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Evaluation of GDF11 And Lipid Profile in Diabetic Dyslipidemia

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Abstract

Background

Diabetic dyslipidemia is a major risk factor for atherosclerosis and cardiovascular complications. Growth differentiation factor 11 (GDF11), a member of the transforming growth factor beta (TGF-β) superfamily, has a role in metabolic regulation, vascular health, and aging, but its role in diabetic dyslipidemia remains unclear.

Patients and Methods: A comparative analysis was conducted between diabetic patients with dyslipidemia and those without. Serum GDF11 concentrations and lipid parameters were measured, along with additional markers of oxidative stress and inflammation. Statistical analysis was performed to assess group differences and correlations.

Result: GDF11 shows a strong positive correlation with total cholesterol and triglycerides in diabetic patients, suggesting a role in lipid abnormalities, while no significant association is seen with HDL or LDL. A modest positive correlation is also observed between GDF11 and AST, indicating a possible link to liver involvement. Hematological findings reveal distinct patterns between diabetic patients with and without dyslipidemia. Those without dyslipidemia exhibit higher WBCs, granulocytes, lymphocytes, and platelets, reflecting greater inflammatory and immune activity. In contrast, patients with dyslipidemia have elevated erythrocytic indices (hemoglobin, RBC count, hematocrit, and MCHC), indicating altered red cell production. Additionally, RDW and platelet indices are higher in the non-dyslipidemia group, suggesting increased variability in cell size. Overall, dyslipidemia in diabetes is associated with enhanced erythropoietic changes, while non-dyslipidemic patients display stronger inflammatory and platelet responses

Conclusion: Higher GDF11 levels are strongly associated with higher LDL, total cholesterol, and triglycerides. GDF11 also rises with HDL, but less strongly compared to LDL/TC. This suggests that GDF11 may play a role in lipid metabolism regulation, possibly being more tightly linked to lipid markers (LDL, TG, TC) than protective HDL.

تقييم عامل اختلاف النمو 11 ومستوى الدهون في مرضى السكري المصابين باضطراب شحميات الدم

سرى هادي إبراهيم، نغم يحيى غافل

المخلص

المقدمة: يُعدّ اضطراب شحوم الدم السكري عامل خطر رئيسيًا لتصلب الشرايين ومضاعفات القلب والأوعية الدموية. وقد ثبت أن عامل تمايز النمو 11، وهو عضو في عائلة بيتا لعوامل النمو المحولة، له دور في تنظيم التمثيل الغذائي، وصحة الأوعية الدموية، والشيخوخة، إلا أن دوره في اضطراب شحوم الدم السكري لا يزال غير واضح.

. طرق العمل: أُجريت تحاليل مقارنة بين مرضى السكري المصابين باضطراب شحوم الدم وغير المصابين به. وقيست تركيزات عامل تمايز النمو 11 في المصل ومعايير الدهون، بالإضافة إلى علامات إضافية للإجهاد التأكسدي والالتهاب. وأُجري تحليل إحصائي لتقييم الاختلافات بين المجموعات وارتباطاتها

النتائج: يُظهر عامل نمو التمايز 11 ارتباطًا إيجابيًا قويًا مع الكوليسترول الكلي والدهون الثلاثية لدى مرضى السكري، مما يشير إلى دوره في اضطرابات الدهون، في حين لا يُرى أي ارتباط كبير مع البروتين الدهني عالي الكثافة أو البروتين الدهني منخفض الكثافة. كما لوحظ ارتباط إيجابي متواضع بين عامل نمو التمايز 11 وانزيم الكبد مما يشير إلى وجود صلة محتملة بإصابة الكبد. تكشف النتائج الدموية عن أنماط مميزة بين مرضى السكري المصابين بخلل شحميات الدم وغير المصابين به. يُظهر أولئك الذين لا يعانون من خلل شحميات الدم ارتفاعًا في خلايا الدم البيضاء والخلايا الحبيبية والخلايا الليمفاوية والصفائح الدموية، مما يعكس نشاطًا التهابيًا ومناعيًا أكبر. في المقابل، يُظهر المرضى المصابون بخلل شحميات الدم مؤشرات كريات الدم الحمراء المرتفعة (الهيموغلوبين وعدد خلايا الدم الحمراء و مكداس كريات الدم الحمراء وتركيز الهيموغلوبين الكروي المتوسط)، مما يشير إلى تغير إنتاج خلايا الدم الحمراء. بالإضافة إلى ذلك، يكون عرض توزيع خلايا الدم الحمراء ومؤشرات الصفائح الدموية أعلى في المجموعة غير المصابة باضطراب شحميات الدم، مما يشير إلى زيادة التباين في حجم الخلايا. بشكل عام، يرتبط اضطراب شحميات الدم لدى مرضى السكري بزيادة التغيرات المكونة للكريات الحمراء، بينما يُظهر المرضى غير المصابين باضطراب شحميات الدم استجابات التهابية وصفائح دموية أقوى.

الاستنتاج: ترتبط مستويات عامل تمايز النمو 11 المرتفعة ارتباطًا وثيقًا بارتفاع الكوليسترول الضار منخفض الكثافة والكوليسترول الكلي والدهون الثلاثية. يرتفع عامل تمايز النمو 11 أيضًا مع البروتين الدهني عالي الكثافة ولكن بدرجة أقل مقارنةً بالبروتين الدهني منخفض الكثافة / الدهون الثلاثية. وهذا يشير إلى أن عامل تمايز النمو 11 قد يلعب دورًا في تنظيم استقلاب الدهون، وربما يكون مرتبطًا بشكل أوثق بمؤشر الدهون البروتين الدهني منخفض الكثافة والدهون الثلاثية والكوليسترول الكلي من البروتين الدهني عالي الكثافة الوقائي.

1. Introduction

Diabetes mellitus (DM) is a long-term metabolic disease arising from both genetic and environmental determinants, mainly marked by sustained hyperglycemia and commonly presenting with symptoms such as excessive urination and hunger. (Pasquel et al. 2021) Dyslipidemia involves disturbances in lipid metabolism, typically presenting with elevated triglycerides, cholesterol, and LDL alongside reduced HDL, and is recognized as a major contributor to cardiovascular complications. (Anusha et al. 2020) In individuals with type 2 diabetes, diabetic dyslipidemia frequently develops and is strongly associated with an increased risk of atherosclerotic cardiovascular disease. (Bahiru et al. 2021) Atherosclerosis is a progressive vascular condition in which lipid deposits and inflammatory responses lead to plaque formation, narrowing, or rupture of arteries, with elevated LDL identified as a central causal factor. (Gusev 2023) Atherosclerosis can be diagnosis by a novel biomarker such as Midkine, NLRP3 inflammasome and Peroxisome Proliferator-Activated Receptor Gamma (PPARγ) Midkine is a heparin-binding growth factor involved in cell growth, inflammation, and tissue repair; it contributes to atherosclerosis by promoting inflammatory cell recruitment and vascular smooth muscle cell migration, leading to plaque development. (Majaj and Weckbach 2022) The NLRP3 inflammasome contributes to endothelial dysfunction by initiating oxidative stress and inflammatory signaling, thereby accelerating the development of atherosclerotic plaques. (Khair et al. 2024) Peroxisome Proliferator-Activated Receptor Gamma (PPARγ) is defined as a nuclear receptor that regulates genes involved in glucose and lipid metabolism, endothelial function, and inflammation, and PPARγ helps protect against atherosclerosis by improving endothelial function and maintaining healthy blood vessels. Reducing inflammation, a key driver of plaque formation. Stabilizing plaques and slowing their progression, lowering the risk of cardiovascular events. (Staels 2005) Superoxide dismutase (SOD), a key antioxidant enzyme, protects cells by neutralizing reactive oxygen species, thereby supporting vascular integrity and cellular balance. (Application 2018) Growth Differentiation Factor 11 (GDF11), belonging to the TGF-β superfamily, is expressed in various organs and has been implicated in processes such as tissue regeneration, metabolism, and cardiovascular regulation. (Simoni-Nieves et al. 2019) Human evidence indicates that lower circulating levels of GDF11/8 are associated with higher incidence of cardiovascular events, including myocardial infarction, stroke, and heart failure, pointing to its potential as a protective biomarker. (Walker et al. 2025)

2. Materials, Patients and Methods

2.1. Patients' Constant and Enrollment

A cross-sectional study was conducted at the AL-Hassan Metabolism, Endocrine, and Diabetics Center in Karbala, Iraq, from February 2025 to April 2025. There were 172 individuals in total with diabetic types 1 and 2, 90 of whom were diabetic dyslipidemia patients (52 men and 38 women), 4 with type 1 and 86 with type 2, and 80 of whom were diabetic without dyslipidemia (36 men and 46 women), 28 with type 1 and 54 with type 2. people in the age range of 20 to 70. Essential data, including age, gender, family history, education, healthy diet, exercise, diabetic duration, medication history, height, and weight, were collected to calculate the body mass index (BMI) of the participants. Serum samples were collected to evaluate the following parameters: fasting blood glucose (FBG) and growth differentiation factor 1qAA1 (GDF11). Triglyceride (TG), total cholesterol (TC), low-density lipoprotein (LDL),

2.2 Inclusion criteria for case group

1. Patients with dyslipidemia as a complication of type I and type II diabetes mellitus
2. patients with Type I and Type II
3. patients who assent to the application of informed consent.
4. patients who are proficient in reading, speaking, and writing Arabic

2.3 Blood Sample Collection

After an overnight fast, 5 ml of blood was drawn from each patient via vein puncture. The samples were collected in EDTA tubes for plasma extraction.

2.4 Biochemical Assay Methods

The following techniques were used to measure each different parameter following the commercial instructions:

1. Estimation of complete blood count.
2. Estimation of Fasting Serum Glucose Level: Glucose oxidase method.
3. Estimation of serum liver enzyme level. (AST and ALT).
4. Estimation of serum cholesterol, triglycerides, HDL, and LDL: Enzymatic assays.
5. Estimation serum atherogenic biomarker (midkine, NLRP3 inflammasome, PPARy)
6. Estimation of serum oxidative stress superoxide dismutase.
7. Estimation of serum growth differentiation factor 11 (GDF11).

Measurement of Body Mass Index (BMI): BMI was calculated using the formula BMI = Weight / (Height)², and individuals were categorized as normal weight, overweight, or obese based on BMI values. (Asil et al. 2014) (Dewi, Rimawati, and Purbodjati 2021)

Statical Analysis

The analysis was performed using IBM SPSS 28. Data normality was evaluated using the Shapiro–Wilk test and Q–Q plots. Continuous variables that were not normally distributed were presented as median (IQR), while categorical variables were expressed as frequencies and percentages. Group comparisons: Mann-Whitney U test for continuous data and Fisher's exact test for categorical data. Correlations: Spearman's rank correlation was used to examine relationships between GDF11 and other continuous variables, with significance set at $p < 0.05$. Diagnostic analysis: ROC curve analysis assessed GDF11's ability to distinguish between diabetes with and without dyslipidemia, reporting AUC, 95% CI, optimal cut-off, sensitivity, and specificity. All analyses were two-tailed, and $p < 0.05$ was considered statistically significant.

3.Results

3.1 Assessment of Sociodemographic Characteristics of The Study Groups

The results relate different variables with diabetes with and without dyslipidemia and the associated p-value. In Table1, the groups showed considerable similarity in terms of BMI, gender, domicile, education, dietary habits, and physical activity. Marked disparities were noted in age and occupation, with the dyslipidemia cohort being older and more frequently employed.

Table1: Sociodemographic Characteristics of The Study Groups

Variable	Diabetes With Dyslipidemia	Diabetes Without Dyslipidemia	P-Value
Age	53 (48-57)	44 (20-56.5)	**0.002**
BMI	29.1 (27.7-31.9)	27.2 (24.5-32.3)	0.166
Gender (Male)	52 (57.8%)	36 (43.9%)	0.093
Gender (Female)	38 (42.2%)	46 (56.1%)	
Residence (Urban)	85 (94.4%)	82 (100%)	0.06
Residence (Rural)	5 (5.6%)	0 (0%)	
Education (Illiterate)	44 (48.9%)	45 (54.9%)	0.73
Education (High School)	3 (3.3%)	3 (3.7%)	
Education (University)	43 (47.8%)	34 (41.5%)	
Occupation (No)	24 (26.7%)	56 (68.3%)	**<0.001**
Occupation (Yes)	66 (73.3%)	26 (31.7%)	
Healthy Diet (No)	66 (73.3%)	56 (68.3%)	0.504
Healthy Diet (Yes)	24 (26.7%)	26 (31.7%)	
Exercise (No)	72 (80%)	59 (72%)	0.282
Exercise (Yes)	18 (20%)	23 (28%)	

3.2 Assessment of diabetic-related clinical and metabolic parameter

The current study indicates that individuals with diabetes and dyslipidemia Patients with Type 1 DM are much less common in the dyslipidemia group (4.4%) compared to those without dyslipidemia (34.1%), so $p < 0.001$, highly significant while Type 2 DM is more common among patients with dyslipidemia (95.6% vs. 65.9%) as shown in Table2. This supports the strong significance above. Fewer dyslipidemia patients use insulin (15.6%) compared to those without dyslipidemia (41.5%). So highly significant $p < 0.001$ Although overall diabetes duration and fasting glucose did not differ significantly. In oral DM medication Highly significant $p < 0.001$. More dyslipidemia patients use oral DM medications (84.4% vs. 59.8% the dyslipidemia group showed highly significant AST ($p < 0.001$), and oral-medication duration were similar between groups. There is no statistically significant difference in HDL levels between the two groups. Since the p-value is much greater than 0.05, There is a highly significant difference in total cholesterol between the groups. Diabetes without dyslipidemia has notably lower cholesterol levels, and because the p-value is less than 0.001, this difference is very unlikely. The groups differ significantly in triglyceride levels. Diabetes without dyslipidemia shows much lower triglycerides, and the very small p-value confirms a strong statistical difference. There is a significant reduction in LDL levels in diabetes without dyslipidemia compared to diabetes with dyslipidemia. With a p-value below 0.001, this difference is highly significant.

Table2: Assessment of Diabetic-Related Clinical and Metabolic Parameter

Variable	Diabetes With Dyslipidemia	Diabetes Without Dyslipidemia	P-Value
Type (Type 1)	4 (4.4%)	28 (34.1%)	**<0.001**
Type (Type 2)	86 (95.6%)	54 (65.9%)	
Insulin (No)	76 (84.4%)	48 (58.5%)	**<0.001**
Insulin (Yes)	14 (15.6%)	34 (41.5%)	
Insulin Duration (Years)*	2.5 (1-4.75)	7 (5.25-8.5)	**<0.001**
Oral DM Med (No)	14 (15.6%)	33 (40.2%)	**<0.001**
Oral DM Med (Yes)	76 (84.4%)	49 (59.8%)	
Oral Med Duration (Years)*	6 (3-10)	9 (1-12)	0.596
FBG (mg/dL)	181.6 (172.7-303.5)	190 (139.2-250)	0.068
HDL (mg/mL)	31.5 (27-40)	33 (28-40.5)	0.499
Total Cholesterol (mg/dL)	195.5 (184.4-225)	166.6 (127.58-180.2)	**<0.001**
Triglycerides (mg/dL)	164.05 (123.8-198.5)	85.4 (63.1-123.22)	**<0.001**
LDL (mg/dL)	85.3 (69.92-93.97)	68.1 (52.25-72.73)	**<0.001**
AST (U/L)	21.34 (19.37-23.53)	19.77 (15.74-21.68)	**0.009**

3.3 Assessment Hematological Parameters Across the Study Groups

The current study indicates that higher red cell mass with less fluctuation, lower white cell and platelet counts, and slight changes in platelet distribution are all seen in diabetic individuals with dyslipidemia as presented in Table3. These modifications could be a reflection of dyslipidemia-related metabolic and inflammatory changes, indicating a unique hematological profile in this population.

Table 3: Hematological Parameters Across the Study Groups

Variable	Diabetes with Dyslipidemia	Diabetes without Dyslipidemia	p-value
WBC (10^9/L)	6.7 (5.7-8.3)	7.2 (6.2-9.2)	**0.013**
Lymphocytes (10^9/L)	2.3 (1.75-2.4)	2.4 (2.3-2.8)	**0.021**
Lymphocyte (%)	34.8 (28.38-37.3)	30.9 (27.7-40.4)	0.278
MID Cells (10^9/L)	0.3 (0.2-0.4)	0.3 (0.2-0.4)	0.249
MID Cells (%)	5.25 (4-7.38)	4.6 (3.9-5.4)	**0.02**
Granulocytes (10^9/L)	4 (3.62-4.9)	4.4 (3.9-6.2)	**0.011**
Granulocytes (%)	61.2 (58.5-64.58)	65.1 (53.08-67.6)	**0.038**
Hemoglobin (g/dL)	14.25 (12.83-15.15)	13.35 (11.3-14.4)	**<0.001**
MCH (pg)	27.3 (25.1-28.78)	26.9 (25-28.8)	0.902
MCHC (g/dL)	33.5 (32.6-34.3)	32.4 (30.8-33.4)	**<0.001**
RBC (10^{12}/L)	5.41 (4.93-5.64)	4.63 (4.45-4.97)	**<0.001**
MCV (fL)	79.5 (74.2-84.65)	82.35 (78.3-87.3)	0.078
HCT (%)	41.9 (38.4-45.6)	39.95 (34.5-42.4)	**<0.001**
RDW (fL)	49.1 (46.8-51.1)	51.7 (47.8-55.8)	**<0.001**
RDW (%)	11.3 (11-11.95)	12.6 (11.8-14.28)	**<0.001**
Platelets (10^9/L)	237 (223-283.5)	275 (223-335)	**<0.001**
MPV (fL)	8.9 (8.6-9.85)	9 (8.7-9.4)	0.543
PDW (fL)	11.7 (11.4-13.07)	11.8 (11.4-12.1)	0.286
PDW (%)	42 (39.8-43.4)	40.5 (39.8-41.8)	**0.01**
Platelets (%)	0.21 (0.2-0.26)	0.25 (0.2-0.31)	**<0.001**
P-LCR (%)	20.05 (18.8-27.18)	21.1 (18.3-22.55)	0.537
P-LCC (10^9/L)	52 (42-67)	60 (47-75)	**0.004**

3.4 Assessment of Spearman's Correlation Between Growth, Differing Nation Factor Levels, And Study Parameter

GDF11 levels are closely associated with hematological indices, particularly red blood cell characteristics (positive with MCHC, RBC, HCT, and hemoglobin; negative with RDW). Among metabolic factors, BMI shows a negative association, while cholesterol and triglycerides show weak positive associations. Age shows a small positive correlation, and many other clinical parameters show no significant association, see Table4 and Fig.1A-E.

Table4: Spearman's Correlation Between GDF11 Levels and Study Parameter

Variable	Spearman Coefficient (r)	p-value
Age	0.168	**0.028**
BMI	-0.214	**0.005**
DM Duration (Years)	0.120	0.116
Insulin Duration	0.231	0.114
Oral Med Duration (Years)	0.107	0.226
FBG (mg/dL)	0.084	0.275
HDL (mg/mL)	-0.012	0.879
Total Cholesterol (mg/dL)	0.299	**<0.001**
Triglycerides (mg/dL)	0.156	**0.041**
LDL (mg/dL)	0.052	0.494
AST (U/L)	0.077	0.316
WBC (10^9/L)	-0.305	**<0.001**
Lymphocytes (10^9/L)	-0.157	**0.039**
Lymphocyte (%)	0.136	0.075
MID Cells (10^9/L)	0.101	0.186
MID Cells (%)	0.264	**<0.001**
Granulocytes (10^9/L)	-0.311	**<0.001**
Granulocytes (%)	-0.169	**0.027**
Hemoglobin (g/dL)	0.217	**0.004**
MCH (pg)	-0.004	0.962
MCHC (g/dL)	0.454	**<0.001**
RBC (10^{12}/L)	0.291	**<0.001**
MCV (fL)	-0.088	0.249
HCT (%)	0.175	**0.021**
RDW (fL)	-0.115	0.134
RDW (%)	-0.500	**<0.001**
Platelets (10^9/L)	-0.091	0.234
MPV (fL)	-0.143	0.061
PDW (fL)	-0.084	0.272
PDW (%)	0.094	0.221
Platelets (%)	-0.149	0.051
P-LCR) (%)	-0.099	0.194
P-LCC (10^9/L)	-0.213	**0.005**
GDF 11 (ng/L)	1.000	**<0.001**

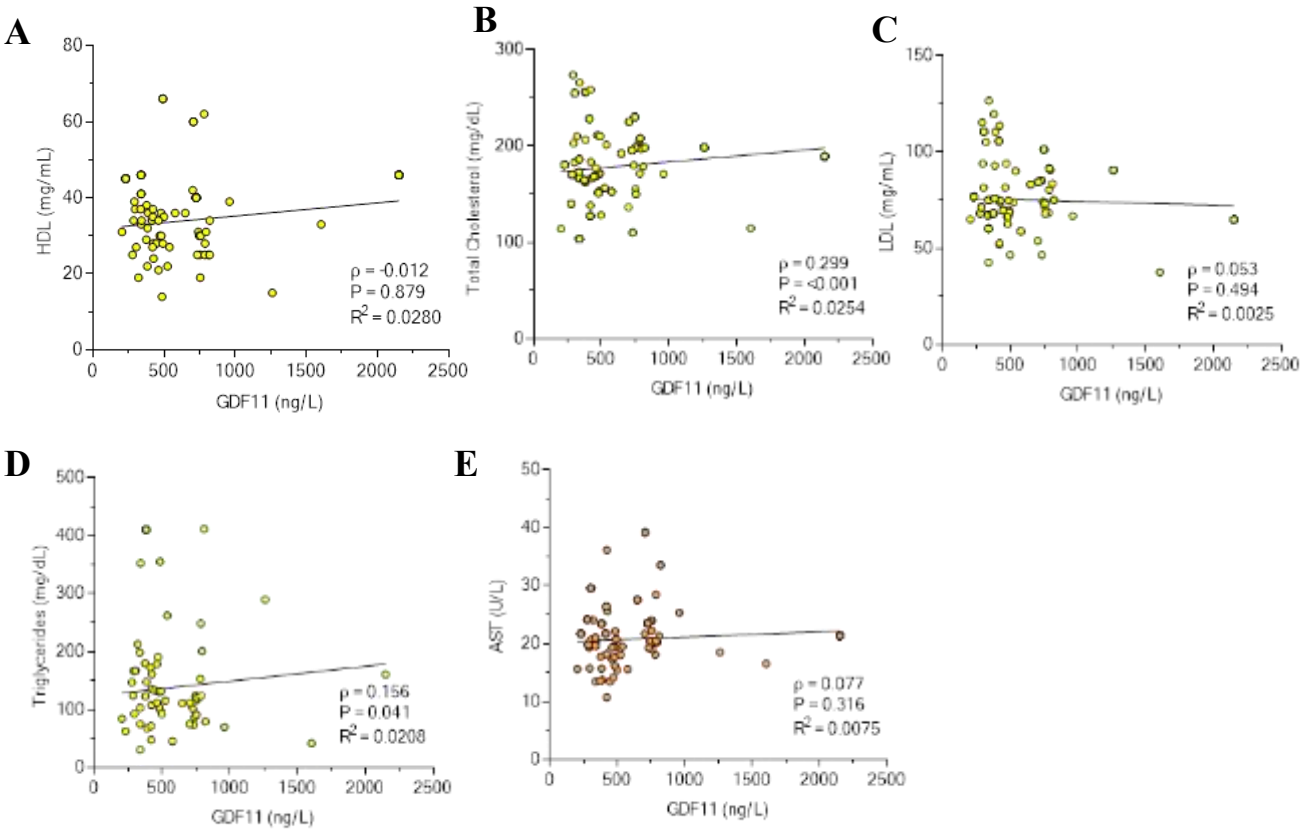

Figure1: Correlation Between Serum GDF11 Levels and Metabolic and Hepatic Parameters. Scatter plots depict the relationships between circulating GDF11 concentrations and (A) total cholesterol, (B) LDL cholesterol, (C) triglycerides, (D) AST activity, and (E) HDL cholesterol. A weak but statistically significant positive correlation was observed between GDF11 and total cholesterol ($\rho = 0.299$, $P < 0.001$, $R^2 = 0.025$), indicating that GDF11 explains approximately 2.5% of total cholesterol variability. No significant association was found between GDF11 and LDL cholesterol ($\rho = 0.053$, $P = 0.494$, $R^2 = 0.002$). GDF11 demonstrated a weak yet statistically significant positive correlation with triglyceride levels ($\rho = 0.156$, $P = 0.041$, $R^2 = 0.021$). In contrast, no significant correlation was detected between GDF11 and AST activity ($\rho = 0.077$, $P = 0.316$, $R^2 = 0.008$). Overall, GDF11 shows weak associations with lipid parameters and no meaningful relationship with hepatic enzyme activity.

4. Discussion

As shown in table 1, both older age and employment status are significantly linked to dyslipidemia among diabetic patients. These observations align with previous research, which recognizes increasing age as a contributing factor to metabolic disturbances in individuals with diabetes. (Ahmmed et al. 2021) According to table 2 patients with dyslipidemia were diagnosed with type 2 diabetes (95.6%). This aligns with existing evidence indicating that lipid disorders are characteristic of insulin-resistant type 2 diabetes rather than type (Masduki 2020). **The** lower insulin use and shorter insulin duration among dyslipidemia patients suggest greater dependence on oral agents and less intensive glycemic control, which may exacerbate lipid imbalance (Haile and Timerga 2020). Meanwhile, AST levels were significantly higher in the dyslipidemia group, reflecting possible hepatic steatosis and metabolic stress, a well-recognized feature of diabetic dyslipidemia. (Manikat, Ahmed, and Kim 2023) Overall, the data reinforce that Type 2

diabetes with dyslipidemia represents a metabolically burdened phenotype, characterized by hepatic involvement and increased cardiovascular In table 3**:** The hematological findings reveal distinct alterations between the two diabetic groups. Patients with dyslipidemia showed lower WBC, lymphocyte, and granulocyte counts, suggesting a suppressed or dysregulated inflammatory response, whereas higher hemoglobin, RBC count, hematocrit, and MCHC indicate possible hemoconcentration or compensatory erythropoiesis linked to chronic metabolic stress. In contrast, platelet count and plateletcrit were lower, and PDW slightly higher, pointing to platelet activation and altered morphology, both associated with endothelial dysfunction in dyslipidemia states. Collectively, these trends imply tha**t** diabetes with dyslipidemia carries a hematological profile consistent with low-grade inflammation and heightened cardiovascular risk (Onalan, Gozel, and Donder 2019)

In table 4 : The data presented reveals several significant correlations between GDF11 levels and various clinical parameters. Notably, GDF11 levels exhibit a positive correlation with total cholesterol, hemoglobin, RBC count, MCHC, HCT, and MID cell percentage, suggesting a potential role in hematologic and lipid metabolism. Conversely, negative correlations are observed with WBC count, granulocyte percentage, RDW percentage, and P-LCC, indicating possible associations with inflammatory and red blood cell distribution parameters. Additionally, GDF11 levels are weakly positively correlated with age and negatively correlated with BMI, aligning with findings from previous studies that report a decline in GDF11 levels with aging and its association with metabolic parameters (Adela, Reddy, and Banerjee 2015)

5. Conclusion

The study shows that GDF11 levels have no significant correlation with most lipid profile components (HDL and LDL) in individuals with diabetic dyslipidemia. Although **a** weak positive association was observed between GDF11 and total cholesterol, the relationship was minimal and explained very little of the variation. Overall, GDF11 does not appear to play a major role in lipid metabolism **among** diabetic patients with dyslipidemia. Further studies with larger samples are needed to clarify its potential metabolic significance.

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