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Sequential cerebral salt wasting complicating SIADH in a patient following head trauma

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ABSTRACT

Hyponatremia is the most commonly encountered dyselectrolytemia following head trauma. The two main mechanisms responsible for non-iatrogenic hyponatremia are cerebral salt wasting (CSW) syndrome and the syndrome of inappropriate antidiuretic hormone secretion (SIADH). SIADH is the commonest dyselectrolytemia cause of hyponatremia following traumatic brain injury (TBI) whereas CSW is the most elusive and challenging diagnosis of the causes of hyponatremia from intracranial causes. The need to distinguish between CSW and SIADH is critical because the management of CSW is volume restitution and sodium restoration whereas for SIADH, the management is exact opposite - water restriction. Our recent experience with a 67-year old Caucasian female post-TBI illustrated very interesting observations. To our knowledge, this is the first case of the sequential development of symptomatic hyponatremia from SIADH followed by the development of hyponatremia from CSW in the same patient during the same admission. Furthermore, our case further highlighted the contrarian observation that with a high index of suspicion for CSW and its early diagnosis, volume depletion and hypovolemia from polyuria may not be a distinguishing presenting factor, when contrasted with SIADH.

Case Report

Implication for health policy/practice/research/medical education:
We report a 67-year-old female who presented with progressively symptomatic hyponatremia due to the SIADH secretion complicating TBI and neurosurgical intervention. She responded initially to fluid restriction, 3% NaCl infusion and oral NaCl tablets. Nevertheless, by hospital day 8, she had quickly developed polyuria, dumping over 3 liters of urine in the first 4-6 hours of that morning with rapid recurrence of hyponatremia, again. She was, this time, diagnosed with CSW and was managed differently with 0.9% NaCl volume expansion. To our knowledge, this is the first case of the sequential development of symptomatic hyponatremia from SIADH followed by the development of hyponatremia from CSW in the same patient during the same admission. Furthermore, our case further highlighted the contrarian observation that with a high index of suspicion for CSW and its early diagnosis, volume depletion and hypovolemia from polyuria may not be a distinguishing presenting factor, when contrasted with SIADH.

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Introduction

Hyponatremia is the most commonly encountered dyslectrolytemia following head trauma (1-4). There is a global consensus that in the context of cerebral diseases, the two main mechanisms responsible for non-iatrogenic hyponatremia are cerebral salt wasting (CSW) syndrome and the syndrome of inappropriate antidiuretic hormone secretion (SIADH) (1-4). Although CSW was first described nearly 70 years ago by Peters et al (5), before SIADH was first reported, SIADH is the more common

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cause of hyponatremia following head and brain trauma or injury (6-10). Nevertheless, CSW is the most elusive and challenging diagnosis of the causes of hyponatremia from intracranial causes (9). The distinction between CSW and SIADH can be difficult and indeed, there has been speculation as to whether, CSW is truly a distinct disease entity (11). The need to distinguish between CSW and SIADH is critical because the management of CSW is volume restitution and sodium restoration whereas for SIADH, the management is exact opposite - water restriction (7-9). Another consensus view is that in CSW, there is always hypovolemia and often hypotension in contradistinction with the usual presence of hypervolemia and volume expansion in SIADH (7-9). Our recent experience with a 67-year old Caucasian woman following a fall and associated head injury followed by a neurosurgical operation illustrated very interesting observations. The one index patient presented initially with post-traumatic symptomatic hyponatremia secondary to SIADH and by the second week of the admission had transformed to CSW syndrome that required a radically different management approach. To our knowledge, this is the first case of the sequential development of symptomatic hyponatremia from SIADH followed by the development of hyponatremia from CSW in the same patient during the same admission. Furthermore, our case further highlighted the contrarian observation that with a high index of suspicion for CSW and its early diagnosis, volume depletion and hypovolemia from polyuria may not be a distinguishing presenting factor, when contrasted with SIADH.

Case Report

The patient is a 67-year-old Caucasian female with history of alcohol dependence admitted on account of traumatic brain injury (TBI) following a fall, down a flight of stairs. There was reported loss of consciousness, lasting for more than one hour. Non-contrast CT head examination showed multiple facial and skull vault fractures as well as blowout fracture of the right orbit, with several areas of intracranial hemorrhage including epidural hematoma with a midline shift, and severe edema of the right cerebellar hemisphere. Her Glasgow Coma Scale score on arrival to our facility was 14. However, she decompensated rapidly, necessitating intubation and ventilatory support. The following day, she underwent a right sub-occipital craniotomy and evacuation of epidural hematoma, with placement of an external ventricular drain. Her immediate post-operative course was uneventful and she was extubated the following day. The rest of her facial and orbital injuries were managed non-operatively as per recommendations by the ENT and the Ophthalmology services, respectively.

Her admission serum sodium level was normal at 141 mmol/L. However, on hospital day 4, she had developed progressive hyponatremia with serum sodium of 132 mmol/L likely secondary to post-TBI SIADH (Figure 1). The patient was initially started on oral NaCl salt tablets 2 g four times daily by the surgical service together with total fluid restriction. However, the serum sodium level continued to steadily decline and on hospital day 6, the serum sodium level had decreased to as low as 123 mmol/L (Figure 1). 3% hypertonic NaCl infusion was started, initially at the rate of 30 cc/h. The patient responded to the treatment and within 24 hours of starting the infusion, the serum sodium level had improved to 128-130 mmol/L (Figure 1). Indeed, serum Na was normal at 140 mmol/L by the morning of hospital day 9 (Figure 1).

Nevertheless, subsequently, by noon on the same hospital day 9, the patient had suddenly become polyuric dumping >3 L of urine, while still receiving the 3% hypertonic NaCl infusion and oral NaCl tablets (Figures 1 and 2). By the afternoon of hospital day 9, the serum sodium had quickly plummeted down to 128 mmol/L (Figures 1 and 3).

Nephrology consultation was obtained for acutely worsening hyponatremia. Except for some mild discomfort in the left side of the head, she had denied any other new symptoms. There was no headache, nausea or vomiting. Appetite was great and she had just completed a good lunch meal without incident before we saw and examined her. Vital signs were stable - Temperature 36.7°C, heart rate 94 bpm, respiratory rate 16/min, blood pressure 142/77 mm Hg, with a pulse oximetry reading of 98% on room air. She was alert, cheerful and non-focal, moving all four limbs

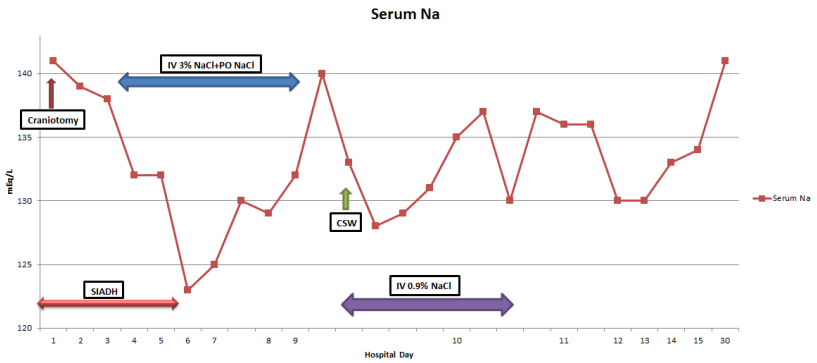


Figure 1. Translational changes in serum Na during the index admission with SIADH followed by CSW.

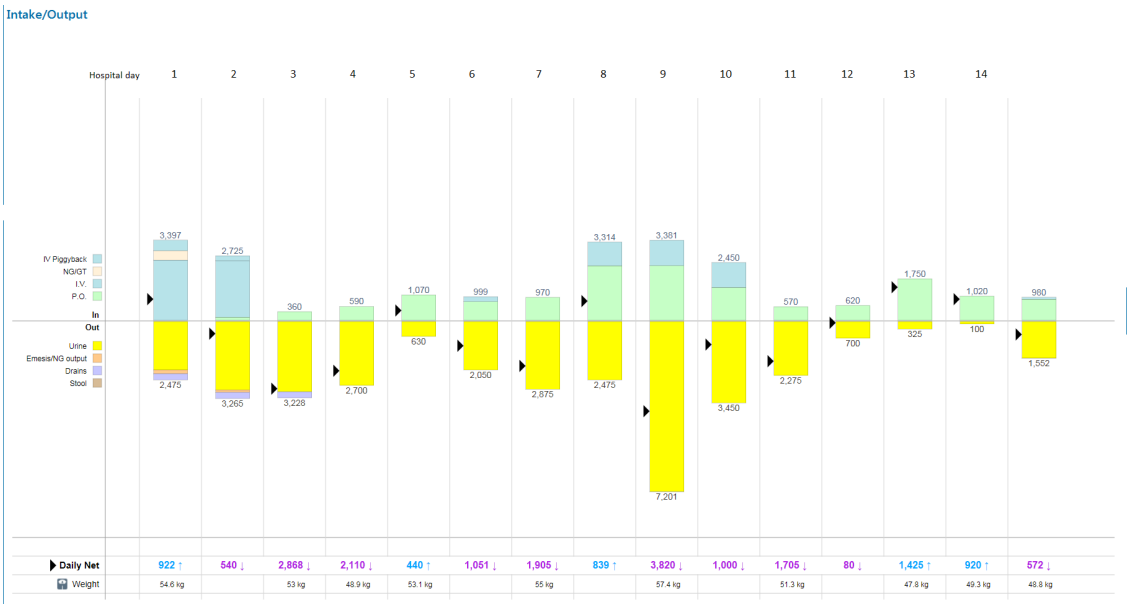
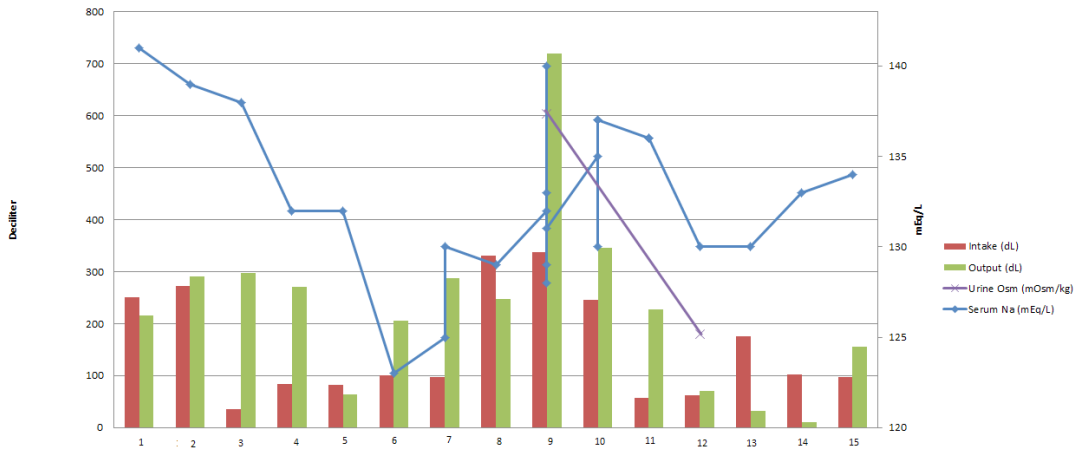


Figure 2. Intake and output record showing massive polyuria of over 7 L on hospital day 9.



Hospital Day	1	2	3	4	5	6	7	8	9					10				11		12	13	14	15
Intake (dL)	250.5	272.5	36	84	82	99.9	97	331.4	338.1					245				57		62	175	102	98
Output (dL)	215	290	297.5	270	63	205	287.5	247.5	720					345				227.5		70	32.5	10	155
Urine Osm (mOsm/kg)									603											179			
Serum Na (mEq/L)	141	139	138	132	132	123	125	130	129	132	140	133	128	129	131	135	137	130	137	136	136	130	130	133	134

Figure 3. Composite of translational changes in serum Na, urine output, urine osmolality and fluid intake during the admission.

appropriately. There was no lower extremity edema. The healing craniotomy wound was otherwise unremarkable. Overnight intake/output showed a deficit of 4335 cc with a charted urine volume of 6575 cc (Figure 2).

The clinical diagnosis was made of CSW complicating post-TBI SIADH managed with 3% NaCl infusion and NaCl salt tablets. Simultaneous urine Na was inappropriately high at 233 mOsm/kg with an accompanying urine osmolality of 603 mOsm/kg, consistent with the diagnosis of CSW (Figure 3). Therefore, the 3% hypertonic NaCl was promptly discontinued and was replaced by 0.9% NaCl infusion, at 100 cc/h. Fluid restriction was lifted. Overnight, serum Na had improved up to 135 mmol/L and the rate of 0.9% NaCl was reduced to 50 cc/h and this was discontinued after another 12 hours. Simultaneously, oral NaCl tablets were also discontinued. Polyuria resolved promptly (Figure 2). Subsequently, the serum sodium level stabilized and remained above 130 mmol/L until discharge to a rehabilitation unit (Figures 1 and 3). During the admission, a urinary catheter was placed due to post-op urinary retention. Following removal of the urinary catheter, she developed symptomatic urinary tract infection (UTI) with urine culture growing E. coli, and she was treated uneventfully with Levaquin. A repeat serum Na completed two weeks post-discharge was normal at 141 mmol/L (Figures 1).

Discussion

We have described our experience managing first, symptomatic hyponatremia from SIADH complicating TBI and subsequent neurosurgical intervention. This was then followed by another episode of acute hyponatremia with polyuria and CSW, all occurring during the same admission. Necessarily, the management paradigm was quickly switched from fluid restriction and salt loading to 0.9% NaCl infusion to forestall volume depletion and hypotension usually associated with CSW (1-9). To our knowledge, this is the first description of the sequential development of symptomatic hyponatremia due to SIADH that is followed by the development of hyponatremia from CSW in the same patient during the same admission. Furthermore, our case further highlighted the contrarian observation that with a high index of suspicion for CSW and its early diagnosis, volume depletion and hypovolemia from polyuria may not be a distinguishing presenting factor, when contrasted with SIADH (7-9). Moderate to severe hyponatremia is acknowledged to increase inpatient mortality (12-14).

Conclusion

It is therefore mandatory that appropriate diagnosis of CSW and/or SIADH be promptly made and specific therapeutic options deployed as early as possible to limit patient morbidity and mortality.

Acknowledgments

This work is dedicated to the memory of a very dear friend and uncle, Mr. B.C. Uchendu, who passed away back home in Lagos, Nigeria, some months ago early in 2017.

Authors' contribution

MACO; conception, design, acquisition of data, data analysis, interpretation of data, literature review, drafting the article and final approval of manuscript. NA; drafting the article and final approval of manuscript. EJA; acquisition of data, interpretation of data, literature review, drafting the article and final approval of manuscript. UCO; drafting the article and final approval of manuscript. AK; design, acquisition of data, data analysis, interpretation of data, literature review, drafting the article and final approval of manuscript.

Conflicts of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the article.

Ethical considerations

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors. The patient has given her informed consent to publish this case report.

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