Sodium imbalance (pediatrics)

Sodium Dysbalance represents one of the most common ionic changes. Sodium is the main cation of ECT and is of key importance to the osmolality of this body compartment. In ICT the concentration of sodium is not the same everywhere, it varies between 3 - 35 mmol/l, but it is always lower than in ECT. The difference in Na concentrations between ICT and ECT (**sodium concentration gradient**) is the result of the active metabolic activity of cells, especially the so-called **sodium pumps** = Na-K-ATPase. Due to the action of this mechanism located in the cell membrane, sodium ions are constantly expelled from ICT to ECT, potassium ions move in the opposite direction. In situations of energy depletion, the activity of Na-K-ATPase is paralyzed and the movement of both mentioned ions changes to the opposite.

Dietary sodium intake is highly individual, 4 – 15 g NaCl (i.e. 70 – 250 mmol/24 hours). The largest part of the ingested sodium is excreted by the kidneys, however, significant extrarenal losses can occur through the skin during excessive sweating (especially in children with a high concentration of Na in sweat - patients with cystic fibrosis). With good kidney function and no increase in the proportion of extrarenal losses, it is possible to calculate with a balanced sodium balance, i.e. p.o. intake corresponds to its excretion in urine. This aspect enables a practical assessment of sodium balance, when the amount of Na excreted in the urine can be converted to NaCl intake.

NaCl intake in g/24 h = Na excreted in urine in mmol/24 h: 17

Example: if the amount of excreted Na was 148 mmol/24 h, it means that the child took in 148 : 17 = 8.7 g of NaCl.

Changes in sodium concentration are always accompanied by a redistribution of body water in individual compartments. ECT hyponatremia leads to a decrease in its osmolality with a subsequent movement of water into the ICT. This redistribution is accompanied by a rise in ECT osmolality. ECT hypernatremia induces exactly the opposite changes. Correct kidney function is determining for all the above data. Sodium ions freely penetrate the glomerular membrane and their concentration in the glomerular filtrate is identical to the S-Na value. In the proximal tubule of the kidney, 50-70% of sodium is resorbed, and <1% of the total filtered sodium eventually reaches the final urine. The value determining what part of the filtered sodium is excreted in the urine is referred to as the fractional excretion of FE Na. The determination of FE Na is used to evaluate renal tubular function and is also one of the auxiliary parameters to distinguish the initial cause of acute renal failure (difference between renal and prerenal ASL).

FE-Na = U-Na / S-Na : U-Kr / S-Kr (FE-Na norm is < 1%)

Hyponatremia

->**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.

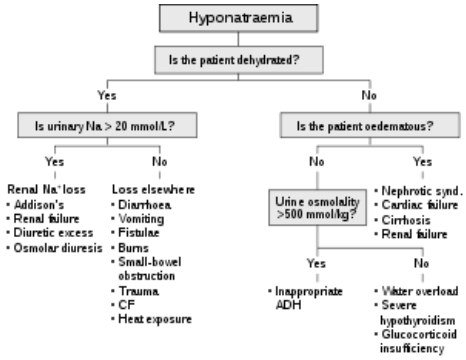
In general, children are more likely to develop **hyponatremic encephalopathies** than children. In addition to altered water distribution, hypoxemia is also involved, due to decreased cerebral perfusion and respiratory distress due to pulmonary edema (neurogenic pulmonary edema).

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..

External links

- Template:Akutně

Kategorie:Vložené články Kategorie:Patofyziologie Kategorie:Patobiochemie Kategorie:Pediatrie Kategorie:Nefrologie Kategorie:Vnitřní lékařství Kategorie:Fyziologie

Hypernatremia

Hypernatremia is denifed as S-Na > 150 mmol/l. If all feedback mechanisms are preserved and the child is conscious, thirst is a typical manifestation of hypernatremia. Another defense mechanism is an increase in ADH secretion and the production of highly concentrated urine. In most cases, this change of sodium concentration in serum occurs when the patient is weakened by some underlying acute or chronic illness, exacerbated by insufficient water intake. *Extracelular osmolality* is compensated by the formation of idiogenic intracellular osmols in the CNS. A significant amount is already present after 24 hours . **Correction of natremia** must therefore be really slow, as it follows that the body is more at risk of rapidly induced sodium loss with the risk of developing cerebral edema than existing hypernatremia.

The clinical picture is modeled by water redistribution and its transfer from ICT to ECT. This is why patients have a long-preserved skin turgor, which is sometimes dough. The brain is the most sensitive to moving water from ICT to ECT. Cerebral dehydration and cell volume shrinkage of varying degrees may occur here. The brain can adapt to a 10-15% reduction in this volume, but> 20% accelerated loss of ICT leads to severe structural changes in the CNS, most of which are irreversible.

Clinical manifestations

Most often we find non-specific symptoms: lethargy, confusion, "squeaky" crying, increased neuromuscular irritability, stiff neck, convulsions, unconsciousness, markedly depressed large fontanel. The symptoms can be very dramatic, as some patients may experience separation of the meningeal sheaths from the brain accompanied by intracranial / intracerebral haemorrhage, and also development of demyelination. There is a big difference between body temperature measured in the rectum and at the foot (> 8 ° C), there may be hypotension or hypertension, manifestations of vasculitis, intravascular coagulation. Rhabdomyolysis may occur in association with hypernatraemia.

Diagnostic algorithm

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

Hyperatremia due to water and sodium deficiency (water deficit is> Na deficit) + decreased ECT volume

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U-Na < 20 mmol/L + U-osmo < 300 mmol/kg → renal loss
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- diabetes insipidus centralis,
- diabetes insipidus renalis.

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U-Na < 20 mmol/L + U-osmo > 600 mmol/kg → extrarenal loss
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- hyperventilation,
- hyperpyrexia.

Hypernatremia due to free water deficit (represents a deficit of "only" water, ie hypernatremia is relative = Na content in the organism is normal) + reduced ECT volume

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U-Na < 20 mmol/L + U-osmo < 300 mmol/kg → renal loss
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- diabetes insipidus centralis,
- diabetes insipidus renalis.

```
U-Na < 20 mmol/L + U-osmo > 600 mmol/kg → extrarenal loss
```

- hyperventilation,
- hyperpyrexia.

Hyperatremia from sodium overdose + normal or elevated ECT

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U-Na > 20 mmol/L + U-osmo is > S-osmo
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- excessive supply of NaCl or $NaHCO_3$,
- incorrect preparation of infant formulas.

Increased natriuresis and urine osmolality are a manifestation of the body's compensation in intact kidney function.

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U-Na < 20 mmol/l  -> renal retention of natrium
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- primary hyperaldosteronism,
- morbus/syndrome Cushing.

In most cases, when U-osmo increases, U-Na decreases and vice versa. Exceptions are hyponatremic conditions of SIADH and CSWS and hypernatremic conditions of diabetes insipidus renalis and centralis.

Additional comment

The cause of hypernatremia is often multifactorial and requires a comprehensive consideration. In addition to the clinical findings, anamnestic data are extremely important, especially the method of preparation of the infant formula in newborns and infants. Hypernatremia in infants and toddlers is also easy with fever and inadequate fluid intake. The body surface area of newborns, infants and toddlers is relatively large in relation to ECT volume and sweating losses (sweat is a prototype of hypotonic fluid except patients with cystic fibrosis) are therefore significant.

Hypernatremia therapy

Hypernatremia associated with > 10% weight loss requires correction by infusion.

$$\text{calculation of water deficit in liters} = 0,6 \cdot \text{weight(kg)} \cdot \left(1 - \frac{\text{normal S-Na}}{\text{current S-Na}}\right)$$

Accurate determination of free water deficit is difficult, especially in hypernatremic dehydration, where most water losses are intracellular, there are no signs of loss of circulating volume, and on the contrary, ECT volume is ensured. In practice, we proceed as follows: for the first 1-2 hours we serve 1/1 FR or 1/1 Ringer sol. We also serve solutions where Na is about 60 mmol/L lower than the current sodium (usually 2/3 - 1/1 solutions). With a good clinical condition of the child and a tendency to normalize laboratory parameters, it is possible to terminate the therapy p.o. rehydration solution. A safe decrease S-Na by max. 0.5 mmol/L/h, resp. S-osmo decrease by max. 1 mmol/L/h. On the contrary, a daily decrease of more than 12 mmol/L carries the risk of cerebral edema. We prolong the adjustment of ECT and ICT to approximately twice the correction time compared to normonatremic dehydration. We usually plan a total correction of 72 hours.

In hypernatremia > 170 mmol/L, the S-Na should not fall below 150 mmol/L during the first 48-72 hours after starting treatment. At Na> 175 mmol/L we consider furosemide 1-5 mg/kg i.v. Dialysis is a last resort in refractory cases. If convulsions occur during hypernatremia therapy, they are probably related to the development of cerebral edema. In this case, it is advisable to slow down the infusion correction, or to apply a smaller dose of hypertonic NaCl solution.

Recommended monitoring

- á 1 hour control of HR, RR, BP, state of consciousness, fluid balance,
- á 4 hours control of Na, K, glycemia a ABB,
- at sodium level below 150 mmol/L we continue the correction as in isotonic dehydration.

Links

Related Articles

- Hyponatremia (pediatrics)
- Hypernatremia (pediatrics)
- Indoor Environment (Paediatrics)
- Serum osmolality
- Sodium imbalance
- Hyponatremia
- Hypernatremia

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