

The Diagnostic Crossroads of Severe PET and C -TMA: A Case of Complement -Mediated Mimicry

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Background : Complement -mediated thrombotic microangiopathy (C-TMA), previously known as atypical haemolytic uraemic syndrome (a-HUS), in pregnancy is a rare, life-threatening condition which closely overlaps with severe pre-eclampsia (PET), creating diagnostic uncertainty for clinicians. Up to 60% of patients have an underlying genetic predisposition.

Aims : To explore C-TMA as a differential for critically unwell patients with acute renal failure, thrombocytopenia and haemolysis, and differentiate cases from severe PET.

Case: A 31 year old primigravida, at 36 weeks gestation with a history of previous myomectomy, and antenatal foetal growth restriction, presented with severe abdominal pain and vaginal bleeding. CTG identified foetal bradycardia and a Category 1 caesarean section was performed. Intraoperative findings revealed a massive placental abruption without evidence of uterine rupture and the patient was anuric.

Post-operatively, the patient required haemodialysis and non-invasive ventilation with oligo-anuric renal failure complicated by hyperkalaemia and fluid overload. Her serum creatinine peaked at 613, Hb reached a nadir of 64, and platelets 39. Low C3 with preserved C4, haptoglobin <0.10, and a normal ADAMTS13 supported a diagnosis of C-TMA, and Eculizumab was commenced. Nil identifiable genetic cause was found. She has had complete normalisation in her renal function post partum.

Discussion :

C-TMA is characterised by the uncontrolled activation of the alternative complement pathway, causing acute kidney injury, thrombocytopenia, and haemolysis. Historically, over 50% of cases progressed to end-stage renal disease. Eculizumab, a monoclonal antibody targeting C5, inhibits terminal complement activation, thereby preventing complement -mediated thrombosis and improving renal outcomes. Distinguishing C-TMA from severe pre-eclampsia is critical, as complement -mediated injury persists despite delivery, and early targeted therapy is needed to prevent irreversible renal damage.