





Case Report

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Pregnancy-related acute kidney injury in a 31 year old primigravida

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ABSTRACT

Pregnancy-related acute kidney injury (AKI), a common obstetrics complication, could be associated with low detection rates particularly in resource-poor settings; hence, an optimized treatment plan is needed for better outcome. We present the case management of a 31-year-old primigravida who had tonic-clonic seizures, progressively declining urine volume and anuria. She had no prior risk factor for kidney disease. She was unconscious, pale, had jaundice and blood pressure was 160/110 mmHg. Laboratories showed deranged kidney and liver function, anemia, and low platelets. With a Stage 3 AKI, she was successfully delivered of a live fetus and managed on a multidisciplinary basis with four low-heparin hemodialysis sessions, cardio-protection, and antibiotics and she recovered normal kidney function.

Keywords: Acute kidney injury, Convulsion, Eclampsia, Kidney function, Preeclampsia, Pregnancy

INTRODUCTION

Pregnancy-related acute kidney injury (PR-AKI) has remained the most common cause of maternal and perinatal mortality.^[1] Advanced maternal age pregnancies, dysfunctional cardiorenal and metabolic states, and low socioeconomic status are associated with higher prevalence of PR-AKI.^[2] Severe (Stage 3) AKI and the need for renal replacement therapy (RRT) are known poor prognostic factors that could result in adverse outcome.^[3] Widespread endothelial dysfunction, proteinuria, and edema characterize PR-AKI; hence, its 14-fold increase in maternal mortality.^[4] Recent diagnostic criteria involve at least two blood pressure (BP) readings not <130/80 mmHg.^[5] A multidisciplinary management approach involving the nephrologist, obstetrician, hematologist, and social workers, among others, improves antenatal care and can prevent maternal and perinatal death.^[6] We report the management of a 31-year-old primigravida with severe PR-AKI who had hemodialysis, and recovered kidney function.

CASE REPORT

A 31 years old unbooked primigravida with gestational age (EGA) of 32 weeks was brought in from a peripheral facility with a 2-week history of progressive body swelling, reduction in urine volume, and anuria of a day duration. She had two episodes of tonic-clonic convulsions, became unconscious to her presentation. Her antenatal visits had been uneventful, her last visit (six days prior to her presentation) BP and hematocrit were 112/78 mmHg and 33% respectively [Table 1].

She was unconscious (Glasgow coma score of 7), jaundiced, pale, had edema and widespread coarse crackles were heard in the lung fields. Her admitting BP and hematocrit were 210/126 mmHg and

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16%, respectively. Her serum creatinine had risen from 123 μmoL to 381 $\mu moL.$ Her fetal heart rate was 132 beats.

Provisional assessment

Acute kidney injury (AKI), and to rule out the "hemolysis, elevated liver enzymes and low platelets" (HELLP) syndrome.

The obstetrician, anesthetics, hematologist, and social workers were invited for reviews. Bedside pre-operative work up showed proteinuria 3+, ketones 1+; bilirubinuria, prothrombin time (18 s), partial thromboplastin with kaolin (52 s), and international normalized ratio (1.4). The erythrocyte sedimentation rate was 32 mm/h and serology was negative for hepatitis B, hepatitis C, and human immunodeficiency viruses. She was resuscitated, received oxygen, frusemide, and intravenous labetalol 50 mg in 200 mL of saline infusion and underwent an emergency cesarean section and was delivered of a live female fetus with pre- through peri-operative packed cell transfusion. She was continued on oxygen and intravenous doses of labetalol, frusemide, ceftriaxone, metronidazole, and rabeprazole.

She regained consciousness 4 h on admission, BP was 182/98 mmHg, and then the first low-dose heparin hemodialysis session. The urea reduction ratio for the four dialysis sessions was 51%, 61%, 66%, and 58%, respectively. Her kidney function progressively improved. She was discharged on the 20th day on admission when her clinical state and kidney function became stable and have been stable in subsequent follow-up visits.

DISCUSSION

The eclamptic state, defined as a new onset hypertension and proteinuria during the last trimester of pregnancy or postpartum, typically occurs from the 20th gestational week to the postpartum period as it was with the index case.^[1-3] Documented incidence rates range from 0.8% to 8.8%, being higher in resource-challenged settings (RCSs).^[2,3] Preeclampsia involves systemic vascular endothelial dysfunction (endotheliosis) and vasoconstriction-induced vasospasms, leading to BP surges, proteinuria, and hematologic, kidney and hepatic dysfunction, and with or without the brain, commonly affected.^[3-5] Vasoconstriction-induced vasospasms in the renal vasculature lead to acute tubular necrosis (ATN) and glomerular dysfunction.^[4,7]

The World Health Organization estimates showed global annual deaths from pregnancy-related conditions at 280,000; hence, preeclampsia is documented to be the leading cause of PR-AKI worldwide with incidence of 36.6% in the United States, and mortality rates 2.73–4.3% compared to 23–33% in RCSs, with a reported incidence of 34.4% in Nigeria.^[8,9] Staging AKI according to the kidney disease improving global outcome criteria,^[10] the index patient had stage 3 AKI, evidenced by serum creatinine rise of 309% (\geq 300% of baseline within 7 days or concentration of \geq 354 umol/L within 48 h or \geq 50% rise from baseline within 7 days or any requirement for RRT). Stage 3 AKI and AKI requiring dialysis are associated with poor prognostic outcome.^[7]

Severe forms of PR-AKI could be associated with the HELLP syndrome, with intravascular mediated microangiopathy hemolytic anemia (MAHA), with histological findings of fibrinoid necrosis which is induced by malignant-phase hypertension-associated-severe vasoconstriction and vasospasm, with vascular leakage.^[2,4,5,9] Proteinuria, hyperuricemia, (common in MAHA, hemoconcentration and/or hemodilution); peripheral burr cells, and echinocytes

Table 1: Liver function tests, nematological, and renal biochemical prome of patient.									
Variables	ALT	AST	Total protein	Albumin	B1	B2	WCC	НСТ	PLTs
	IU/L	IU/L	g/dL	g/dL	μmol/L	µmol/L	cells/µL	%	cells/µL
	6-35	6-40	60-80	35-50	<26	<20	4-11	39-49	150-400
ANC visit								33	
Admission	232	467	88	38	185	114	9.8	16	62
7DOA	88	121	80	28	64	37	6.7	32	111
Discharge	18	22	82	40	16	12	5.2	40	179
Renal	NI-	CDC	17		0	~~	OFD	0/ OFD 1 1	** * * 1
Itellul	Na	SBC	K	Urea	Cr	% Creat rise	GFK	% GFR decline	Uric acid
Biochemistry	mmol/L	mmol/L	mmol/L	mmol/L	 μmol/L	% Creat rise %	mL/min	% GFR decline %	mmol/L
Biochemistry	mmol/L 135–145	SBC mmol/L 22-30	mmol/L 3.5–5.0	mmol/L 3-7	<u>μmol/L</u> 50–100	% Creat rise %	GFR mL/min	% GFR decline %	Uric acid mmol/L 0.16-0.44
Biochemistry Last ANC visit	Na mmol/L 135–145	SBC mmol/L 22-30		Urea mmol/L 3-7	Cr μmol/L 50–100 123.0	% Creat rise %	GFR mL/min 58	% GFR decline %	0.16-0.44
Biochemistry Last ANC visit Admission	Na mmol/L 135-145	88C mmol/L 22-30 18	K mmol/L 3.5-5.0 5.4	Urea mmol/L 3-7 14.0	Cr μmol/L 50–100 123.0 381	<u>% Creat rise</u> <u>%</u> 309	GFR mL/min 58 15	% GFR decline % 74	0.16-0.44
Last ANC visit Admission 7 th DOA	INA mmol/L 135–145 133 134	SBC mmol/L 22-30 18 21	K mmol/L 3.5–5.0 5.4 4.4	Urea mmol/L 3-7 14.0 6.8	Cr μmol/L 50–100 123.0 381 109	% Creat rise %	6FK mL/min 58 15	% GFR decline % 74	0.16-0.44 0.93 0.61
Biochemistry Last ANC visit Admission 7 th DOA Discharge	Na mmol/L 135–145 133 134 137	SBC mmol/L 22-30 18 21 23	K mmol/L 3.5-5.0 5.4 4.4 3.5	Urea mmol/L 3-7 14.0 6.8 4.1	Cr μmol/L 50–100 123.0 381 109 92	<u>% Creat rise</u> <u>%</u> 309	GFK mL/min 58 15	<u>% GFR decline</u> %	0.16-0.44 0.93 0.61 0.38

ALT: Alanine transaminase, AST: Aspartate transaminase, B1: Total bilirubin, B2: Conjugated bilirubin, WCC: White cell count, HCT: Hematocrit, PLTs: Platelets, ANC: Antenatal care, DOA: Day on admission, GFR: Glomerular filtration rate, SBC: Serum bicarbonate concentration.

are common findings in PR-AKI.^[1,3] With deranged clotting profile, coagulopathy should always be ruled out.^[2,3] The absence of common risk factors for PR-AKI such as family history, previous occurrence, poor obstetric history or fetal death, in the index patient further reinforces our assertion that being a primigravida, and poor compliance with antenatal care (ANC) regimen, were significant predisposing factors.^[4-7] This is coupled with the fact that other risk factors such as teenage pregnancy, older patient (>35 years), obesity, diabetes, background hypertension, kidney disease, and hematologic and connective tissue diseases were largely ruled out.

Placenta hypoperfusion and ischemia induce greater expression of maternal anti-angiogenic factors that induce the systemic endothelial dysfunction and microangiopathy. This leads to proteinuria, glomerular endothelioses, swelling and obliteration of the endothelial fenestrations, and loss of the capillary space. These progresses to severe reductions in renal plasma flow and glomerular filtration rate.^[10] Severe PR-AKI can progress from ATN to acute cortical necrosis, particularly in chronic hypoperfusion states such as hypertension, diabetes, and chronic kidney disease.^[9] The loss of cerebral autoregulation could also lead to vascular dilatation, increased permeability, and cerebral edema, which can progress to cerebral ischemia and encephalopathy.^[2,11]

Managing PR-AKI involves ruling out causative factors such as acute fatty liver of pregnancy, and MAHA-associated thrombotic thrombocytopenic purpura and hemolytic uremic syndrome. Optimal BP control, augmenting renal blood flow, dialysis, optimizing nutrition, and terminating seizures with anticonvulsants are essential components of management.^[8,10,11]

CONCLUSION

Pregnancy-related AKI remains a major global health challenge, more so in developing countries. Preeclampsia maintains its dominance as a pivotal challenge in emerging economies where its synergism with suboptimal ANC occasioned by socioeconomic deprivation still persists. While eradicating the primary cause, effective BP control during pregnancy, in PR-AKI, with dialysis, remains pivotal steps in preventing and managing severe cases. Long-term follow-up is necessary to sustain kidney function.

Ethical approval

The Institutional Review Board approval is not required.

Declaration of patient consent

Patient's consent is not required as patients' identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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