Lesson of the week

Treating hepatic hydrothorax

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When a patient presents with a large unilateral symptomatic pleural effusion many doctors will feel tempted to insert a chest drain. We caution against placement of a chest drain as a therapeutic reflex, particularly if the diagnosis is hepatic hydrothorax.

Case reports

Case 1
A 70 year old man was admitted to our department for shortness of breath. He was known to have cryptogenic liver cirrhosis. About two years before this admission he had developed ascites, and treatment with salt restriction, spironolactone, and frusemide (furosemide) was started. One year before admission his right kidney had been removed because of renal cancer. Subsequently he began to have recurrent right pleural effusion. Before he was admitted, weekly thoracenteses had become necessary as he continued to be short of breath.

On admission the patient seemed well except for mild tachypnea. We noted dullness to percussion above the right lung. The abdomen was mildly distended, but ascites was not evident. Jugular venous pressure was not raised, and the patient had no peripheral oedema. Blood count (except for platelets 65,000/mm$^3$) and prothrombin time were normal; bilirubin was 30.8 µmol/l, and protein 71 g/l. The table shows additional results. Chest radiography confirmed a large right pleural effusion; abdominal ultrasonography showed only a small amount of ascitic fluid. Thoracentesis showed a transudate with protein 10 g/l and pH 7.49.

A chest drain was inserted and 2790 ml of fluid was drained. Subsequently the daily volume of drained fluid was 1500-2000 ml, and this volume remained unchanged. After one week an attempt at pleurodesis, using 300 mg of minocycline, failed. Thereafter the patient was treated with continuous positive airways pressure of 7.5 cm H$_2$O for several hours several times a day. Drainage was stopped during periods of continuous positive airways pressure, but the total daily volume of drained fluid remained unchanged. We tried pleurodesis again. This time continuous positive airways pressure was applied before the instillation of minocycline, continued while the drain was clamped for two hours, and stopped two hours after declamping the drain. This second attempt also failed.

The patient became increasingly weak. Laboratory results were compatible with loss of fluid and protein (see table). Staphylococcus aureus, Pseudomonas aeruginosa, and Enterococcus faecalis grew in a culture from pleural fluid, and the pH of the fluid dropped to 7.20. The patient had no fever but developed mild leukocytosis. He was given pipercillin with tazobactam and vancomycin. Finally he was transferred to a hospital with facilities for transjugular intrahepatic portosystemic shunting and thoracic surgical intervention. Several days after his transfer he died from septic shock, without having been treated with either of these procedures. An autopsy was not performed.

Case 2
A 65 year old man with cirrhosis of the liver (Child's disease class C) due to hepatitis B presented with a recurrent symptomatic hepatic hydrothorax. Therapeutic thoracentesis on admission caused a moderate pneumothorax. No chest drain was inserted. The lung was re-expanded by simple aspiration, using an 18 gauge catheter. Transjugular intrahepatic portosystemic shunting was performed, and the hydrothorax did not recur. Signs of encephalopathy shortly after the procedure responded to treatment with lactulose. Some months later the patient had a liver transplant.

Discussion

One patient with hepatic hydrothorax died after the insertion of a chest drain, but a second case illustrates a more successful approach. The question is whether the death of the first patient resulted from complications of an established medical procedure or from the performance of a procedure that was not justified.

Hepatic hydrothorax is defined as pleural effusion in a patient with cirrhosis of the liver in the absence of any other explanation for the accumulation of pleural fluid.¹ No established standard exists for the required diagnostic procedures. Hepatic hydrothorax occurs in about 5% of patients with cirrhosis of the liver, depending on the criteria applied.² The most important cause seems to be transfer of fluid from the peritoneal cavity through small defects in the diaphragm.³ The effusion is generally unilateral and usually right sided, and it is not necessarily accompanied by clinically evident ascites.¹ Laboratory features of the fluid are similar to those of ascites. In addition, spontaneous bacterial empyema similar to spontaneous bacterial peritonitis may occur.⁴ Medical treatment consists of restricting sodium and giving diuretics. If medical treatment fails and therapeutic thoracenteses are needed repeatedly, three options are available for treatment: pleurodesis through a chest drain, thoracoscopy with attempt of diaphragmatic repair followed by talc poudrage, and transjugular intrahepatic portosystemic shunting.

Problems associated with pleurodesis through a chest drain

Inserting a chest drain, with subsequent chemical pleurodesis, is still considered a possible therapeutic option. If the pleural effusion remains large, pleurodesis may be attempted. We caution against performing this procedure if the pleural effusion is not due to liver cirrhosis.

Results of blood tests in a patient with hepatic hydrothorax and a chest drain

<table>
<thead>
<tr>
<th>Variable</th>
<th>On admission</th>
<th>Day 4</th>
<th>Day 12</th>
<th>Day 20*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urea (mmol/l)</td>
<td>7.8</td>
<td>14.3</td>
<td>21.0</td>
<td>29.3</td>
</tr>
<tr>
<td>Creatinine (µmol/l)</td>
<td>88.4</td>
<td>97.2</td>
<td>124.0</td>
<td>141.0</td>
</tr>
<tr>
<td>Sodium (mmol/l)</td>
<td>137.0</td>
<td>133.0</td>
<td>119.0</td>
<td>114.0</td>
</tr>
<tr>
<td>Albumin (g/l)</td>
<td>28.0</td>
<td>24.0</td>
<td>23.0</td>
<td>18.0</td>
</tr>
</tbody>
</table>

*The patient was transferred to a hospital with facilities for transjugular hepatic portosystemic shunting.
strategy, although its limitations are recognised.\(^3\) Successful pleurodesis through a chest drain has been described in some patients with hepatic hydrothorax.\(^4\) However, this approach often fails, presumably because of insufficient apposition of the pleural surfaces as a result of rapid accumulation of pleural fluid.\(^5\) Two patients with hepatic hydrothorax died after a chest drain had been inserted.\(^6\) Prolonged drainage through a chest drain causes massive depletion of electrolytes and protein, and renal failure and impaired immunological function are common sequelae. Iatrogenic infection of the pleural space may further complicate treatment, as was the case in case 1. Using continuous positive airways pressure to lower the peritoneopleural pressure gradient is theoretically appealing. Drainage stopped completely in our patient during application of continuous positive airways pressure, but the total volume of fluid drained daily remained unchanged. A widely cited abstract reports successful pleurodesis by using tetracycline and continuous positive airways pressure in six patients with hepatic hydrothorax, although one of the patients died from an infection of the pleural space.\(^10\) No other publication confirms these data. Pleurodesis with continuous positive airways pressure was not successful in our patient. Two recent case reports describe a reduction in the volume of drained fluid after octreotide was given.\(^11\) Drainage through a chest tube in conjunction with continuous positive airways pressure or octreotide must be considered experimentally at this point.

Two case series describe the outcome of thoracoscopy with closure of the diaphragmatic defect if feasible and subsequent talc poudrage in altogether 26 patients with hepatic hydrothorax.\(^11\)\(^\text{11}\) The procedure was successful in 40% and 75% of the patients. Success rates were higher if the diaphragmatic defect could be identified and lower if the only intervention was insufflation of talc. Drainage through a chest tube was continued on average for nearly two weeks after thoracoscopy. The results of transjugular intrahepatic portosystemic shunting for hepatic hydrothorax have been reported in four case series including altogether 81 patients.\(^12\)\(^\text{18}\) The procedure was effective in relieving symptomatic hydrothorax in 60-80% of the patients. Its main complication is the development of hepatic encephalopathy, but encephalopathy that is resistant to medical treatment and requires occlusion of the shunt is rare. Although insufficiency of the shunt occurs often during follow up, it responds to revision in most cases.

Available data and physiological considerations present a strong argument against placing a chest drain in patients with hepatic hydrothorax. The procedure is life threatening and largely ineffective. Case 2 shows that even a hydropneumothorax does not necessarily require tube drainage and may be amenable to simple aspiration.\(^19\) If maximal medical treatment fails, transjugular intrahepatic portosystemic shunting seems to be the procedure of choice for symptomatic hepatic hydrothorax. Since hepatic hydrothorax usually indicates advanced cirrhosis of the liver, liver transplantation should be considered.

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