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ORIGINAL RESEARCH

Comparative Clinical and Electrophysiological Profiles of Chronic Inflammatory Demyelinating Polyneuropathy in Patients with and without Diabetes Mellitus: An Observational Study

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Background: Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) is an immune-mediated, treatable neuropathy with clinical and electrophysiological features that overlap with diabetic polyneuropathy, complicating diagnosis and delaying immunotherapy. This study compared the clinical and electrophysiological characteristics of CIDP in patients with and without diabetes mellitus (DM) to determine whether diabetes modifies disease presentation or severity.

Methods: This cross-sectional comparative study was conducted at the National Institute of Neurosciences and Hospital, Dhaka, Bangladesh, from January 2018 to December 2023. Consecutive patients aged ≥15 years with CIDP diagnosed per EFNS/PNS 2010 criteria and reclassified using 2021 EAN/PNS guidelines were enrolled and stratified into CIDP+DM and CIDP−DM groups. Clinical data, disability scores (MRCSS, I-RODS, INCAT), and electrophysiological parameters were compared using Mann–Whitney U and chi-square tests.

Results: Of 143 patients (21 CIDP+DM; 122 CIDP−DM), those with diabetes were older at symptom onset (median 55 vs 45 years, $p = 0.034$) and demonstrated significantly greater distal muscle weakness ($p = 0.045$), though overall disability scores were comparable. Electrophysiological assessment confirmed demyelination in both groups; however, CIDP+DM patients exhibited more pronounced axonal involvement, evidenced by reduced median and ulnar compound muscle action potential amplitudes ($p = 0.034$–0.046) and increased tibial F-wave abnormalities ($p = 0.042$). In age- and sex-adjusted multivariable analyses, diabetes independently modified CIDP by reducing distal muscle power ($\beta = -1.13$; $p < 0.001$), median CMAP at elbow ($\beta = -1.32$ mV; $p = 0.0006$), ulnar CMAP at wrist ($\beta = -1.97$ mV; $p = 0.0004$) and elbow ($\beta = -1.55$ mV; $p < 0.001$), tibial F-wave presence ($\beta = -0.46$; $p = 0.0002$), and overall function with lower MRCSS ($\beta = -3.20$; $p = 0.04$) and higher INCAT scores ($\beta = 1.81$; $p = 0.004$).

Conclusion: Diabetes modifies CIDP, producing a mixed demyelinating–axonal phenotype with greater distal motor impairment.

Keywords: chronic inflammatory demyelinating polyradiculoneuropathy, diabetes mellitus, electrophysiology, demyelination, axonal neuropathy

Introduction

Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) is an immune-mediated, treatable neuropathy characterised by progressive peripheral nerve demyelination, resulting in motor weakness, sensory loss, and areflexia. It presents with heterogeneous clinical and electrophysiological patterns encompassing both typical and atypical variants.[1–3] Global

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prevalence ranges from 0.8 to 8.9 per 100,000, reflecting variations in diagnostic criteria and ascertainment methods.[1,4,5] CIDP most commonly manifests between the fourth and sixth decades, typically as symmetric, proximal-distal sensorimotor neuropathy, although focal or asymmetric variants are recognised.[3,6] Diagnosis relies on identifying characteristic electrophysiological features—conduction block, temporal dispersion, and F-wave abnormalities—that distinguish CIDP from other acquired or hereditary neuropathies.

Diabetes mellitus (DM) is highly prevalent in the same age demographic and represents the leading cause of distal symmetric polyneuropathy (DSPN).[7–9] Whilst DSPN is classically axonal, pathological and electrophysiological studies demonstrate that diabetes can also produce segmental demyelination and inflammatory changes in peripheral nerves, mimicking or coexisting with CIDP.[8,10,11] This overlap complicates diagnosis, as diabetic patients developing weakness, numbness, or sensory ataxia are often presumed to have DSPN rather than inflammatory neuropathy. Consequently, CIDP may remain under-recognised or misclassified in diabetic populations, delaying immunotherapy initiation and potentially causing irreversible axonal loss.[9,12]

Distinguishing CIDP from diabetic neuropathy is further confounded by overlapping clinical and electrodiagnostic findings. Individuals with diabetes are reportedly up to eleven times more likely to meet electrophysiological criteria for CIDP compared with non-diabetic individuals.[10] Although both conditions may exhibit slowed conduction velocities and reduced amplitudes, CIDP is distinguished by immune-mediated demyelination, whereas DSPN reflects chronic metabolic and microvascular injury. Accurate differentiation is crucial because CIDP responds to immunomodulatory therapies—corticosteroids, intravenous immunoglobulin (IVIg), or plasmapheresis—with approximately two-thirds of patients achieving meaningful improvement.[13,14] Emerging evidence suggests that CIDP with diabetes may represent a distinct phenotype characterised by more severe axonal loss, altered nerve conduction patterns, or differential treatment responsiveness.[9,11,12] Understanding these interactions is therefore vital for improving diagnostic precision, optimising therapy, and preventing long-term disability.

This study aimed to compare the clinical and electrophysiological characteristics of CIDP in patients with and without diabetes mellitus, exploring whether diabetes modifies nerve involvement patterns or clinical presentation. Clarifying these differences may support more accurate diagnosis and timely immunotherapy initiation, ultimately improving patient outcomes.

Patients and Methodology

This hospital-based, cross-sectional comparative study was conducted at the Department of Neurology, National Institute of Neurosciences and Hospital (NINS), Dhaka, Bangladesh. Consecutive eligible and consenting patients with chronic inflammatory demyelinating polyneuropathy (CIDP) attending outpatient or inpatient neurology services between January 2018 and December 2023 were recruited. Patients aged ≥15 years with clinical and electrophysiological diagnoses of CIDP were included. Initial diagnosis followed the European Federation of Neurological Societies/Peripheral Nerve Society (EFNS/PNS) 2010 criteria, providing a standardised framework based on clinical features, nerve conduction studies, and supportive findings. To align with contemporary diagnostic standards, all cases were subsequently reclassified according to the revised European Academy of Neurology/Peripheral Nerve Society (EAN/PNS) 2021 guidelines, which refine CIDP subtypes and incorporate supportive investigations such as imaging and biomarkers to improve diagnostic accuracy and reduce misclassification. Both newly diagnosed and follow-up patients were eligible, with no sex restrictions. This study was part of the Service Evaluation and Improvement process, conducted in accordance with the NINS Research Ethics Service guidelines. We used data from January 2018 to December 2023, during which patients received standard care and research ethics committee approval was not required. However, we obtained ethical approval from the Institutional Review Board of NINS (IRB/NINS/2025/450) to allow wider sharing of our findings, as required by our institution's policies. Written informed consent was secured from all participants. The study adhered to the principles outlined in the Declaration of Helsinki.

Baseline demographic and clinical data—including age, sex, disease duration, presenting symptoms, comorbidities, and prior treatment—were recorded using a structured proforma. Laboratory investigations included fasting glucose, HbA1c, renal and liver function tests, thyroid profile, serum vitamin B12, and relevant autoimmune markers. HbA1c measurements were part of the baseline laboratory assessment. Diabetes mellitus (DM) status was determined according

to established international criteria: fasting plasma glucose ≥7.0 mmol/L, 2-hour plasma glucose ≥11.1 mmol/L following a 75-g oral glucose load, or glycated haemoglobin (HbA1c) ≥6.5%.[15] Participants were categorised into two groups: CIDP with diabetes mellitus (CIDP+DM) and CIDP without diabetes (CIDP−DM).

Patients were excluded if they had systemic diseases known to cause neuropathy, including HIV-1 infection, systemic lupus erythematosus, monoclonal gammopathy of undetermined significance (MGUS), plasma cell dyscrasias, chronic hepatitis, inflammatory bowel disease, or Hodgkin lymphoma. Those with toxic or drug-induced neuropathies, hereditary neuropathies based on family history or phenotype, or those who declined consent were also excluded. To minimise confounding, participants were screened for thyroid dysfunction and chronic kidney disease.

Clinical, Functional and Electrophysiological Assessment

Each participant underwent standardised neurological and functional evaluation. Muscle strength was assessed using the Medical Research Council Sum Score (MRCSS). MRCSS was calculated based on bilateral assessment of the following muscle groups: Shoulder abduction, Elbow flexion, Wrist extension, Hip flexion, Knee extension and Ankle dorsiflexion. Each muscle group was graded from 0 to 5 according to the standard Medical Research Council scale, yielding a maximum possible score of 60. Functional disability was evaluated with the Inflammatory Rasch-built Overall Disability Scale (I-RODS) and the Inflammatory Neuropathy Cause and Treatment (INCAT) disability score, providing comprehensive appraisal of neuromuscular performance and activity limitation. Additionally, standard nerve conduction studies (NCS) were performed using a Nihon Kohden Neuropack S3 system. Motor nerves assessed included median, ulnar, tibial, and peroneal; the sural nerve was examined for sensory conduction. Measured parameters included motor conduction velocity, distal latency, compound muscle action potential (CMAP) amplitude, F-wave latency, and conduction block. Conduction block was defined as >30% reduction in CMAP area between proximal and distal stimulation sites. All procedures adhered to established electrophysiological protocols.[16] Electrophysiological studies were conducted according to standardised nerve conduction protocols by trained neurophysiology technicians and interpreted by neurologists experienced in electrodiagnostic testing. While examiners were not formally blinded to diabetic status due to the routine clinical setting, nerve conduction studies follow objective measurement protocols that minimise observer-dependent variability.

Statistical Analysis

The primary objective was to compare the two cohorts regarding clinical characteristics—disease duration, symptom pattern, and functional disability—and electrophysiological profiles, focusing on demyelinating parameters, conduction block, F-wave abnormalities, and nerve involvement patterns. Descriptive statistics summarised demographic, clinical, and electrophysiological variables. Categorical variables were expressed as frequencies and percentages; continuous variables as median (interquartile range). Group comparisons employed the chi-square test for categorical data and the Mann–Whitney *U*-test for continuous data. Additionally, an age- and sex-adjusted multivariable linear regression model demonstrated that diabetes modifies the clinical and electrophysiological characteristics of CIDP. To minimise model instability, the multivariable analyses were restricted to age- and sex-adjusted models only, avoiding overfitting by limiting the number of covariates relative to the sample size. A two-tailed p value <0.05 was considered statistically significant. Analyses were performed using SPSS version 29.0 (IBM Corp., Armonk, NY, USA).

Result

Of 143 patients meeting inclusion criteria, 21 (14.7%) had diabetes mellitus (CIDP+DM) and 122 (85.3%) did not (CIDP−DM) (Table 1). Notably, CIDP+DM patients were significantly older at symptom onset (median 55.0 vs 45.0 years, $p = 0.034$) and predominantly male (95.2% vs 72.1%, $p = 0.023$). Whilst ICU admission was more frequent in CIDP+DM (14.3% vs 4.1%), this difference did not reach statistical significance ($p = 0.061$). Cranial nerve involvement, muscle atrophy, and sensory symptoms—including tingling, numbness, and neuropathic pain—occurred at similar frequencies between groups ($p > 0.05$). However, distal muscle weakness was markedly more pronounced amongst diabetic patients (median muscle power score 2 vs 3, $p = 0.045$), whilst proximal weakness, gait imbalance, and global disability scores (MRCSS, I-RODS, INCAT) remained comparable. Cerebrospinal fluid protein concentrations were elevated in both groups, consistent with albuminocytologic dissociation, without

Table 1 Clinical and Demographic Profiles of the Study Groups

Variable	CIDP+DM (N=21)	CIDP-DM (N=122)	P value
Age (Median, IQR) at onset of symptoms	55.0 (36.5, 61.0)	45.0 (29, 52)	0.034
Male sex	20 (95.2%)	88 (72.1%)	0.023
Type 2 DM, n (%)	19 (95%)		
Duration DM (years)	12.19±11.3		
Comorbidity other than DM (%)	0 (0%)	6 (4.9%)	0.299
ICU needed	3 (14.3%)	5 (4.1%)	0.061
Cranial nerves involvement	3 (14.3%)	9 (7.4%)	0.292
Proximal muscle power (Median, IQR)	4 (3, 4)	4 (3, 4)	0.847
Distal muscle power (Median, IQR)	2 (1, 3)	3 (2, 4)	0.045
Motor weakness of the limbs	21 (100.0%)	117 (95.9%)	0.345
Muscle atrophy (Wasting)	8 (38.1%)	36 (29.5%)	0.431
Areflexia	21 (100%)	111 (91.0%)	0.150
Hyporeflexia	0 (0%)	11 (9.0%)	0.152
Muscle twitching	2 (9.5%)	9 (7.4%)	0.733
Tingling sensation	20 (95.2%)	105 (86.1%)	0.242
Numbness	19 (90.5%)	107 (87.7%)	0.717
Burning pain	6 (28.6%)	25 (20.5%)	0.407
Radicular pain	2 (9.5%)	9 (7.4%)	0.509
Cramping pain	8 (38.1%)	32 (26.2%)	0.263
Gait imbalance	21 (100%)	116 (95.1%)	0.299
Any visual problem	2 (9.5%)	2 (1.6%)	0.043
MRCSS (Median, IQR)	40.0 (33, 45)	44 (34, 48)	0.333
IRODS (Median, IQR)	14 (6, 30)	18 (4.8, 26.8)	0.657
INCAT (Median, IQR)	6 (3.5, 8.5)	5.0 (3.0, 8.0)	0.287
CSF protein (mg/dl) (Median, IQR)	128.0 (89.0, 198.2)	152.0 (101.5, 240.8)	0.480
HbA1c, %	8.21±1.43	5.05±0.44	<0.001
Retinopathy, n (%)	6 (28.6%)	0 (0%)	<0.001
Nephropathy, n (%)	4 (19%)	0 (0%)	<0.001

Notes: Chi square test for all categorical variables, where percentages are compared. Mann Whitney *U*-test for all numerical variables where median and inter Quartile Range (IQR) are compared.
Abbreviations: MRCSS, Medical Research Council Sum-Score; IRODS, Inflammatory Rasch-built Overall Disability Scale; INCAT, Inflammatory Neuropathy Cause and Treatment; CSF, Cerebrospinal Fluid; IQR, Interquartile Range.

significant intergroup differences (median 128.0 vs 152.0 mg/dL, $p = 0.480$). Typical CIDP predominated in both cohorts (85.7% in CIDP+DM vs 83.6% in CIDP−DM, $p = 0.808$), with comparable distributions of atypical presentations and electrophysiological classifications (Table 2). Conduction block—a demyelination hallmark—was more common amongst diabetic patients (95.2% vs 78.7%), though this difference approached but did not reach statistical significance ($p = 0.073$). Sural sparing occurred

Table 2 CIDP Electrophysiological Parameters

Variables	CIDP+DM (N=21)	CIDP-DM (N=122)	P value
CIDP subtypes			
Typical	18 (85.7%)	102 (83.6%)	0.808
Atypical	3 (14.3%)	20 (16.4%)	
Acute CIDP	0 (0.0%)	8 (6.6%)	
EDX types			
CIDP	20 (95.2%)	109 (89.3%)	0.401
Possible CIDP	1 (4.8%)	13 (10.7%)	
Sural sparing	3 (14.3%)	29 (23.8%)	0.335
Conduction block	20 (95.2%)	96 (78.7%)	0.073

Notes: Chi square test for all categorical variables, where percentages are compared. Mann Whitney *U*-test for all numerical variables where median and inter Quartile Range (IQR) are compared.

Abbreviations: EDX, Electrodiagnostic test; CIDP, Chronic Inflammatory Demyelinating Polyneuropathy.

with similar frequency between groups (p = 0.335). Whilst both groups demonstrated demyelination, CIDP+DM patients exhibited significantly lower motor amplitudes, indicating pronounced axonal involvement (Table 3). Median nerve compound muscle action potential (CMAP) amplitude at the elbow was significantly reduced in CIDP+DM (median 1.2 vs 2.4 mV, p = 0.046). Ulnar nerve CMAP amplitudes were markedly lower at both wrist (2.6 vs 4.2 mV, p = 0.034) and elbow (1.1 vs 2.6 mV, p = 0.013). Lower-limb motor studies revealed severe amplitude reductions in both cohorts, with numerically lower peroneal and tibial nerve amplitudes in CIDP+DM, consistent with enhanced distal axonal loss. Distal latencies and conduction velocities were comparable across groups.

Sensory conduction parameters were markedly reduced across all nerves in both groups, with many patients demonstrating absent responses and no significant intergroup differences (Table 3). Critically, F-wave analysis revealed

Table 3 Electrophysiological Profiles of the Nerves of Study Population

Nerves	CIDP+DM	CIDP-DM	P value
Median nerve			
Distal latency (ms)	8.4 (6.2, 11.6)	7.4 (5.1, 11.2)	0.534
Amplitude potential (mV) - wrist	3.4 (1.1, 5.2)	3.9 (2.9, 6.7)	0.110
Amplitude potential (mV) - elbow	1.2 (0.3, 3.2)	2.4 (1.1, 4.0)	0.046
Conduction velocity (m/s)	23.4 (17.1, 33.6)	29.3 (20.7, 37.4)	0.113
Ulnar nerve			
Distal latency (ms)	6.2 (4.3, 7.5)	5.2 (3.9, 8.1)	0.663
Amplitude wrist (mV)	2.6 (1.3. 4.8)	4.2 (2.3, 6.1)	0.034
Amplitude elbow (mV)	1.1 (0.4, 3.3)	2.6 (1.1, 4.3)	0.013
Conduction velocity (m/s)	28.5 (18.4, 36.6)	30.7 (20.9, 40.7)	0.194

(*Continued*)

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Table 3 (Continued).

Nerves	CIDP+DM	CIDP-DM	P value
Peroneal nerve			
Distal latency (ms)	5.3 (0.0, 10.9)	5.3 (0.0, 10.0)	0.887
Amplitude potential (mV) - ankle	0.2 (0, 1.0)	0.5 (0, 1.9)	0.227
Amplitude potential (mV) - knee	0.1 (0, 0.5)	0.2 (0, 0.8)	0.227
Conduction velocity (m/s)	20.6 (0, 35.0)	20.4 (0, 31.4)	0.986
Tibial nerve			
Distal latency (ms)	4.5 (0.0, 11.0)	5.0 (0.0, 9.5)	0.888
Amplitude (mV)- ankle	0.3 (0.0, 1.6)	0.5 (0.0, 2.5)	0.495
Amplitude (mV) – popliteal fossa	0.2 (0.0, 1.3)	0.1 (0.0, 0.8)	0.986
Conduction velocity (m/s)	19.6 (0.0, 32.1)	13.4 (0.0, 28.1)	0.671
Sensory nerve study			
Median nerve amplitude potential (uV)	0 (0, 0)	0 (0, 0.01)	0.25
Ulnar nerve amplitude potential (uV)	0 (0, 0)	0 (0, 0)	0.839
Sural nerve distal latency (ms)	0 (0, 0)	0 (0, 1.9)	0.298
Sural nerve amplitude potential (uV)	0 (0, 0)	0 (0, 0.01)	0.197
Sural nerve conduction velocity (m/s)	0 (0, 0)	0 (0, 18.5)	0.144
F-wave study			
Median nerve F wave (ms)	36.3 (0, 59.9)	33.6 (0, 50.8)	0.525
Ulnar nerve F wave (ms)	36.9 (0, 43.7)	33.6 (0, 47.4)	0.555
Tibial nerve F wave (ms)	0 (0, 0)	0 (0, 53.1)	0.042

Note: Mann Whitney *U*-test for all numerical variables where median and inter Quartile Range (IQR) are compared.
Abbreviations: ms, milliseconds; mV, millivolts; uV, microvolts.

that diabetic patients exhibited significantly higher rates of absent or prolonged tibial F-waves (median 0 vs 0–53.1 ms, $p = 0.042$), indicating more severe motor conduction abnormalities in a length-dependent pattern. Median and ulnar F-wave latencies did not differ significantly.

Further, multivariable linear regression model adjusted for age and sex demonstrated that diabetes independently modified CIDP clinical and electrophysiological features (Figure 1). Diabetes significantly reduced distal muscle power (β = −1.13, 95% CI: −1.49 to −0.76, p < 0.001), median nerve CMAP at elbow (β = −1.32 mV, 95% CI: −2.07 to −0.58, p = 0.0006), ulnar nerve CMAP at wrist (β = −1.97 mV, 95% CI: −3.05 to −0.90, p = 0.0004), ulnar nerve CMAP at elbow (β = −1.55 mV, 95% CI: −2.26 to −0.85, p < 0.001), and tibial F-wave presence (β = −0.46, 95% CI: −0.69 to −0.22, p = 0.0002). Diabetes also worsened overall disability with reduced MRCSS (β = −3.20, 95% CI: −6.29 to −0.12, p = 0.042) and increased INCAT score (β = +1.81, 95% CI: +0.61 to +3.02, p = 0.004), confirming a distinct mixed demyelinating-axonal phenotype independent of age and sex effects.

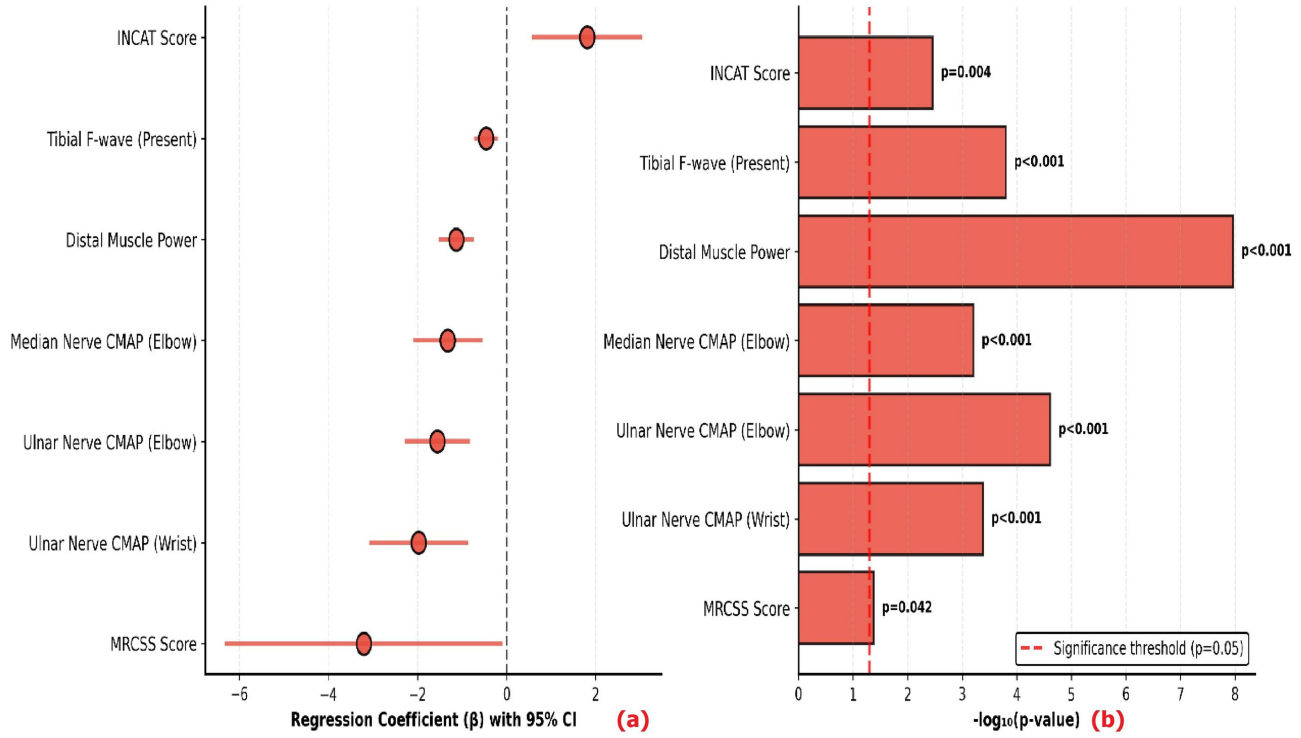

Figure 1 A multivariable linear regression model, adjusted for age and sex, shows that diabetes affects the clinical and electrophysiological characteristics of CIDP. Forest plot (**a**) presents the age- and sex-adjusted effects of diabetes on CIDP, while bar chart (**b**) displays the statistical significance of these effects. A negative value indicates a worse outcome for CIDP patients with diabetes.

Discussion

This study is the first large-scale investigation in Bangladesh to compare the clinical and electrophysiological profiles of patients with chronic inflammatory demyelinating polyneuropathy (CIDP) with and without diabetes mellitus. The results show that, while all patients display typical demyelinating features of CIDP, those with diabetes mellitus present at an older age, have greater distal weakness, and show more evidence of axonal involvement. These findings challenge the traditional view of CIDP as solely a demyelinating disorder in individuals with diabetes and provide new insights to the existing literature.

Consistent with prior reports, the median age of onset was significantly higher amongst diabetic patients (55 years vs 45 years), reflecting the typical later onset of type 2 diabetes and confirming trends reported by Gorson et al[17] and Iijima et al[18] The male predominance observed across the cohort aligns with the known epidemiology of CIDP.[4] Importantly, the longer duration between symptom onset and diagnosis amongst diabetic patients likely reflects diagnostic uncertainty, as symptoms such as paraesthesia and distal weakness are often attributed to diabetic polyneuropathy rather than immune-mediated neuropathy. This pattern of delayed recognition has been described previously[19,20] but remains a critical clinical challenge requiring heightened awareness.

Typical CIDP was the most frequent phenotype in both groups, and disease severity scores (MRCSS, INCAT, I-RODS) did not differ significantly, although diabetic patients exhibited relatively lower distal muscle power. This finding is particularly noteworthy, as it suggests enhanced distal axonal vulnerability consistent with the "double-hit" hypothesis—wherein metabolic injury from diabetes exacerbates immune-mediated demyelination. This represents a significant addition to existing literature, providing clinical evidence for a synergistic pathological interaction between metabolic and inflammatory processes.

Electrophysiological assessment confirmed demyelinating changes in both groups; however, CIDP+DM patients showed significantly greater amplitude reduction—especially in the median and ulnar nerves—suggesting coexisting axonal loss. Notably, the tibial F-wave was more frequently absent or delayed in diabetic cases, indicating more severe distal motor involvement in a length-dependent pattern. Whilst demyelinating parameters were comparable, the diabetic subgroup displayed a distinctive mixed axonal–demyelinating pattern with less frequent sural sparing, consistent with combined

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diabetic and inflammatory pathology. These findings extend earlier electrophysiological and histopathological studies demonstrating that CIDP with diabetes often shows greater axonal degeneration and reduced compound muscle action potential (CMAP) amplitudes than idiopathic CIDP.[17,21] Crucially, our study provides robust quantitative evidence of this phenomenon in a well-characterised cohort.

The pathophysiological basis of this overlap is multifactorial. Chronic hyperglycaemia promotes microvascular dysfunction, endoneurial hypoxia, and oxidative stress, all of which may compromise Schwann cell integrity and lower the threshold for immune-mediated demyelination.[9,22] Consequently, diabetic patients may experience superimposed inflammatory demyelination on a background of metabolic neuropathy, resulting in a more severe clinical and electrophysiological phenotype. Although this study suggests increased axonal loss in CIDP+DM, distinguishing whether such changes represent secondary axonal degeneration from demyelination or primary diabetic axonopathy remains challenging and warrants further investigation.

Distinguishing CIDP from diabetic neuropathy remains a major diagnostic challenge due to overlapping clinical and electrodiagnostic features. Epidemiological data demonstrate variable associations: some studies report a 2- to 11-fold higher likelihood of meeting CIDP criteria amongst diabetic individuals,[10,23] whilst others—such as the Minnesota cohort—found no significant association.[24] This inconsistency underscores ongoing uncertainty regarding whether diabetes is a true risk factor for CIDP or whether diagnostic criteria over-identify CIDP-like features in diabetic polyneuropathy. Nevertheless, clinicians should maintain vigilance for CIDP when diabetic patients present with progressive or asymmetric weakness, areflexia, or conduction block—features atypical of distal symmetric polyneuropathy.[8,9,23]

Although patients with both conditions may have more severe neuropathy, previous studies suggest that their treatment response to corticosteroids, intravenous immunoglobulin (IVIg), or plasma exchange remains comparable to that of non-diabetic patients when disease duration is accounted for.[25] Therefore, accurate and timely diagnosis is critical, as CIDP is treatable even in the presence of diabetes. Delayed recognition may permit irreversible axonal damage and long-term disability.

This study has limitations. It was conducted at a single tertiary centre, and the relatively small number of diabetic patients limits generalisability. Larger multicentre, longitudinal studies integrating advanced imaging, nerve biopsy, and biomarker data would help clarify whether diabetes modifies CIDP pathogenesis or simply amplifies its severity. To minimise model instability, the multivariable analyses were restricted to age- and sex-adjusted models only, avoiding overfitting by limiting the number of covariates relative to the sample size. Further we acknowledge the absence of detailed data on diabetes duration and complications, which could potentially influence neuropathy severity. Despite these limitations, our findings support the notion that CIDP with diabetes represents a distinct, more severe mixed neuropathic phenotype characterised by older age at onset, greater distal weakness, and electrophysiological evidence of additional axonal degeneration—an important contribution to the evolving understanding of CIDP heterogeneity.

Conclusion

Diabetes modifies the clinical and electrophysiological spectrum of CIDP, producing a distinct mixed demyelinating–axonal phenotype characterised by older age at onset, greater distal weakness, and more pronounced axonal loss. Clinicians should maintain a high index of suspicion for CIDP in diabetic patients presenting with atypical or progressive neuropathic symptoms, as timely recognition and immunotherapy can significantly improve outcomes and prevent irreversible disability. Future multicentre, longitudinal studies integrating advanced neuroimaging, nerve biopsy, and inflammatory biomarkers are warranted to elucidate whether diabetes independently modifies CIDP pathogenesis and to determine optimal immunotherapeutic strategies for this distinct patient population.

Patient and Public Involvement

Patients and the public were not involved in the design, conduct, reporting, or dissemination plans of this research.

Provenance and Peer Review

Not commissioned; externally peer reviewed.

Data Sharing Statement

All data generated in this study are available within the manuscript.

Ethics Approval

Ethical approval was granted by the Institutional Review Board of NINS (IRB/NINS/2025/450). Written informed consent was obtained from all participants. The study adhered to the principles outlined in the Declaration of Helsinki.

Patient Consent for Publication

Informed consent was obtained from all participants or their legal representatives.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

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