Original Research Article

Assessment of Cardiovascular Risk among Cigarette Smokers and Non-smokers using Atherogenic Indices: A Hospital Based Cross-Sectional study

Tatiparthi Satya Shika[1*], C Venkateshwarlu[2], Varsha Reddy[3], Raghuveer[4], Manjula[5], Anmol Manaswini[6]

[1,6]Post-Graduate, [2]Professor and Unit chief, [3]Assistant Professor, [4]Senior Resident, [5]Professor and Head, Department, General Medicine, Mallareddy Institute of Medical Sciences, Hyderabad, Telangana, India

[*]Corresponding author email: **satyashikareddy@gmail.com**

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Abstract

Smoking is positively related to lipid abnormalities. This is a hospital based cross sectional observational study conducted during August, 2019 to December, 2019 in cigarette smoking males attending OPD of General Medicine Department, Malla Reddy Institute of Medical Sciences, Hyderabad. Total of 480 subjects were included in the study, divided as two groups: cigarette smokers n=340 and non-smokers n=140. There was no significant difference between the mean values of age. Study showed significant increased levels of total cholesterol, LDL-C, TG and VLDL & significant low levels of HDL-C in the smokers. The mean of Atherogenic lipid ratios was significantly high among the cigarette smokers compared to the non-smokers, showing increased atherogenic risk among the smokers. Hence, Atherogenic indices seem to be a better predictor of atherogenic risk in comparison to old Conventional Lipid Parameters in smokers.

Key words

Atherogenic indices, Smokers, Non-Smokers, Conventional Lipid Parameters.

Introduction

Smoking is associated with altered serum lipid levels and is an independent risk factor for atherosclerosis. According to the World Health Organization (WHO), tobacco use causes 6 million deaths each year, with 80 percent of the world's 1 billion smokers living in low and middle income nations. In India, cigarette smoking has been acknowledged as a public health issue [1].

Studies have shown that smoking may cause abnormal plasma lipid pattern, with increased concentration of Total Cholesterol (TC), Triglycerides (TG), LDL Cholesterol (LDL-c), VLDL Cholesterol (VLDL-c) and decreased levels of HDL Cholesterol (HDL-c). Apart from reports of Dyslipidemia in Smokers, Normolipidemic smokers and Non-smokers may still be at risk of cardiovascular diseases, as their routine reports appear normal leading to erroneous rulings.

To better characterize and understand the atherogenic potential of conventional lipid profile, its parameters have been made into mathematical ratios like the Castelli's Risk Index I (CR-I), Castelli's Risk Index II (CR-II), Atherogenic Index of Plasma (AIP) [2 3], Atherogenic Coefficient (AC) [4], Cholindex (CI). These ratios have been used over the conventional lipid parameters in predicting the risk of Cardiovascular diseases in various clinical settings.

Aim and objective

Aimed to study the Atherogenic Indices among the Cigarette Smokers compared to Non-Smokers and to compare these indices with conventional lipid parameters to assess the Cardiovascular risk.

Materials and methods

It is a hospital based cross sectional observational study conducted during August, 2019 to December, 2019 in cigarette smoking males attending OPD General Medicine department of Malla Reddy Institute of Medical Sciences, Hyderabad. Four hundred and eighty (480) apparently healthy cigarette smokers and non-smokers aged between 21 to 60 years were included in the study after obtaining the informed consent.

Exclusion criteria: (i) Individuals with thyroid disorders, diabetes mellitus, CKD, cancer (ii) tobacco chewers, gutka chewers, (iii) individuals on lipid lowering medication.

A detailed history, lifestyle, Smoking habits, BMI, BP Recording, Random Blood Glucose level, Conventional Lipid Parameters were collected using a structured questionnaire, clinical examination, and blood sampling.

After an overnight fast, 5 ml of venous blood was drawn. This fasting venous blood sample of the patients was collected under all aseptic precaution into the appropriate plain tube. After a clot was allowed to retract, the blood was centrifuged at 3000 rpm for 15 minutes and serum is separated into another plain container for laboratory analysis of lipids. Atherogenic indices were calculated on Microsoft excel 2007. The atherogenic indices were calculated as follows: CR-I = TC/HDL-c , CR-II = LDL-c/HDL-c, AIP = log (TG/HDL-c) [2-4], AC = (TC-HDL-c)/HDL-c , CI = LDL-c – HDL-c (TG <400 mg/dl), LDL-c– HDL-c + 1/5 of TG (TG≥400 mg/dl).

Statistical analysis was done using t-test, using sophisticated SPSS version 23. p value less than 0.05 was considered as statistically significant.

Results

Table - 1, demonstrating the mean difference between the two groups smokers and non-smokers using t-test, p value <0.05 was considered significant, p value <0.001 was considered highly significant (HS). There was no significant difference in age between the two groups, the mean values of serum lipids (TC,

LDL-c, HDL-c, TG, VLDL-c) were significantly high in smokers than non-smokers.

Table - 2 demonstrating the mean difference of atherogenic lipid ratios (CR-I, CR-II, AIP, AC, CI) in smokers and non-smokers. The mean values of atherogenic indices were significantly higher (p value <0.001) in the smokers group than non-smokers.

Comparison of atherogenic indices of smokers and non-smokers is shown in **Figure - 1**, represented as bar charts.

Table - 1: showing t-test result of serum lipid profile in cigarette smokers and non-smokers.

	Smokers (Mean±SD, n=340)	**Non-Smokers (Mean±SD, n=140)**	**p-value**
AGE (Yrs)	46.1±11.0	45.38±12.0	NS
Cholesterol Total (mg/dL)	225.15±36.6	189.35±32.8	.001** HS
LDL-c (mg/dL)	149.5±25.7	127.0±28.9	.001** HS
HDL-c (mg/dL)	39.6±9.5	45.26±9.3	.001** HS
TG (mg/dL)	195.37±92.3	146.2±43.1	.001** HS
VLDL-c (mg/dL)	38.6±18.6	29.14±11.5	.001** HS
hsCRP (mg/L)	2.01±0.29	0.358±0.015	.001**HS

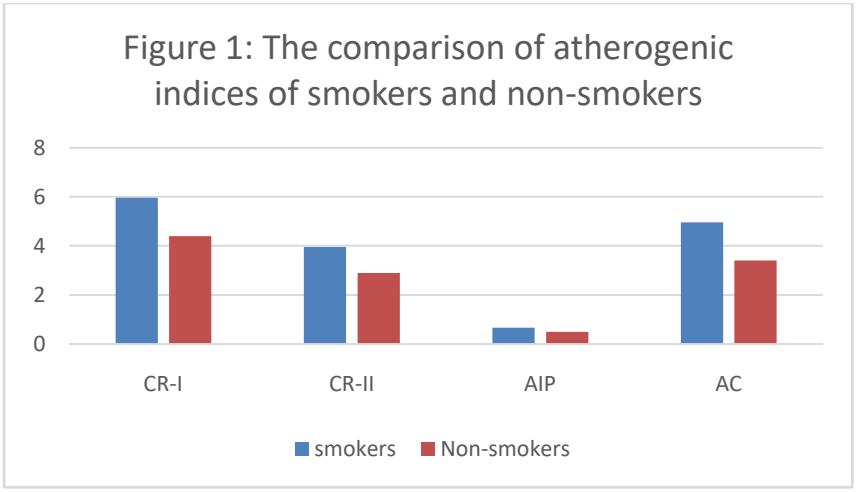
LDL-c: low density lipoprotein cholesterol, HDL-c: high density lipoprotein cholesterol, TG: triglyceride, VLDL-c: very low density lipoprotein cholesterol

Table - 2: Showing the mean difference of atherogenic lipid ratios in smokers and non-smokers.

	Smokers (Mean±SD, n=340)	**Non-Smokers (Mean±SD, n=140)**	**p-value**
CR-I	5.96±1.5	4.4±1.3	.001** HS
CR-II	3.96±1.0	2.9±1.0	.001** HS
AIP	0.66±.19	0.50±.18	.001** HS
AC	4.96±1.5	3.40±1.3	.001** HS
CI	109.95±25.9	81.73±32.4	.001** HS

Table demonstrating the mean difference between the two groups using t-test, *p value <.05, **p value <.001 HS (highly significant). CR-I: Castelli's Risk Index-I, CR-II: Castelli's Risk Index-II, AIP: Atherogenic index of plasma, AC: Atherogenic coefficient, CI: Cholindex.

Figure - 1: The comparison of atherogenic indices of smokers and non-smokers.



Discussion

Smoking is an important risk factor in the development of cardiovascular diseases (atherosclerosis, coronary artery disease and peripheral vascular disorders). Previous studies have established the role of deranged lipid profile in the progression of coronary artery disease.

Lipid abnormality was based on the expert panel of the National Cholesterol Education Programme (NCEP) cutoff values, TC, TGs, LDL-c, VLDL-c levels, respectively exceeding 200 mg/dl, 150 mg/dl, 100 mg/dl, 30 mg/dl and HDL levels below 45 mg/dl were considered abnormal [5]. There was significant difference observed in lipid levels of this study, the mean values of TC (225.15±36.6), HDL-c (39.6±9.5), LDL-c (149.5±25.7), TG (195.37±92.3), VLDL-c (38.6±18.6) in smokers were above the normal cutoffs and was enough to predict cardiovascular risk when compared to NCE cutoffs. Results of our study have showed increased TC, LDL-c, TG, VLDL-c and decreased level of HDL-c in smokers compared to non-smokers. Gepner, et al. [6] and Gamit, et al. [7] also observed higher levels of TC, LDL-c, VLDL-c, TG and lower levels of HDL-c in smokers in their study.

Previous research has shown that atherogenic indices can be used to predict cardiovascular risk in a variety of clinical contexts. The normal range of CAS1 is stated as ≤3.5 [4] and ≤3.0 for CAS2 [8] in the prediction of cardiovascular risk. Another study found that people with a CAS2 value of more than 5 have a higher risk of coronary events [9].

Gaziano, et al. proposed the TG/HDL ratio, which proved to be more accurate than CAS1 and CAS2 in predicting myocardial infarction [10]. The substantial correlation between TG/HDL and coronary heart disease risk supports a metabolic link between TG and cholesterol ester-rich lipoproteins in raising myocardial infarction risk, according to the scientists. The TGs/HDLc ratio was discovered to be a robust independent marker of severe coronary disease, according to da Luz, et al. [2] Noninvasive approaches were used by Bampi, et al. to determine the TG/HDL ratio as a determinant of coronary artery disease [11]. The logarithmic transformation of the TG/HDL ratio into AIP was later used to improve the significant prediction of atherosclerosis [12]. According to Bhardwaj, et al. [3] AC reflects the atherogenic potential of the full lipoprotein fraction range. This is because it is a CVD risk indicator since it measures cholesterol in LDL, VLDL, and IDL (intermediate-density lipoproteins) fractions in relation to anti-atherogenic HDLc.

A positive association was found between TC, LDLc, TGs, VLDLc, and atherogenic indices in the present study. HDLc, on the other hand, was found to be inversely associated to atherogenic indices. Our findings are consistent with those of Niroumand, et al. [13], who found a strong positive association of AIP with TC, TGs, and LDLc and an inverse correlation with HDLc in a cross sectional investigation of non-communicable disease risk factors surveillance. In individuals with coronary artery disease, Pusapati, et al. [14] discovered a negative relationship between HDLc and atherogenic indices.

The mechanism by which smoking increases atherosclerosis remains a mystery. There is evidence that atherosclerosis is an inflammatory disease [15] caused by oxidized LDLc's harmful effects on the vascular endothelium [16]. Nicotine has been shown to raise circulating TGs, LDLc, TC, and VLDLc levels while lowering HDLc levels [17]. Nicotine alters plasma lipids through a variety of mechanisms, include: sympathetic nervous system stimulation, resulting in increased catecholamine synthesis, and cholinergic receptors in the brain, autonomic ganglia are stimulated. Nicotine binds to these receptors, causing the sodium calcium channel to open, allowing sodium and calcium to enter, and neurotransmitters [18] to be released.

Lipolysis and the transfer of free fatty acids to the liver are upregulated by catecholamine and other neurotransmitters. Hepatic reesterification of these acids occurs, with an increase in the production of liver free fatty acids, TGs, and VLDLc into the bloodstream [19]. Free radicals typically oxidise LDL particles and their associated substances stuck in the endothelium. Inflammatory responses in the endothelium are generated in response to oxidised LDL particles, resulting in the recruitment of monocytic white blood cells into the artery wall, which eventually change into macrophages.

In the absence of HDL clearance of lipids from these specialised cells, the absorption of oxidised LDL particles by tissue macrophages activates a cascade of immunological responses that leads to the creation of foam cells and the subsequent production of an atheroma. Free radicals typically oxidise LDL particles and their associated substances stuck in the endothelium. Inflammatory responses in the endothelium are generated in response to oxidised LDL particles, resulting in the recruitment of monocytic white blood cells into the artery wall, which eventually change into macrophages.[17] The ingestion of oxidized LDL particles by tissue macrophages activates a cascade of immunological responses that leads to the creation of foam cells and the subsequent production of an atheroma, In the absence of HDL clearance of lipids from these specialised cells [17, 19]. Because of its involvement in removing fats from arterial walls, HDL is referred to as "good cholesterol." It transfers cholesterol and TGs out of arterial walls, lowers macrophage buildup, prevents and regresses atherosclerosis, and so protects cardiovascular disease over time.

Conclusion

Smoking is positively related to lipid abnormalities. Atherogenic indices appear to be stronger predictors of cardiovascular risk, particularly when the absolute values of the lipid profile appear normal or not significantly disturbed. These atherogenic indices as a tools/test can be used to identify individuals with risk of cardiovascular events in future due to atherogenesis.

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