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A Rare Case of Severe Hyponatremia Associated with Acyclovir Use and Exacerbated by Furosemide

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ABSTRACT

Hyponatremia, a common electrolyte disorder, is rarely associated with acyclovir therapy. We report a 64-year-old woman with a history of hypertension and type 2 diabetes mellitus who presented with altered mental status and severe hyponatremia (serum sodium: 112 mmol/L) after receiving acyclovir for herpes zoster and a single dose of intravenous furosemide. Initial management with hypertonic saline and fluid restriction failed to improve her condition. Acyclovir was subsequently discontinued, leading to a gradual resolution of hyponatremia and normalization of sodium levels. This case underscores the potential role of acyclovir in inducing hyponatremia, possibly through syndrome of inappropriate antidiuretic hormone secretion (SIADH), and highlights the importance of recognizing and managing this rare but serious adverse effect.

Keywords: Hyponatremia; Acyclovir; SIADH; Furosemide; Herpes Zoster; Electrolyte Imbalance

INTRODUCTION

Hyponatremia, defined as a serum sodium level below 135 mmol/L, is the most common electrolyte imbalance, affecting approximately 1% of the general population. Its prevalence increases significantly in medical settings [1].

Hyponatremia can cause a spectrum of symptoms, ranging from mild confusion, nausea, and vomiting to severe manifestations such as respiratory distress, sleep disturbances, seizures, and coma. Contributing factors include diuretics, adrenal insufficiency, and inappropriate antidiuretic hormone secretion (SIADH)[2].

Acyclovir has been rarely associated with hyponatremia, with only a few documented cases in the literature. Here, we report a case of symptomatic severe hyponatremia in an elderly female patient, where acyclovir was identified as the causative agent.

Case Presentation

A 64-year-old woman with a history of hypertension and type 2 diabetes mellitus presented to the medical emergency department with altered mental status and recent memory loss lasting four days. Her symptoms began after receiving a single dose of intravenous furosemide (40 mg) for hypertension, prescribed by a local doctor.

The patient's medications included metformin (850 mg), glimepiride (1 mg), and valsartan (50 mg), all taken once daily. Additionally, she had been prescribed oral acyclovir (800 mg, five times daily) for the past four days for herpes zoster involving the left periorbital region.

On arrival, the patient was vitally stable but disoriented to time, place, and person. A detailed physical examination was unremarkable. Laboratory investigations, including thyroid function tests, complete blood count, renal and hepatic function tests, and midnight serum cortisol levels, were all within normal limits, except for severe hyponatremia with a serum sodium level of 112 mmol/L(Figure 1).

Initial Management

The patient was treated with an infusion of 100 mL of 3% saline in the emergency department, which temporarily improved her condition. However, four hours later, she reverted to her initial altered mental status. A single dose of intravenous dexamethasone (40 mg) was administered but had no effect. The patient was then admitted to the medical ward for further evaluation and management.

LITERATURE REVIEW AND DIAGNOSIS

Based on a review of the literature, we identified previous reports associating acyclovir use with syndrome of inappropriate antidiuretic hormone secretion (SIADH) and hyponatremia. Suspecting this as a potential cause, we implemented fluid restriction (800 mL/day) and oral sodium chloride supplementation three times daily. Despite these measures, the patient’s sodium levels remained persistently low.

Given the lack of response to these interventions, acyclovir was discontinued on the third day of admission. Subsequently, her serum sodium levels steadily improved, reaching 128 mmol/L by the 6th day (Figure 1). During this time, the patient became asymptomatic, and her herpes zoster lesions resolved. MRI of the brain, performed on the third day to rule out central causes of altered mental status, was unremarkable.

Outcome and Follow-Up

The patient was discharged on the sixth day of admission with a serum sodium level of 128 mmol/L and advised to follow up in two weeks. At follow-up, her sodium level had normalized to 138 mmol/L (Figure 1) and she remained asymptomatic.

This case highlights acyclovir-induced hyponatremia, with exacerbation by furosemide. Although rare, such cases warrant clinical attention due to the potential for severe electrolyte disturbances.

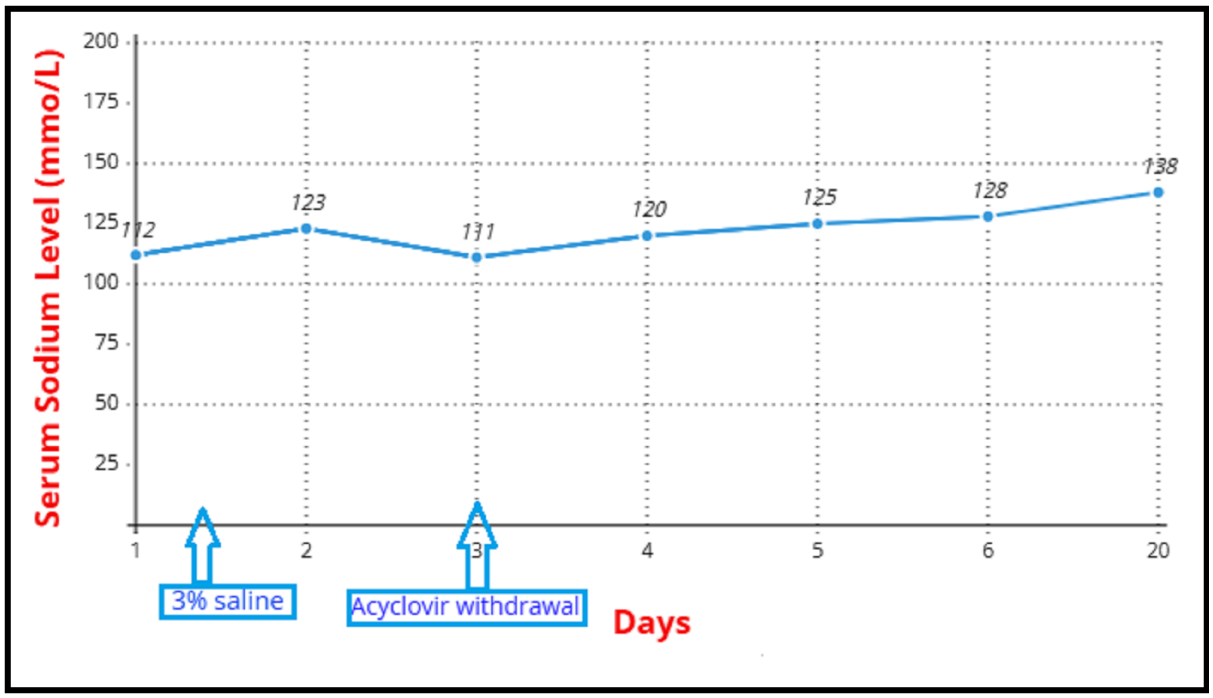

Figure 1: Trend of serum sodium level over span of hospital stay

DISCUSSION

Hyponatremia is infrequently associated with acyclovir, with only a few reported cases of patients developing hyponatremia while being treated with acyclovir for varicella-zoster infection[3-6]. These cases suggest that acyclovir may contribute to hyponatremia, although it has also been proposed that the infection itself could trigger syndrome of inappropriate antidiuretic hormone secretion (SIADH), leading to hyponatremia.

The exact mechanism of SIADH remains unclear, but it is often associated with conditions such as infections, malignancies, and central nervous system insults, including infarctions or hemorrhages[2]. Some studies further suggest that acyclovir may directly play a role in inducing SIADH[3].

Other potential causes of hyponatremia, such as hypothyroidism and Addison's disease[2], were excluded in our patient, as her laboratory results confirmed normal adrenal and thyroid function. It is possible that the patient was already hyponatremic due to acyclovir use, which was then exacerbated by the administration of a low dose of diuretics.

Given that the patient's herpes zoster lesions had resolved, we did not reintroduce acyclovir to confirm the recurrence of hyponatremia. This case highlights the need for further research to establish a definitive link between acyclovir and SIADH, as well as to clarify the underlying mechanisms of this association.

CONCLUSION

In conclusion, this case report highlights the rare but significant association between acyclovir use and hyponatremia, potentially exacerbated by concomitant furosemide administration. Although acyclovir-induced hyponatremia is infrequently reported, it remains a critical consideration in patients presenting with altered mental status and electrolyte disturbances, particularly in those receiving diuretics. The exact mechanism remains unclear, but the potential role of acyclovir in inducing syndrome of inappropriate antidiuretic hormone secretion (SIADH) warrants further investigation. This case underscores the importance of careful monitoring of electrolytes in patients receiving acyclovir, particularly those with additional risk factors for hyponatremia, and calls for further studies to better understand the pathophysiology and management strategies for this rare but concerning side effect.

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