Title: A Case of Type II Heparin Induced Thrombocytopenia

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Case presentation

A 47-year-old male with past medical history of non-insulin dependent Type 2 diabetes mellitus, hyperlipidemia, Class 3 severe obesity, and obstructive sleep apnea, presented with thrombocytopenia (platelet count of 23,000) that was discovered on outpatient blood work. The patient had been admitted to a different hospital two weeks previously for bilateral pulmonary emboli (he had traveled internationally two weeks before experiencing symptoms). During this previous admission, he had been administered thrombolytic therapy, treated with unfractionated heparin for five days, and discharged on dabigatran with a platelet count of 160,000. On presentation at the authors' hospital, the patient received a V/Q scan that showed a high probability of pulmonary embolism. Venous ultrasound/doppler of the lower extremities revealed acute occlusive deep vein thrombosis of bilateral popliteal veins, as well as duplicate femoral and popliteal veins bilaterally. He was initially treated with unfractionated heparin for less than two hours before switching to dabigatran twice a day for three doses. He was subsequently treated with intravenous argatroban for one day and then transitioned to apixaban 10 mg twice daily. The patient's thrombocytopenia persisted throughout the admission, with platelet count ranging from 19,000 to 24,000. After three days, he was discharged with instructions to follow up at the hospital's outpatient internal medicine clinic. Enzyme-linked immunoassay for heparin-platelet factor 4 (PF4) antibody was positive, supporting a diagnosis of Type II heparin-induced thrombocytopenia (HIT). Carbohydrate antigen (CA) 19-9 was also elevated, suggesting malignancy as a possible cause of hypercoagulability. At a one-month follow-up visit, the patient's platelet count had increased to 122,000. Platelet count remained stable at two-month follow-up, and CA 19-9 had normalized.

Discussion

Heparin induced thrombocytopenia (HIT) is divided into two subcategories. HIT Type I is a benign thrombocytopenia caused by platelet aggregation; it is not immune mediated. HIT Type II is a rarer disorder involving antibodies that form against heparin-platelet factor 4 (PF4) complexes, leading to hypercoagulability and thrombocytopenia. The incidence of HIT Type II ranges from approximately 0.1-5%. Prior research suggests the incidence of HIT may be higher with unfractionated heparin than with low molecular weight heparin. HIT may present with skin necrosis in addition to thrombosis. A decreased platelet count is typically seen within ten days of starting heparin. Multiple laboratory tests are available for HIT, including enzyme-linked-immunoassay and serotonin release assay. Because heparin-PF4 antibodies may be elevated without clinical manifestations of HIT, diagnosis of HIT involves both laboratory testing and clinical correlation. Because HIT is a pro-thrombotic disorder, treatment involves anticoagulation. Alternatives to heparin include direct thrombin inhibitors (argatroban, bivalirudin), and Factor Xa inhibitors (fondaparinux). This case underscores the importance of close monitoring and follow-up for patients treated with heparin. Furthermore, the findings highlight that patients who present with deep vein thrombosis/pulmonary embolism need to be evaluated thoroughly for sources of hypercoagulability, even in the presence of other risk factors such as recent travel. Finally, duplicated lower extremity veins present a risk of masking deep vein thrombosis and need to be documented for future patient encounters.