



Congress Abstract

Paroxysmal Nocturnal Hemoglobinuria Presenting with Acute Kidney Injury During Pregnancy: A Diagnostic Challenge for Nephrologists

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ABSTRACT

Background: Paroxysmal nocturnal hemoglobinuria (PNH) is a rare acquired clonal hematopoietic disorder characterized by complement-mediated intravascular hemolysis and thrombosis. Renal involvement is clinically relevant: impaired renal function (eGFR <90 mL/min/1.73 m²) was reported in 42.8% of 4,439 patients in the International PNH Registry. Pregnancy is a high-risk setting. A 2025 meta-analysis of 190 pregnancies in 135 women with PNH reported fetal survival of 82% with eculizumab versus 69% without it, while preterm birth occurred in 32% and 44%, respectively. Coexisting hemolysis, thrombocytopenia, and renal dysfunction during pregnancy may mimic thrombotic microangiopathy (TMA).

Case Presentation: A 33-year-old woman in her third pregnancy had longstanding thrombocytopenia previously considered immune/idiopathic. In March 2025, she developed acute kidney injury (AKI), with serum creatinine 143 µmol/L and eGFR 42.8 mL/min/1.73 m². Nephrology admission revealed dark urine, anemia, thrombocytopenia, proteinuria up to 5 g/L, hematuria, and 24-hour proteinuria of 1.98 g/day. Marked intravascular hemolysis was demonstrated by LDH >2136 U/L, indirect bilirubin 20.9 µmol/L, and haptoglobin 0.1 g/L. Renal function subsequently recovered. TMA was initially suspected, including thrombotic thrombocytopenic purpura (TTP) and atypical hemolytic uremic syndrome. ADAMTS13 activity was 93%, arguing against severe ADAMTS13 deficiency. Persistent hemolysis prompted flow cytometry, which identified a PNH clone: type II erythrocytes 0.14%, type III erythrocytes 38.24%, FLAER-/CD24- granulocytes 7.8%, and FLAER-/CD14-monocytes 34.4%. Urinary hemosiderin was positive, reticulocytes were 8.9%, and LDH remained >1684 U/L. PNH with chronic intravascular hemolysis was diagnosed. At approximately 19 weeks, a multidisciplinary team considered eculizumab; because renal function was preserved and there was no transfusion dependence, anticoagulant prophylaxis and close monitoring were chosen. At approximately 27 weeks, pregnancy was complicated by severe preeclampsia and subsequently resulted in preterm delivery with antenatal fetal death.

Conclusion: PNH should be considered in pregnant patients with unexplained AKI and/or proteinuria accompanied by thrombocytopenia and intravascular hemolysis. Preserved ADAMTS13 activity and identification of a PNH clone were pivotal in distinguishing PNH from TTP. Early recognition and multidisciplinary nephrology-hematology-obstetric management are essential because renal, thrombotic, and pregnancy-related complications may be severe.

Keywords: Paroxysmal Nocturnal Hemoglobinuria; Pregnancy; Acute Kidney Injury; Proteinuria; Intravascular Hemolysis; Thrombotic Microangiopathy; ADAMTS13; Preeclampsia