Ukrainian Journal of Nephrology and Dialysis
Scientific and Practical, Medical Journal

**Founder:**
- National Kidney Foundation of Ukraine

**ISSN 2304-0238;**
**eISSN 2616-7352**

**Journal homepage: https://ukrjnd.com.ua**

**Case Report**

**doi: 10.31450/ukrjnd.3(91).2026.01**

**Ramadi Satryo Wicaksono[1], Johanes Aprilius Falerio Kristijanto[1,2]**

# Post-traumatic intravesical clot retention causing recurrent post-renal acute kidney injury in a pregnant woman: A case report

[1]Department of Internal Medicine, Bangil Regional General Hospital, Pasuruan, Indonesia
[2]Faculty of Medicine, Universitas Wijaya Kusuma Surabaya, Surabaya, Indonesia

Citation: Wicaksono RS, Kristijanto JAF. Post-traumatic intravesical clot retention causing recurrent post-renal acute kidney injury in a pregnant woman: A case report. Ukr J Nephrol Dialys. 2026;3(91):3-8. doi: 10.31450/ukrjnd.3(91).2026.01.

**Abstract.** *Intravesical blood clot obstruction is a recognized cause of postrenal acute kidney injury (AKI), but recurrent obstruction after apparent renal recovery may be difficult to recognize. We report a 30-year-old pregnant woman at approximately 27 weeks' gestation who developed severe AKI after a motor vehicle accident with gross hematuria. At the first admission, serum creatinine was 8.210 mg/dL, and ultrasonography showed bilateral pelvicalyceal ectasia without a definite obstructing lesion. After bladder drainage, renal function rapidly improved, with serum creatinine decreasing to 1.130 mg/dL without hemodialysis. Ten days later, the patient was readmitted with recurrent gross hematuria, anuria, hyperkalemia, metabolic acidosis, and recurrent AKI. Repeat ultrasonography demonstrated a large intravesical blood clot, supporting clot-related obstruction as the most likely mechanism. Repeat bladder drainage and one emergency hemodialysis session were followed by marked renal recovery. The subsequent course was complicated by fetal loss, postpartum hemorrhage, puerperal sepsis, multiorgan dysfunction, and death.*

*The clinical importance of this case lies in the recurrence of severe postrenal AKI after near-complete renal recovery. In patients with trauma-associated gross hematuria, rapid improvement after bladder drainage does not exclude persistent or recurrent clot-related obstruction. Repeat urinary tract imaging should be considered when hematuria, anuria, or kidney dysfunction recurs, particularly when initial imaging shows upper urinary tract dilatation without a clear cause.*

**Keywords:** *acute kidney injury, hematuria, urinary tract obstruction, hydronephrosis, pregnancy complications.*

**Article history:**
*Received May 05, 2026*
*Received in revised form June 09, 2026*
*Accepted June 18, 2026*

**Conflict of interest.** The author declares no conflict of interest.



Correspondence should be addressed to Ramadi Satryo Wicaksono:
ramadi_satryo@yahoo.com

УДК: 616.61/63-007.272:616.61-008.6-036.11]:618.3-06

**Рамаді Сатріо Вічаксоно[1], Йоханес Апріліус Фалеріо Крістіянто[1,2]**

# Посттравматична тампонада сечового міхура згустками крові як причина рецидивного постренального гострого пошкодження нирок у вагітної: клінічний випадок

[1]Відділення внутрішньої медицини, Регіональна загальна лікарня Бангіл, Пасуруан, Індонезія
[2]Медичний факультет Університету Віджая Кусума Сурабая, Сурабая, Індонезія

**Резюме.** *Обструкція сечових шляхів згустками крові в сечовому міхурі є відомою причиною постренального гострого пошкодження нирок (ГПН), однак повторну обструкцію після очевидного відновлення функції нирок можна своєчасно не діагностувати. Представлено клінічний випадок 30-річної вагітної на терміні близько 27 тижнів, у якої після дорожньо-транспортної пригоди з макрогематурією розвинулося тяжке ГПН. Під час першої госпіталізації рівень креатиніну становив 8,21 мг/дл, а ультразвукове дослідження виявило двобічне розширення чашково-мискової системи без чіткої причини обструкції. Після дренування сечового міхура функція нирок швидко покращилася, рівень креатиніну знизився до 1,13 мг/дл без проведення гемодіалізу. Через десять днів пацієнтку повторно госпіталізували з макрогематурією, анурією, гіперкаліємією, метаболічним ацидозом і рецидивним ГПН. Повторне ультразвукове дослідження виявило великий згусток крові в сечовому міхурі, що підтверджувало обструкцію згустком як найбільш імовірний механізм ГПН. Після повторного дренування сечового міхура та одного ургентного сеансу гемодіалізу функція нирок суттєво покращилася. Подальший перебіг ускладнився втратою плода, післяпологовою кровотечею, післяпологовим сепсисом, поліорганною недостатністю та летальним наслідком.*

*Клінічна особливість цього випадку полягає в рецидиві тяжкого постренального ГПН після майже повного відновлення функції нирок. У пацієнтів з посттравматичною макрогематурією швидке покращення після дренування сечового міхура не виключає стійкої або повторної обструкції згустками крові. Рецидив макрогематурії, анурія або повторне погіршення функції нирок потребують повторної візуалізації сечових шляхів, особливо за наявності дилатації верхніх сечових шляхів без встановленої причини.*

**Ключові слова:** *гостре пошкодження нирок, гематурія, обструкція сечових шляхів, гідронефроз, ускладнення вагітності.*

**Introduction.** Postrenal acute kidney injury (AKI) is a potentially reversible but time-sensitive renal emergency resulting from obstructive uropathy, in which impaired urinary drainage increases upstream pressure within the urinary tract [**1**]. Although obstructive uropathy is commonly caused by calculi, malignancy, strictures, neurogenic bladder, or prostatic disease, intravesical blood clot retention represents an uncommon cause of lower urinary tract obstruction [**2**]. In patients with pelvic trauma, hematuria, bleeding tendency, or coagulopathy, intravesical clot burden may obstruct urinary outflow, causing urinary retention, hydronephrosis, and abrupt deterioration of renal function. Recognition may be particularly challenging in gravid or peripartum patients, in whom AKI is often attributed to hemodynamic, infectious, or obstetric complications [**3**]. We report a fatal case of recurrent obstructive uropathy-induced postrenal AKI caused by post-traumatic intravesical blood clot retention in a gravid woman later complicated by retained placenta and puerperal sepsis.

**Ramadi Satryo Wicaksono**
**ramadi_satryo@yahoo.com**

**Case report.** A 30-year-old gravid woman at approximately 27 weeks' gestation first presented to our emergency department on 16 July 2025 after a single motor vehicle accident, with weakness, dyspnea, and gross hematuria. Initial laboratory evaluation showed hemoglobin 9.56 g/dL, leukocytes 13.28 $\times 10^3$/μL, BUN 75.99 mg/dL, serum creatinine 8.210 mg/dL, sodium 128.8 mmol/L, and potassium 4.84 mmol/L. Urinalysis revealed marked proteinuria, hematuria, leukocyturia, nitrituria, and bacteriuria, with erythrocytes >10,000/HPF. Given the trauma-related gross hematuria and suspected urinary outflow obstruction, three-way bladder catheter drainage was performed. Grossly bloody urine was initially drained and followed by progressive clearing of the urine. Abdominal ultrasonography demonstrated bilateral pelvicalyceal ectasia (Fig. 1), suggestive of bilateral hydronephrosis, but no definite obstructing pathology was identified along the urinary tract; cholelithiasis with gallbladder sludge was also noted. Hemodialysis was not performed during this first admission. After urinary drainage and ceftriaxone therapy for urinary tract infection, renal function improved by 18 July 2025, with BUN decreasing to 40.50 mg/dL and serum creatinine to 1.130 mg/dL. The patient remained clinically stable and was discharged on 21 July 2025.

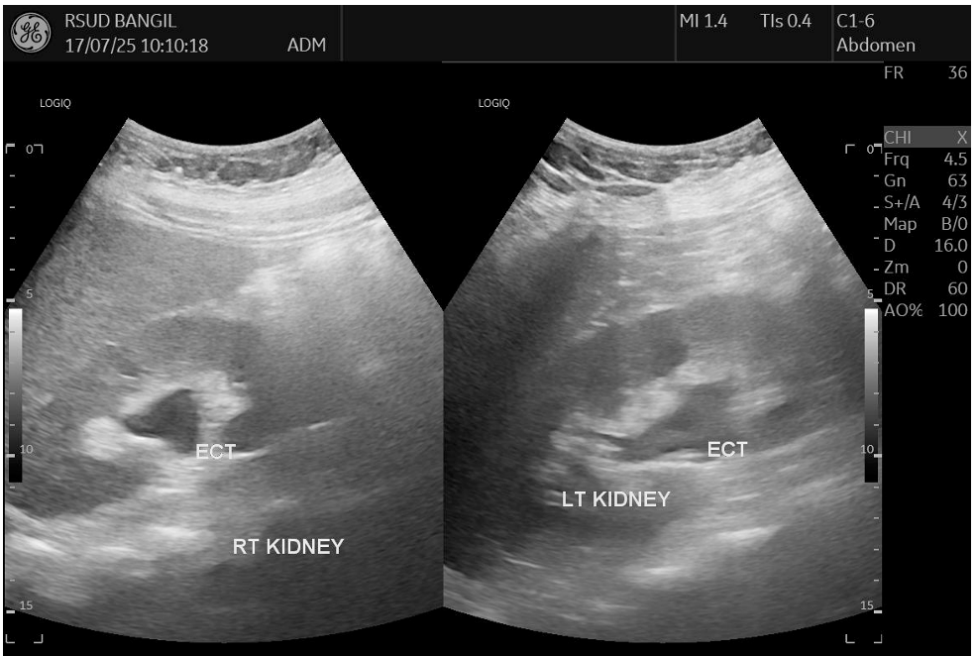


Fig. 1. First-admission abdominal ultrasonography showing bilateral pelvicalyceal ectasia suggestive of hydronephrosis.

On 31 July 2025, the patient was readmitted with recurrent gross hematuria, recurrent AKI, and hyperkalemia. Laboratory evaluation showed hemoglobin 8.87 g/dL, leukocytes 31.37 ×103/μL, BUN 87.60 mg/dL, serum creatinine 5.000 mg/dL, sodium 130 mmol/L, and potassium 6.00 mmol/L. Repeat urinalysis demonstrated persistent proteinuria, hematuria, leukocyturia, and marked bacteriuria. Obstetric ultrasonography performed on 1 August 2025 confirmed a live intrauterine fetus and demonstrated a large intravesical blood clot (Fig. 2), providing radiologic evidence of clot retention. Together with recurrent gross hematuria, prior bilateral pelvicalyceal ectasia, and subsequent improvement after bladder drainage, this finding supported clot-related obstructive uropathy as the most likely mechanism of recurrent postrenal AKI.

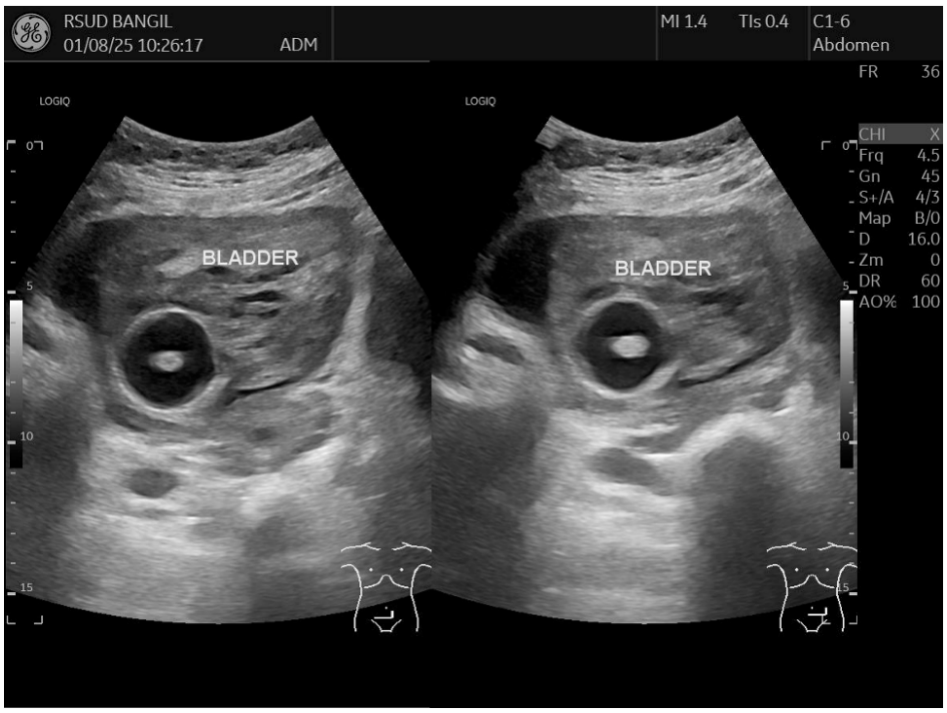


Fig. 2. Abdominal ultrasonography showing a large intravesical blood clot. Echogenic intraluminal material was observed within the urinary bladder, consistent with clot retention and supporting obstructive uropathy as the mechanism of recurrent postrenal acute kidney injury.

On the same day, hyperkalemia worsened, with potassium increasing to 6.20 mmol/L, and arterial blood gas analysis showed metabolic acidosis, with pH 7.30, bicarbonate 10.6 mmol/L, and base excess –16. Renal function further deteriorated, with BUN increasing to 133.98 mg/dL and serum creatinine to 5.895 mg/dL, accompanied by anuria. Given the combination of recurrent AKI, anuria, metabolic acidosis, and hyperkalemia, a single session of emergency hemodialysis was performed, together with repeat three-way bladder drainage and supportive nephrological management. Cystoscopy and formal clot evacuation were not performed because of the patient's gravid condition; therefore, management was limited to three-way bladder drainage, one session of emergency hemodialysis during this second admission, and supportive nephrological care. Renal function subsequently improved, with BUN decreasing to 49.10 mg/dL and serum creatinine to 1.540 mg/dL. The patient was discharged on 9 August 2025 in clinically improved condition, with BUN 52.85 mg/dL and serum creatinine 1.789 mg/dL.

On 14 August 2025, the patient delivered a nonviable fetus at home. The placenta was not expelled within 30 minutes, and she was subsequently referred for retained placenta requiring manual placental removal. She was readmitted later that day in critical condition with postpartum hemorrhage, severe anemia, recurrent AKI, hyperkalemia, hypoglycemia, and profound metabolic acidosis. Laboratory evaluation on admission showed hemoglobin 6.70 g/dL, leukocytes 29.04 $\times 10^3$/μL, BUN 116.73 mg/dL, serum creatinine 5.057 mg/dL, sodium 128.50 mmol/L, potassium 5.58 mmol/L, glucose 50 mg/dL, and albumin 3.0 g/dL. Arterial blood gas analysis demonstrated severe metabolic acidosis and hypoxemia, with pH 7.18, $pCO_2$ 17.7 mmHg, $pO_2$ 33.0 mmHg, bicarbonate 6.8 mmol/L, total $CO_2$ 7.0 mmol/L, base excess –21, and oxygen saturation 52.0%.

Chest radiography obtained during this admission showed pneumonia. Given suspected puerperal sepsis and pneumonia, broad-spectrum antibiotic therapy, including meropenem, was administered. Follow-up laboratory assessment on 15 August 2025 showed potassium decreased to 3.70 mmol/L. Repeat blood gas analysis demonstrated persistent metabolic acidosis with respiratory compensation, with pH 7.43, $pCO_2$ 12.8 mmHg, bicarbonate 8.6 mmol/L, total $CO_2$ 9.0 mmol/L, and base excess –16. At this stage, hemodialysis was not performed because of profound anemia and hemodynamic instability.

During the third admission, the patient developed progressive respiratory failure, puerperal sepsis, coagulopathy consistent with disseminated intravascular coagulation, refractory shock, and multiorgan dysfunction. On 17 August 2025, she required intubation; laboratory evaluation showed hemoglobin 4.60 g/dL, leukocytes 19.02 $\times 10^3$/μL, platelets 120 $\times 10^3$/μL, prolonged activated partial thromboplastin time of 54.80 seconds, prolonged prothrombin time of 23.80 seconds, procalcitonin >50 ng/mL, C-reactive protein >200 mg/dL, and albumin 2.0 g/dL. Despite intensive supportive care, her condition deteriorated further, and she died on 18 August 2025 after cardiac arrest in the setting of septic shock and multiorgan dysfunction. The overall clinical course, including serial BUN and serum creatinine changes, major interventions, and key clinical events, is summarized in Figure 3.

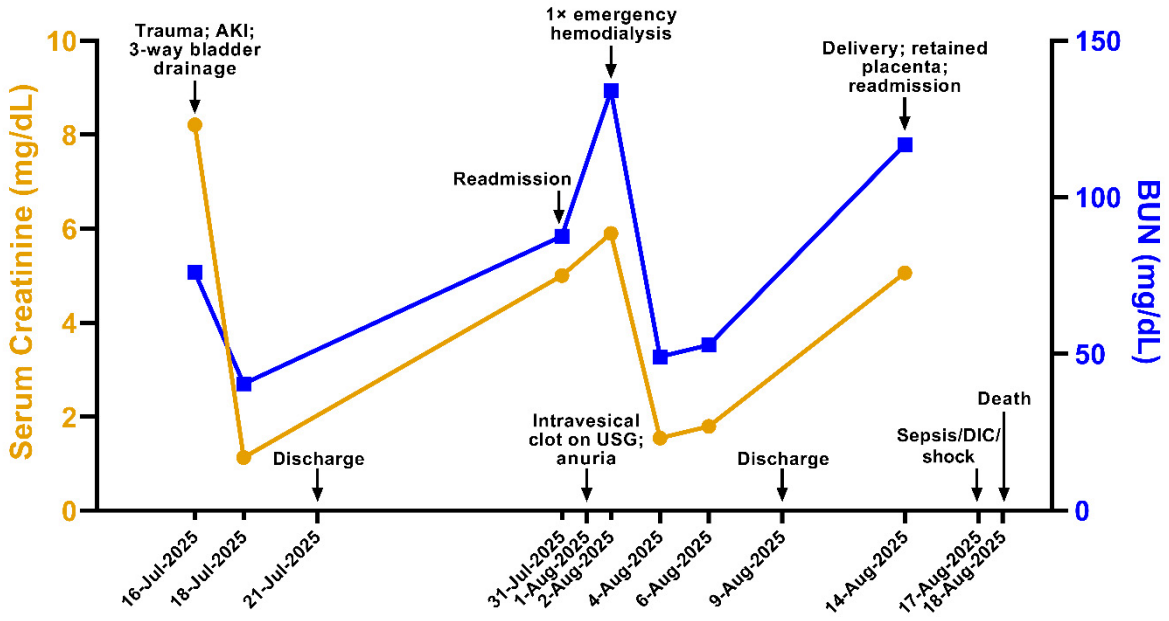


Fig. 3. Clinical course of recurrent postrenal acute kidney injury and subsequent systemic deterioration. Serial BUN and serum creatinine values demonstrate recurrent renal dysfunction with partial recovery after urinary drainage and a single emergency hemodialysis session, followed by recurrent AKI during postpartum septic deterioration. Key clinical events are annotated along the timeline, including trauma, intravesical clot confirmation with anuria, delivery with retained placenta, sepsis/DIC/shock, and death.

**Discussion.** We present a rare case of recurrent postrenal AKI caused by intravesical blood clot retention after trauma-related gross hematuria in a gravid patient. The diagnosis was supported by a relapsing clinical pattern: initial severe azotemia with bilateral pelvicalyceal ectasia improved after three-way bladder drainage, followed by recurrent gross hematuria, anuria, hyperkalemia, metabolic acidosis, and repeat ultrasonographic visualization of a large intravesical clot during the second admission. Although urinary retention volume, formal urology documentation, and cystoscopic confirmation were not available, the convergence of recurrent hematuria, anuria, hydronephrosis-suggestive pelvicalyceal ectasia, radiologic clot visualization, and partial renal recovery after bladder drainage and supportive nephrological care supports clot-related obstructive uropathy as the most likely mechanism of recurrent postrenal AKI. Intravesical clot retention can impair bladder emptying, increase upstream urinary tract pressure, reduce glomerular filtration, and precipitate electrolyte disturbance, metabolic acidosis, and abrupt renal deterioration [1]. Obstructive uropathy is a potentially reversible cause of AKI, accounting for approximately 5–10% of acute renal failure cases, and timely decompression remains central to renal recovery [2, 4]. In this case, the recurrent rise and fall of BUN and serum creatinine after drainage, one emergency hemodialysis session for metabolic acidosis and hyperkalemia, and supportive care was more consistent with a recurrent postrenal process than with isolated intrinsic renal injury.

An important clinical lesson from this case is that apparent improvement after bladder drainage should not be equated with complete resolution of clot-related obstruction. During the first admission, the transition from grossly bloody to clearer urine and the improvement in renal function suggested clinical stabilization; however, recurrent AKI with anuria and subsequent ultrasonographic confirmation of a large intravesical clot indicated that residual or recurrent clot burden may have persisted or re-formed. This observation suggests that patients with trauma-related gross hematuria and postrenal AKI may require serial reassessment before discharge, including careful monitoring of urine output, catheter drainage characteristics, renal function, and repeat ultrasonography when obstruction remains clinically plausible. Earlier confirmation that the bladder was free of recurrent clot might have supported prolonged observation or earlier re-intervention and may have reduced the risk of recurrent obstructive AKI, although causality and the potential effect on fetal or maternal outcome cannot be established from a single case.

The gravid condition added substantial diagnostic and prognostic complexity. Pregnancy-associated AKI is a heterogeneous syndrome that may result from hemorrhage, sepsis, hypertensive disorders, thrombotic microangiopathy, and other pregnancy-related complications; therefore, its underlying renal mechanism should be defined carefully rather than attributing renal dysfunction solely to obstetric illness [3]. Maternal AKI is associated with adverse pregnancy outcomes, including preterm birth, fetal growth restriction, stillbirth, and perinatal death [5]. In this case, recurrent AKI was accompanied by severe azotemia, hyperkalemia, metabolic acidosis, and systemic deterioration before the subsequent delivery of a nonviable fetus. These abnormalities may have contributed to an unfavorable fetal environment through impaired maternal metabolic homeostasis, uremia, electrolyte disturbance, acid-base imbalance, volume instability, and reduced uteroplacental reserve [6]. However, a direct causal relationship between recurrent obstructive AKI and fetal death cannot be established from a single case.

The final deterioration reflected the transition from a potentially reversible postrenal process to systemic critical illness. After delivery of a nonviable fetus at home, the patient developed retained placenta requiring manual removal, postpartum hemorrhage, severe anemia, recurrent AKI, profound metabolic acidosis, pneumonia, puerperal sepsis, and coagulopathy consistent with disseminated intravascular coagulation. Obstetric DIC is a life-threatening complication that may arise from pregnancy- and puerperium-related disorders, particularly severe infection and hemorrhagic complications [7]. During this final admission, hemodialysis was not performed because profound anemia and hemodynamic instability limited the feasibility of renal replacement therapy. The subsequent development of respiratory failure, refractory shock, multiorgan dysfunction, and death indicates that the outcome was ultimately determined not only by recurrent obstructive AKI, but also by severe septic, hemorrhagic, and coagulopathic deterioration.

This case has several limitations. Cystoscopy and formal clot evacuation were not performed because of the patient's gravid condition and clinical context, and urinary retention volume was not documented. Nevertheless, the serial clinical course, catheter drainage findings, anuria during recurrence, ultrasound evidence of bilateral pelvicalyceal ectasia and later intravesical clot, and partial renal recovery after drainage provide clinically meaningful support for recurrent obstructive uropathy. Therefore, the novelty of this case lies not only in the rare etiology of intravesical blood clot–induced postrenal AKI during pregnancy, but also in the need to confirm sustained resolution of obstruction before discharge when recurrent clot formation remains clinically possible. Early multidisciplinary assessment involving nephrology, urology, obstetrics, and critical care is essential when AKI occurs with trauma-related hematuria during pregnancy, because timely recognition of a treatable obstructive component may allow renal recovery before maternal systemic complications and fetal compromise become irreversible.

**Conclusions.** Intravesical blood clot retention is a rare but clinically important cause of recurrent postrenal AKI and should be considered in patients

with trauma-related gross hematuria, anuria, hydronephrosis-suggestive imaging, and relapsing renal dysfunction. This case emphasizes that apparent improvement after bladder drainage does not necessarily exclude residual or recurrent clot-related obstruction; therefore, serial reassessment of urine output, catheter drainage, renal function, and repeat imaging may be warranted before discharge. Timely recognition and sustained confirmation of obstruction resolution are essential, as renal recovery may be possible before recurrent AKI is compounded by fetal compromise, puerperal sepsis, coagulopathy, shock, and multiorgan dysfunction.

**Ethics statement.** The authors certify that they have obtained all appropriate patient consent.

**Conflicts of interest statement.** The authors have no conflicts of interest to declare.

**Funding sources.** This study was not supported by any sponsor or funder.

**Authors' contributions.**

***Ramadi Satryo Wicaksono:*** contributed to case conceptualization, clinical supervision, interpretation of clinical data, critical revision of the manuscript, and final approval of the manuscript.

***Johanes Aprilius Falerio Kristijanto:*** contributed to data collection, literature review, manuscript drafting, figure preparation, manuscript revision, and final approval of the manuscript. Both authors read and approved the final version of the manuscript.

**Data availability statement.** The data supporting the findings of this case report are available from the corresponding author upon reasonable request. However, access to patient-level data may be restricted to protect patient privacy and confidentiality.

**Use of artificial intelligence.** During the preparation of this manuscript, the authors used ChatGPT (OpenAI, Version 4.0) to assist with language refinement, text consistency, and paragraph structure. The authors reviewed and verified all AI-edited content to ensure it accurately reflects their original ideas and take full responsibility for the academic integrity of the work.

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