Tension pneumothorax and pulmonary embolism during neurosurgery: a case report

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A tension pneumothorax arising during surgery is a rare occurrence, however it is a medical emergency that requires immediate decompression. Early diagnosis of a pneumothorax in patients with hypoxemia during general anesthesia, especially when accompanied by a pulmonary embolism during surgery, is not straightforward as it differs from the clinical symptoms seen in patients who have spontaneous ventilation. However, if it develops into a tension pneumothorax, serious hemodynamic side effects may occur. Therefore, the anesthesiologist needs a sufficient understanding of differential diagnosis methods, such as lung sound auscultation and C-arm fluoroscopy. These can be easily utilized in the operating room, and rapid treatment can be performed through consultation with thoracic surgeons.

Keywords: Pulmonary embolism; Tension pneumothorax; Thoracostomy

INTRODUCTION

The incidence of pneumothorax, in which air flows into the pleural cavity, is not common during general anesthesia, however it can occur on account of various causes such as trauma, positive pressure ventilation, rupture of air bullas, and insertion of central venous catheters [1,2]. In addition, cardiovascular collapse may occur when it progresses to a tension pneumothorax, which is characterized by tachycardia, dyspnea, hypotension, and pallor due to hypoxemia, mediastinal movement and reduced venous return. Therefore, early detection of a pneumothorax and immediate intervention are mandatory before hemodynamic exacerbation occurs [2,3]. However, a pneumothorax in patients

with mechanical ventilation during general anesthesia often exhibits a different pattern from those with spontaneous ventilation. If a pulmonary embolism is combined with a pneumothorax, early diagnosis and treatment may be more difficult [1].

We experienced a patient with hypoxemia due to a pneumothorax and pulmonary embolism during neurosurgery with left cerebellopontine angle meningioma. We report this with a review of the literature on the diagnosis and treatment of pneumothorax.

CASE REPORT

A 44-year-old male patient, height 165 cm, and weighting

54 kg, was scheduled for neurosurgery with left cerebellar craniomeningioma. He had received a previous surgery for left cerebellopontine angle meningioma two years prior and also had a history of undergoing a ventricular peritoneal shunt with hydrocephalus four months prior. A chest radiograph (CR) performed before surgery showed subsegmental atelectasis in both lower lobes. In a chest computed tomography (CT), bulla was observed in the left apical lung, and a pulmonary function test showed mild obstructive pulmonary disease with FEV1 1.70 L, FVC 2.61 L, and FEV1/FVC 65%. Electrocardiography showed no specific findings.

After entering the operating room, the Glasgow coma scale of the patient was 10. The vital signs of before induction of anesthesia were: blood pressure (BP) of 125/76 mmHg, heart rate (HR) of 83/min, and pulse oximetry (SpO₂) of 99%. After administering glycopyrrolate 0.2 mg, anesthesia was induced with 3.0 µg/mL of propofol target concentration infusion (TCI) and 3.0 ng/mL of remifentanyl TCI using a target concentration infusion pump (Orchestra® with Base Primea, Fresenius-kabi, Korea). Thereafter, total intravenous anesthesia (TIVA) was maintained until the end of surgery. 50 mg of rocuronium was administered and endotracheal intubation was performed. No neuromuscular blockers were added to monitor for motor-evoked potential during surgery. Mechanical ventilation was initiated with an inhalation oxygen fraction (FIO₂) of 0.5, tidal volume of 7 mL/kg, respiratory rate (RR) of 17/min, and positive end-expiratory pressure (PEEP) of 5 cmH₂O. To maintain the ET-CO₂ at 30–35 mmHg, RR was adjusted appropriately. Immediately after induction of anesthesia, vital signs were BP 130/92 mmHg, HR 72/min, SpO₂ 100%, and ET-CO₂ 31 mmHg. Peak airway pressure of the patient was 17 cmH₂O.

After induction of anesthesia, a central venous catheterization (CVC) was performed through the left subclavian vein for fluid administration. After inserting a 22-gauge catheter into the right radial artery and connecting it to a FloTrac manometer, the patient's cardiac output (CO), BP, and stroke volume variation (SVV) were continuously monitored through a Vigileo monitor (Edwards Lifesciences Corporation, USA).

Patient's posture was then changed from a supine to a right lateral decubitus position and the operation began. During the operation, the patient's BP was maintained at 100–150/45–95 mmHg, the HR was maintained at 65–110/

min, and the SpO₂ was 97–100%. Peak airway pressure was controlled within 20 cmH₂O. The fluid was administered with reference to the SVV. Until the end of surgery, the total amount of fluid administered was 6,200 mL of crystalloid and five units of concentrated packed red blood cells.

The operation was completed after 13 hours from the beginning of the surgery. The patient's FiO₂ was raised to 1.0 for an emergence. Mechanical ventilation was ceased and we conducted manual ventilation. Subsequently, the patient's posture was changed from the right lateral decubitus position to the supine position, and 100 mg of sugammadex was administered for complete reversal of muscle relaxation.

During the 10 minutes of emergence, the patient's BP showed 120–150/65–80 mmHg, however the SpO₂ began to decrease from 97% to 91%. The patient's airway pressure was increased to 35 cmH₂O, and an adequate tidal volume was not delivered well during manual ventilation. The SpO₂ was further reduced to 84% at 20 min after the emergence. It was decided not to extubate the patient's endotracheal tube, and 20 mg of cisatracurium was administered for muscle relaxation and mechanical ventilation was applied again. The crackle sound was confirmed by auscultation in both lungs. We suspected pulmonary edema based on the patient's poor pulmonary function and the edema of his face. After discussion with a neurosurgeon, 20 mg of Lasix and 100 mL of 15% mannitol were administered intravenously.

At 30 minutes after the emergence, the patient's BP was 110/65 mmHg, HR was 108/min, SpO₂ was 82%, and arterial blood gas analysis (ABGA) revealed pH 7.29, PaCO₂ 54 mmHg, PaO₂ 46 mmHg, HCO₃ 26.0 mmol/L, SaO₂ was 76%. At 40 minutes after the emergence, the patient's BP decreased from 110/65 mmHg to 60/35 mmHg, SpO₂ was 80%, HR was 111/min, and ABGA was pH 7.24, PaCO₂ 79 mmHg, PaO₂ 45 mmHg, HCO₃ 33.9, SaO₂ 72%. The left lung sound was significantly more reduced than the right lung sound during a subsequent chest auscultation. Considering this, a pneumothorax was suspected, and CR was taken with C-arm fluoroscopy that was placed in the operating room. A tension pneumothorax with severe collapse of the left lung was identified (Fig. 1A and B). Immediately, a thoracic surgeon was consulted and a tube thoracostomy was performed. After 10 minutes of tube thoracostomy, the pa-

Fig. 1. (A) The right lung field of the patient by C-arm fluoroscopy in the operating room. (B) The left lung field of the patient by C-arm fluoroscopy in the operating room showing tension pneumothorax (arrow). (C) Chest anterior-posterior radiography in the intensive care unit showing subsegmental atelectasis in both lower lobe and right pleural effusion and left-sided pneumothorax with chest tube.

Fig. 2. Postoperative chest CT scan. (A) Axial view of chest CT. (B) Coronal view of chest CT. There are multiple embolisms in interlobal pulmonary artery and the first branch of right pulmonary artery (arrow).

tient's BP rose to 121/73 mmHg, SpO₂ was 91%, and HR was 90/min. The results of ABGA improved slightly to pH 7.29, PaCO₂ 63 mmHg, PaO₂ 54 mmHg, HCO₃ 30.3 mmol/L, and SaO₂ 83%. When oxygen was administered with FiO₂ of 0.8, the SpO₂ was increased to 98%, and the patient was transferred to the intensive care unit (ICU). 60 min after entering the ICU, the results of ABGA performed with FiO₂ of 0.6 showed pH 7.46, PaCO₂ 37 mmHg, PaO₂ 67 mmHg, HCO₃ 25.6 mmol/L, and SaO₂ 95%. Concurrently, an additional chest radiographs performed in the ICU revealed re-expansion of the left-sided pneumothorax (Fig. 1C).

The result of the blood test performed the day after the ICU admission showed that the D-dimer rose to 7.88 µg/mL and the FDP to 19.48 µg/mL. An MRI confirmed a pulmonary embolism in the right pulmonary artery (Fig. 2A and B). Heparin and inotropic were administrated. However, the patient continued to undergo hemodynamic instability and eventually died six days after surgery.

DISCUSSION

Tension pneumothorax that occurs during surgery is rare, however it is a medical emergency that requires immediate decompression. A tension pneumothorax is a complex phenomenon manifested by central hypoxemia and its compensatory response and mechanical compression on the intrathoracic structures [2]. However, hemodynamic changes may appear slowly depending on the degree of compression on the intrathoracic structures. Therefore, it is important to diagnose a pneumothorax early based on the exacerbation of hypoxemia, tachycardia, and respiratory distress [1,4]. When a pneumothorax occurs, symptoms and signs such as chest pain, dyspnea, respiratory distress, and tachypnea may be observed in patients who were previously spontaneously breathing, and an initial approach for a differential diagnosis may be made. Thus, the incidence of hypotension (16%) and cardiac arrest (2%) in a pneumothorax in patients

with spontaneous ventilation have been reported less frequently. However, the clinical symptoms and signs, such as chest pain, dyspnea, and tachypnea, cannot be seen in patients on mechanical ventilation. Therefore, fatal conditions such as hypotension and cardiac arrest have been reported more frequently with the diagnosis of a tension pneumothorax [1]. In this case, only a decrease in the SpO₂ was initially observed, and the BP was already decreased when the diagnosis of a tension pneumothorax was confirmed. Therefore, the anesthesiologist should understand the early warning signs of a pneumothorax in patients on mechanical ventilation and should perform a rapid differential diagnosis and initiate treatment.

Diagnosis of pneumothorax can usually be confirmed through a patient's medical history, physical examination and, if possible, CR and CT scans [5,6]. CT examination is considered the gold standard for pneumothorax diagnosis, however it is not easy to move serious patients to the laboratory for CT examination. Thus, there is a time limitation for a quick diagnosis in the operating room. For a CR, the sensitivity is not high, however it is easier to access in the operating room than a CT scan [7]. For this patient, a CR was taken with C-arm fluoroscopy in the operating room. This was valuable for the diagnosis of a tension pneumothorax. In addition, a quick consultation with a thoracic surgeon and a tube thoracostomy was immediately performed in the operating room. Recently, lung ultrasound has been used to diagnose a pneumothorax using certain characteristic signs such as lung sliding, comet tail artifacts, and the A line sign [8,9]. Therefore, when reflecting on the recent situation where the prevalence of ultrasound in the operating room has increased, it is necessary for anesthesiologists to be more aware of lung ultrasound as it can assist in the differential diagnosis of a pneumothorax.

The treatment of patients with a tension pneumothorax is decompression by needle thoracostomy or tube thoracostomy [2,10]. Temporary decompression may be performed while checking the air leakage by needle thoracostomy before tube thoracostomy. Nonetheless, the definitive treatment method for tension pneumothorax remains tube thoracostomy. If a tension pneumothorax is suspected, it is essential to conduct early diagnosis and treatment through a prompt consultation with a thoracic surgeon. In this case, it took a long time to diagnose the tension pneumothorax,

and the patient's BP decreased due to the tension pneumothorax. Fortunately, diagnosis and tube thoracostomy were performed before the cardiovascular collapse worsened.

It is known that the incidence of tension pneumothorax increases in proportion to the presence and size of the bulla and the degree of adhesion of the lungs [4]. Rupture of larger bullas may occur more easily than smaller ones. In addition, the larger size of the bulla, the higher airflow and higher pressure in the pleural space when ruptured, making it easier for the development of a tension pneumothorax. This patient also had a bulla in the left upper lobe which was observed on CT scans performed prior to surgery. During general anesthesia, the patient's lateral position results in significant ventilation and perfusion mismatch. Overall, dependent lungs are well perfused and poorly ventilated, non-dependent lungs are well ventilated, but poorly perfused [11]. Therefore, special attention should be given to patients undergoing non-thoracic surgery if the bulla is present in the non-dependent lung. In this case, since CVC was performed through the left subclavian vein, it was initially suspected that a pneumothorax occurred as a complication due to CVC. However, when taking into account that there is no significant change in SpO₂ and airway pressure of the patient for 10 hours after maintaining anesthesia with FiO₂ 0.5 after CVC, the possibility of a pneumothorax caused by CVC is considered low. In this patient, it is assumed that the bulla in the left upper lobe ruptured during surgery, and the application of PEEP during mechanical ventilation may have affected the rupture of the bulla. The possibility of a pneumothorax due to the rupture of the bulla cannot be inferred quickly, and the diagnosis was delayed. It seems to have developed into a tension pneumothorax and lowered BP due to the continuous positive mechanical ventilation and PEEP.

After the decrease in SpO₂ in this patient, we suspected pulmonary edema based on the crackle sound in both lungs during the initial auscultation. It took a long time to diagnose the pneumothorax. This was because, although the fluid was administrated based on SVV according to early goal-directed fluid therapy, it was decided that a great deal of fluid was administrated during anesthesia and edema was observed around the patient's face. Excessive accumulation of extravascular lung water due to IV fluid overload may result clinically in pulmonary edema [12]. In this patient, the

lung sounds on the left side were markedly decreased when the patient was re-examined after the SpO₂ decreased, and a pneumothorax was suspected. Therefore, chest imaging was performed with a C-arm fluoroscopy. At the time of re-auscultation, the patient's pneumothorax developed into a tension pneumothorax, the collapse of the left lung became severe and the lung sounds seemed to decrease. At the early stages of the event, the left lung sound may have been decreased more than the right side, but we were not aware of the difference between the two lung sounds. Auscultation remains a valuable diagnostic tool today, despite the development of more sophisticated diagnostic tools for lung disease. In pulmonary edema, an examination using a portable CR device is a useful tool for evaluating alveolar and interstitial edema and pleural effusion, although the evaluation of the heart and blood vessel size is limited [12]. In an emergency that requires a differential diagnosis, such as pulmonary edema or a pneumothorax, an anesthesiologist need to fully understand the correct use and diagnostic methods of stethoscopes, CR, and ultrasound. These, can be easily accessed in the operating room and are still useful.

In this patient, the occurrence of a pulmonary embolism along with a pneumothorax in the operating room was not considered. The pulmonary embolism was detected by increased D-dimer levels and post-operative CT scan. The incidence of pulmonary embolism during surgery is reported 0.4% during head and neck surgery [13]. The typical findings of pulmonary embolism are hypotension and tachycardia [14]. However, depending on the degree of pulmonary artery invasion of embolism, only hypoxemia may be seen as in the case without hypotension [13]. A pulmonary embolism in patients with mechanical ventilation results in a separation phenomenon with decreased ETCO₂ and increased PaCO₂ [15]. This is the result of dead space that is increased by the embolism. In addition, reduced CO and ventilation-perfusion imbalance may contribute to hypoxemia and hypercapnia. Therefore, in patients at high risk for a pulmonary embolism, when the carbon dioxide partial pressure graph is reduced for no specific reason, careful monitoring of PaCO₂ is necessary, and if separation occurs, a pulmonary embolism can be suspected. However, in this case, ETCO₂ during surgery remained constant at 28-30 mmHg and did not decrease. In this patient, a pulmonary embolism was accompanied by a pneumothorax, however, because it

occurred in the distal portion of the right pulmonary artery, it did not seem to have a significant hemodynamic effect on the patient in the operating room. However, it seemed that it affected the patient's morbidity and persistent hypoxemia with the pneumothorax.

In conclusion, early diagnosis of a pneumothorax in patients with hypoxemia during general anesthesia is not easy because it differs from the clinical symptoms seen in patients who spontaneously ventilate. However, if it develops into a tension pneumothorax, serious hemodynamic side effects may occur. Therefore, the anesthesiologist requires a sufficient understanding of differential diagnosis methods, such as lung sound auscultation and CR, which can be easily applied in the operating room. In addition, subsequent rapid treatment through consultation with thoracic surgeons is essential.

CONFLICT OF INTEREST

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

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