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CASE REPORT

REVERSIBLE HYPONATREMIC ENCEPHALOPATHY TRIGGERED BY DIURETIC-INDUCED DYSELECTROLYTEMIA IN STAGE IIIB CKD: A CAUTIONARY CASE REPORT

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ABSTRACT:

Electrolyte imbalances are a major concern for chronic kidney disease (CKD) patients, particularly with diuretic use. Unmonitored loop and thiazide diuretics can cause severe fluid and electrolyte disturbances, leading to acute neurological and metabolic problems. Timely identification and correction of these imbalances are essential to prevent morbidity and mortality in patients with impaired kidney function.

We present a 65-year-old male with stage 3b chronic kidney disease, type 2 diabetes, hypertension, and a stroke history. After 7 days of severe diarrhea, reduced food intake, and increasing weakness, he experienced an acute change in mental status. Upon admission, he exhibited encephalopathy, rapid breathing, and dehydration. Lab results indicated severe hyponatremia (Na^+127 mmol/L), hypokalemia (K^+3.1 mmol/L), hypochloremia, and elevated serum creatinine (6.6 mg/dL), along with high-anion-gap metabolic acidosis. His medication review revealed unsupervised use of furosemide and hydrochlorothiazide for leg edema, contributing to his electrolyte imbalances and metabolic instability.

This case emphasizes the nephrotoxic risks associated with excessive diuretic use in individuals with impaired kidney function and demonstrates the critical need for careful monitoring of electrolytes and tailored medication strategies in the management of chronic kidney disease (CKD). It underscores the importance of collaborative efforts across disciplines to avert drug-induced metabolic disturbances and to maintain both renal and neurological stability. This case underscores the nephrotoxic risks of excessive diuretic use in patients with impaired kidney function, highlighting the need for careful electrolyte monitoring and tailored medication strategies in CKD management.

KEY WORDS: Chronic Kidney Disease; Diuretics; Dyselectrolytemia; Encephalopathy; Hyponatremia; Metabolic Acidosis.

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INTRODUCTION

Hyponatremia, characterized by a serum sodium level falling below 135 mEq/L, represents the most common electrolyte imbalance observed in clinical settings. Research suggests that it impacts around 20% to 35% of patients admitted to hospitals.[1] In the elderly population, the occurrence may reach up to 24.7%.[2] Although mild cases of hyponatremia can be asymptomatic, more severe or swiftly progressing instances can result in serious neurological issues, such as confusion, seizures, and coma, primarily due to cerebral edema caused by osmotic water movement into brain cells.[3] The causes of hyponatremia are diverse, including factors such as the syndrome of inappropriate antidiuretic hormone secretion (SIADH), fluid loss, heart failure, kidney disorders, and the administration of specific drugs, particularly diuretics.[4] For example, SIADH is characterized by the excessive secretion of antidiuretic hormone, which results in water retention and dilutional hyponatremia.[5]

Individuals suffering from chronic kidney disease (CKD), particularly in advanced phases such as CKD stage IIIB, exhibit a heightened vulnerability to

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electrolyte disturbances, notably hyponatremia. The compromised renal function characteristic of CKD reduces the kidneys' capacity to eliminate excess water and maintain sodium equilibrium, consequently elevating the likelihood of hyponatremia.[1] Furthermore, the occurrence of hyponatremia among CKD patients correlates with fluid overload and negative consequences, including a rise in mortality rates.[6] Diuretic-induced hyponatremia represents a considerable issue, especially concerning thiazide diuretics. Thiazides hinder the reabsorption of sodium and chloride in the distal convoluted tubule, resulting in diminished free water clearance and a risk of hyponatremia.[5] Conversely, loop diuretics target the thick ascending limb of the loop of Henle and are less prone to induce hyponatremia due to their role in decreasing medullary hypertonicity and the responsiveness to antidiuretic hormone.[4] Factors that increase the risk of thiazide-induced hyponatremia encompass advanced age, female sex, low body weight, and the simultaneous use of drugs that affect water excretion.[1]

Hyponatremic encephalopathy occurs as a result of swift decreases in serum sodium, which can cause cerebral oedema. The symptoms can vary from slight confusion to severe manifestations such as seizures and coma. [3] Timely diagnosis and careful adjustment of sodium levels are essential to avert complications, including osmotic demyelination syndrome (ODS), a disorder marked by demyelination in the central pontine area due to the rapid correction of chronic hyponatremia. According to European guidelines, it is advised to restrict serum sodium correction to no more than 10 mEq/L within 24 hours to mitigate the risk of ODS.[1] This case report emphasizes a patient diagnosed with CKD stage IIIB who experienced severe, but reversible, hyponatremic encephalopathy as a result of diuretic administration. This case illustrates the critical need to identify the risk of diuretic-induced hyponatremia in patients with CKD, as well as the necessity for diligent monitoring and management to avert negative consequences.

CASE DETAILS

This case was documented in May 2025 at a Tertiary Care Teaching Hospital, Guntur, Andhra Pradesh, India. A 65-year-old male patient, who is a retired educator with a documented history of type 2 diabetes mellitus (T2DM), essential hypertension (HTN), stage 3b chronic kidney disease (CKD), and a previous ischemic cerebrovascular accident (CVA), arrived at the emergency department exhibiting acute confusion and diminished responsiveness. His presenting symptoms included a 7-day history of profuse watery diarrhea, inadequate oral intake, generalised weakness, and intermittent low-grade fever. In the last 48 hours, the patient experienced altered mental status, disorientation regarding time and place, and displayed lethargy with slow verbal responses. Additional symptoms included a non-productive cough, worsening shortness of breath upon exertion, and swelling in both lower limbs. The patient's urinary output showed an initial increase in frequency, which was followed by oliguria in the past 24 hours.

A thorough assessment of his medication history disclosed a chronic intake of Telmisartan 20 mg, Metoprolol Succinate 25 mg, Glimepiride in conjunction with Metformin, and Pantoprazole. Furthermore, he had been intermittently self-administering loop diuretics (Furosemide) and hydrochlorothiazide over the last month for edema management, without any clinical supervision. No recent modifications in prescribed dosages or dietary practices were noted. He denied the consumption of alcohol or tobacco and maintained regular follow-ups for his comorbid conditions. His adherence to medication and dietary restrictions was reportedly good until the onset of his gastrointestinal symptoms. Upon clinical assessment, the patient presented as afebrile (98.6°F) yet exhibited signs of dehydration and mild respiratory distress. He was noted to be tachycardic (122 bpm), had an elevated blood pressure reading of 139/90 mmHg, and a respiratory rate of 28 breaths per minute. His oxygen saturation while breathing room air was recorded at 94%. The general examination revealed significant bilateral pitting pedal edema, periorbital swelling, and dry mucous membranes. Neurologically, he scored 13/15 on the Glasgow Coma Scale (GCS) and displayed indications of an acute confusional state without any lateralizing deficits. Pulmonary auscultation indicated the presence of basal crepitations bilaterally, while the cardiovascular examination was largely unremarkable aside from the noted sinus tachycardia. Both the abdominal and musculoskeletal examinations showed no signs of focal tenderness or rigidity.

Laboratory investigations indicated significant hyponatremia (Na^+ 127 mmol/L), hypokalemia (K^+ 3.1 mmol/L), and hypochloremia, accompanied by an increased serum creatinine level of 6.6 mg/dL, which suggests acute-on-chronic kidney injury. Arterial blood gas analysis revealed a low serum bicarbonate level alongside a high anion gap metabolic acidosis. The complete blood count indicated leukocytosis (WBC 10,300 cells/mm^3). Urinalysis demonstrated mild proteinuria without any active sediment. A chest radiograph showed bilateral mild interstitial infiltrates. Electrocardiography revealed sinus tachycardia without any signs of ischemic changes or QT prolongation. The clinical and biochemistry investigations aligned with a diagnosis of drug-induced dyselectrolytemia accompanied by metabolic acidosis, likely triggered by the excessive administration of diuretics in a patient suffering from pre-existing chronic kidney disease (CKD), which was further exacerbated by volume depletion resulting from ongoing diarrhoea. An initial provisional diagnosis of acute symptomatic

hyponatremia and hypokalemia, along with pre-renal azotemia and metabolic acidosis, was established.

The patient was started on immediate correction with intravenous hypertonic saline (3% NaCl) to treat hyponatremia, in conjunction with oral potassium chloride supplementation. Loop and thiazide diuretics were promptly halted. To manage the underlying metabolic acidosis, oral sodium bicarbonate therapy was initiated. Oxygen support was provided through nasal prongs, and rigorous monitoring of fluid balance, daily serum electrolytes, renal function, and neurologic status was established. Supportive care involved the commencement of a renal-safe antihypertensive, Telmisartan, and his diabetic regimen was adjusted to incorporate a combination of Dapagliflozin and Metformin, taking into account its renal protective properties. Gastric prophylaxis was altered to Rabeprazole and Domperidone, and multivitamin supplementation was introduced to mitigate potential nutritional deficiencies. In the following 72 hours, the patient exhibited a progressive neurological recovery, achieving full restoration of both orientation and consciousness. His serum sodium levels increased to 132 mmol/L, potassium levels normalised to 3.7 mmol/L, and creatinine levels decreased to 5.3 mg/dL. Bowel movements became stable, and his urine output was sufficient. There was a significant clinical enhancement in volume status, accompanied by the resolution of peripheral edema. Throughout the hospital stay, he maintained hemodynamic stability.

On the sixth day, he was discharged in a stable state. His vital signs at the time of discharge were within normal vitals: Blood Pressure of 128/80 mmHg, Heart Rate of 82 bpm, Respiratory Rate of 18 cycles/min, SpO_2 96% on room air, and a temperature of 98.4°F. There was a notable improvement in renal function, with serum creatinine levels at 4.9 mg/dL, and serum electrolytes had returned to normal ranges. The discharge medication regimen included Metoprolol Succinate 25 mg once daily, Clopidogrel + Aspirin 75/10 mg once daily, Rabeprazole + Domperidone twice daily before meals, Dapagliflozin + Metformin twice daily after meals, a Multivitamin Tablet once daily, Sodium Bicarbonate 650 mg three times a day, and Bromhexin + Guanfenesin syrup for the remaining cough. Atorvastatin 10 mg was recommended only as necessary, under strict medical supervision. The patient and caregivers received education regarding the significance of adhering to fluid and salt limitations, the necessity of closely monitoring renal parameters and electrolytes, and the importance of avoiding unsupervised diuretic use. He was arranged for a follow-up appointment with nephrology within 10 days and instructed to report immediately if there was a recurrence of confusion, a decrease in urine output, or any symptoms indicative of fluid overload.

DISCUSSION

This case underscores the significant repercussions of unsupervised diuretic administration in a patient diagnosed with CKD stage 3b, culminating in severe hyponatremia, hypokalemia, metabolic acidosis, and encephalopathy. The occurrence of diuretic-induced dyselectrolytemia is a thoroughly documented issue, particularly prevalent among elderly individuals with prior renal impairment. [7] The interaction of volume depletion, impaired renal function, and unregulated diuretic treatment formed a critical situation that resulted in acute neurological manifestations in this patient. Similar case presentations have been reported in earlier research. For instance, Fried et al. in 1997 recognised thiazide-induced hyponatremias as a frequent reason for hospitalisation among elderly patients, especially those with diminished renal reserve. [8] The present case included the administration of both loop and thiazide diuretics, which increased the risk. While loop diuretics generally disrupt medullary concentration gradients and encourage natriuresis, thiazides hinder the reabsorption of sodium and chloride in the distal tubules, frequently resulting in more severe hyponatremia. [3]

The emergence of encephalopathy in this patient highlights the critical nature of acute hyponatremia. According to Sterns' findings in 2015, a swift decrease in serum sodium levels can lead to cerebral edema, which may present as changes in mental status, seizures, and even coma. [1] In this particular instance, the patient exhibited a serum sodium concentration of 127 mmol/L along with altered consciousness; however, his neurological symptoms were reversible following a gradual adjustment of sodium levels. This aligns with the guidelines that recommend restricting sodium correction to no more than 10 mmol/L within 24 hours to prevent

Timeline Table

Timeframe	Clinical Events/Interventions
Day of Admission (May 17, 2025)	Confusion, encephalopathy, severe dyselectrolytemia diagnosed
Day 1-3 (17-05-2025 to 19-05-2025)	IV 3% NaCl, K^+ supplements, bicarbonate, and diuretics stopped
Day 4–6 (20-05-2025 to 22-05-2025)	Neurological improvement, biochemical normalisation, and discharge planning
Day 6 (Discharge) (22-05-2025)	Asymptomatic, stable renal and electrolyte status

osmotic demyelination syndrome. [9] Furthermore, the existence of high-anion-gap metabolic acidosis indicates the cumulative impact of gastrointestinal fluid loss alongside impaired renal acid excretion. In individuals with chronic kidney disease (CKD), the capacity to produce bicarbonate and eliminate acid is diminished, and concurrent diarrhoea worsens this imbalance. [10] The prompt commencement of sodium bicarbonate treatment played a significant role in achieving metabolic stabilisation and enhancing renal function in our case.

The phenomenon of polypharmacy, coupled with insufficient medical supervision, significantly contributed to the pathogenesis. Research indicates that individuals with chronic kidney disease (CKD) frequently engage in self-medication or receive inadequate guidance regarding the nephrotoxic risks associated with over-the-counter drugs, such as diuretics. [11] In this particular case, the educational efforts and counselling provided at the time of discharge concentrated on preventive measures, which are consistent with the recommendations set forth by KDIGO (kidney disease: Improving Global Outcomes). These guidelines underscore the importance of patient education in reducing drug-related nephrotoxicity in CKD. [12]

The patient's positive response to treatment, characterised by the return of consciousness and the normalisation of electrolyte levels, illustrates the possibility of complete recovery through timely and suitable intervention. This case underscores the importance of diligent electrolyte monitoring in patients with chronic kidney disease (CKD), especially in those who have a background of diuretic usage and loss of gastrointestinal fluids. Collaborative efforts among physicians, pharmacists, and caregivers are crucial to avert recurrence and enhance patient outcomes.

CONCLUSION

This case highlights the significant risks associated with the unsupervised use of diuretics in individuals suffering from moderate to advanced chronic kidney disease. In such patients, the compromised ability of the kidneys to manage electrolytes can swiftly lead to severe dyselectrolytemia and neurological impairment. The combined impact of volume depletion, existing chronic kidney disease, and the use of two diuretics together resulted in acute symptomatic hyponatremia, hypokalemia, and metabolic acidosis, ultimately leading to reversible encephalopathy.

The prompt identification of symptoms, the withdrawal of offending agents, and the precise correction of electrolyte and acid-base imbalances resulted in complete neurological recovery, underscoring the potential for reversibility of these complications when intervention is timely. This case underscores the significance of personalised diuretic management, regular monitoring of electrolytes, and educating patients to avert preventable iatrogenic incidents in the management of CKD.

Learning Points:

- Hyponatremic encephalopathy caused by diuretics can manifest suddenly and may be reversible, even in patients with chronic kidney disease (CKD) at stage 3b.
- The unsupervised use of both loop and thiazide diuretics markedly heightens the risk of electrolyte imbalances.
- Timely and specific correction of sodium levels and acid-base equilibrium can lead to complete neurological recovery.
- Educating patients and their caregivers is essential to avert the recurrence of issues in CKD patients undergoing diuretic treatment.

DECLARATIONS

Patient Perspective: The patient and his family expressed their gratitude for the medical care and counselling received during hospitalisation. They acknowledged that the episode was primarily due to the unsupervised use of diuretics and emphasised their commitment to adhering strictly to prescribed medications and regular follow-ups. The patient reported complete recovery with improved awareness of the importance of monitoring electrolyte levels and avoiding self-medication in the future.

Ethical Approval and Consent: This case report has been compiled in alignment with the ethical standards set forth by the institution and the Declaration of Helsinki. The patient provided written informed consent for the publication of this case report along with any related clinical details or images. The patient has been assured that all identifying information will be anonymised to ensure confidentiality.

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CONFLICT OF INTEREST
Authors declare no conflict of interest.
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AUTHORS' CONTRIBUTION

The following authors have made substantial contributions to the manuscript as under:

Conception or Design:	PKY, RRN
Acquisition, Analysis or Interpretation of Data:	PKY, RRN, DLK
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All the authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.