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A Case of Hyponatremia after Traumatic Brain Injury (TBI)

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Background

Sodium is the major extracellular cation and one of the most important osmotically active solutes.
Hyponatremia is defined as a serum sodium concentration of less than 135mmol/L and occurs in up to 15% of the general adult inpatient population. Hyponatremia may increase the risk of seizures, thereby brain edema, mass effect, and death. Hyponatremia is also associated with delirium and mild cognitive impairment, mobility and balance impairment and falls. After brain injury, hypotonic hyponatremia occurs most frequently because of the syndrome of inappropriate ADH secretion (SIADH) or the cerebral salt wasting syndrome (CSWS). In this report, we would like to share the experience of SIADH after TBI.

Case description

A 61-year-old hypertensive man was found lying unconscious. He was diagnosed with traumatic subarachnoid hemorrhage and intracranial hemorrhage in frontal lobe.
On the 74th day after the hemorrhage onset, the patient was referred to our department for rehabilitation. At that time his motor power was fair+ in the upper extremities and fair in the lower extremities. His sitting balance was poor and he could stand up with maximal assistance and gait with maximal assistance (FAC 1). Initial MMSE was 4 and initial K-MBI was 47. After comprehensive rehabilitation, His functional state was improved, then sitting balance was fair and he could stand up with minimal assistance and gait with moderate assistance.
After 16 days of admission, he showed delirious behavior and complained of nausea and headache. We studied brain CT and laboratory test. The brain CT findings showed no evidence of normal pressure hydrocephalus compared previous brain CT. In laboratory findings serum sodium level fell to 120mmol/L.
Serum osmolality was 264, urine osmolality was 507 and urine sodium was 132. He was clinically euvolemic, and the thyroid function test and rapid ACTH test were normal. So, the clinical impression was SIADH.
Because of symptomatic acute hyponatremia, hypertonic (3%) saline was injected with bolus. With the strict fluid restriction and the supplement of salt in every mealtime, his serum sodium level remained over 130mmo/L, and the delirium symptom and general condition was improved.
In the process of administering cerebrolysin for cognitive improvement, a total of 350cc of normal saline was added for 4 days, and serum sodium level was lower. When his serum sodium level was below 125mmol/L, his delirious behavior and cognitive function was worse. We administered 3% normal saline if his serum sodium level was 125 or lower, with the strict fluid restriction and the supplement of salt in every mealtime. Two weeks later, his serum sodium level remained 126nmol/L and his general function and delirium symptom were slightly improved.

Conclusions

Hyponatremia due to SIADH or CSWS is not rare complication in TBI patient. And the symptom of hyponatremia in TBI patient is various, such as delirium or poor general condition or lethargy. So, it is confused to other complication such as normal pressure hydrocephalus (NPH). Hyponatremia in TBI may increase mortality and related to poor outcome with delirium or longer hospital stay. We experienced a SIADH after TBI with a slow correction. Awareness of the causes of electrolyte disturbances can lead to the appropriate prevention of severe disturbances, particularly in TBI patient and it is very closely associated outcome of patient.

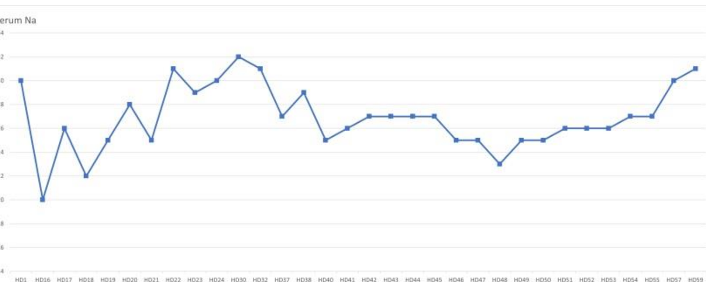

Fig1. serum Na(mmol/L) at 6am (HD: hospital day).

	HD1	HD16
serum Na(mmol/L)	130	120
serum Osm(mOsm/kg H2O)	287	264
urine Osm(mOsm/kg H2O)	611	507
urine Na(mmol/L)	132	132
	PSEUDO	TRUE

Fig2. The hyponatremia shown in HD1 was pseudohyponatreamia, and from HD16 it is true hyponatremia.

rapid ACTH test	HD21
ACTH(pg/ml)	15
cortisol(ug/dl)	7.3
cortisol(ug/dl)	19.1
cortisol(ug/dl)	23.3
TFT	HD21
TSH(uIU/ml)	0.9605
freeT4(ng/dl)	1.1
T3(ng/ml)	23.3

Fig3. Both the rapid ACTH test and the Thyroid function test (TFT) were normal.

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