ISSN: 2776-0960 Volume 4, Issue 4 April, 2023

ResearchJet Journal of Analysis and Inventions
https://reserchjet.academiascience.org

COURSE AND FEATURES OF CHRONIC KIDNEY FAILURE IN CHILDREN

Bakhavadinova Zamira Mukhamattairovna
Senior Teacher Department of Pediatrics and Polyclinic Pediatrics of Children's Diseases of Andijan State Medical Institute

ABSTRACT

The paper reviews literary data on diagnostics and treatment of chronic renal failure (CRF) in children, highlights the importance of its conservative therapy early onset, as well as describes indications for dialysis

Keywords: Chronic renal failure, children, treatment.

Chronic renal failure is a symptom complex caused by irreversible death of nephrons in primary or secondary chronic kidney diseases and is characterized by an increase in creatinine above 2 mg% (0.17 mmol/L) and urea above 50 mg% (7 mmol/L) for more than 6 months [1, 2]. In 2002, K/DOQI (Kidney Disease) clinical practice guidelines were published. Outcomes Quality Initiative is a group of experts known as the Renal Disease Outcome Quality Initiative and created by the American National Kidney Foundation) on Chronic Kidney Disease (CKD), according to which kidney disease should be considered chronic if its signs are traced for 3 months or more [5]. Classification of stages of any chronic kidney disease based on glomerular filtration rate (GFR) is proposed (can not be used in children under 2 years old):

Stage 1 – kidney function is preserved (GFR ≥90 ml/min/1.73 m2);
Stage 2 – slight decrease in kidney function (GFR 60-89 ml/min/1.73 m2);
Stage 3 – moderate decrease in kidney function (GFR 30-59 ml/min/1.73 m2);
Stage 4 – severe damage to kidney function (GFR 15-29 ml/min/1.73 m2);
Stage 5 – terminal CRF (GFR <15 ml/min /1.73 m2).

According to this classification, the diagnosis of CRF it can be delivered in the 3rd stage [3, 4]. For a more objective assessment of GFR in chronic pathology of the urinary system, it is recommended to use the formulas for calculating GFR. In children, the Schwartz formula is used: With cr (ml/min / 1.73 m2) = k = L/Pcr, where k is a constant that changes with age, depending on gender; L is body length, cm; Pcr is the concentration of creatinine in blood plasma, mg%. The value of k for low–weight children in the first year of life is 0.33; for children of the first year of life with normal body weight – 0.45; for children of 2-12 years and girls –

ResearchJet Journal of Analysis and Inventions
https://reserchjet.academiascience.org

(13-21 years) - 0.55; for adolescent boys (13-21 years) – 0.70 (G.J.Schwarz et al., 1987). If the blood creatinine concentration is presented in mmol/l, the constant (k) for children under 12 years of age and adolescent girls is 48.4; for adolescent boys - 61.6 (the recalculation coefficient is taken into account: mg% 88 = mmol/l) [1].

Most of the existing data on the epidemiology of CRF in children are concentrated mainly at the stage of terminal CRF (eSRD) [6-9]. According to the register of the European Renal Association – European Dialysis and Transplant Association (ERA-EDTA), the annual incidence of eSRD in Europe was 7.1 in 1980-1984 and 9.9 per 1 million children in the next 15 years [10]. In the USA, approximately 14 new pediatric patients with eSRD appear per 1 million children per year [11]. In Russia in 2007, 538 children were registered for renal replacement therapy (RRT) or 16.1 per 1 million children [8], in 2009 – 706 children (30.4 per 1 million children).

Unlike adults, in whom chronic glomerulonephritis, diabetes mellitus and arterial hypertension (AH) are the predominant causes of CRF, children are characterized by congenital and hereditary kidney diseases, which, according to various authors, account for 60 to 70% of cases [3, 6, 9]. Among congenital anomalies the development of the urinary system to the development of CRF is most often caused by congenital obstructive uropathies, complicated microbial-inflammatory processes, reflux nephropathy and dysplasia of renal tissue. Among hereditary kidney diseases have a significant proportion of tubulopathies [13, 14]. The structure of the causes of CRF also depends on age: malformations of the urinary system are characteristic of the newborn period (agenesis or hypoplasia of the kidneys, polycystic, megaureter, cystic dysplasia); in infancy and pre-school age, the same spectrum of congenital diseases is observed, as well as the consequences of the transferred pathology (hemolytic-uremic syndrome, renal vein thrombosis, etc.); in preschool and acquired kidney diseases (chronic subacute glomerulonephritis, nephritis in SLE, interstitial nephritis, etc.) play an increasingly important role at school age.

From a certain point on, the mechanisms of progression of kidney damage are the same in any disease, whether it is primary glomerular lesion, tubulointerstitial process or congenital dysplastic changes in parenchyma. With the loss of a significant part of the active nephrons, compensatory changes in hemodynamics occur in the remaining ones: expansion of afferent and less pronounced expansion of efferent arterioles, which leads to hyperperfusion, increased

ResearchJet Journal of Analysis and Inventions
https://reserchjet.academiascience.org

hydrostatic pressure in the glomerular capillaries and hyperfiltration. As a result there is remodeling of the vascular wall of arterioles, aggravation of intraclubular hypertension, violation of the integrity of the glomerular basement membrane, leakage of ultrafiltrate into the mesangium, damage to podocytes and tubular epithelium. Endothelial and mesangial cells, podocytes, tubular epithelium in response to damaging factors are able to produce substances with proinflammatory and prosclerotic activity (cytokines, growth factors), closing a vicious circle. The result of the pathological process is nephrosclerosis – a morphological substrate CPN, regardless of its root cause. In general, congenital anomalies are characterized by a slower progression to eSRD compared to acquired glomerulopathies.

Characteristics of chronic renal failure (according to V.I. Naumova et al., 1991)			
Stages and degrees of CPN	Symptoms of CRF in		International equivalent of CPN stage terms
	glomerulopathy	tubulointerstitial kidney diseases	
I. Tubular renal failure			
The creatinine content in the blood is within the normal range			
II.Total renal failure			
I at a blood creatinine concentration of 0.17-0.44 mmol/l	Hypertension, hemorrhagic syndrome, acidosis, restriction of GFR and tubular functions	Osteopathy, anemia, acidosis, restriction of GFR and tubular functions	Too
II at a blood creatinine concentration of 0.44-0.88 mmol/l	Also; damage to internal organs	Also, internal organ damage, hemorrhagic syndrome	Renal failure; polyuric stage
III at a creatinine concentration in the blood above 0.88 mmol/l	Symptoms of uremia, regardless of etiology	Symptoms of uremia, regardless of etiology	End Stage Renal Disease; oligoanuric stage

There are many classifications of CRF based on different principles – clinical manifestations, GFR level, blood urea. The most successful seems to be the division of CRF depending on the level of serum creatinine. Moreover, the 3rd, 4th and 5th stages of CKD correspond to the 1st, 2nd and 3rd stages of CKD according to this classification.

ResearchJet Journal of Analysis and Inventions
https://reserchjet.academiascience.org

Frequency and severity of clinical symptoms CRF varies depending on the underlying disease. Before the increase in blood creatinine, as a rule, there are already violations in the composition of the internal environment of the body caused by damage to the tubular functions of the kidneys – acidosis, hypokalemia, anemia, hypocalcemia, in combination with isostenuria, sometimes osteopathy. This stage is called tubular insufficiency, characterized, along with the above symptoms, by a small transient increase in blood urea. Tubular insufficiency for the longest time It is clearly traced in time and in tubulo-interstitial diseases, but can also be isolated in most cases of glomerulonephritis. Total CRF is characterized by an increase in not only urea, but also creatinine, the severity of hypocalcemia, anemia, metabolic acidosis increases, hyperkalemia is possible, hypertension is noted, systemic organ damage. The development of CRF in infancy is accompanied by pronounced metabolic disorders, since the metabolism in children of the first year of life at 5 times higher than that of teenagers. Clinically, this is manifested by anorexia, vomiting, metabolic acidosis, rapid development of clinical manifestations of renal osteodystrophy.

Treatment of CRF before the transfer to RRT is reduced to the symptomatic correction of individual clinical laboratory syndromes. The most common and serious of them in children is protein–energy deficiency - a condition when the intake of protein and calories from food does not cover the needs of the body due to a decrease in appetite and dyspeptic phenomena developing due to intoxication, acidosis, anemia. Protein-energy deficiency has a distinct negative impact on the physical and psychomotor development of the child, aggravates the course of nephropathy, brings the timing of the start of RRT, reduces survival on dialysis, increases the risk of transplantation. Therefore, rational nutrition is the basis for the management of a patient with reduced kidney function. Currently, it is not recommended to reduce protein intake below 1.5 g/kg/day in young children age and less than 0.8 g /kg / day in adolescents with predialysis CRF. Moreover, at least 70% of the protein should be of animal origin. The energy value of the diet (120-42 kcal / kg / day, depending on age) is provided with sufficient intake of carbohydrates and fats. Preference is given to vegetable fats with a high content of polyunsaturated fatty acids ω-6 and ω-3, which contribute to increased bile separation, increased cholesterol excretion from the body. All patients with CRF who are on a protein-restricted diet are shown the appointment of ketoanalogs essential amino acids (ketosteril) at the rate of 1 tablet per 5 kg of body weight per day in 3-4 doses with meals. In the presence of edema and

ResearchJet Journal of Analysis and Inventions
https://reserchjet.academiascience.org

hypertension, a strict restriction in the diet of table salt is necessary. In the absence of edema and hypertension in patients with stable hyperazotemia in order to improve kidney function, salt is given in doses (1-2 g / day) [1]. This amount can be provided with specially salted products (soaked herring, pickled cucumber). If the normal level of potassium concentration in the blood serum is exceeded, potassium-rich foods (dried fruits, citrus fruits) are limited, potatoes in uniform, greens, juices, nuts, cocoa, oat flakes, etc.).

Thus, the development of CRF in children differs significantly from that in adults. Complications and lesions of organs and systems in a growing organism occur earlier and are more pronounced. Although pediatric patients with eSRD make up only a small part of the total number of patients with uremia, they place high demands on the healthcare system due to the need not only to correct primary renal disorders, but also to ensure normal growth, development and social adaptation.

REFERENCES

1. Баранов А.А., Сергеева Т.В. Амбулаторная нефрология. Амбулаторная педиатрия. М.: Союз педиатров России. 2009; 1: 156 .

2. Наумова В.И., Папаян А.В. Почечная недостаточность у детей. Л.: Медицина, 1991; 288 .

3. Иванов Д.Д. Хроническая болезнь почек и хроническая почечная недостаточность у детей. Нефрология. 2006; 10: 3: 100–26.

4. Смирнов А.В., Есаян А.М., Каюков И.Г., Кучер А.Г. Концепция хронической болезни почек в педиатрии. Нефрология , 2005, Т.7, № 3, с. 7 –11.

5. K/DOQI Clinical Practice Guidelines for Chronic Kidney Disease Evaluation Classification Stratification. AJKD 2002; 39: 2: Supp.1: 1–266.

6. Байко С.В., Сукало С.В. Эпидемиология почечной недостаточности у детей в Республике Беларусь. Нефрология и диализ. 2009; 11: 4: 370.

7. Молчанова Е.А., Валов А.Л., Каабак М.М. Первые результаты формирования Российского регистра хронической почечной недостаточности у детей. Нефрология и диализ. 2003; 5: 64–68.