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Original Article

Nerve Conduction Velocity and Total Antioxidant Capacity among Cigarette Smokers

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Abstract

Cigarette smoking has been implicated in establishment and progression of several diseases. Smoking causes free radical induced oxidative stress. Increased oxidative stress in smokers can alter the conduction velocity of sensory and motor nerves. With this background the present study was planned to see the effect of chronic smoking on nerve conduction velocity and total antioxidant capacity (TAC) and to find correlation if any between TAC and nerve conduction velocity.

The study was conducted on 45 smokers and 45 non- smokers. Conduction velocity was measured in sensory and motor component of ulnar and median nerves of smokers and non-smokers. Plasma TAC was estimated by ELISA. In our study statistically significant decrease

in sensory nerve conduction velocity of ulnar (34.82±21.45 m/s) and median nerve (42.77±23.29 m/s)of smokers was observed as compared to sensory nerve conduction velocity of ulnar nerve (53.19±14.47 m/s) and median nerve (60.20±14.56 m/s) of non-smokers (P<0.001). Whereas no significant changes were observed in motor nerve conduction velocity of ulnar and median nerve in smokers compared to non-smokers. Total antioxidant capacity was statistically decrease in smokers (28.12±13.23 mmol TE/L) as compared to non-smokers (74.34± 23.35 mmol TE/L) (P<0.0001). A negative correlation was observed between smoking index and TAC indicating that smoking causes oxidative stress. A positive correlation was observed between TAC and sensory and motor nerve conduction velocity in both the nerves. Correlation between TAC and sensory nerve conduction velocity of ulnar and median nerve was found to be statistically significant. On the basis of study it is concluded that oxidative stress induced by smoking contributes to significant decrease in sensory nerve conduction velocity.

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Introduction

Cigarette smoking is a serious health problem. Cigarette smoke is a complex mixture of chemical compounds containing many free radicals and

oxidants. Free radicals include superoxide anion, nitric oxide and hydroxyl radicals (1). The deleterious effects of the free radicals are kept under check by endogenous antioxidant defense system (2). Increased production of free radicals associated with smoking may exceed the capacity of endogenous antioxidant defense system resulting in oxidative stress (3). Smoking is risk factor for several chronic diseases (4). It has been observed that increased oxidative stress can affect the nerve blood flow as well as nerve conduction (5). Cigarette smoking have been implicated in causing subclinical changes in myelin sheath of peripheral nerves and resulting demyelination causes conduction deficit (6). Total antioxidant capacity provides better information about overall antioxidant status of individual as compared to that obtained by measurement of individual components (7) It is well documented that smoking has profound effects on cardiovascular and respiratory system (4). But there is hardly any study available on the effect of oxidative stress on somatic nerves among cigarette smokers.

With this background the present study was planned to see the effects of cigarette smoking on nerve conduction velocity and total antioxidant capacity and to find correlation if any between TAC and nerve conduction velocity in cigarette smokers.

Materials and Methods

This observational cross sectional study was carried out in Department of Physiology at Himalayan Institute of Medical Sciences, Dehradun. Prior approval from institutional ethical committee was obtained. The study was conducted on 45 smokers and 45 non-smokers of age group 20-40 years. The sample size of 45 subjects in each group was obtained using the formula for differences of means at 90% power and á error of 0.05 (8). The subjects were recruited from staff members and residents of institute. After explaining the procedure and purpose of study a written informed consent was obtained. Smokers were interviewed about duration of smoking as well as average number of cigarette smoked per day. Smokers with history of smoking for more than 2 years, with no history of hypertension, diabetes peripheral neuropathy, nerve injury were included in the study. Control group included subjects who have not smoked in life and not addicted to tobacco chewing, gutaka, pan masala. Subjects in control group were healthy with no history of diabetes, hypertension, nerve injury and autonomic dysfunction. Smoking index (Smoking index=average number of cigarette smoked per day in last seven days ×duration of smoking in years) of each smoker was calculated to quantify smoking (9).

Study Protocol – The subjects were asked to report to Department of Physiology in morning after light breakfast and to avoid smoking 2 hours before reporting. Their medical history, personal history, drug history especially history of intake of antioxidants were taken and anthropometric parameters i.e height, weight were recorded.

The nerve conduction velocity was assessed using Neuro Pac (Octopus) -2 channel machine. All the recording were taken in sitting position at 25°C. In the present study sensory and motor nerve conduction velocity of median nerve and ulnar nerve of right forearm were measured using standard protocol (10). Recording were obtained at following instrument setting : For motor studies: sensitivity : 2-5 mv mm^{-1}, low frequency filter: 2-5 Hz, high frequency filter: 10 KHz, sweep speed: 1-2 ms mm^{-1}. For sensory studies: sensitivity: 10-20 µv mm^{-1}, low frequency filter: 2-3 Hz, sweep speed: 2-7 ms mm^{-1}. Electodiagnostic references values were considered as the normal values for motor and sensory nerve conduction (11).

Biochemical analysis

5 ml of blood sample was collected maintaining all the aseptic precautions in vacutainers containing silicate gel. The vacutaines were then centrifuged at 3000 rpm for 30 minutes. Then the serum was kept in tube at –20°C in deep freezer.

Estimation of TAC was done using Bio vision TAC Assay Kit (Catalog No K274-100). The ELISA test was performed according to manufactures direction for use.

Statistical analysis

SPSS version 19 was used for statistical analysis. Unpaired t test was used to see the difference between mean values of nerve conduction velocity and total antioxidant capacity of smokers and non-smokers group and to assess the difference in baseline parameters of two groups. Pearson's correlation was used to find out correlation between nerve conduction velocity and total antioxidant capacity. Significance for the difference was set at P<0.05.

Results

Demographic characteristics and smoking history of study subjects are given in Table I. Based on smoking index criteria the study subjects were moderate smokers (SI=101-200) with smoking index 145±167.87. In our study smokers had lower sensory nerve conduction velocity of ulnar and median nerves as compared to non-smokers and the difference was statistically significant (P<0.001). Whereas no significant changes were observed in motor nerve conduction velocity of ulnar and median nerve in smokers compared to non-smokers. Smokers had lower total antioxidant capacity (28.12±13.23 mmolTE/L) as compared to non-smokers (74.34±23.35 mmolTE/L) and the difference was statistically significant (P<.0001) (Table II). A negative correlation was observed between smoking index and TAC as well as smoking index and sensory nerve conduction velocity in ulnar and median nerve and motor nerve conduction velocity in ulnar and median nerve. A positive correlation was observed between TAC and sensory and motor nerve conduction velocity in both the nerves. Correlation between TAC and sensory nerve conduction velocity in ulnar and median nerve was found to be statistically significant (P<.01) (Table-III).

TABLE I: Demographic characteristics of Non-smokers and Smokers

Parameters	*Non-smokers (n=45)*	*Smokers (n=45)*
Age (years)	37.12±6.17	36.03±6.68
Height (cm)	167.24±7.95	166.33±9.07
Weight (kg)	62.39±12.11	57.48±12.71
Duration of smoking (yrs)	-nil	11.82±8.28
cigarettes/day	-nil	11.91±9.79
Smoking Index	-nil	145±167.87

TABLE II: Nerve conduction velocity and total antioxidant capacity in Non-smokers and Smokers.

Parameters	*Non-smokers (n=45)*	*Smokers (n=45)*	*P value*
MNC (Ulnar Nerve) m/s	56.15±13.29	55.84±13.81	.926
MNC (Median Nerve) m/s	55.47±14.55	51.16±16.14	.258
SNC (Ulnar Nerve) m/s	53.19±14.27	34.82±21.45	.001
SNC (Median Nerve) m/s	60.20±14.56	42.77±23.29	.001
TAC mmol TE/L	74.34±23.35	28.12±13.23	.0001

MNC- Motor nerve conduction, SNC-Sensory nerve conduction, TAC-Total antioxidant capacity.

Unpaired t Test. p≤.05: significant; p≤.01: highly significant.

TABLE III: Correlation among SI, TAC, MNC (UN), MNC (MN), SNC (UN), SNC (MN) of the Smokers (n=45).

Variables	*SI*	*TAC*	*MNC (UN)*	*MNC (MN)*	*SNC (UN)*	*SNC (MN)*
SI	1	-.877**	-.062	-.386*	-.719**	-.723**
TAC	-.877**	1	.032	.268	.956**	.944**
MNC (UN)	-.062	.032	1	.666**	-.042	-.018
MNC (MN)	-.386*	.268	.666**	1	.145	.172
SNC (UN)	-.719**	.956**	-.042	.145	1	.975**
SNC (MN)	-.723**	.944**	-.018	.172	.975**	1

Pearson's correlation (r-values given). *p≤.05: significant; **p≤.01: highly significant.

MNC- Motor nerve conduction, SNC-Sensory nerve conduction, TAC-Total antioxidant capacity, SI-smoking index, UN-Ulnar nerve, MN Motor Nerve.

Discussion

Cigarette smoking causes a lot of impacts on human body (4). Neural tissue is highly susceptible to oxidative damage caused by free radicals since it consist of pool of unsaturated lipids which are liable to peroxidation and oxidative modification (5). The present study showed that there was statistically significant decrease in the sensory nerve conduction velocity of ulnar and median nerves as compared to motor nerve conduction velocity. The results of our study were in accordance to those observed by Agarwal et.al. (12) and Motital et.al. (8). The cigarette smoking affects neural function by various mechanisms .Smoking induces subclinical changes in myelin sheath leading to demyelination of nerves

and consequently decrease in conduction velocity (13). Chronic smoking can induce alteration in membrane permeability properties of tissue and can result in changes in signal transduction and electrolyte imbalance (14). In our study there were no significant changes observed in motor nerve conduction velocity of ulnar and median nerve. Similar observations were made by Motital et.al. in their study (8). This may be due to the fact that sensory nerves are thinner than motor nerves and have shorter intermodal distance. As a rule the thinner nerves are affected early than thicker nerves by any damage. Hence sensory nerves are affected earlier than motor nerves (15).

Smoking enhances oxidative stress not only through the production of reactive oxygen radicals but also through weakening of antioxidant defense system. The ability of an individual to counter- act the damaging effect of free radical depends on antioxidant capacity of body fluid (16). In the present study decrease in plasma TAC was observed in smokers as compared to non -smokers. A decrease in total antioxidant capacity in smokers suggests an increased oxidative stress (17). In the study the smoking index was negatively correlated with TAC indicating that as severity of smoking both in duration of as well as average number of cigarettes smoked per day (smoking index) increases, total antioxidant capacity decreases. i.e smoking weakens antioxidant defense system. Cigarette smoking is one of the most important exogenous factors which causes 3 fold higher incidences of oxidative stress in smokers (2). In our study TAC was found to be positively correlated with nerve conduction velocity of smokers Smokers had lower levels of TAC as compared to non-smokers indicating oxidative stress in smokers which may be responsible for decrease in nerve conduction velocity of sensory nerves. Irfan Rahman et.al. in their study have also shown decrease in antioxidant capacity in healthy smokers indicating systemic oxidative stress (16). Thus studies show that smoking induces oxidative stress as the result of an imbalance between the generation of free radicals and antioxidant system. But hardly any study have shown the correlation between total antioxidant capacity and nerve conduction velocity of sensory and motor nerves of smokers. Based on the above findings it can be said that oxidative stress caused by smoking results in lipid peroxidation and estruction of lipid component of biological membranes (18) which caused a decrease in sensory nerve conduction velocity among smokers.

Limitations

The limitation of our study is small sample size.

Conclusion

Our observations suggest that chronic smoking is associated with reduction in sensory nerve conduction velocity of ulnar and median nerves. There is presence of an oxidative stress in smokers as indicated by decrease in plasma total antioxidant capacity. The oxidative stress may be responsible for nerve conduction deficits among cigarette smokers.

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