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CASE REPORT

Optimal imaging conditions for the diagnosis of pleuroperitoneal communication

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Accepted 20 March 2019

SUMMARY

A 70-year-old woman with end-stage renal disease caused by a polycystic kidney disease developed massive right-sided pleural effusion 10 days after the initiation of peritoneal dialysis (PD). Although pleuroperitoneal communication (PPC) was suspected, computed tomographic peritoneography on usual breath holding did not show leakage. Therefore, we instructed her to strain with maximal breathing, which caused a jet of contrast material to stream from the peritoneal cavity into the right pleural cavity and allowed the identification of the exact site of the diaphragm defect. Following the thoracoscopic closure of the defect, she was discharged without recurrence of hydrothorax on PD. Hydrothorax due to PPC is a rare complication of PD. Notably, numerous previous modalities used to diagnose PPC lack sufficient sensitivity. Thus, an approach to spread the pressure gradient between the peritoneal cavity and the pleural cavity on imaging may improve this insufficient sensitivity.

BACKGROUND

Peritoneal dialysis (PD) is an established renal replacement therapy for patients with end-stage renal disease. With the global burden of chronic kidney disease, this therapy, which is more cost-effective than haemodialysis, has become indispensable in renal replacement therapy in many countries.[1] The annual global growth rate of PD is estimated to be 8%, which is higher than that of haemodialysis.[2] However, there are many complications of PD, one of which is pleuroperitoneal communication (PPC), which causes an exacerbation of respiratory distress and transition to haemodialysis. Regrettably, there is still no examination for PPC with a sufficient sensitivity, and clinicians often face difficulties while diagnosing PPC. Here, we report the case of a woman who developed hydrothorax to PPC after the initiation of PD. Our approach was to increase the pressure gradient between the peritoneal cavity and the pleural cavity on CT peritoneography, which was useful for diagnosis.

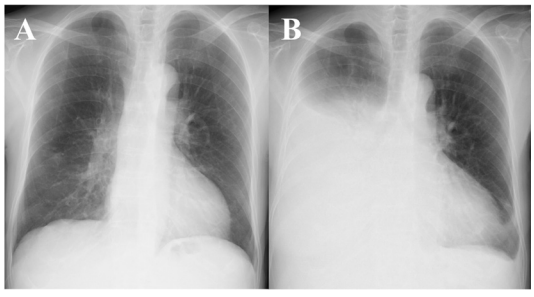

Figure 1 Chest radiography. (A) There was no pleural effusion before the initiation of peritoneal dialysis (PD). (B) A massive right pleural effusion shifting the mediastinum was detected 10 days after the initiation of PD.

CASE PRESENTATION

A 70-year-old woman with end-stage renal disease due to polycystic kidney disease (PKD) opted to undergo PD. Ten days after the initiation of automated PD consisting of 30 mL/kg peritoneal dialysate infusion three times a night, she suddenly complained of dyspnoea. Her blood pressure was 147/78 mm Hg, heart rate was 92 beats/min, temperature was 36.8°C, respiratory rate was 24/min and percutaneous oxygen saturation in room air was 88%. Her heart exam revealed normal S1 and S2 with no murmur, and her chest examination revealed attenuated breath sounds in addition to slight coarse crackle in the right lung field. The results of blood examination were as follows: white blood cell count=6.3×10^9/L, haemoglobin=112 g/L, platelet count=29.4×10^9/L, serum sodium=141 mmol/L, serum potassium=3.9 mmol/L, serum urea nitrogen=55.7 mg/dL, serum creatinine=6.64 mg/dL, serum albumin=3.4 g/dL, serum protein=6.6 g/dL, creatine kinase=64 IU/L, brain natriuretic peptide=113.8 pg/mL and C reactive protein<0.1 mg/dL. Electrocardiography revealed a normal sinus rhythm without ST-T segment abnormality. Chest X-ray radiography revealed massive right-sided pleural effusion (figure 1). At this point, she was suspected to have PPC. However, CT peritoneography using 40 mL/kg peritoneal dialysate mixed with 1.5 mL/kg contrast material on usual breath holding showed no leakage. Therefore, the phase of imaging was changed from the usual inspiratory level to the maximal inspiratory level. Accordingly, the patient was instructed to strain as if defecating, which resulted in the identification of a jet of contrast material from the peritoneal cavity flowing into the right pleural cavity and the exact site of the diaphragm defect (figure 2), which led to an accurate diagnosis of PPC. Due to the fact that PD during the daytime restart with 20 mL/kg peritoneal dialysate per session after the cessation of PD to improve her respiratory condition had induced hydrothorax again, she was expected to undergo a thoracoscopic surgery to continue PD. We could not identify the defect, although, we investigated

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To cite: Nakayama T, Hashimoto K, Kiriyama T, *et al*. *BMJ Case Rep* 2019;**12**:e228940. doi:10.1136/bcr-2018-228940

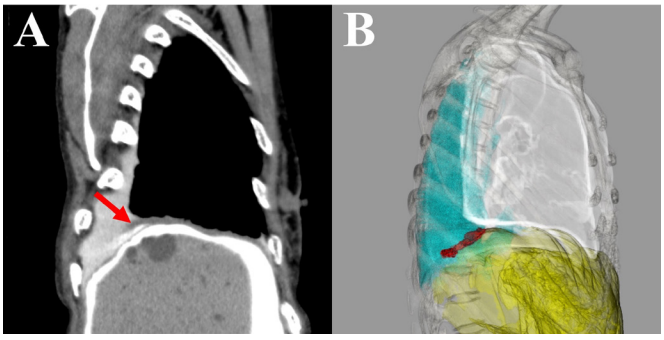

Figure 2 CT peritoneography. (A) Sagittal view showing a jet of contrast material (red arrow). (B) A three-dimensional reconstructed image showing the whole image with a jet of contrast material (red linear mass).

the diaphragm thoroughly. Accordingly, using CO_2 pneumoperitoneum through a PD catheter, we attempted to identify the exact defect (figure 3). There was no air leakage from the defect measuring <5 mm in diameter until the abdominal pressure was sufficiently increased with the CO_2 pneumoperitoneum, and then closure of the defect and pleurodesis were performed.

OUTCOME AND FOLLOW-UP

After the surgery, the patient had an uneventful course for more than 5 months after hospital discharge without hydrothorax recurrence on the originally planned PD.

DISCUSSION

There are many causes of hydrothorax in patients on PD, including PPC in addition to heart failure, hypoalbuminaemia, fluid overload, infections, malignancies and immune disorders. Hydrothorax due to PPC occurs in approximately 1.6%–10% of all patients on PD, mostly within ≤30 days of initiating PD and on the right side.[3–5] The cause of PPC is thought to be acquired diaphragmatic defects because of the elevated intra-abdominal pressure from the peritoneal dialysate infusion, congenital diaphragmatic defects or lymph disorders.[4 6] PKD may be a risk factor for hydrothorax associated with PPC because the intra-abdominal pressure of patients with PKD with large renal or hepatic cysts may be higher than that of patients without PKD, and the collagen in patients with PKD may be abnormal.[7 8] Our patient with PKD developed symptomatic hydrothorax on the right side 10 days after the initiation of PD. PPC should be considered in the differential diagnosis of hydrothorax in patients on PD (especially just after the start of PD) on the right side or with PKD because delayed diagnoses can exacerbate respiratory distress and transition to haemodialysis.

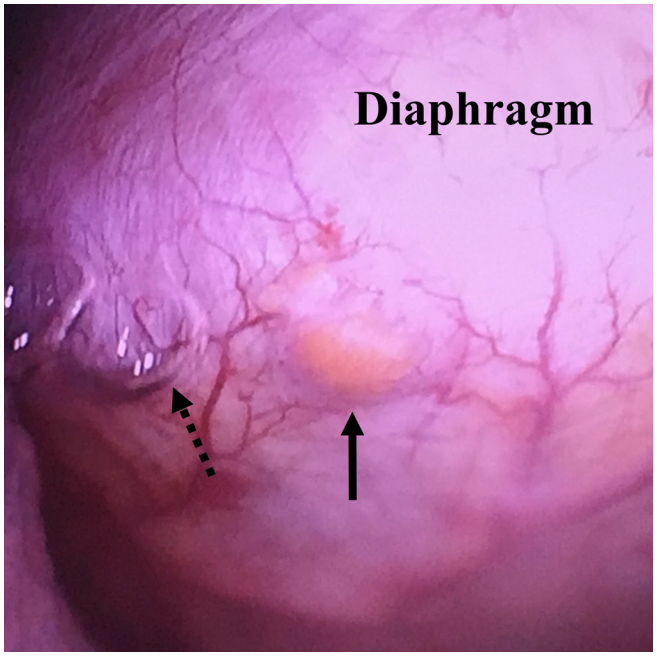

Figure 3 Thoracoscopy showing CO_2 leakage (dotted black arrow) and the diaphragm defect (solid black arrow).

No diagnostic methods for PPC, which mainly consist of image inspection and analysis of the pleural fluid, are recognised as a gold standard. Several imaging modalities, including peritoneal scintigraphy with technetium-99m-labelled macroaggregated albumin, CT, MRI and single-photon emission CT (SPECT), have been implemented for the diagnosis of PPC, but all of them lack the sufficient sensitivity. The sensitivity of CT and MRI peritoneography is approximately 30%, and even peritoneal scintigraphy without showing the exact defect has a sensitivity of 50%.[9 10] Because SPECT has a short history, accumulating more cases is needed to verify its sensitivity.[11] Analysis of pleural fluid to serum glucose gradients also has been used, but it has many problems, such as the absence of a consensus criterion value.[12]

We proposed an approach to increase the pressure gradient between the peritoneal cavity and the pleural cavity on imaging, so that the insufficient sensitivities of the imaging modalities for the diagnosis of PPC could be improved. To the best of our knowledge, there has been no report regarding pressure gradient on imaging for the diagnosis of PPC. As mentioned above, PPC is known to cause the migration of the peritoneal dialysate from the peritoneal cavity into the pleural cavity, leading to hydrothorax. We believe that this migration occurs only when the pressure gradient exceeds a certain value that may depend on the defect size. In our case, thoracoscopy could not demonstrate air leakage until the patient's abdominal pressure was sufficiently increased with the CO_2 pneumoperitoneum through a PD catheter. Judging from this finding, our hypothesis seems to be reasonable. The abdominal pressure reached approximately 230 mm Hg on straining, which is extremely high compared with the pressure of 0.5–1.5 mm Hg before the infusion of the peritoneal dialysate or the 5 mm Hg after the infusion of 15–25 mL/kg peritoneal dialysate, which suggests that migration is likely to occur on straining during defecation or during exercise.[13 14] Therefore, we hypothesise that, in some cases, the insufficient sensitivities of imaging modalities for PPC can be attributed to the pressure gradient on imaging not reaching a value necessary to induce leakage, and an approach that increases the pressure gradient on imaging potentially can minimise the problem. In fact, CT peritoneography did not demonstrate our patient's leakage until she breathed maximally and strained to increase the pressure gradient. Considering that all people, including patients on PD, strain in daily life, we believe that these approaches are non-invasive and have a low risk. Thoracoscopic surgery has an overall success rate of appropriately 70%; however, the success rate increases when the defect is confirmed.[15 16] In a previous study, almost 40% of the patients with PPC elected to permanently transfer to HD without receiving the surgery.[17] Therefore, it is very important that the defect is confirmed before the surgery, which may have affect patients' choice of future dialysis therapy.

PPC associated with PD should be promptly diagnosed because delayed diagnosis can exacerbate respiratory distress

and transition to haemodialysis, but it is not always easy because of the lack of imaging test sensitivity or the presence of many other possible diagnoses. Our novel approach to increase the pressure gradient between the peritoneal cavity and the pleural cavity on imaging may be useful for improving this insufficient sensitivity in a conventional and non-invasive manner.

Patient's perspective

When I had a hard time breathing after the unfamiliar start of peritoneal dialysis, I felt uneasy and even thought of discontinuing PD. However, the prompt diagnosis of the cause relieved my anxiety and encouraged me to have the operation. If computed tomographic peritoneography had not led to a definite diagnosis, I would opt to transition to haemodialysis without a thoracoscopy for further evaluation.

Learning points

- Clinicians should recognise that dyspnoea in patients on peritoneal dialysis rarely results from pleuroperitoneal communication (PPC).
- Previously used imaging methods for PPC have not achieved a sufficient sensitivity partially because of the low-pressure gradient between the peritoneal cavity and the pleural cavity.
- Approaches to increase the gradient, such as breathing maximally and straining on imaging, may increase the imaging sensitivity for the diagnosis of PPC.

Contributors All authors were involved in the patient's clinical care and approved the final manuscript. TN and KHa were involved in designing the study, collecting the data and writing the manuscript. TK was involved in designing the study and collecting the data. KHi was involved in collecting the data and revising the manuscript.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent for publication Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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