

**Utility Of Serology In The Diagnosis Of Preeclampsia and HUS In Pregnancy-  
Related Acute Kidney Injury**

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Antepartum haemorrhage is a leading cause of maternal mortality and acute kidney injury (AKI) in developing countries.<sup>1</sup> In general, AKI is ascribed to volume loss, patients receive supportive treatment but are not investigated for other complications of pregnancy that contribute to AKI, and affect diagnosis and treatment. In the present report, we present an unusual case of pregnancy-related AKI (PR-AKI) with a valuable learning lesson.

A 28-year-old primigravida presented to a primary care facility with vaginal bleeding and pain abdomen at 28 weeks of gestation. She was not told to have any abnormality at her only antenatal visit at 16 weeks of gestation. At presentation, she was hypotensive (90/70 mm Hg), received volume resuscitation and was referred to our hospital, by when she had become anuric. Table 1 shows the investigations, that revealed evidence of microangiopathic hemolysis, normal prothrombin and activated thromboplastin times and azotemia. A non-viable fetus was detected on ultrasound. Vaginal delivery was induced, and she received hemodialysis and blood transfusions. As she stabilized, the blood pressure increased and she needed antihypertensive therapy. Subsequent investigations showed elevated levels of soluble fms like tyrosine kinase (sFLT-1), low placental growth factor (PlGF), and a high sFLT-1/PlGF ratio, suggestive of preeclampsia.<sup>2-4</sup> There was evidence of alternate pathway complement activation, which triggered further investigations– complement factor-H level was low and autoantibodies to complement factor-H (aCFH) were present. A diagnosis of acute kidney injury (AKI) secondary to aCFH related hemolytic uremic syndrome (HUS) and preeclampsia was made. Plasmapheresis could not be done due to financial reasons; the patient improved with hemodialysis and supportive care. Urine output improved after 14 days and creatinine started to decline. The antihypertensive drugs were gradually withdrawn and stopped after 1 month. Six months after discharge, she is normotensive with serum creatinine of 1.7 mg/dL.

This patient presented with PR-AKI following ante-partum hemorrhage. However, serological testing (sFLT-1/PlGF >88) was strongly in favour of pre-eclampsia.<sup>2-5</sup> We think that volume loss and kidney injury masked the diagnosis of preeclampsia by

attenuating hypertension and proteinuria. This patient also showed evidence of aCFH and alternate pathway complement activation. Preeclampsia is known to activate complement pathway in a predisposed patient, like the presence of aCFH, which was the case in this patient.<sup>6</sup> To conclude, the pathogenesis of pregnancy-related AKI is complicated, and a high index of suspicion is needed to uncover these abnormalities.

Table 1: Details of laboratory investigations

Parameter	Value
Hemoglobin	79 g/L
Platelet count	50,000/mml
Prothrombin time	14 sec
Activated partial thromboplastin time	32 sec
Schistocytes	2%
Haptoglobin	0.68 $\square$ mol/L
Uric Acid	559 mmol/L
Lactate dehydrogenase	77.7 $\square\square$ kat/L
Aspartate aminotransferase	4.94 $\square$ kat/L
Alanine aminotransferase	2.56 $\square$ kat/L
Total Bilirubin	17.1 $\square$ mol/L
Urine Albumin	3+
Soluble fms-like tyrosine kinase (sFLT-1)	5829 pg/mL
Placental growth factor (PlGF)	23.6 pg/mL
sFLT-1/PlGF ratio	246.9 (< 88)
C3 complement level	0.12 g/L
Complement factor H (CFH)	462.2 mg/ml (>600)
Alternative pathway functional assay	-0.08 % (27-55)
Anti-CFH levels	35.12 AU/mL (<18)

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