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Case Series

Postpartum kidney crisis: hidden burden of postpartum acute kidney injury

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ABSTRACT

Postpartum acute kidney injury (PPAKI) is a serious complication of pregnancy. This case series analyses seven patients aged 25-35 years, predominantly with hypertensive disorders such as preeclampsia, HELLP syndrome and eclampsia, with few complicated by sepsis and hemorrhagic shock. Dialysis was necessary in the majority of cases, with one mortality. The severity and multifactorial etiology of PPAKI are highlighted by diagnostic and treatment challenges. Results were greatly impacted by prompt intensive care unit treatment, renal support, and a multidisciplinary approach. In order to prevent irreversible renal damage, this series emphasizes the importance of early detection, aggressive management, and customized postpartum surveillance in high-risk obstetric populations.

Keywords: Acute kidney injury, HELLP syndrome, Preeclampsia, ICU, Multidisciplinary management

INTRODUCTION

Acute kidney injury (AKI) occurring in the postpartum period, known as postpartum acute kidney injury (PPAKI). Postpartum kidney disease is a critical yet often underrecognized complication of pregnancy. Sepsis and postpartum hemorrhage (PPH) are the leading causes of PPAKI in underdeveloped countries, whereas in developed countries, severe preeclampsia, PPH, and HELLP (hemolysis, high liver enzymes, and low platelets) syndrome are main contributory factors.[1] Rare causes include hemolytic uremic syndrome (HUS), which is a leading cause of end-stage renal disease (ESRD) in this group.

PPAKI constitutes 28–72% of pregnancy related acute kidney injury cases.[2] Challenges remain in the accurate diagnosis of PPAKI due to physiological alterations in renal function during pregnancy, the absence of universally accepted diagnostic criteria specific to postpartum kidney disease, and variations in healthcare access and antenatal care across different populations. Although the overall incidence of severe PPAKI requiring renal replacement therapy (RRT) is low, it is estimated at approximately 1 in 20,000 pregnancies.[3]

CASE SERIES

All women in our case series were in age group of 25-35 years, with a mean age of 28 years. Amongst seven young females, five had preterm delivery and two had term deliveries. Six cases had emergency caesarean section and one had preterm vaginal delivery. Four patients had antenatal history of preeclampsia. Notably, cases with severe preeclampsia and HELLP demonstrated significant thrombocytopenia (platelet count <50,000/µl) and elevated liver enzymes, indicative of systemic endothelial injury and microvascular thrombosis contributing to AKI. Additionally, markedly reduced urine output in four cases, in three of the cases presenting anuria (nil urine output), underscores the severity of renal compromise. Patients with sepsis and hemorrhagic shock displayed profound hypotension (BP 80/50 mmHg), leading to ischemic kidney injury, exacerbated by multi-organ dysfunction (MODS) and high urea/creatinine levels (up to 209/5.09 mg/dl). Those requiring mechanical ventilation or high

oxygen support were at a greater risk of multi-organ failure, further complicating renal outcomes.

The management of these cases primarily involved ICU admission, fluid restriction, and higher antibiotics. Most cases (six out of seven) required dialysis, with the number of sessions varying based on severity. Case fourth required continuous renal replacement therapy (CRRT) for 120 hours, while fifth case underwent seven cycles of plasma exchange (PLEX). Sixth case had 16 dialysis count, and seventh case required ten sessions, renal biopsy was done in view of persistently deranged KFT, which was suggestive of lupus nephritis. two doses of IVIG and one dose of cyclophosphamide was given for the same. The outcomes were favorable for most cases, except fourth case, which resulted in mortality on day six. The length of hospital stays varied, with the discharge timeline ranging from 15 days to 50 days for survivors.

Table 1: Patients demographic details, antenatal history and postpartum complications.

Case	Age (years)	Gestation at delivery in weeks	Mode of delivery	Antenatal history	Postpartum complications
Case 1	26	39	Cesarean	None	Acute fatty liver of pregnancy
Case 2	25	32	Vaginal delivery	Preeclampsia	HELLP
Case 3	35	37	Cesarean	Preeclampsia	HELLP and sepsis
Case 4	31	30	Cesarean	Preeclampsia	Eclampsia and HELLP
Case 5	26	34	Cesarean	Preeclampsia	HELLP
Case 6	28	36	Cesarean	Preeclampsia	Hemorrhagic shock and MODS
Case 7	25	32	Cesarean	None	Eclampsia

Table 2: Analysis of course in hospital.

Case	Urine output on admission	Urea/ creatinine	Total ICU stay (days)	Dialysis (cycles)	Outcome	Total hospital stays (days)
Case 1	Nil	54/4.67	4	7	Alive	15
Case 2	Nil	110/4.02	5	2	Alive	21
Case 3	Less than 30 ml/hour	123/5.4	4	2	Alive	15
Case 4	Nil	209/5.5	6	CRRT-120 hours	Mortality	Died on day 6 after admission
Case 5	Less than 30 ml/hour	138.1/3.37	4	Plex-7	Alive	20
Case 6	Nil	138.1/3.37	17	16	Alive	56
Case 7	Less than 30 ml/hour	147/2.1	5	10	Alive	39

DISCUSSION

The cases analyzed in this study, highlight the diverse and complex etiologies of postpartum kidney disease, emphasizing the critical role of early recognition and intervention. Hypertensive disorders including preeclampsia, HELLP syndrome, and eclampsia, were prominent contributors to renal dysfunction, leading to complications such as acute kidney injury, thrombotic microangiopathy, and multi-organ failure. Sepsis emerged as another major factor, with cases demonstrating its role in systemic inflammation, endothelial damage, and circulatory collapse, further exacerbating renal impairment. As per analysis in a population-based cohort study conducted in Canada between 2012 and 2016, rates of obstetric acute renal failure rose from 1.66 to 2.68 per 10 000 deliveries between 2003-04 and 2009-10. Majority of acute renal failure increased in deliveries involving hypertensive disorders, and it was particularly noticeable in women who had gestational hypertension with significant proteinuria.[4] In a prospective observational study conducted by Kountouri done between 2013 and 2019, women who had a pregnancy complicated by a hypertensive disorder, had renal dysfunction in 32% of the total cohort, on 6-8 weeks follow-up visit. Independent predictors for the development of renal failure were pre-eclampsia, chronic hypertension, highest measured antenatal serum creatinine, highest measured antenatal 24-hour urinary protein, and blood pressure ≥140/90 mmHg at the postnatal visit.[5] Retrospective study done by Shu et al between the period of 2013 to 2017 suggests all the cases of PPAKI occurred within 1 week after delivery which were similar in our study.[6]

CONCLUSION

This case series emphasis the multifactorial etiology of postpartum kidney disease, with hypertensive disorders, sepsis, and haemorrhagic shock emerging as key contributors to AKI and multi-organ dysfunction. Management strategies primarily involved intensive care support, fluid management, broad-spectrum antibiotics, and renal replacement therapy. Thus, management of PPAKI with multidisciplinary approach gives early recovery and good clinical outcome.

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