Chapter 22
Acute Scrotum

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Abbreviations

AS	Acute scrotum
TT	Testis torsion
ATT	Appendix testis torsion
OE	Orchiepididymitis
DUS	Doppler ultrasound
US	Ultrasound

The expression "acute scrotum (AS)" describes a cluster of different pathologies presenting scrotal pain. The causes vary in incidence according to age (testis torsion—TT—predominate in neonates and adolescents and appendix testis torsion—ATT—in school-aged children). Depending on age distribution AS cohorts may show a predominance of orchiepididymitis (OE) (adult cohorts), ATT (predominantly pre-pubertal pediatric cohorts) or TT (cohorts showing predominance of adolescents and young adults) [1]. In children and adolescents, "surgical" causes largely predominate and OE account only for circa 1/5 of the cases.

Despite not being the most common, TT is the most morbid condition related to AS, and is a surgical emergency: postponing treatment for more than 6 h after the beginning of the pain episode risks testicular infarction and loss of the gonad. AS is an active area of litigation concerning Pediatric Surgery and Urology. AS cases are the third most common diagnosis in claims involving adolescents in the United States and the 12th most common cause linked to successful litigation claims against NHS/United Kingdom [2].

The main causes of AS are:

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1. Testicle torsion (TT)
2. Appendix testis torsion (ATT)
3. Orchiepididimytis (OE)

However, the list of rarer possible causes of AS is extensive:

1. Testicular trauma and/or hematocele
2. Vasculitides affecting the testis and/or the scrotum (Henoch-Schonlein purpura, IgA vasculitis, Kawasaki disease)
3. Pyocele/scrotal abscess/scrotal infections including necrotizing fasciitis (Fournier's gangrene)
4. Idiopathic scrotal edema
5. Inguinal hernia strangulation complicated with testicular ischemia
6. Tense hydroceles/acute hydroceles
7. Adrenal hemorrhage (in neonates)
8. Meconial peritonitis (in neonates)
9. Renal vein thrombosis (mainly in neonates)

The objective of this chapter is to describe the main causes of AE, emphasizing the most relevant and frequent conditions.

1. Testicle torsion

A very clear description of an episode of testicular torsion has been published by Ombrédanne as soon as 1927 [3], but the anatomical cause of the disease, which usually affects both gonads, was only evidenced in the 1960s by Richards Lyon [4]. This finding changed the paradigm of the disease, leading to the adoption of BILATERAL orchidopexy as the state of the art for treatment. A 40% probability of asynchronous contralateral torsion is assumed in patients that are not submitted to contralateral orchidopexy after a TT episode.

The need to educate physicians and the population about the urgency to evaluate any patient presenting acute scrotal pain was soon recognized. Legal problems leading to the prosecution of health personnel accused of misdiagnosis, diagnostic and treatment delay, causing gonadal loss, imposed aggressive surgical protocols, and refinement of diagnosis.

A normal testis is fixed to the posterior scrotal wall, while the vaginal layer surrounds the rest of the gonad's surface. The usual cause of a TT is the intravaginal rotation of a testicle that is not posteriorly fixed to the scrotal wall and remains free into the scrotum, suspended exclusively by the spermatic cord (bell clapper malformation, which usually affects both sides) (Fig. 22.1). The single presence of the bell clapper malformation does not determine the occurrence of TT: an autopsy study involving men without a TT history detected a 12% incidence of bell clapper malformation [2]. The connection of the free testis to the cremaster fibers that form a muscular layer over the spermatic cord also play a part in the pathophysiology of the condition, as a sudden, forceful, and/or asynchronous contraction of this muscle may induce twisting of the gonad, as the cremaster muscular fibers derive from

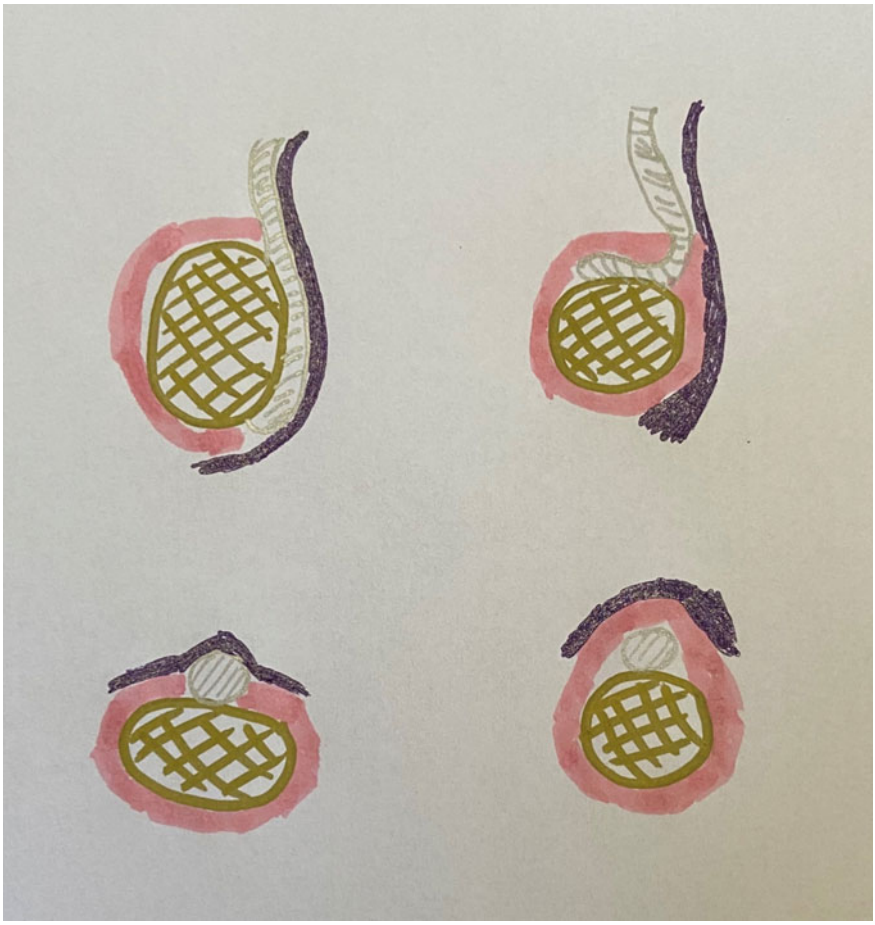

Fig. 22.1 Normal testicle: upper left (sagittal cut), lower left (transversal cut). Bell-clapper deformity: upper right (sagittal cut), lower right (transversal cut). Legend: testicle (gold), epididymis/vas deferens (silver), vaginal layer (pink), scrotal skin (purple) (drawing by the author)

various layers of the abdominal wall muscles and distribute along the cord in a spiral pattern.

Nocturnal erections, protective reflexes and a cold environment may induce a forceful cremasteric contraction, which may explain the higher incidence of TT in winter [5, 6].

The absence of testicular posterior fixation gives rise to one of the most prominent clinical signs that raise the suspicion of testicular malfixation (bell clapper deformity): the longest axis of the affected testicles assumes a horizontal position in the scrotum in a standing patient, especially in post-pubertal males, after the testicle grows to adult proportions.

Absent fixation also explains the torsion of cryptic testes (that twist over an axis formed by the cord and the gubernaculum testis) and extravaginal torsion in the fetus/neonate, as the normal fixation between the vaginal layer and the scrotal wall is usually complete only after 10 days of life.

A torsion between the testicle and the epididymis is extremely rare and depends on a lax testicular mesentery.

The TT determines a sequence of venous and arterial obstruction, also depending on the presence of a "complete" (360°) twist, the number of twists and "tightness" of turns of the cord. The affected gonad suffers progressive ischemia, leading to the sequential destruction of germinal epithelium, Leydig cells, and, finally, testicular necrosis and atrophy. The irreversibility of the ischemic process depends on the duration of the episode: reversibility is highly probable in a 6 h interval, but chances worsen progressively after this time period. Testicular necrosis is almost always the result after 24 h.

TT affects approximately 1:4000 males, with an annual incidence of 0.004% for children under 18 years-old [7] or 4.5/100000 < 35 years-old men [2]. The incidence of the disease is bimodal, affecting preferentially neonates (intrauterine extravaginal torsion due to immaturity, affecting normally descended testes) and post-pubertal adolescents (intravaginal torsion due to bell clapper deformity).

The main characteristic of the disease (except for neonates—see later) is a sudden and very intense scrotal pain episode. Pain may irradiate to the ipsilateral tight, lumbar, or inguinal region. Abdominal pain is also possible, mainly in pre-pubertal males, and frequently causing diagnostic delay, especially if the physician does not include a systematic genital examination in his/her routine physical examination for acute abdominal pain.

Most episodes are nocturnal and may wake the patient up. Spontaneous erections ("wet dreams"), sexual activity, and trauma may also induce TT by a forceful cremasteric contraction.

Some patients (6–63%) describe previous ipsi or contralateral episodes of scrotal pain with spontaneous resolution [8], suggesting that in carriers of bell clapper deformity episodes of torsion with spontaneous resolution may occur. The diagnosis of those patients before an episode of persisting TT is problematic, as there are no image exams that are able to diagnose the presence of the malformation [8]. The only valuable information to confirm this suspicion is the detection of horizontal lying highly mobile testes in those patients, when standing. The sum of a trustable history of repetitive episodes of testicular pain with spontaneous resolution and horizontal-lying testes authorizes the indication of orchidopexies as prophylaxis of TT.

Nausea and reflex vomit may associate. Fever or urinary symptoms are not to be expected. A history of contralateral urgent orchidopexy or the presence of contralateral atrophy after an untreated pain episode enhances the suspicion of TT. The finding of an atrophic testis associated with a previous undiagnosed AS episode entails the indication of contralateral orchidopexy as soon as possible.

The physical examination should be repeated in supine and standing position. Both gonads and testicular cords should be examined in detail. A twisted testis is

usually edematous, hardened, extremely painful and positioned high in the scrotum (Brunzel's sign, "*testis redux*") (Fig. 22.2). The affected gonad and/or the contralateral testicle exhibit a horizontal position, most notable in the orthostatic position and in post-pubertal adolescents (Angell's sign). The examiner may palpate a "naked", free and mobile lower testicular pole, due to the lack of gonadal fixation, in early cases. In patients that are examined very early after the establishment of the torsion, a umbilication presenting in the caudal aspect of the scrotum may be seen (Ger's sign), but this is very uncommon in practice. Palpation of the epididymis out of its usual posterior position is helpful, but not definitive, as the epididymis may be posterior after a 360° rotation. As the disease progresses, local inflammatory changes follow, with scrotal edema and secondary hydrocele. In late cases, after definitive gonadal necrosis and before late atrophy, pain resolves, and the patient may present as a "testicular tumor" (pseudotumoral presentation). The description of a previous pain episode and scrotal ultrasound are the key to the differential diagnosis between late TT and testicular tumors. This is very important because a correct diagnosis allows to protect the contralateral still unaffected gonad from a future torsion episode and spares the patient an oncological approach to a pseudotumoral ischemic testicle.

The absence of the cremasteric reflex is typical of TT, and is valuable for the differential diagnosis of AS, as it is usually present in cases of ATT and in most cases of OE. The absence of cremasteric reflex in a patient presenting AS points to TT as the most probable diagnosis (OR 47.6) [9, 10]. Also, in TT cases the elevation of the affected testicle does not bring relief, which usually happens in OE cases (Prehn's sign).

Some authors have proposed the usage of clinical scores to estimate the probability of TT as the cause of AS. One of those, TWIST (testicular work-up for ischemia and suspected torsion) proposes to consider the presence of testicular edema (2 points), hardness (2 points), vomits/nausea (1 point), high-lying testis (1 point) in patients presenting acute scrotal pain. Patients summing more than 5 points are considered high risk and emergency surgery is indicated (without any complementary exams). The score proved to be highly specific, and the positive predictive value for TT was 93.5% [11, 12].

The difficulties involving the availability of expert sonologists, leading to delays in treatment while waiting for diagnostic confirmation suggest that real-time ultrasound performed by the emergencist (point-of-care ultrasound) should be tested as a supportive approach to the differential diagnosis of TT. This has been tested recently in a 120 AS cohort (12 TT). The correct final diagnosis could be reached in 70% of cases. All TT cases were identified, with one false positive (an ATT case, with 100% negative predictive value and 92.3% positive predictive value), saving an hour between admission and surgery [13].

Considering that the patients should ideally be operated in a maximal interval of 6 h since the beginning of symptoms and that the surgery used to treat TT is low risk in healthy patients, with low morbidity and rare complications, it is accepted that complementary exams can and should be waived if not immediately available. In other words, in cases of high level of suspicion of TT, the physician is authorized

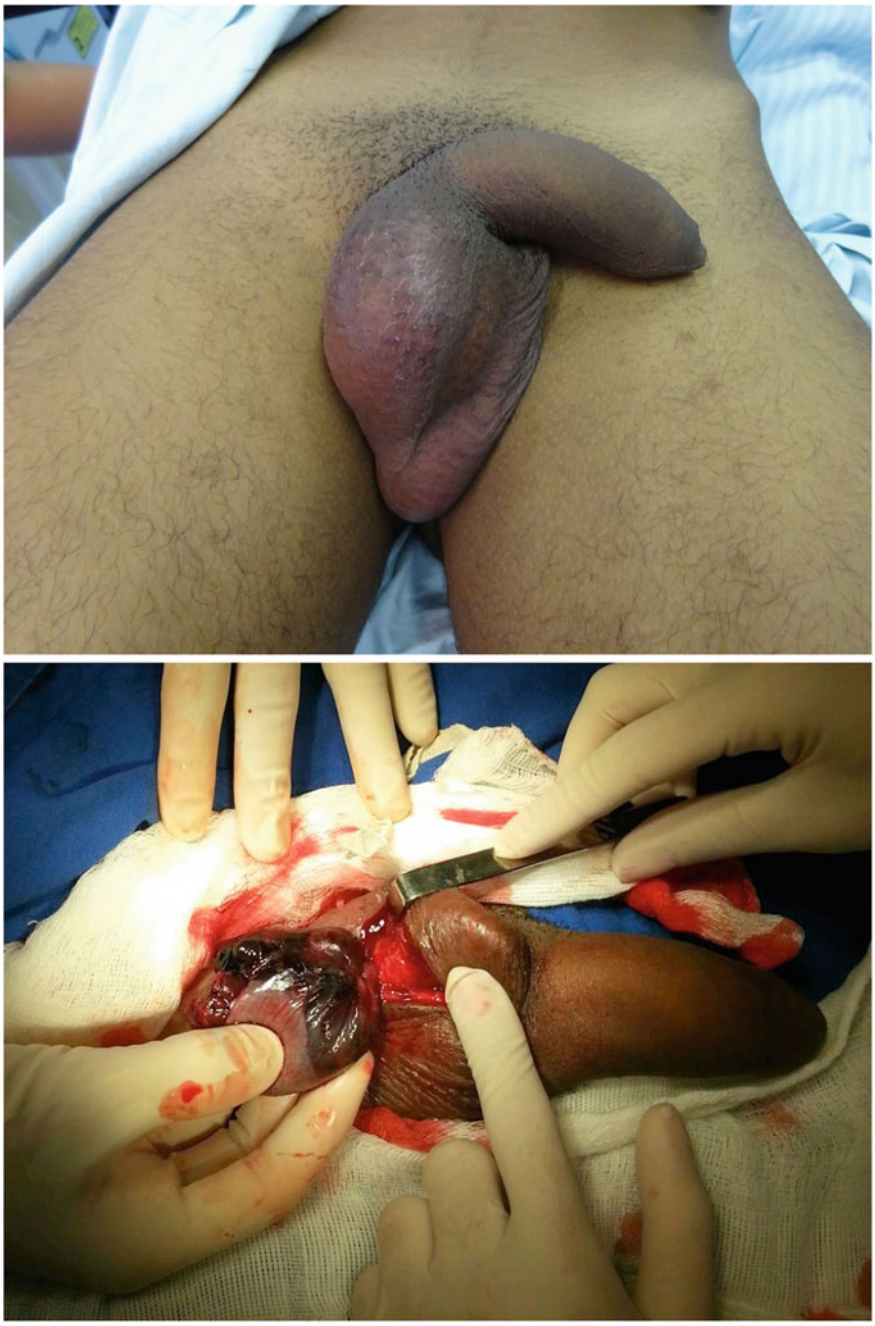

Fig. 22.2 Testicular torsion. Upper picture: elevated and augmented testis. Lower picture: Aspect of the testis and cord rotation in surgery (from the author's archive)

to operate despite not proving his/her hypothesis with complementary exams, especially if the duration of the episode is close to complete the "safe" period of 6 h. As a matter of fact, to NOT operate emergently on a suspect of a TT in due time may be considered malpractice. The delay time to get a doppler ultrasound after emergency evaluation has been recently estimated as approximately 3.5 h in a Canadian paper (surgery was done after a mean 5.1 h after being admitted to the emergency department) [10].

Doppler ultrasound (DUS) is the most used (and most useful) exam to confirm TT. The ultrasonographic aspect of the affected testis varies depending on the interval since the establishment of ischemia. In the first 6 h, the organ parenchyma is normal. From 6 to 24 h, the organ is usually hypoechogenic and increased in volume. After 24 h the testicular parenchyma is heterogeneous and secondary hydroceles are common. Parenchymal heterogeneity has been related to necrosis [10]. Very late presentations (pseudo tumoral) show heterogeneous augmented testes with calcifications. A post-ischemia atrophic testicle is typically hypoechoic and smaller, associated to a hyperechoic epididymis.

The epididymis is also swollen and heterogeneous, hyperechogenic with radiating hypoechoic bands, eventually difficult to be separated from cord structures. The ultrasound technician should also examine the spermatic cord, from the inguinal region down: during a TT episode the spiraled cord corresponding to the region of the twist ("whirlpool sign") or a heterogeneous "pseudo tumor" representing the edematous distal cord ("boggy pseudo mass") can be directly detected [2].

A doppler evaluation is fundamental for the diagnosis of the testicular ischemia caused by absent arterial irrigation of the testis (Fig. 22.3). The interpretation of the exam is more difficult in small testicles. In protracted cases the secondary inflammatory reaction is characterized by peri-testicular hypervascularization, which can be confounded with a normal testicular flow. The sensitivity of US/doppler for the diagnosis of TT varies from 82 to 100% and specificity may reach 98.8% [7]. Positive predictive value has been proposed as very high, but some authors suggest a lower negative predictive value [14], possibly associated to false negative diagnoses attributable to peri-testicular augmented flow.

Testicular scintigraphy was used to detect the absence of vascularization of the affected testis: the diseased organ does not capture the intravenous radioisotope. A peri-testicular "halo" formed by augmented captation of the isotope in inflamed tissues is typically seen, while the normal testis shows similar captation to the neighboring tissues. The exam is rarely available in due time, exposes the patient to radiation, is difficult to interpret in small testicles, and is rarely used to confirm TT diagnosis contemporaneously.

Magnetic resonance imaging (MRI) with intravenous contrast can diagnose TT, using the same theoretical basis of scintigraphy (the absence of captation of intravenous contrast by the ischemic testis) [15]. It has recently been suggested that MRI can indirectly show a bell-clapper malformation, in the presence of hydrocele (demonstrating the absence of posterior fixation and a free-lying testis) [16]. MRI, however, is costly and not readily available in most institutions.

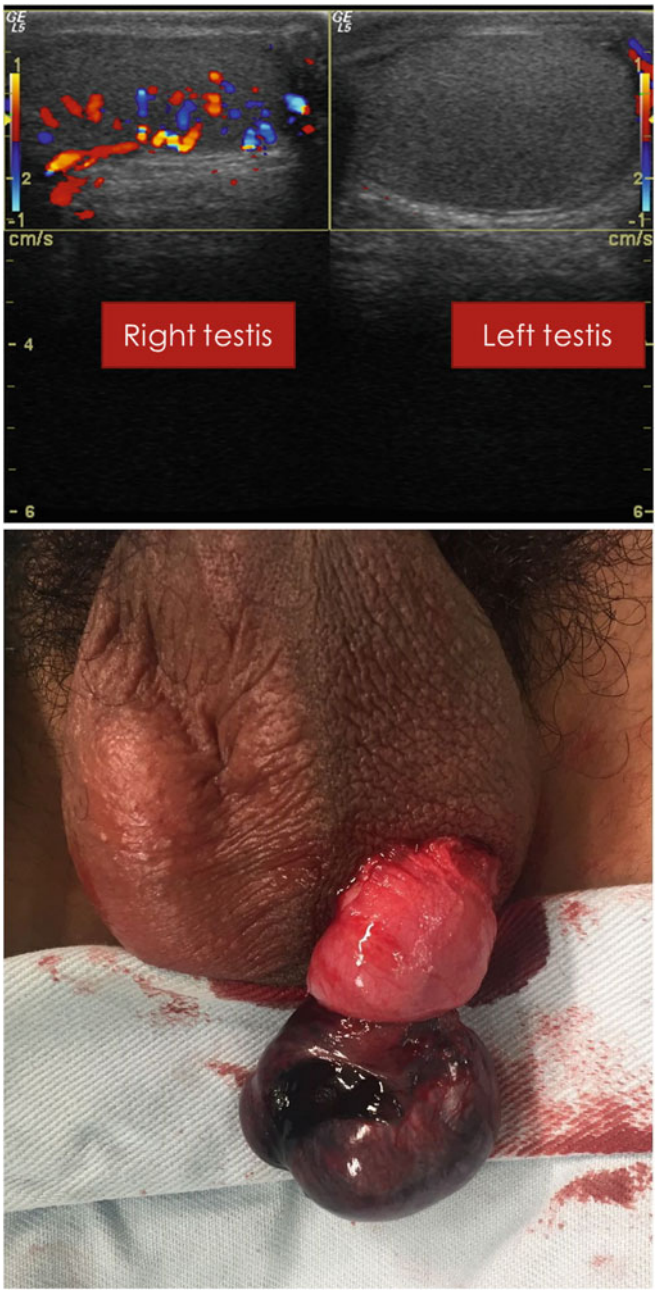

Fig. 22.3 TT affecting the left side. Normal vascularization at the right side, absent vascularization at the left side (upper picture). Aspect of the testis in surgery (lower picture) (case from Dra. Tatiana Fazecas, MD)

Preliminary studies have examined the possible relevance of transcutaneous near-infrared spectroscopy to detect lower oxygen saturation levels in the torsed testis. The method seems promising both in animal research and in a pilot study in humans [2]. Other exams may be used for differential diagnosis, such as ultrasound of kidneys/bladder, urinary tests, and dosing acute inflammatory markers to exclude urinary tract infections associated with OE, urinary tract malformations and vasculitides [17].

The treatment of TT is an EMERGENCY (except for very protracted cases), surgical (usually as an emergency surgery, demanding ipsilateral resolution of the case and contralateral orchidopexy as soon as possible). Even contemporaneously, the literature shows variable proportions for gonad salvation, usually half to two-thirds of the affected gonads, and late atrophy is common [18]. The prognosis for pre-pubertal patients is worse. Some authors described that the loss of the gonad approximately doubles for pre-pubertal children, possibly due to atypical presentations (mainly abdominal pain) and/or low levels of suspicion [19]. Those numbers depend deeply on the timeliness of the patients' presentation to medical evaluation and on their timely transfer to get surgery. Recent papers showed that a relatively small proportion of the patients present to a physician before 6 h after the emergence of pain and parents complain that they had never been educated about the possibility of a TT and its consequences [20]. Also, diagnostic errors made by non-experts were common, especially misdiagnosing OE, leading to serious extension of the episode before treatment by surgeons/urologists (mean delay time to the right diagnosis 84.4 h) [21]. The education of the patients, parents and non-expert physicians, especially those involved in primary care and non-referral emergency units, including specifically designed campaigns, may help to get early diagnosis and surgery, resulting in better salvation numbers.

Non-surgical detorsion of the cord is a classical maneuver described in 1893 [22], and has been revisited recently, associated to simultaneous doppler-ultrasound to attest to the resolution of the ischemic process.

The rotation of a twisted testes is usually "towards the inside" (clockwise in the right, anticlockwise in the left), so de-rotation maneuvers are "towards the outside" (in the direction of the thigh, from medial to lateral, "open-book" way). The twisted testis may show more than one rotation and some testis twist in the opposite direction (circa 1/3 of the patients) [7, 23], so that the physician must be sure to have fully undone the cord's twists. The expression of a successful maneuver is the immediate resolution of pain and the immediate restoration of flow by doppler ultrasound.

De-rotation maneuvers have been criticized due to the need of doppler/US to secure the resolution of ischemia and to the persistent need to operate to avoid other episodes and to fixate the other testis. Modern authors, however, argue that de-rotation buys time. De-rotation maneuvers may be extremely useful if there is no prompt availability of surgery/anesthesia and in patients presenting prohibitive risks for emergency surgery. De-rotation is most effective and efficient in early cases, before the establishment of edema and secondary hydrocele. Subsequent urgent bilateral fixation is needed as soon as possible. Detorsion auxiliary maneuvers

under DUS control have been described recently as effective in 75% of the cases attempted, with higher effectivity in early cases and potential to increase gonadal salvation [24–26].

Surgical treatment of TT after the neonatal period involves trans-scrotal incisions. Most authors prefer a raphe longitudinal incision to access both testes, but some opt for bilateral incisions to get bilateral orchidopexies (in the bottom of the scrotum or using Bianchi's upper-lateral incision). The affected testis is exposed, de-rotated, and observed for 5–10 min in a warm environment. If the organ recovers a normal aspect, fixation follows. In cases of undoubtful necrosis, orchiectomy is usually done. In doubtful cases, the modern approach is to make a long longitudinal incision of the albuginea layer, in order to relieve intratesticular pressure (compartment syndrome) and to cover this incision with a vaginal patch, before fixation. This approach has proved to save some gonads after protracted ischemia [27–29].

As previous worries about possible contralateral damage by immunological mechanisms when conserving a borderline viable testicle has not been proved, the contemporaneous tendency is to be conservative, but black/hemorrhagic testicles more than 5 min after de-torsion (suggesting hemorrhagic necrosis), heterogeneous parenchyma on pre-operative ultrasound, absence of bleeding after incising the albuginea, and episodes with >12 h duration are related to late atrophy [30].

There are a lot of methods for testicular fixation, but unabsorbable material and at least two stitches are preferable, in order not to allow a torsion pivot to form. Some authors fix the testis using a sub-dartos pouch, similar to the technique routinely used to treat cryptorchidism. THE CONTRALATERAL TESTIS MUST BE PEXED IN ANY TT CASE, including cases of post-ischemic testicular atrophy.

Post-operative complications are uncommon. Repetitive episodes of torsion after fixation are rare (0.3%) [31]. Fertility in men affected by unilateral TT (treated either with orchiectomy or orchidopexy) is comparable to the normal population [32].

Time for insertion of substitutive testicular prosthesis in orchiectomy or total atrophy cases is controversial. Many suggest to insert the prostheses only after puberty, in order to avoid a small prosthesis that will need to be changed later in the case of pre-pubertal children. It has been suggested that the insertion of a prostheses in emergency conditions, in the presence of gonadal necrosis, may have a high risk of infectious complications and extrusion. However, some argue that psychological aspects of an empty scrotum have to be considered, and that simultaneous insertion of a prosthesis and orchiectomy is safe [33]. Complications may be more frequent if the insertion of a testicular prosthesis is decided after a long delay time [34].

Perinatal TT patients are a special cohort. They are a minority of TT affecting topical testes, are typically extra-vaginal and usually happen during the third trimester of intra-uterine life, mostly affecting term neonates. Most cases exhibit irreversible late ischemia (histologically calcification, fibrosis, necrosis, and syderophages are usually described after orchidectomy [35]). Bilateral cases are uncommon (circa 1/5 of the cases), 67% asynchronous [2]. A recent metanalysis

shows that salvation of the affected gonads is unusual (7.7%, 2/3 of those documented as post-natal episodes) [36].

In typical cases the babies present unpainful augmentation of scrotal volume ("pseudotumoral"), a firm testis, and scrotal discoloration (bluish/violaceous/pale) from birth. Ultrasound shows a heterogeneous non-vascularized testis with hypoechogenic areas, calcifications, and secondary hydrocele. Acute/treatable TT caused by bell clapper malformation, is uncommon in newborns (less than a quarter of AS cases in this age) [37], presenting as irritability, inconsolable crying and a painful swollen testis. Those cases need emergency surgery and should be treated as typical TT. A previous description of a normal testis by the pediatrician is useful to suggest an acute episode of TT.

The level of urgency involved is controversial: as presentation is usually late, an emergency surgery is debatable. On the other side, if there is any doubt about the possibility of a recoverable organ/recent torsion episode emergency surgery is recommended. Most surgeons argue for urgent surgery, intending to fix and protect the other side from future problems, to detect synchronous previously undetected contralateral torsion and to possibly save some gonads, although uncommon. Those who disagree depart from considering the risk of anesthesia in neonates and the rarity of acute/recent torsion in this population.

An inguinal incision is used in those cases. Contralateral fixation is arguable, as immaturity is the cause for extravaginal TT, and the normal fixation is usually complete after the first 10 days of life. This aspect remains controversial, and many authors opt for contralateral fixation to get better chances of contralateral protection, considering the low risks associated to an orchidopexy, the possibility of an unknown bell-clapper contralateral malformation and the potential disastrous consequences of an eventual contralateral problem [38].

2. Appendix testis torsion

ATT commonly affects Morgagni's hydatid, the appendix testis located near the upper pole and the epididymis (Fig. 22.4). The patients are mostly pre-pubertal (6–12 years-old predominate): ATT is the most common cause of acute scrotum in pre-pubertal children [39]. The main symptom is acute pain, usually less intense than in TT and without irradiation. More mature children may locate the pain on the upper pole of the testis. Children presenting ATT usually do not present nausea and vomits.

Physical examination may evidence a painful distinct mass on the upper pole of the testis, independent of the testis per se, but the progression of the disease frequently complicates the exam, as edema and secondary hydroceles superimpose. In children, especially those with fair and thin scrotal skin and without severe scrotal edema a dark "dot" may be seen transcutaneously, representing the ischemic appendix testis. Transillumination may also help to individualize the mass near the testicular upper pole (Fig. 22.5). In ATT the cremasteric reflex is preserved and the cord is normal to palpation. Doppler ultrasound shows the twisted appendix near the epididymis/superior pole of the testis (a heterogeneous "salt and pepper" oval avascular mass with posterior acoustic reinforcement, hypoechogenic in the earlier

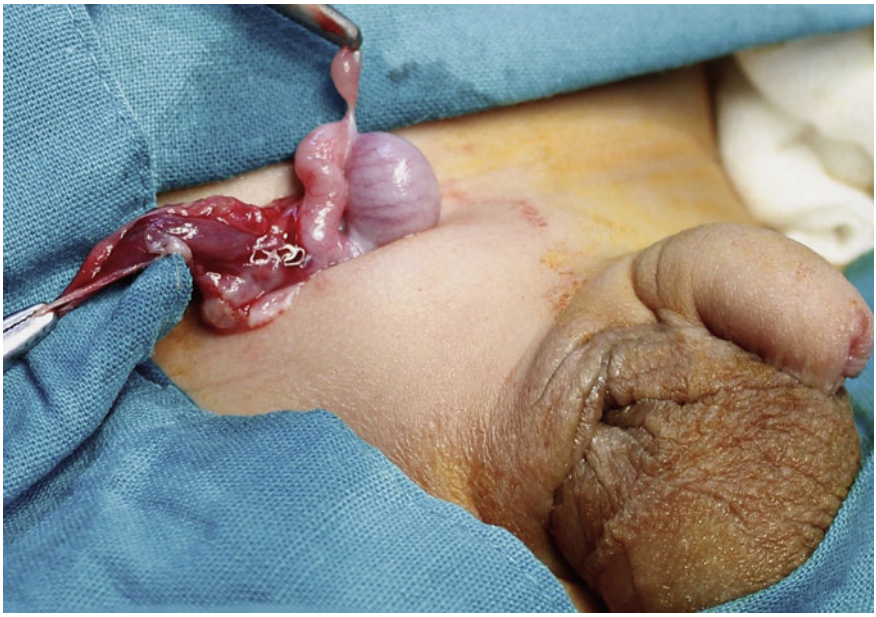

Fig. 22.4 Morgagni's hydatid, the most common form of testis appendix (Dr. Mohamed Fahmy, personal archive)

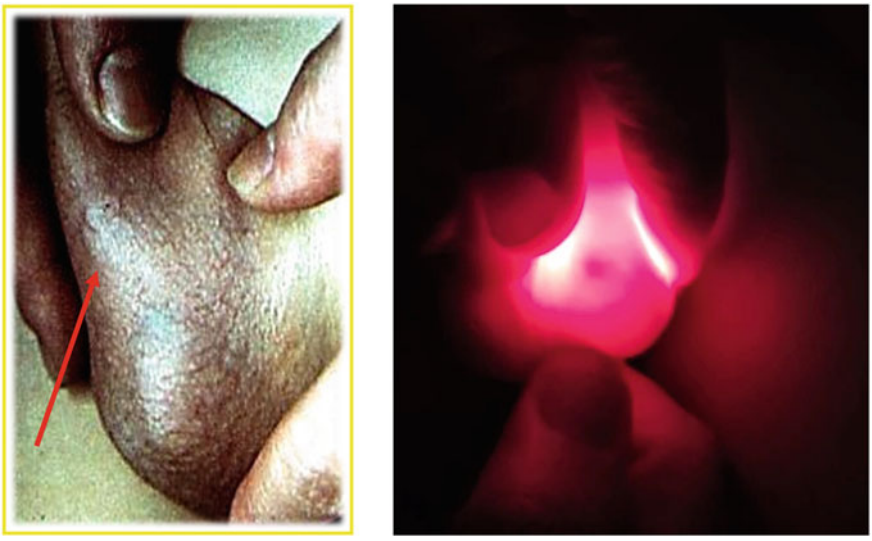

Fig. 22.5 ATT, blue-dot sign as seen in physical examination (left, red arrow) and by scrotal transillumination (right) (case from Dr. Samuel Dekermacher, MD)

cases—echogenicity tends to be higher in later cases) with a normal testicular flow (Fig. 22.6) [40, 41].

Treatment may be surgical (trans-scrotal exeresis of the twisted appendix testis) or conservative (pain treatment and rest). Protracted pain is frequent in cases conservatively treated (approximately a third of the patients) [31]. In some cases the twisted appendix auto-amputates and turns into a freely mobile palpable scrotal calcified nodule, also shown in ultrasound (Fig. 22.5). Metachronous contralateral ATT is very uncommon (2.6%) [31].

3. Orchiepididimytis

OE may be related to viral diseases, lower urinary tract malformations and/or urethral obstructions causing urinary tract infections or urine reflux towards vas

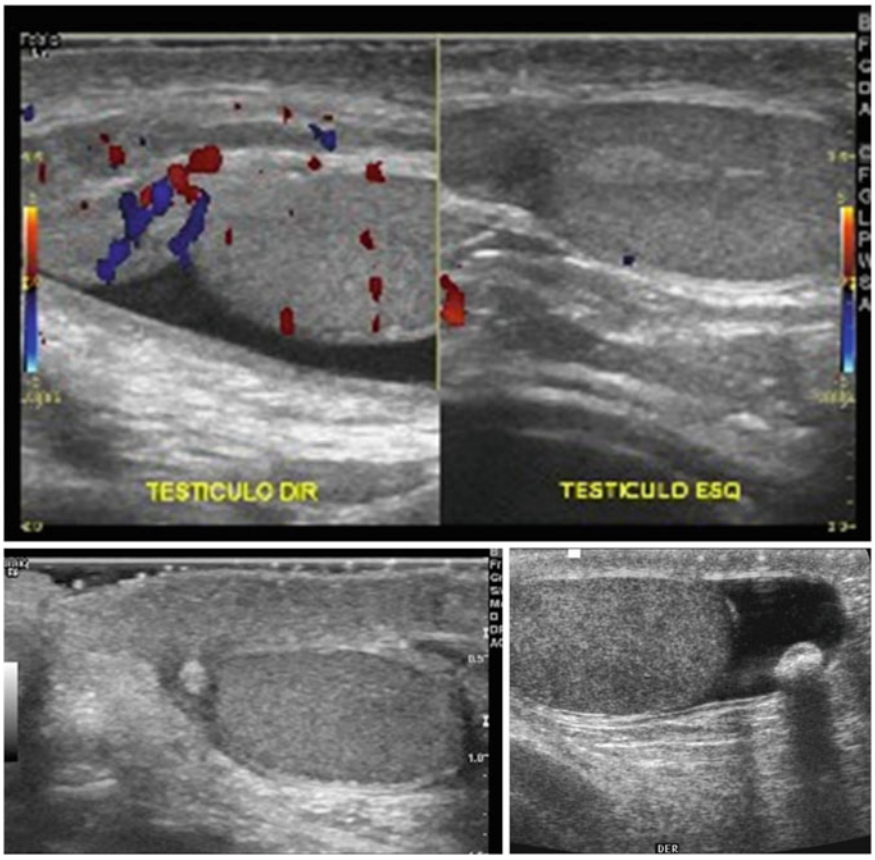

Fig. 22.6 ATT affecting the right side, acute phase (augmented heterogeneous isoechoic appendix testis) (upper picture) and medium term control (hyperechogenic remnant of the appendix testis) (lower left) and late control, with scrotal "calculus" (lower right) (case from Dra. Tatiana Fazecas, MD)

deferens (chemical orchiepididymitis) and voiding dysfunction with incoordination in pre-pubertal children (causing urine reflux under pressure to the vas deferens). In adolescents, sexually transmitted diseases are also to be considered. The incidence of OE in adolescents is estimated in 1.2 cases/1000 patients/year [42].

Mumps is a classical cause of OE, presenting 3–7 days after the typical clinical manifestations of the disease, and is most frequent in post-pubertal boys. 20% of the patients show bilateral affection and 30% may present late partial atrophy of the organ [43]. Mumps orchitis is rarer nowadays due to vaccination. Other viral diseases (especially paramyxo, adeno, and enteroviruses and influenza) may show similar clinical manifestations. Viral orchitis has recently been described in an adolescent after SARS-COV2 infection [42].

In OE cases the affected testis is indurated, painful, and swollen. The epididymis is also edematous and painful to palpation, as well as the cord. There is no abnormality in the organ position and the pain may be eased by elevation of the testis (Prehn's sign). Pain is usually subacute and progressive. Scrotal edema and associated hydrocele are frequent. Some patients present fever. Pyuria is common and dysuria may associate.

US/doppler shows augmented testis AND epididymis, scrotal edema, and secondary hydrocele. In the doppler evaluation, there is an augmented flow to the testis and the epididymis (Fig. 22.7).

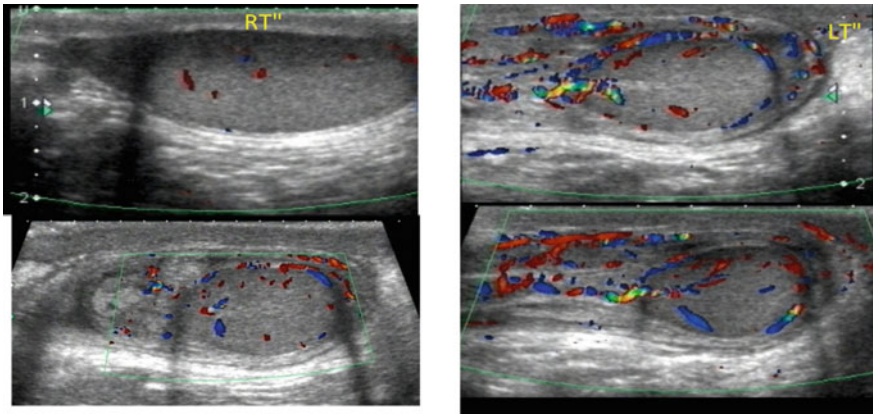

Fig. 22.7 Epididymitis. Doppler ultrasound, showing hypervascularization of the affected (left) testis (upper picture) (case from Dra. Tatiana Fazecas, MD and Dr. Samuel Dekermacher)

Treatment is non-surgical with anti-inflammatories, antibiotics (in patients presenting associated urinary tract infections), elevation of the testicle, rest, and treating voiding dysfunction/urinary tract malformation, whenever present.

4. Other miscellaneous conditions

Testicular trauma is a commonly alleged cause for AS. Patients frequently attribute pain to a recent relatively minor trauma (especially falls, fights against other children, and sports trauma). A wrong diagnosis of post-traumatic orchitis is a common mistake in TT cases. As a matter of fact, testicular trauma very rarely causes AS, as the testicles are highly mobile and rarely exposed to significant blunt trauma. Conversely, testicular trauma may be a CAUSE for TT, by inducing a forceful sudden defensive reflex cremasteric contraction. Ultrasound is extremely useful to exclude significant testicular trauma, presenting rupture/discontinuity of the albuginea, heterogeneous parenchyma, abnormal contour of the gonad, testicular hematomas, and hematocele [44].

Idiopathic scrotal edema is a relatively uncommon acute condition presenting edema and hyperemia involving the scrotal skin/subcutaneous tissue. The hyperemia usually extends to the adjacent skin (inguinal and genital). Some children have a previous history of allergy. There is no testicular pain and the testis/cord are normal to exam. Ultrasound shows exclusively scrotal/inguinal edema, with normal testis, epididymis, and cord. Idiopathic scrotal edema often regresses spontaneously. Regression may be accelerated by anti-hystaminics and/or steroids.

Neonatal adrenal Hemorrhage (presenting as volume augmentation and scrotal discoloration) [45], meconial peritonitis (presenting as scrotal calcified tumors [46]) and left renal vein thrombosis (causing secondary thrombosis of the gonadal vein) [47, 48] should be considered in the differential diagnosis of AS in neonates, but those patients usually show comorbidities, including prematurity and/or perinatal complications and clinical signs of the primary disease that associates with the scrotal signs.

Henoch-Scholein purpura and other vasculitides are rare diseases, often preceded by an infection that may affect the testis. The clinical manifestations are characterized by acute pain, due to small areas of testicular infarction, secondary to the primary affection, that may be bilateral and associate to scrotal edema. Those patients usually show symptoms of their primary disease (classically purpura, arthritis and abdominal pain), but occasionally the testicular affection presents as the initial manifestation. The differential diagnosis is important for those patients, as they exhibit higher risks for surgery and anesthesia in the presence of complications of the vasculitis (carditis, renal affection, pulmonary disease). Treatment of acute scrotum secondary to vasculitides is conservative, associating the treatment of the primary disease to symptomatics.

A torsion of an epidydimal cyst or epididymal appendix (Fig. 22.8) is a rare cause of AS, and is very difficult to differentiate from TAT [49]. Torsion of the epididymis per se is uncommon.

Strangulated hernias are frequently associated to testicular ischemia, by compression of the vascular structures of the cord (Fig. 22.9). The diagnosis is usually

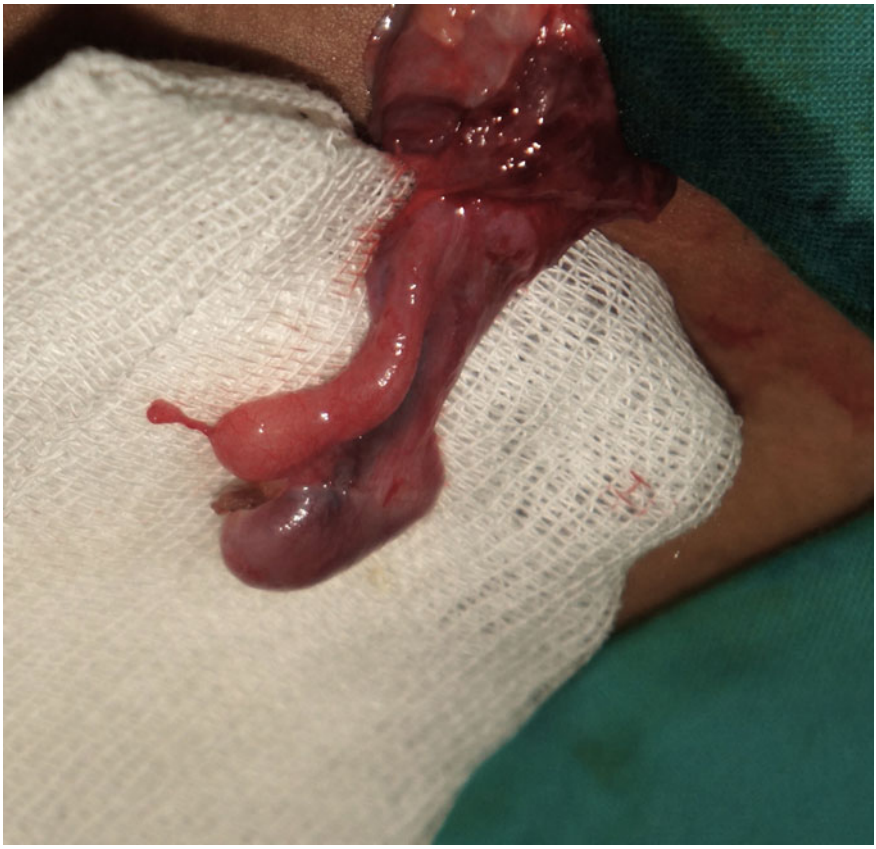

Fig. 22.8 Torsion affecting an epididymal appendix (Dr. Mohamed Fahmy, personal archive)

obvious, and treatment relies on the resolution of the strangulation episode by taxis maneuvers and surgery. An important observation is that in some cases protracted testicular ischemia (by direct compression of the vessels, vascular spasm or vascular trauma during cord manipulation) may be irreversible and lead to progressive atrophy of the gonad. If this risk is predictable the parents must be informed, in order to avoid medicolegal problems.

Fig. 22.9 Strangulated inguinal hernia. Patient diagnosed after 18 h. Upper picture: diffuse hyperemia of scrotal and inguinal skin, indurated and edematous testicle. Lower picture: necrotic testicle and area of intestinal necrosis (case from the author's archive)

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