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CASE REPORT / OLGU SUNUMU

Hyponatremia in a child with tuberculous meningitis in PICU: Cerebral salt wasting syndrome

Çocuk yoğun bakım ünitesinde tüberküloz menenjitli çocukta hiponatremi: Serebral tuz kaybı sendromu

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ABSTRACT

Cerebral salt wasting syndrome (CSW) has been reported in cases with subarachnoid haemorrhage, infections, head injury, brain tumours, trans-sphenoidal pituitary surgery, and neurosurgery. It is characterized by extracellular fluid depletion and hyponatraemia caused by progressive natriuresis with concomitant diuresis.

The relationship between tuberculous menengitis and CSW in children has been desciribed rarely. We describe a case of CSW in an eight years old child with tuberculous meningitis in Pediatric Intensive Care Unit (PICU) whose main biochemical findings were low serum sodium, excessive urine sodium loss and clinical evidence of a contracted extracellular fluid volume.

It is essential that in any child with hyponatremia and meningitis in PICU, an evaluation to be undertaken to differentiate between syndrome of inappropriate antidiuretic hormone secretion (SIADH) and CSW. A wrong diagnosis might lead to inappropriate fluid restriction and worsen the hypovolemia in children with CSW. Supplements of sodium chloride and mineralocorticoids may be useful in managing children with CSW. PICU professionals must be aware of the clinical and laboratory features that distinguish CSW from SIADH.

Key Words: Cerebral salt wasting syndrome, hyponatremia, tuberculous meningitis, children, differential diagnosis

ÖZET

Serebral tuz kaybı sendromu (STKS) subarak-noid kanama, enfeksiyonlar, kafa travması, beyin tümörleri, trans-sfenoidal hipofiz cerrahisi, ve beyin ameliyatı yapılan hastalarda bildirilmiştir. Bu sendrom diürezle birlikte aşırı sodyum atılımı nedeniyle ortaya çıkan hücre dışı sıvı azalması ve hiponatremi ile karakterizedir. Tüberküloz menenjit STKS birlikteliği nadiren rapor edilmiştir.

Bu çalışmada Çocuk Yoğun Bakım Ünitesi'nde (ÇYBÜ) izlenen 8 yaşındaki tüberküloz menenjit ve STKS gelişen bir çocuk hasta sunulmuştur. Hastanın ana biyokimyasal bulguları düşük serum sodyumu, idrarla aşırı miktarda sodyum kaybı idi ve hastada azalmış hücre dışı sıvıya ait klinik bulgular mevcuttu.

Çocuk Yoğun Bakım Ünitesinde menenjitle izlenen bir hastada hiponatremi geliştiği zaman uygunsuz anti-diüretik hormon salınımı sendromu (SIADH) ile STKS ayrımını yapabilmek çok önemlidir. Yanlış bir tanı gereksiz yere sıvı kısıtlaması ve STKS'li çocukta hipovoleminin kötüleşmesine yol açabilir. Sodyum klörür verilmesi ve tedaviye mineralokortikoid eklenmesi STKS'li çocukların yönetiminde yararlı olabilir. ÇYBÜ çalışanları STKS'yi SIADH'dan ayıracak klinik ve laboratuar özellikleri bilmek zorundadır.

Anahtar Kelimeler: Serebral tuz kaybı sendromu, tüberküloz menenjit, çocuklar, ayırıcı tanı

INTRODUCTION

Cerebral salt wasting syndrome (CSW) has been reported in patients with subarachnoid haemorrhage, infections, head injury, brain tumours, trans-sphenoidal pituitary surgery, and neuro-surgery. It is characterized by extracellular fluid depletion and hyponatraemia caused by progressive natriuresis with concomitant diuresis. On the basis of the combination of elevated plasma atrial natriuretic

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hormone levels and decreased or normal plasma aldosterone and vasopressin, it has been suggested that cerebral salt wasting is due to inappropriate secretion of atrial natriuretic hormone. The relationship between tuberculous menengitis and CSW in children in PICU has been desciribed rarely. We describe a case of CSW in an eight years old child with tuberculous meningitis managed in Pediatric Intensive Care Unit (PICU).

Case Report

An 8 years-old boy presented with a history of cough episodes, vomiting and altered consciousness for 10 days prior to application of Pediatric Emergency Department. There was no history of fever or convulsions. On examination, he was found as marasmic and drowsy. No signs of meningeal irritation, raised intracranial pressure or focal neurological deficit were observed.

Laboratory investigations showed his hemoglobin as 14.3 g/dl, total leukocyte count 9600/mm3, 58% neutrophils and 42% lymphocytes and erythrocyte sedimentation rate (ESR) 20 mm/h. The blood levels of sodium was 131 mEq/L, potassium 4.8 mEq/L, urea 23 mg/dl, creatinine 0.5 mg/dl and glucose was 100 mg/dl. The blood pH was 7.44, pCO2: 38 mmHg, pO2: 92 mmHg and bicarbonate 13.5 mEq/L. The Mantoux test was non-reactive. Cerebrospinal fluid (CSF) examination revealed a pleocytosis of 350/cell/mm3, which were 90% lymphocyte, protein 79 mg/dl, glucose 15 mg/dl and Cloride 97 mg/dl. CSF was cultured on Lowenstein-Jensen and Middlebrook solid media and in BACTEC radiometric broth. It was stained with Ziehl-Neelsen stain, that were examined for acid fast bacilli. Mycobacterium tuberculosis was not shown by neither cultures nor staines. Chest X-ray did not show evidence of pulmonary tuberculosis, but enlarged mediastinal nodes were detected by computerized tomography (CT). A CT scan of the cerebrum showed hydrocephaly and granulomas.

Dehydration and hypotansion were described at the second day of hospitalization, while blood sodium concentration was 114 mEq/l, then he was transferred to PICU. Although intravenous fluids were given for correction of dehydration and hyponatremia, the child still remained in a dehydrated state after 24 hours of treatment and the serum sodium level dropped down to 112 mEq/L. The patient's urine output was persistently more than 6 ml/kg/hour and revealed massive natriuresis with sodium excretion ranging from 35 to 140 mmol/l. Urinalysis and ultrasonogram of the kidneys were found to be within normal limits. Antituberculous treatment was started in view of the medical history, and laboratory and CT findings. The diagnosis of CSW was made in the view of hyponatremia in association with polyuria and natriuresis, and the absence of thyroid, adrenal, renal liver or cardiac dysfunction. The blood level of antidiuretic hormone was 11 pg/ml (normal range, 1-14 pg/ml).

The patient was managed with intravenous fluids with 3% NaCl. The serum sodium gradually increased up to 15th hospitalization day and was sustained thereafter. The natriuresis decreased by day 20. The patient underwent an operation due to hydrocephalia and anti-tuberculous therapy has been gone on. He has marked neurologic sequela but he never experienced hyponatremia thereafter.

DISCUSSION

In PICU, hyponatraemia is a common electrolyte disorder in the setting of central nervous system disease and is often attributed to the syndrome of inappropriate secretion of antidiuretic hormone (SIADH). SIADH is characterized by hyponatraemia with an inappropriately concentrated urine, increased urine sodium concentration, and evidence of normal or slightly increased intravascular volume. On the contrary, there are patients with intracranial disease who develop hyponatraemia with similar characteristics but differ in that there is clinical evidence of a contracted extracellular fluid volume. This form of hyponatraemia is due to excessive renal sodium excretion resulting from a centrally mediated process and is termed as CSW. CSW was first defined in 1950 as renal loss of sodium and dehydration in a patient with CNS disease[1].

Atrial natriuretic peptide (ANP) and brain natriuretic peptide (BNP) have several effects that could lead to clinical syndrome of CSW[2]. Their secretion is under normal circumstances stimulated by increased cardiac volume and pressure. Damage to ANP and BNP containing cells in intracranial disorders and passage of these peptides across the blood barrier might cause inappropriate release of natriuretic peptides[3,4,5].

Because of different pathophysiological mechanisms on SIADH and CSW, which require converse therapeutic regimens, early differential diagnosis is mandatory in order to give correct treat-

ment[6]. It is difficult to make differential diagnosis between CSW and SIADH in clinical practice, given the similarity in laboratory values and the overlap in associated intracranial disorders. Determination of extracellular volume remains the primary means of distinguishing these disorders: it tends to be expanded in SIADH and low in CSW. The main biochemical findings of CSW will be low plasma osmolality with inappropriate high urine osmolality (urine to plasma osmolality ratio>1), hyponatraemia with urine sodium loss more than 20 mmol/l, normal/high haematocrit, and plasma urea. Plasma renin activity can be raised or in the high normal range; occasionally it may be depressed.

The clinical presentation of hyponatraemia will depend on the rate of development of hyponatraemia. However, none of these are pathognomonic for either condition. At presen-tation, changes in extracellular volume can be subtle and neither clinical or biochemical estimations are able to ascertain them with consistent accuracy. For this reason, in any hyponatraemic patient with deteriorating clinical status, in the absence of clinical signs of hypovolaemia such as hypotension, a practical approach would be perform with a formal measurement of blood volume using central venous pressure to differentiate between SIADH and CSW.

In SIADH, therapeutic interventions should include restriction of water intake; only in advanced SIADH with total body sodium depletion, sodium should be replaced. In children with CSW, intravascular volume should be vigorously maintained with intravenous saline. In severe hyponatraemia, treatment with diuretics can be attempted. Severe symptomatic hyponat-raemia (associated with seizures and/or coma) should be partially corrected by infusion of hyperosmolar sodium solution, that is, infusion of 3% saline (500 mmol/l) at 1-2 ml/kg/h (0.5-1 mmol/kg/h) for two to three hours, followed by rate of correction to less than 12 mmol/l per day. Rapid correction of hyponatraemia can be associated with pontine myelinoly sis. High dose fludrocortisone (0.2-0.4 mg/day) has been found to be beneficial in some patients with CSW, but with close monitoring of plasma potassium concentrations as notable hypokalaemia may occur in childhood[7]. Mineralocorticoid supplementation could effectively treat the natriuresis and polyuria in such patients [8].

Conclusion

To our knowledge CSW secondary to tuberculous menengitis in childhood is a rare entity. It is essential that in a child with hyponatremia and meningitis, an evaluation be undertaken to differentiate between SIADH and CSW. It is of utmost importance to differentiate the two conditions. A wrong diagnosis in children with CSW might lead to inappropriate fluid restriction and worsen the hypovolemia. Supplements of sodium chloride and mineralocorticoids may be useful in managing these children. Pediatricians, especially Pediatric Intensivisits, must be aware of the clinical features that distinguish CSW from SIADH.

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