Although torsion of a lobe is not uncommon during lung surgery, torsion of the whole lung during a nonpulmonary procedure is rare. Torsion of the lung is defined as the parenchymal rotation of the whole lung on its bronchovascular pedicle. Rotation occurs with equal frequency in the left and right lungs. Predisposing factors include transection of the inferior pulmonary ligament, pleural space adhesions, pneumothorax, and pleural effusion. Pulmonary torsion is most often a consequence of trauma or a complication of thoracotomy. Oddi et al. and Chan et al. reported cases of torsion after a transthoracic esophageal surgery. We report a third such case of intraoperative torsion of the left lung during an esophagectomy.

Case Report

A 52-yr-old white man (weight, 70 kg; height, 170 cm) with a well-documented benign esophageal stricture was admitted to the hospital for an elective thoracoabdominal esophagectomy. The patient's past medical history was significant for a large hiatal hernia, duodenitis, depression, and factor V deficiency. He had smoked one half a pack of cigarettes per day for 20 yr and reported occasional use of alcohol. He had no significant history of cardiovascular or pulmonary disease. His symptoms included severe progressive dysphagia with the intake of solid foods. His medications were onpramazol and dazepam. On physical examination he was well developed, well nourished, and in no acute distress. Results of his laboratory tests were significant only for a mild elevation of the prothrombin time of 14.7 s. Results of his preoperative chest radiograph and electrocardiogram were normal.

In addition to basic anesthesiologist routine monitors, a catheter was placed in the left radial artery for systemic blood pressure monitoring and in the right internal jugular vein for central venous pressure monitoring. Anesthesia was induced with thiopental, fentanyl, and midazolam. Pancuronium was used for muscle relaxation, and the patient's trachea was intubated with an 8.0-mm single-lumen cuffed endotracheal tube. Anesthesia was maintained with oxygen, nitrous oxide, isoflurane, fentanyl, and pancuronium. The patient was placed in the right lateral decubitus position and a left thoracoabdominal incision was made. Exposure of the thoracic esophagus was complicated by adhesions from chronic pleuritis and mediastinitis and required extensive dissection of the lung from the parietal pleura. In addition, the inferior pulmonary ligament was divided and the lung retracted and reflected anteriorly for adequate exposure. With retraction of the left lung there were multiple episodes of oxygen desaturation to 80-82% that required occasional reexpansion of the compressed lung under direct vision. This intraoperative course was otherwise uncomplicated.

After the operation, the patient was admitted to the postanesthesia care unit. Mechanical ventilation was continued. The patient was hemodymanically stable with appropriate oxygenation, although his chest radiograph revealed a left upper lobe infiltrate. On the first postoperative day, the patient was awake, alert, moving all extremities, and following verbal commands. He was weaned from mechanical ventilation and his trachea was extubated. After extubation the patient was in no respiratory distress with good arterial blood gases and arterial oxygen saturation, but there were no breath sounds over the left hemithorax. The postextubation chest radiograph (fig. 1) revealed complete opacification of the left hemithorax, and chest tube drainage was minimal. It was decided to reintubate the patient for aggressive suctioning and bronchoscopy. Fiberoptic bronchoscopy revealed concentric narrowing of the left mainstem bronchus with edema and erythema. A chest radiograph after bronchoscopy and aggressive suctioning showed no improvement in the lung fields, and the patient was sedated and remained connected to the ventilator.

On the second postoperative day, the patient became acutely hypoxic; the pH of the arterial blood gas was 7.52, the partial pressure of carbon dioxide was 35 mmHg, the partial pressure of oxygen was 58 mmHg, and the oxygen saturation rate was 90% while he was ventilated with a fractional inspired oxygen tension of 1.0, and a blood pressure of 70/40 mmHg. Bloody secretions were suctioned from the tracheal tube. Despite aggressive tracheal suctioning, oxygenation did not improve and the patient was taken emergently to the operating room for reexploration of the left hemithorax. In the operating room, after general anesthesia was induced, the single-lumen endotracheal tube was removed and rigid bronchoscopy was performed. This revealed bleeding and total occlusion of the left main bronchus. A right-sided 4.5-French double-lumen endobronchial tube (Mallinckrodt Medical, St. Louis, MO) was placed. Oxygenation im-

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proved with isolated right lung ventilation. Exploration of the left hemithorax via a left thoracotomy revealed bloody pleural fluid and a distended hemorrhagic lung. Further examination revealed torsion of the left lung, which required three counterclockwise rotations to return the lung to its anatomic position. To determine the viability of the lung, a wedge resection of the left upper lobe was examined. This revealed hemorrhagic necrosis, and a left pneumonectomy was performed.

The patient's postoperative course was complicated by pneumonia, sepsis, acute respiratory distress syndrome, respiratory failure requiring tracheostomy, bronchopleural fistula requiring a muscle flap, and a left empyema. The patient eventually recovered and was discharged home.

Discussion

Torsion of the whole lung during a nonpulmonary procedure occurs only rarely. Pulmonary torsion leads to compromise of the pulmonary and bronchial circulations with eventual hemorrhagic infarction and pulmonary necrosis. Therefore, during surgery, it is imperative that torsion of the lung is prevented by careful reflation of the collapsed lung under direct vision; whenever this condition is considered in the postoperative period, a prompt diagnosis with immediate surgical intervention is essential to preserve lung viability, to decrease complications, and to prevent death.

Whole lung torsion occurs with nonspecific signs and symptoms, and thus the physician must maintain a high index of suspicion to make this diagnosis. Presenting features include deterioration of the patient's condition; cessation of bubbling from the chest tube drains, which may reflect a lack of ventilation to the twisted lung; a clinical picture of shock or sepsis; and hypoxemia. Physical examination may reveal absent or diminished breath sounds over the affected lung. Several modalities may help in early diagnosis and intervention. Felson's method describes many radiographic signs that are usually observed with pulmonary torsion. Classically there is opacification of the twisted pulmonary tissue and a change in the position of an opacified lobe or pulmonary vasculature on serial chest radiographs. Other radiographic findings include lobar air trapping, bronchial cutoff, or distortion. Nonradiographic procedures that may aid in the diagnosis include bronchoscopy, angiography, and thoracentesis. Bronchoscopy is usually nonspecific but can show varying degrees of bronchial obstruction, edema, and increased secretions. Pulmonary arteriography can reveal distortion of the pulmonary vascular tree. Thoracentesis can be negative or the fluid can be bloody or chocolate colored, as described by Moser and Proto. Results of ventilation-perfusion scans are usually nonspecific.

Prevention of torsion requires an understanding of the factors that contribute to rotation of the lung on its bronchovascular pedicle. Intraoperative factors include avoidance of direct injury to the hilar vessels during dissection and inadvertent torsion during retraction. Other mechanisms of torsion can include transection of the inferior pulmonary ligament that anchors the lower lobe, absence of a parenchymal bridge between contiguous lobes, presence of a long and slender hilar pedicle, an airless lobe, and pneumothorax. In the two reported cases, the authors suggested that division of the inferior pulmonary ligament was the contributing factor that made torsion possible. We think that contributing factors in this case were dissection of extensive pulmonary adhesions and transection of the inferior pulmonary ligament.

Pulmonary torsion is a rare complication associated with significant complications and death. The diagnosis should be entertained whenever opacification of a hemithorax appears on chest radiographs after operation. Early surgical intervention is recommended.

References


Intraoperative Diagnosis of Torsion of the Left Lung after Repair of a Disruption of the Descending Thoracic Aorta

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LUNG torsion is an uncommon event, although if unrecognized may be fatal. Torsion of a single lobe¹ or of the entire lung² has been reported after intrathoracic procedures. Torsion may also occur after blunt chest trauma,³ occur as a complication of a pneumonia,⁴ a pneumothorax,⁵ a diaphragmatic hernia,⁶ or occur spontaneously when it involves an accessory lobe⁷ or an intraluminal mass.⁸ The authors describe a case of an intraoperative diagnosis of complete torsion of the left lung and the hemodynamic sequelae of surgical detorsion of the lung.

Case Report

A 38-year-old man was admitted after a motor vehicle accident in which he sustained a left flail chest with fractured ribs, left clavicle, and left scapula. The patient received general anesthesia as part of his original evaluation, during which time the patient’s trachea was intubated without difficulty with a single lumen endotracheal tube. The arterial blood gas immediately after intubation showed a pH of 7.21, PaO₂ of 84, a PaCO₂ of 58, and a base deficit of 5.2. The peak inspiratory pressure was initially 40 cm H₂O but decreased to 25 cm H₂O after placement of bilateral chest tubes. A widened mediastinum was noted on chest radiograph, and after aortography, the patient was scheduled for emergency repair of a descending thoracic aorta disruption during general anesthesia.

The patient was transported to the operating room paralyzed and sedated. His lungs were ventilated on 100% oxygen at a rate of 10 breaths/min and a tidal volume of 1000 ml. His arterial blood gas at this time showed a pH of 7.41, a PaO₂ of 425, a PaCO₂ of 40, and a base deficit of 0.1. During an exchange of the single lumen endotracheal tube for a dual-lumen double lumen endotracheal tube, gastric contents were noted in the patient’s oropharynx. Bronchoscopy was performed, which failed to show evidence of aspiration. An arterial blood gas was obtained, which showed a pH of 7.28, a PaO₂ of 164, a PaCO₂ of 54, and a base deficit of 1.9. In addition to routine monitors, a right radial arterial cannula, a left femoral arterial cannula, and bilateral internal jugular catheters were placed.

The patient was positioned in the right lateral decubitus position. After reconfirmation of proper position of the double lumen endotracheal tube with a fiberoptic bronchoscope, the left lung was deflated. The arterial blood gas showed a pH of 7.29, PaO₂ of 49, PaCO₂ of 56, and a base deficit of 3.9. SpO₂ was in the mid-80% range. A decision was made to repair the aortic disruption without cardiopulmonary bypass. The repair was accomplished with a total aortic cross-clamp time of 22 min, during which time the patient’s SpO₂ decreased to 70–75%. An arterial blood gas at that time showed a pH of 7.24, PaO₂ of 41, PaCO₂ of 59, and a base deficit of 2.7. Two bronchoscopies were performed during the period of desaturation; both examinations revealed patent major conducting Airways with a small amount of sanguinous secretions in the left main bronchus. Several efforts to improve oxygenation were either unsuccessful or resulted in unacceptable encroachment of the lung into the surgeon’s field.

Repair of the thoracic aorta disruption was completed, and the transaortic clamps were released. This was immediately followed by a decrease in SpO₂ to ≤ 40% and a decrease in arterial blood pressure to a mean of 40 mmHg. The patient was treated with reinfusion and ventilation of the left lung, progressive boluses of fluid, phenylephrine, sodium bicarbonate, and epinephrine. Over 15 min, the SpO₂ returned to values > 90%.

After final inspection of the repaired aorta, the left lung was ob-