# TESTICULAR TORSION: SPECIFICS OF BIMODAL CLINICAL PRESENTATION, DIAGNOSIS AND TREATMENT

Zorica Jovanović (1), Andjelka Slavković (1), Goran Janković (1), Maja Zečević (8), Milan Slavković (2)

(1) CLINIC FOR PEDIATRIC SURGERY UNIVERSITY CLINICAL CENTER NIS; (2) UNIVERSITY CHILDREN'S CLINIC BELGRADE

**Abstract:** Testicular torsion (TT) is a condition caused by twisting of the spermatic cord, which leads to interruption of blood flow to the testicles. It most often occurs in two different periods of growth and development. It is significantly rarer around birth, before or immediately after, including the first month of life, and much more frequent at the age of 12-18. In addition to age, the pathoanatomical substrate, mechanism of occurrence and clinical presentation are different. In younger adolescents, it is an urgent clinical condition, while in newborns it is usually not. The aim of this paper is to point out these specifics, because the therapeutic approach depends on timely recognition, which can be essentially different. **Key words:** testis, torsion, adolescents, perinatal testicular torsion

### INTRODUCTION

Embryonic development of the testis begins very early, already in the 6th week of gestation, with condensation of the urogenital ridge tissue. In the last trimester of pregnancy, as a result of fetal growth and under the influence of endocrine stimuli, the testis leaves the abdomen and descends into the scrotum through the inguinal canal. Along the way, it is followed by the processus vaginalis, the peritoneal perversion. The testis is fixed in the scrotum by the gubernaculum. The spermatic cord into which the a.testicularis, plexus pampiniformis, and ductus deferens enter occupy an inguinal position proximal to the testis. Since time is a crucial factor in rescuing a torque testis, a good knowledge of anatomy and embryonic development is very important in understanding the pathophysiological mechanism of this condition [2-5].

## DEFINITION

Testicular torsion (TT) is a condition caused by twisting of the spermatic cord, which leads to interruption of blood flow to the testicles. Testicular tissue cannot survive without blood flow, and loss of the affected testicle can occur if not treated immediately. That is why TT is an urgent surgical condition and the most common cause of testicular loss.

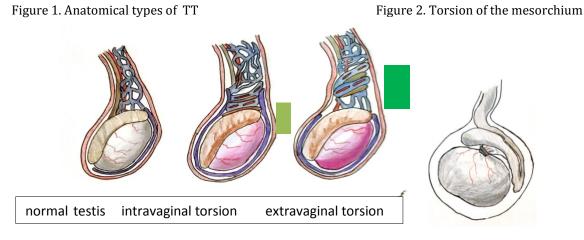
#### INCIDENCE

TT can occur at any age, but most frequently in the age group of 12 to 18 years (puberty) and in the first year of life (12%). On average, 3.8 per 100,000 men under the age of 18 have TT annually, and it is bilateral in 40% of cases [3]. Testicular torsion is the cause in 26% of acute scrotum and in 42% it ends with orchiectomy [3,6,7]. 10% of boys with confirmed TT have a positive family history [6]. The seasonal incidence of testicular torsion ranges from 36.2% in spring, 31% in winter, 19% in summer to 13.8% in autumn. In 81% of cases, torsion occurs when the atmospheric temperature is lower than 15C [7]. It can also occur after physical activity.

#### DISTRIBUTION

The distribution by age is bimodal, as is the pathoanatomical substrate. Extravaginal torsion occurs in fetuses and newborns, because the testes can rotate freely before their fixation through the tunica vaginalis inside the scrotum (Figure 1). It is usually diagnosed in the first 7-10 days of life. Normal testicular suspension ensures a firm fixation of the epididymal-testicular complex on the back and effectively prevents the twisting of the spermatic cord.

Extravaginal torsion accounts for approximately 5% of all torsions. This condition is associated with high birth weight. Bilateral perinatal torsion is rare, although an increase in the number of reported cases has been observed. Currently in the literature this number is around 5.



In adolescents and men who have high attachment of the tunica vaginalis, as well as abnormal fixation of the muscular and fascial sheath, the testis can rotate freely within the tunica vaginalis (intravaginal torsion of the testis) (Figure 1). (Bell clapper), can cause the longitudinal axis of the testis to be oriented transversely, not cephalocaudally. Testis torsion can also occur if there is an abnormal mesorchium between the testis and the epididymis, when the testicle is wider than the mesentery [8] long mesorchium (intravaginal torsion), a rare form, which may explain the ultrasound finding of epididymal hyperemia despite scanty vascular vascularization. (Figure 2)

# TT IN THE ADOLESCENT PERIOD

Testicular torsion is a clinical diagnosis. The clinical presentation of torsion in this age group is characterized by: unbearable unilateral pain in the affected testis, (more often on the left) with sudden swelling because the structures twist (like a puppet on a wire) and the testicle rises. Patients may have fever, nausea and vomiting, abdominal pain, and a history of previous testicular pain. If the patient has an acute scrotum in the clinical presentation, the main goal is to exclude testicular torsion, because it is the only urgent surgical condition [9]. Very often, boys cannot accurately and precisely explain the onset of symptoms, as well as their severityseriousness and duration. Many patients do not come immediately, after the onset of symptoms, further limiting the therapeutic space for testicular rescue [10]. In the anamnesis, some of the patients mention recent trauma or physical exertion, which preceded the difficulties [11]. Some also have nonspecific urinary disorders. High testicular position is indicative of twisted and shortened spermatic cord [12]. It is not of scrotal or testicular origin. For that reason, every adolescent who complains of abdominal pain should undergo an examination of the external genitalia in order to exclude the possibility of scrotal pathology. A retrospective analysis of 73 adolescent patients (mean age 15.3 years) who underwent surgical treatment of testicular torsion showed that patients who had abdominal pain, compared with patients with initial testicular pain, had a significant delay in diagnosis / treatment (mean pain duration of 36 hours versus 5 hours)

Figure 3. Acute scrotum - horizontal position of the right testis



and significantly higher testicular loss rate (81% vs. 4%) [13]. The results of the study suggest that every 10 minutes in delay reduces the chance of testicular survival by 4.8%.

Patients sometimes state that they have previously had similar problems of lower intensity, in terms of unilateral pain in the scrotum, which passed spontaneously in a few hours. The horizontal position of the testicles is most often caused by intermittent torsion of the testicles. The condition is more common on the left testicle. Occasional testicular pain in the presence of an abnormal testicular position should justify the diagnosis of intermittent testicular torsion. Early bilateral orchidopexy is effective. If TT is suspected on the basis of the clinical picture, early urological consultation and urgent surgical intervention are necessary, with rational application of additional diagnostic. TWIST scoring system (Testicular Workup for ischemia and Suspected torsion) was developed to determine the risk of testicular torsion on a clinical basis and reduce the need for ultrasonography. TWIST score uses urological history and physical examination to assess the risk of testicular torsion. Parameters include:

1. Swelling of the scrotum and testicles, where normal landmarks (epididymis) may not be palpable due to swelling (2 points)

2. hard testicle (2)

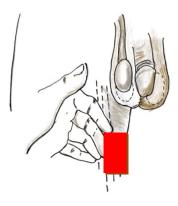
3. absent cremasteric reflex (1)

4. nausea / vomiting (1)

5. high raised testicle (1)

Patients are classified as low-risk, medium-risk, or high-risk. The TWIST score is based on the sum, ranging from 0 to 7. The results of risk stratification for those with low risk of testicular torsion are 0 to 2 points; medium risk, 3 to 4 points; and high risk, 5 to 7 points

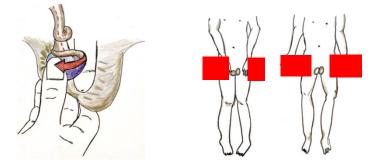
Figure 4-Cremasteric reflex



Stimulation of the inner side of the thigh stimulates the sensory fibres of the genitofemoral and ilioinguinal nerve when the cremaster muscles contract with the resulting elevation of the ipsilateral testis. (Figure 4) The consequences of testicular torsion are: loss of spermatogenesis within 4-6 hours and loss of hormonal function 10-12 hours after occlusion of testicular blood vessels. Survival of the testicles after torsion is very difficult to predict. The papers publish the survival rate if surgery is performed in the first 6 hours after the onset of symptoms 90-100%, and from 6-12 hours, it drops to 50%, and is less than 10% if the symptoms last longer than 24 hours [14]. Survival of the testes after prolonged torsion may indicate that testicular blood flow has not been completely interrupted or that it is intermittent torsion. Therefore, if there is a high degree of suspicion of testicular torsion based on the anamnesis and physical examination, imaging examinations do not have to be performed, but the patient immediately undergoes surgical intervention. Delay in additional diagnostics prolongs the time of testicular ischemia and reduces the survival rate of the testis [14]. In obscure cases Doppler echosonography, which is highly sensitive 88.8% and specific 98.8%, with only 1% false results is used [15]. Testicular torsion treatment: The definitive way of treating testicular torsion is operative, after possible manual derotation. The procedure of manual derotation of the testicles enables the relief of pain, if it is successful, but after that the renewal of the blood flow must be confirmed. Other signs that indicate successful manual detoxification are: change of testicular position from transverse to longitudinal orientation, lower testicular position in scrotum and return of normal arterial pulsations on Doppler ultrasonography. Subsequent orchidopexy is recommended to prevent recurrence of torsion.

Manual detorquation is performed in sedation, if swelling of the scrotum and testicles has not yet developed. In 2/3 of the patients the testis is torqued medially, and in 1/3 laterally. Sometimes detorquation needs to be repeated 2-3 times for complete detorsion[16,17,18]. (Figure 5)

Figure 5Figure 6Manual derotationThe direction of testicular derotation — like opening a book



For manual detorquation of the torsion of the right testicle, the doctor is placed in front of the patient who is standing or lying on his back and with the left thumb and forefinger he is holding the patient's right testicle. The doctor then rotates the right testicle outward 180 ° in the medial-lateral direction. (Fig. 5) For the patient's left testicle, the doctor uses the patient's right thumb and forefinger and rotates the patient's left testicle outward 180 ° from medial to lateral. Inward and toward the midline; therefore, manual testicular derotation involves twisting outward and laterally. (Figure 6) In one series of 104 patients, lateral rotation was found in 46% of patients, and there were no factors that would indicate the direction of torsion with certainty.

Figure 7. TT 360 degrees.



Figure 8. Detorned???, vital testis, after 4h



In older boys with TT, the contralateral bell clapper anomaly is highly prevalent, which supports the standard practice of contralateral testicular fixation in these situations. Fixation remains a matter of the surgeon's personal preference, but the use of an absorbable suture for fixation is associated with recurrence of torsion and should be avoided [20].

Figure 9. Gangrena testis-TT after 4 days



### PERINATAL TESTICULAR TORSION - PTT

Torsion of the spermatic cord of the fetus and newborn is a special and rare entity, different from the one seen in boys in juvenile and adulthood. Testicular torsion occurs prenatally (in the last trimester) or postnatally, in the first thirty days of life. Taylor 1897.g. first described neonatal extravaginal unilateral testicular torsion in the newborn. Papadatos and Moutsouris in 1967 were the first to describe bilateral testicular torsion [21]. The symptomatology is atypical and the clinical presentation is varied. 70% develop before birth and in 30% of boys after birth. The representation of parties is equal [22], and in 20% PTT is bilateral, occurring more often simultaneously, but also asynchronously [23]. Lack of consensus in diagnosis and treatment contributes to confusion and can result in numerous medical (and legal!) problems.

The etiology and pathophysiological mechanism of testicular torsion during pregnancy are unknown. It usually occurs between the 34th and 36th week of gestation, for which there is an ultrasound confirmation, although bilateral torsion has been described in the 32nd week. The incidence of in utero torsion is unknown and is significantly higher because any anarchy or vanishing testis, testicular regression syndrome (TRS), is the end result of TT. Favourable factors for the development of prenatal TT: Testicular hormone insulin-3 (Insl3) - Leydig insulin-like factor (LEY IL) - relaxin-like factor (RLF) is part of the insulin peptide family produced by Leydig cells and is critical for the development of mouse goiter. Underdeveloped gubernaculum and increased testicular mobility and Insl3 mutant mice result in sperm ribbon abnormalities, delayed / absent testicular descent and testicular torsion. 24. Other favouring factors: fetal stress, preeclampsia, gestational diabetes, twin gestation, prolonged presentation, prolonged presentation, multiparous mother, large TM relative to gestational age, presence of prenatal hydronephrosis [21] Elevated intrauterine pressure as well as pressure in the birth canal during childbirth [25] stimulates a strong cremasteric response at a time when there is no tunic - scrotum junction. Prenatal TTs occur in term boys with an average TM of 3.6 kg [26]. Events are long, and the etiology is insufficiently known, there is no typical clinical picture, but the signs and symptoms depend on the time of onset.

The diagnosis of prenatal TT is made on the basis of an imaging examination: ultrasound and MRI of the testis. Enlarged, inhomogeneous testis and two concentric hypoechoic layers of fluid containing irregular thin septa indicating hemorrhage ("double ring of hemorrhage"). The presence of hydrocele and enlarged testis is an early sign of TT due to vascular alteration to local inflammation as well as deviation of the scrotal septum towards the contralateral testis. Postnatal palpable hard mass is a consequence of hemorrhagic necrosis and echogenic ring of edema and fibrosis of the tunic albuginea (ultrasound postnatal finding) [27]. MRI finding of prenatal torsion: the testis is enlarged in relation to the other side, the scrotal wall is thickened, the septum is deviated to the healthy side and there is a hydrocele on the opposite side.

Extravaginal torsion first affects the venous system, causing growing edema, vascular congestion, tissue ischemia, reactive adhesion, and finally arterial obstruction. Fetal pain can be caused by fetal stress due to testicular torsion in utero. In the third trimester, the fetus may feel pain or show a reaction to the injury, hence the propulsion of meconium as a reaction to the torsion of the testicles, which is verified after birth. The study of Ricci et al. who during the urgent ultrasound of the pregnant woman due to reduced fetal activity in the 37th week of gestation noticed an abnormality of one testicle which was confirmed as TT

after birth. Distant results of prenatal torsion: Vanishing testis-testicular regression syndrome (TRS), cryptorchidism, nubbin testis.

The diagnosis of postnatal TT is made on the basis of: anamnestic data, clinical presentation, local status of genital organs and radiological findings. The clinical presentation of postnatal testicular torsion can be symptomatic or asymptomatic. Possible symptoms: Pain (testicular tissue ischemia), nausea, anorexia, vomiting, fever. Since the cremasteric reflex is developed in 48% of healthy infants, this is not helpful in the diagnosis of PTT [28]. Scrotal transillumination is valid only in the case of an associated hydrocele when it is registered as partial.

Testicular ultrasound: Evaluates the preservation of testicular tissue, vascularization and morphological structure. Scrotal US is performed with a high-frequency linear probe (usually from 7 -10Mhz and higher) with optimal resolution and the ability to register the slowest flow. Short-lived torsion is characterized by mixed echogenicity. Prolonged torsion is shown by classifications and a hypervascular hoop of a tunic with a hypodense centre[29]

Figure 10. Male newborn, twenty days old, admitted due to tumefaction in the right scrotum. Clinically and ultrasound evaluated right testis more voluminous, in the lower half hypoechoic and inhomogeneous. No free fluid in the scrotal sac Forms and time of appearance of PTT



• PTT occurred several months before birth: absence of testicles. It will later be treated as a cryptorchid presence of a small, hard, painless, nodular ("nubbin") or vanishing testis.

• PTT a few weeks before birth: painless hard scrotal mass in the upper part of the hemiscrotum, smaller than in the contralateral, normal scrotum, attached to the scrotal wall, without signs of acute inflammation, does not transmit light [30].

• PTT a few days before birth: painless hard scrotal mass of larger or similar size as contralateral normal testis, without acute inflammation, without light transmission [30].

• PTT a few hours before birth: acute scrotal inflammation, the scrotum is painful, bluish or reddish in appearance, the testis is enlarged, hard, sometimes elevated, the ribbon is thickened and painful.

• PTT during the first month of life: without any scrotal symptoms at birth, signs of acute scrotal inflammation will appear later [30].

Bilateral asynchronous TT: Since the tunica vaginalis is only firmly attached to the scrotal wallat the end of the first or second month, this period is critical for asynchronous torsion. It is important to point out that in the early prenatal period, a few weeks before birth and a few days before birth, there are no signs of inflammation, while a few hours before birth and during the first month of life, signs of inflammation are present. Unlike in adults, PTT has an insidious presentation. There are huge variations in the clinical finding depending on when the torsion occurred and when the clinical presentation was initiated. Excluding boys in whom testicular torsion occurs in the first month of life, it is often not possible to determine exactly when PTT actually occurred [31]! Differential diagnosis in the neonatal period: hydrocele, hematocele (consequence of birth trauma, adrenal hemorrhage), trapped inguinal hernia, testicular and paratesticular tumors, scrotal edema, scrotal hematoma, scrotal abscess, meconial peritonitis, epididymitis ... In the literature, there are controversies of surgical therapy in relation to: fetally verified testicular torsion, necessity of surgical exploration, urgency of surgery, place of incision (inguinal / scrotal), treatment of contralateral testis, orchiectomy, nubbin testis. There are several published papers on prenatally ultrasound-detected PTT, but with a hitherto unclear implication for

therapy. Urgent fetal extraction would be justified in the last week of pregnancy if bilateral PTT is suspected, provided the diagnosis is made shortly after torsion. There have been no published papers about it yet! The question is whether postnatal TT should be operated on, and if so, when? Immediately, to delay, or not to operate. There are three possibilities: observation, elective (delayed) operation or urgent operation. Surgical variants include: urgent exploration without / with orchiectomy and contralateral testicular fixation, elective exploration with / without contralateral testicular fixation, or an exploitative posture. Reasons for exploration: The diagnosis of testicular torsion is assumed, but not always certain, the possibility of testicular preservation is small, but it is possible. If the testicle is necrotic, devitalized tissue should be removed. There are also medical and legal aspects. Kaplan and Silber [32] state that even if a prompt diagnosis and exploration is done, only about 5% of the testes can be saved, while Sorensen indicates that this percentage is 40 to 50 [33]. The explanation given by Das and Singer points out that 30% of PTT is in the postnatal period, which gives the urgent surgical approach importance in the possible rescue of the torque testicle [22]. Unilateral PTT is less controversial because it is against exploration if the possibility of testicular rescue is assessed in relation to the potential risk of emergency anesthesia and surgery in the neonatal period [30]. Many authors believe that only bilateral perinatal torsion requires urgent surgical intervention due to the risk of anarchy [34]. A study of a limited group of pediatric urologists indicates that 10% of pediatric urologists prefer urgent exploration, 57% delayed, 33% only observation [30]. The favoured approach is the scrotal incision, justified in emergency interventions [27]. Inguinal incision is justified, in the case of other (possible!) Differential diagnoses (hernias, tumors, undescended testis).

Figure 11:Through a scrotal incision, the torqued right testicle was approached; torsion by 720<sup>o</sup>, "counterclockwise". Testicular recovery after detorsion is partial.



Do you fix the contralateral testis? With intravaginal torsion, there is an anatomical predisposition that torsion of the testicles will also occur on the contralateral side. Extravaginal torsion is a consequence of the lack of fixation of the scrotum and the vaginal tunic [36]. Recommendation: NO contralateral orchidopexy in postnatal unilateral testicular torsion, YES in adult TT because the mechanism of occurrence is different. Only one monorchid man with subsequent intravaginal torsion after postnatal torsion has been described in the literature [37]. Is orchiectomy justified / necessary? Bleeding during incision of the tunica albugina may be the best prognostic sign of potential gonadal vitality. Leaving the ischemic testis in situ, according to many authors, has no consequences for later fertility as in torsion in postpubertal men [38]. Immature spermatogenetic elements are not adequate antigenic stimulation during the neonatal period [39]. The left gonad in situ has both an aesthetic and a psychological role [40]. As there are no data on tumors arising from microscopic foci of viable testicular tissue in the nodular (nubbin) testis, there are disagreements about the preventive removal of such testicular remains [46]. 15% of the nubbin testis has seminiferous tubules.

#### CONCLUSION

A pediatric surgeon / urologist must be consulted immediately in case of an abnormal scrotal appearance! The degree of testicular preservation is directly dependent on the duration of symptoms. With a correct and timely approach, it is possible to increase the preservation rate of the affected testicle. A clear distinction should be made between prenatal ("old") and postnatal torsion of acute TT. Only this last group has a chance of preserving the testicles by urgent surgical exploration.

Whenever possible, especially in bilateral cases of PTT, an effort should be made to keep even necrotic tissue, since such a testicle will have minimal function.

#### **REFERENCES:**

- 1. Barteczko KJ, Jacob MI. The testicular descent in human. Origin, development and fate of the gubernaculum Hunteri, processus vaginalis peritonei, and gonadal ligaments. Adv Anat Embryol Cell *Biol* 2000;156: 1-98.
- 2. Kapoor S. Testicular torsion: a race against time. Int J Clin Pract 2008;62:821-827.
- 3. Zhao LC, Lautz TB, Meeks JJ, Maizels M. Pediatric testicular torsion epidemiology using a national database: incidence, risk of orchiectomy and possible measures toward improving the quality of care. J Urol 2011;186:2009-2013.
- 4. Thomas WE, Cooper MJ, Crane GA, Lee G, Williamson RC. Testicular exocrine malfunction after torsion. Lancet 1984;1357-1360.
- 5. Romeo C, Impellizzeri P, Arrigo T, et al. Late hormonal function after testicular torsion. J Pediatr Surg 2010;45:411-413.
- Sinisi, A.A., Di Finizio, B., Lettieri, F. et al. Late gonadal function and autoimmunization in familialtesticular torsion. Arch Androl 1993; 30: 147
- 7. Climatic conditions and the risk oftesticular torsion in adolescent males.J Urol 2007;178):2585
- 8. Dogra V, Bhatt S. Acute painful scrotum. Radiol Clin North Am 2004 Mar. 42:349-63.
- 9. Tajchner L, Larkin JO, Bourke MG, Waldron R, Barry K, Eustace PW. Management of the acute scrotum in a district general hospital: 10-year experience. *Scientific World* Journal 2009;9:281-286.
- Boettcher M, Bergholz R, Krebs TF, Wenke K, Aronson DC. Clinical predictors of testicular torsion in children. Urology 2012;79:670-674.
- 11. Canning DA, Lambert SM. Evaluation of the pediatric urology patient. In: Wein AJ, Kavoussi LR, Novick AC, Partin AW, Peters CA, eds. Campbell-Walsh Urology, 10th ed. Philadelphia, Pa.: Elsevier Saunders; 2012:3067-3084
- 12. Davenport M. ABC of general surgery in children. Acute problems of the scrotum. BMJ 1996;312:435-437.
- 13. Gold DD, Lorber A, Levine H, Rosenberg S, Duvdevani M, Landau EH, et al. Door To Detorsion Time Determines Testicular Survival. Urology 2019 Aug 10. [Medline].
- 14. Baker LA, Sigman D, Mathews RI, Benson J, Docimo SG. An analysis of clinical outcomes using color doppler testicular ultrasound for testicular torsion. Pediatrics 2000;105:604-607.
- 15. Cuervo JL,Grillo A,Vecchiarelli C,Osio C,Prudent L. Perinatal testicular torsion: a unique strategy.J Ped Surg 2007;42:699-703
- 16. Bomann JS, Moore C. Bedside ultrasound of a painful testicle: before and after manual detorsion by an emergency physician. Acad Emerg Med 2009 Apr. 16:366. [Medline].
- 17. Ramos-Fernandez MR, Medero-Colon R, Mendez-Carreno L. Critical urologic skills and procedures in the emergency department. Emerg Med Clin North Am 2013 Feb. 31:237-60. [Medline].
- 18. Ringdahl E, Teague L. Testicular torsion. Am Fam Physician 2006 Nov 15. 74(:1739-43. [Medline].
- Martin DA,RushtonGH. The Prevalence of Bell Clapper Anomaly in the Solitary. Testis in Cases of Prior Perinatal Torsion. J Urol 2014;
- 20. Riaz-Ul-haq M, MBBS, FCPS, FEBPS; Mahdi AED, Elhassan UE. Neonatal Testicular Torsion; a Review Article. Iran J Pediatr 2012;22): 281-289
- 21. Callewaert PR, Van Kerrebroeck P. New insights into perinatal testicular torsion. Eur J Pediatr 2010;169:705-712.
- 22. Das S,Singer A. Controversies of perinatal torsion of the spermatic cord: a review, survey and recommendations. J Urol 1990;143:231-33
- 23. Lee SD, Cha CS. Asynchronous bilateral torsion of the spermatic cord in the newborn: a case report. J Kor Med Sci 2002;17:712-4
- 24. Sozubir S, Barber T, Wang Y, Ahn C, Zhang S,Verma S, Lonergan D, Lorenzo JA, Nef S, Baker AL. Loss of Insl3: A Potential Predisposing Factor for Testicular Torsion. J Urol 2010;183:2373-9.
- 25. Kogan SJ, Gill B, Bennet B, et al. Human monorchism: a clinicopathological study of unilateral absent testis in 65 boys. J Urol 1986;135:758-61
- 26. Burge DM. Neonatal testicular torsion and infarction: etiology and management. Br J Urol 1987;59:70-3
- 27. Stone KT, Kass EJ, Cacciarelli AA, Gibson DP. Management of suspected antenatal torsion: what is the best strategy? J Urol 1995; 153:782–4
- 28. Chiang MC, Chen HW, Fu RH, Lien R, Wang TM, Hsu JF. Clinical features of testicular torsion and epididymo-orchitis in infants younger than 3 months. J Ped Surg 2007;42:1574-77
- 29. Herman A, Schvimer M, Tovbin J, Sanbank,Bukovski I,Strauss S.Antenatal sonographic diagnosis of testicular torsion.Ultrasound Obstet Gynecol 2002; 20: 522– 524
- 30. Yerkes B E, Robertson MF, Gitlin J, Kaefer M, Cain PM, Rink C R. Management of Perinatal Torsion : today, tomorrow or never ? J Urol 2006;174: 1579-83
- 31. Giannakopoulos X, Chambilomatis P, Filiadis I.Six cases of prenatal and neonatal torsion of the spermatic cord. Int J Urol 1997;4:324–326
- 32. Kaplan GW, Silber I. Neonatal torsion- to pex or not to pex? In: Urologic surgerz in Neonates and young infants. Edited by LR King, Philadelphia: WB Sounders Co,vol 1, chapt. 20,pp 386, 1988.
- 33. Sorsen MD, Galansky SH, Striegl AM, Mevorach R, Koyle AM. Perinatal extravaginal torsion of the testis in the first month of life is a salvageable event. J Urol 1993;62:132-4.
- 34. Pinto KJ, Noe NH, Jerkins GR. Management of neonatal testicular torsion.
- J Urol 1997;156:1196-8
- 35. Guerra LA, Wiesenthal J, Pike J et al: Management of neonatal testicular torsion: which way to turn? Can Urol Assoc J 2008; 2: 376.

- 36. Aaron D. Martin, Rushton GH. The Prevalence of Bell Clapper Anomaly in the Solitary Testis in Cases of Prior Perinatal Torsion. J Urol 2014;191:1573-1577
- 37. Harris BH, Webb HW, Wilkinson AH Jr et al: Protection of the solitary testis. J Pediatr Surg 1982; 17: 950.
- Arda IS,Ozyaylali I.Testicular tissue bleeding as an indicator of gonadal salvageability in testicular torsion surgery. BJU Int 2001;87:89-92
  Frank JD, O<sup>T</sup>Brien M. Fixation of the Testis. BJU Int 2002; 89:331-33
- 40. Driver PC, Losty DP. Neonatal Testicular Torsion. BJU 1998; 82: 855-8