Heparin-induced thrombocytopenia (HIT) as an unusual cause of acute stent thrombosis

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A 53-year-old female presented with 2 weeks of crescendo angina. Ten weeks before admission, she had successful percutaneous coronary intervention (PCI) to the left anterior descending (LAD), and received aspirin, clopidogrel, and unfractionated heparin. Repeat angiography showed a severe ostial stenosis in the intermediate artery (IM) (Panel A). Percutaneous coronary intervention to IM was commenced after unfractionated heparin. Intravascular ultrasound was used to confirm the presence of thrombus and to exclude coronary dissection (Panel C). White thrombus was aspirated, the IM stented, and intracoronary IIb/IIIa bolus administered. The patient developed chest pain and ST-segment elevation 4 h later. Repeat angiography demonstrated acute occlusive thrombus in the distal left main stem (Panel D). A diagnosis of accelerated thrombosis, secondary to heparin-induced thrombocytopenia (HIT), was confirmed by a thrombocytopenia (platelet count decrease 234 x 10^9 per L to 74 x 10^9 per L), and by a positive platelet aggregation assay (Panel E). Heparin was stopped and lepirudin, a direct thrombin inhibitor with no cross-reactivity to heparin, administered. Unfortunately, the patient died 12 h later from subarachnoid, subdural, and intracerebral haemorrhages (Panel F). Heparin-induced thrombocytopenia is characterized by differential thrombosis and haemorrhage as a result of an immune-mediated reaction in the presence of HIT antibodies. Heparin-induced thrombocytopenia antibodies may be present for approximately 120 days after unfractionated heparin, resulting in accelerated HIT with re-exposure. This case highlights the importance of considering HIT as a cause of acute coronary thrombosis during PCI.

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