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Original Research Article

Monocyte to High Density Lipoprotein Cholesterol Ratio in Smokers

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Abstract:

Background: smoking is a leading cause of mortality. Smoking has been linked to low grade inflammation. Recently the monocyte to high density lipoprotein cholesterol ratio (MHR) emerged as an indicator of inflammation. So we aimed to investigate the relationship between MHR and cigarette smoking.

Material and Methods: this was Hospital based cross sectional study. 25-35 years old 200 males were included in the study & divided in two groups. Smoker group comprising 100 smokers having duration of smoking more than 3 years. 100 randomly selected non-smokers were included in the non-smoker group. Detailed history, smoking habits number of cigarette smoked per day and pack year was calculated. Complete blood count was done by procon PE 600 automated analyzer. Total cholesterol, HDL cholesterol, triglyceride was measured on AU 5800 Backmann coulter fully automated biochemical analyzer using enzymatic colorimetric assay. Statistical analysis was done by unpaired student 't' test. Correlation was determined by Pearson correlation Coefficient test.

Result: MHR was significantly higher in smokers as compared to non-smokers (14.4 $\pm$ 2.02, 11.1 $\pm$ 1.98 respectively). Pearson's correlation analysis revealed significant positive correlation between pack years, number of cigarette smoked daily and MHR in smokers group. Monocyte to high density lipoprotein can be used as a surrogate marker of inflammation and endothelial dysfunction.

Keywords: smoking, monocyte to high density lipoprotein cholesterol ratio (MHR) inflammation..

Introduction

Smoking is the most important public health problem according to WHO 2.4 billion people worldwide have consumed tobacco in the form of chewing sniffing or dipping. WHO estimates tobacco related deaths to 8.3 million in 2030 and 1 billion deaths during the 21st century [1].

Toxic ingredients in cigarette smoke circulate throughout the body causing damage in several different ways. The burning tobacco and paper produce more than 4000 chemical compounds in form of gases, vapors particulates like carbon monoxide hydrogen cyanide phenols Ammonia formaldehyde Benzene nitrosamines nicotine and tar [2] 194 million man and 45 million women use tobacco in smoke or smokeless form in India [3].

Smoking has been linked to low grade systemic inflammation as reflected in elevated white blood cell count a well established predictor of myocardial infarction cancer and chronic obstructive pulmonary disease. Cigarette smoke contains oxidant free radicals that are capable of initiating or promoting oxidative damage leading to degenerative pulmonary, cardiovascular disease and cancer [4].

Oxidative damage to unsaturated lipid is a well-established general mechanism for oxidant mediated cellular injury [5].

Smoking is associated with a more atherogenic lipid profile [6] several toxins present in cigarette smoke have immunomodulatory effects. Cigarette smoke constituents induce chronic inflammation at the mucosal surface and modify the host response to exogenous antigen [7].

The ratio of monocyte to high density lipoprotein cholesterol ratio was defined as a cardiovascular prognostic marker indicating the extent of inflammation and oxidative stress. [7,8,9] So we planned the study to analyze the effect of cigarette smoking and its intensity with the lipid profile and MHR ratio.

Material and Methods

The study was approved by institutional ethical committee. study conducted at tertiary Health Care Centre. 200 male participants aged 25 to 35 years were included in the study. Participants were divided in two group: 1) smoker group comprised, 100 smokers having duration of smoking more than 3 years.

2) non-smoker group included 100 non-smokers selected randomly from the general population.

Exclusion criteria.

Patients with presence of chronic disease such as Diabetes mellitus, hypertension, coronary artery disease, heart failure, chronic lung disease, connective tissue disease, chronic kidney disease, metabolic syndrome, leukocytosis, leukopenia or other hematological biochemical or serological abnormalities were excluded. Subjects consuming lipid lowering drugs, steroids, hematinic were excluded.

Detailed data about smoking habits was collected using structural interviewing questionnaires. Smoking characteristics such as the number of cigarettes smoked daily, Number of Pack year was calculated. Pack year represents a combined measure of dose and duration of smoking. Pack year was calculated as the number of cigarettes smoked per day * number of years smoked / 20.

Standing height in centimeter was measured by asking the participants to stand bare feet against the wall on which the measuring scale was inscribed [10]

Weight was measured in kilograms with KRUPS weighing machine in lightweight garments without footwear.

Body mass index was calculated using Quetelet's formula - Body weight in kilogram / height meter squared.

Blood pressure was measured with sphygmomanometer by the standard auscultatory Riva Rossi method.

Random blood sugar was measured.

Venus blood sample was collected after 12 hours of fasting from the antecubital vein with all aseptic precautions. Blood was drawn and transferred into EDTA and plain bulb equally.

Hematological parameters were studied by Procan PE 600 three part differentiated automated hematology analyzer.

Total cholesterol HDL cholesterol triglyceride was measured on AU 5800 Backmann coulter fully automated biochemical analyzer.

LDL cholesterol was calculated by friedwald formula.

Statistical analysis.

Statistical analysis was done by software SPSS version 22. Continuous variables were expressed as mean and standard deviation. Analysis was done by unpaired 't' test and correlation by Pearson correlation test.

Result

The mean age, weight, height and body mass index was matched in both smoker and non-smoker group. There was no statistical difference in age, height, weight and body mass index of smoker and nonsmoker group.

The study included 200 participants. It was observed that monocyte, High density lipoprotein Cholesterol ratio values for the smoker group was significantly higher in smokers than those of non-smoker group (Respectively14.4+/-2.02 and 11.1+/- 1.98).

Triglyceride low density lipoprotein cholesterol, total cholesterol, leukocyte count monocyte values for the smoker group were significantly higher than those of the non-smoker group.

High density lipoprotein cholesterol value was significantly less in smoker group than non-smoker group.

There was significant positive correlation between pack year, number of cigarette smoked daily and monocyte to high density lipoprotein cholesterol ratio.

There was positive correlation between Triglyceride, total cholesterol, low density lipoprotein cholesterol and pack year, the number of cigarette smoked daily in smoker group.

There was negative correlation between high density lipoprotein cholesterol and pack years, the number of cigarette smoked daily in smoker group.

Table 1: Anthropometric Characteristics of Smokers and non-smokers

Variable	Smokers	Non Smokers	P value
Age (years)	29.38 ± 1.95	28.44 ± 1.25	NS
Weight (kg)	57.68 ± 5.07	56.67 ± 4.71	NS
Height (Cm)	158.58 ± 5.03	156.57 ± 4.75	NS
BMI kg/m^2	23.10 ± 1.85	23.28 ± 1.54	NS

Table 2: comparison of Hematological and lipid profile in Smokers and Non Smokers

Variable	Smokers	Non Smokers	P value
WBC X $10^3/mm^3$	14.67 ± 5.19	8.28 ± 3.76	S
Monocyte x $10^3/mm^3$	0.75 ± 0.53	0.55 ± 0.31	S
Total cholesterol mg/dl	194.21 ± 12.05	175.38 ±11.32	S
Triglyceride mg/dl	107.12 ±25.21	96.12 ± 14.25	S
HDL Cholesterol mg/dl	49.01 ± 5.47	53.34 ± 3.14	S
LDL Cholesterol mg/dl	123.4 ± 10.32	106.81 ± 8.85	S
Monocyte to HDL Cholesterol Ratio (MHR Ratio)	14.4 ± 2.02	11.1 ± 1.98	S

Table 3: Correlation analysis between Smoking as pack years, MHR & Blood lipid profile

Variable	pack years	
	r	P Value
MHR	0.254	S
Monocyte x $10^3/mm^3$	0.203	S
HDL Cholesterol (mg/dl)	- 0.104	S
Triglyceride (mg/dl)	0.231	S
Total cholesterol (mg/dl)	0.195	S
Low density Lipoprotein cholesterol(mg/dl)	0.198	S

Table 4: Correlation analysis between the number of cigarettes smoked daily MHR and blood lipid levels.

Variable	The number of cigarettes smoked daily	
	r	P Value
MHR	0.329	S
Monocyte x $10^3/mm^3$	0.295	S
HDL Cholesterol (mg/dl)	-0.279	S
Triglyceride (mg/dl)	0.293	S
Total cholesterol (mg/dl)	0.105	S
Low density Lipoprotein cholesterol	0.132	S

Discussion

In the present study we found that the monocyte to High density lipoprotein ratio was significantly higher in the smoker group than non-smoker group. Total cholesterol and Triglycerides was significantly higher in the smoker group.

According to several recent studies exposure to cigarettes smoke impairs functional structure of endothelial cells. Nicotine and increase oxidative stress generated from smoking induce vascular endothelial dysfunction via the inhibition of endothelial nitric oxide synthase and decreasing generation of nitric oxide [10,11].

Nicotine increases the expression of adhesion molecules in endothelial cells such as E select and intracellular adhesion molecule because of enhanced attachment and transmigration of monocytes in the vessel wall. [12].

It is evident that these results suggest that smoking is an established risk factor for atherosclerosis through several underlying pathways. Monocytes are a distinct type of leukocytes which have a key role in inflammation and atherosclerosis process [13].

Activated monocytes interact with damaged or activated endothelium which result in over expression of pro inflammatory cytokines / adhesion molecules and intracellular adhesion molecule. There after monocytes differentiate into the macrophages that ingest oxidized LDL-C and form dangerous foamy cells[14].

In another study The Count of circulating monocytes was found to be a predictor for new plaque development as well. [15] however HDL-C features anti-inflammatory antioxidants and antithrombotic effect. [14.16.17] HDL- C can prevent inflammatory response by acting directly on monocytes. Recent studies indicate the role of HDL -C in modulating monocyte activation adhesion and in controlling the proliferation of progenitor cells that differentiate to monocyte. HDL-C also prohibits oxidation of LDL-C IN addition to inhibition of macrophages migration. It also removed oxidized LDL-C from foamy cells. [16,17,18,19,20].

Therefore monocytes show a pro inflammatory effect but HDL-C functions as reversal factor during the process. It has been suggested that MHR has a relationship with systemic inflammation and endothelial dysfunction and it is accepted as newly

recognized inflammation based diagnostic and prognostic marker in cardiovascular disease [21,22,23,24]. Recently Acikgoz et all assessed

endothelial functions using flow and nitroglycerine mediated dilation technique and calculation of MHR. The study reported there was a strong inverse correlation between MHR and flow mediated dilatation. Therefore elevated MHR may be a useful marker reflecting impaired endothelial function and systemic inflammation[25].

The relationship between smoking systemic inflammatory response, vascular endothelial injury and atherosclerosis has been well defined

MHR can be used as a surrogate marker of inflammation and endothelial dysfunction.

Conclusion

MHR is a simple easy cost-effective tool that should be used for predicting the systemic inflammatory response and possible endothelial dysfunction in smokers. Cases with high MHR can easily be identified during routine complete blood analysis and could possibly benefit from preventive treatment. Therefore more attention should be given to these indices in the examination of smokers.

Declaration by Authors

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