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A Case Of Refractory Heparin Induced Thrombocytopenia

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INTRODUCTION

Type II heparin-induced thrombocytopenia (HIT) is caused by antibody production which binds complexes between heparin and platelet factor 4 leading to platelet consumption and thrombosis.

Rarely the antibodies continue to activate platelets in the absence of heparin and can present with thrombosis and falling platelet count over 10 days post heparin exposure, in such cases delayed onset HIT can be suspected. It has been noted that 50% of patients with delayed onset HIT have a platelet nadir of <20 10^9/L which is unusual for HIT. [1] Thrombocytopenia can persist for several weeks following the immunising event despite standard treatment with anticoagulation which can be indicative of refractory HIT.

As delayed onset or refractory HIT are rare events there are no trials regarding best treatment.

CASE PRESENTATION

A 71 year old man attended A+E on day 10 post-operatively following tissue aortic valve replacement for aortic stenosis with hypoxia, dyspnoea and pleuritic chest pain.

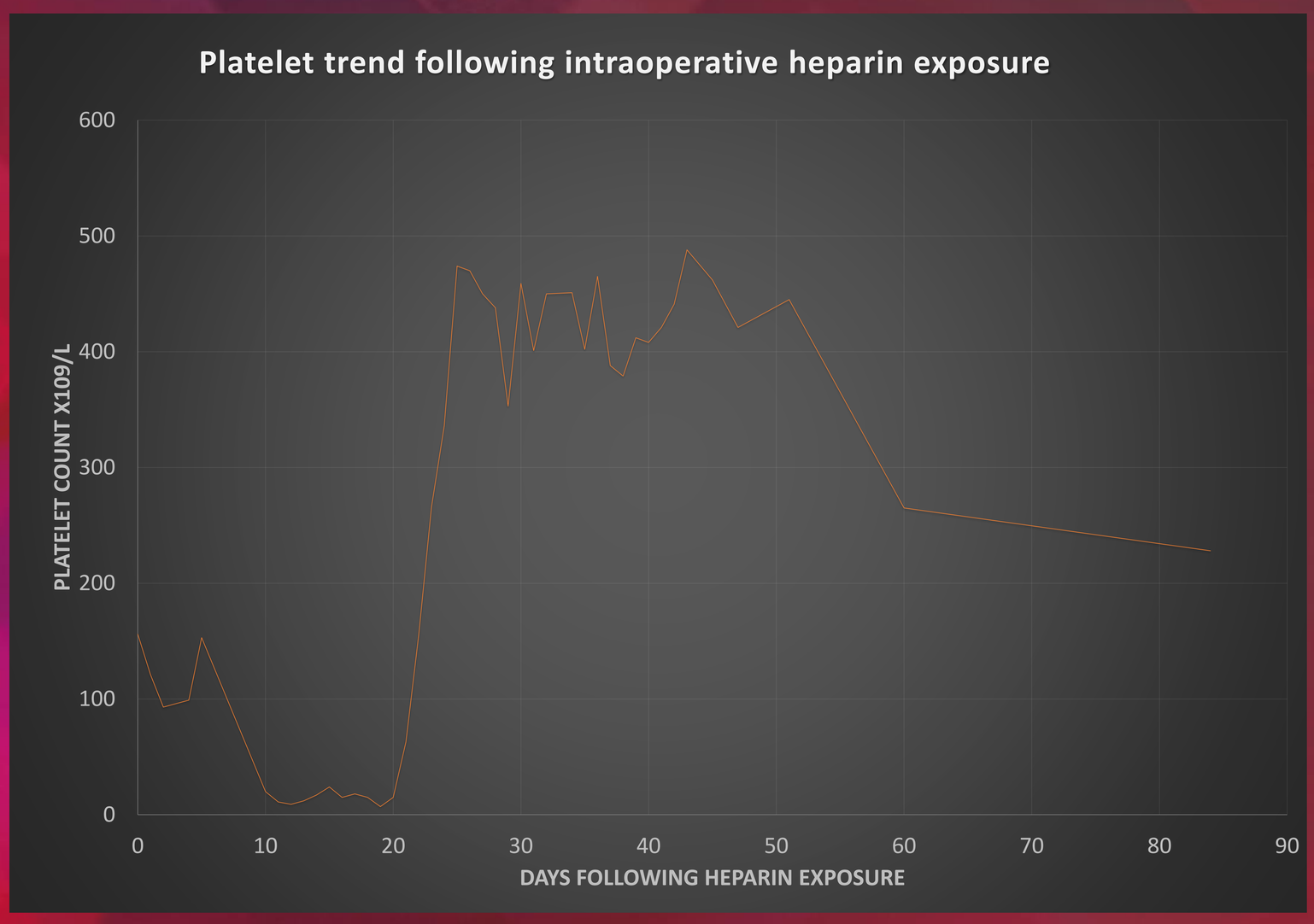
Investigations on admission;

Platelets - 20 x 10^9/L
Hb - 106 g/L,
WCC - 31.46 x 10^9/L
Blood film - left shifted neutrophils
D dimer - 60,938 mcg/L
PT - 15s
APTT - 28s
Fibrinogen - 4.5g/l

CTPA - Saddle PE extending into major branches of the pulmonary artery, with evidence of right ventricular strain.

HIT score - 7
HIT antibodies with Werfen Chemiluminescence AcuStar method (specific IgG PFH- H assay) >125 U/ml.

RESULTS



CLINICAL COURSE

The patient had UFH on cardiopulmonary bypass during cardiac surgery and prophylactic LMWH post operatively for 5 days. The pre-op platelet count was 156 x 10^9/L which fell to 99 x 10^9/L on day 4 post-op but rose to 153 x 10^9/L on day 5 post-op when the patient was discharged. He also received 3000 U intra-arterial UFH during diagnostic coronary angiogram, 77 days pre-op.

Following a diagnosis of HIT, Argatroban was commenced with a target APTT ratio 1.5-3. However, the patient developed haemoptysis and bleeding from his sternal wound. On day 14 post-op, he developed lower limb ischaemia with multiple areas of necrosis on both feet. The APTT ratio was consistently at the lower end of the therapeutic range. We increased the target APTT to 60-70 secs due to worsening ischaemia whilst attempting to minimise any bleeding complications. The platelet count remained between 7 and 24 x 10^9/L at day 20 post-op and a repeat HIT test remained >125 U/ml. Refractory HIT was suspected amongst other differentials. The patient was on antibiotics for pneumonia with good clinical response.

Given the concerns of refractory HIT, IvIg 0.5g/kg/day was commenced on day 21 for 3 days without steroids due to concurrent infection. The platelet count rapidly normalised by day 22 post op to 155 x 10^9/L. The patient improved clinically, with decreasing oxygen requirements and improving peripheral ischaemia avoiding the loss of digits. Fondaparinux was commenced on day 23 post-op and warfarin was started. He was discharged day 44 post-op, following a period of rehabilitation with a normal platelet count and no new evidence of thrombosis.

CONCLUSIONS

Refractory or delayed-onset HIT with features of prolonged severe thrombocytopenia and lack of response to conventional anticoagulants is an important clinical scenario that clinicians need to be mindful of.

Case reports have shown successful treatment with the addition of IVIg +/- steroids, as in this case, although caution is advised given the potential thrombotic risk of IVIg in patients with HIT [1,2,3].

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