CASE REPORT
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Contralateral Tension Pneumothorax during One Lung Ventilation by a Univent® Tube

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Tension pneumothorax during one-lung ventilation (OLV) is a rare but life-threatening complication. A 79-year-old male patient who was diagnosed with lung cancer underwent Univent® Tube (Fuji Systems Corporation, Tokyo) intubation for left upper lobectomy. Two hours after the initiation of OLV, the patient could not tolerate it. Thus, one- and two-lung ventilation were alternatively applied to continue the operation. After the operation, an emergent chest radiograph was taken, and pneumothorax was found at the right (dependent) lung field.

Key Words: Pneumothorax, Pulmonary atelectasis, Ventilation

INTRODUCTION

One-lung ventilation (OLV) is mandatory for thoracic surgery like lung lobectomy and anesthesiologists use double lumen endobronchial tube or Univent® tube for it. The Univent® tube is an endotracheal tube with a movable bronchial blocker integrated into the sidewall of the tube and is technically easy to place properly.[1] The complications related to the Univent® tube include perforation of the blocker cuff lumen by the surgeon, the inclusion of the distal end of the blocker into a resected lung staple line, and the development of a tension pneumothorax.[1,2] We report the development of tension pneumothorax in the ventilated dependent lung during OLV by means of a Univent® tube.

CASE

A 79-year-old, 72 kg, 179 cm man with a history of left pleuritic chest pain was found to have a mass in his left upper lung zone on chest radiography. After computed tomography guided lung biopsy, it was confirmed poorly differentiated carcinoma so he was scheduled to have a thoracotomy and left upper lobectomy. His past medical history included hypertension, benign prostate hypertrophy and brain tumor removal operation 25 years ago. He had made no sequelae from these and never smoked before. Chest radiography and computed tomography revealed calcific granuloma in both upper lobe due to previous infection sequelae and solid mass at left upper lobe apical segment. The lung was not emphysematous and there was no subpleural bleb. Preoperative pulmonary function test and arterial blood gas analysis (ABGA) were normal. Echocardiography showed mild pericardial effusion and normal valvular function with a left ventricular ejection fraction of 53%.

The patient was premedicated by glycopyrrolate 0.2 mg intramuscularly. Upon arrival at the operating room, standard monitoring devices, including ECG lead II, pulse oximetry and oscillometric non-invasive blood pressure were applied. After anesthesia was induced with IV propofol 100 mg and rocuronium 70 mg, tracheal intubation was done with a 7.5 mm Univent® tube easily. This tube was selected due to the possibility of postoperative mechanical ventilation. Anesthesia was maintained with O_2 2 L/min, N_2O 2 L/min and sevoflurane 1.5-2.0 vol%. Under direct visualization of the fiberoptic bronchoscopy (LF-GP, Olympus, Japan) through the tracheal lumen, the endobronchial blocker was manipulated into the left main stem bronchus with no difficulty. Correct placement

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was confirmed by visualizing the inflated cuff of the blocker in the left main stem bronchus below the carina and by auscultation. Then right radial artery was cannulated for continuous blood pressure monitoring and the right internal jugular venous catheter was inserted on the first attempt by anterior approach.

And then patient was turned into the right lateral decubitus position for the planned left thoracotomy. After completion of the patient's surgical position, correct bronchial blocker position was confirmed again using the fiberoptic bronchoscope before skin incision.

During two-lung ventilation, the exhaled tidal volume was 600 mL with a peak inspiratory pressure (PIP) of 16 cmH_2O and a respiratory frequency of 12 breaths/min. SpO_2 was 99% with an inspired fraction of oxygen (FiO_2) of 0.48. After thoracotomy, initiaton of one lung ventilation resulted in the PIP increasing to 30 cmH_2O with an exhaled tidal volume of 545 mL. So we applied pressure control mode with the pressure of 20 cmH_2O then the exhaled tidal volume was 400 mL. Over the next 10 minutes, the SpO_2 gradually decreased from 98% to 92%. So FiO_2 was increased to 1.0, continuous positive airway pressure (CPAP) 5 cmH_2O was applied to the non-dependent lung and positive endexpiratory pressure (PEEP) 5 cmH_2O was added to the dependent lung. After then, the SpO_2 increased to 96%.

Operation was continued without any event but after 2 hours from initiation of OLV, suddenly systolic arterial blood pressure declined from 121 mmHg to 79 mmHg. Simultaneously, exhaled tidal volume was decreased to 300 mL with the inspired pressure of 20 cmH_2O and the SpO_2 was decreased to 88%. At that time ABGA results were pH 7.32, $PaCO_2$ 57 mmHg, PaO_2 61 mmHg and SaO_2 87%. Immediate treatments consisted of resuming twolung ventilation and ephedrine 8 mg was injected intravenously. Then SpO_2 increased to 99% and systolic arterial blood pressure increased to 115 mmHg.

The fiberoptic visualization through the tracheal lumen was done and revealed correct position of the bronchial blocker but some bloody secretion was scattered from the blocked left main bronchus. On the auscultation, the breathing sounds were reduced and crackles were heard around the right infraclavicular area. So decreased SpO_2 was thought due to dependent lung atelectasis caused by bloody secretion passing from the nondependent lung. After the surgeon was informed, the trachea was suctioned thoroughly and we started OLV to proceed the operation. But SpO_2 rapidly decreased to 85% and we had to restart two lung ventilation again.

The surgery was continued, with a repeated unsuccessful attempt at OLV. After completion of the left upper lobectomy, two lung ventilation was resumed with pressure of 20 cmH_2O adding PEEP 5 cmH_2O. Then exhaled tidal volume was 500 mL and ABGA showed; pH 7.30, $PaCO_2$ 61 mmHg, PaO_2 76 mmHg at FiO_2 1.0, SaO_2 94%. During the surgery, the central venous pressure (CVP) was maintained at 8-10 cmH_2O and mean arterial pressure was maintained between 60 and 70 mmHg by infusion of dopamine at the rate of 5 mcg/kg/min.

After the emergent chest radiogragh was performed, pneumothorax was found in the dependent lung and the trachea deviated to the left side (Fig. 1). So the chest tube was inserted into the right thoracic cavity and SpO_2 improved to 100%. The next day, he was extubated and chest tube was removed. He was transferred to the general ward and discharged 14 days later without any complications.

DISCUSSION

During OLV, the occurrence of a pneumothorax in the dependent lung is rare but fatal to a patient. But hypoxemia, increased airway pressure, and decreased tidal volume, which are all clinical manifestations of a pneumothorax, can also occur from bronchospasm, pulmonary edema, pulmonary embolism, and aspiration.[3,4] Therefore, a systematic approach is needed for differential diagnosis.

Intraoperative pneumothorax can occur for various reasons. The surgery itself can cause pneumothorax when there is damage in the bronchus or pulmonary parenchyma during thyroide-

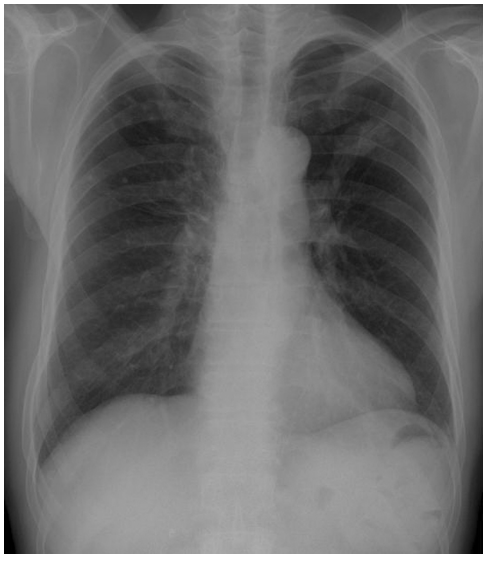

Fig. 1. Preoperative chest radiograph shows calcified granulomas in both upper lobes, and a mass lesion in the left upper lobe.

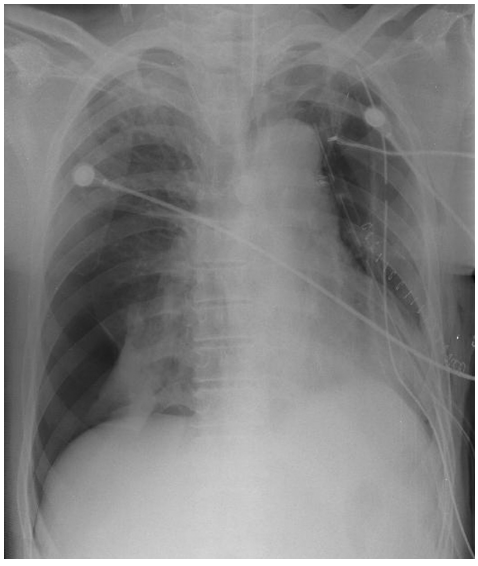

Fig. 2. Postoperative chest radiograph shows pneumothorax at the right lung and a deviated trachea on the left side.

ctomy, lymph node dissection in the neck or thoracotomy. A pneumothorax can also occur following an excessive increase in airway pressure due to the malfunction of anesthetic devices or from lung parenchymal injury during thoracic epidural or central venous catheter placement during anesthesia.[5]

Another rare cause is the ball-valve effect from airway obstruction due to oversecretion in the mechanical ventilated patients who have pulmonary diseases such as intrabronchial hemorrhage, pneumonia, and bronchitis. The obstruction can be bypassed by enlarging airway diameter on inspiration but cannot be bypassed during exhalation when airway diameter is smaller. So the lung becomes overinflated, which can cause a pneumothorax.[6]

As the cause of the pneumothorax in the present case, we first considered pleural damage from central venous cannulation, a bronchial rupture during bronchial blocker placement, and the pleural damage due to surgery itself.

The overall reported incidence of delayed pneumothorax is 0.4 to 0.6% of all central venous access attempts. And pneumothorax is much more common following a subclavian rather than a jugular approach, and generally associated with difficult or multiple approaches.[7] However in the present case, we performed the central venous cannulation successfully at the first attempt by using the anterior approach to the right internal jugular vein. Therefore central venous cannulation cannot be presumed the cause of the pneumothorax.

Mcgillvary et al.[8] has noted that the tip of the blocker is quite rigid and that excoriation of the tracheal mucosa after attempts at blind introduction has been observed. However in the present case, we used a bronchoscope from the beginning to place the blocker in the upper part of the left main bronchus. So it is difficult to say that the placement of the bronchial blocker was the cause of the pneumothorax.

In a study about the causes of a pneumothorax in the dependent lung during thoracic surgeries, Blalock et al.[9] estimated that it is caused by air leakage through some defect in the contralateral respiratory structures or through the mediastinal pleura. However even if there was pleural damage during surgery at the side of the dependent lung, the thoracic pressure at the side of the dependent lung would be higher than the side of the nondependent lung, so the possibility of air inflow would be low. Therefore we think that it could not be the cause of the pneumothorax.

In the present case, when SpO_2 decreased after 2 hours of OLV, we inspected copious bloody secretions were scattered around the trachea and right main bronchus via bronchoscope. Lung auscultation revealed decreased breathing sounds over the right lung fields. Therefore, we first suspected pulmonary atelectasis. So we suctioned the trachea repeatedly, and immediately performed two lung ventilation by manual bagging after removing air from the bronchial cuff. However when we restarted OLV for the surgery, SpO_2 decreased again. So we were able to continue the surgery by alternating one-lung and two-lung ventilation.

Univent® tube we used for OLV had a narrow bronchial blocker lumen for removing secretions and its position in the non-dependent lung may lead to the contamination of the dependent lung by either blood or pus. Besides the bronchoscope cannot easily pass through the tracheal or bronchial lumen like double-lumen endobronchial tube, so it is impossible to remove the secretions in the distal bronchus with the suction port. Therefore, in the present patient the dependent lung may have been contaminated with secretions from the nondependent lung in the process of repetitive removal of air from the bronchial cuff for the adjustment of the blocker's position and for two-lung ventilation. Eventually the accumulation of secretions in the right bronchus may have caused partial obstruction of the airway, and localized barotrauma during continuous mechanical ventilation may have led to tension pneumothorax.

The diagnosis of pneumothorax in the present case was delayed for the following reasons:

1) CVP had not increased as much as would have been expected, perhaps because the contralateral chest was opened.

2) We thought that the cause of the decrease in breath sounds auscultated in the dependent lung was due to atelectasis.

3) The patient had no past history of emphysema or other

bullous disease.

4) We could not clearly inspect the right main bronchus which might be compressed from increased pressure in the pleural space because visualization via bronchoscope was limited due to bloody secretions.

As mentioned above, tension pneumothorax should be suspected when the patient's condition does not improve despite of proper management or resumption of two-lung ventilation. The diagnosis for tension pneumothorax depends on chest X-ray, and bronchoscope is also helpful because it allows for the observation of circumferentially compressed major bronchi.[10] Inspecting the dependent lung is also helpful which may reveal the elevation of the mainstem bronchus or mediastinal herniation into the opposite side hemithorax. When a pneumothorax is strongly suspected and respiratory or circulatory distress are present, anesthesiologist must consider either performing a needle aspiration of the pleural space, or inserting a short intravenous cannula as a pleural space vent.[11]

In conclusion, atelectasis of the dependent lung or ten sion-pneumothorax may occur as a complication when Univent® tube is used for OLV because suction of the secretion can be limited. So anesthegiologist must give all attention to the presenting signs of the pneumothorax and means of treatment.

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