
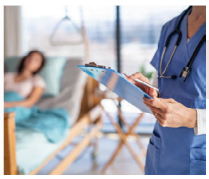

CASE REPORT

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Hypokalemia and Hemoperitoneum from Retrograde Menstruation in a Woman with Peritoneal Dialysis — A Case Report

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ABSTRACT

Introduction: Hypokalemia in peritoneal dialysis (PD) can be caused by dialysate potassium loss, low intake of potassium-rich foods, or gastroenteritis. Hemoperitoneum is a non-mechanical complication of peritoneal dialysis, common in menstruating women. **Case:** A 52-year-old woman with end stage renal disease (ESRD) on PD was admitted to the emergency department with persistent vomiting and bloody dialysate. The patient had a history of type 2 diabetes, controlled with antidiabetic drugs. Her serum potassium was 2.0 mEq/L. Ultrasound showed simple ovarian cysts and uterine myomas. She was diagnosed with severe hypokalemia, hemoperitoneum, type 2 diabetes, and ESRD. The treatment included oral and intravenous potassium chloride, tranexamic acid, and frequent dialysate changes. After a one-month follow-up, no complications were reported. **Discussion:** The hypokalemia was likely associated with gastrointestinal potassium loss and inadequate intake of potassium-rich foods. The temporal relationship between menstruation and bloody dialysate, together with the absence of cyst rupture or peritonitis, supported retrograde menstruation as the probable cause of hemoperitoneum. **Conclusion:** Hypokalemia may result from gastrointestinal losses, inadequate potassium intake, while menstruation might cause bloody dialysate in PD.

Keywords: Case report, end-stage renal disease, hemoperitoneum, hypokalemia, peritoneal dialysis, retrograde menstruation.

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INTRODUCTION

Hemoperitoneum is one of the non-mechanical complications in peritoneal dialysis (PD) and is common in menstruating women. It is the presence of blood in the peritoneal dialysate effluent.[1] As little as 2 mL of blood can render dialysate blood-tinged and can be distressing for the patient. In a female patient, menstruation is the common cause of hemoperitoneum in PD. Two main mechanisms have been proposed to explain hemoperitoneum associated with menstruation. First, retrograde menstruation occurs when uterine contractions expel blood backward into the fallopian tubes and then into the peritoneal cavity. Second, endometrial deposits are shed into the peritoneal cavity.[2] Most bleeding is self-limited with rare complications.[2] A reproductive organ, such as an ovarian cyst rupture, can cause pathological hemoperitoneum. Other causes are solid abdominal organ rupture, for example, spleen rupture, idiopathic thrombocytopaenia purpura (ITP), or iatrogenic.[2]

Hypokalemia is also a common complication in PD. Three mechanisms have been postulated to explain the development of hypokalemia: potassium loss through bowel and dialysate, poor intake of potassium-rich foods, and intracellular shift of potassium. Diarrhea and vomiting contribute to hypokalemia due to loss of potassium through the bowel.[3] A recent study showed the prevalence of potassium levels < 4.0, 3.5, and < 3.0 mmol/L in 37.9%, 17.7%, and 4.4% of PD patients, respectively.[3]

The simultaneous presentation of hemoperitoneum and hypokalemia in a PD patient is extremely rare and can pose significant diagnostic and therapeutic challenges, particularly in low-resource settings. These two conditions may initially present with non-specific symptoms such as abdominal discomfort, weakness, or nausea, leading to delays in diagnosis. Moreover, both complications, if not promptly recognized and managed, can compromise the effectiveness of PD and adversely affect patient outcomes. In women of reproductive age, it is essential to differentiate hemoperitoneum of gynecologic origin from other serious intra-abdominal causes. Persistent hypokalemia may lead to cardiac arrhythmias, muscle weakness, and even respiratory failure.[1,2]

A female PD patient with recurrent hypokalemia and simultaneous hemoperitoneum was presented. This is the first documented case in Indonesia detailing the diagnostic approach and management in a resource-limited setting. We obtained ethical approval from our institutional review board, and this case report adheres to the 2013 Case Report (CARE) guidelines.[4]

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CASE

A 52-year-old female was admitted to the emergency room with profuse vomiting for the past two days. She had a history of recurrent vomiting and diarrhea, leading to frequent hospitalizations. The patient was diagnosed with end-stage renal disease (ESRD) nine months ago. Initially, she underwent hemodialysis but was switched to PD after five months due to persistent hypotension and headaches during hemodialysis. Bloody fluid was also observed in her dialysate for approximately seven days. The patient still had her menstrual cycle, and there was vaginal bleeding during admission. The patient also had a history of heart failure and type 2 diabetes. Current medications included candesartan 8 mg once daily, bisoprolol 2.5 mg once daily, folic acid 1 mg once daily, calcitriol 0.25 mcg twice daily, gliclazide 60 mg once daily, potassium chloride 600 mg three times daily and detemir 10 units subcutaneous once daily. No significance of family and genetic history.

The patient's dietary history consists of a low intake of potassium-rich foods such as fruits and vegetables, likely due to persistent nausea, financial limitations, and dietary misconceptions. The patient reported eating rice primarily, with minimal protein and vegetable intake.

The patient appeared fatigued and pale on initial observation. Blood pressure was 80/50 mmHg, while other vital signs were within normal limits. Physical examination revealed anemic conjunctivae, sunken eyes, and poor skin turgor. Abdominal physical examination was normal, with no sign of peritonitis or palpable mass. The PD transfer set was attached to the patient's abdomen. The patient showed a sample of her blood-tinged dialysate during her dialysis time at home. There was fibrin clot formation in the dialysis catheter, but no signs of infection on the exit site.

The electrocardiogram (ECG) revealed incomplete left bundle branch block and left ventricular hypertrophy. Laboratory results showed a hemoglobin level of 9.8 g/dL and hematocrit of 27.1%. The white blood cell count was 7600/mm^3, with a differential of neutrophils 79%, eosinophils 1%, lymphocytes 17%, and monocytes 3%. The platelet count was 303,000/mm^3. Urea level was elevated at 193 mg/dL, and creatinine was 6.4 mg/dL, with an estimated glomerular filtration rate of 7 mL/min/1.73 m^2. Sodium level was 128 mEq/L, potassium was significantly low at 2.0 mEq/L, and ionized calcium was 0.86 mEq/L. The chloride level was 94 mEq/L, blood glucose was elevated at 347 mg/dL, and albumin was 2.6 g/dL. The diagnosis was severe hypokalemia due to vomiting, hemoperitoneum due to retrograde menstruation, type 2 diabetes with reactive hyperglycemia, hypoalbuminemia, and ESRD on PD.

In the emergency department, symptomatic treatment was given for vomiting, hyperglycemia, and menstrual bleeding. Ringer's lactate 500 mL was administered to correct fluid deficits; potassium chloride 50 mEq intravenous was given to treat hypokalemia; metoclopramide 10 mg intravenous and pantoprazole 40 mg intravenous were administered to control vomiting. Tranexamic acid 500 mg intravenous was given to manage bleeding, and rapid-acting insulin analog 10 units intravenous was administered to correct hyperglycemia. The patient was admitted and referred to an internist for further evaluation and management. The patient was also referred to an obstetrician and gynecologist to search for any pathological cause of menstruation. Abdominal ultrasound showed a left small simple ovarian cyst, two intramural uterine myomas, abnormal chronic renal parenchyma, and slight ascites.

During hospitalization, potassium levels were corrected at a rate of 0.625 mEq/day using intravenous potassium chloride (50 mEq/day), supplemented with oral potassium chloride 600 mg three times daily. Dialysate for CAPD was changed four times daily to resolve the hemoperitoneum, and by the third day of care, vomiting, diarrhea, and hemoperitoneum had resolved. Her serum potassium level was normalized. Hyperglycemia was managed with a daily subcutaneous injection of 10 units of long-acting (basal) insulin (Detemir®) and a once-daily 60 mg dose of oral gliclazide. The patient was discharged on the fourth day of hospitalization with instructions to continue routine medications, including potassium supplementation and oral antidiabetic therapy.

At the follow-up visit one week after discharge in the outpatient clinic, the patient still had nausea, without any vomiting. No other episode of bloody dialysate and diarrhea. Routine medications were maintained, and the patient was advised on medication adherence and potassium intake. At the one-month follow-up, serum electrolytes were within normal limits, and the patient demonstrated good adherence and tolerability to the prescribed medications. No adverse or unanticipated events were reported. Abdominal ultrasonography revealed stable findings, including a small ovarian cyst, intramural myomas, and minimal ascites, consistent with the previous examination.

DISCUSSION

A case of hemoperitoneum with recurrent hypokalemia in a woman undergoing peritoneal dialysis during active menstruation was presented. The etiology of recurrent hypokalemia remains inconclusive, primarily due to diagnostic limitations imposed by financial constraints and the restricted medical investigations covered by Indonesia's national health insurance.

The mechanism of recurrent hypokalemia in our patient is likely multifactorial. One plausible explanation is recurrent episodes of severe gastroenteritis. As reported by Kovačević, *et al.*, and Dhondup, *et al.*, recurrent gastroenteritis can lead to uremic syndrome, resulting in renal decompensation and metabolic alkalosis.[1,5] The alkalosis elevates bicarbonate levels in the bloodstream, promoting renal potassium excretion. Another contributing factor could be inadequate dietary potassium intake. Our patient reported significant dietary limitations, consuming mostly semi-solid foods such as rice and small amounts of meat, with minimal fruits or vegetables due to financial constraints and persistent nausea. These factors likely contributed to insufficient potassium intake.[6,7] A study by Pichitporn, *et al.*, and Dong, *et al.*, emphasized the importance of adequate dietary potassium intake in the recovery of hypokalemia in patients undergoing peritoneal dialysis.[8,9]


This case also presented with significant comorbidities, including heart failure and poorly controlled diabetes mellitus, complicating both the diagnostic approach and electrolyte management. Hyperglycemia may contribute to osmotic diuresis and worsening of intravascular volume depletion, further exacerbating hypokalemia. Despite these considerations, we could not definitively rule out other potential causes of recurrent hypokalemia, such as hyperaldosteronism or salt-wasting nephropathy.[10]

In this case, we corrected recurrent hypokalemia by addressing the symptoms of gastroenteritis and providing intravenous potassium chloride and oral potassium supplementation. This approach aligns with the findings of DeCarolis, *et al.,* who highlighted the cost-effectiveness of oral potassium supplementation over intravenous therapy.[11] While spironolactone is often used to reduce potassium excretion and lower blood pressure in similar cases, it was not indicated in our patient, as her blood pressure was low during initial admission.[1]

Hemoperitoneum with active menstrual bleeding in this patient was benign and resolved after frequent changes of dialysate. Abdominal ultrasound found a simple small cyst on her left ovary, two intramural myomas, chronic renal parenchymal abnormalities, and slight ascites. The ultrasound findings suggest that the ovarian cyst was simple rather than hemorrhagic, and the myomas were intramural, making them less likely to cause the hemoperitoneum.[12,13] Our proposed mechanism for the hemoperitoneum in this patient is retrograde menstruation. Around 10%–12% of hemoperitoneum cases in premenopausal women undergoing PD are caused by menstruation.[14] A study by Harnett, *et al.,* found four PD women with recurrent hemoperitoneum related to retrograde menstruation and rupture of ovarian cysts.[12] Another study by Ozlu, *et al.,* found similar benign hemoperitoneum in a 17-year-old woman, and the bleeding resolved after frequent dialysate changes.[2]

In this case, the temporal correlation between the onset of menstruation and the appearance of bloody dialysate, in the absence of infection or procedural trauma, strongly supports retrograde menstruation as the underlying mechanism. Moreover, the rapid resolution of hemoperitoneum following menstruation and the absence of cyst rupture on ultrasound further reinforce this benign etiology.[15]

Management was guided by clinical judgment and the available resources. The decision to continue PD rather than temporarily switch to hemodialysis was based on the absence of peritonitis, hemodynamic stability after initial resuscitation, and the patient's prior intolerance to hemodialysis. Frequent dialysate exchanges facilitated clearance of intraperitoneal blood, consistent with recommendations in similar cases.[6]

CONCLUSION

The case emphasizes two key clinical insights. First, hemoperitoneum in female PD patients, especially when temporarily associated with menstruation and without signs of peritonitis or trauma, may be a benign and self-limiting condition due to retrograde menstruation; and recurrent hypokalemia in PD patients is often multifactorial, commonly involving gastrointestinal losses, inadequate dietary intake, and comorbid conditions such as diabetes. Despite diagnostic limitations imposed by a resource-constrained healthcare system, this patient was successfully managed through symptom-based therapy, potassium supplementation, and close gynecologic follow-up. The case underscores the value of interdisciplinary collaboration among nephrologists, gynecologists, and internists, which also contributes to the global understanding of PD-related complications, particularly those involving hormonal and metabolic factors, and reinforces the role of clinical vigilance and context-specific decision-making in achieving positive outcomes.

Consent for Publication

Informed consent for publication of this case report was obtained from the patient.

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Author Contributions

W.S., H.H., R.S.Y.W., D.S., and A.E. contributed to the case report conception and design, collected data, and wrote the first draft of the manuscript. All authors reviewed and approved the manuscript.

Conflict of Interest

There is no conflict of interest.

Declaration on the Use of Artificial Intelligence (AI)

The authors declare that no artificial intelligence tools were used in the conduct of this study or preparation of this manuscript.

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REFERENCES

1. Kovacevic I, Sefer S, Ratkovic-Gusic I, Degoricija V, Szavits-Nossan J. Colonic pseudo-obtsruction and severe hypokalemia in an anuric diabetic patient on hemodialysis: case report and review of the literature. Acta Clin Croat. 2006;45(3):193–7.
2. Ozlu SG, Gokce AO, Inozu M, Bayrakci US. Hemoperitoneum related with peritoneal dialysis in a female adolescent: it is not as frightening as it seems. Turkiye Cocuk Hast Derg. 2024;18(2):143–5. doi: 10.12956/tchd.1389383.
3. Yang C, Hu X, Ling X, Xiao C, Duan R, Qiu J, et al. Hypokalemia in peritoneal dialysis: a systematic review and meta-analysis of prevalence, treatment, and outcomes. Kidney Med. 2024;6(12):100923. doi: 10.1016/j.xkme.2024.100923.
4. Riley D, Hao JJ, Kiene H, Kienle G, Mittelman M, Plotnikoff GA. Global advances in health and medicine. Glob Adv Health Med. 2012;1(1):5–7. doi: 10.7453/gahmj.2012.1.1.001.


5. Dhondup T, Qian Q. Acid-base and electrolyte disorders in patients with and without chronic kidney disease: an update. Kidney Dis (Basel). 2017;3(4):136–48. doi: 10.1159/000479968.
6. Lin KK, Lee JJ, Chen HC. Severe refeeding hypophosphatemia in a CAPD patient: a case report. Ren Fail. 2006;28(6):515–7. doi: 10.1080/08860220600767228.
7. Ramakrishna CH, Kumar KS, Padmnabhan S, Kumar VS. Hyponatremia and hypokalemia in a continuous ambulatory peritoneal dialysis patient. Indian J Nephrol. 2007;17(1):20–2. doi: 10.4103/0971-4065.35017.
8. Pichitporn W, Kanjanabuch T, Phannajit J, Puapatanakul P, Chuengsaman P, Parapiboon W, et al. Efficacy of potassium supplementation in hypokalemic patients receiving peritoneal dialysis: a randomized controlled trial. Am J Kidney Dis. 2022;80(5):580–8.e1. doi: 10.1053/j.ajkd.2022.03.013.
9. Dong R, Guo ZY. Gastrointestinal symptoms in patients undergoing peritoneal dialysis: multivariate analysis of correlated factors. World J Gastroenterol. 2010;16(22):2812–7. doi: 10.3748/wjg.v16.i22.2812.
10. De Jaka PS, Alantus DG. Does severe hypokalemia worsen the outcome of diabetic ketoacidosis. InaJEMD. 2024;1(1):25–31. https://doi.org/10.66266/inajemd.v1i1.4.
11. DeCarolis DD, Kim GM, Rector TS, Ishani A. Comparative dose response using the intravenous versus enteral route of administration for potassium replenishment. Intensive Crit Care Nurs. 2016;36:17–23. doi: 10.1016/j.iccn.2015.08.005.
12. Harnett JD, Gill D, Corbett L, Parfrey PS, Gault H. Recurrent hemoperitoneum in women receiving continuous ambulatory peritoneal dialysis. Ann Intern Med. 1987;107(3):341–3. doi: 10.7326/0003-4819-107-2-341.
13. Stolz A, Fourcade J, Klisnick A, Souweine B, Abergel A, Baguet JC, et al. Hemoperitoneum in patients receiving hemodialysis. Am J Kidney Dis. 2000;36(2):E11. doi: 10.1053/ajkd.2000.9010.
14. Lew S. Hemoperitoneum: bloody peritoneal dialysate in esrd patients receiving peritoneal dialysis. Perit Dial Int. 2007;27(3):226–33. doi: 10.1177/089686080702700303.
15. Greenberg A, Bernardini J, Piraino BM, Johnston JR, Perlmutter JA. Hemoperitoneum complicating chronic peritoneal dialysis: single-center experience and literature review. Am J Kidney Dis. 1992;19(3):252–6. doi: 10.1016/s0272-6386(13)80006-6.