Trends in Andrology and Sexual Medicine

Series Editors: E.A. Jannini, C. Foresta, A. Lenzi, M. Maggi

Andrea M. Isidori
Andrea Lenzi

Ultrasound of the Testis for the Andrologist

Morphological and Functional Atlas

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Trends in Andrology and Sexual Medicine

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Foreword

Over the last 10 years, ultrasound examination of the scrotum has become established as a convenient extension of the andrological examination of patients presenting with a complaint of reproductive function. A well-performed ultrasound examination is probably the single most informative diagnostic procedure to facilitate the correct diagnosis of any intra-scrotal abnormality. Pertinently, for the correct interpretation of imaging features, a full clinical history and clinical examination must be integrated with imaging findings, giving a comprehensive overview of the patient's healthcare. Importantly, hormonal assessment and sperm analysis findings are most useful in the presence of any scrotal pathology.

Since 2002, the Department of Experimental Medicine at the "Sapienza" University of Rome has established an ultrasonography unit dedicated solely to andrological cases, equipped with the very latest and technically advanced ultrasound machines. Over this period of time, the unit has performed over 10,000 testicular ultrasound examinations in patients investigated for a variety of conditions. All the images of these examinations have been saved, with a multitude of abnormalities forming the basis of this new atlas-textbook. This core experience has been augmented by contributions of extremely valuable material from a number of international experts to form probably the largest collection in the world, constituting over 1000 ultrasound images, many in full colour, of scrotal pathology.

This unique new reference text for scrotal ultrasonography is also notable for its main emphasis on functional interpretation of the images supplemented by clinical data, thus ensuring that the information will be helpful for clinical management. This approach is intended to increasingly familiarise clinicians with the potential of ultrasonography and, in doing so, encourage incorporation of ultrasound into a central position of the diagnostic pathway in daily clinical practice. Moreover, all the specialists that deal with scrotal pathologies should know the diagnostic potentials offered by the newest imaging modalities (contrast-enhanced ultrasound, elastography), the so-called multiparametric ultrasound examination aiding further development of this modality in the assessment of the scrotal contents.

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We are grateful to all the international collaborators who actively helped assembling this unique collection of cases sending useful pictures of rare conditions: Prof. Amaya Basta, Prof. Demosthenes Cokkinos, Prof. Monica Epelman, Prof. Alex Kirkham, Prof. Malai Muttarak, Prof. Raymond Oyen, Prof. Paul S. Sidhu, Prof. S Boopathy Vijayaraghavan, Prof. Edward Bluth, Prof. Ronald O. Bude, Prof. Kyoung-Sik Cho, Prof. K.C. Dewbury, Prof. Rahul Gupta, Prof. David C. Howlett, Prof. John R. Mernagh, Prof. Martha M. Munden, Prof. Matilde Nino-Murcia, Prof. P. Perimenis, Prof. Osmar De Cassio Saito, Prof. J. Smart, Prof. Simon Strauss, Prof. Krishna M. Surti, Prof. Kunjie Wang, Prof. Neil F. Wasserman and Prof. Aaron F. Wittemberg.

Contents

1 Scrotal and Testicular Anatomy

1.1 Introduction

Ultrasound is considered the primary imaging modality for the scrotum. Knowledge of the clinical (palpatory) and ultrasound findings of the scrotum is crucial. This chapter briefly describes the sonographic appearance of constituents of the normal adult scrotum. The scrotum comprises a sac with several layers, divided into two compartments by the median raphe. Each sac contains a testis and an epididymis with a small volume of fluid. The spermatic cord carries the blood vessels and the vas deferens.

1.2 Layers of the Scrotum

The scrotum is formed by the fusion of the two scrotal swellings, giving rise to an outer cutaneous ridge, or **raphe**, and an inner septum dividing the entire scrotum into two separate pouches, the **median raphe** (Fig. 1.1). The scrotal raphe is continuous with the dartos muscle, which lies in the subcutaneous tissues of the scrotal skin and helps regulate the temperature of the scrotal contents. The layers under the **dartos** muscle are the external spermatic fascia, the cremasteric fascia and the internal spermatic fascia, which is continuous with the transversalis fascia of the abdomen. Overall, the covering of the testes consists of at least six layers: the skin, dartos, external spermatic fascia, **cremasteric layer**, internal spermatic fascia and **tunica vaginalis** [1].

However, on ultrasound the scrotum appears as only three layers—a hyperechoic outer, hypoechoic intermediate and hyperechoic inner layer – with an overall thickness ranging from 3 to 7 mm. Inappropriate air-cooling in the examination room can cause a contraction of the dartos and cremasteric muscle, causing a thickening of the scrotum layers with possible acoustic shadowing or interference.

Inside the internal spermatic fascia is the tunica vaginalis, a portion of the peritoneum that accompanies the descent of the testis into the scrotum during development. After birth, its communication with the main peritoneal cavity obliterates. It consists of a visceral layer adherent to the testis and a parietal layer [2]. The tunica surrounds the testis, except where it is attached to the epididymis. The posterior aspect of the testis – the testis and epididymis site of attachment – is the only part not in continuity with the tunica vaginalis. This detail is important in understating the dynamic of testicular torsion, in particular the bell-and-clapper deformity (see Chap. 5).

A small amount of fluid is normally present between the outer parietal and inner visceral layers of the tunica, which allows the testicle to move freely within the scrotum. This mobility helps protect the testis against traumatic injury. The cleft-like space between the layers of the tunica vaginalis is known as the 'cavum serosum testis' and is usually seen as a thin (1–3 mm), echo-free rim in the area adjacent to the epididymal head. This normal volume of fluid must not be misinterpreted as a hydrocele. Increased fluid allows a better visualisation of the testicular ligament at the bottom of the scrotal sac (Fig. 1.2) and of the upper and lower epididymal ligaments. When the ligament is stretched, it is a clear sign that the vaginal fluid is increased.

Inside the tunica vaginalis is the inelastic tunica albuginea – the connective tissue lining of the testis – from which numerous fibrous partitions, *the septula*, extend vertically into the parenchyma dividing the organ into 200–250 *lobules*. The septula, or septa, converge posteriorly to form the mediastinum testis [3] (Fig. 1.3). The albuginea appears as a hypoechoic thin layer under the hyperechoic tunica vaginalis (visceral layers). An increased fluid collection and thickened hyperechoic vaginalis, with local calcium deposits, are typical accompanying signs of infectious/inflammatory processes occurring within the scrotum (typically epididymitis).

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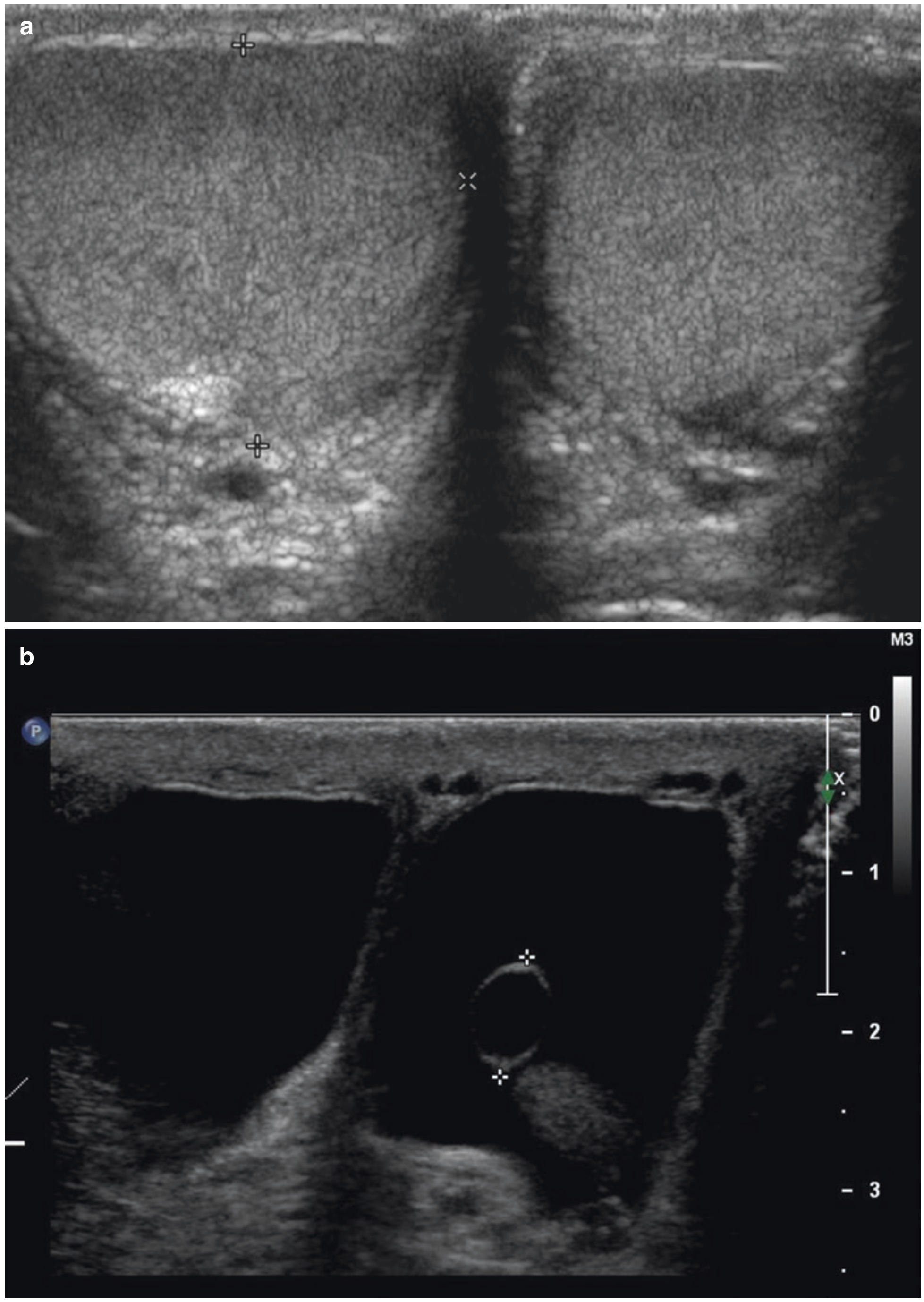


Fig. 1.1 Transverse sonogram of normal scrotum. The testes are confined to each hemiscrotum by the median scrotal raphe (**a**, **b**)

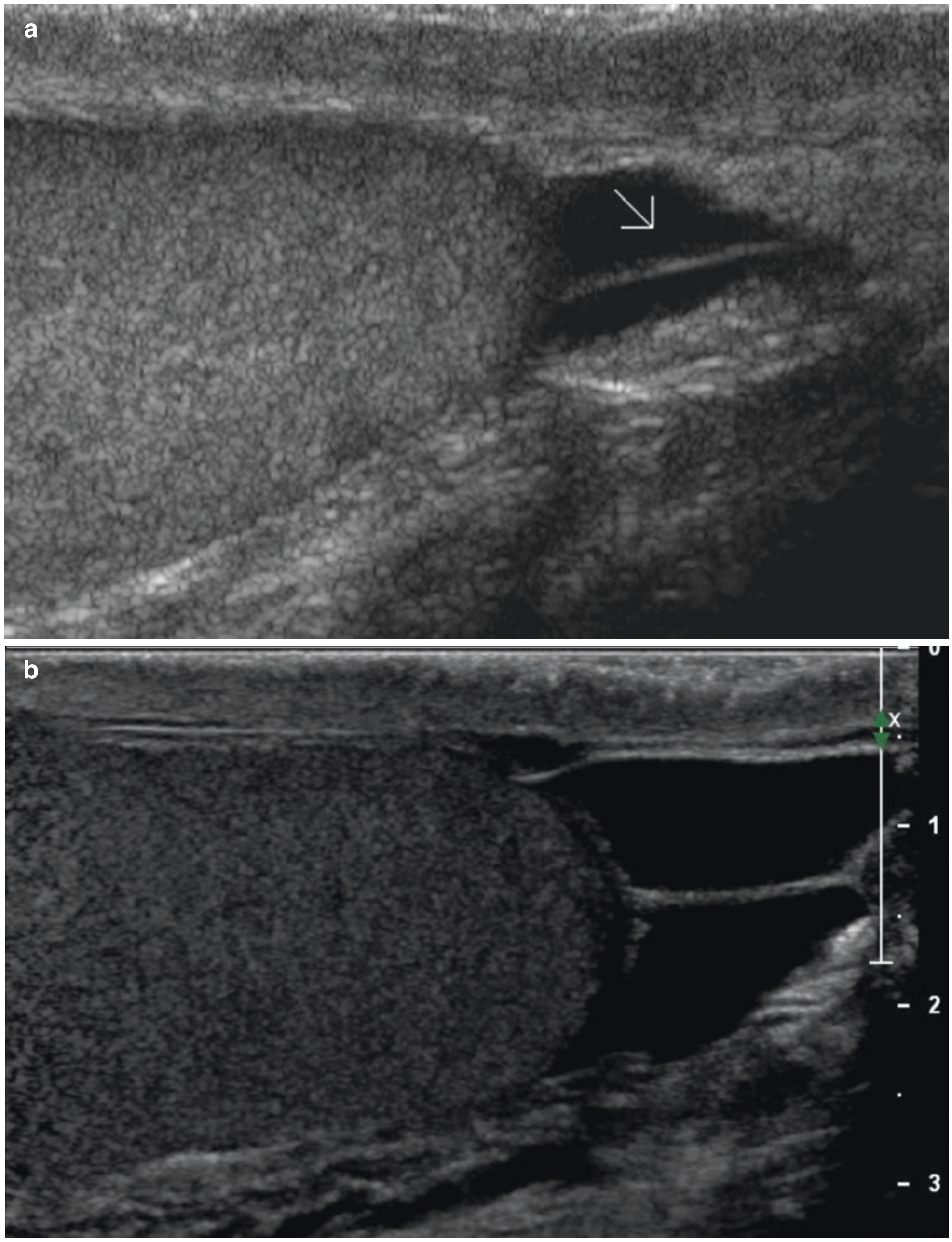


Fig. 1.2 Testicular ligament (*arrow*) (**a**, **b**)

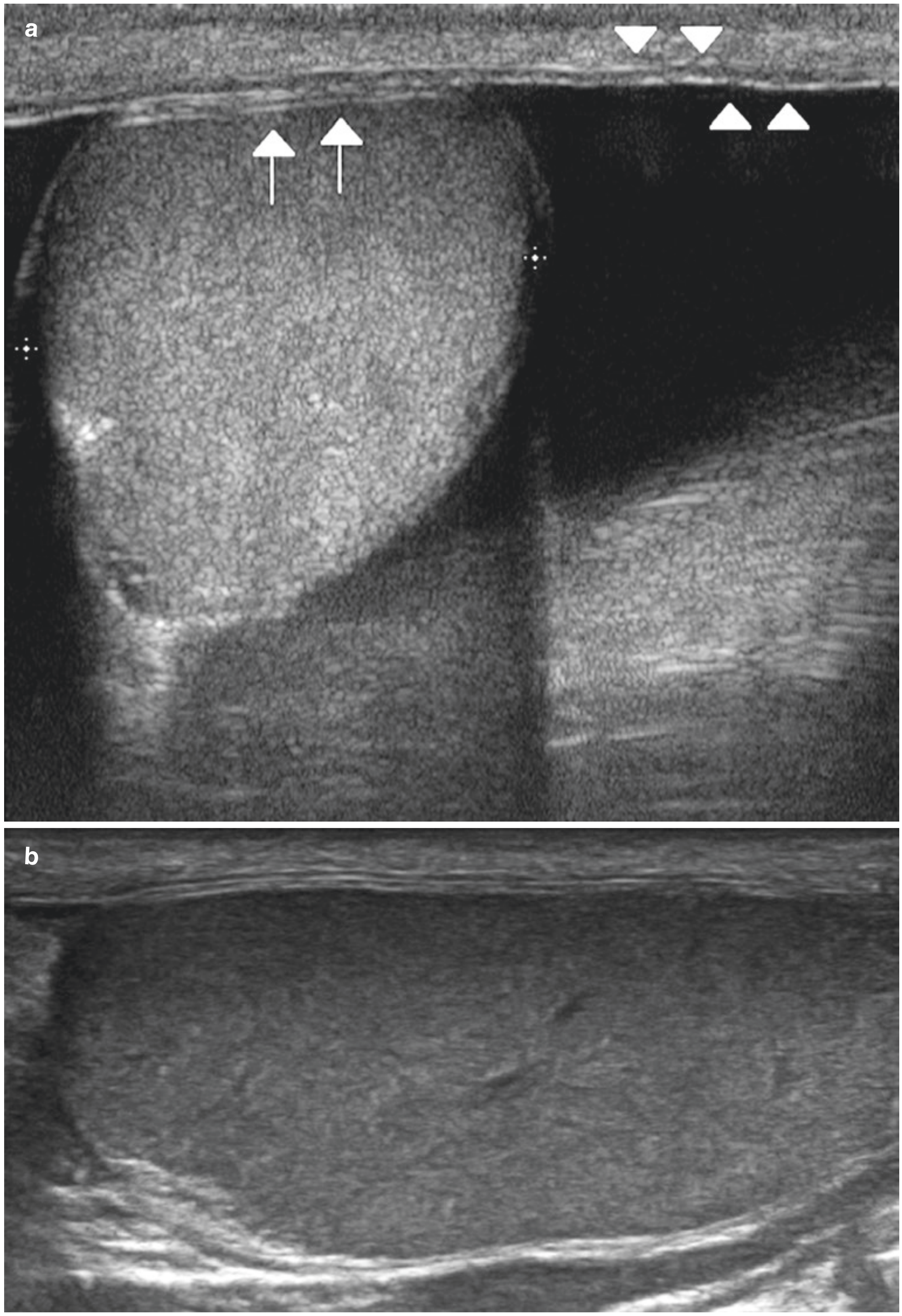


Fig. 1.3 Layers of the scrotum. Normal scrotum. Transverse sonogram of normal scrotum. (**a**) The tunica vaginalis (*arrowheads*) covering the scrotal wall and tunica albuginea (*arrow*) can be seen. (**b**) Longitudinal scans show the three layers of the scrotum. (**c**, **d**) High magnification of the tunica vaginalis and tunica albuginea layers (17-5 mHz linear transducer)

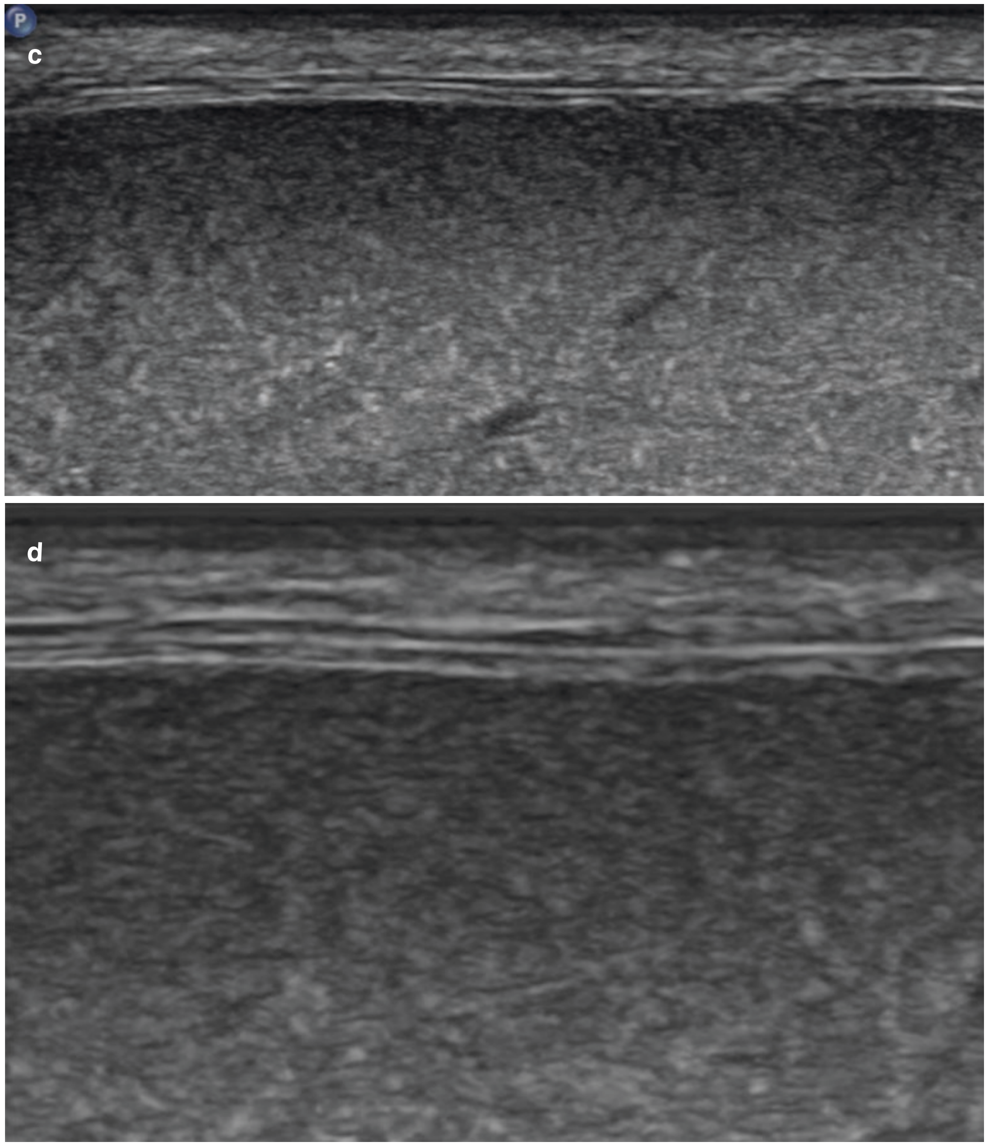


Fig. 1.3 (continued)

1.3 The Testis

The normal adult testis should be symmetrical and ovoid in shape, with the major axis placed vertically and slightly obliquely. The normal testis is located in the scrotum, at least 2 cm below the **external inguinal ring**. In some adult patients, laxity of the inguinal ring can cause the testis to extend to the lower part of the inguinal canal. This location can interfere with normal spermatogenesis. Repeated retraction of the testis into the inguinal canal can also cause inflammation of the epididymis. During scrotal examination physicians should always check the mobility of the testis by pushing it towards the inguinal canal [4].

Even within the scrotal sac, the testis can be positioned abnormally. The most common is a rotation along the vertical axis, with the head of the epididymis located at the bottom and the deferens facing the anterior wall of the scrotum. An altered positioning of the testis due to hyperactivity of the cremasteric reflex is a frequent cause of testicular pain and one of the commonest requests for referral.

The testis is composed of numerous seminiferous tubules separated by radiating septa that divide it into 200–400 lobules. These cannot be identified as separate structures on ultrasound. The lobules contain the seminiferous tubules. The septa run from the innermost fibrous capsule (tunica albuginea) and converge posteriorly to form the central mediastinum testis, which is an invagination of the tunica albuginea [1]. The **septa** (Fig. 1.4) are rarely seen on the sonogram. In some cases they are visible as delicate linear structures, more often identified indirectly by refractory shadowing and disappearing upon slight compression or when the scan direction is changed.

The seminiferous tubules join together to form 20–30 larger ducts, known as **tubuli recti**. These enter the **mediastinum testis**, forming a network of channels within the testicular stroma, called the rete testis. The tubules terminate in 10–15 efferent ductules that drain into the epididymis and then into the vas deferens [3].

The mediastinum testis also contains branches of the testicular artery and testicular vein. It appears as a hyperechoic (echogenic) line, eccentrically located beneath the tunica albuginea on the side facing the epididymis that extends cranio-caudally within the testis. The width of the mediastinum varies widely among individuals, without any diagnostic implications (Fig. 1.5). The mediastinum testis is less obviously seen in the pre- or peri-pubertal testis.

The **rete testis** can be seen in 15–20% of patients as a hypoechoic area with a striated configuration peripheral or adjacent to the mediastinum testis. A small number of **vessels** can occasionally be detected in greyscale sonography as tapering curvilinear hypoechoic structures, with or without echogenic borders extending from the anterior to the posterior margin of the testis. Colour Doppler sonography confirms their nature [4, 5].

The development stage of the germ cell elements and tubular maturation determines the **echotexture of the testicles**. Prepubertal testes are typically of low echogenicity, which increases progressively to reach a medium echogenicity in post-pubertal testicles. The normal adult testis has a homogeneous granular echotexture composed of uniformly distributed medium-level echoes, resembling the echogenicity of the normal thyroid gland (Fig. 1.6). The increase in echogenicity during puberty is primarily the result of growth of the seminiferous tubules, which increase in diameter and develop a lumen [1]. It can be argued that the opening of the seminal tubules secondary to spermiation confers normal echogenicity to the testis. In fact, patients with azoospermia due to Sertoli cell-only syndrome exhibit a very low testis echogenicity (similar to that of the prepubertal testis) (Fig. 1.7) in comparison with other patients affected by secretory azoospermia, for example, due to a late-stage arrest in spermatogenesis (spermatid arrest), whose testis can be normal in size and echogenicity. The echotexture of the testis could provide potentially useful information to physicians. Unfortunately, there are no standard 'reference' values to define the 'normal' echogenicity of the testis. For this reason we suggest the development of an in-house reference based on fixed scanning parameters.

Besides the vessels, the rete testis and mediastinum, the echotexture of the normal testicular parenchyma, including in prepuberty, should appear **homogeneous**. Non-homogeneity should be interpreted as a pathological finding (Fig. 1.8). Similarly, the healthy testis has a firm consistency and cannot be deformed by the pressure of the probe; any variation towards an increase (hard) or reduction (soft) in the palpatory consistency should be interpreted as a pathological finding. Changes in echogenicity and consistency are correlated to reduced semen production. In contrast, steroid hormone production may still be normal even in a severely abnormal testis.

Sequelae of surgical manipulation, orchidopexy, or biopsies can be detected in two thirds of cases as localised alterations in the echogenicity of the parenchyma. **Biopsies** are usually seen as avascular oval or triangular hypoechoic areas beneath the albuginea (Fig. 1.9).

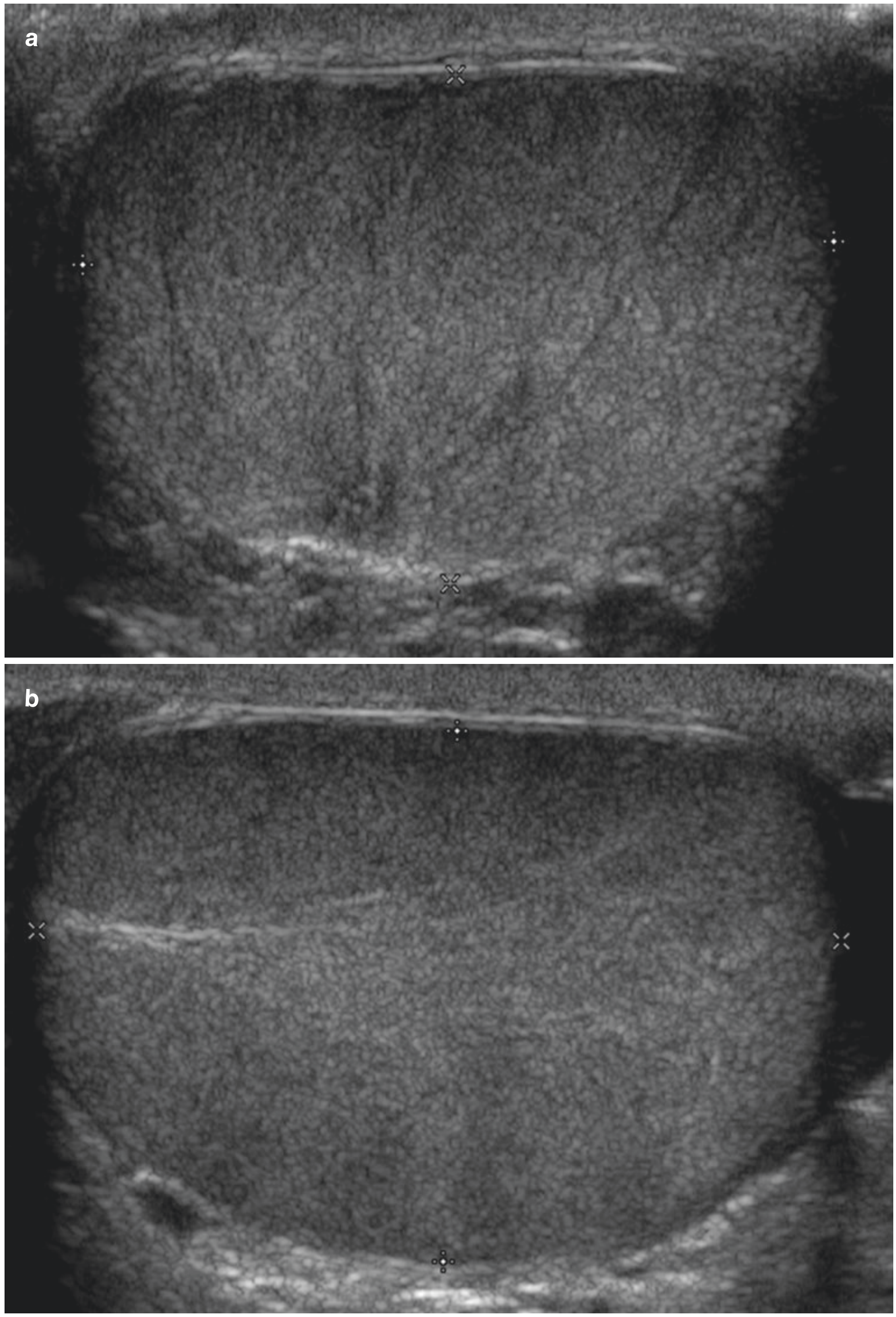


Fig. 1.4 Septa testis. (**a**, **b**) Transverse scans. The septula testis appears on sonogram as delicate linear structures, departing from the mediastinum testis and extending vertically into the parenchyma dividing the organ into 200–250 lobules

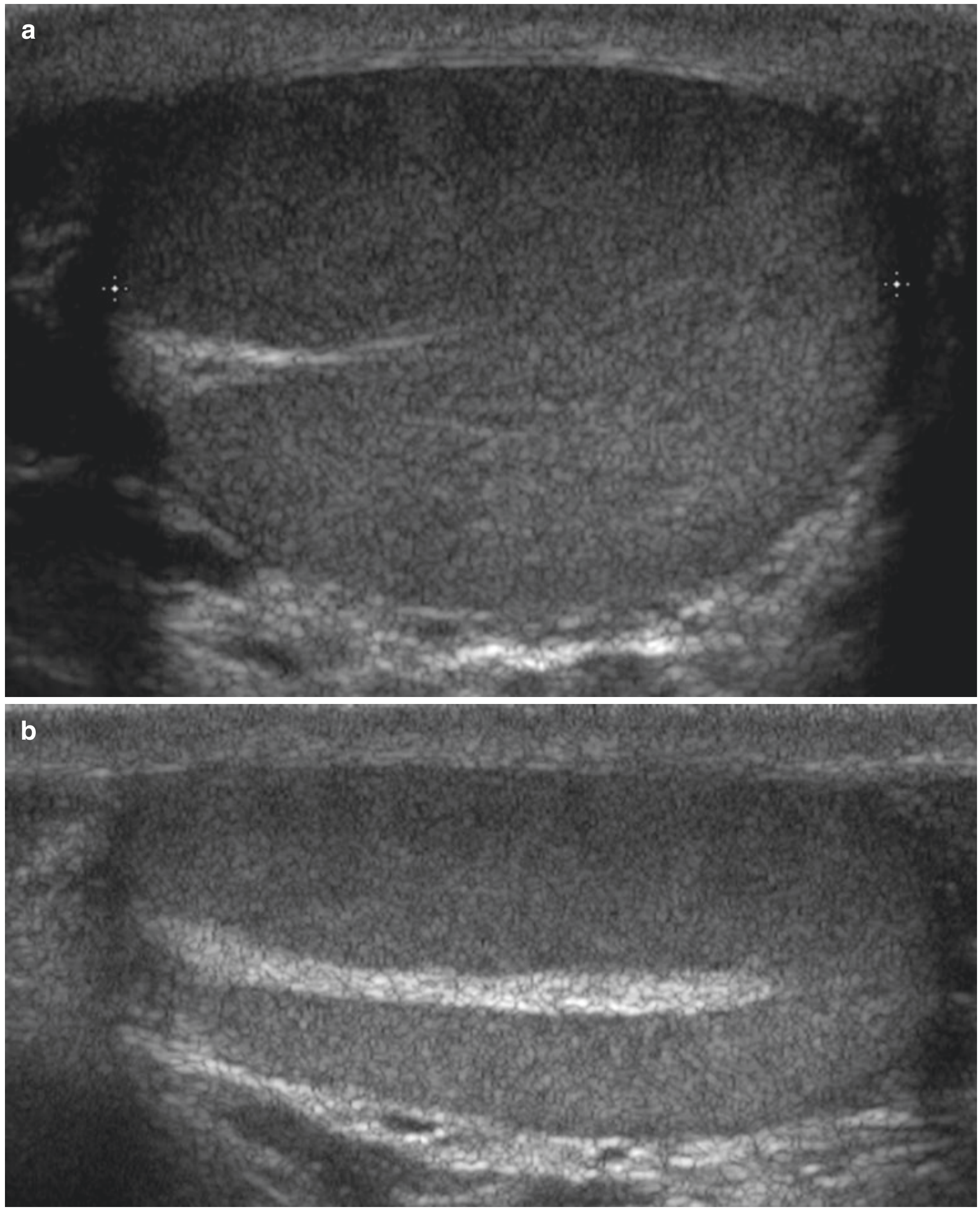


Fig. 1.5 Mediastinum testis. It appears as an eccentrically located hyperechoic (echogenic) triangular shape on transverse scan (**a**) and linear shape on longitudinal scan (**b**) of fibrofatty tissue

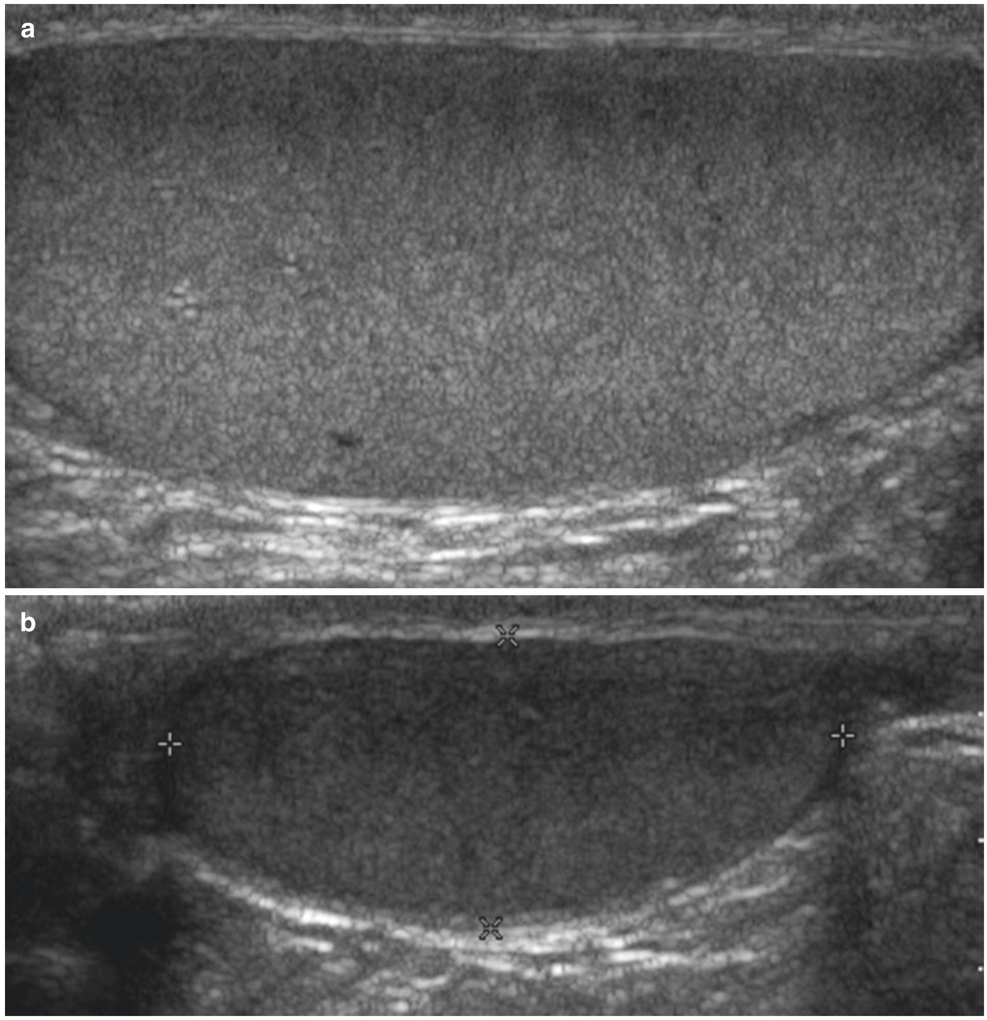


Fig. 1.6 Normal echotexture of the testis. Longitudinal scan reveals the normal homogeneous, granular echotexture composed of uniformly medium-level echoes (**a**) typical of the adult testis, while prepubertal testis (**b**) is typically of low echogenicity

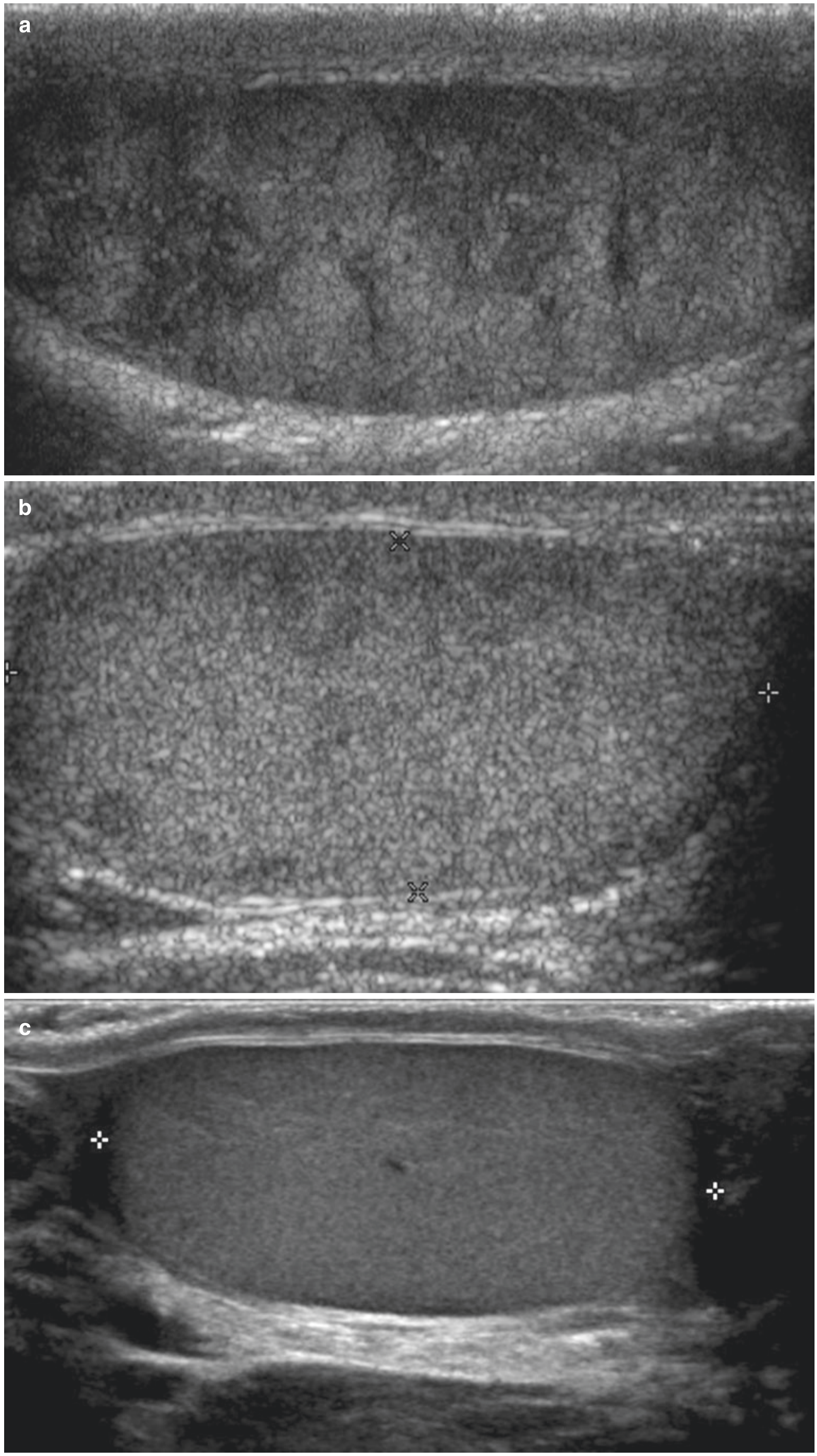


Fig. 1.7 Abnormal echotexture of the testis. Longitudinal scan reveals a very low testis echogenicity due to previous orchitis (**a**) and Sertoli cell-only syndrome (**b**, **c**)

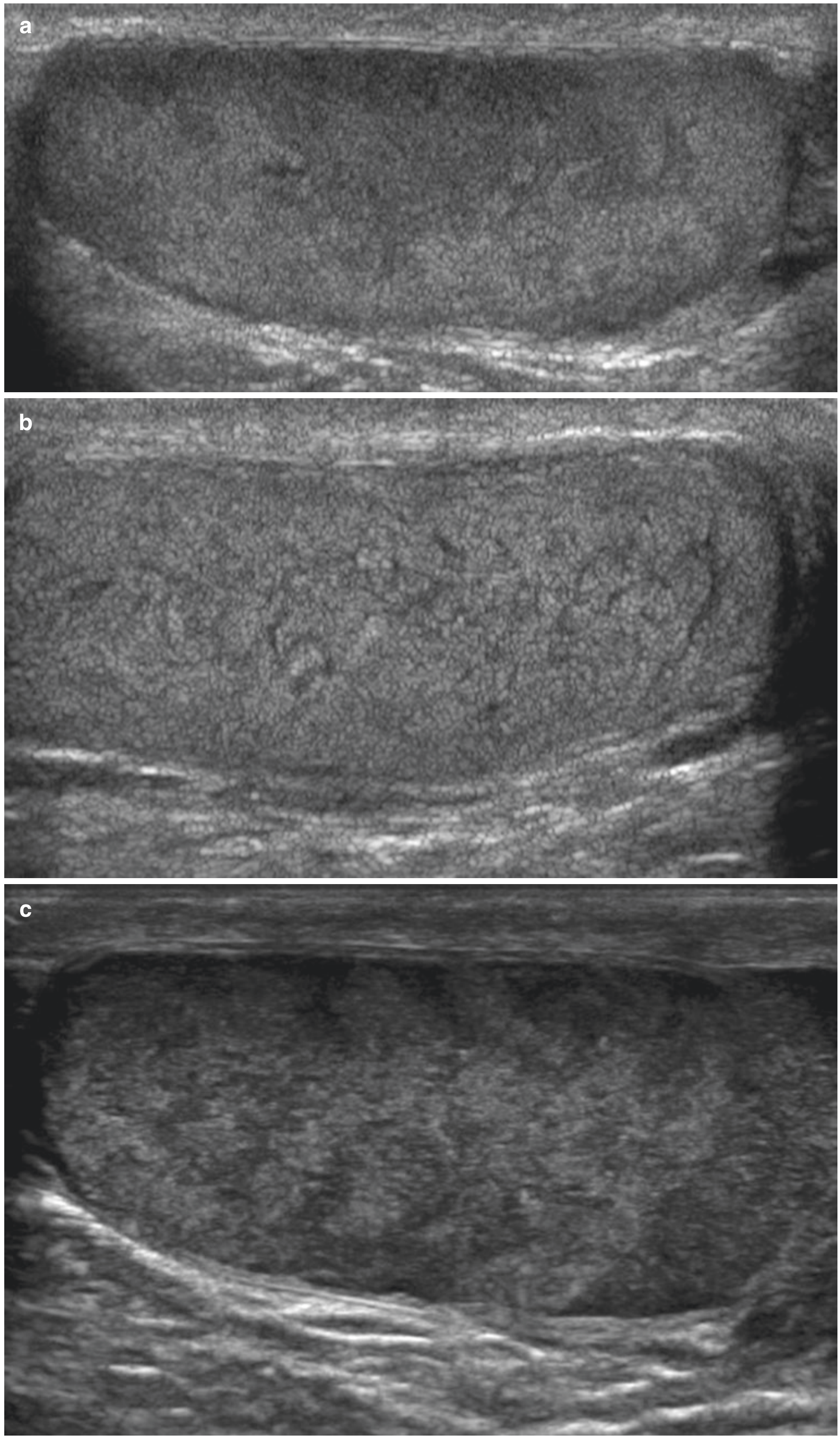


Fig. 1.8 Abnormal echotexture of the testis. Longitudinal scans show the maculated and striated pattern (**a**, **b**) and with areas of patchy low reflectivity that cannot be considered a normal finding (**c**)

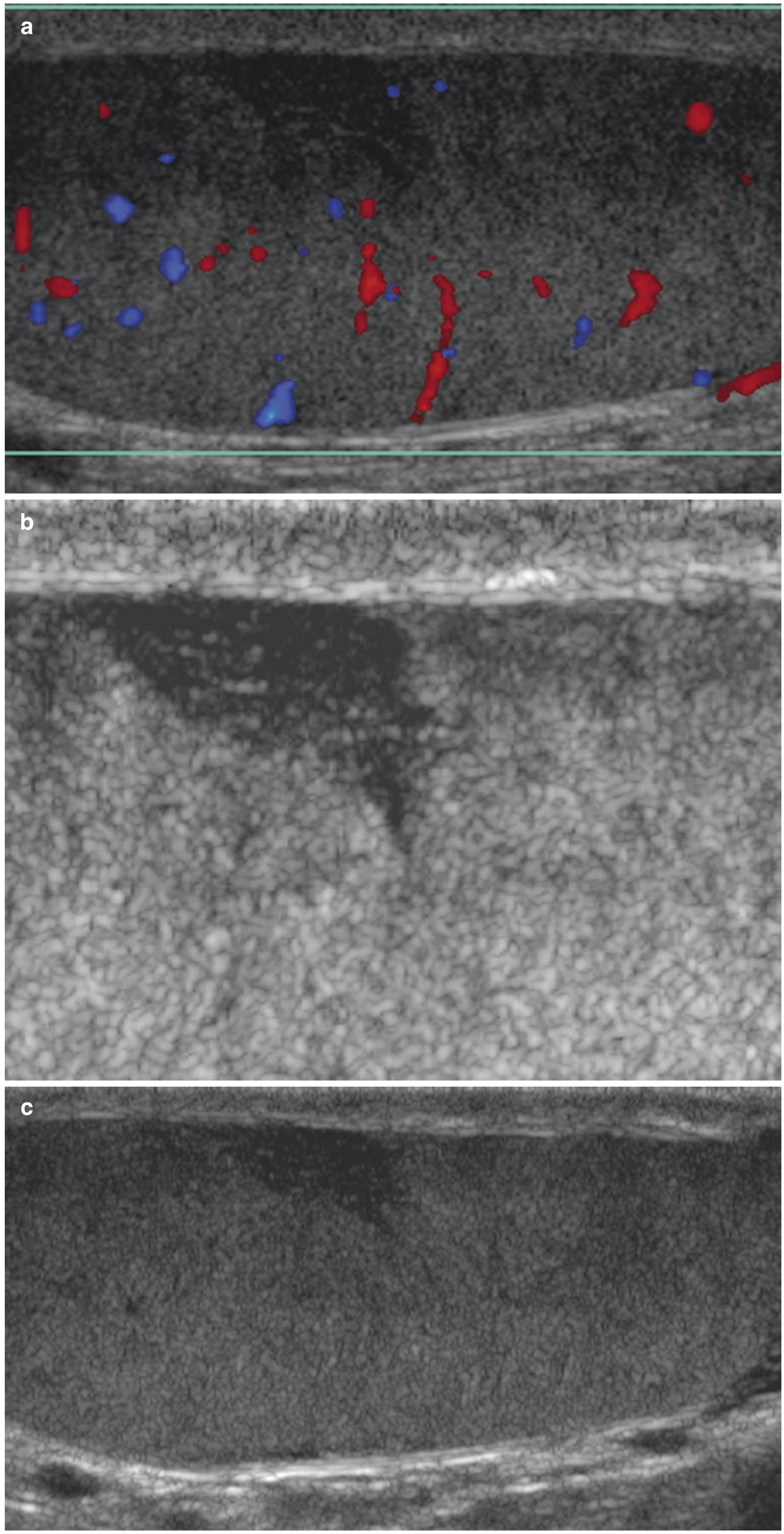


Fig. 1.9 Testicular biopsy. Longitudinal scan shows in the periphery of the testis a cuneiform, avascular area, sign of a precedent scrotal biopsy (**a–c**)

Artefacts can simulate a non-homogeneous appearance of the testis. Most common artefacts are refractory shadows produced by the mediastinum testis or by obliquely oriented testicular septa (Fig. 1.10). They can be avoided by using a slight compression with the transducer or by changing the scan orientation. In addition, fluid collection within (cysts) or surrounding the testis (hydrocele) amplifies the echogenicity of the underlying parenchyma [6].

Testicular volume varies with age. It is generally greatest between the age of 20 and 26, and marginal decline occurs from the 50s onwards. There is also wide variability among subjects and ethnic groups. However, both testes normally have the same size. Any variation greater than 25% between the two testicular volumes should be reported. The size of the testis is closely related to total sperm count, sperm motility and morphology and daily sperm production.

Ultrasound measurement has been shown to have a good correlation with actual testicular volume and is preferred over clinical assessment, especially where intrascrotal disorders such as hydrocele, haematocele, varicocele or epididymitis preclude an accurate clinical measurement.

Correct measurement of testicular volume is crucial, especially in pubertal boys and patients referred for infertility. Testicular volume measured by ultrasound is generally 2–4 mL lower than that estimated with the orchidometer [7]. Testicular volume can be easily calculated by multiplying the three longest diameters and adjusting the value as in the rotational ellipsoid equation: $L \times T \times AP \times 0.52$. This method has an average error of 15%. Accuracy increases with larger volumes and decreases with smaller volumes. With volumes of less than 4 mL, the error may be as high as 50% [8]. Recent studies revealed that a correction factor of 0.71 is probably the more accurate to estimate the calculated testicular volume. This has been confirmed both in animal and human studies using the water displacement methods [8, 9]. The figures obtained with this formula ($L \times T \times AP \times 0.71$) are also closer to those obtained with the orchidometer.

The physician should take the necessary time to scan the maximal longitudinal and transverse axes. The longitudinal diameter (L, between the upper and lower pole) ranges from 44 to 58 mm, the transverse diameter (T, between the medial and the lateral face of the testis) from 18 to 24 mm and the anteroposterior diameter (AP, between the two margins) from 30 to 36 mm (Fig. 1.11). Combination of accurate volume measurement and appraisal of the echotexture of the testis provides important preliminary clinical information on reproductive function.

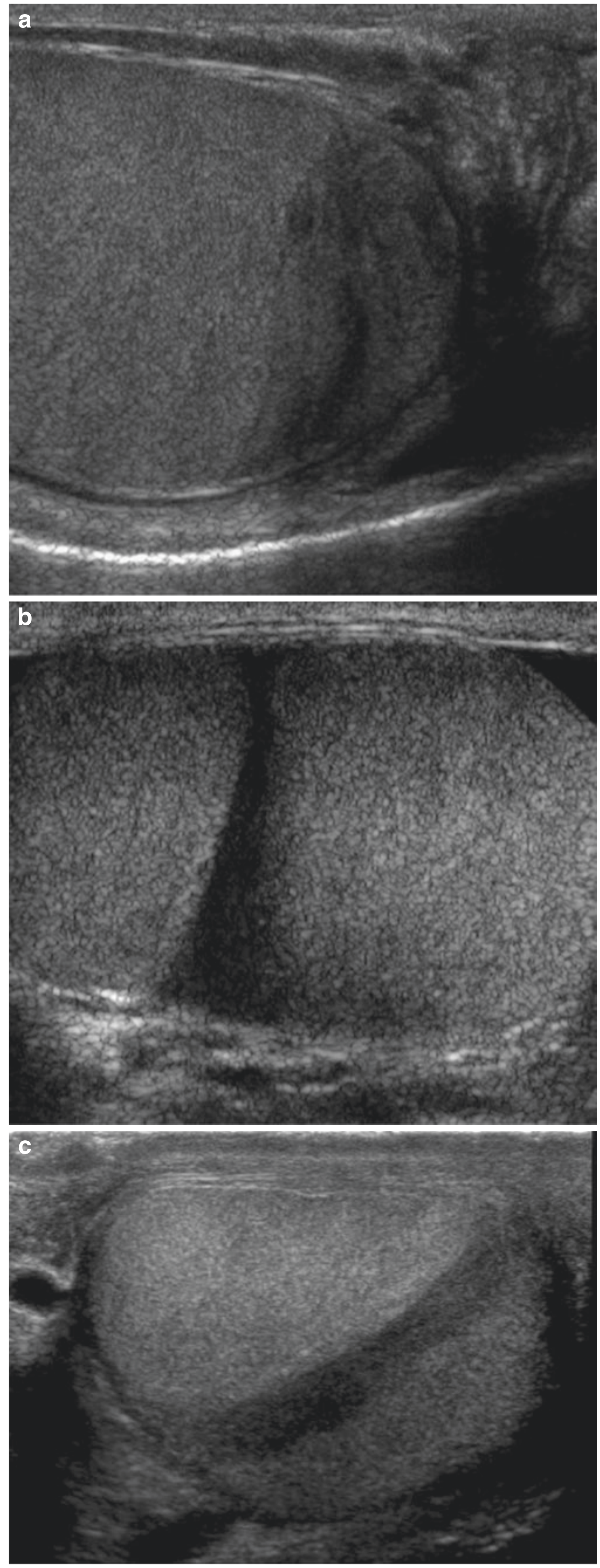


Fig. 1.10 Artefacts in the echotexture of the testis. Artefacts can simulate a non-homogeneous appearance of the testis (**a**) and sometimes can produce an obliquely oriented testicular septa (**b**, **c**)

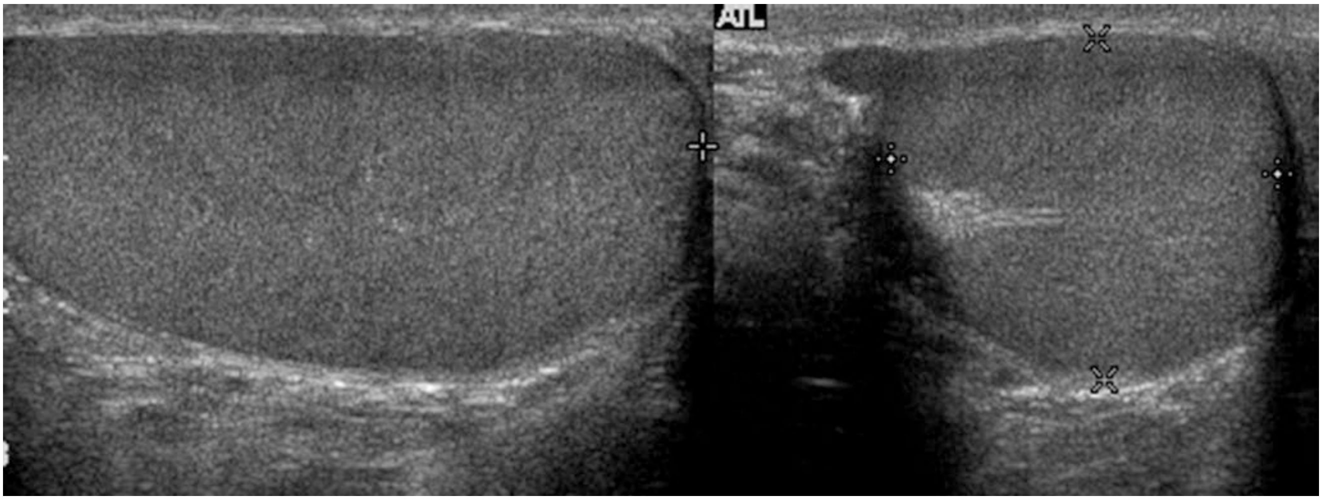


Fig. 1.11 Testicular maximal diameters. Longitudinal and transverse scan of the testis

1.4 The Epididymis

The epididymis is an elongated structure situated dorsally or posterolaterally to the testis. It is normally 5–7 cm long and is composed of three parts: the head, the body and the tail. The **head** is located at the superior pole of the testis, firmly attached by the efferent ductules. This is the largest portion of the epididymis and is round or triangular, with a maximum diameter of 10–12 mm. The rest of the epididymis gradually tapers (**body** 2–3 mm) towards the tail (Fig. 1.12) [1]. The

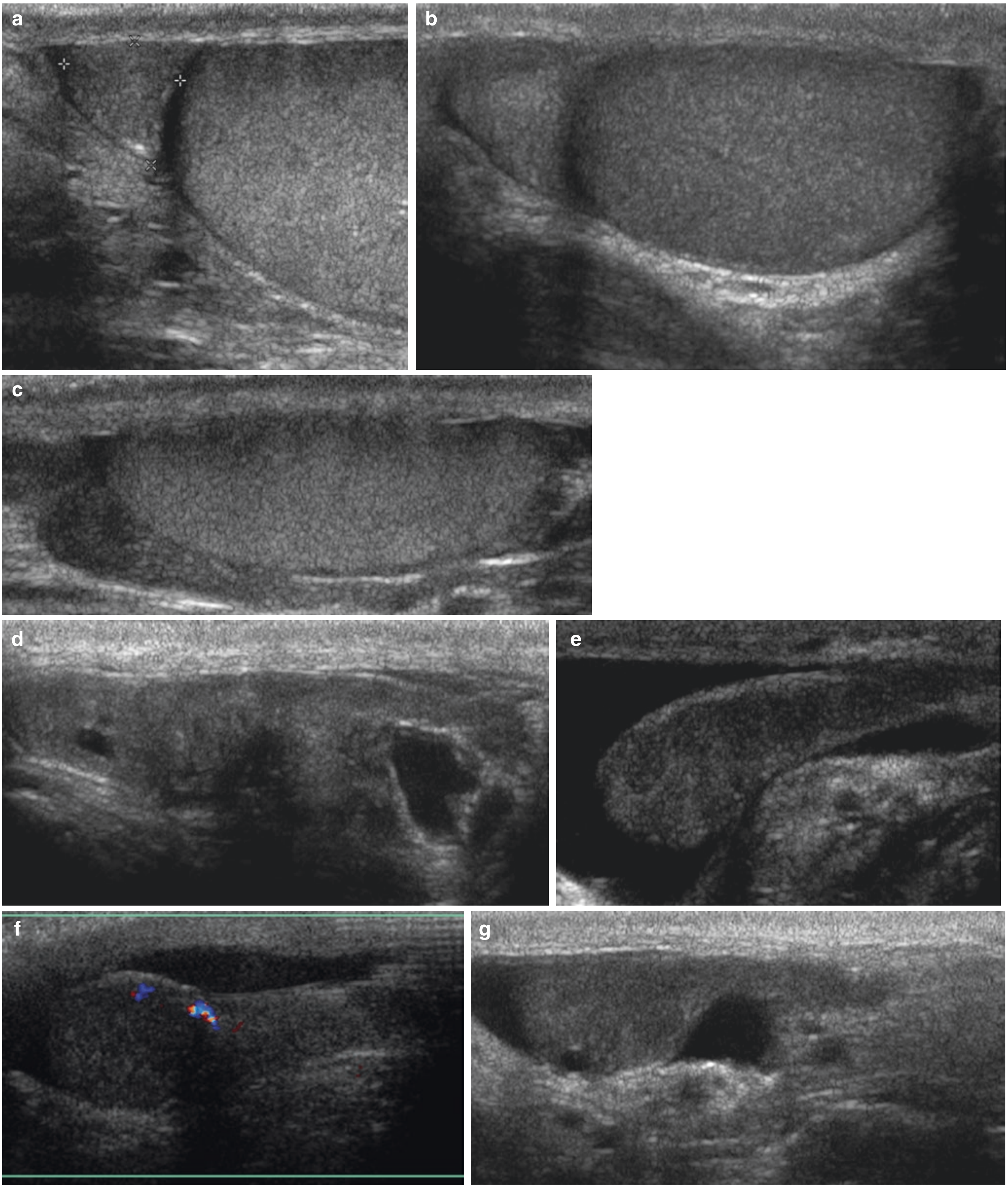


Fig. 1.12 Epididymis. Head (**a**, **b**), body (**c–h**) and tail (**i–k**) of the epididymis. In figure **i** it can be seen within the tail a simple cyst of the epididymis. The echogenicity of the epididymis is generally comparable to that of the testis, even if the head is usually sighted more echogenic than the body and tail

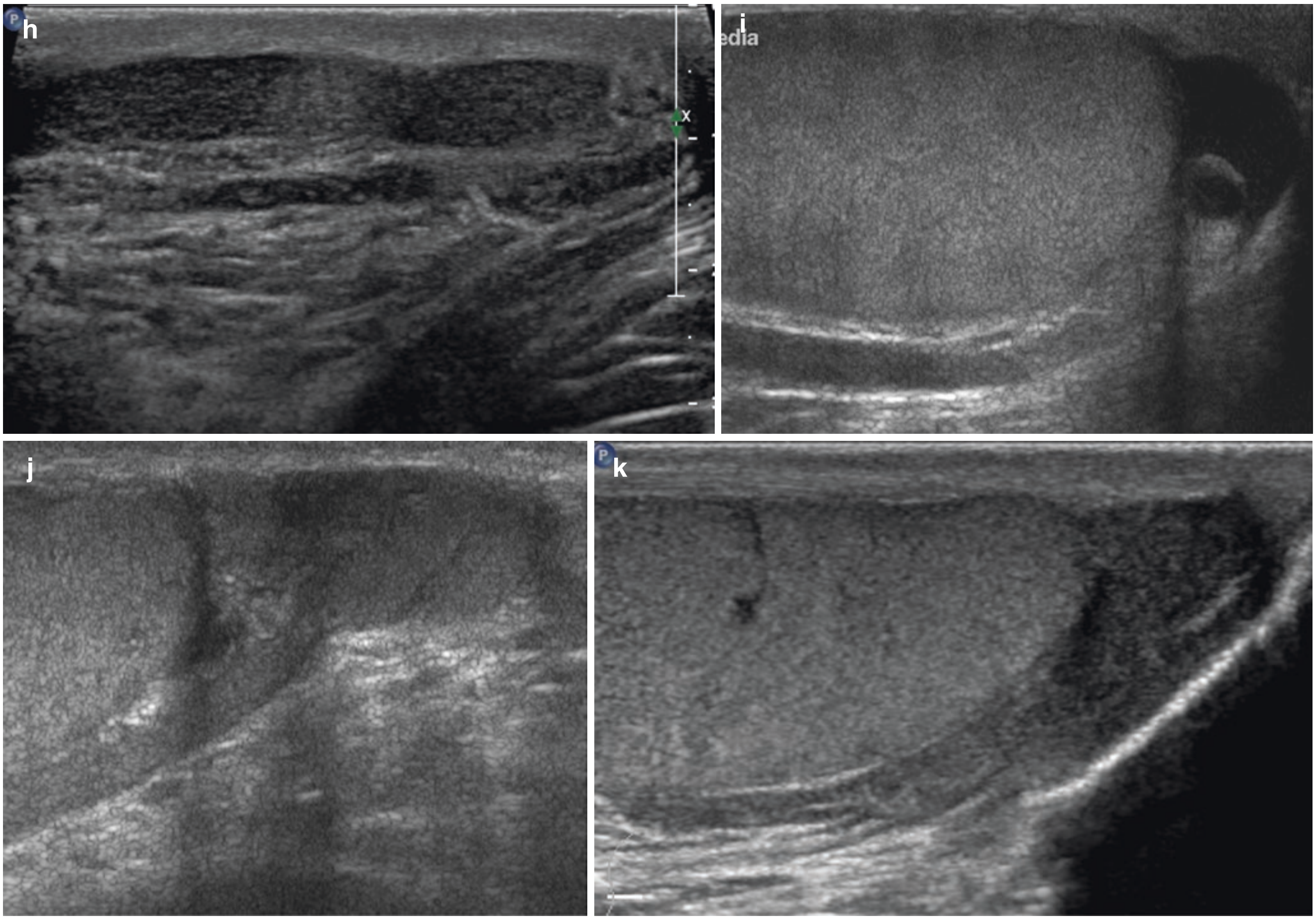


Fig. 1.12 (continued)

head of the epididymis is formed by 10–15 efferent ductules joining together to form a single convoluted duct, the ductus epididymis. This forms the body and the majority of the tail. It is approximately 6 m long and follows a tortuous course from the head to the tail of the epididymis.

The **tail** is located at the inferior pole of the testis and is bound to the testis in a crescent-like manner by fibrous connective tissue. The tail is 1–3 mm thick and is normally flat. Its single duct forms an acute angle at the inferior aspect of the tail and runs cranially to become the vas deferens (Fig. 1.13), which continues into the spermatic cord.

The echogenicity of the epididymis is comparable to that of the testis. The head of the epididymis is usually slightly more echogenic than the body or tail, probably because it contains more tubules and therefore has an increased number of interfaces [10]. Blood flow can be detected using colour flow Doppler or power Doppler imaging. The epididymis has a crucial role in the maturation of the spermatozoa and cannot be reduced to a simple duct. Alterations in the structure of the epididymis can impair sperm motility, morphology and number, independently of any associated obstruction. Inhomogeneous epididymides are frequently associated with a higher number of leucocytes in the seminal fluid.

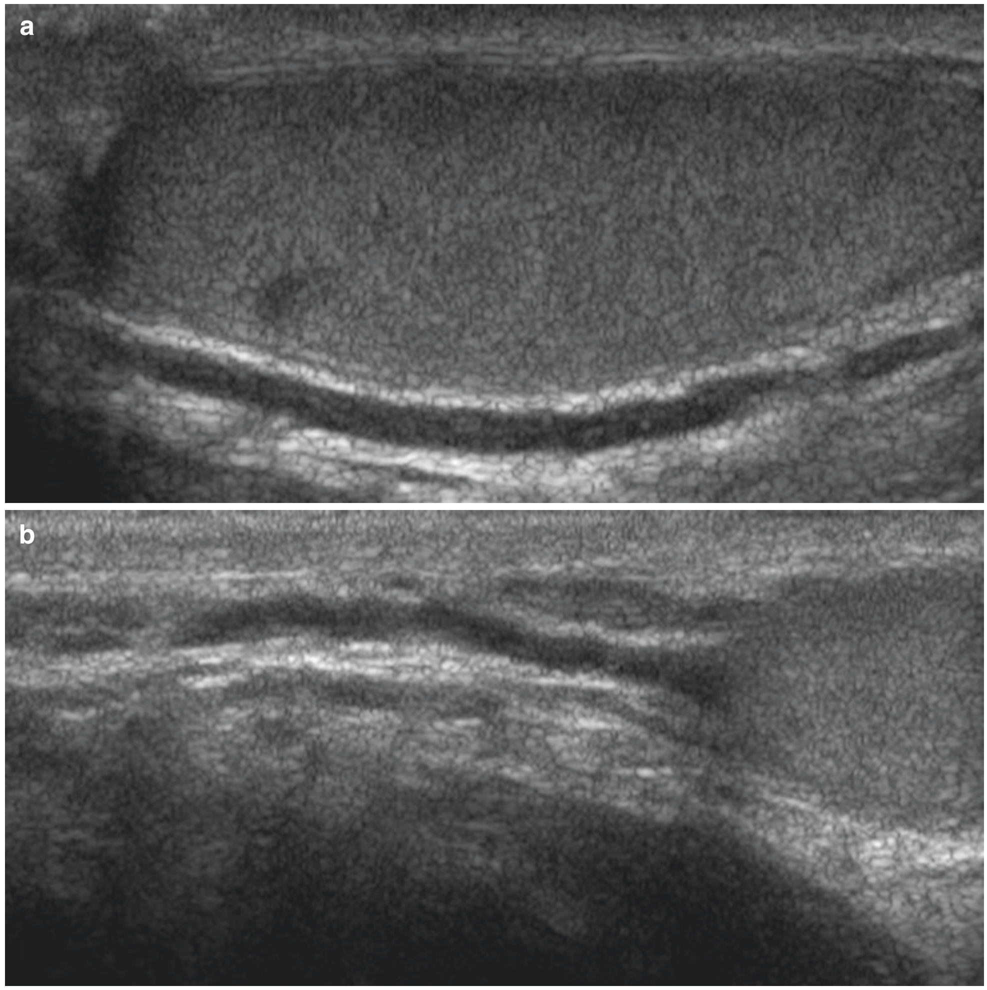


Fig. 1.13 The vas deferens. A single duct forms the tail of the epididymis and runs cranially to become the vas deferens (**a**), which continues in the spermatic cord (**b**)

1.5 The Spermatic Cord

The spermatic cord is a cylindrical complex originating from the posterior margin of the testis and terminating at the internal inguinal ring passing through the inguinal canal. It contains a number of structures, all enveloped by the spermatic fascia: the ductus deferens; the testicular, cremasteric and deferential arteries; the pampiniform plexus of veins; and the nerves and lymphatic system of the testis.

It is difficult to identify sonographically, as its path through the inguinal canal is obscured by the surrounding echogenic fat (Fig. 1.14). The inferior part of the spermatic cord within the scrotum is more easily identifiable, especially by colour Doppler imaging [1, 4].

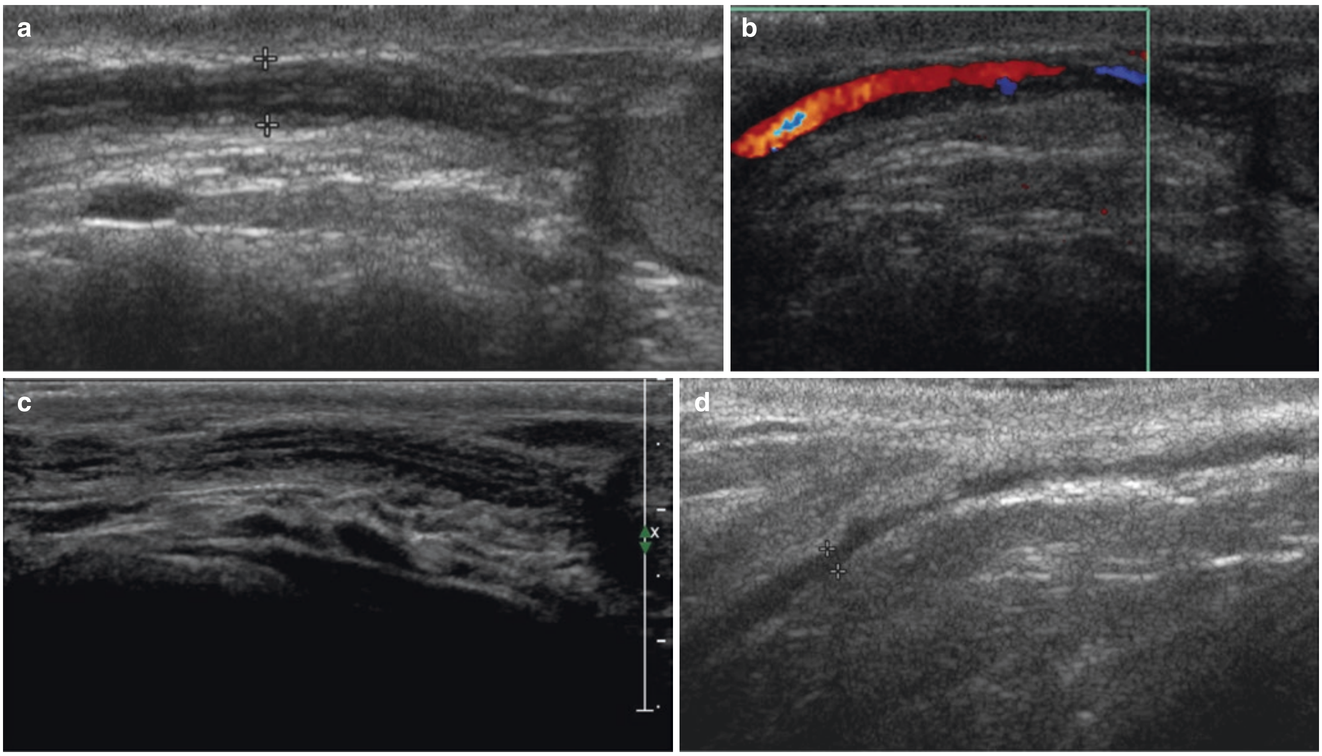


Fig. 1.14 Spermatic cord. Longitudinal scans of the inguinal canal show the spermatic cord, occasionally obscured by the surrounding echogenic fat. It originates from the posterior margin of the testis and terminates at the internal inguinal ring passing through the inguinal canal (**a–d**)

1.6 Arteries

Assessing testicular and epididymal perfusion is an important part of every scrotal examination. Blood is supplied by three arteries passing through the inguinal canal.

The larger **testicular arteries** arise from the anterior aspect of the aorta, immediately below the origin of the renal arteries, and provide the vast majority of the blood supply to the testicular parenchyma. They have a low-resistance pattern on colour Doppler evaluation (Fig. 1.15). The **deferential**

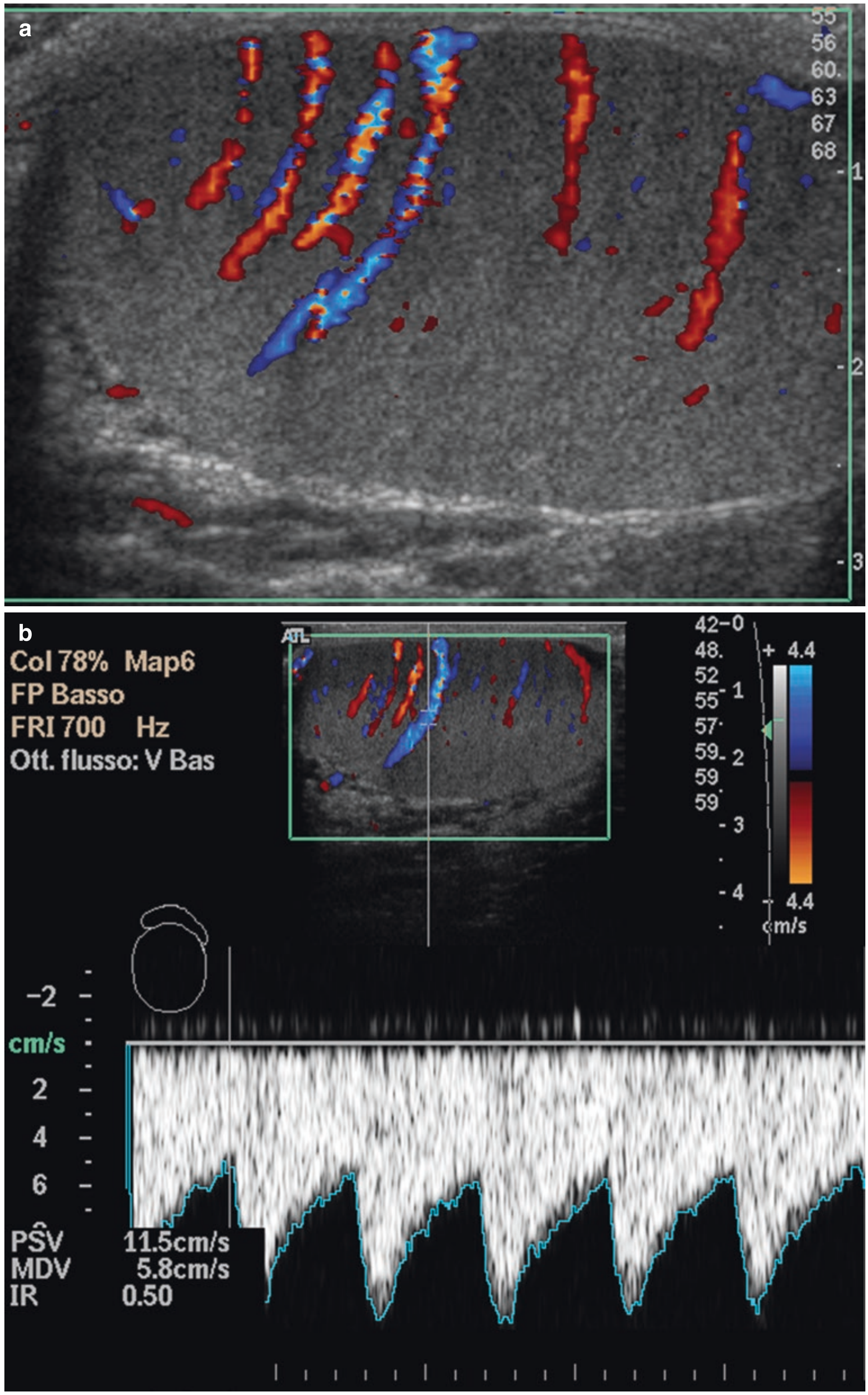


Fig. 1.15 Intratesticular arteries. Colour Doppler scan shows intratesticular arteries (**a**, **c**) with low-resistance pattern on colour Doppler evaluation as shown by detectable end-diastolic flow velocities (**b**, **d**). Capsular subalbugineal arteries (**e**, **f**). Spectral analysis of testicular arteries in peri-pubertal boys (**g**, **h**)

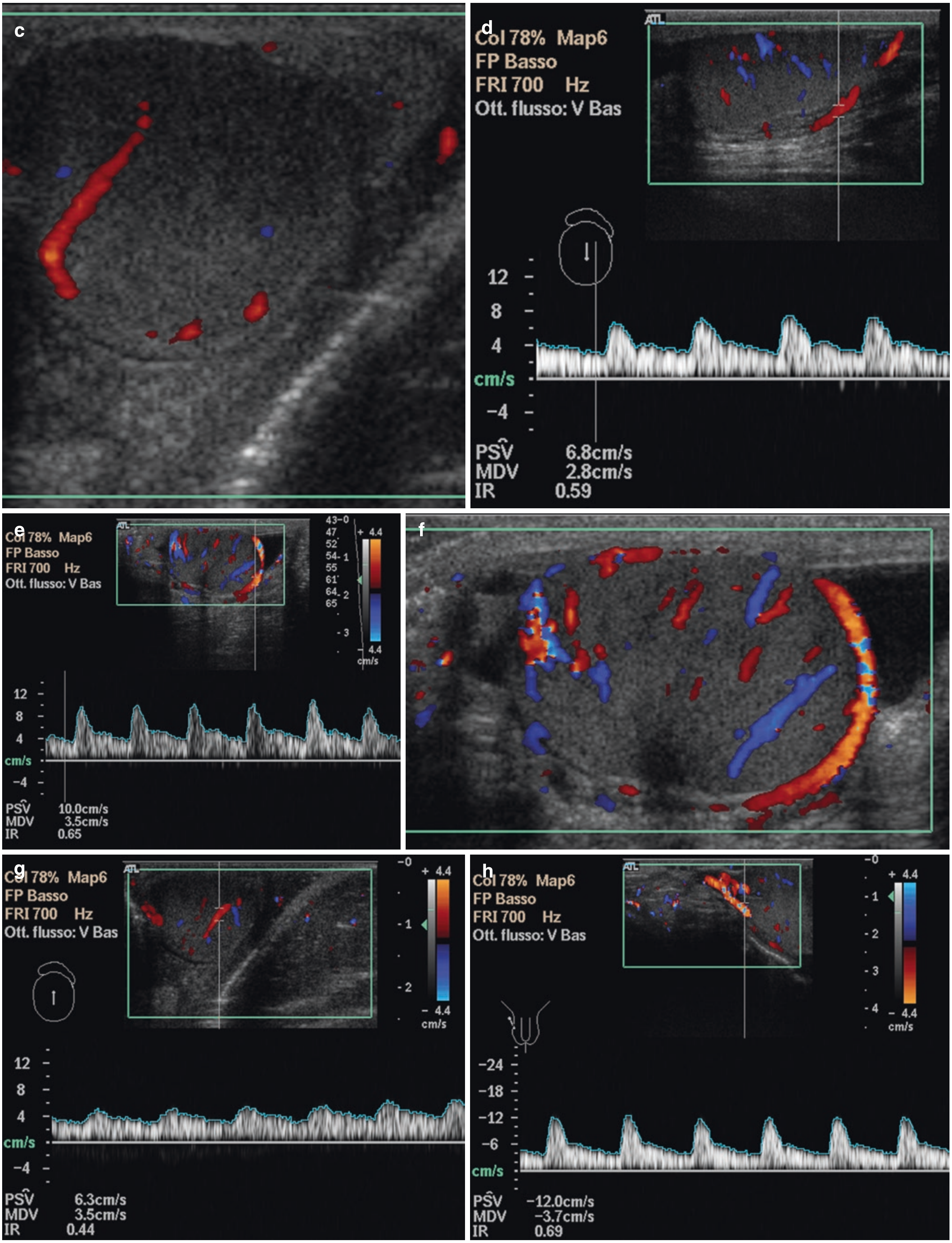


Fig. 1.15 (continued)

arteries originate from the vesical arteries; they have a high-resistance pattern and perfuse the epididymis and vas deferens (Fig. 1.16). The **cremasteric arteries** from the inferior epigastric arteries also have high-resistance flow and supply the scrotal wall. The three arteries have small anastomoses with one another at the level of the cord and scrotal fascia, but

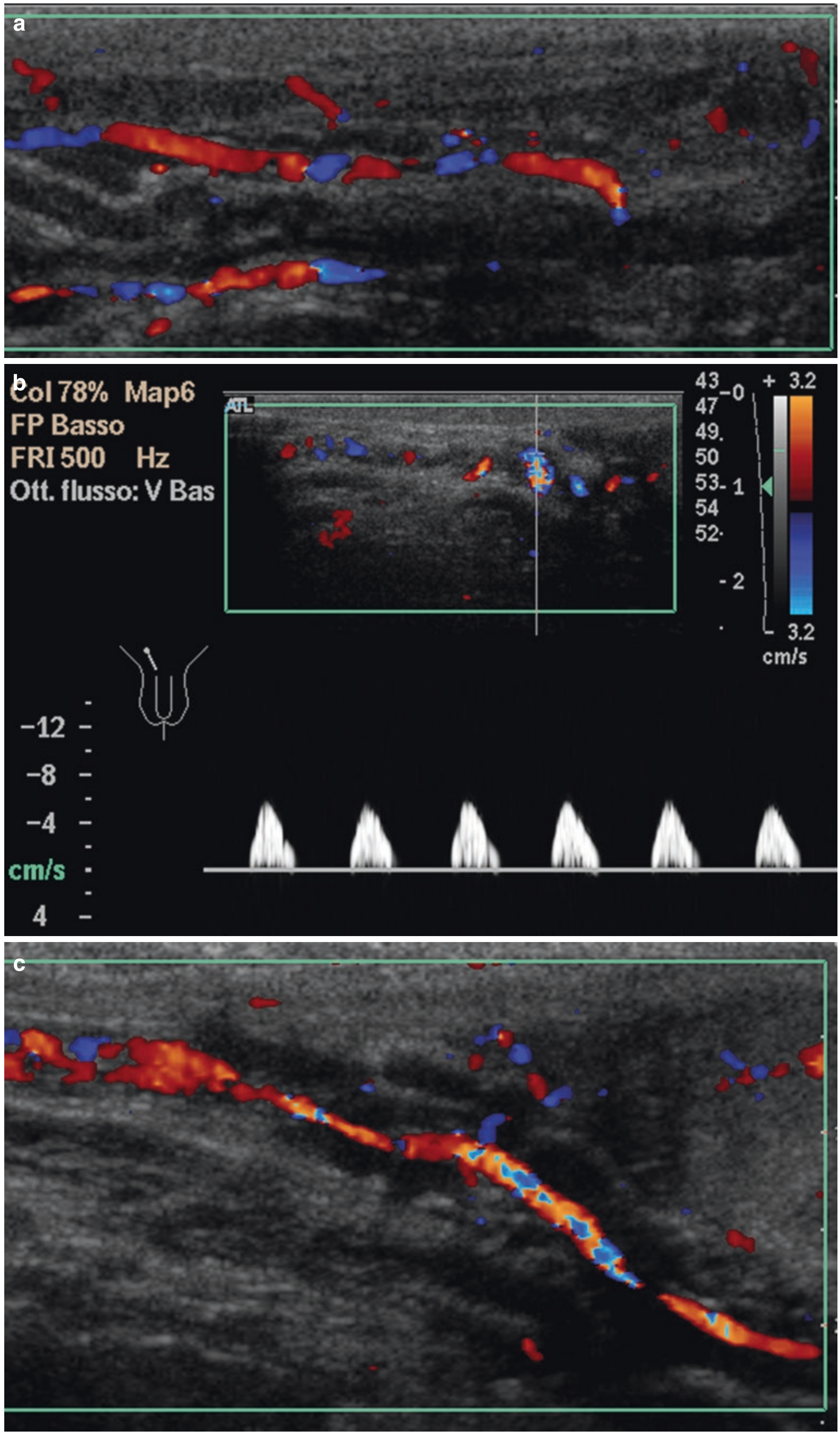


Fig. 1.16 Extratesticular arteries. Colour Doppler (**a**, **c**, **e**) and spectral Doppler (**b**, **d**, **f**) of the extratesticular scrotal artery supply (deferential and cremasteric arteries) show high-resistance waveform with absence of flow in diastole

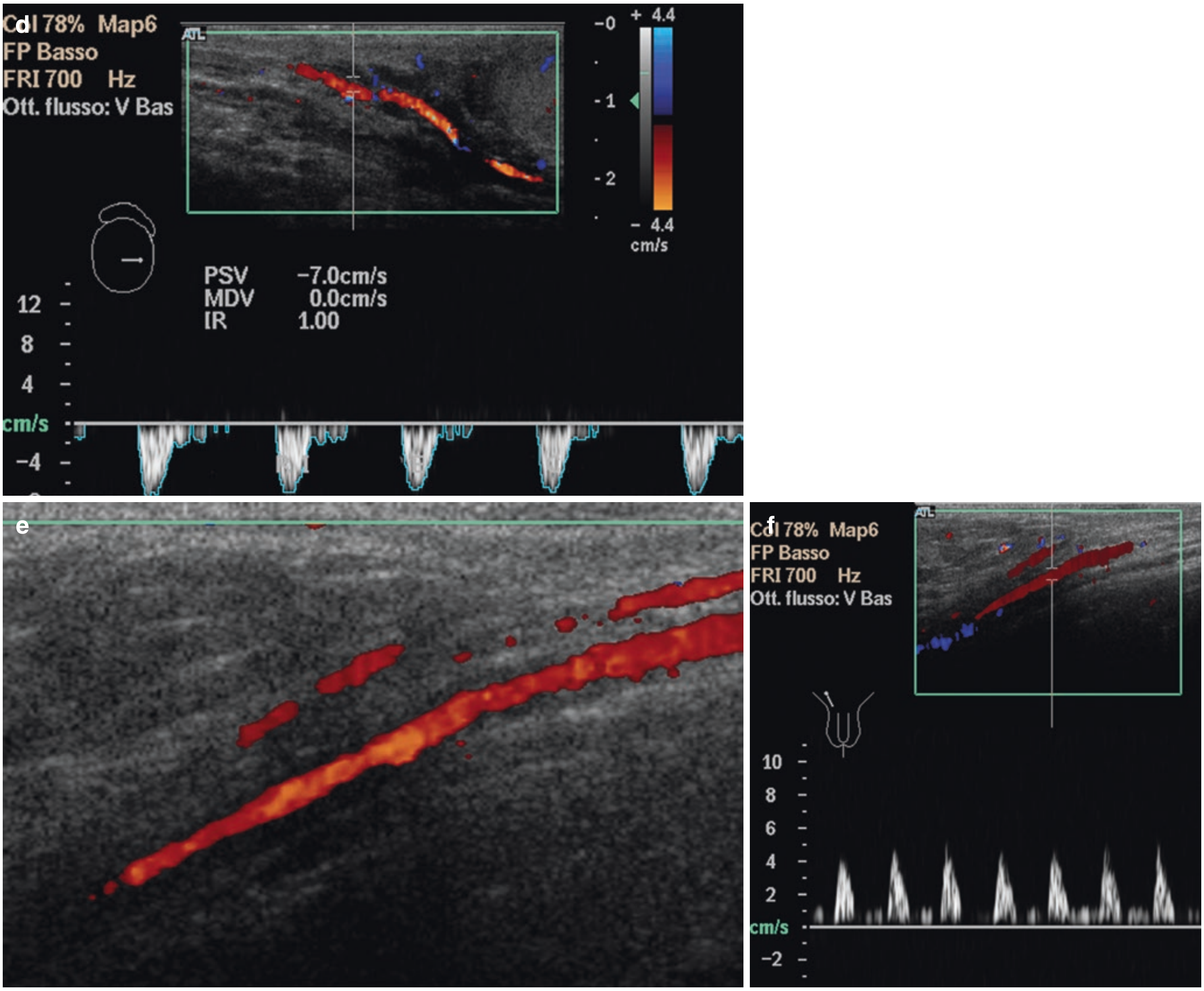


Fig. 1.16 (continued)

there are no known intratesticular anastomoses. The testicular arteries split off from the mediastinum testis, under the tunica albuginea, to form a circumferential group of arteries (**tunica vasculosa**). Smaller arteries branch off from these to the testicular parenchyma towards the mediastinum. In 10–20% of men, a large branch of the testicular artery enters at the mediastinum and runs across the testis to form capsular branches on the opposite side. This is called the **transmediastinal artery** (Fig. 1.17). Transmediastinal/transtesticular arteries are seen unilaterally (50%) or bilaterally (25%), usually in the

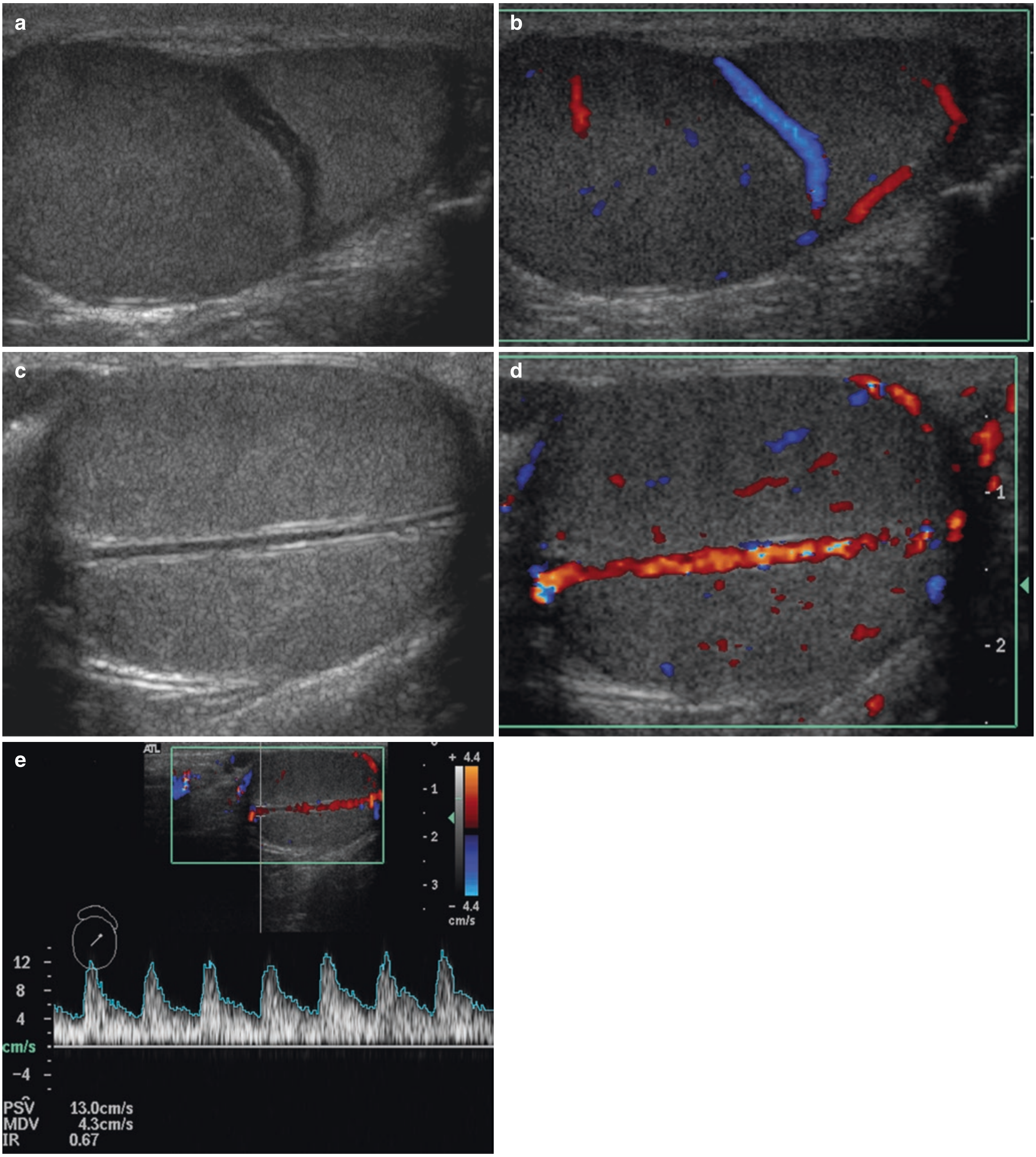


Fig. 1.17 Transmediastinal arteries. Hypoechoic bands of transmediastinal arteries (**a**, **c**) and their aspect on colour Doppler study (**b**, **d**, **e**), which shows low-resistance waveform on colour Doppler evaluation

upper half of the testis. The **transmediastinal veins** accompanying these testicular arteries are more difficult to see (Fig. 1.18) [1, 3, 4, 11]. The testicular veins emerge from the back of the testis and form the loose **pampiniform plexus**. Spectral Doppler analysis of the spermatic cord arteries and intratesticular branches is useful in various conditions. There are a growing number of studies that attempted to evaluate resistance index (RI = PSV − EDV/PSV) of intratesticular branches. This is more easily performed in the subcapsular arteries. The majority of studies found an increased RI, pulsatility index and PSV in intratesticular branches of patients with varicocele, compared to normal subjects. These authors claimed that the increased venous pressure caused by varicocele leads to an increased capsular artery pressure, which is compensated by arterial vasoconstriction, resulting in imparted testicular microcirculation [12]. However, these data are only preliminary, and larger studies are needed to investigate variations of testicular microcirculation in a functional perspective.

The deferential and cremasteric arteries often demonstrate high-resistance waveform—narrow systolic peaks and low or absent diastolic flow. In the presence of paratesticular hyperaemia, however, these spectral features may attenuate and the vessels may acquire the typical low-resistance-type waveforms (broad systolic peak and high diastolic flow) of the testicular artery.

The scrotal wall is supplied by branches of the internal and external pudendal arteries, which also contribute to the blood supply of the spermatic cord, epididymis and, occasionally, the lower pole of the testis.

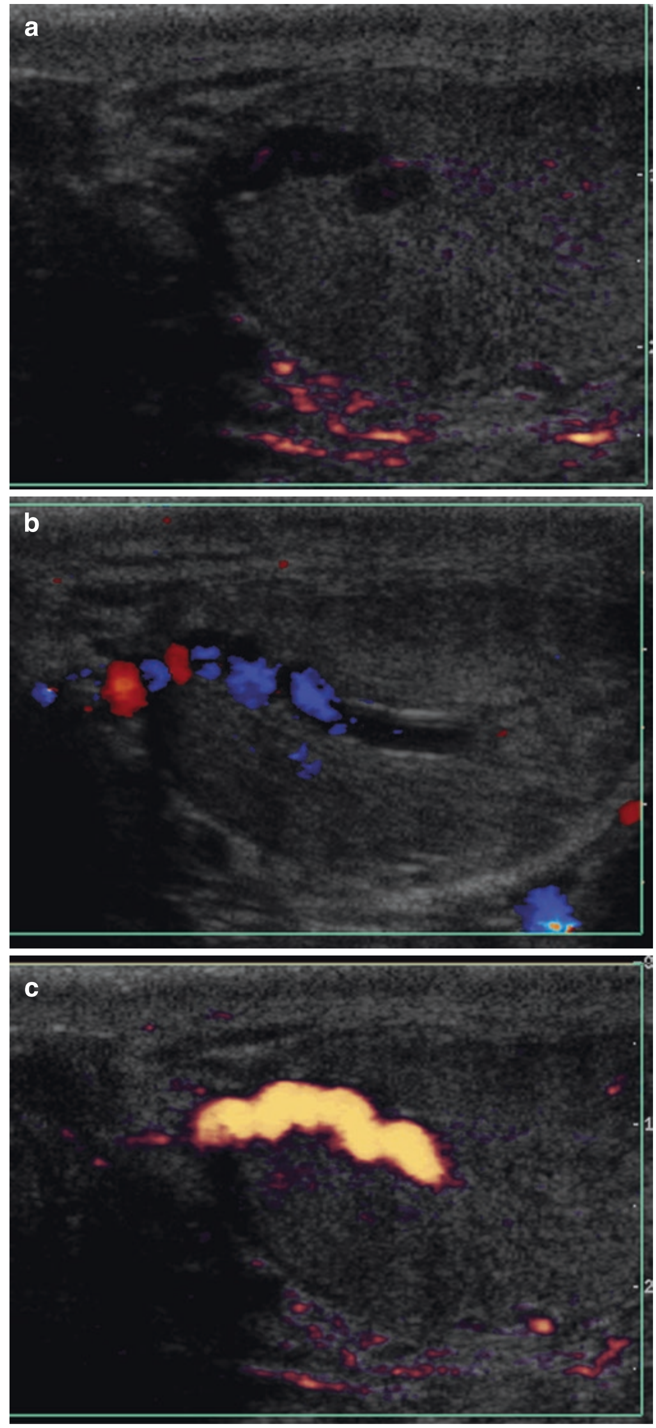


Fig. 1.18 Intratesticular veins. A serpiginous structure (**a**) coursing through the central aspect of the testis with 'tumbling' echoes within. Colour and power Doppler image with the patient performing the Valsalva manoeuvre demonstrating colour flow Doppler (**b**, **c**)

1.7 Appendices

The appendices of the epididymis and testis are embryological remnants that are found towards the upper or the lower pole of the testis or from either the head or the tail of the epididymis.

These structures normally measure only millimetres in length and are best seen by ultrasound when a hydrocele or other scrotal fluid collections are present.

These are isoechoic structures, although calcifications or cysts may develop in the appendices of elderly men (Fig. 1.19). The paradidymis (organ of Giraldes) is composed of some tubules (vasa aberrantia) that persist during embryogenesis without any connection to the testis. It is normally not identified by ultrasound, but sometimes it may swell and distend, forming a cyst-like structure (cyst of Morgagni, Fig. 1.20) that should not be confused with an epididymal cyst [1, 4].

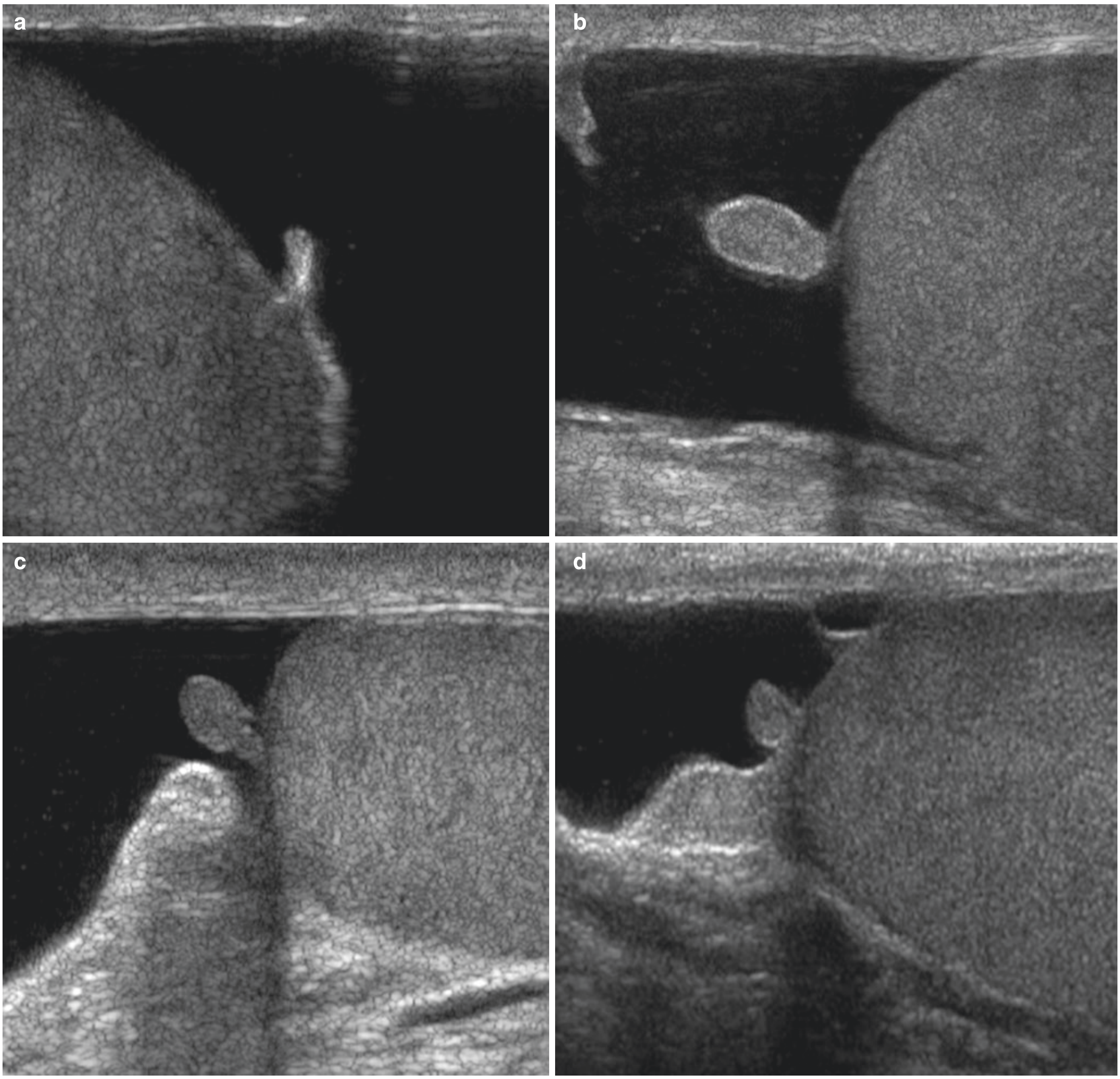


Fig. 1.19 Epididymal and testicular appendages. Hydrocele showing appendix of the testis (**a**, **b**, **c**) and of the epididymis (**d**, **e**). Appendages can calcify (**f–h**)

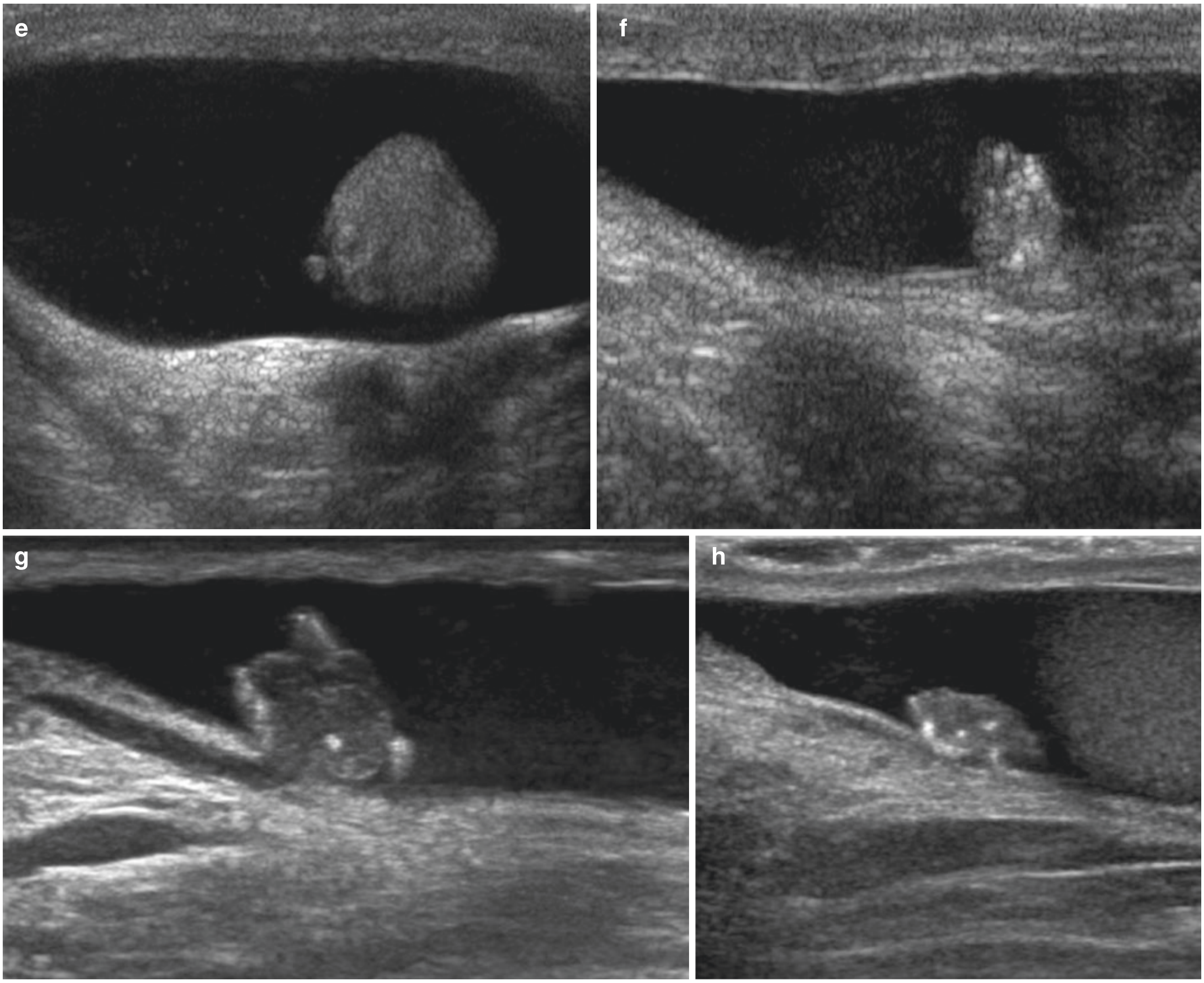


Fig. 1.19 (continued)

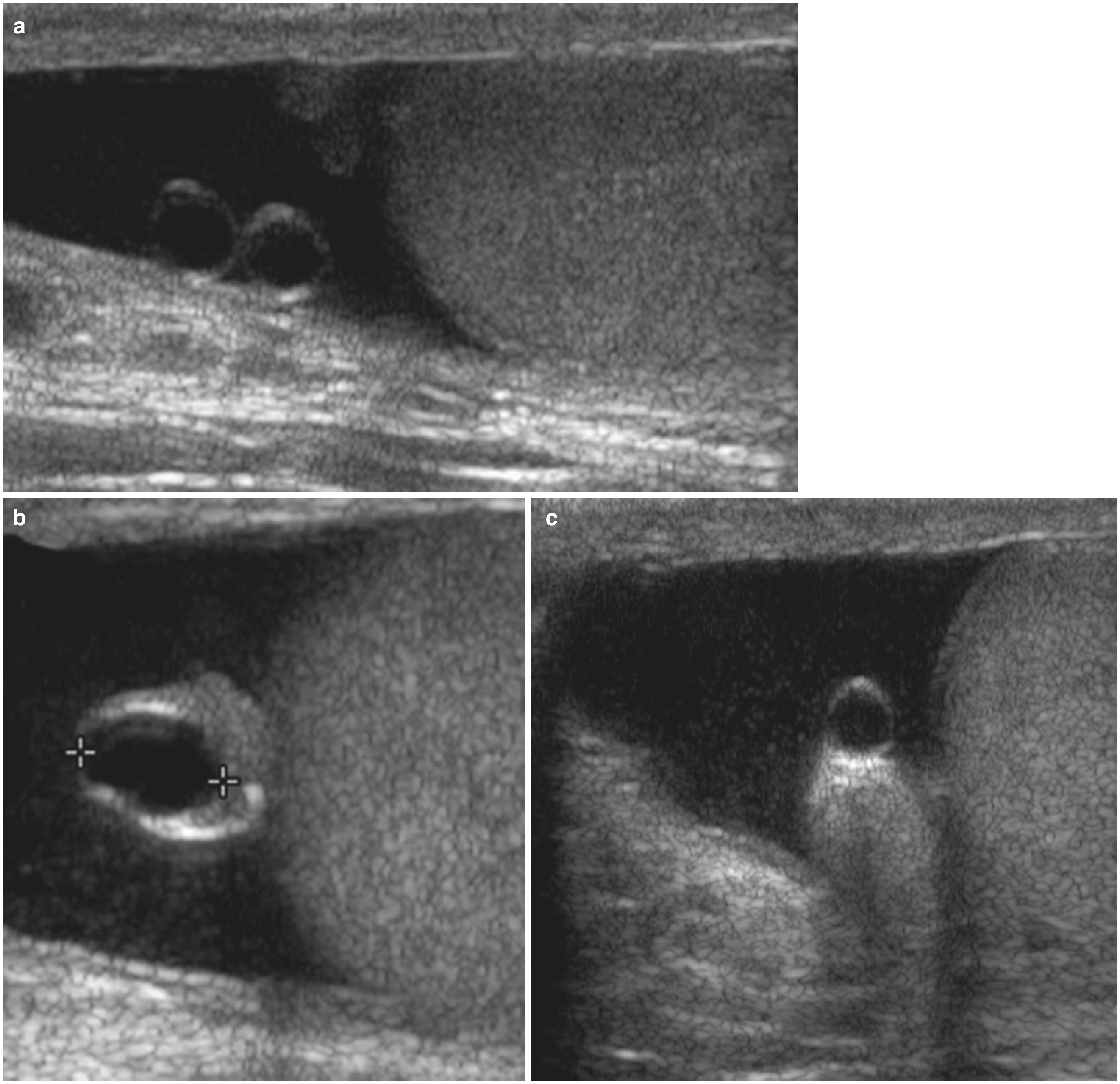
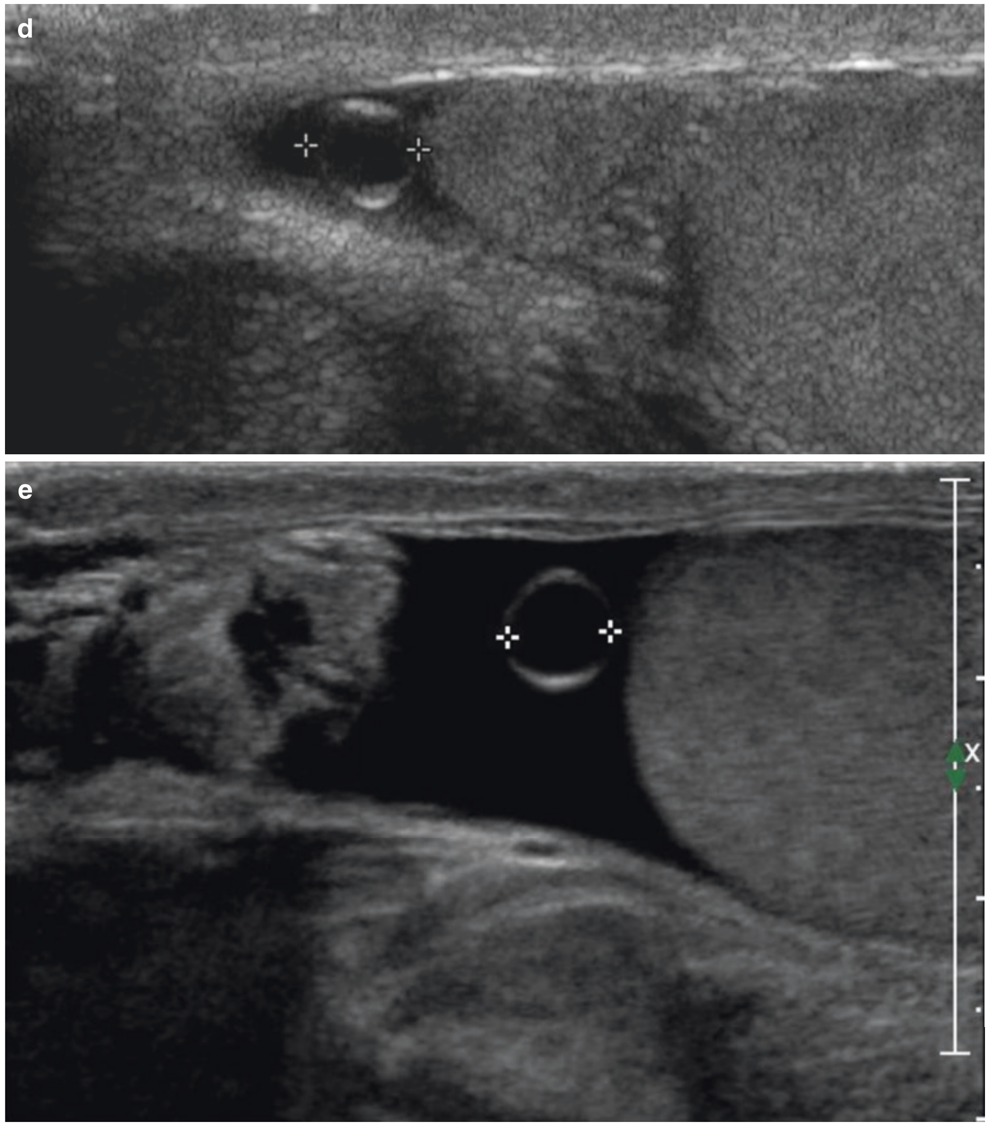


Fig. 1.20 Cyst of Morgagni. Examples of some cysts of Morgagni (**a–e**), which should not be confused with epididymal cysts

Fig. 1.20 (continued)



1.8 Examination Technique

It is good practice to palpate the scrotum before scanning in order to acquire a general impression of the anatomic situation, evaluate the consistency of the testes, appraise the engorgement of the pampiniform plexus while the patient performs a Valsalva manoeuvre and identify any small superficial abnormalities not immediately seen on a sonogram.

The ultrasound examination of the testis should begin with the patient in the supine position. The scrotum should be placed on a towel between the thighs. This firmly supports the scrotum during examination. The patient should hold the penis out of the scanning field, and a towel should be placed over it before applying a generous amount of gel to the scrotum, to avoid artefacts due to scrotal hair.

The scrotum and its contents should be examined using a high-frequency 7–12 MHz transducer with a short focal zone. A large transducer (5.0 cm length) is recommended in order to display the entire testis in a single image. This helps in diameter measurements and corrects volume estimation.

Each testis and epididymis should be examined separately and the anatomy recorded on longitudinal and transverse scans. Images should be acquired in both greyscale and colour Doppler modes.

The normal testis has medium-level echoes. The volume and echogenicity of the two testes should be compared on several transverse images. The asymptomatic testis should be examined first.

If a clinically described mass cannot be visualised, the examiner should manually palpate for the lesion. If it is found, a finger of the non-scanning hand should be placed underneath the lesion while scanning directly over it.

Varicocele should be evaluated with the patient performing a Valsalva manoeuvre both standing and lying down.

Duplex and colour flow Doppler are necessary for a complete evaluation of normal and pathological findings. The superior detection of slow flow velocity is essential to evaluate the small intratesticular and intralesional vessels. The application of colour Doppler is particularly useful in establishing the diagnosis of testicular torsion, scrotal trauma, testicular abscess and varicocele. The evaluation of intratesticular vascularisation, especially of benign and malignant lesions, is recently gaining a major role in the differential diagnosis of small, unsuspected non-palpable lesions [1, 3, 4, 11, 13, 14].

Key Messages

- The echotexture of the adult and prepubertal testis is homogeneous. Apart from the vessels, the rete testis and mediastinum, any non-homogeneity should be interpreted as a pathological finding.
- Size and symmetry of the testes are both important. Any variation greater than 25% between the two testicular volumes should be reported.
- For a correct volume estimate, it is crucial to take the necessary time to scan the maximum diameters.
- Testicular volume calculated by the ellipsoid equation can involve an error of up to 50% for volumes below 4 mL; a modified formula should be used.
- The most accurate formula for calculating testicular volume is $L \times T \times AP \times 0.71$ (rather than $\times 0.52$), especially for small testicles.
- Hypoechogenicity of the immature testis is a normal finding.
- Echogenicity of the testes increases during puberty due to growth of seminiferous tubules and the opening of their lumen. Evaluation of testis reflectivity could provide potentially useful information.
- The testis should be clearly differentiated from the paratesticular structures (including the epididymis).
- All segments of the epididymis can normally be observed. Any enlargement or changes in reflectivity might be pathological and should be reported. Most anatomic structures (e.g. tunica albuginea, ductus deferens, vessels, small amount of liquid in the cavum serosum testis) can be recognised by US, and failure to identify the normal structures must be interpreted on the basis of clinical history.
- Intratesticular blood flow should be evaluated qualitatively for homogeneous and regular distribution. Extratesticular blood flow should be estimated with Doppler sonography to identify high- and low-resistance arteries in the spermatic cord.
- The gel and examination room temperature are important, especially in paediatric patients.
- Thickening of the scrotal layers, the mediastinum and the vessels can cause acoustic shadowing and artefacts simulating a non-homogeneous appearance of the testis.

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2 Non-neoplastic Intratesticular Lesions

2.1 Introduction

Ideally, scrotal ultrasound should distinguish between tumorous and non-tumorous lesions and – in the former category – between benign and malignant tumours. Most testicular tumours present as a palpable mass, but not all palpable masses are malignant tumours, nor are all testicular lesions palpable.

It is now increasingly frequent to find unsuspected non-palpable intratesticular lesions at routine scans for infertility. Up to 10% of these lesions are tumours, half of which prove to be malignant. The uncertainty associated with these findings has often led to inappropriate treatment, e.g. unnecessary orchiectomy. However, greater knowledge and improved diagnostic capabilities are now supporting the clinical management of unsuspected testicular lesions. To provide physicians with an accurate 'non-invasive' diagnosis, ultrasound findings should be merged with clinical and laboratory data, in a rigorous diagnostic process that must adhere to a precise protocol to avoid subjective interpretation.

The first objective in a scrotal ultrasound is to distinguish intratesticular from extratesticular masses. Extratesticular masses are usually obviously benign cystic lesions and, even when solid, are so rarely malignant that a conservative approach is usually adopted.

In the presence of an intratesticular mass, the second objective of the scan is to try to differentiate benign from malignant lesions. About 10 years ago, the school of thought was that it was impossible to distinguish the latter from the few benign masses, at least with any accuracy. Virtually all intratesticular masses were therefore treated by radical orchiectomy.

This approach can no longer be accepted. It is now clear that clinical and radiological findings need to be merged on the basis of predefined criteria, in order to improve the diagnostic process and categorisation accuracy. The first step in describing an intratesticular lesion is to define it as tumorous (proliferating) or non-tumorous (non-proliferating), and the second is to judge it as 'probably benign', 'undetermined' or 'probably malignant' (see below for a selection of possible discriminating criteria). Bear in mind that about 5–15% of all intrascrotal solid lesions are benign tumours.

Even though some of these lesions are very small, categorisation should be attempted anyway. Active surveillance – especially in such cases – can avoid unnecessary surgery or support a testis-sparing approach instead of a radical orchiectomy. We suggest repeated follow-up scans during which the adherence to predefined benignity or malignancy criteria should be checked, possibly in a 'blinded fashion', and compared to the findings of the previous examination. Repeated scanning would allow obtaining the necessary clinical data to interpret functionally the US findings. Such approach requires a deep knowledge of the histopathological pictures associated with certain conditions (i.e. genetic disorders, cryptorchidism, surgical manipulation, hormonal abnormalities).

This chapter covers the most common as well as some extremely rare non-tumorous intratesticular lesions. The differential diagnosis between these lesions and malignant tumours is discussed. The ultrasound features of 'malignancy' and 'benignity' of parenchymal lesions are also presented, while a complete description of benign tumours is given in the next chapter.

2.2 Cysts

Intratesticular cysts – once regarded as rare – are now discovered incidentally in 8–10% of ultrasound scans [1]. As most cysts are asymptomatic, the importance of recognising simple cysts lies in distinguishing them from testicular neoplasms, which may also contain cystic spaces. Simple cysts are seen as sharply demarcated anechoic areas with thin, smooth walls with an acoustically enhanced posterior wall, no solid portions and no structural disturbance of the adjacent testicular parenchyma (Fig. 2.1). The differential diagnosis for scrotal cystic lesions includes intratesticular cysts, epididymal cysts, epidermoid cysts and cystic dysplasia and more importantly germ cell tumours, especially teratomas [2].

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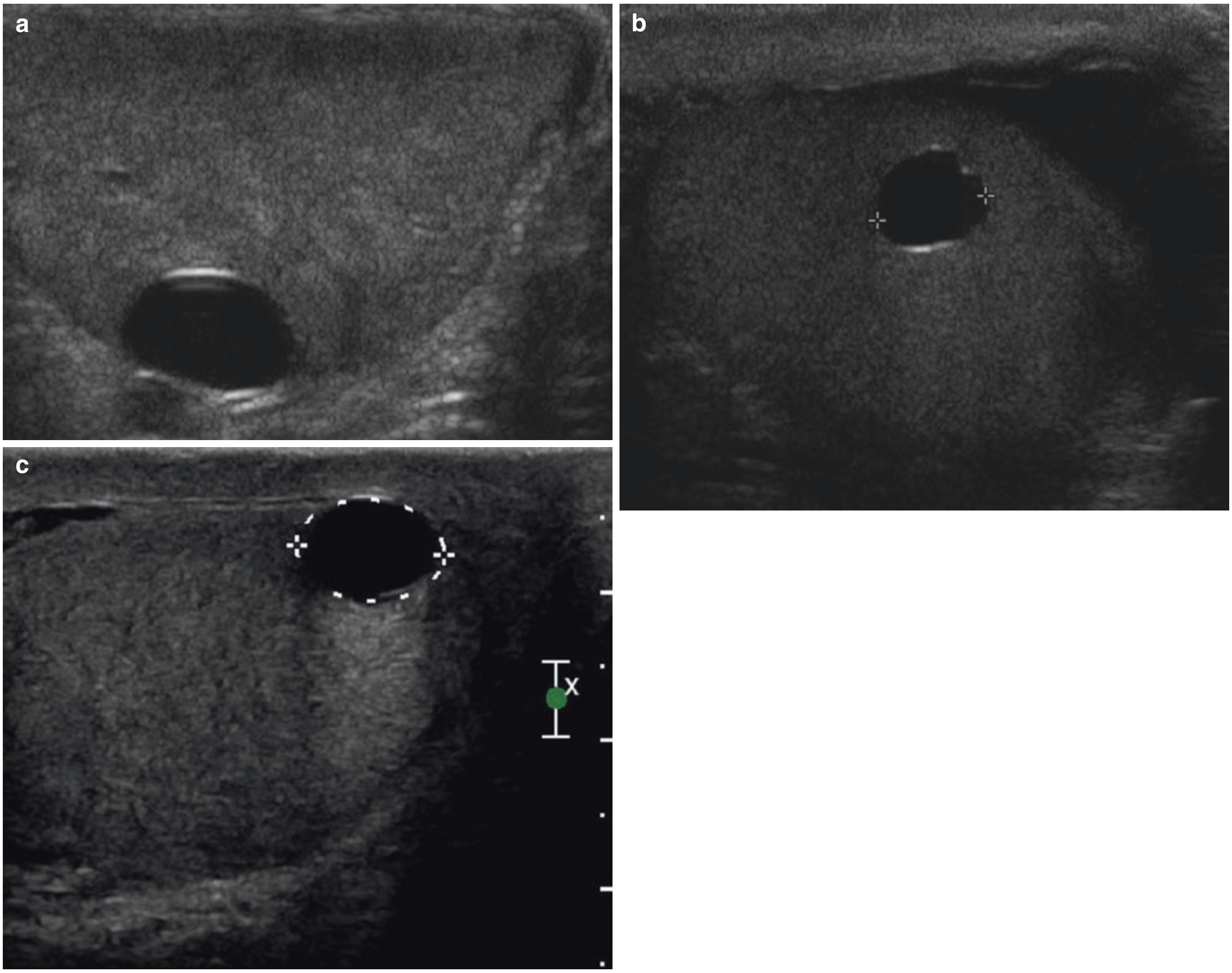


Fig. 2.1 Intratesticular cysts. Isolated cysts within the testis. They fulfil the criteria of any simple cyst: smooth wall, no solid elements, anechoic centre (**a**, **b**) and enhanced through transmission (**c**)

2.2.1 Intratesticular Cysts

Intratesticular cysts are detected incidentally during US. It is postulated that cysts result from an anomalous efferent duct, obstruction of the spermatic ductal system or postinflammatory cystic dilation of individual ducts [3]. The origin is unclear (congenital or acquired infection, trauma or surgery). The cysts are usually solitary but can be multiple (Fig. 2.2). They can occur anywhere in the testis but are often near the mediastinum testis, in the upper half of the didymus. Simple testicular cysts are usually not palpable, and even when large, they are not firm. This is a distinctive feature of benign intratesticular cysts in comparison with cystic tumours, which are generally palpable as firm or hard masses [4]. Cysts of the tunica albuginea can bulge either into the testis or out into the peritesticular space. Even when small, albuginea cysts can often be palpated – in contrast with intratesticular cysts – as a firm mass generally of pinhead size.

Simple cysts vary in size from 2 mm to 2 cm. On US, they have a pathognomonic echo pattern showing a well-defined round anechoic lesion. High magnification (Fig. 2.3) reveals a more echogenic rim around the cyst, caused by compression of the testicular parenchyma and distal acoustic shadowing [5]. It is important to distinguish between a benign cyst and a cystic neoplasm, as cystic testicular lesions are not always benign (testicular tumours may undergo cystic degeneration due to haemorrhage or necrosis). It is therefore crucial to study the entire cyst wall carefully: benign cysts have no solid element attached to the wall. In this case, and in the presence of a homogeneously anechoic inner fluid, the cyst is almost invariably benign: the patient can be reassured and requires no treatment or further follow-up. Occasionally, cysts and solid masses are found within one testis (Fig. 2.4a). The figure shows the different echotexture of the same size cystic and solid lesions. The coexistence of adjacent cystic lesions does not affect the management of the solid one. Very

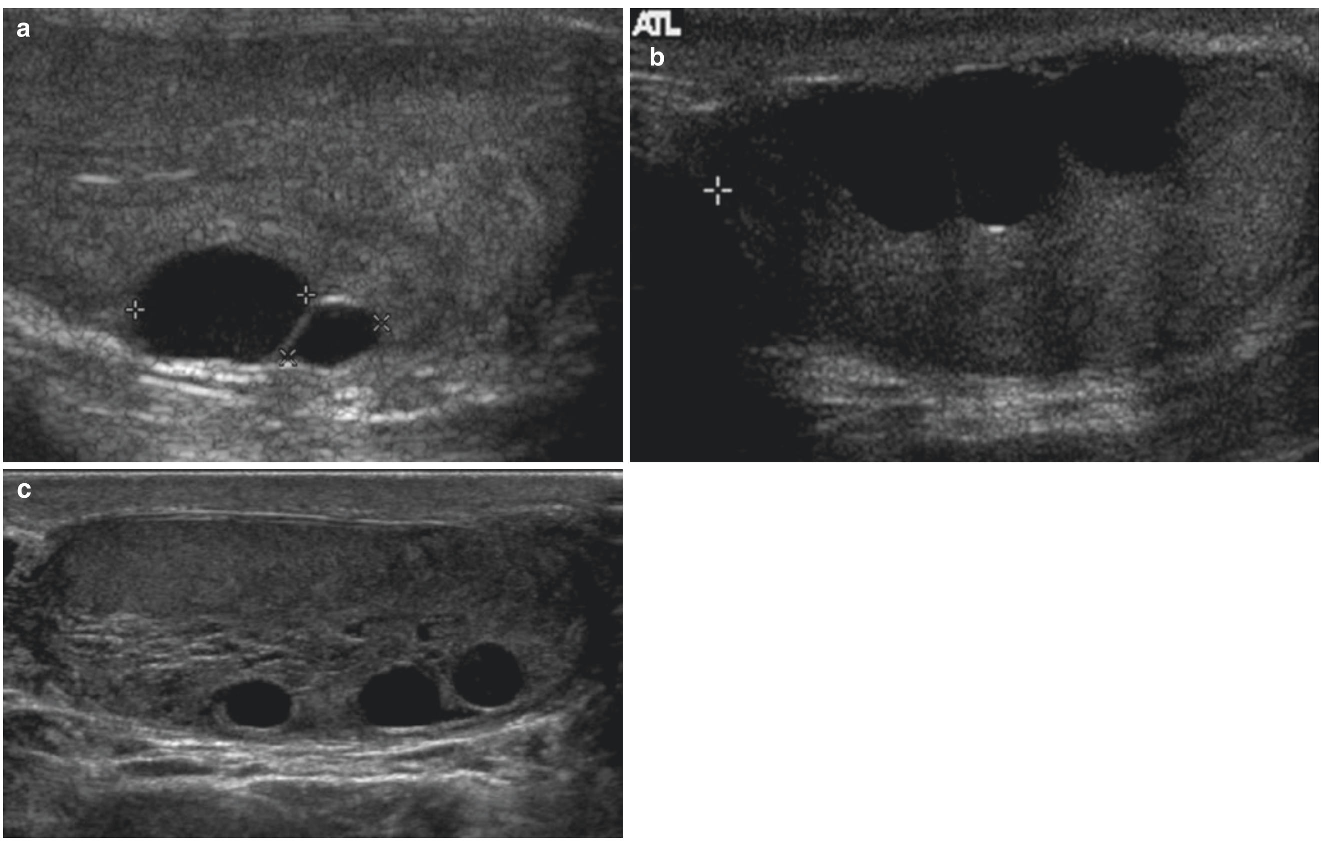


Fig. 2.2 Intratesticular cysts. Longitudinal scan shows multiple intratesticular cysts (**a**–**c**)

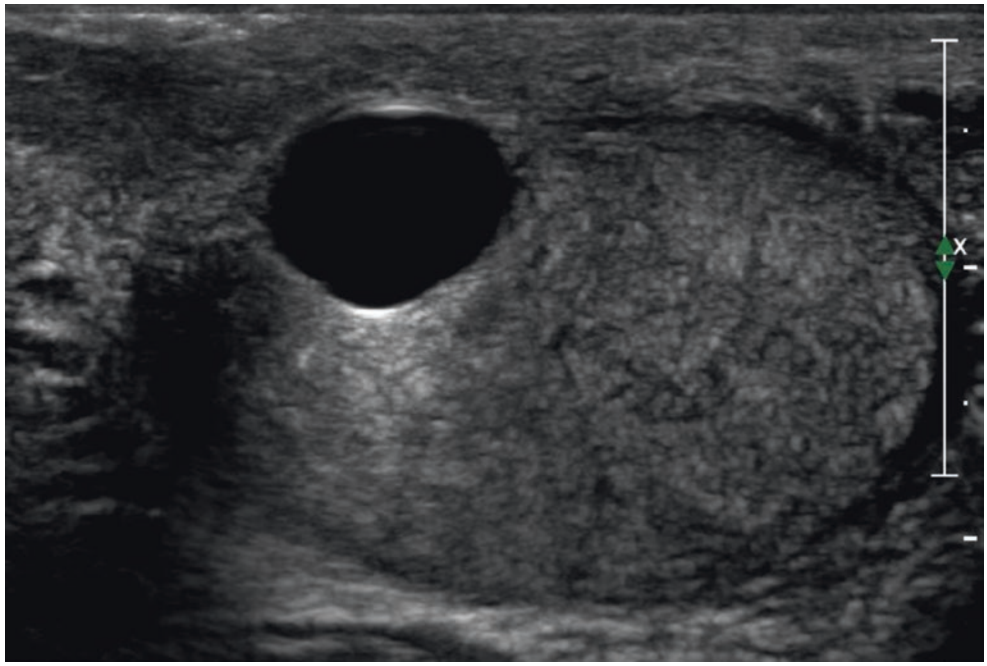


Fig. 2.3 Intratesticular cysts. High magnification of an intratesticular cyst (17-5 mHz linear transducer). Acoustic enhancement is clearly seen

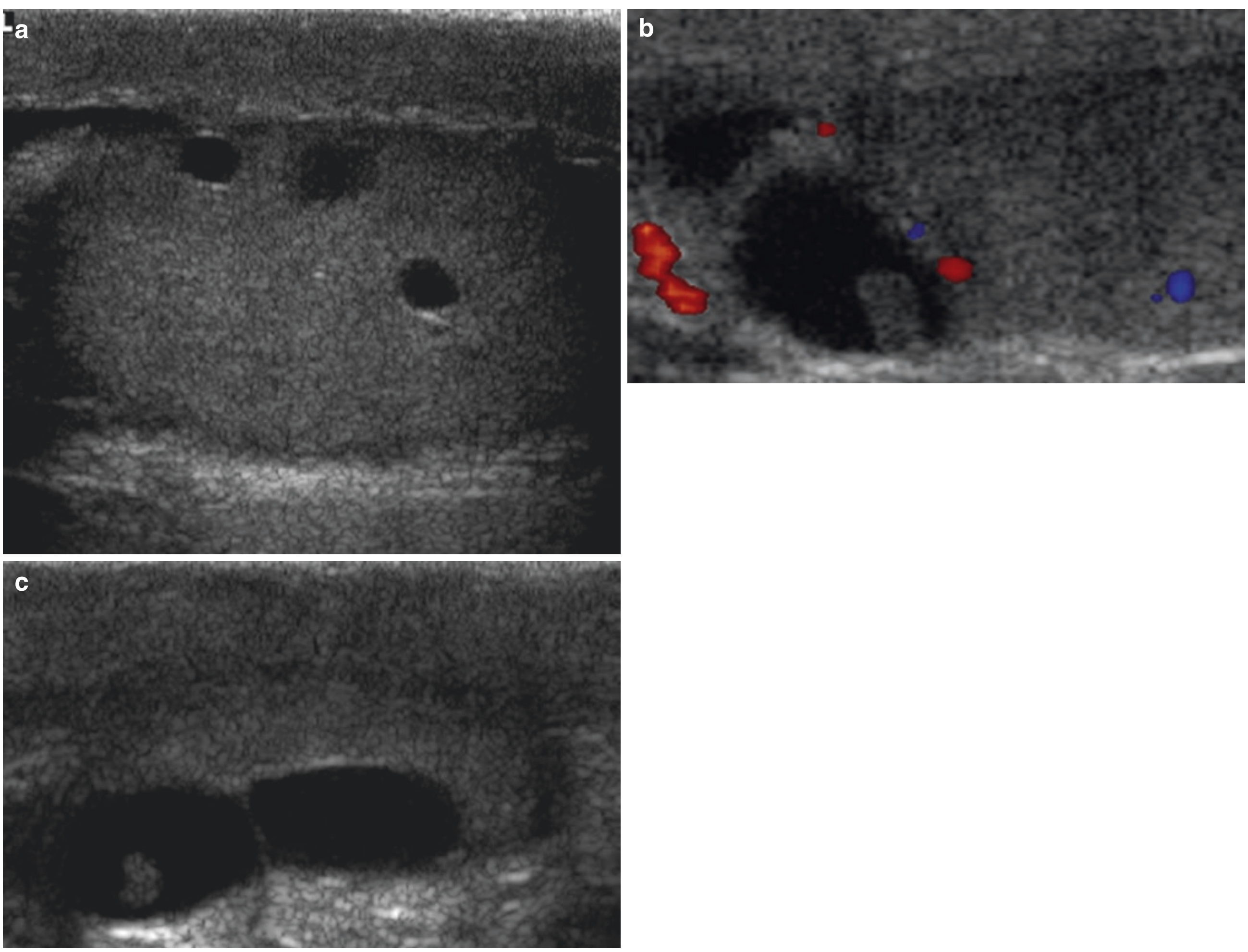


Fig. 2.4 Intratesticular cysts. Longitudinal scan shows the different echogenicity of three intratesticular lesions: a hypoechoic solid mass in the middle, with irregular margins resembling a tumour, and two anechoic benign simple cysts on the side (**a**); a solid avascular papilla detected within an intratesticular cyst (**b**, **c**). The lesion was followed up showing no changes in size or ultrasound appearance. Such appearance has been occasionally found in benign teratomas

rarely, a solid element can be detected even in benign cysts. They should be studied carefully with close follow-up studies (Fig. 2.4b).

In malignant cysts, a clearly detectable solid part is generally visible. Teratomas are tumours with a possibly predominant cystic component. Embryonic cell carcinomas may contain cystic areas, but the mass is predominantly solid. Cystic areas are extremely rare in seminomas or lymphomas. In the differential diagnosis, haematoma and abscess should also be considered. Rarely debris within a benign cyst can simulate a solid component. In this case colour Doppler study (Fig. 2.5) and manipulation (180° rotation) can be helpful.

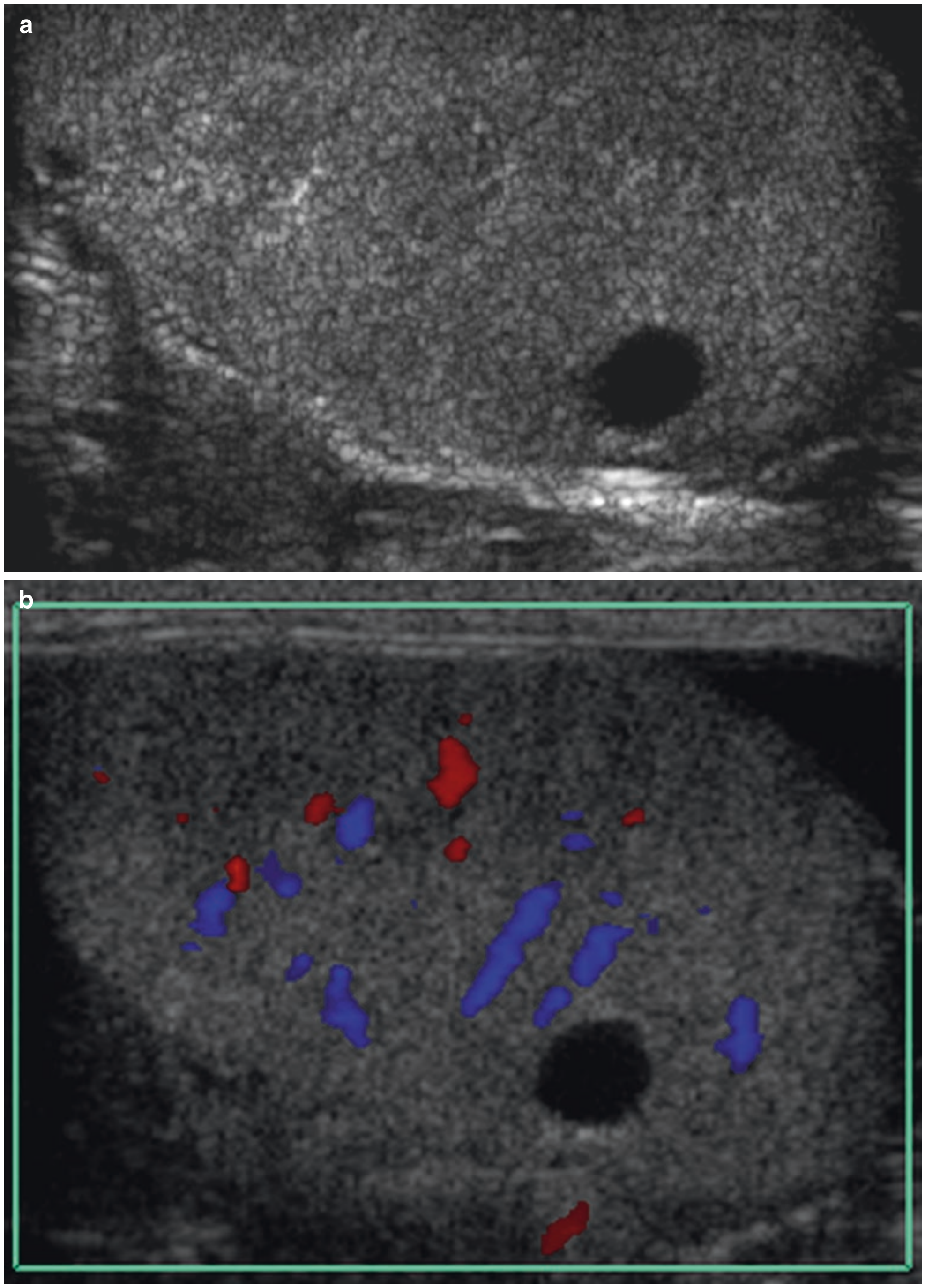


Fig. 2.5 Intratesticular cysts. Use of colour Doppler in the study of an intratesticular cyst. Longitudinal scan shows an anechoic mass that is avascular on Doppler examination (**a**, **b**)

2.2.2 Tunica Albuginea Cyst

The aetiology of **cysts (or granulomas) of the tunica albuginea** is unknown, but they are believed to be mesothelial in origin. Tunica albuginea cysts may be single or multiple and their size varies from 2 to 30 mm (Fig. 2.6). The mean age at presentation is 40 years, and they are also seen in patients in their 50s and 60s [6]. They can be unilocular or multilocular. They have a fairly characteristic feel on palpation as a firm mass, usually of pinhead size, on the surface of the testis. They cannot be distinguished from small subcapsular tumours by palpation alone: in fact, there is a worry that one is feeling the edge of a larger intratesticular tumour. Patients may be asymptomatic or present with pain and swelling. Such cysts must therefore be examined by ultrasound.

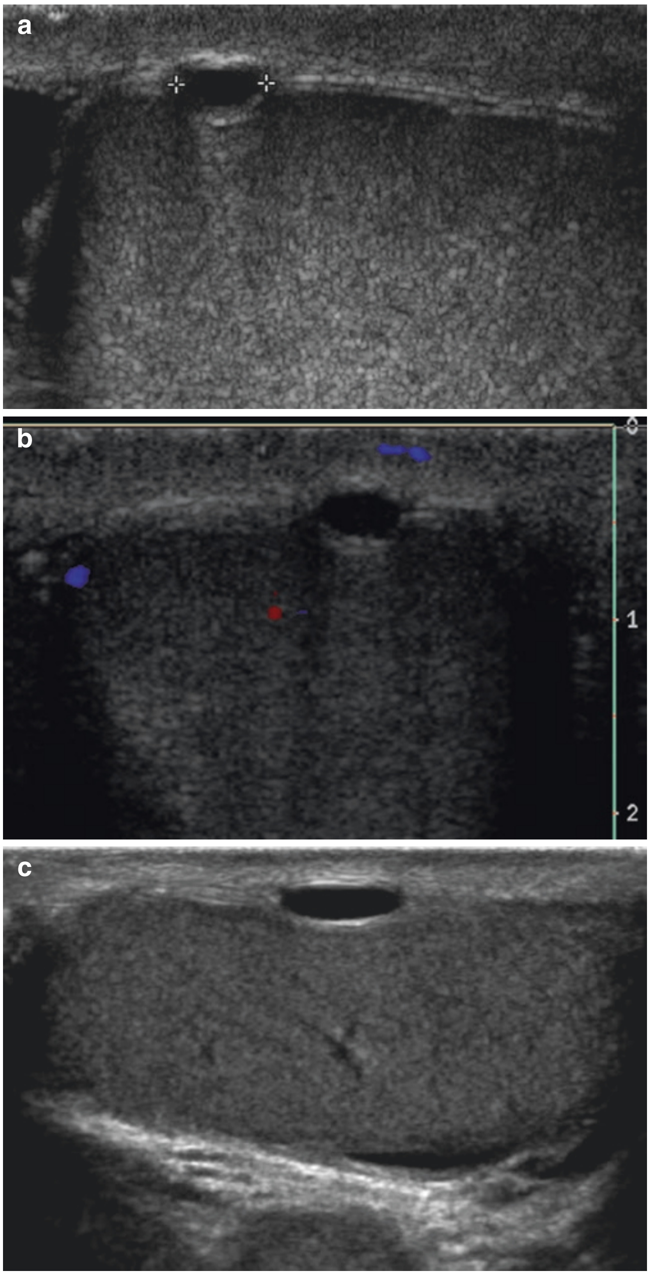


Fig. 2.6 Tunica albuginea cyst. Typically small tunica albuginea cyst. Longitudinal scan: tiny cyst between the layers of the tunica albuginea (**a–c**)

It is often easier to palpate than to locate these cysts by US; therefore it is suggested that the testis is scanned while using a palpating finger to indicate the position of the lesion. They sometimes calcify to leave nothing except a palpable calcification, which casts an acoustic shadow (Fig. 2.7).

It is sometimes difficult to differentiate a tunica albuginea cyst from an intratesticular simple cyst or a tunica vaginalis cyst. On US, tunica albuginea cysts meet all the criteria of a simple cyst and may occasionally have internal echoes or septations. They are usually located along the anterior superior or lateral surface of the testis [7]. Their typical location and ultrasound appearance enable them to be distinguished from intratesticular or paratesticular neoplasms.

The term **granulomatous periorchitis** has been used to describe the presence of a significant number of cysts (more than five per testis). These cysts carry no pathological significance and require no treatment or follow-up. Isolated small cysts are normally incidental findings [8]. Granulomatous periorchitis may be associated with previous or chronic inflammation, hydrocele or autoimmune disorders. In infertile patients, the evaluation of sperm leucocyte antisperm antibodies is recommended.

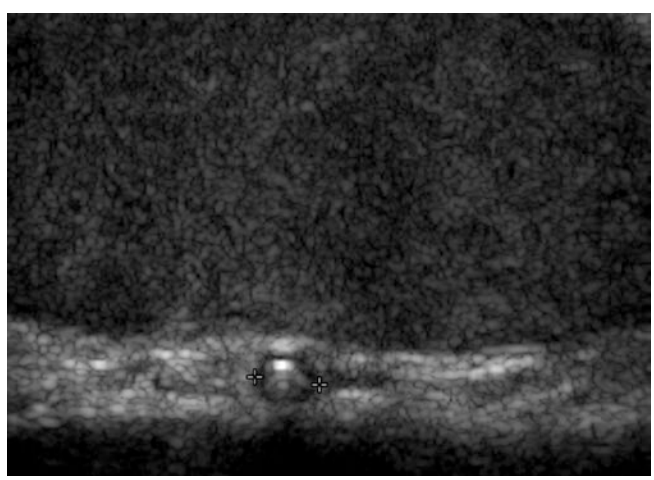


Fig. 2.7 Tunica albuginea cyst. A calcified and palpable tunica albuginea cyst, which casts an acoustic shadow

2.2.3 Tunica Vaginalis Cyst

Cysts of the tunica vaginalis are rare and may arise from the visceral or parietal layer of the tunica vaginalis (Figs. 2.8 and 2.9). Like cysts of the tunica albuginea, they may be single or multiple. Sonographically, they are usually simple anechoic structures (Fig. 2.10), but they occasionally have internal structures or septations [7].

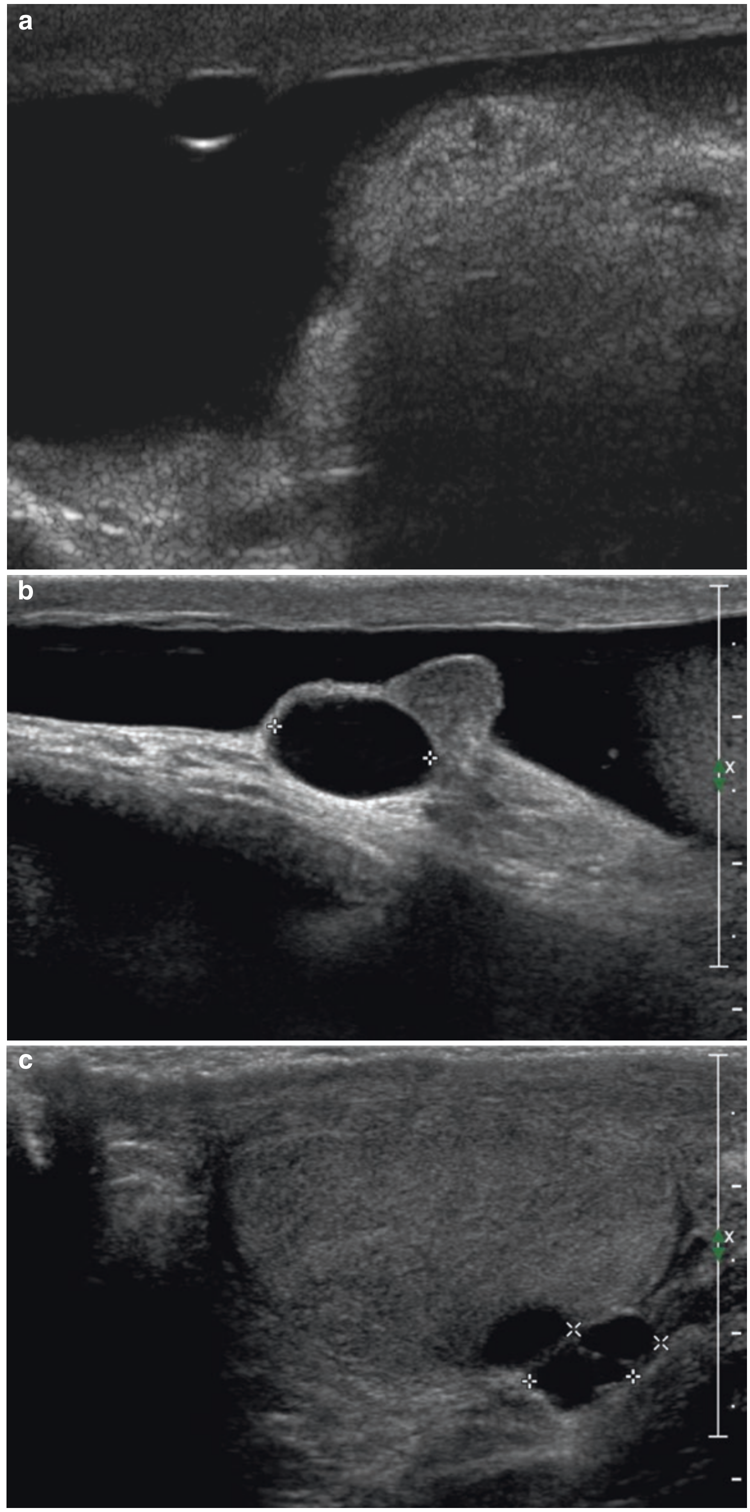


Fig. 2.8 Tunica vaginalis cyst. Longitudinal scan shows two cysts arising from the parietal layer of the tunica vaginalis (**a**, **b**) and an intratesticular cyst and two cysts of the tunica vaginalis (**c**)

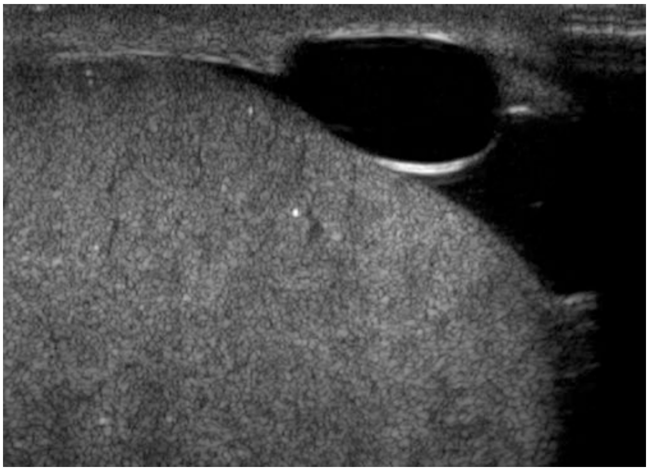


Fig. 2.9 Tunica vaginalis cyst. A large cyst arising from the visceral layer of the tunica vaginalis. This type of cyst is usually palpable

Haemorrhage into tunica vaginalis cysts and torsion have been described and may be a rare cause of acute scrotal pain. An exceedingly rare tumour, the benign cystadenoma of the vaginalis, exhibits clusters of various sizes of cysts, normally arising from the vaginal layer that can resemble varicocele in the greyscale mode (Fig. 2.11).

2.2.4 Dilation of Rete Testis

Benign cystic lesions of the rete testis can be categorised as cystic transformation (ectasia), cystic dysplasia, cystadenoma and simple cyst. These lesions are part of a wider spectrum of cystic lesions of the testis and epididymis and should be included in the differential diagnosis.

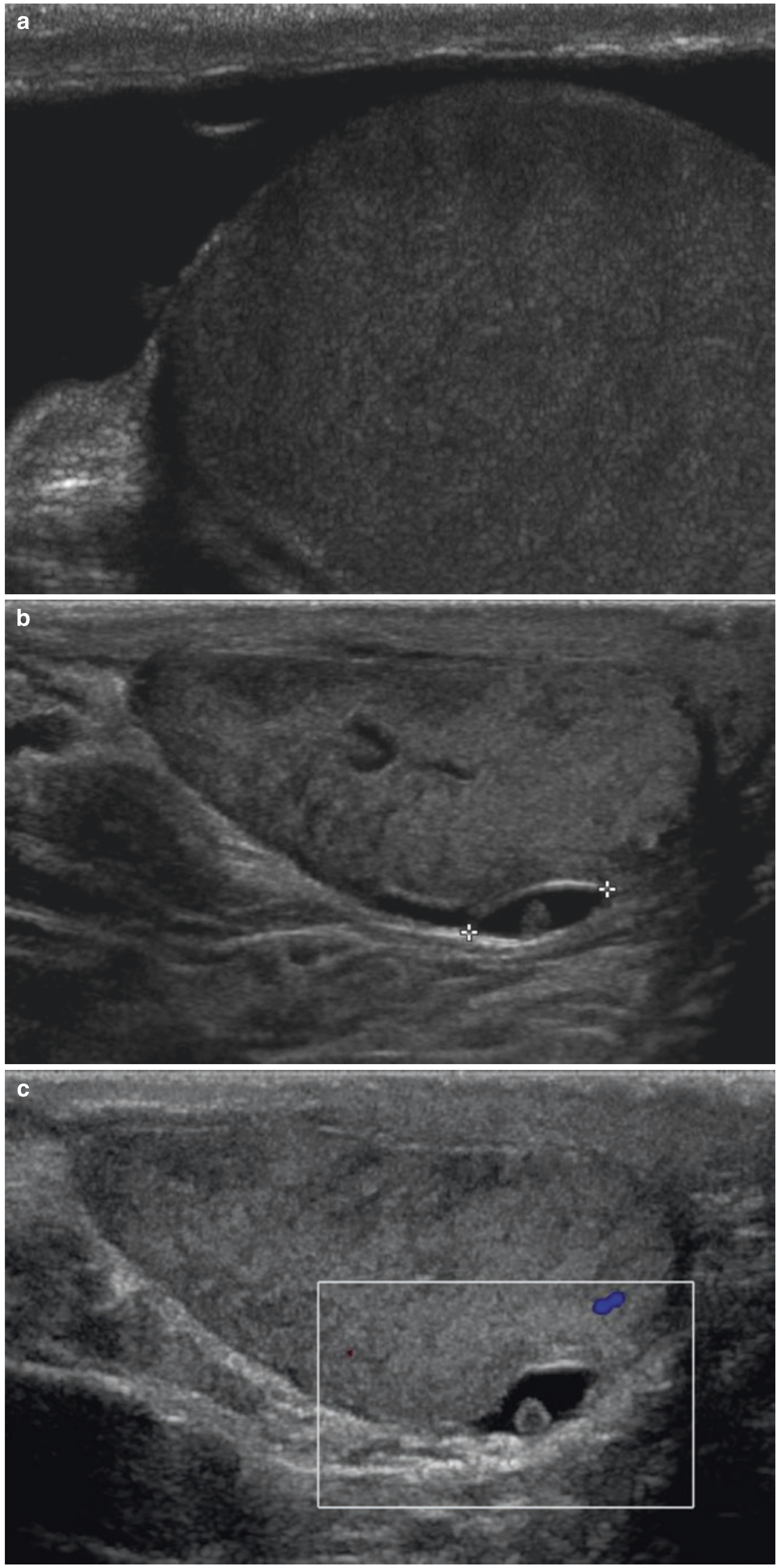


Fig. 2.10 Tunica vaginalis cyst. Longitudinal US scan shows a well-defined cyst with an imperceptible wall and an anechoic centre (**a**); in panels (**b**, **c**), a tunica vaginalis cyst is shown with a solid avascular papilla in a patient with previous cryptorchidism and late orchidopexy

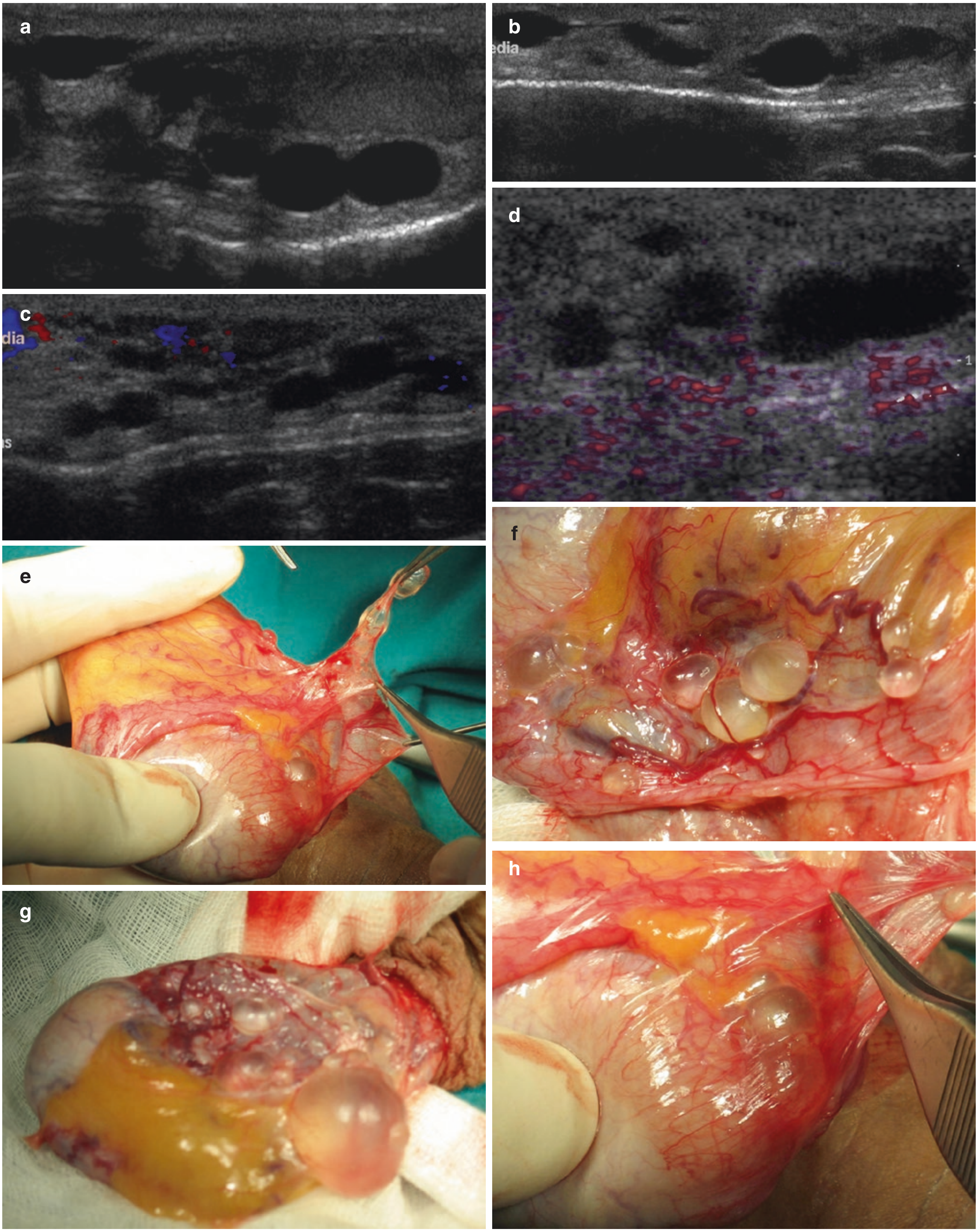


Fig. 2.11 Cystic adenoma of the tunica vaginalis. Multiple cysts arising from a cystic adenoma of the vaginalis (**a**, **b**). The avascular nature of these hypo-anechoic oval structures has to be confirmed by colour Doppler examination (**c**, **d**). More than 15 cysts were found in clusters on the visceral layer of the tunica vaginalis (**e–i**)

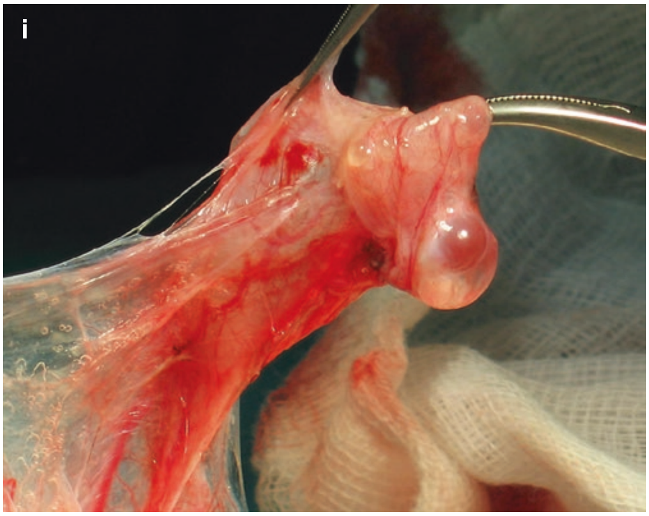


Fig. 2.11 (continued)

2.2.4.1 Cystic Ectasia of the Rete Testis

Cystic ectasia of the rete testis is a benign condition resulting from partial or complete obliteration of the efferent ducts, which causes dilation of the rete testis and should not be erroneously interpreted as a tumour. It is more likely to be an acquired lesion than a congenital problem and has a number of possible causes, including cryptorchidism, mechanical compression of the epididymis or spermatic cord by surgical, neoplastic or infectious processes and ischaemic or hormonally induced atrophic alterations to the epididymal tubules [9]. In some cases there is no apparent cause. It is bilateral in approximately 45% of cases and associated with an ipsilateral spermatocele in approximately 74% [3] and most often occurs in older men. In most cases it is associated with epididymal obstruction. On US, ectasia can be identified as multiple micro- or macro-tubular fluid-filled structures that occasionally form multiple cysts in the region of the mediastinum testis (Fig. 2.12). Their typical location – and the presence of epididymal cysts – can help distinguish cystic transformation of the rete testis from cystic malignant testicular tumours, which can occur anywhere in the testicular parenchyma [10]. Rarely, the ducts resemble dilated intratesticular veins (intratesticular varicocele). Colour Doppler is then required to differentiate between cysts and dilated veins (Fig. 2.13).

An association of atypical solid elements with micropapillary structures resembling a dilated rete testis should raise the suspicion of an exceedingly rare tumour: **adenocarcinoma of the rete testis** (Fig. 2.14) [11, 12]. This condition is clinically associated with haematospermia, older age at presentation, involvement of inguinal lymph nodes, cutaneous manifestation and possibly pulmonary metastases [13].

2.2.4.2 Age-Related Dilation of the Rete Testis

In older patients (over 50), mild **tubular ectasia of the rete testis** is a common finding (Fig. 2.15). This is not generally associated with the typical cystic changes observed in obstructed patients and may not be associated with sperm obstruction, although deterioration in sperm quality might be seen [10]. On US, anechoic, multiple tubular lesions in the region near the mediastinum testis are shown, with no evidence of flow at Doppler US interrogation (Fig. 2.16).

2.2.4.3 Cystic Dysplasia of the Testis

Cystic dysplasia of the testis is a very rare congenital disorder, characterised by multiple irregular cystic spaces in the mediastinum of the testis, which may involve the entire gonad (Fig. 2.17). It usually occurs in infants and young children and is often associated with ipsilateral renal agenesis [14] or dysplasia and other genitourinary tract anomalies. Fewer than 40 cases have been reported [15]. This malformation is due to a development defect of the mesonephric duct, which is the cause of the dilation both of the testicular rete testis and of renal agenesis, and is due to lack of connection between the seminiferous tubule and the efferent duct [16]. The typical clinical presentation is a painless, transilluminating scrotal mass in an otherwise healthy boy. Sonographically, it appears as multiple thin-walled cystic lesions extending out from the mediastinum testis into the testicular parenchyma, causing pressure atrophy of the parenchyma. Histologically, anastomosing cystic spaces lined with cuboidal/flat epithelia and separated by fibrous stroma are observed. The size of individual cysts may vary from 0.1 to 1 cm, and the entire lesion may range from microscopic to 7 cm [17, 18]. Although treatment is surgical, a conservative or nonoperative approach is suggested when feasible. On US, the appearance is similar to acquired cystic dilation of the rete testis. No malignant transformation has been reported.

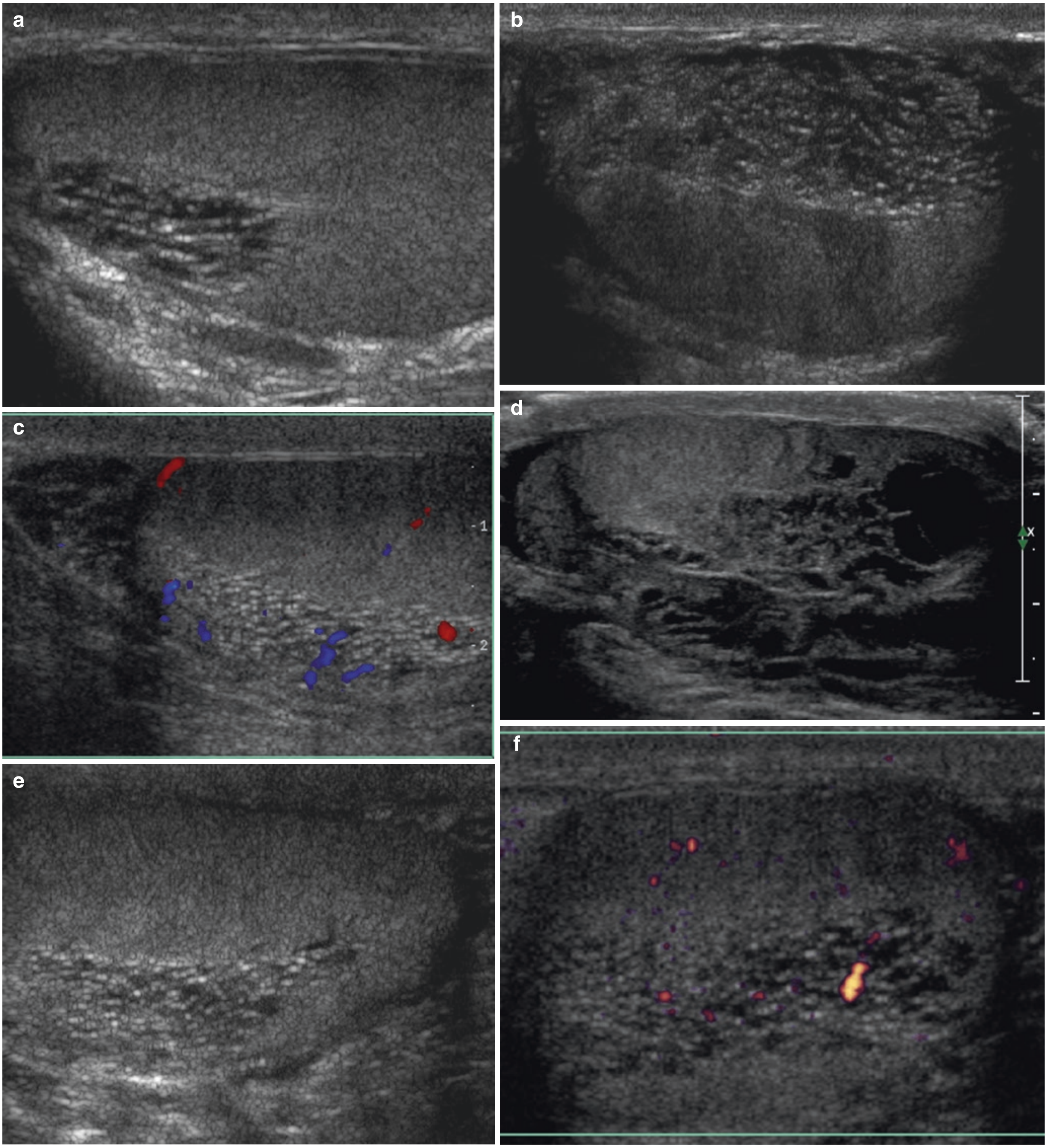
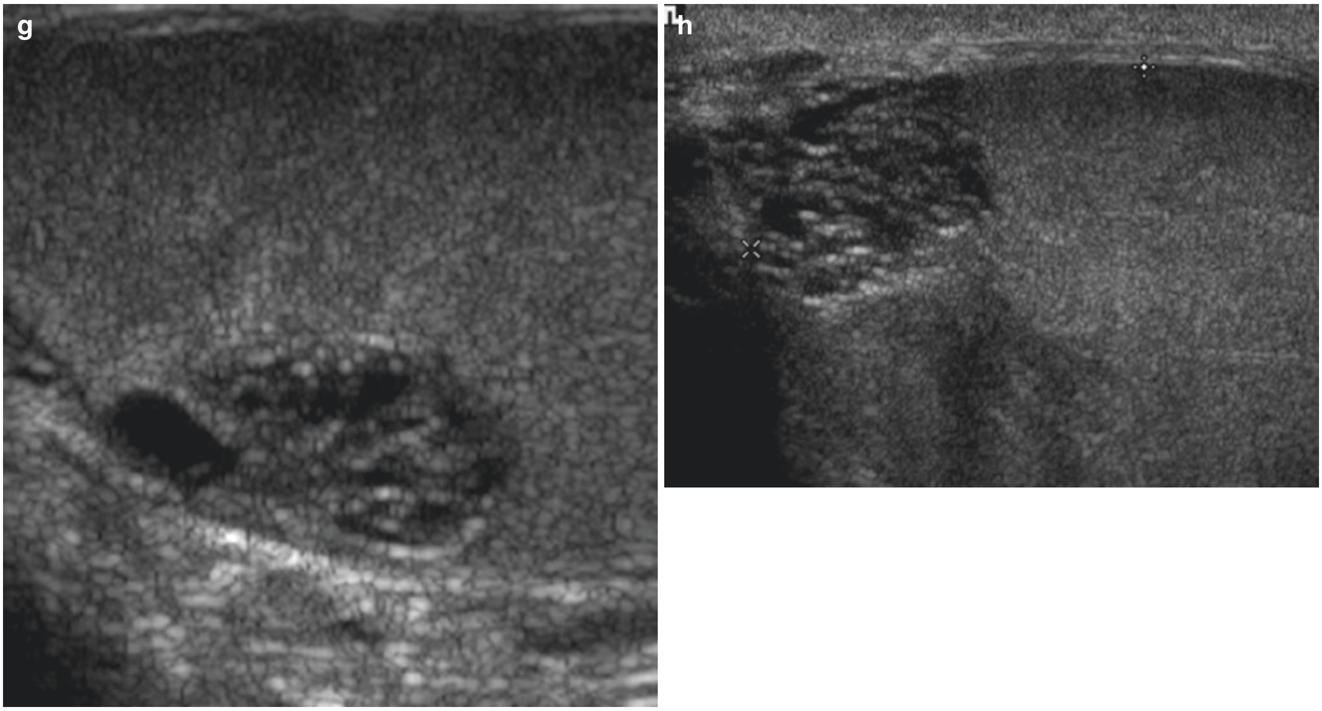


Fig. 2.12 Dilation of the rete testis. Longitudinal scan of the testis demonstrates grapefruit-like cystic dilation of the rete testis (**a–f**). Slight dilation of the efferent ducts in the mediastinum testis (**g**, **h**) should not erroneously be interpreted as a tumour

Fig. 2.13 Dilation of the rete testis. Differences on greyscale and colour Doppler between dilation of the rete testis (**a**, **b**) and intratesticular varicocele (**c**, **d**). Longitudinal scan shows cyst-like lesions in the right testis. During the Valsalva manoeuvre, retrograde flow is demonstrated in the lumen (**d**) in intratesticular varicocele but not in rete testis dilation (**b**)



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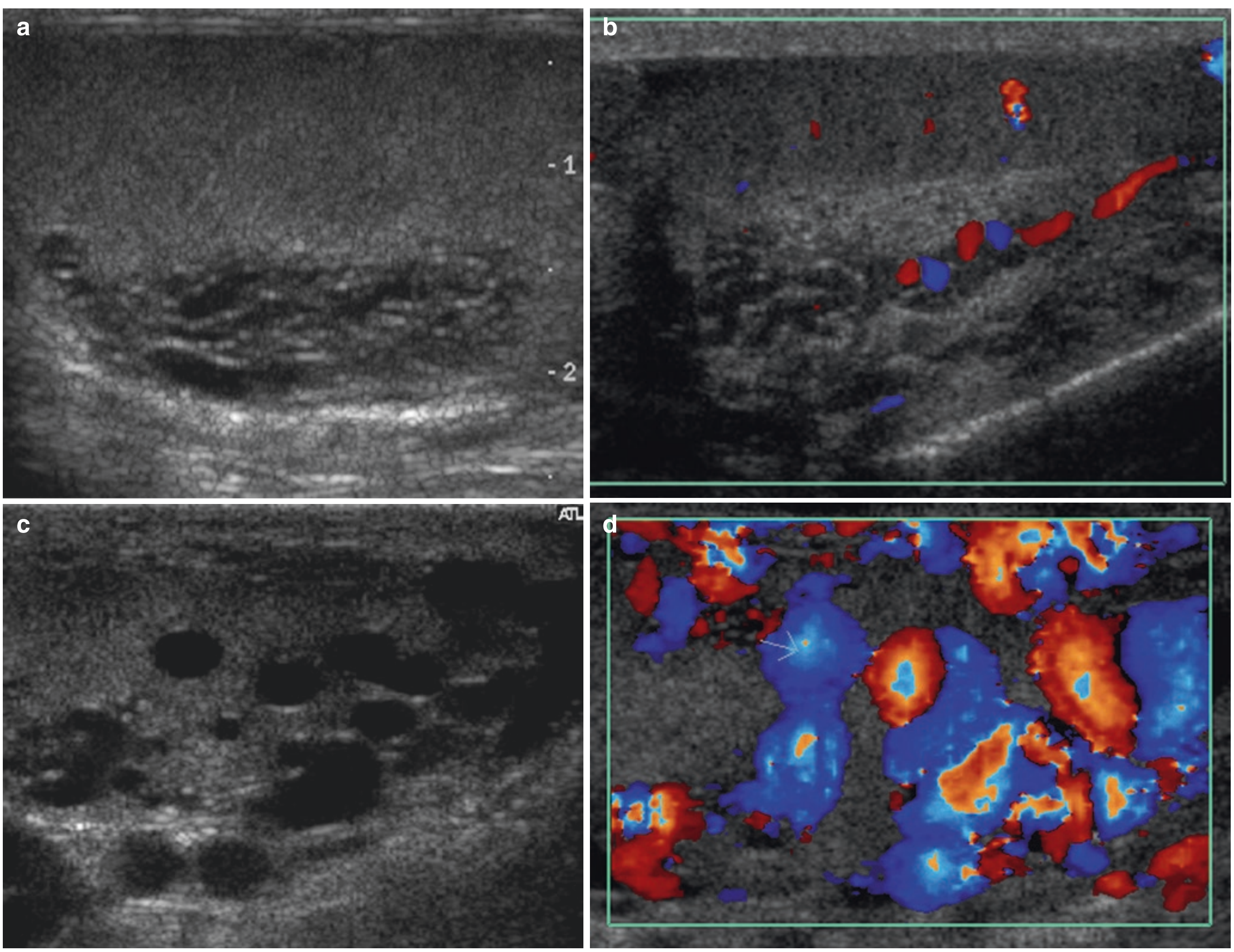


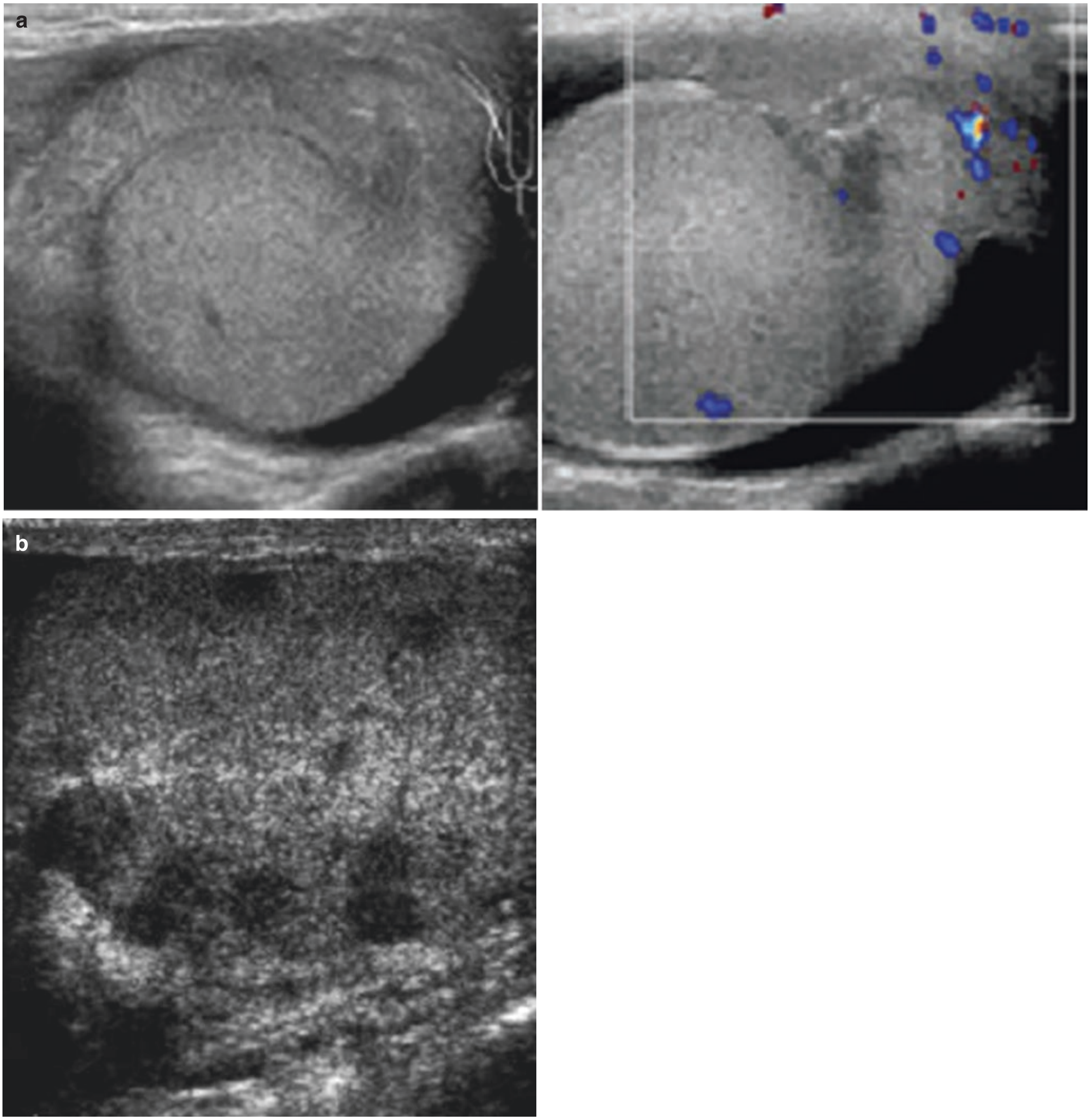


Fig. 2.14 Adenocarcinoma of the rete testis. Sonography revealed a hypoechoic mass at the right epididymis with a poorly defined border. Colour Doppler detected a relatively abundant blood flow in the mass and hydrocele. A diagnosis of poorly differentiated adenocarcinoma of the rete testis was established based on the diagnostic criteria of Nochomovitz and Orenstein (**a**). In image (**b**) ultrasounds of the right testis show multiple lesions within the testis. Orchidectomy revealed a firm tumour. The immunohistochemistry and mucin stains supported the diagnosis of rete testis adenocarcinoma *(Figure* **a** *from: Y. Tian, W. Yao, L. Yang et al, "Primary adenocarcinoma of the rete testis: A case report and review of the literature", Oncology letters (2014) 7: 455–457; Figure* **b** *from: P. Perimenis, A. Athanasopoulos and M. Speakman, "Primary adenocarcinoma of the rete testis", International Urology and Nephrology (2003) 35: 373–374)*

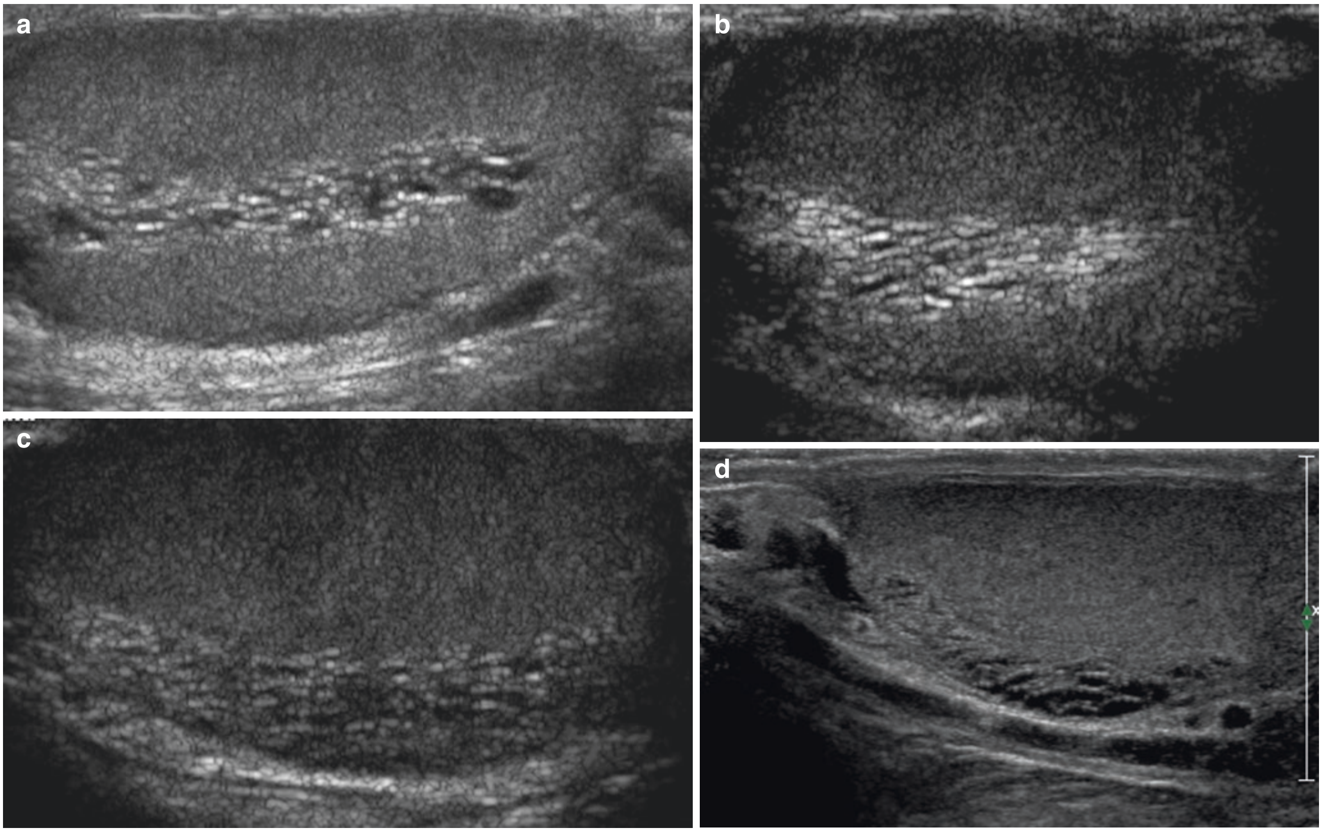


Fig. 2.15 Age-related dilation of the rete testis. Longitudinal images show a consistent number of very small anechoic spaces along the mediastinum testis, which represents the tubular ectasia of the rete testis (**a–d**)

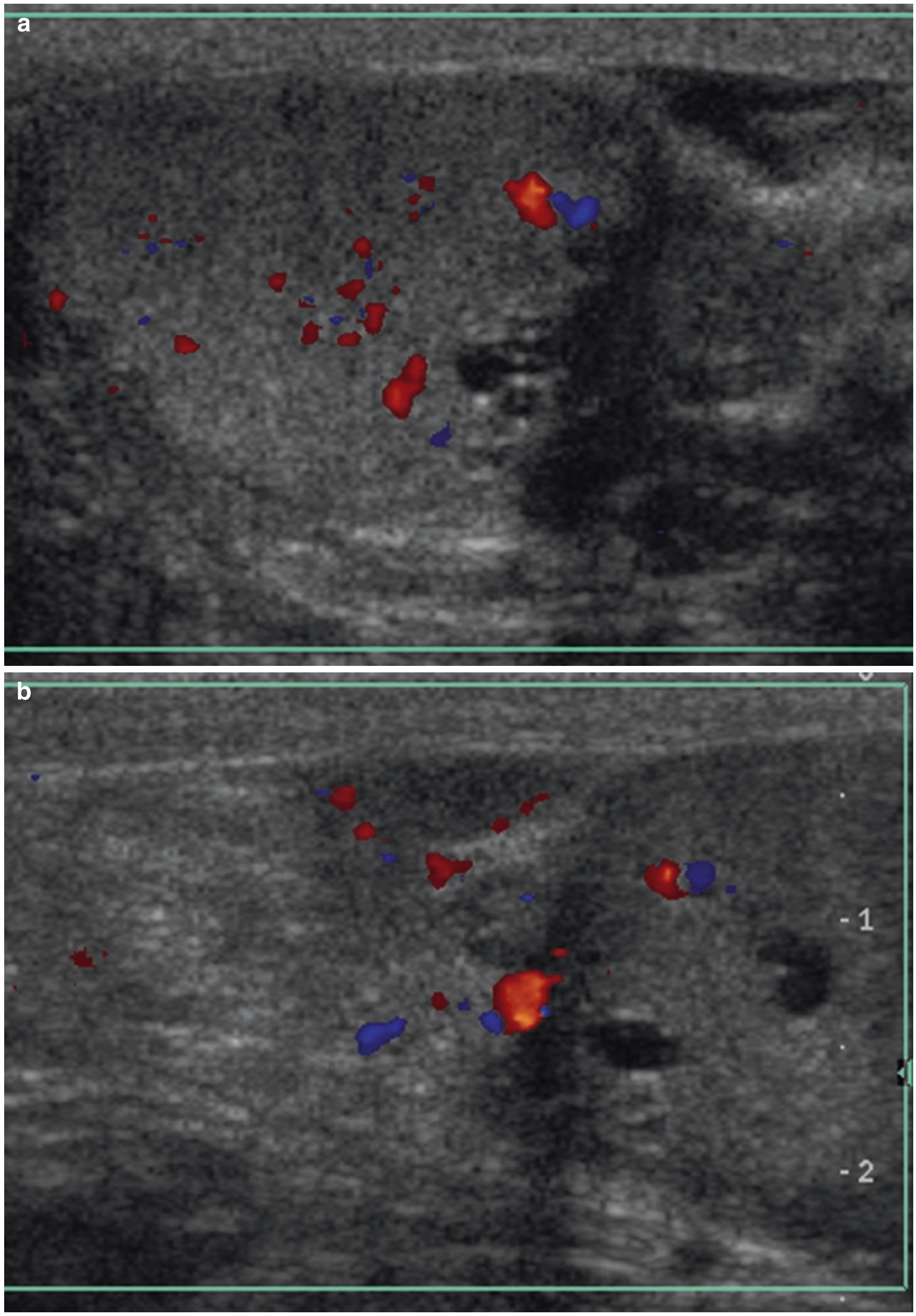


Fig. 2.16 Age-related dilation of the rete testis. Greyscale sonogram of the testis shows sparse anechoic and avascular tubular structures at the mediastinum in the posterior-lateral region (**a**, **b**)

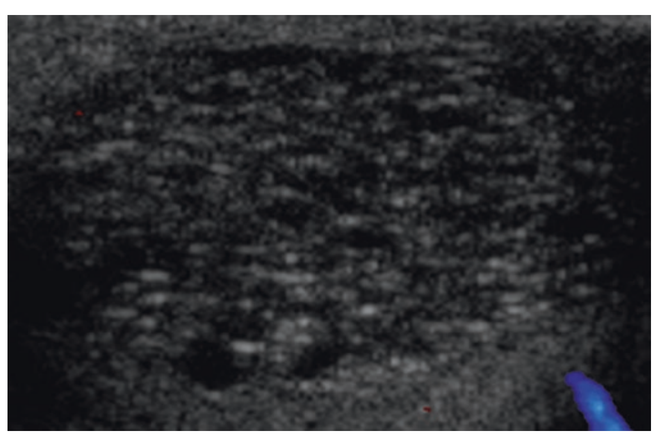


Fig. 2.17 Cystic dysplasia of the testis. Longitudinal sonogram of the testis demonstrates multiple tiny cysts throughout the testis

2.2.4.4 Rete Testis Cystadenoma

Rete testis cystadenoma is a rare cystic lesion of the rete testis, usually presenting as a palpable scrotal mass in patients of various ages. It is normally grossly visible and circumscribed, with solid and cystic components.

2.2.4.5 Vasectomy

Vasectomy involves occlusion of the vas deferens and blockage of the passage of sperm from the epididymis to the ejaculatory duct. After vasectomy, the testes continue to produce sperm, whose passage into the obstructed epididymis and vas deferens causes increased intraluminal fluid pressure. When the obstructed epididymis and efferent ducts are no longer able to compensate for this increase through dilation and increased fluid absorption, intermediate postvasectomy findings may be observed. On ultrasound, these are seen as a variable degree of enlargement of the epididymal body with multiple interfaces. An intratesticular image is occasionally found in the mediastinum testis, consisting of several anechoic and serpiginous tubular structures, with no solid areas and no mass effect on the adjacent testicular parenchyma (Fig. 2.18a). In such patients, the tail and occasionally the head of the epididymis are also involved. On colour Doppler sonography, tubular ectasia of the epididymis is typically hypovascular, but occasionally some flow may be seen at their periphery (Fig. 2.18b).

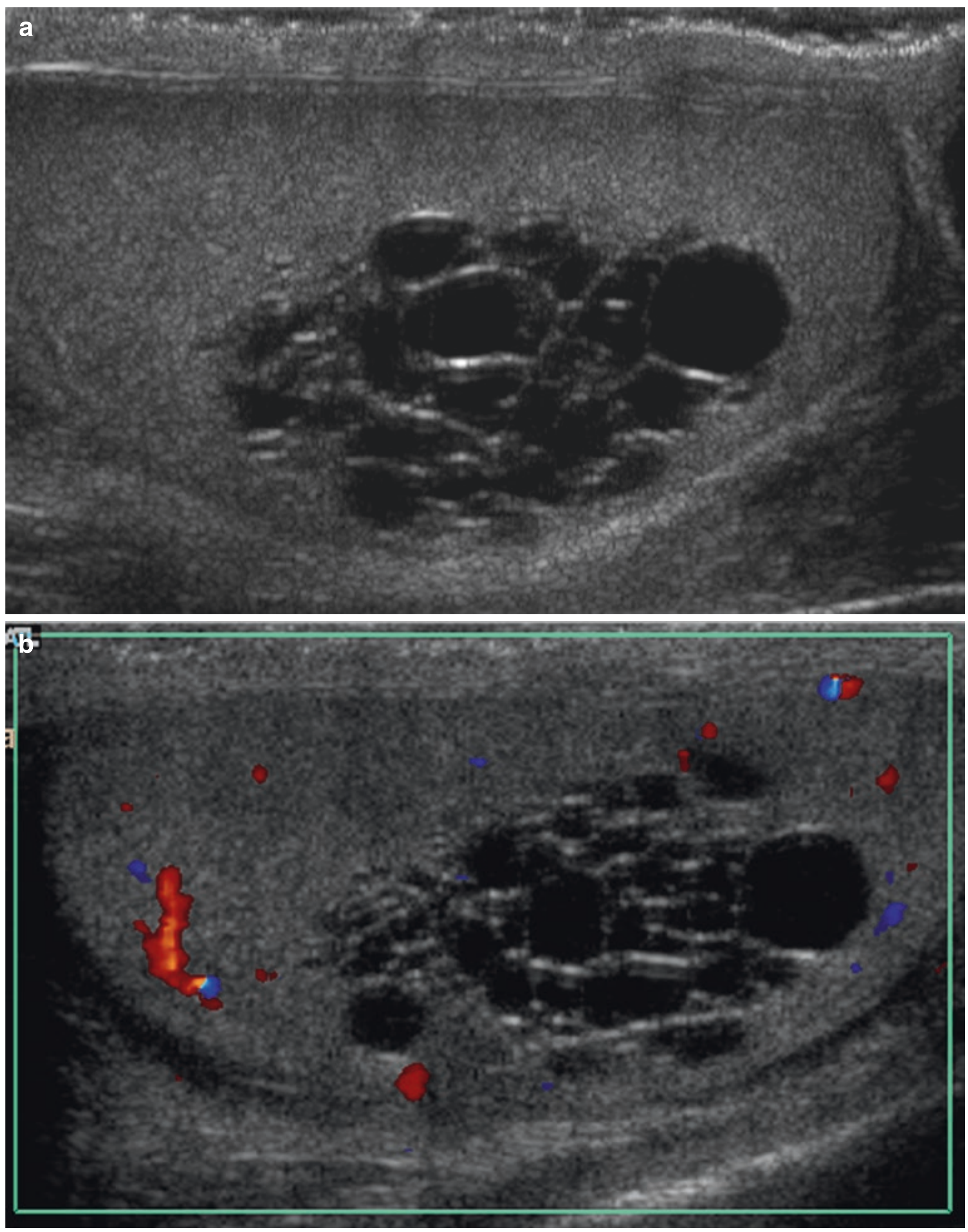


Fig. 2.18 Vasectomy. Hypoechoic cluster cystic configurations in the mediastinum testis (**a**). Colour Doppler imaging shows no evidence of flow (**b**)

2.2.5 Epidermoid Cysts

Epidermoid cyst of the testis, also known as **keratocyst**, is a rare benign testicular tumour of germ cell origin, ranging in size from 1 to 3 cm and whose appearance on ultrasound is variable. Although considered among the most frequent benign lesions of the testis, it accounts for less than 1% of all testicular tumours. The patient's age at presentation is variable but is usually between 20 and 40. Most patients present with testicular enlargement with no clinical symptoms, with a firm (non-tender), usually palpable, painless isolated unilateral lump in the testis. These cysts are not malignant and can be treated by enucleation.

Several theories for the histogenesis of epidermoid cysts have been postulated, including squamous metaplasia of the seminiferous epithelium or rete testis, epidermal inclusion cysts and monodermal development of a teratoma along the line of ectodermal cell differentiation [19]. Current opinion suggests that most epidermoid cysts are derived from epithelial rests or inclusions and are not a part of the malignant spectrum [20]. Pathologically, the cyst is intraparenchymal, has a complete or incomplete inner lining of squamous epithelium and contains keratinised debris or amorphous material with cleft-like spaces within the lumen [20]. A fibrous capsule with or without calcification or ossification may be present [21]. Although cystic in nature, it is filled with keratin and so may appear solid. The US appearance varies with the degree of maturation, compactness and quantity of keratin within the cyst. The lesions are rounded and well defined with marginal vascularity around the wall (Fig. 2.19) but are totally avascular within, with no surrounding parenchymal distortion [22, 23] (Fig. 2.20). Any clear vessel inside the lesion excludes the diagnosis of epidermoid cyst (Fig. 2.21).

The cysts may have an echogenic fibrous or calcified rim, and internal calcifications may also occur. The ultrasound appearance of epidermoid cysts is not constant, but the '**onion ring**' pattern of alternating hyper- and hypoechogenicities caused by laminated layers of keratin has been well described [20]. The combination of an onion ring configuration, negative tumour marker status and avascularity helps differentiate testicular epidermoids from other germ cell tumours [7].

The ultrasound appearance of epidermoid cysts has recently been classified into four types: (a) the classic onion ring pattern (Fig. 2.22), (b) densely calcified mass, (c) cyst with peripheral rim or central calcification and (d) mixed pattern (Fig. 2.23). If an epidermoid cyst is suspected, then testis-sparing surgery may be performed with an on-table frozen section and biopsies of the surrounding parenchyma to exclude any associated testicular intraepithelial neoplasia. In contrast with testicular teratomas, carcinoma in situ has not been reported in the seminiferous tubules adjacent to an epidermoid cyst. For this reason, a tentative diagnosis based on imaging findings can provide tangible information when it is wished to avoid an orchiectomy. However, as the features of epidermoid cysts are variable (Fig. 2.24), some authors recommend the excision of all such lesions, with biopsy of the surrounding normal tissue. Others advocate a more conservative approach, especially if a calcified lesion is identified in an elderly, asymptomatic patient [20, 22].

We currently suggest a surveillance strategy if the size of the lesion does not cause obstruction or discomfort, while the final decision should also take any fertility issues into account.

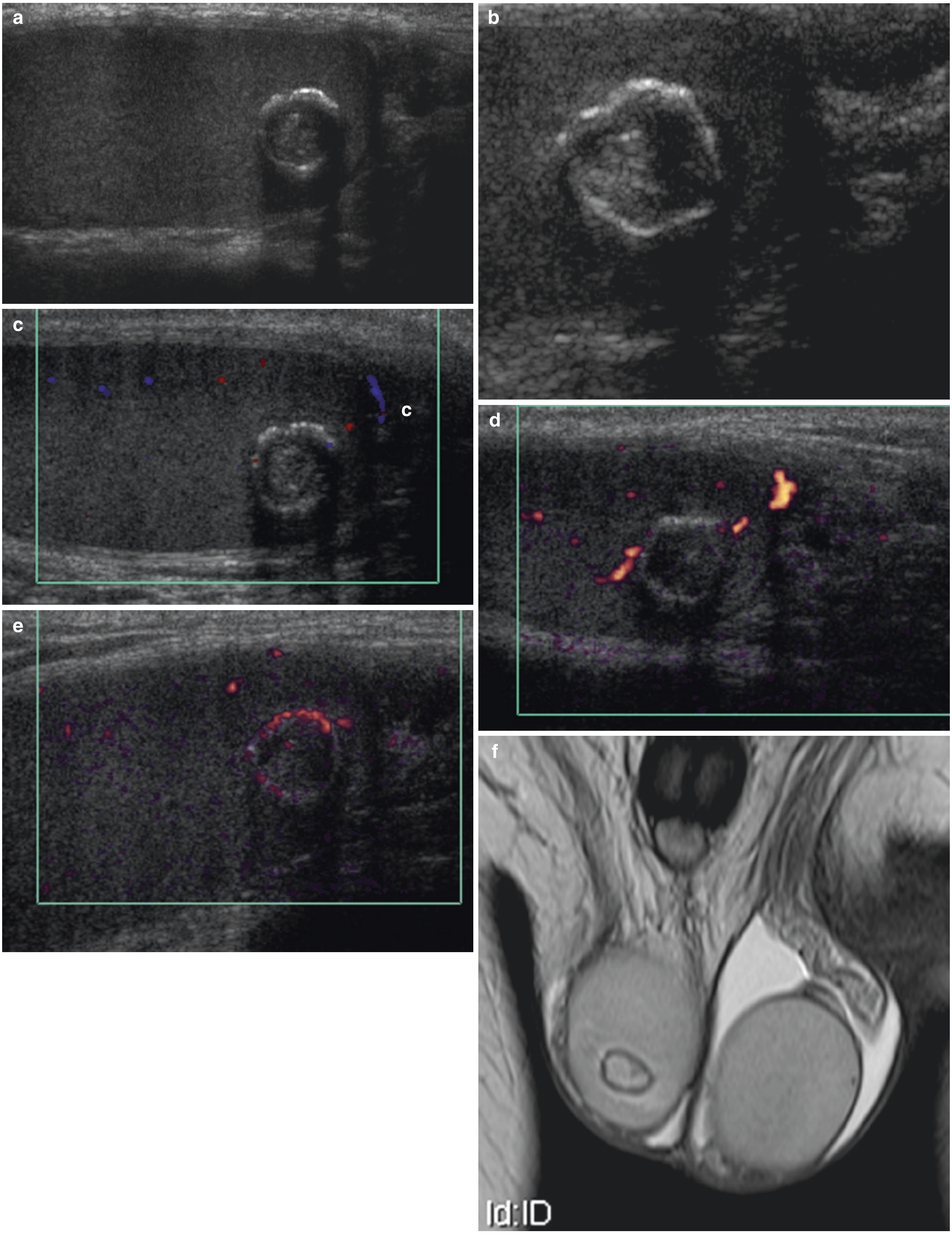


Fig. 2.19 Epidermoid cyst. Longitudinal US images of the right testis (**a**, **b**) show a well-circumscribed rounded intratesticular lesion in the lower pole of the testis. The lesion is surrounded by an echogenic rim, with marginal vascularity around the wall of the cyst (**c–e**). The lesion has high-density internal foci that cause incomplete shadowing. The surrounding testis is normal. Note the clean rim at MRI (**f**)

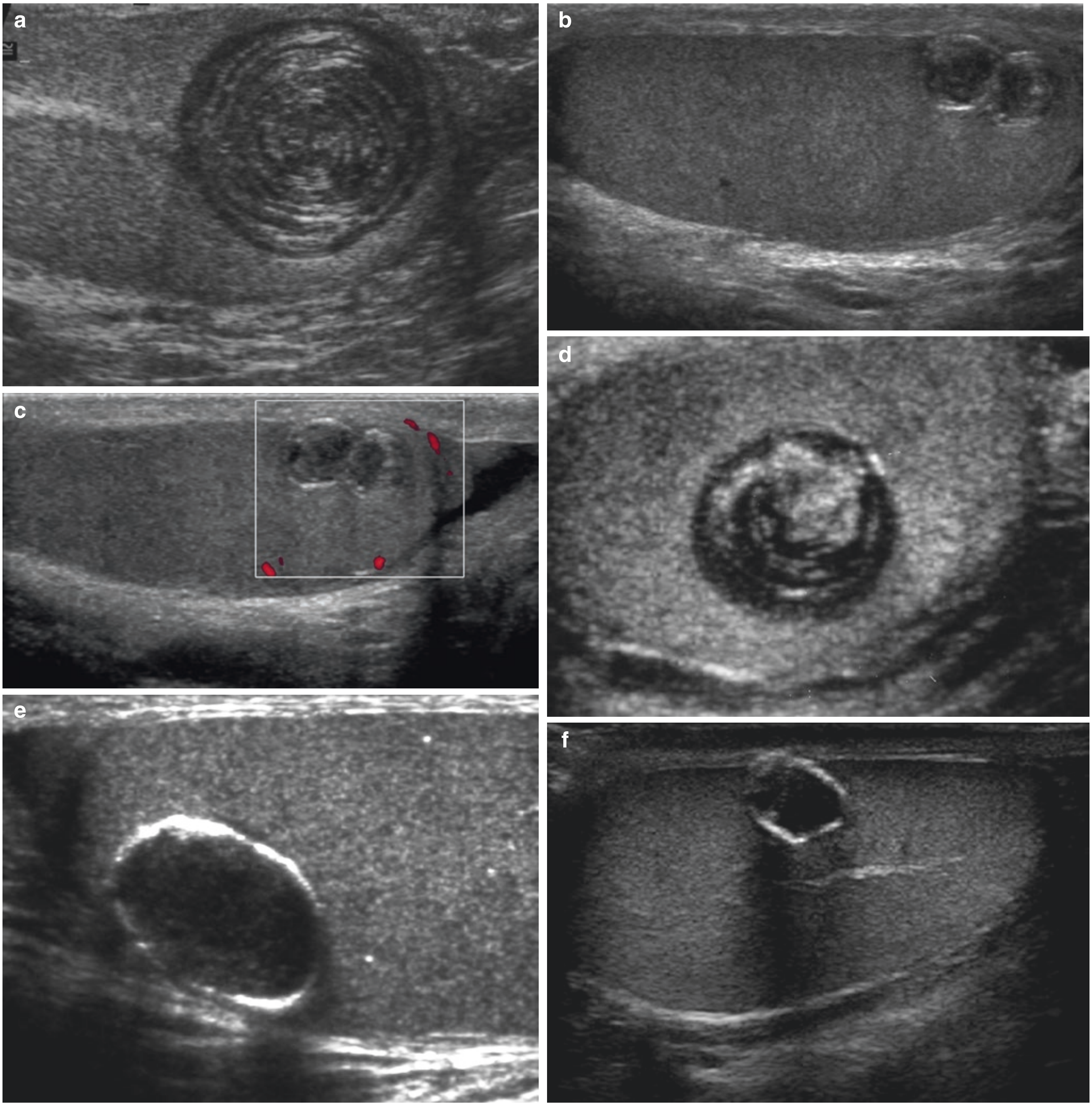
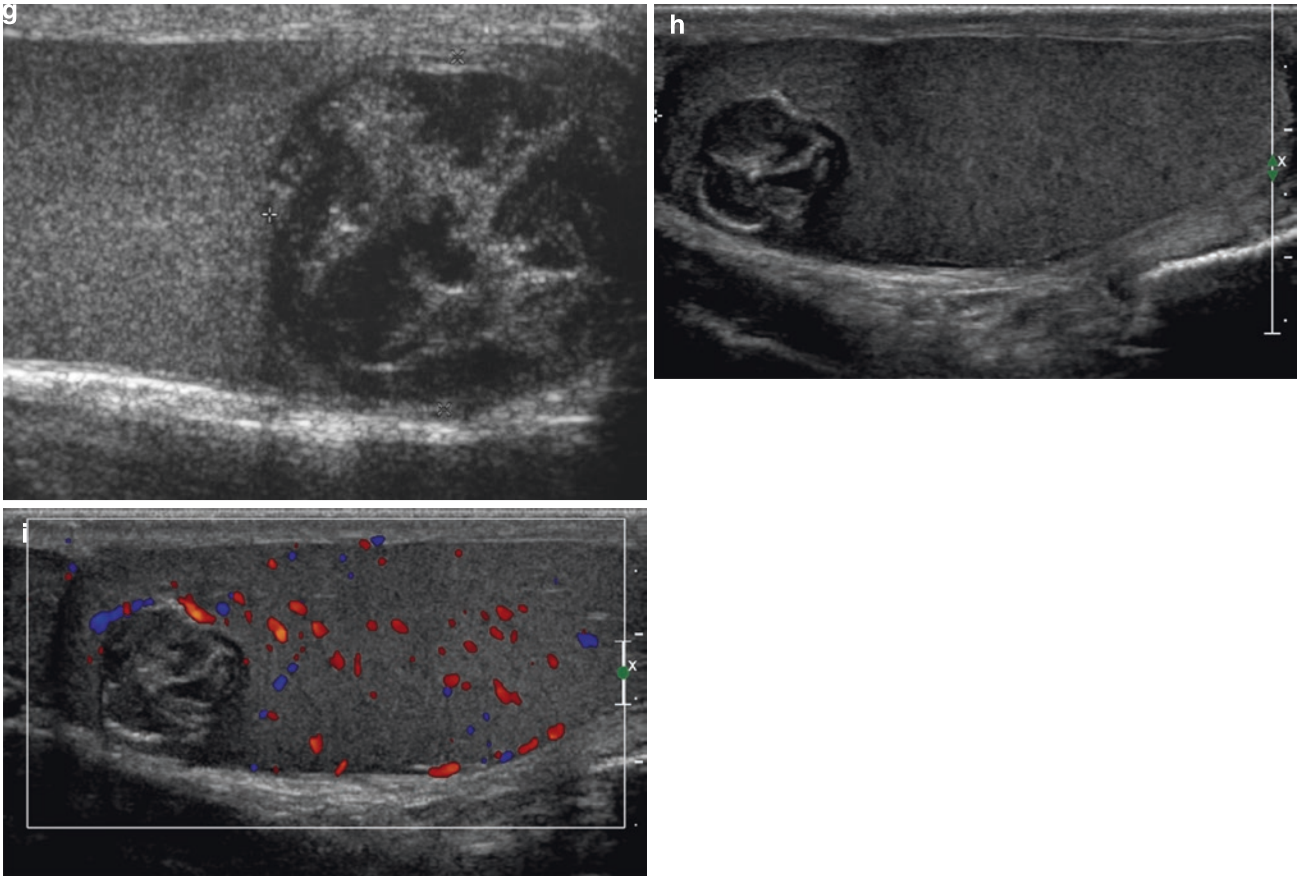


Fig. 2.20 Epidermoid cyst. Typical appearance of an epidermoid cyst: a definitive pattern of concentric rings, representing concentric spheres of keratin (**a**, **b**). No vascularity was shown on colour Doppler or power Doppler sonography (**c**). **Atypical epidermoid cysts**. A less typical appearance, but the anechoic capsules are clearly seen (**d**). **Epidermoid cyst**. In this case there are no concentric rings, but there is an outer, high-echodensity rim (**e**, **f**) with speckled high-density internal foci, causing shadowing; this pattern is also pathognomonic (**g**). **Epidermoid cyst**. Epidermoid cyst with a more speckled appearance (**h**, **i**) *(Figure* **a**, *Courtesy of: R.H. Oyen, MD*; **d–g** *From: Z.V. Maizlin, A. Belenky, J. Baniel et al., "Epidermoid Cyst and Teratoma of the Testis. Sonographic and Histologic Similarities" J Ultrasound Med (2005); 24:1403–1409)*



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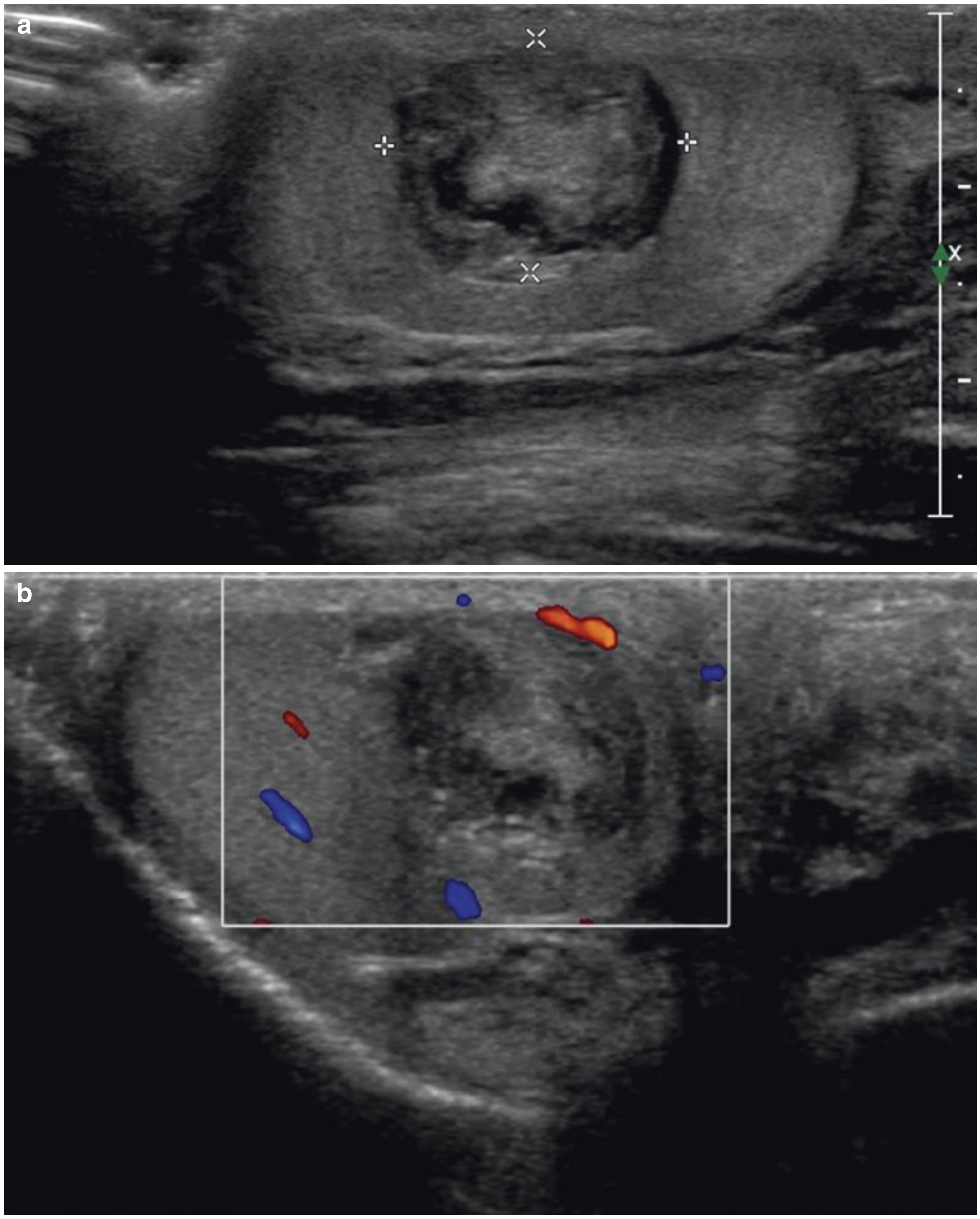


Fig. 2.21 Unusual epidermoid cyst. Greyscale (**a**) and colour Doppler (**b**) longitudinal ultrasound image of the right testicle shows a heterogeneous (hypoechoic and hyperechoic) mass

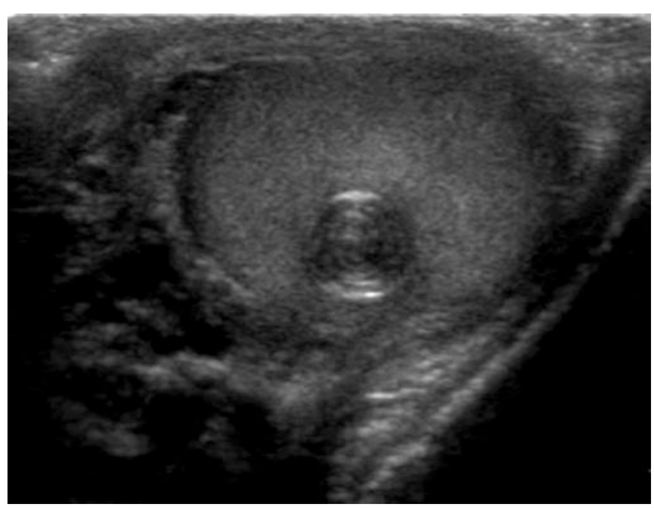
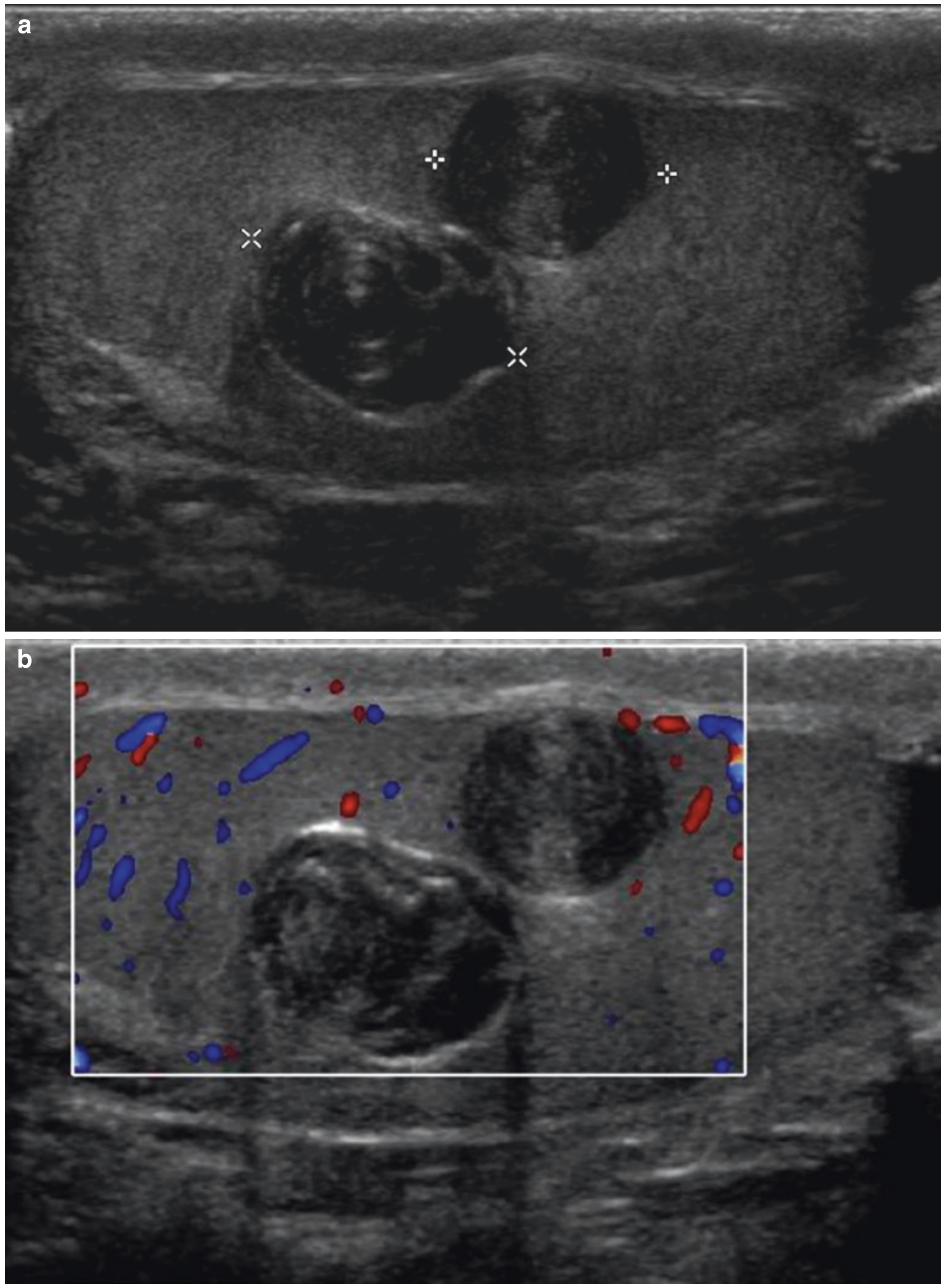


Fig. 2.22 Epidermoid cyst. Longitudinal scan of the testis shows a well-circumscribed intratesticular mass in the lower medial area, which contains alternating rings of hyperechogenicity and hypoechogenicity and has a slightly echogenic centre. The surrounding testis is normal

Fig. 2.23 Multiple epidermoid cysts. Two well-circumscribed, solid, mixed-reflective lesions in the testis, without colour Doppler signal and surrounded by a high-reflective rim (**a**, **b**); an epidermoid cyst on histology



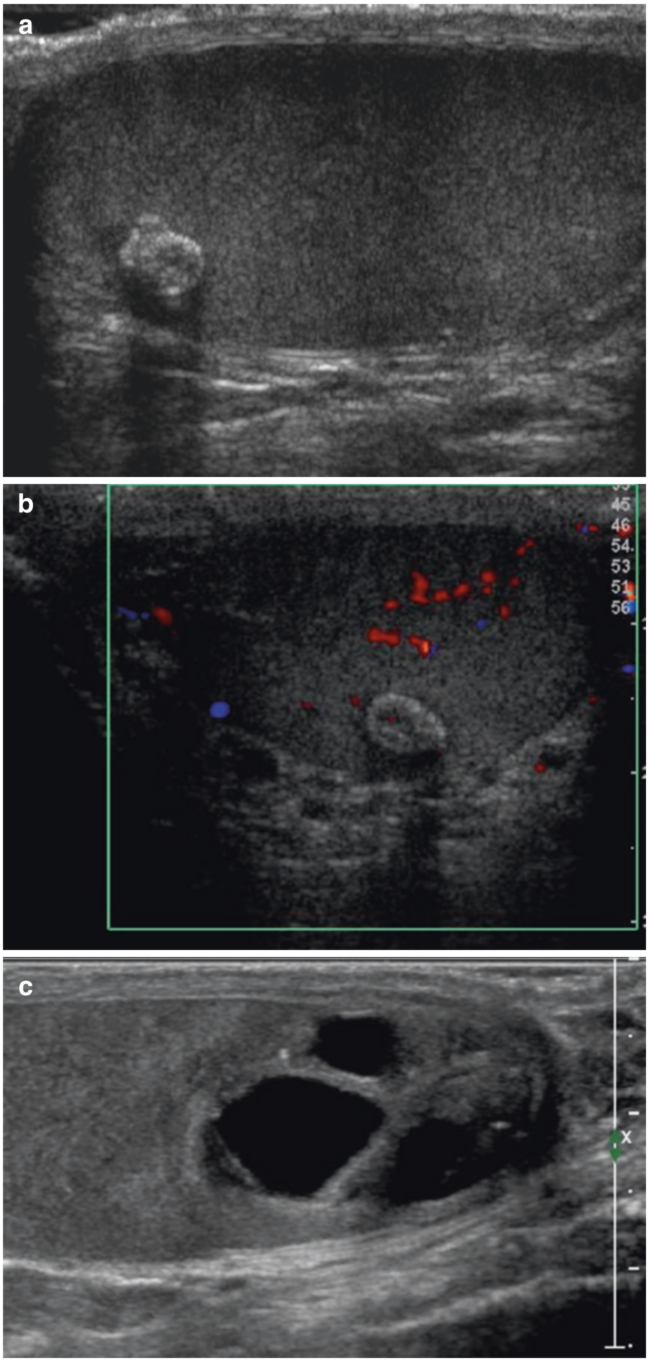


Fig. 2.24 Atypical epidermoid cyst. Atypical appearance of an epidermoid cyst of the testis (**a**, **b**). Dermoid cyst in a 34-years-old patient (**c**)

2.2.6 Dermoid Cyst

Dermoid cysts (Fig. 2.24, panel **c**) differ from epidermoid cysts in that they contain dermal adnexa and are difficult to distinguish from mature teratomas, as defined in the WHO classification. Malignancy is the most important differential diagnosis. A mature teratoma occurring before puberty is regarded as a benign lesion, as only non-metastasising cases have been described to date. The leading symptom, as in all other testicular tumours, is painless enlargement of the testicle. In children, however, this may occasionally be associated with local inflammation and must be differentiated from testicular torsion or epididymitis.

2.3 Calcifications

Scrotal calcification is an uncommon condition characterised by calcium deposits within the seminiferous tubules. It may be within the testicular parenchyma, on the surface of the testis or extratesticular within the tunica vaginalis or epididymis [24].

Solitary parenchymal calcifications not associated with a mass may be due to prior trauma, infection and infarction or represent a burnt-out tumour (Fig. 2.25). They have also been found in association with Klinefelter's syndrome, Peutz-Jeghers syndrome, Down's syndrome and male pseudohermaphroditism. When more than **five microcalcifications** are present on

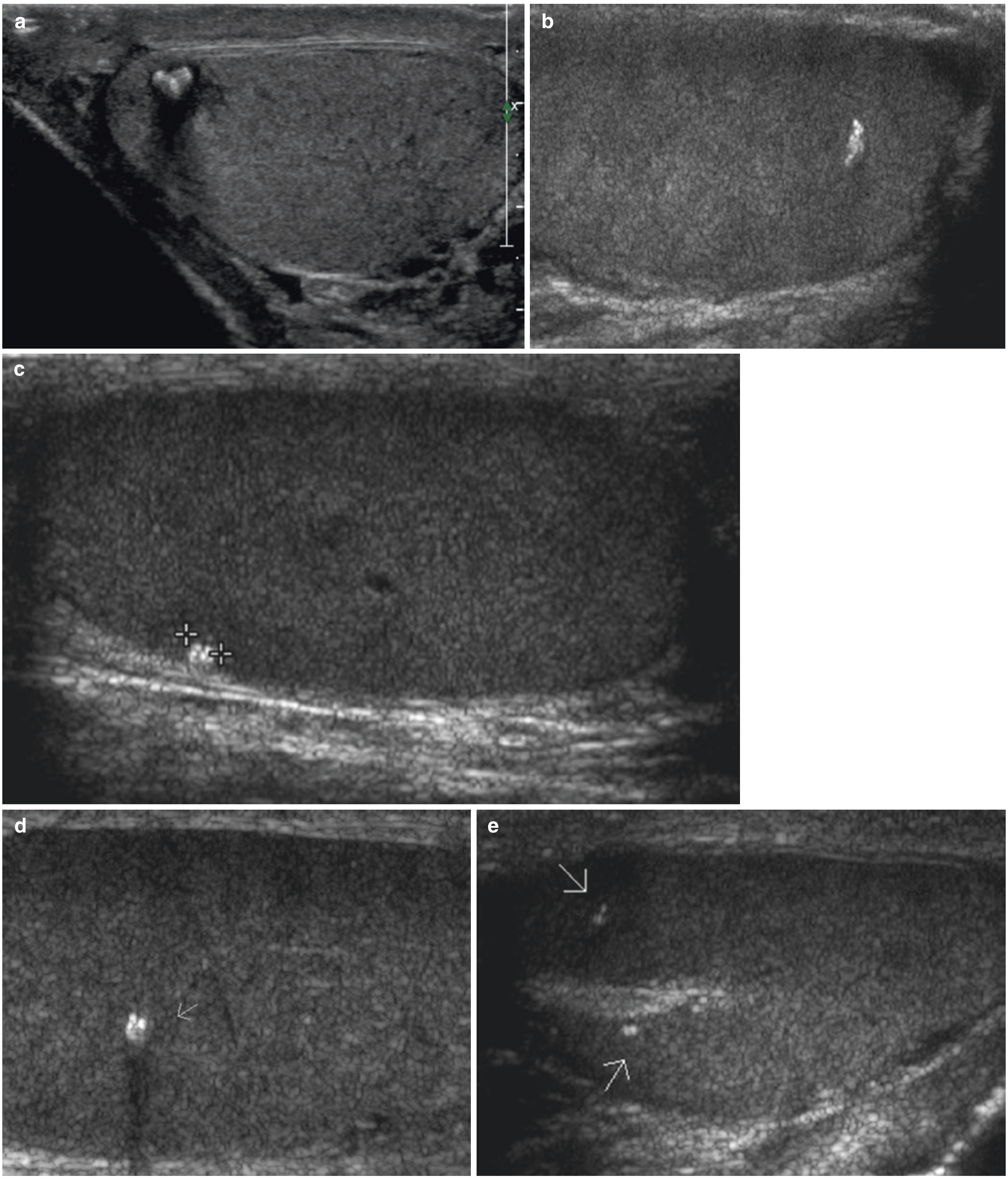


Fig. 2.25 Isolated calcifications. A focal area of calcification within the testis (**a**), cluster of echogenic foci (**b–d**) and several punctate foci (*arrows*) (**e**). Small hyperechoic lesions like these are usually benign and nonprogressive

one image of the testis, this is termed **testicular microlithiasis** [25, 26, 27]. A diffuse microlithiasis gives the appearance of '**starry sky**' to the testicular parenchyma. Although its true prevalence in the general population is still unknown, reported prevalence ranges from 0.6 to 9% [28].Ultrasound (US) reveals multiple 1–3 mm brightly echogenic foci within the parenchyma of the testis [29]. The number of calcified foci and the distribution pattern can vary. Diffuse symmetrical distribution of foci is the characteristic pattern, but asymmetrical distribution, unilateral foci and peripheral clumping have all been described (Fig. 2.26). Acoustic shadowing is not seen, probably due to the small size of the calcifications [30].

Testicular microlithiasis (TM) has been categorised into two histological types of intratesticular microcalcification. The first is the haematoxylin body consisting of amorphous calcific debris, which is associated with germ cell tumours and thought to be the result of a rapid cell turnover. The second is laminated calcifications, which are found in association with

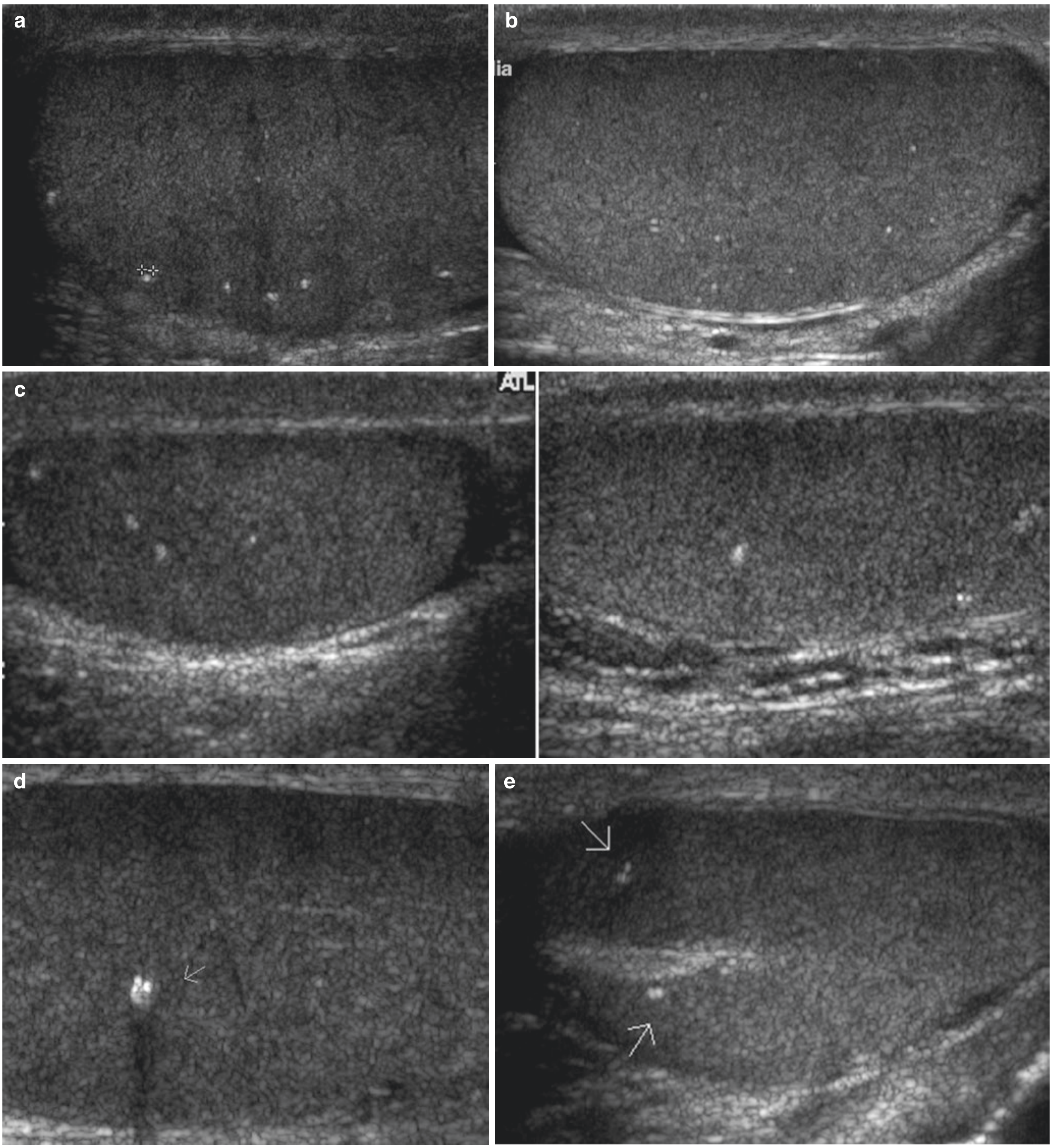


Fig. 2.26 Testicular microlithiasis. Longitudinal US scan shows punctate echogenic foci (more than five) in the central portion of the testis. A selection of sporadic (**a**–**d**) and diffuse (**e**–**i**) microcalcifications

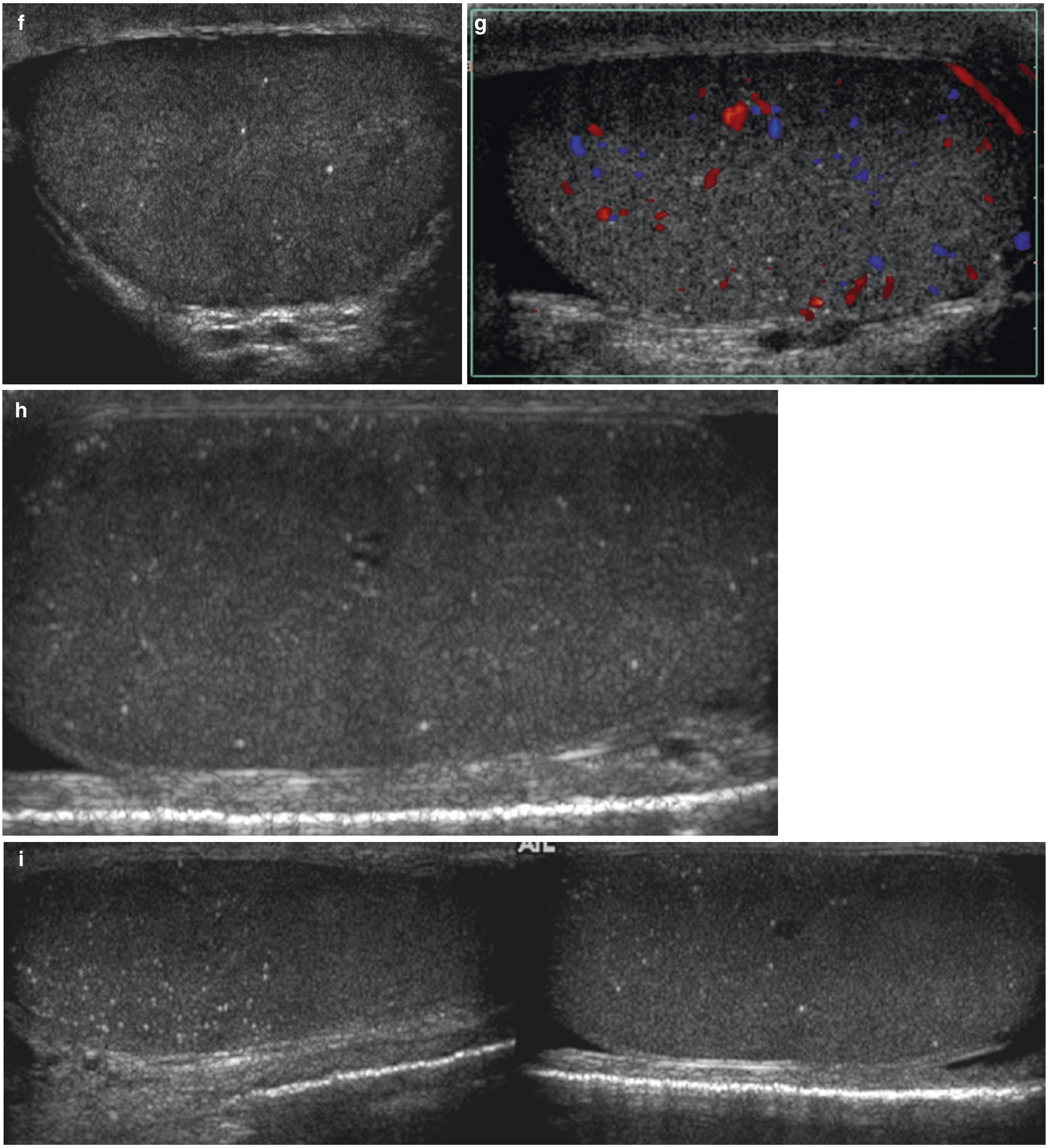


Fig. 2.26 (continued)

germ cell tumours, cryptorchid testes and otherwise normal testes [31]. Testicular microlithiasis has been associated with an increased incidence of testicular tumours (up to 42% in one series), so these patients must be observed with routine ultrasound examinations and serum tumour markers [26].

The extent of the risk of developing a tumour is not yet clearly defined but appears to be greater with large numbers of microcalcifications (Fig. 2.27) (starry sky appearance). Early studies showed a substantially increased risk of developing carcinoma, but most recent papers have drastically

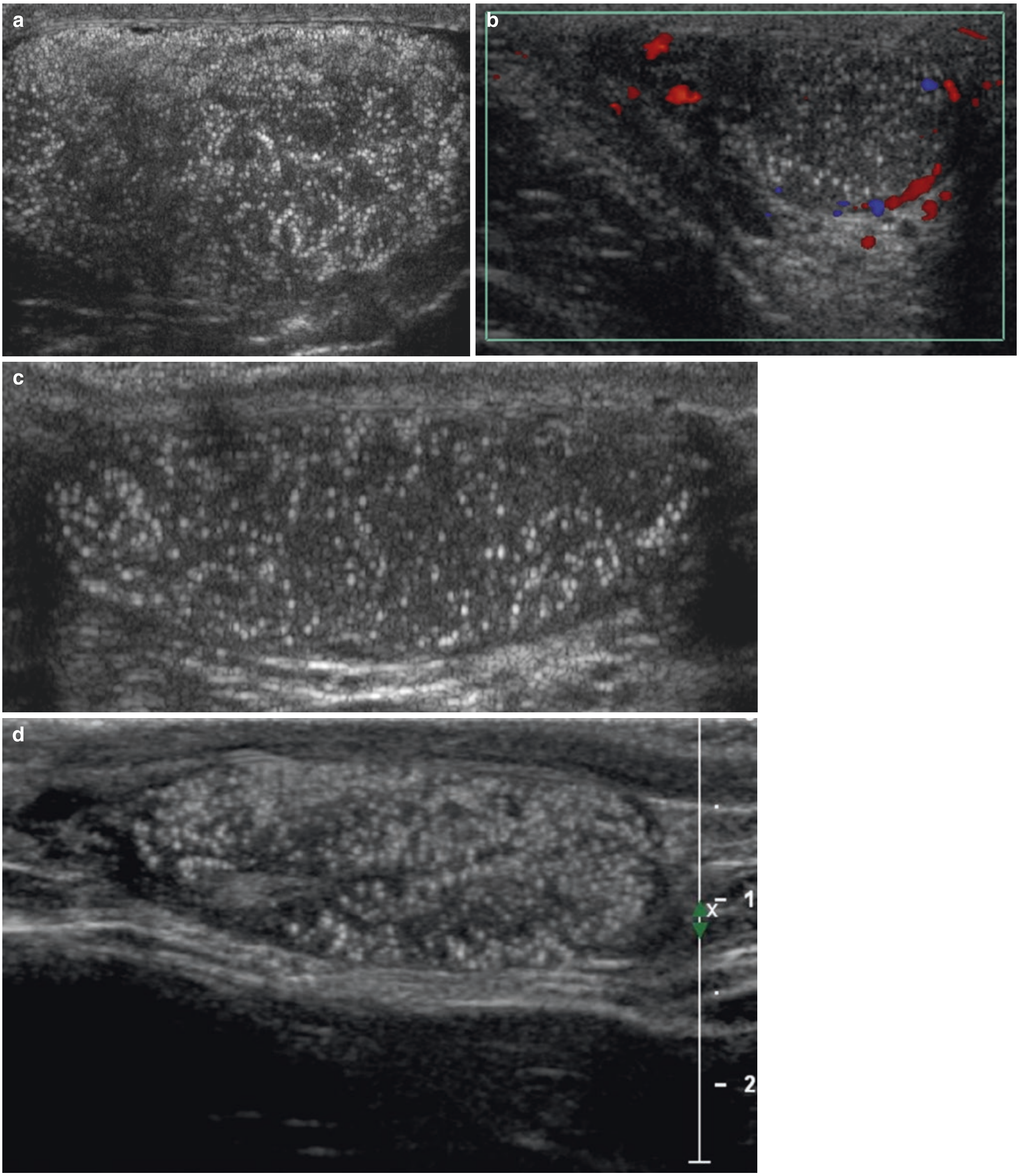
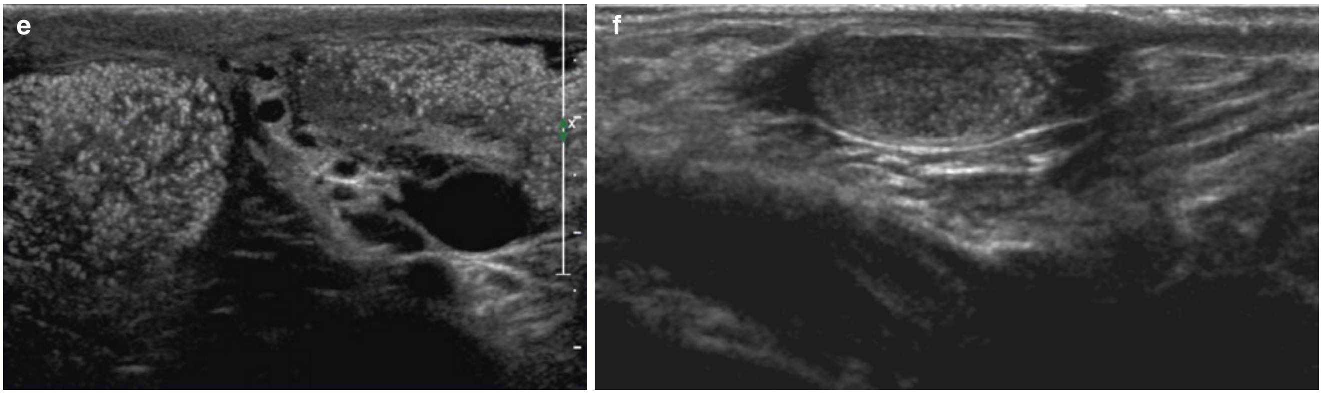


Fig. 2.27 Testicular microlithiasis. Diffuse microcalcifications ('snowstorm') in an apparently healthy testicle (**a**). Multiple diffuse echogenic foci with no acoustic shadowing, which represent punctate calcifications, are shown in the left testis (**b**, **c**) of a 25-year-old man, who had undergone a contralateral right orchiectomy for an embryonal cell carcinoma. Bilateral microlithiasis in a 35-year-old man (**d**, **e**) and in a 3-year-old boy with Klinefelter's syndrome (**f**)



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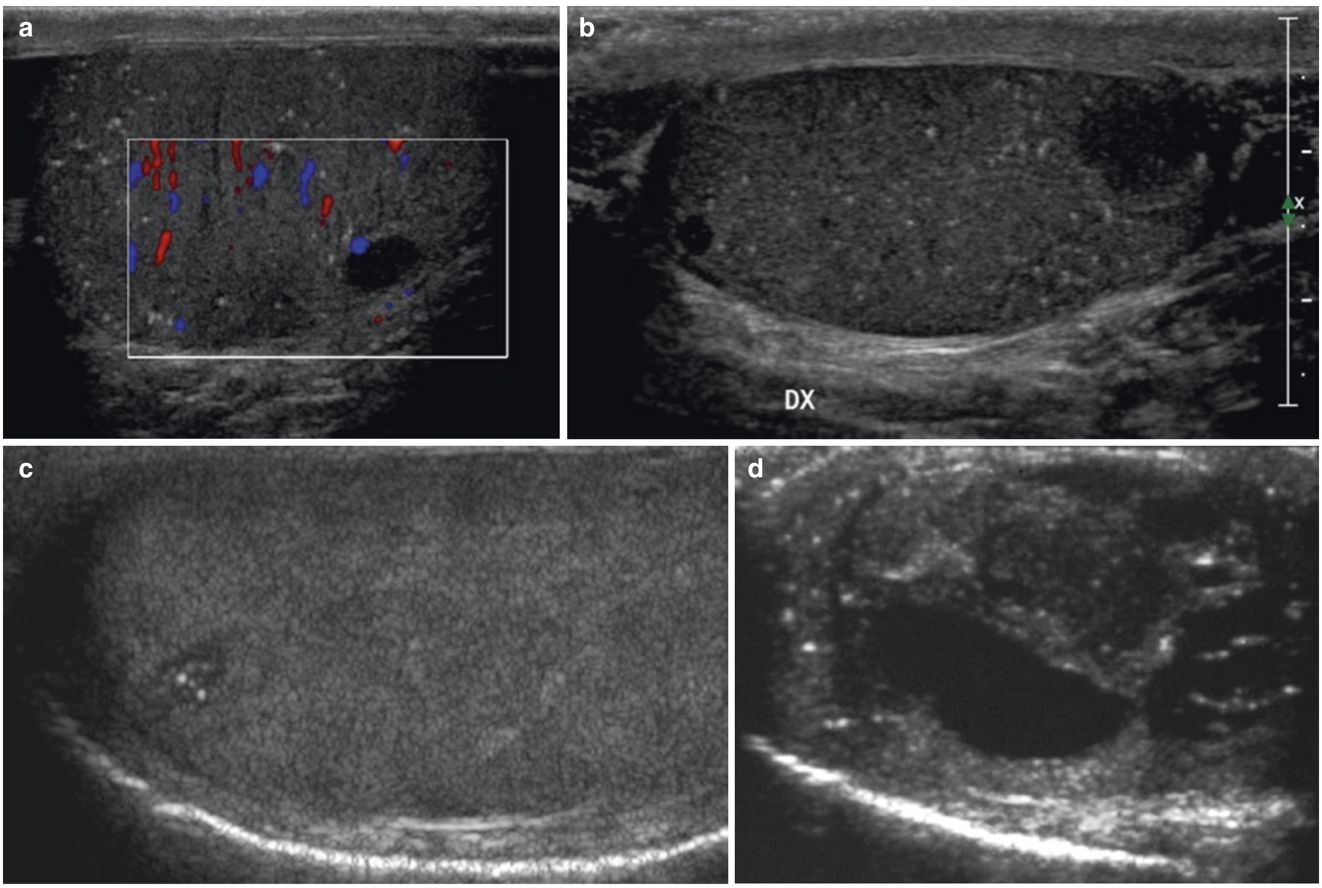

Fig. 2.28 Testicular microlithiasis. Testicular microlithiasis was found at US in association with a hypoechoic seminoma (**a**–**c**) and a malignant teratoma (**d**) *(figure* ***d*** *from: S. Basu, D.C. Howlett, "High-resolution ultrasound in the evaluation of the nonacute testis" Abdom Imaging (2001) 26:425–432)*

reduced the initial figures, although the actual risk is still not clearly quantified. Some centres offer a 6-month ultrasound screening; in the absence of any other suspect lesion, a 12-month screening would probably be more cost-effective. Nevertheless, microcalcifications are more common in patients with oligospermia; therefore assessment of sperm quality is recommended.

All these considerations may suggest the premalignant nature of TM. However, more recent studies show a lower incidence of associated GCT and no interval development of tumour in relatively longer-term follow-up. Recent literature suggests that both TM and testicular GCT may be caused by a common defect, such as tubular degeneration, and TM may present as a marker for such abnormalities; however, given the high association with GCT, it is prudent to follow up TM patients with physical examination and US at least annually and to encourage self-examination [28].

Seminoma, embryonal cell carcinoma and teratomas [22] are the neoplasms most commonly associated with TM (Fig. 2.28). The association of calcification with benign lesions in the testis is also well documented. Benign intratesticular tumours, often originating in the Sertoli and Leydig cells of the seminiferous tubules, are difficult to distinguish from malignant tumours and sometimes demonstrate calcification. Epidermoid tumours have a variable appearance on ultrasound, some having distinguishing features such as a well-demarcated hypoechoic mass with calcification in the wall or a mass surrounded by concentric rings, described as an 'onion ring' appearance. Simple testicular cysts are usually thin walled and anechoic, but they may contain calcification within the rim [22]. Calcification within the extratesticular portion is more frequent than intratesticular calcification and usually represents benign disease.

Localised calcification can be found on the surface of the tunica albuginea. These calcified bodies may slough off into the cavity between the layers of the tunica to form a '**scrotal pearl**' [26]. These are usually solitary, although occasionally they may be multiple and round and measure up to 1 cm in diameter, producing a discrete acoustic shadow. The aetiology of a scrotal pearl is unclear; it originates either as a fibrinous deposit in the tunica vaginalis or as a remnant of a detached appendix testis or appendix epididymis following torsion. Scrotal calculi are often found in association with a secondary hydrocele, thus rendering them impalpable [24]. The tunica vaginalis may occasionally calcify more extensively, producing a linear plaque with acoustic shadowing. Selected non-parenchymal calcifications are shown in Fig. 2.29.

Calcification in or adjacent to the epididymis is a common finding and is usually due to chronic epididymitis. The appendix epididymis and appendix testis may calcify; they can be recognised by their characteristic position and shape [30] (Fig. 2.30).

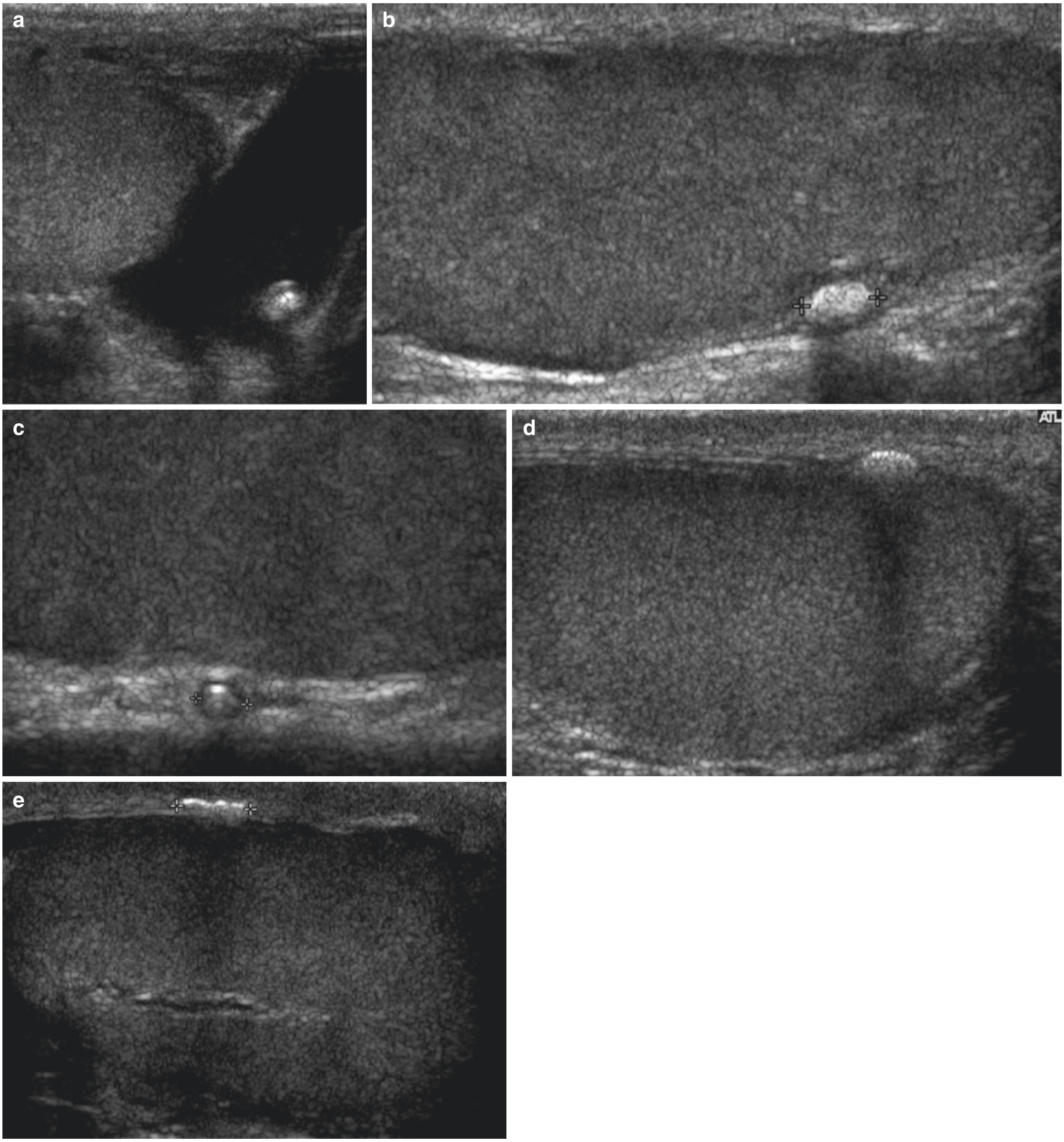

Fig. 2.29 Non-parenchymal calcifications. A selection of non-parenchymal calcifications. Scrotal pearl: a calcified loose body lying between the layers of the tunica vaginalis (**a**). They are often found in association with a secondary hydrocele, thus rendering them impalpable; typical calcified granuloma of the tunica (**b–d**). They can be palpated as a hard lump, usually only the size of a grain of rice, on the surface of the testis. They often appear as a linear plaque of calcification with acoustic shadowing (**e**)

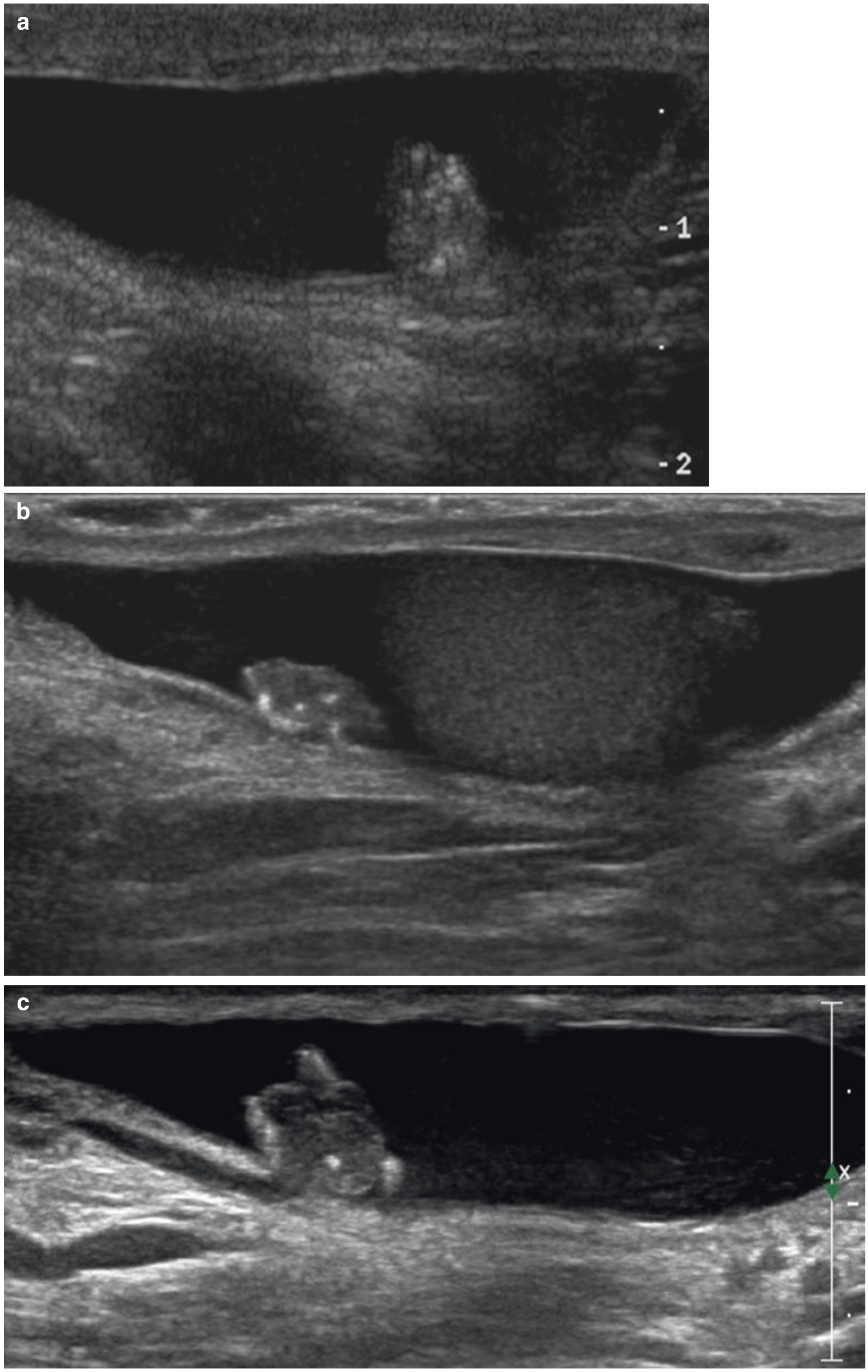

Fig. 2.30 Calcification of the appendix testis. This commonly occurs following a torsion of the appendix testis

2.4 Global and Segmental Ischaemia

Global testicular infarction (Figs. 2.31 and 2.32) usually results from torsion of the spermatic cord, severe epididymo-orchitis or trauma [32]. **Segmental testicular infarction** is an infrequent testicular disorder rarely described in the radiological literature and usually diagnosed after orchiectomy [33]. The predisposing factors for **segmental infarction** include polycythaemia, intimal fibroplasia of the spermatic artery, sickle cell disease, hypersensitivity angiitis, polyarteritis nodosa, thromboangiitis obliterans, arterial embolism and trauma, although most cases are still considered to be idiopathic in nature (Figs. 2.33 and 2.34) [32]. The theory of reduced blood flow – usually caused by venous thrombosis – to certain areas of testicular tissue functioning as end organs explains the pathogenesis of segmental testicular infarction in most cases [34]. This condition normally affects patients in their 20s to 40s, although it has occasionally

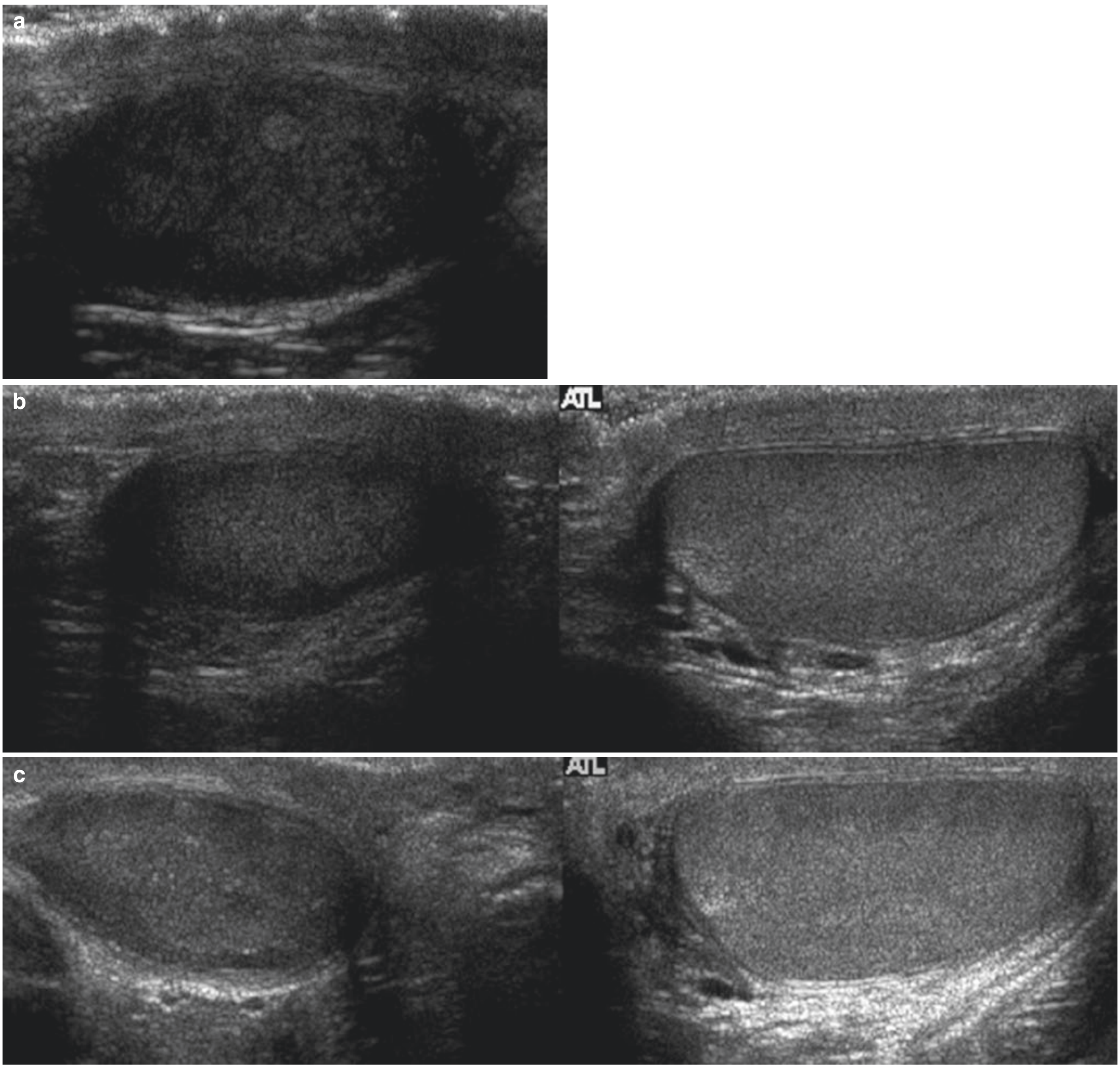

Fig. 2.31 Global testicular infarction. Ischemic atrophy of the left testis of a 45-year-old man, probably secondary to herniorrhaphy (**a**). In (**b**) the left and the right testes are compared. Another global infarction of the testis, secondary to ischaemic damage due to torsion of the spermatic cord (**c**)

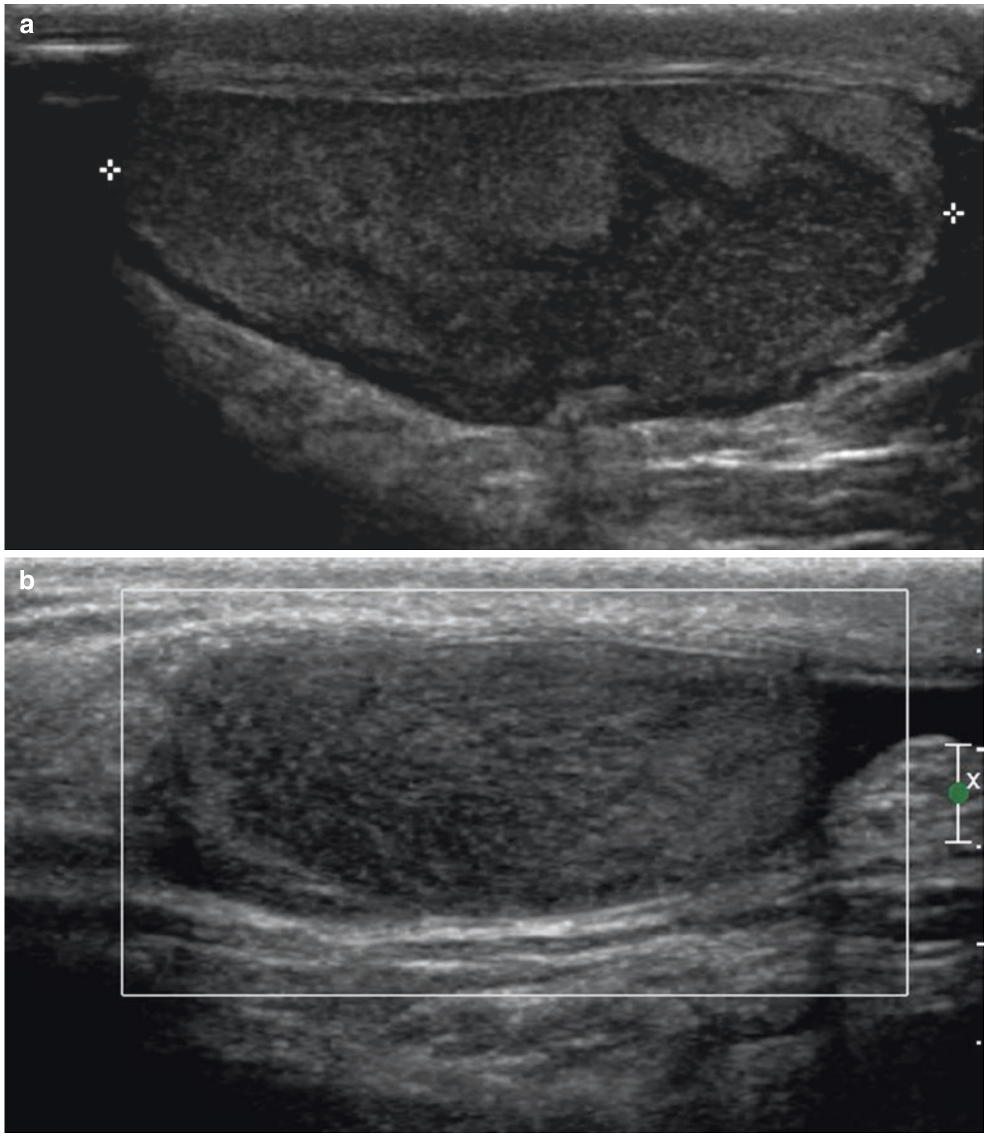

Fig. 2.32 Testicular infarction. Testicular infarction post-missed testicular torsion (**a**) and post-herniorraphy (**b**)

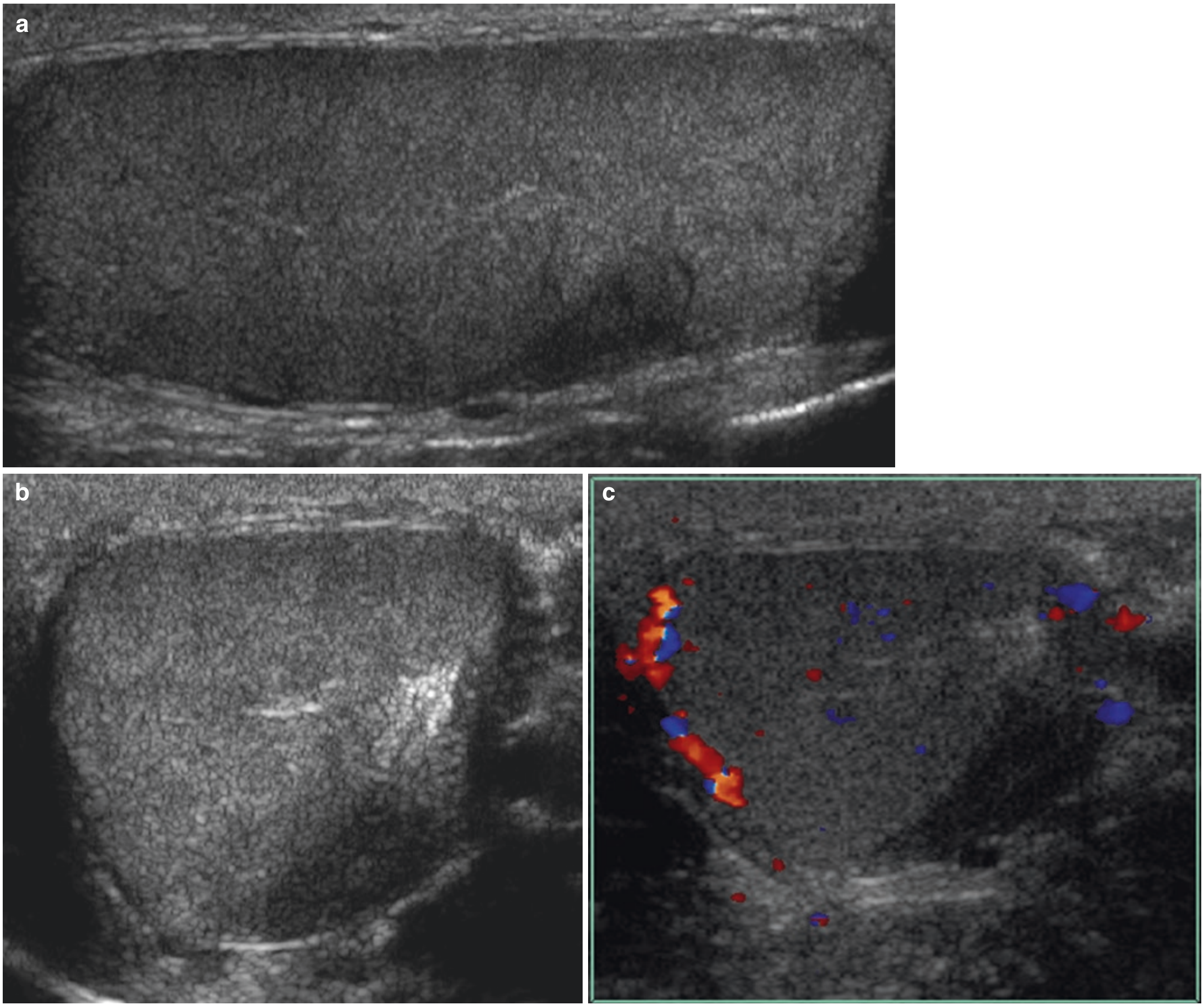

Fig. 2.33 Segmental infarction of the testis. B-mode ultrasound of the lower pole of the right testis demonstrating a triangular, hypoechoic area with an ill-defined margin (**a**, **b**). Colour Doppler ultrasound of the lower pole of the right testis showing lack of colour Doppler flow (**c**), strongly suggesting the presence of segmental ischaemia or area of infarction rather than a tumour

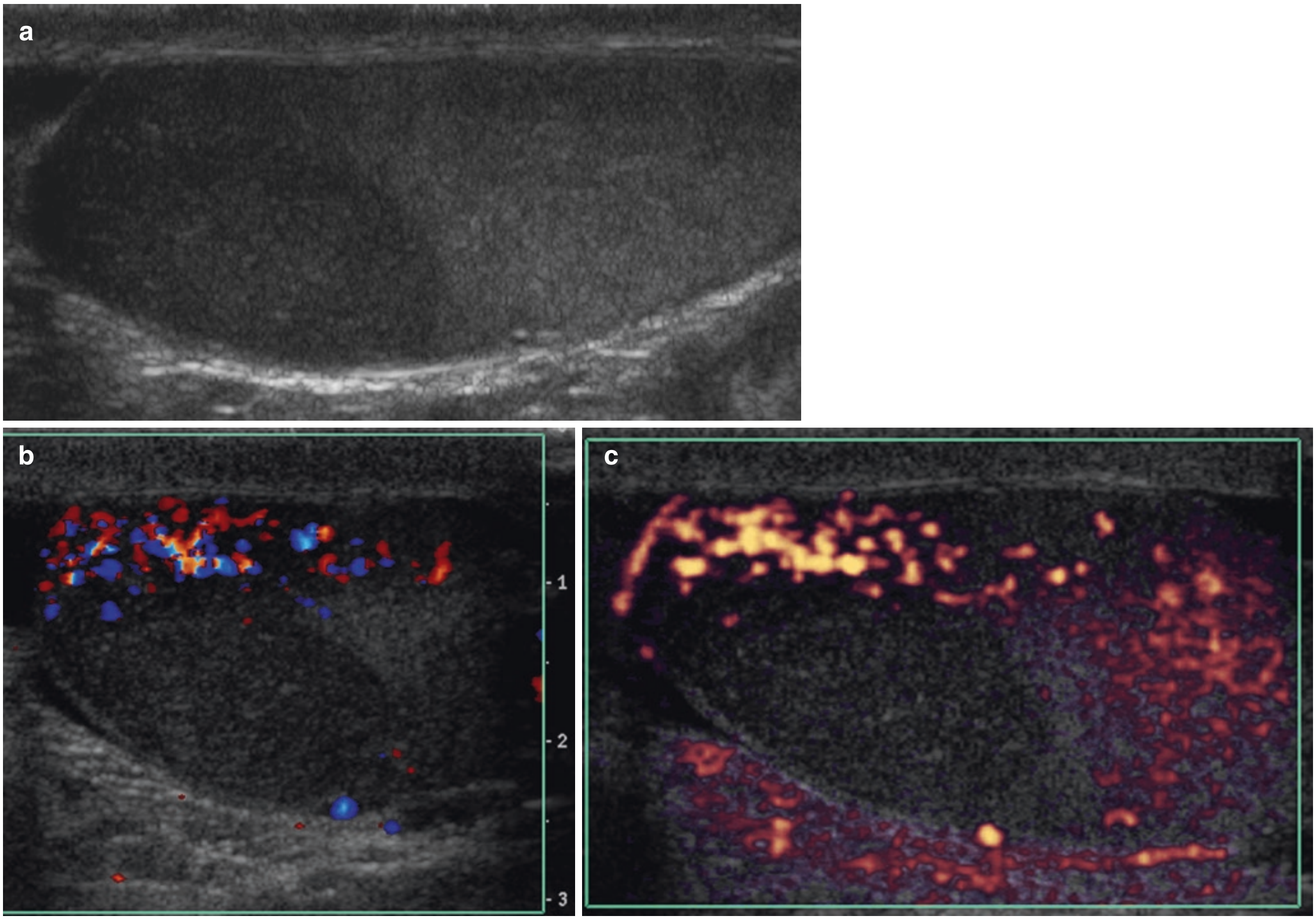

Fig. 2.34 Segmental infarction of the testis. Ultrasound evaluation of swelling revealing segmental infarction of left testis (**a**). Colour and power Doppler sonography (**b**, **c**) showing an avascular lesion in the upper pole of the right testis. Follow-up ultrasound demonstrated regression in the size of the abnormality over time combined with negative tumour markers, allowing a more confident diagnosis of segmental testicular infarction

been reported in neonates [35]. The clinical presentation is non-specific and usually heralded by acute scrotal pain and swelling. A few cases with recurrent testicular pain have also been reported; this sometimes provides an important clue to diagnosis [34]. The diagnostic imaging method of choice is colour Doppler, which shows an area without flow and otherwise normal testicular parenchyma (Fig. 2.35). However, on many occasions, segmental testicular infarction cannot be distinguished from a tumour with low flow, especially if the tumour is small, and confirmation is achieved only following surgery (Fig. 2.36) [32, 36].

Diagnosis may be aided by MRI, which clearly reveals the borders of the segmental testicular infarction not only on T2-weighted sequences but also on enhanced images [37]. The B-mode ultrasound hallmark of global testicular ischaemia and infarction in the acute stage is an enlarged low-reflective testis. This is followed by gradual shrinkage of the testis over time and an increase in reflectivity. Colour Doppler sonography is useful in the evaluation of testicular torsion, where diagnosis is confirmed by the absence of colour flow to one testis in the presence of normal flow to the contralateral testis [32].

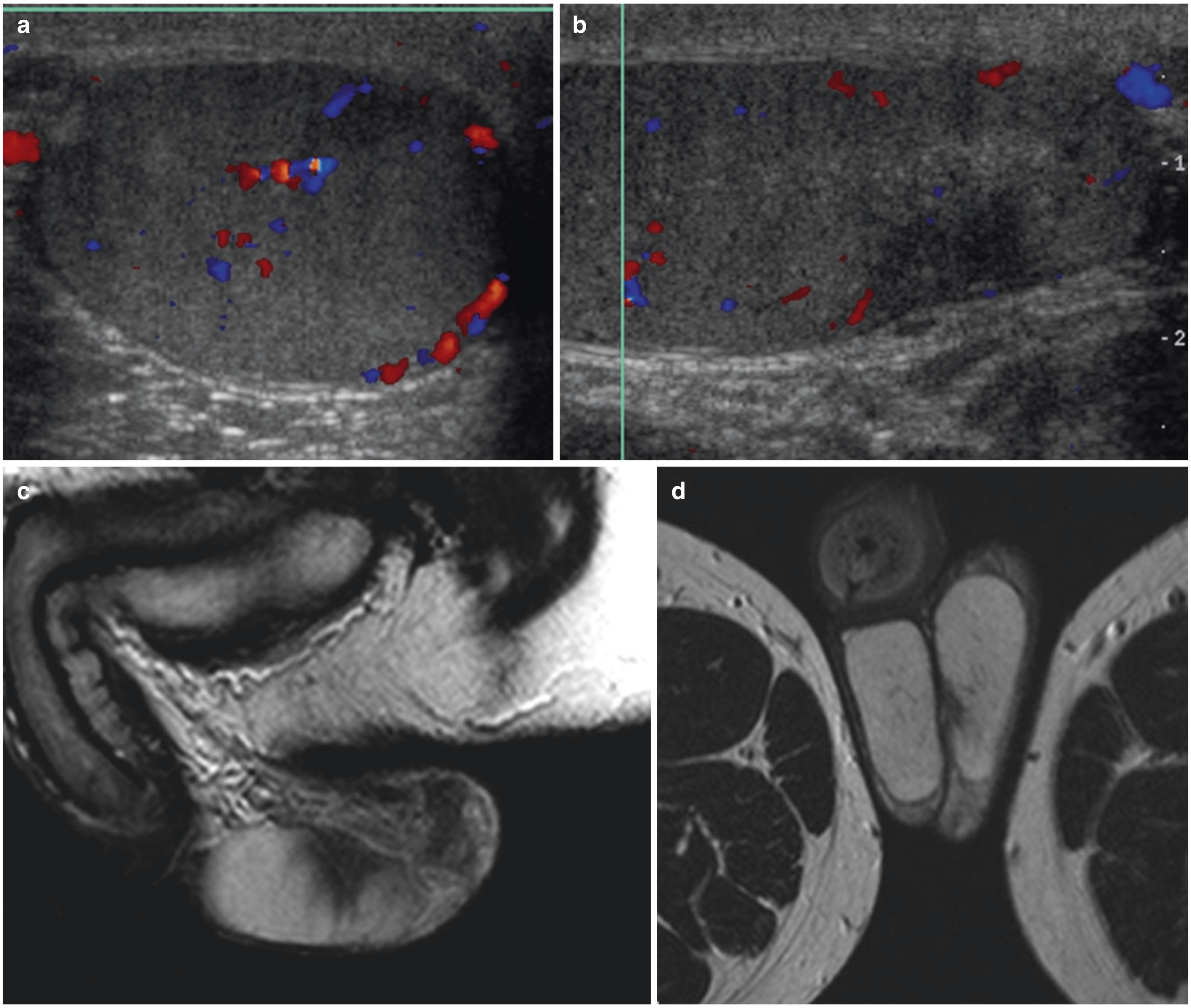

Fig. 2.35 Segmental infarction of the testis. A 48-year-old Caucasian male visited our andrology clinic 48 h after the onset of sudden, spontaneous, moderate-to-severe scrotal pain. Testes ultrasound revealed a segmental infarction of the testis (**a**, **b**). MRI T2-weighted transversal (**c**) and sagittal scans (**d**) of the testis confirmed the diagnosis

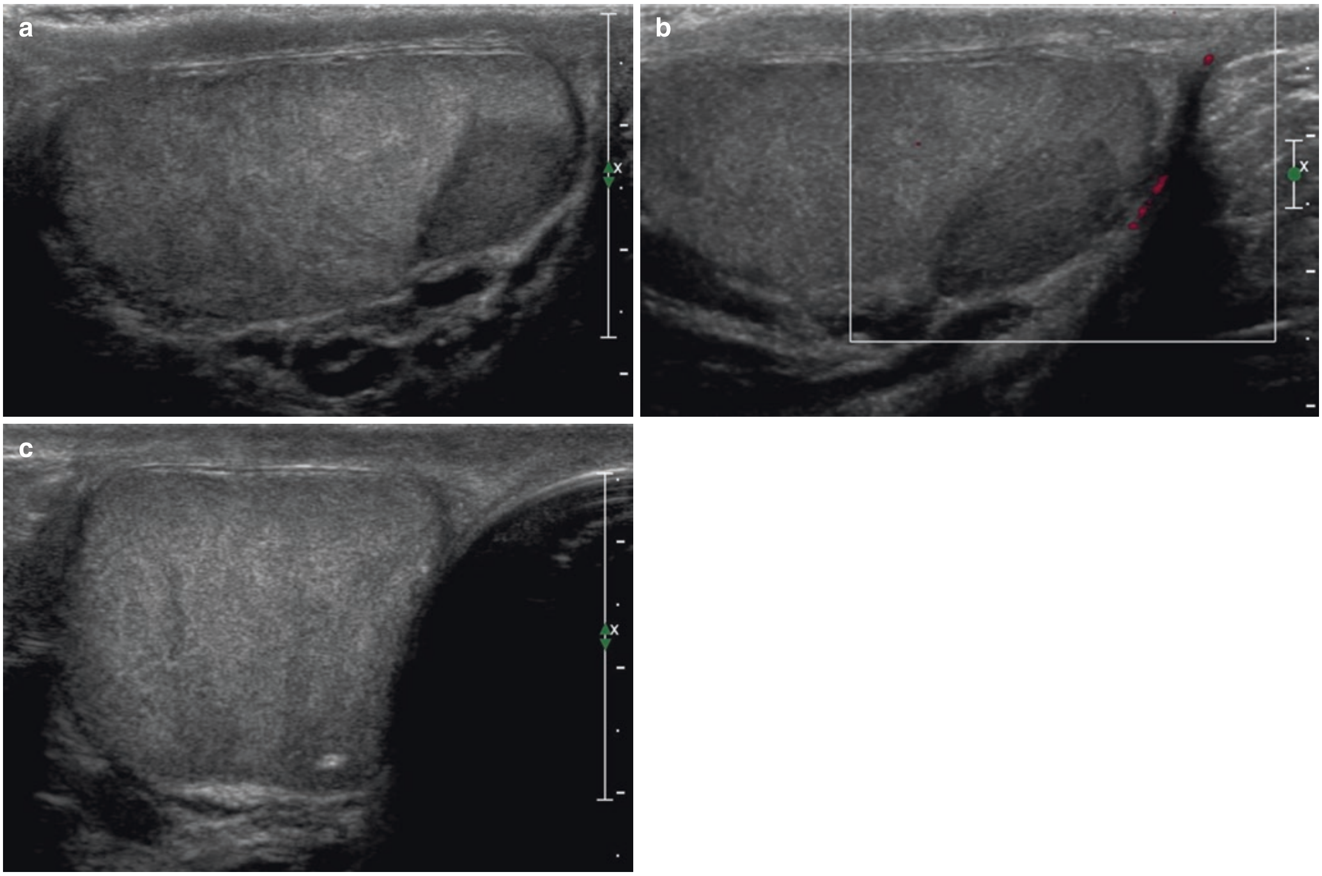

Fig. 2.36 Segmental infarction of the testis. Longitudinal scans revealing a partial left infarction of the testis (**a**, **b**) in a patient previously orchiectomised for a contralateral mixed germ cell tumour

2.5 Others

2.5.1 Abscess

A scrotal **abscess**, or **pyocele**, is usually a complication of epididymo-orchitis (Fig. 2.37) which has crossed the mesothelial lining of the tunica vaginalis [38] but may occur as a complication of testicular torsion or testicular haemorrhage or secondary to trauma or superinfection of a necrotic tumour. It is also associated with systemic infections such as mumps, smallpox, typhoid, scarlet fever and tuberculosis [26]. On US scans, it appears as a complex, heterogeneous fluid collection, with shaggy, irregular walls, intratesticular location, low-level internal echoes, and, occasionally, hypervascular margins [39] (Figs. 2.38 and 2.39). Gas may be present, causing bright specular reflectors and shadowing [38]. It may rupture the tunica vaginalis and produce a fistulous formation on the overlying scrotal skin, especially likely with tuberculous epididymo-orchitis. Sometimes a chronic or partially treated abscess may be difficult to distinguish clinically and sonographically from a testicular tumour.

Lymphoma can also mimic a testicular abscess [26]. Patients present with an acutely painful, swollen scrotum and often have an elevated white blood cell count and fever. They frequently have a history of diabetes, human immunodeficiency virus infection or other immunosuppressive conditions. A scrotal abscess may be further complicated by a potentially life-threatening necrotising infection of the perineum, **Fournier's gangrene** (Fig. 2.40) [40]. Aetiological agents include *Clostridium* bacteria as well as other gas-forming bacteria and anaerobes. Fournier's gangrene is a urologic emergency, and aggressive treatment with surgical debridement and antibiotics is necessary. Some patients may require a suprapubic cystostomy if there is significant penile involvement or a diverting colostomy for rectal involvement [38].

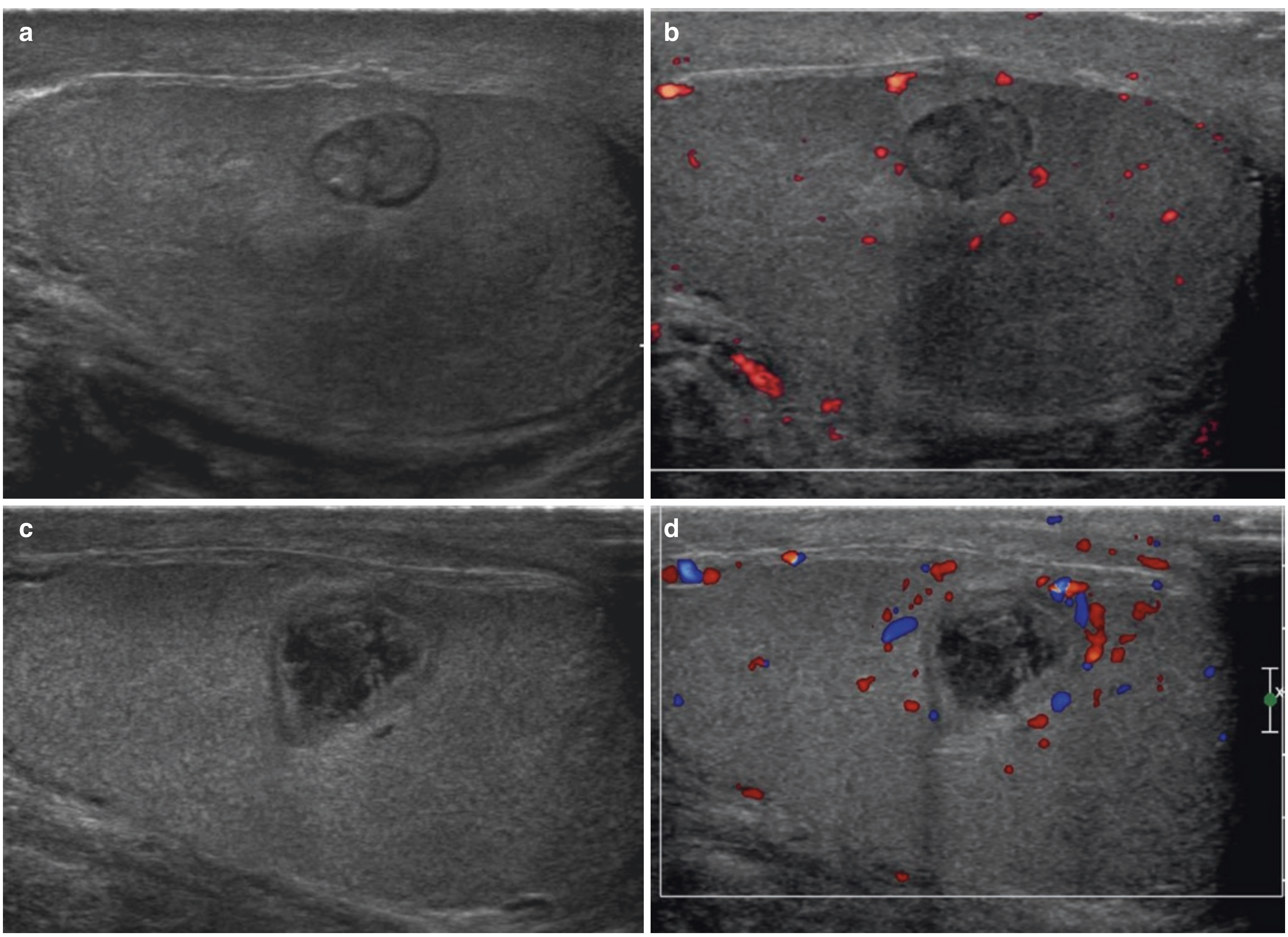

Fig. 2.37 Testicular abscess. A focal lesion with internal echoes developed in a patient after a testicular biopsy (**a**). Colour Doppler image demonstrating absence of flow in the abscess (**b**). The same abscess 1 month later (**c**, **d**)

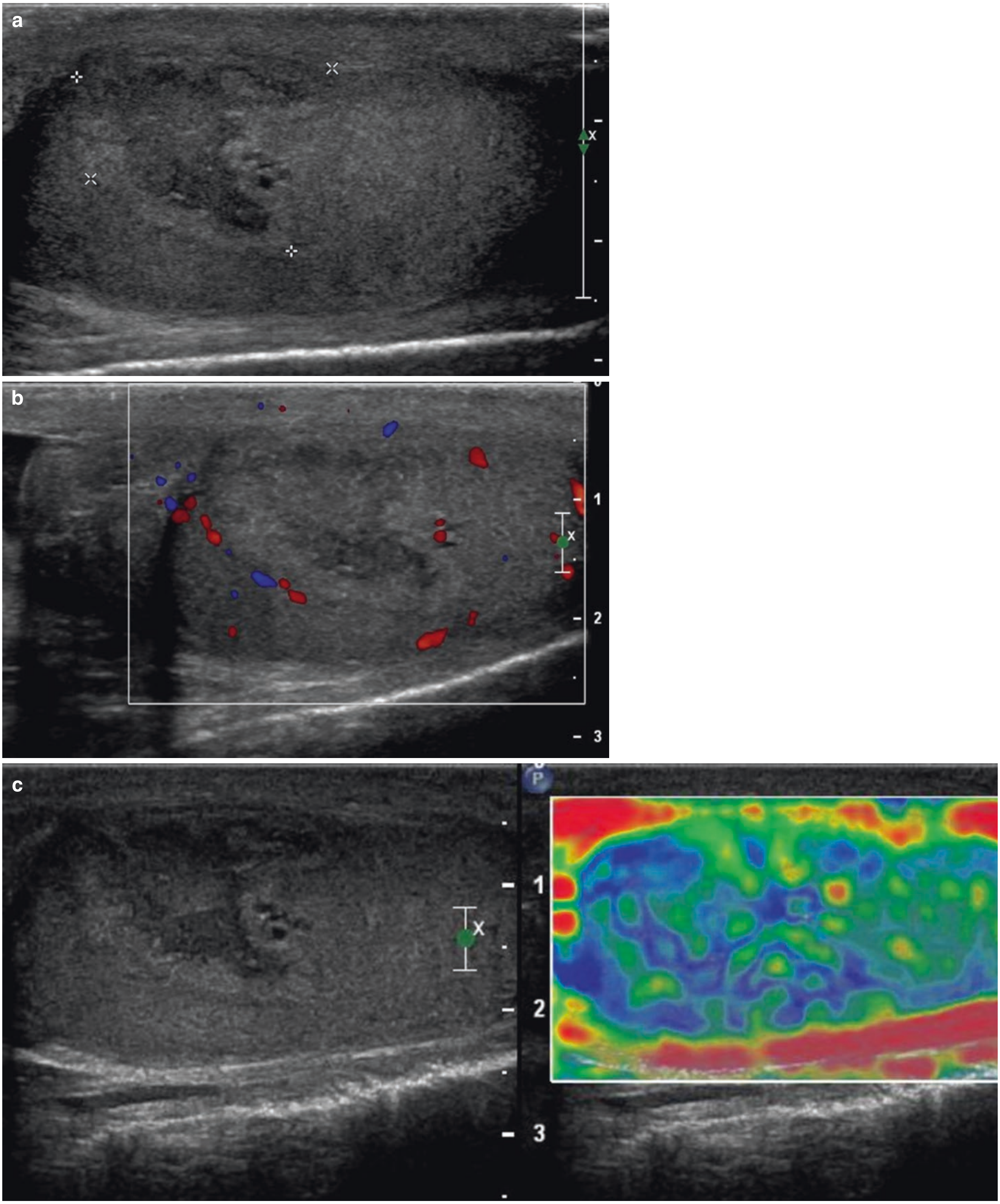

Fig. 2.38 Testicular abscess. Longitudinal greyscale US shows an intratesticular complex lesion with shaggy margins (**a**), avascular at colour Doppler (**b**). Elastography showed low elasticity of the lesion (**c**), strongly suggesting the presence of an intratesticular abscess after testicular injury

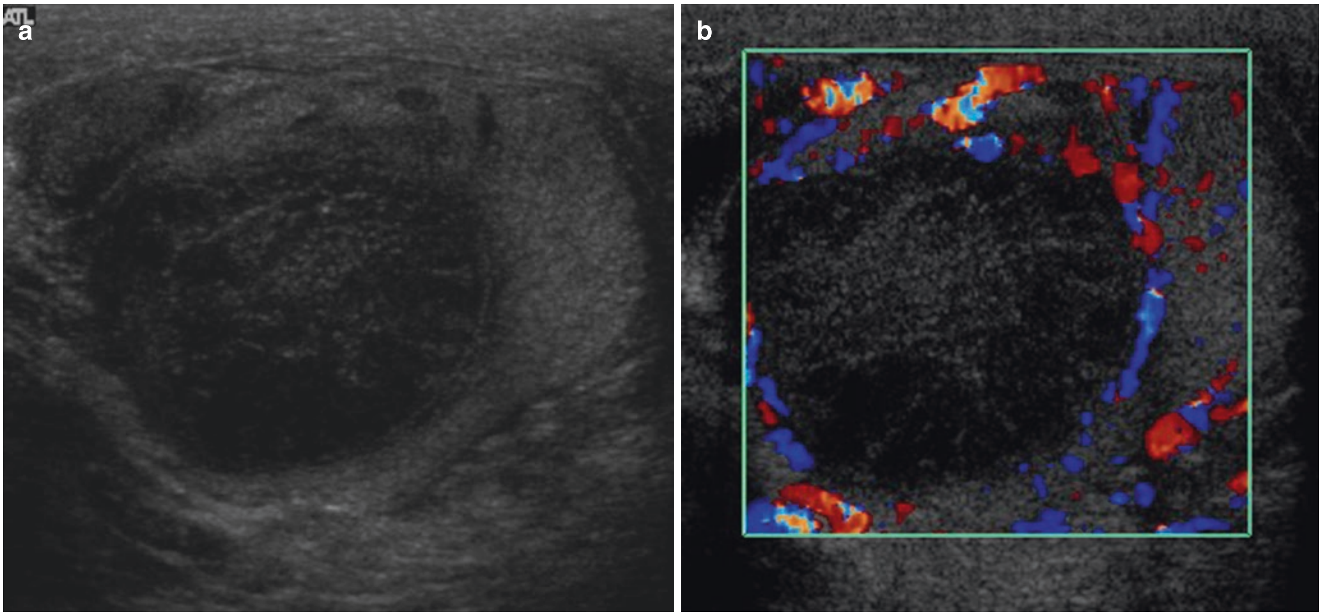

Fig. 2.39 Testicular abscess. Testicular abscess in a diabetic patient in his early 70s with acute and chronic epididymo-orchitis. There is a heterogeneously hypoechoic complex collection within an enlarged left testicle (**a**). Colour Doppler reveals hypervascularity of the testicle with absence of flow within the sonolucent area of necrosis (**b**) *(From: A.F. Wittenberg, T. Tobias, M. Rzeszotarski et al., "Sonography of the Acute Scrotum: The Four T's of Testicular Imaging" Curr Probl Diagn Radiol 2006;35:12–21)*

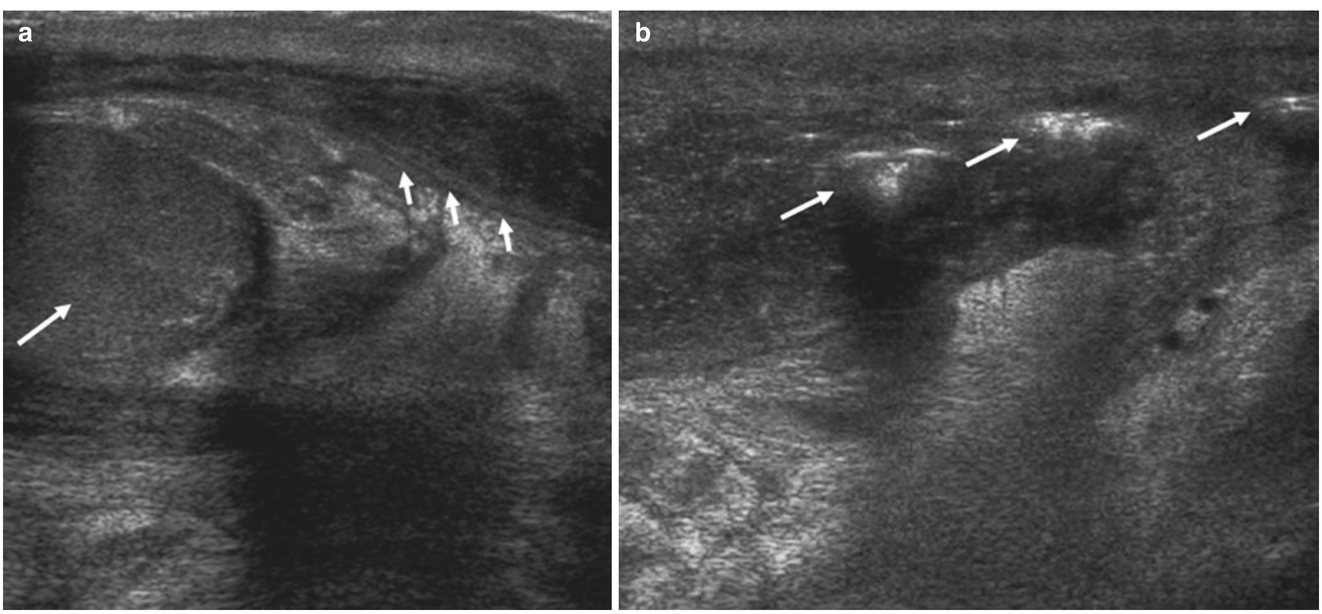

Fig. 2.40 Fournier's gangrene. Marked scrotal soft tissue thickening, with a pocket of low-reflective fluid (*short arrows*) surrounding a normal testis (*long arrow*) (**a**). Multiple areas of high reflectivity (*long arrows*) causing the characteristic 'dirty shadowing' of Fournier's gangrene (**b**) (*From: V.R. Stewart, P.S. Sidhu, "The testis: the unusual, the rare and the bizarre" Clinical Radiology (2007) 62, 289–302)*

2.5.2 Adrenal Rest

Ectopic adrenal rests occur in the testes and are thought to arise from aberrant adrenal cortical cells, which may migrate to gonadal tissues in the foetus. In patients with congenital adrenal hyperplasia, glucocorticoid synthesis is defective, and as a result these aberrant testicular rests fail to involute in the presence of a chronically elevated adrenocorticotrophic hormone [22]. They can be found in Addison's disease, Cushing's syndrome and adrenogenital syndrome. They also occur in congenital adrenal hyperplasia, which is an autosomal recessive disorder caused by the absence of an enzyme of the adrenal cortex. In the appropriate clinical setting, these masses need no further workup, but they do need to be distinguished from Leydig cell tumours, of which 10% are malignant [26]. In fact, they contain large cells that can resemble Leydig cells, with abundant eosinophilic cytoplasm but lacking Reinke crystalloids [41].

Testicular adrenal rests are usually less than 5 mm and are found in the testis and surrounding tissues in 7.5–15% of neonates and 1.6% of adults [41]. On US, adrenal rest tumours present as rounded hypoechoic solid masses, which may be bilateral and multifocal in origin (Figs. 2.41 and 2.42). They usually originate in the hilar region of the testicle and extend peripherally, so that they are located eccentrically in the testis.

The ultrasound features of adrenal rest tumours are well recognised [42]. Focal areas of echogenicity may be present due to fibrosis. Colour Doppler sonography may show these masses to be hyper-, normo- or hypovascular. The vessels coursing through the lesions are not deviated and are considered as useful features to differentiate adrenal rests from other testicular tumours (Fig. 2.43) [41, 43]. The intratesticular nodules of adrenal rests can gradually expand and destroy the testicular parenchyma, resulting in low testosterone production and infertility. The time from the initial detection of testicular abnormalities by US examination until documented infertility can range from 0 to 10 years [44]. These lesions are not premalignant and tend to regress with hormone replacement and hence do not require orchiectomy as the initial form of therapy. A marked reduction or disappearance of the lesions with therapy confirms the diagnosis [45].

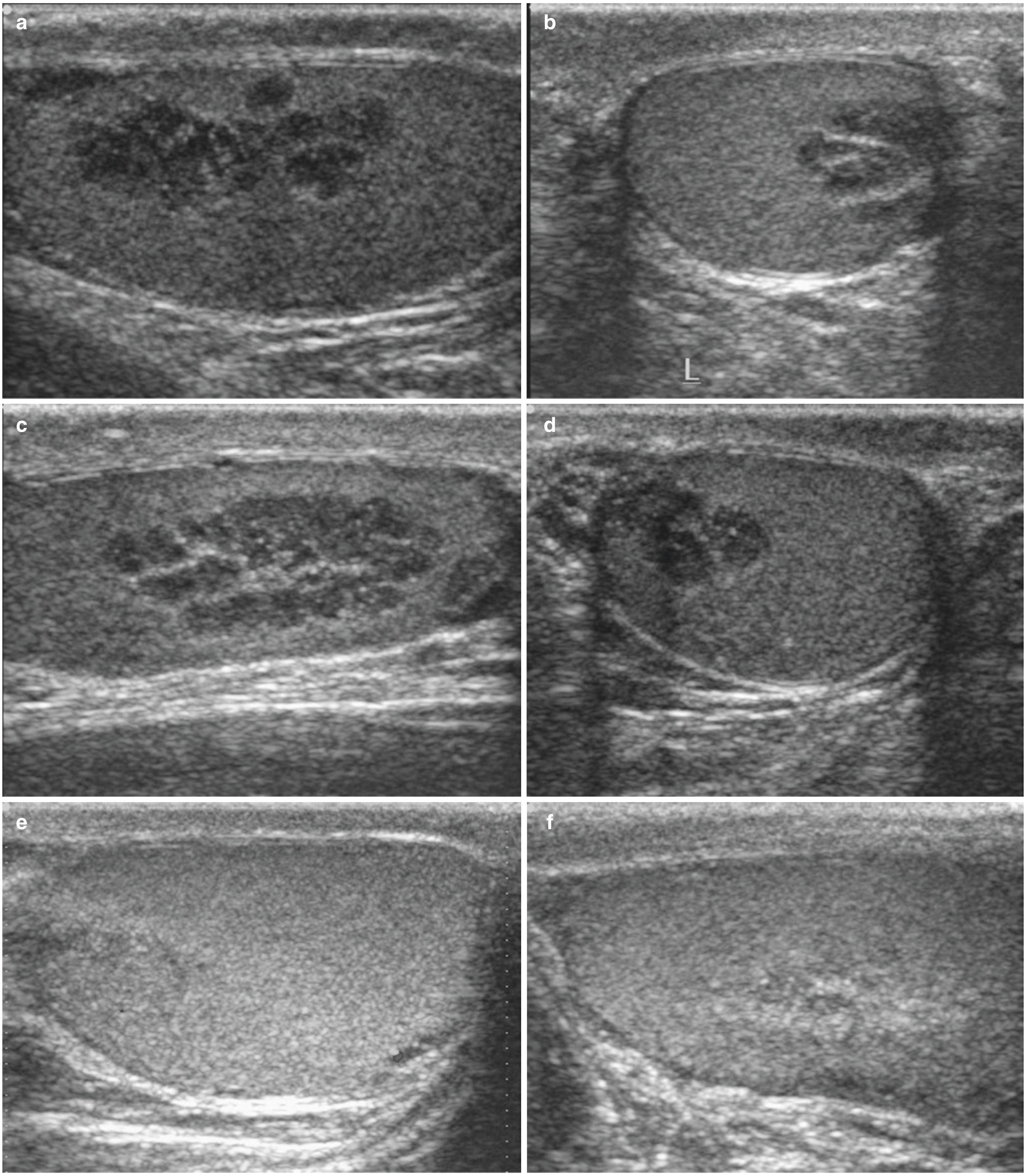

Fig. 2.41 Congenital testicular adrenal rests. Longitudinal and transverse greyscale sonogram of both testes (**a**–**d**) shows hypoechoic intratesticular mass in the region of the mediastinum testis in a 29-year-old man with confirmed diagnosis of CAH. In panels (**e**, **f**), the testes are shown after glucocorticoid therapy (*Courtesy of R.H. Oyen, MD)*

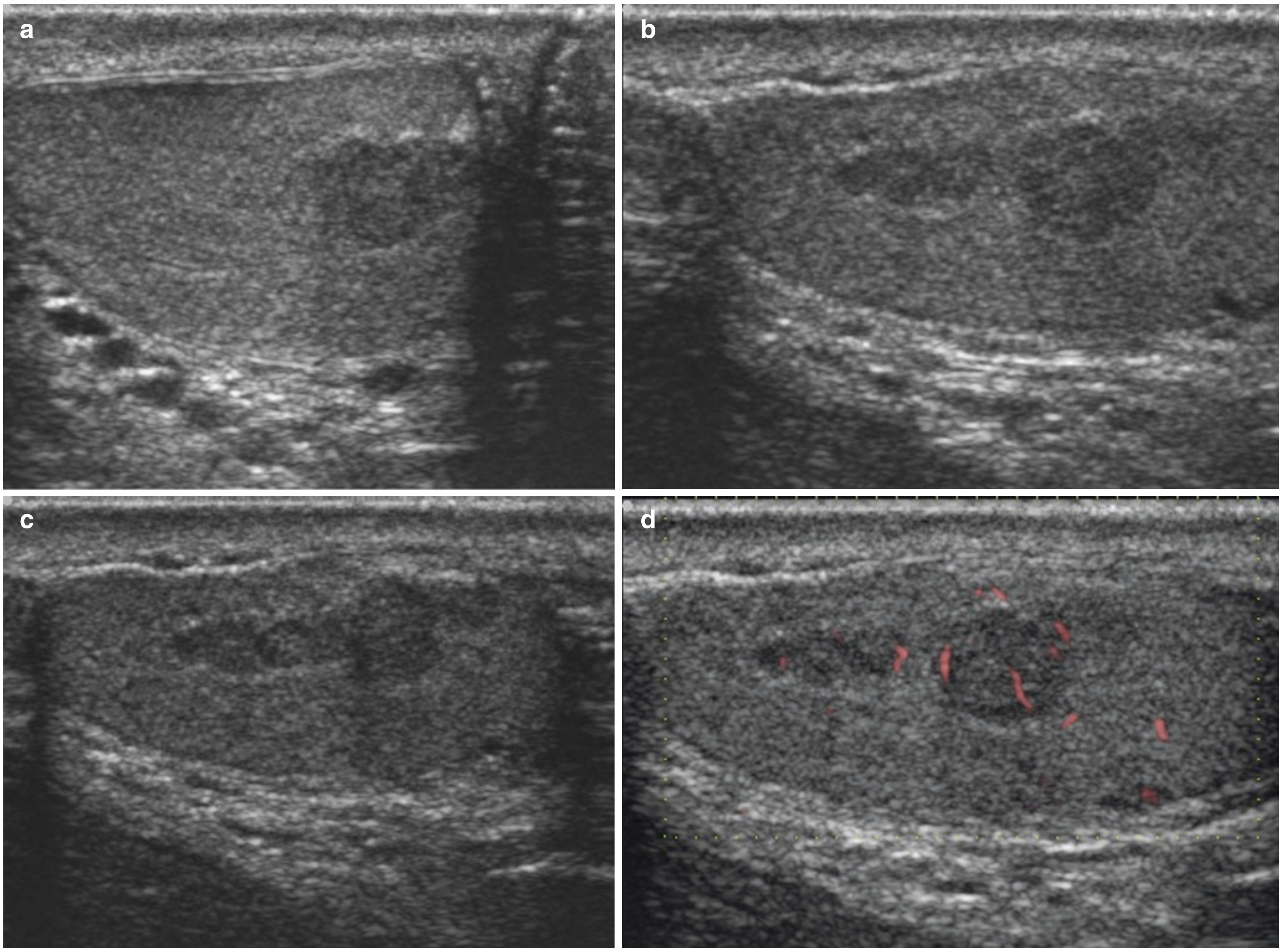

Fig. 2.42 Congenital testicular adrenal rests. Scrotal lesions associated with congenital adrenal hyperplasia. Transverse and longitudinal US scan shows a hypoechoic mass with lobulated margins (**a**–**d**) (*Courtesy of R.H. Oyen, MD)*

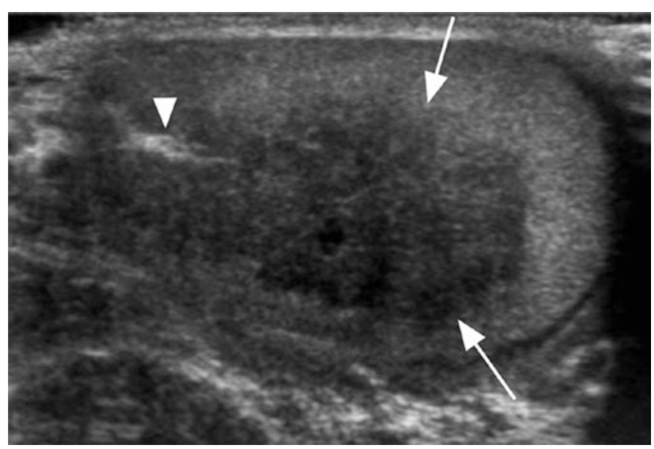

Fig. 2.43 Congenital testicular adrenal rests. Congenital adrenal hyperplasia in a 23-year-old patient with poor hormonal control. The lobulated, moderately well-defined, hypoechoic lesion (*arrows*) surrounds and does not markedly distort the mediastinum testis (*arrowhead*), a characteristic appearance (*From: A.P.S. Kirkham, P. Kumar, S. Minhas et al. "Targeted testicular excision biopsy: when and how should we try to avoid radical orchidectomy?" Clinical Radiology (2009) 64, 1158–1165)*

2.5.3 Sarcoidosis

Sarcoidosis is a multisystemic inflammatory disease of unknown origin involving the formation of non-caseating granulomas, the pathologic hallmark of this condition [7]. Sarcoidosis may involve the epididymis or the testis. Genital involvement occurs in less than 1% of patients with systemic sarcoidosis [40]. The clinical presentation is one of acute or recurrent epididymitis or of painless enlargement of the testis or epididymis. On US, sarcoidosis appears as hypoechoic, irregular or rounded masses in the epididymis, testis or both (Fig. 2.44) [46], more often unilateral than bilateral. Occasionally, hyperechoic calcific foci with acoustic shadowing may be seen. Distinguishing sarcoidosis from an inflammatory process or a neoplasm is difficult by ultrasound alone (Fig. 2.45) [40]. Knowledge that the patient has sarcoidosis is vital to the diagnosis of testicular involvement [47].

Fig. 2.44 Testicular sarcoidosis. Longitudinal greyscale sonograms of the testis in a patient with confirmed sarcoidosis show multiple hypoechoic areas within the testis *(From: D.C. Howlett, N.D.P. Marchbank and D.F. Sallomi. "Ultrasound of the Testis Pictorial Review", Clinical Radiology (2000) 55(8), 595–601)*

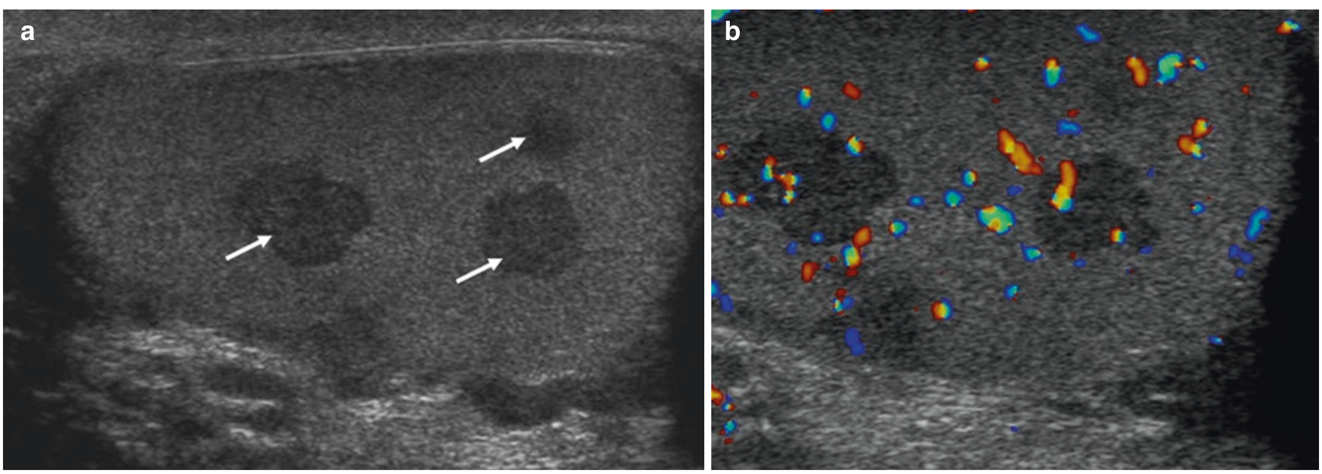

Fig. 2.45 Testicular sarcoidosis. Three low-reflective (*arrows*) focal testicular lesions in a patient with a recent diagnosis of sarcoidosis from a skin lesion biopsy (**a**). Colour Doppler ultrasound of the focal lesions demonstrates some colour Doppler flow but not in keeping with that seen in primary tumours of the testis (**b**) *(From: V.R. Stewart, P.S. Sidhu, "The testis: the unusual, the rare and the bizarre" Clinical Radiology (2007) 62, 289–302)*

2.5.4 Atrophy

Atrophy may produce strong changes in normal testicular echogenicity, often resulting in multiple irregularly shaped hypoechoic areas with disorganised vascularisation, which can resemble tumours. The most helpful distinctive feature of atrophy in contrast with tumour is the reduced size of the testis (Fig. 2.46). However, the problem is that dysplastic testes have a similar appearance and are at increased risk of developing a germ cell tumour.

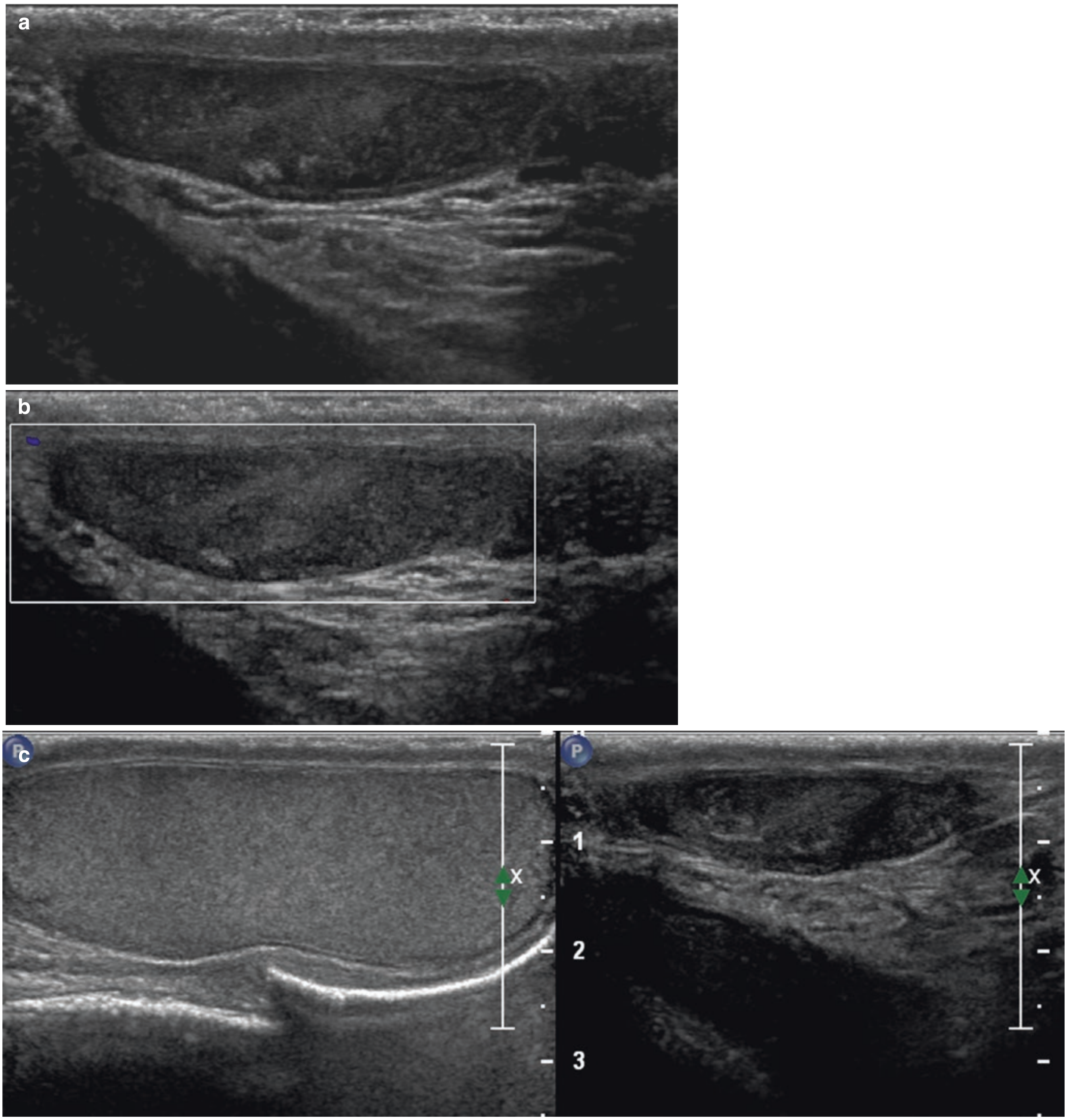

Fig. 2.46 Testicular atrophy. US scan of an atrophic testis (*right panel*), characterised by loss of volume and fibrosis, avascularity and a heterogeneous pattern on ultrasound

2.5.5 Orchitis

Epididymo-orchitis can occasionally produce focal changes in the testis that resemble a tumour. However, although clinical history may be helpful in the differential diagnosis, a number of cases of germ cell tumour have been reported in patients with a previous history of epididymo-orchitis, so this should not be ruled. Furthermore, cases of tumours causing orchitis by obstructing the seminiferous tubules have also been reported.

Orchitis most frequently produces hypoechoic changes in the texture of the testis, rather than discrete masses (Fig. 2.47). Clinical examination, which often reveals reduced testis consistency, and repeated semen analysis and hormonal evaluation, along with follow-up scans, are fundamental in most cases.

2.5.6 Post-biopsy Scars

Surgical manipulation, orchidopexy and biopsies can all leave scars on the testis, seen in two thirds of cases as localised alterations in parenchymal echogenicity. **Biopsies** are normally seen as avascular oval or triangular hypoechoic areas beneath the albuginea (Fig. 2.48). The elongated rather than rounded shape of these small hypoechoic areas, their hypo-reflectivity with low-contrast, irregular margins and, obviously, the patient's clinical history point to the benign nature of these focal lesions, frequently found on routine scans in the infertility clinic. Nevertheless, we strongly recommend that the size and location of these findings is described for future follow-up.

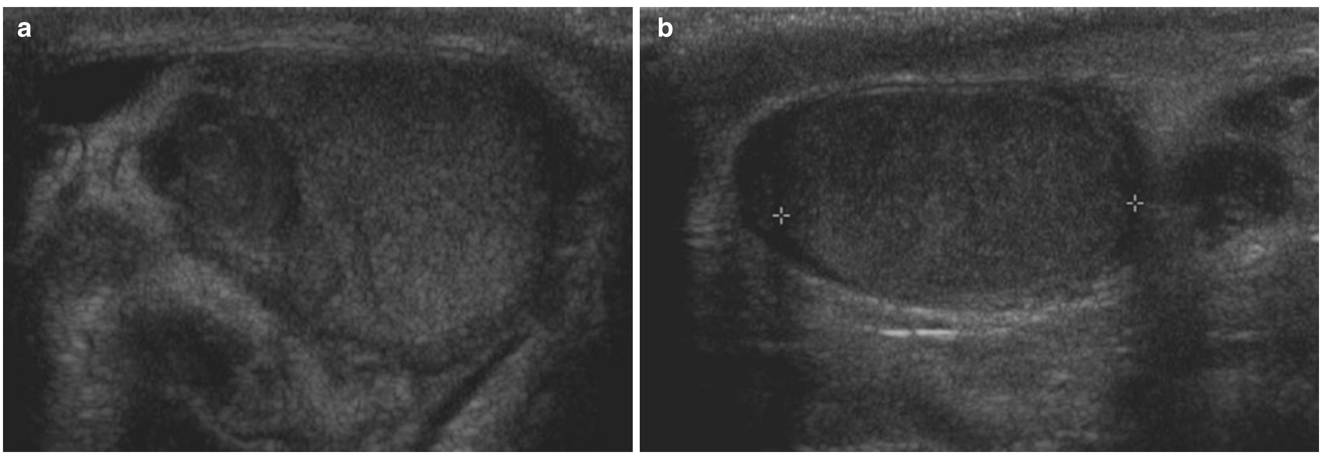

Fig. 2.47 Testicular orchitis. In this patient with a history of untreated orchitis, (**a**, **b**) the left testicle is small and demonstrates hypoechoic area of decreased echogenicity

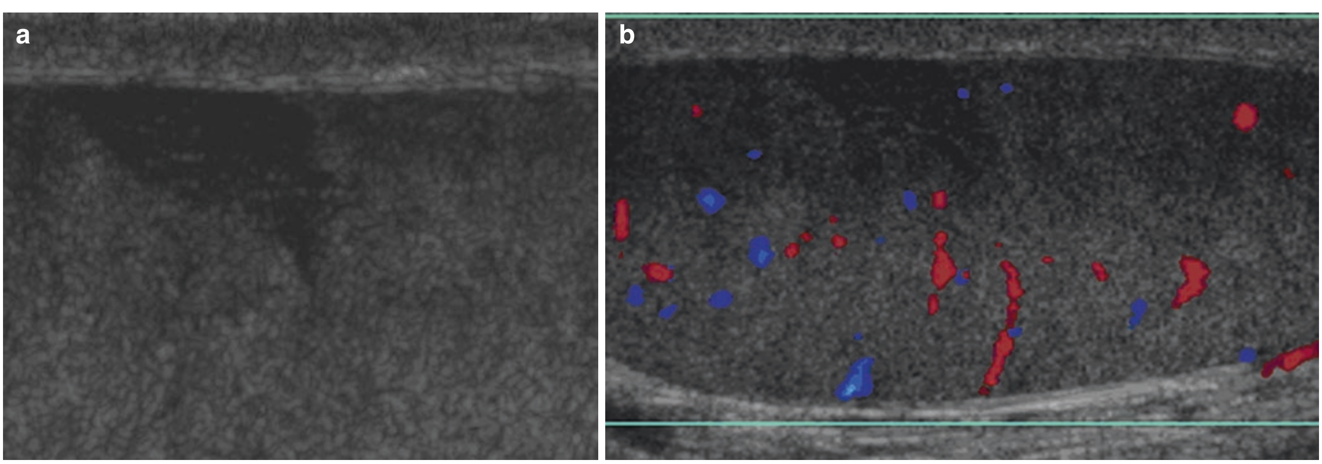

Fig. 2.48 Testicular biopsy. Longitudinal scan shows in the periphery of the testis a cuneiform (**a**), avascular area (**b**), sign of a precedent scrotal biopsy

2.5.7 Intratesticular Varicocele

Rarely, varicocele extends into the testis, presenting multiple cyst-like structures originating at the hilum and extending for a variable depth into the testis. The nature of these lesions is readily demonstrated with colour Doppler (Fig. 2.13) [see Chap. 6 on varicocele].

2.5.8 Fibrosis of the Tunica Albuginea

Small fibrotic foci of the tunica albuginea may develop secondary to inflammation or trauma. They are palpated as small, circumscribed, firm masses. Clinically, fibrotic foci cannot be distinguished from subcapsular tumours. Ultrasound may be silent and the scan may fail to reveal focal or diffuse thickening of the tunica, clinically detected. Plaque-like calcifications of the tunica can be seen as hyperechoic circumscribed lesions, possibly associated with acoustic shadowing. Additional scanning of the testis from the opposite side helps evaluate the testicular tissue behind the calcified area, to rule out a focal tumorous lesion or a burnt-out tumour (Fig. 2.24a).

2.5.9 Testicular Gummas

Testicular gummas are a rare tertiary manifestation of syphilis that develops 2–5 years after the initial infection. Clinical symptoms resemble those of malignant testicular tumours. History taking is fundamental. Patients will test positive for syphilis.

2.5.10 Splenogonadal Fusion

Splenogonadal fusion is a rare malformation where an accessory spleen exists within the scrotum or pelvis fused to the gonadal organs, most commonly on the left side and reported more frequently in males where the accessory spleen within the scrotal sac is easier to palpate [48]. Splenogonadal fusion presents as a painless lump and is associated with other congenital abnormalities, most commonly cryptorchidism and an inguinal hernia. The ultrasound appearances are of a homogeneous mass of slightly lower reflectivity compared to the testicular parenchyma, which may be difficult to separate from the testis. Colour Doppler may demonstrate a central vascular pattern branching to the periphery rather than the disorganised pattern seen in primary testicular malignancy (Fig. 2.49) [49]. The diagnosis of splenogonadal fusion is problematic, and operator awareness of the condition is crucial as the accessory spleen may be confused with a testicular tumour resulting in unnecessary orchidectomy. If the diagnosis is considered preoperatively, a 99mTc-sulphur colloid scan is diagnostic, demonstrating tracer uptake within the accessory spleen and splenic tissue [40, 48].

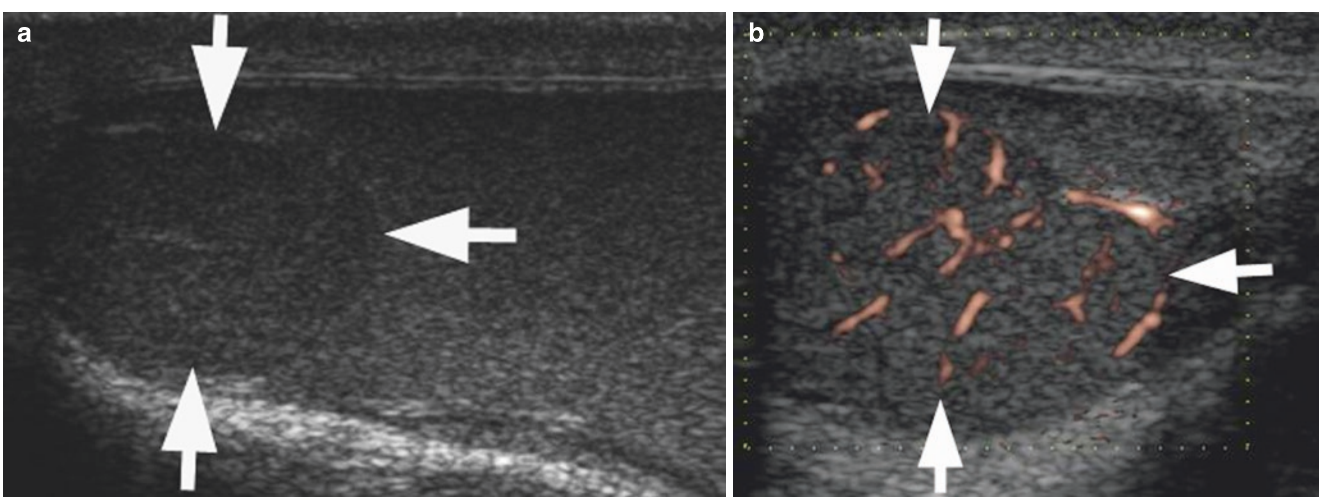

Fig. 2.49 Splenogonadal fusion. (**a**) Longitudinal greyscale sonogram of the testicle in a young adult patient showing a focal mass (*white arrows*) that is slightly hypoechoic compared to the background parenchyma. (**b**) Transverse colour Doppler image of the testicle in the same patient showing markedly increased blood flow within the hypoechoic testicular lesion compared to the adjacent parenchyma. Final pathologic examination on resection revealed splenogonadal fusion *(From: A.M. Basta, J. Courtier, A. Phelps, et al. "Scrotal Swelling in the Neonate" J Ultrasound Med (2015) 34:495–505)*

2.5.11 Intratesticular Haematoma

Testicular rupture, extratesticular haematoma and haematocele are the most common sequelae of testicular trauma. An uncommon finding is an isolated intratesticular haematoma. This has a variable appearance on ultrasound, with a change in its characteristics over time. A history of trauma to the scrotum should suggest the diagnosis, but this is not always forthcoming. Acutely, the haematoma appears as patchily increased reflectivity that becomes poorly reflective and decreases in size as the haematoma retracts, eventually resolving completely. The haematoma presents no colour Doppler flow, a low-reflective rim – thought to represent surrounding oedema – and internal echoes (Fig. 2.50). It is important to distinguish this condition from an intratesticular tumour; thus an accurate history and ultrasound examination are required to reach the correct diagnosis [40].

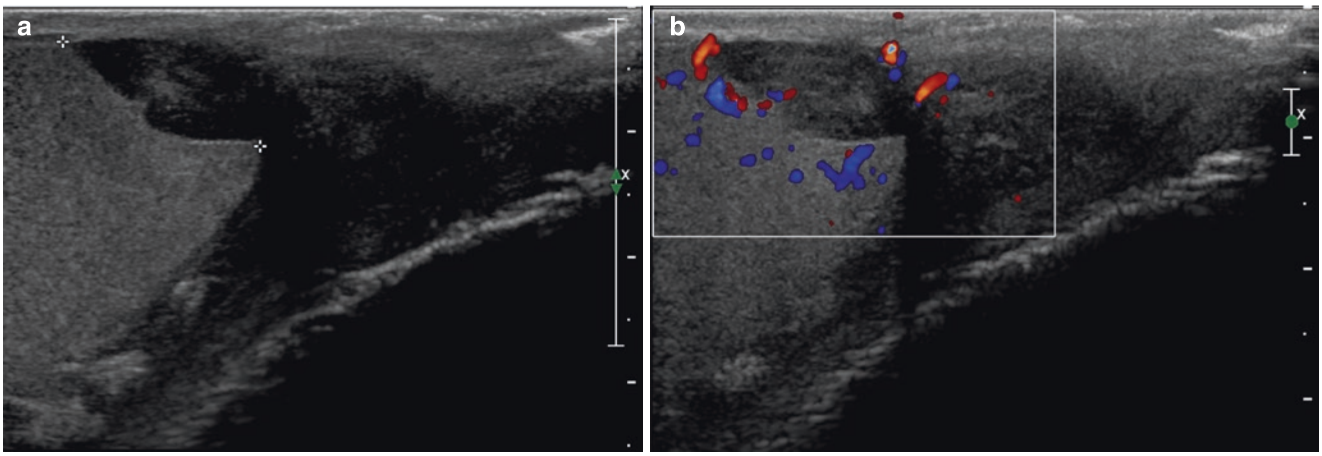

Fig. 2.50 Intratesticular haematoma. A well-circumscribed hypoechoic lesion in the right testis of a patient involved in a motorcycle accident. The lesion contains areas of high reflectivity (**a**). The use of colour Doppler ultrasound confirms the absence of intralesional vascularisation and thus the presence of traumatic intratesticular haematoma (**b**)

Key Messages

- Most testicular tumours present as a palpable mass, but not all palpable masses are malignant tumours, nor are all intratesticular lesions palpable.
- Intratesticular cysts are discovered by chance in 5–10% of ultrasound scans.
- Simple intratesticular cysts are sharply demarcated anechoic areas with a thin wall, posterior acoustic enhancement, no solid portion and no structural disturbance of the adjacent parenchyma.
- Intratesticular cysts that can be palpated as hard masses or contain solid elements should be treated as suspect.
- Albuginea cysts have a fairly characteristic feel on palpation as a firm pinhead-sized mass on the surface of the testis.
- A marginal age-related dilation of the rete testis is a common finding in patients over 50.
- Epidermoid cysts are benign testicular solid lesions presenting with testicular enlargement or firm, non-tender, frequently palpable lumps in the testis. Their typical appearance is the 'onion ring' pattern; however, atypical epidermoid cysts have been described.
- Solitary or isolated (fewer than five) intratesticular calcifications are a frequent, benign finding at routine scan. More than five microcalcifications are an indication for 12-month screening due to an as-yet undefined risk of developing testicular carcinoma.
- Segmental testicular infarction is a probably underestimated condition that can challenge non-experts in differential diagnosis against testicular tumour. The current trend is to treat these lesions conservatively or monitor them closely.
- Size and symmetry of the testes are both important. Any variation greater than 25% between the two testicular volumes should be reported.
- An accurate clinical history is vital in establishing the diagnosis of scrotal abscess, pyocele, sarcoidosis, testicular gummas or intratesticular haematomas. Colour Doppler study confirms the nature of these lesions.
- Adrenal rests become clinically relevant in the presence of chronic ACTH elevation.
- Knowledge of the clinical and ultrasound features of benign intratesticular lesions can save patients from unnecessary orchiectomy. Correct diagnosis obtained by combining the clinical and radiological findings – which may include repeated follow-up scans – could be achieved in a significantly greater number of cases than previously thought.

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3 Neoplastic Intratesticular Lesions

3.1 Introduction

Testicular cancer accounts for 4–6% of all tumours of the male urogenital tract and is the most common malignancy in the 15–35 age group. Testicular tumours can be categorised into germ cell (95%) and non-germ cell (5%) tumours. Germ cell tumours are almost always malignant. Non-germ cell tumours include tumours arising from the stromal cells, primary and secondary tumours from haematopoietic cells and, rarely, other metastatic tumours. About 5% of testicular tumours are benign, of which 90% are stromal cell tumours.

Malignant testicular germ cell tumours are divided into two groups on the basis of their clinical and biological behaviour: seminomatous (SGCT) and non-seminomatous tumours (NSGCT) (Table 3.1).

Ultrasound is the most common technique for evaluation of testicular tumours. MRI can offer additional diagnostic information in differentiating selected cases, while CT is mainly used for staging purposes. No imaging technique can provide a histological diagnosis. For these reasons, malignant and benign tumours – whose ultrasound features may overlap – are presented together in this chapter.

Improved diagnostic techniques and frequent testicular scanning for infertility have drawn attention to a significant number of small, solid and often non-palpable tumours whose diagnosis and management is a significant problem for the andrologist. Recent studies show that many of these incidentally discovered lesions turn out to be non-germ cell tumours [1]. Management options range from orchiectomy to simple excision, frozen section or active surveillance. A thorough clinical history and physical examination combined with accurate, high-resolution scanning enable the best option to be selected in each case. A selection of ultrasound and clinical features that help distinguish among these lesions is reported at the end of this chapter.

Age distribution for testicular cancer is fairly typical. The peak incidence of testicular cancer is in the 25–35 age group [2]; however, some types of tumours – lymphoma, spermatocytic tumour and metastasis – occur later in life [3–22]. There is a third much less pronounced peak in children, due to yolk sac tumours and teratomas. Testicular cancer is more frequent in Caucasians, and its incidence is reported to increase in the last decades of life. The **established risk factors** for testicular carcinoma are prior testicular tumour, cryptorchidism, positive family history, infertility and intersex syndromes. Some authors suggest that the higher incidence of testicular tumours parallels the decline in semen quality and fertility observed in western countries.

It is widely accepted that the syndrome of **testicular dysgenesis** is a risk factor for testicular cancer [10]. Data show that the increased risk for testicular carcinoma is not limited to the cryptorchid testis, but extends to the contralateral testis, even if normally descended. Similarly, the risk is correlated with the severity of cryptorchidism, as if it were the consequence of a greater degree of defective embryogenesis. However, some studies reveal a rising number of testicular cancers in patients with normal seminal parameters [11]. Despite the increase in cases, survival rates for patients with testicular carcinoma have increased to over 90–95% in recent decades.

Most testicular cancers are detected by chance by the patient, as a lump or **painless swelling** of the testis. Approximately 1/3 of patients complain of a sensation of heaviness or fullness in the lower abdomen or scrotum. A **dull or heavy pain** is reported in 20% of cases, confirming that the presence of pain does not exclude the diagnosis of testicular cancer. In approximately 10% of patients, testicular cancer is associated with acute pain, usually caused by haemorrhage or infarction, mimicking torsion or epididymitis. As epididymo-orchitis and germ cell cancer may coexist by chance or due to the tumour's obstruction of the seminiferous tubules, differential diagnosis based solely on clinical grounds can be misleading. Frequently, the only symptom reported by the patient is a vague discomfort in the scrotum. Only a minority (<6%) present with symptoms of metastatic disease.

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Table 3.1 Classification of testicular tumours

Classification
Germ cell tumours derived from germ cell neoplasia in situ
Non-invasive germ cell neoplasia
Germ cell neoplasia in situ
Specific forms of intratubular germ cell neoplasia
Tumours of a single histological type (pure forms)
• Seminoma (typical, anaplastic)
• Embryonal carcinoma
• Teratoma (post-pubertal type, with somatic-type malignancy)
• Choriocarcinoma
• Non-choriocarcinomatous trophoblastic tumours
• Yolk sac tumour (post-pubertal type)
Tumours of more than one histological type
• Mixed germ cell tumours
Germ cell tumours of unknown type
• Regressed germ cell tumours
Germ cell tumours unrelated to germ cell neoplasia in situ
• Spermatocytic tumour
• Teratoma, prepubertal type (dermoid cyst, epidermoid cyst, well-differentiated neuroendocrine tumour, yolk sac tumour prepubertal type)
• Mixed teratoma and yolk sac tumour, prepubertal type
Sex cord-stromal tumours
• Leydig cell tumour
• Malignant Leydig cell tumour
• Sertoli cell tumour (large-cell calcifying and sclerosing Sertoli cell tumour, malignant Sertoli cell tumour)
• Granulosa cell tumour (adult, juvenile)
• Fibroma-thecoma
Mixed germ cell and stromal tumours
• Gonadoblastoma
Lymphoid and haematopoietic tumours
• Diffuse large B-cell lymphoma
• Follicular lymphoma
• Leukaemia/plasmacytoma
Miscellaneous
• Carcinoid (isolated, associated to teratoma)
• Ovarian epithelial-type tumours
Tumours of collecting duct and rete testis
• Adenoma, adenocarcinoma
Secondary tumours (metastasis)
• Lymphoma, leukaemia, myeloma
• Melanoma
• Lung, prostate, kidney, gastrointestinal tract

Tumours can undergo regression, necrosis and scarring (so-called burnt-out germ cell tumours), and therefore some patients may present a normal or small testis, while having a distant metastasis. Hormonally active tumours may present with **endocrine abnormalities** and related symptoms, most commonly gynaecomastia.

Patients with cryptorchidism are 2.5–8 times more likely to develop testicular cancer [12]. The risk is even higher in boys treated after puberty. An increased risk has also been reported for men with Klinefelter's syndrome and gonadal dysgenesis [23].

The main role of US examination in the diagnosis of testicular cancer is to help distinguish intratesticular from extratesticular lesions, as most extratesticular masses are benign while intratesticular masses are more likely to be malignant [13].

Intratesticular masses are categorised as solid, cystic or mixed lesions. Cystic lesions are generally benign, while solid and mixed lesions, with few exceptions (see Chap. 2), should be considered neoplastic. Their appearance on US reflects their gross morphology and underlying histological characteristics. Most malignant testicular neoplasms are hypoechoic to the normal surrounding testicular parenchyma. However, a variety of benign intratesticular processes, such as haematoma, orchitis, abscess, infarction and granuloma, can mimic testicular malignancy. Some tumours are heterogeneous and can be partially cystic. Focal changes within tumours – such as haemorrhage, necrosis, calcification or fatty changes – all produce areas of increased echogenicity and confer a heterogeneous appearance to these lesions. In a hypoechoic testis, such as a prepubertal or atrophic testis, tumours can be isoechoic to surrounding parenchyma. In these cases, colour Doppler ultrasound (CDU) can be helpful.

Overall, the accuracy of ultrasound in detecting and distinguishing intratesticular and extratesticular lesions ranges from 98 to 100% [23].

There are few ultrasound features that can point to a differential diagnosis between SGCT and NSGCT, but exceptions to the typical appearance in these tumours are common. In general, seminomatous tumours are mostly homogeneous and more hypoechoic, whereas non-seminomatous tumours are heterogeneous even when small and frequently show cystic or calcified parts with alternating hypo- and hyperechoic areas. Both SGCT and NSGCT tend to have neat margins. However, large seminomatous tumours can appear non-uniform, and mixed tumours can occur presenting with polycyclic borders.

Lymphoma and leukaemia can be associated with aspecific ultrasound features, ranging from discrete nodules to ill-defined lesions infiltrating the testis multifocally or diffusely. The typical features of the various histotypes are described below, although none can be considered pathognomonic. Diagnosis of the nature of intratesticular tumours is therefore exclusively histological, and the features described herein represent only the most common US findings and do not claim to represent all possible appearances of these lesions.

The use of CDU and power Doppler US is crucial in the evaluation of any intratesticular lesion. Flow studies demonstrate increased vascularity in most malignant tumours and help to better define testicular involvement, margins, cystic degeneration and any necrotic areas of the lesion. However, the presence of hypervascularity is not specific enough for a diagnosis of malignancy (e.g. it is also seen in focal orchitis). It may also be difficult to demonstrate increased blood flow in small tumours or in the presence of calcification artefacts [23]. Moreover, increased or altered vascularisation can be found in both benign tumours and focal hyperplasia.

Having said that, some vascular images are suggestive of malignancies. For example, the arborisation of high-flow, low-resistance vascular branches within the lesion is frequently seen in seminoma.

An important adjuvant investigation in a suspicious testicular mass is the measurement of serum markers: βhCG, αFP, CEA and PLAP are most specific, while LDH and ferritin are less specific (Table 3.2).

Tumour markers also have a well-established role in the staging, prognosis and follow-up of GCT. Elevated αFP is seen in over 95% of children with yolk sac tumours or mixed germ cell tumours with a yolk sac component, while βhCG is significantly raised in nearly all patients with pure or mixed choriocarcinoma, where it reaches very high levels. To a lesser extent, a positive βhCG is also seen in seminomas. αFP elevation is also commonly found in patients with teratomas. Placental alkaline phosphatase (PLAP) may be of some value in monitoring patients with pure seminoma.

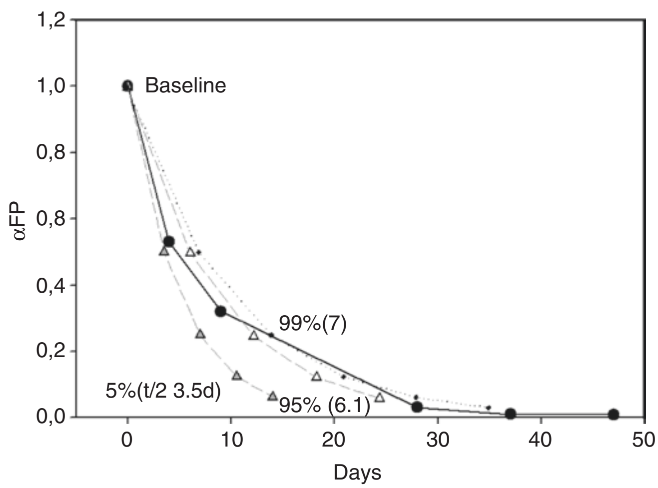
LDH is a much less specific marker; however, it has a direct correlation with most cancers and is useful for staging and prognosis. Ferritin elevation is occasionally observed in seminomas.

Detection of elevated tumour markers significantly affects the positive predictive value given to the ultrasound finding. However, malignant tumours with negative serum markers are encountered frequently, especially in seminomas, and a negative serological diagnosis does not affect the ultrasound diagnosis. Even in unequivocal cases, it is essential to measure tumour markers before surgical removal, in order to have the baseline value for calculation of the marker's clearance rate. Analysis of the curves of serum reduction curves for these markers is very helpful in predicting persistence of disease – e.g. the presence of distant metastasis – or recurrences during follow-up.

In Graph 3.1 the exponential decay of αFP in a patient with a baseline of 26.000 ng/mL is shown (solid circle). It is generally thought that if the decay curve falls between the 95% (open triangle) and 5% (grey triangle), the tumour has been completely removed. This result does not exclude repeated follow-up.

Table 3.2 Tumour markers in testicular cancer

Type	Specific	Less specific
Non-seminomatous	βhCG, αFP, CEA	LDH
Seminomatous	βhCG, PLAP	LDH, Ferritin



Graph 3.1 Exponential decay of αFP in a patient with a baseline value of 26.000 ng/ml is shown (*solid circle*)

3.2 Germ Cell Tumours

3.2.1 Seminomatous Germ Cell Tumours

Seminoma is the most common single-cell testicular tumour in adults and is generally seen in males aged 20–40, with a peak incidence in the fourth and fifth decades of life. Pure seminoma accounts for 35–55% of ger[24]. Seminoma is also a component of 30% of mixed germ cell tumours.

Between 8 and 30% of patients with seminoma have a history of undescended testes, and, due to their high incidence, seminomas are also the most common tumour type in cryptorchid testes (Fig. 3.1).

There are two subtypes: typical seminoma (90–95% of all seminomas) (Fig. 3.2) and anaplastic (5–10%) [23]. Typical seminoma is considered less aggressive than other testicular tumours, with less than 25% of patients presenting metastases at diagnosis. Of these, 20% have retroperitoneal adenopathy and 5% have extra-nodal metastases.

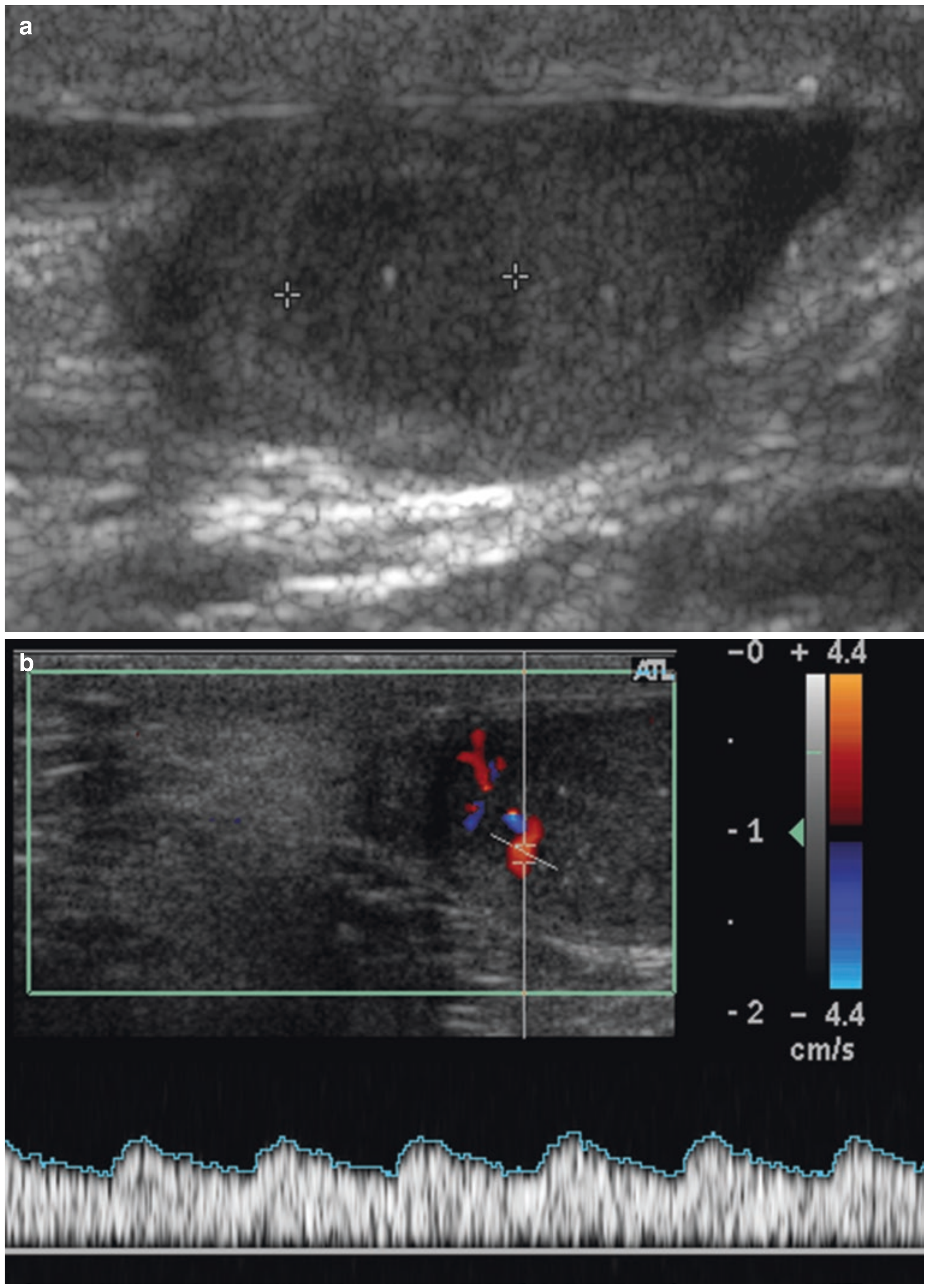
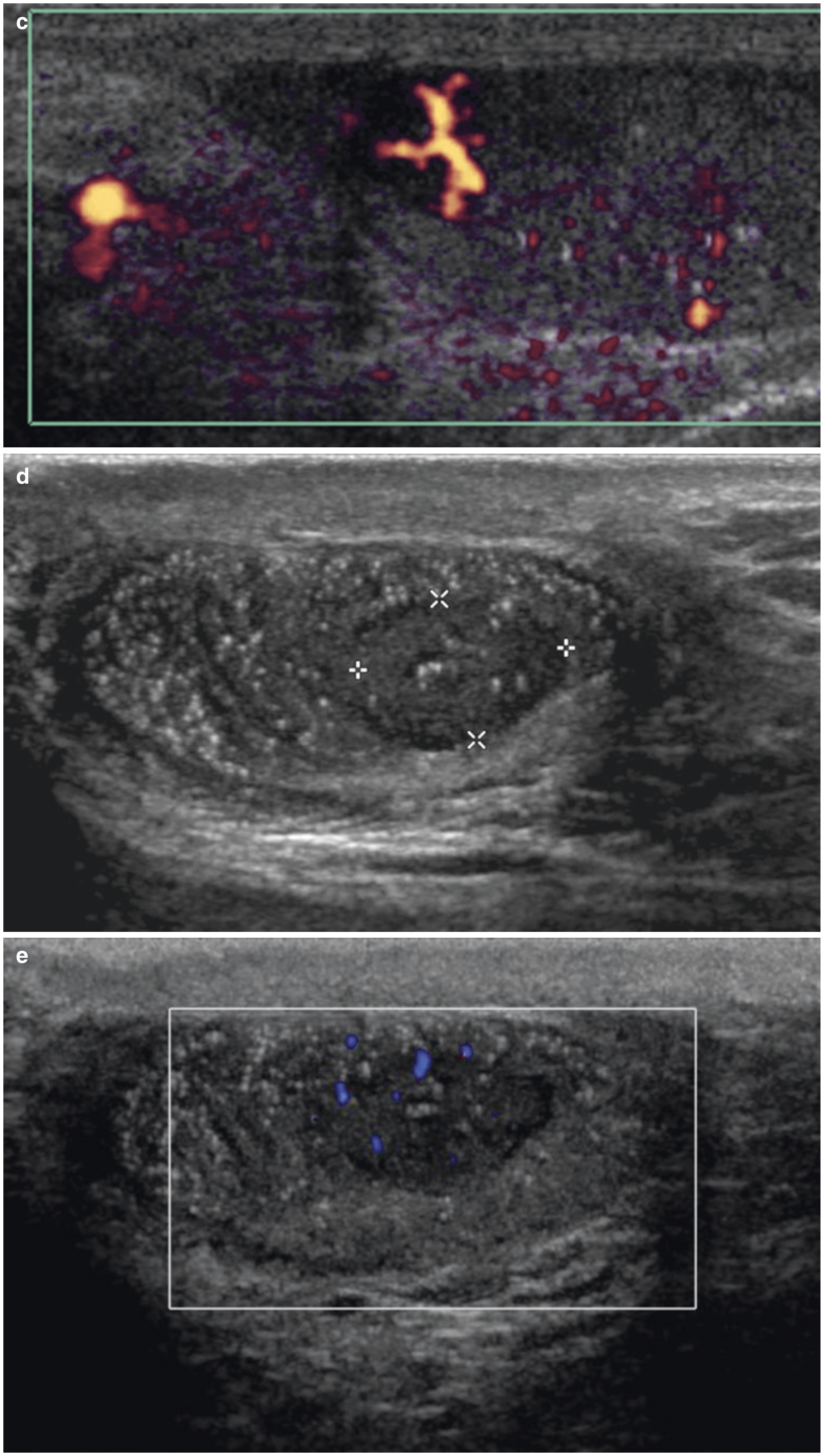


Fig. 3.1 Cryptorchidism and seminoma. Seminoma in a cryptorchid testis of a 28-year-old man treated with orchidopexy at the age of 8 (**a**). Colour and power Doppler study showing vascular branches within the lesion, characterised by low-resistance flows and arborisation (**b**, **c**). Seminoma in atrophic undescended testis. Ultrasound of the right groin demonstrates an atrophic, undescended testis, with massive microlithiasis and containing a rounded hypoechoic mass. The testis was excised, and histology confirmed the presence of seminoma (**d**, **e**)

Fig. 3.1 (continued)



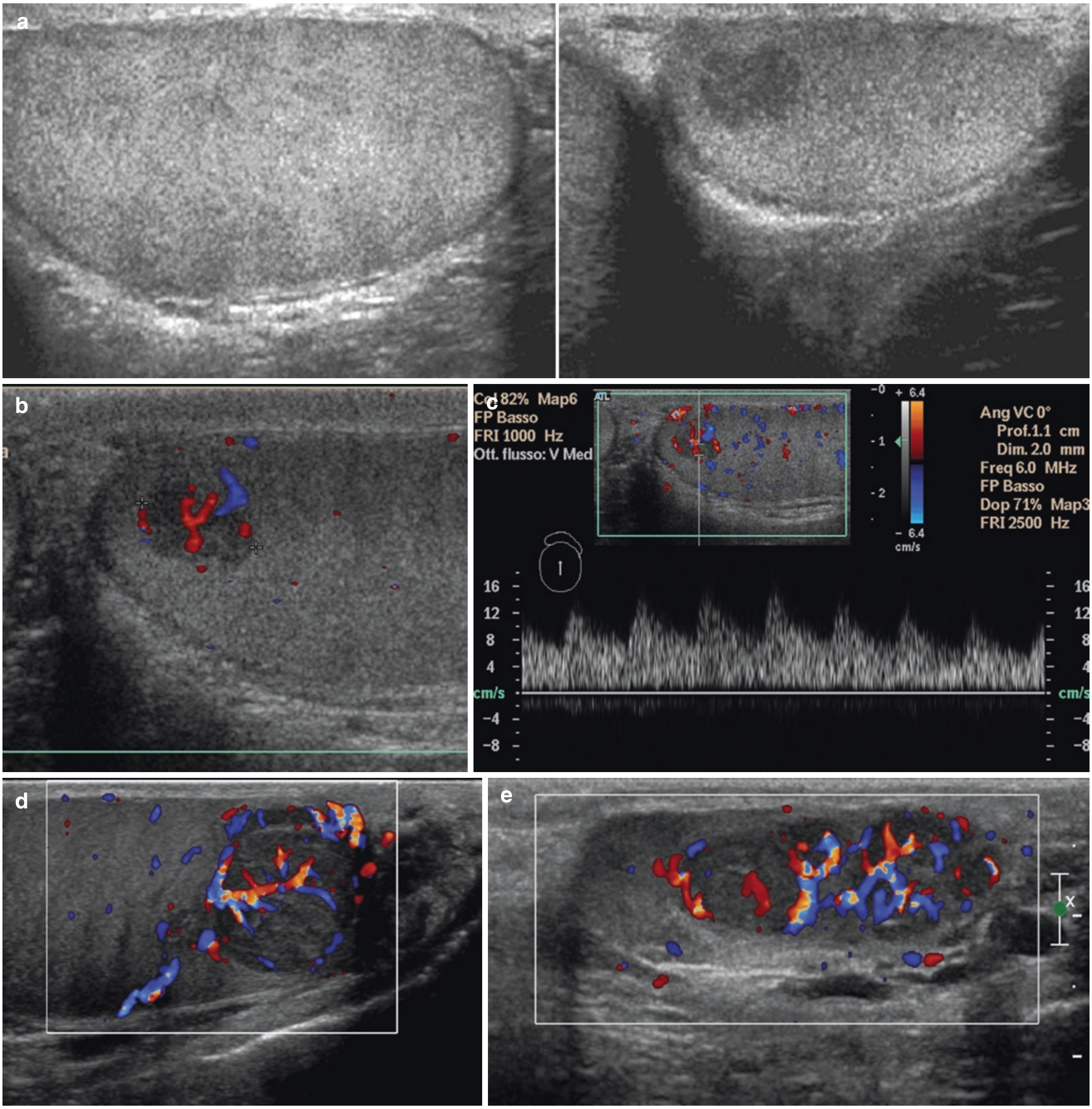


Fig. 3.2 Typical seminoma. Longitudinal scan demonstrates a small, well-defined and hypoechoic mass in the left testis (**a**). A typical finding is the arborisation (**b**, **d**, **e**) of high-flow, low-resistance vascular branches within the lesion (**c**). Surgery confirmed the presence of seminoma. In panel (**e**) intralesional arborisation of the vessels is shown

Macroscopically (Fig. 3.3), seminoma appears as a homogeneously solid, uniformly hypoechoic firm, round or oval tumour that varies in size (Fig. 3.4) from a small nodule in a normal-sized testis to a large mass totally replacing the testicle and causing diffuse testicular enlargement (Fig. 3.5).

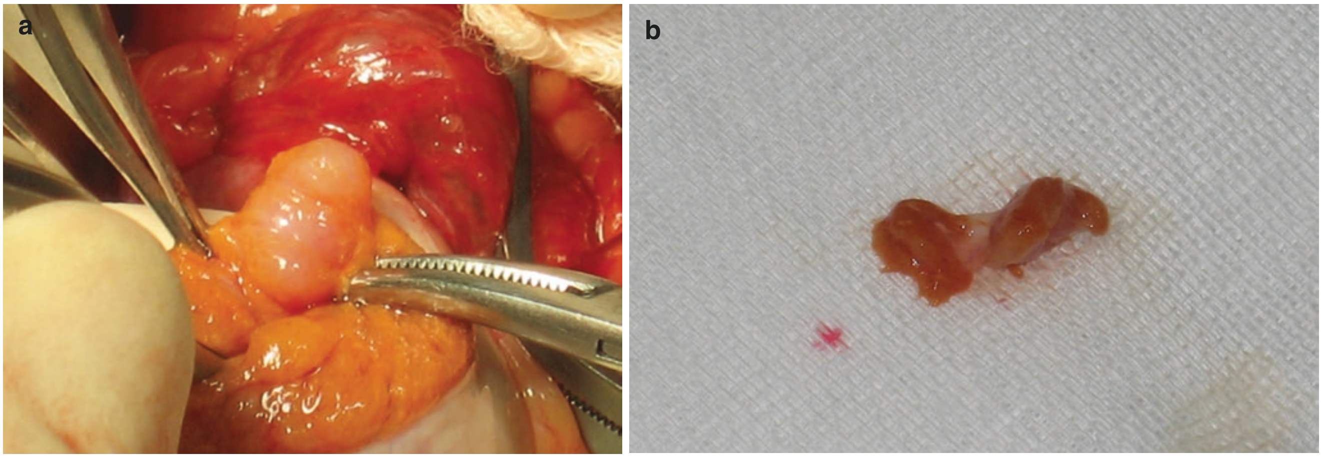


Fig. 3.3 Seminoma: macroscopy. Gross specimen of the tumour illustrated in Fig. 3.2, showing a well-circumscribed glistening tumour, solid and firm (**a**, **b**)

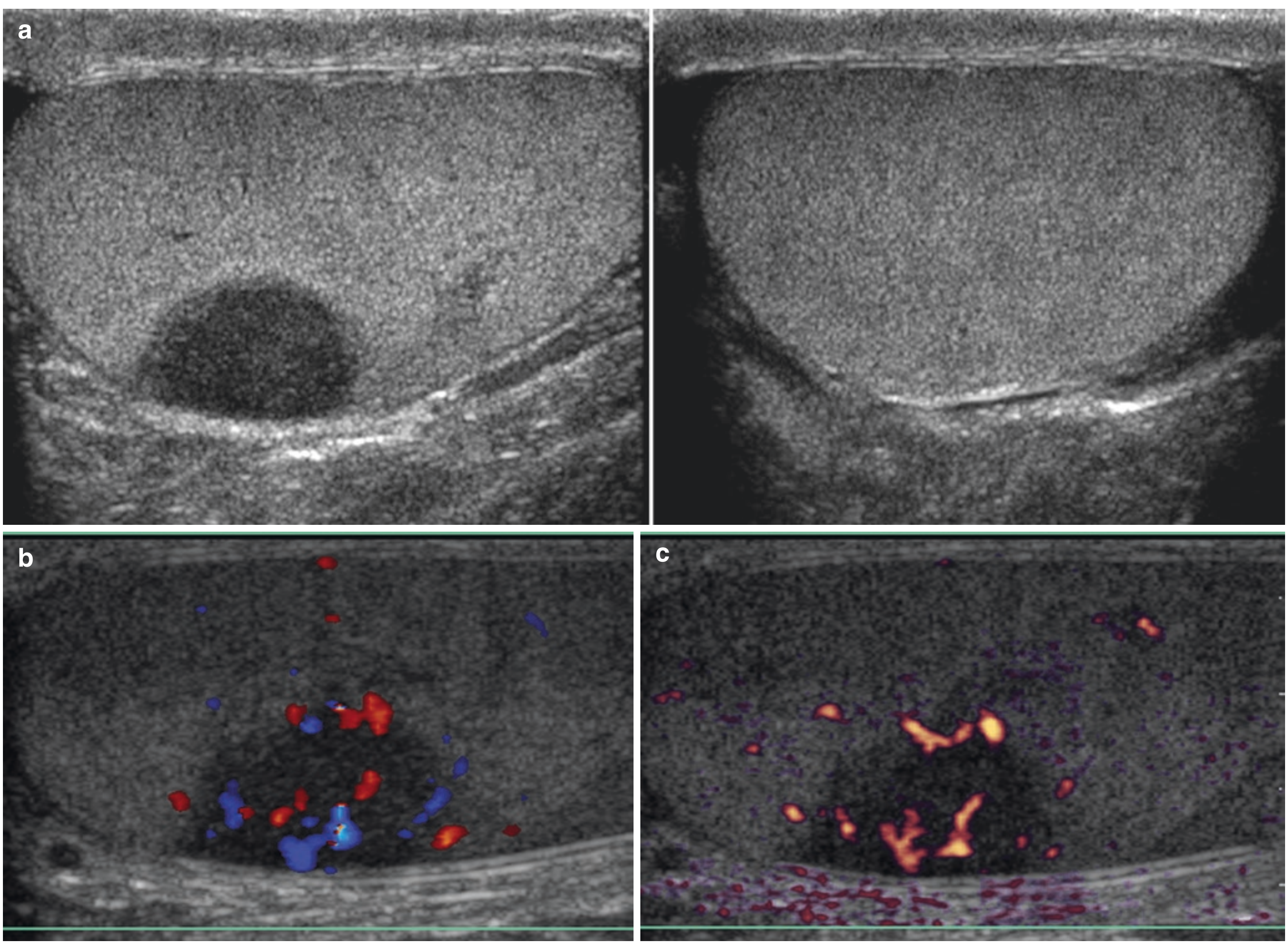


Fig. 3.4 Typical seminoma. Ultrasound of the right testis reveals a medium-sized, well-defined, hypoechoic mass with internal vascularisation at colour Doppler (**a–c**). **Small seminoma**. Hypoechoic lesion (5 mm) near the lower pole of the testis in a patient referred for subfertility. Histologically proven seminoma at orchiectomy. Small seminomas (**d**) are non-distinguishable from Leydigiomas (**e**). **Typical seminoma**. Testicular ultrasound shows a well-circumscribed, hypoechoic solid mass in the lower pole of the testis found to represent a seminoma at orchiectomy (**f**, **g**)

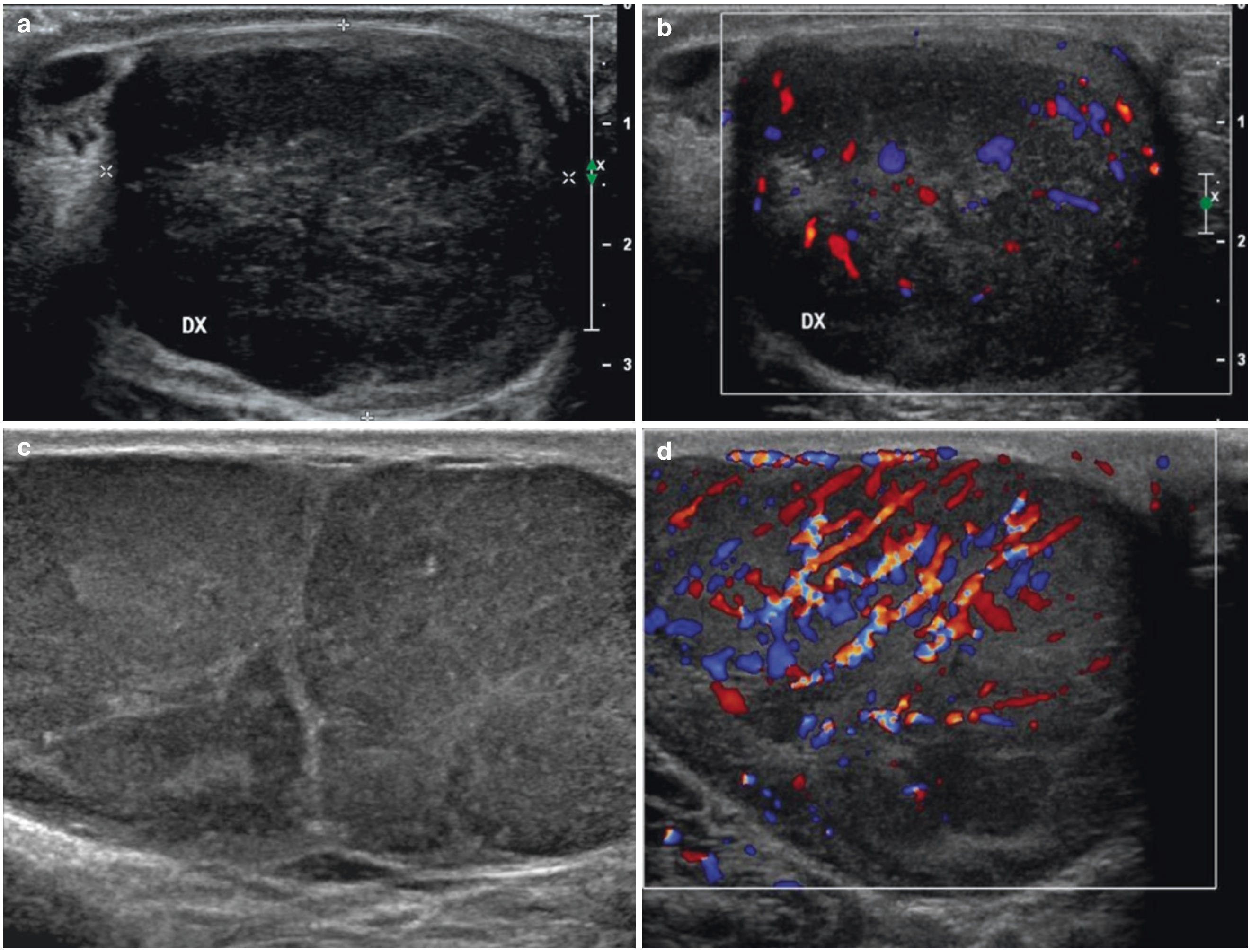


Fig. 3.5 Large seminoma. US scan shows a large mass totally replacing the testicle and causing diffuse testicular enlargement (**a**). Colour Doppler shows peripheral and internal vascularisation (**b**). Another case of large seminoma in a 45-year-old patient (**c**, **d**)

Pure seminoma is occasionally multinodular or presents as a focal lesion with polycyclic lobulated margins (Fig. 3.6), but does not generally exhibit significant internal cystic changes [24, 14]. In lobulated or multinodular tumours, the nodules are most commonly merged to one another (Fig. 3.7). True multifocal lesions with distinct nodules are rare (Figs. 3.8 and 3.9), as are bilateral tumours (Fig. 3.10), occurring in 2% of patients and almost always asynchronous (Fig. 3.4). Rarely, large seminomas become necrotic and appear partly cystic on ultrasound. Seminomas are usually confined within the tunica albuginea.

Seminoma is also associated with testicular microlithiasis (Fig. 3.11) in approximately a third of cases. There is usually increased vascularity on CDU when the mass is larger than 1.0 cm in diameter [2]. A typical finding is the arborisation (panel 3.2b) of high-flow, low-resistance vascular branches within the lesion (panel 3.2c), whereas irregular enriched intralesional and peripheral vascularisation is more frequently seen in NGCT and lymphomas.

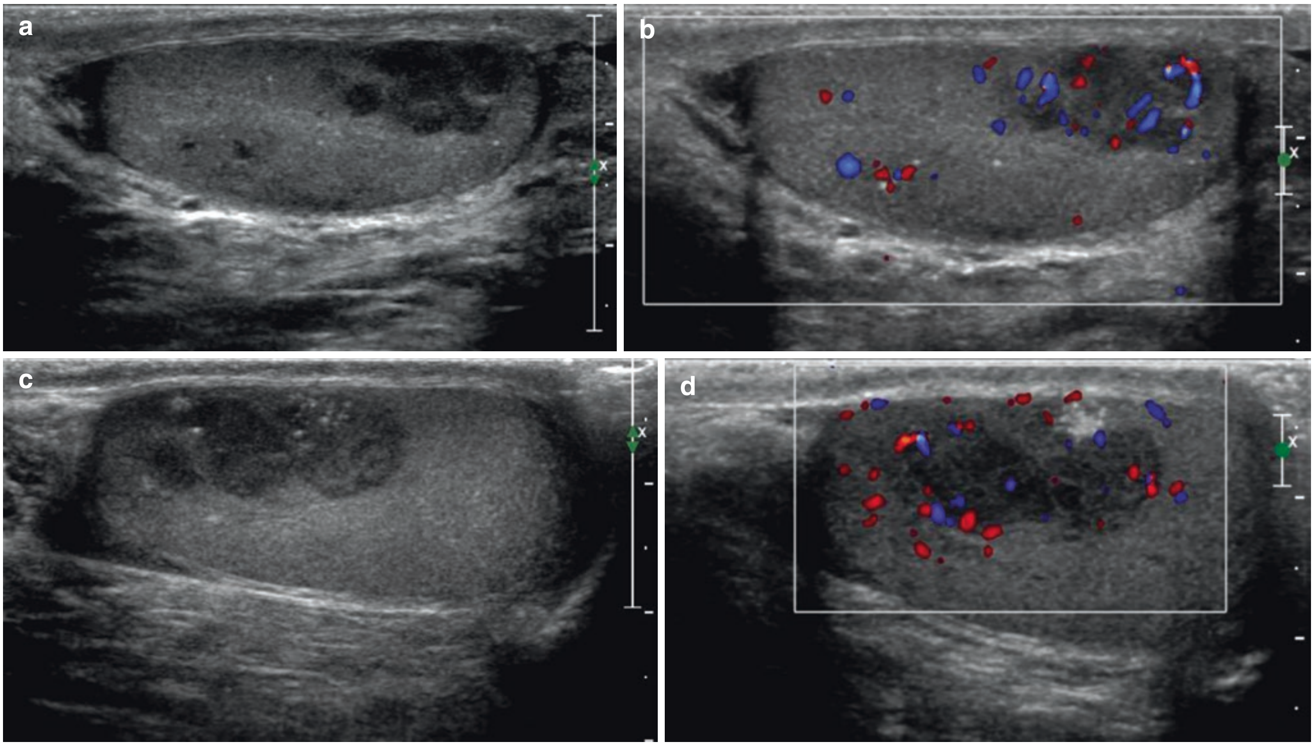


Fig. 3.6 Multilobulated seminoma. Longitudinal scan shows several separate foci of an inhomogeneous and hypoechoic lesion (**a**, **b**) in the left testis of a 28-year-old man and (**c**, **d**) in the right testis of a 21-year-old man

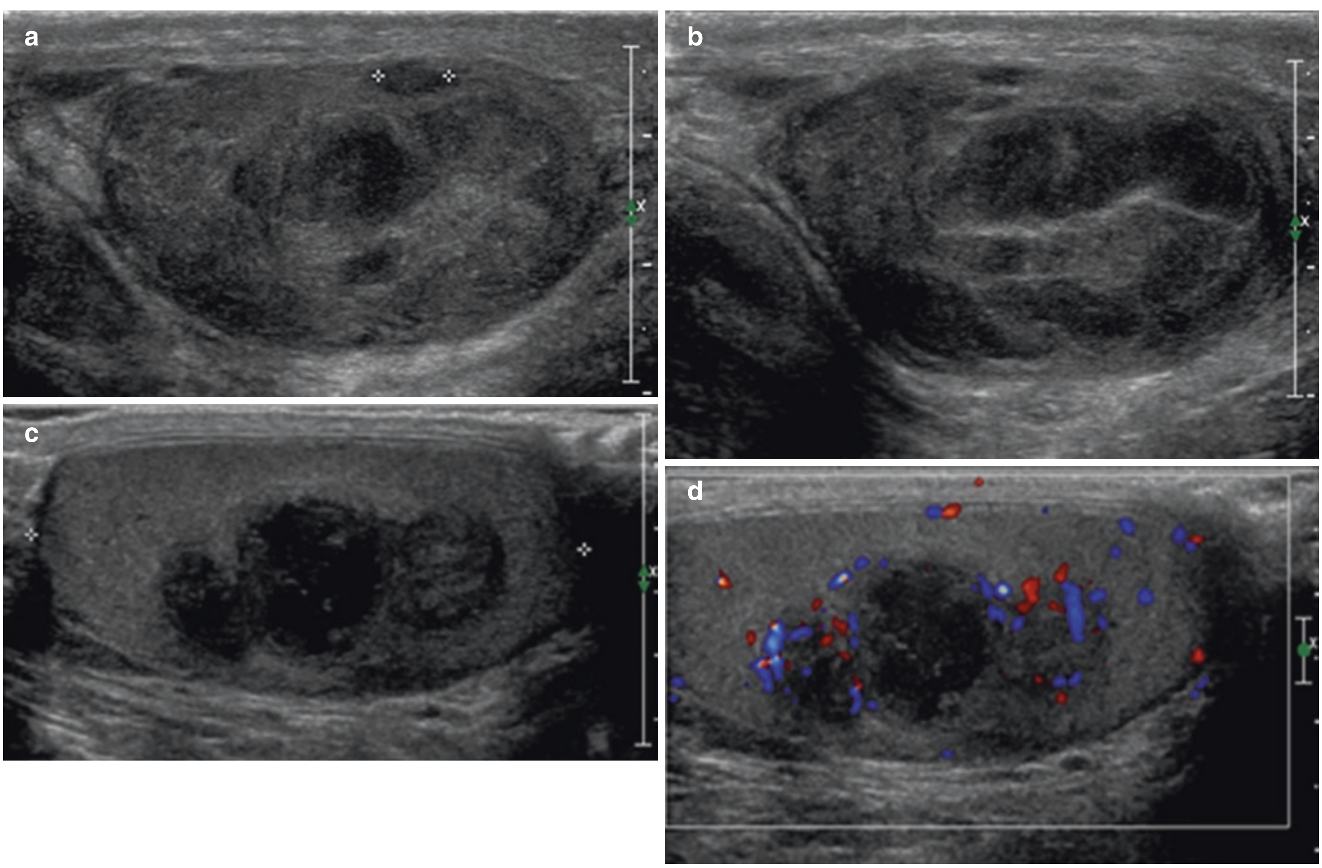


Fig. 3.7 Multinodular seminoma. Longitudinal scan. Multinodular seminoma showing different echogenicity of the nodules

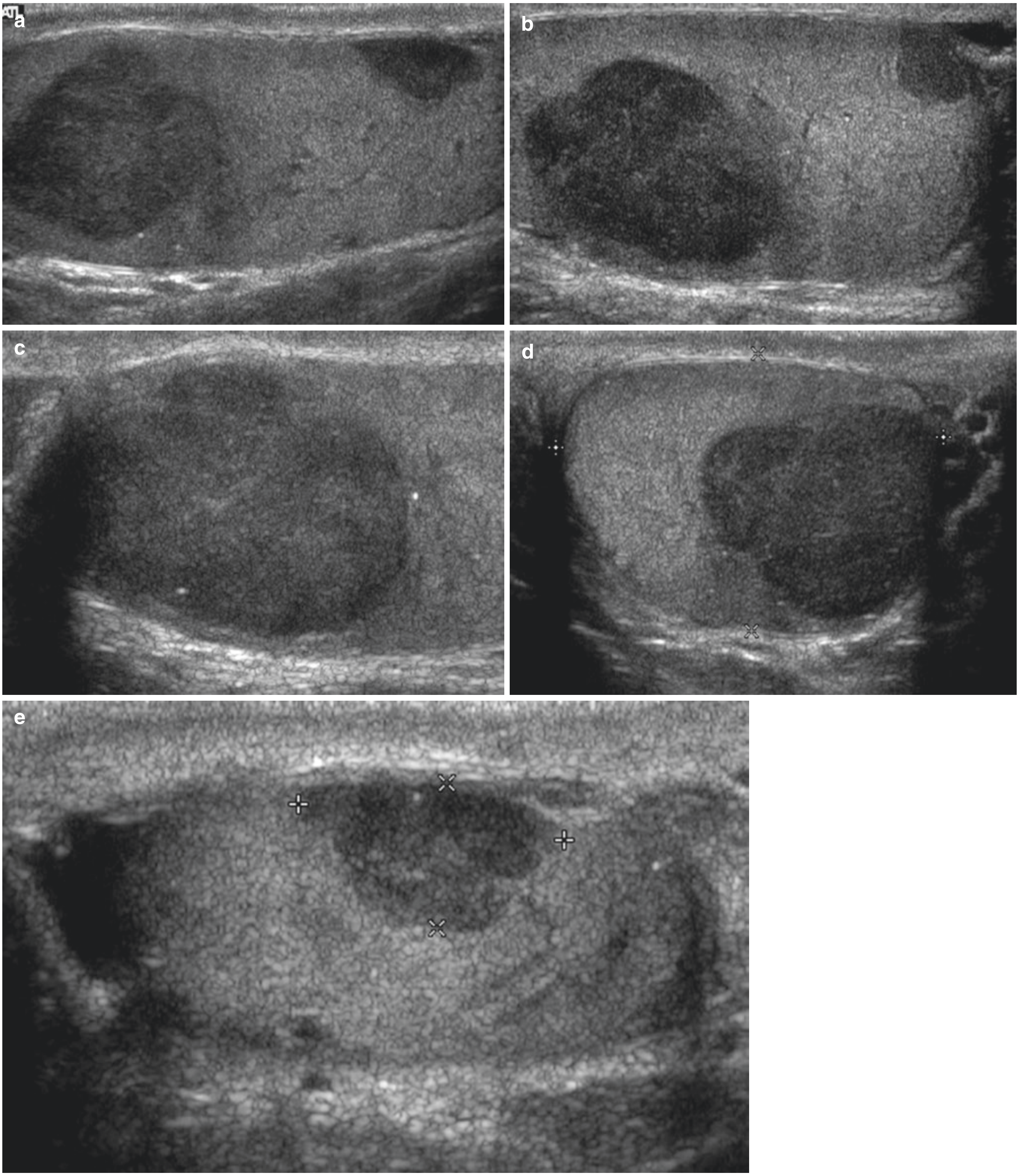
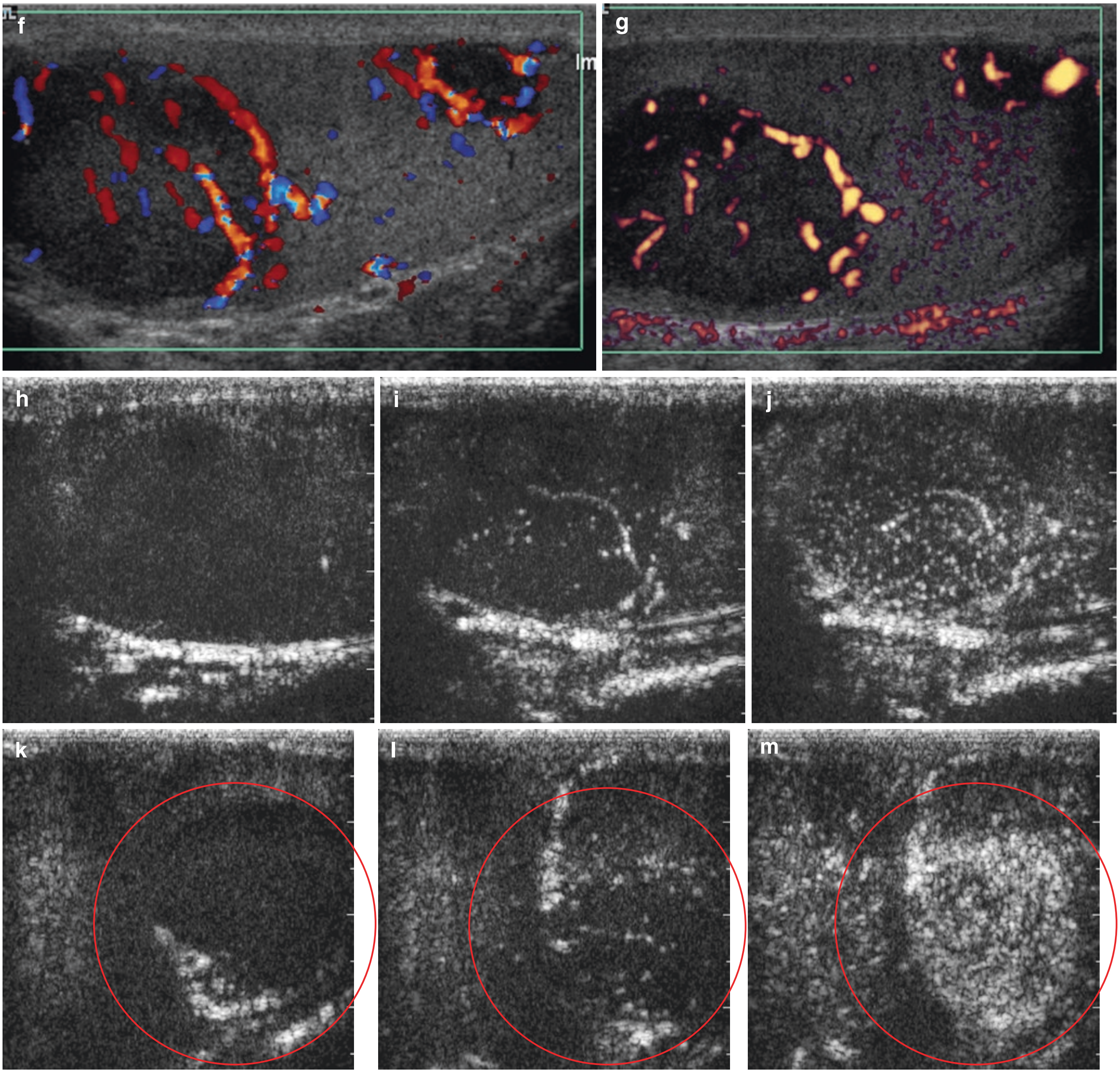


Fig. 3.8 Multifocal seminoma. Multifocal seminoma is generally asynchronous (**a**, **b**). In panels (**c**–**e**), the unclear relationship existent between the lesion and the testicular tunica is shown and in panels (**f**) and (**g**) the presence of intralesional vascularisation. Surgery confirmed the presence of a multifocal seminoma with invasion of the tunica albuginea and infiltration of tunica vaginalis. **Contrast-enhanced ultrasound** (longitudinal (**h**–**j**) and transverse (**k**–**m**) scans). On the arterial phase, US images obtained a few seconds after bolus injection (**h**, **k**) and at following stages of microbubble diffusion into the testis (**i**, **j**, **l**, **m**): the lesion enhances more rapidly and intensely than the adjacent testicular parenchyma, a finding that is consistent with a hypervascularised tumour. On the venous phase, US image showed rapid washout



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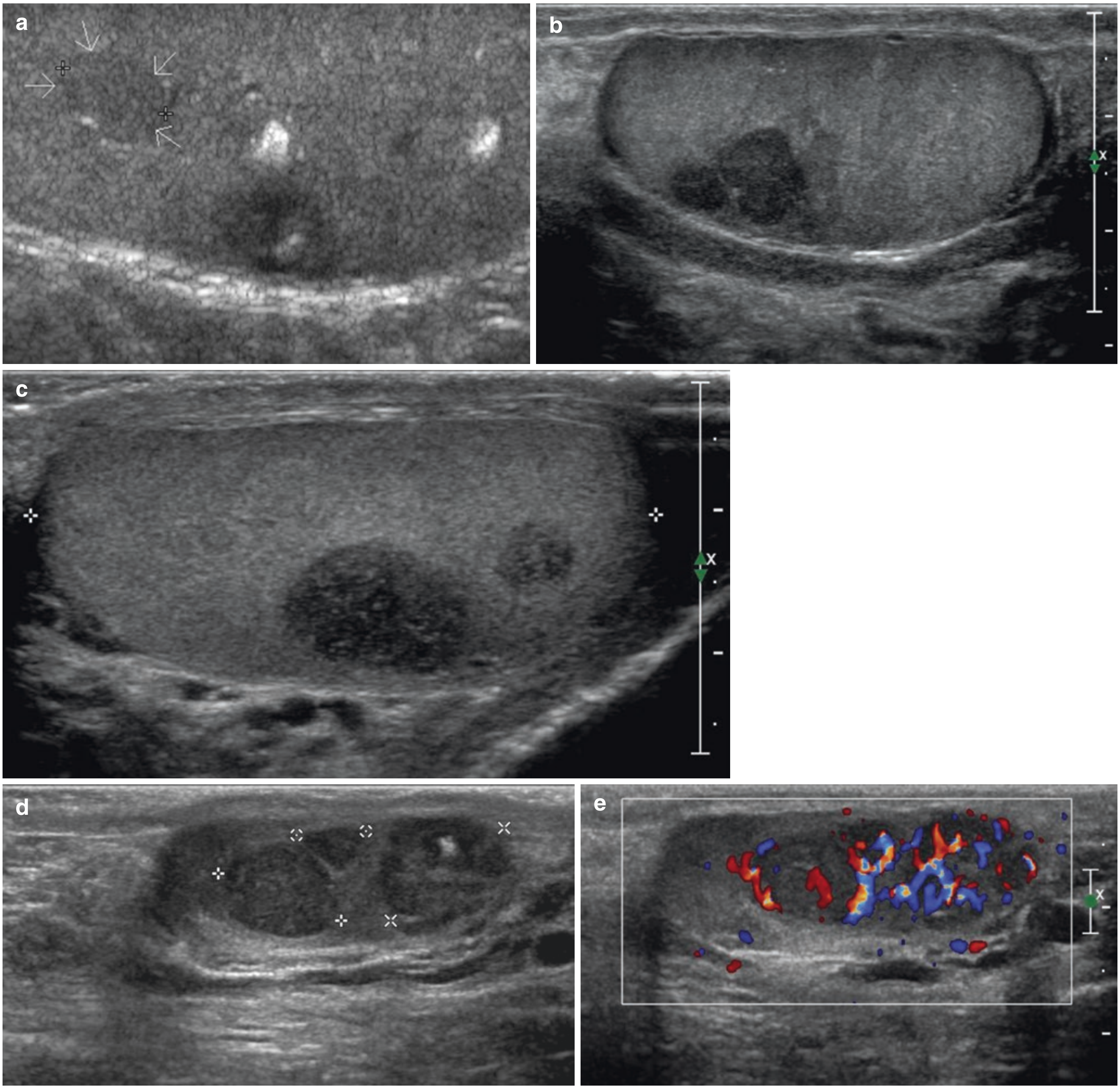


Fig. 3.9 Multifocal seminomas. A selection of four cases of multifocal seminomas, showing the wide range of appearance of these tumours

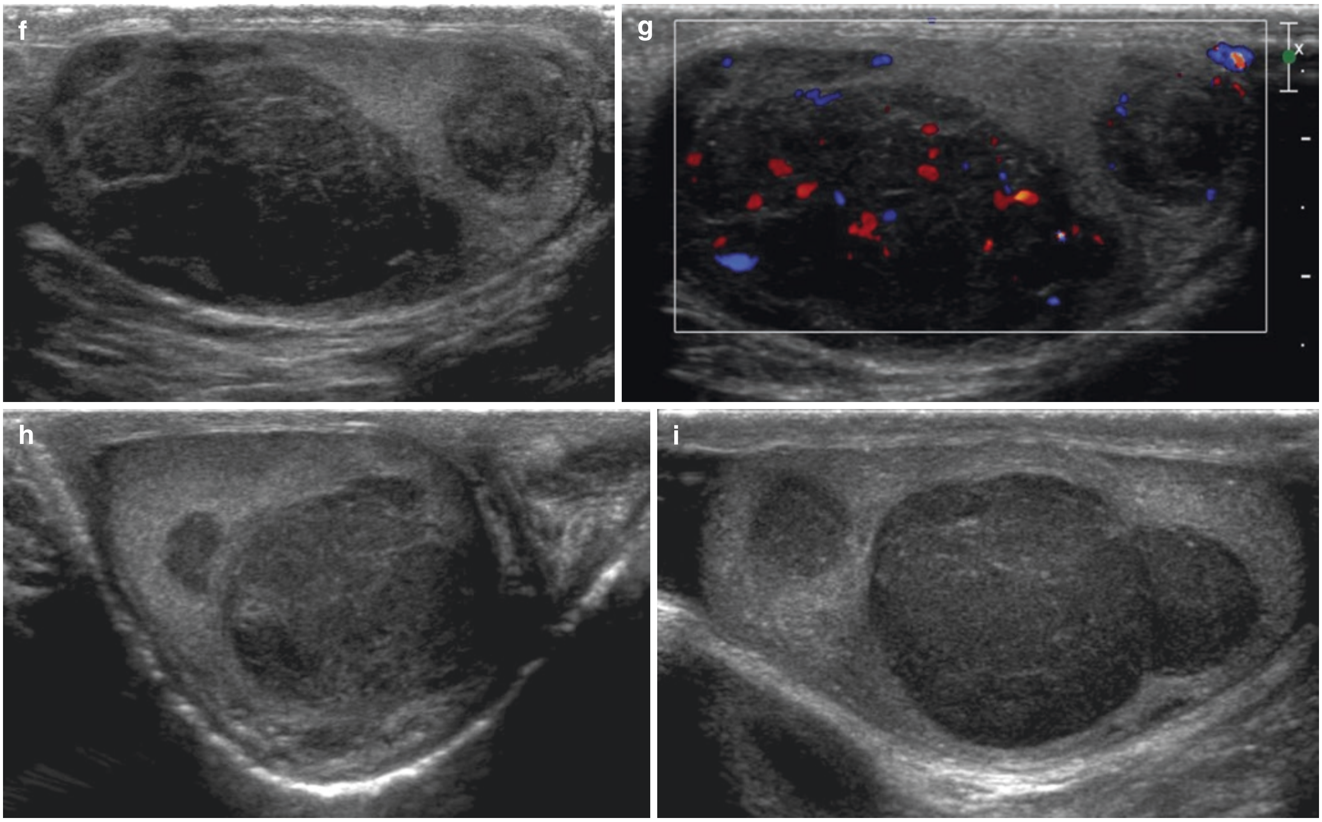


Fig. 3.9 (continued)

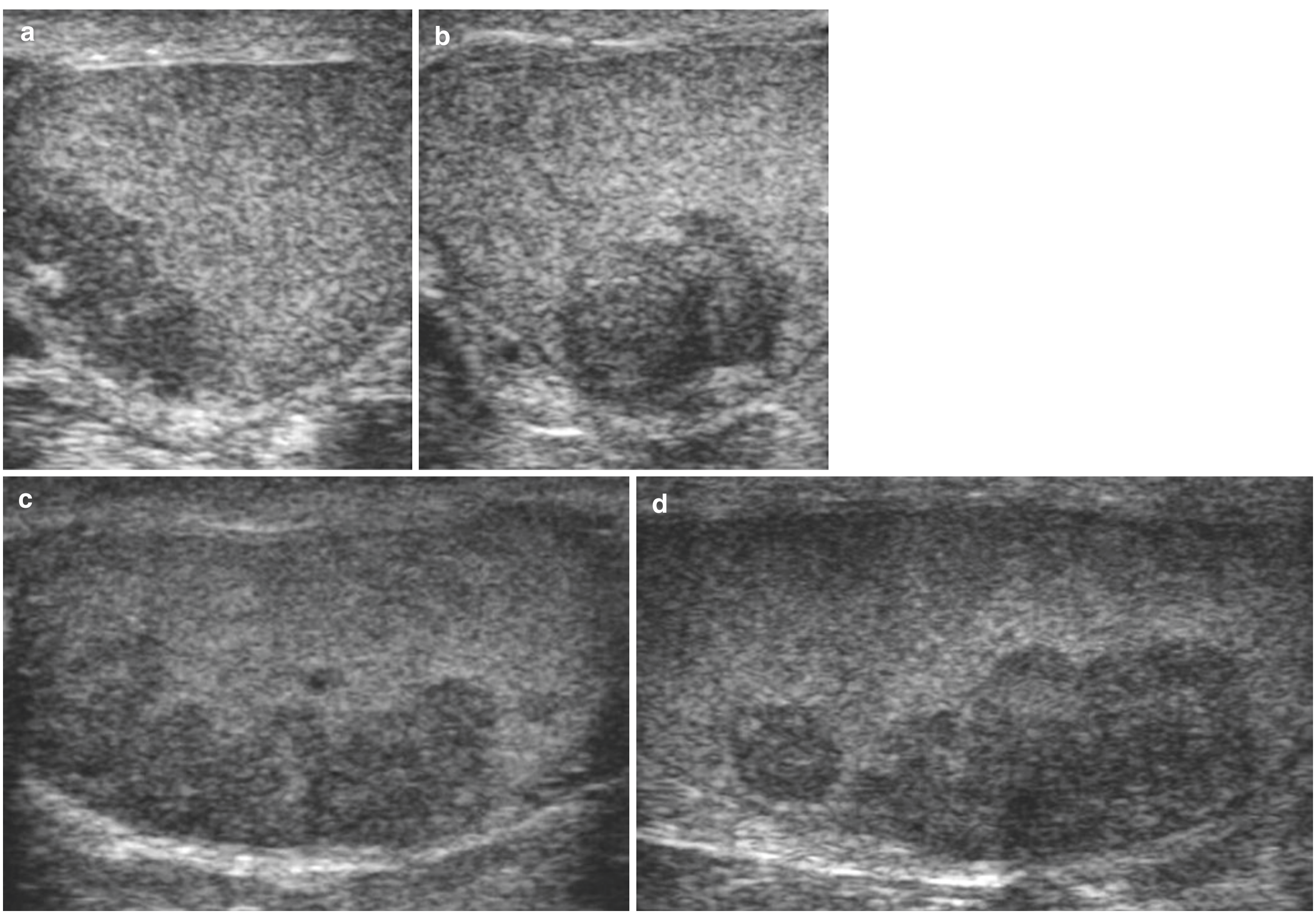


Fig. 3.10 Bilateral seminoma. A selection of three cases of bilateral seminomas *(panels **a–d**, Courtesy of: R.H. Oyen MD)*

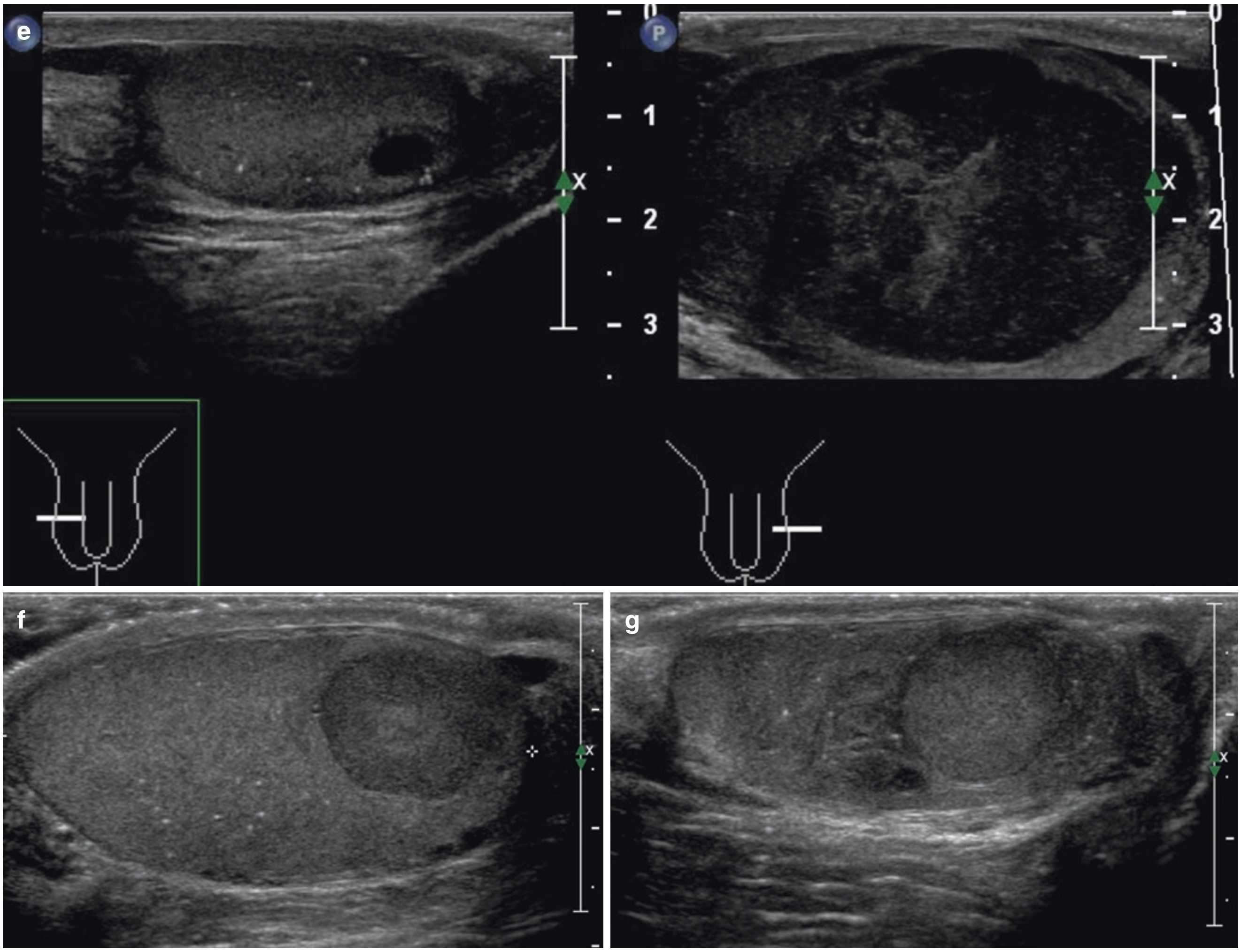


Fig. 3.10 (continued)

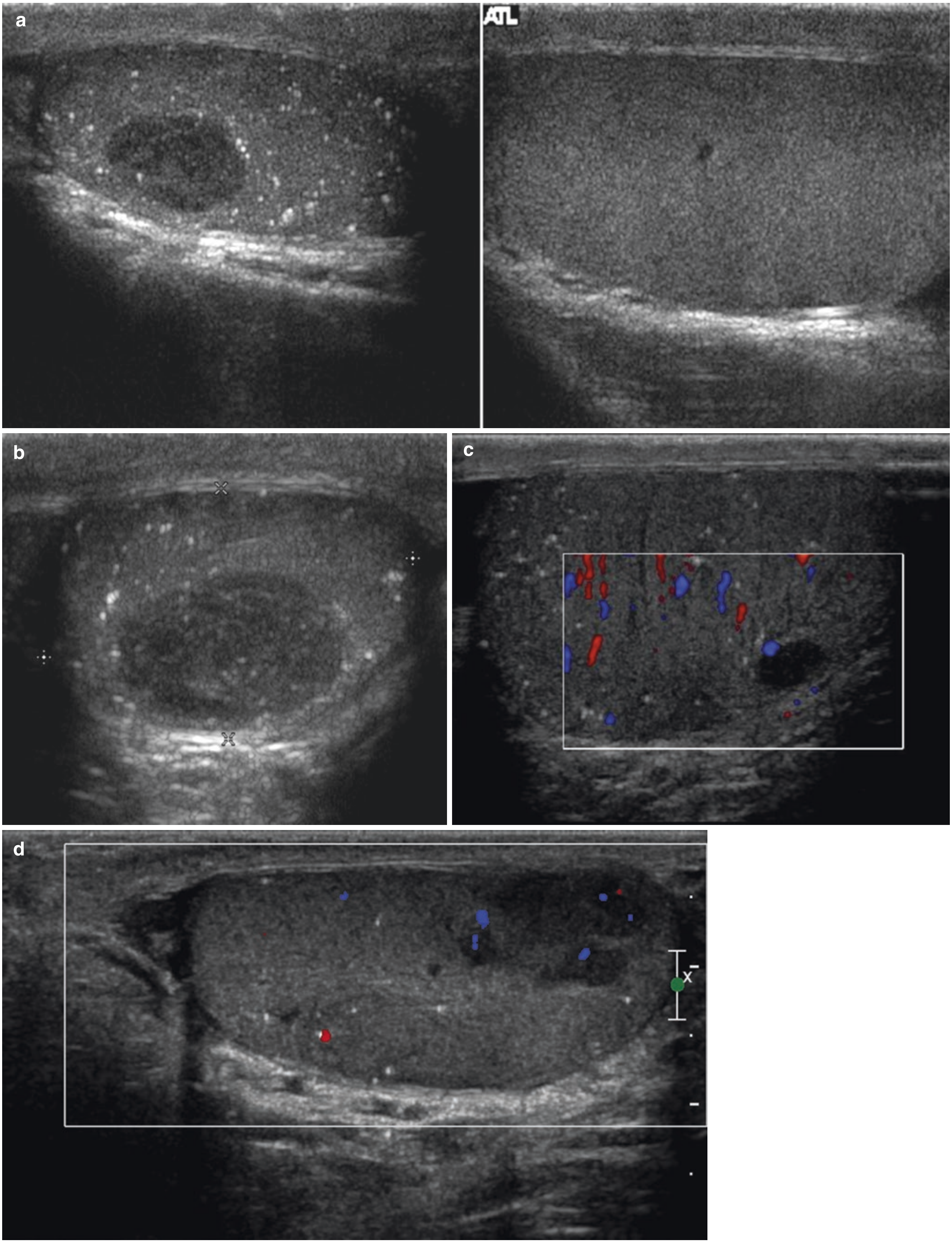


Fig. 3.11 Microlithiasis and seminoma. Seminoma is frequently associated with testicular microlithiasis in approximately a third of cases. There is usually internal vascularity on CDU (**f**–**h**) (panel ***e***, *Courtesy of: D.C. Howlett MD)*

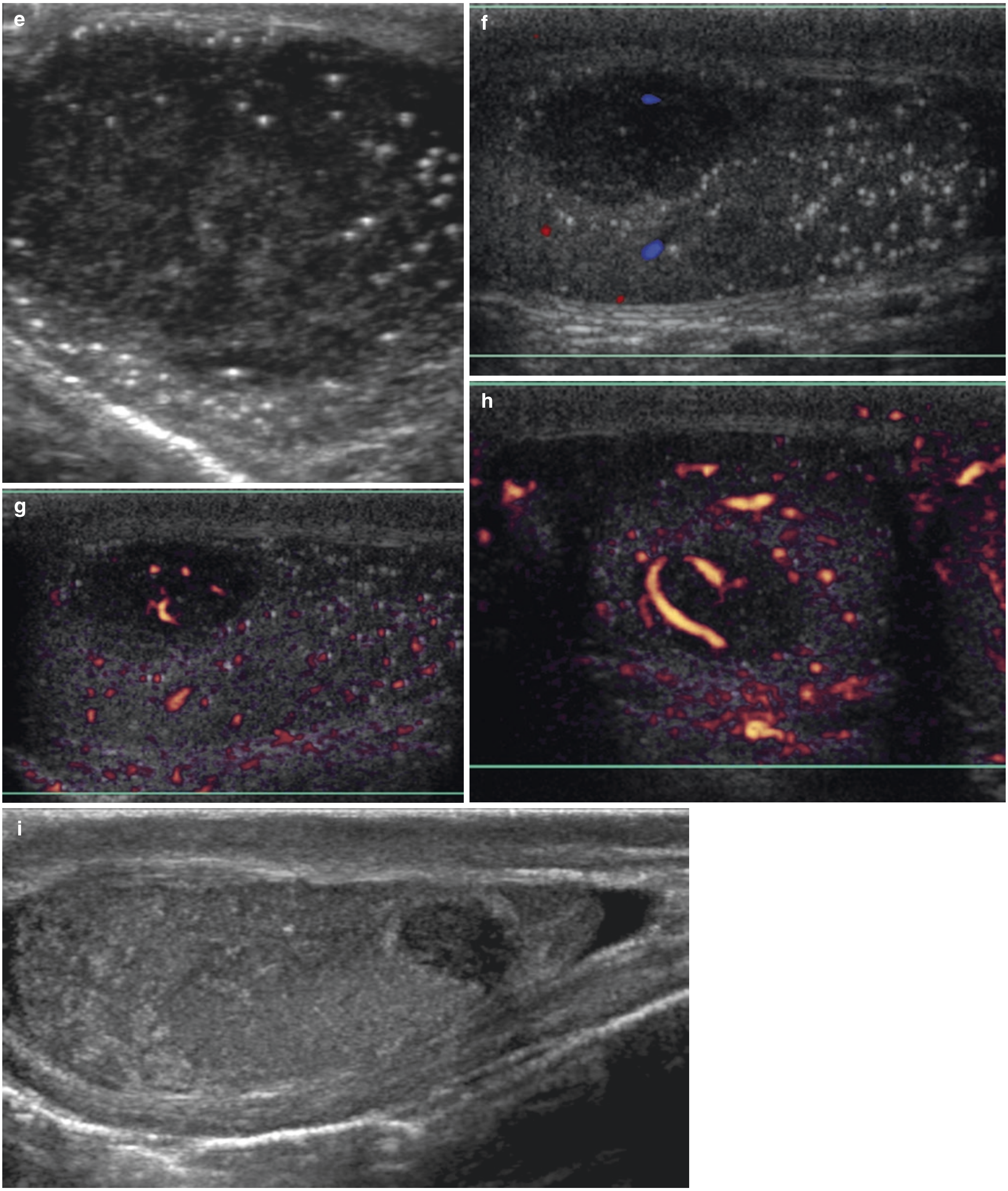


Fig. 3.11 (continued)

3.2.2 Non-seminomatous Germ Cell Tumours

Non-seminomatous germ cell tumours are a collective group comprising various histological subtypes including **embryonal cell carcinoma** (20–25%), **teratoma** (5–10%), **choriocarcinoma** (0.5%), **yolk sac tumours (<1%)** and **mixed tumours** – a mixture of virtually all histological types (20–40%) [24]. They are more aggressive than seminomas and frequently cause visceral metastases to the para-aortic and iliac lymph nodes. In the right testis, the sentinel nodes are in the interaortocaval chain at the second lumbar vertebral body. For the left testis, the sentinel nodes are the left para-aortic nodes in an area bounded by the renal vein, aorta, ureter and inferior mesenteric artery. External iliac node involvement is seen for tumours invading the epididymis, while skin involvement may lead to direct spread to the inguinal nodes. The clinical and US features of the various histotypes are briefly presented.

3.2.2.1 Embryonal Cell Carcinoma

Embryonal cell carcinoma is the second most common histological type of testicular tumour after seminoma and the most frequent in mixed tumours – present in 87% of cases. However, pure embryonal cell carcinomas are rare, accounting for only 2–3% of testicular germ cell neoplasms. They are most common during the third decade of life, with a significant proportion already metastasised at the time of presentation. They are hypoechoic on US but more heterogeneous than seminomas (Fig. 3.12), due to cystic degeneration and calcifications. Embryonal cell carcinomas are frequently

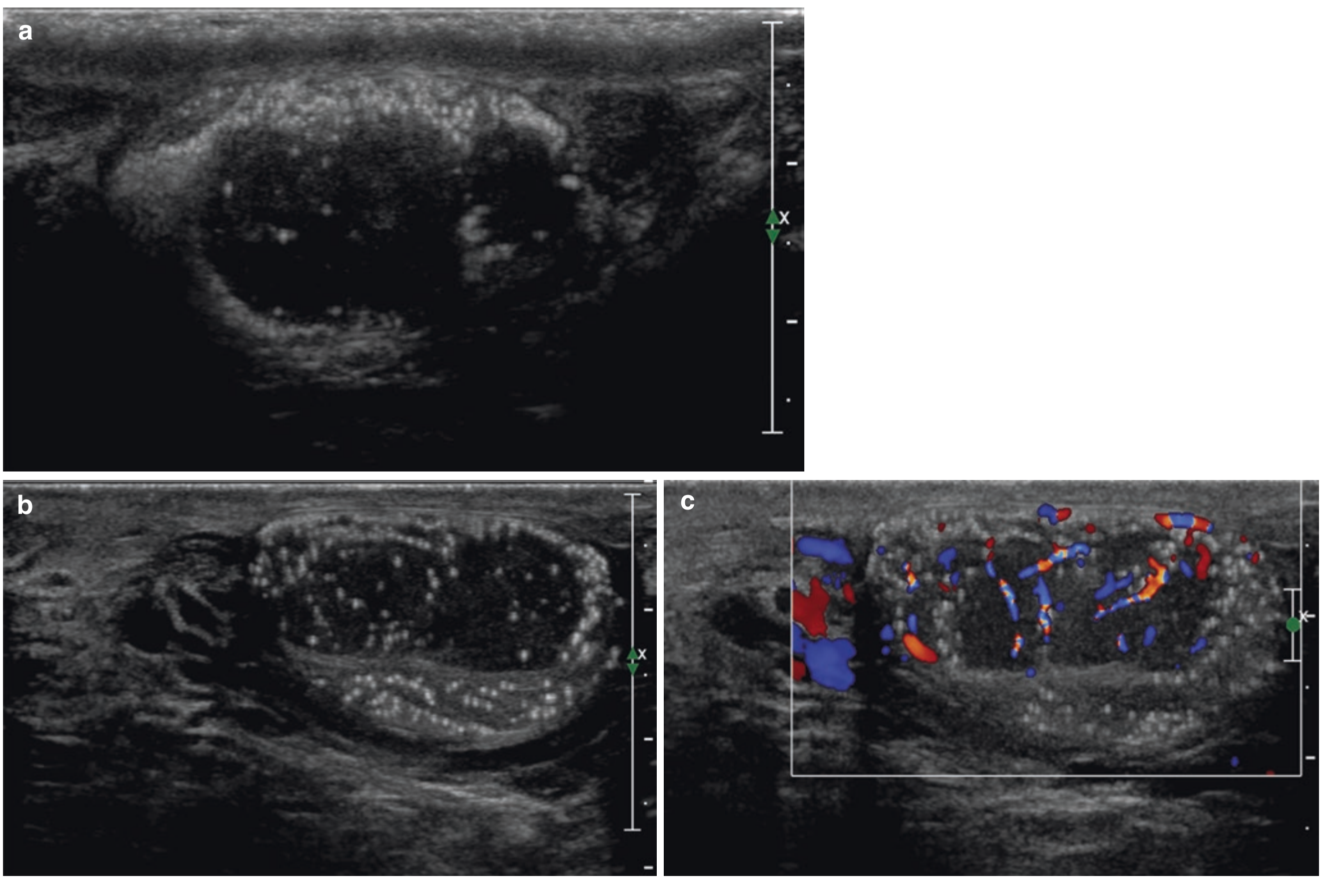


Fig. 3.12 Embryonal carcinoma. Embryonal cell carcinoma. Large tumoural mass in the testis in a patient with massive microlithiasis

large and grow rapidly, and the tunica albuginea is often distorted. Focal areas of haemorrhage may contribute to increased heterogeneity, and the borders of the tumour are less distinct than seminomas (Fig. 3.13) [2]. Cystic areas are present in one third of tumours, and echogenic foci, with or without acoustic shadowing, are not uncommon. The US features of pure embryonal cell carcinoma are not dissimilar to those of mixed NSGCT (Fig. 3.14).

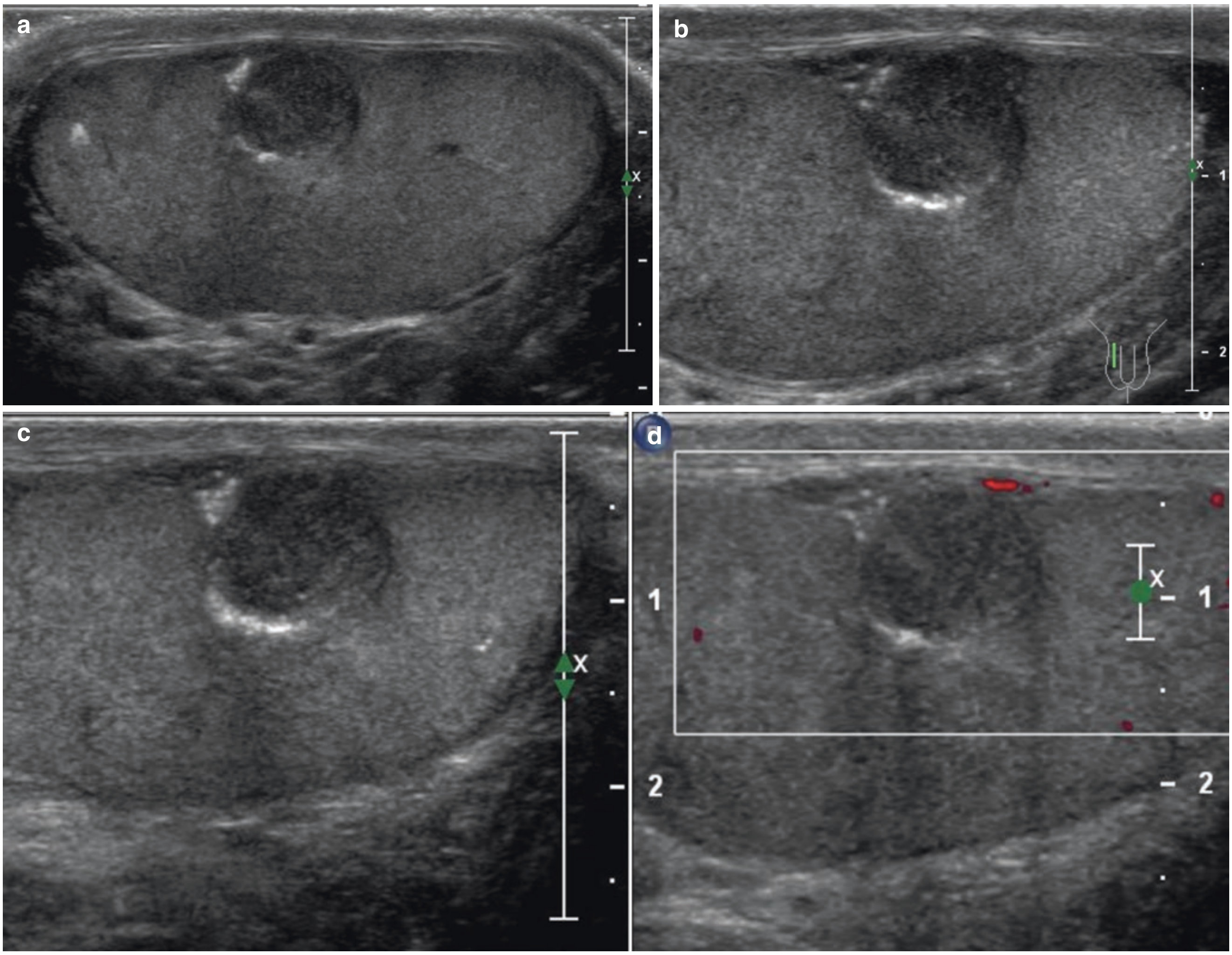


Fig. 3.13 Embryonal carcinoma. Longitudinal scan of the testis of a 25-year-old man with pure embryonal cell carcinoma (pt2). The tumour presents as a hypoechoic mass that distort the tunica albuginea (**a**, **b**), with intralesional calcification. Colour Doppler shows peripheral vascularisation (**c**)

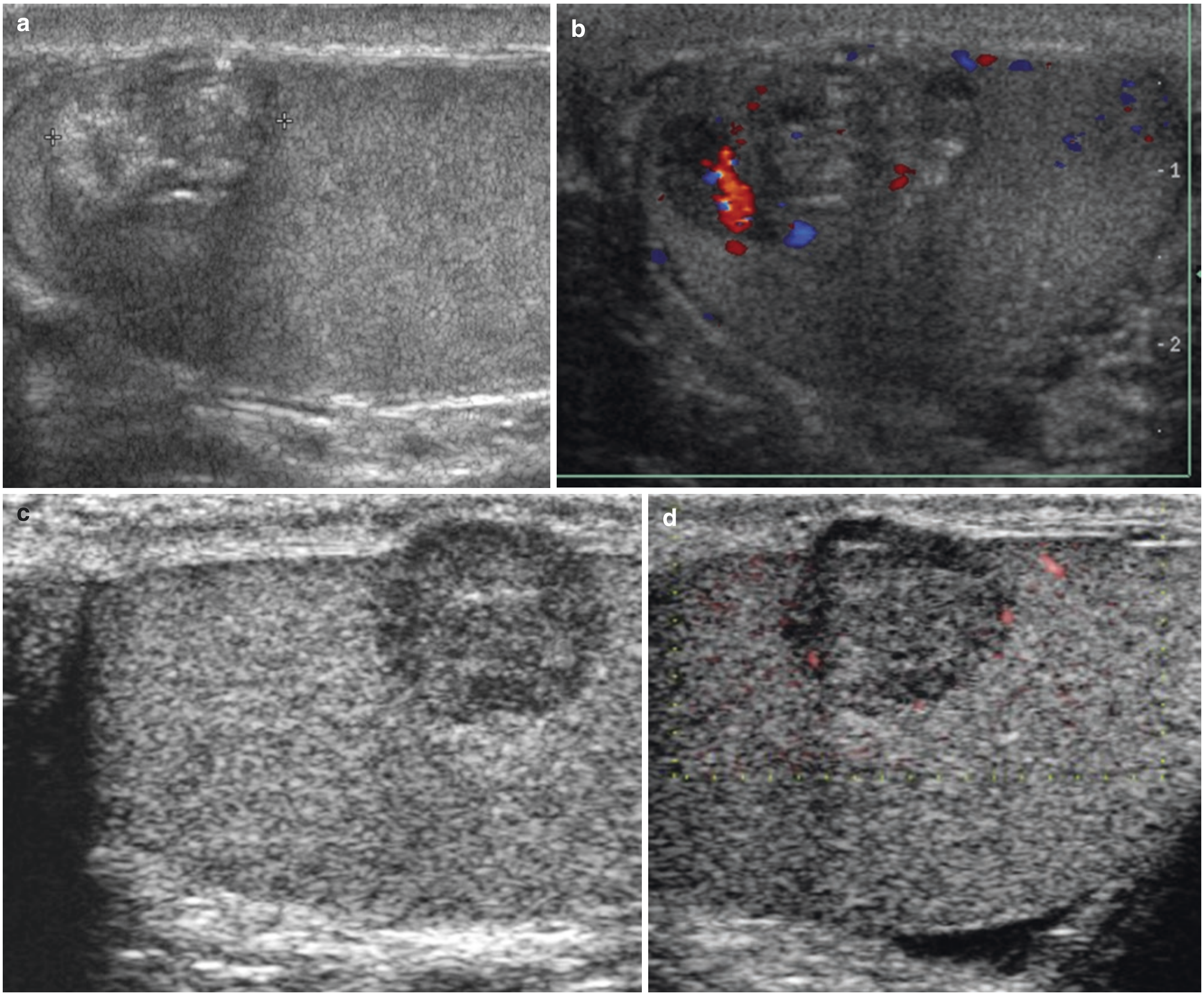


Fig. 3.14 Embryonal carcinoma. US scan of the testis shows a medium-sized, irregular heterogeneous mass at the upper pole of the testis that was confirmed an embryonal carcinoma at histology (**a**, **b**). Pure embryonal carcinoma in a 19-year-old man (**c**, **d**) *(panels **c**, **d**, Courtesy of R.H. Oyen MD)*

3.2.2.2 Teratomas

Teratomas constitute 5–10% of primary testicular neoplasms. They peak in early childhood (less than 4 years), when they are usually benign, in contrast with the more common yolk sac tumour. They peak again in the third decade of life, when they are usually malignant [15].

Teratomas are complex tumours with a disorderly arrangement of adult and foetal tissues, which involve all three germ layers (endoderm, mesoderm and ectoderm). They are histologically classified as mature (Fig. 3.15), immature (Figs. 3.16 and 3.17) or teratoma with malignant transformation, depending on the germ layers present [24]. Small

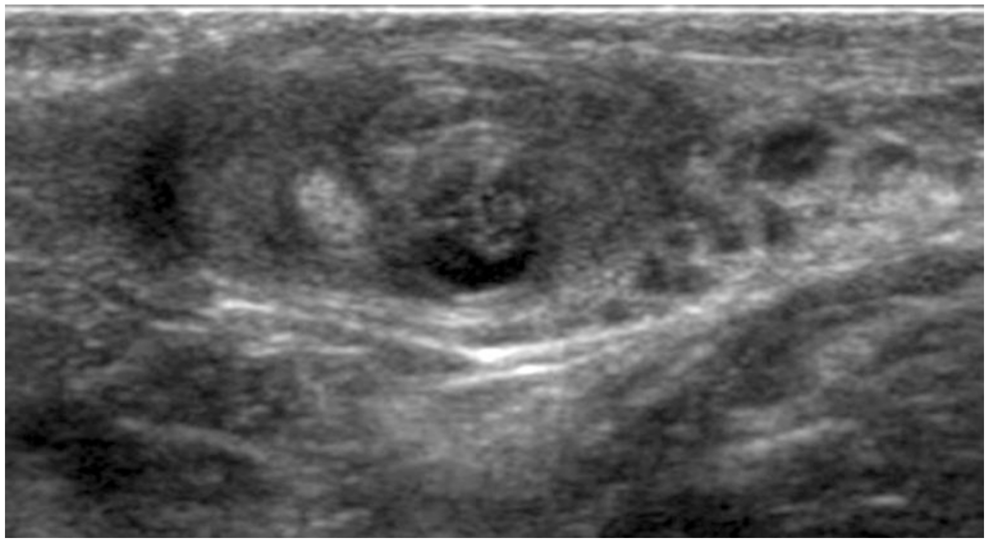


Fig. 3.15 Mature teratoma. Histologically proven mature teratoma in a 28-year-old man with Klinefelter's Syndrome. Longitudinal US scan shows a cystic mass with echogenic borders and peripheral solid components

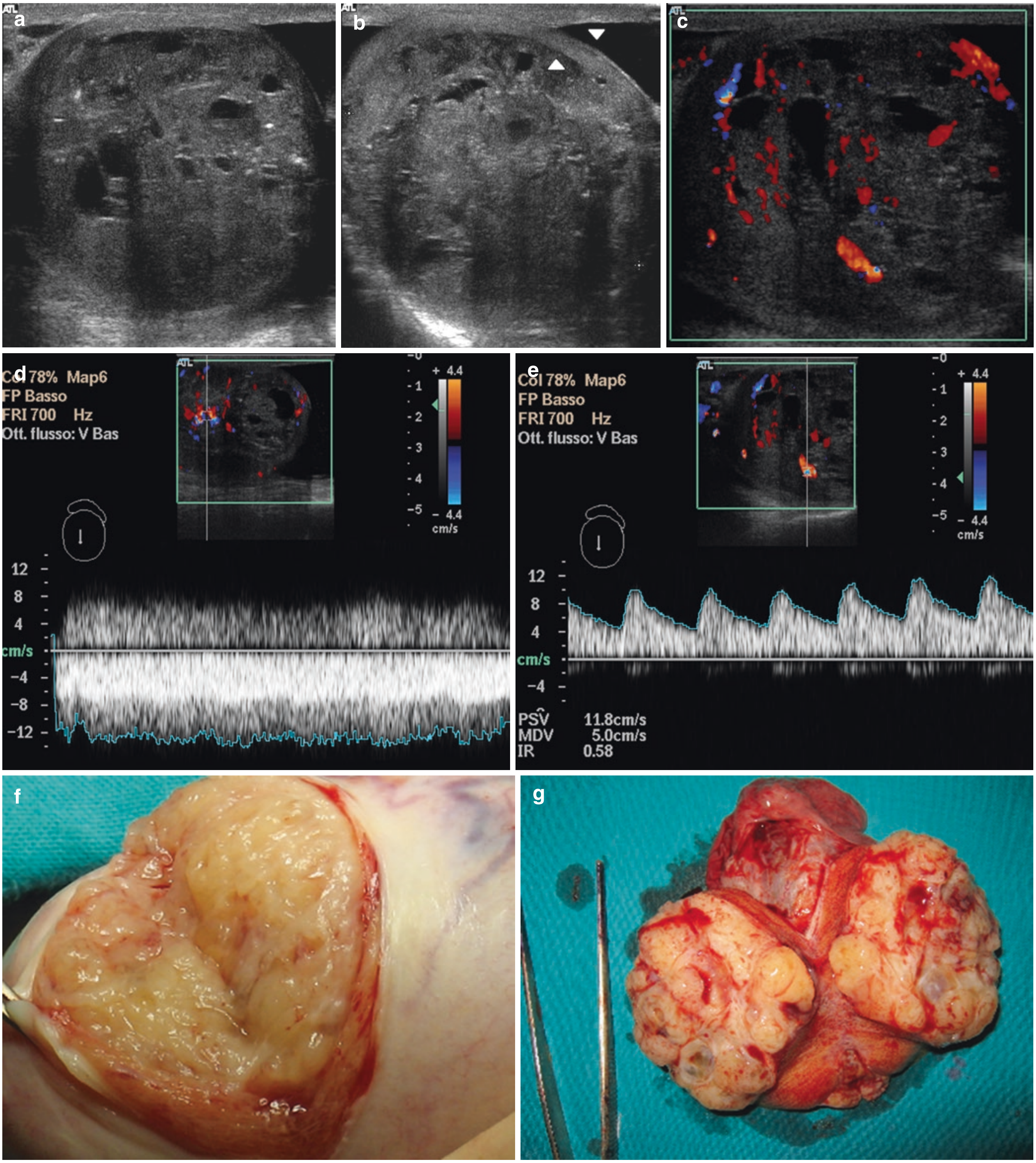


Fig. 3.16 Immature teratoma. US scans of a complex, heterogeneous teratoma found in the testis of a 30-year-old man. The scans show a solid, large, hypoechoic mass with foci of increased echogenicity due to calcifications and fibrosis and anechoic areas resulting from cystic elements (**a**). A rim of normal testis (*between arrows*) is also seen in *panel* (**b**). Colour Doppler scans (**c**) show intralesional veins (**d**) and arteries (**e**) with low-resistance flow on colour Doppler evaluation. In *panels* (**f**, **g**), the gross specimen of the tumour is shown

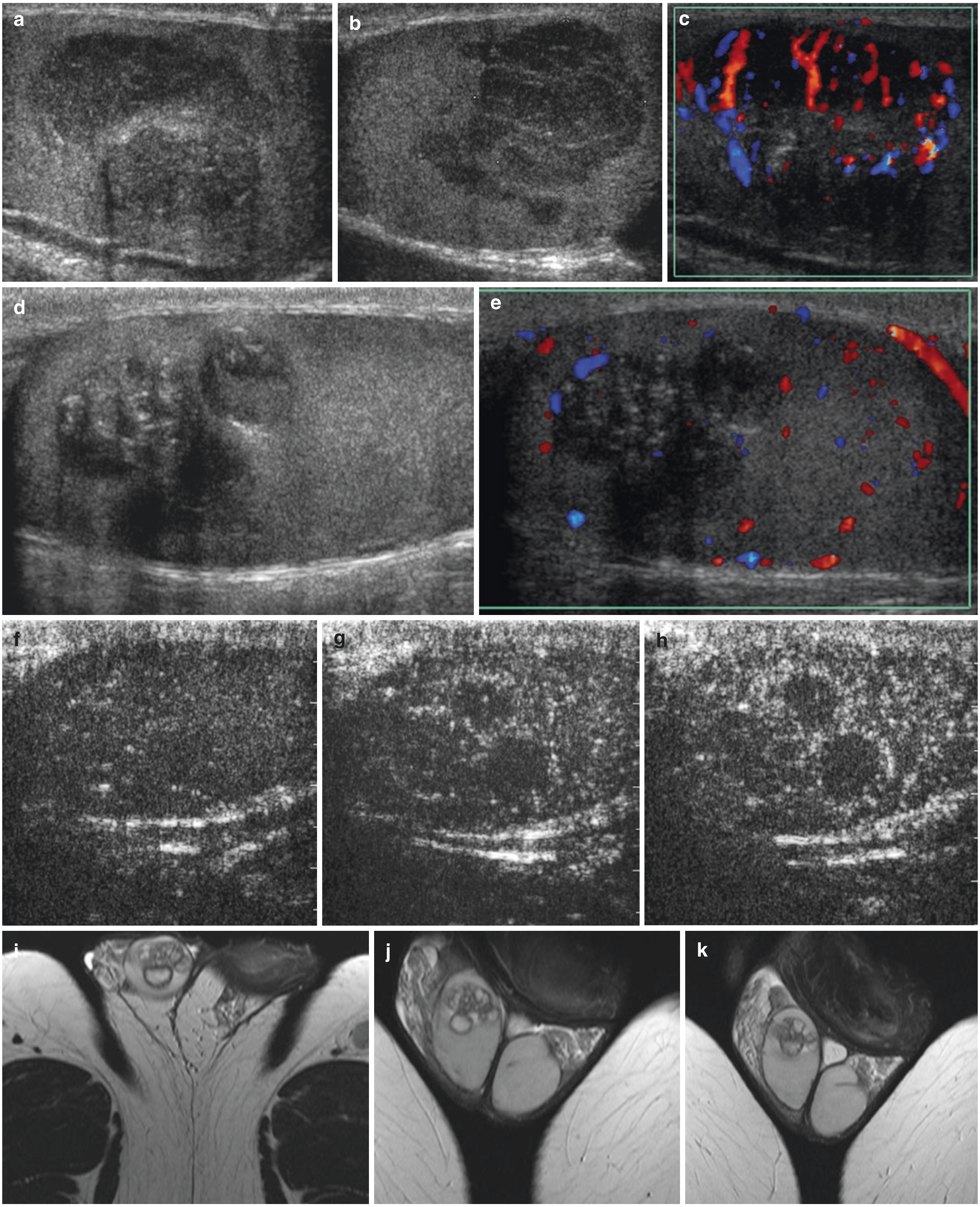


Fig. 3.17 Immature teratoma. Longitudinal scan shows a large solid teratoma in an 18-year-old boy, with irregular margins, markedly heterogeneous, predominantly hypoechoic, with internal hyperechoic foci (**a**, **b**). The tumour has an increased flow at colour Doppler (**c**). **Immature teratoma**. In panels (**d**, **e**), a further case of immature teratoma, in a 27-year-old man, is shown. The tumour was predominantly composed of cysts full of keratin, cartilage and cutaneous annexes, but there were also two foci of immature tissue. In panels (**f**–**h**), there is a selection of US images from the contrast-enhanced study. In panels (**i**–**k**), there is the preoperative MRI study

embryonal cell carcinoma foci are frequent in large teratomas and should be sought extensively. Teratomatous elements can be found in nearly half of mixed germ cell tumours. One third of teratomas metastasise, usually by a lymphatic route, within 5 years.

The US appearance of teratomas varies with their nature. They can be solid intratesticular lesions (Fig. 3.17) but more usually have a predominantly cystic architecture with internal hyperechoic spots (Fig. 3.18) [24]. The heterogeneous US appearance reflects their complex histological nature. Teratomas generally form well-circumscribed masses. Dense echogenic foci causing acoustic shadowing are common, due to focal calcification, cartilage, immature bone, fibrosis and non-calcific scarring. Cysts are also a common feature and may be anechoic or complex, depending on their contents (e.g. serous, mucoid or keratinous fluid) (Fig. 3.19). Care must be taken to differentiate teratomas from benign complex cysts within the testis, including epidermoid cysts.

Conservative treatment has been advocated for prepubertal teratomas, but is not an option for teratomas in post-pubertal testes. Irrespective of their histological characteristics and maturity, adult teratomas can metastasise (mature does not equal benign!).

In contrast with teratomas, epidermoid cysts are benign lesions and have no malignant potential. These are true cysts, although they are filled with echogenic material simulating a

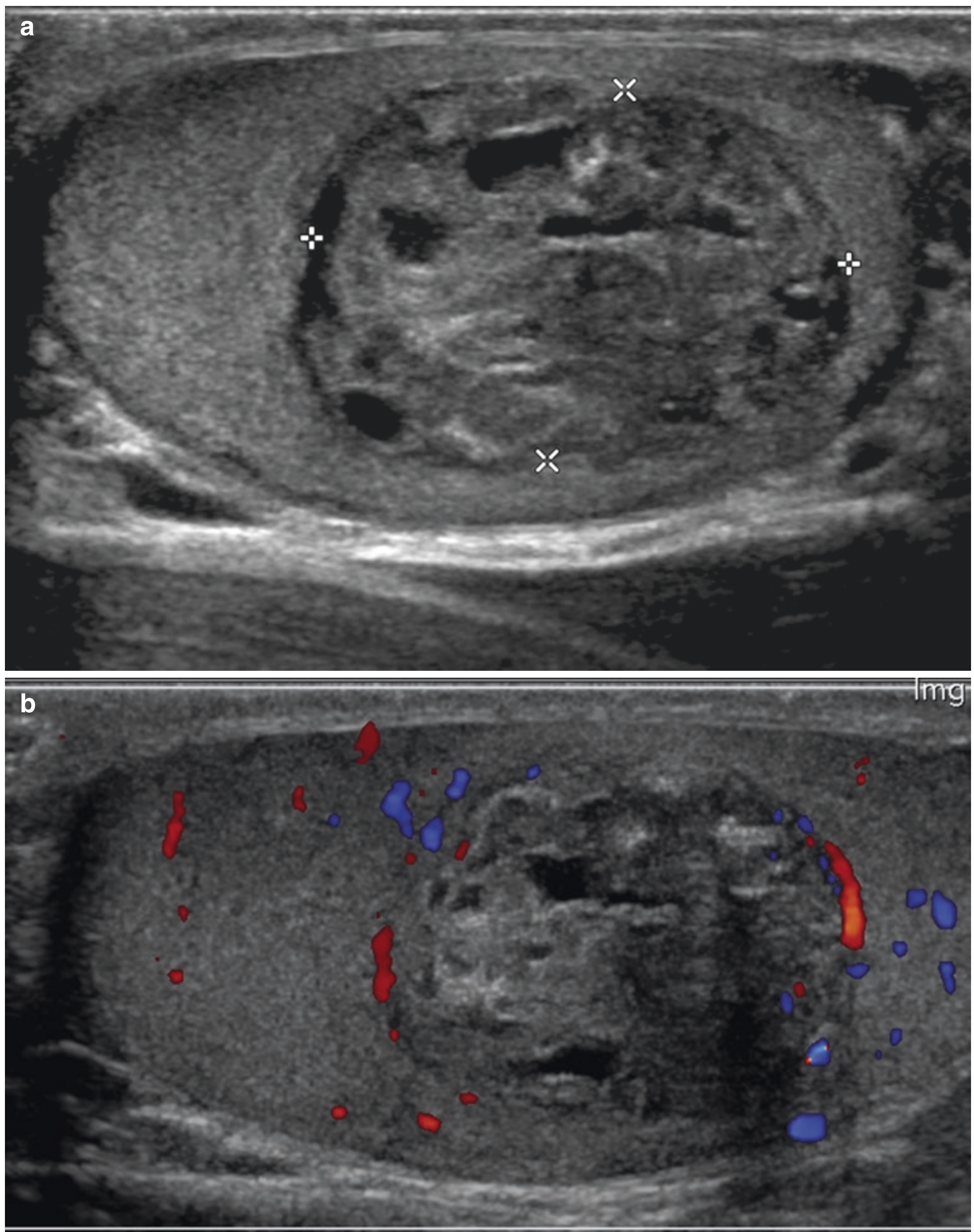


Fig. 3.18 Malignant teratoma (teratocarcinoma). Ultrasound of the right testis of a 24-year-old man. There is a rounded solid mass within the testis with prominent cystic elements and foci of calcification. This was found to be malignant teratoma

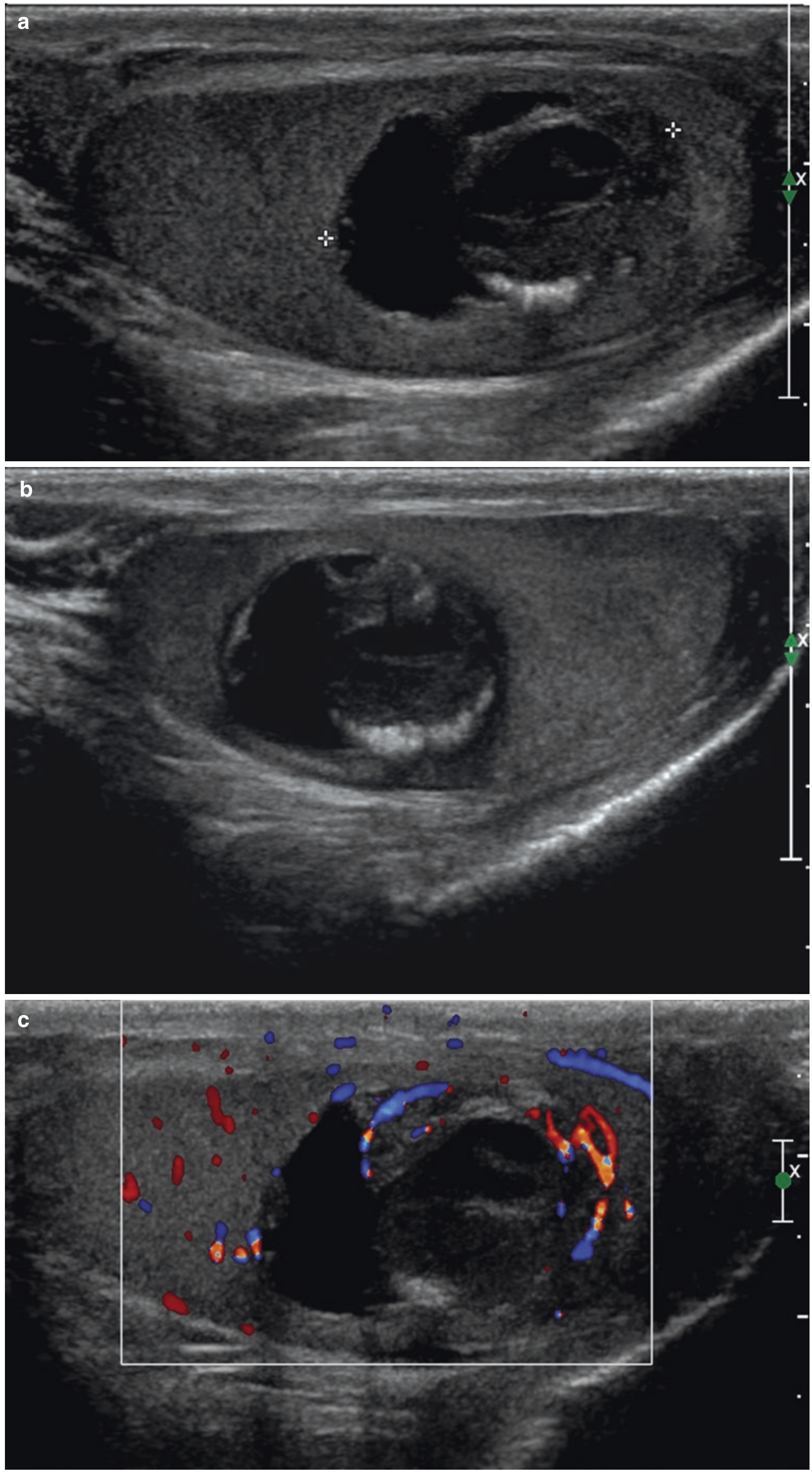


Fig. 3.19 Cystic teratoma. A teratoma with predominantly cystic architecture

solid component, originating from a monodermal layer or squamous metaplasia of surface mesothelium. It is therefore still under debate whether epidermoid cysts should be discussed with teratomas. Due to their clinical behaviour, they are discussed in the previous chapter with other benign intratesticular lesions.

3.2.2.3 Choriocarcinoma

Choriocarcinoma is one of the less common of all germ cell tumours and is most common during the second and third decades of life. Ultrasound is non-specific and may demonstrate heterogeneous solid masses with areas of haemorrhage, necrosis and calcification [16]. Pure choriocarcinoma is the rarest type of germ cell tumour, accounting for less than 0.5% of malignant primary testicular tumours (Fig. 3.20). However, it is detected in 5–7% of mixed germ cell tumours.

Most germ cell tumours spread initially via the lymphatic system, while choriocarcinomas show a tendency towards early haematogenous spread. These tumours are highly malignant, and distant metastases (Fig. 3.21), rather than the scrotal mass, may be the reported complaint. Brain, lung, liver and bone metastases are particularly common with

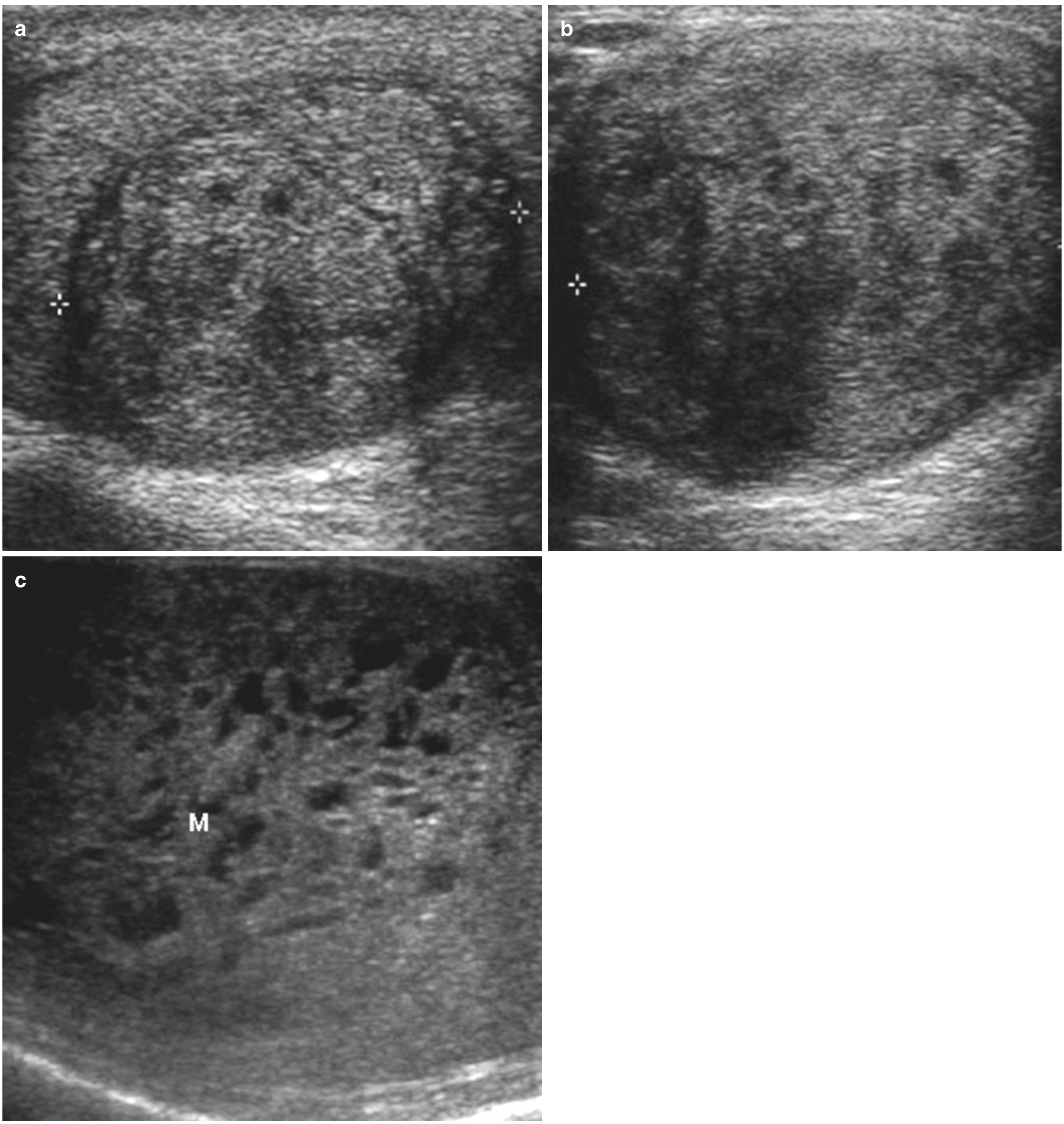


Fig. 3.20 Choriocarcinoma. Longitudinal and transverse scan show a large, heterogeneous mass. Haemorrhage with focal necrosis is present (**a**–**c**) *(panels **a**, **b**, Courtesy of R.H. Oyen MD; Figure **c**, Courtesy of D.C. Howlett)*

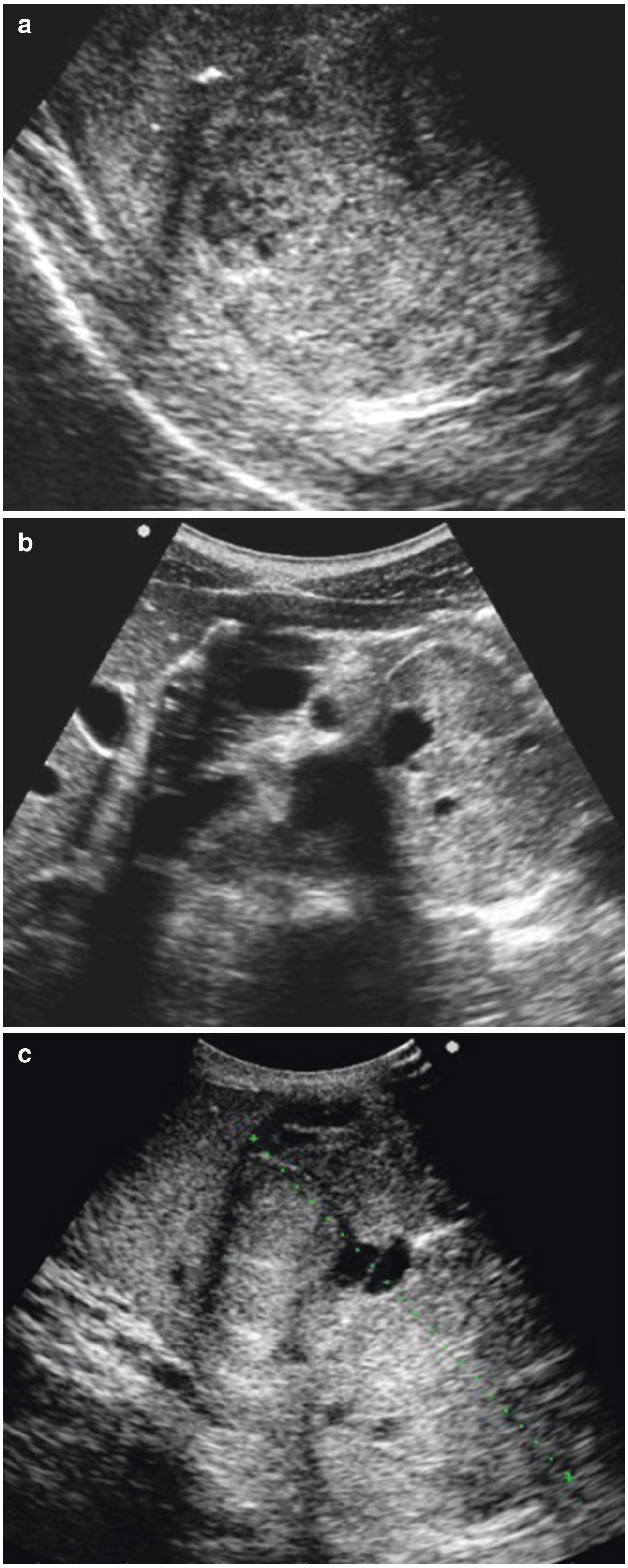


Fig. 3.21 Metastases of choriocarcinoma. Retroperitoneal lymph node metastases of choriocarcinoma of the testis (**a**–**c**) *(Courtesy of R.H. Oyen MD)*

choriocarcinoma. Elevated circulating chorionic gonadotropin levels produced by the tumours are frequent and responsible for the gynaecomastia found in 10% of cases. Pure choriocarcinoma has the worst prognosis; in mixed germ cell tumours, very high levels of hCG are associated with a poorer prognosis. Metastases may be present without any evidence of choriocarcinoma in the testis, either in the presence of a burnt-out tumour or with a primary extratesticular origin. Haemorrhage with focal necrosis of tumour is an almost invariable feature, and calcification may be present, giving a US appearance similar to other NSGCTs.

3.2.2.4 Yolk Sac Tumour

Yolk sac tumour or endodermal sinus tumour (Fig. 3.22) is the most common germ cell tumour in children under 2, accounting for 60% of testicular neoplasms in this age group. The pure form is almost exclusively found in young children, where it is the most common malignant germ cell tumour. It is rare in post-pubertal males but can be present in up to 40% of adult mixed germ cell tumours. It presents as a predominantly solid mass with heterogeneous echogenicity and anechoic spaces [2, 17, 18] (Fig. 3.23). Serum αFP is elevated in over 90% of cases. Imaging of this tumour is aspecific, and in children the only finding may be testicular enlargement without a defined mass.

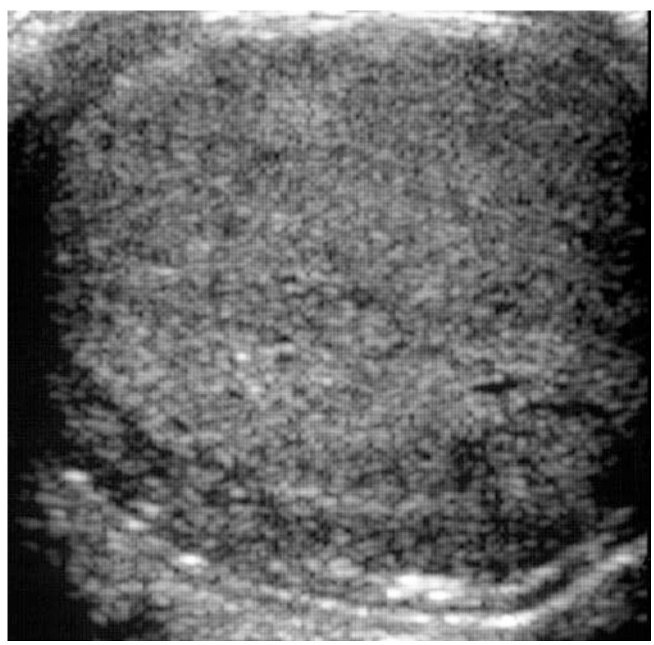


Fig. 3.22 Yolk sac tumour. Ultrasound of the left testis of a 4-month-old boy, which was painless but rapidly enlarging. There is a large, solid mass replacing the left testis, with a rind of normal testis seen peripherally. This mass was highly vascular on colour Doppler examination (not shown). Orchiectomy confirmed the lesion to represent a malignant yolk sac tumour *(From: S. Basu, D.C. Howlett, 'High-resolution ultrasound in the evaluation of the nonacute testis' Abdom Imaging (2001) 26:425–432)*

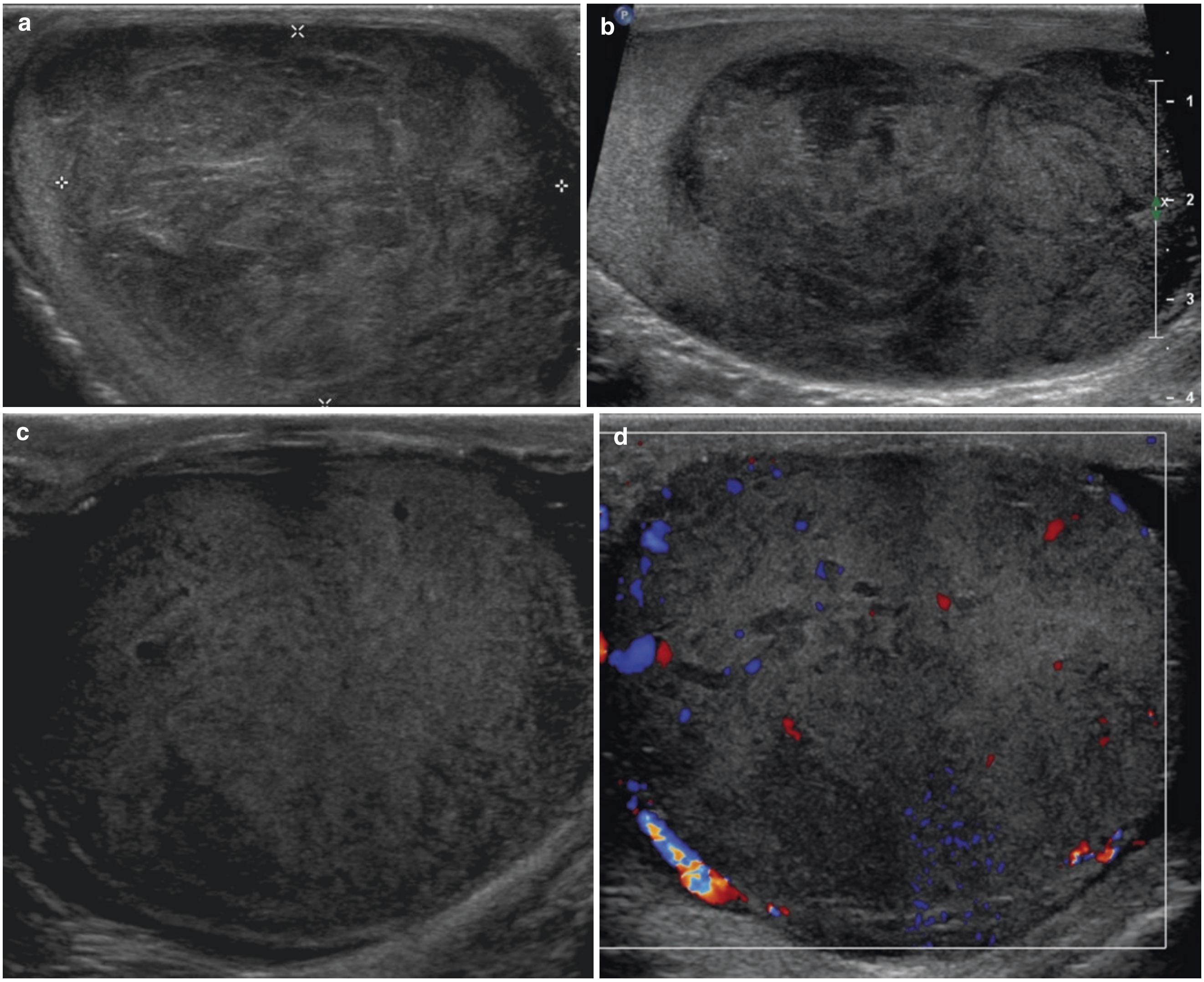


Fig. 3.23 Mixed germ cell tumour. Longitudinal ultrasound of the testis shows a large heterogeneous complex mass with cystic areas (**a**, **b**). Surgery demonstrated the presence of a mixed germ cell tumour, which consisted of seminoma and teratoma. In panels (**b**) and (**c**), a further case of a mixed germ cell tumour is shown, which consisted of seminoma, teratoma and choriocarcinoma

3.2.2.5 Mixed Germ Cell Tumours

Mixed germ cell tumours are any tumours containing more than one histological element. They account for 20–40% of primary testicular tumours and may present in numerous combinations. The average age of presentation is in the third decade of life. Embryonal cell carcinoma (Fig. 3.24) is the most frequent component, followed by teratoma (Fig. 3.25) and seminoma. The least frequent are yolk sac tumour (Fig. 3.26) and choriocarcinoma. The US findings of mixed germ cell tumours reflect the diversity of these lesions and may show areas of cysts, haemorrhage, necrosis and calcifications [16, 19].

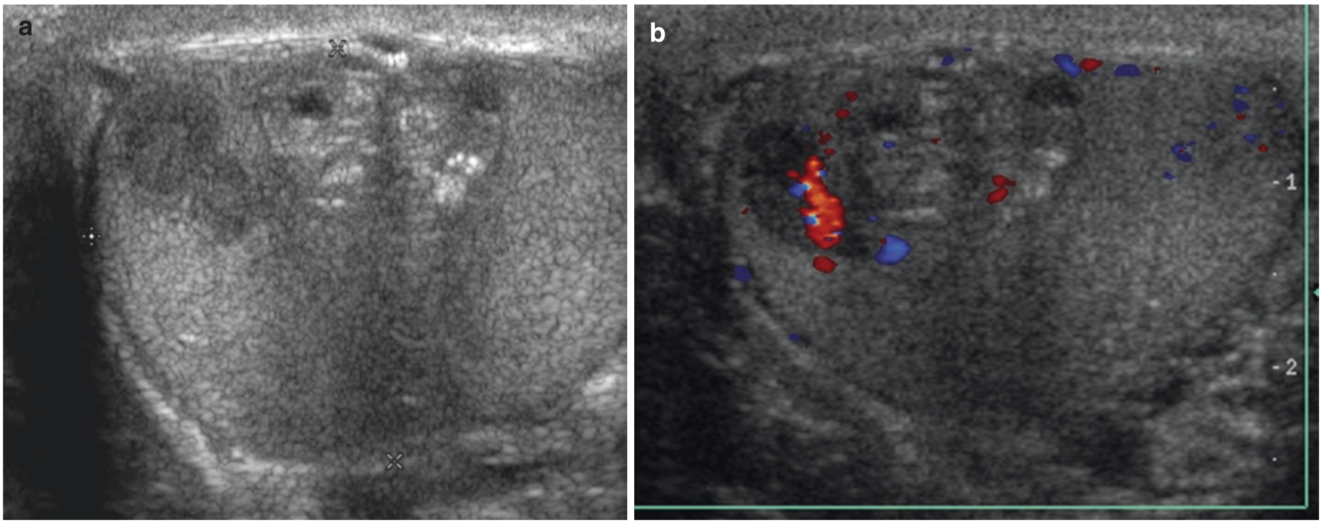


Fig. 3.24 Mixed germ cell tumour. Longitudinal ultrasound of the testis shows a well-defined heterogeneous complex mass with cystic areas (due to tumour necrosis) and hyperechogenic foci (due to calcifications). The mass was hard on palpation (**a**). Colour Doppler interrogation of the mass shows peripheral vascularity (**b**). Surgery confirmed the presence of a mixed germ cell tumour, which consisted of embryonal carcinoma and seminoma

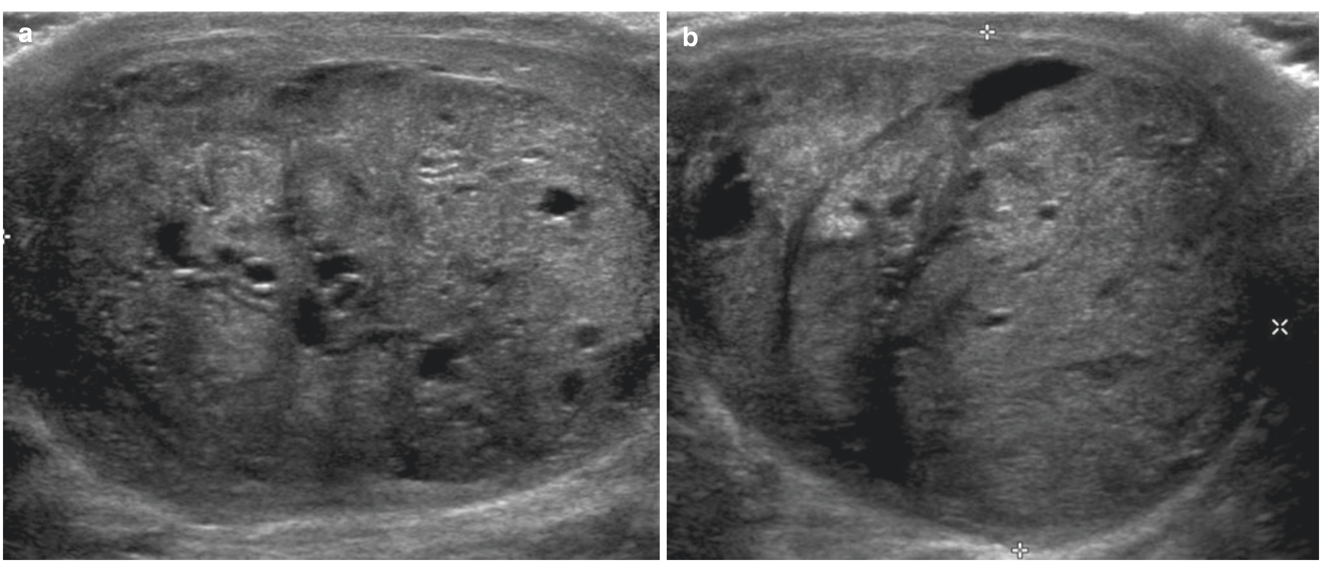


Fig. 3.25 Mixed germ cell tumour. Ultrasound of a large, mixed lesion. At histology it was shown to be a mixed tumour with teratoma, embryonal carcinoma, choriocarcinoma and yolk sac tumour elements

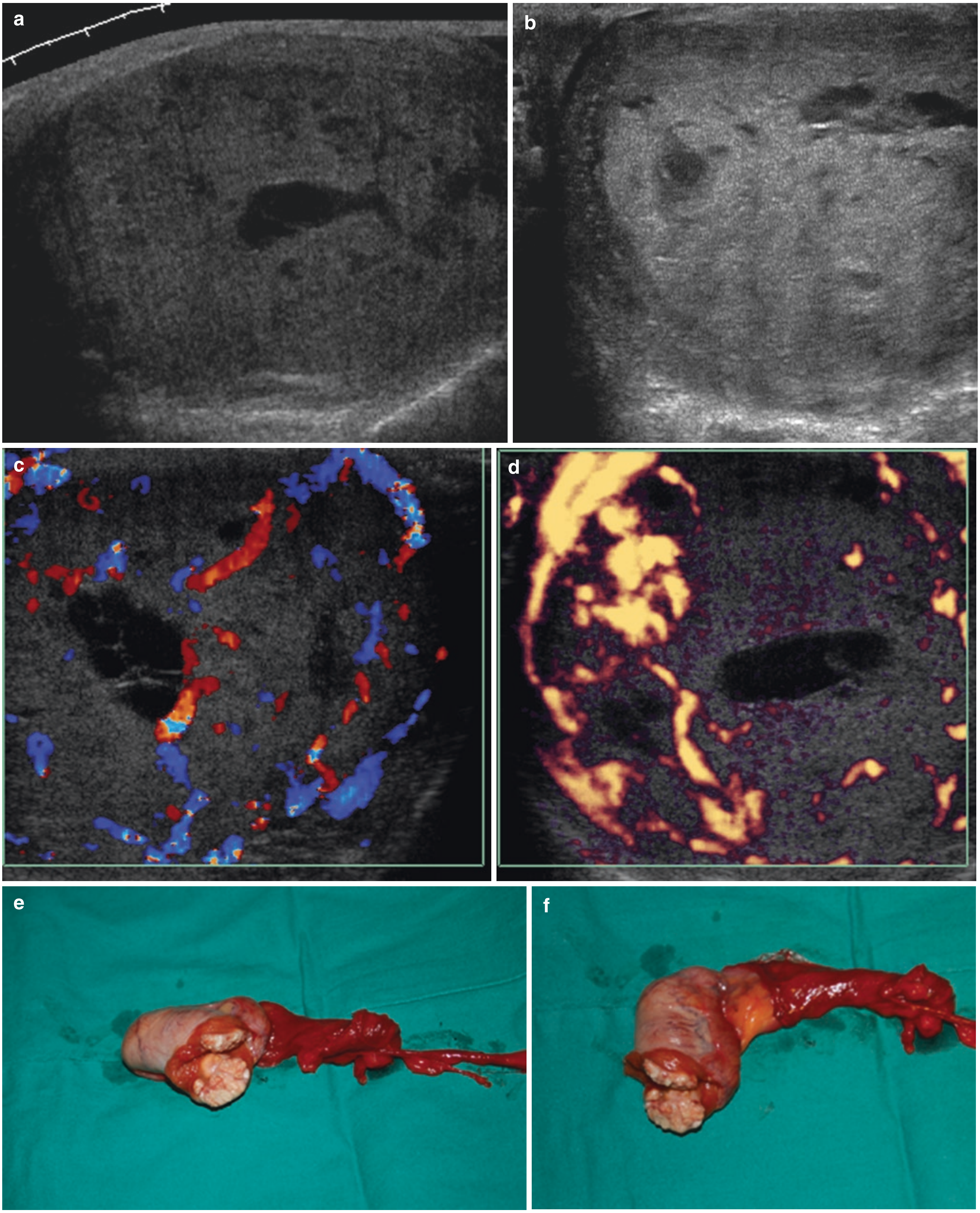


Fig. 3.26 Mixed germ cell tumour. Longitudinal ultrasound of the testis of a 25-year-old man shows a 10 cm mass replacing entirely the left testis. The lesion was solid, relatively homogeneous, with small focal cystic areas due to haemorrhagic necrosis (**a**, **b**). Colour and power Doppler studies revealed peripheral and intralesional vascularity (**c**, **d**). At histology the tumour was a mixed germ cell tumour, with equally represented embryonal carcinoma and yolk sac tumour elements. In *panels* (**e**, **f**), the gross specimen of the tumour is shown

3.2.2.6 Regressed or Primary Extragonadal Germ Cell Tumours

Regressed extragonadal germ cell tumours. A number of older and recent articles report retroperitoneal germ cell tumours in the absence of known primary testicular tumours (Fig. 3.27). These lesions may be primary retroperitoneal neoplasms or metastases from occult testicular tumours. The term 'burnt-out tumour' was introduced to describe cases in which the primary testicular tumour is reduced to a fibrotic scar with intratubular cancerous cells. In the reported cases, the most suspicious findings on US are highly echogenic foci or gross calcifications (>2–3 mm), with or without microlithiasis, in small or atrophic testes. Occasionally, burnt-out tumours can appear as hypoechoic irregular areas in a normal

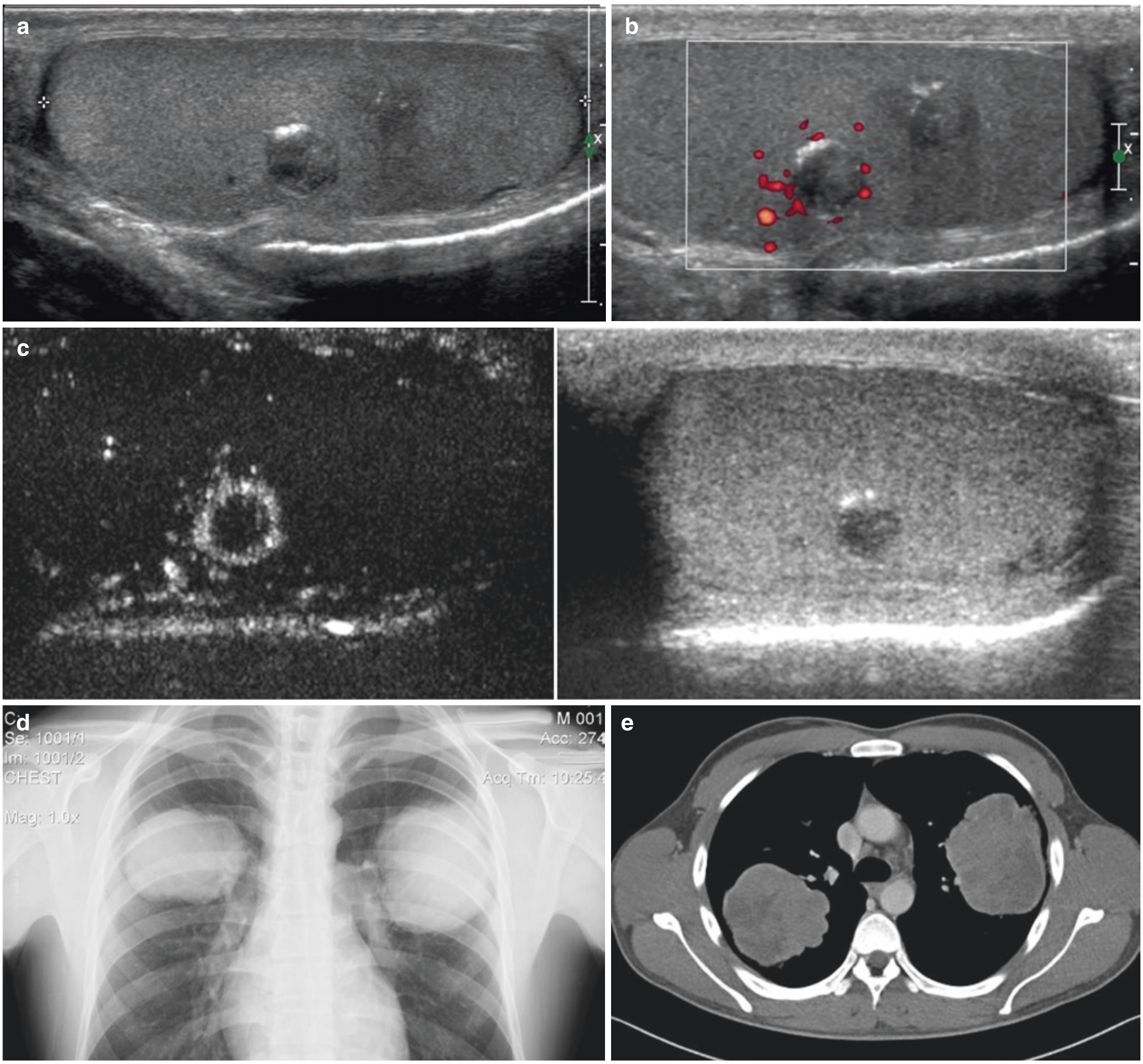


Fig. 3.27 Burnt-out tumour. US scan demonstrates two calcified hypoechoic lesions within an otherwise normal testis in a 24-year-old man (**a**). Lesion was not vascularised (**b**, **c**). Chest X-ray and CT scan shows the presence of pulmonary metastases (**d**, **e**). Histology confirmed embryonal carcinoma cells

testis. It has been proposed that the hyperechoic or calcified areas represent surrounding infarction or fibrosis, while the hypoechoic area is the residual tumour (Fig. 3.28) [20]. Nevertheless, the findings are aspecific.

Primary extragonadal tumours account for less than 5% of all germ cell tumours, and the finding of a testicular lesion has an impact on prognosis and treatment. US have a pivotal role in the search for any primary regressed tumour. US appearance and the histological components of germ cells tumours can change between the primary and secondary lesions or after treatment. A mixed germ cell tumour may evolve into a mature teratoma or into a more aggressive histological type. This further complicates diagnosis in the absence of the early detection of a primary testicular lesion.

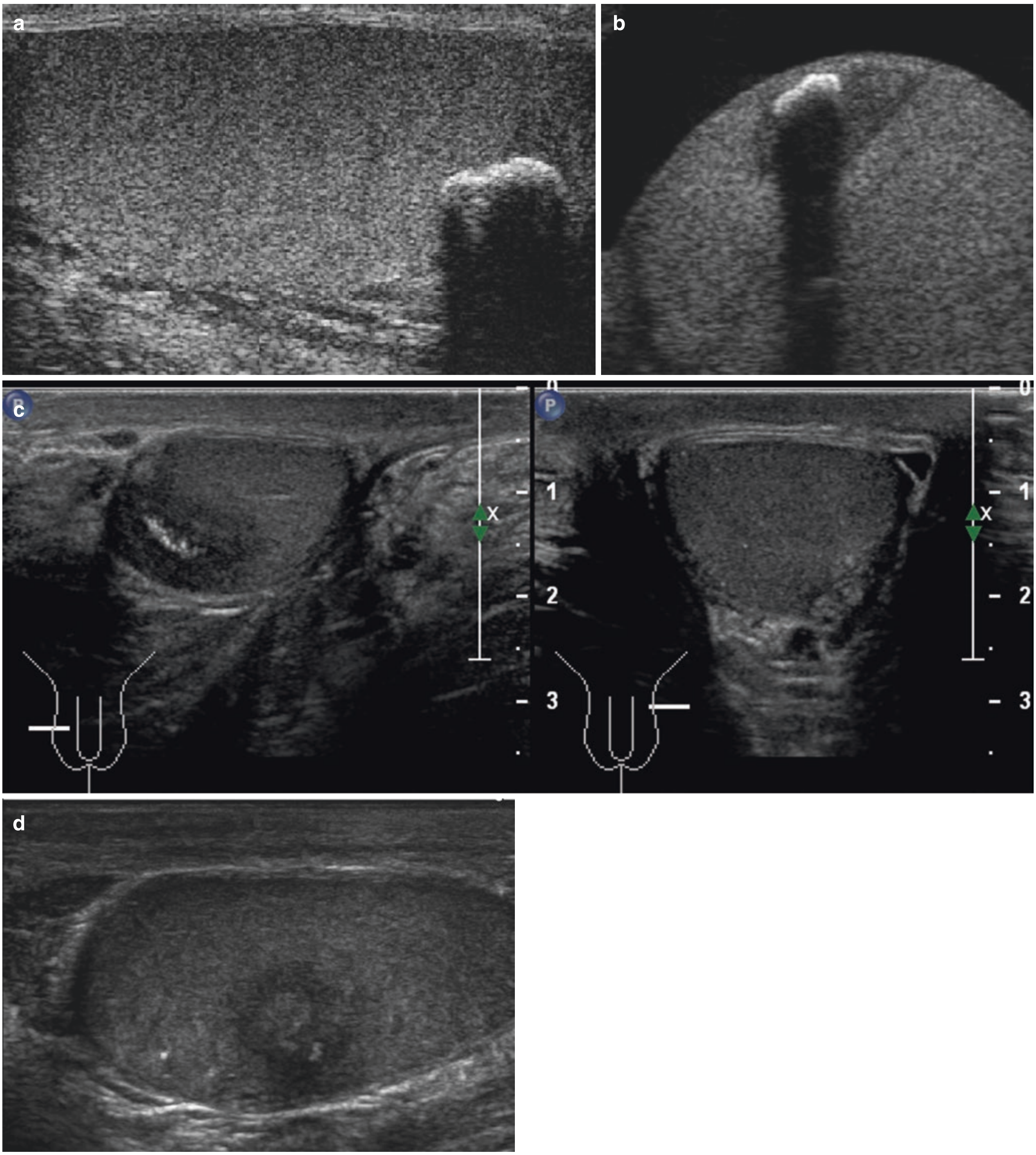


Fig. 3.28 Burnt-out tumour. A selection of burnt-out tumours. US scan show gross calcification near a hypoechoic area (that may represent the residual tumour) in an otherwise normal testis (**a**–**d**) *(panels* ***a***, ***b***, *Courtesy of R.H. Oyen MD)*

3.3 Stromal Cell Tumours

Gonadal stromal tumours account for approximately 3–15% of all testicular neoplasms. The prevalence is higher in children, where non-germ cell tumours account for 10–30% of all tumours. The term gonadal stromal tumour refers to a neoplasm arising from the sex cords or interstitial cells and containing Leydig, Sertoli, thecal, granulosa or lutein cells and fibroblast in various degrees of differentiation. Over 90% of these tumours are benign; however, even with histological analysis, it is difficult to determine their biological behaviour. For this reason a testis-sparing enucleation is generally performed in all cases, or, in selected cases, when clinical and sonographic findings permit a 'watchful-waiting' approach, follow-up can be adopted with serial US every 3 months for a minimum of 18 months, provided these patients have a previous negative point-of-diagnosis CT or MR examination confirming absent abdominal lymph nodes, a normal chest X-ray or CT and negative tumoural markers.

3.3.1 Leydig Cell Tumours

Leydig cell tumours (LCTs), or interstitial cell tumours, are the most common of the non-germ cell testicular tumours, accounting up to 10% of all testicular tumours [21]. Although most are benign, about 10% have a malignant course [25]. They occur predominantly in patients between the ages of 20 and 50 years. Approximately 30% of patients present with symptoms related to hormonal activity: gynaecomastia, precocious pseudopuberty or azoospermia [16]. Impotence and loss of libido may also occur in young men. Some patients complain of testicular swelling, which may or may not be associated with a palpable mass. However, over 10% of patients in a large series had no symptoms related to their tumours, and LCTs were found by chance at US examinations performed for other indications [25].

Recent studies show that Leydig cell tumours are among the most frequent histotypes found in small, non-palpable intratesticular lesions in infertile men (Fig. 3.29), in whom – probably

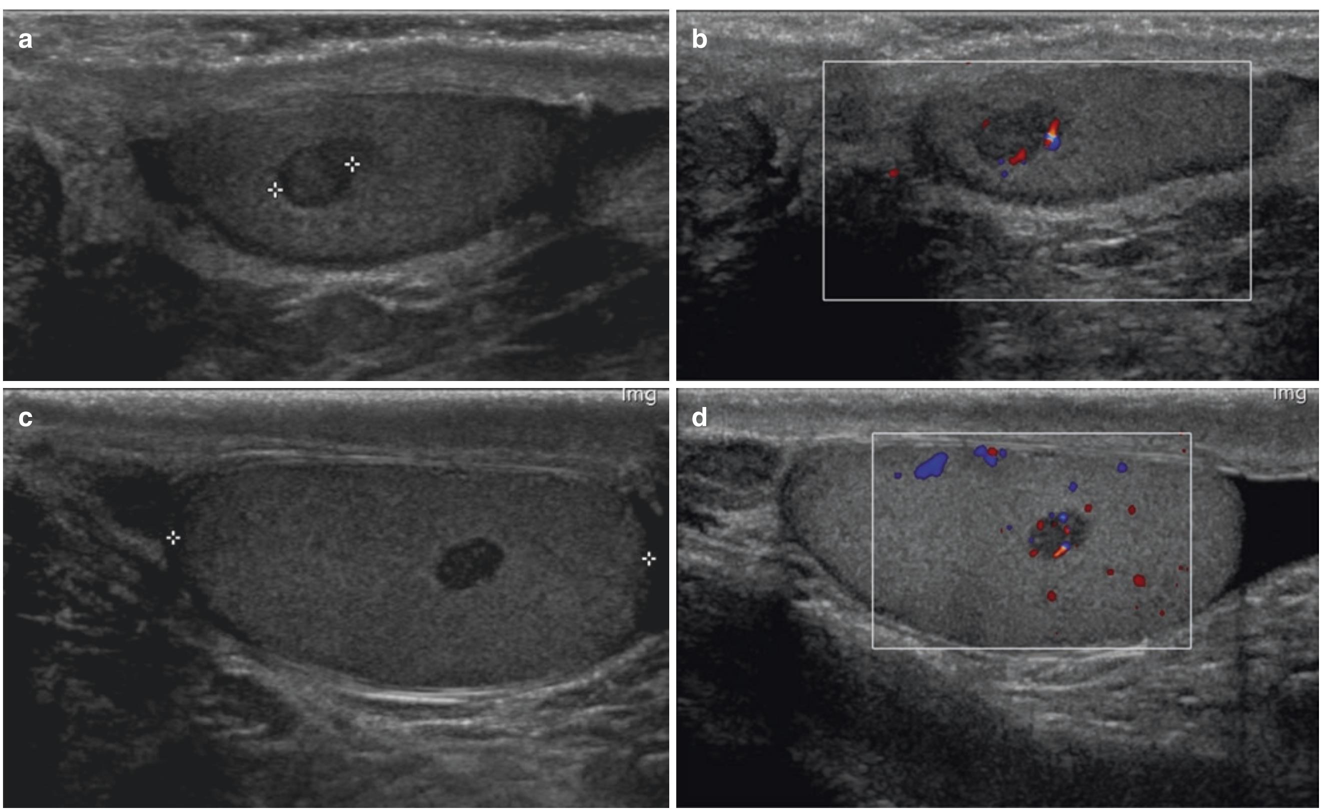


Fig. 3.29 Typical Leydig cell tumour. A selection of cases of solitary, hypoechoic lesions, with peripheral and intralesional vessels, found to be Leydig cell tumours at histology

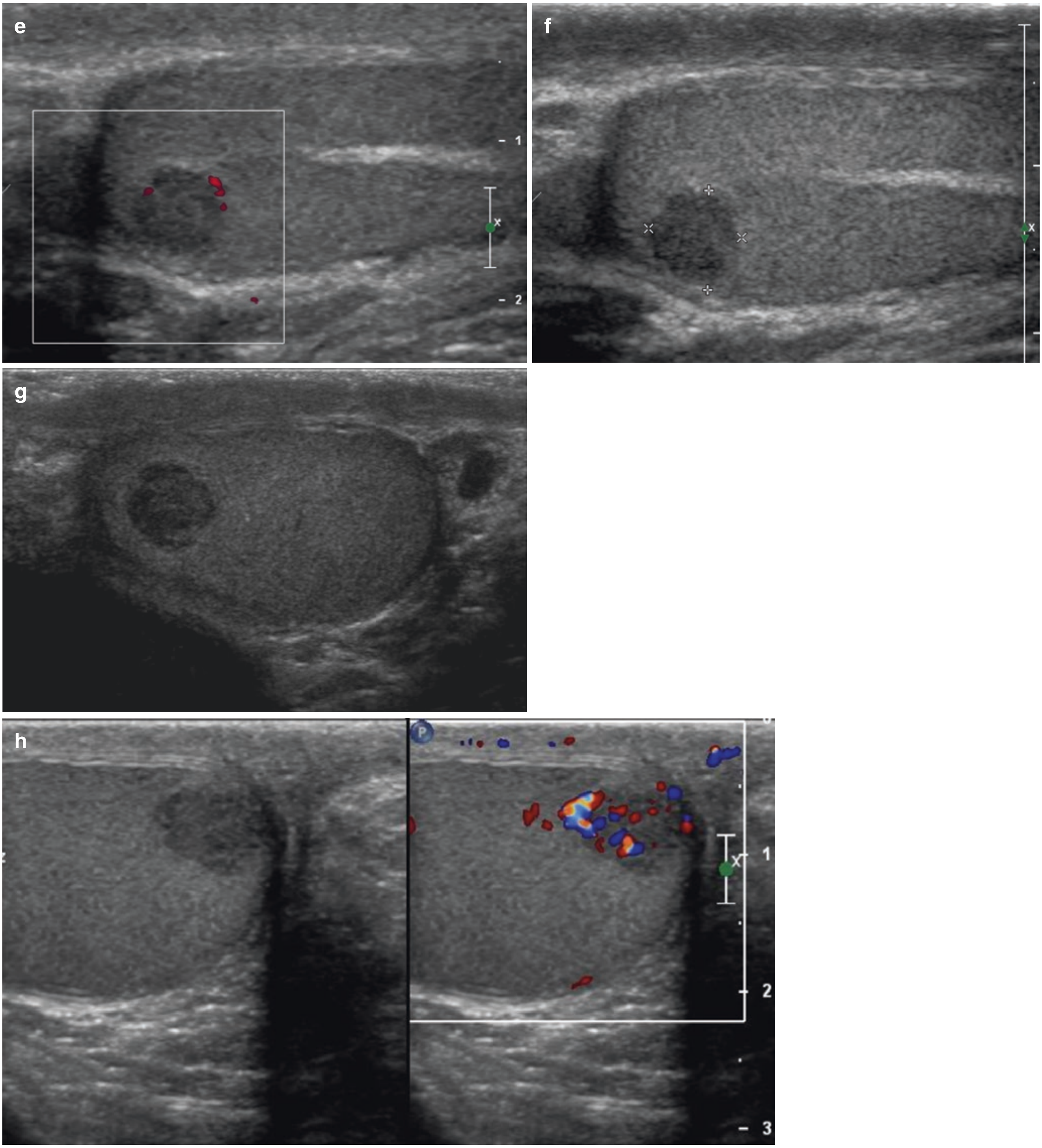


Fig. 3.29 (continued)

due to their reduced size – they seldom produce signs and symptoms of excess hormone (Fig. 3.30). As most Leydig cell tumours secrete oestrogens, the use of hCG stimulation test with measurement of the testosterone-to-oestradiol ratio has been proposed as a more accurate screening test. Alternatively, measurement of steroid hormone precursor (17-OH-progesterone or androstenedione) has been advocated. There is insufficient data to estimate the sensitivity and specificity of these investigations. Contrast-enhanced ultrasound and elastography have been advocated to increase the diagnostic accuracy of plain ultrasound [21, 22] even if the final diagnosis remains histological, even for these tumours (Fig. 3.31).

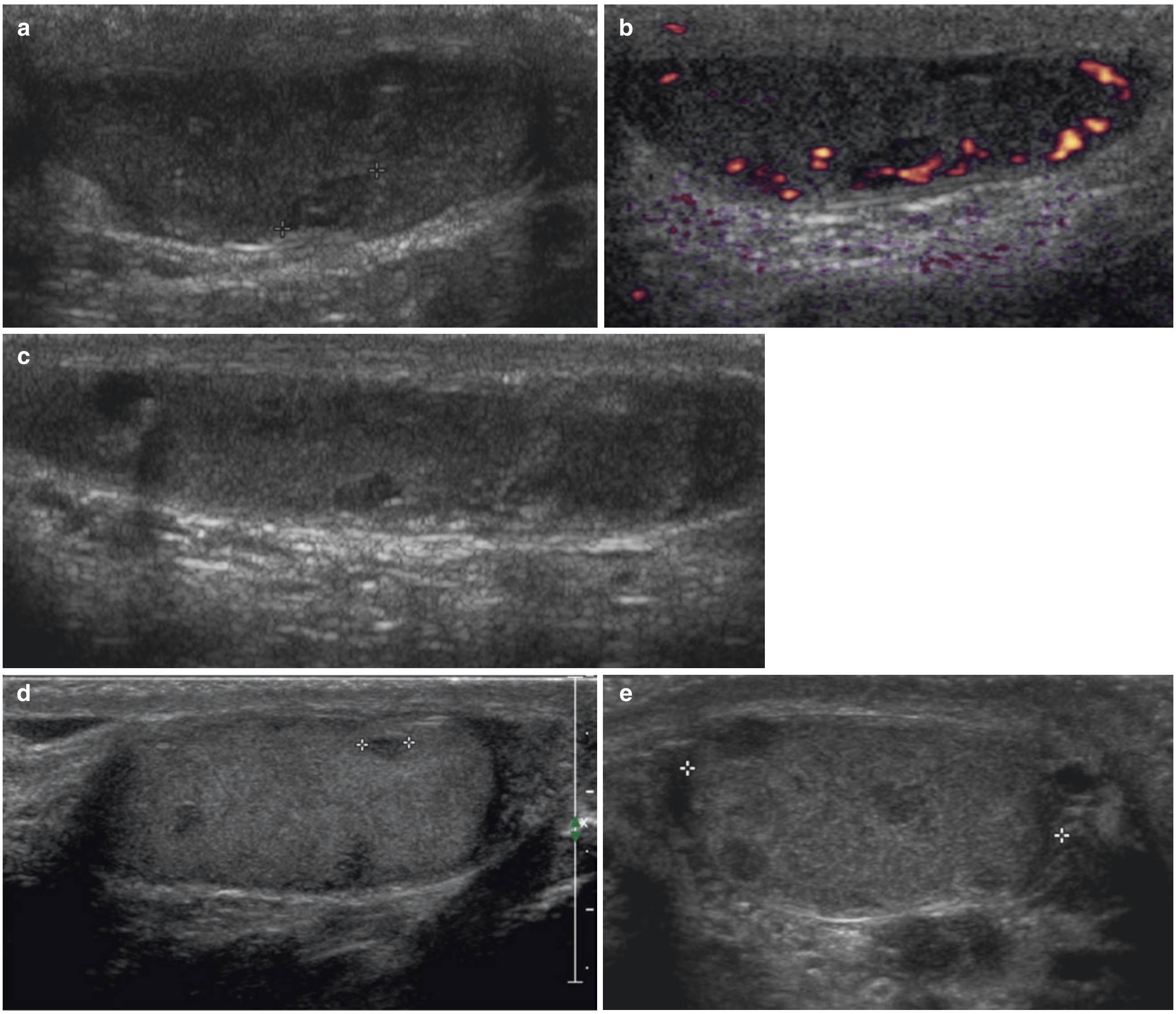


Fig. 3.30 Leydig hyperplasia. Ultrasound scans from a 35-year-old man with Klinefelter's syndrome (**a**–**c**), showing small testes with inhomogeneous echotexture and the presence of multiple irregularly shaped, non-palpable lesions (vs. single, well-defined masses) usually found in patients with gonadal dysgenesis. Clinical history helps in the differential diagnosis between Leydig hyperplasia and tumour. In panels (**d**) and (**e**), further cases of Leydig hyperplasia are shown

Leydig cell tumours are most frequently small, unilateral and solitary (Fig. 3.32), although 3% are bilateral [25]. The literature reports that approximately 10% are malignant, having invaded the tunica at diagnosis. In our experience this percentage is probably smaller. Gonadal tumours are usually seen on ultrasound as homogeneous, hypoechoic, solid, well-demarcated and vascularised lesions (Fig. 3.33). Cystic space resulting from haemorrhage and necrosis is rarely seen and only in larger lesions (Fig. 3.34).

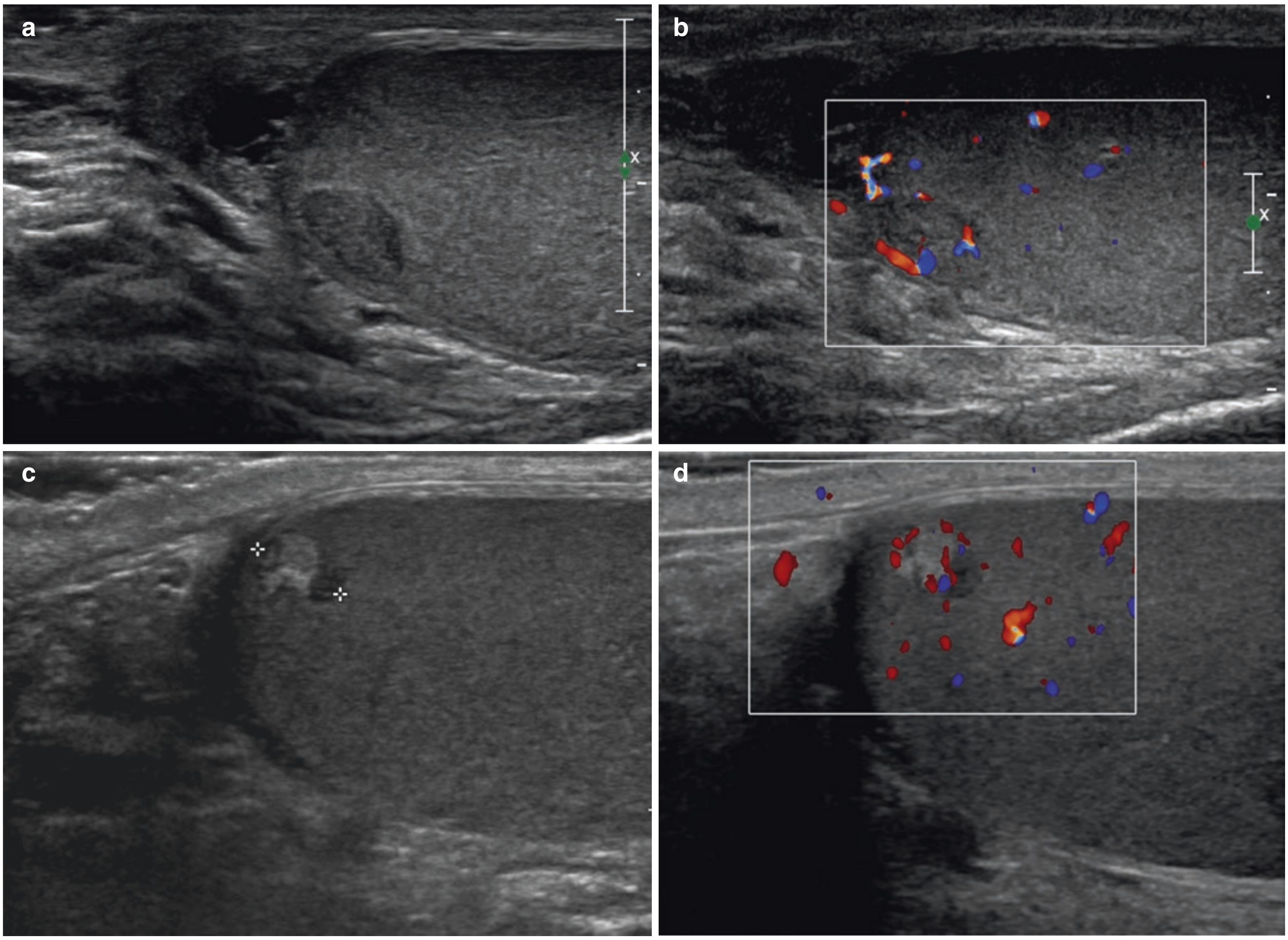


Fig. 3.31 Leydig cell tumour. Sonogram showing two histologically proven Leydig cell tumours, with different echogenicity

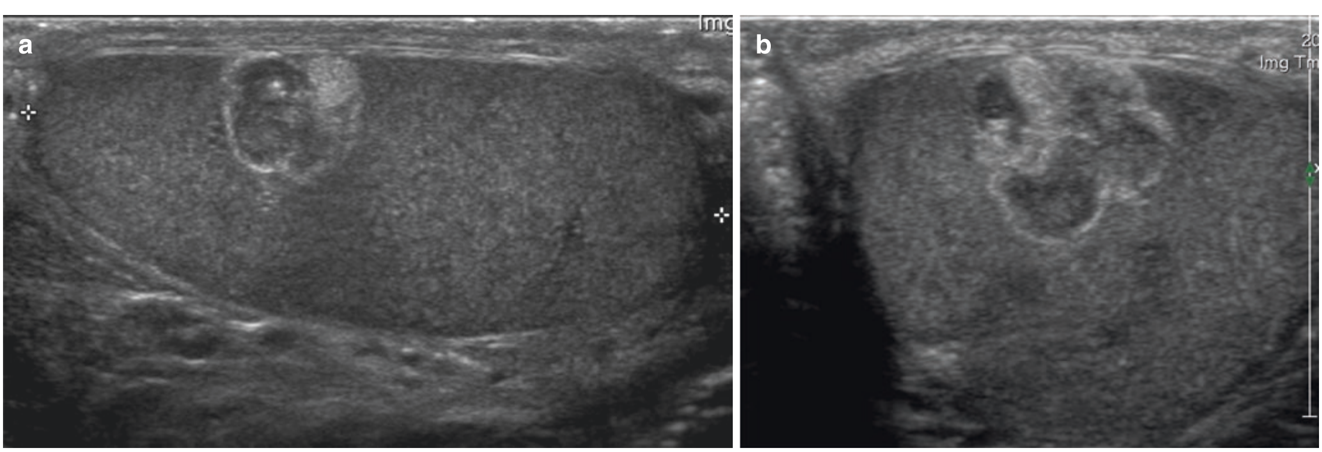
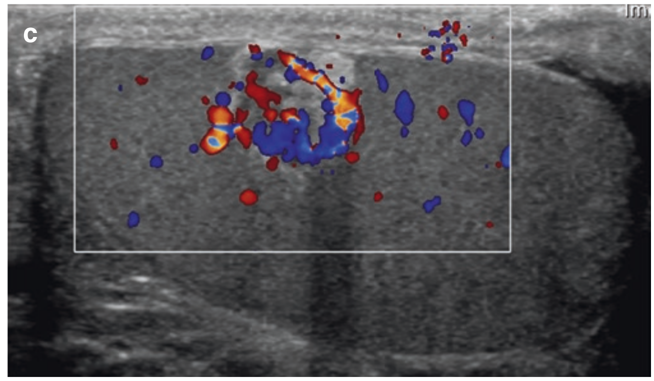


Fig. 3.32 Solitary Leydig cell tumour. Sonogram showing an inhomogeneous tumour in the anterior aspect of the testis (**a**). Colour Doppler image showing intralesional vascularisation of the tumour

Fig. 3.32 (continued)



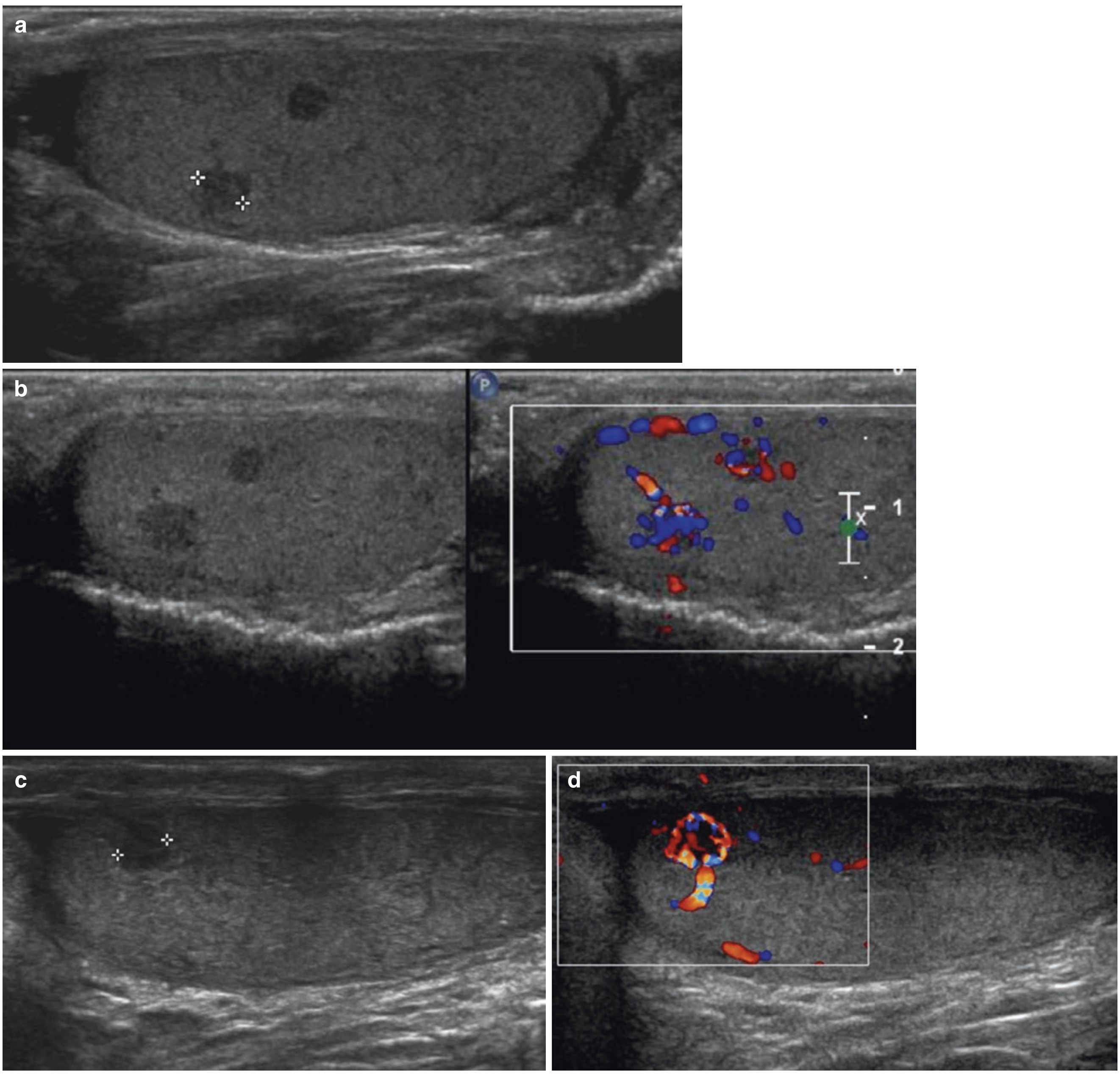


Fig. 3.33 Multifocal Leydig cell tumour. Images from a 45-year-old man with hereditary leiomyomatosis and incidentally revealed lesions in the right testis. Sonogram of the testis shows two small focal lesions (**a**). Colour Doppler image shows intralesional vascularisation in both lesions (**b**, **c**)

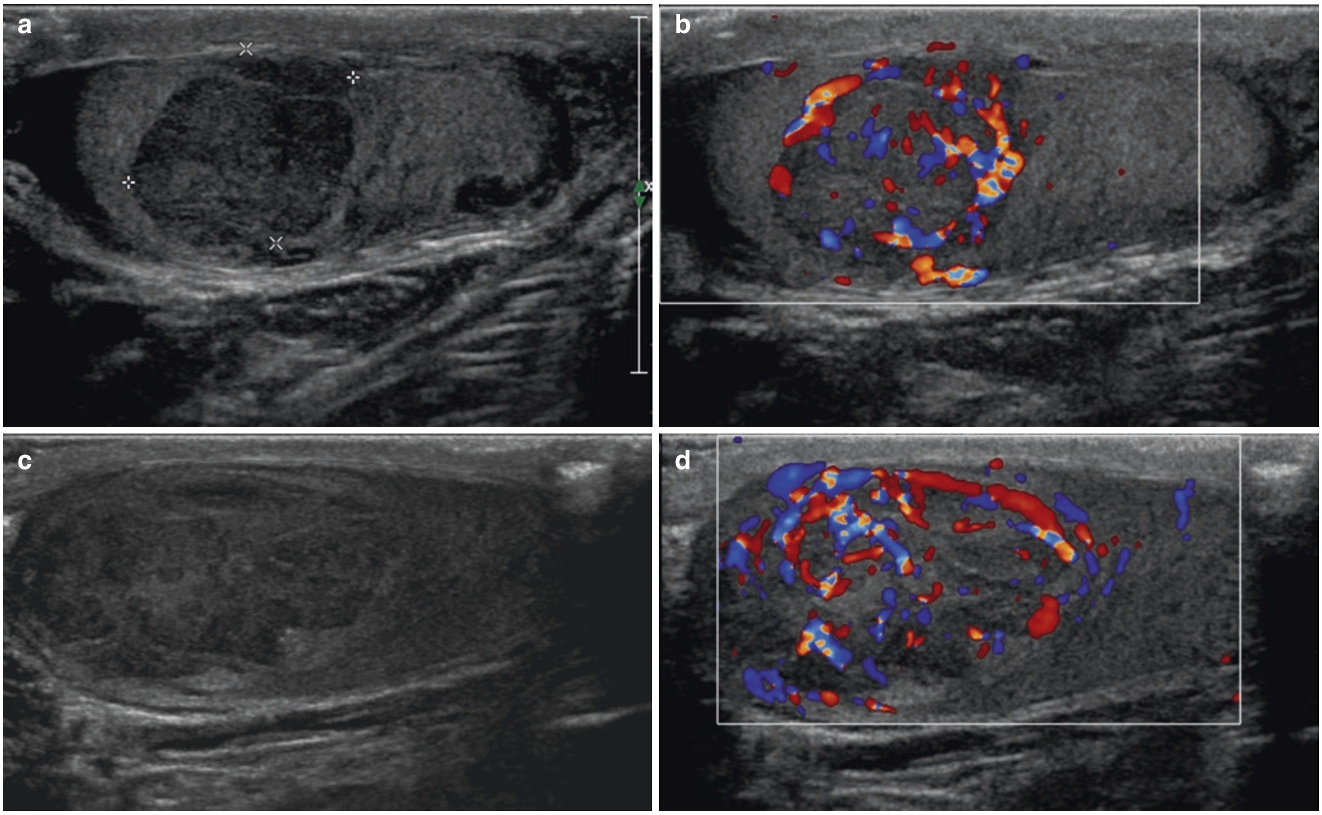


Fig. 3.34 Atypical Leydig cell tumour. Large inhomogeneous masses, highly vascularised, in two different patients, both resulted in Leydig cell tumour at histology

3.3.2 Sertoli Cell Tumours

Sertoli cell tumours are much less common and account for less than 1% of all testicular tumours. The most common presentation is with a painless testicular mass. They are less likely to be hormonally active, but gynaecomastia has been described.

Sertoli cell tumours may occur in undescended testes and in patients with testicular feminisation, Klinefelter's syndrome and Peutz-Jeghers syndrome [26]. There are two clear-cut types of large-cell calcifying Sertoli cell tumour: (a) those associated with complex dysplastic syndromes and which are bilateral and multifocal and (b) those which are not associated with such syndromes and are unilateral and focal.

These tumours appear as single or multiple masses, either hypo- or hyperechoic, with possible calcification. A particular subtype is the large-cell calcifying Sertoli cell tumour (Fig. 3.35), which is most often seen in paediatric patients and can be recognised by its diffusely heterogeneous pattern, with increased echogenicity and large areas of calcification [16]. This subgroup has been associated with Peutz-Jeghers and Carney syndrome.

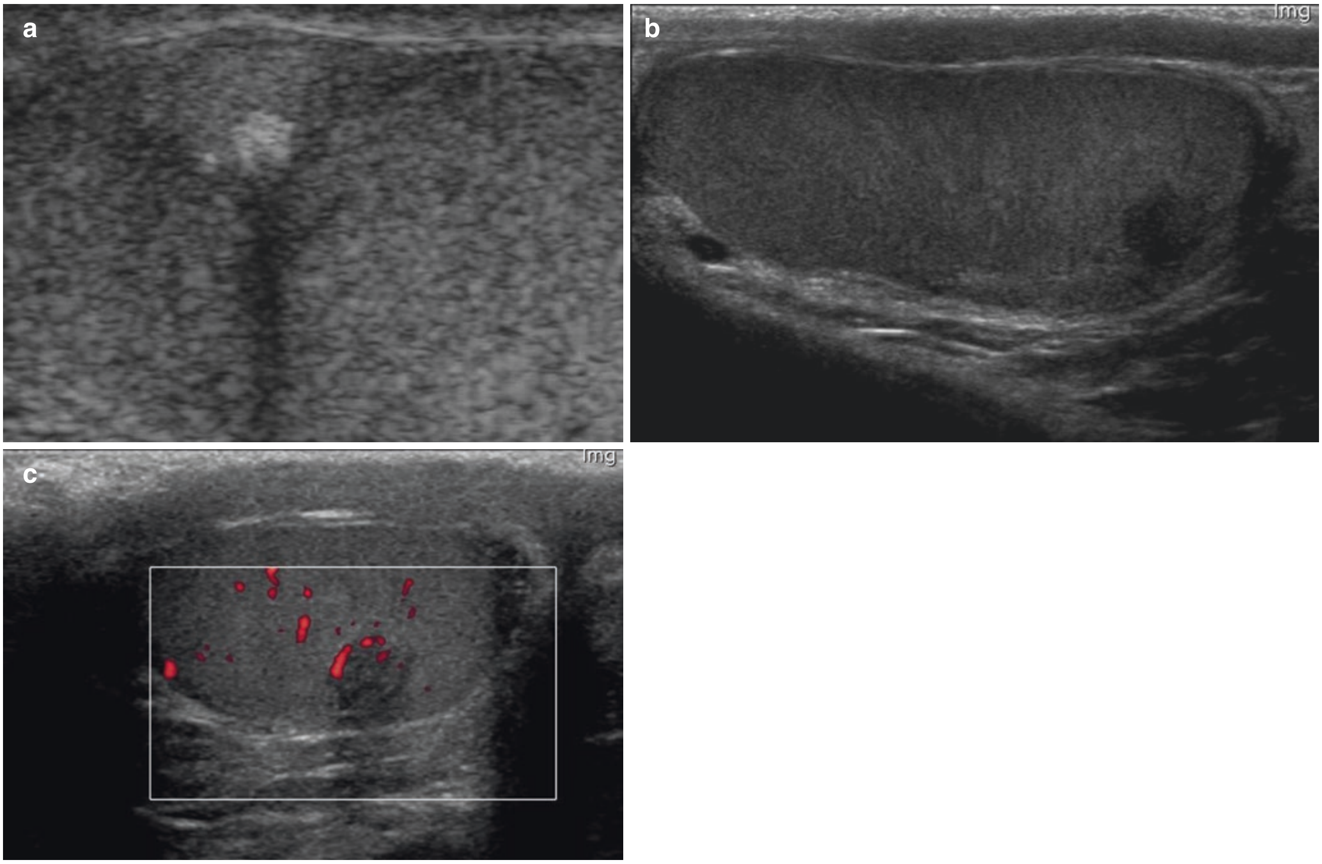


Fig. 3.35 Sertoli cell tumour. Large-cell calcifying Sertoli cell tumour (**a**), with increased echogenicity and large area of calcification *(Courtesy of: R.H. Oyen, MD)* and a hypoechoic Sertoli cell tumour (**b**, **c**), with irregular margins and intralesional vascularisation

3.4 Other Malignant Tumours

Other malignant tumours of the testis include lymphoma, the most common (5% of all testicular tumours), followed by leukaemia and less frequently metastasis arising from prostate, lung, gastrointestinal tract, skin and kidney cancers [24].

3.4.1 Lymphoma

Lymphoma is one of the most common neoplasms in men aged over 50 [23], accounting for 25% of all testicular malignancies in this age group. In men under 50, it represents less than 2% of testicular malignancies [2].

Lymphoma can occur in the testis as a primary or secondary site of involvement. It is also the most common bilateral testicular tumour, with synchronous or asynchronous involvement of the contralateral testis in 8.5–18% of cases [23, 27]. The epididymis and spermatic cord are frequently involved. The histological subtype is virtually limited to non-Hodgkin's large B-cell type [24].

Patients with testicular lymphoma generally present with a marked swelling of the testis and, less frequently, with constitutional symptoms such as weight loss, anorexia, fever and weakness.

Ultrasound usually shows either homogeneously hypoechoic testes in patients with diffuse infiltration (Fig. 3.36) or multifocal hypoechoic lesions of various

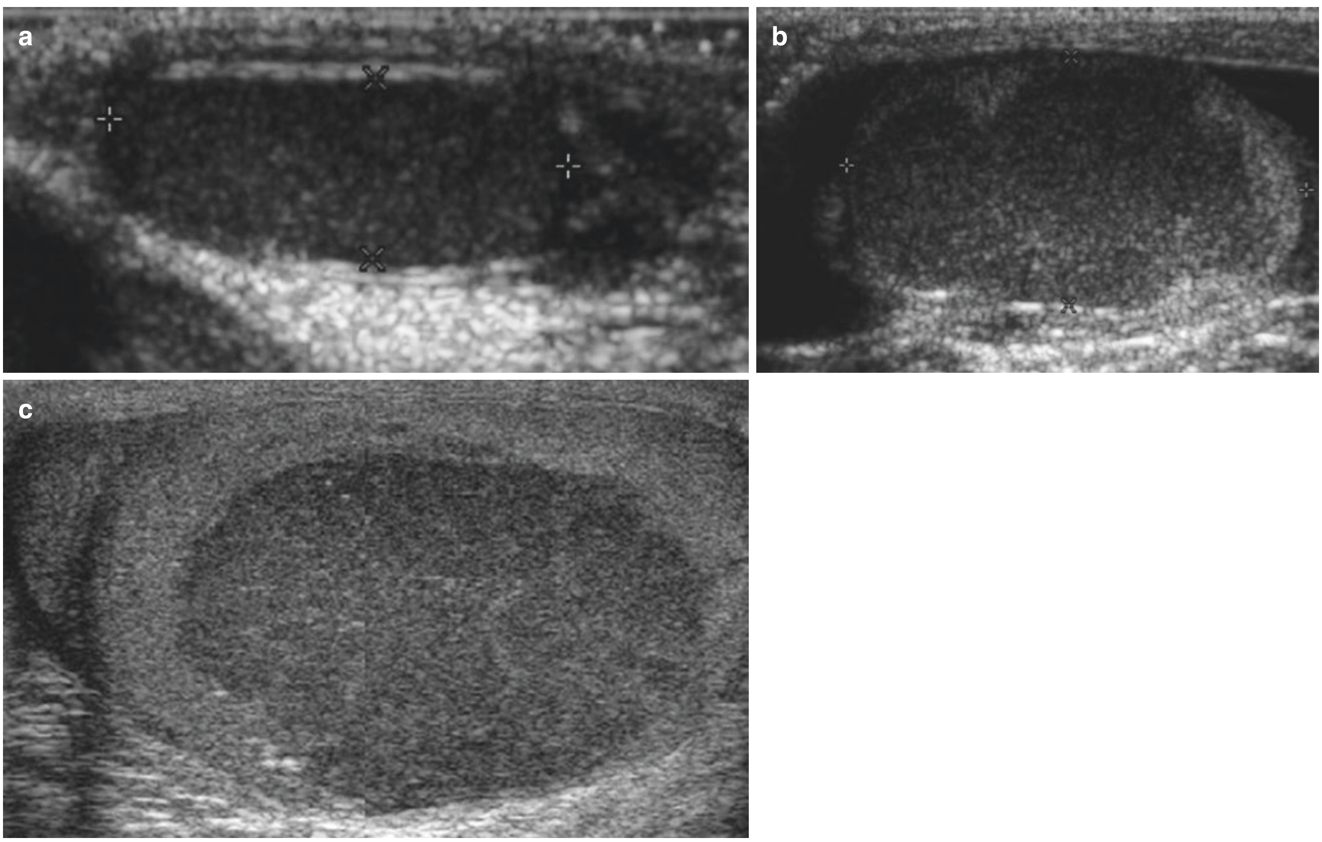


Fig. 3.36 Testicular lymphoma. A 4-year-old boy presents with painless swelling of the right scrotum. Sonographic examination demonstrates an enlarged right testicle that has abnormal heterogeneous echotexture (**a**, **b**). The abnormality was shown to be lymphoma at biopsy. Testicular lymphoma. The whole testis is involved with a hypoechoic, inhomogeneous tumour (**c**, **d**). In panel (**e**) a paratesticular B-cell lymphoma is shown. Transverse US image of the scrotum shows the normal right testis and the enlarged inhomogeneous left testis (**f**), which resulted in a lymphoma at histology (*panels* **a**–**c**, *From: A.F. Wittenberg, T. Tobias, M. Rzeszotarski et al., 'Sonography of the Acute Scrotum: The Four T's of Testicular Imaging' Curr Probl Diagn Radiol 2006;35:12–21; panels* **c**–**e**, *Courtesy of: R.H. Oyen, MD; panel* ***f***, *Courtesy: of Malai Muttarak. MD)*

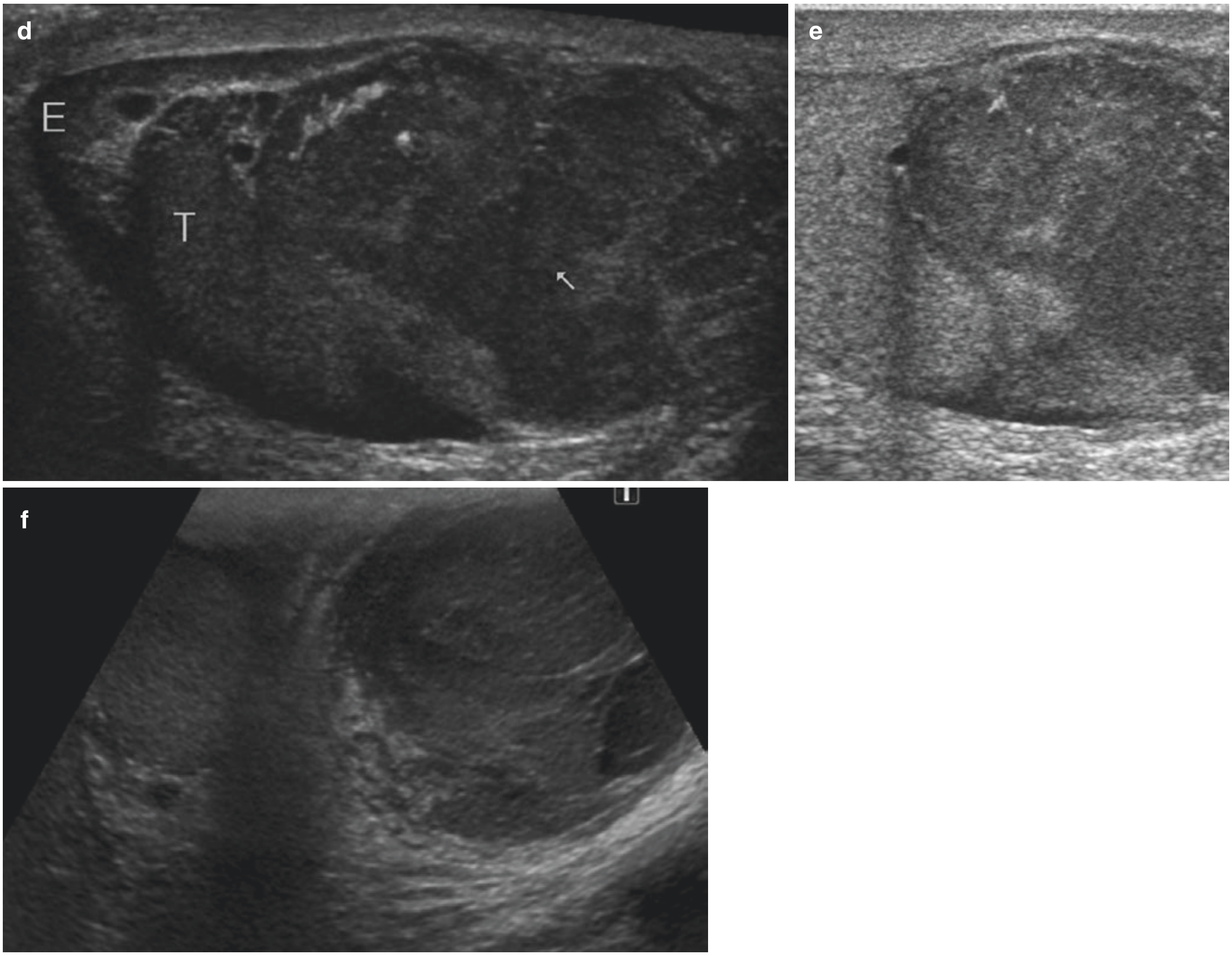


Fig. 3.36 (continued)

sizes (mean lesion size 16 mm, range 8–26 mm) [28–30]. In some cases, parallel hypoechoic lines radiating peripherally from the mediastinum testis are seen; these represent blood vessels crossing through the lesion (Figs. 3.37 and 3.38) [2]. The patient's age at presentation, symptoms, multiplicity and bilaterality of the lesion are all important factors in making the appropriate diagnosis.

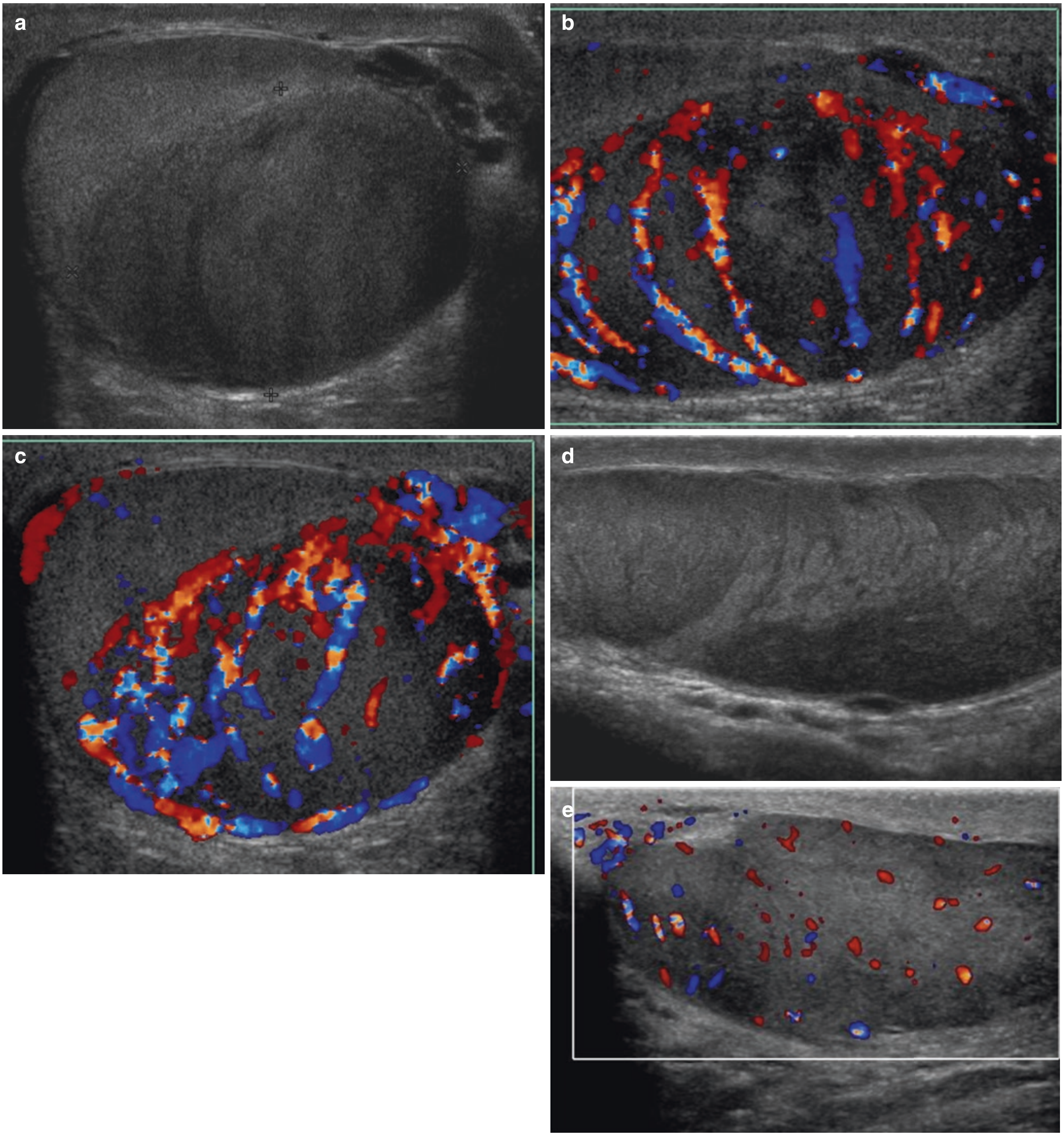


Fig. 3.37 Testicular lymphoma. **Testicular lymphoma** in a 58-year-old man. On greyscale ultrasound, lymphoma appears as a hypoechoic, inhomogeneous tumour nearly filling the testis (**a**). Parallel hypoechoic lines radiating peripherally from the mediastinum testis represent blood vessels crossing through the lesion. Colour Doppler shows increased vascularity of the lesion (**b**, **c**). **Testicular lymphoma** in a 42-year-old man. The testis is enlarged and shows several poorly defined hypoechoic masses that are infiltrating along the septa (**d**, **e**)

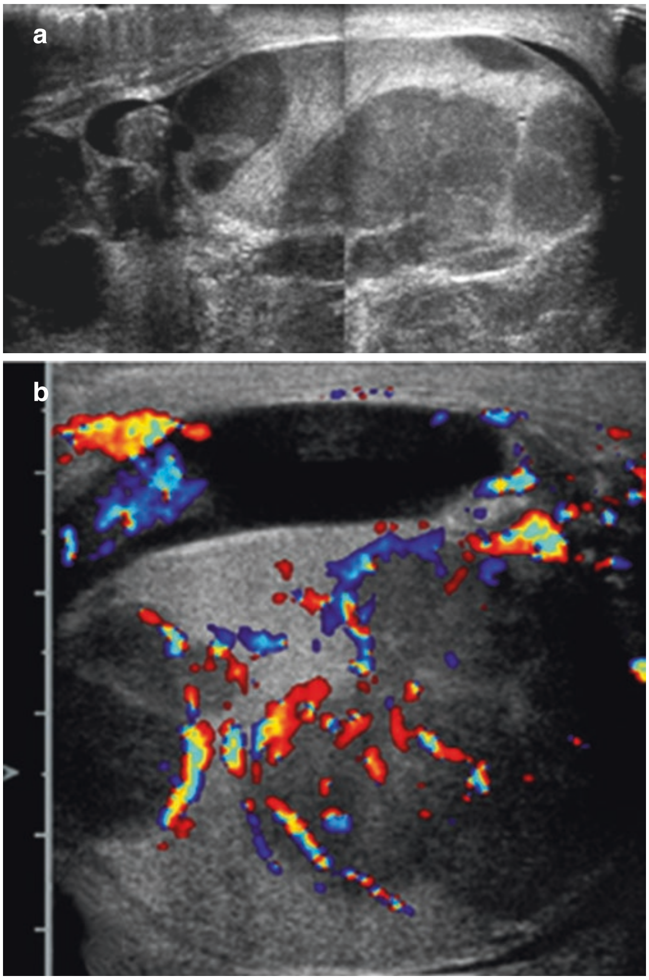


Fig. 3.38 Testicular lymphoma. Multiple echopoor areas are noted throughout the enlarged testis (**a**) with increased peripheral blood flow (**b**) *(From: D.D. Cokkinos, E. Antypa, P. Tserotas, et al. 'Emergency Ultrasound of the Scrotum: A Review of the Commonest Pathologic Conditions' - Curr Probl Diagn Radiol 2011;40:1–14)*

3.4.2 Leukaemia

Primary testicular **leukaemia** is rare, but leukaemia frequently recurs in the testes, particularly in children [24]. Because of the blood-testis barrier, the testis is a 'sanctuary' for leukaemic cells to escape chemotherapy.

Leukaemic infiltration to the testis has been found at autopsy in 40–65% of patients with acute leukaemia and in 20–35% of cases of chronic leukaemia. Testicular involvement is reported in 5–10% of boys with acute lymphoblastic leukaemia, with the majority found during clinical remission [23].

Leukaemia can present different US patterns: unilateral or bilateral, diffuse or focal and hypoechoic or hyperechoic. One type is the infiltrating pattern, with irregular diffuse hypoechoic longitudinal *striae* involving the entire testis (Fig. 3.39). In these cases it may be very hard to recognise the disease on US examination [2, 29].

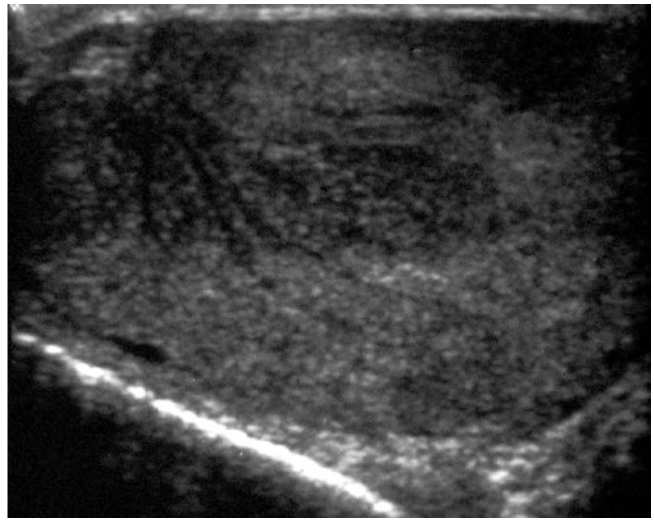


Fig. 3.39 Leukaemia – mass infiltration pattern. Ultrasound of the left testis in a male in remission after bone transplantation for acute lymphoblastic leukaemia. The testis is enlarged and contains several poorly defined hypoechoic masses that are infiltrating along the septa. Similar changes were evident in the right testis, and biopsy confirmed leukaemic infiltration *(From: D.C. Howlett, N.D.P. Marchbank, D.F. Sallomi, 'Ultrasound of the Testis' Clinical Radiology (2000) 55, 595 ± 601)*

Colour Doppler US reveals increased intralesional flow in all areas of lymphomatous or leukaemic involvement, irrespective of lesion size. Differentiation from inflammatory processes of the testis remains difficult. The clinical symptoms are often helpful in the diagnosis [2].

3.4.3 Plasmacytoma

Involvement of the testis is usually a manifestation of diffuse myeloma. The testis may have single or multiple nodules that appear hypoechoic and homogeneous on US examination (Fig. 3.40) [31–33]. Bilateral involvement occurs in approximately 20% of cases.

3.4.4 Testicular Carcinoid

Carcinoid tumour is an extremely rare neoplasm of the testis and represents 0.23% of testicular neoplasm. Testicular carcinoids are classified according to the following three groups: the majority are primary pure testicular tumours, alternatively they occur as part of complex teratomas, and finally, a minority present as secondary metastatic tumours. The ultrasound appearance of testicular carcinoids is not specific, including a well-defined hypoechoic intratesticular mass containing dense calcification (Fig. 3.41); the differential diagnosis includes germ cell tumours [34, 35].

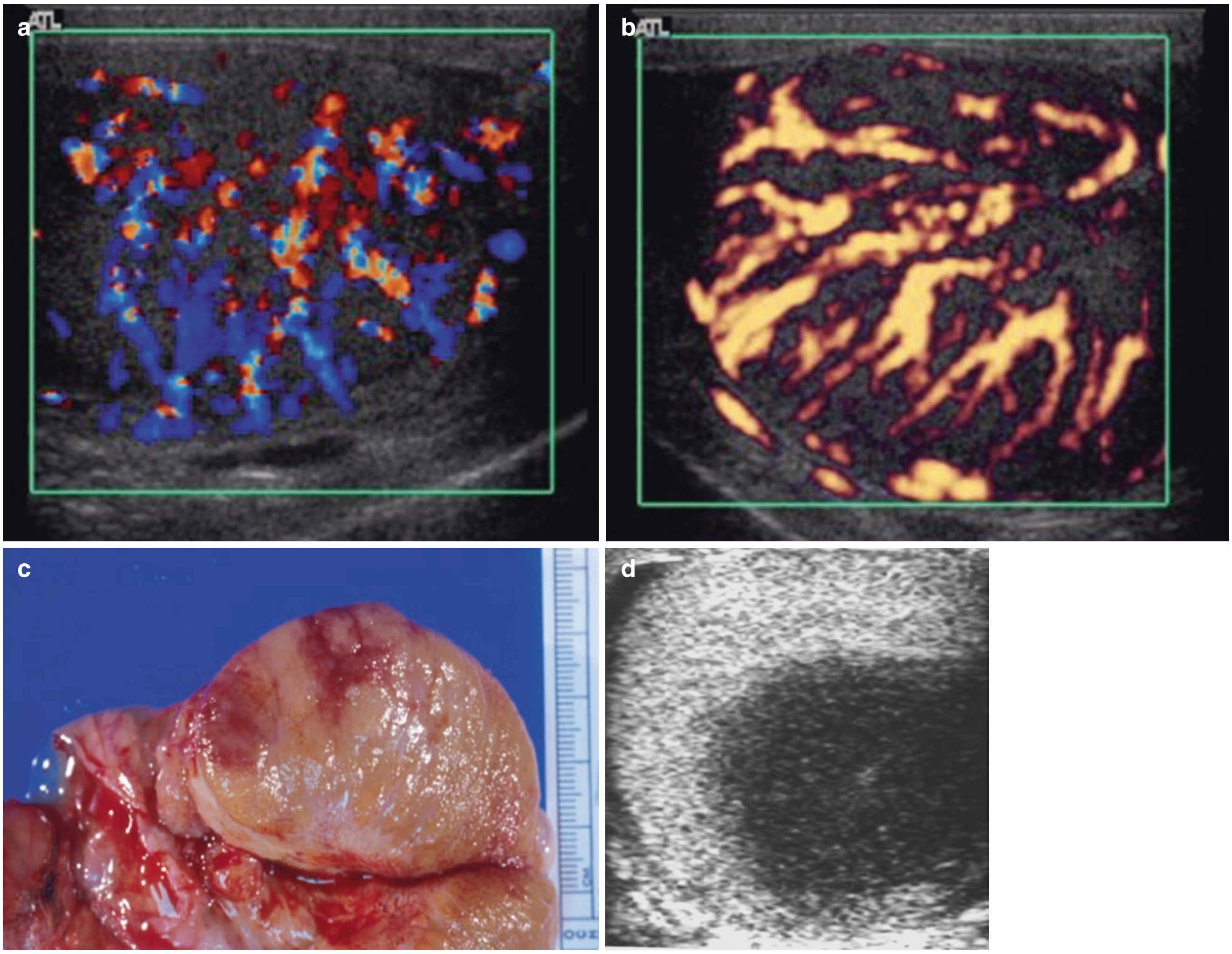
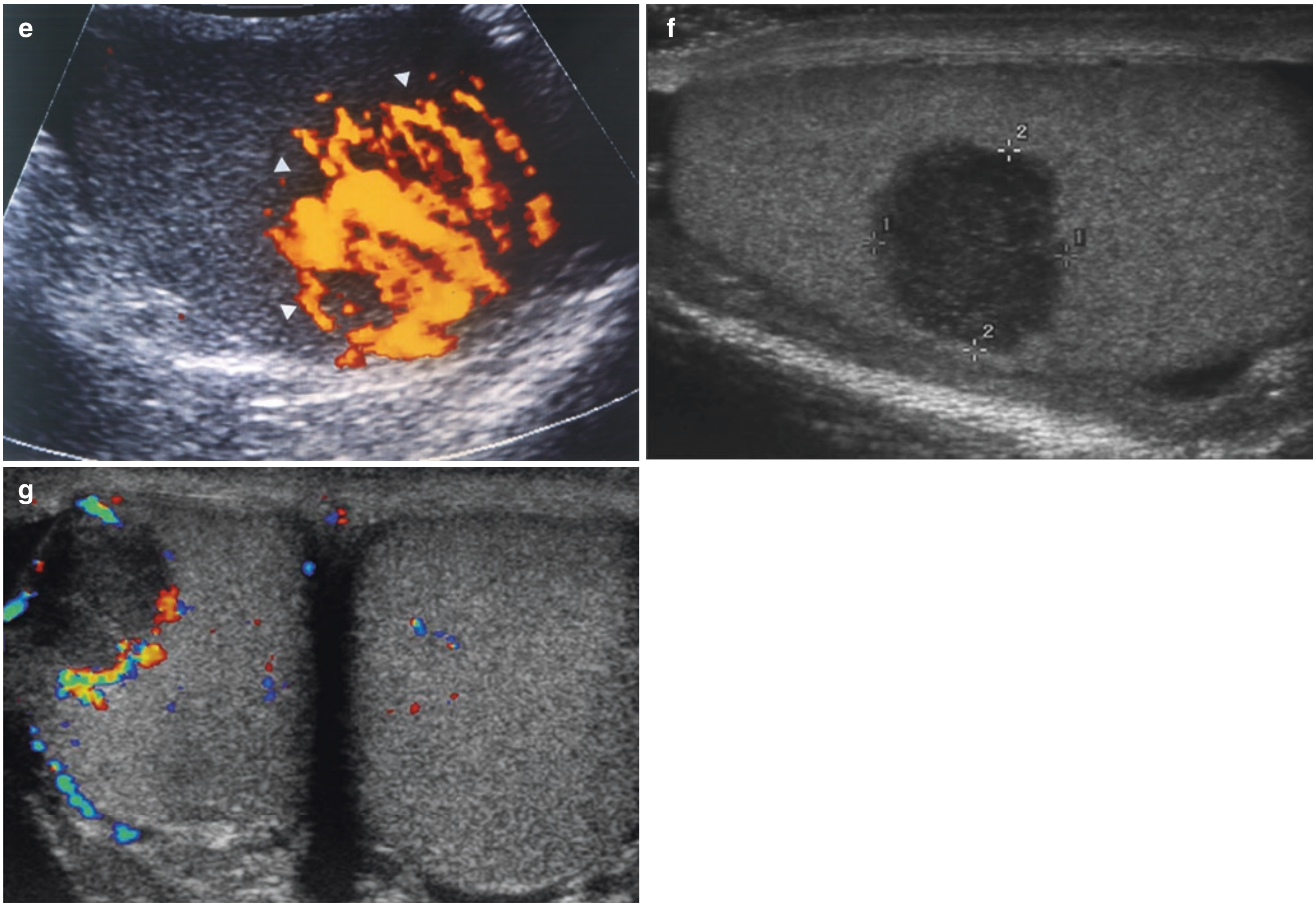


Fig. 3.40 Plasmacytoma. Colour and power Doppler flow image of the mass shows markedly increased vascularity (**a**, **b**). Gross orchiectomy specimen shows lobulated mass-like areas surrounded by haemorrhage (**c**). Longitudinal sonogram showing the plasmacytoma as a slightly heterogeneous, hypoechoic testicular mass (**d**). Power Doppler sonogram of the tumour in the longitudinal plane showing marked hypervascularity (**e**). Longitudinal and transverse high-resolution greyscale sonography of the right testis reveals a slightly lobulated, predominantly hypoechoic lesion. There is increased colour flow around the periphery of the lesion (*panels* ***a–c****, From: F.B. Walker, E.I. Bluth, A. Kenney et al. 'Plasmacytoma of the Testis' J Ultrasound Med 2005; 24:1721–1725; panels* ***d****,* ***e****, From: R.O. Bude 'Testicular Plasmacytoma: Appearance on Gray-scale and Power Doppler Sonography' Journal of Clinical Ultrasound (1999); 27(6): 345–346; panels* ***f****,* ***g****, From: K.M. Surti, P.W. Ralls. 'Sonographic Appearance of Plasmablastic Lymphoma of the Testes' J Ultrasound Med (2008); 27:965–967)*



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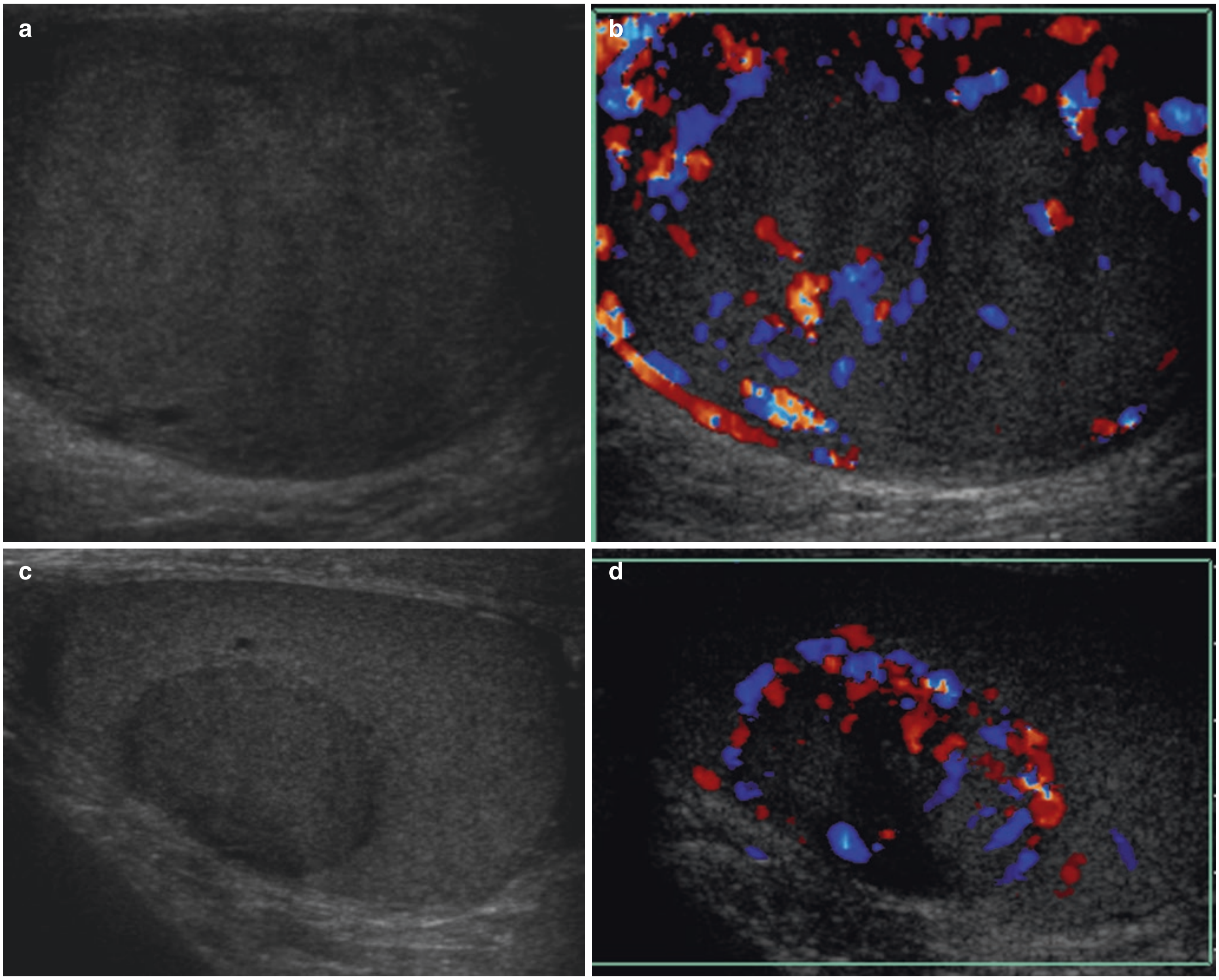


Fig. 3.41 Testicular carcinoid. **Bilateral testicular carcinoid**. Left scrotal sonography shows a diffuse infiltrative mass without calcification in the left testis (**a**). Doppler sonography shows increased vascularity of the mass (**b**). Right scrotal sonography shows a well-defined solid mass without calcification in the right testis (**c**). Doppler sonography shows the hypervascularity of the right testicular mass (**d**) (*From: S. Park, J. Kim, K.S. Cho 'Imaging Findings of a Primary Bilateral Testicular Carcinoid Tumor Associated With Carcinoid Syndrome' J Ultrasound Med 2006; 25:413–416)*

3.4.5 Metastases

Metastatic involvement of the testis by other cancers does occur. The most common primary sites include the prostate, lung, gastrointestinal tract (Fig. 3.42), skin (melanoma) (Fig. 3.43) and kidney, but others have also been reported (including pancreas, bladder, thyroid, neuroblastoma, schwannoma and retinoblastoma). There is nothing specific about the US appearance, but metastases are generally seen in the setting of widespread disease and are rarely the first reason for presentation.

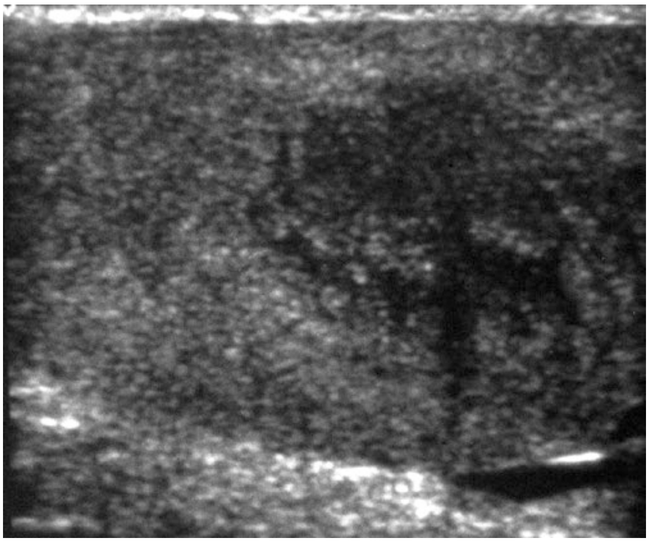


Fig. 3.42 Oesophageal metastases. Ultrasound of the left testis in an elderly male with known disseminated oesophageal carcinoma. The testis is enlarged and hypoechoic and contains a central hypoechoic mass. The appearances are consistent with metastatic infiltration. Figure is in longitudinal section *(From: S. Basu, D. C. Howlett, 'High-resolution ultrasound in the evaluation of the nonacute testis' Abdom Imaging (2001) 26:425–432)*

Fig. 3.43 Melanoma metastases. US scans reveal a hypoechoic inhomogeneous mass, with undefined margins, consistent with the metastatic infiltration of a melanoma *(Courtesy of: R.H. Oyen, MD)*

3.5 Non-palpable Testicular Lesions: Benignity vs. Malignancy

Occasionally a non-palpable intratesticular lesion is discovered by chance on scrotal ultrasound performed for a variety of other indications such as infertility, varicocele orchialgia, etc. The detection of small, non-palpable lesions is increasing and is a growing dilemma for the andrologist. In this chapter, we have stressed that diagnosis in most, if not all, cases of intratesticular lesions can only be made by histological confirmation. Nevertheless, we believe that the role of modern scrotal ultrasound is to attempt a provisional diagnosis of benignity vs. malignancy in order to orientate physicians towards active surveillance or, in dubious cases, a testis-sparing enucleation, as opposed to an inguinal orchiectomy with spermatic cord clamping. Various authors have recently proposed a surveillance approach to benign intratesticular lesions such as segmental infarction or focal Leydig cell hyperplasia.

Having stated all the limitations of US, the paragraph below provides a guide that may help clinicians to interpret scrotal US findings when adopting a conservative approach to intratesticular lesions. However, it should be emphasised that the surveillance strategy proposed for the management of these lesions is still very new. There are no prospective studies that support the use of this strategy as a definitive approach for all undetermined intrascrotal lesions. Furthermore, the uncertainty of some radiological findings should be carefully considered and communicated to patients confidently and clearly, to avoid anxiety or loss at follow-up.

The large majority of malignant intratesticular tumours are focal, solid and hypoechoic to normal testicular tissues (Fig. 3.44), independent of any associated cystic

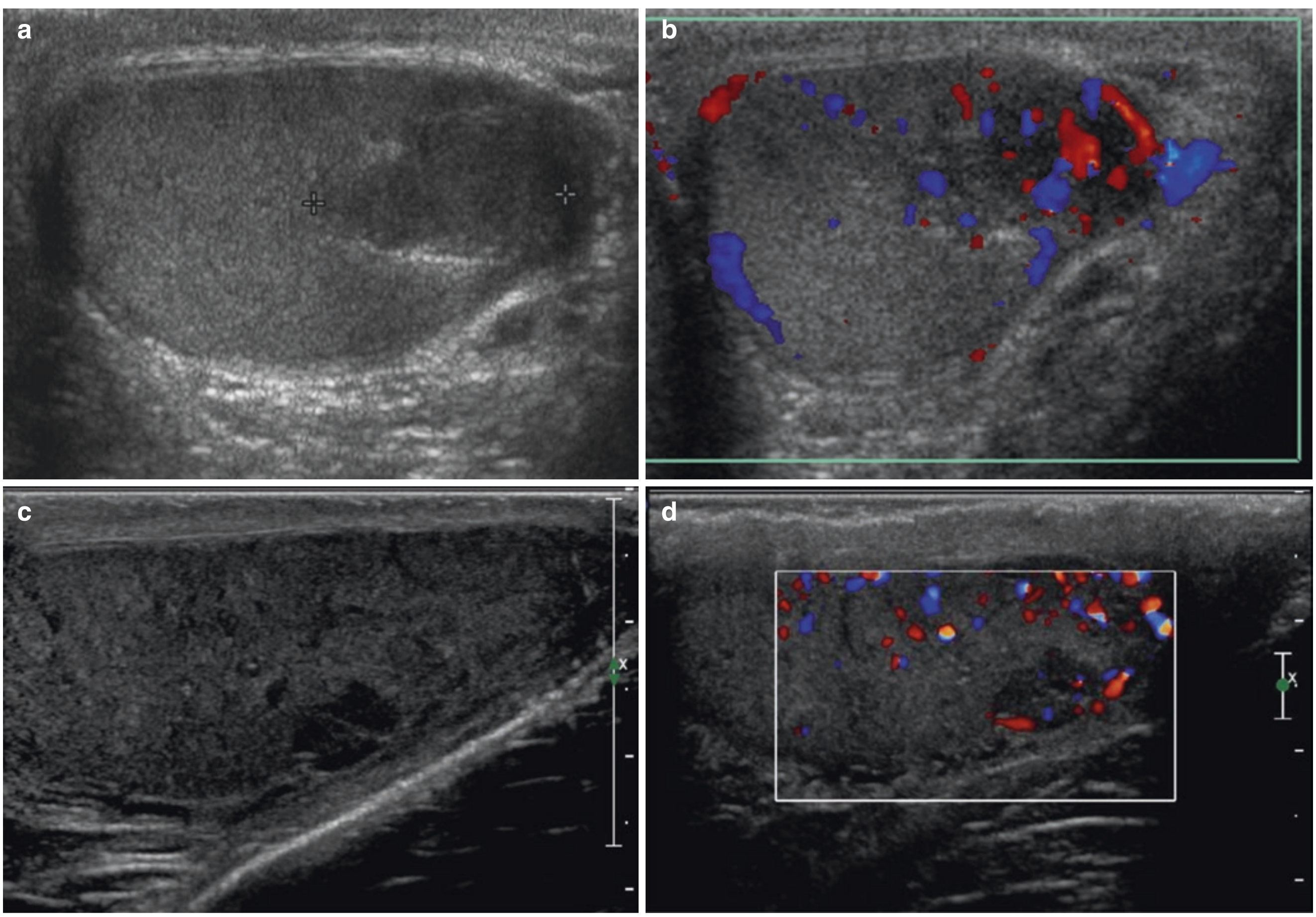


Fig. 3.44 Malignant intratesticular lesion. The majority of malignant intratesticular tumours are focal, solid and hypoechoic to normal testicular tissues. Note the ill-defined margin of the lesion (**a**–**d**)

degeneration. However, there are a number of exceptions, as tumours can be multifocal, calcified and necrotic or occur in a homogeneously hypoechoic testis:

1. A first helpful differentiating feature is the presence of any adjacent remaining normal testicular tissue: even with large malignant tumours, it is almost always possible to retrieve some residual testicular parenchyma (Fig. 3.45), whereas in orchitis and trauma, no normal testis is usually visible. Even in cases of focal orchitis or localised haematoma, the rest of the testis is usually oedematous in the acute stage. Obviously, there are exceptions, such as when the whole testis is replaced by tumour infiltration or in the presence of segmental testicular ischaemia (Fig. 3.46).
2. A second feature is superficial deformity or irregularity of the shape of the testis, considered a typical feature of malignancy. An exception is the presence of severe orchitis with intratesticular abscess or albuginea cysts, which on palpation can easily be confused with the edge of an intratesticular malignancy (Fig. 3.47).
3. A third point is whether testicular masses are accompanied by extratesticular features; epididymitis suggests a possible orchitis (Fig. 3.48) or trauma. Again, however, there are exceptions.
4. A fourth feature is extratesticular fluid collection (hydrocele or haematocele). Small hydroceles are common in testicular cancer, but large hydroceles frequently suggest a benign condition.
5. A fifth feature is the finding of calcifications: microlithiasis in association with a solid intratesticular lesion reinforces the suspicion of malignancy (Fig. 3.49); while an echogenic focus with or without posterior acoustic shadowing in a retroperitoneal lymphadenopathy strongly suggests a burnt-out germ cell tumour.
6. Finally, colour Doppler ultrasound has an increasing role in the characterisation of intratesticular lesions. Over 90% of primary testicular neoplasms of over 1.0 cm diameter show increased vascularity on colour Doppler ultrasound (Fig. 3.50). Flow studies may help to identify tumours that are relatively isoechoic with testicular parenchyma, although focal or diffuse inflammatory lesions cannot be distinguished from neoplasm on the basis of US alone (Fig. 3.51).

Although these features may help categorise the lesions on the basis of personal experience, there is a clear overlap between features suggesting benignity and malignancy, such that no clear discrimination can be made on a single scan. However, integration with clinical and biochemical data as well as a follow-up scan can provide a much more reliable categorisation than previously thought possible (Fig. 3.52).

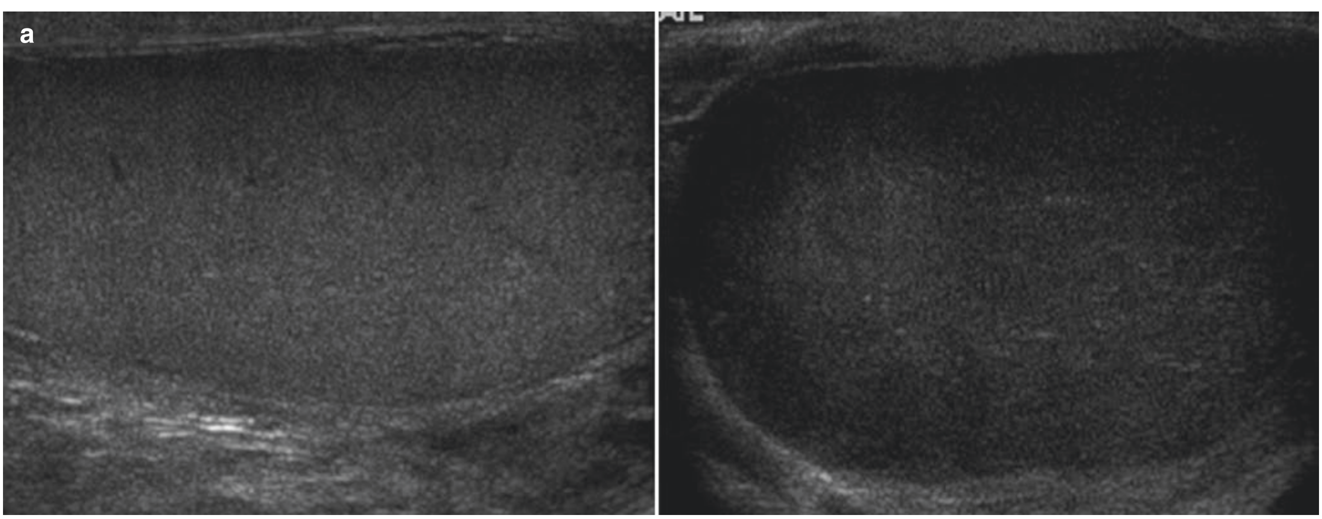
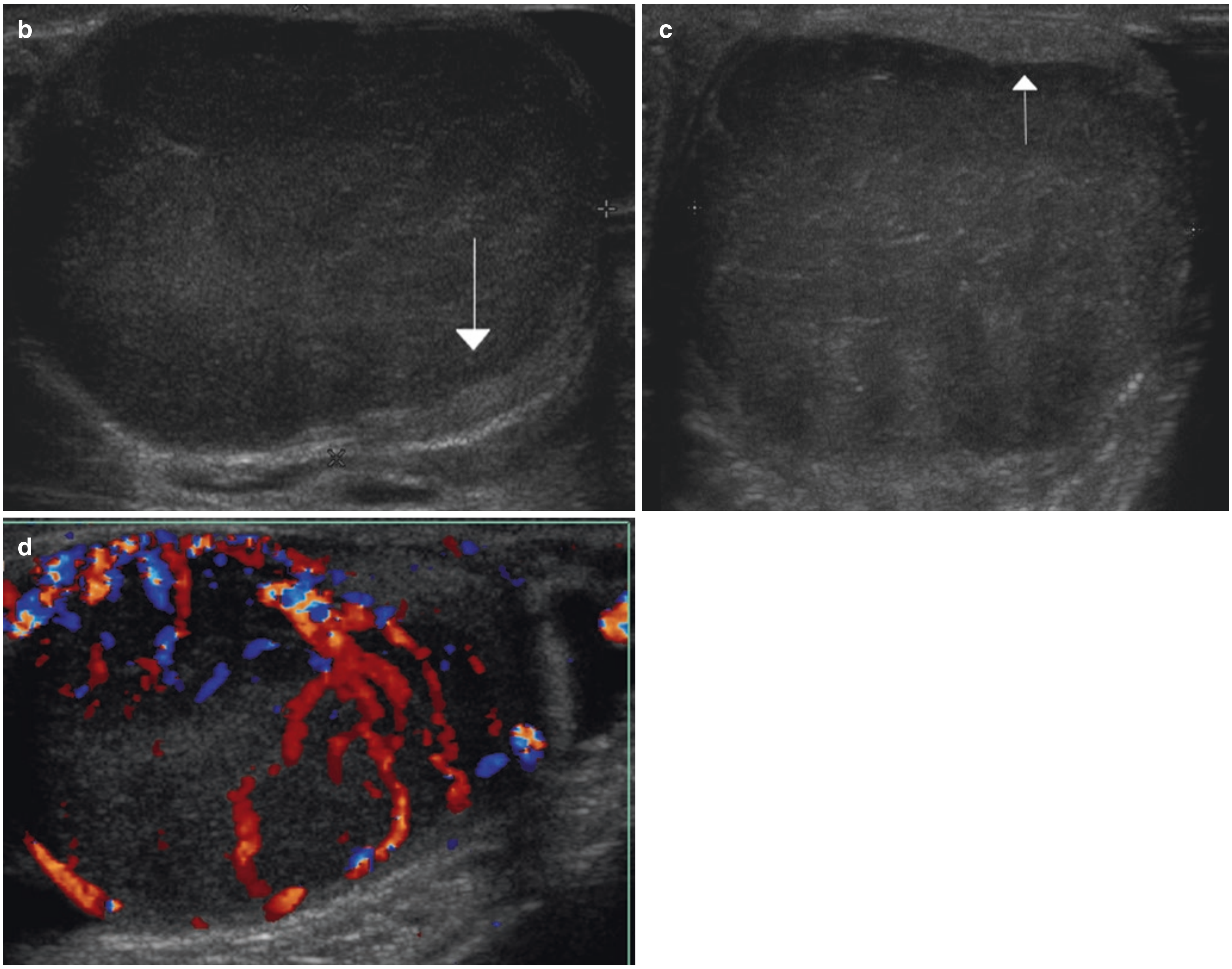


Fig. 3.45 Large seminoma. US scans of a large tumour found in the left testis of a man of 52 years old. The scans show a solid, large, hypoechoic mass. A rim of the normal testis is also seen in figures (**b**) and (**c**) (*arrows*). Colour Doppler scans (**d**) show intralesional vascularisation



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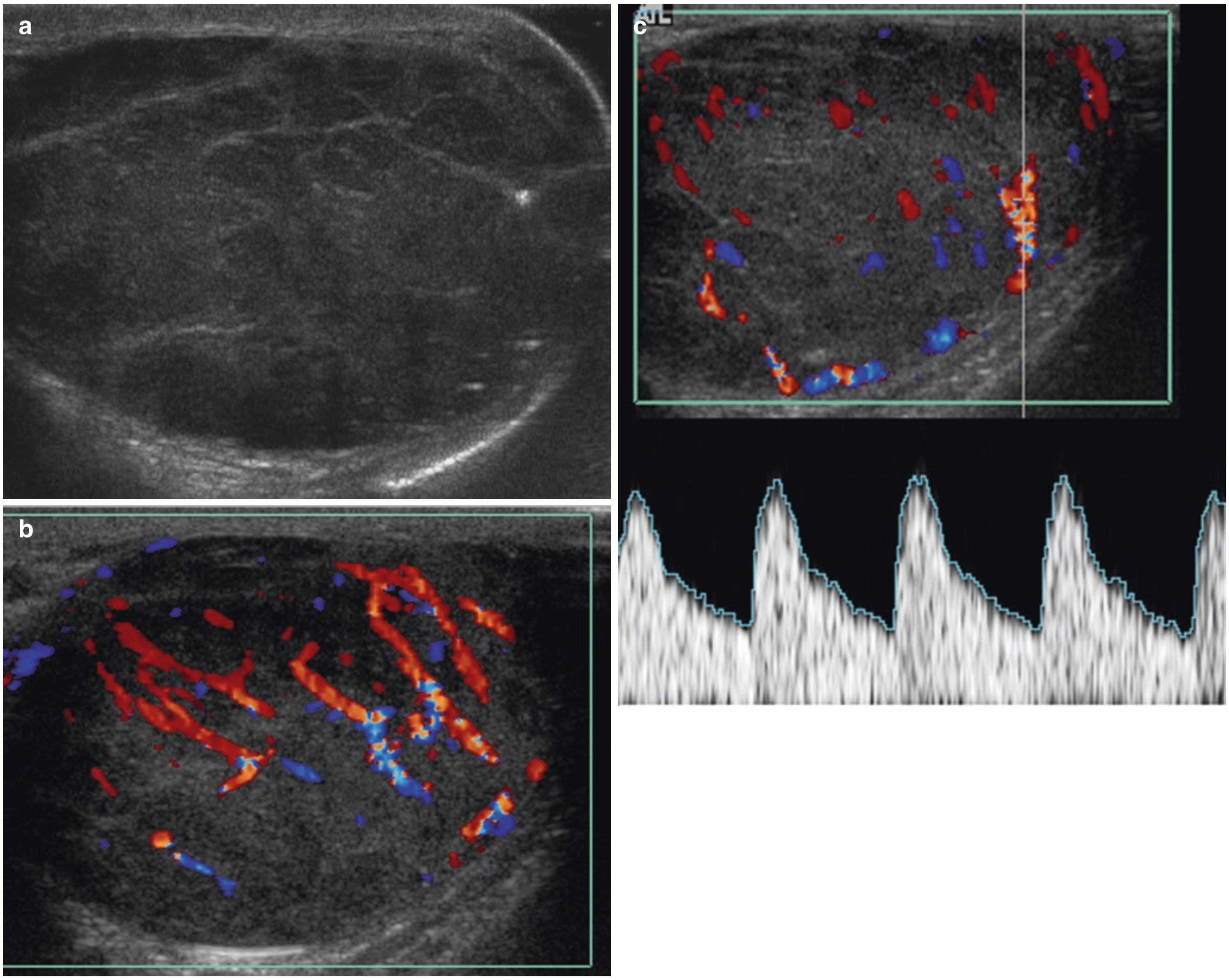


Fig. 3.46 Large seminoma. US scan shows the whole testis replaced by a tumoural mass (**a**). The vascularity seen in the lesion is not distinguishable from the one within testicular inflammation (**b**, **c**). Some malignancies infiltrating the entire testicular parenchyma may have a colour Doppler appearance that is identical to that of diffuse orchitis. The tumour was found to be seminoma at histology

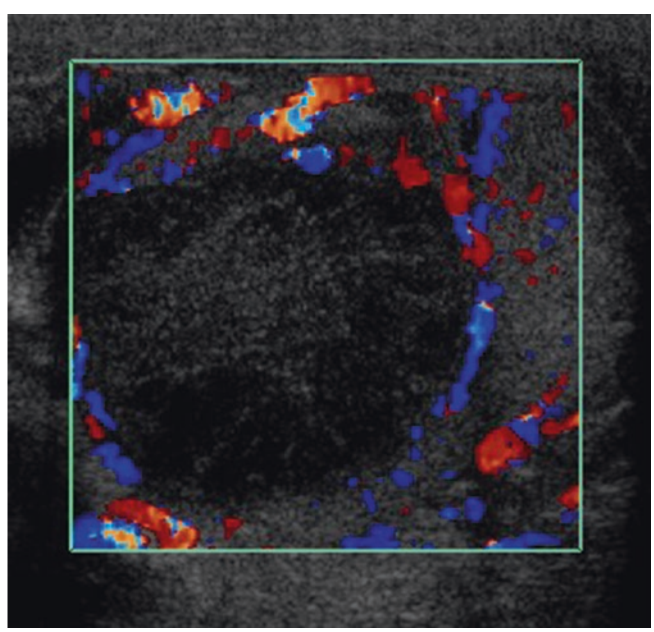


Fig. 3.47 Testicular abscess. Intratesticular abscess can be confused on palpation with an intratesticular malignancy. The distinctive feature is the nearly-absent vascularisation within the lesion *(From: A.F. Wittenberg, T. Tobias, M. Rzeszotarski et al., 'Sonography of the Acute Scrotum: The Four T's of Testicular Imaging' Curr Probl Diagn Radiol 2006;35:12–21. Courtesy of: A.F. Wittenberg, MD)*

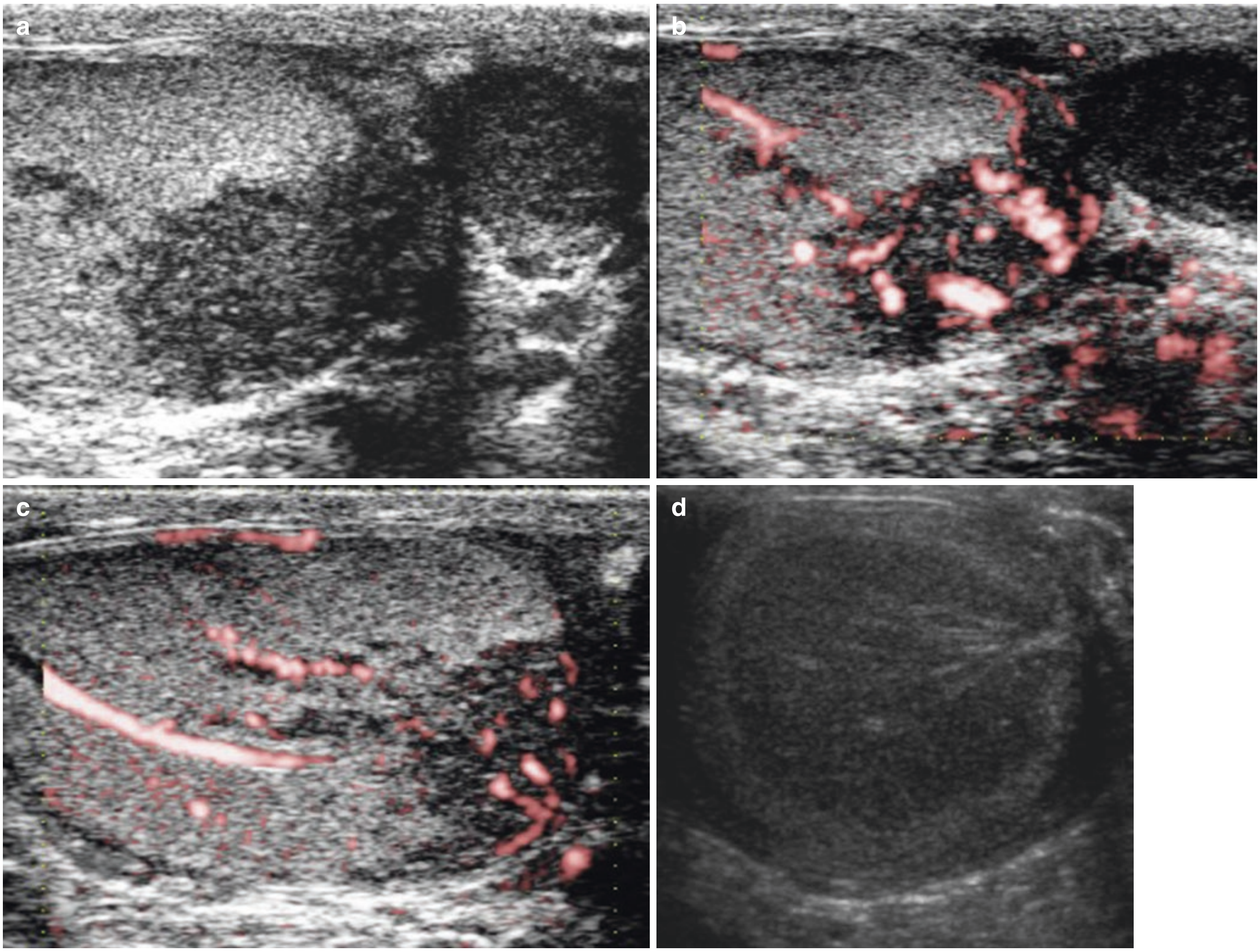


Fig. 3.48 Focal orchitis. Focal orchitis, mimicking a tumour. In focal orchitis the tissue around the lesion is generally oedematous *(panels **a–c**, Courtesy of R.H. Oyen MD; panel **d**, Courtesy of D.C. Howlett, MD)*

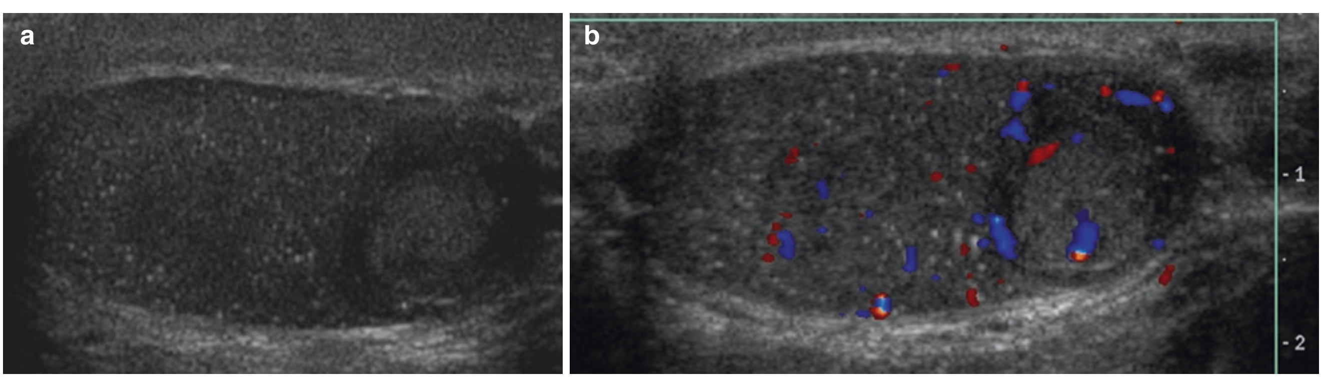
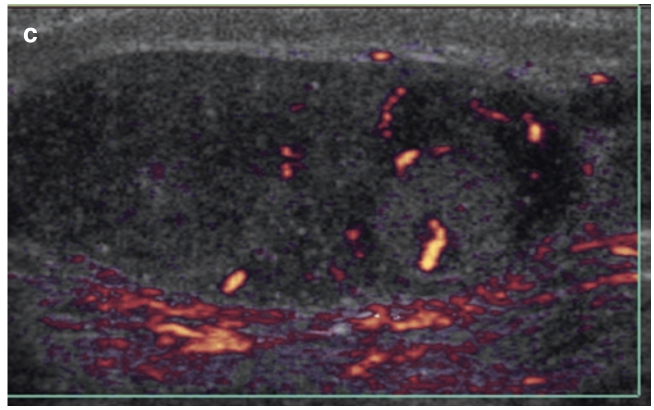


Fig. 3.49 The presence of microlithiasis in association with a solid intratesticular lesion reinforces the suspicion of malignancy

Fig. 3.49 (continued)



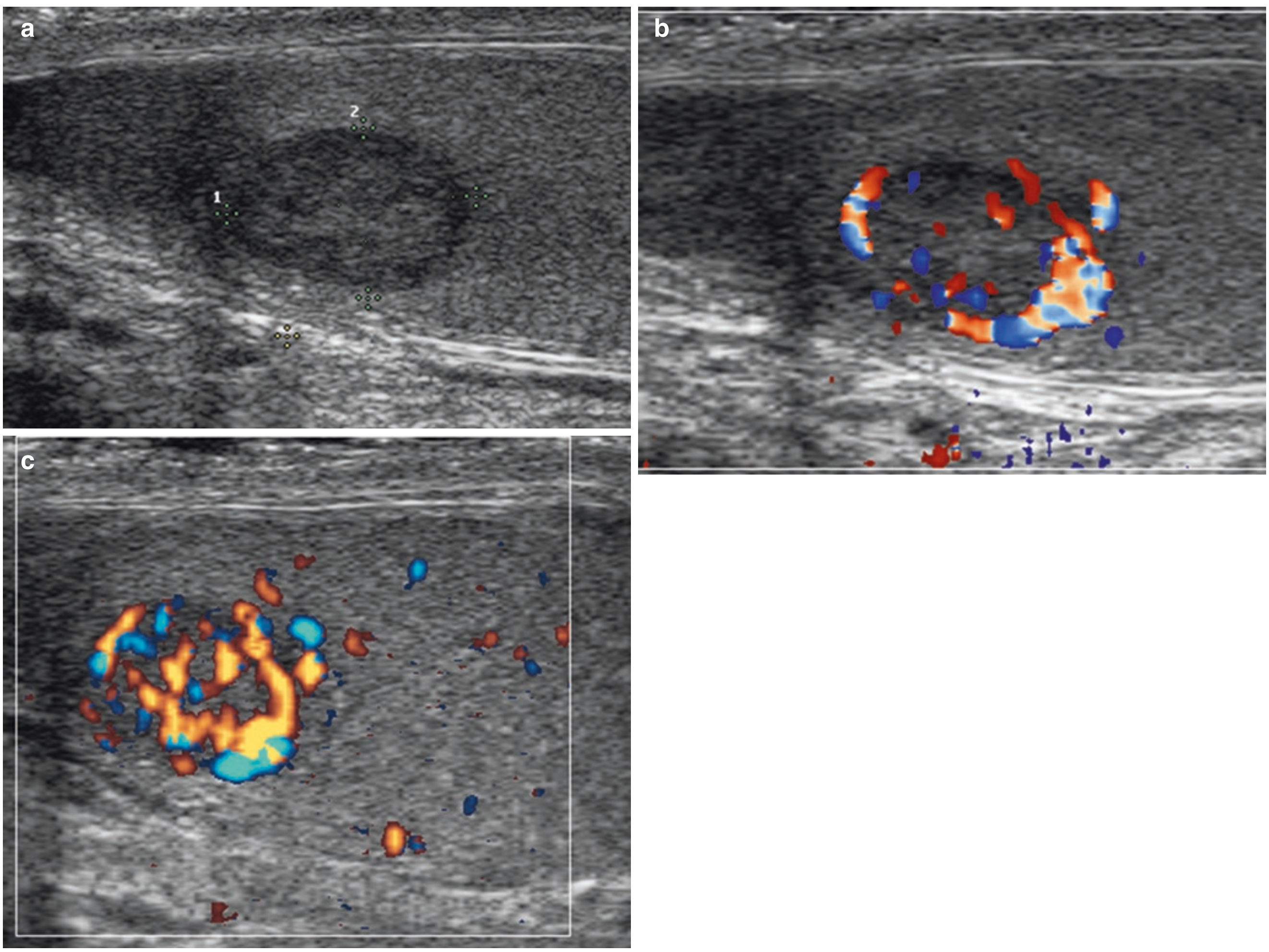


Fig. 3.50 Colour Doppler ultrasound showed increased vascularisation of a testicular lesion, found to be a seminoma at histology

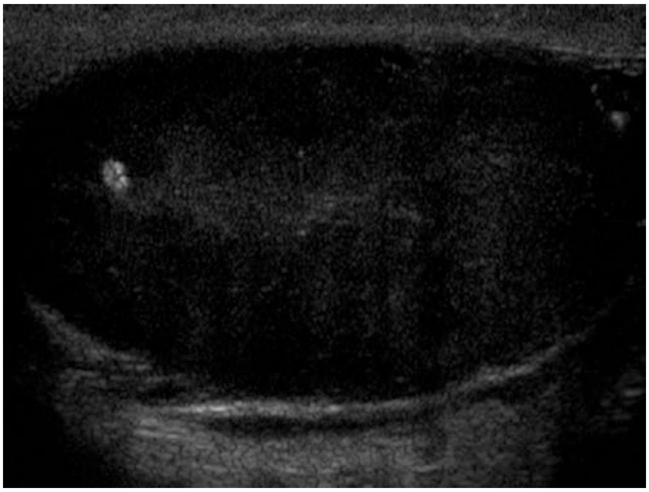


Fig. 3.51 Longitudinal scan of the testis shows a large, hypoechoic testis with calcification. At histology the lesion was a seminoma

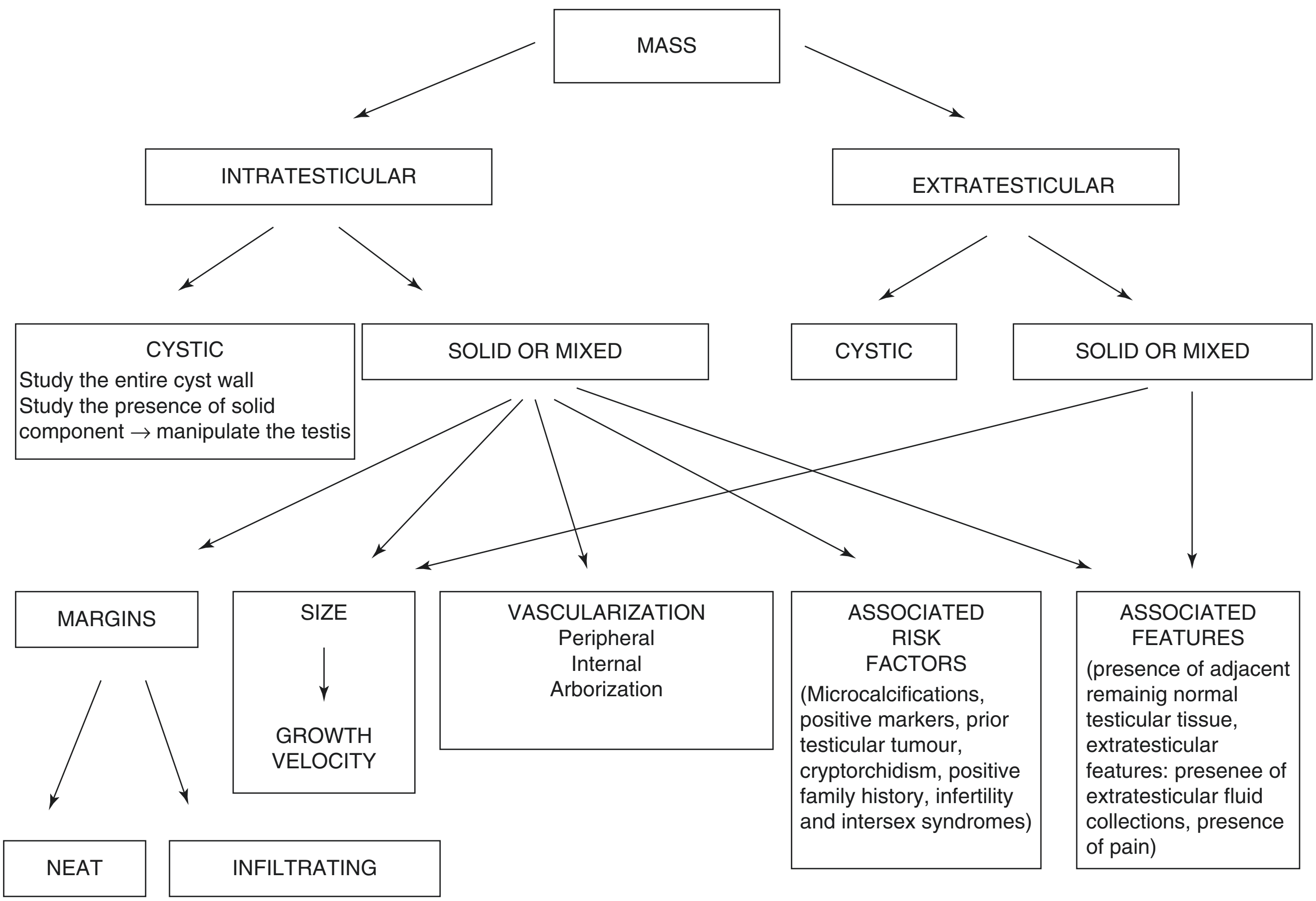


Fig. 3.52 Algorithm of evaluation of a scrotal mass

Key Messages

- Testicular cancer has known risk factors (familiarity, cryptorchidism, testicular dysgenesis, genetic and intersex disorders) that should be sought in every patient. Seminoma is the most common tumour type in cryptorchid testes.
- Although there are some typical US features, testicular tumour must be typed histologically.
- With few exceptions, cystic lesions are generally benign, while solid or mixed lesions are neoplastic. The presence of a solid component in a cystic lesion points to a possible mixed tumour or a teratoma.
- Positive tumour markers greatly increase the likelihood that a testicular lesion is cancerous, although negative tumour markers do not exclude this possibility.
- The use of CDU and power Doppler US is crucial in the evaluation of any intratesticular lesion: flow studies demonstrate increased vascularisation in most malignant tumours. However, predominantly cystic lesions can mislead by showing no intralesional flow.
- Testicular tumours are frequently associated with testicular microlithiasis. In seminomas, this association is described in approximately a third of cases.
- Seminomas are mostly homogeneous and more hypoechoic; they are rarely cystic, whereas non-seminomatous tumours are heterogeneous, even when they are small, and frequently show cystic or calcified parts with alternating hypo- and hyperechoic areas.
- Large seminomas can have necrotic areas and appear non-uniform or mixed. They can exhibit multilobulated borders or asynchronous multinodular lesions, but their margins are generally neat and clear.
- Embryonal cell carcinomas are aggressive tumours, generally hypoechoic on US but more heterogeneous than seminomas, due to cystic degeneration and calcifications. A third contains cystic areas; rapid growth with distortion or infiltration of the albuginea is common.
- Conservative treatment has been advocated for prepubertal teratomas, but is not an option for teratomas in post-pubertal testes. For teratomas, mature does not equal benign.
- Choriocarcinoma can present with very high hCG levels and gynaecomastia.
- Yolk sac tumour is the most common germ cell tumour in children under 2 years, presenting with elevated alpha-FP in over 90% of cases.
- ‘Burnt-out tumour’ describes a primary testicular tumour that regresses to a fibrotic (hyperechoic or calcific) scar with intratubular cancerous cells.
- Primary malignant testicular tumours metastasise to the para-aortic and iliac lymph nodes.
- US appearance and the histological components of germ cell tumours can change between the primary and secondary lesions or after treatment.
- Gonadal stromal tumours account for approximately 3–6% of all testicular neoplasms (more than 2/3 are Leydig cell tumours), and over 90% are benign.
- Leydig cell tumours can present with symptoms related to hormonal activity: gynaecomastia, precocious pseudopuberty, reduced libido and infertility. They are most frequently small and occasionally bilateral.
- Lymphoma is one of the most common neoplasms in men aged over 50. The tumour is frequently bilateral with synchronous or asynchronous involvement of the contralateral testis (8.5–18% of cases).

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4 Extratesticular Lesions

4.1 Introduction

Extratesticular diseases affect the epididymis, the spermatic cord and the scrotal layers. The most frequent findings are epididymal cysts and fluid collections – hydroceles and haematoceles – readily diagnosed with US. Scrotal hernias are also very common findings. However, most exams are performed for epididymitis, which is the most frequent cause of acute or chronic scrotal pain. Primary solid neoplasms are considered rare among paratesticular lesions, although various studies have estimated their prevalence as between 3 and 16% of all patients referred for scrotal ultrasonography [1, 2], indicating that paratesticular tumours are not as infrequent as generally thought (Table 4.1). These tumours are composed of a variety of epithelial, mesothelial and mesenchymal elements. Most neoplasms originate from the spermatic cord and epididymis and, much less commonly, from the testicular tunica. Most solid extratesticular tumours in adults are benign, while up to 40–50% of painless extratesticular masses in children may be malignant [3, 4] (see rhabdomyosarcoma). The most common benign extratesticular neoplasms are lipomas (generally of the spermatic cord) and adenomatoid tumours (mainly of the epididymis) [2]. Malignant tumours include rhabdomyosarcoma of the spermatic cord and mesothelioma of the tunica vaginalis.

Table 4.1 Extratesticular tumours

Benign	Malignant
Adenomatoid tumour	Rhabdomyosarcoma
Lipoma	Fibrosarcoma
Fibroma	Liposarcoma
Haemangioma	Histiocytoma
Leiomyoma	Lymphoma
Neurofibroma	Metastases
Cholesterol granuloma	
Adrenal rest	
Papillary cystadenoma	

4.2 Epididymal Lesions

4.2.1 Epididymal Cysts and Spermatoceles

In our series of over 5000 consecutive patients, **epididymal cysts** of various sizes were found in 26% of cases. Their aetiology remains unclear. A possible traumatic or postinfective origin has been suggested, although in most patients there is no known contributing factor. The cysts are most often located in the head or body of the epididymis of asymptomatic patients (Fig. 4.1) and are detected by chance during exams performed for other reasons. Less frequently, in the case of larger cysts or those in the tail, which are less common, there is a specific request for US evaluation. Epididymal cysts can be found in patients of any age but are most frequent in post-pubertal boys and young adults.

Cysts are normally palpable as painless firm or hard lumps, adherent to the epididymis, but clearly separable from the testicle. There is no evidence that they can directly cause pain. Any associated scrotal pain or discomfort is more likely to be secondary to concomitant epididymitis or compression of adjacent structures. Epididymal cysts can be single or multiple (Panel 4.1e and f), ranging from tiny to very large. However, a mass of over 4 cm in diameter is more likely to be a spermatocele. The distinction between epididymal cysts and spermatoceles is based on pathology, as they cannot generally be differentiated by US (Fig. 4.2). Epididymal cysts are round or oval anechoic lesions with posterior acoustic enhancement. They can be simple or multiseptated, with thin internal septations. Their walls do not contain any solid parts and are generally undetectable on US. Rarely, cysts can become inflamed. In these cases, pain may be present and thickening and hyperaemia of the cystic wall is seen.

Epididymal cysts contain a clear citreous fluid, whereas spermatoceles contain a white-yellow fluid consisting of nonviable sperm, lymphocytes, cellular debris, fat and proteinaceous sediment [5]. This confirms that the two conditions have different aetiologies.

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DOI 10.1007/978-3-319-51826-8_4

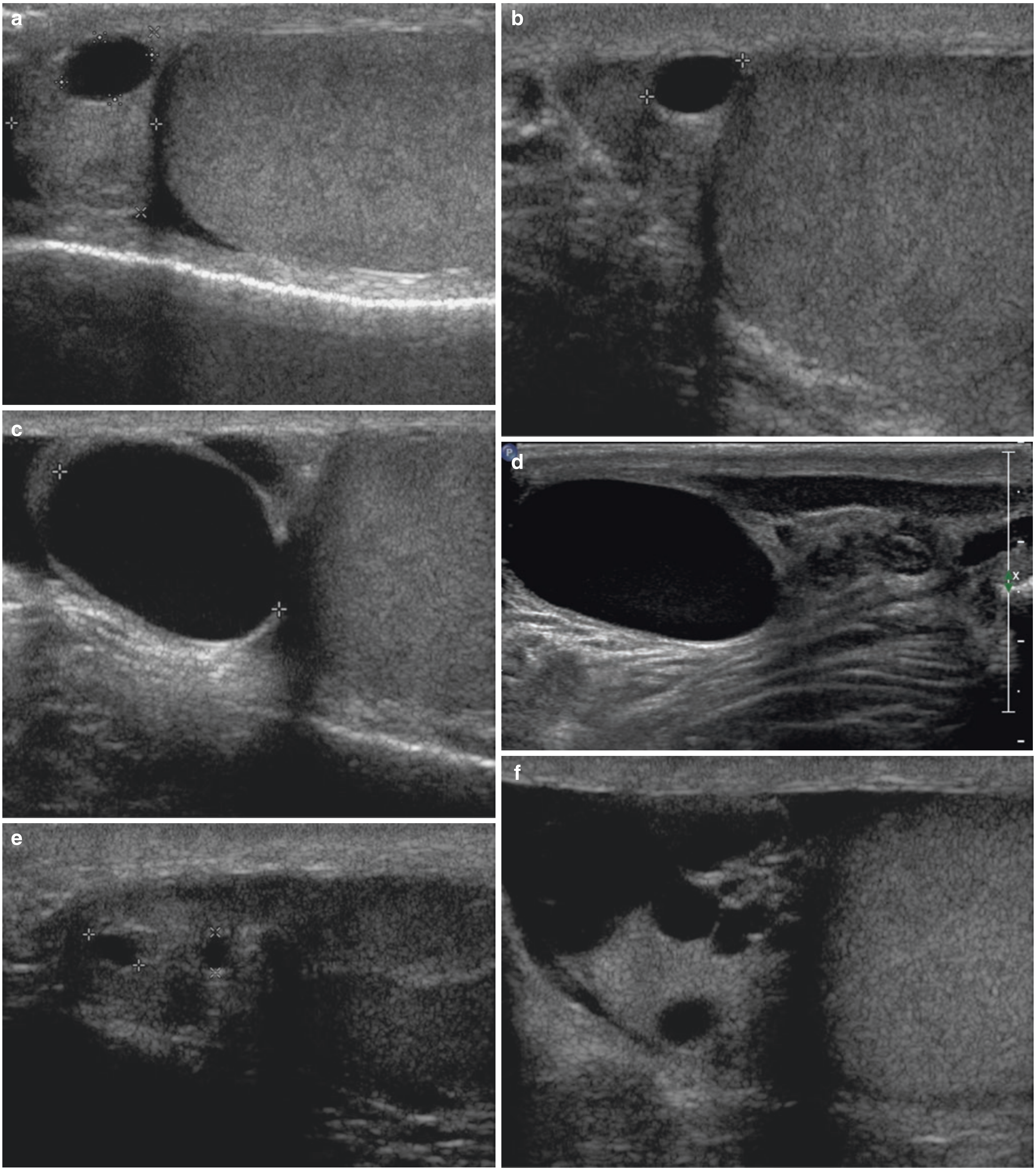

Fig. 4.1 Epididymal cysts. Longitudinal scans of the testis show two small (**a**, **b**) and two large (**c**, **d**) anechoic cysts in the head of the epididymis. Epididymal cysts can be single or multiple (**e**, **f**), and they can be also found in the body (**g**, **h**, *arrows*) and in the tail (**i**) of the epididymis

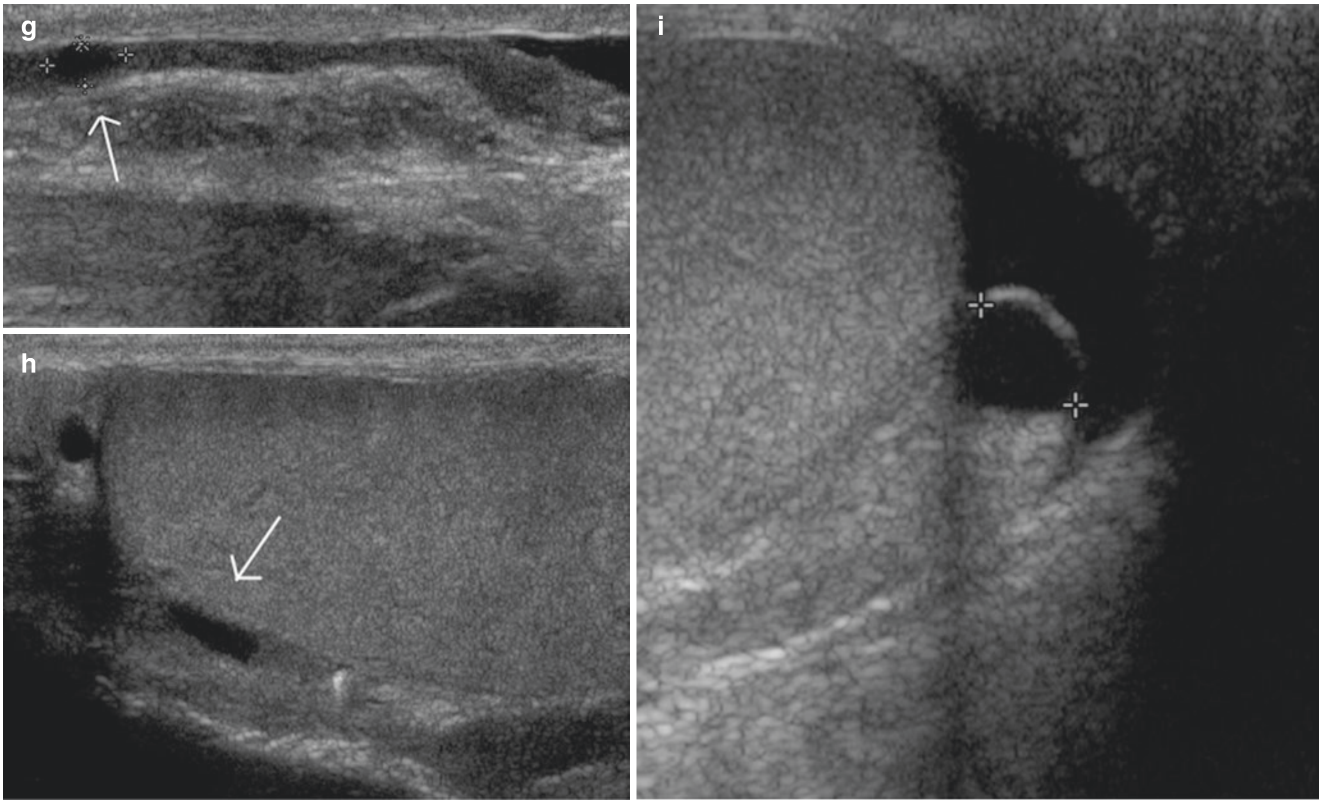

Fig. 4.1 (continued)

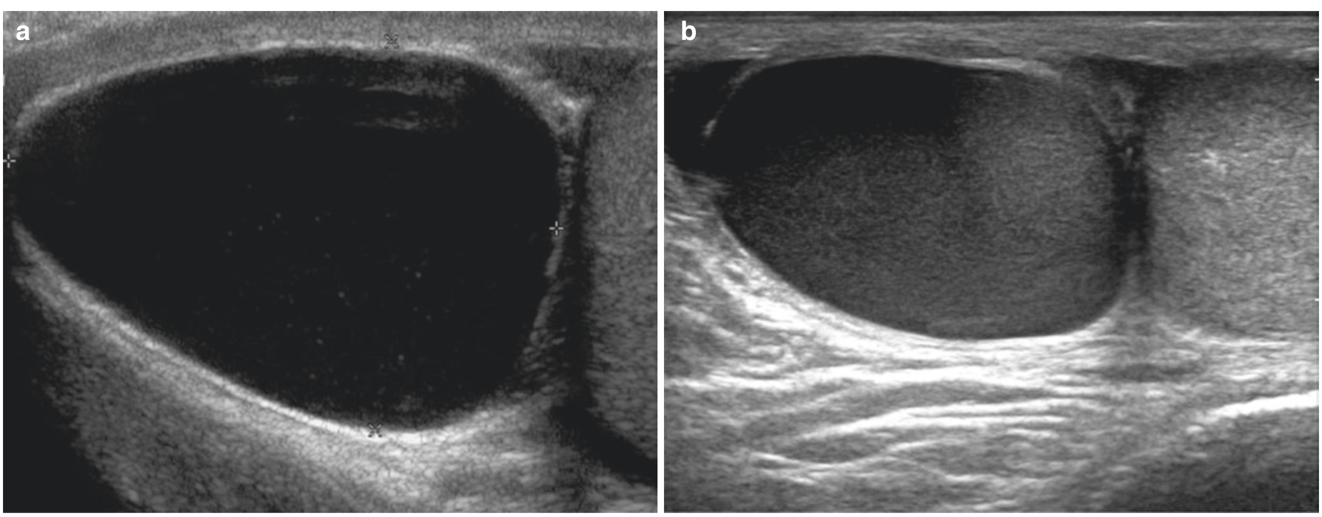

Fig. 4.2 Epididymal cysts. Large epididymal cysts. Longitudinal scan of the head of the epididymis shows a large, well-defined anechoic cyst with good through transmission and posterior acoustic enhancement. A spermatocele can have the same appearance (**a**). Occasionally, debris and particulates can be found in spermatocele and cysts (**b**, **c**) (*panels* **b**, **c**, *Courtesy of R.H. Oyen, MD*)

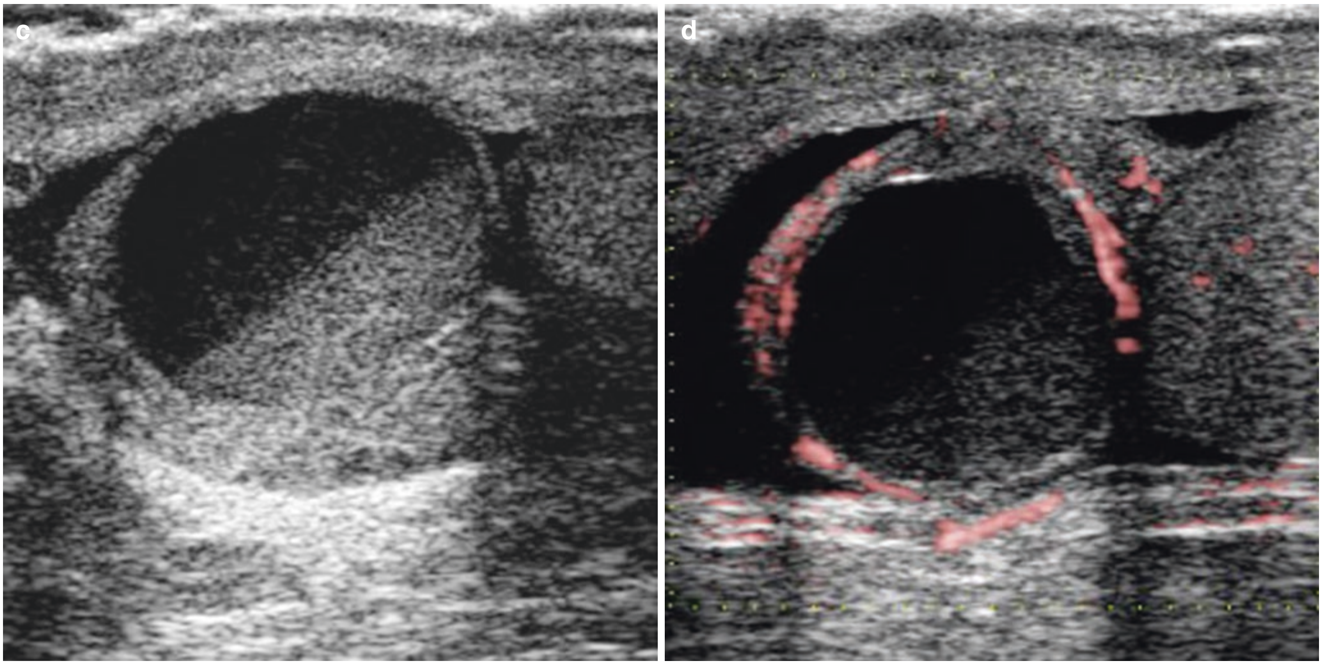

Fig. 4.2 (continued)

Spermatoceles originate from the mechanical obstruction of efferent ductules of the rete testis. They are more frequently found in middle-aged men and are often seen after vasectomy or obstruction secondary to inguinal herniorrhaphy, epididymitis or prostatic diseases. Smaller spermatoceles are also found in association with tubular ectasia of the rete testes and intratesticular cysts (Fig. 4.3) [6]. On palpation, spermatoceles can be identified as a freely movable, transilluminating soft mass (more taut than hydrocele, but not as firm as epididymal cysts) that is separate from and above the testicle. They have a varied US appearance and may be seen as simple or complex cysts. Ultrasound normally shows well-defined hypoechoic lesions, generally 1–2 cm, with posterior acoustic enhancement. Depending on the aetiology, complicated, multiple or multiseptated cysts (Fig. 4.4), with possible internal low-level echoes attenuating the posterior enhancement, are not infrequent. Occasionally, spermatoceles have also been reported to appear as a solid hyperechoic mass with internal calcifications [5] that can mimic paratesticular neoplasms.

Spermatoceles can be very large and in some cases may be difficult to differentiate from hydrocele. On examination, they are firmer than most medium-volume hydroceles, while on US, the detection of the thin wall enveloping the spermatocele is conclusive. Moreover, in large hydroceles, there is normally some fluid in front of the testis, in contrast with spermatocele or cysts. Spermatoceles cause no symptoms, even when large. Torsion and haemorrhage can rarely occur. Aspiration is no longer indicated, as diagnosis is readily made by US and the procedure may introduce infection [7].

A long-standing obstruction of the epididymal tubules can be associated with single or multiple retention cysts but also with a diffuse dilation of the efferent ductules, giving the condition known as **tubular ectasia of the epididymis**. The causes underlying the development of spermatocele and tubular ectasia appear similar to those responsible for intratesticular ectasia of the rete testis [8]. Clinically it is felt as a bulky epididymis or sometimes as a focal mass. Ultrasound reveals a variable degree of enlargement of the epididymal body, which is diffusely hypoechoic with multiple interfaces

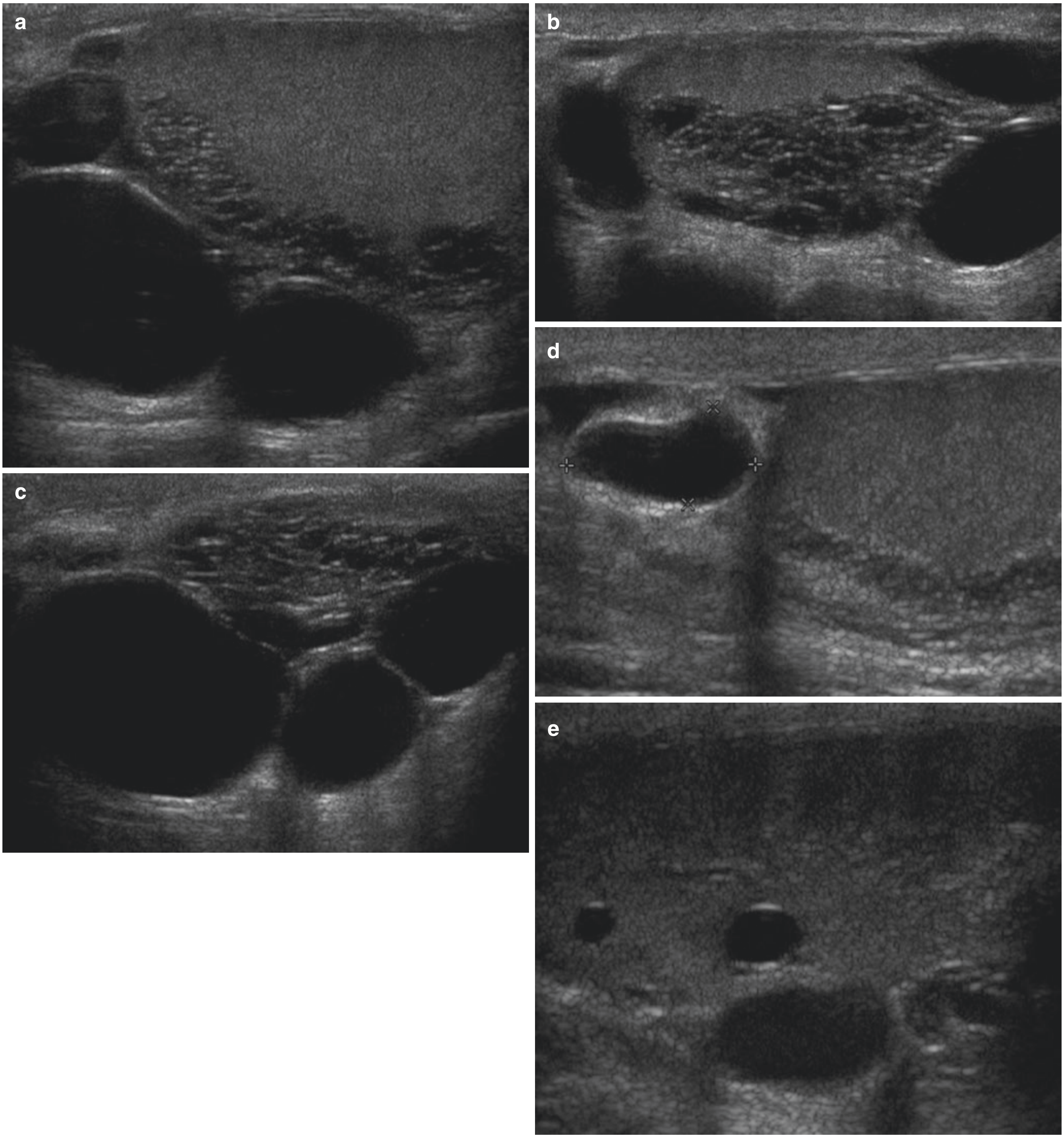

Fig. 4.3 Spermatocele and tubular ectasia of rete testis. Spermatocele are frequently found in association with tubular ectasia of the rete testis (**a**–**d**) and intratesticular cysts (**e**)

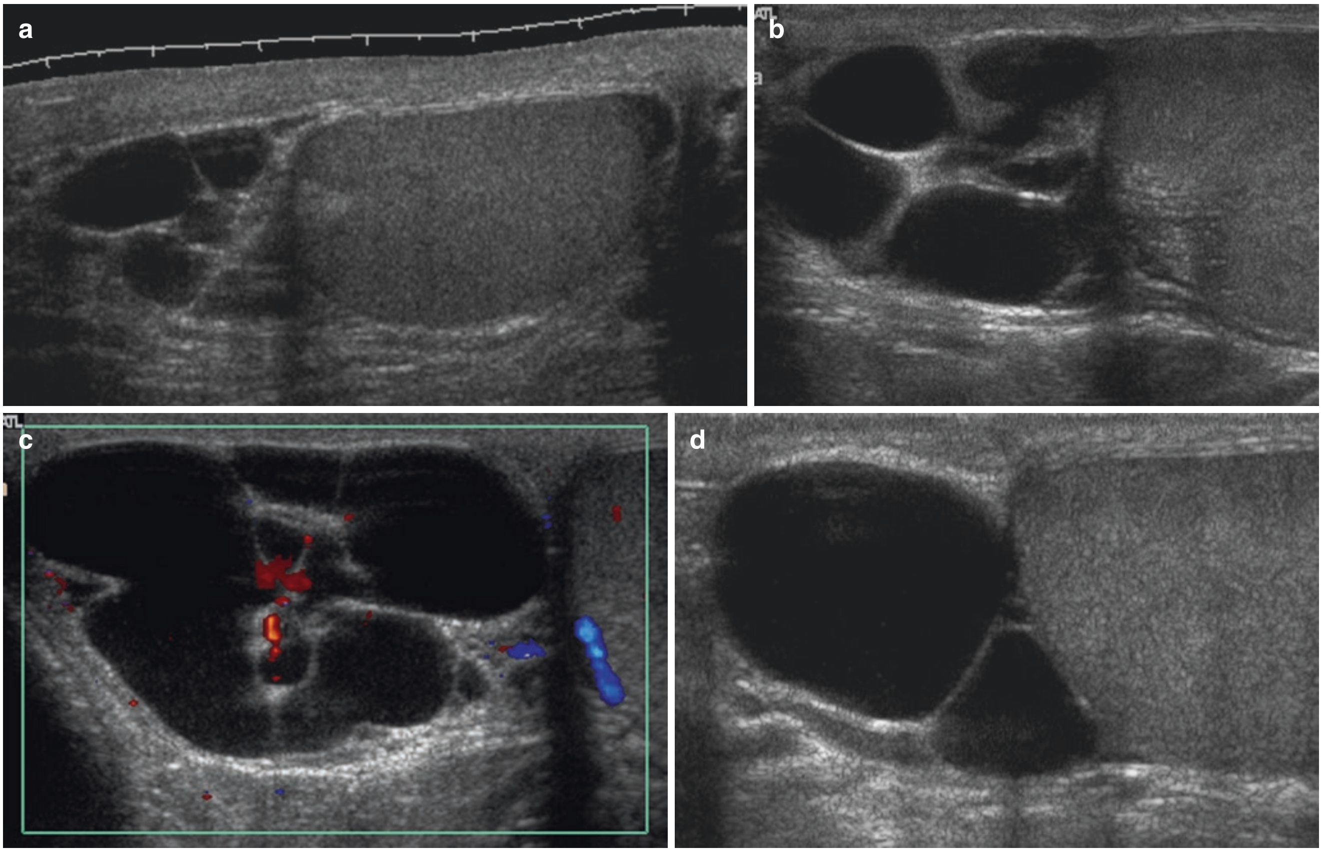

Fig. 4.4 Spermatocele. Longitudinal greyscale sonograms of the right epididymal head show the presence of multiseptated cysts; note that spermatoceles (**a**–**c**) are sonographically indistinguishable from epididymal cysts (**d**)

(Fig. 4.5) [9]. Anechoic and serpiginous tubular structures can only rarely be seen within the epididymis, while they are more frequent in the adjacent mediastinum (rete testis).

Tubular ectasia of the epididymis can be associated with spermatoceles (Panel 4.5i). The epididymal findings may be unilateral or bilateral. In addition to its typical localisation in the body of the epididymis, the tail and the head may also be involved. Colour Doppler examination is important in distinguishing acute epididymitis from tubular ectasia, which could develop as a consequence of untreated infections. On colour Doppler sonography, tubular ectasia is typically hypovascular, thus excluding inflammation. Rete testis dilatation is the most helpful associated feature, enabling diagnosis of tubular ectasia in the absence of known mechanical obstruction.

Rarely, a monolateral tubular ectasia can develop in the absence of any testicular involvement. In these cases, measurement of carnitine or other epididymal markers in the seminal fluid may be necessary to establish the diagnosis.

Finally, a possible complication of long-standing obstruction is '**vasitis nodosa**', a pathological finding characterised by foreign-body giant cell reaction and **sperm granulomas** in the interstitium. The elevated intratubular pressure causes sperm to migrate into the walls of the vas deferens, activating the epithelial cells, which proliferate and start a foreign-body reaction [9] (see paragraphs below).

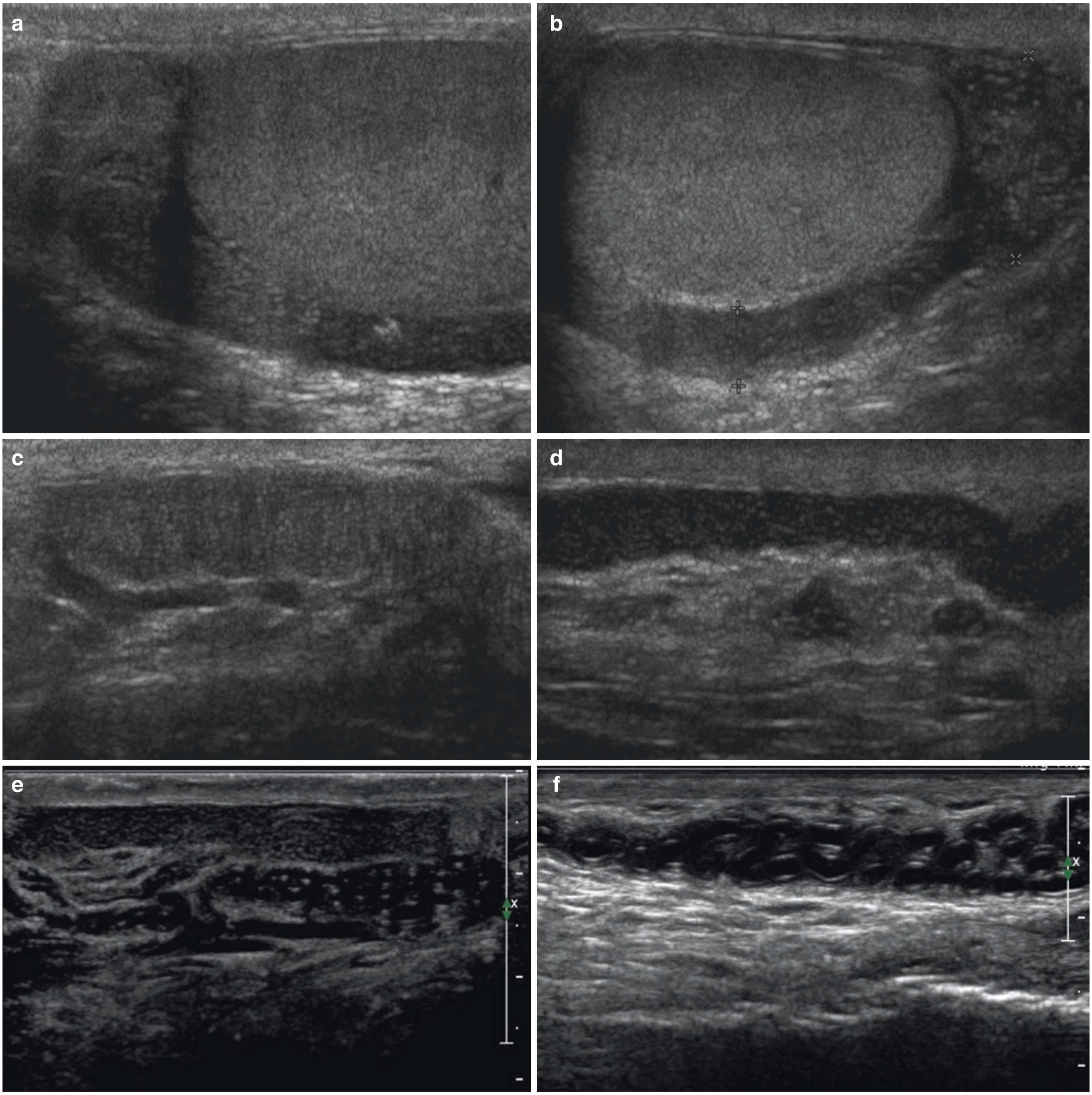

Fig. 4.5 Tubular ectasia of the epididymis. **Tubular ectasia**. Longitudinal ultrasound of the testis shows variable degree of enlargement of the epididymis, which is diffusely hypoechoic with multiple interfaces (**a**–**f**). Colour Doppler examination is important in distinguishing acute epididymitis (**g**) from tubular ectasia, the latter being hypovascular (**h**, **i**). **Tubular ectasia and retention cysts**. A long-standing obstruction of the epididymal tubules can be associated with single or multiple retention cysts (**j**, **k**). **Tubular ectasia and spermatocele**. Tubular ectasia of the epididymis can develop into spermatocele (**l**)

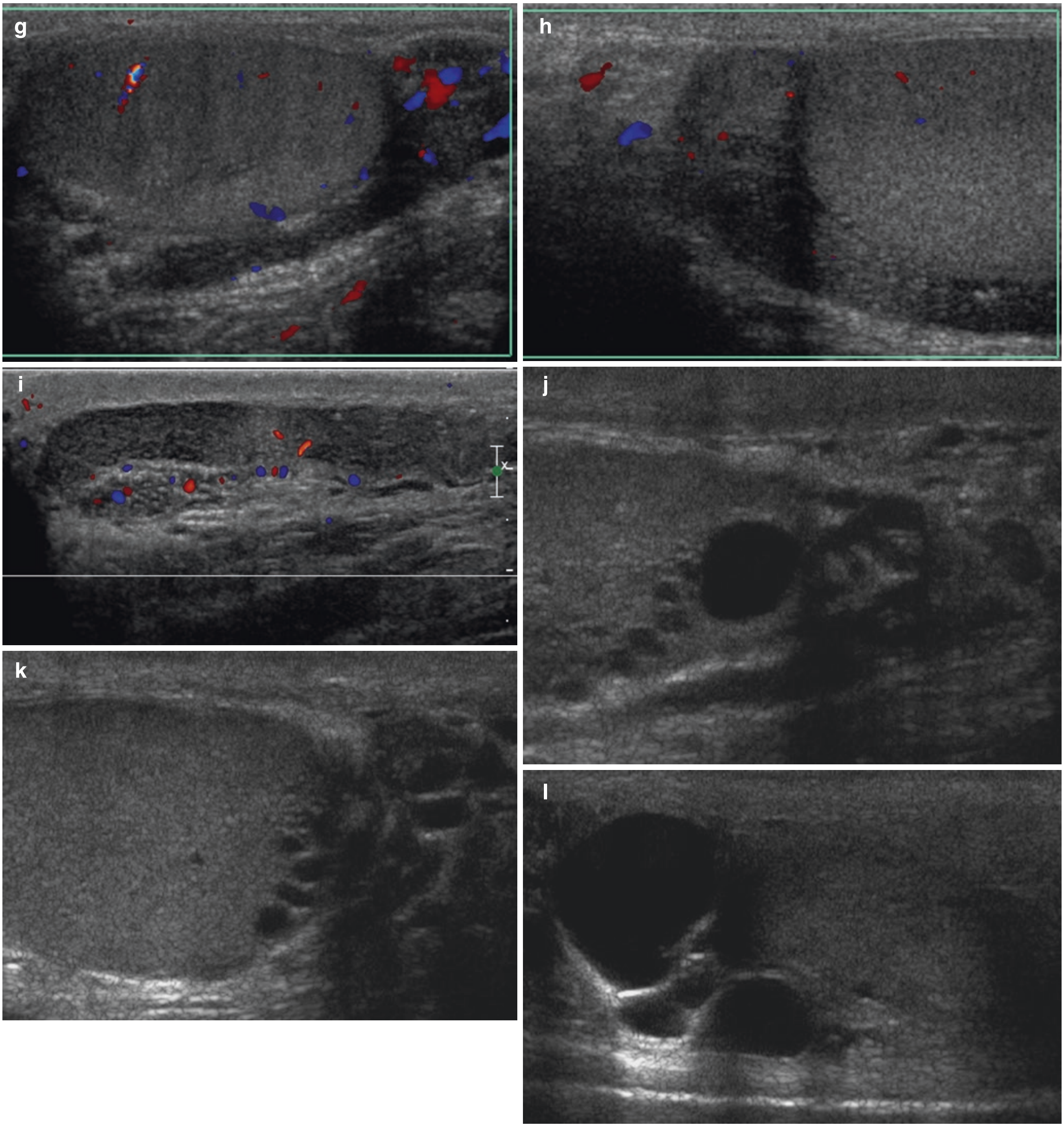

Fig. 4.5 (continued)

4.2.2 Inflammation and Infections of the Epididymis

Epididymitis is the most common cause of acute or chronic pain and is therefore the most frequent reason for consultation. Acute scrotum is often caused by what is generally defined as epididymo-orchitis. However, in the large majority of cases, only the epididymis is involved, and although hyperaemia can be seen in the adjacent testicular parenchyma, the infection does not involve the testes. A notable exception is mumps orchitis.

Epididymitis is classified as acute or chronic on the basis of the infective agent and duration [10]. Acute epididymitis is defined as inflammation, pain and swelling of the epididymis lasting less than 6 weeks. Chronic epididymitis is a condition characterised by long-standing scrotal pain with or without epididymal swelling, usually associated with irreversible structural changes of the epididymis.

The role of US in acute epididymitis is to determine the extent of the inflammatory process and its response to treatment but also to differentiate acute epididymitis from torsion of the spermatic cord. In chronic epididymitis, its role is the differential diagnosis from epididymal tumours or, in the case of granulomatous orchitis, from testicular tumours.

4.2.2.1 Acute Epididymitis and Epididymal Abscess

Acute epididymitis is a common complication of lower urinary tract infections (urethritis, prostatitis, cystitis) that reaches the epididymis retrogradely, via the vas deferens. Spread of the infection via the blood is much less frequent, generally occurring in patients with scrotal trauma. Iatrogenic infections associated with urinary catheters or minor surgical procedures (treatment of hydrocele or scrotal cytoaspiration) account for a significant number of cases. In older men, the most frequent pathogens are *enterococci* (*Escherichia coli*), *Pseudomonas* and *Aerobacter*, while *Chlamydia trachomatis* and *Neisseria gonorrhoeae* are more common in younger adults. A chemical or sterile epididymitis has also been described and is caused by retrograde urine reflux or an ectopic ureter opening into the vas deferens. Mild repeated trauma, typically associated with bicycle or motorbike riding but also coitus interruptus, prolonged ejaculatory latency and some sexual habits can be associated with epididymal discomfort and are a predisposing condition for epididymitis in young adults.

The clinical presentation is with acute, painful, tender hemiscrotum, fever and dysuria [11]. The onset can be insidious and not immediately perceived by the patient. In the acute stage, it may be difficult to differentiate from torsion of the spermatic cord on purely clinical grounds. Epididymitis is more likely in patients over 30, whereas torsion is the prime diagnostic hypothesis in prepubertal boys, in whom epididymitis is rare. Relief of pain on elevation of the scrotum over the symphysis (Prehn's sign) is suggestive of epididymitis, as this manoeuvre exacerbates the pain of spermatic cord torsion. However, epididymitis and torsion are both frequent conditions in males between 15 and 30, and in 15% of patients, early torsion also presents with localised swelling of the epididymis, compromising the clinical differentiation between the two. Laboratory investigations demonstrating leukocytosis, bacteriuria, leukocyturia or associated urethritis can help diagnosis.

The most severe complications of epididymitis are the spread of the infection to the testis (**epididymo-orchitis**) [11] – observed in up to 15% of cases – and the complete or partial obstruction of the efferent ductules, which can lead to infertility. Epididymitis may evolve into **scrotal abscess**. Associated inflammation of the spermatic cord is common, and reactive **hydroceles** often develop. Advanced or untreated cases of epididymo-orchitis may result in **pyocele** [11].

Most cases of epididymitis resolve spontaneously or with adequate medical treatment. However, clinical and US signs of the previous infection can be seen for years. In patients presenting with an enlarged epididymis, scrotal pain on examination or marked heterogeneity of the epididymis, a previous infection should be investigated. These patients often present a significant elevation of the number of white blood cells in the spermiogram, which persists despite no signs of residual infection. There is growing awareness that this persistent sterile **leukospermia**, causing an increased production of ROS (reactive oxygen species), is involved in a consistent number of cases of male idiopathic hypofertility.

The US features of epididymitis (Fig. 4.6) consist of a localised enlargement of the head (45%) and tail (15%) or diffuse involvement of the entire epididymis (40%), which appears heterogeneous, often hypoechoic with scattered hyperechoic foci and associated with mild to severe hyperaemia. During the acute stage, the epididymis can be markedly swollen. The enlargement is paralleled by a reduction in the echotexture, which assumes a coarse distribution (Fig. 4.7). The alternating areas of increased echogenicity are thought to be small epididymal haemorrhages. A reactive hydrocele with skin thickening is frequently seen [12].

The formation of an **abscess** is characterised by the development of a circumscribed hypoechoic lesion within the enlarged epididymis. In the presence of anaerobic infections, gangrene with associated gas formation produces a heterogeneous pattern with hyperechoic foci and artefacts (see Fournier's gangrene). If the testis becomes involved (acute **epididymo-orchitis**), a focal hypoechoic area of orchitis can occur adjacent to the portion of the inflamed enlarged epididymis (Fig. 4.8), with a strongly increased parenchymal flow [13]. Colour Doppler sonography is important in differentiating epididymitis from torsion, with vascularisation

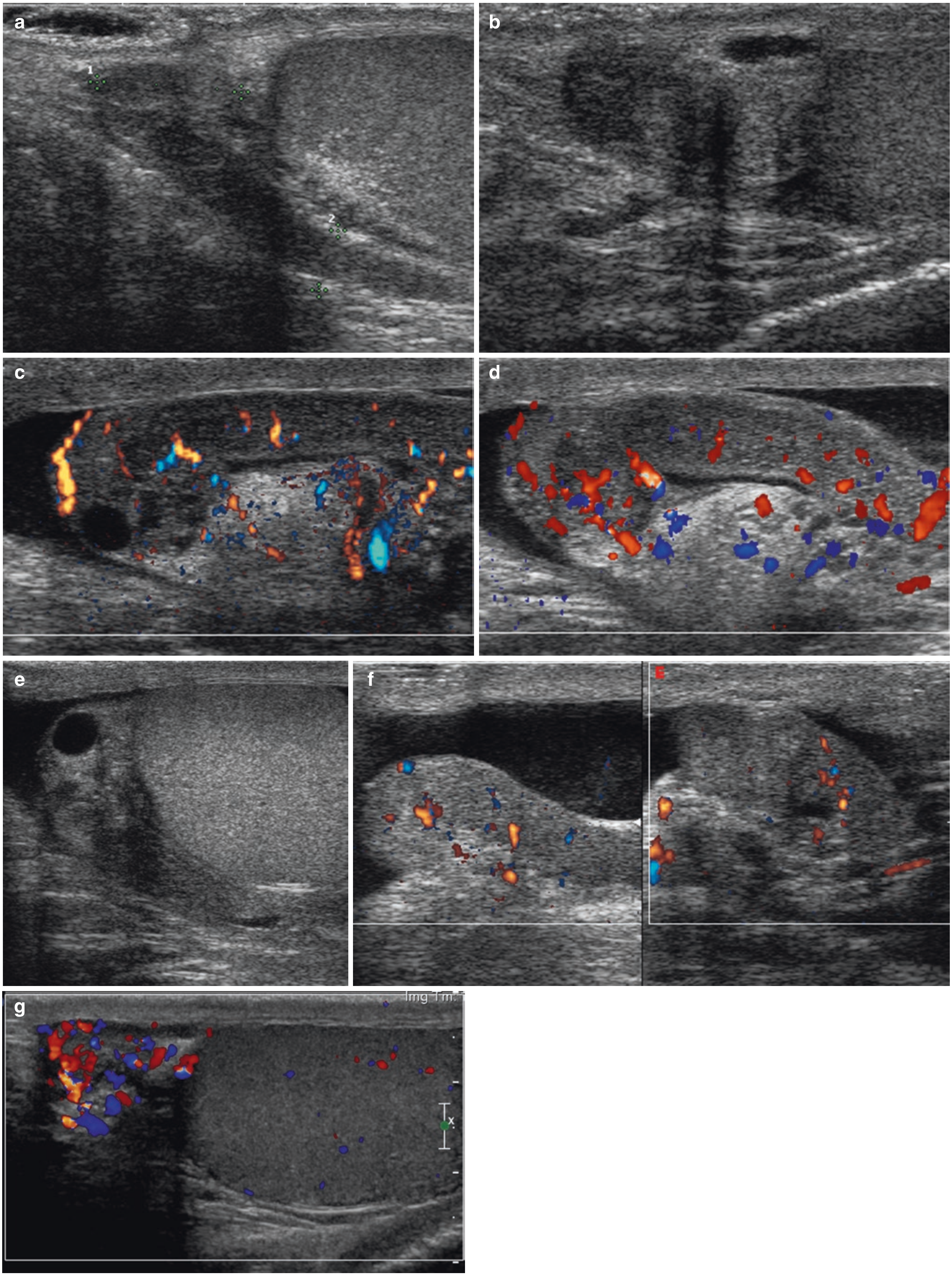

Fig. 4.6 Epididymitis. Epididymitis consists of a localised enlargement of the head and tail or diffuse involvement of the entire epididymis, which appears heterogeneous, often hypoechoic with scattered hyperechoic foci and associated with mild to severe hyperaemia

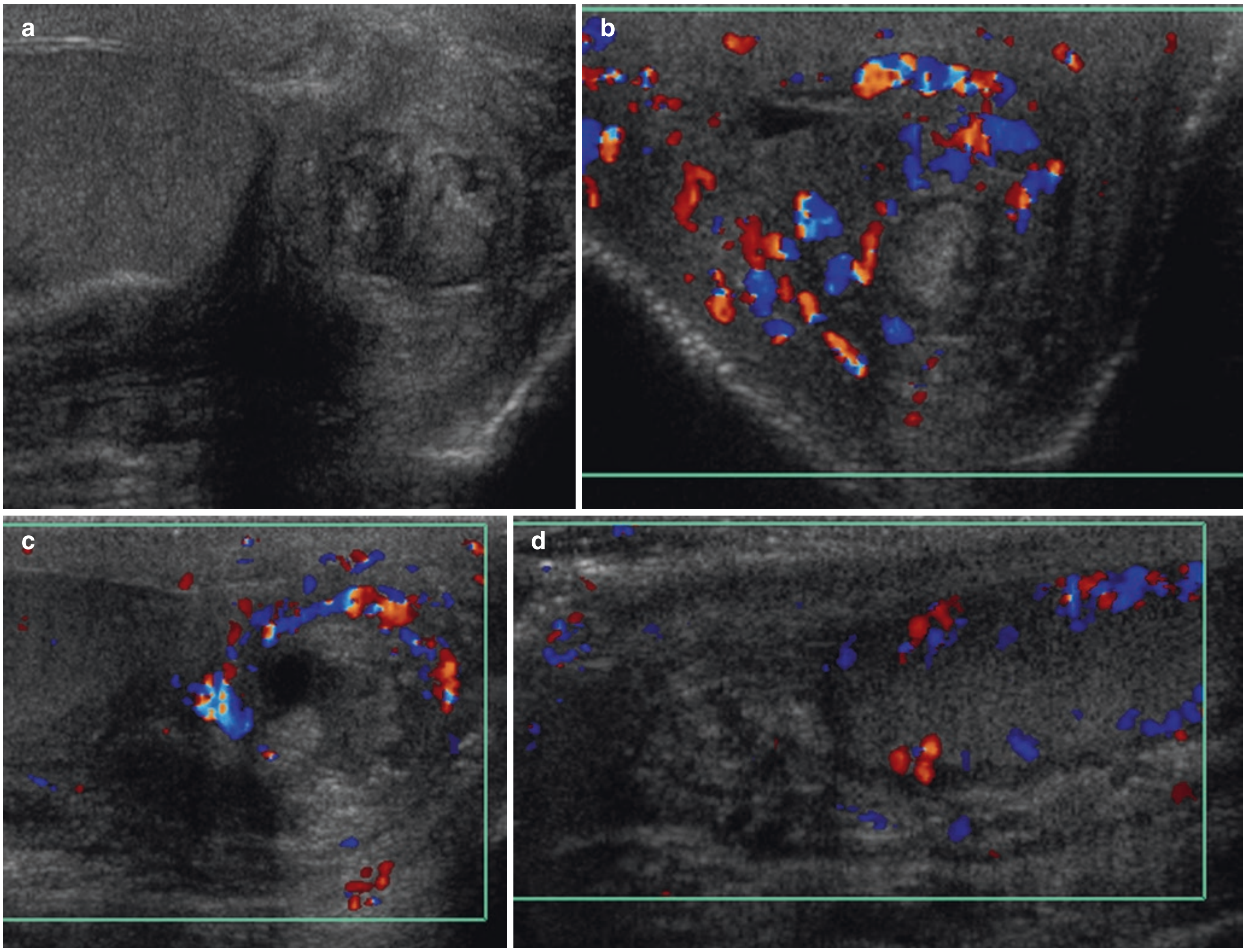

Fig. 4.7 Epididymitis. Ultrasonographic finding disclosed an enlarged, markedly heterogeneous lesion localised in the tail of epididymis, hypervascularised, probably a granuloma secondary to chronic epididymitis

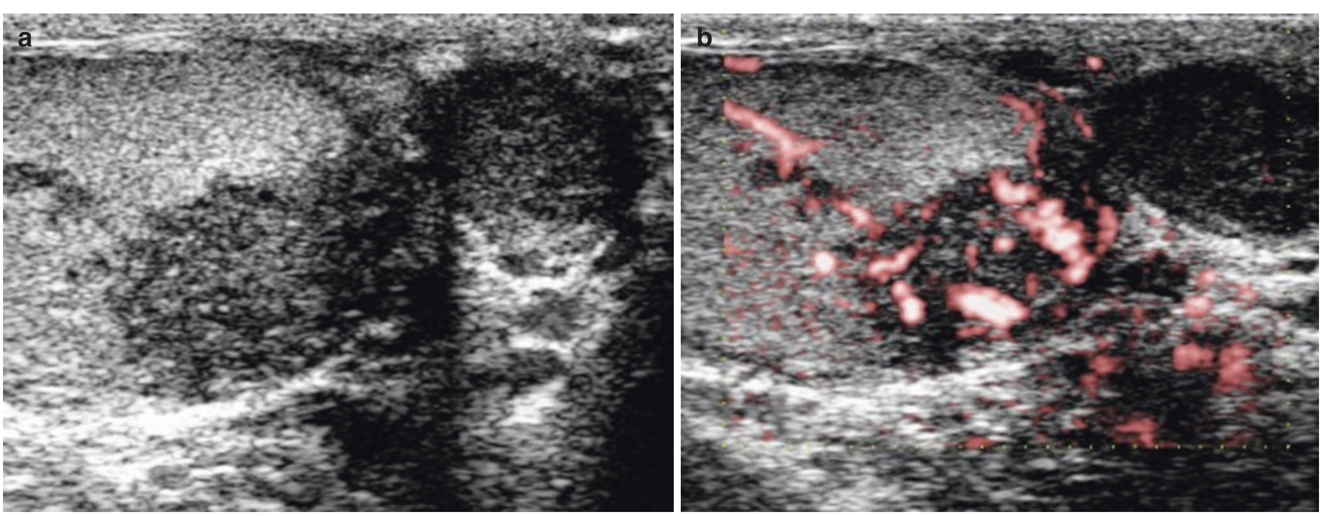

Fig. 4.8 Focal orchitis in epididymitis. If the testis becomes involved, a focal hypoechoic area of orchitis can occur adjacent to the portion of the inflamed enlarged epididymis *(Courtesy of R.H. Oyen, MD)*

invariably increased in the former (Fig. 4.9) and reduced in the latter. Abscesses can be easily recognised by their hypovascular centre with a tissue rim of increased vascularisation. These abnormalities can persist for weeks after the infection has been resolved.

4.2.2.2 Chronic Epididymitis and Fibrous Pseudotumour

Chronic epididymitis results from incomplete treatment of acute epididymo-orchitis or irreversible structural alterations secondary to chronic inflammatory changes and granulomatous reaction. The pathogens typically associated with chronic epididymitis are those causing tuberculosis, brucellosis, syphilis and fungal infections [12]. They reach the epididymis by retrograde extension from the prostate and seminal vesicles, but lymphatic and haematogenous spread are also possible [14]. In contrast with acute epididymitis, the pathogens causing the infection are less frequently detected by first-line analysis (microbiological evaluation of sperm and prostatic fluid), and additional investigation may be required.

The typical US features of chronic epididymitis are those associated with infections from *Mycobacterium tuberculosis*. These include a more frequent involvement of the tail [12],

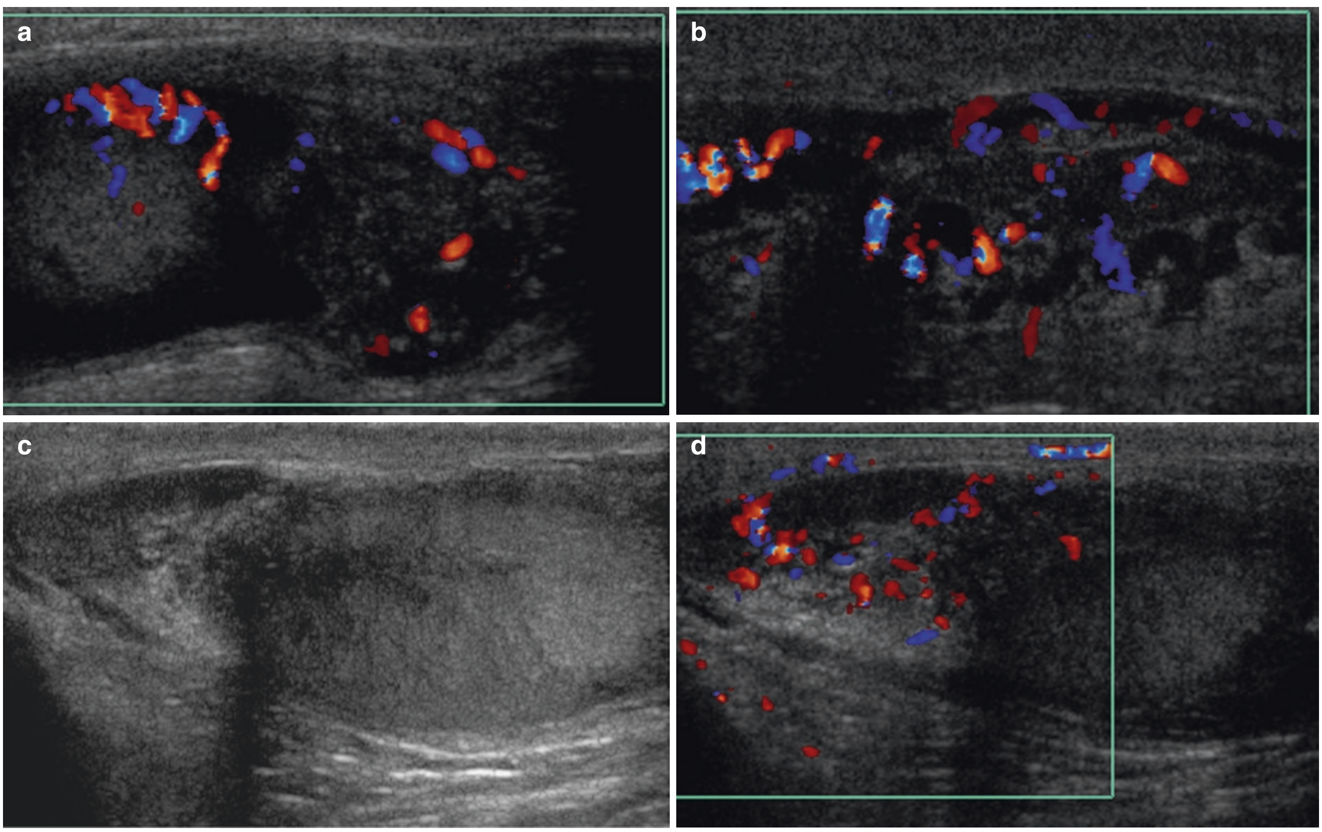

Fig. 4.9 Differential diagnosis between epididymitis and epididymal torsion. Colour Doppler sonography is important in differentiating torsion from epididymitis, with vascularisation invariably reduced in torsion and increased in epididymitis (**a–d**)

with the presence of coarse calcifications in a variably enlarged hypoechoic epididymis (Fig. 4.10) [13]. The organ is generally distorted with an irregular profile, so that the actual size of the epididymis may not be readily appreciated. The examination reveals a hard, irregularly shaped mass adhering to the testis, which can mimic a testicular neoplasm. Thickening of the albuginea and hydrocele are common associated findings. Extension of the infection to the testis can occur but is less common than in acute epididymitis. However, the very firm granulomatous masses can indent the testicular parenchyma, complicating its differentiation from a primary testicular mass. Chronic epididymitis may cause a fibrotic thickening of the tunica albuginea that is sometimes so severe that it is called **pseudotumour of the albuginea** (Fig. 4.11).

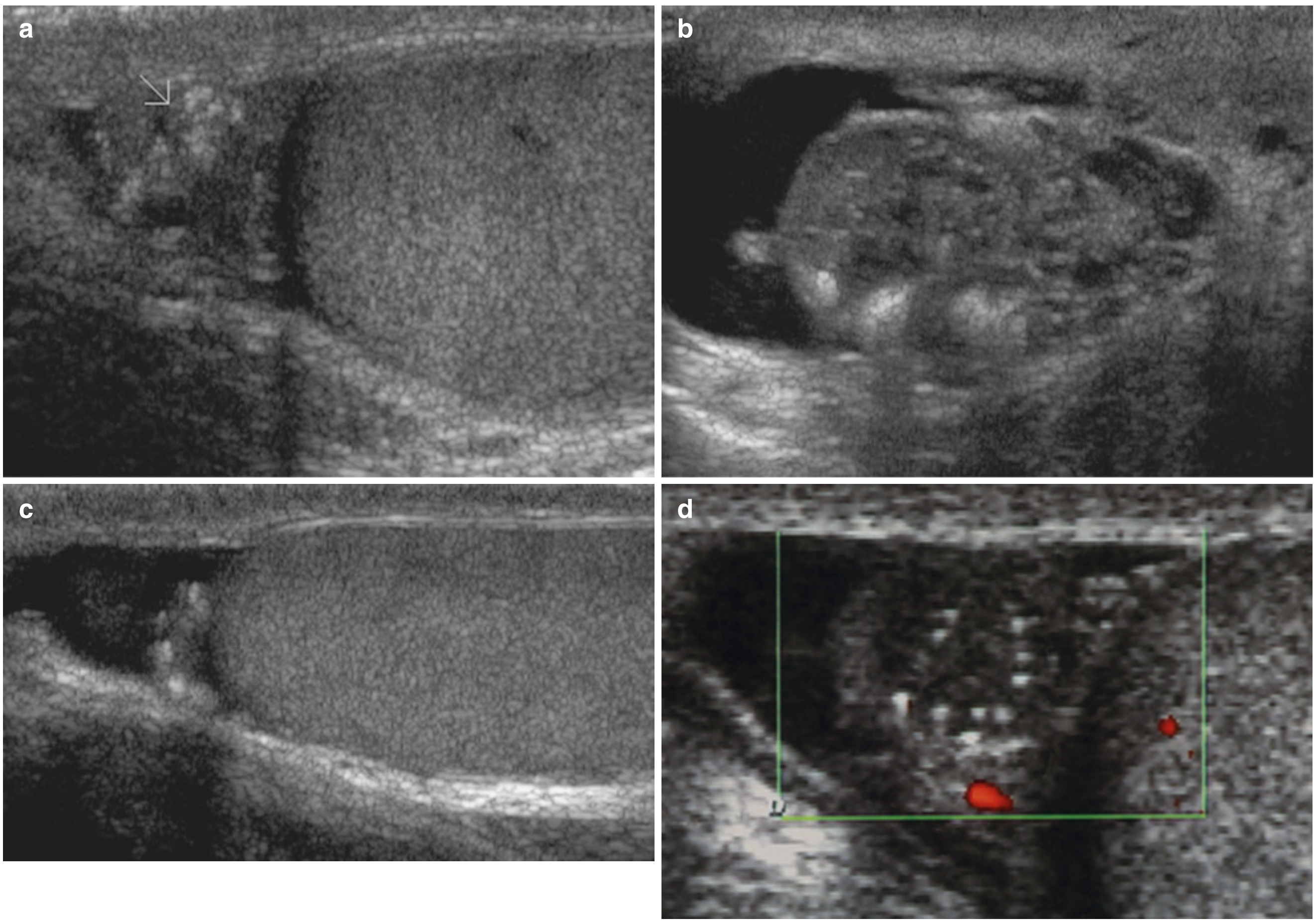

Fig. 4.10 Chronic epididymitis. Longitudinal scans of the testis revealed the presence of enlarged, hypoechoic and inhomogeneous epididymis with coarse calcifications (*panel* **d,** *Courtesy of O.C. Saito, MD)*

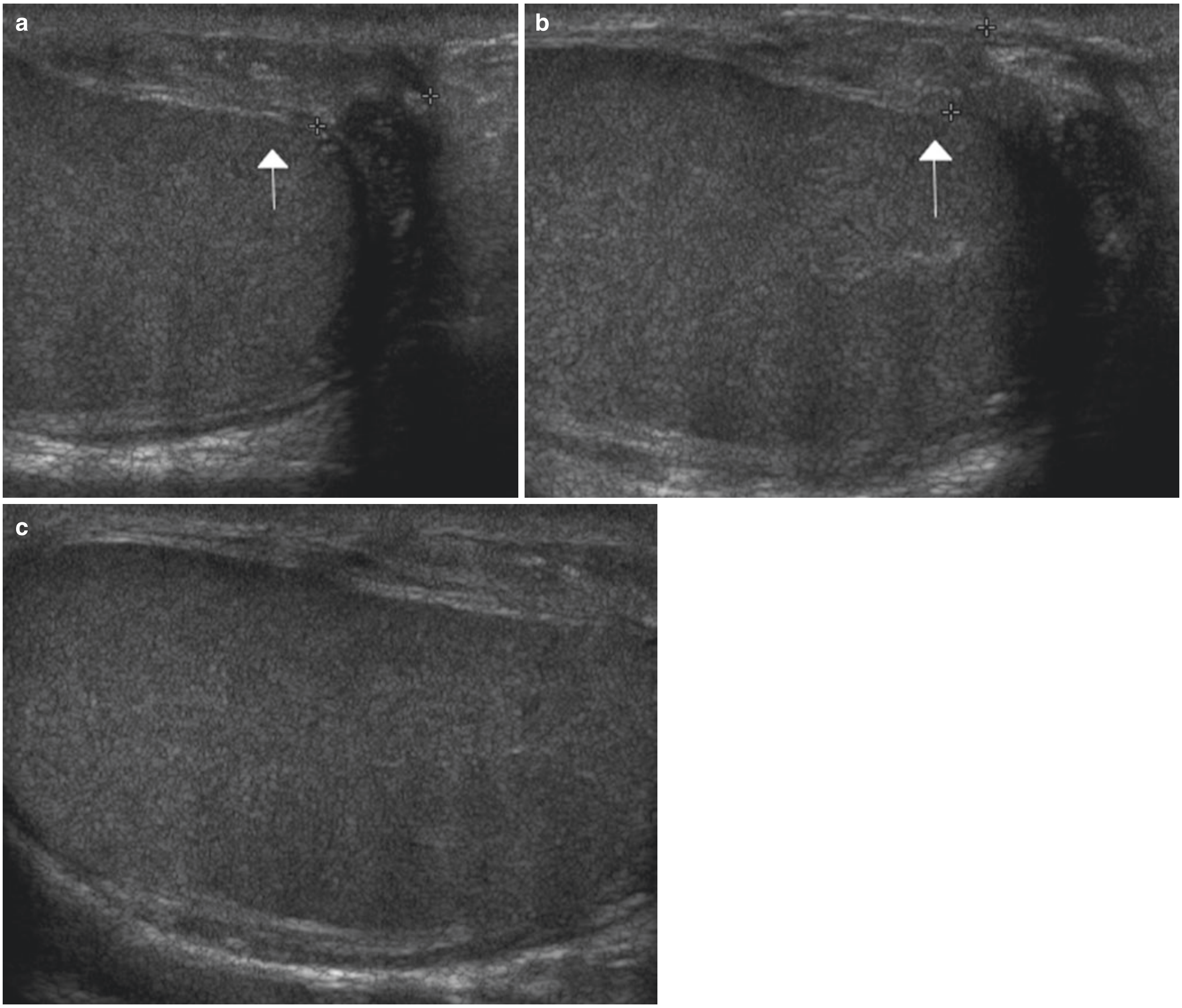

Fig. 4.11 Pseudotumour of the albuginea. Chronic inflammation of paratesticular tissues may cause a fibrotic thickening of the tunica albuginea (*arrow*) that is sometimes so severe to be called pseudotumour of the albuginea (**a**–**c**)

4.2.2.3 Post-inflammatory Epididymal Masses

Sperm granulomas (Fig. 4.12) occur when sperm cells leak out of the vas deferens and generate a foreign-body reaction leading to a necrotising granuloma formation [5, 15]. Sperm granuloma can be clinically detectable as a fibrous lump, painful at palpation. They can be found adjacent to any portion of the epididymis or vas deferens [16].

They are generally less than 1 cm in size. Causative factors include infections, epididymitis, trauma or surgery (vasectomy) [5]. Painful sperm granulomas are a complication of 3–5% of vasectomies and are most frequent within 3 months of the vasectomy, due to nerve compression. On US they appear as solid, smooth, isoechoic or hypoechoic round masses without associated prominent vascularity on colour Doppler imaging.

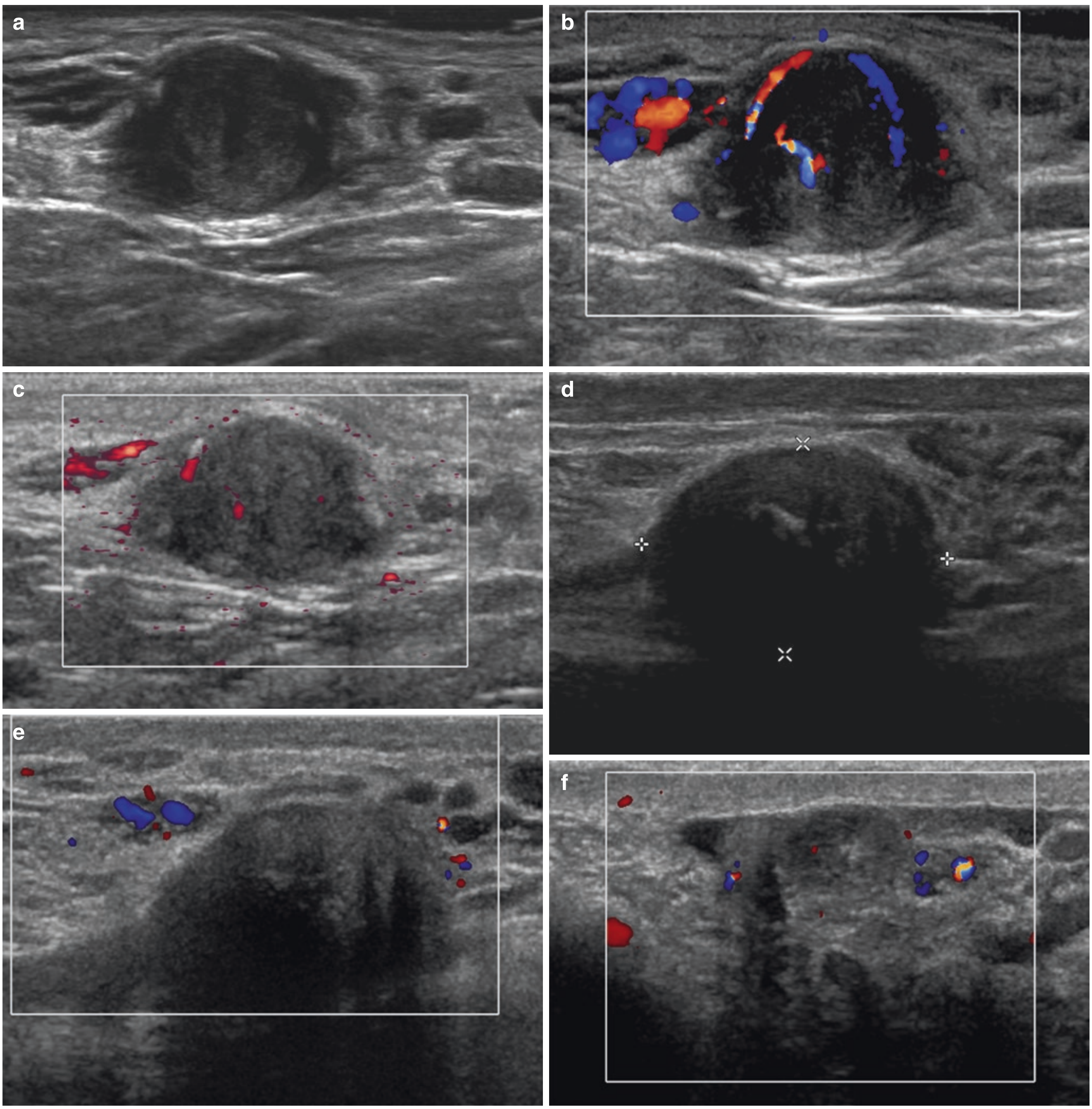

Fig. 4.12 Sperm granuloma. Sperm granuloma occurs when sperm cells leak out of the vas deferens and generate a foreign-body reaction leading to a *necrotising granuloma formation*. They are generally less than 1 cm in size. They can be hypoechoic or isoechoic with the epididymis (**a**–**f**); however, it is the epididymitis to have an overall hypoechoic appearance. Granulomas can also develop post-traumatically as shown in a patient who underwent testicular surgical procedure for suspected testicular torsion (*panels* **e**, **f**)

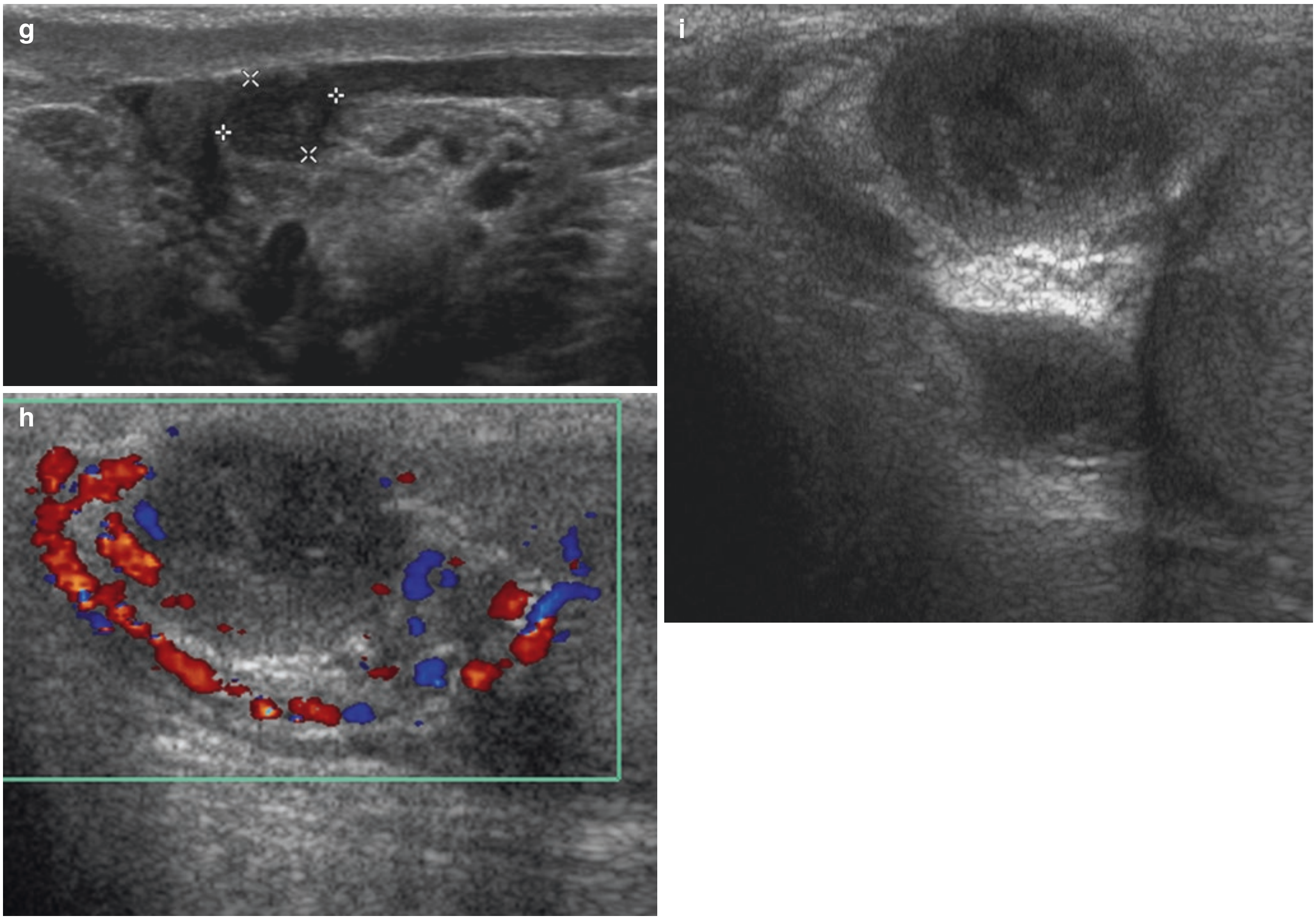

Fig. 4.12 (continued)

They are isoechoic or hypoechoic with the epididymis. They are often only seen when the epididymis moves around them while scanning with palpation [17].

Another paratesticular complication of vasectomy is **epididymitis nodosa**, the epididymal counterpart of vasitis nodosa. This is normally a late postvasectomy complication but has also been described following trauma, herniorrhaphy and prostatectomy, as well as in patients with primary infertility, chronic severe cystitis and bladder diverticula.

The mechanism involved is similar: high-pressure obstruction causing extravasation of spermatic fluid into the vasal interstitium [18]. These lesions are typically small (less than 1 cm) and mostly asymptomatic. The US appearance is similar to that of the adenomatoid tumour.

Sarcoidosis of the genital tract involves the epididymis more frequently than the testis. **Epididymal sarcoidosis** is often asymptomatic, but as the epididymis becomes enlarged, patients may present with a scrotal mass or pain. On US, the epididymis appears enlarged and heterogeneous, and it may have distinct nodules [12].

4.2.3 Epididymal Tumours

4.2.3.1 Benign Epididymal Tumours

Tumour of the epididymis, spermatic cord and paratesticular tissues are rare (ten times less frequent than testicular tumours) and primarily seen in older patients. With the exception of cystadenoma, most of these tumours arise from the mesothelium of the tunica. The large majority are benign. It is not always possible to determine by US whether a tumour originated from the epididymis, the spermatic cord or the tunica vaginalis. For this reason, extratesticular tumours are generally discussed together.

The most common epididymal tumour is the **adenomatoid tumour**, which accounts for approximately 30% of all paratesticular neoplasms, second only to lipoma [1]. It originates from the mesothelium of the tunica vaginalis and is normally located in the tail or near the head of the epididymis. Less frequently, it originates from the tunica albuginea [19] of the testis, with possible intratesticular growth [20]. Adenomatoid tumours are universally considered as benign lesions, although malignant forms have been reported anecdotally. They occur in men of all ages but are most common in the 20–50 age groups. Patients are usually asymptomatic and the tumour is generally unilateral and solitary. Interestingly, adenomatoid tumours are more frequent on the left side. US reveals a smooth, round and well-circumscribed lesion that can vary from a few millimetres up to 5 cm. Associated hydrocele is not a specific feature (15% of cases). Their typical appearance is of an isoechoic (to the epididymis) or hyperechoic (although less than the testis) homogeneous mass, often accompanied by a hypoechoic rim (Fig. 4.13) [15]. Less frequently, they

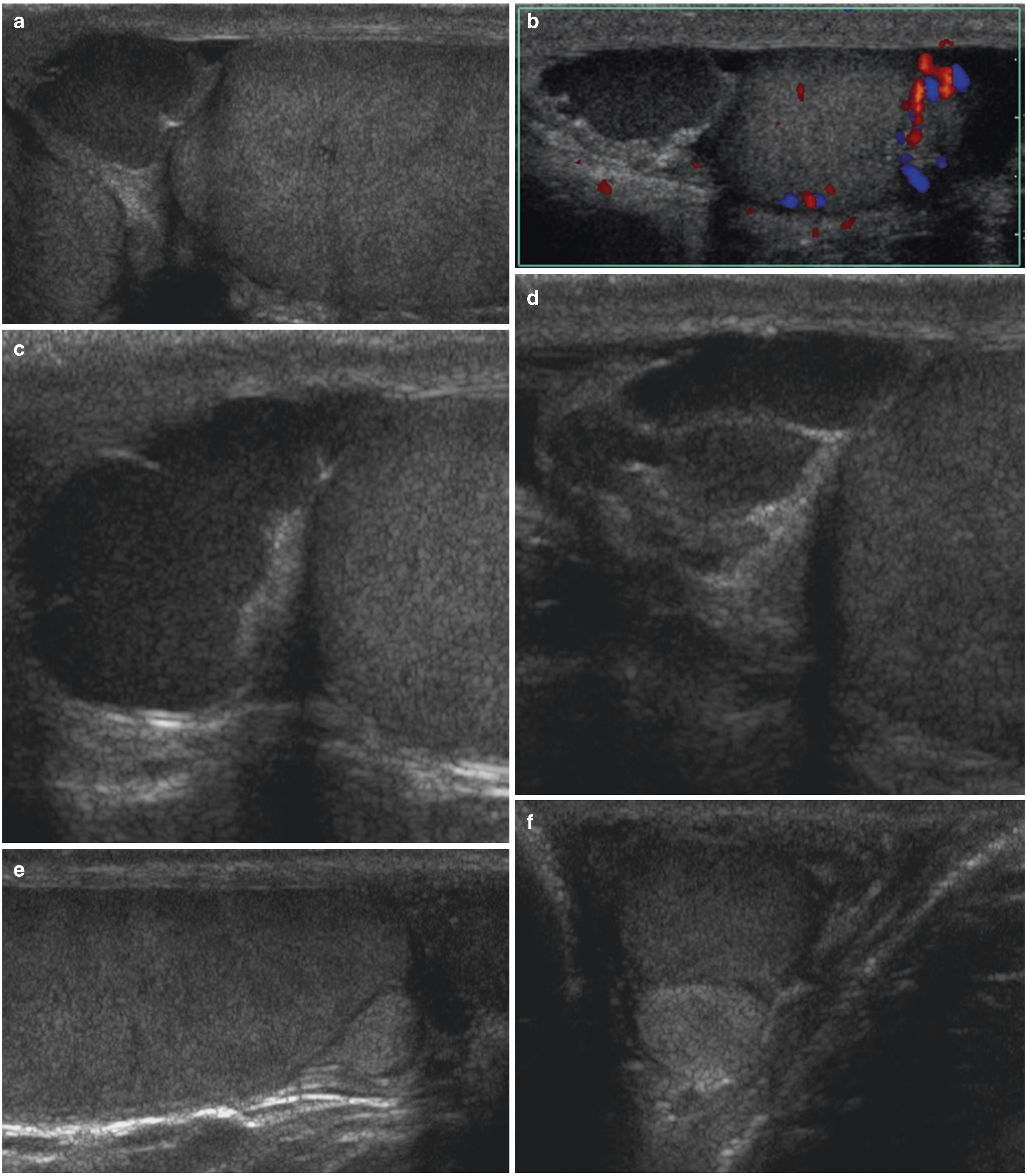

Fig. 4.13 Adenomatoid tumour. US scans reveal a smooth, round, and well-circumscribed and relatively large mass occupying the head of the epididymis. The typical appearance of adenomatoid tumour is of an isoechoic or hyperechoic homogeneous mass, often accompanied by a hypoechoic rim. Panels (**a**–**d**) show an iso-hypoechoic tumour with hyperechoic capsule, whereas in panels (**e**, **f**), there is a hyperechoic lesion with a hypoechoic surrounding rim

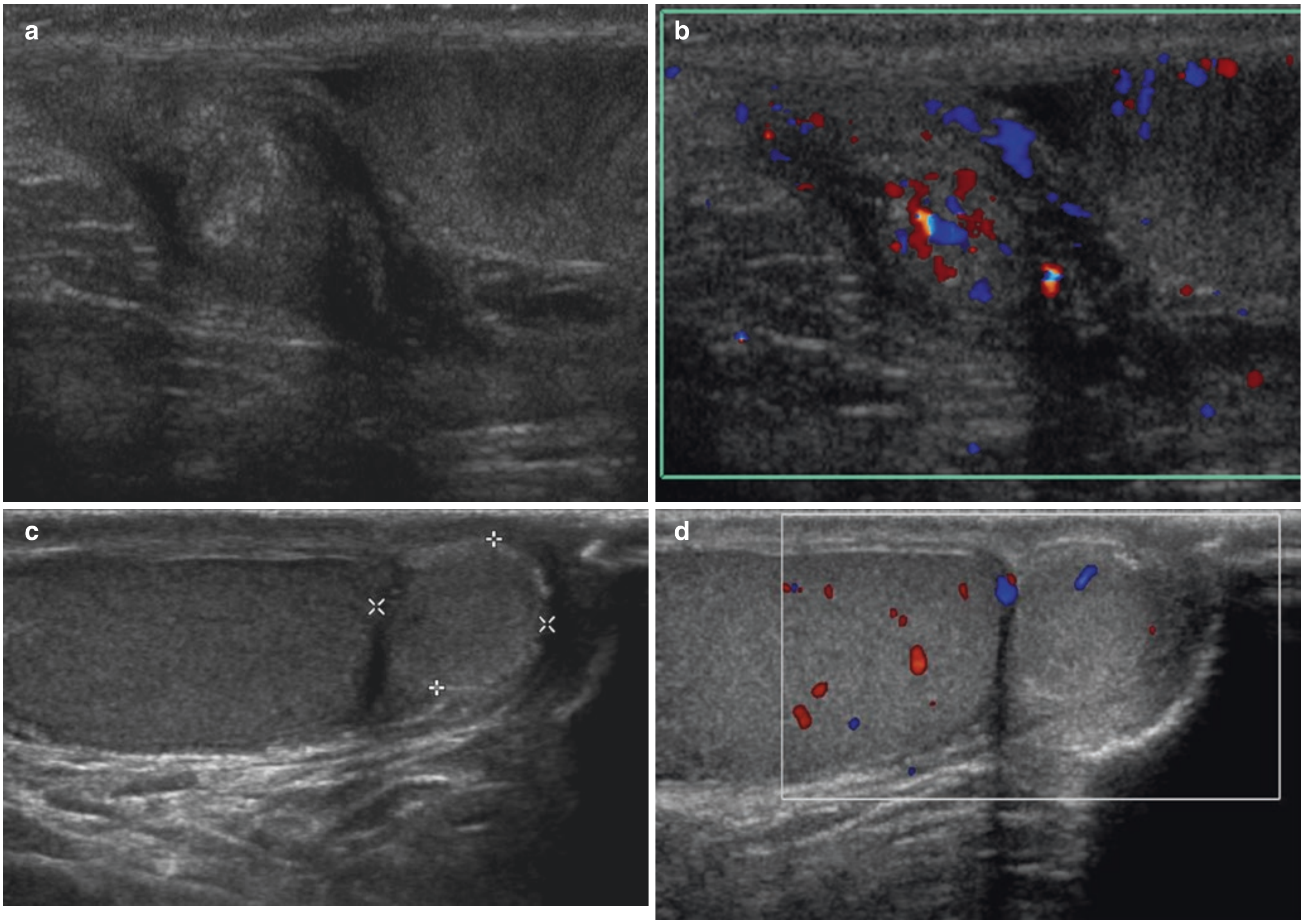

Fig. 4.14 Adenomatoid tumour. Longitudinal scans of the testis reveal a round, inhomogeneous and well-circumscribed lesion adjacent to the head of the epididymis. The lesion has internal vascularisation (**a**, **b**). Another case of adenomatoid tumour in a 35-year-old patient (**c**, **d**)

present as a hypoechoic mass that is indistinguishable from an epididymal granuloma (Fig. 4.14). MR imaging can help determine the paratesticular origin and benign nature of the lesion.

Two other benign epididymal tumours are **leiomyomas** and **papillary cystadenomas**.

On US, epididymal **leiomyomas** are often indistinguishable from adenomatoid tumours. They may display a more heterogeneous pattern, characterised by small echogenic foci with acoustic shadowing consistent with calcifications or multiple, recurrent shadows, unrelated to calcifications, as commonly seen in uterine leiomyomas.

Papillary cystadenomas deserve a special mention due to their association with von Hippel-Lindau (VHL) disease [21], especially when bilateral (40%). They generally manifest as a hard, palpable mass, ranging in size from 1 to 5 cm. The US appearance is typically of a solid nodular mass with very few cystic spaces, but occasionally a multiseptated cystic lesion containing mural nodules and papillary projections is seen (Fig. 4.15). They are usually found in the epididymal head. Efferent duct ectasia is typically present.

Microscopically papillary formations and cystadenomas are mesonephric in origin, arise from the efferent ductules and are the only epididymal tumours of purely epithelial origin. Some consider them to be hamartomas. In the presence of bilateral lesions or predominantly solid lesions larger than 14 mm, the diagnosis of VHL disease should be strongly considered.

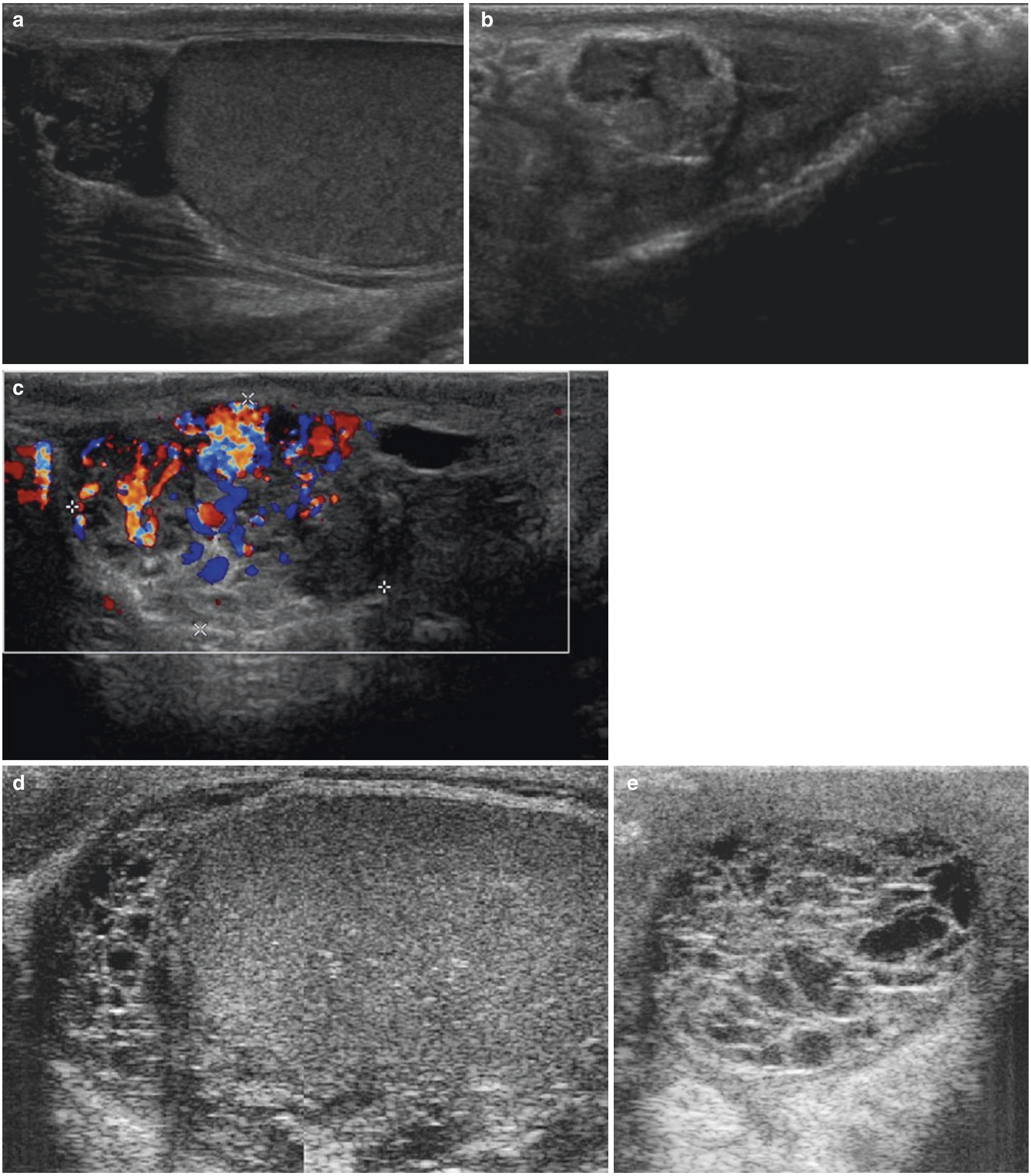

Fig. 4.15 Papillary cystadenoma. The US appearance of a papillary cystadenoma is typically of a solid nodular mass (**a**–**c**, in a patient with von Hippel-Lindau disease) with very few cystic spaces but occasionally a multiseptated cystic lesion (**d**, **e**) *(panels **d**, **e**, Courtesy of: R. H. Oyen, MD)*

Pathologically, papillary cystadenoma must be differentiated from metastatic renal cell carcinoma, which is also common in patients with VHL disease [7]. The differential diagnosis includes adenomatoid tumours, epididymal cysts and, when the mass is predominantly cystic, spermatocele.

Other benign epididymal tumours include **cavernous haemangioma** and **teratoma**.

4.2.3.2 Malignant Epididymal Tumours

Isolated epididymal malignancies are exceedingly rare. A review of the few reported cases reveals large, mainly solid, 'craggy' infiltrating lesions that give a strong suspicion of malignancy, in contrast with the cystic or small, smooth appearance of solid benign lesions.

Malignant tumours of the epididymis generally originate from mesenchymal tissue. **Sarcomas are most common** and **carcinomas** very rare.

The possibility of sarcoma – especially rhabdomyosarcoma – and mesothelioma must be considered. **Metastases and lymphoma** almost invariably follow the involvement of the adjacent testis. Epididymal lymphoma is found in approximately two thirds of cases of testicular lymphoma. This involvement may manifest as multiple nodules or diffuse infiltration. In this case, it must be differentiated from sarcoidosis and tuberculosis, which can also cause simultaneous epididymal and testicular masses. In the latter two conditions, the epididymis is usually involved to a much greater extent than the testis, while in lymphoma the situation is reversed [12].

Metastases to the epididymis are most commonly caused by carcinoma of the prostate. Mechanisms include venous extension or embolisation, arterial embolisation, retrograde lymphatic extension or intraluminal spread via the vas deferens. Metastasis to the epididymis is usually discovered in patients undergoing castration for widely disseminated or locally extensive prostate carcinoma. Other, less common sources are gastric carcinomas [5].

Rhabdomyosarcoma is a relatively frequent primary malignant extratesticular tumour, mainly involving the spermatic cord, although it can also affect the epididymis. It occurs predominantly in children and adolescents. **Mesothelioma** of the tunica vaginalis is another extratesticular tumour that can be malignant in up to 30% of cases.

Malignant tumours of the epididymis, spermatic cord and paratesticular tissue (Fig. 4.16) [22, 23] are all characterised by a fast-growing, hard, painless mass with ill-defined margins. These tumours are not clearly identifiable by US, and diagnosis is made on the basis of patient history and radical orchiectomy. Most of these lesions are solid, with a heterogeneous echogenic internal pattern. Benign lesions are also mainly solid, with the exception of cystoadenoma, which can have a cystic component. However, benign lesions present as very well-defined lesions with no or imperceptible growth. In the case of cystic lesions, the differential diagnosis is towards spermatocele and complex epididymal cysts. Hydrocele is a frequent accompanying sign, especially with mesothelioma (see next paragraph).

4.3 Spermatic Cord Lesions

Pathological findings involving the spermatic cord include an inflammatory process (funiculitis), the presence of cysts and other conditions interrupting the continuity of the deferent duct, resulting in the relatively frequent lipoma and malignant tumours – the juvenile rhabdomyosarcoma – as well as benign tumours.

4.3.1 Funiculitis

The spermatic cord is often involved in the inflammatory process: this is termed funiculitis. The ultrasonic signs are swelling and hyperaemia of the cord (Fig. 4.17). Inflammation of the spermatic cord is usually less marked than that of the epididymis, although in some cases it is the main area affected by inflammation and other symptoms. Severe funiculitis may rarely cause compression of the cord vessels within the inguinal canal, causing testicular ischaemia.

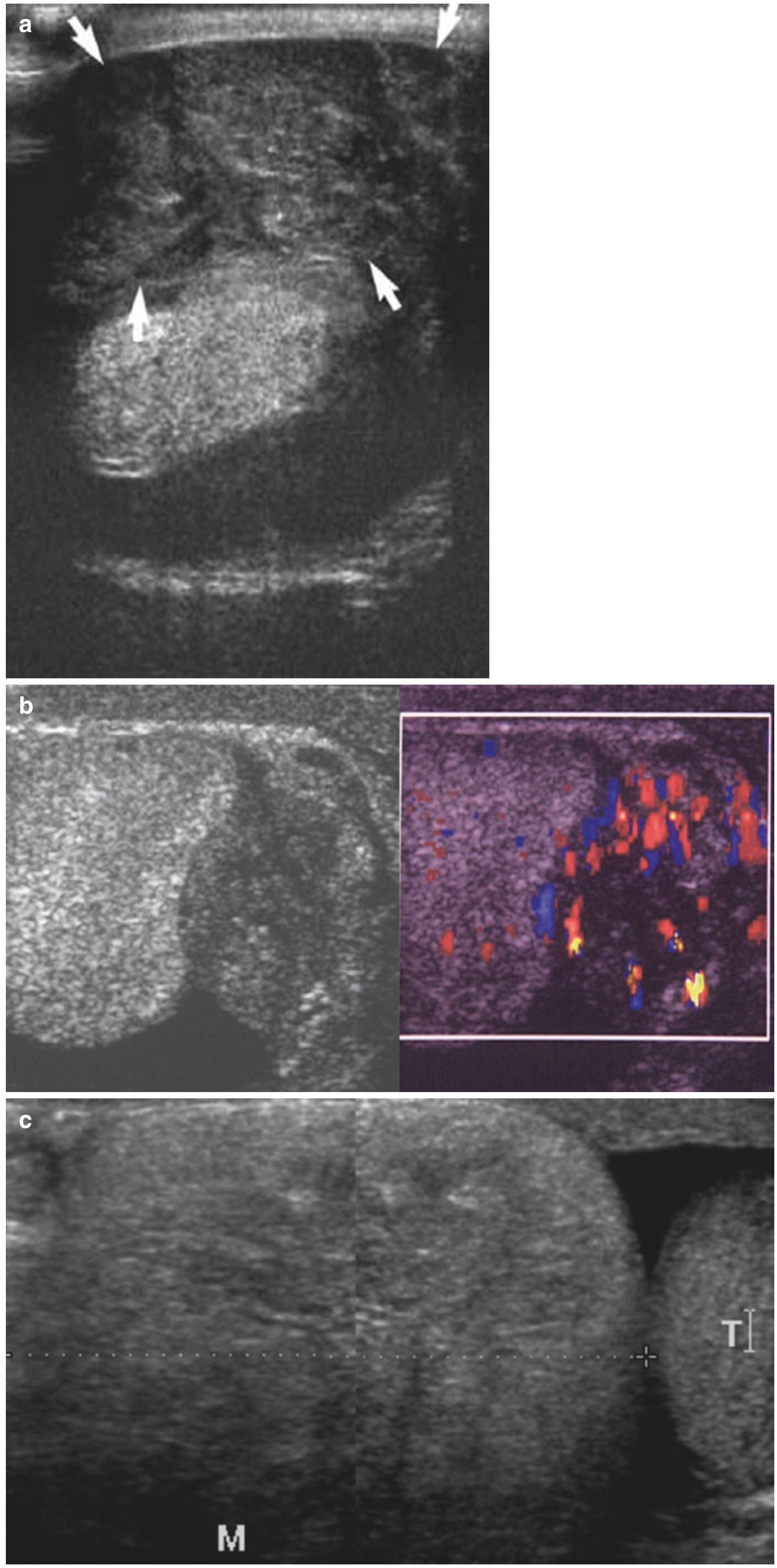

Fig. 4.16 Malignant epididymal and paratesticular tumours. Malignant tumours of the epididymis, spermatic cord and paratesticular tissue are all characterised by a fast-growing, hard mass with ill-defined margins. These tumours are not clearly identifiable by US, and diagnosis is made on the basis of patient history and radical orchiectomy. In figure (**a**), there is a rhabdomyosarcoma. Transverse ultrasound shows heterogeneous, ill-defined mass (*arrows*) within and surrounding the left testis. In figure (**b**) an adenocarcinoma of the epididymis is shown. In figure (**c**), a liposarcoma is shown *(image **a** From: R. Gupta, M. Alobaidi, S.Z. Jafri et al. 'Correlation of US and MRI Findings of Intratesticular and Paratesticular Lesions: From Infants to Adults' Curr Probl Diagn Radiol 2005;34:35–45; image **b** From: J.M. Smart, E.K. Jackson, S.L. Redman et al. 'Ultrasound findings of masses of the paratesticular space' Clinical Radiology (2008) 63, 929**e**938; image **c** Courtesy of D.C. Howlett, MD)*

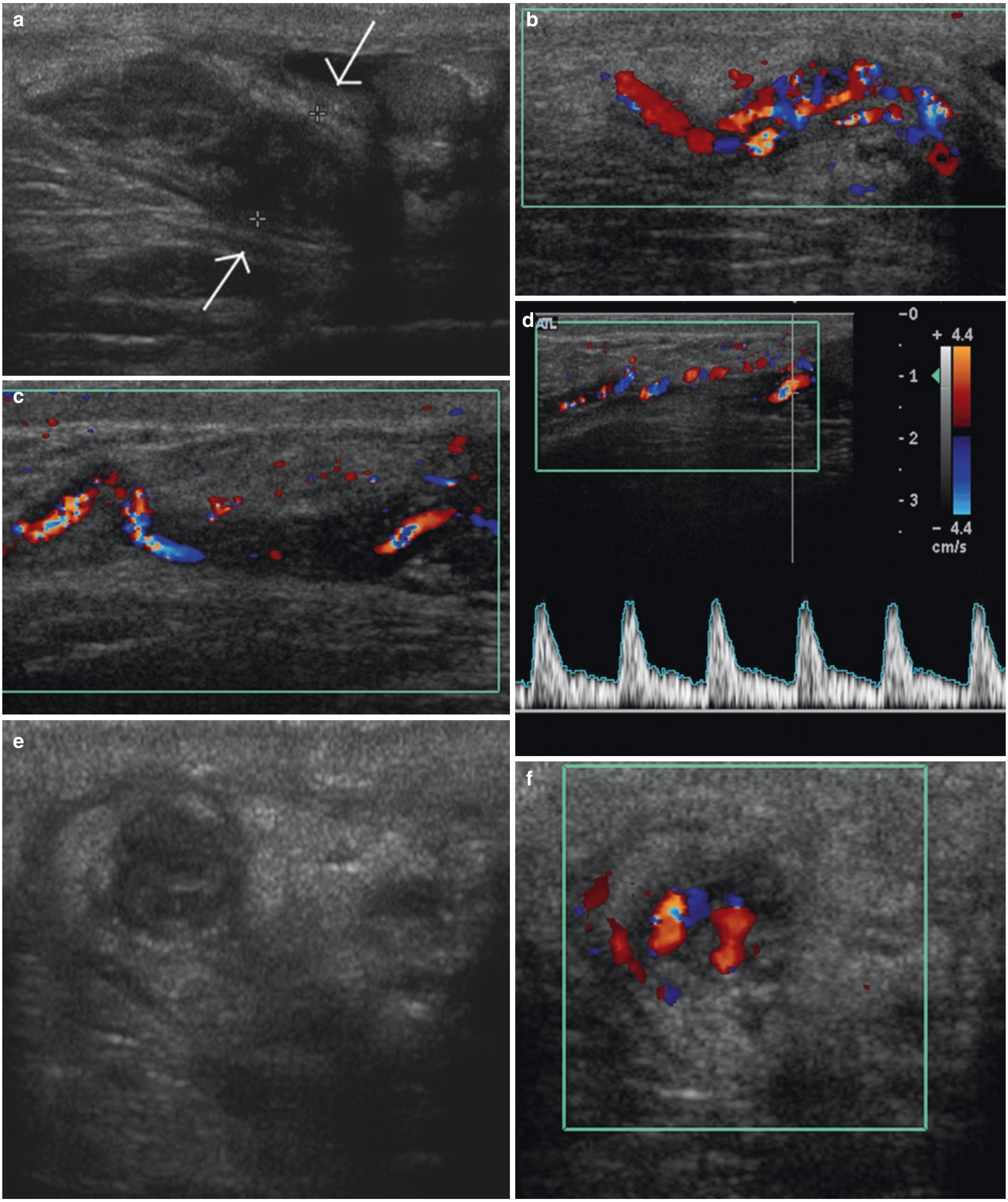

Fig. 4.17 Funiculitis. Longitudinal and transverse scans of the spermatic cord reveal swelling, thickening (**a**, **e**) and hyperaemia (**b**–**d**, **f**) of the spermatic cord. Transverse scan is shown in (**e**) and (**f**) panel. Funiculitis usually occurs in severe cases of epididymo-orchitis, even if sometimes the inflammation of the cord predominates

4.3.2 Spermatic Cord Tumours

Lipoma is the most frequent benign tumour of the spermatic cord (Fig. 4.18). It is most often found by chance, as a non-tender scrotal mass in patients from a wide age range. Lipomas commonly appear as a round, well-defined, low to medium intensity mixed echogenic soft mass that can be easily deformed by the pressure of the transducer. Their echogenicity varies with the varying proportion of fat cells and fibrous, myxoid or vascular tissue, which increase its echogenicity [24, 1]. In consequence, they are often hyperechoic on US, although this is far from sensitive or specific. **Lipomas** can be microscopic or grow progressively to a very large mass (up to 3.2 kg) [21] and can occur at any age. A condition frequently detected in obese men is the lipomatosis of the spermatic cord, which appears as a pasty, medium intensity, fatty infiltration of the spermatic cord. The differential diagnosis is towards hernia or herniation of the perivisceral fat [24–26].

Like hernias, lipomas can also be hyperechoic and involve the spermatic cord. Given the highly variable US appearance, differential diagnosis can only be made by patient history, lesion consistency and size and its borders. MR imaging with fat-suppressed sequences can confirm the diagnosis. However, their rare malignant counterpart, the liposarcoma, cannot be easily distinguished. Large lipomas are therefore generally removed.

Rhabdomyosarcoma (Fig. 4.16) is the most important malignant tumour of the spermatic cord. In contrast with liposarcoma, the second most frequent malignant spermatic cord tumour in adults, rhabdomyosarcoma is frequently seen in children (median age 7 years) [12], accounting for up to 30–40% of all malignant paratesticular neoplasms in the young. US features are non-specific. They are usually characterised by a variable echogenicity with a heterogeneous appearance, due to haemorrhage and necrosis. Colour Doppler shows increased flow and low resistance. A clinical history of a child with a generally painless, fast-growing firm mass, which may envelop or clearly invade the epididymis and testis, should raise suspicions. The differential diagnosis is towards other common paratesticular tumours such as fibromas, leiomyomas and adenomatoid tumours. A CT scan is necessary to estimate local invasion [12, 1].

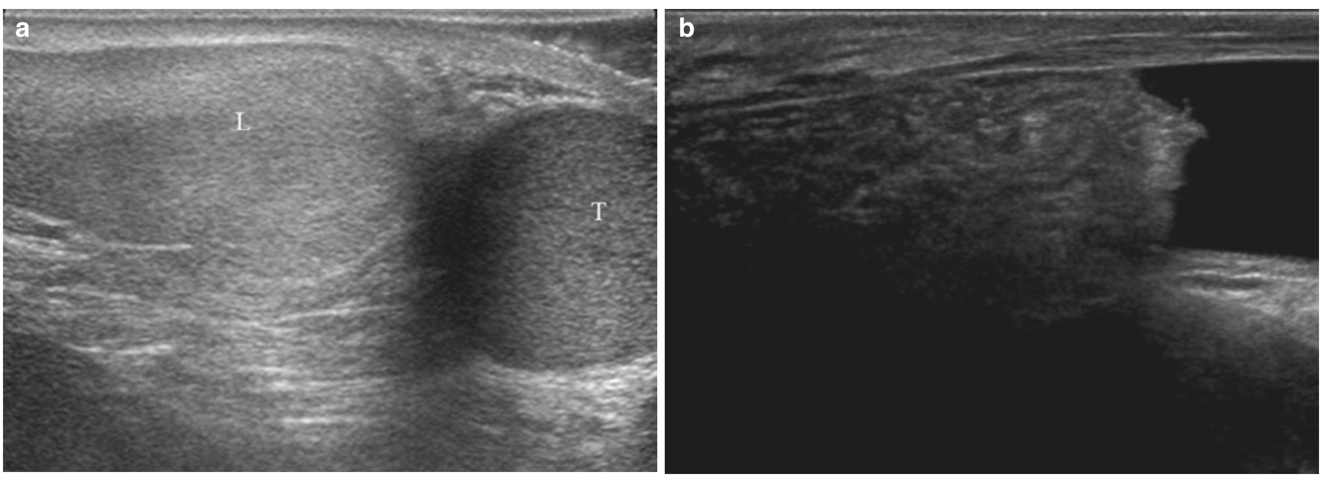

Fig. 4.18 Lipoma of the spermatic cord. (**a**) Longitudinal scan of the scrotum shows a round, well-defined, solid soft mass that can be easily deformed by the pressure of the transducer, characteristics well-suited with lipoma (L). The echogenicity of the tumour varies with the varying proportion of fat cells and fibrous, myxoid or vascular tissue. The differential diagnosis is towards scrotal hernias (**b**) *(panel **a**, Courtesy of M. Muttarak, MD)*

4.4 Tunica Lesions

The tunica vaginalis covers all but the posterior aspect of the testis. Several pathological conditions can involve the tunica vaginalis and the virtual space between the parietal and visceral layers, including reactive inflammatory processes, tumours and fluid collections.

4.4.1 Fibrous Pseudotumour

Fibrous pseudotumour covers a variety of conditions also known as chronic periorchitis, fibrous proliferation of the tunica, benign fibroma, non-specific paratesticular fibrosis, granulomatous periorchitis, nodular fibrous pseudotumour, inflammatory pseudotumour and reactive periorchitis. **Fibrous pseudotumour** is a non-neoplastic lesion caused by a proliferation of reactive fibrous tissue. On physical examination, the lesion may be difficult to distinguish from a testicular or extratesticular neoplasm [5]. The condition has also been clinically described as 'scrotal mouse' due to mobile masses within the tunica vaginalis [12], as these fibrotic proliferations can occasionally detach from the tunica and become freely mobile within the scrotal sac (like scrotal pearls). Briefly, fibrous pseudotumour is a benign, reactive fibrous proliferation of paratesticular tissue, most frequently arising from the tunica vaginalis (approximately 75%) [1, 4]. The underlying pathophysiological mechanism is an inflammatory process, and a reactive nature is suggested in some cases by a history of trauma, surgery, infection or hydrocele. However, in many cases, a relevant history is absent. These masses can be large and mimic neoplasms. US evaluation generally shows one or more solid masses typically attached to or closely associated with the capsule of the testis. The appearance is often of a hypoechoic mass with mixed echogenic areas and lobulated margins, with no evident associated vascularisation. Alternatively, they can appear as a diffuse thickening or plaque-like process of the testicular capsule or epididymis. Nearly 50% of affected patients have an associated hydrocele or haematocele, with 30% having a prior history of trauma or epididymo-orchitis [1]. Various degrees of calcification, with associated acoustic shadowing, are often present [12].

4.4.2 Mesothelioma

The tunica vaginalis is lined by mesothelial cells, which in rare cases may give rise to mesotheliomas. Scrotal mesotheliomas are less common than those of the pleural or peritoneal compartments; however, they do occur and should be recognised as a potentially aggressive malignant disease. Mesothelioma can occur at any age but is most often found in patients aged over 18. Fifteen to twenty percent of mesotheliomas are benign; however distinction between the benign and malignant forms is not possible by US and is mainly based on the lesion's clinical behaviour.

On US scans [27, 28], scrotal mesotheliomas most frequently appear as multiple solid nodules studding or infiltrating the surface of the tunica vaginalis. Known presentations include solitary nodules on the epididymis, papillary projections on the surface of the tunica vaginalis [24, 29] and ill-defined diffuse thickening of the tunica vaginalis in patients with hydrocele. Only a minority of mesotheliomas appear cystic [12].

Benign mesotheliomas can be treated by a simple excision of the hydrocele and do not normally compromise the gonad. Non-papillary benign mesotheliomas commonly arise from the epididymal tail, although they can also originate from the tunica albuginea or spermatic cord. They may even have an intratesticular origin. Sperm granulomas have a similar appearance on imaging [24].

Malignant mesotheliomas are very aggressive tumours that normally infiltrate local soft tissues and metastasise to retroperitoneal lymph nodes. These masses are heterogeneous, with acoustic shadowing. They can be differentiated from benign mesotheliomas by the rapid reformation of fluid, the presence of larger nodular or papillary lesions and the presence of blood in the hydrocele. The differential diagnosis is towards liposarcoma, rhabdomyosarcoma, lymphoma, fibrosarcoma and rarer tumours such as malignant schwannoma and malignant fibrous histiocytoma [1]. All these tumours are often large at the time of presentation. Occasionally, cystic alteration/malformation of the vaginalis or fibrous pseudotumour can mimic mesothelioma (Fig. 4.19).

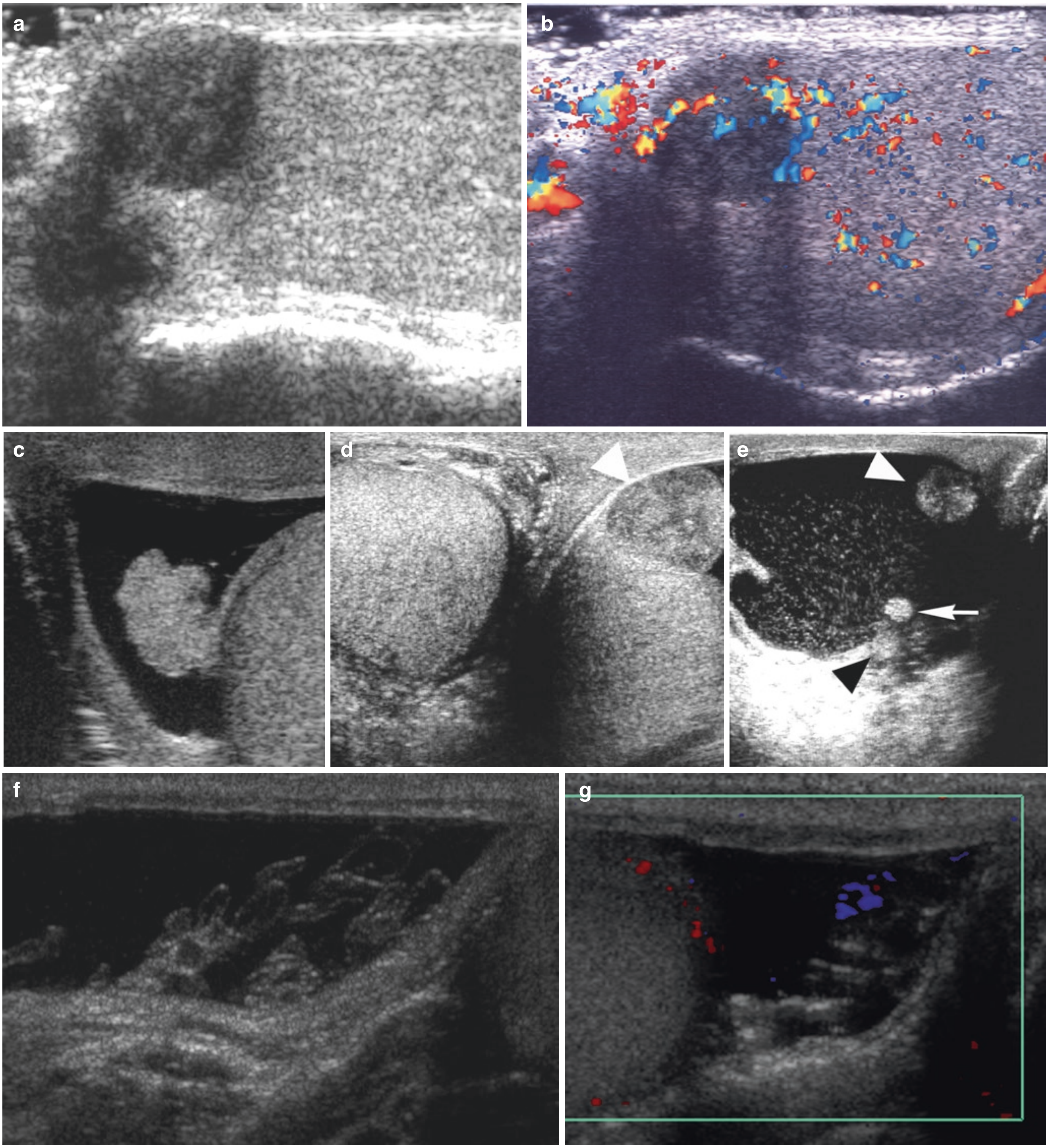

Fig. 4.19 Mesothelioma. In figures (**a** and **b**), an atypical mesothelioma of the tunica vaginalis is shown. Longitudinal sonogram of the right testis shows a hypoechoic upper-pole mass extending beyond the contour of the testicle. Colour Doppler sonogram shows the mass to be vascular, although less so than the testicular parenchyma. In figure (**c**), a typical mesothelioma of the tunica vaginalis is shown. In figures (**d** and **e**), transverse scrotal sonogram shows a nonhomogeneous nodule seemingly arising from the superior pole of the left testicle (*arrowhead*) and another similar nodule arising from the tunica vaginalis (*arrowheads*). A small scrotolith is also present (*arrow*). Occasionally cystic malformation of the tunica vaginalis can mimic mesothelioma (**f**, **g**) (*panels **a**, **b**, From: K. Wolanske, MD, M. Nino-Murcia, MD, 'Malignant Mesothelioma of the Tunica Vaginalis Testis. Atypical Sonographic Appearance'*. Published in: *J Ultrasound Med (2001) 20:69–72; panel **c**, Courtesy of: RH. Oyen, MD; panels **d**, **e**, From: J.B.A. Boyum, N.F. Wasserman, 'Malignant Mesothelioma of the Tunica Vaginalis Testis', J Ultrasound Med (2008); 27:1249–1255)*

4.5 Extratesticular Calcifications

Extratesticular calcifications are a frequent finding on scrotal sonography. Calcifications of the epididymis may be seen in older patients with a previous history of chronic bacterial or tuberculous epididymitis or epididymal trauma (Fig. 4.18).

Calcifications formed between the two layers of the tunica vaginalis are also known as **scrotal pearls**. They are commonly identified in the presence of a hydrocele and may be adherent or mobile. They result from inflammation of the tunica vaginalis or remnants of the appendix testis or epididymis that have detached secondary to torsion and subsequently calcify (Fig. 4.18). Patients with chronic hydroceles or previous haemorrhages or infections may have thin curvilinear calcifications on the surface of the tunica vaginalis (Fig. 4.18). This condition is relatively common and is also known as **pachyvaginalitis** (Fig. 4.20). Calcification in the pampiniform plexus (phleboliths) is less commonly seen in patients with varicocele or those who have undergone varicocele repair. The presence of a number (>3–4) of extratesticular scrotal calcifications should be interpreted as a sign of a previous traumatic event or infection, and appropriate screening is recommended.

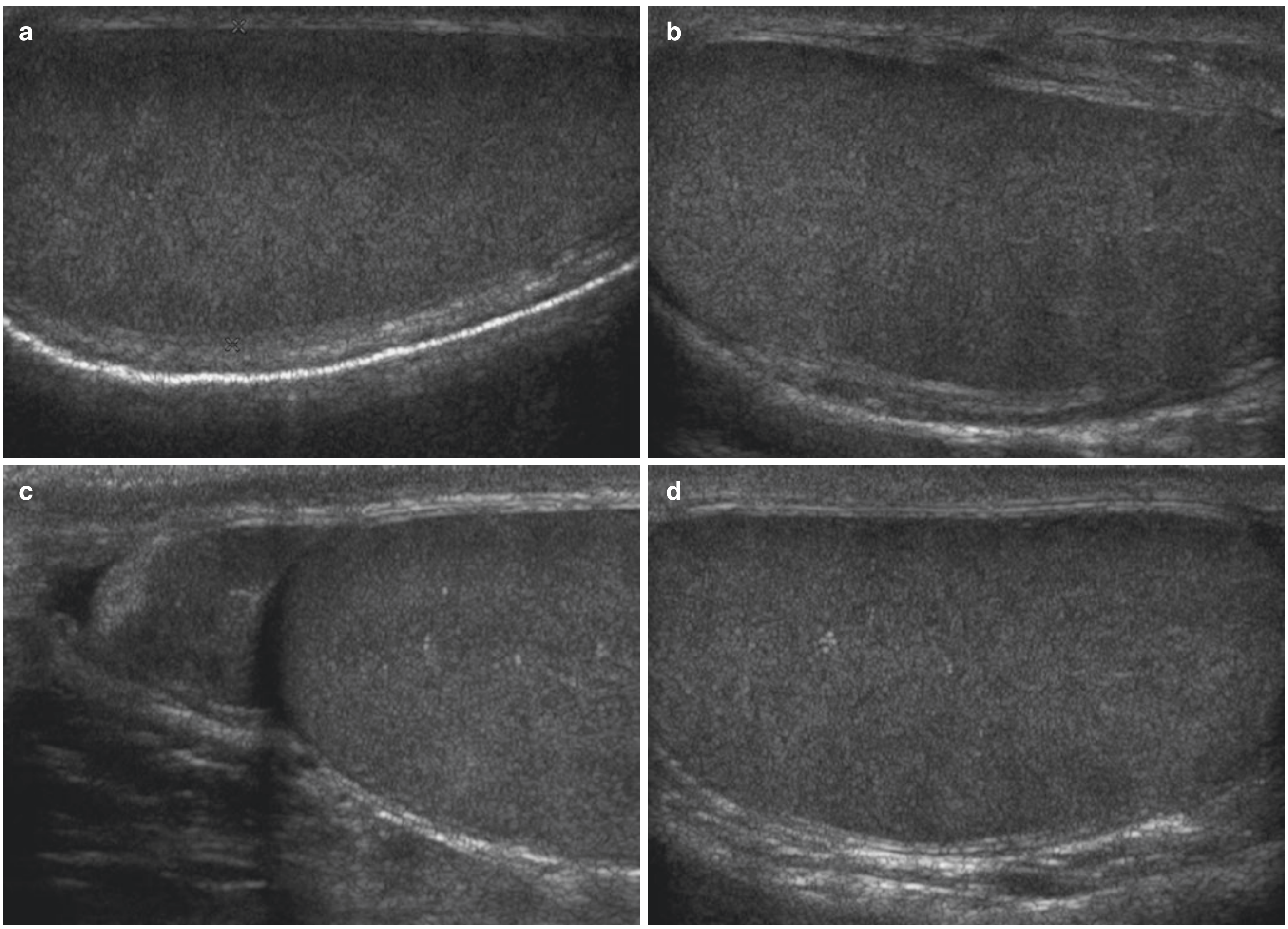

Fig. 4.20 Pachivaginalitis. Longitudinal scans of testes showing thickening of the tunical layers (**a**–**f**) that appear hyperechoic with occasional calcifications, fibrotic changes (**g**, **h**) and detachment of debris (scrotal pearls) (**i**)

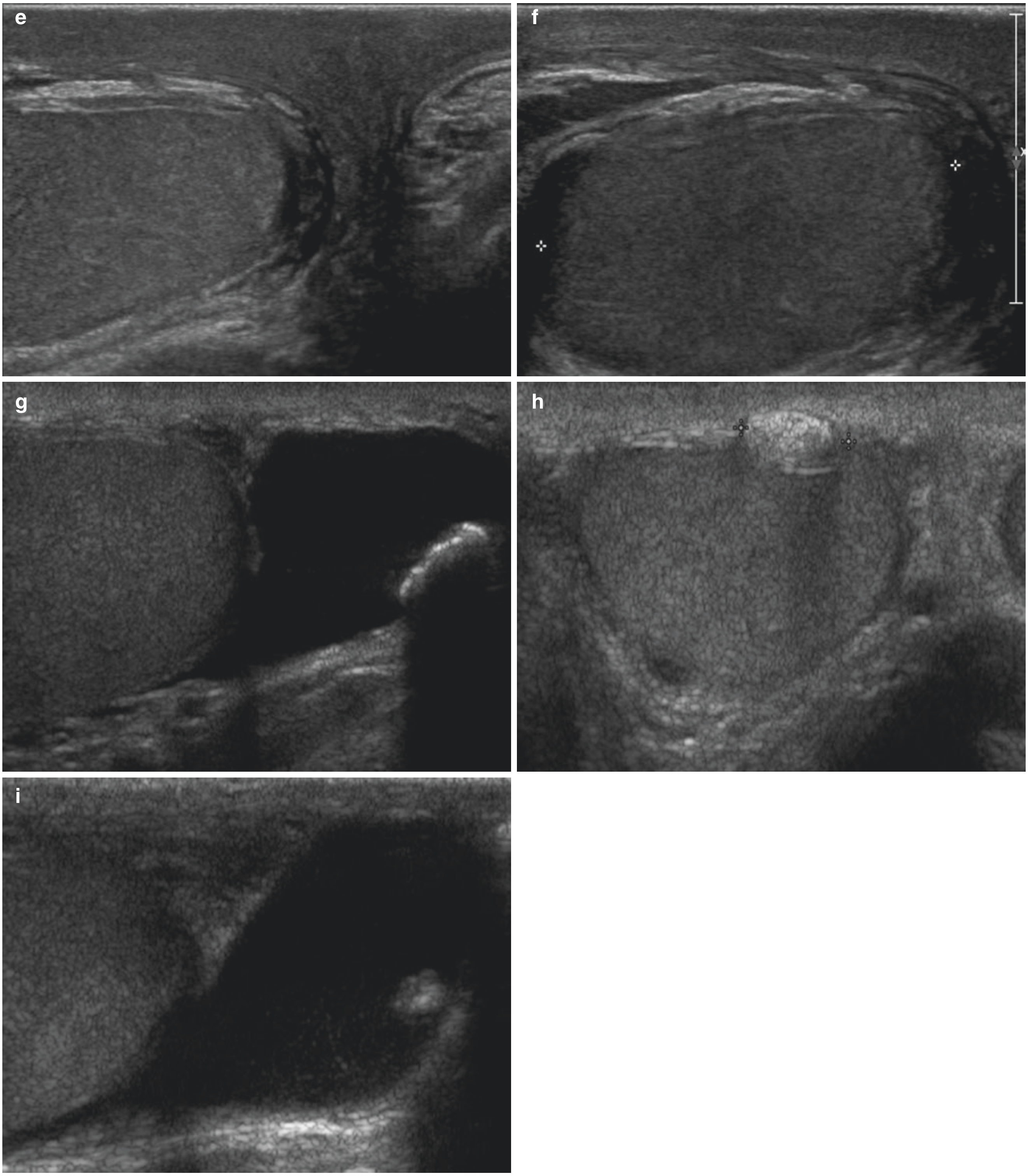

Fig. 4.20 (continued)

4.6 Fluid Collections

The scrotum normally contains a few millimetres of serous fluid between the two layers of the tunica vaginalis, lining the testis and scrotal sac. This serous fluid may function as a lubricant and is usually visible on US examination [5, 12]. Fluids can accumulate in the space between the parietal and visceral layers of tunica vaginalis: normal serous fluid but also blood, pus or urine can produce a swelling of the scrotal sac. As the tunica vaginalis covers the posterior aspect of the testis, fluid collections within its layers are confined to the anterior and lateral aspects of the hemiscrotum [5, 15, 16].

Hydrocele is an abnormally large collection of the serous fluid normally present between the two tunica layers. It is among the commonest causes of painless scrotal swelling and may be congenital or acquired [30]. Congenital hydroceles occur when there is an incomplete closure of the processus vaginalis, allowing communication between the peritoneal cavity and scrotum. They are often seen in baby boys (6% at delivery) and usually resolve by spontaneous absorption by the age of 18 months [5, 12].

Acquired hydroceles may be idiopathic or secondary. The mechanism responsible for idiopathic hydroceles is unclear; they may result from excessive fluid production, the failure of the mesothelial lining to reabsorb fluid or possibly the absence of efferent lymphatics. Impaired reabsorption is considered the most likely cause.

In 25–50% of cases, acquired hydroceles form as a reaction to trauma. Other causes of secondary hydroceles include infection (epididymo-orchitis), infarction, torsion and neoplasm. They are also a common complication of surgical varicocele repair. Retroperitoneal neoplastic infiltration or fibrosis can also affect normal lymphatic drainage and cause hydrocele.

In communicating hydroceles resulting from a patent processus vaginalis, fluid can be moved during clinical examination and increases with raised intra-abdominal pressure. A non-communicating hydrocele is not affected by palpation or positional change. Chronic hydrocele is enveloped by a thick wall and thick septa [24] and is frequently accompanied by extratesticular calcifications. It does not transilluminate. Hydroceles are bilateral in 10% of patients.

A small hydrocele is also found in association with testicular cancer, whereas a large fluid collection is more typically suggestive of an idiopathic hydrocele or an inflammatory cause. The presence of blood cells in the fluid suggests trauma, infection or malignant paratesticular tumours.

US diagnosis of hydroceles is readily made with the detection of the characteristically anechoic collections with good ultrasound transmission surrounding the anterolateral aspects of the testis (Fig. 4.21) [5]. In contrast, large spermatoceles can also involve the posterior aspect of the testis.

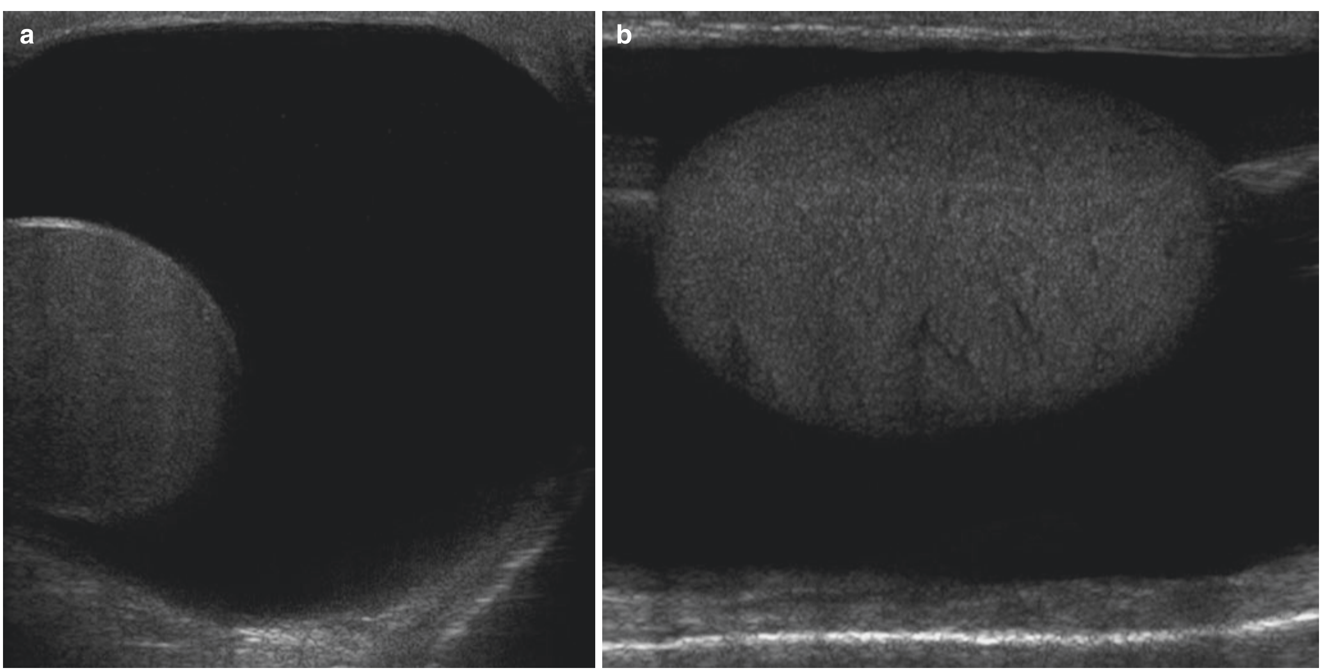

Fig. 4.21 Hydrocele. Hydrocele is a typically anechoic collection with good ultrasound transmission surrounding the anterolateral aspects of the testis (**a**–**e**). The appendix testis is seen projecting from the testis (**f**)

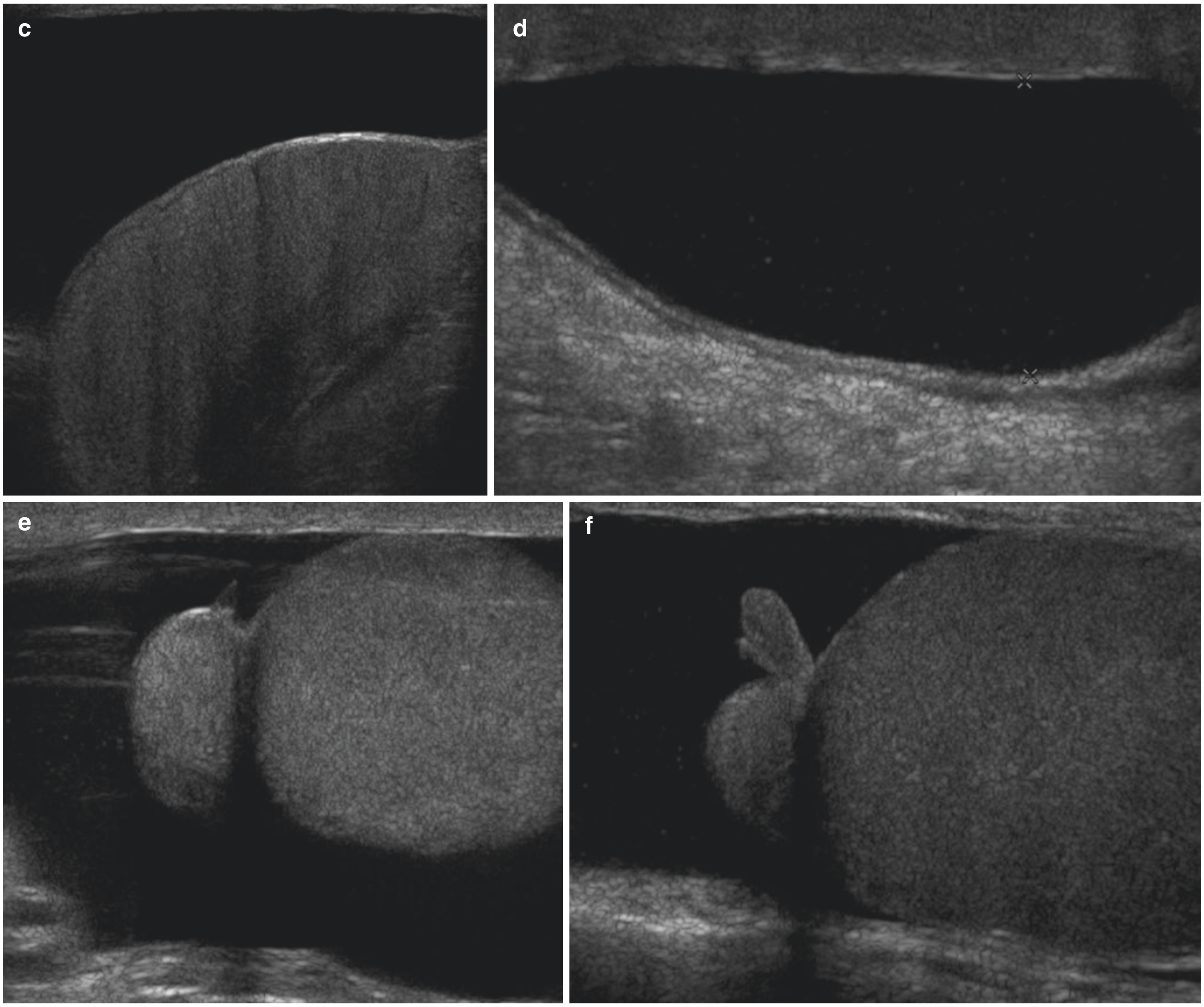

Fig. 4.21 (continued)

The frequent finding of internal echoes floating in the hydrocele is explained by cholesterol crystals, blood (haematocele), pus (pyocele), fibrin strands or hernia. In many cases, freely movable calcifications (scrotal phleboliths, scrotal pearls or hydrocele concretion) are seen [24]. It is generally thought that recently or acutely formed hydroceles should be echo-free collections of fluid, in contrast with chronic conditions. However, with high-resolution systems, very fine swirling echoes, due to crystals, are nearly always seen.

Hydroceles secondary to epididymo-orchitis often contain echogenic material representing an inflammatory exudate (Fig. 4.22). This may later result in echogenic fibrous

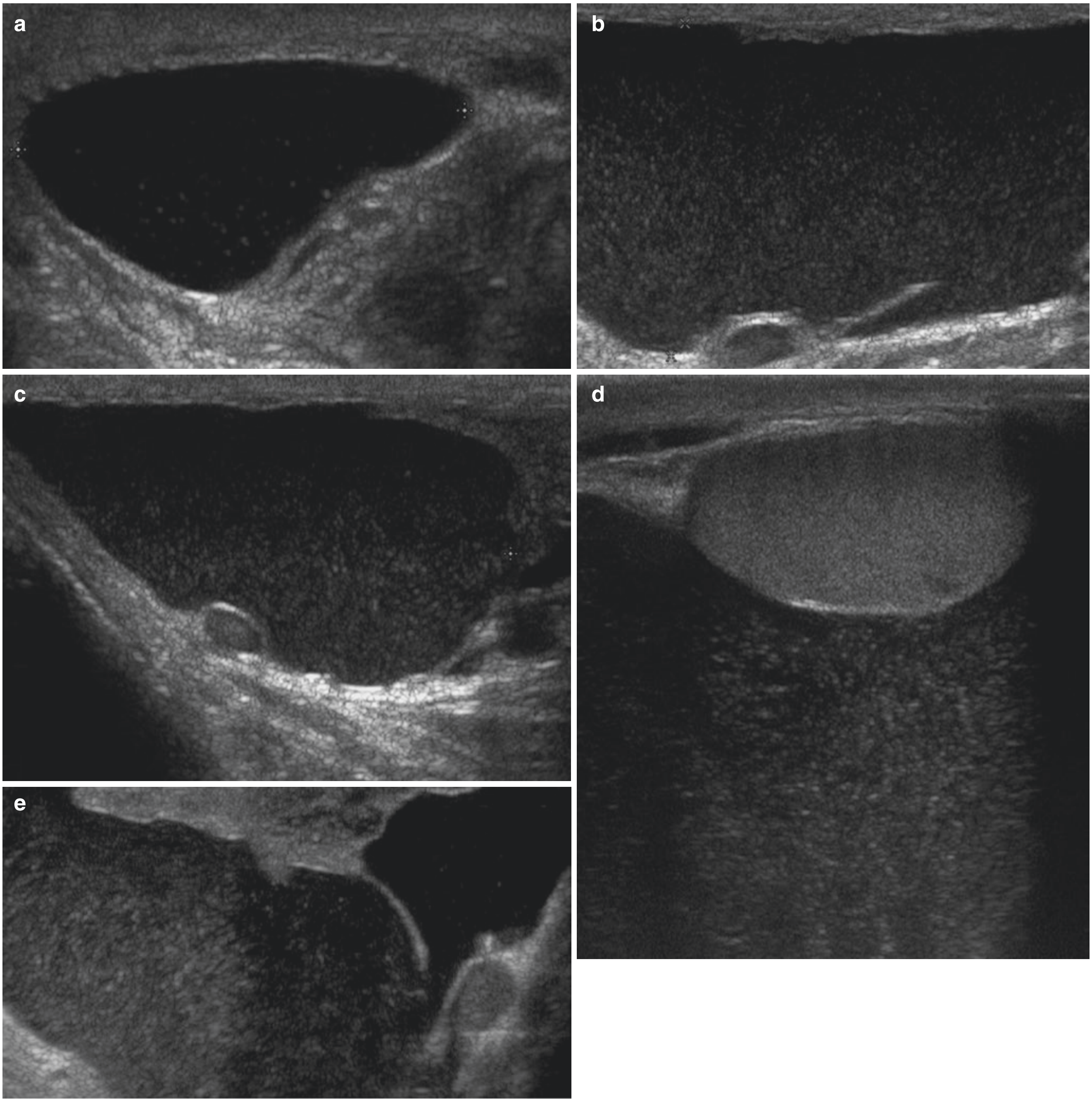

Fig. 4.22 Hydrocele. Hydroceles secondary to epididymo-orchitis often contain echogenic material representing an inflammatory exudate. Panel (**e**) shows two adjacent compartments with the left one filled with echogenic material, while the right side shows a completely anechoic filling

strands crossing the hydrocele fluid. Some chronic hydroceles have multiple septa which are thought by some to be due to chronicity; others believe that they represent a previous infection or inflammation (Fig. 4.23). However, these should not be confused with the testicular ligaments, anatomical structures more easily seen in the presence of hydrocele. Recurrent hydroceles occurring after surgery commonly have multiple coarse septa. Chronic hydroceles may lead to considerable compression of the testis, causing deformity and atrophy. In communicating hydroceles, scanning is performed in both the standing and supine positions, and fluid can be detected along the spermatic cord. Other, less common fluid collections include haematoceles and pyoceles.

Haematoceles are collections of a consistent amount of blood and serum between the visceral and parietal layers of the tunica vaginalis [5]. They often exert a mass effect, distorting the contour of the testis (Fig. 4.24). The most common causes are trauma, torsion, tumour, surgery, diabetes or atherosclerotic disease [12, 31]. These collections are typically complex, with numerous septations, loculations and low- to medium-level internal echoes (Fig. 4.25). In patients with chronic haematoceles, calcification may develop on the tunica vaginalis or internal septations [5]. Haematoceles are frequently seen in patients with varicoceles. Presumably, a traumatic rupture of one of the dilated vessels is a causative factor.

Most haematoceles spontaneously resolve with conservative therapy, although some may become fibrotic and calcified and require surgery if they compress the testis.

Pyoceles (Fig. 4.26) [32, 33] are an uncommon complication resulting from the rupture of an abscess into an existing hydrocele or directly into the space between the layers of the tunica vaginalis. They are generally found subsequent to abscess formation and rupture in epididymo-orchitis. Pyoceles may also result from penetrating scrotal injuries, urethritis, perianal abscess, perianal surgery, vasectomy and scrotal gangrene. On ultrasound, pyoceles and haematoceles show a similar complex pattern, with internal echoes and septation, which can be difficult to differentiate from a chronic hydrocele. Rarely, in patients with anaerobic infections, gas may develop causing bright specular reflectors and shadowing.

Clinical correlation is often needed to differentiate haematocele, pyocele and chronic hydrocele. A distinctive feature of pyocele is the development of scrotal oedema and hyperaemia. Colour Doppler imaging usually reveals increased vascularity in the septa or scrotal wall [5].

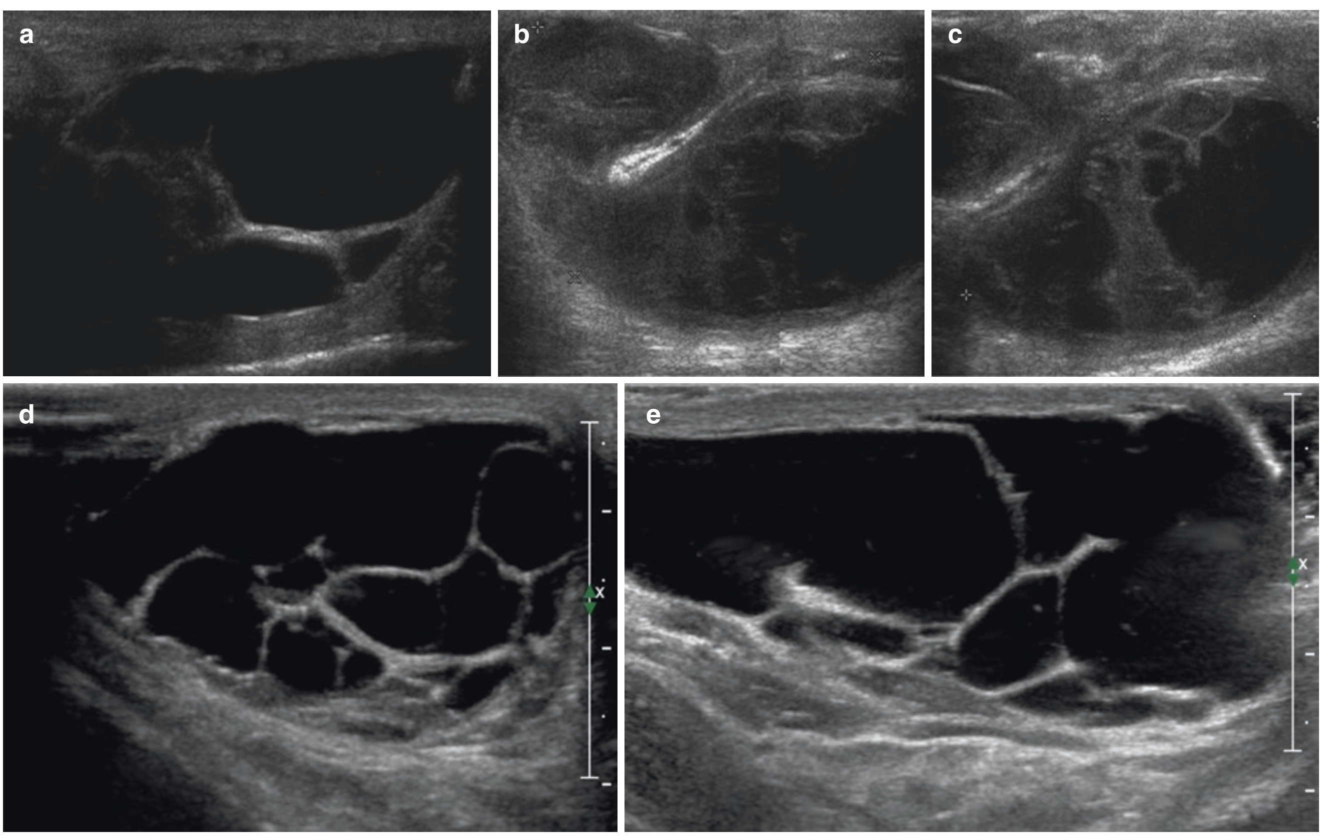

Fig. 4.23 Inflammatory hydrocele. Chronic hydrocele with multiple septa and organised material in the vaginal space

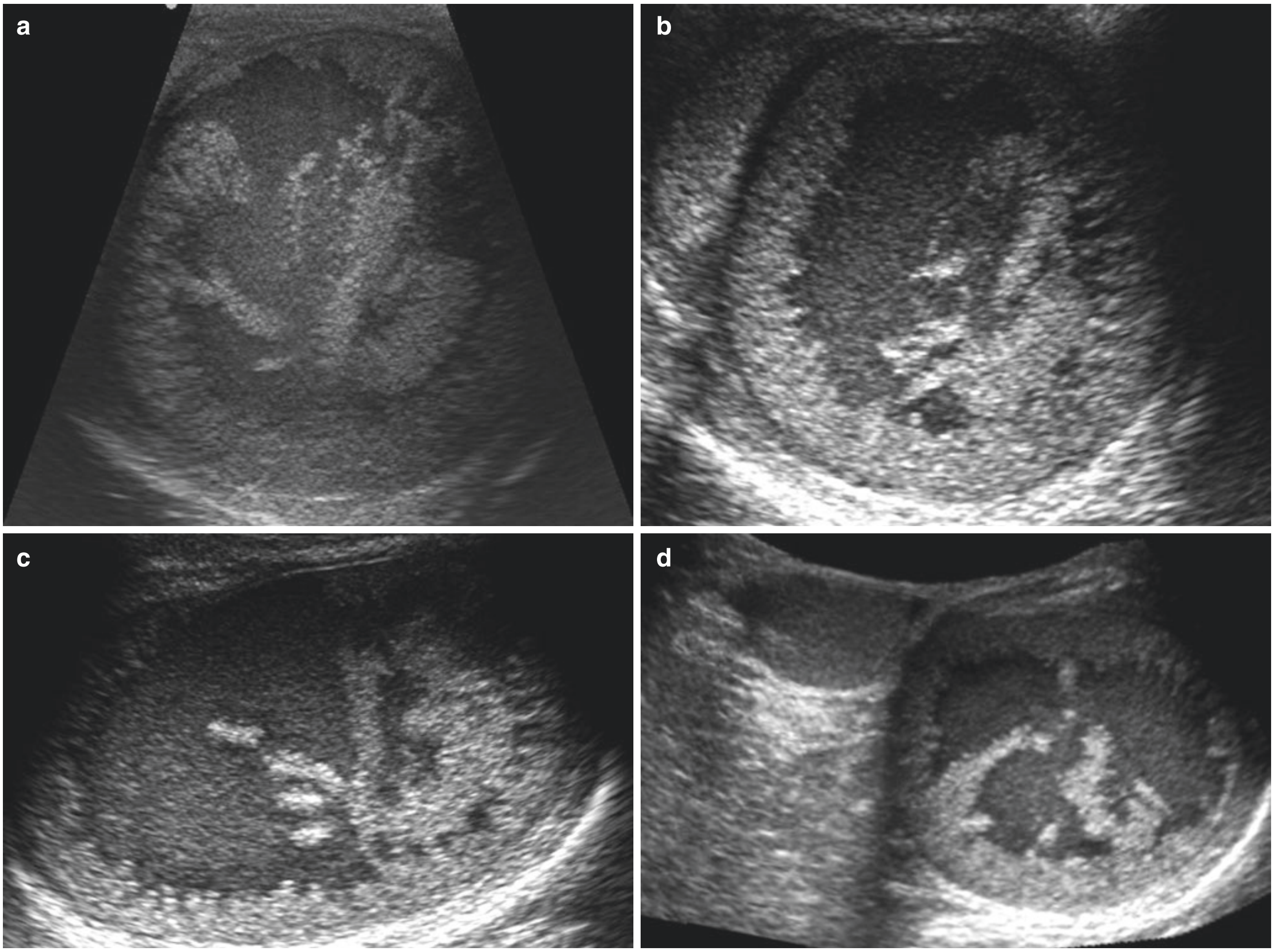

Fig. 4.24 Haematocele. Longitudinal US image of the scrotum revealing a chronic haematocele (*Courtesy of R.H. Oyen, MD*)

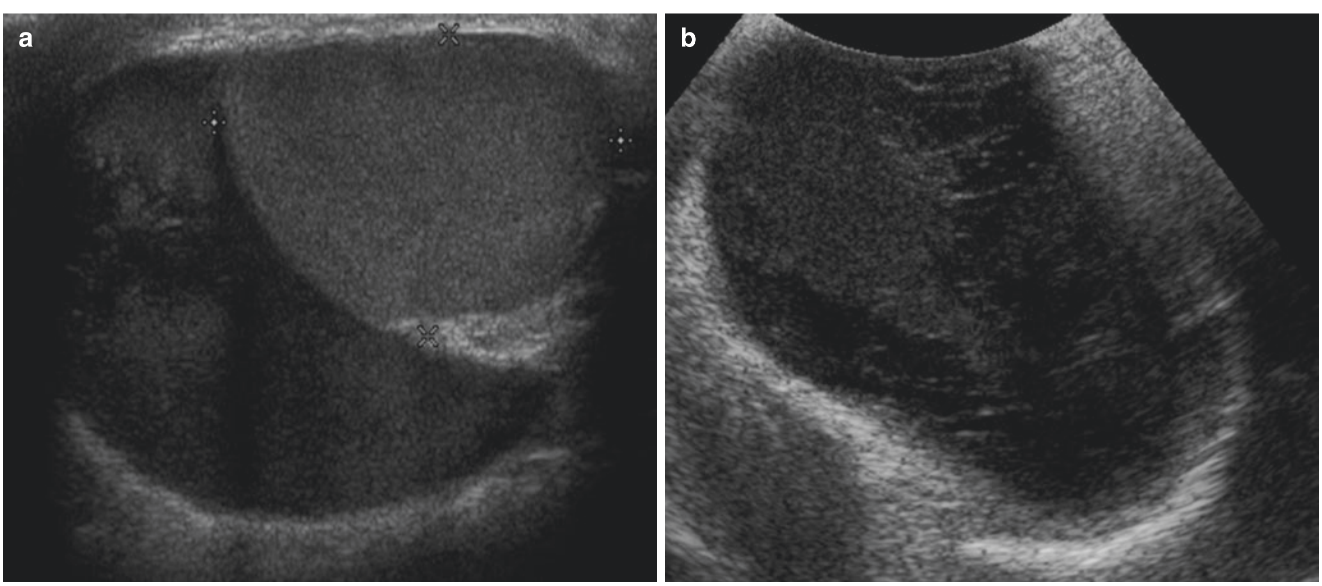

Fig. 4.25 Haematocele. Few hours after a trauma, the blood appears echogenic but is still liquid (**a**). Then haematocele becomes complex and echoic and causes compression of the testis (**b**–**d**)

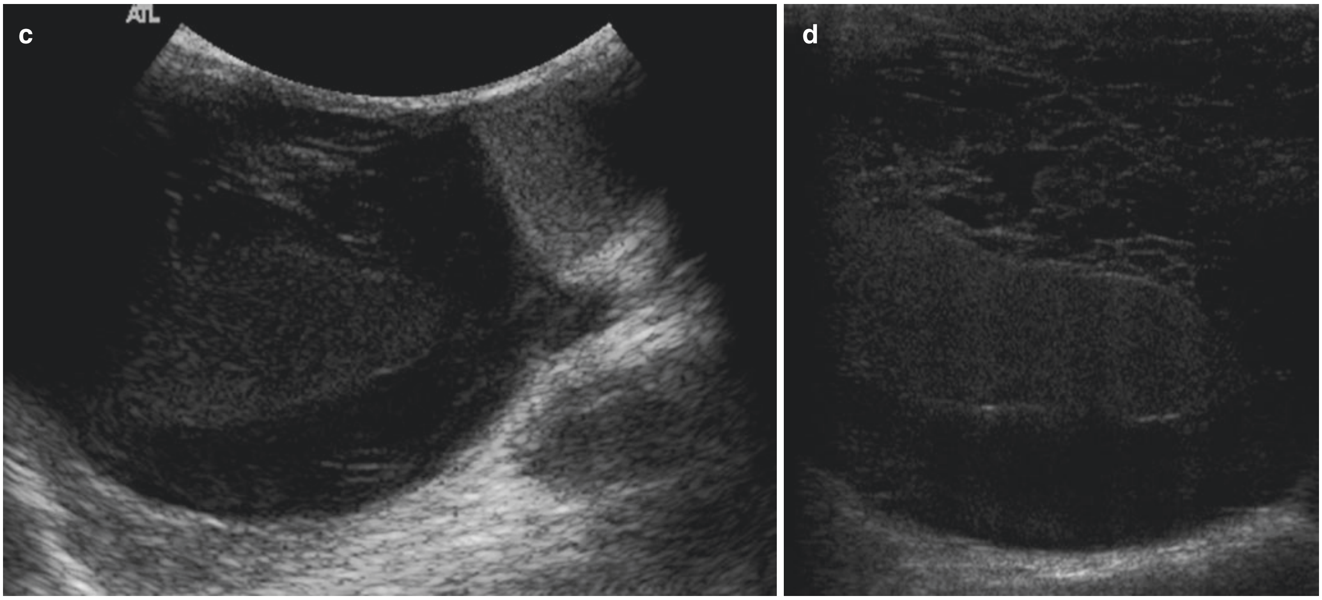

Fig. 4.25 (continued)

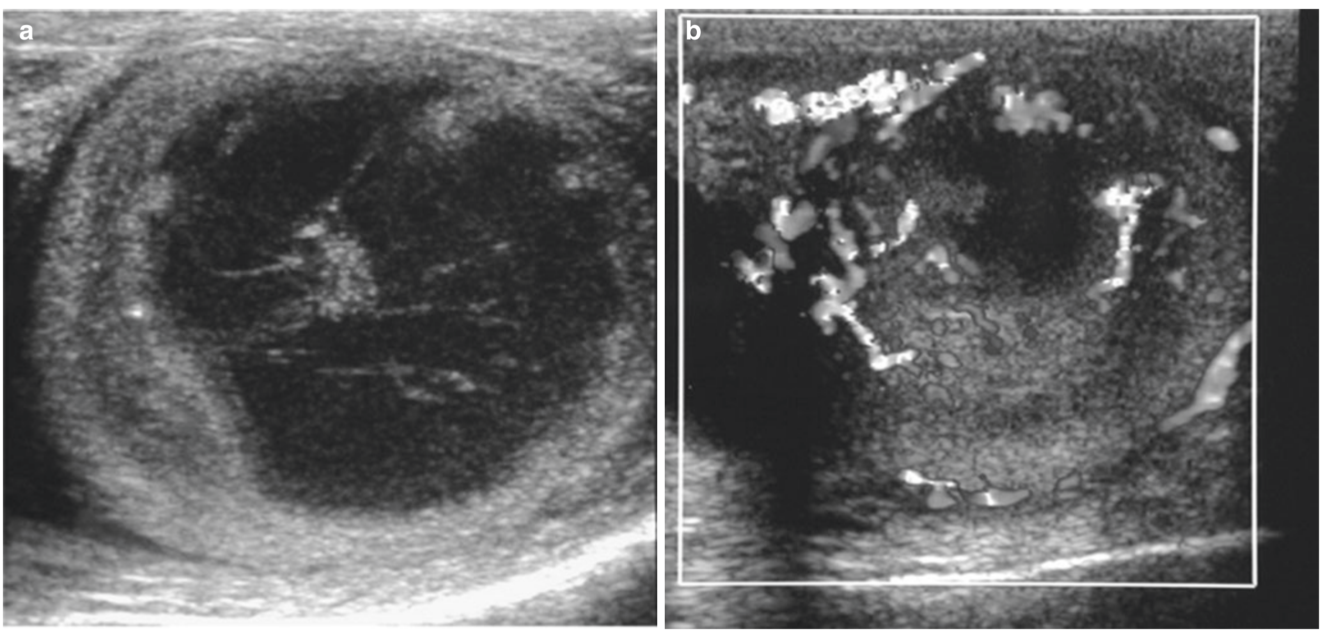

Fig. 4.26 Pyocele. In panel (**a**)**,** transverse scan of the testis shows a heterogeneous right testicular mass. (**b**) Doppler examination of right testis revealing marked vascularity surrounding the mass. In panels (**c**) and (**d**), an epididymo-orchitis with epididymal abscess and pyocele is shown *(panels* ***a–d,*** *From: E.E. Rutherford, K.C. Dewbury, 'Intra-testicular Pseudotumour Mimicking Malignancy: Ultrasound Appearances', Clinical Radiology (2003) 58: 893–895; panels* ***c***, ***d***, *From: S Thinyu, M Muttarak, 'Role of ultrasonography in diagnosis of scrotal disorders: a review of 110 cases', Biomed Imaging Interv J 2009; 5(1):e2)*

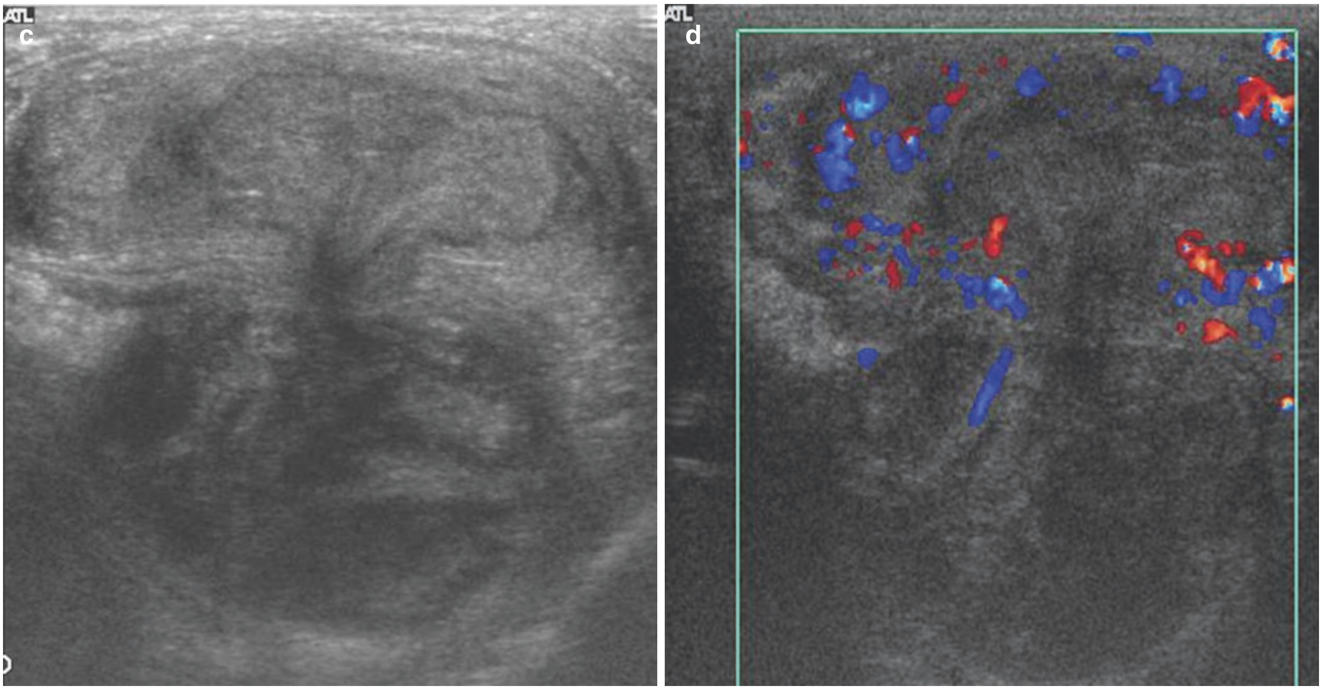

Fig. 4.26 (continued)

4.7 Hernias

Inguinal hernias (Fig. 4.27) are among the most common paratesticular masses. In the vast majority of cases, the diagnosis is obvious on clinical examination, but a minority may be difficult to diagnose and be referred for ultrasound. Hernias occur in 1–4% of babies and with a higher incidence in preterm boys and those with patent processus vaginalis, communicating hydrocele or undescended testis.

Prompt, accurate diagnosis is crucial, as congenital inguinal hernia can complicate with incarceration and strangulation which may result in loss of the gonad (Fig. 4.28) [34].

Ultrasonography can be a useful way to confirm the presence of a scrotal or inguinal hernia or differentiate other aetiologies that may simulate hernia [5]. An inguinal hernia usually presents as a structure of varying shape and echotexture, which moves in the inguinal canal with breathing. The testis and epididymis may be displaced downwards but should retain their normal shape and texture. The colon or small intestine within the inguinal canal appears as a thick-walled, fluid-filled, tubular structure that may show haustra or valvulae conniventes. Real-time display of peristalsis is diagnostic for hernias containing a section of intestine.

Although the clinical diagnosis is usually straightforward, the picture may occasionally be surprisingly difficult to interpret. This is especially true of incarcerated hernias, in which peristalsis is infrequent or absent. Another challenging picture at ultrasound is meconium hernia, which can present with stippled calcifications seen as disseminated bright echoes within a soft mass in the scrotum. A differential diagnosis must always be considered in the presence of complex masses. Diagnosis is facilitated by the continuous display of the lesion in a cranial direction up into the inguinal canal. The use of the Valsalva manoeuvre is critical in demonstrating or defining inguinal hernias, particularly if they are small or contain only omentum. A small protrusion of omental fat should be differentiated from lipoma, lipomatosis or other spermatic cord tissues. An echogenic mass adjacent to the spermatic cord that can be followed into the inguinal canal and demonstrates protrusion towards the scrotum with Valsalva manoeuvre is diagnostic of an omental-type hernia. Lipomas tend to appear as oval defined masses, whereas herniated omentum appears more elongated and should be traceable back to the inguinal canal.

Sonography is also very important in assessing early and late complications of patients who have undergone surgical hernia repair [5, 35]. Clinically, differentiating haematoma from recurrent hernia in the early postoperative period can be difficult. Haematoma appears as a complex, inhomogeneous collection that extends from the inguinal canal, often surrounding the cord and extending into the scrotum [5]. In many cases, the scrotal wall is also thickened, with oedematous changes. Haematomas do not change in appearance with the Valsalva manoeuvre. Evaluation of the ipsilateral testicle with colour or power Doppler imaging is critical to exclude testicular ischaemia or infarction, which can occur secondary to venous congestion from haematoma or after inadvertent ligation of the testicular artery or spermatic vein after laparoscopic herniorrhaphy [5]. Unfortunately, a significant number of testicles are still severely damaged during surgical hernia repair.

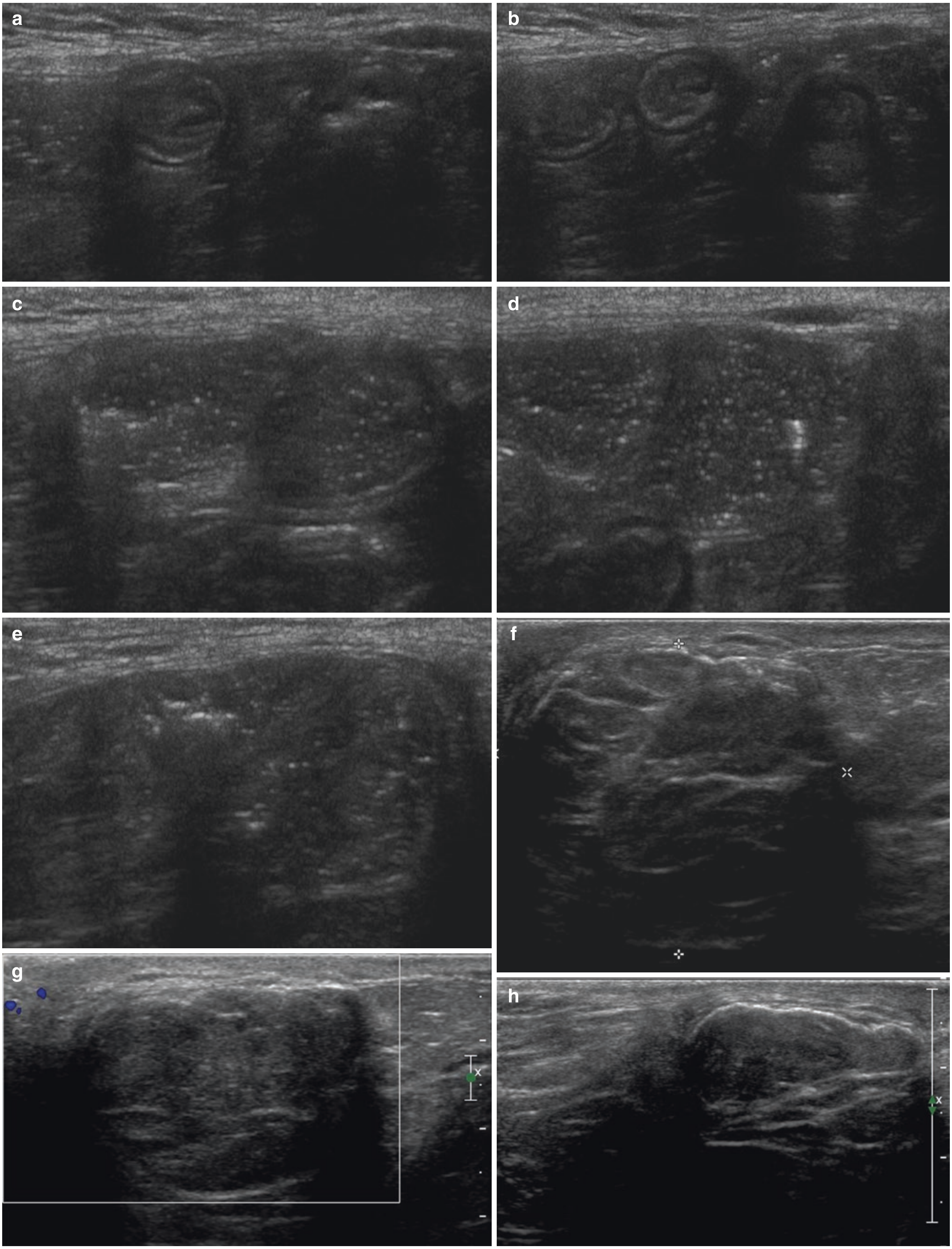

Fig. 4.27 Inguinal hernia. US scan of the inguinal region revealed an inguinal hernia presenting as an inhomogeneous structure of irregular shape which moves in the inguinal canal during Valsalva manoeuvre (**a**–**e**); in panels (**f**–**h**), an inhomogeneous structure is shown, mobile in the inguinal canal in orthostatism, compatible with reducible inguinal hernia

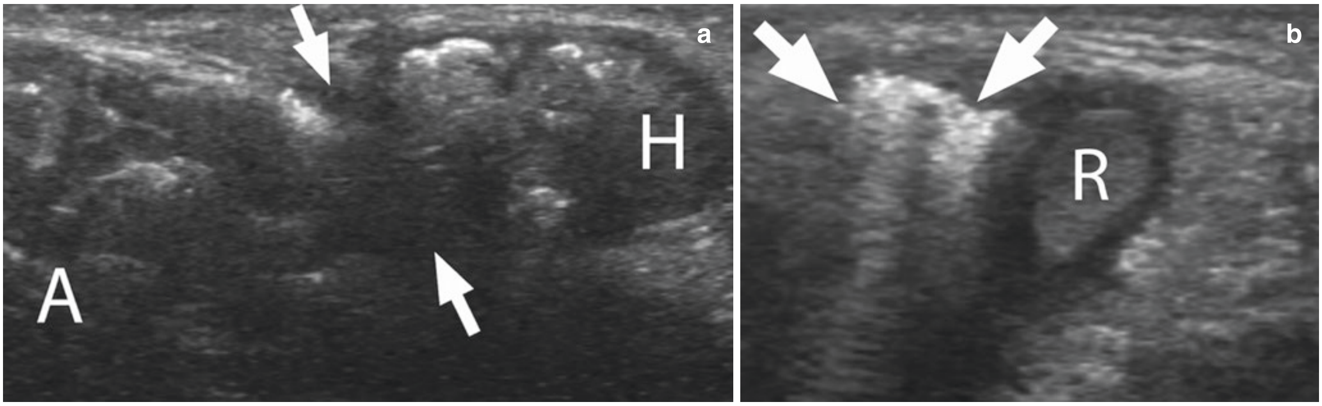

Fig. 4.28 Inguinoscrotal hernia. (**a**) Longitudinal greyscale sonogram of the proximal right inguinal canal in a 2-week-old boy with abdominal distention, dysmorphic features and a non-palpable right testicle. The image shows a right inguinal hernia (H) that contains bowel extending from the abdominal cavity (A) through the proximal inguinal ring (*white arrows*) into the inguinal canal. (**b**) Transverse greyscale sonogram of the inguinal canal in the same patient showing echogenic air (*white arrows*) within bowel loops *(From: A.M. Basta, J. Courtier, A. Phelps, et al. 'Scrotal Swelling in the Neonate' J Ultrasound Med (2015) 34:495–505)*

Key Messages

- Epididymal cysts are round or oval anechoic lesions with posterior acoustic enhancement. They can be simple or multiseptated. Their walls do not contain solid parts. Their aetiology remains unclear and there is no evidence that they cause pain.
- Cysts should be differentiated from spermatoceles originating from the mechanical obstruction of efferent ductules of the rete testis.
- On palpation, spermatoceles can be identified as a freely movable, transilluminating soft mass (more taut than hydrocele, but not as firm as epididymal cysts) that is separate from and above the testicle.
- Epididymitis is the most common cause of acute or chronic pain and is therefore the most frequent reason for consultation. The US features consist of localised or diffuse enlargement of the epididymis, which appears heterogeneous, often hypoechoic with scattered foci and associated with mild to severe hyperaemia.
- Acute scrotum is often caused by what is generally defined as epididymo-orchitis. However, in the large majority of cases, only the epididymis is involved, and although hyperaemia can be seen in the adjacent testicular parenchyma, the infection does not involve the testes. A notable exception is mumps orchitis.
- Acute epididymitis is a common complication of lower urinary tract infections (urethritis, prostatitis, cystitis) that reaches the epididymis retrogradely, via the vas deferens.
- Epididymitis is more likely in patients over 30, whereas torsion is the prime diagnostic hypothesis in prepubertal boys, in whom epididymitis is rare. Relief of pain on elevation of the scrotum over the symphysis (Prehn's sign) is suggestive of epididymitis, as this manoeuvre exacerbates the pain of spermatic cord torsion.
- Laboratory investigations demonstrating leukocytosis, bacteriuria, leukocyturia or associated urethritis can facilitate the diagnosis of acute or chronic epididymitis.
- A sequela of epididymitis is chronic leukospermia. There is growing awareness that this persistent sterile leukospermia, causing an increased production of ROS (reactive oxygen species), is involved in a consistent number of cases of male idiopathic hypofertility.
- Chronic epididymitis may cause a fibrotic thickening of the tunica albuginea that is sometimes so severe that it is called pseudotumour of the albuginea.
- Paratesticular tumours are not as infrequent as generally thought; they are primarily seen in older patients, and the vast majority are benign. Adenomatoid tumour of the epididymis is the most frequent, presenting as a smooth, round and well-circumscribed isoechoic or hyperechoic homogeneous mass.

- Papillary cystadenomas deserve a special mention due to their association with von Hippel-Lindau (VHL) disease [2], especially when bilateral (40%).
- Isolated epididymal malignancies are exceedingly rare. They present as large, mainly solid, 'craggy' infiltrating lesions that give a strong suspicion of malignancy, in contrast with the cystic or small, smooth appearance of solid benign lesions.
- The tunica vaginalis is lined by mesothelial cells, which in rare cases may give rise to mesotheliomas. Fifteen to twenty percent of these are benign; malignant mesotheliomas are highly aggressive cancers.
- Inguinal hernias are among the most common paratesticular masses.
- Fluid collections are frequent. Large hydrocele is more typically suggestive of an idiopathic hydrocele or an inflammatory cause. The presence of blood cells in the fluid suggests trauma, infection or malignant paratesticular tumours.

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5 Acute Scrotum

5.1 Introduction

The great majority of cases of acute scrotum are due to one of three causes: trauma, torsion or epididymo-orchitis. Differential diagnosis also includes a few rare conditions: strangulated hernia, testicular tumour, haematocele and idiopathic scrotal oedema, Henoch-Schönlein purpura and scrotal fat necrosis. In younger subjects, testicular or appendiceal torsions account for over half of cases of acute scrotum, with an additional third due to epididymo-orchitis. The latter is the most frequent cause in adults, followed by trauma. Acute scrotum syndrome, whether of traumatic, ischaemic or inflammatory origin, always merits immediate evaluation as early intervention is very likely to preserve testicular function, whereas a delay will almost invariably lead to chronic irreversible complications.

Clinical symptoms and physical examination are often not sufficient for definite diagnosis, as pain and swelling can limit careful palpation of the scrotal contents [1]. High-resolution ultrasonography combined with colour Doppler ultrasonography facilitates the simultaneous assessment of anatomical information and perfusion changes in many scrotal abnormalities, so these are the imaging techniques of choice in diagnosing the causes of acute scrotal pain [1, 2, 3].

5.2 Testicular Torsion

Testicular torsion is the rotation of the testis along its longitudinal axis. This results in torsion of the spermatic cord with an initial blockage of venous drainage and a subsequent reduction in arterial supply to the testis if complete rotation (>360°) persists. Venous blockage causes oedema and haemorrhage, followed by ischaemia and necrosis when arterial inflow is significantly reduced. The rapid drop in blood flow is due to the fact that the testis is supplied by three arterial vessels that divide in the spermatic cord (testicular, deferential and cremasteric arteries), and there is no significant collateral circulation with the scrotal vascularisation.

Torsion is more common in pubertal boys than in adults. A peak incidence of testicular torsion is also seen in the neonatal period, while it is responsible of less than 20% of cases of acute scrotal syndromes in post-pubertal males [4].

In the clinical setting, symptoms can mimic other non-surgical aetiologies such as epididymo-orchitis and testicular tumours. If the patient presents within an early time window, ultrasound examination is critical in establishing whether the patient needs surgical repair [5]. As testicular torsion causes severe testicular ischaemia (Fig. 5.1), prompt diagnosis and immediate surgery are necessary to preserve the testis. However, a false-negative US evaluation can delay the procedure and place the salvage of the testis at risk. For this reason, and given that US features can vary with time, we strongly recommend that US is performed at least twice (approximately 2 h apart) in patients where a first examination seems to exclude torsion. The testicular salvage rate is 80–100% if surgery is performed within 5–6 h of pain onset (early phase), 70% if surgery is performed within 6–12 h (mid phase) and only 20% if surgery is delayed for more than 12 h [6].

The differential diagnosis of testicular torsion relies on simultaneous assessment of clinical, US and **laboratory findings**. Body temperature, urinary sediment, leucocyte number and markers of inflammation are usually normal in the torsion and are important criteria for its distinction from epididymo-orchitis. **Clinical examination** is often difficult and non-specific, as the scrotum may appear swollen and reddened. Among the few signs that can be searched for are *Ger's sign* (pitting of the skin at the base of the scrotum) and *Brunzel's sign* (the testis lies higher in the scrotum and is horizontal with the patient standing), suggestive of testicular torsion, while *Prehn's sign* is negative (the elevation of the testis fails to relieve pain, as it normally does in inflammatory conditions).

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The presence/absence of the cremasteric reflex can also help in the differential diagnosis. This reflex is elicited by stroking or gently pinching the skin of the upper inner thigh while observing the scrotum. A normal response is contraction of the cremasteric muscles on the ipsilateral side with unilateral elevation of the testis. The cremasteric reflex is rarely intact in patients with testicular torsion but is usually present in patients with torsion of an appendix testis (Table 5.1).

Epidemiological criteria can also help, as epididymitis is uncommon in prepubertal boys whereas torsions are less likely over the age of 30, when epididymitis becomes more common. Nevertheless torsion can occur at any age and the differential diagnosis includes strangulated hernia, haematocele and testicular tumour.

Colour and power Doppler studies are the primary method for diagnosing testicular torsion, with 86%

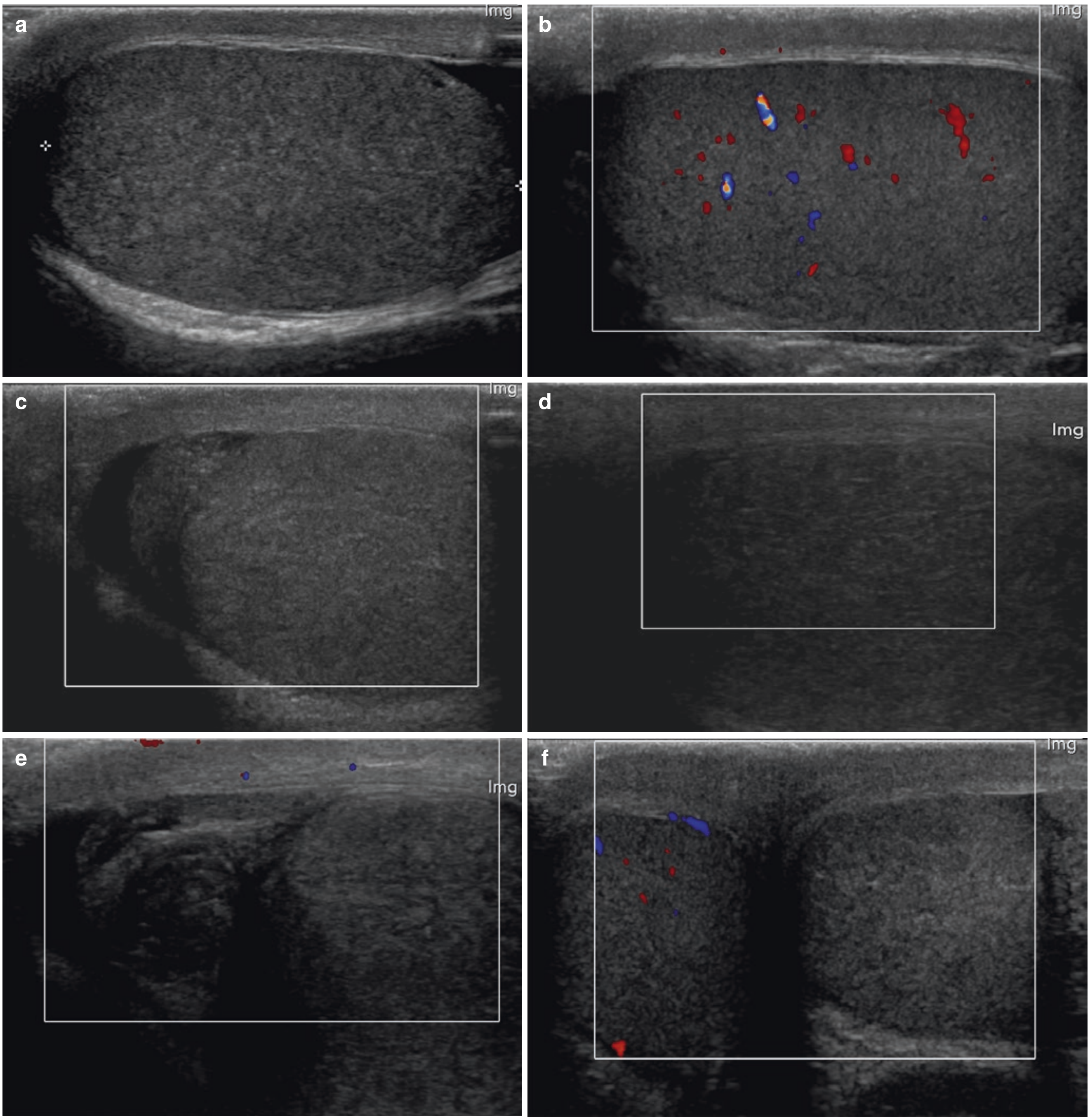


Fig. 5.1 Missed torsion. 18-year-old boy who presented with left scrotal pain for 5 h. Longitudinal US scan of the testes shows an enlarged, unevenly hypoechoic left testis (**a**) compared to the normal right testis (**b**). Colour Doppler images show normal vascular flow in the right testis and absent vascular flow in the left testis (**c–f**). Longitudinal image of the testis 2 (**g**) and 6 months (**h**) after the episode of testicular torsion, surgically treated within 7 hours from the onset of symptoms. The testis revealed changes in the echotexture, which appear inhomogeneous, with an hypoechoic, irregular area in the mid-lower pole of the testis, secondary to ischaemic damage

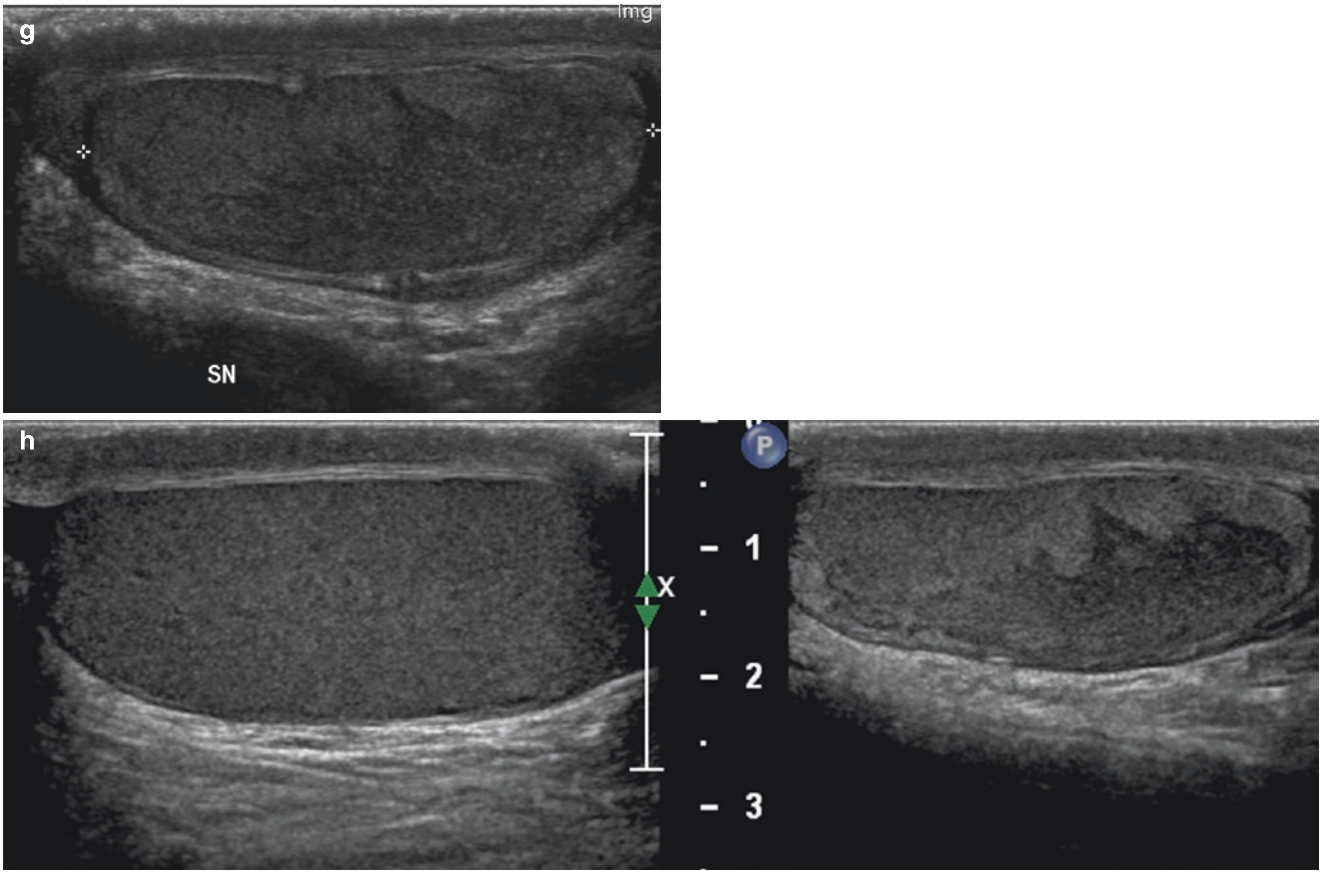


Fig. 5.1 (continued)

Table 5.1 Clinical presentation of the most frequent causes of acute scrotum

Condition	Onset of symptoms	Age	Tenderness	Urine/blood analysis	Cremasteric reflex	Positive sign
Testicular torsion	Acute	Puberty (and perinatal)	Diffuse	Negative	Negative	Brunzel's Ger's
Appendiceal torsion	Subacute	Prepubertal	Localised to upper pole	Negative	Positive	Blue dot
Epididymitis	Subacute (insidious)	Adolescence adulthood (sexual activity) Old age (urinary infections)	Epididymal (and diffuse when extended to the testis)	Positive (occasionally negative)	Positive	Prehn's

sensitivity and 100% specificity [5, 7]. Testicular torsions can be divided by pathophysiology into **intravaginal** and **extravaginal**. The former are more common, especially in peri-pubertal boys, and result from a combination of several factors including abnormal development of the tunica vaginalis. Three factors are likely to contribute to intravaginal torsion: an anomalous suspension of the testis by a long stalk of spermatic cord, the complete cover of the testis and epididymis by the tunica vaginalis and a thin or loose fixation of the posterior aspect of the testis to scrotal wall. The latter is also known as the 'bell-clapper deformity', which has 12% prevalence and is bilateral in 40–80% of cases. The testis, covered by the vaginalis, is in fact free to fall forward and rotate in the tunica vaginalis, much like a clapper inside a bell. When this condition is suspected, a bilateral orchidopexy is usually recommended to prevent testicular torsion.

Predisposing conditions to torsions include undescended testicles, retractile testis and the presence of additional factors such as abrupt rotational movement of the body, exertion, abnormal cremasteric attachment and blunt trauma.

Extravaginal torsion is a rare condition almost invariably seen in newborns (most cases are thought to occur in utero). It is thought to be secondary to a loose attachment of the testis and spermatic cord to the scrotum, allowing rotation of the testis, epididymis and tunica vaginalis as a unit and causing torsion of the cord at the level of the external ring.

US appearances of testicular torsion are variable (Fig. 5.2), depending on its duration and whether presenting in a pre- or post-pubertal testis. In extravaginal torsion in

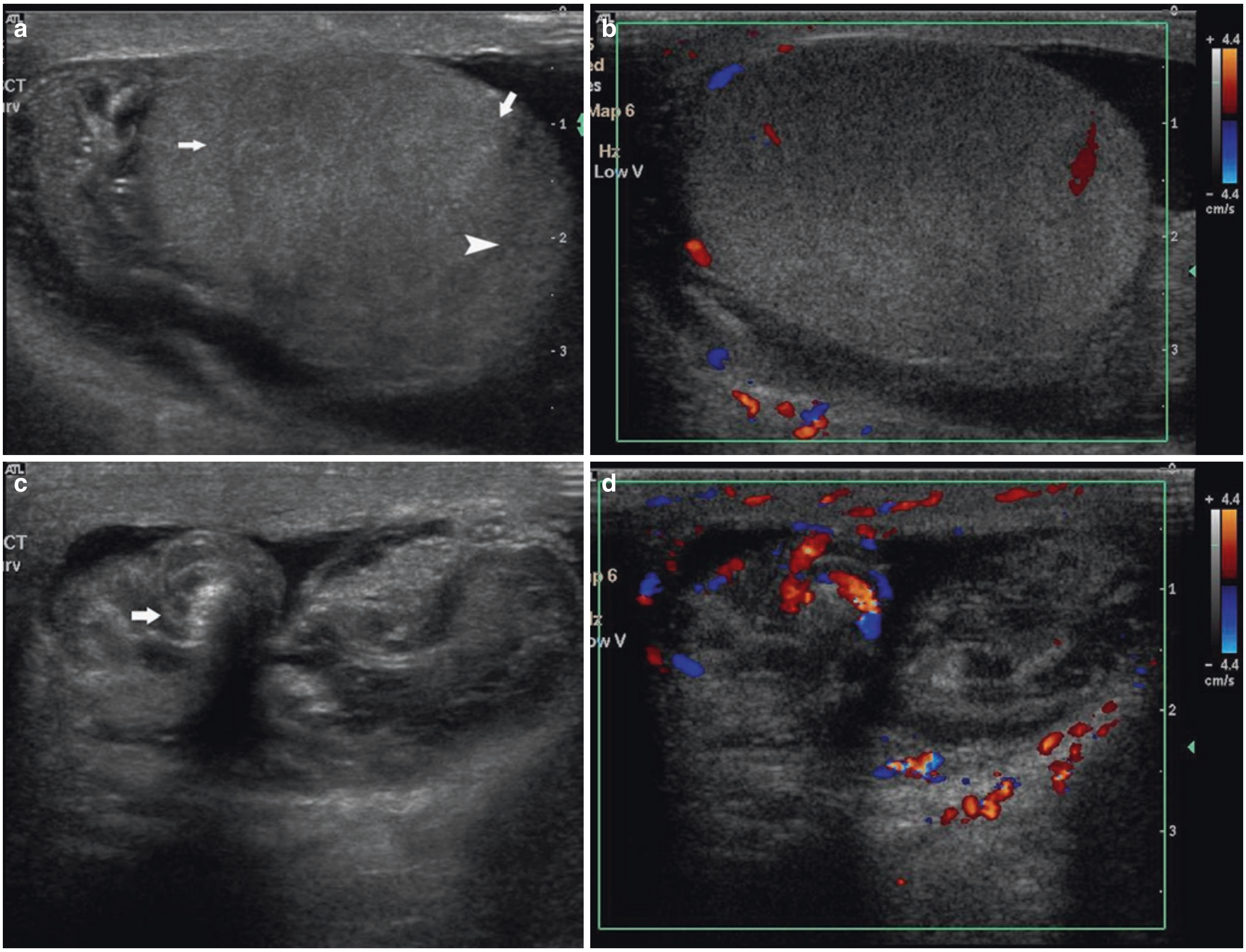


Fig. 5.2 Incomplete torsion of the testis. Longitudinal scan of a horizontally placed testis showing a large hypoechoic area (*arrow*) in the upper two thirds and a normal echo pattern (*arrowhead*) in the lower third (**a**). Colour Doppler image showing no flow in the hypoechoic area with a few vessels seen in the poles (**b**). Greyscale image of the mass of whirlpool with target appearance (**c**). Colour Doppler image of the whirlpool mass showing the vessels going around the central axis (**d**) *(Courtesy of: S. Boopathy Vijayaraghavan, MD)*

newborns, the affected testis appears rounded and enlarged with heterogeneous parenchymal echoes and peripheral echogenic rims. In the first few hours of intravaginal torsion **(early phase)**, the testis often appears normal. After 4 h, the testis is enlarged with diffuse hypoechogenicity (Fig. 5.3) [8]. As time passes, a patchy, heterogeneous testis is seen due to focal haemorrhage and necrosis [1]. The decreased echogenicity cannot be easily detected in children because of the typical low echotexture of the normal prepubertal testis. In contrast, torsion in a prepubertal testis often presents with a diffuse increased echogenicity.

In the **late phase** of torsion, the number of hyperechoic areas increases, a sign of intraparenchymal bleeding. The epididymis also becomes enlarged and heterogeneous with hyperechoic areas due to haemorrhage, although the timescale for this is variable. The fact that epididymal involvement usually follows testicular swelling is of further help in differential diagnosis from epididymo-orchitis, in which the opposite time sequence is generally seen.

In **chronic torsion** (Fig. 5.4), the testis appears small, hard and markedly hypoechoic to anechoic, and colour flow Doppler shows no flow in the testis and increased flow in the paratesticular tissues, including the epididymis-cord complex and dartos fascia.

Reactive hydrocele and thickening of scrotal skin are also seen. The presence of hydrocele may falsely increase the reflectivity of testis because of the automatic depth adjustment of the signal. Hydrocele is typically also seen in epididymo-orchitis, but the colour Doppler US appearance of the two conditions is relatively different: in chronic torsion, flow within the affected testis is invariably reduced or absent, whereas flow within an inflamed testis is increased.

Increased blood flow in the peritesticular tissues can be seen in torsion [9, 1, 2, 3].

Scrotal hyperaemia, also known as the 'rim sign', is the cause of one of the major pitfalls of Doppler ultrasound. The collateral blood supply to paratesticular tissues, which is reactively increased, should not be erroneously interpreted as

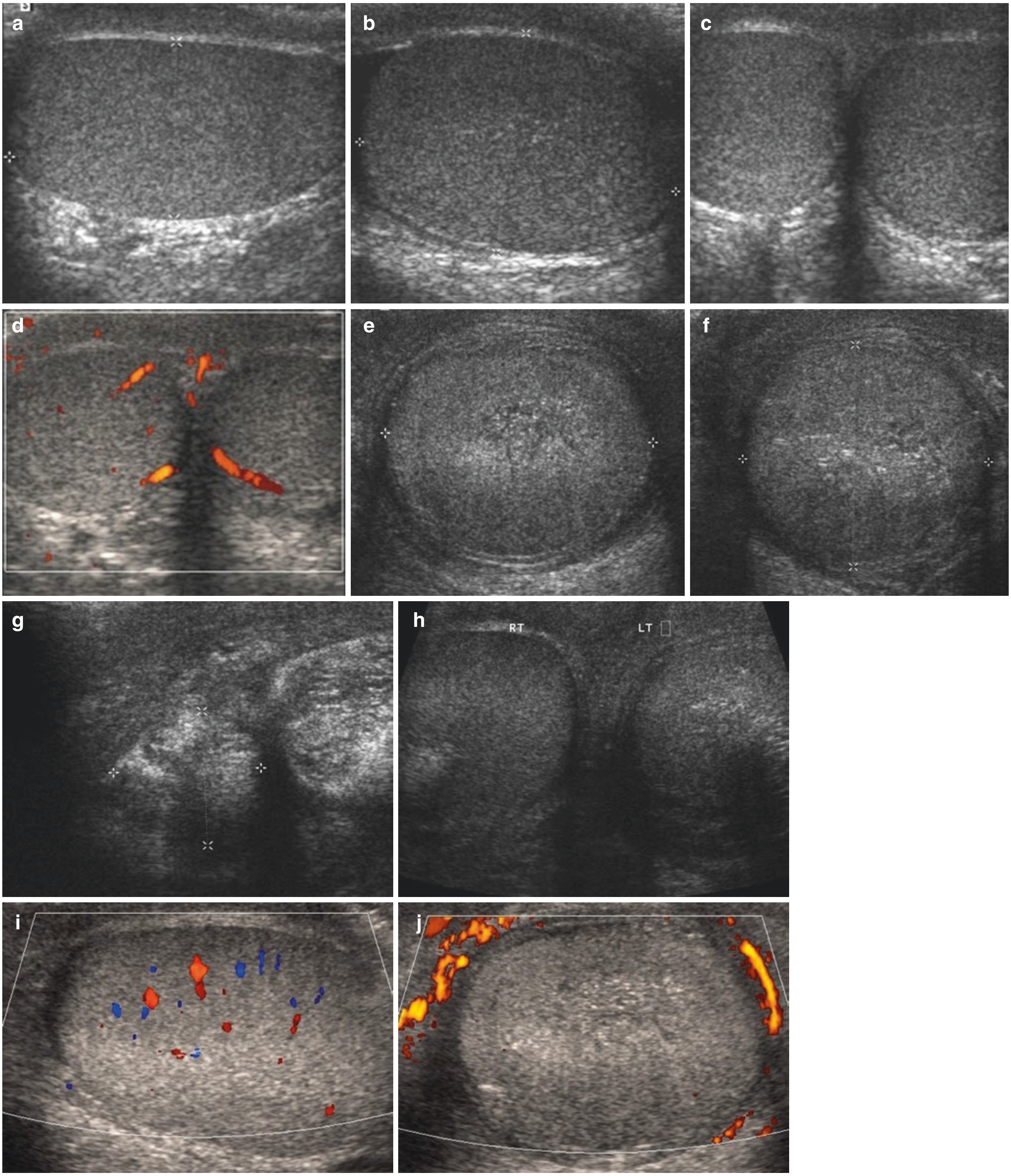


Fig. 5.3 Missed testicular torsion. Missed diagnosis of (incomplete) torsion. (**a**, **b**) Sagittal images of the right and left testis, and (**c**) transverse image of both testes show normal-appearing right and left testes. (**d**) Transverse colour Doppler images of both testes show flow to both testes. The same patient examined 2 weeks later. (**e**, **f**) Sagittal and transverse images of the left testis and (**g**) sagittal image of the left epididymis (**h**). Transverse image of both testes shows a normal right testis and an abnormal heterogeneous left testis with thickening of the skin over the left scrotal sac. (**i**, **j**) Colour Doppler images show normal flow to the right testis but no flow to the left testis *(From: J.R. Mernagh, C.Caco, and J.De Maria. "Testicular Torsion Revisited". Curr Probl Diagn Radiol (2004);33:60–73)*

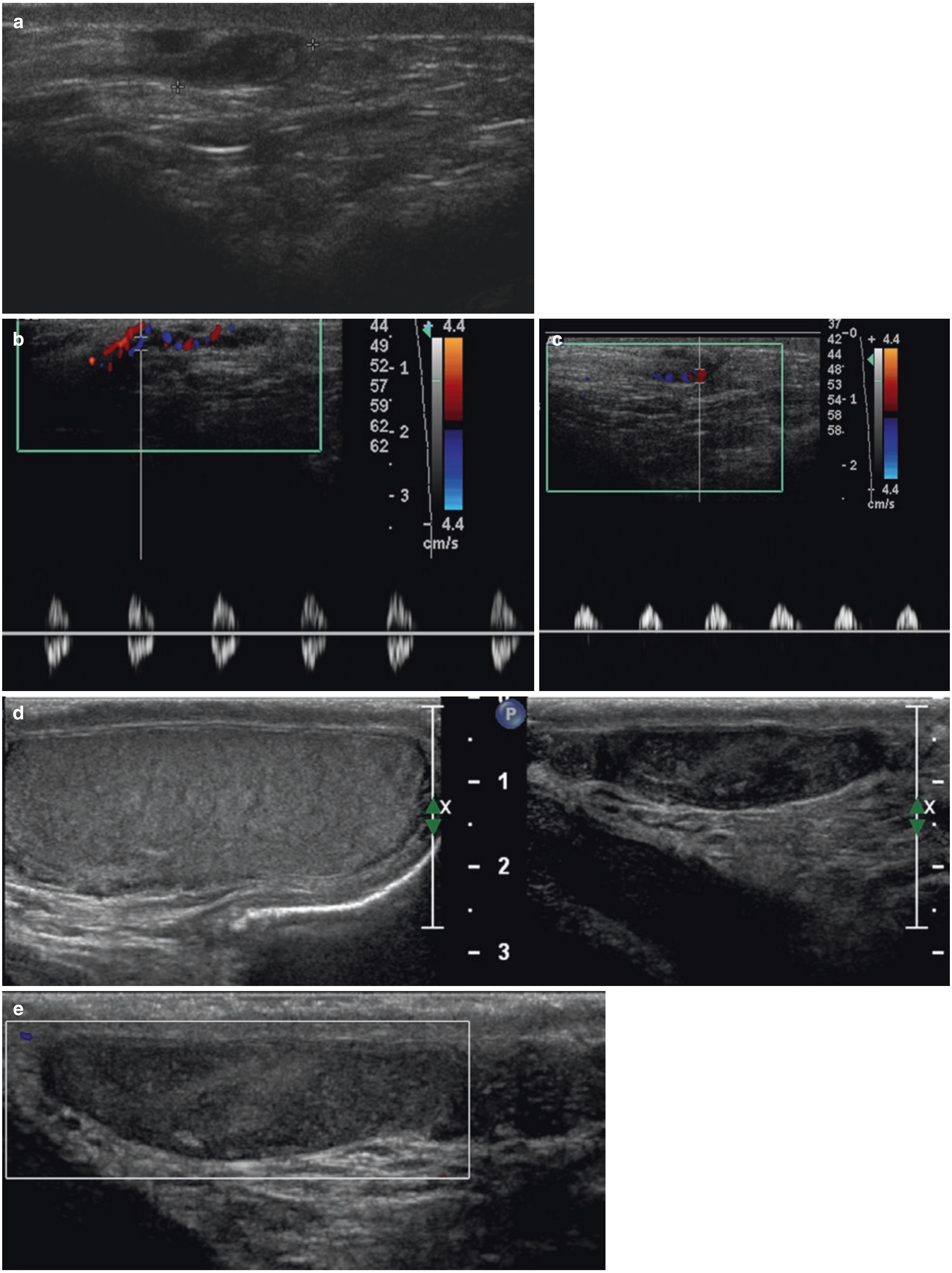


Fig. 5.4 **Atrophy from previous testicular torsion** in a 13-year-old-boy (**a–c**) and in a 45-year-old man (**d–g**). In both cases, the testis is small and inhomogeneous, and the intratesticular flow is disorganised or absent

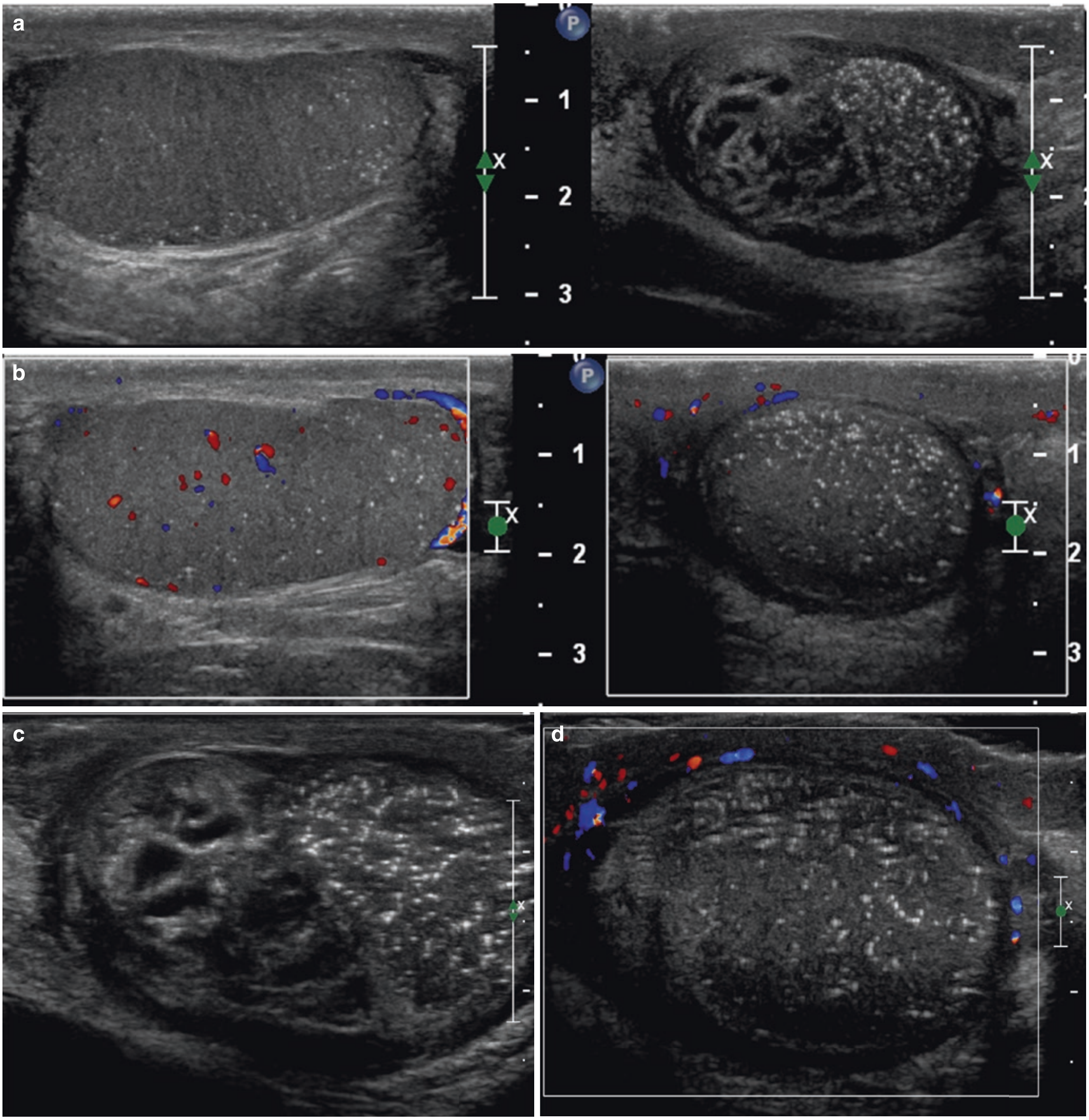


Fig. 5.5 Missed torsion. Missed diagnosis of silent torsion. (**a**) Longitudinal images of the right and left testes and (**b**) longitudinal colour Doppler images of both testes show absence of flow to the left testis. (**d**, **e**) Transverse images of the left testis show an abnormal heterogeneous left testis with microlithiasis, thickening of the skin over the left scrotal sac and no flow

blood flow in the testicular artery. Experimental studies have proven that arterial inflow can still be detected if the torsion is less than 360°, while venous drainage is arrested. To avoid misinterpretation of peritesticular flows, evaluation of parenchymal flow is of great importance. For this reason, **colour-coded duplex ultrasonography** is considered the technique of choice in suspected cases of testicular torsion. It should be noted, however, that this evaluation requires an excellent high-frequency transducer, a machine with adequate power to detect low flow velocities, the patient's cooperation and sufficient experience to judge the normal/abnormal appearance of testicular parenchymal vascularisation (Fig. 5.5).

It must be emphasised that the presence of blood flow in the testis does not exclude the possibility of torsion. Organ perfusion can be initially present, although significantly diminished, gradually disappearing as the oedema increases. In addition colour duplex US provides a 'qualitative', not 'quantitative', assessment, and therefore any reduced vascularisation is considered as comparative to 'what is normally seen' with the particular ultrasound system and settings used.

Once again, the contralateral testis can be used as the control for the original size, echotexture and vascularisation of the testicular parenchyma [1]. However, as previous genital disorders may have altered the appearance of the testes, it is crucial to collect basic information on the patient's medical history even in the acute setting.

With respect to the value of **spectral Doppler** analysis, any pattern may be observed, depending on the stage of the torsion. In general a reversed diastolic flow, caused by oedema and venous compression against the albuginea, suggests ischaemia.

Extratesticular US findings do occur in torsion and should be recognised. The spermatic cord immediately above the testis and epididymis is twisted, causing a characteristic torsion knot or whirlpool pattern (Figs. 5.6 and 5.7) of concentric layers seen on US and MRI. Occasionally, the epididymis cannot be separated from the torsion knot of the spermatic cord.

A clinically important feature of torsion is that it may change the position of the long axis of the testis (Brunzel's

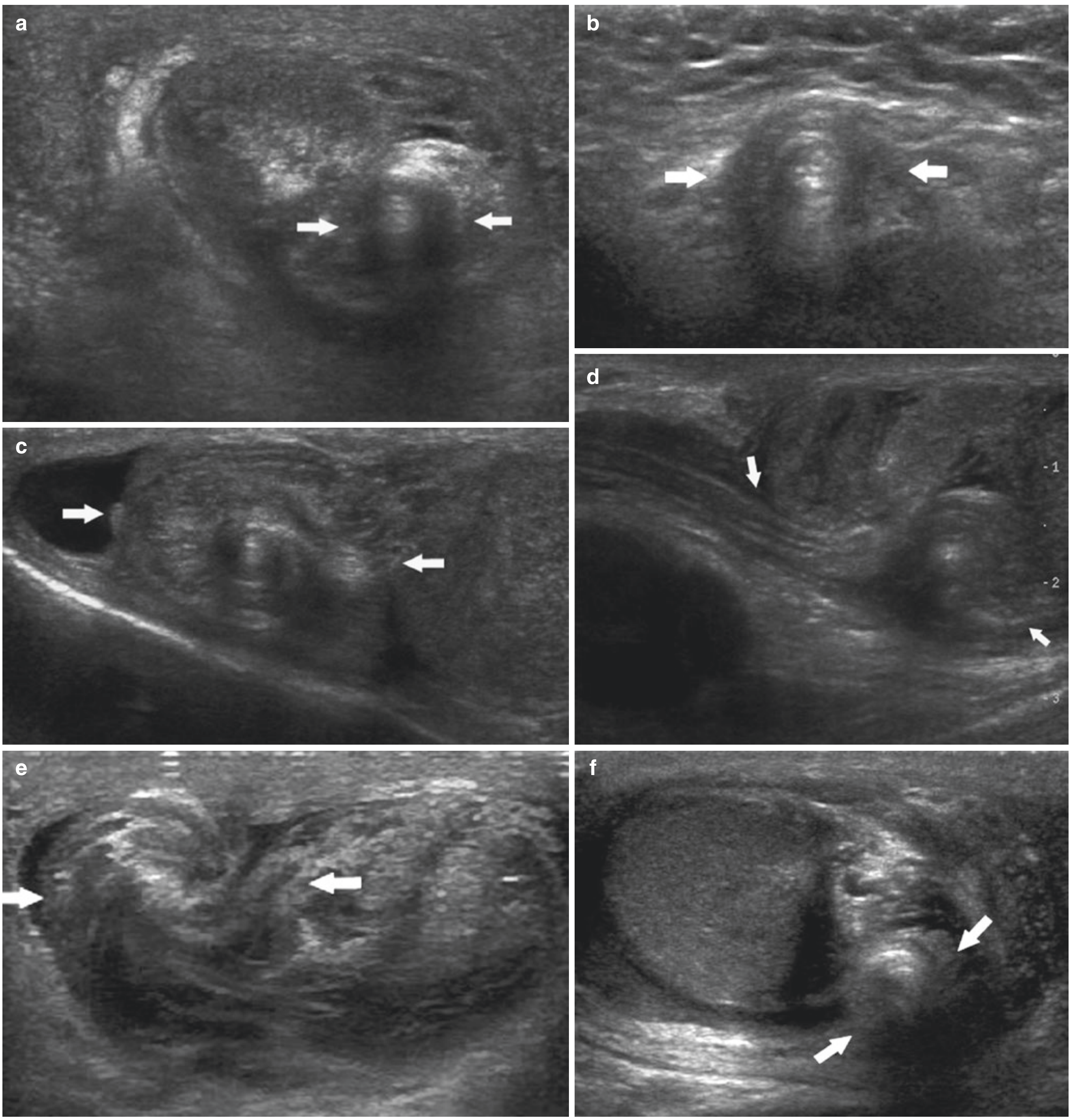


Fig. 5.6 The presence of the whirlpool sign on US is the most specific sign of both complete and incomplete testicular torsions. Various appearances of a **whirlpool mass** (*between arrows*) on static images *(Courtesy of: S. Boopathy Vijayaraghavan, MD)*

sign). This should be interpreted as a sign of predisposition to torsion in boys with **intermittent, recurrent, subacute** scrotal pain of no proven aetiology.

Incomplete or partial torsion (Fig. 5.8) can demonstrate normal flow and is therefore a diagnostic dilemma. Partial torsion is very difficult to discriminate from orchitis, which can also cause partial ischaemia of the testicular parenchyma, thus mimicking torsion (Fig. 5.9) [8]. In addition, physicians should be aware that spontaneous detorsion can occur. This is characterised by increased reactive perfu-

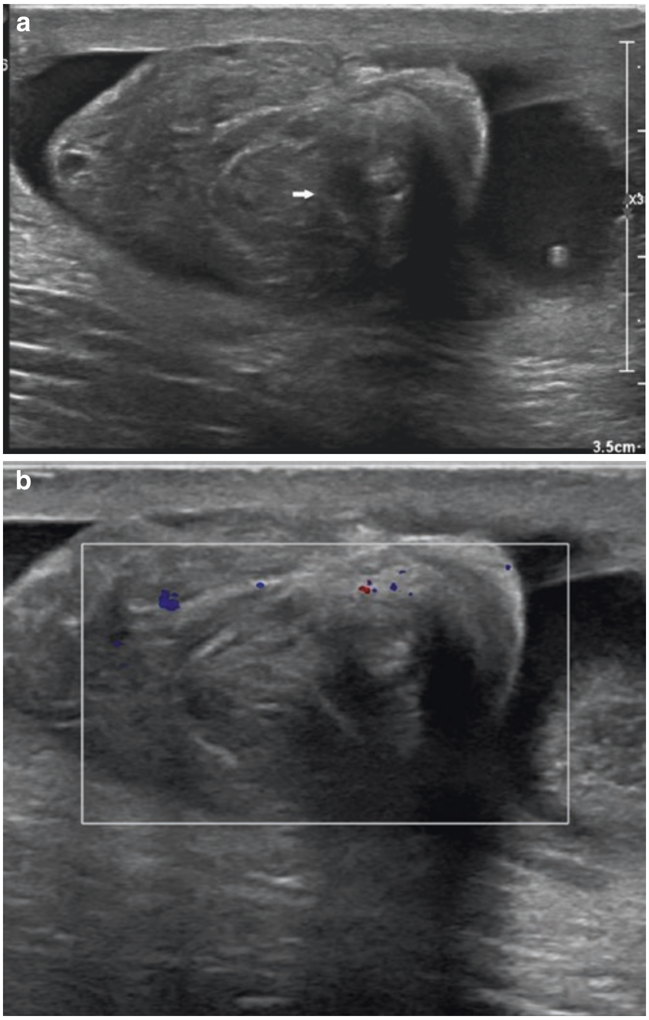


Fig. 5.7 Whirlpool mass. Greyscale (**a**) and colour Doppler (**b**) images of the mass of whirlpool in a patient with complete torsion showing absence of flow in the compressed vessels (*Courtesy of: S. Boopathy Vijayaraghavan, MD)*

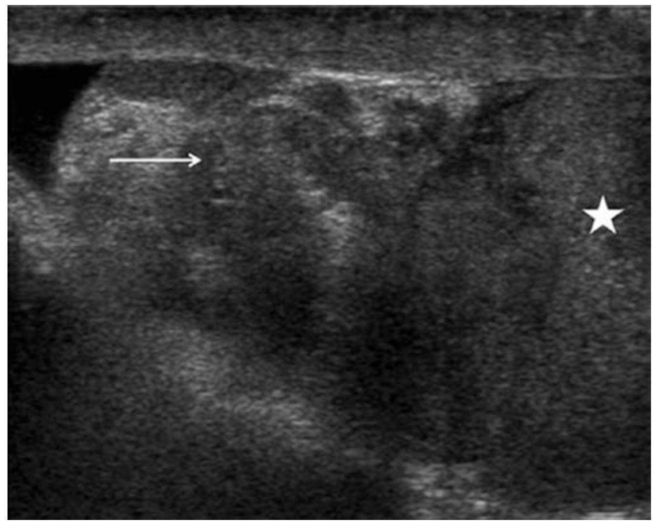


Fig. 5.8 Incomplete torsion of the right spermatic cord. Greyscale longitudinal ultrasound image of the testis in a patient with torsion of the spermatic cord. The testis is heterogeneous and enlarged (*star*), suggesting ischaemia. Lying at the upper aspect is an ill-defined circular mass representing the thickened, oedematous torted spermatic cord (*arrow*). The scrotal wall is also thickened *(From: G.T. Yusuf, P.S. Sidhu. "A review of ultrasound imaging in scrotal emergencies". J Ultrasound (2013) 16:171–178)*

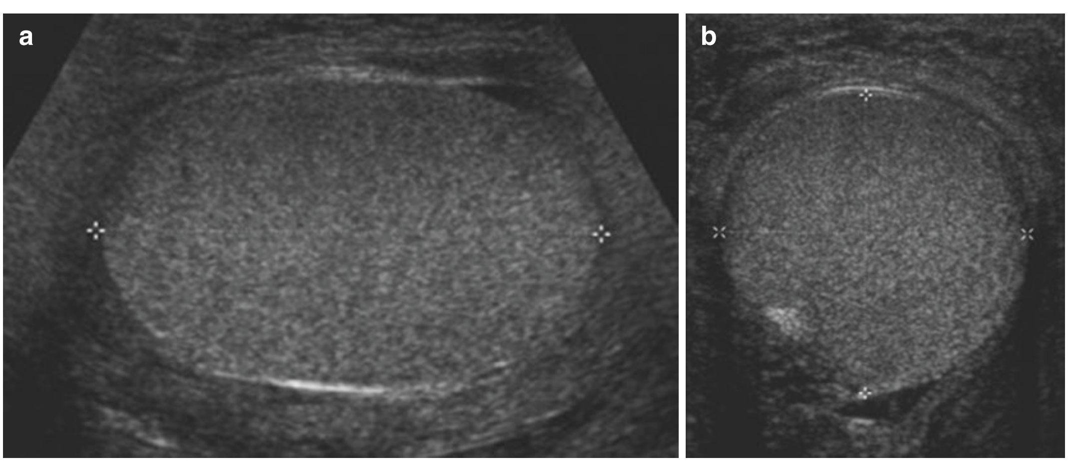


Fig. 5.9 Testicular torsion. Another example of testicular torsion. (**a**, **b**) Sagittal and transverse images of the normal right testis. (**c**, **d**) Sagittal and transverse images of the abnormal left testis. (**e**, **f**) Transverse images of both testes show the normal right testis compared to the abnormal left testis (**g**, **h**). Colour and pulsed Doppler images of the right testis show normal colour and pulsed Doppler signals (**i**, **j**). Colour and pulsed Doppler images of the left testis show no flow (**k**, **l**) *(From: J.R. Mernagh, C.Caco, and J.De Maria. "Testicular Torsion Revisited". Curr Probl Diagn Radiol (2004);33:60–73)*

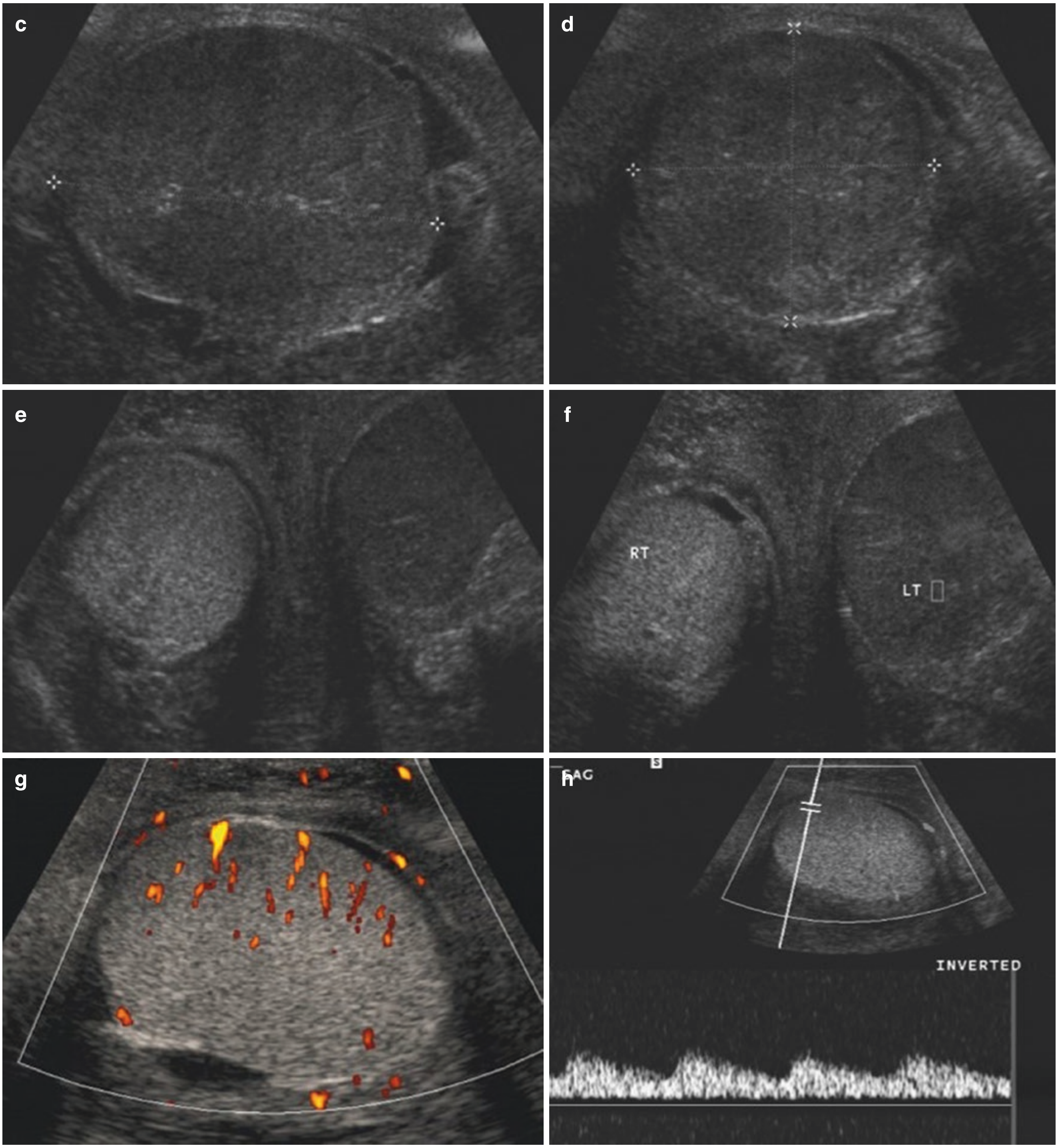


Fig. 5.9 (continued)

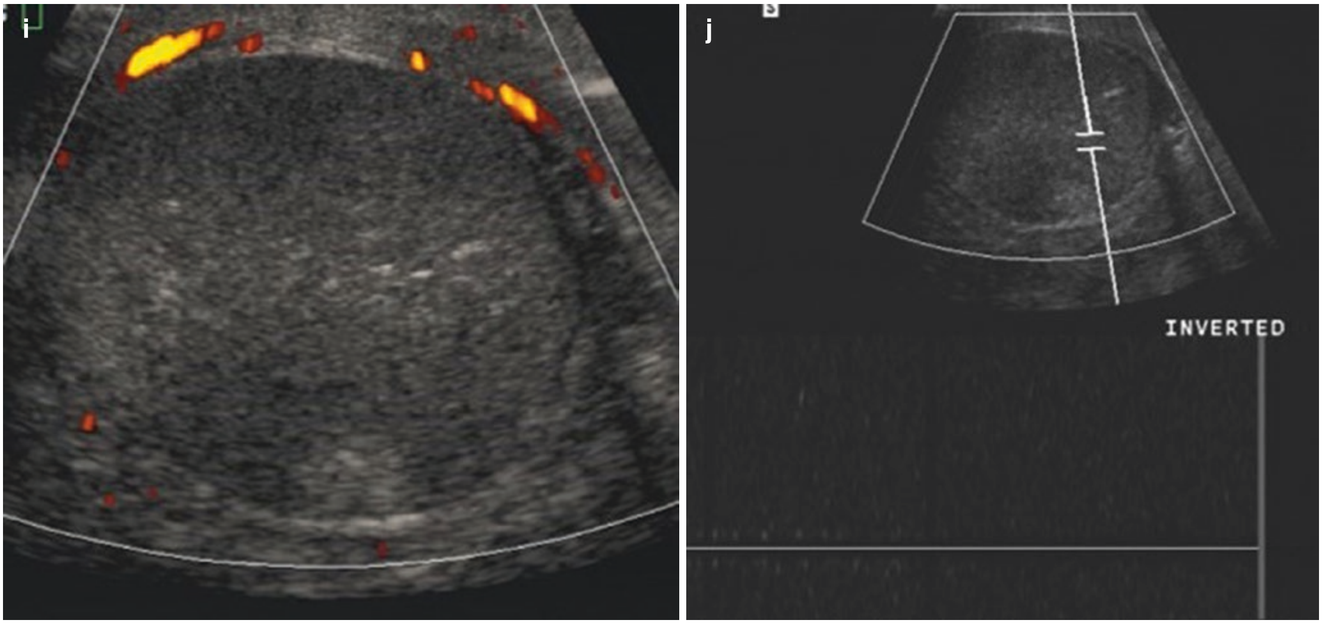


Fig. 5.9 (continued)

sion in the affected testis. Once again it is the clinical history, along with laboratory findings and response to medical treatment, that helps interpret these problematic conditions [5].

The use of **intravascular contrast agents in ultrasound** has grown considerably in recent years and has acquired a role in selected cases, as it may improve the sensitivity of blood flow detection in the acute scrotum [10]. This approach could enable an attempt to quantify vascular inflow and outflow from the testis; however, standardised data are not yet available.

5.3 Torsion of the Testicular Appendix

Torsion of the testicular appendages (Fig. 5.10) is a recognised cause of acute scrotal pain. This is usually due to torsion of the testicular appendix and much less frequently to torsion of the appendix epididymis. Appendiceal torsion can be asymptomatic and accounts for less than 5% of acute scrotum in adults; however, it is nearly as frequent as testicular torsion in children. As treatment of the two conditions differs slightly, a differential diagnosis should be established.

The so-called *blue dot sign* may occasionally be found in appendiceal torsion: in early torsion, the tender appendix can be palpated near the upper pole of the testis and seen as a chrematistic blue dot through the scrotal skin (Table 5.1).

The ultrasound appearance of the twisted testicular appendage has been described as an avascular hypoechoic mass adjacent to a normally perfused testis and surrounded by an area of increased colour flow Doppler perfusion. However, it may also appear as an echogenic extratesticular mass situated between the head of the epididymis and the upper pole of the testis [11]. Compared to the appearance of the normal appendix, which is usually seen only in patients with hydrocele, the torsed appendix is swollen and easily seen as a ‘third body’ in addition to the testis and epididymis. The only accompanying sign is a reactive hydrocele, as the B-mode and colour Doppler aspects of the testis and epididymis are normal [12, 8, 13].

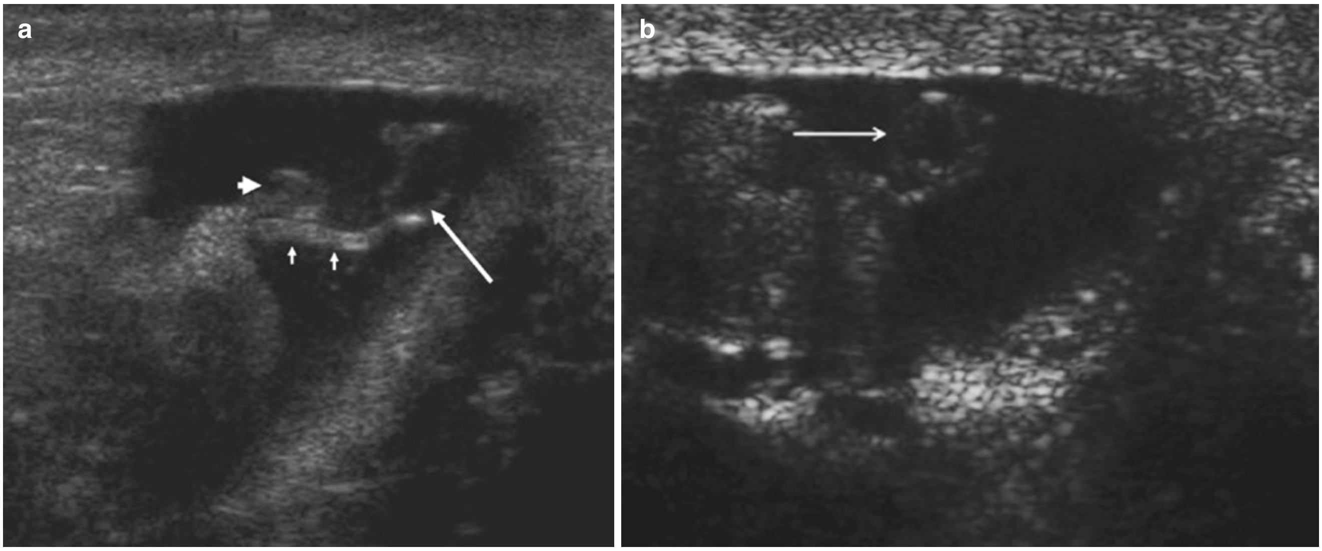


Fig. 5.10 Torsion of the appendix testis. Torsion of an appendix testis. Image through the upper aspect of the testis in a patient with episodes of acute pain, demonstrating a small hydrocele and both an appendix testis (*long arrow*) and an appendix epididymis (*arrowhead*) (**a**). A long stalk to the pedunculate appendix testis is demonstrated (*short arrows*). Greyscale ultrasound image of a patient with acute scrotal pain showing a cystic heterogeneous pedunculated structure arising from the testis, representing an enlarged appendix testis which has undergone torsion (*arrow*). A small surrounding hydrocele is also present (**b**) (panel **a**: *From: V.R. Stewart, P.S Sidhu, "The testis: the unusual, the rare and the bizarre" Clinical Radiology (2007) 62, 289–302; panel* **b**: *from: G.T. Yusuf, P.S. Sidhu. "A review of ultrasound imaging in scrotal emergencies". J Ultrasound (2013) 16:171–178)*

5.4 Trauma

Scrotal trauma is not uncommon and usually results from traffic accidents, athletic injury or direct perineal injury with compression of the scrotum against the pubic bone [1]. Trauma results in contusion, haematoma, haematocele, fracture or rupture of the testis. Untreated testicular injuries may result in ischaemic atrophy, chronic pain or secondary infection [9, 1]. Once the diagnosis is made, however, not all acute post-traumatic events require immediate surgical intervention. The role of US is crucial, especially in the case of blunt trauma, and mainly consists of (a) confirmation or exclusion of testicular rupture, (b) differentiation of soft tissue haematomas from haematocele and (c) follow-up of patients undergoing conservative therapy.

Testicular rupture needs immediate treatment as up to 90% of ruptured testicles can be saved if surgery is performed within 72 h, but less than half if the procedure is carried out later than 72 h [14, 15]. In contrast, a conservative watchful approach can be adopted in intratesticular haematoma, haematoma of the scrotal layers or haematocele if imaging reveals no signs of testicular involvement, including any parenchymal 'suffering' from compression or other indirect mechanisms. Medical treatment may be sufficient to prevent a number of other complications such as infections and development of antisperm antibodies.

Clinical diagnosis is often impossible because of marked scrotal pain and swelling, and therefore colour Doppler ultrasound is essential to establish the diagnosis, evaluate the extent of damage and predict possible complications [1].

Clearly, when interpreting acute scrotum scans, the operator should take into account any pre-existing alterations not necessarily related to the current reason for consultation. Medical history thus remains important prior to ultrasound examination.

The US features of testicular trauma most frequently include single or multiple focal areas of reduced echogenicity, corresponding to **areas of infarction or oedema**. Mixed hypo- and hyperechoic heterogeneous areas corresponding to parenchymal bleeding are normally seen (Fig. 5.11). Haematocele formation is also common and can present without any sign of testicular damage. An irregular or indistinct testicular contour is suggestive of **testicular rupture** (Fig. 5.12), and a break in the continuity of the tunica albuginea, although not always clearly visible, confirms the diagnosis [1]. A discrete plane of fracture has been seen in only a few cases [1, 14], but visualisation of a mixed solid/liquid intrascrotal mass and absence of testis-like structures are indicative of testicular fragmentation. Haematocele (Figs. 5.13 and 5.14) is frequent in testicular rupture but cannot be considered a specific sign. The presence of numerous complex internal echoes distinguishes haematocele from a serous hydrocele.

The appearance of **haematoceles** typically changes with time. In the early stages, they appear hypoechoic, although markedly heterogeneous compared to hydroceles (Fig. 5.15),

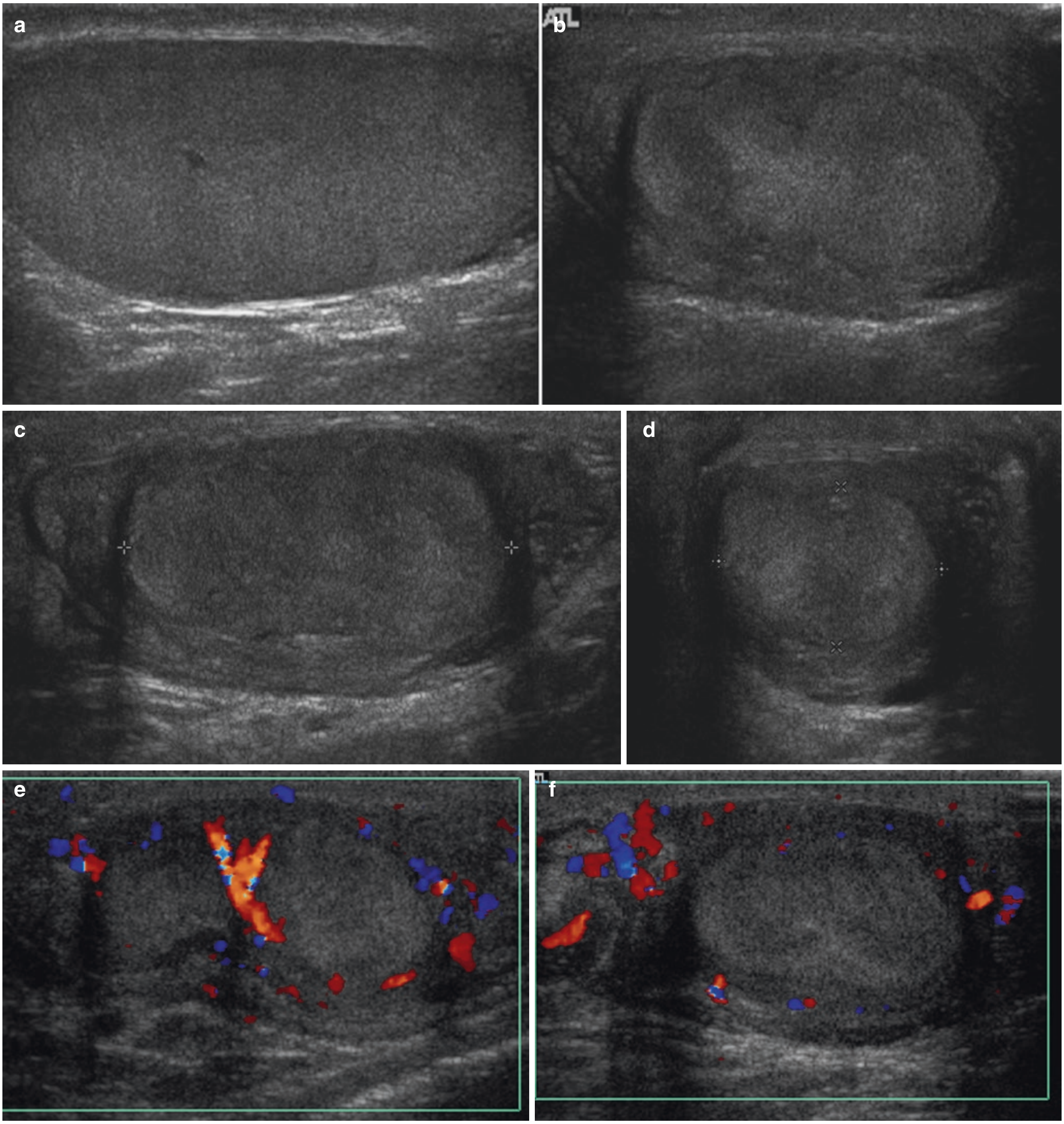


Fig. 5.11 Testicular trauma. US examination of the scrotum of a 24-year-old man after a blunt trauma. Longitudinal and transverse scans of the left hemiscrotum show diffuse oedema, with inhomogeneous echotexture (**a–c**) and loss of definition of the tunica albuginea. Doppler imaging shows an increased but messy blood flow (**d**, **e**). In panels (**f–i**), the scrotal swelling and epididymitis of a 35-year-old patient after testicular injury are shown

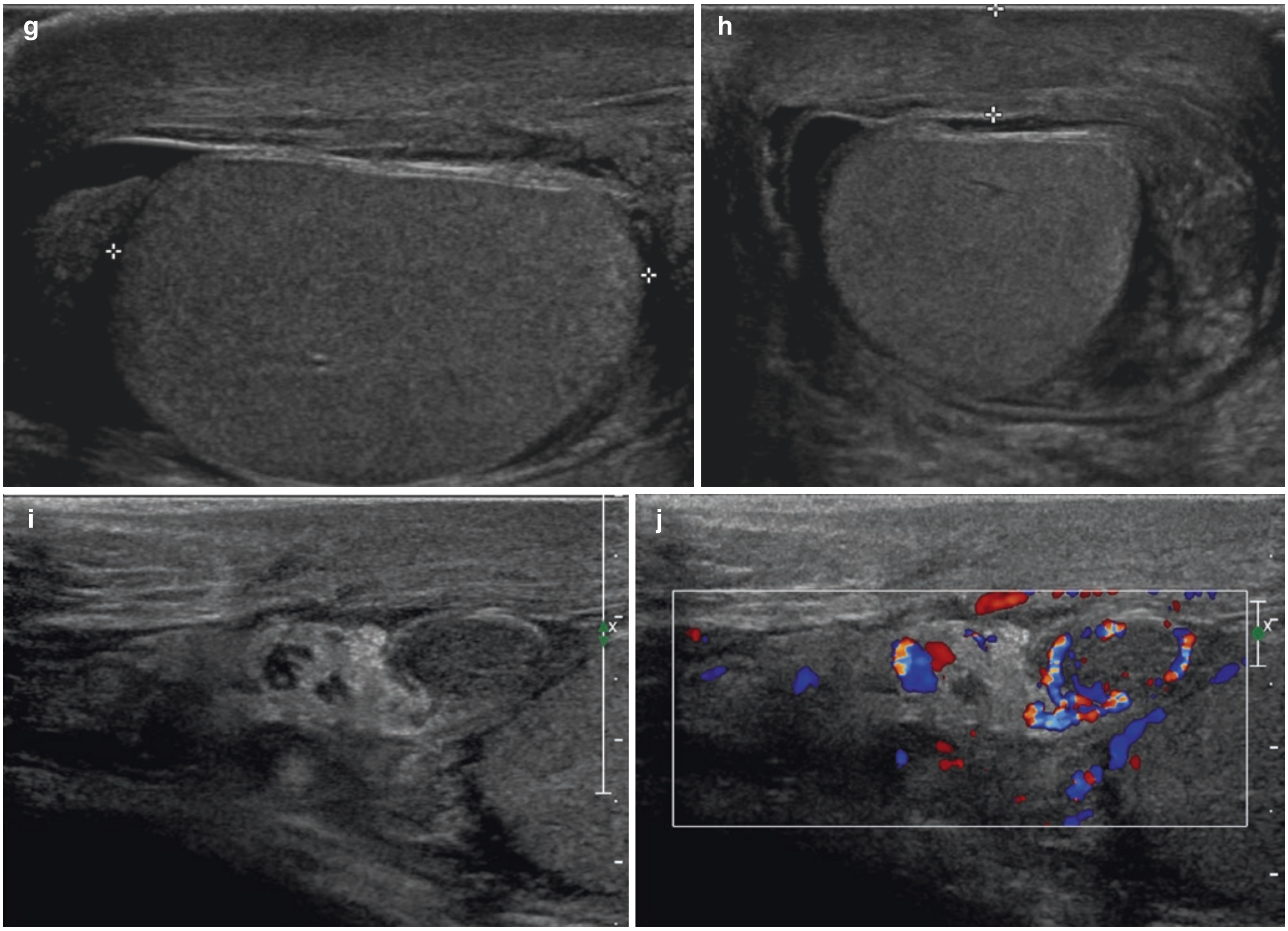


Fig. 5.11 (continued)

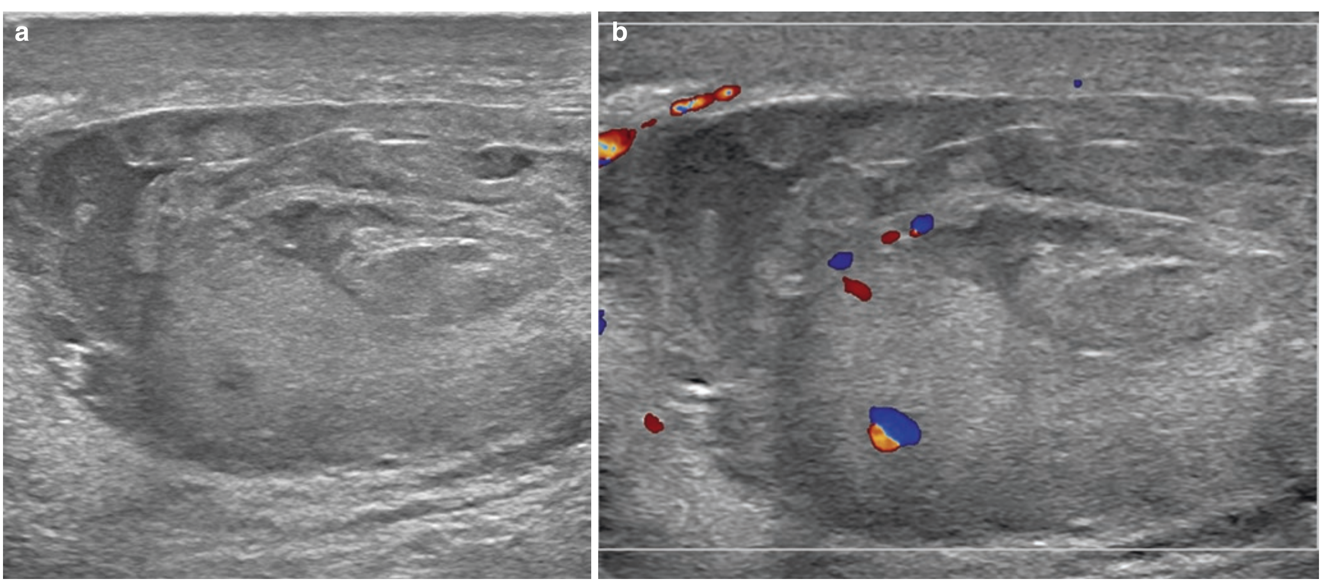


Fig. 5.12 Rupture of the right testis. Longitudinal (**a**, **b**) scrotal US images show an irregular contour of the right testis with heterogeneous echoes, indistinct margins and a hyperechoic haematocele. The scrotal skin was not thickened. Colour Doppler US showed normal vascularity in the upper pole of the right testis but no vascularity in the lower pole *(Courtesy of: M Muttarak, MD)*

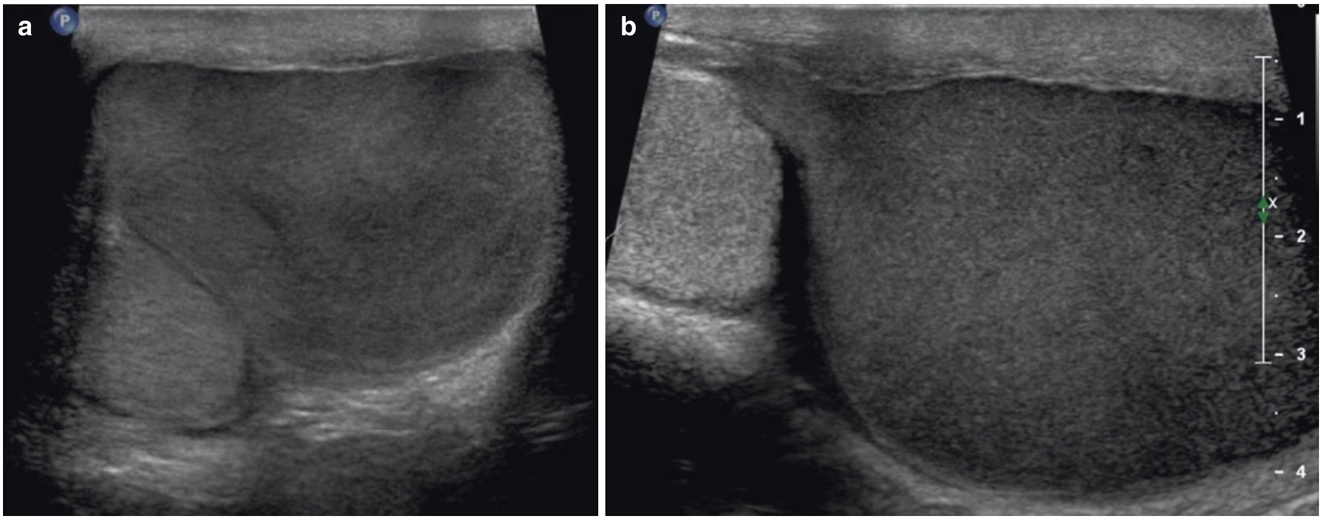


Fig. 5.13 A 19-year-old man who was struck in the scrotum. Longitudinal US image of the scrotum shows large **haematoceles** in the scrotum and a homogeneously echoic right testis

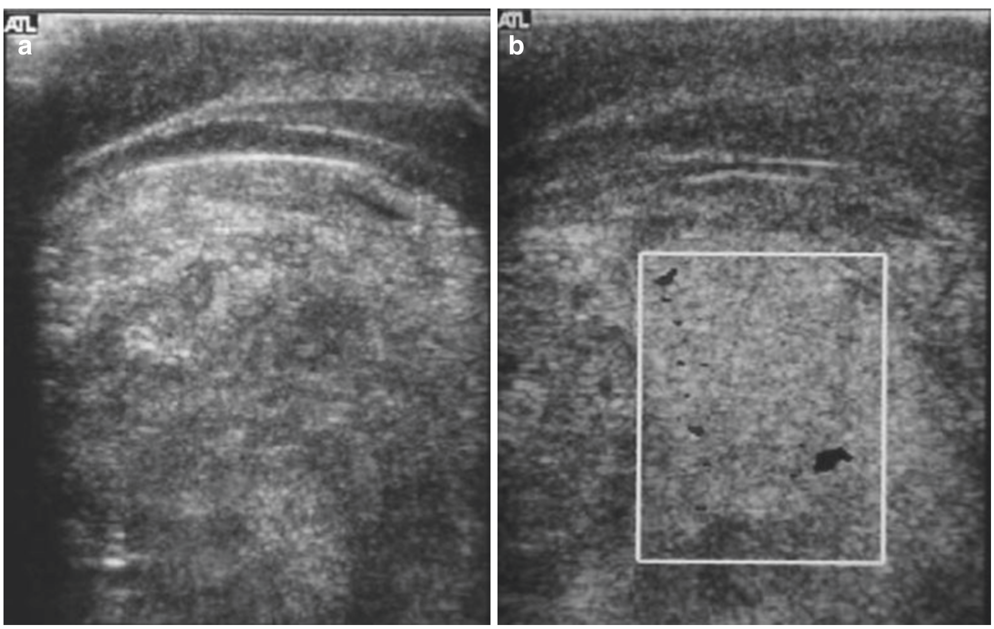


Fig. 5.14 A patient in his mid-20s sustained **blunt perineal trauma** and multiple pelvic fractures as a result of a motorcycle accident. On physical exam, the scrotum was massively swollen. (**a**) Sonographic examination displays the absence of a normal right testicle. The right hemiscrotum is filled with mixed echogenic debris, representing fragments of fractured testicle. (**b**) Doppler imaging shows blood flow within a large segment, likely representing a vascularised fragment of testicle *(From: A.F. Wittenberg, T.Tobias, M. Rzeszotarski, and A.J. Minotti, "Sonography of the Acute Scrotum: The Four T's of Testicular Imaging", Curr Probl Diagn Radiol 2006;35:12–21)*

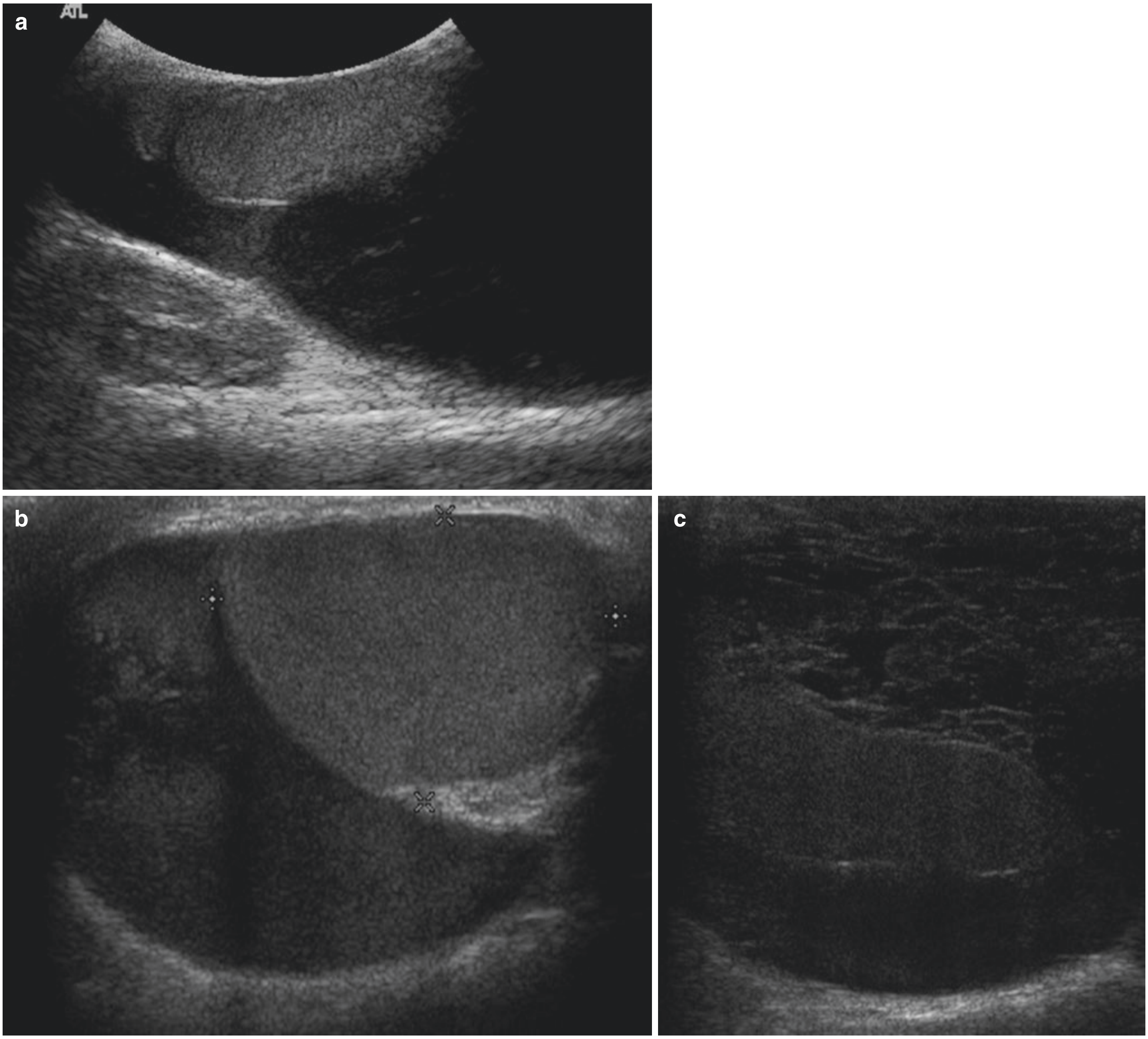


Fig. 5.15 **Testicular trauma** in a 34-year-old-man after being kicked in the right scrotum during a soccer game. Ultrasound of the scrotum revealed large (**a**) and complex haematocele (**b**) with fibrous strand (**c**)

and subsequently become more echogenic as they solidify. Large haematoceles can take several months to reabsorb; they can undergo patchy liquefaction, often leaving a residual hydrocele 'septated' by numerous fibrous strands (Fig. 5.16). Testicular atrophy can develop in long-standing haematoceles. Differential diagnosis includes chronic hydrocele or pyocele. The possibility of super-infection of haematocele after trauma should also be considered.

Intratesticular bleeding causing haematomas is a diagnostic challenge. **Haematomas** (Fig. 5.17) are not uncom-

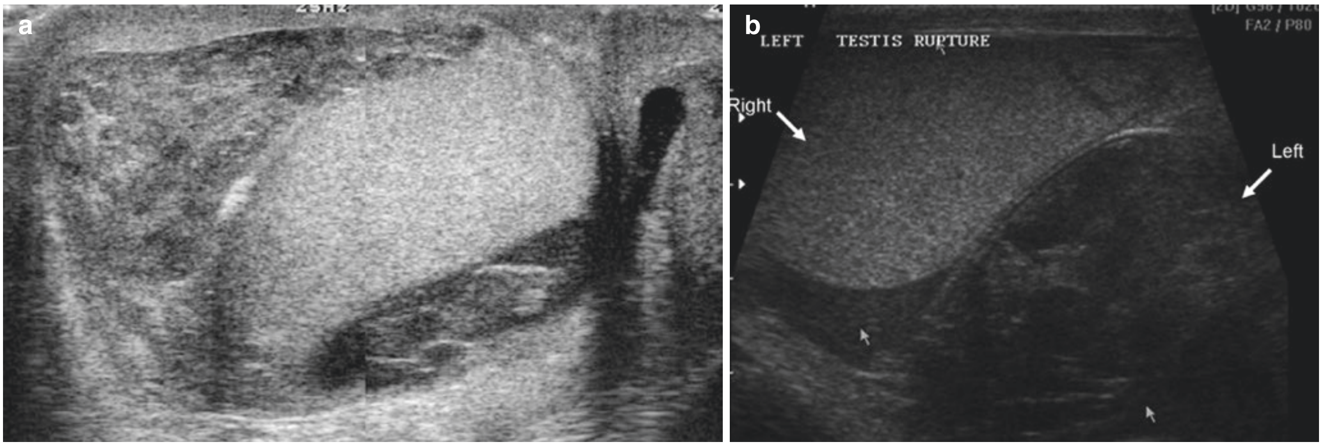


Fig. 5.16 Testicular trauma. Ultrasound scan of the testis showing ruptured testis and haematocele with fibrous strand (**a**). Traumatic rupture of the left testicle (*left arrow*) with complete architectural derangement. The right testicle (*right arrow*) is normal (**b**) (*panel **a**, Courtesy of: R.H. Oyen, MD, panel **b** from: D.D. Cokkinos, MD, E.Antypa, MD, P.Tserotas, et al. Emergency Ultrasound of the Scrotum: A Review of the Commonest Pathologic Conditions - Curr Probl Diagn Radiol 2011;40:1–14*)

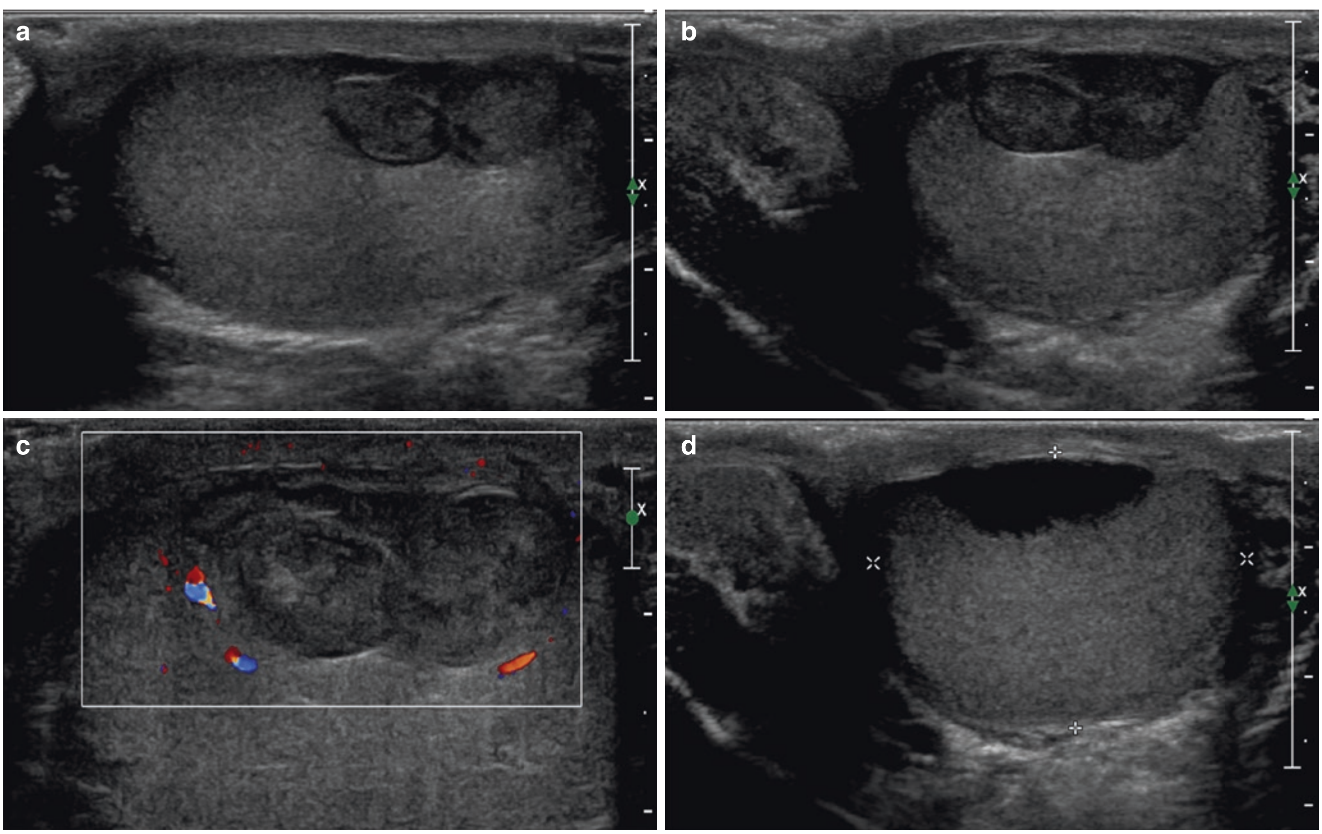
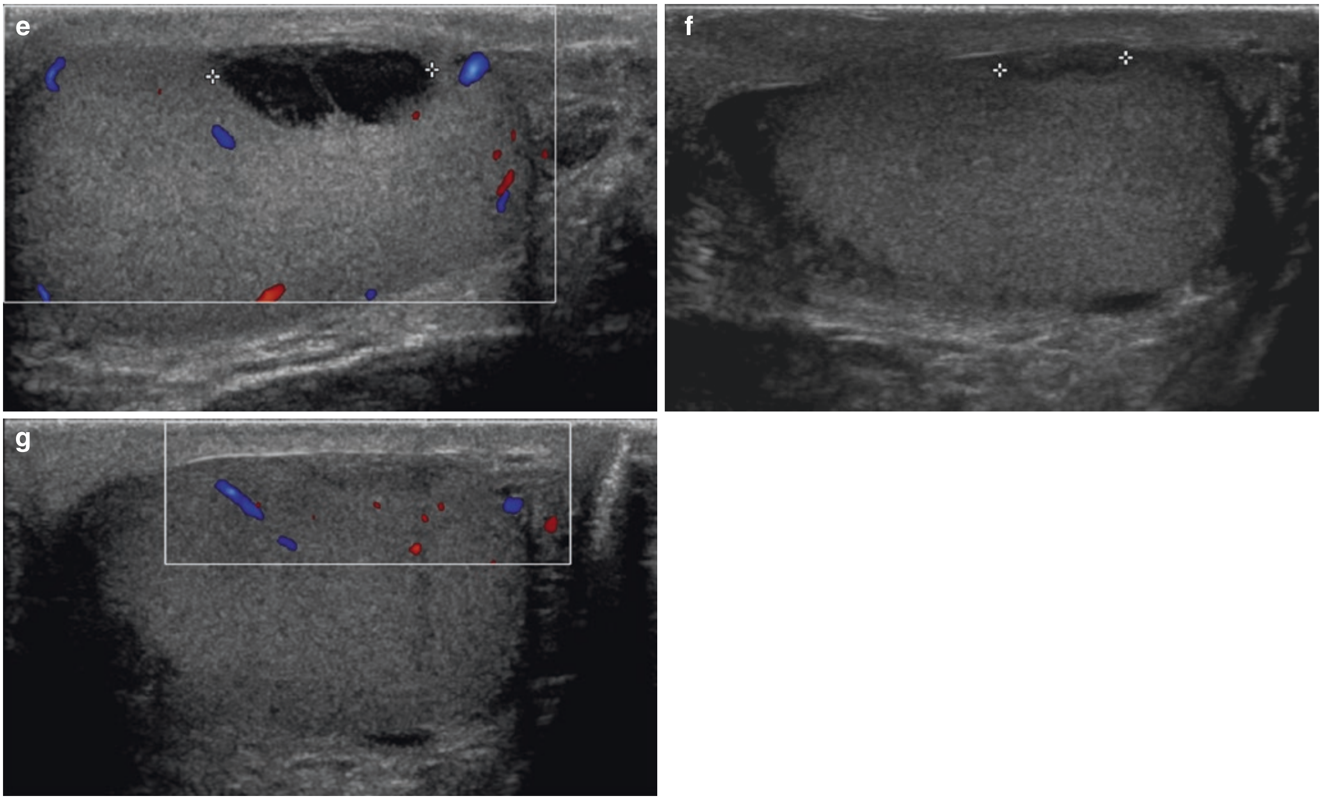


Fig. 5.17 Testicular haematoma in a 20-year-old man involved in motorcycle accident. Longitudinal US image shows an intratesticular bilobate haematoma with clear testicular contour (**a**, **b**). Colour Doppler US image shows normal vascularity of the testis but absence of vascularization inside the lesion (**c**). US images after 2 weeks (**d**, **e**) show a change in the reflectivity of the lesion, still avascular, and after 4 weeks (**f**, **g**) show the almost complete resolution of the hematoma



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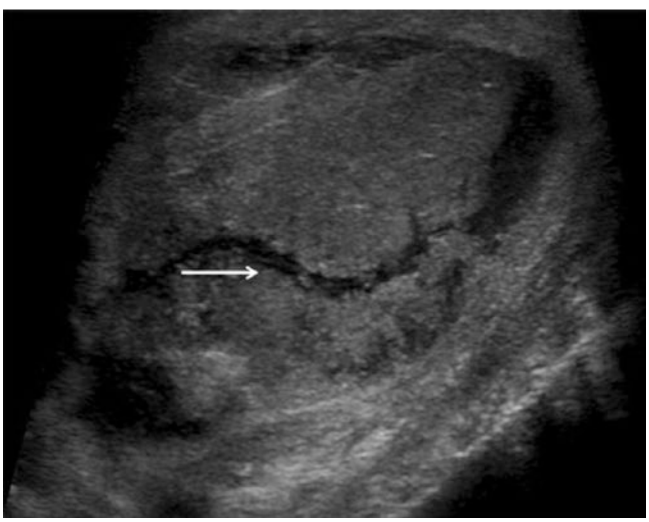


Fig. 5.18 Ruptured testis. A longitudinal greyscale ultrasound image in a patient following testicular blunt trauma. There is a linear hypoechoic line traversing the testis (*arrow*) with mild heterogeneity of the surrounding parenchyma. There is also a small hydrocele and mild thickening of the scrotal wall *(From: G.T. Yusuf, P.S. Sidhu. "A review of ultrasound imaging in scrotal emergencies". J Ultrasound (2013) 16:171–178)*

mon in blunt traumas. They can be seen within the testis but also in the epididymis and scrotal wall. Acute haematomas are hyperechoic and subsequently become hypoechoic (Fig. 5.18) [1, 13]. Intratesticular haematomas can mimic testicular tumours; in these cases, ultrasound follow-up is indicated to reveal the typical temporal changes of haematomas [5]. A more challenging differential diagnosis is from complex intrascrotal haematoma and testicular rupture. Care should be taken to avoid misdiagnosing the two conditions, which require a different approach to treatment. The use of Doppler and colour flow Doppler imaging may aid in distinguishing the normal vascularised testis from a complex haematoma [16].

5.5 Epididymo-orchitis

Epididymitis is the most frequent cause of acute scrotum in adults. In sexually active men, the aetiology is mainly infective (*Chlamydia* and *Neisseriae*), whereas in prepubertal children, abnormalities of the genital tract should be suspected (*Escherichia*). The gradual onset of fever and leucocytosis are distinctive features in the differential diagnosis from torsions. In most cases, only the epididymis is affected (Figs. 5.19, 5.20, and 5.21); its clinical and ultrasound appearance is presented in Chap. 4. The aspect of testicular involvement is briefly presented below.

The great majority of epididymo-orchitis cases originate with a predominant epididymal involvement that later extends to the testis. An exception is viral orchitis (such as mumps orchitis), which primarily attacks the testicular parenchyma. Testicular involvement is therefore clearly detectable as an initial focal (para-epididymal) hypoechogenicity accompanied by enhanced vascularisation that can subsequently cause a diffuse enlargement of the testis (Fig. 5.22). Extensive disruption of parenchymal architecture or cavitations only rarely occurs in infections that are inadequately controlled, alongside abscess formation. To prevent such irreversible complications, we recommend careful follow-up of patients with epididymo-orchitis for 2 weeks after presentation. The differential diagnosis of complicated epididymo-orchitis includes abscesses, obstructions and tumours.

The involvement of the testis in **non-mumps orchitis** mainly occurs via embolisation from the epididymis or supra-infections of damaged testicles (post-surgery, post-trauma, postvasectomy, etc.). However, the term epididymo-orchitis is far too frequently used to describe simple epididymitis. The rare condition of diffuse non-mumps orchitis presents with mild-to-moderate enlargement of the testis, with a fairly even hypoechoic texture (Fig. 5.23) [8]. However, it should be remembered that a surrounding hydrocele can modify the ultrasound beam on

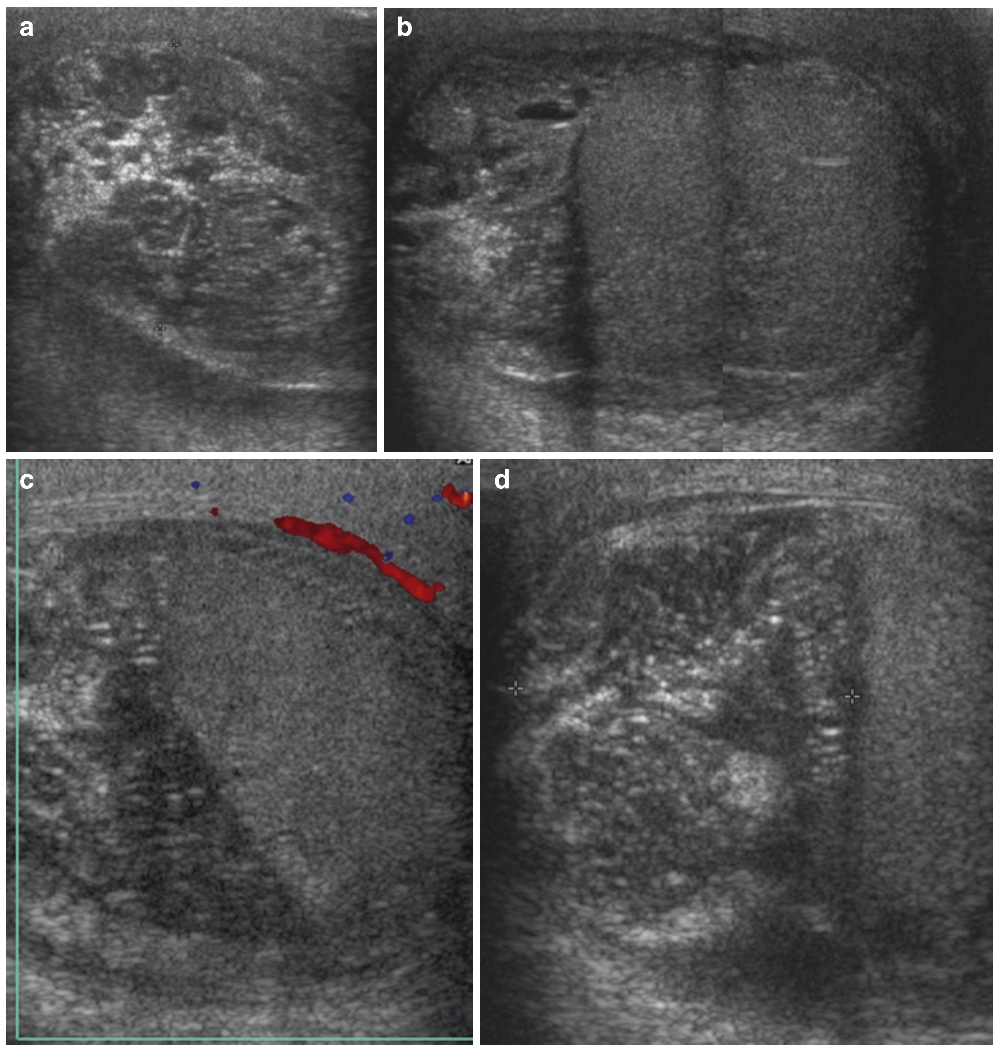


Fig. 5.19 Epididymitis. US scan of the testis of a 17-year-old man, complaining of testicular pain and fever for 3 weeks, revealed a swollen epididymal head with alternated hypoechoic and hyperechoic areas throughout (**a**–**d**)

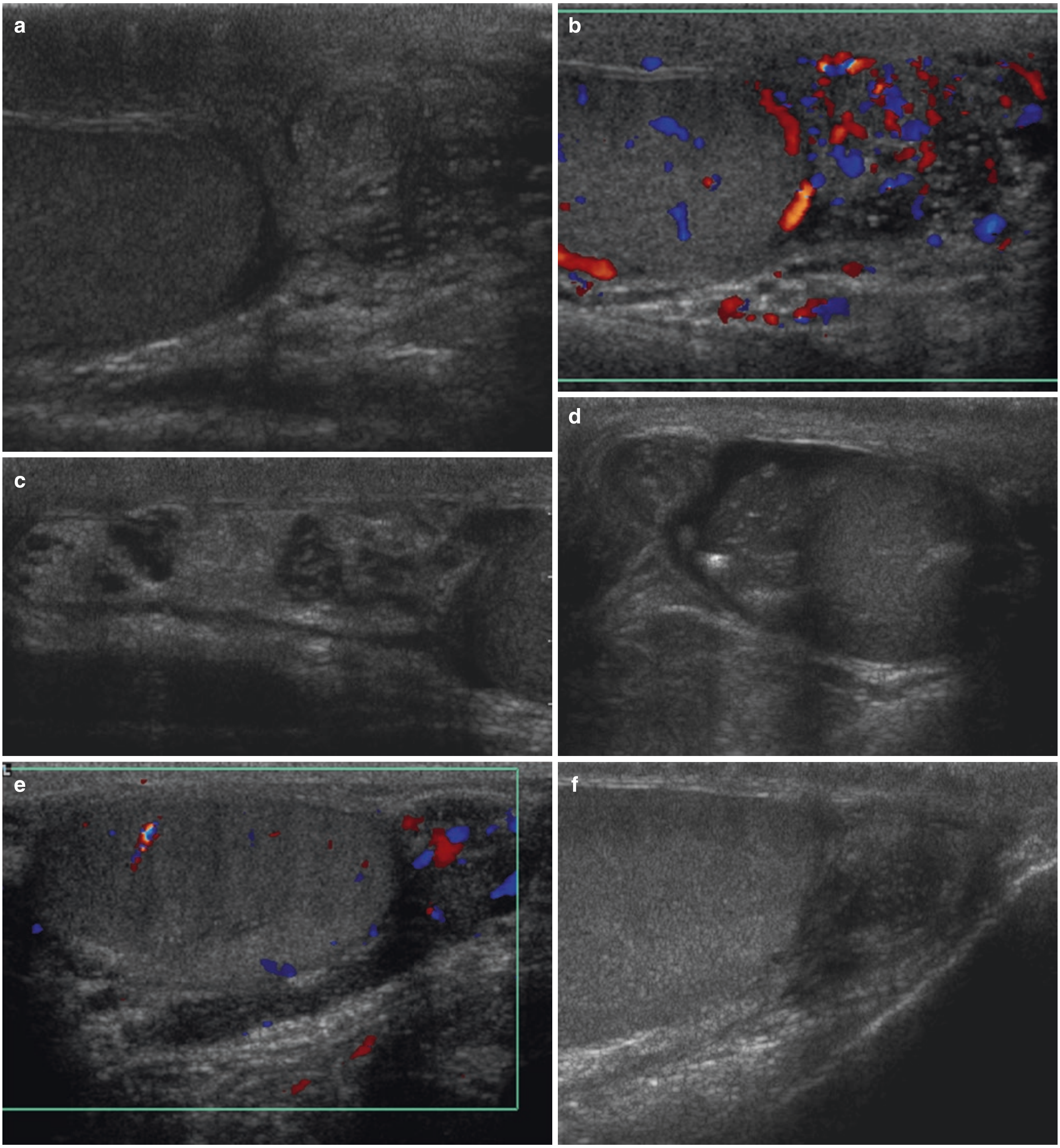
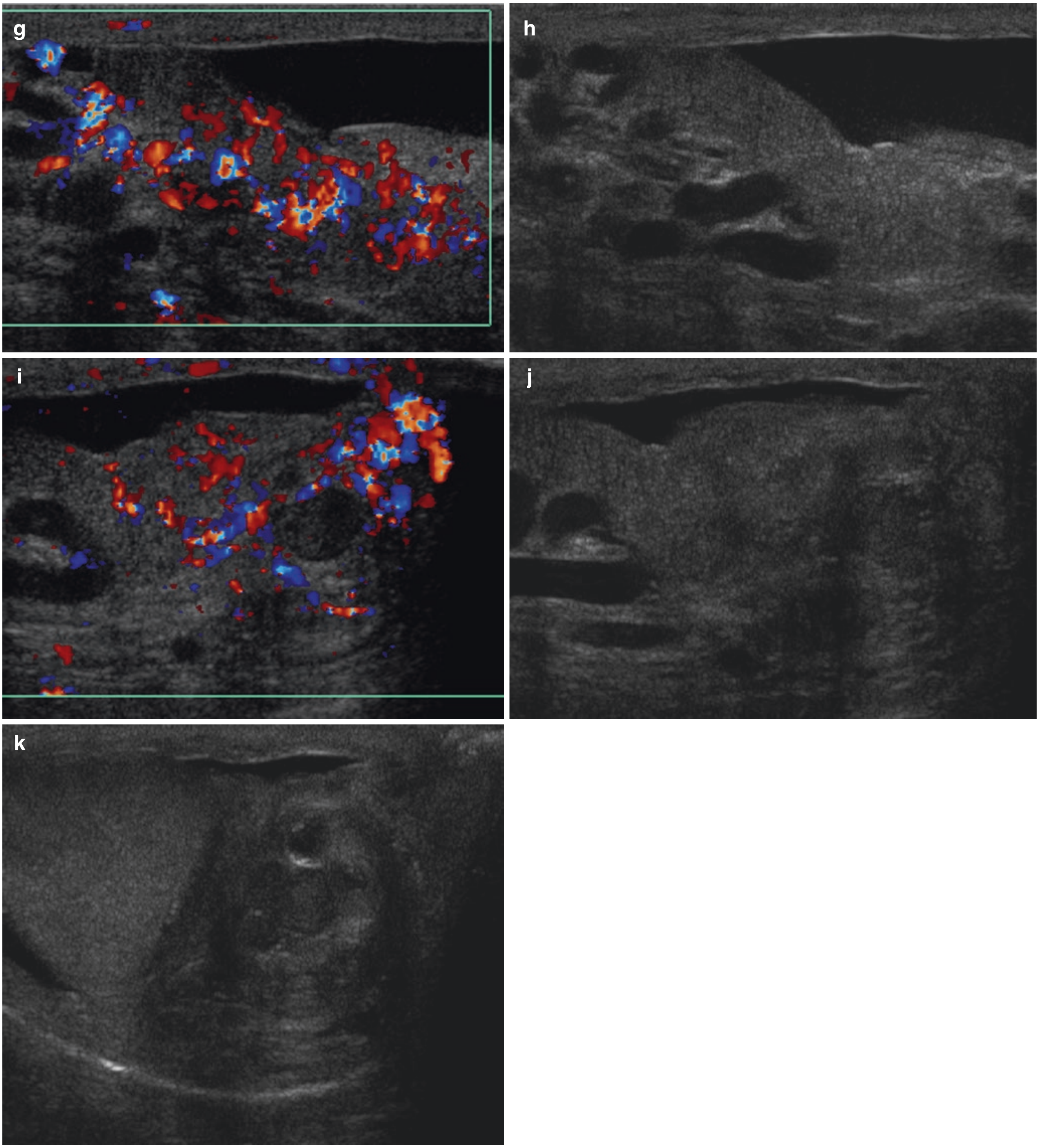


Fig. 5.20 Epididymitis. A selection of epididymitis cases (**a**–**m**): the US features of epididymitis consist of a localised enlargement of the head and tail (**a**–**c**) or diffuse involvement of the entire epididymis, which appears heterogeneous, markedly swollen, often hypoechoic with scattered hyperechoic foci (**d**) and associated with mild-to-severe hyperaemia (**b**, **g**, **i**). The enlargement is paralleled by a reduction in the echotexture (**e**), suggestive of a possible complete or partial obstruction



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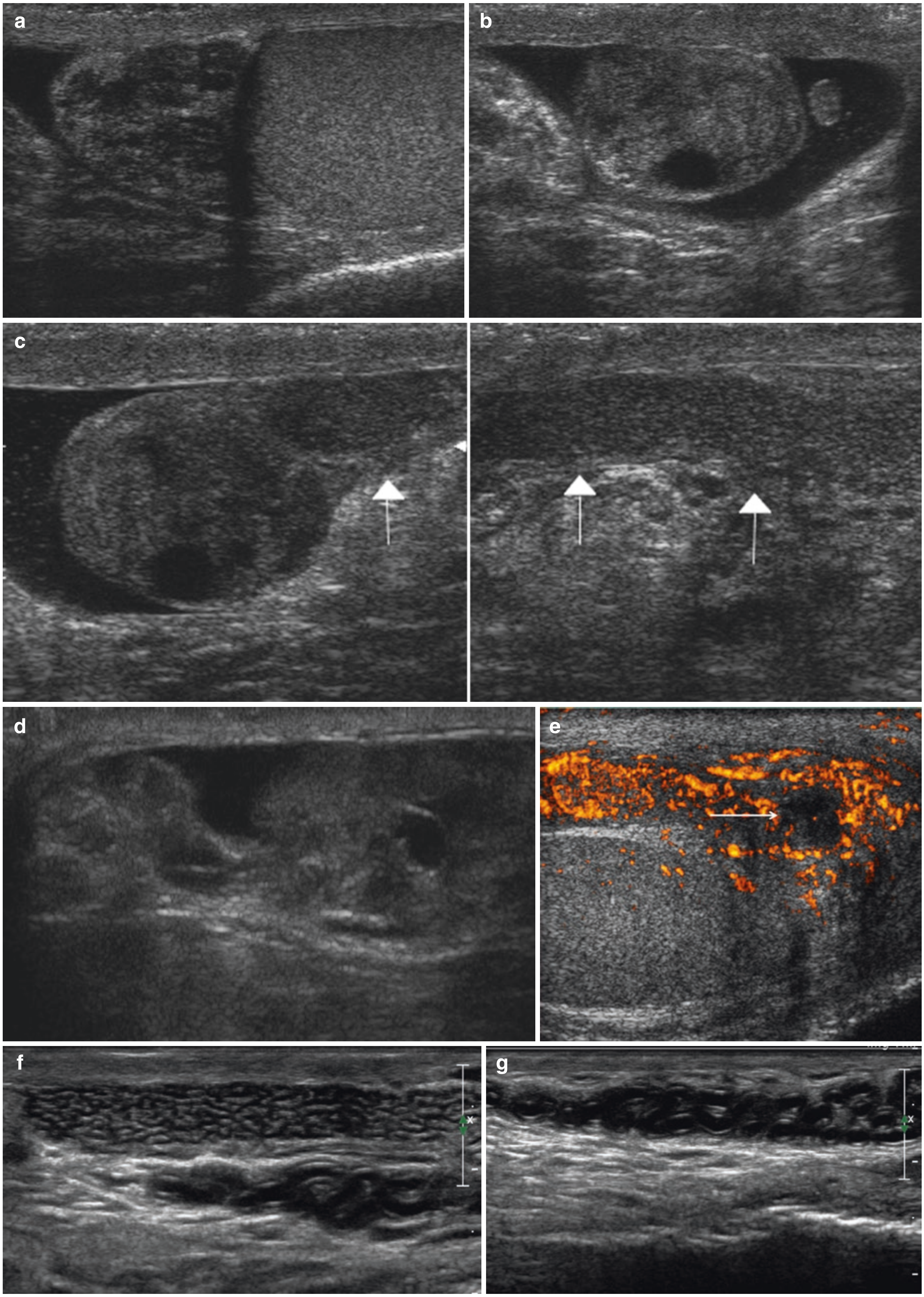


Fig. 5.21 Epididymitis. (**a**, **b**) Epididymitis in a 45-year-old man. The body is enlarged, with a reduced echotexture, which is suggestive of a possible complete or partial obstruction (*arrows*) (**c**, **d**). The tail is more severely affected. In panel (**e**), a focal avascular area within the epididymis representing an area of abscess formation (*arrow*) is shown. Obstruction can be noticed also in the deferent (**f**, **g**) (panel **e** *From: G.T. Yusuf, P.S. Sidhu. "A review of ultrasound imaging in scrotal emergencies". J Ultrasound (2013) 16:171–178)*

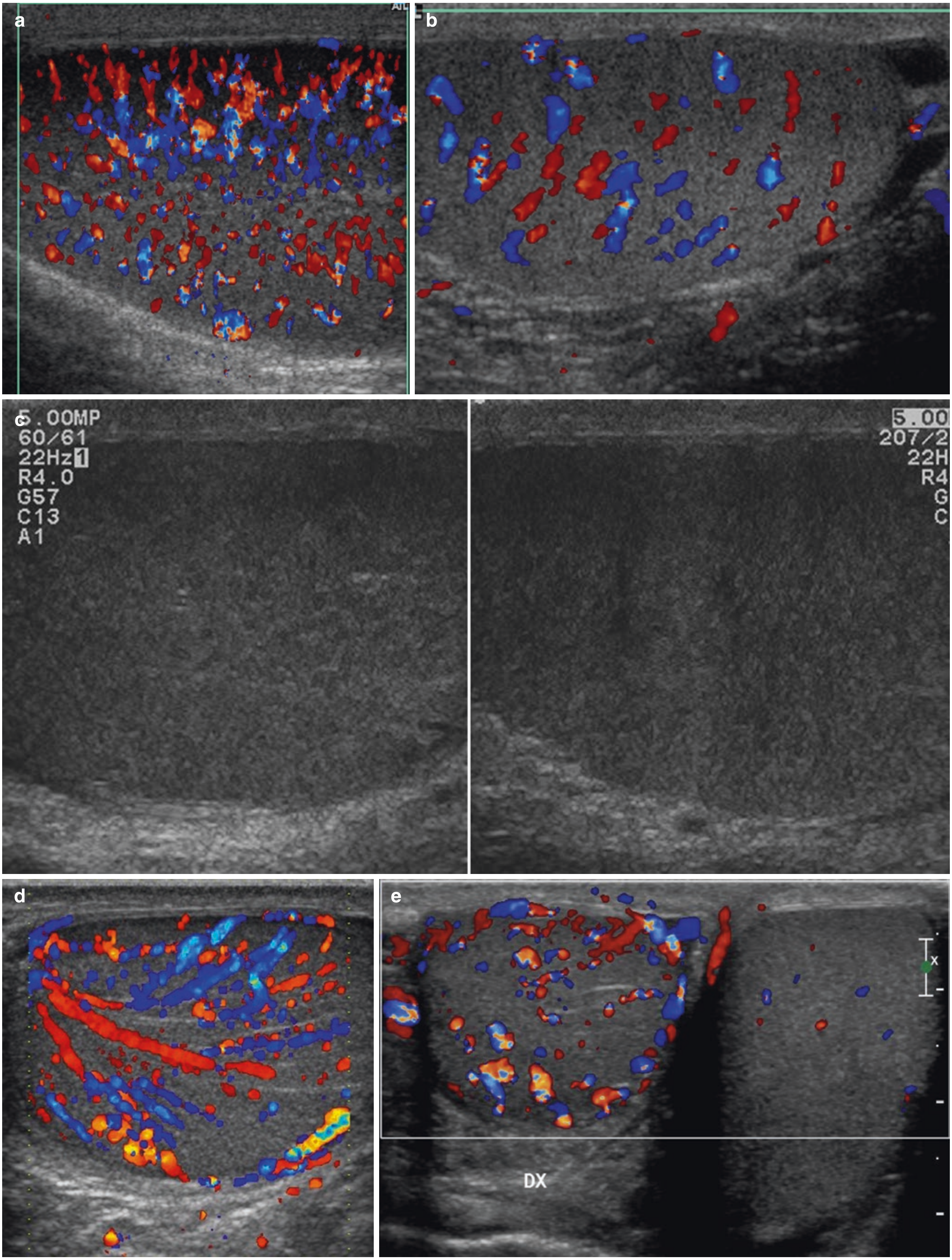


Fig. 5.22 Orchitis. In Doppler study, the testis is intensely hyperaemic. The vessels are enlarged, and a greater vessel number and length are seen than in the normal testis (**a**–**e**) *(panels **a**–**d**, Courtesy of: R.H. Oyen, MD)*

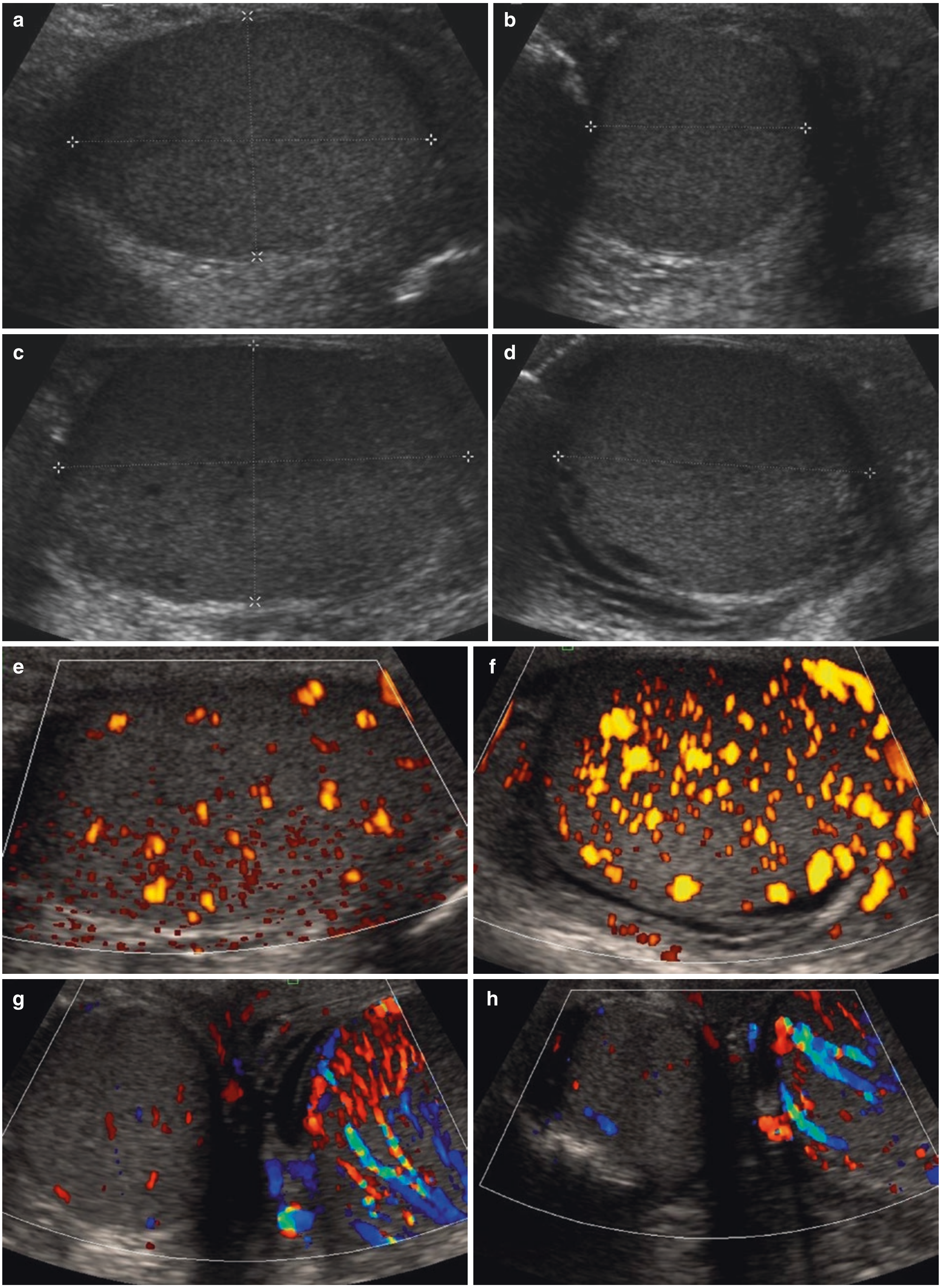


Fig. 5.23 Orchitis. (**a–d**) Sagittal and transverse images of the testes show normal right and left testes. (**e–h**) Two sagittal power Doppler images of the right and left testes and two transverse colour Doppler images of both testes show significantly increased flow in the left testis *(From: J.R. Mernagh, C.Caco, and J.De Maria. "Testicular Torsion Revisited". Curr Probl Diagn Radiol (2004);33:60–73)*

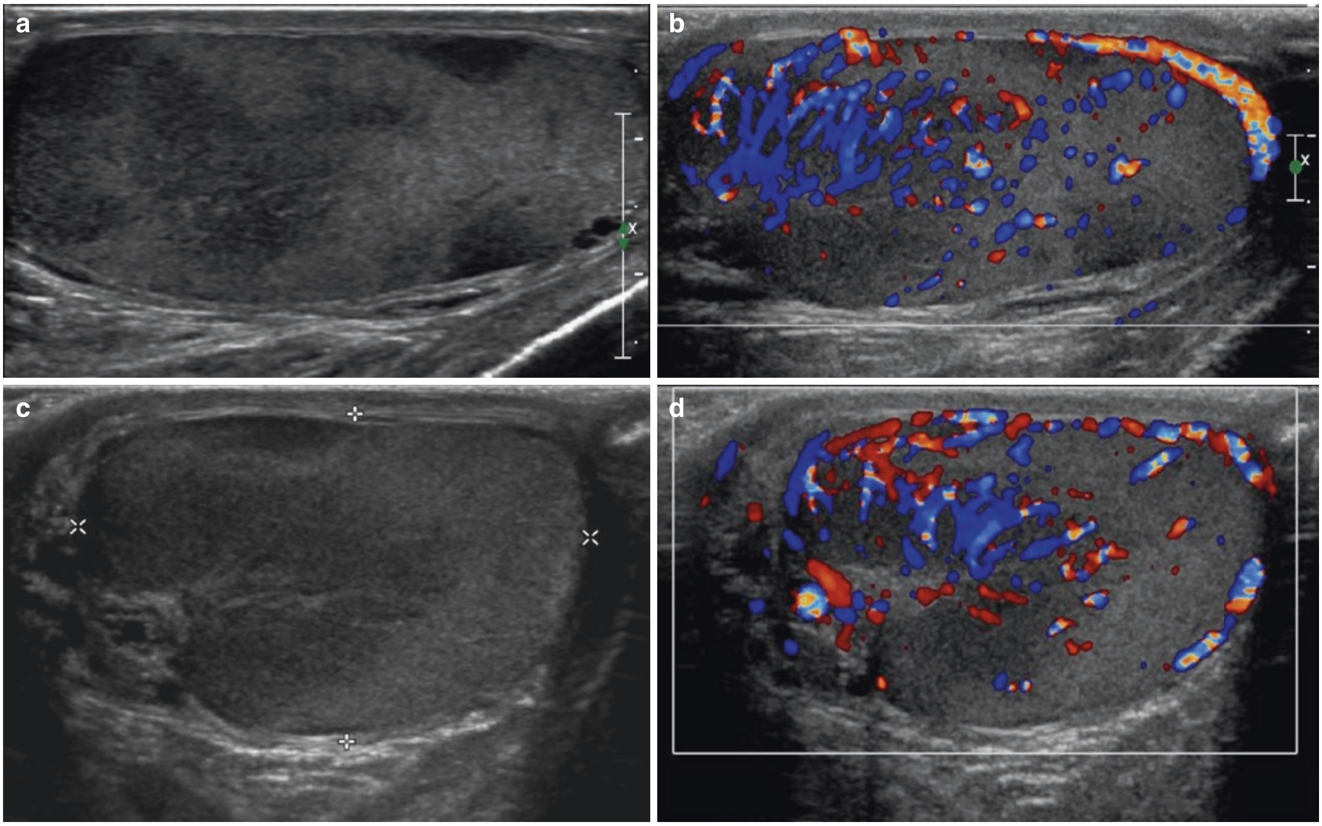


Fig. 5.24 Granulomatous orchitis. Longitudinal and transverse images of the left testis of a 22-year-old man affected by dermatopolymyositis show inhomogeneous echotexture of the testis, with ill-defined, hypoechoic areas highly vascularised. The patient was orchiectomised 6 months earlier for a right seminoma. Histological diagnosis of the left testis revealed granulomatous orchitis (**a**–**d**)

the affected side, resulting in an apparent higher reflectivity of the testis. In addition, although an enlarged, hypoechoic testis is typical of diffuse orchitis, in practice it very common to encounter cases that are hypoechoic but not enlarged, hyperechoic or mixed. The latter is the most difficult to distinguish from large testicular tumours replacing the entire testis (Fig. 5.24). If left untreated, the vascular engorgement of orchitis may compromise venous drainage and produce venous infarction, with the typical initially hyperechoic appearance that subsequently becomes hypoechoic. If adequately treated, orchitis can resolve, but a persistent striated appearance caused by septal accentuation due to fibrosis is a common finding (Fig. 5.25).

Focal orchitis is a less common and more challenging condition. It appears as an area of mixed echodensity in the testis. This is often indistinguishable from tumour by US alone (Fig. 5.26). Focal orchitis can progress into abscess formation, and a clear distinction between the two conditions is not possible, at least in the acute stage (Fig. 5.27).

Mumps orchitis has distinctive clinical and ultrasound features: the testis is affected entirely (never focally), the testis is affected before the epididymis, most cases are accompanied by parotitis (even if not necessarily), and atrophy is a common long-term complication.

Colour Doppler studies are necessary in the evaluation of epididymo-orchitis to establish a differential diagnosis from torsion. In the former, there is a subjective increase in vascularity, above 'what is normally seen', in contrast with a subjective decreased or absent vascularity in torsion. However, this qualitative assessment depends on the particular ultrasound system and settings used as well as operator experience. The absence of hyperaemia does not automatically exclude epididymo-orchitis, as the reactive state often quickly disappears with antibiotic treatment, while venous compression can cause ischaemia. Spectral Doppler patterns normally show increased peak systolic velocity with a variable degree of diastolic flow (Fig. 5.28).

A reversed diastolic flow suggests ischaemia, a rare complication secondary to venous compression or thrombosis.

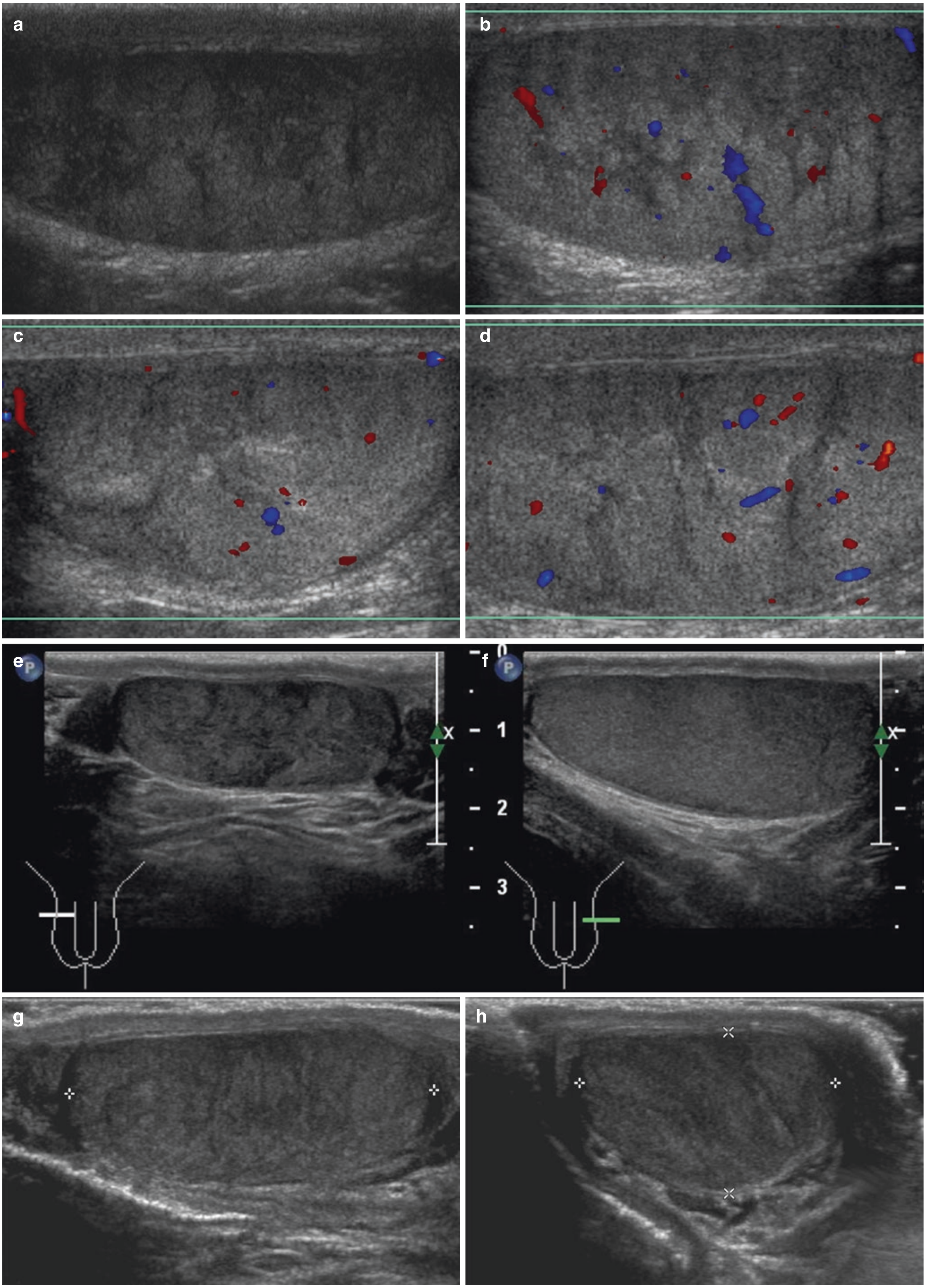


Fig. 5.25 Post-orchitis. Different US scans of abnormal echotexture due to previous orchitis

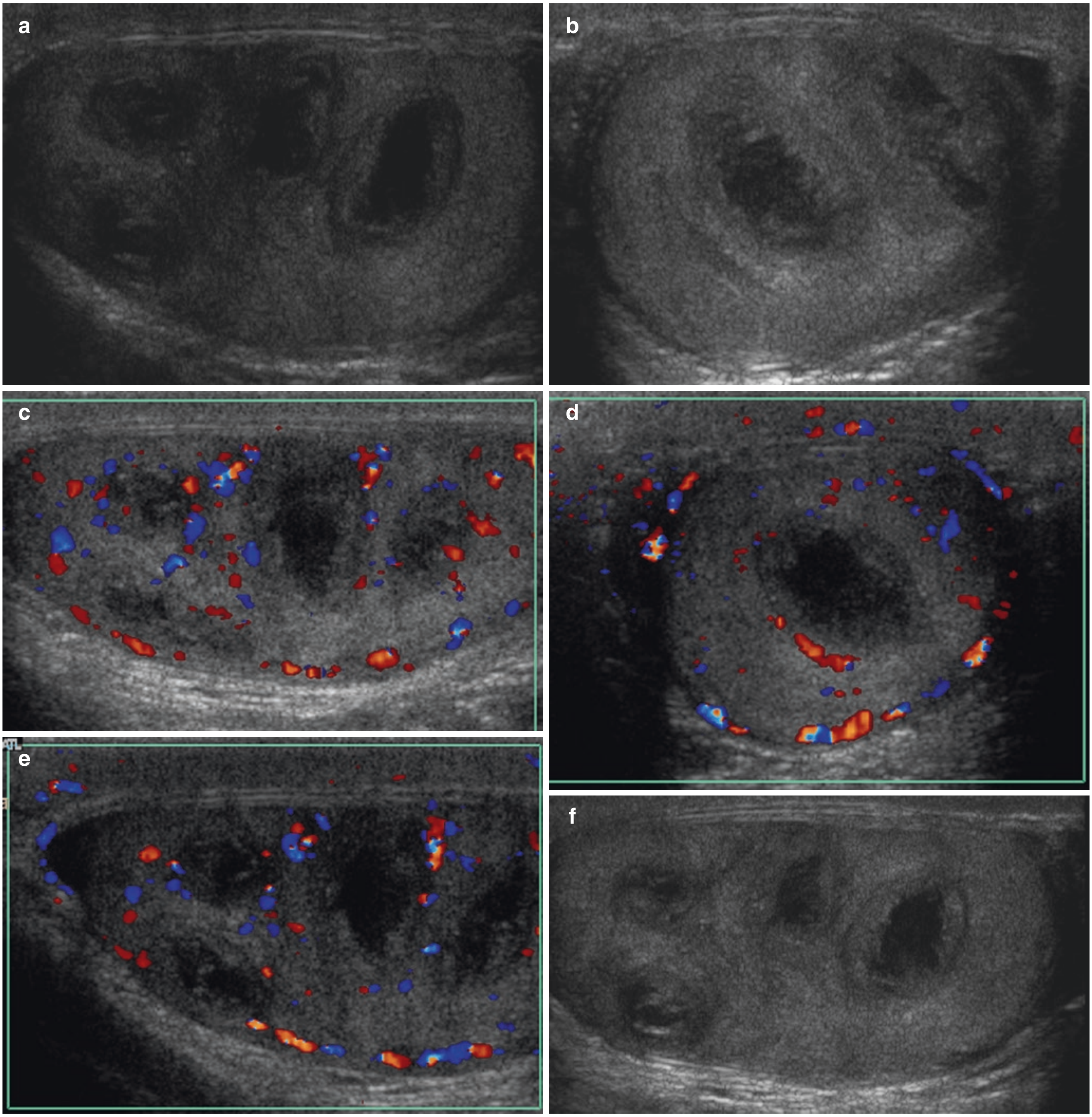


Fig. 5.26 Multifocal orchitis. Several well-defined hypoechoic masses (**a**–**f**) are seen within the right testis, which is mildly enlarged, with inhomogeneous echotexture in the periphery. These areas are characteristic of focal orchitis. On colour Doppler the areas are avascular

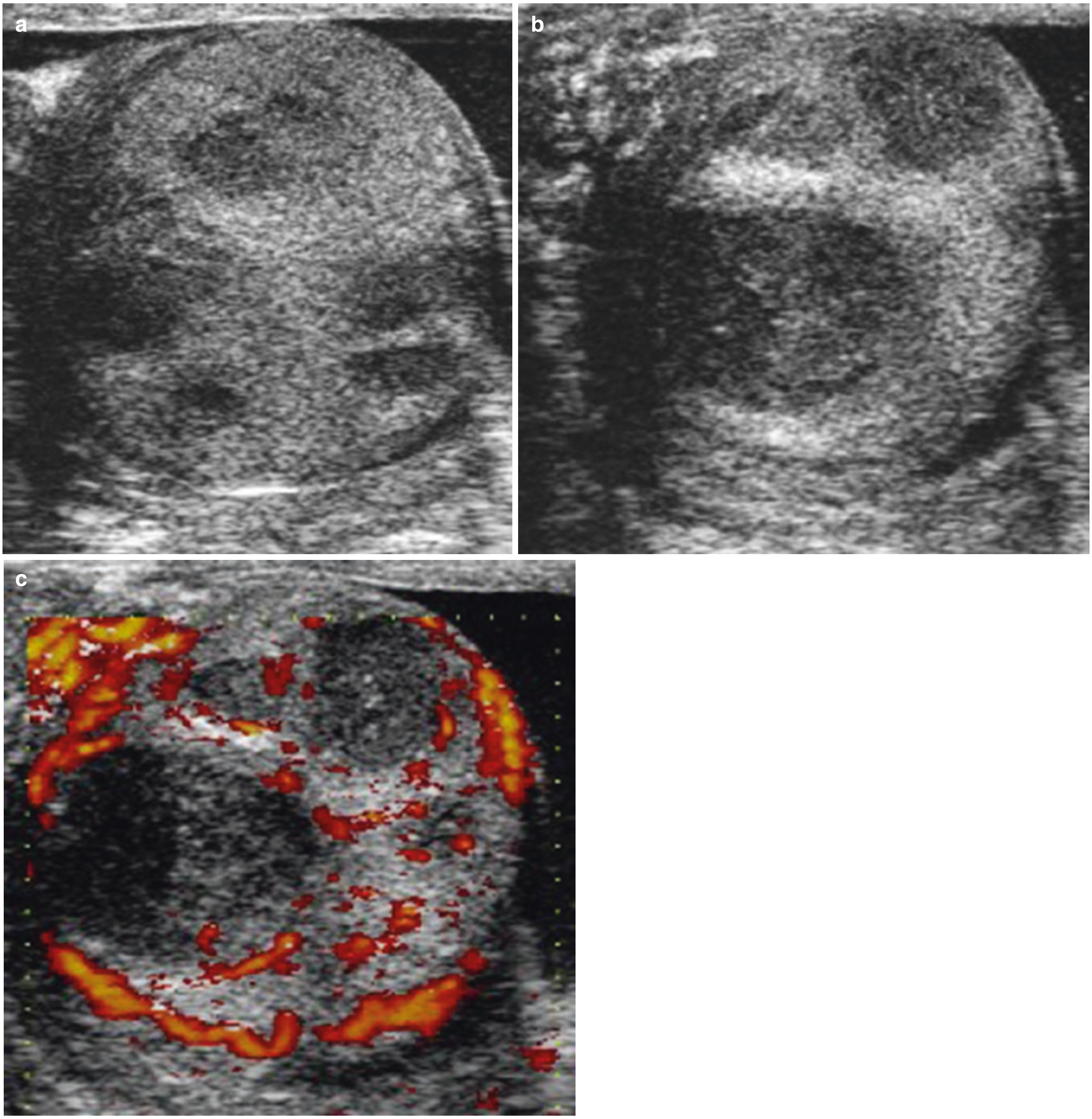


Fig. 5.27 Multifocal orchitis. The testis is of very mixed echodensity but predominantly hypoechoic. There are several areas of focal orchitis within the entire testis. On colour Doppler, the areas are avascular *(Courtesy of R.H. Oyen, MD)*

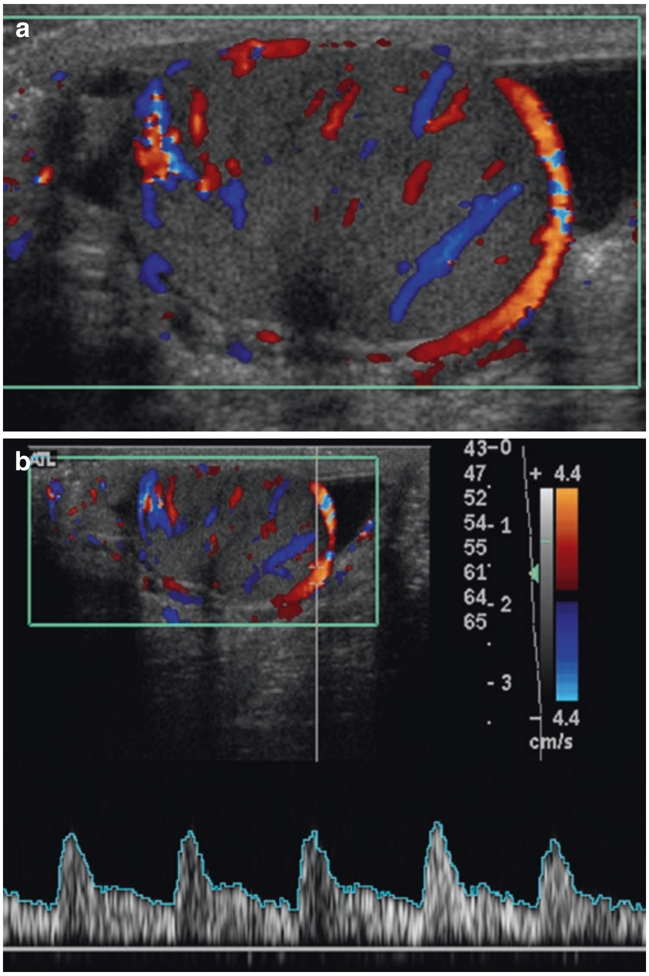


Fig. 5.28 Spectral Doppler is generally unhelpful, varying between low- and high-resistance patterns. Peak systolic velocities are generally elevated

5.6 Miscellaneous

5.6.1 Acute Idiopathic Scrotal Oedema

Acute idiopathic scrotal oedema (AISO) is a rare condition in adults but is well recognised in children. Patients may be asymptomatic, usually apyretic, or complain of scrotal discomfort. Itching is unusual [17]. All diagnostic tests are negative. The aetiology of this condition remains unclear, although many theories have been proposed (infective or allergic phenomenon, trauma or urinary extravasation) [17].

This condition is characterised by a rapid onset of scrotal swelling and erythema – symptoms that can mimic torsion of the testis or appendages, epididymo-orchitis, trauma and Fournier's gangrene. The swelling may be unilateral or bilateral. Typically, patients have no primary source of infection and all laboratory investigations are normal [17]. Ultrasound findings include a normal testicular parenchymal appearance with adequate blood supply to the affected testis, with an associated peritesticular oedema and a considerable amount of fluid in the scrotal wall that results in marked thickening of the subcutaneous scrotal tissues (Fig. 5.29) [18]. Mild reactive hydrocele, enlarged inguinal lymph nodes and a significant increase in the vascularisation of the scrotal skin and subcutaneous tissues are frequently seen [19]. The scrotal wall has the appearance of a series of concentric rings with thin bands of high reflectivity separated by bands of low reflectivity. The scrotal wall in AISO has thus often been described as resembling an onion [17].

The condition is self-limiting, and to prevent unnecessary surgical exploration or antibiotic therapy, it should be recognised and distinguished from other, more serious diseases [20]. Treatment consists of bed rest and scrotal elevation. Analgesics are rarely needed.

5.6.2 Thrombophlebitis of the Pampiniform Plexus

Very rarely, veins in large varicoceles can become inflamed and develop thrombosis. The presentation is of a continuous, persistent mild-to-moderate pain on the left side. Examination reveals an irregular lobulated solid structure in the upper part of the scrotum surrounding the spermatic cord. The US appearance is that of varicocele but with mixed echogenicity within the dilated vessels. Most of the affected vessels show no detectable flow.

5.6.3 Vasculitis and Henoch-Schönlein Purpura

Necrotising vasculitis affecting the testis can be seen in patients with polyarteritis nodosa, Henoch-Schönlein Purpura and rheumatoid arthritis. Patients present with an acute scrotum. The US features are those associated with infarction and intraparenchymal bleeding (see chapter on testicular ischaemia). The failure to diagnose vasculitis has caused many unnecessary surgical explorations, and physicians should be more careful to include these conditions in the differential diagnosis. The most challenging situation is the distinction between vasculitis and torsion in prepubertal boys, where surgical exploration is often unavoidable. On ultrasound the only abnormality detected may be an enlarged testis with hypoechoic echotexture. However, when infarcts develop, the diagnosis is clearer, as they appear as strongly hypoechoic and sharply demarcated wedge-shaped peripheral areas with septal distribution. A round hypoechoic mass may however be seen early in the course of infarct, and in this case, differential diagnosis from an abscess or haemorrhage within a tumour is fairly difficult.

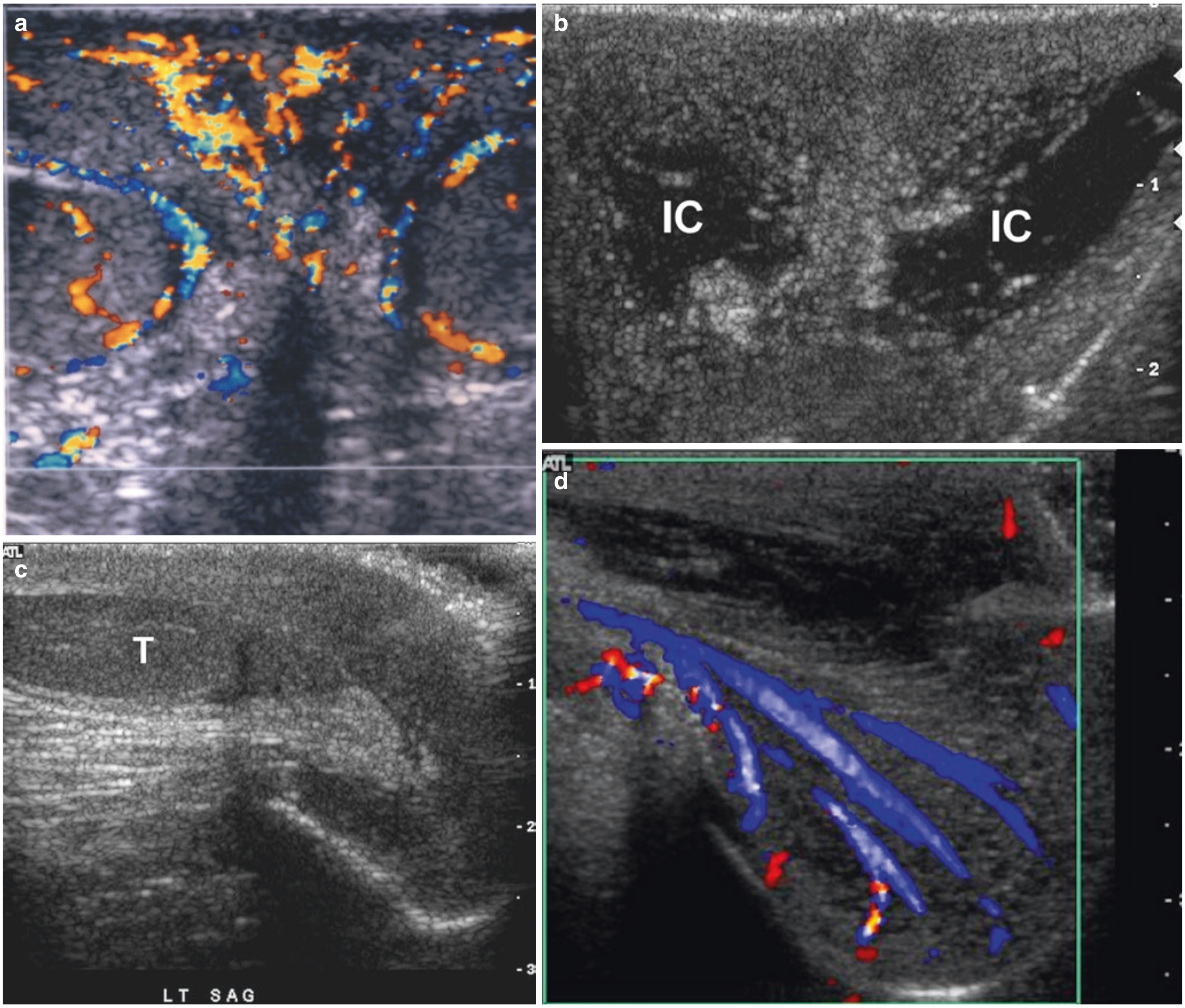


Fig. 5.29 Idiopathic scrotal oedema. Transverse image of the scrotum shows marked scrotal wall thickening and hyperaemia with normal appearance of the intrascrotal contents (**a**). A 6-year-old boy with acute idiopathic scrotal oedema. Transverse and longitudinal scans through the inguinoscrotal area show extreme soft tissue swelling and oedema involving the scrotal and inguinal soft tissues. Transverse colour Doppler scan of the left scrotal soft tissues shows significant hyperaemia. T, testicle; IC, inguinal canal (**b–d**) (panel **a** *From: Martha M. Munden, Lynn M. Trautwein,* "Scrotal Pathology in Pediatrics with Sonographic Imaging" Current Problems in Diagnostic Radiology (2000) vol 29, no. 6, 186–205; panel **b–d** from *Zachary C. Williamson, M. Epelman, A. Daneman et al, "Imaging of the Inguinal Canal in Children" Curr Probl Diagn Radiol 2013;42:164–179)*

5.6.4 Fournier's Gangrene

Fournier's gangrene was originally described in 1883 by a Parisian venereologist, J.A. Fournier, and was thought to be an idiopathic process. Since then, it has been identified as having a polymicrobial origin (most frequently *Streptococcus*, *Staphylococcus* and anaerobics) [21]. Fournier's gangrene is defined as a synergistic, polymicrobial, necrotising fasciitis of the perineum, scrotum and penis characterised by obliterative endarteritis, resulting in gangrene of the subcutaneous tissue and overlying skin [22]. It is an uncommon but not rare condition. The necrotising process commonly originates from an infection in the anorectum, urogenital tract or the skin of the genitalia. Men with alcoholism, diabetes mellitus, leukaemia, morbid obesity and immune system disorders (e.g. HIV, Crohn's disease) and intravenous drug users are at increased risk of developing Fournier's gangrene. The condition can also develop as a complication of surgery.

Proposed pathogenetic mechanisms include small vessel disease, predisposing to tissue ischaemia, defective phagocytosis and increased incidence of urinary tract infections [22].

The typical patient is an elderly male in his sixth or seventh decade of life.

The hallmark of Fournier's gangrene is intense pain and tenderness in the genitalia. The clinical course usually progresses through several phases: prodromal symptoms of fever and lethargy, which may be present for 2–7 days, intense genital pain and tenderness that is usually associated with oedema of the overlying skin, increasing genital pain and tenderness with progressive erythema of the overlying skin, dusky appearance of the overlying skin, subcutaneous crepitation (due to the subcutaneous gases produced by the growing bacteria), obvious gangrene of a portion of the genitalia and purulent drainage from wounds. The systemic effects of this process vary from local tenderness to florid septic shock. In general, the greater the degree of necrosis, the more serious the systemic effects.

Fournier's gangrene is a potentially life-threatening condition that is easily diagnosed solely on clinical grounds when advanced. Any delay in diagnosis, such as misidentification as simple cellulitis or idiopathic scrotal oedema, can significantly delay treatment and leads to an increased morbidity and mortality [21]. Ultrasound findings (Figs. 5.30 and 5.31) include marked thickening of the scrotal skin as well as discrete focal regions of high-amplitude echoes with posterior acoustic shadowing indicative of subcutaneous gas, which is pathognomonic for Fournier's gangrene. Additional ultrasound signs include peritesticular fluid and possible signs of epididymo-orchitis. Gas formation (hyperechoic foci with ill-defined shadowing) is detectable before the clinical presentation of crepitations and purulent drainage, allowing early diagnosis that significantly improves the disease's morbidity and mortality [21].

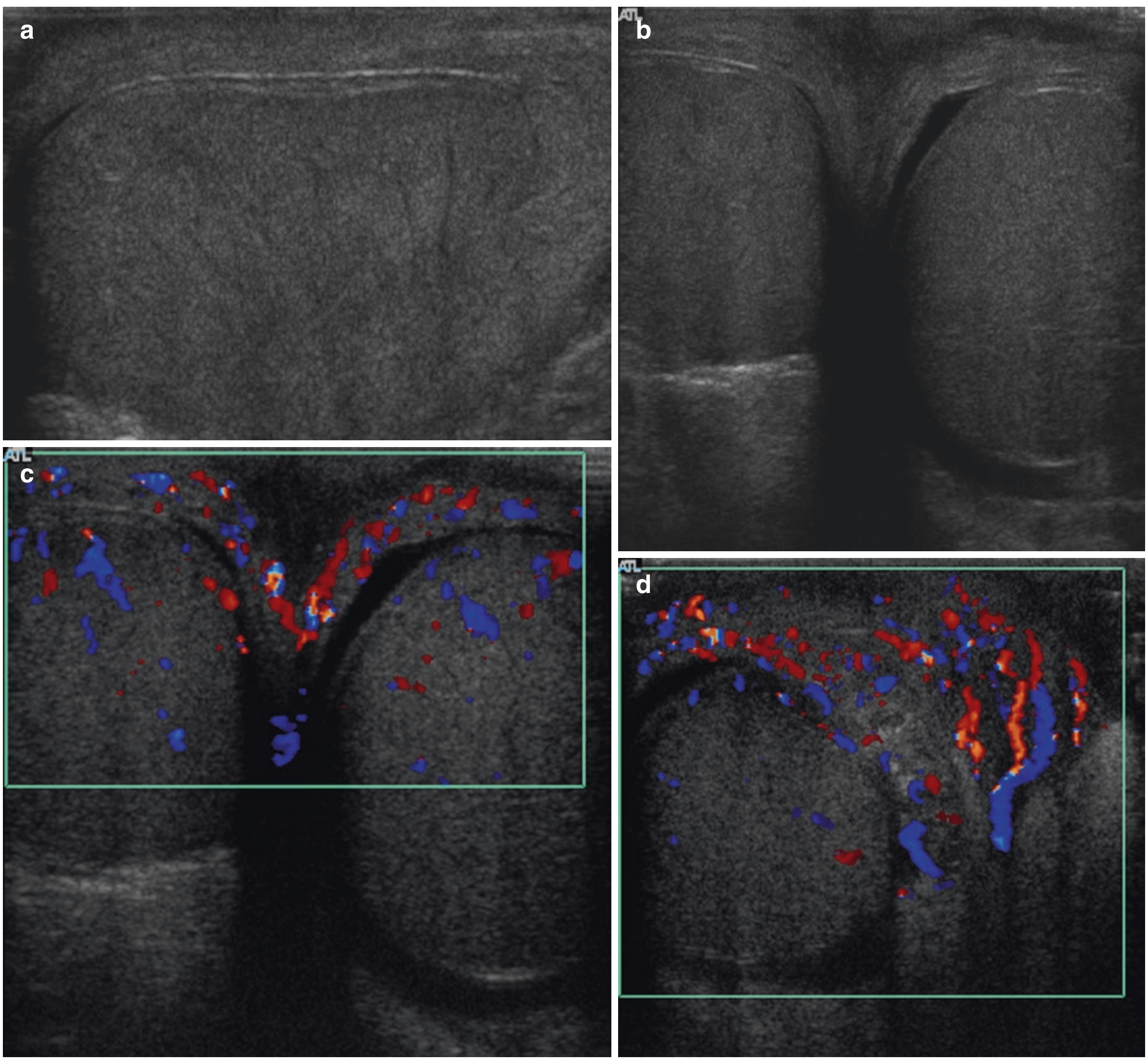
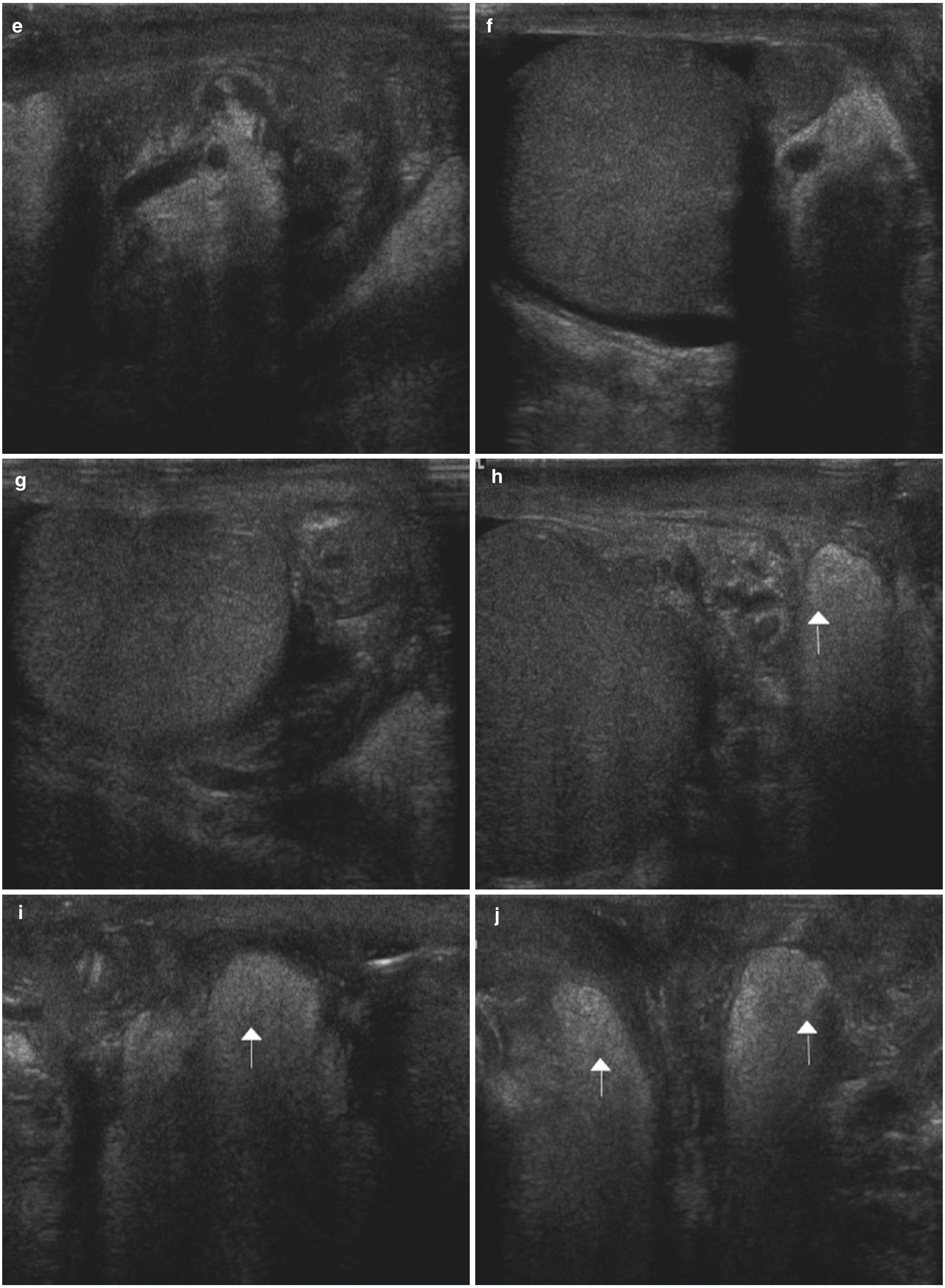


Fig. 5.30 Fournier's gangrene. A bedside ultrasound of a 48-year-old-man revealed diffuse scrotal oedema (**a**) and on colour Doppler increased flow to both testicles (**b**, **c**) with mildly enlarged epididymal heads bilaterally (**e**–**g**). In addition, focal collections of soft tissue gas were noted in the scrotum (**h**–**n**). Subcutaneous scrotal gas appears on US as ill-defined hyperechoic areas with posterior shadowing (*arrows*). Detection of scrotal wall gas is a pathognomonic feature of Fournier's gangrene



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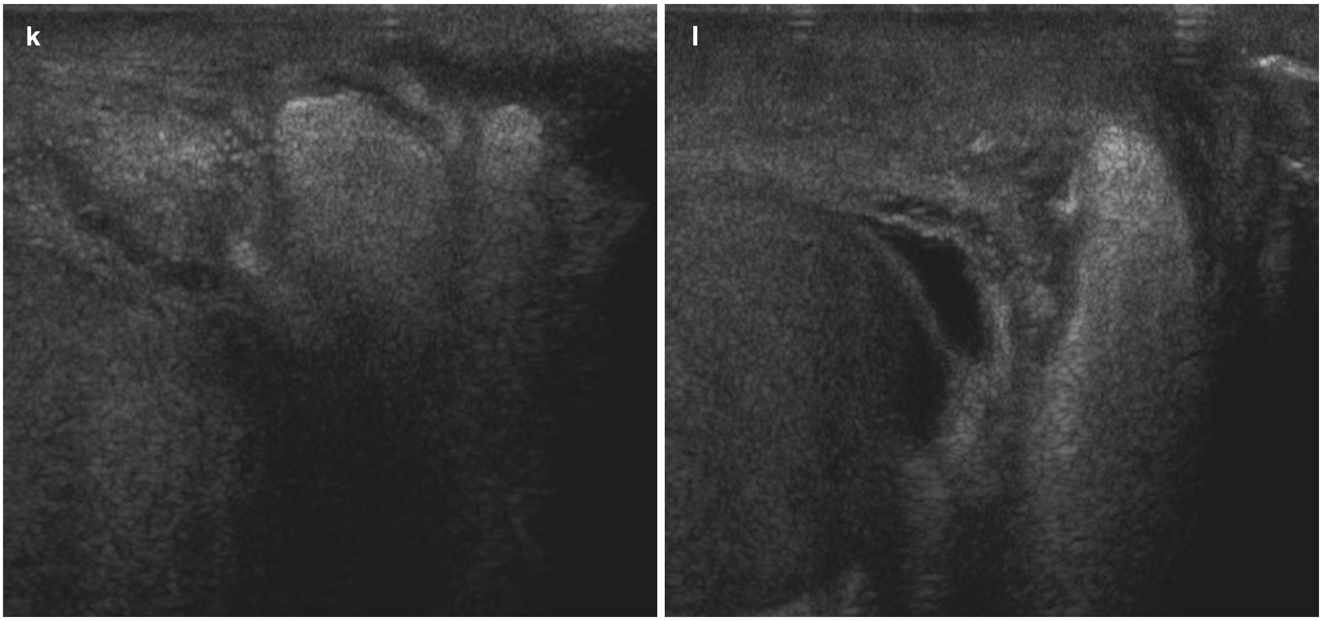

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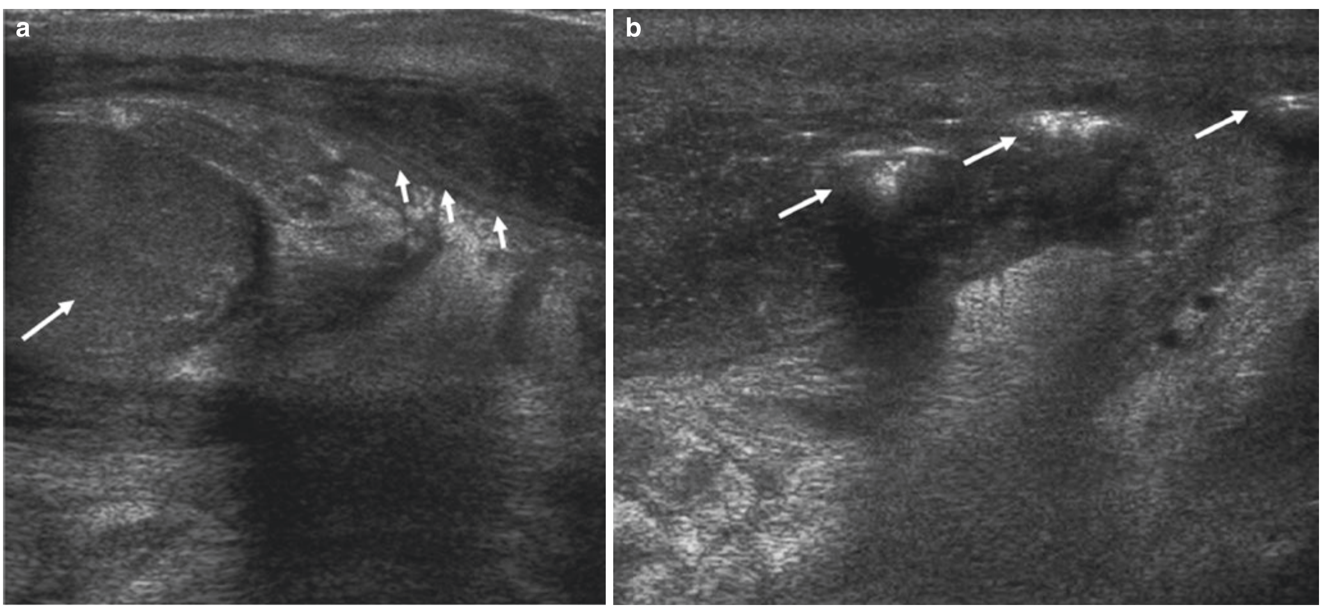

Fig. 5.31 Fournier's gangrene. Marked scrotal soft tissue thickening, with a pocket of low reflective fluid (*short arrows*) surrounding a normal testis (*long arrow*) (**a**). Multiple areas of high reflectivity (*long arrows*) causing the characteristic 'dirty shadowing' of Fournier's gangrene (**b**) (*From: V.R. Stewart., P.S Sidhu, "The testis: the unusual, the rare and the bizarre" Clinical Radiology (2007) 62, 289–302)*

Key Messages

- The great majority of cases of acute scrotum are due to trauma, torsion (of the testis or appendices) or epididymo-orchitis.
- Other rare causes are strangulated hernia, testicular tumour, haematocele and idiopathic scrotal oedema, Henoch-Schönlein purpura and scrotal fat necrosis.
- In prepubertal (or peri-pubertal) subjects, testicular or appendiceal torsion is the most frequent cause. In adults the main causes of acute scrotum are epididymo-orchitis and trauma. Nevertheless, torsion can occur at any age.
- When interpreting US in the setting of an acute scrotum, the operator should take into account any pre-existing alterations of the testicular echotexture and vascularisation. Medical history thus remains important prior to ultrasound examination.
- The differential diagnosis of acute scrotum relies on simultaneous assessment of clinical, US and laboratory findings. Body temperature, urinary sediment, leucocyte number and markers of inflammation are usually normal in torsion and are important criteria for its distinction from epididymo-orchitis. Clinical examination is often difficult and non-specific, as the scrotum may appear swollen and reddened.
- US appearance of testicular torsion is variable, depending on its duration and whether presenting in a pre- or post-pubertal testis.
- As a falsely negative US, evaluation can place testis salvage at risk, and given that US features can vary with time, we strongly recommend that US is performed at least twice in patients where the first examination seems to exclude torsion.
- Colour Doppler and power Doppler are the primary methods for diagnosing testicular torsion. Organ perfusion can be present initially, although significantly diminished, gradually disappearing as the oedema increases. Scrotal hyperaemia, also known as the 'rim sign', is the cause of one of the major pitfalls of Doppler ultrasound. The collateral blood supply to paratesticular tissues, which is reactively increased, should not be erroneously interpreted as blood flow in the testicular artery.
- In epididymo-orchitis the vascularisation of the testis is generally increased, in contrast with a subjectively decreased or absent vascularisation in torsion.
- However, incomplete, partial or intermittent torsion can demonstrate normal or increased flow (reactive hyperaemia) and is therefore a diagnostic dilemma.
- The contralateral testis can be used as the control for the original size, echotexture and vascularisation of the testicular parenchyma.
- Appendiceal torsion can be asymptomatic and accounts for less than 5% of acute scrotum in adults; however, it is nearly as frequent as testicular torsion in children.
- In testicular traumas, clinical diagnosis is often impossible because of marked scrotal pain and swelling, and colour Doppler ultrasound is therefore essential to establish the diagnosis, evaluate the extent of damage and predict possible complications.
- The role of US in testicular trauma consists of confirmation or exclusion of the testicular rupture, differentiation of soft tissue haematomas from haematoceles and follow-up of patients undergoing conservative therapy.
- An irregular or indistinct testicular contour is suggestive of testicular rupture, and a break in the continuity of the tunica albuginea confirms the diagnosis.
- Intratesticular haematomas can mimic testicular tumours; in these cases, ultrasound follow-up is indicated to reveal the typical temporal changes of haematomas. A challenging differential diagnosis is from complex intrascrotal haematoma and testicular rupture. Care should be taken to avoid misdiagnosing the two conditions, which require a different approach to treatment. The use of Doppler and colour flow Doppler imaging may aid in distinguishing the normal vascularised testis from a complex haematoma.
- The great majority of epididymo-orchitis cases originate with a predominantly epididymal involvement that later extends to the testis. An exception is viral orchitis (mumps orchitis), which primarily attacks the testicular parenchyma.
- Focal orchitis appears as an area of mixed echodensity in the testis, often indistinguishable from tumour by US alone.
- Ultrasound findings in Fournier's gangrene include marked thickening of the scrotal skin as well as discrete focal regions of high-amplitude echoes with posterior acoustic shadowing indicative of subcutaneous gas, which is pathognomonic for this condition.

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6 Testicular Varicocele

6.1 Testicular Varicocele

6.1.1 Definition and Aetiology

A varicocele is a dilation of the veins of the pampiniform venous plexus. Depending on the screening method, it is found in about 10–20% of the general male population, increasing to 40% in infertile men [1]. A high incidence of varicocele is also found in athletes (about 29%) [2]. Varicocele is ten times more common on the left than on the right side [3]. Their aetiology remains unclear but is probably multifactorial. The majority are due to incompetent valves in the testicular veins, while a minority result from obstruction of the spermatic vein. Secondary varicocele can occur in the presence of renal vein or inferior vena cava thrombosis, renal tumour and, only rarely, involvement of the testicular vein by retroperitoneal tumour or retroperitoneal fibrosis. Neoplasm is the most likely cause of non-decompressible recent/rapid onset varicocele in men over 40 years of age; it is frequently caused by a renal malignancy invading the renal vein [4, 5] (Fig. 6.1).

In younger subjects, varicocele is normally idiopathic and occurs on the left side. The left spermatic vein enters perpendicular to the left renal vein and is therefore subject to reflux. In contrast, the right spermatic vein enters obliquely into the inferior vena cava, and this difference offers some protection from varicocele on the right side. For this reason an expanding right varicocele should immediately raise the suspicion of external compression of the right spermatic vein or an internal obstruction.

The spermatic veins carry blood centripetally against gravity and have valves for this reason. Missing or incompetent valves in the spermatic veins have often been claimed to explain varicocele, but studies are inconclusive [3, 5]. In addition to the primary cause, retrograde flow into the internal spermatic vein leads to dilation and tortuosity of the pampiniform plexus [6].

6.2 Anatomy

The pampiniform plexus is a complex network of small vessels. Its veins normally range from 0.5 to 1.5 mm in diameter, with a main draining vein up to 2 mm in diameter [4]. It is formally divided into the anterior and posterior plexus. The former drains veins from the testicle and the epididymis and consists of 4–9 veins, with extensive anastomoses among themselves and with the cremasteric and deferential veins (posterior plexus). The veins of the anterior plexus converge into the spermatic vein cranially to the inguinal canal. The routine use of embolisation to treat varicocele has revealed that two or three retroperitoneal veins are not uncommon.

The posterior pampiniform plexus includes the deferential and cremasteric veins. These vessels drain into the internal and external iliac veins, respectively. The contribution of posterior plexus drainage in the pathophysiology of varicocele is minimal; however, since the anterior and the posterior plexus are highly interconnected, their contribution should be considered as a possible confounding factor in post-treatment assessment after varicocele repair.

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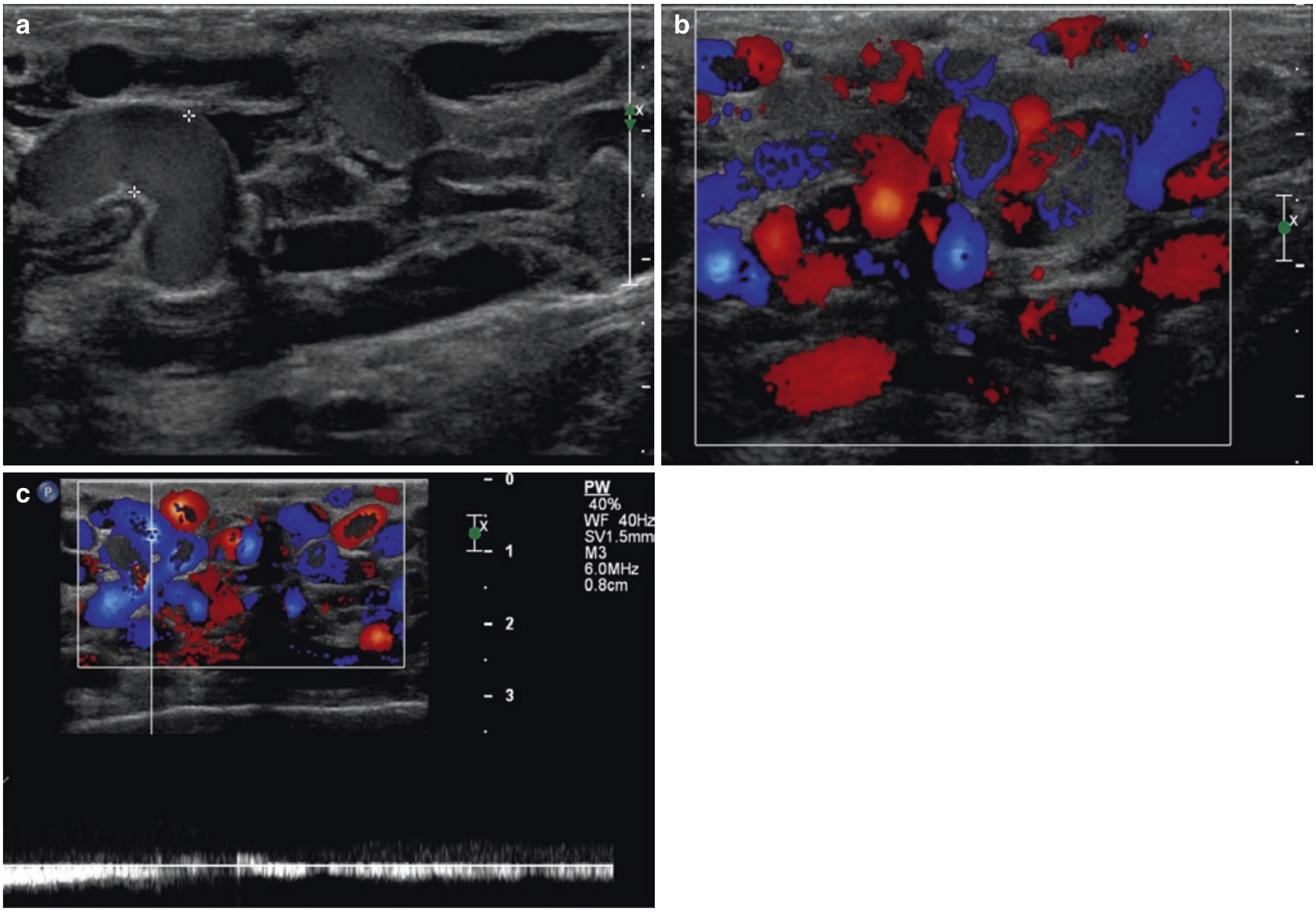

Fig. 6.1 Secondary, non-decompressible varicocele developed in a 65-year-old man due to the presence of a renal tumour invading the renal vein

6.3 Clinical Presentation

The clinical presentation of varicocele is variable. Clinical examinations tend to underestimate its severity and incidence. It is essential that screening for varicocele is performed with the patient standing up: erect posture and increased abdominal pressure lead to stasis and subsequent dilation of the venules of the pampiniform plexus.

Large varicoceles can be palpated in the posterosuperior aspect of the testis as a mass of tortuous veins, sometimes tender, that may feel like a 'bag of worms'. Normally, tenderness decreases with the patients lying down and is exacerbated by the Valsalva manoeuvre. If the varicocele is not reducible in the recumbent position or the patient reports its spontaneous occurrence, a reactive or secondary varicocele is likely and the presence of retroperitoneal lesions must be excluded.

Although clinical evaluation with the Valsalva manoeuvre is a simple, non-invasive test, clinical examination is not without its limitations. Clinical assessment is highly subjective and suffers from low sensibility and specificity, especially in cases of low grade varicocele. The high rate of both false-positive and false-negative diagnosis [7] makes it imperative that physicians acquire extensive experience with the features of varicocele before attempting diagnosis and staging [6].

Clinical examination may also be complicated by concomitant hydrocele, cremasteric contraction, temperature of the examination room, relative volume of the testes, inguinal adiposity and most importantly by the doctor's experience. Both over- and underestimation of the severity of varicocele are frequent, especially the latter.

Emptying of the varicocele in the supine position distinguishes between those due to reflux and those caused by obstruction. For this reason, we normally recommend that patients are first examined in the upright position and then lying down.

Occasionally, a varicocele may cause discomfort and scrotal swelling. The reported feeling of a dull scrotal or inguinal heaviness that increases after a few hours' standing up is suggestive of varicocele. The clinical differential diagnosis is against large inguinal hernias, subacute/chronic epididymitis and testicular malposition.

6.4 Classification of Varicocele

The impact of varicocele and its treatment in male infertility has been a matter of debate for the past 20 years and is not yet resolved. The discrepancies among the several small, inadequately controlled studies in the literature depend on five major issues [6]: (1) there is no unequivocal correlation between the size of the varicocele and the degree of sperm quality impairment; (2) in some patients sperm quality improves after varicocele ablation, but in others it remains the same, due to other concomitant disorders; (3) most studies do not assess whether the varicocele repair was actually successful; (4) the final outcome of delivered pregnancies is strongly affected by female factor infertility; (5) most studies used a clinical rather than ultrasound (US) classification of varicocele; therefore accuracy in staging of the disease cannot be compared objectively.

The clinical and US classification of varicocele are reviewed below.

Dubin and Amelar [8] devised a clinical grading system for varicocele that has the advantage of being universally accepted and easy to use. Grade 1 varicoceles are palpable only during the Valsalva manoeuvre; grade 2 are readily palpable without the need for the Valsalva manoeuvre; and grade 3 are visible on inspection (Fig. 6.2) [6, 9].

Varicoceles greater than 3–4 mm in diameter are usually clinically apparent as a bluish 'bag of worms' surrounding the testis.

Despite the clear advantages of Dubin's classification, US staging of varicocele offers additional information that may in turn have more clinical implications than initially thought. Using greyscale and colour Doppler US, a number of clinical and pathophysiological situations can be identified, which could help better characterise patients with varicocele and identify those who may actually benefit from treatment. Clearly, screening for varicocele remains essentially clinical and is performed by physical examination. However, if there

Fig. 6.2 Inspection of varicocele. Clinical grade 3: readily visible on inspection

Table 6.1 The proposed integrated and revised classification offers a compromise between the clinical (Dubin) and US classification

		Revisited classification (Dubin-Solbiati)	Corresponding to Dubin	Solbiati (1995)	Corresponding Dubin
	B-mode	Reflux		Reflux	
Grade 1	Dilated vessel (>2.5 mm) in inguinal region only	Inguinal reflux only during Valsalva manoeuvre (lasting 2–3 s)	Grade 1	Inguinal reflux only during Valsalva manoeuvre	Grade 1
Grade 2	Supra-testicular vessel dilation (>3 mm)	Supra-testicular reflux only during Valsalva manoeuvre, lasting more than 3 s		Supra-testicular reflux only during Valsalva manoeuvre	
Grade 3	Supra- and peritesticular vessel dilation (>3 mm)	Supra- and peritesticular reflux at rest which increases during Valsalva manoeuvre, lasting more than 3 s	Grade 2	Peritesticular reflux only during Valsalva manoeuvre	
Grade 4	Peritesticular vessel dilation with further dilation during functional manoeuvre, testicular hypotrophy	Peritesticular reflux at rest which may or may not increase during Valsalva manoeuvre	Grade 3	Testicular reflux at rest which increases during Valsalva manoeuvre	Grade 2
Grade 5	Peritesticular vessel dilation that does not increase with functional manoeuvre or intratesticular vessels and testicular hypotrophy	Peritesticular reflux at rest which increases minimally during Valsalva manoeuvre or dilated intratesticular vessels which refill with Valsalva manoeuvre		Peritesticular reflux at rest which increases minimally during Valsalva manoeuvre	Grade 3

In fact, some clinical grade 1 varicoceles could correspond to US grade 2 and 3 (according to Solbiati), especially in inexpert hands. This situation could create some confusion, considering that Dubin's classification is still largely used. In contrast, in our revisited classification, US grade 3 unequivocally corresponds to clinical grade 2. Similarly, US grades 4 and 5 should be confined only to very severe varicoceles that are readily visible and therefore correspond to Dubin grade 3. Briefly, the Solbiati classification offers a more complete stratification of lower grades (Dubin grade 1) but is skewed on the high grades. Our classification is closer to the clinical grading and should thus create less confusion for general practitioners

is any clinical doubt about the diagnosis or the cause, imaging should be requested.

A significant step forward in varicocele characterisation comes from Solbiati [10] and more recently Sarteschi and colleagues [11]. These authors suggest that varicocele should be defined on the basis of the following US features: extension, size and number of dilated veins, affected side, duration of retrograde flow during Valsalva, presence of spontaneous retrograde flow in the upright position, volume and echotexture of the affected testicle (in comparison to the contralateral) and demonstration of retrograde flow in the inguinal canal of the affected side (Table 6.1) (Fig. 6.3).

Both the greyscale and colour Doppler duplex modes must be performed to evaluate varicocele correctly. We strongly discourage an assessment made on the basis of Doppler US alone.

The dilated veins of a varicocele appear as multiple, serpiginous, tubular over 2.5 mm in diameter (Fig. 6.4). On US they resemble a tortuous multicystic collection, usually best seen adjacent or proximal to the upper pole of the testis and epididymis or lateral to the testis. Large varicoceles can extend posterior and inferior to the testis (Fig. 6.5).

The vascular nature of these hypo- or anechoic oval structures must be confirmed by colour Doppler duplex examination. Duplex or colour flow Doppler is necessary to distinguish varicocele from spermatocele or other cystic lesions (Fig. 2.11). Similarly, dilated veins in the mediastinum testes can be distinguished from tubular ectasia of the rete testes by the use of colour flow or pulsed wave Doppler US (Fig. 2.13) [4].

Most refluxing varicoceles show little spontaneous flow on colour Doppler, because blood flow is very slow and below the threshold of the Doppler system (Fig. 6.6). A Valsalva manoeuvre or a cough will cause retrograde flow in a refluxing varicocele, which fills with colour (Fig. 6.7). Retrograde flow may not always be identified in the supine position and may require the patient to be examined when standing, with and without the Valsalva manoeuvre. It is often useful to wait a few minutes before evaluating the patient in the standing position, to allow the varicocele to fill – some varicoceles only become apparent in this position. Whether the reflux is present in the upright position only or also in the recumbent position must be specified.

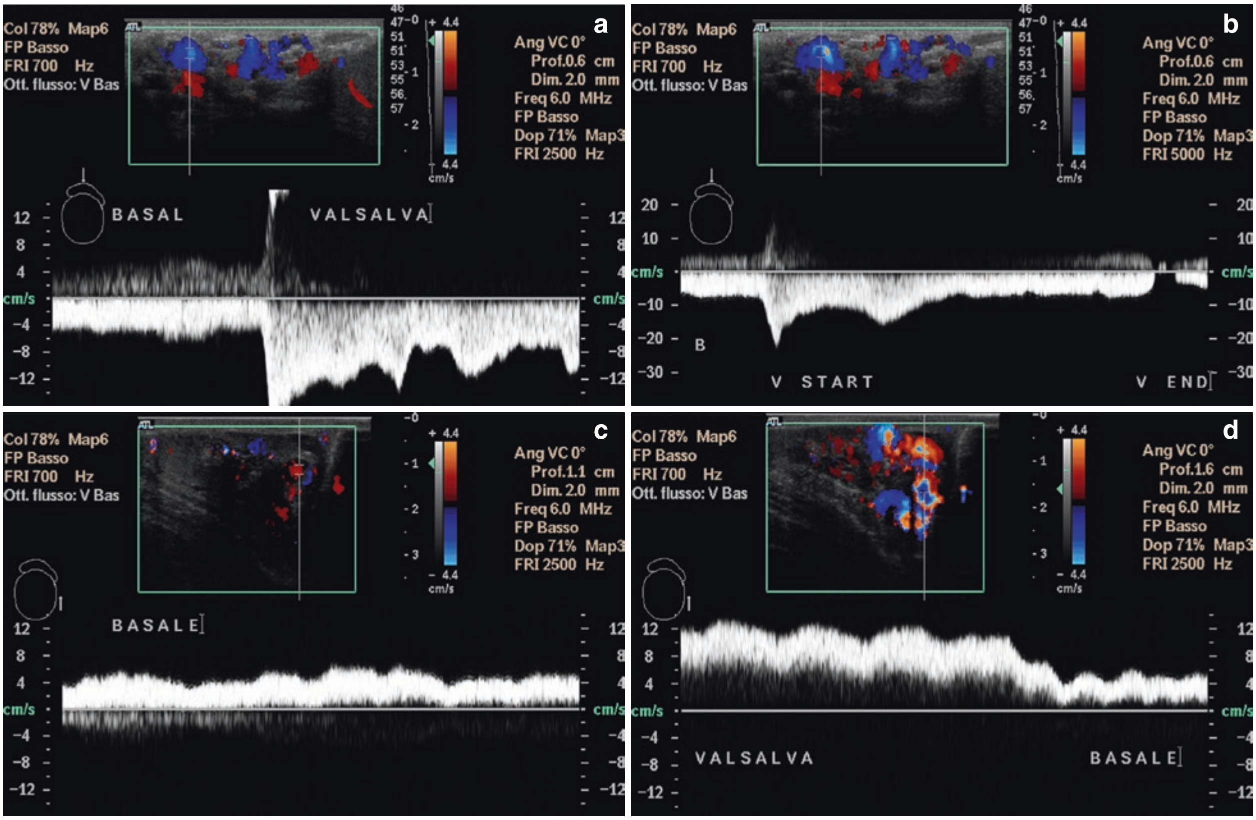

Fig. 6.3 Varicocele is characterised by retrograde filling of the veins of the pampiniform plexus. Images (**a**–**d**) show two patients with a US grade 3 varicocele, characterised by peritesticular reflux at rest which increases during Valsalva

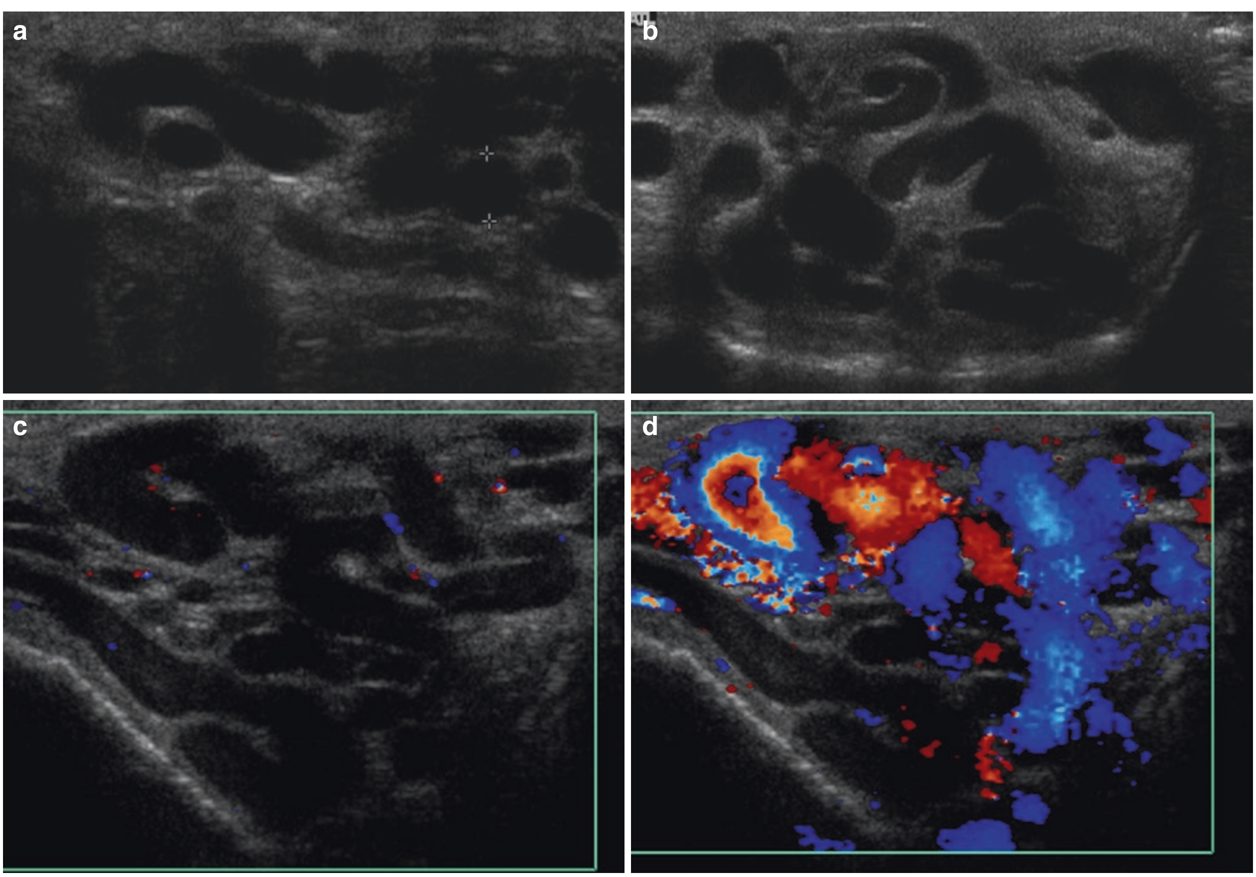

Fig. 6.4 The **dilated veins of varicocele** appear as multiple, serpiginous, tubular structures larger than 2.5 mm in diameter (**a**–**c**), showing retrograde flow at rest and/or during Valsalva manoeuvre on colour Doppler study (**d**)

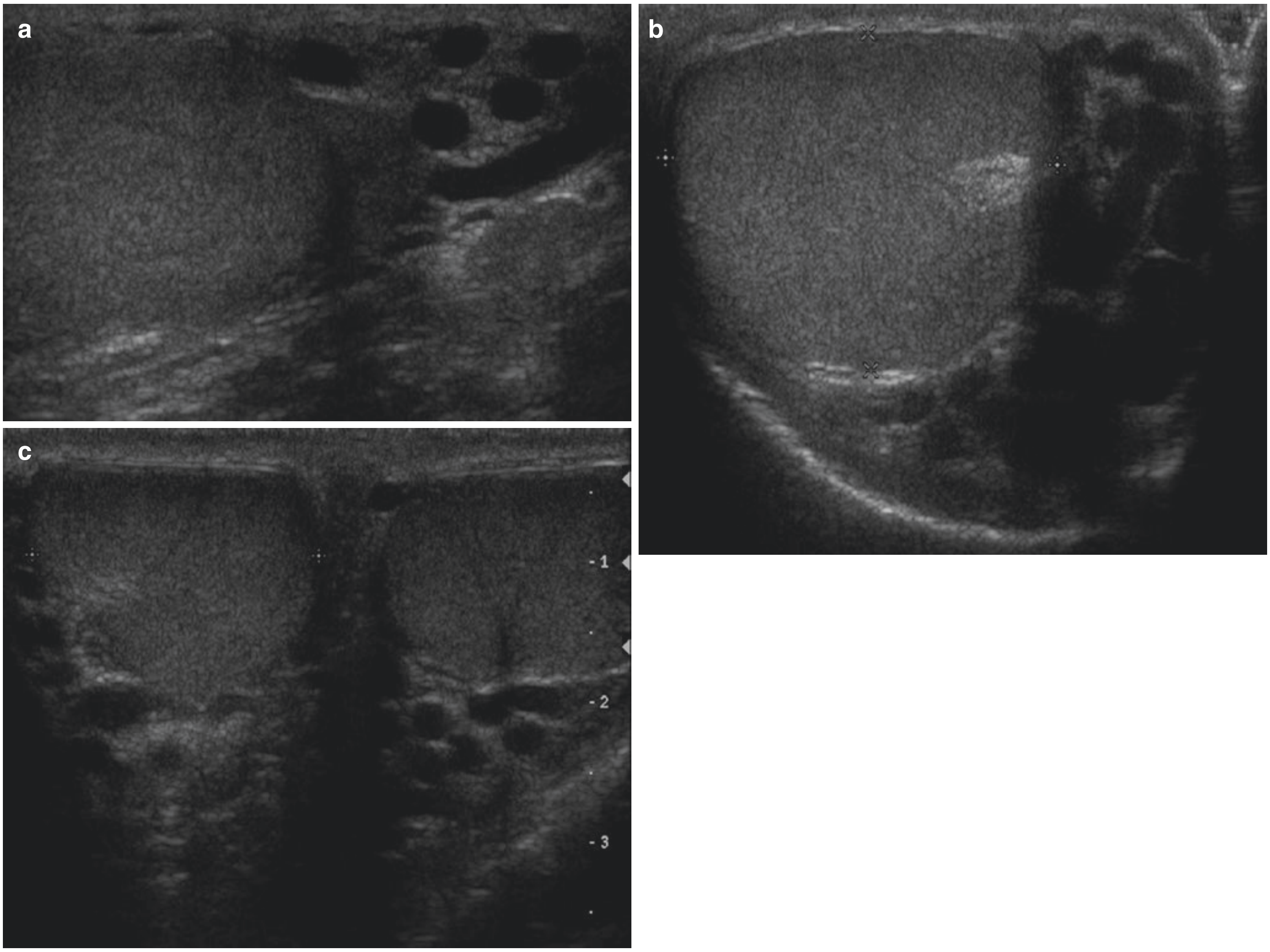

Fig. 6.5 The **dilated vessels** are usually better visualised adjacent or proximal to the upper pole of the testis and epididymis (US grade 1–2 varicocele), lateral to the testis (US grade 2–3 varicocele) or posterior and inferior (US grade 4–5 varicocele) to the testis (US classification)

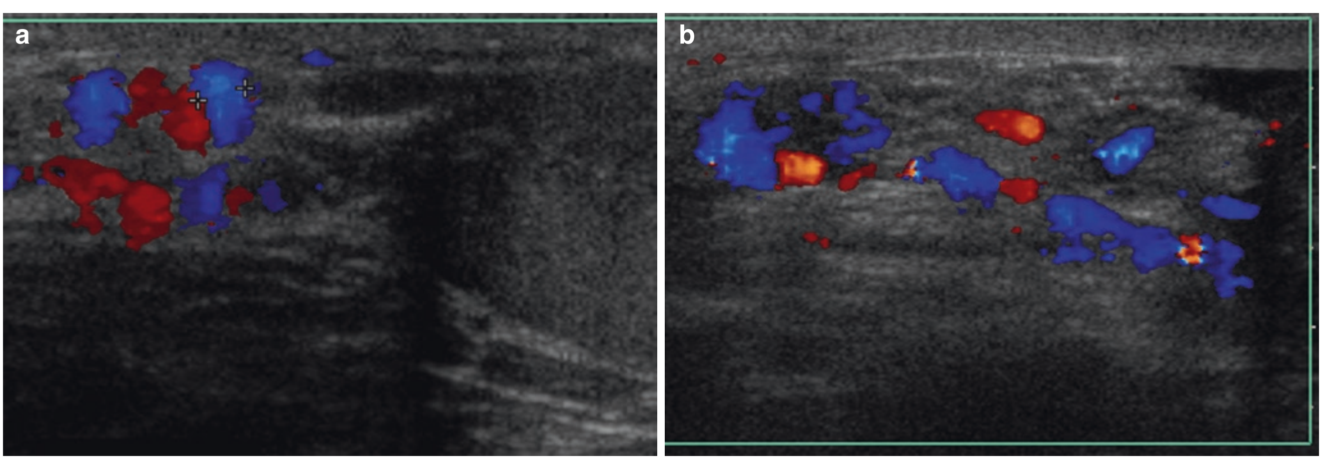

Fig. 6.6 Longitudinal scans of the upper pole of the testis show few, hypoechoic, dilated veins with little spontaneous flow on colour Doppler (**a**, **b**)

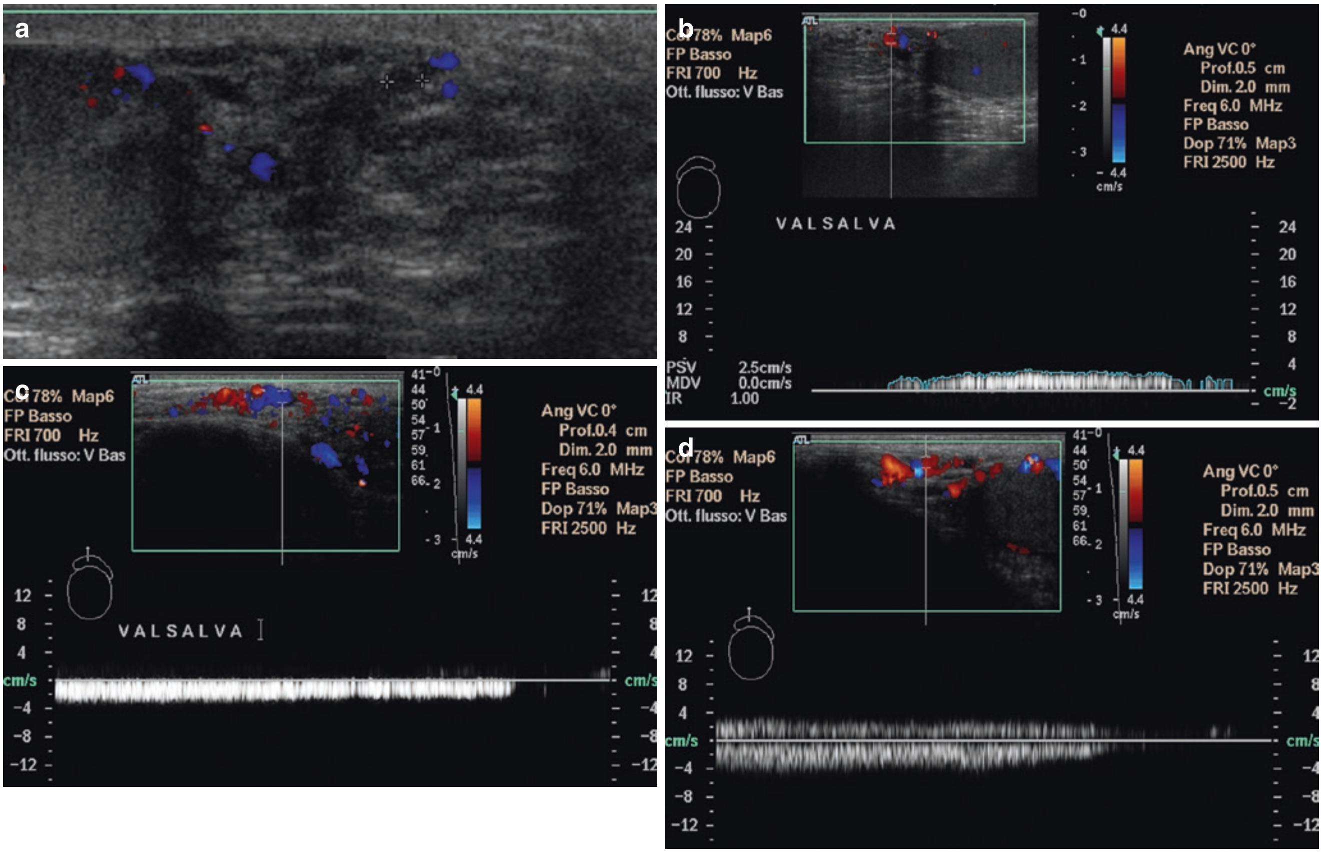

Fig. 6.7 A Valsalva manoeuvre or a cough causes retrograde flow in a refluxing varicocele, which fills with colour. A retrograde flow lasting less than 3 s is detected in these vessels during the Valsalva manoeuvre (US grade I varicocele) (**a**–**d**)

Retrograde flow patterns at rest and during the Valsalva manoeuvre are complex. Figure 6.1 shows all the possible pictures seen at Doppler evaluation in the presence of dilated veins.

It is important to use a high-frequency probe and a machine with good sensitivity to low flow rates. We recommend the use of a fixed PRF value that is set sufficiently low to detect venous flow but high enough to avoid movement artefacts created during the Valsalva manoeuvre. Changes to the CDU settings should be minimised to enable a more consistent appraisal of the severity of varicocele among various patients, prevent inconsistencies at follow-up evaluations, enable a more precise assessment of the treatment's effect and reduce artefacts. This recommendation is also supported by the existence of cases in which no flow can be detected on duplex US despite markedly dilated vessels. In these cases, the operator should refrain from reducing PRF and enhancing the colour gain to document spurious retrograde flow. We suggest they be described as dilation of the pampiniform plexus with partial or complete continence of the venous reflux.

As with any abnormality found on scrotal US, the features of the varicocele should be described first and only then should it be graded or scaled, if necessary. This is even more important given that different classifications have been proposed and confusion may arise if the operator fails to specify the scale used.

A complete description of varicocele should answer all the following points.

1. **Are the vessels of the pampiniform plexus enlarged?** Dilated veins appear on the greyscale as >2.5 mm hypo- or anechoic convoluted oval or tubular structures. Estimation of the average diameter is essential, and a description of their number, whether numerical (>5 or >10 dilated veins) or generic (few, several), should be provided. A finding of three or fewer vessels with an average diameter less than 2.0 mm should be considered as normal. A diagnosis of subclinical varicocele can be made only if the latter case is accompanied by a prolonged retrograde flow that is detected during the Valsalva manoeuvre.
2. **What is the extent of this dilation?** Dilation can be confined to the upper tract of the spermatic cord or extend down along the cord to surround the whole posterior aspect of the testis and its inferior pole

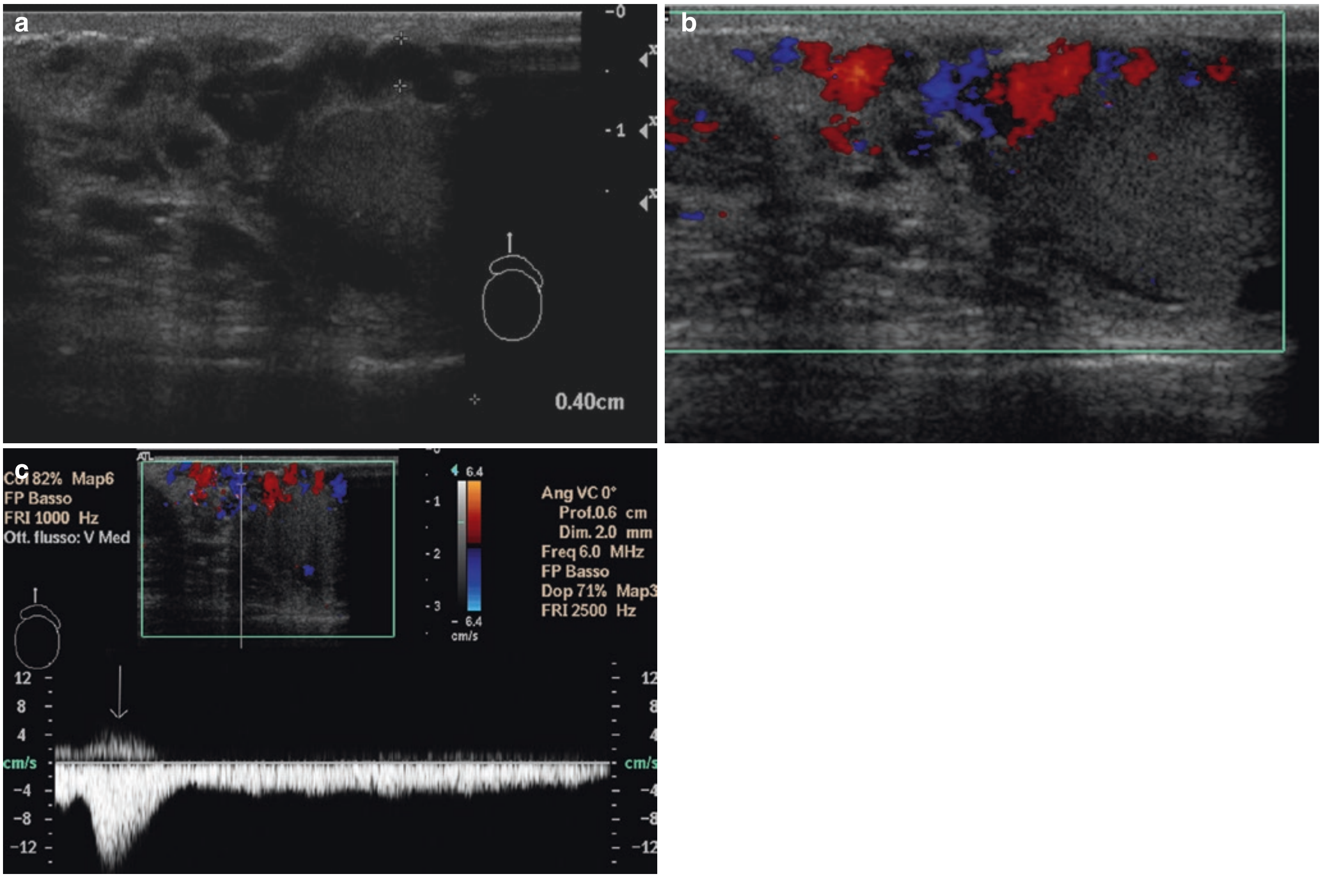

Fig. 6.8 B-mode localisation of dilated vessels is important: veins may be confined to the upper tract of the spermatic cord or extend inferiorly along the cord. In this patient, the vessels can be found surrounding the testis, are >4 mm in size and fill with colour only during the Valsalva manoeuvre. This can be classified as a **US grade 2 varicocele**. Note the impulse when the Valsalva manoeuvre begins (*arrow*)

(Fig. 6.8). The extension is generally correlated to the size of the vessels (Fig. 6.9).

3. **Is there any retrograde flow during the Valsalva manoeuvre?**
Under normal conditions there is only a slow venous flow in a cranial direction. Varicoceles are characterised by retrograde filling of the veins during the Valsalva manoeuvre. The reflux is quantified as permanent (>6 s), which is indicative of a varicocele, intermittent (2–5 s) or brief (<2 s, which is a physiological finding). Figure 6.10 shows all the possible pictures seen at Doppler evaluation in the presence of dilated veins. Intermittent reflux is an area of debate and could be insignificant if there is no palpable varicocele [6]. The pampiniform plexus should be visualised in the longitudinal plane (we recommend a longitudinal rather than a transverse plane scan for the colour-coded duplex US), the sample volume is located on a dilated vessel and the patient is asked to perform the Valsalva manoeuvre for at least 5 s (preferably 10–15 s). The patient is asked to increase abdominal pressure progressively, avoiding abrupt contractions. If a varicocele is present, there is a progressive and sustained retrograde flow in the vessels (Fig. 6.11). The Doppler signal detected in the first 1–2 s is generally an artefact due to a rebound effect of blood filling the intra-abdominal vessels (Fig. 6.12). In contrast, the blood flow detected at the end of the Valsalva manoeuvre, when the patient relaxes the diaphragm, is secondary to the negative pressure developing in the abdominal cavity, which exerts suction on the blood filling the pampiniform plexus. The latter is an anterograde flow and should therefore be considered a normal finding (Fig. 6.13). Evaluation of retrograde flow during the Valsalva manoeuvre should be assessed both in the recumbent and standing position.
4. **Is there any retrograde flow at rest?**
Once the venous dilations have been located and a retrograde flow documented during the Valsalva manoeuvre, the presence of spontaneous reflux should be assessed. Spontaneous retrograde flow is easily documented with the patient standing (Fig. 6.14). A transient reflux can be detected in nearly every patient with visible dilations

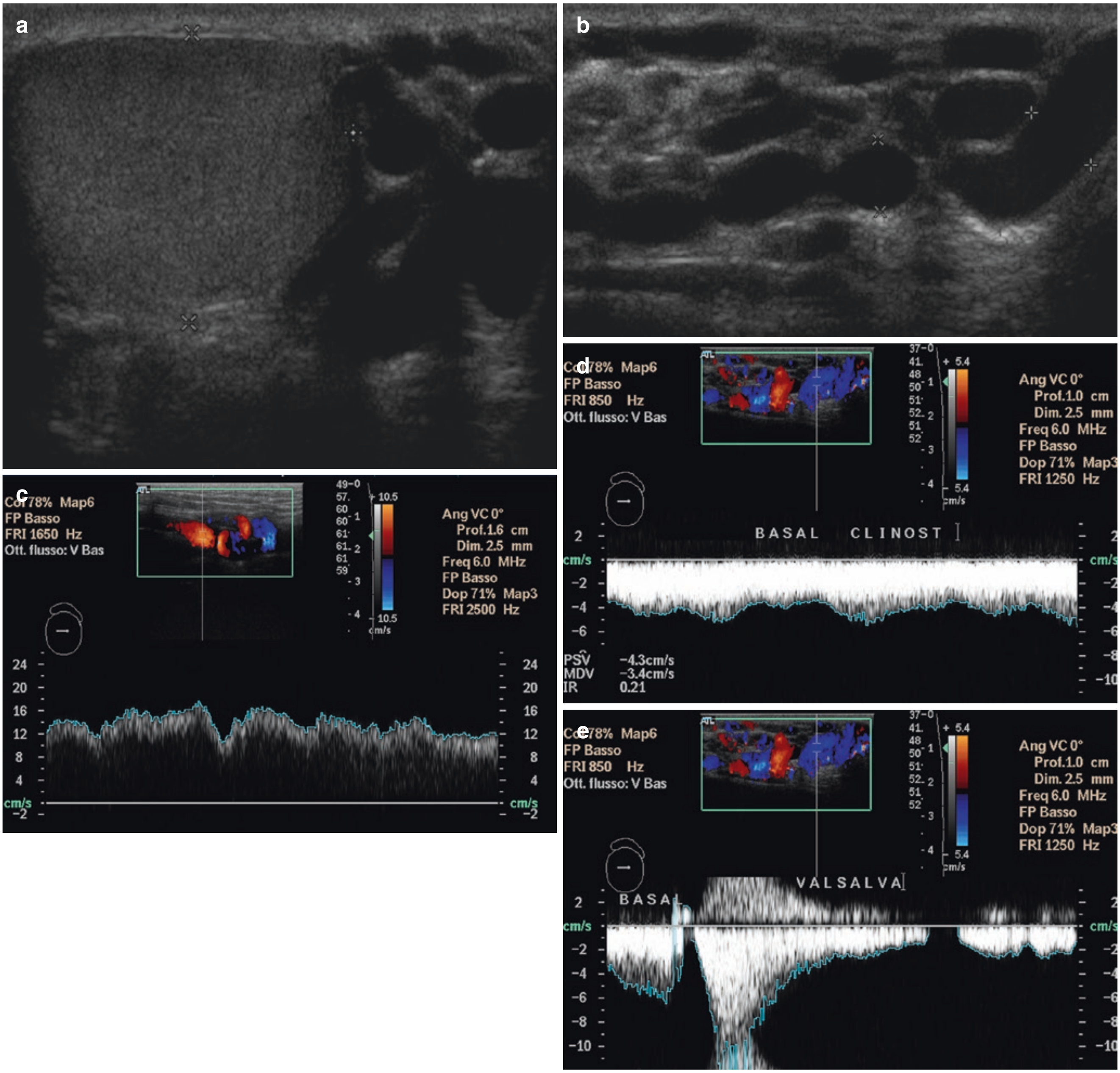

Fig. 6.9 In this case the dilation of the vessels is more pronounced. The veins surround the whole posterior aspect of the testis and its inferior pole. In supine position, at rest, there is a pathological venous flow, which decreases during the Valsalva manoeuvre (**US grade 4 varicocele**)

immediately after shifting from the recumbent to the standing position. Although this may provide some clinical information, we recommend waiting at least 3 min before measuring flow or asking the patient to perform the Valsalva manoeuvre. When spontaneous reflux is present, the operator should describe to what extent this is detectable.

5. **Is the reflux detectable at the inguinal level?**
   When a significant flow is present in the pampiniform plexus, then it must also be evaluated at the inguinal canal, bilaterally. Detection of reflux in the right inguinal canal is the only reliable way to confirm the presence of a right varicocele. In all other cases, it is very difficult to confirm that dilations seen on the right of the scrotum are not veins from the left. Moreover, large left varicoceles can also supply the right venous plexus through communicating vessels. In the latter case, repair should be attempted on the left side only. In the case of a true right (bilateral) varicocele, reflux should be clearly seen in the right inguinal canal.

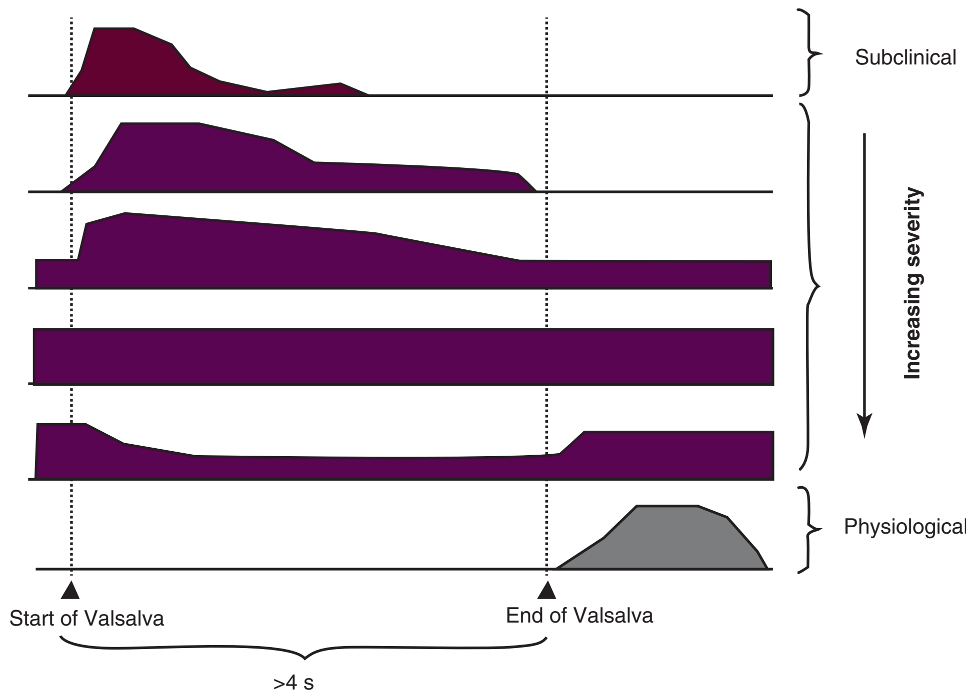

Fig. 6.10 Retrograde reflux in the pampiniform plexus at rest and during Valsalva (between lines): all possible scenarios

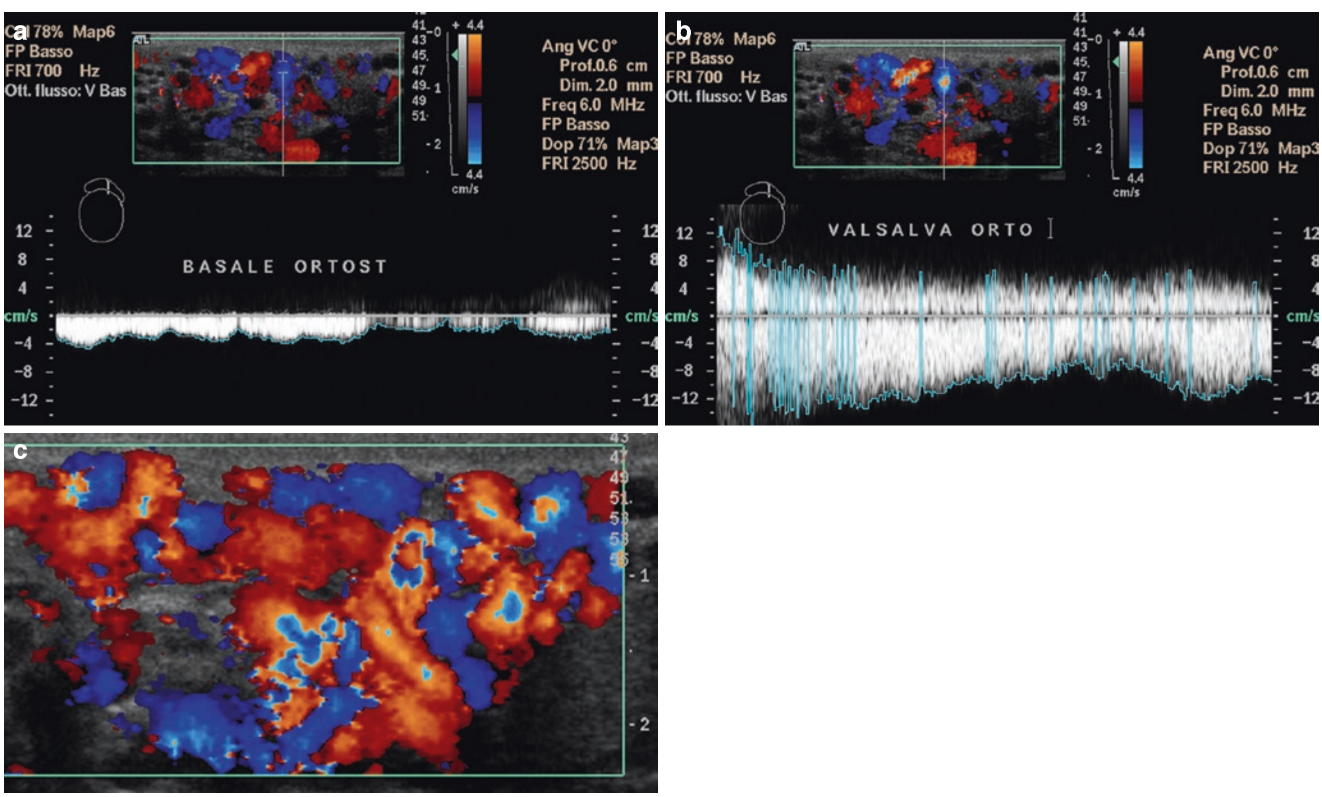

Fig. 6.11 During the examination, the patient is asked to increase abdominal pressure gradually, avoiding any abrupt contraction. If varicocele is present, there is a progressive and sustained retrograde flow in the vessels. Panel (**b**) shows an example of the **aliasing effect** that occurs when low pulse repetition frequencies or velocity scales are used and high velocities are encountered. This patient had a US grade 3–4 varicocele, characterised by a basal spontaneous reflux, which increases with the Valsalva manoeuvre

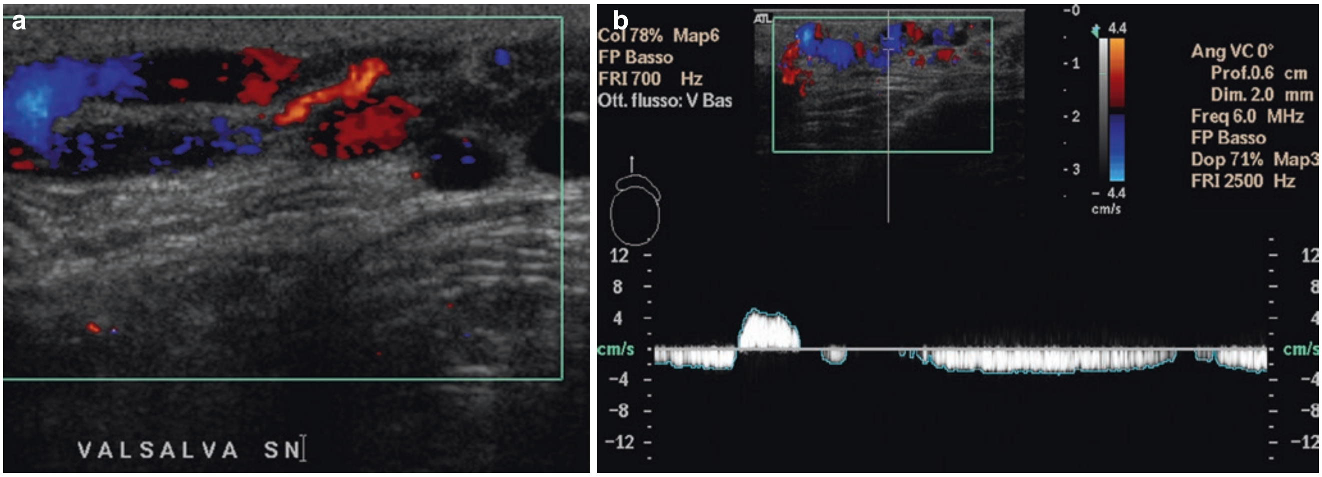

Fig. 6.12 A marginal reflux detectable only during Valsalva manoeuvre. Note the **sign oscillations** of the reflux during Valsalva manoeuvre due to the inconsistency of abdominal contraction

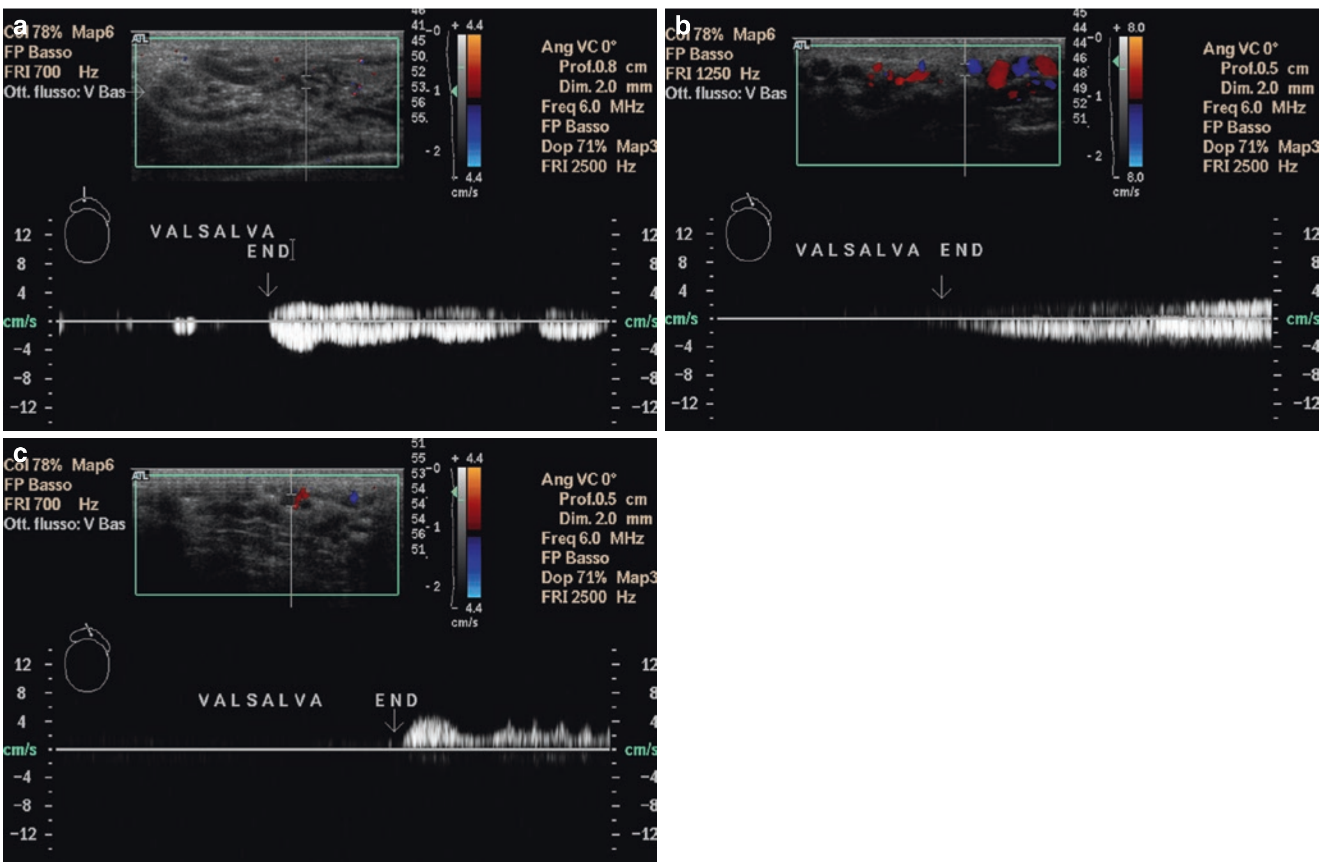

Fig. 6.13 Occasionally, blood flow signal can be detected only **at the end of the Valsalva manoeuvre** (**a–c**, *arrows*). This happens when the patient relaxes the diaphragm, and secondary to the negative pressure developing in the abdominal cavity, suction is exerted on the blood filling the pampiniform plexus. This is a physiological finding

6. **How is spontaneous flow affected by the Valsalva manoeuvre?**
   When spontaneous reflux is present (while the patient is standing), its flow rate can increase, remain stable or decrease during the Valsalva manoeuvre. These three possibilities are likely to reflect progressive stages of the disease. In large varicoceles, the vessels are already stretched to their maximum, completely filled with blood and almost directly continuous with the abdominal venous system: in such cases diaphragmatic contraction does not

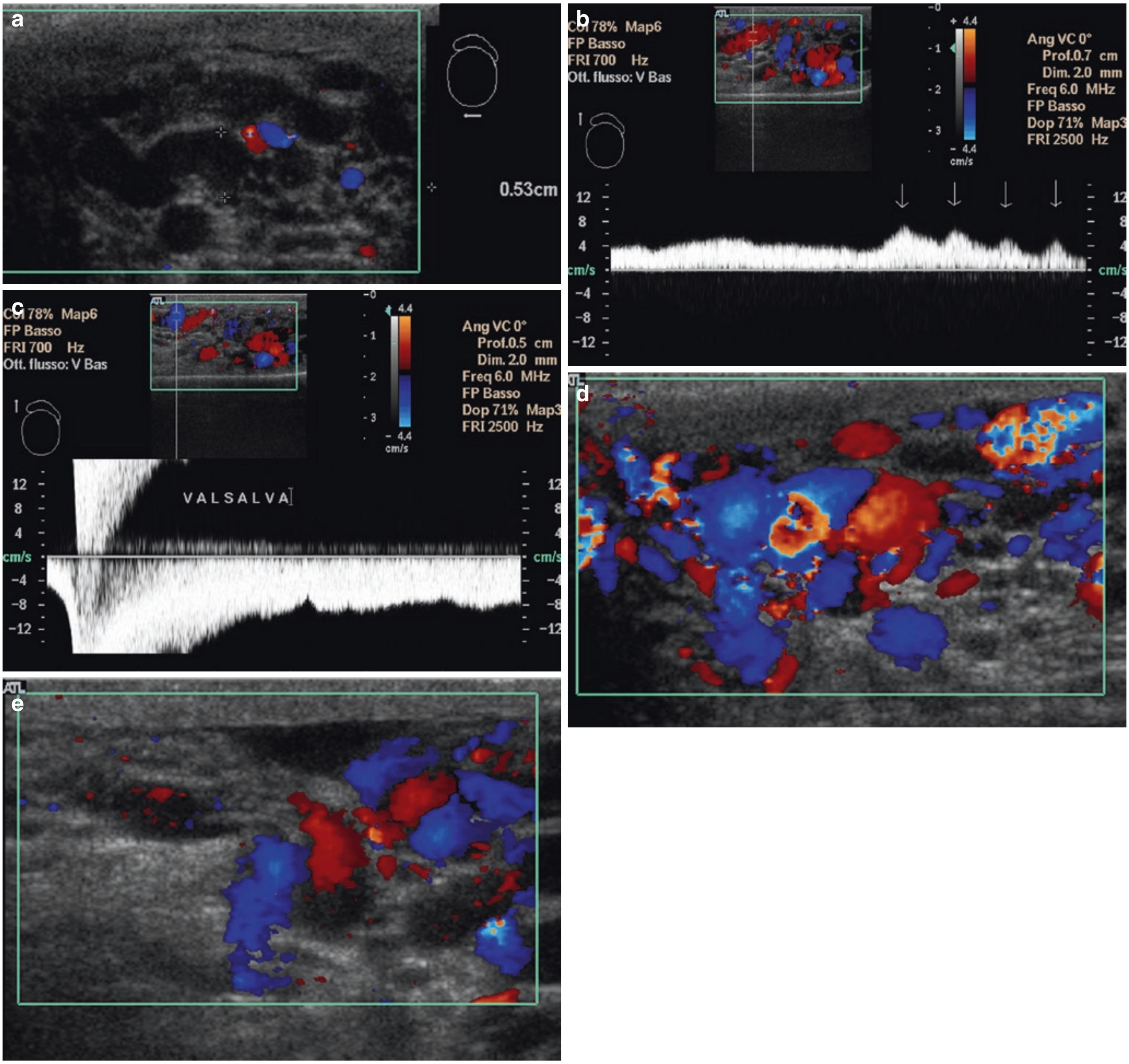

Fig. 6.14 Spontaneous retrograde flow is easily documented with the patient standing. In panel (**b**), note the **impulses** (*arrows*) obtained by asking the patient to count out loud

further affect the magnitude (rate) of the reflux. In younger, thin subjects, a strong contraction of abdominal and inguinal muscles can produce a mechanical closure of the inguinal canal that is reflected in an apparent reduction in reflux (Fig. 6.15).

7. **What is the clinical meaning of enlarged vessels without reflux?**
   A minority of men present an increased number of dilated vessels that fail to increase their calibre or show any retrograde flux during the Valsalva manoeuvre. In these men, an orthodromic flow (directed towards the abdomen) can be seen at the end of the Valsalva manoeuvre. The clinical significance of this condition remains unknown. If reflux is absent, there must be some sort of 'continence' of the spermatic veins (venous valves?). It remains unclear why the scrotal veins are dilated and increased in number.
8. **Distinguishing between the anterior and posterior plexus**.
   Although a distinction between varicoceles of the anterior or posterior plexus is theoretically possible, through identification of the deferent duct and its artery, there is no real distinction between a reno-spermatic and an iliac-spermatic varicocele, as (a) there is still uncer-

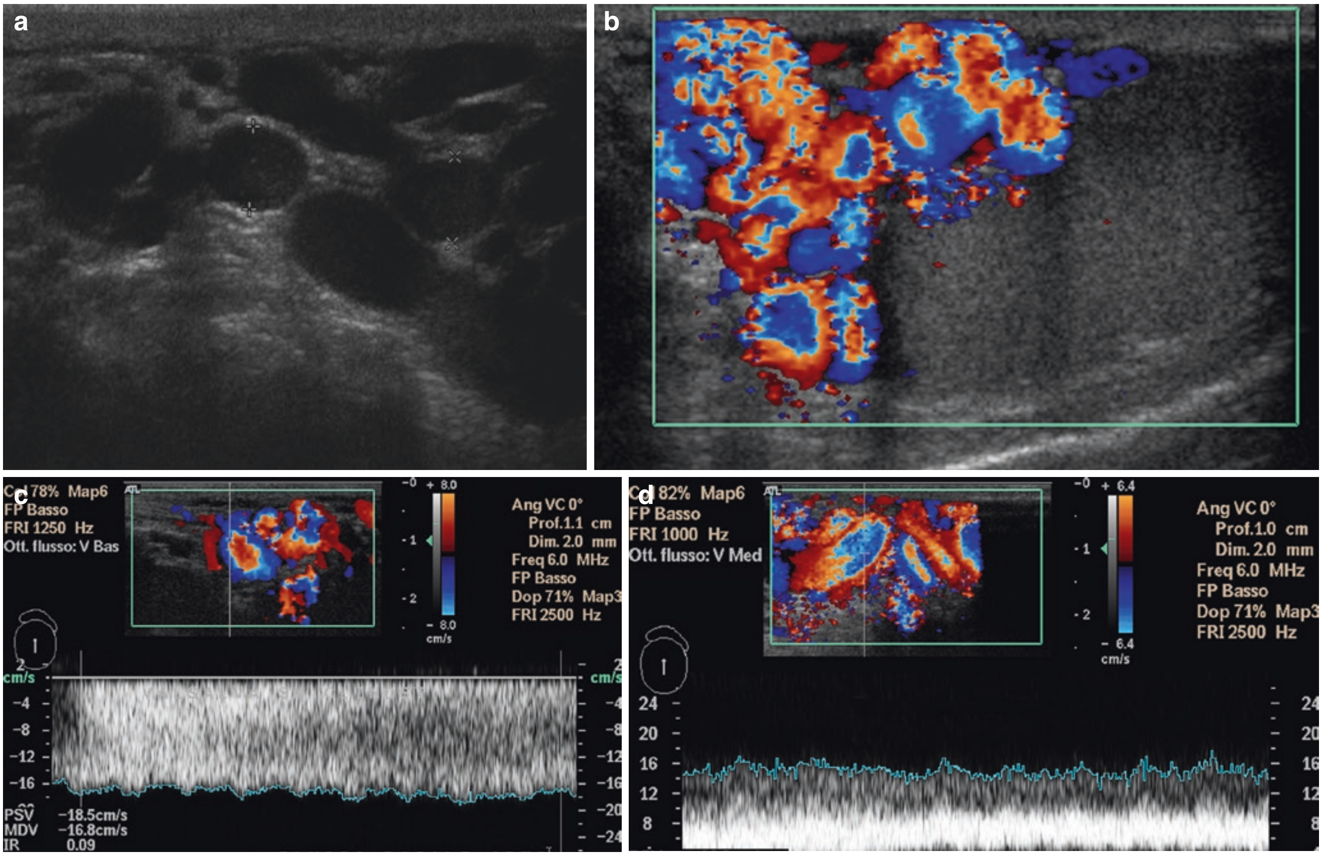

Fig. 6.15 In large varicoceles the vessels are already stretched to their maximum, completely filled with blood and in almost direct continuity with the abdominal venous system: in such cases diaphragmatic contraction does not affect further the reflux magnitude (velocity). This condition is typical of **US grade 5 varicocele**

tainty as to whether the posterior plexus is itself contributing to the development of varicoceles and (b) there are known anastomoses between the two aspects of the plexus.

9. **Post-treatment evaluation**.
US scans are frequently requested to establish the success of varicocele repair. However, they can be misleading as in a significant number of cases, a high performance machine will still detect some colour flow in the dilated vessels (Fig. 6.16). It is important to establish the direction of the flow. In most cases, flow is towards the abdomen and readily increases at the end of the Valsalva manoeuvre. As said above, this could be considered a non-pathological finding. In other cases a true retrograde flow can still be detected but in fewer veins than before the treatment. These cases are likely to produce a recurrence of varicoceles in the long term, normally within 12–18 months of repair. Possible complications of surgical ligation include hydrocele, infections and testicular ischaemia. Embolisation can occasionally be associated with diffusion of the sclerosing agent in the spermatic cord. This may produce a reactive chemical funiculitis, clinically appreciable as a firm enlargement of the cord, that may take several months to resolve.

The responses to these points are the elements needed to correctly classify varicocele using ultrasound. Clearly, US and clinical grading of varicoceles differ, which can create some confusion. Table 6.1 proposes a revised, integrated classification of varicoceles that offers a compromise between the clinical (Dubin) and US classification (Solbiati). In fact, comparison of the latter reveals that some clinical grade 1 varicoceles correspond to Solbiati's US grade 2 and 3, especially in inexpert hands. This situation could create some confusion, considering that Dubin's classification is still largely used. In contrast, in our revised classification, US grade 3 unequivocally corresponds to clinical grade 2. Similarly, US grades 4 and 5 should be attributed only to very severe readily visible varicoceles corresponding to Dubin's grade 3 (as unequivocally occurs in our classification). In short, the Solbiati classification offers a more complete stratification of lower grades (Dubin's grade 1) but is skewed on the high grades, whereas our classification is closer to the clinical grading and will thus create less confusion among general practitioners.

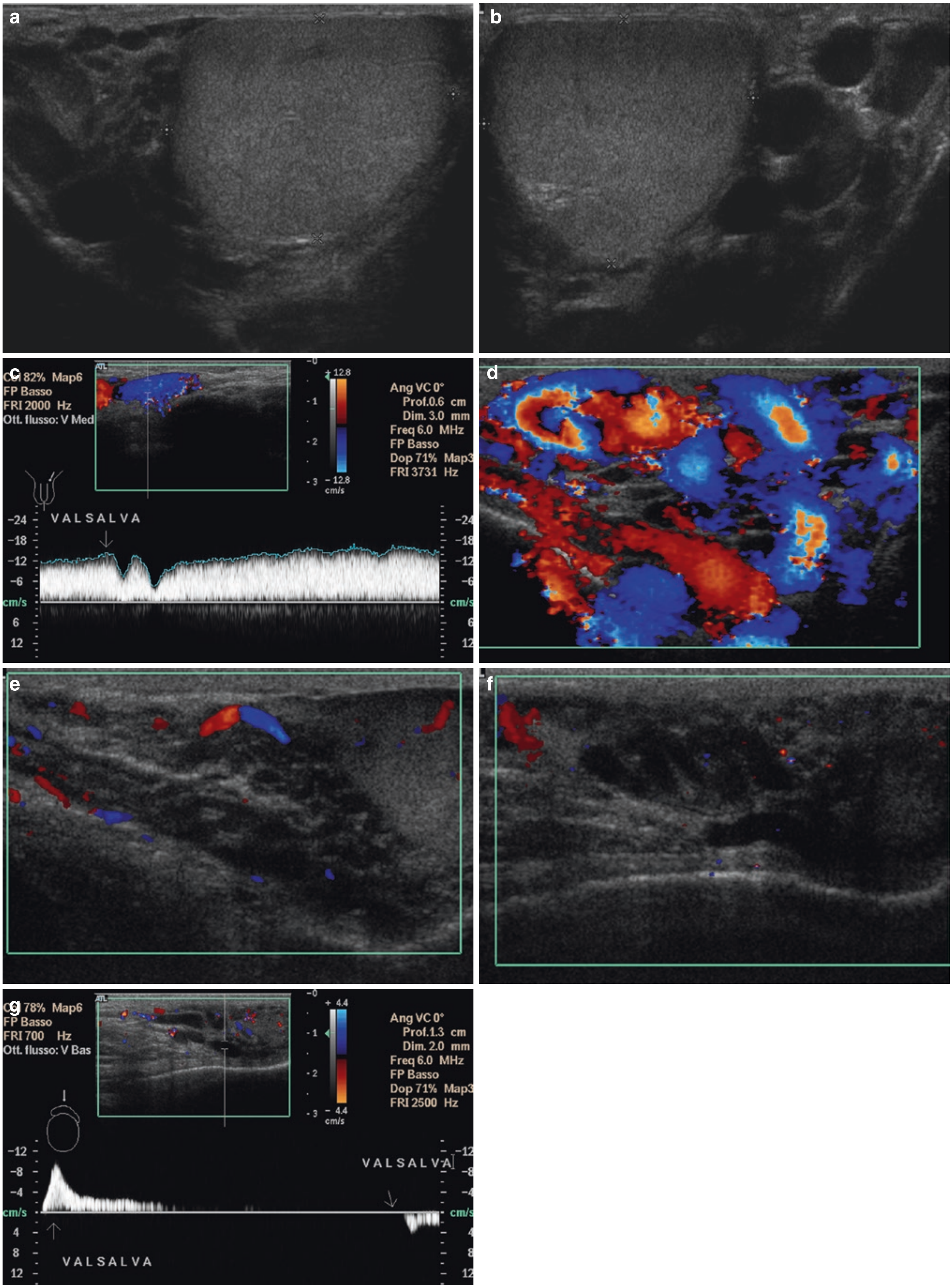

Fig. 6.16 Post-treatment. Longitudinal and transverse scans showing a bilateral varicocele (US grade 5 of the left side). Panels (**e**–**g**) show the same case after treatment by scleroembolisation of the varicocele: the size of the dilated veins is reduced, and the reflux is no longer detected either at rest or during the Valsalva manoeuvre

6.5 Intratesticular Varicocele

Intratesticular varicocele (ITV) is a rare condition characterised by dilation of the intratesticular veins, radiating from the mediastinum into the testicular parenchyma. Both mediastinal and subcapsular veins may be involved, and it is usually but not invariably associated with an ipsilateral extratesticular varicocele [6]. Bilateral ITVs are not uncommon [12] and are most frequently observed in patients who have undergone bilateral orchidopexy. In this group, intratesticular dilation may occur even with small extratesticular varicoceles.

Testicular pain is the most common clinical presentation, attributed to active or passive venous congestion and dilation of the intratesticular veins, which eventually stretch the tunica albuginea [6, 13]; however, even large ITVs may be completely asymptomatic and found by chance in the workup for infertility. The pathogenesis is thought to be similar to that of a varicocele, and more severe impairment of spermatogenesis is common.

On US, it has been described as a tubular or oval structure with a diameter of 2 mm or greater in and around the mediastinum testis with venous flow and a positive Valsalva manoeuvre response (Fig. 6.17) [12, 14]. A cut-off of 2.0 mm

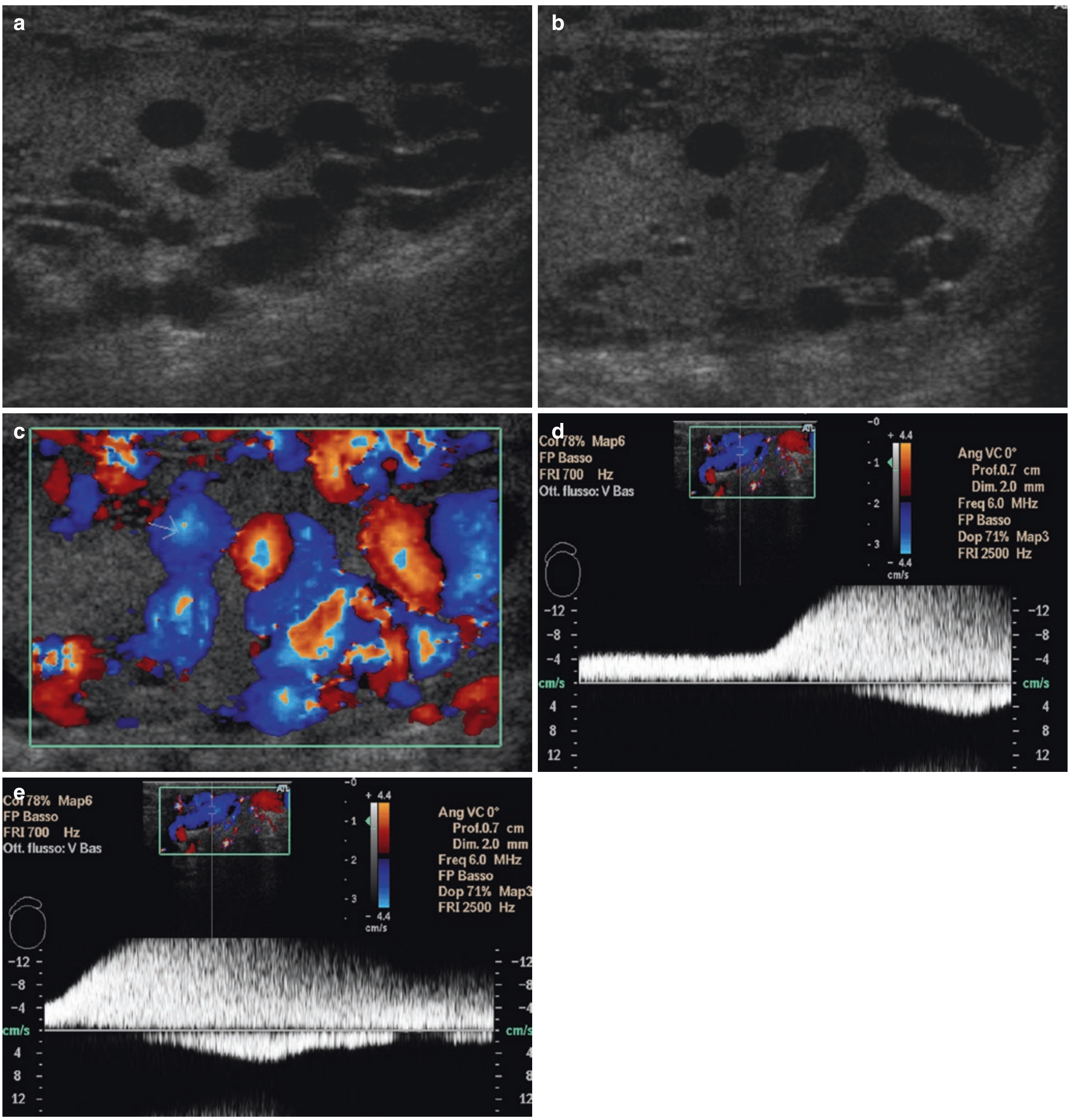

Fig. 6.17 **Intratesticular varicocele (ITV)** appears as tubular or oval structures with diameter ≥2 mm in and around the mediastinum testis. Panels (**c**–**e**) show the duplex colour and Doppler studies during Valsalva

(smaller than for extratesticular vessels) is generally used, but even intratesticular veins smaller than 2.0 mm are clinically significant if they show venous reflux on the Valsalva manoeuvre [6]. The main differential diagnosis in patients with intratesticular varicocele is against cysts, haematoma, abscesses, epidermoid cysts (usually with an echogenic rim) and cystic teratoma. The condition that most closely resembles a serpentine ITV is tubular ectasia of the rete testis, which is also seen in the proximity of the mediastinum (Fig. 2.13) [12, 14].

The use of colour Doppler imaging and the Valsalva manoeuvre, however, confirms the complete filling of the hypo- and anechoic structures with no evidence of an associated soft tissue mass. Colour Doppler images are sufficient to establish the diagnosis of ITV; no further investigations are necessary.

6.6 Adolescent Varicocele

Varicocele involves abnormal dilation of veins in the pampiniform plexus of the spermatic cord and is relatively common in adolescents. Although unusual before the age 10, as many as 15% of adolescent males are found to have a varicocele, which is usually asymptomatic [15]. It is usually found on routine physical examination.

Testicular ultrasound is the most accurate and reproducible method to assess testicular volume and significant testicular size variations [16]. The US features are the same as for adult varicocele. However, in adolescents precise measurement of volume is essential (the orchidometer is not sufficiently accurate). Varicocele may affect testicular growth, especially the rapid growth between the ages of 11 and 16, resulting in a volume discrepancy between the right and left testis [16]. A significant size discrepancy between the affected left testis and the unaffected right testis is the most common objective parameter, signifying possible testicular damage and the need for treatment [15]. It has been suggested that testes associated with larger varicoceles (Fig. 6.18) have a greater volume loss. However, there is little evidence of whether boys with a large varicocele are at a higher risk of growth arrest than those with a small varicocele. It is generally accepted that a varicocele can cause progressive damage in the affected testis, leading to potential problems with future fertility [17, 18].

In practice, size variations of more than 2 mL by ultrasound (for testes below 10 mL) or greater than 25% of the contralateral testis (if volume is greater than 10 mL) are currently the best indicator of testicular damage and should serve as the minimal requirement for surgical repair of the adolescent varicocele (Fig. 6.19) [19].

Sperm quality assessment is generally not possible at such a young age, as the maturation of the gonad is not complete until age 17–18. Alterations in semen findings therefore cannot be automatically attributed to varicocele.

The development of secondary infertility is a strong argument for early varicocele repair. However, not every boy with a varicocele and testicular growth arrest will be infertile, and there is still a need for a test that better distinguishes between adolescents who will develop infertility and those who will remain fertile [19]. Recent research demonstrates the potential importance of heat and oxidative stress patterns on testicular dysfunction with varicocele [20]. However, as said above, the most common indication for surgical correction of an adolescent varicocele remains significant testicular size discrepancy.

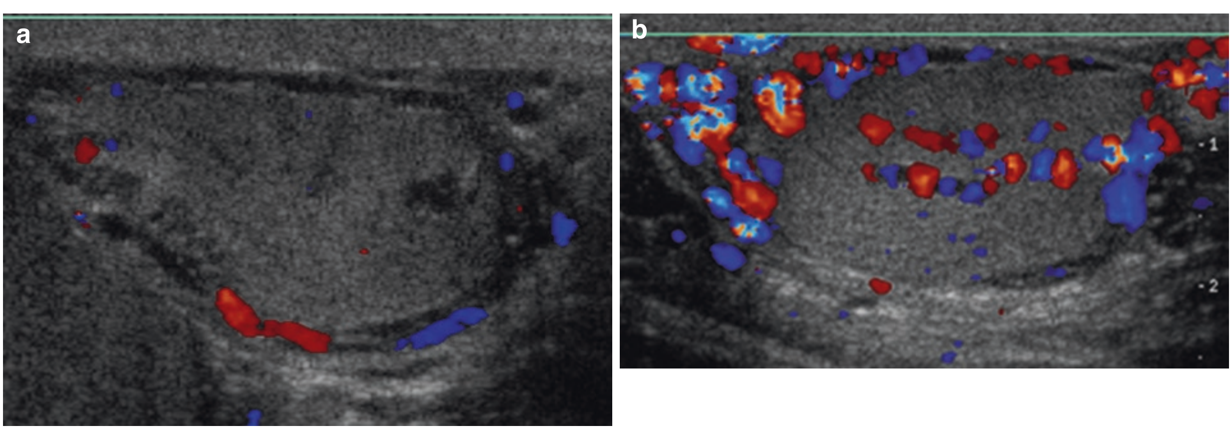

Fig. 6.18 Intratesticular varicocele. Further two cases of intratesticular varicoceles. Panels (**g**, **h**) show the surgical images of the ITV described in panels (**d**, **e**)

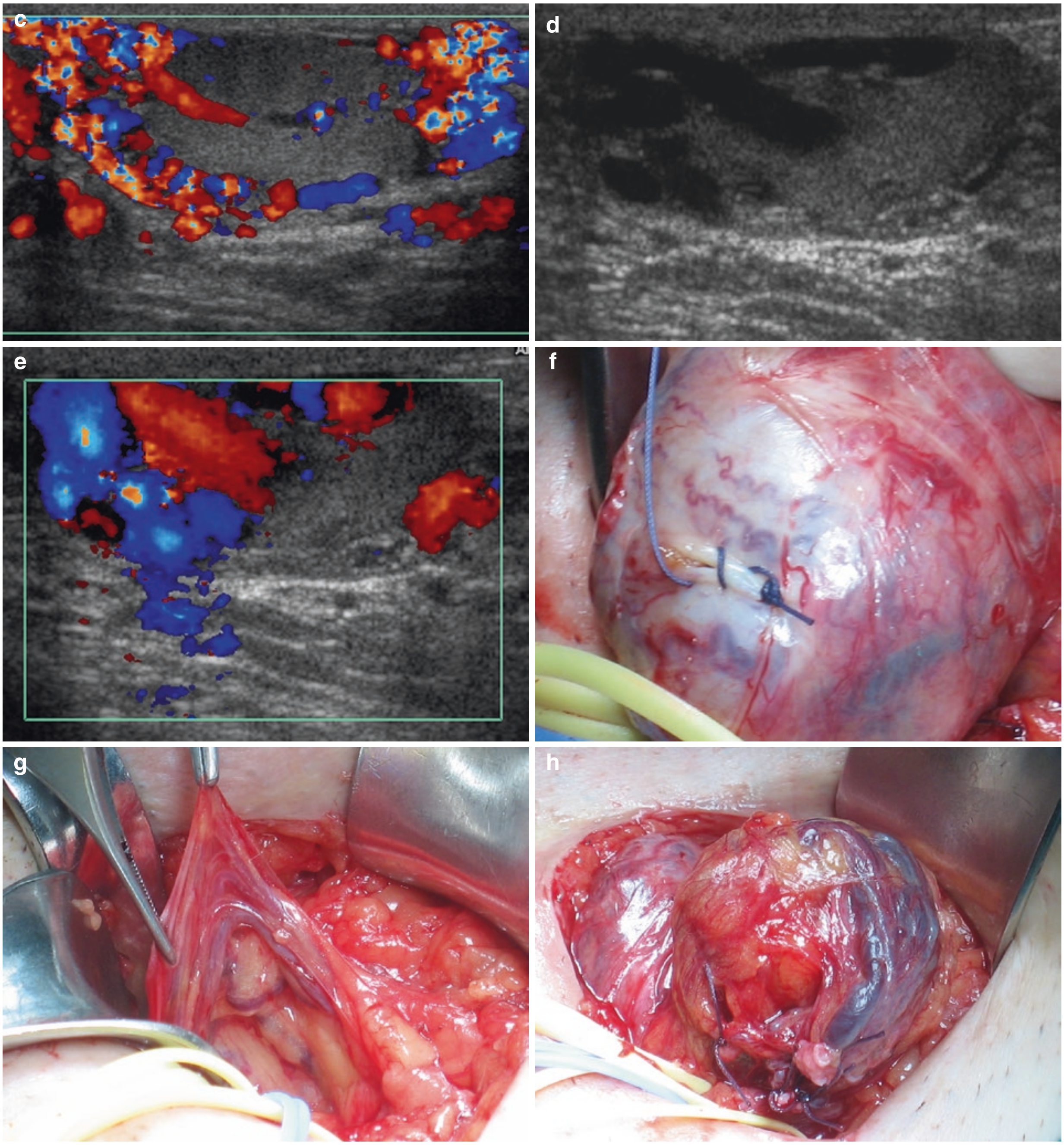

Fig. 6.18 (continued)

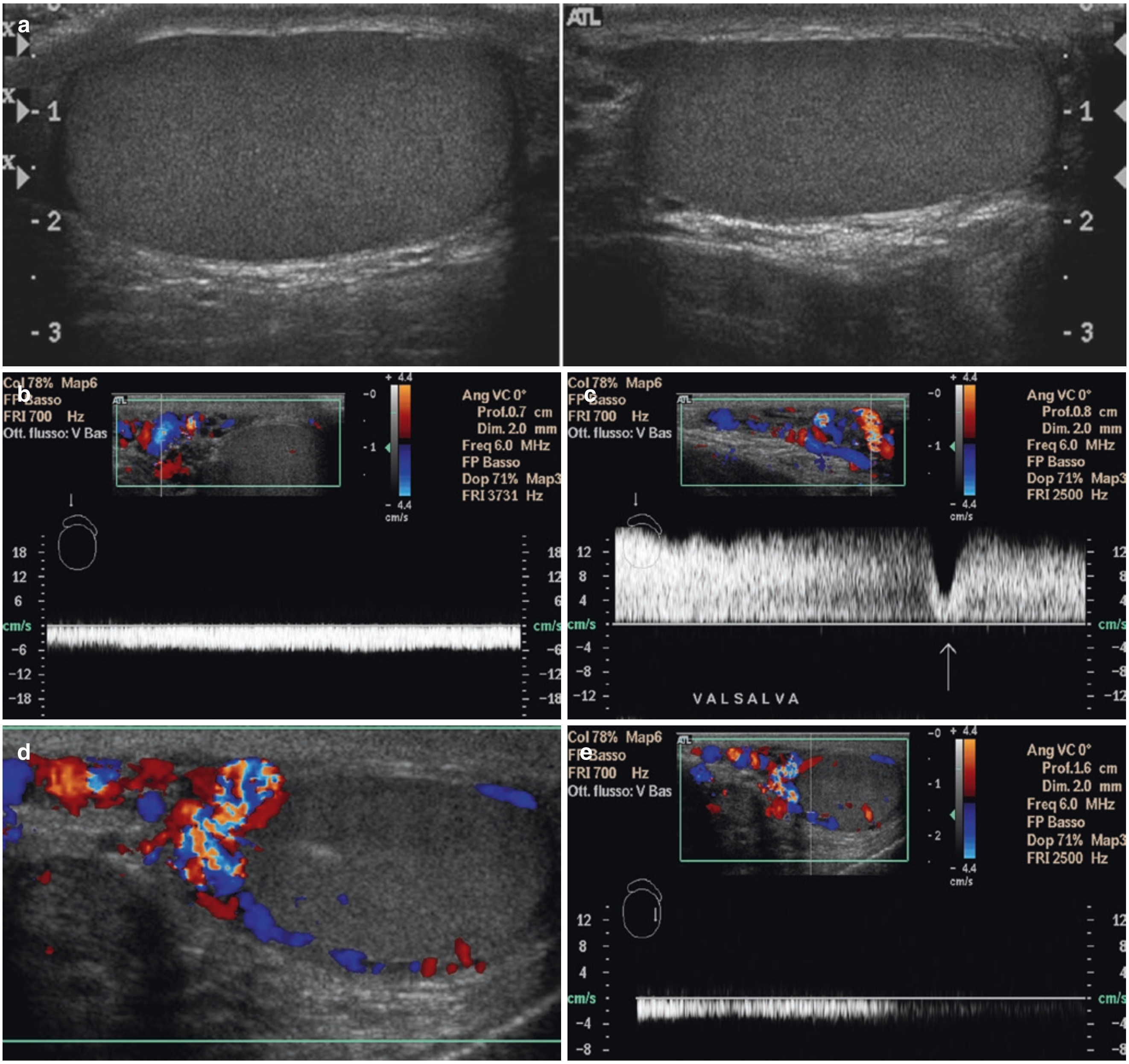

Fig. 6.19 Large left varicocele (**US grade 5**) in a 15-year-old boy. The left testis is reduced in size compared to the contralateral (**a**). Note the flow reduction (*arrow*) at the start of the Valsalva manoeuvre due to abdominal contraction (muscle girdle)

Key Messages

- A varicocele is a dilation of the veins of the pampiniform venous plexus. Depending on the screening method, it is found in about 10–20% of the general male population, increasing to 40% in infertile men. A high incidence of varicocele is also found in athletes (about 29%).
- Neoplasm is the most likely cause of non-decompressible recent/rapid onset varicocele in men over 40 years of age; it is frequently caused by a renal malignancy invading the renal vein. Emptying of the varicocele in the supine position distinguishes between varicoceles due to reflux and those caused by obstruction.
- In younger subjects, varicoceles are normally idiopathic and occur on the left side. Idiopathic varicoceles develop during early puberty.
- Clinical evaluation with the Valsalva manoeuvre is simple, but it may be complicated by concomitant hydrocele, cremasteric contraction and temperature of the examination room, relative volume of the testes, inguinal adiposity and most importantly the doctor's experience. Both over- and underestimation of the severity of varicocele are frequent, especially the latter.
- Dubin and Amelar devised a clinical grading system for varicocele that has the advantage of being universally accepted and easy to use. The clinical examination can be useful for screening.
- A US diagnosis of varicoceles should address all the following issues:
  1. Are the vessels of the pampiniform plexus enlarged?
  2. What is the extent of this dilation?
  3. Is there any retrograde flow during the Valsalva manoeuvre?
  4. Is there any retrograde flow at rest?
  5. Is reflux detectable at the inguinal level?
  6. How is spontaneous flow affected by the Valsalva manoeuvre?
  7. Are there enlarged vessels without reflux?
- Staging of varicocele should include a complete description of extension, size and number of dilated veins, affected side, duration of retrograde flow during Valsalva, presence of spontaneous retrograde flow in the upright position, volume and echotexture of the affected testicle (in comparison to the contralateral) and demonstration of retrograde flow in the inguinal canal of the affected side.
- Intratesticular varicocele is a relatively rare condition characterised by dilation of intratesticular veins. It can occur in association with extratesticular varicoceles but has also been described without large pampiniform dilations, especially in patients who underwent testicular surgical manipulation early in life.
- A diagnosis of varicocele in an adolescent should be accompanied by a precise estimate of volume discrepancies between the affected and non-affected testis and a repeated follow-up of testicular growth to detect any early arrest in development.

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7 Cryptorchidism, Pubertal Development, Hypogonadism and Other Conditions

7.1 Introduction

There are some neonatal and paediatric conditions that deserve a separate description. Although there is overlap between paediatric and adult pathology, differences in anatomy and pathology must be considered when evaluating the paediatric scrotum [1]. These differences, essentially due to the ultrasound appearance of the physiologically or pathologically undeveloped testis (cryptorchid, hypogonadal, etc.), could prove a diagnostic challenge in inexpert hands.

In general, the greyscale examination should always be accompanied by colour Doppler US evaluation to overcome the fact that most diseases appear hypoechoic to the surrounding parenchyma, and as the latter are less echogenic than in the adult, they might be difficult to recognise. For this reason, US scans must necessarily be interpreted in light of the clinical and physical examinations.

However, the compliance of children can be problematic. The examination time may thus have to be cut short, and children's movements can generate artefacts in colour Doppler US. When investigating disease in children, some authors therefore suggest beginning the evaluation on the affected side, in contrast with adults, in order not to miss any disorder [1].

The use of colour Doppler sonography is especially important in children, allowing both perfusion and morphology to be assessed [2]. However, its sensitivity in the detection of testicular flow is limited by the small size of the paediatric testes, the slow blood flow and the difficulties of keeping the child still for a sufficient time. However, colour Doppler evaluation remains crucial, and not only in acute disease, hypoechoic lesions can be missed in the hypoechoic testis of prepubertal boys, and tumours that have a subtle appearance on greyscale examination might light up because of their vascularity.

A detailed description of paediatric testicular malignancies is provided in Chap. 3.

7.2 Cryptorchidism

Cryptorchidism (Fig. 7.1) is a relatively common finding in paediatric practice. The incidence varies from 21% in preterm to 1.8–4.0% in full-term boys [3]. There is a higher risk of cryptorchidism in men with a genetic disorder (Down's syndrome) [4].

The precise developmental mechanisms leading to cryptorchidism are poorly understood; however, abnormalities in both functional anatomic and hormonal factors during embryogenesis and testicular descent are involved [5].

The testes originate within the retroperitoneum and migrate, guided by the contractile cordlike structure known as the gubernaculum testis, down through the internal inguinal ring, inguinal canal and external inguinal ring to the scrotum, at 36 weeks of gestation [3].

The distal bulbous portion of the gubernaculum testis is termed the pars infravaginalis gubernaculi.

Around the time of birth and occasionally during the first weeks thereafter, the testes descend into the scrotum through the processus vaginalis [1]. After complete descent into the scrotum, the gubernaculum testis atrophies; however, it persists if the descent is not complete [6, 5].

After testicular descent, the processus vaginalis is obliterated and the scrotal portion of this processus remains as a peritoneum-lined cavity, the tunica vaginalis, surrounding the anterior surface of the testis. Failure of the testis to descend into the scrotum and patency or anomalous closure of the processus vaginalis result in cryptorchidism, inguinoscrotal hernia and hydrocele [3]. The cryptorchid testis may be located at any point along the descent route (Fig. 7.2) [7]. When

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DOI 10.1007/978-3-319-51826-8_7

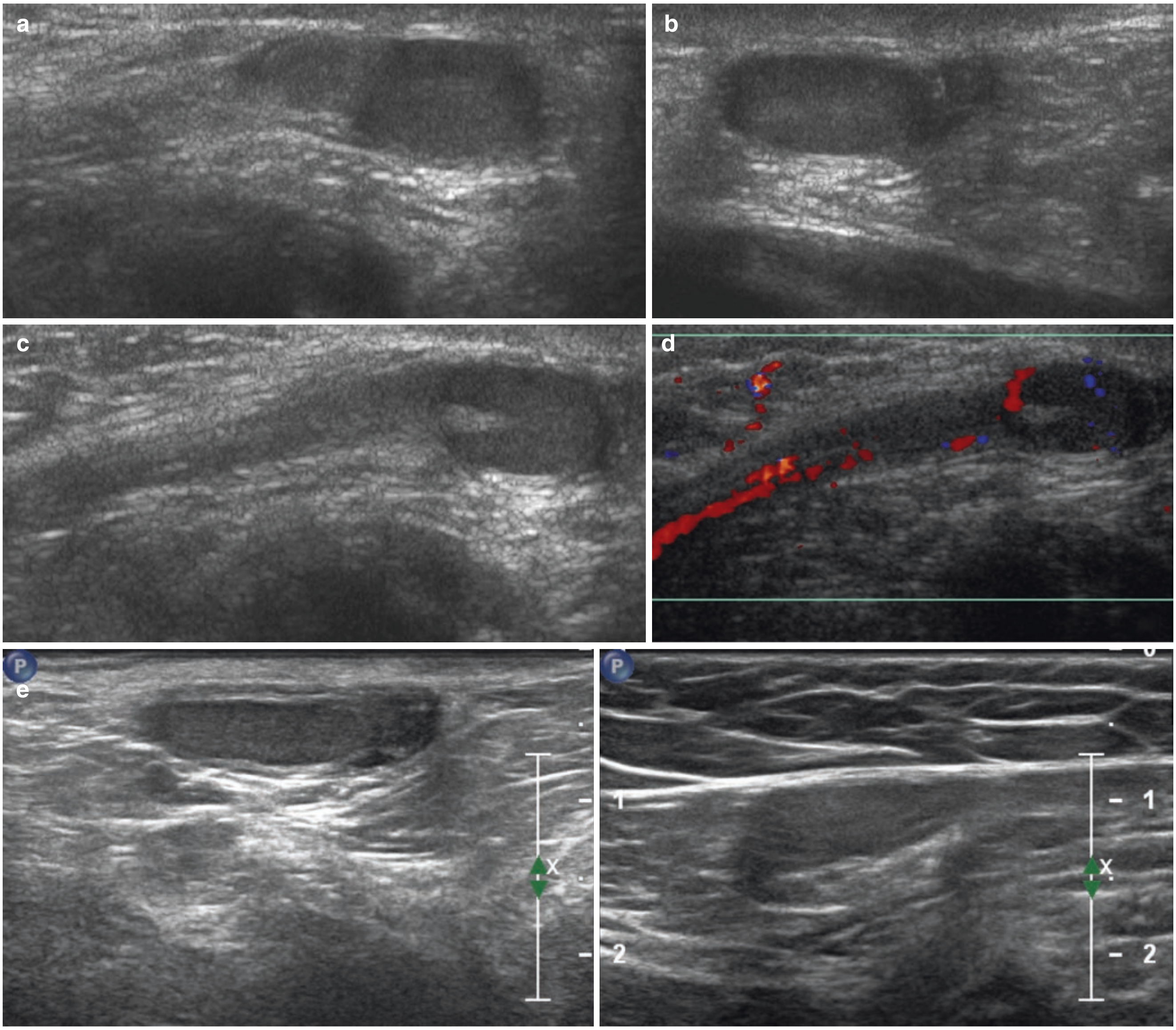


Fig. 7.1 Cryptorchidism. Longitudinal scan shows two small and hypoechoic undescended testes situated in the inguinal canal (**a**, **b**). Note the hilum in panel **c**, which helps in the identification of the testis (otherwise mistaken for the pars infravaginalis gubernaculi). Panel **d** shows the vascularisation of the cryptorchidid testis. Panel **e** shows a testis located in the scrotum (*left image*) and the contralateral located in the inguinal canal (*right image*)

searching for an undescended testis in infants and children, it is helpful to first examine the normally descended testicle and then search for a comparable structure on the contralateral side [8]. The undescended testis, however, is generally smaller and hypoechoic (Fig. 7.3) – not only because of the condition itself but also as the result of greater US attenuation by the overlying tissues – it frequently appears elongated and softer (easily compressed by the probe).

Two important complications of undescended testis are infertility, as the testis must reside in the scrotum to fully mature, and testicular malignancy (Fig. 7.4). The risk of malignancy is 20–46 times higher than in males with normally descended testes. The risk is higher for testes remaining undescended for longer periods, e.g. until the onset of puberty. To reduce the risk of infertility and cancer, orchidopexy is performed (Fig. 7.5); however, despite surgical treatment, the risk of cancer remains greater than normal in both the surgically corrected undescended testis and the contralateral normally descended testis [3, 9].

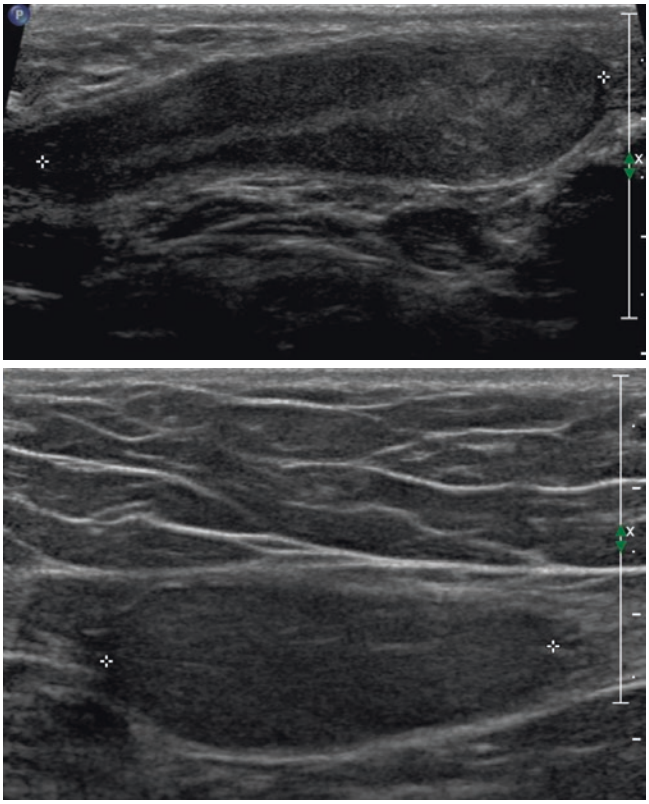


Fig. 7.2 Congenital cryptorchidism. Longitudinal scan shows undescended testes, respectively, of a 40- and a 28-year-old men. The testes are positioned higher to the internal ring, intra-abdominally, and lie in the abdominal fat. In this case the testes are markedly hypoechoic and irregular

Intra-abdominal testes have a four times greater risk of malignancy than inguinal testes. The most frequent malignant degeneration is usually due to seminomatous tumours and embryonal cell carcinoma [1].

Sonography is useful only for identifying testes in the inguinal canal (the most frequent location, 70% of cases) or in the prescrotal region just beyond the external inguinal ring (20%) and should be the initial imaging procedure (Fig. 7.6) [3].

Testes lying intra-abdominally near the external inguinal ring can often be seen (Fig. 7.7), whereas higher intra-abdominal locations make detection with ultrasound more difficult [1] as shadowing from bowel gas impairs the US visualisation of intra-abdominal testes [7]. In such cases, MR imaging offers greater sensitivity [1]. When cross-sectional imaging fails to detect one of the testes, laparoscopic surgery may be required. The diagnosis of testicular agenesia is only made by exclusion. One should be very cautious before dismissing the non-found testis as absent, as untreated intra-abdominal testes are at high risk of malignant degeneration [7].

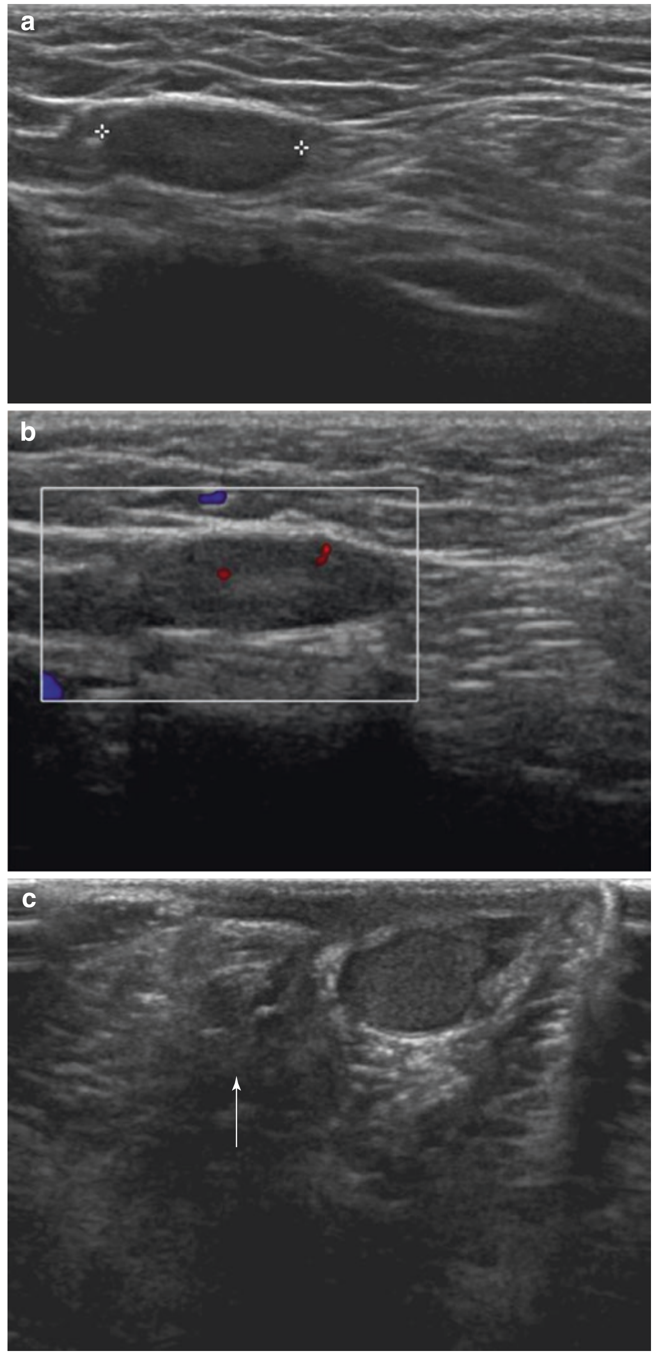


Fig. 7.3 Cryptorchidism. Longitudinal scan shows an undescended testis in the inguinal canal of a 3-year-old boy. The echotexture (**a**) and vascularisation are irregular (**b**). Vascularisation may not always be detectable. The presence of flow generally indicates viability. In panel **c** the empty right hemiscrotum is shown (*arrow*)

The presence of an oval mass in the inguinal canal (isoechoic or hypoechoic with respect to the normally located testis, in echotexture with echogenic mediastinum) is diagnostic [6]. However, care must be taken not to confuse an inguinal lymph node with a cryptorchid testis.

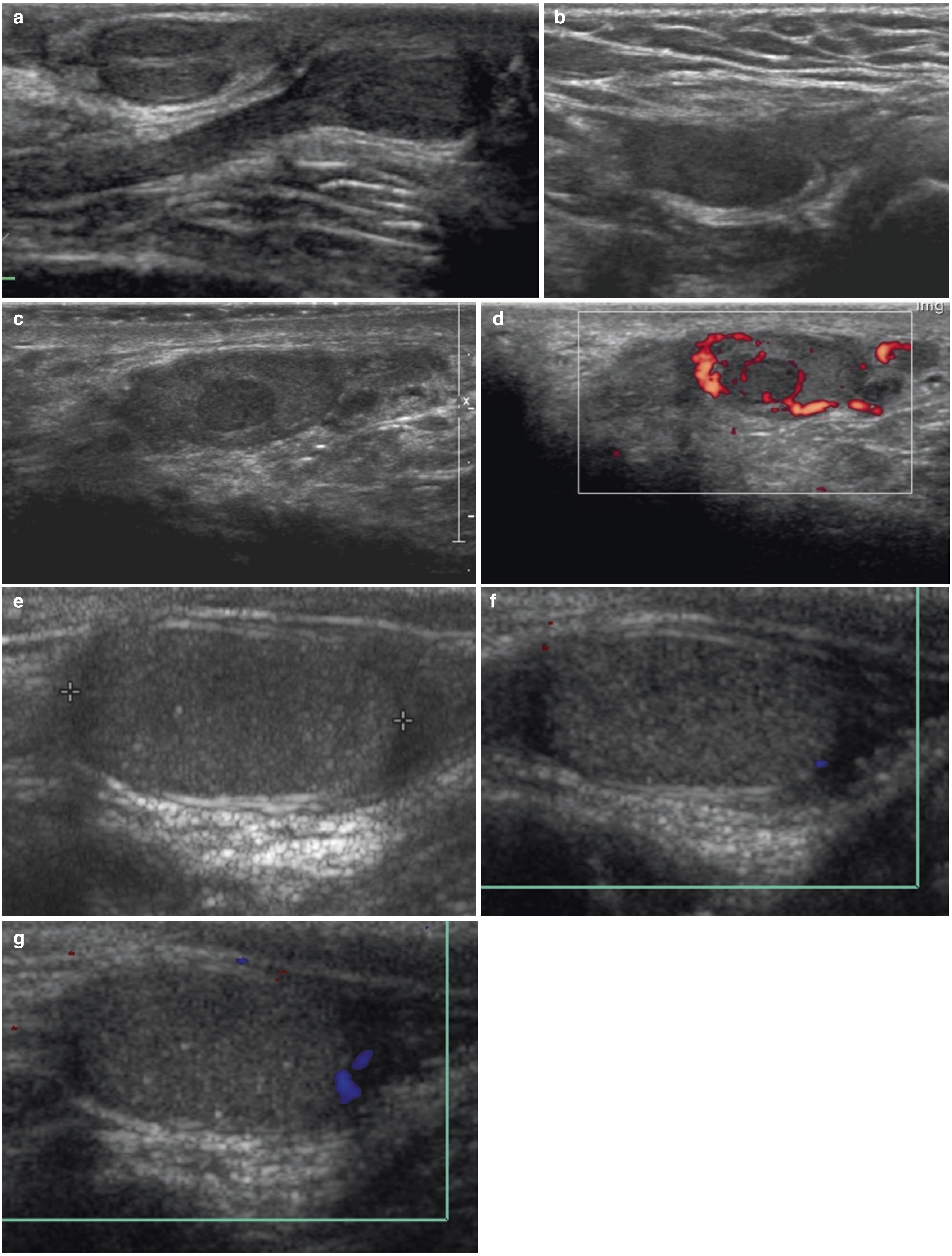


Fig. 7.4 Cryptorchid testes (**a**) in patients with Klinefelter's syndrome. Testicular dysgenesis can be associated with development of seminomas (**b-d**). Panels **e**–**g** show a cryptorchid testis of a 2-year-old boy, with inhomogeneous echotexture and microcalcifications. This testis must be followed up in the long term

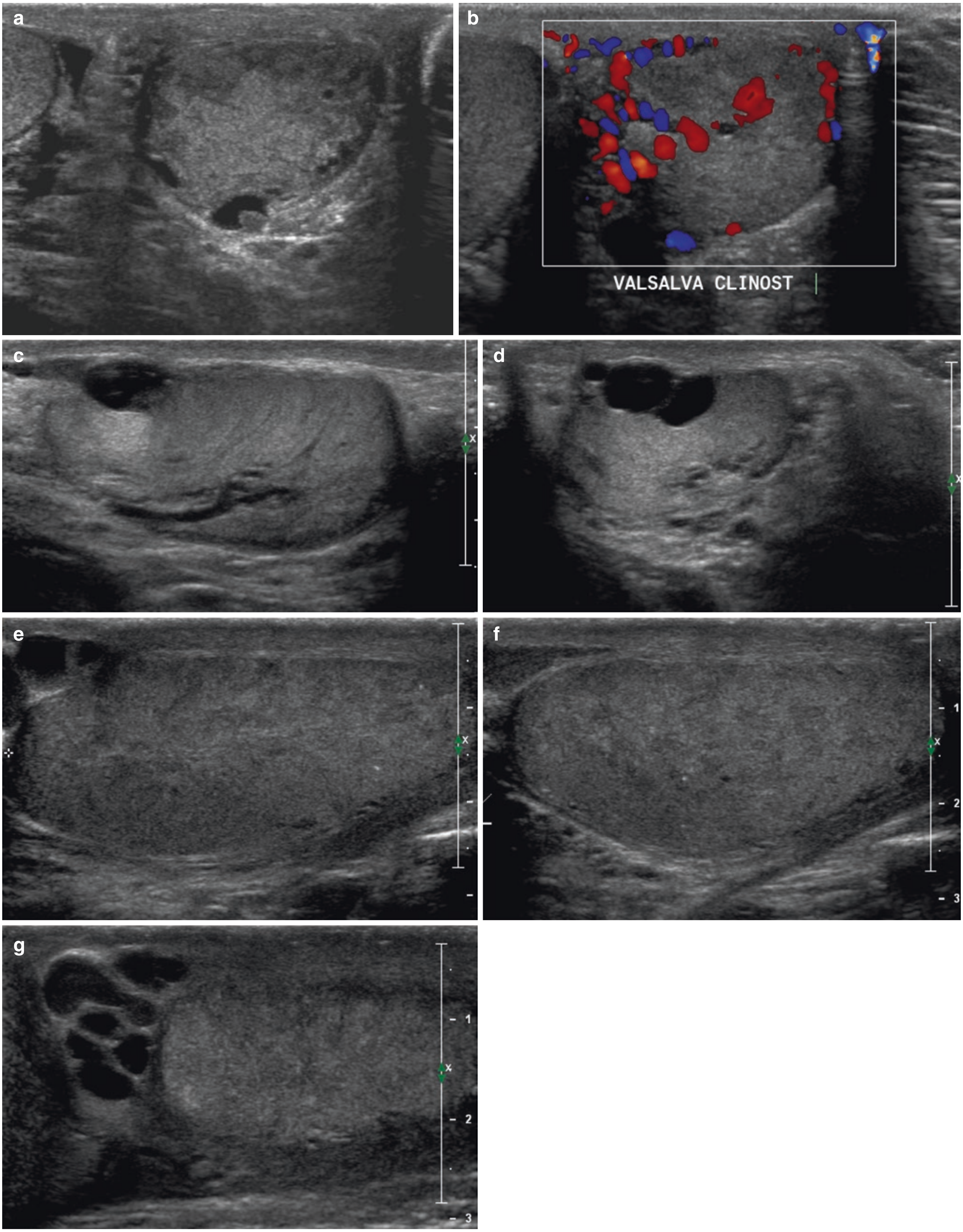


Fig. 7.5 Echo structure of cryptorchid testes after late orchidopexy. (**a**, **b**) US scan of the left testis of a 47-year-old man who had undergone orchidopexy at the age of 8, after 4 years of unsuccessful medical therapy. The testis is inhomogeneous, and intratesticular varicocele can be revealed at Valsalva manoeuvre. (**c**, **d**) Further case of late orchidopexy. (**e**–**g**) US scan of the testes of a 35-year-old man, azoospermic, who had undergone bilateral orchidopexy at the age of 6. Testes are inhomogeneous, and the epididymis shows cystic aspect

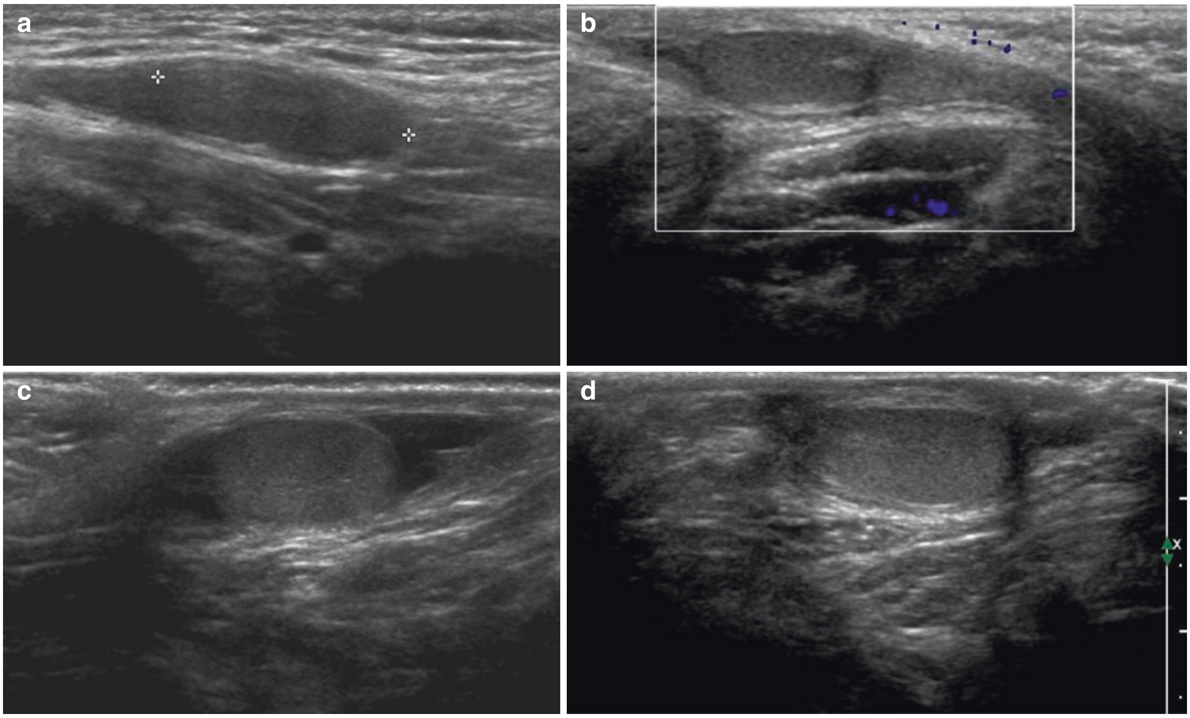


Fig. 7.6 Echo structure of cryptorchidid testes after medical therapy. Longitudinal scan shows the testes of a 1-year-old boy with cryptorchidism, before (**a**, **b**) and after (**c**, **d**) medical therapy with chorionic gonadotropin

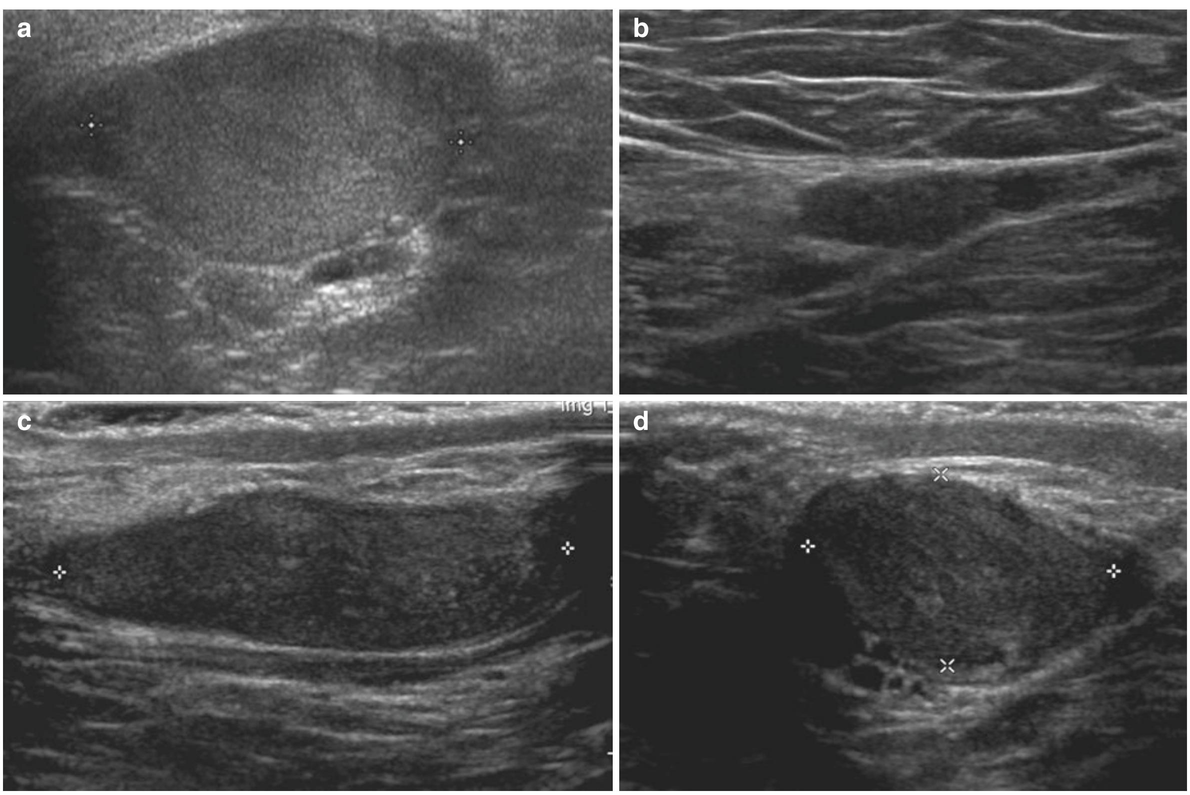


Fig. 7.7 Bilateral cryptorchidism not surgically corrected. Longitudinal scan shows both undescended testes of a 43-year-old man. The left testis is positioned higher to the internal ring, intra-abdominally, within the abdominal fat (**a**). The right testis is smaller than the contralateral, hypoechoic, and is located lower in the inguinal canal (**b**). In panels **c** and **d**, a cryptorchid testis of a 40-year-old man, referred for infertility, is shown. The testis had never been studied

Differential diagnosis depends on two easily identified features: (1) evaluation of mobility along the inguinal canal (Fig. 7.8) – present for the testis and absent for the lymph node (evoke the cremasteric reflex by stimulating the skin on the inner side of the thigh) – and (2) identification of the mediastinum testis (Fig. 7.1) that confirms that the structure is the testis and not a lymph node or the pars infravaginalis gubernaculi, which is the inferior bulbous portion of the gubernaculum testis [9].

Rarely, an undescended testis can be confused with inguinal hernia; real-time peristalsis confirms the presence of intestine [6].

Colour Doppler has at times been able to establish flow within the undescended testis, which is often smaller in size than its intrascrotal counterpart [4]. Torsion can occur in undescended testes, especially in those with malignant degeneration; in these cases colour Doppler evaluation can be very difficult.

In cryptorchidism it is important to describe the location of the affected testis: (1) intra-abdominal (between the caudal pole of the kidney and the internal inguinal ring) and (2) inguinal (between the internal and external inguinal ring) (Fig. 7.9) and prescrotal (at the top of the scrotum, below the inguinal canal and above the scrotal pouch).

In obese children, the testis may appear higher than normal, usually located near the external ring in the superficial inguinal pouch near the neck of the scrotum.

A further task frequently required is to distinguish between truly cryptorchid testes and retractile testes. Retractile or migratory testes (Fig. 7.10) slide back and forth between the scrotum and the external inguinal ring. Because the cremasteric reflex is most active between 2 and 7 years of age, retractile testes are common during childhood. This probably accounts for the inflated reported incidence of about 10% for undescended testis at 5 years of age. Between 25 and 33% of all boys referred to clinics because of suspected undescended testis are found to have normal but retractile testes [10]. Retractile testes usually take a normal intrascrotal position at puberty and no treatment is needed. However, there is concern that retractile testes spending most of their time in the inguinal canal could be damaged. Large prospective studies of the implications of early intervention vs. non-intervention on retractile testes are needed. It is therefore important that the room is adequately heated before performing the scans and that the exam is repeated twice on separate occasions.

US is also useful for therapeutic monitoring of testicular descent in patients undergoing hormone treatment. It is generally thought that the testicular echotexture is not affected by hormonal therapy; however, some changes in volume and vascularisation can occasionally occur.

Finally US is important for monitoring patients after orchidopexy and in follow-up for early detection and staging of possible malignant degeneration (Fig. 7.5).

7.2.1 Inguinal-Scrotal Hernia

An inguinal hernia occurs when tissue or part of the intestine pushes through a weak spot in the abdominal wall in the groin area, causing a bulge in the groin or scrotum. The prevalence of inguinal hernia is higher in preterm neonates. The hernia is more frequently located on the right side, since the right processus vaginalis closes later than the left [7]. Usually, a bulge in the groin or scrotal sac is seen. The bulge may be more noticeable during crying or straining and can come and go.

With greyscale ultrasound, a fluid-filled or air-filled loop of bowel in the funiculus or scrotum can be seen. In cases of herniation of the omentum, hyperechoic areas are found [1]. The diagnosis of hernia is achieved by visualisation of air bubble movement and/or intestinal peristalsis during real-time examination. US examination should include both inguinal canals, since a clinically unapparent contralateral hernia can be found in 88% of cases [7].

7.2.2 Hydrocele in the Neonatal Period

The most common cause of scrotal swelling in the newborn is neonatal hydrocele, which can easily be diagnosed on clinical grounds [1]. A few millilitres of serous fluid are normally present in the potential space between the parietal and visceral layers of the tunica vaginalis [9].

Virtually all hydroceles are congenital in neonates and infants and they are associated with a patent processus vaginalis, which allows peritoneal fluid to enter the scrotal sac. In older children and adolescents, hydroceles are usually acquired and are the result of an inflammatory process, testicular torsion, trauma, or tumour [7]. Septa, which are often seen in adults, do not occur in children [1].

On US the fluid surrounds the anterolateral margins of the testis, as posteriorly the testis is adherent to the epididymis and scrotal wall. The fluid is usually anechoic with good sound transmission and sometimes extends to the inguinal canal [9].

Closure of the processus vaginalis above the testis and below the internal inguinal ring leads to a less common type of hydrocele, also known as **spermatic cord cyst** [7]. This is a localised fluid collection along the spermatic cord, separated from and located above the testicle and the epididymis. It produces an inguinal swelling and is sometimes indistinguishable from a mass on palpation. US is important to differentiate this anomaly from other pathologies present in the groin, such as an incarcerated inguinal hernia, a paratesticular mass (cyst or rhabdomyosarcoma) or an inguinal lymphadenopathy [11].

Another highly uncommon type is abdominoscrotal hydrocele. These are large inguinoscrotal hydroceles that

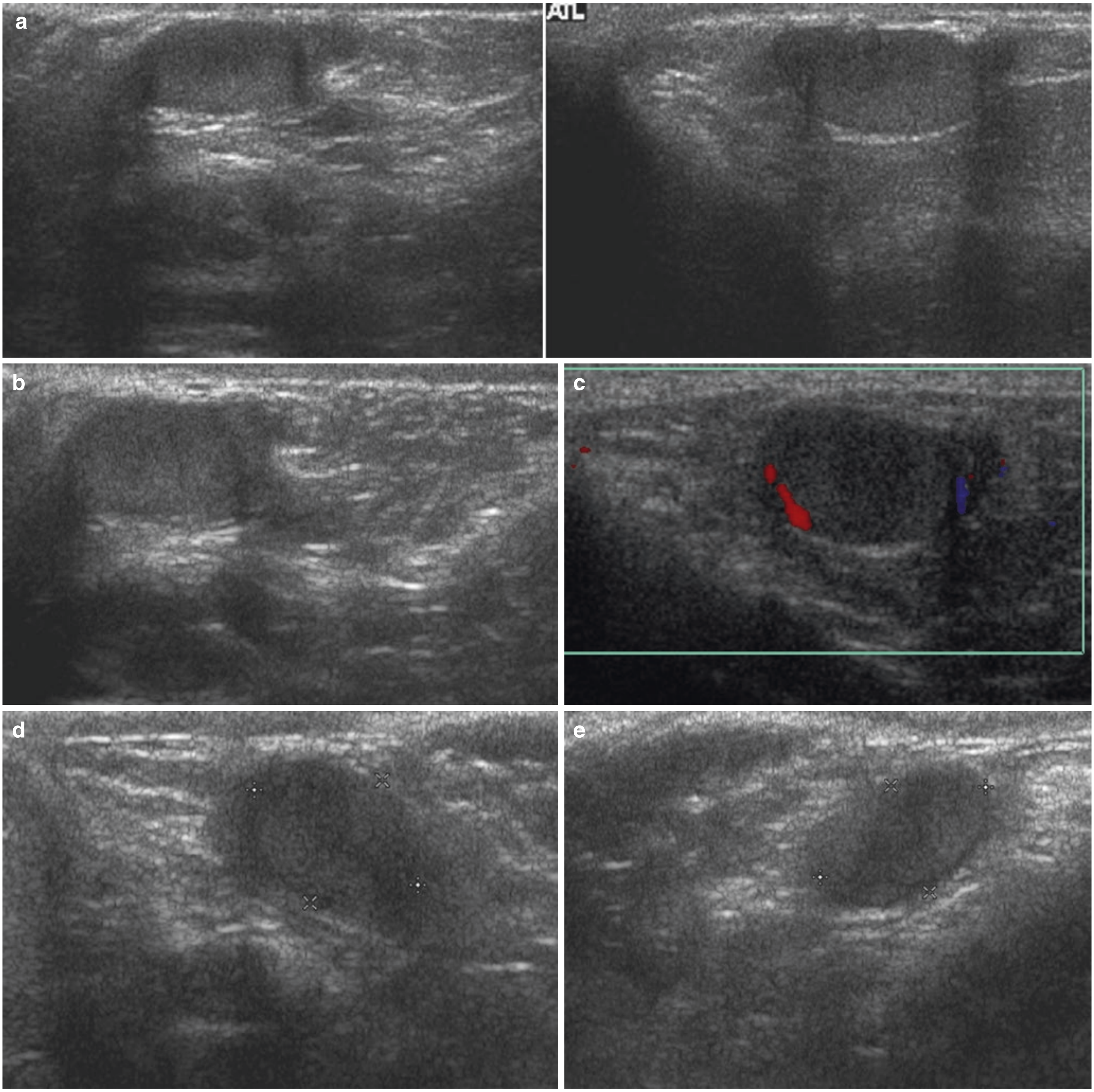


Fig. 7.8 Cryptorchidism. Longitudinal scan shows both undescended testes of a 10-year-old boy. The testes are inhomogeneous and located high in the inguinal ring (**a**–**c**); they can move into the inguinal canal if manipulated (**d**, **e**)

protrude through the internal inguinal ring into the abdominal cavity and manifest clinically as a communicating abdominal-scrotal mass.

Most congenital hydroceles (80%) resolve spontaneously before the age of 2 years. However, spermatic cord and abdominoscrotal hydroceles are usually treated surgically [7].

7.3 Pubertal Development

Estimation of **testicular volume** in boys is important for evaluating normal **pubertal development**. To date, different types of orchidometers, ordinary rulers, callipers and ultrasound have been used to assess testicular size [7].

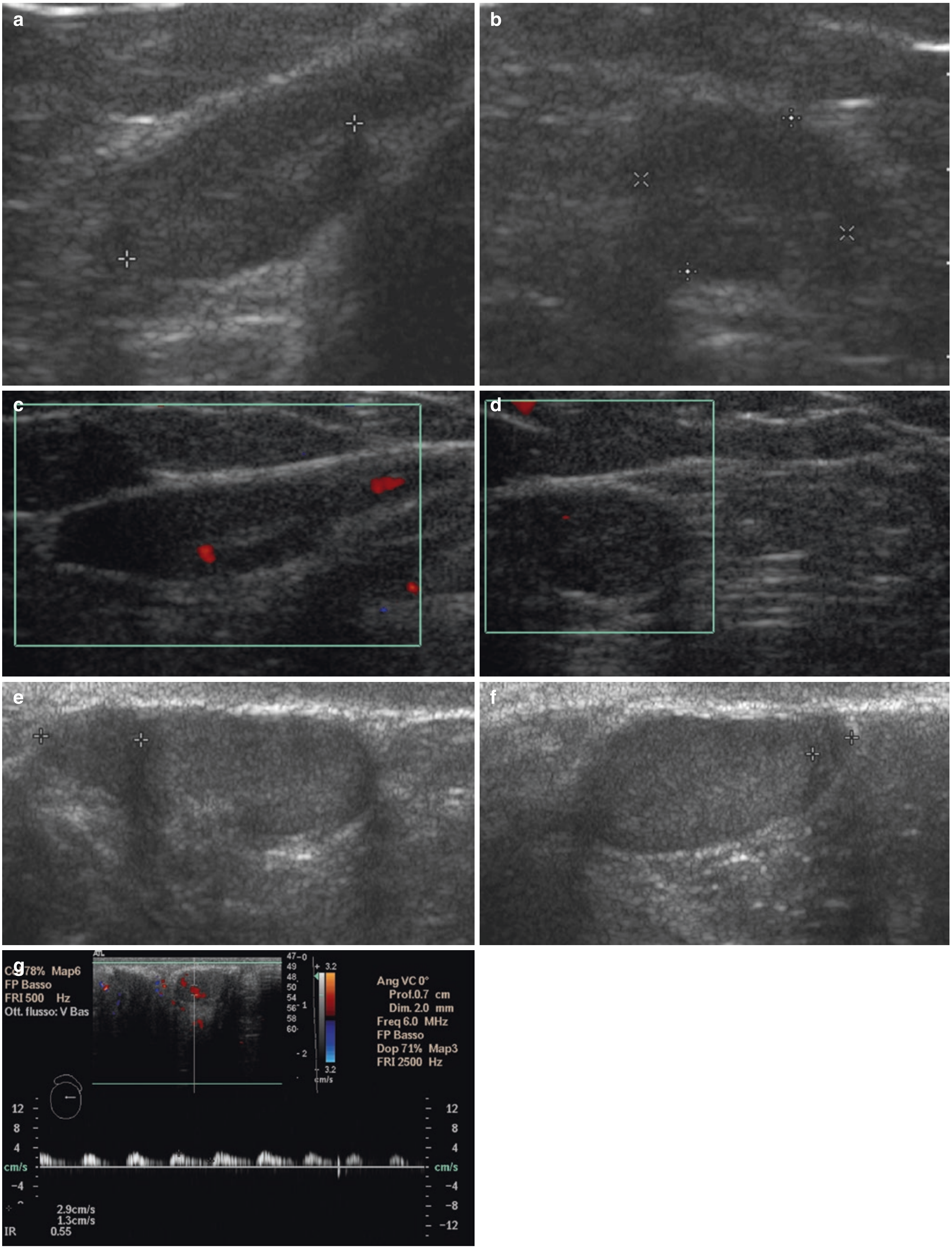


Fig. 7.9 Cryptorchidism and late puberty. US scan of a 14-year-old-boy revealed bilateral undescended testes, located in the inguinal canal. The volume was compatible with G1 stage of puberty (**a**–**d**). The 2-year follow-up scan showing descending testes (**e**–**g**)

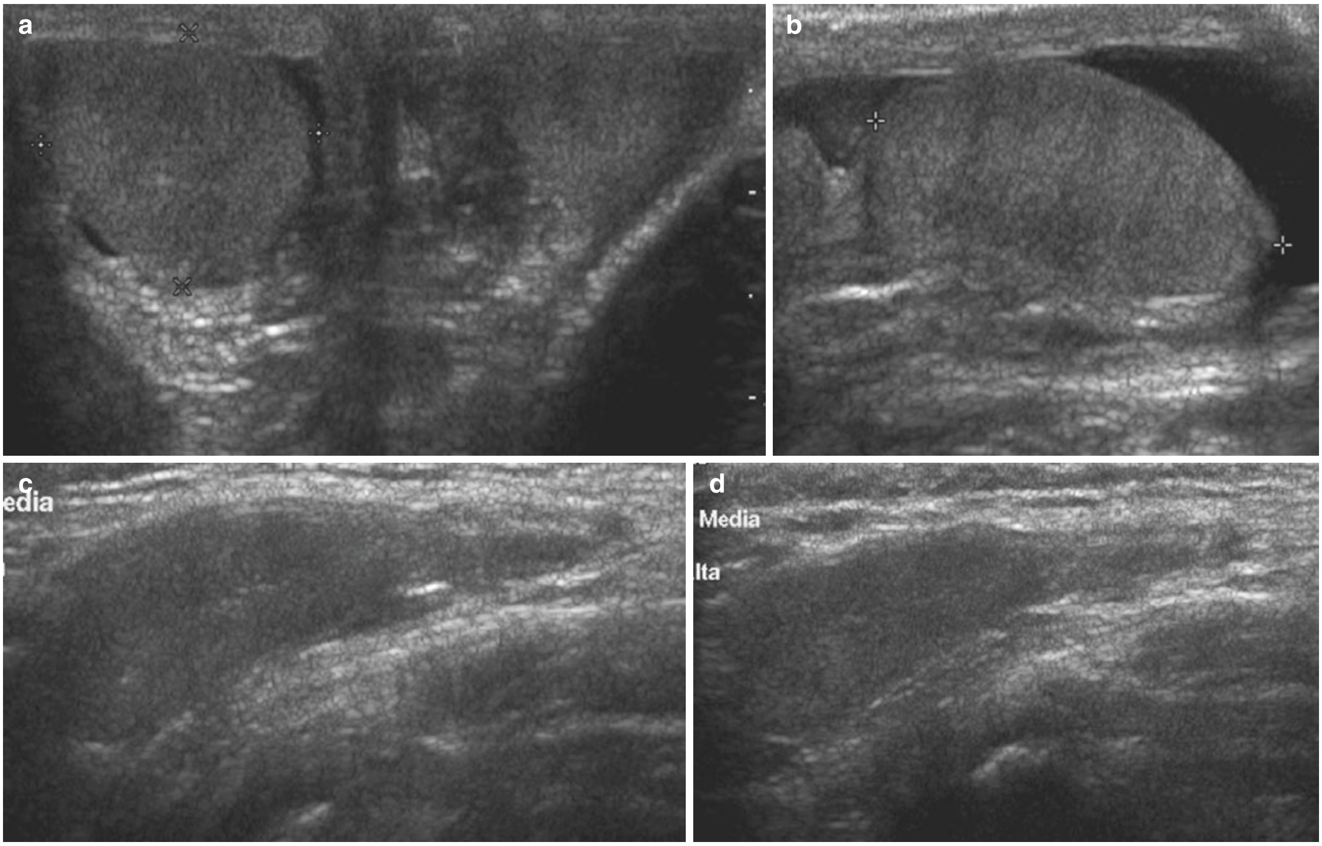


Fig. 7.10 Retractile testis. A retractile testis is defined as a testis above the scrotum that can be manipulated easily into the scrotum and remain there without traction until the cremasteric reflex is induced. Panels **a** and **b** show the testis of a 9-year-old-boy, ascended into the inguinal canal after manual manipulation (**c**, **d**)

Ultrasound is the most reliable, precise and reproducible method for determining testicular volume, especially during the first years of life [12]. However, the formula used by most machines to calculate the volume ($L \times D \times H \times 0.52$) underestimates the real volume of small testicles by up to 40%. Use of a 0.71 correction factor (instead of 0.52) is therefore highly recommended. In order to ensure consistency with subsequent follow-up examinations, the same formula should be used for all scans in the same subject.

The volume of a normal prepubertal testis is between 1 and 2 mL and remains constant in preadolescence. This can create some concern in parents noticing linear growth in the rest of the body, but not in the external genitalia of their son, especially in overweight boys with or without delayed puberty. At the onset of puberty, the testis suddenly increases in size, almost exponentially (Fig. 7.11). The volumetric increase can begin before other pubertal changes or acquisition of other secondary sexual characteristics [7].

Testicular volume enlargement of 4 mL or more is used as a clinical landmark for the onset of puberty (Fig. 7.12) [13]. This generally corresponds to a longitudinal maximum diameter of more than 2.5 cm.

As seminiferous tubules comprise 70–80% of the testicular mass, testicular volume is believed to reflect spermatogenesis. Accurate testicular volume measurement is therefore important for assessment of testicular physiology [14]. Germ cell elements and tubular maturation also determine the **echotexture** of the testicles. The increase in echogenicity during puberty is primarily the result of growth of the seminiferous tubules, which increase in diameter and develop a lumen [15].

In neonates and infants, the testis is seen on US as an ovoid structure of low echogenicity surrounded by an echogenic line, which corresponds to the tunica albuginea [7]. The mediastinum can also be recognised. In neonates and infants, vascularisation is mainly peripheral. Prepubertal testis echogenicity increases progressively from 8 years of age to puberty with the development of germ cell elements (Figs. 7.13 and 7.14), to reach a medium echogenicity in post-pubertal testicles (Fig. 7.15) [16].

The spermatic cord appears as a smooth linear structure limited by a highly echogenic band on longitudinal scans. On transverse scans, it is ovoid with an echogenic margin [7].

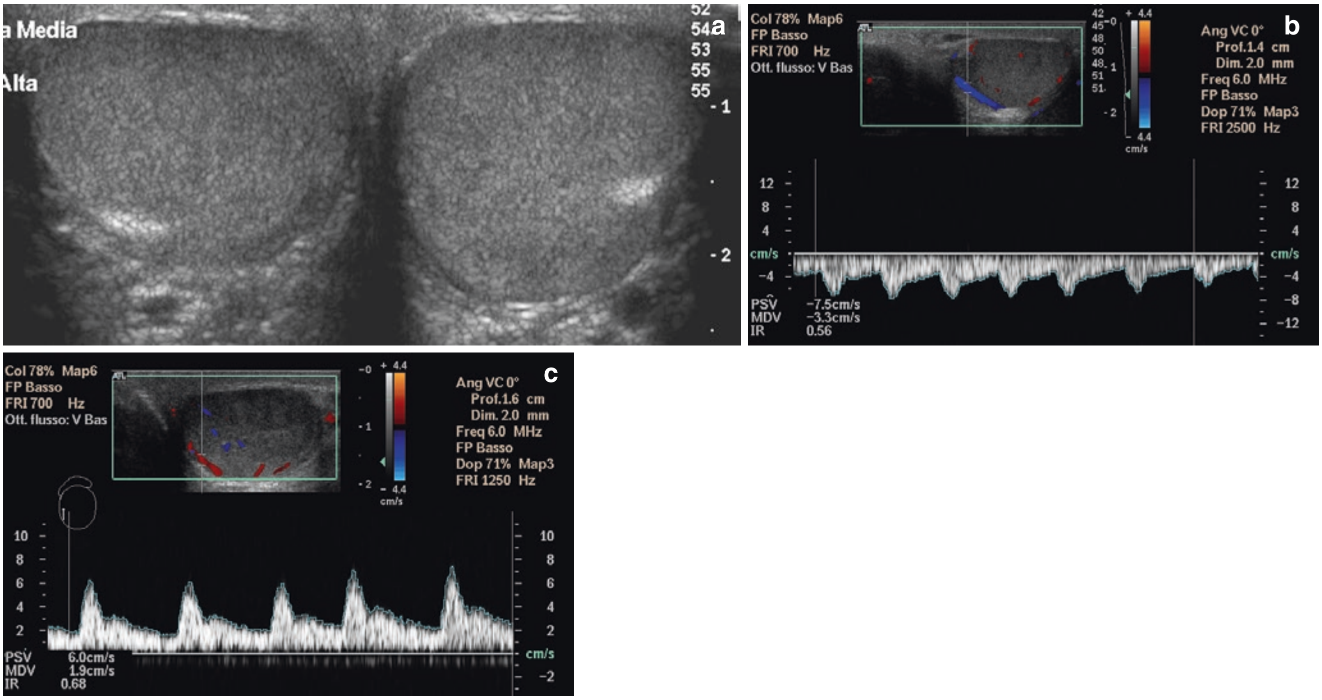


Fig. 7.11 Precocious puberty. Longitudinal scan of the testes of an 8-year-old-boy shows uniform echo structure and echogenicity, with a medium reflectivity, only slightly less echogenic than in adults, as seen in advanced puberty (**a**). The volume was also compatible with advanced puberty (G3–G4). Panels **b** and **c** show the subcapsular arteries with high flow, typical of pubertal development

In pubertal patients, the capsular artery (branch of the testicular artery) is located beneath the tunica albuginea and some centripetal and recurrent intratesticular rami are easily identified. In the small prepubertal testis, the capsular artery is more easily identified (panels b–d in Fig. 7.13) than the intratesticular arteries, which are seen as pulsating dots. The spectral waveform of the intratesticular arteries has a low-flow, low-resistance pattern [7].

Follow-up of the changes observed in the spectral waveform of developing testes is of interest. Clearly, puberty coincides with a dramatic increase in overall blood flow within the testis. Such changes are reflected in greater peak systolic velocities that are typically seen in the capsular branches of the testicular artery. These steep peaks gradually give place to the normal shape of blood flow in the intratesticular arteries, as the development of the testis is accompanied by a growth in the vascular bed. The changes in arterial inflow generally anticipate the increase in volume and echogenicity, suggesting that this event is hormonally driven and is the *primum movens* of puberty. In addition, there is a clear temporal link between the increase in testicular arterial inflow and the development of varicoceles. The relationship between testicular vascularisation and testicular pathology is complex, but it is now clear that its different aspects can be more easily understood by observing the testis during puberty.

7.4 Varicocele in Puberty

Varicocele is a common condition in adolescent boys (Fig. 7.16), with an incidence of 15% [17]. The condition is described in detail in Chap. 6. Technically, varicoceles in adolescents have similar ultrasound features to those in adults. In a morphofunctional approach, however, there are a few aspects that deserve further attention.

Varicocele is an abnormal dilatation of veins in the pampiniform plexus of the spermatic cord. Idiopathic or primary varicoceles typically develop during early puberty but can occasionally be found in preadolescent boys. They can be associated with a time-dependent growth arrest. It is thus very important to measure testicular volumes repeatedly, to monitor any increase in the size difference between the affected and the unaffected testis.

Bilateral varicoceles may impair the growth of both testes. It has been shown that a difference of just 10–15% in volume can be associated with reduced sperm quality. Whether this results in reduced fertility in adulthood is still unknown. However, in adolescents with varicoceles, we suggest that a >20% variation in volume that remains stable or increases further on two separate scans at 3–6 month intervals should be considered as clinically significant.

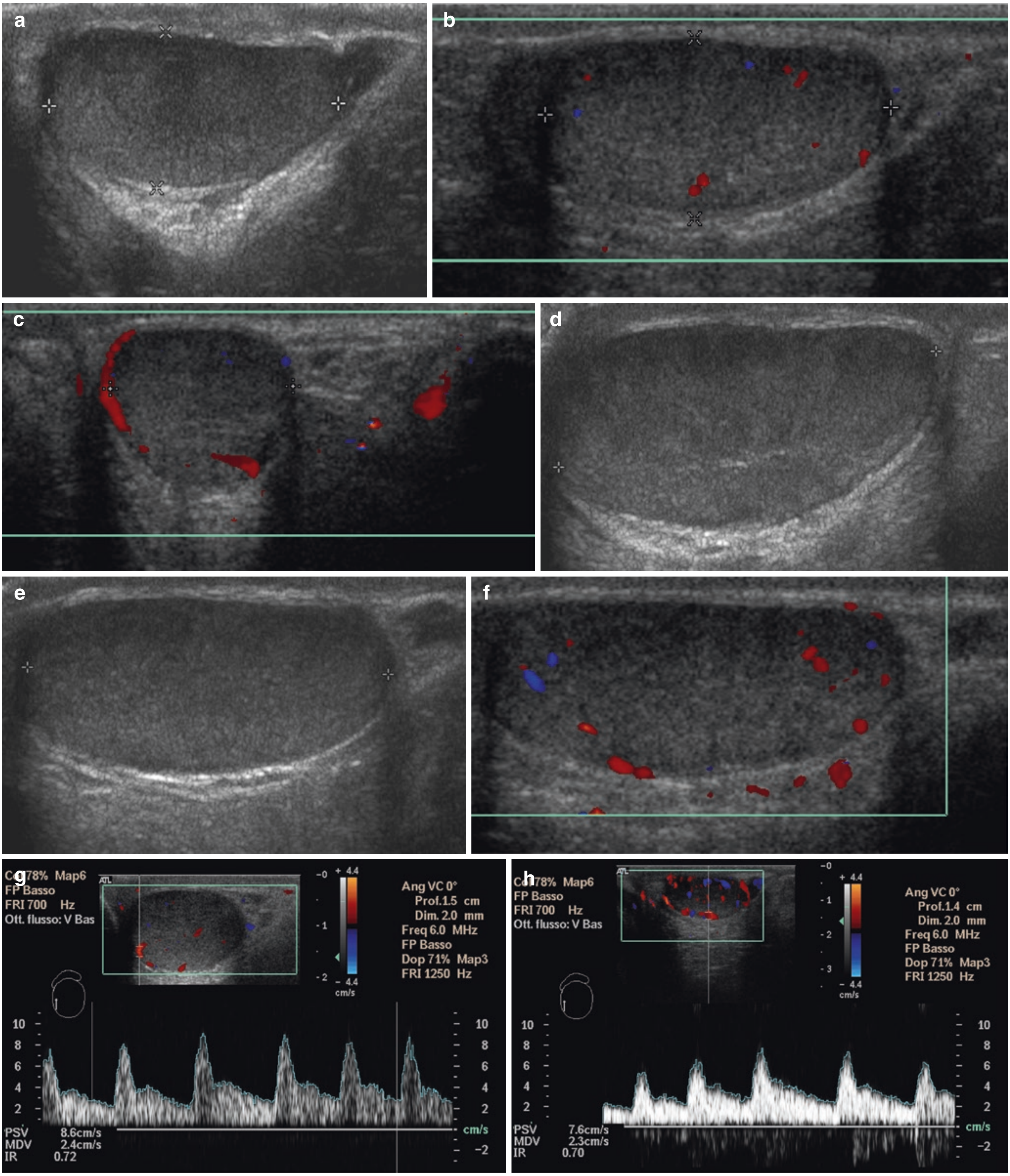


Fig. 7.12 Late puberty G1–G2. Longitudinal scan of the testes of a 15-year-old boy showing small (**right 2.2 cm^3 and left 2.4 cm^3**), hypoechoic prepubertal testes, with little vascularisation (**a**–**c**). He was prescribed testosterone enanthate 50 mg i.m. 1 vl per month. Six months later (**d**, **e**) US revealed increased testicular size (**right 5.4 cm^3 and left 3.7 cm^3**), especially of the right testis (**d**). Intraparenchymal testicular vascularisation appears normal (**f**), with lively flow in both pericapsular arteries, as seen during stages of pubertal development (**g**, **h**)

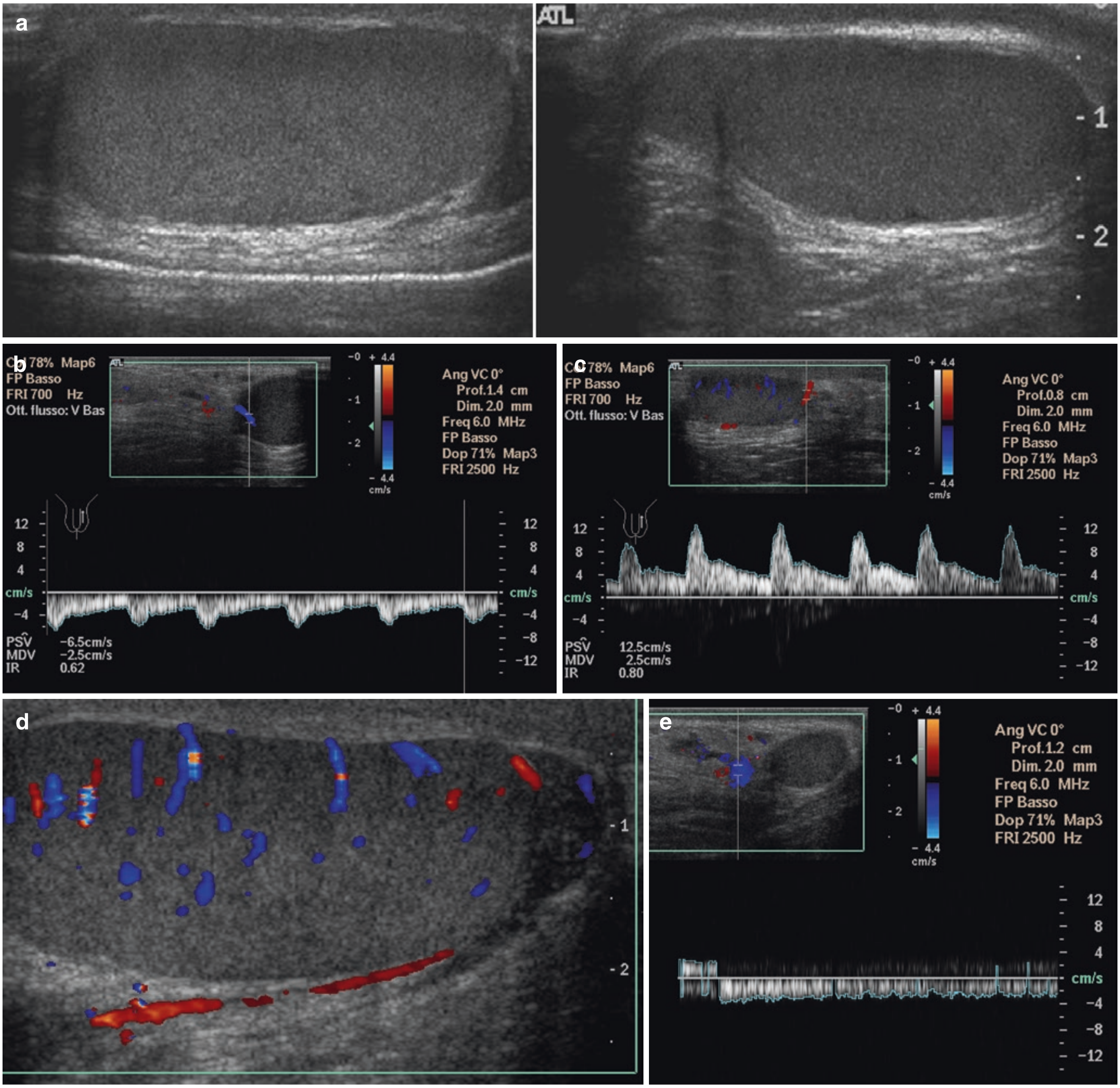


Fig. 7.13 Late puberty G3–G4. Longitudinal scan of the testes of the boy described above (Fig. 7.9) at the age of 16½ shows further growth of the right testis, in contrast with the left. The right testis is compatible with puberty stage G3–G4 (right **11.0 cm³ and left 5.4 cm³**). The left testis appears substantially more hypoechoic than the contralateral (**a**). Intraparenchymal vascularisation seems normal in both testes with lively flow in both pericapsular arteries, as seen during stages of pubertal development (**b–d**). Some hypoanoechoic areas in the left pampiniform plexus, maximum diameter 3 mm, can be attributed to possible venous dilations demonstrating basal flow in upright standing position (**e**). Panels **f–i** show later scans (at age 17 [**f**, **g**] and 17½ [**h**, **i**]). Both testes have grown, but the left is still smaller than the right (**right 15.9 cm³ and left 8.5 cm³**)

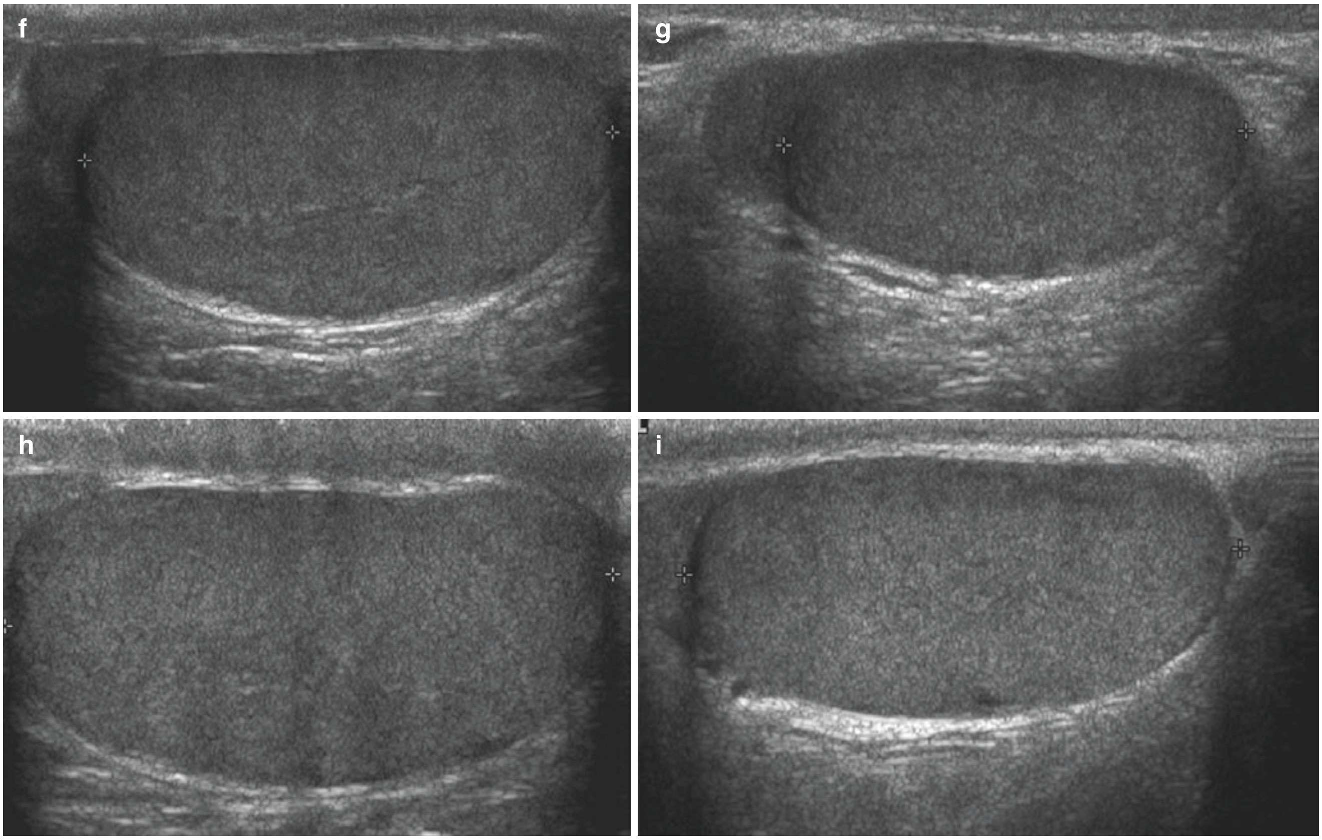


Fig. 7.13 (continued)

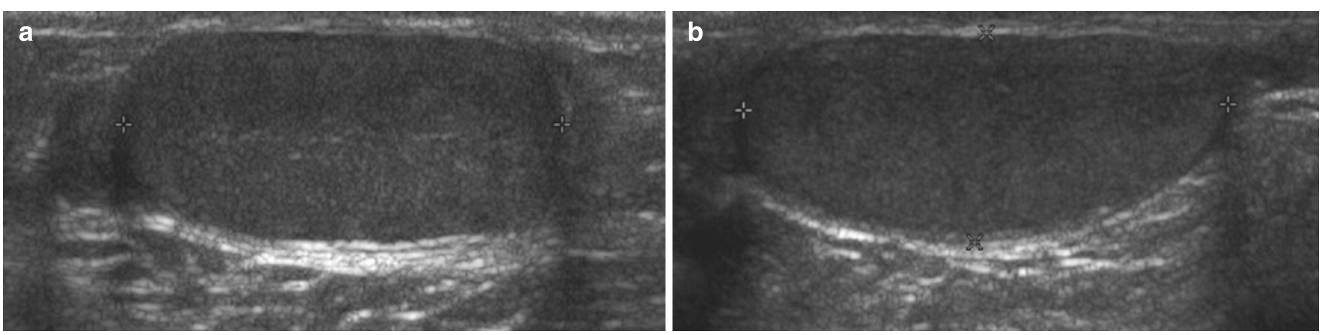
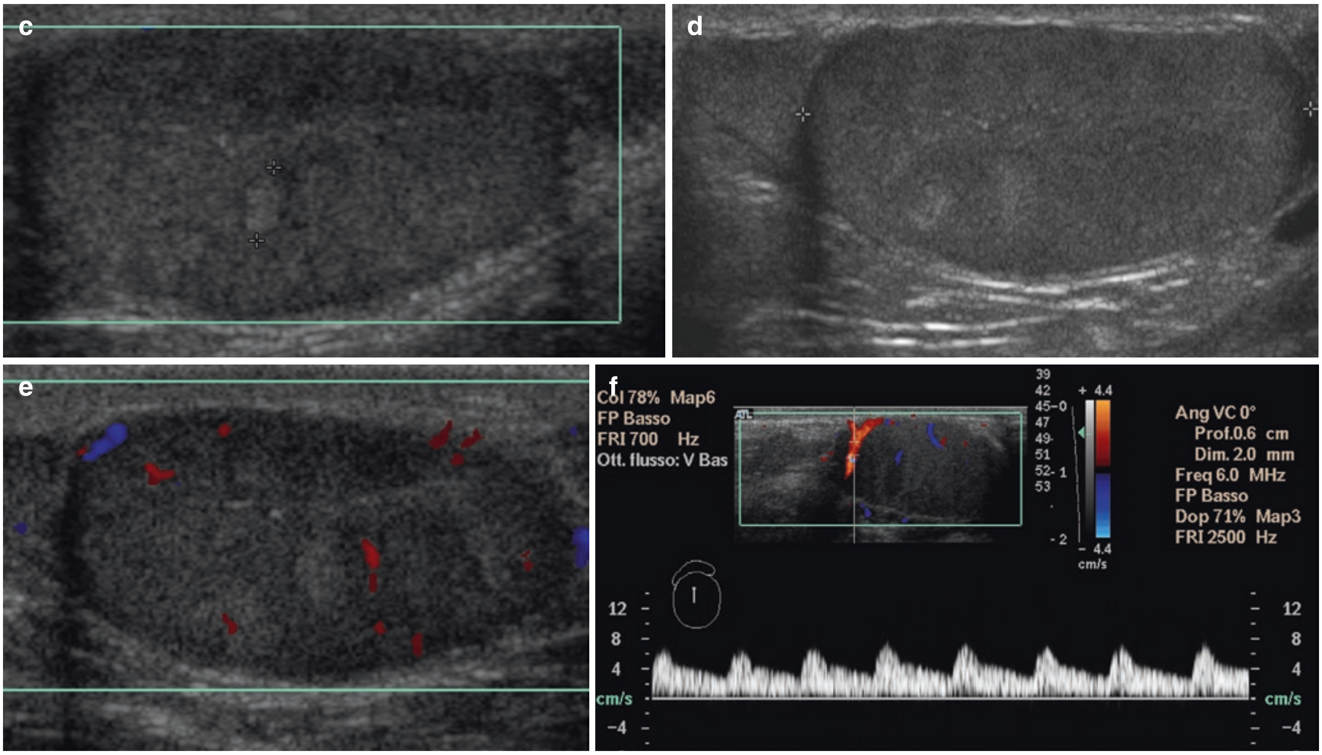


Fig. 7.14 Puberty. Longitudinal scan of the scrotum of a 15-year-old boy shows testes smaller than normal for age (**a**, **b**) but compatible with initial stage G of pubertal development *(G1–G2)*. Both testes have a slightly hypoechoic echo structure and slightly higher than prepubertal echogenicity, indicating progression towards development stages; however the parenchymal structure is uneven, with a hyperechoic hilum. Poor, relatively disorganised intraparenchymal vascularisation (**c**). At 6-month follow-up (**d**), volume was compatible with intermediate stage G (G2–G3), larger than at previous examination. Slightly hypoechoic echo structure and slightly higher than prepubertal echogenicity, indicating progression towards development stages. Poor, relatively disorganised intraparenchymal vascularisation (**e**) but better than at previous examination and some high-flow subcapsular arteries typical of pubertal development observed (**f**)



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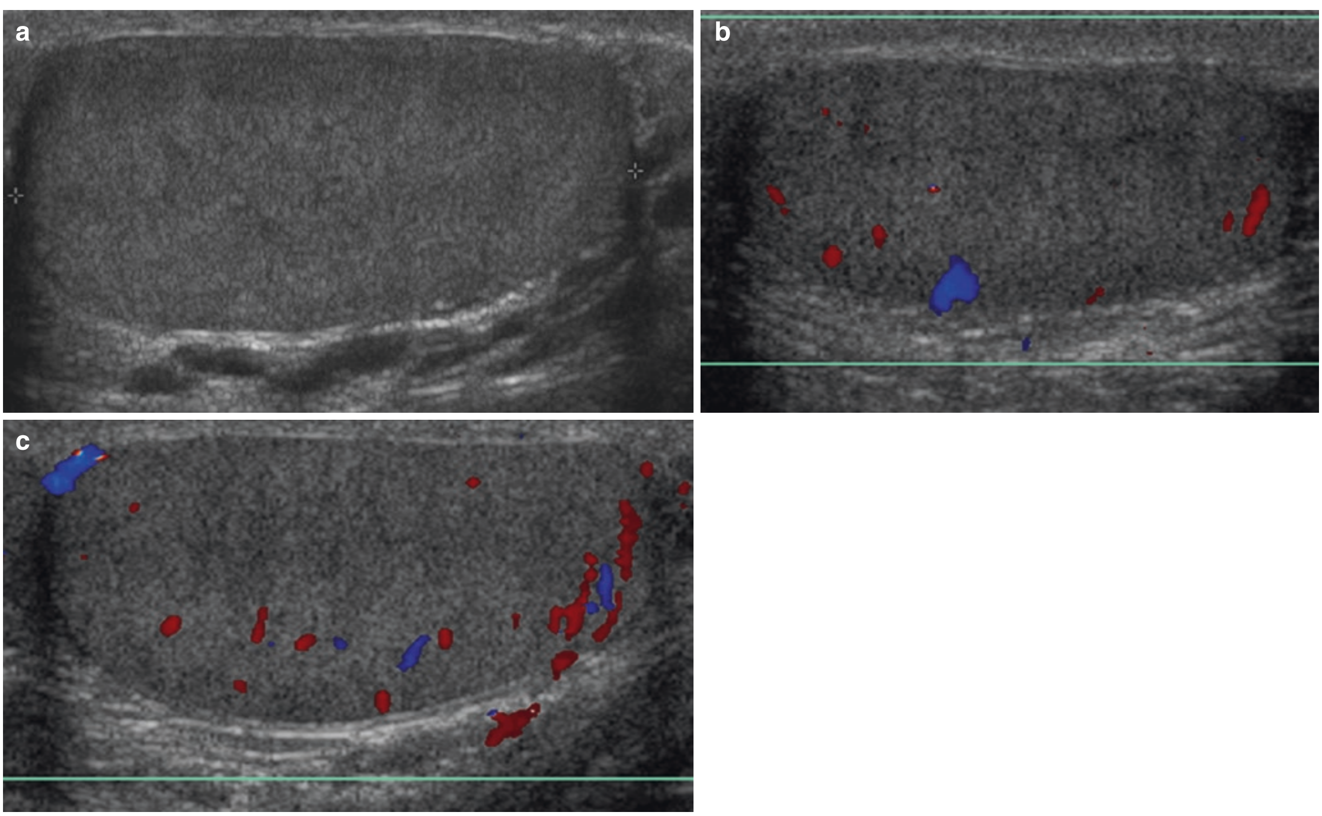

Fig. 7.15 Pubertal examination. Longitudinal scan of the testes of the boy described above (Fig. 7.14), at the age of 17, shows increased testicular reflectivity (**a**) and normal vascularisation (**b**, **c**). The boy did not receive any therapy

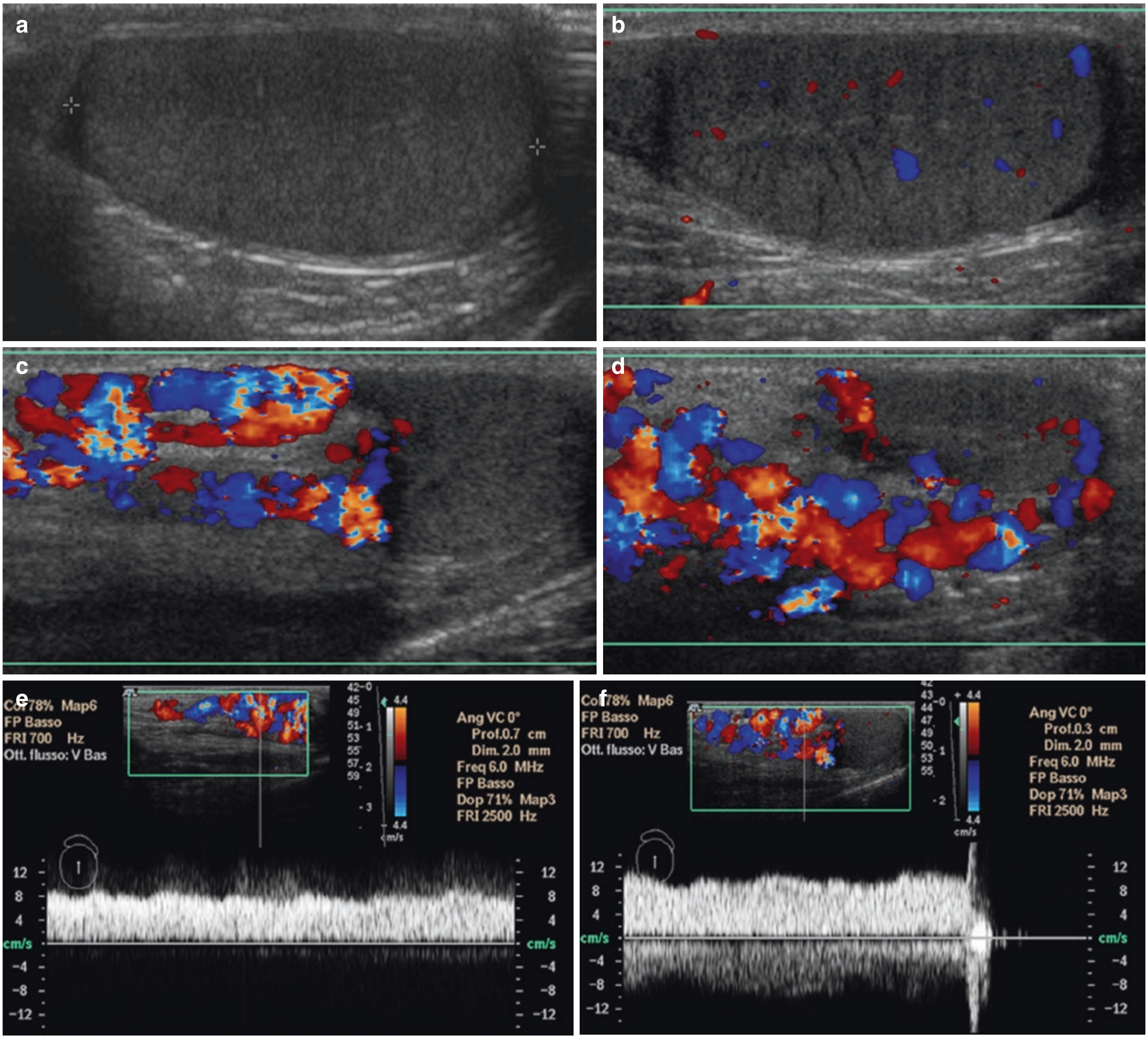

Fig. 7.16 Adolescent varicocele. US scan of the testes of a 12-year-old boy showing grade 4 varicocele. The affected testis (**b**) is smaller than the contralateral (**a**). The veins surround the posterior aspect of the testis (**c**, **d**). In orthostatism, there is a pathological venous flow lasting for >3 s during the Valsalva manoeuvre (**e**, **f**)

Management of varicocele in adolescents remains controversial. The relationship between grade of varicocele, incidence of testicular growth arrest and further likelihood of decreased fertility has not been clearly elucidated [17]. Since gonad maturation requires several years, the spermiogram of adolescents can give results that are abnormal in comparison with the reference range for adults. However, this may simply be a normal stage of development, rather than the result of damage. The spermiogram is thus useless in evaluating the impact of varicoceles on spermatogenesis.

Ultrasound is therefore the only objective assessment of testicular damage. Aspects to watch for are volume discrepancies, accurate staging of varicoceles and abnormalities in the parenchymal echotexture.

Most authors suggest that a US volume difference of greater than 2 mL is currently the best indicator of testicular damage and should serve as the minimum requirement for surgical repair of the adolescent varicocele [18]. We believe that this is inaccurate. First of all, volume differences should be expressed relative to the contralateral testis; therefore a percentage reduction is more accurate. Second, volume may be affected by other conditions (such as cryptorchidism, hydrocele, etc.); therefore the staging of the varicocele is equally important: it is unlikely that a US grade I varicocele is responsible for a large volume reduction. Finally, assessment of testicular echogenicity and echotexture can help in identifying early spermatogenesis impairment. Large varicoceles are associated with changes in parenchymal appearance: the homogeneous granular texture composed of closely packed medium-level echoes is replaced by coarse echoes with reduced reflectivity and thickening of hypoechoic septa. We believe that a prospective interpretation of all these aspects (grading of varicoceles, volume discrepancy and parenchymal echotexture), assessed in a short series of follow-up scans, is the only method to adequately select patients likely to benefit from varicocele repair.

7.5 Hypogonadism

Male hypogonadism is characterised by reduced testicular function: an inability to produce testosterone, spermatozoa or both. Male hypogonadism may be caused by a failure of the testes to respond to stimulation, inadequate release of secretory hormones by the pituitary gland (gonadotropins) or hormone resistance. Although male infertility is commonly considered a problem involving only seminiferous tubular function and spermatogenesis, evidence from men with various forms of tubular disorder or varicocele has demonstrated an exaggerated response of both serum LH and FSH to LHRH. Although total serum testosterone levels are normal in these subjects, the increased gonadotropin response to LHRH indicates a failure in both Leydig cell and seminiferous tubular function. Some compensation for the impaired Leydig cells must occur that is sufficient to return testosterone levels to normal under the influence of increased LH stimulation. These findings confirm that infertile men often possess primary gonadal failure of a subtle nature. Careful ultrasound examination of the testes reveals recognisable features. There is a correlation between the non-uniform reduced reflectivity of the testicular texture and a tendency towards raised gonadotropin levels (Fig. 7.17). The higher frequency of discrete or diffuse areas of Leydig cell in patients with gonadal dysgenesis syndrome, detectable as scattered small hypoechoic foci within the inhomogeneous testes, is described in Chap. 3. This is probably an attempt at compensation for the impaired testicular function.

As hypogonadism has various causes, the appearance of the testis of these subjects can also differ significantly. We present a selection of cases showing the wide variations seen in US pictures in an attempt to correlate the morphological, hormonal and functional features of the hypogonadal testis.

Klinefelter's syndrome is the most frequent chromosome abnormality in men, present in 1 in 500 male neonates. Men with the syndrome usually have small testes (testicular volume of 2–6 mL), which are intrinsically abnormal. Testicular echogenicity (Fig. 7.18) can vary according to the severity of the disease (less pronounced in mild mosaics). The vast majority of sufferers are azoospermic, although recent sperm retrieval

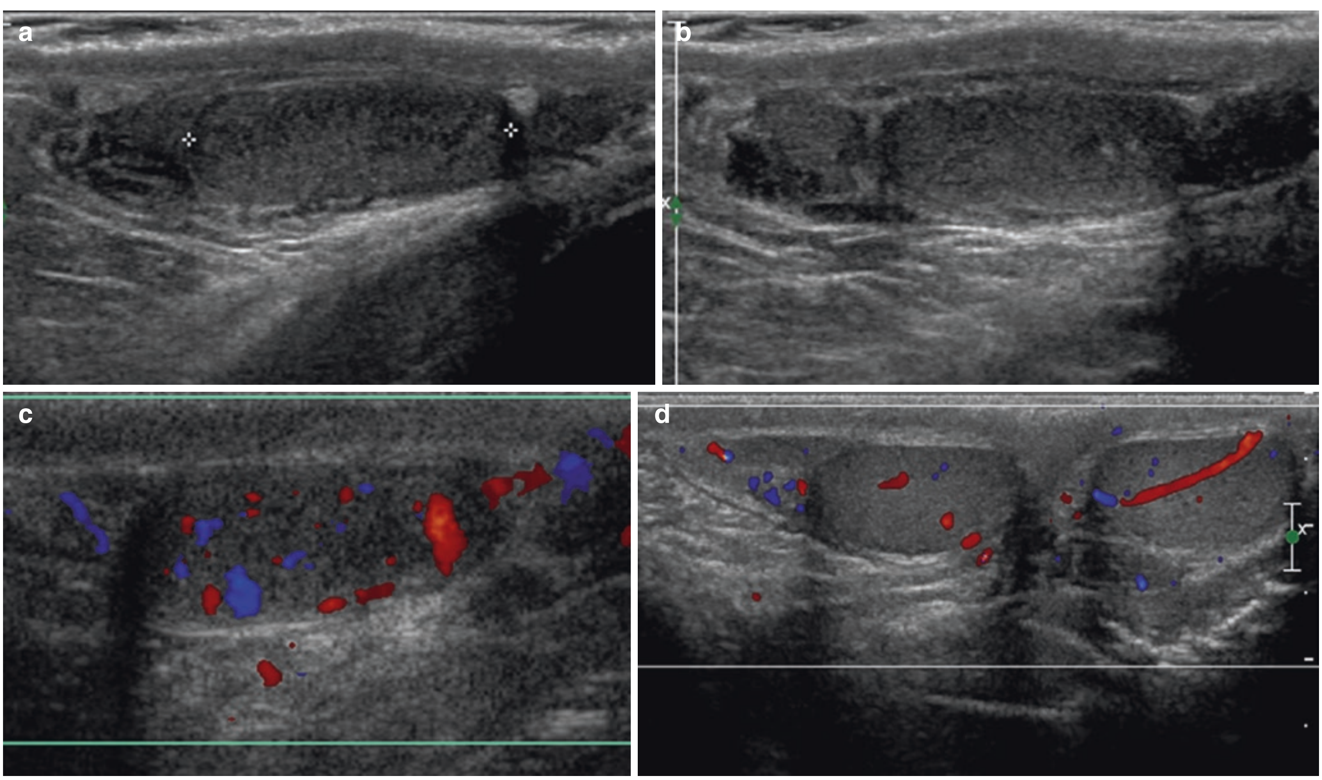

Fig. 7.17 Klinefelter's syndrome. Both testicles show hypoechoic, strongly uneven structure with possibly stromal and vascular hypoechoic areas and some hyperechoic spots, as frequently seen in gonadal dysgenesis syndromes (**a**, **b**). Note the increased vascularisation with irregular vascular spots (**c**, **d**)

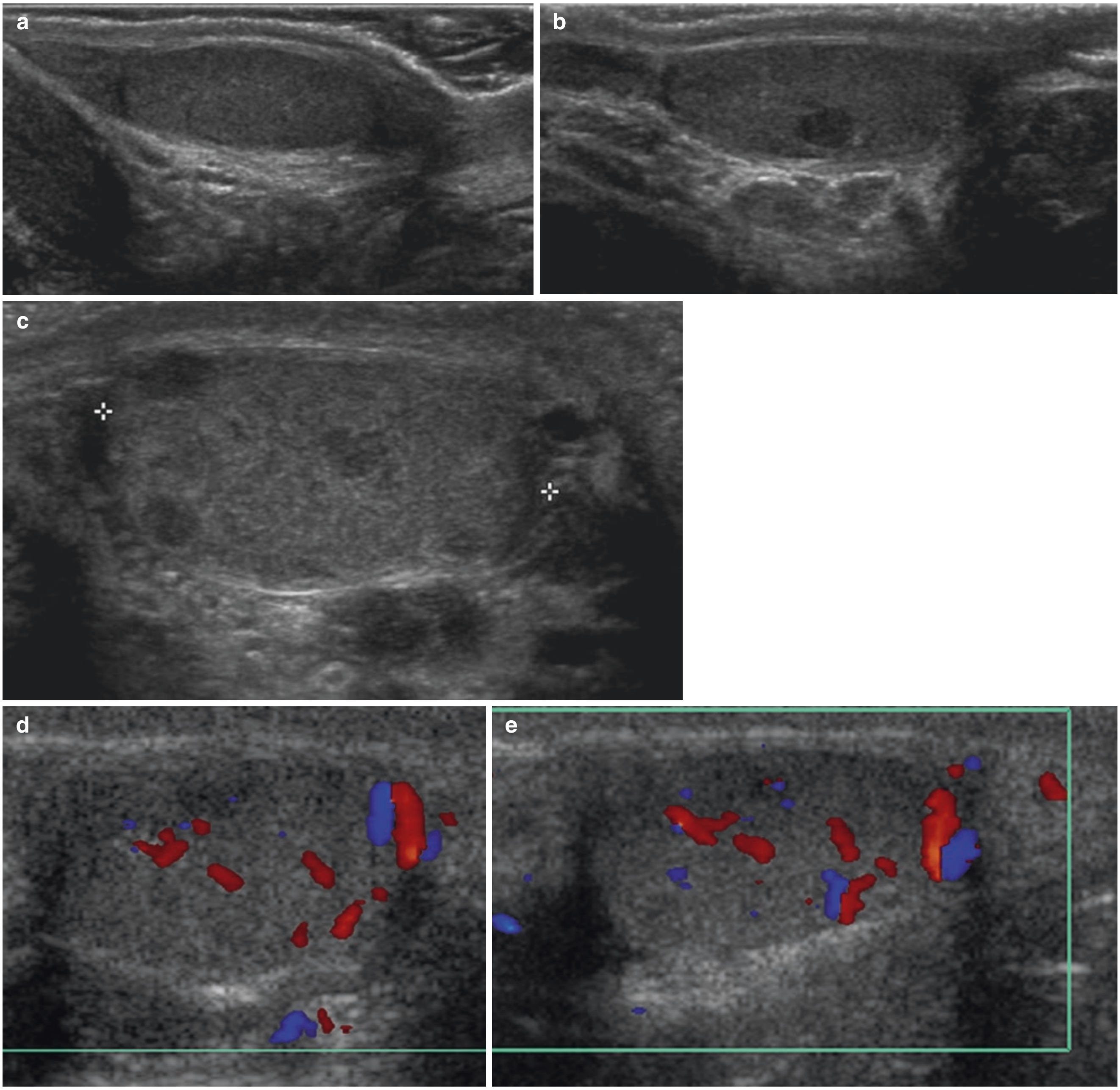

Fig. 7.18 Klinefelter's syndrome. US scan of the testis of an 18-year-old man revealed extremely small, atrophic testes (0.68 cm^3), with hypoechoic echotexture typical of Klinefelter's syndrome (**a**, **b**) and, on the left, a hypoechoic lesion (**b**). Panels **c**–**e** show a further case of Klinefelter's syndrome, in a 20-year-old man. Testicular texture on US is non-uniform (**c**), and vascularisation (**d**, **e**) is increased, likely due to the very high circulating LH and FSH levels

techniques have shown that isolated foci of spermatogenesis can be present in azoospermic men with Klinefelter's syndrome. These cells, even if immature, can be ultimately used to fertilise oocytes in assisted reproduction [19].

The testis is typically seen as a heterogeneous irregular testicular pattern with diffuse foci of hyper- and hypoechoic parenchyma [20]. Testicular histopathology typically reveals atrophy with fibrotic, hyalinised seminiferous tubules and Leydig cell hyperplasia. Clearly, these testicular parenchyma features are reflected in the very irregular hyper- and hypoechoic echogenicity observed on ultrasound scanning. Intratesticular arterial blood flow in men with Klinefelter's syndrome is different from that of the normal testis. It is unclear whether a vascular pattern of high resistance is present prior to the development of testicular atrophy or if it develops during this process. Another possibility is that the high-resistant intratesticular blood flow is secondary to parenchymal atrophy, with fibrotic, hyalinised seminiferous tubules [20].

Hypogonadism due to primary testicular failure is characterised by small testes with mild to severe echo structure abnormalities (the testis usually appears heterogeneous and hypoechoic) (Fig. 7.19). The colour and power Doppler signal is irregularly distributed. As the testis is reduced in size, the vessels are closer to one another, the somatic component is normally increased and focal/diffuse Leydig cell hyperplasia is commonly seen. These features give the appearance of an enhanced flow through the affected testis. The elevated gonadotropins that accompany these cases might be responsible for the increased vascularisation, through the putative effect of LH on testicular vascularisation (Figs. 7.20 and 7.21).

In contrast, secondary hypogonadism due to gonadotropin deficiency or insensitivity is associated with reduced blood flow to the testis (Fig. 7.22). The testicular parenchyma appears less homogeneous and in the former there is a strong clear response to exogenous gonadotropin treatment. In subjects sensitive to gonadotropins, there is a rapid increase in testicular volume (Fig. 7.23). Recent studies suggest that subjects with functional hypogonadotropic hypogonadism may benefit from this treatment. In fact, we have observed an increase in reflectivity of the testis and parenchymal blood flow. This confirms that some of the alterations that are clearly visible in the architecture and vascularisation of the testes of some infertile men are the results of a functional impairment in hormonal signalling.

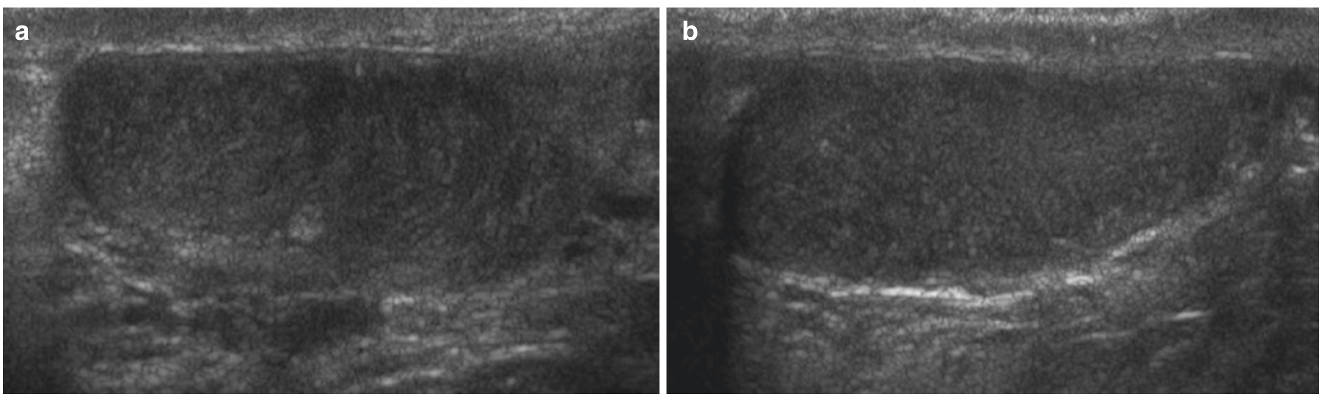

Fig. 7.19 Post-orchitis hypogonadism. US scan of the testes of a 74-year-old man showed non-uniform, small and atrophic testes (**a**, **b**). Atrophy was due to previous bilateral orchitis. Plasma testosterone levels were low and the patient started testosterone therapy

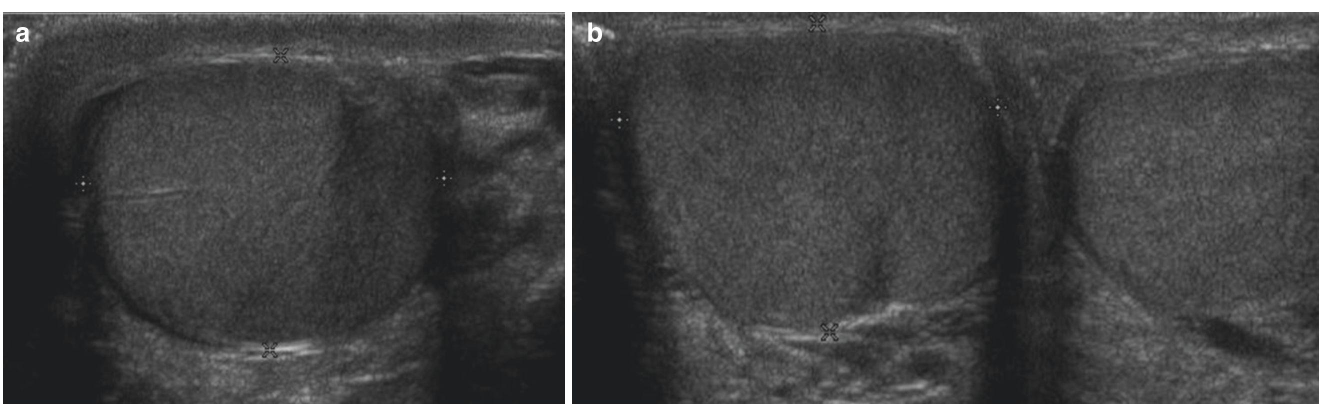

Fig. 7.20 Kennedy syndrome (**a**, **b**). Kennedy syndrome is a neurodegenerative disease associated with decreased effective testosterone due to a defective androgen receptor

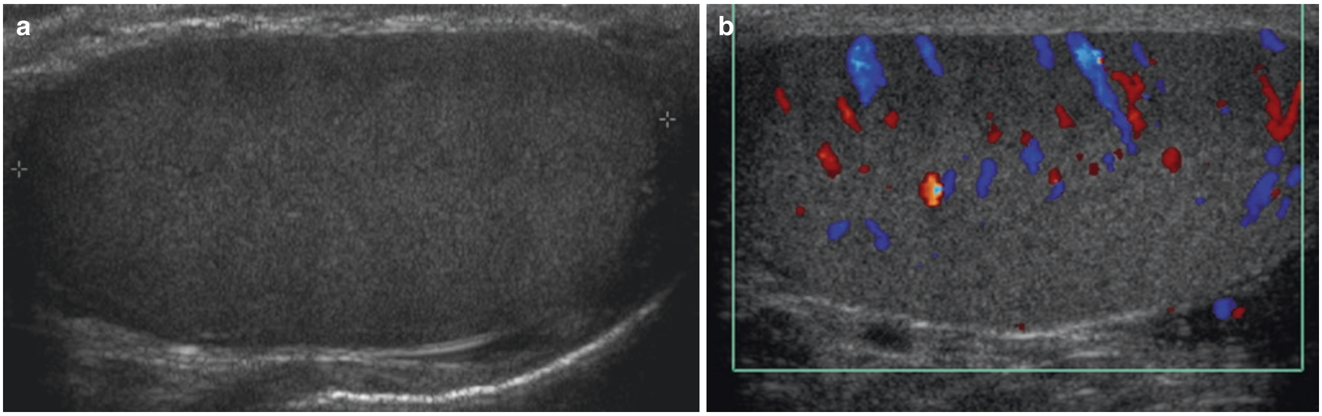

Fig. 7.21 Klinefelter's mosaicism. US scan of a 18-year-old man, referred for azoospermia. Ultrasound revealed relatively normoechoic testes with homogeneous echotexture (**a**) and increased and disorganised vascularisation (**b**). The relatively elevated gonadotropin level explains the increased testicular vascularisation. Karyotyping revealed a 4% chromosomal mosaicism (46, XY/47, XXY)

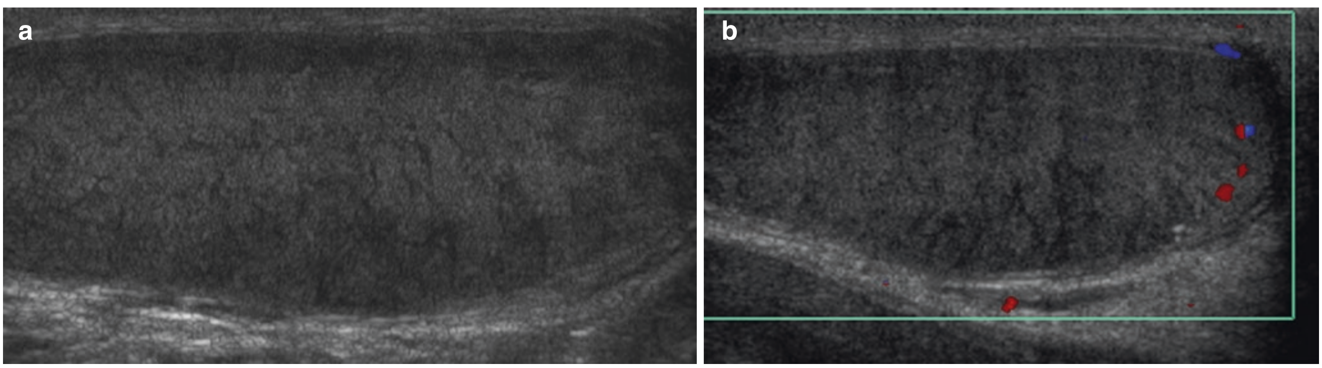

Fig. 7.22 Kallmann idiopathic hypogonadotropic hypogonadism. Patients with IHH may have a history of partial progression through puberty. Serum LH and FSH levels are low-normal or decreased in post-pubertal patients. Gonadotropin levels are inappropriate with respect to serum levels of testosterone or oestradiol. Panels **a** and **b** show the testis of a 32-year-old man with idiopathic hypogonadotropic hypogonadism, treated since puberty with testosterone enanthate. Testicular texture on US is non-uniform and hypoechoic (**a**) and vascularisation (**b**) is nearly absent, due to the low LH and FSH levels

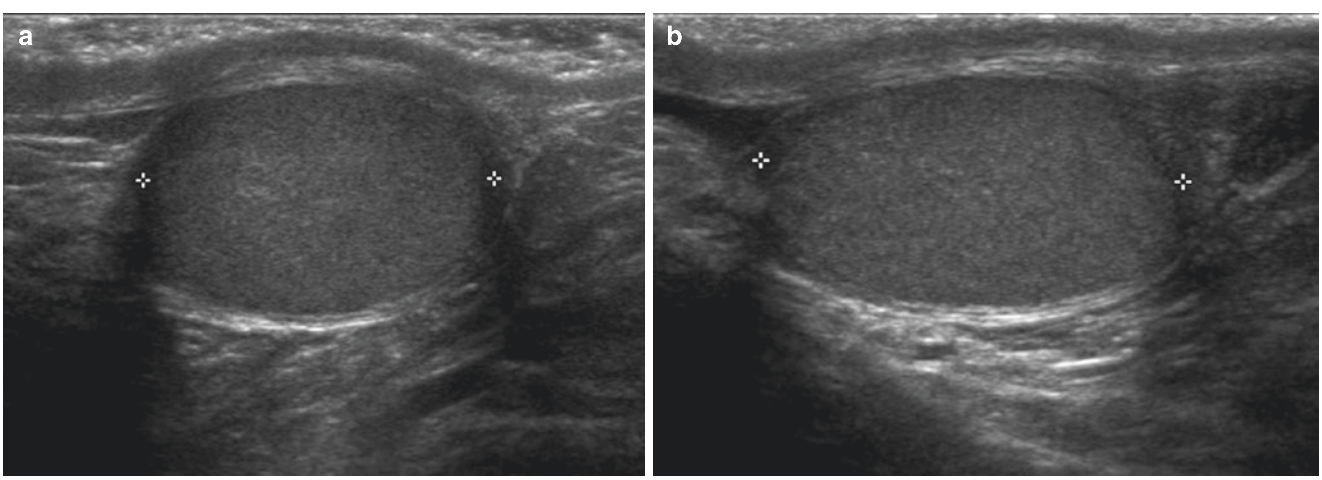

Fig. 7.23 US scan of a patient with Kallmann's syndrome before (**a**, **b**) and after (**c**, **d**) medical therapy with gonadotrophins. Note the volumetric increase of both testes and the change in reflectivity of the testes that show mid-high echoes compared to basal US

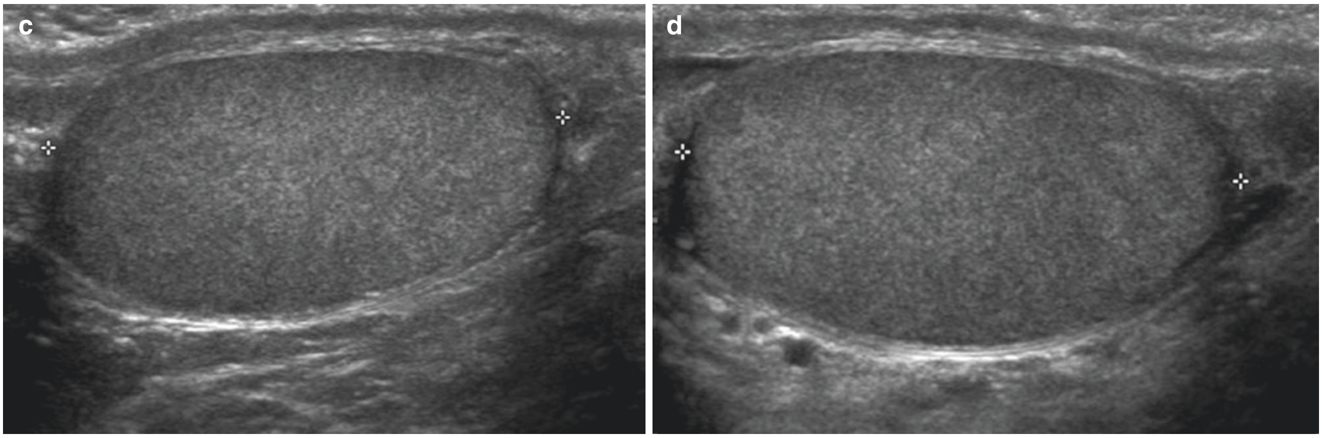

Fig. 7.23 (continued)

Key Messages

- Differences from adults in anatomy and pathology must be considered when evaluating the paediatric scrotum. These are essentially due to the US appearance of the undeveloped testis.
- Compliance is an issue when performing US in children. It may restrict the examination time, and children's movements can generate artefacts in colour Doppler US. Thus, in contrast with adults, some authors suggest beginning the evaluation with the affected side, in order not to miss any disorder.
- Cryptorchidism is a relatively common finding in paediatric practice, whose aetiology includes some not entirely understood defects in the hormonal signalling involved in testicular migration. Failure of the testis to descend into the scrotum and patency or anomalous closure of the processus vaginalis results in cryptorchidism, inguinoscrotal hernia and hydrocele.
- The undescended testis is generally smaller and hypoechoic to the contralateral – not only due to the condition itself but also as the result of greater US attenuation by the overlaying tissues – and frequently appears elongated and of reduced consistency (easily compressed by the probe).
- Two important complications of undescended testis are infertility and testicular malignancy. Intra-abdominal testis carries a four times greater risk of development of malignancy than an inguinal testis. The most frequent malignant degeneration are seminomatous tumour and embryonal cell carcinoma.
- Differential diagnosis in exploring a hypoechoic oval mass in the inguinal canal is easily made by (1) evaluating the mobility along the inguinal canal, which is present for the testis and absent for the lymph node (easily found by evoking the cremasteric reflex through stimulating the skin on the inner side of the thigh), and (2) identifying the mediastinum testis that confirms that the structure is the testis and not a lymph node or the pars infravaginalis gubernaculi, which is the inferior bulbous portion of the gubernaculum testis.
- Torsion can occur in undescended testes, especially in those with malignant degeneration.
- It is important to discriminate between truly cryptorchid and retractile testes. The room should therefore be adequately heated before performing the scans and the exam should be repeated twice on separate occasions.
- An inguinal hernia occurs when tissue or part of the intestine pushes through a weak spot in the abdominal wall in the groin area, causing a bulge in the groin or scrotum; its prevalence is much higher in preterm neonates.
- In neonates and infants, virtually all hydroceles are congenital and are associated with a patent processus vaginalis, which allows peritoneal fluid to enter the scrotal sac.
- Closure of the processus vaginalis above the testis and below the internal inguinal ring leads to a less common type of hydrocele, also known as spermatic cord cyst.
- Estimation of testicular volume in boys is very useful for evaluating normal pubertal development. The best formula to calculate peri-pubertal testicular volumes is $L \times D \times H \times 0.71$.
- Testicular volume during preadolescence is constant. This can create some concern, as the rest of the body grows linearly, but not the external genitalia. At the onset of puberty, the testis suddenly increases in size at an almost exponential rate.

- The increase in echogenicity during puberty is primarily the result of growth of the seminiferous tubules, which increase in diameter and develop a lumen.
- The changes in arterial inflow generally anticipate the increase in volume and echogenicity, suggesting that this event is hormonally driven and is the *primum movens* of puberty.
- In varicoceles in the adolescent, ultrasonography is the only objective method to assess testicular damage, measured as growth arrest or impaired echogenicity.
- It is very important to monitor testicular volumes closely, to check for any widening of the size difference between the affected and unaffected testis.
- Male hypogonadism is characterised by a reduction in testicular function: an inability to produce testosterone, spermatozoa or both. It may be caused by a failure of the testes to respond to stimulation, inadequate release of secretory hormones by the pituitary gland (gonadotropins) or hormone resistance.
- Infertile men often possess primary gonadal failure of a subtle nature.
- The higher frequency of discrete to diffuse areas of Leydig cell hyperplasia in patients with gonadal dysgenesis syndrome is probably an attempt to compensate for the impaired testicular function.
- Klinefelter's syndrome is the most frequent chromosomal abnormality in men, present in 1 in 500 male newborns.
- The typical appearance of the testes in Klinefelter's syndrome is small and hard, with an irregular heterogeneous echotexture scattered with diffuse foci of hyper- and hypoechogenic parenchyma.
- In primary hypogonadism, the accompanying elevated gonadotropin level might be responsible for the increased vascularisation, through various mechanisms, including the putative effect of LH on testicular vascularisation.
- In secondary hypogonadism blood flow is typically reduced, due to gonadotropin deficiency or insensitivity. The testicular parenchyma appears less homogeneous and responds well to exogenous gonadotropin treatment.

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Advanced Ultrasonographic Techniques

8

8.1 Introduction

Ultrasound is a sensitive and accurate technique for the evaluation of testicular anomalies and is widely accepted as the first-line imaging technique for many testicular diseases. Occasionally, however, ultrasound findings are equivocal. Although most focal lesion are malignant and require radical surgery, recognition of benign lesions may be challenging, especially if non-palpable [1].

Recently, some novel methods of investigation have been implicated in order to distinguish benign from malignant testicular lesions. Contrast-enhanced ultrasound (CEUS) is such a new technique taking advantage of intravenous microbubble contrast to provide information on vascular enhancement characteristics of testicular lesions. The benefits of this technique have already been proven in vascular and abdominal imaging where it has been shown to be very sensitive in depicting the presence of vascularity in a lesion.

A further development is the concept of tissue elastography, essentially the assessment of the 'hardness' of a particular tissue. This technique has been used in the thyroid, prostate, liver and breast and lately in the testis [2].

Although in general there are no ultrasound findings that are entirely diagnostic, CEUS and elastography may be helpful in better characterising intratesticular lesions. With increasing understanding, combining these modalities in the ultrasound evaluation of testicular pathology may allow a personalised follow-up plan or targeted ultrasound-guided excision biopsy when thought appropriate, thus potentially reducing the number of unnecessary orchiectomies.

8.2 Contrast-Enhanced Ultrasound (CEUS)

The use of contrast-enhanced ultrasound (CEUS) is firmly established in many organs in the body but less widely used in the testis. CEUS is an ideal technique to ascertain the vascular supply to an organ, as the contrast agent is purely an intravascular agent and increases the sensitivity of detection of blood flow.

CEUS is an inexpensive, easy-to-use diagnostic technique and can be performed immediately after the conventional Doppler study [3, 4]. CEUS has the potential of assessing intratumoural microvascularisation, thus improving conventional US [5].

The contrast media [SonoVue (BR1, Bracco, Milan, Italy), Definity (Bristol-Myers Squibb Medical Imaging, N. Billerica, Massachusetts, USA) or Sonazoid (Daiichi-Sankyo, Tokyo, Japan)] is administered intravenously (2.4–4.8 mL), using a 20-gauge cannula, followed by a 10 mL saline solution bolus. After approximately 20 s, the microbubbles reach the intravascular space of the testis and may be visualised in the parenchyma up to 2–3 min after administration, and then the intensity progressively decreases (washout phenomenon) [5].

The harmonic imaging and 'pulse inversion' techniques used during CEUS suppress normal tissue and enhance the returning echoes of the microbubbles, resulting in a purely vascular perfusion image. Split-screen technology is used and imaging is recorded on a cine loop for at least 120 s for later review.

After microbubble administration testis and epididymis enhance quickly and intensively. The arteries enhance first, followed within few seconds by complete fill-in of the parenchyma.

There is no significant accumulation of microbubbles in the parenchyma of scrotal structures, and enhancement typically fades within about 3 min [5].

CEUS offers additional information in the differential diagnosis of testicular lesions, being superior to non-contrast US, by assessing intratumoural microvascularisation.

In particular, quantitative contrast-enhanced US evaluation improves substantially the diagnostic accuracy of non-enhanced US (up to 93% in the differential diagnosis of non-palpable tumours [6]).

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In several studies [1, 3, 5–10], CEUS has found specific properties of testicular tumours. Both benign and malignant tumours tend to enhance strongly (Fig. 8.1). This helps in the differential diagnosis against non-neoplastic lesions (ischemia, abscesses, cysts, acute scrotal pathology). The finding of a focal intratesticular lesion that is avascular both on colour Doppler and CEUS should raise the possibility of truly avascular lesion (e.g. epidermoid cyst, segmental testicular infarction, etc.) [1, 3, 6]. On the other hand, the presence of necrosis or extensive calcifications, as find in microlithiasis or in burnt-out tumours, can alter microbubble diffusion inside the testis and the lesion, leading to ambiguous CEUS results (Fig. 8.2).

In vascularised neoplasms, the contrast material dynamics may vary in different histologic groups. In particular, malignant lesions are characterised by fast enhancement followed by a rapid washout, whereas benign tumours, despite of presenting an equally rapid enhancement, feature a prolonged washout pattern. This seems to be a distinctive feature of stromal tumours and has been attributed to local oestrogen generation and endocrine gland-derived vascular endothelial growth factor (EG-VEGF), a human angiogenic mitogen, strongly expressed in Leydig cells, which may play a role in Leydig cell tumour growth [11].

The intensity of contrast enhancement contributes to the differential diagnosis between neoplastic and non-neoplastic lesions, which are generally not vascularised or similarly enhanced as the surrounding parenchyma (Fig. 8.3), whereas temporal perfusion dynamics of contrast material enhancement help in the differential diagnosis between malignant tumours and benign lesions.

Although at present, the necessary expertise in performing CEUS examination is limited to specialist centres, there is growing acceptance of CEUS as an essential technique in clinical practice [12].

8.3 Elastosonography

In the last few years, the US techniques strain elastography (SE) and shear wave elastography (SWE) have gained impetus as supplementary, commonly available add-on tools for various applications [1, 3, 10, 13–19]. These techniques provide real-time non-invasive tissue characterisation performed at the same time and as an adjunct to conventional US imaging. Shear wave elastography uses conventional ultrasound imaging techniques to analyse the shear waves generated inside the human body by an ultrasound burst; the propagation speed of the shear waves

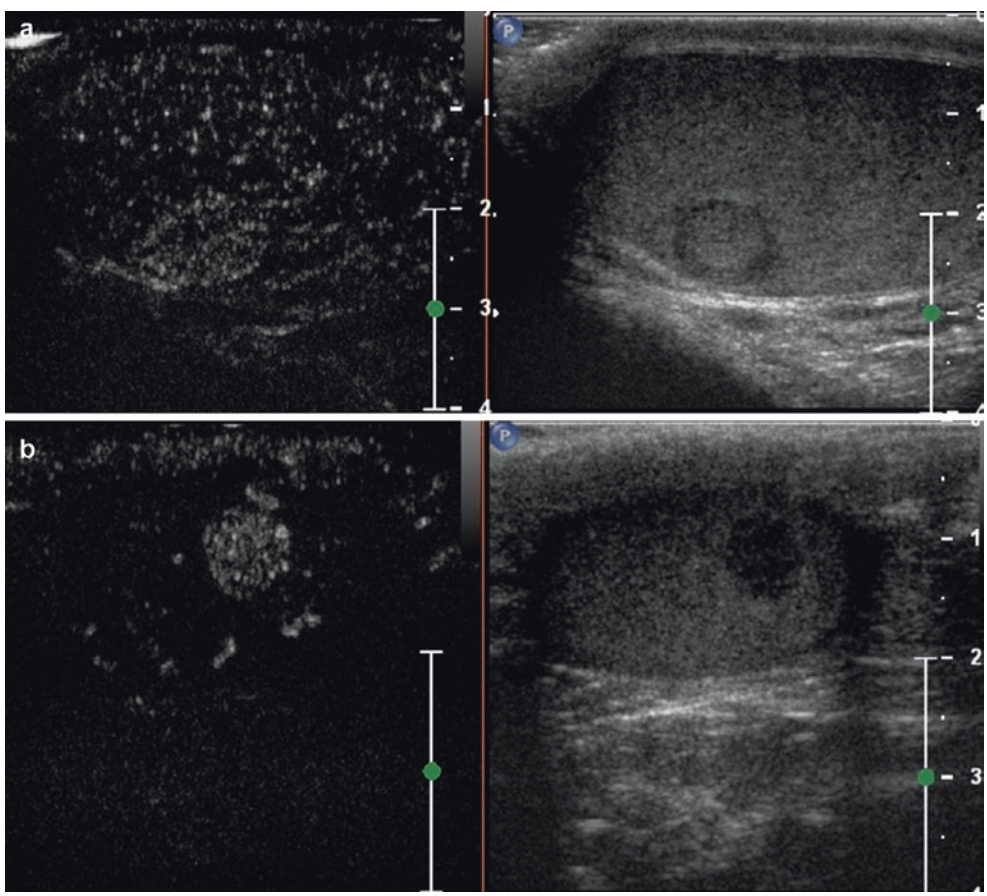


Fig. 8.1 Both benign and malignant tumours tend to enhance strongly. In panel **a** a seminoma is shown and in panel **b** a Leydig cell tumour

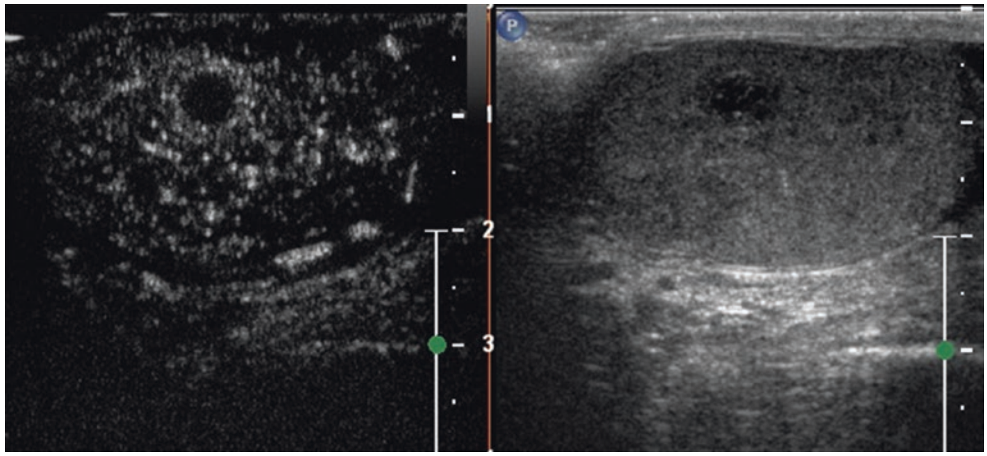


Fig. 8.2 The presence of necrosis altered microbubble diffusion inside the lesion, which was a seminoma at histology

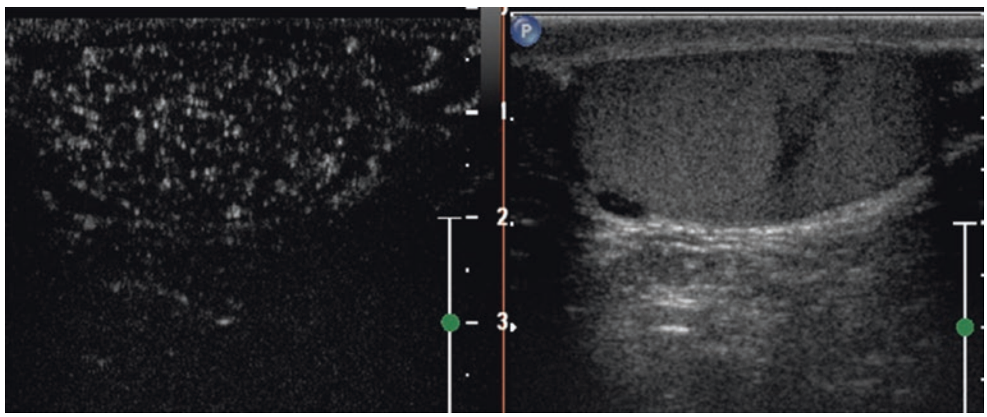


Fig. 8.3 Non-neoplastic lesions are generally not vascularised or similarly enhanced as the surrounding parenchyma

directly correlates with tissue stiffness. SE evaluates the relative elasticity of different tissues in a selected region of interest by using a fast cross-correlation technique and a combined autocorrelation method. It creates an elastogram that is superimposed to the B-mode ultrasound image of the tissue and updated in real time. Its output includes a colour-coded representation (qualitative assessment) of the lesion (Fig. 8.4) and a semiquantitative characterisation by strain ratio, calculated as the ratio of the surrounding parenchyma to the lesions and providing a measure of lesion stiffness [3, 10].

After performing a conventional US, the elastography settings are applied, and SE images and cine loops of each lesion should be saved for qualitative and semiquantitative evaluation of dynamic features. To assess the elasticity score (ES) of a testicular abnormality, the physicians operate the transducer with slight pressure, maintaining contact with the skin, and a perpendicular position to the lesion; a large elastogram region of interest (ROI) box is used to increase the potential number of samples for a strain ratio calculation; at the same time, a compression bar can be visualised on the screen during the examination in order to assess the quality and validity of the operator's freehand pressure. When this bar remains stable for at least 5–10 s, the operator should acquire 10-s cine loops that should be used for subsequent analysis [13].

A colour scale from red through green to blue is used for the elastograms. Red indicates the highest elastic strain (softest tissue) and blue indicates no strain (hardest tissue). Colour-coded elastogram images are graded visually on the stiffness of the nodules relative to the surrounding parenchyma, assigning an elasticity score (ES) to each image.

The normal testis demonstrates a homogeneous medium hardness at elastography [20]. Sometimes the glandular tissue below the tunica albuginea of the testicle presents less relative strain, displayed in light blue, probably due to the limited tissue displacement determined by the fibrous covering. Focal lesions can often be recognised as harder than the surrounding testicular tissue, with some important exceptions concerning hematomas or mixed fluid lesion [3, 21]. Normal testis parenchyma is hard, but compared with the even harder solid tumourous tissue, it has a relatively softer appearance, displaying areas of green and light blue [20].

High elasticity is generally characteristic of lesions which have the same compressibility of the surrounding tissue, intermediate elasticity indicates lesions that are mainly soft but have some stiff areas compared with the normal tissue, and no strain in the entire lesion (low elasticity) is generally

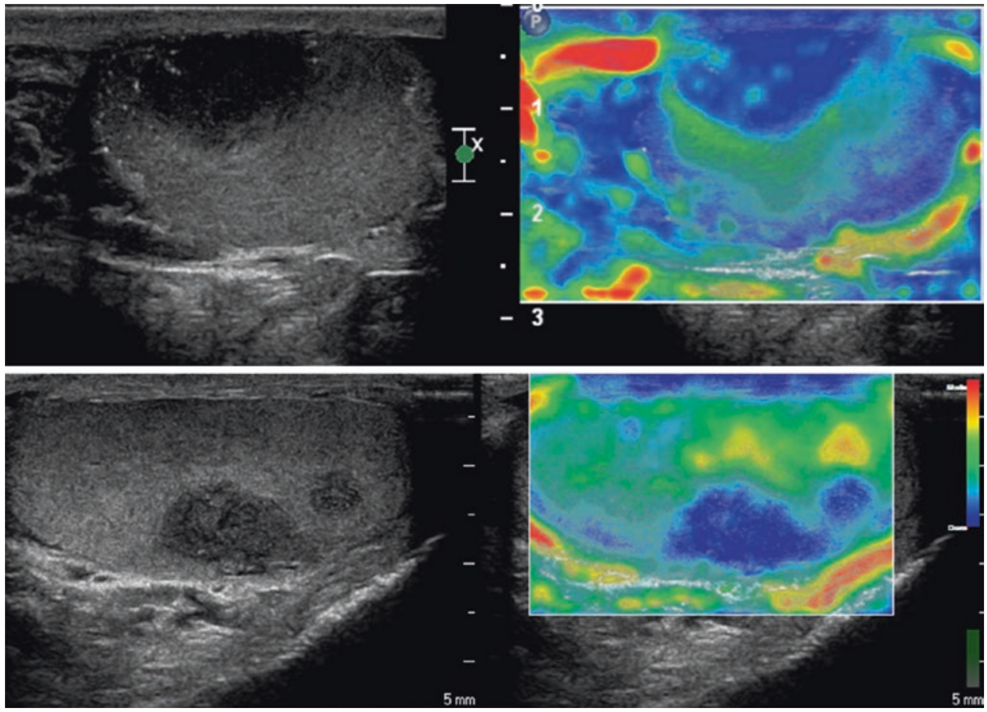


Fig. 8.4 Colour-coded representation of two lesions (both seminoma at histology). *Red* indicates the highest elastic strain (softest tissue) and *blue* indicates no strain (hardest tissue)

characteristic of neoplastic nodules (Fig. 8.5). Benign and malignant neoplasms show a certain overlap in hardness that does not allow a sufficiently confident differential diagnosis (e.g. epidermoid cysts) [3, 6]. Abscesses, partial infarction, Leydig cell hyperplasia, fibrosis and non-tense cysts are generally soft at elastosonography [10, 13].

SE could be considered in the context of evaluating incidental testicular lesions; qualitative assessment in conjunction with a careful clinical history and supplementary diagnostic evaluation might be helpful in differentiating neoplastic from non-neoplastic testicular lesions in challenging cases, but it cannot be used to discriminate benign from malignant neoplasms.

8.4 A Selection of CEUS and Elastography Applications in Testicular Pathology

8.4.1 Seminoma

Seminoma is the most common pure germ cell tumour. It accounts for 35–50% of all germ cell tumours. Seminomas, in comparison with non-seminomatous tumours, occur in a somewhat older population, with an average patient age of 40.5 years. During CEUS seminoma shows rapid enhancement and rapid washout of the contrast within the lesion. The enhancement is usually more intense than in the normal surrounding parenchyma (Figs. 8.6 and 8.7).

On elastography the lesion is normally well evident as a hard lesion (Fig. 8.8).

8.4.2 Non Seminomatous Germ Cell Tumours

Mixed germ cell tumours are much more common than any of the pure histological forms. Virtually any combination of cell types can occur. Embryonal carcinoma is the most common component and is often combined with one or more components of teratoma, seminoma, and yolk sac tumour. The B-mode features reflect the different components of these lesions, showing inhomogeneity, cystic areas, calcification and increased inhomogeneity.

On CEUS, mixed germ cell tumours are rapidly enhanced, but not in a homogeneously way (Fig. 8.9). In the presence of necrosis or extensive calcifications, as seen in burnout tumours, lesions may be unenhanced. In doubtful cases, a total body CT scan is recommended to search for metastases (Fig. 8.10).

On elastography, these tumours show a mixed pattern of elasticity, depending on the component evaluated (Fig. 8.11).

8.4.3 Sex Cord: Stromal Tumours

Approximately 4% of all testicular tumours arise from the cells forming the sex cords (Sertoli cells) and interstitial

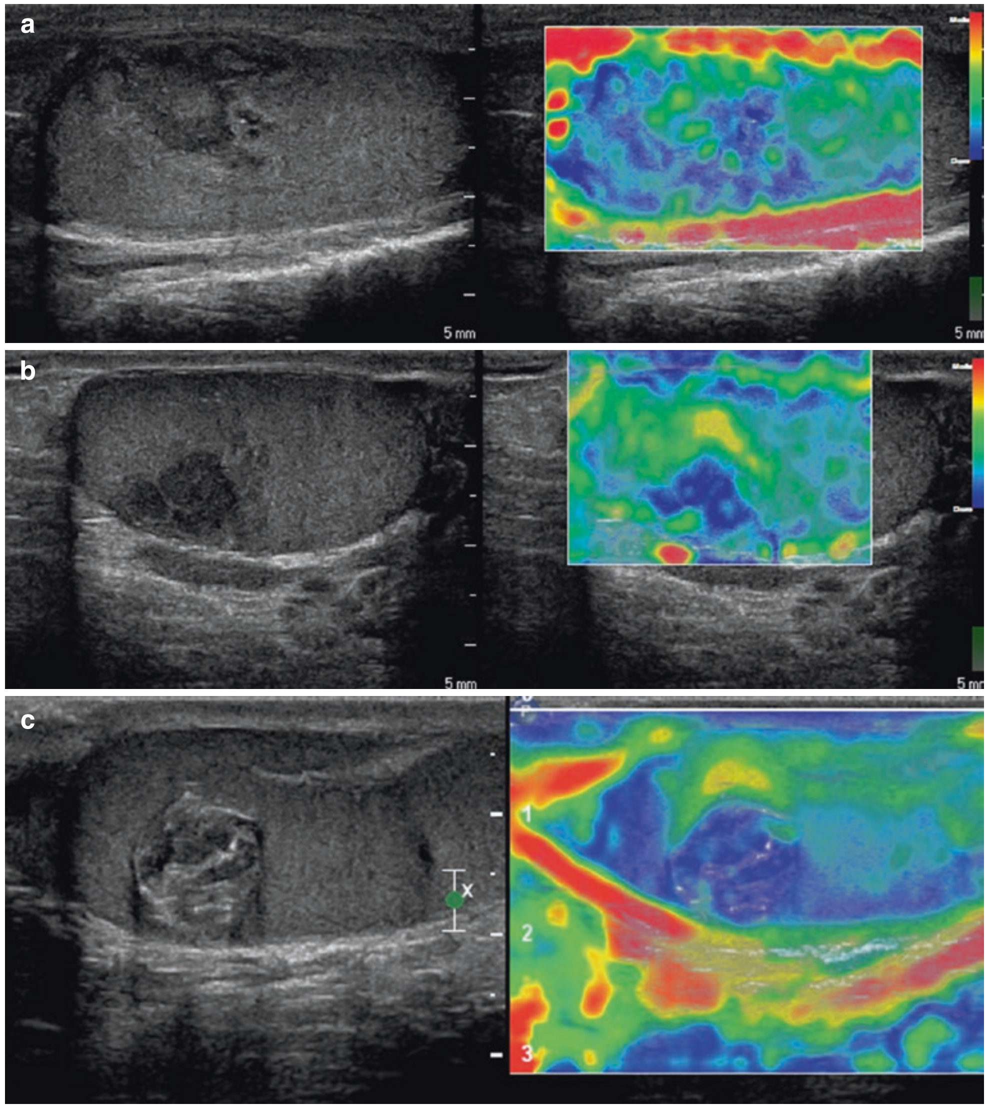


Fig. 8.5 High elasticity is generally characteristic of lesions which have the same compressibility of the surrounding tissue (panel **a**, intratesticular haematoma), intermediate elasticity indicates lesions that are mainly soft but have some stiff areas compared with the normal tissue, and no strain in the entire lesion (low elasticity) is generally characteristic of neoplastic nodules (panel **b**, seminoma; panel **c**, epidermoid cyst)

stroma (Leydig cells). Leydig cell tumours (LCTs) are the most common in this group, accounting up to 10% of all testicular tumours, while Sertoli cell tumours are relatively rare, constituting less than 1% of testicular tumours.

On CEUS LCTs show fast enhancement and delayed washout of the contrast medium that persist longer than the normal testicular parenchyma. The enhancement is higher than the enhancement of the surrounding tissue (Figs. 8.12 and 8.13).

On elastography LCT are generally hard lesions, but they can show also an intermediate stiffness (Fig. 8.14).

Sertoli cell tumours in general appear on CEUS like seminomas, showing a rapid enhancement and a rapid washout.

8.4.4 Epidermoid Cysts

Epidermoid cysts are relatively uncommon benign testicular tumours that account for less than 1% of all testicular neoplasms. Histologically epidermoid cysts are cystic cavities that contain desquamated keratinised epithelium and are lined by stratified squamous epithelium without skin appendages.

On CEUS this lesions are completely unenhanced, sometimes with a rim enhancement, whereas on elastography they show hardness (Fig. 8.15).

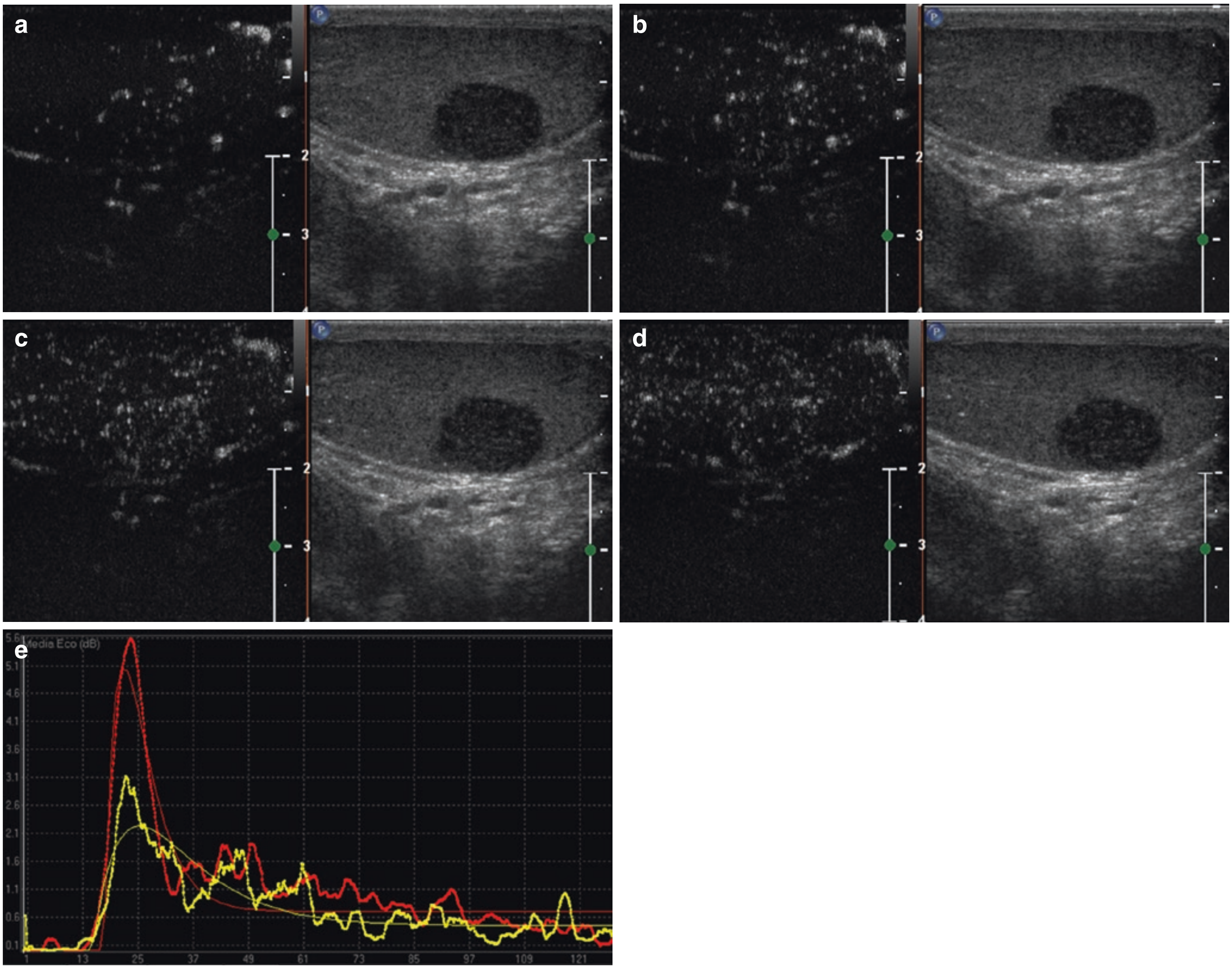


Fig. 8.6 Surgically proven seminoma. The tumour enhance rapidly (**a**, **b**), followed by complete fill-in (**c**) of the lesion. A rapid washout is then appreciable (**d**). Contrast pulse inversion harmonic time-intensity curves of investigated lesion (*red*) and healthy parenchyma (*yellow*) show rapid time of washout

8.4.5 Acute Scrotum

CEUS demonstrate the avascularity of the infarcted area, relative to the surrounding testicular parenchyma. The same appears to intratesticular haematoma and abscess that are avascular on CEUS (Fig. 8.16). Abscess can present rim of enhancement. On elastography they generally show a mixed pattern of firmness (Fig. 8.17).

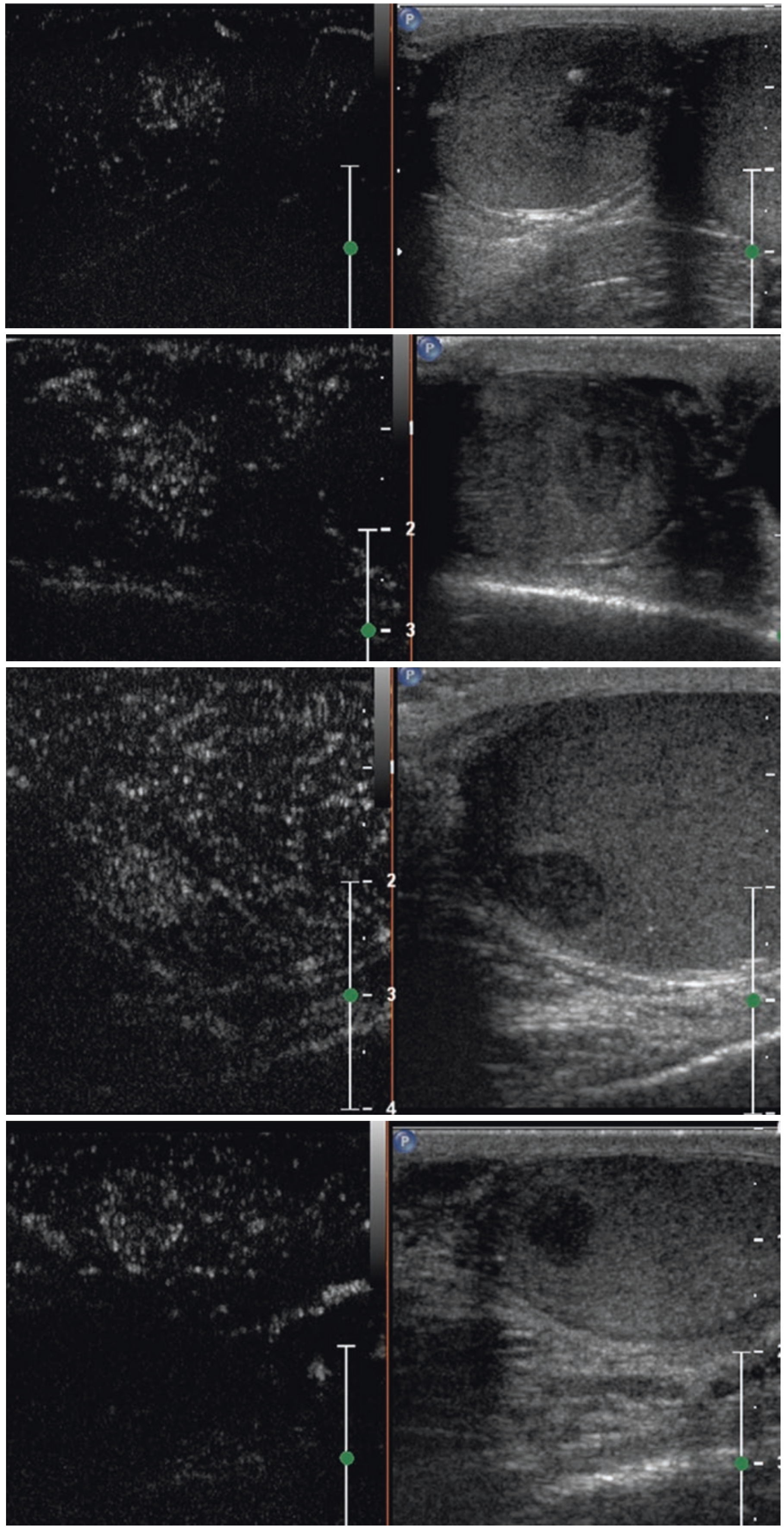
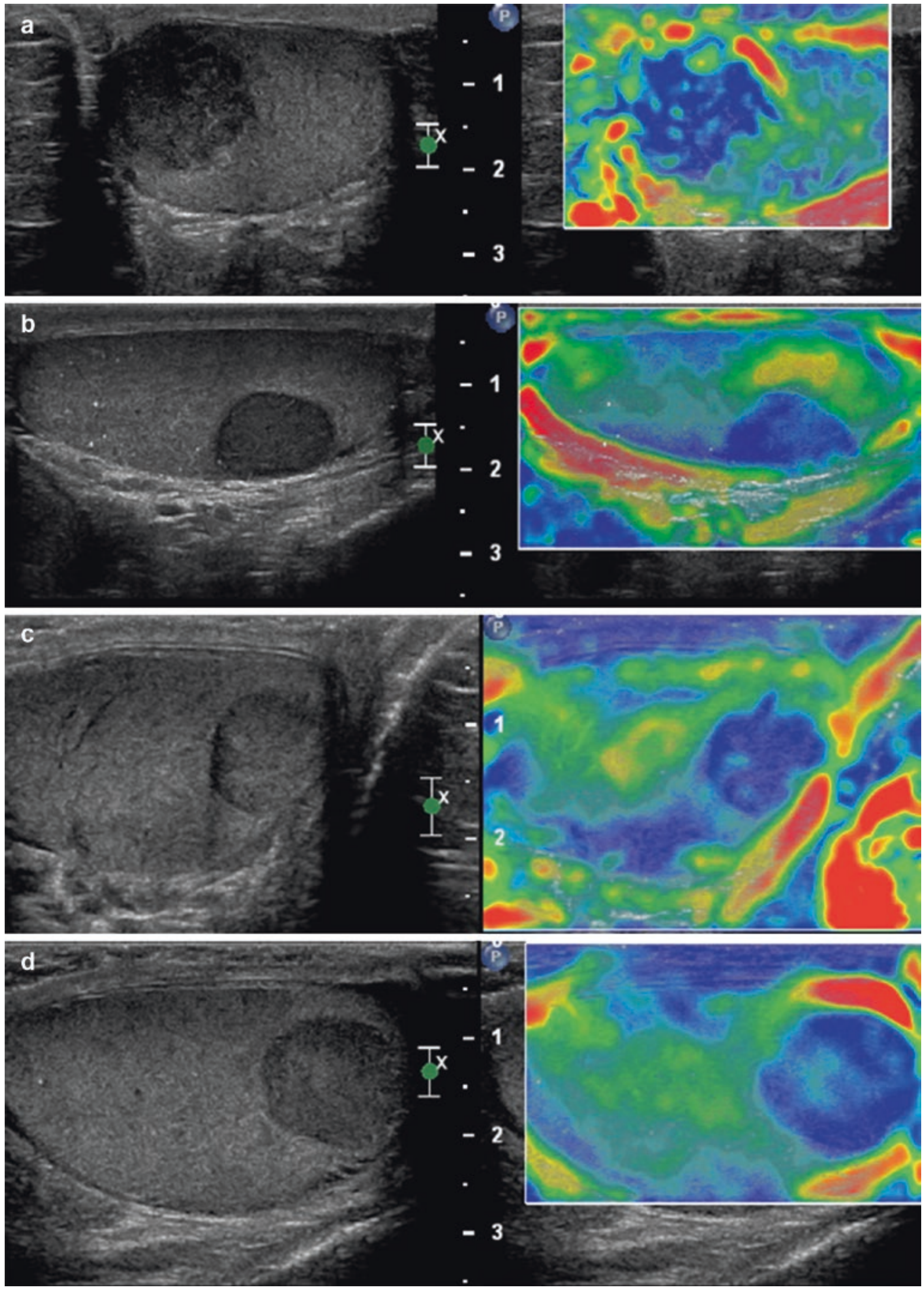


Fig. 8.7 Further cases of seminoma enhancing at CEUS

Fig. 8.8 Seminoma generally appears 'hard' on tissue elastography (**a**–**c**)



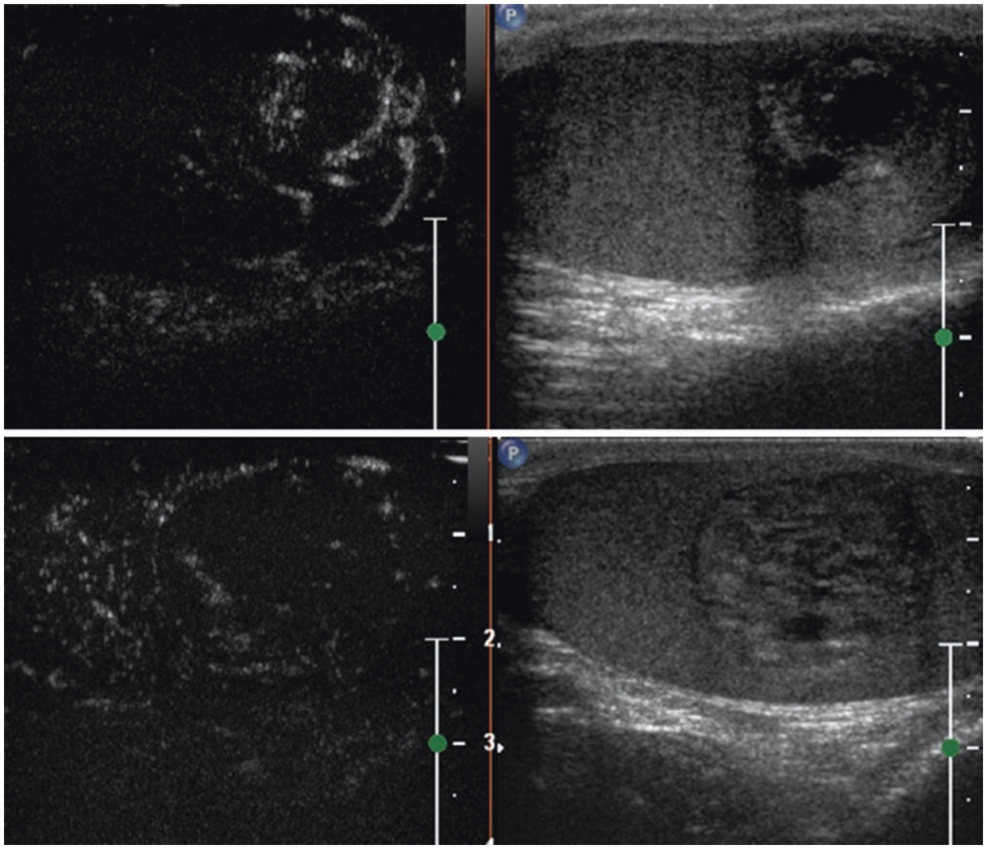


Fig. 8.9 On CEUS, particulate movement of contrast is seen throughout the lesions, confirming the vascularity is present within all the solid components of the lesions

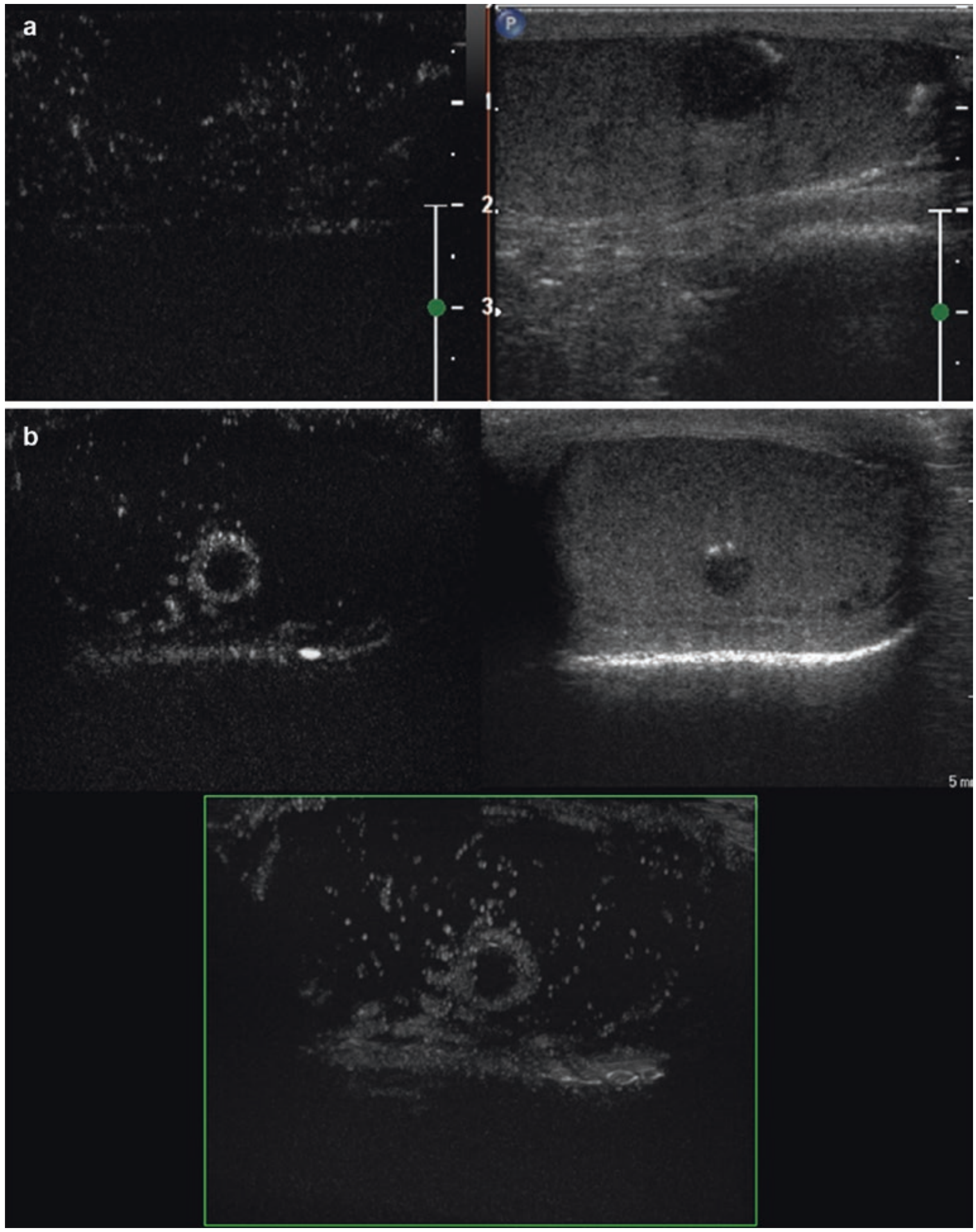


Fig. 8.10 In the presence of necrosis or extensive calcifications, as seen in burnout tumours, lesions may be unenhanced (below are shown two regressed embryonal carcinoma). Microvascular image (MVI) over time gives the observer additional information that is not present in static images. In panel **b** MVI (*green square*) built up during the entire exam (approximately 120 s) confirms the complete absence of intralesional vascularisation

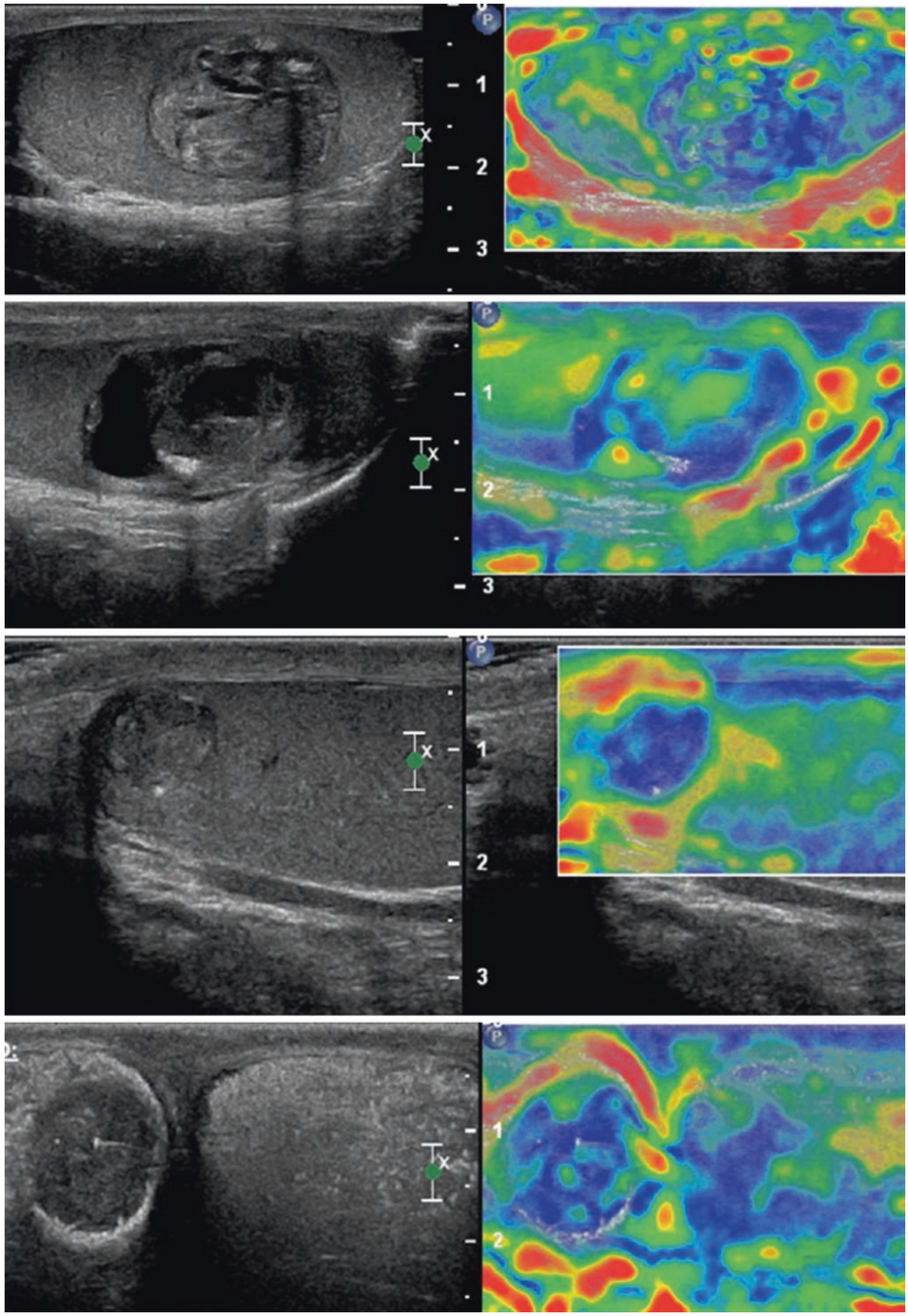


Fig. 8.11 In non-seminomatous germ cell tumours, the pattern of elasticity depends on the component evaluated

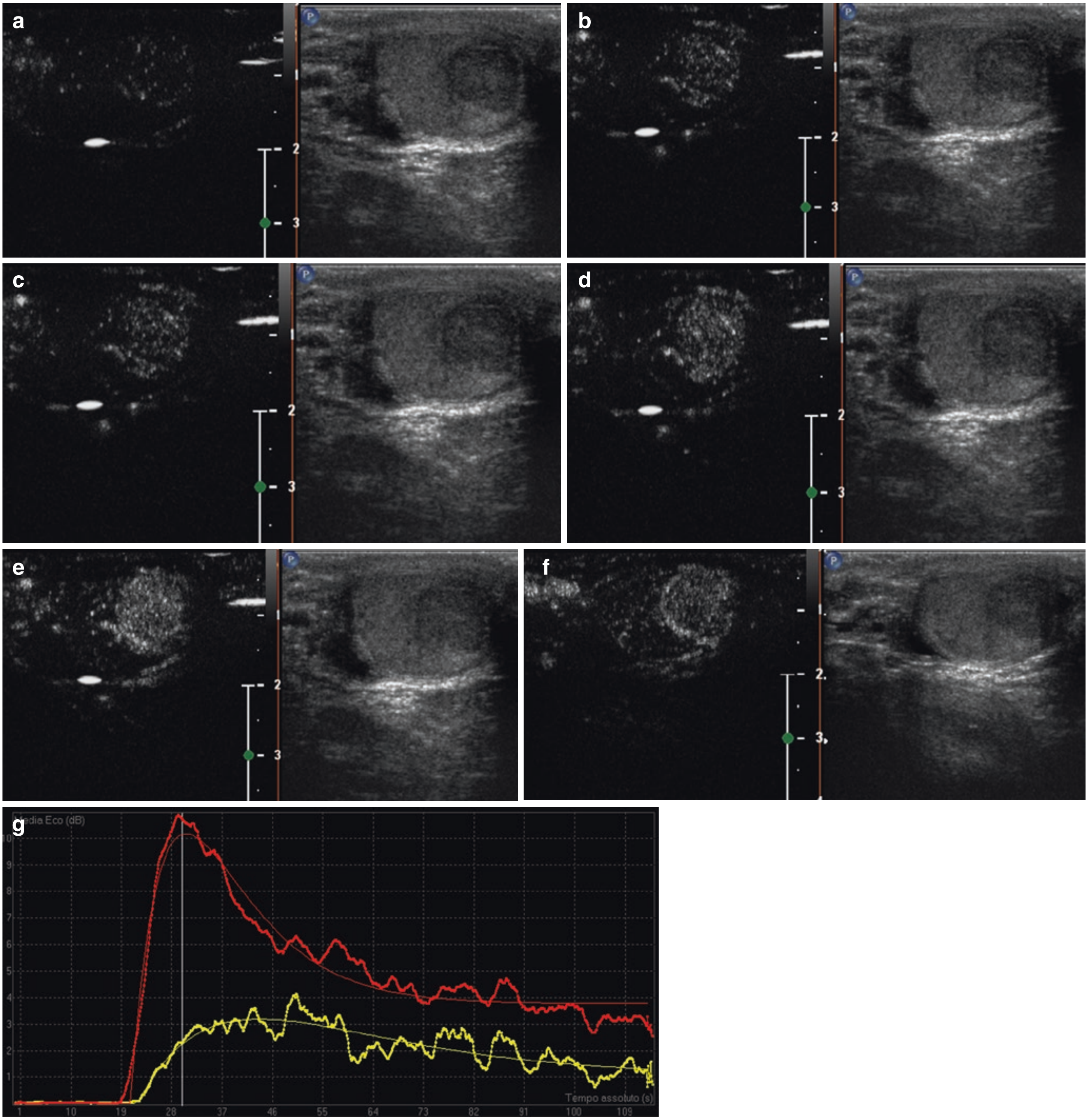


Fig. 8.12 Surgically proven Leydig cell tumour. The tumour enhances rapidly compared to the parenchyma (**a**, **b**) followed rapidly by complete fill-in (**c**–**e**) that lasts over 90 s (**f**). Contrast pulse inversion harmonic time-intensity curves of investigated lesion (*red*) and healthy parenchyma (*yellow*) show delayed time of washout (**g**)

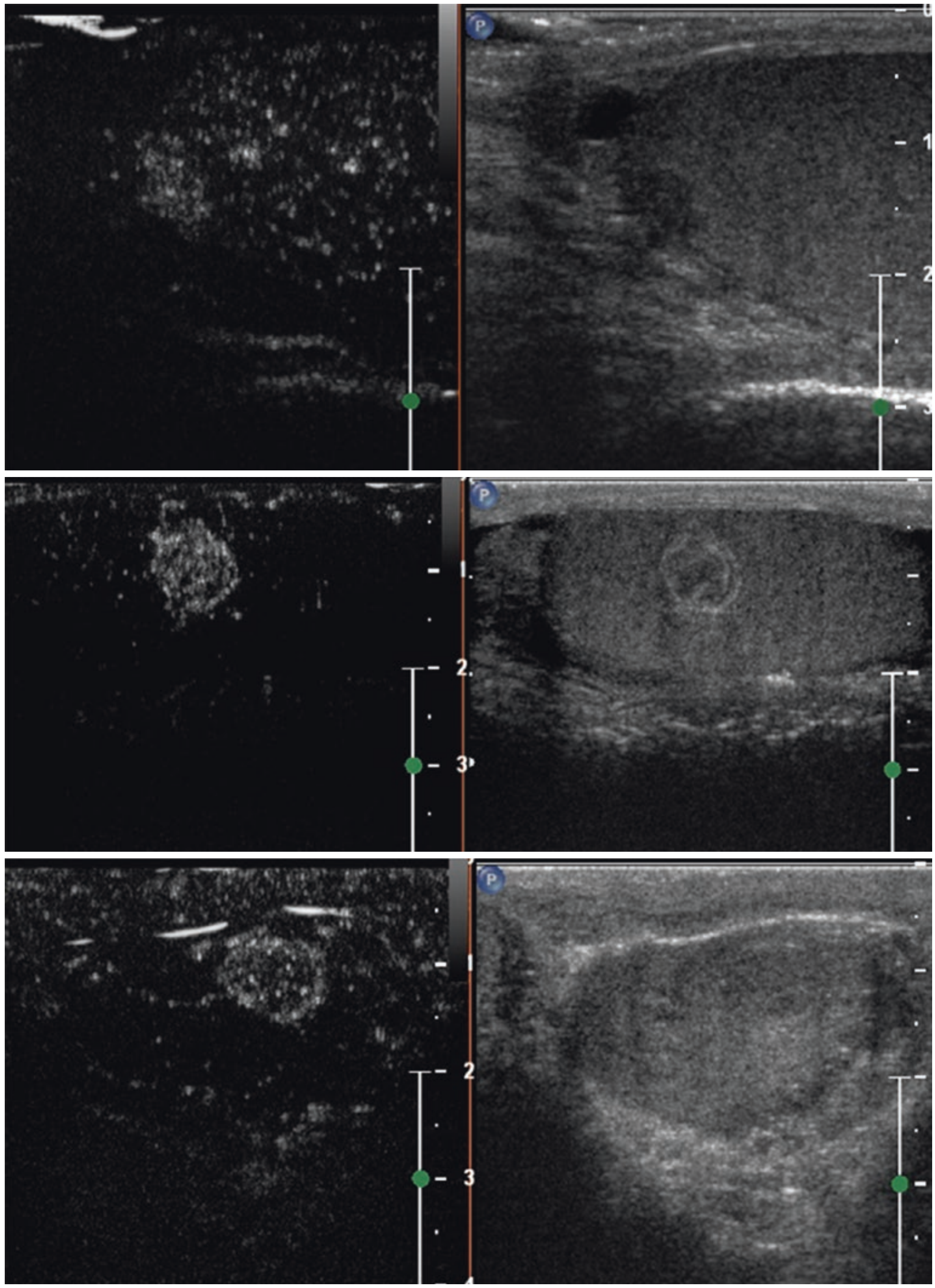


Fig. 8.13 Further cases of Leydig cell tumours at CEUS

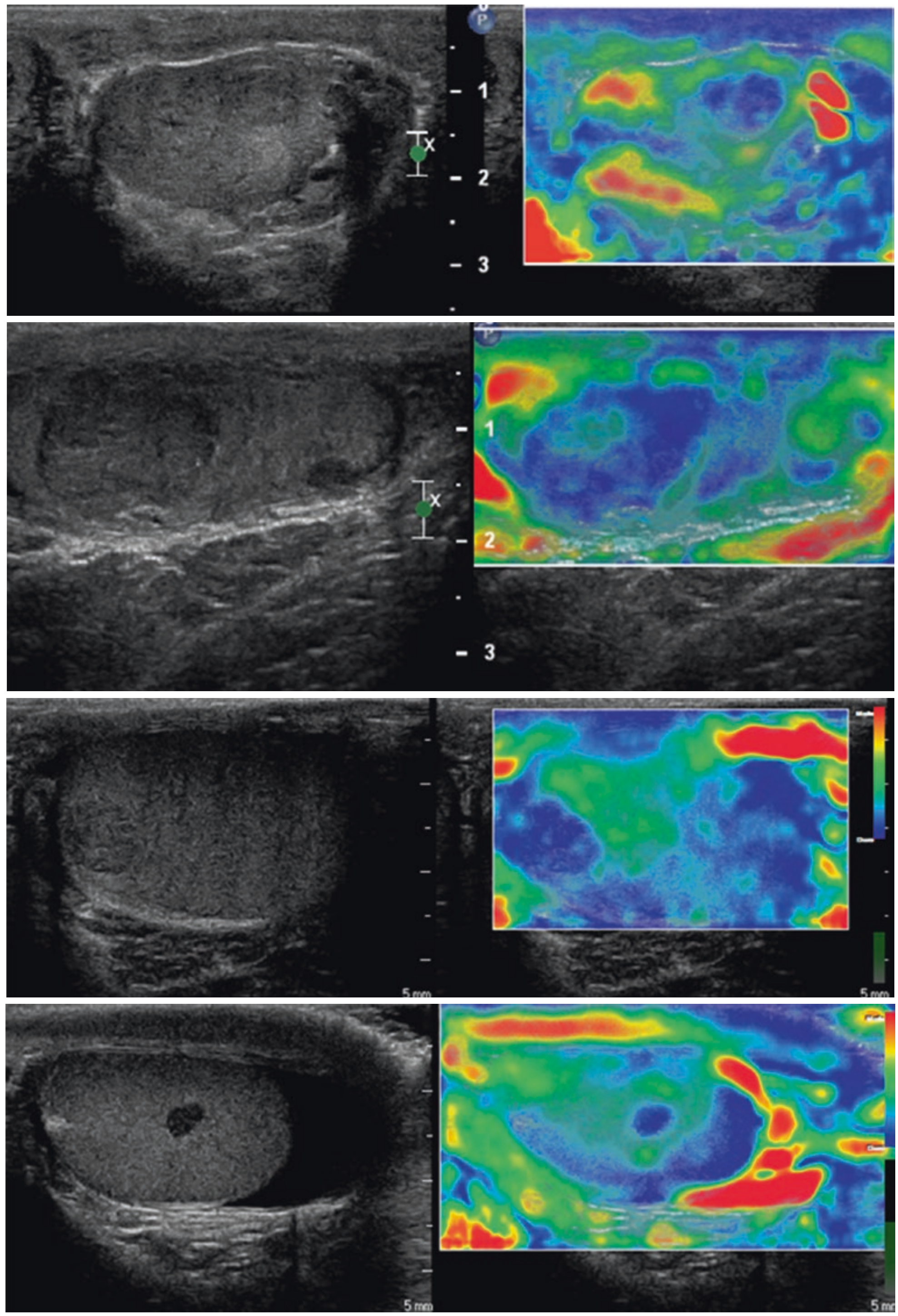


Fig. 8.14 Tissue elastography of Leydig cell tumours demonstrates a distinct 'hard' lesion (*mixed blue/green area*)

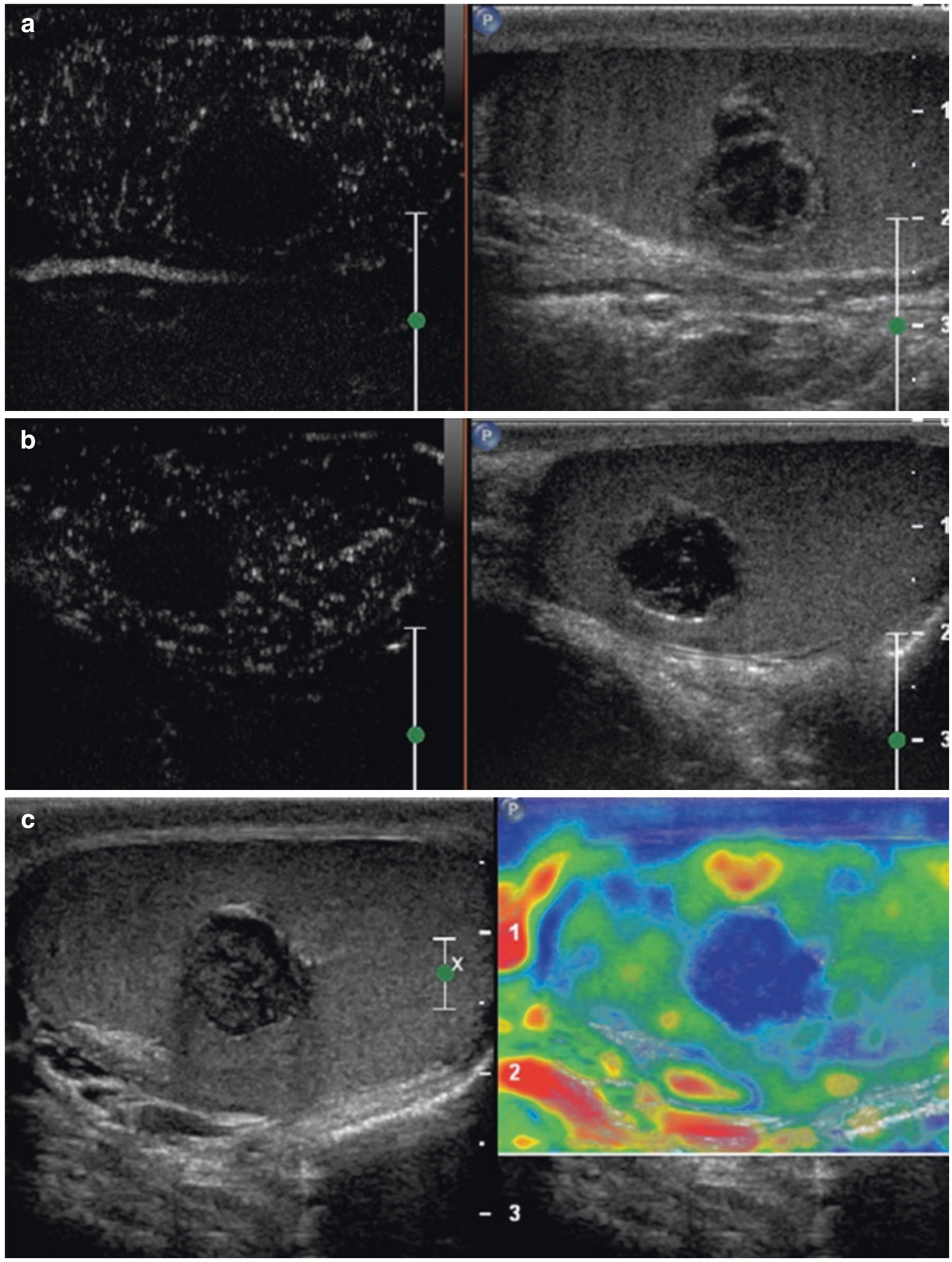


Fig. 8.15 Epidermoid cysts are completely unenhanced at CEUS, sometimes with a rim enhancement, (**a**) whereas on elastography they show hardness (**b**)

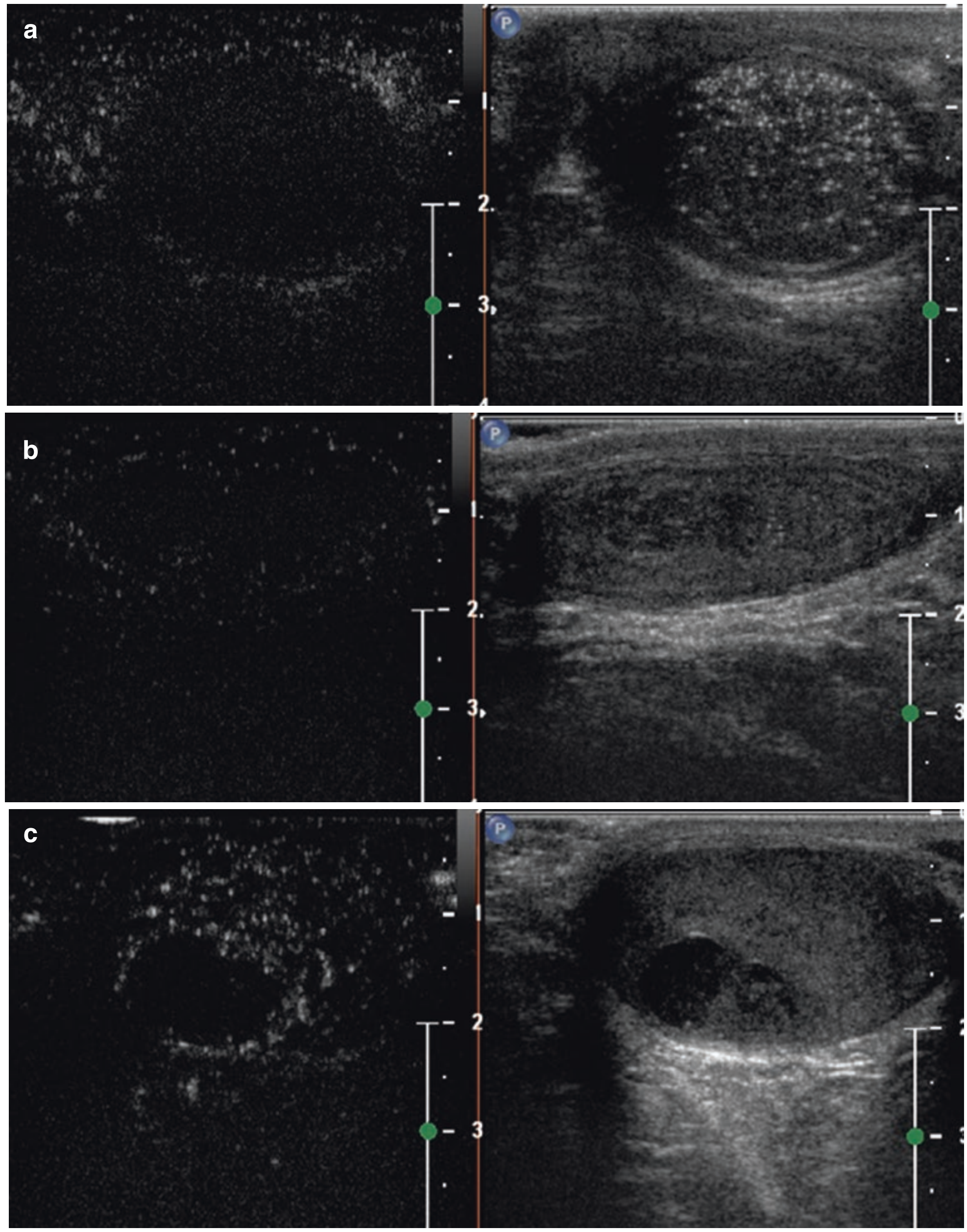


Fig. 8.16 (**a**) Testicular torsion. CEUS shows complete testicular ischemia and slightly increased peritesticular flows. (**b**) Segmental testicular infarction. CEUS demonstrates avascular infarcted area in the superior pole of the testis of a 60-year-old man. (**c**) Intratesticular abscess. CEUS confirm the avascularity of the lesion

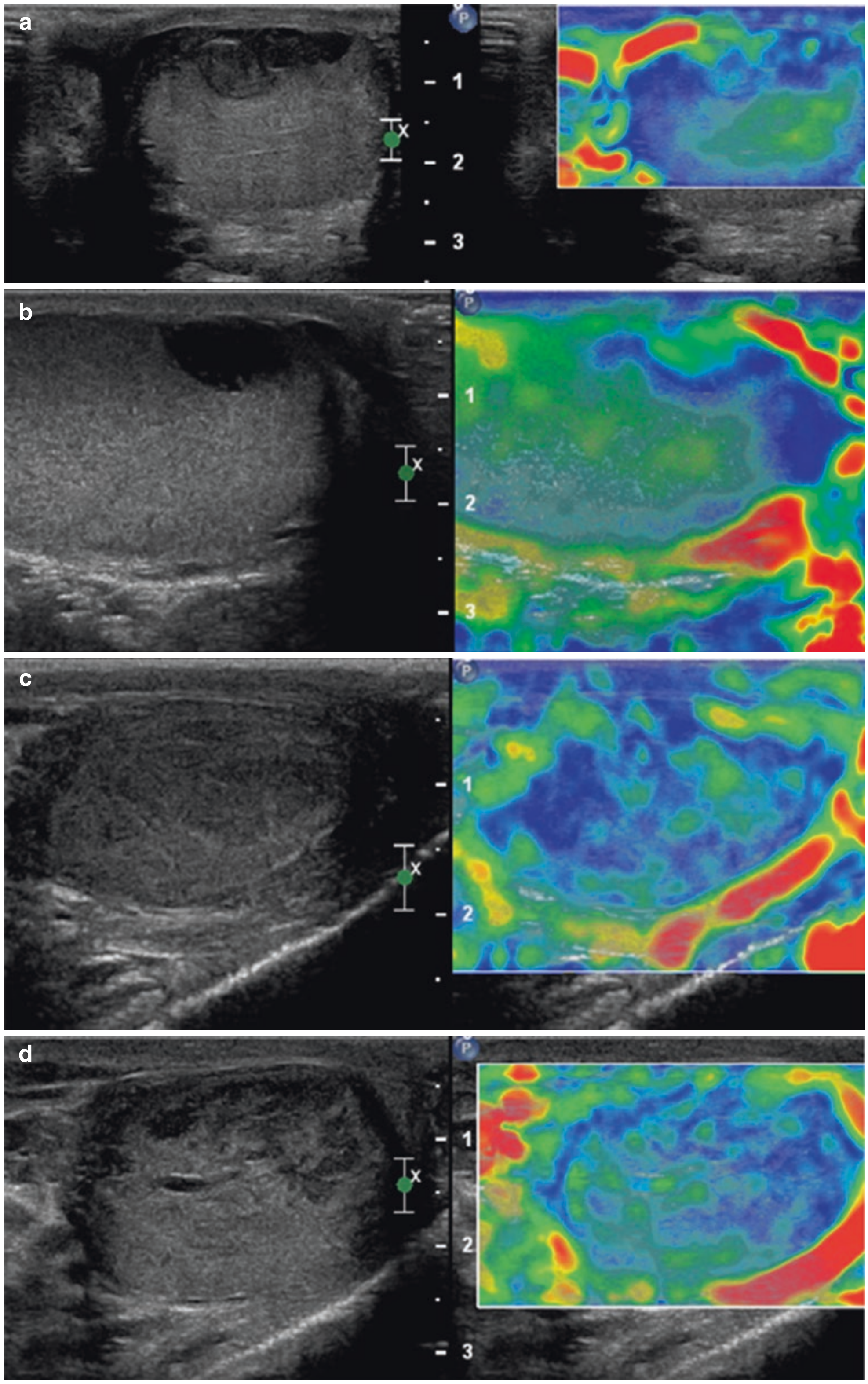


Fig. 8.17 Intratesticular haematoma shows a mixed pattern of firmness (**a**, **b**), as well as segmental ischemia (**c**, **d**)

Key Messages

- Rapid wash-in and rapid washout is a common feature of seminomas, whereas rapid wash-in and delayed washout is a distinctive feature of Leydig cell tumours.
- Sertoli cell tumours behave at CEUS as seminomas.
- The presence of necrosis or extensive calcifications, as find in microlithiasis or in burnt-out tumours, can alter microbubble diffusion inside the testis and the lesion, leading to ambiguous CEUS results.
- The intensity of contrast enhancement contributes to the differential diagnosis between neoplastic and non-neoplastic lesions, which are generally not vascularised or similarly enhanced as the surrounding parenchyma.
- The benign tumours of the testis have a qualitative hardness score that overlap with that of malignant tumours.
- Abscesses, partial infarction, Leydig cell hyperplasia, fibrosis and non-tense cysts are generally soft at elastography.

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