Abdominal Radiology
https://doi.org/10.1007/s00261-020-02529-z

SPECIAL SECTION: MALE PELVIS

Sonography of the penis/erectile dysfunction

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Abstract
Erectile dysfunction (ED) is defined as the persistent inability to achieve and/or maintain an erection for a satisfactory sexual activity. It is secondary to several organic, psychogenic, and combined causes, and represents a serious health dilemma affecting both men and their partners. The diagnostic approach to erectile dysfunction has significantly changed in the last years with the advent of phosphodiesterase-5 (PDE5) inhibitors, and with the recognition that surgical treatment of both arterial insufficiency and penile venous leak have poor long-term clinical outcomes. Although imaging modalities have diminished in importance, differentiating among causes of erectile dysfunction remains mandatory in good medical practice, and ultrasound (US) still remains the cornerstone of the diagnostic workup. US provides an objective, minimally invasive evaluation of penile hemodynamics. Moreover, it provides an excellent depiction of the penile anatomy and of its changes in pathological conditions such as in patients with Peyronie's disease, priapism, and posttraumatic erectile dysfunction.

Keywords Doppler ultrasound · Penis, US · Penis, Erectile dysfunction · Peyronie's disease · Priapism, penis, injuries

Introduction

Erectile dysfunction (ED) is the persistent inability to attain and maintain an erection sufficient for satisfactory sexual performance [1]. It can result from organic and psychogenic causes, or from a combination of them [2].

In patients with ED imaging contributes to differentiate among etiologies, a necessity that has decreased in the last years due to paradigm change in the way these patients are treated. The introduction of phosphodiesterase-5 (PDE5) inhibitors has revolutionized the therapeutics of ED and radically changed the way in which men with ED are assessed and investigated. If ED responds effectively to orally active agents, an extensive diagnostic workup including imaging is not necessary. Imaging, however, is still indicated in patients who do not respond to orally active agents [2]. Moreover, ED may be the first presenting symptom of a multiorgan vascular dysfunction requiring extensive investigation and treatment [3, 4].

According with the European Association of Urology (EAU) guidelines on Erectile Dysfunction [2], Doppler imaging is recommended in patients with primary ED, not caused by organic disease or psychogenic disorder, in young patients with a history of pelvic or perineal trauma, who could benefit from potentially curative revascularization procedures, in Peyronie's disease and congenital penile curvature which might require surgical correction, and for medico-legal reasons, such as to document end stage ED before implantation of penile prosthesis [2].

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Published online: 14 April 2020

Physiology of normal erection

Erection is a complex phenomenon which implies a delicate and co-ordinated equilibrium between the neurological, vascular and the smooth muscle compartment. It includes arterial dilation, trabecular smooth muscle relaxation and activation of the corporeal veno-occlusive mechanism [2]. During sexual stimulation, nerve impulses cause both the release of neurotransmitters from the cavernous nerves and of relaxing factors from the endothelial cells in the penis, resulting in the relaxation of smooth muscle in the arteries and arterioles supplying the erectile tissue and an increase in penile blood flow. At the same time, relaxation of the trabecular smooth muscle increases the compliance of the sinusoids, facilitating rapid filling and expansion of the sinusoidal system. Thus, the subtunical venular plexuses are compressed between the trabeculae and the tunica albuginea, resulting in occlusion of venous outflow. Nitric oxide released is probably the principal neurotransmitter mediating penile erection [5, 6]. Sexual activity triggers the bulbocavernous reflex, resulting in contraction of the bulbocavernous muscle and compression of the crural portions of the corpora cavernosa. During this phase, which is uncommonly reached in a medical setting, intracavernous pressure may exceed the systolic blood pressure, inflow and outflow of blood temporarily cease, and a rigid erection is reached. During detumescence contraction of the trabecular smooth muscle reopens the venous channels, the trapped blood is expelled, and flaccidity returns [5].

Examination technique

Ultrasound evaluation of patients with erectile dysfunction should be performed in the appropriate environment, respecting the privacy of the patient. High-frequency, broadband linear transducers must be used. The patient is examined in the supine position and the penis scanned from its ventral surface using longitudinal and transverse views. Evaluation is carried out after extracavernous injection of vasoactive drugs, except for trauma, priapism, and prosthesis evaluation, in which intracavernosal injection is contraindicated. Preliminary scanning while flaccid is optional in patients investigated for erectile dysfunction and Peyronie's disease [4, 7].

Before intracavernous injection a proper informed consent should be undertaken with the patient outlining the purpose, alternatives, risks, and benefits of the exam. The patient should be informed that a prolonged erection may be hazardous and should return to the hospital if a rigid erection lasts for more than 2 h. The syringe used for injection should have a small needle, preferably 25 gauge or less. In our clinical practice, intracavernous injection is performed in the midshaft by the sonologist (either radiologist or urologist) in sterile conditions, to prevent infection. In our experience, US guidance is not necessary.

Intracavernosal injection is preceded by anxiety and apprehension which may negatively influence the erectile response and cause false-negative results. Therefore, it is important to inform the patient of the expected duration of the examination, and to reassure him about the minimally invasive nature of the procedure, which is not painful, and with a limited number of adverse events.

Several combinations of vasoactive agents have been used for pharmacological induction of erection. Current practice is to use prostaglandin E1 (PGE1), a drug with an excellent safety profile when injected in the cavernosal bodies. The incidence of prolonged erections following pharmacological stimulation for diagnostic ultrasonography is reported as high as 11% after papaverine injection [8], but in our experience correct use of PGE1 has only minimal side effects. Our practice is to inject 10 μg PGE1, followed by a further 10 μg dose if the response is suboptimal, i.e., if the erectile response is lower than that reached at home. Some investigators routinely use 20 μg PGE1, a dosage which we prefer in patients who likely suffer from a mixed, multifactorial etiology, such as in diabetes. A lower initial dosage of 5 μg PGE1 is recommended in young patients in whom psychogenic factors likely prevail and in patients with posttraumatic erectile dysfunction from spinal cord injury.

Color Doppler interrogation and spectral analysis is performed with slow flows settings which are tuned on minimal PRF values that do not produce aliasing. Color gain is tuned just under the noise threshold of the system. Peak systolic velocity in the cavernosal arteries varies significantly according to the sampling location. In general, velocity values are highest at proximal sites, and decrease progressively at distal sites. As a consequence, standardization of the sampling location is needed to reduce variability [9, 10]. Spectral interrogation is obtained at the base of the penis with a Doppler angle $< 60°$. Steering can be used to obtain a good angular correction. The time necessary to obtain erection after pharmaco-stimulation varies greatly from one patient to another. For this reason, continuous monitoring is necessary up to 30–40 min.

Penile anatomy

The penis is made up of three corporal bodies: two corpora cavernosa and a single corpus spongiosum. Corpora cavernosa are the erectile bodies and corpus spongiosum contains the urethra. A septum divides two corpora cavernosa but

contains fenestrations that provide communications between them [7]. Blood supply to the penis is usually through the internal pudendal artery, a branch of the internal iliac artery. After giving off a branch to the perineum, the internal pudendal artery becomes the common penile artery (Fig. 1) which gives rise to the bulbar, urethral, cavernous arteries and ends by becoming the dorsal artery of the penis [11]. The bulbar artery divides into numerous branches supplying the bulb of the corpus spongiosum. Urethral artery is inconstant, its origin and size are variable. When present, it runs for a variable length in the dorsal portion of the corpus spongiosum, occasionally as far as the base of the glans [11]. In the majority of cases the middle and the distal portion of the corpus spongiosum is supplied by a periurethral arterial plexus. The cavernosal artery penetrates the corpus cavernosum, giving off a recurrent branch to the crus and running anteriorly. Along its course, the cavernous artery gives off the helicine arteries. The origin, size and morphology of the arteries to the penis is variable [11, 12]. Cavernosal arteries can be multiple, or can divide along their course in two or more branches [12]. The right and left arteries can be connected with arterial anastomoses. Many collaterals exist connecting the cavernosal arteries, the cavernosal and the dorsal arteries among them and with the periurethral arterial plexus, or with the urethral artery, when present (Fig. 2) [13].

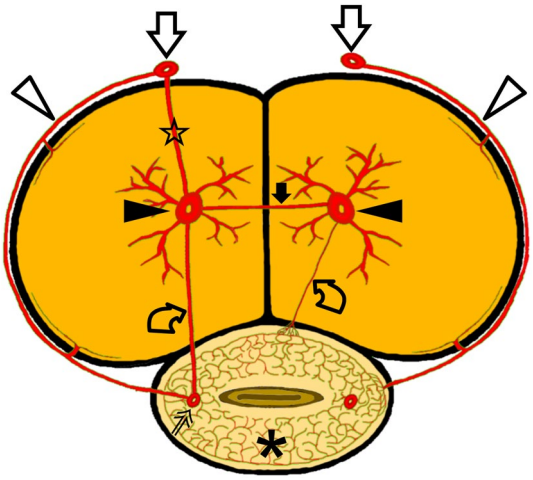

Fig. 2 Arterial vascular anatomy of the penis. Cross-sectional diagram showing the arterial vascular supply to the penis and connecting collaterals. The cavernosal and helicine arteries (arrowheads) are located slightly medially in the corpora. An arterial communication (small arrow) connects the right and left cavernosal artery. Cavernosal-spongiosal communications (curved arrows) branch from the cavernosal arteries and reach either the periurethral arterial plexus (asterisk) or the urethral artery (open-outlined arrow). Extracavernosal communications (open arrowheads) branch from the dorsal arteries (open arrows), surround the erectile bodies, supply the peripheral portion of the cavernosal bodies, merge to the periurethral arterial plexus (asterisk) or connect to the urethral artery (open-outlined arrow). An arterial communication (white star) connects the dorsal and the cavernosal artery

Normal grey-scale and Doppler features

After PGE1 injection, as sinusoidal distension progresses, the echogenicity of the corpora cavernosa decreases from the region around the cavernosal arteries outwards (Fig. 3). The diameter of the normal cavernosal arteries ranges from 0.3 to 0.5 mm in the flaccid state and increases to 0.6–1.0 mm after PGE1 injection (Fig. 4). Cavernosal artery pulsation is evident in normal subjects.

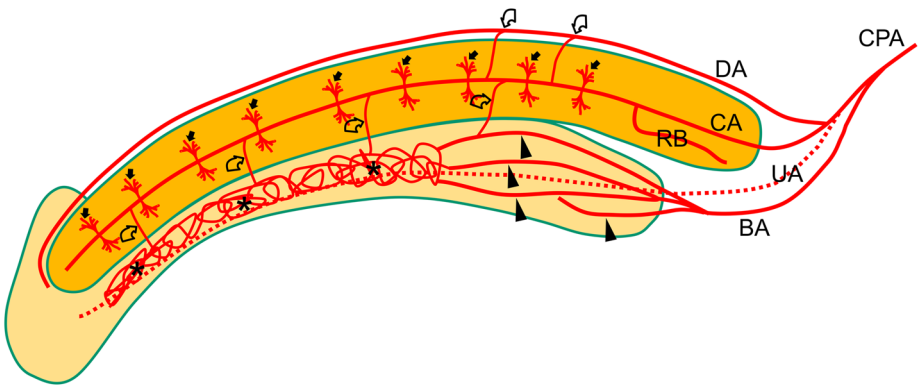

Fig. 1 Arterial vascular anatomy of the penis. The common penile artery (CPA) gives rise to the bulbar artery (BA), to the urethral artery (UA, dotted line), to the cavernosal artery (CA), and ends becoming the dorsal artery (DA) of the penis. The bulbar artery divides into numerous branches (arrowheads) supplying the bulbus of the corpus spongiosum. The urethral artery is inconstant. When present, it can rarely run till the glans, but usually ends in the proximal portion of the corpus spongiosum. The distal portion of the corpus spongiosum is supplied by a periurethral arterial plexus (asterisks). After penetrating into the corpus cavernosum, the cavernosal artery gives off a recurrent branch to the crus (RB). Along its course it gives rise to the helicine arteries (small arrows). A variety of anastomoses connect the different arteries of the penis (curved arrows)

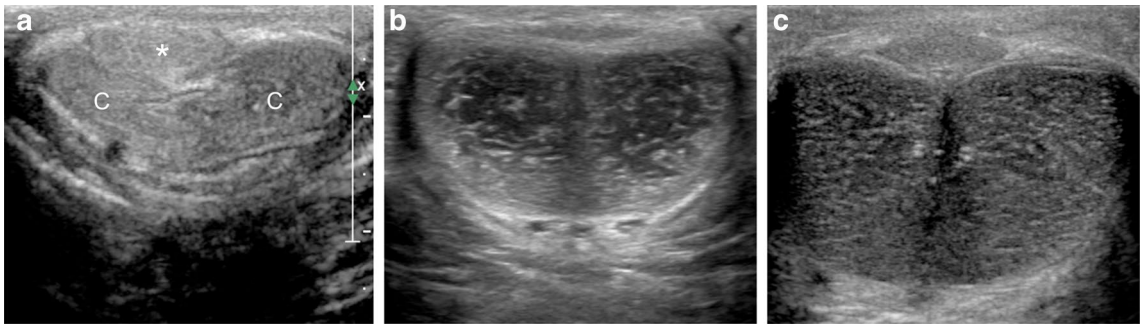

Fig. 3 Normal grey-scale US anatomy of the penis. Axial scans. **a** In the flaccid state, the corpora (C) present with intermediate echogenicity and homogeneous echotexture (asterisk, corpus spongiosum). **b** During the onset of erection distension of the sinusoids starts in the central portion of the corpora, which appears less echogenic than the outer portion. **c** In full erection the corpora are homogeneous by presence of an echogenic network of dilated sinusoids

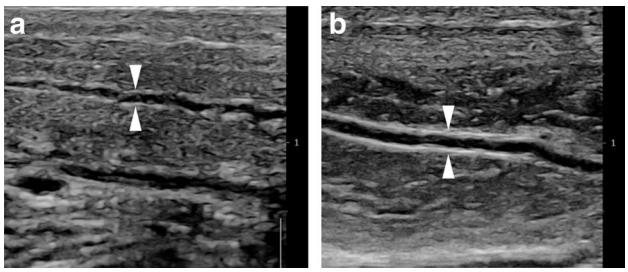

Fig. 4 Normal grey-scale US anatomy of the penis. Longitudinal scans obtained on the ventral aspect of the penis show cavernosal arteries (arrowheads) while flaccid (**a**) and during the onset of erection (**b**). The artery presents as a narrow tubular structure with echogenic wall whose diameter increases during erection from 0.8 to1.6 mm

The arteries to the penis are effectively depicted and investigated with color Doppler US.

During the onset of erection, color Doppler and spectral Doppler interrogation allow depiction of the cavernosal arteries and the helicine branches. In this phase a full evaluation of penile arteries is obtained. It is possible to study the pathway of the cavernosal, dorsal and urethral arteries, to identify presence of anatomical variations and vascular communications [7] (Fig. 5). Variations in penile arterial anatomy is common, and must not be misinterpreted as pathological. Among the most frequent anatomical variants of the cavernosal arteries are origin from the dorsal artery, bifurcation, multiple arteries, asymmetry (Fig. 6).

Doppler waveforms changes in the cavernosal arteries reflect blood pressure changes in the cavernosal bodies [14]. Six phases are described (Table 1, Fig. 7). In the flaccid state (phase 0) monophasic waveforms are recognized with low velocity, high resistance flow, typically of 15–25 cm/s. With the onset of erection (phase 1), there is an increase in systolic and diastolic flows. Peak systolic velocity > 35 cm/s, and diastolic velocity > 8 cm/s are usually recorded in normal subjects. When the blood pressure within the corpora cavernosa begins to rise a dicrotic notch appears at end systole and a progressive decrease of the diastolic flow is observed (phase 2). When the cavernosal pressure equals the diastolic pressure, diastolic flow declines to zero (phase 3). holodiastolic flow reversal (phase 4) reflects cavernosal pressure above the diastolic pressure and full erection. During rigid erection the systolic envelope is narrowed and diastolic flow disappears (phase 5) [15]. The systolic peak reduces or even disappears, reflecting cavernosal pressure approaching or exceeding blood systolic pressure. Cavernosal phase 5 requires contraction of the bulbocavernous muscles and is not commonly observed after pharmacological induced erection. During detumescence diastolic flow appears again.

Arteriogenic erectile dysfunction

ED is defined "arteriogenic" if the primary cause of inability to attain and maintain erection is diminished penile arterial flow [2]. Insufficient arterial blood supply can result from pelvic trauma or, more frequently, from a variety of conditions including atherosclerotic disease, diabetes mellitus, smoking, hyperlipidemia.

The most sensitive and specific sign of arteriogenic ED is recognition of a PSV < 25 cm/s in the cavernosal arteries, provided that a technically correct examination has been performed [16] (Fig. 8).

Valid erections may be observed in patients with extensive arteriogenic lesions and PSV below 25 cm/s, but with preserved veno-occlusive mechanism compensating for the reduced arterial inflow. Usually, these patients respond effectively to PDE5Is.

A secondary diagnostic criterion for arteriogenic ED is asymmetry of the cavernosal PSV between two sides > 10 cm/s [17].

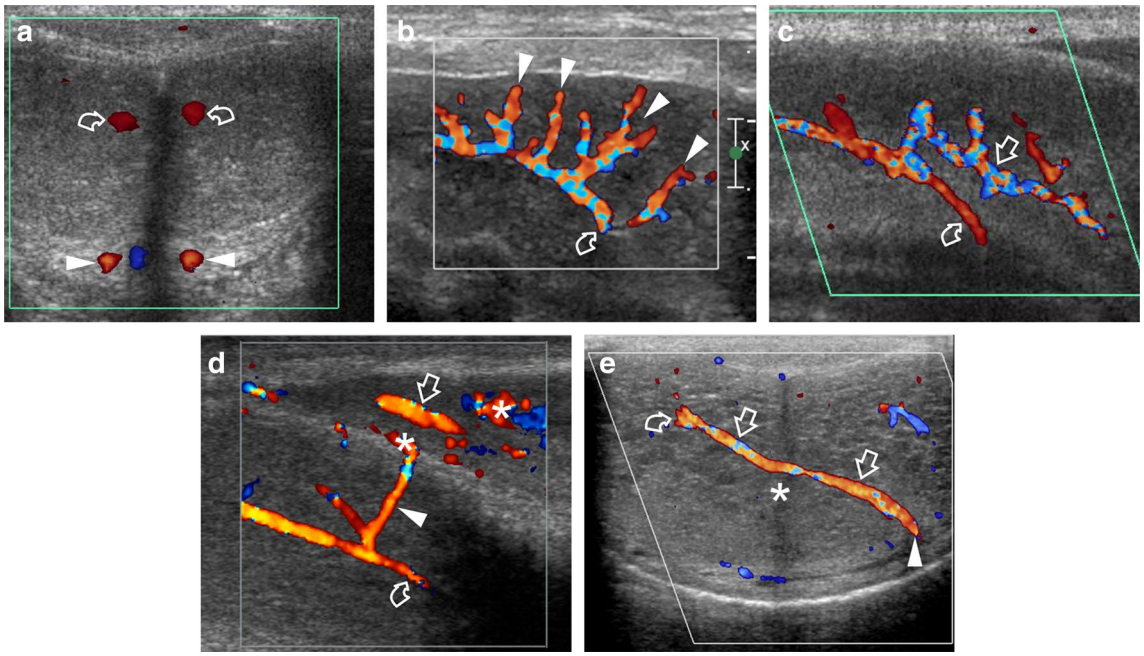

Fig. 5 Arterial vascular anatomy of the penis at color Doppler US. **a** Axial scan shows the cavernosal arteries (curved arrows) and the dorsal arteries (arrowheads) (**b**). Longitudinal scan shows the helicine arteries (arrowheads) branching from the cavernosal artery (curved arrow). **c** Longitudinal scan shows recurrent branch to the crus (open arrow) arising from the cavernosal artery (curved arrow). **d** Longitudinal scan shows cavernosal-spongiosal communication (arrowhead) branching from the cavernosal artery (curved arrow) to the periurethral plexus (asterisks). The urethral artery is also displayed (open arrow). **e** Axial scan shows arterial communication (open arrows) arising from the left dorsal artery (arrowhead), crossing the penile septum (asterisk) and reaching the right cavernosal artery (curved arrow)

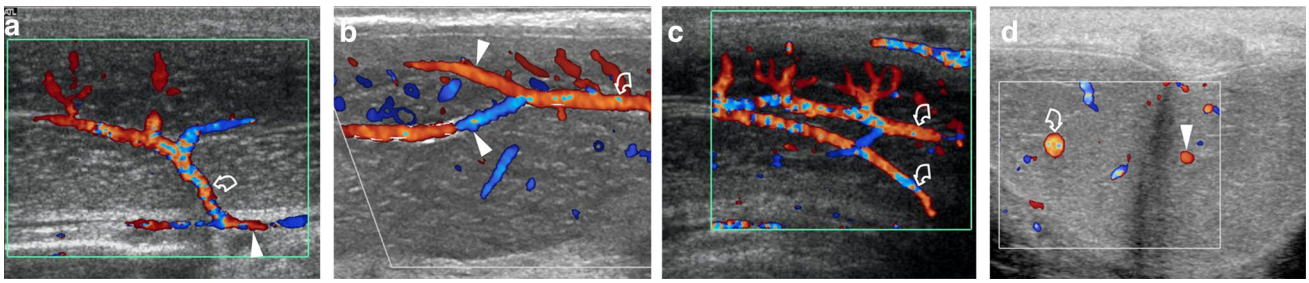

Fig. 6 Variations of the arterial vascular supply to the penis. **a** Longitudinal scan shows the cavernosal artery (curved arrow) branching from the dorsal artery (arrowhead). **b** Longitudinal scan reveals bifurcation (arrowheads) of the cavernosal artery (curved arrow). **c** Longitudinal scan shows double cavernosal artery in the same corpus (curved arrows). **d** Axial scan demonstrates asymmetry with the right cavernosal artery (curved arrow) larger than the left (arrowhead)

In older patients, and in those suffering from diabetes or chronic renal failure, calcifications in the wall of the cavernosal arteries can be identified (Fig. 9). Size change after PGE1 injection is often minimum. Vessel kinking, stenosis or obstruction can be identified. In complete cavernosal artery obstruction, color Doppler imaging can show absence of flow in the obliterated cavernosal artery, or flow inversion through collateral vessels placed distally to the occlusion site with recoding of low velocity flows at spectral analysis. Revascularization can occur through anastomoses with the contralateral patent cavernosal artery, with the dorsal arteries, or through cavernosal-spongiosal communications with reversed flow.

It has been suggested that cavernosal spectral Doppler analysis in the flaccid penis could allow assessment of arteriogenic ED noninvasively [18]. Assessment, however, is unreliable because PSV varies largely and normal thresholds have not been established. Most important, manipulation of the penis during the examination can affect Doppler changes much more significantly while it

Table 1 Normal waveform changes in the cavernosal arteries during the onset of erection

Phase	Doppler waveform	Intracavernosal pressure	Erection status
0	Low velocity, high resistance monophasic flow with minimal or no diastole	Low	Flaccid
1	Increase in systolic and diastolic flows. Low resistances	Low	Turgid
2	High velocity, progressive decrease of the diastolic flow, dicrotic notch at end systole	Progressive increase	Increasing turgidity
3	High velocity, end-diastolic flow disappearance	Equals the diastolic pressure	erection
4	High velocity, holodiastolic flow reversal	Above the diastolic pressure	Full erection
5	Narrowed systolic envelope, disappearance of diastolic flow, reduction of the systolic peak till disappearance	Approaching or exceeding systolic pressure[a]	Rigid erection

[a]This phase requires contraction of the bulbocavernous muscle

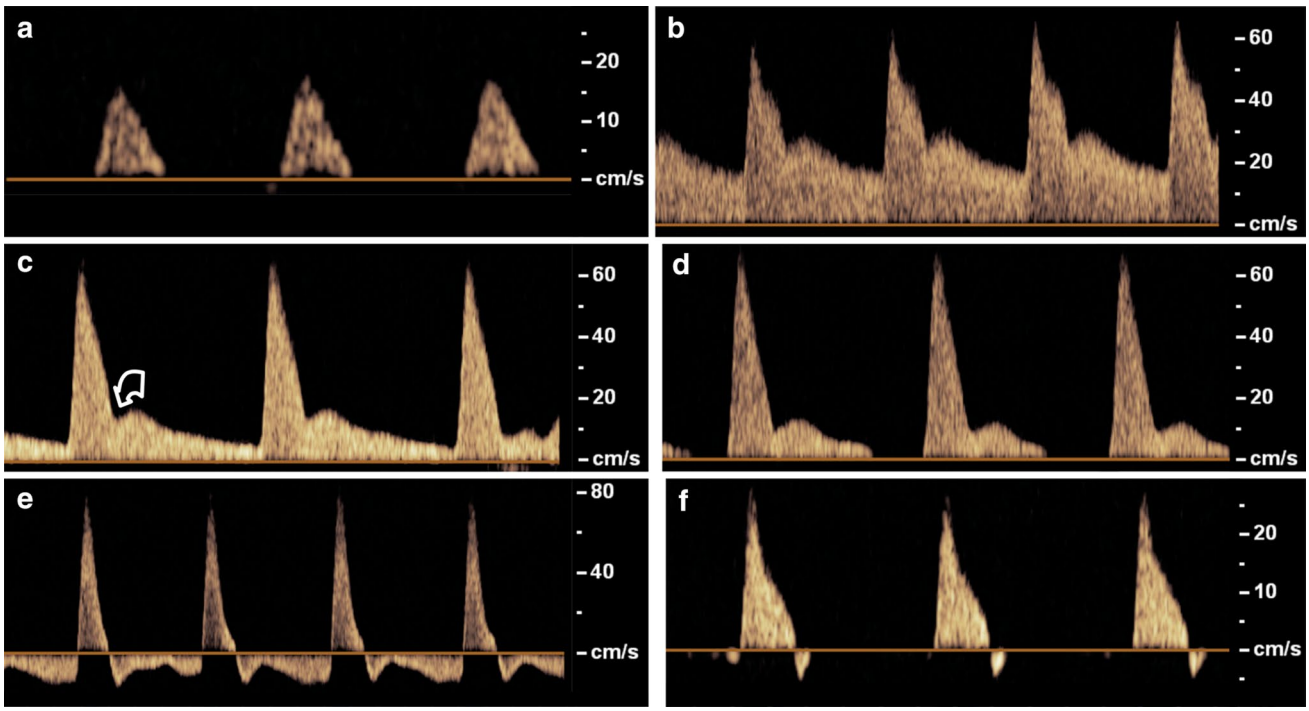

Fig. 7 Normal waveform changes in the cavernosal arteries during the onset of erection. **a** phase 0. Monophasic flow with minimal or no diastole occurring in the flaccid state. **b** Phase 1. Increased systolic and diastolic flow. **c** Phase 2. Dicrotic notch appearance at end systole (curved arrow) and progressive decrease of the diastolic flow. **d**. Phase 3. End-diastolic flow disappearance. **e**. Phase 4. Diastolic flow reversal. **f**. Phase 5. Reduction of the systolic peak during rigid erection

is flaccid than after pharmacological stimulation, when the effect of the drug is prevailing and a maximum arterial inflow has already been obtained.

After injection of an adequate dose of PGE1, several patients show PSV below the normality threshold of 35 cm/s, but above the diagnostic threshold for arteriogenic dysfunction of 25 cm/s. This range of values is commonly observed in older patients with mild erectile dysfunction. They are thought to have reduced response to PGE1 stimulation due to atherosclerotic changes. Stenosis or obstruction of the pelvic arteries should also be considered in these patients.

Veno-occlusive erectile dysfunction

The trapping of blood within the corpora cavernosa is necessary for erection to occur. Venous-occlusive dysfunction is characterized by the inability to achieve and/or maintain adequate erections despite appropriate arterial inflow. This can be caused by vascular abnormalities, such as presence of abnormally patent leakage pathways [19], or in surgical shunting procedures, but in the majority of cases it reveals a pathology of the cavernous tissue [20, 21].

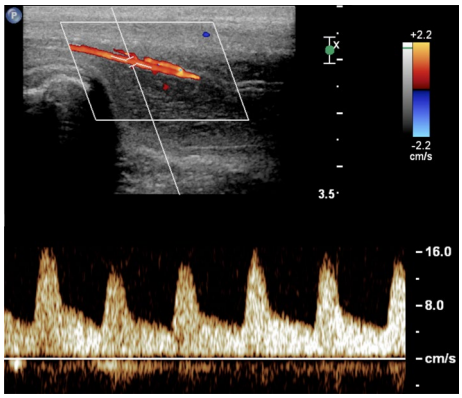

Fig. 8 Arteriogenic erectile dysfunction. Longitudinal scan showing peak systolic velocity in the cavernosal arteries of 16 cm/s

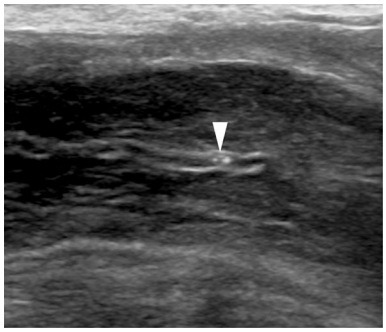

Fig. 9 Arteriogenic erectile dysfunction. Longitudinal scan shows cavernosal artery wall calcifications (arrowheads)

Veno-occlusive dysfunction is the predominant cause of impotence. It can be observed in younger patients without arterial disease, due to congenital imbalance between smooth muscle and elastic/fibrous components of the cavernous bodies [20, 22]. Also, it is observed in anxious men that the excessive adrenergic tone causes structural alterations of the cavernous smooth muscle and endothelium and insufficient trabecular smooth muscle relaxation. Moreover, aging and several risk factors are associated with reduction of the muscle component of the cavernous tissue. An association has been observed between the severity of alteration of the cavernosal smooth muscle content and the severity of veno-occlusive dysfunction [20, 23].

Independent on the underlying cause, characteristic Doppler features are recorded in patients with veno-occlusive erectile dysfunction. After PGE1 injection, Doppler findings suggestive of veno-occlusive dysfunction are EDV in the cavernosal artery > 5 cm/s in the presence of PSV > 35 cm/s, and good visibility of the helicine arterioles. The main leakage pathways from the penis, i.e., corpus spongiosum, dorsal veins, and cavernosal–spongiosal communications remain patent and well visible during the entire examination, with abnormally high flow [24] (Fig. 10).

Assessment of the arterial inflow is a key point for the diagnosis of venous-occlusive dysfunction. Patients with competent veins and normal cavernous tissue might show persistent end-diastolic cavernosal artery flows and patent leakage pathways if penile blood inflow is insufficient to achieve adequate rigidity. It is therefore difficult to distinguish with Doppler US alone between isolated arteriogenic and combined arteriogenic and venous-occlusive dysfunction.

Peyronie's disease

Peyronie's disease is an acquired fibrotic disorder of the tunica albuginea and the septum penis which affects between 3% and 8.9% of all men > 18 years causing curved, painful erections. It is characterized by growth of fibrous inelastic plaques in the tunica albuginea, either circumscribed or diffuse, causing painful erections, penile deformity and shortening. Scarring is prevalent in the dorsal aspect, but could be in any other position or diffuse. The disease is frequently combined with ED [25].

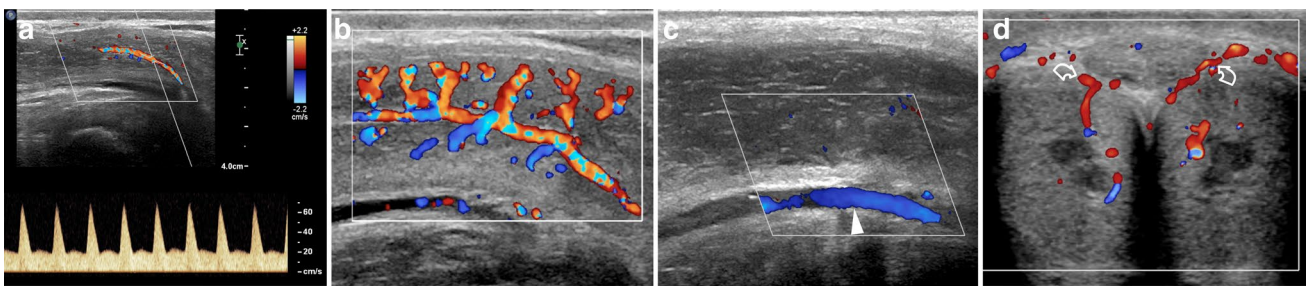

Fig. 10 Venous-occlusive erectile dysfunction. Images obtained after cavernosal injection of 20 µg of PGE1 during the maximum penile turgidity. **a** Doppler interrogation of the cavernosal arteries shows high velocity flows of 65 cm/s and persistent diastolic flows of 20 cm/s. **b** Good visualization of the helicine arterioles branching from the cavernosal arteries. **c**-**d** Evidence of blood leakage through the dorsal vein (**c**, arrowhead) and through the perispongiosal vessels (**d**, curved arrows)

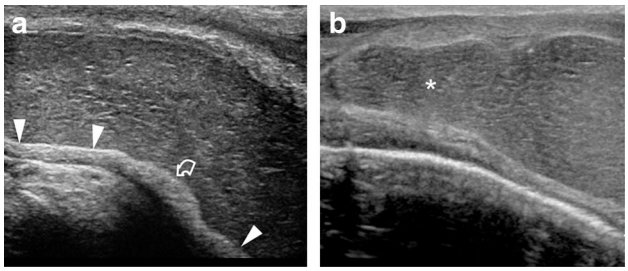

Fig. 11 Severe Peyronie's disease causing shortening, bending and deformation of the penis in two different patients. Longitudinal scans obtained after PGE1 injection. **a** Diffuse thickening of the entire tunica albuginea more prominent on the dorsal aspect (arrowheads) where a circumscribed plaque is seen (curved arrow) causing penile bending. **b** Diffuse thickening of the entire tunica albuginea causing hourglass deformity of the penis with reduction in girth of the tip (asterisk)

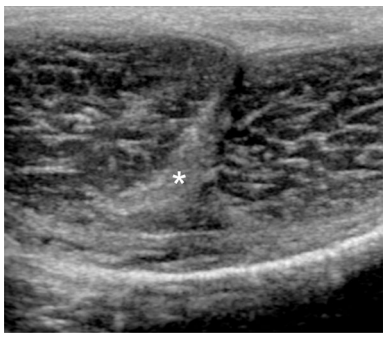

Fig. 12 Peyronie's disease with non-calcified involvement of the penile septum (asterisk)

Diagnosis is made clinically by a combination of penile pain, deformity, and/or palpable plaques. Imaging is obtained to evaluate the extent of the disease, information regarding the erectile function status and to guide treatment [26].

In patients with Peyronie's disease US is the most informative imaging technique. The study is performed after intracavernosal PGE1 injection. A comprehensive evaluation of the tunica albuginea is obtained (Fig. 11). Involvement is usually greater than it seems at palpation, although US measurement of the plaque's size is inaccurate [2]. Both calcified and soft tissue elements can be evaluated, as well as plaque involvement of the penile septum with or without calcification [27, 28] (Fig. 12). Detection of plaque calcifications allows diagnosis of disease stabilization [28] (Fig. 13).

Spatial compounding and adaptive image-processing techniques improves image quality and reduces artifacts [29].

A variety of causes may give ED in patients with Peyronie's disease, some of which are investigated with US [30, 31]. Pain or severe angulation can make coitus difficult or impossible. Penile deformity may result in performance anxiety. Cavernous fibrosis can be responsible for localized venous leakage [19]. Rarely, fibrosis may involve the cavernosal arteries and impair the arterial inflow causing arteriogenic dysfunction [32]. Moreover, many men with Peyronie's disease have coexistent arteriogenic or veno-occlusive dysfunction.

In men with Peyronie's disease, evaluation of erectile function plays a major role for therapeutic management. In patients with normal erection, plication or grafting procedures are undertaken, whereas in case of underlying erectile dysfunction reconstructive procedures associated with prosthesis implantation are considered as well.

Doppler interrogation of the cavernosal arteries allows differential diagnosis between arteriogenic and venous-occlusive dysfunction. Moreover, US and color Doppler US allow evaluating the relationship between the plaques and the penile vasculature, encasement of the neurovascular bundle and of the cavernosal arteries, which may be responsible for poor surgical outcome [28]. Anatomical variations such as arterial vascular communications, and

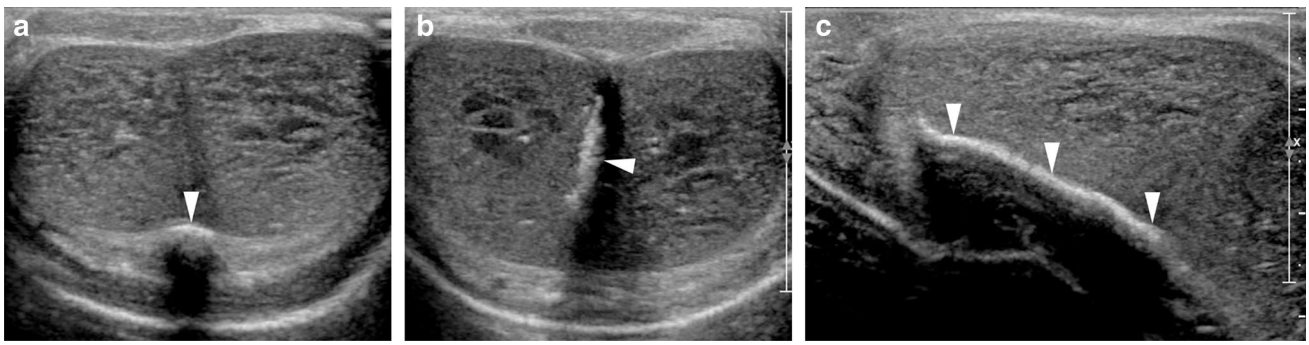

Fig. 13 Severe Peyronie's disease with diffuse thickening of the entire tunica albuginea displaying multiple calcified areas (arrowheads)involving the dorsal portion (**a**), the septum (**b**) and the penile tip (**c**)

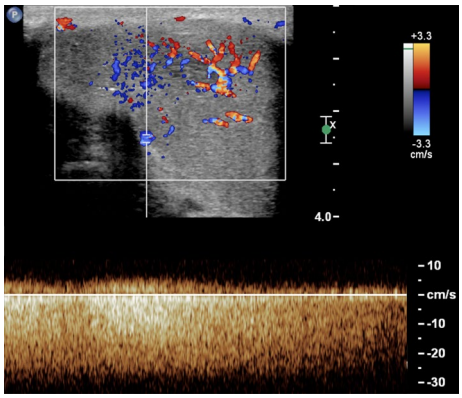

Fig. 14 Severe Peyronie's disease with plaque-related venous leakage. Hourglass deformity of the penis with reduction in girth of the tip and reduced turgidity in the distal portion of the corpora. Doppler interrogation shows venous flows in the site of penile deformation due to incomplete distension of the tunica albuginea

leakage pathways along the penile shaft should be identified, with specific notice to vessels adjacent to the plaques [19] (Fig. 14).

High-flow priapism

Non-ischaemic priapism, also called arterial or high-flow priapism, is a persistent erection caused by unregulated cavernous arterial inflow due, in most of cases, to a blunt perineal trauma causing cavernosal artery laceration [2]. Rarely, high-flow priapism results from cavernosal artery injury following needle insertion or shunting procedures [33–35].

As a consequence of the trauma, a fistula is created between the artery and the sinusoidal spaces with endothelium shear stress and high oxygen tension that stimulate the synthesis and release of nitric oxide. This eventually results in arterial and trabecular dilatation, excess arterial cavernosal inflow, and penile engorgement.

Typically, the patient presents with history of a blunt perineal trauma. Erection is neither fully rigid, nor associated with pain. Full erection can develop after sexual stimulation. Aspiration shows bright red arterial, well oxygenated blood.

A comprehensive history is mandatory for the diagnosis, followed by blood aspiration from the corpora cavernosa.

The management of non-ischaemic priapism is not an emergency, but in the rare cases in which the penis is nearly fully rigid and painful. This is the only situation which requires a rapid workup to prevent cavernosal tissue fibrosis. In most of cases, conservative management with ice apposition and site-specific perineal compression is an option, particularly in children. Androgen deprivation therapy has been reported. Selective arterial embolisation has a high success rate of up to 89% and can be used, especially when conservative management is unsuccessful [36]. Cavernosal artery closure can be temporary, using sealing substances such as autologous clot or embolization microspheres, or permanent, using acrylic glue or coils. No definitive statement can be made on the best substance for embolisation in terms of sexual function preservation [2]. Temporary substances are preferred in our institution.

Color Doppler US is part of the workup of patients with high-flow priapism in many institutions. The study must be performed without intracavernosal drug injection.

Cavernosal tissue laceration and distension of the surrounding lacunar spaces is already seen at grey-scale US as an irregular hypoechoic region within the echogenic cavernous tissue. Color Doppler evaluation allows identification of blood extravasation, of the cavernosal artery injury, and of vessels feeding the fistula in virtually all patients [37] (Fig. 15).

Following cavernosal artery embolisation, priapism resolution can require hours or days. We repeat color Doppler US within few days, to determine if the treatment was successful

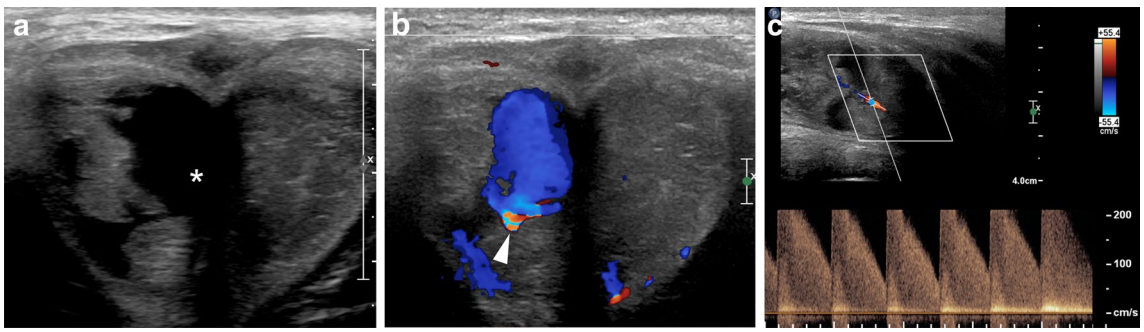

Fig. 15 Postraumatic high-flow priapism. **a**, **b** Axial scans of the penis. **a** Grey-scale US shows a hypoechoic region within the right corpus cavernosum (asterisk). **b** Color Doppler interrogation shows characteristic color blush that is consistent with extravasation of blood from the lacerated cavernosal artery. The cavernosal artery tear is identified as a small color spot displaying aliasing (arrowhead) **c** Doppler interrogation of the cavernosal artery tear shows high velocity, turbulent flows

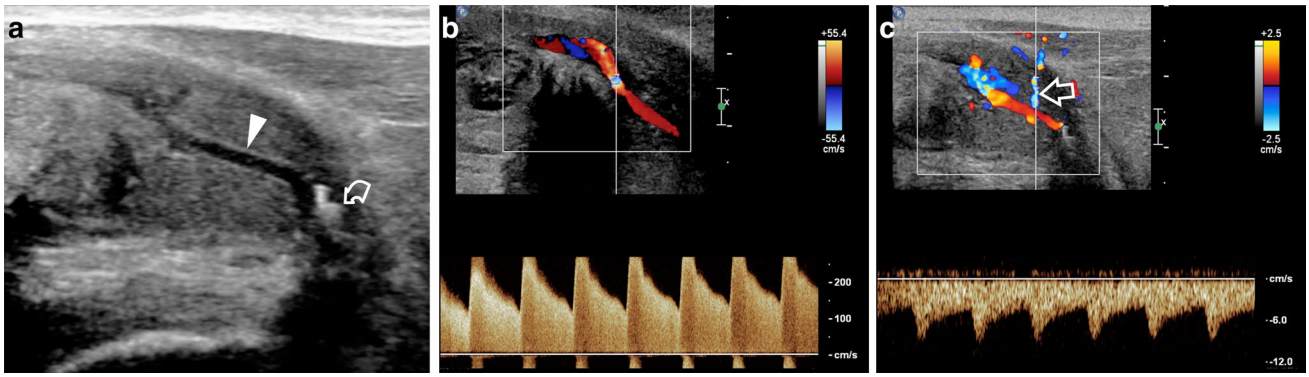

Fig. 16 Postraumatic high-flow priapism. Incomplete closure of the fistula after endovascular coiling of the torn cavernosal artery. **a** Sagittal scan shows the endovascular coil (curved arrow) within the cavernosa artery (arrowhead). **b**, **c** Doppler interrogation shows that the fistula is still patent (**b**) fed by collaterals (open arrow, **c**) distal to the coil

(Fig. 16). The fistula can close, or remain patent despite arteriographic evidence of occlusion, fed by collateral vessels not excluded. Among them, accessory pudendal arteries, recurrent cavernosal branches, and communications with the contralateral cavernosal artery or with the dorsal artery. Often these vessels are not evident before the embolization because of insignificant flow, but become haemodynamically significant after the closure of the principal feeding routes [38]. In case of persistent fistula with reduced flow, we repeat color Doppler US within 2- to 4 weeks to confirm progressive closure of the fistula [37].

Selective cavernosal artery embolization results in resolution of priapism in the majority of patients with preservation of the erectile function [39–41]. Decreased potency can be observed in a minority of patients presenting with nearly complete erection and in those with longstanding priapism [37, 39, 42]. This situation can result in corporeal fibrosis and veno-occlusive dysfunction, a situation that can be observed also in patients with extensive scarring replacing the cavernosal tissue in the site of the injury.

In patients who develop erectile dysfunction after treatment of high-flow priapism color Doppler US should be performed after intracavernosal PGE1 injection to investigate the underlying cause [37].

Low-flow priapism

Ischemic priapism, also called veno-occlusive or low-flow priapism, is a persistent, painful erection marked by little or nocavernous arterial inflow. The corpora are fully rigid and tender, but the glans penis is soft. Collecting a comprehensive history is critical. History must include details relating to presence of previous priapism episodes, erectile function prior to the current priapism episode, onset of full erection and pain, degree of pain, medical or recreational drug use, and associated disease, such as sickle cell anemia or other haemoglobinopathies. Aspiration of blood from the corpora cavernosa shows dark ischemic blood. There is no history of trauma.

Diagnosis is made based on clinic, history, and blood aspiration. In the majority of cases, imaging does not add clinically useful information, but may be performed as an alternative or adjunct to blood gas analysis when the diagnosis is not clear by history and aspiration, and when practitioners inexperienced in diagnosing/treating low-flow priapism order imaging studies.

If possible, US scanning of the penis should be performed before corporal blood aspiration to prevent aberrant blood flow which can mimic a non-ischemic picture.

Ischemic priapism is a urological emergency: immediate intervention is required to minimize irreversible consequences, such as cavernosal tissue necrosis, corporal fibrosis, and irreversible erectile dysfunction [2].

In ischemic priapism smooth muscle biopsies show interstitial edema at twelve hours progressing to destruction of the sinusoidal endothelium at 48 h, and smooth muscle necrosis with fibroblast-like cell transformation. Thrombi can be found in the sinusoidal spaces [43].

At color Doppler US little or no arterial inflow is seen. No flows are recorded in the cavernosal arteries, or low velocity, high resistance flows (Fig. 17). The corpora cavernosa initially present with the same echogenicity and echotexture as in patients with normal erection. When the patient is left supine for few minutes without manipulating the penis, the corpuscolate component of the blood into the corpora cavernosa tends to sediment downwards forming a fluid–fluid level. Later on, the echogenicity of the corpora cavernosa increases, due to edema. Occasionally, echogenic material obliterates the cavernosal arteries [32, 33]. In longstanding ischemic priapism fibrotic replacement of the cavernosal tissue occurs, with irreversible erectile dysfunction. At US,

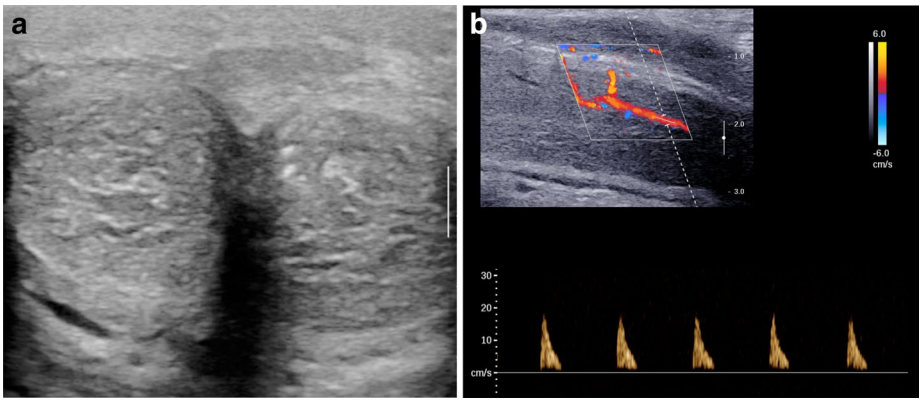

Fig. 17 Low-flow priapism lasting for 11 h. **a** Axial image reveals increased echogenicity of the cavernosal bodies. **b** Color Doppler interrogation shows that the cavernosal arteries are patent with low velocity, high resistance flows. After corporal blood aspiration and intracavernosal α-adrenergic agonist injection detumescence was obtained. The patient regained satisfactory erection

diffuse fibrosis is recognized as ill-defined hyperechogenic tissue replacing the sinusoids, often prevailing around the cavernosal arteries. Little changes occur at Doppler interrogation after PGE1 injection [33]. When impairment of the cavernosal artery inflow is present, small peripheral vessels can be demonstrated feeding the outer portions of the corpora cavernosa through collaterals from extraalbugineal vessels. Use of Contrast-enhanced US (CEUS) is experimental. Enhancement is present in the outer portion of the corpora only, while the tissue around the cavernosal arteries is not vascularized [44].

Stuttering priapism

Intermittent or recurrent priapism, also called stuttering priapism, is characterized by repetitive and painful episodes of prolonged erections, usually transient and self-limiting [2]. This is a hemodynamically distinct situation from ischemic priapism. Cavernosal artery flow is in general preserved, and treatment with blood aspiration/irrigation and intracavernous injections of α-adrenergic agonists is usually successful, with a good erectile function restoration. If left untreated, however, episodes can result in fibrotic cavernosal tissue damage [45].

Imaging is not requested in the standard clinical practice, but may be performed when the diagnosis is not clear. At color Doppler US stuttering priapism resembles a normal erection. High velocity flows of 30–40 cm/s or more are generally recorded in the cavernosal arteries, with diastolic flow disappearance or reversal, depending on the degree of erection reached. Peak systolic velocities reduce in rigid erection. In episodes lasting > 10–12 h a reversible increase of the cavernosal tissue echogenicity can be observed. This is likely due to tissue edema, a reversible tissue alteration [45]. A reactive hyperafflux may develop, presenting at color Doppler US with high velocity, low resistance cavernosal artery flows. This appearance must not be misinterpreted as shifting from stuttering to high-flow priapism. Cavernosal tissue echogenicity is restored within few days [45].

Postraumatic erectile dysfunction

Patients with severe pelvic traumas and patients who had undergone major pelvic surgery, such as radical prostatectomy, are at risk of developing erectile dysfunction due to combined injury to the vessels and nerves [46–48]. The vascular supply to the penis can be assessed by Doppler interrogation of the cavernosal arteries [49]. Young patients with posttraumatic arteriogenic dysfunction can benefit from surgical penile revascularization, in which the penile blood supply is restored connecting the inferior epigastric artery to the dorsal penile artery [50].

Before the operation, color Doppler US is recommended to check for presence of well-developed dorsal arteries, communications between the dorsal and the cavernosal arteries, and communications between the cavernosal arteries of both sides. Size and patency of the epigastric arteries is also evaluated [33, 51].

After the operation, color Doppler US allows evaluation of the vascular anastomosis and of postoperative complications, such as anastomotic dehiscence, thrombosis, stenosis, or pseudoaneurysms [33] (Fig. 18).

Isolated spinal cord injury and isolated damage to the penile nerves can result in neurogenic erectile dysfunction. Early after the trauma, the structure of the cavernosal bodies is normal. A good-to-rigid erection is obtained after intracavernosal injection of very low PGE1 doses of 2.5–5 µg. Longstanding denervation results in cavernosal tissue fibrosis and severe erectile dysfunction, not responsive to high PGE1 doses (≥ 20 µg) (Fig. 19). Penile traumas with injury to the cavernosal tissue can develop focal fibrotic changes within the corpora cavernosa and a variable degree of erectile dysfunction. Among them: intracavernosal haematomas associated or not with cavernosal artery tear and high-flow priapism, isolated

septal haematomas, penile fracture with extensive cavernosal tissue laceration. Fibrotic changes present at US as hyperechogenic areas or nodules with variable attenuation [51]. After intracavernosal PGE1 injection Doppler interrogation is useful to investigate the penile haemodynamics (Fig. 20).

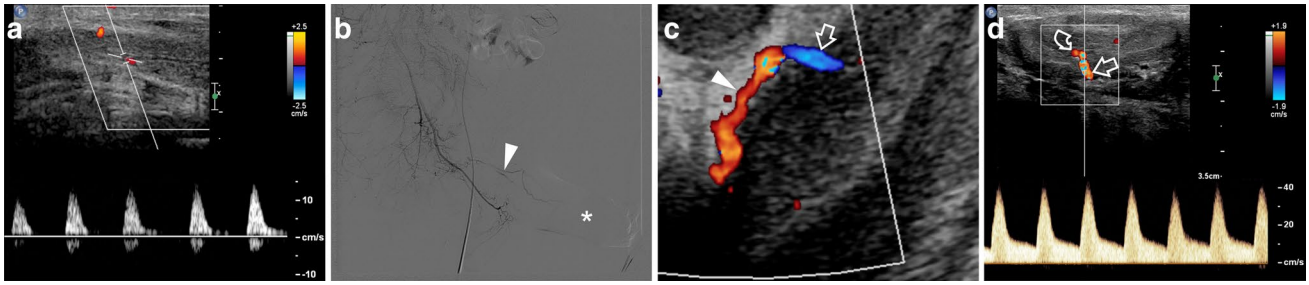

Fig. 18 Severe postraumatic erectile dysfunction in a 32-year-old forester who had multiple pelvic fractures following a crushing accident produced by the fall of a tree. **a** Color Doppler interrogation of the cavernosal arteries obtained after cavernosal injection of 20 μg of prostaglandin E1 shows low velocity flows of 15 cm/s. **b** Angiography shows interruption of the vascular supply to the penis (asterisk) with no opacification of the cavernosal arteries. The dorsal artery (arrowhead) is patent. **c**, **d** After penile arterial revascularization the dorsal artery (arrowhead in **c**) supplies the cavernosal artery (curved arrow in **d**) through a dorsal-cavernosal communication (open arrow in **c**, **d**). High velocity flows of 40 cm/s are recorded in the dorsal-cavernosal communication (**d**). The patient regained satisfactory erection

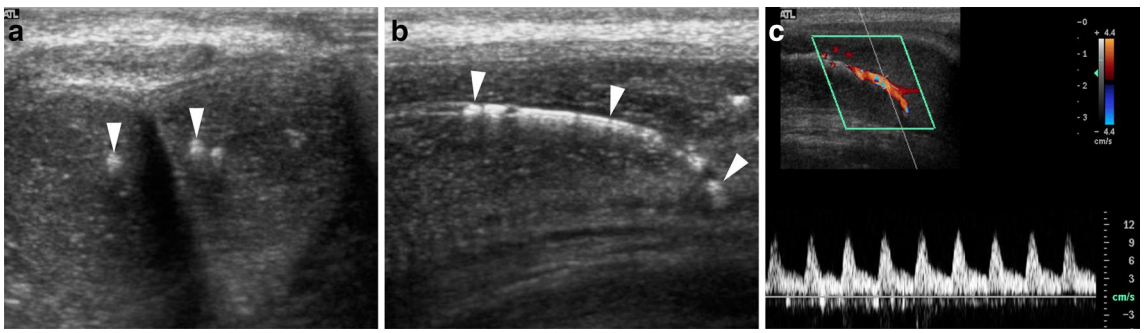

Fig. 19 Severe postraumatic erectile dysfunction in a 33-year-old paraplegic patient who had a spinal cord injury 12 years before after a motorbike accident. Images obtained after cavernosal injection of 20 μg of prostaglandin E1. **a**, **b** Axial (**a**) and sagittal (**b**) grey-scale images show cavernosal arteries with extensive wall calcification (arrowheads). **c** Low velocity flows are recorded at color Doppler interrogation

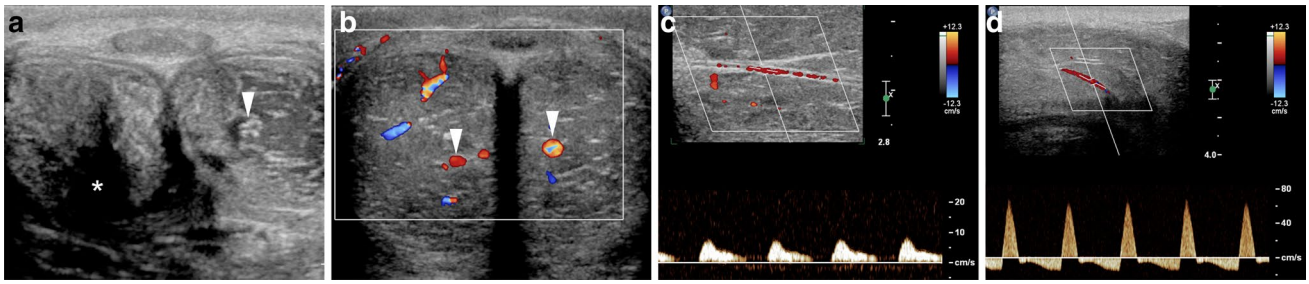

Fig. 20 Patient with direct trauma to the penis resulting in scarring in the right cavernosal crus and tight stenosis of the right cavernosal artery investigated after cavernosal injection of 20 μg of prostaglandin E1. **a** Axial scan showing scarring tissue (asterisk) in the right corpus. The right cavernosal artery is not appreciable, embedded within the scar tissue. The left cavernosal artery (arrowhead) is normal. **b** Axial scan obtained distal to the site of the cavernosal injury. Color Doppler interrogation shows asymmetric distension of the cavernosal arteries (arrowheads). **c**-**d** Doppler interrogation of the cavernosal arteries shows low systolic flows (10 cm/s) in the right, stenotic artery (**c**) and normal flows in the left, non-stenotic artery (**d**)

Prosthesis implantation

MRI is the imaging modality of choice for evaluation of prosthesis dysfunction, while US does not allow a panoramic assessment of all components of the device [52, 53]. US, however, depicts effectively the scrotal pump, corporeal cylinders, and abdominal reservoir [33]. Tubing is not entirely visualized (Fig. 21). US is able to assess prosthesis dysfunction due to oversized or undersized cylinders, displacement and rupture of the cylinders and or the reservoir, presence of fluid collections and of other inflammatory complications around the scrotal pump, cylinders and reservoir. Prosthesis infection requires aggressive therapy with antibiotics and, in case of failure, prosthesis removal. In these latter patients US can be useful in identifying the extent of fibrotic changes before reimplantation [33].

US elastography and CEUS

Ultrasound (US) elastography observes the internal deformations that occur in tissue in response to an applied force, and convert the resulting information to a suitable form for display. Several approaches have evolved regarding both the application of force and the measurement and visualization of tissue response, and these can be grouped in two main categories: strain elastography (SE) and shear wave elastography (SWE) [54].

SE applies a compressive force to tissue and evaluates lesion stiffness, expressed on a qualitative color scale. Depending on the imaging system used, a color closer to either the blue or red end of the spectrum indicates increased stiffness. SWE exploits the fact that shear waves propagate faster in hard than in soft tissue; this can be qualitatively and/or quantitatively assessed with a color-scaled image and measuring the elasticity values in kPa. A color closer to the red end of the spectrum represents a higher kPa value.

It has been suggested that US elastography could potentially improve the evaluation of patients with erectile dysfunction. Experimental studies in animals support use of SWE for evaluation of cavernosal tissue stiffness. Zhang et al. demonstrated that SWE could differentiate between the cavernosal tissue stiffness of aged and of sexually immature rats [55–57]. Hu et al. found higher cavernosal tissue stiffness at SWE in rabbits with hyperlipidemia-induced erectile dysfunction [58].

Recent investigations claim use of US elastography to evaluate changes in stiffness of the cavernosal tissue in patients with erectile dysfunction, as an index of change of the structure and composition of the tissue. Zhang et al. evaluated at SWE cavernosal tissue stiffness of the flaccid penis in healthy volunteers, and concluded that SWE could be a valuable non-invasive method for this purpose [59]. Inci et al. found that SWE is able to assess noninvasively changes with age of cavernosal tissue stiffness during the flaccid state [60]. Higher cavernosal tissue stiffness is found in elderly (Fig. 22).

Altimbas et al. examined 88 patients with erectile dysfunction after intracavernous PGE1 injection and found statistically different scores at strain elastography in the patients with venous-occlusive dysfunction [61]. Hamidi et al. evaluated in the flaccid state with SE the cavernous body fibrosis due to neurovascular bundle damage following radical prostatectomy. Very strong negative correlation was detected between International Index of Erectile Function (IIEF-5) and elasticity scores ($p = 0.0001$) [62]. Similarly, fibrotic changes resulting from ischemic priapism are depicted both with SWE (Fig. 23) and with strain elastography (Fig. 24).

Turkay et al. investigated during the flaccid state with SWE 35 patients with erectile dysfunction and 35 healthy volunteers with similar age, and found different elasticity values between the two groups [63].

Pilot investigations suggest that US elastography can be useful in patients with Peyronie'sdisease to detect and to

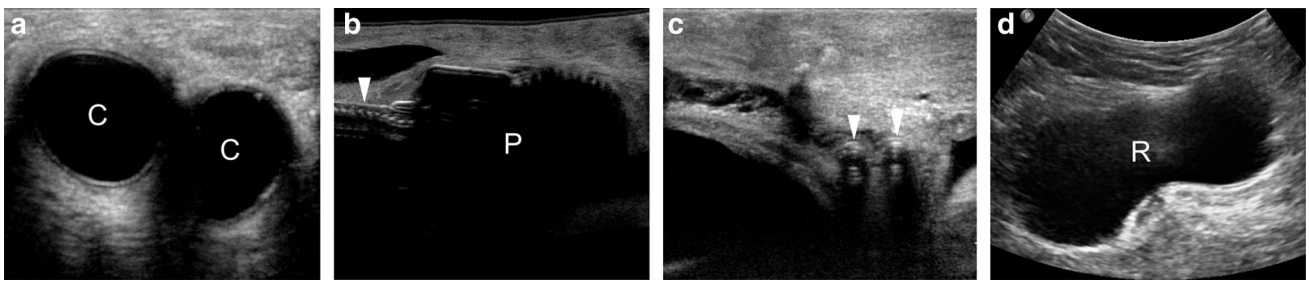

Fig. 21 Inflatable penile prosthesis. US appearance during erection. **a** Axial image of the penile shaft shows the inflated corporeal cylinders (C) as anechoic structures replacing the central portion of the cavernosal bodies. **b** scrotal pump (P) connected with tubing (arrowhead). **c** Tubing in the subcutaneous tissue (arrowheads). **d** Partially deflated abdominal reservoir (R)

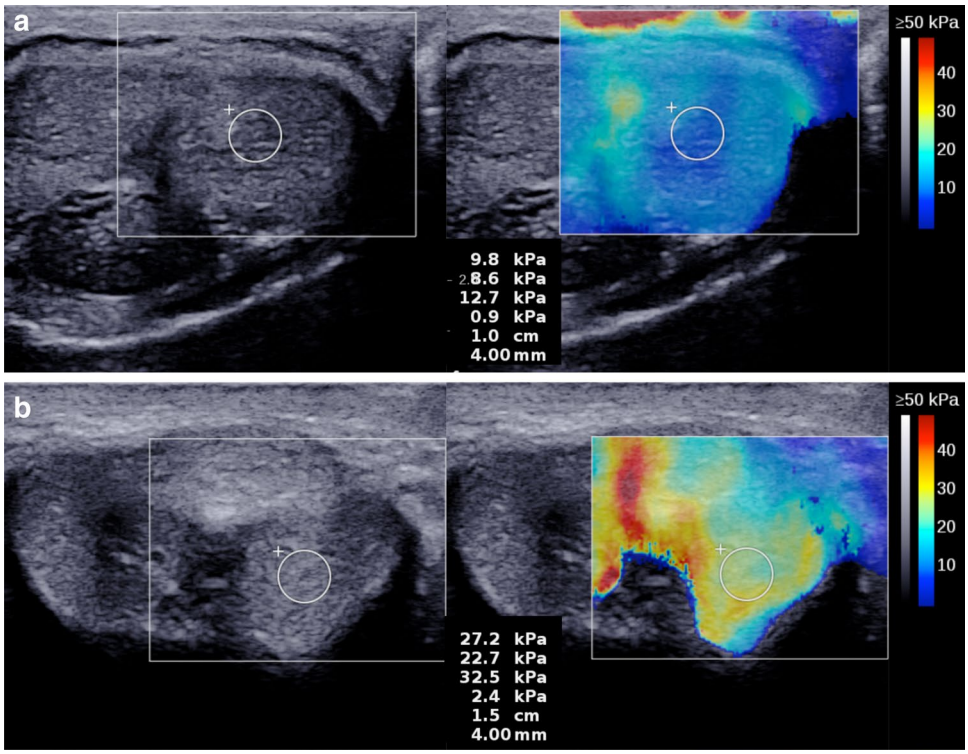

Fig. 22 Shear wave elastography of the penis. Stiffness of the cavernosal tissue in a 27-year-old man (**a**) and in a 68-year-old man (**b**) with normal erectile function. The patients were examined while the penis was flaccid. Shear wave measurements display a lower stiffness of 9.8 kPa in the young, compared with the old subject (27.2 kPa)

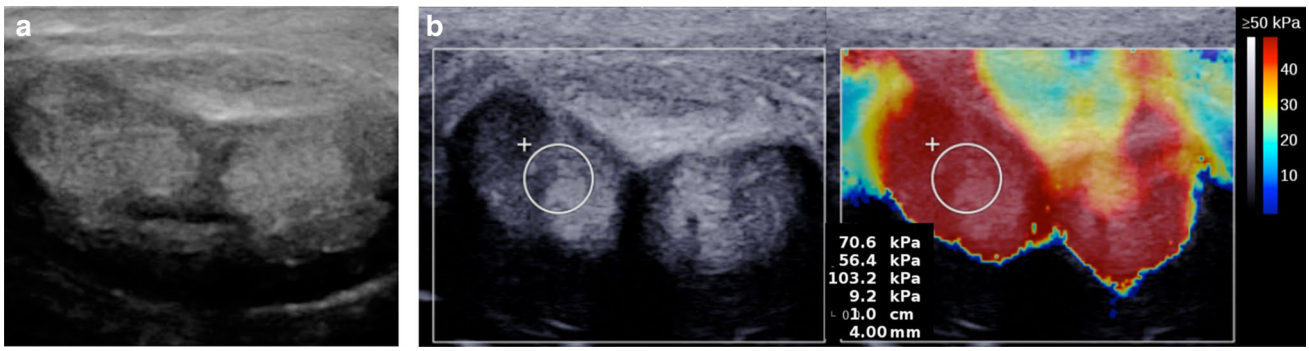

Fig. 23 Shear wave elastography of the penis in a 46-year-old patient with severe erectile dysfunction and cavernosal tissue fibrosis following prolonged ischemic priapism. **a** Axial grey-scale image shows increased echogenicity of the cavernosal bodies. **b** Shear wave elastography reveals high stiffness of 70.6 kPa of the cavernosal bodies, which were also hard at palpation

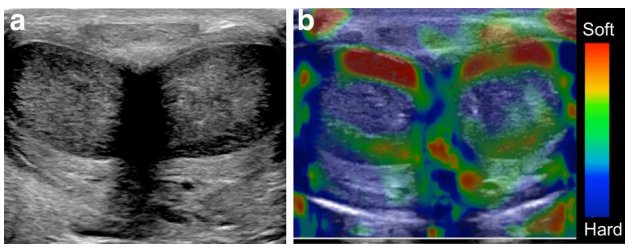

Fig. 24 Strain elastography of the penis in a 38-year-old patient with severe erectile dysfunction and cavernosal tissue fibrosis following prolonged ischemic priapism. **a** Axial grey-scale image shows increased echogenicity of the cavernosal bodies. **b**. strain elastography subjectively displays increased stiffness of the central portion of the cavernosal bodies (displayed in blue)

deliver therapy in plaques that are not evident at palpation and at grey-scale US [64, 65].

Use of US elastography for evaluation of erectile dysfunction is an active research field, but currently cannot be recommended for clinical use. Strain and shear wave elastography provide essentially different information. Most important, it has been shown that the tumescence degree affects the SWE values, as cavernosal tissue becomes more rigid and less elastic with erections [66]. This is not surprising, as the speed at which shear waves propagate through the tissues varies depending on stiffness and viscosity [67], and blood viscosity increases with increasing pressure, as occurs in the cavernosal bodies during the

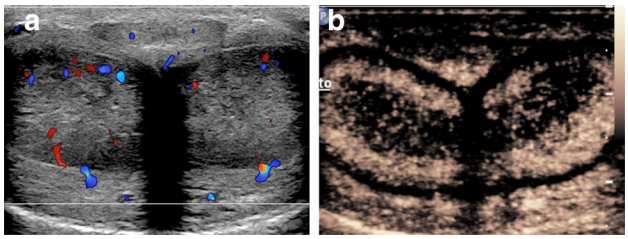

Fig. 25 Diffuse cavernosal tissue fibrosis in a patient with severe erectile dysfunction following prolonged ischemic priapism. **a** Color Doppler interrogation of the cavernosal bodies obtained after injection of 20 μg of prostaglandin E1 shows small peripheral vessels feeding the outer portion of the corpora cavernosa. Cavernosal arteries are obliterated. **b** CEUS shows peripheral enhancement of the corpora cavernosa. The central portion does not enhance

onset of erection [68]. Even if the penis is investigated while flaccid, manipulation with the transducer can induce a variable degree of engorgement, and hamper the measurements. Also, the cavernous septum hampers propagation of the shear wave.

CEUS has a limited role in imaging erectile dysfunction [69]. In complete penile corporeal septation, a rare congenital abnormality that can present occasionally with erectile dysfunction, cavernosal microbubble injection can help diagnosis showing unilateral corporeal enhancement [70]. In erectile dysfunction secondary to trauma and ischemic priapism one study suggests fibrosis is better delineated at CEUS as enhancement defects [71] (Fig. 25). CEUS can also be used to assess patency of shunts in patients with ischemic priapism who have undergone surgical shunting procedures [44].

Conclusion

In the era of PDE5 inhibitors penile Doppler assessment has diminished its importance. However, it continues to have a key role in the evaluation of specific patients presenting with erectile dysfunction, in particular, in trauma, priapism, and Peyronie's disease. In the appropriately selected patient, US and Doppler modes can be valuable to the urologist and help them decide the best treatment option. US elastography and CEUS are useful in selected cases only. The sonologist, either radiologist or clinician, who approaches this field must have a knowledge of the penile US and Doppler anatomy, of the clinical presentation of the most important penile pathologies presenting with erectile dysfunction, and of their appearance at multiparametric US.

Compliance with ethical standards

Conflict of interest All authors declare that they have no conflict of interest.

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