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Carbamazepine-Induced SIADH: A Case Report

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Abstract

Introduction: Carbamazepine is an anticonvulsant widely used for epilepsy, trigeminal neuralgia, and bipolar disorder. While effective, it has been implicated in the development of the syndrome of inappropriate antidiuretic hormone secretion (SIADH), leading to hyponatremia. **Case Presentation:** We report the case of a 66-year-old man with a history of hypertension, hyperlipidemia, schizophrenia, and epilepsy who presented with confusion, blurry vision, and bilateral hand paresthesia. Laboratory evaluation revealed hyponatremia (serum sodium 118 mEq/L), low serum osmolality, elevated urine osmolality, and elevated urine sodium, consistent with SIADH. Despite the therapeutic range of carbamazepine, symptoms persisted for several years due to delayed recognition of the drug-induced etiology. Gradual tapering and eventual discontinuation of carbamazepine resulted in normalization of serum sodium. **Discussion:** Carbamazepine-induced SIADH is a well-documented but often underrecognized complication of antiepileptic therapy. The mechanism involves direct stimulation of vasopressin V2 receptors, resulting in increased aquaporin-2 expression and water reabsorption. Conclusion: This case highlights the importance of recognizing medication-induced SIADH as a potential cause of refractory hyponatremia. Early identification and discontinuation of the offending agent are crucial to prevent morbidity and ensure optimal outcomes.

Keywords

Carbamazepine, SIADH, Hyponatremia, Antiepileptic Drugs, Drug-Induced Endocrine Disorder

1. Introduction

Carbamazepine is an antiepileptic drug commonly prescribed for epilepsy, trigeminal neuralgia, and acute manic or mixed episodes of bipolar I disorder (1).

Its mechanism of action involves inhibition of voltage-gated sodium channels, which reduces neuronal excitability and suppresses synaptic transmission [1].

Although generally well tolerated, carbamazepine has been associated with the syndrome of inappropriate antidiuretic hormone secretion (SIADH), a condition characterized by excessive renal water reabsorption and resultant hyponatremia [2]. The proposed mechanism involves stimulation of vasopressin V2 receptors and upregulation of aquaporin-2 channels in the renal collecting ducts, leading to dilutional hyponatremia despite normal or increased plasma volume [3].

The clinical manifestations of SIADH vary by severity, ranging from mild symptoms such as headache, nausea, and confusion to more serious complications, including vomiting, seizures, cerebral edema, and coma [4]. Severe complications are more likely when serum sodium levels fall below 125 mEq/L [4].

We present a case of carbamazepine-induced SIADH resulting in prolonged hyponatremia that persisted for several years due to delayed recognition of the drug as the underlying cause.

2. Case Presentation

A 66-year-old male with a medical history of hypertension, impaired fasting glucose, hyperlipidemia, schizophrenia, and epilepsy presented to the hospital on March 10, 2019, with complaints of blurry vision, bilateral hand paresthesia, and confusion. Although bilateral hand paresthesia may occur in the setting of electrolyte disturbances such as hyponatremia, it is also a recognized adverse effect of zonisamide therapy, which the patient was taking at the time. Zonisamide related paresthesia was considered as a potential confounding factor in the differential diagnosis. However, the temporal association with severe hyponatremia and subsequent improvement following correction of sodium suggested electrolyte imbalance as the primary contributor to the patient's symptoms. He denied alcohol or tobacco use and had no prior surgical history. Family history was notable for his father's death at age 62 from myocardial infarction and a mother with diabetes and hypertension.

Home medications included carbamazepine 1000 mg daily which the patient had been taking for approximately five years prior to presentation, amlodipine/benazepril 10/40 mg daily, levetiracetam 500 mg two tablets three times daily, quetiapine 100 mg two tablets nightly, and zonisamide 100 mg three capsules twice daily. He had no known drug allergies.

Initial laboratory evaluation demonstrated hyponatremia consistent with SIADH. Key laboratory findings are summarized in **Table 1**.

Despite evidence suggestive of drug-induced SIADH, the initial neurology evaluation attributed the findings to other causes. The patient was started on oral sodium chloride tablets (1 g, two tablets twice daily) for symptomatic management.

Over the next five years, persistent hyponatremia was noted, with serum sodium levels ranging from 118 to 127 mEq/L. On February 29, 2024, the patient remained hyponatremic despite ongoing sodium supplementation. During a fol-

low-up visit on May 30, 2024, his sodium level increased to 131 mEq/L after the neurologist agreed to begin tapering carbamazepine. By August 2024, carbamazepine was gradually replaced with oxcarbazepine. By December 2, 2024, sodium levels had normalized to 135 mEq/L. At his most recent follow-up in July 2025, the patient was no longer taking carbamazepine or sodium tablets, and his sodium remained stable at 135 mEq/L.

Table 1. Initial laboratory findings consistent with syndrome of inappropriate antidiuretic hormone secretion (SIADH).

Test	Result	Reference Range	Interpretation
Sodium (Na)	118 mEq/L	135 - 145 mEq/L	Hyponatremia
Chloride (Cl)	97 mEq/L	98 - 107 mEq/L	Normal
Potassium (K)	4.5 mEq/L	3.5 - 5.0 mEq/L	Normal
Magnesium (Mg)	2.1 mEq/L	1.6 - 2.6 mEq/L	Normal
Creatinine Clearance	>60 mL/min	>60 mL/min	Normal renal function
Serum Osmolality	257 mOsm/kg	275 - 295 mOsm/kg	Hypoosmolar
Urine Osmolality	317 mOsm/kg	100 - 300 mOsm/kg	Inappropriately concentrated
Urine Sodium	>40 mEq/L	<20 mEq/L (low volume)	Consistent with SIADH
Cortisol	17.09 µg/dL	6 - 23 µg/dL	Normal
TSH	2.03 µIU/mL	0.4 - 4.0 µIU/mL	Normal
Free T4	0.84 ng/dL	0.7 - 1.8 ng/dL	Normal
Vitamin B12	1003 pg/mL	200 - 900 pg/mL	Elevated
WBC	$8.9 \times 10^3/\mu L$	$4.5 - 11 \times 10^3/\mu L$	Normal
Carbamazepine Level	9.4 µg/mL	4 - 12 µg/mL	Within therapeutic range
Urine Drug Screen	Negative	—	No evidence of substance abuse
Imaging (CTA Head/Neck, Carotid US, CXR)	Normal	—	No structural or cardiopulmonary abnormalities

3. Discussion

SIADH is a well-documented but under-recognized complication of carbamazepine therapy. It represents a disorder of impaired water balance resulting in dilutional hyponatremia [2] [5]. Although more commonly associated with pulmo-

nary or CNS pathology, drug-induced SIADH remains an important differential in patients presenting with hyponatremia, especially when other causes have been excluded.

Other medications capable of contributing to hyponatremia were also considered. The patient remained on amlodipine/benazepril, levetiracetam, quetiapine, and zonisamide throughout the period during which serum sodium levels normalized following carbamazepine discontinuation. The persistence of these medications despite improvement in sodium levels further supports carbamazepine as the most likely causative agent of the patient's SIADH.

Carbamazepine-induced SIADH occurs via stimulation of vasopressin V2 receptors in the renal collecting ducts, leading to increased aquaporin-2 expression and water reabsorption [3] [6]. This results in dilutional hyponatremia despite an euvolemic state. Unlike many drugs that cause SIADH by increasing ADH secretion centrally, carbamazepine is unique in that it may enhance the action of ADH at the renal level, even in the absence of elevated serum ADH levels.

Although oxcarbazepine is structurally related to carbamazepine and has also been associated with hyponatremia and SIADH, the decision to transition therapy was based on the patient's seizure management needs and the treating neurologist's clinical judgment. In some patients, the risk of hyponatremia may differ between these agents depending on individual susceptibility, dosing, and metabolic response. In this case, tapering of carbamazepine and substitution with oxcarbazepine was associated with normalization of serum sodium levels, suggesting carbamazepine was the primary contributor to the patient's SIADH.

Symptoms typically arise when sodium levels fall below 125 mEq/L. In this case, the patient's symptoms of confusion, fatigue, and nausea were consistent with moderate hyponatremia and resolved with conservative management. Early recognition is essential to prevent progression to life-threatening complications such as seizures, coma, or cerebral edema.

Management includes discontinuation of the offending agent, fluid restriction, and, in severe cases, hypertonic saline [4] [7]. Slow correction of sodium is critical to avoid osmotic demyelination syndrome (ODS), a potentially irreversible neurologic condition characterized by dysarthria, dysphagia, behavioral changes, quadriparesis, and, in severe cases, "locked-in" syndrome [8]. The risk increases when serum sodium is corrected by more than 8 - 10 mEq/L in 24 hours. Vigilant monitoring of sodium correction rates and neurological status is essential during management [8].

The decision to initiate alternative antiepileptic therapy should involve careful consideration of the patient's neurologic history and risk of recurrent SIADH.

4. Conclusion

This case highlights the clinical significance of carbamazepine-induced SIADH as an often underrecognized yet consequential adverse effect of antiepileptic therapy. The patient's prolonged course of hyponatremia, persisting for years despite so-

dium supplementation, shows the potential for diagnostic delay when medication induced etiologies are not considered. Discontinuation of carbamazepine ultimately normalized serum sodium levels, reinforcing the causal association and the necessity of ongoing vigilance in patients receiving this agent. Clinicians should maintain a suspicion for drug-induced SIADH in individuals presenting with unexplained or refractory hyponatremia, particularly in the absence of other systemic causes. Regular electrolyte monitoring, interdisciplinary collaboration, and timely modification of pharmacotherapy are essential to prevent morbidity and optimize neurological and metabolic outcomes. This case further emphasizes the importance of individualized therapeutic decision making and reinforces the need for heightened awareness of carbamazepine's potential endocrine effects in long-term management.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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