

Influenza as a Cause of SIADH Related Hyponatremia: A Case Report

BHAVITA GAGLANI¹, SORAB GUPTA², OCTAVIO CHAVEZ³, RUEDA LIBARDO⁴

ABSTRACT

Syndrome of Inappropriate Secretion of Antidiuretic Hormone (SIADH) is one of the most common causes of hyponatremia in hospitalized patients. The distinct aetiologies and co-morbidities associated with hyponatremia pose substantial challenges in identifying and managing this disorder. Several infectious causes of SIADH are reported but hyponatremia associated with SIADH and influenza virus infection is less commonly seen. We present a case of hyponatremia associated with influenza, which was subsequently diagnosed as SIADH.

Keywords: Anti-diuretic hormone, Flu, Low sodium, Viral infection

CASE REPORT

A 75-year-old paraplegic male presented to the Emergency Department complaining of non productive cough and chest pain for one day. Chest pain was generalized, moderate in intensity and exacerbated by cough. No additional symptoms were reported. He did not take any medications and denied smoking, alcohol consumption or drugs. He was paraplegic due to a gunshot wound injury at lumbar region of the back several years ago. He was afebrile, had a blood pressure of 107/69 mmHg, heart rate of 87 beats per minute and respiratory rate of 18 breaths per minute with oxygen saturation of 98% on room air. Physical exam was only significant for diminished muscle strength 0/5 in lower extremities bilaterally and absent deep tendon reflexes. Laboratory findings were significant for a white blood cell count of 19,340/ μ l with a left shift deviation (87.2% neutrophils) and serum sodium (Na) of 130 mEq/l. Baseline serum Na was 137 mEq/l noted on prior admission for a urinary tract infection four months ago. Kidney function test was normal. A chest x-ray did not show any infiltrates or masses. Urine legionella antigen test was negative. Electrocardiogram showed no abnormality. Rapid flu test was positive for influenza type A. Cultures of urine, sputum and blood were negative for any microbial pathogen. He was started on oseltamivir 75 mg twice daily and fluid restriction was recommended for hyponatremia. Urine work up was significant for a high urine osmolarity of 631 mOsm/kg, low serum osmolality of 261 mOsm/kg with normal urine electrolytes including urinary sodium 46mEq/l. Thyroid stimulating hormone and morning cortisol levels were normal. Chest and brain computed tomography scans were unremarkable. Since all workup for pulmonary and central nervous system disorders remained negative, the patient was diagnosed as having SIADH due to underlying Influenza A virus infection. Patient was followed up after three weeks in the clinic. Leukocytosis was resolved and repeat sodium levels were found to be normal.

DISCUSSION

Causes of SIADH includes wide variety of differential diagnosis such as malignancy, pulmonary conditions (such as severe obstructive lung disease, acute respiratory failure, pneumonia, tuberculosis, abscesses etc.), central nervous system disorders, medications (specially thiazide diuretics) and vasculitis including Kawasaki disease [1], all of which were less likely to be the cause of SIADH in our patient. Hyponatremia is also most often reported within the first two weeks after Spinal Cord Injury (SCI) but it can be observed even later [2]. Our patient had history of SCI many years ago, and

presented with normal range of sodium levels in prior admission, making SCI as less likely cause this acute hyponatremia.

In the study as Lim GW et al., it has been mentioned that serum sodium inversely correlated with neutrophils, C-reactive protein, and N-terminal pro-Brain Natriuretic Peptide (NT-proBNP) [1]. The exact pathogenesis for SIADH is thought to be related to the secretion of inflammatory cytokines- Interleukin (IL)-2, IL-6, IL-1 β , and Tumor Necrosis Factor (TNF)- α strongly stimulates the human Hypothalamic-Pituitary-Adrenal (HPA) axis which have been reported to stimulate parvocellular and magnocellular Arginine Vasopressin (AVP) neurons to secrete more Anti Diuretic Hormone (ADH); thus, causing SIADH [1,3-6]. In our patient, no other clear bacterial source of leukocytosis was present.

The accepted diagnostic criteria for SIADH require evaluation of volemic status, serum sodium concentration, serum osmolality, urine sodium concentration, urine osmolality, thyroid function tests and serum cortisol, alongside a medical history to check salt and water intake, renal function, and diuretic use [3,4,6]. Our patient was diagnosed with SIADH based on hypo-osmolar hyponatremia, urine osmolality >100 mOsm/kg, urine sodium concentration >40 mEq/l, euvolemic state with normal renal, thyroid, and adrenal functions.

The mainstay of treatment for most patients with SIADH is fluid restriction with a target intake of less than 800 ml/day. Oral salt tablets and loop diuretics can be added if optimum response is not achieved with the initial treatment. In the presence of severe or symptomatic hyponatremia, hypertonic saline is the treatment of choice [3-7].

In our patient, successful correction of sodium levels to normal range with water restriction and treatment with oseltamivir for influenza led in our diagnosis of Influenza to be the suspected cause of SIADH related hyponatremia.

CONCLUSION

Association between influenza and SIADH needs to be identified, requiring clinicians to be aware of this condition, which can ultimately allow accurate diagnosis and treatment. Further studies are needed to find out the incidence and pathogenesis of SIADH in patients with influenza so that specific treatment protocols to help correct the hyponatremia in these cases can be devised. Once the cause of SIADH is identified, the ultimate solution is treating the underlying aetiology.

REFERENCES

- [1] Lim GW, Lee M, Kim HS, Hong YM, Sohn S. Hyponatremia and syndrome of inappropriate antidiuretic hormone secretion in kawasaki disease. *Korean Circ J*. 2010;40(10):507-13.
- [2] Kriz J, Schuck O, Horackova M. Hyponatremia in spinal cord injury patients: new insight into differentiating between the dilution and depletion forms. *Spinal Cord*. 2015;53:291-96.
- [3] Mastorakos G, Weber JS, Magiakou MA, Gunn H, Chrousos GP. Hypothalamic-pituitary-adrenal axis activation and stimulation of systemic vasopressin secretion by recombinant interleukin-6 in humans: potential implications for the syndrome of inappropriate vasopressin secretion. *J Clin Endocrinol Metab*. 1994;79(4):934-39.
- [4] Henry DA. In the clinic: hyponatremia. *Ann Intern Med*. 2015;163(3):ITC1-19.
- [5] Anderson RJ. Hospital-associated hyponatremia. *Kidney Int*. 1986;29(6):1237-47.
- [6] Pathak R, Khanal A, Poudel DR, Karmacharya P. Down with the flu: Hyponatremia in a patient with influenza. *N Am J Med Sci*. 2015;7(5):227-28.
- [7] Verbalis JG, Goldsmith SR, Greenberg A, Korzelius C, Schrier RW, Sterns RH, et al. Diagnosis, evaluation, and treatment of hyponatremia: expert panel recommendations. *Am J Med*. 2013;126(10 Suppl 1):S1-42.

PARTICULARS OF CONTRIBUTORS:

1. PGY3 (R3) Resident, Department of Internal Medicine, St. Barnabas Hospital, Bronx, New York, USA.
2. PGY3 (R3) Resident, Department of Internal Medicine, St. Barnabas Hospital, Bronx, New York, USA.
3. PGY2 (R2) Resident, Department of Internal Medicine, St. Barnabas Hospital, Bronx, New York, USA.
4. PGY2 (R2) Resident, Department of Internal Medicine, St. Barnabas Hospital, Bronx, New York, USA.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Bhavita Gaglani,
2562 Laconia ave, Bronx, New York, USA.
E-mail: gaglanibhavita@gmail.com

Date of Submission: **Dec 08, 2016**

Date of Peer Review: **Dec 27, 2016**

Date of Acceptance: **Feb 14, 2017**

Date of Publishing: **May 01, 2017**

FINANCIAL OR OTHER COMPETING INTERESTS: None.