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MODERN METHODS FOR ASSESSING THE COURSE, TREATMENT, AND PROGNOSIS OF CHRONIC RENAL FAILURE IN CHILDREN

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Abstract: Chronic renal failure (CRF) is a non-specific syndrome that develops in hereditary, congenital, and acquired kidney diseases due to the progressive death of nephrons and stroma with a steady decrease in the ability of the kidneys to perform homeostatic functions. More than 50 diseases are known, that end with CRF with different frequencies and speeds (14). The true social significance of diseases for society is currently underestimated. Meanwhile, erased and latent forms of nephropathies are increasingly common in childhood, diagnosed late and early leading to the formation of chronic kidney disease (CKD) and CRF (15,13). The origins of CKD in the adult population often also go back to childhood.

The urgency of the problem.

According to the literature, the incidence of ESRD is increasing in all countries (12,10). In the next 10 years, the number of such patients is expected to double (15). In this regard, the mechanisms of the progression of kidney diseases and the possibilities of renoprotective therapy are being intensively studied all over the world. Unfortunately, modern therapy for kidney diseases is not effective enough and often causes severe side effects (2,15). Meanwhile, a number of interesting publications have appeared that present data on a new strategy for renoprotective therapy in advanced kidney disease in adults and children, which will be used in the 21st century.

Research result. Dynamic observations over 6 years of 70 children who underwent nephropathy in the neonatal period showed that most of them develop interstitial nephritis (IN), neurogenic bladder dysfunction or metabolic nephropathy and pyelonephritis against the background of ongoing rehabilitation measures. Complete recovery was noted only in 15% of patients (15).

The increasing frequency of chronic renal failure is the result of:

1) The current lack of effective measures for the primary prevention of kidney disease leading to CRF;

2) Insufficient effectiveness of "etiological" and "pathogenetic" therapy of kidney diseases, which leads to an increase in chronicity with the subsequent development of CRF;

3) Insufficient effectiveness of rehabilitation measures, as a result of which renal failure progresses with the formation of CRF.

4) An increase in the frequency of congenital and hereditary nephropathies (12.15). The problem of CRF in childhood is closely related to the problem of hereditary and congenital nephropathies. CRF in children was a consequence of congenital and hereditary nephropathy in 41.4%, glomerulonephritis (GN) - 40.5%, secondary pyelonephritis - 15.3%, other causes - 2.7% (18). Not the degree of nephrosclerosis, but the congenital inferiority of the nephron determines the development of renal failure in dysplasia in children. (fourteen).

There are certain geographical and ethical differences in the structure of the causes

of CRF and in some countries hereditary nephropathy is more important, in others congenital malformations complicated by pyelonephritis, in others, the proportion of urolithiasis in the structure of CRF formation increases(23). Therefore, the study of the epidemiology of CRF in both the adult and pediatric populations in our climatic, geographical, and ethical conditions continues to remain an independent problem, which is extremely necessary when determining the amount of medical care for this group of patients.

Usually, CRF is the end of a long-term pathological process, but sometimes in a short time (for example, within six months with malignant nephritis), sclerosis of a large part of the glomeruli and tubular atrophy develops. There are two forms of CKD:

1) General, or total CRF, developing as a result of nephrogenic sclerosis, which is observed in chronic glomerulonephritis (CGN);

2) Predominantly tubular chronic renal failure, which occurs as a result of diseases with initial and predominant damage to the tubules and interstitium, which is observed with nephronophthisis, pyelonephritis, interstitial nephritis, followed by glomerular sclerosis (1).

In the periodization of CRF, the following stages are distinguished: (5)

Stage I (compensated) - glomerular filtration 70-50 ml/min × 1.73 m2, plasma creatinine 0.088-0.265 mmol / l, the number of nephrons 50-25%. There are no subjective complaints.

Stage II (subcompensated) - glomerular filtration 50-30 ml / min × 1.73 m2, plasma creatinine 0.120-0.530 mmol / l, the number of nephrons is less than 30%. Reduced ability of the kidneys to concentrate urine, polyuria and polydipsia. With the deterioration of kidney function, anorexia, symptoms of impaired activity of the gastrointestinal tract, the first manifestations of anemia, metabolic acidosis appear;

Stage III (decompensated) - glomerular filtration 30-10 ml/min × 1.73 m2, plasma creatinine 0.450-0.800 mmol/min, the number of nephrons is less than 10%. The volume of urine decreases, distinct clinical signs of renal failure.

IV stage (terminal or uremia) - glomerular filtration less than 10 ml/min × 1.73 m2, plasma creatinine 0.620-1.110 mmol / l, the number of nephrons is less than 5%. Oliguria, is the defeat of all organs and systems.

The main attention of researchers is drawn to the end stage of CRF, primarily in connection with chronic hemodialysis therapy (32). Meanwhile, new innovations in the field of Renault protection aim at the need to refocus attention on the early stages of renal failure, when there are still opportunities to slow/stop the progression of renal failure (RF), and even restore lost functions (12), delay the onset of renal replacement therapy (RT). Early renoprotective therapy of chronic kidney diseases is a very urgent problem of modern clinical nephrology, since chronic renal failure (CRF) is an inevitable outcome.

It is proposed to ascertain chronic renal failure in children with kidney diseases if they have a decrease in creatinine clearance (Ccr) <20 ml / min. × 1.73 m2 of body surface, an increase in the content of urea in blood serum >35 mg%, and creatinine >2 mg% (>176 μmol / l).

As patients are dynamically monitored, there is a need for laboratory determination of parameters that could suggest the rate of progression of the renal process. The kidneys have a significant reserve capacity and an increase in serum creatine (Pcr) is observed when more than 50% of active nephrons are lost (22). Only from the moment Pcr can serve as a "marker" of the rate of progression of CRF (18). To date, there are no clear criteria by which one can judge whether a patient has the initial stages of renal failure. In congenital and hereditary diseases, in various nephropathies with predominant tubulointerstitial extraction, a decrease in the functional state of the kidneys develops along the tubular pathway, when the Pcr level is within normal fluctuations (18.9).

Such drugs as glucocorticoids and cytostatics are relevant in the onset of GN or in relapses, removing immunoinflammatory activity, they have a renoprotective effect.

However, with a prolonged course of the disease, other, "non-immune" mechanisms begin to predominate: hypertension and glomerular hyperperfusion, aldosterone, angiotensin II, proteinuria (nephrotoxins), hyperuremia, etc. The leading mechanism of action of systemic arterial hypertension are changes in renal hemodynamics: the development of intraglomerular hypertension, hyperfiltration and dysfunction of the modular blood flow (5). Under these conditions, the use of ACE inhibitors, reducing the level of circulating angiotensin II, relieving spasm of the afferent and efferent arterioles, eliminating intraglomerular hypertension, and hyperfiltration. In addition, the decrease under the influence of ACE inhibitors of ANGII circulating in the blood helps to reduce the formation of antidiuretic hormone (ADH) and aldosterone, which have an undoubted nephrosclerogenic effect (12,17). Studies with oral protein loading have shown that high protein intake leads to increased renal hemodynamics, GFR, intraglomerular hypertension and hyperperfusion, which are important factors in the development of glomerulosclerosis (2,12). In a clinical setting, to detect intraglomerular hypertension, a functional renal reserve (FPR) is determined. The degree of preservation of the FPR is determined using a single protein load at the rate of 1 g of protein per 1 kg of the child's body weight. To achieve the high rate of diuresis required for measuring Ccr, it must be combined with a water load of 20 ml per 1 kg of body weight for 30 minutes. An hour before the load, the basal CF is determined and, within an hour after the load, the stimulated CF is determined. RFR calculation is carried out according to the formula: RFR = (GFR-GFR/ GFR) x100, GFR and GFR - basal and stimulated glomerular filtration rate for endogenous creatine (15.17).

ACE inhibitors and ANGII receptor blockers proved to be effective and safe for long-term use of antihypertensive drugs to eliminate intraglomerular hyperfiltration in monotherapy and/or in combination. In everyday practice, a two-fold reduction in proteinuria is considered a guideline for increasing the dose of ACE inhibitors, regardless of the level of A/D achieved (9). The severity of reduction in proteinuria during ACE inhibitor therapy was inversely proportional to the rate of GFR decline (17). Activation of lipid peroxidation (LPO) in the membranes of epithelial cell structures, leading to the loss of their functional activity, changes in the composition of phospholipid fractions is an important mechanism for the development and progression of renal pathology and determines the degree of uremic intoxication in chronic renal failure (6). Therefore, the use of ACE inhibitors does not preclude the use of antioxidants and membrane protectors (12).

When using a double block of the renin-angiotensin-aldosterone system (RAAS) in children with CKD, with the use of ACE inhibitors in patients with a GFR range of 80-60 ml / min × 1.73 m2 after 4-5 years of continuous use of ACE inhibitors in doses that provide physiological hypotension , with initial proteinuria <1.0 g/day, it was possible to stop the progression of PI and increase GFR by 1-2 ml/min/year. Factors that promote and do not contribute to renoprotection have been established (Table 1). ACE inhibitors and sartans with a predominantly renal route at low GFR, and especially in the metabolic syndrome, may exacerbate renal impairment (12). Therefore, in patients with reduced renal function, ACE inhibitor ARBs with a predominantly extrarenal route of elimination (moexipril, monopril, telmisartan, eprosartan) should be used. Renoprotective drug therapy with ACE inhibitors and sartans in CKD should not be interrupted in the absence of primary disease activity.

Possibilities of renoprotection in the progressive course of CKD

Helps restore GFR	Does NOT help restore GFR
GFR over 60ml/min	Rate less than 60 ml/min
Therapy with ACE inhibitors, sartan, mocosidin	Therapy with CCB, beta-blockers, diuretic
Double (ACE inhibitor + sartans) and triple block of the reninangiotensin system (ACE inhibitor + sartan + moxonidine)	ACE inhibitor monotherapy, sartan or moxonidone
Therapy with ACE inhibitors and sartan with extrarenal excretion (fosinopril, eprosartan, telmisartan)	Therapy with ACE inhibitors, sartan with a predominantly renal route of excretion (perindopril, lisinopril, candesartan) course treatment
Lifelong treatment	Not less than 120g/l
Absence of anemia	Presence of proteinuria
Absence of microalbuminuria	Presence of hypertension
BP less than 120/75 mmHg	Smoking
no smoking	Low body weight
Normalization or increase in body weight	The presence of diabetes
Incomplete metabolic syndrome	Hypoalbuminemia
Albumin in the blood more than 35g/l	Male gender
Female before perimenopause	adult age
Children's period	Using self-healing elements
Permanent contact	

The progression of nephropathies and the possibility of renoprotection, a problem that has been recently studied, is promising in future studies of pediatric nephrologists, since many nephropathies, having begun in childhood, continue to progress in adolescents and adults (1,15). The morphological substrate of CRF in CGN is nephrosclerosis, which is the outcome of various chronic kidney diseases (13.5). The type of CRF formation in acquired, congenital, and hereditary nephropathies is different. With dysplasia, the development of renal failure is determined not by the degree of nephrosclerosis, but by the congenital inferiority of nephrons. In childhood, it is renal dysplasia, including polycystic disease, is the predominant cause of CRF (8). In many tubulointerstitial congenital and hereditary diseases, CRF slowly but surely progresses in the absence of bacterial overlay, in the absence of bacterial overlay, and in the absence of an active renal process. The renoprotective treatment essentially begins with the treatment of the etiological factor, with the treatment of the primary disease, where this is known. Unfortunately, the results of "etiological" and "pathogenetic" treatment of most CKD

remain unsatisfactory (9,12) and often end with the formation of CRF. An increase in serum creatine over 0.528 mmol/l (6 mg%) and a decrease in clearance of about 10 ml/ min x 1.73 m2 90.17 ml/sec0 is a contraindication for the use of all forms of immunosuppressive therapy for GN. However, since the rate of progression of CRF accelerates with the preservation or increase in the activity of the inflammatory process in the kidneys in GN and PN, the etiological and pathogenetic treatment of the primary disease is at the same time a measure aimed at inhibiting the progression of renal failure (13). It is known that the presence of nephrotic syndrome, the tubolointerstitial component, the use of glucocorticoids and heparin in patients with GN contribute to the development of hypocalcemia, demineralimzation of bones and the formation of renal osteopathies even before the onset of signs of CRF (18). In this regard, it is considered appropriate to prescribe ergocalciferol or its metabolites in renal osteopathic in patients with chronic renal failure, as well as in the functionally compensated stage of GN, receiving long-term corticosteroids, heparin, diuretics. The volume of conservative measures in children with chronic renal failure is determined by the underlying disease, stage of renal failure, damage to other organs and systems. Great importance in the treatment of chronic renal failure is given to dietary measures: A gross mistake is a sharp restriction (sometimes to complete exclusion) of animal protein from the patient's diet due to the presence of nephropathy with preserved kidney function. An indication for limiting protein in food is a decrease in the level of protein in food is a decrease in the level of CF< 30ml / min x 1.73m2, which corresponds to the III stage of CRF; - up to 70% of the protein in the diet should be of animal origin (meat, fish, milk). Each gram of protein lost in the urine must be replaced by 1.25-1.65g of protein in the diet (2).

Patients with CRF have prescribed 1-2 standard tablets of multivitamins daily. Diet therapy for CRF, starting from the early stages, is important in almost all pathogenetic and clinical syndromes and should be modified depending on the predominant syndrome. For diet therapy of children. Patients with CRF are recommended 3 diet options: without protein restriction - with azotemia not exceeding 52 mmol / l (60-80 mg%) of urea nitrogen; a diet with moderate protein restriction (1.0-1.5 g / kg/day), with a urea level of 57-71.4 mmol / l; a diet with a sharp restriction of protein (up to 0.6-0.7 g / kg/ day) with severe azotemia. The appointment of drugs capable of fixing toxins (sorbents) on their surface is required in stage III CRF (activated charcoal). Activated charcoal should not be administered in the presence of an ulcer and/or gastrointestinal bleeding. Assign at a dose of 1g / kg/day. For 2 weeks to 10 months divided into 3-4 single servings. These are taken 1.5 hours before meals and washed down with 100 ml of water. At the same time, other medicines are taken no earlier than 1 hour after taking the sorbent.

Findings.

Dispensary observation of a patient with chronic renal failure is a complex set of benefits, individually selected taking into account the stage of other organs, and carried out systematically. for life. Therefore, from the moment of ascertaining the already initial stage of renal failure, the task of actively involving the sick child and his relatives in the appropriate treatment regimen becomes important, conservative measures have been and remain the basis for the treatment of chronic renal failure. Thus, the basis for the prevention of the progression of renal failure in the early phase of CRF in recent years has been the use of ACE inhibitors (Capoten, Enap) in monotherapy and in combination with ANG II drugs that reduce arterial hypertension, which reduce arterial hypertension, glomerular hyperfiltration, reduces the level of proteinuria and thereby providing a nephroprotective effect, preventing complications from the cardiovascular system and the central nervous system (7.12).

Used literature.

1.Azimzhanova M.M., Kurban D.D., Mukhamedzhanova D.K. Perinatal, neonatal morbidity and mortality and ways to reduce them in the Republic of Uzbekistan.// Pediatrics-vol. -2000-N 2-3 C 24-26

2.Evseenko D.A., Tsiryulnikov N.I. Morphological changes in the placenta in complicated pregnancy and the state of health of newborns // Pediatrics. 2000.-№ 3. P.11-14.

3.Ignatova M.S. Nephropathy in children: modern genetic aspects // Ross, West Perinat and Pediatrician 2004.-N2-C.44-51.

4.IshkabulovaG.D., KholmuradovaZ.E.Phospholipid structure and the state of lipid peroxidation of erythrocyte membranes in newborns from mothers with preeclampsia associated with chronic pyelonephritis. // Journal of Biomedicine and Practice .2022.-No. 3 -C71-77.

5.Kadyrova G.G. The prevalence of chronic pyelonephritis among women of childbearing age: Aptoref. diss... candidate of medical sciences. - Tashkent, 2000. - 17 p.

6.Kolb V.G., Kamyshnikov V.S. Clinical research methods. // Minsk. - 1976.-311 p.

7.Krylov V.I., Vinogradov A.F., Eremeeva S.I. and others. The method of thin-layer chromatography of lipids of erythrocyte membranes // Laboratory business -1975.-№4 - pp. 205-206.

8.Kulakon V.I., Gurtovoi B.L., Ankirskaya A.S., Antonov A.G. Actual problems of antimicrobial therapy and infection prevention in obstetrics, gynecology and neonatology //Obstetrics and gynecology.-2004.-1C3-6

9.Nurullaen R.B. Urinary tract infections eschemiology and risk factors // Bull. Association of Physicians of Uzbekistan.-2003.-N1.-S.88-94.

10.Savelyeva G.M. Panina OB, Sichinava LG et al. The prenatal period and its significance in the development of the fetus to the newborn //Obstetrics and Gynecology-2004.-No2. -FROM. 60-62

11.Tugusheva F.A. Processes of lipid peroxidation and the protective role of the antioxidant system in the norm and in patients with chronic glomerulonephritis Part 1 // Nephrology.-2001. - Volume 5. -№1.-From 19.27

12.Tokona 3.3., Frolova O.G. Maternal mortality in preeclampsia./Akush.ginekol-1998 No. 5.-S.9 11.

13.Rasud-Zale Yu.G., Bornikov V.T., Gafarov F.B. and Other Features of the course and outcomes of pregnancy and childbirth in women with severe preeclampsia in reproductive history // Zhurn. theoretical and clinical honey. (uzb) -2002 N 6 C 61-64

14.Mazur L.I. Prevention of lesions of the reproductive system in girls/girls with pyelonephritis Nephrological readings (selected issues of clinical nephrology of childhood). Samara -2000 - c 77-85.

15.Shabalov N.P. / Neonatology, I volume. SPb. Special Literature. - 1997. - 494 p.

16.Shcherbavskaya E.A., Geltser B.I. Violation of mineral metabolism and bone tissue formation in a newborn fetus with pregnancy complicated by preeclampsia // Ross West. perinatal, and pediatrics.-2004. - Volume 49, N1-C.10-15

17.Ishkabulova G.D., Kholmuradova Z.E. Functional state of the kidneys in Newborn born From Mothers With Pre-Eklampsia // World Bulletin of Public Health (WBPH).-2022 Semtember, -c75-78

18.Comparative assessment on the effect of different methods of corrective therapy on lipid metabolism and homeostatic renal function.IshkabulovaG.J.1, KhaidarovaK.R.1, Kudratova G.N.1, Kholmuradova Z.E. Department of Pediatrics, Faculty of Therapeutics, Samarkand State Medical Institute Том: 7 Номер: 3 Год: 2020 Страницы: 2794-2800