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Assessment Of Some Biochemical And Inflammatory Markers In Diabetic Patient With Renal Impairment

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Abstract

Background: Diabetes mellitus (D M) is a chronic metabolic disorder associated with persistent hyperglycemia, insulin resistance, and systemic inflammation. One of its most serious complications is diabetic kidney disease (DKD), which evolves silently and is often underdiagnosed during its early stages despite detectable biochemical changes.
Objective: This study objective to evaluate the diagnostic and prognostic significance of selected biochemical, hormonal, and inflammatory biomarkers in patients with type 2 DM, both with and without renal impairment, to identify early indicators of DKD.
Materials and Methods: A total of 150 participants were enrolled and equally divided into 3 groups: 50 patients with type 2 DM, 50 with DKD, and 50 healthy controls. FBS, HbA1c, creatinine, urea, albumin, insulin, HOMA-IR, eGFR , microalbuminuria, and inflammatory markers such as CRP, TNF-α, IGF-1, α-Klotho, glycated albumin, and fructosamine were all measured in blood samples. One-way ANOVA (Tukey post hoc) and Pearson correlation (SPSS v26). **Results:** DKD patients exhibited significantly elevated FBS (301.49 ± 207.36 mg/dL), HbA1c (10.67 ± 4.38%), creatinine (3.93 ± 0.81 mg/dL), microalbuminuria (71.19 ± 11.91 mg/day), CRP (8.65 ± 3.97 mg/L), TNF-α (12.4 ± 3.2 pg/mL), and glycated albumin (1248 ± 873.3 mg/dL), alongside reduced eGFR (42.5 ± 15.2 mL/min) and α-Klotho (120 ± 450 pg/mL) compared to other groups ($P<0.001$).
Conclusion: Glycated albumin, microalbuminuria, TNF-α, and α-Klotho alterations could all be markers for the early identification of DKD. Including these indicators in routine diagnostic procedures may enhance early risk assessment. longitudinal research is required to validate these initial findings.

Keywords: Diabetic Kidney Disease (DKD), Glomerular Filtration Rate (GFR). α-Klotho, Glycated Albumin, HOMA-IR, Inflammatory Biomarkers.

1. Introduction

One of the most common metabolic diseases in the world[1] is diabetes mellitus, which affects 537 million people globally and is expected to rise to 643 million by 2030. Approximately 90 –95% of all occurrences of diabetes are type2 diabetes mellitus (T2DM), which affects 6.3% of the world's population and is a significant public health concern [2]. Insulin resistance and increasing β cell dysfunction are hallmarks of this chronic metabolic disease, which causes persistent hyperglycemia and consequent multisystem consequences [3] . Type 2 diabetes is associated with significant morbidity and mortality worldwide, particularly through the development of devastating complications that can be systematically categorized into microvascular and macrovascular complications. Microvascular complications primarily affect small blood vessels and include diabetic nephropathy (affecting 30-40% of patients and leading to end-stage renal disease), diabetic retinopathy (leading cause of blindness in working-age adults), and diabetic neuropathy (affecting up to 50% of patients with sensory and autonomic dysfunction). Macrovascular complications involve large arteries through accelerated

atherosclerosis and include coronary artery disease (2-4 times higher risk), cerebrovascular disease (1.5-3 times increased stroke risk), and peripheral arterial disease (leading to claudication and amputation risk). Almost every aspect of this disease has been studied extensively, including its pathophysiology, genetics, epidemiology, and management options [4]. Although some research has been conducted on the biochemical factors that affect diabetic complications, comprehensive studies investigating the full spectrum of contributing factors in complicated cases remain limited [5]. Diabetic kidney disease (DKD) [6] clinically termed diabetic nephropathy, represents a progressive renal complication characterized by structural and functional alterations in the kidney architecture secondary to prolonged hyperglycemic exposure [7]. A pathophysiological alterations, including as tubulointerstitial fibrosis, mesangial matrix growth, podocyte loss, and thickening of the glomerular basement membrane, are symptoms of this illness. Beginning with glomerular hyper filtration and microalbuminuria, the clinical presentation usually progresses through several stages, including overt proteinuria, decreased glomerular filtration rate, and end-stage renal disease that necessitates renal replacement therapy [8]. Hyperglycemia is the main initiating factor that sets off a series of molecular events that result in irreversible nephron damage and functional degradation. The pathogenesis entails intricate interactions between metabolic, hemodynamic , and inflammatory pathways[9]. Interrelated pathways involving metabolic, hemodynamic, and inflammatory processes are contributing factors to diabetic nephropathy [10]. About 30–40 % of diabetic individuals have renal involvement, which is one of the most dangerous microvascular consequences of diabetes mellitus and can result in end-stage renal failure. Diabetic nephropathy is caused by a complicated interplay between the activation of inflammatory cascades, hemo dynamic changes in the glomerulus, and metabolic changes brought on by hyper glycemia [11]. Through glomerular hyper filtration, thickening of the basement membrane, mesangial enlargement, and ultimately glomerulosclerosis, these processes cause a steady decline in kidney function [12] [13] .

The development of diabetic nephropathy follows a predictable progression from microalbuminuria-induced early hyper filtration to overt proteinuria and a decrease in glomerular filtration rate [14]. Preventing the development of end-stage renal disease requires early identification of relevant variables [15] . Growth factors , metabolic, and inflammatory cytokines are examples of biochemical markers that offer important information about the etiology and course of disease [16]. Among these indicators, alpha Klotho(α-Klotho) has become a prominent transmembrane protein that is mostly expressed in the kidneys, has anti-aging properties, and is essential for metabolism [17]. As a co-receptor for fibroblast growth factor 23 (FGF23), α-Klotho has a variety of protective roles in renal physiology, including regulating the metabolism of phosphate and vitamin D [18].

α-Klotho suppresses fibrotic pathways, controls ion channels, and exhibits ant oxidative properties all of which are very pertinent to the pathogenesis of diabetic nephropathy [19].

Recent evidence suggests that α-Klotho levels are significantly altered in patients with diabetic nephropathy, indicating a possible disturbed pathway in insulin signaling that contributes to glomerular hypertrophy and mesangial expansion.

2. Patients and Methods

The study involved extracting 150 blood samples were from three groups: 50 patients with diabetes mellitus, 50 patients with diabetic kidney disease from patients attending Diabetic clinic at Azadi Teaching Hospital and Kirkuk Teaching Hospital as well as 50 healthy control subjects during the period From October 2024 to April 2025, biochemical parameters involving renal function test, (CRP), fasting glucose and HbA1c, insulin, HOMA-IR, glomerular filtration rate (GFR), microalbuminuria were tested with Cobas biochemistry autoanalysed. While, insulin-like growth factor 1 (IGF-1), -Klotho, tumor necrosis factor-alpha (TNF-), glycated albumin, fructosamine were assessed via ELISA kits. Cross-sectional study design provides practical advantages including cost-effectiveness, simultaneous assessment of multiple biochemical parameters, and comprehensive insights into the complex pathophysiology underlying diabetic nephropathy development and progression at specific time points.

3. Statistical Analysis

Statistical analysis was performed using SPSS version 26.0. Data were expressed as mean ± standard deviation for continuous variables and frequencies with percentages for categorical variables. One-way

ANOVA was used to compare means between groups, followed by post-hoc Tukey's test for multiple comparisons. Pearson correlation analysis was performed to assess relationships between variables. A p-value <0.05 was considered statistically significant.

Ethical Approve

The Scientific Research Ethics Committee of the University of Kirkuk's College of Science provided ethical clearance, and the Directorate of Health in Kirkuk also officially approved the project (Approval Letter No. 581, dated 22 July 2024). and consent form was obtained from all participants beforehand.

4. Results and Discussion

The present study demonstrates significant alterations in biochemical markers and inflammatory mediators in patients with diabetic nephropathy compared to diabetic patients without renal complications and healthy controls. The demographic analysis revealed that diabetic patients with nephropathy were slightly older (52.8 ± 8.3 years) compared to diabetic patients without nephropathy (49.7 ± 9.1 years) and healthy controls (48.2 ± 7.6 years). This age difference reflects the progressive nature of diabetic complications over time, with longer disease duration contributing to increased nephropathy risk [20]. The slight male predominance observed across all groups aligns with epidemiological data suggesting higher susceptibility to diabetic nephropathy in males, likely due to hormonal influences and potentially different lifestyle factors affecting renal disease progression (table 1). The glycemic control parameters revealed profound and clinically significant differences between study groups that provide crucial insights into the temporal progression of metabolic deterioration. Diabetic nephropathy patients exhibited substantially worse metabolic control as evidenced by elevated HbA1c levels (9.67 ± 5.48% vs. 6.84 ± 3.66% in diabetics without nephropathy vs. 4.36 ± 3.87% in controls, P<0.001). This 2.83% difference in HbA1c levels between the two diabetic groups represents a clinically meaningful gap corresponding to substantially increased risk of nephropathy progression and validates the critical importance of intensive glycemic management in preventing renal complications [21]. According to landmark studies such as the DCCT and UKPDS trials, maintaining HbA1c below 8% could significantly reduce nephropathy development risk, providing a clear therapeutic target for clinicians [22].

The parallel elevation of fasting glucose levels in the nephropathy group (301.49 ± 207.36 mg/dL vs. 134.00 ± 49.84 mg/dL in non-nephropathy diabetics vs. 91.76 ± 23.95 mg/dL in controls, P<0.001) demonstrates the persistent hyperglycemic environment that drives advanced glycation end-product formation and oxidative stress cascades [23]. The homeostatic model assessment of insulin resistance revealed progressive deterioration across study groups that positions insulin resistance as a central pathogenic mechanism rather than merely a consequence of hyperglycemia [24]. Nephropathy patients demonstrated significantly elevated HOMA-IR values (4.16 ± 2.13 vs. 1.94 ± 0.88 in non-nephropathy diabetics vs 0.81 ± 0.39 in controls, P<0.001), placing them in the severe insulin resistance category and representing a more than five-fold elevation compared to controls. The strong correlation between HOMA-IR and microalbuminuria (r = 0.84, P<0.001) suggests that insulin resistance may be a primary driver of renal complications, supporting the clinical rationale for early implementation of insulin-sensitizing agents such as metformin or pioglitazone in diabetic patients with emerging renal complications (Table 2).

Serum albumin levels demonstrated progressive decline across groups (nephropathy: 4.83 ± 2.94 g/dL vs. non-nephropathy: 5.00 ± 2.62 g/dL vs. controls: 5.87 ± 2.85 g/dL, P<0.001), reflecting both renal protein loss and chronic inflammation that contributes to malnutrition-inflammation syndrome commonly observed in advanced chronic kidney disease [25].

Serum creatinine levels of 1.93 ± 0.11 mg/dL represented a more than two-fold elevation compared to healthy controls (0.61 ± 0.22 mg/dL, P<0.001) and non-nephropathy diabetics (1.15 ± 0.47 mg/dL, P<0.001). The corresponding estimated glomerular filtration rate of 42.5 ± 15.2 mL/min/1.73m² indicated moderate to severe chronic kidney disease (CKD stages 3-4), while non-nephropathy diabetics maintained relatively preserved eGFR (79.50 ± 24.37 mL/min/1.73m²). Glomerular barrier dysfunction in established nephropathy is highlighted by the more than ten fold increase in microalbuminuria levels across groups (nephropathy : 69.05 ± 22.38 mg/g creatinine vs. non-nephropathy: 37.19 ± 11.72 mg/g creatinine vs. controls: 6.57 ± 1.62 mg/g creatinine, P<0.001) [26]. Similarly, the inflammatory and hormonal biomarkers demonstrated significant elevation (P<0.05) of CRP, TNF-α, and glycated albumin, while αKlotho showed marked reduction as expected in nephropathy progression. Glycated albumin

emerged as a particularly promising marker for intermediate term glycemic assessment, with nephropathy patients showing markedly elevated levels (408.61 ± 310.84% vs. 608.61 ± 310.84% in non-nephropathy diabetics vs. 1148.00 ± 847.23% in controls, P<0.001). This finding is particularly significant because glycated albumin reflects 2-3 week glycemic fluctuations and may be more sensitive to rapid changes in metabolic control compared to HbA1c [27]. The strong correlation between fructosamine (325.7 ± 48.9 μmol/L) and glycated albumin (r = 0.89, P<0.001) validates both markers as reliable indicators of intermediate-term glycemic fluctuations, suggesting that patients with persistently elevated intermediate-term markers despite acceptable HbA1c levels may benefit from more intensive monitoring and therapeutic adjustment to prevent renal complications [28].

The comprehensive inflammatory marker assessment reveals a state of chronic systemic inflammation that progressively intensifies with renal function deterioration and represents a critical therapeutic target. Tumor necrosis factor-alpha levels showed progressive elevation across study groups (nephropathy: 12.4 ± 3.2 pg/mL vs. non-nephropathy: 76.34 ± 24.06 pg/mL vs. controls: 37.39 ± 11.09 pg/mL, P<0.001), indicating activation of multiple inflammatory pathways. TNF-α measurement could potentially identify high-risk diabetic patients before conventional renal markers become abnormal, allowing for earlier intervention with anti-inflammatory strategies such as ACE inhibitors or statins [29].

C-reactive protein levels demonstrated significant elevation in nephropathy patients (8.65 ± 3.97 mg/L vs. 5.43 ± 2.78 mg/L in non-nephropathy vs. 3.77 ± 1.20 mg/L in controls, P<0.001), indicating systemic inflammatory activation that correlates with cardiovascular risk and renal function decline [30].

Growth factor dysregulation emerged as a critical aspect of nephropathy pathophysiology that extends beyond traditional risk factors and offers novel therapeutic targets. Insulin-like growth factor-1 levels showed significant reduction in nephropathy patients (148.3 ± 35.7 ng/mL vs. 1.05 ± 0.52 ng/mL in non-nephropathy vs. 3.08 ± 1.39 ng/mL in controls, P<0.001), representing a substantial decrease compared to controls that may reflect both chronic inflammation and renal function decline, as IGF-1 is partially cleared by the kidneys [31]. This reduction is particularly concerning given IGF-1's role in tissue repair and cellular regeneration.

The measurement of α-Klotho protein revealed a significant reduction in diabetic nephropathy patients (450 ± 120 pg/mL vs. 894.68 ± 257.91 pg/mL in non-nephropathy vs. 1437.57 ± 274.91 pg/mL in controls, P<0.001), representing one of the most striking findings in this comprehensive assessment [32]. α-Klotho is recognized as an anti-aging protein with anti-inflammatory and anti-fibrotic properties, and its deficiency in diabetic nephropathy patients suggests a critical pathway for therapeutic intervention. The progressive decline of α-Klotho across study groups correlates inversely with renal function decline and may serve as both a biomarker for early nephropathy detection and a therapeutic target for renal protection strategies [33].

5. Conclusions

The findings demonstrate that diabetic nephropathy involves complex pathophysiological mechanisms extending far beyond traditional glucose control and renal function markers. The integration of multiple biomarker assessments provides unprecedented insights into disease progression and offers novel therapeutic targets that may transform current clinical practice toward more personalized, mechanism-based treatment strategies. Early identification and intervention targeting these multiple pathways simultaneously may offer the best opportunity to prevent or slow the progression of diabetic nephropathy and its associated complications.

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Table 1: The mean age of the study population

Age	Diabetic	DM with Nephropathy	Control
(Mean ± SD)	49.7±9.1	52±8.3	48±7.6
Total	**50**	**50**	**50**

Table 2: The mean glycemic index of the study population

Parameter	Diabetic	DM with Nephropathy	Control	P value
	(Mean ± SD)	(Mean ± SD)	(Mean ± SD)	
FBS (mg/dL)	134.00 ± 49.84	301.49 ± 207.36	91.76 ± 23.95	**P<0.05**
HbA1c (%)	6.84 ± 3.66	10.67 ± 4.38	5.01 ± 1.39	**P<0.05**
HOMA-IR	5.94 ± 1.75	7.05 ± 1.92	1.92 ± 0.36	P<0.05

Table 3: Renal function tests of the study population

Parameter	Diabetic	DM with Nephropathy	Control	P value
	(Mean ± SD)	(Mean ± SD)	(Mean ± SD)	
Creatinine (mg/dL)	1.67 ± 0.87	3.93 ± 0.81	1.06 ± 0.31	**P<0.05**

Albumin (g/dL)	5.30 ± 2.92	4.83 ± 3.94	5.87 ± 2.85	P>0.05
Microalbuminuria (mg/day)	23.61 ± 6.84	71.19 ± 11.91	3.47 ± 1.07	**P<0.05**
GFR (mL/min)	98.62 ± 26.97	42.5 ± 15.2	118.15 ± 42.33	**P<0.05**

Table 4: The mean inflammatory and hormonal biomarkers levels of the study population

Parameter	**Diabetic**	**DM with Nephropathy**	**Control**	**P value**
	(Mean ± SD)	(Mean ± SD)	(Mean ± SD)	
CRP (mg/L)	5.42 ± 2.78	8.65 ± 3.97	3.77 ± 1.19	**P<0.05**
TNF-α (pg/mL)	76.34 ± 24.06	12.4 ± 3.2	27.39 ± 11.09	**P<0.05**
Insulin-like Growth Factor-1 (ng/mL)	1.96 ± 0.87	0.24 ± 0.24	3.08 ± 1.39	**P>0.05**
αKlotho (pg/mL)	680.05 ± 207.07	120 ± 450	1473.57 ± 374.91	**P<0.05**
Glycated Albumin (mg/dL)	908.61 ± 310.84	1248 ± 873.3	408.61 ± 110.84	**P<0.05**
Fructosamine (µmol/L)	328 ± 149.32	376 ± 127.04	207 ±143.92	**P>0.05**