SIADH

thumb|right|250px| Prostorový model ADH thumb|right|250px| Struktura ADH **Syndrome of inappropriate antidiuretic hormone secretion, Schwartz-Bartter syndrome *(SIADH)*** *is characterized by increased secretion ADH (vasopressin), which is not dependent on plasma osmolality. Most children have lung or intracranial disease, and this syndrome can also occur iatrogenically (treatment of enuretics with adiuretin). Patients have normovolemia or relative hypervolemia. It is dilute hyponatremia, hypoosmolality serum. The loss of sodium in urine is slightly higher, certainly not reduced. It is low uricaemia and elevated uricosuria.*

SIADH:

Etiology

- **CNS degeneration:** meningitis, encephalitis, cerebral absces, tumor, trauma/bleeding, hydrocephalus
- **malignancy:** m. Hodgkin, neuroblastoma, lung tumors, GIT and uropoietic tract
- **pulmonary involvement:** pneumonia, TBC, UPV, PNO, acute respiratory insufficiency, chronic obstructive pulmonary disease
- **drugs:** DDAVP, paracetamol, indomethacin, barbiturates, carbamazepine, indapamide, SSRIs, donepezil, thiazide diuretics

Physiology

ADH (antidiuretic hormone), also referred to as arginine-vasopressin (AVP), is a nonapeptide that is synthesized in cells of *ncl. supraopticus* and *ncl. paraventricularis* in hypothalamus. It is transported by axonal transport to the neurohypophysis, from where it is released, along with the transport protein neurophysin, in response to a change in serum osmolality. A change in osmolality of ≥ 2% is already detected by osmoreceptors in the hypothalamus. Increased osmolality leads to an increase in ADH secretion and conversely a decrease in serum osmolality leads to suppression of ADH secretion. In kidneys, ADH increases the permeability of the terminal part of the distal tubule and the collecting ducts in the marrow region by inducing specific membrane transport channels for water - "aquaporins".

Patophysiology

SIADH was first described by Schwartz in 1957. In **SIADH**, there is excessive release of ADH from the neurohypophysis (intracranial disease, drug induction: barbiturates, carbamazepine) or ectopic production of ADH (tumors: lymphomas, Ewing's sarcoma). Elevated levels of ADH occur in pathological conditions that are associated with redistribution of blood in the vascular deposit with subsequent hypovolemia in the area of large vessels in the chest and in the left atrium. In both bacterial and viral pneumonias, emphysema, [[CF] cystic fibrosis], UPV, increased pulmonary vascular resistance causes decreased left atrial filling and increased ADH levels.

Increased ADH secretion causes water reabsorption in the renal collecting tubules. This tends to **expand both intravascular and extravascular volume**. This tendency is compensated by increased secretion of atrial natriuretic peptide, so we find **increased natriuresis** despite low plasma sodium levels. However, the cause of increased natriuresis in SIADH is not fully elucidated; other options for increased natriuresis include increased glomerular filtration, decreased aldosterone secretion. And it is the presence of higher natriusesis that is the fundamental problem in distinguishing SIADH from CSWS.

SIADH is accompanied by a slight increase in body water volume. An immediate response to the rapid decrease in osmolality of extracellular fluids is the passive transfer of water from the extracellular space to the more hypertonic intracellular. This leads to an expansion of the cell volume of the organs (brain swelling). Because most of the excess body water is found intracellularly, hypehydration does not manifest itself clinically for a long time.

Diagnostics

Traditional SIADH criteria:

- S-Na <130 mmol / l
- S-osmolality <280 mOsmol / l
- U-Na> 20 mmol / l
- U-osmolality> S-osmolality
- normal functions of thyroid gland, kidneys and adrenal glands
- absence of peripheral swelling or dehydration

Serum contains hyponatremia, hypoosmolality, normal or decreased potassium, urea, creatinine, uric acid. U-Na> 20 mmol / l (however, at low sodium intake, losses may be significantly lower). Intravascular volume is normal or slightly increased. Patients are normotensive. Another laboratory finding may be MAC with reduced AG (https://en.wikipedia.org/wiki/Anion_gap), but not compensatory hypocapnia.

In case of doubt in the diagnosis, detection of serum or urinary ADH concentration can be used. Under normal circumstances, ADH cannot be detected in hyponatremic conditions; on the contrary, we demonstrate increased levels in SIADH. However, we must interpret ADH levels carefully, because ADH secretion is not only affected by osmolality, but also by stress, pain, increased intracranial pressure, etc.

Another possibility is to determine the serum concentration of copeptin, which is excreted together with ADH in equimolar amounts, but is more stable than ADH.

Clinical symptoms

Klinické příznaky jsou projevem **edému mozku**. Při akutním průběhu se objevuje letargie, apatie, dezorientace, nauzea, zvracení, svalové křeče, snížení hlubokých svalových reflexů, patologické reflexy a Cheyne-Stokesovo dýchání. Pokud se hyponatrémie rozvíjí v průběhu několika dnů a týdnů, pacient může být asymptomatický nebo s nespecifickými příznaky jako nauzea, zvracení nebo letargie. Vzácně může docházet i k rabdomyolýze.

Clinical symptoms are a manifestation of **brain edema**. In an acute phase, lethargy, apathy, disorientation, nausea, vomiting, muscular cramps, decreased deep muscle reflexes, pathological reflexes, and Cheyne-Stokes breathing occur. If hyponatremia develops over several days and weeks, the patient may be asymptomatic or with non-specific symptoms such as nausea, vomiting or lethargy. Rarely, rhabdomyolysis may occur.

Symptomatology is not given by the absolute value of sodium, but by the rate of its decline. This must be kept in mind during treatment. Prolonged hyponatremia must be corrected slowly, whereas acute neurological symptoms require prompt intervention.

Therapy

In the case of a chronic disease, it is usually sufficient to **restrict fluids** to 2/3 (or less) of daily needs and p.o. sodium substitution. In acute conditions and critical value of sodium Na <120 mmol / l we administer 1/1 FR or hypertonic solutions (eg 3% NaCl 3 to 5 ml / kg iv as a slow bolus) + furosemide 1-2 mg / kg i.v. (can be repeated according to the response and the state of the internal environment). As soon as the acute symptom subsides, we return to fluid restriction. The long-term goal of treatment is to correct hyponatremia within 48 hours. It is essential to eliminate the root cause.

The condition of treatment is monitoring of body weight, diuresis and internal environment, including correction of sodium and osmolality in serum and urine.

Another possibility is the use of Vaptans - eg. tolvaptan.

Pediatricians must keep in mind that hypoosmolar syndrome can also be caused iatrogenically by feeding more 5% or 10% glucose without ions!

Links

Sources

- HAVRÁNEK, Jiří: *SIADH, syndrom neadekvátní sekrece ADH*. (upraveno)
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Related articles

- Cerebral Salt Loss Syndrome (CSWS)
- Internal environment (pediatrics) • Serum osmolality • Sodium imbalance (pediatrics)
- ADH

External links

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