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Thoracic Compartment Syndrome: A Case Report

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ABSTRACT

Thoracic compartment syndrome has been observed after trauma and after mediastinal and cardiac procedures; however, an adult respiratory distress syndrome (ARDS)-like presentation has not been described as a part of thoracic compartment syndrome. We describe the case of an obese patient who underwent coronary artery bypass (his third such procedure) and hiatal hernia reduction during the same operation, followed by transmyocardial laser revascularization and full chest closure the next day. The patient was hypoxic after chest closure. Two days later, his peak airway pressure increased, and his cardiac and urine outputs decreased. Chest radiography findings suggested ARDS without hemodynamic instability. After we reopened the sternal incisions, the patient's symptoms reversed. Although our patient initially appeared to have ARDS, we believe the organ-volume displacement that occurred during the lengthy dual operation produced a thoracic and abdominal compartment syndrome that responded to decompression of the chest.

INTRODUCTION

Compartment syndromes have been described for several enclosed spaces in the body: the brain in cranial compartment syndrome; the kidney, gut, and liver in abdominal compartment syndrome; and blood vessels, nerves, and muscles in extremity compartment syndrome. Decompressive operations—craniotomy [Kjellberg 1971], laparotomy [McNelis 2002], and fasciotomy [Mubarak 1989]—are the standard of care for compartment syndromes. Thoracic compartment syndrome has been described after trauma and after mediastinal and cardiac procedures [Alexi-Meskishvili 1995; Kaplan 1996]. In such cases, delayed sternal closure has been the treatment modality of choice for providing hemodynamic stability [Riahi 1975]; however, an adult respiratory distress syndrome (ARDS)-like presentation has not been described as a part of thoracic compartment syndrome.

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Compartment syndrome is associated with compartment volume displacement that causes increased pressure and reduced capillary blood flow or venous return, which can compromise organ function. Likewise, ARDS is characterized by high-permeability pulmonary edema (capillary leakage) caused by endothelial cell damage, leading to refractory hypoxemia [Meyrick 1986; Pepe 1986]. We describe the case of a patient with a thoracic compartment–type syndrome resembling ARDS that developed after a lengthy cardiac procedure and a concomitant hiatal hernia reduction and that responded quickly to a decompressive procedure.

CASE REPORT

A 64-year-old obese man (body mass index, 35.6 kg/m^2) was referred to our institution for treatment of coronary artery disease and chest pain. He had undergone 2 previous coronary artery bypass operations, both via median sternotomies. Angiography evaluation performed at our institution revealed severe disease of all 4 previously placed saphenous vein grafts. He also had a very large hiatal hernia that had displaced his abdominal organs into his left chest.

A redo coronary artery bypass operation was performed via a median sternotomy to the posterior descending branch of the right coronary artery, the left anterior descending artery, the second obtuse marginal branch of the circumflex artery, and the diagonal branch artery. The patient's hernia was reduced and repaired during the same operation by slightly extending the sternal incision. The fundus of the stomach had been chronically herniated into the posterior left thoracic cavity. The chest was left open. The next day, the patient underwent transmyocardial laser revascularization, and his sternum was closed. Both operative sessions were uneventful.

Two days later, however, the patient remained hypoxic and could not be weaned from the ventilator. During this time, the patient remained hemodynamically stable. His peak airway pressure increased, and his cardiac output and urine output decreased. The ratio of PaO_2 to the fraction of inspired oxygen (FIO_2) was 48 mm Hg, his oxygen saturation measurements were in the low 80s, and his pulmonary capillary wedge pressure was low, causing us concern that he might have developed ARDS.

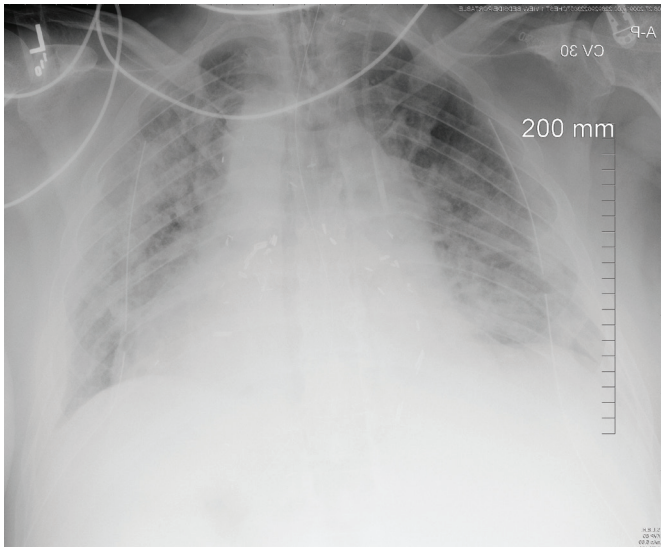

Figure 1. A chest radiogram of the patient's unwired sternum.

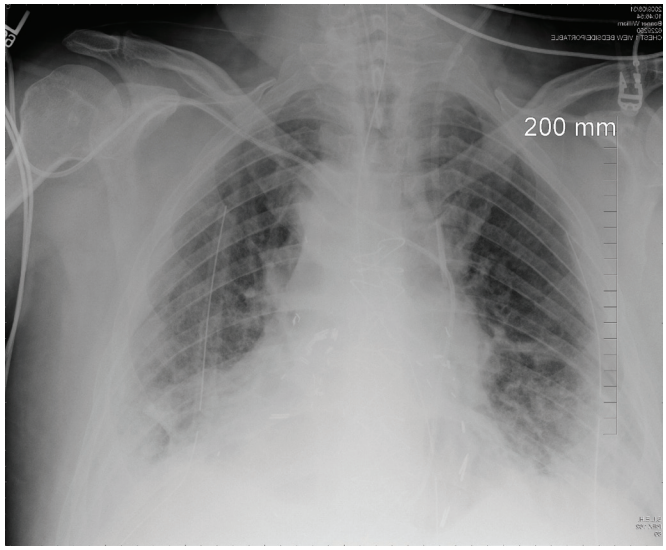

Figure 2. A chest radiogram of the patient's wired sternum obtained after the patient's condition showed clinical and radiologic signs of improvement.

In an attempt to clarify and potentially address the patient's deteriorating clinical status, we explored his condition surgically. Immediately after we opened the sternal incision and its small extension, his oxygen saturation improved to 98%, his peak airway pressure decreased from 38 to 29 cm H_2O, and his urine output increased. All of his bypass grafts were open. The chest was left open with temporary closure of the skin (Figure 1).

After another 5 days, pressure-controlled ventilation with low tidal volumes, diuresis, and other supportive measures had improved the patient's respiratory status enough to allow a second attempt at sternal closure. Once again, however, the patient's oxygen saturation decreased rapidly, to 86%, and his peak airway pressure increased to 36 cm H_2O. When we reopened the sternum, his oxygen saturation increased to 97%. A lung biopsy performed at that time showed nonspecific alveolar edema. The chest was left open, and the same regimen was continued in the intensive care unit. Five days after the second failed attempt, we were able to close the patient's sternum (Figure 2). Thirty-six days after the initial operation, the patient was extubated and discharged home in good condition.

DISCUSSION

Our patient had what we believe to be abdominal, thoracic, or abdominal and thoracic compartment syndrome that presented as ARDS. Compartment syndrome was evident from our patient's compromised venous return and increasing airway pressure after closure of the chest and return of the displaced abdominal organs to their normal location. We believe the patient's concomitant abdominal procedure was a significant contributing factor, along with excess extracellular fluid and fluid retention that were due to his redo sternotomies and lengthy cardiopulmonary bypass.

The resulting increased abdominal pressure compromised the venous return and lowered the cardiac output, resulting in hypoxia akin to ARDS. Because ARDS is known to occur after cardiac surgery and because our patient had clinical features suggestive of ARDS, we suspected it in this patient's case. Although our patient's oxygen saturation improved markedly after we opened the sternal and abdominal incisions, it was uncertain whether the hypoxia was caused by true alveolar capillary membrane damage or from mechanical compression and increased abdominal pressure. In patients with ARDS, damage to the capillary and alveolar epithelia leads to pulmonary edema due to leakage of protein-rich plasma into the interstitial and alveolar spaces [Broaddus 1987]. In our patient, what appeared to be ARDS was probably a compartment-type syndrome marked by increased abdominal pressure caused by abdominal organ displacement.

In instances in which oxygen saturation improves upon opening of the sternum and abdomen, compartment syndrome cannot be excluded. In the case of our patient, relieving the pressure reversed his organ dysfunction. This finding raises the question of whether decompressive procedures like sternotomy should be part of ARDS treatment in order to release the intrathoracic pressure, which, as we observed, dramatically improved oxygenation. Increased thoracic pressure might affect capillary alveolar pressures, which would have an impact on gas exchange.

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