Tissue Factor Overexpression in Rat Arterial Neointima Models: Thrombosis and Progression of Advanced Atherosclerosis

To the Editor:

We read with great interest the recent article by Hasenstab et al. on the role of tissue factor expression in the progression of atherosclerosis. They concluded that tissue factor plays a direct role in neointimal development by coagulation-independent as well as -dependent pathways. We support their conclusions.

To investigate the role of tissue factor in late restenosis after angioplasty, we previously examined 43 patients with ischemic heart disease who underwent elective percutaneous transluminal coronary angioplasty (PTCA) of isolated stenotic lesions in the left coronary artery. Blood samples were drawn from the coronary sinus before, immediately after, and 4 and 24 hours after PTCA. Plasma levels of tissue factor, thrombin-antithrombin III complex (TAT), and prothrombin fragment 1 + 2 (F1 + 2) were measured by enzyme-linked immunosorbent assay.

We found that levels of tissue factor and markers representing thrombin generation, TAT, and F1 + 2 were significantly elevated in the coronary sinus blood 24 hours after PTCA. It is possible that increased tissue factor expression at injured atherosclerotic coronary lesions enhances thrombin generation in the coronary circulation. Follow-up coronary angiography was performed 6 months after PTCA in these patients. We also found a significant positive correlation between increases in tissue factor levels 24 hours after PTCA and late loss index 6 months after PTCA ($r=0.51, P<0.01$). Surprisingly, no significant correlations were found between levels of TAT ($r=-0.14, P=0.45$) or F1 + 2 ($r=0.01, P=0.98$) and late loss index.

We further divided the patients into 2 groups according to the criterion of late restenosis. Patients with late restenosis showed a significantly higher increase in tissue factor levels 24 hours after PTCA than those without restenosis (1.35±0.12- versus 1.03±0.04-fold increases). However, TAT and F1 + 2 levels were not significantly different between patients with and without late restenosis.

These observations suggest that tissue factor is associated with late restenosis of dilated coronary arteries by a mechanism that is independent of the activation of the coagulation systems, and they support the conclusions of Hasenstab et al.

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