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Recombinant Activated Factor VII Administration after Pulmonary Embolectomy: Case Report

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ABSTRACT

Bleeding management in cardiac surgery could be a great challenge for the surgeon and a life-threatening moment for the patient. Despite the fact that recombinant activated factor VII is now widely accepted as a useful adjunct in the management of postcardiotomy coagulopathy, its use in the course of recent thromboembolic event is rarely described. We hereby present a case of rescue recombinant activated factor VII administration to manage a severe coagulation disorder during surgical pulmonary embolectomy performed under cardiopulmonary bypass.

INTRODUCTION

Pulmonary embolism (PE) with an embolus captive in a patent foramen ovale (PFO) is a condition that almost always requires emergency surgical embolectomy. The use of cardiopulmonary bypass (CPB) during this procedure can magnify the coagulation disorder that is already triggered by liver congestion due to right ventriclar dysfunction and the coagulation factor consumption caused by the thrombotic event itself. Bleeding management under these circumstances could be a great challenge. The use of recombinant activated factor VII (rFVIIa) for the management of patients with this coagulation disorder in such a paradoxical setting has rarely been reported [Reyes 2008].

CASE REPORT

A 64-year-old male patient with a medical history of hypertension was admitted for recent onset dyspnea on exertion. Vital signs obtained at the time of admission were normal. Signs of right heart failure, including jugular vein distension, liver enlargement, and mild edema of the lower extremities, were noticed. Except for elevated liver function tests (AST

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1.48 µkat/L, ALT 1.66 µkat/L), laboratory findings including blood count and coagulation were within normal range. Chest computed tomography revealed massive bilateral PE with embolus passing through the PFO (Figure 1A). Echocardiography confirmed this diagnosis (Figure 1B). The left atrial embolus impinged into the left ventricle during diastole (Figure 1C). The right ventricle was dilated and hypokinetic. Duplex sonography of the lower extremities showed thrombosis of the left popliteal vein. There were no signs of systemic embolization. Fibrinolysis was not indicated for the high probability of embolus fragmentation with subsequent pulmonary or systemic embolization. The patient was referred for surgical pulmonary embolectomy, which was performed via a midsternotomy approach using CPB with conventional aortic and bicaval cannulation. The ascending aorta was crossclamped to avoid mechanical dislodgement of the embolus. Cold crystalloid cardioplegia was administered into the aortic root and repeated after 20 minutes. The right atrium was opened, and after the atrial septum was incised a snake-like embolus was seen stuck in the PFO (Figure 2A and 2B). The embolus was extracted, and the PFO was closed by doublecontinuous suture. Longitudinal incision of the pulmonary artery was performed. The procedure continued with aspiration of fragile emboli from the pulmonary branches using silastic tubes of different diameters. The cross-clamp time was 40 minutes. During CPB normothermia was maintained. High doses of vasoactive and inotropic drugs were needed because of right ventricle failure. Weaning from the CPB took 45 minutes, after which the patient experienced severe and diffuse bleeding from the whole surgical field. Blood loss was recuperated. Surgical source of bleeding was excluded. Coagulopathy, caused by coagulation factor deficiency and thrombocytopathy, was assessed using thromboelastography (TEG®; Haemoscope Corporation, Skokie, IL, USA). The patient required transfusion of 10 fresh frozen plasma concentrates, 8 red blood cell concentrates, and 3 pooled platelets. Five grams of fibrinogen were given. The bleeding did not stop and over 60 minutes blood loss was about 1200 mL. It was decided that a reduced dose of rFVIIa (50 µg/kg) would be administered, despite the recent thromboembolic event and considering residual emboli in the pulmonary circulation. After its use, an immediate positive effect was obvious with consequent decrease in catecholamine requirement.



Figure 1. A, Computed tomography (CT) angiography showing emboli in the right and the left atrium (black and white arrows). B, Transesophageal echocardiogram showing an embolus entrapped in a patent foramen ovale (arrow). C, Transthoracic echocardiogram with embolus impingement into the left ventricle (arrow).

The patient was transferred to the intensive care unit, where blood loss, during the next 12 hours, was 300 mL. Then, anticoagulant therapy using low molecular weight heparin was commenced. Repeat sonography of the lower extremities 24 hours later was negative; hence caval filter insertion was not indicated. The patient was weaned off artificial ventilation on the second postoperative day. Further postoperative course was uneventful. Echocardiography before discharge showed normal function of the right ventricle; no signs of pulmonary hypertension were noticed. The patient was discharged on the 18th postoperative day on warfarin, which was recommended as a lifelong therapy.

DISCUSSION

Massive PE with an embolus entrapped in a PFO is a rare finding. Since there are often comorbidities in patients presenting with PE, a mature approach as to the choice of treatment is required.

Patients presenting with high-risk PE could be treated with thrombolytic therapy, which can rapidly decrease pulmonary hypertension, improve right ventricle performance, and thus in turn improve blood oxygen saturation [Goldhaber 1993]. However, possible secondary complications such as intracranial hemorrhage, gastrointestinal bleeding, and recurrence of PE must be considered when thrombolysis is

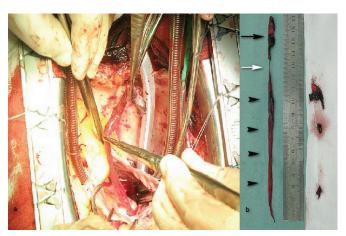


Figure 2. A, Periprocedural image demonstrates extraction of the intracardial embolus. B, About 18 cm long embolus extracted from the atria and the left ventricle (black arrow: right atrial portion of the embolus; white arrow: the portion passing the PFO; head arrows: left atrial and ventricular portion of the embolus).

administered [Thabut 2002]. In case of absolute contraindication for thrombolysis or failure of thrombolytic therapy, both catheter and surgical pulmonary embolectomy should be considered [Meneveau 2010]. About 50% of patients presenting with massive PE are contraindicated for thrombolytic therapy [Kasper 1997]. Furthermore, 8% of patients receiving thrombolytic therapy show no response. Scheduled surgical embolectomy in this group of patients has a better outcome than repeated thrombolysis [Meneveau 2006].

In the case herein reported, the finding of a captive embolus in the PFO with the high risk of embolus dislodgment made surgical embolectomy the treatment of choice. The coagulation factor consumption along with the use of the CPB and liver dysfunction can be considered as a trigger for the coagulation disorder. The use of rFVIIa can substantially reduce the need for exogenous blood transfusion and thus eliminate the risk of both prolonged bleeding and transfusion-related complications. The current risk of a thrombotic episode associated with rFVIIa application is 0.8% to 1.5% [Levi 2005]. In the case of our patient, however, this risk is much lower than the risk of death associated with the operation procedure itself. The administration of rFVIIa in cardiac surgery to deal with uncontrolled blood loss is well reported [Lehr 2010]. Its use in the course of recent thromboembolic event, however, is rarely described in the literature [Reyes 2008].

The case report presented demonstrates effective and safe off-label use of rFVIIa in such a paradoxical setting.

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