Pseudothrombocytopenia After Abciximab (ReoPro) Treatment

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A 46-year-old man received a bolus and continuous infusion of abciximab (ReoPro) after PTCA. A platelet count before the procedure was 270,000/µL. Two hours after the bolus, an automated platelet count from EDTA blood was 92,000/µL (Figure 1). The abciximab infusion was stopped. Four hours after the bolus, the platelet count from EDTA blood was 51,000/µL; at the same time, an automated platelet count from citrated blood was 215,000/µL. A peripheral blood film from EDTA blood (Figure 2) showed marked platelet clumping, which was not present on a film from citrated blood. Platelet counts from EDTA blood reached a nadir of 37,000/µL on day 2 and normalized within 2 weeks. No bleeding complications occurred.

Thrombocytopenia is a well-recognized adverse effect of abciximab therapy and may lead to costly and potentially harmful therapeutic interventions (platelet transfusions, immunoglobulin infusions) or discontinuation of potentially beneficial therapy (ie, abciximab). The thrombocytopenia is thought to be due to immune-mediated platelet consumption or platelet removal from the circulation.

Pseudothrombocytopenia, as in the case described, is an important differential diagnosis of thrombocytopenia because, as an ex vivo phenomenon, it is not associated with bleeding complications and does not require discontinuation of abciximab treatment or intervention with platelet transfusions. It is due to the presence of EDTA as an anticoagulant in the blood-drawing tube. Studies of pseudothrombocytopenia in individuals not receiving abciximab have shown that most of these individuals have antiplatelet antibodies. These are devoid of pathological significance. Although the mechanism of platelet clumping is not entirely clear, it appears that the autoantibodies are directed against the GP IIb/IIIa complex. EDTA is believed to cause a change in the formation of the GP IIb/IIIa complex, making an epitope available to the platelet antibodies. The antibodies then cause platelet activation, with subsequent platelet agglutination. The mechanism of abciximab-associated, EDTA-induced platelet clumping is not clear.

Although pseudothrombocytopenia needs to be ruled out in any patient with thrombocytopenia, this is particularly important in the patient treated with abciximab. The diagnosis of pseudothrombocytopenia will avoid discontinuation of the abciximab infusion and initiation of unnecessary therapies, such as platelet transfusions. When thrombocytopenia occurs after abciximab treatment, a peripheral blood film needs to be reviewed or a platelet count determined in citrated blood.
As Americans, We Should Get This Right
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_Circulation_. 1999;100:1462
doi: 10.1161/01.CIR.100.13.1462

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/100/13/1462.1

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