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A Comparative Study of Plasma Nitric Oxide Levels as Plasma Nitrates a Biochemical Marker of Endothelial Dysfunction in Passive Smokers and Non Smokers

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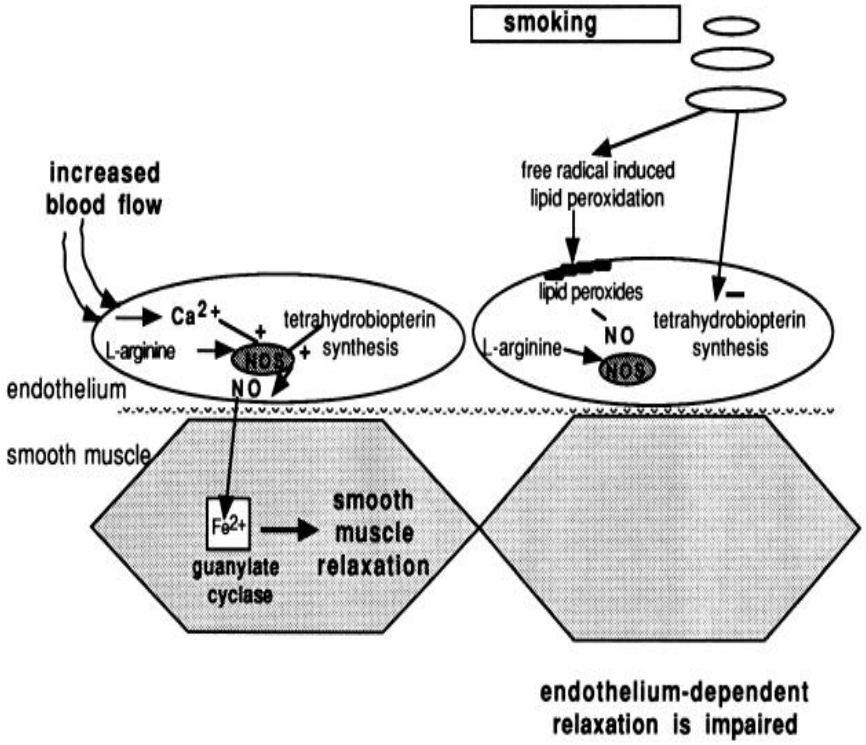
I. Introduction

Cigarette smoking is a risk factor for cardio vascular events. This detrimental effect is not only limited to active smoking but also to passive smoking s which causes vascular endothelial dysfunction, which is an early key event of impaired arterial dilatation, atherogenesis and thrombus formation, which leads to serious cardiovascular complications.

Nitric oxide is an index of endothelial dysfunction, because it is synthesized by endothelial cells from L-arginine by endogenous system nitric oxide synthase(NOS). So it is otherwise known as endothelium derived relaxing factor(EDRF). NO regulates resting vascular tone, local blood flow and tissue perfusion.

Pathogenesis of endothelium dependent relaxation of arteries in smoking:(1,2)

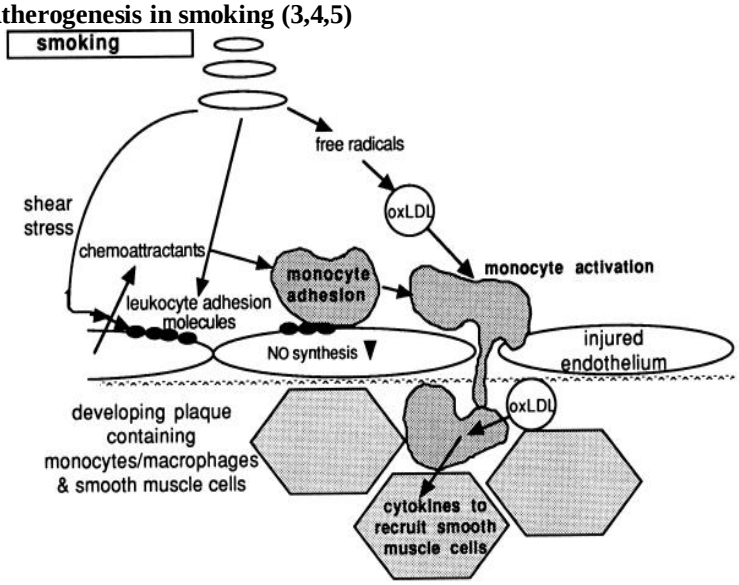


The immediate response of healthy endothelium to an increase in blood flow is an an increase in intra cellular calcium. This increase in calcium concentration stimulates the synthesis of nitric oxide (NO) from l-arinine by the enzyme nitric oxide synthase (NOS).

Essential cofactors for this activity include tetrahydrobiopterin itself synthesized in endothelium.the NO diffuses out of the endothelium to bind to Fe2+ in the haem moiety of guanylate cyclase to stimulate the synthesis of cyclic GMP. The increased concentration of cyclic GMP triggers the smooth muscle relaxation.

Smoking limits the endothelial synthesis of NO in two ways. The increased concentration of blood born free radicals accelerates cellular lipid peroxidation. The lipid peroxides avidly bind to available NO.the synthesis of NO is also reduced due to decreased synthesis of tetrahydrobiopterin. NO is no longer available to stimulate endothelium dependent relaxation, ultimately leading to vasoconstriction and ischemia of various tissues.

Pathogenesis of Atherogenesis in smoking (3,4,5)



Smoking stimulates monocyte adhesion,migration and activation. The altered shear stress at the endothelial surface induced by smoking and absorbed products of tobacco combustion have synergistic effect to increase the expression of Leukocyte adhesion molecules(T CAM-1 and V CAM-1) on endothelium. The alteration of shear stress induced by smoking also may stimulate the release of monocyte chemoattractants.

Reduced nitric oxide synthesis also increase the adhesiveness of the endothelium. The endothelium becomes leaky, permiting the entry of oxidized LDL,which activate the migrated monocyte, macrophages to release cytokines and growth factors to attract more cells (smooth muscle cells ,macrophages) to migrate and proliferate in the intima.

The atherosclerotic lesion has started to develop,continuously stimulated by smoking. Hence both active and passive smoking may lead to impaired arterial relaxation,atherogenesis,thrombus formation which inturn lead to serious cardiovascular events.

Aim: To evaluate the effect of passive smoking on vascular endothelium by estimating nitric oxide(NO) levels as plasma nitrates in passive smokers & non smokers.

Objectives: To estimate the levels of NO as plasma nitrates in passive smokers (cases) and in non smokers (controls) and compare the levels between 2 groups to hypothesise endothelial dysfunction caused by passive smoking.

II. Materials And Methods

Inclusion criteria:

1. Passive smokers includes the individuals who have been regularly exposed to side stream tobacco smoke for atleast 1-2 hrs in a day for 3-6 yrs period either at home or at work place. Active smokers are the one who burns the cigarette and inhales the main stream smoke.
2. Non smokers include the individuals who had never been regularly exposed to tobacco smoke.
3. Individuals of both the sexes in the age group 20-40 yrs.

Exclusion criteria:

1. Individuals with hypertension, diabetes, coronary artery disease.
2. Individuals with peripheral vascular diseases
3. Patients who are on nitrate therapy.

- A case control study was done. Total 100 subjects were included in the study and divided in to 2 groups. Group 1 includes passive smokers (cases) n=50 and Group 2 includes non smokers (controls) n=50.

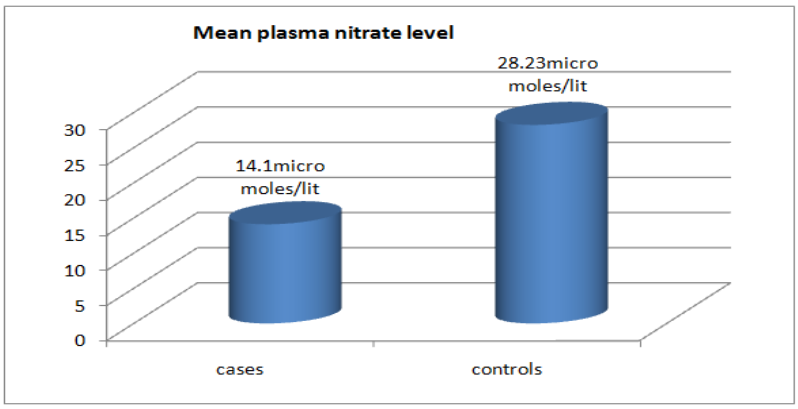
Sample collection:

1. 4 ml of random peripheral venous blood sample was collected in to dry cleanheparinised test tubes.
2. Centrifugation of sample was done for 15 min and plasma separated is stored at -50 degree celsius and that was used for investigation purpose.

Method: Nitric oxide levels in terms of plasma nitrates was measured in cases and compared to that of controls by using Griess method. In a clean dry test tube 0.5 ml of plasma taken , $ZnSO_4$ and NaoH are added mixed well and centrifused for 10 min and thus obtained deproteinised sample is used for the estimation of nitrates by cadmium reduction.

III. Results

Data was analyzed by SPSS17.0 software system. Comparison of two groups done by students t test. Mean plasma nitrate level in controls was 28.23 micro moles/lit and in cases it was 14.1 micro moles/lit, P-value is 0.001 which is statistically significant.



.cases=passive smokers, controls=non smokers

We found significant decrease in plasma nitrates level in passive smokers compared to non smokers dilatation and the formation of thrombus which may lead to serious cardiovascular events.

IV. Discussion

Active smoking is one of the major riskfactor for cardiovascular events. This detrimental effect is no only limited to active smoking but also to passive smoking.

In our study we estimated the plasma nitrates which is the stable product of nitric oxide in passive smokers (people who have been regularly exposed to tobacco smoke for 1-2 hrs in a day).

In the study we found a significant decrease in plasma nitrates level (an index of nitric oxide) in passive smokers when compared to non smokers (people who have not been regularly exposed to tobacco smoke).

Free radicals present in cigarette smoke decreases the synthesis of nitric oxide and also causes the process of atherogenesis . this inturn causes the impaired arterial dilatation and the formation of thrombus which may lead to serious cardiovascular events.

V. Conclusion

In our study the plasma nitrate levels in passive smokers is decreased compared to non smokers which can be considered as biochemical marker for endothelial dysfunction a forerunner
of impaired arterial dilatation(vasoconstriction), atherosclerosis, thrombosis leading to acute cardiovascular events.

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