Journal of Population Therapeutics & Clinical Pharmacology

RESEARCH ARTICLE
DOI: 10.53555/3kaj4y28

CORRELATION AMONG LIVER FUNCTION, INSULIN RESISTANCE, LIPID PROFILE, AND GLYCEMIC MARKERS IN TYPE 2 DIABETES MELLITUS PATIENTS

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Abstract

Background: Type 2 Diabetes Mellitus (T2DM) is associated with impaired glucose metabolism, insulin resistance, dyslipidemia, and hepatic dysfunction. Understanding the interrelationship between these metabolic parameters can improve clinical management and risk assessment.

Objective: To evaluate the correlation among glycemic markers, insulin resistance, lipid profile, and liver function in patients with T2DM.

Methods: A cross-sectional case-control study was conducted on 200 T2DM patients and 200 age- and gender-matched healthy controls at Krishna Mohan Medical College & Hospital, Mathura, from March 2024 to February 2025. Fasting blood glucose, HbA1c, HOMA-IR, lipid profile, and liver enzymes (ALT, AST, ALP) were measured. Data analysis was performed using SPSS v25.0, with independent sample t-tests and Pearson correlation; $p < 0.05$ was considered statistically significant.

Results: T2DM patients exhibited significantly elevated fasting glucose (156.8 ± 42.5 mg/dL), HbA1c (8.4±1.5%), HOMA-IR (4.8±1.9), total cholesterol (211.3±38.4 mg/dL), triglycerides (189.7±56.1 mg/dL), LDL-C (126.5±32.9 mg/dL), and liver enzymes (ALT 49.8±15.6 U/L, AST 45.2±14.2 U/L, ALP 122.6±31.8 U/L) compared to controls (all $p < 0.05$). HDL-C was significantly lower (38.9±8.2 mg/dL vs. 48.6±7.3 mg/dL, $p < 0.001$). HbA1c showed strong positive correlations with fasting glucose ($r = 0.812$) and HOMA-IR ($r = 0.758$), moderate positive correlations with total cholesterol, triglycerides, LDL-C, and weak positive correlations with liver enzymes. HDL-C correlated negatively with HbA1c.

Conclusion: T2DM patients demonstrate poor glycemic control, pronounced insulin resistance, dyslipidemia, and mild hepatic dysfunction. The observed correlations emphasize the interconnected nature of glucose metabolism, lipid abnormalities, and liver function, underscoring the importance of comprehensive metabolic monitoring in T2DM management.

Keywords: Type 2 Diabetes Mellitus, Insulin Resistance, Lipid Profile, Liver Function, Glycemic Markers.

Introduction:

The term diabetes mellitus originates from a combination of the Greek word "diabetes," meaning to siphon or drain, and the Latin word "mellitus," meaning sweet. Historical evidence suggests that the term diabetes was first introduced by Apollonius of Memphis.[1] Diabetes mellitus (DM) refers to a group of metabolic disorders characterized by chronically elevated blood glucose levels. The condition develops due to a complex interaction between genetic predisposition and environmental influences, leading to various forms of the disease.[2] According to CDC statistics presented in 2017, concerning 30.2 million persons aged eighteen or older, or >12 percent of all adults in the USA, have diabetes with type 2 (T2DM). One-quarter of these persons (23.8%) were unaware that they had diabetes. T2DM prevalence rose with age, peaking at 25.2% amongst US seniors (65 and older).[3]
DM is divided into two major categories: Classification of DM and Other Groupings for:

- Type 1 DM (βCell Death normally causes complete reduction of insulin level)
- Type 2 DM, which can range from being primarily caused by insulin confrontation with relative insulin shortage to being primarily caused by insulin resistance due to an insulin secretory abnormality.[4]
- Additional Particular class of hyperglycaemia (Exocrine Pancreas Diseases, Genetic Insulin Action impairment, Genetic - Cells Defects, a disease of endocrine gland, Drug- or Chemical-persuade Diabetes).[5]

Insulin Resistance (IR):

Along with visceral obesity, insulin resistance is a significant marker in the occurrence of the MetS and has been shown to have a significant impact on the emergence of hypertension through several processes.[6] Intake of more sugar is closely connected to the emergence of IR. In several studies, anti-natriuretic impact of insulin (such as insulin-mediated increased salt absorption in the kidney) has been identified as a key mechanism behind the development of hypertension in MetS.[7]

Material and Methods: A 12-month Study was conducted in the of department of Biochemistry in collaboration with department of General Medicine a period from March 2024- February 2025 at Krishna Mohan Medical college & Hospital, Mathura. (U.P.).
Study design: Cross-sectional case control-based study.

Inclusion criteria:

- Men and women with diabetes who are 40-65 age.
- People with diabetes who have a history of the disease in their family.

Exclusion criteria:

- Patients with type 1 diabetes.
- Liver diseases outside fatty liver disease caused by alcohol use, such as autoimmune hepatitis, hemochromatosis, Wilson disease, and drug- induced liver disease.
- Problems with the kidneys.
- Other diseases (e.g. HIV, Zika virus infection, corona virus).
- Pregnant women.
- Lactating women.
- Cancers such as thyroid and pancreatic.

Clinical History:

- Personal information (name, age, place of study, administrative data, length of hospital stay)

- Make sure to include the last two generations of family members who have had diabetes mellitus.

Table 1: Age wise distribution of the patients

Age Groups	Group-1 (Diabetic)=200		Group-2 (as Control) =200	
	No. of participants	%	No. of participants	%
40-45 years	48	24	56	28
46-50	41	20	40	20
51-55	41	21	47	24
56-60	33	16	18	9
61-65	37	19	39	19

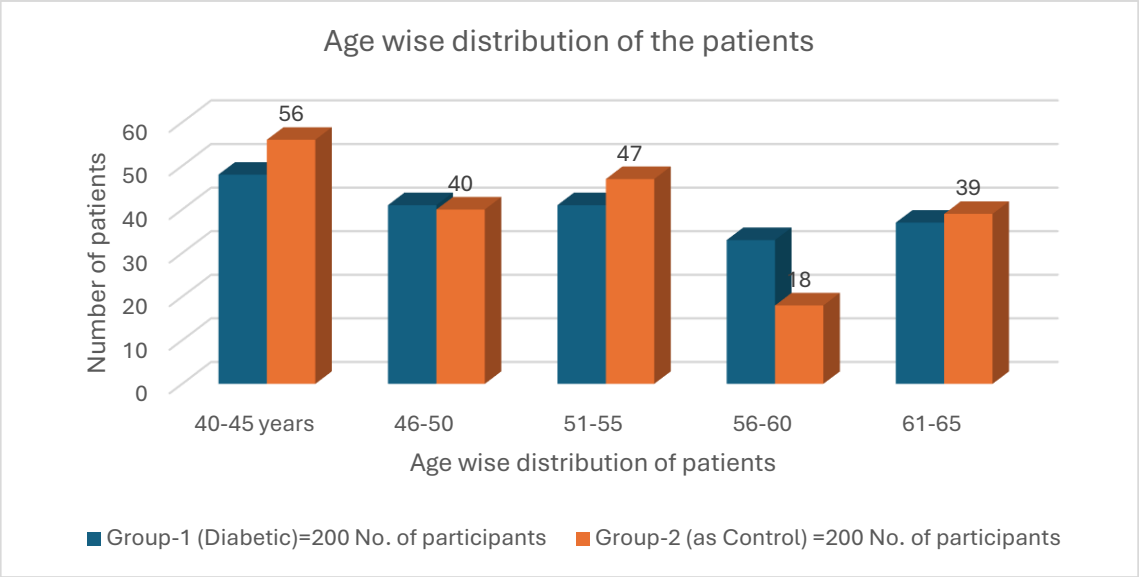

Fig 1: Graphical represents age wise distribution of the patients.

Table 2: table represents gender distribution of the patients

Gender	Group-1 (Diabetic)		Group-2 (as Control)	
	No of participants	Percentage (%)	No of participants	Percentage (%)
Male	109	55	104	52
Female	91	45	96	48
Total	200		200	

In the diabetic group, 109 (55%) participants were male and 91 (45%) were female.
In the control group, 104 (52%) participants were male and 96 (48%) were female.
The difference in gender distribution between the two groups was not statistically significant ($p = 0.52$), indicating that both groups were comparable with respect to gender.

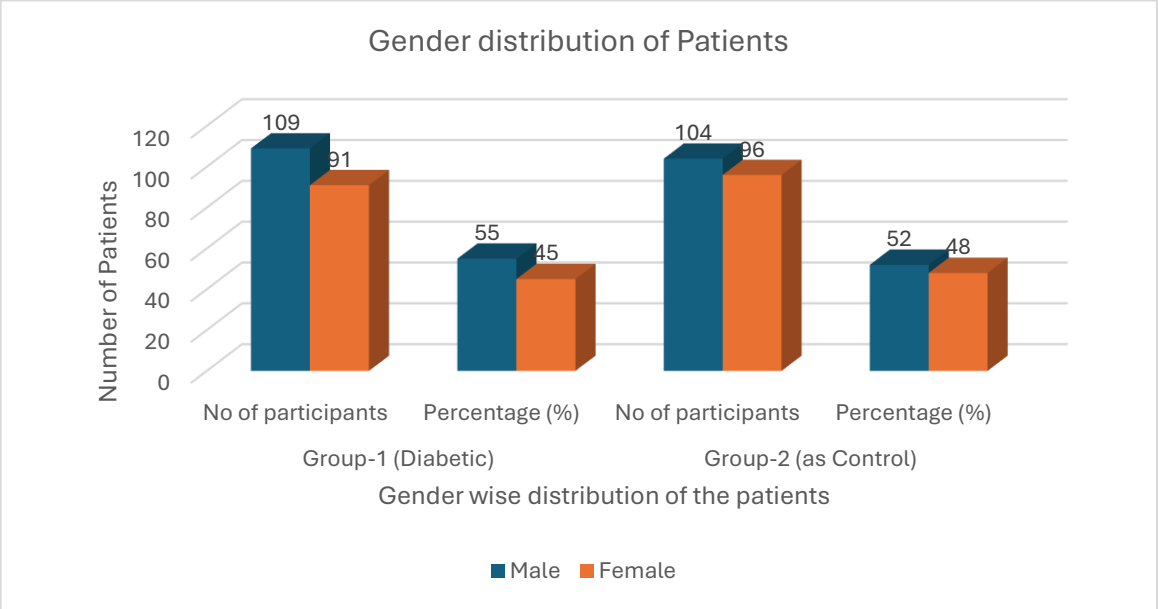

Fig 2: Graphical represents gender distribution of the patients.

Table 3: table represents mean age group of the patients

Age	Mean SD	p-Value
Study group	52.3 ± 7.1	0.33
Control group	51.5 ± 6.8	

The mean age of participants in the diabetic (study) group was 52.3 ± 7.1 years, while that of the control group was 51.5 ± 6.8 years. The difference in mean age between the two groups was not statistically significant (p = 0.33), indicating that both groups were age-matched and comparable in terms of age distribution.

Table 4: Comparison of Biochemical Parameter in study group vs control group.

Biochemical Parameters	Study Group (T2DM) (n = 200)	Control Group (n = 200)	p-value
Fasting Blood Glucose (mg/dL)	156.8±42.5	91.2±10.8	0.001
HbA1c (%)	8.4±1.5	5.3±0.6	0.001
HOMA-IR	4.8±1.9	1.9±0.7	0.001
Total Cholesterol (mg/dL)	211.3±38.4	179.6±31.2	0.001
Triglycerides (mg/dL)	189.7±56.1	128.2±41.5	0.001
HDL-C (mg/dL)	38.9±8.2	48.6±7.3	0.001
LDL-C (mg/dL)	126.5±32.9	108.7±26.5	0.002
ALT (U/L)	49.8±15.6	32.1±9.3	0.001
AST (U/L)	45.2±4.2	30.6±8.1	0.001
ALP (U/L)	122.6±31.8	98.7±26.2	0.016

The comparison of biochemical parameters between the study and control groups showed that Type 2 diabetic patients had significantly higher levels of fasting blood glucose, HbA1c, HOMA-IR, total cholesterol, triglycerides, LDL-C, and liver enzymes (ALT, AST, ALP), while HDL-C levels were significantly lower. These findings suggest that individuals with Type 2 Diabetes Mellitus exhibit poor glycemic control, pronounced insulin resistance, dyslipidemia, and liver dysfunction, highlighting the close interrelationship between glucose metabolism, lipid profile, and hepatic function in diabetes.

Table: 5: Correlation of HbA1c with Biochemical Parameters in T2DM Patients.

Biochemical Parameter	Correlation Coefficient (r)	p-value	Interpretation
Fasting Blood Glucose (mg/dL)	0.812	0.001	Strong positive correlation
HOMA-IR	0.758	0.001	Strong positive correlation
Total Cholesterol (mg/dL)	0.546	0.002	Moderate positive correlation
Triglycerides (mg/dL)	0.603	0.001	Moderate positive correlation
HDL-C (mg/dL)	0.421	0.005	Moderate negative correlation
LDL-C (mg/dL)	0.489	0.003	Moderate positive correlation
ALT (U/L)	0.367	0.012	Weak positive correlation
AST (U/L)	0.342	0.015	Weak positive correlation
ALP (U/L)	0.298	0.021	Weak positive correlation

HbA1c showed a strong positive correlation with fasting blood glucose and HOMA-IR, indicating that higher blood glucose levels and insulin resistance are closely associated with poor glycemic control in T2DM patients. A moderate positive correlation was observed with total cholesterol, triglycerides, and LDL-C, suggesting that dyslipidemia tends to worsen as HbA1c increases. Conversely, HDL-C exhibited a moderate negative correlation, reflecting reduced protective lipid levels in poorly controlled diabetes. Liver enzymes (ALT, AST, and ALP) showed weak but significant positive correlations with HbA1c, implying mild hepatic dysfunction related to chronic hyperglycemia**.**

Discussion

The study revealed that T2DM patients had significantly higher fasting glucose, HbA1c, HOMA-IR, total cholesterol, triglycerides, LDL-C, and liver enzyme levels, while HDL-C was significantly lower compared to controls.[8] These findings indicate poor glycemic control, insulin resistance, dyslipidemia, and liver dysfunction in T2DM patients.[9] The positive correlations of HbA1c with lipid and liver parameters highlight the close interrelationship between glucose metabolism, lipid abnormalities, and hepatic function, consistent with earlier studies.[10]

Conclusion

T2DM is associated with significant disturbances in glycemic, lipid, and liver function parameters. Routine evaluation of these markers is essential for early detection, prevention, and effective management of metabolic and hepatic complications in diabetic patients.

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