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Fallopian Tube Entrapment as a Cause of Catheter Dysfunction in Peritoneal Dialysis: An Unusual Case

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Abstract

Peritoneal dialysis (PD) catheter dysfunction is a common complication, usually related to mechanical factors such as catheter displacement, intraluminal obstruction, or omental wrapping. Fallopian tube entrapment is an exceptionally rare cause of PD catheter dysfunction.

We report the case of a 29-year-old woman with end-stage kidney disease (ESKD) secondary to diabetic nephropathy, undergoing continuous ambulatory PD. One month after laparoscopic catheter insertion, she developed complete dialysate flow obstruction associated with hemorrhagic effluent. Abdominal radiography showed no evidence of catheter migration or kinking, and laboratory findings were unremarkable. Conservative measures, including patient mobilization, laxatives, and intraluminal heparin instillation, failed to restore catheter patency. Exploratory laparoscopy revealed tight wrapping of the catheter by the fallopian tube, resulting in obstruction of the drainage holes. The catheter was successfully released and repositioned in the Douglas pouch. Patency was confirmed intraoperatively, and PD was resumed without further complications.

This case highlights fallopian tube entrapment as a rare but clinically significant cause of PD catheter dysfunction. Because clinical and radiological findings may be nonspecific, surgical exploration may be required to establish the diagnosis. Laparoscopic intervention allows simultaneous diagnosis and treatment while preserving the PD modality. This unusual cause should be considered in female patients presenting with unexplained catheter dysfunction after common mechanical causes have been excluded.

Categories: Nephrology
Keywords: catheter dysfunction, end stage kidney disease (eskd), fallopian tube entrapment, laparoscopy, mechanical complication, peritoneal dialysis (pd)

Introduction

Peritoneal dialysis (PD) catheter dysfunction is a common complication that may compromise dialysis efficacy and lead to technique failure. Although most cases result from mechanical causes, such as catheter migration, intraluminal obstruction, or omental wrapping, entrapment by intra-abdominal structures is rare [1-4].

Fallopian tube entrapment represents an exceptionally uncommon cause of catheter obstruction, with diagnosis often established only during surgical exploration because clinical and radiological findings may be nonspecific [1,5]. We report a rare case of PD catheter dysfunction caused by fallopian tube entrapment, successfully diagnosed and managed by laparoscopic intervention.

Case Presentation

A 29-year-old woman with end-stage kidney disease (ESKD) secondary to diabetic nephropathy had been undergoing PD. The PD catheter was inserted using a laparoscopic technique without immediate complications. She was maintained on continuous ambulatory PD, consisting of three daily exchanges with 1.36% glucose dialysate. Residual renal function was preserved, and catheter function was optimal, with consistently adequate dialysate inflow and outflow.

One month after initiation of PD, the patient was admitted to the Department of Nephrology, complaining of decreased dialysate flow rates and hemorrhagic dialysate for the previous two days. Clinical examination revealed abdominal discomfort and complete absence of both inflow and outflow.

Initial investigations were performed. Abdominal X-ray (Figure *1*) was non-contributory and excluded catheter kinking or migration as causes of dysfunction. Laboratory tests showed no significant abnormalities (Table *1*).



FIGURE 1: Abdominal radiograph showing the peritoneal dialysis catheter in situ, without migration or kinking.

Parameter	Patient value	Reference range	Unit
Blood pressure	112/68	<140/90	mmHg
Heart rate	87	60–100	beats/min
Temperature	37.1	36.0–37.5	°C
Hemoglobin (Hb)	10.7	12–16	g/dL
White blood cell count (WBC)	7.8	4–10	$\times 10^9$/L
Platelet count (PLT)	201	150–400	$\times 10^9$/L
Serum creatinine (Cr)	631.5	45–90	µmol/L
Blood urea nitrogen (BUN)	12.3	2.5–7.5	mmol/L
Sodium (Na^+)	136	135–145	mmol/L
Potassium (K^+)	4.8	3.5–5.0	mmol/L
Blood glucose	4.1	3.9-5.5	mmol/l
Bicarbonate (HCO3-)	23	22-29	mmol/l
C-reactive protein (CRP)	12	<5	mg/L
Procalcitonin (PCT)		<0.5	ng/mL

TABLE 1: Routine laboratory investigations and vital signs at presentation.

Normal reference ranges can vary between different laboratories.

Given the suspicion of omental wrapping or intraluminal obstruction by a fibrin clot, Initial conservative management was attempted in order to restore catheter function. These measures included patient mobilization, administration of laxatives to stimulate bowel peristalsis, and intraluminal instillation of heparin to treat potential fibrin-related obstruction. Despite these interventions, the catheter dysfunction persisted, with continued impairment of dialysate inflow and outflow.

Given the persistence of the obstruction and the failure of conservative measures, the decision was made to perform an exploratory laparoscopy. Intraoperative findings revealed that the PD] catheter was tightly wrapped by the fallopian tube, leading to obstruction of the drainage holes (Figure *2*).

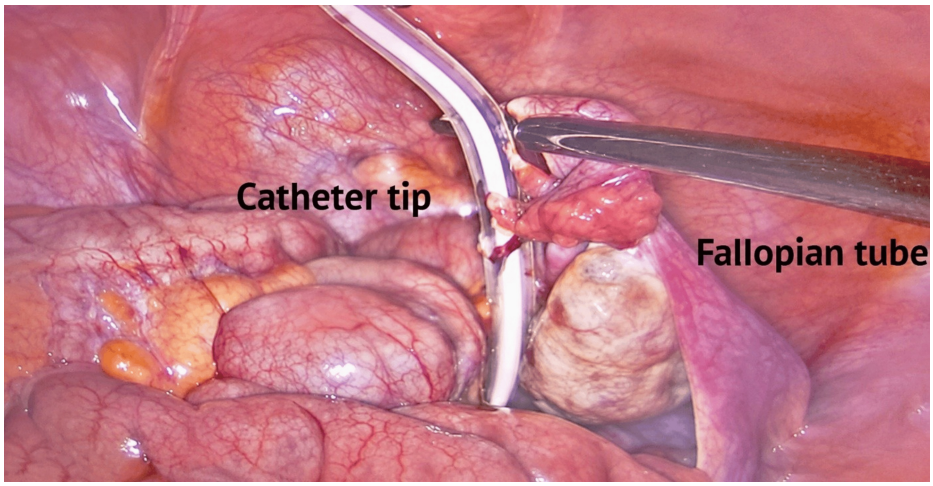


FIGURE 2: Intraoperative laparoscopic view showing entrapment of the peritoneal dialysis catheter tip by the fallopian tube.

The catheter was carefully released from the fallopian tube. Following this maneuver, catheter patency was

assessed intraoperatively and confirmed by satisfactory inflow and outflow of dialysate. The catheter was then repositioned in the Douglas pouch. The procedure was completed without injury to the fallopian tube and without hemorrhagic complications. The postoperative course was uneventful, and PD was subsequently resumed with normal inflow and outflow.

Discussion

PD is an established renal replacement therapy for patients with ESKD, using the peritoneal membrane to allow solute and fluid exchange through the infusion and drainage of dialysate. It relies entirely on the catheter for dialysate exchange; therefore, its correct positioning and proper functioning are crucial for the success of this therapy [1]. Catheter dysfunction represents a major complication of PD [2]. Several mechanisms may contribute to catheter dysfunction, including kinking, displacement, intraluminal thrombosis, omental wrapping, or obstruction by other intraperitoneal structures [1,3]. Reported rates of catheter dysfunction due to mechanical causes range from 2% to 36%, but the incidence has decreased with the adoption of laparoscopic insertion techniques [6]. In our center, mechanical complications were predominantly represented by PD catheter migration, observed in 38.8% of patients, requiring surgical repositioning in 10% of cases. Catheter obstruction accounted for 20.6% of cases [7].

However, other intraperitoneal structures may rarely be involved, such as the appendix, sigmoid colon, or fallopian tube [1,2,8]. Entrapment of visceral structures can lead to significant morbidity, and obstruction due to fallopian tube entrapment is considered an exceptionally rare complication [8,9]. The fallopian tubes are paired muscular ducts that extend bilaterally from the uterine fundus toward the ovaries. Located within the peritoneal cavity along the superior border of the broad ligament, their fimbrial extremity opens directly into the peritoneal space near the ovary. Because of this intraperitoneal position and their mobility, the fallopian tubes may occasionally come into contact with intraperitoneal devices such as a PD catheter.

Although this condition may remain clinically silent, as in our case, some patients may develop symptoms including iliac-region pain and vaginal leakage of fluid [1,2]. This can be explained by the anatomical communication between the peritoneal cavity and the uterine cavity through the fallopian tubes, allowing dialysate to pass into the uterus and subsequently leak through the vagina when intraperitoneal pressure increases. As reported in the literature, radiological investigations may be inconclusive [6]. Similarly, in our patient, X-ray did not identify the cause of catheter dysfunction.

Several factors have been proposed to explain fallopian tube entrapment of a PD catheter, including prior episodes of peritonitis, previous surgery, and proximity of the catheter tip to the fimbrial end of the tube. This complication also seems to be more frequently reported in pediatric patients [1,10,11]. None of these factors were present in our patient. Hemorrhagic effluent in women undergoing PD may be related to menstruation or ovulation. Although our patient was not menstruating, ovulation-related bleeding cannot be excluded. However, its potential role in catheter entrapment remains speculative, with no established causal relationship [2,9].

Management initially relies on conservative approaches aimed at restoring catheter function. These include patient mobilization, stimulation of bowel peristalsis with laxatives, and intraluminal instillation of anticoagulants [12,13]. However, these empirical measures generally show limited effectiveness and often fail to restore adequate dialysate flow, as observed in our patient [12,13]. Laparoscopic management of catheter dysfunction secondary to fallopian tube entrapment has been reported as both a diagnostic and therapeutic approach, allowing catheter preservation with favorable outcomes [2,6,10]. In line with these findings, the decision in our case was to perform exploratory laparoscopy, which revealed a catheter entrapped by the fallopian tube. The catheter was released, restoring patency, and subsequently repositioned without complications.

Conclusions

Fallopian tube entrapment of PD catheters is an exceptionally rare cause of catheter malfunction. Only a limited number of cases have been reported in the literature. Because clinical and radiological findings are nonspecific, the diagnosis is established during surgical exploration. Laparoscopic intervention allows confirmation of the diagnosis and effective management, enabling preservation of the technique. This etiology should be considered in female patients presenting with unexplained catheter dysfunction, particularly when mechanical causes have been excluded.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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