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Acute Kidney Injury and Thrombotic Microangiopathy after Caesarean Section: A Case Report

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Abstract **Case Report**

Pregnancy related acute kidney injury is an infrequent complication associated with postpartum haemorrhage, sepsis, preeclamsia and less commonly with thrombotic microangiopathy. Hereby, we are presenting a case of a young female who underwent cesarean section and developed acute kidney injury in postpartum period decreased urine output. She was diagnosed with thrombotic microangiopathy. She was managed with collaborative intervention and discharged with complete recovery.

Keywords: Acute Kidney Injury, Thrombotic Microangiopathy, Caesarean Section, Eclampsia, Thrombocytopenia.

INTRODUCTION

Acute Kidney Injury (AKI) is a severe medical condition defined by rapid decline in glomerular filtration rate, resulting in accumulation of urea and other nitrogenous waste products and disturbances in fluid and electrolyte balance [1].

The proposed diagnostic criterion for AKI is an abrupt (within 48 hours) reduction in kidney function which is defined as an absolute increase in serum creatinine (level of >0.3 mg/dL) or a percentage increase in serum creatinine level of more than 50% (1.5-fold from baseline) or reduction in urine output (documented oliguria of less than 0.5 mL/kg per hour for more than 6 hours [2]. Acute kidney injury can be classified by its etiology into prerenal, renal, and postrenal failure. Prerenal AKI occurs in clinical settings leading to volume depletion, decrease in effective blood volume, renal vasoconstriction, altered renal hemodynamics, and increased renal vein pressure. Prerenal AKI is not only common but is also potentially reversible. Renal is caused by damage to kidney itself due to toxins, inflammation and injury. Post frenal occurs due to some blockage to urine flow.

The incidence of pregnancy-related acute renal failure in developed countries is 1–2.8%, whereas in the developing countries, this is about 4.2–15% [3-5]. Based on the trimester of pregnancy, acute kidney injury has been divided into 3 groups: first half, second half, and postpartum ARF. Pregnancy-related AKI is usually caused by septic abortion in early pregnancy, by pregnancy toxaemia, HELLP Syndrome, hemorrhages during pregnancy (antepartum and postpartum), and acute tubular necrosis in late pregnancy [6,7]. Puerperal sepsis and thrombotic microangiopathy are seen in the postpartum period. The use of NSAIDs during the postpartum period is a known risk factor for the development of AKI.

The long-term outcome of pregnancy related acute kidney disease is usually favorable unless renal cortical necrosis (RCN) occurs. RCN accounts for only 2% of all cases of ARF and can lead to chronic renal failure (CRF) due to the ischemic destruction of the renal cortex [8]. Timely diagnosis and management of AKI are crucial to minimizing the risk of maternal and fetal complications. The management during pregnancy and after delivery requires a multidisciplinary approach involving obstetricians, nephrologists, and critical care specialists. Hereby, we are presenting a case of acute kidney injury following a cesarean section who was managed and discharged.

CASE REPORT

Twenty six years old G2P1L1 (booked and supervised elsewhere) 36+6 weeks on 9/04/2025 previous LSCS with Rh negative pregnancy with HCV reactive status admitted with complain of pain abdomen with ocassional tightening of abdomen and decreased fetal movements since 1 day. She was planned for elective LSCS with bilateral tubal ligation under all universal precautions. She was married since 7 years. Previous LSCS was done 5 years back in view of oligohydramnios with non – reassuring FHR. She was known case of HCV Reactive status since last 8 months. Her blood group was O Negative. ICT was negative. There was presence of calcium oxalate crystals in urine before surgery. USG was done on 18/03/2025 which showed SLIUP of 35 weeks 4 days, cephalic presentation, placenta anterior and EFW to be 2558 grams, liquor adequate. Injection Betamethasone was covered 12 mg 2 doses 24 hours apart. On opening the abdomen, there were adhesions of uterus with anterior abdominal wall. Bladder was advanced and adherent. Lower uterine segment and previous scar site thinned out. Otherwise, it was an uneventful surgery. She delivered live baby girl weighing 3.44 kg with Apgar 8 and 9. Baby blood group came out to be O Positive. Injection Anti D 300ug was given IM.

Post op day 0 was uneventful with all the vitals being stable. She was put on routine IV antibiotics ceftriaxone, metrogyl and gentamycin. Urine output was adequate. On post operative day 1, in late afternoon urine output decreased and patient developed jaundice. Her pulse rate was 120 bpm, BP 104/60 mm of Hg, RR was 24 / min. There was yellow discoloration all over the body. There was no pedal odema at that time. All the investigations were sent and patient was shifted on piperacillin and tazobactem combination along with clindamycin. She was started on tablet sodium bicarbonate 500mg bd, Tab Rifaximin 400mg tds, Tab ursodeoxycholic acid 300 mg TDS. Haemoglobin was 11.8 g/dl.TLC was raised to 23.6 cumm. Her platelets came out to be 50, 000/cumm. Serum creatinine was 2.9mg/dl. Total bilirubin was 9.8mg/dl and SGOT/PT 280/69. PTI was 71.3 and INR 1.45. LDH was very much raised 2840 U/L. Urine albumin was present. USG whole abdomen and pelvis was grossly normal. 2-D ECHO was done which was normal. For deranged coagulation profile, Vitamin K was started. Injection diclofenac was omitted. Medicine and nephrology opinion was taken and patient shifted to meropenam and colistin and shifted to ICU. Blood culture came out to be sterile. D dimer was 73.9ng/ml. On post operative day 2, patient was transfused 2 units of fresh frozen plasma and one unit of single donor platelets. TLC was 26,000cumm. Platelet count was 26,000 cumm. X ray chest showed minimal free fluid in left pleural cavity. USG on day 2 was normal except minimal non- tapable fluid in left pleural cavity (depth 2mm). On day 2 late evening she developed bilateral pitting pedal odema which was more on left side. Patient passed stools on 2nd day. On postoperative Day 3 she developed lesions at the angles of mouth which was diagnosed herpes labialis and ointment Acyclovir and oral acyclovir was given. On postoperative day 5 she developed oral candidiasis. USG KUB showed renal cortical echoreflectivity with altered corticomedullary differentiation likely renal parenchyma disease with bilateral pleural effusion and ascites. Day 6 pedal odema resolved. She was shifted to oral antibiotic tab Faropenem bd on 13th day and discharged in stable condition.

DISCUSSION

Pregnancy related acute kidney injury (PRAKI) is defined as AKI diagnosed anytime during pregnancy or during postpartum phase (first 6 weeks post delivery).

In India, PR-AKI has decreased from 14.5%min 1987 to 4.3% in 2005 [9]. Specific complications related to each trimester may contribute to kidney injury [10]. In the early stages of pregnancy, AKI is most commonly associated with hyperemesis gravidarum, or Acute Tubular Necrosis (ATN) resulting from a septic abortion. Later in pregnancy or postpartum, AKI can result from severe preeclampsia, HELLP syndrome, thrombotic microangiopathy, acute fatty liver of pregnancy, ATN or acute cortical necrosis associated with haemorrhage [11].

Less commonly, it can follow Nonsteroidal Anti-Inflammatory Drugs (NSAIDs), acute pyelonephritis and urinary tract obstruction. NSAIDs are routinely used for postpartum analgesia, particularly after a caesarean section. Although uncommon, AKI may develop in patients who receive NSAIDs if there are predisposing conditions such as volume depletion or preeclampsia [11]. Sepsis and coagulation abnormalities were the main factors responsible for mortality in the study by Naqvi *et al*., [12].

In our case presence of calcium oxalate crystals in urine before surgery which are indicative of fluid deficiency in body. Since there is thrombocytopenia along with raised LDH and organ damage, thrombotic microangiopathy could be a cause. This case appears to be an example of Acute Renal Failure with Renal Cortical Necrosis probably caused by Thrombotic microangiopathy. The differential diagnosis to be excluded are postpartum hemorrhage, amniotic fluid embolism, HELLP syndrome and sepsis. Thrombotic microangiopathy (TMA) is a heterogeneous group of disorders features by thrombocytopenia, hemolytic anemia and organ failure. Severe post-partum hemorrhage can mimic thrombotic microangiopathy since PPH can lead to renal cortical necrosis due to disseminated intravascular coagulation (DIC) [13]. Our patient did not have PPH and there was no fall in hemoglobin postoperative. Sepsis was ruled out as patient had no fever and blood culture was sterile.

Diagnosis of AKI during pregnancy involves a thorough medical history, physical examination, and review of medication use, as well as laboratory tests, including urine analysis, protein quantification, urine culture, and blood tests to evaluate for microangiopathic haemolysis and thrombocytopenia [14]. Imaging tests, including renal ultrasound or CT, may also be necessary to diagnose cortical necrosis or obstructive uropathy [15]. Occasionally, a renal biopsy may be necessary to confirm the diagnosis and to help with prognosis if evidence of impairment persists [16].

The management of AKI in the postpartum period includes correcting the underlying cause, such as discontinuing possible nephrotoxic agents such as NSAIDs, Angiotensin-Converting Enzyme (ACE) inhibitors, Angiotensin Receptor Blockers (ARBs), treating infections or haemorrhage and hypotension. Fluids and electrolyte imbalances must be corrected, and dietary restrictions on potassium, phosphorus, sodium, and fluid intake may be necessary. In cases of emergency, dialysis or kidney replacement therapy may be necessary to manage severe symptoms, such as pulmonary oedema, hyperkalaemia, or uremic symptoms [17]. Patients with moderate to severe AKI should have an outpatient nephrology review to monitor renal function and blood pressure to prevent the development.

CONCLUSION

Acute kidney injury is a life threatening condition. Multidisciplinary approach and immediate intervention is important to prevent further renal damage and save the life of patient.

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