Unusual Presentation and Novel Solution for Hemodynamic Compromise during Thoracic Surgery

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Cardiovascular collapse during or following major thoracic surgery requires prompt diagnosis and immediate treatment. On occasion, the “classic” or expected presentation of a life-threatening complication does not occur, and such a situation tests the clinician’s diagnostic and problem solving abilities. We describe such a case.

CASE REPORT

A 30-yr-old female with the diagnosis of diffuse large cell lymphoma presented for thoracic surgery. Over a 3-yr period, the patient had received multiple standard and salvage radiation and chemotherapy treatments for lymphomatous involvement of the left lung and left chest wall. Multiple recurrences of tumor in the left anterior chest wall led to the development of a large chest wall ulcer. The left lung exhibited upper lobe consolidation and the presence of a mass. The possibility of sepsis or hemorrhage secondary to the necrotic lesion necessitated excision of a large area of the left chest wall. An exploration of the left thorax and the mediastinum was planned to determine the possibility of resection of the lung mass. A combined thoracic surgery and plastic surgery procedure was planned.

Anesthesia was induced with the iv administration of thioental 4 mg/kg and pancuronium 0.08 mg/kg and inhalation of 1.5% isoflurane. The trachea was intubated with a double lumen endotracheal tube. Anesthesia was maintained with 50% N₂O, 50% O₂, and 1.5% isoflurane. Monitoring included a CVP catheter (Hickman Catheter) in the right subclavian vein, right radial arterial cannula, EKG, Foley catheter, temperature probe, esophageal stethoscope, O₂ analyzer in the inspiratory limb, and end tidal CO₂ analyzer.

A left thoracotomy was then performed in the right lateral decubitus position. Single lung ventilation and anesthesia were provided with 1.5–2% isoflurane and 100% O₂ while the left thoracic cavity was explored. A resection was performed removing the entire left lung. A segment of pericardium (an intrapericardial approach for pneumonectomy facilitated access to major vessels and enabled a wider dissection) and a large section of the left anterior chest wall (including ribs 2–5), including a part of the left sternum, were resected. The pericardium was closed by approximation of the free edges. A lattisimus dorsi muscle flap was used to close the chest wall defect. The patient was then placed in the supine position, and a rectus muscle and skin flap (below the umbilicus) was tunneled cephalad and positioned to reinforce the lattisimus flap. Hemodynamic variables had remained stable throughout the surgical procedure (approximately 8 h) with arterial blood pressures ranging from 90/60–120/80 mmHg, CVP readings from 12–15 cm H₂O, and heart rates from 70–90 bpm. Arterial blood gases with continued one-lung ventilation were excellent (Pao₂ 200–300 mmHg, Paco₂ 35–42 mmHg, pH 7.36–7.42). Blood loss was estimated to be approximately 1000 cc with iv replacement including 2 units of packed red blood cells, 50 cc of 25% albumin, and

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4000 cc of crystalloid. Hematocrit readings were 34% and 30% at 4 and 8 h, respectively. Urine output was steady at 100 cc/h, and the body temperature was 35°C at the end of the operation. The change in position from the lateral decubitus to supine was well tolerated hemodynamically.

In an effort to approximate the abdominal skin folds following the rectus flap procedure, the operating room table was flexed. At approximately 40° of flexion (the upper half of the table raised 20° above the horizontal and the lower half raised 20°), the arterial pressure tracing suddenly disappeared, although the EKG trace (lead II) revealed a normal sinus rhythm without ectopy. The bed was taken out of flexion with immediate return of the arterial pressure tracing showing an arterial blood pressure of 100/60 mmHg. The suddenness with which the arterial pressure tracing disappeared and with which it returned was dramatic. (To further clarify, a comparable wave change can be seen when flushing an arterial line. At one point, a distinct wave form is present and with flushing a straight line immediately occurs.) With this presentation, our attention focused on the mechanics of the arterial pressure system, and to the peripheral vasculature to rule out possible obstruction. Thorough examination of the arterial line site and the entire right upper extremity revealed no abnormality, and a second attempt at flexion produced identical results: a sudden loss of the arterial pressure trace. At this time, a cuff pressure on the same arm was attempted, but could not be detected, and no carotid pulse was present.

The table was returned to the horizontal position, but the arterial trace failed to return. A CVP of 16 cm H₂O was recorded and, although the EKG revealed no ectopy, a change in the QRS wave form was obvious (suggestive of an axis shift). A slowing in heart rate from 90 to 60 bpm occurred. The anterior chest wall reconstruction site was then reopened, and the heart was examined.

The myocardium appeared tense with poor contractility in a tight pericardium. Excision of the pericardial sutures enabled cardiac function to resume, thus relieving what was felt to be an iatrogenic constrictive pericardial closure. No obvious pericardial herniation was seen prior to suture removal. Shortly after suture removal, a mild torsion of the great vessels (superior and inferior vena cavae) with a transient episode of hemodynamic instability occurred. A partial closure of the pericardium was felt necessary to prevent a recurrence. No suitable synthetic pericardial closure material was available. It was decided to close the pericardium without tension by approximating the latissimus dorsi muscle flap to the free pericardial edges, and then incorporating the outer edge of the flap to the margins of the chest wall resection (fig. 1). The rectus muscle and skin flap were then placed on top of the latissimus flap (fig. 2). The rectus muscle and skin flap closure required 40° flexion of the operating room table with no further episodes of hemodynamic instability occurring.

Postoperatively, the patient did well. She remained hemodynamically stable at all times and demonstrated normal neurological exams. Her trachea was extubated on the first postoperative day. She was discharged from the ICU on the third postoperative day.

**DISCUSSION**

Cardiovascular collapse during or following major thoracic surgery may be secondary to massive hemorrhage, a rupture of a bronchial stump following lobectomy or pneumonectomy, acute right-sided heart failure following pulmonary resection, or a pericardial herniation of the heart.¹

Several case reports of cardiovascular collapse secondary to pericardial herniation of the heart with twisting of the superior and inferior vena cavae have been described.²⁻⁵ Herniation has occurred with change in po-

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**Fig. 1.** A latissimus dorsi muscle flap is used to cover the chest wall defect and to support the heart as a "neo-pericardium."

**Fig. 2.** The transverse rectus muscle and skin flap were then placed over the latissimus flap to complete the closure.
sitioning of the patient intraoperatively and postoperatively. A change from the lateral decubitus (the surgical position) to the supine position or positioning with the operative side dependent have been associated with herniation episodes. Positive pressure ventilation and application of suction to an empty hemithorax are other presumed causes.

The presentation of sudden cardiovascular collapse with change in patient position (documented on two occasions) and resolution of the problem with return to the original position (documented on one occasion) made us suspicious of pericardial herniation as the etiology. On reexploration, however, no obvious herniation was detected. The suddenness with which the cardiac output dropped suggests a significant positional change of the heart, impeding myocardial contractility and/or a torsion of the great vessels. The heart had been observed to be functioning well following the initial pericardial closure and during closure of the chest wall defect with the latissimus dorsi muscle flap. This period of observation was extensive (approximately 1 h). Upon reopening the chest, a poorly contracting myocardium in a tight pericardial sac was all that could be appreciated. In the haste to remove the pericardial sutures, the exact position of the heart or the presence of torsion of the great vessels was not observed. The fact that an episode of transient hemodynamic compromise secondary to torsion of the great vessels occurred shortly thereafter may be significant. We can only speculate as to what occurred with the change in patient positioning. We feel that the most likely mechanism for hemodynamic compromise involved the heart, enclosed in a tight pericardial sac, assuming a positional change in the chest with flexion of the table, which resulted in a torsion of the great vessels (inferior and superior vena cava) with a reduction in cardiac output. Following repair, the patient was able to tolerate the flexed position for flap and skin closure without incident.

Presentation of this episode in the operating room, rather than postoperatively, allowed for immediate detection and resolution. The use of the second flap (rectus muscles and skin flap) proved instrumental in detection of the problem, since it necessitated the position change (for closure) which otherwise would not have been done. Moreover, the method of eventual pericardial closure using the latissimus flap demonstrates another novel use of these flaps. Pairolero and Arnold described the use of latissimus flaps for chest wall reconstruction, as well as repair of ventricular aneurysms. The use of the flap, in this case, not only covered the heart, but also suspended it from the chest wall, thus preventing herniation and/or torsion. Since the patient had received preoperative radiation therapy and had an infected wound, any pericardial substitute other than autogenous material may have been inappropriate.

REFERENCES