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A M E R I C A N C O L L E G E O F
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A 62-Year-Old Man With Hypotension and a Large Chest Fluid Collection*

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A 62-year-old man was admitted with increased shortness of breath and a cough associated with yellowish sputum production. His symptoms had been progressively worsening over weeks. Pertinent symptoms at presentation included a 10-lb weight loss, dizziness, and low-grade fevers.

The patient was being followed up in pulmonary clinic for a left lung fluid collection shown in Figure 1. A workup had been initiated. Purified protein derivative results and three sputum tests for acid-fast bacilli were negative. Bronchoscopy showed no endobronchial lesions. BAL grew *Streptococcus pneumoniae* and *Stenotrophomonas maltophilia*. Bronchoscopic mycobacterial and fungal culture findings were negative. No atypical cells were seen. He was treated with trimethoprim-sulfamethoxazole and levofloxacin indefinitely and was referred to the thoracic surgeons. However, he was unavailable for follow-up.

His chronic health problems were non-insulin-dependent diabetes mellitus, chronic pancreatitis, hypertension, emphysema, and gastroesophageal reflux disease. His medications included glipizide, albuterol/ipratropium inhaler, lisinopril, and esomeprazole.

Physical Examination

On hospital admission, the patient looked cachectic. He was mildly confused. His vitals showed a



FIGURE 1. Baseline chest CT scan done 2 years prior to admission. A left chest fluid collection surrounded by a thickened pleura is seen. Left pleural enhancement is noted. An air-fluid level defining a bronchopleural fistula is seen.



FIGURE 2. Chest radiograph on presentation showing complete opacification of the left hemithorax with calcifications along the left pleural surface. There is associated mild contralateral mediastinal shift. Patchy right lung airspace disease is noted.

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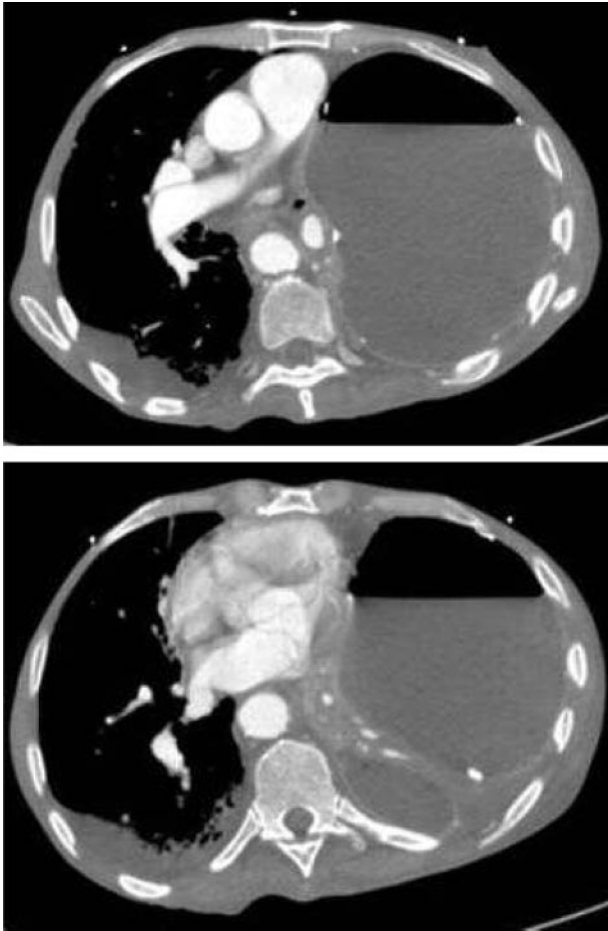


FIGURE 3. *Top*: CT of the chest on presentation showing a significantly enlarged left chest fluid collection. An air-fluid level suggesting hydropneumothorax is seen. There is associated tension phenomenon and contralateral mediastinal shift. Extensive coarse hyperdensities throughout the parietal and visceral pleura of the left lung are seen. In the right lung, a small loculated pleural effusion is seen. *Bottom*: Mass effect on the left ventricle secondary to the fluid collection is noted.

temperature of 38.5°C, a BP of 90/55 mm Hg, a pulse of 110 beats/min (regular), and a respiratory rate of 25 to 28 breaths/min. The oxygen saturation was 95% on 3 L by nasal cannula. Pertinent findings

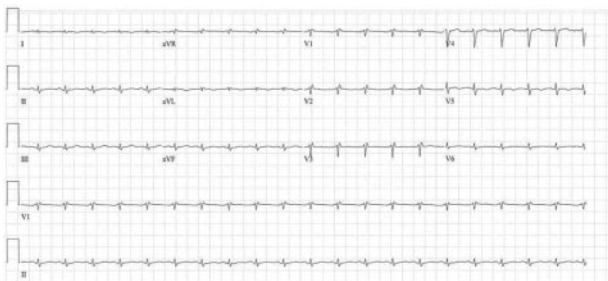


FIGURE 4. ECG on presentation to the ICU showing sinus tachycardia, low-voltage QRS complexes, and incomplete right bundle branch block.

on physical examination included the following: (1) head and neck: poor dentition and no palpable lymph nodes; jugular venous pressure was estimated at 9 to 10 cm above the sternal angle; (2) cardiac: accentuated S2 heart sound over the left upper sternal border; no pericardial friction rub; (3) chest: diffusely decreased air entry and dullness to percussion over the left lung fields; end-expiratory wheezing over the right lung fields; and (4) abdomen: nontender; no hepatosplenomegaly. The extremity and skin examinations were normal, as was the remainder of the neurologic examination.

Laboratory Data

The WBC count was $5.6 \times 10^3/\mu\text{L}$, with the manual differential showing 91% neutrophils. No bands were observed. The hemoglobin was 8.6 g/dL. The platelet count was $223 \times 10^3/\mu\text{L}$. Pertinent serum chemistries included a serum bicarbonate of 26 mmol/L, serum albumin < 1 g/dL, and total protein of 5.0 g/dL. The glucose was 66 mg/dL. Liver function test results were significant for an alkaline phosphatase of 283 U/L. Lipase was normal. The chest radiograph on presentation is shown in Figure 2. The chest CT scan is shown in Figure 3.

Several hours after admission, the patient was found unresponsive. His BP was 60/40 mm Hg. Pulse rate was 130 beats/min, and oxygen saturation was 95% on 6 L by nasal cannula. The patient was intubated, and intensive fluid resuscitation was started. Broad-spectrum antibiotics were administered. A central venous catheter was placed, and the central venous pressure was 23 mm Hg. The ECG is shown in Figure 4.

What is the diagnosis?

What are the next management steps?

Diagnosis: Hemodynamic instability secondary to left ventricular diastolic collapse and cardiac tamponade-like physiology related to an immense encapsulated left chest fluid collection

Management Steps: Immediate drainage of the left-sided fluid collection and cardiopulmonary stabilization followed by definitive surgery

DISCUSSION

Malignancy, uremia, and post-myocardial infarction pericarditis, among other etiologies, may cause large pericardial effusions leading to pericardial tamponade. Cardiac tamponade is usually caused by the acute elevation of intrapericardial pressure. This leads to equalization of cardiac chamber pressures, decrease in stroke volume, compensatory sympathetic activation and, if untreated, hemodynamic collapse.

Cardiac tamponade physiology is not only related to pericardial effusions. Large pleural effusions of various etiologies, in the absence of significant pericardial effusions, have been reported to lead to cardiac tamponade physiology. Multiple reported cases are listed in Table 1. Clinical manifestations of large pleural effusions include hemodynamic instability and pulsus paradoxus. Echocardiographic manifestations include right ventricular or left ventricular diastolic collapse depending on the location of the effusion. Larger effusions and bilateral effusions cause a higher incidence of ventricular collapse and hemodynamic instability. The incidence of these manifestations may be higher in cardiac surgery patients, possibly related to decreased right ventricular reserve. The hemodynamic instability, and echocardiographic findings are usually reversed by drainage of the pleural effusions. It occurs especially in the patients with higher right ventricular systolic pressures.

It has been shown in a canine model that large bilateral pleural effusions lead to an increase in intrapleural pressures, which causes a linear increase in intrapericardial pressures finally leading to right ventricular diastolic collapse. The onset of right ventricular diastolic collapse in both pleural and pericardial effusions occurs at similar intrapericardial pressures. However, compared to pericardial effusions, pleural effusions are associated with a significantly lower incidence of hemodynamic instability and pulsus paradoxus. This may be related to the better preservation of the respiratory variation in intrapericardial pressures in pleural effusions, and to

lower pulmonary vascular pooling due to smaller lung volumes.

Hospital Course

In our patient, the physical examination, central venous pressure, and radiographic findings suggested hemodynamic collapse secondary to tamponade-like physiology related to the left-sided fluid collection. A 32F chest tube was placed into the left fluid collection with ensuing drainage of foul-smelling pus. There was immediate improvement of the patient's hemodynamic status. BP increased to 90 to 100/60 to 65 mm Hg, and the pulse rate decreased to 90 beats/min. Central venous pressure dropped to 13 mm Hg. The patient was stable for several hours. However, hemodynamic status deteriorated secondary to presumed septic shock. Pressors in addition to fluids were administered. Blood cultures grew *Streptococcus viridans*. The echocardiogram showed normal left ventricular function, right ventricular hypertrophy, and severe dilation of the right atrium and right ventricle. No pericardial effusion was noted. After stabilization, the patient was taken to the operating room for resection of the left upper lobe and release of a dense calcific visceral pleura. Pathology showed an extensively inflamed and fibrotic pleura. The left upper lobe had end-stage fibrosis and peribronchial inflammation. A tracheostomy was subsequently done due to difficulty weaning the patient from the mechanical ventilator. Finally, due to persistent fevers, a left lower lobectomy and completion pleurectomy were done with packing of the left chest cavity. He was discharged to a skilled nursing facility after 30 days. The patient's chest radiograph on discharge is shown in Figure 5.

CLINICAL PEARLS

1. *Pericardial effusions are not the only causes of tamponade-like physiology.*
2. *Pleural effusions and lung parenchymal fluid collections lead to an increase in intrapleural pressure that is transmitted to the pericardial space and can cause tamponade-like physiology.*
3. *Compared to pericardial effusions, pleural effusions are associated with a lower incidence of hemodynamic instability and pulsus paradoxus at the same intrapericardial pressures.*
4. *The tamponade-like physiology can be relieved by drainage of these fluid collections.*

Table 1—Pleural Effusions Leading to Cardiac Tamponade Physiology: Cases Reported

Source	Date of Publication	Cause of Pleural Effusion	Radiographic Features	Echocardiogram Findings	Findings Before Drainage of Effusion	Patient Outcome After Drainage of the Effusion
Negus et al	1990	Malignant pleural effusion	Large left pleural effusion (greater than two thirds of the left lung) with tension phenomenon	Not done	BP, 92/68 mm Hg; pulse rate, 108 beats/min; respirations, 26 breaths/min	1,125 mL drained; BP, 140/86 mm Hg; pulse rate, 91 beats/min; respirations, 16 breaths/min
Kisanuki et al	1991	Bronchogenic carcinoma	Large encapsulated pleural effusion in left lower thorax (greater than two thirds of the left lung)	Left ventricular diastolic collapse; no pericardial effusion	BP, 90/60 mm Hg; pulse rate, 90 beats/min	500 mL drained; BP, 120/80 mm Hg; pulse rate, 80/min; left ventricular collapse disappeared
Kaplan et al	1995	Infected hepatic hydrothorax	Large right pleural effusion (greater than two thirds of the right lung)	Right ventricular diastolic collapse; no pericardial effusion	BP, 102/60 mm Hg; pulse rate, 115 beats/min; central venous pressure, 20 mm Hg*	1,500 mL drained; BP, 134/60 mm Hg; pulse rate 109 beats/min; central venous pressure, 1 mm Hg*; right ventricular collapse disappeared
Kaplan et al	1995	Malignant pleural effusion	Large left pleural effusion (greater than two thirds of the left lung)	Left ventricular diastolic collapse; no pericardial effusion	BP, 90/70 mm Hg; pulsus paradoxus, 20 mm Hg	1,100 mL drained; BP, 120/60 mm Hg; pulsus paradoxus resolved; left ventricular collapse disappeared
Alam et al	1999	Hemothorax after cardiac surgery	Large left pleural effusion (greater than one half of the left lung) with blood clots	Right ventricle not well visualized; minimal pericardial effusion	Systolic BP of 90 mm Hg on norepinephrine at 8 to 10 µg/min	Left thoracotomy (2 L drained); norepinephrine drip stopped 2 h afterwards
Alam et al	1999	Post-coronary artery bypass grafting/thick bloody fluid	Moderate bilateral pleural effusions (less than one half of each lung)	Decreased right ventricular function; left ventricular ejection fraction 50%; no pericardial effusion	Cardiac index, 2.1 L/min/m ² on dobutamine at 10 µg/kg/min; mixed venous O ₂ saturation, 40 to 45%	Right thoracostesis: 600 mL of thick bloody fluid; cardiac index, 2.7/min/m ² ; mixed venous O ₂ saturation, 70%; dobutamine stopped 6 h later
Alam et al	1999	After coronary artery bypass grafting	Moderate bilateral pleural effusions (less than one half of each lung)	Not done	Cardiac index, 1.8 L/min/m ² ; pulmonary capillary wedge pressure, 20 to 25 mm Hg on dobutamine at 5 µg/kg/min	Left thoracostesis: 500 mL drained; cardiac index, 2.7 L/min/m ² ; pulmonary capillary wedge pressure, 12 mm Hg; dobutamine stopped 6 h later
Koptides et al	2006	Exudative effusion in patient with end-stage renal disease on dialysis	Large left pleural effusion (greater than one half of the left lung)	Left ventricular diastolic collapse, moderate pulmonary hypertension; no pericardial effusion	Hypotension during dialysis	2 L drained; hemodynamic stability during dialysis was attained
Koptides et al	2006	Hemothorax after thoracentesis of transudative effusion	Large left pleural effusion (greater than one half of the left lung)	Left ventricular diastolic collapse; no pericardial effusion	Hypotension despite aggressive blood product infusion	Hemodynamic stability was attained after left thoracotomy (amount of fluid drained is unknown)

*Central venous pressure correlated with right atrial pressure.

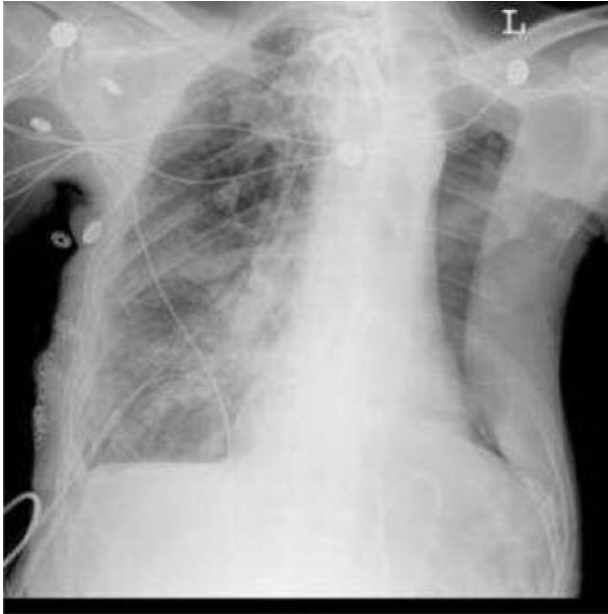


FIGURE 5. Chest radiograph prior to discharge showing evidence of a left thoracotomy with resection of multiple left upper and mid ribs. Right-sided airspace disease is seen, and a right-sided chest tube is noted.

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