Value of D-Dimer Testing in Acute Aortic Dissection

To the Editor:

We read with great interest the recent review on etiology, diagnosis and management in aortic dissection.1 In their discussion on diagnostic strategies, the authors state that useful biomarkers for aortic dissection are currently absent.

We recently reported on 24 patients with acute aortic dissection in whom D-dimer, a degradation product of cross-linked fibrin, was tested as a part of the initial diagnostic strategy.2 We found that, actually, all patients with acute aortic dissection had elevated levels of D-dimer. In contrast, only 31% of control group patients who had chest pain of other origin—most often coronary artery disease—had increased D-dimer concentrations. Our findings might be explained by activation of the extrinsic pathway of the coagulation cascade at the site of vessel (aortic) wall injury by tissue factor.3 Taking the high negative predictive value (100%) of a negative D-dimer test into account, the role of D-dimer testing in suspected acute aortic dissection might resemble its established role in suspected deep venous thrombosis and pulmonary embolism.4

Thus, we agree with Nienaber and Eagle1 that testing of biomarkers for aortic dissection has low specificity. However, facing the high negative predictive value of elevated D-dimer levels makes laboratory testing a useful tool in the diagnostic strategy of suspected acute aortic dissection.

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Response

Thomas Weber et al1 are to be congratulated for their interest in serological screening, or the biomarker approach to the diagnosis of acute aortic syndrome.

Elevated serum D-dimers, however, are found as a reflection of endogenous fibrinolytic activity naturally counteracting the activation of the extrinsic pathway of the coagulation cascade; the latter is triggered by exposure to tissue factor from the dissected aorta (in other words, the formation of clot in the false lumen or any clot). Thus, D-dimers are specific for fibrinolytic activity triggered by thrombus formation as in coronary disease, pulmonary embolism, deep venous thrombosis, or acute dissection.1–3 Not only is specificity low, but sensitivity also is a problem, because thresholds or cutoffs are not defined yet. Moreover, similar to other biomarkers for dissection, such as smooth muscle myosin heavy chains, or soluble elastin, a bedside test is not available, and subacute dissection will be missed as a result of short biological half-life; conversely, dissection in the evolving phase of intramural hematoma is unlikely to spill biomarkers into the circulation.3

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