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Peritoneal Dialysis Complicated by Pleuroperitoneal Communication and Hydrothorax

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Background: Hydrothorax is an uncommon but well-recognized complication of peritoneal dialysis. It is a potentially serious condition that frequently requires cessation of peritoneal dialysis and permanent transition to hemodialysis. Hydrothorax is produced by movement of peritoneal dialysate through pleuroperitoneal fistulas. Pleural fluid analysis typically detects a high glucose concentration, and contrast imaging reveals tracer uptake transgressing the diaphragm. Experience with the treatment of hydrothorax related to peritoneal dialysis is limited.

Case Report: We describe the case of a 54-year-old female on peritoneal dialysis for end-stage renal failure who developed a hydrothorax soon after beginning treatment.

Conclusion: This case describes a classical presentation of hydrothorax in the context of peritoneal dialysis. Treatment is frequently unsuccessful. All clinicians prescribing peritoneal dialysis should be aware of this complication.

Keywords: *Ascitic fluid, hernias–diaphragmatic–congenital, hydrothorax, peritoneal dialysis*

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INTRODUCTION

Hydrothorax is an uncommon but well-recognized complication of peritoneal dialysis (PD). Hydrothorax is defined as a pleural effusion composed of serous fluid and is potentially life-threatening. The pathogenesis of hydrothorax in PD is incompletely understood. Typical signs include pleuritic chest pain and breathlessness. Development of a pleural effusion mandates immediate discontinuation of PD and precludes its resumption in many patients. Experience with patients on PD who develop hydrothorax is limited, and relatively few cases have been reported in the literature. This condition has no standard treatment, and practice is particularly varied among physicians unfamiliar with patients on dialysis. We describe a case of PD complicated by acute and significant hydrothorax and its subsequent management.

CASE REPORT

A 54-year-old female on PD presented to the emergency department in respiratory distress. She had begun continuous ambulatory peritoneal dialysis (CAPD) 6 weeks earlier for end-stage renal failure secondary to tuberous sclerosis. Her Tenckhoff catheter had been inserted surgically 4 weeks prior to the initiation of PD. The initial CAPD prescription comprised 3 daily exchanges of 2 liters of 2.5% dextrose-based dialysate.

The patient developed progressive shortness of breath and right-sided pleuritic pain in the week prior to presentation that gradually worsened to the point of severe breathlessness. She also reported declining daily output from her Tenckhoff catheter during the previous 2 weeks.

Her kidney failure was the result of focal segmental glomerulosclerosis and angiomyolipomas caused by tuberous sclerosis. She also suffered from cerebral, cutaneous, and skeletal involvement. Other significant medical history included right lobular carcinoma in situ with right total mastectomy, appendectomy, uterine fibroids requiring hysterectomy, and hemorrhoidectomy. The patient's medications included darbepoetin, lanthanum carbonate, oxcarbazepine, vigabatrin, and tamoxifen.

On examination at arrival in the emergency department, the patient was in obvious respiratory distress. Her breathing was labored with a respiratory rate of 22 breaths per minute and an oxygen saturation of 89% on 2 liters of supplemental oxygen per minute, delivered via nasal cannula. Other vital signs were within normal limits. Reduced breath sounds and dullness to percussion were noted on the right side. Chest radiograph demonstrated a large right-sided pleural effusion (Figure 1). An intercostal catheter was inserted, and the patient's condition rapidly improved.

A total of 3.5 liters of pleural fluid was drained during the following days. A specimen of straw-colored fluid was collected and sent for analysis. Laboratory findings were consistent with a transudative effusion; no organisms were cultured and no malignant cells were visualized. The pleural

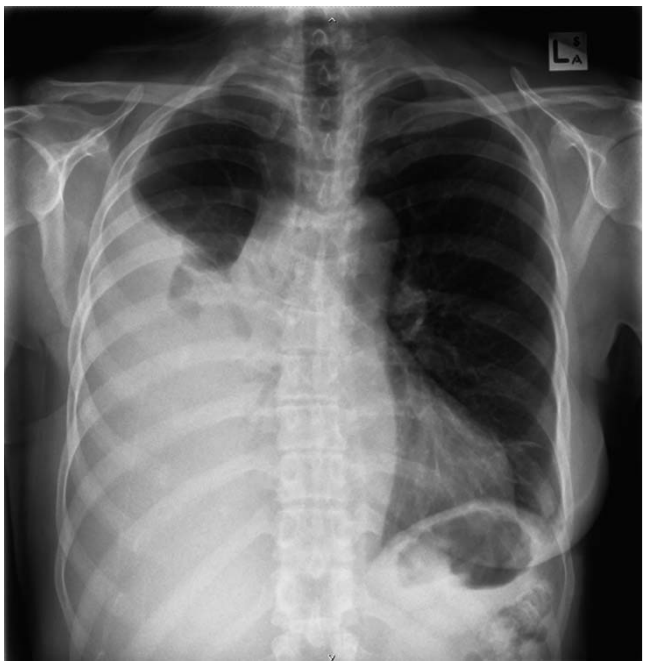

Figure 1. Large right-sided pleural effusion demonstrated on chest radiograph at the time of patient presentation.

fluid glucose concentration was 19.5 mmol/L compared with a serum glucose concentration of 6.8 mmol/L.

Computed tomography (CT) with contrast administered through the Tenckhoff catheter into the peritoneal cavity failed to identify dispersal of contrast material into the right hemithorax. A nuclear isotope scan was subsequently done. Following administration of technetium 99m via the PD catheter, a high volume of radioactive dialysate was detected entering the right hemithorax (Figure 2). No tracer activity was seen in the left hemithorax.

The presence of a pleuroperitoneal fistula was suspected. After her effusion was drained, the patient's body weight was 5 kg heavier than her dry weight of 57.3 kg measured 1 month earlier. PD was withheld, and the patient transitioned to temporary hemodialysis. Video-assisted thoracoscopy 1 week later did not identify any pleuroperitoneal communication. Talc pleurodesis was performed under direct thoracoscopic vision.

The patient recommenced PD 4 weeks after chemical pleurodesis. Her CAPD prescription was modified to 1.5 L of 1.5% dextrose-based dialysate solution exchanged 5 times daily. At 5-week follow-up after recommencing PD, the patient had no evidence of hydrothorax recurrence, and she continued to meet her targets of dialysis adequacy.

DISCUSSION

PD is a commonly used dialysis technique for patients with end-stage renal failure. PD is an effective treatment, but it is not without risk. The most frequent and important complication of PD is infection.[1] Other common complications include catheter site leaks, catheter blockage, abdominal wall herniation, and intestinal perforation.

Hydrothorax is a rare complication, caused by migration of fluid from the peritoneal cavity into the pleural space via pleuroperitoneal fistulas. These diaphragmatic defects are usually congenital and right-sided, explaining the predominance of right-sided effusions.[2] Hydrothorax is estimated to occur in 1.6% of patients on PD.[3] The majority of cases occur within 30 days of commencing PD, and up to 25% are asymptomatic.[3,4] Hydrothorax should always be considered when patients on PD present with pleural effusions; however, the usual differential diagnoses for pleural effusions also apply.

Typical features suggesting hydrothorax include dyspnea and pleuritic pain. Incomplete recovery of distilled fluid from the peritoneal catheter is an important clue to the diagnosis. Intraabdominal pressure is the predominant influence for hydrothorax but does not necessarily correlate with peritoneal dialysate volume.[3,5,6] Hypertonic dialysate also increases risk[7] because hyperosmolality produces an osmotically driven volume flux from tissues into the cavity, further increasing intraabdominal pressure.[8]

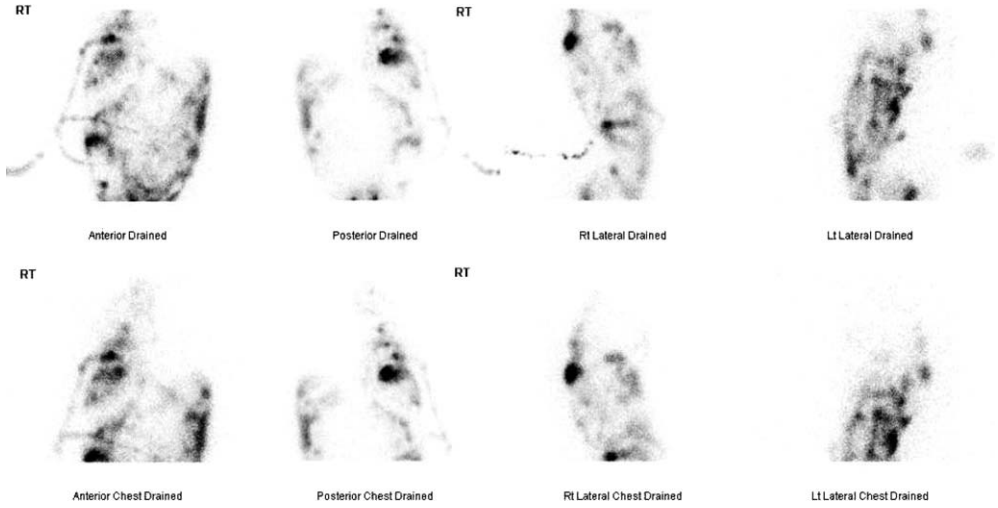

Figure 2. Peritoneal scintigraphy demonstrating a high volume of radioactive dialysate in the right hemithorax with no abnormal tracer accumulation detected in the left hemithorax, thereby confirming a right pleuroperitoneal fistulous communication.

No single test definitively diagnoses hydrothorax. Diagnosis instead requires a combination of biochemical and radiographic findings.

Analysis of the pleural fluid will identify a transudate with an elevated glucose concentration. The glucose concentration in dialysate and peritoneal fluid is higher than the serum glucose concentration. No consensus agreement on a diagnostic pleural glucose level has been reached, and its accuracy for this purpose has not been studied.[9] Although the diagnostic contribution of effusate glucose is unreliable, it is generally accepted that pleural fluid with a glucose concentration $>$16.5 mmol/L or with a concentration greater than that of the serum concentration is consistent with hydrothorax.[4,10] Pleural fluid glucose is influenced by previous dialysate composition, blood glucose, and the rate of pleuroperitoneal leakage. Interpretation in patients with diabetes is difficult and also in patients with small diaphragmatic defects in whom glucose absorption from the pleural cavity may be greater than in patients with larger defects.[11]

Various imaging studies can be helpful in identifying pleuroperitoneal communication. The most informative is peritoneal scintigraphy that has a sensitivity of approximately 50%.[10] A radioisotope in the form of technetium 99m is instilled into the PD fluid, after which a series of images is obtained. Tracer uptake detected in the thoracic cavity confirms a communication between the peritoneum and the pleural space. Alternative techniques that are used less frequently are CT and magnetic resonance peritoneography.

No current guidelines or standards of care for hydrothorax in patients on PD have been developed.[12] A range of conservative and invasive management options has been reported in case series, but no comparative controlled trials have ever been undertaken to compare treatment strategies.[2] The treatment of hydrothorax related to PD often has limited success, and most patients eventually require transition to permanent hemodialysis.[2]

Most authorities suggest beginning with a conservative approach. PD should first be withheld to permit spontaneous resolution of the hydrothorax and diaphragmatic connection. Peritoneal dialysate is hypothesized to act as a sclerosant and to spontaneously seal the pleuroperitoneal tracts in some patients.[5] During this time, patients require conversion to temporary hemodialysis. After approximately 1 month, they may then be reintroduced to PD gradually with low-volume exchanges in a semi-upright position. Ultimately however, the success of this approach is suboptimal, and many patients experience hydrothorax recurrence.[13,14] Removal of pleural fluid through insertion of an intercostal catheter should be restricted to patients in respiratory distress.

Chemical pleurodesis is generally the next step if conservative measures fail. Because of the high failure rate associated with conservative management, many authorities advocate proceeding to pleurodesis at the outset.[15] Chemical pleurodesis involves the instillation of irritants into the pleural cavity via an intercostal catheter with the intention of artificially obliterating the pleural space by inducing adherence of the pleura. Agents available for pleurodesis include talc, tetracycline, doxycycline, and autologous blood. No randomized study data support one agent over another, and their efficacy appears to be similar in small case series and observational studies.[2] Following chemical pleurodesis, patients should wait at least 10 days before recommencing PD to allow time for sufficient scar formation over the defect.[2,16] Most nephrologists defer reimplementation of PD for 2-6 weeks to improve the efficacy of pleurodesis.[17] The likelihood of successfully resuming PD following chemical pleurodesis is approximately 50%.[2,5,18]

Surgical correction is the most efficacious treatment but also the most invasive. Several methods are available, including surgical pleurectomy, mechanical abrasion and pleurodesis, chemical pleurodesis, and diaphragmatic patching. These procedures may be performed through open surgery or video-assisted thoracoscopic surgery. Both surgical techniques are highly effective for this indication, and the likelihood of a patient being able to successfully reinstitute PD without hydrothorax recurrence exceeds 90%.[2] Because of the invasive nature of these treatments, however, some patients do not elect surgery and instead convert permanently to hemodialysis if the conservative approaches fail.

CONCLUSION

Hydrothorax is an infrequent but well-described complication of PD. It is an important diagnosis that may require urgent treatment. No standard practice for this condition exists, and experience with investigations and management is limited. This case report demonstrates a classical presentation of hydrothorax and highlights its relevance for all clinicians encountering patients receiving PD.

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