Research Article

The Complete Resolution Of Pregnancy-Related Acute Kidney Injury In An Eclampsia Patient Complicated By HELLP Syndrome: A Case Report

Kenly Kenly[1,2,3,4]*, Widodo Widodo[1,2], Dana Pramudya[1,2], Andro Pramana Witarto[5,6]

[1]Division of Nephrology and Hypertension, Department of Internal Medicine, Dr. Soetomo General Academic Hospital, Surabaya, Indonesia
[2]Division of Nephrology and Hypertension, Department of Internal Medicine, Faculty of Medicine, Universitas Airlangga, Indonesia
[3]Internal Medicine Subspecialist Study Program, Department of Internal Medicine, Dr. Soetomo General Academic Hospital, Surabaya, Indonesia
[4]Internal Medicine Subspecialist Study Program, Department of Internal Medicine, Faculty of Medicine, Universitas Airlangga, Indonesia
[5]Internal Medicine Specialist Study Program, Department of Internal Medicine, Dr. Soetomo General Academic Hospital, Surabaya, Indonesia
[6]Internal Medicine Specialist Study Program, Department of Internal Medicine, Faculty of Medicine, Universitas Airlangga, Indonesia
*Corresponding author:
Kenly Kenly, kenly_chandra@yahoo.com, Division of Nephrology and Hypertension, Department of Internal Medicine, Dr. Soetomo General Academic Hospital, Jl. Mayjend Prof. Dr. Moestopo No. 6-8 Surabaya 60286, Indonesia

ABSTRACT

Pregnancy-related acute kidney injury (PrAKI) is a significant cause of maternal and fetal morbidity, particularly when complicated by eclampsia and HELLP syndrome. We report a 34-year-old woman in her second pregnancy with seizures, anuria, severe thrombocytopenia, markedly elevated liver enzymes, and intrauterine fetal death at approximately 31–32 weeks of gestation. Laboratory findings confirmed PrAKI alongside eclampsia, HELLP syndrome, pneumonia, and gestational diabetes mellitus. Despite initial fluid management and diuretic therapy, anuria and rising serum creatinine persisted, necessitating hemodialysis initiation on the second day. A total of six hemodialysis sessions were completed, after which kidney function gradually recovered. The hemodialysis was then discontinued due to her significant renal improvement with stable clinical and metabolic parameters within approximately two months, consistent with KDIGO 2012 recommendation to discontinue RRT when intrinsic kidney function has recovered sufficiently to meet patient needs. As the conclusion, the fundamental aspect of PrAKI management is supportive care to maintain renal perfusion.

Keyword: Pregnancy; Acute kidney injury; Eclampsia; HELLP syndrome; Case report.

INTRODUCTION

Pregnancy-related acute kidney injury (PrAKI) is recognized as a significant cause of maternal and fetal morbidity and mortality.[1,2] Its incidence ranges from 40 to 100 cases per 100,000 pregnancies with a higher incidence in low- and middle-income countries (LMICs).[3] It remains to be a common cause for starting dialysis in LMICs although the incidence has generally declined due to the quality improvements in the antenatal and peripartum care.[1,4] Most AKI cases occur in women without prior kidney disease and are often undetected due to the physiological changes in pregnancy.[5–7] To date, there are still no clear definitions of PrAKI.[8] Its development follows a bimodal distribution with two incidence peaks in the first and third trimesters, and is determined by the gestational age and clinical presentations.[1,4]

The main causes of PrAKI during late pregnancy, especially after 20 weeks or in the third trimester, are preeclampsia, eclampsia, and HELLP syndrome (hemolysis, elevated liver enzymes, low platelet count).[1,9] Preeclampsia account for 15–20% of PrAKI cases.[5] HELLP syndrome – one of several serious complications in pregnancy characterized by hepatic dysfunction and thrombotic microangiopathy – may be found in 10–20% of preeclampsia cases and is associated with PrAKI in 7–36% of cases.[10,11] Approximately 2–3% of severe preeclampsia cases can also progress to eclampsia as a continuum, which is defined as unexplained convulsive seizures or coma in pregnancy with hypertensive disorders[11,12] and may complicate preexisting HELLP syndrome. The presence of HELLP syndrome confers a four- to fivefold increased risk of PrAKI; among those affected, approximately 10% develop kidney injury, of whom 10–40% ultimately require dialysis.[10,13]

PrAKI carries significant risks to both maternal and fetal health. It is influenced by various obstetric complications, such as preeclampsia, eclampsia, and HELLP syndrome.[1,9,13] It often complicates the clinical course and requires careful management. Furthermore, the progression of preeclampsia to eclampsia adds further layers of clinical urgency and complexity. Given the overlapping manifestations and the potential for rapid deterioration, early recognition is critical for favorable outcomes. The following case provides a detailed AKI development in a case of

eclampsia complicated by HELLP syndrome, underscoring important diagnostic challenges and therapeutic interventions in managing high-risk pregnancies.

CASE REPORT

A 34-year-old pregnant woman with gestational age of 29/30 weeks was referred from private hospital with seizure around 5 minutes followed by reduced fetal movement. Other complains included dizziness and epigastric pain one day before hospital admission and shortness of breath since she arrived in the emergency room (ER); however, chest pain, blurry vision, headache, fever, and cough were not found. Urine production was 20 mL since she arrived in the ER. Bowel movements was normal. From the previous hospital, she has already received anticonvulsants, $MgSO_4$, oral nifedipine, and fluid maintenance. Her medical history was hypertension and treated with oral nifedipine 10 mg once daily. She is married and has a history of giving birth to her first child weighing 3,000 grams. Her child is currently 9 years old. This is her second pregnancy after 9 years and the antenatal care was routinely done by a midwife.

The abnormal physical examination was positive bibasilar lung crackles and pretibial edema with blood pressure (BP) 132/78 mmHg, heart rate (HR) 139 beats/min, and respiratory rate (RR) 26 breaths/min. For the obstetric status, there were no contraction and fetal heartbeat, and vaginal touché (VT) was not performed. The ultrasound result was single fetus with intrauterine fetal death (IUFD), head presentation, biparietal diameter (BPD) 7.85 cm (~31/32 weeks), head circumference (HC) 28.41 cm (~31/32 weeks), abdominal circumference (AC) 25.20 cm (~29/30 weeks), femur length (FL) 6.00 cm (~31/32 weeks), and estimated fetal weight (EFW) 1,552 grams. The placenta was implanted at the posterolateral part of the uterine corpus. The abnormal laboratory values on admission were hemoglobin 16.4 g/dL, leukocyte 13,620 cells/mm^3, thrombocyte 27,000 cells/mm^3, aspartate aminotransferase (AST) 2,352 IU/L, alanine aminotransferase (ALT) 684 IU/L, blood urea nitrogen (BUN) 24 mg/dL, SCr 1.85 mg/dL, magnesium 4.8 mg/dL, serum albumin 2.8 g/dL, total bilirubin 12.48 mg/dL, direct bilirubin 6.6 mg/dL, random blood glucose (RBG) 394 mg/dL, lactate dehydrogenase (LDH) 3,275 IU/L. The second day laboratory results were hemoglobin 12.3 g/dL, leukocyte 18.980 cells/mm^3, neutrophils 85.5%, thrombocyte 107.000 cells/mm^3, BUN 45 mg/dL, SCr 3.33 mg/dL. The chest X-ray (Fig. 1) showed pneumonia, left pleural effusion, cardiomegaly, and an installed double lumen catheter (DLC) with distal tip passing through the midline and projected as high as 4^{th} to 5^{th} thoracic vertebrae (T4-T5) on the right side. The abdominal ultrasound showed no abnormalities. The head non-contrast computed tomography (NCCT) scan showed no bleeding, cerebral infarction, or cerebral mass; however, mild cerebral edema was found. She was then diagnosed with G2P1 complicated by PrAKI, eclampsia, HELLP syndrome, IUFD, pneumonia, and gestational diabetes mellitus (GDM).

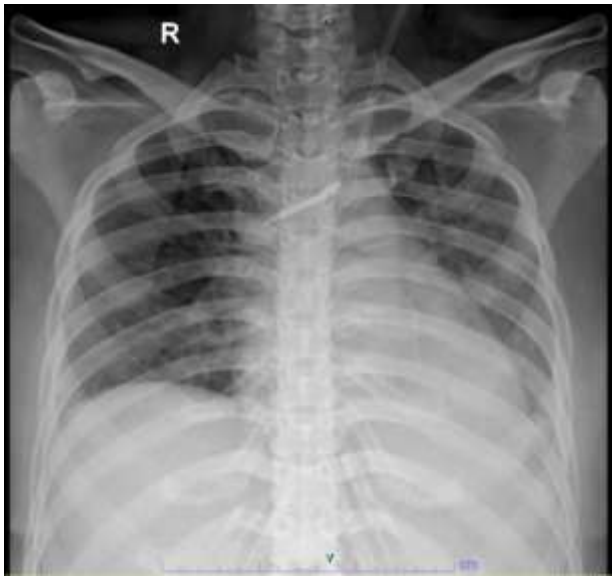
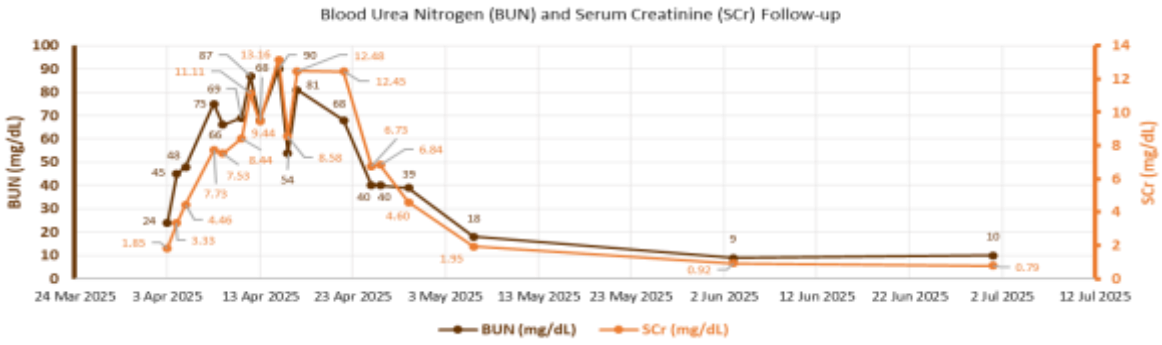


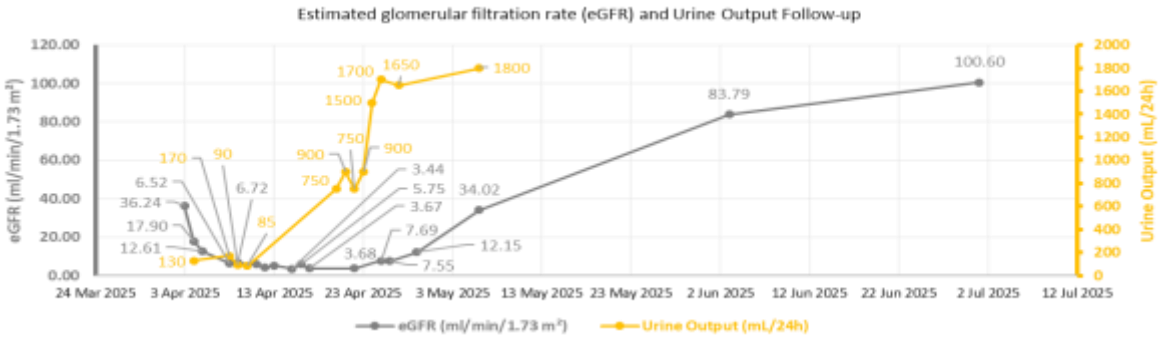
Figure 1. Chest X-ray demonstrating pneumonia, left pleural effusion, cardiomegaly, and an installed double lumen catheter (DLC) with distal tip passing through the midline and projected as high as 4th to 5th thoracic vertebrae (T4-T5) on the right side

The comprehensive management included forming a multidisciplinary team, preparation of intensive care unit, optimal fluid management, $MgSO_4$ initiation, anticonvulsants, antihypertensive drugs, thrombocyte concentrate (TC) transfusion in preparation for delivery, blood glucose regulation using insulin, infection control with antibiotics, and dialysis. In the referral hospital, she received 500 mL of crystalloid with approximately 20 mL of urine production over a 6-hour period before arrival to our hospital. She also received 4 g of $MgSO_4$ as a loading dose, followed by a maintenance infusion at a rate of 1 g/hour, and 20 mg of oral nifedipine as the antihypertensive drug. After her arrival in our hospital, her urine production was still approximately 20 mL. Then, the MgSO4 maintenance infusion was immediately discontinued, and was changed to 500 mL of normal saline as a maintenance infusion for 24 hours. Diuretic was also given due to signs of overload. However, after 24 hours, she was still anuria and her SCr was gradually increasing (Fig. 2). Therefore, hemodialysis via DLC was initiated on the second day (April 4th, 2025) with the following prescription: a duration of 2.5 hours, Q_B 150 mL/min, Q_D 500 mL/min, ultrafiltration (UF) 1.5 L, and heparin-free due to thrombocytopenia. After hemodynamically stable by controlling her BP using intravenous nicardipine and transfusing ten bags of TC to reach thrombocyte >50,000 cells/mm^3, the delivery was then performed by a skilled obstetrician. The elevated liver enzymes were significantly recovered after 72 hours. Despite of adequate treatment, her SCr was still increasing. She was then decided to receive additional hemodialysis sessions while still observing her SCr and urine output. After six hemodialysis sessions, her renal function has improved from May 2025 until now (Fig. 2) and the hemodialysis was then stopped.

Figure 2. Follow-up graphs of renal function tests (RFTs), including (1) blood urea nitrogen (BUN) and serum creatinine (SCr) in the top graph, and (2) estimated glomerular filtration rate (eGFR; based on the 2021 CKD-EPI



(1)



(2)

formula) and urine output in the bottom graph.

DISCUSSION

The management of PrAKI in eclampsia and HELLP syndrome is multifaceted, requiring simultaneous attention to fluid balance to maintain renal perfusion, seizure and BP control, and organ support.[1,4] In the present patient, fluid management was immediately challenging given her anuria on admission, bibasilar crackles, and uncertain volume status. A cautious, limited crystalloid challenge approximately 250 mL is recommended after excluding pulmonary congestion. Loop diuretics have also been widely utilized to treat AKI worldwide and are rarely hazardous during the first 48h; however, preeclampsia patients should not use diuretics unless pulmonary oedema is established.[4] In this case, normal saline 500 mL over 24 hours was used as maintenance, along with a loop diuretic for signs of fluid overload; however, anuria persisted and SCr continued to rise. Such cases with persistent abnormal SCr should be referred to nephrologist for further assessments.[3] Furthermore, renal replacement therapy (RRT) or hemodialysis indications in such patients mirror those in the non-pregnant population, including refractory fluid overload, hyperkalemia, and metabolic acidosis.[3,12] Residual kidney function is considered important in the dialysis initiation of pregnant women, which is recommended to be started from 2 hours three times per week and should be increased to 24–36 hours per week.[14,15] However, current evidence still remains insufficient to establish clear recommendations on when to initiate, how long to continue, or which modality of RRT to select in PrAKI; therefore, dialysis prescriptions should be individualized.[11] Typically, even severe AKI in HELLP syndrome has a high likelihood of kidney recovery of up to 80%.[1]

With regard to eclampsia complicated by HELLP syndrome, the definitive treatment is seizure and BP control, fetal delivery, timed according to gestational age, maternal stability, and severity of disease.[3,12] The drug of choice for preventing and controlling eclampsia is magnesium sulphate ($MgSO_4$), acting through multiple mechanisms including N-methyl D-aspartate (NMDA) receptor modulation, cerebral vasodilation, and calcium channel antagonism.[6,12] Its standard intravenous loading dose at 4 g can be administered to all patients. However, given that $MgSO_4$ is renally cleared, the maintenance dose was appropriately reduced from the standard 1 g/hour to 0.5 g/hour and subsequently discontinued in the setting of oliguria and AKI, with serum magnesium levels monitored every 4–6 hours to prevent toxicity and maintained at <3.7 mmol/L (<9 mg/dL).[6] Conversely, antihypertensive treatment to achieve BP <135/85 is not recommended unless BP is >160/110 mmHg, or even lower threshold for patients with comorbidities or impaired renal function.[16] Labetalol, nifedipine, methyldopa, and hydralazine are widely used as first-line antihypertensive therapy in pregnancy due to their established safety and efficacy profiles, while nicardipine and amlodipine may be considered as second-line intervenous alternatives depending on the local drug availability.[6,16] Concurrent infectious and non-infectious diseases, specifically pneumonia and gestational diabetes, should be managed with appropriate antibiotics and continuous insulin infusion maintaining glucose at 140–180 mg/dL, as recommended for critically ill patients[3,17], to avoid further PrAKI deterioration. Other nephrotoxic agents, including NSAIDs, should also be avoided throughout the clinical course.[3] In this patient, after the delivery, adequate fluid management, and BP, glycemic, and source infection control, her kidney function recovered to normal in approximately two months as the urine output also increasing. Due to her significant renal improvement with stable clinical and metabolic parameters, including SCr, urine output, and fluid balance, we decided to discontinue hemodialysis. This decision aligned with the KDIGO 2012 recommendation to discontinue RRT when intrinsic kidney function has recovered sufficiently to meet patient needs.[18] In this case, preserving residual kidney function and maintaining adequate renal perfusion were the primary considerations guiding our decision. As the conclusion, the fundamental aspect of PrAKI management is supportive care to maintain renal perfusion. Through timely institution of multidisciplinary care, involving nephrologist and obstetrician, the patient achieved complete resolution of kidney function within two months.

CONCLUSION

The fundamental aspect of PrAKI management is supportive care to maintain renal perfusion. Through timely institution of multidisciplinary care, involving nephrologist and obstetrician, the patient achieved complete resolution of kidney function within two months

ACKNOWLEDGMENTS

None.

CONFLICT OF INTEREST
The authors have no conflicts of interest to declare.

CONSENT OF PARTICIPATE
Written informed consent was not required because the manuscript does not include identifiable patient information, photographs, or any images involving patient body parts.

AUTHORS' CONTRIBUTIONS
Conceptualization: KK, WW; Data curation: KK, WW, APW; Investigation: KK, WW, DP; Project administration: KK; Resources: DP; Supervision: WW, DP; Validation: WW, DP, APW; Visualization: DP, APW; Writing – original draft: KK, APW; Writing – review & editing: WW, DP. All authors approved the final version of the manuscript.

DATA AVAILABILITY
No new data were generated or analyzed in this study.

FUNDING
This study received no specific funding.

USE OF ARTIFICIAL INTELLIGENCE TOOLS
The authors did not use artificial intelligence–assisted technologies in the writing, analysis, or preparation of this manuscript.

REFERENCES

1. Taber-Hight E, Shah S. Acute Kidney Injury in Pregnancy. Adv Chronic Kidney Dis. 2020 Nov 1;27(6):455–60. doi:10.1053/j.ackd.2020.06.002 PubMed PMID: 33328061.
2. Meena P, Kattah A. Dialysis During Pregnancy: Are We Any Closer to Clarity? Kidney Int Rep. 2025 Jul 1;10(7):2100–2. doi:10.1016/j.ekir.2025.05.008
3. Nelson-Piercy C, Srisawat N, Kashani K, Lumlertgul N, Murugan R, Rhee H, et al. Pregnancy-associated acute kidney injury — consensus report of the 32nd Acute Disease Quality Initiative workgroup. Nat Rev Nephrol. 2025 Sep 1;21(9):633–46. doi:10.1038/s41581-025-00979-6 PubMed PMID: 40681846.
4. Hall DR, Conti-Ramsden F. Acute kidney injury in pregnancy including renal disease diagnosed in pregnancy. Best Pract Res Clin Obstet Gynaecol. 2019 May 1;57:47–59. doi:10.1016/j.bpobgyn.2018.12.005 PubMed PMID: 30661950.
5. Shah S, Verma P. Pregnancy-Related Acute Kidney Injury: Do We Know What to Do? Nephron. 2023 Feb 1;147:35–8. doi:10.1159/000525492 PubMed PMID: 35793648.
6. Deodhare KG, Pathak N. Hypertension and associated complications in pregnant women with chronic kidney disease. World J Nephrol. 2024 Dec 25;13(4):100680. doi:10.5527/wjn.v13.i4.100680
7. Prakash J, Ganiger VC. Acute Kidney Injury in Pregnancy-specific Disorders. Indian J Nephrol. 2017 Jul 1;27(4):258–70. doi:10.4103/0971-4065.202406
8. Szczepanski J, Griffin A, Novotny S, Wallace K. Acute Kidney Injury in Pregnancies Complicated With Preeclampsia or HELLP Syndrome. Front Med (Lausanne). 2020 Feb 7;7(22):1. doi:10.3389/fmed.2020.00022
9. Moronge D, Sullivan JC, Faulkner JL. Physiology of Pregnancy-related Acute Kidney Injury. Compr Physiol. 2023 Jul 1;13(3):4869–78. doi:10.1002/cphy.c220026 PubMed PMID: 37358509.
10. Gupta A, Ferguson J, Rahman M, Weber-Shrikant E, Venuto R. Acute oliguric renal failure in HELLP syndrome: Case report and review of literature. Ren Fail. 2012 Jun;34(5):653–6. doi:10.3109/0886022X.2012.660856 PubMed PMID: 22364519.

11. Rao S, Jim B. Acute Kidney Injury in Pregnancy: The Changing Landscape for the 21st Century. Kidney Int Rep. 2018 Mar 1;3(2):247–57. doi:10.1016/j.ekir.2018.01.011
12. Gorayeb-Polacchini FS, Moura AF, Luders C, Moura-Neto JA, Leme JEG, Reis da Silva D. Pregnancy in patients with chronic kidney disease undergoing dialysis. Vol. 46. 2024;46(4):e20240067.
13. Liu Q, Ling G jun, Zhang S quan, Zhai W qing, Chen Y juan. Effect of HELLP syndrome on acute kidney injury in pregnancy and pregnancy outcomes: a systematic review and meta-analysis. BMC Pregnancy Childbirth. 2020 Dec 1;20(1):657. doi:10.1186/s12884-020-03346-4 PubMed PMID: 33126866.
14. Wiles K, Skinner L, Smith P. Renal disease in pregnancy. Medicine. 2023 Apr;51(4):293–9. doi:10.1111/tog.12257
15. Ribeiro CI, Silva N. Pregnancy and dialysis. Brazilian Journal of Nephrology. 2020;42(3):349–56. doi:10.1590/2175-8239
16. Conti-Ramsden F, de Marvao A, Chappell LC. Pharmacotherapeutic options for the treatment of hypertension in pregnancy. Expert Opin Pharmacother. 2024;25(13):1739–58. doi:10.1080/14656566.2024.2398602 PubMed PMID: 39225514.
17. American Diabetes Association Professional Practice Committee for Diabetes. 16. Diabetes Care in the Hospital: Standards of Care in Diabetes-2026. Diabetes Care. 2026;49(Suppl. 1):S339–55. doi:10.2337/dc26-S016 PubMed PMID: 41358892.
18. Kellum JA, Lameire N, Aspelin P, Barsoum RS, Burdmann EA, Goldstein SL, et al. KDIGO Clinical Practice Guideline for Acute Kidney Injury. Kidney Int Suppl (2011). 2012;2(1):1–138. doi:10.1038/kisup.2012.1