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Case Report

A Case Report of Cerebral Salt Wasting in a 32 Year-Old, Following a Motor-Vehicle Accident, without Intracerebral Hemorrhage

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Abstract

Introduction: Cerebral Salt Wasting (CSW) is a rare etiology for hyponatremia. It is usually associated with neurotrauma, especially subarachnoid hemorrhage. In CSW, the kidney is unable to retain sodium, resulting in a dramatic hyponatremic hypovolemia, with high urine sodium and volume, resistant to hydration.

Case: A 32 year-old female without significant medical history presented with nausea and vomiting, 1 week after being a pedestrian struck by a vehicle. The original accident caused loss of consciousness, and post-concussive syndrome headaches, but no significant injuries, fractures or internal bleeding. Upon re-admission, she was orthostatic positive. She was lethargic, with a serum sodium of 119 and osmolality of 251. Notably, her urine osmolality was high at 406. Aggressive rehydration was started, however her hyponatremia remained dangerously low. A brief course of hypertonic saline was initiated, however her sodium remained uncorrected. She urinated excessively with each hydration attempt. Given her severe hypovolemic hyponatremia, with high urine output and sodium concentrations, seemingly resistant to rehydration, CSW was considered. She was started on maintenance fluids and fludricortisone. After 3 days, her sodium and hydration status began to resolve.

Discussion: Typical causes of hypovolemic hyponatremia are gastro-intestinal and renal loss. CSW is differentiated as dramatic hypovolemic hyponatremia with high urine sodium and output, that's refractory to typical hydration. It is most commonly found in patients with subarachnoid hemorrhages. However, this case had no brain bleeding. The treatmentis fludrocortisone and observation, as this disease is usually self-limited. Of note, attempts at sodium-based rehydration can exacerbate hyponatremia.

INTRODUCTION

Cerebral Salt Wasting (CSW) is a rare etiology for hyponatremia. It is associated with neurotrauma, including surgery and most commonly, subarachnoid hemorrhage (SAH) [1,2]. It presents as hypovolemic hyponatremia, with high urine volume and urine sodium, refractory to usual hydration [1]. This differentiates it from the more common causes of hypovolemic hyponatremia namely dehydration, gastro-intestinal (GI), and renal loss (such as in laxative and diuretic abuse).

Syndrome of inappropriate antidiuretic hormone (SIADH) can similarly present from neurotruma, with high urine osmolality and hyponatremia. However CSW is differentiated by hypovolemia. Given how attempts at rehydration can exacerbate hyponatremia in CSW, it is a vital differential that cannot go overlooked.

CASE PRESENTATION

A 32 year-old female without significant medical history presented with weakness, nausea and vomiting for one day.

One week prior, she was struck by a car while crossing the street. She suffered a witnessed loss of consciousness for reportedly 5 minutes. Upon arrival of emergency medical services, vitals were stable, she was awake, alert and oriented, but she had no memory of the event. Once in the emergency room, she was given a trauma evaluation. Abrasions were noted on her shoulders, elbows knees, cheek bones and brow. A computed tomography (CT) of her head was negative for hemorrhage, midline shift, or acute intracranial findings. Examination and imaging of her remaining body was unremarkable for significant injuries, fractures or bleeds. Her neurological exam was benign, save for amnesia of the event, and residual headaches. She was diagnosed with a concussion, and post-concussive headaches. No further imaging or tests were done, and she was discharged a few days later.

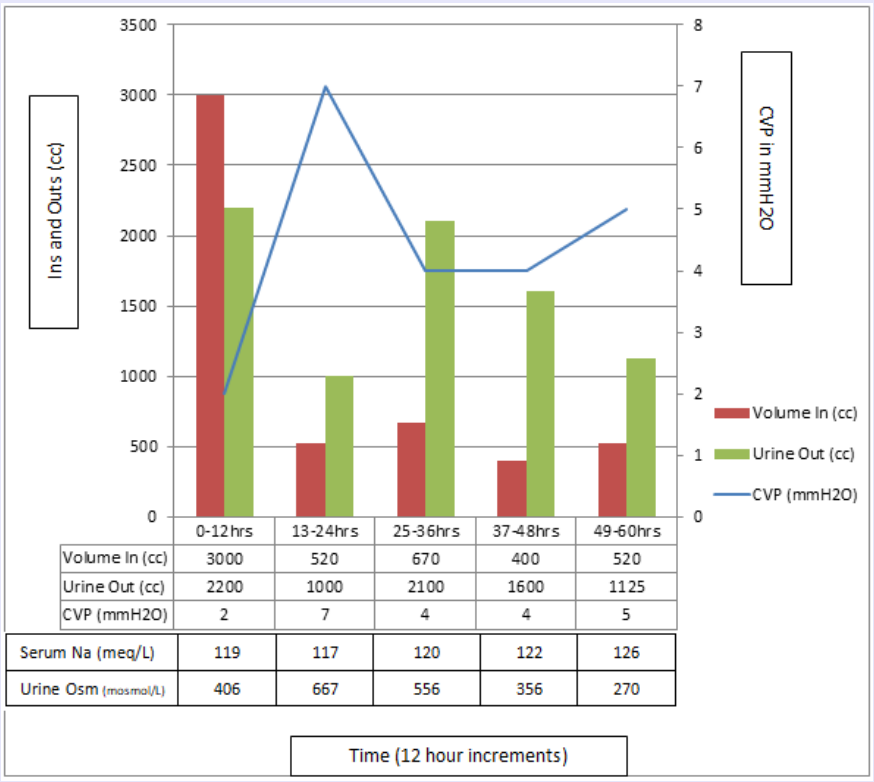
Upon re-admission, her vital signs revealed positive orthostatics. She complained of severe nausea and multiple episodes of vomiting. Physical exam showed dry mucus membranes, and lethargy. While she was complaining of

headaches, there were no focal neurological deficits, including tests for nystagmus, ataxia and meningeal signs. A repeat CT head was ordered and again returned negative.

Her labs revealed severe hyponatremia with a serum sodium of 119meq/L. Blood Urea Nitrogen (BUN) and Creatinine (Cr) were 8mg/dl and 0.5mg/dl respectively. In the setting of significant vomiting and dry mucus membranes, hypovolemic hyponatremia was considered. High urine osmolality (406mosmol/L), in combination with positive orthostatics, and a low-normal ProBrain Natriuretic Peptide (proBNP) of 78pg/ml, supported a low volume state.

A central line was inserted, and her CVP was measured between 2 and 4. She was hydrated with 3L of normal saline over 4 hours and her labs were re-drawn. Over that time, she urinated 2200ml, and her serum sodium decreased from 119 to 117meq/L. Her CVP peaked at 5 before falling back to 2, and she became increasingly lethargic. While an echocardiogram would have aided in assessing volume status, it was not done at the time.

As she was deteriorating, emergency effort was made to correct her sodium. Given her recent head trauma, elements of SIADH, superimposed over emesis-induced hypovolemic hyponatremia was considered, and she was started on 3% saline at 50ml/hr for 4 hours and then stopped. Repeat labs revealed no change in serum sodium (118meq/L). Her urine osmolality however, increased from 406 to 667mosmol/L. Her urine sodium returned at 174meq/L. (Figure 1 and Table 1). Her initial CT scan was followed-up by magnetic-resonance imaging (MRI) of the brain, which again was negative for intracerebral bleeds, or any acute parenchymal findings. Cerebrospinal fluid pressure was not taken.



	0-12hrs	13-24hrs	25-36hrs	37-48hrs	49-60hrs
Volume In (cc)	3000	520	670	400	520
Urine Out (cc)	2200	1000	2100	1600	1125
CVP (mmH2O)	2	7	4	4	5
Serum Na (meq/L)	119	117	120	122	126
Urine Osm (mosmol/L)	406	667	556	356	270

Figure 1 Volume-in (red bar) and Urine-out (green bar) measured in cubic centimeters on the Y-axis, is plotted in 12 hour intervals on the X-axis. Central Venous Pressure (Z-axis) is superimposed in mmH2O.

0-12 Hrs:

Initial serum sodium was 119meq/L, serum osmolality was 251mosmol/L and urine osmolality was 406mosmol/L. The initial 3000cc hydration resulted in a diuresis of 2200cc's, and a decreased serum sodium of 117meq/L (shown in the 0-12 hour block).

13-24hrs:

3% Saline was begun at the 12 hour mark, resulting in a transient CVP elevation and seeming improvement. However, serum sodium and osmolality held at 117meq/L and 150mosmol/L respectively. In response to the hypertonic saline, urine osmolality increased from 406 to 667mosmol/L.

25-36hrs:

As serum concentrations spiked and began to taper, urineosmolality peaked at 667 and began to fall to 556mosmol/L, causing the dramatic diuresis noted in hours 25-36, with a concomitant drop in CVP. 3% saline was abruptly held as a result.

37-48hrs:

CSW was considered, and Fludrocortisone was started during the 37-48 hour block, with limited, maintenance-only, normal saline hydration. Within 24 hours, diuresis began to normalize and CVP began to improve. By day 5, her serum sodium and osmolality had normalized, and her mental status had returned to baseline.

Table 1: Tabular format of graph in Figure 1. Serum electrolytes were taken every 4 hours, along with Urine Osmolality. * indicates time when 3% saline was administered. Target CVP was started Day 2, with instructions to stop fluids if CVP hit 5, and resume when it dropped. Fludricortisone in addition to 100ml/hr of NS with the same CVP target was started during hours 37-48.

	0-12hrs	13-24hrs*	25-36hrs	37-48hrs	49-60hrs
Serum Na(meq/L)	119	117	120	122	126
CVP(mmH2O)	2	7	4	4	5
Treatment	3L NS	200ml 3%NS	100ml/hr NS Target CVP <5	Fludricortisone + 100ml/hr NS	Fludricortisone + 100ml/hr NS
Vol In(ml)	3000	520	670	400	520
Urine Out(ml)	2200	1000	2100	1600	1125
Urine Osm(mosmol/L)	406	667	556	356	270

Given severe hypovolemic hyponatremia, with elevated urine sodium concentrations, extreme diuresis and apparent resistance to hydration, CSW was considered. She was started onfludrocortisone0.4mg every 12 hours and normal saline at 100ml/hr. A conservative maximum CVP was set at <5mmH2O. Serial chemistries involving serum and urine sodium and osmolality were drawn. Over the next 24 hours her serum sodium and osmolality remained low. However, her urine began to improve. Urine sodium decreased from 174 to 110meq/L and urine osmolality decreased from 667 to 356mosmol/L. Renin and aldosterone levels would have been instructive in measuring recovering volume and renal status. Unfortunately, they were not drawn.

On day 3, her serum sodium began to normalize, initially overcorrecting to 126meq/L. Serum osmolality followed, increasing from 250 to 260mosmol/L. With normalization of her serum electrolytes, she began to clinically improve. This occurred despite a persisting negative fluid balance. She was started on 5% Dextrose in water on day 4, and her serum sodium plateaued at 130meq/L. By day 5, both her serum and urine osmolality and sodium reached normal limits, and her lethargy resolved. Her CVP had increased to 6, and her sodium held at 143meq/L. She was monitored for a few more days and discharged in normal health.

DISCUSSION

Pathophysiology

The mechanisms of CSW are poorly understood, and its existence has been contested [1,3]. At its core, CSW is the inability of the kidneys to resorb sodium. The cation thus collects in the distal tubule causing an increased osmotic gradient, resulting in severe sodium and water loss [2,4]. Accordingly, attempts at sodium-based crystalloid hydration only provide more substrate for renal excretion. Fluid in becomes fluid out. Two mechanisms to explain this have been proposed: trauma 1) disrupting the neural input to the kidney, or 2) increasing the release of diuretic hormones [4,5].

The sympathetic nervous system controls many aspects of the nephron, including the renin-aldosterone system. Inhibited renin synthesis could impair aldosterone activity, and result in electrolyte wasting [4]. Unfortunately in this case, serum aldosterone levels were not measured.

An alternative explanation posits that neural injury itself can cause natriuretic hormone release, and inhibit salt balance [1]. One small study [6], found that increasing intracranial pressure from SAH, resulted in higher serum levels of brain natriuretic peptide (BNP), which correlated with increased sodium excretion. Our patient had low-normal proBNP, which correlates with hypovolemia but not the aforementioned study. Furthermore, we did not obtain a cerebrospinal fluid sample to measure intracranial pressure.

Epidemiology

CSW is most frequently observed in neuro-intensive care units [1,2,5,7]. SAH is the most commonly associated insult [2]. The incidence is poorly documented, but one study [7] found that in SAH patients with hyponatremia, only 7% were attributable to CSW, (with 69% attributed to SIADH). CSW has also been reported in cases of meningitis, poliomyelitis, central nervous malignancy and neurosurgery [2]. CSW usually peaks within 10 days of the neurological insult, and resolves on its own with supportive care [8].

Work-Up

The first step in assessing hyponatremia, is determining volume status. Hypovolemic hyponatremia most often occurs from volume loss: either gastro-intestinal due to vomiting or diarrhea, or renal loss from excessive diuresis.

CSW is a more rare cause of hypovolemic hyponatremia. It is associated with recent neural insult, and presents with comparatively high urine sodium and output. It is refractory to hydration. This should be striking as hypovolemic patients should usually retain both urinary sodium and volume.

It is important to differentiate CSW from GI and renal loss, as the latter two are treated with hydration. As mentioned, sodium-based crystalloids can actually exacerbate hyponatremia in CSW.

There is also significant laboratory and comorbidity overlap in CSW and SIADH [2,4]. Both SIADH and CSW are believed to stem from neurotrauma, and both can cause hyponatremia with high urine osmolality. However, they are differentiated by volume status, as CSW is markedly hypovolemic [2,4]. Again, differentiation is vital, as SIADH is treated with volume restriction or hypertonic saline, both of which could exacerbate hyponatremia in CSW [9].

Treatment

The treatment for CSW consists of supportive, maintenance-

only hydration and mineralocorticoids such as fludrocortisone [10]. Dosage is untested, but most case-reports have focused on 0.2mg to 0.4mg/day [10]. In theory, this treatment targets the distal tubule, inducing potassium excretion and sodium resorption, in an effort to counter salt wasting elsewhere in the nephron. As CSW is usually self-limited, there's no chronic treatment [5]. Recall that while first instincts are to replenish water loss, increased urination in response to hydration can exacerbate hyponatremia, but help seal the diagnosis.

Our patient was initially treated with normal saline. But given the fear of superimposed SIADH from a previous neural trauma, and worsening lethargy, hypertonic saline was given. As a result, our patient suffered volatile volume swings, as CVP initially rebounded, then crashed as the kidneys urinated out the hypertonic saline. Conivaptan/Tolvaptan, second line treatments for SIADH should also prove fruitless in CSW, as it will negate what little water resorption the kidneys can perform in the collecting duct.

CONCLUSION

This is an atypical case of CSW, as it occurred in the absence of SAH. However, the patient did suffer a previous concussion with loss of consciousness and residual headaches. While significant, non-hemorrhagic trauma has not been well-documented as a cause of CSW. Our patient presented with hypovolemic hyponatremia, initially presumed to be secondary to volume loss through hyperemesis. However, initial resuscitation with 3L normal saline resulted in 2.2L of sodium-concentrated, urine loss. Given her recent neurotrauma and worsening lethargy, concomitant SIADH was entertained, and hypertonic saline was given in the hopes of arresting her decompensation. While transient improvement was seen, within hours she began dumping salt and volume again in her urine.

More thoughtful consideration implicated CSW as our culprit. Hypovolemic hyponatremia, refractory to both isotonic and hypertonic hydration, with increased urinary volume and sodium, is classic for CSW. In retrospect then, the administration of hypertonic saline was unnecessary and risky. The patient was started on a higher dose of fludrocortis one than usually reported (0.8mg/day instead of 0.2-0.4mg/day), and maintenance-only fluids. Within 24 hours she began to improve.

On day 3, her clinical mentation improved as her labs began to trend towards normal, however correction of her volume status lagged days behind. Rehydration was done slowly for fear of precipitating more hyponatremia. It is notable then, that her mentation improved with her electrolyte replacement well before return of hereuvolemic status. This is logical, as dehydration itself rarely leads to altered mental status. Significant electrolyte disturbances are usually more alarming than oscillations in volume. Given its rarity, ease of mistreatment (as noted here), and paucity of treatment options, CSW remains a challenging diagnosis, which warrants consideration in hypovolemic hyponatremia.

DECLARATIONS

Authors Contribution

Both authors contributed to the treatment of this patient, research, and presentation of this case.

Ethics approval and consent

This is a case report without identifying information, and does not require consent or an ethics review. Accordingly, all work was done in accordance with the Declaration of Helsinki.

Availability of data and material

The in-text figure is original research, created based upon hospital records. Reproduction and data is available upon request. Please make requests of the corresponding author via email.

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