Management of severe pulmonary thromboembolism undetected by intraoperative transesophageal echocardiography: a case report

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Pulmonary thromboembolism is a fatal disease most frequently caused by deep vein thrombosis. It is important to early diagnose, prevent, and manage deep vein thrombosis and pulmonary thromboembolism. However, under general anesthesia, the clinical signs of pulmonary thromboembolism are vague, which complicates its timely diagnosis and treatment. In the present case, transesophageal echocardiography was first performed to diagnose severe pulmonary embolism during pelvic fracture surgery; after computed tomography confirmation, embolectomy was successfully performed.

Keywords: Pulmonary thromboembolism; Transesophageal echocardiography; Computed tomography; Embolectomy

INTRODUCTION

Pulmonary thromboembolism (PTE) is a life-threatening disease. However, due to its vague symptoms under general anesthesia, PTE remains a challenge for anesthesiologists to diagnose and treat [1].

Since deep vein thrombosis (DVT) is known to be the most common cause of PTE, there has been a considerable effort to prevent PTE by early identification and treatment of DVT [2]. The factors that could increase the risk of DVT include major surgery, obstetrics, lower limb problems, malignancy, and reduced mobility [3]. Therefore, the patients with

risk factors should be identified and preventive treatment, such as anticoagulation therapy and pneumocompression, should be started in the early period. Moreover, a D-dimer test can be performed to exclude PTE. Other tools useful to directly or indirectly detect DVT or PTE include computerized tomography (CT) or ultrasonography [3].

During general anesthesia, PTE can cause hypotension and shock. However, differential diagnosis is required for other possible intraoperative conditions. Transesophageal echocardiography (TEE) has the advantage of immediate feasibility in emergency cases. However, due to its relatively low specificity and sensitivity, PTE cannot be completely

ruled out even negative findings [2].

In this report, we present a case of severe PTE undetected by intraoperative TEE and discuss the importance of appropriate identification and accurate management depending on the patient's hemodynamic status.

CASE REPORT

A 168 cm, 71 kg, 53-year-old man with a right pelvic bone fracture and intra-abdominal hematoma was admitted to our hospital on the 7th post-traumatic day. The patient had no past medical history, and his preoperative laboratory tests and chest X-ray were unremarkable. Electrocardiography (ECG) showed 100-120 beats/min tachycardia, but no abnormal findings were observed. Neither DVT nor PTE was observed on CT angiography performed on the 10th post-traumatic day.

In the operating room, standard monitoring was applied. Initial vital signs were relatively stable, with blood pressure (BP) of 103/61 mmHg, pulse rate of 105 beats/min, and peripheral oxygen saturation (SpO_2) of 100%. We started induction with 140 mg propofol, 2 mg remifentanyl, and 50 mg rocuronium. After intubation, for anesthesia, we used 2.5 L/min of air, 1.5 L/min of oxygen, 2.5 vol% of sevoflurane and remifentanyl. Continuous blood pressure monitoring was performed by placing an arterial line on the patient's right radial artery and a central venous catheter on the right jugular vein. Arterial blood gas analysis (FiO_2 0.5) was performed immediately after the induction of anesthesia. The results showed pH of 7.46, partial pressure of carbon dioxide (PaCO_2) of 37.2 mmHg, partial pressure of oxygen (PaO_2) of 193.5 mmHg, hematocrit (Hct) of 24%, and initial central venous pressure (CVP) of 10 mmHg. Plasma solution was given as a peripheral line, hydroxyethyl starch (Volulyte® 6%, Fresenius Kabi Deutschland GmbH, Germany) and normal saline as the central line.

The operation started in the supine position, and the vital signs were stable; SpO_2 was 100%, end-tidal CO_2 (EtCO_2) was 36 mmHg, and CVP was 10 mmHg. At 2 hours 30 minutes after the start of the operation, a sudden massive blood loss was observed in the operative field. At the same time, BP decreased from 100/60 mmHg to 30/15 mmHg, heart rate (HR) increased from 105 to 115 beats/min, and CVP changed abruptly from 10 mmHg to 29 mmHg. Lung, heart

sound was clear, but the EtCO_2 gradually decreased from 36 mmHg to 13 mmHg, and the peripheral oxygen saturation decreased from 100% to 95%. Immediately loading the fluid, FiO_2 increased to 1.0, and norepinephrine 1 mg was continuously instilled at 0.1 $\mu\text{g}/\text{min}/\text{kg}$. When BP was checked at 30/27 mmHg, the HR suddenly decreased to below 50 beats/min. CPR was administered, and 0.1 mg pinephrine was injected 4 times at 3-minute intervals. The arterial blood gas analysis results were as follows: pH 7.28, PaCO_2 55.8 mmHg, PaO_2 98.7 mmHg, and Hct 26% during cardiopulmonary resuscitation (CPR). After 12 minutes of CPR, the patient's EtCO_2 was recovered, and SpO_2 was checked at 100%; however, systolic BP remained at 50-70 mmHg. 0.1 mg phenylephrine was administered twice over 3 minutes, and norepinephrine was administered at 0.2 $\mu\text{g}/\text{min}/\text{kg}$. After 5 minutes, the patient's BP returned to 92/31 mmHg, the arterial blood gas analysis results at that time were pH 7.12, PaCO_2 70.5 mmHg, and PaO_2 262.1 mmHg, Hct 24%.

In view of the hemodynamic change and the arterial blood gas analysis findings, PTE was thought to have occurred. Therefore, we checked TEE; PTE was not seen directly. However, the right atrium and right ventricle were dilated, and global wall hypokinesia was observed. Chest radiograph was performed in the operative field; however, no specificity was found. 3 pint fresh frozen plasma, packed RBC 2 pint were transfused, BP was 91/50 mmHg, HR 115 beats/min checked, while maintaining the booster. In addition, we performed chest CT to confirm the massive PTE in both pulmonary arteries (Fig. 1). Since the amount of PTE was massive, after consultation with a cardiac surgeon, it was decided to perform surgical intervention. The cardiac surgeon performed sternotomy and embolectomy under cardiopulmonary bypass. In the right pulmonary artery and the left pulmonary artery, many thromboses (the larger one 40×8 mm) were removed (Fig. 2). After embolectomy, BP was 90-110/50-60 mmHg, and HR was maintained 90-100 beats/min, arterial blood gas analysis at FiO_2 0.5, pH 7.23, PaCO_2 58.2 mmHg, PaO_2 154.1 mmHg, Hct 9.8% were checked. On the 2nd day postoperatively, the right atrium and right ventricle size returned to the normal size on echocardiography. One month later, CT angiography revealed no PTE. The patient did not have any special neurologic sequelae, and the associated fractures were spontaneously fused through conservative treatment.

Fig. 1. Chest spiral CT scan with a radiocontrast agent showing multiple filling defects both at the bifurcation ("saddle" pulmonary embolism) and in the pulmonary arteries.

Fig. 2. Emboli extracted from the right and left pulmonary artery.

DISCUSSION

PTE is a life-threatening disease in which the pulmonary vessel branch is clogged with blood clots, air, fat, and tumors, blocked by the blood flow, which can lead to unless rapidly diagnosed and treated. The most common cause of PTE is DVT. The risk factors of DVT include major surgery, obstetrics, lower limb problems, malignancy, reduced mobility, history of deep vein thrombosis [3]. Hip fracture patients, such as in the present case, are typically associated with risk factors, such as low extremity fracture, trauma, immobilization, major surgery, and comorbidity [3]. Moreover, Cho et al. reported that, while DVT incidence was low in the

patients with hip fracture in Korea (2.6%), the incidence of DVT increased after 72 hour post-injury [4]. Such patients are recommended to exclude asymptomatic DVT in consideration of vascular evaluation. In this case, although DVT and PTE were not observed in preoperative CT, it was considered that the patient was in the high-risk group of DVT due to immobilization by hip fracture for about 10 days.

The clinical features of pulmonary thromboembolism under general anesthesia include tachycardia, hypotension, decrease in EtCO₂, and elevation of CVP [1]. These signs appear because acute PTE interferes with both circulation and gas exchange [5]. The pulmonary artery is blocked by emboli, which increases the afterload of the right ventricle and dilates the relatively thin walled right ventricle. As a result, tricuspid valve insufficiency and right ventricle ischemia occur due to an increase in the wall tension of right ventricle, which, in turn, causes the right heart failure. The right ventricle dilatation shifts the ventricular septum to the left, reducing the volume of the left ventricle and decreasing cardiac output of left ventricle and leading to cardiogenic shock and death. In addition, desaturation can be induced with a reduced cardiac output and hypoxaemia caused by ventilation-perfusion mismatch with a reduced blood flow in the clogged blood vessels [5].

These signs do not appear only in pulmonary embolism, but require differential diagnosis with appropriate tests. TEE is considered a primary diagnostic tool of life-threatening intraoperative acute PTE. TEE can also exclude other causes, such as right ventricular dysfunction or pericardial tamponade [2,6]. TEE is helpful to diagnose massive pulmonary embolism at the bedside within several minutes, thus minimizing the interference with resuscitation efforts [2]. However, the diagnostic sensitivity of TEE with acute PTE undergoing emergent pulmonary embolectomy during surgery is only 46% [2]. The pulmonary embolism located in the main or right pulmonary artery is easily visualized; however, visualization is not possible in the left pulmonary artery. Lobar pulmonary embolism was also not diagnosed on TEE [7]. With the help of transthoracic echocardiography (TTE), we can expect an additional range of view for examining pulmonary artery, especially the left pulmonary artery with parasternal short axis view.

Moreover, caution should be exercised not to overlook secondary signs of pulmonary arterial obstruction, which is

the right ventricle dysfunction, tricuspid regurgitation, and interatrial septum deviation shown in TEE even if there is no direct visualization of thrombus. Since negative TEE does not exclude a patient when a PTE is suspected, a radiological study to confirm PTE is necessary [2].

According to the 2014 European Heart of Cardiology (ESC) guideline, it is strongly recommended to promptly take a CT image in the event of a shock or hypotension associated acute pulmonary embolism [5]. The PIOPEd II study showed PE detection sensitivity of 87% for CT [8]. Also, CT assessed the right ventricle dysfunction and can predict prognosis of the patients with PTE. Therefore, If CT is negative, it is necessary to find other causes of unstable vital signs [9].

Despite adequate diagnosis of acute PTE, mortality remains rather high, 8-15% [10,11]. If the patient is unstable, or when a CT is difficult to take, even when no thrombus is found in the TEE, the patient strongly suspected to have an acute PTE should begin treatment. If the patient is stable, conservative treatment can be provided, whereas thrombolysis, embolectomy, and inferior vena cava (IVC) filter can be performed in hemodynamically unstable patients [12]. A previous study has reported 89% survival rate for pulmonary embolectomy in the patients with massive PTE [13]. In addition, prolonged chest compression was reported to improve the right side heart flow during cardiopulmonary resuscitation (CPR) in PTE [14]. In this case, chest compression improves the patient's circulation, which may maintain operability until surgical intervention.

In conclusion, in the present case, we suspected PTE through hemodynamic instability and specific signs during surgery in the patient at a high risk of DVT. TEE with aggressive resuscitation was immediately attempted. Although thrombus was not observed directly in TEE, PTE was confirmed by CT, and embolectomy was performed with good results. Therefore, if PTE is strongly expected for patients at high risk of DVT, it is important to immediately confirm the diagnosis by CT and provide immediate management of PTE, even if thrombus is not observed in TEE under general anesthesia.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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