Open Access Library Journal
2025, Volume 12, e13429
ISSN Online: 2333-9721
ISSN Print: 2333-9705

Spastic Tetraparesis Due to CIDP with Nerve Root Hypertrophy

—A Case Report and Literature Review

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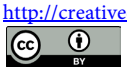
How to cite this paper: El Hayani, O., Benabdeljlil, M., Bnouhanna, W., Rahmani, M., Errguig, L., Birouk, N. and Aidi, S. (2025) Spastic Tetraparesis Due to CIDP with Nerve Root Hypertrophy. *Open Access Library Journal*, **12**: e13429.
https://doi.org/10.4236/oalib.1113429

Received: April 14, 2025
Accepted: May 26, 2025
Published: May 29, 2025



Abstract

Chronic inflammatory demyelinating polyneuropathy (CIDP) with nerve root hypertrophy is a relatively common association, reported in approximately 16 - 54% of cases based on imaging studies. However, it may present a diagnostic challenge, particularly in atypical clinical presentations. We report the case of a 43-year-old patient diagnosed with CIDP who presented with pyramidal syndrome in the lower limbs. Spinal MRI revealed marked hypertrophy of the nerve roots, underscoring the critical role of imaging in supporting the diagnosis and excluding differential diagnoses. Treatment with rituximab, an anti-CD20 monoclonal antibody, has shown promising outcomes in cases of CIDP resistant to conventional therapies, leading to significant clinical improvement and sustained remission in many reported cases, including ours.

Subject Areas

Neurology

Keywords

CIDP, Nerve Roots Hypertrophy, Spinal Cord Compression

1. Introduction

Chronic inflammatory demyelinating polyneuropathy (CIDP) is the most common chronic autoimmune neuropathy, defined as an immune-mediated disorder of the peripheral nerves and nerve roots involving the myelin sheath, character-

ized by a relapsing-remitting or continuously progressive course lasting at least two months. The prevalence of CIDP ranges from 1 to 2 per 100,000 individuals worldwide [1] [2]. While nerve root hypertrophy is typically associated with CIDP, atypical presentations can complicate diagnosis and management. Recent case reports have highlighted instances where CIDP manifests with unusual features such as predominant motor involvement, pyramidal signs, or significant spinal cord compression secondary to nerve root enlargement [3] [4]. In these atypical cases, the clinical course, nerve conduction studies, and spinal MRI findings remain critical in suggesting an inflammatory neuropathic process. Here, we present a clinical case of CIDP with extensive hypertrophic nerve roots manifesting as spastic tetraparesis, underscoring the need for high clinical suspicion and thorough imaging evaluation in atypical presentations.

2. Case Description

A 43-year-old patient was admitted in 2008 at the age of 28 with intermittent diplopia and generalized fatigue. Brain MRI revealed white matter lesions, leading to a diagnosis of multiple sclerosis. He was initially treated with azathioprine and later with cyclophosphamide. In 2015, the patient gradually developed weakness in all four limbs. Clinical examination revealed predominant right-sided spastic tetraparesis, absent deep tendon reflexes, bilateral thenar eminence atrophy, decreased tactile and vibratory sensation in the four limbs, and proprioceptive ataxia. Brain MRI revealed two bilateral signal abnormalities in fronto-parietal subcortical white matter, with the left lesion being larger than the right. Neither lesion extended to the ventricles and without gadolinium enhancement, while spinal cord MRI showed no signal abnormalities.

Nerve conduction studies indicated chronic demyelinating polyneuropathy, as illustrated in **Table 1**. A lumbar puncture demonstrated albumin-cytological dissociation. The patient was diagnosed with CIDP associated with pyramidal signs. Treatment included corticosteroids (1 mg/kg/day) with a slow taper, azathioprine, and baclofen. The patient's condition remained stable, although he developed urinary urgency.

In May 2023, the patient suddenly experienced slurred speech and rotational dizziness following a family conflict. Clinical examination showed pyramidal syndrome in the lower limbs, with muscle strength graded at 4/5 in the left upper limb and both lower limbs, and 3/5 in the right upper limb, atrophy of hand muscles, abolished deep tendon reflexes in the upper limbs, atrophy of the right side of the tongue and deviation to the left during protrusion. There were no palpable thickened nerves.

A spinal cord MRI (**Figure 1**) using a radiculoplexus protocol optimized for peripheral nerve visualization. Imaging was conducted on a 1.5 Tesla. The protocol included the following sequences: T1-weighted spin-echo sequences in sagittal and axial planes to assess anatomy and detect mass effect or nerve root enlargement.

This MRI revealed extensive hypertrophy of the nerve roots in the lumbosacral

and cervical plexuses. Brain MRI showed the same white matter lesions than observed in 2015 (Figure 1). Standard blood tests were normal, serological tests were negative, and lumbar puncture findings were unremarkable.

The patient was referred to neurosurgery for consideration of decompressive laminectomy. However, the intervention was not performed due to stability of symptoms. The diagnosis of hypertrophic CIDP was confirmed, and treatment was switched to rituximab, with maintenance of prednisone at 10 mg/day.

Table 1. Summary of the nerve conduction studies (motor and sensitive) in 2019 and 2024.

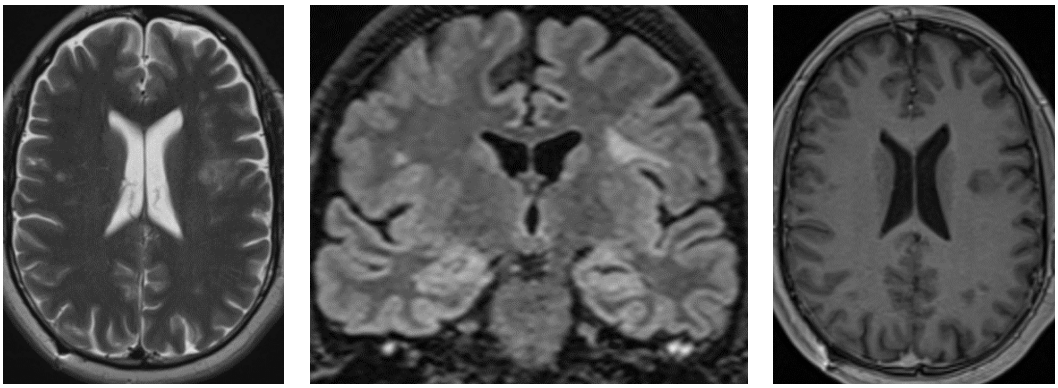
(a)

Nerve/Sites	Motor Amplitude (mv) Distal/Proximal		Latency		Velocity		F wave (ms)	
	2019	2024	2019	2024	2019	2024	2019	2024
Tibial R	1.4 /0.5 (<2)	1.5/1.4	5.89 (<6)	5.04	32 (>40)	27	85.9 (60)	74.3
Tibial L	1.5/1.3	1.4/1.4	8.28	7.85	80	31	78.6	77.9
Peroneal R	0.2/0.3 (<2)	0.1/NO	14.48 (<6)	9.17	25			
Peroneal L	1.7/1.4	0.9/0.9	6.46	7.79	29	27	91 (60)	
Median R	10.9/9.7 (>4.8)	9.9/8.7	3.91(<4)	4.02	28 (>48)	34	60.5 (32)	55
Median L	13.1/11.5	9.6/9.2	3.85	3.06	30	29	48.6	54
Ulnar R	8.8/7 (>4.8)	7.9/5.9	4.64 (<4)	4.13	23 (>48)	27	59.2 (32)	54.8
Ulnar L	5.6/5.6	5.6/5.2	3.7	3.04	30	29	53.8	51.3

(b)

Nerf/Site	Sensitive Amplitude		Velocity	
Radial R	14.5 (<12)	12.3	24	30
Median L	5.6 (>12)	8	26	44
Sural R/L	6.7/3.1 (>8)	0.67/NO	25/15	

NO: not obtained; Note: The normal values are in brackets; the pathological values are highlighted in red.



(A) (B) (C)

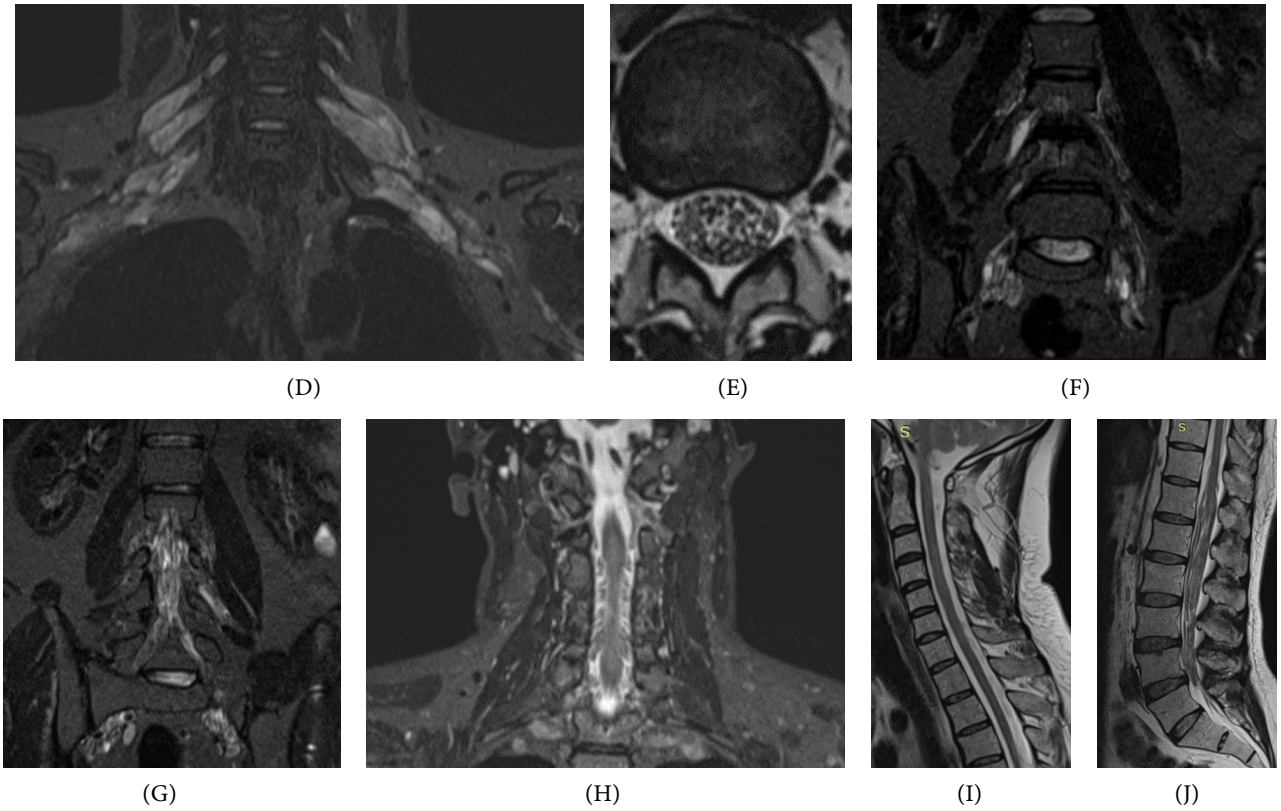


Figure 1. Brain MRI showing: (A), (B): Axial T2 and coronal FLAIR sequences revealing bilateral hyperintensities in the frontoparietal subcortical white matter; (C): Axial T1 with gadolinium contrast showing no contrast enhancement; (D): Coronal STIR sequence showing cervical root hypertrophy; €: Axial STIR sequence showing root hypertrophy; (F), (G): Coronal sequences showing lumbar root hypertrophy; (H): Coronal STIR sequence showing cervical root hypertrophy; (I), (J): Sagittal T2-weighted sequences showing no compression in the cervical and lumbar spinal cord.

3. Discussion

CIDP is an acquired immune-mediated disorder of the peripheral nerve and nerve root, typically presenting with proximal and distal symmetrical weakness, areflexia, and clinical progression lasting more than two months [5]. The annual incidence is estimated to be around 1 per 100,000 persons, with a prevalence ranging from 3 to 9 cases per 100,000 population [6]-[8]. A history of prior infection is found in fewer than 10% of patients [1].

The pathophysiology is explained by macrophage-mediated inflammatory demyelination involving both proximal and distal nerve segments. Macrophages contribute to myelin destruction and promote myelin regeneration, leading to the characteristic "onion bulb" changes observed in CIDP nerve biopsy specimens [9].

The clinical features predominantly involve large myelinated fiber dysfunction, typically leading to proximal and distal limb weakness, ataxia, sensory loss, as seen in our case, and generalized areflexia. Diagnosis remains primarily clinical, supported by electrophysiological evidence suggestive of demyelination [5]. CSF protein levels are almost always elevated, with a sensitivity range of 42% to 77% [10].

However, the CIDP phenotype can be highly heterogeneous, classified into "typical CIDP" and "atypical CIDP" forms account for up to 30% of cases [11]. such as distal acquired demyelinating symmetric neuropathy (DADS) and multifocal acquired demyelinating sensory and motor neuropathy (MADSAM also called Lewis-Sumner syndrome [LSS]). pure motor and sensory CIDP. CIDP variants may be associated with specific antibodies, such as anti-Contactin-1 and anti-Neurofascin. These variants, known as nodo-paranodopathies, can respond to targeted treatments such as Rituximab [11].

According to the 2021 European Academy of Neurology (EAN)/Peripheral Nerve Society (PNS) guidelines, nerve conduction studies typically reveal conduction blocks, abnormal temporal dispersion in the intermediate segment of the nerve trunk, prolonged distal and proximal latencies, and slowed conduction velocities [9] [10]. Our patient fulfils the diagnostic criteria, as shown in **Table 2**.

Table 2. Motor nerve conduction criteria selon EAN/PNS (2021) [10].

Criterion	Description
1) Strongly supportive of demyelination	At least one of the following:
(a)	Motor distal latency prolongation ≥50% above ULN in two nerves (excluding median neuropathy at the wrist from carpal tunnel syndrome).
(b)	Reduction of motor conduction velocity ≥30% below LLN in two nerves.
(c)	Prolongation of F-wave latency ≥20% above ULN in two nerves (≥50% if distal negative peak CMAP amplitude <80% of LLN).
(d)	Absence of F-waves in two nerves (if distal negative peak CMAP amplitudes ≥20% of LLN) + ≥1 other demyelinating parameter in ≥1 other nerve.
(e)	Motor conduction block: ≥30% reduction of the proximal relative to distal negative peak CMAP amplitude, excluding the tibial nerve, and distal negative peak CMAP amplitude ≥20% of LLN in two nerves; or in one nerve + ≥1 other demyelinating parameter except absence of F-waves in ≥1 other nerve.
(f)	Abnormal temporal dispersion: >30% duration increase between the proximal and distal negative peak CMAP (at least 100% in the tibial nerve) in ≥2 nerves.
(g)	Distal CMAP duration prolongation (interval between onset of the first negative peak and return to baseline of the last negative peak) in ≥1 nerve + ≥1 other demyelinating parameter in ≥1 other nerve.
Thresholds for distal CMAP duration prolongation (ms)	
LFF 2 Hz	Median > 8.4, Ulnar > 9.6, Peroneal > 8.8, Tibial > 9.2
LFF 5 Hz	Median > 8.0, Ulnar > 8.6, Peroneal > 8.5, Tibial > 8.3
LFF 10 Hz	Median > 7.8, Ulnar > 8.5, Peroneal > 8.3, Tibial > 8.2
LFF 20 Hz	Median > 7.4, Ulnar > 7.8, Peroneal > 8.1, Tibial > 8.0

Continued

2) Weakly supportive of demyelination	As in (1) but in only one nerve
Notes	
Note 1	Criteria established using a frequency filter bandpass of 2 Hz to 10 kHz for all parameters, except for distal CMAP duration prolongation, which has specific criteria for LFFs of 2, 5, 10, and 20 Hz. Skin temperature should be maintained at ≥33˚C (palm) and ≥30˚C (external malleolus).
Note 2—Extensiveness of motor nerve conduction studies	
Nerves tested	Median, ulnar (stimulated below the elbow), peroneal (stimulated below the fibular head), tibial (one side). If criteria are not fulfilled, the same nerves are tested bilaterally and/or the ulnar and median nerves are stimulated at the axilla and at Erb's point.
Motor conduction block/slowing not considered	Ulnar nerve across the elbow, peroneal nerve across the knee.
Conduction block between Erb's point and wrist	Requires ≥50% CMAP amplitude reduction in the ulnar and median nerves.
Specific techniques	Collision techniques to avoid ulnar nerve components in the median nerve CMAP, Martin-Gruber anastomosis test, ruling out ulnar nerve co-stimulation for median motor conduction block.
If distal CMAP amplitude < 1 mV	Recording from more proximal muscles innervated by the peroneal, median, ulnar, or radial nerves.
Abbreviations	CMAP = Compound muscle action potential, LFF = Low-frequency filter, LLN = Lower limit of normal, ULN = Upper limit of normal.

VandenBergh PYK.EAN/PNS (2021).

Some CIDP patients exhibit subclinical CNS involvement presenting with asymptomatic demyelination, or an associated MS condition [12]. Recent studies have suggested the existence of a condition termed combined central and peripheral demyelination (CCPD), with either one being symptomatic or asymptomatic [13]-[15], as seen in our patient [15]. The diagnosis of MS had been based on nonspecific white matter lesions identified on initial brain MRI, without clear dissemination in space and time, raising the possibility of an initial misdiagnosis. According to the McDonald criteria for MS, a diagnosis requires both clinical and radiological evidence of dissemination in space (lesions in at least two typical CNS regions) and dissemination in time (new lesions over time or simultaneous presence of enhancing and non-enhancing lesions) [6]. In contrast, our patient exhibited no additional CNS events, and the brain MRI findings remained stable, thus failing to meet these criteria fully.

Root hypertrophy, defined as an enlargement of spinal nerve roots, has been observed in patients with CIDP. The first case was reported by Matsuda *et al.* in 1996, describing a patient with progressive sensory disturbances, gait ataxia, and moderate distal muscle weakness. Autopsy findings showed marked and diffuse

enlargement of peripheral nerves and spinal roots, along with onion bulb formations in the sural nerve [16].

This hypertrophy is attributed to chronic, repetitive segmental demyelination and remyelination of peripheral nerves and nerve roots [17]. Along with Charcot-Marie-Tooth disease, CIDP is one of the most common causes of hypertrophic neuropathy [3]. Nerve root hypertrophy is observed in 57 to 80% of typical CIDP cases [11] [17] [18]. This hypertrophy is associated with more severe clinical manifestations, including increased muscle weakness and radicular pain [19].

Nerve conduction studies often show slow conduction velocities and multiple conduction blocks, while CSF analysis typically reveals elevated protein levels ranging from 0.55 to 9 g/l in reported cases (30 - 33], reflecting involvement of proximal nerve segments or roots. A study using ultrasonography to detect cervical nerve root hypertrophy found a significant correlation between the degree of hypertrophy and elevated CSF protein levels [11] [20]. Normal CSF analysis does not exclude the presence of nerve root hypertrophy [21]. Biopsy is recommended in unusual cases when diagnostic investigations are inconclusive, and for patients who are suspected of having CIDP but have not responded to treatment [10]. Differential diagnoses include CMT disease (which differs in age of onset and clinical course), and NF1, characterized by mild, length-dependent polyneuropathy, skin changes, ophthalmological findings, and a positive family history [12]. Other less common differential diagnoses include amyloidosis, lymphoma, and leprosy [3].

Nerve root hypertrophy in CIDP is typically mild or moderate, but in rare cases, it can be massive [11], leading to invasion of the cervicothoracic and lumbosacral foramina and causing spinal cord compression [22]. The dorsal roots are often spared [23]. In such cases, the diagnosis of CIDP may be delayed due to atypical clinical manifestations, including brisk deep tendon reflexes and spasticity [1].

Several cases of spinal cord compression by hypertrophic nerve roots, have been reported [4] [23] [24].

Our case presents CIDP with hypertrophic nerve roots, exhibiting both central and peripheral symptoms but without radiological evidence of spinal cord compression. Brain MRI shows a small number of nonspecific demyelinating lesions that remain stable over time. The exact cause of the central signs in our patient remains unclear. It could suggest infra-radiological spinal cord compression, or it could be explained by functional disturbances due to medullary distress not detected on MRI, given that the nerve roots are hypertrophic from their emergence, even inside the spinal canal (Image H).

Cranial nerves can also be affected by hypertrophy, notably the trigeminal nerve [25]. Although less common, the facial and hypoglossal nerves can also be involved, resulting in facial weakness and tongue atrophy, respectively [26], as seen in our patient. Aïdi *et al*. reported two clinical cases with massive hypertrophy of the nerve roots and brachial plexus, forming supraclavicular pseudo-tumoral masses. The patients also exhibited hypertrophy of the oculomotor nerves and branches of the trigeminal nerve, leading to exophthalmos and oculomotor paral-

ysis. Both patients responded well to corticosteroid therapy [27].

Non-invasive tools, such as MRI, are valuable for diagnosis, particularly in assessing the roots and plexuses using T1-weighted sequences (sagittal or coronal), T2-weighted sequences (axial or coronal), fat-saturated or STIR, 3D isotropic sequences, and post-contrast imaging [28] as the protocol used in our patient. The consensus guidelines from the EAN/PNS include MRI findings as supportive criteria for the diagnosis of CIDP [10]. In cases where CIDP is suspected, MRI can assist in confirming the diagnosis by detecting nerve hypertrophy and increased T2-weighted signal in the plexus. However, its routine clinical use is not currently recommended due to the limited evidence available from systematic studies [29]. These features typically present as contrast enhancement and hypertrophy of the cauda equina and cervical and lumbar plexuses, as observed in our patient (**Figure 1**). However, conventional MRI of roots and plexuses may miss the roots hypertrophy or may only capture restricted regions of the peripheral nerve trunks, potentially missing pathology in smaller nerves. As a result, the distribution and true incidence of affected peripheral nerves may be underestimated. Large field-of-view (FOV) three-dimensional (3D) MR neurography (MRN) has demonstrated greater sensitivity in evaluating the distribution and characteristics of peripheral nerve hypertrophy in patients with CIDP [30].

Patients with nerve roots hypertrophy in CIDP have a significantly longer disease duration than those without hypertrophy suggesting a correlation between chronicity and hypertrophy development. Typically, onset is insidious, with slow clinical progression, as observed in our patient [11]. In many cases, nerve hypertrophy appears to be inversely related to conduction velocity and directly correlated with disease duration but not disease severity [11]. The presence of nerve root hypertrophy may indicate a more severe disease, potentially requiring more aggressive treatment strategies [31].

CIDP with nerve roots hypertrophy respond better to immunotherapy, often requiring a combination of intravenous immunoglobulin (IVIG), plasma exchange, and/or corticosteroids. The therapeutic goal is to improve strength, motor performance, and quality of life. Corticosteroids and immunoglobulins were among the first treatments used for CIDP, and plasmapheresis has also demonstrated efficacy [3]. For patients with no or moderate diffuse nerve enlargement or proximal regional hypertrophy, corticosteroids may be preferred. In cases of significant diffuse or distal regional hypertrophy, IVIG can be a suitable option [32]. Monoclonal antibodies, such as alemtuzumab, rituximab, and eculizumab, have shown efficacy in some studies [12] [33]. In our case, rituximab was preferred due to its accessibility and tolerability. Decompressive surgery with laminoplasty may be indicated for severe spinal cord compression [4].

This case highlights critical diagnostic insights with significant clinical implications. The patient's atypical presentation, combined with nonspecific white matter lesions, emphasized the need for strict application of diagnostic criteria and longitudinal reassessment to avoid misdiagnosis. Spinal MRI with a radiculoplexus

protocol proved essential, as nerve root hypertrophy strongly supported CIDP and guided appropriate management. Clinically, the case underscores the value of early imaging and electrophysiological confirmation, especially in atypical cases, to enable timely diagnosis and targeted therapy. The favorable response to rituximab also suggests that alternative immunotherapies should be considered in refractory cases. Regular clinical, neurophysiological, and imaging follow-up is essential to monitor disease course and prevent complications such as spinal canal stenosis.

4. Conclusion

CIDP with nerve root hypertrophy is common, but other neuropathies with similar features should also be considered. Moreover, hypertrophy can lead to spinal cord compression, potentially causing central signs and complicating the diagnosis of CIDP. This underscores the importance of spinal MRI with a radiculoplexus protocol, as conventional MRI may fail to detect these abnormalities. In such cases, specific management, including surgical cord decompression, may be necessary.

Conflicts of Interest

The authors declare no conflicts of interest.

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