

**Case Report**

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# Acute Hydrothorax Diagnosed Through Scintigraphy in a Patient on Peritoneal Dialysis



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## Abstract

Acute hydrothorax after peritoneal dialysis (PD) is uncommon. Differential diagnoses of various clinical conditions that can result in pleural effusions are necessary. Erroneous diagnoses and subsequent management not only increase the time required for resolving a problem but also deteriorate a patient's clinical condition. We report the case of a 55-year-old woman who received regular PD due to chronic renal failure. She experienced diffused abdominal pain 2 hours after PD. Furthermore, dyspnea developed the following morning. A chest plain film revealed a massive right-sided pleural effusion. Peritoneo-pleural communication caused by a diaphragmatic defect was diagnosed rapidly using lung perfusion scintigraphy.

**Keywords:** Hydrothorax; Pleural effusion; Peritoneal dialysis; Scintigraphy

**Abbreviations:** PD: Peritoneal Dialysis; CAPD: Continuous Ambulatory Peritoneal Dialysis; ED: Emergency Department; Tc-99m MAA: Technetium-99m Macro Aggregated Albumin; PF-S: Pleural-Fluid-to-Serum

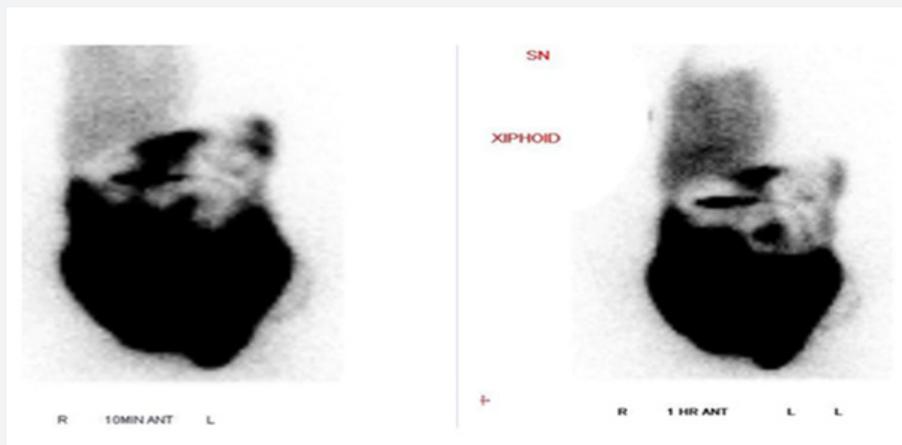
## Case presentation

Our patient, a 55-year-old woman, had a history of hypertension and chronic glomerulo-nephritis-related uremia. She had undergone peritoneal dialysis (PD) catheter insertion in September 2014 and started receiving continuous ambulatory peritoneal dialysis (CAPD) twice daily 1 month after catheter insertion. She experienced intermittent abdominal pain 3 months after the insertion. No fever or dyspnea were noted with the pain. However, a decrease in ultrafiltration volume or rate after the PD session was noted by the patient herself. A review of her medical history did not reveal recent trauma to the chest or previous diaphragmatic surgery. However, shortness of breath was noted 1 day later; hence, she presented to the emergency department (ED). Tachycardia (104bpm) and elevated blood pressure (185/111mmHg) were noted in triage, and her respiratory rate was 22 breaths/min with 98% oxygen saturation. No chest pain or abnormal findings in the electrocardiogram were noted during her ED stay. Laboratory results did not reveal leukocytosis or acidosis. A chest X-ray revealed a massive right-sided pleural effusion (Figure 1). Pleurocentesis was suggested, but the patient refused. We analyzed the dialysate instead of a sample of the pleural effusion fluid; no evidence of infection was found. We arranged lung perfusion scintigraphy, 5 mCi of Technetium-99m macro aggregated albumin (Tc-99m MAA) was injected into the PD fluids, and a rapid accumulation of radioactivity

in the right hemithorax was observed in the images recorded at 30 min and 1 h after PD (Figure 2). Thus, peritoneo-pleural communication (right side) caused by diaphragmatic defect was diagnosed. PD was discontinued immediately and video-assisted thoracic surgery with thoracoscopic diaphragmatic fundoplication and pleurodesis was provided 2 days later. The patient was discharged uneventfully without the recurrence of pleural effusions during a 3-month follow-up.



**Figure 1:** Massive pleural effusion on the right side without cardiomegaly. Arrow indicates continuous ambulatory peritoneal dialysis catheter placement.



**Figure 2:** Lung perfusion scintigraphy images showing rapid and progressive accumulation of radioactivity in the right hemithorax in serial recordings at 30 min and 1 h.

## Discussion

Hydrothorax-related to PD was first reported in 1967 by Edward and Unger [1]. The reported incidence rates of hydrothorax-related PD vary from 1.6% to 10% [2,3]. The incidence rate in new PD patients is <2% [2]. Pleural effusions are usually observed on the right side, presumably because the left side has diaphragmatic protection provided by the heart. Clinical symptoms of pleural effusions include sudden dyspnea, decrease in ultrafiltration rate, and pleuritic chest pain. One study reported that approximately 25% of patients are asymptomatic [4]. In patients with recurrent unilateral pleural effusions or acute respiratory distress after dialysate infusion, trans-diaphragmatic leakage or peritoneal fistulae should be considered. When transudative pleural effusions are confirmed using Light's criteria in patients receiving PD, pleural effusion glucose levels can aid diagnosis. Some authors use a cutoff point of 300mg/dL of pleural effusion glucose for diagnosis [5], whereas others consider a pleural-fluid-to-serum (PF-S) glucose gradient of >50mg/dL, with a sensitivity of 100%, as an indicator [6]. A relatively objective measurement revealed that a PF-S glucose ratio of >1 is consistent with pleuroperitoneal communication because all other causes of transudative pleural effusions have similar or lower glucose concentrations in the pleural fluid compared with the serum (ratios of  $\leq 1$ ) [7]. Any image survey alone is insufficiently sensitive for detection. In most cases, peritoneo-pleural fistulae are diagnosed through scintigraphy or radionuclide scanning (for example, Tc-99m DTPA), with sensitivities of only 40% to 50% [8,9]. However, patients receiving CAPD who present with acute shortness of breath or recurrent unilateral pleural effusions should be examined through peritoneal scintigraphy to eliminate the possibility of a pleuro-peritoneal leak. Several therapeutic approaches can be adopted, including temporary discontinuation of PD, tetracycline instillation into the pleural space, and surgical patch grafting of the diaphragmatic defect. The strategy required to manage the effusion depends on the clinical condition of the patient; however, in all cases, immediate interruption of the PD is required. Surgical intervention was provided to this patient

because she showed rapid accumulation of radioactive material in the right hemithorax.

## Conclusion

In patients who receive regular PD, sudden accumulation of pleural effusion can be diagnosed by minimal invasive scintigraphy instead of pleural effusion tapping. Rapid diagnosis can help us decide whether to discontinue PD to prevent deterioration

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