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A CASE REPORT ON PREGNANCY RELATED ACUTE KIDNEY INJURY

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Key Words
Pre eclampsia, Pregnancy related acute kidney injury, Physiological changes

Acute kidney injury is associated with fetomaternal morbidity and morbidity unless timely evaluated, diagnosed and managed. Complete understanding of renal physiological changes occurring at pregnancy helps in proper intervention. AKI in prerenal cause is due to gravidarum, hyperemesis and uterine hemorrhage due to placental abruption. Intrinsic causes include infections from septic abortion, bilateral cortical necrosis and acute tubular necrosis. Attention should be paid to specific conditions that cause AKI in 2nd and 3rd trimester namely preeclampsia, HELLP syndrome, acute fatty liver of pregnancy and thrombotic micro angiopathies of pregnancy These special conditions share several clinical features of thrombotic microangiopathy which makes diagnosis very difficult. Appropriate causes should be identified and treated intensively, along with dialysis. We here with a case report of pregnancy related acute kidney injury.

INTRODUCTION

AKI in pregnancy usually occur due to obstetrical complications like septic abortion, intrauterine fetal death and puerperal sepsis with previous healthy kidney. There is a huge difference in epidemiological characteristics of PRAKI with respect to incidence, causes and outcomes between developed and developing countries because of many factors like environmental, socio economic and different health delivery systems (1-4). It has declined gradually from1/3000 in 1960's to 1/15000 – 1/20000 in the recent year with respect to number of pregnancies and its incidence has declined from 20 -60% of 1960 to 0-1% in last decade(4). Despite decreasing incidence pf PRAKI, it still accounts 5-20% of total AKI population (2, 5, 6). We hereby report an interesting case of preeclampsia causing AKI but successfully managed by simple medication.

Case Report: A 26 years old unbooked multigravida of 25 weeks 5 days gestation with preeclampsia had attended to antenatal OPD of tertiary care teaching center with c/o anasarca since 20 days and SOB since 15 days. She was not on regular antenatal checkups.

Etiology of AKI in Pregnancy: Development of AKI in pregnancy is by bimodal distribution with two incidence peaks, one at 1st trimester caused due to septic abortion and other in 3rd trimester around delivery due to obstetrical complication. AKI occurs due to multiple etiologies in healthy women .it occurs due to obstetrical complication or pregnancy related medical conditions like preeclampsia or hemolysis, elevated liver enzymes and low platelets syndrome. Some other conditions that are not related to pregnancy like gastroenteritis, malaria, lupus nephritis are also reported to cause P-AKI.

Clinical Examination: She was moderately built, BMI: 21.6, Puffiness of face and pedal oedema, BP: 140/110 mmHg.

Systemic Examination: Heart and lungs sounds: normal, Prior mode of delivery – cesarean 7 years back. Now with breech position FHR: variable 100-110/mt

Investigations:Base Line Investigations:
BP: 140/110mmHg, Hb: 8.2 g/dl, Serum creatinine: 1.21 mg/dl, Proteins: 3+++, Urine protein: 3650 ml/day, Uric acid: 5.3mg/dl. USG scan of kidney: Mild right hydro nephrosis

Diagnosis: 25 years aged multigravida with 25 weeks 5 days gravid with severe preeclampsia

Management: A case admitted to Nephrology ward with chief complaints of anasarca since 20 days, SOB since 15 days. The general measure to reduce renal injury (such as avoidance of nephrotic drugs) should be started. Then administration of IV fluids to maintain renal perfusion (7, 8). This will prevent hypovolemia which ensures fetal wellbeing. The pharmacological therapy of AKI of known complications like preeclampsia, anemia, metabolic acidosis. Angiotensin ll receptor antagonists and angiotensin converting enzymes are contraindicated in pregnancy. Diuretics are not preferred because of high risk of volume depletion. The first line treatment in pregnancy related AKI includes labetalol (9) to correct HB levels ferrous ascorbate was prescribed.

Discussion: AKI in pregnancy require special attention because it involves two lives. Renal in sufficiency in pregnancy is due to prerenal and ischemic causes, it can also be due to pregnancy disorders (10, 11). The incidence of AKI has decreased mainly because of reduction in septic abortion after legalization of abortion, increased antenatal checkups, early identification and treatment of maternal complications of pregnancy like preeclampsia (12). Caring pregnant women with AKI is a challenge for both obstetrician and associated medicate team (13). AKI is a clinical syndrome where there is a decline in kidney function evidenced by increase in serum creatinine levels. AKI is defined as increase in serum creatinine by 0.3mg/dl within 48 hrs. Increase in serum creatinine to 1.5 folds from baseline which occurs within prior 7 days (14). Preeclampsia is one of the main complications in AKI. It is characterized by new onset hypertension (B.P>140/90mmHg) and proteinuria (7300mg/dl) after 20 weeks of gestation (15-17). It also include systemic complications (such as thrombocytopenia, renal insufficiencies, elevated levels of liver enzymes) favours the diagnosis of PE in absence of proteinuria (18, 19). PE occurs during late 2nd or early 3rd trimester, it may also occur up to the time of delivery. The pathogenesis of PE is not fully understood but it involves defects of systemic endothelial factors and placentation (20,-22). Placenta requires extensive angiogenesis for adequate blood supply to meet fetal needs. The angiogenic factors are increased in PE which is hypothesized to decrease the activity of pro angiogenic factors resulting in proteinuria, HTN, endothelial dysfunction (23-25). The treatment of PE depends on illness severity, gestational age, and fetal condition. Before 24 weeks, pregnancy discontinuation is appropriate since studies shown no fetal survival benefits and high risk to the mother.(26,27) Between 25-32 weeks, expectant management is reasonable approach (28). Delivery is treatment of choice in >32 weeks.

Treatment for this case:

BRAND NAME	GENERIC NAME	DOSE	ROUTE	FREQUENCY
Inj.Piptaz	Piperacillin + Tazobactum	4gm + 500mg	IV	BD
Inj.Rantac	Rantidine	2CC	IV	BD
T.Lobet	Labetalol	100mg	Oral	BD
Inj.Neodrol	Methyl prednisolone	1gm	IV	OD
T.Feronia	Ferrous ascorbate	100mg	Oral	BD
T.Shelcal D3	Calcium	500mg	Oral	OD

Better knowledge , high standard of living and on regular antenatal checkups have decreased the incidence of pregnancy related AKI caused by home deliveries, septic abortions, renal ischemia, maternal morbidity and mortality from AKI has decreased drastically by early diagnosis, infection control and appropriate treatment

Conclusion:

In modern era of antibiotics with all aseptic precautions of life threatening complications like pregnancy induced AKI can occurs. They anticipate complications with identification of risk individuals and treatment of underlying causes like sepsis, pre eclampsia, HELLP syndrome, severe hemorrhage has remain the corner stone of management. Previous hypertensive disorder of pregnancy was the most common risk factor for development of AKI. Approximately two third of women recovered from P-AKI

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