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NERVE CONDUCTION VELOCITY IN SMOKERS AND NON SMOKERS: A CASE CONTROL STUDY

Physiology

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ABSTRACT

Introduction: Chemicals that are present in cigarette/bidi smoke have been known to cause subclinical changes in myelin sheaths of eripheral nerves. Despite the antiquity and popularity of smoking, its effect has not been investigated systematically in young adults. **Aim:** To investigate the chronic effects of smoking on Nerve Conduction Velocity (NCV). **Materials and Methods:** The case-control study was conducted in the Department of Physiology, Jawaharlal Nehru Medical College, Aligarh Muslim University, Aligarh, Uttar Pradesh, India, from November 2018 to December 2020. A 40 male smokers (age group 20-60 years) along with 40 age matched healthy male non smokers as controls. The nerve conduction study was performed by using fully computerised Electromyography (EMG) and NCV machine. Sensory Nerve Conduction Velocity (SNCV) test of median and ulnar nerves was performed on subjects. Data was analysed by using unpaired t-test. **Results:** In this study of comparative analysis of total 80 subjects, (40 controls and 40 cases were taken), statistically significant changes (p-value <0.05) were found in the sensory NCV of both the nerves. **Conclusion:** It can be concluded that smoking causes more reduction in NCV.

KEYWORDS

India is among the world's top five tobacco producers and consumers. The World Health Organisation (WHO) attributed 4 million tobacco related death every year and is expected to rise by 8 million death by 2020 [1]. Two major form of tobacco use in India are smoking and chewing [2]. Near about 4200 different chemical constituents have been identified in smoking [3]. Smoke of cigarette/bidi possess a significant health hazard to human beings, especially affecting the haemodynamic of cardiovascular and cause involvement of more than one system of body. Chemicals present in cigarette/bidi smoke like nicotine, tar, carbon monoxide, tar, oxidative gases, polycyclic aromatic hydrocarbons, carbonyls, butadiene, metals, carbon disulphide and benzene etc., have been shown to cause subclinical changes in myelin sheaths of peripheral nerves and results in demyelination which causes poor electrotonic nerve conduction [5]. This may cause nerve dysfunction particularly in the form of decreases in NCV. Chronic hypoxaemia caused by prolonged tobacco exposure cause negative effect on nerves, which results in peripheral neuropathy [6].

Nerve conduction velocity is considered as the most commonly used methods to study the peripheral nerves because of their accuracy in diagnosing conditions related to nerve. It is also helpful is differentiating between the true nerve disorder and conditions which are affected by injury of nerves. Peripheral nerves, that is, ulnar and median nerves in upper extremity are most commonly chosen for NCV as they are easily reachable [7].

Hence, the present study was conducted with an aim to evaluate the effect of smoking on nerve conduction study.

The case-control study was conducted in the Department of Physiology, Jawaharlal Nehru Medical College, Aligarh Muslim University, Aligarh, Uttar Pradesh, India, on smokers and non smokers from November 2018 to December 2020. This study was approved from the Ethical Committee (Letter no. 249) of JN Medical College.

Total 80 sample subjects were taken- 40 as control and 40 as cases. A detailed history and physical examination was carried out for every subject who entered the study as per a designed proforma and the selected cases of smoking and non smoking were assessed for NCV. Both were advised for neuropathy assessment and smokers were asked to report in neurophysiology laboratory after an overnight abstinence of smoking.

All the cases (80) were divided in two groups:
Group 1 (n=40) smokers,
Group 2 (n=40) Non smokers

Inclusion criteria:
Only male smokers aged between 20-60 years, who came to the chosen study centre during the study time period were included in the study as case groups.

Those healthy age matched volunteers from the general population who were interested in participating in the study during the given time period were included as control group.

Exclusion criteria:
Those patients who came to the study centre with hypertension or with any other obvious cause of neuropathy e.g., alcohol abuse, vitamin B12 deficiency, neuropathies associated with exogenous toxic agents, metal or drugs and those patients with history of trauma in the course of nerve to be examined were excluded from the study.

Assessment of Peripheral Neuropathy
Nerve Conduction Velocity Test
Sensory Nerve Conduction Velocity (SNCV): The sensory conduction was measured orthodromically and antidromically. In orthrodomic conduction study, a distal portion of the nerve, e.g., digital nerve was stimulated and Sensory Nerve Action Potential (SNAP) was recorded at a proximal point along the nerve. In antidromic conduction study, the nerve was stimulated at a proximal point and SNAP is recorded distally. In the present study, sensory nerve action potential was recorded antidromically.

Stimuli were supramaximal and of 0.1 ms duration at a frequency of 1 Hz. The filter setting for sensory conduction were 20 Hz-3 KHz, sweep speed was 2 ms/division. The signal enhancement for averaging is generally required for sensory conduction study. The signal enhancement with averaging is proportional to the square root of the number of trials [Table/Fig-1].

Change in amplitude= n; where 'n' is the no. of trials

Nerve	Antidromic/ ortho	Stimulated site	SNAP
Median	antidromic	wrist	Index finger
Ulnar	antidromic	wrist	Little finger

The onset latency of the potential was measured from the stimulus artifact to the initial negative peak. SNCV unlike MNCV was measured by stimulating at a single stimulation site, because the residual latency which comprises neuromuscular transmission time and muscle propagation time is not applicable in sensory nerve conduction. Thus, the SNCV is calculated by dividing the distance (mm) between the stimulating and recording sites by the latency (ms). SNCV=Distance/Latency(m/s)

Statistical Analysis Descriptive statistic were used for analysis of the data.

RESULTS

There was no significant difference in the age between cases and control group in smokers and non smokers.

Table 1: Comparison between sensory nerve conduction velocity (SNCV) of Median and Ulnar in Smokers (n=40) and non smokers (n=40).

Parameters	Smokers (n=40)	Non smokers (n=40)	p-value
Rt. Median nerve velocity	51.22±4.08	55.94±3.02	<0.01
Lt. Median nerve velocity	52.73±4.96	56.55±2.21	<0.01
Rt. Ulnar nerve velocity	51.97±5.03	56.36±3.10	<0.01
Lt. Ulnar nerve velocity	52.26±4.49	55.85±2.68	<0.01

DISCUSSION

From the study, it is seen that statistically significant changes were found in conduction velocity of sensory nerves. Nerve conduction studies provide a means of demonstrating the presence and extent of a peripheral neuropathy [8]. Conduction velocity is usually reduced in demyelinative neuropathies, including smoking. NCV tests can precisely measure the degree of damage in large nerve fibres like median nerve, revealing whether symptoms are being caused by degeneration of the myelin sheath [9]. In the present study, the authors recorded sensory nerve conduction velocities using surface electrodes which require less precision in placement and are therefore quicker to use. Uncertainty of exact site of stimulation, lack of precision of measured conduction distance and uncertainty as to the temperature of the nerve can introduce errors in velocity measurements [10]. By using computerised technique, majority of these errors can be eliminated giving more reliable and reproducible results. The conduction velocity values found in this study are seen similar to those observed by Agrawal D et al., who studied subclinical peripheral neuropathy in chronic obstructive pulmonary disease patient [11]. Smoking causes vasoconstriction and damages blood vessels by atherosclerosis, plaque formation etc. As a result the blood supply and amount of oxygen, delivery to the nerve fibers decreases. Smoking also increases the level of cholesterol in the circulating blood stream which predisposes to the atherosclerosis [12]. The initial change which occurs as a result of smoking is constriction of microvasculature. Such microvascular function impairment occurs early in smoking.

Carbon monoxide released during smoking also damages tunica intima of blood vessels and endothelial cells, which further leads to deposition of fats in the vessel walls [13]. The layer of myelin around the axon is essential for the normal functioning of the nervous system [14]. During the initial period, smoking brings about subclinical changes in the myelin sheath that finally progresses into demyelination [15]. Due to the demyelination, nerve conduction blocks and the conduction velocity decreases [16]. Besides, the carboxyhaemoglobin formed in blood of smokers also decreases nerve conduction [17].

In the present study, there were more statistically significant changes in SNCV, this may be due to the fact that sensory nerves are thinner and are having shorter internodal distances. As a result, the thinner nerves are early affected than the thicker nerves by any damage. Hence, the sensory nerves are more affected than the motor nerve [18]. This may be due to fact that smoking causes decrease in conduction velocity by generating free radicals, by increasing the level of cholesterol.

CONCLUSION

On assessment of peripheral neuropathy through sensory nerve conduction study, it was seen that conduction velocity was decreased in smokers as compared to non smokers, showing the involvement of sensory nerves in smokers.

These results make a strong foundation for future neuropathic changes in apparently healthy adult male smokers as observed in different studies in Chronic Obstructive Pulmonary Disease (COPD) patients. Further studies are needed to confirm the findings with larger sample size.

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