ClinicSearch

Clinical Reviews and Case Reports

Wade Alwan M Yahya *

Open Access

Research Article

Insights into Varicocele: Anatomical Consideration, Prevalence, Incidence, Etiology, Pathophysiology of Infertility with Varicocele, Diagnosis, and Management

Wade Alwan M Yahya [1]*, Rabia A M Yahya [2], Azab Elsayed Azab [3], Karema El.M.Shkal [2]

[1]Surgery Department, Faculty of Medicine, Sabratha University, Libya.

[2]Pharmacology Department, Faculty of Medicine, Sabratha University, Libya.

[3]Physiology Department, Faculty of Medicine, Sabratha University, Libya.

***Correspondence Author:** Wade Alwan M Yahya, Surgery Department, Faculty of Medicine, Sabratha University, Libya.

Received Date: April 19, 2025 | Accepted Date: April 25, 2025 | Published Date: April 30, 2025

Citation: Wade Alwan M Yahya, Rabia A M Yahya, Azab Elsayed Azab, Karema El. M. Shkal, (2025), Insights into Varicocele: Anatomical Consideration, Prevalence, Incidence, Etiology, Pathophysiology of Infertility with Varicocele, Diagnosis, and Management, *Clinical Reviews and Case Reports, 4*(2); **DOI:**10.31579/2835-7957/137

Abstract

Background: Varicocele is characterized by abnormal dilatation and tortuosity of the pampiniform plexus veins. The most prevalent cause of male infertility globally is varicocele, and the most common male infertility surgery performed globally is varicocele repair and surgical procedures that improve semen parameters. Objectives: The current review aimed to highlight the anatomical consideration of varicocele, prevalence, incidence, and etiology of varicoceles, pathophysiology of infertility with varicocele, diagnosis, and management. Previous studies mentioned that 4% and 30% of the general population have varicocele. Varicocele is a major cause of male infertility. Varicocele is 2-3 times more common in men who consult infertility clinics than in men in the general population or with confirmed fertility may be one of the reasons for the association between varicocele and infertility. The prevalence of varicocele in infertile couples ranged from 6% to 47% depending on the geographic location. Varicocele was reported to affect 16.6% of 7.802 men referred for infertility in a European research and makes up 21% of the Greek population. It is noteworthy that varicocele is seen on the left side in 78% to 93% of cases, several ideas have been put up to explain their cause. All patients with a single right-sided varicocele should have the potential of thrombosis or blockage of the inferior vena cava ruled out; situs in versus is another cause of a right varicocele. The detrimental impact of varicocele on testicular function is attempted to be explained by hyperthermia, reactive oxygen species, hypoxia and "adrenal reflux", and endocrine imbalanced. The testicular pathology associated with varicocele is testicular hypotrophy, and a wide range of abnormalities on testicular biopsy. The most frequent findings include sloughing of the germinal epithelium, spermatogenesis halt, reduced spermatogonia per tubule, and hyperplasia of the Leydig cells. Numerous diagnostic procedures can be employed to validate these clinical results such as, Clinical diagnosis. Hormonal assay, Adjunctive diagnostic tests, Thermography, Venography, Radio nucleotide technetium99 pertechnetate scintigraphy, and Ultrasound imaging especially Doppler ultrasonography. The procedure that is most frequently used to treat male infertility is varicocelectomy. Varicocelectomy is indicated for the following clinically relevant varicoceles: infertility, especially when sperm quality or semen parameters are compromised, Hypogonadism, Pain in the scrotum, Hypotrophy of the testicles, especially in children, and problems with massive varicoceles' appearance. Treatment options for varicocele include: percutaneous occlusion, which involves injecting different materials intravenously to block the varicoceles, surgical ligation, Laparoscopic varicocelectomy, Robot-assisted varicocelectomy, Open varicocelectomy, and Microsurgical sub inguinal varicocelectomy. Conclusion: Varicocele is a major cause of male infertility. The prevalence of varicocele in infertile couples ranged from 6% to 47% depending on the geographic location. The detrimental impact of varicocele on testicular function is attempted to be explained by hyperthermia, reactive oxygen species, hypoxia and "adrenal reflux", and endocrine imbalanced. The current review showed that the anatomical consideration of varicocele, prevalence, incidence, and etiology of varicoceles, pathophysiology of infertility with varicocele, diagnosis, and management. Numerous diagnostic procedures can be employed to validate these clinical results such as, Clinical diagnosis. Hormonal assay, Adjunctive diagnostic tests, Thermography, Venography, Radio nucleotide technetium99 pertechnetate scintigraphy, and Ultrasound imaging especially Doppler ultrasonography. Treatment options for varicocele include: percutaneous occlusion, surgical ligation, Laparoscopic varicocelectomy, Robot-assisted varicoclectomy, Open varicocelectomy, and Microsurgical sub inguinal varicocelectomy

Keywords: varicocele; anatomy of varicocele; prevalence; etiology; pathophysiology of infertility with varicocele; diagnosis; management

1.Introduction

Celsus identified two distinct scrotal disorders, the first of which was varicocele, a benign enlargement of the scrotal skin's superficial blood veins. Celsius uses thin, pointed hot irons to cauterize a "varicocele" on the scrotum. The second illness, which Celsus called circocele (varix of blood vessel), is consistent with what we now know about varicocele. Celsius claims that circocele may be a painful condition or "orchidoptosis" that results in atrophy (when one testicle significantly declines and becomes smaller than the other due to malnutrition). He explained the steps taken to expose, isolate, legate, and resect the adjacent venous convolution. Semi-castration was used in the severe situations [1].

Abu-Alqasim Alzahrawi was the most significant Arabian surgeon of the tenth century in the field of medieval medicine. He is said to have lived to be 101 years old and was born in Cordoba. He preferred cauterizations in scrotal surgeries, as is typical in Islamic medicine, even if he mostly agreed with Celsus [2].

Differentiating between varicocele (varicocele scrota) and cirsocele (cirsocele venae spermaticae) was no longer common during the eighteenth century. In his foundational book on testicular disorders in 1843, British surgeon T.B. Curling (1811–1888) articulated a clear position when he replaced the term "cirsocele" with "varicocele" to refer to pathological dilatation of the spermatic veins [1].

Anyone studying varicocele from a historical perspective will unavoidably encounter a terrible incident that occurred in Montpellier on October 28, 1832, and at the time created a great deal of agitation. This regrettable event is frequently brought up in the literature as a reminder to everyone involved to exercise caution when considering any reason for corrective surgery on a varicocele, especially when it comes to underestimating the risk of testicular atrophy [1]. Varicocele was a significant exclusion criteria for military duty for centuries [3]. Macomber and Sounder originally proposed varicocele as a cause of male infertility in 1929 [4]. However, it wasn't until 1952 that the potential link between varicocele and human subfertility was officially acknowledged. This recognition was based on a single case report of a man who had a testis biopsy and was diagnosed with maturation arrest; in this case, bilateral varicocelectomy improved the sperm count and led to coitus pregnancy [5].

Numerous reports that followed suggested that surgical procedures improved semen parameters and that varicocele was more common in men who were not fertile [6]. When Nilsson and colleagues reported in 1979 that they were unable to show any therapeutic impact on infertility in males after ligation of a varicocele, the debate on the significance of varicocele as a cause of male infertility began [7]. Varicocele is currently the most prevalent cause of male infertility globally, and the most common male infertility surgery performed globally is varicocele repair [8].

2. Objectives

The current review aimed to highlight on the anatomical consideration, prevalence, incidence, and etiology of varicoceles, pathophysiology of infertility with varicocele, diagnosis, and management.

3. Anatomical consideration of varicocele

The testicular, vassal, and cremasteric arteries provide the testis with its arterial blood supply. Even when the testicular artery divides, all three arteries anastomose at the testis level to provide a sufficient blood supply [9].

There are several individual variances in the more complex venous drainage. The pampiniformes plexus, the drainage from the testicular vein, pudendal veins, and cremasteric veins are a network of connecting veins located above the testis [10].

The testicular vein often splits from a single vein and enters the inferior vena cava on the right and the renal vein on the left. According to venographic investigations, there exist contacts between the testicular vein and the inferior vena cava below the level of the renal veins, and the left testicular vein can seldom ever enter the latter [11]. Additionally, the left and right testicular venous systems communicate with each other [12]. A quickly growing left-sided varicocele should always prompt a left kidney examination since rare malignant illness of the left kidney spreads down the renal vein and blocks the testicular vein's exit [13].

4. Prevalence and Incidence

About 15% of couples of reproductive age experience infertility, making it one of the major public health concerns [14]. In 40% to 50% of instances of infertility, the male component is implicated [15].

5.

6.

Idiopathic infertility, the most prevalent kind of male infertility, is defined by the presence of one or more abnormal semen parameters without a known cause [16]. Varicocele, another major cause of male infertility, was reported to afflict 16.6% of 7.802 men referred for infertility in a European research [17]. Infertile males with varicocele makes up 21% of the Greek population, according to estimates [18].

The fact that varicocele is 2-3 times more common in men who consult infertility clinics than in men in the general population or with confirmed fertility may be one of the reasons for the association between varicocele and infertility [17–20]. The findings of several research, however, are quite debatable. Between 4% and 30% of the general population has varicocele [21, 22].

Additionally, the prevalence among infertile males varies from 17% to 41% depending on the study [23]. This difference suggests that either the diagnosis of varicocele is very subjective and differs among researchers, or the prevalence of the disorder varies greatly among the various populations.

It's interesting to note that a 1992 WHO multicenter research revealed that the prevalence of varicocele in infertile couples ranged from 6% to 47% depending on the geographic location [24]. Given that congenital or acquired idiopathic testicular failure frequently co-occurs with varicocele, the fact that varicocele is a prevalent issue among infertile men does not necessarily mean that it is the primary cause of infertility [25]. It is noteworthy that varicocele is seen on the left side in 78% to 93% of cases [26]. The use of contemporary diagnostic techniques, such as conventional or Doppler ultrasonography of the scrotum, may be the cause of the higher frequency of bilateral localization reported in more recent investigations [27].

Given that varicocele is more common in infertile men, particularly those with secondary infertility, it is hypothesized that the disorder gradually impairs testicular function in adult males [28]. Varicocele's persistent and steadily growing effect is thought to be the cause of secondary infertility [29].

Some research recommend surgically repairing varicocele to prevent any adverse consequences on testicular function.

However, those studies have a number of shortcomings since they focus on the higher occurrence of varicocele in older couples rather than menti oning any potential decrease in semen characteristics.

As an exception,just one research [30] indicated gradually declining sper m concentration. Furthermore, regardless of a man's capacity for fertility, a woman's age has a detrimental impact on a couple's ability to conceive. However, other studies do not support the higher occurrence of varicocele in males with secondary infertility, thus care should be used when evaluating the increasing harm that varicocele does to the testes [31].

Etiology and pathophysiology of infertility with varicocele

6.1. Etiology

Varicocele, which is more common on the left side and is most likely caused by asymmetry of the internal spermatic veins leading to changes in biochemical characteristics, is the word for an aberrant dilatation of the testicular veins inside the pampiniform plexus [32]. Varicocele is caused by retrograde blood reflux down the internal spermatic vein, which dilates the pampiniform plexus. The venous system must have reflux before there can be any venous dilatation known as a varicocele.

In the past, it was thought that males without varicocele had it because their internal spermatic veins lacked valves [33].

Due to the internal spermatic vein's physical inclination to drain into the renal vein at a right angle, the left side is often where varicocele forms, making it vulnerable to an increase in venous pressure [34]. The structure of the testicular venous drainage system is surely linked to the reported 90% frequency of isolated left side clinical varicocele [35].

A varicocele may be more prevalent on the left side for anatomical reasons. It has been established that the left internal spermatic vein is 8–10 cm longer than the right. Furthermore, the internal spermatic vein on the right obligatorily empties into the inferior vena cava, whereas the internal spermatic vein on the left enters the left renal vein perpendicularly. Increased hydrostatic pressures may be conveyed to the left testicle's venous drainage system as a result of the combined anatomic reality, potentially leading to varicocele development and venous dilatation [36]. Given that 90% of all varicoceles are left-sided, several ideas have been put up to explain their cause [37].

Coolsaet's 1982 theory:

This argument is predicated on the correlation between the nutcracker phenomenon and the absence of effective valves [38].

Sigmund *et al.*'s 1987 theory:

Sigmund identified two hemodynamic types of reflux using bidirectional echo Doppler ultrasound, clinical examination, and retrograde venography: shunt-type (where reflux falls below communicating veins due to valvular incompetence) and stop-type (where reflux is blocked by component valves above communicating veins in the plexus), in which case blood flows into cremasteric and deferential veins [39].

Shafik's theory from 1991:

The three stages of spermatic vein changes—compensated, hidden, and visible varicocele—are caused by venous hypertension, regardless of the underlying reasons [40].

Although additional ideas, such as the existence of venous occlusion by a retroperitoneal mass, may explain the etiology of secondary varicocele, these are the main theories put out to explain the pathogenesis of initial varicocele [41].

6.2. Pathophysiology of varicocele

A varicocele is characterized by abnormal dilatation and tortuosity of the pampiniform plexus veins. In the majority of cases, the teenager is not aware of the varicocele, which is found during a routine physical examination. Idiopathic varicocele is often asymptomatic, manifesting as asymmetry in scrotal enlargement and heaviness in the scrotum, or in rare instances, testicular discomfort [42].

Around 5% of cases of high-grade (large) varicocele occur worldwide. In male adolescents and adults, varicocele is linked to time-dependent growth stoppage; infertility and testicular growth halt are clearly related [43].

Although it is extremely uncommon and causes some worries, a right side varicocele may be seen in conjunction with a left varicocele (bilateral). All patients who arrive with a single right-sided varicocele should have the potential of thrombosis or blockage of the inferior vena cava ruled out; situs inversus is another cause of a right varicocele [37]. The detrimental impact of varicocele on testicular function is attempted to be explained by the following theories:

Hyperthermia:

Varicocele is linked to altered spermatogenesis and increased testicular and scrotal temperatures. According to experimental research, spermatogenesis works best at temperatures below body temperature. Temperature affects several of the enzymes that the testis uses to synthesize DNA at its best [44]. Heat exchange is made possible by the testis's scrotal location and the cooling mechanism supplied by the pampiniform plexus, which encircles the testicular artery and controls the ideal temperature for spermatogenesis [45].

Reactive oxygen species

Among the most potent types of free radicals are reactive oxygen species (ROS), which include hydroxyl radicals, superoxide anion, and hypo-chloride radicals. Varicocelectomy lowered seminal ROS levels and improved antioxidant capacity levels, while varicocele is linked to increased seminal reactive oxygen species and decreased seminal total antioxidant agents [46].

Hypoxia and “adrenal reflux”

Blood stasis in the pampiniform plexus may alter the testis's aerobic metabolism and partial oxygen pressure [47].

Patients with varicocele have been shown to have blood reflux down the testicular vein [48].

Thus, it is postulated that testicular injury results from exposure to adrenal or renal metabolites. Nevertheless, there is no documentation of the adrenal or renal metabolites at the testicular level [49]. In rats with experimental varicocele, adrenalectomy did not lessen the varicocele's effects [50].

Endocrine imbalanced:

The luteinizing hormone (LH) regulates Leydig cells, which are in charge of producing testosterone. According to some research, varicocele may have an impact on the amount of testosterone in the blood, although intra-testicular testosterone plays a crucial role in controlling spermatogenesis [51]. Similar to hypergonadotropic hypogonadism, elevated LH serum levels and an aberrant response to gonadotropin releasing hormone (GNRH) may indicate a disruption of the hypothalamic-pituitary-gonadal axis, which regulates testosterone levels and spermatogenesis [52]. Leydig cell hyperplasia, a documented histological finding in varicocele testicular biopsies, is caused by elevated LH levels [53].

Testicular pathology associated with varicocele:

Testicular hypotrophy

Spermatogenesis is the testicular function most impacted by varicocel [54]. Adolescent testicular development stop is also linked to varicocele [55]. One possible indicator of testicular injury in teenage varicocele is testicular growth stop. Time affects testicular hypotrophy [56]. The risk of testicular growth stoppage was shown to be correlated with the degree of varicocele in adolescents [57].

Histopathology

Males with varicocele have a wide range of abnormalities on testicular biopsy. The most frequent findings include sloughing of the germinal epithelium, spermatogenesis halt, reduced spermatogonia per tubule, and hyperplasia of the leydig cells [58]. The delivery of oxygen and glucose via these structures may be impacted by the often seen thicker basement membrane of somniferous tubules and proliferative endothelium lesions [59].

6. Patient evaluation

6.1. Diagnosis of varicocele

Numerous diagnostic procedures can be employed to validate these clinical results, particularly in minor and subclinical varicocele, notwithstanding the significance of the clinical examination in varicocele diagnosis [60].

Clinical diagnosis:

Numerous varicoceles will go undetected until the physician finds them, but they can cause scrotal pain, particularly if they are big. The patient may observe swelling around the testis if the varicocele is very big [4]. Reflux in the internal spermatic vein without palpable pampiniform plexus is known as subclinical varicocele [61]. In the past, the identification of a group of dilated and convoluted scrotal veins was necessary for the clinical diagnosis of varicocele, and the categorization of varicocele size was basically restricted to palpable and visible forms [62].

There are other grading systems such as:

A-Grahan and Paulson 1980 system [63]:

Grade I: the patient's pampiniform plexus experiences an impulse while doing Valsalva's technique.

Grade II: When the patient is erect, varicocele may be palpated. Grade III: Varicocele is palpable and visible while standing up straight. Grade IV: Varicocele, often secondary varicocele, does not go away while lying down.

B- Oster system 1971 [64]:

Varicocele A: elongation, dilatation, and convolution of the veins around the testis that are felt but invisible.
Visible and palpable varicosity is varicocele B.
Varicocele C: significant alterations that make test distinction challenging and the occurrence of subjective symptoms.

Seminal analysis

Numerous investigations have demonstrated a strong link between varicocele and low-quality sperm. Some simply correlate with a range of distinct characteristics, whereas others do so with all of the parameters [65].

9034 guys from 34 centers across 24 countries who reported as the partner of an infertile couple were included in a 1992 WHO research. The study found that individuals with varicocele had a lower total sperm count per ejaculate and a smaller testis volume [66].

Comparing 40 patients with varicocele to 40 fertile people without, it was shown that all sperm parameters were considerably lower in the former group [67].

According to others, infertility in individuals with varicocele and normal semen parameters is a sign of underlying sperm and molecular function abnormalities [68].

Hormonal assay

Numerous investigations have demonstrated a strong link between varicocele and low-quality sperm. Some exhibit a connection with every metric, whereas others only show a correlation with a number of distinct parameters. Sperm quality may not be directly impacted by prolactine. While FSH is still the preferred hormone in ordinary practice, other hormones may be taken into consideration in specific circumstances. Luteinizing hormone is not a required standard test in the diagnostic work-up of infertile guys. It is still difficult to determine how environmental estrogen exposure affects male reproductive health [69]. A potential hormonal cause of decreased spermatogenesis has been suggested by the discovery that men with varicocele also have aberrant testosterone concentrations, which improve following varicocele treatment [70].

Adjunctive diagnostic tests

Thermography:

Numerous investigations have demonstrated a strong link between varicocele and low-quality sperm. Scrotal thermography has been used to identify the high temperature linked to varicocele; some exhibit a correlation with all measures, while others only show a correlation with a few of distinct parameters. claimed that scrotal thermography might diagnose varicocele with an accuracy comparable to Doppler flow studies [71].

Venography:

Although retrograde spermatic venography is thought to be the most sensitive diagnostic method for scrotal varicocele, its specificity is debatable; that is, while it may be very sensitive for reflux, it is unclear how to interpret it in relation to clinical varicocele and treatment [72].

Radio nucleotide technetium99m pertechnetate scintigraphy :

Another tool for diagnosing varicocele is scrotal scintigraphy, which makes use of radioactive isotopes and gamma cameras (TC99 Technetium). By "pooling" radio nucleotides within the dilated pampiniformes plexus, it can exhibit varicoceles. As with other supplementary testing, scrotal thermography and radioisotope angiography can be helpful in confirming the diagnosis of varicoceles that are clinically obvious; however, these methods should not be utilized to seek for a subclinical varicocele [73].

Scrotal ultrasonography:

Testicular size assessment, detection of obstruction signs, such as rete testicular dilatation, enlarged epididymis with cystic lesion, and lack of vas deferens, exclusion of testicular dysgenesis signs, such as homogeneous testicular architecture and micro-calcifications, and evaluation of blood reflux in men with a vaicocele can all be aided by scrotal ultrasonography [74].

According to reports, color Doppler ultrasonography has a sensitivity of 80–85% and a specificity of 95% [75]. According to a another research, it was 97% [62].

High-frequency sound waves that cause local particle movement inside a medium are what ultrasound is, as the name suggests. As a sound wave travels through a material, the location of the particles will shift, resulting in local periodic displacement of the particles [76].

Principles of applied ultrasonography (us):

Ultrasonography, sonography, or echography are some names for ultrasound imaging. The distance in millimeters between the reflecting surface (tissue) and the ultrasonic source (transducer, or "probe") may be determined using electrical techniques [77].

Ultrasound imaging:

A (amplitude) – mode:

A –mode gives information in only one dimension, which is the axis of the ultrasound beam.

B (brightness) –mode (gray scale image):

When the strength of ultrasound reflection (echo) is displayed as a variation in brightness it is possible to produce two dimensional b-mode images of section within the tissue.

Highly reflective regions are shown as bright areas and echo-free regions are shown as bright area and echo-free regions are shown as (vessels, fluid containing cysts) are shown as dark area.

M (motion) –mode:

M –mode is mainly used in cardiac studies to record motion of vascular structures and the walls of cardiac chambers.

The Doppler ultrasonography:

The Doppler Effect refers to the principle that if sound is reflected from a moving surface, the returning wave is at a different frequency from that of transmitted wave.

Pulsed duplex Doppler US display the flow in area on the corresponding gray scale image as continuous time-velocity wave form.

Color flow Doppler (CFD) US display the color flow superimposed on a gray scale image, thus providing measurement of blood flow within specific blood vessels.

Power Doppler us imaging is Doppler imaging without directionality.

Nearly all scrotal pathologies may now be studied in great detail because to advancements in small parts ultrasonic imaging [78].

Testicular anatomy from ultrasound point of view

Scrotal wall:

The normal scrotal wall is made up of several layers. Sonography is unable to differentiate between them. One echogenic layer that is 2 to 8 mm thick is seen on the scrotal wall [79].

The testis:

With an average measurement of 3.8, 3, and 2.8 cm, the testes are oval structures that measure around 43 x 3 x cm. However, their length, breadth, and antero-posterior diameter range from 3-5 cm, 2-3 cm, and 2-3 cm, respectively; both sides are typically of comparable size [78]. The typical testis shows a homogeneous granular echo texture on sonography, which is made up of evenly spaced medium-level echoes. The mediastinum testis, which runs through the testis in a cranio-caudal direction parallel to the epididymis and is situated at 3:00 or 9:00 o'clock on the transverse section, is commonly observed as a high echogenic line of variable thickness and length in different males, whereas the testis has a smooth oval outline with medium amplitude echogenecity [80]. The testis was separated into the segment centered on the hilum by fibrous septa. The rete testis may be ultrasonographically observed as an ill-defined echo-poor zone near the testicular hilum, occasionally with arbor-like extensions into the parenchyma [78]. These are visible as fine structures at high resolution.

The tunica albuginea:

When seen on an ultrasound scan, the testis appears as a line with a slightly higher echo density surface at a right angle to the ultrasound beam. This is because the tunica albuginea, which surrounds the testis, is folded at the mediastinum testis to produce a linear or sail-shaped intratesticular structure [78].

The epididymis:

The epididymis head, which has a diameter of 6 to 8 mm, is seen sonographically posterior and superior to the testis. The head might be shaped like a triangle, a teardrop, or a crescent [80]. Although the texture may be a little rougher, the echogenicity of the epididymis is comparable to or slightly higher than that of the testis. It's unknown what causes the epididymis to be more echogenic than the testis; it might be that the testis is more uniform.

Histologically, where the epididymis's tubules are bigger and produce more secular echoes [81].

Testicular appendix:

About 30% of people have an appendix testis that protrudes from the testis' superior surface. If it is encircled by the hydrocele fluid, it may occasionally be seen [78].

The spermatic cord:

Sonographically, it is challenging to differentiate the normal spermatic cord from the nearby soft tissue of the inguinal canal since it is located just beneath the skin. Using color follow Doppler sonography or when a hydrocele is present, it may be seen inside the scrotum. The internal spermatic artery may be identified by pulsed Doppler tracing because of its low resistance pattern, which contrasts with the high resistance pattern of the deferential and cremasteric arteries [82].

The arteries that supply the testis are often recognized as a short, convoluted section; however, their spiral shape can be recognized if hyperemia is present or if an ultrasound contrast agent is used. One characteristic that sets the testicular artery apart is its low resistive biphasic wave shape [78].

Both color and grayscale Doppler images clearly show the veins that make up the pampiniform plexus. The internal spermatic vein typically has a diameter of 1-2 mm and does not reflux during valsalva or breathing [78].

Correlation of color Doppler with clinical and venographic findings:

Ten of the fourteen (71%) varicoceles identified by venography in 17 subfertile individuals were clinically palpable, whereas thirteen of the fourteen (92%) were discovered by color Doppler, according to Petro's et al. The color Doppler was positive in the four individuals who had positive venography and normal clinical symptoms [83].

Hoekstra and Witt examined 156 spermatic veins' color Doppler and physical examination results. Veins of questionable palpability had diameters of 2.5+3.5 mm, and reversal of flow was detected in 65% of the cases. They discovered that spermatic veins became palpable at diameters of 3+3.5 mm and that reversal of flow was detectable in all veins larger than 3.5 mm and did not occur in veins smaller than 2.5 mm [84].

It's debatable what non-palpable "color reflux" means. 34 young volunteers with normal sperm counts are the subjects of a study by Meacham et al. In 35% of instances, reflux was found by Color Doppler. However, only 15% had palpable varicocele. Fifty percent of patients without palpable varicoceles had positive Doppler US results. In the lack of palpable varicocele, the author came to the conclusion that it was still unclear how significant reflux was on color Doppler [85].

7. Management of varicoceles

The procedure that is most frequently used to treat male infertility is varicocelectomy. Varicocelectomy is indicated for the following clinically relevant varicoceles:

(1) infertility, especially when sperm quality or semen parameters are compromised.
(2) Hypogonadism.
(3) Pain in the scrotum.
(4) Hypotrophy of the testicles, especially in children
(5) Problems with massive varicoceles' appearance.

Treatment options for varicocele include:

(1) Percutaneous occlusion, which involves injecting different materials intravenously to block the varicoceles, and (2) surgical ligation, which can be done openly or laparoscopically. Inguinal, subinguinal, and high approach (Palomo) techniques are all used in open surgery [86].

Percutaneous varicocele occlusion

More than thirty years have passed since the first description of gonadal vein percutaneous embolization [85].

Both the conventional retrograde occlusion and the more recently reported anterograde method are currently used percutaneous embolization techniques for varicoceles [86]. Because there is no incision or splitting of the abdominal muscles involved, retrograde percutaneous embolization has the major benefit of allowing for a quicker return to regular activities after the surgery than conventional varicocele treatments [87]. With embolization, complications such as contrast response, flank discomfort, embolizing material migration, infection, thrombophlebitis, arterial puncture, and hydroceles are common (9–30%) [88].

Anterograde occlusion can be carried out under local anesthetic, much as the retrograde treatment. Additionally, a shorter operating time (10–15 min) and a lower overall persistence/recurrence rate (5–9%) are linked to the anterograde approach [89]. Testicular shrinkage after treatment, likely due to unknown vascular damage, has been documented in 1% of instances, despite the fact that the complication rate is less than 8% in the majority of series [90].

Furthermore, it has been observed that antegrade blockage might reoccur or persist up to 25% of the time in big varicoceles.

Surgical repair of varicocele

The most common therapy for varicocele is still surgical repair, which can be accomplished by laparoscopic/robotic varicocelectomy, traditional open varicocelectomy (including inguinal and sub-inguinal ligation, and retroperitoneal high ligation), and microsurgical varicocelectomy.

Laparoscopic varicocelectomy

One benefit of laparoscopic varicocelectomy is that it isolates the internal spermatic veins close to the point where they drain into the left renal vein. Since there are just one or two big veins at this level, fewer veins should be assigned.

Furthermore, the testicular artery is frequently clearly distinct from the internal spermatic veins and has not yet branched out. Laparoscopic varicocelectomy has a persistence/recurrence rate of 6–15% [91,92]. Failure often results from the preservation of both the artery and the per arterial plexus of tiny veins (venae comitantes).

According to several studies, recurrence/persistence rates are greater (3.5–20%) when arteries are preserved following laparoscopic varicocelectomy than when the spermatic vessels are ligated [92,93].

Air embolism, unintentional arterial division, genitofermoral nerve injury, hydrocele, intestinal injury, and peritonitis are among the 8–12% of laparoscopic varicocelectomy complications. The most frequent post-operative complication is hydrocele, which results from lymphatic congestion following unintentional closure of the lymphatic vessels [95,86]. The post-operative hydrocele rate can reach 40% if the lymphatics are not purposefully located and maintained [96,97].

Robot-assisted varicoclectomy

There appear to be definite advantages to robotic-assisted varicocelectomy over traditional laparoscopic varicocelectomy, despite the fact that the expense of a surgical robot is undoubtedly a major barrier to its widespread adoption.
The robotic approach has the following benefits: (1) improved dissection precision through 3-dimensional optics; (2) improved instrument handling stability and ergonomics to help surgeons overcome the restricted mobility imposed by the use of straight laparoscopic instruments; and (3) greater degree of freedom in the range and extent of instrument manipulation.

Open varicocelectomy

Different incisions can be used to expose the spermatic arteries at different levels during a conventional open varicocelectomy.

The Palomo procedure, often referred to as high retroperitoneal ligation of varicocele, is carried out by making a horizontal incision that extends medially and inferior to the ipsilateral anterior superior iliac spine. An incision is created in the groin above and lateral to the ipsilateral pubic tubercle, and it extends laterally along the inferior abdominal wall's skin lines in the inguinal approach. In order to distribute the spermatic cord without splitting any abdominal wall muscles or fascia, the open sub inguinal varicocelectomy is incised at the level of the external inguinal ring.

In certain cases, open varicocelectomy can be carried out under local or regional obstruction with or without intravenous sedation, even though it is often done as an outpatient procedure under general or spinal anesthetic [98].

Hydroceles, accidental arterial ligation, testicular atrophy, vas deferens damage, epididymitis, hematoma, and wound infection are among the complications, which occur at a rate of 5–30% [99].

Additionally, compared to alternative treatment methods, the recurrence/persistence rate is substantially greater, ranging from 10 to 45% [100].

Microsurgical sub inguinal varicocelectomy

Varicocelectomy's persistence/recurrence rate (0–2%) and complication rate (1–5%) were greatly decreased with the use of microsurgical methods [101–103]. The operating duration for each side of this outpatient operation is 25 to 60 minutes, and it can be done under local, regional, or general anesthesia. Microsurgical sub inguinal varicocelectomy has become the primary treatment for male infertility experts in the majority of academic institutions. Due in large part to the positive results that are linked with microsurgical varicocelectomy, this procedure is becoming more and more popular. Microsurgical sub inguinal varicocelectomy might further enhance semen parameters, serum testosterone levels, and testicular volume from pre-operative values with low risks of complications, even in cases of persistent or recurrent varicocele following first varicocele treatment [104]. Under 10–25x magnification, testicular arteries are easily seen and maintained.

Techniques that can make artery identification easier include the use of papaverine for irrigation to widen arteries in spasm and intra-operative microvascular Doppler assessment. Compared to an operational microscope, loupes do not offer the same advantages at a lower magnification level [105, 106]. According to certain research, using loupes in place of an operating microscope and an inguinal approach—where spermatic vessels are larger—allows for better spermatic artery preservation and a lower risk of chronic pathologic vein reflux [107]. The testicular artery is the primary arterial supply to the testis and should be protected, even though the vassal (deferential) and cremasteric arteries provide supplementary blood flow to the testis. Lastly, post-operative hydrocele formation, the most frequent consequence of non-microsurgical varicocelectomy, can be practically eliminated with microsurgical varicocelectomy since it enables the detection and preservation of the lymphatics. Recurrence rates are lowered to 1% and post-operative hydrocele and testicular atrophy are almost completely eradicated with the use of microsurgical artery and lymphatic sparing procedures.

When a big varicocele is repaired, the number of motile sperm in the ejaculate increases by 143%, and at one year, the pregnancy rate is 47% [108].

Varicocelectomy can result in sperm returning to the ejaculate in 21–55% of cases, even for men with non-obstructive azoospermia. This eliminates the requirement for testicular sperm extraction for intracytoplasmic sperm injection [109].

Comparative studies of the various treatment options

The failure rate for antegrade sclerotherapy was higher (16%) than for laparoscopic treatment (5%), according to a retrospective research comparing the two procedures [110].

The length of stay after the procedure and the duration of the procedure are similar, but the rate of complications from laparoscopic treatment is much higher (13% vs. 5%), with hydrocele being the most frequent (11%). Although the laparoscopic approach has a lower recurrence rate (5% vs. 16% with anterograde and 19% with retrograde sclerotherapy), it is linked to a higher complication rate (15% vs. 5% with anterograde and 9% with retrograde sclerotherapy), according to a retrospective study comparing laparoscopy, retrograde, and antegrade sclerotherapy.

The most frequent side effect of the laparoscopic procedure was hydrocele, whereas the most frequent side effect of the two sclerotherapies was epididymo-orchitis [111].

For the treatment of left-sided varicoceles in children, Pintus et al. compared laparoscopic high ligation with or without artery sparing, open retroperitoneal high ligation (Palomo procedure) with or without artery sparing, and retrograde sclerotherapy. Compared to sclerotherapy (17%), inguinal open varicocelectomy (15%), and open high ligation with arterial sparing, laparoscopic high ligation with arterial sparing had the highest recurrence rate (25%). The recurrence rate was lower for open high ligation (3.4%) and laparoscopic high ligation (0%) when the arteries were not spared [112]. Open inguinal varicocelectomy, laparoscopy without artery sparing, and retroperitoneal high ligation without artery sparing have all been compared to microsurgical varicocelectomy [113, 114]. The majority of researchers support the microsurgical approach because it is linked to a shorter hospital stay and an earlier recovery, lower rates of recurrence and complications, and potentially higher levels of improvement in sperm count and motility, even though microsurgical varicocelectomy typically takes longer to perform and requires additional surgical training and equipment.

However, it should be underlined that the results of the treatments performed are greatly influenced by the expertise and experience of the surgeons. For example, post-operative results and complication rates can be similar to microsurgical varicocelectomy when performed by a skilled laparoscopist [115]. In this study, we assess the sonographic results that may be used to forecast the result of varicocele repair in the management of male infertility or subfertility associated with varicocele.

8. Conclusion:

It can be concluded that varicocele is a major cause of male infertility**.** The prevalence of varicocele in infertile couples ranged from 6% to 47% depending on the geographic location**.** The detrimental impact of varicocele on testicular function is attempted to be explained by hyperthermia, reactive oxygen species, hypoxia and "adrenal reflux", and endocrine imbalanced. The current review showed that the anatomical consideration of varicocele, prevalence, incidence, and etiology of varicoceles, pathophysiology of infertility with varicocele, diagnosis, and management. Numerous diagnostic procedures can be employed to validate these clinical results such as, Clinical diagnosis. Hormonal assay, Adjunctive diagnostic tests, Thermography, Venography, Radio nucleotide technetium99 pertechnetate scintigraphy, and Ultrasound imaging especially Doppler ultrasonography. Treatment options for varicocele include: percutaneous occlusion, surgical ligation, Laparoscopic varicocelectomy, Robot-assisted varicocelectomy, Open varicocelectomy, and Microsurgical sub inguinal varicocelectomy

References

1. Nöske, H. D., and Weidner, W. Varicocele–a historical perspective. World J Urol, 1999; 17:, 151-157.
2. Gurit e: Abulkasim int geschichte der chirurgie. Olms. Hildesheim. Berlin, Germany, 1964; 1: 620649.
3. Benoff, S., and Gilbert, B. R. Varicocele and male infertility: part I. Human Reproduction Update, 2001; 7(1): 47-54.
4. Anne M. Jequier, Mele Infertility. A Guide for the Clinicien by Handcover. 2000.
5. Tulloch S. A consideration of sterility factors in the light of subsequent pregnancies. Edinburgh Med. J., 1952; 59: 29-34.
6. Dubin I and Hotchkiss R: testis biopsy in sub fertile men with varicocele. Fertil. Steril (1969); 20:50-75.
7. Nilsson S, Edvinson A & Nilsson B: Improvement of the semen and pregnancy rate after ligation and division of internal spermatic vein fact or fiction?. BJU. (1979); 51: 591-596.
8. Khera M and Lipshultz : Evolving approach to the varicocele, Aru, Elim, North Am. (2005), 35: 183-189.
9. Parrott IS, and Hewatt. Ligation of the testicular artery and vein in adolescent varicocele. J urol 1994; 152 (2 pt2): 791.mellinger BC. Varicocelectomy. Tech urol; 1995;1(4):188.
10. Lechter A, Lopez G, Martinez. Anatomy of the gonadal veins: a reappraisal. Surgery; 1991:109(6):735.
11. Campobame P. Bhan Venography in adolescent varicocelectomy: a modified sangical approach. J Pediatr Surg. 1997; 32(9):1298.
12. Shafik A, Mostafa A Olfat S, et al, testicular veins: anatomy and role in varicocelogenesis and other pathologic conditions, Urology. 1990; 35(2):175.
13. 8. World Health Organization. Laboratory manual for the examination of human semen and sperm-cervical macus interaction. 4th Ed. New York USA: Cambridge Univeraky Press, 1999.
14. Snell Rs; The abdomen; part1, abdominal wall in; Clinical Anatomy, 7th Ed, 2004; 4,174-176.
15. Speroff L, Glass RH, Kase NG. Clinical Gynecologic Endocrinology and Infertility. 6th Ed. Baltimore USA: Lippincott Williams & Wilkins; 1999. Infertility, pp. 201-246.
16. Baker HW. Male infertility. Endocrinal Metab Clin North Am. 1994:23:783-793
17. Nieschlag E. Classification of andrological disorders. In: Nieschlag E, Behre HM, editors. Andrology. Berlin, Heidelberg, New York: Spriger-Verlag, 1997. pp. 79-83.
18. Papadimas J, Mantalenakis S. Reproductive Endocrinology in males. Thessaloniki: University Studio Press, 1993.
19. Saypol DC. Varicocele. J Androl 1981; 2:61.
20. Redmon JB, Carey P, Pryor JL. Varicocele-the most common cause of male factor infertility? Hum Reprod Update. 2002; 8:53-58.
21. Pryor JL, Howards SS. Varicocele. Urol Clin North Am. 1987; 14:499.
22. World Health Organization. The influence of varicocele on parameters of fertility in a large group of men presenting to infertility clinics. Fertil Steril. 1992; 57:1289-1293.
23. Kantartzi P-D. [Update on the role of varicocele in male infertility). Master degree dissertation. Thessaloniki: 2006.
24. Saypol DC, Lipschultz LI, Howards SS. Varicocele. In: Lipschultz LI, Howards SS, editors. Infertility in the male. New York: Churchill Livingstone; 1983. pp. 299-313
25. Lemach GE, Uzzo RG, Schlegel PN, Goldstein M. Micromurgical repair of the adolescent varicocele. J Urol. 1998; 160:179-181.
26. Das KM, Prased K, Szmigleski W, Noorani N. Intratesticular varicocele: evaluation using conventional and Doppler sonography. Am J Roentgenol. 1999, 173:1079-1083,
27. Lund L, Tang YC, Roebuck D, et al. Testicular catch-up growth aller varicocele correction in adolescents. Pediatr Surg Int. 1999; 15:234-237.
28. Witt M, Lipschultz L. Varicocele: A progressive or static lesion? Urology. 1993; 42:541-543.
29. Gorelick JI, Goldstein M. Loss of fertility in men with varicocele. Fertil Steril 1993; 59:613-616.
30. Chehval MJ, Purcell MH. Deterioration of semen parameters over time in men with untreated varicocele: evidence of progressive testicular damage. Fertil Steril. 1992; 57:174-177.
31. Jarow JP, Coburn M, Sigman M. Incidence of varicoceles in men with primary and secondary infertility, Urology. 1996; 47:73-76.
32. Lund L, Ernst E, Sorensen H and Oxlund H (1998): Biochemical properties of normal and varicose internal spermatic veins. Scard J. Urol Nephrol, 32:47-50
33. Wishahi M, Detailed anatomy of the internal matic vein and ovarian vein: human cadaver study and operative spermatic venography clinical aspects. J Mol. 1991; 145:780-784.
34. Colpi G, Mancini G. Piedriferro F et al, Disorders of blood flow arterial and venous sexual dysfunction and varicocele. In schill, comhair and hargreave (editors) andrology for clinician. Springer-Verlag, Berlin Germany 2006, 97-142-145
35. Gonzales R, Reddy P, Kaye K. Et al. Comparison of Doppler examination and retrograde spermatic venography in diagnosis of varicocele. Fertil Sicril 1993; 40-96

36. Kaufman, D. G., and Nagler, H. M.The Varicocele: Concepts of pathophysiology—Present and future. World J Urol., 1986; 4: 88-91.
37. Elmore JM. Varicocele in adolescents. Emedicine specialties. Lab. Studies section 5, 2006.
38. Lee J, Binsaleh S, Lo K, and Jarvi K. Varicocele: the diagnosticdilemma. J Androl 2008:29:143-146
39. Sigmund, G., Gall, H., and Bähren, W. Stop-type and shunt-type varicoceles: venographic findings. Radiol, 163(1), 105-110.
40. Shalik A. The physiology of testicular thermoregulation in light of new anatomical and pathological aspect. In zorginiotti (editor): temperature and environmental effects on the testis. Plenum. New york.usa. 1991; 153-172.
41. Colpl G. Mancini G. Peidiferro F and Scoroppo L. Disorders of blood flow. arterial and venous sexual dysfunction and varicocele. In Schill, 2006; 157-160
42. Skoog SJ, Robert KP, Goldstein m et al. The adolscent varicocele, what is new with an old problem in young patients? Pediatrics. 1997; 100(1):112.
43. Goldstein, M. (1997). New insights into the etiology and treatment of male infertility. J Urol, 158(5): 1808-1809.
44. Fujisawa, M., Yoshida, S., Matsumoto, O., Kojima, K., and Kamidono, S. Deoxyribonucleic acid polymerase activity in the testes of infertile men with varicocele. Fertility and Sterility, 1988; 50(5):795-800.
45. Zorgniotti, A. W. Testis temperature, infertility, and the varicocele paradox. Urol, 1980; 16(1): 7-10.
46. Ali, A. S., Osama, E. S., & Mohamed, A. S. (2005). Varicocelectomy is associated with increased seminal antioxidant level in infertile men with varicocele. Benha Med Jl. 2005; 22 (3): 469-4784
47. Sharma, R. K., and Agarwal, A. (1996). Role of reactive oxygen species in male infertility. Urology, 48(6), 835-850.
48. Th, W. P., Mali, M., Oei, H. Y., Arndt, J. W., Kremer, J., Coolsaet, B. L. R. A., and Schuur, K. Hemodynamics of the varicocele. Part II. Correlation among the results of renocaval pressure measurements, varicocele scintigraphy and phlebography. J Urol., 1986; 135(3): 489-493.
49. Turner It, Lopez T. Effects of experimental varicocele require neither adrenal contribution nor venous reflux, J Urol 1989;142(5):1372.
50. Sofikitis, N., & Miyagawa, I. (1993). Left adrenalectomy in varicocelized rats does not inhibit the development of varicocele-related physiologic alterations. Inter J Fertil Menopaus Stud., 38(4), 250-255.
51. Su Im, Goldstein M, and Schlegel Po. The effect of varicocelectomy on serum testosterone levels in infertile men with varicoceles, J Urol 1995; 154(5):1752.
52. Kass, E. J., Freitas, J. E., and Bour, J. B. (1989). Adolescent varicocele: objective indications for treatment. The Journal of Urology, 142(2), 579-582.
53. Sirvent Jj, Berant R, Navarro Ma, et al. Leydig cell in idiopathic varicocele. Euro urol. 1990; 17(3):257,
54. Micies, Lilie V, Isvaneski M. Correlation of hormone and histologic parameters in infertile men with varicocele. Urol irt. 1983; 38(3):187.
55. Paduch DA and Niedzieliki Ị Repair versus observation in adolescent varicocele: a prespective study, J VRE; 1997, 158, 1128-1132.
56. Witt MA, Lipshulte varicoes, a progressive or static lesion? Urology, 1993, 42(5):541
57. Thomas Je, Elder Is. Testicular growth arrest and adolescent varicocele: does the varicocele sus make a difference! J Urol 2002, 168 1649-91
58. Aragona I, Ragazzi R, Pozzan GB, et al. Correlation of testicular volume, histology and Irh test in adolescents with idiopathic varcicocele. Euro Urol, 1994; 26(1):61
59. Hadziselimovic F, Herzog B, Jenny P, et al, Testicular and vascular changes in children and adults with varicocele, J Urol. 1989,142(2 p(2) 581
60. EL-haggers, of meligy a, wagelhs et al role of scrotal scintigraphy in dingnosis of shunt type varicocele. This is for master degree in dermatology and venerreology. faculity of medicine, Cairo University1996; 67-70.
61. Comhaire 4, monteyne r, kunnen m. the value of scrotal thermography as compared with selective retrograde venography of the internal spermatie vein for the diagnosis of subclinical varicocele. Fertil. steril 1976; 13:47-56.
62. Pfeiffer F, Timm Weltkamp, Oliver Bunk. Phase retrieval and differential phase-contrast imaging with low-brilliance X-ray sources Nature Physics 2, 258 -261 (2006)
63. Grahan s, paulson d. hydrocele, spermatocele and varicocele, in Bain j & Hafez e (editors): diagnosis in andrology. Martin's nijhoft publishers. The Hague, Boston, USA, 1980; 121.
64. Oster G, Alan perelson & aharon katchalsky. The techniques of network theory are generalized to include irreversible thermodynamic systems. Complex nonlinear systems can be treated in this way in terms of a well-defined mathematical structure. Nature, 1971; 234: 393-399.
65. Marc D. Hauser, Elissa L. Newport, Richard N. Asli. Segmentation of the speech stream in a non-human primate: statistical learning in cotton-top tamarinds Cognition 78 (2001) B53+B64.
66. Who; reference value of semen variables, in: laboratory manual for the examination of human cemen and semen-cervient mucus interaction, 4th edition, Cambridge university press, UK 1992; 20:154-160.
67. Villanueva-diz e, Vega-Hernandeze, diaz-perez m. Et al. Sperm dysfunction in subferetile patients with varicocele and marginal semen analysis andrologia. 1999; 31:263-267.
68. Benaff; cavironmental toms and varicocele. Assist repon technolandeul 1997:9-261-284
69. Dajani E. Pafalis in the pathological diagnosis of male infertility Cur, Diag Path 2001, 11:246-273
70. Brugh V, lipshultz L. Male factor infertility evaluation and management. Med clin.am, 2004, 88-367-385,
71. Repert Brick, Richardt selection of methods for the detection and estion of trends in water quality, 1991; 27:003-813
72. Negler B, Slppes, Vericecelec current concepts and treatment. In lipshultz I and Howard's (eddon); Infenility in the male. 2nd edition. Churchill Livingstone, London UK, 1991; 313.
73. Wheakly J, Fajman W, Witten F. Clinical experience with the radioisotope varicocele sean as a screening method for the detection of subclinical varioveslesj urol. 1982,128:178-187,
74. Hadziselimovic F, Herzog B and Jenny P the chance for fertility in adolescence boys after corrective surgery for varicocele, J Urol, 1995:154:731-733.

75. Loboscos. C.W Doppler in Gattuccio F, Franco De Rose A, Adelfio Latteri M (editors): Varicocele, cofese edizioni, Palermo, Italy 2000, 73-80.
76. Trush A, Hartshernet introduction in peripheral vascular ultrasound edition, Elsevier, London, UK, 2005, 2-12.
77. Hangiandreou N. Physics tutorial for residents. Topics in us: basic concepts and new technology, Radiographics, 2003; 23:1019-1013
78. Cochilin D. The Scrotum Uin Cocklin, Dubbins, Goldberg and balpren (editors), urogenital ultrasound, a text atlas 2nd edition. Tylor & Francu, Londen, UK, 2006, 183-242
79. Siegel on. The scute scroturs. Radiol clin noch am 1997, 35-959-976.
80. Dewsbury K. Scrotal strasonography and update, bye in 2000: 86:143-52
81. Demures o, Adebays, althegali m. Gray scal and Doppler slirasound of mal tema and extra secular structures, West Af, A Med 2001.20.7-12.
82. Grasso I, Pepe P., and Poanella P Volocimetric evaluation of spermatic vessels wah echo color Doppler in patients with idiopathie varicocele Minerva Urol Nefrol, 1997; 49:179-182.
83. Hagued PG, Mehan DJ, Worlacheck JII, Andrus CII and Parra RO laparascopic varicocelectomy preliminary report of a new technique. J Urol., 1992; 147: 73-76.
84. Liguri G, Trombetta C and Garaffa G. Color Doppler ultrasound investigation of varicocele. world jurol 2004;22(5):378-381.
85. Meacham R, Townand R, Rademacher D, et al. The incidence of varicocele in the general population whene evaluated by physical examination, gray scale sonography, and color Doppler sonography, J, Urol, 1994;151:1535-538.
86. Chan PT, Goldstein M. Varicocele: Options for management. AUA News. 2001; 6:1-6.
87. Iaccarino V. Trattamento conservative Del varicocele: Flebografia selettiva skeroterapia delle vene gonadiche. Riv Radiol 1977; 17:107-17
88. Lima SS, Castro MP, Costa OF. A new method for the treatment of varicocele. Androl., 1978; 10:103-106.
89. Li L Zeng XQ, and Li YH. Digital subtraction angiography-guided percutaneous transcatheter foam sclerotherapy of varicocele: A novel tracking technique. AIR Am I Roentgenol. 2009, 193:978-80
90. Punekar SV, Prem AR, Ridhorkar VR, Deshmukh HL, Kelkar AR. Post-surgical recurrent varicocele: Efficacy of intemal spermatse venography and steel-coil emboliration. Br J Urol. 1996, 77:124-8
91. Tauber T, Weizert P. Pleifer KJ, Huber R. Die antegrade sklerosierung der vena spermatica zur therapie der varikozele Eine randomisierte, kontrollierte, prospective studie verhandlungs ber. Dtsch Ges Urol. 1988, 40-239-40
92. Tauber R, Johusen N. Astograde scrotal sclerotherapy for the treatment of varicocele: Technique and fate results. J Urol 1994, 15:386-90 71
93. Abdulmaaboud MR, Shokeir AA, Farage Y, El-rahman AA, El-Rakhawy MM, Mutabagani II. Treatment of varicocele: A comparative study of conventional open surgery, percutaneous retrograde sclcrutherapy, and laparoscopy, Urol., 1998, 52:294-300
94. Cayan S, Kadioglu TC, Tefekli A, Dadioglu A, Tellaloglu S. Comparison of results and complications of high ligation surgery and microsurgical high inguinal varicocelectomy in the treatment of varicocele. Urology 2000,55:750-754
95. Kass EJ, Marcol B. Results of varicocele surgery in adolescents: A comparison of techniques. J Urol. 1992, 148:694-6.
96. Esposito C, Monguzzi GL., Gonzalez-Sabin MA, Rubino R, Mentinare L., Papparella A, et al. Laparoscopic treatment of pediatric varicocele A multicenter study of the Italian society of video surgery in infancy. J Urol. 2000, 163:1944-6
97. Sayfan J. Adam YG, Soffer Y. A new entity in varicocele subfertility. The "cremasteric reflux" Fertil Steril, 1980, 33:88-90
98. Schwentner C. Radmayr C. Lunacek A, et al. Laparoscopic varicocele ligation in children and adolescents using randomized tral. BJU Int. 2006, 98-861-5 alphan blue: A prospective
99. Hassan JM, Adams MC, Pope JC, 4th, Demarco RT, Bruck JW, 3nd Hydrocele formation following laparoscupic variencelectomy Urol. 2006; 175:1076-9.
100. Marmar JL, Kim Y. Suburgumal resurgical vantecelectuny, A technical erisque and statistical analysts of scaun and pregnancy data 1 Urol 1994; 152-1127-32
101. Heu GL, Ling PY, Hoth CH. Wang Cl, Chen CW, Wen HS, et al. Outpatient Vacarierany performed under 7-419-44 thesis Aan J Andral 2005,
102. Szabo R. Kender Revenge retompoctet maly and of the 1964, 12:024-5
103. Sayfan J. Adam YG, Soffer V. A new entity in varicocele subfertility: The "crensasterie reflux" Fertil Steril 1980; 33:88-90
104. Goldstein M., Gilbert BR. Dicker AP Microsurgical inguinal varicocelectomy with delivery of the testis: An artery and lymphatic sparing technique. J Urol 1992; 148:1808-11
105. Grober ED, Chan PT, Zini A, Microsurgical treatment of persistant or recurrent varicocele. Fertil Steril. 2004; 82:718-22.
106. Cayan S, Acar D, Ulger S. Adolescent varicocele repair: Long-term results and comparison of surgical techniques according to optical magnification use in 100 cases at a single university hospital J Urol. 2005; 174:2003-6
107. Castagnetti M, Cimador M, Catalano P. Evolving management of adolescent varicocele. J Pediatr Urol. 2008; 4:107-12.
108. Gontero P, Pretti G, Fontana F. Inguinal versus subinguinal varicocele vein ligation using magnifying loupe under local anesthesia: Which technique is preferable in clinical practice? Urol. 2005; 66:1075-9
109. Su LM, Goldstein M. Schlegel PN. The effect of varicocelectomy on serum testosterone levels in infertile men with varicoceles. J Urol 1995, 154:1752-5 131
110. Kadioglu A. Tefekli A, Cayan S. Microsurgical inguinal varicocele repair in azoospermic men. Urol. 2001; 57:328-33. 132
111. May M, Johannsen M, Beutner S, et al. Laparoscopic surgery versus antegrade scrotal sclerotherapy: Retrospective comparison of two different approaches for varicocele treatment. Eur Urol. 2006; 49:384-7.
112. Beutner S, May M, Hoschke B, et al. Treatment of varicocele with reference to age: A retrospective comparison of three minimally invasive procedures Surg Endosc. 2007; 21:61-5.

113. Pintus C, Rodriguez Matas MJ, Manzoni C, Varicocele in pediatrik patients: Comparative assessment of different therapeutic approaches. Urol 2001: 57:154-7.
114. Ghanem H, Anh T, E-Nashar A, Shamiool R. Subingainak zmien varicocelectomy verses retroperteneal varicocelectomy: Comparative study of complications and surgical outcome. Urol. 2004; 64:1005-9.
115. Watanabe M, Nagal A, Kusumi N. Minimal invasiveness and effectively of subinguinal microscopic varicocelectomy. A comparative study with retropezitoncal high and lsparessupis approaches. Int J Urol. 2006; 12:2928.