Case Report

Down with the Flu: Hyponatremia in a patient with influenza

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Abstract

Context: Syndrome of inappropriate secretion of antidiuretic hormone (SIADH) is a common cause of hyponatremia. Although it has been associated with different pulmonary infections, there have been only few case reports describing the association of SIADH with influenza. **Case Report:** We report a case of SIADH in a patient with influenza who was successfully treated with fluid restriction. **Conclusion:** It is essential for clinicians to be aware of the association between influenza and SIADH.

Keywords: Inappropriate ADH syndrome, Influenza, Hyponatremia, SIADH

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Introduction

Syndrome of inappropriate secretion of antidiuretic hormone (SIADH) is one of the most common causes of hyponatremia in hospitalized patients, with a prevalence as high as 35%.[1] It is characterized by the impairment of urinary dilution in the absence of any renal disease or any identifiable nonosmotic stimulus that induces antidiuretic hormone (ADH) release. Although SIADH has been known to be associated with different pulmonary infections,[2] only rarely has it been reported with influenza. We present a case of hyponatremia associated with influenza, which was subsequently diagnosed as SIADH.

Case Presentation

A 65-year-oldmale presented to the emergency department with low-grade fever and productive cough for 2 days. He also had mild shortness of breath at rest with pleuritic chest pain. He did not have any neurological symptoms, abdominal pain, nausea, vomiting, change in bowel movements, or loss of appetite or weight. He did not drink any alcohol. He denied any sick contacts or recent travel. Past medical history was significant for coronary artery disease. He did not have any history of recent surgery, central nervous system disorders, malignancy, or known pulmonary disease. The only medication he was taking was aspirin. On examination, he had a temperature of 102.4F, heart rate 120/min, respiratory rate 24/min, blood pressure 110/60 mmHg without orthostatic vital signs, and oxygen saturation 96% on room air. Auscultation of the chest revealed decreased air entry bilaterally without any rhonchi or crepitations. Laboratory examination revealed a serum sodium of 122 mEq/L (135-145), blood urea nitrogen (BUN) 11 mg/dL (8-24 mg/dL), creatinine 1.35 mg/dL (baseline 1.2-1.3), plasma osmolality 276 mOsm/kg (280-290), urine specific gravity of 1.029(1.002-1.030), urine osmolality 777 mOsm/kg, urine sodium 63 mEq/L, thyroid-stimulating hormone (TSH) 3.011 IU (0.5-5.0 mU/L), and random serum cortisol 20.2 μg/dL (7-25 μg/dL). He had a white cell of 8.5×10^9/L. The chest X-ray did not show any infiltrate, consolidation, or mass.

The nasal swab polymerase chain reaction was positive for influenza A. Urinary antigens for Legionella, Mycoplasma, and Streptococcus were negative. Blood

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and sputum cultures were negative. The patient was immediately started on oseltamivir 75 mg twice daily for 5 days. A diagnosis of SIADH was made and his hyponatremia was corrected gradually in 3 days with fluid restriction o less than 800 ml/day. At 2 weeks follow-up, his serum sodium was 135 mEq/L.

Discussion

SIADH is a diagnosis of exclusion.[3] Thus, a careful history to rule out other causes like malignancy, pulmonary conditions, central nervous system lesions, and medications is essential in the evaluation of any patient suspected with SIADH. It has been reported in many pulmonary infections including asthma, atelectasis, acute respiratory failure, and pneumothorax.[4] Influenza has been implicated as a cause of SIADH previously,[5,6] but there is no published data regarding the actual incidence of SIADH in these patients. Our patient was diagnosed with SIADH based on:

a. Hypoosmolar hyponatremia;
b. Urine osmolality >100 mOsm/kg;
c. Urine sodium concentration >40 mEq/L;
d. Euvolemic state; and
e. Normal renal, thyroid, and adrenal function.[7]

Failure of improvement of serum sodium with intravenous normal saline further confirmed the diagnosis of SIADH. He did not have any other apparent cause of SIADH except influenza.

Although the exact pathogenesis for SIADH in influenza is unknown, it is thought to be related to the secretion of proinflammatory cytokines. Interleukin (IL)-2, IL-6, IL-1β, and tumor necrosis factor (TNF)-α have been reported to stimulate parvocellular and magnocellular neurons to secrete more ADH; thus causing SIADH.[8,9]

Fluid restriction is the main treatment modality in SIADH, with a suggested goal intake of less than 800 mL/day.[1] Further treatment options depend on the severity of hyponatremia and the presence of other related symptoms. In the presence of severe or symptomatic hyponatremia, hypertonic saline is recommended. Goal should be to raise serum sodium less than 10-12 mEq/Lin 24 h to ovoid the potential complication of osmotic demyelination with rapid correction.[10] Oral salt tablets and loop diuretics may also be added if optimum response is not seen with fluid restriction alone. Effort should be made to identify and treat the underlying cause. Our patient's SIADH was thought to be secondary to influenza, which was treated with oseltamivir and led to steady improvement in the patient's serum sodium levels.

In conclusion, clinicians should be cognizant of the association between influenza and SIADH to allow for accurate diagnosis and treatment of this condition. Further studies are needed in future to find out the incidence and pathogenesis of SIADH in patients with influenza. Although treatment depends on the severity of hyponatremia and associated symptoms, fluid restriction remains the cornerstone of therapy. Specific treatment aimed at influenza may help correct the hyponatremia in these cases.

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