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Unique presentation of asymptomatic hyponatremia secondary to syndrome of inappropriate antidiuretic hormone (SIADH) in head injury patient.

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Abstract

Patients with severe and moderate head injuries frequentl y experience hyponatremia, although those who suffer fr om mild head injuries hardly ever do so.Traumatic brain i njury (TBI) is known to have a condition called hyponatr aemia caused by SIADH.The syndrome of inappropriate antidiuretic hormone secretion (SIADH) is frequently bro ught on by CNS pathology and is linked to a number of c linical conditions.Initial symptoms may include nausea, malaise, headache, lethargy, and minor cognitive impair ments. Seizures, cardiorespiratory distress, and coma ma y develop in more severe cases.

Clinical euvolemia, the lack of adrenal, thyroid, pituitary, or renal dysfunction, and/or the use of diuretic medicatio ns are further characteristics.

Keywords: Hyponatremia, Syndrome of inappropriate antidiuretic hormone, hematoma.

Introduction

The most common neurological causes of SIADH are subarachnoid haemorrhage (SAH), traumatic brain injury (TBI), brain tumour, and meningitis/encephalitis. Drug-related hyponatraemia may also occur and anticonvulsant drugs, specifically carbamazepine, are of particular relevance after brain injury. Essential diagnostic criteria include low serum osmolality (<280mmol/kg) with a high urine osmolality (>100mmol/kg) and urine sodium concentration (>30mmol/l). The syndrome of inappropriate antidiuretic hormone secretion (SIADH) causes excessive antidiuretic hormone (ADH) release, leading to water retention and, as a consequence, hyponatremia. Laboratory results show hyponatremia < 135 mmol/l. SIADH has been linked to a variety of aetiologies and is most commonly seen in combination with (pulmonary) malignancy, surgery, drugs, and all types of central nervous system disruptions. One of these CNS disruptions is traumatic brain injury (TBI). This article presents a case of SIADH in a 66-year-old female who presented with general tiredness, fever, and hyponatremia following a traumatic hematoma to her parietal occipital region.

Case Report

A 66-year-old female came to emergency department with complaints of generalised tiredness and fever for treatment. Before presenting to emergency department

patient had history of trauma to her head due to slip and fall. No history of headache, loss of consciousness, nausea and other specific systemic complaints. Patient was known case of diabetic and hypertensive and on regular medication. On examination patient was conscious, oriented, febrile and vitals were stable. All routine investigations were done and serum sodium was 112.4 mmol/l. In view of trauma to her head CT Brain was done which showed acute hematoma in right parietal occipital region of scalp (figure 1). In view of hyponatremia 3%NaCl (hypertonic saline) was given. Biochemistry was consistent with SIADH: repeat serum sodium was 118mmol/l, serum osmolality: 267mmol/kg, urine sodium :46mmol/l, urine osmolality :610mmol/kg. Patient was clinically asymptomatic. Surgery opinion was obtained in view of hematoma and orders followed. There was no evidence of hypopituitarism, malignancy, pulmonary disease and nervous system disorders. The aetiology of SIADH is most likely due to hematoma in parietal occipital region. Hyponatremia resolved (figure 2) in 3 days after hypertonic saline and fluid restriction. Patient general condition improved. Patient discharged and being followed up.

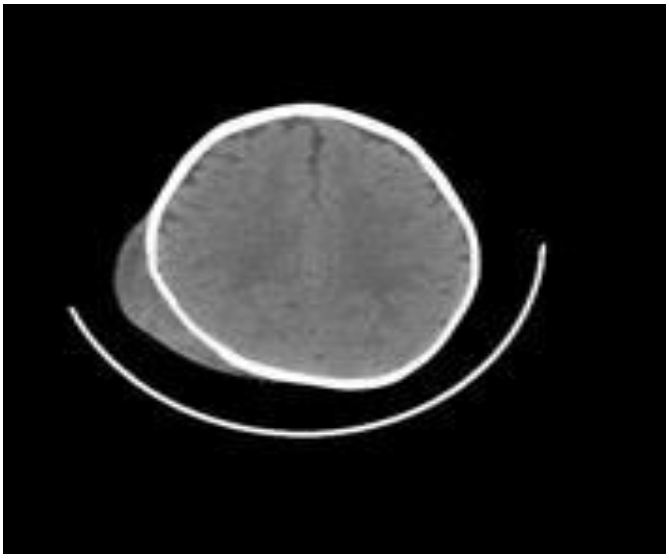

Figure 1: Scalp hematoma in right parietal occipital region

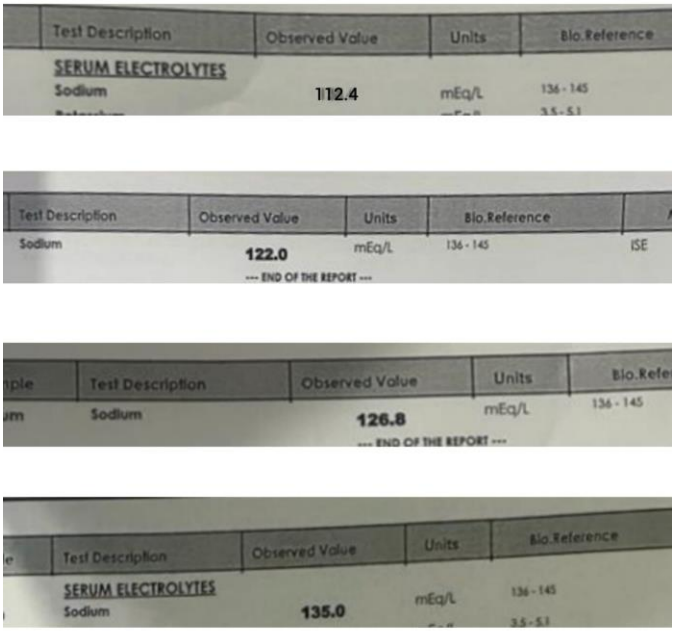

Figure 1: Serum Sodium Levels

Discussion

A common side effect of both traumatic and nontraumatic cerebral injury is hyponatremia, which is frequently linked to the syndrome of inappropriate antidiuretic hormone secretion (SIADH)(1).

However, cerebral salt wasting (CSW) syndrome, a distinct condition, can also be linked to it. The Syndrome of Inappropriate Antidiuretic Hormone (SIADH) secretion is defined by a combination of hyponatremia and hypo-osmolality arising from one of the following:

- Excess ADH release from the hypothalamus/pituitary. This can follow brain injury (traumatic, anoxic, haemorrhagic or inflammatory), stroke or neurological tumours. It is less frequently associated with other neurological disorders.
- Excess ADH production in ectopic sites. Inflammatory conditions or tumours in extracranial sites can be associated with abnormal ADH production. This is particularly likely with respiratory inflammatory disorders or lung tumours.

- Excess ADH production or renal sensitivity to ADH can be triggered by specific medications, especially opiates, NSAIDs, anticonvulsants, antidepressants, antipsychotics, hypoglycaemic agents and anticancer drugs (anti-neoplastic agents).

The syndrome of inappropriate antidiuretic hormone secr etion (SIADH) is frequently brought on by CNS pathology and is linked to a number of clinical conditions (2).Although it is acknowledged that SIADH is a typical early complication of TBI, it very seldom lasts or recurs. The morbidity and mortality of patients with traumatic brain injurycan be decreased with early diagnosis and tre atment(3),those with moderate and severe head injuries fr equently experience hyponatremia, although those with li ght head injuries hardly ever report it.ADH is improperly secreted in SIADH, a condition that is quite prevalent.It can happen in a variety of therapeutic contexts, such as h ead trauma, as was the case in this instance.Traumatic bra in damage can interfere with the ADH secretion's regular regulating processes, causingan excessive release of the h ormone(4).In the example at hand, the traumatic hemato ma probably caused ADH to leak, leading to the emergen ce of SIADH.Hyponatremia that follows can cause a vari ety of symptoms, such as wearines, confusion, nausea, se izures, and in extreme circumstances,even coma.In spite of the patient's considerable decline in serum salt levels i n this instance, the patient showed no symptoms.

Conclusion

We report a case of head trauma inducing asymptomatic hyponatremia. This case underscores the point that mild closed head injury without structural damage should be considered as a cause of SIADH in patients who subsequently develop hyponatremia. By early diagnosis and treating with hypertonic saline and fluid restriction hyponatremia can be resolved.

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