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COURSE AND FEATURES OF ACUTE KIDNEY FAILURE IN CHILDREN

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ABSTRACT
The lecture describes the causes, clinical picture and treatment of acute renal failure in children.

Keywords: renal failure, children, treatment.

Kidney failure this is a violation of the homeostatic functions of the kidneys with the development of azotemia, changes in the balance of acids and bases, water-electrolyte balance, the development of anemia, osteopathy, hypertension and other manifestations due to the inability of the kidneys to perform basic functions. Functional (transient) PN is a transient condition of kidney function inferiority. Organic PN, in turn, can be acute and chronic. Both are based on pronounced changes in the structure of renal tissue and both are accompanied by persistent changes in homeostasis. Acute renal failure is a syndrome characterized by a sudden, rapid and significant drop in renal function, followed by a violation of homeostasis due to inadequate renal perfusion, arterial, venous obstruction, damage to renal cells or obstruction of urine outflow. CRF is an irreversible violation of the homeostatic functions of the kidneys associated with severe progressive renal disease, its final.
Acute renal failure - a nonspecific syndrome developing as a result of acute transient or irreversible loss of homeostatic kidney functions caused by hypoxia of the renal tissue, followed by predominant damage to the tubules and swelling of interstitial tissue, characterized by an acute violation of the homeostatic function of the kidneys due to their organic damage, which is manifested by a rapid and pronounced decrease in diuresis, nitrogen-releasing function, water-electrolyte and acid-base imbalance, it is accompanied by damage to virtually all organs and systems of the body. "Acute kidney injury" (AKI) is a term that supersedes the diagnosis of "acute kidney injury" in light of the need for earlier detection of dysfunction and provision of appropriate therapy. The term AKI can manifest itself both explicitly and subclinically, which is important for the short- and long-term prognosis of recovery of impaired renal functions. The term "OPN" has been preserved only for the most severe cases of acute kidney injury. The terms AKI and AKI are not synonymous, since the international consensus criteria for AKI are intended to describe three levels of renal failure (risk –

R, damage – 1, insufficiency - F) and two clinical outcomes (loss of renal function – L, terminal stage – E; Table 1).

Table. Criteria for stratification of severity of acute kidney injury according to RIFLE criteria (2002)

stage	Criteria for glomerular filtration	Criteria for diuresis
Risk	>Scr* by 1.5 times or <CF** by 25%	<0.5 ml/kg per hour>6h
harm	>Scr by 2 times or < CF at 50%	<0.5 ml/kg per hour >12h
Insufficiency	>Scr by 3 times or < CF by 75% or Scp>354 mmol/l with an increase of at least 44.2 mmol/l	<0.3 ml/kg per hour >24h or anuria > 12 h
Loss of renal function	Persistent acute renal failure, complete loss of renal function > 4 weeks	
Terminal renal failure	Terminal CPN > 3 months	

Stages of acute kidney injury (Table 2)

Table 2. Stages of acute kidney injury according to AK1N (2007)

Stages	Criteria based on serum creatinine level (Scr)	Criteria based on urine volume
1st	> Scr at >26 mmol/L, or from 150 to 200 % basic (1.5-2 times)	<0.5 ml/kg per hour for more than bh
2nd	> Scr by more than 200%, but less than 300% basic (more than 2, but less than 3 times)	<0.5 ml/kg per hour for more than 12h
3rd	> Scr by more than 300% of the base (more than 3 times) or Scr by >350 mmol/L with a rapid increase of more than 44 mmol/l	<<0.3 ml/kg for 24 hours or anuria for 12 hours

Since 2007, modified pediatric criteria (pRIFLE) have been adopted, which are necessary to determine the timing of the start of renal replacement therapy (Table. .3), and in 2012 – the criteria of the organization "Improving global outcomes of kidney disease treatment" (KDIGO) – from the English Kidney Disease: Improving Global Outcomes (Table.4).

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Table.3. Criteria for stratification of severity of acute kidney injury according to Profile (2007)

Stage	Criteria for glomerular filtration (CF)	Criteria for diuresis
Risk	Reduction of CF by 25%	<<0.5 ml/mg per hour x 8h
Damage	Reduction of CF by 50%	<0.5 ml/kg per hour x 16 h
Insufficiency	Reduction of CF by 75% or CF rate<30 ml/min/1.73 m2	<0.3 ml/kg per hour x 24 h or anuria for 12 h
Loss of renal function	Persistent acute renal failure; complete loss of renal function > 4 weeks	
failure	Terminal CPN > 3 months	

Classification. Depending on the level of exposure to the damaging factor leading to the development of a pathological condition, the causes of AKI are divided into prerenal, renal and postrenal.

Postrenal kidney damage is attributed to functional disorders, and renal – to organic. However, a functional disorder with a prolonged course (more than 24-48 hours) passes into the organic form of AKI, since during this time the renal parenchyma is damaged regardless of the initial factor that led to the development of renal failure.

According to the degree of preservation of diuresis, there are non-oliguric (without reducing diuresis) and oliguric forms of AKI. With neoliguric acute renal failure, diuresis remains normal due to a decrease in the reabsorption of water and sodium, an increase in azotemia occurs and GFR decreases. An objective assessment of diuresis is determined by the age of the patient. In the first 2 days. life diuresis is at least 0.5 – 1 ml / kg per hour (transient oliguria of newborns), increasing further to 4 ml / kg per hour, therefore, under true oliguria in children, a decrease in hourly diuresis of less than 0.5 ml / kg per hour should be assumed, and in newborns – less than 1 ml / kg per hour. Anarchy means the absence of urine for 24 hours.

Stage of Recovery

slow recovery of lost functions. The patient's condition gradually stabilizes, the water-electrolyte balance, nitrogen excretion function of the kidneys, blood CBS, erythropoietic function of the bone marrow normalizes. The symptoms of damage to the cardiovascular, digestive and other systems are eliminated. Low relative urine density (1002-1006) and a tendency to nocturia persist for a long time. Normalization of urinary sediment lasts from 6 to 28 months. The diagnosis of PPD is made on the basis of anamnesis, clinical picture, results of laboratory and other research methods.

Diagnosis of AKI, (APN) includes the identification of oligoanuria, determination of the nature of oliguria (physiological or pathological) and diagnosis of the disease that caused the development of AKI. Oliguria is one of the most important manifestations of impaired renal function, so it is necessary to carefully measure diuresis in a patient whose anamnesis makes it possible to suspect the development of AKI. In newborns with oligoanuria, it is initially necessary to exclude malformations of the urinary system. Ultrasound of the organs of the urinary system is used, which allows to exclude or confirm the presence of bilateral anomalies of the kidneys, ureters and various types of infra- and intravesicular obstruction. To diagnose the initial stage of AKI (i.e. renal ischemia), a Doppler study of renal blood flow is used. To exclude the presence of a posterior urethral valve and other types of urinary tract obstruction, miction cystourethrography is usually used in boys.

Detection of oligoanuria requires urgent determination of the level of creatinite, urea nitrogen and blood potassium in order to confirm or exclude AKI. These tests should be repeated daily. With organic acute renal failure, the concentration of creatinine in blood plasma increases by 45-140 mmol / (l x day). With functional oliguria, the creatinine level does not change or rises very slowly for several days.

Currently, the most promising early markers of AKI are lipocalin-2 and serum cystatin C. Lipocalin-2, associated with neutrophil gelatinase (NGAL), or siderocalin, is synthesized by many tissues, stimulated during inflammation and is a marker of acute kidney damage. Any excretion of NGAL into the urine occurs only when it is associated with damage to the proximal renal tubules, where the main lipocaine reabsorption takes place. Cysteine C, although it belongs to the group of markers of acute kidney injury, is not directly a marker of parenchymal damage, but reflects changes in GFR.

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