Journal of Clinical Case Reports

Zhang X, et al., J Clin Case Rep 2016, 6:7
DOI: 10.4172/2165-7920.1000841

Case Report Open Access

Cerebral Salt Wasting Syndrome in Children: One Case and Review of the Literature

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Rec date: Apr 26, 2016; **Acc date:** Jul 23, 2016; **Pub date:** Jul 30, 2016

Abstract

Background: Cerebral Salt Wasting Syndrome (CSWS) refers to the process of intracranial lesions, sodium salt loss by hypothalamus-renal pathway and caused clinical manifestations syndrome of high urinary sodium, hyponatremia, hypovolemia. The clinical manifestations and laboratory are similar to syndrome of inappropriate antidiuretic hormone secretion (SIADH). It is easy to be misdiagnosed.

Case description and management: In this paper, the clinical data of one case of cerebral salt wasting syndrome was diagnosed. By given fluid and sodium replacement, the condition can improve quickly. Through analyzing and reviewing related literature, the understand of diagnosis and treatment of cerebral salt wasting syndrome can be improved.

Conclusions: The main clinical manifestations of cerebral salt wasting syndrome are hyponatremia, high urine sodium, diuresis, hypovolemia. Cerebral salt wasting syndrome in children has very high risks and we should improve the understanding. Early diagnosis and prompt treatment are very important.

Keywords: Children; Cerebral salt wasting syndrome; Syndrome of inappropriate antidiuretic hormone secretion; Hyponatremia; CRP (C Reaction Protein)

Introduction

Cerebral salt wasting syndrome (CSWS) refers to the process of intracranial lesions, sodium salt loss by hypothalamus-renal pathway and caused clinical manifestations syndrome of high urinary sodium, hyponatremia, hypovolemia. The clinical manifestations and laboratory are similar to syndrome of inappropriate antidiuretic hormone secretion (SIADH) and easy to be misdiagnosed. In this paper, the clinical data of 1 case of cerebral salt wasting syndrome was analyzed and reviewed the related literature so that improved the understanding of the diagnosis and treatment of CSWS.

Case Report

A girl, 1 month, Because of cough two days, limbs twitch one day to the hospital. The child was G2P2, full-term birth and natural labor, breastfeeding and no asphyxia. Her mother during pregnancy was healthy. The physical and mental development of the child was the same with the normal of the same age children. Before admission the children had "ribavirin particles, ethylsuccinate particles" et al. but the effect was poor. On admission child with weak breathing, lips cyanosis obvious, anxious to give "Adrenaline, Lobeline, Lamine" *et al.* as salvage therapy. Physical examination: T35.1°C, P152 beats/min, R18 beats/min, WT 3.0 kg, BP 57/36 mmHg, coma, anterior fontanelle flat, neck resistance, three depressions sign obvious. Shortness of breath, lung breath sounds rough and could be heard wet rales. Heart rate 152 beats/min, rhythm of the heart, low heart sound blunt, pathological murmurs was not heard during the valve auscultation area. Abdomen soft, liver below ribs 3 cm, soft muscle tone were increased. Bilateral Babinski sign was positive.

Time	Laboratory tests	Result
After admission Immediately	Blood gas analysis	pH 6.82, PCO2 66.3 mmHg, Na+: 122 mmol/L, Lac 10.7 mmol/L, BE-13.9 mmol/L
	Blood Rt+CRP	WBC: 24.17 × 109/L, Neutrophils%: 42.1, Red blood cells: 3.41 × 1012/L, Hemoglobin: 112 g/L, Platelet: 453 × 109/L, CRP: 24.16 mg/L
	Calcitonin original	25.60 ng/ml
	Myocardialfour	Npro-BNP: 35000.00 pg/ml, Troponin I: 0.193 ng/ml, CK-MB: 35.4 ng/ml, Myoglobin:165.90 ng/ml

	Echocardiography	EF 25%, left ventricular wall motion reduced dispersively and left ventricular function severely reduced
	Chest X-ray	bilateral pneumonia
After 2 hours admission	Blood gas analysis	pH 6.854, PCO_2 60.3 mmHg, Na+: 126 mmol/L, Lac 16 mmol/L, BE-12.3 mmol/L.
After 6 hours admission	Blood gas analysis	pH 7.374, PCO_2 38.5 mmHg, Na+: 129 mmol/L Lac 9.2 mmol/L, BE-2.5 mmol/L
After10 hours admission	Blood gas analysis	pH 7.368, PCO_2 47.6 mmHg, Na+:138 mmol/L Lac 2.2 mmol/L, BE-2.0 mmol/L
After19 hours admission	Blood gas analysis	pH 7.481, PCO_2 33.2 mmHg, Na+: 138 mmol/L Lac 1.8 mmol/L, BE-1.3 mmol/L
The second day after admission	Cerebrospinal fluid routine	Routine yellow, Pan's test (+), cell count: 40 × 106/L, monocytes classification: 0.52, multicore classification: 0.48, central venous pressure 0.41 kPa
	Cerebrospinal fluid biochemistry	Sugar 3.0 mmol/L, chloride 127 mmol /L, cerebrospinal fluid protein: 1581 mg/L
	Cerebrospinal fluid smear	no bacteria
The fourth day after admission	Npro-BNP	11800.00 pg/ml
	Blood Rt+CRP	WBC 8.44 × 109/L, Neutrophils%: 69.3, Red blood cells: 2.88 × 1012/L, Hemoglobin: 87 g/L, Platelets: 317 × 109/L, CRP: 34.3 mg/L
	Echocardiography	EF 58%, left ventricular interval and anterior motion less impressive coordination
	sputum culture and sensitivity	*Streptococcus pneumoniae*
The tenth day after admission	Blood Rt+CRP	WBC: 7.84 × 10^9/L, Neutrophils %: 47.5, Red blood cells: 2.74 × 10^{12}/L, Hemoglobin: 79 g/L, Platelets: 590 × 10^9/L, CRP: <0.50 mg/L
	Calcitonin original	0.21 ng/ml
	Myocardial four	Creatine kinase: 59 U/L, CK-MB: 2.57 ng/ml, Troponin I: 0.020 ng/ml

Table 1: The results of laboratory tests.

Auxiliary examination: Blood gas analysis immediately showed pH 6.82, PCO2 66.3 mmHg, Na+: 122 mmol/L, Lac 10.7 mmol/L, BE-13.9 mmol/L. Blood Rt+CRP (C-Reaction Protein) showed WBC: 24.17 × 109/L, Neutrophils %: 42.1, Red blood cells: 3.41 × 1012/L, Hemoglobin: 112 g/L, Platelet: 453 × 109/L, CRP: 24.16 mg/L. Calcitonin original: 25.60 ng/ml. Myocardial four: Npro-BNP: 35000.00 pg/ml, Troponin I: 0.193 ng/ml, CK-MB: 35.4 ng/ml, Myoglobin: 165.90 ng/ml. Echocardiography showed: EF 25%, left ventricular wall motion reduced dispersively and left ventricular function severely reduced. Chest X-ray showed: bilateral pneumonia. After 2 hours admission blood gas analysis showed pH 6.854, PCO2 60.3 mmHg, Na+: 126 mmol/L, Lac 16 mmol/L, BE-12.3 mmol/L. After 6 hours admission re-examination of blood gas analysis showed pH 7.374, PCO2 38.5 mmHg, Na+: 129 mmol/L, Lac 9.2 mmol/L, BE-2.5 mmol/L. After 10 hours admission re-examination of blood gas analysis showed pH 7.368, PCO2 47.6 mmHg, Na+: 138 mmol/L, Lac 2.2 mmol/L, BE-2.0 mmol/L. After 19 hours admission re-examination of blood gas analysis showed pH 7.481, PCO2 33.2 mmHg, Na+: 138 mmol/L, Lac 1.8 mmol/L, BE-1.3 mmol/L. The second day after admission lumbar puncture was done. Cerebrospinal fluid routine showed yellow, Pan's test (+), cell count: 40 × 106/L, monocytes classification: 0.52, multicore classification: 0.48, central venous pressure 0.41 kPa. Cerebrospinal fluid biochemistry showed sugar 3.0 mmol/L, chloride 127 mmol/L, cerebrospinal fluid protein: 1581 mg/L. Cerebrospinal fluid smear was not found bacteria. The fourth day after admission review of Npro-BNP: 11800.00 pg/ml. Blood Rt+CRP showed: WBC 8.44 × 109/L, Neutrophils%: 69.3, Red blood cells: 2.88 × 1012/L, Hemoglobin: 87 g/L, Platelets: 317 × 109/L, CRP: 34.3 mg/L. Echocardiography showed: EF 58%, left ventricular interval and anterior motion less impressive coordination. The results were shown sputum culture and sensitivity of Streptococcus pneumoniae. After 10 days admission re-examination of blood Rt+CRP showed WBC: 7.84 × 109/L, Neutrophils%: 47.5, Red blood cells: 2.74 × 1012/L, Hemoglobin: 79 g/L, Platelets: 590 × 109/L, CRP: <0.50 mg/L. Calcitonin original: 0.21 ng/ml. Myocardial four: Creatine kinase: 59U/L, CK-MB: 2.57 ng/ml, Troponin: 0.020 ng/ml.

Diagnosis: Purulent meningitis, status epilepticus, CSWS, severe pneumonia, respiratory failure, heart failure.

Treatment: Actively given the child with physiological saline rehydration, active treatment corrected hyponatremia after about 10 hours. And urine output back to normal. After 15 days actively gave endotracheal intubation and anxiliary breathing with breathing machine, antibiotic resistance to infection, reducing intracraninal pressure, eliminating phlegm, smooth wheezing, maintaining water and electrolyte balance etc. symptomatic support treatment, the child got better and discharged from hospital.

Discussion

Peter first reported CSWS in 1950. It refers to the process of intracranial lesions, the sodium salt lossed by the hypothalamus-renal pathway, caused the clinical manifestations syndrome of high urinary sodium, hyponatremia, hypovolemia [1]. In the children's central

nervous system diseases, central nervous system infections, intracranial hemorrhage, tuberculosis or cancer, meningitis and childhood brain tumor surgery are the main causes of CSWS. But for the children reported relatively rare. Celik [2] et al. have reported two cases of status epilepticus in children caused by the cases of CSWS. Smíd [3] reported that after cancer treatment in children with long-term follow-up were found water and electrolyte imbalance also including unexpected lasting CSWS and accounted for 3.6%. In this case CSWS occured because the central nervous system infection associated with status epilepticus. CSWS clinical progress rapidly, if the diagnosis, treatment time, clinical manifestations will quickly ease in a short time and completely back to normal. If diagnosis and treatment delay life would be threaten.

CSWS pathogenesis is not yet entirely clear. Central can affect kidney through humoral and neural mechanisms of sodium reabsorption, one or more disorders are likely to lead to CSWS. Humoral regulation is related with ANP, BNP and other natriuretic factor as endogenous digitalis-like substances. BNP and ANP are strong natriuretic factor. Through competitively inhibiting renal tubular ADH receptor, renal tubular reabsorption of sodium and water is inhibited. Then a lot of sodium and water through the urine cause sodium deficiency, sodium loss more than water. At the same time, from the level of brain stem directly restrain autonomous impulsive came to participate in the nervous system to adjust the kidney. Byeon [4] found that CSWS was associated with an increased level of plasma ANP, BNP. The child testing BNP obviously elevated at the time of admission, consistent with the report. But about ANP, BNP in the role of CSWS pathogenesis remains controversial. Zhang [5] et al. reported 27 cases in children of 68 cases of acute brain injury with hyponatremia, concentrated in severe cases. After acute brain injured caused by central hyponatremia and hypothalamus pituitary system was damaged and produced too many class digitalis substance (EDLS) and antidiuretic hormone (ADH). It seemed concentration of blood center atrial natriuretic peptide (ANP) and b-type brain natriuretic peptide (BNP) declined in no direct effects on the serum sodium concentration. Therefore, the role of ANP, BNP in CSWS remains to be further explored. Hypothalamic lesions of sympathetic nerve tension decreases after intracranial disease. Decreased renal sympathetic nerve activity leads to increase renal blood flow and increase glomerular filtration rate and renin secretion. Renal tubular sodium reabsorption reduction [6]. Resulting blood volume decreased, blood sodium decreased, urinary sodium increased.

CSWS mainly show extracellular fluid to reduce. Because of reduced extracellular fluid may have dehydration performance such as low central venous, orthostatic hypotension, tachycardia, dry mucous membrane, jugular vein collapse, body weight loss, *et al.* [7]. Laboratory tests can be found plasma osmotic pressure is normal or high, hematocrit increase, urinary sodium increase, urine output increase *et al.* Due to lower serum sodium can lead to cerebral edema and increase intracranial pressure. Clinically can appear headache, weakness, irritability, diaorientation, slow reaction *et al.* Occurrence of acute hyponatremia (48 h or less) can cause neurogenic pulmonary edema, cerebral infarction, shortness of breath, epilepsy, brain herniation, even death [8].

Treatments of CSWS mainly add volume and sodium salt, so that positive sodium balance. Specific programs should be intravenous and (or) oral rehydration salts treatment of patients according to the severity of hyponatremia. Intravenous saline is the most commonly method. Under the condition of hopotonic can infuse 3% sodium salt solution. Under hypertonic conditions furosemide cooperate with therapy when necessary. When given 3% salt solution require to monitor central venous pressure. Sodium rehydration rate remains some controversy, too fast or too slow are at risk. There have been reported seriously rapid corrections of hyponatremia caused pontine and pontine outside demyelination [9]. Another report, fludrocortisones by adding sodium from renal tubular auxiliary reabsorption correct the negative balance of sodium salt. It has certain curative effect on CSWS. Celik et al. [10] reported fludrocortisone acetate treated 3 cases of tuberculos encephalitis with CSWS. Sodium and stability of the liquid were obtained. Choi et al. [11] reported a case of 17-year-old boy such as CSWS due to cerebral penetrating cyst by joining fludrocortisone obtained good curative effect.

The child considered had the central nervous system infection with CSWS. The child appeared sudden poor response, convulsions, respiratory failure etc. performance and sodium decreased significantly. BNP was significantly increased. After high concentrated sodium chloride was given the symptoms relieved and returned to normal. The clinical course was in line with the characteristics of CSWS and consisted with the literature reports. Diagnosis of the disease should be identificated with inappropriate antidiuretic hormone (SIADH). Another disease characterized by a high volume of hyponatremia syndrome. Tinggaard [12] et al. and Zhang [5] et al. reported SIADH and CSWS were leading reason of brain damaged in children with hyponatremia hyperlipidemia. Both pathological mechanisms, diagnostic criteria, treatment methods were different. SIADH was mainly for high blood volume and normal total sodium. While CSWS was characterized by a disproportionately low blood volume and decreased total sodium. In SIADH reabsorption of water increase, water and sodium excretion decrease, urine volume reduce or normal. In CSWS reabsorption of water is normal, sodium and water increase excretion, urine output increase. In addition, limited water test and fluid test can help identify. But few patients CSWS and SIADH can coexist. Kaneko [13] reported 1 case of child with brain tumors alternated SIADH and CSWS. Sorkhi [14] reported 102 cases of children with central nervous system disease, 4 cases occurred CSWS, 3 cases of SIADH occurred. CSWS incidence was even higher than SIADH. Therefore, it was great significant to distinguish between the two. There was reported CSWS restricting fluid intake, the misdiagnosis SIADH. Then increase the damage to the nervous system, resulting in a poor prognosis.

Besides, CSWS should also identified with central diabetes insipidus. Central diabetes insipidus have higher mortality rate. It was reported the mortality rate could be as high as 77.8%. And 16 cases of 54 cases occurred [15] CSWS in the early. John [16] and other studies reported that distinguished central diabetes insipidus, SIADH and CSWS had significant value to prevent brain damage caused nervous system performance.

In a short, CSWS is an important reason to severe brain damage caused hyponatremia. A study reported [17] in 281 cases of brain tumors children after operation 15 cases occurred CSWS. Children of CSWS proned to postoperative shock, 47% proned to low sodium convulsions. Research reported [18] hyponatremia caused by CSWS was one of the independent risk factors of childhood brain tumors seizures. Therefore, we should increase awareness of the disease. Once central hyponatremia appears children should be quickly identify CSWS, SIADH and central diabetes insipidus. Determination of Urine volume and urinary sodium has higher clinical value to hyponatremia children. As long as accurate and timely diagnosis, appropriate

treatment, the prognosis is good. About CSWS pathogenesis remains to be further studied.

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