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Research Article

A Rare Encounter of Syndrome of Inappropriate Antidiuretic Hormone Secretion in Localised Herpes Zoster (Shingles)

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I N F O

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A B S T R A C T

Background: There have been many case reports previously stating Syndrome of Inappropriate Antidiuretic Hormone secretion (SIADH) as a manifestation of disseminated herpes zoster virus infection.

Objective: The case report adds light to localised herpes zoster as a rare cause of SIADH, an often-under-diagnosed entity.

Methodology: We report a 70-year-old female who was found in a state of altered sensorium after a presentation of itching and skin lesions over the left thoracic T (T7) region.

Results: Laboratory investigations revealed euvolemic hyponatremia and the patient was diagnosed to have SIADH. Treatment was given for the aetiology of SIADH and to treat the euvolemic hyponatremia.

Conclusion: Treatment of SIADH caused by localised herpes zoster shows quick recovery with the resolution of the herpetic lesions.

Keywords: SIADH, Hyponatremia, Herpes Zoster

Introduction

The clinical manifestations of hyponatraemia vary and depend upon the rapidity of development. These patients are either asymptomatic or present with headache, altered sensorium, seizures, or loss of consciousness. Hyponatremia is defined as a fall in serum sodium less than 135 mEq/L. Euvolemic hyponatremia is characterised by an increase in body water with little or no change in body sodium. One of the causes of euvolemic hyponatremia is SIADH.[1]

Syndrome of inappropriate antidiuretic hormone release (SIADH) is characterised by the inappropriate release of Anti-Diuretic Hormone (ADH) from the pituitary gland or from locations other than the pituitary gland or because of its continuous action on vasopressin receptors in the kidneys (nephrogenic SIADH). Herpes zoster or shingles in adults is caused by the reactivation of latent varicella-zoster virus. After an initial infection that manifests as chickenpox, the virus becomes dormant in dorsal root ganglia with reactivation at a later date. SIADH accounts for nearly 60% of hyponatraemia as per some studies.[2]

Other causes of euvolemic hyponatremia should be excluded like hypothyroidism, glucocorticoid deficiency, stress, and intake of drugs that enhance the release of ADH like carbamazepine, SSRIs, chlorpropamide etc.[3]

There have been many pieces of literature stating that SIADH has happened due to disseminated herpes zoster infection.[4] SIADH secondary to localised herpes zoster is either under-recognised or underreported. In this case report, we report an elderly female who had SIADH secondary to localised herpes zoster infection affecting the T7 dermatome.

Case Report

A 70-year-old female was brought to the emergency department in a state of drowsiness for one-day duration. There was no history of headache, trauma, or seizures. She had noticed painful and itchy skin rashes with blisters in a single, wide stripe or belt-like pattern limited to one side of the body over the T7 dermatome on the left side for 3 days. There was no history of fever, breathlessness, numbness, tingling, weakness of limbs, abdominal pain, altered bowel, or bladder habits. She was a known case of diabetes mellitus on glipizide twice a day and a known case of chronic kidney disease for 10 years not on any treatment.

On examination, there were no signs of dehydration, and Juglar Venous Pressure (JVP) and vitals were normal. On performing a central nervous system examination, the patient was drowsy, but responded to calls and had a Glasgow Coma Scale score of 14/15 (Eye opening 3, Verbal response 5, Motor response 6, E3V5 M6) with pupils equally reacting to light, and bilateral plantar reflexes were flexor. There was no cranial nerve involvement and remainder of the nervous system examination was normal. Crops of vesicles were noted over T7 dermatome on the left side. Breast, gynaecological, and skeletal examinations were found to be normal. Her previously seen creatinine was 2.0 mg/dl. Three months back, her serum electrolytes were found to be normal. All other baseline investigations like Complete Blood Count (CBC), Liver Function Test (LFT), Erythrocyte Sedimentation Rate (ESR), and C-Reactive Protein (CRP) were found to be within normal limits. Her Glycated Haemoglobin (HbA1C) was 7%, and Random Blood Sugar (RBS) was 200 mg/dl. Thyroid Stimulating Hormone (TSH) and 7 am serum cortisol were normal. ABG did not reveal any acid-base disturbances. Imaging of the brain was normal. Other investigations are described in Table 1. Ultrasound abdomen revealed atrophic ovaries and uterus, and bilaterally increased renal cortical echoes and normal-sized kidneys.

On further evaluation, the patient was found to have euvolemic hyponatremia, reduced serum osmolarity with inappropriately concentrated urine. Urinary excretion of electrolytes was normal with normal thyroid and adrenal cortical function. As this elderly patient had presented with altered sensorium due to hyponatremia, which was proceeded by herpes zoster infection over the lower chest wall, an association between the two was suspected. An extensive literature search led to the diagnosis of SIADH secondary to herpes zoster and treatment was started.

The patient was treated with free water restriction, normal saline, 3% NaCl, insulin, oral and topical acyclovir along with soothing agents. Acetaminophen was added for pain. During the course of stay in the hospital, sensorium of the patient improved. Serum sodium gradually returned to normal levels. She did not develop post-herpetic neuralgia. After 3 months of follow-up, the skin lesions had resolved, and serum creatinine was found to be 2.34. Serum electrolytes were within normal limits. She is on regular follow-up for diabetes mellitus and chronic kidney disease without recurrence of hyponatremia.

Table 1.Laboratory Investigations for Hyponatremia

Parameters	Reference Value	Day 1	Day 3	Day 7
BUN (mg/dl)	7-18	15	12	14
Creatinine (mg/dl)	0.8-1.21	2	2.1	1.98
Sodium (mEq/L)	135-145	112	121	134
Potassium (mEq/L)	3.5-5	3.9	4.1	4.1
Chloride (mEq/L)	96-108	100	101	109
Uric acid (mg/dl) in females	2.7-7.3	1.8		
Bicarbonate (mEq/L)	21-32	22	24	25
Serum osmolality (mosm/kg)	285-295	251	279	290
Urine osmolality (mosm/kg)	50-1200	600		
Urine spot sodium (mEq/L)	20-110	50		
Urine spot potassium (mEq/L)	12-62	15		
Urine spot chloride (mEq/L)	55-125	55.8		

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Discussion

Hyponatremia is one of the commonest electrolyte abnormalities noted in hospitalised patients, especially in Incentive Care Unit (ICU) settings. There are numerous causes listed for hyponatremia and one among them is SIADH which accounts for 60% of cases of hyponatremia. The causes of SIADH include infections, intracranial events, malignancies, and drugs.[1]

Varicella zoster virus causing herpes encephalitis and disseminated herpes zoster are known to cause SIADH but localised herpes causing SIADH is rarely encountered in clinical practice.[5] In this patient, clinical features and laboratory values are suggestive of the diagnosis of SIADH. The association between SIADH and localised herpes zoster is not frequent and to date, only a handful of cases have been reported. Most of the reported cases were in the elderly with an underlying co-morbid illness.[6,7]

The underlying mechanisms of SIADH associated with localised herpes zoster include reactivation of varicella zoster virus infection involving the regulatory pathway of anti-diuretic hormone secretion secondary to the centripetal spread via the dorsal root ganglia of the nerves and Trigeminal nerve division 1, ophthalmic nerve (V1) to the nucleus tractus solitarius. There is also non-osmotic stimulation of ADH by painful herpetic eruptions. According to previous case reports, the commonly involved dermatome for Varicella Zoster Virus (VZV) reactivation is that of the trigeminal nerve but this patient had unilateral thoracic 7 dermatomal involvement that led to SIADH.[7]

Most of the patients had hyponatremia until the resolution of Postherpetic Neuralgia (PHN).[8] Our patient did not develop PHN and the hyponatremia resolved with conservative management. Hyponatremia did not recur on long term follow up.

The other uncommon complications associated with herpes zoster are segmental zoster paresis (motor nerves) of upper and lower extremities, acute or chronic encephalitis, ophthalmic zoster with contralateral hemiparesis, myelitis, polyradiculitis, motor neuropathies, zoster sine herpete, and acute retinal necrosis.[9]

SIADH treatment is based on the severity of clinical signs and symptoms of hyponatremia. Earlier, demeclocycline, lithium, urea, and fluid restriction were used for treatment. Hypertonic saline is always given in grave hyponatremia. Quick correction of hyponatremia and shorter hospitalisation achieved by a newer class of drugs named 'Vaptans' mainly tolvaptan and conivaptan, have now achieved an upper hand in the treatment of SIADH by eliminating the requirement for fluid restriction.[10] The patient here was treated for the particular condition causing SIADH along with free water restriction, saline, and optimal blood glucose control.

Conclusion

SIADH is an uncommon complication of localised herpes zoster infection. As the reactivation of herpes zoster is more common among the elderly with an underlying comorbid illness, the development of hyponatremia is associated with a poorer prognosis unless recognised early and treated vigorously.

But in contrast to the treatment of SIADH due to other causes, the one caused by herpes shows a quick recovery with the resolution of herpetic lesions.

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Conflicts of Interest: None

References

1. Sahay M, Sahay R. Hyponatremia: a practical approach. Indian J Endocrinol Metab. 2014 Nov;18(6):760-71. [PubMed] [Google Scholar]
2. Yasir M, Mechanic OJ. Syndrome of inappropriate antidiuretic hormone secretion. Treasure Island (FL): Stat Pearls Publishing; 2021 Jan. [Google Scholar]
3. Mentrasti G, Scortichini L, Berardi R. Syndrome of inappropriate antidiuretic hormone secretion (SIADH): optimal management. Ther Clin Risk Manag. 2020 Jul;16:663-72. [PubMed] [Google Scholar]
4. Semel JD, McNerney JJ Jr. SIADH during disseminated herpes varicella-zoster infections: relationship to vidarabine therapy. Am J Med Sci. 1986 Feb;291(2):115-8. [PubMed] [Google Scholar]
5. Gnann Jr JW. Varicella-zoster virus: atypical presentations and unusual complications. J Infect Dis. 2002 Oct;186 Suppl 1:S91-8. [PubMed] [Google Scholar]
6. Bassi V, Fattoruso O, Santinelli C. Localized herpes zoster infection: a rare cause of syndrome of inappropriate secretion of antidiuretic hormone. Oxf Med Case Reports. 2017 Nov;2017(11):omx065. [PubMed] [Google Scholar]
7. Wang CC, Shiang JC, Chen JT, Lin SH. Syndrome of inappropriate secretion of antidiuretic hormone associated with localized herpes zoster ophthalmicus. Gen Intern Med. 2010 Feb;26:216-20. [PubMed] [Google Scholar]
8. Azimi SZ, Firooz A. Postherpetic abdominal pseudohernia: an uncommon complication of herpes zoster. Dermatologic Ther. 2020 Jul;33(4):e13530. [PubMed] [Google Scholar]
9. Teo HK, Chawla M, Kaushik M. A rare complication of herpes zoster: segmental zoster paresis. Case Rep Med. 2016;2016:7827140. [PubMed] [Google Scholar]
10. Gross P. Clinical management of SIADH. Ther Adv Endocrinol Metab. 2012 Apr;3(2):61-73. [PubMed] [Google Scholar]

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