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Successful management of a case of Cerebral Salt Wanting Syndrome (CSWS) related hyponatremia

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43 year old lady with a history of Rheumatoid Arthritis (On Sulfasalazine and Infliximab IV every 8/52), was brought in to our hospital following a collapse. She had had acute onset of headache six weeks prior to admission with nausea, vomiting and blurred vision but her symptoms had gradually resolved. Her GCS on arrival was 5. Subsequent CT scan brain showed acute right-sided subdural haematoma and subarachnoid haemorrhage and was indicative of coning. She was taken to theatre immediately for decompressive craniotomy and evacuation of her acute subdural haematoma. She was transferred to the ITU.

On day 4 post operatively, she was hyponatremic with sodium 127 mmol/l (base line Sodium was 144 mmol/l pre-operatively). Her sodium continued to fall progressively, reaching 118 mmol/l at day 6. Investigations showed TSH 1.1 mu/l, urine Osmolality 512 mosmol/kg, plasma Osmolality 241 mosmol/kg, urine sodium 167 mmol/l, urea 2.2 mmol/l, creatinine 39 umol/l, K 3.5 mmol/l. She was not dry clinically and her CVP was 5-6 cm H2O. It was difficult to differentiate between Cerebral Salt Wasting Syndrome (CSWS) and SIADH. She was cautiously treated with hypertonic saline and her hyponatremia corrected over a couple of days to134mmol/l

Discussion:

We report a case of hyponatremia secondary to assumed cerebral salt wanting syndrome which was successfully and wisely treated with hypertonic saline in the ITU. The cause of hyponatremia post brain injury is often difficult to ascertain. We recommend patients should be carefully clinically and biochemically assessed. If this is supportive of cerebral salt wasting syndrome then fluid replacement should be commenced and cautious use of hypertonic saline considered. Hypertonic saline at a rate between 50-150 ml/h as a bolus initially, and then review as per current standard of care, so as not to increase the sodium concentration by more than an average of 0.5 mmol/l/hour or an increase no greater than 10 mmol/l in the first 24 hours. This is to prevent the development of osmotic demyelination.

Table 1
Biochemical markers: comparison of SIADH and CSW

Biochemical marker	SIADH	
Extracellular fluid volume	Normal to high	Low
Urinary sodium level	>40 mEq/L	>40 mEq/L
Serum uric acid level	Low	Low
Initial fractional excretion of urate	High	High
Fractional excretion of urate after correction	Normal	High
Urinary osmolality	High	High
Serum osmolality	Low	Low
Blood urea nitrogen/creatinine level	Low to normal	high
Serum potassium level	Normal	Normal to high
Central venous pressure	Normal to high	Low
Pulmonary capillary wedge pressure	Normal to high	Low
Brain natriuretic peptide level	Normal	High
Treatment	Water restriction	Fluids and/or m

CSVV = cerebral sail wasting; SIADH = syndrome of inappropriate antidiuretic hormone.

Table 2
Causes of cerebral salt wasting

- Subarachnoid haemorrhage
- Head trauma
- Intracranial neoplasm
- Metastatic neoplasm
- Infections or carcinomatous
- Meningitis
- Encephalitis
- Central nervous system surgery