Brief Communications

Cerebral salt wasting syndrome in craniopharyngioma

INTRODUCTION

Cerebral salt wasting syndrome (CSWS), which was first described by Peters *et al*. in 1950, is defined by development of natriuresis, hyponatremia and subsequent hypovolemic dehydration in patients with intracranial disorders.[1] Classical causes of the syndrome are traumatic brain injury, stroke, subarachnoid haemorrhage and brain tumour. This syndrome has been reported following pituitary surgery[2] and also in a case of meningoencephalitis.[3] Association of CSWS with craniopharyngioma is a rare entity, but not entirely a new finding.[4] Clinical presentation of CSWS is similar to syndrome of inappropriate secretion of anti-diuretic hormone (SIADH), but differential diagnosis is essential for prescribing appropriate therapy. We report, an intriguing case of CSWS that occurred in a case of craniopharyngioma and underwent a prolonged course.

CASE REPORT

A 45-year-old, 60 kg, male complaining of moderate degree headache for 15 days attended our out-patient department. Magnetic resonance imaging of brain revealed that he had sellar-suprasellar space occupying lesion (24 mm × 20 mm × 26 mm), predominantly solid in nature, without hydrocephalus, suggesting craniopharyngioma [Figure 1]. While waiting for elective surgical removal of that lesion, he became drowsy and admitted in our hospital. He was a patient of known hypothyroidism and was on oral thyroxin (0.1 mg daily). There was no history of convulsion or localising neurological deficit and patient was non-diabetic and non-hypertensive. Following admission, patient was found dehydrated (decreased in skin turgor), his pulse rate was 94/min; blood pressure was 96/60 mm Hg and Glasgow coma scale was 13/15. On investigation, haemoglobin was 10.8 g% and among other findings, total leucocyte count was 6600/cumm, blood glucose was 109 mg/dl, blood urea and serum creatinine were 58 mg/dl and 1.5 mg/dl respectively. Serum electrolyte studies revealed sodium 117 mmol/L and potassium 4 mmol/L. Chest X-ray and electrocardiogram detected no abnormality. Thyroid hormones and serum cortisol were within the normal limit. A central venous line through subclavian route was done and central venous pressure (CVP) was noted 3-4 mm of Hg. As patient was hyponatremic and dehydrated, we started normal saline (NS) infusion at the rate of 150 ml/h (change in serum Na^+ equals to the replacement fluid Na^+ - serum Na^+ divided by total body water + 1 and we wanted to correct serum by 0.5 mmol/L with a total increase of serum Na^+ 8 mmol/L/24 h)[5] to make the patient euvolemic. His urine output in 24 h was also recorded and found to be 3.5 L. At the same time, urine and blood samples were sent for further studies to detect the cause of hyponatremia. Investigations showed serum osmolality 264 mOsm/kg H_2O, urinary osmolality 430 mOsm/kg H_2O and urinary spot sodium 132 mmol/L and a diagnosis of CSWS was made. Infusion of NS was continued along with every 2 h monitoring of serum sodium. Even after 24 h of NS infusion, as serum Na^+ level got slightly increased to 119 mmol/L, we preferred to infuse hypertonic saline (3% sodium chloride) at a rate of 45 ml/h. Patient gradually became conscious and oriented as serum sodium level increased to 130 mmol/L over next 48 h. Following improvement, patient was put up for definitive surgery. During the surgery, conventional neuroanaesthetic technique was used and the patient was reversed well. Intraoperative and immediate post-operative period remained uneventful. On the 4th post-operative day, patient again became drowsy. Although post-operative computed tomography scan revealed nothing abnormal, serum sodium was found to be 122 mmol/L and urine spot sodium 92 mmol/L. Sodium replacements was started immediately with NS infusion. Waxing and waning of the state of hyponatremia persisted for next 27 days and managed accordingly with sodium replacement through nasogastric tube, NS and occasional 3% saline infusion. However, with this treatment his high urinary sodium gradually became normal and serum sodium

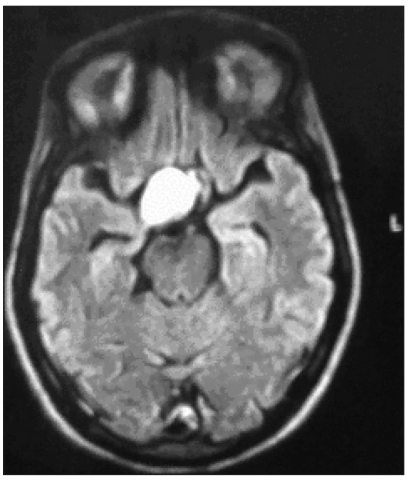

Figure 1: Magnetic resonance imaging brain showing sellar-suprasellar space occupying lesion (24 mm × 20 mm × 26 mm)

gradually reached normal level and patient became clinically stable. Patient was discharged, after 2 weeks of observation as he had been maintaining normal serum sodium level throughout that period.

DISCUSSION

Hyponatremia due to inappropriate SIADH is commonly seen with a number of intracranial diseases, especially head injuries, stroke and subarachnoid haemorrhage. CSWS is often an unrecognised cause of hyponatremia that may occur under similar circumstances.[6-8] It is very crucial to differentiate CSWS from SIADH [Table 1]. The main clinical difference between these two pathologic conditions is total fluid status of patients. In CSWS, there is relative or overt hypovolemia whereas SIADH is associated with normal or hypervolemia. Unfortunately, no single physical finding can accurately and reproducibly measure effective circulating volume. Signs of hypovolemia are usually hypotension, tachycardia, increased capillary filling time, decreased skin turgor, dry mucous membrane. This patient was dehydrated and CVP was found to be <5 mmHg. Other useful point in differentiating CSWS from SIADH is spot urine sodium, which is usually >100 mmol/L in the former.[9] The serum electrolyte imbalance observed in this patient was like that of SIADH. However, signs of volume depletion with urinary spot sodium 132 mmol/l distinguished the case from SIADH.

Treatment of CSWS involves hydration with NS (0.9% sodium chloride) and salt repletion. If hyponatremia becomes very severe, then 3% sodium-chloride may be used. Fludrocortisone acetate 0.2 mg intravenous or oral has been used for treatment of CSWS.[10]

Table 1: Distinguishing features between CSWS and SIADH

Parameter	SIADH	CSWS
Clinical sign of dehydration	Absent	Present
CVP	N/↑	↓
Urine Na^+	↑	↑↑
Serum uric acid	↓	↑↑
BUN	↓	N/↑
BUN/creatinine	↓	N/↑
Haematocrit	N/↓	↑
Serum K^+	N/↓	N/↑
Serum albumin	N	↑
Management	Fluid restriction	Salt and fluid supplementation

CVP – Central venous pressure; BUN – Blood urea nitrogen; N – Normal; ↑ – Increased; ↓ – Decreased; SIADH – Syndrome of inappropriate secretion of anti-diuretic hormone; CSWS – Cerebral salt wasting syndrome

In our case, patient recovered well without use of fludrocortisone acetate or any dreaded complication.

CONCLUSION

Differential diagnosis for SIADH and CSWS is important regarding the selecting mode of treatment as they are completely opposite for each other. Moreover, management of a patient with CSWS for a prolonged period is a challenging job as rapid correction of hyponatremia may lead to central pontine myelinosis with devastating neurological outcome.

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