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A Comparative Study of Nerve Conduction Velocity Among Alcoholics and Non-Alcoholics

Anita Pal[1], Vikas Jain[2], Shrishty Tomar[1], Gaurav Jain[3], A S Rajput[2], Yashvardhan Raghuvanshi[1]

[1]Postgraduate Student, Department of Physiology, Gajra Raja Medical College Gwalior, Madhya Pradesh, India.
[2]Professor, Department of Physiology Gajra Raja Medical College Gwalior, Madhya Pradesh, India.
[3]Associate Professor, Department of Physiology Gajra Raja Medical College Gwalior, Madhya Pradesh, India.

Abstract

Background: Alcoholic Polyneuropathy has been reported to be present in 13%–66% of chronic alcoholics. It has a slow, progressive onset over months to years and it always affects the lower extremity more than the upper extremity and begins distally. A predominant sensory type with axonal type more predominant than demyelination peripheral neuropathy was seen in alcoholic cirrhosis shown in several studies. Montfort R et al. (1995) have found the dose-related toxic effect of alcohol causing autonomic and peripheral neuropathy. To compared the nerve conduction velocity among Alcoholics and non- alcoholics. **Material and Methods:** In our study we have taken 30 cases with a history of more than 10 years of intake of alcohol and 30 controls from age group 30 -60 years. A computerized RMS EMG EP, Mark-II, machine will be used in the study. Filters will be set at 2 Hz AND 5 kHz for motor studies and at 20 HZ and 3 kHz for sensory studies. Study design- A case–control comparative study. Statistical analysis- Unpaired student T-test, regression, and analysis. p-value<.005 will be significant **Results:** After comparing the data gathered by applying an unpaired t-test Our study showed a decreased motor nerve conduction velocity and as well sensory nerve conduction velocity of upper and lower limb There is a statistically significant difference seen in the conduction velocity of alcoholics when compared with non-alcoholics groups of the motor and sensory nerves ($p<0.005$). No effect was observed with duration of alcohol intake on nerve conduction velocity. **Conclusion:** Alcohol-related peripheral neuropathy is a mixed axonal and demyelinating, length-dependent, sensorimotor neuropathy with dominant sensory features.

Keywords: NCS- Nerve conduction study, MNCV-motor nerve conduction velocity, and SNCV – sensory nerve conduction velocity.

Corresponding Author: Dr. Yashvardhan Raghvanshi, Post- graduate student, Department of Physiology, Gajra Raja Medical College Gwalior, Madhya Pradesh, India. Email: yashvardhanraghuvanshi23@gmail.com

Introduction

Alcohol is a psychoactive, dependence-producing substance, it causes a harmful effect on human health and comprises 5.9% of worldwide death. Chronic consumption of Alcohol for many years causes pancreatitis, neurological (neuropathy), psychological and liver diseases, etc.[1] the liver plays a major role in alcohol metabolism by oxidative and non-oxidative pathways.[2,3]

Alcoholic Polyneuropathy has been reported to be present in 13%–66% of chronic alcoholics.[4] It has a slow, progressive onset over months to years and it always affects the lower extremity more than the upper extremity and begins distally.[4-7]

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A predominant sensory type with axonal type more predominant than demyelination peripheral neuropathy was seen in alcoholic cirrhosis shown in several studies.[8-13]
Montfort R et al. (1995) have found the dose-related toxic effect of alcohol causing autonomic and peripheral neuropathy.[14]
Richard and Mayer (1966) studied that in the subjects with acute & chronic alcoholic intoxication, Motor Nerve Conduction Velocities (MNCV) and Sensory Nerve conduction Velocities (SNCV) decreased, and latency period increased in the distal nerves.[15]
Joshi and Patil (2020) concluded in their study that axonal loss is the main cause of polyneuropathy in alcoholics mainly affecting lower extremity nerves. They also found that polyneuropathy worsens with a higher duration of alcohol intake.[16]
Nerve Conduction Studies (NCS) are most commonly used in neurophysiological laboratories not only for the understanding of normal peripheral nerve structure and function but also about various diseases.[17]

Aims and Objectives
Aim: To compare the nerve conduction velocity among Alcoholics and non-alcoholics

Objectives
1. To determine and compare Peripheral Neuropathy via NCS in alcoholics
2. To determine the effect of the duration of alcohol intake on nerve conduction velocity.

Methodology
Study place: Department of Physiology, G. R. Medical College and J.A. Group of Hospitals, Gwalior (M.P.)
Sample Size: 30+30 =60
Study design: case-control comparative study type
Study population: chronic alcoholic subjects of 30-60 years (Duration > 10 years)
Informed consent will be taken from every subject and control.

Inclusion Criteria
1. Study group: Alcohol abusers male of 30-60 years who are consuming alcohol for more than 10 years
2. Control group: Age and sex-matched healthy subjects who are not consuming alcohol

Exclusion Criteria
1. Subjects with Diabetes mellitus
2. Subjects with Hypertension
3. Subjects having neuropathic disorders other than alcoholic
4. Subjects having liver diseases other than alcoholic liver disease
5. Subjects with kidney disorder

Nerve conduction study
A computerized RMS EMG EP, Mark-II, the machine will be used in the study. Filters will be set at 2 Hz AND 5 kHz for motor studies and at 20 HZ and 3 kHz for sensory studies. The sweep speed will be set at 5ms/division for MNCS and 2ms/division for SNCS, and 1-cm disc recording electrodes will be used for sensory studies. Supramaximal stimuli will be delivered to get adequate responses. Data will be collected for following paramters.
Motor nerve conduction velocity will be recorded in the median, ulnar, common peroneal and tibia nerves Sensory nerve conduction velocity will be recorded in median, ulnar nerves and sural nerve.

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RESULTS

Table 1: Anthropometric Data of Cases and Controls

Parameters	Cases (N =30) mean ±SD	Controls (N=30) mean ±SD	p-value
Age (Years)	40.46±8.21	37.13±5.06	0.06
Height (cm)	166.50±3.90	168.6±6.15	0.12
Weight (kg)	62.23±6.54	72.77±9.75	0.00
BMI (Kg/m^2)	22.40±2.06	24.83±2.52	0.00
SBP (mmHg)	134.73±5.74	121.43±6.98	0.01
DBP (mmHg)	85.5±4.38	81.93±3.98	0.00

Table 2: Compare the Motor Nerve Conduction Velocity Among Alcoholics and Non - Alcoholics

Motor Nerve conduction Velocity			
Parameters	**Alcoholics (Mean± SD)**	**Non-alcoholics (Mean± SD)**	**p- value**
Median nerve	50.10±1.14	60.79±1.10	0.000
Ulnar nerve	59.6±1.62	67.131±1.31	0.000
Tibial nerve	42.53±6.96	48.89±7.51	0.001
Peroneal nerve	43.86±5.60	48.04±5.45	0.000

Table 3: Compare the Sensory Nerve Conduction Velocity Among Alcoholics and Non - Alcoholics

Sensory Nerve conduction Velocity			
Sensory nerve	**Alcoholics (Mean ± SD)**	**Non -alcoholics (Mean ± SD)**	**p- value**
MEDIAN NERVE	50.43±4.96	60.254±7.48	0.000
ULNAR NERVE	47.827±9.00	54.85±9.54	0.005
SURAL NERVE	39.72±0.91	45.9±1.02	0.000

There is statistical significant differences seen in conduction velocity of alcoholics when compared with non-alcoholics groups of motor and sensory nerve ($p<0.005$).

DISCUSSION

Alcoholic neuropathy is a primary axonal neuropathy characterized by Wallerian degeneration of the axons and a reduction in the myelination of neural fibers. Controversy surrounds the pathogenic role of alcohol in the development of this neuropathy. Studies on rat models have indicated that alcohol does have a directly neurotoxic effect on the spinal cord and neuronal organelles. Acetaldehyde, a metabolite of ethanol has a direct neurotoxic effect. Ethanol also impairs axonal transport and disturbs cytoskeletal properties.[16]

Walsh and McLeod et al. (2015) studied peripheral Nerve conduction studies in alcoholics with neuropathy and found a slight reduction in maximal motor conduction velocity as compared to controls.[17]

Pillai Karthik Piramanayagam et.al (2016) studied to compare the motor nerve conduction velocities in both the upper and lower limbs of chronic alcoholics and nonalcoholic controls. They found a statistically significantly decrease ($p<0.001$) in motor nerve conduction velocity in chronic alcoholics as compared to non-alcoholic controls.[18]

Our study showed a decreased motor nerve conduction velocity and as well sensory nerve conduction velocity of upper and lower limb similar finding was seen in walsh and mc Leod, Pillai Karthik Piramnarayan, Richard and mayer study.

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In our study we found that a very high significantly decrease in MNCV of median. Ulnar nerve of upper limb and Tibial and Peroneal nerve of lower limb ($p<0.001$) and SNCV of median and ulnar nerve of upper limb and sural nerve of lower limb ($p<0.005$).

The mechanism behind alcoholic neuropathy is not well understood, but many studies have been proposed. These include activation of spinal cord microglia after chronic alcohol consumption,[19] activation of mGlu5 receptors in the spinal cord,[20] oxidative stress leading to free radical damage to nerves, the release of proinflammatory cytokines coupled with activation of protein kinase C,[21] involvement of extracellular signal-regulated kinases (ERKs) or classical MAP kinases,[22] involvement of the opioidergic and hypo-thalamo-pituitary-adrenal system.[23]

CONCLUSION

Our study concludes that alcohol intake is harmful to all body tissues including the peripheral nerves. alcohol intake appears to cause both axonal loss and demyelination.

Alcohol-related peripheral neuropathy is a mixed axonal and demyelinating, length-dependent, sensorimotor neuropathy with dominant sensory features.

No effect was observed with duration of alcohol intake on nerve conduction velocity.

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