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# A Rare Case: Acute Kidney Injury in Pregnancy with Pre-Existing Aortic Stenosis

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## Abbreviations

AKI- Acute Kidney Injury

HELLP- Hemolysis Elevated liver enzymes and Low platelets

AFLP- Acute fatty liver of pregnancy

P-TMA- Pregnancy associated thrombotic microangiopathies.

## Introduction

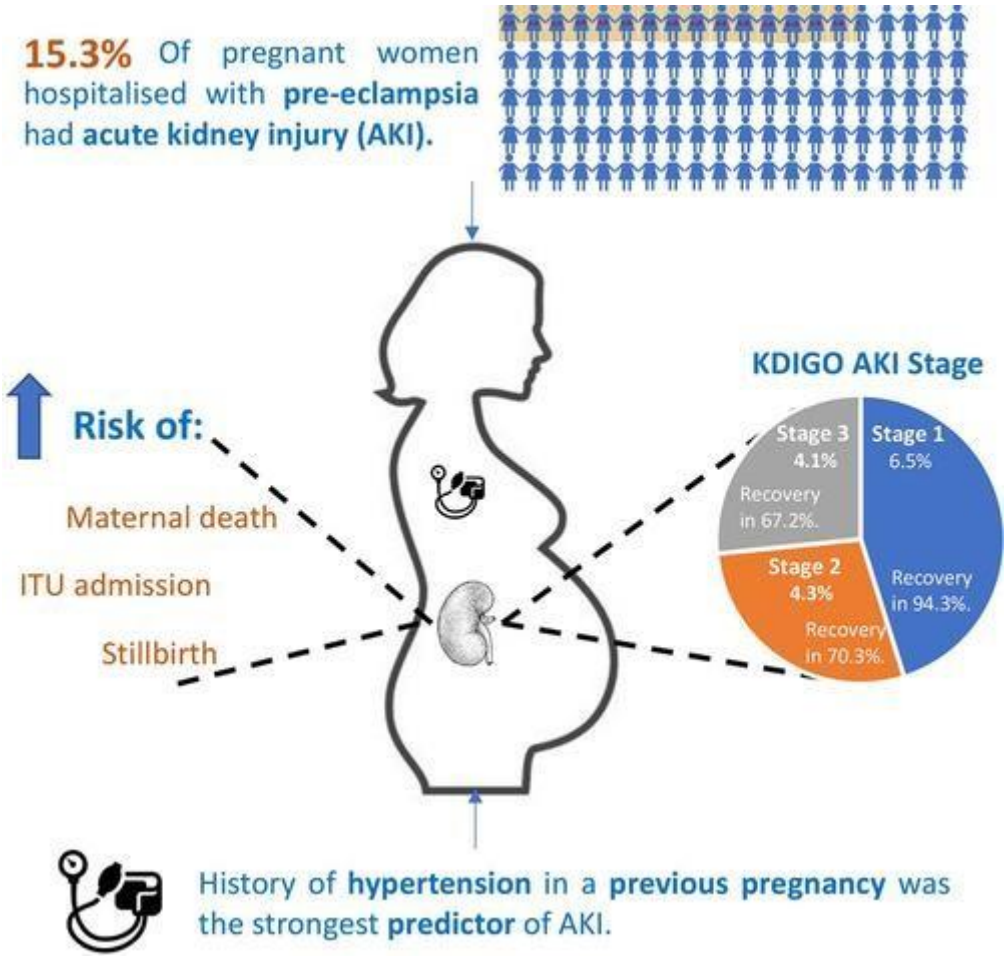
Acute kidney injury (AKI) that occurs during pregnancy or in the post-partum period is a serious obstetric complication with an increased risk of maternal and fetal morbidity and mortality. First trimester causes of AKI in pregnancy include hyperemesis gravidarum and septic abortion. In the third trimester causes such as preeclampsia, acute fatty liver of pregnancy, HELLP syndrome, and the thrombotic microangiopathies (thrombotic thrombocytopenic purpura, atypical hemolytic uremic syndrome) exhibit overlapping features and often present as a diagnostic dilemma.[1,2] Clinical judgment and experience along with laboratory investigations become paramount in making an accurate diagnosis.[3,4]

## Case Presentation

A 26-year-old primigravida with a known case of aortic stenosis with bicuspid aortic valve, who presented at 39+4 weeks of gestation with headache and generalised oedema. Examination findings and laboratory investigations(on admission) were otherwise normal except for raised blood pressure, 2+ proteins in urine and raised PCR(PCR=150). She was started on anti-hypertensive medication on admission. Her past surgical history was significant as she underwent 2 aortic balloon valvoplasty and then Ross procedure for aortic stenosis. She was offered induction of labour for premonitory symptoms and raised blood pressure after detailed counselling.

Intrapartum, she developed oliguria, and she was catheterised to monitor urine output. Her observations (vitals) were otherwise fine and no sign of dehydration and CTG was suggestive of fetal tachycardia. Initially she was encouraged to have oral fluids, later was given IV fluids as fluid challenge, inspite of this she had less than 25cc urine in 4 hours. The fetal tachycardia settled later on.

After senior review all blood investigations repeated again, which revealed rising creatinine and decreasing eGFR. After 30 minutes from last review, she developed hematuria. Her total urine output was less than 50cc in 7hours. Diagnosis of Acute Kidney Injury was made and she was urgently assessed by the medical team and the anaesthetist. After multidisciplinary team review, decision was taken for Category 2 caesarean section. The caesarean section was uneventful, and baby cried immediately after birth, and blood loss at caesarean was 500ml. Post-delivery her urine output and laboratory investigations improved. She was kept on close monitoring and baby and mother were discharged 5 days later with a plan for follow-up.




## Discussion

We observed that preeclampsia/eclampsia is the most common cause of AKI in late third trimester and postpartum periods followed by puerperal sepsis and postpartum hemorrhage [5]. Preeclampsia is a multi- organ disorder characterized by systemic hypertension and renal, hepatic, and cerebral vascular pathology.

These clinical manifestations are consistent with widespread endothelial dysfunction, vasoconstriction, and end-organ ischemia. Abnormal placental angiogenesis during pregnancy resulting from high levels of the anti-angiogenic factors, soluble fms-like tyrosine kinase and soluble endoglin, has been implicated in preeclampsia.[7] Redman et al. have suggested that preeclampsia is an extreme end of a continuous spectrum of inflammatory responses that are a feature of pregnancy itself [8]. This hypothesis is also supported by studies that demonstrate that when a single dose of endotoxin or interleukins is given to pregnant animals, they manifest clinical features of preeclampsia, but not the controls[9.10]. The treatment of AKI in pregnancy is generally supportive, often coupled with expedient delivery, especially when diagnosis is severe preeclampsia, AFLP or HELLP[6]. Given the recent trends of increasing maternal age at the time of pregnancy, as well the availability of modern reproductive methods both of which may be associated with significant co-morbidities, the issues surrounding AKI in pregnancy may become more relevant in the coming years.

| **Blood investigations** | **10/3/22 (on admission)** | **12/03/22 (intra- partum)** | **12/03/22 (postpartum >6) hrs.** |
|---|---|---|---|
| **Haemoglobin** | 122g/L | 112 | 121 |
| **Wbc** | 6 x $10^9$ | 6x$10^9$ | 6x$10^9$ |
| **Platelets** | 2 lacs | 1.8 | 2.1 |
| **Sodium** | 136 | 129 | 135 |
| **Potassium** | 4.2 | 4.5 | 4.2 |
| **Creatinine** | 47 | 144 | 116 |
| **eGFR** | >90 | 49 | 80 |
| **Urea** | 5mmol/L | 6.2 | 5.9 |

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