Testicular Torsion

Learning Objective: At the conclusion of this continuing medical education activity, the participant will understand the pathophysiology, diagnosis and surgical management of testicular torsion.

Israel P. Nosnik, M.D.

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and

Harris M. Nagler, M.D.

Disclosures: Nothing to disclose

Sol and Margaret Berger Department of Urology
Beth Israel Medical Center
Albert Einstein College of Medicine
New York, New York
INTRODUCTION/HISTORICAL PERSPECTIVE

In 1810 Hunter described an 18-year-old man with no previous pain or injury who was “seized with pain” after a few hours of ice-skating followed by testicular atrophy.1 A year later the patient presented again with the same symptoms on the contralateral side. We now understand that this patient presented with the classic symptoms of torsion of the spermatic cord including the acute onset of testicular pain and ischemia, which if left untreated will result in subsequent infarction of the testicle. This case also demonstrates the propensity of the contralateral testis to undergo torsion after ipsilateral torsion.

However, it was not until 1894 that the pathophysiology of testicular torsion was described by Laurenstein who noted, “I am tempted to call the phenomenon torsion of the testis as it is this that twists first followed by the spermatic cord” (fig. 1).2 In 1913 Ombredanne advised early scrotal exploration for acute scrotal pain in children.3 It took until the 1930s to come to the realization that testicular viability decreases dramatically beyond 6 hours after the onset of pain.1

Testicular torsion will annually affect 1 in 4000 males younger than 25 years.4 More than 100 years later acute scrotal pain is the presenting complaint in fewer than 0.2% of emergency department visits. Nevertheless, the acute scrotum is an entity that is feared because of the significance of an error in or a missed diagnosis. The incidence of testicular torsion is bimodal with a peak incidence in the first few days of life and again at puberty (age 13 to 15 years), although it can occur at any age and has been reported in men as old as 78 years.5-7 The prompt evaluation and diagnosis of testicular torsion are of utmost importance as expedient surgical exploration and management can avoid testicular loss and the potential sequelae.

PATHOPHYSIOLOGY

The 2 types of spermatic cord torsion are extravaginal and intravaginal. Each type coincides with a spike in the bimodal age distribution. Extravaginal torsion occurs almost exclusively in neonates (early peak) while intravaginal torsion accounts for more than 80% of testicular torsion in males beyond the neonatal period (late peak) (fig. 2).8

During normal embryological development the testis descends from the abdomen through the inguinal canal via contraction of the gubernaculum, an extratessicular structure which is attached cranially to the cauda epididymis and lower pole tunica albuginea, caudally to the mesenchymal tissue of the scrotum and lies just anterior to the tunica vaginalis.9 Both types of testicular torsion result from twisting of the spermatic cord leading to testicular ischemia. However, extravaginal torsion results from twisting of the tunica vaginalis and testicle before complete descent into the scrotum and fusion of the tunica vaginalis to the scrotal wall. On the other hand, intravaginal torsion is the result of inadequate fixation to the testicular gubernaculum during descent of the testis, thus allowing the testis to rotate freely within the tunica vaginalis. This lack of fixation accounts for the “bell clapper” deformity noted in many cases of testicular torsion (fig. 3).

Intermittent torsion is caused by lack of fixation of the testis, allowing the cord to torque and detorse or reduce spontaneously. Although the lack of fixation is an anatomical defect enabling torsion to occur, it is not clear what actually precipitates torsion. It is hypothesized that the rotational force that twists the spermatic cord is caused by contraction of the cremasteric muscle fibers that are oriented in a spiral manner around the spermatic cord, which may occur during sleep or exercise.

The importance of torsion is the resultant testicular ischemia. The degree of ischemia is affected by the degree of torsion as well as the duration of the event. In animal studies 90 degree twists have been shown to cause no necrosis even after 7 days, whereas 360 degree rotation resulted in necrosis after 12 to 24 hours and 1440 degree rotation resulted in necrosis in only 2 hours.2 This variation likely reflects the difference between disruption of arterial and/or venous flow to the testis. As the testis twists on the cord, there is initially venous occlusion causing tissue congestion before subsequent arterial occlusion causing ischemic changes.

Depending on the duration of the process, the resultant morphological changes range from intense congestion to widespread extravasation of blood into the interstitium of the epididymis and testis. Eventually, hemorrhagic infarction engulfs the entire testis. In late stages the tests can become markedly enlarged and subsequently a sac of soft, necrotic hemorrhagic tissue (fig. 4).10 Canine studies have demonstrated elimination of all
spermatogenic and Sertoli cells by 6 hours of testicular ischemia and the loss of Leydig cells by 10 hours of ischemia. \(^{11}\)

Clinically, it has been observed that beyond 10 hours of torsion most patients will have significant atrophy unless spontaneous reduction has occurred or the torsion is limited to 180 to 360 degrees of rotation. \(^{12}\) Complete or severe testicular atrophy is expected in all cases of testicular torsion of greater than 360 degrees lasting for more than 24 hours. \(^{13}\)

The pathophysiology of injury to the affected testis is a combination of ischemia and reperfusion injury due to restoration of blood flow to the previously devascularized tissue. Reactive oxygen species such as hydrogen peroxide, hydroxyl radicals, superoxide anions, nitric oxide and other toxic by-products arise from activation of the xanthine oxidase pathway in parenchymal cells or from leukocytes that adhere to the reperfusing venule walls before undergoing migration across the endothelium. \(^{14}\)

These reactive oxygen species cause lipid peroxidation in the cellular and mitochondrial membranes which leads to changes in membrane permeability or disruption of membrane permeability allowing for DNA damage. DNA damage can lead to loss of cell viability. \(^{15}\) Although acute testicular torsion is generally unilateral, there is evidence that there may be a bilateral effect on testicular function, and these observations will be discussed.

**DIFFERENTIAL DIAGNOSIS**

Patients with testicular torsion will most often present with an acute scrotum. Before one attempts to render an appropriate diagnosis it is essential that one appreciates the scope of the differential diagnosis. The differential diagnosis of the acute scrotum is broad and includes emergent pathology of abdominal or retroperitoneal contents. Acute non-traumatic scrotal discomfort may be related to pathology of the scrotal contents including the testicle, epididymis and cord structures but may also be caused by pathology directly or indirectly related to abdominal or retroperitoneal structures including ureteral obstruction or ureteral calculi.

Testicular tumors with acute hemorrhage with or without a history of apparently minor trauma may be acutely painful.
Inflammation of the testicular adnexa (epididymitis) may also cause severe scrotal pain. Torsion of testicular appendages (appendix epididymis or appendix testis) or epididymo-orchitis may also have similar presentations. Incarcerated inguinal hernia may cause symptoms mimicking testicular torsion. Ureteral calculi may present with acute referred testicular pain without associated physical findings. It is important to note that the acute scrotum presentation may be part of a constellation of signs and symptoms masking other emergent processes including peritonitis, Fournier’s gangrene and ruptured abdominal aortic aneurysm. These findings may be present due to a patent processus vaginalis or referred pain. Careful abdominal examination is essential in the evaluation of every patient presenting with scrotal pain. For a more complete list of differential diagnoses see Appendix.

Clinical presentation. As with all medical diagnoses, the clinical presentation of acute scrotal pain as well as a thorough history and physical examination are the bedrock upon which clinical diagnosis rests. The interview of a patient presenting with acute scrotal pain should be focused yet thorough with emphasis on patient age, description of the pain, complete urological history and relevant social history.

The pain must be fully characterized in regard to the onset, severity, nature, duration and location. Testicular torsion pain is generally characterized as being of acute onset and severe in nature. A history of similar episodes may suggest intermittent testicular torsion.

Recent trauma or exercise and association with morning erections have been noted in patients with testicular torsion. Patients with torsion may have nausea, vomiting and fever, while symptoms of dysuria, urgency and frequency may suggest an infectious nature. Radiation of pain or referred pain to the scrotum could indicate intra-abdominal pathology such as appendicitis or ureteral lithiasis, and as such, the urologist should maintain a low threshold for further investigation of atypical presentation of non-scrotal causes of testicular pain. However, acute testicular pain may be referred to the lower abdomen. In fact failure to examine the testes in a young boy with lower abdominal pain may lead to an error in diagnosis.

History. A complete medical history should be obtained and focus on genitourinary abnormalities and surgery. A history of testicular fixation for torsion, cryptorchidism or other pathology does not exclude the possibility of testicular torsion as de novo torsion has been reported after orchiopexy. Further questioning should address recent instrumentation of the urogenital tract, genitourinary trauma, exercise and history of genitourinary infection. Social history with a focus on sexual activity must be obtained in adults, adolescents and younger boys. Sexual history, including recent vaginal or anal intercourse, number of partners, safe sex practices and presence or absence of penile discharge or lesions, must be obtained particularly when epididymitis is the suspected etiology of acute scrotal pain and may help guide appropriate antibiotic therapy.

Nausea, vomiting and anorexia are commonly associated with testicular torsion but are less common in cases of epididymo-orchitis and torsion of a testicular appendage. These symptoms have been reported to have a positive predictive value of up to 98% and are present in up to 68% of those with confirmed testicular torsion compared to less than 5% of patients with epidydimo-orchitis and torsion of a testicular appendage. Patients with torsion of a testicular appendage tend to have pain that is less severe in nature. Epididymitis generally presents with a more insidious onset of pain and swelling. It is important to note that although the pain of testicular torsion is generally acute in onset, gradual onset of pain has been reported in up to 25% of cases.

Physical examination. Physical examination of patients with acute scrotal pain should include abdominal, inguinal and genital examinations. It is important that the patient be in a comfortable environment where privacy is ensured to decrease anxiety. Patients with testicular torsion appear markedly uncomfortable, distressed or agitated, and so the examiner should note these signs. Patients with epididymo-orchitis and/or torsion of a testicular appendage, although uncomfortable or in pain, are less agitated and able to remain stationary and calm.

The importance of early examination of the scrotum on presentation is stressed as progressive inflammatory changes may make mask findings that may clarify the diagnosis. The scrotum should be visually evaluated for evidence of trauma, swelling, skin changes (ecchymosis/purpura), masses, color (“blue dot sign” suggesting torsed appendix testis) and, if discernable, the position and lie of the testis within the scrotum. The axis should be noted because elevation of the testis within the scrotum suggests testicular torsion. Also, with torsion the spermatic cord becomes shortened, elevating the testes and changing its axis. However, the axis of the unaffected testis should also be observed for the presence of an abnormal axis or bell clapper deformity which suggests torsion.

Before palpation of the testis the cremasteric reflex should be elicited by lightly stroking or pinching the superomedial aspect of the thigh and observing for elevation of the ipsilateral testis within the scrotum. The presence of ipsilateral cremasteric reflex has been reported to all but rule out the presence of testicular torsion. Rabinowitz demonstrated that the correlation between the presence of ipsilateral cremasteric reflex and absence of testicular torsion was 100% in a 7-year evaluation of 245 patients with acute scrotal swelling. He concluded that the presence of cremasteric reflex was the most predictive finding on physical examination in ruling out testicular torsion and the absence increased the likelihood of testicular torsion.

In a retrospective review of 160 boys (mean age 12.2 years) presenting with acute scrotal pain, Cifci et al also found that ipsilateral loss of cremasteric reflex was the most accurate physical examination predictor of testicular torsion with a sensitivity of 92%, specificity 94% and positive predictive value 94%. Although this more recent study did not show 100% sensitivity and specificity as reported by Rabinowitz, the loss of cremasteric reflex remains an important observation in testicular torsion.
Palpation of the scrotum in the male with scrotal pain is difficult, and so it is generally easier to begin on the unaffected side. The skin should be palpated for thickening or edema, fluid collections (such as scrotal abscesses), tenderness and subcutaneous emphysema. The normal testis is mobile and the epididymis may be palpated posterolaterally. The examiner should note the size, tenderness (localized vs diffuse), lie (high vs low within the scrotum) and axis (horizontal vs vertical) of the testis which may be horizontal due to shortening of the cord proximal to the testis. The remainder of the scrotal contents should be examined, including the spermatic cord as it courses to the external inguinal ring. A finding of a knot or significant tenderness within the cord suggests testicular torsion. Any suspected hydroceles or spermatoceles should be transilluminated.

Physical findings may also help differentiate intravaginal vs extravaginal torsion of the spermatic cord. An enlarged testis with bluish discoloration in the neonate suggests extravaginal torsion. Intravaginal torsion may be suggested by shortening of the spermatic cord with the testis riding high in the scrotum and assuming an abnormal axis. The position of the epididymis may help confirm the clinical diagnosis. An anterior location suggests testicular torsion but the epididymis can be found out of its normal position even with testicular torsion since greater degrees of torsion may return the epididymis to its posterior lateral position. A horizontal lie (bell clapper deformity) of the contralateral testis is highly suspicious and suggests a cause for torsion of the affected testis (fig. 2).

The penis should be examined for discharge or masses. Discharge may suggest an inflammatory or infectious process. Fullness in the groin may suggest an inguinal hernia, and a more thorough examination of the inguinal canal may be hindered by patient discomfort. Inguinal lymphadenopathy may suggest inflammatory changes related to genitourinary infection. The preauricular area should be examined for clinical evidence of mumps, which may be indicative of mumps orchitis as the etiology of scrotal pain.

Manipulation of the testicle may aid in the diagnosis. Elevation of the affected testis may relieve pain when the pain is the result of epididymitis. However, elevation of a torsed testicle increases discomfort as a result of increased ischemia caused by the elevation, which is known as Prehn’s sign. Its diagnostic accuracy has been reported as 83% sensitive and 90% specific with a positive predictive value of 70%.24

Laboratory assessment. Urinalysis should be performed in all patients who present with scrotal pain, regardless of age. Greater than 10 white or 10 red blood cells per high power field suggest epididymitis, although the findings are generally nonspecific and must be interpreted in the context of the history and physical findings. All sexually active males require urethral cultures for Neisseria gonococcus and Chlamydia trachomatis. There are currently no clinically useful serum markers for testicular torsion but marked elevation of serum CK-MM has been noted in animal studies.26 Future studies are necessary to validate these findings in humans.

All cases of acute scrotum should be considered testicular torsion until proven otherwise.19 Emergent urological consultation is mandatory and should supersede any further testing beyond the physical examination because of the critical time requirement to restore blood flow to the testis.

Radiographic studies. Although the diagnosis of testicular torsion is based on history and physical examination in conjunction with findings on surgical exploration, imaging is particularly valuable when the diagnosis is unclear. Before color flow imaging was developed, radionuclide scintigraphy was the principal means of evaluating the scrotum. Scintigraphy has a high degree of sensitivity and specificity. The scintigraphic findings are dependent on the duration of ischemia with the 3 recognized phases of acute, subacute and late. Static images in the acute phase show reduced perfusion and reduced tracer accumulation in the affected testis. In the subacute phase an increase of flow in the scrotal tissue is seen as a result of peritesticular reactive hyperemia, which appears as a halo of hyperactivity around the testis with no accumulation of radiotracer within the testis. The late phase is marked by a reduction in the peritesticular halo with a marked tracer deficit in the testis, indicating the presence of non-vital tissue.27

Beginning in the late 1980s, studies demonstrated that color flow Doppler was at least as accurate as radionuclide scintigraphy. Since then color Doppler ultrasound has become the gold standard for imaging the acute scrotum primarily due to accessibility to the equipment and technicians, reduced cost and decreased time required to perform ultrasonographic evaluation.29,30 The major indication for testicular ultrasound in the acute setting is differentiation between epididymo-orchitis and testicular torsion as a strong enough clinical suspicion should lead to immediate scrotal exploration. In acute epididymo-orchitis there is increased flow within the epididymis and possibly the testis but this flow is generally diminished in torsion which also obstructs the vascular flow to the epididymis, aiding in distinguishing between the 2 conditions. The use of ultrasound for evaluation of neonatal testicular torsion is limited, especially in neonates and prepubertal boys in whom the vessels are difficult to evaluate even by an experienced ultrasonographer.31

Scrotal ultrasonography is performed with the patient supine and a towel placed between the legs to support the scrotum. The best imaging is obtained using a 10 to 14 MHz linear array transducer in direct contact with the scrotal skin. Both testes are examined first within the field of view to compare echogenicity with the contralateral side. The scrotal skin should be measured for thickness. Each testis is then examined in at least 2 planes (longitudinal and transverse) with attention first placed to the unaffected side. The gain should be adjusted accordingly to accurately compare the 2 testes.

Gray scale ultrasound findings vary with the duration of torsion and degree of spermatic cord rotation and are generally nonspecific for testicular torsion. The testis and epididymis may show a normal sonographic appearance 2 to 4 hours after torsion onset.27 Testicular swelling and decreased echogenicity diffusely, and rarely focally, are the most commonly encoun-
ered findings 4 to 6 hours after the onset of torsion. At 24 hours after onset the testis has a heterogeneous echo texture secondary to vascular congestion, hemorrhage and infarction.\textsuperscript{34} Additionally, signs of scrotal edema or reactive hydrocele may be present but these findings remain nonspecific. In a recent prospective study a spiral twisting of the spermatic cord ("whirlpool sign") at the external inguinal ring was seen in 14 of 23 cases of torsion.\textsuperscript{35} The twisting induced an abrupt change in the course, size and shape of the spermatic cord below the point of torsion and appeared as a round or oval homogeneous extratesticular mass with or without blood flow that could be traced cephalad to the normal spermatic cord. A whirlpool sign was also found to be specific for torsion in a retrospective study of 221 sonograms for acute scrotum.\textsuperscript{36} Gray scale ultrasound is greatly enhanced by the use of color and power Doppler ultrasonography which can reliably detect intratesticular flow.\textsuperscript{37} Because gray scale imaging early in torsion is generally normal, the Doppler component of the study is essential.

Color Doppler ultrasonography allows for visualization of blood flow within a viewing area overlaid on the 2-dimensional gray scale image by application of the Doppler principle, which can detect the absence of blood flow within the testis and is a hallmark of testicular torsion and ischemia. Power Doppler, an adjunct to color Doppler, enhances the sensitivity to flow with the displayed hue modulated by the strength of the Doppler signal, which, in turn, is proportional to the number of red cells moving within the sample volume. Thus, the display is a map of the distribution of moving red cells above the sensitivity threshold of the system. Color and power Doppler settings are optimized to display low flow velocities, and demonstrate flow within the testes and surrounding structures (epididymis, other intrascrotal masses and spermatic cord).

It was hoped that the development of power Doppler, which is approximately 15 dB more sensitive than color Doppler imaging for the detection of flow, would improve imaging, particularly in prepubertal boys in whom visualization of intratesticular flow is particularly challenging. Although some improvement in the ability of power Doppler imaging to detect testicular flow has been reported, the ability to detect intratesticular flow in children has not improved considerably.\textsuperscript{38} The role of color and power Doppler in the evaluation of the acute scrotum is well established.\textsuperscript{28, 39, 40}

The absence of testicular flow on color and power Doppler ultrasound is considered diagnostic of ischemia, provided the scanner is optimized for detection of slow flow.\textsuperscript{41} Using the absence of identifiable intratesticular flow as the only criterion for detecting testicular torsion, color Doppler ultrason was 86\% sensitive, 100\% specific and 97\% accurate in the diagnosis of torsion and ischemia in painful scrotum.\textsuperscript{42} However, it is important to note that the presence of testicular blood flow does not exclude the diagnosis of testicular torsion as flow may be visible at the periphery of the testis, particularly during early torsion. The reasons for these false-negative findings include examiner experience, intermittent torsion or lesser degrees of severity of torsion.\textsuperscript{43} This finding is particularly important if the patient is no longer symptomatic since detorsion may have occurred.

In cases of epididymitis color and power flow Doppler will demonstrate increased flow within the epididymis with associated gray scale imaging findings and correlates strongly with the hyperemic characteristics of epididymitis. Spectral waveforms of the testicular artery and cremasteric artery should be obtained. The normal spectral waveform for the testicular artery is low resistance, high flow pattern, whereas the cremasteric artery displays a high resistance, low flow pattern.\textsuperscript{44} The outflow resistance, as calculated by determining the resistive index within the intrascrotal vessels, is defined as (peak systolic velocity – end diastolic velocity)/(peak systolic velocity). The resistance index in a healthy testis is rarely less than 0.5\textsuperscript{31} and a finding below that threshold may suggest outflow obstruction.

Magnetic resonance imaging, specifically dynamic contrast enhanced subtraction techniques, has been evaluated as a potential addition to the diagnostic armamentarium for testicular torsion. The study is particularly sensitive and specific (up to 93\% and 100\%, respectively\textsuperscript{8}) but the technique is hampered by the prohibitive cost, time required to perform and need for 24-hour standby availability of scanners. Dynamic contrast enhanced subtraction magnetic resonance imaging may assist in the diagnosis of testicular torsion in neonates and prepubertal boys given the difficulty of ultrasound evaluation of this population. The use of magnetic resonance imaging remains investigational and requires further evaluation.

MANAGEMENT

Early torsion. Patients presenting with acute onset of pain whose history and physical examination support the diagnosis of testicular torsion should undergo surgical exploration. Adjunctive or confirmatory testing may be obtained if these tests do not result in additional delay in intervention. During the examination the urologist should attempt manual detorsion. With an assistant holding the patient’s hands, the examiner should then gently elevate the testis toward the ipsilateral inguinal ring. The thumb and forefinger are then used to turn the testis laterally while stabilizing the cord above the testis.\textsuperscript{45} If the testis is amenable to manual detorsion, the spermatic cord will lengthen and the testis will assume a normal anatomical position in the scrotum with nearly immediate relief of pain. Should turning the testis laterally not relieve the torsion, the examiner should attempt turning the testis medially. The largest reported series reviewing the direction of spermatic cord rotation for confirmed testicular torsion suggests that 67\% of torsed spermatic cords are rotated medially.\textsuperscript{46} Success rates for manual detorsion ranging from 3\% to 57\% have been reported, supporting at least an early attempt at detorsion to decrease the ischemia time before surgical exploration.\textsuperscript{47-49}

Regardless of whether the testis can be manually detorsed, scrotal exploration and bilateral testicular fixation should still be performed as residual torsion poses a risk to testicular viability.\textsuperscript{46} The risks, benefits and alternatives to the procedure must be discussed with the patient and his
appropriate representative. This discussion should focus on the possibility of testicular loss should a non-viable testis be found on exploration as well as the option of placing a testicular prosthesis in the event of loss in postpubertal males.

Surgical management. The testis can be approached with bilateral transverse incisions, a single midline raphe incision or a single transverse incision to allow adequate exposure of both testes after opening dartos fascia. Attention is first focused on the affected testis, which, after opening the tunica vaginalis, is detorsed to restore perfusion. A viable testis should promptly regain its normal color. A testis with marginal viability should be placed in warm sponges and observed for several minutes. The cord may be examined using an intraoperative Doppler device to ensure return of arterial flow. A necrotic testis found at exploration should be removed by dividing the spermatic cord into 2 or 3 segments and suture ligated to ensure control of the cord vessels.

Once a testis considered viable is replaced in the tunica vaginalis, it should be fixed to the dartos using fine non-absorbable sutures. Some advocate opening the tunica vaginalis widely and fixing the tunica vaginalis to the dartos muscle directly. Animal studies on suture fixation of the testis indicate that using non-absorbable suture results in normal spermatogenesis in 94%, minimal focal tubular necrosis in 23% and complete circumferential adherence to the dartos pouch in fixed testes. These findings suggest that the reactive changes and granulation around non-absorbable suture used to fix the testis may not significantly affect spermatogenesis and provide satisfactory fixation of the testis within the scrotum.

We advocate 3-point fixation, which virtually guarantees no opportunity for the testis to rotate in the x, y or z planes, to ensure no further recurrence of torsion (fig. 5). Care must be taken to avoid injury to the epididymis or vas by fixation sutures. Others recommend fixation at 2 points on the lateral and medial mid pole of the testicle as these areas have been demonstrated to be less vascular and thus less likely to be associated with further vascular compromise.

In a recent series of 11 to 14-year-old patients presenting 6 to 7 hours after the onset of torsion Kutikov et al performed testicular “fasciotomy” by making a longitudinal incision through the tunica albuginea of the affected testis after detorsion of the cord during surgical exploration. The tunica albuginea was then closed without tension using a harvested tunica vaginalis flap. The authors indicated that the affected testis exhibits a compartment syndrome preventing reperfusion and resulting in additional ischemia and cellular damage. The use of this technique remains investigational and further studies are needed to review long-term outcomes. A testicular prosthesis may be inserted for cosmetic reasons in cases of postpubertal torsion when the testis is considered non-viable and removed, although this is best delayed if there is significant scrotal edema.

After fixation or removal of the affected testis, the contralateral testis must be explored. In nearly all cases a bell clapper deformity of the contralateral testis is found. The contralateral testis should be fixed in the same manner as the affected testis using non-absorbable suture to establish fixation as torsion of the previously fixed contralateral testes has been reported. The loss of a solitary testis because of failure to perform an orchiopexy is an unacceptable, catastrophic outcome and should not occur. After establishing excellent hemostasis, the tunical vaginalis and dartos layers are closed using absorbable suture, and the skin is closed in either an interrupted or running subcuticular fashion.

Intermittent torsion. Intermittent torsion is defined as recurrent scrotal pain secondary to torsion of the spermatic cord and spontaneous detorsion. Patients with a history of multiple self-limited episodes of acute scrotal pain but pain-free at the time of presentation are candidates for elective scrotal exploration and testicular fixation. Of a series of patients who underwent scrotal exploration and testicular fixation for intermittent torsion 46% had a bell clapper deformity and 97% had no further episodes of testicular pain.

Late/missed torsion. Missed testicular torsion as a result of late presentation (>24 hours after the onset of pain) should be explored as are cases of early torsion with orchiectomy performed in the event a torsed necrotic testis is found. Historically, there was bias towards the aggressive attempt to salvage torsed testes but a large number of these salvaged testes undergo secondary atrophy with reported rates of 43% at 12 to 24 hours and 85% at >24 hours after the onset of pain. These rates indicate that the salvaged testes may be of no benefit and perhaps could be a liability with increased morbidity and atrophy in a significant number of
cases. Therefore, we recommend removal of the testis that does not demonstrate reperfusion after a prolonged interval.

Secondary effects. Testicular torsion may have effects beyond the loss of the torsed testis. Unilateral testicular torsion seriously interferes with subsequent spermatogenesis in about 50% of patients and borderline impairment in another 20%. Subfertility (sperm count <20 million per ml) occurs in 36% to 39% of patients after torsion. At long-term follow-up semen analysis may be normal in only 5% to 50% of patients. Those with atrophy or who have undergone orchiectomy have a significantly lower sperm count than those without atrophy.

There are several theories to explain bilateral exocrine failure after unilateral torsion including immunological mechanisms, previous episodes of silent intermittent torsion and reflex vasoconstriction. The testis is an immunologically privileged site with a blood-testis barrier composed of tight junctions between Sertoli cells within the seminiferous tubule. Breakdown of this barrier after ischemic damage exposes the antigenic material within the tubule to the immune system resulting in an immunological response that may affect the contralateral testis.

In rats experimentally induced 720 degree torsion caused damage to the contralateral testis and detorsion after 24 hours offered no protection. However, in this same rat model orchiectomy 24 hours later with perioperative antilymphocyte globulin and splenectomy on postoperative day 3 prevented contralateral testicular damage. This result indicates that contralateral damage may be affected by immunological events, and immunosuppression and removal of the antigenic stimulus were protective. The clinical correlate of these experimental findings is that agglutinating antisperm antibodies have been found in 20% of patients after testicular torsion but neither this finding nor the presence immunoflourescent antibodies was significantly correlated with infertility. However, immobilizing antibodies do show a significant correlation with infertility.

On the other hand, oligospermia after unilateral torsion may be the result of a pre-existing bilateral defect. Biopsies of the contralateral testis taken at exploration within 24 hours of the onset of acute scrotal pain show pathological evidence of maturation arrest, germ cell degeneration, tubular hyalinization, immature tubules and focal thickening of basement membranes in 57% to 88% of cases. While the exact cause of these changes is unknown, they may be a result of intermittent silent torsion or underlying testicular abnormalities. Although a preexisting condition may be responsible for testicular changes after torsion, it is unlikely to be the primary cause because there is a clear correlation between the duration of torsion and total sperm count, unless the damage is compounded by another mechanism.

The spermatic cord under distress such as acute torsion induces sympathetically mediated reflex vasoconstriction of the contralateral spermatic vessels. Experimentally induced testicular torsion in rabbits causes an immediate and progressive decrease in contralateral testicular blood flow with a gradual return of normal flow after detorsion. After 2 hours of experimental torsion in the rat a 73% decrease in blood flow was noted in the ipsilateral gonadal artery and a 43% decrease in the contralateral gonadal artery.

In light of the various pathophysiological mechanisms that have been proposed, there have been many experimental treatments to minimize or prevent testicular damage after torsion. Interventions such as immune suppression, chemical sympathectomy to prevent contralateral vasoconstriction, agents to prevent reperfusion injury (calcium channel blockers and taurine) and testicular cooling have been studied in animals. Except for scrotal cooling, which has been suggested to delay ischemic injury to the testis, none of these therapies has been tried in humans.

MEDICOLEGAL

Factors that make torsion an active area of litigation may include the urgency needed in the diagnosis and treatment, diagnostic uncertainties, delays in presentation and/or diagnosis, a relatively common rate of adverse outcome (testicular loss) and psychological impact related to the loss of a testis. In a retrospective review of 39 cases in 18 years from the database of a large company insuring more than 50% of the physicians in the state of New Jersey the liability rates in paid claims were 74% misdiagnosis, 48% improper referral, 19% no radiological study, 13% failure to explore, 13% surgical error, 10% falsified records and 3% unable to determine (multiple liabilities in most cases, therefore greater than 100% total). The most common errors in diagnosis were epididymitis (72%), followed by intermittent torsion (13%), gastroenteritis (8%) and renal colic (5%). Urologists were most frequently faulted for failure to order a radiological study, or errors in surgical technique or judgment.

The rate of payment in claims for an atypical presentation is about half that of typical presentations, suggesting that these cases are somewhat more defensible. Claims with late presentation paid out at a similar rate as cases with early presentations or when the timing of presentation could not be determined. Despite the well accepted negative effect of delayed presentation on the medical outcome, this aspect alone had no effect on legal outcome.

Since the report of Janetschek et al testicular torsion is widely assumed to be associated with infertility and these concerns were heightened by the study of Nagler and deVere White. The true frequency of infertility after torsion is unknown. A cause and effect relationship between torsion and its treatment, and infertility has not been clearly established but, nevertheless, these concerns affect the management of torsion and the medicolegal response.

The astute urologist must always be cognizant of the risk of testicular torsion and have an extremely low threshold for radiographic evaluation and prompt scrotal exploration. Time is of the essence when treating patients with testicular torsion. Early diagnosis and intervention are essential to the preservation of testicular function.
APPENDIX: DIFFERENTIAL DIAGNOSES OF ACUTE/SUBACUTE SCROTUM

Anatomic:
- Torsion of the spermatic cord
- Torsion of the appendix testis
- Torsion of the appendix epididymis
- Communicating hydrocele
- Inguinal hernia
- Hydrocele
- Hydrocele of the cord

Inflammatory:
- Epididymitis
- Epididymo-orchitis
- Trauma/insect bite
- Inflammatory vasculitis (Henoch-Schönlein purpura)
- Idiopathic scrotal edema

Other:
- Dermatologic lesions
- Non-urogenital pathology (eg musculoskeletal)

EDITORIAL COMMENT

The authors have provided a comprehensive overview of a critically important clinical issue for urologists. Much of the data on the effects of testicular torsion are either anecdotal or derived from non-human models that may not accurately reflect the changes that occur after testicular torsion. Diagnosis of this condition can be difficult to make, especially for the patient who initially describes abdominal rather than testicular pain alone.

Litigation for delayed diagnosis of torsion is common. In most cases it appears that the fertility effects of torsion on the contralateral testis occur from the torsion event, and subsequent management of the torsed testicle does not necessarily affect infertility. A high degree of clinical suspicion is necessary for early diagnosis, and confirmatory testing of the diagnosis with color Doppler ultrasound should not be allowed to substantially delay management, as the authors have emphasized.

Peter Schlegel, M.D.
Department of Urology
New York Presbyterian Hospital
Weill Medical College
Cornell University
New York, New York

REFERENCES
1. The most predictive physical examination finding suggesting testicular torsion in patients presenting with acute scrotal pain and high riding testis is
   a. Loss of cremasteric reflex
   b. Scrotal edema
   c. Tenderness to palpation
   d. Blue dot sign
   e. Mass above the testicle.

2. Radionuclide scintigraphy 2 hours after the onset of testicular torsion will demonstrate
   a. Increased flow in the scrotal tissue
   b. Reduction in the peritesticular halo
   c. Reduced perfusion and tracer accumulation in the testis
   d. Increased tracer accumulation in the testis
   e. Increased peritesticular halo

3. After reperfusion of an ischemic testis, lipid peroxidation as a result of reactive oxygen species directly causes
   a. Decreased perfusion of the testicular stroma
   b. Fat necrosis within the scrotum
   c. Apoptosis
   d. Contralateral reflex vasoconstriction
   e. Changes in cellular and mitochondrial membrane permeability

4. The sole cause of bilateral exocrine failure after unilateral testicular torsion has been proven to be mediated by
   a. Immunological exposure of the testicular contents
   b. Prior episodes of silent intermittent torsion
   c. Contralateral reflex vasoconstriction
   d. Genetic predisposition to spermatogenic failure
   e. None of the above

5. The absence of testicular blood flow on power and color Doppler ultrasonography is considered
   a. Diagnostic of epididymo-orchitis
   b. Suggestive of contralateral torsion
   c. Diagnostic of torsion of a testicular appendage
   d. Diagnostic of testicular ischemia
   e. Diagnostic of testicular rupture