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Case Report

A case report of postpartum hemolytic uremic syndrome

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Abstract: Postpartum hemolytic uremic syndrome (PHUS) is a rare thrombotic microangiopathy, and it poses significant diagnostic challenges due to its nonspecific presentation. Our department treated a patient with PHUS. Therefore, the aim of this article is to review and share this rare case treated in our department, providing a thorough understanding of the clinical manifestations and diagnostic criteria of PHUS which is critical for enabling timely therapeutic interventions. Informed consent was granted by the patient to share the case information.

Keywords: Postpartum hemolytic uremic syndrome, diagnosis, plasmapheresis

Introduction

Postpartum hemolytic uremic syndrome (PHUS) a life-threatening obstetric disorder characterized by the diagnostic triad of microangiopathic hemolytic anemia, thrombocytopenia, and acute renal failure, carries grave clinical prognoses [1]. PHUS is associated with a low morbidity rate and a very poor prognosis, with a high mortality rate during the acute phase. Furthermore, the majority of patients develop end-stage renal disease, necessitating long-term renal dialysis treatment. In this report, we present the case of a patient with PHUS who was successfully treated.

Case presentation

A 29-year-old female patient was admitted to the hospital with postpartum onset of yellowish skin discoloration and anuria. This nulliparous patient presented with an unremarkable medical history, including no documented hypertension, diabetes mellitus or other comorbidities during serial antenatal assessments. Prenatal laboratory evaluations revealed normal hepatic and renal function profiles. She subsequently achieved an uncomplicated full-term vaginal delivery of a viable neonate without peripartum complications. Vital signs on admission: temperature 36.3°C, blood pressure 130/80 mmHg, heart rate of 86 bpm, respiratory rate 20/min. Physical examination revealed pallor with generalized icterus, absence of palmar erythema or spider angiomata, and mild pitting edema of lower extremities. No active vaginal bleeding or foul-smelling discharge was noted. An arterial blood gas analysis revealed the following: pH: 7.54; partial press of carbon dioxide ($PaCO_2$): 32.0 mmHg; partial press of oxygen (PaO_2): 66 mmHg; bicarbonate ion (HCO_3^-): 27.4 mmol/L; Lactate 1.4 mmol/L. WBC 29.04×10^9/L, RBC 2.82×10^9/L, Hb 79 g/L, Hct 23.40%, PLT 45×10^9/L, Ret% 3.10%. Peripheral blood smear demonstrated: mild anisocytosis was noted in some mature red blood cells (RBC). Schistocytes were present at 0.8%, and a few polychromatophilic RBC were observed. Coombs (-). Scr 285.20 μmol/L, urea 13.17 mmol/L, ALT 35.80 U/L, AST 250.50 U/L, TBIL 93.02 μmol/L, and DBIL 61.61 μmol/L. PT 12.60s, PTA 75.80%. INR 1.16, FDP 9.29 μg/mL. BNP 10,011.00 pg/mL. PCT exceeding 100 ng/ml. Blood cultures analysis (Pre-admission): gram-negative bacteria were isolated. Renal ultrasound revealed thickened and enhanced parenchymal echogenicity in both kidneys. ADAMTS13 enzyme activity and inhibitory antibody tests (-). The tests for human complement factor I (CFI), human complement factor H (CFH), human complement factor B (CFB), and human complement factor H antibodies (-). Upon admission, empirical anti-

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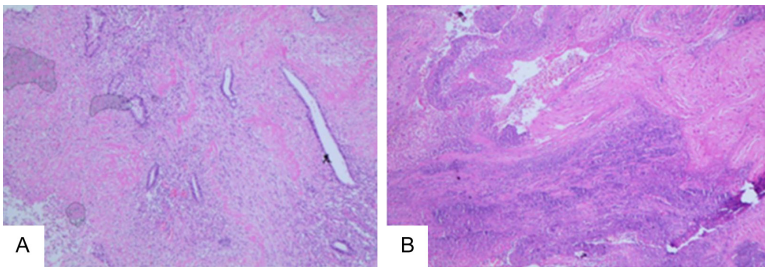

Figure 1. A, B. ×100. Histopathological examination of the intrauterine tissue revealed extensive necrotic debris, scattered fragments of decidual tissue, and small foci of endometrial tissue exhibiting secretory-phase changes, accompanied by marked neutrophilic infiltration.

microbial therapy (meropenem 1g q8h adjusted for renal function), continuous renal replacement therapy (CRRT) with plasma exchange, mechanical ventilation, and uterine contraction augmentation were initiated. Blood and cervical cultures were obtained, with initial blood cultures revealing gram-negative bacilli. Transvaginal ultrasound demonstrated heterogeneous mass at the right superior uterine cavity, suggestive of retained products of conception. Procalcitonin (PCT) level remained elevated, exceeding 100 ng/ml. Subsequent blood culture identified Escherichia coli, Prompting continuation of meropenem per susceptibility testing. After 10 days of sustained treatment in the ICU, the patient's infection markers improved (PCT decreased to 17.83 ng/ml), enabling transfer to nephrology for sustained hemodialysis. However, persistent febrile gynecological ultrasound showed residual intrauterine material. Consequently, she underwent surgical purging, with histopathology revealing extensive necrotic debris and neutrophilic infiltrates (**Figure 1A**, **1B**). Cervical culture grew carbapenem-resistant Acinetobacter baumannii (CRAB), leading to antibiotic escalation to cefoperzone/sulbactam (3g q8h) plus tigecycline (50 mg q12h). Renal biopsy confirmed thrombotic microangiopathy (TMA) with characteristic microthrombi (**Figure 2A-C**). At 3-week post-discharge follow-up, molecular testing revealed a pathogenic CFI/MIN variation. Two-mouth follow-up showed partial renal recovery (urine output 1,800 ml/day), but persistent uremia, requiring ongoing dialysis.

Discussion

Hemolytic uremic syndrome (HUS) is a clinical syndrome characterized by microangiopathic hemolytic anemia, thrombocytopenia, and acute kidney injury [1]. It is subclassified into categories: Typical HUS (STEC-HUS), induced by Shiga toxin-producing Escherichia coli; Atypical HUS (a-HUS): primarily caused by congenital or acquired dysregulation of the alternative complement pathway [2]. PHUS is a pregnancy-associated subtype of aHUS, that typically develops within 10 weeks after delivery. It presents with acute renal injury, thrombocytopenia, and microangiopathic anemia, often progressing rapidly with significant risk. The primary pathological manifestation of PHUS is extensive glomerular microthrombosis, which may progress to fibrinoid necrosis in severe cases [3, 4]. In this case, renal biopsy revealed glomerular ischemic sclerosis, mild segmental microthrombi within glomerular capillary loops, thickened small arterioles with proliferative and swollen endothelial cells, fibrinoid necrosis of the vessel walls, and luminal stenosis. These findings are consistent with the pathological changes of thrombotic microangiopathy (TMA).

Early diagnosis and timely intervention can effectively delay the progression of renal function deterioration in PHUS patients, significantly improving long-term prognosis [5]. However, clinical identification of this disease faces dual challenges: First, due to its rare incidence (<1 per million annually) and nonspecific manifestations such as fever and thrombocytopenia, aHUS is prone to be misdiagnosed as obstetric emergencies like HELLP syndrome, particularly during the perinatal period where diagnostic ambiguity persists [6]. Second, current complement gene mutation detection systems achieve laboratory confirmation in only 40% of cases, with molecular testing for CFH/CFI/MCP requiring 5-7 working days on average. This diagnostic delay compels clinicians to rely on the TMA triad - microangiopathic hemolytic anemia, thrombocytopenia, and acute kidney injury - for immediate clinical decision-marking [7]. Simultaneously, prompt initiation of diagnostic treatment and further complement factor antibody

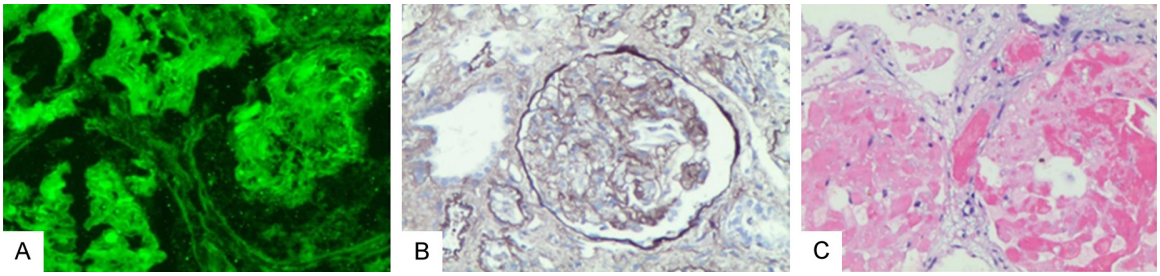

Figure 2. Renal histopathology. A. ×200. Glomerular Immunofluorescence: IgG (-), IgA (-), IgM (+-), C3 (+-), C1q (+-), Fib (+); Mesangial granular deposits: IgG1 (-), IgG2 (-), IgG3 (-), IgG4 (-), HbsAg (-), HbcAg (-), Kappa (-), lambda (-), c4d (-). B. ×400. No amyloid deposition was observed on Congo red staining. C. ×200. Renal arterioles and small arteries exhibited fibrinoid necrosis, with fibrin deposition observed within the vascular lumen and vessel walls. The glomeruli demonstrated marked swelling, containing extensive fibrin thrombi, accompanied by erythrocyte extravasation.

testing, along with specific genetic and molecular analyses, are critical for definitive diagnosis.

At present, no specific therapeutic agents are approved for PHUS, and clinical management primarily relies on multimodal supportive therapy. Current evidence supports combined hemodialysis and plasma exchange as the first-line therapeutic strategy for PHUS. Initiating dialysis within 24 hours of diagnosis or suspicion improves survival during acute renal injury. In addition, early adjunctive plasma exchange removes pathogenic complement factor, replenishes coagulation regulators, and reduces kidney damage [8].

Conclusion

PHUS is a rare but life-threatening obstetric complication characterized by low incidence but disproportionately high mortality and poor prognosis. Recent studies indicate that over 60% of PHUS patients progress to end-stage renal disease. Early diagnosis via multidisciplinary team collaboration and prompt initiation of evidence-based management strategies are critical to mitigate adverse outcomes.

Disclosure of conflict of interest

None.

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