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Original article:

Electrophysiological evaluation of peripheral neuropathy in alcoholic diabetic patients

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ABSTRACT

Background: The reported frequency of sensory and motor polyneuropathy in alcoholic patients varies from 12.5% to 29.6% .Very limited research literature is available for electrophysiological studies in diabetics taking alcohol. With this view, present study was planned to study nerve conduction velocity results of diabetic patients and alcoholic diabetic patients and to assess whether the diabetic patients taking alcohol are associated with more advanced changes of polyneuropathy compared to diabetic alone.

Material and Methods: Nerve conduction study was performed in control group (Group A, n:40) diabetic patients (Group B ,n:30) and diabetic patients taking alcohol(Group c, n:30). By standard method. Mean and SD Values were calculated for all the groups. One way ANOVA was used for multiple group comparisons followed by post hoc Tukey's test.

Results: When diabetic patients (Group B) and alcoholic diabetic patients (Group C) were compared to controls (Group A) there was very highly significant increase in DMLs and DSLs. very highly significant decrease in CMAPs and SNAPs, very highly significant decrease in MNCVs and SNCVs, ($p<.001$). When (Group B) and (Group C) were compared very significant changes were observed for most of the parameters of lower extremity nerves.($P<001$).

Conclusion-Diabetes mellitus more than five years is associated with electrophysiological evidence for sensory and motor generalized polyneuropathy. Alcoholic diabetic patients are associated with more advanced changes of polyneuropathy compared to diabetic only.

Key words: Electrophysiological Evaluation, peripheral neuropathy, Diabetic patients.

Introduction:

The number of people with diabetes has risen from 108 million in 1980 to 422 million in 2014 (1). In 2000, India (31.7 million) topped the world with the highest number of people with Diabetes mellitus and number raised to 69.2 million (8.7%) in 2015(1, 2) It is predicted that by 2030 DM may afflict up to 79.4 million individuals in India It has been estimated that 50% of diabetics suffer from peripheral neuropathy and 50% of these neuropathies are considered at least moderate in severity. Morbidities associated with diabetic neuropathy amount to more than $10 billion in the US. (3,4,5,6,7.8,). The prevalence of alcohol-related peripheral neuropathy has been estimated to occur in two-thirds of chronic alcoholics. The reported frequency of sensory and motor polyneuropathy in alcoholics varies from 12.5% to 29.6% (9,10). The rate of incidence of alcoholic polyneuropathy involving sensory and motor polyneuropathy varies from 10% to 50% of alcoholics depending on the subject selection and diagnostic criteria.

So diabetic and alcoholic polyneuropathy is severe health problem with increasing incidence .Lot of electrophysiological studies research

work is available in diabetic and alcoholic ployneuropathy.

Very limited research literature is available for electrophysiological studies in diabetics taking alcohol. With this view, present study was planned to study nerve conduction velocity results of diabetic patients and alcoholic diabetic patients and to assess whether the diabetic patients taking alcohol are associated with more advanced changes of polyneuropathy compared to diabetic alone.

Materials and methods

Sixty diabetic patients which were referred to Department of Electrophysiology of Krishna Institute of Medical Sciences, Karad were investigated. The patients were referred from Krishna hospital, other hospitals and nursing homes in Karad city as well as neighboring cities of Western Maharashtra. .Nerve conduction study was performed on apparently healthy age and sex matched controls who were non diabetic non alcoholic and not having any major illness.

Inclusion Criteria for controls: Apparently healthy individuals (non diabetic, non alcoholic) between age of 40 years to 60 years and willing to participate in study.

Inclusion Criteria for Diabetic patients:

Patients between age of 40 years to 60 years, suffering from diabetes more than five years. Patients between age of 40 years to 60 years suffering from diabetes more than five years and taking alcohol more than five years. and willing to participate in study.

Exclusion criteria for Diabetic patients:

Patients having any major illness or endocrinal disorder other than diabetes mellitus and not willing to participate in study.

All were male patients and Mean age was 52 years.

Institutional ethical committee approval was received for the study. Patients and subjects were informed the detailed procedure of nerve conduction study and written consent was obtained. Electro diagnostic study included motor and sensory nerve conduction of Median and Ulnar nerves of upper extremity and Peroneal , Tibial, and Sural nerves of lower extremity, by conventional method (11).

For recording sensory and motor nerve conduction, surface metal electrodes were used. For recording of motor nerve conduction, recording electrode was placed close to the motor point of the muscle and reference electrode 3cm distal to it. A supramaximal strength of stimulus was used.. For ulnar nerve stimulation at elbow arm position was maintained at 135^0(11,12, 13).For orthodromic sensory conduction of median nerve and ulnar nerve, surface recording electrodes were used. For stimulation ring electrodes were used. Twenty supramaximal stimuli were delivered and average was recorded. Ground electrode was placed between recording and stimulating electrodes. During nerve conduction study, laboratory temperature was maintained between 21^o C to 23^0C. When skin temperature of limb was below 34^0C, the limb was immersed in a warm water to correct the temperature (11, 12,13).

For each motor nerve studied following parameters were measured:

Distal motor latency (DML).

Compound muscle action potential (CMAP)
Motor Nerve conduction velocity (MNCV)
For each sensory nerve studied following parameters were measured:
Distal sensory latency (DSL)
Sensory nerve action potential (SNAP)
Sensory Nerve conduction velocity (SNCV). 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 ± S.D. One way ANOVA was used for multiple group comparisons followed by post hoc Tukey's test. $P<0.05$ was said to be significant.*, $P < 0.01$ highly significant **, $P < 0.001$ very highly significant ***. $P>0.05$ was considered as Not significant (NS.)

When Diabetic Patients (Group B) and alcoholic diabetic patients (Group C) were compared it was observed that no significant differences were observed for CMAPs of Median and MNCV and DML of ulnar nerve.
($p>.4$)

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When Diabetic Patients (Group B), and alcoholic diabetic patients (Group C) were compared it was observed Very significant differences were observed for most of the parameters studied for lower extremity Sensory and motor nerves.(p<.0001***).

Results:

Table 1: Motor nerve conduction study of Control (Group A), Diabetic Patients (Group B), and alcoholic diabetic patients (Group C)

Name of the Nerve	Control(A) (n = 40) Mean ± SD	Diabetic (n = 30)(B) Mean ± SD	Diabetic and alcoholic patient (n = 30)(C) Mean ± SD	ANOVA Test. F value	P Value.
Median DML(ms)	2.90 ± 0.4	4.02 ± 1.92	5.02 ± 1.11	25.40	<.0001 ** *
MNCV(m/sec)	58.50 ± 6.0	44.8 ± 8.2	40.11 ± 7.3	64.4	<.0001***
CMAP amplitude (mV)	15.76 ± 3.65	8.10 ± 4.2	6.11 ± 4.4	56.55	<.0001***
Ulnar DML(ms)	2.80 ± 0.3	4.2 ± 2.2	4.55 ± 1.11	16.63	<.0001***
MNCV(m/sec)	56.80 ± 5.33	42.56 ± 6.55	41.10 ± 6.88	74.04	<.0001***
CMAP amplitude (mV)	15.4 ± 2.6	7.11 ± 3.46	4.33 ± 2.66	140.55	<.0001***
Peroneal DML(ms)	3.23 ± 0.77	4.12 ± 1.4	5.11 ± 1.42	21.27	<.0001***
MNCV(m/sec)	47.28 ± 6.66	35.95 ± 7.01	32.14 ± 7.11	46.45	<.0001***
CMAP amplitude (mV)	10.44 ± 2.44	7.89 ± 1.70	4.99 ± 3.11	41.48	<.0001***
Tibial DML(ms)	3.75 ± 0.65	5.04 ± 1.66	6.01 ± 0.71	39.20	<.0001***
MNCV(m/sec)	46.10 ± 4.11	36.11± 6.11	31.11 ± 6.88	64.15	<.0001***
CMAP amplitude (mV)	13.70 ± 3.64	4.10 ± 2.9	3.11 ± 1.3	147.59	<.0001***

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Table 2: Sensory nerve conduction study of Control (Group A), Diabetic Patients (Group B), and Diabetic patients taking alcohol. (Group C)

Nerve name	Control (Group A) (n= 40) Mean ± SD	Diabetic (Group B) (n= 30) Mean ± SD	Diabetic & alcoholic (Group C) (n= 30) Mean ± SD	ANOVA Test. F value	P Value
Median Nerve DSL(ms)	2.52 ± 0.33	3.92 ± 0.89	4.21 ± 0.81	10.62	<.0001 ***
SNCV(m/sec)	52.44 ± 5.33	30.80 ± 6.11	28.11 ± 4.18	229.4	<.0001 ***
SNAP amplitude (mV)	12.11 ± 2.50	6.42 ± 3.3	5.41 ± 3.10	54.17	<.0001 ***
Ulnar Nerve DSL(ms)	2.73 ± 0.55	4.20 ± 0.12	5.11 ± 0.66	197.84	<.0001***
SNCV(m/sec)	52.90 ± 4.44	40.10 ± 4.95	38.11 ± 4.11	113.95	<.0001***
SNAP amplitude (mV)	14.34 ± 2.67	8.01 ± 2.99	5.11 ± 3.11	93.84	<.0001***
Sural Nerve DSL(ms)	2.25 ± 0.77	4.24 ± 0.48	5.12 ± 0.51	199.12	<.0001***
SNCV(m/sec)	49.23 ± 3.20	35.12 ± 3.91	30.14 ± 4.11	254.24	<.000***
SNAP amplitude (mV)	13.40 ± 3.40	6.88 ± 1.33	4.78 ± 0.55	136.84	<.0001***

Discussion:

In diabetes consistent hyperglycemia damages the microcirculation structure and function resulting in ischemia involving small blood vessels those supply nerves (Vasa nervosum). This results in axonal loss by Wallerian degeneration causing Distal Polyneuropathy (DPN) .To diagnose this condition nerve conduction studies with measurement of latency and velocity are commonly used as they are considered to be most sensitive, reliable, objective and non invasive means (14,15). In present study in diabetic patients (Group B) compared to controls (Group A) there were very highly significant increase in DMLs and DSLs., very highly significant decrease in CMAPs and SNAPs, very highly significant decrease in MNCVs and SNCVs, (p<.001). (Table 1& 2) These findings indicate that there is strong electrophysiological evidence for sensory and motor polyneuropathy in diabetic patients. A nerve conduction study is necessary to determine whether a neuropathy is primarily demyelinating, axonal, or both. Demyelinating neuropathy characteristically shows a reduction in conduction velocity and prolongation of distal latencies, whereas axonal neuropathy shows a reduction in amplitude of SNAP or CMAP earlier. Our results indicate mixed results. (16)When diabetic patients taking alcohol (Group C) was compared with control (Group A) all above mentioned changes were observed which again indicates that there is strong

electrophysiological evidence for sensory and motor polyneruopathy in diabetic patients taking alcohol .When diabetic patients (Group B) and diabetic patients taking alcohol(Group C) was compared all above mentioned parameters showed further deterioration in nerve conduction study , P** *(P<0.01). Thomas Julian et al. observed that in case of chronic alcoholic patients In general, the nerves in lower limbs were more affected than the upper limbs. Sensory and motor nerves were equally affected and reduced in amplitude was main finding.(17)David K McCulloch et al. observed that in case of diabetic patients the prevalence of symptomatic peripheral neuropathy was much higher who drank alcohol excessively.(18)Nicholas V. Emanuele et al. suggested that , long-term alcohol consumption in well-nourished diabetics results in increased blood sugar levels. Heavy alcohol consumption may increase a person's risk for polyneuropathy. (19)

In preset study also it was observed that mainly lower extremity nerves showed significant changes. Above mentioned findings indicates that diabetic patients taking alcohol have advanced sensory and motor polyneuropathy compared to diabetic alone. In diabetic patients taking alcohol, more deterioration was observed mainly to lower extremity nerves. Alcohol related neuropathy is associated with several risk factors, such as malnutrition, thiamine deficiency, direct toxicity of alcohol and a family history of alcoholism .

Vitamin B12 deficiency has been associated with significant neurological pathology, including peripheral neuropathy (20,21,22) There are other situations that are not a direct cause of nerve compression, but may increase the risk and may predispose the nerve to be compressed specially when the soft tissues are swollen like synovitis, pregnancy, hypothyroidism, diabetes or alcoholism .For compression neuropathies like carpel tunnel syndrome nerve conduction studies are very important(23)

There is some debate over whether the main cause is the direct toxic effect of alcohol itself or whether the disease is a result of alcoholism-related malnutrition . In experimental models of alcoholic polyneuropathy utilizing rats and monkeys has not resulted in convincing evidence that proper nutritional intake along with alcohol results in polyneuropathy (24). In addition, the consumption of alcohol may lead to the buildup of certain toxins in the body. For example, in the process of breaking down alcohol, the body produces acetaldehyde, which can accumulate to toxic levels in alcoholics. This suggests that there is a possibility ethanol (or its metabolites) may cause alcoholic polyneuropathy .

In diabetics taking alcohol we have got marked changes in all parameters studied in mainly in lower extremity nerves. It could be because of synergistic effect of all the various factors like altered metabolism due to diabetes, vitamin deficiencies in alcoholics, direct toxic effects of hyperglycemia and of breaking down products of alcohol. Further large sample size study and more duration of diabetes and alcohol consumption are required to come to final conclusion.

Conclusion:

Diabetes mellitus more than five years is associated with electrophysiological evidence for sensory and motor generalized polyneuropathy. Alcoholic diabetic patients are associated with more advanced changes of polyneuropathy compared to diabetic only.

Recommendations:

Further large scale study is required with large sample size, more duration of diabetes and more duration of alcohol intake

Findings of clinical and biochemical investigation of vitamin deficiencies should be correlated with findings of nerve conduction studies.

Study limitations:

History regarding exact quantity of alcohol intake, type of alcohol, and frequency of alcohol intake is not mentioned.

Associated vitamin deficiencies study was not done. Study is done on small sample size. So further large scale study with all above mentioned details if investigated it will help to come to precise conclusion.

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