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Cerebral Salt Wasting Syndrome Following Neurosurgical Intervention in Tuberculous Meningitis

L NAGOTKAR
P SHANBAG
N DASARWAR

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

From the Pediatric Intensive Care Unit, Lokmanya Tilak Municipal Medical College & General Hospital, Sion, Mumbai 400 022, India.

Correspondence to: Dr Leena Nagotkar, 9458/242, Kannamwarnagar 2, Vikhroli(E), Mumbai 400 083, India. E-mail: nagotkar.leena@gmail.com.

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ventriculoperitoneal shunt surgery for hydrocephalus. 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 ($E_1M_2V_1$). 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 ($E_3M_4V_3$), 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 comatosed with a GCS of 3 ($E_1M_1V_1$). 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 ($E_3M_4V_3$). The patient could be weaned off the ventilator. Haloperidol (Serenace) and benzhexol hydrochloride (Pacitane) were started for involuntary movements and patient was transferred to the ward. Though serum sodium had normalized, fludrocortisone 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 neurosurgical 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

TABLE I SERIAL VALUES OF SERUM SODIUM, URINARY SODIUM AND URINARY VOLUME

Hospital day	Serum sodium (mmol/L)	Urinary sodium (mmol/L)	Systolic BP (mm of Hg)	Urine volume mL/kg/hr	Blood urea nitrogen mg/dL
Case 1 Preoperative	132		96		12
1	128		95	4	
2	125	34	95	8	
3	120	84	70	10	45
4	123	78	80	7	
5	128	30	90	3	
6	130	15	95	2	18
7	132	10	95	<2	
Case2					
1	119		90		30
2	110			6	
3	116			6	
4	109	146	90	7	28
Fludrocortisone added					
5	114	104	90	10	25
6	122		112	8	
7	123	88		8	
8	122			8	
10	125	41	110	7	
12	135			4	
16	133	18		5	
20	140			4	
24	142			2	
28	141	10		<2	

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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