More work needed in examining the relationship between mean platelet volume and inflammation in varicocele pathophysiology

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We read with great interest the article by Coban and colleagues.[1] In this well-designed study, Coban and colleagues evaluate the relationship between varicocele and the mean platelet volume (MPV), mean corpuscular volume, platelet, and platelet distribution width (PDW). They found that MPV increased in patients with varicocele and inclined to decrease again after the varicocele was surgically corrected. A recognizable increase was also available in PDW, but it was not statistically important. The ready presence of these markers at no additional cost may increase their wider use in clinical practice.

Varicoceles are present in 15% to 20% of the normal male population and in about 35% to 40% of men with infertility.[2] Varicocele is associated with other vascular pathologies.[3,4] The pathogenesis of varicocele is retrograde flow into the spermatic vein. The superiority of experimental data from clinical and animal models indicate a detrimental effect of varicoceles on spermatogenesis.[2] Testicular temperature elevation and venous reflux play a significant role in varicocele-induced testicular dysfunction, although the exact pathophysiology of varicocele-induced harm is not completely understood.[2] Varicocele stimulates an inflammatory event that could play a harmful role in spermatogenesis.[5-7]

MPV is a widely used laboratory marker associated with platelet function based on inflammatory conditions. Increased levels of MPV were shown in varicocele,[8-9] cerebrovascular disease, peripheral artery disease, stroke, malignancy, ulcerative colitis, and coeliac disease – all related to endothelial dysfunction on the basis of inflammation.[10,11] IL-1, IL-6 and TNF-α are among the progenitor cytokines that impress the platelet manufacturer. Higher MPV values are also a useful indicator of higher thromboycte activity.[12] The level of platelets is increased in high-grade inflammation, leading to a reduction in the MPV level as a result of the migration of most large reactive platelets to inflammatory sites and intensive consumption of these platelets. In cases of low-grade inflammation, an increase in MPV level occurs as a result of the increased reactive immature platelets.[12] MPV is potential subclinical inflammation marker in patients with familial Mediterranean fever.[13,14]

An important limitation of this study was the relatively small number of patients as varicocele affects about 20% to 25% of the male population. Consequently, MPV, IL-1, IL-6, IL-8 and TNF-α are easy ways to evaluate the relation varicocele pathophysiology and the relevant vascular pathologies. We believe the findings of the current study will lead to further studies examining the relationship between MPV and varicocele.

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