

〈Case Report〉

A case of thoracoscopic repair for pleuroperitoneal communication in a patient undergoing continuous ambulatory peritoneal dialysis

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ABSTRACT Pleuroperitoneal communication (PPC) has been reported as a potential complication of continuous ambulatory peritoneal dialysis (CAPD). It is a rare condition associated with CAPD and there is no established standard treatment. Once PPC develops, CAPD cannot be continued in approximately half of the patients, who would then need to be switched to hemodialysis. We present the case of a patient on CAPD who presented with PPC; we repaired the diaphragmatic defect by thoracoscopic surgery and CAPD could be continued for 31 months after the surgery.

We discuss a potentially effective strategy for PPC syndrome with a review of the literature.

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Key words : Pleuroperitoneal communication (PPC), Continuous ambulatory peritoneal dialysis (CAPD), Thoracoscopic surgery

BACKGROUND

PPC with massive hydrothorax is one of the significant complications of continuous ambulatory peritoneal dialysis (CAPD) and no standard treatment strategy has been established. PPC is estimated to occur in approximately 1.6 to 2.0% of all patients undergoing CAPD; approximately 50% of the patients who develop PPC require switching to hemodialysis^{1, 2)}. Therefore, prompt diagnosis and treatment are necessary to prevent conversion to hemodialysis as much as possible. In this article, we report a case of PPC that was treated by thoracoscopic repair. Diaphragm repair

by thoracoscopic surgery is minimally invasive and is considered as an effective surgical procedure for PPC that would allow continuation of CAPD treatment.

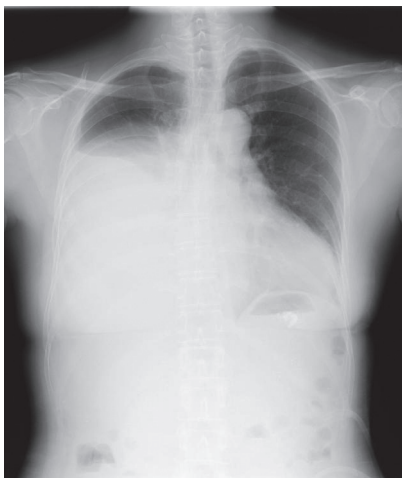
Case Description

A 54-year-old woman with chronic renal failure due to chronic glomerulonephritis was referred to our hospital complaining of breathlessness four days after she was initiated on CAPD. A plain chest X-ray revealed a massive right-sided pleural effusion (Fig.1a). Laboratory examination on admission revealed an increase of the serum uric

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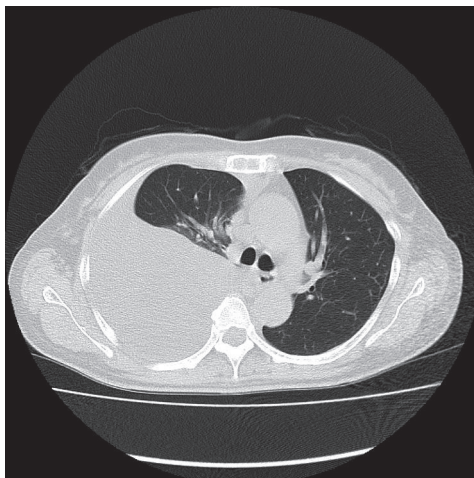


Fig. 1. A plain chest X-ray showing massive right pleural effusion (a). Chest computed tomographic (CT) image showing massive right pleural effusion, atelectasis of the right middle and lower lobes, and mediastinal shift to the left (b).

Table 1. Laboratory Data on Admission

Complete blood count		Blood biochemistry test			
WBC	5,090 / μ L	TP	6.7 g/dL	Na	140 mEq/L
RBC	369 $\times 10^4$ / μ L	Alb	3.8 g/dL	K	5.2 mEq/L
Hb	10.1 g/dL	T-Bil	0.4 mg/dL	Cl	103 mEq/L
Hct	31.6 %	AST	15 IU/L		
Plt	32.7 $\times 10^4$ / μ L	ALT	3 IU/L	Pleural effusion	
		ALP	186 IU/L	Color	light yellow
		LDH	253 IU/L	Specific gravity	1.016
		γ -GTP	15 IU/L	TP	< 0.3 g/dL
		CRE	8.15 mg/dL	Alb	< 0.3 g/dL
		UN	41 mg/dL	LDH	14 U/dL
		UA	8.4 mg/dL	Glu	204 mg/dL
		Glu	75 mg/dL	ADA	< 2.0 U/L
		CRP	0.32 mg/dL		

acid level, renal function impairment, and anemia. However, the white blood cell count and serum C-reactive protein level were normal. Examination of a pleural fluid specimen obtained by thoracentesis revealed a higher pleural fluid glucose level than the blood glucose level (Table 1). Chest computed tomography (CT) revealed atelectasis of the right lower and middle lobes and mediastinal shift to the left due to the massive right pleural effusion (Fig.1b). The protein and LDH levels in the pleural fluid were low, suggesting a transudative pleural effusion. Since physical examination revealed no obvious leg

edema or distended jugular veins and the chest CT showed no obvious pulmonary edema or cardiac dilatation, we ruled out heart failure. Blood test results showed no evidence of an inflammatory reaction and the pleural effusion was not exudative, so that we ruled out bacterial infection as the cause of the pleural effusion. Furthermore, we excluded the possibility of pleural effusion associated with hypoalbuminemia as the pleural effusion was unilateral.

PPC was suspected based on the findings including (1) a massive right-sided pleural effusion after the

initiation of CAPD, (2) the pleural fluid glucose level being higher than the blood glucose level, and (3) the drainage volume being lower than the injected volume of the CAPD solution. Therefore, we scheduled thoracoscopic repair. Under general anesthesia and one-lung ventilation, with the patient in the left lateral decubitus position, three ports were inserted into the 7th intercostal space on the anterior axillary line and posterior axillary line and the 8th intercostal space on the posterior axillary line. No adhesions in the pleural cavity or pleural thickening were observed. CAPD solution stained with indigo carmine dye was injected into the peritoneal cavity via the peritoneal dialysis catheter, and within a short while, several cystic lesions at the center of the right diaphragm became stained in blue (Fig.2). The blue-stained CAPD solution was found to flow into the thoracic cavity through some of these cystic lesions. The cystic lesions in the diaphragm were resected with a thoracoscopic stapler, followed by covering of the staple lines with a polyglycolic acid (PGA) sheet and fibrin glue. Histopathologic examination of the resected diaphragm revealed a muscle tear that was contiguous with only peritoneal tissue at that site, suggesting that there could be a fistula at the site. The patient resumed CAPD on the fourth postoperative day and there was no recurrence of the hydrothorax. The patient was

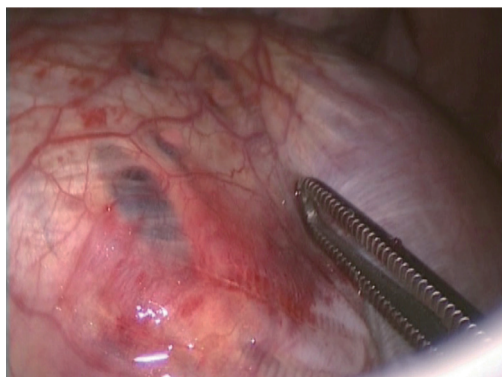


Fig. 2. Some of the cystic lesions were identified at the center of the diaphragm.

able to continue CAPD until the 31st postoperative month.

DISCUSSION

PPC is a condition in which fluid and blood flow between the thoracic and abdominal cavities through small holes in the diaphragm. It is often observed in patients who are undergoing CAPD, or have liver cirrhosis or ascites associated with malignancy. PPC is estimated to occur in 1.6 to 2.0% of patients undergoing CAPD. It is considered as a serious complication, as about a half of all the patients who develop PPC during CAPD need to be switched to hemodialysis¹⁻³).

The precise pathogenetic mechanism of PPC in patients undergoing CAPD remains unclear until date. Several mechanisms have been speculated, including (1) development of defects of the diaphragm due to abnormal intraperitoneal pressures; (2) presence of congenital or traumatic diaphragmatic defects; (3) development of defects or laceration of blebs in the fragile region of the diaphragm; and (4) migration of fluid through lymphatic vessels^{2, 4}).

Since anatomically, ascitic fluid tends to accumulate first under the diaphragm on the right side, PPC often occurs on the right side. In patients undergoing CAPD who present with right-sided pleural effusion, PPC may be suspected if the pleural fluid glucose level is higher than the blood glucose level⁵). Diagnosis is made by confirming passage of the injected CAPD solution into the thoracic cavity using a dye, contrast medium, or radioactive isotope^{3, 6-8}). In our case, the pleural fluid glucose level was 204 mg/dl, higher than the blood glucose level, and it was confirmed intraoperatively that CAPD solution stained with indigo carmine dye leaked into the right thoracic cavity. Reported treatments for PPC include reduction of the injected amount of CAPD solution, temporary hemodialysis, water control with a diuretic,

pleurodesis, and surgical treatment^{1, 3, 4, 6-10}. Pleurodesis is reported to have a success rate of only 41%-54%^{1, 2, 11}. Furthermore, pleurodesis could also lead to peritoneal fibrosis if the injected agent flows into the peritoneal cavity through the diaphragmatic defects⁷.

As surgical procedures to treat PPC, resection, direct suturing, and reinforcement of the responsible lesions in the diaphragm have been performed, either singly or in combination. Sakamoto *et al.* reviewed the data of 53 cases of thoracoscopic surgery for PPC reported since 2000. They found that about 80% of patients who underwent partial resection or suturing of diaphragm also underwent diaphragmatic reinforcement or pleurodesis simultaneously, and in 70% of these cases, the diaphragm was reinforced with a PGA sheet and fibrin glue¹¹. In the current case, we selected to resect the lesions with a thoracoscopic stapler and reinforce the staple lines with a PGA sheet and fibrin glue, as in many of the cases reported in the literature.

The success rate of thoracoscopic repair has been reported to be over 80%, because the path of communication can be directly identified^{3, 5, 7}. On the other hand, success rates in cases where the route of communication is unclear are reported to be in the range of 38%-44%^{3, 11} indicating the importance of confirming the site of communication. The site of communication can be easily identified using a dye or by inducing pneumoperitoneum^{3, 5, 6, 7, 9, 11}. In the case of an identifiable lesion in the diaphragm, ligation or stapling is possible. However, there are some reports of development of lacerations at the resection margins when using a thoracoscopic stapler¹².

Cerfolio and Bryant reported an 80% success rate in 33 of the 41 patients who underwent thoracoscopic pleurodesis with talc for PPC due to refractory hepatic ascites, and the communication path could be identified in only 5 cases¹³. This

indicates that talc pleurodesis is an effective option for patients with unidentified diaphragmatic lesions. In Japan, however, the indications for talc are limited to malignant pleural effusion and refractory pneumothorax, which poses a challenge to its practical application for PPC.

The majority of patients require long-term CAPD therapy due to social considerations; if the residual renal function declines during CAPD, the dialysate volume must be increased. Increase in the intraperitoneal pressure arising from this increase in dialysate volume is a significant risk factor for PPC recurrence. In this case, the patient showed no recurrence and could continue CAPD for 31 months. Eventually, the patient had to be initiated on hemodialysis because of declining renal function and decreased urine output, as well as due to the repeated episodes of CAPD-related peritonitis. In any case, thoracoscopic repair proved beneficial to extend the time to hemodialysis. In previous reports, PGA sheets, fibrin glue, pedicle muscle flap, pericardial fat pad tissue and pleurodesis have been used to reinforce the diaphragm^{11, 13-15}. To prevent recurrence, reinforcement procedures should be determined in each case.

CONCLUSION

Thoracoscopic surgery proved to be an efficacious method for treating PPC in our case. In addition to resection of the defects, the responsible lesions in the diaphragm were reinforced to prevent recurrence of hydrothorax.

CONFLICTS OF INTERESTS

The authors state that they have no conflicts of interests to declare.

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