Emergency care kidney disease in children

Methodical instructions for students of the 5th and 6th grades course of higher medical institutions of education III–IV levels of accreditation

Міністерство охорони здоров'я України
Харківський національний медичний університет

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Невідкладна допомога дітям з захворюваннями нирок

Методичні вказівки
для здобувачів 2 рівня вищої освіти 5-го та 6-го курсу
вищих медичних закладів освіти III–IV рівнів акредитації

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Introduction and definition

Acute and chronic kidney failure are the main pathological conditions that require urgent care in children with kidney disease.

Acute renal failure (ARF) is a symptom complex characterized by a rapid loss of homeostatic functions of the kidneys.

Chronic renal failure – a violation of kidney functions that has been observed for three or more months, occurs as a result of the progressive death of nephrons and stroma in chronic kidney disease (hereinafter CKD), subacute malignant glomerulonephritis or after acute renal failure with an anuric period lasting more than 3 weeks. Factors of acute functional failure of the kidneys can be called the following: infectious diseases; shock states; pathological processes; acute nephritis; glomerulonephritis – damage to the glomeruli (a functionally active unit of the kidney, which performs the function of filtering plasma). Acute functional failure of the kidneys is a frequent diagnosis; approximately 15–25 % of patients are admitted to the intensive care unit with a diagnosis of acute renal failure. If we talk about chronic kidney failure, it develops in older children and may be associated with a hereditary factor. A few years ago, ANN was fatal in 80 % of cases. Today, the fatality rate has decreased to 50 %.

CKD is the final phase of any progressive kidney disease. The main causes of CKD are:

1. Glomerulopathies: a) rapidly progressive GN; b) chronic GN; c) GN in rheumatic diseases; d) toxic glomerulopathies (also associated with taking drugs gold, D-penicillinamine); e) nephropathy in blood diseases, paraneoplastic; f) metabolic nephropathies (diabetic nephropathy, kidney amyloidosis).

2. Tubulointerstitial nephropathies: a) CKD; b) kidney tuberculosis; c) all variants of primary and secondary chronic IN.

3. Vascular diseases of the kidneys: a) hypertensive nephropathy (with essential hypertension (especially malignant); b) renal (polyarteritis nodosa, Wegener's granulomatosis and systemic scleroderma); c) ischemic kidney disease (stenosis, atherosclerotic or cholesterol embolism of renal arteries (bilateral or single functioning kidney).

4. Obstructive nephropathies: urolithiasis, hydronephrosis, retroperitoneal fibrosis, tumors of the kidneys, urinary tract and prostate gland.

5. Hereditary nephropathies and kidney abnormalities: Fanconi syndrome, Alport syndrome, polycystic kidney disease, kidney hypoplasia.

Classification of CKD

There is no generally accepted classification of CKD. Below is the classification CNN (Table 1) was approved by the Ministry of Health of Ukraine (2003).

Today, they are used in general clinical and nephrological practice defining all progressive kidney diseases as "***chronic kidney disease***" with the allocation of 5 stages according to the level of glomerular filtration and the characteristics of these stages according to according to the data of the US National Kidney Foundation (NKF) (*Table 1*).

Table 1

Classification of chronic renal failure

(order of the Ministry of Health of Ukraine dated 20.07.2003 No. 365)

Degrees of CNN	GFR level, ml/min	Creatinine level, mmol/l
1st	< 90...≥ 60	> 0.123...≤ 0.176
2nd	< 60...≥ 30	> 0.177...≤ 0.352
3rd	< 30...≥ 15	> 0.353...≤ 0.528
4th	< 15	> 0.528

Diagnostics

In patients with CKD, except for those receiving dialysis, the stage of the disease should be established based on the glomerular filtration rate and the level of albuminuria (*Table 2, Table 3*). Summary table 4 can be used to predict the course of CKD. It should be noted once again that the stages of CKD are determined according to GFR, not according to the blood creatinine level. This is due to the fact that the level of creatinine in the blood begins to increase when the GFR decreases by half from normal. That is, hypercreatininemia is observed in the presence of more than 50 % of non-functioning nephrons. Determination of GFR is performed with expected hyperfiltration or normal functions with the Roberg-Tareev test and radionuclide renoscintigraphy, in all in other cases, according to the GFR-EPI formula. For pediatric patients, the Schwartz formula is used. Other formulas today are recognized as incorrect and not are used.

$$SHKF = \frac{Factor \times height\ (cm)}{0.0113 \times blood\ creatinine\ (\mu\mu mol/l}, \text{ or}$$

$$SHKF = \frac{Factor \times height\ (cm)}{blood\ creatinine\ (mg/dL)}$$

Table 2

The value of the coefficient depending on the age of the patient

(D.D. Ivanov "Chronic kidney disease: diagnosis and treatment". Educational and methodological manual 2014)

In ik	***K*** for creatinine in mg/dL	***K*** for creatinine in μmol/l
< 1 year	0.33	29
> 1 year	0.45	40
2–12 years in	0.55	49
13–21 years old Boys	0.70	62
13–21 year old girls	0.55	49

Online GFR calculators, taking into account age and gender, is also relevant today https://medsoftpro.ru/kalkulyatory/skf-u-detej.html.

Diagnosis of CKD consists in the selection of risk groups, their screening, determination of the concentrating ability of the kidneys and GFR, albuminuria, and instrumental examination. Laboratory screening methods include clinical blood analysis, measurement of GFR using one of the calculation methods, assessment of the ratio of day and night diuresis, qualitative tests for the presence research methods are blood pressure monitoring, dynamic nephroscintigraphy, ultrasound of the kidneys, and Doppler examination of renal vessels. Normal indicators of GFR, depending on the method of calculation, which depend on the gender and age of the patients, are shown in the *table 3*.

Table 3

Normal values of CF (ml/min/day) in children and teenagers

Children	Unsts	Unsts
Newborns (premature)	25–91 μmol/l	(0.29–1.04 mg/dL)
Newborns (full term)	21–75 μmol/l	(0.24–0.85 mg/dL)
2–12 months	15–37 μmol/l	(0.17–0.42 mg/dL)
1–<3 years	21–36 μmol/l	(0.24–0.41 mg/dL)
3–<5 years	27–42 μmol/l	(0.31–0.47 mg/dL)
5–<7 years	28–52 μmol/l	(0.32–0.59 mg/dL)
7–<9 years	35–53 μmol/l	(0.40–0.60 mg/dL)
9–<11 years	34–65 μmol/l	(0.39–0.73 mg/dL)
11–<13 years	46–70 μmol/l	(0.53–0.79 mg/dL)
13–<15 years old	50–77 μmol/l	(0.57–0.87 mg/dL)

The main criteria for diagnosing CKD are:

– history of kidney damage;

– polyuria and nocturia;

– decrease in relative density and osmolarity of urine;

– decrease in kidney size according to ultrasound (or X-ray examination);

– laboratory data: anemia, GFR decrease (with daily diuresis no less 1.5 l – < 70 ml/min and lack of functional renal reserve; hyperphosphatemia, hypercalcemia, hyperuricemia);

– an increase in blood pressure.

The classification of CKD and CKD in children is given in the *table 4*.

Table 4

Stages of CKD and CKD for children and adolescents

CKD stage	CNN stage	SHKF, ml/min/1.73 m^2	Blood creatinine, mmol/l	Maximum relative density of urine
I	There is none	≥ 90	≤ 0.104	> 1018
II	And A (tubular)	≥ 90	≤ 0.104	≤1018
II	And B (compensated)	89–60	0.105–0.176	<1018
III	II (undercompensated)	59–30	0.177–0.351	
IV	III (uncompensated)	29–15	0.352–0.440	
V	IV (terminal or dialysis)	< 15	> 0.440	

Risk factors of CKD

Risk factors that have a possible influence on the development of CKD. A heavy family history of CKD, a decrease in the size and volume of the kidneys, low birth weight or prematurity, low material wealth or social status.

Risk factors provoking the development of CKD. Presence of type 1 and type 2 diabetes mellitus, hypertension, autoimmune diseases, urinary tract infections, urolithiasis, urinary tract obstruction, toxic effect of drugs.

Risk factors for CKD progression. High degree of proteinuria or hypertension, insufficient glycemic control, smoking and drug use.

Risk factors for end-stage CKD. Late start of renal replacement therapy, low dialysis dose, temporary vascular access, anemia, low blood albumin levels.

Indications for hospitalization are the presence of one or more pathological conditions or complaints:

– edema; – arterial hypertension above 140/90 mm Hg;

– intense pain in the abdomen or lower back;

– intoxication syndrome – vomiting, nausea, weakness, convulsions;

– decrease in the rate of diuresis;

– violation of the water–electrolyte and acid–alkaline state;

– complications of CKD – decompensation of chronic heart failure, uremic pericarditis, pneumonia, pleurisy, hemorrhagic syndrome, hyperkalemia, etc.;

– hemodialysis.

When collecting complaints, pay attention to the presence of arterial hypertension, weakness, convulsions.

Collection of disease anamnesis to accurately determine the time of onset of the disease:

– symptoms of the onset of the disease and subsequent course;

– therapy that was carried out at the pre-hospital stage from the moment of onset of the disease.

Collection of life anamnesis:

– obstetric anamnesis;

– diseases which transferred patient;

– previous ones cases treatment;

– nature of nutrition;

– establish which medical means accepts patient;

– allergy history;

– results previous ones laboratory and instrumental examinations patient (if such were conducted);

– detection factors risk that could lead to the development of CRF (congenital malformations bodies urinary system), glomerulonephritis, pyelonephritis, diabetes, hereditary kidney diseases;

– unfavorable premorbid background;

– presence companions chronic diseases;

– unfavorable social and household conditions;

– the patient's geographic area of residence.

Allergological history. Information on vaccination.

Family history (hereditary and congenital kidney diseases in family members, siblings)

Medical history (use of nephrotoxic drugs)

List of drugs that accepted patient

Suffered injuries, operatives interference

Diseases and habits (tobacco smoking, use of alcohol, toxic substances, drugs)

Clinical manifestations

Clinical manifestations of HNH can be:

1) specific signs of kidney disease, such as glomerulonephritis;

2) symptomatic signs of extrarenal diseases that led to the development of CKD.

In the case of primary kidney disease, clinical symptoms are determined precisely his typical picture. When detecting signs of kidney damage against the background of an extrarenal process, the following are most often characterized and determined by a nephrologist Manifestations of CKD:

– reduction of GFR;

– albuminuria/proteinuria;

– hypertension;

– anemia;

– bone-mineral disorders.

Hypertension is one of the most frequent manifestations of CKD. Hypertension can be the cause of CKD, and conversely, the presence of CKD always causes hypertension *de novo* due to the development of hyperactivity of the sympathetic system and erythropoietin deficiency anemia.

The second most frequent pathological manifestations of CKD after hypertension are anemia and metabolic disorders.

The initial symptoms of chronic renal failure are increased fatigue, nausea, loss of appetite, and an increase in the amount of urine excreted per day. In most cases, these symptoms are masked by manifestations of the underlying disease. The higher the degree of CKD, the more pronounced its clinical manifestations, and in some cases the symptoms of damage to the cardiovascular, in others – nervous, digestive, hematopoietic and other systems prevail. In some cases, the patient's well-being and condition do not correspond severity of renal failure ("silent uremia"). As CKD progresses, the symptoms of the underlying disease activity decrease, which can reduce the impact the main disease on the patient's survival on renal replacement therapy. With chronic GN of the 4th–5th century. In the CKD stage, proteinuria, manifestations of NS and edematous syndrome decrease, acute nephritic syndrome does not occur. It often decreases or the activity of rheumatic diseases, on the background of which nephropathy developed, completely disappears. In some patients with generalized amyloidosis and damage to the adrenal glands, in the case of the development of sylvrate kidney, with disseminated tuberculosis, progressive HNK with systolic dysfunction, the blood pressure level decreases.

Pathological changes in various organs and systems can also be attributed to non-specific manifestations of CKD:

− changes in the digestive organs, changes in the small and large intestines, an increase in the liver;

− damage to the cardiovascular system – most often arterial hypertension (AH), myocardial infarction, pericarditis;

− changes in the lungs – in the form of uremic lungs;

− hematological syndrome, anemia, hemorrhagic diathesis, toxic hyperleukocytosis;

− bone and joint changes, uremic osteodystrophy;

− violation of the function of endocrine organs in;

− damage to the nervous system;

− skin manifestations;

− violation of the electrolyte balance and acid-base state (KOS);

− infectious complications.

Treatment of CKD

There is no specific treatment for CRF, and its prospects are limited.

Regime. The development of CKD requires a change in the patient's lifestyle: hypothermia, significant physical and emotional stress should be avoided; the patient needs optimal working and living conditions, additional rest during work, a longer vacation is also advisable; in the case of pronounced worsening of the condition, intercurrent diseases occur; with stage II and III circulatory insufficiency, disorders of cerebral and coronary circulation, bleeding, bed rest is indicated; pregnancy and childbirth are contraindicated for women with chronic renal failure. Symptomatic treatment of CKD is carried out regardless of its cause with the goal stabilization of the process and includes conservative renoprotective therapy (diet therapy and drug treatment), and at the V stage – renal replacement therapy: hemodialysis, peritoneal dialysis, hemofiltration, as well as kidney transplantation.

The goal of conservative treatment is:

1) preservation of residual kidney function;

2) slowing down the rate of progression (blood pressure control, correction of anemia, prevention of progression of left ventricular hypertrophy, coronary and cerebral atherosclerosis, slowing down of the progression of hyperparathyroidism and renal osteodystrophy);

3) improvement of biochemical constants of the organism's biological environments;

4) elimination of the factor in, as well as can deepen uremic intoxication (water-salt disorders, disturbances of P-Ca metabolism, urinary tract obstructions, infections, injuries, surgical interventions, bleeding, taking nephrotoxic drugs).

Conservative reno-protective therapy is used in CKD III–IV stages at the pre-dialysis stage.

Low-calorie, low-protein, and low-phosphorus diet (MBD) with the use of keto analogs (KA) of essential amino acids (ketosteril), maintaining the adequacy of protein, phosphorus, potassium, and GFR. The effectiveness of the diet is observed as long as the creatinine clearance is >10–15 ml/min/1.73 m^2. MBD includes the use of proteins of high nutritional value (at least half: egg protein, animal and soy proteins). The appointment of MBD (0.6 g of protein/kg/day – 0.4 g/kg/day of animal origin, 0.2 g/kg/day of food plant protein) with a partial replacement of animal protein with highly purified soy protein and with a KA complex with a calculation of 0.1 g/kg/day allows to achieve a decrease in GFR by 0.8 ml/min/1.73 m^2.

The consumption of potassium and phosphorus depends both on the stage of CKD and on the physical activity and age of the patient.

Potassium intake. In connection with the increase in the content of K + in the blood with CKD, it is necessary to limit the use of products with its content. At Hyperkalemia requires medical correction:

– ion-exchange resins (resonium), consumption of products rich in potassium, 10.0 per 100 ml of water 3 times a day;

– 500 ml of 5 % glucose solution + 8 units of insulin IV, drip;

– calcium gluconate – 20–30 ml of 10 % IV;

– soda-buffer – 200 ml in/v.

With an increase in the level of K+ in the blood above 7.0 mmol/l, the risk of complications increases from the heart (extrasystole, atrioventricular block, asystole) and occurs the need for renal replacement therapy.

With chronic obstructive pulmonary disease of the 3rd–4th century. prescription of K+ drugs (asparkam, panangin, calypoz etc.) is contraindicated. When calculating when compiling the MBD menu to determine the amount of products of animal origin, a protein unit is used (equal to 7 g biologically complete protein).

Food products that, due to their low content of protein, potassium, and sodium, can be prescribed to patients with CKD without restrictions include: butter (butter), oil, margarine, unsalted lard, lard, protein-free bread, flour, semolina, sugar, honey, fruit caramel, coffee, tea. Improvement in the general condition of patients, decrease in the level of creatinine and urea in the blood occurs already 1 month after the start of MBD treatment and reaches a maximum after 6 months. Contraindications to a low-protein diet are absolute and relative. Absolute contraindications include: severe anorexia and incessant vomiting; pronounced catabolism; severe, uncorrected by medication, arterial hypertension; poor tolerance of dietary restrictions; sharply reduced residual kidney function (GFR less than 5 ml/min), especially with oliguria resistant to diuretic therapy; condition after surgery, severe infectious diseases, complications of CRF (pericarditis, pleurisy, etc.); urinary tract obstruction and inability to empty the bladder; abuse of alcohol, drugs, mental illness, domestic disorder.

CKD patients are screened for nutritional status disorders (stages 3–4). The treatment of this condition consists in eliminating the factor that causes it the progression of nutritional disorders and the achievement of CNN stabilization:

– correction of hyperglycemia (with diabetes);

– decrease in the activity of the process (in systemic connective tissue diseases, hepatitis, etc.);

– stop taking toxic drugs in;

– elimination of kidney and urinary tract obstruction and restoration of normal urodynamics;

– restoration of normal blood flow in renal arteries (atherosclerotic plaque, fibromuscular dysplasia, etc.);

– treatment of heart failure;

– use of high-energy nutrient mixtures: Renamine, Nutrien Nephro, Survimed Renal, Supro-760, Polyproten Nephro, etc. The peculiarity of their composition is highly purified soy protein, fats, fermented and non-fermented dietary fibers, selenium compounds, succinic acid, B vitamins, vitamin E, folate acid, biotin, nicotinamide, calcium pantothenate.

Table 5

Conservative treatment of chronic renal failure

Indicator	Target values	Medication correction
Blood pressure	Initiate therapy in patients with BP consistently above the 90th percentile for age, sex, and height For children with proteinuria, systolic and diastolic BP should be maintained at or below the 50th percentile for age, sex, and height, unless achieving these goals is limited by signs or symptoms of hypotension	Beginning of therapy 1. ACEI (Enalapril, Ramipril) or ARBs (Olmesartan, Valsartan). 2. Vasodilating β -blockers (Nebivolol). 3. Selective calcium blockers (Diltiazem, Felodipine, Lerkandi, etc.). 4. Blockers of imidazoline receptors (Moxonidin, Physiotens). 4. Diuretics (Torasemide, Xypogamma) * Taking drugs every 2–6 hours, depending on their amount
Hyperparathyroidism	**I–II CKD stage:** PTH – 35–70 pg/ml, Serum phosphorus – 0.87–1.48 mmol/l; **II 1st stage of CKD:** PTH – 70–110 pg/ml (7.7–12.1 pmol/l), Phosphorus – 0.87–1.48 mmol/l; **IV CKD stage:** PTH – 150–300 pg/ml and above (16.6–33.0 pg/ml), 1.13–1.78 mmol/l	For patients 3a–5d: 1. Limitation of phosphorus in food and. 2. Phosphate binders (Renagel). 3. Calcium preparations, active metabolites and synthetic analogues of vitamin D. (Sa D3 Nicomed, Alfa–D3TEVA, Tsinacalcet, Renvella) 4. Control of KLS – correction of acidosis 5. Densitometry
Growth retardation	Normal age values	With a growth deviation of –2 σ Rastan 0.6–1.0 units/kg/week
Albuminuria (proteinuria)	≤ 30 mg/day	BRA II_1, I ACE inhibitor, Diltiazem, Moxonidine

Indicator	Target values	Medication correction
Anemia	6 months–5 years Hb > 110 g/l, 5–12 years Hb > 115 g/l, 12–15 years Hb > 120 g/l Ht ≥ 33–37 %	Depending on the type of anemia – preparations of the elementary hall for, vitamin B12 or their combinations; Erythropoiesis-stimulating means of ESZ (Erythropoietin, Recormon)
Anticoagulant	Hematocrit Curing time Platelets RT TT ARTT Fibrinogen	Heparin 100–150 units/kg/day Dipyridamole 3–5 mg/kg/day
Diuretics	Preference is given to thiazide-like (indapamide) or loop drugs (Torasemide, Xypogamma).	It is worth noting that torasemide has a better cardiovascular risk profile compared to furosemide: the frequency of adverse reactions to torasemide is 14 times lower than to furosemide. However, in the terminal stage of CKD, when hyperkalemia occurs, the diuretic effect of furosemide is more pronounced, as it reduces the potassium content in the blood (!). The dose of indapamide is 0.625 or 1.25 mg/day for long-term use; xypamide – from 10 to 80 mg/day; torasemide – from 5 to 40 mg/day (for diabetic kidney disease); furosemide – from 10 to 50 mg/day. High doses of torasemide and furosemide in CRF are not prescribed because they cause resistance. In view of the potassium-sparing effect of ACE inhibitors/BRAs, the additional appointment of mineralocorticoid receptor antagonists to patients with CKD during diuretic therapy is impractical (!). Spironolactone and eplerenone in a dose of 25–50 mg/day are used with normal concentration of potassium in blood serum
Hyperkalemia	Classification of hyperkalemia: mild – 5.6–6.5 mmol/l; moderate – 6.6–7.5 mmol/l; severe >7.5 mmol/l.	Limit potassium intake (fruits, fruit juices, plant foods). IV 10–20 ml of 10 % calcium chloride solution In order to move potassium into the cells, enter 20–40 ml of 40 % glucose solution + 4–8 units of short-acting insulin. Loop diuretic in patients with preserved diuresis, eg, furosemide 20–40 mg IV, dose may be repeated every 6–8 h. Hemodialysis (rarely peritoneal dialysis) – in case of life-threatening hyperkalemia and in patients with severe renal failure
Correction of water exchange disorders	If the level of creatinine in the blood is 0.35–0.43 mmol/l, which corresponds to a glomerular filtration rate of 10–40 ml/min, and there are no signs of CHF, then you can take a sufficient amount of fluid and maintain diuresis at the level of 2–2.5 L/day (high diuresis contributes to the elimination of waste products from the body). In case of dehydration (polyuria, vomiting, diarrhea, leading to drowsiness, weakness, increased blood coagulation), intravenous administration of 3 liters of 5 % glucose solution per day under control of central venous pressure is recommended	

Indicator	Target values	Medication correction
Hypocalcemia/Hyper calcemia	Vitamin D preparations (alfacalcidol 0.25 mg/day 3 times a week) and/or calcium preparations. In the presence of convulsions, symptoms of Khvostek, Trousseau, calcium preparations are added (calcium acetate 0.5 g 3 times a day 1 hour before or 3 hours after meals); in case of inefficiency, calcium gluconate or calcium chloride is administered intravenously. / The dose is determined by the phosphorus level: with a phosphorus level of 1.454–2.4 mmol/l – 1 tablet 3 times a day during meals; 2.4–2.9 mmol/l – 2 tablets 3 times a day; with a phosphorus level > 2.9 mmol/l – 3 tablets 3 times a day. If, after 2 weeks, the phosphorus level is > 1.78 mmol/l, the dose of sevelamer is increased by 1 tablet after a meal, followed by control after 2 weeks	
Hyperphosphatemia	A diet with a restriction of the daily content of phosphorus in the food products of the daily ration to 800–1000 mg. However, for patients receiving dialysis, this is usually not enough, so hyperphosphatemia drugs are prescribed to correct hyperphosphatemia. All drugs for the treatment of hyperphosphatemia are taken with or immediately after food	
Acidosis	introduction of 4 % soda solution at the rate of 4 ml/kg of body weight IV (reduces acidosis), as well as the use of 10 ml of 10 % calcium gluconate solution	
Sorbents	As a supplement to a low-protein diet oxycellulose initially in a dose of 40 g, followed by an increase in the dose to 100 g/day; methylsilicic acid hydrogel 15 g/day; starch 35 g/day every day for 3 weeks; polyaldehyde 40–60 g/day; carbolene 30 g/day; enterosorbent 3 g/day	
Treatment of infectious complications	is carried out by using antibiotics taking into account nephrotoxicity (aminoglycosides – gentamicin, amikacin; cephalosporins – ceftriaxone have this property). Preference is given to macrolide antibiotics (azithromycin, clarithromycin, roxithromycin), semi-synthetic penicillins (oxacillin, methicillin), protected penicillins of a broad spectrum of action (amoxicillin)	
Extracorporeal methods of treatment:	hemodialysis individually 2–3 times a week; hemosorption – 3–5 procedures per course of treatment; peritoneal dialysis – introduction into the abdominal cavity and removal of special dialysis solutions through a dialysis catheter; hemofiltration, which allows you to remove about 20 liters of fluid from the body in 3–4 hours	
Kidney transplantation	not indicated in the case of kidney tuberculosis, malignant neoplasms, chronic purulent processes, significant defects of the urinary tract, as well as for persons with mental retardation	

ARB/ACEI is prescribed as renoprotection, regardless of the presence of elevated blood pressure!!!

IAPF

- Rami pril 5–10 mg
- Enalapril 10–20 mg

SCONCE

- Cardosal 10–40 / 12.5
- Irbesan 150–300 / 12.5

Dose-dependent effects of ACE inhibitors

- Antihypertensive = 0.2–0.4 mg/gc.
- Hypoproteinuric = 0.5–0.8 mn/kg.
- Antisclerotic = 0.9–2 mg/kg.

The BRIMONEL combination is used for the treatment of hypertension at various stages of CKD

BR (A) **I** (ACE) **MO** (Xonidine) **NE** (bivolol) **L** (ercadipine)

Table 6

Treatment patients with chronic renal failure depend of GRF

(Ivanov D.D. NPK "Actual issues of pediatric nephrourology", Kharkiv, 2018)

With GFR > 30 ml/min	**With GFR ≤ 30–15 ml/min**
○ IACE / BRA + Moxonidine ○ Nebivolol ○ Lercandipine ○ Diuretic	○ Moxonidine + Lercandipine ○ Nebivolol * Solving the issue of starting dialysis

Renoprotection: correction of nitrogenous impurities and MIA syndrome (Malnutrition – Inflammation – Atherosclerosis) in chronic renal failure (Stenvinkel P. et al., 2012)

1. Diet
2. + Ketosteril (1 tablet / 5 kg)
   + Lespenefril (Libera 1 t * 3 days)
3. Enteropassage (normaze, dufalak)
4. Enterosorption (polyphepan, enterosgel)
5. Correction of microflora

The main tasks of diet therapy of chronic obstructive pulmonary disease

- Reduction of nitrogen load
- Provision of essential amino acids
- Compensation of energy costs

Therapeutic nutrition according to the stage of chronic obstructive pulmonary disease

The nutrition of a child with CKD and CKD should be balanced and age-appropriate.

Consumption of animal protein should not be more than 20 g/day

With chronic obstructive pulmonary disease I–III art (predialysis) – restriction of consumption of only POTASSIUM containing products.

With chronic obstructive pulmonary disease IV art (dialysis) – there is no need to limit the consumption of any food, provided that there are adequate 3 dialysis sessions per week.

Indications for starting dialysis

Basic indications and contraindications for program hemodialysis

Indication:

- GFR < 15 ml/min/1.73 m^2;
- Urea content in blood plasma > 35 mmol/l;
- Creatinine content in blood plasma > 0.7 mmol/l;
- The content of "medium molecules" in the blood plasma is >1 U;
- Potassium content in blood plasma > 6.5 mmol/l;
- Reduction of standard bicarbonate in the blood > 20 mmol/l;
- Deficiency of buffer bases > 15 mmol/l ($B\sum$ < -10);
- Development of persistent oligoanuria (< 500 ml/day);

− The onset of pulmonary edema in the background of hyperhydration;
− Fibrinous or less often exudative pericarditis;
− Signs of increasing peripheral neuropathy;
− Edema of the brain;
− Malignant arterial hypertension with signs of congestive heart failure.

Absolute contraindications:

− HF with congestion in the large and small blood circulation regardless of kidney disease. Malignant hypertension and its consequences.
− Infectious diseases of any localization with an active inflammatory process.
− Oncological diseases of any localization in internal organs.
− Gastrointestinal ulcer in the active phase.
− Mental diseases with a negative attitude of patients to hemodialysis.
− Hemorrhagic syndrome of any origin.

Hemodialysis program

A standard dialysis program is three times a week for 4 hours.

The duration of treatment and/or its frequency should be increased in patients with hemodynamic instability and cardiovascular diseases.

Management of water balance is an extremely important parameter for preventing complications during GD in children.

Estimated dialyzer surface area in children

Child's age (years)	< 2	2	4	9	14	> 14
Surface area (m^2)	0.2	0.37	0.56	0.75	0.98	1.1–1.4

Anticoagulation therapy on hemodialysis and prevention of thrombosis

To prevent thrombosis of the extracorporeal system during hemodialysis, it is mandatory to use anticoagulants/antithrombotic substances.

In patients without an increased risk of bleeding, low-dose unfractionated heparin or low-molecular-weight heparin can be used.

Renal replacement therapy using the PD method

Currently, 4 main variants of chronic PD are used:

1) continuous ambulatory peritoneal dialysis (CAPD – continuous ambulatory peritoneal dialysis);

2) permanent hardware peritoneal dialysis (CCPD – continuous cycler-assisted peritoneal dialysis);

3) nocturnal intermittent peritoneal dialysis (NIPD – nocturnal intermittent peritoneal dialysis);

4) tidal peritoneal dialysis (TPD – tidal peritoneal dialysis).

CCPD, NIPD and TPD are carried out using special devices – cyclers in automatic mode (APD – automated peritoneal dialysis).

Manual (ambulatory) PD – is implemented as a variant of permanent ambulatory peritoneal dialysis (PAPD), consists of continuous PD cycles, while

several (2–4) cycles are performed during the day and one at night (dialysis cycle is called the elapsed time from the moment from the beginning of one peritoneal exchange to the beginning of the next).

Hardware PD (APD) is carried out with the help of a device (cycler) and is implemented in several variants:

− permanent hardware PD (PCPD) – continuous conduct of PD cycles, while several (3–5) cycles are carried out at night and one – during the day;

− nocturnal intermittent PD (NPPD) – conducting frequent (5–8) cycles at night, with a break during the day;

− tidal PD (PPD) – carrying out very frequent (24–30) cycles at night, with a break during the day. A feature of PPD is incomplete draining of the dialysate at each cycle and ensuring full continuity of the dialysate process during its implementation.

Indications for transferring a child from hemodialysis to peritoneal dialysis

− Progression of cardiovascular insufficiency.
− Poor individual tolerance of HD.
− The child's non-compliance with the water and salt regime.
− Low weight or young age of the child.
− Problems with vascular access.
− Sepsis, bacteremia.
− Uncorrectable hypertension.
− The distance of the patient's place of residence from the dialysis center
− The child's (parents', guardians') need for free movement
− Diabetes mellitus.

Indications for transfer of a child with PD to hemodialysis

− Permanent lack of possibility to achieve the target Kt/Vurea and CCr in the absence of medical, technical and psychosocial contraindications to GD.

− Inadequate solute transport or fluid removal. Patients with rapid peritoneal transport may have insufficient ultrafiltration and/or excessive loss of protein in the dialysate (a relative contraindication that is revealed after the start of treatment and the first test peritoneal balance – PET test).

− Severe uncontrolled hypertriglyceridemia.
− Unacceptably high frequency of peritonitis or other PD complications.
− Development of technical/mechanical problems.
− Cachexia resistant to active therapy (relative).

Contraindications for the choice of peritoneal dialysis

− Low transport characteristics of the "peritoneal membrane" (absolute contraindication).

− Documented loss of peritoneal membrane function (absolute contraindication).

− The presence of adhesion disease in the abdominal cavity, which limits the movement of dialysate (absolute contraindication).

− Severe chronic obstructive lung diseases (absolute contraindication).
− Fungal lesions of the abdominal cavity (absolute contraindication).
− Had surgical interventions accompanied by drainage of the abdominal cavity (absolute contraindication).
− Chronic peritonitis (absolute contraindication).
− Acute respiratory failure (absolute contraindication).
− Skin infections (absolute contraindication).
− Malignant neoplasms or uncorrected other mechanical defects in the abdominal cavity that cannot be corrected and make effective peritoneal dialysis impossible or increase the risk of infection (absolute contraindication).
− III century obesity (relative contraindication).
− Polycystic kidney disease (relative contraindication).
− Presence of enterostoma and/or urostoma (relative contraindication).
− Congenital or acquired anatomical changes of the abdominal wall (relative contraindication).
− Adynamic ileus (relative contraindication).
− Chronic respiratory failure (relative contraindication).
− Life-threatening hyperkalemia (relative contraindication).
− Pulmonary edema (relative contraindication).
− High level of catabolism (relative contraindication).
− Cachexia (relative contraindication).
− Lack of sufficient motivation and intelligence (relative limitations to independent implementation of PD methodology).
− Limitation of movements or vision (relative limitations to the independent implementation of the PD technique).
− Difficult social or sanitary and hygienic conditions (relative restrictions to independent implementation of PD methodology).

Tests

1. From urine of a 14-year-old boy with the exacerbation of secondary obstructive pyelonephritis Pseudomonas aeruginosa was isolated with a titer of 1000000 microbes per 1 ml. What antibiotic is the most advisable in this case?

A. Ciprofloxacin. *C. Cefazolinum.* *E. Chloramphenicol.*
B. Ampicillin. *D. Azithromycin.*

2. A 9-year-old girl complains of fever up to 37,5 °C, headache, inertness, weakness, loss of appetite, stomachache, and frequent painful urination. Provisional diagnosis of acute pyelonephritis is made. Clinical urine analysis: specific gravity – 1018, no protein, leukocytes – 10–15 in the vision field. What investigation method can verify the diagnosis of urinary system infection?

A. Rehberg test (crea-tinine clearance test).
B. Bacteriological inoculation of urine.
C. Zymnytsky test (mea-surement of daily diuresis).
D. Complete blood count.
E. Clinical urine analyses, dynamic testing.

3. A 13-year-oldgirl complains of fatigability, frequent headaches, cardialgia. Eight years ago she had a case of pyelonephritis. Urine analyses periodically revealed leukocyturia. The child has undergone no further treatment. On examination: increased BP up to 150/100 mm Hg. Ultrasound investigation revealed significant reduction of the right kidney. What process is leading in arterial hypertension pathogenesis in this case?

A. Disturbance of water-electrolytic balance
B. Disturbance of renal circulation
C. Hypersympathicotonia
D. Hyperactivity of renin-angiotensin system
E. Increased cortisol level

4. A 7-year-old boy has been an inpatient for 1.5 months. He had been delivered to the hospital with complaints of edemas all over his body, low urine output, and headache. Clinical urinalysis: proteins – 7.1 g/L, leukocytes – 1–2 in the vision field, erythrocytes – 3–4 in the vision field. During the course of treatment the edemas gradually dissipated, headache abated, diuresis normalized. Daily urine proteins – 3 g/L. Biochemical blood test: total protein – 43.2 g/L, urea – 5.2 mmol/L, cholesterol – 9.2 mmol/L. What glomerulonephritis syndrome is the most likely to be present in the patient?

A. Mixed *C. Isolated urinary* *E. Nephrotic*
B. Nephritic *D. Hematuric*

5. A 9-year-old girl complains of fever up to 38.5 °C, headache, inertness, weakness, loss of appetite, stomachache, and frequent painful urination. Provisional diagnosis of acute pyelonephritis is made. Clinical urine analysis: specific gravity – 1016, no protein, leukocytes – 10–15 in the vision field. What investigation method can verify the diagnosis of urinary tract infection?

A. Zymnytsky test (density measurement of daily diuresis)
B. Rehberg test (creatinine clearance test)
C. Bacteriological inoculation of urine
D. Complete blood count
E. Clinical urine analyses, dynamic testing

6. During regular examination of a 2-year-old boy, he presents with enlarged left kidney, painless on palpation. The right kidney was undetectable on palpation. Excretory urography shows no contrast on the right. Cytoscopy detected hemiatrophy of the urinary bladder trigone, the right ureteral orifice is not detected. What pathology is it?

A. Dystopia of the right kidney
B. Agenesis of the right kidney
C. Hypoplasia of the right kidney
D. Agenesis of the right ureter
E. Ectopic right ureteral orifice

7. An 8-year-old girl with complaints of painful urination, frequent low-volume urination, and leukocyturia was diagnosed with acute cystitis. 10 days before the disease onset she was treated by the gynecologist for acute vulvitis. 5 days ago she presented with mild catarrhal symptoms. Her mother ascribes the child's disease to her overexposure to cold. Specify the most likely infection route?

A. Descending *C. Hematogenic* *E. Lymphogenic*
B. Ascending *D. Contact*

Answers

1	2	3	4	5	6	7
A	*B*	*D*	*E*	*C*	*B*	*B*

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Навчальне видання

Невідкладна допомога дітям з захворюваннями нирок

Методичні вказівки
для здобувачів 2 рівня вищої освіти 5-го та 6-го курсу
вищих медичних закладів освіти III–IV рівнів акредитації

Упорядники Іщенко Тетяна Борписівна
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