Role of Serum Malondialdehyde (MDA) and Vitmin- C Level in Non-Smokers and Chronic Smokers with Acute Myocardial Infarction (AMI) in Male

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Abstract

Introduction: Cigarette smoking is recognized as a serious health hazard as each cigarette tears away 7-11 minutes of human life. The present study aimed to investigate the serum MDA and vitamin c levels in non-smoker and chronic smoker with AMI patient and its association with cigarette consumption. Cigarette smoke contains many oxidants capable of generating reactive oxygen species and reactive nitrogen species. These species play a key role in oxidative stress, leading to the development and progression of many disorders, including hypertension, cancer, diabetes mellitus and cardiovascular diseases.

Methods: This study was conducted in the Medicine Department at DMMC & SMHRC, Nagpur in collaboration with ABVRH, Sawangi (Meghe) during September 2020 to January 2021. Informed consent was obtained from all the subjects. 40 controls who were apparently healthy non-smokers were included after appropriate matching (for age and sex) and 40 apparently healthy chronic smokers were selected 40 chronic smokers with diagnosed acute myocardial infarction were selected from SMHRC. All of them were male subjects

Result: The MDA and vitamin C were compared between Group A and Group B and also between Group B and Group C. There was a significant rise in MDA ($p<0.0001$) and significant decrease in vitamin C ($p<0.01$) in Group B compared to Group A. There was a significant rise in MDA ($p<0.0001$) and significant decrease in vitamin C ($p<0.001$) in Group C compared to Group B.

Conclusions: The increase in serum MDA level and decrease in vitamin C was found in chronic smokers compared to non-smokers. It was also found that there is increase in serum MDA and decrease in vitamin C in smokers with AMI compared with smokers without AMI, and the reason for this inter-subject variability of MDA and vitamin C levels may be due to gene-environmental factors.

Key Words: *Chronic smokers, Myocardial infarction, MDA, Vitamin C*

Introduction

Cigarette smoking is a major health issue and causes large number of deaths worldwide. Smoking has been reported to be a risk factor for oral cancer, oesophageal cancer, lung cancer and even liver cirrhosis.[1]

Cigarette smoke is a complex mixture of chemicals containing more than 4000 different constituents. Some of the compounds identified include pyridine alkaloids such as nicotine, ammonia, acrolein, phenols, acetaldehyde N-nitrosamine, polycyclic aromatic hydrocarbons such as benzopyrine, combustion gases such as carbon monoxide, nitrogen oxides, hydrogen cyanide, and trace metals, α-emitter radioactive elements such as polonium, radium and thorium etc.[2]

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Smoking is a potent but modifiable risk factor of cancer, chronic obstructive pulmonary disease (COPD) and cardiovascular diseases. It acts substantially via reactive species to initiate and promote pathology and is consistently associated with elevated oxidative damage to DNA, lipids and proteins.[2,3] It has been suggested that cigarette smoking done regularly more than 10 per day, constitute a major risk for coronary heart disease (CHD).[4]

Cigarette smokers have an increased risk of cardiovascular diseases (CVD), possibly mediated by elevated levels of oxidized macromolecules owing to heightened ROS production. Smokers are exposed to significant quantities of ROS in both gas and tar phase. Further ROS production mediated through inflammatory processes may exacerbate those produced through direct exposure. Blood of cigarette smokers routinely displays decreased antioxidant capacity and increased oxidized lipids compared to non-smokers. [5]

Malondialdehyde is a organic compound with the formula CH2 (CHO) 2. This reactive species occurs naturally and is a marker for oxidative stress. Reactive oxygen species degrade polyunsaturated lipids present on cell membrane forming malondialdehyde. This aldehyde product is used as a biomarker to measure the level of oxidative stress in an organism.[6]

MDA is a sensitive marker of lipid peroxidation. Free radicals released from smoking degrade polyunsaturated lipids present mainly in cell membranes forming malondialdehyde (MDA). This compound is a reactive aldehyde and is one among the many reactive electrophilic species that cause toxic stress in cells and form covalent protein adducts which are referred as advanced lipoxidation end products.[6]

Antioxidants depletion or deficiency may contribute to oxidative stress. Antioxidants not only protect against the direct injurious effects of oxidants, but also alter the inflammatory events that play an important role in the pathogenesis of oxidative stress related diseases.[7]

Vitamin C is a potent water-soluble antioxidant because, by donating its electrons, it prevents other compounds from being oxidized. The species formed after the loss of one electron is a free radical, semidehydroascorbic acid or ascorbonyl radical, a reactive and possibly harmful free radical. Many studies have demonstrated low plasma concentration of vitamin C in smokers and acute myocardial infarction (AMI) patients.[2,8]

Vitamin C functions as antioxidant by preventing others substances from being oxidized by donating its electrons. However in this process vitamin C oxidized itself. The compound formed after loss of electron is ascorbyl radical which is relatively stable with half life of 10─5 seconds and is fairly unreactive which explains the antioxidant function of ascorbic acid.[9]

The mechanism involved in the reduction of vitamin C level in smokers may be due rapid oxidation of ascorbic acid by free radicals. The negative relationship between vitamin C and MDA may be due to the depletion of vitamin C when the oxidant burden is increased.

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Present study is undertaken to evaluate serum malondialdehyde as indicator of oxidative stress and serum vitamin C as indicator of antioxidant level in non -smokers and chronic smokers with acute myocardial infraction AMI in male.

Materials and Methods

This study was conducted in the Medicine Department at DMMC & SMHRC, Nagpur in collaboration with ABVRH, Sawangi (Meghe) during September 2020 to January 2021. Informed consent was obtained from all the subjects. 40 controls who were apparently healthy non-smokers were included after appropriate matching (for age and sex) and 40 apparently healthy chronic smokers were selected 40 chronic smokers with diagnosed acute myocardial infarction were selected from SMHRC. All of them were male subjects.

Inclusion Criteria: Both chronic smokers and chronic smokers with myocardial infarction, who smoked more than 10 cigarettes per day for more than 09 years, were selected.

Exclusion Criteria: Those excluded from the study were persons abusing alcohol, ex-smokers, patients with diabetes mellitus, hypertension, renal diseases, hepatic impairment, endocrine disorders, obese individuals and

patients on drugs like multivitamins etc.

Sample Collection: 5 ml blood sample was collected venipuncture method in all subjects and care was taken to ensure that in AMI patients, blood sample was collected before thrombolysis of patient. Serum was separated in 3000 rpm for 1 min and analyzed for the MDA and vitamin C level.

Biochemical Analysis:[11,12]

· MDA was measured by thiobarbituric acid reactive substances assay (TBRAS) method.

· Ascorbic acid (Vitamin C) measured by dinitrophenyl hydrazine (DNPH) method.

Statistical Analysis

The data were analyzed using SPSS software program, version 20.0. The mean and standard deviation were measured. Analyzed and interpreted using descriptive and inferential statistics. The probability value is less than 0.05 ($p < 0.05$) and it was considered as statistically significant.

Result

Table no 1 shows the serum levels of MDA and vitamin C in non-smokers (Group A) and chronic smokers (Group B). The MDA of Group A was 238±3.54nmol/l and in Group B was 432±9.0nmol/l. The mean vitamin C in Group A was 1.20±0.10mg/dl and that of Group B was 0.98±0.18mg/dl. MDA was significantly higher ($p<0.0001$) in Group B compared to Group A. Vitamin C level was significantly lower ($p<0.01$) in Group B than in Group A.

TABLE NO: 01: serum levels of MDA and vitamin C in non-smokers (Group A) and chronic smokers (Group B).

Parameter	Non-Smokers (Group A)	Chronic Smokers (Group B)	P- Value
MDA nmol/l	238±3.54	432±9.0	P<0.0001
Vit C mg/l	1.20±0.10	0.98±0.18	P<0.01

Table no 02 shows the serum levels of MDA and vitamin C in chronic smokers (Group B) and chronic smokers with MI (Group C). The MDA of Group B was 432±9.0nmol/l and in Group C was 530±15.90 nmol/l. The mean vitamin C in Group B was 0.98±0.18mg/dl and that of Group C was 0.73±0.20 MDA was significantly higher ($p<0.0001$) in Group C compared to Group B. Vitamin C level was significantly lower ($p<0.001$) in Group C than in Group B.

TABLE NO: 02: serum levels of MDA and vitamin C in chronic smokers (Group B) and chronic smokers with MI (Group C).

PARAMETER	CHRONIC SMOKERS (GROUP B)	CHRONIC SMOKERS WITH AMI (GROUP C)	P- VALUE
MDA nmol/l	432±9.0	530±15.90	P<0.0001
Vit C mg/l	0.98±0.18	0.73±0.20	P<0.01

Discussion

The present study showed that there was a significant elevation of serum MDA ($p<0.0001$) and significant decrease in serum ascorbic acid (vit c) levels ($p<0.01$) in chronic smokers compared to non-smokers.

Cigarette smoking is probably the most addictive and dependence producing form of object-specific, self-administered gratification known to man. According to present estimates, tobacco is responsible for causing more than 5 million deaths every year (World Health Organization, 2008). About 19% of tobacco consumption in India is in the form of cigarettes, while 53% is smoked as bidis. Regular smoking doubles the chances of stroke in men.[13]

It has been estimated that 1016 radicals are present in one puff of cigarette smoke. Free radicals can oxidize lipid, protein and carbohydrate molecules, damaging cell membranes and DNA, thereby altering cellular structure and function. Cell membranes are rich sources of polyunsaturated fatty acids, which are more prone to be attacked by oxidizing radicals causing lipid peroxidation.[14]

Chole et al. reported association of lipid peroxidation with the habit of either chewing betel nut or betel leaf or tobacco or smoking in the control subjects.[15]

Solak et al. also reported that MDA levels in plasma also correlated with the daily exposure to cigarette smoke.[16]

In this study, we have also compared chronic smokers and chronic smokers with acute myocardial infarction. The serum MDA was significantly higher ($p<0.0001$) and serum ascorbic acid was significantly lower ($p<0.001$) in chronic smokers with AMI than in chronic smokers, even when both groups were matched for age, body weight, BMI and smoking habits. Although cigarette smoking is a well-established risk factor for vascular diseases, the genetic mechanisms that link cigarette smoking to an increased incidence of cardiovascular diseases are not well understood.

Several environmental factors are documented to influence redox metabolism, but relatively little is known about genetic effects. TAS, an indicator of redox homeostasis, is under strong genetic control, especially in smokers and this could lead to the difference in MDA and vitamin C levels in chronic smokers with MI than in chronic smokers without MI.[17]

Conclusion

In conclusion, the present findings indicated that cigarette smokers had higher lipid peroxidation levels and lower antioxidant capacity compared to non-smokers. Inter-subject variability of both elevated serum MDA and reduced serum vitamin C levels in chronic smokers with AMI may be influenced by gene-environmental factors.

Conflict of Interest: Nil

Source of Funding: Nil

Ethical Clearance: taken from institutional ethics committee

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