Thoracic Compartment Syndrome Secondary to a Thoracic Procedure*

A Case Report

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Prolonged open sternotomy is a well-known phenomenon in the pediatric and adult cardiac surgery literature. It is usually an adjuvant in the treatment of a severely compromised heart. We present a case of thoracic compartment syndrome that developed postoperatively from a noncardiac thoracic procedure. Management, diagnosis, and literature review are presented. (CHEST 2003; 124:1164–1168)

Key words: cardiac; chest; decompression; lung; surgery

Abbreviation: PEEP = positive end-expiratory pressure

The compartment syndrome is a well-known phenomenon in the orthopedic and trauma literature. It was first described as an osteofascial phenomenon, when increased tissue pressure reduces capillary perfusion below a level necessary to maintain tissue viability. This concept has been expanded on to include any circumscribed body cavity such as the abdomen and thorax. The thoracic compartment syndrome has been described in the thoracic compartment via delayed sternal closure after prolonged cardiac operations. No reports have been found in the literature describing an event of thoracic compartment syndrome after mediastinoscopy and lobectomy.

CASE REPORT

A 71-year-old white woman presented for mediastinoscopy and bronchoscopy for a newly diagnosed left upper lobe lesion. The patient had undergone a right lower lobectomy in 1992 for stage 1 adenocarcinoma of the lung. A new lung lesion was found on her yearly screening chest radiograph. Her history was significant for hypertension, anxiety, and gastroesophageal reflux disease. She also had a 35-pack-year smoking history but had quit 15 years ago. Her surgical history was significant for the lobectomy and lumbar spinal decompression in March 1999. On hospital admission, she was receiving therapy with diltiazem hydrochloride, 300 mg daily; clonidine hydrochloride, 0.2 mg twice daily; valproic acid, 250 mg daily; prazosin hydrochloride, 2 mg daily; furosemide, 40 mg daily; gemfibrozil, 600 mg daily; and alendronate, 10 mg daily.

The physical examination was significant for a well-healed right thoracotomy scar, a lumbar spinal incision, and an II/IV systolic ejection murmur. The patient had no appreciable adenopathy. A CT scan of the chest showed a 2-cm spiculated left upper lobe nodule. Her chest radiograph in 1998 had been negative for any lesions. Preoperative pulmonary function tests revealed an FVC of 2.15 L (91% predicted), an FEV1 of 1.11 L (59% predicted), and a PaO2 of 47 mm Hg. Her preoperative workup included negative results of a persantine thallium test and two-dimensional echocardiogram.

The patient underwent a mediastinoscopy and bronchoscopy (Fig 1). In the recovery room, she developed immediate respiratory distress secondary to tracheal compression and deviation, with blood coming from the wound. She was taken back to the operating room emergently and was explored via a median sternotomy. A large hematoma was evacuated. She was found to have a bleeding small bronchial vessel. Her estimated blood loss was 2,000 mL, and she received 5 U packed RBCs, 6 U platelets, and 7 L crystalloid. She also had a left upper lobectomy at this time, as the mediastinoscopy biopsy findings were negative (Fig 2). Pathology on the lesion showed non-small cell carcinoma with negative lymph nodes.

Postoperatively, the patient developed a worsening acidosis and hypotension despite the use of vasoactive agents (ie, norepinephrine bitartrate, milrinone lactate, epinephrine, dobutamine hydrochloride, and maximal volume resuscitation). Her cardiac index ranged from 1.3 to 2.1 L/min/m², with pulmonary artery BPs of 44/24 mm Hg and a wedge pressure of 16 to 18 mm Hg. The patient was maximally sedated with fentanyl and was paralyzed with vecuronium. Her chest radiograph showed the endotracheal tube position 3 cm above the carina, and no evidence of pneumothorax or other abnormality (Fig 2). A cardiac workup, including both a transthoracic and transesophageal echocardiogram, revealed no evidence of tamponade or focal wall motion abnormalities. Peripheral Doppler echocardiography examination showed no evidence of deep venous thrombosis. There were no sites of ongoing hemorrhage identified. There was no change in her troponin levels or ECG to indicate acute ischemia.

Arterial blood gas levels obtained with tidal volume of 550 mL, positive end-expiratory pressure (PEEP) of 5 cm H2O, 50% oxygen, and volume control ventilation at 15 breaths/min were as follows: pH, 7.26; PaO2, 135 mm Hg; PaCO2, 37 mm Hg; HCO3, 27 mmol/L; PaCO2, 16 mm Hg. The patient’s peak airway pressure was 58 cm H2O, and her arterial lactate level was 8.0 mEq/L. The ventilator was changed to a pressure control mode for fear of causing bronchial stump blowout because of increasing airway pressures. The next set of arterial blood gas levels, obtained with settings of pressure control at 35 mm, a respiratory rate of 15 breaths/min, a tidal volume of 440 mL, and a fraction of inspired oxygen of 50% oxygen with PEEP set at 3 cm H2O, but auto-PEEP set to 11 cm H2O, were as follows: pH, 7.02; PaCO2, 62 mm Hg; PaO2, 262 mm Hg; PaO2/FiO2 ratio, 444; and PaCO2 increases to 47 mm Hg. The patient’s hemoglobin level was stable at 8.5 g/dL. Her chest computed tomography (CT) scan after surgery showed a 2-cm spiculated left upper lobe lesion and no evidence of empyema or pleural effusion.
93 mm Hg. Her minute ventilation on both modes remained similar. Her static compliance was calculated at 60 cm H$_2$O, and her dynamic compliance was 25 cm H$_2$O. Because of worsening acidosis and hypotension, a mixed venous gas was obtained with a pH of 7.21, a mixed venous oxygen pressure of 33 mm Hg, a mixed venous saturation of 59%, and a base deficit of 7.6 mEq/L. Additional fluid resuscitation for her worsening status was undertaken with 4 U packed RBCs and 4 L crystalloid.

The patient was taken back to the operating room (16 h postoperatively) for abdominal and thoracic exploration. Abdominal exploration was negative, with no signs of compromised viscera. Thoracic exploration showed no signs of cardiac tamponade and no hematoma or compromised tissue. The heart was found to be edematous with global hypofunction, and an artificial dam (Gore-Tex; WL Gore; Flagstaff, AZ) was placed leaving the chest open (Fig 3).
Postoperatively, the patient’s cardiac index improved to 2.7 L/min/m² with pulmonary artery pressures of 50/25 mm Hg and arterial blood gas levels measured at pH 7.46, PaCO₂ of 30 mm Hg, and PaO₂ of 89 mm Hg on the same pressure control settings. The patient was kept heavily sedated and paralyzed for 72 h, and then was taken back to the operating room for closure of her chest wound. The patient recovered, with her course further complicated by atrial fibrillation/flutter that required cardioversion and tracheostomy for prolonged ventilator weaning.

**Discussion**

A true thoracic compartment syndrome is a rarity, even to experienced thoracic surgeons. Our patient experienced hemorrhage, which is a well-known postmediastinoscopy complication. Some bleeding, especially from bronchial vessels, can be managed with urgent mediastinoscopy, but our patient demonstrated a very rapid demise, suggesting major vessel injury, namely, the innominate artery.
was reexplored through a median sternotomy with just this injury at the forefront of our differential diagnosis. Once it was realized that a bronchial vessel was bleeding, it was easily controlled. Attention was then turned to the pathol-
gen in her lung as the mediastinoscopy biopsy specimens had been confirmed as negative.

Rahi et al., in 1975 were the first to recognize the syndrome of “tight mediastinum” after prolonged cardiac operations. They treated the “syndrome” with a sternal traction device after full sternal closure.

Since then, almost 200 reported cases have appeared in the cardiothoracic literature. The pediatric literature reports, an incidence of delayed sternal closure due to the increasing complexity of surgery of 5.6% to almost 30% in the neonate. The survival rate in this group is also much lower than that in the adult population, with reports ranging from 50 to 64%. These figures are attributed to the use of extracardiac conduits and preoperatively dilated hearts with poor lung compliance due to excessive pulmo-
nary blood flow.

In the adult cardiac population, the incidence of de-
layed sternal closure and prolonged open sternotomy has ranged from 1.5 to 2.8% with survival figures ranging from 65 to 72%. In most of the cases described, as well as in ours, a marked diminution of the cardiac index was noted. Rising airway pressures on closure of the chest is the usual marker for the development of this syndrome in the cardiac population. However, the syndrome may not develop for hours and possibly days. The lower cardiac output then leads to increasing acidosis and hypotension, which may be exacerbated by the use of vasoactive agents. The decrease in BP combined with an increased end-
diastolic pressure can result in subendocardial ischemia leading to a cycle of progressive deterioration and death. Our patient had no ECG signs of ischemia and a normal transesophageal echocardiogram, thus a high index of clinical suspicion remains the best diagnostic tool. An additional factor that must be taken into consideration in our patient as well as in the trauma population is that the worsening acidosis may be coming from another organ system or from an undiagnosed, potentially lethal injury for which continued resuscitation is a must, but then the thoracic compartment syndrome becomes a diagnosis of exclusion. It is this reasoning that led to an exploratory celiotomy as well as the thoracotomy in our patient.

Kaplan et al. have described a trauma case in which a patients with thoracic and cardiac injuries was treated with delayed sternal closure. Their patient had multiple ep-
isodes of cardiac massage and decompensated in the operating room on attempts to close the thoracic cage. There are anecdotal incidents that we have experienced with prolonged open sternotomy in the trauma patient, but we have had no survivors to date, and these patients have had massive thoracic as well as cardiac injuries.

In a series of > 6,000 cardiac patients, Furnary et al. identified risk factors associated with death after an open sternotomy. These included the following: (1) use of four or more inotropic drips; (2) perioperative stroke; (3) elevation of serum creatinine level to > 265 μmol/L (3.0 mg/dL); and (4) serious ventricular arrhythmias. The presence of both renal failure and ventricular arrhythmias increases the risk of death to almost 50%.

With a review of the cardiac literature, four major causes of prolonged open sternotomy have been identi-
fied. The number one identified reason was coagulopa-
thy with uncontrolled bleeding. Our patient required treatment with multiple blood products, but not to the degree described in the literature. Mestres et al. have described a patient with a coagulopathy so severe that he was sent back to the ICU with large-bore mediastinal tubes connected to a cell saver unit and mediastinal packing.

Additional predisposing factors include cardiomegaly with or without rhythm disturbances, extra-anatomic de-
vices such as conduits or ventricular assist devices, and finally patients with decreased lung compliance or acute pulmonary edema are at significant risk for a delayed sternal closure. Cases described included patients with preoperative congestive heart failure and pulmonary edema. Our patient had abnormal lung compliance by virtue of her COPD and previous thoracotomy.

In cases in which the chest of the patient cannot be closed due to increased risk of compartment syndrome, various methods of achieving chest closure have been described. Options include the use of synthetic materials vs closure with native skin/flaps or just leaving the chest open and packed. A technique described by Jones et al. advocates the use of stents, in addition to coverage, to prevent trauma to the heart from the open bony segments. We used an artificial patch (Gore-Tex; WL Gore) that was sutured to the sternal skin edges. The average time to closure of the chest ranges from 2 to 5 days, with emphasis on the stabilization of patient hemody-
namics before the attempt at closure. There must be evidence of increased cardiac output, decreased filling pressures, and improved lung function prior to closure. Some authors have advocated aggressive diuresis to achieve this end. However, the diuresis of patients who may actually be intravasally underresuscitated can prove to be difficult and harmful.

Furnary et al. found that mediastinitis was much more common in the delayed sternal closure population when bleeding was the primary indication for the open chest. Also, the reported rates of mediastinitis in the general cardiothoracic population of 0.15 to 5% are consistent with the 4 to 6% incidence reported in the literature on prolonged open sternotomy.

CONCLUSION
Thoracic compartment syndrome is well-known in the cardiac literature but is still developing in the noncardiac thoracic/trauma literature. Keys to the diagnosis and treat-
ment of this syndrome include the early recognition of the increasing airway pressure, failing cardiac output, and worsening acidosis. Treatment should include immediate decompression of the chest and maintenance of the open chest until the patient demonstrates correction of the previous hemodynamic instability.
Successful Endoscopic Nd-YAG Laser Treatment of Endobronchial Endometriosis*

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Catamenial hemoptysis is a rare condition that is associated with the presence of intrapulmonary or endobronchial endometrial tissue. Diagnosis of and therapy for this condition are still a matter of debate. We describe a case of endobronchial endometriosis with catamenial hemoptysis. An endobronchial lesion was diagnosed by spiral CT scan, taken at the onset of the menses, and confirmed with flexible bronchoscopy. The patient was successfully treated with endoscopic Nd-YAG laser therapy with a 1-day in-hospital procedure. We suggest that endoscopic laser treatment should be the first line of therapy for central airway endometriosis, provided that the source of bleeding has been conclusively located and all of the lesions can be reached with the bronchoscope.

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Key words: endometriosis; female genital disease; hemoptysis; laser surgery; lung diseases; respiratory tract diseases

Catamenial hemoptysis is a cyclic pulmonary hemorrhage that is synchronous with female menses. It is a rare clinical entity, associated with the presence of intrapulmonary ectopic endometrial tissue, and related both to distal parenchymal and to central airway lesions. Fewer than 40 cases of catamenial hemoptysis have been reported, although endometriosis related to trachea and/or large bronchi involvement is even rarer, with < 10 proven cases reported in the English literature.1–3

Diagnosis of and therapy for this condition are still a matter of debate. The precise source of bleeding is generally not easy to localize because the chest radiograph, CT scan, and bronchoscopy often show normal findings after menses. Histopathologic confirmation of pulmonary endometriosis is also difficult since both biopsy and resected specimens should be obtained just before the onset of the menses.4 The main criterion for the diagnosis is the finding of periodic hemoptysis that is synchronous with menstruation, and most of the reported cases were diagnosed on the basis of the patient’s clinical history, without a supportive histologic demonstration.5

In this report, we present a case of bronchial endometriosis diagnosed by spiral CT scan with virtual bronchoscopy and cured by endoscopic Nd-YAG laser treatment.

CASE REPORT

A 25-year-old nonsmoking woman presented for evaluation of recurrent hemoptysis in July 2001. She underwent her first uneventful pregnancy, and the infant was delivered by a cesarean section in July 2000. In February 2001, just after the onset of menses, she suddenly expectorated roughly 10 mL red blood. Hemoptysis lasted for 1 day and stopped spontaneously. There were no other symptoms. Similar episodes occurred on the first day of every following menses until the time of evaluation. The volume of blood varied from 5 to 15 mL. The findings of a chest radiograph and flexible fiberoptic bronchoscopy, which were performed elsewhere in June 2001, were normal.

The findings of a physical examination and laboratory study were normal. The patient underwent, after and during menses, high-resolution CT scans of the chest, and a conventional enhanced, single-breathhold, spiral CT scan supplemented with multiplanar reconstructions and virtual bronchoscopy. The CT scan performed after menses did not reveal any bronchopulmonary abnormality. The second examination, obtained at the onset of menses, demonstrated some ground-glass opacifications with a patchy distribution at the level of the left lower lobe that was related to the presence of blood in the alveoli (Fig 1). Furthermore, a small area of bronchial mucosal thickening was observed at the origin of the left upper bronchus by virtual bronchoscopy (Fig 2). These findings were not visible with the first CT scan (Fig 3). A pelvic CT scan and gynecologic examination did not

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REFERENCES

1 Haljamae H, Enger E. Human skeletal muscle energy metabolism during and after complete tourniquet ischemia. Ann Surg 1975; 182:9–14