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A Case of Near-Missed Diagnosis of Multiple Sclerosis Presented as Lumbar Disc Herniation with Radiculopathy

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Abstract

This case describes a patient who was initially diagnosed In Spine Surgical Hospital as lumbar Disc Herniation (LDH) and planned for surgical intervention but was later found to have Multiple Sclerosis (MS) after further neurological evaluation. It highlights the importance of maintaining a high index of suspicion, repeating the neurological examination, and involving a multidisciplinary team in intractable lumbar disc cases, particularly before proceeding to surgery. It is well established that the earlier the diagnosis of MS the better is outcome with proper management on the long-term prognosis. In this patient, early labeling as a surgical case contributed to the failure to identify Lhermitte's sign and pyramidal signs that may appear later. Further assessment, together with neurophysiological studies including Nerve Conduction Studies (NCS) and visual evoked potentials (VEP), supported central nervous system involvement and helped confirm MS while excluding lumbar disc herniation as the cause of the presentation. This case reflects a clinical challenge. In our experience, patients with central nervous system pathologies may be misidentified as surgical lumbar candidates when initial assessments are focused primarily on radicular symptoms, thus delaying the diagnosis of MS.

Keywords: Multiple sclerosis (MS); lumbar disc disorder with radiculopathy (LDDR); lumbar disc herniation (LDH); Lhermitte's sign (LS); upper motor neuron (UMN); lower motor neuron (LMN); pyramidal signs (PS); McDonald criteria.

Case Presentation

A 43-year-old male with a history of hypertension presented with an 8-months history of progressively worsening neurological symptoms, including recurrent electric shock-like sensations extending along the lumbar spine into the lower extremities. The symptoms initially started with sciatic-like pain in the left leg and later involved the right leg. Two weeks after symptom onset, a lumbar spine X-ray, followed by an MRI, revealed a lumbar disc herniation (LDH). Despite treatment with Non-Steroidal Anti-inflammatory (NSAIDs), muscle relaxants, and multiple physiotherapy modalities, his condition continued to progress. On further history, the patient reported difficulty walking, which was attributed to severe pain. He subsequently underwent an epidural injection for pain management. There were no urinary symptoms. He was treated with Gabapentin and various analgesics without any improvement. After approximately seven months of persistent symptoms, the patient was offered surgical decompression based on the presumed diagnosis of LDH. Such presentations may mimic mechanical spinal pathology and contribute to delayed or incorrect diagnosis of underlying neurological conditions, including MS (Beiske et al., 2004; Rolak & Fleming, 2007; Solomon et al., 2016). On that basis, the patient sought a second opinion from a neurologist, hoping for an alternative management approach. At that time, despite prolonged conservative management, no clinical improvement was reported or demonstrated.

Neurological Reassessment

On neurological assessment, the patient was alert, oriented, and cooperative. He came walking without any support, driving his own car. Neurological examination revealed mild slow spastic gait, pyramidal signs in the lower limbs, with increased tone proximally more than distally in the left lower limbs. Deep tendon reflexes were exaggerated in both knees, more pronounced on the left side, and a suspected unilateral left-sided Babinski sign. Sensory examination revealed allodynia in both lower

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limbs; however, this did not follow a dermatomal or radicular distribution, nor any defined sensory level. Neck flexion elicited an electric shock-like sensation suggestive of Lhermitte's sign (LS). There was no evidence of cortical sensory loss in all four limbs. Cranial nerves, upper limb examination, and cerebellar assessment were all within normal limits. There was no past medical history of ocular, visual, or bulbar symptoms. On further detailed history, the patient reported intermittent neck pain, electric shock-like pain along the cervicothoracic spine over the past two years which he was not asked nor claimed.

These findings were suggestive of an Upper Motor Neuron (UMN) pathology rather than Lower Motor Neuron (LMN). Following the clinical presentation, neurological assessment, and imaging findings, the working diagnosis was revised to MS and was confirmed using established diagnostic criteria, demonstrating clinical and radiological dissemination in space and time (Dekker & Wattjes, 2017; Thompson et al., 2018).

Investigations

To confirm the final diagnosis and rule out other probable causes, a thorough diagnostic process was used. This included brain and spinal cord imaging, blood tests, and a detailed clinical re-evaluation. A baseline laboratory assessment was performed to exclude metabolic, infectious, and other inflammatory etiologies of the patient's condition. Standard biochemical measures, including liver, thyroid function tests, and Blood Sugar, were unremarkable.

Neurophysiological Studies and Visual Evoked Potential

Immediate nerve conduction studies (NCS) did not support peripheral neuropathy or radiculopathy. Lower-limb NCS showed normal results, ruling out LMND [Figure 1] and [Figure 2].

Motor Nerve Conduction:

Nerve and Site	Latency	Amplitude	Segment	Latency Difference	Distance	Conduction Velocity
Peroneal.R						
Ankle	3.1 ms	13.3 mV	Extensor digitorum brevis-Ankle	3.1 ms		
Fibula (head)	9.0 ms	11.8 mV	Ankle-Fibula (head)	5.9 ms	300 mm	51 m/s
Popliteal fossa	10.4 ms	11.8 mV	Fibula (head)-Popliteal fossa	1.4 ms	80 mm	57 m/s
Tibial.R						
Ankle	3.1 ms	13.1 mV	Abductor hallucis-Ankle	3.1 ms		
Popliteal fossa	10.1 ms	10.0 mV	Ankle-Popliteal fossa	7.0 ms	350 mm	50 m/s
Tibial.L						
Ankle	3.1 ms	15.5 mV	Abductor hallucis-Ankle	3.1 ms		
Popliteal fossa	9.8 ms	12.9 mV	Ankle-Popliteal fossa	6.7 ms	350 mm	52 m/s
Peroneal.L						
Ankle	3.3 ms	12.6 mV	Extensor digitorum brevis-Ankle	3.3 ms		
Fibula (head)	9.0 ms	11.5 mV	Ankle-Fibula (head)	5.7 ms	300 mm	53 m/s
Popliteal fossa	10.4 ms	11.4 mV	Fibula (head)-Popliteal fossa	1.4 ms	80 mm	57 m/s

F-Wave Studies

Nerve	M-Latency	F-Latency
Peroneal.R	3.1	43.2
Tibial.R	3.9	41.1
Tibial.L	3.5	42.8
Peroneal.L	3.1	43.1

Figure 1. Motor Nerve Conduction Study of the lower limbs, with F-wave, showing no evidence of motor radiculopathy or neuropathy.

Afterward, visual evoked potentials (VEP) confirmed a delayed left-eye P100 of more than 120 milliseconds and a normal right eye [Figure 3].

MRI of the brain confirmed the diagnosis of a demyelinating disease of the central nervous system, mostly MS. It showed multifocal T2/FLAIR hyperintense lesions in the periventricular, deep white matter, and subcortical white matter of both frontoparietal and temporal lobes, oriented perpendicular to the ventricles, giving a Dawson's fingers appearance. A focal similar lesion in the right cerebral peduncle and linear signal-intensity changes

Sensory Nerve Conduction:

Nerve and Site	Onset Latency	Peak Latency	Amplitude	Segment	Latency Difference	Distance	Conduction Velocity
Superficial peroneal.R							
Ankle	1.9 ms	2.2 ms	21 µV	Dorsum of foot-Ankle	1.9 ms	100 mm	54 m/s
Sural.R							
Lower leg	1.9 ms	2.3 ms	30 µV	Ankle-Lower leg	1.9 ms	100 mm	53 m/s
Sural.L							
Lower leg	2.0 ms	2.6 ms	30 µV	Ankle-Lower leg	2.0 ms	100 mm	50 m/s
Superficial peroneal.L							
Ankle	1.9 ms	2.2 ms	20 µV	Dorsum of foot-Ankle	1.9 ms	100 mm	53 m/s

Interpretation:

Bilateral lower limb motor and sensory nerve conduction study including F waves were within normal limits.

Conclusion:

This electroneurophysiology study is within normal limits.

Single NCS/EMG study along cannot rule out or confirm the diagnosis.
Follow up and clinical correlation is recommented.

Figure 2. Sensory NCS of the lower limbs, with a conclusion of a normal study.

Visual Evoked Potentials:

LEFT EYE MEASUREMENTS

Text	Lat N75 ms	Lat P100 ms	Lat N145 ms	PP Amp 75-100 uV
Oz-Fz	1:N75 84.5	1:P100 125	1:N145 164	1:N75 P100 7.81

RIGHT EYE MEASUREMENTS

Text	Lat N75 ms	Lat P100 ms	Lat N145 ms	PP Amp 75-100 uV
Oz-Fz	3:N75 60.0	3:P100 106	3:N145 152	3:N75 P100 8.69

Results:

This pattern reversal Visual Evoked Potential Study shows prolonged P100 latency over left eye. Right P100 latency is normal.

Clinical and MRI correlation is recommented.

Figure 3. Visual Evoked Potential (VEP) confirming prolonged P100 latency on the left eye, as compared with the normal right eye.

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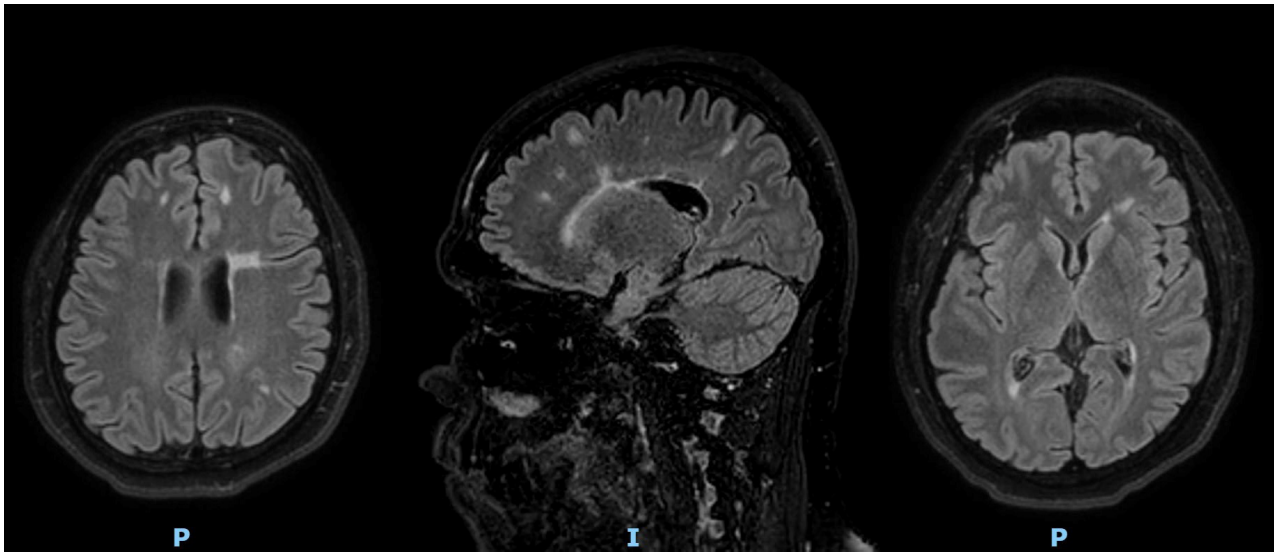

Figure 4. MRI brain, axial and coronal FLAIR, showing the extensive demyelinating lesions with the typical perpendicular Dawson's appearance.

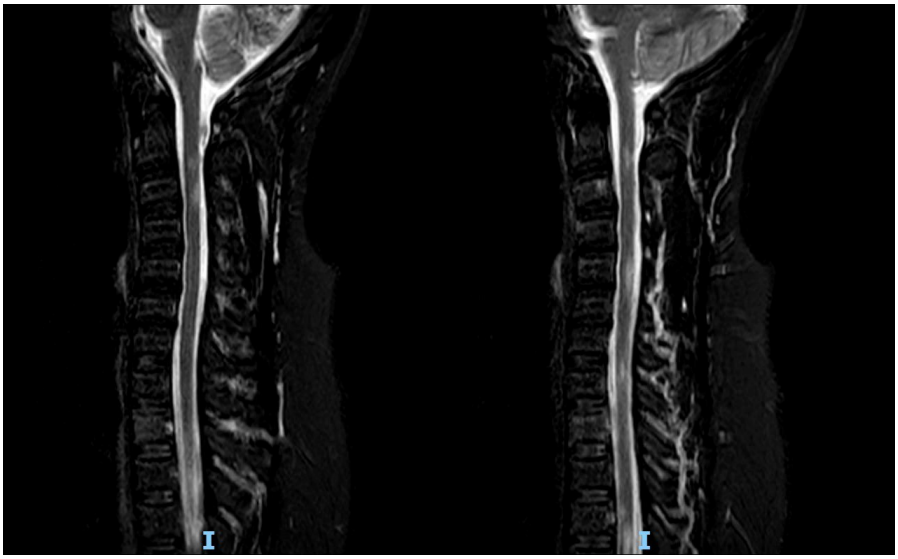

Figure 5. MRI cervical spine showing three-level lesions (C2/3, C5, C6/7), confirming MS.

in the left pons were typical for multiple sclerosis (MS) [Figure 4].

MRI of the cervical spine [Figure 5] showed multifocal short-segment altered signal intensity within the cervical spinal cord at the C2–3 junction, and C6- 7 on the left side, and C5 on the right side. These findings correlated with the patient's electric shock-like sensations triggered by neck flexion and supported the clinical diagnosis of Lhermitte's sign (Dekker & Wattjes, 2017; Kanchandani & Howe, 1982; Wattjes et al., 2015). Other multifocal thoracic spine similar lesions [Figure 5] are scattered from T2-T10 as well, generating the clinical presentation. There was no enhancement seen in the post-contrast study.

Discussion

The diagnostic overlap between early MS and Lumbar Disc Disorder with Radiculopathy (LDDR) frequently leads to clinical anchoring, where symptoms are prematurely attributed to structural abnormalities identified on lumbar imaging (Brownlee et al., 2017; Rolak & Fleming, 2007; Tosi et al., 1998). As illustrated by Tosi et al., even a single cervical demyelinating lesion may closely mimic radiculopathy, prompting clinicians to

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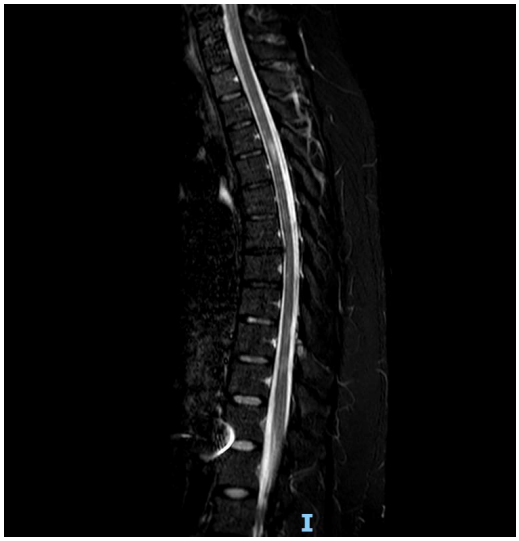

Figure 6. Figure 6. MRI thoracic spine: multiple intramedullary demyelinating lesions scattered along the thoracic area

favor a mechanical etiology over central nervous system pathology (Beiske et al., 2004; Tosi et al., 1998). This bias is particularly consequential in patients presenting with lower-limb paresthesia, where the early application of a "surgical label" may reduce attention to subtle upper motor neuron signs and non-dermatomal sensory patterns (Beiske et al., 2004; Rolak & Fleming, 2007).

A shift toward central nervous system involvement is often suggested by key clinical findings such as Lhermitte's sign. This transient, shock-like sensation triggered by neck flexion reflects dorsal column dysfunction within the cervical spinal cord (Beckmann et al., 2015; Kanchandani & Howe, 1982). Although not specific to MS, its reproducibility on examination should prompt reconsideration of a central lesion rather than isolated spinal radiculopathy.

Magnetic resonance imaging (MRI) remains the cornerstone for diagnosing MS and demonstrating dissemination in space according to the 2017 McDonald criteria (Dekker & Wattjes, 2017; Thompson et al., 2018). Typical findings include ovoid lesions in periventricular, juxtacortical, infratentorial, and spinal cord regions, which support a demyelinating process (Dekker & Wattjes, 2017; Thompson et al., 2018). However, imaging must be interpreted cautiously, as nonspecific white matter changes and age-related findings may confound the diagnosis (Brownlee et al., 2017; Dekker & Wattjes, 2017; Solomon et al., 2019; Thompson et al., 2018) except in the presence of extensive spinal cord lesions. In this context, radiological findings should always be integrated with a detailed neurological examination.

This case underscores the importance of a multidisciplinary approach in patients with persistent or atypical "lumbar" complaints, particularly when symptoms are non-dermatomal or disproportionate to imaging findings. Neurological consultation prior to operative intervention is essential, with careful and repeated clinical assessment that may reveal previously overlooked (or lately appeared during progression) pyramidal signs, gait abnormalities, or LS. Other multifocal thoracic-spinal similar lesions [Figure 6] are scattered from T2–T10 as well, contributing to the clinical presentation. No enhancement was seen on the post-contrast study. Such an approach can reduce unnecessary surgical procedures and facilitate earlier initiation of appropriate immunotherapy (Gaitán & Correale, 2019; Solomon et al., 2016; Solomon et al., 2019).

We find one case presentation documenting three levels cervical, thoracic then lumbar spine surgeries for a patient who is diagnosed later as MS, and one more study presented in 1992 (Korovessis et al., 1996; Ramirez-Lassepas et al., 1992), retrospect follow up of 11 MS patient presented as acute non discogenic radicular pain who were followed up from 6months to 15 years, from 1st presentation to final diagnosis, 6 of them had lumbosacral radiculopathy (Korovessis et al., 1996).

To summarize our perspective, the causes behind such misdiagnosis can be highlighted as

practical points to help identify similar cases presenting to neurosurgical or orthopedic clinics:

1. The initial preliminary diagnosis made at the first visit is often treated as a final diagnosis; studies suggested this fact in a considerable proportion. Adhering to the

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initial diagnosis without re-evaluation and re-investigation increases the risk of diagnostic error (Gaitán & Correale, 2019; Solomon et al., 2019).

2. Most neurological diseases with progressive patterns require repeated history taking and neurological examination at every visit, as new symptoms and signs may evolve and otherwise go unnoticed (Brownlee et al., 2017; Solomon et al., 2019).

3. Multidisciplinary teamwork, particularly in intractable cases and collaboration between neurology, neurosurgery, and physiotherapy, is essential in identifying misdiagnosis and guiding appropriate management plans (Gaitán & Correale, 2019).

4. Maintaining a high index of suspicion and broadening the differential diagnosis toward non-surgical etiologies is crucial. Continuous educational programs for surgical specialties are important, as prolonged practice may shift focus away from internal medicine differentials (Gaitán & Correale, 2019; Rolak & Fleming, 2007).

5. Our key message emphasizes fundamental clinical principles to avoid delayed diagnosis. We advocate for retrospective and multidisciplinary studies initiated from Surgical Spinal centers to determine the true prevalence of similar cases and improve statistical understanding and clinical awareness (Gaitán & Correale, 2019; Solomon et al., 2019).

Conclusion

This case signifies the diagnostic challenges that take place, especially when the very initial signs of MS tend to present as lumbar disc disease with radiculopathy. It is the findings of unusual sensory symptoms, not tracing over an exact distribution on a specified dermatome, Lhermitte's sign, and involvement of the pyramidal tract that should raise concern and urge a thorough neurological assessment for a possible hidden disease of the central nervous system. A thorough clinicoradiological correlation, potentially supplemented by neurophysiological investigations, is crucial to avoid misdiagnosis and the performance of unwarranted spinal surgery. Furthermore, a multidisciplinary assessment is particularly valuable for specific patients experiencing progression in their symptoms before escalation from conservative to surgical management, as the prompt identification of MS facilitates the earlier commencement of effective treatment and the enhancement of functional outcomes.

Disclosure

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