

Negative Pressure Pulmonary Edema Occurring Immediately after Tracheal Extubation

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Abstract

A 65-year-old man underwent a partial hepatectomy for metastatic liver cancer, with no marked changes in hemodynamic or respiratory status. However, severe respiratory insufficiency developed immediately after extubation. Chest radiography showed a butterfly shadow and frothy sputum was suctioned from the tracheal tube on reintubation, and acute pulmonary edema was diagnosed. When acute pulmonary edema occurs immediately after extubation, chest radiography and echocardiography must be performed first to distinguish between cardiogenic and non-cardiogenic pulmonary edema, because of the different potential underlying causes and treatments. In this patient, a cardiogenic cause was ruled out, and we concluded that upper airway obstruction and excessive negative intrathoracic pressure led to pulmonary edema.

Key words; negative pressure pulmonary edema, extubation, anesthetic complication

Introduction

When acute pulmonary edema occurs, differential diagnosis between cardiogenic and non-cardiogenic pulmonary edema is crucial. Cardiogenic pulmonary edema may be due to myocardial infarction, aortic dissection, or acute renal failure, and urgent treatment is usually necessary. During anesthesia, patients cannot

complain of symptoms, so diagnosis is often delayed. Although diagnosis is not always easy, the most important point is to rule out cardiogenic pulmonary edema. On the other hand, well-known causes of non-cardiogenic pulmonary edema include acute respiratory distress syndrome (ARDS), re-expansion pulmonary edema, neurogenic pulmonary edema, and transfusion-related acute lung injury. Negative pressure pulmonary edema must also be included in differential diagnosis of non-cardiogenic pulmonary edema.

Case presentation

A 65-year-old man (height, 166 cm; weight, 65 kg) was scheduled to undergo a partial hepatectomy (S₈) for metastatic liver cancer. His past medical history included a partial lung resection for hamartoma at 47 years old, acute pericarditis at 61 years old, and a lower anterior resection for rectal cancer at 64 years old. Preoperatively, blood tests showed no abnormalities, with total protein (TP) at 7.0 g/dL and albumin (Alb) at 4.1 g/dL. Chest radiography also revealed no abnormalities, with a cardiothoracic ratio of 50%. Furthermore, pulmonary function tests showed vital capacity of 3.44 L, forced expiratory volume in 1 second (FEV_{1.0}) of 2.53 L, and percent predicted FEV_{1.0} (FEV_{1.0}%) at 75%. Electrocardiography findings were normal.

Upon entering the operating room for surgery, vital signs were normal, blood pressure was 118/78 mmHg, heart rate was 80 beats/minute, and oxygen saturation (SpO₂) was 99% (room air). An epidural catheter was placed in the T8-T9 vertebral interspace. Anes-

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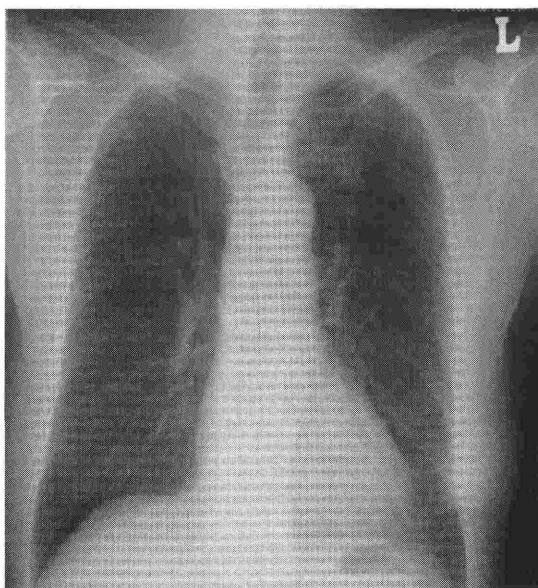
thetia was induced using 80 mg of propofol and 200 μ g of fentanyl, and muscle relaxation was achieved with 6 mg of vecuronium, then tracheal intubation was performed. Anesthesia was maintained with air, oxygen, sevoflurane, fentanyl, vecuronium, and continuous epidural infusion of 1.5% xylocaine at 4–10 mL/hour. Heart rate increased (100 bpm) and blood pressure decreased (80/50 mmHg) from the start of the hepatectomy, thus 2000 mL of colloid solution was infused over 2 hours.

Blood gas data at 1 hour prior to surgery completion showed no abnormalities [pH, 7.34; PaO₂, 205 mmHg; PaCO₂, 40 mmHg; HCO₃⁻, 20.9 mEq/L; lactate, 34 mg/dL (FiO₂ 0.43)]. The operation time was 5 hours 9 minutes, blood loss was 530 mL, intraoperative fluid therapy included 1,700 mL of crystalloid fluid and 2,000 mL of colloid solution, and urine output was 760 mL. Surgery was completed without any major hemodynamic or respiratory changes. Core temperature remained at 37.6°C throughout the operation.

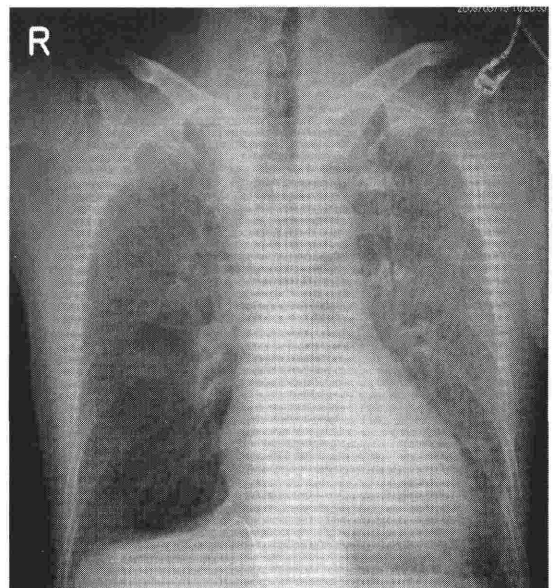
Under spontaneous respiration, tidal volume was approximately 500 mL and respiratory rate was 12 breaths/minute. Hemodynamic status remained stable, thus the muscle relaxant was stopped and the

patient extubated. However, markedly labored breathing (seesaw breathing) and tachypnea (respiratory rate, 20 breaths/minute) developed immediately after extubation. Even with O₂ at 10 L/minute given via a mask, SpO₂ was only 80% and auscultation revealed moist rales. Manual mask ventilation was attempted, however, ventilation was difficult and SpO₂ did not improve. Chest radiography showed a butterfly shadow, bilateral pulmonary edema, and a cardiothoracic ratio of 56% (Photo 1). Since mask ventilation was difficult, the patient was re-intubated under sedation. Tracheal intubation went smoothly without resistance and frothy sputum was suctioned from the tracheal tube. At this time, blood pressure was 80/50 mmHg and heart rate was 120 beats/minute.

A central venous catheter was inserted from the right internal jugular vein for measurements, which showed central venous pressure (CVP) at 5 mmHg. Transthoracic echocardiography showed no left ventricular wall motion abnormality, and no enlargement of the left atrium or left ventricle. Blood tests after the acute change showed the following: pH, 7.22; PaO₂, 85.1 mmHg; PaCO₂, 51.7 mmHg; HCO₃⁻, 20.4 mEq/L (FiO₂, 1.0); hemoglobin, 11.7 g/dL; hematocrit,



Preoperative chest X-ray



Postoperative chest X-ray

Photo 1

34.2%; TP, 3.2 g/dL; and Alb, 1.7 g/dL. The patient was subsequently managed in the intensive care unit while intubated. Postoperative urine output was adequately maintained, pulmonary edema gradually improved, and he was extubated at 3 days after the procedure.

Discussion

Pulmonary edema is a pathological condition caused by increased hydrostatic pressure in the pulmonary capillaries, decreased colloidal osmotic pressure, and increased vascular permeability. The condition is classified as cardiogenic or non-cardiogenic based on the etiology¹. In cardiogenic pulmonary edema, increased left atrial pressure, due to acute cardiac disease or fluid overload, leads to increased hydrostatic pressure in the pulmonary capillaries². On the other hand, causes of non-cardiogenic pulmonary edema include ARDS, in which vascular permeability is increased due to inflammation³, re-expansion pulmonary edema, due to disruption of vascular endothelium and increased vascular permeability when a collapsed lung is re-expanded⁴, transfusion-related acute lung injury, in which dysimmunization to anti-leukocyte antibodies in transfused blood products leads to increased vascular permeability⁵, neurogenic pulmonary edema, due to increased catecholamine levels or increased systemic inflammatory response as a result of events such as subarachnoid hemorrhage⁶, and negative pressure pulmonary edema, in which negative intrathoracic pressure leads to increased venous return and elevated hydrostatic pressure^{1,7-9}.

Our patient had a history of acute pericarditis and may have developed cardiogenic pulmonary edema due to fluid overload (2,000 mL of colloid solution was given over 2 hours). However, hemodynamic and respiratory states were normal before extubation, echocardiography showed no evidence of cardiac insufficiency, and CVP was not abnormally high, thus a cardiogenic cause was ruled out. Consequently, non-cardiogenic pulmonary edema was determined to be the major pathology in this patient and negative pres-

sure pulmonary edema was the most likely consideration. We considered that the acute upper airway obstruction was probably due to either laryngeal edema or pharyngeal secretions related to the large fluid infusion.

Negative pressure pulmonary edema was first reported clinically in 1977¹⁰, with an incidence of 94 per 100,000 surgical cases¹¹. Among patients undergoing tracheal intubation or tracheostomy after airway obstruction, a relatively large percentage (11%) have been reported to develop negative pressure pulmonary edema¹¹. The condition occurs more frequently in men up to middle age with ASA physical status 1 or 2, and is more likely in individuals suffering from sleep apnea syndrome, bronchial asthma, or severe obesity¹¹. Several causes of negative pressure pulmonary edema are known. With acute upper airway obstruction, intrathoracic pressures of -50 to -100 cmH₂O can occur, which markedly increase venous return and elevate pulmonary capillary hydrostatic pressure⁷. Another cause, related to decreased right atrial pressure, is an increase in right atrial and right ventricular volumes, which shifts the ventricular septum leftward and decreases diastolic function^{7,8}. In addition, hypoxemia or hypercapnia due to hypoventilation causes a release of catecholamines, inducing systemic vascular resistance to rise and left ventricular afterload to increase, resulting in decreased cardiac output and pulmonary congestion⁷⁻⁹. An additional cause is protein leakage and hypoproteinaemia due to direct injury to the pulmonary capillaries⁸. In our patient, preoperative TP and Alb were 7.0 g/dL and 4.1 g/dL, respectively, with marked decreases to 3.2 g/dL and 1.7 g/dL, respectively, in postoperative measurements.

The differential diagnosis included aspiration pneumonia, cardiogenic pulmonary edema, and fluid overload. During the clinical course, no events suggestive of aspiration were observed, and cardiogenic pulmonary edema and fluid overload were ruled out based on echocardiography and CVP findings. Negative pressure pulmonary edema was thus considered to be the most likely pathology in this patient.

Conclusion

When acute pulmonary edema occurs, differential diagnosis between cardiogenic and non-cardiogenic pulmonary edema is important, due to the wide differences in underlying causes and required treatments. Patients cannot complain of symptoms while under general anesthesia, so diagnosis is often delayed. However, urgent treatment is necessary if cardiogenic pulmonary edema is suspected, thus 12-lead electrocardiography, chest radiography, and echocardiography examinations should be promptly performed. If cardiogenic pulmonary edema is ruled out, negative pressure pulmonary edema must be considered in differential diagnosis of non-cardiogenic pulmonary edema.

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