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Assessment of aetiological, clinical and electrophysiological profile of peripheral neuropathy and their correlation in a tertiary care center

Nazmul Haque[1*], Mohammad Saiful Basar[2], S M Arafat[3]

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*Corresponding author

ABSTRACT

Background: Peripheral neuropathy is a prevalent neurological condition in clinical practice. Proper examination and management need a sequential approach because to its varying appearance and multiple causes. Neuropathies can be classified into subtypes and etiologies based on clinical observations, electro diagnostic testing, and laboratory studies customized to each patient. The study aims to investigate the clinical characteristics, etiology, and electrophysiological profile of peripheral neuropathy at a tertiary care hospital. **Methods & Materials:** The study examined 70 cases of chronic peripheral neuropathy treated at the Department of Neurology at Shaheed Ziaur Rahman Medical College & Hospital in Bogura, Bangladesh, over a 12-month period (June 2024 to May 2025). The study included a detailed examination of the presenting complaint, clinical examination, electrophysiological studies, and other relevant findings. Data were examined using the statistical software SPSS 20.0. **Results:** Patients were 18–75 years old, with male predominance (M: F = 2.4:1). Most had polyneuropathy (68.6%), predominantly affecting both upper and lower limbs (85.8%) with axonal (60%) or demyelinating (40%) patterns. Etiologies included immune-mediated causes, diabetes, CIDP, infections, hereditary, alcohol, and others. Sensory-motor involvement was most common (54.3%), while pure motor neuropathies were rare (1.2%). **Conclusion:** Peripheral neuropathy is more frequent in men aged 30-70. The majority have distal symmetrical motor sensory polyneuropathy. Diabetes is the leading cause of chronic peripheral neuropathy, followed by immune-mediated neuropathy. Hansen's neuropathy is a common infectious neuropathy.

Keywords: *Chronic peripheral neuropathy, electro diagnostic tests, diabetes mellites, immune mediated.*

1. *Assistant Professor, Department of Neurology, Shaheed Ziaur Rahman Medical College & Hospital, Bogura, Bangladesh (ORCID: 0009-0006-7178-1029)*
2. *Professor, Department of Neurology, Shaheed Ziaur Rahman Medical College & Hospital, Bogura, Bangladesh (ORCID: 0009-0007-6748-5830)*
3. *Assistant Professor, Department of Medicine, Shaheed Ziaur Rahman Medical College & Hospital, Bogura, Bangladesh (ORCID: 0009-0004-1085-9326)*

INTRODUCTION

Peripheral nerve disorders are neurological abnormalities caused by malfunction in the motor, sensory, or autonomic nerves. The etiology of neuropathies and their clinical manifestations are quite varied. Neuropathy can be caused by entrapment, systemic diseases, inflammation, autoimmune disorders, hereditary disorders, ischemia situations, paraneoplastic syndromes, deficiencies, infections, and toxins [1]. Peripheral neuropathy affects 2.4% of the population, but climbs to 8% among those over 55 years old. Neuropathy symptoms can be classified as acute (<4 weeks), subacute (4-12 weeks), or chronic (>12 weeks) [2]. Mononeuropathy refers to the involvement of a single nerve and indicates a localized process. The most prevalent causes are direct trauma, compression, vascular lesions, and neoplastic infiltration. Multiple mononeuropathy, also known as mononeuropathy multiplex, refers to the simultaneous or sequential destruction of multiple non-contiguous nerves. Electrophysiological investigations can pinpoint the location of the lesion more precisely than clinical examinations. Examination can distinguish between axonal loss and localized segmental demyelination. The purpose of this study is to look into the clinical characteristics, etiology, and electrophysiological profile of peripheral neuropathy in a tertiary care hospital.

METHODS & MATERIALS

A prospective observational study included 70 patients of chronic peripheral neuropathy from the Department of Neurology at Shaheed Ziaur Rahman Medical College & Hospital in Bogura, Bangladesh, from June 2024 to May 2025.

The study included patients presenting with symptoms and signs suggestive of persistent peripheral neuropathy, as well as any probable cases of ongoing peripheral neuropathy. Patients with acute or sub-acute neuropathy, myelopathy, traumatic neuropathy, or carpal tunnel syndrome were excluded from the study.

The patient provided a detailed history of their current problems, past medical history, family history, and comorbidities. A thorough neurological examination was conducted. We sent a complete blood hemogram, ESR, random blood glucose, renal function tests, liver function tests, serum thyroid and lipid profiles, and viral indicators. Specific tests include serum vitamin B12, HBA1C, serum electrophoresis, urine for Bence Jones proteins, and RA factor. ANA profile, vasculitis panel, Lymes serology, VDRL, and MRI spine with contrast were performed in selected cases. Electrodiagnostic tests are performed in all patients to determine the pattern of involvement. Demyelinating neuropathy is diagnosed when distal latency exceeds 115% of the upper limit of normal with normal CMAP or more than 150% of normal with low CMAP. Axonal neuropathy is characterized by reduced amplitude despite adequate distal delay and conduction velocity [3]. A split skin smear or nerve biopsy is performed, and genetic testing may be necessary. Data were analyzed using the statistical software SPSS 20.0.

RESULT

Table I shows the majority of patients were in the 31–50 years and 51–70 years age groups (41.43% each), indicating that neuropathy was most common in middle-aged and older adults. Fewer cases were observed in the 18–30 years (7.14%) and

>71 years (10%) age groups. Among males, most cases occurred between 31–70 years (82%), while females showed a similar distribution, with 85% between 31–70 years. Overall, the findings suggest that neuropathy predominantly affects individuals in the middle and late adulthood period, with a male predominance across all age groups.

Table I
Age & Sex distribution of the participants (*n*=70).

Age (in years)	Male (50)n(%)	Female (20) n (%)	Total (70) n (%)
18 - 30	4 (8)	1 (5)	5 (7.14)
31 - 50	20 (40)	9 (45)	29 (41.43)
51 - 70	21 (42)	8 (40)	29 (41.43)
> 71	5 (10)	2 (10)	7 (10)

Table II presents Sensorimotor involvement was the most common presentation (54.29%), while pure sensory (18.57%) and pure motor (2.86%) symptoms were less frequent. The majority of patients had both upper and lower limb involvement (67.14%), whereas isolated lower limb involvement was uncommon (5.71%). Bladder involvement (4.29%) and cranial nerve involvement (2.86%) were rare. Skeletal deformities (2.86%), peripheral nerve thickening (1.42%), dysautonomia (1.42%), and gastrointestinal symptoms (1.42%) were infrequent, indicating that autonomic and structural complications were relatively uncommon.

Table II
Distribution of Clinical Symptoms and Neurological Involvement Among Study Participants (*n* = 70).

Symptom	N (%)
Sensorimotor	38 (54.29)
Pure sensory	13 (18.57)
Pure motor	2 (2.86)
Both upper & lower limbs involvement	47 (67.14)
Only lower limbs	4 (5.71)
Bladder involvement	3 (4.29)
Cranial nerve involvement	2 (2.86)
Skeletal deformities	2 (2.86)
Peripheral nerve thickening	1 (1.42)
Dysautonomia	1 (1.42)
GIT symptoms	1 (1.42)

Table III shows polyneuropathy was the most common pattern (68.57%), predominantly axonal (39/48), with fewer demyelinating cases (10/48), indicating that axonal neuropathy was the leading electrophysiological subtype. Mononeuritis multiplex accounted for 14.29% of cases and was mainly axonal in nature. Polyradiculoneuropathy represented 11.43% of cases and was largely demyelinating, reflecting conditions such as CIDP and related immune-mediated neuropathies. Mononeuropathy was least common (5.71%), with a mixed axonal and demyelinating distribution. Overall, axonal neuropathy predominated in polyneuropathy and mononeuritis multiplex, while demyelination was more characteristic of polyradiculoneuropathy cases.

Table III
Distribution of Neuropathy Patterns and Their Electrophysiological Types (Axonal vs. Demyelinating) Among Patients (*n* = 70).

Pattern	N=70, n (%)	Demyelination (20)	Axonal (50)
Poly neuropathy	48 (68. 57)	10	39
Mono neuritis multiplex	10 (14.29)		
Hansens	5		
Sjogren	1		
Vasculitis	1		
Para neoplastic	1		
No cause found	2	1	8
Poly radiculo neuropathy	8 (11.43)	7	2
CIDP			
Rheumatoid arthritis, with GM1,GD1a ab			
Recurrent CIDP			
Childhood CIDP			
MADASM			
MMNCB			
Lymes			
Diabetes			
Mono neuropathy	4 (5.71)		
Bilateral femoral	3		
Radial nerve	1	2	1

Table IV presents Endocrine causes were the most common etiology, accounting for 48.57% of cases, predominantly due to diabetes (41.43%), highlighting diabetes as the leading cause of neuropathy in this cohort. Immune-mediated neuropathies constituted 30%, with CIDP and its variants forming a significant proportion, indicating a substantial burden of treatable inflammatory neuropathies. Infectious causes were identified in 14.11% of cases, mainly leprosy, while hereditary neuropathies accounted for 7.05%. Alcohol-related (8.23%), paraneoplastic (3.52%), drug-induced (2.35%), and chronic kidney disease–related neuropathies were less frequent. No identifiable cause was found in 6 cases. Overall, metabolic and immune-mediated factors were the predominant contributors to neuropathy in this population.

Table IV
Etiological Distribution of Peripheral Neuropathy Cases (*n* = 70).

Aetiology	Number of Cases	Percentage (%)
Endocrine causes	34	48.57
– Diabetes	29	41.43
– Impaired glucose tolerance	5	7.14
Immune mediated	21	30.00
– CIDP	12	17.14
– Pure motor variant	1	1.43
– Sensory variant	2	2.86
– Recurrent CIDP	1	1.43
– Childhood CIDP	1	1.43
– Pure motor variant (second case)	1	1.43
– MADSAM	1	1.43
– MMNCB	1	1.43
– GM1/GD1a antibody	1	1.43
– Multiple myeloma	1	1.43
– Connective tissue diseases	9	12.86
—— Rheumatoid arthritis	2	2.86
—— Sjogren syndrome	1	1.43
—— Systemic sclerosis	2	2.86
—— Vasculitis	2	2.86
—— Panel negative	2	2.86
Medical disorders	1	1.43
– Chronic kidney disease	1	1.43
Hereditary neuropathy	6	8.57
– CMT 1	2	2.86
– CMT 2	2	2.86
– SPG11	2	2.86
Infectious neuropathy	12	17.14
– Leprosy	8	11.43
– Lyme	1	1.43
– Brucellosis	1	1.43
– HIV	1	1.43
– Others	1	1.43
Alcohol-related neuropathy	7	10.00
Paraneoplastic	3	4.29
– NHL	1	1.43
– Ovarian carcinoma	1	1.43
– Anti-Hu antibody	1	1.43
Drug induced	2	2.86
– Bortezomib	1	1.43
– Leflunomide	1	1.43
No cause found	6	8.57

Table V shows Among the 31 cases, axonal neuropathy was predominant, accounting for 24 cases (77.4%), while demyelinating neuropathy comprised 7 cases (22.6%). Diabetes mellitus and entrapment neuropathy together contributed 12 cases (38.7%), mainly with an axonal pattern. Hansen's disease accounted for 5 cases (16.1%), all predominantly axonal. Impaired glucose intolerance was seen in 4 cases (12.9%), also largely axonal. Alcohol-related neuropathy (2 cases, 6.5%), rheumatoid arthritis (2 cases, 6.5%), and single cases (3.2% each) of HIV, leflunomide exposure, celiac disease, chronic kidney disease, sensory variant CIDP, and idiopathic causes were noted. Demyelination was relatively limited and mainly associated with immune-mediated and idiopathic cases. Overall, metabolic and systemic causes were the major contributors, with a clear predominance of axonal pathology (77.4%) over demyelination (22.6%).

Table V
Etiological Distribution and Electrophysiological Pattern (Axonal vs. Demyelinating) of Neuropathy Cases (*n* = 31).

Cause	N=31	Demyelination (7)	Axonal (24)
DM Entrapment	9	5	11
Sensory variant CIDP	31	-	2
Hansens	5	-	1
Leflunomide	1	-	1
Celiac disease	1	-	1
Impaired glucose intolerance	4	-	1
HIV neuropathy	1	-	-
Alcohol	2	-	1
Metabolic (CKD)	1	-	1
Rheumatoid arthritis	2	1	1
idiopathic	1	1	2

Table IV presents among the three cases, hereditary neuropathy was the most common cause (2/3, 66.7%) and demonstrated a purely axonal pattern. Motor CIDP accounted for one case (33.3%) and showed a demyelinating pattern. Overall, axonal neuropathy predominated (66.7%), while demyelination was limited to the immune-mediated case (33.3%).

Table- IV
Pure Motor Neuropathy.

Cause	N=3	Axonal	Demyelination
Hereditary neuropathy	2	2	-
Motor CIDP	1	-	1

DISCUSSION

Peripheral neuropathy is a frequent neurological condition with varied symptoms and causes. In our study, the age of presentation ranged from 18 to 75 years, with the bulk of cases falling between 51 and 70 years old, followed by 31 and 50 years old. There is a male majority of 67%, which is in conformity with the study done by Sase and coworkers [4] (62%), and Goel and coworkers (61%) [5]. Diabetes and dietary deficits are the leading causes of neuropathy [6, 7]. Other causes include entrapment neuropathy and idiopathic. The prevalence of diabetic neuropathy in our study was consistent with that of Bansal et al [8]. Diabetes is the leading cause of slowly progressing neuropathy. It has varying manifestations. The most prevalent observation is distal symmetrical polyneuropathy, with a length-dependent pattern. In two diabetes cases, one exhibited unilateral meralgia paresthetica, while the other had asymmetrical progressive entrapment neuropathy of the femoral nerve with poor glycemic control. Imaging was used to rule out plexopathy. A middle-aged diabetic with poor glycemic control came with increasing quadriparesis. Workups for genetic, autoimmune, and paraneoplastic panels were negative, and nerve biopsy revealed conflicting results. She was administered rituximab to treat immune-mediated neuropathy. In research by Sase and coworkers [4], GBS was the second most common cause. Mygland and coworkers [9] found that alcoholic and toxic neuropathy were the next most common causes. Immune-mediated neuropathy was the second most common cause in our investigation. Lubec et al. [10] reported CIDP at 6.2%, which is lower than our findings. Entrapment neuropathy was found in 9% of cases in a study by Sase and colleagues [4] and 17% in a study by Vishali et al [7], followed by infective neuropathy. Immune-mediated neuropathy is the second most common cause in our study. The majority of CIDP patients were associated with diabetes, with one case confirming MADSAM through nerve biopsy. The cases included DADS variant, bone marrow biopsy-proven multiple myeloma, pure motor variant, sensory variant, remitting and relapsing type, childhood CIDP, GM1, GM2 antibody positive MMNCB, and GM1, GD1a antibody positive. Two cases of connective tissue disorders with mononeuritis multiplex pattern were diagnosed with rheumatoid arthritis and positive for SSA and SSB antibodies. One patient presented with increasing quadriparesis, dysphagia, oesophageal spasm, functional gut motility dysfunction, and hearing loss. A systemic sclerosis work-up revealed a positive result. In one example, a patient arrived with gradually worsening sensory ataxia and severe dysautonomia. An autoimmune and paraneoplastic panel was negative. The patient reacted to steroids rather than IV immunoglobulins. Two cases were clinically suspected of vasculitis with mononeuritis multiplex presentation, but the panel was negative. The patients responded to drugs. Eight occurrences of Hansen's neuropathy were caused by infections. They appeared with mononeuritis multiplex neuropathy. Five instances had MDT treatment, with one completing the course, one defaulting, and one newly diagnosed with split skin smear. One patient remained with a hypasthetic patch, while another had deformities and peripheral nerve thickening. Two cases had significant fiber involvement. Electrodiagnostic investigations indicated motor involvement in three cases. It highlights the importance of taking drug resistance into account while treating Hansen's illness. Two instances presented with gradual quadriparesis, considerable weight loss, and wasting. Spine MRI revealed root enlargement at the conus, indicating Lymes serology. Treatment with doxycycline improved the condition. One instance with bladder involvement tested positive for brucella antibodies, comparable to others. Six occurrences of alcohol-related neuropathy were reported, with the majority being painful. Thiamine supplementation treated a case of small and large fiber neuropathy significantly. One example of pure sensory neuropathy presented with GIT symptoms for three years. Constraints prevented the use of antigliadin antibodies. A duodenal biopsy revealed lymphocytic infiltration but was negative for periodic acid Schiff base stain. But she replied with doxycycline. Smith et al. [11] discovered that only 31% of chronic idiopathic neuropathy cases were idiopathic, whereas the rest were caused by reduced glucose tolerance. Hughes et al. [12]

did not identify glucose intolerance as a significant risk factor. We recommend considering immune-mediated neuropathy in idiopathic patients, even if the panel was negative, as research for novel antibodies continues.

LIMITATION

This was a single-centered study with a modest sample size. As a result, the study's conclusions may not accurately reflect the situation throughout the country.

CONCLUSION

Diabetes, the most prevalent cause, highlights the importance of glycemic control and lifestyle changes. The rise in immune-mediated neuropathy could be attributed to environmental, lifestyle, genetic, and epigenetic factors. Infectious causes should be recognized as avoidable. More large-scale investigations are needed to have a better knowledge of illness profiles.

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