INTRODUCTION:
Hyponatremia is a common electrolyte disorder in the setting of CNS disease and is often attributed to SIADH. This syndrome is characterised by hyponatremia with an inappropriately concentrated urine, increased urine sodium concentration, normal or slightly increased intravascular volume. By contrast, there are patients with intracranial disease who develop hyponatremia with similar characteristics but differ in that there is clinical evidence of a contracted ECF volume. This form of hyponatremia is due to excessive renal sodium excretion resulting from a centrally mediated process, Cerebral salt wasting CSW. Distinguishing between these two disorders is of crucial importance because therapy indicated for one disorder if used in the other can result in worsening of the clinical condition of the patient.

HISTORY AND CLINICAL PRESENTATION:
A 25 yr old apparently healthy female presented with c/o Seizure activity – tonic posturing of the left upper limb that later involved all 4 limbs, with associated H/O vomiting & headache for past 1 week. On examination she was drowsy, arousable, confused, afebrile, severely dehydrated. She had tachycardia otherwise vitals was stable. She had a GCS – E3V4M5, Systemic examination was unremarkable. She had no Neck stiffness, Fundus examination was normal.
Her seizure was controlled with inj Lorazepam 4mg IV. Investigations revealed that she had normal Blood sugar, CBC, RFT, CHEST X RAY, USG Abdomen but her serum electrolytes showed serum sodium 121 mEq/L serum potassium 3.7 mEq/L LFT normal except for mild transaminitis. CT BRAIN – Normal.
On day 2 patient continued to be drowsy, disoriented, severely dehydrated BP fell to 90/60 from 110/70 the previous day, we repeated her serum electrolytes, her hyponatremia and dehydration persisted in spite of fluid administration. On day 4 patient developed signs of meningeal irritation – kernigs sign was present. CSF analysis showed Protein - 58 Sugar - 29, 4 to 5 cells present, NO organisms in Gram Stain, Negative for AFB, NO growth when cultured. ADA - 8.5 U/L – Negative. We proceeded with MRI BRAIN – T1 hypo / T2 hyper intense, FLAIR suppressible lesion noted in right inferior frontal region, FLAIR hyperintensity in right parietal region. MRI BRAIN CONTRAST – Leptomeningeal and pachymeningeal enhancement, ring enhancing lesion noted in right inferior frontal region – TB meningitis + Tuberculoma.

She was started on CAT 1 ATT and steroids.

But her hyponatremia and dehydration persisted. What is the cause of her hyponatremia, IS IT SIADH OR CEREBRAL SALT WASTING ???
DIAGNOSTIC CRITERIA FOR BOTH SIADH & CSW

- Low plasma Sodium concentration (in our pt) [ <130mmol/l ] 124
- Low plasma Osmolality [ <270mmol/kg ] 267
- Urine Osmolality not minimally low [ >150mmol/kg ] 161
- Urine Sodium concentration not minimally low [ >30mmol/l ] 90
- APPROPRIATE clinical context

A trail of vaptans worsened the hyponatremia and dehydration in our patient

VAPTANS

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VAPTANS stopped 3% sodium chloride

URIC acid levels can help in differentiating between SIADH and CSW

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SrUricacid -1.7

SrUricacid -1.3
DISCUSSION:

Is it CSW..?? That our patient has ..... 
- Hyponatremia
- ↓ Sr osmolality
- ↑ urine osmolality
- ↑ urine Na+
- Hypotension
- Dehydration
- Persistent ↓ uric acid
- Improved with Fluid therapy

Is it SIADH..?? That our patient has ..... 
- Hyponatremia
- ↓ Sr osmolality
- ↑ urine osmolality
- ↑ urine Na+
- Haematocrit normal
- Sr albumin normal

YES
- Hyponatremia
- ↓ Sr osmolality
- ↑ urine osmolality
- ↑ urine Na+
- Haematocrit normal
- Sr albumin normal
- Worsened with VAPTANS
- Sr Potassium
- BUN-Cr ratio

NO
- Hypotension
- Dehydration
- Persistent ↓ uric acid
- Improved with Fluid therapy
- Haematocrit not elevated
- Sr albumin not elevated
- Sr potassium
- BUN-Cr ratio

EABV – Effective Arterial Blood Volume
ECF volume is the primary means of distinguishing CSW from SIADH. Severe dehydration, fall in BP, response to fluid therapy, worsening with fluid restriction and vaptans all these facts favour CSW over SIADH in our patient.

A proposed mechanism of CSW:

**Central Nervous System Disease**

- ↓ Sympathetic Nervous System Outflow
- ↑ BNP, ANP, ? other Natriuretic Factors

- ↓ Proximal Urate Reabsorption
- ↓ Proximal Na Reabsorption
- ↑ Distal Na Delivery
- ↑ Natriuresis Without K Wasting

- ↓ EABV
- ↑ AVP
- ↑ Urinary Concentration

**Hyponatremia**

IMCD – Inner Medullary Collecting Duct, EABV – Effective Arterial Blood Volume, AVP – Arginine Vasopressin, BNP – Brain Natriuretic Peptide, ANP – Atrial Natriuretic Peptide
CONCLUSION:

This review emphasises the need for CSW to be included in the differential diagnosis of hyponatremia in a patient with central nervous system disease.

REFERENCES:

1. Cerebral salt wasting following tuberculous meningoencephalitis in an infant Syed Ahmed Zaki, Vijay Lad, Preeti Shanbag Department of Pediatrics, Lokmany Tilak Municipal General Hospital and Medical College, Sion, Mumbai, India
6. Continuing Nephrological Education (CNE) Nephrol Dial Transplant (2000) 15: 262-268 Hyponatremia in a neurosurgical patient : SIADH Vs CSW Biff F. PalmerDepartment of Internal Medicine , Division of Nephrology , University of Texas Southwestern Medical Center , Dallas , Texas , USA