Case Report

Cardiac Herniation with Torsion of Right Pulmonary Artery: Status Post Urgent Left Pneumonectomy

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Citation


Abstract

Cardiac herniation is a rare complication of intrapericardial lung resection, typically occurring within the first twenty-four hours. Considering that this condition carries a high mortality rate despite prompt management, early diagnosis is key. Since clinical manifestations can be variable, the use of imaging modalities coupled with a high index of suspicion is critical to making the correct diagnosis. In this article, a case of acute cardiac herniation post left pneumonectomy diagnosed with the help of an intraoperative transesophageal echocardiography (TEE) is presented.

Keywords: Echocardiography; Transesophageal; Pneumonectomy; Complication; Intraoperative

1. Introduction

Pneumonectomy, defined as the total resection of a lung, is a common procedure for treating patients with end-stage lung cancer or loss of lung ventilatory function from various etiologies. In 1948, the first case of cardiac herniation was reported following lung cancer resection involving either pericardiectomy or pericardiotomy [1]. This rare complication most often occurs within twenty-four hours after surgery or intraoperatively [2]. In the literature, only one case of
late cardiac herniation has been described up to six months after pneumonectomy [3]. Such cases are extremely rare and are usually due to adhesions between the heart and the pericardium. In this article, a case of cardiac herniation post urgency left intrapericardial and extrapleural pneumonectomy diagnosed with the help of an intraoperative transesophageal echocardiography (TEE) is presented. Written consent has been obtained from the patient.

2. Case Report
A 69 years-old male presented to the emergency room of a community hospital in septic shock (Sepsis-3 [4]), with complaints of hemoptysis and shortness of breath. His past medical history was significant for smoking cessation. The initial laboratory work-up revealed leukocytosis (> 30 × 10⁹/L) and elevated serum lactate (18 mg/dL), despite adequate volume resuscitation. A computed tomography (CT) of the chest identified a left upper lobe cavitary lung lesion measuring 6.7 × 6.7 × 10.9 cm, extending to the left mainstem bronchus and carina. Left-sided pleural effusion with associated pneumothorax were also observed. Subsequently, empiric antibiotic therapy was initiated and the patient was transferred to a tertiary center for thoracic surgery assessment. A chest CT repeated twenty-four hours later revealed a large obstructive necrotic cavity in the left lung and a rapidly growing left pulmonary artery pseudoaneurysm. As a result, the patient was scheduled for emergency left intrapericardial and extrapleural pneumonectomy, as well as partial left atrial resection and serratus anterior flap to the left mainstem bronchus and carina. Left-sided pleural effusion with associated pneumothorax were also observed. Subsequently, empiric antibiotic therapy was initiated and the patient was transferred to a tertiary center for thoracic surgery assessment. A chest CT repeated twenty-four hours later revealed a large obstructive necrotic cavity in the left lung and a rapidly growing left pulmonary artery pseudoaneurysm. As a result, the patient was scheduled for emergency left intrapericardial and extrapleural pneumonectomy, as well as partial left atrial resection and serratus anterior flap to the left mainstem bronchus. General anesthesia was induced with ketamine 50 mg, midazolam 5 mg and rocuronium 50 mg. The left lung was isolated using a left sided double lumen tube size 39 French, assisted by a fiberoptic bronchoscope. A triple lumen central line was inserted in the right jugular vein and arterial pressure was monitored through a right femoral arterial catheter.

The patient was then placed in the right lateral decubitus position. Subsequently, the left thoracic cavity was entered in the fifth intercostal space through a large left postero-lateral thoracotomy. A partial extrapleural dissection was needed due to multiple adherences, finally exposing a completely atelectatic left lung, adherent to the pericardium. Due to rupture risk, the left pulmonary artery (PA) was first identified and controlled by a large opening of the pericardium, followed by division at its origin with double stapling. As previously mentioned, the lung lesion was partially extending into the left atrium on the initial CT scan. Thus, the left atrium was clamped with a Satinsky clamp, partially resected and sewed, with good cardiovascular stability. As well, the left mainstem bronchus was found to be necrotic, with bulky inflammatory and malignant lymph nodes. An extensive dissection into its origin was needed to perform a handsewn resection-suture close to the carina. A resection of the pericardium was finally necessary to remove the left lung. A left serratus anterior muscle flap was then harvested and fixed to the bronchial stump through a left sided thoracotomy at the third intercostal space. Given the elevated risk of empyema post-pneumonectomy due to patient’s septic status, it was decided against closing the pericardial defect with a mesh.

A 28-French chest tube was then inserted, and the chest closed. During the operation, blood loss was minimal (200cc), no vaso-active drugs were administered and no desaturations were recorded. Transesophageal echocardiography was then performed and revealed a mildly dilated heart, globally depressed contractility and an ejection fraction of 40%. Subsequently, thoracic epidural catheterization was performed in the lateral decubitus position for pain control. Shortly after extubation in the supine position, the patient desaturated (85%) and required immediate re-intubation. Despite
this measure, the patient continued to desaturate (55%), while the end tidal carbon dioxide (CO2) decreased from 36 mmHg to 7 mmHg. This was followed by hypotension (55/35 mmHg) and bradycardia (20-30 beats per minute), both unresponsive to multiple phenylephrine and ephedrine boluses. Arterial pressure then improved after 1mg of epinephrine (180/100 mmHg), while the saturation remained low (68%). No evidence of myocardial ischemia was identified on electrocardiogram, no crackles were heard at right lung base and the chest tube drained freely. However, prominent jugular venous distension was noticed, along with severe hypoxemia on arterial blood gas analysis.

At that point, through fiberoptic bronchoscopy, good positioning of the endotracheal tube was confirmed, obstructions or secretions were ruled out, and the tracheal mucosa was noticed to be pale. Subsequently, a repeat TEE revealed a hypokinetic right ventricle with worsening distension and contractility, with a severe right ventricular and pulmonary artery systolic pressure gradient, suggesting right ventricular outflow tract (RVOT) obstruction. Many reasons could explain this finding, first of all, pulmonary embolic disease. Specifically, occurring after pneumectomy with no repaired pericardium, it was then believed that was due to torsion of the right pulmonary artery secondary a cardiac herniation through the pericardium defect. Thus, the patient was rapidly positioned in the right lateral decubitus position for immediate repeat thoracotomy. This resulted in an almost instantaneous increase in EtCO2 and blood pressure.

On initial surgical exploration, it was noticed the heart was posteriorly dislodged into the left chest cavity, creating tension and distortion of the right pulmonary artery. The heart was repositioned in the pericardium, leading to immediate saturation improvement. Given the finding of cardiac herniation, it was decided to close the pericardial defect with a patch of bovine pericardium. Thereafter, the patient was positioned in a supine position and maintained a good saturation. He was subsequently transferred to the intensive care unit under mechanical ventilation.

3. Discussion

Since cardiac herniation was first reported in 1948, about sixty other cases were recorded. Most cases occurred after intrapericardial pneumonectomy, followed by extrapleural pneumonectomy, lobectomy, minimally invasive cardiac surgery, severe chest trauma, and congenital cardiac defects [5]. The incidence of cardiac herniation was determined to be independent to pericardial defect size, and without any side predilection [6]. However, clinical presentation largely depends on the site and size of the pericardial defect. Usually, right-sided herniation causes obstruction/torsion of the superior and inferior vena cava, leading to reduced venous return and hypotension. As well, a decrease in filling pressures, a sharp rise in central venous pressure and tachycardia may be observed.

On their part, left sided cardiac herniations usually lead to compression of the myocardium on the pericardial edge. This can result in myocardial ischemia or infarction, hypotension, and arrhythmias such as ventricular fibrillation. In the present case of left sided herniation, torsion of the right pulmonary artery resulted in right ventricular outflow tract obstruction, leading to acute right sided heart failure. Most often, small pericardial defects remain asymptomatic for life, or are only detected on autopsy. They can also result in nonspecific symptoms such as chest pain or palpitations, which tend to be worsened by lying on the side of the defect. Given the rare incidence of cardiac herniation, it’s resulting hemodynamic collapse can be easily misinterpreted for other more common causes such as coronary artery disease, heart failure, shock or cardiac...
tamponade. Cardiac herniation should be suspected in patients known for pericardial defects with clinical signs that cannot be related to other cardiac etiologies. Most reported cases highlighted the importance of prompt diagnosis and management, considering this diagnosis carries a high mortality rate (50%-100%) [2, 5, 7, 8]. In symptomatic and asymptomatic patients, the diagnosis is most often made through routine/urgent chest x-ray or CT-scan. Furthermore, all reported cases with acute symptoms refractory to pharmacologic treatment were diagnosed during urgent repeat thoracotomy.

Only one case was diagnosed by transthoracic echocardiography (TTE) [2], which has three characteristic features pathognomonic for the absence of pericardium [9]. Since clinical manifestations can be variable, the use of imaging modalities coupled with a high index of suspicion among the treating team is critical to making the correct diagnosis. Patients with hemodynamic instability refractory to pharmacologic treatment after surgery should all have immediate re-thoracotomy, being the only effective resuscitative treatment [6]. Measures to improve hemodynamic status prior to surgical treatment include positioning the patient in the lateral decubitus position with the non-surgical side down, avoiding hyperinflation of the remaining lung, and injecting 1-2L of air into the surgical hemithorax [10]. Finally, this present case of acute left cardiac herniation after pneumonectomy presented with worsening right ventricular function on intraoperative TEE. The use of TEE should be considered in the context of acute hemodynamic or ventilatory instability following pneumonectomy involving pericardiectomy or pericardiotomy. This imaging modality could lead to prompt diagnosis and perhaps sufficient time to prevent mortality.

References


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