CLINICAL VIGNETTE

Cerebral Salt Wasting in a Patient with Recent Head Trauma

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Case Presentation

A 58-year-old man with Type II Diabetes presented after 3 weeks of headaches. He had a recent motor vehicle accident requiring surgery. Headaches had worsened and occurred constantly at night. They were not associated with any muscle weakness, numbness, tingling, or other focal neurological defects. He had tried acetaminophen, which alleviated the pain temporarily. The pain was described as a diffuse pressure like sensation throughout his head and neck. He did not have associated abdominal pain, nausea, nor vomiting. He did report general malaise and poor appetite, eating less than usual. He denied any seizures or changes in mental status. He did not have prior traumatic injuries noted or history of migraine.

The motor vehicle accident resulted in a left tripod fracture requiring open reduction internal fixation with plate/screw as well as subdural hemorrhage. After the motor vehicle accident, he was told he had low sodium and prescribed salt tabs for several days before running out. We did not have access to labs regarding his outpatient management.

On presentation, patient was afebrile, normotensive, with normal vital signs. His physical exam was unremarkable except for dry mucous membranes. Neurological exam revealed fully alert and oriented patient with, no facial droop, moving all extremities, with normal sensation and 5/5 strength in all extremities. Laboratories included normal complete blood count, comprehensive metabolic panel significant for sodium of 118, serum osmolality of 259, and normal urinalysis. CT of the head showed minimal dural thickening consistent with prior subdural hematoma and old fractures without other abnormalities. Nephrology was consulted who attributed hyponatremia to syndrome of inappropriate antidiuretic hormone (SIADH) in the setting of recent head trauma. They recommended sodium chloride tablets BID, fluid restriction, urine sodium/osmolality, and monitoring urine output every two hours, without additional IV normal saline. Headache was thought to be a tension headache from his recent motor vehicle accident. Urine osmolality was 570 with urine sodium of 135 consistent with SIADH. Serum sodium did not improve initially, so sodium chloride tabs were increased to TID. Given continued sodium hovering around 118, 3% normal saline was added and the fluid restriction lifted. He was noted to be drinking significant amounts water. Fludrocortisone 0.2 mg PO BID was also added which allowed 3% sodium to be tapered off. His sodium increased to 130 by discharge with follow-up in nephrology and primary care. Since discharge he has been titrated off of salt tabs with sodium increasing to 133 and plans to titrate off fludrocortisone. His headache has resolved.

Discussion

The syndrome of inappropriate secretion of antidiuretic hormone (SIADH) involves changes in the expulsion of water from our body due to the secretion of antidiuretic hormone (SDH) and malfunction of the physiological suppression of this hormone, leading to decreased urine output. Whenever free water intake exceeds urine output, sodium concentration in blood decreases, leading to hyponatremia.[1] Common laboratory markers include high urine osmolality with urine sodium levels > 40 mEq/L.[1] This is consistent with impaired water excretion with lower water excretion of concentrated urine. Patients without this syndrome, have water intake suppressing excretion of ADH, leading to more dilute and voluminous urine.

There are many known causes for SIADH including CNS disturbances, malignancies, medications, surgery, pain, pulmonary disease, hormonal abnormalities, as well as idiopathic.[1] Cerebral salt wasting can be included with SIADH, but the primary disorder is the body's loss of salt/wasting in the urine due to CNS pathology. This may be seen with subarachnoid hemorrhage, which leads to loss of volume and increased secretion of ADH.[2] Two different mechanisms affecting neurological input have been described. The nervous system regulates sodium, uric acid and water reabsorption in the proximal tubule. Neurological deficits can lead to reduced sodium/uric acid absorption.[2,3] In brain injury, brain natriuretic peptide is released and this factor may also impair sodium reabsorption in the renal tubules. Cerebral salt wasting should be considered volume loss is present, such as hypotension with elevated urine sodium, and CNS injury.[4,5]

The treatment of SIADH includes three different components: treatment of the patient's underlying condition leading to SIADH, initial attempts to increase serum sodium, and further therapy if the SIADH state continues, especially if underlying condition cannot be reversed.[6,7] The mainstays of treatment include fluid restriction and high solute intake. High solute intake can be achieved with a high sodium and high protein diet, or salt tabs and/or urea intake.[6,7] Hyponatremia that does not respond, is severe, or causing symptoms will require rapid

raising of the sodium level with intravenous hypertonic saline. Concentration of sodium/electrolytes in the fluids being administered has to be higher than this concentration in the urine, otherwise there will be no effect and may worsen the hyponatremia.[4,8]

Treatment of cerebral salt wasting is similar to SIADH treatment. However, patients with neurological problems, especially subarachnoid hemorrhage should not be treated with fluid restriction, as these patients may be hypovolemic. In more urgent cases, 3% saline is used to raise the serum sodium. Parenteral therapy can then be transitioned to oral therapy with sodium chloride tablets once sodium has improved and the patient is more stable. If needed, a mineralocorticoid like fludrocortisone also can be used.[9-11] Cerebral salt wasting usually resolves within a few weeks, especially if the underlying neurological problem is resolving.[3]

Conclusion

SIADH and/or cerebral salt wasting should be considered in all hyponatremia patients with neurological afflictions such as masses or hemorrhage. Laboratory findings of low serum sodium, elevated urine osmolality, and elevated urine sodium levels can point toward diagnosis and guide treatment. Keeping these diagnoses on the differential allows more rapid diagnosis and treatment. Wrong treatment, such as isotonic intravenous fluids, may actually worsen hyponatremia, leading to increased morbidity and mortality.

REFERENCES

1. **Rose BD, Post TW**. *Clinical Physiology of Acid-Base and Electrolyte Disorders, 5th ed.* New York: McGraw-Hill; 2001. p.703.
2. **Palmer BF**. Hyponatremia in patients with central nervous system disease: SIADH versus CSW. *Trends Endocrinol Metab*. 2003 May-Jun;14(4):182-7. doi: 10.1016/s1043-2760(03)00048-1. PMID: 12714279.
3. **Palmer BF**. Hyponatraemia in a neurosurgical patient: syndrome of inappropriate antidiuretic hormone secretion versus cerebral salt wasting. *Nephrol Dial Transplant*. 2000 Feb;15(2):262-8. doi: 10.1093/ndt/15.2.262. PMID: 10648680.
4. **Singh S, Bohn D, Carlotti AP, Cusimano M, Rutka JT, Halperin ML**. Cerebral salt wasting: truths, fallacies, theories, and challenges. *Crit Care Med*. 2002 Nov;30(11):2575-9. doi: 10.1097/00003246-200211000-00028. PMID: 12441772.
5. **Carlotti AP, Bohn D, Rutka JT, Singh S, Berry WA, Sharman A, Cusimano M, Halperin ML**. A method to estimate urinary electrolyte excretion in patients at risk for developing cerebral salt wasting. *J Neurosurg*. 2001 Sep;95(3):420-4. doi: 10.3171/jns.2001.95.3.0420. PMID: 11565862.
6. **Rose BD**. New approach to disturbances in the plasma sodium concentration. *Am J Med*. 1986 Dec;81(6):1033-40. doi: 10.1016/0002-9343(86)90401-8. PMID: 3799631.
7. **Ellison DH, Berl T**. Clinical practice. The syndrome of inappropriate antidiuresis. *N Engl J Med*. 2007 May 17; 356(20):2064-72. doi: 10.1056/NEJMcp066837. PMID: 17507705.
8. **Taplin CE, Cowell CT, Silink M, Ambler GR**. Fludrocortisone therapy in cerebral salt wasting. *Pediatrics*. 2006 Dec;118(6):e1904-8. doi: 10.1542/peds.2006-0702. Epub 2006 Nov 13. PMID: 17101713.
9. **Misra UK, Kalita J, Kumar M**. Safety and Efficacy of Fludrocortisone in the Treatment of Cerebral Salt Wasting in Patients With Tuberculous Meningitis: A Randomized Clinical Trial. *JAMA Neurol*. 2018 Nov 1;75(11):1383-1391. doi: 10.1001/jamaneurol.2018.2178. Erratum in: *JAMA Neurol*. 2018 Nov 1;75(11):1443. PMID: 30105362; PMCID: PMC6248117.
10. **Albanese A, Hindmarsh P, Stanhope R**. Management of hyponatraemia in patients with acute cerebral insults. *Arch Dis Child*. 2001 Sep;85(3):246-51. doi: 10.1136/adc.85.3.246. PMID: 11517113; PMCID: PMC1718897.
11. **Kinik ST, Kandemir N, Baykan A, Akalan N, Yordam N**. Fludrocortisone treatment in a child with severe cerebral salt wasting. *Pediatr Neurosurg*. 2001 Oct;35(4):216-9. doi: 10.1159/000050424. PMID: 11694800.