Hydrothorax in continuous ambulatory peritoneal dialysis: therapeutic implications of Tc-99m MAA peritoneal scintigraphy

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Key words: continuous ambulatory peritoneal dialysis (CAPD); end-stage renal disease (ESRD); hydrothorax; Tc-99m macroaggregated human albumin (MAA) peritoneal scintigraphy

Introduction

Continuous ambulatory peritoneal dialysis (CAPD) is an established, effective renal replacement therapy for patients with end-stage renal disease (ESRD). There are many problems inherent in CAPD therapy, including hydrothorax formation. Many techniques have been developed to overcome these complications. However, so far, there is no consensus concerning the pathogenesis and most effective therapeutic approach to the CAPD-related hydrothorax. Although hydrothorax is relatively uncommon, it may be so serious as to result in respiratory distress, necessitating discontinuation of peritoneal dialysis temporarily or permanently. We describe four patients with CAPD-related hydrothorax, which illustrate the complexity of the problem. We also emphasize the usefulness of peritoneal scintigraphy.

Cases

Case 1

A 24-year-old man with ESRD secondary to lupus nephritis had received maintenance haemodialysis therapy since June 1988. Because vascular access could not be established, a Tenckhoff catheter was inserted on January 30, 1991. Unfortunately, dyspnea and tachypnea developed in June, 1991. On admission to hospital, physical examination revealed regular heart beats without gallop. Breathing sounds were attenuated over the lower half of the left chest. The chest X-ray confirmed a massive left-sided pleural effusion (Figure 1a). The echocardiogram revealed concentric left ventricular hypertrophy with adequate global performance, and no pericardial effusion. The abdominal sonogram showed a normal liver echo pattern without evidence of liver cirrhosis. Serum C3 level was 66 mg/dl (normal range 50–120 mg/dl), C4 29 mg/dl (normal range 15–45 mg/dl) and ANA 1:40 (–). Pleural fluid was transudative. Bacterial culture, mycobacterial culture and the cytology of pleural fluid were all negative. Continuous ambulatory peritoneal dialysis (CAPD) was discontinued temporarily and the patient was switched to haemodialysis therapy on June 18, 1991. Thoracentesis was performed. Following splenic injury with subcapsular haematoma, haemoperitoneum developed on June 20, 1991. The pleural aspirate was serosanguinous 3 days later. As conservative management had failed, thoracoabdominal surgery was performed. Following splenic injury with subcapsular haematoma, haemoperitoneum developed on June 20, 1991. The pleural aspirate was serosanguinous 3 days later. As conservative management had failed, thoracoabdominal surgery with splenectomy and tala abrasion pleurodesis were performed. However, no definite defects over the diaphragm could be found during the procedure. CAPD therapy was resumed with small exchange volumes. Finally, maintenance CAPD therapy could be resumed. The follow-up chest X-ray just revealed pleural reactive changes at the left base.

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March 1992. At his request, he came to our institution to start CAPD therapy. On April 8, 1992, a Tenckhoff catheter was implanted and CAPD commenced. On April 16, 1992, the patient felt tight chested with mild dyspnea following infusion of 1500 ml of dialysate (1.5% Dianeal). On physical examination, blood pressure was 170/115 mmHg, pulse rate 85 beats/min and respiratory rate 24/min. The heart beats were regular without gallop, and the breath sounds were diminished with dullness to percussion over the lower third of the right hemithorax. The chest X-ray confirmed a moderate amount of right pleural effusion (Figure 2a). The echocardiogram revealed septal hypertrophy with adequate left ventricular systolic performance and normal left ventricular size. No pericardial effusion was noted. Both HBsAg and anti-HCV antibodies were negative. The abdominal sonogram was negative. Thoracocentesis showed a clear, pale-yellow transudate. Bacterial culture, mycobacterial culture and the
cytology of pleural effusion were all negative. The results of simultaneously drawn serum, pleural fluid and peritoneal effluent are shown in Table 1. On April 22, 1992, Tc-99m MAA peritoneal scintigraphy revealed the appearance of radioactivity in the right hemithorax 30 min after intraperitoneal injection and suggested a PPC (Figure 2b). The patient’s dyspnea worsened over the next 2 days, and the pleural effusion on chest X-ray was even larger. The symptoms were markedly relieved following therapeutic thoracentesis. On April 24, 1992, pleurodesis with oxytetracycline (25 mg/kg) was performed. CAPD therapy was discontinued and he was switched to haemodialysis therapy three times a week. On May 22, 1992, we reinstituted CAPD therapy in Fowler’s position with only 1000 ml of 1.5% Dianeal solution for each exchange. On the next day, however, the dyspnea recurred. The chest X-ray film disclosed reaccumulation of right pleural effusion. He was switched to maintenance haemodialysis therapy and is awaiting renal transplantation.

Case 3

A 30-year-old woman had ESRD due to IgA nephropathy. Eleven months after maintenance CAPD therapy, she was noted to have rhinorrhea, nasal stuffiness, prolonged dry cough, right side chest pain and progressive dyspnea. Physical examination disclosed a blood pressure of 140/80 mmHg, a pulse rate of 80 beats/min and a respiration rate of 20/min. Dullness to percussion and diminutive breath sounds over the lower third of the right thorax were also found. The chest X-ray confirmed a right pleural effusion (Figure 3a). Her liver function, abdominal sonogram and echocardiogram were all normal. The pleural fluid was a cloudy transudate; see Table 1. Bacterial culture, mycobacterial culture and the cytology of pleural effusion were all negative. On November 7, 1995, Tc-99m MAA peritoneal scintigraphy showed accumulation of radiotracer in the peritoneal cavity, and only a hot area in the middle of the right lower chest area 2 h later (Figure 3b). This finding suggested the existence of PPC. She was switched to haemodialysis therapy temporarily. Her chest discomfort improved gradually. Four weeks later, CAPD therapy was resumed with satisfactory results.

Case 4

A 38-year-old male developed ESRD due to chronic glomerulonephritis and was started on emergency haemodialysis therapy for severe metabolic acidosis and pneumonia complicated by respiratory failure in February, 1997. At his request, he was switched to CAPD therapy and a Tenckhoff catheter was implanted on March 3, 1997. He complained of mild dyspnea and chest tightness following the induction of 1500 ml dialysate (1.5% Dianeal) on March 14, 1997. Physical examination revealed signs of a large right pleural effusion. The chest X-ray disclosed the opacification of the lower half of the right hemithorax and infiltration over the left middle lung field (Figure 4a). The echocardiogram revealed only minimal pericardial effusion. The pleural fluid was a straw-coloured transudate. The results of simultaneously drawn serum, pleural fluid and peritoneal effluent are shown in Table 1. Cultures for bacteria, mycobacteria and the cytology of pleural fluid were all negative. On March 24, 1997, Tc-99m MAA peritoneal scintigraphy showed diffusely increased radioactivity in the right hemithorax 15 min after injection of peritoneal radiotracer (Figure 4b) and suggested a PPC. Dyspnea was markedly relieved following therapeutic thoracentesis. On March 28, 1997, pleurodesis with oxytetracycline (25 mg/kg) was performed. CAPD therapy was discontinued temporarily and the patient was switched to haemodialysis therapy. On May 4, 1997, prior to reinstitution of peritoneal dialysis, his chest X-ray revealed no pleural effusion. However, the dyspnea recurred immediately after infusion of 1000 ml of dialysate (1.5% Dianeal). The chest X-ray disclosed reaccumulation of a right pleural effusion. He was switched to maintenance haemodialysis therapy.

The clinical characteristics, method of diagnosis, treatment and outcome for these four cases are summarized in Table 2.

Discussion

CAPD-related hydrothorax indicates that hydrothorax is derived from the peritoneal dialysate and characterized by a transudate with a relatively high glucose level (i.e. intermediate between that of the dialysate and the serum), lower protein content (<1 g/dl) and lower

<table>
<thead>
<tr>
<th>Case</th>
<th>Glucose (mg/dl)</th>
<th>Total protein (mg/dl)</th>
<th>LDH (U/L)</th>
<th>Cell count (L/N)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PF</td>
<td>Serum</td>
<td>PE</td>
<td>PF</td>
</tr>
<tr>
<td>1</td>
<td>324</td>
<td>87</td>
<td>910</td>
<td>93.4</td>
</tr>
<tr>
<td>2</td>
<td>183</td>
<td>118</td>
<td>608</td>
<td>128.0</td>
</tr>
<tr>
<td>3</td>
<td>123</td>
<td>121</td>
<td>386</td>
<td>798</td>
</tr>
<tr>
<td>4</td>
<td>312</td>
<td>181</td>
<td>760</td>
<td>696</td>
</tr>
</tbody>
</table>

PF, pleural fluid; PE, peritoneal effluent.
lactate dehydrogenase (LDH) level of pleural fluid than that of simultaneously drawn serum [1,2]. It is a well known but uncommon complication of CAPD therapy. Its prevalence rate varies with the patient population studied and the criteria applied. In a study of 3195 patients undergoing CAPD therapy from 161 centres, only 50 patients (1.6%) developed acute massive hydrothorax due to PCC at some time (1 day to 8 years) during the study period (1–104 months) [3].

Fig. 3. Case 3. (a) Chest X-ray confirmed the right pleural effusion. (b) Tc-99m peritoneal scintigraphy showed accumulation of radiotracer in the peritoneal cavity, and only a hot area (arrowhead) in the middle of the right lower chest 2 h later.

Fig. 4. Case 4. (a) Chest X-ray film disclosed the opacification of the lower half of the right hemithorax and infiltration over the left lung. (b) Tc-99m peritoneal scintigraphy revealed diffusely increased radioactivity (arrowhead) in the right hemithorax 40 min after intraperitoneal injection.
However, Chow et al. had reported that ~10% of patients in CAPD therapy will develop a pleural effusion secondary to the movement of dialysate from the peritoneal cavity through the diaphragm into the pleural cavity [1]. Moreover, there may be a higher prevalence of asymptomatic hydrothorax only detected by routine check-ups and resolving spontaneously [4]. From February 1990 to July 1998, a total of 127 uraemic cases accepted CAPD therapy in our hospital. Four cases (3.2%) were found to have CAPD-related massive hydrothorax. The complication does not appear to be limited to any specific aetiology of ESRD [1,3]. There was probably no apparent predisposition due to sex or age, although it has been reported predominantly in females [3,5]. Its occurrence preferentially on the right side (88%) has been reported in the literature [3].

The exact mechanism is still somewhat controversial. Various mechanisms of CAPD-related hydrothorax have been postulated. (i) Lymphatic leakage from the thoracic duct [6]. The argument against this mechanism is the rarity of hydrothorax in cirrhosis of the liver, where increased thoracic duct flow and pressure (15–70 cm H$_2$O vs 6–15 cm H$_2$O in normal patients) are common [7]. (ii) Passage of fluid from the abdominal cavity to the pleural cavity by way of lymphatic channels in the diaphragm [8,9]. However, the lymphatic flow rate from the peritoneal cavity into the pleural cavity is too slow to explain the rapid movement and aggregation of the injected Tc-99m MAA [10]. These diaphragmatic lymphatics may be simply a normal route for the drainage and not responsible for the accumulation of pleural fluid [11]. (iii) Unidirectional flow of the peritoneal fluid directly via defects in the diaphragm due to pressure dynamics and perhaps to a one-way ‘ball valve’ [12,13]. It may be the most likely explanation for the development of pleural effusions in uraemic patients with normal liver and heart function on CAPD therapy. In view of the fact that anatomical defects are more common in the right diaphragm, it may explain why hydrothorax is much more common in the right chest. Three of our four cases suffered right hydrothorax, with the exception of Case 1. These diaphragmatic defects may be pre-existing (e.g. congenital defects, foramina around the major vessels and oesophagus, etc.) or acquired (e.g. those resulting from previous trauma or surgery through the diaphragm, or rupture from areas of thinning and separation of the tough collagenous fibres of the tendinous portion of the diaphragm). These defects may be large, up to 5.0 mm, and with epithelialized ducts, but are usually <1 mm in diameter [14]. They may leak and then reseal due to the chronic inflammatory reaction that accompanies ascitic accumulation, then may leak again [15]. Additionally, after the pleural and abdominal fluid reaches an equilibrium for a sufficient time, lessening of the pressure on the diaphragm might allow the healing and closure of defects [16], such as Case 3.

The diagnosis of CAPD-related hydrothorax is usually easy. However, sometimes difficulty can be encountered when CAPD-related hydrothorax co-exists with that of other aetiologies such as inadequate ultrafiltration, congestive heart failure, hypoalbuminaemia (e.g. malnutrition or liver cirrhosis), malignant pleural effusion or tuberculosis pleurisy, especially in the instances of chronic pleural effusion. Therefore, careful history taking and physical examination is mandatory. Moreover, both paracentesis and thoracocentesis should also be done to be certain that the ascites and pleural effusion are both transudates. Cell count with differential, protein concentration, cultures and cytology serve as adjunctive measures to rule out the coincidental exudative processes. Normally, the glucose level of all transudates in pleural fluid is parallel with, and is never higher than, that of the serum. In all of our four cases, the pleural fluids were transudates with relative high glucose levels, which characterized the CAPD-related hydrothorax. The fluid analysis of the pleural fluid and peritoneal effluent is not identical, and may be due to the different capacity for metabolism and absorption of the pleural surface and the peritoneal cavity.

Table 2. Characteristics, diagnosis, treatment and outcome in four cases of CAPD with massive hydrothorax

<table>
<thead>
<tr>
<th>Case</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical characteristics</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex/age (years)</td>
<td>M/24</td>
<td>M/44</td>
<td>F/30</td>
<td>M/38</td>
</tr>
<tr>
<td>Underlying renal disease</td>
<td>Lupus nephritis</td>
<td>Unknown</td>
<td>IgA nephropathy</td>
<td>Chronic GN</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Chest pain or tightness</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Retention of dialysate</td>
<td>2.0 l</td>
<td>1.5 1</td>
<td>2.0 l</td>
<td>1.5 1</td>
</tr>
<tr>
<td>Dialysate volume at diagnosis</td>
<td>18 weeks</td>
<td>1 week</td>
<td>11 months</td>
<td>1 week</td>
</tr>
<tr>
<td>Duration of CAPD before the discovery of hydrothorax</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Site of hydrothorax</td>
<td>Left</td>
<td>Right</td>
<td>Right</td>
<td>Right</td>
</tr>
<tr>
<td>PPC diagnosis by</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PF—serum glucose gradient</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Tc-99m MAA scintigraphy</td>
<td>Slow</td>
<td>Quick</td>
<td>Hot spot only</td>
<td>Quick</td>
</tr>
<tr>
<td>Treatment</td>
<td>Thoracotomy and pleurodesis</td>
<td>Oxytetracycline pleurodesis</td>
<td>Temporarily DC CAPD</td>
<td>Oxytetracycline pleurodesis</td>
</tr>
<tr>
<td>Outcome</td>
<td>Continued</td>
<td>Failed and switched to HD</td>
<td>Failed and switched to HD</td>
<td>Continued</td>
</tr>
</tbody>
</table>

PPC, Pleuroperitoneal communication; GN, glomerulonephritis; PF, pleural fluid; HD, haemodialysis.
COMMUNICATION BETWEEN THESE TWO CAVITIES, AND AID US

Employed successfully to demonstrate the severity of integrity of the diaphragm. Nuclear scans utilizing for massive hydrothorax complicating CAPD.

In conclusion, constant surveillance, including careful history taking, physical examination and follow-up of chest X-rays at regular periods, is necessary to detect pleural effusions in patients receiving CAPD therapy. After diagnostic thoracocentesis, those patients with CAPD-related hydrothorax should undergo special studies to ascertain the anatomical integrity of the diaphragm. Nuclear scans utilizing Tc-99m MAA isotope instilled intraperitoneally can be employed successfully to demonstrate the severity of communication between these two cavities, and aid us in the decision making of the therapeutic approach to this complex problem [2,20,21]. By withholding CAPD therapy and reduction of the abdominal pressure initially, thereafter we can choose pleurodesis with osteotomy (thoracoscope) [22] for the slow appearance of MAA in the pleural cavity. For patients with rapid appearance (<30 min) of MAA and insisting on CAPD therapy, we can adopt open surgery for the possible large diaphragmatic defects which may be the least likely to heal spontaneously or refractory to pleurodesis.

References


Received for publication: 11.11.98
Accepted in revised form: 16.12.98