Chronic renal dysfunction (pediatrics)

Chronic renal insufficiency is a disorder of glomerular filtration, which leads to a decrease in it and an increase in creatinine, possibly other components and waste substances (urea, uric acid); this disorder must last at least half a year.[1]

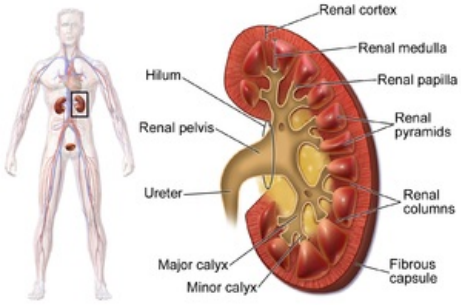
Chronic renal failure ("end-stage renal disease") is a condition where the kidneys are unable to ensure even basic homeostasis in a resting situation without physical and psychological stress. The final stage comes with uremia and death from its complications (disruption of the internal environment with hyperkalemia, severe acidosis, hypertension and brain edema).

- glomerular filtration around 0.16 ml/s/1.73 m² (10 ml/min/1.73 m²);
- weekly urea clearance (Kt)/urea volume of distribution (VV) is < 2.0;
- plasma creatinine > 800 µmol/l (10-fold increase above normal).[1]

The classification of the degree of renal dysfunction according to *the Dialysis Outcomes Quality Initiative, DOQI* is used - 5 degrees according to glomerular filtration disorder.[1]

Etiology in children

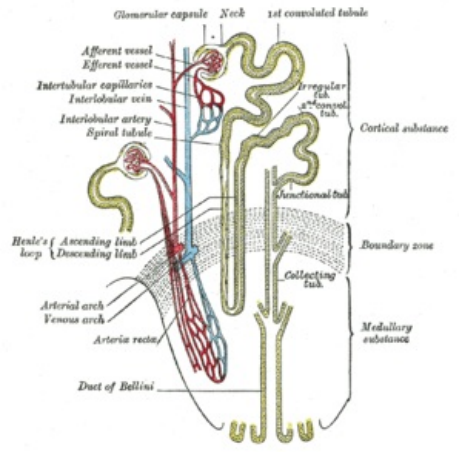
- congenital defects and congenital nephropathy
  - obstructive uropathy including vesicoureteral reflux (25 %);
  - bilateral renal hypoplasia (13 %);
  - cystic kidney disease, especially AR (10 %);
  - juvenile nephroptosis (10 %);
- hemolytic uremic syndrome (7 %);
- chronic glomerulonephritis (22 %);
- in adults, diabetic nephropathy is the most common.[1]



Kidney structure

Pathogenesis

- **decrease in glomerular filtration with a progressive character**; rate of progression depends on diagnosis;
- diuresis is not greatly affected in the first stages – compensated by an increase in the sodium and potassium excretion fraction;
- **disorders of ion and water management**
- **bone disease** (disorder of calcium phosphate metabolism): reduction of glomerular filtration → phosphorus retention → serum phosphorus increases → serum calcium decreases → parathyroid hormone is released (secondary hyperparathyroidism) → increases calcium level by breaking down bones and increases phosphorus excretion by reducing its reabsorption in the tubules (increased excretory fraction of phosphorus) → growth disorder, deformities of long bones (genua vara etc.) and bone pain; deposition of calcium in the vascular system, myocardium and soft tissues → risk of cardiovascular complications – the most common cause of mortality in long-term replacement therapy;
- lack of agents produced in the kidneys: erythropoietin (→ **anaemia**), calcitriol (→ bone disease); or their excess: renin;
- **disorder of urine acidification** with a tendency to metabolic acidosis;
- hyperlipidemia
- **hypertension**
- **nutritional and growth disorder** - multifactorial: reduced energy supply, chronic metabolic acidosis, anaemia, low efficiency in hemodialysis, large protein losses in peritoneal dialysis, late onset of puberty and limited pubertal growth spurt.[1]



Renal duct

Clinical picture

- often only non-specific symptoms: loss of appetite, fatigue, sleepiness, deterioration of school performance, decrease in performance, pallor, headaches; slow development - often escapes attention;
- growth delay (lag-down phenomenon in the percentile chart);
- hypertension, vision disorders, vomiting, impaired consciousness and convulsions. [1]

Therapy

Early inclusion of the patient on the waiting list for transplantation, until then replacement elimination and conservative therapy.

Conservative therapy

- diet: protein in the diet is not limited in children (only in adults); sufficient energy supply - event. nasogastric sodium or PEG feeding; maintenance of fluid balance, limited intake of phosphorus;
- calcitriol and erythropoietin supplementation;
- correction of acidosis with bicarbonate p.o., in smaller children with Shol's solution (mixture of citrates);
- correction of hyperphosphatemia using phosphate binders;
- correction of hyperlipidemia - hypolipidemics (only in older children);
- treatment of hypertension – calcium channel blockers, ACE inhibitors and β-blockers;
- vitamin B, C and folic acid supplementation (but do not give vitamin A and E);
- growth disorder - recombinant human growth hormone.

Replacement elimination therapy

- absolute indication:
  - GF < 0.08 m/s/1.73 m² (5 ml/min/1.73 m²)
  - clinical signs of uremic complications: pericarditis, pleuritis, fluid retention and edema, cardiac insufficiency, clinical and laboratory signs of malnutrition, hypertension unresponsive to treatment, and hyperkalemia that cannot be managed conservatively.

Peritoneal dialysis

- in children by the method of choice
- principle: a special catheter (Tenckhoff) is inserted into the peritoneal cavity, through which a dialysis solution (similar in composition to ECT, but without products that must be removed from the body) is injected inside, hyperosmolar → ultrafiltration through the peritoneal wall, which functions as a semi-permeable membrane;
- it is usually carried out at night, using an automatic device (cycler);
- goal: balance the balance of fluids, ions, manage hypertension, improve the state of nutrition and growth, enable attendance at school;
- risk of peritonitis (turbidity of the drained dialysate, abdominal pain, fever, laboratory signs of inflammation).

Hemodialysis

- principle: extracorporeal blood purification using a dialyzer, in which blood flows on one side of the dialysis membrane and dialysis solution (modified Ringer's solution) on the other; fully automated device (the so-called artificial kidney); an arteriovenous connection (fistula) is formed on the forearm of the non-dominant arm, in which a varix is formed due to high blood pressure, into which 2 needles are inserted;
- takes place in children's dialysis centers; mostly 3 times a week for 4-5 hours;
- indications: contraindications to peritoneal dialysis, insufficient permeability of the peritoneum, adhesions in the abdominal cavity, inability of parents to handle dialysis at home, wishes of adolescents.

Kidney transplantation

- from a technical point of view, it is already possible for children from 10 kg;
- from a deceased or living donor (most often a relative);
- absolute contraindications: active or untreated malignancy, chronic HIV infection, a multi-organ failure that would require transplantation of other organs, positive cross-match in donor-recipient; relative: chronic hepatitis C, severe family and child non-compliance and lack of support from the family;
- in children under 5 years of age, organ transfer is more risky due to the higher reactivity of the immune system;
- the kidneys themselves are usually left in situ; the graft is located extraperitoneally, its vessels are sewn to the vasa iliaca (in smaller children, directly to the aorta and inferior vena cava), the ureter is sewn to the bladder with a modified antireflux plastic;
- immunosuppression: corticoids (methylprednisolone) with gradual reduction and withdrawal, tacrolimus etc.;
- after a successful transplantation, the quality of life increases significantly;
- complications: acute graft rejection (fever, pain, decrease in diuresis, hypertension or asymptomatic rejection);
- chronic graft rejection – chronic transplant nephropathy (decreased glomerular filtration, uncontrolled hypertension);
- risk of infection due to long-term administration of immunosuppressants (urinary tract infections etc.).[1]

Links

Related articles

- Acute renal failure (pediatrics)

References

1. LEBL, – JANDA, J – POHUNEK, P, et al. *Klinická pediatrie.* 1. edition. Galén, 2012. 698 pp. pp. 628-635. ISBN 978-80-7262-772-1.