

## Case Report

# Testicular torsion: a scrotal catastrophe

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### ABSTRACT

Testicular torsion is a serious scrotal emergency having a negative impact on fertility and in its most severe presentation, there is potential loss of the testicle if not diagnosed early. The condition needs to be diagnosed promptly with immediate surgical intervention. Intermittent testicular torsion (ITT) is a forerunner or a red flag for an impending frank testicular torsion. ITT is characterized by sudden onset of testicular pain which may resolve spontaneously before further investigation and treatment. Testicular torsion in adults is usually intravaginal in location and can be diagnosed clinically if the patient presents early with typical clinical signs. A case of ITT who presented with frank unilateral testicular torsion diagnosed clinically and surgically treated with salvage of the affected testes is presented to highlight the importance of history of ITT and typical clinical features. The anatomical aspects and pathophysiology of testicular torsion including the aftermath is discussed. ITT is a forerunner to frank testicular torsion. If offered prophylactic orchidopexy then a frank episode of testicular torsion with all its sequelae can be averted.

**Keywords:** Intermittent, Testicular, Torsion, Diagnosis, Treatment, Complications

### INTRODUCTION

The incidence of testicular torsion in adults is 4.5 in 100,000 males.<sup>1</sup> It is a condition affecting young males. Over 50% of patients with testicular torsion will have had a prior episode of testicular pain due to intermittent testicular torsion (ITT).<sup>1</sup> Intermittent testicular torsion (ITT) in adults usually is ignored and goes unreported.<sup>1</sup> Failure to recognize the existence of ITT can lead to repeated attacks of scrotal pain, swelling and possibly loss of the testicle.<sup>2,3</sup> A case of episodic intermittent testicular torsion presenting eventually with frank testicular torsion is presented along with a brief review of literature.

### CASE REPORT

A 30 year old young male presented with sudden onset of severe, excruciating, right sided scrotal pain referred to

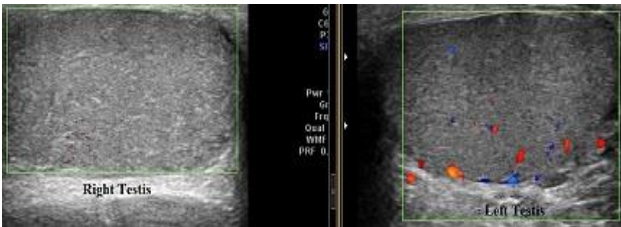
the right lower abdomen. There was no history of difficulty in micturition, dysuria, fever or trauma to the scrotum. However the patient gave a history of intermittent attacks of right sided scrotal pain which resolved spontaneously. Two of the seven episodes of ITT were significant enough that the patient sought medical advice. However investigations revealed normalcy of the testis at the time of evaluation.

On examination the vital parameters were within normal limits. Local examination of the scrotum revealed redness and edema of the right hemi scrotum with the right testis lying higher up in a more horizontal position as compared to the left (Figure 1). The right hemiscrotum was extremely tender precluding further physical examination of the scrotum. Examination of the abdomen did not reveal any positive findings. Patient was immediately investigated. A complete haemogram, renal profile and urine examination was normal. A doppler ultrasound of

the scrotum revealed complete absence of blood supply to the right testis with a secondary hydrocele. The left testis was normal (Figure 2).



**Figure 1: Right testicular torsion with the affected testis lying higher and more horizontal.**



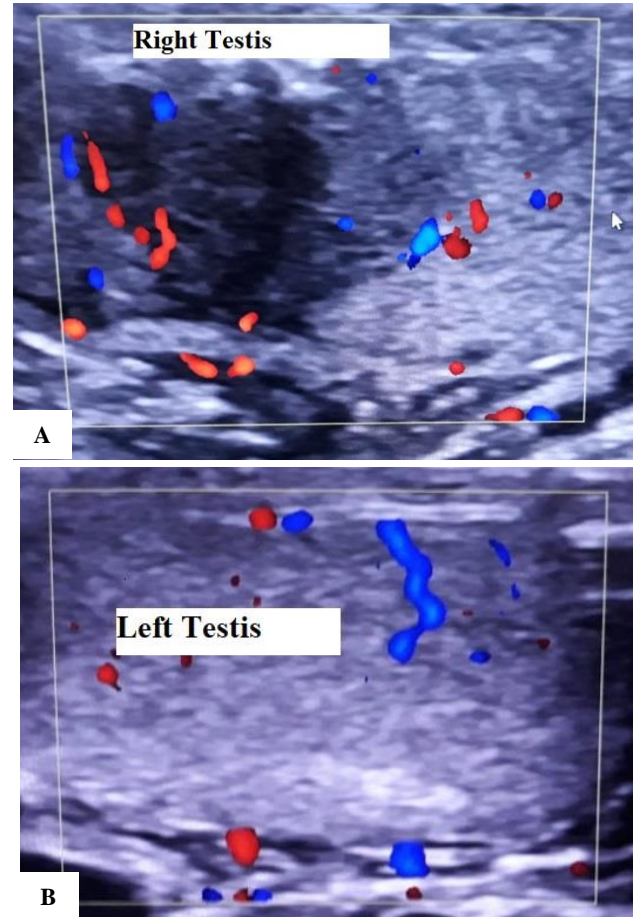
**Figure 2: Preoperative Doppler ultrasound of both the testes showing absence of vascularity of the right side.**



**Figure 3: Intraoperative photo showing the twist marked by the black arrow.**

Ultrasound of the abdomen was normal. Patient was taken up for surgical exploration of the scrotum under regional anesthesia. The right testis had undergone

torsion with 3 twists amounting to 540 degrees (Figure 3). The color of the testis was dusky and edematous. Surgical detorsion or unwinding was done, which led to improvement in the appearance of the testis. A four point fixation was done using interrupted sutures of 4-0 proline. The contralateral testis was also fixed in the same session. Scrotal skin suture removal was done on day 10. A repeat doppler ultrasound was done which confirmed the presence of normal blood supply to the right testis (Figure 4 A and B). There is no testicular atrophy thereafter.



**Figure 4: (A) Postoperative Doppler ultrasound of the right testis showing normal vascularity; (B) postoperative Doppler ultrasound of the left testis showing normal vascularity.**

## DISCUSSION

Testicular torsion is an acute surgical emergency usually encountered in children. However it is also seen in young males. Various anatomical variations of the tunica vaginalis predispose to torsion in a fully descended adult testis.<sup>1,4</sup> An adult testicular torsion is usually intravaginal in location and is attributed to a high insertion of the tunica vaginalis around the cord or a long mesoorchium. In the pediatric age group the torsion is usually extra vaginal in location. Irrespective of age at presentation, the chances of testicular loss is high unless diagnosed and

treated promptly. ITT may cause venous congestion with or without decreasing arterial inflow leading to significant testicular damage. It can cause atrophy of the seminiferous tubules and peritubular fibrosis leading to a decline in spermatogenesis. Clinically the patient presents with rapid onset of pain followed by complete resolution in ITT.<sup>3</sup> Physical examination will reveal a very mobile testis due to a long spermatic cord. Almost all patients with ITT will have normal doppler ultrasound findings by the time investigations are done. This leads to a delay in fixing the testis thereby preventing a severe episode of frank torsion. However scientific evidence is inadequate to support a prophylactic orchidopexy in patients presenting with ITT. In the case presented, the patient had multiple episodes of ITT before he presented with a severe episode of frank testicular torsion.

History is typical in testicular torsion with the absence of genitourinary symptoms before the onset of pain. The pain is excruciating and unremitting with poor response to analgesics. A doppler ultrasound is diagnostic as seen in the case presented. However if doppler ultrasound is unavailable, the surgeon should not wait to confirm the diagnosis, but should proceed with scrotal exploration as that is the best chance for salvage of the affected testis.<sup>5</sup> The interval between onset of symptoms and surgical intervention has an important impact on testicular salvage. If treated between 6 hours of onset of pain, 90-100% of testicles can be saved. If treated within 6-12 hours then there is a 20-50% chance of saving the testis. However if the testicular torsion is treated between 12-24 hours then the salvage rate is very poor (0-10 percent).<sup>6</sup> Doppler ultrasound of the scrotum is a diagnostic tool for testicular torsion. It will reveal reduced blood flow with altered echogenicity of the testis accompanied by detection of a spiral twist of the spermatic cord and a small secondary hydrocele.

A manual detorsion can be attempted in the emergency room. However the success rate is questionable. It is always mandatory to subject this patient to surgical exploration. Testicular torsion is an emergency necessitating early surgical exploration of the scrotum. Ischemia persisting beyond 4 hours seriously threatens the viability of the affected testis. It can also cause serious damage to the contralateral testis as well. Salvage of affected testis is better in younger patients presumably due to less twisting of the cord.<sup>6</sup>

Warm ischemia time has a serious impact within six hours of torsion. The salvage rate in undescended testis undergoing torsion is extremely poor with orchiectomy rate of 60-70% as compared with a descended testis.<sup>6</sup> At the time of scrotal exploration, manual detorsion of the testis is performed and the viability of the testis is assessed. In doubtful cases the testis can be covered with a gauze soaked in warm saline accompanied by administration of 100% oxygen. If the testis shows signs of improvement with respect to its lividity, then a four point fixation of the testis with non-absorbable suture

material is done. However if the testis is gangrenous then an orchiectomy has to be performed. Fixation of the contralateral testis has been done in the same sitting in view of the high likelihood of an episode of torsion happening by virtue of the same anatomical predisposition.

### ***Aftermath***

Unilateral testicular torsion severely interferes with subsequent spermatogenesis in over half of the patients and in another 20% of patients can cause borderline impairment.<sup>7</sup> Subfertility (sperm count less than 20 million per ml) is found in 36-39% of patients who have undergone torsion of the testis. In a long term follow up, semen analysis may be normal in 5-50% of patients. The sperm count and degree of atrophy both correlate closely with the duration of torsion. Damage to the contralateral testis in a patient who has suffered unilateral testicular torsion is a complex pathology.<sup>7</sup>

The abnormalities found in the contralateral testis are extensive apoptosis in the germinal epithelium, atrophic Leydig cells, malformation of late spermatids, fewer late spermatids and pathological changes in the Sertoli cells.

Trauma to the blood testis barrier initiated by torsion may induce the release of apoptotic activating factors (cytokines), which damage the contralateral germinal epithelium. Studies suggest that leaving behind a non-viable severely damaged testis in situ causes more damage to the contralateral testis. Sperm antibodies occur in 0-11% of patients at the time of torsion or at a later date. Hormonal function is relatively well preserved with elevated levels of LH and FSH seen only in patients with torsion persisting beyond 8 hours or those with testicular atrophy. There is 3.2 fold increased risk of developing a testicular tumor, 6-13 years after torsion.<sup>7,8</sup> Injury to the affected testis is usually caused by a combination of ischemia and reperfusion injury by reactive oxygen species which arise from the activation of xanthine oxidation system in parenchymal cells or from leukocytes that adhere to reperfusing venule walls before undergoing diapedesis into the interstitial tissues. There are several hypotheses which have been postulated to explain bilateral exocrine dysfunction after unilateral torsion. These include immunological mechanisms, previous episodes of silent intermittent torsion, congenital dysplasias, and reflex vasoconstriction.

### ***Immunologic or sympathetic orchidopathy***

The testis is an immunologically privileged site. An ischemic damage may lead to breakdown of the blood testis barrier. Antigenic material from the dying testis could be exposed to the immune system and the resultant auto anti-bodies may then affect the unaffected testis. Anti-testis auto antibodies maybe present in 13% of the patients.<sup>8</sup> However autoantibodies, do not correlate with exocrine or endocrine dysfunction. Intermittent silent

torsion can also cause significant damage to both the testes. Oligospermia after unilateral testicular torsion may be an underlying defect in both the testis. Biopsies of the contralateral testis taken at the time of exploration for torsion show evidence of pathology in 57-88% of cases. These include maturation arrest, germ cell degeneration, tubular hyalinization, immature tubules and focal thickening of basement membrane. These changes are usually seen in both the testis following episodes of ITT. Preexisting congenital dysplasias may also explain the disturbance of spermatogenesis in the contralateral testis. The anatomical abnormality predisposing to torsion may be associated with defective spermatogenesis, such as that found in unilateral cryptorchids who are often infertile and have histological deformities even in a normally descended testis as well as have a 10 fold increased risk of torsion. Surgical exploration and fixation is unlikely to be responsible for damage to the contralateral testis. In humans histopathological damage is already present at the time of exploration and several abnormalities occur in patients with or without contralateral fixation.

#### ***Reflex vasoconstriction***

Spermatic cord under distress induces sympathetically mediated reflex vasoconstriction of the contralateral blood vessels with resultant ischemic damage.<sup>7,8</sup> As contralateral testicular hypoxia is caused by torsion of the spermatic cord only, the testis and epididymis do not seem necessary for this and the testicle only under distress appears to be an important structure resulting in contralateral testicular hypoxia. Thus contralateral testicular injury from unilateral torsion is most probably caused by preexisting damage combined with hypoxia resulting from sympathetic reflex mediated vasospasm. Based on these observations, experimental treatments have been postulated to limit testicular damage after torsion. These include cooling of the testis, limiting reperfusion injury, suppressing immune mediated damage and attempting chemical sympathectomy to prevent contralateral vasoconstriction. Chemical sympathectomy works by inhibiting the afferent impulses from the ipsilateral testis after stress thereby preventing contralateral vasospasm and hypoxia.<sup>8</sup>

#### **CONCLUSION**

A patient with history of intermittent testicular torsion characterized by scrotal symptoms should be considered

for prophylactic orchidopexy. Testicular torsion is an emergency necessitating early scrotal exploration. Severe collateral damage is usually caused to the contralateral testis in a patient treated for unilateral testicular torsion leading to subfertility.

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