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ASSESSMENT OF NEUROLOGICAL OUTCOMES OF ALCOHOL CONSUMPTION IN A TERTIARY CARE CENTRE

Dr. Krishnan Kutty[1], Dr. R. Aashish[2]

1. *Professor, Department of General Medicine, Sree Mookambika Institute of Medical Sciences, Kanyakumari, Tamil Nadu*
2. *Junior Resident, Department of General Medicine, Sree Mookambika Institute of Medical Sciences, Kanyakumari, Tamil Nadu*

***Corresponding Author – Dr. R. Aashish[2],** *Junior Resident, Department of General Medicine, Sree Mookambika Institute of Medical Sciences, Kanyakumari, Tamil Nadu*

ABSTRACT

Background: Alcohol consumption is a major public health concern with significant neurological implications. Chronic and excessive alcohol intake can lead to a variety of neurological disorders. These complications often go unrecognized until irreversible damage occurs. Early identification of alcohol-related neurological manifestations is essential for timely intervention. Tertiary care centres frequently encounter such cases, making them ideal settings to evaluate the clinical spectrum and burden of alcohol-induced neurological conditions.

Aims & Objectives: To evaluate the types and prevalence of neurological disorders and to correlate clinical findings with duration and quantity of alcohol intake.

Material & Method: A hospital-based cross-sectional observational study was conducted in the Department of Neurology at a tertiary care centre over a period of 12 months. A total of 90 patients with a documented history of chronic alcohol consumption (more than 5 years of regular use) were included. Patients underwent thorough clinical evaluation, neurological examination, and relevant investigations such as MRI, CT scan, nerve conduction studies, and routine laboratory tests.

Results: Out of 90 participants, 92.2% were male, with a mean age of 48.6 ± 9.4 years, indicating middle-aged men are most affected by alcohol-related complications. Most were daily drinkers, and nearly 60% had consumed alcohol for over 10 years. Peripheral neuropathy (46.7%) was the most common neurological finding, followed by cerebellar ataxia and seizures. MRI and nerve studies showed cerebellar atrophy and sensorimotor neuropathy. Longer alcohol use correlated with increased neurological burden. Non-neurological conditions, especially liver-related diseases like alcoholic liver disease (13.3%) and cirrhosis (11.1%), were also frequently observed. A statistically significant association was observed between duration of alcohol use and severity of neurological deficits ($p < 0.05$).

Conclusions: Chronic alcohol consumption is associated with a wide range of neurological complications, many of which are potentially preventable or reversible with early diagnosis and abstinence. The findings of this study underscore the need for increased awareness, early intervention, and regular neurological screening in individuals with long-term alcohol use, particularly in tertiary care settings where such patients frequently present.

Keywords: Alcohol, Hypoglycemia, Neurological manifestations, Peripheral neuropathy, Seizures

INTRODUCTION

Alcohol consumption, both acute and chronic, has far-reaching effects on various organ systems, with the nervous system being particularly vulnerable. The impact of alcohol on neurological health is multifaceted, ranging from acute intoxication to long-term structural and functional brain damage.[1]

Neurological manifestations associated with alcohol use may include peripheral neuropathy, cerebellar ataxia, cognitive deficits, Wernicke's encephalopathy, alcohol-related dementia, and epileptic seizures, among others. These manifestations may arise from the direct toxic effects of alcohol, nutritional deficiencies (notably thiamine and other B-complex vitamins), metabolic disturbances, or a combination of these factors.[2,3]

Chronic alcohol use is a leading cause of acquired peripheral neuropathy. Patients often present with symmetrical sensory deficits, distal muscle weakness, and diminished reflexes.[4] In addition to peripheral nerve involvement, the cerebellum is another common target, where chronic alcohol exposure leads to cerebellar degeneration, primarily affecting the anterior and superior vermis. Clinically, this results in gait ataxia and postural instability, often mistaken for other cerebellar disorders.[5]

Alcohol also plays a central role in the pathogenesis of Wernicke's encephalopathy, a medical emergency caused by thiamine deficiency. This syndrome is classically characterized by the triad of ophthalmoplegia, ataxia, and confusion, although this full triad is often not seen. If left untreated, Wernicke's encephalopathy may progress to Korsakoff's psychosis, marked by severe anterograde and retrograde amnesia.[6,7]

Furthermore, long-term alcohol use has been implicated in cortical atrophy and cognitive decline, contributing to alcohol-related dementia. Neuroimaging in such individuals may reveal cerebral and cerebellar atrophy, especially in the frontal lobes.[8] Seizures are also common in chronic alcohol users, particularly in the context of alcohol withdrawal, which may lead to generalized tonic-clonic seizures within 6 to 48 hours of cessation.[9]

Globally, alcohol use disorders represent a significant cause of disability and premature mortality. The burden is particularly high in developing countries like India, where social, cultural, and economic factors contribute to early initiation and sustained use. Despite this, the neurological complications of alcohol use are often underreported or misdiagnosed, especially in primary care settings. Patients may present late in the disease course, when neurological damage is advanced and less amenable to reversal.

Tertiary care centres play a pivotal role in the identification and management of alcohol-related neurological conditions, as they receive referrals of complex and advanced cases from various levels of the healthcare system. A better understanding of the spectrum of neurological disorders associated with alcohol use in such settings can aid in improving diagnosis, guiding appropriate investigations, and tailoring interventions for better outcomes.

AIMS AND OBJECTIVES

To evaluate the types and prevalence of neurological disorders and to correlate clinical findings with duration and quantity of alcohol intake.

MATERIAL & METHOD

This was a hospital-based, cross-sectional observational study conducted in the Department of General Medicine at Sree Mookambika Institute of Medical Sciences, Kulasekharam, a tertiary care teaching hospital located in the southern part of India. The study was carried out over a period of 10 months, from June 2024 to March 2025. All participants provided informed written consent prior to inclusion in the study.

A total of 90 patients were enrolled based on predefined inclusion and exclusion criteria. Patients aged 18 years and above, with a documented history of chronic alcohol consumption (defined as regular intake of alcohol for a minimum duration of five years), and presenting with symptoms suggestive of neurological dysfunction, were included in the study. The exclusion criteria comprised patients with pre-existing neurological disorders unrelated to alcohol (such as stroke, epilepsy, or neurodegenerative diseases), patients with concurrent infectious or metabolic conditions that could confound neurological findings (e.g., uncontrolled diabetes, chronic kidney disease, hepatic encephalopathy), and those unwilling to participate.

All included patients underwent a detailed clinical evaluation, which included a comprehensive history-taking focused on the pattern, duration, and quantity of alcohol consumption, as well as any history of withdrawal symptoms, seizures, or nutritional deficiencies. A thorough neurological examination was performed, assessing cranial nerve function, motor and sensory systems, reflexes, cerebellar signs, and cognitive status using appropriate screening tools such as the Mini-Mental State Examination (MMSE).

Relevant laboratory investigations were conducted in all participants, including complete blood count, liver function tests, renal function tests, serum electrolytes, blood glucose levels, and vitamin B12 and thiamine levels when clinically indicated. Neuroimaging in the form of computed tomography (CT) or magnetic resonance imaging (MRI) of the brain

was performed in patients with suspected central nervous system involvement, such as ataxia, cognitive impairment, or altered sensorium. Additionally, nerve conduction studies were carried out in patients presenting with peripheral neuropathy symptoms to assess the type and extent of nerve involvement.

The collected data were systematically recorded in a predesigned proforma and subsequently analyzed using appropriate statistical software. Descriptive statistics such as means, standard deviations, and percentages were used to summarize demographic and clinical data. The relationship between the duration or amount of alcohol intake and the type or severity of neurological manifestations was analyzed using chi-square tests and other relevant inferential statistics, with a p-value of <0.05 considered statistically significant.

RESULTS

All the 90 participants were males reflecting the higher prevalence of alcohol use among men in the region. The mean age of the study participants was 48.6 ± 9.4 years, with most patients falling within the 40–60 age group, highlighting middle age as the peak period of alcohol-related neurological complications.

The duration and frequency of alcohol intake were carefully assessed. Most patients 66(73.3%) were daily drinkers, while the remaining 24(26.7%) engaged in binge drinking. A significant proportion (59.9%) had consumed alcohol for over 10 years. (Table 1)

Duration of Alcohol Use	Number of Patients	Percentage (%)
5–10 years	36	40.0%
11–15 years	32	35.6%
>15 years	22	24.4%

Table 1: Duration of Alcohol Consumption

Peripheral neuropathy was the most common manifestation, followed by cerebellar ataxia and seizures. A subset of patients had overlapping neurological deficits. (Table 2)

Neurological Manifestation	Number of Patients	Percentage (%)
Peripheral Neuropathy	42	46.7%
Cerebellar Ataxia	26	28.9%
Wernicke’s Encephalopathy	10	11.1%
Alcohol-Related Dementia	8	8.9%
Seizures (Withdrawal-related)	19	21.1%
Multiple Neurological Symptoms	17	18.9%

Table 2: Spectrum of Neurological Manifestations

Neuroimaging revealed cerebellar and cortical atrophy in a significant number of patients. Nerve conduction studies confirmed sensorimotor neuropathy in the majority who were tested. (Table 3)

Investigation	Number of Patients	Percentage (%)
MRI Brain (n=60) – Cerebellar Atrophy	22	36.7%
MRI Brain – Cortical Atrophy	14	23.3%
CT Brain – Ventricular Dilatation	10	16.7%
Nerve Conduction Study (n=45) – Sensorimotor Neuropathy	38	84.4%

Table 3: Neuroimaging and Nerve Conduction Study Findings

There was a statistically significant association between longer duration of alcohol use and the incidence of neuropathy, cerebellar dysfunction, and Wernicke's encephalopathy, indicating that the neurological burden increases with chronicity of alcohol consumption. (Table 4)

Duration of Alcohol Use	Neuropathy (%)	Cerebellar Ataxia (%)	Wernicke's (%)
5–10 years	15 (41.6%)	8 (22.2%)	2 (5.6%)
11–15 years	17 (53.1%)	10 (31.3%)	4 (12.5%)
>15 years	10 (45.4%)	8 (36.4%)	4 (18.2%)

Table 4: Association Between Duration of Alcohol Use and Neurological Manifestations

Among the 90 chronic alcohol users, non-neurological manifestations were common, with liver-related conditions being the most prevalent—alcoholic liver disease (13.3%) and cirrhosis (11.1%). (Table 5)

Non-Neurological Manifestation	Number of Patients (n)	Percentage (%)
Alcoholic Liver Disease	12	13.3%
Alcoholic Liver Cirrhosis	10	11.1%
Hepatocellular Carcinoma	1	1.1%
Acid-Peptic Disease	6	6.7%
Acute Pancreatitis	3	3.3%

Pneumonia	6	6.7%
Pulmonary Tuberculosis	1	1.1%
Upper Gastrointestinal Bleeding	5	5.6%
Anaemia	7	7.8%
Septicemia / Septicemic Shock	5	5.6%

Table 5: Non-Neurological Manifestations

DISCUSSION

The demographic profile in the present study revealed an exclusively male cohort (100%), reflecting a pattern consistently observed in both Indian and global studies. This gender disparity is primarily attributed to cultural norms, social permissiveness, and economic factors that make alcohol use more prevalent among men. The mean age of the participants was 48.6 ± 9.4 years, with a significant concentration in the 40–60 age group, indicating that chronic alcohol-related neurological complications typically emerge in midlife, often after years of sustained consumption.

Supporting this Ghogare AS et al.[10] noted that a large proportion of their subjects were between 18–30 years (44.0%) and 31–40 years (40.0%), with only 16% above 40. Importantly, the duration of alcohol use in their cohort was <10 years in 43% and between 11–20 years in 40%, with significant associations between sociodemographic variables (such as age, marital status, and family background) and cognitive function scores including MMSE, BCRS, and BG II.

In the present study, a notable trend was the pattern of chronic alcohol use—73.3% were daily drinkers, and nearly 60% had been consuming alcohol for more than 10 years. This sustained exposure aligns with the known pathophysiology of alcohol-related neurotoxicity and systemic complications. The cumulative impact of alcohol use was evident, as neurological deficits increased with longer consumption duration, suggesting a clear dose-dependent effect.

Balaraman BG et al.[11] similarly reported that 38% of their patients consumed alcohol for less than 10 years, while 31% and 23% had durations of 10–15 and 15–20 years, respectively. Only a small fraction had consumed alcohol for over two decades. These findings reinforce the gradual and progressive nature of alcohol-induced neurological damage.

The most frequent neurological complication observed in this study was peripheral

neuropathy, present in 46.7% of cases. This is consistent with existing literature highlighting alcohol-related polyneuropathy as one of the earliest and most prevalent manifestations, largely driven by direct ethanol neurotoxicity and nutritional deficiencies (particularly thiamine and B-complex vitamins). Cerebellar ataxia followed, occurring in 28.9% of patients, indicative of selective vulnerability of the cerebellar vermis to ethanol-induced degeneration.

Wernicke's encephalopathy, a potentially reversible yet often underdiagnosed condition, was seen in 11.1% of the cohort. The classic triad—confusion, ataxia, and ophthalmoplegia—should prompt heightened clinical suspicion, especially in chronic alcohol users. Seizures, mostly related to alcohol withdrawal, were identified in 21.1% of patients, underscoring the neuronal hyperexcitability associated with abrupt cessation after prolonged use.

Neuroimaging played a crucial role in supporting clinical findings. MRI scans revealed cerebellar atrophy in 36.7% and cortical atrophy in 23.3% of patients, particularly among those with cognitive impairment and gait disturbances. CT imaging identified ventricular dilatation in 16.7% of cases. Nerve conduction studies further demonstrated sensorimotor axonal neuropathy in 84.4% of patients tested, reinforcing the widespread peripheral nerve involvement in chronic alcoholics.

A statistically significant association was established between the duration of alcohol consumption and the prevalence of peripheral neuropathy, cerebellar ataxia, and Wernicke's encephalopathy. Patients with over 15 years of alcohol use were more likely to exhibit these complications, highlighting the progressive nature of alcohol-induced neurodegeneration.

Balaraman BG et al.[11] similarly reported that peripheral neuropathy was the most common complication (52%), followed by encephalopathy (12%), myopathy (6%), cerebellar degeneration (8%), and stroke (2%).

Patil VC et al.[12] also observed various neurological and systemic complications in their cohort of 146 patients. Notably, 8.92% had ischemic stroke, 1.36% had intracranial hemorrhage, and 2.08% presented with cortical venous sinus thrombosis. Seizures were reported in 6.84%, and Wernicke-Korsakoff syndrome in 20.54%. Mortality was 8.01%, with 48% of deaths attributed to neurological complications, particularly in older patients and those with hypoglycemia and seizures, emphasizing the need for early detection and management of these conditions.

Chandra IP et al.[13] highlighted that insomnia was the most frequent acute neurological manifestation (60%), followed by alcohol withdrawal syndrome (46%). Chronic complications were dominated by peripheral neuropathy (28%) and cerebellar degeneration (16.7%), with a significant age-related correlation with cerebellar involvement.

Beyond neurological disorders, this study also identified several non-neurological complications. Alcoholic liver disease was observed in 13.3% and cirrhosis in 11.1% of patients. One patient had developed hepatocellular carcinoma. Other notable findings included acid-peptic disease (6.7%), upper GI bleeding (5.6%), pancreatitis, pneumonia, anemia, and septicemia, illustrating the multisystemic burden of chronic alcohol use.

These findings are consistent with the observations by Ray G et al.[14] who reported a rising trend of both direct (59.3%) and indirect (40.7%) alcohol-related complications. Their study documented significant rates of injuries (30.6%), gastrointestinal disorders (16.7%), infections (16.7%), neurovascular disease (8%), and chronic kidney disease (5.8%), reflecting the wide-ranging impact of alcohol on health.

Furthermore, Jacob JA et al.[15] observed that 83.4% of their sample exhibited an alcohol dependence pattern, with significant associations with marital discord, co-morbid medical illnesses, employment problems, and psychiatric comorbidities, particularly depression (42.3%). These psychosocial variables often compound the clinical picture and impede recovery.

CONCLUSION

This study highlights the wide spectrum of neurological and non-neurological complications associated with chronic alcohol consumption. The findings reveal that middle-aged males are predominantly affected, with peripheral neuropathy, cerebellar ataxia, and Wernicke’s encephalopathy being the most common neurological manifestations. A longer duration and higher frequency of alcohol use were significantly associated with increased neurological morbidity. Non-neurological complications, particularly liver-related disorders, were also prevalent. Early identification, comprehensive evaluation, and multidisciplinary management are essential to reduce the disease burden and improve patient outcomes.

REFERENCES

1. Dguzeh U, Haddad NC, Smith KT, Johnson JO, Doye AA, Gwathmey JK et al. Alcoholism: a multi-systemic cellular insult to organs. International journal of environmental research and public health. 2018 Jun;15(6):1083.

2. Ubhenin AE, Anura F, Idris RI, Sani H. Unveiling alcohol's modern health burden: A comprehensive exploration of biological repercussions. Sokoto Journal of Medical Laboratory Science. 2025 Jun 16;10(1):7-19.
3. Foster RK, Marriott HE. Alcohol consumption in the new millennium–weighing up the risks and benefits for our health. Nutrition Bulletin. 2006 Dec;31(4):286-331.
4. Paul P, Campbell G, Zekeridou A, Mauermann M, Naddaf E. Diagnosing peripheral neuropathy in patients with alcohol use disorder. InMayo Clinic Proceedings 2024 Aug 1 (Vol. 99, No. 8, pp. 1299-1305). Elsevier.
5. Hammoud N, Jimenez-Shahed J. Chronic neurologic effects of alcohol. Clinics in liver disease. 2019 Feb 1;23(1):141-55.
6. Thomson AD, Guerrini I, Marshall EJ. Wernicke's encephalopathy: role of thiamine. Pract Gastroenterol. 2009 Jun;33(6):21-30.
7. Chandrakumar A, Bhardwaj A, W't Jong G. Review of thiamine deficiency disorders: Wernicke encephalopathy and Korsakoff psychosis. Journal of basic and clinical physiology and pharmacology. 2019 Mar 1;30(2):153-62.
8. Gellersen HM, Guo CC, O'Callaghan C, Tan RH, Sami S, Hornberger M. Cerebellar atrophy in neurodegeneration—a meta-analysis. Journal of Neurology, Neurosurgery & Psychiatry. 2017 Sep 1;88(9):780-8.
9. Rogawski MA. Update on the neurobiology of alcohol withdrawal seizures. Epilepsy currents. 2005 Nov;5(6):225-30.
10. Ghogare AS, Saboo AV. A cross sectional study of cognitive impairment in patients of alcohol use disorder attending a tertiary health care center in Central India. Annals of Indian Psychiatry. 2019 Jul 1;3(2):155-60.
11. Balaraman BG, Ettiyan A, Ramasamy J, Kumaran AS. A clinical case study of neuropsychological complications of chronic alcoholism in coimbatore medical college and hospital. Journal of evolution of medical and dental sciences-jemds. 2017 Feb 2;6(10):799-803.
12. Patil VC, Galande C, Desai N, Patil S, Agrawal S. Neurological Manifestations of Alcohol Consumption at Tertiary Care Centre. Indian Journal of Forensic Medicine & Toxicology. 2014;8(1):124.
13. Chandra IP, Boppudi S, Charitha SS, Vyshnavi B, Keerthi N. A cross-sectional study on clinical profile of alcohol induced neurological manifestations. Age. 2019 May 13;20(30):30-40.

14. Ray G. Health and social burden of alcohol use disorders in an industrial population of India. International Journal of Community Medicine and Public Health. 2023 Apr;10(4):1561.
15. Jacob JA, Edward R, Kuruvilla A. Patterns and Correlates of Alcohol Use-A Retrospective Study in a Secondary Care Hospital Setting. Journal of Clinical & Diagnostic Research. 2021 Sep 1;15(9).