A 72-year-old white woman presented with progressive dyspnea, orthopnea, and a weight gain of 10 lb over a period of 2 weeks. The patient’s history was remarkable for coronary heart disease for which she underwent percutaneous transluminal coronary angioplasty of a filiform circumflex artery stenosis 1 year before admission, and for a chronic hepatitis C evolving to cirrhosis with recurrent variceal hemorrhage requiring endoscopic sclerotherapy.

On admission, the patient had tachypnea with a respiratory rate of 30 breaths/min. The heart rate was 100 beats/min with a regular rhythm. The BP was 110/70 mm Hg. There were no murmurs, nor was there a third heart sound. The jugular veins were not distended. A right-sided dullness on thoracic percussion was noted. A chest radiograph revealed a large right-sided pleural effusion but no evident cardiomegaly (Fig 1). Thoracentesis was performed, and 2,000 mL of clear fluid with a protein level of 1 g/dL and a lactate dehydrogenase of 180 U/L were removed. The WBC count was 200/µL. Results of fluid cultures and cytologic studies remained negative. An ultrasound of the abdomen showed minimal perihepatic ascites. Echocardiography revealed a normal left ventricular function. The patient was started on furosemide, 80 mg qd, and spironolactone, 200 mg qd. Over the next week, the pleural effusion and dyspnea recurred. With repeated thoracentesis, an additional 2,000 mL of fluid were aspirated. Diuretics were discontinued because symptomatic arterial hypotension developed. A therapeutic procedure was performed (Fig 2).

The procedure consisted of the placement of a 10-mm diameter transjugular intrahepatic portosystemic shunt (TIPS), as seen on the radiograph in projection on the right upper abdomen (Fig 2). The portosystemic pressure gradient dropped from 20 to 11 mm Hg. The pleural effusion resolved within days (Fig 2), and dyspnea did not recur. Shunt patency was documented by Doppler ultrasound. Unfortunately, portosystemic encephalopathy evolved over the following days, which could be adequately controlled with enteral lactulose administration.

What is the diagnosis?
Figure 1. Admission chest radiograph demonstrating large right-sided pleural effusion in a patient with liver cirrhosis.

Figure 2. Chest radiograph of the same patient after performance of a therapeutic procedure.
**Diagnosis: Hepatic hydrothorax**

Hepatic hydrothorax is defined as pleural effusion in a cirrhotic patient without primary pulmonary or cardiac disease. The frequency of hepatic hydrothorax in cirrhotic patients is reported to be around 5%. The pleural effusion is predominantly right sided (85% of cases) but may be bilateral. Although most often accompanied by significant ascites, it can occur in its absence. Hepatic hydrothorax originates secondary to ascitic fluid movement from the abdominal cavity to the pleural space via defects in the diaphragm. These defects include holes and blebs on the central tendinous portion of the diaphragm. The cyclic negative intrathoracic pressure during breathing and the positive intra-abdominal pressure create an unidirectional flow of ascites across the diaphragm.

Signs and symptoms are primarily those of liver cirrhosis and ascites. Rarely, pulmonary symptoms such as cough and dyspnea predominate. In a patient with right-sided pleural effusion and known liver cirrhosis with ascites, and no concomitant pleural or cardiac disease, a hepatic hydrothorax is most likely. At thoracentesis, protein levels are low (<2.5 g/dL) and similar to what can be found in cirrhotic ascites. The polymorphonuclear leukocyte count should not exceed 500/µL; otherwise, a concomitant spontaneous bacterial empyema exists. Radiolabeled colloid injected into the peritoneal cavity can be used to demonstrate the communication between the peritoneal and pleural space.

The principles of management are the same as for treatment of cirrhotic ascites. In most patients, symptoms are controlled effectively with sodium restriction and diuretics (distal-acting agents plus loop diuretics). Thoracentesis is the most effective method for rapid relief of symptoms. If repeated thoracentesis is required for symptom control, placement of a TIPS is the treatment of choice. In patients with refractory hepatic hydrothorax, relief of symptoms can be achieved in 60 to 80% of patients following TIPS placement. Major complications are shunt occlusion, portosystemic encephalopathy, and postprocedure sepsis.

However, the prognosis for patients with hepatic hydrothorax remains poor despite symptom control following TIPS placement, and liver transplantation as a definitive treatment modality should be considered.

**References**