Review Article

The role of microsurgical varicocelectomy in treating male infertility

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Abstract: Varicoceles are the most common cause of male infertility. They afflict 15–20% of the general male population and 40% of males with primary infertility. Although multiple treatment modalities exist, including radiographic embolization and laparoscopy, open subinguinal microsurgical varicocelectomy is currently the gold standard of treatment for this condition. In this article, we discuss the role of varicocelectomy in the treatment of the modern infertile male and present a practical, safe, and reproducible technique for the microsurgical approach.

Keywords: Infertility; microsurgery; pampiniform plexus; sperm; testicle; varicocele; varicocelectomy

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Introduction

Varicocele is the most common diagnosis in men presenting with concerns regarding their fertility (1). While the incidence of varicocele has been estimated to be between 15–20% in the general population, this value increases to 35–40% amongst men who present with primary infertility (2,3). Although it is estimated that approximately 80% of men with varicoceles will be fertile and not experience any sequelae as a result of their diagnosis, there is a preponderance of data suggesting that, for some men, the presence of a varicocele can negatively impact both Sertoli and Leydig cell function in a progressive fashion (4-9). Consequently, the evaluation and treatment of varicoceles has become one of the backbones of modern male reproductive medicine.

Pathophysiology and anatomy

Defined as a dilatation or tortuosity of the venous plexus that drains the testicle, a thorough understanding of testicular physiology and relevant anatomy provides the foundation upon which varicocele treatment is based.

While there are several hypotheses that attempt to explain the correlation between varicoceles and subfertility, the most commonly acknowledged mechanism is that of testicular hyperthermia. The male scrotum is maintained at a temperature approximately 1–2 ℃ cooler than core body temperature. This thermoregulation is essential to proper testicular function and is achieved by a number of mechanisms (10). Chief among these is thin scrotal skin, which lacks an insulating layer of subcutaneous fat and the presence of a counter-current heat exchange system based around the pampiniform plexus. First proposed in 1959, this system allows arterial blood to be cooled by testicular venous return prior to reaching the testis (11). Dilatation of this network of veins allows the pooling of blood, which can then act as a radiator and raise the temperature of the entire scrotum (12,13). Although it is unclear exactly why these lower temperatures are so essential for proper testicular function, the detrimental effects of elevated scrotal

temperatures are well documented, both clinically and on a histopathologic level. Varicoceles have been shown to result in the testicular atrophy of both subfertile men and fertile controls while demonstrating an association with relatively thin germinal epithelium and late stage maturation arrest on testicular biopsy (4,14,15).

From an anatomic standpoint, blood supply to the testis, epididymis, and vas deferens is achieved by the following vessels: the internal spermatic artery (also known as the gonadal artery), the deferential artery, and the cremasteric artery (also known as the external spermatic artery) (16). The internal spermatic artery and the deferential artery arise from the aorta and inferior vesical artery, respectively. As such, they both follow the spermatic cord within the internal spermatic fascia and are considered "deep". The cremasteric artery arises from the inferior epigastric artery and supplies the surface of the cord at the level of the external inguinal ring before traveling within external spermatic fascia distally and ultimately anastomosing with the internal spermatic and deferential. It is also considered "deep", but it is important to note that it travels in a distinct fascial compartment from its two cohorts and runs between the internal and external spermatic fascial layers. All three vessels are ultimately responsible for supplying blood to the testis and epididymis, although the majority of blood flow comes from the larger caliber internal spermatic artery.

In regards to venous drainage, all urologists are familiar with the network of veins known as the pampiniform plexus. It is this structure that, when dilated, is responsible for the pathognomonic "bag of worms" first described by Dubin and Amelar in 1970 (17). However, we advocate that successful microsurgeons should strive for a deeper understanding of the spermatic cord's venous vasculature. Fortunately, this is relatively simple if one applies what is readily known about fascial compartments of the spermatic cord and its aforementioned arteries. It is a well-established anatomic maxim that veins typically follow arteries and this statement holds true for the spermatic cord. Consequently, there are three groupings of veins: the internal spermatic veins (also known as the gonadal veins), the deferential veins, and the cremasteric veins. The internal spermatic and deferential veins course within the internal spermatic fascia while the cremasteric veins travel between the external and internal fascial layers. The cremasteric veins are only accessible at the inguinal/subinguinal level and by virtue of this are missed when performing laparoscopic or retroperitoneal repairs. This is significant as studies have shown these vessels to be involved in up to 50% of varicoceles (18). The internal spermatic, deferential, and cremasteric veins intertwine and anastomose several times over as they approach the testis, creating the familiar pampiniform plexus. Collateral venous drainage of the testis is then provided by the gubernacular veins at the base of the testis that later coalesce with superficial scrotal veins. With a subinguinal microsurgical approach, the internal spermatic and cremasteric veins are ligated while venous drainage is maintained through the gubernacular and deferential vessels. If the surgeon elects to deliver the testis and ligate the gubernacular veins, drainage is maintained solely by the differentials.

Other vital structures that are contained within the spermatic cord include the cremasteric muscle fibers, the genital branch of the genitofemoral nerve, and the vas deferens. The cremasteric muscle fibers and the genital branch of the genitofemoral nerve travel alongside the cremasteric vessels between the external and internal layers of spermatic fascia while the vas deferens travels within the internal spermatic fascia and is intimately associated with its own named vessels.

As with all veins, the internal spermatic, deferential, and cremasteric veins possess valves that are designed to help prevent retrograde flow. It is the failure of these valves that is thought to precipitate varicocele formation. Clinically, varicoceles are most commonly found on the left side. This finding is thought to be due to the longer course that the spermatic vessels must take on this side prior to inserting on the renal vein. This longer course means that veins on this side are exposed to greater hydrostatic pressure, potentially leading to preferential valve failure. It is also worth noting is that the insertion of the left internal spermatic vein into the left renal vein is directly perpendicular, allowing for direct transmittance of elevated renal vein pressures down the entire length of the vein. On the contralateral side, the right internal spermatic vein inserts directly on the inferior vena cava after a much shorter course and at an oblique angle. This course is protective and shields the right gonadal vein from experiencing elevated pressures. As a result, isolated right-sided varicoceles are quite rare and the presence of one should always prompt imaging to rule out more sinister pathology.

Treatment indications

Below we will discuss indication for treating varicoceles for men with infertility, hypogonadism, and pain. (This manuscript does not specifically address varicoceles in the

pediatric population, and this condition has its own set of criteria governing the decision to pursue therapeutic correction.)

Male infertility

As per the American Society for Reproductive Medicine and Society for Male Reproduction and Urology's Practice Committee report "*Report on varicocele and infertility*: *a committee opinion*" varicoceles should be treated when each of the following criteria are met:

(I) The varicocele is palpable on physical examination of the scrotum;
(II) The couple has known infertility;
(III) The female partner has normal fertility or a potentially treatable cause of infertility;
(IV) The male partner has abnormal semen parameters (19).

In regards to infertility, scrotal ultrasound has a very limited role in the evaluation of varicoceles. Repair of "subclinical" varicoceles that are visible on ultrasound but not apparent on physical exam has not been shown to improve fertility and as such is not considered standard of care. However, US may be utilized to evaluate for the presence of varicoceles in men in whom an adequate scrotal exam is not possible. Although official, data-driven criteria are lacking, most experts agree that multiple spermatic veins >2.5–3.0 mm in diameter at rest and with Valsalva tend to indicate the presence of a clinically significant varicocele (20).

While intrauterine insemination (IUI) and *in vitro* fertilization/intracytoplasmic sperm injection (IVF-ICSI) are options for couples experiencing varicocele-related infertility, patients should be educated that these approaches circumvent, rather than treat, their underlying pathology. Varicocele repair offers a durable solution for pure varicocele-related infertility with 80% of patients demonstrating improvement in semen analysis parameters and 40% ultimately achieving spontaneous pregnancy (21). Comparatively, IUI and IVF-ICSI are required for each attempt at pregnancy and as a result are much less cost effective (22-24). Even if IUI or IVF-ICSI are ultimately required due to female factors, a recent meta-analysis examining a total of 870 IVF cycles showed that couples in which the male partner underwent varicocelectomy prior to IVF were over twice as likely to achieve a live birth when compared to those who elected observation (25). Varicocelectomy may also improve the male partner's semen analysis parameters to a point where the couple, who may have previously only qualified IVF-ICSI, can become candidates for IUI. Conversely, for couples in which time is a factor, varicocele repair can be deferred until after a pregnancy has been achieved through the use of assisted reproductive technologies (ART). This approach allows the male partner's semen parameters to recover while the female partner is pregnant and optimizes his semen parameters for any future pregnancy attempts.

For men with non-obstructive azoospermic (NOA), varicocele repair has been shown to return sperm to the ejaculate for approximately ~40% of patients with a live birth rate of 6% (26). This is encouraging as return of sperm to the ejaculate may obviate the need for testicular extraction. However, the overwhelming majority of these patients will still require some form of ART and as such some authors have found that directly proceeding to microsurgical testicular sperm extraction (M-TESE) may be a more cost-effective option (27).

Adult men with clinically significant varicoceles and normal semen analysis parameters should be offered observation with annual or biennial semen analyses given the risk for progressive testicular dysfunction (5-8).

Hypogonadism

The idea of varicocelectomy as a treatment for hypogonadism is an emerging and controversial concept. Although early studies did not demonstrate any increase in serum testosterone, these were small in scale and did not specifically screen for hypogonadal men. Rather, the men were primarily infertile with varying hormonal profiles (28,29). Since that time, several studies have demonstrated an increase in serum testosterone for subpopulations of infertile men with both low serum testosterone and hypogonadal symptomology (30,31). In their recent meta-analysis, Li *et al.* found that, on average, men undergoing varicocelectomy experienced an increase in their serum testosterone of 97.48 ng/dL (32). This has led some authors to advocate for adding hypogonadism to the list of indications for varicocelectomy and propose early intervention as a way to avoid future androgen deficiency (8,33). Although these findings are promising, it is important to note that the idea of varicocelectomy as treatment for hypogonadism is still nascent. It not yet considered to be standard of care and has yet to be recommended by any governing body.

Symptomatic varicoceles

The final indication for varicocelectomy is that of pain or discomfort. Typically described as an aching or dull pain localizing to the ipsilateral testicle or inguinal region, there is a tremendous amount of variability in the characterization, intensity, and frequency of this discomfort. Patients may often have other chronic pain syndromes or have sought care from several other providers prior to presentation. We believe that a methodical and conservative approach beginning with lifestyle modifications, judicious non-steroidal anti-inflammatory use, and pelvic floor physical therapy frequently benefit this population prior to proceeding with surgical repair. When conservative measures fail, patients should be counseled that surgery may not relieve their discomfort. There is significant variability regarding the outcomes of varicocelectomy when performed for pain with series' success rates ranging from 53–94% (8).

Options for repair

The foundation upon which varicocele repair is based is that, by interrupting the venous return from the engorged veins of the pampiniform plexus, collateral flow can be forced to egress through the gubernacular and deferential veins and the pooling of blood will be prevented. This must be accomplished while preserving all vital structures of the testicular cord, to include the internal spermatic artery, vas deferens, and spermatic cord lymphatics. A variety of surgical approaches ranging from the classic Ivanissevich and Palomo repairs to laparoscopy have been developed to achieve these goals (34,35). However, due to its high success rate and minimal complication rates, microsurgical varicocelectomy has been well established as the gold standard for varicocele repair in the modern era (36-39). With proper technique and experience, recurrence rates and incidence of hydrocele approach 1–2% and 0–1%, respectively (37). Embolization remains a non-surgical option, but is typically reserved as a second-line treatment for patients who fail surgery as its own failure rate is approximately 4%, significantly higher than that of the microsurgical approach (40). There is also the not-insignificant concern about gonadal radiation exposure in an already vulnerable population.

Technique

In regards to incision site selection, there are essentially two options for microsurgical varicocelectomy: that of the more traditional inguinal approach or a sub-inguinal incision. The inguinal approach, originally described by Ivanissevich, is centered at the external inguinal ring and requires the incision of the external oblique aponeurosis (34). This has the advantage of avoiding distal arborization, leading to the discovery of fewer veins that require ligation and a decreased chance of encountering multiple arteries (41). This can facilitate safe and expeditious dissection, but the incision of the external oblique aponeurosis and subsequent repair is typically associated with greater post-operative discomfort. As such, we prefer the subinguinal approach at our institution.

For the subinguinal approach, the location of the external inguinal ring is identified by invaginating the scrotal skin with an index finger parallel to the spermatic cord as it passes over the pubic tubercle. An oblique 2.5 to 3-cm incision is made along Langer's lines just below this level. Camper's and Scarpa's fasciae are then divided with electrocautery. The wound is deepened with blunt dissection utilizing Army/Navy retractors until the level of the spermatic cord is reached. The cord is most easily identified as it passes over the pubic tubercle. Gentle traction on the ipsilateral testicle and palpation at this level can help identify the cord in challenging circumstances. Once identified, the cord is encircled with the use of the index finger and the thumb, although some surgeons may prefer to use a Babcock instrument. A 1-inch Penrose drain is then looped underneath the cord and used to gently deliver the cord from the wound. If there is any difficulty in elevating the cord, we advocate encircling the cord more distally as posterior cremasteric attachments are frequently encountered as the cord immediately exits the external ring. It is at this point that the floor of the incision is inspected to ensure the entirety of the cord has been isolated and elevated.

With the cord isolated and elevated, the operating microscope is brought into the field and the cord is examined under 20× magnification. The cord is then draped over the operating surgeon's index finger at the distal interphalangeal joint and its contents are evenly spread. Gerald forceps without teeth are used to bluntly perforate the external oblique fascia and access the inside of the spermatic cord, and Bovie electrocautery is used to incise the external and internal spermatic fasciae. Some authors use Jacobsen hemostats to secure the spermatic cord fascial edges for traction, and but we simply let the fascial edges splay laterally with back pressure on the spermatic cord

using the surgeon's index finger. As this approach requires a skilled assistant, some surgeons may prefer to rest the cord on knife handle or a tongue depressor sheathed within a Penrose drain. A key technical aspect of this procedure is to achieve exposure that transforms the spermatic cord from a cylindrical 3-dimensional structure to a more 2-dimensional configuration, facilitating identification of individual vessels and structures. This dissection is carried proximally to the level of the external inguinal ring. Some authors have described placing the testicle on mild traction to further expose the section of the cord that traditionally rests within the inguinal canal (42).

A 3-mm micro-Doppler is introduced before the fine dissection begins. We request that the anesthesiologist keep the patient's systolic blood pressure >100 mmHg to facilitate identification and differentiation of arterial signals. The field is copiously irrigated with 1% lidocaine solution to combat vasospasm, although a 1:5 dilution of papaverine (30 mg/mL) may also be used. Both of these maneuvers are worth emphasizing as they are frequently overlooked and early identification of the testicular artery is absolutely essential for safe dissection. It is with this exposure that we begin to search for the artery with our micro-Doppler probe. Once this has been identified, great care is taken to protect it and re-confirm its identity throughout the dissection. If difficulty is encountered in identifying the artery, the surgeon should confirm that the systolic blood pressure is still > 100 mmHg. If the artery is still not identified, additional trouble shooting should include confirming that the irrigant being used is lidocaine alone and not lidocaine with epinephrine, as this latter solution will cause arterial vasospasm and suppress the Doppler signal. The vas deferens and its vascular packet are then identified and, when possible, brought to the lateral or medial edge of the cord and excluded from the operative field.

It is at this point that we begin to identify and ligate the internal spermatic and cremasteric veins. Fine tipped Bishop forceps are used to grasp the adventitial tissues while a micro-tipped Jacobson clamp facilitates dissection. 3-0 or 4-0 silk ties are passed behind the veins, the micro Doppler is used once more to confirm their identity, and they are then ligated and divided. In order to improve efficiency, we advocate passing the midpoint of the silk tie underneath the vessel before cutting the tie in two, essentially turning what was a single silk tie into two. This minimizes any potential trauma from multiple passes under the same vessel. Some surgeons may prefer to use clips to ligate the vessels, although we have found the use of ties to be more precise in our experience. Any hemorrhage is immediately controlled with judicious use of fine-tipped bipolar electrocautery and saline irrigation. Any lymphatics are identified and preserved in order to minimize the risk of a post-operative hydrocele.

Unfortunately, it is not uncommon to encounter periarterial veins that are intimately associated with the testicular artery or are scarred in as a result of some previous inflammatory response. In these scenarios, we recommend changing the angle of dissection. This can be achieved by approaching the structure in question from the opposite side of the table or even flipping the cord over for a posterior approach. When these maneuvers are unsuccessful, we endorse carrying the dissection superiorly, even if this requires opening the external ring. Due to distal venous arborization, an exceptionally challenging distal venous dissection may be exponentially more feasible at a more proximal location.

Once all visible veins have been ligated and divided, the internal spermatic artery, or arteries, is inspected visually and with the Doppler once more to ensure patency. The vas is inspected as well. The cord and the wound are then irrigated copiously and the cord is returned to its orthotopic position. It is at this point that the cord is inspected in-situ for any collateral venous supply which is subsequently ligated when present. The wound is inspected for bleeding and infiltrated with a generous amount of 0.25% bupivacaine. A cord block is also performed. Scarpa's and Camper's fasciae are then closing with a running 3-0 absorbable-braided suture and the skin is closed with a 4-0 subcuticular monofilament. The wound is then reinforced with Steri-strips and a dry sterile dressing. Finally, a scrotal support with fluffy dressings is applied.

Post-operatively, the patient is instructed to avoid any significant exertion or lifting for approximately 1–2 weeks. He is then seen at a standard 2-week post-op check to evaluate for proper healing. Semen analyses are obtained at 3-month intervals following surgery and patients are advised that they may not see optimization of their semen analysis parameters until 6 to 9 months following surgery.

Complications

Complications unique to microsurgical varicocelectomy include recurrence, hydrocele, testicular artery injury and vasal injury. Fortunately, these are all quite rare and their rates compare favorably to those of other surgical

approaches and radiographic embolization (37,40,43).

Testicular artery and vasal injury are considered by far to be the most catastrophic complications and should be considered "never ever" events as they can result in loss of the testicle and obstruction of the ipsilateral delivery of sperm, respectively. While vasal injury can be addressed with immediate repair using standard vasectomy reversal technique, arterial injury is almost never salvageable. Pediatric patients can arguably tolerate proximal ligation of the testicular artery reasonably well due the plasticity afforded by their age and collateral flow from the deferential and cremasteric arteries, but adults are almost completely dependent on their testicular artery for adequate gonadal perfusion (44). Fortunately, the incidence of testicular artery injury during varicocelectomy is less than 1% and careful use of the micro-Doppler and adherence to basic surgical principles should drive the incidence of this complication to almost zero (45,46).

Hydrocele following varicocelectomy is the result of inadvertent ligation of efferent lymphatics and is managed identically to idiopathic adult hydrocele. In the past, non-microsurgical approaches resulted in hydrocele formation in approximately 7% of patients while the introduction of the operating microscope has reduced this incidence to 0–1% (37,43,47). In our experience, patients with a history of prior inguinal or groin surgery are at a slightly higher risk for this and are counseled accordingly. The vast majority of hydroceles occurring after varicocelectomy in our experience are reactive and resolve on their own over several weeks to months after surgery. For the rare patient who develops a persistent hydrocele post-varicocelectomy, hydrocelectomy remains the gold standard to resolve the condition.

Varicocele recurrence rates are similarly low and range from 1–2%. These are often thought to result from the collateralization of cremasteric veins that can be missed before they enter at the level of the external inguinal ring (16). Also, smaller veins that may have been missed during a patient's initial surgery can become engorged due to physiologic shifts and similarly incite recurrence. In these scenarios, both repeat surgery and radiographic embolization may be considered viable options as they both display success rates in excess of 90% (10,40,48).

Conclusions

Varicoceles remain the most common cause of male-factor infertility. Fortunately, they are also the most treatable. Microscopic varicocelectomy is a safe and reliable outpatient procedure that allows the majority of affected patients to optimize their fertility in a durable fashion. We believe that the detailed technique presented above offers a safe and effective approach that skilled microsurgeons can refer to while performing this staple of men's microsurgery.

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Footnote

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