Testicular ascent as a mechanism for intra-abdominal torsion

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Testicular ascent, while uncommon, can occur. A testicle that has ascended out of the scrotum can torsse and may present as an acute inguinal mass or acute abdomen. Testicle ascent can occur even if previous intra-scrotal location has been documented.

Case report

A 16-year-old Caucasian male presented to the emergency department with a chief complaint of a right groin lump. His caregiver stated that he had first noticed the mass earlier that morning and that it had since enlarged and become red and painful. Review of symptoms was positive for mild gastrointestinal complaints lasting several days. The patient’s past medical history was significant for descended testicles bilaterally, mental retardation, cerebral palsy, neuromuscular scoliosis, posterior spinal fusion, seizure disorder, and gastroesophageal reflux disease. He was taking Lamictal, Topamax, and Ativan.

Physical examination revealed mild tachycardia and an 8 x 6-cm tender, erythematous mass in the right groin. Laboratory studies were significant for a leukocytosis. A CT scan of the abdomen and pelvis revealed bilateral undescended testicles. The right testicle was enlarged and had an enhancing rim and associated fat stranding, suspicious for torsion. Additional findings included a 2-cm walled-off fluid collection within the lower right inguinal canal and a small amount of free pelvic fluid. A scrotal ultrasound scan revealed absence of blood flow to the right testicle. The patient subsequently underwent a right orchiectomy and left orchiopexy. Intraoperative findings revealed 720 degrees of right spermatic cord torsion with a necrotic right testicle that did not “pink up” upon untwisting. There was also a right hernia sac devoid of bowel that was repaired with a high-ligation herniorrhaphy. The final pathologic diagnosis was hemorrhagic infarction of the right testicle, consistent with the effects of torsion.

Figure 1. 16-year-old male with testicular ascent. Both testicles are in their respective inguinal canals, with enlargement and rim enhancement of right testicle (white arrow). Note also fat stranding in the right inguinal canal and anterior subcutaneous tissues, and a small amount of free fluid in the pelvis (white dot).
The annual incidence of testicular torsion in males younger than 25 is approximately 1/4000. Approximately 90% of cases result from intravaginal torsion, in which the tunica vaginalis covers not only the testicle and epididymis but also part of the spermatic cord (1). The resulting “bell-clapper deformity” allows the testicle to rotate freely within the tunica vaginalis, predisposing to torsion. Of these cases, 65% occur in males between the ages of 12 and 18 (1). The remaining cases are due to trauma, testicular tumors, testicles with a horizontal lie, cryptorchidism, and spermatic cords with a long intrascrotal portion.

Torsion of the spermatic cord initially obstructs venous return. Testicular ischemia results as venous and arterial pressures equalize, subsequently compromising arterial flow. The degree of ischemia depends directly on both the duration and degree of rotation of the spermatic cord. One study quoted testicular salvage rates of 90%, 50%, and 10% if detorsion occurred less than 6 hours, 12 to 24 hours, and more than 24 hours after the onset of symptoms, respectively (1).

The differential diagnosis of acute scrotal pain includes testicular torsion, trauma, epididymitis, orchitis, incarcerated inguinal hernia, varicocele, idiopathic scrotal edema, and torsion of the appendix testis. Physical exam findings for testicular torsion may include scrotal edema and tenderness, enlargement of the involved testicle, and malposition of the epididymis. The most sensitive finding is the absence of the cremasteric reflex, in which the testicle fails to elevate at least 0.5 cm in response to stroking the ipsilateral medial thigh. This finding has been found to be 99% sensitive for testicular torsion in males older than 30 months (1).
Immediate surgical exploration is performed when clinical history and physical examination findings are unequivocal and/or imaging is not available expeditiously [2, 3]. Radiographic modalities commonly used to investigate testicular torsion include Doppler ultrasonography and radionuclide imaging; they are recommended only if clinical suspicion is low, as surgical exploration remains the preferred method of investigation in probable cases. Doppler findings include decreased or absent blood flow to the affected testicle as compared to the normal testicle. Echogenicity of the torsed testicle may initially be decreased as a result of edema but may appear increased after infarction has occurred. False-negative results may be due to intermittent torsion or early torsion when arterial inflow is still preserved. The sensitivity and specificity have both been found to be about 90% [1], but may be increased with the use of spectral waveform analysis and if the whirlpool sign is observed by the sonographer [2]. Ultrasonography may also reveal characteristic findings in other testicular diseases, such as orchitis, epididymitis, hydrocele, and tumor. Scintigraphy reveals decreased uptake by the torsed testicle as compared to the normal testicle. The sensitivity is estimated to be approximately 100% [1], but radionuclide testing takes longer to perform and is thus not the initial imaging study of choice to evaluate for torsion.

Torsion of the cryptorchid testicle

Primary cryptorchidism is the failure of one or both testicles to attain an intra-scrotal position by birth. It is estimated that 33% of premature males and 3% to 5% of term males will have at least one undescended testicle at birth. By three months of age, the prevalence is reduced to 0.8%, with no further spontaneous decrease throughout childhood [4].

The literature contains several case reports of torsion of a cryptorchid testicle, and of these reports, many have been associated with a coexistent testicular tumor. One article stated that as of 1997, there had been 39 case reports of intra-abdominal testicular torsion in neonatal patients, and nearly 60% of these cases were associated with malignancy [5]. Two case reports of adolescent males were associated with nonneoplastic history [6, 7], while several reports among young adult males were all associated with testicular malignancy; seminoma was the most common histologic type [8, 9, 10, 11]. One retrospective study investigated the outcomes of 11 patients who were diagnosed with torsion of a cryptorchid testicle between the years of 1984 and 2004. Patients ranged from 1 month to 18 years, with a median age of 7.5 months. Approximately 75% of cases involved the left testicle, and clinical signs included inguinal swelling, erythema, and tenderness as well as an "empty" hemiscrotum. Three cases were managed by orchiectomy; six cases resulted in subsequent "vanishing" of the affected testicle, and one case resulted in a normal testicle 21 years postoperatively (the remaining case was lost to follow-up) [12].

Testicular ascent

A review of our patients' medical charts revealed multiple instances of documented descent of both testicles by physicians after one year of age. While the reliability of previous exams cannot be established definitively, this does suggest the diagnosis of secondary cryptorchidism. Secondary cryptorchidism, or testicular ascent, is used to describe testicles that had been in correct scrotal position but at a later date could not be manipulated into that position. The notion of testicular ascent is well supported by various surgical statistics. Orchiopexy rates are estimated at 2% to 3% of all males up to the age 17 despite a cryptorchidism rate of only 1% at 1 year [13]. Furthermore, at one institution, 40% of orchiopexy procedures were performed for boys greater than 4 years of age, suggesting either failure of screening or an even higher incidence of acquired cryptorchidism than expected [14]. One study found that those undergoing orchiopexy who had previously documented normal testicular position ranged from 2% to 20% [13]. The incidence of testicular ascent has been estimated at 2% in cases where normal position was previously documented, with a mean age at diagnosis of 7 years [13]. It tends to be a unilateral phenomenon, with the affected testicle residing prescrotal/distal to the external inguinal ring. The etiology is unknown, but leading theories include cremaster spasticity (the cremaster muscle contracts in response to circulating androgens in an indirect fashion) as well as relative cranial growth of the patient. The presence of a persistent processus vaginalis has been found in approximately half of documented cases [13]. The pathology of the ascended testicle consists of germ cells that are not normal but less severely deranged than those found in primary cryptorchidism [13, 14], while other parameters of testicular viability suggest similar effects on fertility, as seen in primary cryptorchidism [15]. Treatment consists primarily of orchiopexy, as only one-third of these patients exhibit a sustained response to hormonal therapy [13, 14].

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