CHRONIC KIDNEY FAILURE IN CHILDREN

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Annotation: *Chronic renal failure is a clinical symptom complex associated with a decrease in glomerular filtration rate by more than 50%, impaired hemostatic function of the kidneys, and a decrease in the number and function of the remaining nephrons. Kidney and urinary tract diseases in children when glomerular filtration rate is less than 20 ml/minute (1.73 m2), serum creatinine is more than 1 mg% (0.177 mmol/l) and urea is more than 50 mg% (8.3 mmol/l) - SBY can be diagnosed.*

Keywords: *glomerulosclerosis, total kidney failure, glomerulonephritis, oligoanuria, pericarditis, hyperphosphatemia, hypokalemia.*

Etiology: In SBY, homeostasis disorder - kidney function disorder, areas of total injury are identified. In SBY -urine osmolality- decreases due to the decrease in the mass of functioning nephrons.

The causes of SBY in children under 5 years of age are considered to be: congenital kidney structural disorders, obstructive anomalies, acquired diseases after 5 years of age are considered to be: focal segmental glomerulosclerosis, chronic glomerulonephritis, hemolytic uremic syndrome, progressive hereditary nephropathy (hereditary nephritis, kidney dysplasia).

• "Total kidney failure" - the pathological process spreads to all parts of the nephron.

• "Partial kidney failure" - limited (isolated) damage to certain functions of the kidney.

• Glomerular-type predominant renal failure.

• Renal failure with predominant tubular type.

• "Terminal renal failure" - many nephrons do not work, compensatory activity is lost.

4 stages of SBY are distinguished:

Stage 1: Compensated (polyuric) hemostatic constant without violation (when a load test is performed) passes with a decrease in the reserve capacity of the kidney.

Stage 2: Subcompensated - is a stage with partial kidney dysfunction, unstable hyperazotemia and anemia in the kidney.

Stage 3: Decompensated - is the stage of SBY that disappears with clear signs.

Stage 4: Terminal, uremic stage - oligoanuria, organ and system damage.

Pathogenesis: A progressive decrease in the number of functioning nephrons leads to a decrease in the kidney's concentration properties and reserve capacity. Renal failure - less than 50% of nephrons, which can be detected when we perform functional tests, indicates damage to the nephrons. Dysfunction due to hypertrophy of functioning nephrons - 75-80% of nephrons are sclerotic. These nephrons normally control osmotic diuresis. As a result of sclerosis of the nephrons, the concentration property of the kidney decreases, polyuria develops, and a large amount of electrolytes (K and Na) is excreted in the urine. In the functionally compensated stage of SBE, the above disorders are observed. When the diseases leading to SBE escalate and more than 90% of nephrons die, the decompensated stage begins (terminal), extracellular fluid homeostasis is disturbed, and hyperazotemia, acidosis, hyperhydration, electrolyte disturbances (hyperkalemia, hypocalcemia, hyponatremia, cardiac arrhythmia) and oligoanuria are observed. In addition to the causes of SBE, high protein in the diet aggravates kidney failure, while a low protein diet prevents the development of SBE.

Persistent proteinuria and systemic hypertension of any etiology lead to increased damage to the kidney-injury and sclerosis of the endothelium of glomerular capillaries. Slow and latent viral or other types of infections (mycoplasma, chlamydial, herpes, enterovirus) - damages the kidneys and urinary tract, leads to the development of an immunopathological process, resulting in SBE. Intestinal dysbacteriosis, constipation, chronic diseases of the gastrointestinal tract, and endotoxicosis can also cause SBE.

The following key syndromes are important in SBE:

Azotemia: the level of creatinine in the blood serum is a generally accepted very specific criterion indicating a defect in the kidney's nitrogen excretory function. Electrolytic disorders: mainly caused by the dysfunction of the tubules. Uremia has the following disorders: hyponatremia, hyperkalemia, hyperphosphatemia, hypermagnesemia, hypocalcemia, and various changes are observed in some patients.

Acidosis: hyperchloremia (congenital and congenital in patients, mainly metabolic disorders, nephropathy, alkalosis) is associated with disturbances in

tubular acidification and ammonogenesis, limitation of accumulation of acidic substances in urine.

Anemia: reduction of erythropoietin synthesis in YuGA occurs as a result of reduction of erythropoiesis, reduction of erythrocyte life span, disruption of protein-synthetic function of liver and iron deficiency, blood loss with urine as a result of the effect of uremic toxins.

Osteodystrophy: Stimulates calcitonin synthesis, kidney 1,25 dehydrooxychole-calceferol derivative, decreased intestinal calcium absorption, hypocalcemia and acidosis, hyperphosphatemia, stimulates the activity of the thyroid gland, increased parathyroid hormone levels, calcitonin deficiency causes osteodystrophy.

Hypertension: due to deficiency of prostaglandin synthesis in the kidney, hyperhydration and hypervalemia, sodium retention, on the other hand, excessive production of renin from YUGA, deficiency of renin inhibitors, or excessive production of renin. Angiotensin 2 is formed as a result of the action of the converting enzyme from angiotensin 1.

Hyperphosphatemia and acidosis develop when SKF decreases by 30%, hyperkalemia, when SKF decreases by 10% (relative to sodium), hyperkeratinemia when SKF decreases by 50%.

Clinical appearance of SBY at the initial stage:

Complaints: fatigue, weakness, decreased appetite, refusal to eat, subfebrile body temperature, polydipsia.

Clinic: retardation of physical development, weight loss, white-yellow skin, loss of sweating, nausea, vomiting, intestinal dysfunction, polyuria, nocturia. Laboratory tests: normal or hypostenuria, mild or moderate normochromic anemia.

Clinical presentation of the tubular type of SBY:

Complaints: rapid fatigue, indifference to training and games, loss of interest, severe headache, anorexia, polyuria, polydipsia.

Clinic: general symptoms for all options; increasing pallor of the skin, deposition of urochrome in skin folds, dry skin, intestinal dysfunction (gastroenterocolitis, nausea, vomiting), nocturia, polyuria.

Glomerulopathy: hypertension, edema.

Tubulopathy: retardation of physical development, osteopathy, polyuria with signs of dehydration, electrolyte disorders, hyponatremia (headache, nausea, convulsions), hypokalemia (muscular hypotonia, apathy), decreased blood pressure.

Laboratory test results:

• decrease in filtration of balls,

• mild or moderate hypochromic or normochromic anemia,

• increased amount of urea in blood serum, creatinine is normal

• mild metabolic acidosis,

• hypostenuria.

Clinical presentation of the 1st degree of Total SBY:

Complaint: More pronounced than tubular insufficiency.

Clinic: common symptoms for all variants, liver-brown skin, hemorrhagic syndrome, gastroenterocolitis, hypertension, osteoporosis, bone demineralization, moderate anemia.

Tubulopathy: worsening bone disorders (bone pain, gait disturbances, limb deformities).

Laboratory test results:

• Reduction of CF by 40-50%, • hypochromic anemia (Nv 80-90 g/l)

• high levels of urea and creatinine,

• metabolic acidosis,

• hyperphosphatemia,

• hypokalemia,

• hypocalcemia.

Clinical presentation of 2 levels of Total SBE:

Complaints and clinical signs: manifested in connection with the development of the pathological process.

Cardiovascular system:

• hypertension,

• heart rhythm disorder,

• pericarditis,

• Changes in the ECG (tachycardia, changes in the amplitude of P, R, T waves, positioning of the RS-T segment below the isoline).

• heart failure.

Respiratory system:

• banal pneumonia

• specific inflammation of "uraemic pneumonitis",

• fibrotic pleurisy,

• nephrogenic lung tumor.

Gastrointestinal tract:

• anorexia,

• nausea, vomiting,

• bad breath,
• gastroenterocolitis,
• motorism.

Nervous psychic sphere:

1. Subclinical stage - slowing of impulse transmission along peripheral nerve fibers. Decreased vibrational sensitivity.

2. The stage of transition to neurological disorders - neurosthenic syndrome, muscle excitability, finger tremors, painful convulsions in calf muscles, "restless legs syndrome".

3. Stage of stable neurological disorders: PNS and muscle damage, polyneuropathy, MNS damage-encephalopathy, stroke.

MENTAL DISORDERS:

• Asthenic,
• asthenodepressive state,
• emergence of fear.

Visual impairment:

• decreased visual acuity,
• darkening of the eyelids,
• "Red eye" syndrome,

Hearing impairment:

Hearing loss of a sound-receiving nature.

Laboratory test results:

• Sudden decrease in HF,
• Severe anemia of mixed type,
• Hypostenuria,
• Decompensated metabolic acidosis,
• An increase in the amount of urea and creatinine in the blood serum,
• hyperphosphatemia,
• Hyperkalemia.

Clinical presentation of 3 levels of Total SBY:

Clinical appearance:

• development of all clinical and laboratory signs of uremia
• decreased diuresis
• muscle atrophy
• changes in the skin and mucous membranes (stomatitis)
• decompensated metabolic acidosis up to coma.

Diagnostics

- Assessment of the patient's anamnesis, heredity, and clinical course
- urine test
- functional examination
- checking homeostasis
- diagnosis of damage to various systems.

Treatment:

Diet: Table #7 is recommended.

Instructions for this diet:

- CF rate 40-10 ml/min* 1.73m2
- when serum creatinine is up to 0.44 mmol/l
- when serum creatinine and urea are up to 16.8 mmol/l

Diet No. 7 according to V. I. Naumov is aimed at less restriction of protein, increasing the amount of fat at the expense of vegetable oils, and providing the necessary energy.

Meat, fish, cottage cheese are limited, cereals, pasta dishes, vegetables, butter, vegetable oil, eggs, milk, kefir, fruits are given.

At the 1st stage of SBY, symptomatic treatment is carried out aimed at eliminating the main disease and symptoms of kidney failure.

In stage 2a, table 3B (the amount of protein is limited to 1.5 g/kg) When azotemia is high, a strict diet with a daily amount of protein of 0.6-0.7 g/kg is carried out. The diet is enriched with carbohydrates and fats. The Giardano-Givanetti diet is very convenient for this; meat, fish and yogurt will be completely limited. Children aged 11-14 years with SBY are given the Giordano-Givanetti diet.

For breakfast: 2 yolks (egg yolk), vinaigrette 200g, tea 200ml, protein-free bread 30g, oil 15g.

Second breakfast: pastila 30 g, apple biscuit 180 g, grape juice 100 ml.

Lunch: 300 g of rice-fruit soup, 200 g of grape-wheat porridge, 200 g of fresh fruit jelly, 40 g of protein-free bread, 10 g of butter. Second lunch: 200 g of fresh fruits.

For dinner: 210 g of fried potatoes with apples, 200 ml of sugar tea, 30 g of lozenges, 30 g of protein-free bread, 15 g of butter. Transferring the child to this diet is carried out at stage 2a when the amount of residual nitrogen is 30 mmol/l.

When azotemia decreases, the amount of protein in the diet is increased by 1.-1.5 g/kg. The amount of salt in the diet is determined taking into account hypertension, edema, daily diuresis and sodium excretion (balags should be equal to 0). Salt restriction in polyuria causes hyponatremia. In case of

hypokalemia, foods rich in K (bananas, apricots, grapes, black plums) are included in the diet. Vitamins E and B are recommended at all stages of SBY.

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