IRAQI Academic Scientific Journals
Samarra Journal of Pure and Applied Science
SJPAS
مجلة سامراء للعلوم الصرفة والتطبيقية
www.sjpas.com
p ISSN: 2663-7405
e ISSN: 2789-6838

Possible effects of Osteocalcin and some biochemical parameters on kidney Function in Iraqi Diabetic Patients

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https://doi.org/10.54153/sjpas.2025.v7i4.1175

Article Information
Received: 19/03/2025
Revised: 15/06/2025
Accepted: 22/06/2025
Published: 30/12/2025

Keywords:
Biochemical parameters, Chronic Kidney Disease, Diabetic patients. Kidney function, Osteocalcin

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Abstract

Chronic kidney disease (CKD) is a severe medical illness that is only worsening. Chronic renal failure (CKF) is the end stage of CKD, which is characterised by increasing loss of kidney function. We were to see if diabetes played a role in how osteocalcin (OCN) affected people who had renal failure. Between November 2021 and January 2022, 40 patients—20 with chronic renal disease with diabetes mellitus (T2DM) and 20 without—were transferred from a hospital to Emamain Al Khadhemain-education. The organisation is in charge of 28 healthy people. Minute. Each sample was separated into blood serum for ten minutes at 3000 revolutions per minute using the centrifuge. Age, BMI, and gender equality were among the information gathered from each participant. The Medical Research Unit of the University of Rivers' Faculty of Medicine conducted this investigation. In comparison to the control group (27.70 ± 3.46 mg/dL), CDK without T2DM and CDK with T2DM both displayed a substantial decrease in OCN (7.32 ± 1.52, P-value = <0.001; 12.35 ± 2.38, P-value = <0.001). Along with variations in calcium, phosphorus, urea, creatinine, vitamin D3, and other parameters, there were variations in insulin and fat.

Introduction

Research has extensively explored how the bone-kidney axis contributes to the physiological regulation of mineral ion concentrations. Numerous studies have shown that malfunctioning in one organ can affect the functioning of the other organ. For instance, people with chronic kidney disease (CKD) may exhibit a range of problems associated with anomalies of the bones, such as extra skeletal calcification (coming from a dysregulation of mineral ions) or fractures (resulting from an increased fragility of the bones). Skeletal anomalies in CKD-mineral bone disease (CKD-MBD) are primarily explained by reduced osteoblastic differentiation, which impacts the anabolic responses of the bone [1].

Osteocalcin (OCN), comprising around 46 to 50 amino acids, is a vitamin K-dependent, non-collagenous protein produced predominantly by bone-forming osteoblasts [2]. Research has indicated that elevated blood levels of OCN are related to lower bone density and a greater risk of fractures, notably hip fractures, in aging populations, a consequence of increased release of

OCN from resorbed bone tissue [3]. Circulating OCN is eliminated by the kidney and liver [4], and it builds up in the blood as renal function declines. Parathyroid hormone (PTH) intact and alkaline phosphatase levels are strongly associated with the progressive rise in serum OCN levels observed in CKD patients. More significant indicates the severity of the bone lesions [5].

Bone strength is dependent on both its quantity and composition. The most prevalent non-collagenous protein in bone is called OCN, yet little is known about its purpose. Previous research by other researchers has shown that OCN operates as a hormone to control muscle hypertrophy, testosterone synthesis, and glucose metabolism in distant tissues. It also appears to reduce bone mass by inhibiting the formation of new bone [6]. Osteoblasts create most of the non-collagenous bone protein, called OCN. The protein has three carboxylated glutamic acid residues. Uncarboxylated OCN has limited affinity for Ca^{2+}, whereas carboxylated OCN shows strong attraction to it and adopts an α-helical shape upon binding. The mechanisms of mineralization, hydroxyapatite growth inhibition, and osteoclast precursor chemotactic activity have all been linked to carboxylated OCN [7].

In those with CKD, high serum phosphate levels have been associated with detrimental health outcomes such as cardiovascular disease, the advancement of renal disease, and overall mortality [8,9]. High blood phosphorus levels have been associated with an increased risk of developing CKD and cardiovascular diseases. However, studies have not adequately addressed the relationship between phosphorus consumption in food and the start of CKD [10].

Due to higher dietary phosphorus density, patients with diabetes who have normal renal function are more likely to develop CKD. In cases of chronic kidney dysfunction, the kidneys exhibit an impaired ability to regulate serum phosphorus levels, regardless of significant fluctuations in dietary intake [11]. Normal renal physiology ensures a phosphorus, irrespective of dietary and gastrointestinal factors, preserving a tight serum phosphate range. A disruption in this process, manifested by elevated serum phosphate due to impaired excretion, is a hallmark of end-stage renal failure [1].

Proper evaluation of renal function is critical for the clinical management of patients suffering from kidney disease or other disorders influencing kidney activity. Renal function tests facilitate early diagnosis, guide therapeutic monitoring, and help assess disease evolution over time. The National Institutes of Health estimates that 14% of people worldwide have CKD. Diabetes and hypertension are the two leading causes of CKD worldwide [9, 12-14].

Serum OCN levels were higher in patients with chronic renal disease. It was found that elevated circulating levels of OCN, rather than poor renal filtration, suggest faster bone turnover in both normal individuals and patients with mild to severe renal impairment [15]. Individuals suffering from CKD may exhibit excessive levels of OCN build-up in their serum due to either reduced renal clearance, higher bone metabolism, or both. In patients with CKD, serum OCN levels are continuous, closely associated with PTH and alkaline phosphatase, and are related to PTH. Interestingly, it is interesting to note that these increases in serum OCN levels correspond with the severity of bone lesions [2]. Recent investigations emphasize the regulatory roles of bone-derived elements over various bodily systems, such as renal, parathyroid, muscular, adipose, and pancreatic functions. For instance, Fibroblast growth factor 23 (FGF23), which is produced from bone, is an essential modulator of renal phosphate

metabolism [16]. It is unknown why people with CKD are yet more prone to develop insulin resistance. Therefore, further research with a carefully constructed study is needed to investigate whether there is reduced bone turnover; thus, further research with a carefully constructed study is necessary to examine whether reduced bone turnover is required. Therefore, further research with a carefully constructed study is needed to determine whether reduced bone tumor activity in CKD is due to it. Recent studies have revealed that it affects energy consumption [17,18].

Several studies have emphasized the critical role of the bone-kidney axis in maintaining mineral ion homeostasis and regulating systemic mineral metabolism. Numerous investigations have conclusively shown that the functioning of one organ can be impacted by sickness in another. For instance, abnormalities in bone often result in extra skeletal calcification (caused by an imbalance of mineral ions) and fractures (caused by increased osteoporosis) in individuals with CKD. In CKD-MBD, skeletal abnormalities are partially attributed to impaired osteoblast differentiation, leading to a diminished anabolic response of osteoblasts. Several investigations have identified a proportional association linking insulin resistance with progressive impairment in kidney function [19]. A complex cation such as calcium is often misunderstood; essential positive and negative calcium balances are crucial to people with chronic renal illness. Enhancing dietary calcium intake, such as calcium carbonate supplementation, promotes calcium retention, whereas insufficient intake may predispose individuals to osteoporosis. Raising calcium intake has little effect on the extremely low calcium levels found in the urine of people with CKD. Low calcium levels in the urine are caused by low calcium absorption. Additionally, the research demonstrated that the blood's calcium content does not precisely reflect the body's total calcium content because elevated blood calcium levels completely restore the body's calcium homeostasis while blood calcium levels stay constant [20].

The main risk factor for decreased kidney function is T2DM. The markers of chronic renal damage in diabetes are impairment of urea and creatinine. Even though T2DM-related renal function impairment is very burdensome, there aren't many local data on urea and creatinine impairment.

This case-control study aimed to assess the levels of OSN in the sera of diabetic patients, compare these levels with those of healthy controls, and investigate the correlations between OSN and various biochemical parameters.

Materials and Methods

The study included 40 diseases in the Governorate, divided equally into two groups. The participants' ages ranged from 20 to 55. The study also included 20 samples of healthy individuals in the same age group. The samples were collected from patients attending Al-Kadhimiya Teaching Hospital in Baghdad Governorate in November 2021 and January 2022.

The samples were collected from the patients based on laboratory tests. They were diagnosed by a nephrologist and found to have chronic kidney disease. 20 patients were classified as having both kidney disease and diabetes, and 20 patients had chronic kidney disease but not diabetes.

Serum Preparation

Blood samples were collected by withdrawing 5 ml of venous blood using a sterile, single-use medical syringe. The samples were placed in plastic tubes with a tight cap and were free of anticoagulants. The tubes were left at room temperature (25°C) until coagulation occurred. They were then centrifuged for 10 minutes at 3,000 rpm. The serum was then withdrawn using a micropipette, and the resulting serum was divided into five aliquots in Eppendorf tubes to avoid sample degradation during thawing and repeated freezing. The serum was stored in a freezer at -20°C until the required tests were performed.

Sample Concentration Measurement

Cobas C 111 (Roche, Germany) assesses glucose, urea, OCN levels, and lipid profiles. Insulin, vitamin D, OCN levels, creatinine levels, and lipid profiles were measured and evaluated using the ELISA technique (Diagnostics Automation, USA).

Statistical Analysis

The statistical program (SPSS) was used, using Duncan's test to compare the three groups at a probability level of 0.05 $p < 0.05$, to calculate the mean and standard deviation (SD).

Results and Discussion

Age of patients and control groups

Table1 and Figure1 shows the ages of studied groups, matching of age between groups is essential for the best statistical implications. Our results show that there was non-significant difference between patients and control with a p-value of 0.58.

Table 1: Age in the studied groups.

Factor	Controls	CDK without T2DM	CDK with T2DM	P-value
Age (Years)	34.95±5.64	44.10±3.37	45.40±4.78	0.58

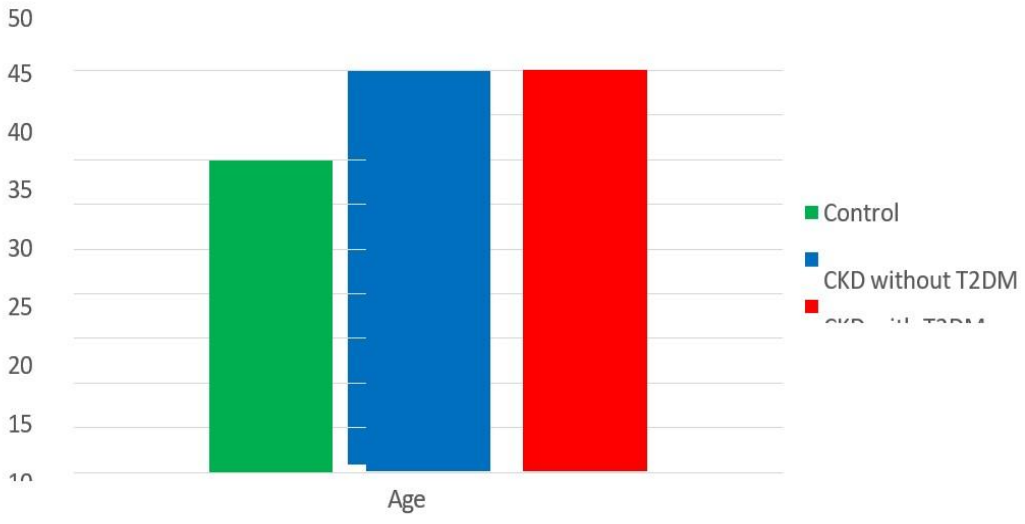

Fig. 1: Age in the studied groups.

Biochemical parameters in patients and control

Lipid Profile and BMI

The Comparison of lipid profile and BMI as shown in table 2 and figure 2 between studied groups. There was significant difference p-value≤0.05 between CKD with T2DM when compared with the control group in all parameters mention in the table2 (BMI and lipid profile). Also, there were significant difference p-value<0.05 between CKD without T2DM when compared with control group, only we have non-significant difference p-value ≥0.05 in all parameters of lipid profile and BMI when compare CKD patients with and without T2DM.

The explanation of this difference could refer that CKD is linked to a dyslipidemia characterized by high levels of TGs and low levels of HDL-C. The levels of LDL-C (and total cholesterol as a result) are usually not high; nonetheless, there is a correlation between proteinuria and cholesterol as well as TGs. CKD causes a reduction in lipoprotein lipase and LDL receptor activity, while the rise in TGs during CKD is attributed to a slower breakdown of TG-rich lipoproteins, with production rates remaining unchanged.

Table 2: Comparison of lipid profile and BMI in studied groups.

Parameter	Control (mean±SD)	CKD without T2DM (mean±SD)	CKD with T2DM (mean±SD)	P-value Control vs. CKD without T2DM	P-value Control vs. CKD with T2DM	P-value CKD without T2DM vs. CKD with T2DM
BMI	25.14±3.5	28.48±4.17	30.32±4.21	0.027	<0.001	0.316
HDL	53.75±5.34	44.85±3.08	46.75±6.26	<0.001	<0.001	0.468
VLDL	19.23±2.2	36.49±3.22	42.04±15.61	<0.001	<0.001	0.151
LDL	79.27±23.35	214.14±21.35	236.89±61.55	<0.001	<0.001	0.179
Cholesterol	152.25±22.03	222.50±20.77	241.60±55.28	<0.001	<0.001	0.230
Triglyceride	96.15±11.02	182.45±16.12	210.20±78.07	<0.001	<0.001	0.151

* Data are presented as mean ± standard deviation (SD). Statistical significance was evaluated using ANOVA followed by post hoc analysis.

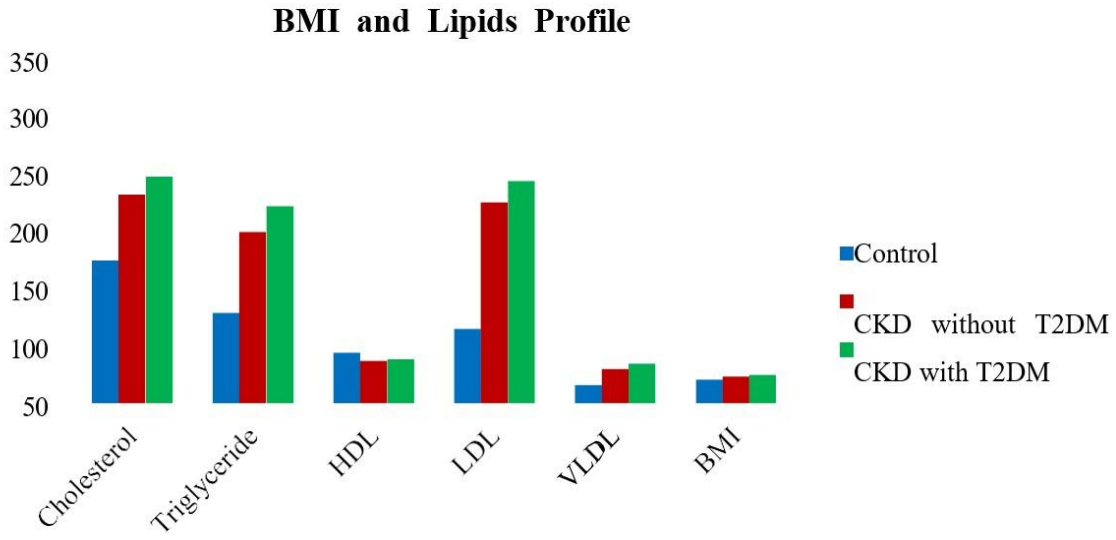

Fig. 2: Comparative Analysis of Lipid Profiles and BMI Among Study Groups.

OCN, Vitamin D3 and Insulin

The mean serum OCN levels were markedly reduced in both CKD without T2DM (7.32 ± 1.52 mg/dL) and CKD with T2DM (12.35 ± 2.38 mg/dL) compared to the control group (27.70 ± 3.46 mg/dL), with a highly significant difference ($p < 0.001$). Regarding vitamin D3, a considerable decline was detected in CKD without T2DM (8.81 ± 1.51 mg/dL) and CKD with T2DM (11.84 ± 1.50 mg/dL) relative to the control subjects (33.20 ± 3.66 mg/dL), with the comparison between the two CKD groups also showing statistical significance ($p = 0.001$). Meanwhile, insulin concentrations appeared elevated in both CKD groups when compared to controls (control: 12.53 ± 4.30 mg/dL; CKD without T2DM: 16.65 ± 5.36 mg/dL; CKD with T2DM: 16.44 ± 5.81 mg/dL). However, the difference between CKD patients with and without diabetes did not reach statistical significance ($p = 0.991$), as shown in Table 3 and Figure 3.

Table 3: Comparison of OCN, Vitamin D3, and Insulin levels among the studied groups.

Parameter	Control	CDK	Parameter	Control	CDK	Parameter
OCN	27.7±3.46	7.32±1.52	12.35±2.38	0<0.001	0<0.001	0<0.001
Vitamin D3	33.2±3.66	8.81±1.51	11.84±1.5	0<0.001	0<0.001	0.001
Insulin	12.53±4.30	16.65±5.36	16.44±5.81	0.039	0.054	0.991

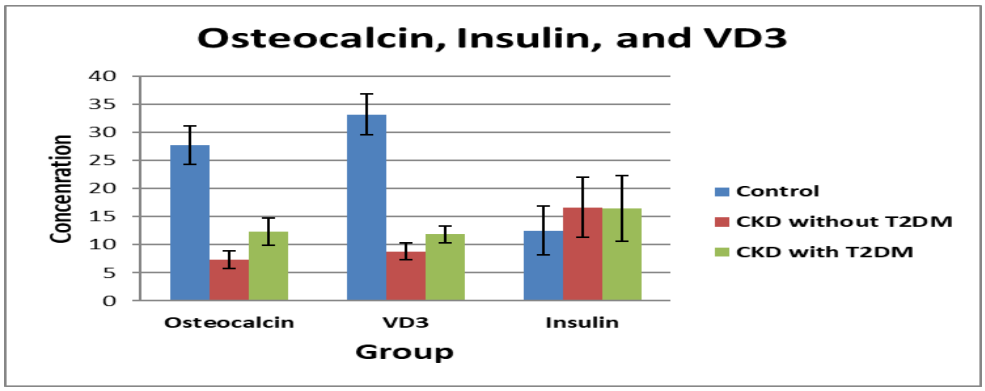

Fig. 3: Serum OCN, Vitamin D3, and Insulin Levels in the different study groups.

Patients with CKD exhibited elevated serum OCN levels, which may be due to reduced renal clearance and heightened bone metabolism. The rises in serum OCN levels that accompany the advancement of kidney disease are strongly associated with PTH levels [13,14]. Post-transplantation, the levels of OCN and PTH decrease gradually as renal function is restored [15]. OCN was regarded as a marker of bone turnover because it is released into the circulation in its uncarboxylated form after osteoclast-mediated bone resorption [13]. Additionally, OCN functions similarly to a hormone in the modulation of glucose metabolism; OCN levels that are lower than normal are frequently associated with metabolic disease and diabetes mellitus [16]

Calcium and phosphorus

Serum calcium concentrations were notably reduced in patients with CKD, both without T2DM (7.67 ± 0.92 mg/dL) and with T2DM (7.18 ± 1.03 mg/dL), compared to the control group (9.27 ± 0.34 mg/dL), with highly significant p-values (<0.001). However, the difference in calcium levels between the two CKD groups was not statistically significant ($p = 0.154$). Regarding serum phosphorus, levels decreased significantly in CKD without T2DM

(2.54 ± 0.68 mg/dL) and CKD with T2DM (3.00 ± 0.53 mg/dL) in comparison to controls (3.81 ± 0.19 mg/dL), with p-values also <0.001. In contrast to calcium, phosphorus levels showed a significant difference between the CKD subgroups (p = 0.017), as summarized in Table 4 and illustrated in Figure 4.

CKD results in reduced kidney function, which disrupts calcium and phosphorus metabolism. This disruption often leads to imbalances that impact bone health and elevate the risk of cardiovascular issues. In particular, high phosphorus levels combined with low calcium levels can result in weakened bones, a heightened risk of heart attack or stroke, and potentially fatal outcomes [20-22].

Table 4: Comparison of serum calcium and phosphorus levels among the studied groups.

Factor	Control	CKD without T2DM (mean ± SD)	CKD with T2DM (mean ± SD)	p-value (Control vs. CKD without T2DM)	p-value (Control vs. CKD with T2DM)	p-value (CKD without vs. with T2DM)
Phosphorus	3.81±0.19	2.54±0.68	3.00±0.53	0.000	0.000	0.017
Calcium	9.27±0.34	7.67±0.92	7.18±1.03	0.000	0.000	0.154

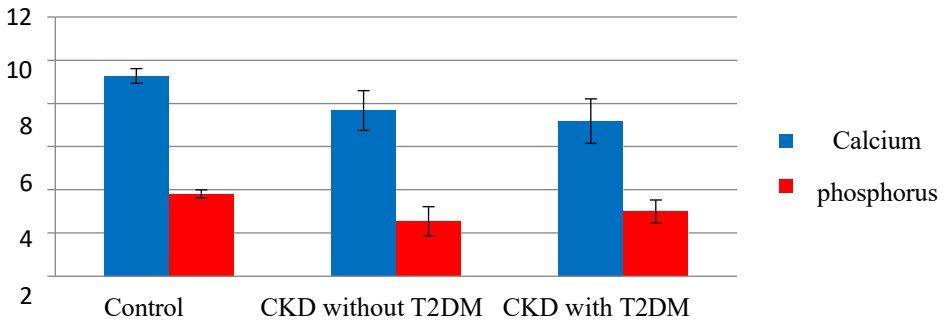

Fig. 4: Serum calcium and phosphorus levels in the different study groups

Creatinine and urea in studied groups

As illustrated in Table 5 and Figure 5, patients with CKD, regardless of T2DM, exhibited significantly elevated serum creatinine levels compared to the control group. Specifically, creatinine levels averaged 1.17 ± 0.45 mg/dL in CKD without T2DM and 1.08 ± 0.35 mg/dL in CKD with T2DM, both markedly higher than the control group's 0.63 ± 0.13 mg/dL (p < 0.001 for both comparisons). However, the difference between CKD subgroups was not statistically significant (p = 0.686).

Table 5: Comparison of serum creatinine levels across the studied groups.

Factor	Control	CKD without T2DM (mean ± SD)	CKD with T2DM (mean ± SD)	p-value (Control vs. CKD without T2DM)	p-value (Control vs. CKD with T2DM)	p-value (CKD without vs. with T2DM)
Creatinine	0.63±0.13	1.17±0.45	1.08±0.35	0.000	0.000	0.686

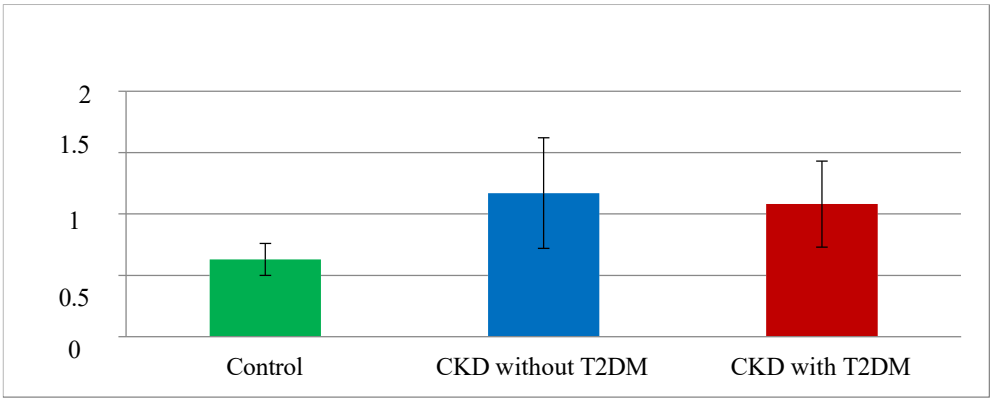

Fig. 5: Serum creatinine levels in control and CKD patients with and without T2DM.

As presented in Table 6 and Figure 6, serum urea concentrations were significantly elevated in CKD patients, regardless of T2DM status, compared to the control group. Specifically, mean urea levels reached 53.50 ± 7.50 mg/dL in CKD without T2DM and 49.60 ± 15.31 mg/dL in CKD with T2DM, both showing a highly significant difference from the control value of 33.40 ± 7.91 mg/dL ($p < 0.001$ for both comparisons). Analysis indicated that the two CKD subgroups did not differ significantly ($p = 0.485$).

Table 6: Serum Urea levels in the studied groups.

Factor	Control	CKD without T2DM (mean ± SD)	CKD with T2DM (mean ± SD)	p-value (Control vs. CKD without T2DM)	p-value (Control vs. CKD with T2DM)	p-value (CKD without vs. with T2DM)
Urea	33.40±7.19	53.50±7.50	49.60±15.31	0.000	0.000	0.485

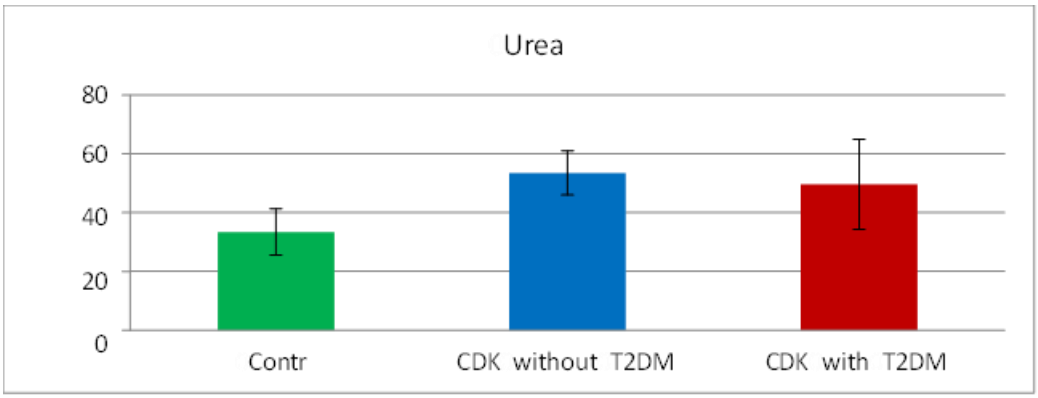

Fig. 6: Serum Urea levels control and CKD patients with and without T2DM.

Table 7. shows the difference between male and females in OCN, D3, and Insulin. The direct effect of sex on the progression of CKD is not evident in individuals. Nevertheless, while monitoring different paths of estimated glomerular filtration rate decline, no sex difference was noted in patients with nonglomerular disease; however, CKD progressed more rapidly in females with glomerular disease. In a similar vein, a prospective investigation into the risk factors for the overall progression of patients with CKD, the study revealed no differences based on gender in insulin but with significant difference in OCN and D3.

Table 7: Comparison of OCN, Vitamin D3, and Insulin levels between males and females.

Parameters	Patients male Mean±SD	Patients Female Mean±SD	P-value
OCN	33.7±4.46	50.7±9.45	0.04
Vitamin D3	20.2±3.66	16.2±3.97	0.018
Insulin	7.53±4.10	9.53±4.34	0.33

Existing studies have demonstrated that bone marrow-derived osteoclasts increase bone disintegration as oestrogen levels fall in the postmenopausal age. A high rate of bone turnover results from greater bone production, which is driven by increased bone breakdown. Bone synthesis and breakdown are mutually reinforcing and supportive [6]. Patients with postmenopausal type 2. These face the exact opposite problem. T2DM patients have a higher rate than those with T1DM or osteoporotic postmenopausal women [5]. When measures of bone turnover were investigated, results revealed a decrease in bone turnover, particularly in people with well-controlled diabetes [12]. Furthermore, this clarifies the phenomenon of delayed bone loss seen in T2DM patients [8]. Kestenbaum et al. [10] discovered that persons with type 2 diabetes did not experience a significant reduction in bone mineral density until twenty years after the disease was diagnosed. Also found a delayed bone loss pattern in people with type 2 diabetes.

Insulin resistance in peripheral tissues, alongside β-cell dysfunction, is recognized as a key pathophysiological mechanism in the progression of type 2 diabetes mellitus (T2DM). This resistance often stems from impaired insulin signaling and dysregulated insulin secretion, exacerbating hyperglycemia [19]. Clinically, exogenous insulin administration is commonly employed to mitigate the consequences of persistent hyperglycemia and maintain glycemic control [13]. At the cellular level, insulin resistance is characterized by deficient transmission of signals from the insulin receptor to downstream substrates involved in critical metabolic and mitogenic pathways [24].

Vitamins have an essential role in glucose metabolism. Upon entering pancreatic β-cells, vitamin D binds to the vitamin D receptor (VDR), facilitating the activation of insulin gene transcription and promoting insulin secretion [22]. VDR expression has been identified in multiple tissues responsive to insulin, including pancreatic β-cells and adipocytes [19]. Emerging evidence suggests that vitamin D may enhance β-cell function, improve insulin sensitivity, and attenuate systemic inflammation, thereby supporting metabolic homeostasis [16].

The results of this study suggest a connection between an elevated risk of developing CKD and high LDL and low HDL levels. These results are consistent with previous findings [21]. Chang and associates also confirmed that high LDL levels are still a risk factor for osteoporosis, even though low HDL levels have been linked to an increased risk [4]. Only limited investigations have explored the link between dyslipidemia, osteoporosis, and CKD [5,6]. Hyperphosphatemia typically occurs in advanced CKD stages, especially when the remaining

nephrons fail to eliminate excess phosphorus to combine calcium and phosphorus adequately. Calcium and phosphorus may combine within the circulatory system, resulting in an insoluble complex in the aqueous environment. In addition to heart illness or calciphylaxis, this process may result in extraskeletal calcification [2]. Phosphorus retention insinuates excessive PTH production and release indirectly by inhibiting the generation of calcitriol and lowering ionised Ca2+ levels. The findings are consistent with those demonstrating that increased serum PTH levels in end-stage CKD are positively connected with serum phosphorus and negatively correlated with serum GFR and serum calcium [3]. It is commonly known that the many cascades that cause renal bone disease are primarily triggered by hyperphosphatemia [20]. The current study observed that blood urea and creatinine levels were markedly increased in end-stage CKD patients compared to earlier stages (3–4), corroborating previous literature [25]. This is linked to patients with end-stage renal disease experiencing a steady drop in GFR. As GFR decreases, blood levels of urea and creatinine rise [25]. A significant correlation between elevated blood PTH levels and the end stage of chronic renal disease has also been reported [25]. These findings align with previous studies showing that serum PTH concentrations progressively increase as renal dysfunction advances, particularly in patients reaching stage 5 CKD. This highlights the strong association between hyperparathyroidism severity and the extent of kidney impairment [20]. A significant side effect of CKD is a loss of renal function, which can result in secondary hyperparathyroidism [20]. This decrease in renal function causes an increase in the synthesis of PTH because CKD directly causes several changes in bone and mineral metabolism.

Evidence indicates that individuals with chronic kidney disease (CKD) frequently present with elevated serum phosphorus levels (hyperphosphatemia) and reduced calcium concentrations (hypocalcemia), both of which are hallmark features of advanced secondary hyperparathyroidism [20]. Conversely, subjects with preserved renal function typically maintain normal mineral metabolism and do not exhibit these biochemical abnormalities [25].

Conclusions

This study examines the relationship between CKD and type 2 T2DM, and it has been confirmed that CKD significantly raises urea and creatinine levels, which substantially affect and reveal biochemical markers. It has been confirmed that CKD significantly raises urea and creatinine levels, which affects and reveals several significant findings. Elevated bone calcium levels associated with CKD suggest increased bone turnover. It has been confirmed that CKD significantly raises urea and creatinine levels, which affect the development of type 2 diabetes (T2DM). In addition, patients with CKD showed lower glomerular filtration rates (GFR) compared to controls, indicating decreased kidney function.

Also, significant decreases in vitamin D levels were observed in controls and CKD patients, suggesting that CKD plays a role in hyperlipidemia and vitamin D deficiency. Hyperlipidemia was also common in CKD patients, with markedly high levels of Cholesterol, triglycerides, and high—and low-density lipoproteins. Quantitative changes further complicate the management of CKD.

High levels of OCN are associated with increased bone turnover and may play a role in CKD. The results showed that OCN levels were significantly lower in CKD patients with or without

T2DM compared to the control group. This suggests that OCN may play an essential role as a marker in bone health and turnover in these patients.

Thus, this study highlights the complexity of biochemical interactions in CKD patients, stressing the need for comprehensive management strategies that are not limited to kidney function but also emphasize the management of associated metabolic disorders such as T2DM and lipid metabolism.

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IRAQI Academic Scientific Journals
Samarra Journal of Pure and Applied Science
SJPAS
مجلة سامراء للعلوم البحتة والتطبيقية
www.sjpas.com
p ISSN: 2663-7405
e ISSN: 2789-6838

التأثيرات المحتملة للأوستوكالسين وبعض المعايير الكيميائية الحيوية على وظائف الكلى لدى مرضى السكري العراقيين

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البحث مستل من رسالة الماجستير الباحث الأول

معلومات البحث:
تاريخ الاستلام: 2025/02/10
تاريخ التعديل: 2025/03/10
تاريخ القبـــول: 2025/03/15
تاريخ الـنشر: 2025/12/30
الكلمات المفتاحية:
مرض الكلى المزمن، أوستيوكالسين، المعايير الكيميائية الحيوية، وظائف الكلى، ومرضى السكري.

معلومات المؤلف
الايميل:
الموبايل:

الخلاصة:
مرض الكلى المزمن هو مرض خطير يزداد سـوءًا.ويعد الفشـل الكلوي المزمن هو المرحلة النهائية من مرض الكلى المزمن، والذي يتميز بفقدان متزايد لوظائف الكلى. كان هدف هذه الدراسة هو معرفة مستوى الاوستيوكالسين لدى مرضى السكري الذين لديهم عجز كلوي .تم جمع العينات خلال الفترة من تشـرين الثاني 2021 الى كانون الثاني 2022، قسمت العينات الى 20 مريض بالعجز الكلوي والسكري النوع الثاني معا و20 مريض فقط لديه عجز كلوي بدون سكري و28 شخص اصحاءوتم جمع العينات من مستشفى الإمامين الكاظمين التعليمية. باستخدام جهاز الطرد المركزي تم الحصـول على مصـل الدمواسـتخدام الجهاز بمعدل 3000 دورة في الدقيقة لمدة عشـر دقائق. تم مطابقة الاعمار لمجاميع الدراسـة.تم أجراء الجزء العملي في وحدة الأبحاث الطبية بكلية الطب بجامعة النهرين. وبالمقارنة بمجموعة المرضـــى مع الاصـــحاء كان مســـتوى الاوســـتيوكالســـين منخفض معنويا (27.70 ± 3.46 مجم/ديسـيلتر)، أظهر كل من CDK بدون مرض السـكري من النوع الثاني و CDK مع مرض السكري من النوع الثاني انخفاضًا كبيرًا في أوستيوكالسين) 7.32 ± 1.52 p=0.001 (و12.35 ± 2.38: قيمة< P = 0.001 .(إلى جانب الاختلافات في الكالسيوم والفوسفور واليوريا والكرياتينين وفيتامينD وغيرها من المعايير، كانت هناك اختلافات في الأنسولين والدهون.