

ACUTE KIDNEY INJURY IN PREGNANT WOMEN: ETIOLOGIES, MANAGEMENT STRATEGIES, AND MATERNAL-FETAL OUTCOMES IN A TERTIARY CARE SETTING

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ABSTRACT

Background: Acute kidney injury (AKI) in pregnancy is a life-threatening complication associated with significant maternal and neonatal morbidity and mortality. Predisposing factors include hypertensive disorders (e.g., preeclampsia), hemorrhage (notably postpartum hemorrhage), and sepsis. Early diagnosis and expeditious management—including hemodynamic stabilization and renal replacement therapy—are paramount for improving outcomes.

Methods: A retrospective observational study was performed at the Karnataka Medical College and Research Institute, Hubli, Karnataka, over six months (May 2024–November 2024). Forty-nine pregnant or postpartum women (up to six weeks post-delivery) with AKI were identified from hospital records. Data on demographics, obstetric parameters, etiology of AKI, mode of delivery, renal replacement therapy (dialysis) requirements, and maternal-fetal outcomes were analyzed. Key findings were summarized using bar charts and pie charts to illustrate distributions of age, parity, mode of delivery, etiologies, dialysis requirements (including number of cycles), maternal mortality, causes of death, and trends in serum urea/creatinine.

Results: Hypertensive disorders (40.8%) were the most frequent etiology, followed by postpartum hemorrhage (30.6%) and sepsis (14.3%).

- Dialysis was required in 24 (49.0%) women; among these, 9 succumbed to complications (37.5% mortality in the dialysis subgroup).
- Overall maternal mortality was 28.6% (n=14), primarily attributed to multiorgan dysfunction syndrome, septic shock, and coagulopathy.
- Fetal/neonatal deaths occurred in 24.5% (n=12), often linked to extreme prematurity, low birth weight, or severe maternal compromise.

Conclusion: AKI during pregnancy and the postpartum period remains a serious clinical challenge, especially in resource-constrained settings. Early detection of predisposing conditions, prompt resuscitation, and the availability of renal replacement therapy can substantially improve maternal and neonatal survival. Strengthening antenatal care, timely referral pathways, and critical care infrastructure are crucial strategies to curtail mortality and morbidity.

INTRODUCTION

Acute kidney injury (AKI) in pregnancy is a formidable complication often culminating in severe maternal and fetal morbidity and mortality. Although its overall incidence has declined in many high-income countries, it remains disproportionately high in low- and middle-income regions, including India, where late referrals and inadequate healthcare resources contribute to worse outcomes [1,2].

Multiple mechanisms underlie AKI in pregnancy. Hypertensive disorders (particularly severe preeclampsia, eclampsia, and HELLP syndrome) can precipitate renal hypoperfusion and endothelial dysfunction [3,4]. Hemorrhage—especially postpartum hemorrhage (PPH)—leads to hypovolemia and ischemic renal injury if not rapidly corrected [5]. Infections, such as puerperal sepsis or surgical site infections, trigger systemic inflammation that further exacerbates renal damage [6]. These conditions often coexist, rendering management more complex and outcomes more guarded [7].

The gravity of AKI in pregnancy extends beyond maternal well-being, as fetal complications—ranging from preterm delivery and low birth weight to intrauterine death—are frequent when maternal hemodynamics are severely compromised [8]. Early recognition of high-risk cases and prompt initiation of therapy (including renal replacement, if needed) are cornerstone interventions. With an increasing focus on enhancing obstetric care and critical care collaborations, there is an urgent need to evaluate the specific etiologies, management protocols, and outcomes associated with AKI in pregnancy to inform best practices [9].

In this retrospective study, we aim to elucidate the clinical profile of pregnancy-related AKI in a tertiary care setting, delineate major causative factors, and assess maternal-fetal outcomes. By focusing on graphical representations of data—including age, parity, etiologies, dialysis requirements, and mortality—this report seeks to highlight the pressing need for comprehensive care pathways that can reduce adverse outcomes.

MATERIALS AND METHODS

Study Design and Setting

A retrospective, single-center, observational study was conducted at the Karnataka Medical College and Research Institute, Hubli, Karnataka. The study period spanned six months (May 2024–November 2024).

Inclusion and Exclusion Criteria

- **Inclusion:** Pregnant or postpartum women (up to six weeks post-delivery) diagnosed with AKI, defined by either
 1. a rise in serum creatinine ≥ 0.3 mg/dL within 48 hours, or
 2. a ≥ 1.5 -fold increase in baseline creatinine within seven days.
- **Exclusion:** Women with known pre-pregnancy end-stage renal disease on maintenance dialysis, incomplete medical records, or onset of AKI beyond six weeks postpartum.

Data Collection

Patient records were screened for:

1. Demographic variables: Age, parity, referral status.
2. Obstetric details: Gestational age, mode of delivery.
3. Etiology of AKI: Hypertensive disorders, hemorrhage, sepsis, etc.
4. Interventions: Conservative management, dialysis cycles, surgical procedures (e.g., hysterectomy, bilateral internal iliac artery ligation).
5. Laboratory values: Serum urea, creatinine (initial and peak values), and other pertinent parameters.
6. Maternal-fetal outcomes: Maternal mortality with causes of death, fetal/neonatal survival, and renal recovery status at discharge.

Data were de-identified, coded, and entered into a secure electronic database for analysis.

Ethical Considerations

Institutional guidelines for retrospective reviews were followed, ensuring patient anonymity and data confidentiality. Formal approval was obtained from the local institutional review board.

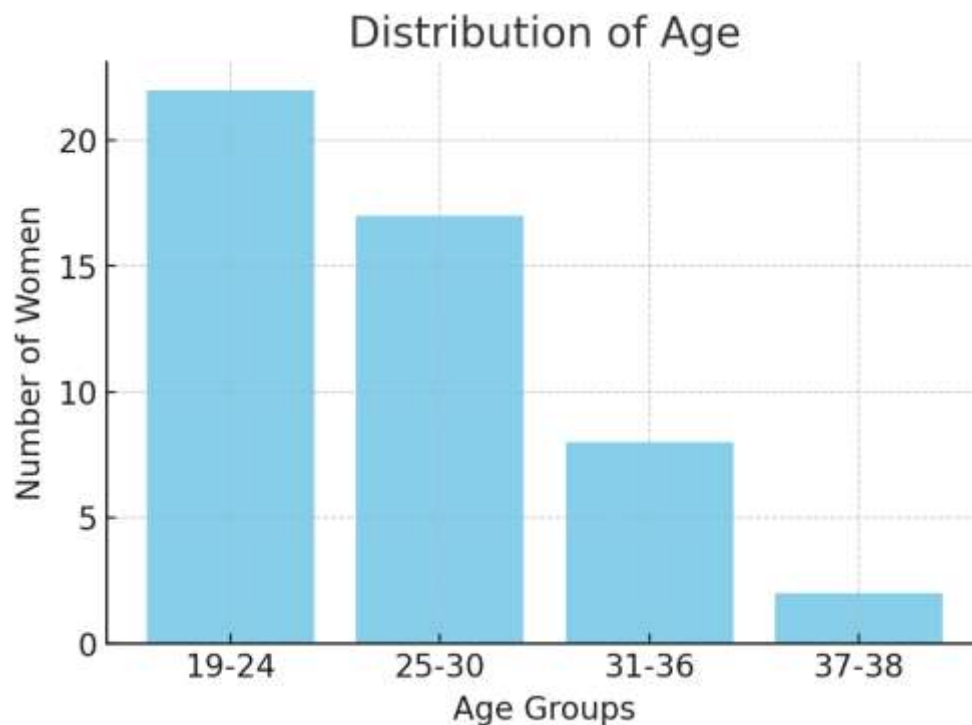
Statistical Analysis

Descriptive statistics were used given the non-comparative nature of this study. Categorical data (e.g., mode of delivery, cause of maternal death) are presented as frequencies and percentages, whereas continuous data (e.g., age, serum creatinine) are summarized as mean $\pm$ standard deviation or median with interquartile range, as appropriate. Bar charts or pie charts illustrate key distributions:

1. Age
2. Parity
3. Mode of delivery
4. Etiology of AKI
5. Dialysis requirement and number of dialysis sessions
6. Maternal deaths and corresponding causes
7. Trends in serum urea and creatinine levels
8. Mortality among patients who underwent dialysis

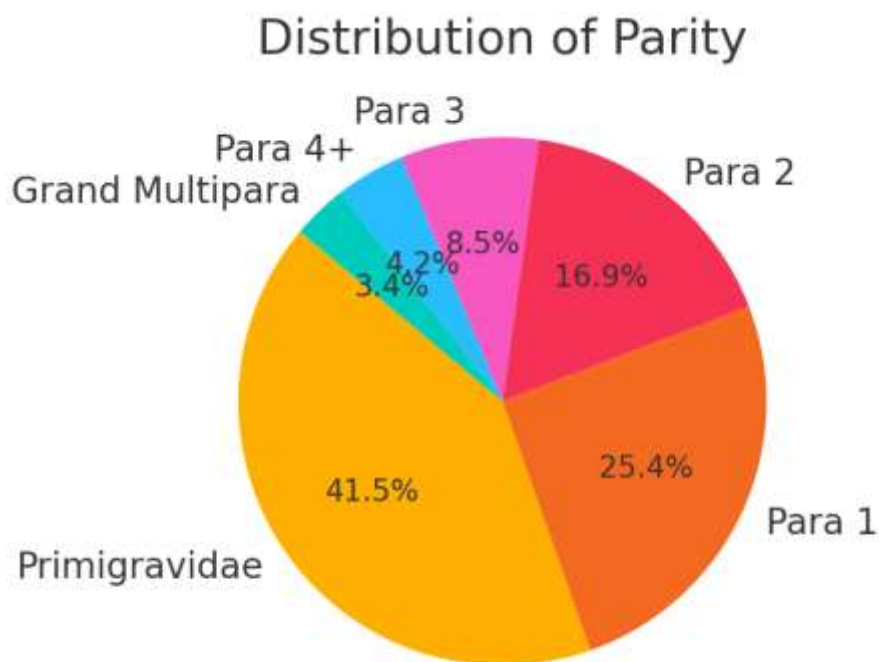
RESULTS

1. Distribution of Age



Bar Chart demonstrates the age distribution of the 49 women. The mean age was 25.9 ± 4.5 years (range: 19–38 years), with the largest subset (approximately 45%) being 20–25 years old.

2. Distribution of Parity



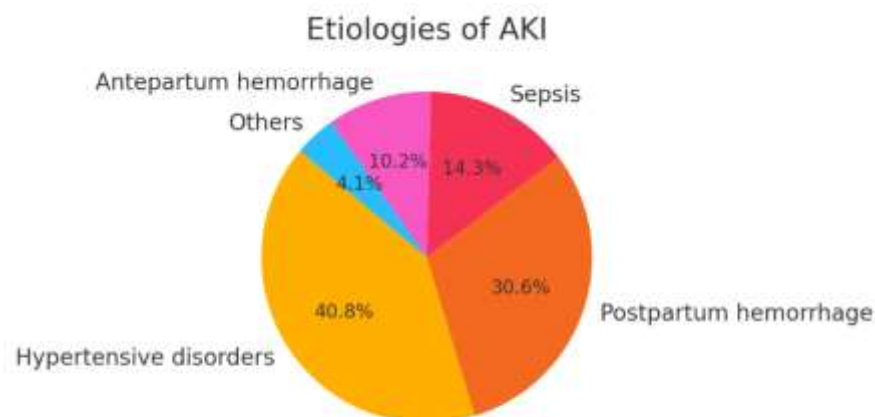
Pie Chart illustrates the parity distribution. Primigravidae constituted nearly 50% of cases, whereas others ranged from para 1 to grand multipara (up to G7P6L6). Higher parity often correlated with comorbidities such as anemia and hypertensive disorders.

3. Mode of Delivery



Bar Chart compares cesarean section versus vaginal delivery. A total of 28 women (57.1%) underwent cesarean delivery (both elective and emergency), and 21 (42.9%) delivered vaginally. Emergency cesarean sections were frequently done in the context of severe preeclampsia or abruptio placentae.

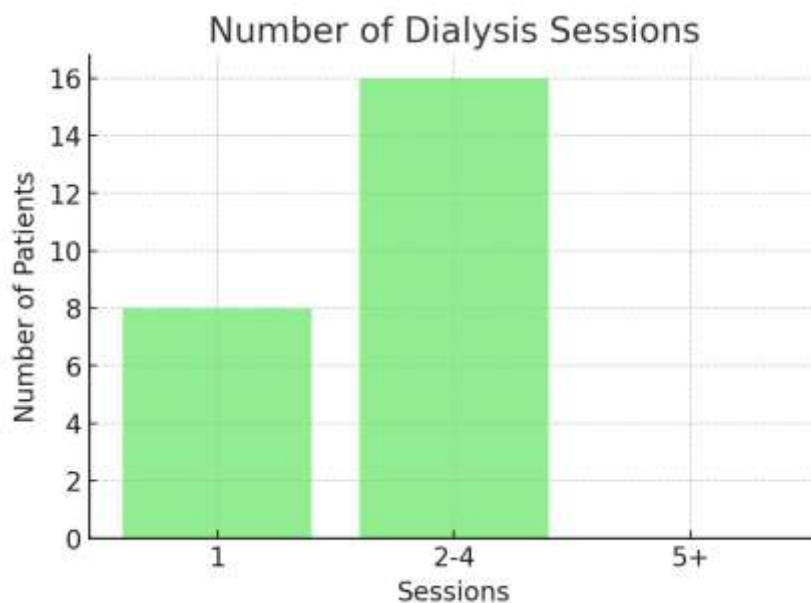
4. Etiologies of AKI



As shown in **Pie Chart**, the most common etiologies were:

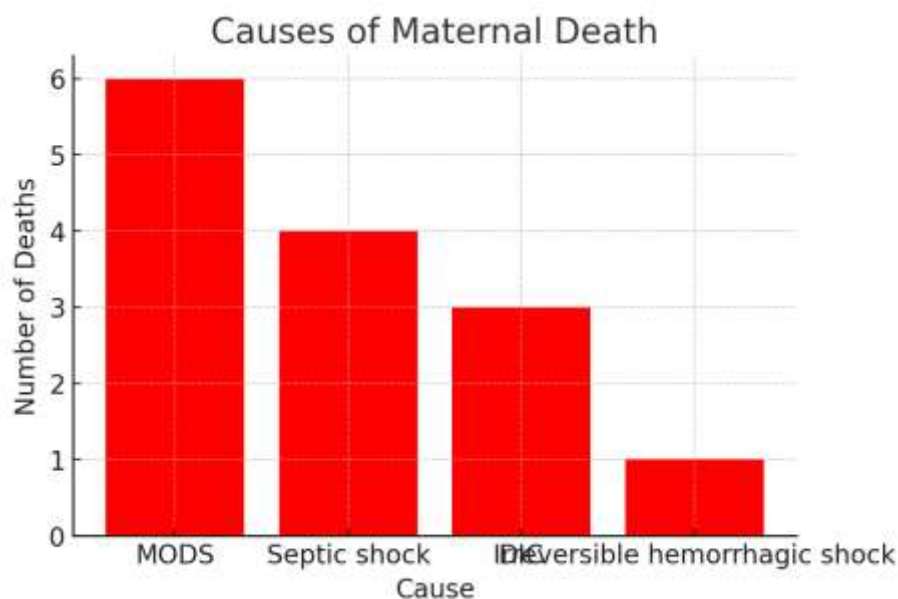
- Hypertensive disorders (40.8%), including severe preeclampsia, eclampsia, and HELLP syndrome.
- Postpartum hemorrhage (30.6%), attributed to atonic or traumatic causes.
- Sepsis (14.3%), often puerperal or secondary to surgical site infection.
- Antepartum hemorrhage (10.2%), predominantly abruptio placentae.
- Other causes (4.1%), such as severe anemia or gastroenteritis.

5. Requirement of Dialysis and Number of Sessions



Bar Chart illustrates the number of dialysis sessions per patient. Approximately half the cohort—24 of 49 women (49.0%)—required renal replacement therapy (RRT). Most patients (n=16) received 2–4 sessions, guided by oliguria, azotemia (urea >100 mg/dL, creatinine >3 mg/dL), and metabolic disturbances (e.g., refractory acidosis).

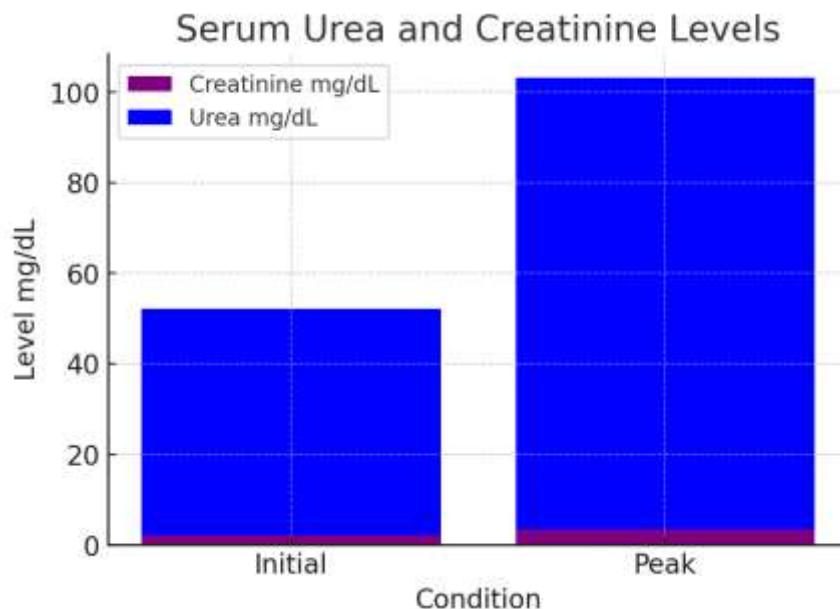
6. Maternal Death and Cause of Death



Overall maternal mortality was 28.6% (14/49). **Bar Chart** delineates the primary causes of death:

- **Multiorgan dysfunction syndrome (MODS):** 6 (42.9% of deaths)
- **Septic shock:** 4 (28.6% of deaths)
- **Disseminated intravascular coagulation (DIC):** 3 (21.4% of deaths)
- **Irreversible hemorrhagic shock:** 1 (7.1% of deaths)

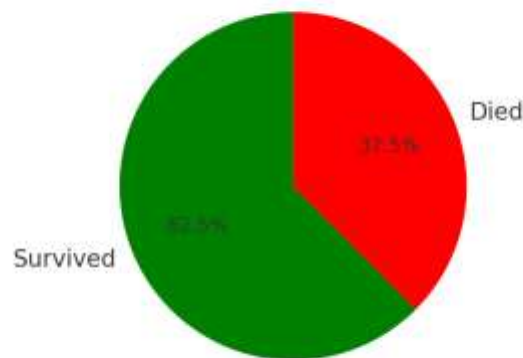
7. Serum Urea and Creatinine Values



Bar Chart presents the initial and peak urea/creatinine levels observed during hospitalization. On admission, average creatinine was 2.1 ± 0.7 mg/dL, escalating to 3.3 ± 1.2 mg/dL in severe cases. Urea levels were >100 mg/dL in patients who eventually required dialysis.

8. Mortality Among Patients Who Underwent Dialysis

Mortality Among Patients Who Underwent Dialysis



Pie Chart depicts survival versus mortality in this subset. Of the 24 patients who received dialysis, 9 died (37.5% mortality within the dialysis subgroup). The principal factors contributing to death in these women included septic shock, persistent hypotension despite volume resuscitation, and advanced multiorgan failure.

Additional Procedures

Emergency interventions—such as hysterectomy and bilateral internal iliac artery ligation—were undertaken in 24 patients (49.0%) to control catastrophic hemorrhage. Many of these patients had multiple comorbidities, including severe anemia and coagulopathy, further compounding renal injury.

Fetal/Neonatal Outcomes

Fetal/neonatal mortality was 24.5% (12/49), primarily associated with:

- Extreme prematurity and low birth weight.
- Intrauterine fetal demise.
- Neonatal sepsis.

Twenty (40.8%) mothers recovered fully with normalization of renal function by discharge, while 15 (30.6%) were discharged with partial renal recovery and advised close nephrology follow-up.

DISCUSSION

The findings of this study reinforce the pivotal role of hypertensive disorders, hemorrhage, and sepsis in precipitating pregnancy-related AKI [9,10]. Hypertensive complications, notably severe preeclampsia and HELLP syndrome, were the most frequent etiologic factors. Their underlying pathophysiological mechanisms—endothelial injury, vasospasm, and microangiopathic hemolysis—are well-known to incite acute renal dysfunction [11].

Postpartum hemorrhage was another crucial contributor; its severity often led to hypoperfusion and ischemic renal damage, which can rapidly progress to acute tubular necrosis [12]. Urgent surgical interventions (e.g., hysterectomy, bilateral internal iliac artery ligation) are frequently lifesaving, but delays in referral and persistent hypovolemia can further compromise renal perfusion [13].

Sepsis-related AKI was also notable, reflecting the high burden of puerperal infections and the complex interplay of inflammatory mediators in causing renal injury [6,14]. Timely antibiotic administration, hemodynamic support, and renal replacement therapy can improve survival, but resource constraints often limit comprehensive management in low- and middle-income settings [15].

Maternal mortality was alarmingly high (28.6%), with multiorgan dysfunction and septic shock being predominant end points. Notably, more than one-third of women requiring dialysis

ultimately died (9/24). These statistics underscore the severity of illness and the need for early detection of impending renal failure [16]. Moreover, a streamlined referral system that facilitates prompt intervention and critical care support is indispensable to enhancing outcomes.

Fetal and neonatal outcomes mirror maternal instability: a 24.5% fetal/neonatal mortality rate underscores the significant impact of maternal compromise on placental perfusion and neonatal well-being. Addressing modifiable risk factors—such as early correction of anemia, meticulous blood pressure control, and prompt recognition of peripartum sepsis—can mitigate some of these adverse outcomes [17].

CONCLUSION

AKI in pregnancy remains a formidable challenge in tertiary care, often stemming from hypertensive disorders, obstetric hemorrhage, and sepsis. This retrospective analysis illustrates that nearly half of the affected women required dialysis, with a substantial proportion succumbing to multiorgan dysfunction. Adverse fetal and neonatal outcomes further highlight the shared maternal-fetal vulnerabilities. Efforts to curtail maternal mortality and morbidity must focus on:

1. **Early identification** of AKI in high-risk pregnancies.
2. **Timely referral** to centers equipped with critical care and dialysis facilities.
3. **Aggressive management** of the underlying etiology, whether hemorrhage, hypertension, or sepsis.
4. **Strengthening antenatal services** and ensuring multidisciplinary obstetric and nephrology collaboration.

Enhancing healthcare infrastructure, refining care pathways, and reinforcing training in obstetric emergencies can substantially improve both maternal and fetal outcomes in resource-limited contexts.

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