CASE REPORT

Sweet hydrothorax: a common presentation of a rare condition

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ABSTRACT

Sweet hydrothorax is a known, yet rare, complication of peritoneal dialysis. It can be life-threatening. This case is about a 70-year-old lady who presented with acute respiratory failure due to massive right-sided hydrothorax that developed insidiously over 3 months of starting peritoneal dialysis. Thoracentesis and technetium scan confirmed the diagnosis. Treatment was successful with hemodialysis.

Key words: Diaphragmatic defect, peritoneal dialysis, pleural effusion, pleuroperitoneal communication, sweet hydrothorax, technetium scan

Key messages

- Peritoneal Dialysis (PD) can be complicated by sweet hydrothorax.
- Symptoms onset after initiation of PD and symptoms resolve with its discontinuation suggest the diagnosis.
- High gradient glucose level between pleural fluid and plasma is a hallmark feature of sweet hydrothorax.
- Patients with life-threatening symptoms can consider hemodialysis or surgical options.

INTRODUCTION

Peritoneal dialysis (PD) is a form of dialysis offered to patients with end-stage renal disease (ESRD). It is associated with less mortalities in the first 2 years^[1] and can provide the best dialysis modalities based on patient's health and home situation as well as dialysis-center factors. Clinicians should be familiar with its complications. One rare complication that can be life-threatening is PD-associated hydrothorax. Although it has been described in nephrology literature, limited number of cases has been published in general medicine journals. Herein, we present a case of acute respiratory failure due to massive hydrothorax related to PD.

nephropathy complicated by ESRD, for which she was started on PD 3 months ago, presented to the hospital with deteriorating dyspnea associated with nonproductive cough over 2 weeks.

She has been compliant with her dialysis sessions that were well-tolerated. She was treated for bronchitis before presentation with no improvement. The patient had good functional status before the presentation and lived independently. She denied productive cough, pleuritic chest pain, lower extremity edema, or fever. No history of diabetes mellitus, immobility, or calf swelling was reported. Vitals showed blood pressure of 133/84 mm Hg and heart rate of

CASE HISTORY

A 70-year-old female patient with past medical history of well-controlled rheumatoid arthritis and hypertensive

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96 beats per minute, hypoxia with 70% saturation on room air, and tachypneic with respiratory rate in 20 breaths per minute. Physical examination was significant for decrease breath sounds and dullness on percussion over the right lower lobe. No active arthritis, pitting edema, or elevated jugular venous pressure was noted. Laboratory tests showed mild leukocytosis and hyperglycemia [see Table 1].

Chest x-ray revealed a massive right-sided pleural effusion that was not present on an x-ray taken few weeks before starting the PD (see Figures 1 and 2). Chest computed tomographic angiography showed no pulmonary emboli (see Figure 3). She was admitted to intensive care unit and was put on high flow oxygen therapy.

A thoracentesis of 1.3-L slightly yellow tinged clear fluid provided significant symptom relief. Analysis was consistent with transudative effusion and high glucose level in pleural fluid in comparison with serum glucose level (see Table 1). Technetium 99m (Tc 99m) scintigraphy showed leakage of Tc 99m into right pleural space [Figure 4]. This suggests peritoneopleural communication due to a large, most likely congenital, right diaphragmatic defect.

PD was discontinued and patient was started on hemodialysis. Follow-up evaluation showed neither recurrence of symptoms nor recurrence of pleural effusion on chest x-ray (see Figure 5).

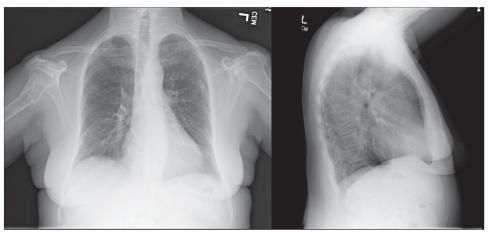


Figure 1: The lungs are well expanded and clear bilaterally. No focal consolidation, pleural effusion, or pneumothorax is seen. Stable mild cardiac enlargement

Table I: Laboratory and biochemical values on admission ^[1]		
WBC	15.9	4–11 Thou/mm ³
BUN	30	6–20 mg/dL
Creatinine	4.09	0.4-1.03 mg/dL
Glucose	162	70–110 mg/dL
INR	l	0-1.9
Total protein	6.7	5.9–7.5 g/dL
Albumin	3.5	3.3-4.5 g/dL
ALT	9	3-37 U/L
AST	18	8-34 U/L
Alkaline phosphatase	68	25-105 U/L
Total bilirubin	0.21	0.2-1 mg/dL
Pleural glucose	230	mg/dL
Pleural LDH	21	U/L
Plasma LDH	162	U/L
Pleural RBC	8	U/mm³
Pleural WBC	91	U/mm³
Pleural neutrophil	6	%
Pleural pH	7.9	
Pleural cholesterol	<4	mg/dL
Pleural total protein	<1.0	g/dL

WBC = white blood cell, BUN = blood urea nitrogen, INR = international normalized ratio, ALT = alanine transaminase, AST = aspartate transaminase, LDH = lactate dehydrogenase, RBC = red blood cell

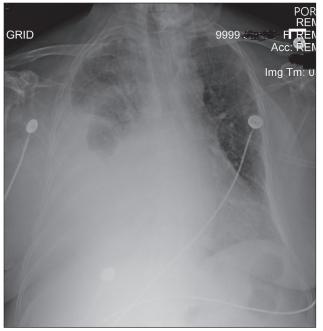


Figure 2: Near complete opacification of the right hemithorax secondary to a large effusion and atelectasis or consolidation. Recommend imaging follow-up after treatment to ensure resolution

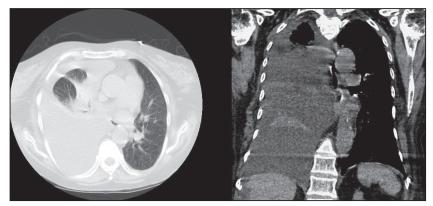


Figure 3: Very large right pleural effusion that occupies nearly the entire right hemithorax with compressive atelectasis and consolidation in the right perihilar region. Recommend imaging follow-up after treatment to ensure resolution

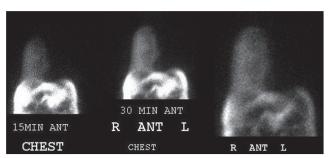


Figure 4: Tc-99m scintigraphy: 4.5 mCi of technetium 99m was injected through peritoneal dialysis catheter. Subsequently, scintigraphy of the chest was performed at 15 (on the left), 30 (on the middle), and 120 min (on the right)

DISCUSSION

Hydrothorax is a known complication of PD that occurs in about 1.6%–6% of patients with this form of dialysis, whether they are adult or children. [2] It is more prevalence in female patients compared to males. [3] It tends to occur on the right side, [4] and develops usually within the first year

of starting PD.^[5] Later occurrence after 8 years of using PD has been reported as well.^[4]

The clinical manifestation of PD-associated hydrothorax is similar to symptoms associated with other etiologies of pleural effusion including dyspnea, nonproductive cough, and chest discomfort. Mild pleural effusion can be asymptomatic. However, large pleural effusion may cause severe symptoms at presentation. Symptoms onset after starting PD are suggestive of the disease process.^[6]

Pleural fluid analysis is indicative of transudative effusion. High glucose concentration in pleural fluid is diagnostic, hence the name "sweet hydrothorax," and difference of more than 50 mg/dL was found to have 100% specificity. In our case, the patient had no history of diabetes mellitus and the most recent HbA1c was 5.4%. However, the pleural glucose level was high compare to plasma glucose level. This is related to dextrose solution that is being used in PD, which

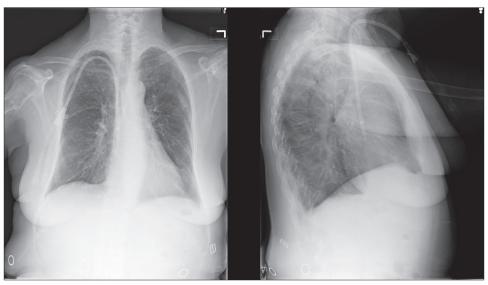


Figure 5: Chest x-ray, few weeks after starting hemodialysis, showed no pleural effusion. Perm catheter is in place

leaks into pleural space. Severe hyperglycemia in diabetic patients has been reported as a complication as well. [9]

Several imaging can be performed to confirm the diagnosis. To can be injected into peritoneal cavity to evaluate the leakage of dialysis fluid. Video-assisted thoracic surgery can be a diagnostic and therapeutic option for patients who prefers to stay on PD.^[5,10] Thoracic surgery to repair the defect or pleurodesis could be also considered in patients who failed conservative management.^[8]

We believe that the acute respiratory failure was due to large pleural effusion that is attributed to starting PD. This is supported by pleural fluid analysis and improvement of symptoms following pleurocentesis and hemodialysis. To study confirmed the diagnosis.

This case demonstrates the significance of sweet hydrothorax as a potentially life-threatening complication of PD in patients with peritoneo-pleural communication. Awareness of this condition and its imaging findings as well as the pleural fluid analysis are important for diagnosis and management.

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Conflicts of interest

There are no conflicts of interest.

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