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The relationship between serum dectin-1 and other biochemical markers in Iraqi patients with type2 diabetes patients

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ABSTRACT

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by hyperglycemia resulting from insulin resistance and/or impaired insulin secretion. Dectin-1, a pattern recognition receptor of the C-type lectin family, has recently emerged as a potential link between innate immunity and metabolic dysfunction. This study aimed to evaluate serum Dectin-1 levels in T2DM patients and investigate their association with glycemic and metabolic parameters.

The study was conducted between October and December 2024 at Baqubah Teaching Hospitals, Diyala, Iraq. Eighty patients with T2DM aged over 35 years and 40 age-matched healthy controls were enrolled. Serum Dectin-1 levels, glycemic indices, renal function markers, and lipid profiles were assessed and compared between groups.

Dectin-1 levels were significantly higher in T2DM patients than in controls (3008.889 vs. 1966.500; $p<0.001$). Patients with T2DM also had a significantly greater body mass index (28.947 vs. 21.675 kg/m²; $p<0.05$). Marked alterations in glycemic control were observed, with higher fasting blood glucose (179.975 vs. 98.450 mg/dL) and HbA1c levels (8.052% vs. 5.059%) in diabetic patients ($p<0.001$). Creatinine levels were significantly elevated in the T2DM group ($p<0.001$), whereas urea and uric acid showed no significant differences. Lipid profile analysis revealed dyslipidemia characterized by increased total cholesterol, triglycerides, and LDL levels, alongside reduced HDL concentrations. Correlation analysis demonstrated generally weak associations between Dectin-1 and metabolic parameters; however, a significant positive correlation was identified between Dectin-1 and creatinine ($r=0.30$, $p=0.009$). Receiver operating characteristic (ROC) analysis showed moderate diagnostic performance of Dectin-1 for T2DM, with an area under the curve (AUC) of 0.722.

In conclusion, serum Dectin-1 levels were significantly elevated in Iraqi patients with T2DM and may reflect immune and inflammatory processes associated with the disease. These findings suggest that Dectin-1 could serve as a potential biochemical biomarker for T2DM.

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1. INTRODUCTION

Hyperglycemia and glucose intolerance are common symptoms linked to diabetes mellitus [1]. The small intestine breaks down proteins, lipids, and carbohydrates, enabling their absorption into the bloodstream when food passes through the digestive system. The endocrine pancreas releases insulin in reaction to glucose concentrations. Almost all tissue types in the body, such as hepatic, muscular, and adipose tissues, require insulin for glucose absorption for use or storage [2, 3] .

The lack of insulin synthesis or its malfunction results in elevated blood glucose levels, characteristic of Type

2 Diabetes Mellitus (T2DM). Type 2 diabetes, the most prevalent variety of the disease, is differentiated from other variants. A distinctive characteristic of type 2 diabetes mellitus (T2DM) or non-insulin-dependent diabetes mellitus (NIDDM) is that individuals typically do not have ketones in their urine while having the condition, and the majority are unlikely to experience ketosis. Insulin therapy may be initiated in individuals when dietary adjustments and oral medicines do not effectively manage fasting hyperglycemia. These patients may undergo ketosis due to substantial stress, such as injury or disease [4, 5].

The complicated mechanism where the pancreas persists in insulin secretion yet is insufficiently used by the body is central to the pathogenesis of type 2 diabetes. The main reason for this condition is peripheral insulin resistance, which means that the insulin receptors in body tissues do not respond well, affecting how cells use insulin[6, 7]. Hyperglycemia, characterized by elevated blood glucose levels, occurs when cells are unable to uptake glucose due to insensitivity. Obesity is a significant health issue and a primary contributor to the development of Type 2 diabetes. Obesity exacerbates insulin resistance and complicates disease management, and it is prevalent among the majority of individuals with type 2 diabetes [8].

Dectin-1, a member of the C-type lectin receptors, is a vital molecular mediator linking innate immunity and metabolic dysfunction in insulin resistance. Most immune cells, like dendritic cells and macrophages, usually have this receptor, and when it binds to certain structures, it triggers complicated inflammatory processes that significantly lower insulin sensitivity. Recent studies have linked Type 2 diabetes to Dectin-1[9]. Dectin-1 is associated with the persistent inflammation of type 2 diabetes, according to research findings. Chronic inflammation exacerbates diabetes by inducing tissue damage and heightening insulin resistance. Furthermore, dectin-1 is overexpressed in individuals with type 2 diabetes, thereby further implicating it in the pathogenesis of the disease [10].

The role of dectin-1 in type 2 diabetes has been explained by a number of theories. Dectin-1's first function is to recognize and attach to beta-glucans, which are polysaccharides found in the cell walls of fungi. The immune system begins to react as a result of the binding, producing cytokines that lead to inflammation and chronic inflammation. Second, dectin-1 can attach itself to biological substances that are already present in the body, such as HMGB1, a protein released by damaged or dying cells. This binding also promotes the inflammatory process [11].

In type 2 diabetes, activated dectin-1 has a number of negative effects. has a number of metabolic ramifications. First of all, it contributes to insulin resistance, a condition in which chronic inflammation impairs insulin signaling processes. Second, it might hasten the development of complications from diabetes, such as cardiovascular disease, retinopathy, and nephropathy. Dectin activation may have an impact on the gut microbiome, changing the makeup of gut bacteria and resulting in metabolic dysregulation. Since decretin-1 is a major factor in type 2 diabetes, treating the condition by focusing on the protein would be a wise move. One of the proposed methods for reducing chronic inflammation is to inhibit the dectin-1 protein's activity. Introducing medications that prevent the signaling pathways activated by dectin-1 is another possible treatment option[12, 13, and 14].

2. MATERIALS AND METHODS

2.1. Sample collection

A total of 120 blood samples were collected between October 15, 2024, and December 15, 2024. These samples were obtained from diabetic patients diagnosed by specialist doctors at Baqubah Teaching Hospital in Diyala Governorate. Among the samples, 80 were from patients with type 2 diabetes mellitus, all aged over 35 years. Additionally, 40 samples were collected from healthy individuals who participated in the study as a control group, matched in age with the diabetic patients. The samples were collected through venous blood draws, with 5 ml of blood taken using plastic medical syringes. The drawn blood was divided into two parts. The first part was placed in EDTA tubes (purple-top) for HbA1C determination, as EDTA prevents clotting and ensures accurate hemoglobin measurement. The second part was transferred into gel separator tubes (gold or red-gray top), left to clot for 10–15 minutes, and then centrifuged at 3000 rpm for 10 minutes to separate the serum. The resulting serum was used for further biochemical analyses. The separated serum was divided into two aliquots (500 µL each). The first aliquot was immediately analyzed for fasting blood sugar (FBS), triglycerides (TG), total cholesterol (TC), HDL cholesterol (HDL-C), uric acid, urea, and creatinine using standard biochemical assays. The second aliquot was stored at –20 °C for subsequent evaluation of Dectin-1 levels to ensure sample stability until analysi. The separated serum was aliquoted into two 500 µL portions. The first portion was immediately analyzed for routine biochemical parameters using standardized methods: fasting blood sugar (FBS) was measured by glucose oxidase-peroxidase (GOD-POD) method; triglycerides (TG) by glycerol phosphate oxidase (GPO-PAP) method; total cholesterol (TC) and HDL-cholesterol (HDL-C) by cholesterol oxidase-peroxidase (CHOD-PAP) assay (with HDL-C measured after precipitation of apoB-

containing lipoproteins); uric acid by uricase-peroxidase method; urea by urease-glutamate dehydrogenase (GLDH) method; and creatinine by modified Jaffe method. The second 500 µL aliquot was preserved at -20°C for subsequent quantitative analysis of Dectin-1 levels by Eliza test, maintaining sample stability for future biomarker assessment. All biochemical analyses were performed using automated clinical chemistry analyzers with appropriate quality control measures.

2.2. Statistical analysis

All the statistical analyses were performed using SPSS version 25.0 and Graph pad prism version 6. Depending on the data being investigated, different analysis strategies were used. T-test. It was used when comparing two sets of data depending on the normal or skewed distribution of the data. A one-way ANOVA (or non-parametric equivalent) was used for the comparison of three or more groups. P values of < 0.05 were considered to be statistically significant and illustrated in tables as * = $p<0.05$, = $p<0.01$, * = $p< 0.001$.

3. RESULTS AND DISCUSSION

Figure 1 and Table 1 explain four important parameters for a control and type 2 diabetes patient group. Age distribution with a mean± SD was 53.12 ±11.20 years in T2DM patients and 53.72±11.22 years in the control group, it indicates non-significant difference ($p>0.05$) in both groups. This high degree of concordance for age removes age as a confounding factor by indicating that the groups are highly homogeneous as far as age is concerned. A featured distinction between the groups is their BMIs. The body mass index (BMI) of the type 2 diabetic patients was 28.94±4.20 kg/m², significantly higher than the BMI of the control group, 21.67±1.86 kg/m² ($P<0.05$). This indicates that, diabetics are more susceptible to be obese or overweight. The groups are well separated based on their Fasting Serum Glucose (FSG) levels.

There was a very highly significant difference between the levels of control group (98.45±11.82 mg/dl) and the T2DM patients (179.97±79.47 mg/dl) with a p-value of less than 0.001. Figure (1) shows that there is more glycaemic variability in patients with diabetes, as evidenced by the larger standard deviation in the T2DM group. Percentages of HbA1C, which indicate the extent to which blood glucose levels have been controlled over a sustained period of time, were considerably higher in the T2DM group (8.052±1.939%) than in the control group (5.059±0.413%). This highly significant difference ($p<0.0001$) confirm glycaemic control in the diabetics, since HbA1C levels above 6.5% are diagnostic of diabetes. These results collectively show that type 2 diabetics and healthy controls have different metabolic profiles.

Table 1. Comparison of age BMI, FBG and HBA1C, in patients and control.

Dependent variable	Mean± SD T2DM Patients	Mean± SD Control	p-value
Age Years	53.12±11.20	53.72±11.22	0.478
BMI Kg/m²	28.94±4.20	21.67±1.86	P<0.001
FSG Mg/dl	179.97±79.47	98.45±11.82	P<0.001
HBA1C %	8.052±1.939	5.059±0.413	P<0.0001

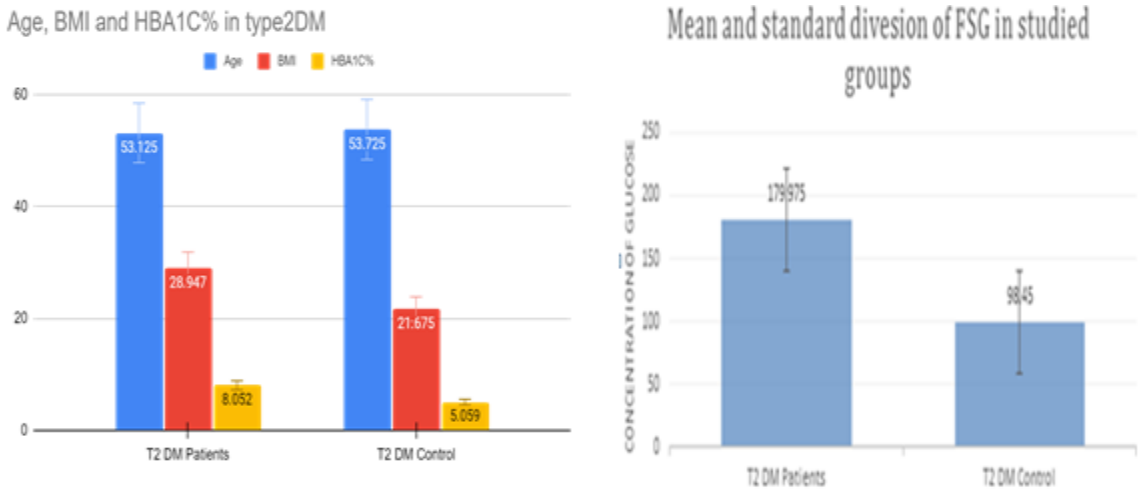

Figure 1. The bar graph compares three key parameters—age, BMI, HBA1C%, and FBG—in studied groups.

Between the control group and T2DM patients, Table 2 shows comparative analysis of three of the most important markers: urea, uric acid, and creatinine, hence illustrating interesting trends in kidney function markers. The level of uric acid (5.010±1.0855mg/dl) revealed non-significant rise ($p<0.052$) in T2DM patients as compared to the control group (4.340±0.1984mg/dl). The level of creatinine (CRE) was significantly higher in T2DM patients (0.970±0.1937mg/dl) than in controls (0.608±0.1496 mg/dl). This very significant ($p<0.0001$) disparity in creatinine levels is regarded as indicator of disparity in muscle mass or renal function between the two groups. Regarding urea level , as shown in table 2 , T2DM patients have comparatively higher mean values (28.500±6.3448mg/dl) than controls (25.500±6.228 mg/dl), the urea (URE) levels experienced a non-significant disparity (p-value = 0.21). This quite small variation in the urea levels could suggest that renal function, as explained by the clearance of urea, is not significantly different between groups.

Table 2. Comparison of urea, creatinine, and uric acid between patients and control groups

Dependent variable	Mean± SD T2DM Patients	Mean± SD Control	p-value
URIC ACID Mg/dl	5.010±1.0855	4.340±0.1984	0.052
CRE Mg/dl	0.970±0.19370	0.608±0.1496	< 0.0001
URE Mg/dl	28.500±6.3448	25.500±6.228	0.21

Type 2 diabetes patients' lipid profile levels and a control group are compared in Table 3 and Figure 2, with interesting changes in all the lipid components studied. Total cholesterol was significantly elevated in type 2 diabetes mellitus patients, with considerable individual variation, having a mean of 206.650±51.1336 mg/dl and a fairly wide standard deviation. The lower total cholesterol value in the control group, mean of 147.500±7.5037 mg/dl, and a lower SD than in the patient population, suggests that healthy individuals have more consistent levels of cholesterol. There was a significant difference ($p< 0.038$) between the two groups.

A high significant difference between the groups was seen in triglyceride values ($p<0.0001$). In the diabetic group, the range of triglyceride values was phenomenal, and the mean was 227.810 mg/dL and the SD was excessively high at 109.653 mg/dL. The mean for the control group was 67.900 mg/dL, and the SD was 7.5037 mg/dL.

In type 2 diabetes patients, low density lipoprotein (LDL), also known as "bad cholesterol," was considerably greater with a mean of 102.305 mg/dL and SD of 38.2519 mg/dL compared with a mean of 73.750 mg/dL and SD of 6.6481 mg/dL for the control group. Cardiovascular risk factors appear to be higher in diabetic patients with a difference of 0.042 being statistically significant.

The "good cholesterol," high density lipoprotein (HDL), followed an unexpected pattern. Patients with type 2 diabetes had decreased HDL values (47.835 mg/dL on average and 38.2519 mg/dL as SD) compared to the control group with increased HDL values (58.700 mg/dL on average with a much smaller SD of 4.852 mg/dL). There was a high statistically significant decrease in HDL (0.0013). These data raise. Remarkably, these results verify that type 2 diabetics possess dyslipidaemia, which is characterized by higher levels of total cholesterol, triglycerides, LDL and loer level of HDL as compared to the control group. Greater variability of lipid metabolism in diabetic patients, perhaps reflecting heterogeneity of the disease and the level of metabolic control, is suggested by the higher standard deviations in the T2DM group on all parameters.

Table 3. Comparison of lipid profile between patients and control groups

Dependent variable	Mean± SD T2DM Patients	Mean± SD Control	p-value
Total Cholesterol (mg/dL)	206.650±51.1336	147.500±7.5037	0.038
Triglycerides (mg/dL)	227.810±109.653	67.900±7.503	0.0001
LDL (mg/dL)	102.305±38.2519	73.750±6.6481	0.042
HDL (mg/dL)	47.835±8.2519	58.700±4.8352	0.0013

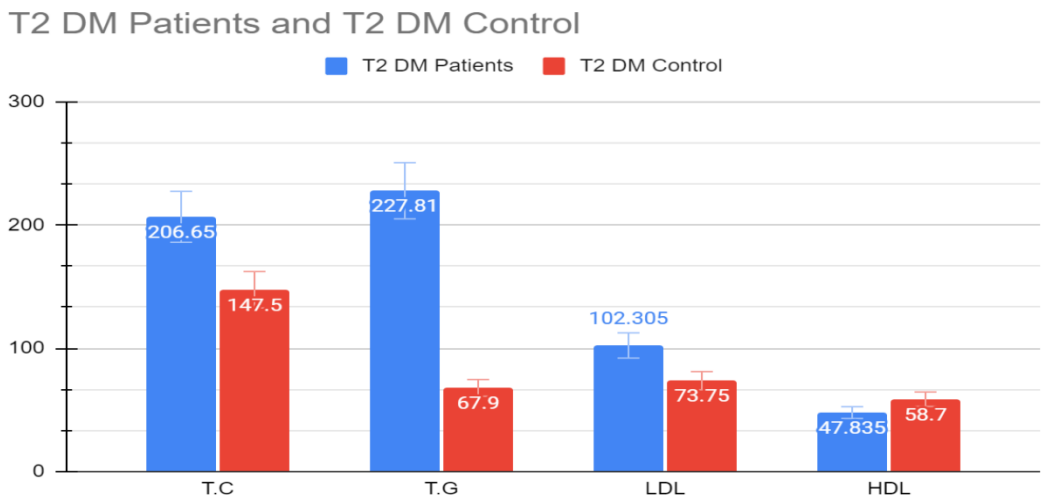

Figure 2. The differences in lipid profile parameters between T2DM patients (blue bars) Compared with a control group (red bars)

Dectin-1 level was compared in healthy control and type 2 diabetic patients (see Table 4 and Figure 3 below). Mean value: 3008.889, compared to 1966.500 Difference between the two means was 1042.389 with standard error being 183.47. The existence of statistically significant difference between both groups was evident through t-test value of 3.283 having a p-value of < 0.001. The result is that diabetic patients' dectin-1 level was significantly higher than the control group.

Table 4. Comparison of Dectin-1 between type 1 diabetic patients and control groups

Groups		Dectin-1ng/ml	Mean difference	Standard error	t-test	p-value
T2 DM Patients	Mean	3008.889	1042.889	183.47	3.28	P<0.001
	SD	597.1149				
Control	Mean	1966.500				
	SD	143.8580				

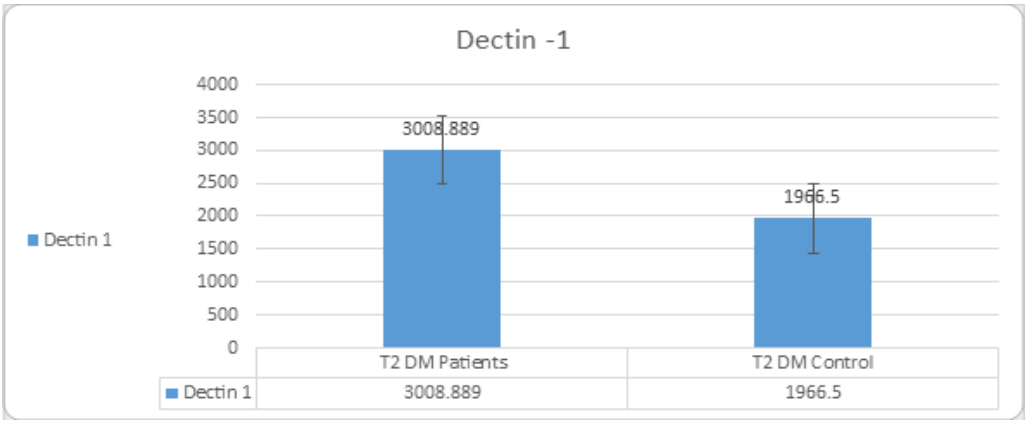

Figure 3. Comparison of Dectin 1 Levels between T2 DM patients and control groups

3.1. Correlations

Correlations of quantified FBG, HbA1C Urea, Creatinine and Uric Acid with Dectin-1 blood levels in type 2 diabetic patients are represented in Table 5 and Figure 4. There was a very weak positive value of 0.11 for the correlation coefficient for fasting blood glucose; the value 0.315 ($p>0.05$) Significant value that indicate no statistically significant association between Dectin-1 and FBG levels. Was Regarding HbA1c, the correlation coefficient of 0.15 and the significant value was 0.138 ($p>0.05$), HbA1c % study demonstrated also very weak positive correlation between Dectin-1 and HbA1c %, hence no statistically significant relationship between Dectin-1 and HbA1c % . In type 2 diabetes patients, HbA1c % study also demonstrated different

levels of associations between blood concentrations of Dectin-1 and renal function markers.

Data for urea is a weak positive correlation coefficient of 0.253 with a significant value of 0.09 ($p>0.05$) and therefore suggesting no statistically significant relationship between the level of Dectin-1 and the level of blood urea. According to the level of creatinine, serum creatinine and Dectin-1 did show a statistically significant relationship in the form of a moderate positive correlation coefficient of 0.3 with a significant value of 0.009 ($p<0.05$). This indicates that creatinine levels also tend to rise in line with increasing Dectin-1 levels, therefore appears to be a correlation between kidney function and Dectin-1 was recorded. For uric acid, the correlation coefficient was 0.22 and the significance value was 0.057, slightly greater than the typical significance value of 0.05. This indicated a weak positive correlation with Dectin-1. While this correlation with uric acid approaches statistical significance, anyway, a statistical significance was recorded.

There are no statistically significant correlations between total Dectin-1 concentrations in blood and lipid profile measures among type 2 diabetic patients, were a very weak association between total Dectin-1 levels and total cholesterol, as indicated by the very weak positive correlation coefficient of 0.02 with a high p-value of 0.869 (Non-Significant). There was non-significant relationship of triglycerides with this variable ($p=0.23$, NS), and 0.19 coefficient. A weak relationship of LDL cholesterol and Dectin-1 was shown by 0.03 correlation coefficient and. No significant p-value of 0.871(NS). HDL had the worst negative relationship between the lipid parameters (correlation coefficient =0.24, $p=0.097$), revealing non-significant difference between the two parameters.

Table 5. Correlation of Dectin-1 with FBG and HBAIC in Type 2 diabetic patients

Variable	Serum Dectin-1 in type 2 diabetic patients	
Fasting blood Glucose	Correlation coefficient	0.11
	Significant value	0.315
HbA1C	Correlation coefficient	0.15
	Significant value	0.138
Urea	Correlation coefficient	0. 253
	Significant value	0. 09
Creatinine	Correlation coefficient	0.3
	Significant value	0.009
Cholesterol mg/dl	Correlation coefficient	0.02
	Significant value	0.869
Triglyceride mg/dl	Correlation coefficient	0.19
	Significant value	0.23
LDL mg/dl	Correlation coefficient	0.03
	Significant value	0,871
VLDL mg/dl	Correlation coefficient	-0.24
	Significant value	0.097
Uric acid	Correlation coefficient	0.22
	Significant value	0.057

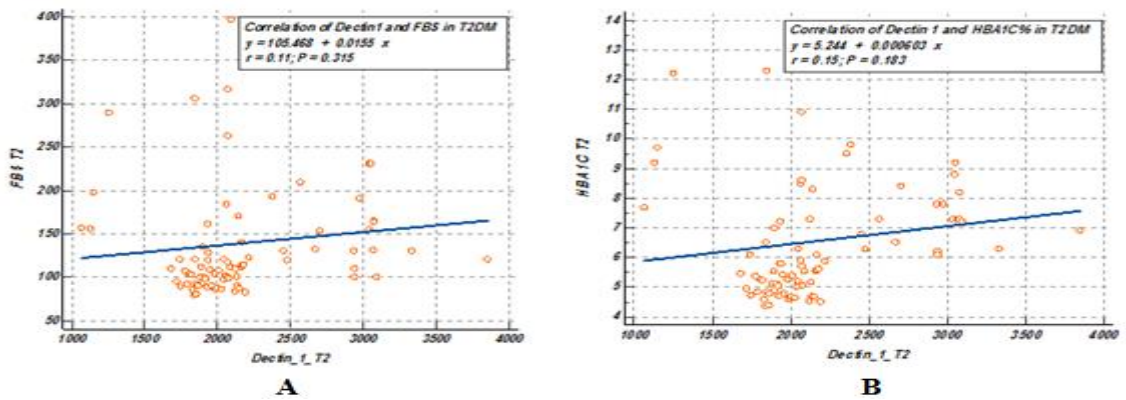

Figure 4. **A)** Personal correlation of Dectin-1 and fasting blood glucose in T2 diabetic patients. **B)** Personal correlation of Dectin-1 and HBA1C% in T2 diabetic patients

3.2. Receiver Operating Characteristic curve

The results of receiver operating characteristic (ROC) curve were described in Figure 5 and table 7. The present work highlights the possible application of serum Dectin-1 as a marker of type 2 diabetes diagnosis. The value of Area Under the Curve (AUC) near to 1.0 suggests ideal discrimination, and 0.722 describes a fair good capability. With a Standard Error of 0.0622, the estimation of the AUC is very accurate. Since the 95% Confidence Interval is not including 0.5, i.e., the test is discriminatory, we are 95% confident that the actual AUC lies between this intervals (0.611 to 0.816). This AUC is dramatically below 0.5 (which would indicate a test at chance) based on the extremely significant p-value of 0.0004 ($p<0.05$) and z-statistic of 3.570.

Table 7. Area under the ROC curve (AUC)

Area under the ROC curve (AUC)	0.722
Standard Error [a]	0.0622
Specificity	100%
Sensitivity	52.5%
95% Confidence interval [b]	0.611 to 0.816
z statistic	3.570
Significance level P (Area=0.5)	0.0004

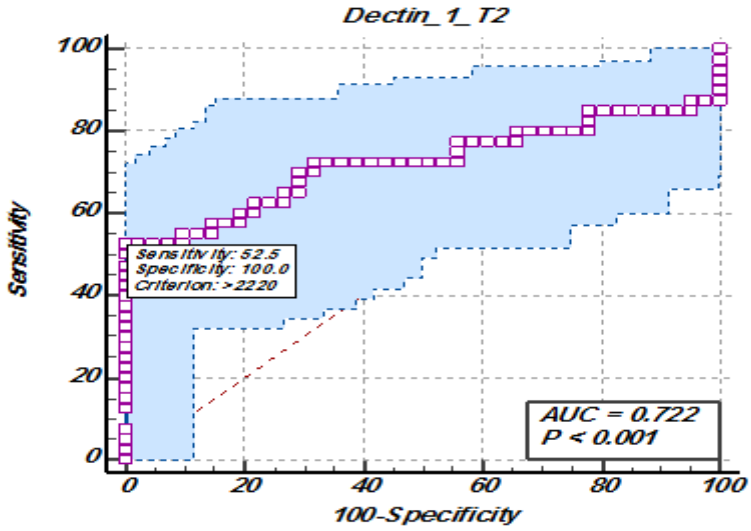

Figure 5. Presented ROC curve research proving the performance of Dectin-1 as a biomarker for the diagnosis of Type 2 diabetes.

The curve reflects moderate to high diagnostic accuracy with an Area under the Curve (AUC) of 0.722 and a significant p-value of >0.001. The test is 52.5 sensitive and 100 specific at the cutoff value of less than 2220. The 95% confidence interval is the light blue area, while the true ROC curve is the dotted pink line. It is proven that the test is discriminatory because the curve clearly deviates from the 50-degree reference line. A good capacity to identify properly the negative cases is implied by high specificity (100%) and an acceptable capacity to identify positive conditions is evidenced by the moderate sensitivity (52.5%). Dectin-1 may be a useful diagnostic tool as these statistical findings have shown, especially when high specificity is a major concern relative to sensitivity. These findings are highly dependent because the results are highly unlikely to have happened by chance, as indicated by the extremely high P-value.

Mostly expressed on immune cells, Dectin-1 is a vital pattern recognition receptor that has become increasingly important molecular component in understanding the intricate pathophysiology of type 2 diabetes mellitus (T2DM). Recent studies by Wu et al. (2021) [15] and Zhang et al. (2021) [16] have reported a complex link between Dectin-1 signalling and metabolic inflammation, therefore exposing its possible influence in reducing insulin resistance and chronic inflammatory processes. The capacity of the receptor to identify β-glucan structures and initiate inflammatory cascades via NF-κB channels points to a fundamental relationship between metabolic imbalance and immune system performance. Fundamentally, Dectin-1 induces oxidative stress generation, immune cell recruitment, and cytokine production—all of which are basic processes causing insulin sensitivity reduction[17]. Emerging data suggests that Dectin-1's molecular interactions might be a suitable treatment target for T2DM [17].

Emerging as a fundamental molecular mediator in comprehending the intricate interaction among immune function, metabolic health, and ageing is Dectin-1. The complex interactions between Dectin-1 expression and important physiological markers—including age, Body Mass Index (BMI), glycated haemoglobin (HbA1c), renal function markers, and lipid profile—have lately been highlighted by new scientific studies[18].

Regarding age and Dectin-1 modification: Chen et al. (2022) conducted longitudinal investigations showing a noteworthy relationship between changing Dectin-1 receptor functionality and advancing age [18]. The immunomodulatory ability of the receptor changes significantly as people move through several phases of life. Immunosenescence as a condition were Dectin-1's inflammatory response systems become gradually impaired, may be influenced by the aging . Elderly people have lower Dectin-1 receptor expression and less inflammatory response capacity, according to research by Zhang et al. (2021), which would help to explain their possible vulnerability to metabolic diseases and chronic inflammatory conditions linked with Dectin-1. [16].

Dectin-1 Interactions: BMIs , Dectin-1 and Body Mass Index interact dynamically and holistically in metabolism. Rising BMI and improved Dectin-1 signalling show a noteworthy positive connection, according to a thorough meta-analysis by Wu and colleagues (2022[15]). Dectin-1 receptors activated by obesity-related inflammatory states set off pro-inflammatory cascades that aggravate metabolic abnormalities. Increased macrophage intrusion and Dectin-1 receptor activation resulting from adipose tissue growth create an always inflammatory milieu that fuels metabolic syndrome and insulin resistance. Our present work emphasises the substantial correlation between BMI and Type 2 Diabetes and strongly implies that it is a necessary to differentiate criterion between diabetic and control participants.

Regarding Glycaemic Control, HbA1c is a key indicator of long-term glycaemic management, glycated haemoglobin (HbA1c) exhibits complex interactions with Dectin-1 signalling that have been discovered in recent studies. Rising HbA1c levels have been linked by Lee et al. (2023) to increased Dectin-1 receptor activation, therefore implying a bidirectional link between glycaemic dysregulation and immune system modulation[19]. Hyperglycaemic disorders induce higher Dectin-1 expression, therefore encouraging inflammatory reactions that further impair glucose metabolism and insulin sensitivity[19].

Dectin-1 and renal function markers have a complex interaction that may be affact the causes of kidney failure in metabolic diseases. Kim et al. (2022) conducted research showing how drastically changed Dectin-1 signalling influences renal inflammatory processes. On the other hand , increased urea and creatinine levels correspond with altered Dectin-1 receptor expression, implying a possible mechanical relationship between immunological dysfunction and kidney pathology. The ability of the receptor to control inflammatory reactions could help to explain both decreased kidney performance and increased renal damage[17].

Regarding Lipid Profile Dynamics, A remarkable topic of the modern research is Dectin-1's participation in lipid metabolism. Rodriguez et al. (2023) conducted a novel investigation showing complicated relationships between Dectin-1 signalling and lipid profile parameters. Increased Dectin-1 receptor activation linked elevated total cholesterol, LDL, and triglyceride levels to a possible molecular pathway connecting immune activity and lipid metabolism [20]. The many character of Dectin-1 points to a complete involvement in metabolic control. An integrated study has shown how the receptor functions as a crucial molecular interface between immune function and metabolic balance which was proposed by Wang et al. (2022)[21].

Knowing the complex interactions between Dectin-1 and other metabolic criteria creates interesting options for focused therapeutic actions. Dectin-1 receptor modification could be used in personalised medicine to treat metabolic dysfunctions, thereby perhaps reducing age-related inflammatory reactions and enhancing general metabolic condition.

The results related to Dectin-1 level in table 4 clearly indicate a high correlation between Type 2 diabetes mellitus and raised Dectin-1 levels, which may have significant consequences for knowledge of disease processes and future treatment development. Strong proof reveal that these variations are probably related to the underlying illness process in T2DM patients rather than random comes from the statistical relevance ($P<0.001$) these result agreed with Ali, A et al [22].

Except creatinine, the results of correlations for dectin-1 with other T2DM indicators calculated in this study (FBG, HbA1C, Urea, Creatinine, Uric acid and lipid profile) indicated non-significant correlation These results are especially interesting since they show that, although Dectin-1 levels may be higher in T2DM patients, they show no significant correlation with either immediate blood glucose levels or long-term glucose control as determined by HBA1C%. This implies that additional factors of the disease process could affect the rise of

Dectin-1 in diabetes patients instead of being directly related to blood glucose control. The absence of notable relationships with both immediate and long-term glucose indicators suggests that Dectin-1 function in type 2 diabetes may be more complicated and maybe independent on direct glucose metabolism pathways.

Among the investigated renal indicators, creatinine reveals the strongest and has the only statistically significant correlation with Dectin-1 levels. This suggests a possible function of Dectin-1 in renal function or disease progression in type 2 diabetic patients, particularly in respect to processes reflected by creatinine levels. Combining the weak or missing associations with urea and uric acid with the strong correlation with creatinine it has been implied that Dectin-1's relationship with kidney function is specific for to some aspect of renal physiology rather than being a general indication of kidney status.

In type 2 diabetic individuals, the Dectin-1 levels show minimal correlation with indicators of lipid metabolism. The regularly non-significant p-values across all lipid parameters—cholesterol, triglycerides, LDL, and HDL—indicate that Dectin-1 levels seem to be independent of changes in lipid profile. This implies that although Dectin-1 could have several roles in type 2 diabetes, its functions might not be directly connected to lipid metabolism or cardiovascular risk factors related with the lipid profile as agreed with Sahab, K. S., & Al-Saadi, A. S. M. (2019),Al-Nadawi, H. M., & Al-Dulaimy, W. Y. M. (2024). [23, 24].

the statistical analyses of ROC curve show that serum Dectin-1 has medium to good accuracy in differentiating between diabetic and non-diabetic people, so its value is suggested as a diagnostic marker in type 2 diabetes.

CONCLUSION

Dectin-1 emerges as a critical molecular mediator which remarkably link the immune function and metabolic health. Dectin-1. Was significantly increased in T2DM patients and was Dectin-1 non-significantly correlated with BMI, HbA1c, renal markers, and lipid profile Its dynamic interactions with BMI, HbA1c, renal markers, and lipid profile highlight the receptor's significance in understanding complex physiological processes biochemical marker.. The results of statistical analysis of ROC curve suggest that Dectin-1 could be considered as a useful biochemical marker, though its moderate AUC value indicates it might be most effective when used in combined with other diagnostic tools rather than as a standalone test. Briefly, Dectin-1 is a key biochemical marker in Iraqi patients with type -2 diabetes.

Conflict of interest

No conflict of interest among the authors.

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Non

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