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Refractory Abciximab-Induced Thrombocytopenia

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A variety of drugs can cause thrombocytopenia. We report on one patient who developed severe refractory thrombocytopenia following abciximab use.

Case Report

A 73-year old white female with a history of coronary artery disease and multiple percutaneous coronary interventions presented with severe, retrosternal chest pain. Electrocardiogram suggested anterolateral ischaemia. She was diagnosed as having unstable angina. Prior to abciximab administration platelet counts were 189,000/mm^3. 4-hour post-catheterization platelet counts were 1,000/mm^3. Peripheral blood smear showed no platelet clumping. Coagulation profile and fibrinogen level were normal. Heparin antibodies and d-dimers were not detected. Platelet antibodies were positive. Despite stopping all suspected agents (aspirin, clopidogrel, heparin and abciximab), repeated platelet transfusions and intravenous steroids, the platelet counts remained suppressed. Intravenous immunoglobulins were given. The platelet counts cyclically trended between 2,000 and 67,000/mm^3, before gradually normalizing by the sixth post-catheterization day.

Discussion

Abciximab-induced thrombocytopenia is likely related to immunologic mechanisms. Abciximab is unique in the way that thrombocytopenia typically occurs within 24 hours of drug administration compared to other drug-induced thrombocytopenias, which require a period of drug administration to induce sensitization. It is hypothesized that the rapid occurrence of thrombocytopenia following initial exposure to abciximab is due to "preformed naturally occurring" antibodies against neoepitopes exposed by alteration of the GPIIb/IIIa molecule. In our patient, thrombocytopenia was refractory and platelet antibodies were present. Eventually platelet counts increased following high doses of steroids and intravenous immunoglobulins. The delayed platelet count recovery despite these therapies suggests that another mechanism may underlie the continuing thrombocytopenia.

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