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Acute kidney injury in obstetrics: Varied pathologies and outcomes

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Abstract

Background: Acute Kidney Injury (AKI) is the sudden loss of renal function. Multiple causes of AKI exist that include those that afflict the general population, and those that are associated with pregnancy. AKI in pregnancy is of particular challenge as there are physiological alterations of renal function in pregnancy. The diagnosis is by abnormalities of renal function tests in addition to the abnormalities reflecting underlying pathologies. Management depends on underlying causes and the prognosis can often be positive.
Material and methods: We present three cases of AKI in pregnancy, their evaluation and management.
Results: The first patient had AKI due to septic abortion and was treated by dialysis. The second patient had AKI due to preeclampsia with severe features and was treated by termination of pregnancy and multiple anti-hypertensives. The third patient had AKI due to hyperemesis gravidarum and was treated aggressively with fluids and anti-emetics.
Conclusion: Early recognition and initiation of appropriate therapy can lead to favourable outcomes in AKI.

Keywords: Pregnancy, renal failure, renal replacement, dialysis

Introduction

Acute Kidney Injury (AKI), which replaces the term-Acute Renal Failure, is abrupt loss of renal function. The etiology is varied and can be due to pre-renal, renal/interstitial and post-renal causes. In addition to the causes that afflict general population, there are pregnancy specific pathologies that can result in AKI in pregnancy. The recognition of AKI is not usually a challenge but complications can be life-threatening.
We present three obstetric patients who had AKI due to varied causes.

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Case 1

A 36 years para 5 living 5 tribal woman was brought to casualty with history of 4 days of fever with chills, decreased urination since 2 days and drowsiness since 12 hours, as elicited from the husband. She was conscious, oriented, irritable and lethargic. She was pale, dehydrated, febrile (102degF) and had grade 2 pitting pedal edema. Her pulse was 126/min, blood pressure was 98/66mmHg and respiratory rate was 18/min. Significant investigation reports were as follows: Haemoglobin-5.2g/dL, WBCs-33,000 X 10^9/L, S. Sodium-113mmol/L, S. potassium-5.3mmol/L, S. chlorine-88mmol/L, B. Urea-188mg/dL. S. creatinine-6.3mg/dL. Urine pregnancy test was negative. Ultrasound showed evidence of minimal? Retained products of conception. Bilateral kidneys showed altered parenchymal echotexture with moderate loss of cortico-medullary differentiation. As grave risk was being explained, it was revealed that the patient had got an abortion done, 2 weeks ago, by insertion of some material per vaginally.
She was immediately shifted to Nephro-Urology, where she was managed with blood transfusion, higher order antibiotics, and fluids. She had four cycles of hemodialysis on four consecutive days. Over the next 7 days, patient gradually improved. She was discharged on day 11 with advice for strict follow-up. However, she was lost to follow-up.

Case 2

A 21 years primigravida was referred from primary health centre for the management of severe hypertension. She was at 35 weeks and 3 days of gestation, diagnosed with preeclampsia 3 weeks ago. She had been on tablet labetalol 100 mg twice daily. She had no other significant history. She had headache and blurring of vision since 12 hours.
On examination, she had facial puffiness, periorbital edema, and grade 3 pitting pedal edema. Pulse was 98/min, blood pressure was 194/130mmHg, respiratory rate was 22/min. She was afebrile. She had mild basal crepitations bilaterally. Haemoglobin-9.2g/dL, WBCs-9X10^9/L, Platelets-87 X 10^9/L, S. Sodium-133mmol/L, S. potassium-3.3mmol/L, S. chlorine-110mmol/L, B. Urea-71mg/dL. S. creatinine-2.2mg/dL. S. LDH-1170U/L, S. Uric acid-9.2mg/dL. Obstetric ultrasound showed oligohydramnios (AFI 3.3cm) with fetal growth restriction and absent end diastolic flow with brain sparing effect. Ultrasound Abdomen showed mild pleural effusion. There was bilateral papilloedema on fundoscopy.
Patient was administered oral and injectable antihypertensives but no control was seen. Magnesium sulfate was withheld in view of abnormal renal parameters. She underwent emergency Caesarean section and delivered a preterm, alive male baby of 1.78Kg. Baby was admitted to the NICU in view of low birth weight and low APGAR scores. Over the next 3 post-operative days, BP continued to be in the range of 150-160/90-100mmHg. Renal parameters were still abnormally high. Urine output was satisfactory. Nephrourology opinion was sought. There was no renal abnormality on ultrasound. 2-D echocardiography was normal. Antihypertensives were changed from labetalol and amlodipine to amlodipine, metoprolol and furosemide. Over the next 10 days both blood pressure and all renal parameters returned to baseline. She was discharged on day 14.

Case 3

A 27 years, multiparous woman presented to the casualty at 11 weeks gestation with drowsiness following severe nausea and vomiting of over 15-20 episodes in the last 2-3 days. She could barely tolerate liquids but not any solid foods. She was admitted once previously at 8 weeks with severe hyperemesis gravidarum for intravenous hydration. She was only on prenatal vitamins and occasionally doxyllamine for nausea and vomiting.

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She looked exhausted, irritable with signs of gross dehydration. She weighed 42 Kg and had lost 4.5Kg in the last 6 weeks. Pulse was 130 beats per minute, blood pressure was 90/58 mm Hg, and temperature of 37.4C. She had a hematocrit of 46%, serum creatinine of 3.7 mg/dL and blood urea nitrogen of 96 mg/dL, serum sodium -131 mmol/L, potassium-4.4 mmol/L, and chloride 79 mmol/L. Urinalysis showed proteinuria (2+), and ketones positive. Arterial blood gas values showed pH 7.47, CO2 34 mm Hg, and bicarbonate 24 mmol/L. Serological tests were negative for hepatitis B, hepatitis C, and HIV. Her thyroid-stimulating hormone (TSH) was 0.01 IU/mL, free thyroxine (T4) was 3.9 ng/dL. Renal ultrasound showed normal-sized kidneys with parenchymal echogenicity but no hydronephrosis, obstruction, or calculi. Her obstetric ultrasound showed no abnormality.

Despite intravenous fluid administration, her urine output was about 400mL on the first day. Aggressive intravenous fluid management was continued. She was administered injection Thiamine. The vomiting episodes were controlled with injection ondensetron. Serial measurements of serum creatinine and blood urea nitrogen showed improvement in renal function over the next 5 days. On day 14, her blood urea and serum creatinine were 22 mg/dL and 1.3 mg/dL, respectively. She was discharged with advice for follow-up. Two weeks after discharge, follow-up showed normalization of renal function, with a blood urea nitrogen of 16 mg/dL and serum creatinine of 0.7 mg/dL. Her TSH and free T4 had normalized. She had occasional vomiting until about 15 weeks and had completely stopped thereafter. She delivered a healthy female baby of 3.1Kg at term.

Discussion

Acute Kidney Injury (AKI) is the sudden loss of renal function leading to retention/accumulation of nitrogenous wastes in the body. Consensus definitions of AKI have been laid down by RIFLE (Risk, Injury, Failure, Loss, End Stage Renal Disease), AKIN (Acute Kidney Injury Network) and KDIGO (Kidney Disease: Improving Global Outcomes) modifications of AKIN. However, there exists no set definition of AKI in pregnancy, owing partly to, physiologic changes in renal function in pregnancy.

The incidence is pegged at 1 in 20,000 [1] pregnancies with some authors reporting as high an incidence as 1 in 50 [2] pregnancies.

AKIs are traditionally categorized as pre-renal, renal/interstitial and post-renal, by etiology. The commonest causes of AKI in pregnancies less than 20 weeks are pre-renal diseases that include hyperemesis gravidarum, acute tubular necrosis secondary to septic abortions, and infections [3]. In advanced pregnancies, and in the postpartum period, severe preeclampsia, HELLP syndrome, Acute Fatty Liver of Pregnancy (AFLP), thrombotic microangiopathy, drug-induced (NSAIDs) and Acute Tubular Necrosis associated with massive haemorrhage are common predisposing conditions [3].

Septic abortion, an unfortunate occurrence in the modern world, poses life-threatening complications and commonly causes multiorgan failure. Acute kidney injury by acute tubular necrosis is known to be the probable mechanism [4]. It is known that the interstitial/renal causes of AKI have higher mortality and morbidity [5] as compared to renal and post-renal causes, usually requiring renal replacement. Our first patient resorted to unsafe abortion and consequently had renal failure. Ultrasound showed normal sized kidneys which points towards an acute etiology. The management of sepsis must be aggressive even as the prognosis is often guarded. The requirement for high order injectable antibiotics is often non-negotiable and must be decided appropriately, bearing the on-going renal insult in mind. In our case, hemodialysis initiated at the appropriate time may have been the key in saving her life.

Preeclampsia, the obstetric enigma, afflicts 3-5% of pregnancies with varying consequences [3]. This multifactorial hypertensive disorder of pregnancy results from endothelial injury by

placental toxins. The corner stone of management is adequate control of blood pressure by antihypertensive medications. The definitive treatment is by termination of pregnancy. Overall decision depends on control of hypertension, gestational age and feto-maternal complications. The incidence of AKI in preeclamsia is heightened when HELLP syndrome is associated [3]. Renal replacement is required when there is evidence of ischaemic Acute Tubular Necrosis. In our case, although the patient did have severe preeclampsia, aggressive control of hypertension did suffice without the need for renal replacement.

Hyperemesis gravidarum is the exaggerated end of the nausea and vomiting spectrum of pregnancy which is characterized by 5% loss of pre-pregnancy body weight [6] and ketonuria. It often involves volume depletion and dyselectrolytemia and significantly impacts quality of life. The pathogenesis is hypothesized to be multifactorial that include hormonal changes, altered gastrointestinal motility, and genetic factors. Laboratory parameters such as electrolyte and acid base imbalances, increased hematocrit, increased blood urea and serum creatinine, transient hyperthyroidism, and low magnesium and calcium levels may be present in varying combinations. Hyperemesis may cause nutritional deprivation, electrolyte imbalance, Wernicke's encephalopathy, and renal failure. Severe volume depletion causing prerenal failure, which our patient had, is rare. Although ou patient had acute kidney injury, the treatment, was largely supportive with fluids, vitamins, and anti-emetics and the response was prompt.

Conclusion

Acute Kidney Injury has multiple precipitating factors and pregnancy-specific causes. Three pathologies, namely, Septic abortion, Preeclampsia and Hyperemesis gravidarum, which lead to Acute Kidney Injury with varying severity was discussed in the present article Timely recognition and treatment led to betterment in all three patients.

Although AKI can be a life-threatening complication of common pregnancy-related pathologies, early diagnosis and prompt initiation/maintenance of treatment can often result in favourable outcomes.

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