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ACUTE KIDNEY INJURY ASSOCIATED WITH PREGNANCY: RENAL OUTCOMES

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Abstract

Background: One of the most difficult and dangerous pregnancy problems is acute kidney injury (AKI). We discuss our observations regarding the clinical characteristics and results of 60 patients who had AKI due to pregnancy who were seen throughout the research period. Examining the prevalence, characteristics, and effects of acute kidney injury (AKI) during pregnancy in the Indian population was the aim of this study.

Materials and Method: in the study the patients were women who were pregnant with AKI. To evaluate the acute kidney injury the parameters involves is RIFLE (Risk, Injury, Failure, Loss of Function and End stage Renal Disease). Patients were examined using demographic data, a detailed history, a clinical examination, and laboratory tests. The main result was a change in maternal renal function, including progression to chronic kidney disease and restoration to normal renal function (CKD). The delivery method, pregnancy-related issues and maternal mortality were all regarded as secondary outcomes.

Results: The present study included 60 patients in total, with an average age of 26 years. 41.2% of the individuals were primigravida, and 49.9% had severe anaemia. AKI was primarily brought on by pre-eclampsia and postpartum haemorrhage. During the three-month follow-up period, it was noted that the renal outcome had significantly improved, with 30 patients achieving full renal recovery and remaining patients developing CKD with mild to no recovery. Serum glutamic oxaloacetic transaminase and glutamic pyruvic transaminase levels were increased in all individuals but later recovered to normal.

Conclusion: According to our study, women with AKI due to pregnancy frequently experience multi-organ problems and need mechanical ventilation and renal replacement therapy. As a result, managing AKI caused by pregnancy offers a challenge that necessitates an accurate assessment of the contributing factors to enable effective therapy.

Keywords: Acute Kidney Injury, Pregnancy, preeclampsia, serum creatinine

Introduction

Acute kidney injury (AKI), which is characterised by a sudden and persistent reduction in kidney function, includes both subclinical kidney damage and renal impairment.[1] Pregnancyrelated AKI (PR-AKI) is a significant factor in the morbidity and death of both the mother and the foetus during pregnancy.[2] Because to advancements in prenatal and postnatal treatment, the prevalence of PR-AKI fell from 7% to 4.68% between 2000 and 2014. AKI prevalence during pregnancy in India ranged from 0.02 to 11.5%. Based on physiological changes happening during pregnancy, the progression of

AKI is evaluated.[3] The development is sometimes regarded as bimodal since it differs from early to late in the gestational phase and even throughout the postpartum stage.[4] Any stage of pregnancy can experience pregnancyrelated AKI, however the third trimester and the postpartum period have the highest frequency (39%) of fetal/newborn mortality. It is discovered to be connected to septic shock or dehydration brought on by hyperemesis during the first trimester.[4] The third-trimester disease is linked to pre-eclampsia/eclampsia, antepartum and postpartum hemorrhage, puerperal sepsis, hemolytic uremic syndrome, disseminated intravascular coagulation (DIC), hemolysis, increased liver enzymes, and low platelet levels syndrome.[5] A greater rate of maternal and perinatal morbidity and mortality is caused by an increase in the risk of co-occurring preeclampsia and pregnancy-related hypertension diseases. Although there are particular medicines available for the management of AKI, such as renal replacement therapy and targeted medication therapy, their effectiveness has not been demonstrated in a human clinical trial.[6] Clinicians can diagnose PR-AKI earlier with the support of early risk factor identification, which calls for specialized treatment interventions. Little urine production and blood chemistries are signs of mild renal impairment. One of the known indicators is serum creatinine.[7] In severely ill patients, serum creatinine may eventually fail to reflect a true decline in glomerular filtrate rate (GFR). The techniques for supplementary diagnosis used to diagnose AKI are the foundation of the current investigation. Urine dipstick tests, urine microscopy, renal ultrasonography, and renal biopsies are some of the diagnostic methods for structural renal disease and/or urinary obstruction.[8] The occurrence of AKI during pregnancy is a critical clinical issue since both the mother and the foetus must be taken into consideration. Moreover, certain pregnancy diseases that are still poorly understood may contribute to it. Since only a few retrospective studies have provided information on the incidence, aetiology, and effects of PR-AKI, a significant component of AKI in India, there was a need for a prospective investigation. The current study aims to evaluate the renal outcomes of individuals with PR-AKI as well as the aetiology of PR-AKI.

Material and methods

In the tertiary care hospital, a prospective observational study was conducted. Every patient's individual written consent was obtained. AKI is defined as either a rise in serum creatinine of 26.5 mol/L or higher within 48 hours, an increase to 1.5 times baseline, or a decreased urine volume of 0.5 ml/kg/h for six hours, according to the Kidney Disease Improving Global Outcomes (KDIGO).[9]

Inclusion criteria

This study included all consecutive AKI patients, regardless of origin, such as pre-renal, renal, or post-renal, and monitored them for the development of chronic kidney disease over a period of three months.

Exclusion criteria

Individuals with bilateral contracted kidneys, preexisting diabetes mellitus, renal disorders, hypertensive nephropathy, recipients of renal transplants, or CKD were excluded from the study.

Methodology

Demographic information, a thorough medical history, a clinical examination, and laboratory tests like a complete hemogram, blood urea, blood creatinine, serum calcium, serum phosphorous, serum uric acid, serum protein ratio, serum sodium, serum potassium, blood sugar, and bilirubin were all used to analyse a group of patients. A comprehensive obstetric examination was performed on each subject. During hospitalisation, alternate-day renal functional tests were carried out. A specific investigation into the delivery method was made. All patients were treated either conservatively or with hemodialysis in accordance with accepted guidelines. Their clinical traits were evaluated at the one- and three-month follow-up periods after discharge.

The key outcome was the maternal renal outcome, which included a rebound to normal renal function and a progression to CKD requiring or not requiring dialysis. Secondary outcomes included the manner of birth, pregnancy problems such antepartum, intrapartum, or postpartum, admission to the

intensive care unit (ICU), and mother death. The Statistical Package for the Social Sciences (SPSS) version 20.0 was used to conduct the statistical analysis. Quantitative data were shown as mean standard deviation (SD), while qualitative data were shown as n (%). A $P<0.05$ was regarded as statistically significant in every analysis.

Results

This study comprised 60 pregnant women with AKI in total. The patients were 26 years old on average. The gestational period was 34.3 weeks on average (4.2). As seen in Table 1, 37 study participants gave birth naturally while 23 underwent caesarean sections.

Table 1: Baseline clinical characteristics

Parameters	Total (n=60)
Average Age(years)	26
Gestation period	
<28	4
28 to 34	14
34-37	11
>37	31
Mode of Delivery	
Vaginal	37
C-Section	23

Fever (82.5%), edoema (81.45%), dyspnea (72.60%), oliguria (65.8%), vaginal haemorrhage (35%), anuria (22%), altered level of consciousness (19%), and convulsions (14%) were the clinical signs that were documented at the time of presentation. Those with reduced or no urine production also reported many presenting symptoms.

Pre-eclampsia/eclampsia (n = 19; 33.3%), antepartum haemorrhage (APH) (n = 8; 14%), postabortion sepsis (n = 9; 15.7%), and puerperal sepsis (n = 36; 63) were among the factors that affected the study participants and led to the development of AKI. In several circumstances, a number of variables contributed to AKI.

Pre-dialysis serum creatinine was 6.5 ±2.5 mg/dL (range 2.2-16.22 mg/dL), and pre-dialysis blood urea was an average of 129± 49 mg/dL (range 40-229 mg/dL). Prior to the start of the diuretic phase of the hospitalisation, there was oliguria or anuria for 12.8–6.4 days (range 5-30 days).

The range of 2.7-6.3 mmol/L for the mean serum potassium was 4.3± 0.8 mmol/L. All patients received dialysis as necessary in addition to antibiotic treatment for infections. The average number of 4-hour long dialysis sessions needed by each patient was 6.9 (range 0–20). Seven patients were managed conservatively and all seven recovered fully without the need for dialysis. Among the fifty patients who received HD, three initially had PD due to hemodynamic instability and were subsequently switched to HD. All patients had thorough ultrasonography and gynecological assessment. Eight patients had their retained sperm and egg removed, and one had to have a hysterectomy due to uncontrollable uterine haemorrhage.

Nine of the 60 study participants passed away (15%), whereas 30 (or 50%) showed total renal recovery, 15 (or 25%), had partial recovery, and 6 (or 10%) showed no recovery. Table 2 compares laboratory variables and their impact on outcomes for study participants.

Table 2: Comparison of laboratory variables and their impact on outcomes in individuals with acute renal damage due to pregnancy

Sr. No	Parameters	% Complete Recovery (n=30)	% Partial Recovery (n=15)	% No Recovery (n=6)	% Expired (n=9)
1	Haemoglobin	30 (9)	26.6 (4)	16.6 (1)	77.7 (7)
2	Thrombocytopenia	27 (8)	40 (6)	33.3 (2)	66.6 (6)
3	Jaundice	10 (3)	26.6 (4)	0 (0)	77.7 (7)
4	D-dimer elevated	30 (9)	33.3 (5)	50 (3)	55.9 (5)
5	Activated Partial Thromboplastin	30 (9)	33.3 (5)	50 (3)	77.7 (7)
6	Decreased palsma fibrinogen	27(8)	33.3 (5)	50(3)	44.6 (4)

Only one patients of the 12 patients with total anuria fully recovered, and the other five required ongoing dialysis therapy. The remaining six patients, or 50% of those with anuric AKI, had partial renal recovery and were dialysis independent. ATN (n = 40), which is commonly accompanied by septicemia (n = 28), pre-eclampsia (n = 14), APH (n = 7) and PPH (n = 2), either alone or in combination, was the cause of AKI in the study subjects. 13 patients (22.81%) showed cortical necrosis, which was more common in late pregnancy (11 of 13; 84.62%) than in early pregnancy (only one of 13; 7.21%). The four most frequent causes of late pregnancy cortical necrosis were eclampsia, followed by intrauterine mortality, puerperal sepsis, APH, and PPH in that order. Nine patients (15%) died. Septicemia (n = 4; 44.44%), pre-eclampsia (n = 3; 33%), APH (n = 1), and PPH (n = 1) were the reasons of death. Pre-eclampsia (n = 11; 57%), APH (n = 7), PPH (n = 3; 60%), and septicemia (n = 7; 47.2%) were the most frequent causes of the 28 cases of foetal fatality that were recorded.

We found that foetal loss occurred in 87.5% of patients with APH and 60% of those with PPH. Maternal sepsis and PPH were most frequently linked to postpartum mortality (40%) and 40%, respectively, but pre-eclampsia and APH were more frequently linked to IUD. E. coli (n = 12), Pseudomonas (n = 7) and Klebsiella (n = 5) were among the common organisms produced on blood culture. Acute tubulointerstitial nephritis, localised and segmental glomerulosclerosis, membranoproliferative glomerulonephritis, membranous glomerulonephritis, and cortical necrosis were all identified in the 21 patients (36.84%) who underwent renal biopsy.

Duration of oliguria, haemoglobin level, blood urea, and SCr did not affect mortality, according to analysis of the individual dependent variables using a Bonferroni-adjusted significance level of 0.01. These values were P = 0.629, 0.266, 0.224, and 0.637, respectively. The length of oliguria was significantly related to renal recovery (P = 0.001).

Discussion

AKI associated with pregnancy has a reported incidence of 5–40%.[10] Studies have found that while this frequency is down in both developed and developing nations, it is declining less rapidly in the latter.[11] This decline in incidence could be attributed to prevention of pregnancy-related complications such as septic abortion, early and more effective preeclampsia treatment, and timely lower caesarean delivery on a selective basis. I have. In our study, the mean age of participants was 26 years, whereas in the study by Grunfeld et al. and Chug et al. The slightly lower average age of study participants may reflect the younger average age of marriage. In 1976, Chug et al.[12] We found that both 29 late pregnancies (40.35%) and 43 early pregnancies (59.7%) experienced her AKI. About half of the participants had abortioninduced renal failure, compared to the study by Chugh et al. This difference can be explained by the fact that more patients are now receiving prenatal care than before. More than half of expectant women still don't follow antenatal care, even in recent years. 37 individuals (64.9%) in this research had not received any prenatal care. From our analysis Chugh et

al.[13] found comparable results, where 31.9% of patients had anuria, 55.6% oliguria, and 12.5% nonoliguria. Other studies estimate the maternal mortality rate to be between 14% and 32%. The maternal mortality rate (15.78%) in the present study was lower than that reported by Chugh et al (30%).[14] This may be because septic abortion is less common (22%) and patients started hemodialysis sooner (85%). The time from the onset of renal failure to referral to our laboratory ranged from 1 day to 30 days in our study, with a mean of 6.5 days. The mean blood creatinine in the present study was 6.54 mg/dL, which was lower than 15.4 mg/dL in the study by Chugh et al. [13] Hemodialysis was given to the majority of our patients (85.96%) since, barring medical necessity, it is the recommended form of dialysis for AKI patients at our facility. In the current study, late pregnancy (11 of 13 instances, or 84.62%) was associated with cortical necrosis more commonly than early pregnancy (just two cases). Yet, several studies have shown that cortical necrosis can happen both early and late in pregnancy. Cortical necrosis impacted 25% of the eclampsia group in the Chugh et al. experiment, and serious mortality occurred in 75% of cases. In their investigation, the prevalence of APH was 6.94%; cortical necrosis affected 80% of these individuals, and mortality was 100%. Cortical necrosis was present in 16.7% of the PPH group, and death was 50%. 33.3% of those with puerperal sepsis showed cortical necrosis, and 44.4% of them died.[15] In the study by Jai Prakash et al., only 3.17% of the patients had APH; none of them exhibited cortical necrosis, but 20% of patients with PPH did.

In the study by Grunfeld et al., there was no maternal mortality in the group of PPH patients, but 50% of them developed cortical necrosis. 28 cases (49.12%) of foetal loss were observed in our investigation. Maternal sepsis (40%) and PPH (40%) were more frequently linked to post-natal fatalities, while pre-eclampsia and APH were more frequently linked to intra-uterine deaths in patients.[16] According to a study from Casablanca, 5.5% of patients experienced foetal death and 9.1% of patients experienced maternal death. According to earlier research, septic shock and PIH were the most frequent causes of maternal death in our analysis. 75 women with AKI due to pregnancy were reported by Erdemoglu et al. Ages of the cases ranged from 21 to 46, and 36% happened after giving birth.[17] Sepsis was the primary cause of pregnancy-related AKI in 14.6% of cases, followed by hemorrhage in 12%, fetal toxicity in 75.2%, and sepsis after abortion in 14.6%. 33.3% of patients required dialysis and the maternal mortality rate was 10.6%. Arora et.al [18] Among the 57 patients they examined, sepsis (33.3%), hemorrhage (28.1%), and hypertensive disorders (26.3%) were found to be the three leading causes of pregnancy-related AKI. Septic abortion (50%) and postpartum AKI (2.5%) were the most common causes of pregnancy-related AKI, followed by APH (15%), preeclampsia (15%), acute gastroenteritis (7.5%) and PPH. (5%), acute pyelonephritis (5%), and AKI. Carpenter et.al We investigated 569 cases of pregnancy-related AKI and found septic abortion (50%) and postpartum AKI (2.5%). Sixty percent of patients required dialysis and the mortality rate was 20%.[19] In 59 patients with pregnancy-related AKI, Sivakumar et al. They reported that 74.57% of cases occurred in the postpartum period, 16.94% in the third trimester, 6.77% in the second trimester, and 1.69% in the first trimester. In their investigation, postpartum sepsis was the leading cause of AKI.

Conclusion

Puerperal sepsis, together with the development of pre-eclampsia, APH, and PPH, appears to be the most common etiological cause in pregnancy-related AKI, which is a common medical issue. Long-term oliguria or complete anuria are negative prognostic indicators. Managing PRAKI is a therapeutic problem that necessitates an accurate assessment of potential contributing factors to enable effective treatment. Morbidity and death linked to PR-AKI may be reduced with early diagnosis and prompt treatment.

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