Case Report

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Management of intraoperative acute pulmonary embolism in a patient with subarachnoid haemorrhage undergoing femoral fracture repair

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Abstract

Acute pulmonary embolism (APE) during surgery can be life-threatening. We herein report a case of a 56-year-old man with subarachnoid haemorrhage who underwent surgical repair of a femoral fracture. During surgery with the patient under general anaesthesia, his oxygen saturation and end-tidal carbon dioxide decreased dramatically. An emergency transoesophageal echocardiogram demonstrated mobile echogenic densities in the right pulmonary artery and enlargement of the right atrium, and these findings were suggestive of APE. Considering the patient's history of subarachnoid haemorrhage, anticoagulation with heparin or thrombolysis therapy for APE was contraindicated. We recommended inferior vena cava filter placement to prevent recurrence of the APE. Unfortunately, the patient and his family members refused the filter implantation, and the patient was discharged.

Keywords

Acute pulmonary embolism, general anaesthesia, transoesophageal echocardiogram, subarachnoid haemorrhage, inferior vena cava filter, case report

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Introduction

Acute pulmonary embolism (APE) during surgery can be life-threatening. The incidence of APE ranges from 0.3% to 30.0% in different surgical groups, and the highest

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incidence is found in orthopaedic patients.[1] Symptoms such as dyspnoea, chest pain, haemoptysis, and syncope can be indicators of APE.[2,3] However, the presence of these typical characteristics is difficult to determine in patients under general anaesthesia, increasing the difficulty of early diagnosis and timely treatment. Intraoperative transoesophageal echocardiography (TEE) can provide direct evidence of APE. This report describes a case involving a patient who developed intraoperative APE during general anaesthesia. This case demonstrates the value of using echocardiography in the diagnosis of APE.

Case presentation

A 56-year-old man with a history of smoking was admitted to the emergency department for treatment of a femoral fracture. Falling from a height had led to a slight disturbance of consciousness. Brain computed tomography (CT) revealed a laceration in the right temporal lobe of the brain and subarachnoid bleeding. Neurosurgical consultation showed no abnormalities. Chest CT showed changes indicating hypostatic pneumonia in both lungs. The patient had no history of hypertension, diabetes mellitus, or heart disease. The results of laboratory tests were within normal limits. The patient did not undergo routine anticoagulant therapy before surgery because of the subarachnoid haemorrhage.

On the third day of hospitalisation, the patient was scheduled to undergo surgery for internal fixation of the femoral fracture. In the operating room, peripheral intravenous access was established in the right upper limb, and standard monitors were placed. The Allen test was performed, and the left radial artery was then cannulated for continuous blood pressure monitoring. The monitors showed a blood pressure of 124/76 mmHg, heart rate of 108 bpm, and oxygen saturation of 95%. Considering the patient's anxiety, the anaesthesiologist administered 70 μg of dexmedetomidine intravenously over 10 minutes. For anaesthetic induction, the patient received midazolam (4 mg), sufentanil (50 μg), etomidate (15 mg), and cisatracurium (12 mg). After intubation, the anaesthesiologist auscultated the chest and confirmed good bilateral air entry. Anaesthesia was maintained with remifentanil (0.1–0.2 μg/kg/minute), sevoflurane (2%) in an air–oxygen mixture, and dexmedetomidine (0.4 μg/kg/h). Cisatracurium (5 mg) was administered at 1-hour intervals to maintain muscle relaxation. Considering the intraoperative application of vasoactive drugs, the anaesthesiologist inserted the central venous line through the right internal jugular vein.

Approximately 1 hour after the surgery was started, the anaesthesiologist noticed that the patient's end-tidal carbon dioxide ($ETCO_2$) had decreased from 35 to 15 mmHg within a few seconds. The oxygen saturation decreased to 80%, and the blood pressure dropped to 80/50 mmHg. However, no significant change in the airway pressure was noted. The anaesthesiologist immediately adjusted the mechanical ventilation to manual ventilation and auscultated the bilateral breath sounds. The heart rate gradually increased to 140 bpm, and an electrocardiogram showed atrial fibrillation. The central venous pressure increased to 28 cmH_2O. Intravenous administration of ephedrine (6 mg) was repeated, and 100% oxygen was administered via the endotracheal tube; this was followed by administration of epinephrine (10 μg). However, these protocols did not improve the patient's haemodynamics. Instant arterial blood gas analysis (ABGA) showed hypercapnia (Table 1). Emergency TEE demonstrated mobile echogenic densities in the right pulmonary artery and enlargement of the right atrium, and these findings were suggestive of APE (see Supplemental Video 1). Multidisciplinary consultation started immediately.

Table 1. Arterial blood gas analysis results.

Parameter	T_0	T_1	T_2
pH	7.38	7.13	7.26
pCO_2 (mmHg)	41	58.5	56.7
pO_2 (mmHg)	70	52	63.4
SO_2 (%)	94.6	67.8	93.2
Lac (mmol/L)	0.7	1.4	1.6
ctO_2 (mmol/L)	19.6	13.4	17.7
HCO_3^- (mmol/L)	24.7	22.6	26.5
ABE (mmol/L)	−0.1	−5.7	−3.1
SBE (mmol/L)	0.2	4.2	2.0

T_0: time point at which the patient entered the operating room.
T_1: time point at which respiratory and haemodynamic changes occurred.
T_2: 10 minutes after T_1; time point at which the patient's condition became stable.
pCO_2, partial pressure of carbon dioxide; pO_2, partial pressure of oxygen; SO_2, oxygen saturation; Lac, lactate; ctO_2, oxygen content; HCO_3^-, bicarbonate; ABE, actual base excess; SBE, standard base excess.

The following treatment was administered: epinephrine (0.05 μg/kg/minute) and norepinephrine (0.03 μg/kg/minute), which were administered continuously; methylprednisolone (200 mg); and 5% sodium bicarbonate liquid (100 mL).

The patient was transferred to the intensive care unit, and the operation was postponed. A follow-up lower extremity Doppler study indicated intraluminal obstructions in the left superficial femoral vein and popliteal vein. Echocardiography suggested slight enlargement of the right heart, a severe increase in the pulmonary artery pressure, and moderate mitral regurgitation. The D-dimer level was high at 36.25 μg/mL (reference range, 0.0–0.3 μg/mL). These results further supported the diagnosis of APE noted on TEE. Considering his history of subarachnoid haemorrhage, the patient could not undergo anticoagulation with heparin or thrombolysis therapy for APE. We recommended placement of an inferior vena cava (IVC) filter to prevent APE recurrence after a multidisciplinary risk–benefit discussion when the patient's haemodynamic parameters became temporarily stable. Unfortunately, the patient and his family members refused the IVC implantation, and the patient was discharged.

The patient provided written informed consent for publication of this case report. Approval by an ethics committee was unnecessary because of the nature of this study (case report).

Discussion

APE is associated with significant morbidity and mortality during the perioperative period.[4] The source of the PE may be thrombosis, gas, amniotic fluid, or tumour tissue. The most common cause of PE is deep vein thrombosis.[5] In our case, the most likely source of the PE was thrombosis caused by venous thrombosis in the lower extremities. First, orthopaedic surgery was a high-risk factor for pulmonary thromboembolism, although no thrombus was present before the surgery. Second, anticoagulant therapy was not performed before surgery because of mild subarachnoid haemorrhage.

TEE is generally considered to be the primary diagnostic technique for identifying intraoperative PE because of its high safety, availability, and utility in the operating room and its lack of interference with resuscitation efforts; its high diagnostic utility in nonsurgical settings has also been reported.[9,10] A report of patients undergoing pulmonary embolectomy for severe PE showed a survival rate of 89% and suggested that the survival rate was associated with early diagnosis and surgical intervention.[11] Trained doctors who perform TEE in a timely manner can quickly diagnose PE and guide haemodynamically unstable resuscitation in patients with APE.[12] In the present case, TEE examination revealed thromboembolism, which serves as direct

confirmation of PE, thus leading to early medical intervention and good outcomes.

We recommend performance of a predictive trial to estimate the risk of PE in orthopaedic patients undergoing surgical intervention. Anaesthesiologists can use two scoring tests: the improved Wells scoring system and the revised Geneva scoring system.[13,14] Based on the cumulative score of either of these tests, the patient can be classified as being at low, intermediate, or high risk of PE. In high-risk patients, as in our case, surgery must be postponed and a chest CT angiography scan must be performed.[15] In intermediate-risk patients, however, we recommend the use of a prophylactic anticoagulant prior to surgery.

Based on the combination of the TEE results and clinical manifestations, the most likely cause of the haemodynamic and respiratory changes in cases such as ours is pulmonary thromboembolism. Once diagnosed, the treatment options include thrombolysis, IVC filter placement, and surgical embolectomy. Anticoagulant therapy is a basic method for the effective treatment of PE; in patients with cerebral haemorrhage, however, the effectiveness and safety of anticoagulant therapy have not been fully confirmed by clinical data.[16,17] Thrombolytic therapy is another treatment choice for PE because it can quickly restore lung perfusion. However, it carries a significant risk of bleeding, especially when predisposing conditions or comorbidities exist. In particular, a head injury within 3 weeks is considered an absolute contraindication for thrombolysis.[5] In the present case, we did not consider thrombolytic or anticoagulant therapy for the APE when weighing the risk of bleeding with the possible benefits of clinical thrombolysis.

We suggested that our patient undergo implantation of a recyclable IVC filter to prevent APE recurrence. IVC filters can reportedly be used when there are absolute contraindications to anticoagulation and a high risk of venous thromboembolism recurrence.[18] One study showed that the use of IVC filters and standard anticoagulant therapy significantly reduced the incidence of PE compared with anticoagulant therapy alone, but the treatment had no effect on mortality.[19] In addition, surgical thrombectomy can be life-saving in patients with PE. Because of the complication of cerebral haemorrhage, however, the effectiveness of this method is only based on a few reported cases.[20]

In summary, intraoperative APE is a major complication in orthopaedic patients. The anaesthesiologist must perform a risk assessment before surgery. The timely use of TEE during surgery allows the anaesthesiologist to make a rapid and accurate diagnosis and adopt effective responses and management. Moreover, PE may occur before surgery in patients with cerebral haemorrhage, and clinical treatment is complicated. We must find better ways to prevent this condition and treat such patients.

Declaration of conflicting interest

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Supplemental material

Supplemental material for this article is available online.

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