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Case Report

Glucose Gradient Combined with Pleural Effusion Volume Observation in Pleuro-Peritoneal Communication: Presentation of Two Cases

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Abstract

Peritoneal Dialysis (PD) is an established effective renal Replacement Therapy (KRT) for patients with End-Stage Renal Disease (ESRD). PD accounts for 9% of all KRT and 11% of all dialysis worldwide. An uncommon but well-recognized complication is hydrothorax due to Pleuroperitoneal Communication (PPC) occurring in 1.6–10% of PD patients . An increased intra-abdominal pressure results in dialysate leaking across the diaphragm into the pleural space causing PPC. It is most commonly to be right-sided. Some PPCs appear rapidly. Some exist with long-term PD. Origin of PPC can be detected via contrast CT peritoneography and peritoneal scintigraphy using Technetium-99m tagged macro aggregated albumin or Tc-99m sulfur colloid. Thoracentesis is consistent with a transudative effusion with a high glucose concentration (50 mg/dL greater than simultaneous blood glucose). However, glucose measurement may be equivocal if prolonged effusion results in significant glucose absorption. Here we reported two cases of PPC verified by glucose gradient combined with pleural effusion volume observation, which might provide an easy, reliable, and cost-effective method for diagnosing PPC.

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Introduction

Peritoneal Dialysis (PD) is an established effective renal Replacement Therapy (KRT) for patients with End-Stage Renal Disease (ESRD). PD accounts for 9% of all KRT and 11% of all dialysis worldwide [1,2]. An uncommon but well-recognized complication is hydrothorax due to Pleuroperitoneal Communication (PPC) occurring in 1.6–10% of PD patients [3,4]. An increased intra-abdominal pressure results in dialysate leaking across the diaphragm into the pleural space causing PPC. It is most commonly to be right-sided [5]. Some PPCs appear rapidly. Some exist with long-term PD [6]. Origin of PPC can be detected via contrast CT peritoneography and peritoneal scintigraphy using Technetium-99m tagged macro aggregated albumin or Tc-99m sulfur colloid. Thoracentesis is consistent with a transudative effusion with a high glucose concentration (50 mg/dL greater than simultaneous blood glucose). However, glucose measurement may be equivocal if prolonged effusion results in significant glucose absorption [7]. Here we reported two cases of PPC verified by glucose gradient combined with pleural effusion volume observation which might provide an easy, reliable, and cost-effective method for diagnosing PPC.

Case Reports

Case 1

A 44-year-old man with ESRD due to Immunoglobulin A nephropathy (IgAN) presented with coughing for two months and was admitted to the inpatient department on July 22, 2022. He started on Continuous Ambulatory Peritoneal Dialysis (CAPD) five months before presentation. He had a history of hypertension and denied any history of abdominal trauma or surgery involving the diaphragm. Physical findings disclosed a blood pressure of 126/86 mmHg, a pulse rate of 72 beats/min, and a respiration rate of 20/min. His brain natriuretic peptide and acute infection index were regular. Chest examination revealed decreased breath on the right chest, without crackles or wheezing. Computerized Tomography (CT) showed new bilateral pleural effusion (Figure 1). Right thoracocentesis was performed and pleural fluid analysis showed clear yellow fluid with 43 white blood cells, mononuclear cells of 79.1%, and segmented cells of 20.9%. Fluid lactate dehydrogenase (LDH) was 24, Adenosine Deaminase (ADA) was 0.3u/l, glucose(GLU) was 9.74mmol/l (blood glucose was 4.55mmol/l at the same time), and protein was 2.5g/L. The effusion was transudative, without any malignant components, and the methylene blue test was performed as follows. Twenty mg of methylene blue was injected into the infusion of two L of 1.5% dextrose dialysate fluid which was planned to be dwell for six hours. However, the patient suffered from severe abdominal pain and vomited after two hours of dwelling so the methylene blue had to be stopped. We observed pleural effusion changes from thoracic closed drainage every two days. Hydrothorax increased rapidly after CAPD and resolved after discontinuing PD

(Figure 1). The patient's clinical symptoms resolved completely after discontinuing PD. Follow-up CT scan showed resolution of pleural effusion after the patient started hemodialysis (Figure 1).

Case 2

A 29-year-old male with progressive shortness of breath for two days was admitted to the inpatient department in March 2023. He was on CAPD for one month for ESRD due to nephritis ten years ago. He denied any surgery involving the diaphragm. Physical examination showed a blood pressure of 150/107 mmHg, a pulse rate of 98 beats/min, a respiratory rate of 20/min, and diminished breath sounds on the lower right lung field, without any crackles or wheezing. CT scan showed a new right pleural effusion. Right-sided thoracocentesis showed pleural fluid with 36 white blood cells, mononuclear cells of 72%, and segmented cells of 28%. Fluid lactate dehydrogenase (LDH) was 25, adenosine deaminase(ADA) was 0.4u/l, glucose(GLU) was 6.96mmol/l (blood glucose at the same time was 4.95 mmol/l), and protein was 3.4g/L. Observation of pleural effusion changes was performed as described in Case 1. After discontinuing PD and starting hemodialysis, symptoms resolved, and a follow-up CT scan showed a resolution of pleural effusion (Table 1).

	Glucose(mmol/l)			ALB(g/L)		
	effusion	serum	(PF/S)	effusion	serum	(PF/S)
Case1	9.74	4.55	2.14	2.5	35.3	0.07
case2	6.96	4.95	1.40	3.4	31.1	0.109

Table 1: Biochemical criterion of pleural fluid and serum.

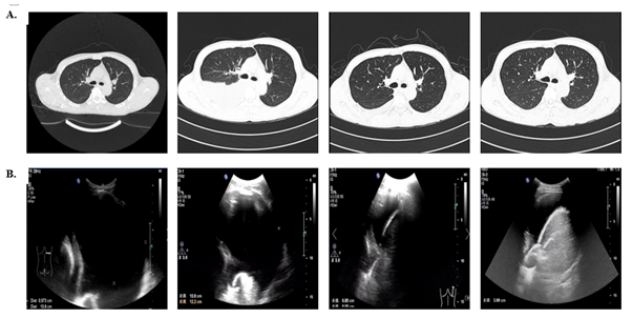

Figure 1: Changes of chest CT scan in before and after HD in case 1.

A. Chest CT scan indicated(from left to right) no pleural effusion before PD, massive right pleural effusion after five months of PD, small right pleural effusion after dry belly during night, no pleural effusion after three months of switching to HD

B. Chest color Doppler indicated(from left to right) massive right pleural effusion before thoracentesis, massive right pleural effusion after two days of thoracic drainage under CAPD, small pleural effusion after two days of thoracic drainage after dry belly during night, small pleural effusion after another two days of thoracic drainage after dry belly during night (Figure 2).

Liquid-based smears show lymphocytes, red blood cells, and a small amount of mesothelial cells, without any malignant components.

Discussion

PPC is one of the rare and serious complications in PD. Both of our patients finally switched to PD. Pleural effusion could occur when

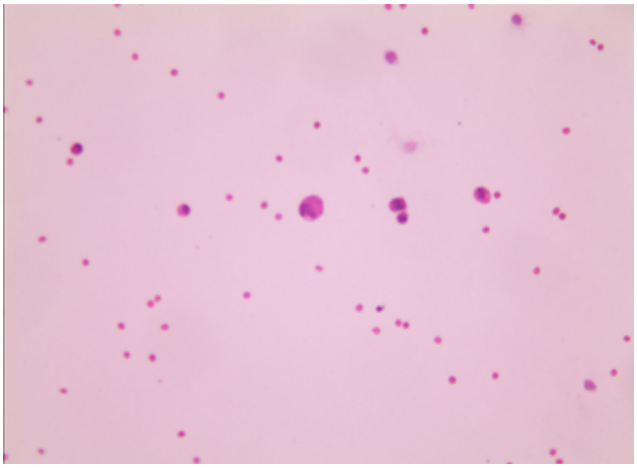

Figure 2: Pathology of pleural fluid.

the PD fluid enters the chest cavity through the thoracoabdominal fistula. For some patients, hydrothorax develops rapidly after PD initiation, and for others, it exists after long-term PD, suggesting an acquired manner. For the latter, PPC developed from attenuated tissue repeatedly exposed to raised intra-abdominal pressure. Most PPCs would emerge within one year. Case 1 in this article emerged after five months of PD treatment while case 2 developed pleural effusion within one month. Both of which developed within one year.

The mechanisms for hydrothorax development include congenital or acquired diaphragmatic defects, increased pleuroperitoneal pressure gradients, and lymphatic drainage disorders [8]. Some reports referred to young age, hernia, abdominal surgery, and peritonitis as risk factors of PPC [9]. Others also included trauma and polycystic kidney disease. Both of our patients had hypo-albuminemia, anemia, and low Body Weight Index (BWI), all of which may also be risk factors for PPC, which could contribute to the rupture of the weak diaphragm in the long-term high-pressure ladder. Their pleural fluid is all exudate (according to the Light standard), and the pleural fluid is grape-shaped. Pathology of pleural fluid in case one indicated lymphocytes, red blood cells, and a small amount of mesothelial cells.

The symptoms of PPC may be similar to congestive heart failure but will not be relieved by using hypertonic dialysate. Patients might present cough, chest tightness, shortness of breath, dyspnea. The two cases presented with cough and shortness of breath without edema. Their imaging and clinical biochemistry excluded acute infection or acute heart failure. Radiographic or ultrasound would most frequently indicate right-sided pleural fluid though it presents as left-sided or bilateral in some cases. Pleural effusions associated with PPC leaks are typically transudative with a low cell and protein content. All of these would remind us of PPC.

Available imaging methods for diagnosing PPC include intraperitoneal infusion of contrast material through the catheter during plain abdominal radiography and CT. These are time-consuming, require adequately trained personnel, and may be associated with complications related to intra-abdominal iodinated contrast media administration [10]. PPC can be detected via contrast CT peritoneography and peritoneal scintigraphy using Technetium-99m tagged macro-aggregated albumin or Tc-99m sulfur colloid. PD fluid containing labeled radioactive isotopes can diagnose chest and abdomen fistula, but this method has low sensitivity, only 40% to 50%, and is suitable

for medical settings. CT intraperitoneal biopsy Shadow can diagnose PPC and locate the fistula. However its sensitivity is only 33%, and patients with smaller diaphragmatic fistulas are prone to leakage diagnosis, and this technique may cause damage to residual kidney function using [11,12]. Television thoracoscopy technology allows for direct observation of the chest and diaphragm. A pathological condition of the device is affected, and it has the advantage of minimal trauma, but this method is effective for the device. High technical requirements and relatively high prices. Intraperitoneal methylene blue instillation followed by thoracentesis has been utilized in certain instances. However, there is a risk of chemical peritonitis. In case 1, the patient presented severe abdominal pain that led to shock syndromes. Although experimental PPC uses methylene blue test widely. The positive rate of the methylene blue test is low, and some patients may experience severe changes after using methylene blue. It is not recommended to use it in the clinic.

67% of PPC patients were with pleural fluid/serum glucose gradient greater than 100mg/dL (approximately 5.56mmol/L), 13% of PPC patients with a concentration gradient of 50-100mg/dL(between 2.78 and 5.56 mmol/L), while the remaining 20% of PPC patients with concentration gradient less than 50mg/dL (approximately 2.78 mmol/L) [13]. Glucose content is higher in pleural fluid than in serum. It is the so-called "sweet hydrothorax." Pleural fluid glucose concentration greater than that of serum (pleural fluid to serum glucose ratio >1) in a patient receiving PD is consistent with PPC [14,15]. Both of our cases were consistent with it. However, glucose measurement may be equivocal if prolonged effusion results in significant glucose absorption. Comparing glucose concentration in pleural fluid and blood during the same period is a simple-to-operate, cost-effective way to diagnose PPC. Combined sugar level test with volume change observation of pleural fluid by thoracentesis and drainage, through CAPD and IPD can be faster and more accurate for the diagnosis. The patient developed pleural effusion quickly after CAPD otherwise when dwelling during the night. This is a safe, sensitive, and economical way for diagnosis.

For treatment, temporary (2-4 weeks) PD cessation results in the resolution of pleural effusion, whereas refractory hydrothorax suggests a unidirectional communication. There have been occasional reports of successful management utilizing low-volume cycler PD regimens. Successful thoracoscopic pleurodesis (oxytetracycline, talc, autologous blood) has been reported. However, this invasive procedure is painful and with an increased risk of infection. Surgical repair of the diaphragmatic hernia should be for patients with visible diaphragmatic defects. The patient would be able to successfully transition back to PD after four weeks of postoperative intermittent hemodialysis. Some patients would switch to hemodialysis. Clinical significant symptoms relieved and pleural effusion disappeared quickly in our cases.

In conclusion, PPC is an uncommon but well-recognized complication in PD. Glucose gradient combined with pleural effusion volume observation could be a fast, accurate, simple, easy-to-implement, and cost-effective method for diagnosing PPC. PPC treatment is frequently unsuccessful. Nephrologists prescribing peritoneal dialysis should be aware of this complication.

Author's Contribution

Li-fang Yang and Wan-jun Lu contributed to patients managements; Jian-hong Gao contributed to data analysis. Corresponding author contributes to the design of the whole study and paper writing.

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Conflict of Interests

The author declares that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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