



Case Report

An Unusual Complication in a Patient on CAPD – Retroperitoneal Hemorrhage: A Case Report

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ABSTRACT

Background: Hemoperitoneum is an infrequent but recognized complication of continuous ambulatory peritoneal dialysis (CAPD), most commonly attributable to abdominal trauma, gynecological causes (follicular cyst rupture, menstrual reflux, ovulation, endometriosis), or intraperitoneal sources such as hepatic or splenic cyst rupture, catheter-related trauma, and vascular anomalies. Hemoperitoneum originating from a retroperitoneal source, particularly cyst rupture in autosomal dominant polycystic kidney disease (ADPKD), is exceedingly rare and has been reported only twice previously in the literature.

Case Presentation: We describe a 45-year-old postmenopausal woman with a 20-year history of ADPKD and ESKD on CAPD, who presented with hemorrhagic peritoneal dialysate outflow followed by complete outflow obstruction, anasarca, and altered sensorium. Imaging demonstrated a large intraperitoneal collection with air foci alongside a separate lesser-sac collection, in the absence of any demonstrable retroperitoneal-to-peritoneal communication or active contrast extravasation. No alternative source of hemorrhage was identified after multidisciplinary evaluation. The patient was managed with temporary discontinuation of CAPD, hemodialysis via a non-tunneled internal jugular catheter, blood transfusion, and intraperitoneal antibiotic therapy, with gradual clinical improvement.

Conclusion: Retroperitoneal hemorrhage should be considered in the differential diagnosis of hemoperitoneum in patients with ADPKD on peritoneal dialysis. Prompt recognition of pain and hemorrhagic dialysate in this population is essential to enable timely intervention and salvage of the peritoneal dialysis modality.

Keywords: Hemoperitoneum; Peritoneal Dialysis; Autosomal Dominant Polycystic Kidney Disease; Retroperitoneal Hemorrhage; Continuous Ambulatory Peritoneal Dialysis

Introduction

Hemoperitoneum is a well-recognized, though infrequent, complication among patients undergoing continuous ambulatory peritoneal dialysis (CAPD). The most common cause is direct or catheter-related trauma, while gynecological etiologies—including follicular cyst hemorrhage, retrograde menstrual flow, ovulation, and endometriosis—predominate among women of reproductive age. Other intraperitoneal sources include spontaneous rupture of hepatic or splenic cysts and vascular anomalies. In contrast, hemoperitoneum arising from a retroperitoneal source is markedly uncommon and has been described almost exclusively in the context of cyst rupture in patients with autosomal dominant polycystic kidney disease

(ADPKD) or acquired cystic kidney disease [1-3]. To date, only two such cases have been reported: one in a patient with ADPKD, involving an intracystic hematoma [2], and another in a patient with acquired cystic kidney disease, involving cystic rupture with an associated perirenal hematoma [3]. Notably, in both prior reports and in the present case, hemoperitoneum occurred despite the absence of any demonstrable retroperitoneal-to-peritoneal communication on abdominal imaging, and no alternative bleeding source could be identified. We present a further case illustrating this rare complication, with the aim of raising clinical awareness among nephrologists managing ADPKD patients on peritoneal dialysis.

Case Presentation

Background and Renal History

A 45-year-old woman was diagnosed with ADPKD in 2005, following presentation with bilateral loin pain and renal dysfunction (serum creatinine 3 mg/dL); the diagnosis was confirmed on abdominal imaging demonstrating bilateral polycystic kidneys and supported by a positive family history of ADPKD (affecting her younger brother and son), although genetic testing was not performed. She was managed medically but subsequently lost to follow-up until 2022, when she developed recurrent generalized tonic-clonic seizures; magnetic resonance imaging of the brain revealed subarachnoid hemorrhage secondary to a ruptured berry aneurysm, for which she underwent aneurysmal clipping with decompressive craniotomy and was subsequently maintained on antiepileptic therapy. At that time, her serum creatinine was 4.5 mg/dL with a urine output of 2 L/day, and she was discharged on conservative management.

In 2023, she presented with fluid overload and uremic symptoms (serum creatinine 15 mg/dL) and was initiated on hemodialysis via a left femoral catheter, subsequently transitioned to a right internal jugular venous (IJV) catheter. A left brachiocephalic arteriovenous fistula was created but failed after four dialysis sessions. Maintenance hemodialysis was continued via non-tunneled right and, later, left IJV catheters. The left IJV catheter had been placed as an emergency access once the right IJV became unusable and the left AVF had failed; despite partial thrombosis of the left IJV noted on subsequent Doppler evaluation, this access permitted three hemodialysis sessions to relieve excess fluid before catheter dysfunction necessitated further intervention.

In March 2024, she was readmitted with fluid overload and uremic symptoms after a one-month interruption in dialysis due to right IJV catheter

dysfunction. Doppler ultrasonography demonstrated complete right IJV, partial left IJV, and complete left femoral vein thrombosis. Given exhausted vascular access options, a percutaneous soft peritoneal dialysis (PD) catheter (Covidien percutaneous catheter set; two-cuff Tenckhoff catheter, 42 cm) was inserted, with satisfactory catheter position confirmed on abdominal radiography, and the patient was initiated on automated peritoneal dialysis cycles, subsequently transitioning to CAPD (three exchanges/day; 2 L dwell volume; 4-hour dwell time; 1.5% dextrose solution) with a daily ultrafiltrate of approximately 900 mL. She remained clinically stable on this regimen for approximately nine months prior to the current presentation.

Obstetric and Family History

The patient was para 2, living 2, with both deliveries full-term normal vaginal deliveries; there was no history of gestational hypertension or pregnancy-related acute kidney injury. Her last childbirth was 23 years prior, and she had attained menopause 4 years earlier. Family history was notable for ADPKD in her younger brother and son, though genetic testing had not been performed in any family member; there was no family history relevant to the obstetric course described above.

Presenting Complaint

The patient developed hemorrhagic PD outflow followed by complete outflow obstruction over one week, prompting cessation of CAPD exchanges at home. She subsequently presented to our institution with anasarca, New York Heart Association (NYHA) class IV dyspnea (respiratory rate 22/min, oxygen saturation 96% on room air), and altered sensorium.

Clinical Examination

On examination, the patient was drowsy (Glasgow Coma Scale 9/15) with pallor and pedal

edema; she was afebrile with a blood pressure of 100/70 mmHg and pulse rate of 92 beats/min. The abdomen was soft and non-tender. The PD catheter was in situ with no evidence of exit-site or tunnel-site erythema, tenderness, or purulent discharge.

Investigations

Laboratory investigations are summarized in Table 1.

Table 1. Laboratory and Peritoneal Dialysis Fluid Investigations at Presentation

Parameter	Value
Complete Blood Picture (Hb/TLC/Platelets, $\times 10^3$)	7.5 g/dL / 7,500 / 1.75 lakh/ μ L
Renal Function Tests (Urea/Creatinine)	307 mg/dL / 20.07 mg/dL
Serum Electrolytes (Na^+/K^+)	146 mEq/L / 3.8 mEq/L
Serum Calcium / Phosphorus / Uric Acid	8.3 mg/dL / 6.5 mg/dL / 6.7 mg/dL
Total Bilirubin	0.7 mg/dL
AST / ALT	9 U/L / 14 U/L
Serum Albumin / Total Protein	2.0 g/dL / 5.7 g/dL
PT / INR / APTT	14 sec / 1.2 / 33 sec
PD Fluid Cell Count	1,100 cells/ μ L (60% neutrophils, plenty of RBCs/HPF)
PD Fluid Cytology	Negative for malignancy

Abbreviations: ALT – alanine aminotransferase; APTT – activated partial thromboplastin time; AST – aspartate aminotransferase; Hb – hemoglobin; INR – international normalized ratio; K^+ – potassium; Na^+ – sodium; PD – peritoneal dialysis; PT – prothrombin time; TLC – total leukocyte count.

Ultrasonography of the abdomen revealed bilateral polycystic kidneys with a large intraperitoneal hemorrhagic collection measuring 16.5×8 cm. Contrast-enhanced computed tomography (CECT) of the abdomen demonstrated bilaterally enlarged

kidneys (right $17 \times 9 \times 5.2$ cm; left $15.3 \times 8 \times 4.9$ cm) with multiple hypodense cystic lesions showing foci of hyperdensity consistent with intracystic hemorrhage, and scattered cystic calcifications (Figure 1).



Figure 1. Chest radiograph confirming an appropriately positioned non-tunneled left internal jugular venous catheter tip (arrow) at the cavoatrial junction. Patient identifiers have been redacted.

A well-defined intraperitoneal collection of fluid attenuation with internal air foci, measuring approximately $23 \times 6.7 \times 18$ cm, extended from the left subhepatic space—abutting the left hepatic lobe and greater curvature of the stomach—inferiorly to the inframesocolic compartment at the L5–S1 level, with lateral extension into the right paracolic gutter. A separate collection measuring $6.2 \times 9.7 \times 3.6$ cm was noted in the inferior recess of the lesser sac, anterior and posterior to the distal pancreatic body. No drainage procedure was performed in the interim, and the interval reduction in collection size on repeat CECT after three days ($4.7 \times 8 \times 9.2$ cm and $3 \times 6.2 \times 6$ cm, respectively) most likely reflected spontaneous resorption; air foci again were present, with no active contrast extravasation. Notably, no retroperitoneal-to-peritoneal communication was identified on either study. PD fluid analysis showed a cell count of 1,100 cells/ μ L with 60% neutrophils and abundant red blood cells (formal red cell count not performed); a PD fluid sample was sent for culture prior to initiation of antibiotics and remained negative, and cytology was negative for malignancy.

Management and Clinical Course

Saline flushes administered through the CAPD catheter yielded approximately 1 L of hemorrhagic

outflow (Figures 2 and 3); CAPD was subsequently discontinued and the fluid sent for analysis. Given the prior successful, if temporary, use of the left IJV for hemodialysis access in 2023 despite partial thrombosis on Doppler, and in the absence of any alternative site (complete right IJV and left femoral vein thrombosis), vascular access was re-established via a non-tunneled left IJV catheter, with satisfactory tip position confirmed on chest radiography (Figure 4), and the patient was transitioned to daily 6-hour hemodialysis sessions with satisfactory ultrafiltration. She received transfusion of one unit of packed red blood cells and two units of fresh frozen plasma during dialysis. General surgery and urology consultations were obtained, and the patient was monitored with serial abdominal girth measurements. Intraperitoneal vancomycin and ceftazidime were started empirically after the PD fluid sample was sent for culture.

By the fourth hospital day, the patient's sensorium had improved. At the time of writing, she remains hospitalized, with plans to either resume CAPD after a two-week interval or proceed to permanent left IJV catheter placement for continuation of maintenance hemodialysis.



Figure 2. Coronal contrast-enhanced CT image showing bilaterally enlarged kidneys with multiple parenchymal cysts (arrows), several with hyperdense contents consistent with intracystic hemorrhage, in a patient with ADPKD and ESKD; the percutaneous CAPD catheter is seen in situ (arrowhead).



Figure 3. Blood-tinged CAPD drainage bag showing hemorrhagic peritoneal effluent, obtained at presentation.



Figure 4. Hemorrhagic peritoneal dialysis effluent collected in the drainage bag following saline flush of the CAPD catheter.

Discussion

Hemoperitoneum in patients on peritoneal dialysis is an uncommon complication that typically arises from trauma, gynecological pathology, or intraperitoneal visceral or vascular sources. A retroperitoneal origin—specifically, hemorrhage from a ruptured renal cyst in ADPKD—represents a distinctly rare and likely under-recognized mechanism. Only two prior cases have been documented in the literature: one in a patient with ADPKD, describing an intracystic hematoma [2], and the other in a patient with acquired cystic kidney disease, describing cystic rupture with a perirenal hematoma [3]; both ultimately manifested as bloody peritoneal effluent. A parallel has similarly been drawn to hemoperitoneum in polycystic kidney disease more broadly [1].

A striking feature shared by the present case and both prior reports is the absence of a radiologically demonstrable communication between the retroperitoneal and peritoneal compartments, despite clear clinical and biochemical evidence of hemorrhagic dialysate. This suggests that microscopic or transient tracking of blood across retroperitoneal fascial planes into the peritoneal cavity may occur without leaving an imaging-detectable defect, particularly in the setting of markedly distorted renal and perirenal anatomy from long-standing polycystic disease. The coexistence of intracystic hemorrhage, cyst calcification, and large-volume fluid collections with air foci in this patient further underscores the complexity of localizing the precise bleeding source in such cases, and highlights the limitations of cross-sectional imaging in confirming a retroperitoneal-to-peritoneal tract.

From a management standpoint, hemorrhagic PD effluent in a patient with ADPKD warrants a systematic evaluation that includes assessment for catheter-related trauma, gynecological causes, and intraperitoneal visceral pathology, in addition to a high

index of suspicion for retroperitoneal cyst rupture even when initial imaging does not demonstrate a clear communication. Notably, the PD effluent cell count in this patient (1,100 cells/ μ L with 60% neutrophils) met the International Society for Peritoneal Dialysis (ISPD) criteria for PD-associated peritonitis (effluent white cell count $>100/\mu$ L with $>50\%$ polymorphonuclear cells), despite negative PD fluid cultures—a pattern of culture-negative peritonitis reported in up to 20% of episodes in the literature. In this case, temporary cessation of CAPD, transition to hemodialysis via an available central venous route, hemodynamic support with blood products, and empiric intraperitoneal antibiotic coverage administered in line with ISPD peritonitis treatment guidance permitted clinical stabilization and preservation of the option to resume peritoneal dialysis. This approach mirrors the conservative, supportive strategies described in the two previously published cases, both of which similarly avoided surgical intervention and achieved resolution with observation and correction of coagulation and volume status [2,3].

This case adds to the limited existing literature on retroperitoneal hemorrhage presenting as hemoperitoneum in ADPKD patients on peritoneal dialysis and reinforces the importance of considering this rare etiology in the differential diagnosis of hemorrhagic dialysate, particularly given the vascular fragility and structural complexity of the polycystic kidney. Timely recognition of abdominal pain and hemorrhagic peritoneal dialysate in such patients is essential, as it may herald a rare retroperitoneal source of hemoperitoneum; a high index of suspicion, prompt multidisciplinary evaluation, and supportive management can help salvage both the patient and the peritoneal dialysis modality.

Conclusion

Retroperitoneal hemorrhage should be considered in the differential diagnosis of hemoperitoneum in ADPKD patients on peritoneal dialysis, even without demonstrable retroperitoneal-

to-peritoneal communication. Prompt recognition and supportive multidisciplinary management may help preserve the peritoneal dialysis modality.

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