Case Series

Distinguishing chronic inflammatory demyelinating polyneuropathy from diabetic neuropathy: A case series from Northeast India

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ABSTRACT

Chronic inflammatory demyelinating polyneuropathy (CIDP) is an immune-mediated neuropathy that may mimic diabetic sensorimotor polyneuropathy. Timely identification of CIDP in diabetics is critical, as it is potentially reversible with immunotherapy. We report a case series of three diabetic patients presenting with subacute progressive lower limb weakness. All three showed demyelinating features on nerve conduction studies and elevated cerebrospinal fluid (CSF) protein, consistent with albuminocytologic dissociation. Two cases showed classical electrophysiological features of CIDP, whereas one case had bilateral sural nerve involvement, conventionally suggestive of diabetic neuropathy, but responded dramatically to intravenous immunoglobulin. All three patients had functional recovery and improved nerve conduction post-treatment. Differentiating CIDP from diabetic neuropathy is essential, especially in the presence of atypical features or progression. Elevated CSF protein and treatment response remain key diagnostic clues, even in patients with features typically attributed to diabetes.

Key words: Chronic inflammatory demyelinating polyneuropathy, Demyelination, Diabetic neuropathy, Intravenous immunoglobulin, Nerve conduction study, Sural Nerve

Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) is an immune-mediated disorder of the peripheral nerves characterized by progressive symmetrical weakness and sensory loss evolving over more than 8 weeks. Diabetic peripheral neuropathy (DPN), on the other hand, is a common complication of long-standing diabetes mellitus, presenting with distal, symmetrical, predominantly sensory symptoms. Differentiating CIDP from DPN is often challenging due to overlapping symptoms, particularly in diabetic patients. Comparison between CIDP and DPN is shown in Table 1. However, accurate identification is crucial as CIDP is treatable with immunomodulatory therapies such as intravenous immunoglobulin (IVIG), corticosteroids, and plasma exchange, whereas DPN is managed with glycemic control and symptomatic relief only [1]. The European Academy of Neurology/Peripheral Nerve Society 2021 criteria for CIDP highlight the importance of electrophysiological and supportive diagnostic tools such as cerebrospinal fluid (CSF) protein elevation, imaging, and treatment response. In India, studies such as the one by Sharma *et al*. have demonstrated the diagnostic and therapeutic challenges of CIDP in diabetic patients, especially when axonal features or sural nerve involvement coexist [2].

We present three such cases from a tertiary care center, each illustrating different diagnostic scenarios and the importance of early and accurate treatment.

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Case 1: CIDP in a Diabetic Chronic Kidney Disease (CKD) Patient

A 55-year-old male with 15 years of poorly controlled diabetes, hypertension, and CKD stage 4, baseline creatinine 4.2, presented with progressive bilateral lower limb weakness for 6 months and difficulty rising from a chair for 1 month. He retained upper limb strength, bowel/bladder control, and bulbar function. Examination revealed bilateral pedal edema. Hemoglobin A1c was 10%. Nerve conduction study (NCS) showed distal latencies >150% of standard in bilateral median and ulnar nerves with conduction blocks. Lower limb nerves were non-stimulable. CSF analysis showed 3 cells and protein 98 mg/dL, consistent with albuminocytologic dissociation. After nephrology clearance, he received IVIG (first cycle: 2 g/kg; subsequent cycles: 1 g/kg every 4 weeks). The patient began walking unaided, and repeat

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NCS showed improved distal latency and stimulable lower limb nerves. He was started on azathioprine (2 mg/kg/day) for maintenance.

Case 2: Classical CIDP in an Insulin-dependent Diabetic Female

A 58-year-old female, a known diabetic on insulin, presented with bilateral lower limb weakness for 2 months. Examination revealed diminished lower limb reflexes, impaired vibration and joint position sense, and bilateral lower limb wasting. Power was 3/5 in the lower limbs; the upper limbs were normal. Bowel, bladder, cranial nerves, and cerebellar systems were intact. NCS showed increased distal latency, conduction block, and slowed conduction velocity in the lower limbs, with normal sensory studies. CSF showed 3 cells and protein 78 mg/dL, consistent with albuminocytologic dissociation. She received IVIG (first cycle: 2 g/kg; subsequent: 1 g/kg every 4 weeks) and, at 3-month follow-up, was ambulant without support. NCS parameters also improved significantly. She was started on azathioprine for maintenance therapy.

Case 3: CIDP Mimicking Diabetic Neuropathy with Sural Involvement

A 64-year-old diabetic female with well-controlled sugars presented with progressive bilateral lower limb weakness. She was bedbound at admission but retained bowel/bladder and bulbar functions. On examination, deep tendon reflexes were absent in the lower limbs. There was wasting, pedal edema, and impaired joint position/vibration sense. Power was 2/5. NCS showed non-stimulable lower limb nerves and bilateral sural involvement, commonly considered a feature of diabetic neuropathy. However, CSF analysis revealed 4 cells and protein 86 mg/dL, consistent with albuminocytologic dissociation. The duration of symptoms was 4 months. The F wave in the upper limbs was normal. Electrodiagnostic criteria for CIDP were partially fulfilled. A trial of IVIG (first cycle: 2 g/kg; subsequent: 1 g/kg every 4 weeks) was initiated. After 3 cycles, the patient showed dramatic improvement and began walking independently. She was maintained on azathioprine.

Comparison between all three cases is shown in Table 2.

Table 1: Comparison between CIDP and diabetic neuropathy

Feature	CIDP	Diabetic neuropathy
Onset	Subacute to chronic, progressive	Chronic, insidious
Pattern	Proximal and distal, symmetrical	Distal symmetrical
Motor involvement	Prominent	Mild or absent
Reflexes	Absent or reduced	Reduced
Sensory loss	Large fiber involvement (vibration, position)	Small fiber±large fiber
NCS findings	Demyelinating (CB, ↓CV, ↑DL)	Axonal features (↓amplitude)
CSF findings	Albuminocytologic dissociation	Normal or mildly elevated protein
Treatment response	Good with IVIG/steroids	No response to immunotherapy

NCS: Nerve conduction study, CSF: Cerebrospinal fluid, CIDP: Chronic inflammatory demyelinating polyneuropathy, IVIG: Intravenous immunoglobulin, CB: Conduction block, CV: Conduction velocity, DL: Distal latency

DISCUSSION

CIDP is considered a potentially reversible neuropathy, with a prevalence of 1–9/100,000 population [3,4]. The clinical presentation in our three cases highlights the subacute progression of lower limb weakness, a hallmark of CIDP. Two patients had classical electrophysiological findings, whereas the third case showed bilateral sural nerve involvement, typically seen in diabetic neuropathy. However, the presence of elevated CSF protein and dramatic improvement with IVIG therapy confirmed the diagnosis of CIDP in all three patients.

Electrodiagnostic studies are central to distinguishing CIDP from diabetic neuropathy. CIDP typically presents with prolonged distal latencies, slowed conduction

Table 2: Comparison of all three cases

Feature	Case 1	Case 2	Case 3
Age/sex	55/Male	58/Female	64/Female
Diabetes duration	15 years (uncontrolled)	Insulin dependent	Well-controlled
CKD	Stage 4	No	No
Symptoms duration	6 months	2 months	4 months
Reflexes	Diminished	Diminished	Absent
Vibration/joint position sense	Not reported	Impaired	Impaired
Power (lower limbs)	3/5	3/5	2/5
NCS upper limb	Distal latency↑, CB	Normal	Normal
NCS lower limb	Non-stimulable	↓CV, CB	Non-stimulable+ sural involved
CSF cells	3	3	4
CSF protein (mg/dL)	98	78	86
Treatment	IVIG×3 + Azathioprine	IVIG+ Azathioprine	IVIG×3 + Azathioprine
Outcome	Walking unaided	Walking unaided	Walking unaided

CKD: Chronic kidney disease, NCS: Nerve conduction study, CSF: Cerebrospinal fluid, IVIG: Intravenous immunoglobulin, CB: Conduction block, CV: Conduction velocity

velocities, and conduction blocks. In contrast, DPN is associated with length-dependent axonal loss without demyelinating features. Notably, the third patient in our series had sural nerve involvement, which has traditionally been viewed as a feature against CIDP diagnosis. However, emerging literature and our findings suggest that sural involvement does not preclude CIDP, particularly when other supportive features are present [3,5].

CSF analysis revealed albuminocytologic dissociation in all three patients, further supporting the diagnosis. This finding, while not specific, is seen in nearly 80–90% of CIDP cases. Importantly, all three patients responded well to IVIG, with improved mobility and nerve conduction parameters. The use of IVIG, especially in diabetic patients with renal impairment (as in Case 1), should be preceded by nephrology consultation, as the risk of worsening renal function is known [6,7].

Our findings align with previous Indian and international studies that emphasize the need to maintain a high index of suspicion for CIDP in diabetic patients presenting with progressive weakness, proximal involvement, areflexia, or rapid functional decline [8-10]. The third case underlines the critical insight that CIDP can present with sural involvement, and such patients should not be prematurely labeled as having pure diabetic neuropathy.

CONCLUSION

This case series emphasizes the importance of recognizing CIDP in diabetic patients who present with progressive weakness and demyelinating features on NCS. Key diagnostic clues include conduction block, elevated CSF protein, and a favorable response to immunotherapy. Even in the presence of sural nerve involvement, conventionally associated with diabetic neuropathy, CIDP must be considered if the clinical course and supportive investigations are consistent. Early diagnosis and initiation of immunotherapy can result in significant functional recovery.

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