Case Report: Pre-eclampsia with Low Sodium Syndrome (PALS) in a Patient with Chronic Kidney Disease

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Background

Preeclampsia is a multifaceted hypertensive disorder of pregnancy, traditionally defined by new-onset hypertension after 20 weeks of gestation accompanied by proteinuria or systemic involvement in the absence of other causes [1,2]. Although its classical presentation, hypertension, proteinuria, and potential end-organ dysfunction, is widely recognised, atypical manifestations warrant heightened clinical awareness. One such atypical variant is Preeclampsia with Low Sodium Syndrome (PALS), in which hyponatraemia coexists with severe features of preeclampsia.

Hyponatraemia during preeclampsia remains rare but has increasingly been reported in the literature, with an estimated incidence of 14.6% among women with preeclampsia compared to no cases in normotensive pregnancies [3]. This phenomenon is associated with severe clinical features, including HELLP syndrome, renal dysfunction, low birth weight, and increased neonatal morbidity [4,5]. Neurological complications, such as seizures and cerebral oedema, underline the importance of prompt recognition and intervention [5].

The underlying mechanisms of PALS are not fully understood but are thought to involve a syndrome of inappropriate antidiuretic hormone secretion (SIADH), triggered by stress, pain, or defective placental vasopressinase production [6]. Additionally, nephrotic syndrome, common in preeclampsia, may exacerbate fluid imbalances through hypoproteinaemia and non-osmotic ADH release [5]. Management strategies centre on maternal stabilisation through fluid restriction, diuretic therapy, and, in severe cases, hypertonic saline. Ultimately, timely delivery remains the definitive cure for both preeclampsia and hyponatraemia, with sodium levels typically normalising 48–72 hours postpartum [1,8].

Case Description

A 20-year-old woman (G1P0) at 21 weeks' gestation was referred to a tertiary centre for severe preeclampsia in the setting of chronic kidney disease. She had a known family history of renal failure requiring dialysis but had not undergone a comprehensive nephrological workup prior to pregnancy.

The patients clinical findings included both hypertension and proteinuria, presenting with blood pressure readings consistently above 160/110 mmHg, accompanied by nephrotic-range proteinuria (4–5 g/day). This was associated with renal dysfunction, marked by a serum creatinine that was 81 µmol/L on admission and showed persistent elevations. The clinical picture was complicated by severe hyponatraemia, the patients serum sodium was critically low (117 mmol/L), classified as hypervolaemic hyponatraemia. Clinically, she exhibited facial oedema, thirst, and mild pitting oedema. The initial management of this patient included antihypertensives, fluid balance and fetal monitoring. In terms of antihypertensives she was commenced on methyldopa and nifedipine to control blood pressure. To address the fluid balance, strict fluid restriction was advised to counter SIADH-like physiology. Diuretic therapy was employed but yielded minimal improvement in serum sodium levels (range: 117–123 mmol/L). In regards to the fetus there were no ultrasonographic evidence of significant fetal growth restriction. However, the fetus was small for gestational age (320 g) at delivery. Despite ongoing treatment, maternal condition worsened, culminating in the decision to medically terminate the pregnancy at 22 weeks. A fetus, weighing 320 g, was delivered vaginally.

The patient exhibited elevated creatinine levels, reaching a peak of 102 µmol/L postpartum. During pregnancy, proteinuria remained at nephrotic levels, indicating significant renal impairment. Postpartum, renal function showed partial improvement, with creatinine levels stabilising between 69–81 µmol/L and an estimated glomerular filtration rate (eGFR) of 59–90 mL/min.

Autoimmune screening, including tests for antinuclear antibodies (ANA), anti-double-stranded DNA (anti-dsDNA), and antiphospholipid antibodies, returned negative results. Thrombophilia screening identified a heterozygous prothrombin mutation, while lupus anticoagulant and Factor V Leiden mutation were absent.
A renal ultrasound was performed, revealing no structural abnormalities or evidence of renal vein thrombosis. The patient's hyponatraemia resolved within 72 hours following pregnancy termination. Although renal function improved post-delivery, it did not return to baseline levels, confirming a diagnosis of chronic kidney disease.

Discussion

This case underscores the severe and atypical nature of PALS, particularly when superimposed on pre-existing chronic kidney disease. While preeclampsia itself poses a significant risk to maternal renal function, underlying CKD can both obscure and exacerbate the clinical picture.
Management primarily involves restricting fluid intake as the first-line approach to addressing SIADH-induced hyponatraemia. Diuretic therapy may be utilised to help reduce fluid overload; however, careful monitoring is required to prevent haemodynamic instability.
In cases where sodium levels are critically low, particularly when accompanied by neurological symptoms, hypertonic saline may be administered. Ultimately, delivery remains the definitive treatment, as the resolution of the placenta-driven pathology typically leads to the normalisation of sodium levels postpartum [5,6].
In the present case, the severity of both renal dysfunction and refractory hyponatraemia prompted termination of pregnancy at 22 weeks. Postpartum follow-up revealed partial reversal of the renal impairment, though persistent CKD was confirmed. This outcome aligns with evidence suggesting that preeclampsia superimposed on CKD may lead to irreversible renal injury [5].

Conclusion

This case highlights the complexity of managing an atypical presentation of preeclampsia, Preeclampsia with Low Sodium Syndrome, in a patient with underlying chronic kidney disease. The refractory hyponatraemia and renal compromise underscore the importance of early recognition, rigorous monitoring, and timely intervention. Future pregnancies should be guided by a multidisciplinary care plan that addresses both obstetric and renal risks. Further research is needed to elucidate the pathophysiology of PALS and to refine management strategies for this rare but potentially life-threatening condition.

References

1. Espinoza J., et al. Gestational Hypertension and Preeclampsia. The American College of Obstetricians and Gynaecologists. 2020 Jun;135(6). doi:10.1097/aog.0000000000003891
2. Hypertension in pregnancy: Diagnosis and management: Guidance [Internet]. [cited 2025 Mar 18]. Available from: https://www.nice.org.uk/guidance/cg107
3. Sompolinsky Y, Hagai A, Remer C. 562: Hyponatremia among preeclampsia patients - feature of severity - American Journal of Obstetrics & Gynecology [Internet]. 2020 [cited 2025 Mar 18]. Available from: https://www.ajog.org/article/S0002-9378(19)31948-9/fulltext
4. Montebello A, Thake J, Vella S, Vassallo J. Pre-eclampsia complicated by severe hyponatraemia. BMJ Case Reports. 2020 Dec;13(12). doi:10.1136/bcr-2020-237827
5. Sandhu G, Ramaiyah S, Chan G, Meisels I. Pathophysiology and management of preeclampsia-associated severe hyponatremia. American Journal of Kidney Diseases. 2010 Mar;55(3):599–603. doi:10.1053/j.ajkd.2009.09.027
6. Powel JE, Rosenthal E, Roman A, Chasen ST, Berghella V. Preeclampsia and low sodium (PALS): A case and systematic review. European Journal of Obstetrics & Gynecology and Reproductive Biology. 2020 Jun;249:14–20. doi:10.1016/j.ejogrb.2020.03.052