A Comparative Study of Motor Nerve Conduction Velocities in Chronic Alcoholics and Non Alcoholics

Pillai Karthik Piramanayagam[1], Thenmozhi R[2]

ABSTRACT

Introduction: Alcohol is a dependence-producing substance and it has been widely used in the world. It can cause various number of diseases, social and economic nuisance to the community. Study was done to compare the motor nerve conduction velocities in both upper and lower limbs of chronic alcoholics and non-alcoholic controls.

Material and Methods: The study design was Case control study and carried out in the Department of Physiology. Nerve conduction studies were performed by using RMS EEG-32 Super Sec Recorders and Medicare System (Pvt) Ltd Chandigargh. 60 males candidates in the mean age group of 25-50 years in the chronic alcoholic group and 60 non-alcoholic group were selected. Nerve conduction studies done in the Motor nerves -Median, Ulnar, Tibial. The Motor nerve distal latency, Motor nerve action potential and Motor nerve conduction velocity were recorded.

Results: In Median, ulnar motor nerves of both upper limbs and Tibial motor nerve of both lower limbs were statistically very highly significant increase ($p<0.001$) in distal latency and statistically very high significantly decrease ($p<0.001$) in amplitude of compound nerve action potential and motor nerve conduction velocity in chronic alcoholics as compared to non-alcoholic controls.

Conclusion: The electrophysiological studies can be useful in the diagnosis of asymptomatic polyneuropathy in chronic alcoholics subjects. Nerve conduction studies can be a tool used to detect earlier neurological involvement before clinically present with complications.

Keywords: Alcoholics, electrophysiological technique, motor nerve conduction velocity,latency

INTRODUCTION

Alcohol is a psychoactive substance and its consumption is harmful to human health and it represents 5.9% of worldwide deaths. The alcohol causes more than two hundred diseases and various conditions such as cancer of the mouth, oesophagus and larynx, liver cirrhosis, pancreatitis, social consequences, such as road-traffic accidents, workplace-related problems, family and domestic problems, and interpersonal violence may bring to social attention.

Alcoholics may experiences burning feet and loss of pain sensation in feet all due to peripheral neuropathy, Distal, symmetrical polyneuropathy, radial nerve compression and wrist drop may occur. Myopathy associated with muscle cramps, tenderness, and weakness In long term sequence cerebral atrophy may occur.[1]

The disability and death occurs very early in life. Alcoholism is characterized by a strong urge to consume alcohol and an inability to limit the amount of drinking despite adverse consequences. The younger generation uses alcohol nowadays to decrease the mental tension and to remove social inhibitions. The wide ranging effects of long term consumption of alcohol on various systems in their functioning, either through change in general metabolism, nutritional deprivation, hepatic malfunction and psychosomatic or psychological disturbances.

Alcoholic neuropathy occurs due to high alcohol levels in the body, it causes the neuronal damage and it is frequent among chronic alcoholics, there are no uniform data about its prevalence. The concept of peripheral neuropathy and chronic alcoholism in mens has been known since past, but the relationship between both is to be determined. study of the disease of peripheral nerve test has been greatly developed by use of application of neurophysiological technique.[2-8]

By advocating the neurophysiological tests to detect subclinical, central and peripheral neuropathy at an early stage of disease would reduce the risk and may promote healthy life in chronic alcoholics. Study was done to compare the motor nerve conduction velocities in both upper limbs and both lower limbs in chronic alcoholics and non-alcoholic controls.

MATERIAL AND METHODS

Study design was Case control study and was carried out in the Department of Physiology after getting approval from the institutional ethical committee. Complete central nervous system examination was performed. Nerve conduction tests were performed by using RMS EEG-32 Super Sec Recorders and Medicare System (Pvt) Ltd Chandigargh. Study duration was one year**.** Candidates of non alcoholics controls and alcoholics without drinking alcohol were asked to come in Department of Physiology in the morning hours at 10.00 am for measurement of anthropological parameters and electrophysiological tests. All candidates were explained in full detail about research work. Written and informed consent were obtained from each subject at the starting of the research work.

Inclusion criteria - 120 male canditates of age group between 25-50 years was selected from general population.

Exclusion criteria – Patients with Tuberculosis, Diabetes, thyroid disorder, hypertension, smokers, stroke,anti- epileptics,

[1]Assistant Professor, [2]Associate Professor and HOD, Department of Physiology, Tirunelveli Government Medical College, Tirunelveli-627011, India

Corresponding author: Dr.Pillai Karthik Piramanayagam, Assisant Professor, Department of Physiology, Tirunelveli Government Medical College, Tirunelveli-627011

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anti-psychotics, antidepressant drugs, malignancy.

The candidates were divided into two groups:

Study group consisted of 60 chronic alcoholic men drinking alcohol for greater than 21 units/week for more than 5 years of duration without discontinuation and not having any clinical neuropatholigical symptoms.[3] Control group consisted of 60 good health people of same age group, socio economic cadre, Basal metabolic index and not drinking alcohol.

Brief details of candidates and the types, quantities, number of times consuming alcohol and duration of consumption of alcohol intake were recorded. Alcohol dependence screening in alcoholics and controls were done by using alcohol dependence data Questionnaire (SADD). It was used to detect Alcohol consumption in units and were quantified.

The study was conducted in Motor nerves of Median, Ulnar, Tibial of both upper and lower limbs and the Parameters selected were Motor distal latency (MDL), Motor nerve action potential (MNAP) and Motor nerve conduction velocity (MNCV).

STATISTICAL ANALYSIS

In our study simple "t" test and paired "t" test were done. The mean, standard deviation, standard error, 95% confidential interval were analysed. p value significance <0.001 obtained

RESULTS

There was statistically very highly significant increase ($p<0.001$) in latency of both upper and lower limbs motor nerves like median nerve, ulnar nerve and tibial nerve in chronic alcoholics as compared to controls. There was also statistically very highly significant decrease ($p<0.001$) in amplitude and motor nerve conduction velocity of both upper and lower limbs motor nerves like median nerve, ulnar nerve and tibial nerve in chronic alcoholics as compared to controls (Table 1-3).

DISCUSSION

Alcohol produces both immediate effects in the behavioural pattern and in thinking processes. It can damage to health over time after long term alcohol consumption.

In advanced stage of the disease the nerve damage may become permanent. Thiamine deficiency may persist.[9] Treatment with nutritional supplement in patients with chronic polyneuropathy may have symptoms.[10] Alcoholic polyneuropathy may affect the quality of life of the patients. The effects of the disease varies from mild discomfort to severe disability.[11]

Our study showed a decreased motor nerve conduction velocity which correlates with Maudsley and Mayer et al[4] who compared MNCV which was more prominent decrease in chronic alcoholics, with neuropathy as compared to those without neuropathy. In chronic alcoholics. There was also decreased in motor nerve conduction velocity in distal segments of the nerves in the upperlimb and minor decrease in sensory nerve conduction velocity as compared to controls.

Parameters Selected	Median motor nerve	Non alcoholics (n=60)	Chronic alcoholics (n=60)	P value
		Mean±SD	Mean±SD	
MDL (ms)	Rt	3.839±0.041	4.197±0.049	p<0.001
	Lt	3.825±0.094	4.168±0.050	p<0.001
CMAP (uv)	Rt	8.124±0.047	7.531±0.149	p<0.001
	Lt	8.130±0.018	7.530±0.196	p<0.001
MNCV (m/s)	Rt	55.273±0.522	51.254±0.053	p<0.001
	Lt	55.169±0.543	51.729±0.521	p<0.001
P<0.05 is significant, P<0.001 is highly significant				

Table-1: Comparison of motor distal Latency, motor nerve compound muscle potential, Nerve conduction velocity of median motor nerves in controls and chronic alcoholics

Parameters Selected	Ulnar motor nerve	Non alcoholics (n=60)	Chronic alcoholics (n=60)	P value
		Mean±SD	Mean±SD	
MDL (ms)	Rt	3.27±0.110	3.303±0.047	p<0.001
	Lt	3.183±0.118	3.256±0.042	p<0.001
CMAP (uv)	Rt	5.5 ±1.00	5.48 ±0.269	p<0.001
	Lt	5.68±0.006	5.63±0.781	p<0.001
MNCV (m/s)	Rt	61.52±0.856	58.504±0.400	p<0.001
	Lt	61.49±0.871	58.627±0.430	p<0.001
P<0.05 is significant, P<0.001 is highly significant				

Table-2: Comparison of distal Latency, compound muscle action potential, Nerve conduction velocity of Ulnar motor nerve

Parameters Selected	Tibial motor nerve	Non alcoholics (n=60)	Chronic alcoholics (n=60)	P value
		Mean±SD	Mean±SD	
MDL (ms)	Rt	5.31±0.152	6.17±0.090	p<0.001
	Lt	5.24±0.121	6.16±0.102	p<0.001
CMAP (uv)	Rt	4.63±0.631	3.3±0.110	p<0.001
	Lt	4.79±0.668	3.25±0.164	p<0.001
MNCV (m/s)	Rt	52.667±1.443	49.472±0.572	p<0.001
	Lt	52.56±0.985	50.051±0.193	p<0.001
P<0.05 is significant, P<0.001 is highly significant				

Table-3: Comparison of distal Latency, compound muscle action potential,Nerve conduction velocity of Tibial motor

Richard and Mayer M.D.,et al[5] studied in the subjects with acute and chronic alcoholics and found decrease in Motor nerve conduction velocities and sensory nerve conduction velocity. The latency period was prolonged.

Walsh and McLeod et al[6] studied peripheral Nerve conduction tests in chronic alcoholics with poly neuropathy and saw a slight reduction in maximal motor nerve conduction velocity as compared to non alcoholic controls. The amplitude of sensory nerve action potentials was reduced with a small increase in latency.

T.G.H.C Fernandis et al[7] assessed peripheral nerve conduction studies in normal healthy subjects and two 2 groups of chronic heavy alcoholic drinkers consuming two types of distilled alcohol and legal spirit and found both arm limb and legs motor and sensory nerve conduction velocity were significantly decreased in both group of alcoholics.

Dr. Shafiqe Ahmed et al[8] studied motor nerve conduction velocities and terminal latencies in ulnar and median nerve of upper extremity, and posterior tibial and common peroneal nerve of lower extremity in chronic alcoholics and normal healthy controls and found a decrease in Motor nerve conduction velocity and terminal latencies in alcoholics both with neuropathy and without neuropathy in all nerves of both upper and lower extremities.

E. B. Casey et al[9] studied nerve conduction studies in alcoholic subjects with minimal or no clinical evidence of peripheral neuropathy and found conduction velocity was decreased in patients with chronic neuropathy.

There are views of diversities regarding the histopathological changes in alcoholic neuropathy Mawdsley and Mayer[4] considered segment demyelination was the most probable lesion accounting for reduction, in MNCV, while Blackstock[2] were of opinion that axonal degeneration was the main pathological event. Laseelly found evidence of segmental demyelination secondary to axonal degeneration. Gioranne Tredici and Mario Ninazzi (1974) in their study showed typical aspect of chronic partial denervation due to wallerean like degeneration of fibers They found that the ratio between myelin component to myelinated axons was prolonged in pathological nerves despite the decrease in myelin, indicating more involvement of axonal part. The cause of alcoholic polyneuropathy is unknown. Thiamine deficiency and neural toxins has been considered as main factor for demyelination. The present study has shown that in a group of alcoholic patients without or with minimal clinical evidence of peripheral neuropathy, motor nerve conduction velocity diminished[7]

Coers and Hildebrand, 1965; Walsh and McLeod, 1970[6]; Blackstock et al., 1972)[2] found Reduction in amplitude of nerve action potentials with preservation of normal conduction velocity suggested that axonal degeneration is occurring.

Walsh and McLeod[6] found histological changes of axonal degeneration in sural nerves from alcoholic neuropathy. A common pattern of axonal degeneration is that changes start at and spread from the distal ends of the fibres. The pattern of abnormality demonstrated electro-physiologically in alcoholic neuropathy suggests that nerve fibres losses their function.

Mawdsley and Mayer[4] suggested that segmental demyelination, with associated reduction in velocity, might occur at an early stage of the disease, as originally suggested by Denny-Brown (1958). The slight reduction in maximal conduction velocity in both motor and sensory nerves in alcoholic neuropathy and in other neuropathies in which axonal degeneration is found could be explained by failure of conduction in the most rapidly conducting fibres, conduction continuing normally in the slower fibres.[4] Abnormalities seen in alcoholics could be attributed to a direct toxic effect of ethanol or its metabolites on peripheral and autonomic nerves. Deficit of B vitamins especially thiamine was thought to play a leading cause in the pathogenesis of alcoholic peripheral neuropathy.

Constant use of alcohol contributes to metabolic changes in the nerve cells and degeneration of the axial flux. Every axon begins to form from the most distal sections of the cell body whose integrity depends on the consistency of the streams. This tells why the longest part of axons are primary to be involved. As the disease progresses, the axonal flux becomes less and less efficient, and the degeneration begins to prolong to portions of the axons nearer to the cell body and is accompanied by destruction of the myeline sheaths. This is known as retrograde degeneration (Savoldi, 1995; Manzo and Costa, 1998).[12] The pathological cause of alcoholic polyneuropathy is still under controversy. While some have told their opinion that it results from nutritional deficiency, and especially from deficiency of vitamin Thiamine (B1), there is both clinical and experimental evidence of a direct toxic effect of alcohol on motor nerves

Limitations

This Study was a case control study and it need for Quantitative analysis. Complication occurs with the dose of alcohol, duration and abstinence of alcohol on Peripheral nerves may help to establish mathematical models for more detailed description of interaction between alcohol consumption and neurological dysfunction. More studies are recommended to see whether the neurological dysfunction in alcoholics, improvement with abstinence and lifestyle modification along with appropriate nutritional treatment to reduce the morbidity and mortality in alcoholics.

The electrophysiological tests like nerve conduction studies can be used to detect subclinical central and peripheral neuropathy at an early stage of alcohol intoxication. So implementation of suitable preventive measurement and therapy methodologies can be applied to decrease the complication of alcohol.[13]

CONCLUSION

The neural changes in alcoholic neuropathy, segment demyelination, reduction in number of neurons and axonal degeneration were the main cause for decrease in motor nerve conduction velocity. The reduction in myelin content and thiamine deficiency has been considered as main factor for demyelination.The direct toxic actions of alcohol have also been documented in the cause for nerve lesion.

Our study confirms the involvement of peripheral and central nervous systems in chronic alcoholics. The electrophysiological tests can be used to detect subclinical central and peripheral neuropathy at an early stage of disease.

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