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REVIEW ARTICLE

Torsion of spermatic cord in children: A review

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Abstract The current opinion on spermatic cord torsion is discussed in this review, with special attention to natural history, value of diagnostic tools, evidence for surgical management, outcome and management of atypical forms of torsion.

Introduction

Acute spermatic cord torsion, more commonly and not so exactly named testicular torsion (TT), is one of the rare true emergencies in pediatric urology. Although there is no doubt about the necessity to restore impaired blood flow to the testis, there is still much debate with regard to the most effective diagnostic algorithm, indications for emergency surgery, management of the reperfused testis and management of less typical forms of TT (intermittent, late diagnosed and neonatal TT). We reviewed the current literature in an attempt to delineate an evidence-based strategy.

Incidence and pathophysiology

Testicular torsion occurs in approximately 1 in every 4000 men under the age of 25 years. The reported rate of TT in syndrome of the acute scrotum is variable. The most reliable data come from studies in which all children with symptoms of acute scrotum were indicated for surgical revision. In these, torsion of the appendix testis was the most common pathology (45—57%), followed by torsion of the spermatic cord (26—27%), and epididymitis as a much less common condition (only 10—11%) [1,2].

TT can be encountered at any age, but most commonly during adolescence and the neonatal period. Adolescent TT

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is intravaginal. A long mesorchium allowing for greater mobility of the testis which hangs freely within the tunica space (bell clapper deformity) is believed to be the principal predisposing condition. This anatomical anomaly is nearly always bilateral. Hayn et al. found this abnormality in 47 boys who were operated upon for spermatic cord torsion or intermittent cord torsion in all cases on the affected side and contralaterally in 88% and 90%, respectively [3]. An autopsy study performed by Caesar et al. [4] revealed bell clapper deformity in 12% of the male population. As the incidence of spermatic cord torsion is much lower, an inciting event must occur. The sudden contraction of the cremasteric muscle, which is attached onto the cord in a spiral fashion, as a response to mechanical, sexual or thermic stimulation (e.g. a jump into cold water) may cause a rotational effect on the testis as it is pulled upward [5]. Perinatally, the mechanism of torsion is different. Absence of normal fixation between testicular coverings and tunica dartos results in abnormal mobility in the scrotum and extravaginal torsion.

The primary consequence of TT is ischemia of the testis due to venous and subsequent arterial occlusion followed by reperfusion injury upon repair. Hemorrhagic necrosis and cell apoptosis lead to the accumulation of testicular neutrophils (inflammatory response) and increased testicular oxidative stress. According to experimental animal studies, germ cells are much more vulnerable than Sertoli cells and spermatogenesis can be eliminated despite returned blood flow and preserved Sertoli cell function [6]. However, the exact mechanism of cellular injury remains only partially understood.

The two most important factors determining testicular damage are the length of time from onset of symptoms to cord detorsion, and the degree of cord twist. It is commonly accepted that salvage of the human testis declines sharply with a symptom duration greater than 6 h [7]. However, necrosis is possible as early as within 4 h where a high degree of twisting exists [8]. In animal studies, blood flow changes in testicular tissue were insignificant during torsion and after detorsion if the cord was twisted to 360° or less. In the 720-° torsion group, blood flow to the twisted testis was reduced significantly [9]. Tryfonas et al. found complete or severe atrophy in all patients with torsion of more than 360° and a symptom duration of more than 24 h [10].

Salvage rate of testes after detorsion is reported as 62—85% for descended testes and 29—40% for undescended testes [11]. The majority of authors report the testis as 'salvaged' if a palpable mass with Doppler signs of perfusion is preserved. However, this does not correspond to salvaged testicular function. Although, duration of ischemia correlates well with testicular atrophy, there is no clear correlation with semen quality. Semen analysis is normal in only 5—50% of patients [12], indicating alteration of the contralateral testicle. There are many theories explaining this 'collateral damage'. The theory of an autoimmune process triggered by the rupture of the hematotesticular barrier leading to formation of anti-sperm antibodies has never been confirmed. Evidence against an autoimmune reaction is found in the article by Anderson et al. [13], who did not detect any cases of anti-sperm antibodies after TT. Hagen et al. [14] found pre-existing dysplastic changes in the contralateral testicle in 30 of 34 patients (desquamation of the germinative epithelium, atrophy of the Leydig cells and malformation of spermatoblasts). This may represent consequences of reperfusion injury episodes during previous asymptomatic intermittent torsion or congenital abnormality. Pre-existing contralateral abnormalities were verified by Anderson et al. [13]. Altered microcirculation of the opposite testis due to vasospasm caused by a sympathetic mediated reflex may affect testicular function and ultimately fertility as well [15].

Incidence and pathophysiology

- Torsion of the spermatic cord represents 26—27% of children with acute scrotum.
- Two most important factors determining testicular damage are time from onset of symptoms to cord detorsion and degree of cord twist.
- Poor semen quality after testicular torsion may represent consequences of previous symptomatic intermittent torsion or congenital abnormality.

Diagnosis

History and clinical findings

The goal of diagnosis is to identify boys with TT as soon as possible. Rapid diagnosis shortens the duration of ischemia; correct diagnosis lowers the incidence of unnecessary surgery. Acute and rapidly increasing pain is the most common symptom. It may be localized not only in the scrotum but also into the groin and lower abdomen, imitating an acute abdominal event. It may occur during sleep and arousal of the patient; it may follow direct trauma (in 4—8% of cases), increased physical activity or sudden change of temperature (dive into cold water). Some patients refer to intermittent acute pain, uni- or bilateral, with spontaneous resolution in their history. Vegetative symptoms like nausea, vomiting, paralytic ileus, lethargy may be encountered. In neonates, signs of restlessness may be associated with the torsion, but usually the first sign is a scrotum of abnormal appearance during delivery or diaper changing.

On early examination, painful palpation and scrotal edema are found in 88% and 44% of patients, respectively [2]. Skin redness and elevated body temperature are usually absent. Painful enlarged testicle and epididymis are typical early signs; the testicle is placed more cranially due to the shortening of the cord. After several hours the scrotal skin becomes red; the massive local swelling makes clinical examination difficult, sometimes nearly impossible. Clinical examination of the abdomen is normal. Cremasteric reflex is a very useful diagnostic tool. Rabinowitz [16] described 100% correlation between the presence of the ipsilateral cremasteric reflex and the absence of testicular torsion during a 7-year evaluation of 245 boys with acute scrotal swelling. Later, one case of preserved cremasteric