CASE REPORTS


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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 lobe or of the entire lung has been reported after intrathoracic procedures. Torsion may also occur after blunt chest trauma, occur as a complication of a pneumonia, a pneumothorax, a diaphragmatic hernia, or occur spontaneously when it involves an accessory lobe or an intraluminal mass. 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-yr-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 41-French 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-
served to rapidly reinflate. The chest was closed, and the SpO₂ decreased once again to the mid-80% range with a stable arterial blood pressure. The peak inspiratory pressure, which had previously been 48 cm H₂O, increased to 52 cm H₂O. It was elected to perform bronchoscopy once again to confirm airway patency and suction secretions. On examination of the bronchial lumen of the double lumen endotracheal tube, the left mainstem bronchus appeared to be symmetrically compressed 3 mm past the end of the bronchial lumen. A more experienced bronchoscopist was called to evaluate the airways and confirmed that the lumen of the left mainstem bronchus appeared to be obliterated. The right mainstem bronchus appeared to be normal. The surgeons were asked to re-explore the chest. The ribs were retracted, revealing an apparently normal, inflated, and ventilated left lung. The hilum of the left lung was then examined, and it was noted that the entire lung had undergone a 180° torsion about the left pulmonary bronchus and vascular pedicle. Detorsion of the lung was immediately followed by a precipitous decrease in the SpO₂ to ≈ 50%, a mild decrease in the mean arterial blood pressure, and an increase in heart rate from 110 to 135 beats/min. The patient responded to the judicious administration of ephedrine and manual ventilation of the lungs over a period of 10 min with a return of SpO₂ to > 80%. An arterial blood gas showed a pH of 7.29, PaO₂ of 48, PaCO₂ of 63, and a base deficit of 1.0. Bronchoscopy revealed a small amount of serosanguinous secretions in the left mainstem bronchus with none in the right mainstem bronchus. The oxygen saturation subsequently increased to 80% over the next 20 min with expansion of an apparently normal left lung. The chest was closed, and the patient was turned in the supine position. Over the next 30 min and after changing the double lumen endotracheal tube to a single lumen tube, the oxygen saturation further increased to 93%.

The patient’s trachea remained intubated over the next 10 days, during which time his SpO₂ remained in the low 90% range. His chest radiograph showed patchy infiltrates over the left lung field, considered to be a result of a severe lung contusion. Repeated bronchoscopies noted serosanguinous secretions in the left mainstem bronchus, which gradually decreased over 5 days. The patient was extubated and transferred to the ward from which he was discharged 16 days after his admission.

**Discussion**

Signs and symptoms of intraoperative pulmonary torsion may be subtle or attributable to other processes. In the case described, the patient demonstrated a 4 cm H₂O increase in peak inspiratory pressures with a decrease in the oxygen saturation from the low 90% to the mid-80% range. These findings were attributable to the patient’s history of pulmonary contusion or possible aspiration injury and to recent chest closure. Particularly misleading was the appearance of normal left lung excursion before chest closure. We believe that the lung was torsioned at this time and that the apparently normal excursion of the left lung may have resulted from transmitted movement from the right lung via the mediastinum or perhaps a result of incomplete occlusion of the left mainstem bronchus. This case demonstrates that apparent excursion of the lung within the thorax does not rule out torsion.

On noting an increased inspiratory pressure with a decreased oxygen saturation, we used bronchoscopy to confirm endotracheal tube positioning and to lavage and suction the main bronchi. This examination demonstrated an obliterated left mainstem bronchial lumen, the differential for which includes extrinsic compression of the bronchus, endobronchial mass, or bronchial wall edema. When occlusion of the bronchus is only partial, the diagnosis of torsion may not be as apparent, although bronchoscopy is usually helpful in ruling out other entities, such as aspiration pneumonitis. In the case described, reexploration of the chest ultimately led to the discovery of lung torsion.

A common sequel to detorsion of the lung is spillage of necrotic material from the torsed lung tissue into adjacent lung tissue. Several reports described this complication, and because of this hazard, separation of the lungs via double lumen endotracheal tube is recommended before detorsion of the involved tissue. We used lung separation as a precaution, although noted only limited bronchorrhoea.

Detorsion may lead to profound hemodynamic consequences even in the absence of hemorrhagic bronchorrhoea. In the case described, the oxygen saturation decreased precipitously with marked tachycardia and hypotension. We hypothesize that the principle effect involves the flow into the left atrium of deeply hypoxic and acidic blood released from the pulmonary veins of the torsioned lung. This results in an initial decrease in the oxygen saturation, which, together with acidic metabolites, results in a significant decrease in cardiac output. Further, a decrease in cardiac output may result in a decrease in mixed venous oxygen saturation, perpetuating hypoxemia. Reports of thrombus in the pulmonary vein after detorsion by Schuler and in reports by Hendriks et al and Inoue et al of cerebral infarction after detorsion suggest that showering of emboli from the pulmonary veins is another possible sequel to detorsion, although we found no evidence of this in the described case.

In this case, mobilization of the lung was performed to improve surgical exposure and may have increased the likelihood of torsion. Deflation of the lung and division of the inferior pulmonary ligament have been described by Felson as maneuvers which may predispose to the development of pulmonary torsion.
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We have reported a case of intraoperative diagnosis of whole lung torsion and the hemodynamic sequelae of detorsion. Reports of the intraoperative diagnosis of torsion are rare, whereas the incidence of torsion during intrathoracic procedures may be as high as 0.2%. We conclude that in the diagnosis of torsion is easily missed. We attribute this to the subtlety of the presenting symptoms and advocate a high index of suspicion. When indicated, bronchoscopic examination of the airways may be diagnostic. Awareness of maneuvers that predispose patients to the development of torsion may help predict high-risk cases.

References

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Severe Maternal Hypotension and Fetal Bradycardia after a Combined Spinal Epidural Anesthetic

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INTRATHecal sufentanil alone or combined with bupivacaine is commonly administered via a combined spinal epidural (CSE) technique to provide analgesia for patients in labor. Although side effects of these agents have been reported, there is debate about whether spinal sufentanil used alone or with bupivacaine is associated with clinically significant hypotension. We describe a case of severe maternal hypotension with associated fetal bradycardia after the intrathecal administration of 7.5 μg sufentanil and 2.5 mg bupivacaine.

Case Report

A 32-yr-old, 60-kg, gravida 3, para 2 parturient requested epidural analgesia during active stage I labor. Her past medical history was unremarkable, and she had no complications with two epidural anesthetics with previous births. Informed consent was obtained for a CSE anesthetic. Maternal blood pressure was 135/80 mmHg (recorded in the right upper arm with an appropriately sized oscillometric blood