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Incidence, Characteristics and Outcome of Acute Kidney Injury in the Postpartum Period: A Prospective Observational Study from a Tertiary Care Centre in North Bengal, India

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ABSTRACT

Background: Pregnancy-related acute kidney injury (PR-AKI) remains an important cause of maternal morbidity and mortality in low- and middle-income countries. Postpartum AKI accounts for a substantial proportion of PR-AKI, yet contemporary data from eastern India are limited. This study examined the incidence pattern, clinical characteristics, aetiology and outcome of postpartum AKI at a tertiary referral centre.

Methods: A hospital-based prospective observational study was conducted in the Department of Obstetrics & Gynaecology, North Bengal Medical College & Hospital, over 12 months (November 2022–October 2023). Sixty-eight women with postpartum (<12 weeks after delivery) AKI referred to the centre were enrolled after applying defined inclusion and exclusion criteria. Demographic, clinical and laboratory data were recorded on a standard proforma. AKI was staged using KDIGO criteria. Categorical variables were summarised as frequencies and percentages and continuous variables as mean ± standard deviation; analysis was performed using SPSS v21.0.

Results: The mean age was 29.03 ± 5.70 years, with 61.8% of patients aged 26–35 years. Half (50%) had received no antenatal care. Vaginal delivery occurred in 60.3% and caesarean section in 39.7%. Oligo-anuria was universal (100%), followed by oedema (67.6%) and breathlessness (55.9%). At presentation 64.7% were in KDIGO stage III. Puerperal sepsis was the leading cause (41.2%), followed by preeclampsia (33.8%), intrauterine death (22.0%), antepartum haemorrhage (14.7%) and postpartum haemorrhage (11.8%). Dialysis was required in 45.6% of patients. Complete renal recovery occurred in 54.4%, partial recovery in 19.1%, while 14.7% remained dialysis-dependent; maternal mortality was 11.8%.

Conclusion: Postpartum AKI in this setting presents late and at an advanced KDIGO stage, driven predominantly by puerperal sepsis and hypertensive disorders of pregnancy—both largely preventable. Strengthening antenatal coverage, promoting institutional aseptic delivery, and early recognition and aggressive management of sepsis, haemorrhage and hypertension are key to reducing the burden and improving maternal and renal outcomes.

Keywords*: Acute kidney injury; Postpartum; Pregnancy-related AKI; Puerperal sepsis; Preeclampsia; Dialysis; Maternal outcome; KDIGO.*

INTRODUCTION

Pregnancy-related acute kidney injury (PR-AKI) is a heterogeneous disorder that encompasses all causes of acute renal impairment from early pregnancy up to three months postpartum.[1] It is an important obstetric complication associated with significant maternal and fetal morbidity and mortality.[2] Although its incidence has fallen sharply in high-income countries because of improved general and obstetric care, PR-AKI remains a serious public health problem in developing countries, where it is estimated to account for 3–5% of all cases of acute kidney injury.[3,4]

The aetiology and presentation of PR-AKI differ markedly between high- and low-income settings.[5] In resource-limited regions the dominant contributors are infection, nephrotoxins and obstetric and surgical complications.[6] The principal conditions associated with renal injury in the antenatal and postpartum periods are preeclampsia and eclampsia, obstetric haemorrhage, HELLP syndrome and puerperal sepsis.[7] Early-pregnancy AKI is most often pre-renal, secondary to dehydration from hyperemesis gravidarum or to septic shock; in the third trimester and immediate puerperium it is more commonly associated with preeclampsia/eclampsia, antepartum and postpartum haemorrhage, puerperal sepsis, haemolytic uraemic syndrome and HELLP syndrome.[8]

Complete renal recovery is usual when patients are treated appropriately and promptly. However, inadequate initial resuscitation, delayed initiation of antibiotics, prolonged referral intervals and consequent delay in starting dialysis are the main drivers of poor outcomes.[9] The maternal mortality attributable to PR-AKI ranges from 13% to 24% in developing countries, and fetal, neonatal and perinatal mortality are also disproportionately high in this group.[10]
Despite the heavy burden borne by low- and middle-income countries, relatively few studies have characterised the incidence, risk factors and outcomes of postpartum AKI in obstetric populations.[11] A clear understanding of these features is essential for clinicians to anticipate, prevent and manage the condition. The present study was therefore undertaken to determine the incidence pattern, clinical and laboratory characteristics, aetiology and outcome of acute kidney injury occurring in the postpartum period at a tertiary care centre in North Bengal.

Aim and Objectives

Aim. To study the incidence, characteristics and outcome of acute kidney injury in the postpartum period.
Specific objectives: (i) to determine the causes of AKI in the postpartum period; and (ii) to determine the clinical characteristics and outcome of postpartum patients with AKI.

MATERIALS AND METHODS

Study design, setting and duration

This was a hospital-based, prospective, observational study carried out in the Department of Obstetrics & Gynaecology, North Bengal Medical College & Hospital, over a period of 12 months (November 2022 to October 2023). The hospital serves as a tertiary referral centre receiving obstetric patients with renal injury from rural areas and from block primary health centres for further management.

Study population and sample size

The study enrolled women presenting with postpartum (<12 weeks after delivery) AKI referred to the centre during the study period. Using an expected PR-AKI incidence of 4.27% reported by Mir et al. (2017)[12], a 95% confidence level and a relative precision of 5%, the calculated sample size was approximately 68. Sixty-eight consecutive eligible patients were therefore included.

Eligibility criteria

Inclusion criteria: women giving valid informed consent and presenting with AKI within 12 weeks of delivery.
Exclusion criteria: pre-existing renal disease before pregnancy, chronic hypertension antedating pregnancy, renal calculi, renal scarring, small kidney on ultrasonography, and unwillingness to participate.

Data collection

After institutional ethics committee clearance and written informed consent, demographic, clinical and laboratory data were obtained by interview, clinical examination and review of medical records, and entered on a standard proforma. Recorded variables included age, parity, antenatal care status, mode of delivery, presenting symptoms and signs, and the most abnormal values within the first 24 hours of admission. Antecedent obstetric conditions documented included gestational hypertension, preeclampsia, eclampsia, HELLP syndrome, postpartum haemorrhage, infection and acute fatty liver of pregnancy. Laboratory work-up comprised complete blood count, random blood sugar, liver function tests, blood urea, serum creatinine, coagulation profile (PT-INR, APTT), viral markers (HIV, HBsAg, HCV) and routine urine examination. AKI was staged according to the Kidney Disease: Improving Global Outcomes (KDIGO) criteria.

Statistical analysis
Categorical variables were expressed as numbers and percentages and continuous variables as mean ± standard deviation (SD). The unpaired Student t-test was used for parametric data, the Mann–Whitney U test for non-parametric data, and the chi-square test for categorical data, as appropriate. A p-value < 0.05 was considered statistically significant. All analyses were performed using the Statistical Package for the Social Sciences (SPSS) version 21.0 (IBM Corp., Chicago, IL, USA).

RESULTS
A total of 68 women with postpartum AKI were studied. The key demographic, clinical, laboratory and outcome findings are summarised below.

Demographic profile
Patients ranged in age from 18 to 42 years, with the maximum incidence in the 26–35-year band (61.8%) and a mean age of 29.03 ± 5.70 years (Table 1). Regarding antenatal care, half of the women (50%) had received no antenatal care, 30.9% had been seen only by a traditional birth attendant, and just 19.1% had received adequate care from a gynaecologist (Table 2). Multiparous women constituted 57.4% of the cohort and primiparous women 42.6% (Table 3). Vaginal delivery had occurred in 60.3% and lower-segment caesarean section in 39.7% (Table 4).

Table 1. Age distribution of patients (n = 68).

Age group	Frequency	Percentage (%)
18–25 years	19	27.9
26–30 years	22	32.4
31–35 years	20	29.4
>35 years	7	10.3
Total	**68**	**100**

Mean age: 29.03 ± 5.70 years.

Table 2. Antenatal care (ANC) status (n = 68).

ANC status	Frequency	Percentage (%)
Regular	13	19.1
Irregular (TBA only)	21	30.9
None	34	50
Total	**68**	**100**

Table 3. Parity distribution (n = 68).

Parity	Frequency	Percentage (%)
Primiparous	29	42.6
Multiparous	39	57.4
Total	**68**	**100**

Table 4. Mode of delivery (n = 68).

Mode of delivery	Frequency	Percentage (%)
Vaginal delivery	41	60.3
Lower-segment caesarean section	27	39.7
Total	**68**	**100**

Clinical presentation
Oligo-anuria was the universal presenting feature (100%), followed by oedema (67.6%), breathlessness (55.9%), hypotension (48.5%), fever (42.6%), hypertension (35.3%), encephalopathy (23.5%) and abdominal pain (19.1%) (Table 5).

Table 5. Clinical presentation at admission (n = 68; features not mutually exclusive).

Clinical feature	Frequency	Percentage (%)
Oligo-anuria	68	100
Oedema	46	67.6
Breathlessness	38	55.9
Hypotension	33	48.5
Fever	29	42.6
Hypertension	24	35.3
Encephalopathy	16	23.5
Abdominal pain	13	19.1

Laboratory findings

Most patients were anaemic, with a mean haemoglobin of 9.94 ± 1.89 g/dL. The mean blood urea and serum creatinine were 113.65 ± 35.87 mg/dL and 5.95 ± 1.43 mg/dL, respectively. The mean total leucocyte count was 19,439.71 ± 4,407.06 cells/mm³, in keeping with the high proportion of septic cases, and the mean platelet count was 0.92 ± 0.27 × 10^6/mm³ (Table 6).

Table 6. Laboratory investigations at admission (n = 68).

Investigation	Mean	± SD
Haemoglobin (g/dL)	9.94	1.89
Blood urea (mg/dL)	113.65	35.87
Serum creatinine (mg/dL)	5.95	1.43
WBC count (cells/mm³)	19,439.71	4,407.06
Platelet count (×10^6/mm³)	0.92	0.27

Severity, hospital stay and management

Most patients had a prolonged admission: 47.0% stayed for more than 10 days and 41.2% for 7–10 days, with a mean hospital stay of 10.59 ± 3.49 days (Table 7). At presentation, the majority were already at an advanced stage of renal injury—64.7% in KDIGO stage III, 25.0% in stage II and 10.3% in stage I (Table 8). Dialysis-requiring AKI was seen in 31 patients (45.6%); of these, 21 (30.9%) received intermittent haemodialysis and 10 (14.7%) peritoneal dialysis, while 37 (54.4%) were managed conservatively (Table 10).

Table 7. Duration of hospital stay (n = 68).

Duration	Frequency	Percentage (%)
<7 days	8	11.8
7–10 days	28	41.2
>10 days	32	47
Total	**68**	**100**

Mean duration: 10.59 ± 3.49 days.

Table 8. KDIGO stage on admission (n = 68).

KDIGO stage	Frequency	Percentage (%)
Stage I	7	10.3
Stage II	17	25
Stage III	44	64.7

KDIGO stage	Frequency	Percentage (%)
Total	**68**	**100**

Aetiology of postpartum AKI

Puerperal sepsis was the single most common contributing factor, present in 28 patients (41.2%), followed by preeclampsia in 23 (33.8%), intrauterine death in 15 (22.0%), antepartum haemorrhage in 10 (14.7%), postpartum haemorrhage in 8 (11.8%), HELLP syndrome in 4 (5.9%), acute fatty liver of pregnancy in 4 (5.9%) and thrombotic microangiopathy in 2 (2.9%). As many patients had more than one contributing factor, the percentages exceed 100% (Table 9, Figure 1).

Table 9. Aetiological factors contributing to postpartum AKI (n = 68; categories not mutually exclusive).

Cause	Frequency	Percentage (%)
Puerperal sepsis	28	41.2
Preeclampsia	23	33.8
Intrauterine death	15	22
Antepartum haemorrhage	10	14.7
Postpartum haemorrhage	8	11.8
HELLP syndrome	4	5.9
Acute fatty liver of pregnancy	4	5.9
Thrombotic microangiopathy	2	2.9

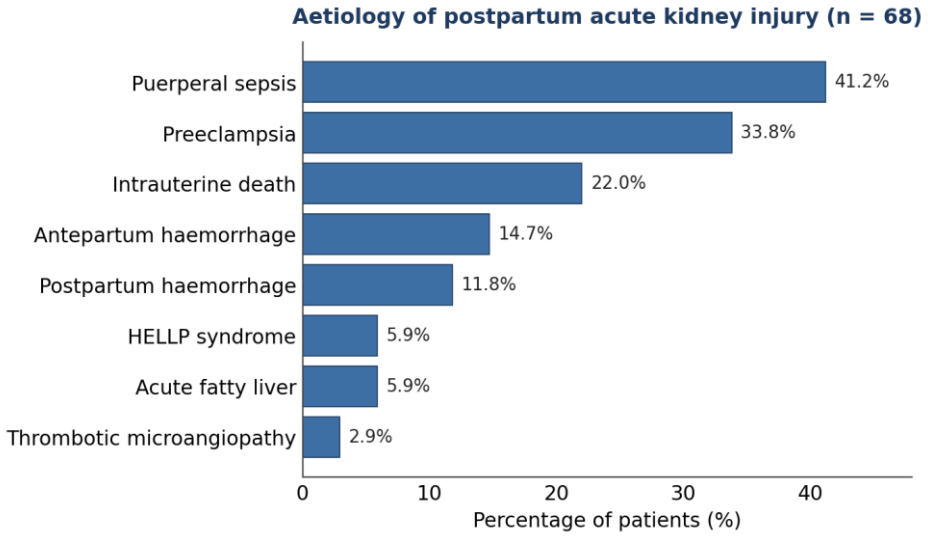

Figure 1. Aetiology of postpartum acute kidney injury (n = 68). Percentages exceed 100% because several patients had more than one contributing factor.

Table 10. Treatment given (n = 68).

Treatment	Frequency	Percentage (%)
Intermittent haemodialysis	21	30.9
Peritoneal dialysis	10	14.7
Conservative treatment	37	54.4
Total	**68**	**100**

Outcome

Most patients made a complete renal recovery (54.4%), 19.1% recovered partially and 14.7% remained dialysis-dependent. Maternal mortality was 11.8% (Table 11, Figure 2).

Table 11. Maternal renal and survival outcome (n = 68).

Outcome	Frequency	Percentage (%)
Complete recovery	37	54.4
Partial recovery	13	19.1
Dialysis-dependent	10	14.7
Death	8	11.8
Total	**68**	**100**

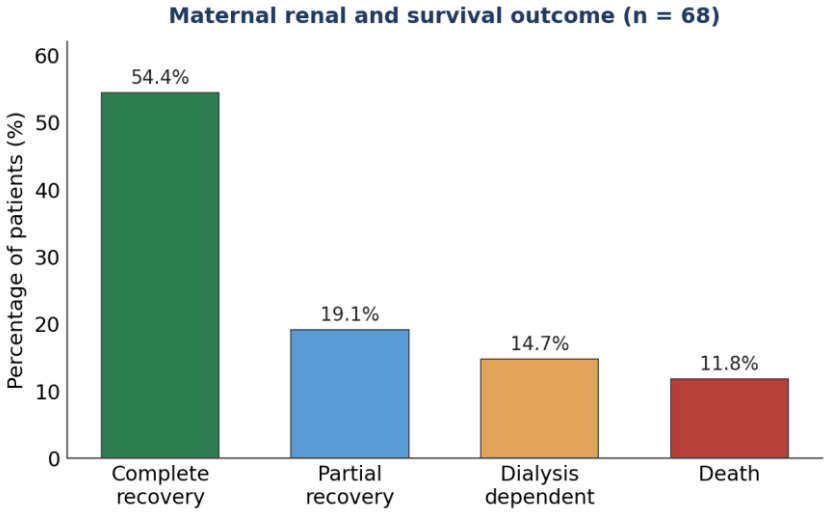

Figure 2. Maternal renal and survival outcome of postpartum AKI (n = 68).

DISCUSSION

Acute kidney injury is a clinical syndrome characterised by an abrupt decline in glomerular filtration rate with accumulation of nitrogenous waste products. Although a rare complication of pregnancy in high-income settings—where incidence has fallen to roughly one in 20,000 births[13]—pregnancy continues to account for 15–20% of AKI in developing countries[4], and postpartum AKI represents 26–70% of all PR-AKI.[14] Its severest, dialysis-requiring form carries appreciable mortality and short-term morbidity and an increased long-term risk of chronic kidney disease. The present single-centre study of 68 referred patients provides a contemporary description of this condition in North Bengal.

The mean age in our cohort (29.03 ± 5.70 years) is consistent with the range of 24–28 years reported across comparable Indian and South Asian series, including Mir et al.[12], Tanwar et al.[15], Parween et al.[16], Bokhari et al.[10] and Mondal et al.[17] The preponderance of patients in the 26–35-year band reflects the age distribution of the local obstetric population rather than a specific age-related risk.

A striking finding was the poor antenatal coverage: only 19.1% of women had received adequate antenatal care, half had received none, and the remainder had been seen only by a traditional birth attendant. This pattern is almost identical to that reported by Bokhari et al. (19% adequate care, 48% none)[10] and underscores the central role of inadequate antenatal supervision in the genesis of severe, late-presenting postpartum AKI. Multiparity predominated (57.4%), in line with most prior reports.[17] Vaginal delivery accounted for 60.3% of cases, closely matching the 60–61% reported by Mir et al.[12] and Afreen et al.[18], although centres performing predominantly caesarean deliveries have reported the reverse distribution.[17] The clinical spectrum—universal oligo-anuria followed by oedema, breathlessness, hypotension, fever, hypertension and encephalopathy—mirrors that described by Mir et al.[12] and Mondal et al.[17] almost feature for feature, suggesting a reproducible presentation of severe postpartum AKI across the region. Consistent with this severity, the mean serum creatinine was high (5.95 mg/dL) and most patients were anaemic, while the elevated mean leucocyte count reflects the large infective contribution. Importantly, 64.7% of patients were already in KDIGO stage III at presentation, again closely matching Mir et al. (64% stage III)[12] and reflecting delayed referral.

Puerperal sepsis was the leading aetiological factor (41.2%), followed by hypertensive disorders (preeclampsia 33.8%; HELLP 5.9%) and obstetric haemorrhage (antepartum 14.7%, postpartum 11.8%). This hierarchy is concordant with multiple Indian series in which sepsis is the commonest cause of postpartum AKI[4,19,20], and contrasts with high-income settings where preeclampsia, PPH and HELLP syndrome predominate.[21] The kidney is frequently injured in sepsis through haemodynamic compromise, endothelial dysfunction, intrarenal inflammation and microthrombosis; in our population,

anaemia, prolonged labour, repeated unsterile vaginal examinations and prolonged rupture of membranes are the usual antecedents. These observations reinforce that the dominant causes here are largely preventable.

Dialysis was required in 45.6% of patients, comparable to the 46–47% reported by Mir et al.[12] and Gullipalli et al.[20], though higher dialysis rates (70–100%) have been described in more severely affected referral cohorts.[16,17] Renal outcomes were favourable in the majority: complete recovery in 54.4% and partial recovery in 19.1%, figures that align with the complete-recovery rates of approximately 51–54% reported by Goplani et al. and Kumar et al.[22,23] Maternal mortality in our series was 11.8%, similar to Khalil et al. (15%)[24] and Afreen et al. (10.4%)[18], and considerably lower than the 28–33% reported by Choudhary et al. and Tanwar et al.[25,15]; sepsis and coagulation abnormalities were major contributors to mortality in earlier reports.[26] The comparatively low mortality in the present series is plausibly attributable to earlier dialysis access and improved supportive care.

The principal limitations of this study are its single-centre design, the modest sample size and the referral-based population, which together select for more severe disease and limit generalisability; longer-term renal follow-up beyond the index admission was also not captured. Nonetheless, the consistency of our findings with contemporaneous regional data lends them external validity and highlights actionable priorities.

Taken together, the results indicate that postpartum AKI in this setting is largely a disease of preventable obstetric complications presenting late. Strengthening antenatal coverage, promoting institutional delivery with strict asepsis, and ensuring early recognition and aggressive management of sepsis, haemorrhage and hypertensive disorders—coupled with timely referral and dialysis—offer the clearest route to reducing both the incidence and the associated maternal morbidity and mortality.

CONCLUSION

Acute kidney injury in the postpartum period is a serious, potentially preventable obstetric complication that endangers both mother and child. In this prospective cohort, puerperal sepsis and hypertensive disorders of pregnancy were the predominant causes, most patients presented late at KDIGO stage III, and nearly half required dialysis. While complete or partial renal recovery was achieved in the majority, maternal mortality remained substantial at 11.8%. Monitoring of high-risk women, improved antenatal coverage, aseptic institutional delivery, and early detection and aggressive management of hypertension, haemorrhage and sepsis can reduce the incidence of postpartum AKI and its associated maternal morbidity and mortality in developing-country settings.

Declarations

Ethics approval: The study was approved by the Institutional Ethics Committee of North Bengal Medical College & Hospital, and written informed consent was obtained from all participants.
Conflicts of interest: The authors declare no conflicts of interest.
Funding: None.
Author contributions: SB conceived and conducted the study, collected and analysed the data, and drafted the manuscript. GM supervised the study, provided scientific and methodological guidance, and critically revised the manuscript. Both authors approved the final version.

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