Effects of Hydrocele on Morphology and Function of Testis

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Abstract

Hydrocele is generally believed as innocent. But there is increasing evidence of noxious influences of hydrocele on testis resulting in morphological, structural and functional consequences. These effects are due to increased intrascrotal pressure and higher temperature-exposure of the testis. Increased intrascrotal pressure can cause testicular dysmorphism and even testicular atrophy. The testicular dysmorphism is reversible by early hydrocele surgery, but when persist, possibly indicate negative influence on future spermatogenesis. Spermatic cord compression by hydrocele is responsible for testicular volume increase. Such testes lose 15%-21% volume after hydrocele surgery. Tense scrotal hydrocele can cause acute scrotal pain from testicular compartment syndrome, which is relieved by evacuation of hydrocele. Higher resistivity index of subcapsular artery of testis and higher elasticity index of testicular tissue are caused by large hydrocele. As an aftermath, testis suffers ischaemia with long-term effect on spermatogenesis. High pressure of hydrocele along with ischaemia and oedema is found to result in histopathological damage to testis like total/partial arrest of spermatogenesis, small seminiferous tubules, disorganized spermatogenetic cells, basement membrane thickening and low fertilty index in children. Higher temperature exposure of testis interferes with spermatogenesis. In adults it results in lower quality of semen in the form of oligospermia and low motility of sperms. Congenital hydrocele can be observed upto 2y age and noncongenital hydrocele upto 1y period for spontaneous resolution in the absence of associated pathology indicating earlier operation like inguinal hernia, cryptorchidism, tense hydrocele, testicular torsion, testicular mass,etc. In adults with tense hydrocele early operation is indicated.

Keywords: Hydrocele; Testicular dysmorphism; Intrascrotal pressure; Histological damage to testis; Resistivity index of testis; Reduced spermatogenesis

Introduction

Hydrocele is fluid accumulation in between the visceral and parietal layers of tunica vaginalis (TV) testis. In pediatric age-group, hydrocele is generally due to persistence of part of processus vaginalis (PV). In communicating hydrocele (CH), patent PV communicates with peritoneal cavity. Noncommunicating hydrocele (NCH) is limited to scrotum usually, or extends upto external inguinal ring. Abdominoscrotal hydrocele (ASH) is occasionally seen; in this condition, large scrotal hydrocele communicates with abdominal component, through narrow inguinal canal in an hourglass fashion. The most accepted theory of origin of ASH suggests that obliteration of PV results in fluid accumulation in TV leading to increased intrascrotal pressure. When intrascrotal pressure exceeds the intra-abdominal pressure (IAP), inguinal portion of a large hydrocele is pushed into the low-pressure abdominal compartment through the inguinal canal, forming a dumbbell configuration with central constriction at the internal inguinal ring.^[1] Hydrocele is prevalent in upto 57.9% of male full-term newborn infants. Extravaginal (communicating) type was present in 86% of all infants, intravaginal (noncommunicating) in 9.5%, and both types together in the same neonate was observed in 4.5%.^[2] Unlike chronic hydrocele, acute hydrocele is secondary to acute process within TV or torsion of testis or its appendages.

Hydrocele is painless, generally believed as innocent, not affect testis and most primary hydroceles regress. The difference in intrascrotal temperatures between hydrocele side and normal side in adults was found not significant by Krahn in 1963, but hydrostatic pressures in the hydroceles were significantly higher (8-21 cm H2O) than that in normal side T.V. sac (0-3.5 cm H₂O). ^[3] Biopsy specimens from affected side and normal side in 9 patients showed no difference in morphology of seminiferous tubules by the same author. ^[3] In 1989, Singh discovered that sperm concentration and their motility in semen was found affected by the duration of hydrocele. ^[4] In 1990, the then published research works revealed that testicular dysmorphism (TD), atrophy, reduced spermatogenesis can be resultant from hydrocele, ^[5] and when associated with certain pathological

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conditions, hydrocele operation should not be delayed even in children. ^[6] Later more and more scientific evidences are accumulating both in adults and in children including infants, of the deleterious effects of apparently innocuous hydrocele. It was surprising to find no mention of any adverse effects of hydrocele to testis in recent reviews of hydrocele. ^[7,8] The present review is an expedition to elucidate the effects of different types of hydrocele in children and adults on the tissue-pressure within testis, its blood supply, the morphologic and histopathological alterations, spermatogenesis and the fertility potential of the individual in the long run. Minimizing further complications from a hydrocele is dependent on the employment of timely therapeutic techniques following diagnosis. ^[9]

Method of Data Collection and Analysis

Using the search-engine "google" and applying search items as 'effects of hydrocele on testis', 'effects of hydrocele on testis in children', 'testicular compartment syndrome', 'testicular dysmorphism', 'effects of hydrocele on fertility', 'histopathological changes of testis in hydrocele', literature search was done and publications were listed. Publications related to animals were excluded. Publications after 1960 only are collected. Relevant data from articles were compiled, analysed and conclusions drawn. Data were categorized into headings and subheadings based on different effects on testis by hydrocele in adults and in children. Based on available evidences, finally, current recommendation to the timing of hydrocele treatment is arrived at.

Effects of Intrascrotal Pressure on Testis

Effects on morphology of testis

Dedhia found that hydrocele can adversely affect morphology of testis. [10] Dandapat et al. studied the effect of unilateral hydrocele of TV of 2-5y duration in 120 patients of 30-40y age group.^[5] The testes of unaffected side acted as controls. On the hydrocele side, 22% of testes showed flattening and 8% atrophy, while 70% of testes showed no pressure effect . Thickening of the epididymis and cord was observed in 12% and 18% of cases respectively. In adults, tendency for rounding rather than flattening of the ipsilateral testis due to hydrocele was highlighted by others. [11] Anatomical changes in testicular morphology, such as the development of fusiform testes, have been observed in infants with large, tense ASH and are reportedly reversed after hydrocelectomy. In a review of infantile ASHs with TD including 73 patients (94 testes), TD (defined as flattened and elongated or fusiform testes) was reported in 65/94 (69.1%) of the affected testes as detected during surgery. Epididymal abnormalities were identified in 12/94 (12.8%) of the affected epididymes. ^[12] Testicular dysmorphism has also been reported by many authors. [5,10,12-33] [Table 1]. In a case report of ASH, testis was situated between abdominal and scrotal components bilaterally. ^[1] Testicular dysmorphism is mostly associated with ASHs. Hydrocele sac pressures are higher than that in the abdominal compartment (>11 cm of H2O). [34-40] Thus, a hydrocele in childhood cannot be simply explained by the patency of the PV at a caliber that allows only the passage of abdominal fluid into scrotum. In a hydrocele, the contractile activity of the persisting smooth muscle of the incompletely obliterated PV actively propels the fluid into the sac, but prevents its retrograde flow. The reported distal intussusception of PV probably also supports its propulsive activity.^[40]

The two theories regarding the mechanism by which testicular dysgenesis (TD) develops include (1) increased hydrostatic pressure on the testicular parenchyma or the vessels due to large tense hydrocele and (2) intrinsic developmental abnormality of the testis which is a part of the ASH complex or a coexisting finding. ^[12,22] Excess hydrostatic pressure of hydrocele resulted in testicular atrophy, [27,29] epididymal abnormalities, such as splayed and thickened and/or elongated epididymis, [22] epididymal tissue incorporated in the hydrocele sac, ^[26] and complete lack of epididymis. [22,25,27] Complete postoperative recovery in most of the testes with dysmorphism [23-25] suggests that early operative intervention can prevent functional damage. However, some testes were persistently hypoplastic despite early surgical intervention. [23] Such testicular hypoplasia can be the result of pressure damage or secondary to operative vascular compromise. [22] A high incidence of contralateral abnormalities was observed in infants with ASH. This observation may suggest that unilateral TD is more related to developmental abnormalities than to the pressure effect of hydrocele fluid on the testis. In such cases postoperative recovery from TD could not be expected. The morphologic testicular changes observed in infants with ASH suggest possible long-term functional deficiency including fertility risk. In a recent review of ASH involving 578 cases, Gadelkareem observed TD in 69.10%-90% cases. [32] Testicular dysmorphism is often reversible in pediatric patients. Unilateral/ bilateral testicular atrophy, impaired spermatogenesis or azoospermia have been reported in ASH. [32]

Jedrzejewski et al. observed in boys aged 8-36m by scrotal ultrasound scan (USS) that in presence of scrotal hydrocele, the volume of testes decreased with increasing age. ^[28] In 120 patients of 20-40y age with bilateral hydrocele of 2-8y duration, Singh et al. demonstrated that overall 12.5% of testes, and in severe hydrocele, 46.4% of testes had decrease in size. ^[33]

Jozsa et al. proved that the persistence of NCH in patients with mean age of 40m, for at least 1 month, resulted in the destruction of appendix testis, indicated by the absence of androgen receptor and estrogen receptor expressions and by epithelial destruction. ^[41] This is probably the effect of elevated hydrostatic pressures. Such knowledge may suggest alteration in the age of surgical treatment of pediatric patients with NCH. Yang et al. operated 284 boys 18 m-56 m age, with hydrocele laparoscopically. Interestingly, almost all the preoperative NCH (283/284) were discovered with open internal rings and patent PV rather than with closed internal rings. But there were no fluctuating sizes of these hydroceles. They detected "valve-like pore" on top of the 'noncommunicating' hydrocele observed in their laparoscopic explorations. This had a key role in making the fluid flow only from peritoneal cavity into hydrocele. In support of this, they detected blue staining of the aspirated fluid of the hydrocele. ^[42] Hydrocele of some neonates and infants can spontaneously

| Table 1: Published data of morphological and histological changes in testis due to hydrocele. | | | | | | | | | | | |
|---|---------------------|-------------------------------|--------------|-------------------|--|---|---|--|--|--|--|
| Author | Year of publication | No. of patients/ testes | Age group | Type of hydrocele | Dysmorphism | Histological change | Semen analysis | | | | |
| Krahn et al ^[3] | 1963 | 9 | Adults | | - | Nil | - | | | | |
| Gratania et al ^[34] | 1969 | - | Adults | | - | Nil | - | | | | |
| Dedhia et al | 1981 | - | Adults | | Adversely affect morphology | Adversely affect spermatogenesis in 65% cases | - | | | | |
| Singh MP et al ^[35] | 1989 | 43 | Adults | SH | N/A | Histological alteration in 52.2% of biopsies | - | | | | |
| Singh MP et al ^[4] | 1989 | 43 | Adults | SH | N/A | N/A | Sperm concentration &motility affected by duration of hydrocele | | | | |
| Dandapat et al ^{is} | 1990 | 120 | 30-40 y | - | Flattening in 22%, atrophy of 8% testes | total arrest of spermatogenesis in 8% of the testes, partial arrest in other 10%. disorganized spermatogenic cells seen in the tubules of 42%, interstitial cell fibrosis in 85% of biopsies | - | | | | |
| Chamberlain et al ^[13] | 1995 | 2 testes | 2.75 m | ASH | 2 Fusiform testes | Not done | - | | | | |
| Ferro et al ^[14] | 1995 | 4/5testes | 8 m | " | 1 Elongated testis | Reduced parenchyma | - | | | | |
| Sereles and Kogan ^[15] | 1996 | 4 testes | 4 m | " | Tubular & dysplastic testes | | - | | | | |
| Nagar and Kessler ^[16] | 1998 | 15/19 testes | 3.9 m | " | Flattened &elongated testes | | - | | | | |
| Cooper et al ^[17] | 1999 | 2/3testes | 8 m | " | 2 fusiform 1 elongated | | - | | | | |
| Avolio et al. [18] | 2000 | 9/10 testes | 7-18 m | u | 1 fusiform | | - | | | | |
| Srinath et al ^[36] | 2004 | 25 | 27-48 y | Unilateral SH | No effect on volume | distortion of the seminifer- ous tubules arrangement, reduction in the number of seminiferous cell count due primarily to a decrease in spermatids | - | | | | |
| Mihmanli. ^[19] | 2004 | 23 | 21-72 Y | - | Volume higher. Volume decrease postoperatively | Not studied | - | | | | |
| Kinoshita et al ^[37] | 2006 | 1/1 | 9 m | ű | 1 normal | | - | | | | |
| Turgut et al [20] | 2006 | - | - | | Rounding of testis. | | - | | | | |
| Adaletli. ^[21] | 2006 | 17 | 2-7Y | - | Volume higher. Volume decrease postoperatively. 9 elongated testes 5 | Not studied | - | | | | |
| Bayne et al. [22] | 2008 | 6/9 testes | 5-12 m | u | Splayed epididymis, 4 Thickened and/or elongated epididymis | | - | | | | |
| Cozzi et al. ^[23] | 2008 | 18/23 testes | 3-21 m | u | 18 Flattened and elongated testes | Not studied | - | | | | |
| Cuervo et al. ^[24] | 2009 | 6/6 | 3-8 m | ű | 1 fusiform testis | | - | | | | |
| Kajbafzadeh et al. ^[25] | 2010 | 7/10 | 3-84 m | ű | 9 Flattened and elongated testis | | - | | | | |
| Park et al ^[26] | 2010 | 1/1 | 11 m | ű | Normal testis Epididymal tissue Incorporated in hydrocele sac (1) | | - | | | | |
| Halilbasic et al ^[27] | 2011 | 1/2 | 11 m | ű | 1 testicular atrophy and lack of epididymal tissue | | - | | | | |
| Jedrzejewski et al ^[28] | 2012 | 41 | 8-36 m | SH | Volume decreased with increasing age | Not studied | - | | | | |
| Vaos et al ^[12] | 2014 | 1/1 | 10 m | ASH | Flattened, elongated fusiform testis, splayed epididymis | Not studied | - | | | | |

| Gupta et al. [38] | 2015 | 92 cases | 20-40 y | Unilateral SH | N/A | basement membrane thickening, interstitial fibrosis, disorganization of seminiferous tubules & impaired spermatogenesis | - |
|-----------------------------------|-------|-------------------------------------|----------------------|----------------------------|---|---|--|
| Kamble et al ^[29] | 2015 | 1 | 25 y | ASH | Atrophic testis | N/A | - |
| Kurokawa et al ^[30] | 2016 | 76/76 | 4 y | 34 SH, 42 sp. cord H | Smaller testes in SH compared to testes with sp. cord H | Smaller seminiferous tubules. | - |
| Tatekawa Y ^[31] | 2017. | 1 | 8m | | Bilateral fusiform testes. | Not studied | - |
| Gadelkareem | 2018 | 578 | Pediatric & adult | | Flattened & elongated/ fusiform testis 69.1-90% | Not studied | - |
| Kokaoglu et al ^[39] | 2018 | 52 testes with SH; 36 control | 6.32 m | SH | Volume not reduced. Elastograph value high by SWE | Not studied | - |
| Singh RK et al ^[33] | 2019 | 120 cases | 20-40 y | Bilateral H | Smaller testes in 12.5% | Not studied | Oligospermia in 15%, abnormal shape & motility of sperms |

Abreviations used: H=hydrocele; SH=scrotal hydrocele; ASH= abdominoscrotal hydrocele; sp.cord=spermatic cord; N/A= not available; SWE= shearwave elastography.

resolve. Thus patients more than 2 years old were recommended with surgical repairs. In their clinical practice, Yang et al. observed that typical CHs and NCHs with volume greater than 10 ml with high tension could hardly resolve after 18 months of age. In addition, anxiety generally existed among parents of this group of patients. ^[42]

Adaletli et al. compared the volume of testes on the side of CH with that of testes on the normal side in 17 children aged 2-7y. Before hydrocelectomy, the volume of testes on hydrocele side was higher than that of normal side. After hydrocelectomy, volume of testes on hydrocele side decreased by 15% whereas no change in volume was observed on normal side. [21] Mihmanli et al. found in adults that the high volume of such affected testes reduced by 21% after hydrocelectomy. The resistive (RI) and pulsatility (PI) indices of the intratesticular arteries of testes with hydrocele were significantly higher than those on normal side testes. Significant decreases in RI and PI values by 21% and 36% respectively were detected after hydrocelectomy. ^[19] The increase in testicular volume and vascular resistance are due to an increase in impedance to venous and lymphatic flow. The pressure of the hydrocele causes obstruction in the venous and lymphatic outflow. This stasis is in turn reflected as swelling and an increase in the volume of the testis. The decrease of testicular volumes postsurgery showed that the pressure effects were eliminated, and, possibly this considerably benefits the testes. Increase in testicular volume of testes with spermatic cord hydrocele, compared to that of testes with testicular hydrocele in children was demonstrated by Kurokawa et al.. [30] Turgut et al. demonstrated that the mean testicular volumes of adult patients with unilateral hydrocele of more than 6m duration were significantly less than those with hydrocele of less than 6m duration. Unilateral idiopathic hydrocele has a tendency for rounding rather than flattening the ipsilateral testis. [20]

Testicular compartment syndrome

Dagrosa et al., reported 15-year-old boy who presented with 2 days of increasing testicular pain and swelling, diagnosed to have a large hydrocele with compromised testicular perfusion

by doppler USS. He was managed by emergent operative drainage and repair of a "tension hydrocele" with immediate regain of testicular perfusion. ^[43] There are a few reports of such "testicular compartment syndrome" due to idiopathic hydrocele in adult patients. ^[44-46] Pressure within the hydrocoele needs to exceed the systolic blood pressure. ^[47] The diastolic blood flow is found absent in testis with hydrocele. ^[45] The elevated hydrocele pressure plays an important role in the malfunction of spermatogenesis as well as in hypoxemic hypoxia of the testis. ^[47]

A tense hydrocele in adult patients may damage spermatogenesis. But testicular functional status cannot be evaluated properly preand postoperatively in the pediatric patients due to failure to obtain semen analysis and limitations to do testicular biopsies. Shear wave elastography (SWE) is an ultrasound technique used to track shear waves passing through tissues by quantifying the elasticity of structures and nodules. Recently it has been found that with small hydroceles, there is no difference in testicular volume and stiffness values of testes with and without hydrocele as determined by SWE in children. ^[48] Kokaoglu et al. using SWE compared the testicular volumes and median elastography of testes of children with average age of 6.32 m. 52 testes with NCH and 36 testes without hydrocele were studied. SWE values of testes with NCH were significantly higher compared with those of controls, despite the absence of a significant difference in testes volumes. It is concluded that NCH may be damaging to testicular tissues.^[39] It is logical that the operation age of children with NCH need re-evaluation in the light of the above findings.

Effects on resistivity index (RI) and elasticity index (EI) of testis

Turgut et al. also documented an increase in resistivity index (RI) of the subcapsular artery on the hydrocele-affected side, which might be a sign of ischemia. ^[20] Nye and Prati, on the other hand, observed an absent testicular diastolic flow on hydrocele-side. ^[45] Hence, early treatment of hydrocele, particularly those that are large and tense, might be necessary to prevent testicular

damage. ^[11,49] Tension hydrocele is a rare condition that produces alterations of form and circulation, along with acute pain of testes. Zawaideh et al. evaluated retrospectively a series of five patients with mean age of 57y with a long history of hydrocele and increasing acute scrotal pain. [50] Patients were investigated with gray scale US of both testes, as well as with color-doppler and spectral analysis, comparing the results with those after hydrocele aspiration and symptoms relief. The involved testicles had "flattened" appearance and parenchymal doppler signals showed increased intratesticular vascular resistance. One patient had a low diastolic flow, compared to the contralateral testis, with an increased RI value, one had no diastolic flow, two patients had retrograde diastolic flow, and the remaining one had no intratesticular flow visible. After decompression, there was disappearance of pain and improved flow with normalization of testicular vascularity. A case of clinically acute scrotum in 14-month-old boy who underwent emergency exploration was found to be due to intense inflammation of hydrocele wall confirmed by histopathology.^[51]

Histopathologic alterations in testis due to hydrocele

Gratania found no difference in histology between affected testes and normal testes. [34] Ferro in 1995, found that among his pediatric patients operated for ASH, an 8m old child's testis was elongated. Biopsy examination revealed reduced testicular parenchyma. ^[14] Kurokawa et al. examined whether hydroceles in children caused histopathological damage to testes by comparing the spermatic cord hydroceles with the testicular hydroceles. Testes with spermatic cord hydroceles were significantly longer than those with testicular hydroceles. The large testes (over 20 mm) with spermatic cord hydroceles showed significantly larger diameter of seminiferous tubules compared to small testes (under 13 mm) with testicular hydroceles. The larger testes with spermatic cord hydroceles could be caused by testicular stromal edema due to an increase of resistance to venous and lymphatic flow by hydrostatic compression on the spermatic cord, whereas the smaller testes with testicular hydroceles could be caused by the shrinkage of the seminiferous tubules due to hydrostatic pressure to the testes. ^[30] Singh et al. who performed biopsies on testes during hydrocele operations in adults showed alteration in 52.2% of the biopsies depending on size and duration of hydrocele. [35] Politof et al.. published their results of investigation of the prognosis for fertility in children who had undergone surgery for unilateral or bilateral hydrocele. ^[6] Testicular biopsy findings were positive, and therefore the fertility prognosis was very poor in 75% of patients with associated pathology such as cryptorchidism, varicocele, and pre-existing torsion. Children with hydrocele and pathological findings are significantly older than hydrocele patients with no associated pathology. Hydrocele on its own has no effect on subsequent fertility. Hydrocele does not require immediate surgery. In the presence of hydrocele and certain associated pathology, however, surgery is indicated. [6] Effect of simple hydrocele on later fertility were examined in one report in children undergoing hydrocele repair. The authors found that a poorer but not significant fertility index was observed on the hydrocele side when ipsilateral testicular and epididymal findings were taken into consideration.^[22]

Dandapat et al.. studied the biopsies of testes from hydrocele side of unilateral hydrocele of TV of 2-5 y duration in 120 patients of 30-40 y age group. ^[5] They reported 8% of the testes had total arrest of spermatogenesis, while other 10% exhibited partial spermatogenesis arrest at spermatocyte level. All testes from control side were histologically normal. Large numbers of disorganized spermatogenic cells were seen in the tubules of 42%, also interstitial cell fibrosis in 85% of biopsies, basement membrane thickening (78% of testes on affected side vs 12% of testes on the control side), as well as thickening of tunica albuginea and of TV (85% of testes) on hydrocele side. Similar changes were detected in 8% of testes on the control side. Arrest of spermatogenesis and consequent testicular atrophy can be the result of high fluid pressure in the tunica vaginalis, pressure on the blood supply of the testis from oedema within a tight fibrous sheath enclosing the TV as well as due to increased intrascrotal temperature. Disorganization and sloughing of tubules observed in histopathology might be due to obstruction of the vas or epididymis from fluid pressure. Interstitial fibrosis might have been due to prolonged contact with protein-rich fluid inside the TV. In conclusion, big hydroceles of the TV testis of long duration impair spermatogenesis and may lead to subfertility or infertility. The duration of hydrocele and testicular change are directly proportional.^[5] Similar conclusion was arrived by other authors too. ^[36,38] The fact that such changes occur is a strong argument for advising surgical treatment of all hydroceles particularly in men in the reproductive age group. Large and tense vaginal hydroceles significantly affect the morphology and histology of testis. The testis appears first to protect itself against an increased ambient pressure by a thickening of the tunica albugenia. Hydrocele is a pathological finding by sonography in 7% of infertile men is significant. ^[52]

Effects of intrascrotal temperature on spermatogenesis

There is evidence in children and adults that, raised intrascrotal temperature possibly harm testicular function including impairment of spermatogenesis. ^[11] But the most temperature-sensitive testicular cells in adulthood, pachytene spermatocytes and round spermatids, are not yet present in infantile testes. In conclusion, there is no convincing evidence supporting a link between genital heat stress in young boys and poor semen quality in adulthood. ^[53] In the most temperature-sensitive testicular cells, i.e., pachytene spermatocytes and round spermatids, apoptosis is induced via the mitochondrial pathway activating the Bax protein. Another potential molecular mechanism involves a cold-inducible RNA-binding protein (CIRP), which is expressed in vitro in murine pachytene spermatocytes and round spermatids cultured at 32°C and is suppressed in culture temperatures of 37°C. ^[53]

Discussion

Semen and blood samples from 99 healthy men were analysed by Hjolland et al.. in relation to scrotal skin temperature obtained by

a 24-h continuous monitoring protocol. A negative correlation was found between high scrotal temperature and sperm output. Sperm concentration decreased 40% per 1°C increment of median daytime scrotal temperature. Similar results were found for total sperm count, follicle stimulating hormone (FSH), and inhibin B.^[54] Dedhia found that hydrocele can adversely affect spermatogenesis in 65% cases. ^[10] In 1989, Singh et al. detected in 43 randomly selected scrotal hydrocele patients, FSH and leutinising hormone (LH) levels were raised in 50 and 75% of patients respectively whereas testosterone level was found subnormal in 50% patients undergone investigations. LH and FSH levels also showed some relation to duration and size of the hydrocele. Similarly, sperm concentration and their motility was found affected by the duration of the disease. ^[4] Singh et al. investigated the effect of hydrocele on spermatogenesis in 120 patients of 20-40 y age with bilateral hydrocele of 2-8 y duration. On semen examination, there was oligozoospermia seen in 15% of cases which improved post hydrocele-operation, some cases also showed defect in sperm morphology and sperm motility.^[33] The arrest of spermatogenesis is likely due to increased pressure on the blood supply of the testis from edema and from a rise in intrascrotal temperature. [5,44,55] Water, which has a very high specific heat compared to other liquids, is the major component of hydrocele. Water is resistant to transmission of heat. High heat from water being trapped within the scrotum gives the scrotum a temperature too high for optimum spermatogenesis. Hydrocele has a direct link to male infertility. Hydrocele produce infertility due to compartment syndrome, compression, increased temperature of hydrocele, and ischaemia.

When to operate hydrocele?

There has been debate on the necessity of surgery in pediatric patients with hydrocele, as well as on the best timing of intervention. Many researches have already found that hydrocele may interfere with spermatogenesis. Recently Acer-Demir et al. retrospectively reviewed hydrocele progression in 355 pediatric patients. [56] They recommended that until strong evidence of hydrocele-induced testicular damage in children is available, following up congenital hydroceles until at least 1 year and preferably 2 years of age, and following up noncongenital hydroceles for at least 6 months and preferably 1 year if there is no associated pathology indicating earlier surgery like inguinal hernia, cryptorchidism, tense hydrocele, testis torsion, or testis mass. [8,56,57] Non-communicating hydroceles are often resolved by two years of age in infants while it is resolved within six months in boys (older than one year with onset of hydroceles) in approximately 75% cases. Indications of surgery in the first year of life are an associated inguinal hernia and hydroceles that become huge in size. ^[58] In adults, hydroceles do not need treatment, if there is normal testicle or if the patient remains asymptomatic.^[7] In view of future functional changes in testes having morphological changes associated with hydroceles. early operation is advocated in tense hydroceles.^[13]

Conclusion

All hydroceles are not innocent as previously considered. Tense hydroceles, large hydroceles (vertical length >12.5 cm), hydroceles of long duration (>48 m duration), and abdominoscrotal hydroceles can cause serious harm to testis. The deleterious effects may be in the form of mechanical pressure resulting in alteration of morphology, histological damage to seminiferous tubules, and impedence of testicular blood supply manifested as acute scrotal pain or ischaemia of testicular tissue. Exposure to high temperatures of hydrocele can result in thermal damage to spermatogenetic cells. One or more mechanisms may be damaging in each case. In the longrun, future spermatogenesis can be adversely affected resulting in oligospermia or azoospermia. Damage to testis should be prevented by timely intervention of hydrocele.

Competing Interests

The authors declare that they have no competing interests.

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