Effect of Abciximab on Prothrombin Activation and Thrombin Generation in Acute Coronary Syndromes Without ST-Segment Elevation

To the Editor:

I read the article by Merlini et al. with interest, particularly when the authors concluded that abciximab did not prevent thrombin activation and generation among patients with acute coronary syndrome not undergoing percutaneous coronary intervention. These results were observed despite adequate anticoagulation with intravenous heparin infusion based on the target activated partial thromboplastin time at 24 and 48 hours.

We compared these activities among 70 patients with ST-segment myocardial infarction randomized to reduced-dose alteplase plus abciximab and direct angioplasty plus abciximab. Likewise, we found that abciximab did not suppress the activation and generation of thrombin among those receiving combined pharmacological reperfusion therapy. However, the levels of prothrombin fragment F1+2 and thrombin/antithrombin III complexes were highest at the first hour, and gradually fell to baseline by 24 hours. Although the clinical conditions differed between these 2 studies, administration of a fibrinolytic agent accelerated clot dissolution, which might have led to an earlier and higher peak of thrombin activation and generation activities. But the reason for the persistent elevation of prothrombin fragment F1+2 at 1 month in their patients remained unclear.

In contrast, thrombin activation and generation activities did not change significantly among those randomized to direct angioplasty plus abciximab. Although baseline levels were similar between the 2 groups, these patients had considerably lower levels of prothrombin fragment F1+2 (2.3±1.6 versus 4.2±1.6 nmol/L; P<0.001) and thrombin/antithrombin III complexes (15.7±12.5 versus 33.5±19.9 μg/L; P=0.007) at the first hour. Mechanical dissolution of clots together with potent antiplatelet inhibition in an expanded lumen after angioplasty could have contributed to the favorable hemostatic effects of direct angioplasty. Taken together, these findings suggest that more potent antithrombotic agents are required to prevent thrombin activation and generation among patients who are treated medically for acute coronary syndromes or ST-segment myocardial infarction.

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