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Pregnancy Related Acute Kidney Injury-A case report

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Abstract

Pregnancy-related Acute Kidney Injury (AKI) is a global health problem and is associated with high risk of both maternal and fetal morbidity and mortality. Even though the incidence shows a decreasing trend in the developing countries like India, there are still significant number of cases owing to socioeconomic factors and inadequate antenatal care. We report a case of a 29years old primigravida at 26 weeks + 2 days, case of uncontrolled Chronic Hypertension and Hypothyroidism referred with oligohydramnios (AFI-6.4cm) and FGR. On evaluation she was found to have altered renal parameters and moderate anemia and underwent Emergency Caesarean in view of impending signs of eclampsia and worsening renal parameters. Postoperatively renal parameters further worsened and Nephrology evaluation with Renal Biopsy- reported as Glomerulonephritis- FSGS, Chronic interstitial Nephritis, acute tubular necrosis and hypertensive vascular changes. Currently she is on antihypertensives and monitoring of renal parameters. Diagnosis of pregnancy-related AKI is challenging due to the lack of standard criteria and overlap of clinical manifestations among different etiology. Chronic hypertension and altered renal parameters in pregnancy mandates early suspicion and treatment to prevent sequential complications to the mother and baby.

We here aim to clarify the grey areas in early diagnosis and management of a case Pregnancy related Acute Kidney Injury which may help clinicians in their practice.

Keywords: Pregnancy related Acute Kidney Injury, Chronic hypertension.

Introduction

Pregnancy-related acute kidney injury (AKI) has declined in incidence in the developing countries in last three decades, although it remains an important cause of maternal and fetal morbidity and mortality [1]. Whereas the incidence of pregnancy-related acute kidney injury has increased in the developed countries, theorized to be the result of an increase in pregnancies in advanced maternal age [2]. Due to the physiological changes of pregnancy, PRAKI is difficult to diagnose, hard to predict and often diagnosed after significant damage to the kidney has occurred. Pregnancy-related causes of AKI such as preeclampsia, acute fatty liver of pregnancy, HELLP syndrome, thrombotic micro Angio pathies (thrombotic thrombo cytopenic purpura, atypical hemolytic - uremic syndrome) exhibit over lapping features and are often diagnostic dilemmas.

Differentiating among these conditions may be difficult or impossible based on clinical criteria only. In difficult and rare cases, a renal biopsy may need to be considered for the exact diagnosis and to facilitate appropriate treatment[1]

Acute kidney injury (AKI) is marked by sudden decline in glomerular filtration rate, causing decreased excretion of nitrogenous waste products like urea, creatinine, and uremic products. In normal pregnancies, the serum concentration of creatinine falls as a result of the increase in glomerular filtration rate. Elevation of the Serum creatinine levels >0.9 mg/dl or 75 µmol in pregnancy considered outside the normal range for pregnancy and calls for critical evaluation of renal function [5].

In India, PRAKI requiring dialysis fell from 15% in 1982–1991 to 10% in 1992–2002, with a concurrent decrease in maternal mortality from 20% to 6.4%, respectively [3]. This marked decline might be due to the reduction in sepsis associated with abortion and childbirth, as well as improved management of post partum haemorrhage and placental abruption.

This report is of a 29-year-old Primigravida diagnosed with Pregnancy Related Acute Kidney Injury and describes the challenges in diagnosis and management. A multi- disciplinary team approach aided in avoiding a major mishap.

Case Report

A 29-year-old Primigravida at 26 weeks+ 2days with history of hypothyroidism and uncontrolled Chronic Hypertension was referred from a local hospital with oligohydramnios (AFI-6.4cm) and FGR. Her Blood Pressure recording at the time of antenatal visit was found to be 160/100mmHg and further evaluation showed raised levels of S. Creatinine-3.2mg/dl, Urea-72mg/dl, decreased Hemoglobin levels of - 8.7gm% and a Urine albumin level of 3+. Her Previous blood investigations1st trimester showed normal values (Urea-13gm%, Creatinine- 0.7mg/dl. After admission, her RFT was serially monitored, which showed worsening of the renal parameters (Cr-3.8mg/dl, Ur-79mg/dl). Ultrasound Abdomen - Bilateral raised renal echogenicity with maintained cortico-medullary differentiation. The necessity of early termination of pregnancy in view of worsening renal parameters and uncontrolled hyper tension was explained to the mother and the relatives.

Her BP showed a persistent rise to 190/110mmHg associated with acute onset of severe headache with multiple episodes of vomiting. She was taken up for Emergency Caesarean at 26wks +3days in view of impending eclampsia and worsening renal parameters. Extracted a preterm male baby of weight 585gms. Baby

was kept in NICU with supportive care but expired on NND 4.

Post operatively, she was restarted on antihypertensives. Cardiology evaluation revealed normal ECG and ECHO. Ophthalmology evaluation showed no evidence of any disc edema or hypertensive retinopathy. However, the renal parameters were further worsening- 24hr urine protein- 3023gms, Urine P:C-3.61, Creat-4.3 mg/dl, Urea-90mg/dl, cANCA, pANCA, ANA- neg. Systemic steroids were started in view of suspected underlying Glomerulonephritis and IV Iron and weekly Erythropoietin as supportive management for anemia. In view of persistently deranged renal parameters, Renal Biopsy was done, which showed Glomerulonephritis-FSGS, Chronic interstitial Nephritis, acute tubular necrosis and hypertensive vascular changes

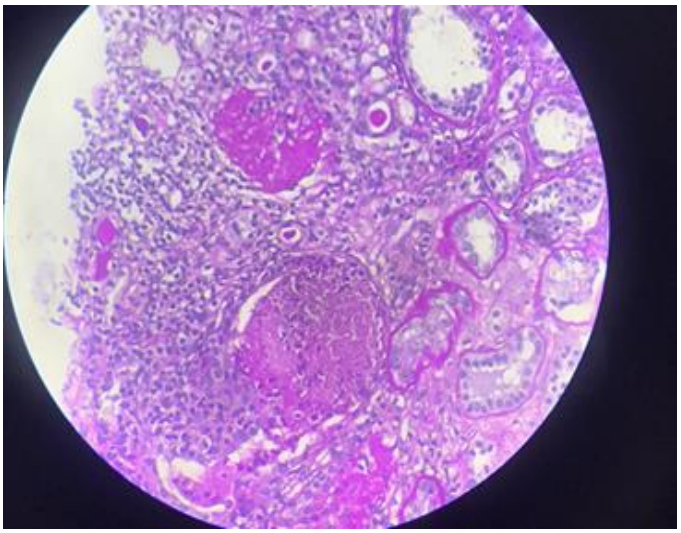

Fig [1]- PAS staining showing global sclerosis of one glomeruli and necrosis in the other glomeruli. Background shows dense inflammatory infiltrate

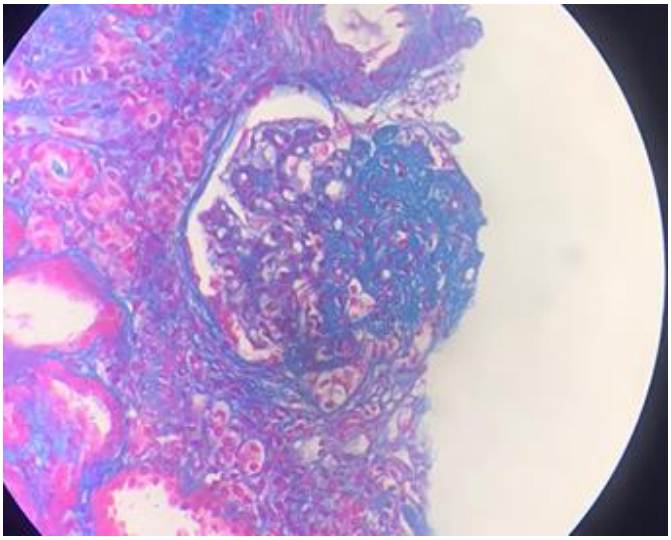

Fig 2: Masson trichrome staining showing segmental sclerosis of glomeruli

At present, S. Creat- 4.8mg/dl, Urea- 53mg/dl, P:C- 3.6, Hb- 8.7mg/dl. She is on antihypertensives -T. Nifedipine R 10mg BD and T. Losartan 25mg BD with Nephrology follow up.

Discussion

Acute kidney injury in pregnancy is an unusual but life-threatening complication. Among developed countries, PRAKI has decreased to 4% - 4.4% [(6)]. The incidence of acute kidney injury in pregnancy (PRAKI) has markedly decreased over the last three decades in India, particularly due to decreased incidence of post abortion AKI. However, PRAKI still accounts for 3%-5% of cases of total AKI. Currently, in India, majority of PRAKI (70%-90%) occurs in the postpartum period and in late third trimester similar to the developed countries [(4)].

Pregnancy related AKI represents a unique disease process. Any disease process that causes AKI in a non-pregnant patient can cause PRAKI, but there are causes of AKI that are unique to the pregnant state. The causes of PRAKI can be divided into those that occur before twenty weeks of gestation, and those that occur thereafter. The most common

causes of PRAKI before twenty weeks gestation include sepsis (urosepsis, septic abortion or pneumonia) and hypovolaemia (hyperemesis gravidarum and gastroenteritis). Additional causes include drugs such as diuretics which can lead to hypovolaemia and non-steroidal anti-inflammatory drugs. After twenty weeks gestation, causes unique to pregnancy include preeclampsia (PE), acute fatty liver of pregnancy, microangiopathic haemolytic syndromes (TTP, HUS), HELLP syndromes and renal cortical necrosis. Other disorders that should also be considered include systemic lupus erythematosus flare, antiphospholipid syndrome and pre-existing renal disease

Worldwide, Pre-eclampsia is the most common cause of PRAKI [3]

Pregnancy related AKI can be masked by the physiological changes in pregnancy. A serum creatinine value taken before pregnancy or early in the first trimester along with the routine booking

bloods will be invaluable as a comparison to rule out CKD. As this is not routinely performed, PRAKI is often missed until the symptoms are profound. This may result in permanent renal injury before clinical interventions are initiated. As many of the causes of PRAKI overlap and can be difficult to diagnose, a systematic approach to PRAKI diagnosis is needed. Upon presentation, a thorough history, review of medications and examination are essential.

Initial investigations should include a urine dipstick, microscopic analysis of the urine, a full blood count, liver function test, urea creatinine and electrolytes, lactate dehydrogenase and a renal ultrasound.

Management of PRAKI requires identification of the underlying cause and rapid correction of the insult, as the consequences can be dire to both the mother and the unborn foetus.

Prevention of mild PRAKI can often be accomplished by the early recognition of hypovolaemia and treating it. The aim of management of PRAKI should be the maintenance of euvolemia, restoration of normotension, avoidance of nephrotoxic agents and maintenance of urine output above 0.5ml/kg/hour. The intravenous fluid of choice are crystalloids. Additional supportive management like correction of hyper kalaemia and acidosis should be initiated as soon as detected. Hyper kalaemia is often asymptomatic in patients with AKI, but the consequences can be devastating like cardiac arrhythmias, conduction defects, weakness and paralysis. If the acute measures (insulin, β

adrenergic agonists or sodium bicarbonate) fail, urgent Renal Replacement Therapy (RRT)is mandatory, as refractory hyper kalaemia is a life-threatening condition. Similarly, acidosis due to AKI is associated with adverse outcomes (cardiac arrhythmias, depression of cardiac function, immune system dysfunction

and generation of a pro-inflammatory state. If acidosis is persistent and does not respond to

supportive measures, RRT is indicated [9]. There are no guidelines on the timing of RRT initiation. However, RRT should be considered in all patients with PRAKI when there are life-threatening indications, such as fluid overload, acid-base and electrolyte abnormalities or oliguria.

Once PRAKI has been diagnosed and managed, the postpartum management of such a patient should include a screening urea and creatinine, along with a urinary dipstick analysis. If any abnormality is detected, prompt referral to a nephrologist is warranted for further management of ongoing renal disease due to, or worsened by the PRAKI [3].

Sandeep Saini et al conducted a study to look into the etiologies, prevalence, and outcome of pregnancy-related

acute kidney injury and reported sepsis as the major cause of AKI despite increase in the number of institutional deliveries [8]. A Nigerian study compared the etiologies with their short-term outcomes and concluded that Obstetric haemorrhage is the most common cause of PRAKI [7]. Abhinav Banerjee et al compared Standard Conservative Treatment and Early Initiation of Renal Replacement Therapy (RRT) in Pregnancy-related Acute Kidney Injury and found no difference in maternal mortality and morbidity with early and late RRT. But they observed that the fetal outcome was significantly better for patients with early RRT than conservative treatment in PRAKI patients. [9] With no definite protocol for management of Pregnancy Related Acute Kidney Injury in pregnancy, more evidence-based literature and case reports on this condition is the need of the hour.

Conclusion

PRAKI is a silent and often overlooked syndrome, which is associated with dire consequences for both the mother and the fetus. The effect of PRAKI in maternal and fetal outcome is very notable.

The significance of pre-conceptional counselling in chronic hypertensive patient should be recognized. Every pregnant lady with chronic hypertension should be evaluated at the booking visit with renal parameters and requires a multi-disciplinary approach involving Nephrology and Cardiology. Hence, a clarity to the grey areas regarding the early diagnosis and management of Pregnancy Related Acute Kidney Injury is salient.

The magnitude of the disease burden should be acknowledged by each clinician and a keep a low threshold for evaluation and diagnosis of AKI in pregnancy.

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