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Case Report

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Unusual cause of hyponatremia in a young patient with varicella zoster infection: a case report

Abstract

Hyponatremia is one of the more common electrolyte abnormalities in clinical medicine. Some sources estimate that the number of patients who experience sodium levels of <135meq/L can be as high as 30% of all hospitalized patients. SIADH is the usual cause of Hyponatremia in patients with Varicella Zoster infection. Severe hyponatremia can cause substantial morbidity and mortality. The most common manifestations are CNS-related and include lethargy, confusion, disorientation and agitation. Serious manifestations include seizures, hypoxia and coma. We present here a report of an adult patient who presented with Varicella Zoster infection with seizures, agitation and unresponsiveness due to hyponatremia. Patient was on homeopathic medications and salt free diet since 3 days and was advised to take only free water to prevent exacerbations of skin lesions.

Keywords: varicella zoster, hyponatremia, homeopathy

Volume 7 Issue 5 - 2019

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Received: August 13, 2019 | **Published:** September 05, 2019

Introduction

Hyponatremia, defined as serum sodium below 135mEq/L, is one of the most frequently encountered electrolyte disorders in critically ill patients.[1] Patients at risk include septic patients, postoperative patients, older patients on thiazide diuretics, patients with malignant or psychiatric illness, and endurance athletes. A serum sodium concentration <100mEq/l, carries a high risk of brain damage due to seizure activity. There is concern about injury to the brain for rapid and over correction of hyponatremia. We present here a patient who presented with Varicella Zoster infection and symptomatic hyponatremia due to volitional intake of free water as per advice of homeopathy physician. Hypertonic saline, fluid restriction and close monitoring in the intensive care unit led to complete recovery of the patient with no residual neurological deficit.

Case report

A 42-yr old man, with no known comorbidities, presented with fever, convulsions and vesicular eruptions over thorax, extremities, head & neck due to Varicella Zoster infection of three days duration. He was treated at home by a family homeopathy physician who prescribed alternative medications and salt free diet and was also advised to take only free water to prevent exacerbation of skin lesions and irritability. In the emergency room, patient was convulsing, unresponsive, agitated and combative with Glasgow Coma Scale (GCS) of 7/15 and had oral bleed due to tongue bite. He was sedated, intubated and ventilated and remained stable hemodynamically. Initial blood investigations revealed serum sodium concentration of 113mEq/l with normal renal and liver functions. Other biochemical parameters were as shown in Table 1. Cortisol and thyroid levels were within normal limits. Septic work up was unremarkable.

Table 1 Biochemistry and urine laboratory test results

Test	Unit	On admission	6hrs after	12 hrs after	Day 2	Day 3	Day 4	Day 18
Serum Sodium	mmol/L	113	118	121	124	128	132	137
Serum Chloride	mmol/L	79	92	94	99	100	101	104
Serum K	mmol/L	4.3	3.6	3.8	3.9	4	4.4	4.6
Serum Osmolality	mOsmol/kg	236						
Urine Na	mmol/L	59	80					
Urine Osmolality	mOsmol/kg	608						

CT of the head showed no evidence of cerebral edema, meningeal enhancement, stroke or space occupying lesions. Cerebrospinal fluid studies were normal. He was managed as symptomatic euvolemic hyponatremia, due to SIADH aggravated by increased free water ingestion and salt restriction. EEG showed nonspecific generalized slowing with no epileptic discharges. The patient received 2 doses of 150 mls 3% hypertonic saline in first 4 hours until his sodium reached 120 mEq/l, followed by normal saline infusion according to his calculated fluid deficit subsequently. Rate of sodium correction was meticulously monitored at 1, 6 and 12 hours. He was simultaneously treated with antiviral (Acyclovir) and antibiotics along with supportive management. He was successfully weaned off ventilator within 48 hours once his sodium normalized with no residual neurological deficit and was discharged home after 4 days of hospitalization. Serum

sodium levels on the day of discharge were 132mEq/L with Glasgow Outcome scale of 1. Follow up two weeks later was unremarkable, with serum sodium levels of 137mEq/L and complete recovery from chicken pox with some residual scars.

Discussion

Hyponatremia can be due to various causes (Table 2). SIADH is a known entity in patients with viral infections including Varicella Zoster which could have been one of the factors in our patient as he was euvolemic with urine Osmolality more than that of serum.[2] However, hyponatremia in our patient was also exacerbated due to excess ingestion of salt free fluids and salt restriction leading to severe symptomatic hyponatremia, on the advice of homeopathic physician. The best way of symptomatic hyponatremia is to administer hypertonic saline solution. This was achieved promptly in our patient by giving hypertonic saline 150mls, total 2 doses in 6 hours, along with fluid restriction with regular monitoring of serum sodium at 1,6 and 12 hours without overcorrecting it which led to his complete neurological recovery. Guidelines for managing hyponatremia are based mostly on retrospective studies and expert opinion, and recommendation is to promptly increase serum sodium concentration (by at least 4-6mEq/L) without overcorrecting it (i.e. > 10mEq/l in 24 hours and/or 18mEq/L in 48 hours).[3,4] Risk of rapid correction of serum sodium (>10 mEq/l in 24 hours or 18 mEq in 48 hours) in acute Hyponatremia includes Osmotic Demyelination syndrome which has worse prognosis.[5]

Table 2 Causes and characteristics of hyponatraemia*

	Volume of extracellular fluid		
Variable	**Contracted**	**Normal or near normal**	**Expanded**
Causes	Diarrhea; vomiting; excessive sweating; poor water intake; diuretic use	Syndrome of inappropriate secretion of antidiuretic hormone; hypothyroidism; adrenal insufficiency	Congestive heart failure; cirrhotic liver disease; nephrotic syndrome
Serum osmolality	Low	Low	Low
Urine osmolality (mOsm/L)†	>500	>100	>100
Urine volume	Usually decreased	Varies with intake	Usually decreased
Urine sodium concentration (mmol/L)	<20‡	>40	<20‡
Response to 0.9% saline infusion	Clinical and biochemical improvement	No change or worsening of hyponatraemia	Little change in hyponataemia, worsening of edema

*For simplicity, conditions such as cerebral salt wasting and "reset osmostat" have been omitted.

†Values less than 100 imply an appropriate pituitary and renal response to hyponatremia, as would be expected with psychogenic polydipsia. The variability in urine osmolality results in low sensitivity and specificity for causes of hyponatremia in which the osmolality is greater than 100 mOsm/L.

‡May be greater than 20 with diuretic use.

The findings of currently available Cochrane reviews of studies of homeopathy do not show that homeopathic medicines have effects beyond placebo.[6,7] The treatment of hyponatremia is determined by 3 major factors: severity of hyponatremia, that is, the presence or absence of severe CNS symptoms such as lethargy, delirium, seizure, and coma; onset of hyponatremia: acute (within 48 hours) or chronic (>48 hours); and volume status.[8] The biggest challenge of treating symptomatic hyponatremia is how to prescribe saline therapy and maintain the correction rate in the target range. It cannot be overstated that frequent measurements of the serum sodium must be performed in concert with whichever calculations are used; as clinical estimates of TBW are rather crude. Nguyen and Kurtz reviewed potential errors with simplified formulae. If the rate of correction is too fast or too slow, we may need to alter the infusion rate and/or administer furosemide dose may be necessary.[9]

In our case we followed the protocol based on FWE along with frequent monitoring of serum sodium and other electrolytes. Overcorrection of hyponatremia could be managed by administration of desmopressin along with careful administration of fluids to keep up with unwanted urinary water losses. This is especially common in patients who have chronic hyponatremia, or whose initial sodium concentration is ≤105mEq/L, patients with hypokalemia, alcoholism, malnutrition and those with liver disease.[10] Recently, conivaptan, a V1A/V2-receptor antagonist, was approved for treating hospitalized patients with euvolemic Hyponatremia. However, the risk of rapid correction is still present; therefore, frequent checks of serum sodium are needed.[11] Patient education leaflets and sites never mention about possibility of hyponatremia in Chicken pox patients leave alone risk of hyponatremia with salt restricted fluids as was seen in our patient. This could be one area which we need to look at which could prevent complications in a seemingly benign Varicella Zoster diagnosed patient who otherwise have a god recovery.[12]

Funding details

None.

Acknowledgments

None.

Conflicts of interest

Authors declare that there is no conflict of interest.

Citation: Kantor S, Chandwani J, Abri SA, et al. Unusual cause of hyponatremia in a young patient with varicella zoster infection: a case report. *J Microbiol Exp.* 2019;7(5):224–226. DOI: 10.15406/jmen.2019.07.00263

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