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Case Report

Spinal cord compression from hypertrophic nerve roots in chronic inflammatory demyelinating polyradiculoneuropathy – A case report

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ABSTRACT

Background: Spinal cord compression secondary to nerve root hypertrophy is often attributed to hereditary neuropathies. However, to avoid misdiagnosis, rare immune-mediated neuropathy such as chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) should not be overlooked. This report presents a case of multilevel nerve root hypertrophy leading to significant cord compression from CIDP.

Case Description: We report a 56-year-old gentleman with type two diabetes mellitus who presented with subacute cervical cord syndrome following a fall. Mixed upper and lower motor neuron features were noted on examination. Magnetic resonance imaging showed significant pan-spinal proximal nerve root hypertrophy, compressing the cervical spinal cord. Initial radiological opinion raised the possibility of neurofibromatosis type 1 (NF-1), but neurophysiology revealed both axonal and demyelinating changes that were etiologically non-specific. C6 root and sural nerve biopsies taken at cervical decompression displayed striking features suggestive for CIDP. Although NF-1 is the most observed condition associated with root hypertrophy, other important and potentially treatable differentials need to be entertained.

Conclusion: While rare, CIDP can cause significant spinal cord compression. Furthermore, clinical manifestations of CIDP can mimic those of inherited peripheral neuropathies. Neurologists and neurosurgeons should be aware of this condition to optimize subsequent therapeutic decision-making.

Keywords: Charcot-marie-tooth disease, Chronic inflammatory demyelinating polyradiculoneuropathy, Hypertrophic neuropathy, Neurofibromatosis

INTRODUCTION

Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) is immune-mediated neuropathy of peripheral nerves and nerve roots involving myelin sheath. The symptoms usually include symmetrical weakness of both proximal and distal muscles, sensory impairment, absent or diminished tendon reflexes, with signs of demyelination in nerve conduction studies, as well as in nerve biopsy specimens. These can be insidious, progressively increasing over 2 months with relapsing or chronic and progressive phase.[11] Nerve root hypertrophy can be found

distinctively in NF-1 and Charcot-Marie-Tooth (CMT) disease.[6] However, reports have also demonstrated spinal canal stenosis secondary to nerve roots hypertrophy in CIDP.[21] We report the case of extensive hypertrophic nerve roots causing cord compression where the radiological picture is similar to a background inherited neuropathy, but biopsy finding confirmed changes in line with inflammatory characteristics of CIDP.

CASE DESCRIPTION

A 56-year-old gentleman with a background history of type 2 diabetes mellitus (6 years of sound control and no associated complications), hypertension, and medically controlled hyperthyroidism presented to his local hospital with a 6-week history of progressive lower limb weakness, voiding difficulties, constipation, and burning paraesthesia of the soles of his feet, after a minor fall and head injury at work. He denied radicular pains in upper or lower limbs, or preceding neurological difficulties. He was otherwise systemically well. Due to the sphincter disturbance, he underwent emergency imaging of the lumbar spine which showed massive thickening of the cauda equina and lumbar roots and prompted imaging of the rest of his spine. This showed a similar picture with superimposed degenerative changes involving the cervical spine with evidence of mid-cervical cord compression due to a combination of discogenic disease and nerve root hypertrophy [Figures 1 and 2]. Imaging of the brain was comparatively unremarkable.

Given the massive root hypertrophy, an initial diagnosis of neurofibromatosis type 1 (NF-1) was entertained. The patient was referred urgently to the regional NF-1 unit in Greater Manchester. On examination, no cutaneous stigmata of NF-1 could be appreciated, nor was there evidence of peripheral nerve hypertrophy. Upper and lower motor neuron signs were present with evidence of mild symmetrical wasting of distal lower limb muscles, predominant pyramidal distribution of weakness in the lower limbs and to a milder extent in the upper limbs with global areflexia and extensor plantar responses. Vibration sense was impaired to the anterior superior iliac spine with a reduction in pinprick to the mid-thighs and altered sensation to T8 with some additional non-specific sensory disturbance over the ulnar distributions of the hands bilaterally. He was unable to stand or walk unaided. It was felt that the presentation was atypical for NF-1, and an opinion was sought from the regional neuromuscular service while arrangements were made for cervical decompression. Genetic testing for NF-1 was taken.

The neuromuscular service's subsequent review revealed similar examination findings [Table 1] although the patient was now globally weaker with Medical Research Council Grade 2 power scores in the proximal lower limbs. Neurophysiology showed evidence of a mostly length-dependent mixed axonal and demyelinating neuropathy, which was etiologically non-specific. The changes were compatible with diabetes, primary inflammatory neuropathy, or even NF-1. A biopsy of both sural nerve and proximal root biopsy was recommended at cervical cord decompression [Figure 3] to increase diagnostic yield and rule out the presence of dual pathology, that is, the neuropathy and radiculopathy having separate causation.

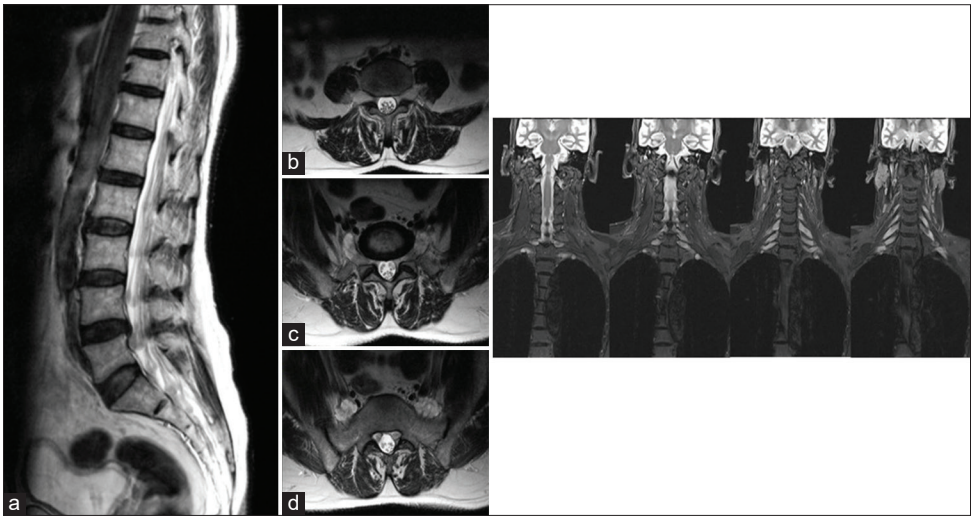

Figure 1: Left: Lumbar nerve root involvement. (a) Sagittal T2 (off midline) shows multiple intradural nerve root nodular lesions approaching the exit foramina. (b) Axial T2 through L3 foramina shows enlarged L3 roots at the exit foramina with some thickening of the intradural cauda equina roots. (c) Axial T2 through L4 exit foramina shows thickened L4 roots extending outwards from foramina, while intradural cauda equina roots are thickened. (d) Axial T2 through S1 segment of sacrum shows lobulated L5 roots ventral to sacral alar, while S1 nerve roots at lateral recesses are enlarged. **Right:** Coronal STIR of the brachial plexus in sequential images from posterior to anterior. All the cervical roots are thickened and hyperintense. The first image shows the intradural components in the spinal canal.

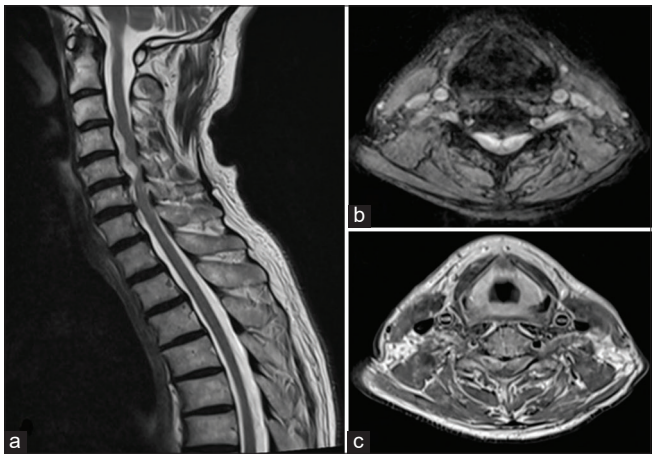

Figure 2: (a) Sagittal T2 shows hyperintense segmental lesions ventrolateral to the cervical cord at C4 and C5 vertebral levels compressing the cervical cord. (b) Axial T2 and (c) axial post-Gadolinium T1 at lower border of C5 vertebral level show bilateral C6 root lesions compressing the cervical cord which lies in the dorsal midline.

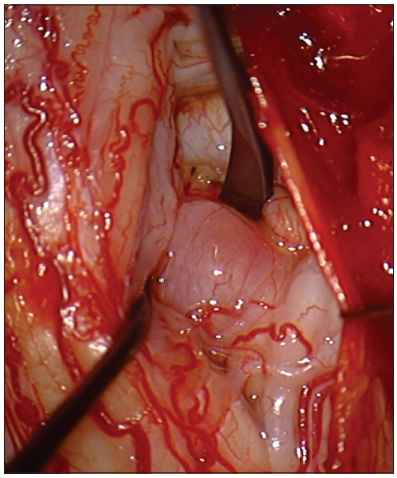

Figure 3: Cervical decompression: Intraoperative finding of grossly thickened nerve root.

Surgery was performed in standard prone position under total intravenous anesthesia with neurophysiology monitoring. A C4-C6 laminoplasty was performed. After opening the dura mater, the motor evoked potentials amplitudes in the lower limbs improved. The arachnoid was thickened, and large tumor-like swellings involving the C5 and C6 nerves bilaterally, ventral to cord could be appreciated. These appeared to arise from the motor roots as stimulated by the intraoperative nerve stimulator except the left C6 nerve root which was subsequently biopsied. A frozen section demonstrated dispersed large, myelinated axons throughout the lesional tissue, contradicting a diagnosis of a peripheral nerve sheath tumor. Instead of debulking, an expansive duraplasty, followed by expansive laminoplasty was therefore performed. A left sural nerve biopsy was also taken.

Histopathology of both the root and nerve biopsies showed striking features classical for a chronic inflammatory demyelinating neuropathy with marked loss of large, myelinated fibers, endoneurial edema, thinly remyelinated axons, Schwann cell onion skinning, and T cell inflammatory infiltrates [Figure 4]. The hypertrophy apparent in the nerve root was due to marked expansion of an edematous endoneurial matrix.

Six weeks after surgical decompression, the patient commenced monthly pulses of immunoglobulin therapy and is making slow and steady improvements to the point where he is mobilizing independently [Table 2]. Genetic tests for NF-1 subsequently returned negative.

DISCUSSION

This 56-year-old gentleman presented with subacute cervical myelopathy caused by a combination of degenerative spinal disease and proximal nerve root enlargement. Proximal nerve root enlargement has a broad differential diagnosis, of which CIDP is an important etiology.[10] CIDP is an acquired auto-immune polyneuropathy which is usually treatable with corticosteroids, intravenous immunoglobulin, or therapeutic plasma exchange.[12] The first description of CIDP as a chronic polyneuropathy can be found in the literature as early as 1958;[1] however, the pathogenesis is yet to be fully understood. The general consensus is an immune-mediated response directed to peripheral nerve myelin even though this hypothesis is not proven formally.[9,11,19,24] There is increasing evidence suggesting that the prevalence of CIDP is higher in patients with diabetes mellitus. Bril *et al*., in their Scopus-based literature review, highlighted the prevalence to be nine-fold higher when compared to CIDP in non-diabetic patients.[2] The most recent European multicenter cohort study by Rajabally *et al*. supported this increased prevalence and demonstrated a two-fold rise than in the general population.[16] However, it remains unclear whether the two disorders are pathogenetically correlated.[8] Understanding CIDP co-occurring in diabetes is essential as in contrast to diabetic polyneuropathy, CIDP is treatable.[23] Typically, CIDP presents with a slowly progressive proximal and distal motor and sensory neuropathy.[12] In the presented case, the patient denied any neurological symptoms before the fall. There were no pre-existing problems with instability, sensory dysfunction, or weakness. The relative paucity of symptoms relative to signs is often considered a diagnostic clue to genetic neuropathies.[10] However, there was no evidence of foot deformities, history of childhood gait abnormalities, family history of neurological difficulties, cutaneous or ophthalmological lesions suggestive of either Charcot-Marie-Tooth type 1 (CMT1) or NF-1, and two

Table 1: (A) Median motor conductions on both sides show severe distal motor latency delay and reduced motor potential amplitude, motor potentials are temporarily dispersed. Forearm motor conduction slowing on both sides and F latencies show moderate delay in latency. Ulnar motor conduction on both sides show delay in distal motor latency and reduced motor potential amplitude, dispersed particularly proximally, mild forearm motor conduction slowing, and moderate motor conduction slowing across the elbow on both sides. Moderate to severe reduction in lower limb motor conduction (common peronial? Tibialis) (B) Median digit III and ulnar digit V-no recordable sensory potentials. Radial sensory conduction on both sides-reduced sensory potential amplitude and no significant sensory conduction slowing. Lateral antebrachial cutaneous nerve sensory conduction on the right side-markedly reduced sensory potential with sensory conduction slowing. In both lower limbs show no recordable sensory potential. (C) Needle EMG done in tibialis anterior and gastrocnemius on both sides as well as right vastus lateralis in the lower limbs; includes first dorsal interosseous. EDC on both sides in the upper limbs and right deltoid. Active denervation is noted in right first dorsal interosseous and to a lesser degree in the right tibialis anterior. Prominent chronic denervation is noted in all these muscles and the changes are relatively more prominent in bilateral extensor digitorum communis in the upper limb and the left tibialis anterior in the lower limb.

A						
Motor nerve conduction studies						
Nerve	**Onset lat**	**Amp**	**Amp difference**	**CV**	**F-Flat**	**Distance**
	ms	**mV**	**%**	**m/s**	**ms**	**mm**
Medianus motor left						
Wrist-APB	7.13	2.3			52.4	
Elbow-Wrist	12.5	1.70	-26.1	43.8		235
Medianus motor right						
Wrist-APB	8.02	2.7			40.8	
Elbow-Wrist	14.1	1.95	-27.8	40.3		245
Ulnaris motor left						
Wrist-ADM	4.40	2.5			50.2	
Bl. elbow-Wrist	9.90	1.53	-38.8	41.8		230
Ab. Elbow-Bl. elbow	13.7	1.47	-3.9	23.7		90.0
Ulnaris motor right						
Wrist-ADM	5.10	3.3			4.79	
Bl. elbow-Wrist	10.7	2.7	-18.2	44.6		250
Ab. Elbow-Bl. elbow	13.5	2.5	-7.4	32.1		90.0
Deep peroneal motor left						
Fib Head-Tib Ant	9.48	3.2				
Pop Foss-Fib Head	12.7	3.2	0	21.7		70.0
Peroneus motor left						
Ankle-EDB	-	-				
Peroneus motor right						
Ankle-EDB	-	-				
Tibialis motor left						
Ankle-Abd hal	-	-				
Tibialis motor right						
Ankle-Abd hal	-	-				
Deep peroneal motor right						
Fib Head-Tib Ant	5.75	3.2				
Pop Foss-Fib Head	9.52	2.8		18.6		70.0
B						
Sensory nerve conduction studies						
Nerve	**Onset lat**	**Peak lat**	**-ve, Amp**	**CV**	**Distance**	
	ms	**ms**	**μV**	**m/s**	**mm**	
Digits to wrist sensory left						
Med III-Wrist	-	-	-			
Uln V-Wrist	-	-	-			
Digits to wrist sensory right						
Med III-Wrist	-	-	-			
Uln V-Wrist	-	-	-			

(*Contd*...)

Table 1: (*Continued*)

B					
Sensory nerve conduction studies					
Nerve	**Onset lat**	**Peak lat**	**-ve, Amp**	**CV**	**Distance**
	ms	**ms**	**μV**	**m/s**	**mm**
Stim 5-Wrist	-	-	-		
Lateral antebrachial sensory right					
Elbow-Forearm	3.06	3.81	1.10	40.2	123
Stim 2-Rec 2	2.93	3.40	1.30	42.0	123
Peroneus superficial sensory left					
Shin- Ankle	-	-	-		
Peroneus superficial Sensory Right					
Shin- Ankle	-	-	-		
Stim 2-Ankle	-	-	-		
Radialis sensory left					
Forearm- Wrist	1.74	2.44	2.7	-	
Forearm 2-Wrist	1.82	2.46	3.2	49.5	90.00
Radialis sensory right					
Forearm- Wrist	1.78	2.31	4.7	-	
Forearm 2-Wrist	1.84	2.31	5.0	46.2	85.0
Suralis sensory left					
Calf-Ankle	-				
Suralis sensory right					
Calf-Ankle	-				
Stim 2-Ankle	-				

C									
Muscle	**Interpretation**	**Spontaneous Activity**				**Voluntary Activity**			
		Fib	**PSW**	**Fasc**	**Amp**	**Dur**	**Poly**	**IP**	**Firing**
Left inteross dors I	Moderately inactive	0	0	1+	+	+	Normal	-	
Right deltoideus ant	Moderately inactive	0	0		+	+	Normal	-	
Right ext dig communis	Profoundly inactive	0	0	1+	++	+	Normal	-	1+
Left ext dig communis	Profoundly inactive	0	0		+	+	+	-	
Right inteross dorsal i	Moderately subacute	1+	1+		+	Normal	Normal	-	
Right vastus lat	Moderately inactive	0	0		+	Normal	+	-	
Left gastroc caput med	Moderately inactive	0	0		+	Normal	Normal	-	
Left tibialis anterior	Moderately inactive	0	0		+	Normal	Normal	-	1+
Right gastroc caput med	Moderately inactive	0	0		+	Normal	Normal	-	
Right tibialis anterior	Moderately inactive	0	1+		+	Normal	Normal	-	

genetic neuropathies that have been found to cause nerve root hypertrophy,[10]

Magnetic resonance imaging (MRI) remains an essential radiological diagnostic modality. CIDP may show hypertrophy of the cervical and lumbar spinal roots, brachial and lumbosacral plexuses, and contrast enhancement in active disease.[5] However, there is marked variability of nerve root enlargement seen with CIDP in the literature. Tanaka *et al.* identified that increased short tau inversion recovery (STIR) on MRI was more sensitive than the relative diameter of nerve roots.[22] In contrast, Oudeman *et al.* demonstrated that qualitative assessment of hypertrophy and signal hyperintensity on STIR or MR neurography was of limited value when differentiating CIDP from normal healthy volunteers. Rarely, as identified by occasional case reports, significant nerve root enlargement causing cord compression has been found in CIDP.[14,18]

In contrast, cervical cord compression by multilevel segmental neurofibromas is frequently seen[3,25] in NF-1, and hence, this was our first diagnostic impression based on MRI imaging. In our nationally commissioned NF-1 service, this disease pattern is seen in individuals with extensive internal tumor burden or in the so-called spinal variant where all segmental nerve roots are involved with little cutaneous manifestations of NF-1. However, neurofibromatosis, CIDP and even CMT1 can all demonstrate T2 hyperintensity and

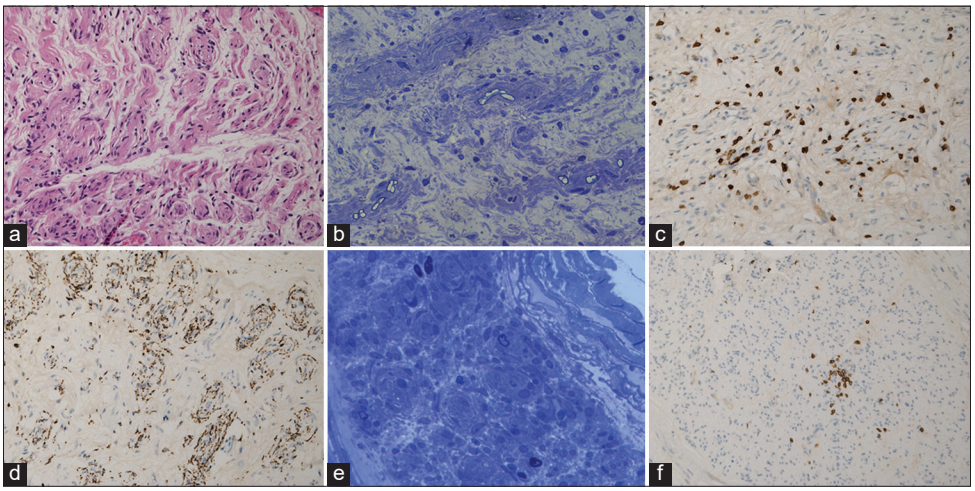

Figure 4: C6 nerve root biopsy: (a) H&E (hematoxylin and eosin) stained section showing endoneurial edema and a minifascicular architecture (in longitudinal and transverse profile) due to Schwann cell proliferation militating against a neoplastic or infectious process. (b) Semi-thin resin section (toluidine blue stained) showing a marked reduction in the density of large myelinated fibers and a prominent edematous stroma. The pathology is further characterized by thinly myelinated fibers surrounded by multi-layered sheaths of Schwann cells, which all favor a demyelinating process. (c) CD3 immunostaining for T lymphocytes showed prominent focal endoneurial lymphocytic activity. (d) CD68 immunostaining highlighted phagocytic activity co-localized to the cords of Schwann cells. **Sural nerve biopsy:** (e) Semi-thin resin section (toluidine blue stained) showing striking loss of large myelinated fibers, endoneurial edema and concentric Schwann cell proliferation. (f) CD3 immunostaining again showing a focal endoneurial inflammatory infiltrate of T-lymphocytes.

Table 2: MRC scores interpretation: (0) no contraction; (1) palpable contraction but no visible movement; (2) movement without gravity; (3) movement against gravity; (4) movement against a resistance lower than the resistance overcome by the healthy side; (5) movement against a resistance equal to the maximum resistance overcome by the healthy side.

Motor power assessment	4 months from symptom onset		6 months from symptom onset (3 weeks postoperative)	
	Right	Left	Right	Left
Shoulder abduction	5	5	4+	4+
Biceps	5	5	5	5
Triceps	5	5	4–	4–
Wrist extension	5	5	4–	4–
Wrist flexion	5	5	4–	4–
Finger extension	5	4	4–	4–
Intrinsic hand muscles	4+	4+	4–	4–
Hip flexion	2	2	4–	4–
Hip extension	5	5	4+	4+
Knee flexion	3	2	4–	4–
Knee extension	2	2+	4+	4+
Ankle dorsiflexion	4	4	4–	4–
Plantar flexion	5	5	4	4

gadolinium enhancement with nerve root hypertrophy, and thus it can be difficult to distinguish between them by MRI alone.[13,18,20] Furthermore, CMT1 can similarly cause significant cervical cord compression from pre-ganglionic nerve root hypertrophy.[7]

To differentiate the cause of hypertrophy, nerve conduction study and electromyography were performed. These also evaluate the presence, degree and pattern of conduction slowing along motor and sensory nerves in proximal and distal segments which indirectly provides evidence of demyelination, classically seen in CIDP and CMT1. Reduction in compound muscle action potential for motor nerves and sensory nerve action potential for sensory nerves represents the degree of axonal damage and loss of fibers. In our patient, there was electrophysiological evidence of underlying mixed demyelinating and axonal generalized large fiber polyradiculoneuropathy [Table 1]. Despite the absence of conduction block and the presence of uniform changes, the changes were supportive of CIDP,[17] particularly as the degree of slowing was not typical for that of CMT1.[10] However, a range of electrophysiological changes can be seen in NF-1, including a demyelinating neuropathy with axonal features.[4]

With ongoing diagnostic dilemma, the neurosurgical team proceeded with decompressive surgery for severe cord compression, keeping in mind there may be concomitant hereditary nerve root hypertrophy. Subsequent biopsy (C6 nerve root and sural nerve) revealed thinly myelinated large axons surrounded by multi-layered sheaths of Schwann cells traversing an edematous expanded stromal background, with

endoneurial T lymphocyte-mediated inflammatory activity, and phagocytic activity mediated either by macrophages and/or Schwann cells [Figure 4]. There was no evidence of a peripheral nerve sheath tumor. Onion bulb formation can be seen in both CMT1 and CIDP on neuropathology; however, the significant inflammatory changes, as seen in this case, are typically absent in hereditary CMT1.[15] Therefore, these features were supportive of an inflammatory demyelinating polyneuropathy. Moreover, a diagnosis of CIDP was reached based on radiological, electrophysiological,[17] and crucially in this case nerve biopsy.

In this case, we theorize that the acquired narrowed cervical spinal canal through disc disease and nerve root hypertrophy left little room for compensation following this gentleman's fall, resulting in subacute cervical myelopathy which ultimately led to his diagnosis of CIDP. This case further highlight that the degree of nerve enlargement on MRI imaging does not correlate with clinical symptoms, as before his fall, the gentleman was asymptomatic.

CONCLUSION

CIDP can possess diagnostic uncertainty with atypical presentations and shares similarities with genetic disorders such as neurofibromatosis and Charcot-Marie-Tooth disease. This case illustrates a relatively rare presentation of CIDP in the setting of spinal cord compression and the importance of keeping a broad differential diagnosis, taking into careful consideration the clinical, neurological, and pathological findings.

Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

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