

Severe Asymptomatic Hyponatremia in an Elderly Man

Lawson Ekpe¹, Emin Emin², Onuche Lawrence² and Osuji Kingsley³

¹Department of Chemical Pathology, University of Calabar, Calabar, Nigeria

²Department of Chemical Pathology, University of Calabar Teaching Hospital, Calabar, Nigeria

³Department of Chemical Pathology, Ambrose Alli University, Ekpoma, Nigeria

ABSTRACT

Hyponatremia is regarded as the most common electrolyte imbalance among hospitalised patients. Most hyponatremic conditions present with various degrees of symptoms depending on the level of sodium. Herein, we present a case of hyponatremia in a 65-year male, which was being managed as a case of diabetic nephropathy. On admission, initial results showed a sodium level of 120 mmol/L. Subsequent electrolyte assay after a week on medications showed serum sodium of 103 mmol/L with no associated symptoms of hyponatremia, except nausea and an episode of vomiting. There was no associated focal neurological deficit in the patient. Glasgow coma scale was 15. A repeat electrolyte assay done 24 hours later showed a serum sodium level of 102 mmol/L. The simultaneous level of potassium was 2.6 mmol/L and 2.7 mmol/L, respectively. From this case, it is important to note that the level of hyponatremia does not necessarily have a direct relationship with the severity of symptoms of hyponatremia.

Key Words: Hyponatremia, Electrolytes, Diabetic nephropathy.

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INTRODUCTION

Electrolyte imbalance is a common finding in clinical practice.¹ The normal serum sodium concentration falls between 135-145 mmol/L. Sodium is associated with many body functions such as fluid maintenance, fluid balance and regulation of blood pressure. Hyponatremia is defined as a serum sodium concentration of less than 135 mmol/L. It is a disorder of water balance reflected by an excess of total body water relative to electrolytes and is usually associated with low plasma osmolality (<275 mOsm/kg). Hyponatremia is generally caused by an increase in renal water reabsorption due to release of arginine vasopressin hormone along with water intake, and can occur in situations of volume depletion, volume overload, or normal volume. Sodium loss is minor compared with gains in water in most types of hyponatremia. Hyponatremia has been documented in literature as the commonest electrolyte abnormality globally among hospitalised patients.²⁻⁴ In most cases, hyponatremia is an incidental finding picked up by laboratory studies.^{4,5} It causes major diagnostic and management problems in practice. Hyponatraemic disorders are divided into euvolaemic, hypervolaemic and hypovolaemic.

In the evaluation of a hyponatraemic patient, history-taking should focus on identifying the potential cause, duration, and symptomatology. Clinical examination should include assessment of volume status. When there are obvious symptoms, these may help the attending clinician to investigate, diagnose, and treat hyponatremia conditions. Common signs and symptoms of hyponatremia include seizures, muscle cramps, nausea and vomiting, confusion, restlessness, coma and irritability, among others.

In this case report, we present a case of severe hyponatremia with no symptoms, which is very rare for this low serum sodium level. Low values of serum sodium with little or no clinical features have been documented in literature, especially in Caucasians. In our environment, this case report might be suggestive of the fact that severe asymptomatic hyponatremia may not be so rare as purportedly reported by some authors and researchers.

CASE REPORT

A 65-year male was referred to our Teaching Hospital from a private health facility. His presenting complaints were hiccups, poor appetite, nausea and leg swelling of seven days' duration. Full history was taken and he was found to be a known diabetic patient for more than 25 years, a known hypertensive for more than 10 years, and recently had become hepatitis B seropositive. Drug history showed that he was on anti-hypertensive drugs, like normoretic, losartan, and clopidogrel. On general physical examination, he was well oriented in time, place and person, afebrile, pale, mildly dehydrated with pedal edema up to the upper third of the lower limb. His pulse rate was 72 bpm,

Correspondence to: Dr. Lawson Ekpe, Department of Chemical Pathology, University of Calabar, Calabar, Nigeria

E-mail: lawsonekpe2002@yahoo.com

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with a blood pressure of 142/80 mmHg (sitting). His body mass index (BMI) was 39 kg/m². On chest examination, he had a respiratory rate of 26 breaths per minute and bilateral fine crepitations on auscultation. On central nervous system examination, he was conscious and alert, with no focal neurological deficit. Other systemic examination was not remarkable. He was admitted and managed as a case of diabetic nephropathy with super-imposed acute interstitial nephritis and urinary tract infection (UTI). Investigations done included full blood count, serum electrolytes, urea and creatinine, glycated hemoglobin, chest X-ray, urine culture, urinalysis, and echocardiography. Initial results showed hemoglobin of 8.6 g/dl and a serum sodium of 120 mmol/l. The sample for serum electrolytes was collected under an aseptic procedure and put into a plain bottle. The serum electrolyte assay was done by experts on Labjeniks® Q-Lyte 60 electrolyte analyser (Made in Germany), which was well-calibrated. Quality control (QC) check was done prior to assay of the sample. Various levels of QC were passed and had other agreeable electrolyte results. The laboratory is currently participating in an external quality assessment by the Fellow of the Royal College of Pathologists (Chemical Pathology) of Australasia. The patient had elevated glycated hemoglobin, mild cardiomegaly, potassium of 2.6 mmol/l, and the urine culture showed a pathogenic organism. The patient was subsequently placed on appropriate antibiotics, continued on antidiabetic agents (metformin and pioglitazone), antihypertensives (candesartan, torsemide, metolazone), antiviral agent (lamivudine) and low molecular weight heparin (clexane). Following further complaints of gastritis and nausea with an episode of vomiting, rabeprazole and metoclopramide were added to his drugs. The patient was then managed and followed up with other investigations as liver function tests (including albumin and total protein), lipid profile, thyroid function tests, which were all unremarkable.

One week after admission and intake of his above-listed medications, a repeat kidney function test result showed sodium 103 mmol/L, urea 8.8 mmol/L, chloride 71.6 mmol/L, bicarbonate 25 mmol/L, potassium 2.6 mmol/L, and creatinine 116 µmol/L. Following these results, a repeat test was done 24 hours later and the results obtained were: sodium 102 mmol/L, urea 12.2 mmol/L, chloride 96 mmol/l, bicarbonate 24 mmol/L, potassium 2.7 mmol/l and creatinine 129 µmol/L. Continuous check and evaluation showed that hypokalemia eventually got corrected as the value increased to 3.2 mmol/L over a month.

Urine electrolytes and osmolality were not done to ascertain the cause and type of hyponatremia. A thorough check, history, and medications were assessed to elicit the cause of the hyponatremia. Based on those results, signs and symptoms of hyponatremia were checked, and none were found. It followed that the serum sodium level of 102 and 103 mmol/L did not have any clinical manifestation in the patient, except nausea and one episode of vomiting. This is really strange and it prompted us to check the literature on this subject. The managing team decided to augment his sodium level gradually to restore it to

normal. This was corrected gently over days to weeks till it was restored to the normal value of 136 mmol/L.

DISCUSSION

The prevalence of hyponatremia has been recorded in various literature to be as high as 18%.³ Hyponatremia can be mild, moderate or severe.⁶ Severe hyponatremia is serum sodium less than 125 mmol/L and is usually associated with increased morbidity and mortality. It is known that patients who develop hyponatremia during hospitalisation have increased mortality rates compared with those who have hyponatremia on admission.⁵ Lower levels of hyponatremia are associated with more severe symptoms, especially neurological symptoms.^{7,8} Generally, notable signs and symptoms of severe hyponatremia may include headache, obtundation, muscle cramps, falls, seizures, coma and even death.^{7,8} In the literature, low values of plasma sodium have been reported with mild symptoms. Joseph *et al* reported serum sodium of 99 mmol/L with minimal symptoms in a case presentation. This serum sodium seems to be one of the lowest values of hyponatremia in a patient without symptoms documented in the published literature.⁴ Our patient had serum sodium of 103 mmol/l and there were no marked clinical signs or symptoms except for a nauseating effect and one episode of emesis. Halawa *et al* proposed that of the 363 patients seen in a Swedish hospital over a 3-year period, hyponatremia values of < 125 mmol/L occurred in only 11 of them with an only neurological manifestation of hyponatremia.⁹ A low level of sodium up to the range of 110 mmol/L is thought to be extremely dangerous with high mortality and morbidity reaching 33-80%. Sterns *et al* in their study seem not to corroborate with the general popular widespread belief of high mortality linked to severe hyponatremia.¹⁰ In their study, involving 62 patients, with 64 episodes of severe hyponatremia, only 8% had mortality, mostly related to underlying diseases. In those with a sodium level of 105 mmol/L or below, the mortality rate was 5%. They suggested that rapid correction of hyponatremia is not justified just because of low serum sodium levels.¹⁰ Very low sodium is not without signs or symptoms in many people. Renneboog *et al* observed that mild chronic hyponatremia induces a high incidence of falls, tiredness and attention-deficit as a result of gait and attention impairment. A major complication could be cerebral edema, arising from the fluid shift in the brain tissue which can eventually lead to brainstem herniation, respiratory arrest and death.¹¹

In conclusion, this case demonstrates that in some cases of severe hyponatremia, clinical symptoms may not be very obvious as seen in our patient. Therefore, a serial electrolyte assay and high index of suspicion should be the way forward in managing such patients.

PATIENT'S CONSENT:

Written and informed consent was obtained from the patient and the head of the department to publish the data. Confidentiality of the patient was maintained. All processes involved were done within ethical acts.

CONFLICT OF INTEREST:

The authors declared no conflict of interest.

AUTHORS' CONTRIBUTION:

LE: Supervised, literature review.

EE: Analysed sample, literature review.

OL: Analysed sample, data collection.

OK: Data collection.

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