Heparin-Induced Thrombocytopenia with Thrombosis of the Aorta, Iliac Arteries and Right Axillary Vein Successfully Treated by Low-Molecular Weight Heparin

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Heparin-induced thrombocytopenia is a common finding. Besides an early mild form, due to a direct effect of heparin without clinical relevance, about 5% of patients receiving this drug suffer, within 5-14 days, from a delayed, immune-mediated, sustained and deeper thrombocytopenia [1]. This appears to be due to platelet activation via the Fc receptor [2] and to endothelial cell injury caused by immunoglobulin binding [3] and may predispose to thrombotic complications. Heparin-induced thrombocytopenia and thrombosis syndrome (HITTS) is rarer, occurring in less than 0.2% of patients receiving heparin [4], but is associated with a very high morbidity and a mortality of 18-36% [5]. Thrombosis involves arteries more frequently than veins, leading to acute myocardial infarction, stroke, pulmonary embolism, limb gangrene and end-organ damage [6]. Thrombosis of the aorta has been reported in only very few cases [4,7-9], in some of which it was incomplete, and often had a poor outcome. We report here a case of heparin-induced complete thrombosis of the aorta, iliac arteries and right axillary vein, together with hemorrhagic manifestations, successfully treated with low-molecular weight heparin and oral anticoagulants. The propositus is a 46-year-old bank director, who was admitted on June 23, 1993, to a peripheral hospital because of a traumatic right femoral fracture. His history was unremarkable for coagulation disturbances and he was not taking any drug. Subcutaneous heparin, 12,500 U twice daily, was started prophylactically on admission. Platelet count was $166 \times 10^9/l$. He underwent successful surgery for bone repair on June 28. On July 2, while still on heparin at the same dosage, he suffered from sudden acute ischemia of the left leg. A CT scan showed a thrombus totally obstructing the aorta 15 mm above its bifurcation and extending to the origin of the iliac arteries. Platelet count was $45 \times 10^9/l$ and the partial thromboplastin time (PTT) 52 s. He underwent surgical arteriotomy and thrombectomy and was given heparin by infusion at increasing rates, up to 45,000 U/24 h from July 5. Platelet count ranged between 43 and $67 \times 10^9/l$ and PTT between 22 and 52 s. On July 5, bilateral leg ischemia occurred again and femoral artery recanalization with Fogarty catheter had to be performed twice. Major bleeding occurred intraoperatively and 3 units of red blood cells were transfused. Heparin infusion was continued at 45,000 U/24 h. Thrombocytopenia persisted and PTT rose up to 149 s. Right axillary vein thrombosis and a large hematoma of the right arm occurred. Clinical worsening and complications were most likely related to continuing the patient on heparin. However, we first

saw the patient on July 7, when we were called for a consultation. We made the diagnosis of HITTS and immediately stopped heparin. On the same day the patient was transferred to our University Hospital and given low-molecular weight heparin (Fraxiparine), 4,000 U daily and acenocoumarol, 3 mg on July 7 and 1 mg on July 8 and 9. Platelet count rose to $116-139 \times 10^9/l$ on July 8 and then up to $450 \times 10^9/l$ on July 12; PTT returned to normal or near-normal; the prothrombin time (PT) was 15-25% (INR 2.5-3.5) already on July 9, when Fraxiparine was stopped, and small amounts of acenocoumarol were then needed to keep it at therapeutic levels. The following course was uneventful. Peripheral

Letter to the Editor

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