Cerebral Salt Wasting Syndrome

**Definition**

Cerebral salt wasting syndrome (CSWS) is defined as the renal loss of sodium and extracellular fluid volume following acute or chronic central nervous system injury with normal kidney function.\(^1\,2\,3\) Due to the hemoconcentration effects, both serum urea nitrogen and hematocrit are elevated.\(^18\) However, excessive sodium losses in the urine lead to decreases in serum osmolality.\(^7\)

Natriuresis increases sodium and water excretion through the renal system, which results in increases of urine output, renal sodium excretion, urine osmolarity and specific gravity.\(^10,18\)

**Epidemiology**

Hyponatremia is a common fluid and electrolyte imbalance resulting from different neurological and neurosurgical diseases such as head injuries, tumors, infections, or strokes.\(^4\) Approximately 60% of patients with subarachnoid hemorrhages develop hyponatremia.\(^5\)

**Pathophysiology of the Disease**

CSWS can be triggered by stress responses to surgical procedures, general anesthesia, or sepsis.\(^6\) Neurological conditions related to CSWS include brain tumors, trauma, surgery, hemorrhages, and infections.\(^2\)

The mechanism of CSWS is not completely understood. Two pathophysiological mechanisms of CSWS have been proposed. They include disregulation of sympathetic response and increased serum levels of atrial natriuretic peptide and brain natriuretic peptide.\(^10,11,12,13\)

There are three common types of natriuretic peptides, the atrial natriuretic peptide (ANP), brain natriuretic peptide (BNP), and C-type natriuretic peptide (CNP). ANP and BNP are released when the heart is stretched due to increased volume.\(^11,12,13\) Atrial natriuretic peptide is derived from the atrium. It is a hormone with a wide range of effects, which include natriuresis, diuresis, vasodilation, and inhibition of the renin-angiotensin - aldosterone axis and the sympathetic nervous system.\(^1,13\) BNP is mainly secreted from the ventricles of the heart. It directly inhibits sodium reabsorption from the renal tubule and inhibits the release of renin and aldosterone from the juxtaglomerular cells.\(^14\)

CNP is found in the brain and CSF. It is released from the endothelial cells and acts as a vasodilator but with minimal natriuretic activities.\(^1\)

Natriuretic peptides have renal effects and vasodilation effects. The renal effect promotes sodium and water excretion, which decrease intracranial pressure (ICP). The vasodilation effect prevents cerebral vasospasm.\(^9,15\) These effects can be protective mechanisms to prevent cerebral ischemia after a brain injury or subarachnoid hemorrhage. However, in CSWS, excessive release of ANP and BNP lead to hypovolemia, which may exacerbate vasospasm.\(^16\) Hyponatremia in CSWS can disrupt the blood brain barrier and cause vasogenic edema and increase ICP.\(^16\)

**Manifestations of CSWS**

Manifestations of cerebral salt wasting syndrome include hyponatremia, polyuria, and increased urine sodium.\(^3\,17\)

Diuresis results in hypovolemia, hemoconcentration, and increase urine output. Signs and symptoms of hypovolemia include tachycardia, postural hypotension, reduced skin turgor, and low central venous pressure.\(^10,14\)

There is no consensus on the criteria for diagnosing CSWS. Several clinical findings and laboratory tests have been used to establish the diagnosis of CSWS. Both serum and urine analysis results are altered in CSWS.

Urinalysis results demonstrate increased urine sodium concentration. Urine osmolarity is increased due to excessive amount of sodium is excreted in the urine. Urine output may be normal but is usually increased because water is excreted with sodium.\(^9\)

Negative fluid balance in CSWS results in hemoconcentration.\(^19\) Typical serum results for CSWS include elevated hematocrit, blood urea nitrogen (BUN) and creatinine ratio, and serum protein concentration, with decreased sodium concentration.\(^3\)

**Treatment Options**

The goal of therapy is to maintain a positive sodium balance and prevent volume depletion.\(^14\)

Patients with CSWS are volume depleted with sodium wasting, therefore resuscitation with isotonic sodium solution is crucial.\(^7,14\) Some authors suggest using 3% NaCl in patients with severe hyponatremia and for stable patients with mild hyponatremia, oral salt supplements (tablets) may be adequate.\(^17\) Sodium replacement must be calculated carefully. Rapid replacement of sodium may result in central pontine myelinolysis or extrapontine myelinolysis.\(^5\)

The amount of fluid replacement should match urine losses. Restriction of fluids in patients with CSWS leads to hypotension, shock, increased risk of cerebral vasospasm, cerebral ischemia, and infarction.\(^8,18\)

Fludrocortisone, a mineralocorticoid may be order to enhanced sodium reabsorption in the distal renal tubules.\(^16\) Adverse effects of fludrocortisone include pulmonary edema, hypokalemia, and hypertension. Fludrocortisone is only available in oral preparations. For patients who are unable to take oral medications, a less effective medication, hydrocortisone via intravenous injection, may be used as an alternative.\(^9\)

**Central Pontine Myelinolysis and Extrapontine Myelinolysis**

If severe chronic hyponatremia is corrected too rapidly, central pontine myelinolysis (CPM), and/or extrapontine myelinolysis (EPM) may result.\(^1,9\) Mortality rates of CPM/EPM are approximately 30%.\(^20\)

The etiology of CPM/EPM is unclear. It is suggested that during hyponatremia, extracellular osmolality is lower than...
intracellular osmolality. To avoid brain cell edema due to influx of extracellular fluid, neurons remove some of the intracellular electrolytes and organic osmolytes to equalize the intracellular and extracellular osmolality\(^2\).

The rapid replacement of sodium to correct hyponatremia increases the osmolality of extracellular fluid. If the organic osmolytes are unable to synthesize or transport into the brain cells, it will create an osmolality gradient (lower osmolality inside the cells) and fluid shifts (efflux) from intracellular space to extracellular space and results in shrinkage of the cells\(^2\).

Myelolysis has a biphasic presentation. The initial phase is related to hyponatremia, where the patient presents with malaise, nausea, headache, lethargy, confusion, and seizure\(^2\). Patients may improve in a couple of days if hyponatremia is corrected. Presentation of the second phase is related to myelolysis. Several days after correction of hyponatremia, patients develop neurological symptoms including spastic quadriplegia, pseudobulbar palsy, mental disorders, mild confusion, coma, and death\(^3\).

**Nursing Implications**

Patient with CSWS are hypovolemic - monitor the central venous pressure for the status of patient's intravascular volume if available\(^2\). Accurately record patient's intake and output balance. Even when the fluid loss in urine is totally replaced; patient will still has a negative fluid balance, because the insensible fluid loss (breathing, perspiration, and feces) for a normal person is approximately 500mL/day. Observe for any signs of hypovolemia such as tachycardia or hypotension.

Natriuresis in CSWS leads to electrolyte imbalances. Monitor the patient's cardiac status for side effects of electrolyte imbalance\(^1\) such as dysrhythmias. Monitor the patient's neurological status for any complication of hyponatremia such as confusion, lethargy, seizures, and coma\(^2\). During sodium replacement, monitor for any signs and symptoms of CPM/EPM.

If patient is on fludrocortisone, closely monitor the patient's serum potassium (hypokalemia) and glucose (hyperglycemia) concentrations\(^1\).

**References**


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