Two Cases of Valvular Thrombosis Secondary to Heparin-Induced Thrombocytopenia Managed Without Surgery

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A 83-year-old woman underwent aortic valvular replacement with a bioprosthesis for symptomatic aortic stenosis. Anesthesia and surgery were uneventful. Unfractionated heparin (UFH) had been administered for cardiopulmonary bypass followed by anticoagulation on day 0 (surgery), and had been continued thereafter. Routine postoperative blood count showed an increase in platelet counts to >200 G/L on day 6. On the ninth day, the patient complained of paresthesia of the left foot. A computed tomography scan with intravenous radiocontrast material was performed. Thrombosis of both iliofemoral axes and a thrombus on the ascending aorta were found (Figure 1 and Figure 2). A blood count on day 9 revealed thrombocytopenia at 44 G/L; antiPF4 antibodies (Enzyme-Linked ImmunoSorbent Assay HPIA is Heparin Platelet Factor 4 Induced Antibodies [ELISA] Asserachrom HPIA Stago, Asnières France) were positive (optical density >2), and the platelet aggregation test confirmed the diagnosis of heparin-induced thrombocytopenia. Anticoagulation with UFH was stopped and replaced with intravenous sodium danaparoid targeted to anti-Xa concentration of 0.5 to 0.8 IU/mL. The patient underwent surgical iliofemoral thrombectomy that was uneventful.

Transesophageal echocardiography (TOE) revealed thrombosis of the aortic valve (with a thrombus of 18 mm) extending into the first centimeters of the ascending aorta (Figure 3 and online-only Data Supplement Movie I). Because the patient was asymptomatic, and due to the estimated risks of the surgical procedure, the decision to pursue sodium danaparoid treatment was made; the above-mentioned thrombi were to be monitored using TOE. Platelet counts returned to 100 G/L on day 14. Sodium danaparoid was...
overlapped with antivitamin K (fluindione), which was started on day 18. Sequential TOE revealed total regression of the intracardiac thrombus after 3 weeks (no picture available).

A 54-year-old woman underwent mitral valve replacement for mitral stenosis with a mechanical prosthesis. Anesthesia and surgery were uneventful. Unfractionated heparin had been administered for cardiopulmonary bypass anticoagulation on day 0 (surgery) and continued thereafter. On postoperative day 11, a routine TOE was performed because of an increased valve gradient revealed by previous transthoracic echocardiography; nonobstructive thrombosis of the mitral valve was confirmed (Figure 4A). The patient had been anticoagulated with UFH since day 0, and the platelet counts were 230 G/L. Treatment with aspirin was added while administration of UFH was pursued. Two days later (postoperative day 13), a new TOE revealed thrombus extension with 2 mobile thrombi of 18 mm (Figure 4B; online-only Data Supplement Movie II and Movie III). Platelet counts were >200 g/L, but anti-PF4 antibodies (HIA IgG ELISA kit, Hyphen, Biomed) were positive (optical density = 1.25). As a diagnosis of heparin-induced thrombocytopenia with thrombosis (HITT) was highly suspected, treatment with UFH was stopped, and anticoagulation performed with intravenous sodium danaparoid targeted to anti-Xa concentration of 0.5 to 0.8 IU/mL.

Because the patient was asymptomatic, and due to the risk of surgery in patients with HITT, the decision to pursue medical treatment was made, with further monitoring of the patient by TOE. The patient’s evolution was favorable, with total disappearance of the thrombi observed on treatment day 32 (Figure 5 and online-only Data Supplement Movie IV).

No clinical thromboembolic event occurred for these 2 patients.

These 2 cases illustrate the difficulties encountered every day when diagnosing heparin-induced thrombocytopenia or HITT in cardiac surgery patients. They also raise the question of the adequate strategy when HITT presents with valvular thrombosis. In the case of the non-HITT-related valvular thrombosis, treatment is usually discussed as being between a medical or a surgical strategy. Roudaut et al recommended taking into account the size of the thrombus in the presence of nonobstructive thrombosis.1 For thrombi <5 mm, a medical strategy is recommended with echocardiography follow-up and antiplatelet therapy; for thrombi >5 mm, especially when thrombi are large, mobile, and pedunculated, surgical strategy...
would be the preferable choice. Taking into account these recommendations, both patients should have undergone surgical thrombectomy, even without HITT.

The specificities of HITT-related valvular thrombosis are not well documented. Because of difficulties of anticoagulation during cardiopulmonary bypass in patients with heparin-induced thrombocytopenia (risk of severe hemorrhage and transfusion), the medical strategy was considered to be the best option for these 2 patients. The favorable evolution in both cases with medical treatment alone highlights the need for a specific strategy for valvular thrombosis in the context of HITT.

These 2 cases confirm several other reports in which nonsurgical therapy was successful in patients with HITT-related prosthetic valvular thrombosis and left atrial thrombosis. Taken together, these reports suggest that when analyzing the risk-to-benefit ratios of different strategies in patients with valve thrombosis after cardiac surgery secondary to HITT, medical therapy alone may be considered as an acceptable alternative.

Disclosures
None.

References
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