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Original article

Study of clinical profile of peripheral neuropathy in chronic alcoholics

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Abstract

Introduction: Axonal neuropathy with decreased densities of nerve fibres is the most likely diagnosis based on electrophysiological and pathological data. However, in individuals with a lengthy history of neuropathic complaints and substantial axonal sprouting, tiny and unmyelinated fibre densities were more severely diminished than big and myelinated fibre densities.

Materials and methods: The present cross sectional study was conducted in the Department of Medicine, Dr. D. Y. Patil Medical College, Hospital and Research Centre, Pune, Maharashtra, from August 2019 to September 2021. Total 100 chronic alcoholic heavy drinkers were chosen after a detailed history and clinical examination. The study protocol was reviewed by the concerned Ethical Committee and was granted ethical clearance.

Results: After explaining the purpose and details of the study, a written informed consent was obtained from the patients. Out of 100 patients 44 patients with duration of exposure to alcohol was >15 years had highest UNES score of 33.6818 followed by 5-15 years of exposure had UNES score 28.0952 and those with <5 years of exposure had mean UNES score of 21.7857. On One-way analysis of variance statistically significant association was observed between duration of alcohol consumption and UENS score (p=0.001).

Conclusion: The present study concluded that a longer duration of alcohol use as well as increased age were shown to be related with a more severe type of peripheral neuropathy.

Keywords: Peripheral neuropathy, chronic alcoholics

Introduction:

Axonal neuropathy with decreased densities of nerve fibres is the most likely diagnosis based on electrophysiological and pathological data.[1, 2] However, in individuals with a lengthy history of neuropathic complaints and substantial axonal sprouting, tiny and unmyelinated fibre densities were more severely diminished than big and myelinated fibre densities.[3] Subperineurial oedema is more common in thiamine deficiency neuropathy, while segmental de/remyelination resulting from enlargement of successive nodes of Ranvier is more common in alcoholic neuropathy.[4]

It is common for a number of tests to be conducted in order to provide an accurate diagnosis of ALN. Esophagogastroduodenoscopy and X-rays of the gastrointestinal system may also be necessary if a patient vomits and suffers from nausea for no apparent cause. ALN may be diagnosed with the use of electromyography and nerve conduction testing. This procedure may be necessary in certain instances. During a neurological exam, the patient's sensory abilities and reflexes may be assessed. [5]

As a preventable illness, it's ironic that we need to take a hard look at this growing and dangerous threat. Hence the present study was undertaken with the purpose to study the clinical profile of patients diagnosed with alcoholic peripheral neuropathy as well as tried to correlate the duration of alcohol consumption and assess the nerve conduction velocity among them.

Materials and methods:

The present cross sectional study was conducted in the Department of Medicine, Dr. D. Y. Patil Medical College, Hospital and Research Centre, Pune, Maharashtra, from August 2019 to September 2021. Total 100 chronic alcoholic heavy drinkers were chosen after a detailed history and clinical examination. The study protocol was reviewed by the concerned Ethical Committee and was granted ethical clearance. After explaining the

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purpose and details of the study, a written informed consent was obtained from the patients.

Inclusion criteria

- Male subjects consuming 60 gm/day or more of ethanol for 5 years or more/ Male subjects consuming 15 drinks or more /week.
- Female subjects consuming 20-40gm/day or more for 5 years or more/ female subjects consuming 8 drinks or more/week

Exclusion criteria

- Patients with diabetes .
- Patients with chronic renal failure.
- Patients with Liver disease/LFT abnormalities other than low albumin.
- Patients with malignancy.
- Patients on drugs known to cause peripheral neuropathy.
- Patients with a family h/o inherited neuropathies.
- Patients with h/o exposure to heavy metals and toxins.
- Patients with h/o lumbar or cervical radiculopathy.
- Patients with nutritional deficiencies.
- Patients with collagen vascular diseases.
- Patients with hypothyrodism, dysproteinemias, amyloidosis and AIDS.

Results:

The overall mean age of the study population was 39.91 years. Majority of them belongs the age group of 41-45 years (33%) followed by 46-50 years (22%), 31-35 years and 36-40 years (18% each) and 25-30 years (9%). Positive family history of alcohol intake in the study population was observed in 38% of the patients.

Mean duration of alcohol consumption was highest (16.36 years) in the age group of 46-50 years followed by (15.81 years) in 41-45 years, (11.83 years) in 31-35 years, (11.50 years) in 36-40 years (5.88 years) in 25-30 years. On One-way analysis of variance the distribution was found statistically significant.

Table 1: Distribution of sensory symptoms in the study population

Sensory symptoms	Frequency	Percent
Pins & needles sensation of feet	81	81.0
Burning feet	53	53.0
Numbness of feet	38	38.0
Hyperalgesia of feet	28	28.0
Allodynia	16	16.0
Unsteadiness in darkness	11	11.0
Total	100	100.0

Major sensory symptom reported by the patients was Pins & needles sensation of feet (81%) followed by Burning feet (53%), Numbness of feet (38%), Hyperalgesia of feet (28%), Allodynia (16%) and Unsteadiness in darkness (11%).

Table 2: Distribution of Autonomic symptoms in the study population

Autonomic symptoms	Frequency	Percent
Erectile dysfunction	27	27.0
Sweating disturbances	17	17.0
Postural Giddiness	9	9.0
Bladder disturbances	3	3.0

Major autonomic symptoms reported by the patients were erectile dysfunction (27%), sweating disturbances (17%), postural giddiness (9%) and bladder disturbances (3%).

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Table 3: Distribution of Motor Weakness of Great Toe in the study population

Motor Weakness of Great Toe	Frequency	Percent
Normal	75	75.0
Weak	25	25.0
Total	100	100.0

Motor Weakness of Great Toe in the study population was observed in the 25% of the patients.

Table 4: DTR response in lower and upper limb in the study population

DTR Response	Lower Limb		Upper Limb	
	Frequency	Percent	Frequency	Percent
Absent	18	18.0	5	5.0
Normal	47	47.0	83	83.0
Sluggish	35	35.0	12	12.0
Total	100	100.0	100	100.0

DTR response in the lower and upper limb was found absent in (18% and 5%) and sluggish response was seen in (35% and 12%) of the study population

Table 5: Distribution of type of Neuropathy in the study population

Neuropathy	Frequency	Percent
Sensory Neuropathy	54	54.0
Sensory + Autonomic Neuropathy	21	21.0
Sensorimotor Neuropathy	19	19.0
Sensorimotor + Autonomic Neuropathy	6	6.0
Total	100	100.0

Major type of neuropathy observed was Sensory Neuropathy (54%) followed by Sensory + Autonomic Neuropathy (21%), Sensorimotor Neuropathy (19%) and Sensorimotor + Autonomic Neuropathy (6%).

Table 6: Association between duration of alcohol consumption and UENS score in the study population

Duration of alcohol consumption	N	UNES Score		p-value
		Mean	Std. Deviation	
<5	14	21.7857	3.37818	0.001 (Sig.)
5-15	42	28.0952	3.69449	
>15	44	33.6818	3.92827	
Total	100	29.6700	5.55424	

Test applied: One-way ANOVA

Out of 100 patients 44 patients with duration of exposure to alcohol was >15 years had highest UNES score of 33.6818 followed by 5-15 years of exposure had UNES score 28.0952 and those with <5 years of exposure had mean UNES score of 21.7857. On One-way analysis of variance statistically significant association was observed between duration of alcohol consumption and UENS score (p=0.001).

Discussion:

A strong association was found between age and the occurrence of alcoholic neuropathy. Subjective symptoms and signs rose dramatically with age. Because the frequency of alcohol misuse in women is lower in our area of the nation, all of the patients in our research were male. Vittadini G and colleagues found that more than 70 percent of their patients with alcohol-induced neuropathy were men.[6]

In our research, 38 of the 100 individuals with alcoholic neuropathy had a family history of alcohol consumption. People with a strong family history of alcohol misuse are more likely to develop alcoholicneuropathy, and the consequences of alcoholism and abuse tendencies are also more pronounced in these patients. Alcohol consumption and alcohol-related end organ damage, such as neuropathy, have been linked to a hereditary susceptibility in families previously studied.[7]

Mean duration of alcohol consumption was highest (16.36 years) in the age group of 46-50 years followed by (15.81 years) in 41-45 years, (11.83 years) in 31-35 years, (11.50 years) in 36-40 years (5.88 years) in 25-30 years. On One-way analysis of variance the distribution was found statistically significant (p=0.001). When Vittadini Get al. conducted an alcoholic neuropathy epidemiological investigation, they found a link between alcohol use and the development of neuropathy. Neuropathic symptoms may begin to emerge after only five years, but polyneuropathy takes at least ten years to develop.[6] In a study of 57 cases of alcoholic neuropathy, Thomas Zambelis et al.[i] discovered a link between alcoholic neuropathy and time spent drinking.

Alcoholic neuropathy is associated with a wide range of symptoms, including pins and needle feeling in the feet, burning feet, numbness in the lower limbs, hyperalgesia in the lower limbs, allodynia, and unsteadiness in the dark, among others. Neuropathy patients suffering from alcoholism have aberrant sensory symptoms such as burning sensations in the lower extremities, according to Koike H and colleagues.[8]

In our study the major autonomic symptoms reported by the patients were erectile dysfunction (27%), sweating disturbances (17%), postural giddiness (9%) and bladder disturbances (3%).

In the autonomic function tests, H reflex was observed in 43%, Heart Rate Variability in 30% and Sympathetic Skin response in 27%. During our investigation, we found that two individuals showed objective autonomic indications prior to the onset of subjective symptoms. Only one-fourth of the alcoholic neuropathy patients in our study reported autonomic symptoms, indicating that there is no simultaneous involvement of somatic and autonomic fibres in this disease. Patients with alcoholic neuropathy should be examined for subclinical autonomic neuropathy, even in the absence of subjective indications of autonomic dysfunction. Screening bedside autonomic function testing should be done in all patients with alcoholic neuropathy because autonomic impairment starts before symptoms and indications of peripheral neuropathy.

There was no association between the severity of alcoholic somatic neuropathy and the presence of autonomic neuropathy, as reported by Nicolosi C et al. in their investigation.[9] M.W. Agelinka et colleagues found that 40% of alcoholic neuropathy patients displayed aberrant HRV to deep inspiration and prolonged hand grip in the form of autonomic neuropathy.[10] A total of one patient in their research was found to have both autonomic and peripheral neuropathy. In their series, Roser Monforte et al. discovered autonomic neuropathy in 24.3% of patients, and they found a link between the length of alcohol exposure and the development of this condition . [9,10,11]

Conclusion:

The present study concluded that a longer duration of alcohol use as well as increased age were shown to be related with a more severe type of peripheral neuropathy.

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