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Determination of Xanthine Oxidase and Calprotectin and Some Biochemical Parameters in Diabetic Nephropathy Serum of Iraqi Patients

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Abstract **Objective:** Diabetic Nephropathy (DN) is a major long-term severe microvascular complication of diabetes mellitus and the current leading cause of end-stage kidney disease. It accounts for up to 40% of patients requiring renal replacement therapy. The risk of end-stage kidney failure has been associated with an increase in the level of a new biomarker, xanthine oxidase and calprotectin, used to assess the severity of kidney injury. **Methods**: Blood samples were collected from 30 individuals diagnosed with DN disease and 30 individuals with diabetes but without kidney disease, who visited the National Diabetes Centre at the University of Al-Mustansiriyah, along with 28 control subjects. An enzyme-linked immunosorbent assay was used to assess serum xanthine oxidase (XOD) and Calprotectin (Calp) and a UV-Visible spectrophotometer was used to evaluate other biochemical parameters. **Results**: The mean serum XOD activity (2.04±0.38 ng/ml) in DN was significantly greater than the corresponding values for the controls (0.95±0.12 ng/ml) and the mean serum Calp (173.46±29.56 ng/ml) in DN Patients was significantly greater than the corresponding values for the controls (43.26±5.88 ng/ml). The optimal cut-off value for CALP sensitivity of 86.3% and specificity of 82.1% and the AUC for control and DN is 0.844 and for control versus diabetic, it is 0.853, while XOD activity showed a high sensitivity of 76.5% and specificity of 89.9% to distinguish between control and DN, so the results show the AUC = 0.833 for control vs. DN and AUC for control and diabetes is 0.863. These high sensitivity and specificity values highlight the reliability of CALP and XOD as significantly associated with diabetic nephropathy.

Key Words Calprotectin, Diabetic Nephropathy, Xanthine Oxidase, ROC

INTRODUCTION

A class of metabolic disorders known as diabetes mellitus is typified by hyperglycaemia brought on by deficiencies in either insulin action or secretion or both. Diabetes-related chronic hyperglycaemia is linked to long-term harm, malfunction and failure of many organs, particularly the heart, blood vessels, kidneys, eyes and nerves. Therefore, preventing diabetes and its complications is of utmost importance. The kidneys are affected by diabetes in phases and enlarges and the glomerular filtration rate (GFR) is disrupted when diabetes first appears. The majority of current scientific and clinical research has suggested kidney failure and sclerosis. Regular screening, early detection and adequate treatment of chronic problems can all help lower the morbidity and mortality rates associated with diabetes mellitus. Thus, kidney development will be the main topic of our conversation [1].

Globally, diabetic nephropathy is now the leading cause of end-stage renal disease (ESRD). In order to enhance cardiovascular and renal outcomes for individuals with chronic kidney disease, it is still crucial to manage traditional risk factors like smoking, hypertension and hyperlipidaemia. However, there is emerging acknowledgment that nontraditional risk factors, such as decreased haemoglobin levels, raised serum creatinine levels, hypoalbuminemia, and/or increased urine albumin excretion, may potentially be significant in people with chronic renal disease [2]. High blood sugar levels cause the kidneys to create too much reactive oxygen species (ROS), which may aggravate diabetes-related renal problems. The underlying mechanisms of DN disease encompass elevated blood sugar levels, various hemodynamic factors, changes in metabolism, inflammatory responses and the development of fibrous tissue [3].

The primary source of reactive oxygen species (ROS) production in the bloodstream is xanthine oxidase (XOD), a crucial enzyme that converts purine bases to uric acid [4]. Additionally, clinical research has suggested a strong correlation between poor glycaemic management and increased xanthine oxidoreductase (XOR) activity [5,6]. Vascular endothelial impairment is independently linked to higher XOR activity in diabetics. Thus, by reducing ROS and oxidative stress, xanthine oxidoreductase (XOR) suppression may prevent DKD better than uric acid regulation [7].

Calprotectin (CALP) is a heterodimer containing S100A8 and S100A9 monomers. It is found in neutrophil cytosols, monocyte membranes and inflammatory cell membranes. Every monomer features two binding sites for Ca^{+2} in the N- and C-terminal EF hands, linked by a hinge area. It is released from these cells upon activation or adhesion to the endothelium [8,9]. Due to the over-expression of CALP, it has been found that the blood concentrations of this inflammatory marker are elevated in various chronic low-grade inflammatory conditions, including obesity and insulin resistance [10,11].

The condition known as diabetes mellitus and its subsequent complications, including high blood pressure and kidney failure, have been linked to increased levels of CALP in both plasma and urine [12]. CALP exhibits a range of functions related to living organisms. This acts as an indicator for the differentiation of phagocytes intracellularly and as a substance related to damage that communicates with different receptors extracellularly [13,14]. The stimulating effect of Toll-like receptor 4 (TLR4) and glycation result (AGE) receptors plays a significant role in the underlying mechanisms of micro- and macrovascular complications associated with diabetes mellitus (DM). A soluble protein in white blood cells, calprotectin is actively released by phagocytes during the stress response. It is linked to inflammation and is reported to be elevated in individuals with insulin resistance [15].

METHODS

Blood samples were collected from 30 individuals (15 female and 15 male) diagnosed with DN disease, alongside 30 individuals (15 female and 15 male) with diabetes but without kidney disease, who visited the National Diabetes Centre at the University of Al-Mustansiriyah. Additionally, a control group of 28 individuals (14 female and 14 male) was included from December 2024 to February 2025, with all participants aged between 35 and 70. The diagnosis had been previously established by the treating physicians according to the medical records and the diagnostic criteria adopted at the centre (ADA/KDIGO guidelines).

A volume of 8 mL of venous blood was collected for every sample, in addition to 6 mL being collected in an anticoagulant-containing serum separator tube, after 30 minutes of clotting at room temperature, the sample was centrifuged at 3000 rpm for 10 minutes. After careful removal, the serum was aliquoted into storage tubes. A spectroscopy instrument, such as a UV-Visible spectrophotometer, directly measures biochemical markers such as FBG, cholesterol, triglycerides and kidney function. Both XOD and CALP were measured using ELISA kits from Shanghai Biological (China) and Cusabio (China); the other two components were maintained at -20°C, as well as all study groups had their BMIs determined.

The Excluded Criteria The included diabetic patients are free from acute illness or infection at the time of the study. Those with diseases were excluded such as, high blood pressure, smoking, chronic liver disease and heart diseases.

Statistical Methods

This study, Analysis System 9.1 used to analyse data and evaluate significant differences ($p<0.05$), before performing one-way ANOVA, the assumptions of normality and homogeneity of variances were assessed. Independence of observations was ensured by the study design. Since these assumptions were met, one-way ANOVA was performed to compare the three study groups and LSD used SPSS-19 to calculate the correlation coefficient (r) and the ROC curve analysis used to assess each illness indicator diagnosis accuracy.

RESULTS

Table 1 shows that the DN disease group had higher FBG, BMI, urea, UA, creatinine, cholesterol and triglycerides compared to the diabetic and control groups. The DN group had significantly higher fasting blood glucose levels (397.93±14.99 mg/dl), nearly tripling the diabetic group (229.7±8.26 mg/dl) and quadrupling the control group (83.75±1.91 mg/dl) at $p = 0.05$. DN patients had metabolic problems and renal impairment, indicated by BMI, urea and uric acid levels. Although less pronounced than in the DN group, the diabetic group still differed from the control group. Triglyceride levels in the diabetic group (302.77±16.53 mg/dl) were substantially higher than those in the control group (72.89±3.48 mg/dl), confirming diabetes-related dyslipidaemia.

The both CALP and XOD levels in DN, diabetic and control groups are compared in Table 2, at $p = 0.05$ shows the

Table 1: DN, Diabetic and Control Group Parameter Comparison.

Groups	FBG mg/dl	BMI	Urea mg/dl	U.A mg/dl	Creatinin mg/dl	Chol. mg/dl	Tri. mg/dl
DN	397.93± 14.99 a	41.97±1.15 a	90.2±7 a	9.98±0.26 a	2.32±0.15 a	337.7±17.33 a	352.1±14.49 a
Diabetic	229.7± 8.26 b	38.71±0.76 b	54.93±3.00 b	7.04±0.30 b	1.55±0.09 b	233.07±7.14 b	302.77±16.53 b
Control	83.75±1.91 c	34.57±1.02 c	39.86±1.25 c	4.73±0.29	0.91±0.07 c	108.36±4.13 c	72.89±3.48 c
LSD	35.48*	16*	15.86*	0.84*	0.36*	39.68*	43.84*

* The significant differences between groups were shown at $p = 0.05$.

Table 2: CALP and XOD Levels for DN, diabetic and control groups.

Parameters	CALP ng/ml	XOD ng/ml
DN	173.46±29.56 a	2.04±0.38 a
Diabetic	78.2±8.27 b	1.52±0.24 b
Control	43.26±5.88 c	0.95±0.12 c
LSD	50.20*	0.761*

*A significant difference between the groups is shown at p = 0.05.

Table 3: Pearson correlation between parameters for Diabetic.

Parameter		FBS mg/dl	BMI	Urea mg/dl	U.A mg/dl	Creatinin mg/dl	Chol. mg/dl	Tri. mg/dl	CALP ng/ml	XOD ng/ml
FBG mg/dl	r	1	0.166	-0.532	-0.130	0.014	0.030	-0.102	-0.049	0.093
	p	-	0.381	0.002	0.492	0.943	0.875	0.592	0.797	0.625
BMI	r	-	1	0.089	0.197	0.120	0.043	0.052	0.026	-0.092
	p	-	-	0.641	0.296	0.529	0.822	0.787	0.892	0.627
Urea mg/dl	r	-	-	1	-0.031	0.300	0.165	-0.100	-0.143	-0.136
	p	-	-	-	0.870	0.107	0.384	0.599	0.452	0.474
U.A mg/dl	r	-	-	-	1	0.007	-0.140	-0.174	0.146	-0.218
	p	-	-	-	-	0.972	0.461	0.359	0.441	0.248
Creatinin mg/dl	r	-	-	-	-	1	-0.184	-0.237	-0.084	0.060
	p	-	-	-	-	-	0.330	0.208	0.659	0.752
Chol. mg/dl	r	-	-	-	-	-	1	0.350	-0.230	0.241
	p	-	-	-	-	-	-	0.058	0.222	0.200
Tri. mg/dl	r	-	-	-	-	-	-	1	-0.422*	0.311
	p	-	-	-	-	-	-	-	0.020	0.094
CALP ng/ml	r	-	-	-	-	-	-	-	1	-0.265
	p	-	-	-	-	-	-	-	-	0.157
XOD ng/ml	r	-	-	-	-	-	-	-	-	1
	p	-	-	-	-	-	-	-	-	-

** Correlation has significance at the 0.01 level (two-tailed).
* Correlation is significant at the p = 0.05 level (2-tailed).

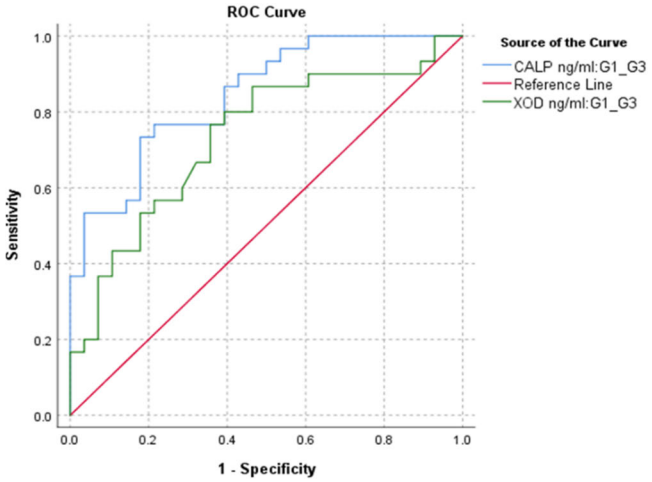

Figure 1: ROC curve for CALP

levels of CALP at the DN group had the highest mean value (173.46±29.56 ng/ml), then the diabetic group (78.2±8.27 ng/ml) and the control group (43.26±5.88 ng/ml), the statistically significant differences were indicated by letters (a, b, c) given to each group. The DN group (2.04±0.38 ng/ml) and the diabetic group (1.52±0.24 ng/ml) did not substantially vary in XOD levels. Both groups had considerably greater levels than the control group (0.95±0.12 ng/ml). LSD values of 50.20 for CALP and 0.761 for XOD, which quantify group comparison dependability, corroborated these differences' statistical significance.

Table 3 Pearson correlation analysis demonstrated a significant moderate positive correlation between fasting blood glucose and serum urea (r = 0.532, p = 0.002). In addition, a significant moderate negative correlation was observed between triglycerides and calprotectin levels (r = -0.422, p = 0.020). No statistically significant correlations were found between XOD and any of the investigated biochemical parameters, nor between calprotectin and the remaining clinical variables (p>0.05).

Figure 1 determines the ROC curve study underscores the prospective use of CALP may serve as a potential biomarker, while the AUC values indicate superior diagnostic efficacy of CALP levels in differentiating between groups. The AUC for control and DN is 0.844 (p = 0.001) and for control versus diabetic, it is 0.853 (p = 0.001). Multiple comparisons provide statistically significant results (p<0.05), demonstrating the ability of CALP to differentiate between diabetes and the control group.

The ideal minimum CALP value distinguishes between the control and DN groups at 64.82 ng/ml, resulting in a sensitivity of 86.3% and a specificity of 82.1%. The threshold for differentiating control from diabetes is 55.37 ng/ml, exhibiting a sensitivity of 84.5% and a specificity of 83.4%. Conversely, the elevated sensitivity and specificity values underscore the dependability of CALP as a diagnostic indicator for identifying diabetes-related complications.

In addition, Figure 2 shows the AUC curve indications for XOD (ng/ml) diagnostic performance. And the AUC = 0.833 (p = 0.002) for control vs. DN, indicating considerable discrimination. The results show AUC for control and diabetes is 0.863 (p = 0.031), indicating statistical significance (p<0.05). These data demonstrate level of XOD

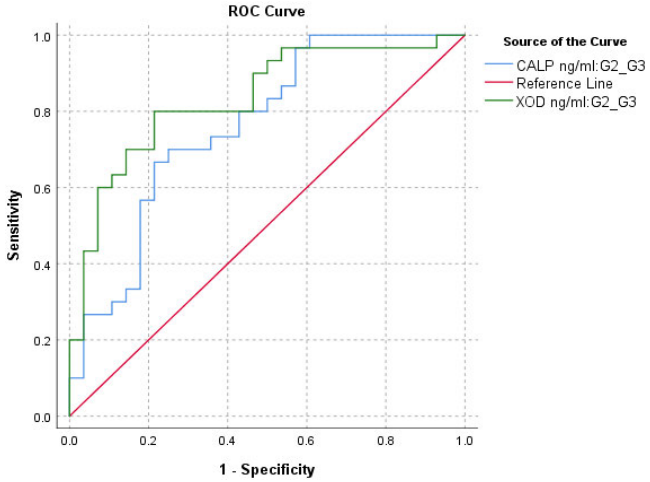

Figure 2: ROC curve for XOD

capacity to differentiate diabetic from non-diabetic circumstances. The optimum cut-off value for separating control from DN is 1.15 ng/ml, with 76.5% sensitivity and 89.9% specificity. A somewhat higher cut-off value of 1.25 ng/ml for control and diabetes has 89.0% sensitivity and 83.7% accuracy.

DISCUSSION

Diabetes-associated kidney disease is a renal condition caused by diabetes and the kidney is the primary contributor to chronic renal disease globally, accounting for renal illness in approximately one-third of individuals receiving dialysis [1].

In that research, the results demonstrated notable differences ($p<0.05$) in the BMI parameters between the patient group and healthy subjects, as shown in Table 1. This finding is consistent with other research [16] and the investigation revealed a notable elevation of glucose levels in comparison to the control group. In addition to factors contributing to this condition, which typically manifests after age 40, some factors involve a decline in β-cell functionality, variations in insulin production and its effectiveness and a rise in insulin resistance [17,18].

The analysis of this research indicated a statistically significant rise in total cholesterol levels among individuals with diabetes compared to the control group. Still, observation linked to low physical activity or a suppression of cholesterol breakdown [19]. Furthermore, the levels of triglycerides were observed to be markedly elevated in individuals with diabetes in comparison to the control group, which results in a lack of insulin, which leads to elevated blood sugar levels and the release of fatty acids from fat stores [19]. The research suggests that significantly higher blood urea levels and creatinine in diabetic patients are associated with kidney damage more than in control patients, which agrees with Ullah *et al.* [20]. The abnormally high serum creatinine and urea levels indicate that hyperglycaemia is associated with kidney diseases [21].

A kidney problem is indicated by elevated urea and creatinine levels, which result from muscle creatine breakdown. The kidneys filter urea into urine, but the body reabsorbs and uses some.

Korsmo *et al.* [23] showed that uric acid levels varied across the patient cohort and control groups. Insoluble urate crystals may accumulate in the renal tubules and medullary interstitial region due to elevated XOR activity [24]. This disease may cause urate stones and renal tubule destruction, leading to kidney failure [25, 26].

Hasan *et al.* [27] reported that the XOD values in the kidney disease group were considerably higher than those in the diabetic and health groups (2.04±0.38, 1.52±0.24 and 0.95±0.12). High serum glucose levels in diabetes enhance XOD activity in renal glomerular endothelial cells, causing ROS production (H_2O_2 and O_2^-) and oxidative stress, leading to renal endothelium damage [3]. Clinically, elevated XOD activity may reflect increased oxidative stress in diabetic nephropathy. Since oxidative stress contributes to endothelial dysfunction, inflammation and progressive renal injury, XOD may serve as a potential biomarker for identifying patients at higher risk of kidney damage. Furthermore, these findings support the potential therapeutic relevance of targeting XOD using xanthine oxidase inhibitors to slow disease progression.

The patients' serum CALP concentrations increased significantly ($p<0.05$): 173.46±29.56, 78.2±8.27 and 43.26±5.88 compared to healthy persons. Multiple studies [15] support this result. Calprotectin, an S100 protein, is essential for proinflammatory signalling. From a clinical perspective, increased calprotectin concentrations indicate ongoing inflammatory activity. As inflammation is a key mechanism in the development and progression of diabetic kidney disease, calprotectin may help identify patients with active renal inflammation before advanced renal dysfunction becomes clinically evident. CALP activates critical signalling pathways, including TLR4 and AGE receptors, to cause micro- and macrovascular problems associated with DM [9]. Based to those processes, it may because nephropathy associated with diabetes [10].

The elevation of serum urea and creatinine confirms declining renal function in patients with diabetic nephropathy. Clinically, these conventional renal markers remain essential for disease staging; however, they often increase after substantial nephron loss. Therefore, combining traditional renal markers with inflammatory and oxidative stress biomarkers such as calprotectin and XOD may improve early risk stratification [3].

Table 3 although a significant inverse correlation between triglycerides and calprotectin was observed, this finding should be interpreted cautiously because of the relatively small sample size and the cross-sectional design of the study, which precludes establishing causality. Clinically, the positive association between FBG and urea suggests that poor glycaemic control may be accompanied by worsening renal function. This finding reinforces the importance of strict glycaemic control to delay renal impairment.

Figure 1 shows that CALP may be a sensitive and specific biomarker for early identification and diagnosis of diabetic sequelae, notably DN [28]. It's excellent AUC = 0.844 values and strong diagnostic characteristics indicate clinical usage for screening high-risk people and following the growth of the disease, as shown in Figure 1. The ability to discriminate diabetic and kidney patients from healthy controls makes it useful in diabetes diagnosis and encourages future study to prove its clinical uses [29].

Figure 2 shows that XOD can identify oxidative stress-related changes in diabetes and DN. High specificity and sensitivity make it useful in medical diagnostics and its AUC value of 0.833 allows early metabolic abnormality detection and tailored treatment interventions. Table 3 XOD levels separate diabetes and renal patients from healthy controls. Its high AUC, sensitivity and specificity make it a diabetes biomarker. These results suggest assessing XOD's potential in oxidative stress measurement and clinical evaluation [22]. In Table 3, urea, the nitrogenous byproduct of amino acid metabolism, is positively correlated with fasting blood glucose. Urea is the main metabolite of tissue and dietary protein. Consequently, diabetes-related high urea levels imply a damaged kidney [30].

CONCLUSIONS

This research found that diabetes damage associated with the kidney and diabetes dramatically increases CALP, suggesting its utility as an inflammatory marker. At the same time, XOD was significantly associated with diabetic nephropathy. The results obtained confirm the diagnostic and prognostic utility of these biomarkers in assessing metabolic and inflammatory changes associated with diabetes and its consequences. The rock curve ROC analysis demonstrated that xanthine oxidase and calprotectin may have potential clinical value as a biomarker; however, prospective longitudinal studies are required to determine its prognostic utility.

Limitations

This study was limited by its relatively small sample size, recruitment from a single diabetes centre and cross-sectional design, which may limit the generalizability of the findings and prevent assessment of temporal changes in biomarker levels. The cross-sectional design of this study precludes establishing causal relationships or determining the prognostic value of the investigated biomarkers. Future longitudinal studies with larger sample sizes are warranted to evaluate whether XOD and calprotectin can predict the progression of diabetic kidney disease.

Ethical Statement

The study was approved by the Ethics Committee of [Al-Nahrain University] (Approval No. 2\21-RECSNU, Date: 09/12/2025). All procedures were conducted in accordance with ethical standards.

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