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Aspects of early detection and prevention of essential micronutrient deficiency at different levels of chronic kidney disease

Radjabov N.M.
Bukhara State Medical Institute, Bukhara, Uzbekistan

Abstract: Chronic kidney disease (CKD) is an important public health problem. As kidney function declines, patients with CKD gradually develop end-stage kidney disease and must undergo dialysis or kidney transplantation to maintain their lives, placing a heavy economic burden on families and society. Therefore, it is necessary to effectively prevent and delay the progression of CKD. Essential micronutrients play an indispensable role in CKD, and the purpose of this study is to systematically review their benefits in the disease and summarize the risks of their excess.

Keywords. Chronic kidney disease, obesity, cardiovascular disease, cooper, ferrum, zinc.

Introduction. The current global prevalence of chronic kidney disease (CKD) is estimated at 13% and is rising rapidly amid rising obesity, diabetes, and hypertension [1,5,9,11,15]. Current international guidelines define this condition as decreased renal function, manifested by a glomerular filtration rate (GFR) less than 60 ml/min/1.73 m 2 and/or markers of renal damage, lasting at least 3 months, regardless of the underlying cause. The classification of CKD consists of five stages based on the decline in GFR from early disease to kidney failure [2, 6, 7, 9]. The causes of CKD are varied. Diabetes and hypertension are leading causes in middle- and high-income countries. Herbal use may cause kidney toxicity. Environmental pollution with heavy metals and organic compounds is also one of the causes of CKD [4,8,11,17,19]. CKD has become a major public health disease worldwide, with a significant negative impact on mortality and quality of life that increases with age. WHO global health estimates that in 2012, 864,226 deaths were attributed to CKD, ranking the disease as the fourteenth leading cause of death, accounting for 12.2 deaths per 100,000 people. The Global Health Observatory predicts that the mortality rate from CKD will continue to rise, reaching 14 per 100,000 by 2030 [2,7,9,14,15,20,21,22,23,24.25.26,27,28,29].

In recent years, the existence of an organic link between albuminuria and ultrastructural and functional disorders of podocytes has been confirmed in a number of experimental and clinical studies. [6,7,17,18]. It has been shown that these changes occur long before the appearance of microalbuminuria. [4,8]. The data

obtained confirmed that podocytes were involved in the processes much earlier and increased interest in them. This is due to the fact that the detection of changes in this cell and nephropathy allows you to diagnose and stop the process of kidney damage before the appearance of clinical signs.

Micronutrients are essential micronutrients needed for normal body function and include iron (Fe), zinc (Zn), selenium (Se), copper (Cu), iodine (I) and manganese (Mn) in amounts ranging from 50 to 18 micrograms. milligrams per day. in patients with CKD, this may be abnormal as a result of poor nutrition, hypercatabolism caused by uremia, persistent inflammation, or the dialysis procedure itself (4). Optimal micronutrient status can help maintain optimal immune function, reducing exposure to infections and improving quality of life for patients with CKD. Previous research has shown that micronutrient homeostasis may help regulate immune disorders, enhance growth and development, and reduce infections, cardiovascular complications, anemia, and mineral and bone diseases (5). Additionally, loss of micronutrient homeostasis in patients with end-stage renal disease (ESRD) significantly contributes to increased morbidity and mortality. Therefore, micronutrient homeostasis should be considered in all stages of CKD, and all clinicians caring for patients should be aware of micronutrient requirements. This review summarizes the benefits and risks of micronutrients in patients with CKD.

Purpose. We aimed to study the effectiveness of drugs on the studied laboratory and instrumental markers when using drugs containing zinc microelement in addition to standard treatment in order to increase the effectiveness of treatment of nephropathy in these patients in the early stages.

Material and methods. As a research source, 180 patients diagnosed with the early stages of CKD before dialysis, who referred to the multidisciplinary medical center of Bukhara region and received inpatient treatment, were taken. They were divided into 3 groups. The first group consisted of 37 patients with CKD stage 1, the second group consisted of 94 patients with CKD stage 2, and the third group consisted of 49 patients with CKD stage 3a/b.

All patients in the follow-up were compared before and after treatment with standard clinical and laboratory tests, including microalbuminuria, type IV collagen, aldosterone, and cystatin-C. In all patients, the amount of zinc, iron and copper in blood serum, the amount of transferrin and ferritin from ferrokinetic indicators, and the amount of zinc in urine were determined.

Result. The patients involved in the study were prescribed zinc-preserving drugs (containing 20 mg zinc-preserving tablets 1 time per day) for 6 months.

After 6 months, the amount of GFR, cystatin S, zinc, copper and type IV collagen, ferritin in the blood serum was determined and a comparative analysis was carried out in order to evaluate the level of inflammation.Scientific literature reports that there is an inverse proportional relationship between the elements of copper and zinc, that is, a sharp decrease in the microelement of zinc in the body leads to a relative increase in the microelement of copper (S.V. Berestenko and co-authors, 2007).

Reliable changes in a number of indicators were also observed in h patients. The amount of cystatin-C in the blood serum decreased and the GFR indicator increased, and a reliable difference was noted ($r<0.01$).

Also, the effect of the used drugs on laboratory markers according to the stages of CKD was studied. In 27.6% of CKD stage 1 patients who received the zinc preparation, GFR showed indicators higher than 120 ml/min/1.73 m2, that is, normalized, in 16.8% of CKD stage 2 patients. In 19.6% of patients with CKD stage 2, GFR increased by 1.3 times after treatment, in which GFR was detected using cystatin S and diagnosed with CKD stage 1.

It was observed that 14.7% of patients with CKD stage 3a/b passed to CKD stage 2. In order to study the effect of zinc on the development of fibrosis in the kidney, when comparing the indicators of type IV collagen before and after treatment, it was observed that it decreased by 1.38 times in patients with CKD stage 1, 1.3 times in patients with CKD stage 2, and 1.21 times in patients with CKD stage 3, and these changes showed that zinc micronutrient supplementation reduces the degree of fibrosis.

When zinc and copper micronutrient indicators were compared before and after treatment, it was observed that the zinc micronutrient indicator increased more reliably than the copper indicator. That is, zinc increased by 1.69 times, copper by 1.17 times, in patients with stage 2 of the disease by 1.65 times and 1.36 times, and in patients with stage 3a/b by 1.63 and 1.16 times after treatment. observed ($p=0.001$).

We also emphasize the possibility that such changes in mim microelements are related to changes in the amount of zinc.

In patients with CKD, deficiency of micronutrient zinc was more evident than other micronutrients, and these changes were observed from the early stages of the disease.

In the first chapter of the thesis, information is presented based on the analysis of the existence of a strong positive correlation between urinary excretion of zinc trace element and CKD levels ($r= 0.7$; $p<0.001$). A strong negative correlation was found

between the blood serum level of zinc trace element and the clinical stages of CKD (r= -0.64; p<0.001).

A sharp decrease in the amount of zinc in the early stages of the disease indicates that this trace element is a diagnostic marker in the development of CKD and, in turn, a prognostic marker for evaluating the progression of the disease.

Conclusions. When comparing ferritin level before treatment and after treatment, its decrease is not at the level of certainty (r=0.05), it is proven once again that anemia is eliminated, polydeficiency anemia and chronic inflammatory anemia are more likely to develop in CKD anemia.

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