Hyponatremia (pediatrics)

->**Hyponatremia** is defined as **S-Na < 130 mmol/l**. The risk of serious complications occurs when S-Na decreases <120 mmol/l, especially if this decrease occurs rapidly, ie within 48 hours = **acute hyponatremia**. The body's primary defense mechanism is the production of highly dilute urine and the excretion of free water. If these patients have altered renal function, hyponatremia may be exacerbated.

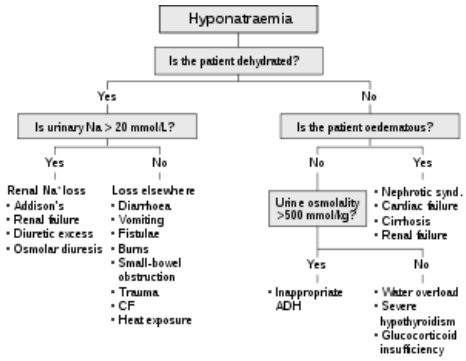
Clinical picture

Cellular hyperhydration occurs, especially in the CNS (brain edema), so the predominant symptomatology is neurological. Patients tend to be apathetic, have anorexia nervosa, nausea, vomiting, hypotension, Cheyne-Stokes breathing. Other times, unrest dominates, cephalogia, ev. convulsions or unconsciousness. Mortality in these cases reaches up to 50%! Where the development of hyponatraemia is creeping, ie >&thinb;48 hrs. the symptomatology is very discreet.

In general, children are more likely to develop **hyponatremic encephalopathies** than children. In addition to altered water distribution, hypoxemia is also involved, due to decreased cerebral perfusion and respiratory distress due to pulmonary edema (neurogenic pulmonary edema).

Diagnostics

In the introduction, it is necessary to determine S-Na and S-osmolality (S-osmo). Hyponatremia accompanied by normal or elevated S-osmo is referred to as **pseudohyponatremia**. Its most common causes are hyperglycemia, severe hyperlipidemia or hyperproteinemia, parenteral delivery of osmotic diuretics. In hyperglycemia, S-osmo increases and at the same time water moves from ICT to ECT, ie an increase in glycemia of 3 mmol / l leads to a decrease in sodium by 1 mmol / l. In hyperproteinemia / hyperlipidemia, S-Na decreases, but S-osmo remains unchanged. The so-called **true hyponatremia** is always accompanied by hypoosmolality, and the next diagnostic step is the determination of osmolality urine (U-osmo), urinary sodium waste (U-Na) and the evaluation of hypovolemia/ euvolemia/hypervolemia.



Estimation of volume depletion can sometimes be very misleading, eg in children with swelling y or where hyponatremia has developed due to increased urinary sodium loss (renal impairment, diuretics), signs of volume depletion usually missing. This is because these patients tend to have an intact mechanism of thirst and p.o. they receive a considerable amount of hypotonic fluids. Despite the aforementioned limiting factors, if hyponatremia is accompanied by a U-Na finding of 20 mmol/l, then there is a significant loss of circulating volume.

By combining the values of S-Na, S-osmo, U-Na, U-osmo and assessing the condition of ECT, we can differentially diagnose the following types of hyponatremia:

S-Na < 130 mmol/l + S-osmo > 280 mmol/kg → pseudohyponatremia

- *hyperosmotic:* hyperglycemia, an osmotic diuretic (eg mannitol);
- *isoosmotic:* hyperproteinemia, hyperlipidemia.

S-Na < 130 mmol/l + S-osmo < 280 mmol/kg + U-osmo < 100 mmol/kg

- psychogenic polydipsia;
- water intoxication;
- otherwise set osmostat.

S-Na < 130 mmol/l + S-osmo < 280 mmol/kg + U-osmo > 100 mmol/kg + hypovolaemia/dehydration

a. + U-Na < 20 mmol/l→ extrarenal losses:

- GIT;
- cystic fibrosis;
- hyperthermia;
- burns;
- ascites / other fluid sequestration;

b. + U-Na> 20 mmol/l → renal losses:

- salt-wasting nephritis;
- diuretics;
- mineralocorticoid deficiency (Addison's disease, CAH);
- renal tubular acidosis;
- pseudohypoaldosteronism;

- CSWS.

S-Na < 130 mmol/l + S-osmo < 280 mmol/kg + U-osmo > 100 mmol/kg + euvolemia

a. U-Na > 20 mmol/l;

- SIADH;
- otherwise set osmostat;

b. U-Na < 20 mmo/l → repeat the algorithm

c. other etiology

- hypothyroidism;
- deficiency glucocorticoids;
- postoperative period;
- UPV;
- nausea/stress/pain.

S-Na < 130 mmol/l + S-osmo < 280 mmol/kg + U-osmo > 100 mmol/kg + hypervolaemia / edema

a. U-Na > 20 mmol/l:

- acute renal failure;
- chronic renal failure.

b. U-Na < 20 mmol/l:

- cardiac insufficiency;
- cirhosis;
- nephrotic syndrome.

In a simplified scheme, we can divide hyponatremia into 3 basic groups:

- pseudohyponatremia;
- hyponatremia with low serum and urine osmolality (U-osmo < 100 mmol/kg);
- hyponatremia with low serum osmolality, but U-osmo > 100 mmol/kg. This 3. typ we further differentiate according to the patient's hydration status.

Hypovolemic hypotonic hyponatremia

It most often occurs in extrarenal (gastroenteritis, profuse sweating, burns) or renal (mineralocorticoid deficiency, diuretics) losses sodium u. The symptoms of ECT loss are clinically dominated: decreased skin turgor, cold cyanotic acre, tachycardia, orthostatic hypotension, oliguria, azotemia. With extrarenal sodium loss, functionally intact kidneys excrete urine with high osmolality but low sodium, U-Na < 10 mmol/l. In conditions with metabolic alkalosis, only a low urinary chloride concentration can indicate a circulating volume deficit: Cl <10 mmol/l. More pronounced azotemia with excretion of hypo/isotonic urine with high natriuresis U-Na > 20 mmol/l conditions with renal sodium loss occur. CSWS occupies an important position here.

Isovolumic hypotonic hyponatremia

SIADH is a representative of this type of hyponatremia, although in reality there is also relative hypervolemia. However, this fact cannot be objectified by physical examination.

Hypervolemic hypotonic hyponatremia

Patients have generalized swelling and excrete small amounts of concentrated urine with low sodium concentrations U-Na < 10 mmol/l (heart failure, nephrotic syndrome, liver cirrhosis). Higher sodium concentration U-Na > 20 mmol/l is in patients with acute tubular necrosis and/or acute renal failure.

Additional comment

A combination of several factors usually leads to the development of postoperative hyponatremia: improper tactics parenteral fluid delivery, lack of diuresis monitoring, and nonosmolar stimuli leading to increased secretion ADH. In infancy, poorly prepared hypotonic formula may contribute to the development of hyponatremia. The possibility of severe hyponatremia in patients with enuresis treated with DDAVP is current.

Hyponatremia therapy

Two basic principles must be respected:

- fluid intake restrictions;
- correction of the inducing pathological mechanism. We assess the urgency of active correction of hyponatremia according to the severity of the situation in which the degree of hyponatremia, the rate at which

the deficit arose and the patient's clinical picture play a dominant role.

deficit calculation Na in mmol = 0,6 × kg. t.h. × (normal S-Na - detected S-Na)

we usually serve 1/3, max. 1/2 of the calculated deficit.

In practice, we proceed as follows: If Na is 120–125 mmol/l and the patient has no signs of dehydration, the mere restriction of fluids and p.o. sodium supplementation may lead to gradual adjustment. If the patient does not have clinical difficulties (convulsions) and Na is > 115–120 mmol/l, we give 1/1 FR, resp. solution, where Na is about approx 60 mmol/l higher than the current value of sodium. If the patient has cramps or is Na < 115–120 mmol/l we give i.v. bolus 1,5–2 mmol Na/kg during 10 min., resp. until the end of convulsions (e.g. 3–4 ml 3% NaCl, where 1 ml = 0,5 mmol). The rise of S-Na by max. 0,5–0,7 mmol/l/ hod., resp. S-osmo increase of max. 1,0–1,4 mmol/l/ hod. The daily rise in sodium should not exceed the value 12–15 mmol/l.

With a rapid correction of chronic hyponatremia, when the daily rise Na is > 15 mmol/l there is a risk pont demyelinating syndrome. The risk is higher if hyponatremia lasts longer. This situation is all the more treacherous because in the beginning there is a clinical improvement, only in the next period (days to a week) neurological symptoms manifest themselves (behavioral changes, pyramidal symptoms, quadruspasticity, pseudobulbar paralysis, coma). Patients with concomitant malnutrition, potassium or hypoxia depletion are more prone to the development of osmotic demyelinating syndrome (which has its correlates in the CT/MRI brain).

With very low sodium, it is sometimes advantageous to determine the so-called target sodium in a certain time, ie the value we want the serum sodium to reach in 12 hours. This target value lies at the level Na < 128 mmol/l. E.g. we have current sodium 115 mmol/l and we want the target sodium in 12 hours to be 127 mmol/l. We will use the formula below:

deficit calculation Na in mmol = 0,6 × kg t.h × (target S-Na − found S-Na v mmol)

Simply put, with severe clinical symptomatology, therapy must be aggressive to achieve a "safe zone". 120–125 mmol/l. An increase in sodium during the first hour of treatment can be tolerated 4–8 mmol/l, however, the subsequent correction must be gradual. In refractory cases, dialysis should be considered..

Links

Source

- HAVRÁNEK, Jiří: Dysbalance natria.

related articles

- Dysbalance natria (pediatrics)
- Hyperrememia (pediatrics)
- Indoor environment (pediatrics)
- Serum Osmolality
- Hyperrememia

External links

- Template:Akutně

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