Zagazig University Medical Journal
www.zumj.journals.ekb.eg
https://doi.org/10.21608/zumj.2024.294428.3426 Volume 30, Issue 5, August 2024
Manuscript ID ZUMJ-2406-3426 (R1)
DOI 10.21608/ZUMJ.2024.294428.3426
ORIGINAL ARTICLE

Prevalence, Clinical Features and Outcome of Post-Partum Acute Kidney Injury: A single Tertiary Center Study

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Submit Date 2024-06-03
Revise Date 2024-06-12
Accept Date 2024-06-23

ABSTRACT

Background: Pregnancy-related acute kidney injury (Pr-AKI) is a significant factor associated with increased maternal and fetal morbidity and mortality in developing countries. The aim of our study was to investigate the clinical data and associated outcomes in patients diagnosed with post-partum AKI (PP-AKI) at our center.

Methods: This prospective study at Zagazig University Hospital, Egypt, from October 1, 2023, to March 1, 2024, included patients with PP-AKI. These women were compared to a matched cohort of healthy pregnant women without pre-existing AKI. Multivariate analysis is used to identify risk factors.

Results: Out of 900 pregnant patients who delivered during the study period, 52 patients developed PP-AKI with prevalence of 5.7%. The majority were multiparous (59.6%), and most delivered by cesarean section (67.3%). The most common cause of AKI is preeclampsia (51.9%), followed by ante-partum hemorrhage (40.4%) and HELLP syndrome (36.5%). Stage 3 AKI (KDIGO classification) was the most prevalent occurring in 23% of cases. Maternal death occurred in 13.5% of cases, while fetal death was 17.3%. Follow- up showed that 42.3% had complete recovery, 30.7% CKD and 13.4% remained dialysis-dependent. Risk factors for AKI included high WBC, low Hb, low platelets, and high bilirubin levels, with p-values of 0.004, <0.0001, and 0.031, respectively. Additionally, nulliparous women had significantly lower odds of developing AKI (0.248, p = 0.017). However, the significance of these risk factors disappeared in multivariate analysis.

Conclusion: PP-AKI is a relatively common pregnancy complication with significant maternal and fetal mortality risks. Preeclampsia and ante-partum hemorrhage are major risk factors. Larger, long-term follow-up studies are recommended to confirm these findings.

Keywords: Pregnancy, Acute kidney injury, Post-Partum AKI

INTRODUCTION

The term "acute kidney injury" (AKI) describes the sudden or rapid loss of kidney function. It is defined as an abrupt and typically reversible decline in glomerular filtration rate [1], leads to elevated nitrogenous waste products, such as urea and creatinine that are typically eliminated by the kidneys [2]. Pregnancy-related acute kidney injury (Pr-AKI) emerges as a significant public health challenge, as it is a major contributor to morbidity and mortality in both mothers and their fetuses [3].

Moreover, AKI is linked to a reduction in kidney function that raises the risk of adverse cardiovascular events, prolongs hospital stays, and increases the likelihood of developing chronic kidney disease (CKD) and end-stage renal disease (ESRD) [4,5]. As a result, early detection and treatment of pregnancy-related AKI are critical and may even save lives. In 1960, the occurrence of Pr-AKI was around 20-40%. This has decreased to less than 10% in recent years (2005), likely due to factors like wider access to safe abortion and advancements in prenatal care such as early detection of pre-eclampsia [6]. Developed countries have experienced a further decrease in Pr-AKI incidence, reaching a range of 1 to 2.8%. This significant improvement likely results from the reduction of septic abortions and advancements in perinatal care [7,8]. Despite the significant decline in developed countries, Pr-AKI remains a significant concern in developing regions, with a prevalence ranging from 4.2% to 15% [9,10]. The unique challenges of treating AKI in pregnancy necessitate a deep understanding of its underlying causes. Only then can healthcare providers formulate appropriate treatment strategies that prioritize both maternal and fetal well-being. The risk of AKI extends beyond pregnancy itself, as women can also develop this complication after giving birth. PP-AKI can develop as a consequence of severe pregnancy complications such as pre-eclampsia, a condition marked by high blood pressure and potential organ damage; HELLP syndrome hemolysis (the breakdown of red blood cells; elevated liver enzymes, indicating liver dysfunction); and in rare cases, atypical hemolytic-uremic syndrome (aHUS) may also be a contributing factor. Notably, the risk of post-partum AKI (PP-AKI) is particularly high when these complications arise before delivery and persist untreated [12,13]. The severest form of PP-AKI requiring dialysis is often associated with significant mortality and morbidity [14]. Accordingly, early detection and identifying the risk factors are pivotal in managing and ameliorating the outcomes of PP-AKI.

To address the potential link between PP-AKI and severe pregnancy complications, we investigated PP-AKI prevalence, clinical features, risk factors, and maternal and fetal outcomes in our hospital over six months. This data will inform future clinical guidance.

PATIENTS AND METHODS

This prospective observational cohort study involved patients who developed PP-AKI from October 2023 to April 2024. Each patient provided informed written consent to participate in this research, which was authorized by the university hospital's institutional ethics board (ZU-IRB #11099). Every procedure followed the Helsinki Declaration. The study was conducted at the nephrology unit of internal medicine department and gynecology and obstetrics department, at Zagazig University Hospitals. We included patients with PP-AKI. All adult females >18 years old age who developed PP-AKI during the study period are included in the study. Moreover, a group of 25 healthy pregnant patients with no PP- AKI were included as a control group. The study included all eligible patients identified during the 6-month study period (comprehensive sample).

Operation design:

Informed consent was taken from all the participants, and all patients were subjective to full clinical history, full clinical examination and Laboratory investigations (Renal function panel, CBC, CRP, Immunological screening (e.g ANA, Anti DsDNA, C3, C4), coagulation profile and urine microscopy and 24 hours urine protein. Histopathology of renal biopsy tissue in selected cases. Kidney biopsy was indicated when the laboratory evaluation for AKI is non-diagnostic, and a definitive diagnosis will help to facilitate appropriate treatment outweighing the risks of biopsy. Using a prospective approach, we identified patients who developed PP-AKI based on the following criteria as defined by the Kidney Disease: Improving Global Outcomes (KDIGO) 2012 guidelines [15]: Rapid decline in kidney function within 48 hours postpartum, meeting the following key diagnostic criteria: (1) AKI with an onset occurring immediately following delivery and for up to 6 weeks postpartum; (2) Serum creatinine increase Absolute value exceeding 0.3 mg/dL (>26.5 µmol/L) ; (3) at least a 50% increase in serum creatinine (1.5 times baseline); and (4) normal renal function throughout pregnancy and before delivery. AKI was staged into 3 stages according to the KDIGO 2012 criteria [15]. According to the definition of AKI, PP-AKI was recorded within 48 hours post delivery. Preeclampsia was defined as the first diagnosis of

blood pressure over 140/90 mmHg with 2+ proteinuria on a dipstick after 20 weeks of gestation. Severe preeclampsia is diagnosed when a pregnant woman experiences both high blood pressure (systolic blood pressure above 160 mmHg and diastolic blood pressure above 110 mmHg) and signs of significant protein in the urine (at least 5 grams per liter or a high reading on a dipstick test). Additionally, symptoms like headaches, vision problems, or upper abdominal pain may indicate organ involvement and suggest a more serious case. Eclampsia is a severe complication of preeclampsia, characterized by the development of new-onset grand mal seizures [16]. HELLP syndrome is characterized by three key findings: a low platelet count (less than 100,000 per microliter), elevated liver enzymes (specifically, aspartate aminotransferase or AST greater than 70 units per liter), and signs of hemolysis. The definition of sepsis was established by the American College of Chest Physicians [17]. According to the World Health Organization (WHO), puerperal sepsis is an infection of the genital tract that manifests after membrane rupture or labor and can occur anytime within the first 42 days postpartum. The diagnosis is based on the presence of at least two of the following criteria: pelvic pain, fever, abnormal discharge, or slow uterine shrinking [18]. Significant blood loss following childbirth, exceeding 500 milliliters (mL) after vaginal delivery or 1000 mL after cesarean section, was classified as postpartum hemorrhage (PPH) [18]. Primary outcomes include maternal and fetal mortality. Secondary outcomes include: 1- Complete renal recovery was defined as serum creatinine levels returning to less than 1.4 mg/dL at discharge or during the 12-week follow-up period. 2- Partial renal recovery was defined as serum creatinine levels remaining at or above 1.4 mg/dL at discharge or on follow-up of 12 weeks 3- Cases progressed to CKD. 4- Patients progressed to ESKD. Demographic and laboratory data are collected and entered in an excel spread sheet for statistical analysis.

Statistical analysis:

Data was entered into a Microsoft Excel spreadsheet to ensure accuracy and facilitate initial organization; the statistical software package SPSS version 21.0 was used to perform all data analysis. Descriptive statistical analysis was performed to summarize the data set. Categorical variables were analyzed by calculating frequencies and proportions. Quantitative variables were analyzed using means and standard deviations (SD). Comparison between groups using independent T test between non-categorized data and Chi-square in categorized data. Binary logistic regression analysis is used to define odds of risk factors using univariate and multivariate analysis value is ≤0.05 is significant.

RESULTS

Among 900 pregnant females who delivered in the same study period, 52 patients developed PP-AKI with prevalence rate of 5.7%. Their age was 25.7±5.2 years. 40.4% was nulliparous, while 59.6% was multiparous. 67.3% were delivered by cesarean section and 32.7% delivered normally. The majority of AKI stages were stage 3 in 44.2% of cases followed by stage 1 in 36.5% and stage 2 in 19.2%. Pre-eclampsia was the most prevalent cause of PP-AKI (51.9% of cases), followed by antepartum hemorrhage in 40.4%, combined causes were present in 75%, figure 2.

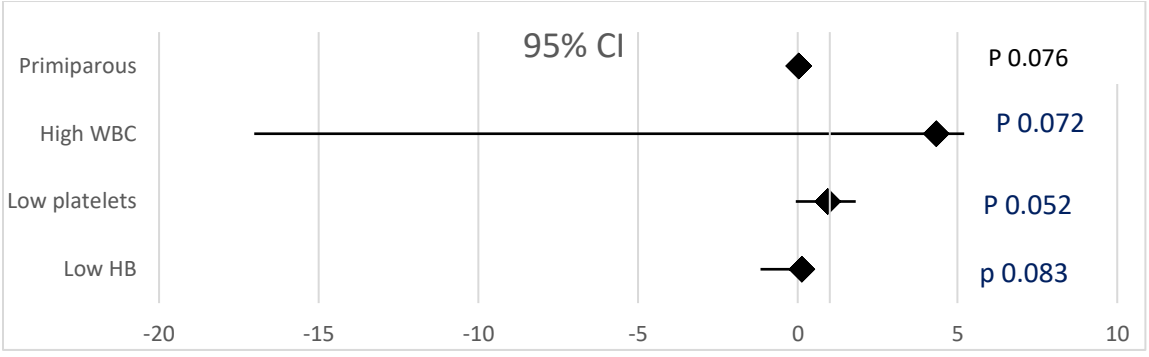

Figure 1: Forest plot of risk factors of post-partum AKI

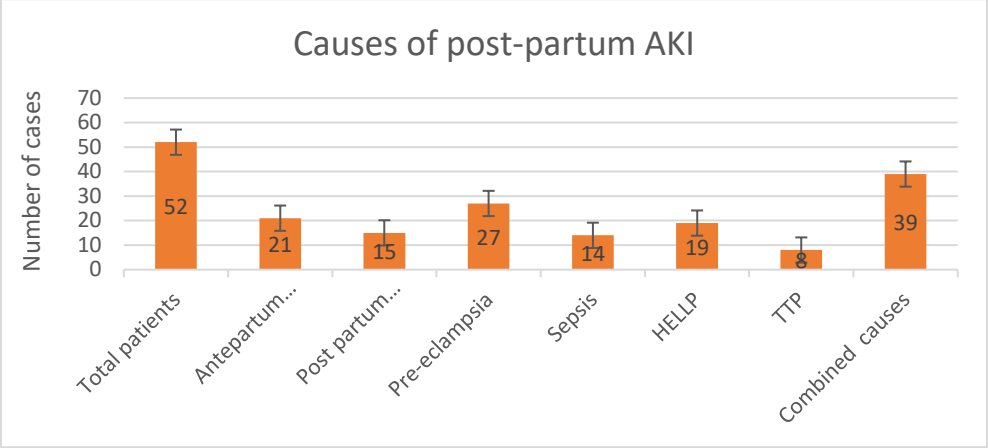

Figure 2: Causes of PP-AKI

The major clinical presentations were anuria in 30.8% of cases followed by shortness of breath (SOB) in 17.3%. Kidney biopsy has been done in 6 cases, 4 biopsies showed acute tubular necrosis (ATN), while 2 cases showed thrombotic microangiopathy (TMA), figure 3,4.

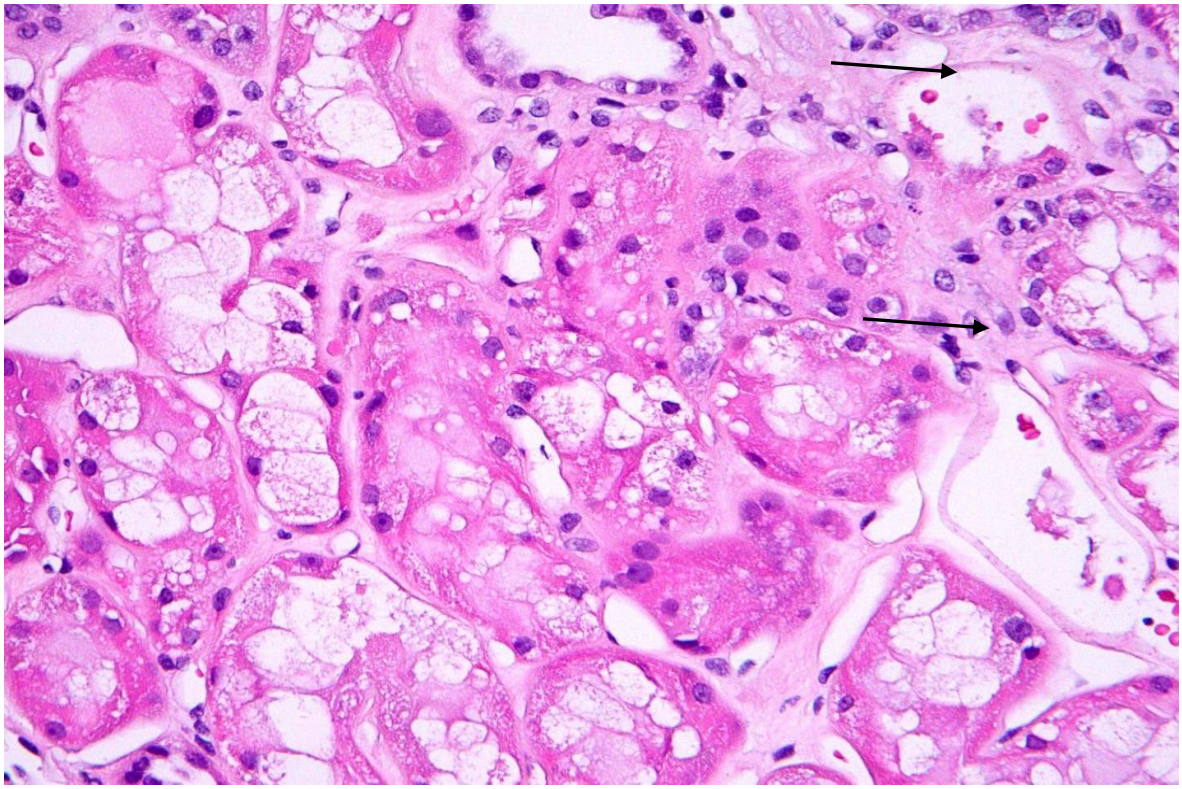

Figure 3: Kidney Biopsy showed acute tubular necrosis (ATN) (black arrows)

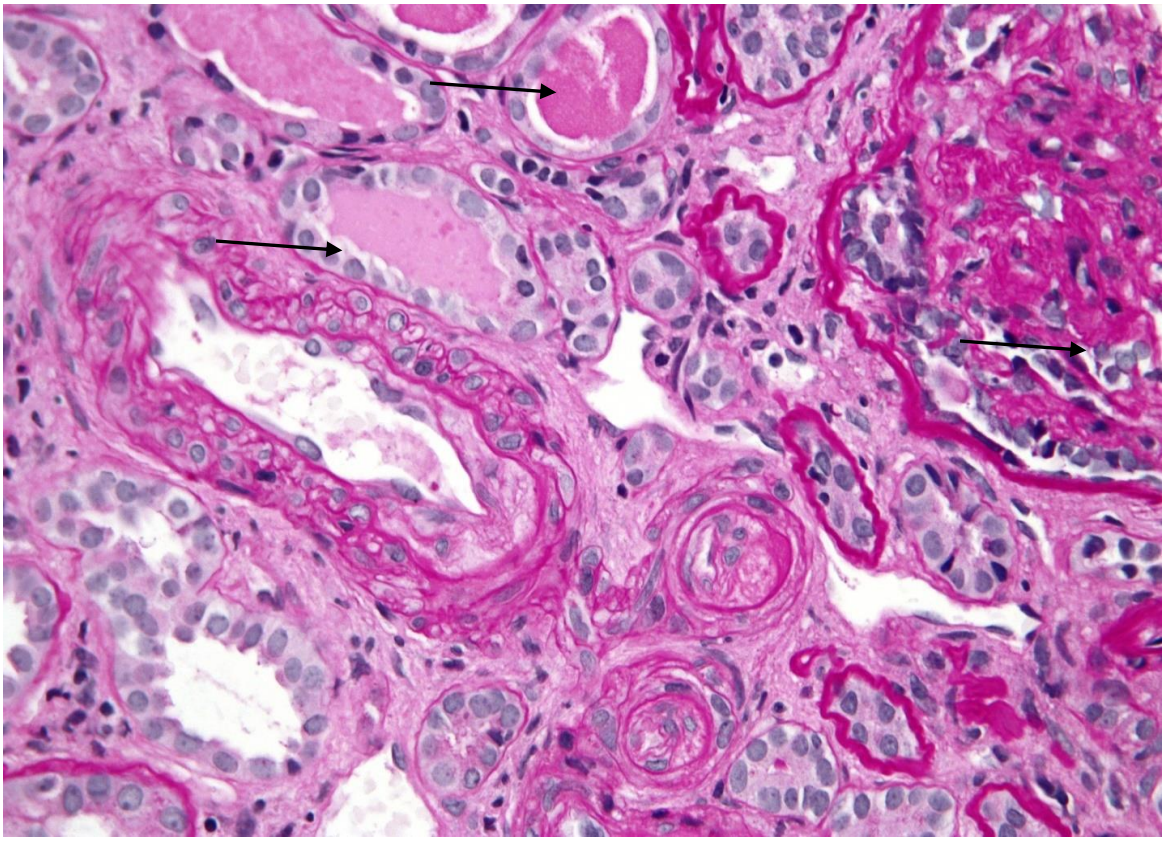

Figure 4: Kidney Biopsy showed Thrombotic microangiopathy (TMA) (black Arrows)

Primary outcomes: Maternal mortality occurred in 7 cases (13.4%) and fetal mortality in 9 cases (13.4%), figure 5. TTP is significantly prevalent in expired group compared to survived group (P 0.000). However, Pre-eclampsia is statistically prevalent in survived group (p 0.034) suggested that it does not have a significant role in patients' mortality. Furthermore, AKI stage 3 was the prevalent stage in expired group, however, it was statistically insignificant (p0.068). HB and platelets were statistically lower in expired group (P= 0.000 and 0.001 respectively), Figure 1. While Creatinine and bilirubin were statistically higher in expired group (p =0.027 and 0.000 respectively). Refer to table 4.

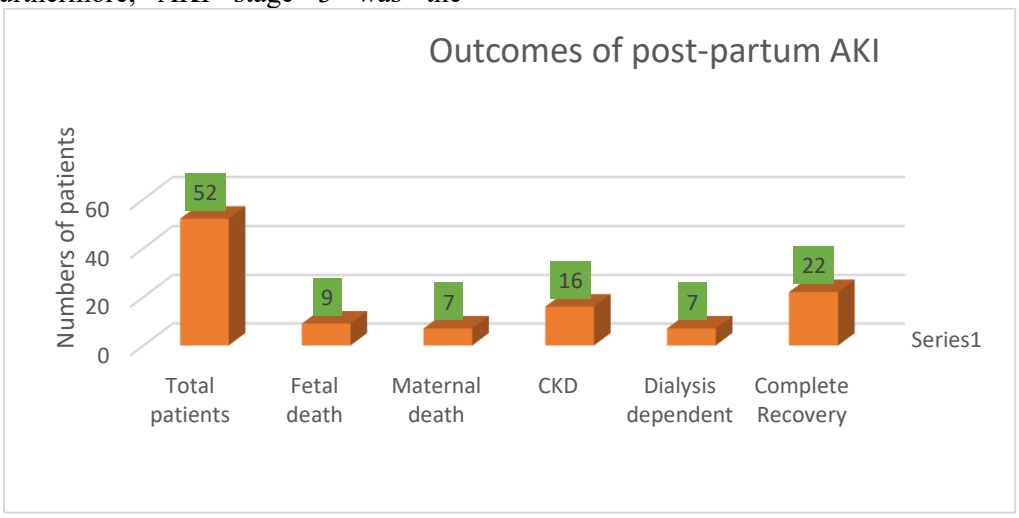

Figure 5: Outcomes of post-partum AKI

Table 1: clinical and demographic characteristics of AKI population

Parameters	Number (52) (%)
	25.7±5.2
Pregnancy status **Nulliparous** **Multiparous**	 21 (40.4) 31 (59.6)
Delivery status **Normal delivery** **Cesarean section**	 17 (32.7) 35(67.3)
Clinical presentation: **SOB** **Anuria** **Fever** **Hypotension** **Hypertension** **Nausea, Vomiting** **Headache** **Abdominal pain**	 9 (17.3) 16 (30.8) 5 (9.6) 5 (9.3) 1 (1.9) 7 (13.5) 6 (11.5) 1 (1.9)
Ante-partum Hemorrhage	21 (40.4)
Post partum Hemorrhage	15 (28.8)
Pre-eclampsia	27(51.9)
Sepsis	14 (26.9)
HELLP syndrome	19 (36.5)
Thrombotic thrombocytopenic purpura (TTP)	8 (15.4)
Combined causes	39 (75)
Fetal death	9 (17.3)
Maternal death	7 (13.4)
Chronic kidney disease (CKD)	16 (30.7)
Dialysis dependent	7 (13.4)
Complete recovery	22 (42.3)
AKI stages: **Stage 1** **Stage 2** **Stage 3**	 19 (36.5) 10 (19.2) 23 (44.2)

HELLP (Syndrome of hemolysis, elevated liver enzymes and low platelet), AKI (Acute kidney injury)

Comparing to control group, there were significant low Hemoglobin level and platelets, p 0.006 and <0.0001 respectively, while there were significant high WBC, 24 hours urinary protein and serum bilirubin level (p <0.0001, <0.0001 and 0.037 respectively), no significant differences were found regarding serum albumin level (p 0.204), age (P 0.528), delivery mood (P0.528) and pregnancy status (P 0.113), table 2. Comparison between AKI stages showed no significant differences between AKI stages regarding age, delivery mood, pregnancy status causes of AKI and primary and secondary outcomes.

Table 2: Comparison between post-partum AKI and control groups.

	Control group N=24 (%)	AKI group N=52 (%)	P
Age	26.5±4.6	25.7±5.2	0.528
Delivery status **Normal**	 9(37.5)	 17 (32.6)	 0.681

Cesarean	15 (62.5)	35(67.3)	
Pregnancy status: **Nulliparous** **Multiparous**	 14 (58.3) 10 (41.6)	 21 (40.3) 31 (59.6)	 0.113
Preeclampsia	0	27 (51.9)	0.0001
Ante partum hemorrhage	0	15 (28.8)	0.003
Post partum hemorrhage	0	24 (46.1)	0.000
HEELP syndrome	0	19 (36.5)	0.001
Sepsis	0	14 (26.9)	0.005
TTP	0	8 (15.3)	0.042
Death	0	7 (13.4)	0.059
Fetal death	0	9 (17.3)	0.030
Diabetes	10 (41.6)	10 (19.2)	0.039
Hypertension	6 (25)	8 (15.3)	0.315
WBC9/l	6.4±1.5	11.0±5.6	0.000
Hemoglobin (HB) gm/dl	9.4±0.9	8.5±1.3	0.006
Platelets×9/l	254.5±89.2	126.1±109.5	0.000
Creatinine mg/dl	0.62±1.5	5.20.62±1.52.8	0.000
24h urine protein/mg	158.1±78.5	1286.1±678.2	0.000
Albumin gm/dl	4.1±0.3	3.9±0.5	0.204
Bilirubin mg/dl	1.0±0.1	1.8±1.8	0.037

TTP (Thrombotic thrombocytopenic purpura), HELLP (Syndrome of hemolysis, elevated liver enzymes and low platelet)

Refer to table 3. In univariate analysis, risk factors for AKI included high WBC, low Hb, low platelets, and high bilirubin levels, with p-values of 0.004, <0.0001, and 0.031, respectively. Additionally, nulliparous women had significantly lower odds of developing AKI (0.248, p = 0.017).

Table 3: Comparison between AKI stages

	AKI stage 1 N=19 (%)	**AKI stage 2 10 (%)**	**AKI stage 3 23 (%)**	**p**
Age	24.6±5.3	26.2±6.1	26.4±4.7	0.508
Pregnancy status **Primiparous** **Multiparous**	 11(57.8) 8 (42.1)	 4(40) 6 (60)	 6 (26.0) 17 (73.9)	 0.112
Delivery mood: **Normal** **Caesarean section**	 7 (36.8) 12 (63.1)	 3 (30) 7 (70)	 7 (30.4) 16 (69.5)	 0.889
preeclampsia	12 (63.1)	4 (40)	11 (47.8)	0.431
Antepartum Hemorrhage	8 (41.1)	3 (30)	10 (43.4)	0.755
Post partum Hemorrhage	4 (21.0)	5 (50)	6 (26.0)	0.243
HELLP syndrome	8 (41.1)	2 (20)	9 (39.1)	0.472
Sepsis	4 (21.0)	5 (50)	5 (21.7)	0.187
TTP	1 (5.2)	1 (10)	6 (26.0)	0.154
Death	1 (5.2)	1 (10)	5 (21.7)	0.279
Fetal death	4 (21.0)	2 (20)	3 (13.0)	0.768

TTP (Thrombotic thrombocytopenic purpura), HELLP (Syndrome of hemolysis, elevated liver enzymes and low platelet), AKI (Acute kidney injury)

However, the significance of these risk factors disappeared in multivariate analysis, Table 5. AKI stage 3 was the most important risk factor for mortality in univariate analysis (OR 2.916, 95% CI 1.133-4.507, P 0.026),

Table 4: Comparison between survived and expired groups

	Survived N=69 (%)	**Expired patients N=7 (%)**	
Age	26.2±5.1	23.1±2.9	0.116
AKI	45 (65.2)	7 (100)	0.059
Delivery status **Normal** **Cesarean**	 24 (34.7) 45 (65.2)	 2 (28.5) 5 (71.4)	0.741
Pregnancy status: **Nulliparous** **Multiparous**	 32 (46.3) 34 (49.2)	 3 (42.8) 4 (57.1)	0.859
Preeclampsia	24 (34.7)	0	0.039
Ante partum hemorrhage	17 (24.6)	4 (57.1)	0.067
Post partum hemorrhage	15 (21.7)	0	0.169
HELLP syndrome	18 (26)	1 (14.2)	0.492
Sepsis	14 (20.2)	0	0.187
TTP	1 (1.4)	7 (100)	0.000
AKI stages: **Stage 1** **Stage 2** **Stage 3**	 18 (26) 9 (13) 18 (26)	 1(14.2) 1 (14.) 5 (71.4)	0.068
Diabetes	17 (24.6)	3	0.297
Hypertension	14 (20.2)	0	0.187
WBC^9/l	9.8±5.3	7.3±1.7	0.222
Hemoglobin (HB) gm/dl	9.0±1.1	6.8±0.7	0.000
Platelets×^{9}/l	180.9±115.9	26.1±7.3	0.001
Creatinine mg/dl	3.5±3.1	6.2±2.4	0.027
24h urine protein/mg	899.8±796.4	1226.4±337.9	0.288
Albumin gm/dl	4.0±0.51	3.9±0.58	0.550
Bilirubin mg/dl	1.9±0.9	4.8±2.2	0.000

TTP (Thrombotic thrombocytopenic purpura), HELLP (Syndrome of hemolysis, elevated liver enzymes and low platelet)

Secondary outcomes: For 3 months follow up period, 22 (44.2%) patients showed complete recovery, 16 patients (30.7%) developed CKD, while 7 (13.4%) patients progressed to ESRD and continue on kidney replacement therapy. For more details, refer to table 1, figure 5.

Table 5: Univariate and multivariate analysis for Risk factors for AKI

	Univariate analysis			**Multivariate analysis**		
	odds	**95%CI**	**p**	**odds**	**95%CI**	**P**
Primiparous	0.248	0.079-0.779	0.017	0.029	0.001-0.14	0.076
High WBC	1.562	1.155-2.211	0.004	4.330	0.878-21.35	0.0720.
Low platelets	0.983	0.974-0.992	0.000	0.936	0.876-1.000	0.052
Low HB	0.545	0.314-0.945	0.031	0.131	0.013-1.30	0.083

However, it became insignificant on multivariate analysis (OR 0.469, 95% CI 0.086-2.402, P =0.354). Table 6

Table 6: Univariate and multivariate analysis for Risk factors for motility

	Univariate analysis			Multivariate analysis		
	odds	95%CI	p	odds	95%CI	P
AKI stage 3	2.916	1.133-4.507	0.026	0.469	0.086-2.402	0.354
Ante-partum Hemorrhage	4.078	0.828-20.079	0.084	0.440	0.043-4.457	0.487

DISCUSSION

PP-AKI, a rare but critical postpartum complication, can be life-threatening. However, timely treatment improves outcomes. This prospective cohort study was designed to focus on pregnant patients who developed PP-AKI to evaluate the characteristics and possible risk factors of this complication. In this work, the mean age of women included in the study was 25.7±5.2 years. Multiparous were the prevalent in (59.6% of cases and nulliparous were 40.4%. In a previous comparable recent study, which has been done by Yusupha Sanyang and his colleges [19], the mean age of their participant was 29.2±3.3 and 78.3% were multiparous while 21.7% were nulliparous. The prevalence rate of postpartum AKI in the study population was 5.7%. In a similar study from India [20], the prevalence was 3.26%. While the global incidence of PR-AKI appears to be declining [21], recent trends in the United States and Canada suggest a potential increase in cases within these developed nations [22]. A French study of 59,302 women admitted to the ICU for postpartum complications found that among the 182 women who developed AKI, 54.1% progressed to stage 3 AKI and 70.3% required ICU treatment. Despite the severity of these cases, there were no reported deaths in this study. This suggests that while PP-AKI remains a significant concern, advances in critical care management may be improving survival rates in developed countries. Our study identified hypertensive disorders of pregnancy as the leading cause of PP-AKI, specifically including pre-eclampsia in 51.9% of cases followed by antenatal hemorrhage in 40.4% and HELLP syndrome in 36.5%. Research suggests a geographic disparity in the leading causes of PP-AKI. Among women in high-income countries, hypertensive disorders of pregnancy, like pre-eclampsia, are well-documented risk factor [22]. In contrast, infection and postpartum hemorrhage are the primary causes of AKI in many low- and middle-income regions [24,25]. This difference may be attributed to several factors, including variations in access to prenatal care, underlying health disparities between populations, and the increased prevalence of infectious diseases in developing countries. Previous studies conducted in China have consistently identified pregnancy-induced hypertension (PIH) and postpartum hemorrhage (PPH) as the main predisposing factors behind pregnancy-related AKI [26]. The exact causes and development mechanisms of PP-AKI remain poorly understood. Pregnancy is characterized by a significant increase in maternal blood flow, with cardiac output rising by 60% to 80% immediately after delivery. However, this rapid increase is followed by an equally rapid decrease within 10 minutes, returning to near-normal levels within an hour postpartum. This dramatic shift in hemodynamics during the peripartum period is suspected to play a role in the development of PP-AKI, but the precise mechanisms remain unclear [27-28]. Several factors are thought to contribute to the development of PP-AKI. These include severe hypovolemia, often caused by severe post-partum hemorrhage, and pregnancy related coagulopathy. Additionally, elevated levels of atrial natriuretic peptide (ANP) and brain natriuretic peptide (BNP) observed postpartum may play a role. Both ANP and BNP possess potent diuretic effects, potentially contributing to diuresis seen in the early post-partum period [28]. In our study, ATN was the prevalent in the biopsy findings in 5 cases out of 7 cases underwent kidney biopsy followed in TMA in the other 2 cases. Our findings align with previous research [29], where ATN was the most prevalent histological feature observed in biopsies (33%), whereas other research by Sani S. et al. reported a higher prevalence of TMA (54%), followed by ATN [9]. In our study, there were relatively fair outcomes, with complete renal recovery seen in 44.2% of cases, while 30.7% showed partial renal recovery and only 13.4% of cases continue on Kidney replacement therapy (KRT). Maternal death happened in 7 cases (13.4%) and fetal death in 9 cases 17.3%. Our study findings regarding

outcomes in pregnancy-related AKI seem to differ from some prior reports suggesting positive results with treatment [26,30]. A study from India [20] reported encouraging results, with 81.1% of patients achieving complete renal function recovery. Only a small fraction (2.7% each) experienced partial recovery or remained dialysis dependent. Notably, no deaths were reported. However, in a study done by Naresh Pahwa, maternal mortality has occurred in 18.5% of patients which is higher than our rate [31]. The difference between our study outcomes and previous studies raises the red flag for early detection and intense intervention to ameliorate those outcomes. In our study, the relative risk factors which have increase the rate of PP-AKI were pregnancy in multiparous women, pregnant women with low hemoglobin level, low platelets level and high white blood cells (WBC) and high bilirubin level. Those laboratory abnormalities may reflect early detection of sepsis, thrombotic microangiopathy and HELLP syndrome and should be considered while evaluating pregnant patients. However, the effect of those factors declined in multivariate analysis but they still to be considered during patient evaluation.

Limitations:

Our study has some potential limitations. First, the relatively small size of the control group so the comparable results may show some bias. Second, the nature of the control group is lacking the same potential risk factors predisposing to AKI (Pre-eclampsia and ante-partum hemorrhage) and only included healthy pregnant women which might affect the outcomes of the comparable results. Third, the follow up period is short as longer follow up period is required for more accurate recording the clinical outcomes. Fourth, some co-morbidities and history of medications used are lacking which may increase the strength of the study in respect of risk factors.

CONCLUSIONS

Post-partum AKI is a relatively common pregnancy complication with significant poor maternal and fetal outcomes. Preeclampsia and ante-partum hemorrhage are major risk factors. Early identification of patients with these conditions is crucial, allowing for the prompt implementation of intensified care and potentially improving outcomes. Strategies such as enhancing antenatal care, preventing PIH, and promoting sepsis prevention awareness within healthcare institutions hold promise in reducing the prevalence of PP-AKI. Larger, long-term follow-up studies are recommended to confirm these findings.

Declaration of interest and Funding information: The authors report no conflicts of interest.
Authors' contributions: A.A. and E.E. formulated and designed the study; T.G. was a major contributor in writing the manuscript; A.A and T.G. analyzed the data; E.E. and A.A. revised the paper. All authors read and approved the final manuscript.

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To Cite:

Elsayed, I., Ghonimi, T., Abd El-Hameed, A. Prevalence, Clinical Features and Outcome of Post-Partum Acute Kidney Injury: A single Tertiary Center Study. *Zagazig University Medical Journal*, 2024; (1641-1652): -. doi: 10.21608/zumj.2024.294428.3426