Original Article

Prevalence and Electrophysiological Patterns of Peripheral Neuropathy in Cirrhosis: An Etiology-Specific Cross-Sectional Study

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Abstract

Background: Peripheral neuropathy (PN) is a significant yet under-recognized neurological complication of chronic liver disease (CLD). Understanding its pattern across etiologies and severity strata can guide earlier recognition and management. **Material and Methods:** A cross-sectional study was conducted at a tertiary-care teaching hospital. A total of 120 consecutive adult patients with confirmed cirrhosis were evaluated clinically; nerve conduction studies (NCS) were performed to confirm and classify PN. Disease severity was categorized by Child-Pugh criteria, and between-group differences were analyzed using appropriate statistical tests. **Results:** We included 120 participants (male 88, 73.3%; female 32, 26.7%). The overall prevalence of PN was 60.8% (73). The predominant phenotype was pure sensory (27, 37.0%), followed by mixed sensorimotor (20, 27.4%) and pure motor (17, 23.3%). Prevalence increased significantly with Child-Pugh severity: Class A (3, 15.0%), Class B (45, 67.2%), and Class C (25, 75.8%) (P<0.001). Across etiologies, PN was highest in Hepatitis B (19, 65.5%), followed by cryptogenic (8, 61.5%) and alcoholic cirrhosis (44, 59.5%) (P=0.028). Purely axonal degeneration was the predominant electrophysiological pattern (95.9%). **Conclusion:** PN is highly prevalent in cirrhosis, manifesting primarily as an axonal sensory neuropathy. Its frequency increases significantly with hepatic decompensation and varies by viral and metabolic etiologies. Findings support routine electrophysiological screening in cirrhotic patients.

Keywords: Peripheral neuropathy; Cirrhosis; Nerve conduction studies; Child-Pugh score; Axonal degeneration.

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Introduction

Chronic liver disease (CLD) is a major global health burden. While its hepatic complications are well-documented, extrahepatic manifestations like peripheral neuropathy (PN) remain underdiagnosed in clinical practice. Historically, PN prevalence in cirrhosis ranged widely from 19% to 80% based on electrophysiological studies, but the condition often remains clinically silent until advanced nerve damage occurs.[1] The pathophysiology of CLD-associated PN is multifactorial, involving metabolic derangements, hyperammonemia, systemic inflammation, and viral-mediated immune mechanisms.[2] However, the exact relationship between PN phenotypes, specific CLD etiologies (such as alcoholic vs. viral hepatitis), and the degree of hepatic decompensation remains incompletely characterized.

We conducted a cross-sectional study to document the prevalence, electrophysiological patterns, and severity of PN in adults with varied cirrhosis etiologies. By systematically evaluating these parameters in a tertiary-care setting, this study aims to clarify the etiological and severity-based drivers of hepatic neuropathogenesis to refine screening strategies.

Materials and Methods

Study design and setting: A cross-sectional observational study was conducted at the neurophysiology unit in collaboration with the department of medicine of a tertiary-care teaching hospital. Consecutive sampling was used over a 24-month period.

Participants: Adults (18–75 years) with a confirmed diagnosis of hepatic cirrhosis (via ultrasonography, endoscopy, or biopsy) presenting for evaluation were included. Patients with diabetes mellitus, prior nutritional deficiencies, significant comorbid cardiac/renal/neurological diseases, active sepsis, or human immunodeficiency virus (HIV) were strictly excluded.

Sample size: The target sample size was 120, determined a-priori using a prevalence-based formula (assuming a projected peripheral neuropathy prevalence of 55%, with a 95% confidence interval and an allowable margin of error of 8%).

Data collection: A structured clinical and neurological examination recorded sensory, motor, and reflex abnormalities. Fasting laboratory investigations assessed hepatic synthetic function and viral serologies. Standardized nerve conduction studies (NCS) and electromyography (EMG) were performed

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using a 4-channel system.

Variables and outcomes: Primary outcomes included the prevalence of peripheral neuropathy (defined by abnormal NCS), pattern classification (axonal, demyelinating, or mixed), and distribution (sensorimotor, purely sensory, or purely motor). Secondary tabulations examined associations with cirrhosis etiology and severity (Child-Pugh classification).

Ethical considerations: Ethics approval and informed consent were obtained prior to recruitment. All participant identifiers have been masked for blinding in this submission, strictly adhering to the journal's double-blind peer-review policy and STROBE guidance.

Statistical analysis: Data were analyzed using SPSS v25. Descriptive statistics summarized counts and percentages. Between-group categorical associations were tested using Chi-square or Fisher's exact tests; continuous variables across groups were compared using one-way ANOVA. Significance was set at $P<0.05$.

Results

Participant characteristics: We included 120 cirrhotic patients (male 88, 73.3%; female 32, 26.7%). The largest age group was 41-45 years (46, 38.3%), followed by 51-60 years (31, 25.8%). The majority of the cohort resided in rural areas (111, 92.5%). Based on Child-Pugh criteria, most patients presented with moderate hepatic decompensation, classified as Class B (67, 55.8%), followed by Class C (33, 27.5%), and Class A (20, 16.7%) [Table 1 & Figure 1].

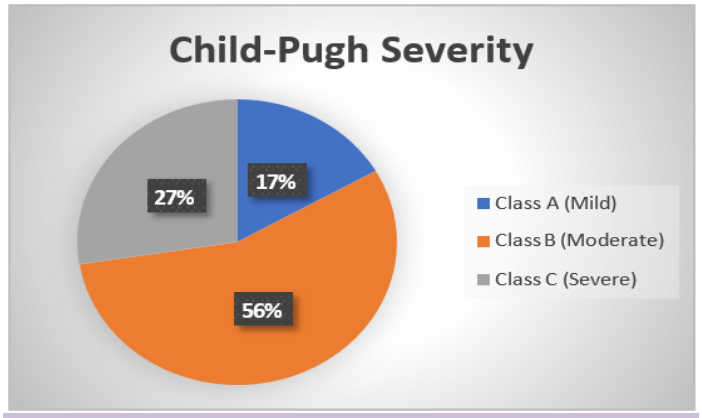

Figure 1: Severity of Child-Pugh.

Table 1: Demographic and Clinical Profile of Patients

Parameter	Count (n)	Percentage (%)
Gender		
Male	88	73.3
Female	32	26.7
Child-Pugh Severity		
Class A (Mild)	20	16.7
Class B (Moderate)	67	55.8
Class C (Severe)	33	27.5
Cirrhosis Etiology		
Alcoholic	74	61.7
Hepatitis B	29	24.2
Cryptogenic	13	10.8
Hepatitis C	4	3.3

Table 2: Peripheral Neuropathy Association with Disease Severity and Etiology

Variable	PN Present (n=73)	PN Absent (n=47)	P-value
Child-Pugh Class			
Class A (n=20)	3 (15.0%)	17 (85.0%)	<0.001
Class B (n=67)	45 (67.2%)	22 (32.8%)	
Class C (n=33)	25 (75.8%)	8 (24.2%)	
Cirrhosis Etiology			
Alcoholic (n=74)	44 (59.5%)	30 (40.5%)	0.028
Hepatitis B (n=29)	19 (65.5%)	10 (34.5%)	
Cryptogenic (n=13)	8 (61.5%)	5 (38.5%)	

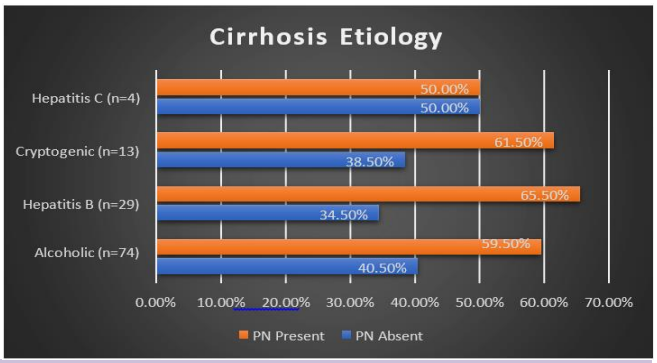

Figure 2: Graphical representation of Cirrhosis Etiology

Peripheral neuropathy prevalence and severity: Overall, electrophysiological testing confirmed peripheral neuropathy in 73 patients (60.8%). The pure sensory type was the most prevalent phenotype (27, 37.0%), followed by mixed sensorimotor (20, 27.4%) and pure motor (17, 23.3%). A highly significant progressive increase in neuropathy prevalence was observed with advancing liver disease severity: Class A (3, 15.0%), Class B (45, 67.2%), and Class C (25, 75.8%) ($P<0.001$) [Table 2 & Figure 2].

Etiology and electrophysiological patterns: Prevalence varied significantly by etiology ($P=0.028$), peaking in Hepatitis B-related cirrhosis (19, 65.5%), followed by cryptogenic (8, 61.5%)

and alcoholic (44, 59.5%). Across all cases, pure axonal degeneration was the overwhelmingly dominant electrophysiological pattern (95.9%). Alcoholic, cryptogenic, and Hepatitis C etiologies exhibited a 100% axonal pattern, whereas a mixed axonal-demyelinating pattern was exclusively observed in a subset of Hepatitis B cases (2, 10.5%) (P<0.001) [Figure 3].

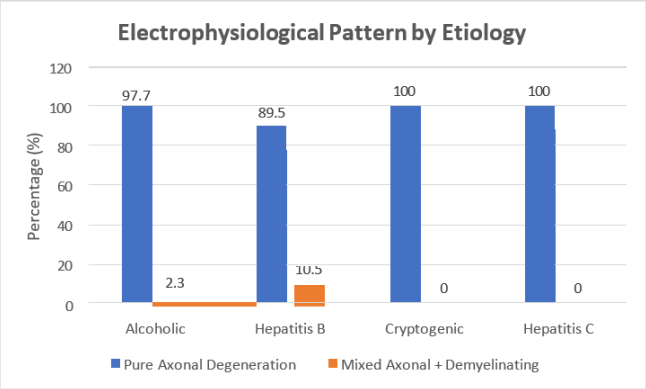

Figure 3: Graphical representation of Graphical representation of Cirrhosis Etiology Lifestyle factors.

Active and former smokers demonstrated a significantly higher prevalence of neuropathy (26, 72.2%) compared to non-smokers (47, 56.0%) (P=0.042). While alcohol consumption history showed a higher prevalence, the independent association was not statistically significant (P=0.162).

Discussion

In this hospital-based cross-sectional cohort (n=120), peripheral neuropathy was highly prevalent (60.8%), manifesting predominantly as a distal, symmetric, and purely sensory axonal degeneration. The composite burden of neuropathy rose significantly across Child-Pugh severity strata, from 15.0% in mild disease to 75.8% in severe decompensation (P<0.001). Furthermore, the disease etiology heavily influenced neuropathy rates, with Hepatitis B patients bearing the highest burden (65.5%, P=0.028), and active smoking was identified as an independent risk factor (P=0.042).

These patterns are biologically and clinically coherent. The overwhelming predominance of axonal degeneration supports a "dying-back" neuropathy, where the longest distal sensory axons are preferentially compromised by metabolic insufficiency, hyperammonemia, and oxidative stress associated with hepatic failure.[3] The lack of a significant independent association between alcohol use and neuropathy—despite alcohol's known neurotoxicity—suggests that the hepatic dysfunction itself is the primary neuropathogenic driver. Conversely, the unique presence of demyelinating features in Hepatitis B-related cases points toward distinct, virus-specific, immune-mediated mechanisms of nerve injury.[4,5] Smoking retained an independent association, suggesting that cumulative oxidative and nitrosative stress exacerbates nerve fiber damage.[6]

Strengths of this study include strict predefined eligibility criteria, an a-priori sample size, and the use of robust electrophysiological testing rather than relying solely on clinical presentation, which often underestimates subclinical neuropathy. Limitations include the single-center scope and the cross-sectional design, which precludes establishing temporality between hepatic decline and neuropathic onset. The small sample size for certain etiologies (e.g., Hepatitis C) also limits the generalizability of those specific subgroup findings.

Future work should adopt longitudinal designs to clarify causality and determine whether neuropathy status provides independent prognostic value beyond conventional MELD or Child-Pugh scores. Evaluating whether targeted interventions for hyperammonemia or oxidative stress can halt electrophysiological decline remains a critical clinical frontier.[7]

Conclusion

Peripheral neuropathy is a highly prevalent, largely subclinical complication of chronic liver disease, manifesting primarily as an axonal sensory neuropathy. Its prevalence scales directly with the severity of hepatic decompensation and varies significantly across viral and metabolic etiologies, identifying severe Child-Pugh classes and Hepatitis B patients as high-risk subgroups. These findings support the routine integration of electrophysiological screening in cirrhotic patients to guide early recognition and comprehensive systemic management.

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Conflicts of interest

There are no conflicts of interest.

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