Pathogenesis of Varicocele

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The logically inaccurate designation varicocele, defined by Ambroise Paré in the sixteenth century as a “vascular plexus filled with melancholic blood”, is taken to mean a phlebectasia of individual or all regions of the pampiniform plexus. To understand the pathogenesis of this phlebectasia, one must consider a large number of etiological factors responsible individually or in combination for the genesis of a varicocele.

Knowledge of normal anatomy and physiology is the prerequisite for understanding the etiology and pathogenesis of the disease.

Anatomy

The venous drainage of the testis takes place chiefly via a deep network of veins and a superficial network of veins anastomosing with it. The deep venous network consists of the external spermatic vein, the deferens vein, and the internal spermatic vein. The pampiniform plexus is formed from the testicular vein, the epididymal vein, and the accompanying veins. It drains near the inguinal ring after these veins have joined to form the internal spermatic vein, which opens almost perpendicularly on the left side into the left renal vein. The right internal spermatic vein joins the vena cava at an angle of 30–40° about 4 cm below the junction point of the right renal vein. The extended pampiniform plexus constitutes the anatomical substrate of varicocele.

In the literature, numerous factors have been accorded responsibility for the pathogenesis of idiopathic varicocele. Nevertheless, many points remain under dispute. All authors agree on a clear preponderance of varicocele on the left side. Ivanissevich [12] found idiopathic varicocele on the left in 4470 cases. Oster [26] also found an exclusive localization on the left in his 837 cases. Various other authors report a strong preponderance of occurrence on the left, but also found bilateral varicoceles and varicoceles on the right only in an average of 2% – 3% of the cases [9, 16, 20, 29, 32]. However, it must be borne in mind that according to Ählberg et al. [1] the right spermatic vein opens into the right renal vein in 10% and even more frequently according to Brown et al. [2]. Comparable junctional conditions to those on the left side would then be present. Grillo-Lopez [8] made the interesting discovery in 1971 that a situs inversus was present in three cases of isolated varicocele on the right side.
It could be concluded from all these investigations that an idiopathic varicocele very seldom occurs only on the right side, if at all, under normal drainage conditions into the vena cava.

The possible etiological explanations for the almost exclusive occurrence of idiopathic varicocele on the left side are listed below:

a) Congenital and hereditary weakness of the connective tissue and of the vessel walls [9, 10, 15], which predisposes to status varicosus.

b) Defective development of the cremaster muscle and congenital atonia of the scrotum [7].

c) Raised hydrostatic pressure due to greater length of the left internal spermatic vein [9, 30].

d) Unfavorable inflow conditions at the junction with the left renal vein due to: 90° junction [23, 25], raised pressure of the renal vein compared to the vena cava [19, 28], or additional elevation in pressure by clamping of the renal vein at the angle between the aorta and the superior mesenteric artery [10, 15, 24].

e) Compression from outside by lymph nodes [25], or a course between inferior mesenteric artery and aorta [5, 18].

f) Congenital absence of valves or closure insufficiency of valves present [1, 3, 11, 13, 14, 21, 28, 31].

Point f) requires elucidation. The congenital absence or closure insufficiency of valves was postulated as the cause for the development of a varicocele as early as 1909 [11], but this hypothesis was rejected in the 1930s, in some cases on the basis of inexact investigation [25]. However, this etiological factor gained increasing attention in recent years as a result of investigations of pathological anatomy on autopsy material and the introduction of transfemoral retrograde phlebography [3, 14, 21].

The nature and position of the valves or their absence or insufficiency were demonstrated very inhomogeneously, and in some cases in a contradictory fashion. Mellin [22] reported in 1970 that the internal spermatic vein has no valves at all; this had already been established by the Göttingen anatomist Henle in the middle of the nineteenth century. Ivanissevich and Gregorini [13] assumed valvular insufficiencies were an etiological factor in the genesis of varicocele in 1918 although they were unable to prove the presence of valves. Gösafay [6], Åhlberg [1], Brown [2], and Kohler [17] were also merely able to confirm radiologically the lack of valves in the left internal spermatic vein by retrograde and anterograde phlebography. Comhaire and Kunnen pointed out in 1974 that a junctional valve can sometimes be lacking in men [3].

In 1974 we investigated the drainage conditions of both internal spermatic veins to clarify the position of valves in the two internal spermatic veins. We also hoped to be able to give at least a hypothetical explanation from the anatomical conditions found for the almost exclusive occurrence of an idiopathic varicocele on the left [21]. The average length of the spermatic vein was 42 cm. Two or more spermatic veins were almost always found. They frequently anastomosed with each other in a manner resembling a rope ladder (Fig. 1). Six of the 50 autopsied cadavers had had an untreated varicocele on the left side. This was shown by the markedly phlebectatic pampiniform plexus and could be confirmed by retrospective anamnestic studies. A