Case Report

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

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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...
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distinctively in NF-1 and Charcot-Marie-Tooth (CMT) disease. However, reports have also demonstrated spinal canal stenosis secondary to nerve roots hypertrophy in CIDP. 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.
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. CIDP is an acquired auto-immune polyneuropathy which is usually treatable with corticosteroids, intravenous immunoglobulin, or therapeutic plasma exchange. The first description of CIDP as a chronic polyneuropathy can be found in the literature as early as 1958; 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. 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. 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. However, it remains unclear whether the two disorders are pathogenetically correlated. Understanding CIDP co-occurring in diabetes is essential as in contrast to diabetic polyneuropathy, CIDP is treatable. Typically, CIDP presents with a slowly progressive proximal and distal motor and sensory neuropathy. 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. 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 conduction velocities 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 peroneal? 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.

### 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  |               |     |        |          |
| Elbow-Wrist            | 12.5      | 1.70 | -26.1         | 43.8| 235    |          |
| Medianus motor right   |           |      |               |     |        |          |
| Wrist-APB              | 8.02      | 2.7  |               |     |        |          |
| Elbow-Wrist            | 14.1      | 1.95 | -27.8         | 40.3| 245    |          |
| Ulnaris motor left     |           |      |               |     |        |          |
| Wrist-ADM              | 4.40      | 2.5  |               |     |        |          |
| 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  |               |     |        |          |
| 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   |          |

### 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...)
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.¹⁰ 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.¹⁰ 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 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

| Nerve | Onset lat | Peak lat | -ve, Amp | CV | Distance |
|-------|-----------|----------|----------|----|----------|
| Stim 5-Wrist | - | - | - | |
| Lateral antebraclial sensory right | 3.06 | 3.81 | 1.10 | 40.2 | 123 |
| Elbow-Forearm | 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 | 1.74 | 2.44 | 2.7 | - | |
| Forearm-Wrist | 1.82 | 2.46 | 3.2 | 49.5 | 90.00 |
| Radialis sensory right | 1.78 | 2.31 | 4.7 | - | |
| Forearm-Wrist | 1.84 | 2.31 | 5.0 | 46.2 | 85.0 |
| Suralis sensory left | - | | | |
| Calf-Ankle | - | | | |
| Suralis sensory right | - | | | |
| Calf-Ankle | - | | | |

| 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 | - | - |
gadolinium enhancement with nerve root hypertrophy, and thus it can be difficult to distinguish between them by MRI alone.\textsuperscript{13,18,20} Furthermore, CMT1 can similarly cause significant cervical cord compression from pre-ganglionic nerve root hypertrophy.\textsuperscript{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, specifically as the degree of slowing was not typical for that of CMT1.\textsuperscript{10} However, a range of electrophysiological changes can be seen in NF-1, including a demyelinating neuropathy with axonal features.\textsuperscript{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. Therefore, these features were supportive of an inflammatory demyelinating polyneuropathy. Moreover, a diagnosis of CIDP was reached based on radiological, electrophysiological, 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 highlights 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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