

Post-Partum Microangiopathic haemolytic anaemia, thrombocytopenia and AKI: post partum haemorrhage vs complement mediated aHUS

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Introduction

Thrombotic microangiopathy (TMA) can present during pregnancy and commonly in the post-partum phase. It can be a challenging diagnostic conundrum to dissect between; pre-eclampsia & HELLP syndrome, pregnancy associated atypical haemolytic uraemic syndrome (aHUS) and thrombotic thrombocytopenia purpura (TTP). At the National Renal Complement Therapeutics Centre (NRCTC) we have seen a cohort of women presenting with Acute Kidney Injury (AKI) and microangiopathic haemolytic anaemia (MAHA) after a post-partum haemorrhage.

Methods

We conducted a retrospective case review of all women referred to the NRCTC with thrombocytopenia, AKI and MAHA in the post-partum period. A detailed review differentiated the cases into those with a large post-partum haemorrhage (PPH; blood loss >1 litre) and those without. Complement biomarkers and complement genotyping were undertaken in all cases.

Results

Between April 2013 and June 2019 ~750 individuals have been referred to the NRCTC with suspected aHUS. 178 were females of child bearing age (16-45 years old). Of these 10 had a history of a PPH. 3/10 were treated with eculizumab due to initial diagnostic uncertainty. The remaining 7 patients received supportive treatment. 6 out of the 7 patients who received supportive treatment recovered renal function. Three patients had renal biopsies which showed a range of pathology including TMA, Acute Tubular Necrosis and Cortical necrosis. In the PPH group no complement mutations were identified. This group was compared to the NRCTC cohort of post-partum complement mediated aHUS with complement mutations.

Discussion

Both PPH and complement mediated aHUS can present with AKI, MAHA and thrombocytopenia with differentiation initially challenging. In PPH, this phenotype is not associated with complement mutations unlike postpartum aHUS where >70% will have mutations. In PPH supportive treatment usually results in spontaneous resolution of the AKI, thrombocytopenia and MAHA unless cortical necrosis is present.