Response: Is Mean Platelet Volume Increased in Behcet’s Disease with Thrombosis?

Behcet’s disease (BD) is a chronic vasculitis with obscure etiology and patients with BD are recognized to be at increased risk for venous and/or arterial thrombosis. Therefore, the disease has been termed as a hypercoagulable/prethrombotic state. Several risk factors associated with the coagulation-fibrinolytic systems have been extensively studied in BD. Tunc et al. (2005) found that plasma platelet-activating factor and P-selectin activities were significantly higher in thrombotic BD than nonthrombotic group. Nakano et al. (2003) found that platelet aggregation was increased in patients with BD when compared with normal individuals. In addition, aggregation response to adenosine diphosphate (ADP) was increased, unchanged or impaired with low-dose ADP (Stathakis 1977; Haim et al. 1984; Wilson et al. 1988). We recently reported that mean platelet volume (MPV) was higher in patients with BD than control and we concluded that increase in MPV was independent from the disease activity and thrombosis was associated with the higher MPV values (Acikgoz et al. 2010). In contrast to our results, Lee and Kim (2010) found that MPV was lower in patients with BD than control groups. In addition, there was no difference in white blood cell (WBC) and MPV in BD patients with or without clinical manifestations such as thrombosis, genital ulcers and uveitis. Accordingly, they concluded that MPV was lower in patients with BD than the control groups and that the BD patients without thrombosis showed no significant difference in WBC and MPV when compared to the BD patients with thrombosis. These results were certainly different from our study. However, many studies conducted on the hemostatic state of BD frequently presented contradicting results.

Studies from Turkey consistently indicated increased activated protein C resistance and Factor V Leiden mutation prevalence in BD patients, whereas studies from Israel and Tunisia did not (Guermazi et al. 1988; Mader et al. 1999). Fusegawa et al. (1991) found increased protein C and S levels in patients with BD. In contrast, Hampton et al. (1991) showed a significantly lower protein C antigen in BD patients without active thrombosis. On the other hand, Lenk et al. (1998) demonstrated normal Protein C and S activities in BD patients without active thrombosis. Similarly, investigations on the fibrinolytic parameters yielded contradictory results in the literature. The tissue-type plasminogen activator (tPA) antigen values were reported as normal or reduced (Aitchison et al. 1989; Hamton et al. 1991; Haznedaroglu et al.1996; Demirer et al. 2000). Normal and increased tPA activity has also been found (Hamton et al. 1991; Haznedaroglu et al.1996; Demirer et al. 2000). The Factor VIII was studied either alone or with von Willebrand factor and was increased or remained unchanged in BD (Wechsler et al. 1987; Conard et al. 1988; Hamton et al. 1991). Together with our study, conflicting results regarding hemostatic parameters in BD may be attributed to genetic variations of the study populations from different ancestries, geographic variations, duration and intensity of the disease, psycho-social stresses, life style and nutritional differences.

In conclusion, alteration in platelet aggregation is the main event leading to imbalance in hemostasis, which may eventually lead to thrombotic disorders. Because BD is a chronic thrombotic disorder, the relationship between platelet function and thrombotic complication warrants further investigation.

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