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CLINICAL, ELECTROPHYSIOLOGICAL PROFILE AND TREATMENT RESPONSE IN CHRONIC INFLAMMATORY DEMYELINATING POLYNEUROPATHY WITH AND WITHOUT DIABETES MELLITUS: A RETROSPECTIVE STUDY FROM ODISHA

Nihar Ranjan Biswal[1], Aujjwalya Kumar Jena[2], Chinmayee Swain[3]

[1]*Assistant Professor, Department of Neurology, SCB Medical college & Hospital, Cuttack, Odisha, India.*
[2]*Assistant Professor, Department of Neurology, SCB Medical college & Hospital, Cuttack, Odisha, India.*
[3]*Senior Resident, Department of Neurology, SCB Medical college & Hospital, Cuttack, Odisha, India.*

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Corresponding Author:
Dr. Nihar Ranjan Biswal,
Assistant Professor, Department of Neurology, SCB Medical college & Hospital, Cuttack, Odisha, India.
Email: niharpediatric@gmail.com

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ABSTRACT

Background: Chronic Inflammatory Demyelinating Polyneuropathy (CIDP) is an immune-mediated neuropathy that causes demyelination of peripheral nerves, resulting in weakness and sensory loss. Differentiating CIDP from diabetic sensorimotor polyneuropathy (DSP) remains challenging because both may present with overlapping electrophysiological abnormalities. This study compared the clinical profile, electrodiagnostic findings, and treatment outcomes of CIDP patients with and without diabetes mellitus (DM) to identify features that may aid diagnosis and guide management.

Materials and Methods: This retrospective analysis included 182 patients with CIDP (91 with diabetes [CIDP+DM] and 91 without diabetes [CIDP-DM]) attending the Neurology and Neuromuscular Clinics at SCB Medical College and Hospital, Cuttack, Odisha, between January 2019 and December 2022. Demographic, clinical, laboratory, and nerve conduction data were extracted from patient records. CIDP diagnosis followed Koski criteria, and diabetes was defined as per ADA guidelines.

Results: The mean age of participants was 66.7 ± 13.2 years, and the mean HbA1c was 7.2 ± 1.8%. CIDP+DM patients had higher systolic blood pressure (142.1 ± 22.4 vs. 133.5 ± 16.2 mmHg; $p = 0.03$) and poorer glycaemic control (7.8 ± 1.9% vs. 5.7 ± 0.4%; $p < 0.0001$). Microvascular complications, including retinopathy (16% vs. 2%), nephropathy (12% vs. 1%), and hypertension (64% vs. 34%), were more prevalent among CIDP+DM patients ($p < 0.001$).

Discussion: The study reveals that CIDP patients with diabetes have more severe neuropathic involvement and greater large-fibre dysfunction but are less likely to receive immunotherapy than their non-diabetic counterparts, despite having comparable treatment responsiveness. This under-treatment may reflect diagnostic uncertainty due to overlapping electrophysiological features with DSP. The only independent factor associated with treatment success was shorter duration of neuropathy, emphasizing the importance of early recognition and intervention.

Conclusions: CIDP with diabetes is associated with worse neuropathic severity and more comorbidities, yet similar potential for treatment response when adequately treated. Diabetes should not preclude immunotherapy in suspected CIDP. The study highlights the need for refined diagnostic criteria and serum or tissue biomarkers to accurately identify immune-mediated neuropathies among diabetic populations, preventing under-treatment of a potentially reversible condition.

Keywords: Chronic Inflammatory Demyelinating Polyneuropathy (CIDP); Diabetes Mellitus; Diabetic Sensorimotor Polyneuropathy (DSP); Nerve Conduction Studies; Immunotherapy; Intravenous Immunoglobulin; Peripheral Neuropathy; SCB Medical College.

INTRODUCTION

Chronic Inflammatory Demyelinating Polyneuropathy (CIDP) is a chronic immune-mediated disorder of the peripheral nervous system that leads to segmental demyelination of motor and sensory nerves. The disease is characterized by slowed motor conduction velocities, prolonged distal motor latencies, and conduction block, reflecting the underlying demyelinating pathology.[1,2] In general, the electrodiagnostic criteria that support the diagnosis of a demyelinating neuropathy include one or more of the following: abnormal distal motor latency in more than half of the tested nerves, decreased motor conduction velocity in over 50% of nerves, or abnormal F-wave latency in more than 50% of nerves.[2,3]

Diagnosing CIDP among patients with diabetes mellitus (DM) poses a significant challenge. This difficulty arises because diabetic sensorimotor polyneuropathy (DSP), especially in poorly controlled diabetes, may also demonstrate mild demyelinating changes, thereby mimicking or overlapping with CIDP.[4] Furthermore, several studies have documented the coexistence of CIDP and DSP in diabetic individuals.[5-11] Some authors have suggested that the presence of two electrodiagnostic abnormalities consistent with demyelination is necessary to establish a diagnosis of CIDP in diabetic patients, whereas only one such abnormality may suffice in non-diabetics.[12]

This diagnostic overlap has important therapeutic implications, as CIDP is typically responsive to immunomodulatory treatment — such as intravenous immunoglobulin (IVIg), corticosteroids, or plasma exchange — whereas DSP is not.[3,12] Hence, differentiating these two conditions is essential to ensure that patients with potentially treatable immune-mediated neuropathy are not misclassified as having irreversible diabetic neuropathy.

In light of this, the present study aimed to compare the clinical features, electrophysiological characteristics, and treatment outcomes between diabetic (CIDP+DM) and non-diabetic (CIDP-DM) patients with CIDP attending SCB Medical College and Hospital, Cuttack, Odisha. A secondary objective was to explore whether these clinical and electrodiagnostic parameters could assist in distinguishing CIDP from diabetic neuropathy in routine clinical practice.

MATERIALS AND METHODS

This study included CIDP patients attending the Neurology and Neuromuscular Clinics at SCB Medical College and Hospital, Cuttack, between January 2019 and December 2022. Relevant demographic details, clinical findings, laboratory results, and electrophysiological data were retrospectively retrieved from patient records. The study protocol was reviewed and approved by the Institutional Ethics Committee.

All included patients were 18 years or older and had a confirmed diagnosis of CIDP. Both diabetic (CIDP+DM) and non-diabetic (CIDP-DM) subjects were included (n = 182; 91 per group). Diabetes mellitus (either type 1 or type 2) was diagnosed according to the American Diabetes Association (ADA) criteria, which include abnormal fasting glucose, elevated HbA1c, random glucose with symptoms, or an abnormal oral glucose tolerance test.[13]

The diagnosis of CIDP was made by an experienced neurologist, based on the clinical presentation and the presence of demyelinating features on nerve conduction studies (NCS), following the Koski criteria.[2] For CIDP+DM subjects, coexisting diabetic sensorimotor polyneuropathy (DSP) was identified if the following criteria were met: at least one abnormal sural NCS, one abnormal peroneal NCS, and one neuropathic symptom or sign.[1,14,15] Patients with proximal diabetic radiculoplexopathies (e.g., diabetic lumbosacral plexoradiculoneuropathy) were excluded based on their asymmetric and painful presentation.

Each participant underwent a detailed neurological examination, including assessment using the Toronto Clinical Neuropathy Score (TCNS), vibration perception threshold (VPT) testing, and comprehensive nerve conduction studies (sural, peroneal, and tibial nerves).[2,16]

Nerve conduction studies were performed using a Sierra Wave electromyography system (Cadwell Laboratories Inc., Kennewick, WA, USA). Recordings followed the standard protocols of the Canadian Society of Clinical Neurophysiology and the American Association of Neuromuscular and Electrodiagnostic Medicine.[17,18] For accuracy, limb temperature was maintained at or above 32°C for upper limbs and 31°C for lower limbs.

Compound Muscle Action Potentials (CMAPs) for peroneal and tibial nerves were measured baseline-to-peak at the ankle and knee, whereas sural Sensory Nerve Action Potentials (SNAPs) were measured from baseline to negative peak. The F-wave latency was recorded as the minimum reproducible latency obtained from ten supramaximal stimuli.

Vibration perception thresholds were assessed using a Neurothesiometer (Howell Scientific, London, UK). Testing was performed three times on the dorsum of each great toe, proximal to the nail bed, and the average of three readings was used for analysis. “Null” tests were randomly introduced to confirm response accuracy.

Treatment Response Classification

Patients were classified as Responders (R) or Non-Responders (NR) based on combined physician and patient evaluation. Responders included those who improved or stabilized after progressive decline, while non-responders either worsened or showed no change following treatment.

All data were analyzed using R software. Continuous variables were presented as mean ± standard deviation (SD) for normally distributed data and as median with interquartile range (IQR) for non-normal data. Categorical variables were analyzed using the Chi-square (χ^2) test, and continuous variables were compared using ANOVA or the Kruskal–Wallis test when appropriate.

To account for multiple comparisons, Bonferroni correction was applied, and the level of statistical significance was set at $p < 0.05$ divided by the number of comparisons performed.

RESULTS

A total of 182 patients diagnosed with Chronic Inflammatory Demyelinating Polyneuropathy (CIDP) were included in the study conducted at SCB Medical College and Hospital, Cuttack, , Odisha. Of these, 91 subjects had diabetes mellitus (CIDP+DM) and 91 were non-diabetic (CIDP-DM). The overall mean age of the study population was 66.7 ± 13.2 years, and the mean haemoglobin A1c (HbA1c) among all participants was 7.2 ± 1.8%.

As shown in Table 1, demographic characteristics such as age, sex distribution, and body mass index were comparable between the two groups. However, CIDP+DM patients had significantly higher systolic blood pressure (142.1 ± 22.4 mmHg vs. 133.5 ± 16.2 mmHg; $p = 0.03$) and poorer glycaemic control with higher HbA1c values (7.8 ± 1.9% vs. 5.7 ± 0.4%; $p < 0.0001$).

Table 1: Clinical and electrodiagnostic features of 91 CIDP-DM and 91 CIDP+DM subjects (n = 182)

Parameter	CIDP-DM (n = 91)	CIDP+DM (n = 91)	P value
Age (years)	67.4 ± 12.9	66.1 ± 13.5	0.48
Male sex, n (%)	66 (73%)	64 (70%)	0.69
BMI (kg/m²)	27.6 ± 4.8	27.9 ± 5.9	0.83
Type 2 DM, n (%)	—	89 (98%)	—
Duration of DM (years)	—	17.2 ± 13.8	—
Duration of PNP (years)	9.9 ± 7.6	10.2 ± 8.1	0.77
Systolic BP (mm Hg)	133.5 ± 16.2	142.1 ± 22.4	0.03
Diastolic BP (mm Hg)	79.5 ± 10.4	82.2 ± 12.5	0.39
VPT upper right	6.6 ± 5.1	7.8 ± 4.7	0.14
VPT upper left	6.4 ± 5.0	7.9 ± 5.3	0.13
VPT lower right	24.9 ± 13.7	32.6 ± 13.2	0.003
VPT lower left	24.6 ± 13.5	31.3 ± 12.8	0.009
TCNS, Median [IQR]	13 [8, 16]	13 [9, 17]	0.44
Symptoms, Median [IQR]	4 [3, 5]	4 [3, 6]	0.28
Sensory, Median [IQR]	4 [2, 4]	4 [3, 5]	0.001 *
DTR, Median [IQR]	6 [4, 8]	6 [4, 8]	0.40
Retinopathy, n (%)	2 (2%)	15 (16%)	0.0008*
Nephropathy, n (%)	1 (1%)	11 (12%)	0.0009 *
Hypertension, n (%)	31 (34%)	58 (64%)	0.0004 *
Hyperlipidemia, n (%)	34 (37%)	39 (43%)	0.47
HbA1c (%)	5.7 ± 0.4	7.8 ± 1.9	<0.0001 *

ascular and microvascular complications were notably more frequent among CIDP+DM subjects — including retinopathy (16% vs. 2%), nephropathy (12% vs. 1%), and hypertension (64% vs. 34%) — all statistically significant ($p < 0.001$). Sensory assessment revealed that the diabetic group had higher vibration perception thresholds (VPT) in the lower limbs (right: $p = 0.003$; left: $p = 0.009$), and greater abnormalities in the sensory component of the Toronto Clinical Neuropathy Score (TCNS) ($p = 0.001$).

Table 2: Nerve Conduction Parameters

Parameter	CIDP-DM	CIDP+DM	P value
Sural nerve amplitude potential (mV)	6.9 ± 5.8 (1.4–29.6)	2.3 ± 2.8 (0–13.8)	<0.0001*
Sural nerve distal latency (ms)	3.4 ± 0.5 (1.6–4.6)	3.6 ± 0.6 (2.0–5.0)	0.10
Sural nerve conduction velocity (m/s)	41.5 ± 5.1 (32–56)	38.3 ± 5.2 (27–55)	0.038
Peroneal amplitude (mV) – ankle	2.9 ± 2.7 (0–12.3)	2.1 ± 2.3 (0–11.0)	0.14
Peroneal amplitude (mV) – knee	2.4 ± 2.5 (0–11.2)	1.9 ± 2.3 (0–12.6)	0.39
Peroneal distal latency (ms)	7.8 ± 4.9 (3.1–31.0)	6.0 ± 1.3 (3.3–9.3)	0.004
Peroneal CV (m/s) – fibular head	34.1 ± 7.1 (16–46)	32.5 ± 6.5 (17–45)	0.27
Peroneal CV (m/s) – popliteal fossa	36.1 ± 7.0 (18–52)	33.0 ± 6.3 (23–47)	0.32
Peroneal F-wave (ms)	55.3 ± 25.8 (0–95.1)	58.8 ± 15.9 (0–80.3)	0.42
Conduction block (%)	13.8 ± 23.0	10.1 ± 43.6	0.59
Tibial nerve amplitude (mV)	5.5 ± 6.0 (0–24.0)	5.8 ± 5.3 (0.4–22.8)	0.76
Tibial amplitude – popliteal fossa (mV)	3.9 ± 4.5 (0–18.0)	4.1 ± 3.7 (0.2–16.0)	0.89
Tibial distal latency (ms)	6.9 ± 3.1 (3.8–18.2)	5.5 ± 1.1 (3.3–8.3)	0.002
Tibial distal latency – popliteal fossa (ms)	18.1 ± 5.3 (1.1–31.5)	17.3 ± 3.2 (10.1–23.6)	0.17

Tibial conduction velocity (m/s)	34.6 ± 6.5 (21–52)	35.4 ± 6.0 (22–49)	0.55
Tibial F-wave (ms)	65.7 ± 5.5 (53.5–77.0)	63.9 ± 8.2 (43.0–80.4)	0.28

In nerve conduction studies (NCS), CIDP+DM subjects demonstrated lower sural nerve sensory amplitudes (2.3 ± 2.8 mV vs. 6.9 ± 5.8 mV; p < 0.0001), slower sural conduction velocity (38.3 ± 5.2 m/s vs. 41.5 ± 5.1 m/s; p = 0.038), and shorter distal motor latencies for both the peroneal (6.0 ± 1.3 ms vs. 7.8 ± 4.9 ms; p = 0.004) and tibial nerves (5.5 ± 1.1 ms vs. 6.9 ± 3.1 ms; p = 0.002). These findings suggest greater axonal involvement and delayed peripheral conduction among diabetic CIDP patients.
Treatment characteristics are summarized in Table 3. A significantly higher proportion of CIDP-DM patients received immunomodulatory or immunosuppressive therapy (93% vs. 60%; p < 0.0001). Intravenous immunoglobulin (IVIG) was the most commonly used therapy in both groups but was administered more frequently to CIDP-DM subjects (87% vs. 53%; p < 0.0001). Similarly, corticosteroid therapy with prednisone (67% vs. 20%; p < 0.0001) and azathioprine (54% vs. 13%; p < 0.0001) were more often prescribed in the non-diabetic group.
Although the overall response to treatment did not differ significantly between groups (p = 0.68), the response to IVIG was more favorable in CIDP-DM patients (82%) compared to CIDP+DM patients (58%) (p = 0.005). The average number of IVIG cycles administered was also higher among CIDP-DM subjects (23.6 ± 38.9 vs. 7.5 ± 12.0; p = 0.019). Plasma exchange (PLEX) was used less frequently overall but showed similar efficacy between groups.

Table 3: Treatment details of 91 CIDP-DM and 91 CIDP+DM subjects

Parameter	CIDP-DM	CIDP+DM	P value
N	91	91	—
Response to treatment (n = 132)			0.68
Non-responders, n (%)	40 (44%)	45 (49%)	
Responders, n (%)	51 (56%)	46 (51%)	
Treatment Provided, n (%)	85 (93%)	55 (60%)	<0.0001 ¥
IVIG, n (%)	79 (87%)	48 (53%)	<0.0001 ¥
Prednisone, n (%)	61 (67%)	18 (20%)	<0.0001 ¥
PLEX, n (%)	13 (14%)	5 (6%)	0.037
Azathioprine, n (%)	49 (54%)	12 (13%)	<0.0001 ¥
Mycophenolate mofetil, n (%)	12 (13%)	9 (10%)	0.44
Loading dose IVIG (2 g/kg)	1.91 ± 0.4	2.01 ± 0.4	0.22
Number of IVIG treatments	23.6 ± 38.9 (1–198)*	7.5 ± 12.0 (0–58)	0.019
Response to IVIG treatment, n (%)	59 (82%)	30 (58%)	0.005
Number of PLEX treatments	1.5 ± 0.9 (1–3)	4.6 ± 0.7 (4–5)	0.0003 ¥
Response to PLEX treatment, n (%)	11 (79%)	4 (67%)	0.61
Clinical Status (n = 132)			0.11
Worse, n (%)	22 (24%)	10 (11%)	
No change, n (%)	18 (20%)	28 (31%)	
Stabilized, n (%)	30 (33%)	21 (23%)	
Improved, n (%)	21 (23%)	32 (35%)	
NCS after treatment (n = 121)			0.83
Worse, n (%)	12 (13%)	9 (17%)	
Stable, n (%)	70 (78%)	39 (75%)	
Improved, n (%)	9 (9%)	4 (8%)	

Among the total of 182 patients, 132 received active treatment and were subsequently evaluated for clinical and electrophysiological outcomes. Their details are provided in Table 4. When stratified according to response status, 100 patients were categorized as responders (R) and 82 as non-responders (NR). Responders had a significantly shorter duration of polyneuropathy (8.4 ± 6.1 years vs. 12.3 ± 7.3 years; p = 0.003), suggesting that earlier initiation of therapy may be associated with better clinical outcomes. No significant differences were noted between responders and non-responders in age, gender, BMI, or systolic blood pressure.
Nerve conduction studies also revealed marginally higher sural and peroneal nerve amplitudes in responders, although these did not reach statistical significance. Overall, patients with shorter disease duration, fewer comorbidities, and early therapeutic intervention demonstrated improved functional and electrophysiological outcomes.

Table 4: Clinical and electrodiagnostic features of 82 Non-Responders (NR) and 100 Responders (R) to treatment for CIDP

Parameter	NR	R	P value
N	82	100	—
Age (years)	68.2 ± 12.1	64.1 ± 14.2	0.15
Male sex, n (%)	55 (67%)	73 (73%)	0.42
BMI (kg/m²)	28.5 ± 4.8	28.3 ± 5.9	0.88
Duration of DM (years)	18.8 ± 13.5	14.7 ± 7.8	0.24
Duration of PNP (years)	12.3 ± 7.3	8.4 ± 6.1	0.003

Systolic BP (mmHg)	137.6 ± 20.8	134.2 ± 19.1	0.46
TCNS, Median [IQR]	13.5 [11, 15]	13 [8, 16]	0.40

Nerve Conduction Parameters

Parameter	NR	R	P value
Sural nerve amplitude potential (mV)	4.2 ± 5.8 (0–29.8)[1]	5.8 ± 5.1 (0–19.1)	0.17
Peroneal amplitude (mV) – ankle	2.3 ± 2.3 (0–7.5)	2.6 ± 2.8 (0–12.6)	0.56
Peroneal CV (m/s) – fibular head	33.8 ± 7.6 (15–46)	32.9 ± 7.2 (15–47)	0.57
Conduction block (%)	17.4 ± 16.9	13.7 ± 31.2	0.54

DISCUSSION

This study conducted at SCB Medical College and Hospital, Cuttack, Odisha, compared the clinical characteristics, electrophysiological findings, and treatment responses of patients with Chronic Inflammatory Demyelinating Polyneuropathy (CIDP) with and without diabetes mellitus (DM). The current analysis, which includes one of the larger cohorts of CIDP patients assessed in this setting, demonstrates that CIDP patients with diabetes (CIDP+DM) exhibit a distinct clinical and electrophysiological pattern compared to CIDP patients without diabetes (CIDP-DM).

In our cohort, CIDP+DM subjects showed greater neuropathic severity, more pronounced large-fibre involvement, and a higher prevalence of vascular comorbidities such as hypertension, retinopathy, and nephropathy. These findings are consistent with the earlier observations of Gorson et al,[5] who reported that patients with diabetes have a more complex neuropathic profile but demonstrate a comparable rate of treatment response when appropriately managed. Despite this, CIDP+DM patients in our study were less likely to receive active immunotherapy, including intravenous immunoglobulin (IVIG) and corticosteroids, compared to their non-diabetic counterparts.

Interestingly, although CIDP+DM patients had higher vibration perception thresholds and greater sensory impairment on the Toronto Clinical Neuropathy Score, the response rate to treatment did not significantly differ between the two groups. The only factor that correlated significantly with treatment responsiveness was the duration of polyneuropathy, with a shorter disease duration associated with better outcomes. The presence or duration of diabetes itself did not predict response. While CIDP-DM patients exhibited a higher response rate to IVIG therapy than CIDP+DM patients, this trend might be influenced by sample variability and treatment selection bias, as also noted in previous reports.[19]

The under-treatment of CIDP in diabetic patients remains a concerning observation. Existing diagnostic criteria for CIDP, although highly specific,[3,20] may lack sensitivity when applied to diabetic individuals. The overlap between CIDP and diabetic sensorimotor polyneuropathy (DSP) in clinical presentation and electrophysiological findings often leads to diagnostic uncertainty. In such cases, demyelinating changes caused by immune-mediated mechanisms may be masked by pre-existing diabetic nerve injury. Consequently, CIDP in diabetic individuals may go undiagnosed or insufficiently treated, as observed in our study, highlighting the pressing need for a reliable biomarker to differentiate between immune-mediated and metabolic nerve damage.

Previous studies have shown that patients with diabetes can demonstrate electrophysiological findings atypical for classic DSP, but insufficient to meet CIDP diagnostic thresholds.[4] A modest reduction in conduction velocity is common in DSP; however, more pronounced slowing, even when not meeting the formal criteria for CIDP, may suggest underlying demyelination.[4] This phenomenon has been linked to differences in glycaemic control, where poorer control (mean HbA1c ≈ 9.6%) correlates with axonal damage, while relatively better control (HbA1c ≈ 7.5%) with demyelinating features.[21] Thus, patients with well-controlled diabetes yet presenting with significant neuropathy and demyelinating features should raise suspicion for CIDP, warranting appropriate immunomodulatory therapy. Our findings suggest that such differentiation is often overlooked in clinical practice.

This study has several limitations. As a retrospective analysis, it relied on available clinical records and electrophysiological data. Although physician and patient evaluations were used to assess outcomes, validated disability scales such as the Inflammatory Neuropathy Cause and Treatment (INCAT) scale, Rasch-built Overall Disability Scale (RODS), and the Overall Neuropathy Limitation Scale (ONLS) were not consistently available during the study period.[22-24] Future studies should incorporate these standardized tools to enable a more objective definition of treatment response. Additionally, misclassification remains a possibility, as demyelination on NCS is not pathognomonic, and there are currently no serum biomarkers to definitively confirm CIDP. Although sural nerve matrix metalloproteinase-9 has been proposed as a potential tissue biomarker,[25] nerve biopsy is invasive and not routinely feasible in all patients.

Another important challenge is distinguishing CIDP+DM from proximal diabetic neuropathies, such as diabetic lumbosacral plexoradiculoneuropathy, which may share overlapping clinical features. However, in this study, cases presenting with typical features of proximal diabetic neuropathy were excluded to minimize

diagnostic overlap. The study also identified a selection bias against aggressive treatment in diabetic CIDP patients, possibly due to concerns about metabolic complications or misclassification. If all CIDP+DM cases had been treated similarly to CIDP-DM, the observed response rates might differ.

Overall, the differentiation between CIDP and diabetes-related neuropathies carries critical therapeutic and prognostic implications. Our findings show that CIDP+DM patients have more severe neuropathy and greater electrophysiological impairment, yet receive less treatment, despite having a similar potential for therapeutic response. As demonstrated in the ICE study,[19] neuropathy severity alone does not preclude clinical improvement with immunotherapy. Therefore, improved awareness and more refined diagnostic approaches are essential to ensure that diabetic patients with potentially reversible immune-mediated neuropathies receive timely and appropriate treatment.

Future research should aim to establish specific electrophysiological thresholds and identify serum or tissue biomarkers that can reliably differentiate CIDP from diabetic polyneuropathy. Such advancements could greatly enhance diagnostic accuracy and treatment outcomes in this complex clinical intersection.

CONCLUSION

In this comparative study from SCB Medical College and Hospital, Cuttack, patients with CIDP and diabetes exhibited more severe neuropathic involvement and comorbidities, yet were less likely to receive adequate immunotherapy compared to non-diabetic CIDP patients. Despite this treatment gap, response rates were comparable across groups, emphasizing that diabetes should not deter active management of CIDP.

The duration of polyneuropathy emerged as a key determinant of treatment success, underscoring the importance of early diagnosis and intervention. Given the diagnostic overlap between CIDP and diabetic sensorimotor polyneuropathy, there is an urgent need for specific electrophysiological cut-offs and biomarkers to accurately distinguish these conditions. Enhancing clinical recognition of CIDP in diabetic populations can substantially improve patient outcomes and prevent under-treatment of a potentially reversible neuropathy.

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