

Electrophysiological Evaluation of Peripheral Neuropathy in Chronic Alcoholic Patients

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Electro diagnostic findings helps us to find out type of neuropathy and associated neurological conditions like radiculopathies etc. Knowledge of which will be useful for prognosis of disease , planning line of treatment and for further research. Nerve conduction studies were carried out in 55 alcoholic patients. Alcoholic patients were divided into three groups depending upon number of years , taking alcohol. Group 1) up to two year, Group 2) More than two years to four years, Group 3) More than four years to six years and more. Nerve conduction study was carried out as per standard method. Amplitudes of CMAPs and SNAPs were affected earlier than conduction velocities. lower extremity nerves were initially affected than upper extremity nerve. Sensory and motor nerves were equally affected. More was the duration of alcohol intake more was the deterioration in nerve conduction study. Axonal loss was the main cause of poly neuropathy mainly affecting lower extremity nerves. So further research is required to find out exact cause of changes and how much because of toxic effects of alcohol and how much because of vitamin and nutrition deficiencies and other factors associated with alcohol consumption.

Keywords: Alcoholic Patients; Electrophysiological Evaluation; Peripheral Neuropathy.

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, esophagus and larynx, liver cirrhosis, pancreatitis etc¹ India's annual alcohol intake increased by 38 per cent between 2010 and 2017 and alcohol consumed globally per year has risen by 70 per cent since 1990² Chronic consumption of alcohol leads to advent of peripheral neuropathy³

Polyneuropathy has been reported to be present in 13%–66% of chronic alcoholics⁴. Even in clinically evident polyneuropathy the prognosis often can be established on the basis of electrodiagnostic findings. Furthermore, the electromyographer can readily identify other primary or superimposed disorders, such as polyradiculopathy, that may be clinically indistinguishable from polyneuropathy . In present study patients consuming alcohol and clinically suspected or diagnosed as a peripheral neuropathy were evaluated. Hypothesis for present

research work is, alcohol damages peripheral nerves and more is the duration of alcohol intake more is the damage. So aim of present research work is to find out type of neuropathy, extent of damage and effects of long duration of alcohol intake on peripheral nerves with the help of nerve conduction studies. Knowledge of present research work will be useful for determination of prognosis and better management of neuropathy patients.

MATERIALS AND METHODS

It was a cross sectional, comparative study. Present research work was carried out in department of physiology KIMS. karad. Patients from Krishna hospital who were willing to participate in study were included in present study. Study duration was from Nov. 2018 to March. 2019. Institutional Ethics committee permission was taken. (KIMSDU/IEC/06/2018).

55 patients clinically suspected or diagnosed of peripheral neuropathy and taking alcohol which were referred to department of Physiology were investigated.

Inclusion Criteria for Controls

Apparently healthy individuals (non diabetic, Non Alcoholic) between the age of 40 years to 60 years willing to participate in the study. No history suggestive of exposure to heavy metals and neurotoxic industrial agents like, lead. Etc.

Inclusion Criteria for Alcoholic Patients

Patients between age of 40 years to 60 years willing to participate in study. Patients taking alcohol and Clinically diagnosed or suspected patients of polyneuropathy.

Nerve conduction study was also performed on apparently healthy 60 age matched controls who were non diabetic non alcoholic and not having any major illness. Alcoholic patients were divided into three groups depending upon number of years, taking alcohol. (Group A): up to Two years (Sample size 20). (Group B): More than two years to Four years (Sample size 20), (Group C) More than four years up to six years or more (Sample size 15). Average age of patients was 53 years and all were male patients. Average age of controls was 52 years, and all were male subjects.

Exclusion Criteria for Patients

Patients not Willing to participate in study, having any major illness or endocrinal disorder like diabetes mellitus or renal failure etc. Patients having history suggestive of exposure to heavy metals and neurotoxic industrial agents like, lead. Etc were excluded from the study.

Institutional ethical committee approval was taken for the study. Patients and subjects were informed the detailed procedure of nerve conduction study and written consent was taken. Electro diagnostic study included motor and sensory nerve conduction of Median, Ulnar Peroneal, Tibial, and sural nerves by conventional method⁴ For stimulation supra maximal strength was used. For recording sensory and motor nerve conduction, surface metal electrodes were used. For recording motor conduction, recording electrode was placed close to the motor point and reference electrode three centimeter distal to it. For median nerve stimulation, stimulus was given at wrist and at elbow near volar crease of brachial pulse. For recording motor conduction of Ulnar nerve, Abductor Digiti Minimi muscle was used. Stimulus was given at wrist and at elbow in cubital tunnel behind medial epicondyle. For ulnar nerve stimulation at elbow arm position was maintained at 135°^{5,6}. For motor conduction of Peroneal nerve, Extensor Digitorum Brevis (EDB) muscle and for motor conduction of Tibial nerve Adductor Hallucis (AH) muscles were selected. Reference electrode was placed three centimeter distal to recording electrode.

For Sensory conduction of median nerve surface recording electrode was placed three centimeter proximal to distal wrist crease on median nerve. For Sensory conduction of ulnar nerve surface recording electrode was placed three centimeter proximal to distal wrist crease on ulnar nerve. Reference electrode was placed three centimeter proximal to recording electrode. For sensory conduction of Sural nerve the surface recording electrode was placed midway between heel and lateral malleolus and reference electrode was placed three centimeter distal to recording electrode. For sensory nerve conduction study, twenty supramaximal stimuli were delivered and average was recorded. Ground electrode was placed between recording and stimulating electrodes.

For each motor nerve studied following parameters were measured:

- Distal motor latency (DML). milliseconds.(ms.)
- Compound muscle action potential amplitude (CMAP) . mill volt. (MV)
- Motor Nerve conduction velocity(MNCV) . Meters /Sec. (M/S)
- For each sensory nerve studied following parameters were measured:
- Distal sensory latency (DSL). milliseconds.(ms.)
- Sensory Nerve action potential . (SNAP) Microvolt's. (μ V)
- Sensory Nerve conduction velocity (SNCV) . Meters /Sec. (M/S)

During nerve conduction study, laboratory temperature was maintained between 21o C to 23oC. When skin temperature of limb was below 34oC, the limb was immersed in a warm water to correct the temperature. (4,7) For nerve conduction studies, Recorder and Medicare System (RMS) machine from Chandigarh (India) was used.

Statistical Analysis

Statistical analysis was performed using SPSS, version 20.0. Data was summarized into mean \pm S.D. One way ANOVA was used for multiple group comparisons followed by post hoc Tukey's test. $P > 0.05$ Not significant (NS.), $P < 0.05$ was said to be significant. *, $P < 0.01$ highly significant **, $P < 0.001$ very highly significant ***

RESULTS AND DISCUSSION

In present study all were male patients. In present study when two year alcohol intake group (A), was compared with control group , no significant differences were observed for sensory and motor nerve conduction velocities, and Distal motor latencies , distal sensory latencies of upper extremity and lower extremity nerves. ($P > 0.05$ not. Sig)). However When four year alcohol intake group (B) , was compared with control group , significant decrease in Amplitudes of CMAPs and SNAPs and significant increase in DSLs were observed for lower extremity nerves. When six year alcohol intake group (C) was compared with control , no significant differences were observed for sensory and motor nerve conduction velocities of up.(Table 1)per limbs. However significant decrease in SNAPs were observed in upper extremity sensory nerves. Very Significant decrease in sensory and motor nerve conduction velocities, Amplitudes of CMAPs and SNAPs were very significantly decreased in lower extremity nerves.($P < .001$). (Table 3) Distal motor latencies , distal sensory latencies of lower limb nerves were very significantly increased.($P < .001$). Our study showed that lower extremity nerves were initially affected than upper extremity nerves.(Table 1, 2 ,3) Our study showed that the amplitudes of CMAPs and SNAP s were significantly reduced

Table 1. Mean and SD. Values of DMLs, CMAPs, and MNCVs of upper extremity nerves

| Nerve | Groups | DML.(ms.) | CMAP(MV) | NCV. (M/S) |
|-----------------------|----------------------------|----------------|-----------------|------------------|
| | | Mean & SD. | Mean & SD | Mean & SD |
| Median nerve. (Motor) | Control (N=60) | 2.88 \pm .41 | 15.89 \pm 3.1 | 57 \pm 5.4 |
| | two years alcoholic(N=20) | 2.78 \pm .42 | 14.19 \pm 5.2 | 57.11 \pm 4.6 |
| | four years alcoholic(N=20) | 2.22 \pm .41 | 14.12 \pm 4.4 | 56.18 \pm 3.71 |
| | six years alcoholic(N=15) | 2.61 \pm .35 | 14.14 \pm 3.4 | 56.11 \pm 4.11 |
| | ANOVA | | | |
| | F Value | 14.179 | 2.11 | 0.267 |
| | P Value | <0.0001 *** | 0.102 NS | 0.84 NS |
| ulnar nerve. (Motor) | Groups | DML.(ms.) | CMAP(MV) | NCV |
| | | Mean & Sd. | Mean & Sd. | Mean & Sd. |
| | control(N=60) | 2.82 \pm .41 | 15.82 \pm 2.8 | 57.88 \pm 5.88 |
| | two years alcoholic(N=20) | 2.79 \pm .11 | 14.87 \pm 3.1 | 56.11 \pm 5.11 |
| | four years alcoholic(N=20) | 2.66 \pm .14 | 14.11 \pm 3.1 | 55.14 \pm 5.12 |
| | six years alcoholic(N=15) | 2.67 \pm .11 | 14.00 \pm 3.2 | 55.11 \pm 5.14 |
| | ANOVA | | | |
| | F Value | 1.901 | 2.66 | 1.924 |
| | P Value | 0.1136 NS | 0.051 NS | 0.84 NS |

Table 2. Mean and SD. Values of DSL, SNAP, and SNCV of upper extremity nerves

| Groups | | DSL.(ms.) Mean & SD | SNAP. (µV) Mean & SD | NCV. (M/S) Mean & SD |
|----------------------------------|-----------------------------|-------------------------|-------------------------|--------------------------|
| Nerve Median nerve. (Sensory) | control(N=60) | 2.52 ±.34 | 12.68± 2.44 | 53.21±5.22 |
| | two years alcoholic(N=20) | 2.51±.33 | 11.98±1.44 | 54.21±3.18 |
| | four years alcoholic(N=20) | 2.49±.41 | 9.11±1.22 | 54.14±4.1 |
| | six years alcoholic(N=015) | 2.30±.11 | 7.14±1.33 | 53.14±5.11 |
| | ANOVA | | | |
| | F Value | 1.81 | 39.875 | 1.924 |
| | P Value | .149 NS | <.0001 *** | 0.772 NS |
| Nerve Ulnar nerve. (Sensory) | Control (N=60) | 2.71 ±.55 | 13.22± 2.58 | 53.27±4.44 |
| | two years alcoholic (N=20) | 2.68±.14 | 12.14±3.11 | 53.14±4.12 |
| | four years alcoholic (N=20) | 2.70±.17 | 8.12±1.14 | 53.44±3.12 |
| | six years alcoholic (N=15) | 2.80±.11 | 6.11±2.71 | 52.17±3.17 |
| | ANOVA | | | |
| | F Value | .271.846 NS | 44.19<0.0001 *** | .3450.794 NS |
| | P Value | | | |

Table 3. Mean and SD. Values of DMLs, DSLs, CMAPs ,SNAPs, MNCVs and SNCVs. of lower extremity nerves

| Groups | | DML.(ms.) Mean & SD | CMAP (MV) Mean & SD | NCV (M/S) Mean & SD |
|----------------------------|----------------------------|-------------------------|-------------------------|-------------------------|
| Peroneal nerve. (Motor) | control(N=60) | 3.22 ±.76 | 7.88± 2.2 | 47.55±3.56 |
| | two years alcoholic(N=20) | 3.14±.77 | 5.14±1.2 | 46.81±3.77 |
| | four years alcoholic(N=20) | 4.12±.17 | 4.18±1.4 | 46.11±3.17 |
| | six years alcoholic(N=15) | 5.21±.11 | 3.11±1.1 | 41.17±2.18 |
| | ANOVA | | | |
| | F Value | 45.89 | 41.36 | 14.34 |
| | P Value | <.0001 *** | <.0001 *** | <.0001 *** |
| Tibial nerve. (Motor) | control(N=60) | 3.64 ±.58 | 12.97± 4.21 | 47.12±3.56 |
| | two years alcoholic(N=20) | 3.54±.28 | 10.11±5.12 | 47.14±4.44 |
| | four years alcoholic(N=20) | 4.12±.44 | 8.12±2.31 | 46.14±3.11 |
| | six years alcoholic(N=15) | 5.12±.14 | 6.14±3.14 | 45.11±4.12 |
| | ANOVA | | | |
| | F Value | 43.80 | 17.03 | 1.405 |
| | P Value | <.0001*** | <.0001 *** | .24 NS |
| (Sensory) sural nerve. | control(N=60) | 2.22 ±.36 | 13.45± 3.11 | 50.21±3.12 |
| | two years alcoholic(N=20) | 2.11±.34 | 11.11±2.12 | 50.41±4.12 |
| | four years alcoholic(N=20) | 3.11±.25 | 10.11±3.11 | 50.17±3.17 |
| | six years alcoholic(N=15) | 4.14±.33 | 7.14±3.17 | 48.11±2.37 |
| | ANOVA | | | |
| | F Value | 159.84 | 24.34 | 1.904 |
| | P Value | <.0001 *** | <.0001 *** | .113 NS |

earlier than conduction velocities, Which indicates that alcoholic neuropathy is mainly because of axonal loss. Due to alcohol there is metabolic alterations in the nerve cells and degeneration of the axial flux. Each axon begins to degenerate from the most distal sections of the cell body whose integrity depends on the consistency of the streams. This explains why the longest axons are the first to be involved. Lower extremity nerves are longest so these nerves show earlier changes⁸

Sensory and motor nerves were equally affected. More is the duration of alcohol intake more is deterioration in nerve conduction study. (Table 3) When all parameter were compared within the two year, four year and six year alcohol intake groups DMLs and DSLs were significantly increased and NCVs were decreased. When fast conducting axons get degenerated then nerve conduction velocities decreases⁹ Self reported history of alcohol intake of all the patients for type of alcohol and frequency and quantity of alcohol intake was variable and unreliable so it is difficult to correlate. The cause for polyneuropathy in alcoholics is multifactorial, from both nutritional deficiencies and alcohol metabolism's direct toxic effects on neurons. History, physical examination and nerve conduction study can help to differential this condition from other forms of neuropathy. No specific lab test is available for diagnosis.^{10, 11, 12} Thiamine deficiency, direct toxicity of alcohol and Vitamin B12 deficiency is associated with alcoholic polyneuropathy. .^{2,7,8,13,14,15} So further research is required to find out exact effects of alcohol, toxic ingredients in alcohol, associated vitamin and nutrition deficiencies responsible for polyneuropathy. .

Implications

Knowledge of present research work will be useful to study prognosis of disease, planning treatment of alcoholic neuropathy patients. . Knowledge of present research work will be also useful for planning further research in alcoholic neuropathy to find out exact effects of alcohol, toxic ingredients in alcohol, associated vitamin and nutrition deficiencies.

Limitations

History of alcohol intake like exact duration of alcohol intake, type of alcohol intake is as per patients history .Effects of alcohol, toxic

ingredients in alcohol, associated vitamin and nutrition deficiencies are not separately studied.

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CONCLUSION

Based on the results observed in our study, we conclude that the axonal loss is the main cause of poly neuropathy mainly affecting lower extremity nerves .More is the duration of alcohol intake more is deterioration in nerve conduction.

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