Extensive pulmonary embolism after severe postpartum haemorrhage: management with an inferior vena cava filter

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ABSTRACT
We report the case of a 36-year-old woman with an inferior vena cava thrombosis and extensive pulmonary embolism six days after a severe postpartum haemorrhage. She had undergone caesarean section with bleeding managed by massive transfusion, hysterectomy, and two attempts at uterine artery embolization. Systemic thrombolysis and catheter-directed thrombolysis in intensive care were abandoned due to recent and incomplete uterine artery embolization. A temporary inferior vena cava filter was chosen because of significant risk of massive pulmonary embolism. This was a controversial decision because guidelines from different professional groups offer conflicting recommendations. The therapeutic options for the management of massive postpartum pulmonary embolism when thrombolysis is contraindicated are discussed.

Keywords: Inferior vena cava thrombus; Pregnancy; Pulmonary embolism; IVC filter

Introduction

Venous thromboembolism is a significant cause of morbidity and mortality during pregnancy and the immediate postpartum period.[1] When life-threatening pulmonary embolism is associated with inferior vena cava (IVC) thrombus, a rare postpartum complication,[2] and systemic thrombolysis is contraindicated, therapeutic decisions are a challenge. We report a case of IVC thrombus and extensive pulmonary embolism and discuss management options.

Case report

A 36-year-old woman with a pre-pregnant body mass index of 20 kg/m^2, underwent caesarean delivery for cholestasis of pregnancy in a peripheral urban hospital at 39 weeks of gestation. Previous medical history included two uneventful caesarean deliveries but was otherwise unremarkable. At delivery, uterine atony resistant to oxytocin and sulprostone led to a postpartum haemorrhage that was managed with transfusion of eight units of red blood cells (RBC), two units of fresh frozen plasma (FFP), prothrombin complex and tranexamic acid. In view of continuing bleeding, and without access to arterial embolization, a hysterectomy was performed under general anaesthesia.

After initial stabilization, bleeding resumed and the patient was transferred to a tertiary hospital for arteriographic embolization and intensive care. An abdomino-pelvic computed tomography (CT) scan revealed a large intra- and retro-peritoneal haematoma, with active bleeding from the right ovarian and umbilical arteries. Arteriography demonstrated aneurysmal lesions at the stumps of both uterine arteries with hypertrophy of the left uterine artery and atrophy of the right, without extravasation of contrast from the ovarian arteries. Embolization was performed using absorbable gelatin fragments, resulting in complete occlusion of both uterine arteries and partial occlusion of both internal iliac arteries. Despite initial angiographic control, haemorrhage recurred a few hours later with sudden tachycardia and acute anaemia with a haemoglobin of 6 g/dL. Further arteriography revealed early recanalization of both uterine arteries and a second emergency embolization was carried out using non-absorbable particles. Management of haemorrhage required transfusion with 25 units of RBC, 18 units of FFP, 3 units of platelets, prothrombin complex 40 mL, tranexamic acid 1 g and fibrinogen concentrate 1.5 g (Clottafact®, LFB, Paris, France). In the absence of re-bleeding and renal failure, prophylactic anticoagulation was started 24 h after embolization. Subsequent progress in the intensive care unit was uneventful, and she was transferred to the ward on the third day after admission.

On the sixth day after caesarean delivery she appeared dyspnoeic. She was conscious, her blood pressure was 120/60 mmHg, heart rate 85 beats/min and oxygen saturation 96% breathing room air. Her respiratory rate was 20 breaths/min with exertional dyspnoea during speech,

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but chest auscultation was unremarkable. Electrocardiographic changes demonstrated an S1Q3 pattern consistent with a diagnosis of pulmonary embolus (PE). Plasma biochemical analysis did not show elevated levels of troponin Ic or brain natriuretic peptide (BNP). Blood gas analysis revealed mild respiratory alkalosis (pH 7.46, $PaCO_2$ 3.97 kPa) without hypoxemia (PaO_2 13.6 kPa). Transthoracic echocardiography demonstrated good left ventricle (LV) systolic function with right ventricle (RV) dilation without acute cor pulmonale and pulmonary hypertension.

The thoracic CT scan showed a massive PE in the proximal right pulmonary artery, extending to three lobes, and in the left lower lobe artery (Fig. 1). The contrast-enhanced abdominal CT scan also revealed a non-obstructive 2.5 cm thrombus located in the IVC above the renal veins (Fig. 2).

An intravenous bolus of 3000 U unfractionated heparin was given, followed by a continuous infusion of 100 U/h, gradually increased to 1850 U/h with an activated partial thromboplastin time ratio target between 2.5 and 3. She was confined to bed. In view of the recent caesarean delivery, dysplastic uterine arterial stumps, failure of embolization attempts with incomplete occlusion of the internal iliac arteries, both systemic and regional catheter-directed thrombolysis were considered too hazardous. Implantation of a vena cava filter was also considered potentially hazardous due to the need for a superior vena cava approach and locating the filter above the renal veins which, in the event of filter occlusion, could quickly compromise renal function. Despite this, it was considered to be the best option and a retrievable cava filter was inserted through the internal jugular vein under local anaesthesia, and positioned 1 cm above the thrombus. The procedure was uneventful and renal function was unimpaired.

She remained in intensive care for two days, and spent a further three weeks on the gynaecology ward. The IVC filter was removed after six weeks, following a contrast-enhanced CT scan that confirmed disappearance of the IVC thrombus. She continued oral anticoagulation therapy for one year.

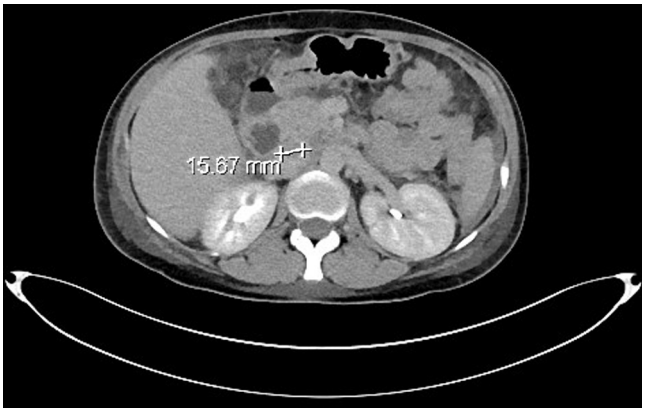

Fig. 2 CT scan showing extensive thrombosis in the inferior vena cava

Discussion

A PE may be called massive when it is associated with arterial hypotension or cardiogenic shock associated with RV failure. Haemodynamically stable patients with proven RV dysfunction on echocardiography and with high troponin and BNP levels are usually diagnosed as suffering from sub-massive PE.[3] Our patient did not present with massive PE, but echocardiographic and angiographic findings suggested that the extent of pulmonary artery obstruction could increase rapidly and raise RV afterload. The embolus was extensive and involved four of the five lung lobes. The presence of a 2.5 cm thrombus in the IVC was particularly worrying since migration would have caused complete pulmonary artery occlusion and sudden cardiac arrest.

Our patient had a number of risk factors for thromboembolism: age >35 years, multiparity, caesarean

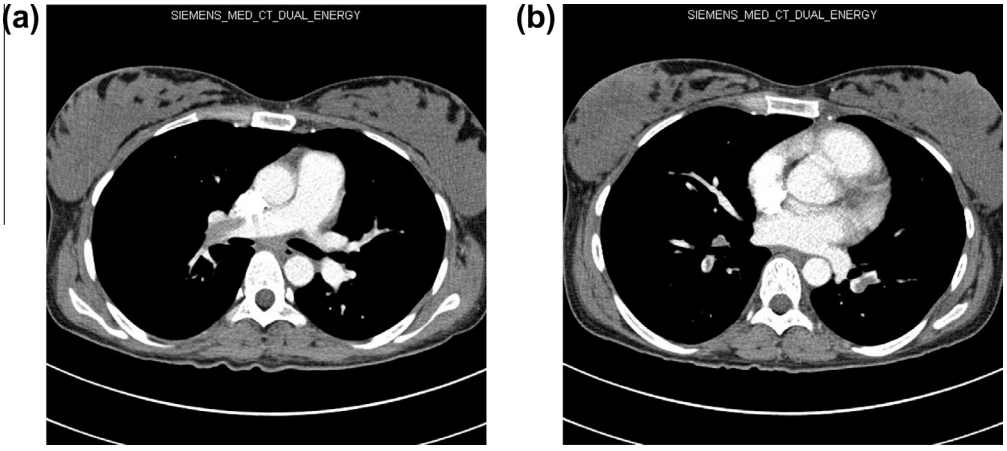

Fig. 1 CT scan showing a large thrombus in the right main pulmonary artery (a) and left lower pulmonary artery (b)