Original Article

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Risk Factor Evaluation in Pregnancy Related Acute Kidney Injury

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Abstract

Background: Pregnancy Related Acute Kidney Injury (PRAKI) is common in developing countries. It often complicates the normal pregnancy and puerperal period with a significant proportion of morbidity and mortality related to it. Identification and early intervention of the risk factors associated to it can prevent this life-threatening complication. To evaluate the risk factors associated with PRAKI in a tertiary level hospital.

Materials and methods: This case control study was carried out in Dhaka Medical College Hospital for a period of one year following approval of ethical review committee. A total of 50 subjects were enrolled as cases who had PRAKI and 67 apparently healthy pregnant women were enrolled as controls. All patients underwent detailed history taking, physical examination and hematological and biochemical parameter. Data collection was done through a structured questionnaire and collected data were analyzed using the statistical software SPSS 23.

Results: Among cases, the frequency of past obstetric history were Pregnancy related hypertensive disorder (22%) abortion (8%) intra uterine death/stillbirth (6%) and ectopic pregnancy (2%). Among the pregnancy related risk factor pre-eclampsia/eclampsia PIH, sepsis, PPH, gestational diabetes mellitus, urinary tract infection, APH were significantly higher in compared to control (All p< 0.001 except the later one p value of which 0.003). Multivariate logistic regression analysis showed pre-eclampsia/ eclampsia [OR 1.104, 95% CI (1.001-1.998) p=0.049] PIH [OR 1.119, 95% CI (1.023-1.890) p=0.048] sepsis [OR 1.873, 95% CI (1.032-2.998), p=0.039] post-partum hemorrhage [OR 2.223, 95% CI (1.921-2.990) p=0.038], increased total leucocyte count [OR 1.338, 95% CI (1.015-3.476), p=0.049] decreased platelet count [OR .880, 95% CI (.682-.997) p p=0.016], increased D-dimer [OR 4.564, 95% CI (2.321-12.881), p=0.001] and prolonged APTT [OR 1.899, 95% CI (1.023-2.442) p=0.003] were independently associated with development of PRAKI.

Conclusion: Pregnancy related acute kidney injury (PRAKI) is a major health problem especially in the middle-income families and carries very high mortality and morbidity. Improved antenatal care and obstetric care reduce PRAKI in developed country. History of a previous hypertensive disorder of pregnancy, eclampsia, sepsis was the risk factors for development of AKI. Improvement of pregnancy care and early and appropriate Nephrological care may reduce PRAKI and mortality.

Key words: Acute kidney injury; Creatinine; Preeclampsia; Pregnancy; PRAKI (Pregnancy Related Acute Kidney Injury); Renal insufficiency.

Introduction

Pregnancy-related acute kidney injury (PRAKI) remains a significant cause of maternal and fetal morbidity as well as mortality. Recent data from Canada and the US show pregnancy-related AKI to be increasing between 2003 to 2010. In Canada, rates have risen from 1.6 to 2.3 per 10,000 deliveries and in the US from 2.3 to 4.5 per 10,000 deliveries.[1,2] It remains a common cause for requiring dialysis in low and middle-income countries and is associated with high maternal and neonatal morbidity and mortality rates. The incidence of pregnancy-related Acute Kidney Injury (AKI) has reduced over recent decades in India because of improvements in reproductive health care system and decreased incidence of postabortion sepsis.[2,3] In last 20 years, a study from Pakistan reported that pregnancy- related AKI is increasing due to the lack of adequate primary health-care facilities and immoral practice of induced abortion There is a limited understanding of underlying risk factors in these settings to enable appropriate triage and targeting of scarce resources.[1,4,5]

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PRAKI is also increasing due to level of obesity, co-morbidity like hypertension, diabetes and increasing maternal age. Several study from India shows PRAKI to be decreased between 1960 to 2019.Decreased rate is being reported from 17-43% due to improve socio-economic setting.[1,4]

PRAKI is improving in high income setting due to legislation of pregnancy termination and improvement of antenatal care.

Normal pregnancy accompanies several physical and physiological changes. Understanding these changes is crucial in evaluating risk factors of AKI in pregnant women. Initially, the size of the kidney increases by 1.0–1.5 cm in length. Primarily, the change is in the collecting system. This is the result of tissue hypertrophy, renal vascular and interstitial space expansion. The renal pelvis, calyces, and ureters are also dilated, expanding to the pelvic brim. It is postulated that nearly 90% of normal pregnant women develop physiological hydronephrosis. Renal plasma flow elevates by 45%–70% and can reach 85% in the second trimester. After conception, the Glomerular Filtration rate (GFR) increases continuously and the increment can be up to 25% at the 4th week of gestation. It reaches a peak at 65% increase from the baseline at around the 13th week of gestation. This consequently leads to hyperfiltration state, bringing other physiological changes such as an increase in uric acid clearance, increased excretion of calcium, glycosuria, increased filtration of amino acids, and increased creatinine clearance and decreased in BUN and serum creatinine, sodium, magnesium, bicarbonate level.[6-10]

Pregnancy related acute kidney injury has two incidence peaks: one in the first trimester, caused by infections (Usually involving septic abortion), and the other in later part of pregnancy, due to late obstetric complications.[4,5,11] As in the general population, the causes of AKI in pregnant women are divided into 3 groups: pre-renal, renal, and post-renal. The prerenal causes are more common in the earlier stage of pregnancy due to hyperemesis gravidarum or acute tubular necrosis in the context of septic abortion but also due to the later stage of pregnancy cause like as obstetric hemorrhage: antepartum (Placenta previa, placental abruption, placenta accreta) post-partum (Atony, trauma, uterine rupture). In the later stages, AKI development is more frequent and usually associated with a renal cause like as preeclampsia, acute fatty liver of pregnancy, Hemolytic Uremic Syndrome (HUS) HELLP syndrome (Hemolysis, elevated liver enzymes and low platelet count) and sepsis.[1,2,7,9,12] In the United State PRAKI rates risen from 2.3 to 4.5 per 10000 delivery in any time of gestational period and 0.48% to 2.17% per 10000 delivery in postpartum period.[13] In India several study shows AKI is common in later trimester and among their pre-eclampsia is 35% and puerperal sepsis is 25%
In Bangladesh an observational study state that septicemia (43%) and eclampsia (19%) were the leading cause of PRAKI, others causes were PPH, APH, ruptured ectopic pregnancy.[14] But the elaborated study on risk factor evaluation of PRAKI is scarce, thus the current study has been designed.

Materials and methods

This case-control study was carried out in the Department of Medicine in collaboration with Nephrology and Obstetrics & Gynaecology at Dhaka Medical College Hospital after getting approval of this protocol and then formal ethical clearance was taken from the ethical review committee of Dhaka medical college for conducting the study within 12 months period during April 2020 to March 2021. Total 117 pregnant women or women on puerperium who matched the inclusion and exclusion criteria were approached for inclusion in the study. They were categorized into case and control group. Fifty (50) Patients diagnosed to have pregnancy related acute kidney injury were categorized as cases and sixty-seven (67) pregnant subjects with normal renal function was categorized as control group after excluding chronic kidney disease or pre-existing renal disease. Patients/attendance was briefed about the study and a written informed consent was taken from each of the participants. A standardized case report form was formulated to document demographic data, clinical symptoms and signs, co-morbidity including hypertension, gestational diabetes, urinary tract infection, sepsis, connective tissue disease, acute diarrheal disease, each patient at the time of presentation. All patients underwent detailed physical examination with special emphasis on acute kidney injury and

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preexisting co-morbidity. Detailed obstetric history was taken regarding the number of pregnancies, outcome of previous pregnancies, abortion, ectopic pregnancy, pre-eclampsia/eclampsia, abruption placentae, placenta previa, intra uterine death, ante and postpartum hemorrhage. Blood sample were collected from study subjects to estimate blood hemoglobin, total leucocyte count, Platelet count, serum creatinine, AST, ALT, total bilirubin, D-dimer, PT, APTT. With all aseptic precaution 6 ml of venous blood was drawn from ante cubital vein by a disposable plastic syringe and each of 2 ml was delivered immediately into an EDTA tube (For plasma D-dimer, PT, APTT) and a plain tube without anticoagulant (For serum creatinine, ALT, AST, total bilirubin). Plasma was collected from the remaining portion of whole blood in the citrate tube by centrifuging at 3000 rmp for 5 minutes. All sample were stored at -20^0 C except for D-dimer, PT, APTT which were immediately processed. All the hematological and biochemical tests were performed at the Department of Clinical Pathology, DMC and Department of Hematology and BMT, DMC. Estimation of blood hemoglobin, total leucocyte count, platelet count by automated hematological analyzer, estimation of serum creatinine by modified kinetic Jaffe reaction, estimation of ALT and AST by kinetic method, estimation of serum total bilirubin by chemical method, estimation of D-dimer by latex agglutination assay, estimation of PT by step dilution method, estimation of APTT by thrombin clotting technique.

PRAKI is defined as AKI diagnosed anytime during pregnancy or postpartum phase (First 6 weeks post- delivery)

AKI in pregnancy: A creatinine level of ≥1 mg/dl (≥88 µmol/l) within 48 hours or a rapid rise (By definition in 48 hours) of 0.5 mg/dl (44 µmol/l) above baseline should be investigated. (KDIGO clinical practice guide line for AKI 2012)

Acute kidney injury in postpartum phase: Increase in S. Cr by ≥0.3 mg/dl (≥26.5 µmol/l) within 48 hours or Increase in S.Cr 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.

All the above information were recorded in a data collection form consisting of relevant questionnaire. After completion, data analysis was done by SPSS 23.

Table I Socio-demographic characteristics of the study population (n=117)

Variables	Case (n=50) n (%)	Control (n=67) n (%)	p value
Age group (Years)			
18-20	2 (4)	4 (6)	
21-30	8 (16)	21 (31.3)	0.125[c]
31-40	40 (80)	42 (62.7)	
Mean±SD	33.5±5.33		
(19-40)	31.43±6.51		
(18-40)	0.062[a]		
Socio-economic class			
Poor	17 (34)	24 (35.8)	
Middle income group	21 (42)	36 (53.7)	0.130[b]
Rich	12 (24)	7 (10.4)	

During the period of April 2020 to March 2021among the study participants (n=117) mean age of the cases and controls was 33.5±5.33 (SD) years and 31.43±6.51 (SD) years respectively, p>0.062. Majority of the patients were in age group 31-40 years (80% in cases and 62.7% in controls). Mean age of the patients was statistically similar (p=0.125). Majority of the respondents belonged to middle income group (42% in cases and 53.7% in controls). No significance difference (p=0.130) was observed between two groups.

Table II Anthropometric and clinical profile of the study population (n=117)

Variable	Case (n = 50) mean ± SD	Control (n = 67) mean ± SD	p-value
BMI (kg/m^2)	28.23±3.02 (20-36.5)	25.08±3.47 (20-32)	<0.001[a]
Blood pressure			
Systolic (mm of Hg)	153.02±14.45 (110-175)	123.13±11.63 (100-130)	<0.001[a]
Diastolic (mm of Hg)	104.40±8.30 (100-120)	80.97±11.65 (60-90)	<0.001[a]

Mean weight, BMI, systolic and Diastolic Blood pressure were statiscally higher in cases (p<0.001) then control.

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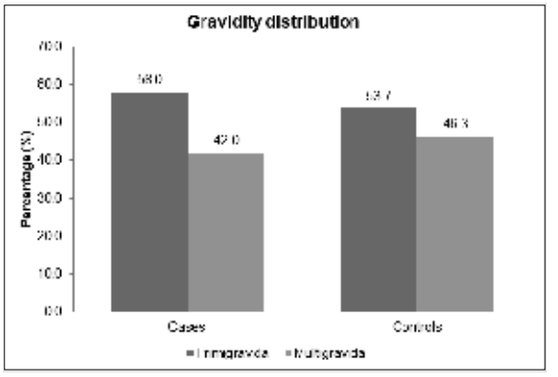

Figure 1 Distribution of the respondents by gravidity (n=117)

In cases, 58% patients were primigravida which was the majority and in controls, 53.7% were primigravida which were majority. Distribution of gravidity was similar in both groups (p=0.521).

Table III Obstetric profile of the study population (n=117)

Obstetric profile	Case (n=50) n (%)	Control (n=67) n (%)	p value
Gestational period			
1st trimester	0	1 (1.5)	
2nd trimester	5 (10)	14 (20.9)	0.299[c]
3rd trimester	18 (36)	18 (26.9)	
Puerperium	27 (54)	34 (50.7)	
Past obstetric history*			
Not Applicable (primigravida)	29 (58)	36 (53.7)	0.521[b]
Uneventful	9 (18)	24 (35.8)	0.021[b]
Abortion	4 (8)	2 (3)	0.083[c]
Pregnancy Related Hypertension Disorder	11 (22)	3 (4.5)	<0.001[c]
Ectopic pregnancy	1 (2)	1 (1.5)	0.979[c]
IUD/Stillbirth	3 (6)	1 (1.5)	0.083[c]

In cases, 54% patients and in controls, 50.7% were in puerperium which was the majority. Cases had statistically similar gestational age to controls (p=0.299). In this study, 35.8% of multigravida controls' have uneventful past obstetric history, while adverse event was statistically significant among cases which was Pregnancy Related Hypertension Disorder (22%) but other condition like abortion (8%) ectopic pregnancy (2%) and IUD (intra uterine death)/stillbirth (6%) which was statistically non-significant.

Table IV Distribution of pregnancy related risk factor among the study populations (n=117)

Adverse Maternal Outcome	Case (n=50) n (%)	Control (n=67) n (%)	p value
Pre-eclampsia/Eclampsia	16 (32)	0	
Pregnancy induced hypertension	17 (34)	0	
Sepsis	26 (52)	0	
Post partum heamorrhage	25 (50)	0	
Gestational diabetes mellitus	15 (30)	0	
Urinary tract infection	13 (26)	1 (1.5)	<0.001
Ante Partum Hemorrhage	9 (18)	0	
Hyperemesis gravidarum	6 (12)	1 (1.5)	0.112
HELLP	1 (2)	0	0.245
Acute diarhhoeal disease	1 (2)	0	0.245

Multiple response Adverse Maternal Outcome among the cases were pre-eclampsia/ eclampsia (32%) PIH (34%) sepsis (52%) postpartum hemorrhage (50%) gestational diabetes mellitus (30%) urinary tract infection (26%) (All p<0.001) ante partum hemorrhage (18%) (p<0.003) other condition like hyperemesis gravidarum (12%) HELLP (High elevated liver enzyme and low platelet) (2%) acute diarrheal disease (2%). Cases had significantly frequent adverse maternal outcome than control.

Table V Laboratory Parameter among the study population (n=117)

	Case n=50	Control n=67	p value
Hematological Profile			
Haemoglobin (gm/dl)*	7.39 ±0.90 (5.31-12.36)	11.83±1.83 (10.12-13.30)	<0.001[a]
Total leucocyte count (TLC) ($\times 10^9$ per litre)**	11.34 (9.89 -17.36)	8.9 (7.5 -11.21)	<0.001[b]
Platelet Count (PC) ($\times 10^9$ per litre)**	129.50 (82-193)	182 (158-238)	<0.001[b]
Biochemical Profile			
Serum Creatinine (mg/dl)*	1.78±0.35 (1. 4-2.30)	0.58±0.04 (0.55-0.71)	<0.001[a]
D-dimer (ng/ml)**	1280 (1190-1610)	845 (302-1088)	<0.001[b]
AST (U/L)**	28 (18-47)	17 (15-31)	<0.001[b]
ALT (U/L)**	28.50 (13-47)	12 (9-30)	<0.001[b]
Total bilirubin (µmol/L)**	0.62 (0.50-0.86)	0.6 (0.39-0.72)	0.341[b]
PT (Seconds)**	10 (9-12)	9 (7-12)	<0.001[b]
APTT (Seconds)**	31 (22-38)	23 (21-36)	<0.001[b]

This table shows Hemoglobin, Platelet count was significantly lower in cases than control (p<0.001 in All). Total Leucocyte Count, D-dimer, AST, ALT, PT and APTT were significantly higher in cases than control (p<0.001 in All).

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Table VI Predictors of pregnancy related acute injury in the study population (n=117)

Risk Factors	OR	95% CI Lower limit-Upper limit	p value
BMI	1.804	0.490-1.998	0.508
Pre-eclampsia/Eclampsia	1.104	1.001-1.998	0.049
Pregnancy induced hypertension	1 19	1.023-1.890	0.048
Sepsis	1.873	1.032-2.998	0.039
Post partum heamorrhage	2.223	1.921-2.990	0.038
Increased leucocytes count	1.338	1.015-3.476	0.049
Decreased platelet count	0.880	0.682-0.997	0.016
Increased-D-dimer	4.564	1.546-5.650	0.001
Prolonged PT	1.583	0.089-2.234	0.090
Prolonged APTT	1.899	1.023-2.442	0.030

Multivariate logistic regression was done to measure level of significance.

Multivariate logistic regression showed pre-eclampsia/eclampsia, PIH, sepsis, postpartum hemorrhage, increased total leucocytes count, decreased platelet count, Increased D-dimer, Prolonged APTT were independently associated with development of PRAKI.

Discussion

The mean age of the cases and controls were 35.5±5.33 years and 31.43±6.51 years accordingly. The mean age of the cases was statistically similar but average age of PRAKI patients slightly lower to the present study.[15,16] Majority of the patients belonged to middle-income group with statistical similarity in both groups, 42% in cases and 53.7% in controls.

Among cases about 54% were in the puerperium and 36% were in 3rd trimester which were similar to the previous study.[17,18] Usually, the development of AKI during pregnancy follows a bimodal distribution with two incidence peaks: one in the first trimester caused by septic abortion and other in the third trimester and/or around delivery due to late obstetrical complications.[19] Regarding gravidity, 58% of cases and 53.7% of controls were primigravida which were the majority in both groups.

In this study, 35.8% multigravida controls have uneventful past obstetric history. While in past obstetrics history pregnancy related hypertensive disorder (22%) was statistically significant in case then control. Adverse maternal outcome among the cases were pregnancy induced hypertension (34%) preeclampsia/eclampsia (32%) sepsis (52%) postp artum hemorrhage (50%) gestational diabetes mellitus (30%) urinary tract infection (26%) ante partum heamorrhage (18%) hyperemesis gravidarum (12%) HELLP (High elevated liver enzyme and low platelet) (2%) acute diarrheal disease (2%). According to study design, this study included only patients with no complications as controls. AKI is a heterogeneous syndrome in pregnant women and is caused by multiple etiology. It occurs typically in otherwise healthy women who developed obstetrical complication or acquired pregnancy-related medical condition such as pre-eclampsia, HELLP syndrome, etc.[19] However, several etiologies not related to pregnancy (Acute gastroenteritis, malaria, pyelonephritis, lupus nephritis, and acute interstitial nephritis) are also reported to cause PRAKI.[19] It is useful to consider the causes of PRAKI in relation to obstetrical complications and pregnancy-associated-specific diseases. In this study pregnancy-induced hypertension, sepsis and postpartum heamorrhage were predominant maternal adverse conditions. Several previous reviews reported these conditions as etiology of PRAKI.[10,20,21]

Hemoglobin and platelet count, were significantly lower among cases in comparison to controls. Total leucocyte count, D-dimer, AST, ALT, total bilirubin, PT, and APTT were higher among cases in comparison to controls. Considerable changes occur in the urinary tract system during normal pregnancy including increased size of kidneys due to renal vascular and interstitial space volume expansion. The physiological hydronephrosis of pregnancy characterized by a dilation of the calyces, renal pelvis, and ureter occurs in over 90% of pregnant women.[22] This anatomical abnormality may be present until the 16th postpartum week and promotes urinary stasis in the ureter, leading to the development of urinary tract infection. The dilatation of the urinary system is due to the hormonal effects of progesterone, external compression by the gravid uterus, and morphological changes in the ureteral wall. The systemic vasodilatory state typical of pregnancy, increases renal perfusion as well as Glomerular Filtration Rate (GFR). Renal plasma flow can increase up to 85% in the second trimester of pregnancy. The GFR can reach 40%–50% of baseline throughout pregnancy and subsides in the

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first 3 months postpartum. These hemodynamic abnormalities result in decreased serum creatinine in pregnant women to 0.4–0.5 mg/dl. Systematic vasodilation leads to the stimulation of antidiuretic hormone, resulting in decreased plasma osmolality and plasma sodium by 4–5 mEq/L.[22] Minute ventilation increases due to progesterone-induced stimulation of the central respiratory center in the brain. This results in a decrease in pCO_2 and a mild chronic respiratory alkalosis, which is compensated for renal excretion of bicarbonate. A decrease of about 4 mEq/L in bicarbonate concentration is common in the pregnant women. So during pregnancy the renal system becomes already vulnerable. So, systemic adverse conditions like sepsis, hypertensive emergency, obstetrical heamorrhage aggravates and promotes renal lesion and causes acute kidney injury. Multivariate logistic regression showed, pregnancy induced hypertension, preeclampsia/eclampsia, sepsis, postpartum heamorrhage, Increased total leucocyte count decreased platelet count, increased D-dimer, prolonged APTT all were independently associated with development of PRAKI but previous one study found preeclampsia and history of a previous hypertensive pregnancy as important risk factors.[3] A systematic review mentioned preeclampsia and the hypertensive disorders as dominant risk factors for PRAKI another study found HELLP syndrome associated with postpartum hemorrhage as a high risk for PRAKI.[23,24] Similarly, third study mentioned preeclampsia, hemorrhagic shocks as rick factors for PRAKI.[18] Regarding laboratory parameters, a single study found elevated D-dimer, prolonged prothrombin time, elevated total leukocytes count were significant risk factors for PRAKI which corroborates with the findings of present study.[25]

Limitation

Single centre and small sample size study.

Conclusion

From this study it can be concluded that pregnancy induced hypertension, Pre-eclampsia/ eclampsia, sepsis, postpartum heamorrhage, leukocytosis, decreased platelet count, increased D-dimer, prolonged APTT, were significantly associated with development of PRAKI.

Recommendation

Multicentre and large samples size study is recommeded to see the proper picture.

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Contribution of authors

PB-Conception, acquisition of data, data analysis, drafting & final approval.
MRP-Conception, acquisition of data, data analysis, drafting & final approval.
MMR-Design, critical revision & final approval.
TS-Data analysis, interpretation of data, critical revision & final approval.
CSHK-Design, critical revision & final approval.

Ethical approval

The protocol was approved by The Ethical Committee of Dhaka Medical College
Approval memo number – ERC-DMC/ECC/2020/ 406 and date- 23/12/2020.

Disclosure

All the authors declared no conflict of interest.

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