J Nephropharmacol. 2024; 13(2): e11674.

https://jnephropharmacology.com DOI: 10.34172/npj.2024.11674

Journal of Nephropharmacology

Management of severe hyponatremia in a chronic kidney disease patient; a case report

Anass Qasem[1*], Syed Arman Rabbani[2], Martin Thomas Kurian[1], Sathvik B Sridhar[2]

[1]Department of Nephrology, Ibrahim Bin Hamad Obaidullah Hospital, Ras Al-Khaimah, United Arab Emirates
[2]Department of Clinical Pharmacy and Pharmacology, RAK College of Pharmacy, RAK Medical and Health Sciences University, Ras al Khaimah, United Arab Emirates

ARTICLE INFO

Article Type:
Case Report

Article History:
Received: 14 Jan. 2024
Accepted: 3 Jun. 2024
ePublished: 29 Jun. 2024

Keywords:
Hyponatremia
Chronic kidney disease
Hemodialysis
Hypertonic saline

ABSTRACT

Hyponatremia is a common electrolyte abnormality in chronic kidney disease (CKD). Managing severe hyponatremia in CKD is challenging, requiring the correction of biochemical imbalances and fluid overload, often through traditional hemodialysis. However, this can lead to rapid serum sodium correction, potentially causing neurological complications. We present a case of a CKD patient with a suspected stroke, who exhibited dizziness, confusion, and an unsteady gait, and was found to have azotemia, metabolic acidosis, and severe hyponatremia (101 mmol/L). We managed the case by gradually correcting the serum sodium with hypertonic saline. Subsequently, we initiated traditional hemodialysis using a dialysate sodium concentration of 130 mEq/L when the serum sodium levels increased to 122 mmol/L. The patient was discharged in a vitally and hemodynamically stable condition, with a serum sodium level of 137 mmol/L. This report highlights the intricacies of managing severe hyponatremia in a CKD patient with a suspected stroke.

Case Report

Implication for health policy/practice/research/medical education:
In this case report, we found, slow and gradual correction of serum sodium and subsequent use of traditional hemodialysis with low sodium dialysate offers a viable strategy to manage chronic kidney disease patients with severe hyponatremia.
Please cite this paper as: Qasem A, Rabbani SA, Kurian MT, Sridhar SB. Management of severe hyponatremia in a chronic kidney disease patient; a case report. J Nephropharmacol. 2024;13(2):e11674. DOI: 10.34172/npj.2024.11674.

Introduction

Hyponatremia, defined as a serum sodium concentration of below 135 mmol/L, represents a commonly encountered electrolyte abnormality in various clinical conditions, including chronic kidney disease (CKD) (1,2). The decreased ability of the kidneys to regulate water and sodium excretion predisposes patients with CKD to hyponatremia (3). Managing CKD patients with severe hyponatremia presents significant clinical challenges. It requires correction of biochemical imbalances, azotemia, and fluid overload, which can be achieved through traditional hemodialysis (4). However, this approach can lead to a rapid correction of serum sodium, potentially leading to neurological complications, including seizures and osmotic demyelination syndrome (ODS). Management can be further complicated by the presence of cardiovascular (5) and cerebrovascular comorbidities (6). Continuous renal replacement therapy (CRRT) is often the preferred treatment for such cases due to its gradual correction capabilities (7). However, its use may be limited by the patient's clinical condition and its availability in healthcare settings.

We report the management of severe hyponatremia in a CKD patient with suspected stroke, presenting with dizziness, confusion, and unsteady gait along with metabolic acidosis and azotemia.

Case Presentation

A 55-year-old male with a known medical history of hypertension, diabetes mellitus, and gout, was brought to the emergency department (ED) with complaints of dizziness, confusion, and an unsteady gait. The patient exhibited these symptoms for approximately three and a half hours before presentation. Furthermore, he had hiccups, headache, weakness, and slurred speech. There was no history of loss of consciousness, urinary or bowel incontinence, convulsions, or fever.

On admission (day 0) the patient's vital signs were as

***Corresponding author**: Anass Qasem, Email: anass.qasem@ehs.gov.ae

follows: temperature 36.4 °C, heart rate 86 beats per minute, respiratory rate 18 breaths per minute, blood pressure 156/80 mm Hg, and oxygen saturation 98% on room air. Physical assessment revealed pallor, lower limb pitting edema, and crepitations in the left lower lung area. His laboratory investigations revealed a critically low-serum sodium level of 101 mmol/L along with a serum creatinine level of 889.2 µmol/L and an estimated glomerular filtration rate (eGFR) of 5 mL/min/1.73 m^2. Additionally, the patient exhibited metabolic acidosis (pH-7.2, PCO2- 29 mmHg, PO2- 22 mmHg, HCO3-12.5 mmol/L) and elevated levels of creatine kinase (CK) at 5890 U/L and myoglobin at 19860 ng/mL.

Ultrasound of the kidneys, ureters, and bladder (KUB) revealed bilateral grade III nephropathy and mild ascites. Furthermore, a multi-slice non-contrast computerized tomography (CT) scan of the brain revealed a defined sub-cortical hypo-dense area in the left occipital region, a tiny focus of low attenuation in the left pons, and early ischemic white matter changes in the periventricular areas. The CT findings were suggestive of acute as well as chronic ischemic changes in the brain. Thrombolytic therapy was not initiated as the patient presented outside the optimal time window. Subsequently, conservative management for stroke was initiated.

The patient was shifted to the intensive care unit (ICU) for the management of severe hyponatremia. The patient was started on hypertonic saline (sodium chloride 3%) at rate of 50 mL/h under close observation with target serum sodium correction of 8 mmol/L in the first 12 hours and a total of 12 mmol/L in 24 hours. The ICU care plan focused on a slow and meticulous correction of hyponatremia, with frequent sodium levels monitoring every 2 hours. Over the first 3 days of admission, the patient serum sodium gradually increased with minimal change of serum creatinine level.

On day 4th of the admission, the patient showed considerable improvement and was subsequently transferred to the high dependency unit (HDU). At this point, the serum sodium levels had risen to 122 mmol/L. Hemodialysis was initiated through a right double lumen temporary femoral hemodialysis catheter, utilizing dialysate with a low sodium of 130 mmol/L, potassium 2 mmol/L, calcium 1.25 mmol/L, magnesium 0.5 mmol/L with blood flow rate of 200 mL/min and dialysate flow rate of 600 mL/min. The patient's sodium increased to 126 mmol/L at the end of the dialysis. The serum sodium levels of the patient did not change between the dialysis treatments and during the next hemodialysis, serum sodium level raised to 128 mmol/L. There was complete resolution of patient symptoms with no neurological deficits. Thereafter, the patient was put on maintenance hemodialysis through right internal jugular permcath, which was inserted on day 10th of the admission. On day 12th of the admission, the patient was discharged in a vitally and hemodynamically stable condition with serum sodium of 137 mmol/L (Figures 1 and 2).

Discussion

Management of severe hyponatremia in a patient with CKD, particularly when complicated by suspected cerebrovascular events requires careful considerations of both the clinical condition of the patient and the available medical resources. Rapid correction of hyponatremia, whether through quick hypertonic saline or traditional hemodialysis (using a dialysate with elevated sodium levels) can lead to ODS. This condition is primarily driven by the swift movement of water and electrolytes in the brain, a response to sudden shifts in serum osmolarity (8). ODS development following overcorrection of sodium by traditional hemodialysis is well documented (9). Therefore, the international guidelines recommend

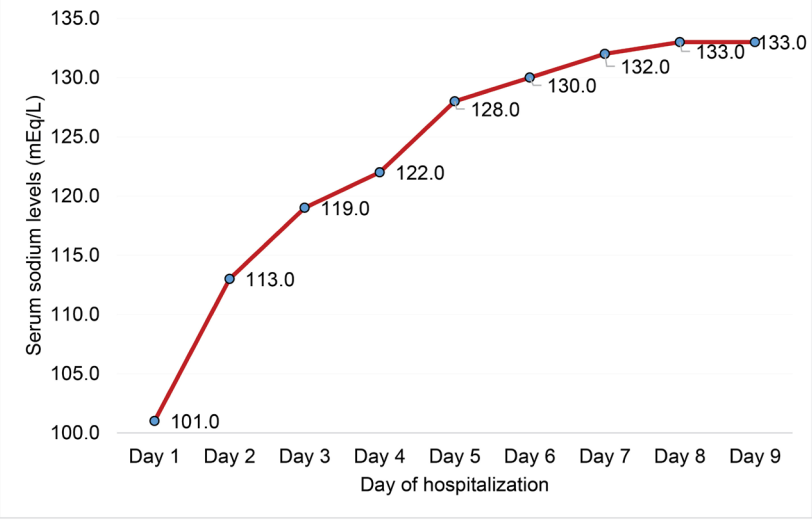

Figure 1. Slow and gradual correction of serum sodium levels.

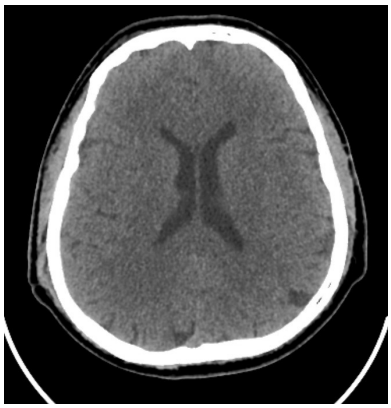

Figure 2. CT of brain without contrast.

that serum sodium correction should not exceed 10 to 12 mEq/L and 18 mEq/L within the first 24 and 48 hours, respectively (10).

Considering the aforementioned factors, it is imperative to adopt a cautious and gradual approach in correcting severe hyponatremia in CKD patients, as in patients with normal renal function. In our patient, we achieved this through a dual strategy. Initially, the patient received slow and gradual hypertonic saline with a target serum sodium correction of 8 mmol/L in the first 12 hours with a total of 12 mmol/L in 24 hours. Subsequently, hemodialysis was initiated only after the serum sodium of the patient reached 122 mmol/L, employing a dialysate with a reduced sodium concentration of 130 mmol/L. This approach completely resolved the patient symptoms with no neurological deficits. CRRT serves as an optimal approach for managing these types of patients. However, our hospital did not have CRRT facility, therefore, given the clinical condition of our patient and the absence of CRRT facility necessitated the use of the mentioned approach.

Previous studies have utilized similar approaches for the management of severe hyponatremia in CKD and end-stage renal disease (ESRD) patients. Pattanashetti et al successfully managed severe hyponatremia in two CKD patients with advanced azotemia using conventional hemodialysis. They employed a dialysate sodium concentration of 128 mEq/L in a resource-limited facility without CRRT (11). In a similar case, Wendland et al corrected the serum sodium level of a patient with ESRD from 112 mEq/L over the course of three hemodialysis sessions. They utilized a dialysate sodium concentration of 130 mEq/L and a blood flow rate to 50 mL/min (12).

Conclusion

In conclusion, slow and gradual correction of serum sodium and subsequent use of traditional hemodialysis with low sodium dialysate offers a viable strategy to manage CKD patients with severe hyponatremia. This approach minimizes the risks of sodium overcorrection and neurological complications, particularly in settings with limited resources.

Authors' contribution

Conceptualization: Anass Qasem, Syed Arman Rabbani, Martin Thomas Kurian, Sathvik B Sridhar.
Data curation: Anass Qasem, Martin Thomas Kurian.
Formal analysis: Syed Arman Rabbani, Sathvik B Sridhar.
Investigation: Anass Qasem, Martin Thomas Kurian.
Validation: Syed Arman Rabbani, Sathvik B Sridhar.
Writing–original draft: Anass Qasem, Syed Arman Rabbani, Martin Thomas Kurian, Sathvik B Sridhar.
Writing–review & editing: Anass Qasem, Syed Arman Rabbani.

Conflicts of interest

The authors declare that they have no competing interests.

Ethical issues

This case report was conducted in accordance with the World Medical Association Declaration of Helsinki. The patient has provided written informed consent for publication as a case report. Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors.

Funding/Support

None.

References

1. Arzhan S, Lew SQ, Ing TS, Tzamaloukas AH, Unruh ML. Dysnatremias in Chronic Kidney Disease: Pathophysiology, Manifestations, and Treatment. Front Med (Lausanne). 2021;8:769287. doi: 10.3389/fmed.2021.769287
2. Inoue M, Nakai K, Tanaka S, Mitsuiki K, Tokumoto M, Tsuruya K, et al. Prevalence of hyponatremia and associated factors in patients with chronic kidney disease: the Fukuoka Kidney Disease Registry (FKR) study. Clin Exp Nephrol. 2023;27:1023-1031. doi: 10.1007/s10157-023-02395-1
3. Kovesdy CP. Significance of hypo- and hypernatremia in chronic kidney disease. Nephrol Dial Transplant. 2012;27:891-8. doi: 10.1093/ndt/gfs038
4. Zhang R, Wang S, Zhang M, Cui L. Hyponatremia in patients with chronic kidney disease. Hemodial Int. 2017;21:3-10. doi: 10.1111/hdi
5. Breen T, Brueske B, Sidhu MS, Murphree DH, Kashani KB, Barsness GW, et al. Abnormal Serum Sodium is Associated With Increased Mortality Among Unselected Cardiac Intensive Care Unit Patients. J Am Heart Assoc. 2020;9:e014140. doi: 10.1161/JAHA.119.014140
6. Barkas F, Anastasiou G, Liamis G, Milionis H. A step-by-step guide for the diagnosis and management of

hyponatraemia in patients with stroke. Ther Adv Endocrinol Metab. 2023;14:20420188231163806. doi: 10.1177/20420188231163806
7. Tandukar S, Kim C, Kalra K, Verma S, Palevsky PM, Puttarajappa C. Severe Hyponatremia and Continuous Renal Replacement Therapy: Safety and Effectiveness of Low-Sodium Dialysate. Kidney Med. 2020;2:437-449. doi: 10.1016/j.xkme.2020.05.007.
8. Voets PJGM, Maas RPPWM, Vogtländer NPJ, Kaasjager KAH. Osmotic demyelination syndrome and thoughts on its prevention. J Nephrol. 2022;35:339-342. doi: 10.1007/s40620-021-01081-3
9. Huang WY, Weng WC, Peng TI, Ro LS, Yang CW, Chen KH. Central pontine and extrapontine myelinolysis after rapid correction of hyponatremia by hemodialysis in a uremic patient. Ren Fail. 2007;29:635-8. doi: 10.1080/08860220701392314
10. Pasovski G, Vanholder R, Allolio B, Annane D, Ball S, Bichet D, et al. Hyponatraemia Guideline Development Group. Clinical practice guideline on diagnosis and treatment of hyponatraemia. Eur J Endocrinol. 2014;170:G1-47. doi: 10.1530/EJE-13-1020
11. Pattanashetti N, Bharati J, Kohli HS, Gupta KL, Ramachandran R. Successful management of severe hyponatremia in CKD-VD: In a cost limited setting. Hemodial Int. 2019;23:E69-E71. doi: 10.1111/hdi.12716
12. Wendland EM, Kaplan AA. A proposed approach to the dialysis prescription in severely hyponatremic patients with end-stage renal disease. Semin Dial. 2012;25:82-5. doi: 10.1111/j.1525-139X.2011.00981.x