Varicocele

9

Karthik Gunasekaran

9.1 Introduction

The pampiniform plexus is a venous network that drains blood from the testis. A varicocele is an abnormal dilatation of the pampiniform plexus and usually occurs on the left side. The incidence of varicocele is almost as high as 15 % in the general population afflicting up to 35 % of men with primary infertility and 81 % of men with secondary infertility. Although other studies have found an equal incidence of varicocele among both primary and secondary infertility, this discrepancy remains to be explored (Jarow et al. 1996; Gorelick and Goldstein 1993; Saypol 1981). A recent Cochrane review suggested that varicocele is the most frequent physical abnormality found in subfertile men (Kroese et al. 2012). The varicocele has always been a controversial topic and highly debated among specialists in the field. The effect of varicocele on semen parameters, its association with infertility, and its effect on pregnancy rates have been avidly debated. Subfertile men show a higher incidence of varicocele prompting researchers to believe that the varicocele is a cause of infertility (WHO 1992). Further, repair of varicocele has seemingly documented some beneficial effects (Newton et al. 1980). However, critics are skeptical as the effects of varicocele repair are inconsistent across studies.

The aim of this chapter is to throw light on the current concepts on the etiology of varicocele, its pathophysiology, its association with infertility, and choice of treatment and finally to look at the cost–benefit analysis in treating it. This hopefully would translate into guiding the physicians in giving a well-informed choice to the patient.

K. Gunasekaran, MBBS, MS, FIAGES, FIMSA, FECSM
The Metromale Clinic: Super Specialty Clinic for Male Sexual Health and Fertility, No. 1, Crescent Park Street, T. Nagar, Landmark: Near BR Mathsya, Chennai, India
e-mail: gunasek@gmail.com

K. Gunasekaran, N. Pandiyan (eds.), *Male Infertility*,
DOI 10.1007/978-81-322-3604-7_9

9.2 Anatomy and Etiology

Though a varicocele is more common on the left, bilateralism seems to be on the rise with recent data suggesting over 30–80 % of varicocele being bilateral (Gat and Bachar 2004). When an isolated right-sided varicocele is identified, it warrants further investigation to rule out a retroperitoneal anomaly, although it could also be a normal anatomical variant. Why are varicoceles more common on the left side? The left internal spermatic vein follows a perpendicular course that is approximately 8–10 cm longer than the right before it enters the left renal vein. This results in increased hydrostatic pressure in the left internal spermatic vein. Also, the left internal spermatic vein because of its perpendicular insertion allows the direct transmission of renal vein pressure to the left internal spermatic vein. The right spermatic vein enters the inferior vena cava at an angle, thus preventing direct pressure transmissions from occurring (Siegel et al. 2006). Furthermore, the left internal spermatic vein lacks functional valves that can lead to regression of blood. There could be a partial obstruction of the left internal spermatic vein because of the compression of the left renal vein between the aorta and upper mesenteric artery (nutcracker syndrome) (Naugton et al. 2001).

9.3 Etiopathogenesis of Varicocele

The etiology of varicocele is multifactorial; similarly the pathophysiology too involves multiple mechanisms. Increased scrotal temperature, high intratesticular pressures, reflux of toxic metabolites, hypoxia and reactive oxygen species, and effect on hormones have all been suggested as possible mechanisms (Fuzisawa et al. 1989; Comhaire 1991). The increased temperature of the scrotum results from reflux of warm blood from the abdominal cavity. This may be a result of the damage or absence of valves in both the internal spermatic vein and cremasteric or external spermatic veins (Goldstein and Eid 1989). The venous tone of the spermatic plexus increases resulting in high hydrostatic pressures.

One study demonstrated that the pressures were actually 19.7 mmHg higher than when compared to the control groups (Shafik and Bedeir 1980). This increased venous pressure can compromise arterial pressure which is required to maintain homeostasis of intratesticular pressure. In the lab looking at fragments from the spermatic vein has revealed alterations in the longitudinal muscle layers, in addition to a decrease in the number of nerve elements and vasa vasorum present in the vessel wall. However, we do not know whether the decrease in the vasa vasorum is a change that suggests causation due to the varicocele or whether this is simply a reactive change of no clinical significance. Nevertheless, these findings suggest a defective contractile mechanism of blood transport through the venous plexus (Tilki et al. 2007). Dysfunction may result by means of chronic vasoconstriction of the spermatic epithelium caused by an increased concentration of toxic metabolites from the adrenal gland (Naugton et al. 2001). Oxidative stress which results from excessive reactive oxygen species (ROS) is increasingly being recognized as a major factor in infertility Said et al. 2005, 1; Agarwal et al. 1994; De Lamirande and Gagnon 1992).

The human body possesses a minimum quantity of ROS for regulating normal sperm function; however, in about 25–40 % of infertile men, the semen contains an excessive amount Marmar 2001; Padron et al. 1997). ROS are needed for regulating normal sperm functions such as sperm capacitation, the acrosome reaction, and sperm–oocyte fusion (De Lamirande and Gagnon 1992; Marmar 2001; Padron et al. 1997).

Elevated ROS and diminished antioxidant capacity have been associated with varicocele (Hendin et al. 1999). However, since both fertile and infertile men show similar findings, it is unclear whether ROS is a cause or consequence. Oxidative stress has also been associated with increased DNA fragmentation in patients with varicocele (Smith et al. 2006). Altered production of steroids in the testis is also a proposed mechanism of varicocele affecting fertility. Some early reports suggested decreased testosterone levels in men with varicocele, whereas others suggested that this was not the cause (Hudson et al. 1983; Swerdloff and Walsh 1975).

9.4 Varicocele and Semen Parameters

The jury has still not given a verdict on the effect that varicocele has on semen parameters and its subsequent effect on male infertility. Semen parameters can either be normal, or there can be different findings like oligozoospermia, asthenozoospermia, teratozoospermia, or a combination of findings. Some studies suggest a gradual deterioration of seminal parameters in the presence of varicocele, ultimately leading to azoospermia (Papadimas and Mantalenakis 1983). Other studies reveal that semen parameters may not be affected at all as there is no significant difference between infertile men and men in the general population with or without varicocele (Redmon et al. 2002). Interestingly, a large-scale study by the WHO showed significantly lower sperm concentration in infertile men with varicocele compared to men with idiopathic infertility, but did not give any evidence regarding the impact of varicocele on sperm motility and morphology (WHO 1992). Strictly speaking a cause–effect relationship between the presence of varicocele and its impact on semen parameters remains unproven. Another plausibility explanation is that varicocele may be an incidental finding in men with idiopathic infertility and men with isolated seminal plasma abnormalities.

9.5 Diagnosis of Varicocele

9.5.1 Physical Examination

A physical exam consisting of inspection and palpation still remains the best method to diagnose a varicocele. Large varicoceles are easily visible and on palpation they give “a bag of worms feel” as described by Dublin. Dublin et al. also graded varicocele based on size. Easily visible varicoceles are grade 3 or large, grade 2 or medium refers to varicoceles that are palpable without a Valsalva, and grade 1 or small refers to varicoceles that are palpable only with Valsalva (Dublin and Amelar 1977).

The grading may be of clinical importance as Steckel and colleagues observed that men with larger varicocele had poorer semen parameters preoperatively and showed an improvement in semen parameters after their repair compared to the repair of small- or medium-sized varicocele (Steckel et al. 1993). It may be important to observe decompression of varicocele in the supine position after examination in the standing position. A varicocele that does decompress after lying down may signify other pathology like a cord lipoma or a hernia.

9.5.2 Doppler Testing

Reflux seen with a Valsalva maneuver can point to a varicocele. Recording the signal and visualizing the images help clinch a diagnosis. Arterial and venous flow should not be confused together. A persistent and reproducible venous rush is to be looked for. A pencil probe Doppler can be used in most cases (Greenberg et al. 1977).

9.5.3 Ultrasound

Ultrasound is being increasingly combined with Doppler for the diagnosis of varicocele. Color Doppler ultrasound had a sensitivity of 93 % and specificity of 85 % when compared to physical examination. It is especially useful in diagnosing a not so easily palpable varicocele (Chiou et al. 1997). Whether subclinical varicocele, picked up only by color Doppler ultrasound, warrants any intervention is a subject of intense debate (Petros 1991).

9.5.4 Venography

Venography is used more as a treatment option than as a diagnostic procedure. It is very sensitive, but not that specific (Ahlberg et al. 1996). It is performed using the Seldinger technique via the right femoral or right internal jugular vein (Sigman and Jarrow 2002). Given the invasive nature of the procedure, routine use is not recommended. However, it has been used as the procedure of choice for detecting persistent varicocele post-surgery (Seyferth et al. 1981).

9.6 Indications for Varicocelectomy

9.6.1 Symptoms of a Varicocele

Large varicoceles are usually symptomatic. Scrotal pain may be an important symptom. Other causes of scrotal pain need to be ruled out before any intervention. Different surgical approaches have been tried with varying results (Karademir 2005; Yeniyol et al. 2003; Chawla et al. 2005).

9.7 Varicocele and Its Effect on Fertility

Varicocele and its effect on semen parameters have been a subject of intense debate. Varicocele can affect sperm concentration, count, and motility. Some studies suggest that varicocele may affect spermatogenesis, regardless of fertility status. Sperm counts in both fertile and infertile men with varicocele were lower compared to controls, but the fertile group showed higher sperm counts (Nagao et al. 1986). The lower concentration and motility reported according to researchers could be attributed to germ cell apoptosis, to increased concentration of reactive oxygen species, or to the presence of antisperm antibodies (Yeniyol et al. 2003). According to MacLeod and colleagues, varicocele induces what is known as a stress pattern on sperm morphology in over 90 % of his infertile patients (MacLeod 1965). Others opined that the stress pattern may not be pathognomonic of a varicocele (Saypol 1981). It is currently difficult to come to any conclusion regarding the effect of varicocele on semen parameters.

9.8 Varicocele in Azoospermia

The complete absence of sperm in the ejaculate in a neat and centrifuged sample is azoospermia. The incidence ranges from 1–15 % of all subfertile men (Jarow et al. 1989; Pagani et al. 2002). The prevalence of varicocele in men with azoospermia is 5–10 % (Matthews et al. 1998; Kim et al. 1999). As early as 1955, Tuloch reported spermatogenesis in a patient treated with varicocele. Motile sperm appeared in the ejaculate of 21–55 % of men following varicocele repair (Lee et al. 2007). This suggests that varicocele repair may obviate the need for assisted reproduction techniques. However, one should also remember that these effects may only be temporary, as in a recent study, more than 50 % men relapsed back to azoospermia in 1 year (Pasqualotto et al. 2006). The varicocele correction or the mere appearance of sperm in the ejaculate post-surgery alone cannot predict whether a natural conception would occur. Also one should be aware of the etiology of azoospermia before initiating treatment. Y-chromosome micro-deletions and karyotype abnormalities are clinically significant findings in men with azoospermia. About 16.6 % of azoospermia men have Y-chromosome micro-deletions or karyotype anomalies (Kleiman et al. 1999). A few studies have addressed the effects of varicocele repair in infertile men presenting with coexisting genetic abnormalities. In a study of varicocele repair in men with infertility and Y-chromosome micro-deletions vs. no deletions, the men with no deletion ($n=4$) were found to have an improvement in their semen parameters, while men in the deletion group ($n=5$) did not exhibit any improvement (Dada et al. 2007). Importantly, one must understand that, most of these studies are based on small case series, much larger randomized controlled trials may solve the debate on whether varicocele surgery can be offered to select subgroups of men with azoospermia. Currently there is low-quality evidence to suggest that varicocele surgery may be no better than expectant management (Biyani et al. 2009).

9.9 Varicocele and ICSI

A common question troubling infertility specialists who treat patients with low sperm counts and coexistent varicocele is whether to surgically correct the varicocele or to offer ICSI directly. The points in favor of a varicocele repair would be that if the procedure is successful, then there would be a paradigm shift in the concept of *ICSI for all patients presenting with male infertility (*ASRM 2004). On an additional note, if the couple has a good prognosis of a future natural pregnancy, a varicocele repair would be cost-effective (Penson et al. 2002).

9.10 Treatment of Varicocele

9.10.1 Antegrade Sclerotherapy

Tauber described sclerotherapy for varicoceles as early as 1988 (Tauber and Johnson 1994). High scrotal incision is made and the cord is hooked out. The large spermatic vein is injected with contrast medium. It is then injected with sclerosing agent mixed with air. Zucchi et al. compared sclerotherapy to surgery and showed that results were comparable (Zucchi et al. 2005).

9.10.2 Percutaneous Occlusion

The procedure is usually performed on an outpatient basis with IV sedation. Various techniques like balloon or coils can be employed to achieve occlusion of the internal spermatic vein. Though a surgical procedure can be avoided, still the effect of radiation lingers and patients are concerned about this. There is also a risk of recurrence and balloon migration. Success rates for venous occlusion are less than that of surgery (Pryor and Howards 1987).

9.11 Surgical Strategies

9.11.1 Retroperitoneal Approach

The idea is to ligate the internal spermatic vein retroperitoneally. It is hypothesized that at this level the number of veins that needs *ligation is less and hence recurrence is less* (Cockett et al. 1984). This is also known as the modified Palomo *technique. Palomo in his original description ligated the artery along with the internal spermatic vein* (Palomo 1969). In the modified Palomo technique, the artery is spared (Wright and Goldstein 1994). A known pitfall to this technique is that there is no scope for ligation of the cremasteric vessels which can cause recurrence (Coolsaet 1980).

9.11.2 Laparoscopic Varicocelectomy

This technique is very similar to the modified Palomo technique with the exception of a laparoscopic entry with carbon *dioxide insufflation. Hydrocele appears to be a common complication associated with varicocele. Some surgeons do a bunch ligation of the artery, vein, and lymphatics in the laparoscopic approach. Contrary to popular belief, ligation of the testicular artery does not result in testicular atrophy, and a single group proved lower recurrence rates with the en masse technique (*Kass and Marcol 1992). However, Goldstein and colleagues had lower success rates when the testicular artery was divided (Wright and Goldstein 1994).

9.11.3 Inguinal Approach

This technique aims to tackle the internal spermatic vessels in the inguinal canal. The vessels here are more visible because of the size, and an important advantage is that the external cremasteric vessels may be ligated here (Ivanissevich 1960). A Doppler probe may be utilized to identify and spare the artery along with an operating microscope (Goldstein et al. 1992). Some authors advocate the delivery of the testicle through the inguinal wound to ligate all the testicular venous channels (Goldstein et al. 1992). However, this claim has been refuted by others (Ramaswamy and Shlegel 2006).

9.11.4 Subinguinal Approach

The main advantage of this technique is that the inguinal canal is not breached. As the external oblique is not cut, there is less pain and also the chances of injuring the ilioinguinal nerve are less (Marmar and Kim 1994). The external cremasteric vessels can be ligated at this level. The main disadvantage is that this is a more taxing technique due to the fact that the internal spermatic veins are more branched at that level. A microscope may come in handy when this technique is employed due to the smaller size of the vessels here.

Conclusion

Varicocele and its effect on fertility always have been and will continue to be a subject of intense debate in the years to come. Our limited understanding of the *pathophysiology of varicocele and response to treatment continues to evolve over time. Evidence suggests that there could be a significant improvement in sperm concentration and motility in carefully selected patients albeit without any evidence to suggest improvement in spontaneous pregnancy rates. For now, subclinical varicocele is best left alone. Microsurgery through the subinguinal route may be the mainstay of treatment.*

References

Agarwal A, Ikemoto I, Loughlin KR. Relationship of sperm parameters with levels of reactive oxygen species in semen specimens. J Urol. 1994;152:107–10.
Ahlberg NE, Bartley O, Chidekel N, Fritjoffson A. Phlebography in Varicocelescroti. Acta Radiol Diagn (Stockh). 1996;4:517–28.
Biyani CS, Cartledge J, Janetschek G. Varicocele. BMJ Clin Evid. 2009;2009.
Chawla A, Kulkarni G, Kamal K, Zini A. Microsurgical Varicocelectomy for recurrent or persistent varicoceles associated with orchalgia. Urology. 2005;66:1072–4.
Chiou RK, Anderson JC, Wobig RK, et al. Color Doppler ultrasound criteria to diagnose varicoceles: correlation of a new scoring system with physical examination. Urology. 1997;50:953–6.
Cockett AT, Takihara H, Consentino MJ. The varicocele. Fertil Steril. 1984;41:5–11.
Comhaire F. The pathogenesis of epididymo-testicular dysfunction in varicocele: factors other than temperature. Adv Exp Med Biol. 1991;286:281–7.
Coolsaet BL. The varicocele syndrome: venography determining the optimal level of surgical management. J Urol. 1980;124:833–9.
Dada R, Kumar R, Shamsi MB, Sidhu T, Mitra A, Singh S, et al. Azoospermia factor deletions in varicocele cases with severe oligozoospermia. Indian J Med Sci. 2007;61(9):505–10. http://dx.doi.org/10.4103/0019-5359.34519.
De Lamirande E, Gagnon C. Reactive oxygen species and human spermatozoa. II. Depletion of adenosine triphosphate plays an important role in the inhibition of sperm motility. J Androl. 1992;13:379–86.
Dublin L, Amelar RD. The Varicocele and infertility. In: Amelar RD, Dublin L, Walsh PC, editors. Male infertility. Philadelphia: Saunders; 1977. p. 57–68.
Fuzisawa M, Yoshida S, Kojima K, Kamidono S. Biochemical changes in testicular varicocele. Archs Androl. 1989;22:149–59.
Gat Y, Bachar GN, Zuckerman Z, et al. Varicocele: a bilateral disease. Fertil Steril. 2004;81(2):424–9.
Goldstein M, Eid JF. Elevation of intratesticular and scrotal skin surface temperature in men with varicocele. J Urol. 1989;142:7.
Goldstein M, Gibert BR, Dicker AP, dwosh J, Gnecco C. Microsurgical inguinal varicocelectomy with delivery of the testis: an artery and lymphatic sparing technique. J Urol. 1992;148:1808–11.
Gorelick JI, Goldstein M. Loss of fertility in men with Varicocele. Fertil Steril. 1993;59(3):613–6.
Greenberg SH, Lipshultz LI, Morganroth J, Wein AJ. The use of Doppler stethoscope in the evaluation of varicoceles. J Urol. 1977;117:296–8.
Hendin BN, Kolettis PN, Sharma RK, Thomas AJ, Agarwal A. Varicocele is associated with elevated spermatozoal reactive oxygen species production and diminished seminal plasma antioxidant capacity. J Urol. 1999;161:1831–4.
Hudson RW, PEREZ-Marrero RA, Crawford VA, Mc Kay D. Seminal plasma testosterone and dihydrotestosterone levels in males with varicoceles. Int J Androl. 1983;6:135–42.
Ivanissevich O. Left Varicocele due to reflux: experience with 4.470 cases in forty-two years. J Int Coll Surg. 1960;34:742–5.
Jarow JP, Espeland MA, Lipshultz LI. Evaluation of the azoospermic patient. J Urol. 1989;142:62–5.
Jarow JP, Coburn M, Sigman M. Incidence of varicoceles in men with primary and secondary infertility. Urology. 1996;47(1):73–6.
Karademir K, Senkul T, Baykal K, et al. Evaluation of the role of Varicocelectomy including external spermatic vein ligation in patients with scrotal pain. Int J Urol. 2005;12:484–8.
Kass EJ, Marcol B. Results of varicocele surgery in adolescents: a comparison of techniques. J Urol. 1992;148:694–6.
Kim ED, Leibman BB, Grinblat DM, Lipshultz LI. Varicocele repair improves semen parameters in azoospermic men with spermatogenic failure. J Urol. 1999;162(3 Pt 1):737.
Kleiman SE, Yogev L, Gamzu R, Hauser R, Botchan A, Lessing JB, et al. Genetic evaluation of infertile men. Hum Reprod. 1999;14(1):33–8. http://dx.doi.org/10.1093/humrep/14.1.33.

Kroese AC, de Lange NM, Collins JA, Evers JL. Surgery or embolization for varicocele in subfertile men. Cochrane Database Syst Rev 2012;10:CD000479. doi:10.1002/14651858.CD000479.pub5

Lee JS, Park HJ, Seo JT. What is the indication of varicocelectomy in men with non obstructive azoospermia? Urology. 2007;69:352–5.

MacLeod J. Seminal cytology in the presence of Varicocele. Fertil Steril. 1965;16:735–57.

Marmar JL. The pathophysiology of varicoceles in the light of current molecular and genetic information. Hum Reprod Update. 2001;7:461–72.

Marmar JL, Kim Y. Subinguinal microsurgical varicocelectomy: a technical critique and statistical analysis of semen and pregnancy data. J Urol. 1994;152:1127–32.

Matthews GJ, Matthews ED, Goldstein M. Induction of spermatogenesis and achievement of pregnancy after microsurgical varicocelectomy in men with azoospermia and severe oligoasthenospermia. Fertil Steril. 1998;70:71–5.

Nagao RR, Plymate SR, Berger RE, Perin EB, Paulsen CA. Comparison of gonadal function between fertile and infertile men with Varicoceles. Fertil Steril. 1986;46:930–3.

Naugton DK, Nangia AK, Agarwal A. Varicocele and male infertility: part II. Pathophysiology of varicoceles in male infertility. Hum Reprod Update. 2001;7:473–81.

Newton R, Schinfeld JS, Schiff I. The effect of varicocelectomy on sperm count, motility and conception rate. Fertil Steril. 1980;34:250–4.

Padron OF, Brackett NL, Sharma RK, Lynne CM, Thomas Jr AJ, Agarwal A. Seminal reactive oxygen species and sperm motility and morphology in men with spinal cord injury. Fertil Steril. 1997;67:1115–20.

Pagani R, Brugh 3rd VM, Lamb DJ. Y chromosome genes and male infertility. Urol Clin North Am. 2002;29:745–53.

Palomo A. Radical cure of varicocele by a new technique: preliminary report. J Urol. 1969;61:604–7.

Papadimas J, Mantalenakis S. Hormonal profile in infertile men. Arch Androl. 1983;11:73–80.

Pasqualotto FF, Sobreiro BP, Hallak J, Pasqualotto EB, Lucon AM. Induction of spermatogenesis in azoospermic men after varicocelectomy repair: an update. Fertil Steril. 2006;85:635–9.

Penson DF, Paltiel AD, Krumholz HM, Palter S. The cost-effectiveness of treatment for varicocele related infertility. J Urol. 2002;168:2490–4.

Petros JA, Andriole GL, Middleton WD, Picus DA. Correlation of testicular color Doppler ultrasonography, physical examination and venography in the detection of left varicoceles in men with infertility. J Urol. 1991;145(4):785–8.

Pryor JL, Howards SS. Varicocele. Urol Clin North Am. 1987;14:449–513.

Ramaswamy R, Shlegel PN. Microsurgical inguinal varicocelectomy with and without testicular delivery. Urology. 2006;68:1323–6.

Redmon JB, Carey P, Pryor JL. Varicocele – the most common cause of male factor infertility? Hum Reprod Update. 2002;8:53–8.

Said TM, Agarwal A, Sharma RK, Thomas Jr AJ, Sikka SC. Impact of sperm morphology on DNA damage caused by oxidative stress induced by beta-nicotinamide adenine dinucleotide phosphate. Fertil Steril. 2005;83(1):95–103.

Saypol DC. Varicocele. J Androl. 1981;2:61–71.

Seyferth W, Jecht E, Zeitler E. Percutaneous sclerotherapy of varicoceles. Radiology. 1981;139:335–40.

Shafik A, Bedeir GA. Venous tension patterns in cord veins in normal and varicocele individuals. J Urol. 1980;123:383–5.

Siegel Y, Gat Y, Bacher GN, Gornish M. A proposed anatomic typing of the right internal spermatic vein: importance for percutaneous sclerotherapy of varicocele. Cardiovasc Intervent Radiol. 2006;29(2):192–7.

Sigman M, Jarrow JP. Male infertility. In: Walsh PC, Retik AB, Vaughan ED, Wein AJ, editors. Campbells urology, vol. 2. 8th ed. Philadelphia: Saunders; 2002. p. 1475–515.

Smith R, Kaune H, Parodi d, et al. Increased sperm DNA damage in patients with varicocele: relationship with seminal oxidative stress. Hum Reprod. 2006;21:986–93.
Steckel J, Dicker AP, Goldstein M. Relationship between Varicocele size and response to Varicocelectomy. J Urol. 1993;149:769–71.
Swerdloff RS, Walsh PC. Pituitary and Gonadal Hormones in patients with varicocele. Fertil Steril. 1975;26:1006–12.
Tauber R, Johnson N. Antegrade scrotal sclerotherapy for the treatment of varicocele: technique and late results. J Urol. 1994;151:386–90.
The Male Infertility Best Practice Policy Committee of the American Urological Association and The Practice Committee of the American Society for Reproductive Medicine. Report on varicocele and infertility. Fertil Steril. 2004;82 Suppl 1:S142–5.
Tilki D, Kilic E, Tauber R, Pfeiffer D, Stief CG, Tauber R, et al. The complex structure of the smooth muscle layer of spermatic veins and its potential role in the development of varicocele testis. Eur Urol. 2007;51:1402–9; discussion 10.
WHO. The influence of varicoceles on parameters in a large group of men presenting to infertility clinics. Fertil Steril. 1992;57:1289–93.
World Health Organization. The influence of varicocele on parameters of fertility in a large group of men presenting to infertility clinics. Fertil Steril. 1992;57:1289–93.
Wright EJ, Goldstein M. Ligation of the testicular artery during micro surgical varicocelectomy: incidence and implications. Abstract presented at the 89th Annual meeting of the American Urological association. Monterey; 1994.
Yeniyol CO, Tuna A, Yener H, Zeyrek N, Tilki A. High ligation to treat pain in Varicocele. Int Urol Nephrol. 2003;35(1):65–8.
Zucchi A, et al. Treatment of varicocele: randomized prospective study on open surgery versus Tauber antegrade sclerotherapy. J Androl. 2005;26:328–32.