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Case Report

Rare cause of neck pain and gait imbalance: a case report

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ABSTRACT

Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) is an uncommon cause of neck pain and radiculopathy that can be easily misdiagnosed. The lack of diagnostic criteria makes the diagnosis of such cases challenging. However, the correlation between clinical presentation with laboratory and radiological findings can help diagnose these patients. Surgical decompression is rarely indicated in the absence of any cause of neuronal compression. Medical treatment of CIDP is currently the main line of treatment. We are presenting a case of a 32-year-old male with no known medical conditions who presented to our spine clinic with a history of chronic neck pain associated with gradual and progressive left upper limb weakness and imbalance. Clinical imaging showed hypertrophied nerve roots of the cervical spine. The patient was treated with intravenous immunoglobulins and corticosteroids, which improved his symptoms significantly.

Keywords: Chronic inflammatory demyelinating polyradiculoneuropathy, Neurological deficit, Neck pain, Weakness, Imbalance

INTRODUCTION

Neck pain accompanied by neurological symptoms, such as motor weakness or sensory disturbances, is a frequent clinical presentation in primary care and specialized settings. It can arise from a broad spectrum of etiologies, ranging from localized compressive neuropathies to systemic nervous system diseases. While common etiologies, such as cervical disc herniation or spondylosis, are often identified through clinical evaluation and imaging, atypical presentations or overlapping pathologies can pose significant diagnostic challenges, especially if the central nervous system is involved.[1-5]

In cases where history and physical examination are insufficient, using advanced diagnostic tools becomes important. Imaging modalities such as magnetic resonance imaging (MRI), computed tomography (CT), and ultrasound (US) play an important role in identifying structural abnormalities. However, in patients with neurological deficits without clear structural pathology, functional studies like nerve conduction studies (NCS) and electromyography (EMG) are crucial for evaluating peripheral nervous system involvement. Additional investigations, such as cerebrospinal fluid analysis through lumbar puncture, can also provide valuable insight into diagnosing inflammatory or demyelinating processes.[6,7]

This case report highlights a diagnostic journey of a 32-year-old gentleman who presented to our spine clinic with a history of persistent neck pain associated with radicular pain and weakness of the left upper limb with no evidence of any disc disease causing a neurological compression. However, further clinical investigations confirmed the presence of chronic inflammatory demyelinating polyradiculoneuropathy (CIDP). This report adds to the existing literature by highlighting an unusual presentation of CIDP and discussing the challenges and clinical

reasoning involved in differentiating it from more common conditions.

CASE REPORT

A 32-year-old right-hand-dominant male, working as a private driver, presented to our spine clinic with chronic neck pain associated with progressive left upper limb weakness. His symptoms were accompanied by an 8-year history of gait imbalance, which had previously led to a traumatic left ankle fracture requiring surgical fixation 4 years prior. The weakness in his left upper limb was insidious in onset, gradually progressive, and associated with numbness and tingling sensation in both hands. The patient denied any history of dizziness, headache, photophobia, palpitation, bowel or bladder disturbance, or saddle anesthesia. Additionally, there was no history of associated rigidity.

On examination, the patient exhibited a bilateral resting tremor and a wide-base gait imbalance, though he could walk unassisted. There was no visible spine deformity, but he had a painful cervical range of motion. Neurological findings included a positive Romberg test, difficulty with the heel-to-toe walk, a positive Lhermitte sign, and positive Tinel and Durkan compression tests bilaterally.

Deep tendon reflexes were absent in all limbs, and muscle strength in the left upper limb was graded as 4/5 in a non-myotomal distribution, compared to 5/5 in the right upper limb. Lower limb strength was 5/5 across all myotomes. Notably, there was no rigidity in neurological examinations.

Urgent magnetic resonance imaging (MRI) of the cervical spine was performed to evaluate for spinal cord compression and myelopathic changes. The imaging revealed hypertrophied cervical nerve roots, as shown in Figure 1.

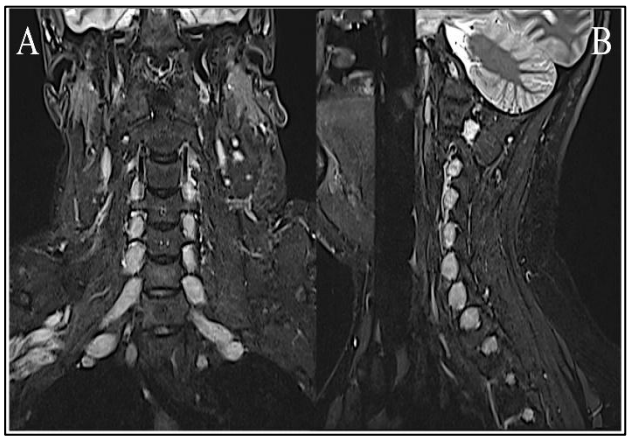

Figure 1: (A) Coronal T2 fat-saturated sequence MRI demonstrating bilateral hypertrophied nerve root involving the nerve root from C1 till T1; (B) sagittal T2 fat-saturated sequence MRI demonstrating the hypertrophied exiting nerve root from C1 till T1.

Subsequently, the patient was referred to a neurologist for further evaluation. Laboratory investigations, including a complete blood count and renal function tests, were within normal limits on admission. A lumbar puncture was attempted, but the procedure was challenging due to the patient experiencing an electrical shock-like sensation in his legs. Despite this, 1 mL of clear cerebrospinal fluid (CSF) was obtained. CSF analysis revealed no cellular abnormalities, and no organisms were identified.

The patient was diagnosed with chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) and initiated intravenous immunoglobulin (IVIG) therapy. He received an initial course of IVIG at a dose of 0.4 g/kg daily for 5 days, followed by maintenance therapy of 60 mg monthly for 6 months. Clinically, he demonstrated moderate improvement, significantly reducing neck pain and better balance.

However, slight residual upper limb weakness persisted, though it did not affect his daily activities. He has been on regular follow-ups with the neurologist over the past 2 years. He experienced occasional symptom relapses, requiring additional monthly IVIG infusions.

DISCUSSION

CIDP is a rare disorder of the peripheral nerves and nerve roots that is often missed or misdiagnosed. It is characterized by chronic immune-mediated inflammation, and in many cases, the initial presentation includes mild weakness or sensory deficits, which can make timely diagnosis difficult.[8] Although the underlying pathophysiology is poorly understood, it is believed to involve both cellular and humoral autoimmunity.[9,10]

Patients typically present with progressive bilateral distal motor deficits, sensory loss, and hyporeflexia. However, the lack of clear diagnostic criteria for CIDP makes its diagnosis challenging and often delays early identification and treatment.[11-13] In our case, the patient presented with a history of gait imbalance, progressive weakness, and numbness in his upper limbs, raising concerns about spinal cord compression. This highlights the importance of excluding other neurological causes in these patients.

The diagnosis of CIDP requires correlating clinical, laboratory, and radiological findings. The diagnosis should rely on the following elements complete blood count, electrolyte analysis, and screening for systemic diseases like HIV, along with protein electrophoresis, electrophysiological studies, NCS, and EMG, are critical for identifying features such as conduction blocks, increased motor and sensory latencies, and absent F waves, CSF analysis can also aid in diagnosis, with elevated protein levels without pleocytosis being a typical finding in CIDP, radiological imaging, particularly MRI, plays an essential role in excluding central nervous system pathology: MRI remains the investigation of choice, nerve roots biopsy in selected patients.[8,12,14]

Our patient underwent many investigations to reach his final diagnosis, including an MRI scan of the spine, which showed significant nerve root swelling but no evidence of significant compression of the spinal cord to explain his symptoms. Interestingly, while CIDP sometimes has been linked to diabetes, our patient had no history of diabetes.[15,16] The current recommendation for treating CIDP is mainly medical, with intravenous immunoglobulins and corticosteroids being the first line of treatment.[17-20] Plasmapheresis has also been shown to have good results.[17,21-23] However, in the case of CIDP associated with radicular hypertrophy causing spinal canal stenosis, surgical decompression becomes a valid treatment option.[24]

CONCLUSION

CIDP is a rare neurological disease that can lead to gait imbalance. Although spinal cord compression is the most common and important cause of spine pain associated with numbness and limb weakness, spine surgeons should be aware of other conditions, such as CIDP, that can present with signs and symptoms similar to spinal cord compression and myelopathy. Early identification and treatment will lead to better clinical outcomes.

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