Torsion of the Testicle

It Is Time to Stop Tossing the Dice

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Abstract: In this review, long-held myths and misperceptions about the evaluation and management of testicular torsion are discussed, and recommendations for the management of patients who present with acute scrotal pain are presented.

Key Words: testicular torsion, epididymitis, scrotal pain, testicle, malpractice, epididymo-orchitis

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CASE

A 6-year-old boy was transferred from an outside hospital for evaluation of abdominal pain. His pain had begun earlier that morning when he awakened with nausea and vomiting. The initial complaint of pain was localized to the abdomen and a computed tomographic scan was performed because of suspected appendicitis. The patient's scrotum and testicles were never examined during the first evaluation. During the evaluation in the pediatric emergency department, the history of testicular trauma while swimming the day prior was elicited. The possibility of a testicular blunt trauma injury was entertained prior to the color Doppler ultrasound (CDS). The examiners, which included a pediatric urologist, noted testicular tenderness and possible bruising. However, no history or physical findings clearly diagnostic of testicular torsion (TT) were noted by the examiners. The cremasteric reflex was intact. The ultrasound demonstrated no blood flow to the left testicle (Fig. 1). During surgical exploration of the scrotum, the testicle was found to be torsed 720 degrees and could not be salvaged. Interestingly, the torsion occurred in a lateral direction and not medially, which is the most common direction.

MEDICOLEGAL RISKS

Testicular torsion is the third most common cause of a malpractice lawsuit in adolescent males 12 to 17 years.[1] The misdiagnosis of TT is not a recent problem, an unavoidable event, or one owned primarily by emergency physicians. In 2001, Matteson et al[2] reviewed closed case files specifically involving TT from the years 1979 to 1997 of a large medical malpractice insurance company based in New Jersey. The major liabilities for paid claims were an error in diagnosis (74%), the most common misdiagnosis was epididymitis (72%), urologists were named most frequently (48%) and atypical presentations of testicular torsion were common (31%). Another review of claims and indemnity payments for urologists by an insurance company in New York State (1985–2004) found that TT was tied as the fourth most commonly misdiagnosed condition.[3] That review describes 7 claims for a missed diagnosis and 8 claims of alleged negligent management of TT.[3] Unfortunately, although we are in good company, these reports serve as evidence that inaccurate assumptions about the evaluation of acute scrotal pain are resulting in lost testicles and large medicolegal pay outs.

Torsion of the testicle is a relatively rare condition occurring with an annual incidence of 4.5 in 100,000 in males 1 to 25 years.[4] Consequently, the incidence of TT presenting to emergency departments is low in contrast to other scrotal complaints, and there are signs and symptoms that are admittedly more commonly found with the testicle undergoing torsion.[5–8] This, however, is the perfect storm that sets up health care providers for overconfidence in their clinical diagnosis as well as the perpetuation of clinical myths and misperceptions.

In this review, long-held myths and misperceptions about the evaluation and management of TT are discussed and recommendations for the management of patients who present with acute scrotal pain are presented.

Myth 1: Testicular Torsion Can Be Consistently Ruled Out by Physical Examination Alone

The literature confirms that it is not possible to consistently and accurately differentiate testicular torsion (TT) from epididymo-orchitis (EO) and other scrotal pathologic abnormality by physical examination alone. For decades, there have been isolated physicians who have tried to warn us that the diagnosis of TT is fraught with problems. In 1967, Leape[9] warned that the presentations of the various conditions that cause scrotal pain and most frequently TT fail to "conform to the accepted clinical picture." Accurately sorting through the various conditions that can cause a painful testicle can be challenging, and experienced urologists have stated that initial clinical impressions are frequently flawed.[6,9–14] In a 25-year review of 199 children presenting with an acute scrotum, Sidler et al[15] stated that, "no discriminating features in either history or examination conclusively differentiated the correct diagnosis." And, ironically, one author who expressed confidence in the physical examination reported a 12.5% incidence of TT misdiagnosis.[16]

One of the major tripwires is the belief that the presence of a cremasteric reflex essentially rules out a TT. In fact, there are a number of series that report loss of the cremasteric reflex in 100% of patients presenting with TT.[11,17–19] And, these same authors tout the loss of the cremasteric reflex as a highly reliable sign that is diagnostic for TT. Unfortunately, this is not true. First, the cremasteric reflex is a fickle examination finding and it is well documented that the cremasteric reflex is frequently absent in up to 30% of males with normal testicles.[20,21] In fact, if cremasteric reflexes are tested regularly, one quickly realizes that this reflex is often subtle or barely perceptible. It is also absent in other conditions that present as acute scrotal pain. Caldamone et al[19] reported in their series that the cremasteric reflex was absent in 100% of their patients with TT, but it was also absent in 33% of patients with hydatid torsion and 25% of patients with epididymitis. Consequently, reliance on the cremasteric reflex for a decision to go to surgery or imaging should be a cause for concern.

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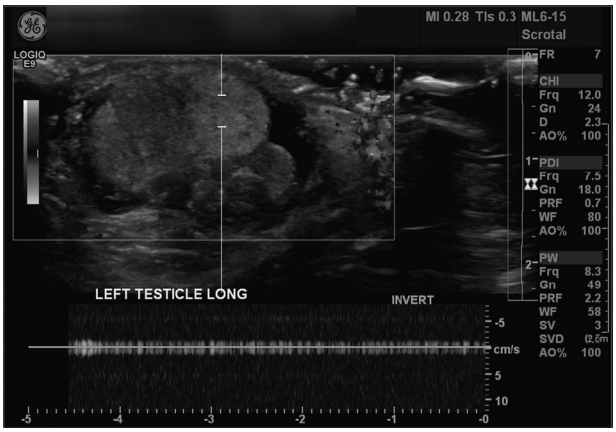

FIGURE 1. Color Doppler ultrasound study demonstrating no blood flow to the left testicle.

Although the cremasteric reflex is commonly absent in conditions other than TT, it is also present in a significant number of patients with TT. Most of the case series are small, but the documentation of a persistent cremasteric reflex with a TT in even 1 patient should be sufficient enough evidence to undermine confidence in this examination. Beni-Israel et al[5] reported that 5 (29%) of 17 patients with TT had a normal cremasteric reflex. Persistence of the cremasteric reflex was reported in 10 of 25 or 40% of TT patients by Van Glabeke et al.[22] In another small series, a normal cremasteric reflex was observed in 3 of 8 patients requiring orchiectomy for necrotic testicles after TT.[10] Paul et al[23] reported that the cremasteric reflex was present in 12% of 17 patients with torsion and described the cremasteric reflex as "suboptimal" in the diagnosis of TT. Other authors have described the cremasteric reflex in 8%, 10%, 20%, and 30% of their patients diagnosed with TT.[12,24–26] To use this examination for an imaging or consultation decision in the diagnosis of TT is an unnecessary gamble. Unfortunately, that is happening.

Scrotal erythema, edema, and testicular swelling are other physical findings that are commonly reported and described in patients with TT. Unfortunately, these findings are also very common in patients with EO and torsion of the appendix testis (TAT).[8–10,16,17,22,27–29] For example, Cass et al[28] reported scrotal erythema in 19% of patient with EO and 18% of patients with TT. Scrotal edema was present in 11% of the EO patients and in 17.5% of patients with TT. Swelling and tenderness of the testis were present in 68% of the patients with acute TT and in 20% of the patients with acute epididymitis. Mushtaq et al[16] described scrotal swelling in 30% of TAT, 75% of TT, and 97% of EO patients. Mäkelä et al[8] reported hemiscrotum swelling in 44% of TT, in 39% of TAT, and in 88% of EO cases. Waldert et al[26] described scrotal swelling in 75% of TT patients, 35% of TAT patients, and 92% of EO patients.

Another physical finding often considered strongly suggestive of epididymitis is pain around the upper pole of the testicle or epididymis. However, it also occurs with torsion of the testicle and the testicular appendage. A review of 543 surgical explorations of children and adolescents reported that the pain was restricted to the upper pole of the testis in 18.7% of patients with TT and in 40.8% of patients with torsion of the testicular appendage.[22] Karmazyn et al[12] reported upper pole pain in 7.3% of their patients with TT.

In addition, an enlarged epididymis may be common in patients with epididymitis, but it is also found in patients with TT. Multiple ultrasound studies have documented enlargement of the epididymis in the presence of a TT.[12,26,30–32] Karmazyn et al[12] reported that 43% of the children with TT had a swollen epididymis compared with 77% of the children with epididymitis and torsion of the testicular appendix. In the large series by Waldert et al,[26] the 2 patients with TT who were misdiagnosed as EO had persistent blood flow on color Doppler ultrasound and had an enlarged epididymis on the affected side. Ingram et al[32] report a missed torsion with confusion caused by the appearance of an enlarged epididymis. Nussbaum Blask et al[31] specifically studied the epididymis in a retrospective review of ultrasounds and found it to be enlarged in 47 of 50 pediatric patients with the known diagnosis of TT.

Although the lie of the testicle in torsion is frequently transverse, a vertical orientation is also common in patients with TT.[27,29] Abul et al[29] reported that a transverse elevated lie of the testis was observed in only 4 (36.4%) TT patients. A normal lie was described in 54% of the TT patients described in a small series by Kadish and Bolte.[11] A vertical orientation of the testicle was described in 17% of the TT patients in an article by Ceiftci et al.[24] A horizontal lie was reported as present in only 46% of TT patients evaluated by Eaton et al.[25] Murphy et al[10] described an abnormal position of the torted testicle in only 52% of their 31 TT patients. The abnormal testicular position was reported as a horizontal lie in 26% of those patients.[10] An abnormal orientation was described in only 21 of 41 patients with TT by Karmazyn et al.[12] In addition, one would think that retracted testicles would be seen primarily with TT, but in the series by Van Glabeke et al,[22] it was missing in 20% of torsion patients and present in 35.3% of patients with epididymitis. Other authors described testicle elevation, a "high lie" or "high position" in only 33%, 50% and 55% of their patients with TT.[5,10,25]

Myth 2: Testicular Torsion Can Be Differentiated From Other Causes of Scrotal Pathology by Its Pathognomonic History

Torsion of the testicle and its appendages and epididymitis have a significant overlap in their reported symptoms. Acute excruciating scrotal pain of relatively short duration before arrival in the emergency department is highly suggestive of TT.[28,29] However, rapid onset can be seen in epididymitis and torsion of the testicular appendage and gradual in TT. Cass et al[28] reported that 16% of their patients with TT had a gradual onset and a sudden onset in 51% of their patients with acute epididymitis. Sakellaris and Charissis[33] reported that acute testicular pain was noted in 15 (51.7%) of patients with epididymitis. Klin et al[34] reported in their chart review of 65 males who presented with an acute scrotum that 100% of their TT patients (n = 12) and 50% of EO patients (n = 21) presented with an acute onset of pain. Waldert et al[26] reported sudden onset of pain in 60 (96%) of 62 TT patients, in 121 (72%) of 168 TAT patients, and in 12 (50%) of 24 EO patients.

Although urinary signs and symptoms are expected to occur with EO patients, they can also be found in patients with TT. In the series by Cass et al,[28] urinary symptoms of frequency and/or burning were documented to be present with equal frequency in both TT and EO patients (7%). Lewis et al[7] described 2 sexually active TT patients who presented with pyuria and were initially misdiagnosed and treated as a sexually transmitted disease.

Nausea and vomiting are common with torsion of the testicle,[5,35] but they also occur with epididymitis. Mushtaq et al[16] reported vomiting in 33% of the TT patients and in 14% of the EO patients. Waldert et al[26] reported nausea and vomiting in 32% of TT patients, in 3% of TAT patients, and in 12.5% of EO patients. Lyronis et al[17] reported nausea and vomiting in

62.8% of TT patients and in 12.9% of patients with EO. Jefferson et al[36] felt that nausea and vomiting had strong positive predictive values (96% and 98%) because they found it documented in 69% and 60% of the boys with spermatic cord torsion and in none in patients with EO. Yet, in this retrospective review, there were only 5 patients with EO in contrast to 83 patients with TT.[36]

Reports of testicular pain are common to patients presenting with TT, TAT, and EO. Mushtaq et al[16] noted that testicular pain was reported in 92% of TAT patients, in 88% of TT patients, and in 76% of EO patients. Testicular pain was reported by Mäkelä et al[8] to be present in 68% of TT, in 94% of TAT patients, and in 58% of EO patients. Lyronis et al[17] reported testicular pain in 61.2% of patients with EO and in approximately 100% of patients with torsion of appendages and spermatic cord torsion.

Reports of abdominal pain alone are also found commonly among these conditions.[8,25–28,37,38] Cass et al[28] reported that 12.5% of their TT patients presented with only abdominal or inguinal pain. Anderson and Williamson[39] reported that 32 (5%) of 597 patients with TT and a fully descended testicle did not describe any scrotal pain and that 22% of the entire group had abdominal pain, which often preceded and exceeded the scrotal pain. In fact, the appendix was removed in 3 patients before the true diagnosis was made. Inguinal pain alone was described in 6% of the cases.[39] Isolated abdominal pain is not pathognomonic for TT and also occurs with TAT and EO patients.[8,16,28,40] The retrospective review by Mushtaq et al[16] reported complaints of abdominal pain in 9% of TAT patients, 28% of TT patients and 21% of EO patients. Mäkelä et al[8] reported that 7% to 8% of boys complained of abdominal pain in each 3 groups of TT, TAT, and EO.

Diagnostic Accuracy Also Poor for Other Causes of Scrotal Pain

It should also be pointed out that the overlap in historical and physical examination findings affects the diagnostic accuracy of the other common conditions presenting with scrotal pain. In the series by Mushtaq et al,[16] TT was misdiagnosed in 12.5% of cases, TAT in 17%, and EO in 44%. Or conversely, the history and clinical examination provided the correct diagnosis in only 87.5%, 83%, and 56% of boys, respectively. Lewis et al[7] reported that 17 (7%) of 233 patients presenting with TAT, EO, and TT were initially misdiagnosed. Of these patients, 4 (11%) of 37 patients with TT were missed at the time of initial presentation.[7] In a 2010 article by Soccorso et al,[41] the outcome of their policy of early scrotal exploration to minimize testicular loss was described. Only 3 of 138 boys were treated conservatively and 135 underwent scrotal exploration. The 135 boys who underwent scrotal exploration were divided into 3 groups depending on the suspected diagnosis. Of the 47 boys with suspected TAT, 7 (15%) had a TT. The second group included 46 boys who were felt to be more consistent with TT. In this group, 13 (28%) had TAT. The last group was 42 boys in whom the surgeons could not make a preoperative definitive diagnosis. In that group, 93% were TAT cases and 3 (7%) were TT cases. The authors concluded that surgical exploration in all cases of pediatric acute scrotum offers an accurate diagnosis and treatment and minimizes the risk of testicular loss.[41]

Myth 3: Testicular Torsions That Present After 6 Hours Are Not Salvageable and No Longer Need to Be Evaluated in a Timely Manner

Just as there are misconceptions about the reliability of the physical examination, the issue of testicle survival is more complicated than what is frequently stated. It is a commonly held belief that a testicle torsed longer than 6 hours is outside the timeframe for survival. Consequently, patients who arrive more than 6 hours from the onset time of pain are potentially managed less urgently because of the belief that the testicle is no longer viable. The perpetuation of this myth is putting potentially viable testicles at risk.

Although dead testicles have been noted at surgery and atrophy has been observed at follow-up when symptoms have been present for less than 6 hours,[10,35,39] paper after paper and series after series describe longer periods after which significant percentages of testicles have gone on to survival.[7,10,14,16,17,19,26,35,39,42–47] Almost all of the larger series describe patients whose testicles survive 12 to 24 hours of torsion or longer. Lewis et al[7] reported that the salvage success rate was 50% among patients who presented between 6 and 48 hours after pain onset. In the large series by Anderson and Williamson,[39] there were 222 normal testicles at follow-up and 4 nonviable tests in patients with symptoms between 0 and 6 hours. If the duration of symptoms was 7 to 12 hours, 10 testicles were nonviable, 88 testicles were normal at follow-up, and 1 testicle was atrophic. For symptom duration of 13 to 18 hours, 8 testicles were nonviable, 16 testicles were normal at follow-up, and 10 testicles were atrophic. Of 58 patients with symptoms for 19 to 24 hours, 30 testicles were nonviable, 18 testicles were normal, and 10 testicles were atrophic at follow-up. Of the 62 patients with symptoms for 25 to 48 hours, 46 testicles were nonviable, 3 testicles were normal, and 13 testicles were atrophic at follow-up. Importantly, for the 152 patients who were symptomatic for more than 48 hours, 4 testicles were normal and 8 testicles were atrophic, and the remaining 140 testicles were nonviable.[39] Mushtaq et al[16] reported a viable testis in 5 of 14 patients who were symptomatic for more than 12 hours (36% salvage rate) and in 2 of 7 patients who were symptomatic for more than 24 hours (22% salvage rate). Corbett and Simpson[13] described 23 patients in their series with confirmed TT. All patients presenting after less than 12 hours had testicular survival as did 2 of 3 patients who presented at 12 to 20 hours and 1 of 2 who presented at 24 hours.[13] Taskinen et al[48] described the course of 17 patients with TT. Six of 17 patients underwent orchiectomy and detorsion, and orchiopexy was performed in 11 patients. The detorsion group had symptoms for 15 hours (range, 6–168 hours), and the orchiectomy group had symptoms an average of 42 hours (range, 24–96 hours). In 1983, Scott et al[49] reported on a series of 27 patients with TT.[49] In that series, most of the testes that were torsed for 12 hours or less were salvaged. The 5 testicles removed by orchiectomy were torsed for more than 24 hours. Atrophy was more common in the testicles salvaged after the longest period of torsion. Jones et al[50] published a review of all TT cases in the armed services of the United Kingdom between 1972 and 1983. That article described a survival rate of 46% in patients who presented with pain greater than 24 hours.[50] Sessions et al[35] described their experience with 200 patients. The symptom duration before presentation for evaluation in all surviving testicles was 0.5 hours to 6 days (median, 5 hours).[35] Klingerman and Nourse[43] reported that their longest interval to survival was 48 hours. Cavusoglu et al[47] reported that the mean duration of pain at presentation was 1.35 days (range, 12 hours to 3 days) when the testis was salvaged by detorsion. Arce et al[44] reported on a small series in which the testicles of all 6 patients survived. One patient had a torsion of 540 degrees for 18 hours and another 360 degrees for 12 to 14 hours. In another small series of 33 patients with TT described by Hegarty et al,[45] 6 of his patients with pain for more than 24 hours had viable testes. However, 2 of these patients had subsequent testicular atrophy. Bayne et al[46] reported that

mean pain symptom duration in boys who were transferred but subsequently did not undergo orchiectomy was 9.8 hours. Lyronis et al[17] reported that the mean duration of pain at presentation was 11.4 hours for patients who had successful detorsion. Kaye et al[51] reported that in boys whom the torsed testis was salvaged, the pain was present for an average of 20.5 hours (range, 2–96 hours). These being the mean than many of their patients with surviving testicles were beyond the 6-hour timeframe. Barbalias and Liatsikos[52] reported a patient whose testicle survived after 7 days of torsion. Shukla et al[53] reported that 10 of 13 testicles were salvaged after 13 to 24 hours of symptoms and 3 of 8 testicles with symptoms of 25 to 48 hours in duration. Visser and Heyns[54] calculated the early salvage rate (testis viable at exploration) and late atrophy rate in 2 meta-analyses of 1140 patients in 22 series and 535 patients in 8 series, respectively. Their meta-analysis clearly documented much higher percentages of survival beyond 6 hours than commonly recognized and also confirmed that longer survival was more commonly associated with testicular atrophy. Finally, several authors have taken a strong stance on this topic. Lewis et al[7] state that a testis should not be presumed necrotic and unsalvageable if less than 48 hours have elapsed since the onset of symptoms. Waldert et al[26] expressed their disagreement with studies that consider urgent exploration as unnecessary when pain has been present for more than 24 hours. In their study, the salvaged testicles had a median duration of symptoms of 4 hours, but the range was 1 to 168 hours of pain.[26]

These reports and others provide undeniable evidence that a significant number of testicles remain viable hours beyond the 6-hour limit. The explanations vary and include thickness of the spermatic cord, degrees of twisting, and the persistence of blood flow to the testicle in some patients.[55] Visser and Heyns[54] concluded that the two most important factors determining testicular salvage after torsion were the duration and the degree of testicular rotation. The degrees of twisting or torsion most assuredly contribute to the loss of blood flow to the testicle, and therefore, not all torsions are the same.[35,44] Waldert et al[26] did color Doppler scans on 100% of their patients presenting with scrotal pain. Two of 62 patients with TT were missed. The 2 TT patients had a misdiagnosis of EO and both had a 90-degree torsion with residual arterial blood flow but no venous drainage.[26] However, it seems that even testicles with larger degrees of torsion have survived relatively unscathed. Karmazyn et al[12] described the degrees of torsion in their 33 patients with TT.[12] The torsion was complete in 21 (360–1440 degrees) and partial in 12 (90 degrees in 1 child and 180 degrees in 11). Cass et al[28] reported 9 patients with symptoms of 6 to 11 hours who were apparently successfully reduced (6 were lost to follow-up) and 5 patients (3 were lost to follow-up) with symptoms of 12 to 23 hours. The recorded degrees of twist in the first group was 360, 360, 180, 360, 720, and 900 degrees. The degrees of twist in the second group (12–23 hours) was 360 degrees in 2 patients, found reduced in 2 patients, and not recorded in the others.[28] Elsaharty et al[56] reported on the association of scrotal trauma and TT. In that small case series, prolonged testicular pain after trauma were successfully reduced in 3 of 4 patients. The successful detorsion occurred after 3 days (360 degrees), 2 days (720 degrees), and 24 hours (spontaneous resolution with anesthesia).[56] The presence of continued testicular blood flow in some patients with TT is well documented in the literature. Consequently, the presence of testicular flow does not definitely exclude TT.[7,11,56–59] In a 2004 article, Kalfa et al[59] reported that the diagnosis could not be established with color Doppler ultrasound (CDS) in 13 (30%) of 44 cases because intratesticular perfusion was still present in 10 cases. In a 2007 multicenter study, Kalfa et al[60] reported that, in 50 (24%) of 208 cases, the testicular vascularization was judged as normal or increased compared with the other testis resulting in the failure of CDS to establish the diagnosis of spermatic cord torsion. This persistence of blood flow explains why some testicles have survived for days after the initial torsion event. It also explains why misdiagnosis or delays in surgical management of TT patients have occurred when Doppler ultrasound studies demonstrate blood flow to the testicle.[7,11,32,36,56,58] Venous obstruction is the first hemodynamic consequence of TT, and obstruction of arterial inflow follows with the end result of testicular ischemia. The viability of the testes partly depends on the number of twists or the degree of turning of the spermatic cord. One study looked at color Doppler sonographic findings at different degrees of spermatic cord torsion in an animal model.[61] Torsion of the spermatic cord was created in 5 dogs by exposing and rotating the ipsilateral testis 0, 180, 270, 360, 450, and 540 degrees. Flow became undetectable at 450 degrees of rotation in 4 animals and at 540 degrees of torsion in the fifth animal.[61] Sanelli et al[62] described a 12-year-old boy in which color Doppler flow was readily detected bilaterally and was relatively symmetric. Pulsed Doppler sampling revealed asymmetric high impedance flow and a lobular appearance of the left spermatic cord with a coiled configuration of the vessels. Surgical exploration demonstrated a 360-degree torsion of the left spermatic cord.[62] The study of Karmazyn et al[12] reported normal or increased flow on ultrasound in 10 children with TT. Three of these had testicular exploration delayed more than 12 hours, and the spermatic cord was twisted from 540 to 1440 degrees. In the study by Taskinen et al,[48] preoperative color Doppler ultrasonography showed some circulation in 40% of the patients with TT. Bentley et al[55] described a series of patients with spermatic cord torsion and preserved testis perfusion. In their small series of 4 patients who presented with TT and preserved perfusion, the testicles were torsed 180, 360, 540, and 720 degrees. One of these patients who was initially missed (180 degrees) returned 4 days later and had a successful detorsion.[55]

Myth 4: Color Doppler Ultrasound Is a Consistently Reliable Tool for Confirming the Diagnosis of Testicular Torsion

Reliance on the history and physical examination alone is hazardous and the inaccuracy of those elements has been well documented now for decades. Concurrently, although the accuracy of imaging is quite good, it is also well documented to have a degree of error and inaccuracy.[7,29,59,60,63] The failure of both history and physical examination and color Doppler ultrasound to definitively make the diagnosis in significant percentages of patients is demonstrated in the 2007 multicenter study by Kalfa et al.[60] In that study, 208 patients had spermatic cord torsion proven at surgery. However, the clinical diagnosis of TT before any ultrasonographic examination was judged as highly probable in 78.5% of the cases, possible in 10.2%, and unlikely in 11.3% of these torsed patients. Moreover, CDS failed to establish the diagnosis of spermatic cord torsion in 50 cases (24%) because the testicular vascularization was judged as normal or increased compared with the other testis.[60] In a study published in 2005, Lam et al[63] expressed high confidence in color Doppler ultrasound for the diagnosis of TT. Yet, in that large series, 323 patients had an initial negative ultrasound finding, but 29 were explored eventually on clinical indications. Four of these patients (1.2% of 323) were diagnosed intraoperatively as TT.

The most important finding on ultrasound seems to be the identification of the torsion knot in the spermatic cord.[44,64–67]

Although direct visualization of the twisted cord with high-resolution ultrasonography with a probe frequency of 10 to 12 MHz seems to be a much more reliable indicator of the diagnosis of spermatic cord torsion, it too will not be diagnostic in a small percentage.[59,60] In the large study by Kalfa et al,[60] high-resolution ultrasonography detected the twist as a snail shell-shaped mass, measuring 7 to 33 mm, in 199 (96%) of 208 TT patients. This was contrasted against color Doppler ultrasound that identified only 76% of the TT patients.

DISCUSSION

The "Classic" Case

Is there a category of patient presenting with an acute scrotum that seems diagnostically obvious for EO and can be managed without imaging? Even the immediate exploration policy (without imaging) described in the older studies did not result in every patient with testicular pain going to surgery. Despite an aggressive, immediate exploration policy, Cass et al[28] reported that not every acute scrotum was surgically explored. Those cases not explored had admission findings "diagnostic of acute epididymitis, that is a swollen tender tail of the epididymis with a normal size, non-tender testis and had a clinical course typical of acute epididymitis."[28] In this 1980 study, in which Doppler, ultrasound, and isotope scanning were not performed, they explored twice as many patients with acute epididymitis as patients with acute torsion of the testis or its appendages.[28] However, it is most likely that this is the same category of patients where the correct diagnosis is being missed and surgical delays are occurring.[2] Another large series describing 209 consecutive emergency scrotal explorations seems to confirm this perspective. It reported that 5 (6%) of 82 cases of confirmed TT that the surgeon did not consider this the most likely diagnosis. And, the conclusion of the authors was, "In the absence of supportive radiological investigations, a small but significant number of twisted testes will be missed if conservative management is adopted."[14] If imaging is not performed on every patient with a painful scrotum, one is gambling. Even if the criteria for timely consultation and evaluation for surgical exploration are broadened and consistently accomplished, it does not seem to matter whether you are an emergency medicine physician or urologist; a small but significant number of twisted testes will be missed.[2,3,6,9–14,16]

The Older Patient

It is well documented that TT occurs most frequently in teenagers and young adults and the frequency of epididymitis is greater in the older, sexually active male. In that context, is it safer to make the diagnosis purely on clinical grounds in the older, sexually active patient? However, the increased incidence of epididymitis and the decreased frequency of TT may result in a relatively greater risk of diagnostic error in the older male. One study reported that the odds of having an orchiectomy actually double for each 10-year increase in age.[4] In the study of closed malpractice claims, the mean age of patients in that series was 24.3 years and included 4 patients older than 40 years.[2] This review of the literature could find no evidence that the increased incidence of orchiectomy in older males was related to more frequent misdiagnoses. Another study demonstrated a greater incidence of orchiectomy for the older age groups and the older patients trended toward presenting later (however, not statistically different).[68] The difference in that study was presumed to be greater degrees of observed cord twisting in older patients, a mean of 585 degrees in the adults versus 431 degrees in the younger age group.[68]

Diagnostic Interdependence

Although the history and physical examination are not reliable, they cannot and should not be discarded. Instead, a diagnostic triad that includes imaging is a necessity.[69] Cautious, methodical and thorough evaluations using all 3 diagnostic tools are mandatory. Even then, it is documented that there will be patients who present with mild pain, few associated symptoms, a relatively normal examination and apparent blood flow on color Doppler sonography who have an intermittent torsion or less than 360 degrees of torsion.[25,32,55] The bottom line is that the standard of care should be a timely color Doppler ultrasound or, if Doppler is not available, radionuclide testicular scan of any patient who presents to the emergency department complaining of scrotal or testicular pain and the history and examination is not consistent with TT.[70–72] Unfortunately, although both the current imaging modalities have some degree of diagnostic error, either imaging or timely surgical exploration are our remaining diagnostic options.[26,59,60,63,73–75] If after diagnostic steps are accomplished and the diagnosis remains unclear, a concurrent consultation with a urology colleague to share in the decision making should be accomplished. If based on the history and examination the potential risk of TT remains, surgical exploration may be appropriate despite apparently normal imaging studies.[29,55] The bottom line is that the inherent challenges with making the diagnosis of TT are now well documented and it is time to stop taking diagnostic gambles with the acute scrotum.

Review Limitations

Most studies presented in this review are retrospective chart reviews. Consequently, some of the information reported is dependent on what was and was not documented in the medical records. Nevertheless, the author feels that the validity of the recommendations made in this article can be sufficiently supported by the weight of multiple clinical case reports and large case series that present consistent and similar findings. The evidence that the misdiagnosis of TT continues unabated despite advances in the diagnostic tools available to the clinician is well documented.

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