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# Chronic Immune Sensorimotor Polyradiculopathy: Case Report of A Rare Variant In The UAE

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## 1. Abstract

Chronic Inflammatory Sensory Motor Polyradiculopathy (CISMP) is a rare and debilitating neurological condition characterized by progressive loss of both sensory and motor functions. Its clinical presentation is often nonspecific, posing diagnostic challenges, particularly in younger patients and especially in the context of other diseases such as diabetes.This case report highlights a 24-year-old female with type 1 diabetes who developed a complex spectrum of neurological complications including diabetic polyneuropathy, peripheral neuropathy, and neuropathic pain.The initial therapeutic approach with intravenous immunoglobulin (IVIG) resulted in a satisfactory clinical response, and with subsequent maintenance doses, the patient's condition significantly improved.This case underscores the importance of a multidisciplinary approach in the management of CISMP and emphasizes the need for personalized, tailored approach to treatment strategies to optimize both neurological and metabolic outcomes.

## 2. Introduction

Although it is uncommon for a clinician to come across a patient with immune-mediated demyelinating radiculopathies that are restricted to either proximal sensory or motor roots, it is not unheard of. The involvement of sensory roots is termed as Chronic Inflammatory Sensory Polyradiculopathy (CISMP) and its motor counterpart as Chronic Inflammatory Motor Polyradiculopathy (CIMP), of which only one case has ever been reported [1].

In the previous years, the term CIDP (chronic inflammatory demyelinating polyneuropathy) was used to describe patients with symmetric sensorimotor impairment that involves proximal and distal extremities with associated slowing and/or block of conduction on nerve conduction and other electrodiagnostic testing [1].CISP was first described in 2004 by Sinreich et al. [2].Based on an analysis of 15 patients with sensory ataxia and areflexia but without weakness, their patients had prolonged SSEP latencies and albumin-cytologic dissociation [2-4].Chronic Inflammatory Sensorimotor Polyradiculopathy (CISMP) is a rare and progressive neurological disorder characterized by inflammation and demyelination of peripheral nerves, resulting in sensory and motor dysfunction. This condition is cumulative of a heterogeneous group of immune-mediated neuropathies characterized by relapsing- remitting or chronically progressive clinical course [5, 6].CISMP was first described by Khadilkar et al. [7], in 2017, in their case report of 2 patients who responded well to corticosteroids and had progressive lower limb weakness in addition to sensory ataxia and areflexia [7]. The pathophysiology of CISMP involves immune-mediated damage to the myelin and, in some cases, the axons. The key diagnostic markers are nerve conduction studies and CSF analysis to rule out other differentials.Only a handful of cases have been reported of either of the immune-mediated radiculopathies mentioned in this section.While the precise etiology is unknown, early recognition is crucial as timely initiation of therapy can significantly improve patient outcomes and prevent irreversible disability.

## 3. Case Report

A 24-year-old female, known case of Type 1 Diabetes for the last 8 years, presented to Ibrahim Bin Hamad Obaidallah Hospital (IBHOH) after an episode of seizure induced by hypoglycemia. The patient also reported leg pain and inability to walk for the past few months.According to the family, the patient began experiencing bouts of weakness, lower limb pain and difficulty walking, which started 4 months before. She would find it difficult to get up from a sitting or lying down position, and would often feel dizzy with postural changes. In addition to this, she also had frequent hypoglycemic episodes. During this time, she had started receiving treatment at another medical facility. The patient was given Pregabalin and Duloxetine for her neuropathic pain, however, the treatment was ineffective even after potentiating the medicine doses.During the current episode of seizure, the patient's blood sugar was checked and found to be very low (1.9mmol). Subsequently, after receiving glucagon, the patient's blood glucose improved to normal levels. There is no prior history of seizures.Previously, the patient was taking very high doses of Insulin, around 40 units long- acting and 20-25 units short-acting Insulin about 3 times per day. However, 4 months before her current complaint, she began receiving 2 units Lispro and 8 units Glargine thrice daily.

## 4. Examination Findings

On examination, the patient did not appear to be in any acute distress, was conscious and fully oriented with stable vitals. She was unable to sit up or place her legs down from the bed. When examined, the following findings were noted (Table 1). The patient was then admitted to the medical wards for further management. CBC was normal; other than the low potassium and low glucose, there were no other electrolyte abnormalities. Coagulation profile, liver and kidney function tests were established as normal. Mildly elevated T4 but normal T3 and TSH, while LH, FSH, Prolactin and Estradiol levels also came out normal.Through laboratory studies, adrenal insufficiency and other endocrinological conditions, could be ruled out.The most important differential to be considered was diabetic neuropathy, however, in this case, the patient is young and the incidence of neuropathy in Type 1 diabetes mellitus is much lower when compared to Type 2 diabetes.Another differential of Guillain-Barré Syndrome (GBS) was found to be unlikely primarily due to the

long history of weakness with lower limb pain. The patient would require a nerve conduction study to exclude conduction block and radiculopathy, which are features of CIDP.In addition to the laboratory studies, the following investigations were performed (Table 3):Nerve Conduction Studies (Table 4):

## 5. Findings and Discussion

Nerve conduction study is carried for the sensorimotor fibers in the upper and the lower limbs of the 24-year-old diabetic Type 1 female patient who gives a history of weakness of lower limbs that started on October, 2022 and progressed to the extent that she cannot walk while the patient is transferred to our department with a provisional diagnosis of Chronic Immune Demyelinating Polyradiculoneuropathy andthe study included the median nerves, ulnar nerves including the late "F" response, the peroneal nerves and the sural nerves showing a diffuse axonal sensorimotor neuropathic process of symmetric pattern primarily affecting the lower limbs of moderate degree of severity.

## 6. Conclusion

Nerve conduction study shows axonal symmetric peripheral polyneuropathy.Considering the patient's presentation, with lower limb pain and weakness, bilateral upper and lower limb areflexia, with sensory and motor involvement, alongside the lab, NCS and other investigations, a diagnosis of chronic immune sensorimotor polyradiculopathy was then made.A day after the NCS results, the patient was started on the initial dose of Immunoglobulin infusion, IVIG 2gm/kg over 5 days in divided doses. Oxcarbazepine was added for the neuropathic pain.The patient's condition improved and she was discharged with a scheduled follow-up in the Neurology clinic 3 weeks later. During her follow up, she complained of proximal weakness mainly in the lower limbs with pain, tingling and banding sensation in the feet and hands. She was then admitted for receiving her maintenance IVIG dose, 1gm/kg over 2 days duration and then discharged. At this time, Oxcarbazepine was discontinued.The patient was following up in the neurology clinic semi-regularly. Last follow up, that is one year after the first admission, revealed that the patient no longer complains from any limb weakness or loss of sensations. CNS examination was normal except for absent reflexes in both upper and lower limbs, with reduced power 4+/5 in both lower limbs. No sensory loss observed. No cranial nerve or cerebellar abnormalities. No other focal weakness or deficits present with normal vision. The patient was satisfied with her results and clinical outcome.

## 7. Discussion

Chronic inflammatory sensorimotor demyelinating polyradiculopathy is a rare acquired, immune-mediated neuropathy affecting peripheral nerves and nerve roots. This chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) variant is typically characterized by a relapsing-remitting or progressive course of symmetric weakness of proximal and distal muscles [8]. The condition predominantly affects males, with a male to female sex ratio of 1.5:1 to 4:1 and primarily occurs in adults with incidence increasing with age [8].The cause for CIDP and its variants is unknown, yet there is evidence that it is triggered by immune-mediated reactions. Characteristic pathologic features of CIDP include segmental demyelination and remyelination of peripheral nerves, which may result in onion bulb formation [9].In around 10% of the cases, autoantibodies against nodal and paranodal proteins have been identified, which include IgG4 subclass proteins directed towards the proteins near the Node of Ranvier. Specific targets in CIDP are as follows [10]:

- NF 155 – paranodal protein expressed by glial cells
- NF140 and NF186 proteins present at the nodes
- CNTN1 and Contactin-associated protein 1 (CASPR1).

**Table 1**: Examination findings.

| Upper Limb Power (bilateral) | 5/5, (MRC muscle power scale) |
|---|---|
| Lower Limb Power (bilateral) | 3/5, with feelings of heaviness, (MRC muscle power scale) |
| Upper and Lower limb reflexes | Absent |
| Proprioception and vibration | Impaired in bilateral toes |
| Sensations | Decreased sensation up to C6 level (subjective) |
| Cranial Nerves | Intact |
| Cerebellar signs | Absent |

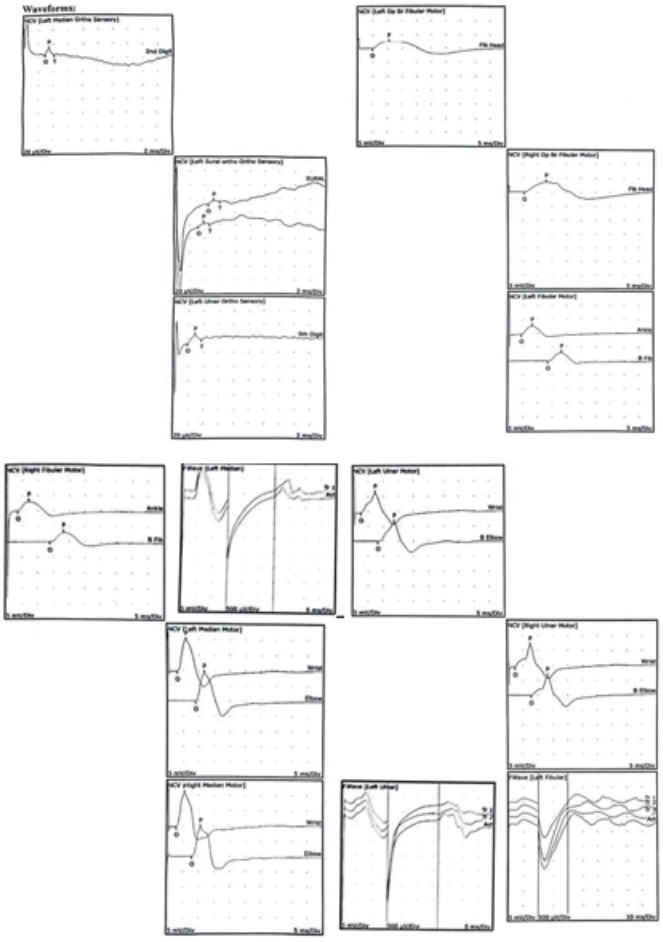
**Table 2**: Laboratory findings of the patient after admission.
*The value in the patient was below normal
#The value in the patient was below normal
LH: Luteinizing Hormone; FSH: Follicle Stimulating Hormone; TSH: Thyroid Stimulating Hormone; LP: Lumbar puncture; WBC: White Blood Cells.

| Laboratory test | Value | Reference range |
|---|---|---|
| Hemoglobin (g/dL) | 11.6* | 12.1 – 15.1 |
| Potassium level (mEq/L) | 3.37* | 3.5-5.2 |
| HbA1C (%) | 8# | <5.7; target for diabetic <6.5 |
| Cortisol AM (nmol/L) | 346.1 | 146-690 |
| Cortisol post Synacthen test (nmol/L) | 673.8 | >420 |
| **Urine** | | |
| Bacteria | Few | None |
| Leukocyte esterase (per HPF) | 10 | 0-5 |
| Nitrates | Negative | Negative |
| **Cerebrospinal Fluid Analysis (LP)** | | |
| Character | Bloody | Colorless |
| WBC (per mm3) | 124# | 5 |
| Glucose (mmol/L) | 5.8# | 2.77 - 4.44 |
| Protein (mg/L) | 1596# | 150 - 450 |
| Lactic acid (mmol/L) | 1.4 | 1.1 - 2.4 |
| CSF culture | Negative | Negative |

**Table 3**: Imaging studies.

| Other Investigations | Results |
|---|---|
| **CT brain without contrast** | Normal CT scan. No well-established recent ischemic insult or fresh hemorrhagic episode. |
| **MRI cervical spine without contrast** | Revealed a straightened cervical curve denoting paravertebral muscle spasm with no central spinal canal or foraminal significant discogenic neural compromise. There was no evidence of cord edema, plaques, or syrinx formation. |
| **MRI thoracic spine without contrast** | No central spinal canal or foraminal significant discogenic neural compromise. No significant disc bulge or frank herniation. No significant abnormalities. |

**Table 4**: Never conduction studies waveforms.



It is interesting to note that biopsies from patients with these antibodies have not shown the findings typically expected in a patient with CIDP such as the onion bulb

formation and macrophage-mediated demyelination. Clinical features also appear to be different from the typical CIDP [10].In regards to the clinical presentation, the most common subtype is the typical CIDP, comprising around 50-60% of the affected cases [11,12].Its features include symmetric, sensorimotor polyneuropathy characterized by proximal and distal muscle weakness that exceeds the extent of sensoryloss. Diagnosis of CIDP is dependent on progression or relapse of the disease over greater than eight weeks. Symptoms are progressive over several months or years. Some patients present with rapidly progressive symptoms and are categorized as AIDP or acute onset CIDP [12,13]. Patients describe difficulty climbing stairs or rising from a seated position, owing to the proximal muscle weakness present. Patients report difficulty in fine motor tasks such as opening jars or doors, buttoning shirts, and often give an account of frequent falls and tripping over their feet due to foot drop [9].Patients with CIDP also have sensory involvement though less prominently as motor symptoms. Instead, sensory impairment in CIDP is usually greater for vibration and position sense, compared to pain and temperature. Patients thus have gait ataxia which is usually mistaken for posterior cord involvement [9,14].Other variants of CIDP that are recognized by the European Academy of Neurology (EAN) and Peripheral Nerve Society (PNS) include:

1.Multifocal CIDP - Also known as asymmetric sensorimotor and Lewis Sumner Syndrome, accounts for 5-10% of cases with a characteristic asymmetric multifocal presentation which results in sensory and motor signs and symptoms in individual nerves. Patients may either have autonomic symptoms, neuropathic pain, and CN involvement [16].

2.Focal CIDP - Uncommon presentation that features sensorimotor deficits typically isolated to the brachial or lumbosacral plexus [16].

3.Motor CIDP – Rare; involves motor nerves and sparing of sensory fibres. Weakness is symmetric and may involve any part of the body [12].

4.Sensory CIDP - Characterized clinically by symptoms and signs consistent with large fiber sensory dysfunction, including ataxia, pain, paresthesias, and dysesthesias. There is no weakness or autonomic dysfunction and the electrophysiologic testing is not abnormal [15].

5.Distal CIDP – Also called Distal-acquired demyelinating symmetric neuropathy (DADS) is a sensory-predominant variant of CIDP which is more slowly progressive than the typical variant [12]. Phenotypically, DADS patients have a monoclonal gammopathy compared to the other variants. 50% of these monoclonal IgM patients have anti-myelin associated glycoprotein (anti-MAG) antibodies which have a different disease mechanism and is different from CIDP and hence, are not considered as a variant of CIDP [17].

6.Proximal CIDP – Also called chronic inflammatory sensory polyradiculopathy (CISP), consisting of inflammatory demyelination confined to dorsal (sensory) nerve roots. This presents clinically with a symmetric sensory ataxia with marked vibration and proprioceptive deficits, indicative of large fibre sensory dysfunction [18].Additionally, it is important to distinguish CIDP from Autoimmune nodopathy as both conditions require a different treatment approach. In any patient presenting with a progressive or relapsing-remitting polyneuropathy involving both motor and sensory axons alone with areflexia, the diagnosis of CIDP should be considered [19,20].Symptoms usually must be present for at least 8 weeks. NCV of both sensory and motor nerves and EMG must be performed in all patients with suspected CIDP. NCV features suggesting CIDP include partial conduction blocks, slow conduction velocity, disappearance of F waves, etc. Patients with other focal or generalized nerve diseases, such as diabetic neuropathy, can also present with such features on electrophysiologic testing and therefore, due to electrodiagnostic overlap, careful assessment is key to discriminate patients with diabetic neuropathy and DADS variant of CIDP [21].There is no specific lab test to diagnose CIDP but testing must be done to rule out the mimics as discussed above. Lumbar Puncture and CSF analysis is performed for all suspected patients with CIDP if electrophysiologic testing is non-diagnostic [15].Albumino-cytologic dissociation is a hallmark of CIDP, wherein the CSF protein is elevated (>45 mg/dL) while the CSF white cell count remains normal. If the patient is diabetic, then the elevated CSF protein level attributed to CIDP would be greater than 100 mg/

dL.Neuroimaging modalities such as MRI of the spine, brachial plexus, and lumbosacral plexus is usually done for the atypical presentations of CIDP as these conditions have features that overlap with structural, infectious causes of polyradiculopathy [15].Biopsies are reserved for patients with unyielding diagnostic testing and there still lies a high suspicion of an infiltrative process. Since CIDP has a patchy demyelination pathology, a biopsy may be limited in giving a true positive or negative, yet despite these drawbacks, if done properly, a nerve biopsy can provide solid evidence of demyelination [22].Early and effective treatment is key in stopping the immune attack against the myelin sheath and preventing long-term disability. Initial immunomodulatory therapy is recommended in patients with mild-moderate disease with rapid progression. IVIG, plasma exchange or glucocorticoids are effective as per the EAN/PNS Guidelines [23,24]. In 2024, FDA approved the use of Efgartigimod alfa-hyaluronidase as a biologic therapy for the use in CIDP. It is an IgG antibody used to promote autoantibody degradation in conditions such as Myasthenia gravis, although the effective use of this medication in CIDP has not been defined [25].For patients with immune-mediated neuropathies due to nodal and paranodal antibodies, it is recommended to use biologic therapy such as Rixutimab as these patients fail to respond to the standard immunomodulatory therapies. If there is no response after the initial therapy, the patient must be re-evaluated to verify the diagnosis to better tailor the management plan. If after initial therapy there is suboptimal response, then an escalated dose or a different modality must be tried. Other immunosuppressant agents that have been used include cyclosporine, methotrexate, azathioprine, mycophenolate and cyclophosphamide [25].

## 8. Prognosis and Outcome

There is limited data regarding the long-term prognosis of CISMP and CIDP, although response to treatment is well recorded. Approximately 40% of the patients with CIDP achieve cure or go into remission but usually not without deficits.

## 9. Conclusion

This case study highlights the diagnostic and therapeutic challenges of CISMP, a CIDP variant, especially in the context of diabetes, which can obscure the clinical picture. By detailing the presentation, management and the patient's response to treatment, we hope to contribute to the growing body of literature on immune-mediated polyradiculopathies and further assist clinicians in recognizing and managing similar conditions more effectively, ultimately improving outcomes and quality of life of patients facing these complex and challenging disorders.

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