Original Article

Evaluation of Prevalence and Pattern of Peripheral Neuropathy among Patients with Liver Cirrhosis

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Abstract

Background: Peripheral neuropathy is a complication of liver cirrhosis. It contributes to morbidity and significantly decreases the quality of life of the patients. The prevalence and pattern vary with disease severity and etiology. The current study aimed to determine the prevalence and patterns of peripheral neuropathy in liver cirrhosis and to evaluate its association with clinical and biochemical findings. **Material and Methods:** This hospital-based cross-sectional study was done on 60 cases of liver cirrhosis confirmed by clinical, biochemical, and radiological evaluations. Complete clinical examination followed by neurological evaluation was done for each case. Nerve condition studies were done in all cases. The severity of cirrhosis was classified based on Child–Pugh score. An appropriate statistical test was used for analysis of the data. **Results:** Peripheral neuropathy was present in 56.7% of patients. The most common pattern was symmetrical distal polyneuropathy (82.4%). Axonal neuropathy was the predominant electrophysiological type (60%). Neuropathy prevalence increased significantly with worsening Child–Pugh class (A: 30%, B: 60%, C: 86.7%; p = 0.005). Alcoholic cirrhosis showed significantly higher prevalence (72.2%) compared to non-alcoholic causes (p = 0.004). Patients with neuropathy had higher age, longer disease duration, and higher Child–Pugh scores. **Conclusion:** Peripheral neuropathy is a frequent complication of liver cirrhosis, particularly in alcoholic and advanced disease. Early detection using clinical and electrophysiological assessment is essential for timely management.

Keywords: Liver Cirrhosis, Peripheral Neuropathy, Child–Pugh score, Sensory-Motor neuropathy.

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Introduction

Liver cirrhosis is an end-stage of chronic liver disease, which is progressive liver fibrosis with the distortion of liver parenchyma.[1] The prevalence of the condition is common, affecting millions of people across the world. The common etiological factors are chronic viral hepatitis (especially hepatitis B and C), alcohol related liver disease, non-alcoholic fatty liver disease (NAFLD), and autoimmune liver diseases.[2] In addition to the familiar liver findings, cirrhosis is now known to be a systemic disorder affecting multiple organ systems such as the nervous system, cardiovascular system and renal function.[3] Peripheral Neuropathy (PN) is one of the extrahepatic complications that is gaining clinical attention. It occurs due to damage or dysfunction of the peripheral nervous system. Clinically manifested as sensory disturbances, motor weakness, and autonomic dysfunctions. Several interrelated pathophysiological mechanisms can cause peripheral neuropathy in patients with cirrhosis. Chronic liver dysfunction causes an increase in neurotoxic molecules like ammonia, bile acids, and other metabolites that are typically filtered by the healthy liver.[4] In addition, nutrient deficiencies are also frequent in cirrhotic patients, especially B1 (thiamine), B6 (pyridoxine), B12 (cobalamin), and vitamin E, which occur because of decreased storage capacity in the liver, decreased food intake, and malabsorption.[5] Chronic alcohol use is a significant contributor to cirrhosis, and has been shown to have independent effects on peripheral nerves that are mediated through direct neurotoxic effects and thiamine deficiency.[6] The prevalence of peripheral neuropathy in patients with liver cirrhosis has been reported to range from 20% to 80% depending on the diagnostic criteria used, the severity of liver disease, and the population studied.[7] However, there appears to be a wide variation in standardized assessment protocols and a better understanding of the pattern of neuropathy in the patient population. In cirrhosis, the clinical picture of PN is most frequently represented by a symmetrical length-dependent sensorimotor polyneuropathy more severe in lower than upper extremities.[8] Sensory nervous symptoms such as tingling, burning, numbness, and painful paresthesia are common precursors for motor involvement. Autonomic neuropathy is less commonly evaluated, but may present with postural hypotension, gastrointestinal dysmotility,

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and sweating abnormalities, which also affect patient quality of life in these patients.[9] Although peripheral neuropathy is a significant cause of morbidity in cirrhosis, it is underdiagnosed in routine practice. The insidious and subtle onset of symptoms, together with the overwhelming emphasis on managing hepatic complications, often causes suboptimal recognition of neurological involvement. Early recognition of PN is very important, as it may be partially reversible with the right interventions such as nutritional support, cessation of alcohol intake, and optimized management of liver disease.[10] Additionally, the occurrence and extent of neuropathy could be a predictor for disease progression and survival. Based on the clinical relevance of this complication, the need for systematic assessment of prevalence and trends of peripheral neuropathy in liver cirrhosis patients in different clinical contexts requires focused research. These studies can yield important insights into the natural history of neuropathy in cirrhosis, potential risk factors that place patients at risk for neurological involvement, and clues to rational screening and management. The present study aims to determine the prevalence and describe the profile of PN in patients with liver cirrhosis, adding to the evidence that already stresses the relevance of complete neurological examination in the management of patients with cirrhosis.

Materials and Methods

This cross-sectional observational study was conducted in the Department of General Medicine in a Tertiary Care teaching Hospital, Telangana. Institutional Ethical approval was obtained for the study. Written informed consent was obtained for the study after explaining the nature of the study and outcomes in the vernacular language.

Inclusion criteria

1. Patients with clinically, biochemically, and radiologically confirmed cases of liver cirrhosis.
2. Patients aged 18 years and above
3. Willing to sign informed consent voluntarily

Exclusion criteria

1. History of neuropathy due to diabetes mellitus, renal failure, or vitamin B12 deficiency
2. On medication with neurotoxic drugs such as antitubercular drugs without pyridoxine supplementation or chemotherapy agents.
3. Critically ill and uncooperative patients.

Sample size calculation: Diagnostic criteria for liver cirrhosis: The diagnosis of cirrhosis was based on clinical grounds and with the aid of some supporting laboratory tests. Radiological confirmation was achieved by ultrasonographic features of cirrhosis such as a decreased size or nodular liver, portal vein dilatation, splenomegaly, and ascites. In addition, endoscopic evidence of esophageal varices was also considered supportive for the diagnosis.

Following informed consent, a detailed clinical history was taken and included the duration and etiology of cirrhosis, alcohol consumption, and symptoms suggestive of peripheral neuropathy, including numbness, tingling, distal weakness, or a burning sensation. A complete general and neurological examination was performed with special emphasis on peripheral nervous system assessment, including sensory and motor functions, reflexes, vibration sense, and proprioception.

Assess the severity of liver disease: The severity of cirrhosis was determined by Child–Pugh classification, which includes patients in Class A, B, and C.

Neurological assessment: Clinical assessment of peripheral neuropathy and categorisation was done according to neurological findings. Nerve conduction studies (NCS) were undertaken when available to confirm and classify neuropathy as being of axonal, demyelinating, or mixed type. Patients who were not able to undergo electrophysiological testing due to edema or swelling of the feet were excluded from the NCS evaluation. All other eligible patients were subjected to electrophysiological testing except those mentioned above.

Statistical analysis: Data were entered into Microsoft Excel and analyzed using appropriate statistical software, SPSS version 25. Continuous variables were expressed as mean ± standard deviation, and categorical variables were expressed as frequencies and percentages. The Chi-square test was used for comparison of categorical variables, and a p-value <0.05 was considered statistically significant.

Results

Baseline demographic and clinical characteristics of the patients are presented in [Table 1]. Analysis of the table shows that a total of 60 cases were included in the study. The mean age of the study population was 52.4 ± 11.6 years. Males were in the majority with 70% of cases, while females were 30% of cases. The mean duration of cirrhosis was 34.2 ± 18.5 months. Alcohol was the most common etiology of cirrhosis, observed in 60.0%, followed by Hepatitis B in 13.3% of patients, Hepatitis C in 10.0%, and non-alcoholic steatohepatitis (NASH) in 11.7%. Cryptogenic or other causes accounted for 5.0%. Based on Child–Pugh classification, 33.3% belonged to Class A, 41.7% to Class B, and 25.0% to Class C, indicating that the majority had moderate to advanced liver disease.

Table 1: Baseline demographic and clinical characteristics of study population

Characteristic	Value
Age (years), mean SD	52.4 ± 11.6
Male, n (%)	42 (70.0%)
Female, n (%)	18 (30.0%)
Duration of cirrhosis (months), mean SD	34.2 ± 18.5
Etiology of cirrhosis, n (%)	
Alcohol	36 (60.0%)
Hepatitis B	8 (13.3%)
Hepatitis C	6 (10.0%)
Non-alcoholic steatohepatitis (NASH)	7 (11.7%)

Other / Cryptogenic Child-Pugh class, n (%)	3 (5.0%)
Class A	20 (33.3%)
Class B	25 (41.7%)
Class C	15 (25.0%)

Prevalence of peripheral neuropathy is given in [Table 2]. Peripheral neuropathy was present in 34 out of 60 patients, giving an overall prevalence of 56.7%, whereas 26 patients (43.3%) did not have evidence of neuropathy. This finding indicates that peripheral neuropathy is a common neurological complication among patients with liver cirrhosis.

Table 2: Prevalence of peripheral neuropathy in patients with liver cirrhosis

Parameter	Frequency	Percentage (%)
Peripheral neuropathy present	34	56.7
Peripheral neuropathy absent	26	43.3
Total	60	100

The pattern and type of peripheral neuropathy are presented in [Table 3]. Among the 34 patients with peripheral neuropathy, the majority demonstrated a symmetrical distal (length-dependent) pattern, seen in 28 patients (82.4%), while asymmetrical or multifocal neuropathy was observed in 6 patients (17.6%). The most commonly reported symptom was numbness, present in 30 patients (88.2%), followed by tingling or paresthesia in 28 patients (82.4%). Burning sensation was reported by 22 patients (64.7%), and distal muscle weakness was noted in 16 patients (47.1%). Nerve conduction studies were performed in 30 patients after excluding 4 patients with severe pedal edema that interfered with electrophysiological testing. Axonal neuropathy was the predominant electrophysiological pattern, identified in 18 patients (60.0%). Demyelinating neuropathy and mixed neuropathy were each observed in 6 patients (20.0%). These findings suggest that axonal degeneration is the most common neuropathic process in cirrhotic patients.

Table 3: Pattern and type of peripheral neuropathy (N = 34)

Parameter Distribution	Frequency	Percentage (%)
Symmetrical, distal (length-dependent)	28	82.4
Asymmetrical/multifocal	6	17.6
Symptoms reported		
Numbness	30	88.2
Tingling/paresthesia	28	82.4
Burning sensation	22	64.7
Distal weakness	16	47.1
Nerve conduction study (NCS) findings (n =30) Axonal type	18	60
Demyelinating type	6	20
Mixed type	6	20
Note: NCS was performed in 30 patients (excluding 4 with severe pedal edema).		

Association between severity of cirrhosis and peripheral neuropathy is given in Table 4. The prevalence of peripheral neuropathy increased progressively with worsening Child–Pugh class. Neuropathy was present in 6 out of 20 patients (30.0%) in Child–Pugh Class A, 15 out of 25 patients (60.0%) in Class B, and 13 out of 15 patients (86.7%) in Class C. Statistical analysis demonstrated a significant association between higher Child–Pugh class and the presence of peripheral neuropathy (Chi-square = 10.54, df = 2, p = 0.005). This indicates that increasing severity of liver dysfunction is strongly associated with a higher risk of neuropathy.

Table 4: Association between severity of cirrhosis (Child-Pugh class) and peripheral neuropathy

Child-Pugh class	Neuropathy present (n= 34)	Neuropathy absent (n = 26)	Total	Prevalence Within class (%)
Class A	6	14	20	30.0
Class B	15	10	25	60.0
Class C	13	2	15	86.7
Total	34	26	60	56.7
Chi-square test: X2 =10.54, df = 2., p = 0.005				

Association between etiology of cirrhosis and peripheral neuropathy is presented in [Table 5]. Peripheral neuropathy was most prevalent among patients with alcoholic cirrhosis, where 26 out of 36 patients (72.2%) were affected. Lower prevalence rates were observed in Hepatitis B (37.5%), Hepatitis C (33.3%), NASH (28.6%), and cryptogenic cirrhosis (33.3%). Comparison between alcoholic and non-alcoholic cirrhosis revealed a statistically significantly higher prevalence of neuropathy in alcoholic cirrhosis (Chi-square = 8.14, df = 1, p = 0.004). This finding suggests that alcohol-related liver disease has a stronger association with peripheral nerve involvement.

Table 5: Association between etiology of cirrhosis and peripheral neuropathy

Etiology	Neuropathy present (n)	Total patients in etiology	Prevalence (%)
Alcohol	26	36	72.2
Hepatitis B	3	8	37.5
Hepatitis C	2	6	33.3
NASH	1	7	28.6
Other / Cryptogenic	1	3	33.3
Chi-square test (alcoholic vs. non-alcoholic combined): X2 = 8.14, df = 1, p = 0.004			

Comparison of clinical features between patients with and without peripheral neuropathy is depicted in [Table 6]. Analysis of the table showed that patients with peripheral neuropathy were significantly older than those without neuropathy (56.2 ± 10.1 years vs. 48.5 ± 12.4 years, p = 0.01). They also had a significantly longer duration of cirrhosis (38.5 ± 19.2 months vs. 28.9 ± 16.8 months, p = 0.04). The mean Child–Pugh score was significantly higher among patients with neuropathy compared to those without neuropathy (9.2 ± 2.1 vs. 6.8 ± 1.9, p < 0.001), indicating more severe liver disease in affected patients. Ascites was significantly more common in patients with neuropathy (70.6% vs. 38.5%, p = 0.01). Although esophageal varices were more frequently observed among neuropathy patients (82.4% vs. 61.5%), the association did not reach statistical significance (p = 0.07).

Table 6: Comparison of clinical features between patients with and without peripheral neuropathy

Feature	Neuropathy present (n=34)	Neuropathy absent (n=26)	p-value
Age (years), mean ± SD	56.2 ± 10.1	48.5 ± 12.4	0.01
Duration of cirrhosis (months), mean ± SD	38.5 ± 19.2	28.9 ± 16.8	0.04
Child-Pugh score, mean ± SD	9.2 ± 2.1	6.8 ± 1.9	<0.001
Presence of ascites, n (%)	24 (70.6%)	10 (38.5%)	0.01
Esophageal varices, n (%)	28 (82.4%)	16 (61.5%)	0.07

Discussion

The overall results of our study indicate that peripheral neuropathy was found in 56.7% of cases of liver cirrhosis. This highlights that peripheral neuropathy is a relatively common complication in chronic liver disease but is frequently underrecognized. The prevalence of cirrhosis-related neuropathy has been reported to be from 30% to 70%, which also varies with different populations studied and with different methods of diagnosis.[11] In the present study, alcoholic cirrhosis was the most frequent etiology and had a significantly higher prevalence of neuropathy (72.2%). This is in line with observations of previous studies showing chronic alcohol use is an independent neurotoxin and a significant cause of axonal peripheral neuropathy. Alcohol induced neuropathy is caused by direct toxicity to peripheral nerves and indirect alcohol-induced nutritional deficiencies, especially thiamine and other B-complex vitamins that are important for the metabolism of neurons.[12] However, the presence of neuropathy in the non-alcoholic cirrhosis group indicates that hepatic failure contributes to neuropathy beyond alcohol toxicity. Liver disease severity was associated with peripheral neuropathy and was higher in those with Liver Class C (86.7%) compared to those with Liver Class A (30%). This progressive increase in neuropathy associated with Child-Pugh scores shows that when liver failure worsens, neuropathic changes become more frequent in prevalence. These proposed mechanisms involve build-up of ammonia and other neurotoxic by-products, liver dysfunction in the process of detoxification, chronic systemic inflammation, and oxidative stress followed by axonal injury.[13] A similar result was found in previous work showing that patients with advanced cirrhosis have more severe neurophysiological abnormalities.[14] The common pattern of neuropathy observed was symmetrical distal polyneuropathy, seen in 82.4% of affected patients. This length-dependent pattern is typical of metabolic and toxic neuropathies, suggesting a systemic process of peripheral nerves, which is distal to proximal. The common symptoms were paresthesias, burning sensation, distal weakness and numbness, reflecting mainly sensory involvement, although motor involvement was relatively late. The results are similar to previous electrophysiological studies, which demonstrated sensory-predominant axonal neuropathy in cirrhosis patients.[15] The electrophysiology pattern most frequently seen was axonal neuropathy (60%) followed by demyelinating (20%) and mixed (20%). However, the predominance of axonal damage suggests that the neuropathy in cirrhosis is related to chronic metabolic and toxic injury, rather than primary demyelinating disease as reported by other studies.[16,17] Axonal degeneration is frequently observed during systemic diseases with chronic metabolic disorders and is usually irreversible unless early diagnosis and treatment.[18] Patients with peripheral neuropathy in this study were comparatively older, had more severe cirrhosis (Child–Pugh score) and longer disease duration than those without peripheral neuropathy. This indicates that the cumulative disease burden and the progressive development of liver disease play an important role in nerve injury. Additionally, ascites was more common in patients with neuropathy, further indicating that advanced portal hypertension and decompensated liver disease are associated with neurological complications. Neurophysiological abnormalities have been previously described in similar studies in which severity of liver disease was associated with abnormalities.[19] Finally, the results of this study highlight that peripheral neuropathy is a common complication of liver cirrhosis, especially in the advanced stages and those with alcoholic cirrhosis. Early clinical recognition and electrophysiological evaluation are important for timely diagnosis and management, which may help in improving quality

of life and preventing further neurological deterioration.

Conclusion

Peripheral neuropathy is a common neurological complication in patients with liver cirrhosis, with a prevalence of 56.7% in the present study. The condition is predominantly seen as a symmetrical distal sensory neuropathy, with axonal degeneration being the most frequent electrophysiological pattern. Alcoholic cirrhosis and advanced stages of liver disease (higher Child–Pugh class) are strongly associated with increased risk of neuropathy. The severity of liver dysfunction, longer disease duration, and older age further contribute to its occurrence. Early recognition through clinical evaluation and nerve conduction studies is important for timely diagnosis and management to improve patient outcomes and quality of life.

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Conflicts of interest

There are no conflicts of interest.

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