CASE REPORT

Cerebral Salt Wasting Syndrome in a Patient with Normal Pressure Hydrocephalus with Possible Change in Pressure Setting of Adjustable Shunt by Metal Detector

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Abstract

Aim and background: Hyponatremia is a common electrolyte disorder in the setting of neurological disease. Among patients with neurological disorders, syndrome of inappropriate antidiuretic hormone secretion (SIADH) is the common malady, and cerebral salt wasting syndrome (CSWS) is rarely encountered. In SIADH, there is usually increased or normal intravascular volume, and in CSWS, there is central volume depletion. Thus, proper clinical examination of central volume status should be done before making a diagnosis.

Case description: A 73-year-old man presented with drowsiness 2 years ago. Serum sodium was low, and he was advised by a physician to take extra salt supplementation. His confusion worsened, he developed gait instability, urinary incontinence, and was diagnosed as having normal pressure hydrocephalus (NPH). A ventriculo peritoneal (VP) shunt was placed. Later in the year, he was again admitted to the hospital with drowsiness. He was diagnosed with SIADH and treated with water restriction and tolvaptan. A year later, he became drowsy and was admitted to this hospital. Serum sodium was low, and he had central volume depletion. He was diagnosed with CSWS. The worsening was due to the change in VP shunt pressure as he walked through a metal detector, which led to excessive drainage of cerebrospinal fluid (CSF). He was treated with hydration, salt supplementation, and shunt pressure adjustment, leading to improvement in his condition.

Conclusion: CSWS is a rare metabolic manifestation of NPH. Patients with VP shunts should be cautioned against walking through a metal detector with a potential electromagnetic field.

Clinical significance: Clinical assessment of jugular venous pressure (JVP) is important in order to differentiate between SIADH and CSWS.

Keywords: Case report, Cerebral salt wasting syndrome, Hyponatremia, Metal detectors, Normal pressure hydrocephalus.

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Introduction

Hyponatremia is a commonly encountered dys electrolytemia in the setting of a central nervous system disease. The usual cause is syndrome of inappropriate antidiuretic hormone secretion (SIADH).[1] The patient usually has an increased or normal intravascular volume due to inappropriate hypersecretion of ADH. A much rarer cause of hyponatremia is cerebral salt wasting syndrome (CSWS).[2] In the series by Sherlock et al., 7% had CSW compared to 69% who had SIADH.[1] In CSW, there is central volume depletion due to inappropriate salt wasting in the urine.

We describe a case where the patient presented with drowsiness, was diagnosed with normal pressure hydrocephalus (NPH), and had persistent hyponatremia, which was attributed to CSWS. The patient was admitted after acute deterioration following walking through a metal detector in a shopping mall.

Case Description

A 73-year-old man presented with drowsiness and mild confusion in December 2023. He had similar episodes of drowsiness and disorientation in January 2021, for which he had consulted a local physician. Various investigations were done. His serum sodium was found to be low, and hence he was advised to take extra salt with his diet. However, his confusion worsened, and in March 2021, he developed gait instability and urinary incontinence and was diagnosed with NPH, following which a ventriculo peritoneal (VP) shunt was placed. Again, in November 2021, the patient became very drowsy and was admitted to a hospital. At that point, his serum sodium was 112 mEq/L, potassium 5.7 mEq/L, uric acid 1.5 mg/dL, urine spot sodium 143 mEq/L, and urine osmolality 535 mOsm/kg. He was diagnosed with SIADH and was discharged with the advice of water restriction (500–1 L/day). Tolvaptan was commenced at 15 mg/day. After discharge from the hospital, his drowsiness persisted, and his serum sodium was always on the lower side.

In Dec 2022, the patient became extremely drowsy and unresponsive and was admitted to this hospital. His vital parameters

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were normal, and there was no focal neuro-deficit. He had central volume deficit. His blood pressure was 110/80 mm Hg with postural hypotension of 10 mm Hg. Investigations revealed serum sodium 121 mEq/L, potassium 5 mEq/L, uric acid 3 mg/dL, calculated serum osmolality 275 mOsm/kg, urine spot sodium 182 mEq/L, and calculated urine osmolality 380 mOsm/kg. On further evaluation, an MRI brain was done, which did not reveal any new findings besides NPH. An X-ray of the VP shunt was done as per the advice of the neurosurgeon, and his stent pressure setting was found to have changed from 70 to 90 mmH_2O, which had caused less drainage of cerebrospinal fluid (CSF). The patient gave a history of having walked through a metal detector in a shopping mall, following which his neural condition deteriorated. He was hydrated with normal saline (2–3 L/day) and given oral salt supplementation, and his clinical condition improved. He was discharged.

On admission in December 2023, the patient presented again with drowsiness and mild confusion. Vital parameters were within normal limits, respiratory rate was 20–24/minute, SpO_2 98% on room air, and BP 120/80 mm Hg. All relevant investigations were done, and his serum sodium was 118 mEq/L, potassium 4.4 mEq/L, urine spot sodium 212 mEq/L, TSH 2.9 mIU/L, cortisol 17.3 mcg/dL, and calculated serum osmolality 280 mOsm/kg. The NCCT brain was done, which was normal. X-ray of the VP shunt revealed that his VP shunt pressure had changed again from 70 to 90 mmH_2O, which caused less drainage of CSF, increased intracranial pressure, and resulted in worsening of his condition. The pressure valve setting was reset to 70 mmH_2O. The patient had central volume depletion due to cerebral salt wasting and was treated with normal saline (4 L/day) and oral salt supplementation (3 gm thrice daily). The serum sodium increased to 130 mEq/L, and the patient's sensorium gradually improved. He was ambulated and discharged.

On the follow-up clinic visits, the patient could walk to the clinic and talk normally. His sensorium was better, and urinary symptoms had improved. Serum sodium stabilized at 130 mEq/L. He was advised not to go near a magnet or walk through metal detectors.

A diagnosis was made of CSWS, which worsened each time the pressure setting of the VP shunt valve was altered due to magnetic interference, resulting in increased intracranial pressure.

Discussion

In case of hyponatremia induced by some cerebral affliction, it is important to distinguish between SIADH and CSWS. Both present with similar clinical manifestations; however, their pathophysiology and treatments are completely different.[3] Inappropriate treatment can aggravate the symptoms and result in mental changes, seizures, and even death.

In patients with NPH, SIADH has been reported to occur.[4] This is because of increased mechanical pressure on the hypothalamus due to the buildup of CSF, which causes the release of ADH. ADH acts on the V2 receptors in the collecting duct of the kidney and increases reabsorption of water, leading to high extracellular fluid volume. In this case, the patient was initially misdiagnosed as having SIADH and was treated with water restriction, although his central volume status was depleted, which made his condition worse.

The cause of hyponatremia in this case was due to CSWS. In CSWS, due to brain injury, there is release of circulating factors like brain natriuretic peptide (BNP) that impair renal tubular sodium reabsorption.[4] There is impaired sympathetic response, which inhibits the release of renin and aldosterone, resulting in a reduction in sodium, water, and urate reabsorption. Hence, patients with CSWS have low extracellular fluid volume but high BNP.

Cerebral salt wasting syndrome in NPH has not been reported, to our knowledge. A case was reported where CSWS was caused by external lumbar drainage in a patient with NPH.[5]

Walking through a metal detector has been reported to affect valve pressure settings in patients with an adjustable VP shunt.[6] Though the risk is low, as security scanners definitely emit weaker magnetic fields than MRI machines, the risk is still present. Some modern adjustable VP shunts, like the Codman (Integra Life Sciences), have protective features to minimize magnetic interference, which older models, like St. Jude Medical (Abbott), may not have.

A patient with a VP shunt should inform security personnel in shopping malls and airports about the risk and preferably carry a medical certificate stating this. It is of vital importance that they have a manual pat-down or some alternative screening procedure.

Conclusion

In conclusion, apart from SIADH, CSWS, which is an extremely rare metabolic manifestation of NPH, should be considered in the differential diagnosis of hyponatremia in patients with idiopathic NPH. Clinical assessment of jugular venous pressure (JVP) is very important in order to differentiate between SIADH and CSWS. Thus, proper clinical examination of central volume status should be done before making a diagnosis.

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