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Case of intraoperative acute pulmonary embolism diagnosed by transesophageal echocardiography under general anesthesia and successfully managed with extracorporeal membrane oxygenation

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Abstract

Acute pulmonary thromboembolism (PTE), which carries a high mortality rate, is difficult to diagnose when it occurs intraoperatively. Therefore, patient prognosis depends on a prompt diagnosis by anesthesiologists. A 49-year-old woman underwent right lower extremity dissection due to a contusion of the right lower extremity caused by trauma. Eleven days after surgery, she underwent debridement for necrosis of the amputation wound. Intraoperatively, a drop in blood pressure and tachycardia were observed, and PTE was suspected based on a rapid deterioration in oxygen saturation and a drop in end-tidal carbon dioxide partial pressure. Transesophageal echocardiography (TEE) showed a thrombus filling the right pulmonary artery, and a diagnosis of PTE was made. The patient was treated using venoarterial extracorporeal membrane oxygenation, and thrombectomy was performed the next day to save her life. In this case, we were able to diagnose and treat the intraoperative acute PTE at an early stage. In addition, the appropriate choice of treatment saved the patient's life without complications.

Keywords

Transesophageal echocardiography, pulmonary thromboembolism, venoarterial extracorporeal membrane oxygenation, general anesthesia, debridement

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Introduction

Perioperative acute pulmonary thromboembolism (PTE) is a relatively rare complication. Once it occurs, however, it can lead to serious outcomes and, in some cases, even death. The development of PTE during surgery under general anesthesia is difficult to diagnose, which can lead to a delay in diagnosis and subsequent treatment.[1] We report a case in which intraoperative PTE was diagnosed by transesophageal echocardiography (TEE), allowing prompt and effective treatment.

Case presentation

A 49-year-old woman (160 cm tall, weighing 50 kg), with histories of depression and adjustment disorder, was injured in a collision accident with a train, and was rushed to our hospital in a state of cardiopulmonary arrest and with her left leg amputated by the trauma. After arriving at the hospital, she was immediately intubated and ventilated, and resuscitation was continued. She was diagnosed as being in cardiac arrest due to hemorrhagic shock, and emergency thoracotomy and aortic occlusion were performed. Her heartbeat resumed within approximately 20 min, following massive fluid infusion and blood transfusion. Subsequently, the aortic occlusion was converted to percutaneous aortic blockade and the chest was closed, and transection of the right hip and angioembolization of the right superior gluteal artery were performed. She was extubated on the ninth hospitalization day and was given 1 L/min of oxygen by nasal inhalation to

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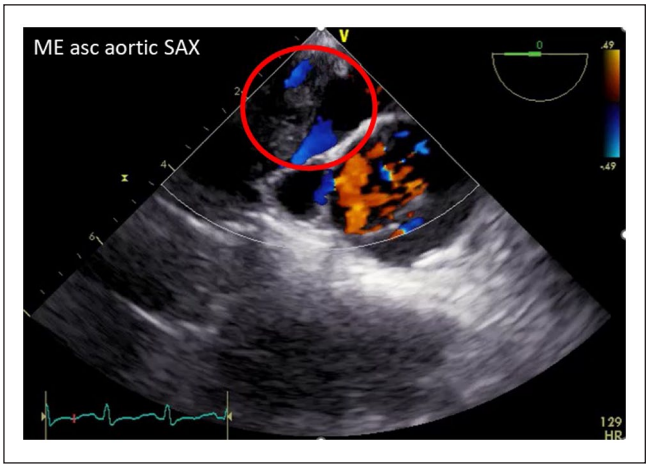

Figure 1. Transesophageal echocardiography findings.
Transesophageal echocardiography showed a thrombus in the right pulmonary artery (red circle).

maintain SpO_2 at above 97%. Blood laboratory test results at this time revealed a prothrombin time–international normalized ratio of 1.09, activated partial thromboplastin time of 25.5 s, fibrinogen level of 238 mg/dL, antithrombin-III (AT-III) of 107.9%, and elevated D-dimer level of 23.59 µg/mL. Thoracoabdominal X-rays showed fractures at Th10, 11, and L3–5, and magnetic resonance imaging showed findings of spinal cord injury at the Th7–11 levels. Along with other treatments, as required, perioperative deep vein thrombosis (DVT) prophylaxis was applied, in the form of withholding of anticoagulation, application of elastic stockings to the lower extremities and intermittent pneumatic compression, without performing lower extremity vascular echocardiography. The patient was scheduled for debridement on the 11th day after right hip dissection surgery, and heparin was to be started thereafter. For debridement, anesthesia was induced with propofol at a target concentration of 2.0 µg/mL, along with remifentanil 0.2 µg/kg/min, followed by rocuronium 50 mg. Anesthesia maintenance was performed with propofol at a target concentration of 1.5–2 µg/mL and remifentanil 0.1–0.2 µg/kg/min, with rocuronium, as needed, for muscle relaxation. Phenylephrine was administered as needed to maintain blood pressure. Intraoperatively, the patient was monitored using standard monitoring techniques, including direct measurement of arterial pressure, electrocardiography, SpO_2, and bispectral index. Blood gas analysis showed a partial pressure of arterial oxygen (PaO_2) of 121.3 mmHg and a partial pressure of arterial carbon dioxide ($PaCO_2$) of 38.5 mmHg at a fraction of inspiratory oxygen (FiO_2) of 0.5 at the start of surgery. Approximately 1 h after the start of surgery, her SpO_2 decreased suddenly from 98% to the 60% level, heart rate increased (from 77 to 94 bpm), and blood pressure decreased (from 78/52 to 64/45 mmHg). End-tidal carbon dioxide tension ($EtCO_2$) also decreased (from 33 to 25 mmHg) at the same instant. Although her blood pressure increased with the administration of a vasopressor, poor oxygenation persisted and $EtCO_2$ remained low (19 mmHg). Blood gas analysis performed at this time showed a PaO_2 of 53.9 mmHg and $PaCO_2$ of 49.9 mmHg at an FiO_2 of 1.0. Based on these findings, we suspected the development of PTE and performed transthoracic echocardiography (TTE). Evaluation revealed pressure overload in the right ventricle. Then, TEE was performed, which revealed a thrombus in the right pulmonary artery (Figure 1). After consulting with a cardiovascular surgeon, venoarterial extracorporeal membrane oxygenation (VA-ECMO) with femoral artery pumping and femoral vein debridement was performed. Following VA-ECMO initiation, her oxygenation improved and hemodynamics stabilized. Since the patient had recently undergone traumatic amputation of her lower leg and there were wounds all over her body, thrombolytic therapy was not administered since it could have increased the risk of bleeding and infection. Contrast-enhanced computed tomography (CT) was performed, which revealed a thrombus in the pulmonary artery (Figure 2). The next day, thrombectomy was performed under an open chest. No anticoagulation was used preoperatively, and the activated clotting time remained between 150 and 180 s while on VA-ECMO. After pulmonary artery thrombectomy, an organic thrombus was also found at the inferior vena cava side of the right atrium, and a dark red to partly white thrombus was observed in the right pulmonary artery (Figure 3). The patient's post-thrombectomy course was uneventful, with the maintenance of hemodynamics and oxygenation. Postoperatively, the patient was admitted to the intensive care unit under sedation and intubation and was extubated on the third postoperative day. Thereafter, she had a good course with no recurrence of PTE. She had no neurological complications.

Discussion

Venous thromboembolism and DVT, including PTE, remain serious complications during surgery. Acute onset of PTE is, reportedly, one of the leading causes of death in hospitalized patients, including surgical patients.[2,3]

Intraoperatively, under general anesthesia, patients cannot complain of the typical symptoms of PTE, including dyspnea and chest pain, making it difficult to diagnose acute-onset PTE intraoperatively.[1] Sudden and unexplained tachycardia, hypotension, hypoxemia, and decreased $EtCO_2$ are possible signs of acute PTE,[4] with the differential diagnosis including anaphylactic shock and hemorrhage. Our patient showed no cutaneous signs of anaphylaxis, such as redness or rash, and had no bleeding from the operative field, ruling out both anaphylaxis and hemorrhage. In our patient, since she had just undergone surgery, contrast-enhanced CT could not be performed, and hence, we performed TTE to diagnose the cause of the hypoxia and hemodynamic instability. PTE was strongly suspected based on the observation of pressure overload in the right ventricle, and detection of thrombus by

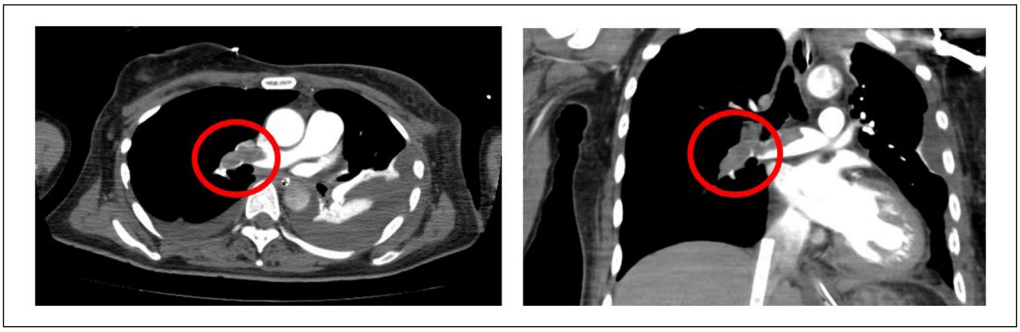

Figure 2. Contrast-enhanced computed tomography (CT) imaging findings.
Contrast-enhanced CT images showed a thrombus in the right pulmonary artery (red circles).

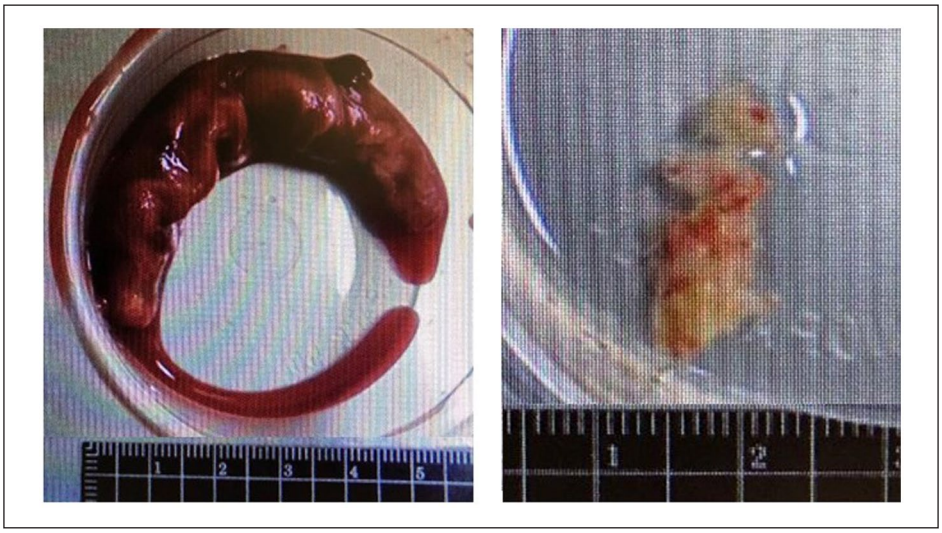

Figure 3. Removed blood clots
The thrombus was surgically removed.

TEE, which led to a definitive diagnosis and early intervention. In this case, although the diagnosis was confirmed by TEE, the process of making a diagnosis by the combination of clinical evaluation and imaging studies, based on a rapid drop in $ETCO_2$ and confirmation of right heart load findings on ECG and TTE, was equally important. In addition, circulatory dynamics at the onset of PTE are an important predictive factor of the outcome. Regarding the circulatory dynamics at the onset of PTE, the mortality is 65% in cases with cardiac arrest and 30% in those in a state of shock; in patients with stable circulatory dynamics, the mortality is said to be about 2%.[5] In our patient, although she was in shock and was considered to have a poor prognosis, early intervention saved her life.

There are four available therapeutic options for PTE: anticoagulation, thrombolytic therapy, catheterization, and pulmonary artery thrombectomy.[6–8] The treatment strategy is selected from among them according to the patient's circulatory dynamics, respiratory status, and bleeding risk. In addition, current guidelines recommend using VA-ECMO as an aid to circulation in patients with acute-onset PTE and refractory shock in high-risk groups until surgical embolectomy or catheterization is applied.[3] In our case, since the patient was in shock right at the onset, VA-ECMO was performed until surgical treatment could be performed, since she was considered a high-risk candidate based on her clinical course and the high risk of bleeding. Thrombolysis should be considered if circulation cannot be maintained and at facilities where ECMO cannot be used to control circulation immediately.[9] In retrospect, reflecting on our management of this case, we should have conducted an ultrasound of the leg veins or performed contrast-enhanced CT when we observed the elevated D-dimer level after extubation on day 9. Anticoagulation therapy should also have been considered at that time. PTE progresses rapidly and is often fatal. Hence, its early diagnosis and treatment are important for saving the patient's life,[10] and the disease should be considered as one of the possible differential diagnoses in cases with unexplained changes in hemodynamics and oxygenation perioperatively, as in this case. In

addition, while there are several reports of PTE in the perioperative period that were treated with anticoagulation,[1,10,11] there are only a few reports of its surgical treatment. Therefore, this case is significant to report.

Conclusion

We experienced a case of intraoperative PTE that was suspected based on the presence of unstable hemodynamics and hypoxemia, and in which an early definitive diagnosis of PTE was made using TEE, which led to successful early stabilization of the circulatory dynamics by VA-ECMO and salvage of the patient.

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Authors' contributions

AK and YI contributed to the intensive care management of the patient, conceptualization of the case report, and writing of the original draft. YI edited the manuscript. AK was the overall supervisor of this case. All authors read and approved the final manuscript.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and the accompanying images.

Declaration of conflicting interests

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Ethics approval

Our institution does not require ethical approval for reporting individual cases or case series.

Informed consent

Written informed consent was obtained from the patient(s) for their anonymized information to be published in this article.

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