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Pneumothorax of undetermined etiology occurring during general anesthesia

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Introduction

Pneumothorax during positive-pressure ventilation is an important and potentially lethal complication. Needle-related causes of pneumothorax (i.e., nerve block or central vascular catheter placement) or airway-management-related causes (i.e., instrumentation or barotraumas) are well recognized and occur in up to 3% of pneumothorax cases [1]. The incidence of pneumothorax due to barotraumas in patients receiving mechanical ventilation is between 4% and 15% [2].

Idiopathic pneumothorax is due to the formation and rupture of subpleural blebs and is related to the patient's level of tobacco consumption [3]. We report here a patient who developed a collapse of the right lower lung field during general anesthesia, as a result of pneumothorax of undetermined etiology.

Case history

A 65-year-old man (height, 162 cm; weight, 46 kg) was scheduled to receive a pylorus side gastrectomy for gastric cancer. The patient had a history of smoking (20 cigarettes per day for 50 years).

Arterial blood gas analysis under room air was performed (pH, 7.43; PaO_2, 93 mmHg; $PaCO_2$, 39.5 mmHg). A central venous catheter was placed in the right subclavian vein 4 days before the operation. A chest X-ray taken after central line insertion showed the catheter to be in an acceptable position in the superior vena cava, with no evidence of pneumothorax (Fig. 1). Preoperative laboratory data, respiratory function, and an electrocardiogram were all within normal ranges. After the patient entered the operating room, an epidural catheter was placed at the 8th and 9th thoracic level (T8–9 interspace), and no blood or central spinal fluid was drawn. General anesthesia was induced with fentanyl 0.1 mg, thiopental 250 mg, and vecuronium bromide 9 mg, and the trachea was intubated.

Anesthesia was maintained with N_2O (50%), O_2 (50%), sevoflurane (1%–1.5%), and intermittent epidural administration of 1% lidocaine. The patient was mechanically ventilated with tidal volumes of 450–550 ml (end-tidal CO_2, 35–40 mmHg) and an inspiratory peak pressure of 14–18 mmHg. A catheter was inserted via the left radial artery for arterial blood pressure monitoring and blood gas analysis. Arterial blood gas analysis before skin incision under 50% oxygen revealed pH 7.42, PaO_2 190 mmHg, $PaCO_2$ 37.6 mmHg, and SaO_2 100%.

About 2 h after the start of the surgery, we noticed a decrease in PaO_2 to 88 mmHg (pH, 7.39; $PaCO_2$, 39.4 mmHg). Intratracheal suctioning was carried out using a bronchofiberscope, and we drew a considerable quantity of secretion from both lungs. The position of the tracheal tube was correct, and a bronchofiberscopic examination showed no abnormalities in either lung. We then considered the possibility that the pneumothorax was a complication of the central venous catheter insertion, but this possibility seemed unlikely because of the absence of diminished respiratory sounds in the upper and middle lung field. Because the operation was scheduled to finish within another 30 min, we decided to proceed conservatively and gave the patient 100% O_2.

A chest radiograph was taken immediately after the end of surgery, and we observed that the right lower lung field had collapsed as a result of a pneumothorax of the inferior lobe, and that no major collapse or abnormality in the apex and middle lung field had occurred

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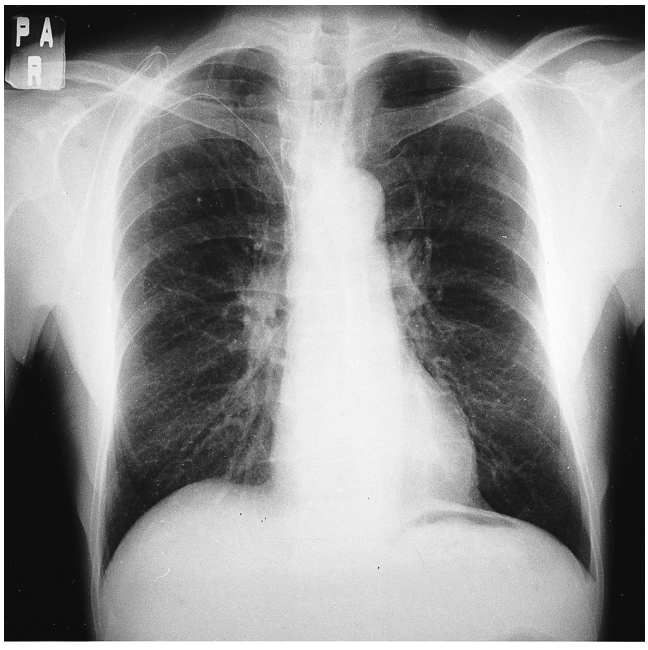

Fig. 1. A chest X-ray taken after central line insertion showed the catheter to be in an acceptable position in the superior vena cava, with no evidence of pneumothorax

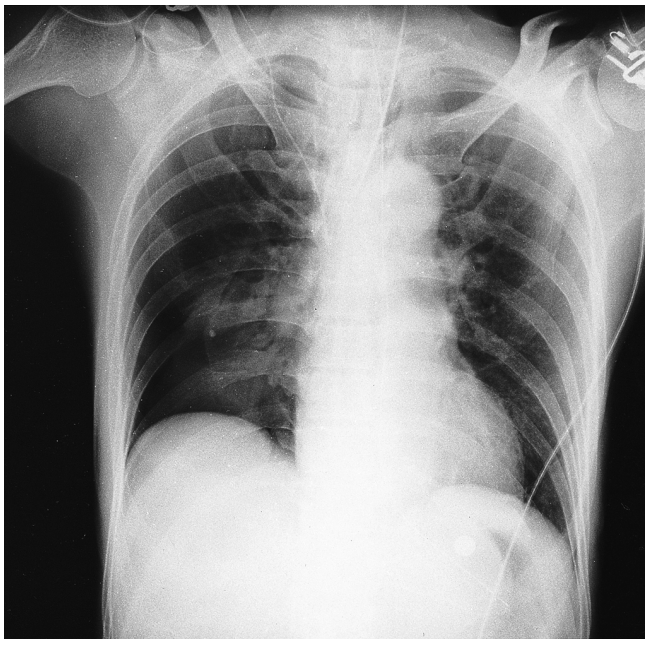

Fig. 2. A chest radiograph was taken immediately after the end of surgery. The right lower lung field had collapsed as a result of a pneumothorax of the inferior lobe, and no collapse or abnormality in the apex and middle lung field had occurred

(Fig. 2). At this point, PaO_2 was 105 mmHg (FiO_2 1.0). A chest drainage tube was immediately inserted into the right pleural cavity. After the muscle relaxant had been reversed with atropine 1.0 mg and neostigmine 2.0 mg, the patient was transferred to the intensive care unit with a tracheal tube. The tracheal tube was removed when PaO_2 had improved to 220 mmHg under 50% oxygen at 1 h after admission to the intensive care unit. Afterward, the patient's respiration and hemodynamics were stable, and he was discharged from the intensive care unit to the ward the next day.

Discussion

In the present case, although we suspected the occurrence of a pneumothorax during surgery, we could not diagnose it until the surgery had ended. Fortunately, a serious tension pneumothorax did not occur, but we should have searched for the cause of the decrease in PaO_2 earlier by taking a chest X-ray during the operation. Early detection of perioperative pneumothorax might have been possible by a careful monitoring of the decrease in lung compliance and a change in the pressure-volume loop configuration with continuous spirometry [4,5]. In this case, manual and mechanical ventilation did not reveal any change in lung compliance, and we did not detect the pneumothorax until a chest radiograph had been taken. Pneumothorax is usually either needle-related, as when caused by central vascular catheter placement or nerve block, or airway-management—related, as when caused by instrumentation or barotraumas [1].

Pneumothorax occurs during central venous catheter placement with a frequency of 1.6%–3% [1,6,7]. In the present case, the central venous catheter was placed via the right subclavian vein 4 days before the operation. No evidence of pneumothorax was revealed by a chest X-ray obtained after catheter placement. However, the diagnosis of pneumothorax may be delayed for hours or days after central venous catheterization [8]. One case has been reported in which a patient developed a tension pneumothorax under general anesthesia with mechanical ventilation 10 days after central venous line placement [8]. Accordingly, we initially suspected the occurrence of a needle-related pneumothorax in the present case. However, because of the location of the pneumothorax, we had to conclude that this etiology was unlikely. If the pneumothorax had occurred in the upper lung field, we probably could have diagnosed it by auscultation. In the present case, it would have been difficult to detect the pneumothorax by auscultation, and we should have been careful when the collapse of the lung occurred in the lower lung.

Thoracic epidural catheters have also been reported to accidentally puncture the pleural cavity and cause pneumothorax [9,10]. In the present case, because we were able to place the epidural catheter without any

difficulty, the procedure of epidural catheterization could be eliminated as a cause of the collapse of the lower lung field. We effectively used the epidural catheter with a continuous infusion of 0.25% bupivacaine postoperatively for 4 days without any complications.

Barotrauma is also an important cause of pneumothorax in patients receiving mechanical ventilation. In patients undergoing general anesthesia, pneumothorax due to barotrauma has been reported to occur with a frequency of about 0.5% [1]. Peak inspiratory pressures over 40–50 cm H_2O are associated with an increased risk of alveolar rupture during mechanical ventilation. Accordingly, it has been recommended that tidal volume be adjusted to maintain peak plateau pressures at or below 35 cm H_2O during ventilation [2,11]. In the present case, control ventilation was maintained with a peak plateau pressure of 14–18 cm H_2O. Therefore, it was also unlikely that barotrauma was the cause of the pneumothorax.

Nakamura et al. have reported that spontaneous pneumothorax is related to the level of tobacco consumption, as analyzed by the Brinkmann index, and occurs more often in tall, thin individuals as analyzed by the Kaup index [3]. In the present case, the patient was not very tall, but he was thin enough to qualify for this risk factor. More importantly, the patient had a 50-year history of smoking (Brinkmann index, 1000), and was considered to be at significantly higher risk for pneumothorax than a non-smoker. The Kaup index of this patient was 12.9, which is lower than the average for a normal man (14.84) and fell within the criteria for higher risk of pneumothorax. It is generally understood that the formation and rupture of subpleural blebs causes spontaneous pneumothorax. Positive-pressure ventilation and the use of nitrous oxide may easily cause perioperative pneumothorax in a patient with subpleural blebs. Unfortunately, in this patient we could not find any visual evidence of blebs with a CT scan. It is possible that small blebs were present that we could not differentiate. Therefore, we conclude that the pneumothorax could have been caused by either or both of these two causes. We considered that there might have been adhesion between the upper and lower lobes of the right lung and the pleural cavity, and adhesion might have minimized the collapse of the upper and lower lobes of the right lung.

In summary, perioperative pneumothorax without any obvious cause may occur in any lung field under general anesthesia, and it is important for anesthesiologists to recognize the necessity of performing a chest X-ray immediately when a pneumothorax is suspected.

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