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A COMPARISON OF NCS PARAMETERS OF SENSORY NERVES OF LEFT SIDE LIMB OF STUDY POPULATION WITH NORMAL REFERENCE RANGE VALUE

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ABSTRACT

The Tenth Revision of the International Classification of Diseases and Health Problems (ICD-10) defines the dependence syndrome as being a cluster of physiological, behavioural, and cognitive phenomena in which the use of a substance or a class of substances takes on a much higher priority for a given individual than other behaviours that once had greater value. Comparing the sensory nerves in both left and right shows that latency is prolonged and action potential is reduced in sural nerves but previous studies show that there was a decrease in amplitude of sensory nerve action potential in radial nerves. In our study patients, clinically there was no evidence of neuropathy but NCS revealed conduction velocity to be decreased in motor and distal sensory nerves where alcoholic patients who clinically had no evidence of neuropathy were investigated and found that there was a decrease in conduction velocity of motor and sensory nerves. The prevalence of alcoholic neuropathy in our study was 72%. The prevalence of neuropathy in chronic alcohol abuse range from 10 to 75%. In another study by Chopra and Tiwari et al the prevalence of alcohol neuropathy ranges from 25 to 66%.

Key words: Acetaldehyde dehydrogenase, Alcohol dehydrogenase, Alcohol use disorder identification test, Diagnostic and statistical manual of mental disorders, Fatty acid ethyl esters, Gamma amino butyric acid, International classification of diseases, Health problems.

INTRODUCTION

In this section presents introduction of this research work. Alcohol is metabolized by several pathways.Alcohol dehydrogenase (ADH) and aldehyde dehydrogenase (ALDH) are the most important enzymes involved in the metabolism. [1]Other enzymes that play a role in the alcohol metabolism are cytochrome P450 2E1 (CYP2E1) and catalase. Large amounts of alcohol consumption is metabolized by CYP2E1 and catalase metabolizes a small fraction of alcohol. Fatty acid ethyl esters (FAEEs) also help in eliminating alcohol.[2]

Genetics behind metabolism

Certain amount of alcohol can only be metabolised regardless of how much a person consumes. Factors responsible for alcohol metabolism that varies with every individual are:[3]

1) liver size
2) body mass

Research proves that there are variations of the ADH and ALDH enzymes in every individual. For example, a fast ADH enzyme or a slow ALDH enzyme results in toxic acetaldehyde to build up in the body that finally results in alcohol dependence [4]. In this paper presents section 2 of this paper explains the detail on the related works. In section 3 presents the materials and

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methods adopted and section 4 presents the details of the experiments and discussions. Finally section 5 concludes the paper by sharing our inferences and future plans.

RELATED WORKS

In this section presents focuses the related works of this research work. The goals of alcohol dependence are achievement of abstinence, reduced frequency and relapse, health and psychosocial functioning improvement [5]. Controlled alcohol consumption as a first step in the1. process of treatment is followed but it's difficult to achieve reduction in alcohol consumption in severely2. dependent patients.[6] The difficulty is probably due to the3. developed addiction memory and impaired control of drinking. In the detoxification phase withdrawal symptoms varies with every individual.[7] Symptoms arise after 4-6 hours of abstinence and becomes predominant on the second day.[8] Serious complications seen are seizures, hallucinations, and delirium.[9] Severe withdrawal symptoms should be treated as an in patient basis for 5 to 7 days. Benzodiazepines helps to reduce central nervous system irritability.[10] Other treatment factors include supplementation of thiamine and intravenous fluids administration.[11] Anticonvulsants and antipsychotic agents are also used if corresponding manifestations occur.[12] Essential components of a comprehensive treatment program includes the psychosocial treatment modalities.[13] Adjunctive pharmacologic relapse prevention therapy is needed only for a small percentage of few alcohol dependent patients. Long term care is required for alcohol dependent patients. Course of alcohol dependence is defined by periods of abstinence and recurring periods of relapses.[14] If there is a relapse following a longer period of abstinence, intensified level of treatment should be considered and should not be considered as a complete failure of treatment. [15] Patients with co-morbid medical, psychiatric, family and social problems come under the category of poorest adherence to treatment.

MATERIALS AND METHODS

In this section presents the materials and methods of this research work.

Study Design: A Cross sectional study

CASE DEFINITION

Inclusion Criteria

Patient fulfilling the criteria of alcohol dependent syndrome without symptoms of neuropathy.
Age above 18 years
Patients willing to participate in the study

Exclusion Criteria

1. Patients suffering from neuropathy of other causes(infectious or non infectious)
2. Patients with symptoms of neuropathy.
3. Patients unwilling to participate in the study.

Sampling Frame: Patients attending at Coimbatore region hospitals, India

Sample Size: 100 cases

Data Collection

Alcohol dependent syndrome patients were selected based on CAGE criteria. In these patients diabetes mellitus and other possible causes for neuropathy was ruled out by history and clinical examination. All the patients were subjected to nerve conduction study were from the clinical settings of Sri Ramakrishna Multi-Specialty Hospitals, Coimbatore, Tamil Nadu, India.

RESULTS AND DISCUSSIONS

In this section focuses the results and discussions of this research work.

T**able 1. NCS parameters of the sensory nerves of the left side were compared to the normal reference range values**

NERVE	PARAMETERS			
		LATENCY	CMAP	NCV
MEDIAN	Patient	2.34	58.26	55.08
	Normal	3.7	12.81	43.76
	P value	0.373	**0.001**	1
ULNAR	Patient	2.13	42.01	50.88
	Normal	3.5	9.32	43.33
	P value	0.410	1	1
SURAL	Patient	2.1	12.70	51.64
	Normal	4.2	7.69	42.22
	P value	**0.0049**	**0.002**	1

Table 2. Distribution Of Neuropathy In Asyptomatic Alcohol Dependance Syndrome Patients

ABNORMAL NCS	72%
NORMAL NCS	28%

Table 3. Age and Outcome Correlation

AGE GROUP	NEUROPATHY	
	ABNORMAL N(%)	NORMAL N (%)
30-40	53%	47%
41-50	79%	21%
51-60	100%	0%
>60	50%	50%

Table 4. Liner Correlation for Age Group and Neuropathy

Cut off value	43
Area under the curve	0.677
95% CI	0.576 to 0.767
Significance level P (Area= 0.5)	0.0071

A statistically significant correlation was established in the latency and action potential of the sensory sural nerve studied on the left sided limb. Comparing the sensory nerves in both left and right shows that latency is prolonged and action potential is reduced in sural nerves (Table 1). Based on nerve conduction study 72% of asymptomatic patients had evidence of neuropathy. Electrophysiological evaluation was normal in 28% of the study population (Table 2). Statistically significant correlation was established between age and neuropathy with a $p<0.001$ in the study population. Among the people in age group 40-60 years, 50% had neuropathy. As age increases, the probability of neuropathy also increases. Age group of more than 60 years, the presence and absence of neuropathy was 4% respectively in each group.There was positive correlation between age and development of neuropathy with a p value < 0.001 (Table 3).

In chronic alcohol consumers, 43 years of agewas taken as the cut off point for developing neuropathy with a sensitivity of 72.2% and a specificity of 78.6% (Table 4). A significant p value= 0071 was established. In response to CAGE questionnaire, there was statistical significance between patients with neuropathy in our study. This is similar to the John Rebecca et al study where there was a statistical significance comparing CAGE questionnaire with presence of neuropathy.According to the previous studies most common electro diagnostic finding in patients with alcohol abuse is symmetrical axonal sensory motor neuropathy in the form of decreased amplitude, of sensory and compound motor action potential and decreased velocity.A statistically significant correlation of the motor nerve action potential was established in median, ulnar and common peroneal nerves.Comparing the motor nerve parameters in left and right shows that there is a siginificant reduction in action potential in median, ulnar and peroneal nerves.

CONCLUSION

Finally this work concludes, A statistical significance was found for correlation between units of alcohol consumption and development of neuropathy with a p value<0.001. Various studies done are controversial to the findings in our study. First study to be mentioned here is a study done by Montforte et al states that development of neuropathy does not correlate with age, units of alcohol or nutritional factor. Similarly Bhansali et al and John Rebecca et al in their studies also state that there was no significant difference noted between the groups with neuropathy and the total life time dose of alcohol consumption. Hence establishment of significant correlation between units of alcohol consumption and neuropathy is still controversial. Future studies or researches can give a clarity about the relationship between the units of alcohol and its adverse effects on the peripheral nerves.

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