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Heart Failure due to Tension Hydrothorax after Left Pneumonectomy

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ABSTRACT

Tension hydrothorax is a rare complication of pneumonectomy for pleural mesothelioma and an exceptionally rare cause of heart failure. We describe a patient who had undergone extrapleural pneumonectomy, chemotherapy, and radiation for pleural mesothelioma and who developed heart failure symptoms within months of the completion of treatment. Investigation showed a massive left pleural effusion resulting in tension hydrothorax, mediastinal shift, and evidence of right heart failure with constrictive physiology and low cardiac output. Therapeutic thoracentesis resulted in increase in cardiac output and symptomatic improvement.

CASE PRESENTATION

A 65-year-old male patient with a history of exposure to asbestos was diagnosed with pleural mesothelioma. He underwent a left-sided extrapleural pneumonectomy with en block resection of the diaphragm, left partial pericardial resection, and lymphadenectomy, with a Gore-tex reconstruction of the left diaphragm and part of the pericardium. The patient then underwent chemotherapy followed by radiation to the left chest.

The patient recovered after surgery and resumed normal activities. However, 6 months later, he developed progressive dyspnea, fatigue, and ascites. On physical exam, he was afebrile, with blood pressure 101/86 mmHg, pulse rate 86 per minute, oxygen saturation of 97% on room air, and normal respiratory rate. He had 20 cm of water jugular venous distention. There were diminished heart sounds and absent left lung sounds, moderate ascites, and bilateral pitting edema. The patient could not walk across the room without stopping because of dyspnea and extreme fatigue.

Laboratory findings showed normal blood counts, electrolytes, and liver function tests. Chest radiography showed complete left hemithoracic opacification with right tracheal deviation consistent with a tense hydrothorax (Figure 1). An echocardiogram showed preserved left ventricular systolic function, pseudonormal diastolic filling pattern, a septal bounce, mild right ventricular dilation with reduced systolic function, severe right atrial dilation, severe tricuspid regurgitation, moderate pulmonary hypertension, severe dilation of the inferior vena cava, and trivial pericardial effusion (Figure 2). Paracentesis revealed a transudate with negative cytology. A right-heart catheterization (Table) was consistent with moderately elevated filling pressures and low cardiac index of 1.5 L/min per m2 [Klopfenstein 1994]. Simultaneous ventricular pressure tracings displayed concordance. There were no consistent criteria for constriction or restriction. A cardiac magnetic resonance imaging (MRI) demonstrated a small left ventricle, enlarged right-sided chambers, septal bounce, and no late gadolinium enhancement or myocardial edema. The left hemithorax was filled with fluid with mass effect over the heart, predominantly the left ventricle (Figures 3 and 4).

After 6 L of fluid removal via diuresis, hemodynamics showed no improvement (Table). The differential diagnosis included constrictive pericarditis or tension hydrothorax with compression of the heart with tamponade or constriction physiology. We decided to begin with the least invasive intervention of thoracic drainage.

After removal of 1500 mL of fluid via thoracentesis, hemodynamics improved with drop in right atrial pressure to 8 mmHg and increase in cardiac index to 2.6 L/min per m2 [Klopfenstein 1994]. The patient's symptoms also improved dramatically. A PleurX catheter was placed for continued drainage. Diagnostic thoracoscopy and laparoscopy were negative for malignancy

DISCUSSION

We describe a patient with a history of malignant mesothelioma who underwent extrapleural pneumonectomy with extensive reconstruction of the pericardium and hemidiaphragm, radiation therapy, and chemotherapy and developed slowly

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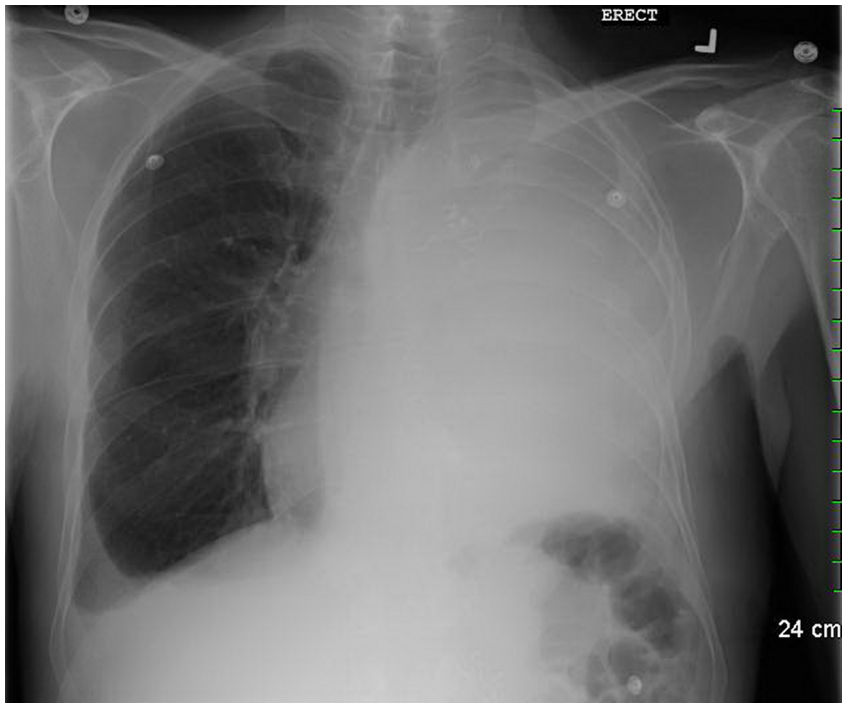

Figure 1. Chest X-ray

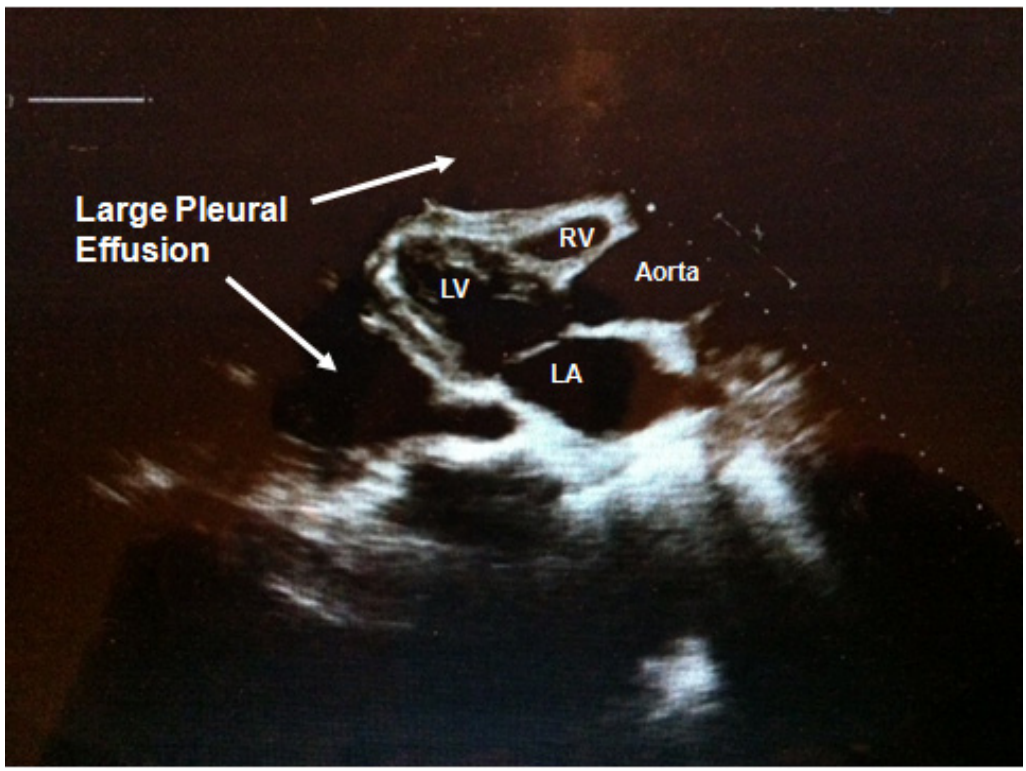

Figure 2. Echocardiogram

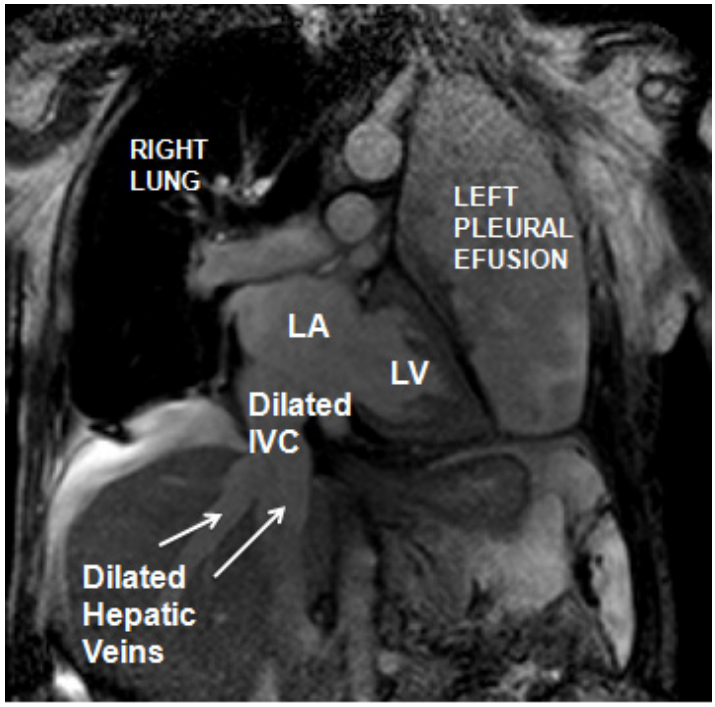

Figure 3. MRI image demonstrating severely dilated right heart and inferior vena cava

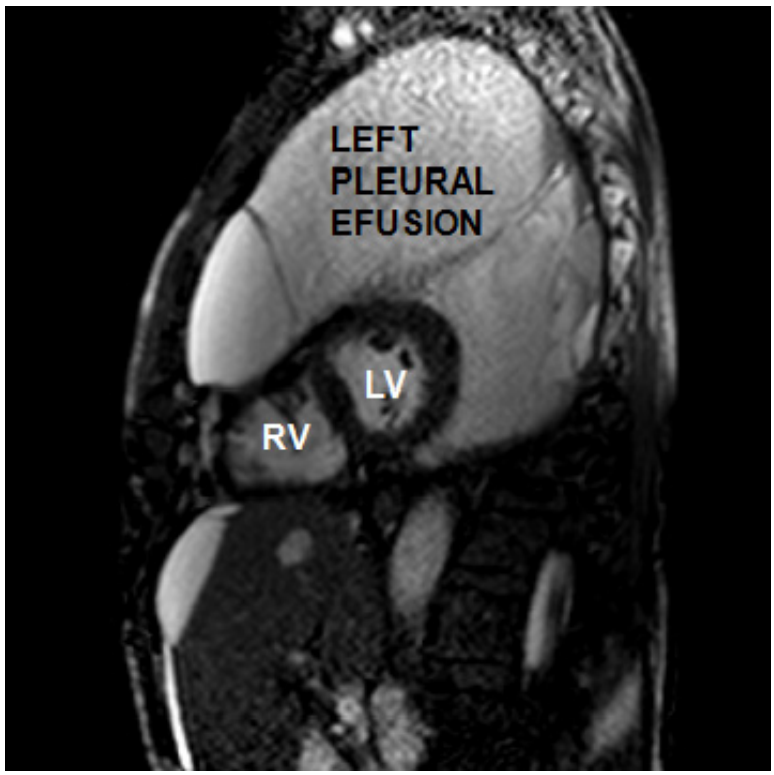

Figure 4. MRI image demonstrating left ventricle compression by a massive hydrothorax

Right-Heart Catheterization Results

	Before Diuresis/before Thoracentesis	After Diuresis/before Thoracentesis	After Serial Thoracentesis
Mean right atrial pressure, mm Hg	15	15	8
Right ventricular pressure, mm Hg	36/18		
Pulmonary artery pressure, mm Hg	36/23 (29)	50/21	44/19
Cardiac output (Fick), L/min	3.25	1.9	4.74
Cardiac index (Fick), L/min per m^2	1.53	1.03	2.58
Systemic vascular resistance, dynes × sec/cm^5		3637	1242
Pulmonary vascular resistance, dynes × sec/cm^5		409	140

progressive signs and symptoms of predominantly right HF, characteristic for constrictive pericarditis, but also had shortness of breath and low cardiac output as in cardiac tamponade. It appeared that tension hydrothorax was the cause.

Tension hydrothorax is a rare condition in which a large pleural effusion causes a shift of the mediastinal structures to the opposite side. Hemodynamic compromise ultimately occurs due to compression of the heart and great vessels. The etiology of the patient's pleural effusion may be a result of a slow accumulation after his surgical procedure, radiation, or possibly even his chemotherapeutic regimen. The patient's hemodynamic evaluation and MRI did not fit into cardiac tamponade or constriction, though the evidence suggested that a constrictive physiology was more likely. The intracardiac pressures were elevated on both sides but differed by more than 5 mmHg. Left and right ventricular pressures were concordant meaning no exaggerated ventricular interdependence. Moderate pulmonary hypertension and ventricular concordance are more typical for restrictive physiology, but there were no signs of infiltration on cardiac MRI.

The complex physiology, with the pericardium on the left side being replaced with synthetic material and increased extracardiac pressure affecting mostly the left side of the heart, likely contributed to the mixed hemodynamic picture and resulted in left ventricular compression, low cardiac output, congestion with dilation of right chambers, and the syndrome of right ventricular failure. Also, the patient's complicated surgery with likely pericardial adhesions may have altered some of the hemodynamic evaluation.

This case illustrates the difficulty of the evaluation of heart failure symptoms and points to a rare but important cause in tension hydrothorax. Utilizing the entire clinical picture pointed to tension hydrothorax being the most likely etiology, which was supported by the improvement in the patient's hemodynamics.

There is experimental evidence of pressure transmission from pleural effusion to the heart, resulting in hemodynamic compromise [Vaska 1992]. In dogs, tamponade confirmed by right ventricular diastolic collapse on echocardiogram was induced by either intrapericardial or intrapleural fluid infusion. Interestingly, the same increase in intrapericardial pressure was much better tolerated when caused by cardiac compression by pleural fluid than when saline was infused directly into the pericardium. The resulting drop in cardiac output was less with intrapleural infusion [Klopfenstein 1994].

Review of the literature shows few prior cases of cardiac tamponade physiology complicating pleural effusions. The reported case closest to our patient (coincidentally from our institution), was a patient with pleural mesothelioma after trimodal therapy, also with tension hydrothorax and a mediastinal shift to the right, with right ventricular diastolic collapse and shortness of breath, but no signs of right-sided failure [Sawar 2006].

Right ventricular failure was present in only one reported case of tamponade due to left-sided pleural effusion, in a patient with sepsis and end-stage renal disease [Kopterides 2006]. In other cases, patients had different reasons for systemic congestion and ascites, such as massive pulmonary embolism [Gamez 2008] or Budd-Chiari syndrome [Venkatesh 1995].

In summary, we describe a case of tension hydrothorax presenting as new-onset heart failure with ascites and shortness of breath. Unlike cases reported before, our patient had a mixed picture with features of constriction, restriction, and tamponade, all of which improved after several sessions of thoracentesis with eventual placement of a PleurX catheter. This case illustrates that the complexity of modern cancer treatments creates unique situations, adding to many faces of heart failure and requiring careful consideration of management strategies to achieve the best possible outcomes.

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