Hepatic hydrothorax

Case Report 1

A 67-year-old man was admitted with a 1-week history of shortness of breath. He was a heavy smoker with alcohol consumption exceeding 40 units per week. On examination he was apyrexial and had signs of a right-sided pleural effusion. His liver was enlarged 3 cm below the costal margin. There was no ascites or splenomegaly. His jugular venous pressure was normal and there was no pedal oedema. On his initial investigations, he had normal urea and electrolytes and normal liver function tests except for a low albumin level of 28 g/litre.

The chest X-ray confirmed a large right-sided pleural effusion. The pleural fluid had a low protein/albumin content (pleural fluid albumin 8 g/litre), but in view of the history of heavy smoking, large pleural effusion and hepatomegaly, a diagnosis of possible bronchogenic carcinoma with liver metastasis was made. The computed tomography (CT) scan of the chest failed to reveal a cause for the pleural effusion. The pleural fluid cytology, bronchoscopy and echocardiogram were normal. An ultrasound of the abdomen showed an irregular liver with possible single metastasis and minimal free fluid in the abdominal cavity.

He had a chest drain inserted which continued to drain more than 3 litres of fluid daily for 5 days. A thoracoscopy and pleural biopsy were normal. He had two attempts at pleurodesis with talc and tetracycline both of which failed to stop fluid from reaccumulating. He then had a tunnelled pleural drain inserted. This still continued to drain large amounts of fluid for the next 8 days. Therefore a parietal pleurectomy was carried out.

A repeat ultrasound at this stage showed no ascites but an irregular liver suggestive of cirrhosis and a possible malignant lesion within the liver. He had a modestly raised alpha-fetoprotein level of 65 ng/ml (normal range 0–10 ng/ml). He was referred to the authors and a diagnosis of hepatic hydrothorax was made. He was treated with salt restriction and spironolactone. He responded well to treatment and stopped reaccumulating fluid.

A biphasic CT of the abdomen and hepatic angiography confirmed the diagnosis of multifocal hepatoma. His liver biopsy was consistent with cirrhosis secondary to alcohol.

Case Report 2

A 47-year-old woman was admitted with a history of shortness of breath for a couple of days. There was no history of chest pain, cough or fever. On examination she had signs of right-sided pleural effusion. There were no signs of chronic liver disease and her abdominal examination was normal. Her liver function tests were mildly abnormal with an albumin level of 30 g/litre, aspartate aminotransferase level of 52 IU/ml and bilirubin level of 22 μmol/litre. Her chest X-ray confirmed a large right-sided pleural effusion. Her echocardiogram was normal and the abdominal ultrasound showed a small amount of ascites mainly in the pelvis with non-homogenous liver, borderline enlarged spleen and bilateral pleural effusions.

She also had a cystic structure in the right iliac fossa and therefore she had a transvaginal ultrasound done at the same time. This showed a 3 cm cystic mass with echoes within it. The radiologist suggested the possibility of an ovarian tumour with Meig's syndrome. Her CA125 (cancer antigen 125) was marginally raised and her pleural fluid did not show any abnormal cells. The pleural fluid protein was 24 g/litre (albumin 16 g/litre) and her pleural biopsy was normal.

Her ovarian cyst on subsequent discussion and computed tomography scan was confirmed to be benign. She was then referred to the authors and was diagnosed to have hepatic hydrothorax.

On further enquiries, she gave history of a blood transfusion 20 years ago and investigations confirmed her to have cirrhosis from chronic hepatitis C virus infection. She was treated with salt restriction and diuretics. Her pleural effusion disappeared following this treatment.

Introduction

The presentation of chronic liver disease is varied. Ascites is a common complication of cirrhotic liver disease and occurs with a pleural effusion in 5–10% of cases (Cardenas et al, 2004). On rare occasions, pleural effusion is the presenting feature of liver disease with little or no ascites. This presentation can cause diagnostic problems and lead to unnecessary investigations and treatment.

Discussion

The large pleural effusion in a cirrhotic patient, in the absence of primary pulmonary or cardiac disease, has been termed a hepatic hydrothorax (Cardenas et al, 2004). The fluid in the peritoneal cavity is drawn by negative pressure into the chest across defects in the diaphragm. These defects are usually very small and caused by anatomical thinning of the tendinous portion of the diaphragm. The dynamics of respiration enhance the movement of ascites into the pleural cavity. The negative intrathoracic pressure together with the positive intra-abdominal pressure promotes flow of fluid selectively from peritoneal to pleural cavity (Lieberman et al, 1966). This may result in a large pleural effusion being created in the absence of clinically detectable ascites as seen in these cases.

The traditional concept of transudate and exudate is not useful in patients with liver disease. However, the serum ascites albumin gradient (SAAG) calculated by subtracting ascitic fluid albumin from serum albumin is useful in determining the presence of portal hypertension. A SAAG above 11 g/litre indicates portal hypertension with 97% accuracy (Saravanan and Cramp, 2002). In both case reports the SAAG was more than 11 g/litre which could have provided the clue for the correct diagnosis purely on the basis of pleural fluid analysis.

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The treatment of hepatic hydrothorax is similar to that of ascites, often responding well to dietary sodium restriction and diuretics as seen in these two cases. Chest drain insertion and chemical pleurodesis are usually ineffective (Borchardt et al, 2003). In resistant cases, transjugular intrahepatic porto-systemic shunt (TIPSS) is an effective treatment (Siegerstetter et al, 2001). Liver transplantation should be considered in selected cases (Cardenas et al, 2004).

Conclusions

Cirrhosis of the liver should be considered in the differential diagnosis of low protein pleural effusion, even if there is minimal or no ascites. Intercostal chest drainaige and chemical pleurodesis are usually ineffective in the treatment of hepatic hydrothorax. TIPSS should be considered in resistant cases. **BJHM**

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