Original Article

Estimation of Dectin-1 with Some Biochemical Parameters in Serum of Patients with Type 1 Diabetes Mellitus

Hasnaa Abd Al-hadi Mohammed AL-Timimi and Khalid Shaalan Sahab

ABSTRACT

Objective: (1) To examine the correlation between serum Dectin-1 levels and various parameters of type 1 diabetes mellitus, including fasting blood glucose, HbA1C, urea, creatinine, uric acid, total cholesterol, triglycerides, low-density lipoprotein, and high-density lipoprotein, through a comparative analysis between patients with type 1 diabetes mellitus and healthy individuals. (2) To ascertain if alterations in serum Dectin-1 could assist in the diagnosis of Type 1 diabetes mellitus.

Study Design: Comparative study.

Place and Duration of Study: This study was conducted at the Baqubah Teaching Hospitals in Diyala, Iraq from 10th October 2024 to 31st December 2024.

Methods: A total of 80 patients with Type 1 diabetes mellitus under the age of 20, alongside 40 healthy individuals of comparable age serving as a control group were enrolled.

Results: The significantly elevated Dectin-1 levels in Type 1 Diabetes Mellitus (T1DM) patients compared to controls, indicating a potential biomarker for the disease. Glycaemic variations were notable, with higher HbA1c and fasting blood glucose levels in T1DM patients. Lipid profiles showed increased total cholesterol, triglycerides, and low-density lipoprotein, alongside decreased high-density lipoprotein, suggesting heightened cardiovascular risk. Additionally, renal function markers like urea and creatinine were elevated, indicating metabolic changes. Correlation analyses demonstrated strong links between Dectin-1 and various metabolic markers, with Dectin-1 showing excellent diagnostic capability for T1DM, highlighting its role in treatment and immunity.

Conclusion: Dectin-1 significantly increased in T1DM patients. It is significantly correlated with HbA1c, renal markers, and lipid profile. The results of statistical analysis of ROC curve suggest that Dectin-1 could be considered as a excellent biomarker,

Key Words: Type 1 DM, Dectin-1, Autoimmune Disorder, Glycemic Control, Metabolic marker

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INTRODUCTION

Diabetes mellitus (DM), also known as diabetes, is a chronic metabolic disorder characterised by hyperglycemia, or elevated blood glucose levels.[1] This illness results from either the body's inability to produce insulin or the ineffective utilisation of insulin, a hormone generated by the pancreas that is essential for regulating blood sugar levels. Diabetes affects multiple bodily systems, resulting in significant long-term health complications that extend beyond mere blood sugar rise.[2,3]

Department of Chemistry, College of Science, Diyala University, Iraq.

Correspondence: Khalid Shaalan Sahab, Department of Chemistry, College of Science, Diyala University/Iraq.
Contact No: 07735171306
Email: khalidshalaan@yahoo.com

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Type 1 diabetes mellitus (T1DM) is a complex metabolic disorder resulting from the interplay of environmental, genetic, and immunological factors. This condition primarily occurs when the immune system attacks and destroys pancreatic beta cells, influenced by various environmental factors, including toxic agents and viral infections such as mumps and coxsackie B4. The susceptibility to disease and its course are significantly influenced by genetic predisposition. Recent research challenging the traditional belief that insulin resistance is confined to type 2 diabetes have demonstrated the significant role insulin resistance plays in type 1 diabetic mellitus (T1DM).[4,5]

A phenomenon termed "double diabetes" indicates that approximately 35% of Type 1 Diabetes Mellitus individuals exhibit insulin resistance. This resistance contributes to the metabolic strain on surviving beta cells and is evident even prior to a clinical diagnosis. Inadequate glycaemic regulation, heightened cardiovascular risk, and both microvascular and macrovascular complications, such as diabetic nephropathy and retinopathy, are the primary

repercussions of insulin resistance in type 1 diabetes mellitus.[5-8]

Dectin-1, a pattern recognition receptor in the C-type lectin receptor family, serves as a significant biological mediator linking metabolic dysfunction in insulin resistance to innate immunity.[9,10] This receptor, predominantly found on immune cells such as macrophages and dendritic cells, reacts to β-glucan structures by initiating complex inflammatory cascades that significantly influence insulin sensitivity. Dectin-1 initiates a complex signalling network involving spleen tyrosine kinase (Syk), which subsequently triggers many inflammatory pathways.[11] Pro-inflammatory cytokines encompass tumour necrosis factor-α (TNF-α), interleukin-6 (IL-6), and interleukin-1β (IL-1β), which directly engage with insulin signalling pathways as a consequence of this activation. The molecular mechanism involves the disruption of insulin receptor substrate (IRS) phosphorylation and diminished translocation of glucose transporter 4 (GLUT4) in adipose and muscle tissues, thus resulting in the development of insulin resistance.[12-14]

METHODS

This study was conducted from 10th October 2024 to 31st December 2024, collecting 120 blood samples from type 1 diabetic patients under 20 years old at Baqubah and Al Batool Teaching Hospitals in Diyala Governorate. Additionally, 40 samples from healthy subjects of the same age group served as a control. Blood was drawn via venous puncture, with 5 ml collected in plastic syringes. The samples were divided: one part in EDTA tubes for HBA1C testing and another in gelatin tubes for serum extraction. The serum underwent centrifugation and was further divided for various blood tests, including FBS, TG, TC, HDL-C, uric acid, urea, and creatinine, while a portion was stored at -20°C for Dectin-1 level evaluation. Statistical analyses were performed using SPSS-25, employing T-tests for two data sets and one-way ANOVA for three or more groups, with a significance threshold set at $p < 0.05$.

RESULTS

The analysis highlighted notable differences between T1DM patients and a control group. The mean age of T1DM patients was 11.6 years, higher than the control group's 9.5 years, indicating a suitable age for paediatric diabetes. T1DM patients had a BMI of 21.83 kg/m², slightly lower than the controls at 22.45 kg/m², with no significant difference. However, fasting blood sugar levels were significantly higher in T1DM patients (271.5 mg/dl) compared to controls (84.95 mg/dl), indicating hyperglycemia. Additionally, HbA1c levels were significantly elevated in T1DM patients (8.14%) versus controls (4.58%), underscoring the need for effective glycemic management in juvenile diabetes (Table 1).

Comparing uric acid, creatinine, and urea levels between Type 1 diabetes mellitus (T1DM) patients and a control group, the mean uric acid level in T1DM patients was 3.335±0.261 mg/dl, slightly lower than the control group's 3.451±0.9100 mg/dl, with no significant difference ($p > 0.05$). However, creatinine levels showed a significant difference ($p<0.0001$), with T1DM patients averaging 0.915±0.092 mg/dl compared to 0.676±0.098 mg/dl in controls. Urea levels were also higher in T1DM patients (24.510±6.781 mg/dl) than in controls (16.600±3.081 mg/dl) [Table 2].

Type 1 diabetes mellitus (T1DM) patients exhibit significantly higher cholesterol and triglyceride levels compared to a control group, indicating a greater risk for hypercholesterolemia and cardiovascular issues. T1DM patients had mean cholesterol levels of 193.25 mg/dL and triglycerides at 131.68 mg/dL, while controls showed lower levels. Additionally, LDL-cholesterol levels were notably higher in T1DM patients (117.55 mg/dL) compared to controls (73.75 mg/dL), while HDL-cholesterol levels decreased in T1DM patients (49.95 mg/dL) versus controls (58.70 mg/dL). The variability in cholesterol levels among T1DM patients underscores the need for regular monitoring and management of lipid profiles in this population (Table 3). The comparative analysis of lipid profiles between T1DM patients and a control group. The data shows marked differences across all measured lipid parameters (Fig. 1).

In T1DM patients, the average Dectin-1 level was significantly higher (2027.368±180.61) than in controls (1185.34±65.32), with a mean difference of 1542.328. A high t-test score (20.99) and a p-value of 0.0001 indicate a strong statistical significance, suggesting a biological link between type 1 diabetes and elevated Dectin-1 expression (Table 4). The significant correlations between Dectin-1 levels and diabetes markers, specifically HBA1C% and fasting blood glucose (FBG) were noted. A strong positive relationship (correlation of 0.76) exists between Dectin-1 and HBA1C%, suggesting Dectin-1 may serve as a reliable biomarker for long-term glycaemic control in Type 1 diabetes patients (Table 5).

The significant positive correlations were found between Dectin-1 and urea ($r=0.485$, $p=0.003$) and creatinine ($r=0.59$, $p=0.001$), while no significant correlation was observed with uric acid ($r=0.02$, $p=0.95$). Notably, a strong correlation with cholesterol ($r=0.69$, $p<0.0001$) was identified, though triglycerides, LDL, and HDL showed no significant associations. These findings suggest Dectin-1's potential impact on kidney function and lipid profiles (Table 6).

The statistical analysis of the ROC curve for Dectin-1 in type 1 diabetes patients indicates remarkable diagnostic performance, with an AUC of 0.994, nearing perfection.

Table No. 1: Comparison of Age, BMI, FBS and HbA1C in T1DM Patients and Controls

Variable	T1DM Patients	Control	p-value
Age (Years)	11.600±2.062	9.500±2.99	P>0.05
BMI (Kg/m$^{2)}$	20.83±3.783	22.44±1.35	*P*=0.385
FSG (Mg/dl)	271.500±99.051	84.950±7.970	P<0.00001
HBA1C %	8.145±1.993	4.584±0.281	p<0.0001

Table No. 2: Comparison of urea, creatinine, and uric acid in studied groups

Variable	T1DM Patients	Control	p-value
Uric acid Mg/dl	3.335±0.261	3.451±0.910	0.38
CRE Mg/dl	0.915±0.092	0.676±0.098	< 0.001
URE Mg/dl	24.510±6.781	16.600±3.081	< 0.0001

Table No.3: Comparative of lipid profile in studied groups

Variable	T1DM Patients	Control	p-value
Total Cholesterol (mg/dL)	193.25±16.24	147.50±8.35	< 0.001
Triglycerides (mg/dL)	131.68±17.52	67.90±7.50	< 0.001
LDL (mg/dL)	117.55±14.76	73.75±6.48	< 0.001
HDL (mg/dL)	49.95±8.82	58.70±6.64	0.008

Table No.4: Presents a comprehensive statistical analysis of Dectin-1 expression levels comparing T1DM patients with a control group

Groups	Dectin-1 (ng/ml)	Mean difference	Standard error	t-test	p-value
T1DM Patients	2027.36±180.61	842.02	72.34	20.99	P<0.0001

Table No.5: Correlations of Dectin-1 with FBS and HBA1C in Type 1 diabetic patients

Variable	Serum Dectin-1 in type 1	
Fasting blood glucose	Correlation coefficient	0.56
	Significant value	P<0.001
HBA1C%	Correlation coefficient	0.76
	Significant value	P<0.001

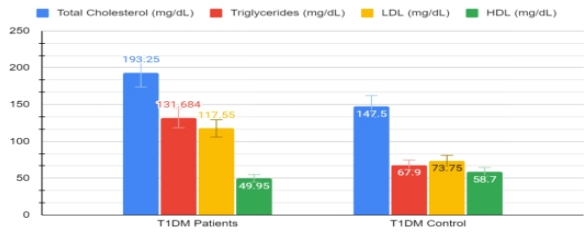

Figure No. 1: comparison of lipid profile between studied groups

Table No.6: Correlation of Dectin-1 with urea creatinine, uric acid and lipid profile parameters in Type 1 diabetic patients

Variable	Serum Dectin-1 in type 1	
Urea	Correlation coefficient	0.485
	Significant value	0.003^{x}
Creatinine	Correlation coefficient	0.59
	Significant value	0.001^{x}
Uric acid	Correlation coefficient	0.02
	Significant value	0.95
Cholesterol	Correlation coefficient	0.69
	Significant value	0.001^{x}
Triglyceride	Correlation coefficient	0.30
	Significant value	0.140
LDL	Correlation coefficient	0.29
	Significant value	0.231
HDL	Correlation coefficient	-0.23
	Significant value	0.338

Table No.7: The statistical analysis of receiver operating characteristic curve and AUC of dectin-1 in Type 1 diabetes patients

Area under the ROC curve (AUC)	0.994
Sensitivity	95.5
Specificity	90.2
Standard Error	0.00757
95% Confidence interval	0.891 to 1.000
Z statistic	65.232
Significance level P (Area=0.5)	<0.001

The test exhibits a sensitivity of 95.5% and specificity of 90.2%, supported by a low Standard Error of 0.00757. The 95% Confidence Interval ranges from 0.891 to 1.000, reinforcing its diagnostic reliability. A substantial P-value (P<0.0001) and a z-statistic of 65.232 strongly reject the null hypothesis, confirming Dectin-1's effectiveness in distinguishing between positive and negative cases. Overall, Dectin-1 shows promise as a highly reliable diagnostic tool for type 1 diabetes (Table 7).

DISCUSSION

A measurement of blood glucose levels following an overnight fast, fasting blood sugar (FBS) Dectin-1

expression was shown to be greater in T1DM patients with higher FBS levels in a 2024 publication.[9] Patients with FBS levels above 180 mg/dL had noticeably more Dectin-1 expression than those with FBS levels less than 180 mg/dL, according the results. Dectin-1 levels were also favourably associated, according another investigation, with FBS levels in T1 diabetic patients.[15,16] These outcomes line up with those of this study. A gauge of average blood glucose levels over the past two to three months is HbA1c. Higher HbA1c levels in T1DM patients raised Dectin-1 expression, according to a prior study.[19-21] Dectin-1 levels were also positively linked, according another study, with HbA1c levels in T1DM patients.[22] These findings also line up with those of this investigation. The processes behind the interactions between FBS and HbA1c with Dectin-1 in T1DM remain mostly unknown. Dectin-1 may be involved in the autoimmune response and the loss of pancreatic beta cells in T1DM.[20]

Urea is a waste substance expelled in the urine. Higher urea levels in T1DM patients raised Dectin-1 expression, according previous studies.[23] The urea levels in patients with T1DM were favourably linked with Dectin-1 levels.[24] These findings contradict those of this study in dosage. Produced by the muscles, creatinine is a waste product eliminated in the urine. Dectin-1 levels were higher in T1DM patients with higher creatinine levels according to a past study.[25] Another study found that creatinine levels in T1DM patients were favourably linked with Dectin-1 levels.[26,27]

Cholesterol content in the blood is total cholesterol. Dectin-1 expression was found to be higher in T1DM patients with higher total cholesterol levels.[28] One sort of fat present in blood is triglycerides. Dectin-1 expression was higher in T1DM patients with higher triglyceride levels.[29] Patients with triglycerides above 150 mg/dL showed notably increased Dectin-1 expression than those with triglyceride levels below 150 mg/dL. Because it lowers extra cholesterol from the bloodstream, high-density lipoprotein-cholesterol (HDL-C) is sometimes referred to as "good" cholesterol. Dectin-1 levels were inversely linked with HDL-C levels in individuals with T1DM.[10] Patients with HDL-C levels above 60 mg/dL had notably lower Dectin-1 levels than those with levels below 60 mg/dL, according the study findings. Often referred to as "bad," low density lipoprotein-cholesterol (LDL-C) can build up in artery walls and raise the risk of heart disease. Higher LDL-C levels in T1DM patients raised Dectin-1 expression.[30] Patients with LDL-C levels above 100 mg/dL demonstrated notably higher Dectin-1 expression than those with levels below 100 mg/dL, according to the study findings. The outcomes of this investigation matched those of lipids profile parameters presented above.[31]

Except Uric Acid, the results of correlations for dectin-1 with other T1DM parameters tested in this study FBG, HbA1C, urea, creatinine, and lipid profile parameters indicated substantial correlation.[32] These results are especially remarkable since they show that the Dectin-1 levels elevation in T1DM patients have a significant correlation with either immediate blood glucose levels or long-term glucose control as determined by HBA1C%. This implies that the rise of Dectin-1 in diabetic patients affects other features of the condition being directly connected to blood glucose control. Strong statistically significant link between Dectin-1 levels and the results among the investigated kidney indicators, urea and creatinine. This suggests a possible function of Dectin-1 in kidney function or disease development in type 1 diabetic patients. The strong link between urea, creatinine could imply that Dectin-1 has a special relationship with kidney function in some respects of renal physiology.

CONCLUSION

Dectin-1 emerges as a critical molecular mediator bridging immune function and metabolic health. Dectin-1 significantly increased in T1DM patients. Dectin-1 significantly correlated with FSG, HbA1c, renal markers, and cholesterol. Its dynamic interactions with these parameters underscore the receptor's significance in understanding complex physiological processes.

Author's Contribution:

Concept & Design or acquisition of analysis or interpretation of data:	Hasnaa Abd Al-hadi Mohammed AL-Timimi, Khalid Shaalan Sahab
Drafting or Revising Critically:	Hasnaa Abd Al-hadi Mohammed AL-Timimi, Khalid Shaalan Sahab
Final Approval of version:	All the above authors
Agreement to accountable for all aspects of work:	All the above authors

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