Published: April 30, 2024 | Pages: 102-106

CHANGES IN PHOSPHORUS METABOLISM IN CHRONIC HEART FAILURE WITH RENAL DYSFUNCTION AND EFFECTS OF VITAMIN D SUPPLEMENTATION

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ABSTRACT: The article presents changes in phosphorus indicators in patients with chronic heart failure and advanced kidney dysfunction. Also, on the basis of standard treatment, the combined use of sodium glucose type 2 inhibitor empagliflozin and vitamin D has been found to have a positive effect on electrolyte balance and kidney dysfunction.

KEYWORDS: Chronic kidney disease (CKD), left ventricular ejection fraction (LVEF), hyperphosphatemia, Phosphorus values.

INTRODUCTION

The urgency of the problem. According to experts of the World Health Organization, the spread of chronic non-communicable diseases is called an epidemic of the 21st century [9]. Among them, cardiovascular diseases and their severe complications, CHF, are considered the main cause of death [13]. Asymptomatic myocardial dysfunction occurs in approximately 10 million Europeans [10].

According to studies, analysis of comorbidity in SYuE shows that more than 50% of patients have chronic kidney disease (CKD) [9, 13]. In radionuclide renoscintigraphy, it was found that 73% of patients with CHF had impaired kidney function. But only 13.5% of them have CKD in their anamnesis [11, 12].

Angiotensin-converting enzyme inhibitors, angiotensin receptor antagonists, ß-blockers, and mineralocorticoid receptor antagonists have been used for many years in the treatment of CHF. In recent years, drugs such as sacubutril-valsartan and glucose sodium cotransporter type 2 inhibitors (GNKT-2i) have also been included in the standard treatment of CHF [8].

CHF found that the use of glucose-sodium cotransporter 2 inhibitors in patients with low left ventricular ejection fraction (LVEF) is highly effective. Currently, dapagliflozin, empagliflozin, canagliflozin and other drugs belonging to this group have been created [14]. However, despite

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the positive results achieved in the treatment of CHF in recent years, the death rate from it is still high. In most cases, it is a comorbidity of the disease, and among them, CKD is one of the leading causes of death.

In most cases of CKD, hyperphosphatemia is observed and it can be attributed to impaired excretion of this mineral [6]. There are few studies on CHF disorders of phosphorus metabolism. Plischke and coauthors identified 99 patients with CHF who had a left ventricular ejection fraction of 33±10% in an outpatient setting for 35 months. It found that organophosphates were associated with CHF (hazard ratio-HR=26.044) and other cardiovascular events (HR=26.944) with hospitalizations and composite endpoints (HR=13.294)[3].

The cause of hyperphosphatemia in heart failure is, firstly, hypoxia that occurs in the severe stages of this complication and, as a result, the induced release of microelements from cells [1], secondly, increased absorption of phosphates in swollen and ischemic intestines [5, 1, 2], and finally, it is related to impaired kidney function [4, 6].

In addition to the above, but until now, in patients with advanced renal dysfunction, CHF is used in combination with vitamin D, ß-blockers, mineralocorticoid receptor antagonists, sacubitril/valsatan, angiotensin-converting enzyme inhibitors, and sodium-glucose cotransporter type 2 inhibitors that are part of their complex. the effect is not covered in the literature.

Materials and Methods. 120 CHF Ⅱ and Ⅲ FC patients with advanced renal dysfunction were included in our study. In them, serum creatinine, which is a traditional test method for assessing kidney dysfunction, and glomerular filtration rate (GFR) calculated using it were taken as criteria. Patients included in the follow-up were divided into two main and control groups according to the treatment received at the beginning. The main group consisted of 80 patients, and their average age was 66.5±5.7, men 43 (53.75%) - women 37 (46.25%). Among them, patients with CHF Ⅱ and Ⅲ FC were 14 (17.5%) and 66 (82.5%), respectively. GFR was equal to 80.6 ± 5.5 ml in 1 minute per 1.73 m2 body surface.

The control group consisted of 40 patients with an average age of 67.6 ± 5.5 years, 20 (50%) men and 20 (50%) women. Among them, patients with CHF Ⅱ and Ⅲ FC were 8 (20%) and 32 (80%), respectively. GFR was equal to 78.4 ± 5.2 ml in 1 minute per 1.73 m2 body surface.

The main and control group of patients involved in the study were divided into two subgroups based on vitamin D levels during the examinations. The first subgroup was made up of patients whose blood serum vitamin D level decreased from normal values (Vit D≤30, ng/ml) and the second subgroup was made up of patients whose level was maintained (Vit D≥30, ng/ml). 40% (32) of patients in the main group and 42.5% (17) of the control group were found to have reduced vitamin D levels.

Patients with reduced vitamin D content of the main group were prescribed CHF complex standard treatment (sacabutril-valsartan, ß-blocker, mineralocorticoid receptor antagonist-eplerenone, sodium glucose cotransporter type 2 inhibitors-empagliflozin) and vitamin D at 4000 units per day for 8 weeks. A maintenance dose of 2,000 units was then recommended for 4 weeks. Only complex standard (sacabutril-valsartan, ß-blocker, mineralocorticoid receptor antagonist-eplerenone, sodium glucose cotransporter type 2 inhibitors-empagliflozin) treatment was applied to patients with normal vitamin D levels. At this point, we would like to point out that there is no

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information published in the available literature about the effectiveness of sodium glucose cotransporter type 2 inhibitors and vitamin D when used in combination with GFR patients on the basis of CHF.

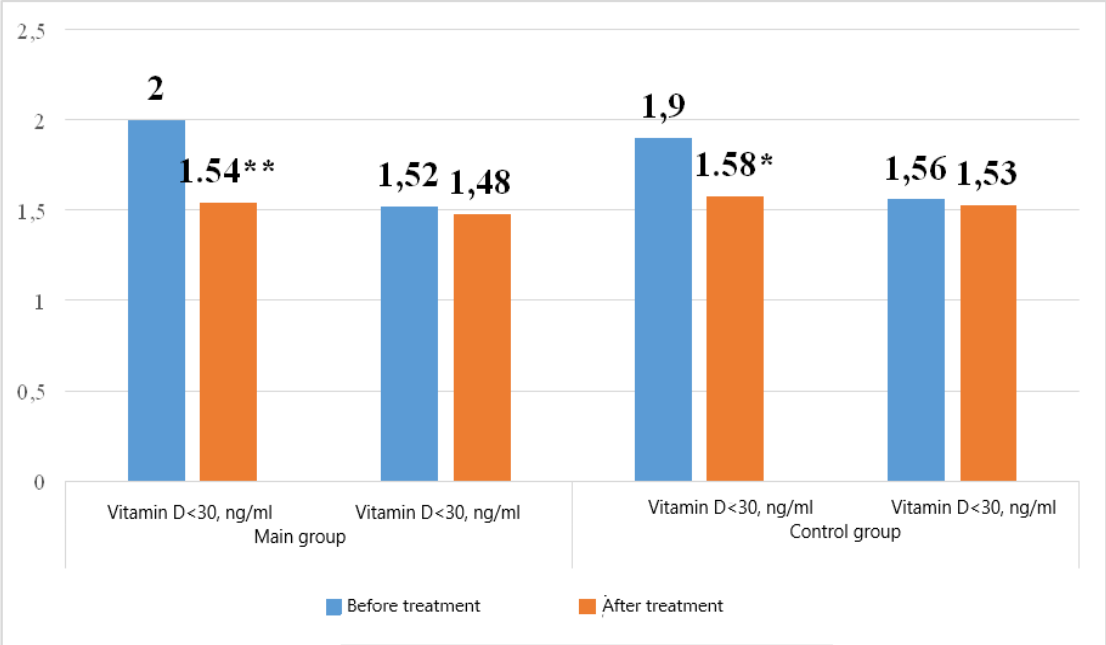
Patients with reduced vitamin D serum levels in the control group were prescribed CHF complex standard treatment (sacabutril-valsartan, ß-blocker, mineralocorticoid receptor antagonist-eplerenone) and vitamin D 4000 units per day for 8 weeks. A maintenance dose of 2,000 units was then recommended for 4 weeks. Patients with normal vitamin D levels were recommended only complex standard treatment (sacabutril-valsartan, ß-blocker, mineralocorticoid receptor antagonist-eplerenone).

Vitamin D and phosphorus indicators in the blood serum were determined in all subjects involved in the study before and after 6 months of treatment, as well as common laboratory tests. Kidney functional status was evaluated by calculating GFR using creatinine.

Research results and discussion. An increase in the amount of phosphorus in patients with GFR on the basis of CHF has been shown in a number of scientific studies. Here it should be noted that most of the observations were made in the severe stages of GFR. We studied phosphorus parameters in GFR associated with CHF and evaluated the effect of standard treatment of CHF supplemented with vitamin D. Figure 1 below shows the dynamics of phosphorus indicators in the main and control group of patients.

Figure 1.

Phosphorus indicators in blood before and after treatments in patients under observation



Note: * - the reliability of the difference between indicators before and after treatment: * - r<0.05 - r<0.01.**

As shown in the figure, serum phosphorus decreased 1.29 times from 2.1 ± 0.3 mmol/l before treatment and 1.54 ± 0.25 mmol/l after treatment in the subgroup with vitamin D deficiency in the main group. . When they were compared, a highly reliable difference was noted (r<0.01). Phosphorus improved from 1.52±0.24 mmol/L to 1.48±0.15 mmol/L in

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the subgroup with normal vitamin D levels, but no reliable difference was observed.

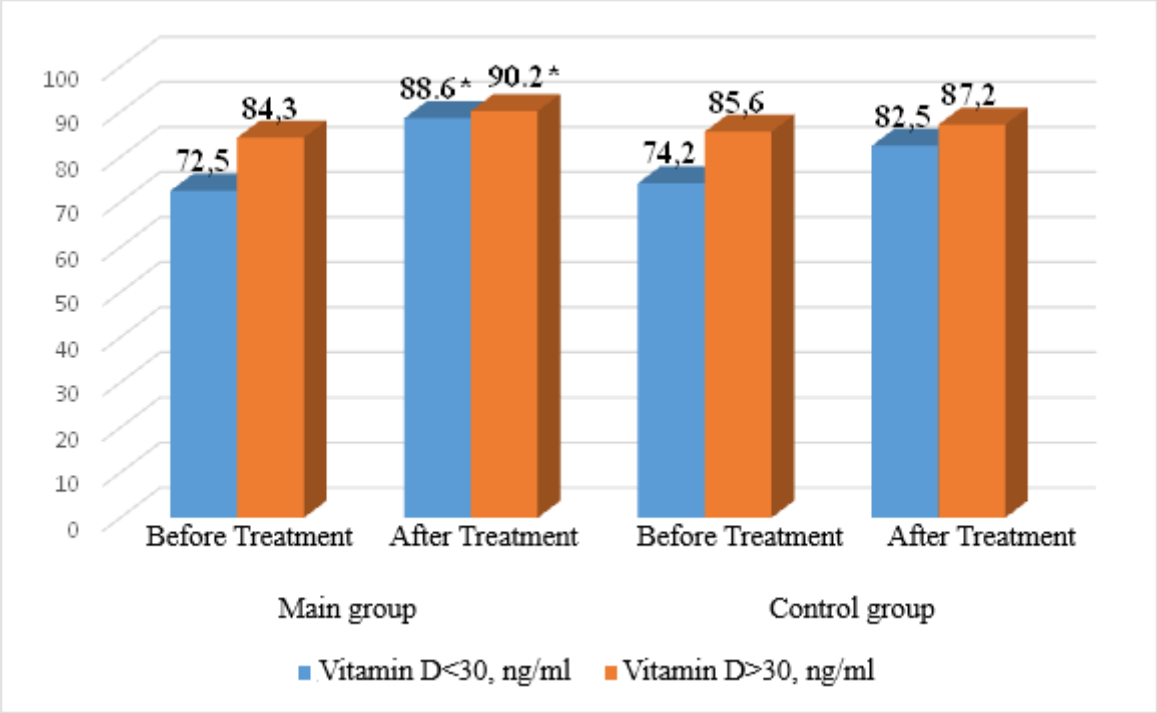
Phosphorus values in the control group of patients receiving vitamin D were 1.9±0.32 mmol/l before treatment and 1.58±0.2 mmol/l in the first group, and there was a reliable (r<0.05) difference between them. observed. In the second group, the uni content decreased from 1.56±0.3 mmol/l to 1.53±0.25 mmol/l, but no reliable difference was detected.

The results obtained CHF Prescribing vitamin D with empagliflozin-containing complex therapy in patients with advanced renal dysfunction has a positive effect on phosphorus indicators.

Also, the effect of the complex treatment procedures on the functional state of the kidneys was studied. Figure 2 below shows the post-treatment changes in creatinine and glomerular filtration rate calculated using it.

Figure 2.

Indicators of glomerular filtration rate after treatments in the main and control group patients.



Note: * - the reliability of the difference between indicators before and after treatment: * - r<0.05.

Glomerular filtration rate improved from 72.5±5.2 to 88.6±5.6 mL/min per 1.73 m2 of body surface area in the baseline group with low vitamin D, and a significant difference (r<0.05) was observed. Its amount increased from 84.3±6.8 to 90.2±6.4 ml per 1 minute per 1.73 m2 of body surface in the normal group (p<0.05). In the control group, these indicators were 74.2±5.4 to 1.73 m2 body surface area in the first subgroup, 82.5±6.2 ml per minute in the second subgroup, and 85.6±7.5 to 1.73 m2 body in the second subgroup, respectively. level increased by 87.2±6.9 ml in 1 minute. However, no reliable difference (r>0.05) was found in both groups.

Serum creatinine decreased from 98.2 ± 7.4 to 72.6 ± 5.3 µmol/L in vitamin D-deficient patients in the baseline group with a highly reliable difference (r<0.01) after standard treatment. The second,

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i.e. vitamin D level, decreased from 84.1 ± 8.7 to 70.4 ± 6.2 µmol/l in those with normal levels, and a reliable difference (r<0.01) was observed. In the control group, before treatment, it was 97.5±9.6 and 82.4±9.2 µmol/l in both subgroups, and after treatment it was 85.4±8.7 and 80.4±7, improved to 6 µmol/L. However, there was no significant difference (r>0.05) between the groups.

CONCLUSION

In patients with chronic kidney disease due to chronic heart failure, determination of vitamin D levels in blood serum and coordination of treatment with its help leads to reduction of pathological processes in kidneys and improvement of their function. They have a nephroprotective effect, reduce oxidative stress inflammation and fibrosis processes in the kidneys and stabilize the development of chronic kidney disease. This is confirmed by the results obtained by prescribing the vitamin D preparation for treatment in patients in the main and control groups in our study.

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