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RESEARCH ARTICLE

CARBAMEZAPINE INDUCED SEVERE ASYMPTOMATIC HYPONATREMIA: CASE REPORT

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ABSTRACT

Background: Severe hyponatremia is rare when carbamezapine is used as a monotherapy. It acts by inappropriate anti-diuretic secretion. Here we present a case of female with severe asymptomatic hyponatremia. Case report: A 61 year female which was a known case of trigeminal neuralgia and hypertension presented to opd for her routine checkup. She was taking tab carbamezapine 300mg TDS, temisartan 40 mg and amlodipine 5 mg once a day. Her blood pressure was 126/82 mm of Hg, pulse rate was 72 bpm. Her systemic investigation was normal. Her baseline blood investigation was sent as a part of routine checkup and her sodium was reported to be 109 mEq/l. Her sodium was repeated again and was found to be 111 mEq/L. she was admitted in view of hyponatremia in the ward. Her serum osmolarity was 228 mosm/kg , urine sodium was 92 mEq/l, urine osmolarity was 306mOsm/kgH20, uric acid was 2.1 mg/dl. Tsh was normal. Our patient was diagnosed with SIADH . Her carbamezapine was stopped, salt intake was increased and fluid were restricted. 5 day after her sodium improved to 126 m Eq/l. she was discharged and on followup her sodium was 132m Eq/l after 1 week. Conclusion : hyponatremia is a life threatening condition and is multifactorial. acute hyponatremia is more symptomatic than chronic. Hence we should be very careful in managing these patients

INTRODUCTION

Hyponatremia is defined as a serum sodium (Na+) level of < 136 mmol/L, and is considered significant when levels are between 115 and 125 mmol/L (1). Acute hyponatremia (less than 48 hours) can cause neurologic complications such as seizures and coma and it necessitates urgency of care to prevent complications. Carbamazepine, an anticonvulsant and psychotropic drug, is used commonly for the treatment of epilepsy, neuralgia, mental retardation and psychiatric disorders[2]. The incidence of hyponatremia (Na<134 mEq/L) in 451 carbamazepine-treated patients was 13.5% and that of severe hyponatremia ((Na<128 mEq/L)) was 2.8%[3.] The main mechanism by which carbamazepine causes hyponatremia is by increased antidiuretic hormone (ADH) secretion, but it has also been related to increased sensitivity of the renal tubules to ADH activity as well as increased aquaporin 2 channel expression in the renal tubule[4] In the following case, we present the case of a patient with asymptomatic hyponatremia secondary to recent carbamazepine use.

CASE PRESENTATION: 61 year female known case of trigeminal neuralgia and hypertension presented to opd for her routine checkup. She was taking tab carbamezapine 300mg TDS, temisartan 40 mg and amlodipine 5 mg once a day. Her blood pressure was 126/82 mm of Hg, pulse rate was 72 bpm. Her cardiovascular, neurology, respiratory examination was normal. Her baseline investigation were sent as a part of routine investiagation as shown in table 1. her sodium. Her baseline blood investigation was sent as a part of routine checkup and her sodium was reported to be 109 mEq/l. Her sodium was repeated again and was found to be 111 mEq/L. she was admitted in view of hyponatremia in the ward. Her serum osmolarity was calculated which was 228 mosm/kg , urine sodium was sent which was 92 mEq/l, urine osmolarity was 306mOsm/kgH20. hence a possibility of hypoosmolar euvolumeic asymptomatic hyponatremia was kept. For futher investigation her uric acid was 2.1 mg/dl. Tsh was within normal limit. Since her uric acid was low with asymptomatic euvolumeic hyposomolar hyponatremia a possibility of SIADH was kept secondary to carbamezapine. Her xray chest and ct head was done which was normal. Her carbamezapine was stopped, salt intake was increased and fluid were restricted. 5 day after her sodium improved to 126 m Eq/l. she was discharged and on followup her sodium was 132m Eq/l after 1 week.

Table 1.

hemoglobin	12.30g/dl	urea	17.80 mg/dl
Tlc	7.20 thou/mm3	Creatinine	0.51 mg/dL
PLATELTS	164 thou/mm3	Calcium	9.06 mg/ dL
ESR	38	phosphorus	3.39mg/dL
SGOT	28 U/l	Sodium	109 mEq/L
SGPT	18U/l	potassium	5 mEq/L
Alk posphate	111U/l	chloride	73.34 mEq/L
protein	8.05 g/dL		

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DISCUSSION

Severe hyponatremia is rare when carbamazepine is used as monotherapy [5], however if it does develop, it is most common in the first 3 months [6], with a median onset of 38.5 days. Our patient had been on carbamazepine therapy for more than 30 days. Carbamazepine, an anticonvulsant and psychotropic drug, is used commonly for the treatment of epilepsy, neuralgia, mental retardation and psychiatric disorders[2]. In our patient it was used to treat trigeminal neuralgia. The incidence of hyponatremia (Na<134 mEq/L) in 451 carbamazepine-treated patients was 13.5% and that of severe hyponatremia ((Na<128 mEq/L)) was 2.8%[3]. The main mechanism by which carbamazepine causes hyponatremia is by increased antidiuretic hormone (ADH) secretion, but it has also been related to increased sensitivity of the renal tubules to ADH activity as well as increased aquaporin 2 channel expression in the renal tubule[4.] Hyponatremia is defined as a serum sodium (Na+) level of < 136 mmol/L, and is considered significant when levels are between 115 and 125 mmol/L. [1]

Hyponatremia is subclassified as mild (130-134 mEq/l), moderate (125 – 129) and severe (<125 mEq/L). The symptoms of hyponatremia are mainly neurological and relate to brain edema. Chronic hyponatremia can be severe yet remarkably asymptomatic because the brain has adapted by decreasing it tonicity over weeks as was in our case. Serum uric acid is low (< 4 mg/dL) in patients with SIAD consistent with suppressed proximal tubular transport in the setting of increased distal tubular sodium and water transport. In our study also the uric acid was 2.1 mg/dl consistent with SIADH. Since SIADH is due to multiple causes her xray was done to rule out pulmonary lesion which are an important cause of SIADH. Her ncct head was also done which was normal. Our patient showed good response to management with fluid restriction and discontinuation of carbamazepine therapy. Predictors of poor response to therapy are serum osmolality > 500 mmol per kg, 24 hour urine output > 1.5 L, increase of > 2 mmol of serum Na+ in 24 hours, and a serum Na+ level less than the sum of UNa+ and K+ levels.[7]

Conclusion

Hyponatremia can be misdiagnosed if not recognized promptly; suspicion should be high when risk factors are present and the patient has been prescribed antiepileptic drugs. Patients can suffer from neurologic complications if the imbalance is not corrected. Therapy should be limited to fluid restriction and discontinuation of the causative agent since the use of other medications is not recommended.

Conflict of Interest: None

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