The Importance of Erythrocyte Aggregation in Blood Rheology: Considerations on the Pathophysiology of Thrombotic Disorders

To the Editor:

I have read with great interest the review by Kroll et al on platelets and shear stress. They state that rheologic factors lead to platelet-dependent arterial thrombosis.

Blood rheology is complex and mainly determined by variables such as blood viscosity, hematocrit, erythrocyte aggregation (EA), and deformability. Hemorheologic factors, through their effects on microcirculation, have long been implicated in the pathogenesis of cardiovascular diseases. EA is one of the important hemorheological determinants that may create problems at the level of microcirculation. It is stated that EA has a direct effect in the formation of thrombosis at low shear conditions, i.e., in veins. Vessel injury in a vein with slowed or arrested blood flow will usually lead to a thrombus that is rich in fibrin and red blood cells (red thrombus). Conversely, a thrombus that forms in the arterial circulation where flow is relatively undisturbed will consist primarily of platelets and some stabilizing fibrin (white thrombus). Although platelet aggregation plays an important role in the mechanism of thrombus formation in the arterial system as suggested by Kroll et al., the supplementary effect of EA in arterial occlusive diseases should not be ignored. Elevated EA might have an indirect role in the formation of arterial thrombosis through its effect on platelets. It is stated that, with the increase in EA, blood viscosity also increases and local blood flow decreases. These events cause local acidosis and platelet aggregation leading to endothelial cell damage.

I would like to share our experience on some rheologic studies in patients with vascular thrombotic diseases. In a recent study, we showed that EA was elevated in patients with coronary heart disease. Atherosclerosis is an age-related degenerative process, and men are more prone to it than are premenopausal women. With this in mind, we investigated whether age and menopause had an effect on EA and found that EA rates were increased in men and postmenopausal women compared with the premenopausal period. However, no age-related change was determined in men. Another disorder, diabetes mellitus, is a prototype disease associated with macrovascular and microvascular complications. Hemorheologic factors have long been implicated in the pathogenesis of diabetic complications. We showed that EA was elevated in patients with diabetic nephropathy. In these studies we found that elevated EA was an independent risk factor for vascular occlusive disorders. Glacet-Bernard et al. have also found that elevated EA was an independent risk factor for central retinal vein occlusion. We had an interesting observation in patients with Behçet’s disease. Not all patients with this disorder succumb to various thrombotic complications. In patients with vascular complications of Behçet’s disease, we found higher EA rates compared to those without vascular problems (manuscript submitted).

It has long been known that elevated EA is an independent risk factor for thrombosis both in arterial and venous systems. Pharmacologic approach to the elevated EA has been a subject of investigation in recent years. Beta blockers, calcium channel blockers, and ticlopidine are only a few drugs shown to decrease elevated EA. However, the exact mechanism(s) of elevated EA in the pathogenesis of thrombotic disorders have not been fully established yet and further studies are required to elucidate them. I believe that at present, a preferably nonpharmacological approach on the management of factors known to affect blood rheology such as stress, cigarette smoking, and abnormal lipid profile might be of benefit to prevent thrombotic diseases.

REFERENCES

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