Profound Thrombocytopenia Caused by Abciximab Infusion following Percutaneous Coronary Intervention

BY BLAKE DALEY, D.O., DAVID MIRANDA, M.D., ANKUR KALRA, M.D., AND DANIEL PEASE, M.D.

A 63-year-old male presented to the emergency department (ED) following a witnessed out-of-hospital cardiac arrest at a local sporting venue. Prior to arriving in the ED, the patient had received multiple defibrillation shocks for ventricular fibrillation (v-fib) without achieving a perfusing rhythm or return of spontaneous circulation (ROSC). Vitals on arrival in the ED were notable for no attainable blood pressure, no heart rate and 90% O2 saturation following rapid sequence intubation. Physical examination was remarkable for no carotid or femoral pulses, no respiratory effort and no withdrawal to painful stimulation. Laboratory data revealed a venous pH of 7.06, lactate of 2.6 mmol/L, troponin I of .192 ng/mL, hemoglobin of 14.1 g/dL, and platelet count of 228,000/mm$^3$. Initial 12-lead electrocardiogram, following ROSC, demonstrated ST-segment-elevation in anterior and precordial leads V1-V5 with a right bundle branch block (Figure 1). Prompt catheterization laboratory activation and subsequent coronary arteriography revealed a culprit lesion in the mid-left anterior descending (LAD) coronary artery (Figure 2).

A drug-eluting stent was placed in the mid-LAD via percutaneous coronary intervention (PCI). The glycoprotein IIb/IIIa inhibitor abciximab was added to the anti-platelet armamentarium, which included aspirin and clopidogrel. The patient had received a loading-dose of intravenous unfractionated heparin as part of upstream therapy.

Subsequent laboratory examination revealed profound thrombocytopenia. Testing at three-hour intervals revealed a baseline platelet count of 228,000/mm$^3$ followed by counts of 76,000/mm$^3$, 13,000/mm$^3$ and 9,000/mm$^3$ (Figure 3). Physical findings included bleeding from the oral mucosa and epistaxis. Abciximab infusion was stopped and a hematology consultation was obtained. Given the significant risk of bleeding associated with such severe thrombocytopenia, platelets and fresh frozen plasma were transfused, despite the attendant risk of occluding the freshly inserted stent.² The patient’s platelet count continued to rise steadily following these interventions, and there were no significant bleeding events. A peripheral smear did not demonstrate evidence of disseminated intravascular coagulation.

FIGURE 1
Presenting 12-Lead ECG Demonstrating ST-Elevation in Precordial Leads V1-V5
Learning points

- Patients receiving abciximab are at risk of severe thrombocytopenia.
- Abciximab-induced thrombocytopenia develops very rapidly.
- All clinicians involved in a patient’s care following PCI should be vigilant about checking platelet counts.

Discussion

Post-PCI thrombocytopenia represents an infrequent but clinically challenging complication of anti-platelet intravenous glycoprotein IIb/IIIa administration. Abciximab-induced thrombocytopenia has been shown to occur in up to 2.9% of patients. In contrast to type II heparin-induced thrombocytopenia, which may develop over several days, abciximab-induced thrombocytopenia develops rapidly. Because of this, platelet counts should be checked at 2 and 4 hours after initial infusion of abciximab. In the event of significant thrombocytopenia following PCI with abciximab, consultation with cardiology prior to discontinuation of antiplatelet therapy or administration of any blood products is absolutely necessary to balance the risk of stent thrombosis versus life-threatening bleeding in this high-risk situation. MM

Blake Daley, David Miranda and Daniel Pease are residents and Ankur Kalra is a fellow in the Division of Cardiology and department of medicine at Hennepin County Medical Center.

REFERENCES