Case Report

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Subarachnoid Hemorrhage with Cerebral Salt Wasting Leading to Cerebral Ischemia

Sudhir Suggala[1], Rahul Gupta[2], K V V S N Murthy[3], Manojkumar Bhutte[4], Krishna Chaitanya Joshi[5]

[1]Consultant, Nagarjuna Hospital, Kanuru, Vijayawada, Andhra Pradesh, India, [2]Senior Consultant, Department of Neurosurgery, Fortis Hospital, Noida, Uttar Pradesh, India, [3]In Charge Professor, Department of Neurosurgery, Government General Hospital, Vijayawada, Andhra Pradesh, India, [4]Assistant Professor, Department of Neurosurgery, Dharwad Institute of Mental Health and Neuro Sciences, Dharwad, Karnataka, India, [5]Assistant Professor, Department of Neurosurgery, MS Ramaiah Medical College, Bangalore, Karnataka, India

Abstract

Cerebral vasospasm is a well-known complication after aneurysmal subarachnoid hemorrhage. This along with natriuresis in the form of cerebral salt wasting (CSW) leads to intravascular volume depletion, potentiating cerebral edema and leading to cerebral infarction and focal neurological deficit(s). CSW and syndrome of inappropriate antidiuretic hormone (SIADH) have been reported to occur in cerebral pathologies. Hyponatremia resulting from CSW may be difficult to distinguish from SIADH. However, the distinction between them is imperative because treatment is just the opposite. In this manuscript, we report a case of CSW culminating in cerebral vasospasm, cerebral edema, and ischemia that manifested as aphasia during the post-operative period.

Key words: Cerebral salt wasting, Cerebral vasospasm, Subarachnoid hemorrhage, Syndrome of inappropriate antidiuretic hormone secretion

INTRODUCTION

Cerebral salt wasting (CSW) syndrome is defined by the development of extracellular volume depletion due to a renal sodium transport abnormality in patients with an intracranial disease with normal adrenal and thyroid function. It was first described by Peters *et al.* in 1950.[1] Various cerebral pathologies leading to this syndrome include head injury, brain tumor, intracranial surgery, stroke, tubercular meningitis, etc.[2] It is an important under-recognized cause of hyponatremia in patients with subarachnoid hemorrhage (SAH) and aneurysm clipping. The clinician can misinterpret it as a syndrome of inappropriate secretion of antidiuretic hormone (SIADH) which has a totally different treatment protocol. We report a case of CSW syndrome presenting as delayed vasospasm and aphasia due to cerebral ischemia.

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CASE REPORT

A 53-year-old male came with a history of sudden onset of severe headache, vomiting, and transient loss of consciousness. On examination, he was conscious, alert and oriented and had neck stiffness. A cerebral computed tomogram (CT) revealed SAH (Fischer Grade 3) with blood in anterior interhemispheric fissure and basal cisterns (Figure 1a). CT angiography revealed a medium sized anterior communicating artery aneurysm directed anteriorly, inferiorly, and to the right side (Figure 1b). He underwent a left pterional craniotomy and clipping of aneurysm on 3rd day of ictus. There were no intraoperative complications, and the initial post-operative course was uneventful. Since the patient was accepting normal oral feeds and CT scan (on 3rd postoperative day) was normal (Figure 2a), he was shifted toward from the intensive care unit.

On the 7th postoperative morning, he became drowsy, aphasic, and developed a right hemiparesis (Grade 4/5). At this stage, the blood pressure was 100/60 mm Hg, the central venous pressure was 1-cm of water (5-10 cm H_2O), and plasma sodium was 118 mmol/L. The last 24 h, urinary output was 1200 ml with a measured

Corresponding Author: Dr. Sudhir Suggala, Nagarjuna Hospital, Kanuru, Vijayawada - 520 010, Andhra Pradesh, India. Phone: +91-9949416094. E-mail: sudhirsuggala@gmail.com

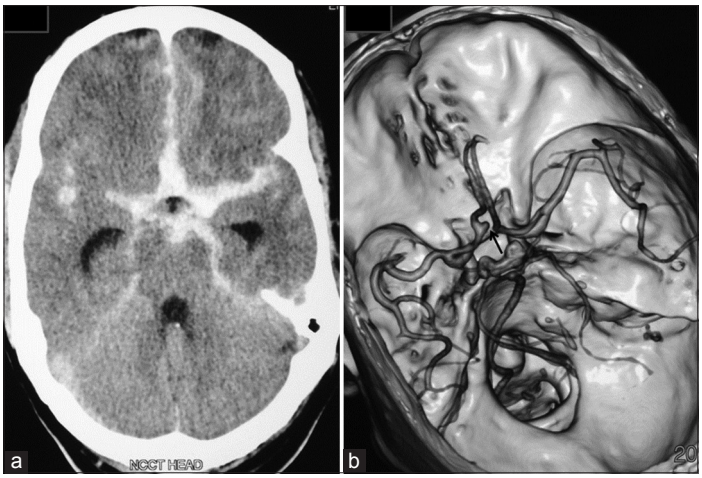

Figure 1: (a) Computed tomogram (CT) head showing subarachnoid hemorrhage in anterior interhemispheric fissure and basal cisterns, (b) CT angiography showing the aneurysm

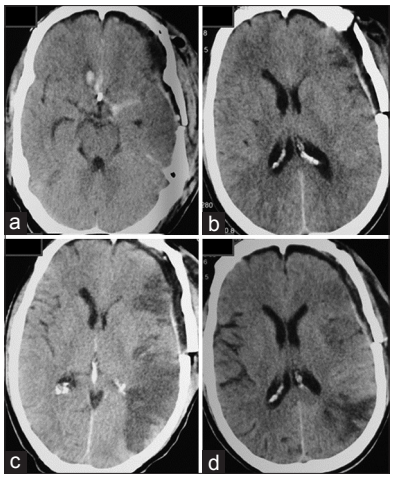

Figure 2: Serial computed tomogram head: (a) postoperative day 3, (b) postoperative day 7, (c) postoperative day 12, (d) postoperative day 18

fluid intake of 1800 ml during the same period. He was shifted back to the intensive care unit and a further workup revealed a serum osmolality of 278 mosm/kg (normal 285-295 mosm/kg), blood urea nitrogen (BUN) of 54 (normal 22-46 mg/dl), creatinine 0.8 (normal 0.6-1.2 mg/dl), serum uric acid 2.3 mg/dl (normal 2.0-7.0 mg/dl), urinary sodium 108 mmol/L (normal value in dehydration <20 mmol/L), urine osmolality 308 mosm/kg (normal <100 mosm/kg), and urine specific gravity of 1.030 (normal <1.003 or less). The patient appeared hypovolemic and the entire clinical picture was suggestive of CSW. CT scan of the brain (7th postoperative day) was normal (Figure 2b).

The sodium deficit was calculated to be 714 meq. Half correction of sodium was done at the rate of 1-mmol/L/h in next 24 h. Over the next 24 h, his serum sodium levels improved to 128 mmol/L and he became less drowsy. Volume depletion was corrected with dextran 40 and normal saline to attain a central venous pressure between 8 and 10. Urine output between 80 and 150 ml/h was achieved and urinary sodium levels reduced to 52 mmol/L. The patient continued to improve neurologically, and serum sodium level returned to normal at (136 mmol/L) in next 3 days. He started communicating and his hemiparesis resolved. Another cerebral CT, on postoperative day 12, revealed patchy infarcts in left middle cerebral artery territory (Figure 2c).

The patient gradually improved in a week and became more alert. The biochemical investigations were normal, and he was shifted to the ward. Another CT scan (18th postoperative day) (Figure 2d) revealed the resolution of infarcts. His aphasia improved gradually over next 3 months.

DISCUSSION

CSW is a process of extracellular volume depletion and is characterized by extracellular volume depletion, low central venous pressure, increased urinary sodium (>40 meq/L), increased urinary osmolality, increased BUN/creatinine levels, and low to high serum osmolality. Two postulated mechanisms for CSW are the excess secretion of natriuretic peptides (atrial natriuretic peptide [ANP], brain natriuretic peptide [BNP], C-type ouabain-like peptide), and the loss of sympathetic stimulation to the kidney.[3] After SAH, the reason for volume depletion and hyponatremia is natriuresis, which can be due to CSW, SIADH, hypothyroidism, adrenal insufficiency, osmotic dieresis, use of diuretics, and renal failure. Cerebral vasospasm remains one of the major threats to patients with aneurysmal SAH and natriuresis make them prone for delayed cerebral infarction.[4]

CSW and SIADH have been reported to occur in cerebral pathologies, but the distinction between them is imperative because the treatment is just the opposite. A retrospective review of data for 316 patients who presented with SAH and hyponatremia found that the diagnosis was SIADH in 69% and CSW in 6.5%.[5] SIADH is characterized by normal to high extracellular fluid volume, high urinary sodium levels (>40 mEg/L), high urine osmolality, low serum osmolality, low to normal BUN/creatinine ratio, normal to high CVP, and normal BNP levels.[6] If CSW syndrome is misdiagnosed as SIADH and treated with fluid restriction, there is a risk of hemoconcentration and hypotension leading to decreased cerebral perfusion and vasospasm. Furthermore, if not corrected early, it may induce cerebral edema and increased intracranial pressure due to low osmolality.

Our patient had clinical features suggestive of CSW like hyponatremia associated with hypovolemia, elevated urea, increased urinary sodium, and increased urinary osmolality.[6] He developed neurologic deficit simultaneously with hyponatremia and natriuresis. He rapidly responded to volume replacement with colloids and sodium rich fluids. Whether the cerebral ischemia led to the development of CSW or CSW preceded the onset of the infarct is a matter of debate. However, our prompt exclusion of SIADH as the cause of hyponatremia prevented the patient from deterioration, and the infarcts were patchy and resolved quickly. Fluid restriction, which is the treatment for SIADH would have been disastrous.

The appropriate treatment for CSW syndrome includes maintaining the body fluid volume and electrolyte concentration. Corticosteroids (like fludrocortisone) are also recommended for the treatment of CSW syndrome but were not used in this case. Corticosteroids act on the distal tubule of the kidney, thereby directly increasing the sodium absorption. However, they should be used judiciously as there is a risk of fatal side effects such as hypertension, hypocalcemia, and pulmonary edema.

The patient had normal urine output at the onset of CSW, which was slightly awkward. Serum levels of ANP, BNP were not done, in this case, which could have further confirmed the diagnosis. Fractional excretion of uric acid, which improves after hyponatremia, correction in SIADH unlike CSW could have been done to substantiate the diagnosis.

CONCLUSION

We like to convey that: (1) Hyponatremia might herald or coincide with the onset of cerebral infarction after surgery for aneurysmal SAH and can manifest even a week later, and (2) CSW is an important cause of hyponatremia, which has to be differentiated from SIADH.

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