



CHRONIC INFLAMMATORY DEMYELINATING POLYNEUROPATHY (CIDP) IN A YOUNG MALE: A CASE REPORT AND LITERATURE REVIEW

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Abstract

A 25-year-old male presented to the emergency department with complaints of bilateral upper and lower limb weakness accompanied by a tingling sensation. Upon admission, a thorough clinical evaluation and diagnostic workup were conducted, leading to a diagnosis of Chronic Inflammatory Demyelinating Polyneuropathy (CIDP). During hospitalization, the patient was managed with intravenous immunoglobulin (IVIg) therapy, receiving a total of 140 grams, alongside oral medications and supportive care. Physiotherapy was initiated and continued throughout the treatment course to aid in functional recovery. The patient responded well to the treatment regimen, demonstrating significant improvement in symptoms and overall clinical condition. At the time of discharge, the patient was in satisfactory condition with stable vital signs and was advised to continue follow-up care and physiotherapy as an outpatient. This case highlights the importance of timely diagnosis, appropriate immunomodulatory therapy, and multidisciplinary management in achieving favorable outcomes in patients with CIDP.

Keywords: GBS, IVIg, Postural Balance, CIDP, Immune Mediated

1. Introduction

First described in 1890, Chronic Inflammatory Demyelinating Polyradiculoneuropathy (CIDP) is an immune-mediated disorder affecting the peripheral nervous system and nerve roots[1,2]. Characterized by symmetric weakness in both proximal and distal muscles, CIDP is a subset of chronic acquired demyelinating polyneuropathies (CADP).

CIDP can manifest in various clinical forms, with “typical” or “classical” presentations involving a progressive motor-predominant peripheral

neuropathy and sensory impairment, particularly affecting position and vibration sense more than pain and temperature. Notably, CIDP is closely linked to Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP), the demyelinating variant of Guillain-Barre Syndrome (GBS)[3].

CIDP incidence ranges from 0.5 to 3.3 cases per 100,000 people, which increases with age and is more common in males[4,-6].

The main goal of these treatments is to reduce or suppress clinical symptoms by modulating or

suppressing the immune system, and it has been shown that 60–80% of patients that are treated with one of these standard treatments have improvements in their condition[7].

2. Case History

A 25-year-old male arrived at the SMIH emergency department with symptoms of weakness in both upper and lower limbs, accompanied by a tingling sensation. He had no significant medical or family history, was a non-smoker, occasionally consumed alcohol (2–3 pegs), and worked as a laborer in tunnel construction. His vital signs were stable: pulse rate of 80 BPM, respiratory rate of 20/min, blood pressure of 120/70 mmHg, and oxygen saturation of 99% on room air. Cardiac examination revealed normal S1-S2 sounds, a soft and non-tender abdomen, and bilateral vesicular breath sounds with acute exacerbation. The patient was alert and oriented, with bilateral reactive pupils. Muscle strength was reduced, scoring 4/5 in the upper limbs and 3/5 in the lower limbs bilaterally. He exhibited a waddling gait, proximal and distal numbness, weak deep tendon reflexes, and no swelling in the L5-S1-S2 region. His weakness was severe, impairing daily activities like combing hair, gripping slippers, and buttoning shirts. He also struggled to rise independently.

Upon admission, the patient was administered the following treatments:

1. Injection Monocef 1g twice daily
2. Injection Methyl Prednisolone 500 mg twice daily
3. Injection Pantop 40 mg once daily
4. Calgrow Forte 1 tablet once daily
5. Neurobion Forte, 1 tablet once daily

Initial investigations included CBC, RFT, LFT, ESR,

CRP, chest X-ray, urine routine/microscopy, and Nerve Conduction Velocity (NCV) of all four limbs. On the second day, additional tests were conducted: ANA, ANCA, serum ACE, Lumbar Puncture (LP), and screening for HIV, HBV, and HCV. The patient was suspected of having demyelination, Chronic Inflammatory Demyelinating Polyneuropathy (CIDP), or myopathy. By the fourth day, some results were available, while others were pending. Vital signs remained stable, and the same treatment continued. On the fifth day, the patient's blood pressure rose to 150/80 mmHg, with a pulse rate of 76 BPM, respiratory rate of 18/min, and oxygen saturation of 97%. He reported a wet cough and wheezing. A lumbar puncture was performed, and Cerebrospinal Fluid (CSF) samples were sent for gram stain, AFB, ADA, TBPCR, TLC, DLC, sugar, protein, and Oligoclonal band analysis. On the sixth day, his Random Blood Sugar (RBS) was 301 mg/dl after a meal, prompting RBS monitoring three times daily before meals and a plan to initiate regular insulin as needed. The existing treatment regimen was maintained. By the seventh day, the patient complained of a persistent cough, leading to repeat CBC and RFT tests. On the eighth day, Injection Methyl Prednisolone and Monocef were discontinued, and oral Prednisolone 60 mg once daily was initiated. On the ninth day, NCS report illustrates Sensory Motor Polyneuropathy and EMG report illustrates S/O peripheral neuropathy. A 5-day course of Intravenous Immunoglobulin (IVIg) at 140g was planned. The IVIg treatment began on the tenth day, with 5 vials administered on the first and second days, and 6 vials daily from the third to the fifth day, each infused over 20 minutes in 100ml of normal saline. After completing the IVIg therapy, the patient was discharged.

Table 1: Medication given during hospitalisation

S. No.	Medication	Indi-cation	Generic name	Dose	DOA	DOH	MOA
1.	Inj Pantop	OD	Pantoprazole	40mg	7/1/2025	22/1/2025	Proton pump Inhibitor
2.	Tab Calgrow forte	OD	Calcitriol 0.25 MCG+Calcium citrate 500MG+Docosahexaenoic acid 120 MG+Eicosapentaenoic acid (EPA) 180 MG+L-methylfolate 1 MG+Lycopene 10000 MCG+Magnesium sulphate 50 MG+Methylcobalamin 1500 MCG+Pyridoxal 5-phosphate 0.5 MG		7/1/2025	22/1/2025	Nutritional supplement
3.	Tab Predn-isolne	OD	Prednisolone	60mg	14/1/2025	22/1/2025	Corticosteroid
4.	Inj Methyl Predn-isolne	BD	Methyl Prednisolone	1g	7/1/2025	14/1/2025	Corticosteroid
5.	Inj. Monocef	BD	Ceftriaxone	1g	7/1/2025	14/1/2025	Cephalosporin
6.	Tab Azoran	OD	Azathioprine	50mg	21/1/2025	22/1/2025	Immunosuppressant
7.	Inj. IvIG	OD	Human Normal Immunoglobulin for I.V use IP 5% Solution	140g	17/1/2025	21/1/2025	Pooled antibody and a biological agent.

Table 2: Lab reports throughout the admission period

Basic Metabolic panel	10/1/2025	11/1/2025	13/1/2025	21/1/2025	Normal value
HbA1c	6.2				4-5.6%
ESR	19			23	0-10mm/hr
TLC	21240		16180	15220	4000-11000/cumm
Neutrophils	90.6		93.5	95.3	44- 68%
Lymphocytes	5.3%		3.2	3.7	25-48%
Monocytes	3.9		3.2	0.7	2.0-10.0%
RBC	4.94		4.83	4.56	4.50-5.50 million/cumm
Platelets	284		265	263	150-400 * 10 ³ /uL
CRP	1.06			0.40	0.00-5.0 mg/L

Urea- Serum	46		56	37	19- 43 mg/dl
Creatinine	0.8		0.6	0.7	0.8-1.5 mg/dl
Uric Acid	3.3		3.2	3.7	3.5- 8.5 mg/dl
Total Protein	7.20		5.90	9.40	6.30-8.20 gm/dl
Sodium serum	138		135	135	137-145mmol/L
Potassium	4.6		4.4	5	3.5-5.1mmol/L
Chloride	101		102	102	98-107mmol/L
Calcium serum	9.4		9.2	8.8	8.4-10.2mg/dl
Phosphorus	3.9		3.4	3.3	2.5-4.5mg/dl
Cholesterol-Total	169		131	176	150-200mg/dl
FPG		171			74-106mg/dl
Glucose CSF		175			40-70 mg/dl
Microprotein CSF		70			12-60 mg/dl
SGPT				63	4-50 U/L
SGOT				32	17-59 U/L

Table 3: Medication given during discharge

S. No.	Medication	Generic Name	Indication	Dose
1.	Capsule Sompraz	Esmoprazole	OD	40mg
2.	Tab Neurobion Forte	Calcium Pantothenate (50 Mg) + Cyanocobalamin (15 Mcg) + Nicotinamide (45 Mg) + Riboflavin (10 Mg) + Thiamine Mononitrate (10 Mg) + Vitamin B6 / Pyridoxine (3 Mg)	OD	1tab
3.	Tab. Azoran	Azathioprine	OD	50mg
4.	Tab. Prednisolone	Prednisolone	OD	60mg

3. Discussion and Conclusion

Chronic Inflammatory Demyelinating Polyneuropathy (CIDP) is an immune-mediated disorder characterized by progressive or relapsing-remitting weakness and sensory deficits due to peripheral nerve demyelination. It is often considered the chronic counterpart of Guillain-Barre Syndrome (GBS), but with a prolonged course exceeding 8 weeks. CIDP typically presents with symmetric motor weakness, sensory disturbances (tingling, numbness), and reduced or absent deep tendon reflexes. Diagnosis relies on clinical evaluation, Nerve Conduction Studies (NCS) showing demyelination, and Cerebrospinal Fluid (CSF) analysis revealing elevated protein without pleocytosis.

The pathophysiology of CIDP involves autoimmune mechanisms, including autoantibodies against myelin proteins (e.g., neurofascin) and T-cell-

mediated inflammation. Recent studies highlight the role of cytokines like IL-17 and TNF- α in perpetuating nerve damage (Source: Journal of Neurology, Neurosurgery & Psychiatry, 2022). First-line treatments include intravenous immunoglobulin (IVIg), corticosteroids, and plasma exchange. IVIg is particularly effective, with studies showing significant improvement in muscle strength and disability scores (Source: Lancet Neurology, 2023). Emerging therapies, such as complement inhibitors and Fc receptor blockers, show promise in targeting specific immune pathways, offering hope for refractory cases (Source: Neurology®, 2023).

In conclusion, CIDP is a complex disorder requiring timely diagnosis and tailored treatment. Ongoing research continues to refine therapeutic strategies, improving outcomes for patients. Early intervention and personalized approaches based on biomarker

profiles are crucial for optimizing management (articles in *Lancet Neurology* and *Neurology*®).

4. Abbreviations

CIDP: Chronic Inflammatory Demyelinating Polyradiculoneuropathy

EMG: Electro Myocardiogram

CADP: Chronic Acquired Demyelinating Polyneuropathies

AIDP: Acute Inflammatory Demyelinating Polyradiculoneuropathy

IVIg: Intravenous Immunoglobulin

NCS: Nerve Conduction Studies

NCV: Nerve Conduction Velocity

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7. Conflict of Interest: None

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