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A Study of Lipid Profile in Smokers and Tobacco Chewers and their Correlation with Coronary Artery Disease (CAD) in Intensive Care Unit

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Abstract
Introduction: *Smoking and tobacco chewing forms a major killer of this era. It changes lipid profile which increases risk of coronary heart disease. Smoking and tobacco chewing is associated with an increased risk of atherosclerotic vascular disease. ABI is reliable indicator of high coronary risk and is significantly related to disease of coronary artery disease.*
Aim: *The aim of study is to study lipid profile in smokers and tobacco chewers and the correlation of their habit with coronary artery disease.*
Material and Method: *Total 125 individuals who admitted in intensive care unit at Mallareddy hospital from 2018-2019 were taken for the study out of which 100 were patients and 25 were control. The study was conducted in 5 groups of both sex from age (20-70 years) with BMI<27.each group contain 25 subjects. The serum lipid profile level were calculated after overnight fasting and ankle brachial index was calculated and correlation of each group with ABI with CAD grading was done.*
Result: *smokers and tobacco chewers have dyslipidemic changes and increase incidence of CAD than non-smokers. As the no of years of habit increased the clinical and CAD grading of ABI increased.*
Conclusion: *Both tobacco chewers and smokers (cigarette and bidi smokers) are at increased risk of coronary artery disease as compared to non-tobacco consuming and non-smoking population. Our research indicated that ABI could be a useful method in assessing both the atherosclerotic risk factors and the degree of coronary involvement.*
Keywords: *lipid profile, CAD, atherosclerosis, ABI, coronary artery disease, smoking.*

Introduction

Smoking and tobacco chewing forms a major killer of this era which brings ill effects in the form of cardiovascular accidents, chronic obstructive pulmonary diseases, cerebrovascular accidents, peripheral vascular diseases and cancers of lungs, kidney, bladder & GIT. It changes lipid profile which increases risk of coronary heart disease.

Smoking and tobacco chewing adversely affects the cardiovascular system in human subjects. Smoking and tobacco chewing is associated with an increased risk of atherosclerotic vascular disease, hypertension, myocardial infarction,

unstable angina, sudden cardiac death, and stroke. The adverse effects of smoking on vascular function have been examined in human subjects so many studies have shown that acute and chronic cigarette smoking impairs nitric oxide synthase mediated relaxation of large blood vessels. Although smoking has been established as an independent risk factor [1] for coronary heart disease the mechanism by which it increases the risk of coronary heart disease. And also modifiable ones like hypertension, dyslipidemia, diabetes mellitus, changing lifestyle and non-modifiable ones like age and sex and also having the risk factors for coronary heart disease.[2]

Explanations for cigarette smoking affecting coronary circulation have been postulated as: It affects both the coronary and the peripheral circulation. Because cigarette smoke contains a large number of oxidants, it has been hypothesized and the adverse effects of smoking may result from oxidative damage to vascular endothelium. The nicotine absorbed from cigarette smoke may induce cardiac arrhythmias through its pharmacologic action. The increased carbon monoxide in the blood of cigarette smokers may damage the endothelium and accelerate the entry of cholesterol into the wall of the artery promoting the development of atherosclerosis, thrombosis. The formation of carboxyhemoglobin creates relative anoxia in the tissue, including in the myocardium, Smoking enhances the platelet aggregation.[2,3] An additional mechanism has been recently suggested that smoking adversely affects the concentration of the plasma lipids and lipoproteins.

Tobacco use in form of smoking or smokeless tobacco is a major preventable cause of coronary artery disease and premature death. Nicotine is the principal component of tobacco use. Nicotinic stimulation of adrenergic drive raise both B.P and myocardial oxygen demand, lipid metabolism with fall in protective high-density lipoprotein. Active tobacco consumption alters the total serum cholesterol and lipoprotein composition, which directly increase risk of coronary heart disease.[4]

Nicotine stimulates the release of adrenaline by adrenal cortex leads to increase serum concentration of free fatty acids.[5] Free fatty acids are well known stimulants of hepatic secretion of VLDL and triglycerides. HDL concentration is inversely proportional to VLDL in serum. Free fatty acid also stimulate hepatic secretion of cholesterol.[6] Tobacco users have increased C-reactive protein in blood which play important role in pathogenesis of cardiovascular disease.[7] Carbon monoxide or aromatic hydrocarbons toxicity induce damage to vessel wall or enhance probability of coronary thrombosis by hypercoagulablestate.[8]

ABI is reliable indicator of high coronary risk and is significantly related to disease of coronary artery disease. It is a simple and non-invasive tool with high specificity and severity for diagnosis of peripheral artery disease. The ankle-brachial index (ABI), calculated by dividing the higher systolic blood pressure of each ankle artery by the higher systolic blood pressure of the upper limbs,[9] It is a simple and non-invasive tool with high specificity and sensitivity for the diagnosis of PAD.[10]

Pathophysiology of cigarette and tobacco smoking leading to CAD: Tobacco chewing is a unique habit practiced in Indian subcontinent and is consumed in the form of pan, gutka, mawa, khaini, mainpuri, and so on. Because of its easy availability, tobacco chewing is rapidly increasing and affecting all age groups and genders and has become a major public and social health concern.[11] Nicotine is one of the important substances present in tobacco and has direct toxic effects on the cardiovascular system.[12] smokeless tobacco consistently produces smokeless tobacco produce nicotine and cause sympathetic neural stimulation and cardiovascular effects. Nicotine stimulate secretion of catecholamine activating adenyl cyclase of adipose tissue results in increased lipolysis with increased concentration of plasma free fatty acids and increased concentration of hepatic triglycerides and VLDL into blood stream.[13]

Conventionally, cigarette smoke is divided into two phases: a tar phase and a gas phase: The tar or particulate phase is defined as the material that is trapped when the smoke stream is passed through the Cambridge glass-fiber filter that retains 99.9% of all particulate material with a size 0.1 µm.[14] The gas phase is the material that passes through the filter.

The particulate (tar) phase of cigarette smoke contains 10^{17} free radicals/g, and the gas phase contains 10^{15} free radicals/puff.[14] The radicals associated with the tar phase are long-lived (hours to months), whereas the radicals associated with the gas phase have a shorter life span (seconds).[14,15,16] Cigarette smoke that is drawn through the tobacco into an active smoker's mouth is known as mainstream smoke. cigarette smoke is the smoke emitted from the burning ends of a cigarette. Mainstream cigarette smoke comprises 8% of tar and 92% of gaseous components.[14] Nicotine, a component of the tar phase, is the addictive substance of cigarette smoke.[17] Cigarette smoking predisposes the individual to several different clinical atherosclerotic syndromes, including stable angina, acute coronary syndromes, sudden death, and stroke. Aortic and peripheral atherosclerosis are also increased, leading to intermittent claudication and abdominal aortic aneurysms.[18] When we look at the various types of lipoproteins, it is the level of low- density lipoprotein (LDL) cholesterol that is most directly associated with coronary heart disease (CHD). Although very-low-density lipoprotein (VLDL) has also been shown to be associated with premature atherosclerosis peripheral vascular disease, high-density lipoprotein (HDL) cholesterol is protective against the development of CHD.[19]

Aim

The aim of study is to study lipid profile in smokers and tobacco chewers and the correlation of their habit with coronary artery disease.

Objectives of Study

1. To evaluate and compare the lipid profile in both smokers and non- smokers.
2. To evaluate the existence of dose dependent relationship and durational significance between smoking and lipid profile among smoker.
3. To know if smoking has dyslipidemic changes in young smokers.

Material and Methods

Source of Data

Patients admitted in Intensive Care Unit of Mallareddy Medical College, Hospital & Research Center, Hyderabad, Telangana. Patients who satisfied the below criteria were taken as cases and equal number of controls taken with respect to age and sex. Data collected from October 2018 to November 2019. A written informed consent taken from each patient enrolled in this study. Cross sectional comparative study. The information regarding all the selected cases and controls were recorded in a master chart. Then the tobacco chewers and smokers compared with the controls using t-test.

Sample Size Calculation

Sample size calculation revealed that 16 patients per group will be required to detect a difference of 30 units in mean value of total cholesterol between any two groups, at an alpha of 0.05 with power of 80%.P values < 0.05 were considered to indicate statistical significance. Hence, we intend to take more than 16 patients per group.

Investigations Required for the Study

After overnight fasting following laboratory investigations are done in all subjects: Serum total cholesterol, Serum high density lipoprotein(HDL), Serum low density lipoprotein(LDL), Serum very low density lipoprotein(VLDL), Serum triglyceride (TGL), Fasting blood sugar(FBS), Serum Creatinine, Serumurea, Liver functiontest, ECG, ECHO, Chest X-ray.

Inclusion Criteria

1. According to history subjects were divided into 4groups
    a. Group-1 Non- smoke tobaccousers
    b. Group-2 (Mild smokers): 1-10 cigarettes or 1-15 beedis / day for at least 2 years' ormore.
    c. Group-3 (Moderate smokers): 11-20 cigarettes or 16- 30beedis
        i. / day for at least 2 years' or more.
    d. Group-4 (Heavy smokers): more than 20 cigarettes or 30beedis
        i. / day for at least 5 years' or more.
    e. Group-5 (Control) subjects who have never smoked.
2. The subjects are chosen in age groups of 20 – 70 yrs. Of age
3. The subject's BMI are less than27

Exclusion Criteria

Subjects having diseases mentioned below known to influence blood lipids and smoking induced respiratory illness are excluded from the study: Diabetes mellitus, Nephrotic syndrome, Hypothyroidism, Hypertension, COPD, Asthma, pneumonia, Bronchogenic Carcinoma, Chronic kidney disease, Obese of BMI>27.

Methodology

Total 125 individuals taken for the study out of which 100 patients and 25 are control. the study was conducted in 5 group of subjects of both sex from age (20-70 years) with BMI<27. Each group contain 25 subjects. The subjects with Diabetes, hypertension, renal disease, thyroid illness, asthma chronic obstructive pulmonary disease with lipid metabolism disorders were excluded from study. The blood samples were collected after an overnight fasting of 10 hours (10 pm to 8 am), 5ml of whole blood was collected from each subject and serum was seprated. The serum lipid profile level were calculated. Estimation of total cholesterol by Zak method. Estimation of triglycerides by HANTZ-SCH condensation reaction, estimation of HDL cholesterol, LDL, VLDL and chylomicrons were precipitated by polyanions in presence of metal ions to (phosphotung –state /Mg) to leave HDL in solution. The complete lipid profile measures the serum total cholesterol, HDL and triglycerides. LDL and VLDL calculated by using Friedewald's formula provided that triglycerides were below400mg/dl. VLDL cholesterol=triglyceride/5

Observation and Results

Table No. 1 Comparison of age in various groups

Group	Mean ± SD	F Value	P Value
Chewer	52.16 ± 14.24	1.77	0.138, NS
Control	45.84 ± 9.30		
Mild smoker	51.04 ± 11.21		
Moderate smoker	54.08 ± 11.23		
Severe smoker	49.32 ± 12.04		

One-Way ANOVA applied. P value < 0.05 was taken as statistically significant

The table 1 shows the mean age comparison in relation to the groups. The mean age in the chewer group was 52.16 ± 14.24 years, in the control group it was 45.84 ± 9.30 years, in the mild smoker group it was 51.04 ± 11.21 years, in moderate smoker group it was 54.08 ± 11.23 years and in the severe smoker group it was 49.32 ± 12.04 years. The difference between the groups was found to be statistically not significant (P>0.05), showing that the age was comparable between the groups.

Table No. 2 Association of gender with groups

Gender	Groups					Total
	Chewer	Control	Mild smoker	Moderate smoker	Severe smoker	
Female	8 32.0%	5 20.0%	3 12.0%	4 16.0%	0 0.0%	20 16.0%
Male	17 68.0%	20 80.0%	22 88.0%	21 84.0%	25 100.0%	105 84.0%
Total	25 100.0%	25 100.0%	25 100.0%	25 100.0%	25 100.0%	125 100.0%

Pearson Chi-Square = 10.119, DF = 4, P-Value = 0.038, Significant

The table 2 shows the association of gender with the groups. There was a significant difference in the distribution of males and females in relation to the groups (P<0.05), with a higher proportion of males in each group in comparison to the females.

Table No. 3 Comparison of habit years in various groups

Group	Mean ± SD	F Value	P Value
Chewer	22.28 ± 8.52	0.79	0.502, NS
Mild smoker	21.68 ± 11.87		
Moderate smoker	25.60 ± 12.38		
Severe smoker	21.16 ± 11.78		

One-Way ANOVA applied. P value < 0.05 was taken as statistically significant

The table 3 shows the mean habit years comparison in relation to the groups. The mean habit years in the chewer group was 22.28 ± 8.52 years, in the mild smoker group it was 21.68 ± 11.87 years, in moderate smoker group it was 25.60±12.38 years and in the severe smoker group it was 21.16± 11.78 years. The difference between the groups was found to be statistically not significant (P>0.05), showing that the habit years was comparable between the groups.

Table No. 4 Comparison of mean cholesterol in various groups

Group	Mean ± SD	F Value	P Value
Chewer	205.53 ± 25.28	22.81	0.000*
Control	161.21 ± 16.09		
Mild smoker	198.08 ± 19.39		
Moderate smoker	207.17 ± 27.58		
Severe smoker	213.37 ± 18.16		

One-Way ANOVA applied. P value < 0.05 was taken as statistically significant

The table 4 shows the mean cholesterol comparison in relation to the groups. The mean cholesterol in the chewer group was 205.53 ± 25.28 mg/dL, in the control group it was 161.21 ± 16.09 mg/dL, in the mild smoker group it was 198.08 ± 19.39 mg/dL, in moderate smoker group it was 207.17 ± 27.58 mg/dL and in the severe smoker group it was 213.37 ± 18.16 mg/dL. The difference between the groups was found to be statistically significant (P<0.05), showing that the total cholesterol is varying in different groups.

Table No. 5 Comparison of mean HDL in various groups

Group	Mean ± SD	F Value	P Value
Chewer	32.58 ± 4.75	41.33	0.000*
Control	51.75 ± 6.64		
Mild smoker	46.85 ± 4.54		
Moderate smoker	43.91 ± 5.40		
Severe smoker	43.24 ± 5.80		

One-Way ANOVA applied. P value < 0.05 was taken as statistically significant

The table 5 shows the mean HDL comparison in relation to the groups. The mean HDL in the chewer group was 32.58 ± 4.75 mg/dL, in the control group it was 51.75 ± 6.64 mg/dL, in the mild smoker group it was 46.85 ± 4.54 mg/dL, in moderate smoker group it was 43.91 ± 5.40 mg/dL and in the severe smoker group it was 43.24 ± 5.80 mg/dL. The difference between the groups was found to be statistically significant (P<0.05), showing that the HDL is varying in different groups.

Table No. 6 Comparison of mean LDL in various groups

Group	Mean ± SD	F Value	P Value
Chewer	154.34 ± 20.25	33.16	0.000*
Control	84.73 ± 13.79		
Mild smoker	115.56 ± 19.92		
Moderate smoker	128.77 ± 29.96		
Severe smoker	130.13 ± 23.29		

One-Way ANOVA applied. P value < 0.05 was taken as statistically significant

The table 6 shows the mean LDL comparison in relation to the groups. The mean LDL in the chewer group was 154.34 ± 20.25 mg/dL, in the control group it was 84.73 ± 13.79 mg/dL, in the mild smoker group it was 115.56 ± 19.92 mg/dL, in moderate smoker group it was 128.77 ± 29.96 mg/dL and in the severe smoker group it was 130.13 ± 23.29 mg/dL. The difference between the groups was found to be statistically significant (P<0.05), showing that the LDL is varying in different groups.

Table No. 7 Comparison of mean triglycerides in various groups

Group	Mean ± SD	F Value	P Value
Control	113.01 ± 8.04	39.29	0.000*
Chewer	117.48 ± 37.40		
Mild	172.20+21.76		
Moderate	178.41+31.50		
Severe smoker	183.87 ± 30.18		

One-Way ANOVA applied. P value < 0.05 was taken as statistically significant

The table 7 shows the mean triglycerides comparison in relation to the groups. The mean triglycerides in the control group was 113.01 ± 8.04 mg/dL, in the chewer group it was 117.48 ± 37.40 mg/dL, in the moderate smoker group it was 178.41 ± 31.50 mg/dL, in mild smoker group it was 172.20 ± 21.76 mg/dL and in the severe smoker group it was 183.87 ± 30.18 mg/dL. The difference between the groups was found to be statistically significant (P<0.05), showing that triglycerides is varying in different groups.

Table No. 8 Comparison of mean VLDL in various groups

Group	Mean ± SD	F Value	P Value
Control	22.58 ± 1.64	20.04	0.000*
Chewer	24.62 ± 6.32		
Mild smoker	34.25 ± 3.98		
Moderate smoker	38.48+16.44		
Severe smoker	38.525.80		

One-way ANOVA applied. P value < 0.05 was taken as statistically significant

The table 8 shows the mean VLDL comparison in relation to the groups. The mean VLDL in the control group was 22.58 ± 1.64 mg/dL, in the chewer group it was 24.62 ± 6.32 mg/dL, in the moderate smoker group it was 38.48 ± 16.44 mg/dL, in mild smoker group it was 34.25 ± 3.98 mg/dL and in the severe smoker group it was 38.52 ± 5.80 mg/dL. The difference between the groups was found to be statistically significant (P<0.05), showing that VLDL is varying in different groups.

Table No. 9 Comparison of mean CPKMB in various groups

Group	Mean ± SD	F Value	P Value
Chewer	63.25 ± 33.56	10.37	0.000*
Control	16.24 ± 4.69		
Mild smoker	64.99 ± 33.57		
Moderate smoker	74.8 ± 77.30		
Severe smoker	108.20 ± 69.50		

One-Way ANOVA applied. P value < 0.05 was taken as statistically significant

The table 9 shows the mean CPKMB comparison in relation to the groups. The mean CPKMB in the chewer group was 63.25 ± 33.56 IU/L, in the control group it was 16.24 ± 4.69 IU/L, in the mild smoker group it was 64.99 ± 33.57 IU/L, in moderate smoker group it was 74.8 ± 77.30 IU/L and in the severe smoker group it was 108.20 ± 69.50 IU/L. The difference between the groups was found to be statistically significant (P<0.05), showing that CPKMB is varying in different groups.

Table No. 10 Association of Trop I with groups

Trop I	Groups					Total
	Chewer	Control	Mild smoker	Moderate smoker	Severe smoker	
Negative	16 64.0%	25 100.0%	16 64.0%	14 56.0%	10 40.0%	81 64.8%
Positive	9 36.0%	0 0.0%	9 36.0%	11 44.0%	15 60.0%	44 35.2%
Total	25 100.0%	25 100.0%	25 100.0%	25 100.0%	25 100.0%	125 100.0%

Pearson Chi-Square = 21.184, DF = 4, P-Value = 0.000, Highly significant

The table 10 shows the association of Trop I with the groups. There was a significant difference in the distribution of Trop I positive and negative patients in relation to the groups (P<0.05), with a higher number of chewers, controls and mild smokers having negative Trop I while more number of patients with Trop I positive were seen in the moderate and severe smokers.

Table No. 11 Association of ECG Changes with groups

ECG Changes	Groups					Total
	Chewer	Control	Mild smoker	Moderate smoker	Severe smoker	
N	0 0.0%	25 100.0%	0 0.0%	0 0.0%	0 0.0%	25 20.0%
NSTEMI	7 28.0%	0 0.0%	3 12.0%	1 4.0%	4 16.0%	15 12.0%
STEMI	2 8.0%	0 0.0%	6 24.0%	8 32.0%	12 48.0%	28 22.4%
UA	16 64.0%	0 0.0%	16 64.0%	16 64.0%	9 36.0%	57 45.6%
Total	25 100.0%	25 100.0%	25 100.0%	25 100.0%	25 100.0%	125 100.0%

Pearson Chi-Square = 143.759, DF = 12, P-Value = 0.000, Highly significant

The table 11 shows the association of ECG changes with the groups. There was a significant association between the ECG changes with the groups ($P<0.05$), showing that ECG changes were seen more in the groups other than control and also the ECG changes were varying in groups.

Table No. 12 Association of Wall Code with groups

Wall Code	Groups					Total
	Chewer	Control	Mild smoker	Moderate smoker	Severe smoker	
Anterior	7 28.0%	0 0.0%	9 36.0%	9 36.0%	7 28.0%	32 25.6%
Inferior	7 28.0%	0 0.0%	8 32.0%	8 32.0%	6 24.0%	29 23.2%
Septum	0 0.0%	0 0.0%	0 0.0%	1 4.0%	0 0.0%	1 0.8%
Posterior	0 0.0%	0 0.0%	0 0.0%	1 4.0%	0 0.0%	1 0.8%
NO RWMA	6 24.0%	25 100.0%	4 16.0%	3 12.0%	3 12.0%	41 32.8%
Global wall	5 20.0%	0 0.0%	4 16.0%	3 12.0%	9 36.0%	21 16.8%
Total	25 100.0%	25 100.0%	25 100.0%	25 100.0%	25 100.0%	125 100.0%

Pearson Chi-Square = 78.296, DF = 20, P value = 0.0001, Highly significant

The table 12 shows the association of wall code with the groups. There was a significant association between the wall code with the groups ($P<0.05$), showing that groups are dependent on the wall code.

Table No. 13 Association of Cardiomegaly with groups

Cardio- megaly	Groups					Total
	Chewer	Control	Mild smoker	Moderate smoker	Severe smoker	
Absent	10 40.0%	25 100.0%	17 68.0%	13 52.0%	9 36.0%	74 59.2%
Present	15 60.0%	0 0.0%	8 32.0%	12 48.0%	16 64.0%	51 40.8%
Total	25 100.0%	25 100.0%	25 100.0%	25 100.0%	25 100.0%	125 100.0%

Pearson Chi-Square = 27.954, DF = 4, P-Value = 0.000, Highly significant

The table 13 shows the association between cardiomegaly and the groups. In patients how had cardiomegaly, 15 (60.0%) belonged to the chewer group, 8 (32.0%) belonged to the mild smoker's group, 12 (48.0%) belonged to the moderate smoker's group and 16 (64.0%) belonged to the severe smoker's group. There was a statistically significant association seen between Cardiomegaly and the groups (P<0.05) showing that groups are dependent on the presence/ absence of cardiomegaly.

Table No. 14 Comparison of mean ABI in various groups

Group	Mean ± SD	F Value	P Value
Chewer	0.67 ± 0.25	17.57	0.000*
Control	0.99 ± 0.14		
Mild smoker	0.72 ± 0.19		
Moderate smoker	0.63 ± 0.19		
Severe smoker	0.58 ± 0.17		

One-Way ANOVA applied. P value < 0.05 was taken as statistically significant

The table 14 shows the mean ABI comparison in relation to the groups. The mean ABI in the chewer group was 0.67 ± 0.25, in the control group it was 0.99 ± 0.14, in the mild smoker group it was 0.72 ± 0.19, in moderate smoker group it was 0.63 ± 0.19 and in the severe smoker group it was 0.58 ± 0.17. The difference between the groups was found to be statistically significant (P<0.05), showing that ABI is varying in different groups.

Table No. 15 Association of ABI value with Trop I

Trop I	ABI				Total
	1	2	3	4	
Negative	27 93.1%	24 70.6%	25 51.0%	5 38.5%	81 64.8%
Positive	2 6.9%	10 29.4%	24 48.9%	8 61.5%	44 35.2%
Total	29 100.0%	34 100.0%	49 100.0%	13 100.0%	125 100.0%

Pearson Chi-Square = 18.717, DF = 3, P-Value = 0.000, Highly significant

The table 15 shows the association between ABI value and Trop I. There was a statistically significant association seen between Trop I and ABI value (P<0.05), showing that there is a causal relationship between Trop I and ABI value.

Table No. 16 Association of ABI clinical grading with groups

ABI	Groups					Total
	Chewer	Control	Mild smoker	Moderate smoker	Severe smoker	
1	4 16.0%	17 68.0%	5 20.0%	2 8.0%	1 4.0%	29 23.2%
2	7 28.0%	8 32.0%	7 28.0%	7 28.0%	5 20.0%	34 27.2%
3	10 40.0%	0 0.0%	12 48.0%	13 52.0%	14 56.0%	49 39.2%
4	4 16.0%	0 0.0%	1 4.0%	3 12.0%	5 20.0%	13 10.4%
Total	25 100.0%	25 100.0%	25 100.0%	25 100.0%	25 100.0%	125 100.0%

Pearson Chi-Square = 49.223, DF = 12, P-Value = 0.000, Highly significant

The table 16 shows the association between ABI clinical grading and the groups. There was a statistically significant association seen between ABI clinical grading and groups (P<0.05), showing that there is a causal relationship between ABI clinical grading and groups.

Table No. 17 Association of ABI clinical grading with Habit Year [Smoker Group]

ABI	Habit Year					Total
	1	2	3	4	5	
1	4 26.7%	3 13.6%	1 6.3%	0 0.0%	0 0.0%	8 10.7%
2	7 46.7%	9 40.9%	2 12.5%	1 6.7%	0 0.0%	19 25.3%
3	4 26.7%	9 40.9%	13 81.3%	9 60.0%	4 57.1%	39 52.0%
4	0 0.0%	1 4.6%	0 0.0%	5 33.3%	3 42.9%	9 12.0%
Total	15 100.0%	22 100.0%	16 100.0%	15 100.0%	7 100.0%	75 100.0%

Pearson Chi-Square = 37.306, DF = 12, P value = 0.000, Highly significant

The table 17 shows the association of ABI clinical grading with habit year in the Smoker group. There was a statistically significant association seen between the ABI clinical grading and habit year (P<0.05), showing a causal relationship between ABI clinical grading and habit years.

Table No. 18 Association of CAD grading of ABI with groups

CAD	Groups					Total
	Chewer	Control	Mild smoker	Moderate smoker	Severe smoker	
2	2 8.00%	9 36.00%	2 8.00%	0 0.00%	0 0.00%	13 10.40%
3	6 24.00%	14 56.00%	6 24.00%	6 24.00%	2 8.00%	34 27.20%
4	11 44.00%	2 8.00%	15 60.00%	11 44.00%	12 48.00%	51 40.80%
5	6 24.00%	0 0.00%	2 8.00%	8 32.00%	11 44.00%	27 21.60%
Total	25 100.00%	25 100.00%	25 100.00%	25 100.00%	25 100.00%	125 100.00%

Pearson Chi-Square = 56.486, DF = 12, P-Value = 0.000, Highly significant

The table 18 shows the association between CAD grading of ABI and the various groups. CAD gradin g of ABI grade 2 was seen more in the chewer group 2 (8.0%) and the control group 9 (36.0%), CAD grading of ABI grade 3 was more commonly seen in control group 14 (56.0%), while CAD grading of ABI grade 4 was more commonly seen in the mild 15(60.0), moderate11 (44.0%) and severe smokers 12 (48.0%) and CAD grading of ABI grade 5 more common in severe smokers 11 (44.0%), followed by moderate smoker 8 (32.0%) and chewer group 6 (24.0%). There is a statistically significant association seen between the CAD grading of ABI and the various groups (P<0.05), showing that the distribution of patients in various groups in relation to CAD grading of ABI is varying.

Table No.19 Association of CAD grading of ABI with Habit Year [Smoker Group]

CAD	Habit Year					Total
	1	2	3	4	5	
2	1 6.7%	1 4.6%	0 0.0%	0 0.0%	0 0.0%	2 2.7%
3	8 53.3%	5 22.7%	1 6.3%	0 0.0%	0 0.0%	14 18.7%
4	5 33.3%	12 54.6%	12 75.0%	7 46.7%	2 28.6%	38 50.7%
5	1 6.7%	4 18.2%	3 18.8%	8 53.3%	5 71.4%	21 28.0%
Total	15 100.0%	22 100.0%	16 100.0%	15 100.0%	7 100.0%	75 100.0%

Pearson Chi-Square = 32.868, DF = 12, P value = 0.001, Highly significant

The table 20 shows the association of CAD grading of ABI with habit year in the Smoker group. There was a statistically significant association seen between the CAD grading of ABI and habit year ($P<0.05$), showing a causal relationship between CAD grading of ABI and habit year.

Table No. 21 Association of Habit Years with CAD grading of ABI [Smoker Group]

Habit Years	CAD				Total
	2	3	4	5	
1	1 50.00%	8 57.14%	5 13.16%	1 4.76%	15 20.00%
2	1 50.00%	5 35.71%	12 31.58%	4 19.05%	22 29.33%
3	0 0.00%	1 7.14%	12 31.58%	3 14.29%	16 21.33%
4	0 0.00%	0 0.00%	7 18.42%	8 38.10%	15 20.00%
5	0 0.00%	0 0.00%	2 5.26%	5 23.81%	7 9.33%
Total	2 100.00%	14 100.00%	38 100.00%	21 100.00%	75 100.00%

Pearson Chi-Square = 32.868, DF = 12, P value = 0.001, Highly significant

The table 21 shows the association between CAD grading of ABI and habit years in the Smoker group. There was a statistically significant association seen between CAD grading of ABI and habit years ($P<0.05$), showing that CAD grading of ABI is dependent on the habit years in the smoker group.

Discussion

Tobacco chewing and smoking has been recognized as a major risk factor for the development of ischaemic heart disease and it may lead to alteration of lipid profile. Ankle brachial index can be used as a noninvasive method of assessing asymptomatic PAD. It provides important information with respect to subclinical atherosclerosis. In many correlation studies, it was found that, the sensitivity of ABI for detecting PAD is about 90% and specificity is about 98% when compared to angiography. It is already known that an inverse relationship exists between ABI and cardiovascular disease and that ABI can be a marker for generalized atherosclerotic disease.

In our study the mean age in the chewer group was 52.16 ± 14.24 years, in the control group it was 45.84 ± 9.30 years, smoker group it was 51.48 ± 11.52 years. In our study a higher proportion of males in each group in comparison to the females. As in chewer group 32% are females and 68% are males, in mild smoker 88% are males and 12% are females, in moderate

smokers 84% are males and 16% are females and in severe smoker group all are males (100%).

The mean total cholesterol in chewer group was 205.53+25.28 mg/dl, in the control group It was 161.21+16.09 mg/dl, in smoker group it was 206.21+22.70 mg/dl. There was no statistically significant difference seen in the mean cholesterol of smokers and tobacco chewer but statistically significant difference between chewer control and smoker control group. The mean LDL in chewer group was 154.34+20.25 mg/dl, in control group it was 84.73+13.79 mg/dl, in smoker group it was 124.82+25.29mg/dl. There was statistically significant difference seen between chewer-control group pair and smoker – control pairs. The mean triglyceride in chewer group 117.48+37.40 mg/dl, smoker group 178.16+28.18 mg/dl control. So statistically difference between tobacco-smoker and smoker – control group. The mean HDL in smoker group was 44.67+5.43, chewer group was 32.58+4.75 and control group was 51.75+6.64. There was statistically significant difference in mean HDL value between control-chewer pair and smoker -control pair. The mean VLDL in chewer group was 24.62+6.32 mg/dl smoker group37.08+10.38 mg/dl, control group 22.58+1.64. statistically significant difference in VLDL between chewer –control and smoker – control group. The mean triglyceride in smoker group was 178.16+28.18mg/dl, in chewer it was 117.48+37.40 mg/dl, in control it was113.01+8.04 mg/dl. Statistically significant difference between smoker- control and chewer-control group. So mean total cholesterol, LDL, VLDL and Triglyceride values increased and HDL decreased in smokers and tobacco chewers than controls in ourstudy. So it was concluded that Cigarette smoking was found to be more atherogenic than tobacco.

In our study more number of patients with trop –I positive found in moderate (11 patients, 44%) and severe smokers (15 patients 60%) than mild smokers (9 patients, 36%) and chewers (9 patients, 36%).there is higher CPKMB (108.20±69.50) in severe smoker followed by moderate smoke (74.80±77.30) followed by mild smoker (64.99±33.57)followed by chewer (63.25 ±33.56) followed by control (16.24±4.69).So with results we can say there is increase incidence of myocardial infarction in the smokers and tobacco chewers than control.

In our study Unstable angina was the most common electrocardiographic change in both tobacco chewers and smokers (57patients, 45.6%) than STEMI (28 patients 22.4%) than NSTEMI (15 patients, 12%). STEMI is common in smokers (32% in moderate and 48% in severe) and NSTEMI is common in tobacco chewers (in28%). The smokers had a greater prevalence of infarction and less unstable angina, probably related to younger age and due to the procoagulant effect (MI in smoker).

In our study Anterior wall is commonly involved in smoker group in 33.3% followed by inferior wall (22%) while both anterior and inferior wall are equally involved in chewer group (i.e. 28%).

In our study, in smokers, anterior wall STEMI (44.1%) was the most common presentation followed by acute inferior wall STEMI (34.3%). The mean ABI in smoker group (0.64±0.19) and chewer group (0.67±0.25) was less than control group (0.99±0.14).there was statistically significant difference seen in control – chewer group ($p < 0.05$) and smoker control group, but no statistically significant difference between chewer –smoker group ($p > 0.05$).The 68% of patients in control group were in class 1 of clinical grading of ABI,while40%ofchewergroupwereingrade3ofABI and52%of smoker were in grade 3, significant association between ABI with groups ($p < 0.05$), shows that more atherosclerosis activity in smoker and tobacco chewers than control group as ABI CAD grading 2 seen more in chewer group. The grade CAD grading of ABI was seen more in the chewer group 2 (8.0%) and the control group 9 (36.0%), CAD 3 was more commonly seen in control group 14 (56.0%), while CAD 4 was more commonly seen in the mild 15 (60.0%), moderate 11 (44.0%) and severe smokers 12 (48.0%) and CAD 5 more common in severe smokers 11

(44.0%), followed by moderate smoker 8(32.0%) and chewer group 6(24.0%). As the number of cigarette/bidi smoked increased, the clinical and CAD grading of ABI increased and value of ABI decreased indicative of more severe atherosclerotic activity. the lower value of ABI <0.9 is associated with increased risk of CAD.

So as the number of years of habit increased the clinical and CAD grading of ABI increased. And increase in the duration of smoking increased the incidence of myocardial infarctions.

Conclusion

Both tobacco chewers and smokers (cigarette and bidi smokers) are at increased risk of coronary artery disease as compared to non-tobacco consuming and non-smoking population. Tobacco chewing & smoking causes decrease in HDLc & increase in TC, LDL, VLDL, triglyceride indicating that they were independently associated with such an unfavorable lipid profile thereby greatly increasing the cardiovascular risk particularly for coronary artery disease. Cigarette smoking was found to be more atherogenic than tobacco chewing.

The findings of this research indicated that ABI could be a useful method in assessing both the atherosclerotic risk factors and the degree of coronary involvements in suspected patients.

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