Image Report

Stem Cell INVESTIGATION

HIT the nail on the head

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A 70-year-old male with CAD, DM and HTN presented with progressive symptoms of angina and was found to have an aortic murmur. Cardiac catheterization revealed severe calcified triple vessel coronary artery disease with aortic stenosis. He underwent Coronary Artery Bypass Grafting with aortic valve replacement. Five days after the procedure, he developed thrombocytopenia with greater than 50% reduction in platelet count. His right fingertips developed thrombosis and skin necrosis (*Figures 1*,*2*). There was strong suspicion for heparin-induced thrombocytopenia (HIT) given his high 4T score. He was started on fondaparinux. Anti-platelet factor 4 antibodies and serotonin release assay (SRA) were sent and resulted positive. Gradually, his platelet counts began to increase with fondaparinux. Once they were greater than 50,000, he was taken for angiogram and thrombectomy to the right radial artery with improved circulation. He will continue on fondaparinux for a month and follow up with Hematology as an outpatient.

HIT is a lethal immune reaction to heparin.

HIT results from an autoantibody directed against endogenous platelet factor 4 in complex with heparin causing thrombocytopenia by consumption and arterial and venous thrombosis by platelet activation. Mortality rate can be as high as 20% without treatment.

The 4 T's score is used to estimate the likelihood of HIT based on the degree of Thrombocytopenia, Timing of platelet count drop, presence of Thrombosis, and absence of oTher causes of thrombocytopenia, giving pretest probabilities of low, intermediate or high based on points.

Anti-platelet factor 4 antibody and SRA are two tests used to diagnose HIT for patients with intermediate to high probabilities. SRA is considered gold standard.

The differential diagnosis of HIT includes disseminated intravascular coagulation/sepsis, immune thrombocytopenia, post-transfusion purpura, thrombotic microangiopathy, drug-induced thrombocytopenia, venous thrombosis unrelated to heparin, lupus or antiphospholipid antibody syndrome, and delayed-type hypersensitivity reactions to heparin.

Figure 1 The patient developed right fingertip thrombosis and skin necrosis.

Treatment consists of holding any heparin products and starting non-heparin anticoagulation such as argatroban, Danaparoid or Fondaparinux which can later be bridged to Coumadin or other anticoagulants once platelet counts reach above 150,000. Patients should avoid all heparin

Figure 2 The patient developed right fingertip thrombosis and skin necrosis.

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products lifelong.

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Footnote

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