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CASE REPORT

Pleural Effusion Developing in Two Patients on Continuous Ambulatory Peritoneal Dialysis

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ABSTRACT

Two patients with end-stage renal disease on continuous ambulatory peritoneal dialysis (CAPD) presented with pleural effusions. The aspirated fluid was categorised as transudate, based on alkaline pH, low protein and lactic dehydrogenase level. A striking feature of the pleural fluid was its very high glucose content that resulted from translocation of dextrose containing peritoneal dialysate into the pleural space via a pleuroperitoneal connection. One patient was transferred to hemodialysis, which led to complete resolution of pleural effusion. The other patient was switched to automated peritoneal dialysis, using small dwell volumes with consequent reduction in size of the pleural effusion. Pleuroperitoneal leak should always be considered in the differential diagnosis of pleural effusion in CAPD patients. Although isotopic peritoneography can demonstrate reflux of the tracer in the pleural space, measurement of pleural fluid glucose is a simpler and reliable way of diagnosing pleuroperitoneal communication.


INTRODUCTION

Continuous ambulatory peritoneal dialysis (CAPD) has emerged as an effective alternative to hemodialysis for the treatment of end-stage renal disease. It offers a more independent lifestyle and flexibility to continue to work or travel. However, continuous presence of dialysate in the peritoneal cavity can lead to increased intra-abdominal pressure (IAP), particularly in the upright body position such that the increase in IAP is directly proportional to the volume of instilled dialysate. IAP in CAPD patients can peak further at 120 - 150 cm H₂O during coughing or straining, compared to basal pressure of 2-10 cm H₂O. Excessive increase in the intra-abdominal pressure can facilitate cross-movement of peritoneal dialysate into the pleural space if a pleuroperitoneal communication, resulting from a congenital or acquired defect in the diaphragm exists in these patients. The incidence of such a defect has been estimated to be 1.6% in CAPD population.

The purpose of reporting these cases is to increase the awareness of the condition.

CASE REPORT

Patient 1: A 44-year man developed end-stage renal disease (ESRD) secondary to diabetic nephropathy. His attendance at follow-up clinics was rather irregular. He presented to the emergency department with pulmonary oedema necessitating an emergency start of hemodialysis. Hemodialysis therapy was continued via a right internal jugular permcath for 3 weeks before he opted for CAPD as long-term treatment for ESRD. Hence, a Tenckhoff catheter was placed and CAPD was initiated following a break-in period of 2 weeks. His CAPD prescription consisted of three 2L exchanges with 1.36% glucose and one 2L night-time exchange with Icodextrin.

Three weeks later, he attended the peritoneal dialysis (PD) clinic with cough and shortness of breath on exertion. There were no other cardiac, pulmonary or systemic symptoms. Physical examination showed signs of left pleural effusion. Blood pressure was 135/89 mmHg; there were no signs of fluid overload. Chest X-ray (CXR) (Figure 1a) confirmed a left pleural effusion. ECG, cardiac enzymes and echocardiogram were unremarkable. Consequently, a thoracocentesis was performed. Aspirated fluid was straw-coloured, categorised as a transudate, based on alkaline pH, low protein and lactic dehydrogenase level. A striking feature of the pleural fluid was its very high glucose content that resulted from translocation of dextrose containing peritoneal dialysate into the pleural cavity via a pleuroperitoneal connection. One patient was transferred to hemodialysis, which led to complete resolution of pleural effusion. The other patient was switched to automated peritoneal dialysis, using small dwell volumes with consequent reduction in size of the pleural effusion. Pleuroperitoneal leak should always be considered in the differential diagnosis of pleural effusion in CAPD patients. Although isotopic peritoneography can demonstrate reflux of the tracer in the pleural space, measurement of pleural fluid glucose is a simpler and reliable way of diagnosing pleuroperitoneal communication.
Patient 2: An 18-year female patient with ESRD due to lupus nephritis was commenced on hemodialysis. In view of multiple failed arteriovenous fistulas, she was switched to CAPD. Her CAPD prescription was gradually intensified to two 2.5L exchanges with 1.36% glucose, one 2L exchange with 2.27% glucose and one 2L night-time exchange using Icodextrin, to achieve the target Kt/V of > 1.7. She presented with progressive dyspnea for 3 weeks. Physical examination showed signs of fluid overload and bilateral pleural effusions. CXR verified pulmonary congestion and pleural effusions on both sides (Figure 2a).

She was treated with rapid peritoneal exchanges using hypertonic (3.86% glucose) dialysate that provided an ultrafiltration of 2.2 litres over 2 hours and relief from pulmonary congestion. At the time of discharge, her PD prescription included one 2.5 L exchange with 1.36% glucose, two 2L exchanges with 2.27% glucose and one 2L night-time exchange with Icodextrin.

Clinical and radiological review next month revealed significant resolution of the left sided pleural effusion but a paradoxical increase in the right sided pleural effusion (Figure 2b). A diagnostic right pleural tap was carried out. Aspirated fluid turned out to be a transudate (Table I; patient-2) with very high concentration of glucose indicating translocation of peritoneal dialysate into the pleural cavity. ANA was positive (1:160) but serum complement levels were normal.

Hemodialysis was not an option in view of unavailability of vascular access. She was switched to automated peritoneal dialysis using smaller exchange volumes. Follow-up X-rays showed marked reduction in the size of right sided pleural effusion.

DISCUSSION

Analysis of the pleural aspirate is crucial to ascertain its nature and etiology in CAPD patients presenting with pleural effusion unless clinical context clearly points to fluid overload or heart failure. Pleural effusion in both the patients had characteristics of a transudate, i.e. alkaline pH, pleural fluid-to-serum protein ratio of less than 0.5, and lactic dehydrogenase level of less than 60% of the serum level. A salient feature of the pleural fluid was its very high glucose content (sweet hydrothorax) due to the presence of dextrose (glucose monohydrate) - the most commonly employed osmotic agent in PD solutions. The true anhydrous glucose concentrations in dialysates are 1.36%, 2.27% and 3.86%, equivalent to 76 mmol/L, 126 mmol/L and 214 mmol/L of glucose, respectively. The ratio of pleural fluid to serum glucose is dynamic and varies with the type of fluid instilled into the peritoneal cavity. There is no absolute glucose concentration level that can be used to diagnose pleuroperitoneal leak; any pleural-fluid glucose concentration greater than that of serum is considered to be diagnostic of pleuroperitoneal leak.

Isotopic peritoneography using Tc-99m DTPA produced fascinating picture of radioisotope tracer refluxing into the pleural space in patient 1; but this test has a sensitivity of only 40 - 50%. Others have utilised CT peritoneography to demonstrate the pleuroperitoneal communication but the diaphragmatic defect can sometimes be too small to be picked-up by peritoneography. Hence, measurement of pleural fluid glucose is considered a much simpler, quicker, and reliable test for diagnosing pleuroperitoneal leak.

Pleural effusion developed soon after starting CAPD in patient-1, whereas it developed as a late complication in patient-2. It is plausible that increase in the dwell volumes in patient-2 (to achieve the desired Kt/V) and the resultant high intraperitoneal pressure precipitated the development of hydrothorax. Hence, it was possible to continue PD using smaller dwell volumes in supine position using night-time automated PD - maneuvers that are known to avoid excessive rises in intra-abdominal pressures. Patient-1 was switched to

Table I: Biochemical results of blood and pleural fluid of the two patients.

<table>
<thead>
<tr>
<th>Patient-1</th>
<th>Patient-2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pleural fluid</td>
<td>Serum</td>
</tr>
<tr>
<td>pH</td>
<td>7.69</td>
</tr>
<tr>
<td>Protein (g/L)</td>
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</tr>
<tr>
<td>LDH (U/L)</td>
<td>52 U/L</td>
</tr>
<tr>
<td>Glucose (mmol/L)</td>
<td>39</td>
</tr>
</tbody>
</table>

Figure 1: Patient-1: (1a) CXR showing left pleural effusion. (1b) Isotopic peritoneography demonstrating reflux of the tracer material into the left pleural space (*).
hemodialysis to provide rest to his peritoneum for 3 - 4 months. While this in itself can sometimes lead to healing of the diaphragmatic fistula, any recurrence of pleural effusion on reinstitution of CAPD requires either permanent transfer to hemodialysis or pleurodesis/surgical repair of the diaphragm.9

REFERENCES