Journal of Infectious Diseases & Therapy
ISSN: 2332-0877

Goyal et al., J Infect Dis Ther 2020, 8:3

Case Report Open Access

Herpes Simplex Virus Type 1 Encephalitis with Syndrome of Inappropriate Antidiuretic Hormone Secretion: A Case Report

Rajiv Goyal*, Jason Lee, Robert Kleyman, Hamish Patel and Domenick Sorresso
HCA Healthcare/USF Morsani College of Medicine GME Programs, Regional Medical Center Bayonet Point, Tampa, USA

***Corresponding author:** Rajiv Goyal, HCA Healthcare/USF Morsani College of Medicine GME Programs, Regional Medical Center Bayonet Point, Tampa, USA, E-mail: rajivgoyal689@gmail.com

Received date: June 23, 2020; **Accepted date:** July 10, 2020; **Published date:** July 17, 2020

Abstract

Patient's that present with concerns for viral encephalitis with concomitant syndrome of inappropriate antidiuretic hormone secretion (SIADH) should raise suspicion for herpes simplex virus type 1(HSV-1) encephalitis. Early recognition and treatment with high dose acyclovir can improve neurological symptoms associated with HSV encephalitis and SIADH.

Keywords: Herpes Simplex Encephalitis; SIADH; Hyponatremia; Case Report; CNS Infection

Introduction

HSV-1 encephalitis is a highly devastating central nervous system (CNS) infection, which accounts for approximately 10%-20% of the 20,000 annual cases of viral encephalitis [1,2]. There are few case reports that have reported the occurrence of SIADH in patients diagnosed with HSV-1 encephalitis [3,4]. Here we present a case of HSV-1 encephalitis with concomitant SIADH.

Case Presentation

A 74-year-old Caucasian male with a past medical history of hypertension, hyperlipidemia, gastroesophageal reflux disease, and coronary artery disease presented to our emergency department (ED) with a chief complaint of generalized malaise with associated fevers, diaphoresis, and nausea, which began three days prior to arrival after suffering a bee sting. The patient's workup was unremarkable, and he was discharged home from the ED.

The next day, the patient returned to the ED with his wife, who reported worsening confusion and dysarthria. On evaluation, he was oriented to self and was unable to answer any questions about his history or symptomatology. Vital signs were unremarkable, except for a temperature of 100.3°F for which he received acetaminophen. On neurological examination, no focal deficits were appreciated. Computed tomography (CT) brain was unremarkable for any acute intracranial processes. Due to concerns for bacterial meningitis, a lumbar puncture was performed and patient was empirically started on ceftriaxone, vancomycin, and ampicillin. Initial comprehensive metabolic panel revealed a plasma sodium of 124 mEq/L, with a urine sodium of 115 mEq/L and urine osmolality of 344 mOsm/kg on urinalysis, consistent with SIADH.

Due to worsening mentation and temperature rising from 100.3°F to 102.6°F, despite acetaminophen administration; the patient was transferred to the intensive care unit for closer observation and management. He developed respiratory distress and was subsequently intubated and sedated. An electroencephalogram (EEG) was performed, and we observed findings significant for diffusely slow waveforms consistent with diffuse cerebral dysfunction.

	Quality or Value
Color	colorless
Appearance	clear
White blood cells (wbc)	77 wbc/uL
Polynuclearwbc	1.30%
Mononuclear cells	98.70%
Total red blood cells (rbc) counted	0 rbc/uL
Glucose	58 mg/dl
Lactate dehydrogenase	65 unit/L
Total protein	104.0 mg/dl (high)

Table 1: Cerebral spinal fluid (CSF) laboratory results.

Magnetic resonance imaging (MRI) of the brain was significant for asymmetric, right greater than left, temporal lobe flair abnormality involving the right insular cortex and right limbic system, concerning for HSV encephalitis (Figure 1). CSF laboratory results (Table 1) were consistent with a viral etiology based on elevated protein and lymphocytic predominance. CSF serology was positive for HSV-1 and patient was immediately started on high-dose intravenous (IV) acyclovir 800 mg every 8 hours. The patient's condition continued to decline, becoming hypotensive and requiring vasopressor support. Repeat MRI of the brain three days later demonstrated worsening edema and restricted diffusion involving the right temporal lobe, insula, and right limbic system with a new mass effect onto the right lateral ventricle. Neurosurgery evaluated the patient and deemed that he was not a candidate for any surgical intervention.

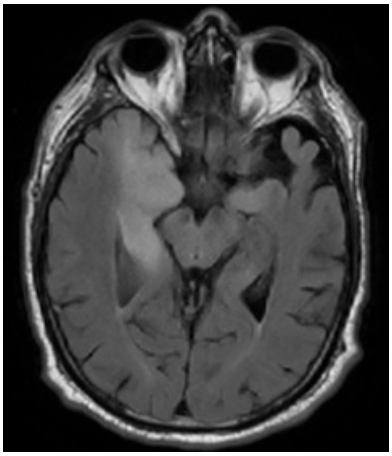

Figure 1: MRI of the brain with findings classic for HSV-1 encephalitis with bilateral temporal lobe flair abnormalities.

During his hospitalization, several attempts were made to wean mechanical ventilation however unsuccessful due to patient's overall declining neurological condition and need for ventilatory support. Given overall prognosis, decision was made to pursue comfort measures and proceed with terminal extubation.

Discussion

HSV-1 is by far the most common herpes simplex virus responsible for herpes encephalitis [5] about one-third of cases occur in individuals with a primary HSV-1 infection, while two-thirds of individuals are those who were already seropositive for the virus, irrespective of a history of recurrent orofacial herpes [6] most individuals affected by HSV-1 encephalitis display signs and symptoms of the infection within one week, including altered level of consciousness, change in personality, confusion, focal cranial nerve deficits, aphasia, ataxia, and focal seizures [7]. Several studies have demonstrated a possible link between HSV-1 encephalitis and SIADH [3,4] SIADH is a condition whereby a lack of suppression of antidiuretic hormone (ADH) release leads to impaired water excretion [8,9]. While ADH secretion relies on the hypothalamus and posterior pituitary gland, HSV-1 encephalitis classically involves the temporal lobe, insula, or cingulate.

Patients with HSV-1 usually present with fever, headache, dysphasia, disorientation, altered mental status and sometimes convulsions. Similar to the patient in our report, HSV-1 is common in older patients and encephalitis is usually due reactivation of the latent HSV-1 infection. Cranial MRI can be strongly indicative of encephalitis, showing hyper-intensity if the temporal lobe and hippocampal region. Furthermore, CSF findings for HSV-1 normally show a lymphocytic pleocytosis, elevated protein and normal glucose levels [4].

Our patient was found to have low sodium levels with high urine sodium and osmolality in the setting of SIADH. Hyponatremia can be due to a multitude of etiologies, from malignancies, drugs, human immunodeficiency virus (HIV) infection, and central nervous system (CNS) disturbances. In our case, the patient had a primary CNS infection with concomitant euvolemichyponatremia leading to SIADH. The exact mechanism is still unclear. One theory suggests that if the limbic system is involved, the inflammation can spread to nearby structures including the hypothalamic system causing disruption of hormonal control on the pituitary gland [4].

The presence of hyponatremia amongst patients with possible viral encephalitis could be helpful in the early diagnosis of HSV-1 encephalitis before CSF results are available.3 Clinically, a patient that presents with concerns for viral encephalitis with concomitant SIADH should raise suspicion for HSV encephalitis. Typically, treatment of the underlying disease process should lead to the resolution of hyponatremia. This was the case with our patient who presented with serum sodium of 124 mEq/L which improved to 137 mEq/L with IV acyclovir and normal saline 0.9%.

Conclusion

Patient's that present with concerns for viral encephalitis with concomitant SIADH should raise suspicion for HSV encephalitis. Early recognition and treatment with high dose acyclovir can improve neurological symptoms associated with HSV encephalitis and SIADH.

HCA Disclaimer

This research was supported (in whole or in part) by HCA and/or an HCA affiliated entity.The views expressed in this publication represent those of the author(s) do not necessarily represent the official views of HCA or any of its affiliated entities.

References

1. Levitz RE (1998) Herpes simplex encephalitis: a review. Heart Lung 27:209-212.
2. Whitley RJ (1990) Viral encephalitis. N Engl J Med 323:242-250.
3. Basaran S, Yavuz SS, Bali EA (2019) Hyponatremia is predictive of hsv-1 encephalitis among patients with viral encephalitis. Tohoku J Exp Med 247:189-195.
4. Kuriki A, Ishihara K, Satoh H, Sugie M, Kato H, et al. (2008) Syndrome of inappropriate secretion of anti-diuretic hormone associated with limbic encephalitis due to herpes simplex virus infection--a case report. Rinsho Shinkeigaku 48:184-190.
5. Corey L, Whitley RJ, Stone EF, Mohan K (1988) Difference between herpes simplex virus type 1 and type 2 neonatal encephalitis in neurological outcome. Lancet (London, England) 1:1-4.
6. Whitley RJ, Gnann JW (2002) Viral encephalitis: familiar infections and emerging pathogens. Lancet (London, England).359:507-513.
7. Whitley RJ (2006) Herpes simplex encephalitis: adolescents and adults. Antiviral Res 71:141-148.
8. Yasir M, Mechanic OJ (2020) Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH). In: Treasure Island (FL).
9. Ellison DH, Berl T (2007) Clinical practice. The syndrome of inappropriate antidiuresis. N Engl J Med 356:2064-2072.