Cerebral salt loss syndrome

Cerebral salt-wasting syndrome (CSWS) is defined as the development of excessive natriuresis with subsequent hyponatremic dehydration in patients with intracranial disease. The cause of hypoosmolality and hyponatremia is completely different from SIADH. Hyponatremia and serum hypoosmolality from sodium loss are present, volume ECT is reduced and hypovolaemia, sodium urine loss is very high. Uricaemia and uricosuria are normal, but there may be less waste uric acid.

Etiology

The most commonly reported are trauma and CNS tumors, intracranial hemorrhage, neurosurgery, tuberculosis meningitis, etc. The exact incidence is unknown. About 60% of children with intracranial injury or CNS tumor have hyponatremia. The cause is usually equal to CSWS and SIADH.

Pathophysiology

CSWS was first described by Peters et al. in 1950. The pathophysiological mechanism is still not precisely elucidated. One hypothesis assumes an increased sympathetic nerve activity due to a CNS lesion, followed by an increase in renal perfusion pressure and the release of dopamine in a natriuretic effect. A better known hypothesis assumes the release of natriuretic factors (atrial natriuretic peptide - ANP, brain natriuretic peptide BNP, C-type natriuretic peptide CNP, oubain-like compound OLC due to an intracranial lesion. BNP was first detected in a pig brain sample (hence its name), but its predominant formation is in the area of heart ventricles.

Diagnostics

The basis of diagnostics, similar to SIADH, are laboratory tests. In serum we find, similarly to SIADH, hyponatremia with hypoosmolality and in urine extreme natriuresis and increased urinary osmolality. The sodium balance is negative and the diuresis is in the polyuria range. It is usually a depletion of extracellular fluid.

- S-Na: <135 mmol / l
- S-osmolality: <280 mmol / l
- U-Na:> 25 mmol / l
- polyuria, dehydration [1]
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Differential diagnostics

In the differential diagnosis, we must distinguish other diseases associated with hyponatremia: congestive heart failure, renal or liver failure, hypothyroidism, [[Addison's disease | adrenal insufficiency]], iatrogenic induced hyponatremia (hypotonic infusions, diuretics). Paradoxically, it may be the most difficult to distinguish CSWS from SIADH. Both syndromes have several laboratory features in common, but the treatment is completely different. SIADH is also most commonly associated with CNS lesions in childhood. Assessing the patient's hydration is crucial in differential diagnosis. While CSWS shows signs of dehydration, SIADH is characterized by euvolemia or mild hypervolemia (but no swelling). However, distinguishing between the two syndromes can be very difficult in some cases. Therefore, we also assess the parameters of diuresis, excretion fraction for sodium and sodium balance. An interesting parameter is the *uric acid excretion fraction* (FEUA). Its normal values are <10%. Patients with both CSWS and SIADH have FEUA elevated. However, after correction of hyponatremia, FEUA normalizes in SIADH but remains elevated in CSWS. Therefore, some authors suggest using FEUA as a differential diagnostic marker in a very vague clinical picture. Although, as mentioned above, great pathophysiological importance is attached to natriuretic factors, they do not figure (yet?) As criteria in the mosaic of CSWS diagnostics.

	SIADH	CSWS[1]
creatinine clearance	increased	normal
excretion fraction	normal/decreased	increased
sodium balance	balanced	negative
diuresis	normal/decreased	increased
extracellular fluid	normal/increased	decreased
dehydratation	absent	present
body weight	normal/increased	decreased
ADH	normal/elevated	elevated
natriuretic peptides: ANP, BNP	normal/elevated	elevated
S-proteins	normal	elevated
S-K	reduced/normal	elevated/normal
urikemia	reduced	normal/reduced
urikosuria	elevated	normal/elevated

Clinical picture

CSWS usually occurs within the first week after the onset of the brain lesion and resolves spontaneously within 2-4 weeks, but sometimes persists for many months. The clinical picture is conditioned by hyponatremia, intravascular volume depletion (dehydration symptomatology) and basic CNS damage.

Therapy

The goal of CSWS treatment is to replenish intravascular volume and maintain a stable sodium. In the acute phase, we administer isotonic and hypertonic NaCl solutions. Hyponatremia must be corrected slowly (an increase of 0.5–0.7 mmol/l/hour or 12–18 mmol/l/day is allowed), otherwise there is a risk of developing *pontine myelinolysis* even with the risk of death. After stabilization, we switch to enteral NaCl supplementation, some authors report the beneficial effect of mineralocorticoids (*fludrocortisone acetate*). Careful monitoring of body weight, water and sodium balance is essential. In cases where it is not really possible to distinguish between SIADH and CSWS, or in the presence of both syndromes, i.v. administration urea.

Links

Related Articles

- Antidiuretic Hormone Secretion Syndrome (SIADH)
- Indoor environment (pediatrics) • Serum osmolality • Sodium dysbalance (pediatrics)
- ADH

External links

- Case report CSWS as a complication in a patient with obstructive hydrocephalus (http://www.solen.cz/pdfs/ped/2008/02/13.pdf)

Source

- HAVRÁNEK, Jiří: *CSWS, cerebral salt wasting syndrome* . (managed)

References

1. http://www.solen.cz/pdfs/ped/2008/02/13.pdf