Cerebral-induced salt loss syndrome

This article has been translated from WikiSkripta; ready for the **editor's review**.

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. There is hyponatremia and serum hypoosmolality from sodium losses, there is reduced ECT volume and hypovolemia, there is a very high urine sodium loss. Uricemia and uricosuria are normal, but there may be lower waste uric acids.

Etiology

trauma and CNS tumors, intracranial hemorrhage, neurosurgical procedures, tuberculosis meningitis etc. are most often mentioned. The exact incidence is unknown. About 60% of children with intracranial injury or CNS tumor show hyponatremia. CSWS and SIADH are equally likely to be the cause.

Pathophysiology

CSWS was first described by Peters et al. in 1950. The pathophysiological mechanism is not exactly clarified even to this day. One hypothesis postulates increased sympathetic nerve activity as a result of a CNS lesion with subsequent increase in renal perfusion pressure and release of dopamine, which has natriuretic effects. A more well-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) as a result of an intracranial lesion. BNP was first demonstrated in a pig brain sample (hence its name), but its predominant production is in the cardiac ventricles.

Diagnostics

Laboratory tests are the basis of diagnosis, similar to SIADH. Similar to SIADH, hyponatremia with hypoosmolality is found in the serum and extreme natriuresis and increased urinary osmolality in the urine. Sodium balance is negative and diuresis is in the polyuria range. There is depletion of extracellular fluid.

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

Differential diagnosis

As part of the differential diagnosis, we must distinguish other diseases associated with hyponatremia: congestive heart failure, renal or liver failure, hypothyroidism, [[Addison's disease|adrenal insufficiency]], iatrogenically induced hyponatremia (hypotonic infusions, diuretics). Paradoxically, it may be most difficult to distinguish CSWS from SIADH. Both syndromes share several common laboratory signs, but the treatment is quite different. SIADH is also most often associated with CNS lesions in childhood. Assessment of the patient's hydration is fundamental in the differential diagnosis. While we find signs of dehydration in CSWS, euvolemia or mild hypervolemia (but without the presence of edema) is typical for SIADH. However, distinguishing the two syndromes can be very difficult in certain cases. Therefore, we also assess diuresis parameters, excretion fractions for sodium and sodium balance. An interesting parameter is the ``excretory fraction of uric acid *(FEUA). Its normal values are < 10%. Patients with both CSWS and SIADH have elevated FEUA. However, after correction of hyponatremia, FEUA normalizes in SIADH but remains elevated in CSWS. Some authors therefore suggest using FEUA as a differential diagnostic marker in a very unclear clinical picture.* Although, as mentioned above, great pathophysiological importance is attached to natriuretic factors, they do not appear (yet?) as criteria in the CSWS diagnostic mosaic.

	SIADH	CSWS[1]
creatinine clearance	increased	normal
excretory fraction Na	normal/reduced	increased
sodium balance	balanced	negative
diuresis	normal/reduced	increased
extracellular fluid	normal/increased	reduced
dehydration	absent	present
body weight	normal/increased	reduced
ADH	normal/increased	increased
natriuretic peptides: ANP, BNP	normal/increased	increased
S-proteins	normal	increased
S-K	reduced/normal	elevated/normal
uricemia	reduced	normal/reduced
uricosuria	increased	normal/increased

Clinical picture

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

Therapy

The goal of CSWS treatment is to replenish intravascular volume and maintain stable natremia. 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 permitted), otherwise there is a risk of developing "pontine myelinolysis" even with risk of death. After stabilization, we switch to enteral NaCl supplementation, some authors report on 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 simultaneous presence of both syndromes, i.v. urea administration.

Links

Related Articles

- Syndrome of inappropriate secretion of antidiuretic hormone (SIADH)
- Internal Environment (Pediatrics) • Serum Osmolality • Sodium Dysbalance (Pediatrics)
- ADH

External links

- Case report of 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*. (edited)

References

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

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