The American Journal of Medical Sciences and Pharmaceutical Research ISSN 2689-1026 Volume 08 - 2026

Role Of Fetuin-A In The Clinical And Laboratory Manifestations Of Calcification In Chronic Kidney Disease

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Received: 30 Mar 2026 | Received Revised Version: 14 Apr 2026 | Accepted: 28 Apr 2026 | Published: 20 May 2026

Volume 08 Issue 05 2026 | Crossref DOI: 10.37547/tajmspr/Volume08Issue05-16

Abstract

The article presents the results of a study on changes in calcium-phosphorus balance in patients with chronic kidney disease, depending on the level of fetuin-A in the blood serum, and on the calcification processes that occur in the body. It also discusses changes in calcium, phosphorus, PTH, and vitamin D, as well as the occurrence of cardiac calcification, in the study groups with normal and low levels of fetuin-A. The conclusions based on the conducted studies also recognize the promising research that needs to be done in this regard.

Keywords. Chronic kidney disease, fetuin-A, calcium, phosphorus, vitamin D, calcification.

Cite This Article: Yuldoshev Turgun Ravshanovich, Sabirov Maksud Atabaevich, & Munavvarov Burkhan Abdujalilovich. (2026). Role Of Fetuin-A In The Clinical And Laboratory Manifestations Of Calcification In Chronic Kidney Disease. The American Journal of Medical Sciences and Pharmaceutical Research, 8(05), 75–78. https://doi.org/10.37547/tajmspr/Volume08Issue05-16

1. Introduction

Chronic kidney disease (CKD) has been formed as an independent nosology, due to its diverse etiological basis, its course and consequences, especially as it progresses to the final terminal stages, vary. For example, in some people, conservative or predialysis stages of CKD last for 5-10 years, while in others, the early stages of the disease, based on one or two repeated acute infectious diseases, progress to the terminal stage of chronic renal failure (CRF) in a few months [1]. In one case, anuria suddenly develops in stage 5 of CKD and the patient dies from hyperkalemia, while in another, someone continues for years at this stage without starting renal replacement therapy. From this point of view, the process of receiving renal replacement therapy in the terminal stage of CKD also varies. These aspects are not sufficiently explained by the markers of kidney damage, inflammatory mediators, and even cytokine mechanisms that are currently widely used in clinical practice [9, 11]. In this regard, new kidney biomarkers are emerging in modern clinical medicine. One of these elements is the biomarker fetuin-A. The role of this marker in the calcium-phosphorus imbalance that occurs in SCD is unique. Moreover, the fact that fetuin-A appears in the body as an anti-calcification

factor has been reflected in many literatures [4, 5, 6]. It is known that the kidneys play an important role in maintaining the balance of calcium and phosphorus. Renal failure negatively affects all aspects of calcium-phosphorus metabolism, disrupting it. In patients with SCD, when the GFR decreases below 60 ml/min/1.73 m2 , phosphorus filtration decreases and its serum concentration begins to increase [7, 8, 12] . This leads to an increase in parathyroid hormone (PTH) secretion by the parathyroid gland. PTH reduces the reabsorption of phosphorus and thus tries to normalize its serum level. However, when the GFR decreases below 30 ml/min, this mechanism fails, and persistent hyperphosphatemia develops. This, in turn, stimulates increased PTH secretion. Hyperphosphatemia inhibits the activity of 1 α -hydroxylase in the proximal tubules, resulting in a decrease in the release of 1 ,25(OH)2D3 - calcitriol and its concentration in the blood. Calcitriol deficiency leads to impaired calcium absorption in the small intestine and the development of hypocalcemia. If hypocalcemia persists for several months, hyperplasia of the parathyroid gland develops. This, in turn, leads to excessive production of PTH, that is, increased secretion. Hypocalcemia, vitamin D deficiency, and hyperphosphatemia are the most important factors for PTH. Hypocalcemia, relative or absolute calcitriol deficiency, and hyperplasia of the parathyroid gland begin to develop in the initial stage of renal dysfunction (SCC stage 3 (ECG 60–30 ml/min/1.73 m 2) and are recognized in the literature as progressing with the progression of renal failure [4]. Therefore, the diagnosis of chronic kidney disease with modern biomarkers in the field requires an assessment of their role in the course of the disease and the formation of its consequences.

The purpose of the study.

Analysis of clinical-laboratory changes of calcium-phosphorus imbalance in relation to serum fetuin-A level in patients with chronic kidney disease .

Material and methods. 103 patients with CKD in the conservative and predialysis stages treated at the nephrology department of the National Medical Center were selected for the study. All patients included in the study had their serum fetuin-A levels determined, and based on their values, they were divided into two groups (N 27.31 – 205.98 ng/mL), i.e., those with a fetuin-A level below the norm (n-76, Fetuin-A – 15.11±5.41) and those with a fetuin-A level within the norm (n-27, Fetuin-A – 57.8±6.48). The clinical and laboratory changes in calcification processes (calcium, phosphorus, vitamin D, PTH, signs of cardiac calcification on echocardiography) were analyzed in the study groups. Also, the degree of dependence of cardiac calcification on the concentration of fetuin-A in the blood was calculated using the online computer program https://medstatistic.ru/calculators/calchi.html. The results were statistically analyzed.

Results and their discussion.

According to the results, calcium fetuin-A in the normal group was on average 1.99 ± 0.08 mlmol/l. and it was found to change unreliably (r>0.05) compared to the control group, and in the group with f etuin-A below the norm, calcium averaged 1.87 ± 0.07 mlmol/l, and it was observed to decrease reliably (r<0.01) compared to the control group. When the main groups were compared, it was shown that the value of hypocalcemia was not reliable (r>0.05) in the group with fetuin-A level below the norm compared to the group with normal fetuin-A level . Hyperphosphatemia showed a similar picture. In particular, the amount of phosphorus in the blood is 1.42 ± 0.08 mlmol/l in the group with normal fetuin-A . and it changed unreliable (r>0.05) compared to the control group, and in the group with f etuin-A below the norm, it was observed that phorphor was 1.65 ± 0.09 mlmol/l, and it exceeded reliably (r<0.001) compared to the control group. When the main groups were compared with each other, it was found that the phosphorus value changed unreliably (r>0.05) in the group with fetuin-A below the norm compared to the group with the normal fetuin-A level (Table 1.1).

Table 1.1

Clinical-laboratory picture of calcification processes depending on fetuin-A concentration in blood

Parameters	**Control group (n -20)**	**Fetuin-A is normal group with (n - 27)**	**Fetuin-A group below the norm (n - 76)**	**Statistical analysis** (between groups)
FetuinA (N 27 , 1 – 205 , 98 ng	9 3.8 ± 1.67	57.8±6.48 ***	15.11±7.41 *** ^^	p<0.01

/ mL)				
calcium	2, 1 7 ± 0.0 6	1.9 9 ± 0.0 8	1.87 ± 0.07 **	p>0.05
phosphorus	1.07 ± 0.06	1.42 ± 0.08	1.6 5 ± 0.09 ***	p>0.05
PTG *(N 15-65pg/ml)*	4 6 , 9 2 ±4 , 1 1	68.6 ± 4.09 **	7 9.5 ± 3.18 *** ^	p<0.05
25(ON) D *(N 30-60 ng /ml)*	37.14 ± 2.41	2 4, 3 ± 3 ,1 7**	1 3.9 ± 2.1 6 *** ^^	p<0.01
Cardiac calcification	20/0	4/23	47/29	$\chi 2 = {}^{4.986}$; (p = 0.026)

Note: * - PTG – parathyroid hormone (parathormon); 25(ON) D – 25-hydroxyvitamin D.

* - difference values are significant compared to the indicators of the control group (*- r<0.05, **- r<0.01, ***- r<0.001); ^ - where the values are significant compared to the differences between the main research groups (^ - r<0.05, ^^ - r<0.01, ^^^ - r<0.001).

* The degree of dependence of cardiac calcification on the concentration of fetuin-A in the blood was calculated using the online computer program https://medstatistic.ru/calculators/calchi.html

If we pay attention to the indicators of calcium-phosphorus imbalance in patients with SCD in our study groups, although the decrease in calcium or increase in phosphorus in groups with normal levels of fetuin-A compared to healthy individuals was insignificant (p>0.05), the indicators still changed. Also, their significant (p<0.01) change in groups with normal levels of fetuin-A, while the difference in values in the main groups was insignificant (p>0.05), indicates the presence of calcium-phosphorus imbalance in groups with normal levels of fetuin-A. However, their significant change in individuals with low levels of fetuin-A indicates that this is a clear violation of the process of calcium-phosphorus metabolism .

The calculated PTG and vitamin D, the next indicators of calcium-phosphorus balance, changed as follows in our research groups. In particular, the level of PTG in the blood in the group with normal fetuin-A was 68.6 ± 4.09 pg /ml on average, which was significantly (r<0.01) higher than the control group, while the PTG in the blood serum of patients in the group with low fetuin-A was 79.5 ± 3.18 pg/ml , which is more reliable than the control group. (r<0.001) was shown to be exceeded. When the main groups were compared, it was found that the PTG value was significantly (r<0.05) higher in the group with fetuin-A than in the group with normal fetuin-A level (Table 1.1).

Vitamin D (25(OH)D) also showed the same pattern as PTH. That is, 25(OH)D in the group with normal fetuin-A was 24.3 ± 3.17 ng/ml , which was significantly (p<0.01) lower than the control group, while in the group with low fetuin-A, the level of 25(OH)D decreased by 13.9 ± 2.16 ng/ml , which was significantly (p<0.001) lower than the control group. When the main groups were compared, it was found that the group with low fetuin-A had significantly (p<0.01) lower vitamin D values than the group with normal fetuin-A levels (Table 1.1).

If we pay attention to the PTH and 25(OH)D indicators in patients depending on the level of fetuin-A , in the groups with normal fetuin-A levels, although they changed slightly above the normal threshold, a significant increase (p<0.01) in PTH and a significant decrease (p<0.001) in 25(OH)D compared to healthy individuals indicates the presence of calcium-phosphorus imbalance in these patients. However, a significant change (p<0.001) in the groups with lower fetuin-A levels, as well as a significant difference in values in the main groups (p<0.05, p<0.01), indicates a pronounced manifestation of the process of calcium-phosphorus metabolism, that is, the intensification of calcification processes

It is known that impaired calcium-phosphorus metabolism in patients with SCD leads to renal osteodystrophy, which

is manifested by the development of osteopenia or osteoporosis in the bones, and these processes can be diagnosed by densitometry. This is reflected in a number of publications devoted to this area [2, 3]. Also, these changes are reflected not only in the bone, but also in other organs (blood vessels, kidneys, etc.) and tissues, including the heart. In our studies, data on the presence of calcification processes in the heart of patients using echocardiography were also analyzed. According to this, 29 of 76 patients with a lower than normal level of fetuin-A were found to have calcification processes in the heart during echocardiography. Also, 4 of 27 patients with a normal level of fetuin-A were found to have cardiac calcification. When the results were statistically analyzed, the Chi- square test for the occurrence of cardiac calcification in relation to the serum fetuin-A concentration in patients with SCD showed a reliable value of 4.986 ($p = 0.026$). This confirms the statistically significant relationship between the frequency of cardiac calcification and the serum fetuin-A level

Conclusion

Thus, from the early stages of SCD, calcium-phosphorus metabolism is disturbed, which is manifested by a decrease in calcium and vitamin D in the blood, an increase in phosphorus and PTH. This imbalance has serious consequences not only in the blood, but also in other organs and tissues. It is known to specialists that, along with bone fractures due to renal osteodystrophy, the exacerbation of blood vessel and heart calcification processes is an important pathogenetic chain of the development of chronic heart and kidney failure (glomerulosclerosis). Now, the change in these processes depending on the level of fetuin-A in the blood, that is, the exacerbation of calcification processes when this biomarker decreases, is a large-scale problem. Therefore, the study of such biomarkers in the development of SCD and the continuation of research on their dynamics are among the main tasks facing us.

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