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Massive cardiac and pulmonary thrombi after cardiac surgery

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1. Case presentation

A 32-year-old obese male with bicuspid aortic valve and severely dilated ascending aorta (59.5 mm) underwent hemi-arch aortic replacement and valvuloplasty on cardiopulmonary bypass. Due to progressive respiratory failure, veno-venous extracorporeal membrane oxygenation (ECMO) was started on postoperative day 4. On postoperative day 15, ECMO flow fell and the patient was progressively desaturating. Chest X-ray was normal. *Trans*-Oesophageal echocardiography revealed a mobile thrombus in the right atrium (Fig. 1, Panel A, asterisk) passing the tricuspid valve toward the right ventricle during systole (Supplementary video 1). Blood tests showed normal coagulation but low platelets (26,000/mm^3 versus 160,000/mm^3 at day 0). Following redo-sternotomy, a right atrial thrombus measuring 3 × 12 cm (Fig. 1, Panel B, left, Supplementary video 2) was removed. Intra-operatively, a second thrombus measuring 1.5 × 10 cm was discovered in the pulmonary artery and removed (Fig. 1, Panel B, right).

What is the diagnosis?

2. Discussion

Based on the thrombocytopenia, thrombus development and elevated heparin antibodies (1.8 U/mL, reference < 1.0 U/mL), heparin-induced thrombocytopenia (HIT) was suspected and conversion from a heparin to a danaparoid based ECMO system was made. Platelet count doubled within 48 h and normalised in 7 days.

HIT is caused by auto-antibodies to complexes between platelet factor 4 (PF 4), a protein released by activated platelets, and heparin [1]. These auto-antibodies form immunocomplexes and trigger platelet activation and aggregation [1]. The consequent increase in thrombin generation creates a paradoxical prothrombotic state in these patients [1]. It typically occurs between 5 and 10 days after heparin initiation, though in patients who received heparin in the last 90 days, HIT can develop more quickly because of persistent anti PF 4 - heparin antibodies [1]. Unfractionated heparin is associated with a higher risk of HIT than low molecular weight heparins and surgical patients have a higher risk than medical patients [2].

Anti PF4 – heparin antibodies should only be tested when clinically relevant. They are frequent in patients after treatment with heparin, but only 2 to 15% will develop clinically relevant HIT [2]. The '4T Scoring
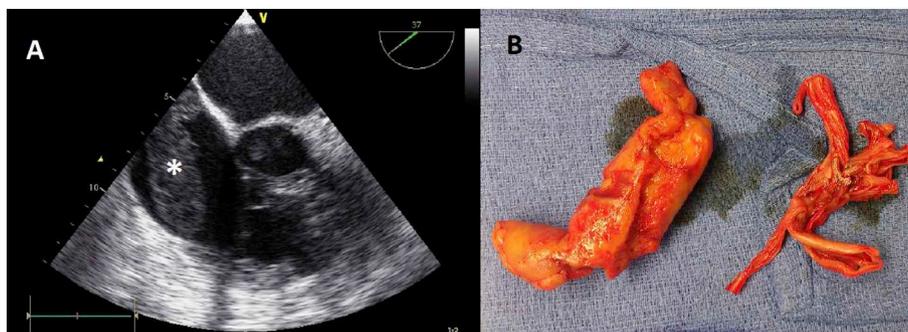

Fig. 1. Panel A: Mobile thrombus in the right atrium, passing toward the tricuspid valve during systole, on transoesophageal echocardiography (Asterisk). Panel B: Intra-operatively removed right atrial thrombus (3 × 12 cm, left) and second thrombus (1.5 × 10 cm, right) removed from pulmonary artery.

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System', based on timing and degree of thrombocytopenia, thrombosis and other causes of thrombocytopenia, can be used to determine pre-test probability of HIT [1]. Treatment of HIT consists in prompt cessation of heparin and administration of alternative anticoagulants, such as argatroban, bivalirudin, danaparoid or fondaparinux [2].

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ejim.2018.09.020.

Conflict of interest

None.

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