Hyponatremia and Rapid Autocorrection in High-Frequency Ketamine Use: A Case for Monitoring Sodium in Chronic Ketamine Patients

Mohamed E. Ahmed MD, Eric Rosenthal DO

Affiliations: UMASS Health Alliance hospital, Fitchburg Family practice



Introduction or Background

Ketamine is prescribed for pain refractory to traditional analgesics. Chronic ketamine therapy is monitored for urologic and hepatobiliary toxicity but not for sodium and water balance.

Case presentation

39-year-old man on long-term ketamine who escalated to 15–20 rectal doses daily developed vomiting, diarrhea, and excessive water drinking

Investigation and workup

Initial labs showed Na level 122. Saline plus potassium corrected his sodium too fast (13 mmol/L/24h)

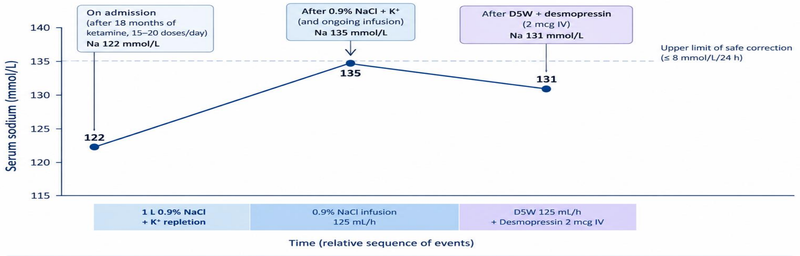
Management & Outcome

Saline was stopped and reversed with D5W and desmopressin, relowering Na to 131; here covered without brain injury and was counseled on safe ketamine and fluid use

Discussion

Ketamine can disturb water homeostasis through 3 opposing mechanisms: ketamine-precipitated SIADH, transient central diabetes insipidus and dose-related nausea and vomiting driving both fluid loss and compensatory free water intake. Because these mechanisms can move serum sodium in either direction, serum sodium warrants inclusion among screening tests in chronic ketamine therapy.

Figure 1. Serum sodium response to treatment

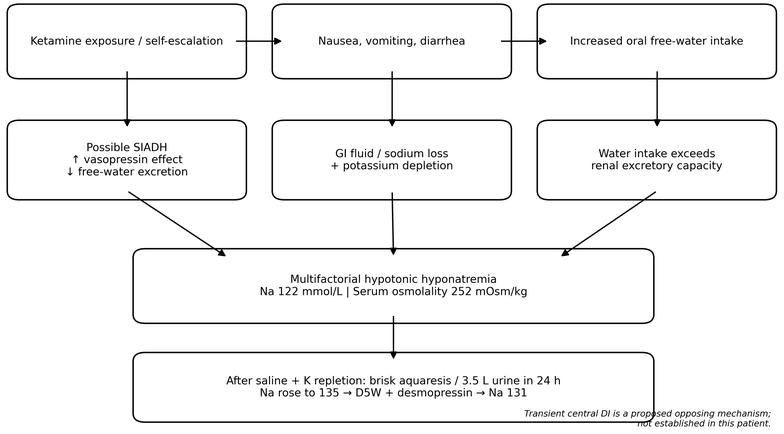


Key points:

- Admission sodium was 122 mmol/L (hypotonic hyponatremia).
- Sodium increased to 135 mmol/L (13 mmol/L rise) after 0.9% NaCl and potassium repletion (exceeding the recommended correction limit).
- Desmopressin (2 mcg IV) with D5W was given when sodium reached 135 mmol/L, and sodium was relowered to 131 mmol/L.

The graph shows the three documented sodium values in sequence. Exact elapsed times between measurements were not provided.

Potential Ketamine-Associated Disruption of Water Homeostasis



Conclusion/Implications/Lessons Learned

Conclusion

- Ketamine can shift serum sodium in either direction, resulting in inappropriate antidiuresis or transient central diabetes insipidus.
- Dose-related vomiting drives sodium loss and compensatory polydipsia.

Implication

- The hyponatremia was multifactorial, with every stimulus being rapidly reversible.
- Water loss overcorrected the sodium without any hypertonic saline, which risked osmotic demyelination.

Lessons learned

- Serum sodium should be added to chronic ketamine surveillance labs, as current monitoring targets address only urologic and hepatobiliary toxicity.
- Clinicians should establish a correction ceiling and a desmopressin plan up front.

References

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With sincere gratitude to my Family Medicine faculty for their continued guidance, teaching, and support throughout my residency.