

DOI: <https://dx.doi.org/10.18203/2320-1770.ijrcog20231567>

Original Research Article

Study of clinical profile and outcome in pregnancy related acute kidney injury

Jagruti Brahmbhatt¹, Kirti Devada^{1*}, Manishkumar Patel²

¹Department of Obstetrics and Gynecology, Government Medical College Baroda, Gujarat, India

²MBBS, PG Certificate in Public Health, Gujarat, India

Received: 21 April 2023

Accepted: 11 May 2023

***Correspondence:**

Dr. Kirti Devada,

E-mail: dr.kirti112@gmail.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Background: Acute kidney injury (AKI) is a clinical syndrome characterized by a sudden decrease in glomerular filtration rate leading to decreased excretion of nitrogenous waste like urea, creatinine and other uremic toxin. Even minor changes in serum creatinine are associated with increased in-patient mortality. To study various etiological factors responsible for AKI to do better management of condition for prevention of adverse effects on maternal and fetal outcome. To study outcome of disease in form of recovery, morbidity and mortality. To record maternal and foetal outcome.

Methods: A prospective, observational study of 50 patients conducted at department of obstetrics and gynaecology, Medical College Baroda and Sir Sayajirao General Hospital from the time period of 1 year.

Results: There was no significant association of initial serum creatinine with maternal outcome. However, there was significant association of last serum creatinine and Percentage Improvement in serum creatinine with maternal outcome as last serum creatinine was significantly lowest in completely recovered patients as compared to dead and discharged on request patients and percentage Improvement in serum creatinine was significantly high in completely recovered patients as compared to dead and discharged on request patients. There was no significant association of initial and last serum creatinine with fetal outcome; however, percentage improvement in serum creatinine was significantly high in alive fetuses as compared to aborted, dead and undelivered fetuses.

Conclusions: Pregnancy-related AKI is a common medical problem and understanding its association with various etiopathologies has significant impacts on maternal and fetal outcome.

Keywords: Acute kidney injury, Fetuses, Pregnancy, Serum creatinine

INTRODUCTION

Acute kidney injury is a clinical syndrome characterized by a sudden decrease in glomerular filtration rate leading to decreased excretion of nitrogenous waste like urea, creatinine and other uremic toxin. The reported incidence of obstetric AKI in the developed countries is 1-2.8%, while in developing countries it is 9-25%.¹ AKI that requires dialysis is rare in developed countries, with an incidence of 1:20,000 or less, of all gestations.^{2,3}

Recent epidemiological studies demonstrate wide variation in etiologies and risk factors describe the increased mortality associated with this disease (particularly when dialysis is required) and suggest a relationship to the subsequent development of chronic kidney disease and progression to dialysis dependency. Emerging evidence suggests that even minor changes in serum creatinine are associated with increased in-patient mortality. Mortality remains very high and the risk for maternal and fatal complications are also significant.⁴⁻⁸

Normal pregnancy accompanies a number of physical and psychological changes. The urinary tract is a system that demonstrates remarkable alterations in both anatomy and physiology. Understanding of these changes is crucial in evaluating renal diseases in pregnant women. AKI in pregnancy bears a high risk of development of bilateral renal cortical necrosis and, consequently, of chronic renal failure. Obstetric complications constitute the most common cause of renal cortical necrosis (50-70%).³⁻⁵ Abruptio placenta, septic abortion, eclamptic toxemia, post-partum haemorrhage (PPH) and puerperal sepsis are the pregnancy related situations responsible for causing renal cortical necrosis.⁵

This study will help us to have

Better understanding of etiopathologies associated with Pregnancy related acute kidney injury (PRAKI).

Early diagnosis and classification of PRAKI; thereby enhancing our ability to manage and improve care for patients with or at risk for AKI.

METHODS

This was one-year prospective observational study. Labour room, obstetrics ICU, Obstetrics and Gynaecology department of SSG Hospital. Study will be carried out in all patients admitted in labour room, obstetrics ICU and of Obstetrics and Gynaecology who meet my inclusion criteria.

All the patients fulfilling my inclusion criteria during the study period of one year. Sample size is calculated with help of www.openepi.com site. Approximately 50 samples were selected.

Inclusion criteria

Patients attending labour room, obstetrics ICU, of SSG Hospital who develops kidney injury with following findings will be included in study.

Increase in SCr by ≥ 0.3 mg/dl (≥ 26.5 micromol /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.

Exclusion criteria

Known case of renal disease (Renal insufficiency from any cause, GN). History of hypertension and diabetes before gestation. History of NSAID or analgesic abuse. Previous

urological surgery. History of renal stone. History of reflux nephropathy. Renal scarring shown on ultrasound. Smaller size kidney for age and size.

Methodology

All the patients attending Labour room, Obstetrics ICU, Obstetrics Ward fulfilling my inclusion criteria will be recruited in the study. Patients fulfilling my inclusion criteria and willing to give written informed consent about study after explanation of purpose of study will be included.

Patients will be provided with written informed consent in English and local language.

All patients will undergo detailed history taking, clinical examination, radiological assessment, biochemical and haematological investigations will be carried out. Specific inquiries will be conducted regarding age, parity, gestational age, antenatal care taken or not, no. of antenatal visit, mode of delivery number of blood component transfusions, operative procedure.

All patients will be classified according to AKIN classification. All patient's outcome will be recorded in form of maternal and fetal outcome.

Statistical analysis

The recorded data was compiled and entered in a spreadsheet computer program (Microsoft Excel 2007) and then exported to data editor page of SPSS version 15 (SPSS Inc., Chicago, Illinois, USA). For all tests, confidence level and level of significance were set at 95% and 5% respectively.

RESULTS

This prospective observational study was conducted at labour room, and obstetrics ICU, of the department of obstetrics and gynaecology of SSG Hospital over duration of 1 year. After applying inclusion/exclusion criteria and taking informed consent, 50 pregnant patients with kidney injury were included in the study. All patients were classified according to AKIN classification. Following were the results related to the study.

Mean age of the patients in our study was 26.56 ± 5.04 years. Majority of the women in study were in the age group 21-30 years (68.00% patients), followed by 18.00% patients in 31-40 years age group. Mean height and weight of the patients was 152.48 ± 5.91 cm and 49.7 ± 6.97 kg, respectively. Majority of the patients belonged to Hindu religion (82.00% patients) and from lower socioeconomic class (98.00% patients). Most of the patients were unbooked (68.00% patients); gravida 1 (34.00% patients) followed by gravida 2 (24.00% patients) and gravida 3 (24.00% patients); and nullipara (32.00%) followed by para 2 (28.00%).

Table 1: Distribution of demographic and clinical characteristics of study subjects.

Demographic and clinical characteristics	Frequency	%
Age distribution in years		
<=20	7	14.00%
21-30	34	68.00%
31-40	9	18.00%
Mean ± Stdev	26.56±5.04	
Median (IQR)	26 (23-30)	
Height (cm)		
Mean ± Stdev	152.48±5.91	
Median (IQR)	152 (148-156)	
Weight (kg)		
Mean ± Stdev	49.7±6.97	
Median (IQR)	49 (45-52)	
Religion		
Hindu	41	82.00%
Muslim	9	18.00%
Socio-economic status		
High	1	2.00%
Low	49	98.00%
Booking status		
Booked	16	32.00%
Unbooked	34	68.00%
Gravida		
G1	17	34.00%
G2	12	24.00%
G3	12	24.00%
>G3	9	18.00%
Parity		
P0	16	32.00%
P1	11	22.00%
P2	14	28.00%
P3	6	12.00%
>P3	3	6.00%
Gestational age (in weeks)		
Preterm	37	80.43%
Term	9	19.57%
Mean ± Stdev	32.93±5.83	
Median (IQR)	34.93 (30.429-36)	
Family history		
All siblings has SCT	1	2.00%
Not significant	49	98.00%
Hemodialysis		
No	42	84.00%
Yes	8	16.00%
Systolic blood pressure (mmHg)		
Mean ± Stdev	135.83±24.48	
Median (IQR)	140 (120-150)	
Diastolic blood pressure (mmHg)		
Mean ± Stdev	86.67±14.04	
Median (IQR)	90 (80-100)	
Hemoglobin (gm%)		
Mean ± Stdev	7.39±2.58	

Demographic and clinical characteristics	Frequency	%
Median (IQR)	6.85 (5.700-8.800)	
Total count (/cumm)		
Mean ± Stdev	18596.6±8613.41	
Median (IQR)	17435 (12800-23060)	
Platelet count		
Mean ± Stdev	153208±77717.91	
Median (IQR)	153000 (92000-204000)	
Serum bilirubin (mg/dl)		
Mean ± Stdev	2.45±3.08	
Median (IQR)	1.1 (0.800-2.100)	
Serum urea (mg/dl)		
Mean ± Stdev	63.36±25.18	
Median (IQR)	59 (47-76)	
Pulse (minute)		
Mean ± Stdev	99.96±21.03	
Median (IQR)	96 (87.500-110.500)	
Platelet concentrate		
Mean ± Stdev	0.64±1.54	
Median (IQR)	0 (0-0)	
Packed cell volume		
Mean ± Stdev	3.1±2.65	
Median (IQR)	3(2-4)	
Cryoprecipitate		
Mean ± Stdev	0.54±1.42	
Median (IQR)	0 (0-0)	
Fresh frozen plasma		
Mean ± Stdev	2.96±5.38	
Median (IQR)	0 (0-4)	
International normalized ratio		
Mean ± Stdev	1.32±0.44	
Median (IQR)	1.15 (1.100-1.370)	
Single donor platelet		
Mean ± Stdev	0.2±0.61	
Median (IQR)	0 (0-0)	

Mean gestational age was 32.93±5.83 weeks. Family history was found in only 1 patient (2.00%) who had all siblings with SCT. Hemodialysis was required in 8 (16.00%) patients. Mean SBP was 135.83±24.48 mmHg; mean DBP was 86.67±14.04 mmHg; mean hemoglobin was 7.39±2.58 g%; mean total count was 18596.6±8613.41/cumm; mean platelet count was 153208; mean INR was 1.32±0.44; mean serum bilirubin was 2.45±3.08 mg/dl; and mean serum urea was 63.36±25.18 mg/dl. The patients received mean platelet concentrate (0.64±1.54); mean packed cell volume (3.1±2.65); mean cryoprecipitate (0.54±1.42); mean fresh frozen plasma (2.96±5.38); and mean single donor platelet (0.2±0.61).

As compared to initial serum creatinine, last serum creatinine was significantly lower (1.1 vs 2.6, P<.0001) (Figure 1).

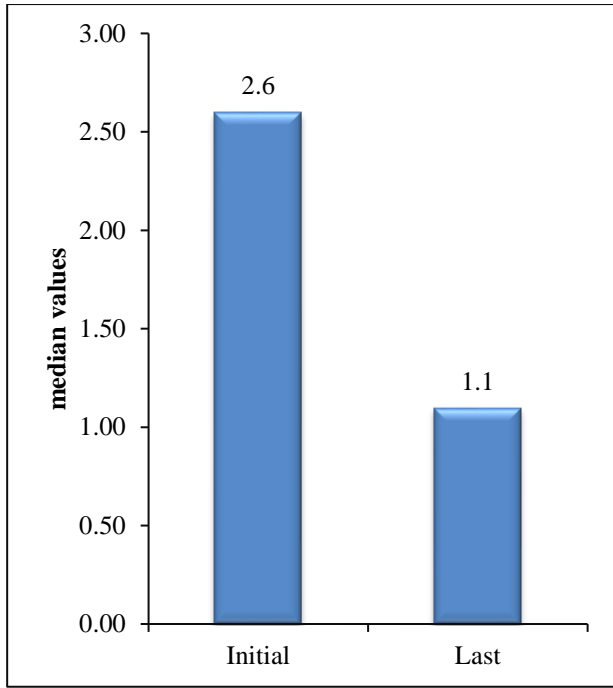


Figure 1: Comparison of initial serum creatinine with last value.

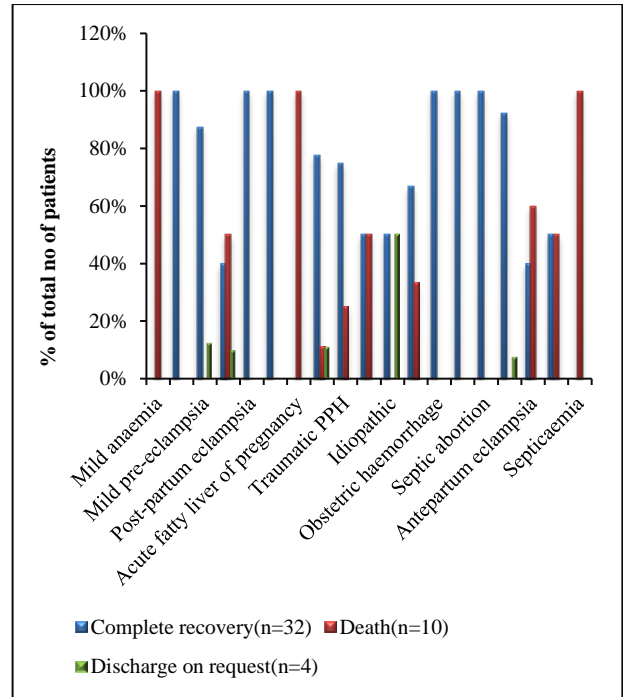


Figure 2: Association of etiological factors with maternal outcome.

Table 2: Association of serum creatinine with maternal outcome.

S. creatinine (mg/dl)		Complete recovery (n=32)	Death (n=10)	Discharge on request (n=4)	P value*
Initial	Mean ± Stdev	2.84±0.8	2.87±1.72	2.82±1.32	0.631
	Median (IQR)	2.65 (2.250-3.100)	2.56 (2.200-2.800)	2.25 (2.100-3.550)	
Last	Mean ± Stdev	1.08±0.28	3.22±1.8	2.44±1.74	0.0002
	Median (IQR)	1.1 (0.900-1.200)	2.4 (2.400-4.300)	2.18 (1.335-3.550)	
% Improvement in serum creatinine	Mean ± Stdev	59.68±14.47	-29.05±81.33	17.31±36.98	0.0001
	Median (IQR)	60.06 (52.849-71.198)	0 (-41.935-0.415)	0 (-1.750-36.364)	

*-Kruskal Wallis test.

Table 3: Association of etiological factors with fetal outcome.

Etiological factors	Fetal outcome				Total	P value*
	Abortus (n=2)	Alive (n=19)	Dead (n=28)	Undelivered (n=1)		
Mild anaemia	0 (0.00%)	0 (0.00%)	0 (0.00%)	1 (100.00%)	1 (100.00%)	<.0001
Postpartum haemorrhage	0 (0.00%)	1 (100.00%)	0 (0.00%)	0 (0.00%)	1 (100.00%)	0.645
Mild pre-eclampsia	0 (0.00%)	2 (16.67%)	10 (83.33%)	0 (0.00%)	12 (100.00%)	0.176
Severe anaemia	0 (0.00%)	3 (25.00%)	8 (66.67%)	1 (8.33%)	12 (100.00%)	0.185
Post-partum eclampsia	0 (0.00%)	1 (100.00%)	0 (0.00%)	0 (0.00%)	1 (100.00%)	0.645
Surgical site infection	0 (0.00%)	1 (100.00%)	0 (0.00%)	0 (0.00%)	1 (100.00%)	0.645
Acute fatty liver of pregnancy	0 (0.00%)	0 (0.00%)	1 (100.00%)	0 (0.00%)	1 (100.00%)	0.849
Severe pre-eclampsia	0 (0.00%)	5 (55.56%)	4 (44.44%)	0 (0.00%)	9 (100.00%)	0.613

Continued.

Etiological factors	Fetal outcome				Total	P value*
	Abortus (n=2)	Alive (n=19)	Dead (n=28)	Undelivered (n=1)		
Traumatic PPH	0 (0.00%)	3 (75.00%)	1 (25.00%)	0 (0.00%)	4 (100.00%)	0.462
HELLP syndrome	0 (0.00%)	1 (50.00%)	1 (50.00%)	0 (0.00%)	2 (100.00%)	0.975
Idiopathic	1 (50.00%)	1 (50.00%)	0 (0.00%)	0 (0.00%)	2 (100.00%)	0.006
Atonic PPH	0 (0.00%)	2 (66.67%)	1 (33.33%)	0 (0.00%)	3 (100.00%)	0.759
Obstetric haemorrhage	0 (0.00%)	1 (100.00%)	0 (0.00%)	0 (0.00%)	1 (100.00%)	0.645
Hepatitis E	0 (0.00%)	1 (100.00%)	0 (0.00%)	0 (0.00%)	1 (100.00%)	0.645
Septic abortion	1 (100.00%)	0 (0.00%)	0 (0.00%)	0 (0.00%)	1 (100.00%)	<.0001
Abruption placentae	0 (0.00%)	0 (0.00%)	14 (100.00%)	0 (0.00%)	14 (100.00%)	0.002
Antepartum eclampsia	0 (0.00%)	0 (0.00%)	5 (100.00%)	0 (0.00%)	5 (100.00%)	0.225
Placentae previa	0 (0.00%)	1 (50.00%)	1 (50.00%)	0 (0.00%)	2 (100.00%)	0.975
Septicaemia	0 (0.00%)	0 (0.00%)	2 (100.00%)	0 (0.00%)	2 (100.00%)	0.651

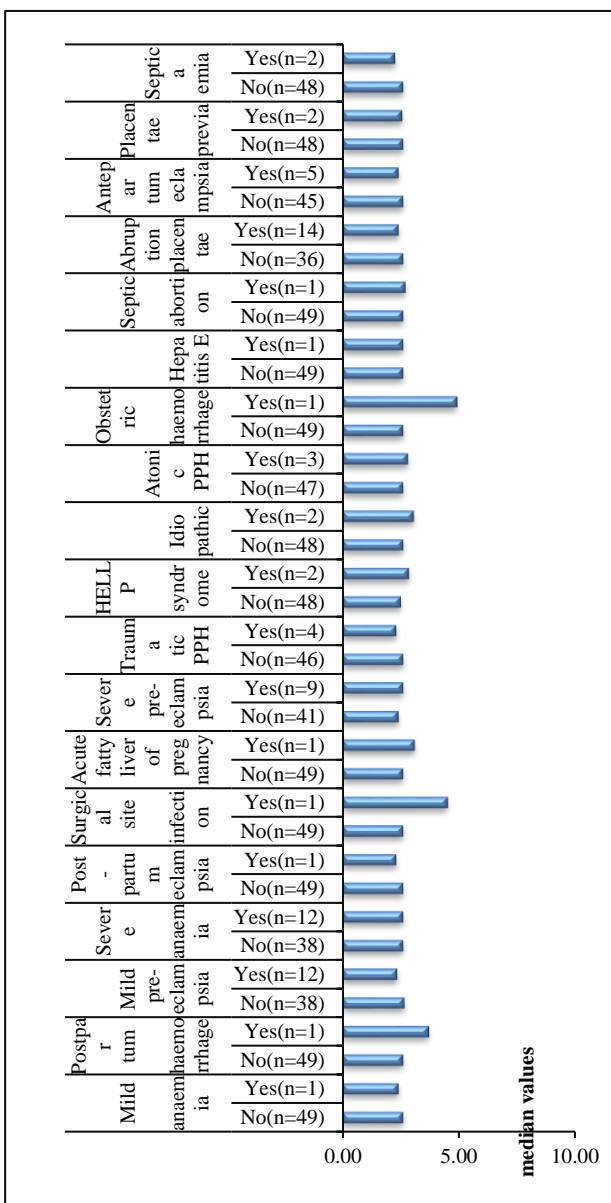


Figure 3: Association of initial serum creatinine with etiology.

In this study, mode of delivery in most of the patients (66.00%) was vaginal delivery, followed by emergency LSCS in 26.00% patients. In this study, 64.00% patients recovered completely, 4 patients got discharged on request, 4 patients did not follow-up, and death happened in 10 (20.00%) patients. In this study, 56.00% fetuses were dead, 38.00% fetuses were alive, 2 got aborted and 1 was undelivered.

There was no significant association of etiological factors with maternal outcome (Figure 2).

There was no significant association of initial serum creatinine with maternal outcome (P=0.631). However, there was significant association of last serum creatinine and % Improvement in serum creatinine with maternal outcome as last serum creatinine was significantly highest in dead as compared to completely recovered patients and discharged on request patients (2.4 vs 1.1 vs 2.18, P=0.0002), and % improvement in serum creatinine was significantly highest in completely recovered patients as compared to dead and discharged on request patients (60.06 vs 0 vs 0, P=0.0001). Among the patients who died, rather than improvement, there was a 30% deterioration in the serum creatinine.

There was no significant association of etiological factors with fetal outcome, except Mild anaemia (which was present in the only undelivered one), Septic abortion (found in only abortus), idiopathic causes (seen in alive and abortus) and Abruption placentae (in 14 dead fetus only) (P<0.05) (Table 3).

There was no significant association of etiological factors with mode of delivery (P>0.05) except mild anemia, idiopathic cause (P<.0001), septic abortion (P<.0001) and placentae previa (P<.0001). There was no significant association of initial SCr, last SCr, and % improvement in serum creatinine with mode of delivery (P>0.05).

There was no significant association of initial SCr, last SCr, and % improvement in serum creatinine with etiology

($P > 0.05$), except antepartum eclampsia where initial and last SCr were comparable, but % improvement in SCr was significantly higher in patients without antepartum eclampsia as compared to those with antepartum eclampsia (55.56 vs 0, $P = 0.021$) (Figure 3).

DISCUSSION

AKI in pregnancy bears a high risk of development of bilateral renal cortical necrosis and, consequently, of chronic renal failure. Obstetric complications constitute the most common cause of renal cortical necrosis (50-70%). Abruptio placenta, septic abortion, eclamptic toxemia, post-partum hemorrhage (PPH) and puerperal sepsis are the pregnancy-related situations responsible for causing renal cortical necrosis. Regardless of cause, the management of AKI is mainly supportive, with dialysis being indicated when medical management fails to treat the complications.⁹ We aimed to understand AKI characteristics in pregnancy and identify the factors related to its maternal and fetal outcomes. We also determined the outcome of disease in form of recovery, morbidity, and mortality.

Mean age of the patients in our study was 26.56 years. Majority of the women in study were in the age group 21-30 years. Mean height and weight of the patients was 152.48 ± 5.91 cm and 49.7 ± 6.97 kg, respectively. Comparable to our study findings, Godara SM et al, reported that the mean age of the study patients was 26.4 years and the most common age group affected was 20-25 years, while in the studies of Bekele D et al and Mahesh E et al the mean age was 28.9 years and 25 years, respectively.¹⁰⁻¹²

In our study, most of the patients were gravida 1 followed by gravida 2 and gravida 3 and nullipara followed by para 2. Mean gestational age was 32.93 ± 5.83 weeks. In contrast to our study, in study by Godara SM et al, majority were multigravida and primigravida.¹⁰

Mean SBP was 135.83 ± 24.48 mmHg; mean DBP was 86.67 ± 14.04 mmHg; mean hemoglobin was 7.39 ± 2.58 g%; mean total count was 18596.6 ± 8613.41 /cumm; mean Platelet count was 153208; mean INR was 1.32 ± 0.44 ; mean Serum bilirubin was 2.45 ± 3.08 mg/dl; and mean Serum urea was 63.36 ± 25.18 mg/dl. In study by Godara SM et al the mean predialysis blood urea was 129 ± 49 mg/dL and serum creatinine was 6.5 ± 2.5 mg/dL.¹⁰

In this study, mode of delivery was vaginal delivery in most of the patients (66.00%), followed by Emergency LSCS in 26.00% patients. Maternal mortality rate in our study was 20.00% where as 64.00% patients recovered completely, 8% patients got discharged on request, and 8% patients did not follow-up. A relatively higher rate of maternal mortality in developing countries like India is typically associated with the delay on the part of the patients and their attendants. In study by Bekele D et al, 83% women were discharged with improvement, 5 (12%)

died, and 2 (5%) absconded. Of the five maternal deaths, three women had pre-eclampsia-related AKI and two had puerperal sepsis-related AKI.¹¹ Mahesh E et al, reported similar mortality rate as that of our study.¹²

Fetal mortality rate was also remarkably high in our study. In this study, 56.00% fetuses were dead, 38.00% fetuses were alive, 4% got aborted and 2% was undelivered. Out of 19 alive newborn babies, 12 neonates were handed over. Only 7 neonates required NICU admission. In study by Bokhari et al, the rate of fetal mortality was 60%.¹³ Patel et al, reported that fetal mortality rate was 41.7% in their study.¹⁴

In this study, icterus was present in 7 (14.00%) and edema was present in 31 (62.00%) study subjects. Pandey et al, reported that the most common presenting symptom was oliguria seen in 58.53% (24) of patients followed by oedema feet in 53.65% (22) of patients.¹⁵

In this study, in majority of the study subjects (28.00%) etiology was abruption placentae, followed by mild pre-eclampsia and severe anaemia in 24% patients each. Godara SM et al, reported that in majority, the etiology of AKI in the study patients included post-abortion sepsis and hemorrhage in early pregnancy.¹⁰ Bekele D et al, reported that the most common cause of pregnancy-related AKI was pre-eclampsia or eclampsia (31/42, 74%), followed by puerperal sepsis (6/42, 14%).¹¹

There was no significant association of etiological factors with maternal outcome. Mild anaemia, Septic abortion (present in abortus), and Abruptio placentae showed a significant association with fetal outcome; severe pre-eclampsia showed a significant association with NICU admission cause ($p < 0.05$). Mild anaemia ($P < 0.0001$), idiopathic cause ($P < 0.0001$), septic abortion ($P < 0.0001$) and placentae previa ($P < 0.0001$) showed significant association with mode of delivery ($P < 0.05$). Bokhari et al¹³ reported that IUD was the outcome of pregnancy in 60% women. PPH was the cause of obstetric AKI in 41% women in our study group. This result is relatable to the findings of a study conducted by Ali et al that depicted obstetric hemorrhage causing AKI in 58% of patients in their study population.¹⁶

No significant association of initial serum creatinine with maternal outcome ($P = 0.631$) was found but, a significant association of last serum creatinine and % improvement in serum creatinine with maternal outcome existed in our study. There was no significant association of initial and last serum creatinine with fetal outcome, NICU admission, and mode of delivery; however, % improvement in serum creatinine was significantly higher in alive foetuses as compared to aborted, dead and undelivered foetuses. Our study results corroborated with other studies as well. Pandey et al.¹⁵

There was no significant association of serum creatinine with etiology. However, percentage improvement in SCr

was significantly lower in patients with antepartum eclampsia. To our knowledge, there is no study that has investigated the relationship of creatinine and etiology of AKI in pregnant women.

CONCLUSION

Pregnancy-related AKI is a common medical problem, and abruptio placentae, followed by mild pre-eclampsia and severe anemia seems to be the most common etiological factors. The requirement of transfusion and haemodialysis becomes imperative in such patients. No association of etiological factors with maternal outcome was present. Improvement in serum creatinine was highest in live patients and fetuses. % improvement in SCr was lower in patients with antepartum eclampsia. Mild anaemia, septic abortion, and abruptio placentae were significantly associated with fetal outcome. Septic abortion and placenta previa were significantly associated with mode of delivery. Overall, the maternal outcome was good but the fetal outcome was dismal due to AKI in pregnancy.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Mehta RL, Kellum JA, Shah SV. Acute kidney injury network: Report of an initiative to improve outcomes in acute kidney injury. *Crit Care*. 2007;11: R31.
2. Stratta P, Besso L, Canavase C, Grill A, Todros T, Benedetto C, et al. Is pregnancy related acute renal failure a disappearing clinical entity? *Ren Fail*. 1996;18:575-84.
3. Makusidi AM, Liman HM, Yakubu A, Hassan M, Isah MD, Chijioke A. Hemodialysis among pregnancy related acute kidney injury patients: A single center experience in North Western Nigeria. *Indian J Nephrol*. 2016;26:340-2.
4. Okunola OO, Ayodele OE, Adekanle AD. Acute kidney injury requiring hemodialysis in the tropics. *Saudi J Kidney Dis Transpl*. 2012;23(6):1315-9.
5. Prakash J, Tripathi K, Malhotra V, Kumar O, Srivastava PK. Acute renal failure in eastern India. *Nephrol Dial Transplant*. 1995;10:2009-12.
6. Prakash J, Tripathi K, Pandey LK, Gadela SR, Usha. Renal cortical necrosis in pregnancy related acute renal failure. *J Indian Med Assoc*. 1996;94:227-9.
7. Selcuk NY, Tonbul HZ, San A, Odabas AR. Changes in frequency and etiology of acute renal failure in pregnancy (1980-1997). *Ren Fail*. 1998;20:513-7.
8. Altintepe L, Kazim G, Tonbul HZ. Etiology and prognosis in 36 AKI cases related to pregnancy in central Anatolia. *Eur J Gen Med*. 2005;2:110-3.
9. Ibrahim A, Ahmed MM, Kedir S, Bekele D. Clinical profile and outcome of patients with acute kidney injury requiring dialysis - An experience from a haemodialysis unit in a developing country. *BMC Nephrology*. 2016;17(1):1-5.
10. Godara SM, Kute VB, Trivedi HL, Vanikar A V., Shah PR, Gumber MR, et al. Clinical profile and outcome of acute kidney injury related to pregnancy in developing countries: a single-center study from India. *Saudi J Kidney Dis Transpl*. 2014;25(4):906-11
11. Bekele D, Ahmed M, Ibrahim A, Kedir S, Chan G. Profile and outcomes of women with pregnancy-related acute kidney injury requiring dialysis at a center in Ethiopia. *Int J Gynecol Obstet*. 2017;138(2):138-41.
12. Mahesh E, Puri S, Varma V, Madhyastha PR, Bande S, Gurudev KC. Pregnancy-related acute kidney injury: An analysis of 165 cases. *Indian J Nephrol*. 2017;27(2):113.
13. Bokhari SRA, Inayat F, Jabeen M, Sardar Z, Saeed S, Malik AM, et al. Characteristics and Outcome of Obstetric Acute Kidney Injury in Pakistan: A Single-center Prospective Observational Study. *Cureus*. 2018;10(9).
14. Patel M, Sachan R, Radheshyam, Sachan P. Acute renal failure in pregnancy: Tertiary centre experience from north Indian population. *Niger Med J*. 2013;54(3):191.
15. Pandey D, Redkar N. Acute renal failure in pregnancy at a tertiary level hospital in Mumbai: An epidemiological profile. *Ann Int Med Dent Res*. 2016.
16. Ali A, Ali MA, Ali MU, Mohammad S. Hospital outcomes of obstetrical-related acute renal failure in a tertiary care teaching hospital. *Ren Fail*. 2011;33(3):285-90.

Cite this article as: Brahmbhatt J, Devadal K, Patel M. Study of clinical profile and outcome in pregnancy related acute kidney injury. *Int J Reprod Contracept Obstet Gynecol* 2023;12:1849-55.