Original Article

Risk factors and fetomaternal outcome in pregnancy-related acute kidney injury

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Abstract

Introduction: Pregnancy-related acute kidney injury (PRAKI) is acute kidney injury (AKI) occurring during pregnancy, labor, and postpartum period. AKI is defined as suddenly impaired kidney function with the retention of nitrogenous and other waste products. In high population country like India, not all deliveries are done tertiary care. Even not all are registered one if delivery is conducted at a hospital setup. The majority of patients are being managed by available obstetrician at local places. Early diagnosis and timely management of complications related to pregnancy are very important to avoid PRAKI. We aim to study maternal risk factors and fetomaternal outcome in PRAKI. **Materials and Methods:** A prospective study is conducted between 2021 and 2022 in the Department of Obstetrics and Gynaecology, VMMC, and Safdarjung Hospital, New Delhi. For antenatal and delivered women up to 6 weeks, 50 patients were recruited according to KDIGO (Kidney Disease International Global Outcomes) criteria. Patients were followed with CBC, serum electrolytes, serial KFT, urine input/output monitoring, and USG-KUB. Dialysis was done if indicated. Complete renal recovery was considered if S.Cr ≤1.0 mg/dl within 6 weeks of diagnosis of AKI. For statistical significance, a *P* value of less than 0.05 was considered. **Results:** The majority of patients were unbooked, 21–25 years of age, and belonged to lower socioeconomic status (54%). Risk factors were: preeclampsia (28%), puerperal sepsis (24%), PPH (20%), abruption (14%), pyelonephritis (4%), acute gastroenteritis (4%), gestational hypertension with superimposed preeclampsia (2%), antepartum eclampsia (2%), and thrombotic microangiopathy (2%). Hemodialysis is required in 23 (46%). Complete renal recovery was seen in 40 (80%) and partial renal recovery in 3 (6%). Maternal mortality was 14% and causes were: puerperal sepsis (57%), preeclampsia with severe features with MODS (29%), and antepartum eclampsia with hepatorenal failure (14%). Fetal outcome: 76% live birth, 24% intrauterine death, and 16% early neonatal death. **Conclusion:** Most common risk factors for PRAKI are preeclampsia followed by puerperal sepsis and PPH where all are preventable causes.

Keywords: Acute kidney injury (AKI), Postpartum hemorrhage (PPH), preeclampsia, pregnancy, pregnancy-related acute kidney injury (PRAKI)

Introduction

Pregnancy-related acute kidney injury (PRAKI) is acute kidney injury occurring during pregnancy, labor, and the postpartum period up to 6 weeks. Acute kidney injury (AKI), previously termed as acute renal failure, is used to describe suddenly impaired kidney function with retention of nitrogenous and other waste products normally excreted by the kidneys. According to KDIGO (Kidney Disease Improving Global Outcomes) guidelines: AKI is defined as;

- Increase in SCr by ≥0.3 mg/dl (≥26.5 μmol/l) within 48 hours; (or)
- Increase in SCr to ≥1.5 times baseline, which is known or presumed to have occurred within the prior 7 days, (or)
- Urine volume <0.5 ml/kg/h for 6 hours.

Pregnancy-related acute kidney injury (PRAKI) contributes to 3–7% of overall acute kidney injury (AKI) cases in the Indian

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subcontinent. PRAKI is a major cause of maternal and fetal morbidity and mortality in developing countries. The incidence of PRAKI was 1.56%, and the mean age of the study population was 25 years.[1] With improvement in antenatal and postnatal care, the incidence of PRAKI in India has steadily declined from 22% in the 1960s to 9% in the 1980s, and further down to 3–7% in the 2000s; however, the levels continue to remain higher than the levels seen in developed countries (1 in 20,000 pregnancies).[2-5]

Physiologic and anatomic changes in the kidney during pregnancy

There are considerable changes that occur in the urinary tract system during normal pregnancy: kidneys increase in size by about 1–1.5 cm due to renal vascular and interstitial space volume expansion. The physiological hydronephrosis of pregnancy characterized by a dilation of the calyces, renal pelvis, and ureter occurs in over 90% of pregnant women.[6]

This anatomical abnormality may be present until the 16th postpartum week and promotes urinary stasis in the ureter, leading to the development of urinary tract infection. The dilatation of the urinary system is due to the hormonal effects of progesterone, external compression by the gravid uterus, and morphological changes in the ureteral wall. The systemic vasodilatory state, typical of pregnancy, increases renal perfusion and glomerular filtration rate (GFR). The GFR increment causes an increase in uric acid clearance, leading to hypouricemia. Proteinuria also increases and urinary protein excretion of 300 mg/day is considered physiological in normal pregnancy. Renal plasma flow can increase up to 85% in the second trimester of pregnancy. The GFR can reach 40%–50% of baseline throughout pregnancy and subsides in the first 3 months postpartum. These hemodynamic abnormalities result in a decrease in serum creatinine in pregnant women to 0.4–0.5 mg/dl. Systematic vasodilation leads to the stimulation of antidiuretic hormone, resulting in a decrease in plasma osmolality and plasma sodium by 4–5 mEq/L.[7]

Minute ventilation increases due to progesterone-induced stimulation of the central respiratory center in the brain. This results in a decrease in pCO2 and a mild chronic respiratory alkalosis, which is compensated for by the renal excretion of bicarbonate. A decrease of about 4 mEq/L in bicarbonate concentration is common in pregnant women.[8]

Common causes of PRAKI

PRERENAL: Hyperemesis gravidarum, Hemorrhage, Heart failure.

INTRARENAL: Acute tubular necrosis, acute cortical necrosis, acute fatty liver of pregnancy, preeclampsia/HELLP (hemolysis, elevated liver function tests, and low platelet count), thrombotic thrombocytopenic purpura/atypical hemolytic–uremic syndrome, pyelonephritis, pulmonary embolism, amniotic fluid embolism, lupus nephritis, acute intestinal nephritis.

POSTRENAL: Hydronephrosis due to uterine compression, injury to the ureter/bladder/urethra during cesarean section or vaginal delivery, ureteral obstruction from stones or tumor, obstruction at the bladder outlet.[9]

Management of PRAKI can be complex for the following reasons

(1) There are two patients to consider, the mother and her fetus.
(2) Renal and cardiovascular adaptations of pregnancy affect diagnosis and management.
(3) A multidisciplinary team approach is warranted (including specialists in critical care medicine, maternal–fetal medicine/high-risk obstetrics, nephrology, and neonatology).

Renal recovery: 1) Partial renal recovery—decreasing level of serum creatinine. 2) Complete renal recovery—decline in S. Cr to ≤1.0 mg/dl within 6 weeks of diagnosis of AKI.

Materials and Methods

A prospective study was conducted between 2021 and 2022 in the Department of Obstetrics and Gynaecology, VMMC, and Safdarjung Hospital. We included antenatal and postnatal/postpartum women up to 6 weeks after delivery, 50 patients were recruited according to KDIGO (Kidney Disease International Global Outcomes) criteria of AKI.

A detailed history included maternal age, gravida, parity, last menstrual period (LMP), socioeconomic history, education status of both husband and wife, family income, past medical history, and number of ANC visits taken. A thorough general physical examination and complete obstetric examination have been performed. Risk factors/obstetrics cause of AKI noted. Clinical characteristics of the patients in terms of access to antenatal care, duration of gestation, type and place of delivery, fetal outcome, duration of symptoms prior to admission, urine output, hematological and biochemical profiles, duration of hospitalization, recovery of renal function, and patients' survival were recorded.

Women diagnosed with AKI managed and followed up during pregnancy, labor, and the postpartum period up to 6 weeks as per standard guidelines. All routine ANC investigations were done and noted. Labor events and fetal outcomes were noted. Serial KFT, serum electrolytes, CBC, and other required laboratory investigations for the management of AKI have been done and followed. Daily urine input/output charting was done. A number of dialyses noted. Renal recovery at the time of discharge and during follow-up period up to 6 weeks after a diagnosis of AKI were noted. Renal recovery is taken as a decline in S. Cr to m1.0 mg/dl within 6 weeks of diagnosis of AKI.

The presentation of the categorical variables was done in the form of number and percentage (%). On the other hand, the quantitative data were presented as the means r SD as median with 25th and 75th percentiles (interquartile range). The data normality was checked by using Kolmogorov–Smirnov test. In the cases in

which the data were not normal, we used nonparametric tests. The comparison of the variables across follow-up which were quantitative and not normally distributed in nature was analyzed using Wilcoxon signed-rank test.

The data entry was done in the Microsoft EXCEL spreadsheet and the final analysis was done with the use of Statistical Package for Social Sciences (SPSS) software, IBM manufacturer, Chicago, USA, version 25.0. For statistical significance, a *P* value of less than 0.05 was considered statistically significant.

Results

All 50 (100%) study subjects were unbooked, 42 were referred from a referral center with prior ANC visits either at their center or somewhere, while eight patients had no prior ANC visits.

The majority of 28 (56%) study subjects belonged to the age group 21–25 years. 27 (54%) patients belong to lower socioeconomic status according to the modified Kuppuswamy scale. The distribution of maternal outcome is shown in Table 1 and fetal outcome in Table 2.

The mean value of hemoglobin (g/dl) was 9.51 ± 2.17 with a median (25th–75th percentile) of 9.35 (8.75–11). Similarly, the mean value of total leucocyte count (cells/mm^3) was 12853.8 ± 7123.63 with a median (25th–75th percentile) of 9800 (8825–12900). Similarly, the mean value of platelet count (cells/mm^3) was 139348.94 ± 35693.15 with a median (25th–75th percentile) of 134000 (110500–170000). The distribution of USG kidney is shown in Figure 1.

The mean value of sodium (mEq/L) of study subjects was 133.94 of st with a median (25th–75th percentile) of 134. (131–137) Similarly, the mean value of potassium (mEq/L) of study subjects was 3.78 L) of with a median (25th–75th percentile) of 3.7 (3.5–3.9).

The median (25th to 75th percentile) of blood urea (mg/dL) of study subjects on day 1 was 29.5 (27–41.75).

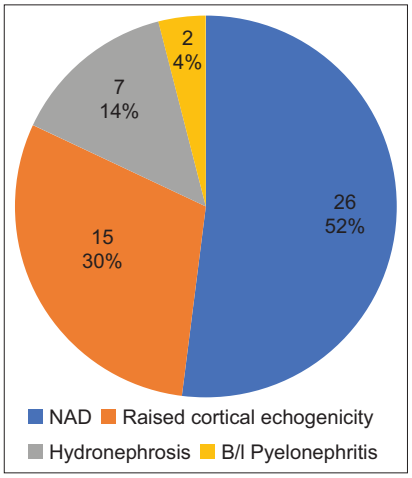

Figure 1: Distribution of USG-KUB of study subjects

Significant improvement was seen in blood urea (mg/dL) of study subjects with partial renal recovery and with complete renal recovery (*P* value < 0.001). The median (25th to 75th percentile) of blood urea (mg/dL) of study subjects with partial renal recovery

Table 1: Distribution of maternal outcome of study subjects

Maternal outcome	Frequency	Percentage
HDU/ICU stay		
Yes	50	100.00%
Recovered and discharged/maternal mortality		
Maternal mortality	7	14.00%
Recovered and discharged	43	86.00%
Cause of death		
APE (Ante partum eclampsia) with Hepatorenal failure	1	14.29%
Puerperal sepsis	4	57.14%
Preeclampsia with severe features with MODS	2	28.57%
Hemodialysis		
No	27	54.00%
Yes	23	46.00%
Renal recovery—partial/complete		
Complete	40	80.00%
Partial	10	20.00%
Risk factors of AKI		
Preeclampsia	14	28.00%
Puerperal sepsis	12	24.00%
PPH	10	20.00%
Abruption	7	14.00%
Pyelonephritis	2	4.00%
Acute gastroenteritis	2	4.00%
APE (Ante partum eclampsia)	1	2.00%
Gestational hypertension with superimposed preeclampsia	1	2.00%
Thrombotic Microangiopathy	1	2.00%

Table 2: Distribution of fetal outcome of study subjects

Fetal outcome	Frequency	Percentage
Preterm/term		
Preterm	20	40.00%
Term	30	60.00%
Birth weight (gm)		
<1.5 kg	5	10.00%
1.5–<2.5 kg	15	30.00%
≥2.5 kg	30	60.00%
Mean±SD	2576.1±649.72	
Median (25th–75th percentile)	2665 (2153.75–3107.5)	
Range	1190–3540	
NICU admission		
No	38	76.00%
Yes	12	24.00%
Live birth/still birth		
Live birth	38	76.00%
Still birth	12	24.00%
Early neonatal death		
Early neonatal death among live birth	6	16.00%
Total live birth	32	84.00%

was 63 (22–100) which was significantly higher as compared with blood urea (mg/dL) of study subjects with complete renal recovery with median (25th to 75th percentile) of 17 (14.5–18).

The median of serum creatinine (mg/dL) of study subjects on day 1 was 2.1 (1.9–2.675).

Significant improvement was seen in serum creatinine (mg/dL) of study subjects with partial renal recovery and with complete renal recovery (*P* value <0.001). The median (25th to 75th percentile) of serum creatinine (mg/dL) of study subjects with partial renal recovery was 2.4 (1.1–4.4) which was significantly higher as compared with serum creatinine (mg/dL) of study subjects with complete renal recovery with median (25th to 75th percentile) of 0.8 (0.7–0.9).

Discussion

In our study, the majority of subjects were between 21 and 25 years with a mean age of 25.18 ± 3.8 years. A similar result was observed by Mahesh E *et al.*, in which total 165 patients with PRAKI were observed and the mean age of the study population was 25 years.[1] Similarly, In an prospective observational study of Gopalakrishnan N *et al.* mean age was 25.4 ± 4.73 years.[10] Mishra Vineet V *et al.* conducted a study that included patients with an average of 26.2 years.[11] So, our study is comparable with the above references as they also involve the same age group.

In our study, risk factors causing PRAKI were as follows: preeclampsia 14 (28%), puerperal sepsis 12 (24%), PPH 10 (20%), abruption 7 (14%), pyelonephritis 3 (6%), acute gastroenteritis 2 (4%), IHCP 1 (2%), gestational hypertension 1 (2%), APE (antepartum eclampsia) 1 (2%), and thrombotic microangiopathy 1 (2%). Mishra Vineet V *et al.* did a prospective study and found that obstetric hemorrhage (38.46%) is the most common cause of obstetric AKI. Late referral in 18 (34.61%), puerperal sepsis in 6 (33.33%), obstetric hemorrhage in 5 (27.77%), and combined sepsis and hemorrhage in 5 (27.77%) are the common contributing factors.[11] In study of Vinturache A *et al.* the causes of AKI were: sepsis (59%), preeclampsia, and eclampsia (56%) were the leading causes of PRAKI[12] So, the present study is comparable with the above references and has similar risk factors.

In our study, 54% of patients underwent hemodialysis and 46% of patients were discharged without hemodialysis. In the study of Mishra Vineet V *et al.* 4 (7.69%) patients became dialysis dependent.[11] So, the present study is not comparable with the above reference. Patel A *et al.* did a study on association and contributing factors in pregnancy related acute kidney injury at a tertiary referral hospital and found that 59.5% of cases required hemodialysis.[13] So, our study is similar to the above reference. Proper and early ANC care and treatment can prevent AKI to develop and further prevent it to reach at the stage of hemodialysis.

In our study, complete renal recovery occurred in 80% of patients and 20% of patients with partial renal recovery, out of which 7 (14%) patients died and 3 patients continued hemodialysis beyond 6 weeks. In Mishra Vineet V *et al.* study, the outcome was favorable with complete renal function recovery in 55.76% of patients.[11] In the study of Sharma M *et al.*, at the end of 90 day follow up, complete recovery of renal function occurred in 53.8%, partial recovery in 23.1%, and end stage kidney disease (ESKD) in 7.7%.[14] So, our study is comparable with the above references and has similar renal recovery rates.

In our study majority (86%) of patients were discharged, maternal death occurred in 7 (14%) patients. The causes of death were as follow: puerperal sepsis (57.14%), severe PE (28.57%), and APE (14.29%). In study of Sharma M *et al.* maternal mortality in AKI was 15.4%[14], in Mishra Vineet V *et al.* study 32.69%[11], in Saini S *et al.* study 25%[15], Adejumo OA *et al.* had maternal mortality of 34.4%[16]. In study of Mahesh E *et al.* maternal mortality was 20%, with sepsis 59%, preeclampsia, and eclampsia 56% as the causes of death[1].

In our study, significant improvement was seen in serum creatinine of patients on day 1 with on discharge of patients. The median (25th to 75th percentile) of serum creatinine (mg/dL) of patients on day 1 was 2.1 (1.9–2.675) which was significantly higher as compared with serum creatinine (mg/dL) of patients on discharge/after 6 weeks with median (25th to 75th percentile) of 0.8 (0.7–1) with $P < 0.001$. So, in our study 40 (80%) patients have serum creatinine 1 mg/dL at 6 weeks. Liu YM *et al.* did a study and found that the SCr level returned to normal in 18 (72%) patients out of 25, although 6 of these women had persistent proteinuria, including 3 who had miscellaneous causes of AKI. Seven days after the termination of pregnancy, the maternal condition improved, and serum creatinine between the day of admission and the seventh day postpartum.[17] Both studies had statistically significant improvement in serum creatinine.

In our study, 30 (60%) patients were term and 20 (40%) were preterm. Sharma M *et al.* study had a preterm birth in 23.1%.[14] Liu YM *et al.* had 10 (62.5%) preterm delivery.[17]

In the present study, in majority patients 30 (60%); birth weight (kg) was ≥2.5 kg, in 15 (30%) patients; birth weight (kg) was 1.5 to <2.5 kg and birth weight was <1.5 kg in only 5 (10%) out of 50 patients. The mean value of birth weight (kg) of study subjects was 2576.1 ± 649.72 gm. In Sharma M *et al.*, the mean birth weight in newborns delivered by patients with AKI was (2.53 ± 0.73 kg).[14]

In our study, out of 50 babies, 38 (76%) were live birth, 12 (24%) babies were IUD, and early neonatal death occurred in 6 (16%) out of 38 babies. In study of Mahesh E *et al.*, fetal mortality was 22%.[1] Sharma *et al.*, perinatal death occurred in 26.9% and stillbirth in 7.7%.[14] Saini S *et al.*, fetal mortality is 23.5%.[15] Adejumo AO *et al.*, fetal mortality is 50%.[16] Liu YM *et al.*, the rate of stillbirth was 27.0%.[17] Awowole IO *et al.*, perinatal mortality rates were 34%.[18]

Conclusion

Our study concluded that the majority of our patients were unbooked and 21–25 years of age, mostly belonged to lower socioeconomic status. A total of 50 study subjects were included, 42 were referred and 44 patients were postpartum and 6 were antepartum. Our study concluded the major risk factors causing AKI in pregnancy in our tertiary care hospital were: preeclampsia, puerperal sepsis, PPH, abruption, pyelonephritis, acute gastroenteritis, gestational hypertension with superimposed preeclampsia, APE (antepartum eclampsia, and thrombotic microangiopathy. All 50 study subjects stayed in HDU/ICU, out of which 43 (86%) were recovered and discharged. Out of 10 patients with partial renal recovery, in seven maternal mortality occurred while three patients continued hemodialysis beyond 6 weeks of delivery in the nephrology department. The majority of 26 (52%) study subjects USG-KUB were NAD (No abnormality detected), 15 (30%) USG-KUB showed raised cortical echogenicity, seven (14%) USG-KUB showed hydronephrosis, and two (4%) patients USG-KUB showed B/L pyelonephritis. The maternal mortality rate was 14% in our tertiary care hospital. The cause of mortality of study subjects was: puerperal sepsis in 4 (57.14%), preeclampsia with severe features with MODS in two (28.57%), and APE with hepatorenal failure in one (14.29%) out of seven. Fetal outcome: 38 (76%) were live birth, 12 (24%) babies were IUD and early neonatal death occurred in 6 (16%) out of 38 babies. Early ANC registration with regular ANC visits is necessary to prevent such high-risk pregnancy.

Key messages

PRAKI is a common complication seen in routine practice which can be prevented by timely management of all the risk factors. Early ANC registration and regular follow-up can improve both the maternal and fetal outcomes in this condition.

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Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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