НИРКИ KIDNEYS

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Calcium and phosphorus imbalances as biochemical markers in chronic kidney disease: a case-control study

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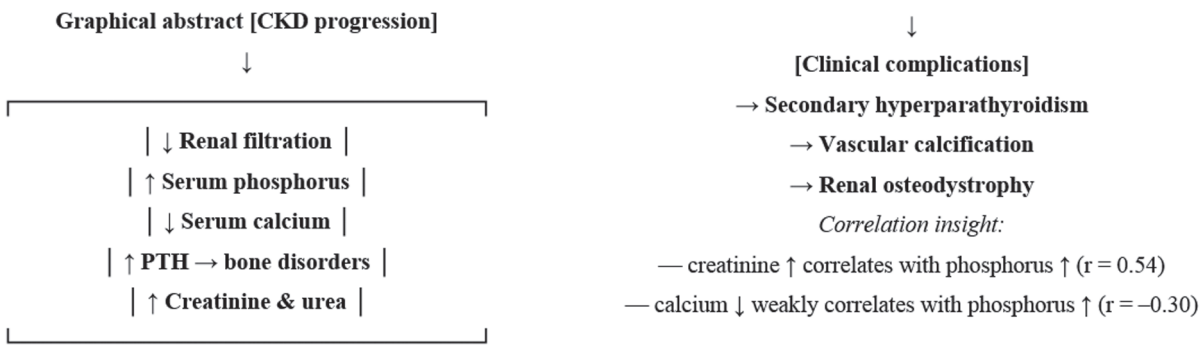
Abstract. ***Background.*** *Progressive loss of renal function is the hallmark of chronic kidney disease (CKD), which is often linked to biochemical abnormalities, especially in the calcium and phosphate metabolism. In the pathophysiology of secondary hyperparathyroidism, vascular calcification, and bone mineral abnormalities, these disruptions are crucial. The purpose of this research was to examine the blood calcium and phosphorus profiles of hemodialysis patients with CKD, to clarify if these profiles correlate with indices of renal function and compare results to those of healthy people in order to determine whether these profiles are diagnostically or prognostically relevant.* ***Materials and methods.*** *A comparative, cross-sectional research was carried out at Al-Hussain Hospital between December 2024 and June 2025, comprising 60 patients with end-stage renal disease who were between the ages of 25 and 72 years and were receiving long-term hemodialysis. At the same time, 30 healthy age- and gender-matched controls were included in the study. The amounts of serum urea, creatinine, calcium, and phosphorus were measured and analyzed using the conventional biochemical techniques. An examination of statistical data was carried out using SPSS version 26, with the level of significance set at $p < 0.05$. To analyze the connections between the parameters, Pearson's correlation was used.* ***Results.*** *In comparison with healthy controls, patients with chronic kidney disease showed substantially higher levels of serum phosphorus (5.37 ± 0.47 mg/dL) and creatinine (7.46 ± 1.15 mg/dL), as well as lower calcium levels (5.54 ± 0.41 mg/dL) ($p < 0.0001$ for all). The link between creatinine and phosphorus was somewhat positive ($r = 0.54$); however, calcium and phosphorus levels were negatively associated ($r = –0.30$).* ***Conclusions.*** *The presence of hyperphosphatemia and hypocalcemia is quite common in individuals with advanced CKD, and both conditions are directly linked to reduced renal function. Phosphorus levels may be used as a surrogate measure for renal impairment, as shown by the modest association that exists between phosphorus and creatinine. Monitoring calcium and phosphorus on a regular basis, in addition to traditional indicators, is essential for the early diagnosis of changes in mineral metabolism and the prompt treatment of problems connected to CKD.*

Keywords: *chronic kidney disease; biomarkers; hyperphosphatemia; hypocalcemia; hemodialysis; bone mineral disorder; creatinine; phosphate-calcium axis*



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Introduction

Damage to the kidneys over time causes a major upset in the body's biochemical equilibrium, particularly with regard to calcium and phosphorus levels; this disorder is known as chronic kidney disease (CKD). In addition to being major factors in skeletal and cardiovascular problems, these two minerals are essential for evaluating the metabolic status of CKD patients [1]. Research has shown that hyperphosphatemia develops when the kidneys aren't working properly, and that calcium levels drop when vitamin D activation is low and absorption in the intestines is poor [2]. The secretion of parathyroid hormone (PTH) is stimulated by these disturbances, resulting in secondary hyperparathyroidism. Vascular calcification is promoted and bone disease (renal osteodystrophy) is exacerbated by this situation [3, 4]. These mineral metabolism problems continue to be a leading cause of mortality in CKD patients and have been significantly associated with increased cardiovascular risk [5].

The current research investigates the ways in which changes in calcium and phosphorus levels might serve as early indicators of difficulties connected to CKD. In addition to this, it highlights the clinical significance of these mineral abnormalities in terms of early identification and preventative therapy, as well as looks into the underlying processes that are responsible for their impact on patient health. The condition known as hyperphosphatemia is brought on by chronic renal disease, which causes phosphate excretion to be hindered. The disruption of calcium and phosphate balance that results from this condition, in conjunction with the reduced synthesis of calcitriol, is a contributing factor in the development of hypocalcemia and secondary hyperparathyroidism (SHPT) [6, 7]. The conversion of 25-hydroxyvitamin D to its active form, calcitriol, is diminished in patients with CKD due to the decreased activity of renal 1α-hydroxylase. Because of this decline, intestinal calcium absorption is impaired, which ultimately results in hypocalcemia. Furthermore, the retention of phosphate hinders the production of calcitriol, which only serves to exacerbate the calcium imbalance [8]. The parathyroid glands are stimulated to release PTH when hypocalcemia is present, which ultimately results in SHPT. Bone resorption is increased in order to release calcium when PTH levels are elevated; nevertheless, this compensatory process leads to bone abnormalities in patients with chronic kidney disease [9]. Systemic disruptions in mineral metabolism are included in chronic kidney disease-mineral and bone disorder (CKD-MBD). These disturbances include anomalies in calcium, phosphate, PTH, and vitamin D levels, which ultimately result in bone pathology and vascular calcification [1].

Materials and methods

Concerning data of samples

The research was conducted on 60 patients, both male and female, with end-stage renal disease (ESRD) who were receiving long-term hemodialysis treatment at the hemodialysis unit at Al-Hussain Hospital. The patients' ages ranged from 25 to 70 years old. It was from December 2024 to June 2025 that the research was conducted. In order to serve as a control group, 30 healthy individuals, both men and females, were selected. Through the use of a specialized questionnaire, the personal information of each participant, both those who were ill and those who were well, was collected.

Study design is presented in Fig. 1.

Conditions for exclusion

Exclusion criteria for patients with hepatitis B, recent hospitalization, current phosphate binder therapy, cancer, inherited or acquired blood disorders, acute or chronic inflammation, recent hemorrhage, and any other condition that could affect their hematological parameters were outlined in the study.

Collection of samples

Before each hemodialysis session, four milliliters of blood were drawn from patients with CKD. The blood sample was then placed in a test tube, and after that, it was separated in order to produce blood serum. The samples were analyzed for levels of creatinine, blood urea, calcium, and phosphorus in order to determine the severity of the kidney function impairment. Additionally, all of the tubes were transported via an icebox until they arrived at the laboratory.

Statistical analysis

The data collected in this research was subjected to statistical analysis using SPSS version 26, with the independent sample t test being used to determine variance and the person coefficient being used to determine correlation at a p-value of less than 0.05 [10].

Results

Distribution of patients according to sex

As shown in Table 1, the current research demonstrated a statistically significant difference at a p-value of less than 0.05. The high number of patients in the male group was recorded at 40 (66.67 %), while the female group had 20 (33.33 %).

Distribution of patients according to age

Table 2 presents an illustration of the age distribution of patients who have been diagnosed with chronic renal failure.

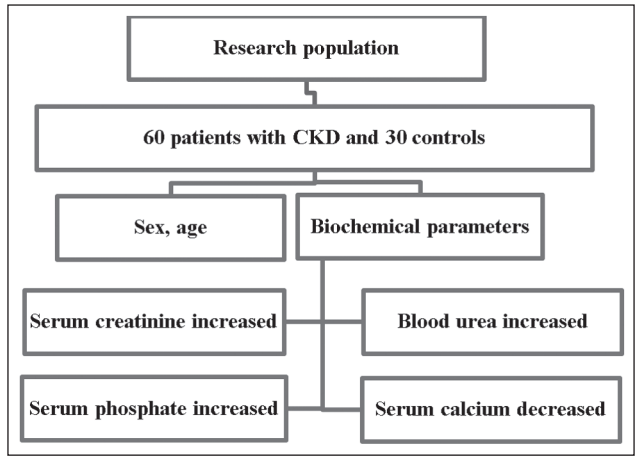

Figure 1. Flow chart of study design

It reveals that the largest proportion of patients, which accounts for 45 %, are within the age group of 57–72 years, followed by 35 % of patients aged 41–56 years, and only 20 % of patients aged 25–40 years. The study also demonstrated a significant difference between the age groups.

Evaluation of renal function test and electrolyte in renal patient and control group

The research obtained a statistically significant difference with a p-value of less than 0.05. In this investigation, the biochemical parameters (Table 3) that were evaluated revealed substantial differences in the levels of urea, creatinine, calcium, and phosphorus among patients who were diagnosed with CKD in comparison to healthy controls.

Table 1. Distribution of CKD patients according to sex

Sex	N	%
Males	40	66.67
Females	20	33.33
Total	60	100
p-value 0.046		

Table 2. Distribution of CKD patients according to age

Age, years	N	%
25–40	12	20.0
41–56	21	35.0
57–72	27	45.0
Total	60	100
p-value 0.009		

According to Table 3, all four biochemical markers showed statistically highly significant differences between patients and controls ($p < 0.0001$). Urea and creatinine levels are markedly elevated in CKD patients, indicating renal impairment. Serum calcium is significantly lower in CKD patients, reflecting disturbances in mineral metabolism. Phosphorous levels are significantly higher, consistent with hyperphosphatemia common in CKD due to reduced excretion.

Person correlation between biochemical parameters

Table 4 shows that there is a modest positive association ($r = 0.54$) between serum creatinine and phosphorus levels, suggesting that both parameters tend to increase together when renal function deteriorates. Weak or negligible correlations were found between other parameters.

Discussion

Chronic kidney disease patients are 80 % male and 20 % female. Males are more likely to require renal replacement therapy such transplants or dialysis to achieve ESRD faster than females [11]. Biological and behavioral factors explain the gender disparity. Estrogen preserves kidney function in women, but testosterone may promote renal damage and fibrosis in males [12]. Men are also more likely to smoke, eat more protein, and be exposed to occupational nephrotoxins, which raise CKD risk [13].

Health system factors may apply. According to research, men are more likely to be sent to nephrology services early, undergo dialysis, and get kidney transplants than women [11]. Due to access and treatment disparities, CKD groups may have varied demographics. Thus, the finding that males made up a bigger fraction of the sample may indicate biological differences in disease onset and healthcare access and delivery [14]. Recognizing and addressing these issues is essential to gender-equitable CKD treatment.

Table 3. Biochemical parameters of kidney function in study samples, mg/dL (mean ± SD)

Parameter	Patients	Controls	p-value
Urea	135.90 ± 2.53	30.78 ± 2.19	< 0.0001
Creatinine	7.46 ± 1.15	1.05 ± 0.13	< 0.0001
Calcium	5.54 ± 0.41	9.34 ± 0.14	< 0.0001
Phosphorous	5.37 ± 0.47	3.34 ± 0.14	< 0.0001

Table 4. The correlation among the study parameters of patients

Parameters	Urea	Creatinine	Calcium	Phosphorous
Urea	1.00	–0.31	–0.03	0.17
Creatinine	–0.31	1.00	–0.01	0.54
Calcium	–0.03	–0.01	1.00	–0.30
Phosphorous	0.17	0.54	–0.30	1.00

Global epidemiological study shows that CKD prevalence rises with age, and this distribution matches that trend. Due to structural and functional kidney degradation, such as nephron loss, glomerulosclerosis, and vascular stiffness, GFR normally declines with age [15]. Physiological changes in elderly people raise the likelihood of chronic kidney disease even without other health issues.

The main risk factors for CKD, hypertension and type 2 diabetes, are also more frequent in older people. These co-occurring diseases gradually damage renal function [16]. This study largely comprised patients aged 57–72, consistent with the Chronic Kidney Disease Prognosis Consortium's findings that CKD incidence and severity grow dramatically after 50 [17].

Younger people may have better renal reserve and reduced chronic disease risk, which may explain why 20 % of patients are in this age range. Hereditary nephropathies, autoimmune diseases, and congenital anomalies cause CKD in this age group [18]. While uncommon, early-onset CKD has a longer disease course and a higher risk of lifelong complications, making it important to detect and treat. This aging chronic kidney disease burden emphasizes the need for preventive nephrology therapy and age-specific screening programs to reduce the risk of ESRD and its healthcare costs [19].

Compared to healthy controls, CKD patients have significant biochemical alterations in urea, creatinine, calcium, and phosphorus. Chronic renal illness causes kidney function decrease and mineral metabolism disturbances, which these changes reflect. Compared to controls, CKD patients exhibited substantially higher urea and creatinine levels (135.90 ± 2.53 mg/dL and 7.46 ± 1.15 mg/dL, respectively) with a p-value < 0.0001. Our findings support serum urea and creatinine as primary markers of GFR and total renal function. Kidney failure reduces excretion, causing blood nitrogenous waste accumulation [20].

Additionally, CKD patients had significantly lower blood calcium levels (5.54 ± 0.41 mg/dL) compared to controls (9.34 ± 0.14 mg/dL). Reduced renal conversion of 25-hydroxyvitamin D to 1,25-dihydroxyvitamin D may impede intestinal calcium absorption in chronic kidney illness, causing hypocalcemia [21]. Hyperphosphatemia and PTH resistance in renal and bone tissues aggravate secondary hyperparathyroidism [22].

Patients with CKD exhibited significantly higher blood phosphorus levels (5.37 ± 0.47 mg/dL) compared to controls (3.34 ± 0.14 mg/dL) (p-value < 0.0001). Severe CKD induces hyperphosphatemia due to renal phosphate excretion reduction. Arterial calcification, increased phosphorus levels, and cardiovascular morbidity and mortality have been associated to chronic renal disease [23, 24]. This study shows that serum calcium and phosphorus are negatively correlated in CKD patients, supporting mineral metabolism dysregulation. Low calcium and high phosphorus, creatinine, and urea values indicate renal excretory dysfunction and bone-mineral metabolism issues. Preventing cardiovascular events and bone disorders in CKD patients requires early detection and treatment of these abnormalities.

Table 4 shows a modest positive correlation (r = 0.54) between blood creatinine and phosphorus levels rising concurrently as renal function declines. This is pathophysiologically feasible since CKD decreases renal excretion of inorganic phosphate and creatinine, a nitrogenous waste product [21]. Hyperphosphatemia causes arterial calcification, cardiovascular disease, and increased mortality, particularly in advanced chronic renal disease [25]. Phosphorus and calcium had a moderate negative correlation (r = –0.30) supporting their adverse physiological relationship. In chronic kidney disease, poor renal phosphate clearance raises blood phosphorus. This increases PTH and FGF-23, which limit calcitriol synthesis and calcium absorption. This relationship affects hypocalcemia and secondary hyperparathyroidism in CKD-MBD [26].

A weak negative connection with creatinine (r = –0.31) and calcium (r = –0.03) and a minor positive correlation with phosphorus (r = 0.17) were the only meaningful relationships. Urea is a sensitive indication of nitrogen retention, although protein ingestion, catabolism, and hydration status affect its variability, as these modest associations suggest [1]. The counterintuitive inverse association between urea and creatinine may be owing to individual differences in CKD patients' creatinine production or tubular secretion, particularly in muscular wasting or malnutrition. Since creatinine and calcium show no significant relationship (r = –0.01), hormonal and gastrointestinal factors may have a higher influence on calcium levels in CKD than glomerular filtration alone. Our data show that vitamin D, phosphate, and parathyroid hormone maintain calcium homeostasis.

To conclude, the correlation analysis shows that multiple pathophysiological pathways cause CKD. When creatinine and phosphorus are considerably connected, both minerals are reliant on renal excretion. When calcium and phosphorus are negatively related, mineral balance is upset. Our data show that chronic renal disease therapy requires extensive biochemical monitoring to decrease systemic risks and improve patient outcomes.

Conclusions

Biochemical abnormalities including hypocalcemia and hyperphosphatemia are related with chronic kidney disease, which decreases renal function. These anomalies induce secondary hyperparathyroidism, CKD-MBD, cardiovascular disease, and mineral and bone disorders. High blood creatinine and phosphorus levels show how mineral metabolism and renal clearance are linked. Preventing CKD complications requires early detection and treatment. Biochemical markers must be consistently monitored in chronic kidney disease treatment to improve patient outcomes and quality of life. These indicators include calcium, phosphorus, PTH, and creatinine.

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Authors' contributions. Noora Q. Al-Khafaji — conceptualization, data curation, investigation, methodology, project administration, resources, software, original draft, review & editing; Hanan B. Saadon, Sarah Jassim Abed — conceptualization, data curation, investigation, methodology, project administration, original draft, review & editing.

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Порушення рівнів кальцію та фосфору як біохімічні маркери при хронічній хворобі нирок: дослідження типу «випадок — контроль»

Резюме. Актуальність. Прогресуюча втрата функції нирок є ознакою хронічної хвороби нирок (ХХН), що часто супроводжується біохімічними порушеннями, зокрема обміну кальцію та фосфатів. Ці розлади відіграють ключову роль у патофізіології вторинного гіперпаратиреозу, судинної кальцифікації і порушень мінерального обміну кісткової тканини. **Мета:** вивчити профілі кальцію та фосфору в крові пацієнтів із ХХН, які перебувають на гемодіалізі, з'ясувати, чи корелюють вони з показниками функції нирок, та порівняти результати з даними здорових осіб для визначення діагностичної або прогностичної значущості. **Матеріали та методи.** Порівняльне поперечне дослідження було проведено в лікарні Аль-Хуссейн з грудня 2024 року по червень 2025 року. У ньому взяли участь 60 пацієнтів з термінальною стадією ХХН віком від 25 до 72 років, які тривалий час отримували гемодіаліз. Також у дослідження було включено 30 здорових осіб контрольної групи, порівнянних за віком та статтю. Рівні сечовини, креатиніну, кальцію та фосфору в сироватці крові вимірювали та аналізували за допомогою стандартних біохімічних методів. Статистичний аналіз проводили за допомогою програми SPSS версії 26 із рівнем значущості $p < 0,05$. Для аналізу зв'язків між параметрами використано коефіцієнт кореляції Пірсона. **Результати.** Порівняно зі здоровими особами контрольної групи пацієнти із хронічною хворобою нирок мали значно вищі сироваткові рівні фосфору ($5,37 \pm 0,47$ мг/дл) та креатиніну ($7,46 \pm 1,15$ мг/дл), а також нижчий уміст кальцію ($5,54 \pm 0,41$ мг/дл) ($p < 0,0001$ для всіх). Зв'язок між креатиніном та фосфором був дещо позитивним ($r = 0,54$), однак рівні кальцію та фосфору мали негативну кореляцію ($r = -0,30$). **Висновки.** Гіперфосфатемія та гіпокальціємія є досить поширеними явищами в осіб із прогресуючою ХХН, і обидва стани безпосередньо пов'язані зі зниженою функцією нирок. Рівень фосфору може бути використаний як сурогатний показник ниркової недостатності, що підтверджується його незначною кореляцією з умістом креатиніну. Регулярний моніторинг рівнів кальцію та фосфору на додаток до традиційних показників є важливим для раннього виявлення порушень мінерального обміну та своєчасного лікування ускладнень, пов'язаних із ХХН.

Ключові слова: хронічна хвороба нирок; біомаркери; гіперфосфатемія; гіпокальціємія; гемодіаліз; порушення мінерального обміну; креатинін; фосфатно-кальцієва вісь