Inferior vena cava thrombectomy in a patient with heparin-induced thrombocytopenia via inflow occlusion technique on beating heart

Faruk Cingoza, Murat Tavlasoglua,*, Mehmet Ali Sahina and Mustafa Kurkuoglu
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INTRODUCTION

The administration of heparin frequently induces a reduction in platelet counts. This phenomenon is called heparin-induced thrombocytopenia (HIT) and classified as either type I or II. To avoid confusion between the syndromes, ‘HIT type I’ has been renamed as ‘non-immune heparin associated thrombocytopenia’, and ‘HIT type II’ is simply called ‘HIT’. HIT occurs more often with unfractionated heparin (UFH) than with low-molecular-weight heparin (LMWH) and, almost certainly, more often with LMWH than with fondaparinux [1]. Inflow occlusion on beating heart is a technique that was used more commonly in cardiac surgery prior to cardiopulmonary bypass (CPB) era. Nowadays, this technique is seldom preferred in cases with pulmonary valvotomy, aortic valvotomy, atrial septectomy, cardiac injury and extraction of intracardiac thrombus or foreign body [2]. These previously described operations can also be performed by using CPB. However, in patients with previous HIT history, CPB may no longer be suitable during surgical intervention due to possible adverse effects of heparin in terms of HIT relapse. In this article, we present a successfully performed caval vein thrombectomy with inflow occlusion technique on beating heart in a patient with a history of deep vein thrombosis, pulmonary embolism and HIT type II.

CASE REPORT

The patient was LMWH treated followed by warfarin sodium due to right lower extremity deep vein thrombosis caused by trauma in 2003, and then left lower extremity deep vein thrombosis and pulmonary embolism in 2005. He has been on oral warfarin sodium therapy since 2005. In 2011, the patient was operated on for anal fissure in a different medical centre, and received LMWH in the postoperative period. During this time period, thrombocytopenia was observed, and after a haematology consultation, based on laboratory tests, the diagnosis of HIT type II was confirmed. This information was rechecked from the patient’s past records, brought during admission.

He was admitted to our clinic seven months after the initial diagnosis of HIT type II with complaints of angina pectoris and shortness of breath. The patient was consulted by haematologist. Blood samples were taken for antibody level measurement. In addition, it was declared that a different anticoagulant agent rather than heparin would be suggested depending on the test results. Trans-thoracic and transesoophageal echocardiography revealed a mass lesion in the lumen of the inferior caval vein in the cavoatrial junction. Cardiac magnetic resonance examination confirmed the thrombus in the lumen the of inferior caval vein. The patient was scheduled for inferior vena cava thrombectomy before the test results could be obtained, because haemodynamic compromise occurred in the clinical follow-up. The operation was planned to be performed with inflow occlusion technique on the beating heart. Then warfarin sodium was stopped three days prior to the operation while fondaparinux sodium was begun twice a day. The operation was successfully performed and no postoperative complications were observed.

Keywords: Inflow occlusion • Heparin-induced thrombocytopenia • Fondaparinux sodium • Caval thrombectomy
over the diaphragm. Distal and proximal ends of caval veins were secured (Fig. 1). The patient was ventilated at an oxygen concentration of 100%. After the proximal and distal ends of the inferior caval vein were occluded and clamped, venotomy was performed and thrombectomy was successfully achieved in the usual manner (Supplementary video 1). Then venotomy was repaired with 6/0 prolene suture material in the usual manner (Fig. 2a). Figure 2b demonstrates the extracted organized thrombus material.

Positive blood sample results were obtained in the postoperative period and confirmed the accuracy of our approach in order to minimize possible complications.

**DISCUSSION**

HIT is an immune-mediated disorder resulting when immunoglobulin G antibodies are produced that recognize a self-protein, platelet factor 4, when platelet factor 4 has formed complexes with heparin [3]. Multimolecular complexes of heparin, platelet factor 4 and immunoglobulin G form on platelet surfaces, and occupancy of the platelet Fc receptors by HIT–immunoglobulin G produces platelet activation. Heparin chains bind to platelet factor 4 determined by their chain length, a possible explanation of why UFH is more likely to cause HIT than LMWH [3, 4]. Platelet activation in HIT also leads to the activation of the coagulation cascade. Once these procoagulant events are triggered, the prothrombotic risk remains for days to weeks, even after heparin has been stopped [3, 4]. Most often, HIT presents as an unexpected platelet count fall beginning 5 to 10 days after heart surgery [3]. The produced immunoglobulins in G form may interfere with heparin in cases of re-exposure. Therefore we have abandoned the use of CPB and scheduled the inflow occlusion technique on the beating heart.

We could consider some other options for anticoagulation such as bivalirudin or lepirudin, however the ecarin clotting time monitoring that is mandatory for correct dosing [3, 5] is not available in our clinic. Moreover, the provision of bivalirudin and lepirudin is difficult because they are not widely used in our country. We did not have enough experience with the option of using CPB on bivalirudin or lepirudin therapy. We did not consider performing hypothermic circulatory arrest because the thrombus was in the operable area.

We considered accessing the inferior vena cava through an abdominal incision; however, the thrombus was mainly at the cavoatrial junction, thus the abdominal approach would necessitate further incision through the diaphragm and even sternotomy. Therefore we abandoned this approach, but lower partial sternotomy could be considered for a minimal invasive approach and better cosmetic results.
The inflow occlusion technique was more frequently used before the invention of CPB machines. Although it has lost its popularity with the use of CPB in daily practice, it may be preferred in procedures like the removal of the right atrial thrombus or catheter, right atrial or ventricular pace leads [2] and caval venous thrombectomy accompanied by HIT. In patients with acute HIT, postponing surgery for several weeks may be necessary, and different anticoagulation agents should be used during cardiac surgery such as bivalirudin (preferably, if ecarin clotting time monitoring is available), lepirudin (if ecarin clotting time monitoring is available and renal function is normal), epoprostenol plus heparin, tirofiban plus heparin or danaparoid (if drug and anti-factor Xa monitoring are available). However, these anticoagulant agents do not meet the well-known advantages of UFH during CPB including its high efficacy for preventing thrombosis of the CPB circuit; rapid and simple intraoperative monitoring by activated clotting time and neutralization of heparin by protamine sulphate [3]. Where heparin use is avoided, some different surgical techniques may be applied including off-pump coronary artery bypass surgery, requiring only about one half to one third the level of anticoagulation compared with conventional CPB, using different anticoagulation regimens (bivalirudin, lepirudin or danaparoid). Additionally, the inflow occlusion technique may be used safely in patients having a risk of HIT accompanied by caval thrombosis. The advantage in this case was the intact blood flow from the superior caval vein through the pulmonary circulation. This gave us an additional 20-min period during the inferior caval vein clamping. The thrombus was removed. Postoperatively, platelet counts were monitored daily and no decrease was observed. The whole postoperative course was uneventful.

SUPPLEMENTARY MATERIAL

Supplementary material is available at ICVTS online.

Conflict of interest: none declared.

REFERENCES