



BILATERAL PLEURAL EFFUSION IN CONTINUOUS AMBULATORY PERITONEAL DIALYSIS MANAGED BY VATS PLEURODESIS

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ABSTRACT

Pleuroperitoneal leak as a cause of pleural effusions in peritoneal dialysis is a rare but important complication to consider in continuous ambulatory peritoneal dialysis (CAPD) patients presenting with recurrent progressive dyspnoea. Generally, these effusions are unilateral and right-sided, resulting in shortness of breath and reduced ultrafiltration volume, which are initially managed by peritoneal rest. We describe a case of bilateral pleural effusions in a 57-year-old female on chronic CAPD who developed recurrent progressive dyspnoea but maintained adequate dialysis output. A chest radiograph revealed bilateral pleural effusions with high glucose content, and scintigraphy confirmed the existence of a definite pleuroperitoneal communication. She was managed by temporary substitution to haemodialysis, followed by suturing of the shunt and successful video-assisted thoracoscopic surgery (VATS) pleurodesis with an aldehyde-based surgical glue. Unexplained recurring dyspnoea in chronic CAPD should raise the suspicion of a possible pleuroperitoneal leak, even in patients without an apparent loss of ultrafiltration. Pleurodesis using an aldehyde-based adhesive was effective and tolerated well by our patient and may be considered in managing cases of recurrent pleural effusion.

KEYWORDS

Pleural effusion, pleuroperitoneal leak, peritoneal dialysis, pleurodesis, surgical adhesive

LEARNING POINTS

- Recurrent dyspnoea in a chronic peritoneal dialysis patient should raise the diagnosis of a possible pleuroperitoneal leak, even if no apparent loss of ultrafiltration was observed.
- Minimally invasive surgical pleurodesis using surgical adhesive can be considered in cases of refractory pleuroperitoneal leak.



INTRODUCTION

Peritoneal dialysis (PD) is accompanied with its own set of complications, either infectious or non-infectious, that are associated with technique failure. Pleural effusion due to pleuroperitoneal leak is a rare but well-recognised complication of PD, with a reported incidence ranging from 1.0%–6.0% in adult and 3% in paediatric patients on chronic continuous ambulatory PD (CAPD)^[1,2]. No definite eliciting factor can be identified in most cases; however, it is suggested that dialysate flows along a congenital or acquired anatomic communication between the pleura and peritoneal cavities, exacerbated by a rise in intra-abdominal pressure along with the negative intra-pleural pressure formed during spontaneous breathing^[1].

Cases of dialysate leak into the pleural cavity due to pleuroperitoneal communication (PPC) have been described previously^[3]. Typically, pleuroperitoneal leak manifests as unilateral 'sweet' pleural effusion that is right-sided in about 88% of cases, which is evident within 1 year following PD initiation. It results in shortness of breath and reduced ultrafiltration (UF) volume without signs of heart failure or peripheral oedema^[2]. The first line of treatment includes peritoneal rest, although pleurodesis may be necessary in recurrent cases^[1]. Here we describe an unusual case of bilateral pleural effusions in a patient on chronic CAPD with maintained UF successfully treated with pleurodesis using an aldehyde-based surgical adhesive.

CASE DESCRIPTION

A 57-year-old woman routinely undergoing CAPD was referred to our hospital with worsened shortness of breath and dry cough for 4 days. She denied any history of leg swelling. She had been diagnosed with end-stage kidney disease (ESKD) secondary to hypertension and had started CAPD 1 year prior to admission, regularly receiving Dianeal® 2.5% for 5–6 hours dwell time with a target dialysis dose of 1700 ml. Although previously asymptomatic, she had multiple complaints of dyspnoea due to pleural effusions 2 months after the commencement of CAPD, although no problems with peritoneal drainage were observed. At the time, no definite cause was identified, and she was treated

with an additional session of 4-hours haemodialysis (HD) per week in another hospital. CAPD was not discontinued and no alteration was made to her PD prescription. The patient reported that her symptom was only relieved when HD was provided. She denied any history of trauma to the chest.

On admission, the patient was weak and moderately hypoxic, with decreased breath sounds and dullness to percussion in the right lung and left lower lung base. She had no peripheral oedema. A chest X-ray showed massive right-sided hydrothorax with minimal left pleural effusion (Fig. 1A). Thoracentesis successfully drained 800 ml of serous fluid, and fluid analysis revealed a high level of glucose 261 mg/dl (serum glucose 143 mg/dl), protein 1.1 g/dl (serum protein 6.2 g/dl), lactate dehydrogenase (LDH) 105 U/l (serum LDH 707 U/l), all of which point to a transudative effusion. Pleural fluid pathology revealed no malignant cells. Laboratory examination showed no alarming abnormalities in liver and kidney function, as well as normal serum albumin level (4.1 g/dl). An echocardiogram excluded the possibility of acute decompensated heart failure. Scintigraphy with radioactive technetium sulfur colloid (^{99m}Tc)-pertechnetate demonstrated rapid uptake of contrast (within 5 minutes of contrast injection) in both pleural cavities at 60 min, confirming the presence of a definite pleuroperitoneal communication (Fig. 2).

CAPD was ceased temporarily, and the patient was transitioned into HD. Video-assisted thoracoscopic surgery (VATS) was performed, revealing a 1 × 2 mm circular tear at the posterolateral diaphragm, which was then sutured followed by chemical pleurodesis with an aldehyde-based surgical glue, BioGlue® (CryoLife Europa Ltd.) (Fig. 3). Approximately another 900 ml of pleural fluid was removed and the patient tolerated the procedure well. HD was successfully discontinued after 4 weeks, and CAPD was resumed with an initial daily volume of 1000 ml using alternating Dianeal 1.5% and 2.5%, which was gradually increased by 250 ml per week.

Subsequently, the patient tolerated CAPD without any complaints of dyspnoea and the follow-up chest X-ray after 2 months revealed resolution of effusions in both pleural spaces (Fig. 1B).

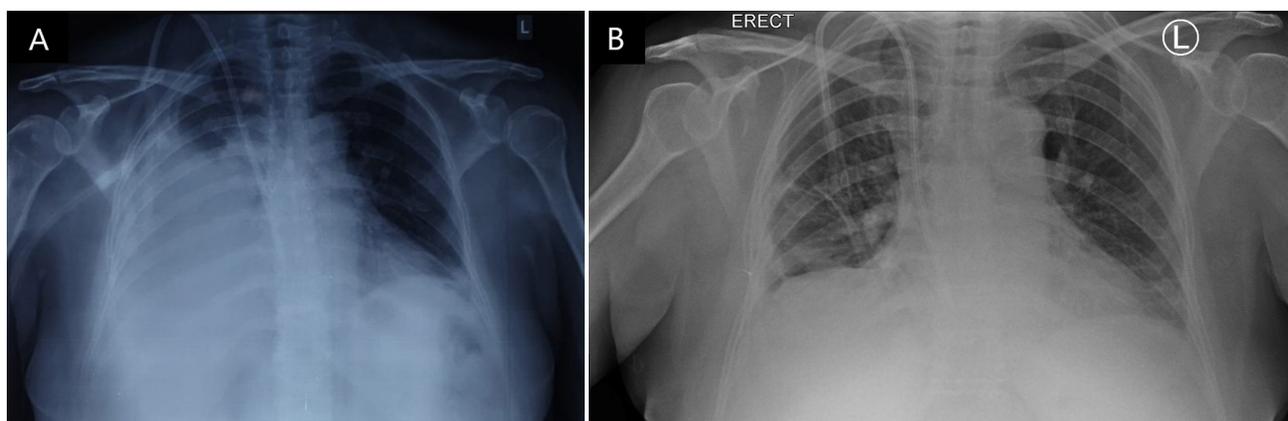


Figure 1. Resolution of pleural effusion as shown on chest X-ray. A) Chest X-ray on admission demonstrating massive right hydrothorax and minimal left pleural effusion, and B) follow-up chest X-ray 2 months postsurgical intervention.

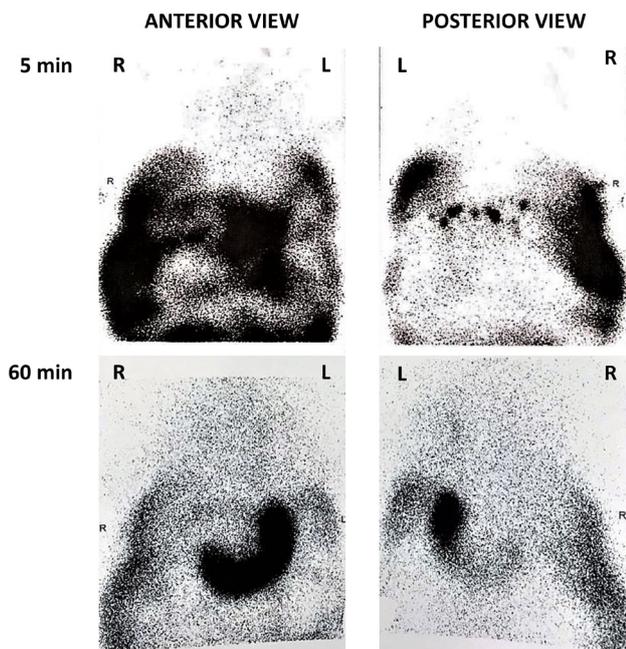


Figure 2. Peritoneal scintigraphy demonstrated extravasation of dialysate containing radioactive ^{99m}Tc -pertechnetate into bilateral pleural cavities.

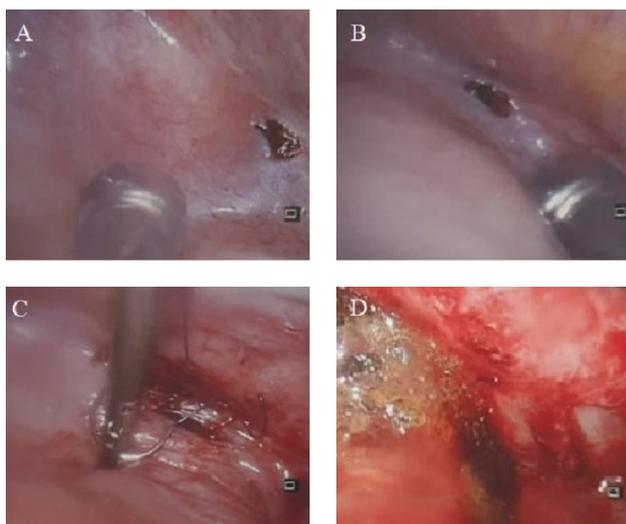


Figure 3. A) and B) VATS revealed a 1 × 2 mm circular tear at the posterolateral diaphragm, C) suture of the pleuroperitoneal communication, and D) pleurodesis with an aldehyde-based surgical glue.

DISCUSSION

We describe an unusual case of bilateral pleural effusions in a setting of chronic CAPD. The majority of the reported pleuroperitoneal leak cases were right-sided, while only about 4% of cases occurred bilaterally^[3]. A pleuroperitoneal leak should be suspected in non-oedematous PD patients presenting with unexplained dyspnoea accompanied by a sudden reduction in ultrafiltration volume or poor dialysis adequacy, although 25% of patients remain asymptomatic^[1,2]. The condition should be readily differentiated from other aetiologies of pleural effusion commonly associated with end-stage renal disease patients, including fluid overload, congestive heart failure, hypoalbuminemia, pneumonia and

other systemic infections. In this case, despite the recurrent dyspnoea, our patient maintained adequate dialysate output since the onset of symptoms, which possibly led to the missed early suspicion of a possible PPC.

No diagnostic methods for PPC are considered a gold standard. An initial diagnosis can be made by thoracocentesis with fluid analysis consistent with peritoneal dialysate, a transudate with a high pleural glucose content^[4], as seen in our patient. It was suggested that effusion with a pleural fluid-to-serum (PF-S) glucose gradient exceeding 50 mg/dL is highly specific of a leak from a PPC^[5]. Furthermore, the finding of transudative fluid should also be differentiated from other causes such as congestive heart failure, cirrhosis, hypoalbuminemia, or nephrotic syndrome, which were not observed in the described patient. Alternatively, PF-S glucose ratio >1.0 in circumstances where the gradient is <50 mg/dl is consistent and more sensitive to PPC, whereas other causes of transudative and exudative effusions may produce a ratio of <1 and <0.5, respectively^[4]. Other diagnostic methods such as CT peritoneography have also been implemented in locating the position of the leakage. However, this method is useful in cases of large PPC, but lacking sensitivity (33%) in detecting smaller pleuroperitoneal leaks^[6]. Peritoneal scintigraphy using radioactive isotope technetium offers visualisation of the free flow of dialysate, even in small and slow leaks, as well as assessing the location and size of the anatomic defect. In one study, this method was effectively capable of confirming the presence of a PPC in 70% of cases within 30 minutes following contrast injection^[6].

There is no specific guideline available in managing hydrothorax complicating CAPD. Experts suggest that initial management should begin with temporary cessation of PD for 2 to 6 weeks^[1]. Although most patients may require permanent transition to HD, Chow et al.^[1] reported spontaneous closure of the diaphragmatic defect in approximately 50% of cases, allowing successful reinstitution of CAPD. In recurrent cases where the conservative approach fails, mechanical (direct mechanical abrasion) or chemical pleurodesis is the acceptable next step^[1]. Successful cases of chemical pleurodesis using povidone-iodine, tetracycline, talc, and autologous blood have been reported. However, there is currently not enough data to suggest which sclerosing agent is superior to the other and the choice of sclerosants should depend on availability, cost, and potential side effects.

Surgical management of PPC has a reported success rate approaching 100% in resuming long-term PD, especially when a specific defect has been confirmed^[7]. Common options for pleural closure include the use of non- or absorbable sutures, fibrin glue or surgical mesh. Minimally invasive videothoracoscopy (VATS) eliminates the need for thoracotomy and has become a popular diagnostic and therapeutic procedure in managing PPC, as it allows direct visualisation and repair of small diaphragmatic defects. VATS-assisted pleurodesis was found to be safe and highly effective in preventing recurrence of CAPD-associated

pleuroperitoneal leak with an over 80% success rate compared to conventional pleurodesis^[7].

We utilised a surgical adhesive, BioGlue[®], containing 45% purified bovine serum albumin and 10% glutaraldehyde that spontaneously produces a stable and rigid but flexible sealant after the two components bind to each other^[8]. BioGlue[®] has the advantage of being biocompatible and safe to use in many cardiothoracic surgeries^[9]. Potaris et al.^[8] have successfully utilised BioGlue[®] sealant to seal air leaks from pulmonary parenchyma and bronchopleural fistulas in 38 patients with no observed empyema or any complications related to its toxicity. Several studies showed that BioGlue[®] was associated with greater adhesive strength and burst pressure than fibrin sealant in managing a pleural defect^[10]. A clinical trial also demonstrated the clear benefit of using BioGlue[®] compared to conventional surgical treatment in treating alveolar air leaks in terms of duration of air leak, drain removal time and hospital stay; however, no difference in efficacy was found between BioGlue[®] and fibrin glue^[11].

To our knowledge, management of a pleuroperitoneal leak using a biocompatible aldehyde-based surgical glue has not been reported. Further studies need to be conducted to determine its efficacy and safety in sealing PPC in PD patients. Our patient was managed by temporary cessation of PD followed by VATS-assisted surgical suture and BioGlue pleurodesis. She successfully recommenced CAPD after 4 weeks with a gradual increase in inflow volumes, and showed resolution of effusion without any recurrence of dyspnoea.

CONCLUSION

This case emphasises the importance of considering a pleuroperitoneal leak as a differential diagnosis in chronic PD patients presenting with recurrent dyspnoea due to pleural effusion with no evident cause, even in those without an apparent loss of UF. Management by pleurodesis with an aldehyde-based surgical adhesive is a safe and suitable option to consider in cases of refractory PPC.

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