

Testicular torsion

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Abstract:

Testicular torsion is an easy diagnosis and yet a has a difficult and challenging outcome. The management is time sensitive and most frequently encountered in the casualty. A through history and examination along with a testicular doppler and ultrasound are vital for a diagnosis and to avoid complications. Complications may include testicular infarction, necrosis, and sub/infertility. Managing these complications are quite challenging for the doctor, patient and the attenders. This article therefore focusses on the management of testicular torsion and its complications and on how we dealt with our patients and helped them in overcoming the challenges.

1. INTRODUCTION:

Testis is formed retroperitoneally, adjacent to the kidneys. At about the third month of intrauterine life, the gubernaculum testis develops and extends from the genital tubercle to the inferior pole of the testis via the inguinal canal. The peritoneum later encircles the testis completely forming a mesentery, the mesorchium. The inferior, main part of the gubernaculum attaches to the scrotal skin pouch, and the minor, superior part disappears. (1,2)

Many congenital anatomical abnormalities of the testis and its adnexae have been considered as the factors responsible for the causation of torsion. Those include hypermobile testis, loose and abnormal connections between testis and adnexae, and polyorchidopathia. Normally, the testis is suspended in a vertical position, but testis lying along a horizontal plane has been found to increase incidence of torsion, especially subclinical intermittent torsion. Cryptorchid testes have long been recognized to cause torsion and demonstrated in experimental studies and case reports. Testicular tumor and torsion spermatocele predispose in favor of torsion. Anomalous spermatic cord attachment, bifurcation and short cord have been

hypothesized to predispose to torsion. Some of the abnormalities of tunica vaginalis like capacious tunica vaginalis, “clapper bell deformity” (faulty mesentery of the testis), and high investment of the tunica have been thought to contribute to testicular. Other factors hypothesized to increase torsion risk are a voluminous scrotum, an elongated globus minor (body of epididymis), thrombosis of the pampiniform plexus veins, excessive mobility of the vas deferens, a hyperactive cremasteric reflex, maldeveloped gubernaculum testis, vascular hamartoma, and previous scrotal surgery and orchidopexy. In addition to these predisposing factors, muscular exertion in the form of swimming, skating, sexual exertion, sudden flexing of the thighs, and weight lifting, has been reported to precipitate torsion in the young adults. A recent report suggested that persistent Mullerian duct syndrome might contribute to development of torsion. Reports have indicated that torsion is more likely to occur in cold climates especially in places with temperatures below 15C and less likely to occur in hot conditions and summer months.(3,4)

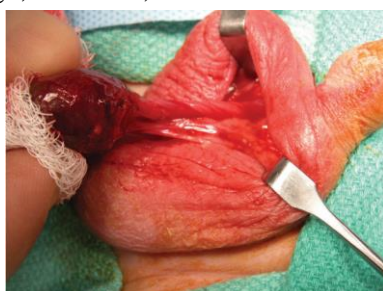
Torsion results from twisting of the spermatic cord, which causes ischemic changes such as swelling, degeneration, necrosis, and infarction, depending on the degree and duration of twisting. Torsional twisting usually occurs away from the midline, probably secondary to the direction of the cremasteric muscle fibers. The degree of the torsion varies from 180 to more than 720. This variation in torsion contributes to the variant presentations of acute torsion from severe to sub-acute and chronic torsion. Torsion blocks both arterial supply and venous drainage. This contributes to hemorrhage, edema,

ecchymosis, and cellulites. The edema further results in altered blood flow dynamics to the testis and accentuates arterial blockage, hypoxia, and gangrene of the testis. (5,6)

2. METHODOLOGY:

We did a retrospective analysis of all torsion testis cases who came to our hospital in the last 5 years and analysed the outcomes of these patients.

Patients included were diagnosed with testicular torsion irrespective of the age and reason.



Torsed testicle with visible twisting of spermatic cord and testicular necrosis.

3. RESULTS:

Parameters		no of patients
Total no of patients	55	
Age group	0-17 years	39
	18-25 years	13
	Above 25 years	3
Side	Right	15
	Left	40
Reason for testicular torsion	Tumours	1
	Congenital	49
	Injury	5
History of pain	Less than 6 hours	32
	6-12 hours	18
	Greater than 12 hours	5
Testicular scan	Torsion with intact blood supply	29
	Torsion testis with impaired blood supply	26
Surgery	Orchidopexy	27
	Orchidectomy	28

4. DISCUSSION:

Testicular torsion is an emergency condition which may lead to irreversible

ischemic injury if not treated immediately. It is because of the torsion of the spermatic cord within the tunica vaginalis in children and adolescents. It may be because of a lack of normal fixation of testis or epididymis to the muscular layers of cord or scrotum. It occurs only in about 20 - 25% of all cases of acute scrotum in children. (7)

A study reported 19 cases with median age of 10 years, in our research mean age was 15 years. The most common symptoms are pain, swelling/erythema, nausea and vomiting. The incidence of TT is more common in left side, both in our study and the literature. (5-7)

Duration of symptoms before surgery is an important predictor of outcome in TT. Studies reported that no testicle with a history longer than 12 hours could be salvaged. Some studies observed testicular necrosis, even atrophy after orchidopexy in patients with the history shorter than 10 hour. (5-7)

A few researchers have reported testicular atrophy during follow-up in 27% of patients after orchidopexy within 4 hours of onset of symptoms. In our study 35 patients had symptoms for less than 6 hours and the rest for more than 6 hours. (5-7)

Some studies have reported salvage procedures even after 24 hours of onset of symptoms. Some have reported that even after salvage procedures, patients have pain for an average of 20 hours. (7,8)

While the degree of rotation might be a determining factor for the viability of the involved testis, Previous reports suggest that there is no significant difference in testicular TT with rotation degree of 360 to those with more than 360 degree. (9,10)

Testicular torsion can be caused by high and transverse position, thickening of epididymis, retractile testis, and sometimes lack of the cremasteric reflex. It is reported that a lack of the cremasteric reflex is another feature highly associated with TT, and was effective as a predictor in half of their patients. (11,12)

Some studies have reported cases of testicular atrophy in their follow-up within

orchidopexy group, and we had no atrophy cases during the follow-up. Literature suggests a higher number of TT during colder months, with a significant increase during winter. In a recent study of 70 cases with TT, 31 (44%) were in winter season, and 22 (31%) patients referred during autumn season. This was consistent in our study too, with a majority of cases being reports during December and January. (5,6,9)

Early diagnosis and exploration can prevent medico legal risks. TT requires emergency attention in order to optimize the testicular salvage rate. Imaging/radionuclide scanning should not be a cause of delay, also ultrasonography may produce false negative results, so early surgical exploration is mandatory, and second look exploration can be more effective in salvageability. Cold season, torsion degree higher than 360° and a history longer than 12 hours were not specific signs of testicular non-viability. The school age group which is the most vulnerable one, should be informed about this, to prevent testicular damage.(1,5,8,10,11)

5. REFERENCES:

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