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Trauma – A Probable Cause of Siadh!! : A Case Report

Ocurrence of Recurrent hyponatremia due to syndrome of inappropriate antidiuretic hormone secretion following traumatic brain injury

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ABSTRACT:

One of the common electrolyte abnormalities seen in clinical practice is hyponatremia. SIADH accounts for 60% cases of all hyponatremias. Usually hyponatremia associated with TBI is transient and reversible. Hyponatremia in patients with brain injury is mostly due to inappropriate secretion of antidiuretic hormone (ADH) due to pituitary dysfunction after head injury. We present a case of 60-year-old female with history of TBI-associated hyponatremia with features of delayed recovery and recurrent hyponatremia. In this report, we emphasize the importance of identifying patients with attenuated recovery from brain injury, with complication of SIADH and acute hyponatremia. Special care needs to be taken on preventing the recurrence by avoiding the risk factors in such individuals. It is necessary to differentiate TBI-associated SIADH from other important causes of hyponatremia such as cerebral salt wasting, and hypocortisolism.The purpose of this case report is to highlight the need to look into rare causes of syndromes in clinical practice which are often missed.

I. CASE REPORT:

A 60-year-old female,homemaker,resident of chennai admitted in chettinad hospital and research institute with complaints of seizures, giddiness and vomiting since 5days following a traumatic brain injury. Patient presented to ER with 2-3 episodes of nonconvulsive seizures each lasting for 2 to 3 minutes with loss of consciousness with mild post ictal confusion, giddiness was insidious in onset and gradually progressive not associated with head movements, with no aggravating and relieving factors followed by vomiting with 2-3 episodes per day which is watery in consistency, containing food particles,nonbilious, non blood stained and non foul smelling with no aggravating and relieving factors,no episodes of headache and black out.

Patient had a history of Road traffic accident a motor vehicle collision 5 days back following which she developed the above mentioned complaints.GCS-15/15, No history of seizures, vomiting, ENT bleed at the time of the accident. Patient underwent CT BRAIN which showed no abnormality and was treated symptomatically and discharged with a diagnosis of concussion and closed head injury.

She was taking medications at the time of presentation for chronic kidney disease and systemic hypertension

ON EXAMINATION patient was drowsy, arousable at the time of admission during seizure episode,conscious, cooperative and oriented to time, place, person after seizures settled.afebrile. Vitals- Blood pressure:130/80mm hg; Pulse:85 bpm, Respiratory rate :18 cpmand SPO2: 98% @RA

She was clinically euvolemic with a urine output of 100ml/h and an overall minor positive net external fluid balance (total daily fluid input 2600ml, total daily fluid output 2450ml).

On Systemic examination:

Cardiovascular system: s1s2+, no murmurs.

Respiratory system: Bilateral air entry equal and no added sounds**.**

Per Abdomen: Soft and non tender and no organomegaly .

Central nervous system: No focal neurological deficit

INVESTIGATIONS: TLC: 5000; HB: 11.6g,other parameters within normal limits,serum sodium 110mmol/l (reference range: 135–143), serum osmolality 247mmol/kg (280–300), urine sodium 65mmol/l (>30) and urine osmolality 340mmol/kg suggesting SIADH .Her thyroid function was normal: fT_4 15.5pmol/l (9.1–19.6), TSH 2.29mU/l (0.3–5), and cortisol levels 17.03µg/dl (6.7-22.6)

An MRI brain and pituitary was performed to rule out secondary brain damage and surgical conditions

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like SDH,EDH,axonal injury or any vascular pathology post RTA, which was normal. An insulin tolerance test was not performed as repeated cortisol levels were considered sufficient especially a value >550 nmol/l.

TREATMENT GIVEN: Fluid restriction was strictly followed with total fluid intake calculated from previous day's urine output plus insensible water losses for the day.Inspite of that hyponatremia persisted, sopatient was treated with 3% NaCl and tolvaptan 15mg once a day and patient was symptomatically improved ,repeat sodium levels were in normal limits and patient was discharged after 5 days.

Following discharge she experienced giddiness and was readmitted with h/o altered level of consciousness with no seizures, recurrence of moderate hyponatremia was found which was again consistent with SIADH, requiring long-term fluid restriction and sodium correction.(She was most recently admitted in October 07, presenting with nausea, vomitings in the context of not adhering to his fluid restriction). She was clinically euvolaemic. Biochemistry was consistent with SIADH: serum sodium 122 mmol/l, serum osmolality 253 mmol/kg, urine sodium 54 mmol/l, urine osmolality 345 mmol/kg, TSH 0.55 mU/l (0.3–5), fT_4 12.6 pmol/l (9.1–19.6), cortisol at 0600 h 347 nmol/l (100–540). Serum sodium improved with strict adherence to fluid restriction.

Psychogenic polydipsia is an important consideration in those presenting with recurrent euvolaemichyponatraemia, but the fluid restriction required in this case to achieve normonatraemiawas severe and inconsistent to rule out a diagnosis of psychogenic polydipsia. There was no evidence of hypopituitarism, malignancy, intercurrent pulmonary disease or nervous system disorders.
The etiology of this chronic SIADH was considered to be most likely secondary to prior TBI.

II. DISCUSSION:

Transient hyponatremia due to SIADH is common in patients who have suffered TBI. Born et al. (2) evaluated 109 patients with a GCS ≤7 in the first 24 h after a severe head injury. During follow-up , 36 of 109 patients developed SIADH at various time periods until day 19. Though SIADH is a common early complication of TBI, it rarely persists or recurs. Only a few published case reports were available regarding patients suffering from persistent or recurrent hyponatremia due to chronic SIADH after TBI (3) (4) (5). Though there is no information about follow-up , no further seizures were reported.Management of hyponatremia in the initial episode is by hypertonic saline and fluid restriction, whilw the next two episodes were managed with fluid restriction alone. There is no further hyponatremic episodes reported by the patient beyond the mentioned time.

The pathophysiological mechanisms responsible for SIADH after TBI remain uncertain (1) (3). Injury to pituitary stalk or posterior pituitary region causing inappropriate non-osmotic hypersecretion of ADH is the common mechanism observed(1). In most of the patients ,hyponatremia resolves with management in short duration and hence considered as transient effect . Considereing the clinical presentation of our patient, it seems permanent selective damage to these structures is possible.

Recent expert guidelines of management of hyponatremia suggested the treatment based on the cause , presence of neurological symptoms which indicate the severity and the speed at which onset of hyponatraemia occurred (6) (7). Irrespective of the cause , the correction of acute symptomatic hyponatraemia is by hypertonic (3%) saline given either via bolus or continuous intravenous infusion (6) (7). Treatment of chronic hyponatraemia or hyponatraemia of indeterminate duration should involve a controlled and limited correction to avoid the neurological sequelae of osmotic demyelination (6) (7). For most cases of mild-to-moderate SIADH, fluid restriction is regarded as first line therapy (6). Long-term fluid restriction is often complicated by poor adherence, especially when cognitive damage has been sustained. Pharmacological therapy is considered in individuals where either the cause persists or fluid restriction is not effective as expected. Oral urea can increase urinary solute excretion and induce an osmotic diuresis (6) (8). This has shown promising results in the treatment of long-term SIADH with recurrent hyponatremia, but is generally poorly tolerated due to its unpleasant taste (8). Euvolaemic and hypervolaemichyponatremia is better managed with vasopressin receptor antagonists. Tetracycline antibioticDemeclocycline, which inhibits the action of antidiuretic hormone in the renal distal tubule has a significant role in the management of chronic hyponatremia, but the authors are not aware of any published data on its role in SIADH following TBI (6) (8). The recommended treatment dose is 600–1200 mg/day 6). This case highlights a possible long-term effect of TBI on the endocrine system and the challenges of complying with a long-term fluid restriction and the potential role of

demeclocycline in patients with chronic hyponatremia due to post traumatic SIADH

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