



## Obstetric Acute Kidney Injury - Experience at a Tertiary Health Care Centre

Dr. Anushka S Mehta

Dr. Silkey V Mittal

Dr. Pushpa A Yadava

Corresponding author - Dr Parav D Shah

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### INTRODUCTION:

Acute kidney injury is a heterogeneous syndrome defined by rapid (over hours to days) decline in the glomerular filtration rate which results in reduced clearance of nitrogenous products such as urea and creatinine.<sup>(1-2)</sup> It is a major cause of maternal morbidity and mortality especially in developing countries. The incidence of obstetric AKI has decreased in the developed countries (1-2.8%) due to better antenatal care. However in developing countries the incidence is high (4.25%). In developing countries it is mainly due to limited accessibility of health care facilities.<sup>(6-7)</sup> To understand about the obstetric AKI we should first know about the physiological renal changes in pregnancy.

Pregnancy causes an increase in renal plasma flow by 50–70%, while GFR increases by 30–50%. Unlike diabetic nephropathy, pregnancy-induced hyperfiltration is not deleterious to kidneys since both afferent and efferent arterioles are dilated without intraglomerular hypertension. This physiological hyperfiltration results in lowering the normal level of serum creatinine to 0.4–0.5 mg/dL. Thus, even a serum creatinine level of 1 mg/dL is suggestive of substantial reduction in GFR. A diagnosis of pregnancy-related AKI is suggested when serum creatinine rises by 0.5 mg/dL within 48 hours of a potential renal insult.

Severe AKI associated with pregnancy is less frequent today although acute renal ischemia is still often associated with septic abortion in early trimester and severe preeclampsia and obstetric haemorrhage in late trimesters. The above mentioned aetiologies being most common; HELLP (Haemolysis, Elevated liver enzymes, Low platelet) syndrome, septicaemia, severe hyperemesis gravidarum resulting in dehydration, intrauterine death, acute fatty liver of pregnancy and thrombotic microangiopathies are also contributory to obstetric AKI.<sup>(3-5)</sup>

Thorough history will point toward the potential offending factor in AKI. Focused physical examination looking for signs such as fever, volume depletion/overload, jaundice, anemia, purpura, ecchymosis, jaundice, hepatic tenderness, and palpable kidneys should be undertaken. Laboratory investigations to know about electrolytes, renal function, complete blood count should be obtained. Meticulous urinalysis will be rewarding. Dipstick evidence of hematuria, proteinuria, and infection should be sought. Presence of glomerular red blood cells (RBCs) and RBC casts favor glomerular disease such as lupus nephritis. White blood cell (WBC) casts and pyuria favor APN. In prerenal azotemia, the urine fractional excretion of sodium is less than 1 and is more than 1 for the intrinsic renal and post renal cause.

Identifying the risk factors for AKI in pregnancy and their timely correction can help preserve the renal function. Although if the renal function deterioration has begun then identification of the cause and appropriate management in the intensive care unit along with prompt volume resuscitation with either crystalloid solutions or blood products for shock, controlling the blood pressure, correction of sepsis with appropriate antibiotics and correction of electrolytes with renal function forms the basis of the management of obstetric AKI. Involving a nephrologist for a multidisciplinary approach is a must for management of AKI. Following are the common etiologies of AKI in pregnancy.



Causes of AKI in pregnancy in early trimester are septic abortion, hyperemesis gravidarum. In late trimester, the causes are Preeclampsia/ HELLP syndrome, Obstetric hemorrhage (including antepartum and postpartum hemorrhage), TTP (thrombotic thrombocytopenic purpura) /HUS (hemolytic uremic syndrome), Acute fatty liver of pregnancy, Lupus nephritis.

#### OBJECTIVES-

1. To study the causes leading to obstetric AKI in order to aid in future studies for preventive aspects of progression to AKI.
2. To study the impact of AKI in maternal morbidity and mortality.

#### I. METHODOLOGY:

This retrospective study was carried out over a period of 8 months from July 2019 to February 2020 in the Department of Obstetrics and Gynaecology, at SVPIMS Hospital, Ahmedabad, Gujarat. Total 25 cases were studied and analysed. Medical records including progress notes, detailed history, laboratory investigations repeated periodically, management protocols were studied and analysed. Moreover, data about blood transfusions, the time duration from the onset of AKI to initiation of dialysis, number of dialysis, urinalysis, biochemical and hematological tests, length of hospitalization, and changes in the maternal renal functions at the time of discharge were also recorded in a predesigned data record form.

Diagnosis of AKI was established according to the criteria of Acute Kidney Injury Network (AKIN) [11]. 14

STAGES OF AKI	S. CREATININE	URINE OUTPUT
STAGE 1	$\geq 1.5$ -1.9 times the baseline	$< 0.5$ ml/kg/hr for more than $\geq 6$ hours.
STAGE 2	$\geq 2$ -2.9 times the baseline	$< 0.5$ ml/kg/hr for $\geq 12$ hours.
STAGE 3	$\geq 3$ times the baseline	$< 0.3$ ml/kg/hr for $\geq 24$ hours, Anuria for $\geq 12$ hours.

AKIN stage 3 patients were enrolled for hemodialysis, i.e., serum creatinine levels  $> 4$  mg/dL with an increase of more than 0.5 mg/dL or serum creatinine three times of baseline value or oliguria with urine output less than 0.3 mL/kg/hour for 24 hours or anuria for 12 hours. Renal recovery was analyzed in terms of renal function testing at the time of discharge from the hospital. Complete recovery was defined as the return of serum creatinine level to normal or  $< 1.4$  mg/dL. Return of serum creatinine value to  $\geq 1.4$  mg/dL after three months of AKI diagnosis was defined as partial recovery. The third category was a dialysis-dependent end-stage renal disease (ESRD) or all-cause mortality. Sepsis was defined according to the criteria of Systemic Inflammatory Response Syndrome (SIRS). Pre-eclampsia was considered

as hypertension with proteinuria after 20 weeks of gestation and its progression to eclampsia in patients presenting with seizures.

#### >Inclusion Criteria:

All pregnant and postpartum patients admitted to the hospital having the following:

- 1) Oliguria: 24 hour urine output  $< 400$ ml
- 2) Anuria
- 3) S. creatinine  $> 1.5$  mg/dl
- 4) Rise in S.creatinine  $> 0.3$ mg/dl within 2 days

#### >Exclusion Criteria:

Pregnant patients with existing chronic kidney disease or insufficiency prior to pregnancy, diabetes and chronic hypertension were excluded from the study.



## II. RESULTS:

TABLE 1 : DEMOGRAPHIC DISTRIBUTION

<b>AGE</b>	<30 yrs	11 (44%)
	>30 yrs	14 (56%)
<b>PARITY</b>	primigravida	14 (56%)
	multigravida	11 (44%)
<b>ANTENATAL CARE</b>	Regular	5 (20%)
	not taken	20 (80%)
<b>EDUCATION STATUS</b>	Below primary	15 (60%)
	Below HSC	7 (28%)
	Above HSC	3 (12%)
<b>REGISTRATION</b>	Registered	10 (40%)
	Emergency	15 (60%)



<b>SOCIO ECONOMIC STATUS</b>	Upper	1(04%)
	Middle	4 (16%)
	Lower	20 (80%)

As shown in **Table 1**, obstetric AKI was seen in 44% of the patients below 30 years and 56% of the patients above 30 years. There were 56% primigravida patients and in multigravida 44%. Only 20% of patients had regular antenatal visits. 60% of patients in our study had received

below primary level of education considering that the majority of our study included rural population. 60% of the patients were unbooked patients. In our study, 96% of the patients were from the middle class and lower class.

#### FIGURE1 : ETIOLOGY OF PREGNANCY RELATED ACUTE KIDNEY INJURY

As shown in **Figure 1**, Out of the 25 cases studied the most common etiology for Obstetric AKI was found to be Hypertensive disorders in pregnancy (52%); Preeclampsia (44%) and

Eclampsia (8%) . The second most common cause was Obstetric haemorrhage (32%) Antepartum Haemorrhage (20%) and Postpartum haemorrhage (12%).

**TABLE 2 : CAUSES OF AKI RELATED TO PARITY**

PARITY	Pre eclampsia	Eclampsia	APH	PPH	Septicemia	HELLP Syndrome
PRIMIPARA	7 (63.6%)	1 (50%)	2 (40%)	1 (33%)	1 (100%)	2 (67%)

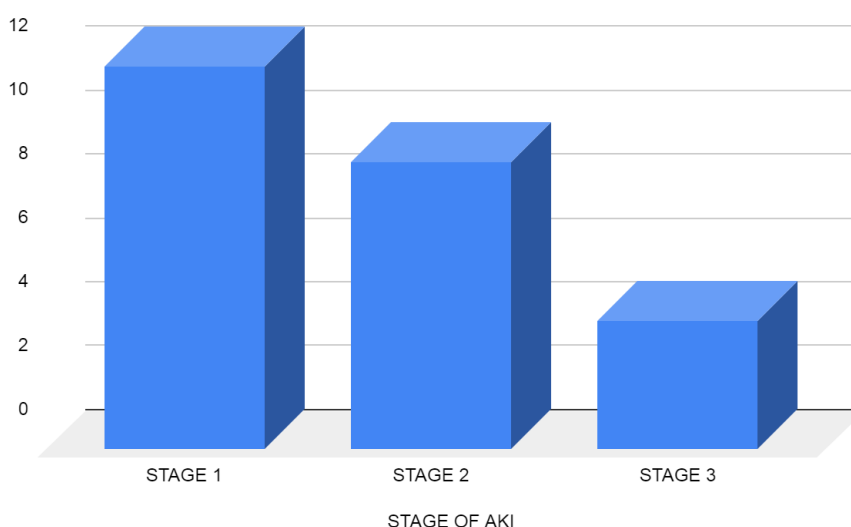


MULTIPAR A	4 (36.3%)	1 (50%)	3 (60%)	2 (67%)	0	1 (33%)
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As shown in **Table 2**, Pre eclampsia was more common in primiparous patients (63%) as compared to multiparous patients (36.3%). While, obstetric hemorrhage was more common in

multipara patients (60% of antepartum hemorrhage and 67% of postpartum hemorrhage) as compared to primipara patients (40% of antepartum hemorrhage and 33% of postpartum hemorrhage).

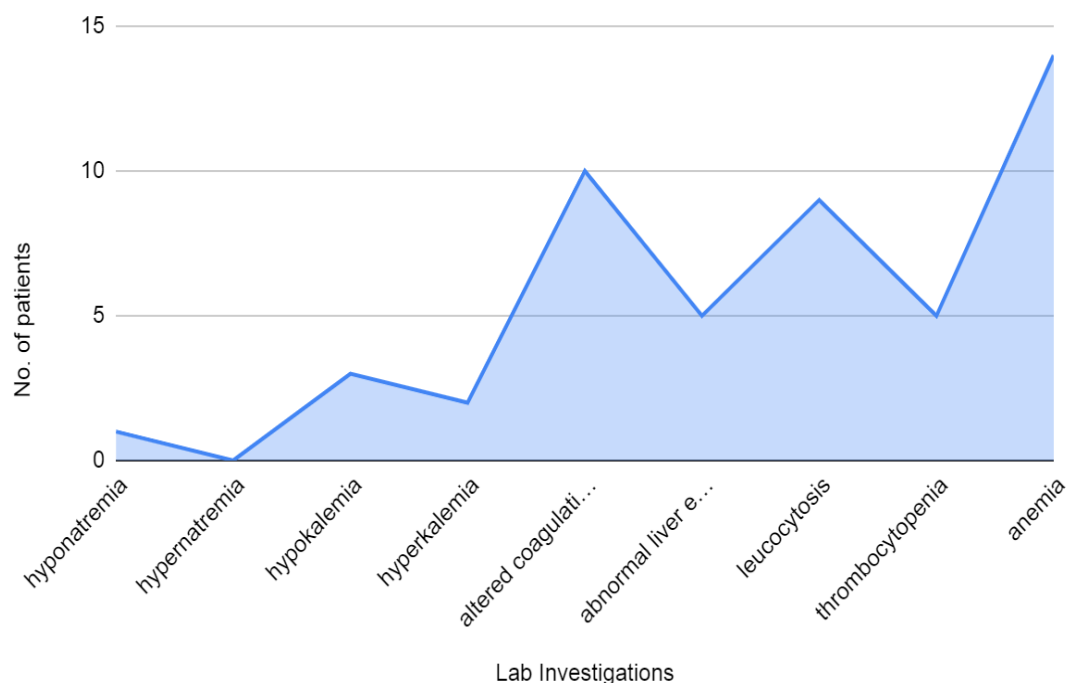
**TABLE 3 : CLASSIFICATION INTO STAGES OF AKI**



As shown in Table 2, out of the 25 patients of Obstetric AKI, 4 patients out of 25 (16%) patients were classified as stage 3 AKI while 9 patients (36%) were categorized under stage 2 AKI and 12 patients out of 25 (48%) under stage 1 AKI.

As Classification of stages of AKI is based on Serum creatinine, it shows that Serum creatinine is an important biochemical parameter for progression of disease severity.

**FIGURE 2 : LABORATORY INVESTIGATIONS IN OBSTETRIC AKI**



As shown in **figure 2**, out of the 25 cases of obstetric AKI, 56% of the cases had anemia followed by altered coagulation profile among 40% patients. Leukocytosis was seen in 36% patients

and electrolyte abnormalities of sodium and potassium were the least common with obstetric AKI [hyponatremia (4%), hyperkalemia (8%), hypokalemia (12%)].

**FIGURE 3: LABORATORY PARAMETERS DERANGED AND THE RESULTANT SEVERITY OF AKI**

	MILD AKI	MODERATE AKI	SEVERE AKI
Anemia	6 (50%)	4 (44.44%)	4 (100%)
Altered coagulation	3 (25%)	5 (55.56%)	2 (50%)
leukocytosis	2 (16.67%)	4 (44.44%)	3 (75%)
Thrombocytopenia	2 (16.67%)	2 (22.22%)	1 (25%)
abnormal liver tests	1(8.33%)	3 (33.33%)	1 (25%)

As shown in **figure 3**, anemia was seen to be present in 50% of the mild cases, 44% of moderate cases and in 100% cases of severe AKI. Altered coagulation profile was seen in 25% mild

cases, 55.56% moderate cases of AKI and 50% of severe cases. Leukocytosis was seen in only 16.67% mild cases, 44.44% moderate cases and 75% of severe or stage 3 AKI.



**TABLE 3: MATERNAL OUTCOME IN ACUTE KIDNEY INJURY**

OUTCOME	NUMBER OF PATIENTS	PERCENTAGE
Recovery	16	64%
Partial recovery	2	8%
Dialysis dependant	4	16%
Death	3	12%

As shown in **Table 3**, Out of 25 cases of obstetric AKI, 18 patients recovered. Complete recovery was seen in 16 patients (64%) and partial

recovery was seen in 2 (8%) patients. 4 patients (16%) required dialysis. 3 (12%) patients died during the course of treatment.

**TABLE 4 : FETAL OUTCOME**

	NO.OF PATIENTS
Full term live birth	10 (40%)
Preterm live births	7 (28%)
IUDs	6 (24%)
Stillbirth	1 (4%)
ABORTION	1 (4%)

As shown in **table 4**, Out of 25 cases of obstetric AKI, 40% Patients were born full term, 28% were preterm, 24% fetus died in utero, while 4% fetuses were still born and 4% fetuses were aborted.

### III. DISCUSSION:

In our study the most common age group of presentation was above 30 years(56%) . In studies done by Vineet et al <sup>(8)</sup>, and Goplani et al <sup>(6)</sup> , the mean age of presentation was found to be 26-30 years.

Majority of patients in our study were unbooked patients (60%) and presented as emergency cases. Only 20% of the patients in our study received regular antenatal care, highlighting the fact that primary prevention of obstetric AKI should commence in the antenatal period itself, proper blood pressure monitoring, and maintaining

normal hemoglobin levels, weight, urine albumin early dx, idx and timely identification from the first visit and during each visit by appropriate hematinics. A study conducted by Sushma et al (16) also reported that 66% patients in their study were unbooked and did not receive adequate antenatal care.

Hypertensive disorders of the pregnancy (52%) with 44% cases of preeclampsia and 8% cases of eclampsia, were found to be the most common cause of obstetric AKI in our study followed by Obstetric hemorrhage which constituted 32% of the total causes of AKI in our study. This goes in accordance with the study done by Gayathri et al which shows hypertensive disorders as the most common cause<sup>(9)</sup>(39%). Hypertensive disorders being the most common cause is, in fact, preventable and hence, patients



should be assessed regularly from the first antenatal visit and blood pressure should be well controlled during pregnancy to prevent the complications, Obstetric AKI, being one of them.

Goplani et al <sup>(6)</sup> , reported Obstetric haemorrhage as the most common cause of obstetric AKI (38%) . However, obstetric hemorrhage was an important contributing cause of AKI in our study highlighting the importance of control of hemorrhage since the antenatal period as well as measures to prevent antepartum and postpartum hemorrhage as an important factor for preventing the number of cases from progressing to AKI.

16% of the patients were classified as stage 3 AKI and required dialysis eventually. Bokhari et al. (15) reported 68.3% patients who needed dialysis. This may be influenced by the fact that 80% patients in their study were already admitted with stage 3 AKI as compared to our study where only 16% patients had stage 3 AKI on admission. Early treatment and timely referral are crucial in the management of this complication and prevent the long term implications of renal insult.

We reported maternal mortality of 12% which is similar to the study done by Khalil et al (15%) and Godara et al <sup>(10)</sup> (15%). Goplani et al reported a maternal mortality of 18.5% and Chaudhari et al <sup>(11)</sup> reported a mortality rate of 33%. This significant mortality rate of AKI in all studies shows the magnitude of the problem of Acute Kidney Injury in obstetrics and thus, need for initial prevention of complications and prompt interventions once the renal insult has started to occur.

We observed in our study that the majority of patients of stage 1 and stage 2 AKI were managed with blood products and fluid resuscitation, while the two patients that were referred from primary health care centres due to postpartum hemorrhage, progressed to stage 3 AKI. One patient of HELLP syndrome died during the course of treatment. It was also observed that in majority of patients in the stage 2 and stage 3 of AKIN had anemia and altered coagulation profile, anemia being more common of the two, suggesting the importance of prevention of factors like

hemorrhage that lead to anemia and urgent treatment by blood transfusions if at all occur.

Anemia was the most common laboratory parameter to be deranged in all severe cases of AKI, which occurred more in cases of hypertensive disorders and obstetric hemorrhage, highlighting the importance of prompt blood and blood products transfusion to treat hemorrhage (antepartum and postpartum hemorrhage). Leukocytosis was seen in 3 out of 4 cases of severe or stage 3 AKI showing tha septicemia whether cause or an aggravating factor to the cause of AKI needs to be aggressively treated.

Out of the three deceased patients, two patients who had presented with Post-partum haemorrhage died within two days and sixteen hours of admission respectively. The third patient deceased was a case of HELLP syndrome. Obstetric hemorrhage which along with being a common contributing factor to AKI is also a dangerous factor contributing to mortality in patients with acute kidney injury needs prevention in the first place. This can be effectively done by a skilled obstetrician who can anticipate the occurrence of hemorrhage and be well equipped and well prepared with measures to control obstetric hemorrhage. However, once it has occurred, immediate control of the source of hemorrhage, prompt blood and blood products transfusion at a tertiary care centre is the only saviour.

In our study, 40% patients were liveborn at full term, Out of the rest 60% patients with poor fetal outcome, 28% were delivered preterm, 24% fetuses died in utero, 4% were still born and 4% were aborted. Sushma et al (16) also reported poor fetal outcome in 44% patients. This highlights the importance of prevention and treatment of obstetric AKI for maternal as well as fetal well being.

#### IV. CONCLUSION:

The spectrum of AKI in obstetrics is a serious contributor of long term maternal morbidity and mortality. However, we have observed that almost all causes of AKI are preventable and proper antenatal management since the first point of contact with the care provider, along with prior anticipation of the causes that may lead to AKI can drastically reduce the morbidity and mortality in





obstetric patients which is around 12-15% due to AKI.

An obstetrician should have a broad knowledge about the physiological alterations in the renal system in pregnancy to apply the best evidence based strategies in treating patients with AKI considering both maternal and fetal effects of the pathology and its management. Early diagnosis and prompt management in a tertiary care hospital can avoid potential life threatening AKI and its sequel.

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