EDITORIAL COMMENT

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Clinical varicoceles are present in approximately 15% of the general male population. The majority of men with varicoceles are asymptomatic and fertile, and only 15–20% of them are experiencing some physical discomfort or fertility problems [1]. Varicoceles frequently appear during early puberty and are recognized to be easily corrected with surgery. There is the hypothesis that the presence of varicoceles is associated with progressive testicular damage, including testis hypotrophy from adolescence onwards, and consequent reduction in fertility.

The authors emphasized the negative role of varicoceles on testicular growth during puberty. However, their results show that mean left testicular volume did not differ among subjects with second or third grade of unilateral left varicoceles. After surgery they observed a significant increase of volume of both testes. A healthy European man has, on average, a testicular volume of 18 ml per testis. The normal range lies between 12 and 30 ml [2]. Testicular volume is largely correlated with sperm production so almost normal testicular volumes in studied subjects predict normal sperm production and normozoospermia, which was found in 85% of men treated for varicoceles.

In subjects with testicular volume exceeding 6 ml and azoospermia or oligozoospermia, elevated FSH indicates primary impairment of spermatogenesis. Within wide margins, the extent of FSH elevation is correlated with the number of seminiferous tubules lacking germ cells (Sertoli cell–only syndrome) [3]. The most frequent cause of Sertoli cell–only syndrome is a deletion of one or more of the AZF regions of the Y chromosome [4]. Complete removal of the AZFa region is associated with Sertoli cell–only syndrome, while complete removal of the AZFb region is associated with spermatogenic arrest [4]. Defects at late, postmeiotic spermatogenic stages are FSH–independent [2] and normal testicular volume with normal FSH levels is observed in these subjects. Recent data shows that new genetic causes may be involved in postmeiotic spermatogenesis disturbances [5, 6].

High gonadotropin levels in serum in combination with low testosterone levels indicate testicular origin of hypogonadism (primary hypogonadism) that is not caused by varicoceles, but rather primary defects of testes such as bilateral cryptorchidism, Klinefelter syndrome, or testicular dysgenesis. In these cases, testicular volume usually is lower than 6ml and testicular growth retardation during puberty is observed, irrespectively of the presence of varicoceles.

Although the treatment of varicocele in adolescents may be effective, there is a significant risk of overtreatment. Approximately 50% of adolescent patients with varicoceles experience a spontaneous increase of testicular volume without any intervention [7]. Kozakowski et al. [8] have shown that among 77 patients, the assessment of peak retrograde flow on the duplex Doppler ultrasound can be a valuable tool in predicting persistent, progressive asymmetry of the testes. They concluded that varicoceles associated with a peak retrograde flow of 38 cm/s or greater and 20% or greater asymmetry of testis volume should be considered for varicocelectomy [8]. Patients with peak retrograde flow greater than 30 cm/s need to be monitored carefully. Those with peak retrograde flow less than 30 cm/s are less likely to require surgery. In recently published paper from this same center, in group of 684 adolescent males, only 0.9% patients were found to have intratesticular varicoceles and high peak retrograde flow [9]. The authors concluded that adolescents with intratesticular varicoceles, not extratesticular ones, should be scheduled for surgery rather than followed. Bogaet et al. [10] investigated the effects of early screening and treatment of varicoceles by antegrade sclerotherapy in adolescent boys on the frequency of paternity later in life. They have shown that there is no beneficial effect of treating varicoceles early in puberty with regard to the chance of successful later paternity [10].

In conclusion, we must remember that the mechanisms by which varicoceles might affect fertility have not yet been explained, and neither have the mechanisms by which surgical treatment of the varicoceles might restore fertility. Infertility in adults may also be associated, despite disturbances in spermatogenesis, with several aspects of abnormal function of spermatozoa such as attachment of the spermatozoon to the egg, failure to undergo a normal acrosome reaction, or the inability of spermatozoa to penetrate the zona pellucida.

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