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Atypical postpartum decline: A diagnostic conundrum

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Abstract

Postpartum clinical deterioration can occasionally mask uncommon but serious underlying conditions. We present the case of a 29-year-old multiparous woman who developed abdominal distension, fever and worsening clinical status following an uneventful vaginal delivery. Initially managed as puerperal sepsis, her atypical course prompted further evaluation, ultimately revealing a bladder rent requiring surgical intervention. Prompt surgical repair led to complete recovery. Since its manifestations often mimic puerperal sepsis, recognition is frequently delayed, increasing the risk of morbidity. This report highlights key clinical indicators, the diagnostic utility of ascitic-fluid biochemical analysis (including ascitic creatinine values), the importance of timely surgical decision making and need for a high index of suspicion for visceral injury in preventing morbidity in cases of atypical postpartum decline ^[1-10].

Keywords: Spontaneous bladder rupture, puerperal sepsis mimic, postpartum complications, fundal bladder rent, peritonitis

Introduction

The postpartum period is usually characterized by recovery and physiological adaptation; however, serious complications may arise unexpectedly that may mimic commonly encountered postpartum infections. Such conditions often present with nonspecific features such as abdominal distension, fever, declining urine output, or unexplained ascites, posing a diagnostic challenge. Thorough evaluation and timely intervention are essential to reduce associated morbidity.

Case Report

Mrs. X, a 29-year-old multiparous woman (P2L2) with an uneventful antenatal course, delivered a healthy male baby weighing 3 kg by full-term vaginal delivery with right mediolateral episiotomy on 3 August 2024 at a peripheral hospital. She was discharged on the third postpartum day after passing urine and stools normally. On the sixth postpartum day, she presented to the same hospital with fever, abdominal pain, and foul-smelling vaginal discharge for two days. Laboratory investigations revealed leukocytosis (WBC 24,220/mm³), and ultrasonography showed an overdistended uterus with heterogeneous endometrium. A diagnosis of puerperal sepsis was made, and she was started on intravenous antibiotics. On the following day, she developed urinary retention, requiring catheterization, and was referred to our hospital on the evening of 10 August 2024 (postpartum day 7) with a catheter in situ. At admission, she was febrile and tachycardic (pulse rate 118/min, BP 110/70 mmHg, temperature 100 °F) with mild abdominal distension. Local examination revealed a gaping episiotomy wound with foul discharge. Per-vaginal examination showed a contracted uterus (~20 weeks' size) with foul-smelling discharge. A provisional diagnosis of puerperal sepsis was made. She was started on intravenous Piperacillin-Tazobactam and Metronidazole with supportive care. Investigations showed hemoglobin 10.3 g/dL, WBC 19,950/mm³, and creatinine 1.25 mg/dL. Urine output was normal. Initially, her condition improved, becoming afebrile with adequate urine output. However, on the twelfth postpartum day, she developed recurrent fever and progressive abdominal distension. Ultrasound revealed multiple small hypoechoic areas in the spleen, septated periuterine collections, and pelvic fluid. CECT abdomen demonstrated multiple loculated abdominopelvic collections, peritonitis, splenic abscesses, hepatomegaly, and mild enteritis, suggesting an infective pathology. The patient continued to deteriorate with hypotension, tachycardia, hematuria, and worsening distension despite broad-spectrum antibiotics. Ascitic fluid analysis revealed elevated protein (2.3 g/dL) and low sugar (< 10 mg/dL). A multidisciplinary decision was made for emergency exploratory laparotomy on the thirteenth postpartum day. Intraoperatively, approximately 600 mL of pus and blood was drained. The abdomen showed dense adhesions with necrotic tissue between the bladder and uterus. A rent at the fundus of the bladder extending posteriorly was identified, with associated pyoperitoneum and peritoneal inflammation. The uterus and right adnexa appeared normal, while the left adnexa could not be visualized due to adhesions. Bladder repair was performed in two layers after thorough peritoneal lavage, and a suprapubic catheter was inserted.

The postoperative period was uneventful except for a minor wound infection, managed conservatively. She was extubated on postoperative day 5, and the catheter was kept in situ for 15 days. The patient made a full recovery.



Fig 1: Figure showing intra operative finding of bladder rent at fundus extending posteriorly 3x2cm²

Discussion

Postpartum visceral injuries presenting as sepsis-like illness remain diagnostically challenging. Spontaneous rupture of urinary bladder is an extremely uncommon cause of acute abdomen in the puerperium with an incidence of approximately 1 in 126,000 women [1]. The first documented case was described by Kiebel in 1995 [1]. The reported mortality approaches 25%, primarily due to diagnostic delay. Risk factors include bladder overdistension, infection, ischemic necrosis, prolonged labor, and postpartum atony [2, 3, 7]. Most patients present between 1-7 days postpartum with abdominal pain, distension, and fever. Stabile *et al.* [5] conducted a systematic review published in 2021, analyzing cases reported between 1990 and 2020, comprising approximately 48 postpartum cases documented worldwide. Their review emphasized the median time to presentation to be 3 days and identified abdominal pain, distension, and oliguria as dominant features.

Clinical diagnosis is often challenging because it mimics puerperal sepsis. Absence of hematuria and normal urine output can delay recognition [3, 4, 8, 9]. Pseudo-renal failure may occur when urine is absorbed from the peritoneal cavity, causing raised serum creatinine despite normal renal function [4, 8]. Ascitic-fluid creatinine and urea levels provide a rapid, inexpensive, and high-yield diagnostic tool for differentiating urine leakage from infective ascites. A high ascitic creatinine relative to serum creatinine strongly suggests urinary ascites. Qiao *et al.* [8] and Hadian *et al.* [9] highlighted that early estimation of ascitic-fluid creatinine is a simple and reliable diagnostic tool for differentiating spontaneous bladder rupture from infectious causes and advocate early ascitic testing as a lifesaving step, especially when imaging is inconclusive. Radiologic diagnosis is

frequently missed in early stages. While ultrasonography demonstrates fluid, it cannot identify rupture sites. CT cystography remains the most accurate modality [5, 8]. Farahzadi *et al.* [6] and Singh *et al.* [4] described cases where small or sealed fundal rents were missed due to inflammatory adhesions and bowel interference.

Management depends on rupture type and clinical stability. Intraperitoneal ruptures require surgical repair, whereas selected extraperitoneal tears may be treated conservatively [7, 8, 10]. Marcos da Silva Barroso, *et al.* [10] emphasized that prompt surgical intervention remains the mainstay for preventing sepsis and multiorgan failure.

Conclusion

Spontaneous bladder rupture, though rare, must be considered in postpartum women presenting with abdominal distension, persistent fever, or unexplained ascites. Voiding history and vigilant monitoring of postpartum urinary output are quintessential, as the absence of hematuria or normal renal parameters does not exclude the diagnosis. While the classical presentation includes new-onset ascites, suprapubic pain, anuria, and hematuria, atypical presentations may manifest as sepsis with pseudo-renal failure and no hematuria. Simple bedside analysis of ascitic-fluid creatinine, supported by timely cross-sectional imaging, can prevent fatal delays, and early surgical intervention, often necessitating laparotomy ensures excellent recovery. This case highlights that apparent postpartum sepsis may conceal a surgically correctable visceral injury, and emphasizes that subtle clinical clues, sound judgment, and evidence-based reasoning remain indispensable for identifying the true underlying cause.

Conflicts of interest

Not available

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Not available

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