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Cardiac arrest following induction of anesthesia in a patient with acute massive pulmonary thromboembolism

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Introduction

Patients with massive pulmonary thromboembolism (PE) occasionally develop acute right ventricular dysfunction followed by circulatory collapse or sudden death. Early diagnosis and aggressive treatment of this condition are therefore essential [1]. The present report is a case of cardiac arrest following induction of anesthesia for pulmonary thrombectomy in a patient with acute massive PE after lobectomy.

Case report

A 61-year-old woman had right upper lobectomy for lung cancer. She started walking on the third postoperative day. However, she noted recurring intermittent palpitation and chest discomfort following mild exertion, which disappeared following oxygen inhalation. Chest X-rays obtained on the 11th postoperative day showed a hyperlucent region in the right middle lung field and large quantities of right intrathoracic effusion. The patient experienced shortness of breath that became progressively worse until the 12th postoperative day. The patient presented with tachycardia and tachypnea. Her arterial blood gas values during oxygen inhalation at 4 l·min^{-1} through a face mask were as follows: PaO_2, 73 mmHg; $PaCO_2$, 29 mmHg; and pH, 7.48 on the 14th postoperative day.

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Computed tomographic (CT) examination revealed massive thrombosis in the deep vein of the right lower extremity and bilateral pulmonary arteries. Pulmonary angiography disclosed acute dilatation of the right atrium and ventricle, enlargement of bilateral pulmonary arteries, and a large quantity of residual contrast medium in the right ventricle during aortic visualization, indicating decreased pulmonary blood flow and cardiac output. An inferior vena caval filter was positioned percutaneously via the right internal jugular vein just below the bifurcation of the renal vein. A Swan–Ganz catheter was then inserted via the right cubital vein and direct emergency pulmonary thrombectomy under cardiopulmonary bypass (CPB) was then scheduled. Urokinase (200000 units) was administered intravenously.

The patient's condition just prior to induction of anesthesia was as follows: heart rate 98 beats/min, arterial blood pressure 100/70 mmHg, pulmonary arterial pressure (PAP) 77/33 (mean 45) mmHg. We planned initially to introduce assisted circulation accompanied by percutaneous cardiopulmonary bypass support (PCPS) before induction of anesthesia. However, the drainage cannula could not be inserted via the femoral vein to the right atrium because of the filter positioned in the inferior vena cava. Therefore, we decided to use the subclavian vein as the drainage root of PCPS after induction of anesthesia.

Induction of anesthesia was performed using midazolam (10 mg) and continuous fentanyl infusion (0.1–0.3 μg·kg^{-1}·min^{-1}) under 100% oxygen. Moderate hypotension became apparent during insertion of a double-lumen central venous cannula into the right internal jugular vein for central venous pressure monitoring and drug administration. However, this was effectively overcome by intermittent i.v. bolus administration of ephedrine (4 mg). Soon after the cannulation, abrupt profound hypotension, severe cyanosis, and rapid decrease in oxygen saturation to 68% were noted.

Cardiac resuscitation, including closed and open cardiac massage, was continued for 30 min until CPB was initiated.

Following sternotomy, the main pulmonary artery was opened. Extensive thrombosis was found throughout most of the pulmonary arterial branches. A large amount of the thrombosis was removed using forceps and a Fogarty catheter. The patient could not be weaned from CPB despite administration of a large dose of inotropics. Therefore, PCPS was initiated at $2\,l \cdot min^{-1}$ through the drainage cannula inserted via the left subclavian vein and intra-aortic balloon pumping was also applied. These procedures allowed the patient to be weaned from CPB and the operation was performed. She was then transferred to the intensive care unit. Her postoperative maximum mean PAP remained around 30–35 mmHg. The patient died on the second day after pulmonary thrombectomy due to acute renal failure.

Discussion

Several problems related to our anesthetic management became apparent in the present case. The patient already presented severe dyspnea, cyanosis, and mean PAP of 45 mmHg prior to induction of anesthesia. McIntyre and Sasahara [2] reported that a mean PAP of 36 mmHg correlates with 50% pulmonary vascular obstruction. Mitchell and Trulock [3] reported that 50% obstruction of the pulmonary vasculature usually results in right ventricular dysfunction, leading to a patient mortality rate of between 43% and 80% in the first 2 h, and 85% after 6 h. Considering these results, the patient suffered from rapidly deteriorating right ventricular dysfunction, and thus was highly susceptible to latent circulatory collapse. In the present case, we underestimated the severity of the patient's condition, and thus hastily induced anesthesia without providing adequate circulatory support.

PCPS has been used to stabilize hemodynamics in patients with massive PE when hypotension persists or right ventricular dysfunction develops. Norita et al. [4] reported successful treatment for patients who developed cardio respiratory collapse or cardiac arrest, and subsequently underwent emergency pulmonary thrombectomy under PCPS.

Prophylactic percutaneous placement of the inferior vena caval filter is appropriate generally when there is recurrent PE with deep venous thrombosis, and when anticoagulation therapy is ineffective or contraindicated [5]. In the present case, however, we initially inserted the unretracted filter despite evidence of marked right ventricular depression. This filter ultimately presented an impediment to insertion of the drainage cannula for PCPS. Based on this knowledge, the cannula should have been introduced first in order to perform life-saving PCPS, and then the filter could have been inserted if necessary after pulmonary thrombectomy was completed. Furthermore, immediately after the patient arrived in the operating room, the drainage cannula should have been inserted via the subclavian or the jugular vein under local anesthesia [6].

Hypotension induced by midazolam and fentanyl [7], as well as a reduction in venous return that was exacerbated by an elevation in intrapleural pressure due to positive pressure ventilation [6], may have triggered a serious decrease in pulmonary blood flow and cardiac output, resulting in cardiac arrest. Splinter et al. [8] reported a pregnant patient with massive PE who underwent successful emergency Cesarean section followed by pulmonary thrombectomy. They used ketamine since it preserves hemodynamics and is well tolerated by cyanotic patients. Thus, cautious use of ketamine in combination with an initial infusion of inotropic agents is preferable for induction of anesthesia.

A Swan–Ganz catheter might provide some information concerning the diagnosis and treatment of PE. However, the present patient presented with acute massive PE, therefore the Swan–Ganz catheter should not have been inserted due to the high risk of worsening the pulmonary vascular obstruction which could ultimately have led to cardiac arrest. If in a life-threatening situation, acute massive PE is confirmed by some deteriorating symptoms and/or noninvasive monitoring including CT examination, lung scintigraphy, and transesophageal echocardiography, open pulmonary thromboectomy should be considered as soon as possible in order to ensure better prognosis.

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