


Cerebral Salt Wasting Syndrome Following Neurosurgical Intervention in Tuberculous Meningitis

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ABSTRACT

Cerebral salt wasting is characterized by inappropriate natriuresis and volume contraction in the presence of cerebral pathology. Diagnosis can be difficult and therapy is challenging. We report two children with tuberculous meningitis and hydrocephalus who developed cerebral salt wasting following neurosurgical intervention. The first patient was managed with rigorous salt and water replacement whereas the second patient required the addition of fludrocortisone for control of salt-wasting.

Key words: Cerebral salt wasting; Hydrocephalus; Tuberculous meningitis.

INTRODUCTION

Hyponatremia frequently develops in patients with a variety of acute central nervous system diseases. Two pathophysiological mechanisms have been suggested to cause non-iatrogenic hyponatremia: cerebral salt wasting syndrome (CSWS) and the syndrome of inappropriate secretion of antidiuretic hormone (SIADH)(1-4).

Considering the divergent nature of treatment and the potential adverse effects of improper fluid therapy, it is important for the treating clinician to be able to differentiate between SIADH and CSWS, since therapy of SIADH is fluid restriction whereas in CSWS, rigorous salt and volume replacement are necessary. We describe two patients with tuberculous meningitis and hydrocephalus who developed cerebral salt wasting syndrome following neurosurgical intervention.

CASE REPORT

Case 1: A 4-year-old boy was transferred to the Pediatric Intensive Care Unit (PICU) following...
ventriculoperitoneal shunt surgery for hydro-
cephalus. He was diagnosed elsewhere to have
tuberculous meningitis. Antituberculous therapy and
steroids were started following which there was
transient improvement. Computerized tomography
(CT) scan of brain revealed mild hydrocephalus.
Two weeks later, the child again developed altered
sensorium. Since repeat CT scan brain showed
moderate hydrocephalus, the patient was transferred
to our hospital for neurosurgical intervention.
Following the shunt placement, there was no change
in sensorium ($E_1 M_2 V_1$). Serum sodium at this time
was 128 mmol/L and decreased to 120 mmol/L on
day 3 despite discontinuing mannitol. The child
appeared mildly dehydrated, systolic blood pressure
dropped to 70 mm of Hg and the urine output was 10
mL/kg/hour. Fluid correction was given volume-to-
volume with normal saline. Thereafter the child
stabilized with a gradual drop in the urine flow rate
and urinary sodium. The sensorium improved
($E_3 M_4 V_3$), blood pressure was stable and the child
transferred to the ward for further management.

Case 2: A 6-year-old girl was transferred from a
private hospital where she was receiving intravenous
antibiotics for a presumptive diagnosis of pyogenic
meningitis. The patient was transferred in view of
repeated convulsions and deteriorating sensorium.
At admission, the patient was comatose with a GCS
of 3 ($E_1 M_1 V_1$). An urgent CT scan brain showed
basal exudates with moderate hydrocephalus. The
patient was intubated and ventilated. An urgent
neurosurgery reference was sought and emergency
ventricular tap done. Antituberculous therapy with
steroids was started. The sensorium however,
remained the same. Serum sodium was 119 mmol/L
and blood pressure 90/60 mm of Hg. Mannitol was
discontinued. Despite volume-to-volume correction
with normal saline, the serum sodium dropped to 109
mmol/L. Urinary volume was 6 mL/kg/hour and
urinary sodium 146 mmol/L. Sodium correction was
done with 3% saline and fludrocortisone started in
the dose of 0.1 mg/day which was increased to 0.5
mg/day with monitoring of the serum potassium.
Over the next 5 days, there was a gradual increase in
the serum sodium to 125 mmol/L, with improvement
in the sensorium to a GCS of 10 ($E_3 M_4 V_3$). The
patient could be weaned off the ventilator.

Haloperidol (Serenace) and benzhexol hydro-
chloride (Pacitane) were started for involuntary
movements and patient was transferred to the ward.
Though serum sodium had normalized, fludro-
cortisone was continued because of high urinary
volume. Sensorium improved to normal and
intravenous fluids omitted. The urinary volume
gradually decreased to <2mL/kg/hour on the 28th
hospital day when fludrocortisone was stopped and
the patient discharged.

The serial values of serum sodium, urinary
volume and urine sodium of both patients are shown
in Table I.

**DISCUSSION**

Cerebral salt wasting syndrome has been described
with a variety of cerebral insults including
tuberculous meningitis and also following neuro-
surgical interventions(3,4). In our patient it is not
clear whether the CSWS occurred due to tuberculous
meningitis *per se* or due to the neurosurgical
intervention which was done on the day of
admission.

Hyponatremia, an increase in urinary sodium
excretion and high urinary osmolality are common to
both CSWS and SIADH. Physical examination and
laboratory results can assist a clinician in
differentiating between the two conditions, and thus
determine proper treatment. Evaluation of volume
status is crucial. Patients in the intensive care setting
may be assessed by measurement of their central
venous pressure (CVP). A high urine output further
supports the diagnosis of a salt-losing state rather
than SIADH(5,6). In our patients since hypovolemia
was obvious on clinical examination, CVP lines
were not inserted.

Blood urea nitrogen (BUN) increases in patients
with volume contraction as occurs with CSWS
whereas in patients with SIADH with a volume-
expanded state, the BUN is usually on the lower side.
Patient 1 had an elevated BUN, though this may not
always be observed(7).

Definitive diagnosis can be made by
documenting elevated levels of the natriuretic
peptides atrial natriuretic peptide (ANP) and brain
natriuretic peptide (BNP) although this may not be a universal finding(5,8). These could not be done in our patients due to lack of facilities.

Appropriate management of CSWS is fluid replacement with saline with the addition of the mineralocorticoid-fludrocortisone if necessary (2,5,6,9). Fludrocortisone acts directly on the renal tubule to reduce sodium excretion and significantly decrease the negative sodium balance(9). CSWS tends to be a transient phenomenon usually resolving within 3-4 weeks(10).

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REFERENCES


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