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Original Research Paper

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DYSLIPIDAEMIA AND ITS IMPACT IN PRE-DIABETIC AND DIABETIC POPULATIONS: AN ANALYSIS OF VARIOUS PARAMETERS

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KEYWORDS :

INTRODUCTION:

Prediabetes is characterized by elevated blood glucose levels that do not yet meet the criteria for diabetes. This condition is identified through indicators such as impaired fasting glucose (IFG), impaired glucose tolerance (IGT), or glycated hemoglobin (HbA1c) levels ranging from 5.7% to 6.4%. It represents a pre-morbid condition that heightens the likelihood of developing type 2 diabetes mellitus (T2DM), sharing similar underlying mechanisms of insulin resistance and/or impaired insulin secretion. Individuals diagnosed with prediabetes face a higher risk of microvascular complications, including retinopathy, nephropathy, and neuropathy, compared to those with normal glucose levels. Additionally, prediabetes is linked to an elevated risk of cardiovascular diseases.

Diabetic dyslipidemia refers to a specific set of lipoprotein abnormalities, which include elevated triglyceride levels, reduced high-density lipoprotein (HDL) cholesterol, and an increase in small, dense low-density lipoprotein (LDL) particles. This condition is prevalent in type 2 diabetes, affecting approximately 70% of patients. Diabetic dyslipidemia is a significant contributor to atherosclerotic cardiovascular disease (ASCVD), the leading cause of mortality in this population. The impact of cardiovascular disease (CVD), which is the leading cause of illness and death globally, is especially pronounced in individuals with type 2 diabetes mellitus (T2DM).[3-4] The share of CVD cases linked to diabetes is on the rise within the broader population.

As the incidence of diabetes continues to increase, particularly among ethnic minority groups, it becomes crucial to focus on reducing CVD risk in this demographic for public health reasons. While lifestyle changes and statin therapy are the primary strategies for mitigating CVD risk in diabetic patients, these individuals still face a significant likelihood of experiencing negative cardiovascular events.[5-8]

Aim:

The aim of this study is to analyse the relationship between metabolic biomarkers (fasting blood sugar, HbA1C, cholesterol levels, and creatinine) and lifestyle factors (smoking status, weight, and blood pressure) among pre-diabetic and diabetic individuals. We aim to identify key patterns that contribute to the development of metabolic disorders and CVD risks.

METHODS

Study Design: A retrospective cross-sectional analysis of clinical and laboratory data collected from a medical facility. Data from participants regarding blood sugar, lipid profiles, renal function, and demographic variables were included.

Data Collection: clinical assessments (blood sugar, lipid profiles, creatinine, BMI, etc.) and the measurement methods (biochemical methods, standard clinical devices).

Statistical Analysis: Descriptive statistics were used for summarizing data. T-tests, ANOVA, and multivariate regression were applied to assess the associations between metabolic variables and demographic/lifestyle factors.

RESULTS

- **Demographic Characteristics:**
- Gender Distribution: 34% male, 66% female.
- Age Range: 18–90 years, average 56 years (SD = 13.7).

- **Blood Glucose Levels:**
- Average fasting blood sugar (FBS): 139.9 mg/dL (SD = 26.4).
- Post-Lunch Blood Sugar (PLBS): 303.5 mg/dL (SD = 65.5).
- HbA1C: 7.2% (SD = 1.3).

- **Lipid Profiles:**
- Total Cholesterol: 197.8 mg/dL (SD = 42.2).
- Triglycerides: 286.6 mg/dL (SD = 89.4).
- HDL: 36.3 mg/dL (SD = 5.8).
- LDL: 102.7 mg/dL (SD = 48.3).

- **Renal Function:**
- Serum Creatinine: 0.91 mg/dL (SD = 0.25).
- Urine Microalbumin: 26.7 mg/dL (SD = 3.9).

- **Blood Pressure:**
- Systolic BP: 123.1 mmHg (SD = 7.6).
- Diastolic BP: 78.3 mmHg (SD = 6.1).

- **Smoking Status:**
- Smokers: 5%, Non-Smokers: 95%.

Demographic Characteristics

Table 1: Participant Demographics

Characteristic	N	Percentage (%)
Total Participants	245	100%
Male	83	34%
Female	162	66%
Mean Age (SD)	56 years	(13.7)
Age Range	18-90 years	

Comparative Analysis

Comparison of Lipid Parameters Between Pre-Diabetic and Diabetic Patients

Lipid Parameter	Diabetic Patients	Pre-Diabetic Patients	Normal Range (mg/dL)
Total Cholesterol (T CHOL)	217.6 ± 38.4	220.8 ± 42.6	<200
Triglycerides (TGL)	254.5 ± 78.9	260.0 ± 83.2	<150
HDL	40.05 ± 4.6	40.04 ± 4.5	>40 (men); >50 (women)
LDL	126.7 ± 48.1	128.8 ± 45.9	<130
VLDL	50.9 ± 10.3	52.0 ± 11.2	10–30

Comparison of Lipid Profiles Between Smokers and Non-Smokers

Lipid Parameter	Smokers	Non-Smokers	Normal Range (mg/dL)
Total Cholesterol (T CHOL)	224.9 ± 35.6	217.1 ± 39.4	<200
Triglycerides (TGL)	260.6 ± 80.4	254.4 ± 75.6	<150
HDL	40.74 ± 3.5	40.00 ± 3.6	>40 (men); >50 (women)
LDL	132.1 ± 41.7	126.2 ± 42.6	<130
VLDL	52.1 ± 9.2	50.9 ± 10.1	10–30

Statistical Analysis Summary

- **Correlation Analysis:** A correlation matrix (see supplementary data or appendix) revealed weak associations between FBS and PLBS (r = 0.02). There were no significant correlations between most metabolic parameters and age.

- **T-Tests (Gender Differences):**
- A significant difference was found in triglyceride levels between males and females (p = 0.036), with males having higher triglyceride levels.
- There were no significant gender differences for other parameters like FBS, HbA1C, LDL, or blood pressure.

DISCUSSION

Dyslipidemia is strongly associated with cardiovascular disease (CVD), especially in individuals with impaired glycemic control, such as pre-diabetes and diabetes. Numerous studies, including those by Goldberg et al. (2001) and Mooradian (2009),[9-10] have confirmed the characteristic lipid profile in diabetic dyslipidemia—elevated triglycerides, low HDL cholesterol, and small dense LDL particles. In this study, diabetic patients showed average triglyceride levels of 254.49 mg/dL and HDL levels of 40.05 mg/dL, aligning with these findings. Similarly, pre-diabetic individuals also exhibited elevated triglycerides (260.04 mg/dL) and low HDL levels (40.04 mg/dL), indicating the early onset of dyslipidemia, which resembles the diabetic lipid profile.

The study also highlights the importance of the Atherogenic Index of Plasma (AIP), which reflects the ratio of triglycerides to HDL and is a significant predictor of cardiovascular events. As Dobiasova et al. (2001)[11-12] demonstrated, AIP values above 0.24 are associated with increased atherosclerosis risk. In this study, AIP values for all participants were significantly above this threshold, placing the entire population at high CVD risk. Particularly concerning were the high AIP values among diabetic patients aged 60–70 years (around 0.80), largely due to elevated triglycerides (305.80 mg/dL) and low HDL (36.60 mg/dL). This combination significantly heightens the risk of atherosclerotic events in this age group.

Age further exacerbates dyslipidemia-related risks. Studies by Abbott et al. (1983) and Grundy et al. (2004)[13-14] show that the severity of dyslipidemia worsens with age, particularly in diabetic patients. Our data support this, as participants aged 60–70 years exhibited the highest triglyceride levels (305.80 mg/dL) and VLDL levels (61.16 mg/dL), indicating a greater risk for atherosclerosis in older populations.

Pre-diabetic individuals are also at a high risk of cardiovascular disease. Although their lipid abnormalities are generally less severe than those of diabetic patients, the similarity in triglyceride, LDL, and total cholesterol levels highlights the need for early intervention. As Grundy et al. (2005) [15] emphasized, insulin resistance in pre-diabetic individuals contributes to dyslipidemia, further justifying aggressive risk management in this population.

Managing cardiovascular risk in both pre-diabetic and diabetic patients is crucial. The high prevalence of abnormal lipid profiles (96.5% of participants) indicates an urgent need for intervention. As Collins et al. (2003) [16] recommended, lipid-lowering medications like statins, combined with lifestyle modifications, should be prioritized. Regular monitoring of triglycerides, LDL, and HDL levels is essential for managing dyslipidemia in these populations.

In addition, smoking, a known risk factor for CVD, worsens dyslipidemia. This study found that smokers had elevated total cholesterol, triglycerides, and LDL levels compared to non-smokers, which supports findings by Bakhru and Erlinger (2005) and Law et al. (1997) [17-18]. Given these results, smoking cessation should be a priority in risk-reduction strategies for diabetic and pre-diabetic populations.

In conclusion, both pre-diabetic and diabetic individuals are at elevated risk for cardiovascular events due to dyslipidemia. Early detection and management of lipid abnormalities, along with interventions targeting smoking cessation, are essential to reducing this risk.

CONCLUSION

Lipid disorders are modifiable risk factors commonly associated with DM and prediabetes, with a major impact on the early development of ASCVD. Their approach should be individualised according to patients' CV risk and comorbidities; this usually comprises a combination of lifestyle and pharmacologic interventions, while monitoring the LDLc values—the primary target—and non-HDLc and apoB as secondary (or co-primary) targets, alongside TG, HDLc and, if available, Lp(a). While in patients with DM the current guidelines' recommendations for LDLc lowering, while preventing CV events, include a high-intensity statin therapy wherever possible.

Patients with diabetes, particularly T2DM, often have dyslipidemia. Modern therapy of patients with diabetes demands that we aggressively treat lipids to reduce the high risk of ASCVD in this susceptible population and in those with very high TG to reduce the risk. Apart from the medical therapy, life style and dietary modification plays an important role. Future research should focus on lifestyle modifications and gender-specific interventions.

REFERENCES:

1. Wild S, Roglic G, Green A, Sicree R, King H. Global prevalence of diabetes: estimates for the year 2000 and projections for 2030. Diabetes Care 2004; 27:1047–1053.
2. Fox CS, Pencina MJ, Wilson PW, Paynter NP, Vasan RS, D'Agostino RB., Sr Lifetime risk of cardiovascular disease among individuals with and without diabetes stratified by obesity status in the Framingham heart study. Diabetes Care 2008; 31:1582–1584.
3. Kannel WB, McGee DL. Diabetes and cardiovascular disease. The Framingham study. Jama 1979; 241:2035–2038.
4. Fox CS, Coady S, Sorlie PD, D'Agostino RB, Sr, Pencina MJ, Vasan RS, et al. Increasing cardiovascular disease burden due to diabetes mellitus: the Framingham Heart Study. Circulation 2007; 115:1544–1550.
5. Geiss LS, Wang J, Cheng YJ, Thompson TJ, Barker L, Li Y, et al. Prevalence and incidence trends for diagnosed diabetes among adults aged 20 to 79 years, United States, 1980–2012. Jama 2014; 312:1218–1226.
6. Mooradian AD. Cardiovascular disease in type 2 diabetes mellitus: current management guidelines. Arch Intern Med 2003; 163:33–40.
7. Waters DD, Guyton JR, Herrington DM, McGowan MP, Wenger NK, Shear C, et al. Treating to New Targets (TNT) Study: does lowering low-density lipoprotein cholesterol levels below currently recommended guidelines yield incremental clinical benefit? Am J Cardiol 2004; 93:154–158.
8. Fitchett DH, Leiter LA, Goodman SG, Langer A. Lower is better: implications of the treating to new targets (TNT) study for Canadian patients. Can J Cardiol 2006; 22:835–839.
9. Goldberg, I. J. (2001). Diabetic Dyslipidemia: Causes and Consequences. The Journal of Clinical Endocrinology & Metabolism, 86(3), 965–971. DOI: 10.1210/jcem.86.3.7304
10. Mooradian, A. D. (2009). Dyslipidemia in type 2 diabetes mellitus. Nature Reviews Endocrinology, 5, 150–159. DOI: 10.1038/nrendo.2009.11
11. Dobiasova, M., & Frohlich, J. (2001). The Plasma Parameter log(TG/HDL-C) as an Atherogenic Index: Correlation with Lipoprotein Particle Size and Esterification Rate in apoB-Lipoprotein-Depleted Plasma (FERHDL). Clinical Biochemistry, 34(7), 583–588. DOI: 10.1016/S0009-9120(01)00263-6
12. Millan, J., Pintó, X., Muñoz, A., Zúñiga, M., Rubiés-Prat, J., Pallardo, L. F., ... & Masana, L. (2009). Lipoprotein ratios: Physiological significance and clinical usefulness in cardiovascular prevention. Vascular Health and Risk Management, 5, 757–765. DOI: 10.2147/VHRM.S6264

13. Abbott, R. D., Garrison, R. J., Wilson, P. W., Epstein, F. H., & Castelli, W. P. (1983). Joint distribution of lipoprotein cholesterol classes: the Framingham study. Arteriosclerosis, Thrombosis, and Vascular Biology, 3(3), 260–272. DOI: 10.1161/01.ATV.3.3.260
14. Grundy, S. M., Benjamin, I. J., Burke, G. L., Chait, A., Eckel, R. H., Howard, B. V., ... & Smith, S. C. (2004). Diabetes and cardiovascular disease: A statement for healthcare professionals from the American Heart Association. Circulation, 109(23), 2568–2572. DOI: 10.1161/01.CIR.0000133180.59184.2C
15. Grundy, S. M., Brewer, H. B., Cleeman, J. I., Smith, S. C., & Lenfant, C. (2005). Definition of metabolic syndrome: Report of the National Heart, Lung, and Blood Institute/American Heart Association conference on scientific issues related to definition. Circulation, 109(3), 433–438. DOI: 10.1161/01.CIR.0000111245.75752.C6
16. Collins, R., Armitage, J., Parish, S., Sleigh, P., & Peto, R. (2003). MRC/BHF Heart Protection Study of cholesterol lowering with simvastatin in 20,536 high-risk individuals: A randomised placebo-controlled trial. The Lancet, 361(9374), 2005–2016. DOI: 10.1016/S0140-6736(03)13637-3
17. Bakhru, A., & Erlinger, T. P. (2005). Smoking cessation and cardiovascular disease risk factors: Results from the Third National Health and Nutrition Examination Survey. PLoS Med, 2(6), e160. DOI: 10.1371/journal.pmed.0020160
18. Law, M. R., Wald, N. J., & Thompson, S. G. (1997). By how much and how quickly does reduction in serum cholesterol concentration lower risk of ischaemic heart disease? BMJ, 314(7075), 1383-1389. DOI: 10.1136/bmj.314.7075.1383