Pleuroperitoneal leak (PPL) - A diagnostic dilemma resolved by peritoneal scintigraphy in a patient on continuous ambulatory peritoneal dialysis (CAPD) - A case report

VASANTH G KGANESAN
Department of Nephrology,
CHRISTIAN MEDICAL COLLEGE

Abstract:
Hydrothorax secondary to pleuroperitoneal communication is an unusual complication of continuous ambulatory peritoneal dialysis. Many modalities have been used to diagnose pleuro peritoneal shunt related hydrothorax. Pleural fluid analysis is a simple, cost effective approach but it is not always helpful to diagnose this entity. When pleural fluid analysis is non-contributory, Tc-99m gammagraphy, computed tomography scan and magnetic resonance image can be used for diagnosis. Among these Tc-99m Sulphur colloid scintigraphy is a very sensitive method to detect if biochemical analysis are inconclusive.

Keyword: Hydrothorax, Pleuroperitoneal shunt, CAPD, Scintigraphy

Introduction:
Continuous ambulatory peritoneal dialysis (CAPD) is an established effective renal replacement therapy for patients with end-stage renal disease (ESRD). There are various causes for hydrothorax in patient with ESRD.

Hydrothorax due to pleuroperitoneal shunt after CAPD is a rare complication. Erroneous conclusion could be detrimental for patient care and management. Simple pleural fluid analysis will be helpful for diagnosing most cases of pleuroperitoneal leak. However if biochemical analysis is inconclusive, imaging modalities like peritoneal scintigraphy, contrast CT scan are required to prove the presence of pleuroperitoneal shunt. Case report A 28 years old gentleman with Stage 5 CKD due to idiopathic fibrillary glomerulonephritis was initiated on continuous ambulatory peritoneal dialysis (CAPD) in October 2012 presented to CAPD clinic two months after catheter insertion with history of breathing difficulty for 5 days. He was not a diabetic and there was no past history of tuberculosis. On examination he was tachypneic and there was mild pallor with no pedal edema. He had a urine output of around 1000 ml per day and was on 3 exchanges of 2 litres 2.5% dextrose solution for 6 hours each with night dry. His blood pressure was 160/80 mmHg and pulse rate was 100 per minute. He maintained saturation of
98% with 1 litre of nasal oxygen. JVP was not elevated. On auscultation there were diminished breath sounds on right side of chest without crepitations or murmurs. Chest x ray showed right sided massive pleural effusion (Fig-2). Chest x ray prior to catheter insertion was normal (Fig-1).

Fig-1 xray before CAPD Fig-2 Xray at presentation- 2months after CAPD

Diagnostic & therapeutic thoracentesis was done and fluid was sent for analysis. Patient's breathing difficulty came down after pleural tapping. His blood pressure medications were optimized. Subsequent echo cardiogram showed left ventricular hypertrophy with ejection fraction of 56% and there was no pericardial effusion. His blood and pleural fluid analysis were reported as follows. His pleural fluid was sterile and transudative by Light's criteria. Simultaneous peritoneal fluid glucose level was 806 mg% and cell count was 10 cells/cumm. There was no feature suggestive of peritonitis. His ultrasound abdomen was normal except for bilateral contracted kidneys. After 5 days he underwent CT peritoneography to demonstrate pleuroperitoneal leak. After draining pleural fluid a baseline CT thorax was obtained, then 50 ml of contrast was mixed with 2L of PD fluid and infused into peritoneal cavity. A repeat CT thorax cuts were taken after 2 hours (fig -4). There was no increase in Hounsfield(HF) units to suggest a leak. No obvious communication was demonstrated.

Fig-3 xray showing reaccumulation of fluid

Since it was a transudative pleural effusion and there were no features of volume overload, cardiac failure or hypoalbuminemia a presumptive diagnosis of pleuroperitoneal leak was made. However, biochemical parameters were inconclusive of leak since pleural fluid glucose content was not high (<300 mg%) and there was no significant pleural fluid serum glucose gradient.
He underwent CT peritoneography to demonstrate pleuropertitoneal leak. After draining pleural fluid a baseline CT thorax was obtained, then 50 ml of contrast was mixed with 2L of PD fluid and infused into peritoneal cavity. A repeat CT thorax cuts were taken after 2 hours (fig-4). There was no increase in Hounsfield(HF) units to suggest a leak. No obvious communication was demonstrated.

**Fig-4 CT peritoneography with limited cuts**
CT chest showing no change in Hounsfield unit in chest film after two hours of contrast in CAPD fluid compared to baseline CT chest (only limited films showed)

Subsequently he underwent Tc-99m Sulphur colloid scintigraphy. In this 80 mBq of Tc-99m Sulphur colloid was instilled into the 2 L CAPD fluid bag and infused through PD catheter. Dynamic images were acquired for 1 hour followed by delayed static views at 2 hours. Tracer activity was demonstrated in right pleural space suggestive of pleuro peritoneal leak (Fig-5).

**FIG-5-Scintigraphy showing tracer activity in right pleural cavity confirming pleuropertitoneal shunt**

His CAPD was temporarily discontinued and he was initiated on hemodialysis through right internal jugular catheter. Options were discussed with patient and his relatives and subsequently he underwent right intercostal drainage followed by pleurodesis with betadine. Post pleurodesis there was complete obliteration of right pleural space(Fig-6).

X ray after pleurodesis

It was planned to restart CAPD after 6 weeks. Fortunately patient received a deceased donor kidney transplantation while on hemodialysis and PD catheter was removed 6 weeks after transplantation. Discussion Peritoneal dialysis (PD) is an established effective renal replacement therapy for patients with end-stage renal disease (ESRD). An uncommon complication is hydrothorax due to pleuroperitoneal communication which occurs in 1.6–10% of PD patients. The first description of a pleuroperitoneal leak causing a pleural effusion in a PD patient was in 1967. The peritoneo-pleural communication can be either bi-directional or uni-directional. Presence of preexisting congenital diaphragmatic defect leads to development of hydrothorax immediately after starting CAPD. This confirms bi-directional flow of dialysate through pre-existing congenital defect. Negative intra-pleural and positive intra-abdominal pressure generated during the descent of diaphragm open small acquired defects and behave as ball valves allowing unidirectional transit of dialysate from peritoneum to pleural cavity which usually occurs late. These late occurrence is due to increased intraabdominal pressure and
subsequent separation of diaphragmatic collagen fibres and pleural bleb formation which rupture subsequently leads to acquired opening. The fluid that accumulates in patients with CCF, cirrhosis, pancreatitis, uremia, urinothorax, PD, and nephrotic syndrome may also have the characteristics of a transudate. Patients usually present with dysnea and chest pain, which is more marked during CAPD inflow. The initial suspicion may be aroused due to reduced dialysate outflow. Tension hydrothorax, respiratory failure and ultrafiltration failure are more severe sequelae, which can lead to discontinuation of CAPD . Patients with adult polycystic kidney disease have a higher incidence of hydrothorax complicating PD, presumably due to increased intraabdominal pressure . Intra-abdominal pressure reaches 120 – 150 cm H2O with coughing or straining, compared to 0.5 – 2.2 cm H2O in normal people and 2 – 10 cm H2O in patients with peritoneal fluid . Elevated intraabdominal pressure predisposes the patient to thinning of the diaphragm. High volume (40 mL/kg) PD exchange at initiation in children causes more hydrothorax compared to those children starting with low volumes (10 mL/kg). Pleural fluid analysis is a simple cheap diagnostic test. The following diagnostic indices can be used for diagnosis of pleuroperitoneal shunt.

1. Pleural fluid glucose concentration above 300 mg/dL was considered diagnostic, but some studies have proposed using a pleural fluid to serum gradient of 50 mg/dL for diagnosis.  

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Pleural fluid</th>
<th>Serum</th>
<th>Peritoneal fluid</th>
</tr>
</thead>
<tbody>
<tr>
<td>LDH</td>
<td>33</td>
<td>857</td>
<td>-</td>
</tr>
<tr>
<td>PROTEIN</td>
<td>0.3 g/dl</td>
<td>6.3 g/dl</td>
<td>-</td>
</tr>
<tr>
<td>ALBUMIN</td>
<td>0.1 g/dl</td>
<td>3.9 g/dl</td>
<td>-</td>
</tr>
<tr>
<td>GLUCOSE</td>
<td>139 mg%</td>
<td>142 mg%</td>
<td>806 mg%</td>
</tr>
<tr>
<td>CELL COUNT</td>
<td>07/cumm</td>
<td>7200</td>
<td>10 cells/cumm</td>
</tr>
<tr>
<td>ADENOSIN DEAMINASE</td>
<td>3.2 U/L</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>HEMOGLOBIN</td>
<td>-</td>
<td>10.1 g%</td>
<td>-</td>
</tr>
</tbody>
</table>

1 Measurement of D-lactate in pleural fluid is useful test. L-lactate is endogenous source. But PD fluid contains both D and L-lactate. Demonstration of D-lactate in pleural fluid is diagnostic of PPS. However D lactate assay is not easily available in most centers.

2 Alternative peritoneal dialysis solutions such as icodextrin may not contain glucose. In this setting, the addition of povidone-iodine to a fluid aspirate may result in a characteristic blue-black color. But this method is not approved by many authors.

4. Pleural fluid glucose concentration more than serum glucose concentration (P/S > 1) is another way of diagnosis suggested in some studies. But sometimes it is difficult to get conclusive reports from glucose analysis alone. Pleural fluid glucose concentration could be affected by size of pleuroperitoneal communication, rate of fluid movements across membranes, posture of the patient, glucose concentration at the time of...
diagnosis and rate of glucose absorption from pleural cavity. Methelene blue instilation in to PD fluid and demonstration in pleural fluid is also useful method but due to the fear of chemical peritonitis it is not considered routinely. Contrast computed tomography peritoneography was associated with 33% sensitivity in one study. It will give further anatomical details about peritoneum and adhesions. Radionuclide scintigraphy using technetium-99m tagged macro-aggregated albumin or Tc-99m sulfur colloid confirms abnormal pleuroperitoneal communication in a noninvasive fashion and is associated with sensitivity of 40% to 50%. It has less radiation exposure and no adverse effect on peritoneum. Scintigraphy with Tc99m (2-5 mCi) is associated with a number of advantages given that it is a non-invasive technique, it is low-cost and it can provide several images without increasing the radiation that the patient is exposed to. This imaging technique begins with the intraperitoneal administration of the radioisotope. New images are taken two hours after administration (in order to blend the mixture) and then all the contrast is expelled when the peritoneal liquid is drained. The test is positive if the radioisotope is detected in the pleural cavity. In our patient pleural fluid glucose concentration was 139 mg%( <300 mg%) and serum glucose concentration was 142 mg%( no significant gradient) and ascitic fluid glucose concentration was 806mg%. However we strongly suspected pleuroperitoneal leak since it occurred very early after initiation of CAPD and there was no other causes for this transudative effusion. Hence we proceeded with a sensitive method like scintigraphy and demonstrated a pleuroperitoneal leak.

Conclusion:

Scintigraphy is more sensitive than other imaging modalities and it should be considered if there is a difficulty in demonstrating a pleuroperitoneal leak using pleural fluid analysis alone.

REFERENCES:
2) Mufazzal Ahmad, Anshu Rajnish "Role of Peritoneal Scintigraphy in the Management of Hydrothorax Caused by Continuous Ambulatory Peritoneal Dialysis" Indian Journal of Nuclear Medicine, Vol. 17, No. 2 & 3, June & Sept., 2002