ORIGINAL ARTICLE

Atherogenic Indices in Newly Diagnosed Obese and Lean Patients of Type 2 Diabetes Mellitus: A Comparative Study

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Abstract

Background: Dyslipidemia is one of the driving forces in the pathogenesis of atherosclerosis and its resultant cardiovascular disease. Both these conditions are characterized by an increase in proatherogenic lipids compared to anti-atherogenic lipids. Atherogenic Indices have been developed to predict CVD risk without increasing the cost of testing; however, most of the studies done to date have used these indices in patients who have already suffered a coronary event. Dyslipidemia is most prevalent in cases of type 2 diabetes mellitus (T2DM). Therefore, this study was designed to assess atherogenic risk (via atherogenic indices) in newly diagnosed treatment-naïve obese and lean patients of T2DM.

Materials and methods: Treatment-naïve, newly diagnosed patients of T2DM were recruited and grouped into obese (BMI ≥ 25 kg/m^2) and lean (BMI <18.5 kg/m^2) groups. Blood was collected in a fasting state for the estimation of glycemic parameters and fasting lipid profile. Atherogenic indices (LDL-C/HDL-C, non-HDL-C, TC/HDL-C, atherogenic coefficient, lipoprotein combined index, and atherogenic index of plasma (AIP)) were calculated using predefined formulas.

Results: LDL-C/HDL-C, non-HDL-C, TC/HDL-C, atherogenic coefficient, lipoprotein combined index, and AIP were higher in the obese group compared to the lean group. However, these calculated indices were above the recommended cutoffs in both obese and lean patients with T2DM.

Conclusion: This study is the first to document increased atherogenic risk in both obese and lean patients (newly diagnosed) with T2DM. Although CVD risk is higher among the obese patients, aggressive control of plasma lipids is required in all patients with T2DM, irrespective of BMI.

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Introduction

Diabetes mellitus is defined as a group of metabolic diseases characterized by hyperglycemia, resulting from defects in insulin secretion and/or insulin action.[1] The incidence of type 2 diabetes mellitus (T2DM), a subtype of the disease, continues to rise exponentially throughout the globe, riding on the obesity wave.[2] An increase in adiposity can itself lead to derangement of fat metabolism and result in dyslipidemia. Dyslipidemia is also seen in patients with T2DM as a result of disturbance of glucose–insulin homeostasis. Atherosclerosis/cardiovascular disease (CVD), a sequela of dyslipidemia, contributes immensely to the disease morbidity and mortality. Dyslipidemia refers to an imbalance in a patient's fasting lipid profile, marked by low levels of high-density lipoprotein cholesterol (HDL-C) and elevated levels of low-density lipoprotein cholesterol (LDL-C) and triglycerides (TG).[3] Among these, LDL-C has been considered the primary factor in the development of atherosclerosis, often labeled as "bad cholesterol." However, it was noted that even after lowering LDL-C to the suggested levels, 50% remnant CVD risk remained. This forced scientists to rethink their approach and led to the development of numerous lipid ratios/atherogenic indices [LDL-C/HDL-C, non-HDL-C, TC/HDL-C, atherogenic coefficient (AC), lipoprotein combined index, and atherogenic index of plasma (AIP)] for the enhancement of the predictive power of the lipid profile without increasing the cost of testing.[4]

The LDL-C to HDL-C ratio representing the proatherogenic to antiatherogenic potential is a proven CVD risk marker with studies[5,6] documenting a higher level in patients with CVD compared to non-CVD patients. The ratio has also been shown to increase progressively with a decrease in the caliber of coronary vessels.[7] A ratio value greater than 2.517 is considered a sensitive predictor of CVD.

The TC-to-HDL-C ratio represents the ratio of total cholesterol to antiatherogenic cholesterol. It has been shown to correlate strongly with LDL particle number[8] and is documented to be a strong cardiovascular risk marker.[9–11] Non-HDL-C denotes the serum cholesterol of all the apoB-containing lipoproteins. High non-HDL-C may be responsible for the remnant CVD risk seen in individuals despite lowering LDL-C to the recommended ranges. It is calculated by deducting the HDL-C values from TC. Studies[12,13] have reported it to be a better CVD risk predictor in patients with obesity, T2DM, and metabolic disorders displaying atherogenic dyslipidemia.

Atherogenic coefficient or the non-HDL-C/HDL-C ratio denotes the balance between pro-atherogenic and anti-atherogenic lipoproteins and serves as an indicator of lipid dysregulation. It is said to be a better risk predictor for CVD compared to AC.[14] Lipoprotein combined index (LCI) is defined as the ratio of the product of total cholesterol, triglycerides, and LDL-C to HDL-C.[5] It, alongside AIP, i.e., log-transformed TG by HDL-C ratio, is considered to be an accurate marker for CVD risk prediction.[15,16]

Therefore, having compared the metabolic profile in newly diagnosed obese and lean patients of T2DM previously,[17] this study was done to further analyze the levels of nontraditional lipid ratios in newly diagnosed obese (BMI ≥25 kg/m^2) and lean (BMI <18.5 kg/m^2) patients of T2DM before starting antihyperglycemic agents. This is the first study to compare atherogenicity (as measured by atherogenic indexes) across these groups using the Asia-Pacific guidelines for BMI stratification.

Materials and Methods

The study was done in the department of Biochemistry, and patients were recruited from the outpatient department of the Department of Endocrinology of a tertiary care hospital in Delhi, India.

Ethical Aspects

Ethical clearance from the Institutional Ethics Committee-Human Research via diary

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number IEC-HR/2019/41/25. Written informed consent was obtained for all participants, and the study was conducted in lines of the Declaration of Helsinki.

Participant Selection

Newly diagnosed patients of T2DM (age group 20–65 years) were recruited. They were divided based on BMI (WHO Asia-Pacific Guidelines)[18] into obese (BMI ≥25 kg/m^2) and lean (BMI <18.5 kg/m^2). WHO criteria were used for the diagnosis of T2DM.[19] The following patients were excluded: Patients with renal, hepatic, or thyroid disorders, pregnant and lactating women, chronic alcoholics, and those suffering from severe comorbid illnesses. Patients on antihyperglycemic agents were also excluded from the study.

Participant Sampling and Routine Biochemical Testing

Peripheral blood was collected from all participants after an overnight fast of 12 hours. Blood collected in the plain vial was allowed to clot, following which it was centrifuged for 10 minutes at 4000 rpm. The serum thus obtained was used for the estimation of serum analytes (lipid profile and routine biochemical investigations). Blood collected in a fluoride vial was used for the estimation of glucose levels. HbA1c was estimated from blood collected in EDTA tubes on a BIORAD D-10 analyzer. Fasting lipid profile and blood sugar levels were measured by the enzymatic method on the RANDOX RX Imola AutoAnalyzer (RANDOX, UK).

Calculation of Lipid Indices

The following formulas were used for the calculation of lipid Indices:

- LDL-C/HDL-C = LDL-C (mg/dL)/HDL-C (mg/dL).
- TC/HDL-C = TC (mg/dL)/HDL-C (mg/dL).
- Non-HDL-C = TC (mg/dL) –HDL-C (mg/dL).
- Atherogenic Coefficient = Non-HDL-C (mg/dL)/HDL-C (mg/dL).
- Lipoprotein combined index = [TC (mg/dL) × TG (mg/dL) × LDL-C (mg/dL)]/HDL-C (mg/dL).
- AIP = log [TG (mg/dL)/HDL-C (mg/dL)].[20]

Statistical Analysis

SPSS v26.0 (IBM Corporation, USA) software was used to analyze the data. Following testing for normality, unpaired Student's t-test was used to compare parameters between the two groups. A *p*-value of less than 0.05 was considered to be statistically significant.

Results

Each group consisted of 22 male subjects and 8 female subjects. The mean age (years) was 52.1 ± 10.6 in the lean group and 51.5 ± 10.4 in the obese group. The mean BMI (kg/m^2) was 17.9 ± 0.8 in the lean group and 27.2 ± 2.7 in the obese group. Physical and biochemical characteristics of the groups are depicted in Table 1. LDL-C/HDL-C, non-HDL-C, TC/HDL-C, AC, LCI, and AIP ($p = 0.006$) were higher in the obese group compared to the lean group, as depicted in Table 2.

Discussion

Type 2 diabetes mellitus is characterized by hyperglycemia and tends to be associated with dyslipidemia. These metabolic alterations in the plasma serve as a primer for the formation of an atheroma and its sequelae. While the majority of the patients suffering from T2DM tend to be obese, lean patients can also develop the disease. This study was designed to compare the levels of atherogenicity (via atherogenic indexes) in newly diagnosed obese patients (BMI ≥25 kg/m^2) and lean (BMI <18.5 kg/m^2) and of T2DM.

Lipid transport in the body is mainly carried out by lipoproteins. The various types of lipoproteins are differentiated based on their cholesterol-to-triglyceride ratio and apolipoprotein present. A recent review article by Singh and Prabhakaran[21] has highlighted the atherogenic potential of apoB-containing lipoproteins compared to non-apoB-containing molecules. The various atherogenic indices used in this study are also designed to compare the proatherogenic and antiatherogenic potential of lipid molecules. Various atherogenic indices studied showed a similar trend when compared between the two study groups. LDL-C/HDL-C, non-HDL-C, TC/HDL-C, AC, LCI, and AIP were higher in the obese group compared to the lean group. However, only AIP was found to be statistically significant. AIP is the logarithmic transformation of the triglyceride-to-HDL-C ratio and is said to be

Table 1: Comparison of physical and biochemical parameters in obese and lean patients with T2DM

*Variables**	*Obese (n = 30)*	*Lean (n = 30)*	*p-value*
Waist circumference (cm)	100.1 (13.7)	76.7 (6.3)	–
Percentage body fat (%)	35.9 (5.7)	22.1 (5.9)	0.001***
Fasting plasma glucose (mg/dL)	207.28 (73.8)	254.3 (63.1)	0.01**
2-hour postprandial plasma glucose (mg/dL)	329.16 (88.1)	361.2 (76.6)	0.131
HbA1C (%)	9.42 (2.1)	11.5 (2.6)	0.001***
TC (mg/dL)	194.1 (35.4)	187.1 (35.2)	0.43
TG (mg/dL)	146.2 (53.7)	142.6 (66.4)	0.81
LDL-C (mg/dL)	137.5 (36.9)	124.3 (31.1)	0.13
HDL-C (mg/dL)	30.8 (7.5)	34.2 (11.1)	0.16

*values are expressed as mean (SD); **Significant; ***highly significant

Table 2: Comparison of atherogenic indices in obese and lean patients with T2DM

*Variables**	*Obese (n = 30)*	*Lean (n = 30)*	*p-value*	*Recommended cutoffs*
LDL-C/HDL-C	4.8 (2.1)	4.0 (1.6)	0.09	2.517
TC/HDL-C	6.7 (2.4)	5.9 (1.8)	0.14	3.5
Non-HDL-C	163.3 (37.5)	152.8 (34.8)	0.25	<130 mg/dL
Atherogenic coefficient (AC)	5.7 (2.4)	4.9 (1.8)	0.14	3.04
Lipoprotein combined index (LCI)	156253.4 (106258.8)	115817.9 (96340.8)	0.08	78830.7
AIP	0.7 (0.2)	0.6 (0.2)	0.006**	0.2

*values are expressed as mean (SD); **Significant; ***highly significant; values were above recommended levels in both groups

a very sensitive marker of atherogenicity. In addition, it has the following additional benefits compared to traditional indexes. Logarithmic transformation of AIP corrects for the lack of a normal Gaussian distribution and can also serve as an indirect surrogate marker of LDL-C particle size. An interesting finding was that the calculated indices were above the recommended cutoffs in both groups.

This is the first study to demonstrate an increased risk of CVD in both newly diagnosed lean and obese individuals with T2DM. The majority of studies (8–16) to date have utilized these indices to compare CVD risk between patients with established coronary events and control populations, consistently reporting higher levels in those with CVD. However, no previous studies have evaluated these indices specifically in newly diagnosed, treatment-naïve individuals with T2DM.

As previously reported (17), lean individuals with T2DM exhibited poorer glycemic control compared with age-matched obese counterparts, whereas dyslipidemia is more pronounced in the obese group than in lean individuals. This pattern suggests that excess adiposity in obese individuals may confer a greater CVD risk through adverse lipid profiles, potentially exerting a stronger influence than dysglycemia and its sequelae alone.

Nevertheless, as demonstrated in the present study, lean individuals with T2DM also carry a significant risk for CVD and therefore require equally vigilant, structured, and systematic follow-up.

Implications and Future Prospects

Our study demonstrates that obese patients with T2DM are at a higher cardiovascular risk compared to lean patients with T2DM. In addition, both groups were above the recommended threshold of certain lipid ratios, indicating that cardiovascular risk assessment is necessary for all patients with T2DM, irrespective of BMI. Therefore, there is a need to control blood lipid levels aggressively in patients with both lean and obese T2DM to decrease the cardiovascular risk associated with the disease. Future studies are needed to elucidate.

Limitations

A small sample size is a limitation of this study. In addition, matched healthy controls were not recruited.

Statement and Declarations

Source of Funding

None.

Conflict of Interest

None.

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