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**Regression of intracardiac heparin-induced thrombosis after aortic root surgery**  
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its anticoagulant effects, as well as frequent development of clots and prolonged postoperative bleeding [4].

Ancrod prevents the development of clots by selectively depleting fibrinogen. It can be reversed with FFP or cryoprecipitate. However, ancrod is not approved by the Food and Drug Administration and is available for compassionate use only [4].

Lepirudin, a direct inhibitor of thrombin, is a safe and effective anticoagulant, although there is no specific antidote [5, 6]. Three to five times more potent than heparin, it does not crossreact with heparin-induced antibodies. It leads to rapid recovery in platelet count and prevents progression of thrombotic complications. Monitoring the activated partial thromboplastin time (aPTT) is required [4, 5].

#### *Back-Up Plan if Off-Pump Approach Not Possible*

At our institution, patients with HITT who require cardiac surgery are maintained on lepirudin and warfarin preoperatively. If the procedure cannot be performed off-pump, abciximab is given intraoperatively (bolus of 0.25 mcg/kg followed by an infusion of 0.125 mcg/kg per minute). Abciximab inhibits platelet aggregation, the primary etiology of thrombosis in patients with HITT. Abciximab acts by binding to intact glycoprotein IIb/IIIa receptor and preventing binding of fibrinogen, von Willebrand factor, and other adhesive molecules. A standard dose of heparin is given as a bolus. Abciximab is continued until 10 minutes before reversal of heparin, which is accomplished using the usual dose of protamine. Because abciximab disrupts platelet function, platelets are given (12-18 units) immediately after heparin reversal.

Warfarin should be given only after therapeutic levels (PTT of 1.5 to 2.5 the normal) of lepirudin have been achieved. Ideally, warfarin should not be given until there is substantial resolution of thrombocytopenia, as thrombotic complications have been described in patients when alternative anticoagulants were discontinued prior to resolution of thrombocytopenia. Lepirudin can be discontinued when the INR is therapeutic for 48 hours or after 5 days of warfarin therapy, whichever is longer [1, 4].

Prophylactic platelet transfusions are not recommended, as petechiae and other clinical evidence of bleeding are uncommon. In addition, thrombotic events occurring early after transfusion of platelets have been reported. Platelet transfusions should be reserved for patients with serious hemorrhagic complications [5]. Platelet inhibitors, such as aspirin and prostin, should be used to decrease the adhesiveness of platelets, which can limit thrombosis.

Surgical thromboembolism may be appropriate for select patients with large vessel or intracardiac thromboembolism. For patients who cannot receive heparin, an off-pump approach with total inflow occlusion is preferred. A backup plan should be in place, however, regarding alternative methods of anticoagulation in the event that CPB is required.

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## Regression of Intracardiac Heparin-Induced Thrombosis After Aortic Root Surgery

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Heparin-induced thrombocytopenia and thrombosis syndrome (type II) is associated with thromboembolic complications and a mortality rate up to 30%. We describe a patient who developed intracardiac and aortic Dacron prosthesis heparin-induced thrombosis after aortic root conservative surgery. Successive transoesophageal echocardiographies demonstrated a progressive regression of intracardiac thrombosis with oral anticoagulation by warfarin and antiplatelet therapy combining aspirin and clopidogrel.

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**H**eparin-induced thrombocytopenia type II (HITT) is an idiosyncratic immune-mediated reaction most commonly caused by an immunoglobulin G antibody that binds to platelets in the presence of heparin and causes platelet activation [1]. Heparin-induced thrombocytopenia type II is defined by a fall of platelet count by more than 50% of the base line value occurring at least 5 days after heparin therapy onset, accompanied by heparin-dependant antiplatelet antibodies detected by the

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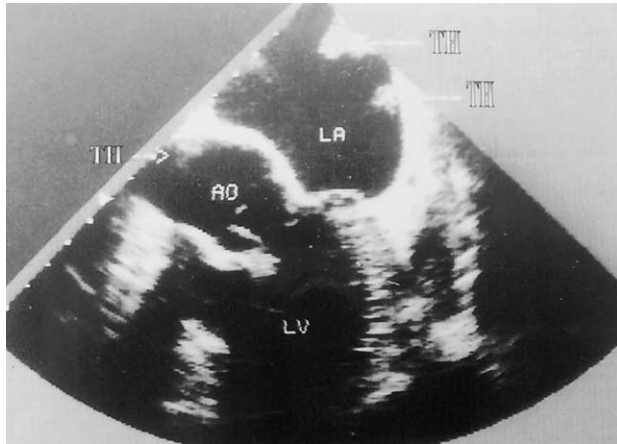


Fig 1. Transesophageal echocardiography images revealed thrombosis (TH) into the left atrium (LA) and in the Dacron aortic graft (AO). (LV = left ventricle.)

sensitive C-serotonin release assay. The real frequency of HIT is unknown and ranges between 0.2% and 2% [2, 3], although it is more common after full dose intravenous heparin administration. The risk for thromboembolic events in patients with HIT has been reported more than 60% [1]. In contrast, intracardiac thrombosis due to HIT remains exceptional and treatment of this complication is unclear [4, 5]. We describe a patient with multiple intracardiac and aortic prosthesis thrombosis due to HIT after aortic root surgery successfully treated without cardiac reoperation.

A 70-year-old patient underwent elective aortic root remodeling, described by David and Feindel [6], for an ascending aorta aneurysm with a prosthetic Dacron graft. Preoperative platelet count was 213,000 per  $\mu\text{L}$ . The immediate postoperative course was uneventful. Anticoagulation therapy with subcutaneous heparin (7500 U three times daily) was necessary because of chronic atrial fibrillation. One week after surgery the patient developed an acute ischemic leg syndrome with a drop of platelet count down to 44,000 per  $\mu\text{L}$ . Transthoracic echocardiography was normal. An emergency aortic bifemoral bypass was indicated due to the presence of extensive and disseminated "white clot" in the aortic bifurcation and legs arteries. At the same time heparin-dependent antibodies were detected by the sensitive C-serotonin release assay and treatment with Orgaran (AntiXa levels at 0.5 IU/mL; Orgaran, Roseland, NJ, USA) was started and then switched to oral anticoagulation with warfarin (INR: 3 to 3.5) 5 days after vascular surgery. Platelet count rose back up to 250,000 per  $\mu\text{L}$ .

Evolution was satisfactory up to the tenth postoperative day after vascular surgery, when the patient developed a transient ischemic attack. A transoesophageal echocardiographic (TEE) scan demonstrated multiple thrombosis in the right and left atrium, and attached to the proximal and distal suture of the aortic prosthesis (Figs 1 and 2). An adjunctive therapy with aspirin and

clopidogrel was started in association with warfarin. Eight days later a TEE control illustrated regression of the intracardiac thrombosis. The antiplatelet therapy was continued and a new TEE control 1-month later revealed the total disappearance of intracardiac thrombosis.

### Comment

This observation demonstrates the urgent need for rapid recognition of HIT because continuing heparin therapy may lead to severe thromboembolic complications. A drop of platelets count below 100,000 per  $\mu\text{L}$  with thromboembolic complication suggests the presence of heparin-induced antibodies that need to be confirmed by laboratory tests. This report also demonstrates that, despite rapid diagnosis and treatment of HIT, the risk for severe thrombosis persists for several weeks after exposure to heparin. Warkentin and Kelton [7] indicated in a retrospective study that patients with a diagnosis of isolated HIT without thrombosis have a 30-day risk of thrombosis of more than 50%, despite cessation of heparin administration. This study suggests that alternate anticoagulant or antiplatelet therapy should be necessary when the diagnosis of HIT is done, even without thrombosis complications.

Peripheral vascular thrombosis that occurred in our patient was largely reported previously. But heparin-induced intracardiac and Dacron prosthesis thrombosis remains exceptional. Intracardiac thrombosis may be underestimated in asymptomatic patients and TEE should be systematically performed in all patients with HIT to search for intracardiac thrombosis [5].

The therapeutic strategy in this patient was unclear. Regarding the risk of a new thromboembolic event in the case of medical treatment, compared with the risk of cardiac reoperation with the difficult management of cardiopulmonary bypass without heparin administration. Thrombolytic agents have been successfully used in life-threatening conditions without bleeding complications [4]. Antiplatelet therapy is a good alternative and, in

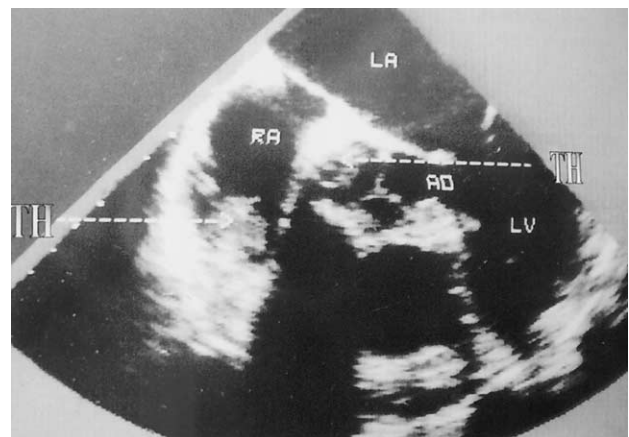


Fig 2. Transesophageal echocardiography images revealed thrombosis (TH) into the right atrium (RA) and near the proximal suture of the Dacron aortic graft (AO). (LA = left atria; LV = left ventricle.)

this patient, association of warfarin, aspirin, and clopidogrel have demonstrated a rapid regression of thrombosis.

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## Uncommon Presentation and Surgical Correction of Unroofed Coronary Sinus Syndrome

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**A 59-year-old man with signs and symptoms of congestive heart failure, occurring a few months after an infective episode, underwent cardiac investigations revealing severe biventricular dysfunction, persistent left superior vena cava with almost completely unroofed coronary sinus, and critical stenosis of the proximal right coronary artery. Surgical correction of the congenital malformation associated with revascularization of the right coronary allowed a prompt recovery of clinical conditions and ventricular function.**

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In 1965, Raghiv and colleagues [1] described the developmental complex characterized from termination of the left superior vena cava (LSVC) in the left atrium, atrial septal defect, and absence of coronary sinus. This malformation was recognized in 8 patients of a surgical series reported in 1979 in which the partition between the coronary sinus and the left atrium was totally or almost totally absent [2]. Pathophysiology of the defect consists of bi-directional atrial shunting, increased pulmonary blood flow and decreased systemic oxygen saturation [3]. Cyanosis and the risk of cerebral complication make surgical correction advisable. We present an uncommon case of an adult patient who was hospitalized because of an episode of congestive heart failure, in whom the demonstration of the congenital malformation was totally unexpected.

A 59-year-old man with a history of hypertension, hypercholesterolemia, past smoking, and chronic obstructive pulmonary disease who was treated with bronchodilators, after a 1-month period of mild, intermittent pyrexia accompanied by progressively reduced exercise tolerance, was referred to our cardiology department because of clearly evident congestive heart failure (New York Heart Association functional class IV). Referral diagnosis was postmyocarditis dilated cardiomyopathy. Peripheral edema and orthopnea were cured with aggressive medical treatment (diuretics and vasodilators), and the echocardiogram revealed dilated right (moderate tricuspid valve regurgitation) and left ventricles (transverse diastolic diameter, 69 mm), systolic left ventricular (LV) dysfunction (ejection fraction, 35%), akinesia of the inferoposterior LV wall, persistent LSVC without a precise demonstration of ASD (atrial septal defect). Cardiac catheterization demonstrated systemic oxygen saturation of 91%, pulmonary artery systolic pressure of 51 mm Hg, and a cardiac index of 1.96 L/min/m<sup>2</sup>. Injection into the LSVC revealed a wide defect in the roof of the coronary sinus with bi-directional shunt (Fig 1). Coronarography demonstrated a severe stenosis of the right coronary ostium. Ventriculography showed akinesia of the LV inferior wall, LV diastolic volume of 233 mL, systolic volume of 157 mL, and global hypokinesia with an LVEF of 33%. Blood gas analysis revealed an arterial PO<sub>2</sub> of 55 mm Hg with room air, with a PCO<sub>2</sub> of 35, a hemoglobin level of 17.2 g/L, and a hematocrit value of 53%.

During the operation the pericardium was found to be adherent to the epicardium as is usually seen after a recent pericarditis. After the skeletonized right internal mammary artery was harvested, cardiopulmonary bypass was established by cannulation of the ascending aorta and the three caval veins selectively. During aortic cross-clamp time with cardioplegic arrest, the intact atrial septum was opened through the right atriotomy, and the anatomy of the defect was demonstrated; a small remnant of the coronary sinus wall was present just upstream the large orifice in the right atrium, whereas at least three quarters of the theoretical course of the coronary sinus beneath the left atrium was unroofed, and the LSVC was drained at the left upper corner of the left atrium, close to

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