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Renal Outcomes of Pregnancy-Related Acute Kidney Injury: a Single Centre Experience in India

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ABSTRACT

Objective: *This study aimed to investigate the incidence, clinical characteristics, and outcomes of acute kidney injury (AKI) during pregnancy in Indian population.*

Materials and methods: *A prospective observational study was conducted in pregnant patients admitted to Pt. B.D. Sharma PGIMS, Rohtak, Haryana, India. Acute kidney injury was assessed using Risk, Injury, Failure, Loss of function, and End-stage renal disease (RIFLE) criteria. Patients were analyzed on the basis of demographic data, detailed history, clinical examination, and laboratory investigations. The primary outcome was maternal renal outcome, including return to normal renal function and progression to chronic kidney disease (CKD). The secondary outcomes included the mode of delivery, complications of pregnancy, intensive care unit (ICU) admission, and maternal death.*

Results: *A total of 51 patients with an average age of 29.5 years were included in the present study. About 49.9% of subjects had severe anemia and 41.2% were primigravida. The main cause of AKI was pre-eclampsia and postpartum hemorrhage. There was a marked improvement in renal outcome with 33 patients having complete renal recovery and six patients developed CKD was observed during three months follow-up period. The peaked median value of blood urea was 62.0 mg% in patients with normal renal function, 178.5 mg% in those with CKD and 120.0 mg% in expired patients (P=0.001). A statistically significant change in serum potassium (P=0.010) and creatinine levels (P<0.001) was observed during the follow-up period. Liver enzymes, including serum glutamic oxaloacetic transaminase and serum glutamic*

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pyruvic transaminase, were high in all patients at the time of admission, but decreased to normal on follow-up.

Conclusion: *Our study indicates that pregnancy-related AKI patients present with multiorgan complications and many of them require mechanical ventilation and renal replacement therapy. Most of these patients have poor outcome. Hence, the management of pregnancy-related AKI presents a challenge that requires proper evaluation of causative factors to facilitate appropriate treatment.*

Keywords: postpartum hemorrhage, pre-eclampsia, serum creatinine, serum potassium.

INTRODUCTION

Acute kidney injury (AKI) is an abrupt and sustained decrease in kidney function, which encompasses both subclinical kidney damage and renal impairment (1). Pregnancy-related AKI (PR-AKI) remains a major cause of maternal and fetal morbidity and mortality (2). The incidence of PR-AKI has declined from 7% to 4.68% between 2000 and 2014, with improvement in antenatal and postnatal care (2, 3). In India, the prevalence of AKI during pregnancy ranged from 0.02 to 11.5% (4-6).

The development of AKI is assessed based on physiological changes occurring during the gestation period. The development is routinely considered as bimodal as it is often different for early and late part of the gestation period even for the postpartum period. Pregnancy-related AKI can occur at any stage of pregnancy, but in the third trimester and the postpartum period is associated with a high (39%) incidence of fetal/neonatal mortality. During the first trimester it is found to be associated with septic shock or dehydration due to hyperemesis. In the third trimester, the condition is associated to pre-eclampsia/eclampsia, antepartum and postpartum hemorrhage, puerperal sepsis, hemolytic uremic syndrome, disseminated intravascular coagulation (DIC), hemolysis, elevated liver enzymes, and low platelet levels syndrome (7). Increasing the risk of superimposed pre-eclampsia and pregnancy-induced hypertensive disorders accounts for higher maternal and perinatal morbidity and mortality rate (8, 9).

Renal replacement therapy and targeted drug therapy are specific therapies available for the management of AKI, but their efficacy has not proven to be significant in a human clinical trial (10, 11). Early identification of risk factors helps clinicians for timely diagnosis of PR-AKI that requires specific therapeutic interventions (12). Mild renal dysfunction is manifested by small urine output and blood chemistries. Serum creatinine is one of the established biomarkers (13). Eventually true fall in glomerular filtrate rate (GFR) may not be reflected by serum creatinine in critically ill patients (14).

The present study is based on the adjunctive diagnosis tools to diagnose AKI. Urine dipstick testing, urine microscopy, renal ultrasonography, renal biopsies help to diagnose existing structural renal disease and/or any obstruction in the urinary system (15).

The development of AKI in pregnancy is a major clinical challenge, since two patients (mother and fetus) have to be considered, and it may be caused by specific pregnancy diseases which are not fully understood so far. Since PR-AKI forms a formidable pool of AKI in India and only few retrospective studies shed light on the incidence, etiology and potential complications of PR-AKI, there was a need for a prospective study. The present study aimed to look for the causes responsible for PR-AKI and to assess the maternal and perinatal renal outcomes of patients with PR-AKI. ❑

METHODS AND MATERIALS

Study setting

The present prospective observational study was carried out in the Department of Obstetrics and Gynecology of Pt. B.D. Sharma PGIMS, Rohtak, Haryana, India, between 1st July 2015 and 31st August 2016. The study was approved by the Ethics Committee at each hospital site. Individual written consent was obtained from each patient. The study was conducted in line with the Declarations of Helsinki.

Acute kidney injury was defined on the basis of Risk, Injury, Failure, Loss of function, and End-stage renal disease (RIFLE) criteria (16). According to the Kidney Disease Improving Global Outcomes (KDIGO) (17), AKI is defined either as an increase in serum creatinine by ≥26.5 µmol/L within 48 h, increase to more than 1.5 times baseline, or reduced urine volume <0.5 mL/kg/h for six hours.

Inclusion criteria

All consecutive patients with AKI irrespective of etiology, i.e., pre-renal, renal, or post-renal, were included in this study and followed-up for three months for the occurrence of chronic kidney disease (CKD).

Exclusion criteria

Patients with preexisting diabetes mellitus, renal diseases, hypertensive nephropathy hypertension, bilateral contracted kidney, renal transplant recipients, or CKD were excluded from the study.

Study methodology

Selected patients were analyzed based on demographic data, detailed history, clinical examination, and laboratory investigations including complete hemogram, blood urea, serum creatinine, serum calcium, serum phosphorous, serum uric acid, serum protein ratio, serum sodium, serum potassium, blood sugar, and bilirubin. Each patient underwent a complete obstetric examination. Alternate day renal functioning tests were performed during hospitalization. A specific inquiry was conducted regarding the mode of delivery. All patients were managed by either conservative treatment or hemodialysis, according to standard indications. After discharge, their clinical characteristics were assessed during the one- and three-month follow-up period. Patients' evaluation was done as per their clinical outcome at three-month follow-up by dividing them into three groups: Group A (patients in whom renal function recovered to normal), Group B (patients who progressed to CKD) and Group C (patients who died after admission).

Outcomes

Maternal renal outcome, including return to normal renal function and progression to CKD re-

Parameter	Total (N=51)
Age (years)	25.9 (4.2)
Parity, n (%)	
Primipara	21 (41.2)
Multipara	30 (58.8)
Gestation period	
<28	4 (7.9)
≥28 to <34	12 (23.6)
≥34 to <37	9 (17.6)
≥37	26 (50.9)
Biochemical parameter	
SBP (mm Hg)	140.2 (32.5)
DBP (mm Hg)	90.4 (30.3)
Hemoglobin (gm)	7.5 (2.9)
TLC (10^3/cmm)	15.4 (7.6)
Platelets (10^3/cmm)	151.5 (124.1)
Blood urea (mg)	68.6 (65.9)
Serum creatinine (mg)	2.2 (2.1)
Serum sodium (mEq/L)	135.5 (7.2)
Serum potassium (mEq/L)	4.6 (0.8)
Serum calcium (mg)	8.3 (0.9)
Serum phosphate (mg)	4.6 (1.1)
Serum uric acid (mg)	7.9 (2.9)
Serum protein (gm)	5.8 (1.2)
Blood glucose (mg)	98.1 (41.8)
Serum bilirubin (mg)	0.9 (0.1)
ALT (I.U)	28.3 (18.2)
AST (I.U)	35.2 (16.9)
Degree of anemia	
Normal	7 (13.7)
Mild	3 (5.9)
Moderate	16 (31.4)
Severe	25 (49.0)
Mode of delivery, n (%)	
Vaginal	32 (62.7)
LSCS	19 (37.2)
Urinary output, n (%)	
Normal	22 (43.2)
Oliguria	17 (33.3)
Anuria	12 (23.5)
Associated complications, n (%)	
DIC	8 (15.7)
ICU admission	18 (33.3)
Mode of treatment, n (%)	
Hemodialysis	14 (27.5)
Conservative	37 (72.5)
Perinatal outcome	
Full term	19 (37.3)
Preterm	8 (15.7)
IUGR	2 (3.9)
IUD	20 (39.2)
Neonatal death	2 (3.9)

Data shown as mean (SD) unless otherwise specified. ALT=alanine transaminase; AST=aspartate aminotransferase; DIC=disseminated intravascular coagulation; ICU=intensive care unit; IUD=intra-uterine death; IUGR=intra-uterine growth retardation.

TABLE 1. Baseline characteristics

quiring or not requiring dialysis, was the primary outcome. Obstetrical outcome, including mode of delivery, complications of pregnancy such as antepartum or intrapartum or postpartum, intensive care unit (ICU) admission, and maternal death, were the secondary outcomes.

Statistical analysis

Statistical analysis was performed using Statistical Package for the Social Sciences (SPSS) version 20.0. Qualitative data were presented as n (%) and quantitative data as mean [standard deviation (SD)]. The change in continuous variables was assessed by Friedmen test, followed by multiple comparisons using Wilcoxon sign rank test with Bonferroni correction. Between-group comparison of continuous variables was done by Wilcoxon rank sum test and Kruskal Wallis test, followed by multiple comparisons using Dunn test with Bonferroni correction. Categorical variables were compared by Fisher exact test. In all analysis, a $P<0.05$ was considered as statistically significant. ❑

RESULTS

A total of 51 pregnant patients with AKI were included in this study. Subjects' average age was 25.9 years, ranging from 20 to 40 years. Out of the 51 patients, 49.0% had severe anemia and 41.2% were primigravida. Mean parity was 2.2 (1.2) and mean duration of gestation 34.8 (4.1) weeks. Thirty-two (62.7%) patients delivered vaginally and 19 (37.2%) underwent a cesarean section. At presentation, 23.5% of patients had anuria and 33.3% oliguria. An adverse event of DIC was observed in eight patients, whereas 18 (33.3%) were sick, required mechanical ventilator support and admission to ICU. Dialysis was required in 14 (27.5%) cases, while the remaining patients were managed conservatively. The incidence rate of intrauterine fetal death was 20/19 live births and neonatal death was observed in two cases. Out of the 51 patients, eight were premature and 19 mature. During hospitalization, pulmonary edema was observed in six patients and bilateral pleural effusion in one patient, and all seven patients were managed by hemodialysis. One (1.9%) patient had right lower lobe consolidation. Table 1 displays the baseline characteristics of enrolled patients.

Statically, no significant differences were observed in age, parity number, and gestation period among study participants. There was a statistically significant ($P=0.001$) difference in ICU admission with mechanical ventilator support among deceased patients in comparison with normal patients and those with CKD. The associated complications were observed among all patients. Disseminated intravascular coagulation was more frequent among deceased patients (41.7%) than normal ones and patients with CKD (9.1 and 16.6%, respectively). The peaked (maximum) value of blood urea was 62.0 (28.0-218.0) mg/dL in patients with normal renal function,178.5 (155.0-230.0) mg/dL in those

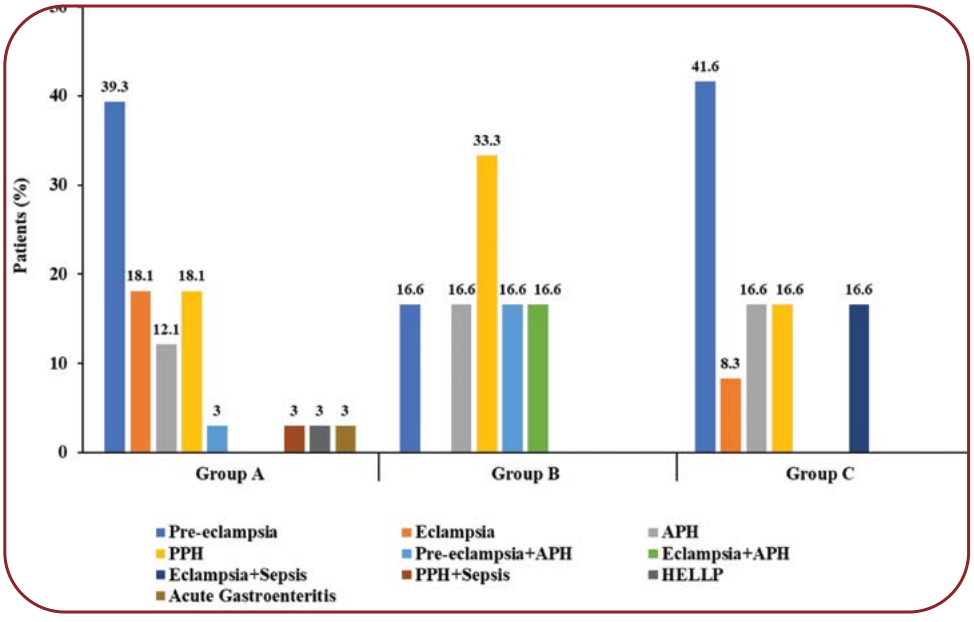

FIGURE 1. Etiology of pregnancy related acute kidney injury in recovered, CKD patients, and expired patients
(Group A: patients in whom renal function recovered to normal; Group B: patients who progressed to CKD; Group C: patients who expired after admission; APH=antepartum hemorrhage; PPH=postpartum hemorrhage; HELLP=hemolysis, elevated liver enzymes, and low platelet count)

TABLE 2. Main characteristics of the groups: demographic and clinical data

Parameter	Normal patients (A) (n=33)	CKD patients (B) (n=6)	Deceased patients (C) (n=12)	P value
Age (years)	24.0 (20.0-35.0)	23.5 (20.0-40.0)	24.5 (20.0-32.0)	0.633 0.697[a], 0.937[b], 0.509[c]
Parity number	2.0 (1.0-5.0)	2.0 (1.0-7.0)	2.0 (1.0-4.0	0.887 0.945[a], 1.000[b, c]
Gestation period (weeks)	36.0 (24.0-40.0)	36.5 (31.0-38.0)	36.5 (30.0-39.0)	0.885 1.000[a, c], 0.939[b]
SBP (mm Hg)	146.0 (82.0-210.0)	114.0 (70.0-200.0)	153.0 (90.0-180.0)	0.573 0.459[a], 1.000[b], 0.523[c]
DBP (mm Hg)	98.0 (0-160.0)	82.0 (0-120.0)	100.0 (0-160.0)	0.799 0.775[a], 1.000[b, c]
Hb (gm%)	8.1 (3.0-15.7)	5.7 (3.7-9.7)	6.1 (3.1-12.3)	0.178 0.231[a], 0.219[b], 1.000[c]
TLC (10^3/cmm)	13.5 (3.0-30.0)	13.5 (78.0-23.0)	19.0 (85.0-38000.0)	0.159 1.000[a], 0.087[b], 0.365[c]
Platelets (10^4/cmm)	13.0 (2.0-70.0)	12.0 (7.0-17.5)	10.0 (2.0-53.0)	0.582 1.000[a, c], 0.451[b]
Blood urea (mg/dL)	49.0 (13.0-119.0)	91.5 (21.0-210.0)	51.0 (23.0-459.0)	0.137 0.070[a], 0.868[b], 0.247[c]
Max. value	62.0 (28.0-218.0)	178.5 (155.0-230.0)	120.0 (57.0-220.0)	0.001 <0.001[a], 0.003[b], 0.376[c]
Serum creatinine (mg/dL)	1.5 (0.5-5.8)	3.8 (0.8-6.8)	2.21 (0.7-12.5)	0.706 0.066[a], 0.198[b], 0.663[c]
Max Value	1.4 (0.9-7.9)	7.1 (4.8-11.5)	3.8 (1.5-8.0)	<0.001 <0.001[a, b], 0.317[c]
Serum sodium (mEq/L)	136.0 (119.0-148.0)	136.5 (121.0-142.0)	133.5 (127.0-153.0)	0.955 1.000[a, b, c]
Serum potassium (mEq/L)	4.4 (3.2-6.1)	5.0 (4.6-5.6)	4.7 (3.2-6.0)	0.102 0.074[a], 0.337[b], 0.532[c]
Serum calcium (mg/dL)	8.5(6.5-10.4)	8.1 (6.5-10.1)	8.3 (6.7-9.7)	0.571 1.000[a, c], 0.451[b]
Serum phosphate (mg/dL)	4.5 (1.3-7.2)	4.7 (4.0-6.8)	4.5 (3.9-6.6)	0.492 0.595[a], 0.483[b], 1.000[c]
Serum uric acid (mg/dL)	6.8 (3.0-15.2)	8.5 (7.0-11.1)	6.9 (4.5-12.4)	0.411 0.299[a], 1.000[b], 0.342[c]
Serum protein (gm%)	5.4 (3.9-9.2)	5.5 (3.5-6.8)	6.6 (4.7-7.3)	0.071 1.000[a], 0.039[b], 0.162[c]
Blood glucose (mg/dL)	81.0 (44.0-162.0)	110.0 (61.0-182.0)	107.0 (56.0-282.0)	0.013 0.105[a], 0.012[b], 1.000[c]
Serum bilirubin (mg/dL)	0.9 (0.6-1.4)	0.9 (0.8-1.2)	1.0 (0.8-1.1)	0.012 0.498[a], 0.005[b], 0.386[c]
SGOT (I.U.)	20.0 (16.0-134.0)	31.0 (20.0-40.0)	33.5 (20.0-70.0)	<0.001 0.049[a], <0.001[b], 0.729[c]
SGPT (I.U.)	26.0 (21.0-122.0)	41.5 (22.0-54.0)	44.0 (27.0-63.0)	<0.001 0.074[a], <0.001[b], 0.732[c]
Mode of delivery, n (%) Vaginal LSCS	 21 (63.3) 12 (36.4)	 4 (66.7) 2 (33.3)	 7 (58.3) 5 (41.7)	 0.912
Urinary output, n (%) Oliguria	 16 (48.5)	 5 (83.3)	 8 (66.6)	 0.228
Associated complications, n (%) DIC ICU admission	 3 (9.1) 6 (18.2)	 1 (16.6) 1 (16.6)	 5 (41.7) 11 (91.7)	 0.037 0.001

Data shown as median (range) unless otherwise specified.
[a], A *vs.* B; [b], A *vs.* C; [c], B *vs.* C; DBP=diastolic blood pressure; DIC=disseminated intravascular coagulation; Hb=hemoglobin; ICU=intensive care unit; LSCS=lower segment caesarean section; SBP=systolic blood pressure; SGOT=serum glutamic oxaloacetic transaminase; SGPT=serum glutamic pyruvic transaminase; TLC=total leukocyte count.

with CKD and 120.0 (57.0-220.0) mg/dL in deceased patients (P=0.001). The peak serum creatinine during AKI was significantly different among all patients (P<0.001). The median blood glucose (mg/dL) was 81.0 in normal patients, 110.0 in CKD patients and 107.0 in deceased patients (P=0.013). All patients showed a significant increase in the median values of serum glutamic oxaloacetic transaminase (SGOT) and serum glutamic pyruvic transaminase levels (SGPT); P<0.001 (Table 2). The etiology of PR-AKI in recovered patients, those with CKD and deceased patients is shown in Figure 1.

There was a marked improvement in the renal outcome, with 33 patients having complete renal recovery, while six patients developed CKD during the three-month follow-up period. A significantly decreasing trend in blood urea was observed in recovered patients (P<0.001) at all points of measurement. Blood levels of uric acid

TABLE 3. Comparison of histological, renal, and biochemical parameters over three months in recovered and CKD patients

Parameter	Admission (a)		Discharge (b)		One month (c)		Three months (d)		P value	
	Recovered	CKD	Recovered	CKD	Recovered	CKD	Recovered	CKD	Group A	Group B
Hb (gm%)	8.1 (3.0-15.7)	5.7 (3.7-9.7)	8.9 (6.7-12.6)	8.6 (7.8-9.3)	9.0 (8.0-13.8)	8.4 (8.0-9.3)	8.8 (8.0-12.7)	8.7 (8.0-9.3)	0.127 0.170[a], 0.129[b], 0.196[c]	0.115 0.075[a, b, c]
Difference	0.155		0.483		0.868		0.254			
TLC (10^3/cmm)	13.5 (3.0-30.0)	13.5 (7.8-23.0)	10.5 (5.0-21.0)	10.1 (5.6-18.0)	9.5 (6.8-21)	9.6 (5.6-11.5)	8.8 (7.0-12.5)	9.1 (7.0-12.5)	<0.001 0.003[a], <0.001[b, c]	<0.001 0.046[a], 0.043[b], 0.027[c]
Difference	0.876		0.080		0.066		0.362			
Platelets (10^3/cmm)	130.0 (20.0-700.0)	120.0 (700.0-175.0)	210.0 (35.0-500.0)	175.0 (120.0-260.0)	255.0 (190.0-350.0)	240.0 (120.0-250.0)	250.0 (190.0-360.0)	205.0 (174.0-280.0)	<0.001 0.001[a, b, c]	0.015 0.017[a], 0.046[b], 0.028[c]
Difference	0.668		0.282		0.067		0.051			
Blood urea (mg/dL)	49.0 (13.0-119.0)	91.5 (21.0-210.0)	30.0 (14.0-85.0)	92.5 (63.0-59.0)	24.0 (14.0-48.0)	80.0 (48-102)	20.0 (14.0-45.0)	57.0 (26.0-84.0)	<0.001 <0.001[a, b, c]	0.232 0.917[a], 0.249[b], 0.141[c]
Difference	0.037		<0.001		<0.001		<0.001			
Serum creatinine (mg/dL)	1.3 (0.5-5.8)	3.8 (0.8-6.8)	0.8 (0.4-2.1)	4.25 (1.7-4.9)	0.7 (0.4-1.2)	2.9 (1.5-6.4)	0.6 (0.4-1.0)	2.7 (1.2-6.4)	<0.001 0.001[a, b, c]	0.275 0.463[a], 0.913[b], 0.833[c]
Difference	0.053		<0.001		<0.001		<0.001			
Serum sodium (mEq/L)	136.0 (119.0-148.0)	136.5 (121.0-142.0)	136.0 (119.0-148.0)	139.0 (131.0-140.0)	138.0 (130.0-149.0)	139.5 (36.0-142.0)	136.0 (132.0-142.0)	140.0 (135.0-145.0)	0.253 0.370[a], 0.610[b], 0.127[c]	0.267 0.400[a], 0.750[b], 0.930[c]
Difference	0.891		0.917		0.277		0.184			
Serum potassium (mEq/L)	4.4 (3.2-6.1)	5.0 (4.6-5.6)	3.9 (3.0-4.6)	4.2 (3.2-5.1)	3.8 (3.5-5.0)	4.8 (3.5-5.6)	4.1 (3.5-4.6)	4.4 (3.5-5.2)	0.010 0.001[a], 0.003[b], 0.044[c]	0.267 0.046[a], 0.273[b], 0.080[c]
Difference	0.0389		0.281		0.021		0.317			
Serum calcium (mg/dL)	8.5 (6.5-10.4)	8.1 (6.5-10.1)	8.9 (7.5-9.8)	8.5 (6.8-10)	8.9 (7.8-10.7)	8.4 (7.8-10.2)	8.9 (7.6-10.0)	8.4 (8.0-9.6)	0.097 0.007[a], 0.092[b], 0.090[c]	0.665 0.752[a], 0.345[b], 0.463[c]
Difference	0.739		0.274		0.215		0.216			
Serum phosphate (mg/dL)	4.5 (1.3-7.2)	4.7 (4.0-6.8)	4.2 (2.4-7.2)	4.2 (2.4-7.2)	4.0 (2.4-5.5)	4.9 (4.3-5.5)	4.1 (3.0-4.8)	4.7 (3.7-5.9)	0.283 0.360[a], 0.077[b], 0.070[c]	0.580 0.400[a], 1.000[b], 0.753[c]
Difference	0.422		0.291		0.003		0.045			
Serum uric acid (mg/dL)	6.8 (3.0-15.2)	8.5 (7.0-11.1)	5.0 (3.1-13.0)	6.6 (4.6-7.9)	4.2 (3.1-6.3)	6.1 (4.6-7.9)	4.0 (3.0-5.8)	5.5 (3.9-7.4)	<0.001 <0.001[a, b, c]	0.004 0.028[a], 0.075[b], 0.027[c]
Difference	0.133		0.032		0.006		0.008			
Serum protein (gm%)	5.4 (3.9-9.2)	5.5 (3.5-6.8)	6.7 (4.5-8.2)	6.9 (5.8-7.8)	7.0 (6.1-8.6)	7.2 (4.6-8.0)	7.1 (6.5-8.2)	7.5 (7.1-8.2)	<0.001 <0.001[a, b, c]	0.006 0.028[a], 0.075[b], 0.027[c]
Difference	0.969		0.635		0.852		0.134			
Blood glucose (mg/dL)	81.0 (44.0-162.0)	110.0 (61.0-182.0)	88.0 (64.0-132.0)	101.0 (82.0-122.0)	88.0 (68.0-108.0)	100.0 (92.0-122.0)	88.0 (54.0-98.0)	94.0 (68.0-100.0)	0.915 0.499[a], 0.658[b], 0.804[c]	0.104 0.600[a], 0.917[b], 0.249[c]
Difference	0.083		0.067		0.059		0.184			
Serum bilirubin (mg/dL)	0.9 (0.6-1.4)	0.9 (0.8-1.1)	0.8 (0.7-1.0)	0.8 (0.7-0.9)	0.8 (0.7-0.8)	0.8 (0.7-0.9)	0.8 (0.7-0.9)	0.9 (0.7-1.0)	0.867 0.055[a], 0.290[b], 0.129[c]	0.155 0.125[a], 0.142[b], 0.174[c]
Difference	0.265		0.422		0.126		0.763			
SGOT (I.U.)	26.0 (21.0-122.0)	41.5 (22.0-54.0)	25.0 (18.0-45.0)	29.0 (24.0-43.0)	24.5 (18.0-32.0)	25.5 (21.0-32.0)	24.0 (20.0-29.0)	26.0 (18.0-29.0)	0.027 0.067[a], 0.009[b], 0.007[c]	0.029 0.046[a, c], 0.028[b]
Difference	0.059		0.059		0.508		0.358			
SGPT (I.U.)	20.0 (16.0-134.0)	31.0 (20.0-40.0)	20.0 (14.0-34.0)	22.5 (19.0-30.0)	20.0 (16.0-36.0)	20.5 (18.0-24.0)	19.5 (16.0-36.0)	21.0 (17.0-23.0)	0.254 0.157[a], 0.096[b], 0.055[c]	0.401 0.141[a], 0.080[b, c]
Difference	0.051		0.296		0.263		0.143			

Data shown as median (range)

[a], a vs b; [b], a vs c; [c], a vs d; Hb=hemoglobin; SGOT=serum glutamic oxaloacetic transaminase; SGPT=serum glutamic pyruvic transaminase; TLC=total leukocyte count.

were higher in both groups at the time of admission. Among recovered patients there was a statistically significant change in serum potassium (P=0.010) and creatinine levels (P<0.001) during the follow-up period. Serum protein levels were low at the time of admission but increased to normal on follow-up. Liver enzymes, including SGOT and SGPT, were high in all patients at the time of admission, but decreased to normal on follow-up (Table 3). ❑

DISCUSSION

Acute kidney injury in pregnancy commonly occurs due to obstetrical complications such as ante-partum or postpartum hemorrhage, intrauterine fetal death, septic abortion, abruptio placentae and puerperal sepsis. The renal physiological alterations arising during pregnancy need to be assessed appropriately for a precise diagnosis and management of AKI. The present study prospectively evaluated 51 patients for three months after admission to the institute.

The incidence of PR-AKI was more frequent in the third trimester (55%) (18), and a similar result was observed in the present study (50.9%), but a recent study showed conflicting results, where the majority of subjects were in the postpartum period (60%), followed by the third trimester (1). The major causes of PR-AKI in the third trimester included postpartum hemorrhage and pregnancy-induced hypertension. Furthermore, in concordance with this result, some studies have shown that hypertensive disorders, hemolysis, elevated liver enzymes, and low platelet count (HELLP) (19), thrombotic microangiopathies, sepsis, septic shock, postpartum hemorrhages were most common causes of PR-AKI (20-22).

The main etiologies of PR-AKI are different in developing and developed countries. Pre-eclampsia and postpartum hemorrhage (4, 7, 23) were the notable causes of obstetric AKI, similarly to the present study. Multiple studies on etiological factors in PR-AKI reported sepsis as the most common cause of AKI (1, 3, 21, 24). It is found to be associated with improper handling, poor hygienic environment, non-sterilized and/or inappropriate intervention, traditional birth assistant (25). Pre-eclampsia and postpartum hemorrhage were the leading causes of PR-AKI, comparable with the findings of Mir *et al* showing that postpartum hemorrhage was contributed to 25% and 14% incidence of pre-eclampsia (22). This suggests the need for improving the quality of antenatal and perinatal care.

Increasing antenatal and postnatal care and improved medical services has led to the dwindling incidences of maternal mortality related to PR-AKI in developing countries. The mortality rate has decreased from 20% in 1980 to 4% (China) and 5.8% (India) (3, 4). However, the rate of PR-AKI related mortality is still high in developing countries like India. The present study reported 23.5% of maternal mortality, which is in agreement with findings reported by previous studies (1, 26). Prakash J *et al* noted a high perinatal mortality (~45%) throughout three decades (3). A study from developed country reported a PR-AKI related fetal mortality of only 5.5% (27). Previous literature reported the highest mortality rate of 50%. Similarly, the present study also revealed a high rate of neonatal death (43.1%) (28). The higher rate of fetal mortality may be associated with poor antenatal care.

In terms of renal outcomes, Albino *et al* noted a complete recovery of renal function in 25.7% of patients in their randomized clinical trial (29), whereas the present study reported a complete recovery of renal function in 64.7% of patients. Oliguria is attributed to poor renal perfusion and it may be a consequence of hypovolemia and pain and triggers the sympathetic nervous system (30). Analysis of data from FINNAKI study (31) reported oliguria in 92% of patients, in contrast to the result of the present study (33.3%).

In the present study, the mean peak values of blood urea and serum creatinine were 120.0 and 3.8 mg/dL, respectively in CKD patients, and 178.5 and 7.1 mg/dL, respectively in deceased patients. Similarly, Krishna *et al* observed mean serum creatinine levels of 7.7 mg/dL in deceased patients and 8.3 mg/dL in those who survived (26). The present study has reported a post-discharge follow-up of parameters in PR-AKI patients.

The strengths of our study include the availability of clinical characteristics of PR-AKI and feasible length of follow-up. Limitations include the small sample size, lack of information about monitoring in ICU, and impact of conservative treatment measures. ❑

CONCLUSION

Our study indicates that pre-eclampsia, eclampsia, antepartum and postpartum hemorrhage were common causes of AKI in late pregnancy. All of them are treatable and preventable etiologies. About one third of these patients required ICU admission, mechanical ventilation and renal replacement therapy. Complicated cases were associated with poor obstetric outcome as well as increased mortality, and adverse health outcomes, including worse-

ning of kidney function leading to chronic kidney disease. The management of PR-AKI is a clinical challenge that requires proper evaluation of potential causative factors in order to facilitate appropriate treatment. Early diagnosis and timely intervention may help to reduce morbidity and mortality associated with PR-AKI. ❑

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