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Pleuroperitoneal communication after bacterial peritonitis and total gastrectomy for *gastric neuroendocrine tumors*: a case report and brief literature review

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Abstract

Background: Peritoneal dialysis (PD) is associated with various complications, some of which may result in its discontinuation. Pleuroperitoneal communication (PPC) is commonly recognized by the presence of a diaphragmatic defect and pressure elevation in the abdominal cavity due to the dialysate. PPC is unpredictable and its presence prevents the continuation of PD. We present the clinical course and pathological findings of PPC in a PD patient after bacterial peritonitis and total gastrectomy for gastric neuroendocrine tumors. We provide a brief review of PD-related complications that develop due to a non-infectious pathology, including those related to catheter use and an elevated intra-abdominal pressure.

Case presentation: A 65-year-old Japanese man, who had been receiving PD treatment for 1 year, visited our hospital owing to a cloudy dialysate. Bacteria were detected in the dialysate. He had been previously diagnosed with gastric neuroendocrine tumors and gastrectomy had been planned. On admission, we started a 14-day antibiotic treatment for PD-related peritonitis. The patient showed a good clinical course. Gastrectomy was performed as planned, and the postoperative course was uneventful. During the perioperative period, PD was temporally changed to hemodialysis. Five weeks after the gastrectomy, PD treatment was resumed with gradual increase in the exchange volume. After returning to PD overnight, using an automated peritoneal dialysis machine, the patient complained of breathing difficulty and he gained weight. Right-sided pleural effusion was observed on a chest radiograph, and PPC was confirmed by scintigraphy when a mixture of technetium-99m and dialysate was seen entering the right hemithorax within 120 min. The patient did not consent to surgery for the PPC and he hoped to continue to receive PD treatment conservatively. We advised the patient to undergo dialysate exchange in a semi-seated position, and he was prohibited from lying down during the daytime. He continued PD treatment without signs of pleural effusion and over-volume.

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Conclusions: This case of PPC occurring after bacterial peritonitis and total gastrectomy for gastric neuroendocrine tumors in a PD patient demonstrates the necessity of recognizing the PPC pathology in PD management and establishing methods for preventing PPC development after bacterial peritonitis or surgical procedures.

Keywords: Peritoneal dialysis, Pleuroperitoneal communication, Bacterial peritonitis, Gastrectomy, Non-infectious PD-related complications

Background

Peritoneal dialysis (PD) has various complications that occasionally result in its discontinuation. Pleuroperitoneal communication (PPC) is a pathology that is commonly identified by the presence of a diaphragmatic defect and pressure elevation in the abdominal cavity caused by ascites due to liver cirrhosis or peritoneal dialysate; the latter causes iatrogenic ascites, which has been found to trigger PPC [1, 2]. Patients with pleural effusion induced by PPC commonly present with coughs and dyspnea because the chest cavity is narrower than the abdominal cavity. Therefore, PPC requires appropriate strategies for treatment, especially in renal replacement therapy, to enable the continuation of PD for as long as possible. Previous case reports showed a 50% success rate of PD continuation with conservative adjustments [3, 4], such as PD volume and schedule adjustments, or when PD was temporarily combined with hemodialysis. Treatment options for PPC include chemical pleurodesis [4] or surgical diaphragmatic repair via video-assisted thoracoscopy (VATS). Being female and having autosomal dominant polycystic kidney disease (ADPKD) were identified as risk factors for PPC, with ADPKD—one of the factors related to increasing intra-abdominal pressure—being associated with PPC onset [5, 6]. However, some reports argue against the involvement of these factors; consequently, there is currently no consensus regarding the associated etiological factors [7, 8].

We present a case of a patient who developed PPC 1 year after PD initiation. PPC was suspected to have been caused by bacterial peritonitis and total gastrectomy, which were performed for the treatment of gastric neuroendocrine tumors. Moreover, we provide a brief review of PD-related complications that develop due to a non-infectious pathology, including those related to catheter use and elevated intra-abdominal pressure.

Case presentation

A 65-year-old man with end-stage diabetic kidney disease who was receiving PD was scheduled for a gastrectomy to treat gastric neuroendocrine tumors. In the perioperative period, we planned to suspend PD and switch to hemodialysis temporarily as renal replacement therapy for 1 month after gastrectomy. The PD prescription for the patient consisted of three exchanges per day of 2.5% dextrose-based dialysate overnight using an automated PD machine.

One month before the operation, the patient experienced PD-related infectious peritonitis, which required hospitalization for treatment. On admission, he had no specific complaints of fever or abdominal pain. He had a cloudy dialysate and a leukocyte count of 600/mm^3 in effluent microscopic examination. Although there was no melena, a remarkable decease in hemoglobin level from 11.0 to 6.2 g/dL, without iron deficiency, was noted (transferrin saturation, 31.5%; serum ferritin level, 47.0 ng/dL). The hemoglobin decrease was suspected to have been caused by bleeding from the gastric tumor. The following inflammatory markers were within the normal limits: white blood cell count (3830/μL) and C-reactive protein level (0.06 mg/dL). Additionally, *Staphylococcus epidermidis* and *Staphylococcus capitis* were detected in a PD effluent culture submitted on the day the patient was hospitalized. The administration of antibiotics was started and red blood cell transfusion was performed, and the surgery was rescheduled because it was considered that the patient may not survive the peritonitis. Consequently, re-culturing of the PD effluent showed negative results, and leukocytes were absent in the effluent. Subsequently, the surgery was performed as planned. The operation was completed successfully, and the postoperative course was good. After the gastrectomy, the patient was temporarily shifted to hemodialysis for 1 month and his condition stabilized. After 5 weeks, we restarted PD with a small initial volume and infrequent exchanges of dialysate during the day. We gradually increased the regimen to the previous prescription using the automated PD machine at night. Although we performed PD steadily during the day without symptoms, we noted a lower dialysate volume than expected and the patient complained of slight dyspnea. He also gained weight from the first day PD was administered at night. A chest radiograph revealed reduced permeability in the right lung field, suggesting right-sided pleural effusion (Fig. 1a). To confirm the presence of a peritoneal-pleural leak, we conducted scintigraphy with a mixture of technetium-99m and dialysate, which

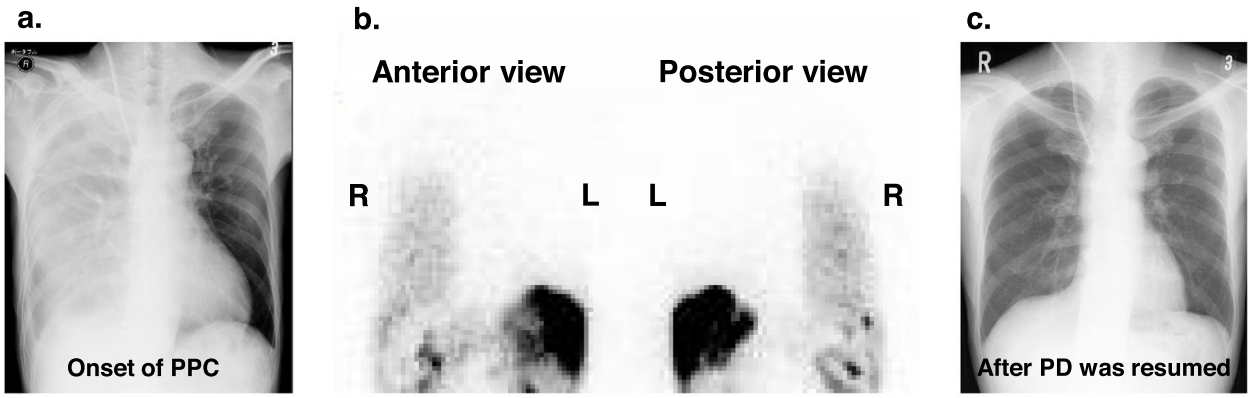

Fig. 1 Pleural effusion detected by imaging examination. **a** Abundant pleural effusion can be detected, predominantly on the right side of the lung field, as seen on chest radiography performed after the patient complained of slight dyspnea with weight gain. **b** Pleuroperitoneal scintigraphy with a mixture of technetium-99m and dialysate reveals a transdiaphragmatic leakage predominantly on the right side of the pleural cavity within 120 min after intraperitoneal injection. **c** After conservative management, no pleural effusion is detected on a chest radiograph

we infused into the peritoneal cavity through a PD catheter under aseptic conditions. During the examination, the patient was advised not to move the spine, and continuous still images in both anterior and posterior views were acquired at frequent time intervals. Isotopic peritoneography revealed radioactive dialysate entering the right pleural cavity at 120 min (Fig. 1b); therefore, we strongly suspected PPC. Although we offered to perform surgical detection and repair of the suspected defect in the diaphragm, the patient did not consent to it and opted to continue PD treatment conservatively. Therefore, we adjusted his prescription to involve dialysate exchange in the daytime alone in a semi-sitting position. We also prohibited the patient from lying down during the daytime, and he was only allowed to stand or be in a semi-sitting position. He continued to receive PD treatment, without signs of pleural effusion (Fig. 1c) and an over-volume status, with three exchanges per day, each with 2.5% dextrose-based dialysate lasting for 2 h, and one exchange per day with an icodextrin-based solution for 8 h. After adding diuretics (40 mg of furosemide and 7.5 mg of tolvaptan), the patient was discharged at 73 days after his admission, and he resumed visits to our PD outpatient clinic.

Discussion

PPC is known as a crucial comorbidity in PD patients. The prevalence of PPC is reported to be 1.6–10% [3, 7], with a time-to-onset ranging from months to years [7]. The exact etiology of PPC occurrence in patients undergoing PD is unclear. The mechanism underlying the development of ascites in the pleural effusion is considered to be related to the increased intra-abdominal pressure due to the accumulation of PD dialysate or liver cirrhosis in the presence of a diaphragmatic defect, such as those around the major vessels and the esophagus or through the diaphragmatic foramina or lymphatics [1, 2]. Previous studies have reported that once blebs that form on fragile regions in the diaphragm due to intra-abdominal pressure are ruptured, they could cause a hydrothorax and a one-way valve [9, 10].

PPC can be detected via pleural fluid analysis and imaging techniques, such as computed tomography using a contrast agent or peritoneal scintigraphy. Pleural fluid analysis commonly reveals an elevated glucose concentration or a high pleural fluid-to-serum gradient. Previous reports showed that a transudative pleural fluid glucose concentration greater than the serum glucose concentration (pleural fluid to serum glucose ratio > 1) in a patient receiving PD was consistent with PPC [11]. In addition, PPC presence should be doubted if the pleural fluid glucose level is higher than the serum glucose level [12]. Recent reports described that the positive detection rate of computed tomography and scintigraphic peritoneography for PPC was one third and 40–50% of cases, respectively [1, 13, 14]. Therefore, even if the pleural fluid glucose level and/or the imaging test do/does not prove peritoneal-pleural leakage, PPC may still be present in PD patients with pleural effusion. In our case, peritoneal scintigraphy revealed radiotracer activity in the thoracic cavity, which indicated a high possibility of PPC.

Although specific guidelines or consensus regarding the definitive treatment of PPC have not been established, a few options can be considered. These options include conservative treatment, chemical pleurodesis, or surgical repair of the diaphragmatic leak site. The conservative approach for a hydrothorax related to PPC in PD patients should be considered first; however, limited success was shown in a previous report of a case series [7]. The rate of continuation of PD with conservative treatment for PPC was found to be approximately 50–

60% [3, 4]. Conservative management of PD patients with PPC includes the reduction of the PD dialysate volume in patients with residual renal function to decrease the intra-abdominal pressure [15, 16] and a temporary shift to hemodialysis in patients with anuria until the spontaneous closure of minor defects in the diaphragm, which takes approximately 4–6 weeks [17]. Chemical pleurodesis is the second option and it is performed with talc, tetracycline, doxycycline, or autologous blood infusion into the chest cavity under VATS guidance [18–20]. Pleurodesis for PPC has been confirmed to have a success rate of 90% [4] and a large confidence interval (50–100%) which was attributed to the fact that pleural adhesion caused by sclerosing substances is not always firmly formed, resulting in PPC recurrence [18, 21, 22].

In recent years, surgical repair via VATS has been advocated [23]. However, this option has the following limitations: it is more invasive than other options, the defect site may not be detected owing to the microscopic size in most cases [21, 24], and the recurrence rate of pleuroperitoneal leak after VATS is uncertain. In previous reports, the priority of these PPC treatment options was variable, and because there are currently no evidence-based guidelines for PPC treatment, the treatment decision is dependent on the patient's clinical status and the patient's choice, as seen in our case.

In our case, although the connection between gastrectomy and the occurrence of PPC was doubtful, it was also difficult to determine whether PPC was due to surgical manipulation or incomplete wound healing at the surgical site. This was because gastrectomy was performed via open surgery, which, unlike laparoscopic surgery, is not affected by elevated intraperitoneal pressure, and the wound healing progressed well for 1 month. Although we could not find any report of PPC related to a surgical procedure, some reports have described a relationship between malignant tumors and PPC-presenting patients with ovarian cancer [25] or abdominal ascites related to gastric cancer [26]. However, in these reports, PPC was the result of cancer or was associated with a malignant pathology and not with surgical complications.

Regardless of whether the PPC in our case was due to the surgical procedure or not, we should consider the optimal period of peritoneal rest until the resumption of PD. For surgery in PD patients, there is a general concern about possible leakage of dialysate from the postoperative wound [4, 27, 28]. Previous reports suggested that the peritoneal rest period after surgery in PD patients should be 4–6 weeks, but it is unclear whether this time is sufficient to prevent postoperative complications. The effective rest period in our case was 5 weeks. The optimal period of peritoneal rest with PD discontinuation after pleurosclerosis or a diaphragm repair procedure for a PPC defect was described as up to 8 weeks [4, 29, 30]. It is currently difficult to determine the appropriate period of postoperative peritoneal rest to prevent complications, and this may be a good focus for prospective clinical trials in the future.

Another suspected cause of PPC in our patient was the bacterial peritonitis that occurred prior to gastrectomy. Although bacterial peritonitis has not been indicated as a major pathology that induces weakness of the peritoneum and diaphragm, some reports have suggested an association between PPC and peritonitis [31]. Prevot et al. reported a case of pyopneumothorax and peritonitis due to a perforated duodenal ulcer accompanied by PPC [32]. The cause of the pyopneumothorax was acute peritonitis in the presence of PPC; however, the authors did not mention an association between peritonitis and the onset of PPC. Thus, there is insufficient evidence to confirm a relationship between PPC and bacterial peritonitis.

Mini review: notable PD-related complications other than those related to infections

We often encounter various clinical complications in the management of patients undergoing PD. PD-related complications are generally classified into infectious or non-infectious pathologies. During these four decades since PD was first established, the practice of PD has comprised a history of resolving complications associated with infectious diseases [33]; however, it has been shown that the number of cases of PD discontinuation due to infectious diseases is decreasing due to increasing knowledge and emerging guidelines for its treatment [34, 35]. In contrast, there is concern regarding the increasing rate of non-infectious complications that could lead to technique failure, and approximately 20% of PD patients switch to hemodialysis owing to these complications [36]. We reviewed some clinically significant non-infectious PD-related complications and the main treatment options (Fig. 2). These complications occasionally cause common clinical symptoms, such as localized edema and dialysate flow failure, and they decrease the effectiveness of PD treatment.

Catheter-related complications

Non-infectious catheter-related complications include malposition, obstruction, and leakage. Most of these complications result in flow failure, abdominal pain, bleeding, and sometimes, infections. Moreover, various pathologies exist for catheter obstruction, including kinking, intraluminal obstruction by fibrin or clot, external wrapping by the omentum or fallopian tubes, and external adhesion of the dilated intestine due to constipation. There are various solutions for eliminating these complications depending on their cause(s). Some of these complications are conservatively improved by the administration of a laxative, by reducing or

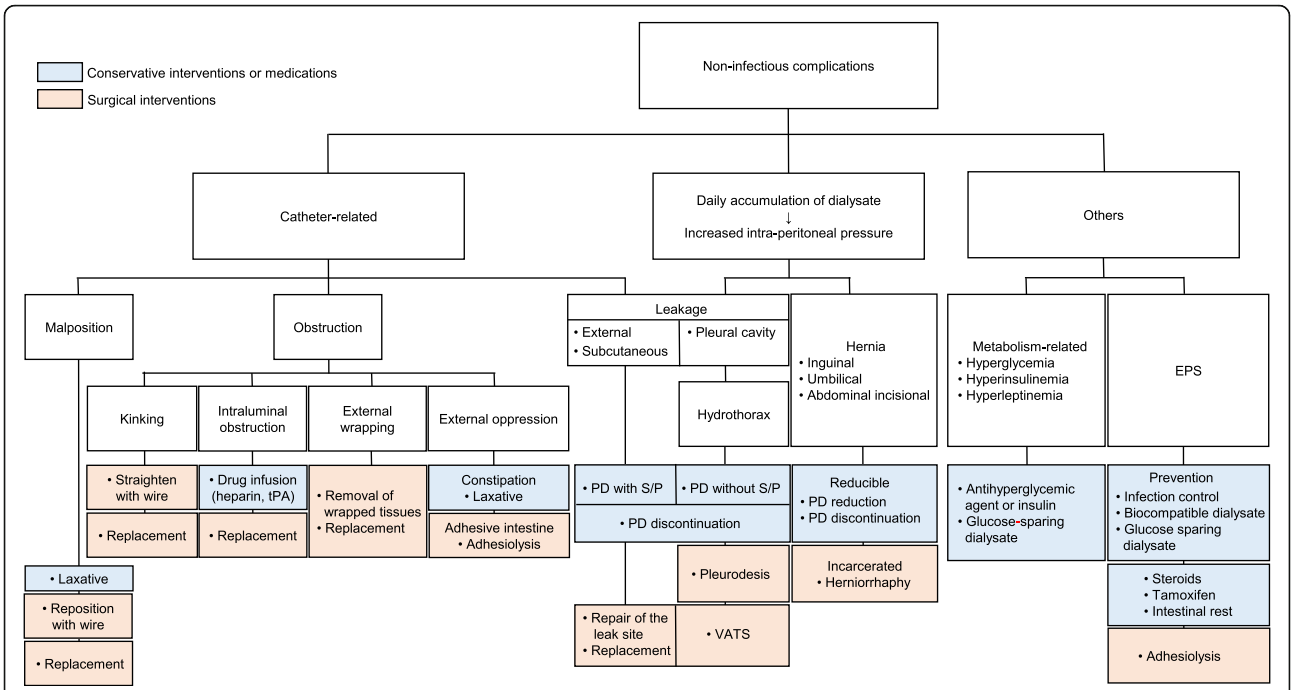

Fig. 2 Schematic overview of non-infectious PD complications. In relation to malposition, a laxative is considered first to smoothen the intestinal movement, and the manipulation of the catheter using a semi-flexible wire or surgical repositioning is the next strategy. In case of kinks, a wire technique to straighten the kinking site or surgical replacement is utilized. Surgical replacement is adapted for an intraluminal-occluded catheter in cases of ineffective drug infusion or for a catheter wrapped by external tissues which cannot be removed. In case of external oppression by the bowel tract, laxatives are suitable; however, when external oppression is induced by a firmly adhesive intestine, surgical adhesiolysis should be considered. Leakage could be conservatively improved by the reduction or discontinuation of dialysate exchange or by changing the patient's position during dialysate dwell time. Surgery should be considered if catheter-related leakage is prolonged or repetitive. Hydrothorax induced by IAP elevation requires pleurodesis or surgical repair using VATS in the absence of improvement by conservative measures. Although hernia is often restored spontaneously by decreasing IAP via the reduction or discontinuation of PD, surgical repair is required if there is a recurrence or if the herniated intestine is incarcerated. In metabolic disorders mainly induced by a high glucose-containing dialysate, we consider the use of medications and changing the PD regimen with a glucose-sparing dialysate. To avoid EPS progression, infection control and a biocompatible or glucose-sparing dialysate should be considered. In the inflammatory phase, steroid or tamoxifen administration may be considered, and if ileus is present, adhesiolysis should be chosen. EPS, encapsulating peritoneal sclerosis; IAP, intraperitoneal pressure; PD, peritoneal dialysis; S/P, spine position; t-PA, tissue plasminogen activator; VATS, video-assisted thoracoscopy

discontinuing dialysate exchanges, and in some cases, by invasive procedures, such as catheter repositioning through a fluoroscopically guided manipulation using a semi-flexible wire [37], laparoscopic catheter repositioning, catheter replacement in an open abdomen, or surgical removal of obstructing tissues from the catheter tip [38]. A previous report presented the effect of the instillation of a thrombolytic agent (tissue plasminogen activator) against intraluminal occlusion [39]. Catheter-related leakage around the catheter to external or subcutaneous tissue is mostly due to inadequate surgical techniques such as incomplete suturing of the catheter cuff to the peritoneum, wound healing delay, or anatomic abnormalities, such as a patent process of the peritoneum.

Complications induced by increased intraperitoneal pressure (IAP)

IAP increases due to the accumulation of dialysate fluid in the peritoneal cavity and is the inevitable pathology surrounding PD treatment. According to the pathological conditions that PD patients present with, a hernia or hydrothorax could occur when the structures that support the tissues around the abdominal cavity, such as the inguinal canals and the umbilicus, are potentially weak or defective or when the patient has a prior surgical incision or a diaphragmatic defect that occurred prior to PD initiation. Hydrothorax is a typical example of a leakage due to increased IAP, and a defect in the diaphragm becomes a traffic route resulting in PPC. Being female and having ADPKD have also been reported as prevalent factors related to PPC, followed by hydrothorax [5, 6], but there are also reports that suggest that there is no clear association between hydrothorax and sex [7] or ADPKD [8]. Thus, the factors that induce PPC have not been established. Given that PPC can occur at any time during the management of a PD patient, PPC should be suspected in cases of unexplained pleural effusion.

Conversely, a hernia is more likely to be encountered clinically. A hernia is often restored spontaneously by decreasing the IAP via the reduction or discontinuation of PD treatment. However, surgical repair is required if there is a recurrence or the herniated intestine is incarcerated and necrotic [40]. A previous large-scale retrospective study conducted in the USA indicated that cystic disease resulted in a 2.5-fold increase in the risk of anatomic complications; female sex resulted in an 80% reduction in the risk, and a Kt/V of over 2.0 resulted in a 52% reduction in the risk of hernia [41].

Other notable complications

Previous studies have reported the importance of metabolic disorders in PD patients [36]; hyperglycemia and hyperinsulinemia increased the prevalence of cardiovascular disease events in PD patients compared to patients undergoing hemodialysis [42]. In particular, dialysates containing high levels of glucose could elevate plasma insulin levels with intraperitoneal glucose loading in a dose-dependent manner and induce the onset of diabetes [43] and the harmful effects of a hemodynamic status [44]. Therefore, not only medications such as antihyperglycemic agents or insulin should be considered, a combined regimen including a dialysate containing as low levels of glucose as possible and a glucose-sparing dialysate containing icodextrin should also be considered [45].

Another critical issue in PD management is encapsulating peritoneal sclerosis (EPS). EPS is defined as a series of pathologies in which various stages of the pathologies progress, such as the stage of peritoneal inflammation, peritoneal membrane thickening, and encasement of bowel loops, which finally cause infiltration failure and frequent bowel obstructions. Although previous reports that suggested an association between EPS progression and significant morbidity and high mortality have prevented the widespread use of PD, EPS has been on a decreasing trend due to improvements in the treatment of peritonitis and the regimen of the dialysate [46]. Recently, the risks of and treatment guidelines for EPS were published in Japan [47], and various interventions were recommended to avoid EPS progression. Concerning the risk factors for EPS, such as peritonitis, low dialysate pH, or high levels of glucose degradation products in the dialysate, thorough hand hygiene to prevent bacterial contamination and the use of highly biocompatible or glucose-sparing dialysate to reduce peritoneal membrane deterioration are recommended. In the inflammatory phase, steroids or tamoxifen may be used to reduce peritoneal fibrosis and sclerosis; however, the outcomes obtained from the administration of these agents are limited [48, 49]. The results of surgical interventions have improved, and adhesiolysis performed by an experienced team should be considered early [49]. In order to accomplish effective RRT, discussions about shifting to hemodialysis should be initiated while still continuing PD with adequate safety [48].

Conclusions

PPC is a rare but common complication in PD patients. PPC is due to the elevation of pressure in the abdominal cavity via various pathways; therefore, it is important to identify the appropriate treatment options that are available for PPC in each case. Moreover, more information is required to establish the optimal peritoneal rest period after surgical procedures. Assessing information regarding the optimal period of peritoneal rest will result in improved patient outcomes, as well as meet patients' desire to maintain their normal lifestyle through continued PD treatment. Here, we describe a PD patient who developed PPC after bacterial peritonitis and gastrectomy. Our case showed that even if we take measures to prevent pleuroperitoneal leakage, PPC could occur at any time. We hope that our data will contribute to the establishment of a consensus or guidelines for PPC management in PD patients.

Abbreviations

PD: Peritoneal dialysis; PPC: Pleuroperitoneal communication; ADPKD: Autosomal dominant polycystic kidney disease; VATS: Video-assisted thoracoscopy; IAP: Intraperitoneal pressure; EPS: Encapsulating peritoneal sclerosis

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Authors' contributions

JI and HK, as attending doctors, were involved in the treatment protocol. Clinical follow-up and material preparation, such as data imaging collection, and histological examination, were performed by JI, HK, EK, MK, KS, and HE. The manuscript was written by JI. KN commented on the manuscript as a supervisor. All authors have read and approved the final version of the manuscript.

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Availability of data and materials

The datasets used in this case report are available from the corresponding author on reasonable request.

Ethics approval and consent to participate

In this report, all procedures were carried out in accordance with the ethical standards of the institutional review board of our center (IRB approval number 0454).

Consent for publication

Informed consent was obtained from the patient and his family.

Competing interests

The authors declare that they have no competing interests.

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