Vascular Diseases of the Testis

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Abstract

Palpable scrotal mass, acute scrotal pain, and enlarged scrotum are common scenarios in clinical practice. These symptoms are frequently caused by abnormalities and alterations of the vessels within the scrotal sack. This chapter reviews syndromes related to the vasculature of the scrotum, including both the arteries and veins. A miscellany number of less frequent diseases are also included to complete differential diagnostic evaluations. In addition, a full description of the anatomy of the vessels within the scrotal sack and a brief reminder of the embryological development of these vessels provide morphological background and complete this chapter.

Glossary of Terms

Extratesticular varicocele Abnormal enlargement or dilatation of the internal spermatic vein and the pampiniform venous plexus of the testis due to the inversion of venous blood flow within the spermatic cord.

Intratesticular arteriovenous malformation Arteriovenous malformation within the testicular parenchyma.

Intratesticular varicocele Abnormal enlargement or dilatation of the intraparenchymal testicular venous net.

Segmental testicular infarction Arterial ischemic testicular damage presenting with a typical ultrasound diffuse segmental pattern due to a number of various etiologies.

Testicular amyloidosis Intratesticular deposit of amyloid.

Testicular hemangioma Benign soft tissue neoplasm of the testis commonly presenting abnormal vascular growth pattern.

Testicular torsion Twist of the spermatic cord causing testicular ischemia.

Venous testicular infarction Ischemic damage to the testicle due to localized edema (commonly associated to a local inflammation-infection process) occluding the venous drainage of different portions/the entire testis.

Introduction

Palpable scrotal mass, acute scrotal pain, and enlarged scrotum are common scenarios in clinical practice. These entities are frequently caused by abnormalities and alterations of the vessels within the scrotum. Although these diseases are relatively rare, and seldom life threatening, they are commonly extremely painful, causing patient major discomfort; in cases of erroneous diagnoses, frequently, litigations follow (e.g., amputation of testis in case of overseen testicular torsion).

This chapter reviews the processes of scrotal and testicular vascular diseases, affecting both arteries (i.e., testicular torsion and segmental testicular infarction) and veins (i.e., intra- and extratesticular varicocele and venous testicular infarction). Despite their rarity, a number of diseases, including vasculitis, amyloidosis, arteriovenous malformations, and benign vascular tumors (i.e., hemangioma), have been also briefly reviewed to complete the differential diagnosis of these processes. In addition, a detailed description of the intrascrotal vascular anatomy and a brief description of the embryological development of these vessels have been included to provide morphological background and to complete this chapter.

Embryological Development of Male Gonadal Blood Supply

The blood supply to the primitive testis is derived from the 30 pairs of mesonephric arteries that arise from the aorta, about a third of which will contribute to the urogenital arterial rete that supplies the kidneys and the developing testes. The lowest of the pairs does not become obliterated and forms the testicular artery.

The testicular artery runs through the mesonephric fold above the testis and then passes caudally as an unbranched medial descending limb to enter the tunica albuginea at the lower pole. From there, it circles the lower pole to run cranially as the branched lateral ascending limb on the ventral and dorsal surfaces of the testis.

Vascular Anatomy of the Testes

External Testicular Arterial Supply

The arterial supply to each of the testis most often arises from the anterolateral surface of the aorta at L2-L3, just below the renal artery. However, these vessels may originate directly from the renal artery or even from one of its branches (Mostafa et al. 2008).

The right testicular artery runs over the psoas muscle and inferior vena cava (IVC), anterior to the genitofemoral nerve, the ureter, and the pelvic part of the external iliac artery to meet the spermatic cord at the internal inguinal ring. Its left counterpart passes behind the inferior mesenteric artery and the left colic artery but otherwise takes a course similar to that of the right artery (Skowronski and Jedrzejewski 2003; Yalçin et al. 2005).

Numerous investigators (Hundeiker and Keller 1963; Kormano and Suoranta 1971; McMinn 1995; Williams et al. 1995) have reported that the right testicular artery lies anterior to the IVC and posterior to the third part of the duodenum, right colic, ileocolic arteries, root of mesentery, and the terminal part of the ileum. The left testicular artery lies posterior to the inferior mesenteric vein, left colic artery, and the lower part of the descending colon. They cross anteriorly to the genitofemoral nerve, the ureter, and the lower part of the external iliac artery, passing to the deep inguinal ring to enter the spermatic cord. Terayama et al. (2005) showed that the testicular arteries run from the abdominal aorta to the testes, with various configurations (straight, meandering, spiral, or coiled). They suggested that these configurations may play several roles in protecting normal spermatogenesis, such as allowing wide mobility of the testes on physical attack, heat emission with the entangled pampiniform plexus, and reduction of the blood flow rate.

Different researchers (Merklin and Michels 1958; Nathan et al. 1976; Onderoglu et al. 1993; Ozan et al. 1995) described abnormal variations of the testicular artery course, origin, or number. In less than 20 % of cases, it passes behind the inferior vena cava, and in 12 % it arches over the renal vein (Notkovich 1955; Mirapeix et al. 1996). Loukas and Stewart (2004) reported an accessory left testicular artery originating from the ventrolateral wall of the descending aorta communicating with the left renal artery in addition to the normal right and left testicular arteries. Deepthinath et al. (2006) reported extratesticular arteries. Bhaskar et al. (2006) reported an abnormal course and branching of the right testicular artery arising from the anterior surface of the abdominal aorta dividing into two branches: one coursed inferiorly behind the inferior vena cava as the testicular artery proper, whereas the other passed behind it emerging on the anterior surface of the right kidney. After crossing, it bifurcated into an ascending branch to the right suprarenal gland and a descending branch ending in the posterior abdominal wall. Naito et al. (2006) reported two cases of the left testicular artery arching over the left renal vein before running downward to the testis. Mano et al. (2006) indicated that even with situs inversus, testicular vessels did not exhibit an inverted morphology. Rusu (2006) associated anatomic variants with the bilateral doubled testicular arteries: on the right side the medial testicular artery emerged from abdominal aorta, whereas the lateral one arose from the superior renal artery. On the left side, both arteries emerged as a common trunk from the abdominal aorta.

They descend in a straight course to its termination in the majority of cases, although they may show a convoluted course in up to 15 % of cases. In 6–8 % of the cases, each artery may divide high on the cord into an inferior testicular artery and an internal testicular artery (Harrison and Barclay 1948; Harrison and McGregor 1957).

Patterns of Testicular Artery Termination

There are three sites at which the testicular artery may terminate. In the majority of cases (80 %), the testicular artery runs distally until it reaches a position close to the testicular mediastinum, descending parallel along its entire length and terminating at the upper end of mediastinum testis

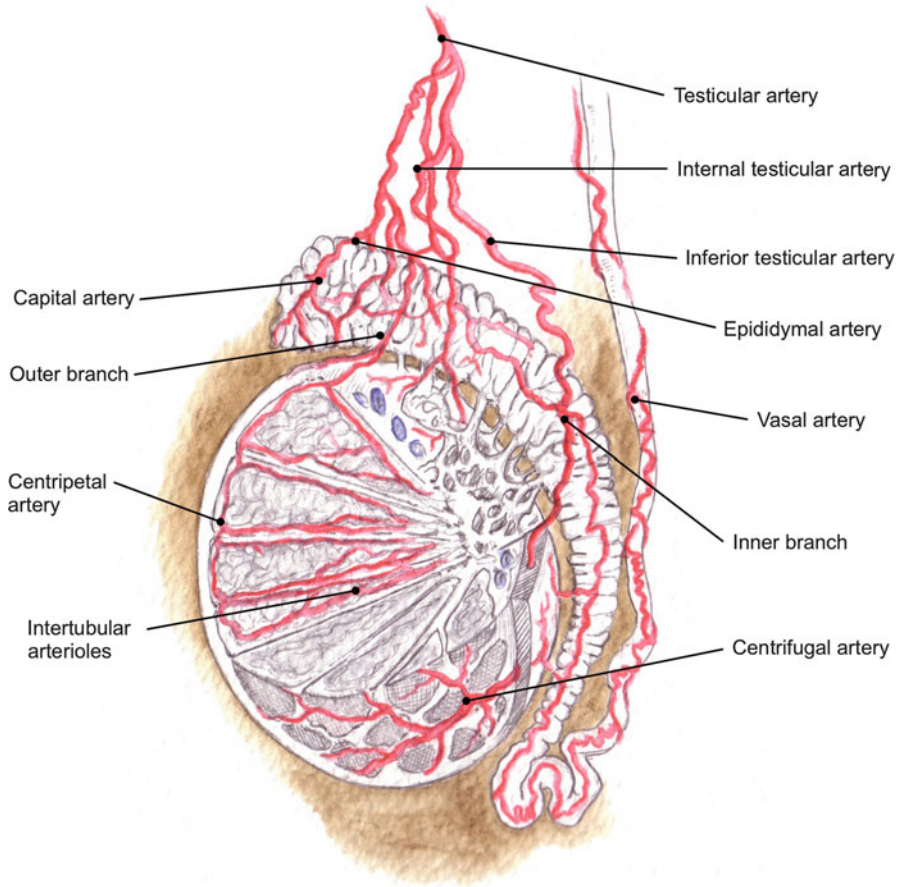

Fig. 1 Arterial vascular supply to the testis

close to the upper testicular pole. The testicular artery terminal branches are placed 3–4 cm from the upper end of the testis in approximately 15 % of cases, while in the remaining 5 % the testicular artery descends without a single division all along the mediastinum testis (Asala et al. 2001; Pais et al. 2004; Fig. 1).

Internal Arterial Distribution

As the testicular artery leaves the inguinal canal and approaches the upper end of the testis (if it has not done so at a higher level), it divides into two main branches: (i) an outer branch, the internal testicular artery, and (ii) an inner branch, the inferior testicular artery. Surrounded by the pampiniform plexus, the tortuosity of the testicular arteries increases as they approach to the testicle. This configuration may be considered a heat-exchange system to cool the arterial blood at this level.

The inferior testicular artery runs between the posterior testis surface and the epididymal body, branching and entering through the posterior border of the testis at several different points. In contrast, a number of branches from the internal testicular artery pass through the tunica albuginea to enter the tunica vasculosa. Taken together these vessels form an aggregation which appears as a true vascular hilum in the posterior testicular aspect. Considerable variation is found in the branching and distribution of these vessels as they enter the testicular parenchyma, but they commonly ramify at the level of the tunica vasculosa to get inside the parenchyma as the

coiled centripetal arteries. The centripetal arteries run toward the rete testis and, in turn, put out branches that completely reverse the course and return also as centrifugal arteries. Both sets of arteries divide further inside the parenchyma and end as intertubular arterioles (Cordier et al. 1938; Kormano 1967; Kormano and Suoranta 1971; McMinn 1995). The capillaries that arise from the arterioles enter the interstitial tissue and are separated from the germinal and supporting cells by a basement membrane that constitutes the "blood-testis barrier" (Ploen and Setchell 1992).

The rete testis is therefore supplied by small vessels from the testicular mediastinum and from the small centripetal branches of the internal testicular artery. The tunica albuginea has its own set of capillary networks that are not related to the proper testis vascularization. Conversely, they arise independently from the branches of the testicular artery, from the vessels supplying the rete, and from the branches of the epididymal artery. The epididymal artery branches directly from the testicular artery at a level proximal to the epididymis. It supplies the head and the body of the epididymis through one or more capital arteries. The tail is vascularized by a complex network involving the epididymal, vasal, and testicular arteries, with supplementation from the cremasteric artery. This system provides an extensive anastomotic loop among these vessels (Cooper 1830; Hill 1909).

The artery of the vas deferens (i.e., deferential artery) arises as a branch from the inferior vesical artery giving several branches to supply the vas deferens along its course and terminates with several capsular branches close to the mediastinum testis. Some of the vasal branches anastomose freely (i.e., following an irregular pattern) with branches of the testicular artery along the mediastinum and close to the lower end of the testis to form an epididymal-deferential loop (Harrison 1949; Yalçin et al. 2005).

The cremasteric artery arises from the inferior epigastric artery close to the deep inguinal ring to enter the inguinal canal supplying the cremasteric contents. It runs outside the internal spermatic fascia and so supplies an extremely limited amount of blood to the structures within. However, some terminal branches may reach the lower pole of the testis and anastomose with the epididymal-deferential loop, with a terminal branch of the testicular artery (80 %) or with branches of the epididymal artery (20 %) (Harrison 1949).

Vascular Testicular Areas

According to the amount of vascular supply, a number of different vascular areas can be distinguished in the testes: (i) rich vascular areas (upper polar, mediastinum testis, and posterolateral segments), (ii) moderate vascular areas (middle third of the lateral surfaces), and (iii) poor vascular areas (anterior border and anterolateral surfaces) (Harrison and Barclay 1948; Fig. 2).

Extratesticular Venous Pathways

The very variable number and distribution of veins draining the testis, epididymis, and vas deferens connect with a deep and superficial venous network.

The most common pathway of the deep venous network has three components: (i) an anterior set composed of the pampiniform plexus and the testicular vein, (ii) a middle set composed of the deferential vein, and (iii) a posterior set composed of the cremasteric veins (Asala et al. 2001).

The veins emerging from the testis and from the superficial plexus overlying the anterior part of the epididymis form the anterior portion of the deep venous network. These vessels typically provide as many as 10 branches, which anastomose to form a mesh-like complex of large veins, the pampiniform plexus. Pampiniform plexus courses about the testicular artery and in front of the vas deferens in the spermatic cord. As noted, the pampiniform plexus is closely associated with the tortuous branches of the testicular artery below the external inguinal ring, a relationship that may act as a heat-exchange mechanism (Hill 1909; Asala et al. 2001).

The vessels of the pampiniform plexus become reduced to three or four veins and then to two as

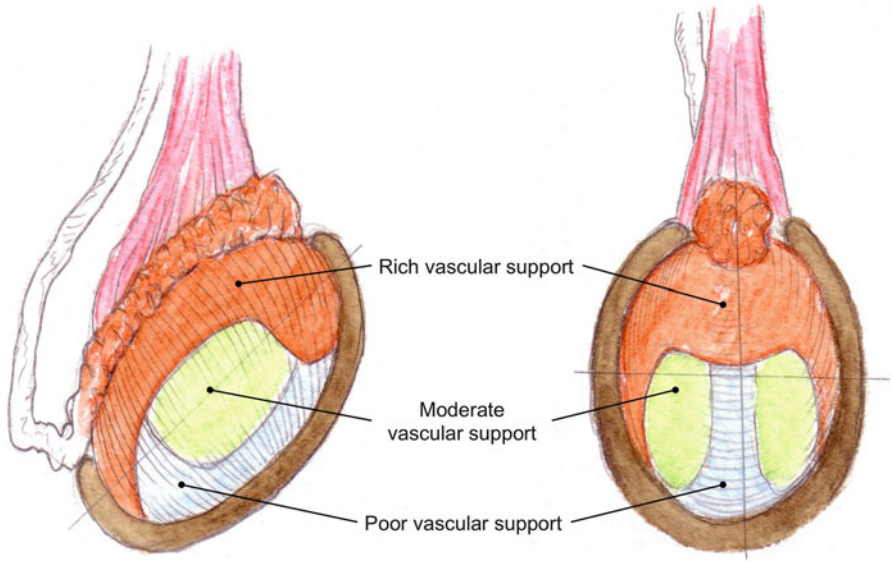

Fig. 2 Distribution of vascular areas in the testis

they pass through the external inguinal ring. The two veins combine into a single vein that ascends in the pelvis as the testicular vein lateral to the artery and posterior to the ureter. The left testicular vein deviates medially from the accompanying artery below the lower pole of the kidney and ascends vertically to uniformly enter the left renal vein, usually lateral to the adrenal vein, joining it most often at a right angle. The right testicular vein enters the anterolateral surface of the vena cava obliquely at a site below the renal vein, at the level of the second lumbar vertebra, and below the exit of the testicular artery from the aorta. In 10 % of cases, it drains into the renal vein (Asala et al. 2001).

The middle set is made up of the funicular and deferential veins. The funicular veins drain the posterior part of the epididymis into the inferior epigastric and external iliac veins. The deferential veins accompanying the vas deferens drain into the venous plexus of the testicular cord as well as the prostatic plexus and vesical plexus (Asala et al. 2001).

The posterior set, collecting from the cremasteric veins, becomes separated from the cord near the external ring. It drains into the internal saphenous or the inferior epigastric vein.

The superficial venous network is made up of the scrotal veins which drain through the external pudendal vein into the internal saphenous vein or through the superficial perineal veins into the internal pudendal vein. Within this system, the cremasteric vein joins the venous plexus of the spermatic cord and the inferior epigastric vein. Small anastomotic channels occur frequently between the superficial and deep systems and between the elements of each (Hill 1909; Asala et al. 2001; Fig. 3).

Intratesticular Venous Drainage

The veins arise diffusely from a dense microvascular bed about the tubules. They join collecting venules, which pass either peripherally or centrally, in contrast to the arteries, which are organized peripherally in the testis. The peripherally directed veins reach the tunica vasculosa and continue on the anterolateral aspect of the testis, where they form large channels on the surface. The centrally directed veins, providing the principal drainage of the testis, run to the rete testis, pass through the posterior surface of the tunica albuginea at the mediastinum, and are joined by

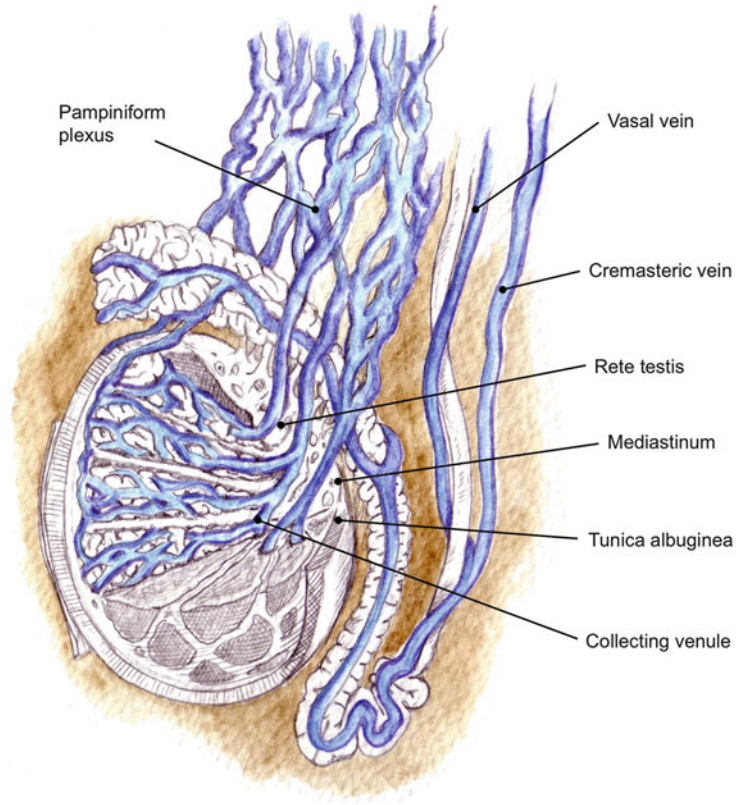

Fig. 3 Venous drainage of the testis

veins from the anterior portion of the epididymis before they reach the pampiniform plexus.

Testicular Arterial Diseases

Testicular Torsion

Torsion (rotation) of the testis with twisting of the spermatic cord produces a blockage of blood flow at this level. Torsion represents a surgical emergency; if left untreated total loss of the testis on the affected side may follow (Bennett et al. 1987). The damage to the testis depends on the degree and duration of local ischemia and reperfusion damage.

Epidemiology and Risk Factors

Testicular torsion is one of the most common surgical emergencies occurring in young males, with a calculated annual incidence of 1:4,000 among those under 25 years old. Although it has been seen from birth to 77 years of age, there are two peaks in incidence; it is most frequent at puberty (65 % of all torsions) with another much smaller peak in the first year of life (Anderson and Williamson 1988; Melekos et al. 1988).

Any malformations or abnormalities that permit greater testicular mobility or facilitate scrotal rotation increase the risk of torsion. The most common of all these malformations occurs when the tunica vaginalis wraps improperly around the spermatic cord and prevents the testis to attach to the posterior scrotum (Ringdahl and Teague 2006). The tunica vaginalis normally covers the anterior surface of the testis and extends over varying distances across the epididymis and the spermatic cord. Where the coverings extend up the cord (so-called "bell-clapper" variant), the testis is suspended freely within the tunical cavity, within which it may rotate around its narrow vascular pedicle (Caesar and Kaplan 1994; Fig. 4).

Other predisposing factors include rapid increase in testicular volume such as germ cell

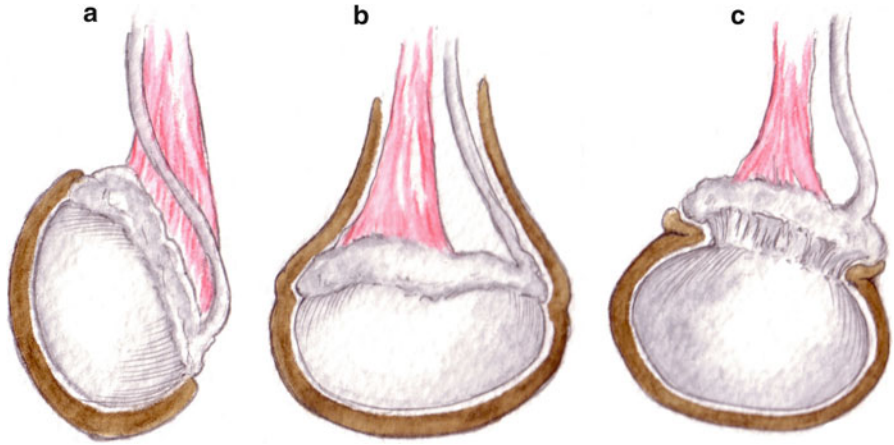

Fig. 4 Anatomic variations in testicular torsion. (**a**) Normal anatomy. (**b**) The "bell-clapper deformity." (**c**) Loose epididymal attachment to the testis

neoplasias (typically producing an "intravaginal" torsion in puberty), testicles with horizontal lie, a history of cryptorchidism, a spermatic cord with a long intrascrotal portion (typically in neonatal period producing an "extravaginal" en-masse twist), and, less commonly, genital trauma (Ringdahl and Teague 2006).

Pathophysiology

Ischemic testicular damage resulting from torsion is related to both the number of turns on the spermatic cord, i.e., severity of ischemia, and the duration of rotation-induced ischemia. Hence, all cases with a torsion of greater than 360° and lasting 24 h or longer will results in ipsilateral testicular loss secondary to severe ischemic atrophy if the testis is left in situ. However, severe atrophy has also been reported with similar degrees of rotation after as little as 4 h of torsion (Tryfonas et al. 1994). The threshold for irreversible damage seems to be around 8 h, before which testicular atrophy is rare and after which it is the rule (Bartsch et al. 1980).

Effect of Testicular Torsion on Fertility. Different experimental studies have shown that bilateral testicular damage and reduced fertility may result from unilateral torsion (Rabinowitz et al. 1985). The proponed mechanism of this injury is a disruption of the "blood-testis barrier" and the release of sperm antigens from the damaged testis (raised in serum after torsion), causing an immune response (that can be managed with immunosuppressive agents) in which both humoral and cellular mechanisms have been proposed. Fertility is related to the duration of torsion only if the ischemic testis is retained in the scrotum after untwisting. Early removal of the damaged testis enables preservation of contralateral histology and maintenance of normal fertility (Bartsch et al. 1980; Rabinowitz et al. 1985).

Prepubertal torsion in absence of antigens from immature spermatozoa does not appear to affect fertility. A number of experimental clinical studies showing normal contralateral testicular growth and semen analysis after torsion support this idea (Rabinowitz et al. 1985; Tryfonas et al. 1994). In addition, significant preexisting histological abnormalities were identified in half of the patients and suggest that infertility may be related to a preexisting testicular pathology (Hadziselimovic et al. 1986).

Necrotic testes should be removed in this age group, and the possibility of adjunctive pharmacological immune manipulation at the time of surgery is a possible option for the future. Another etiology of diminished testicular function has been proposed by groups that have routinely biopsied the normal testis after untwisting.

Clinical Presentation

The classical presentation of testicular torsion is the sudden onset of severe, unilateral pain that is often associated with nausea and emesis. Pain is present with palpation and at rest. A history of similar intermittent episodes may suggest intermittent testicular torsion.

Childhood and Peripubertal Torsion. Testicular torsion usually causes sudden onset of pain as compared with other causes of acute scrotum (in up to 90 % of cases) (Anderson and Williamson 1988), but the history does not always follow this classical pattern. The duration of pain is critical in determining the risk of prolonged ischemia and subsequent testicular loss. There is a 90 % chance of salvaging the testicle when ischemia has been present for less than 6 h, which decreases to 50 % at 12 h and 10 % at 24 h (Ringdahl and Teague 2006). Scrotal pain, which may be the earliest and predominant symptom, was associated with groin, abdominal, or thigh pain in 34 % of these patients. Referred urinary symptoms may be noted in up to 5 %, while nausea and vomiting may be seen in approximately 40 % of patients (Anderson and Williamson 1988).

On the affected side the body of the testis is tender and swollen and rides high in the scrotum, with a thickened tender cord. The cremasteric reflex is usually absent, and a detectable secondary hydrocele is found in 52 % of patients. Mild fever and scrotal erythema, edema, or ecchymoses should be readily identifiable. These are in general late signs, commonly associated with low testicular salvage rates (Anderson and Williamson 1988). However, the absence of edema or erythema or the presence of a cremasteric reflex does not rule out the possibility of acute testicular torsion, especially if the onset of pain was recent.

Neonatal Torsion. About 10 % of all torsions occur in the neonatal period and these can be subdivided into two groups, depending on whether the condition is seen at birth (prenatal torsion, 70 %) or occurs later in previously normal testes (postnatal, 30 %) (Das and Singer 1990). Prenatal torsion occurs extravaginally and is thought to be caused by the action of the cremaster muscle on the mobile testis and its coverings. It presents at an early nappy change or at the postnatal check as a firm asymptomatic testicular mass. The testicle may be in a high or inguinal position, with bruising of the scrotal skin. The differential diagnosis of neonatal scrotal swelling includes hydrocele, hernia, testicular tumor, and meconium peritonitis.

Most authors agree that immediate exploration of the prenatally twisted testis rarely if ever results in ipsilateral testicular salvage. In their review, Das and Singer (1990) found no viability at exploration in 80–100 % of cases, and these testes were removed, with fixation of the other side. Adjunctive tests (Doppler ultrasonography and isotope imaging) may be applicable in this group to confirm the diagnosis but will also be more difficult to perform. The need to exclude tumor and fix the other side to prevent bilateral involvement is a strong argument for urgent exploration. If tumor is a possibility, an inguinal incision is preferred. The increased risks of anesthesia in the newborn can be minimized by management in a specialized center (La Quaglia et al. 1987; Fig. 5).

Diagnosis

History and Physical Examination. A child with acute scrotal pain must be presumed to have spermatic cord torsion regardless of age until proven otherwise; however, in some cases, an accurate evaluation may save the child an unnecessary surgical exploration. In examining a boy with possible torsion, the surgeon may be the latest in a succession of doctors to upset him. It is therefore important to spend time reassuring him and his parents and gaining his confidence to obtain a worthwhile examination.

The first step in diagnosing torsion is a thorough history, including onset, location, and intensity of the pain. Observation of the child's general appearance and level of distress should be recognized. The acute scrotum should be examined carefully to determine the true etiology. Testicular assessment should be an automatic part of the abdominal examination in any boy, and this good habit will avoid missing those torsions presenting with more "abdominal" symptoms.

In general, the examiner should begin with the normal side where a horizontal lie to the testis

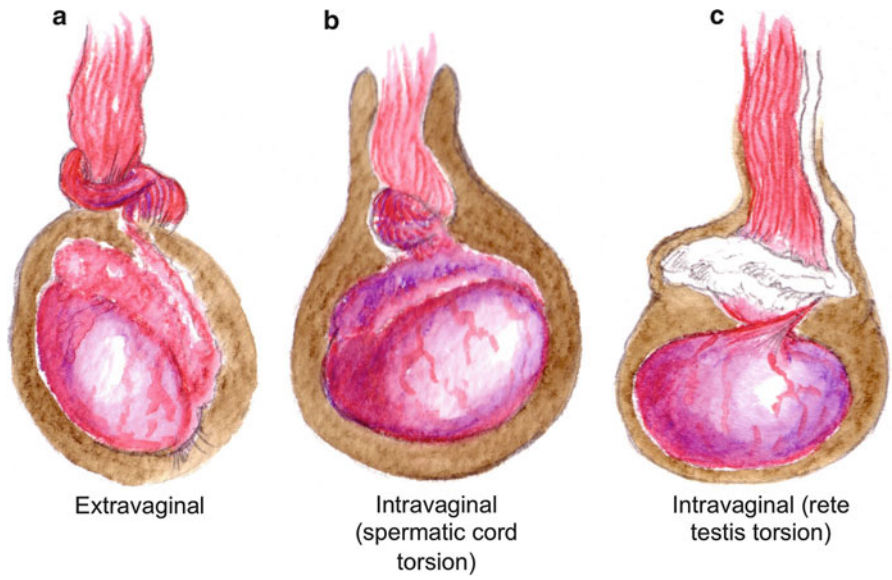

Fig. 5 Extravaginal (**a**) and intravaginal (**b** and **c**) testicular torsion

suggests a "bell-clapper" deformity (Anderson and Williamson 1988). To begin the scrotal examination, the inguinal canal should be inspected on each side for signs of asymmetry or mass. Testicular torsion may present with varied clinical findings, but the involved testis often demonstrates signs such as higher riding in the hemiscrotum, a transverse orientation, an anterior epididymis, absent cremasteric reflex, and tenderness of the testis and epididymis. Associated scrotal wall swelling or erythema is suggestive of spermatic cord torsion if presentation is delayed. Assessing the cremasteric reflex is a key component of the physical examination. However, there have been case reports describing patients with a positive cremasteric reflex and testicular torsion (Schmitz and Safranek 2009). Other examination findings are also useful but less reliable than absence of the cremasteric reflex (Fig. 6).

Neonatal extravaginal testicular torsion can occur prenatally, resulting in a firm, enlarged, nontender mass in the hemiscrotum that is usually associated with dark discoloration of the overlying skin. A normal scrotal examination at birth and subsequent development of erythematous, tender, edematous hemiscrotum suggests postnatal extravaginal testicular torsion and should be addressed immediately with surgical intervention if the neonate is clinically stable.

Imaging. In recent years the high rates of so-called unnecessary explorations have led to the search for adjunctive tests to improve the accuracy of diagnosis. Reports of patients with positive blood flow on Doppler ultrasonography who in fact had torsion reinforce the view that the diagnosis should be clinical and that the priority is expeditious surgical management (Steinhardt et al. 1993). Negative explorations are a necessary consequence of this approach (Bartsch et al. 1980; Tryfonas et al. 1994).

Because no examination finding can completely rule out torsion, ultrasound with Doppler or scintigraphy may be done to confirm or rule out the diagnosis (Marcozzi and Suner 2001; Lavallee and Cash 2005; Abul et al. 2005; Ringdahl and Teague 2006). However, both methods are subject to availability of equipment and personnel and require the cooperation of the patient. They should not be used routinely unless the delay involved in obtaining them is minimal. They can provide useful information in patients in whom the diagnosis of torsion seems unlikely or the duration of symptoms indicates a dead testis if torsion is the cause. If suspicion for torsion is

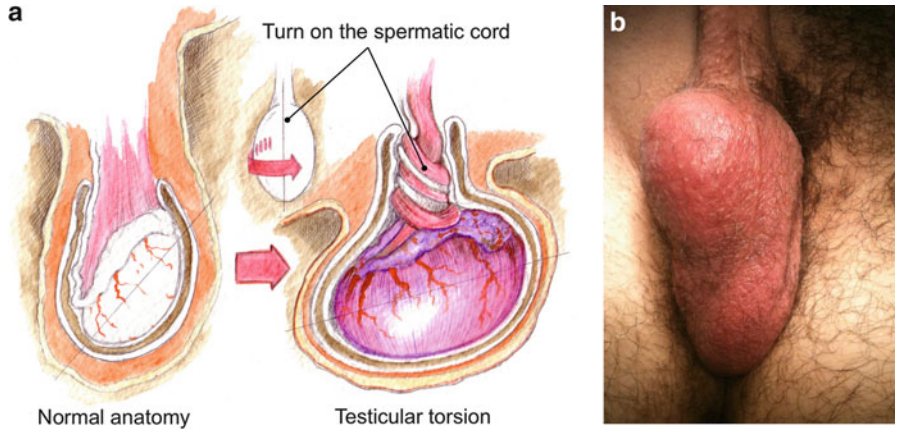

Fig. 6 Mechanism of testicular torsion (**a**) and clinical findings in the scrotum (**b**). The involved testis demonstrates a higher riding in the hemiscrotum, a transverse orientation, and an anterior epididymis

high, it is reasonable to proceed directly to surgery. A misdiagnosis of testicular torsion leading to an unnecessary "scrotal exploration" does not lead to medical litigation. On the contrary, a patient who has a testicular torsion misdiagnosed and loses a testis may proceed to successful medical litigation (Cavusoglu et al. 2005).

Differential Diagnosis

The differential diagnosis of the acute scrotum includes testicular torsion, torsion of the appendix testis or epididymis, epididymitis/orchitis, hernia/hydrocele, and other more rare conditions (i.e., trauma, sexual abuse, tumor, idiopathic scrotal edema, dermatitis, cellulitis, and vasculitis) (Das and Singer 1990). Although any acute scrotal swelling must be regarded as testicular torsion until proved otherwise, the ability to confidently identify these other conditions may avoid unnecessary surgery.

Torsion of the Appendix or Epididymis. There are five different testicular appendages described. The most prevalent is the appendix testis, which lies on the upper pole of the testis and is a vestigial remnant of the Müllerian system, found in around 90 % of males and bilaterally in 60 %. The other appendages are all remnants of the Wolffian system and are found on the epididymis (in half of the male population) or more rarely on the cord itself or in the epididymo-testicular groove. About 95 % of twisted appendages involve the appendix testis. There is one peak in presentation at <10 years old, which is earlier than the peak for torsion (Melekos et al. 1988). Abrupt pain suggests spermatic cord torsion or torsion of one of the testicular appendices. However, the pain may be of a more gradual onset, and associated symptoms of nausea, vomiting, and abdominal pain are more rare (Fig. 7).

Examination may reveal a focal tenderness, usually at the upper pole of the testis, with a nontender testis and epididymis. It is often associated with a reactive hydrocele. A palpable tender nodule at the upper pole alludes to the diagnosis, and a visible engorged hydatid or "blue dot" (i.e., reflective of a necrotic appendix), seen through the scrotal skin, is pathognomonic (Melekos et al. 1988).

A clear case of a twisted appendage can be managed conservatively with a combination of bed rest, scrotal support, and nonsteroidal anti-inflammatory drugs (NSAIDs). If the scrotum is explored to confirm the diagnosis, the appendage is commonly excised. Although an asymptomatic appendage on the other testis is quite likely, it is not explored, except where a bell-clapper deformity is found incidentally, for which a bilateral orchidopexy is required.

Epididymitis/Orchitis. Gradual onset of the pain is more consistent with inflammation of the epididymis/testis. Idiopathic epididymitis classically has a gradual onset and is not associated with

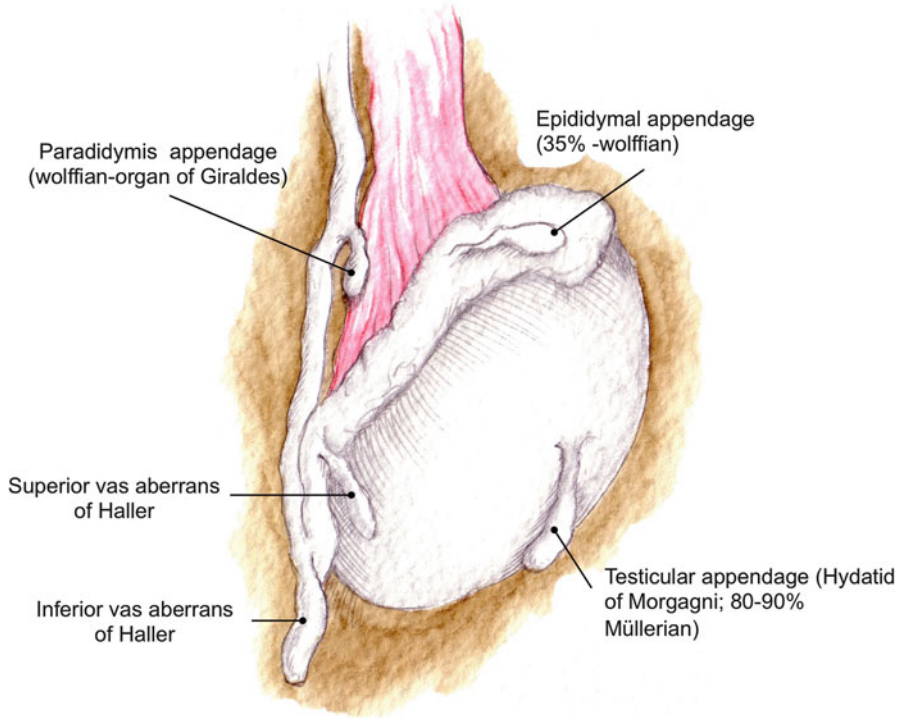

Fig. 7 Testicular appendages. The scheme evidences the most frequent location of testicular and epididymal appendages

nausea or emesis but can have similar clinical signs, including a firm, tender, enlarged testis with an erythematous and edematous scrotum.

Infective inflammation causes also scrotal pain and swelling. This condition is rare in children. They are usually associated with a gradual onset of pain, fever, and urinary tract symptoms. Commonly, no other sign of urinary infection is present and microbiology of the urine is rarely confirmatory. When urinary infection is confirmed, a complete work-up to exclude underlying urinary tract anomalies has to be performed (Das and Singer 1990).

Hernia/Hydrocele. A fullness or mass suggestive of a hernia or hydrocele of the spermatic cord may be identified by palpation at the inguinal canal. A hydrocele can be distinguished from hernia by transillumination, as well as from the absence of a mass in the inguinal canal. In the absence of volume changes within the hydrocele, the processus vaginalis is usually not patent, and the newborn hydrocele resolves by 1 year of age without surgery. Depending on the features of the hernia, it can be managed conservatively or, on the contrary, operated to avoid derived complications.

Idiopathic Scrotal Edema. Under this condition the skin is commonly reddened and thickened, but the underlying testis and epididymis are not tender. The edema may extend upward into the groin or downward into the perineum in direction to the anal verge. It generally occurs in boys below 7 years of age, is frequently bilateral, and resolves over 2–3 days with no active management. Few of these patients proceed to surgery, because the diagnosis is usually apparent (Melekos et al. 1988).

Other diagnoses that may rarely mimic torsion include tumor, varicocele, and trauma. Although these are all associated with scrotal swelling, specific aspects of their histories and clinical findings make them easier to differentiate from torsion. Henoch-Schönlein purpura can present with an acutely tender scrotum, but the characteristic skin manifestations elsewhere are diagnostic;

simultaneous torsion is rare but has been reported in this condition (Das and Singer 1990).

Management

The simple suspicion of testicular torsion (i.e., abrupt onset of testicular pain) represents a true surgical emergency, and thus exploration should be undertaken as soon as possible. Some advocate attempting to untwist the testis by rotating it away from the midline, to minimize the ischemic period. Therefore, manual detorsion at the emergency room is an option. Nevertheless, although it may bring relief from symptoms, this maneuver may also be unsuccessful and cause great distress to the patient. Even if it works it should not delay the prompt surgical confirmation of the diagnosis and definitive management.

Patients with intermittent torsion may have few or no clinical findings despite a history that is highly suggestive (Anderson and Williamson 1988; Melekos et al. 1988). It appears that many torsion cases can resolve by spontaneous untwisting of the cord, but this is a warning that should not be ignored (Schulsinger et al. 1991). Hence, if a patient presents with a history of intermittent testicular pain, the surgeon should have a good reason for not carrying out an urgent bilateral orchidopexy.

The affected testis is commonly approached through a midline scrotal incision and the diagnosis of testicular torsion is then confirmed. The cord is untwisted and the testis wrapped in a warm saline-soaked swab. While the result of reperfusion is awaited in the affected testis, the contralateral testis may be explored and conveniently fixed (i.e., scrotal orchidopexy or septopexy) to prevent metachronous testicular torsion on that side. This should be carried out whether a bell-clapper deformity is found or not (Ringdahl and Teague 2006). Orchidopexy/septopexy should be performed with the tunica vaginalis everted and at least two 5-0 polypropylene sutures between the tunica albuginea of the testis and the dartos (Bartsch et al. 1980; Anderson and Williamson 1988; Tryfonas et al. 1994). Testicular salvage depends on the degree of torsion and on the time that has elapsed until repair. Detorsion within 6, 12, and 24 h of torsion results in a salvage rate of 90 %, 50 %, and less than 10 %, respectively (Anderson and Williamson 1988).

The twisted testis is reappraised thereafter and carefully observed for reperfusion signs. If the ipsilateral testis appears healthy, an orchidopexy/septopexy is performed. Conversely, if the affected testis is obviously black and dead, it may not be able to be salvaged. Likewise, if obviously necrotic testes are left after surgery, a high proportion of them will spontaneously discharge through the wound (Anderson and Williamson 1988). Therefore, nonviable necrotic testes (32 % of cases despite emergent scrotal exploration and detorsion) should be removed by orchiectomy.

Special Situations

Previous Testicular Surgery. Testicular torsion has been reported associated with previous ipsilateral orchidopexy and testicular biopsy. Inadequate fixation at the previous operation or single adhesions forming an axis for the testis to twist around were blamed. Eversion of the tunica and the use of nonabsorbable sutures at orchidopexy were suggested to prevent this occurrence (Thurston and Whitaker 1983). Sutureless fixation using the dartos pouch technique is the preferred method of fixation in these cases (Bellringer et al. 1989).

Solitary Testes. Investigation of patients with impalpable testes often reveals a blind-ending vas and vessels leading to a variable sized nubbin of testicular remnant in the scrotum. These absent testes may constitute 10 % of patients with the initial diagnosis of cryptorchidism. These features reveal a perinatal or "silent" torsion as the etiology of testicular loss (Tureck et al. 1994). In all of these cases, the remaining testis should be fixed, and this policy should be applied to solitary testes after trauma or tumor excision. There will otherwise remain a theoretical risk of torsion in these patients (Mishriki et al. 1992).

Segmental Testicular Infarction

Segmental testicular infarction is uncommon. Although the vast majority of these cases are

idiopathic in origin, different predisposing factors have been reported including polycythemia, intimal fibroplasia of the spermatic artery, sickle cell disease, and trauma (Fernandez-Perez et al. 2005).

Clinically, segmental infarction presents with testicular pain (Fernandez-Perez et al. 2005). Conversely, germ cell tumors are incidental findings and commonly present asymptomatic. The diagnosis is often made following orchiectomy for a suspected germ cell tumor (Han et al. 1994). However, improved imaging (i.e., color Doppler ultrasound) allows for a more confident assessment.

Ultrasound shows a wedge-shaped low reflective area (Fernandez-Perez et al. 2005) without posterior acoustic enhancement and associated frequently with focal expansion of the testes (Sriprasad et al. 2001). The absence of intralesional color Doppler flow is paramount in differentiating segmental testicular infarction from germ cell tumors (increased color Doppler flow) (Bushby et al. 2001) and, together with the clinical presentation, may lead to conservative testes-sparing surgery (Sriprasad et al. 2005).

Testicular Venous Diseases

Varicocele

A varicocele is an abnormal enlargement or dilatation of the internal spermatic vein and the pampiniform venous plexus of the testis due to the inversion of venous blood flow within the spermatic cord.

Varicoceles are progressive lesions that may hinder testicular growth and function over time and are the most common and correctable cause of male infertility worldwide, given that approximately 40 % of men with primary infertility have a varicocele and more than half of them experience improvements in semen parameters after its repair (Bennet 1889; Tanrikut et al. 2011).

Although the understanding of the pathophysiology, treatment, and outcomes of this entity has evolved greatly over the past decades, experts continue to debate the efficacy of surgical repair in improving fertility. The decision to treat adolescents with varicocele is controversial. In one hand, if all adolescents with varicocele were treated, a large percentage of them would go on an inappropriate, costly, and potentially unnecessary procedure. In the other hand, however, waiting until the onset of a possible irreversible infertility in the adulthood would be equally unacceptable. The task for the treating physician is, therefore, to identify those adolescents who are at greatest risk for infertility in adulthood, in an effort to offer them an early surgical intervention.

Epidemiology

Varicoceles are one of the most commonly identified scrotal abnormalities. Although rare in pediatric populations, its prevalence markedly increases with pubertal development to approximately 15 % by the late teenage years, a rate similar to that in adult populations (Buch and Cromie 1985; Khera and Lipshultz 2008). Due to differences in venous drainage (see below), 90 % of varicoceles occur on the left side. However, bilateral varicoceles occur in more than 50 % of patients (Amelar and Dubin 1987). In addition, this condition can be detected in 19–41 % of patients with primary infertility and 45–81 % of those with secondary infertility (Dubin and Amelar 1971; Witt and Lipshultz 1993; Nagler and Martinis 1997; Agarwal et al. 2007).

History

Varicoceles were recognized as early as the first century by Celsus, who described dilation of scrotal veins and noted an association between a varicocele and testicular atrophy (Kaufman and Nagler 1987). Later, in the sixteenth century, Ambroise Paré (1500–1590) described this vascular abnormality as containing "melancholic blood." In 1856, Curling was the first to describe a varicocele's association with infertility when he reported decreased "secreting powers of the gland" in the presence of a varicocele (Curling 1856). In the twentieth century, many other surgeons reported an association with an "arrest of sperm secretion" and subsequent improvement in fertility after varicocele repair. It was not until 1955, however, that Tulloch (2001) first reported the results of treating infertility with varicocele repair. Currently, a varicocele repair is the world's

most commonly performed male infertility procedure (Schlesinger et al. 1994).

Features for Varicocele Formation

The formation of a varicocele has been attributed predominantly to (i) anatomic variance, (ii) increased pressure in the left renal vein, and (iii) incompetence or congenitally absent valves.

Anatomic Variance. Several anatomic differences between the right and left testicular (internal spermatic) veins are thought to contribute to this predominance. Although highly variable, the left system usually consists of one or more veins within the spermatic cord that coalesce in the retroperitoneal space to become the testicular vein. The left testicular vein inserts into the left renal vein at a right angle, whereas the right testicular vein joins the IVC at an oblique angle. The relative greater blood flow in the IVC is thought to augment drainage on the right (Shafik et al. 1990).

Increased Pressure in the Left Renal Vein. The left testicular vein is 8–10 cm longer than the right, with a proportional increase in pressure. Increases in left renal vein pressure also have been noted secondary to two nutcracker phenomenon mechanisms (Coolsaet 1980): (i) a proximal nutcracker phenomenon which describes compression of the left renal vein as it passes between the aorta and superior mesenteric arteries and (ii) a distal mechanism that involves retrograde blood flow through the deferential and external spermatic veins caused by compression of the left common iliac vein as it courses under the left common iliac artery.

Congenitally Absent or Incompetent Valves. Absent or incompetent valves have been classically pointed out as the primary cause of varicocele, contributing in a great extent to its formation and severity (Johnson et al. 2004). However, subsequent research has shown that varicocele may be absent with incompetent valves or even without them (Wishahi 1992; Braedel et al. 1994). Conversely, in the presence of normal valves, dilation of the testicular vein can cause functional incompetence as a result of loss of coaptation (Gorenstein et al. 1986).

Varicocele is diagnosed only in humans and it seems to be associated with the erect position whereby one-way valves in the spermatic veins ensure the exit of waste products against gravity. Dysfunction of the internal spermatic vein valves with age increases the pressure up to eight times above the physiologic level in the venous drainage, which deviates testicular venous flow to other horizontal routes.

Several microscopic venous changes can be pointed out as alterations that may cause a varicocele. However, it is difficult to know if these changes are the cause or the consequence of the varicocele formation process. The main histological alterations described in the wall of the spermatic plexus veins of varicocele patients include the following: (i) the alteration of the vasa vasorum of the tunica adventitia, (ii) the reduction of the longitudinal and oblique smooth muscle cells, (iii) the increase in connective tissue, and (iv) the decrease in the circular smooth muscle cells of the tunica media (Iafrate et al. 2009).

Risk Factors

Although the exact mechanisms have yet to be elucidated, several physical findings have been found to be associated with an increased risk for developing varicocele in adolescence. A low body mass index has been found to be associated with the development of varicocele. It has been shown that varicoceles are less frequent in obese men, possibly because abdominal fat protects the left renal vein from becoming compressed between the aorta and superior mesenteric artery (Nielsen et al. 2006; Practice Committee of the American Society for Reproductive Medicine 2006). Eventually, a retroperitoneal mass that obstructs the spermatic vein along its course may produce a varicocele.

In addition, physical development in adolescence also carries physiologic changes in serum testosterone levels that may contribute to varicocele formation by increasing blood flow to the testicle and thus causing venous dilation (May et al. 2006; Kumanov et al. 2008). Increased penile length and circumference as well as rapid pubertal development have been found independent risk factors for the development of varicocele.

Effects of Varicocele and Pathologic Findings Related to Testicular Dysfunction

Varicoceles have been associated with (i) progressive and age-related testicular atrophy (Greenberg et al. 1978), (ii) alterations in semen composition and morphology producing infertility, and (iii) abnormal testosterone and follicle-stimulating hormone levels (Cayan et al. 1999).

The most well-documented abnormality associated with clinical varicocele is testicular hypotrophy. Although testicular size remains an easily observable clinical phenomenon in the presence of varicocele, its relationship to testicular dysfunction, as defined by abnormal semen analysis results, remains more difficult to quantify in adolescents.

In adults, the most common findings on semen analysis are decreased motility, decreased sperm density, and increased number of pathologic sperm forms (Paduch and Niedzielski 1996). Furthermore, a variety of histological changes related to testicular dysfunction have been documented in the results of testicular biopsy in males with varicocele, including Leydig cell hyperplasia, decreased number of spermatogonia per tubule, decreased spermatogenesis and maturation arrest, sloughing of germinal epithelium, and interstitial fibrosis (Castro-Magana et al. 1990; Turek and Lipshultz 1995). Because the adult testicle is composed mostly of seminiferous and germinal cells, it is not surprising to find a correlation between testicular volume and function as defined by semen analysis in this group.

Proposed Pathologic Mechanisms

In spite of the existence of a growing body of literature defining all the above mentioned associations, the exact mechanism whereby varicocele induces these pathologic changes has yet to be conclusively elucidated. In the meantime, debate continues on the pathologic effects of varicocele. Although many theories have been postulated on the subject, the harmful effects of varicocele on both fertility and testosterone production are likely caused by a combination of factors that include testicular hyperthermia, hormonal dysfunction, increased or decreased testicular blood flow, reflux of toxic metabolites, and/or seminiferous tubular hypoxia (Pryor and Howards 1987).

Hyperthermia. Elevated scrotal and testicular temperature is the most widely accepted mechanism for testicular dysfunction (Khera and Lipshultz 2008). The prevailing theory is that poor venous drainage leads to disruption of the countercurrent exchange of heat from the spermatic cord, which elevates scrotal temperatures. The elevated scrotal temperature leads to impaired spermatogenesis, affecting negatively to germ cell function, proliferation, and subsequent fertility (Amelar and Dubin 1987; Goldstein and Eid 1989). Increased scrotal temperatures have been shown to result in decreased testosterone synthesis by Leydig cells, injury to germinal thermolabile cell membranes, decreased protein biosynthesis, decreased amino acid transport, and altered Sertoli cell function and morphology (Fujisawa et al. 1988, 1989; Gorelick and Goldstein 1993; Mieusset and Bujan 1995; Simsek et al. 1998).

Hypoxia. Several recent publications have proposed a direct link between impaired testicular drainage and tissue hypoxia. These studies suggest that tissue damage is the result of impaired testicular microcirculation secondary to increased hydrostatic pressure rather than global hypoxia (Cayan et al. 1999; Gat et al. 2005; Lee et al. 2006).

Reflux of Renal and Adrenal Metabolites. Although the reflux of metabolites derived from the kidney and the adrenal gland has been proposed as a mechanism for testicular damage in men with varicocele, research conducted to date has been inconclusive, and experimental studies have shown that toxic effects of varicocele do not require adrenal contribution and that reflux even may not occur (Turner and Lopez 1989). Further research is required to determine if adrenomedullin plays a role in the formation of varicocele.

Oxidative Stress. Venous blood from varicoceles of infertile men has shown increased production of nitric oxide and its active metabolites that are known to play a role in sperm dysfunction. These increased levels have been shown to correlate with the severity of varicocele. Thus, Allamaneni and colleagues (2004) reported a

positive correlation between seminal "oxidant" levels and varicocele grade. They demonstrated that higher seminal oxidant levels are seen in men with grade 2 and 3 varicoceles compared with men with grade 1 varicoceles. Another recent study demonstrated that levels of oxidants are significantly higher in semen of infertile men than in semen of fertile men (Khera et al. 2007). Varicocele also reduces antioxidant defenses, potentially adding to the localized oxidative stress (Agarwal et al. 2009).

Clinical Presentation

Varicoceles are typically asymptomatic. Although in adult men this clinical condition can be associated with testicular pain and hypogonadism, the most relevant effects are represented by semen parameter alteration and male infertility. In a minority of patients, an aching pain is present that improves when the patient lies down.

Subclinical varicoceles are not palpable on physical examination but rather are diagnosed radiographically. Most authors agree that subclinical varicoceles are varicoceles less than 3 mm in diameter (Hoekstra and Witt 1995). There has been much debate as to the clinical relevance of subclinical varicoceles. Studies have shown that subclinical varicoceles have no significant impact on fertility and that repairing subclinical varicoceles has no significant impact on improving fertility rates (Jarow et al. 1996).

Idiopathic varicocele is more prominent in the upright position and disappears in the supine position. Secondary varicoceles, especially on the right side, can be caused by retroperitoneal tumors or lymphadenopathy and do not change size as noticeably as in the supine position. Although a left-sided varicocele is not a urological emergency, a right-sided varicocele in the absence of a left-sided varicocele should prompt an evaluation for a retroperitoneal process. If only the right side is involved, there exists a possibility that a retroperitoneal tumor is present and compressing the vein. Further imaging with computed tomography or ultrasound of the abdomen and pelvis is indicated for varicoceles that are of sudden onset, do not reduce in size with the patient supine, or occur on the right side.

Identifying Patients at Risk

Varicocele, if left untreated, will continue to affect testicular growth (with loss of volume and progressive deterioration in semen parameters) in a concrete subset of patients. In adults, treatment is straightforward and is proposed whenever (i) there is a palpable varicocele, (ii) there is documented infertility, (iii) it has been confirmed that there is no female infertility problem, and (iv) there is at least one abnormality found on semen analysis.

In the adolescents, significant controversy exists regarding the appropriate methods of evaluation for surgery. The diagnosis of varicocele leads to additional questions about possible infertility and the need to establish clinical criteria for varicocele repair. Currently, a variety of clinical tests are available for identifying adolescents at risk for infertility associated with varicocele. However, a consensus has yet to be reached on the most appropriate combination of tests in evaluation of adolescent varicocele. This topic remains at the forefront of debate and research. Currently, clinical tests proposed in evaluation for surgical intervention in adolescents with varicocele include (i) physical examination, (ii) radiologic evaluation, (iii) biochemical tests, and (iv) semen analysis.

Physical Examination. Physical examination includes identification of varicocele grade and the measurement of testicular volume. Patients should be examined in standing and supine positions. While standing, patients should be asked to perform a Valsalva maneuver so the physician can assess reversal of venous flow. The classic finding is a left-sided scrotal mass with a "bag of worms" consistency that increases in size with standing or Valsalva maneuver and decompresses when in supine. Classically, varicoceles are graded according to the following criteria (Trum et al. 1996):

- Grade 1 (small): palpable only with Valsalva maneuver
- Grade 2 (medium): palpable with the patient standing
- Grade 3 (large): visible through the scrotal skin and palpable with the patient standing

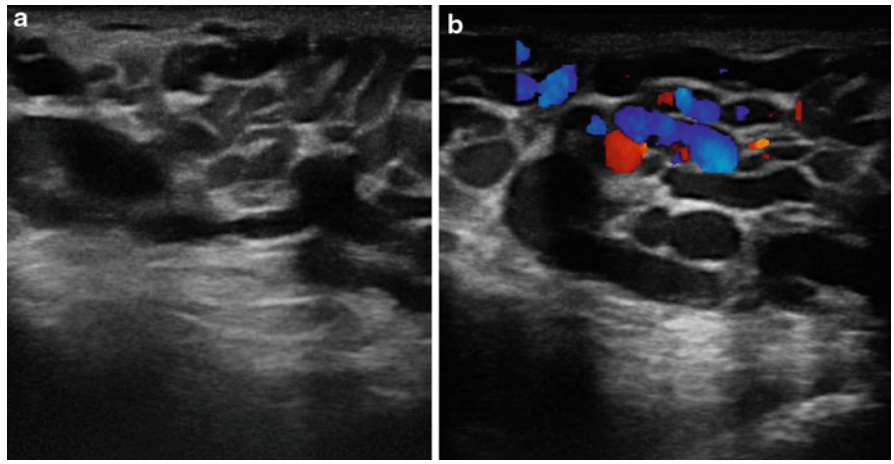

Fig. 8 Ultrasound exploration of left varicocele. No standard or clearly defined criteria for varicocele diagnosis by ultrasound exists to date. However, most clinicians agree that this type of diagnosis should be based on the demonstration of veins dilated between 2 and 3 mm (**a**) and reversal of venous flow with Valsalva maneuver (**b**)

An important part of the physical examination in all boys with varicocele is an accurate assessment of testicular consistency (firmness) and volume. Although the assessment of testicular consistency is subjective, a careful simultaneous comparison of both testes may give the clinician additional qualitative information about the overall condition of the ipsilateral testis. Several methods are available to measure the size of the testis, including visual comparison, calipers, Prader orchidometer (comparative ovoids), Takahira orchidometer (disc elliptical rings), and ultrasonography.

Radiologic Evaluation. If the diagnosis is in question, ultrasound is the test of choice, with a sensitivity of 97 % and specificity of 94 % (Trum et al. 1996). The duplex ultrasound has significantly improved our ability to diagnose varicoceles. Since its introduction, the use of other radiographic tools, such as venography, the Doppler stethoscope, and radionucleotide scans such as technetium 99 m-pyrophosphate, has greatly decreased.

Duplex ultrasound to diagnose a varicocele should be performed only when there is uncertainty on a physical examination, to corroborate or confirm results of the physical examination, or to identify recurrent or persistent varicoceles. Although there are currently no standard or clearly defined criteria for diagnosing a varicocele by ultrasound, most clinicians would agree that this type of diagnosis should be based on a patient having veins dilated between 2 and 3 mm and reversal of venous flow with Valsalva maneuver (Fig. 8).

From the 3-dimensional measurements on ultrasound, the relative testicular volumes may be also calculated and used to guide further treatment. Ultrasonography should be considered the standard criterion for assessing testicular volume. Results of ultrasonography have consistently shown high correlation with actual testis volume and have been highly reproducible, with improved detection of bilateral varicoceles and increased sensitivity in the evaluation of volume differentials as compared with orchidometer.

Biochemical Tests. Biochemical tests are based on the integrity of the testis and any effect of the varicocele on the hypothalamic-pituitary axis. At the testis level, serum inhibin levels reflect the integrity of the seminiferous tubules and the function of Sertoli cells.

Semen Analysis. Semen analysis in men with varicoceles reveals decreased motility, decreased sperm density, and more pathologic forms. By applying strict morphological criteria to semen analysis, varicocele repair improves the seminal parameters in approximately 70 % of patients,

with the improvement in motility being the most common. Studies suggest that the effect of varicocele on semen quality is similar in adults and adolescents (Pasqualotto 2007).

Semen analysis cannot adequately be performed until the subjects have progressed to the point in pubertal development necessary for adequate ejaculation, and sample procurement continues to raise several ethical questions. Nevertheless, it would seem from current trends in the literature that recommendations for the role of semen analysis in evaluation of adolescent varicocele are forthcoming.

Indications for Treatment

There is active controversy concerning the repair of varicoceles. Although varicoceles cause abnormal semen analysis and varicocelectomy can normalize these abnormalities (Segenreich et al. 1997; Beutner et al. 2007), there is no irrefutable evidence that this translates into higher rates of pregnancy (Evers and Collins 2003) possibly because of a lack of good randomized data, improper study protocols, and poor reporting of data. A Cochrane review (Evers and Collins 2008) recently reported that the evidence does not currently support varicocele repair as a reliable treatment for male infertility.

Conversely, the Male Infertility Best Practice Policy Committee of the American Urological Association recommended that a varicocele repair should be offered to the male partner of a couple attempting to conceive when all four of the following conditions are present (Practice Committee of the American Society for Reproductive Medicine 2004):

- The female partner has normal fertility or a potentially correctable cause of infertility.
- The couple has documented infertility.
- A varicocele is palpable or, if suspected, is corroborated by ultrasound.
- The male partner has one or more abnormal semen parameters or sperm function test results.

Adult men who have a varicocele and abnormal semen parameters and do not wish to conceive currently but might in the future could be considered for a varicocele repair.

Adolescent boys with varicoceles should be considered for a varicocele repair if there is testicular pain or a reduction in the ipsilateral testicular volume. If there is no identifiable reduction in ipsilateral testicular volume, these young men can be followed with annual physical examinations or a semen analysis. These patients and their families should be fully apprised of the concerns and controversies surrounding adolescents with a varicocele (Behre et al. 1989; Cayan et al. 2002b; Zampieri et al. 2008).

Treatment

The best method for treatment of adolescent varicocele has yet to be established. There have been no randomized, controlled, prospective clinical studies that compare the various techniques in adolescents or adults. Treatment options include open and laparoscopic surgical approaches for varicocele ligation and percutaneous transvenous embolization. In addition, several investigators have described innovative minimally invasive surgical approaches for the treatment of adolescent varicocele, including microsurgical approach, laparoscopic single-port surgery (Kaouk and Palmer 2008), and robotic-assisted techniques (Shu et al. 2008).

Open Surgical Approach. Open surgery remains the mainstay of varicocele treatment. Paduch and Skoog (2001) found that high retroperitoneal ligation of the testicular artery and veins (Palomo procedure) is the treatment of choice in adolescents, given that no testis atrophy after high ligation was observed and the classic Palomo repair was associated with a statistically significant decrease in surgical failure rate (Okuyama et al. 1988; Kass and Marcol 1992; Marsuda et al. 1993).

The scrotal approach was one of the first operations used for varicocele repairs. In 1904, Hartman was the first to describe radical resection of the scrotum and external clamping of varicoceles (Noske and Weidner 1999). The scrotal approach is no longer performed because of the increased risk of injury to the testicular artery and its high rate of recurrence. Currently, there are

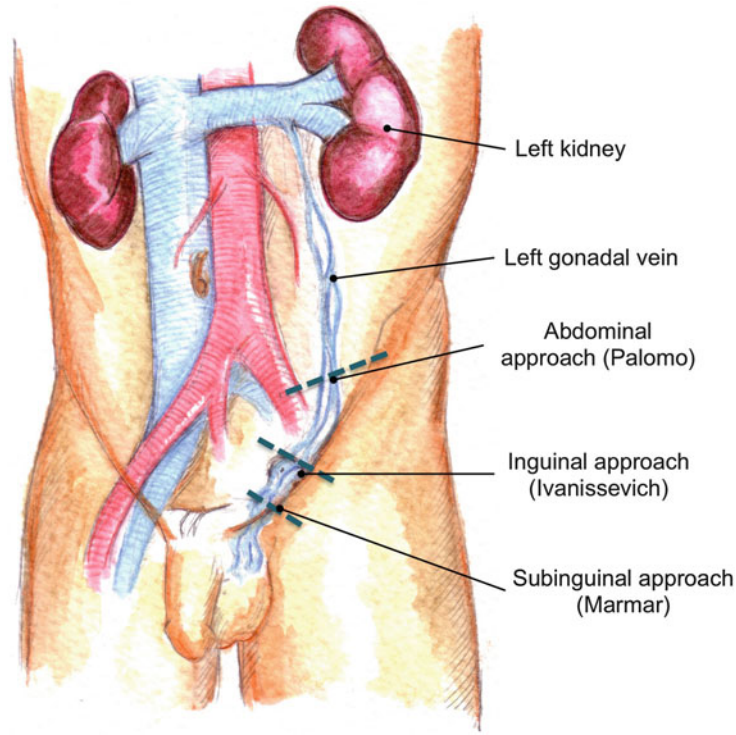

Fig. 9 Different approaches to open varicocele surgical repair

three main surgical approaches to a varicocele repair: inguinal, subinguinal, and retroperitoneal. The retroperitoneal approach can be performed either open or laparoscopically.

The open retroperitoneal approach (Palomo) involves a muscle-splitting approach. The peritoneum is retracted medially and the spermatic vessels are ligated lateral to the ureter. A laparoscopic retroperitoneal approach also has been described with artery-sparing and non-artery-sparing techniques. There has not been any increased efficacy with the laparoscopic approach over the open approach, and the laparoscopic approach is performed with decreased frequency (Jarow et al. 1993).

The inguinal approach (Ivanissevich technique) involves making an incision superior to the external inguinal ring and incising the external oblique fascia. A 3–4 cm "mini" inguinal incision can be made. Varicoceles in this region generally present with a typical vascular pattern in which the artery is next to, or adheres to, several veins. There is generally a separate isolated vein nearby. The testicular artery adheres to the undersurface of a large vein in approximately 50 % of cases (Beck et al. 1992).

The subinguinal approach does not require a fascial incision and theoretically offers less postoperative pain and faster recovery. The veins tend to branch at this level, however, and a higher number of smaller caliber veins need to be ligated (Fig. 9).

Hopps et al. (2003) demonstrated that internal spermatic arteries at the subinguinal level were more than three times as likely as those identified at the inguinal level to be surrounded by a network of adherent veins. The arteries at this level tend to be end arteries, and inadvertent injury to these arteries carries a higher rate of testicular injury (Chan et al. 2005; Al-Kandari et al. 2007).

Percutaneous Embolization. The first reports of percutaneous embolization of varicoceles occurred in 1978 (Lima et al. 1978). Since that time, advancements have been made in this technique with the use of coils, balloons, and sclerotherapy. Sclerotherapy has been particularly useful for occlusion of smaller collateral veins (Comhaire and Kunnen 1985). Percutaneous

embolization of varicoceles typically is not the initial treatment for varicoceles because of higher recurrence rates and failed procedure rates. Pryor and Howards (1987) found that the overall success rate, taking into account failed primary attempts and the recurrence rate, was 68 %. Therefore, percutaneous varicocele embolization should be reserved for recurrent or persistent varicoceles when the anatomy causing the varicocele needs to be radiographically defined. A less commonly used percutaneous approach to treating varicoceles is antegrade scrotal sclerotherapy. The success rate for this technique in the few published series varies between 87 % and 95 % (Fretz and Sandlow 2002; Beutner et al. 2007). However, the initial reflux grade and the number of collateral vessels of the spermatic vein are the most important factors for predicting success of this technique. The rate of complications with these technique is approximately 6 % (Fretz and Sandlow 2002; Beutner et al. 2007).

Advances in Surgical Repair of the Varicocele

The past several years have seen explosive growth in the literature about minimally invasive varicocelectomy including laparoscopic, robotic, and percutaneous techniques. This has enabled surgeons to offer multiple options to young patients to maximize surgical outcomes and minimize morbidity.

Arterial Sparing Surgery and Micro-Doppler Use. Two major advances in the surgical repair of the varicocele have been the surgical microscope and the intraoperative Doppler ultrasound. The main advantage of microsurgical repair over non-microsurgical repair is the significant reduction in postoperative complications, such as testicular artery injury, hydrocele formation, and varicocele recurrence.

The issue of preserving the testicular artery (i.e., microsurgical technique) has been questioned in adults, in whom inadvertent arterial ligation may lead to poorer postoperative sperm quality (Turek and Lipshultz 1995). Patients with artery-sparing procedures had semen analyses with higher sperm concentration, better motility, increased semen volume, and a higher rate of morphologically normal sperm than those with prior artery ligation (Zampieri et al. 2007).

In contrast, microscopic inguinal or subinguinal approach with arterial preservation should be considered as a viable option for adolescent varicocele treatment. The microsurgical low inguinal or subinguinal approach has been reported as the method with the highest success rate (99 %) and the lowest morbidity (0 % hydrocele). The main potential disadvantage of this approach is the need for an operating microscope to spare the arteries and lymphatics and the increased number of veins at this level.

The micro-Doppler is another advance that has improved the outcomes in varicocele repair. Hallak et al. (2005) found that microsurgical varicocelectomy combined with intraoperative Doppler ultrasound improved preservation of the testicular artery and increased the number of veins ligated. The Doppler is a valuable tool in helping reduce intraoperative complications and improving the number of testicular veins ligated, because by definitively excluding the artery, more veins can be taken without concern for arterial injury. Although no studies thus far have demonstrated the use of the Doppler causing a more significant improvement in semen parameters or overall outcomes, a recent study demonstrated that the number of veins ligated during varicocele repair does correlate positively with an increase in total sperm motility (Shindel et al. 2007).

Laparoscopic and Robotic-Assisted Varicocelectomy. Laparoscopic techniques represent appealing options in that the patient is spared the morbidity of a groin incision. The transabdominal intraperitoneal approach also offers the possibility of bilateral repair through the same small incisions.

There is only one report of a randomized controlled trial of laparoscopic and open repair that found that the rate of varicocele relapse was statistically similar (1.84 % vs. 1.35 %, respectively) between laparoscopic and open varicocelectomy but that the likelihood of wound complications and scrotal edema and the necessity of analgesia were all increased significantly in the open repair group. The laparoscopic group also had a decreased length of stay and shorter operative

time (Podkamenev et al. 2002). In terms of varicocele recurrence, Barroso et al. (2009) recently reported a comparison between open and laparoscopic varicocelectomy and noted that recurrence was seen in 2.9 % and 4.4 %, respectively.

One of the drawbacks to laparoscopic varicocelectomy is that the patient traditionally was required to have three incisions to facilitate a laparoscopic camera and two working instruments. Hence, Link et al. (2006) described a technique for two- trocar laparoscopic varicocelectomy. An additional evolutionary step forward was also reported by Kaouk and Palmer (2008) with their series of single-port laparoscopic varicocelectomy. With the advent of the robotic da Vinci Surgical System (Intuitive Surgical, Inc, Sunnyvale, CA, USA), the use of a robotic platform for minimally invasive surgery has expanded (Shu et al. 2008).

Complications

The most common complications after a varicocele repair are the formation of a hydrocele, varicocele recurrence, and testicular artery damage. The rates of these complications are highly contingent upon the surgical approach and a surgeon's skills. The complication rates after a varicocele repair have declined significantly since the introduction of the microscope and intraoperative Doppler.

The complication rates for hydrocele formation with non-microsurgical technique range from 3 % to 39 % (Szabo and Kessler 1984; Amelar 2003; Szabo and Kessler 1984), whereas hydrocele formation is rarely reported with a microsurgical technique (Goldstein et al. 1992). These improved results are thought to be caused by the greater ability to identify and preserve individual lymphatics. The recurrence rate for microscopic inguinal varicocelectomy has been reported between 1 % and 2 % compared with 9 % and 16 % for non-microscopic inguinal varicocele repair (Goldstein et al. 1992; Cayan et al. 2000; Marmar and Kim 1994). The recurrence rate for non-microscopic subinguinal varicocele repair is reported to be between 5 % and 20 % (Szabo and Kessler 1984; Marmar et al. 1985).

Outcomes and Beneficial Effects of Varicocele Repair

Our understanding of how a varicocele repair affects overall semen and hormone parameters has evolved over the past several decades. A varicocele repair also has been shown to improve serum follicle-stimulating hormone and testosterone levels (Su et al. 1995). In 1987, Kass and Belman (1987) were the first to demonstrate a significant increase in testicular volume after varicocele repair in adolescents. Finally, pregnancy rates after varicocele repair have been shown to increase with intrauterine insemination despite the absence of significant changes in gross semen analyses (Daitch et al. 2001). It is believed that improved functional factors not measured on routine semen analysis may explain these increased intrauterine insemination success rates.

Impact on Fertility. Simultaneously, the impact of varicocele repair on fertility is still one of the most controversial issues due to a lack of conclusive, well done randomized controlled trials (Ficarra et al. 2006). Nevertheless, correction of varicoceles has been shown to improve not only sperm motility, density, and morphology but also specific functional sperm defects. Improvements in the sperm penetration assay, oxidant determination, and DNA fragmentation have been achieved after a varicocele repair (Schatte et al. 1998; Mostafa et al. 2001; Zini et al. 2005; Ohl et al. 2007).

Impact on Semen Parameters. Abdel-Meguid et al. (2011) analyzed the changes from baseline in mean semen parameters in patients randomly allocated to receive microsurgical varicocelectomy or an observation protocol. Semen analyses were obtained at baseline and 3, 6, 9, and 12 months after the randomization. This study clearly showed the superiority of treatment group in comparison with the observational ones in terms of improvement in all semen parameters. Conversely, none of the semen parameters revealed significant changes from baseline in the control group (Abdel-Meguid et al. 2011). The favorable effects on semen parameters were also recently reconfirmed by two meta-analyses (Baazeem et al. 2011; Schauer et al. 2012). Both meta-analyses suggest that varicocelectomy leads to

significant improvements in sperm count and motility regardless of surgical technique.

Although many studies demonstrated an improvement in semen quality, the appropriate length of time required following varicocelectomy for semen quality evaluation is not well established. Al Bakri et al. (2012) concluded that the best semen quality recovery occurs after 3 months from surgery and then does not improve further thus supporting this follow-up period as a key to evaluate the effectiveness of varicocele repair (in terms of seminal changes) improving the decision-making process. Such a result was not affected by the different approaches used to repair the varicocele.

Impact on Pregnancy Rate. Concerning the impact of varicocelectomy on pregnancy rate of infertile couples, available meta-analyses continue to be influenced by previous heterogeneous and methodologically poor randomized clinical trials (RCTs). However, recent RCTs and non-randomized controlled studies showed significant advantages also in terms of pregnancy rate in patients who received varicocelectomy in comparison with observation. Different studies have shown a benefit of varicocelectomy on pregnancy rates (Abdel-Meguid et al. 2011; Baazeem et al. 2011; Mansour Ghanaie et al. 2012), while Giagulli and Carbone (2011) showed that the correction of varicocele aimed at restoring fertility could be more appropriate for men of couples with infertility longer than 2 years. Obviously, more data are needed.

Impact on Artificial Reproductive Techniques. The varicocelectomy does not seem to offer any significant advantages in terms of pregnancy rate in couples who underwent intracytoplasmic sperm injection (ICSI) (Pasqualotto et al. 2012). However, in a recent randomized study comparing couples in which male partners underwent varicocele repair versus couples who underwent expectant therapy, the authors showed a significant lower percentage of spontaneous first-trimester miscarriage (13.3 % vs. 69.2 %) in the first group (Mansour Ghanaie et al. 2012). However, it is possible that the improvement in semen parameters achieved after varicocele correction could offer a significant advantage also in terms of downstaging or shift of the level of artificial reproductive techniques needed to bypass male factor infertility (Cayan et al. 2002). More data on this important goal should be reported in the future.

Impact on Testosterone Levels. The effects on testosterone levels of varicocele and varicocele repair have, until recently, been equivocal. The results of earlier studies that did not find a statistically significant rise in testosterone following varicocele repair are difficult to interpret due mainly to methodological differences in the way testosterone levels were obtained (Lee et al. 2007; Di Bisceglie et al. 2007; Zheng et al. 2009; Rodriguez-Peña et al. 2009). Although the majority showed a postvaricocelectomy rise in testosterone compared to baseline, the change did not commonly reach statistical significance, probably secondary to short patient samples and the very wide range of testosterone values recorded after varicocelectomy.

Several recent studies (Sathya Srini and Belur Veerachari 2011; Zohdy et al. 2011; Hsiao et al. 2011) have added high-quality data to an existing base of research that, when combined, produce a consistent body of evidence for both the harmful effects of varicocele on testosterone production and the restoration of normal testosterone levels following varicocele repair in men with below normal testosterone levels. These studies strengthen considerably the evidence base about the positive relationship between varicocele repair and testosterone levels.

Predictive Markers

There has been much interest in identifying predictive markers to assess which male patients would most benefit from varicocele repair. Marks et al. (1986) reported four preoperative factors associated with an increased likelihood of postoperative pregnancies: (i) a lack of testicular atrophy, (ii) sperm density more than 50 million per ejaculate, (iii) sperm motility of 60 % or more, and (iv) and serum follicle-stimulating hormone values less than 300 ng/mL. Kamal and colleagues (2001) found that men with more than five million sperm per milliliter had a spontaneous pregnancy rate of 61 % after varicocele repair

compared with an 8 % spontaneous pregnancy rate in men with less than five million sperm per milliliter. Although previous reports have suggested that the gonadotropin-releasing hormone stimulation test may be useful in predicting clinical outcomes after microsurgical varicocelectomy, recent studies have found this not to be the case (O'Brien et al. 2004). The gonadotropin-releasing hormone stimulation test is not commonly used in clinical practice.

There is also a great need for further research to improve selection of patients who may most benefit from surgical correction of varicocele. Advanced molecular biology techniques used for the evaluation of the infertile men have increased the understanding of the physiology of spermatogenesis.

It is estimated that 13 % of men with azoospermia carry microdeletions of the long arm of chromosome Y (Nakahori et al. 1996; Pryor et al. 1997; Seifer et al. 1999). Men with varicocele and azoospermia or severe oligoasthenospermia may suffer from point mutation or deletion of genes important in spermatogenesis. Y-chromosome microdeletion analysis is not universally available, and the assay technique has not been standardized fully. In the near future, by screening for aberrations of genes involved in regulation of spermatogenesis, better criteria for management of patients with varicocele may be established (Paduch and Skoog 2001; Sigman and Jarrow 2007).

Intratesticular Varicocele

Intratesticular varicoceles represent a rare entity (<2 % in a symptomatic population) (Das et al. 1999), which can be associated with extratesticular varicoceles. In the same manner as extratesticular varicoceles, they are more often seen on the left side. However, bilateral intratesticular varicoceles have been described (Das et al. 1999). Intratesticular varicoceles will behave clinically in a similar fashion to an extratesticular varicocele. Commonly, they appear with testicular pain attributed to stretching of the tunica albuginea secondary to venous congestion (Browne et al. 2005) and increase in size with retrograde flow on Valsalva maneuver.

Diagnosis relies on ultrasound findings. The ultrasound appearances of an intratesticular varicocele are anechoic serpiginous or cystic structures radiating from the mediastinum testis, though involvement of the subcapsular veins has been described (Atasoy and Fitoz 2001). Color and spectral Doppler examination is characteristic and allows differentiation from other cystic structures such as prominent rete testis or an intratesticular cyst.

The clinical significance and associated incidence of infertility is yet to be established (Das et al. 1999). Treatment includes sclerotherapy, embolization, or spermatic vein ligation (Morvay and Nagy 1998; Demirbas et al. 2001).

Venous Infarction

Venous infarction of the testis is an extremely rare entity that may occur in association with severe epididymo-orchitis. In these patients, localized edema occludes the venous drainage of different portions of the testis or the entire testis, and thus producing segmental or complete testicular infarction (Bird and Rosenfield 1984; Rencken et al. 1990). Ultrasound shows a segmental or complete low reflectivity and an absence of color Doppler flow (Eisner et al. 1991). Indirect evidence of venous infarction is suggested by reversal of testicular arterial flow in the spermatic cord (Sanders et al. 1994).

Other Vascular Diseases of the Testis

Testicular Amyloidosis

Amyloid deposition may occur in nearly every location of the genitourinary tract (Falk et al. 1997). Deposits of amyloid in the testis may occur as a primary disease. However, testicular amyloidosis is much more frequently secondary to well-defined pathologic conditions, such as chronic granulomatous diseases, Crohn's disease,

tuberculosis, rheumatoid arthritis, pyogenic diseases, and neoplasias (Ishi et al. 1983).

Testicular amyloidosis is characterized by the extracellular deposition of eosinophilic, amorphous material (i.e., protein) in a diffuse manner in the walls of blood vessels and in the lamina propria of the seminiferous tubules. Microscopically, it shows commonly a typical fibrillar pattern (Tripathi and Desautels 1969).

Amyloid deposits can lead to solid organ dysfunction secondary to infiltration and disruption of the normal tissue architecture with enlargement of the affected testis. However, patients may present also with bilateral testicular enlargement or even with normal testicular volume. Although in most patients, the seminiferous epithelium shows complete spermatogenesis, many tubules may present with hypospermatogenesis (Nistal et al. 1989). Likewise, primary testicular amyloidosis has been described as a rare cause of infertility (Handelsmann et al. 1983; Bonacina et al. 1992; Schrepfermann et al. 2000; Haimov-Kochman et al. 2001). In these patients, azoospermia is caused by the complete replacement of testicular parenchyma with amyloid.

The differential diagnosis should include testicular infarction (torsion) if the entire testis is affected and a burned-out germ cell tumor if amyloid deposition changes are circumscribed to one region. Staining with Congo red and electron microscopic study are critical to confirm the diagnosis of amyloidosis. Electron microscopy is a well-recognized method and considered the reference standard for the diagnosis of amyloidosis (Nistal et al. 1989).

Testicular Vasculitis

Isolated testicular vasculitis is rare (Warfield et al. 1994; Joudi et al. 2004; McGuire et al. 2006; Atis et al. 2010). It is usually found in young people and still remains unclear whether such cases represent a truly isolated vasculitis or solely an unusual primary presentation site. Therefore, the most common appearance of testicular vasculitis is as part of a multiorgan or systemic disease. Involvement of the testicles is seen less frequently in Wegener's granulomatosis, Henoch-Schönlein purpura, giant cell arteritis, and rheumatoid arthritis, whereas testicle involvement is commonly associated with polyarteritis nodosa (Belville et al. 1982; Lee et al. 1983; Huisman et al. 1990). The results of postmortem studies suggest that the testis is involved in 38–86 % of cases of polyarteritis nodosa. At the same time, less than 18 % of these cases are symptomatic, and most will show other manifestations of polyarteritis nodosa (Shurbaji and Epstein 1988).

The pathogenesis of isolated testicular vasculitis remains unknown. Many questions regarding its focal presentation or the risk of subsequent progression remain also to be determined. Likewise, it is not known whether this unique clinical picture has a better prognosis than does systemic disease (Gordon et al. 1993).

The patient commonly presents with symptoms and signs in favor of inflammation. Therefore, other conditions presenting as pain in the testicle including testicular neoplasm have to be included in the differential diagnosis. Testicular torsion and segmental testicular infarction may be excluded by means of Doppler ultrasound. However, ultrasound examination may fail to show any abnormality but can also demonstrate the existence of a hypoechogenic mass (Shurbaji and Epstein 1988; Warfield et al. 1994; McGuire et al. 2006). In the majority of reported cases, clinical or laboratory evidence of disease in other organ systems on presentation was present or developed subsequently within a short time period (Lee et al. 1983). Serological markers (i.e., C-reactive protein and von Willebrand factor) are possible indicators of endothelial injury in systemic vasculitis but may not reflect the activity in isolated organ disease. Magnetic resonance imaging (MRI) is a more sensitive technique that can demonstrate focal testicular infarction, but, at present, the only "diagnostic tool" for vasculitis is histological confirmation.

Testicular necrotizing vasculitis is impossible to diagnose without tissue analysis. The microscopically observed changes are almost identical in all vasculitis seen in other systemic disorders.

After orchiectomy, histopathological findings are used to investigate the existence of necrotizing vasculitis. Histopathological characteristics observed in necrotizing vasculitis are mainly restricted to blood vessels. Fibrinoid necrosis is the morphological hallmark of the disease. The walls of small- and medium-sized testicular arteries are affected. Notably, hemorrhagic necrosis occurs in other pathologic conditions, such as testicular torsion, infarction, and inflammation.

Systemic treatment with potentially toxic immunosuppressive therapy with the added risk of sterility, despite the lack of clinical and objective laboratory evidence of systemic disease, presents a difficult clinical dilemma. In view of the high relapse rate associated with polyarteritis nodosa, long-term follow-up for these patients is essential. However, the absence of serological markers of disease activity makes monitoring of any future relapse quite difficult.

Hemangioma of the Testis

Hemangioma is one of the most common soft tissue tumors and the most common tumor in infancy and childhood (Rosenthal 1946; Hidalgo et al. 1995). However, its isolated occurrence in the testis is rare. The first reported case of testis hemangioma was by Morehead and Thomas in 1944 (Morehead and Thomas 1944). Since then, most subtypes have been reported and have been described as cavernous (Kleiman 1944; Morehead and Thomas 1944; Lozano et al. 1994; Sendra-Torres et al. 1995), capillary (Kuraoka et al. 1994; Hidalgo et al. 1995), histiocytoid (Banks and Mills 1990), and juvenile (Uchida et al. 1997), with cavernous hemangioma being the most common.

Their clinical presentation may vary from testicular enlargement to acute testicular infarction. Typically, no serum elevations of beta-human chorionic gonadotropin and alpha-fetoprotein are detected. The ultrasound study presents a vascularized hypoechoic mass similar to germ cell tumors (Lozano et al. 1994).

Hence, the most common preoperative diagnosis of a solid mass in the testis would be a germ cell tumor or other testicular malignancy because they are more prevalent in this population (Lozano et al. 1994). Therefore, this neoplasm, although rare, should be considered in the differential diagnosis of a testicular mass, given that preoperatively distinguishing this tumor from the more common testis tumors (i.e., germ cell, sex cord, stromal) is not feasible at present.

Intratesticular Arteriovenous Malformation

Arteriovenous malformations are commonly described inside the scrotum. However, to our knowledge there are only two reports of intratesticular arteriovenous malformations (itAVM) in the current literature (Strom and Franciosis 1976; Kutlu et al. 2003). The ultrasound appearance is that of multiple tubular anechoic structures that display vascular flow, readily differentiated from an intratesticular varicocele by the lack of retrograde blood flow on the Valsalva maneuver (Kutlu et al. 2003; Browne et al. 2005). Differential diagnosis includes intratesticular hemangioma and testicular malignancy (Ricci et al. 2000). While the presence of calcification between the tubular anechoic structures is highly suggestive of a hemangioma, the presence of a mass with a disorganized pattern is commonly seen in a primary testicular malignancy (Stewart et al. 2004).

Conclusions

Vascular pathology of the vessels within the scrotal sack is not unfrequent. While testicular torsion represents a true surgical emergency, it is still controversial if varicocele has to be treated to improve patient fertility. The consequences derived from unconscious management can be devastating in both cases, given that testicular atrophy may follow an unresolved spermatic cord twist or a costly overtreatment may be inflicted in cases of indolent varicocele. An adequate knowledge of these conditions is essential for an optimum decision-making process.

The above chapter has reviewed in depth these and other scrotal vascular conditions to provide the treating physician a wide-scope perspective to deal with them.

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