

A Case Report of Spinal Cord Edema and Cervical Spondylosis Masquerading as Chronic Inflammatory Demyelinating Polyneuropathy (CIDP)

Muhammad Sohail Ajmal Ghoauri¹, Nauman Ismat Butt^{2*}, Dur-e-Sabeh³, Muhammad Bilal Rasheed², Muhammad Umair Javed⁴, Faizan Ali Khan²

¹Department of Neurology, Bahawal Victoria Hospital, Quaid-e-Azam Medical College, Bahawalpur, Pakistan.

²Department of Medicine & Allied, Azra Naheed Medial College, Superior University, Kot Arian, Lahore, Pakistan.

³Department of Medicine, Bahawal Victoria Hospital, Quaid-e-Azam Medical College, Bahawalpur, Pakistan.

⁴Department of Pulmonology, Bahawal Victoria Hospital, Quaid-e-Azam Medical College, Bahawalpur, Pakistan.

Abstract: A 64-year-old previously-healthy male presented with 2-year history of progressive neurological symptoms of numbness and muscle weakness involving all 4 limbs. There was gait disturbance, urinary and fecal incontinence. On examination, the left upper limb had normal tone, diminished deep tendon reflexes and power of 4/5 with wasting both in proximal and distal muscles. There were reduced pinprick pain and temperature sensations below the elbow bilaterally with intact vibration and proprioception. Both lower limbs had increased tone, diminished deep tendon reflexes, power of 3/5 with wasting both in proximal and distal muscles with unequivocal plantar reflex bilaterally. There were reduced pinprick pain and temperature sensations below the knee on right and below the ankle on left with intact vibration and proprioception. Nerve Conduction Studies (NCS) were done which showed axonal type of denervation in all limbs. MRI scan of Cervical Spine showed T2W hyperintense signals and narrowing of spinal canal from C3 to C7 region. The final diagnosis was spinal cord edema in the cervical region and cervical spondylosis causing spinal cord compression.

Keywords: Chronic Inflammatory Demyelinating Polyneuropathy (CIDP), Spinal cord edema, Cervical spondylosis, Nerve Conduction Studies (NCS), MRI scan, Case report.

INTRODUCTION

Neurological disorders are a major cause of disability and mortality worldwide [1]. Spinal cord compression leads to progressive neurologic disability and subsequently affects life quality [2]. Spinal cord edema is a rare disorder that can be caused by various etiologies including infections, autoimmune disorders, and trauma [3]. Cervical spondylosis is another common degenerative condition that can cause spinal cord compression leading to neurological symptoms [4]. Because, the early symptoms of spinal cord edema and cervical spondylosis leading to cervical myelopathy may be non-specific and subtle a significant delay may occur before a correct diagnosis may be made [5]. Other causes of a delayed diagnosis includes overlapping, incomplete neurological assessment by the primary physicians and a lack of awareness regarding this condition [5, 6]. Behrbalk *et al.* reported that there was a 2.2 year delay between symptoms onset and diagnosis. Furthermore, majority of patients have hand numbness and may be initially diagnosed and treated for carpal tunnel syndrome [6]. Surgical decompression of spinal cord compression may lead to halting disease progression but the spinal regenerative capacity is limited and long-standing disease is often irreversible [2]. Therefore delay in treatment is

associated with poor prognosis and substantial disability.

CASE PRESENTATION

We present the case of a 64-year-old previously-healthy male who presented with 2-year history of progressive neurological symptoms starting from numbness and tingling of right lower limb which progressed to muscle weakness and wasting of proximal and distal parts involving both lower and upper limbs in asymmetrical pattern causing disturbances in gait. The patient also reported urinary and fecal incontinence for last two months. There was no history of trauma, episodes of unconsciousness, fever, weight loss, night sweats, joint pains, cough or any palpable lumps. He used to work as a laborer but had quit his job due to his disease. He was married with 4 children and denied extra-marital sexual contact. He did not smoke or use illicit drugs.

On examination, the patient was conscious and cooperative, well oriented in time, space and person. The right upper limb had normal tone, reflexes, and muscle power. The left upper limb had normal tone, diminished deep tendon reflexes and power of 4/5 with wasting both in proximal and distal muscles. There were reduced pinprick pain and temperature sensations below the elbow bilaterally with intact vibration and proprio-

*Address correspondence to this author at the Department of Medicine & Allied, Azra Naheed Medial College, Superior University, Kot Arian, Lahore, Pakistan. Email: nauman_ib@yahoo.com

ception. Both lower limbs had increased tone, diminished deep tendon reflexes, power of 3/5 with wasting both in proximal and distal muscles with unequivocal plantar reflex bilaterally. There were reduced pinprick pain and temperature sensations below the knee on right and below the ankle on left with intact vibration and proprioception. He had a high-stepping gate. All cranial nerves and cerebellar functions were intact. The cardiac, respiratory and abdominal examinations were unremarkable. Based on clinical findings, an initial diagnosis of Chronic Inflammatory Demyelinating Polyneuropathy (CIDP) was made and the patient was admitted.

On investigation, CBC, ESR, LFTs, RFTs and urinalysis were normal. Serology for TPHA, HBV, HCV and HIV were negative. Serum B12, HbA1c and TSH were normal. Nerve Conduction Studies (NCS) were done which showed axonal type of denervation in all limbs. There were no signs of demyelination on NCS to suggest CIDP. MRI scan of Cervical Spine showed T2W hyperintense signals and narrowing of spinal canal from C3 to C7 region indicating spinal canal edema and cervical spondylosis as shown in Fig. (1).



Fig. (1). MRI Scan of Cervical Spine Showing T2W Hyperintense Signals and Narrowing of Spinal Canal from C3 to C7.

Based on findings of NCS and MRI scan, the final diagnosis was spinal cord edema in the cervical region and cervical spondylosis causing partial spinal cord compression and a neurosurgical opinion was sought. The patient and his attendants refused neurosurgery and a plan was made for conservative therapy encompassing bed-rest, NSAIDs, guided physiotherapy and rehabilitation programme. On follow up, the patient was partially improved, was tolerating treatment without any adverse effects and was doing regular physiotherapy.

DISCUSSION

Chronic inflammatory demyelinating polyneuropathy (CIDP) is an uncommon, auto-immune disease caused by aberrant immune response leading to demyelination of peripheral nerves [7]. The exact etiology and triggering factors of CIDP remains unknown.

The clinical features include progressively worsening muscle weakness, impairment in sensory function and diminished deep tendon reflexes [8]. CIDP is a chronic disease with a non-specific variable disease history with gradual progression, stepwise progression or a relapsing-remitting course [7, 8]. Electromyography (EMG) and Nerve Conduction Studies (NCS) are usually used to diagnose CIDP [9]. Other differential diagnosis that need to be considered in a patient with progressive neurologic decline include Sub-acute Combined Spine Degeneration, Motor Neuron Disease, Neuro-syphilis, and spinal myelopathy. Our patient had progressively worsening neurological findings and initially we suspected a diagnosed of CIDP on basis of diminished deep tendon reflexes, unequivocal plantars and asymmetrical sensorimotor loss. However based on findings of NCS and MRI scan, our patient was diagnosed with partial spinal cord compression caused by spinal cord edema in the cervical region and cervical spondylosis making our patient's presentation a rare and atypical one for Spinal Myelopathy.

CONCLUSION

In conclusion, we present a case of a patient with progressive neurological symptoms who was initially thought to have Chronic Inflammatory Demyelinating Polyneuropathy (CIDP) but later on work-up spinal cord edema and cervical spondylosis were identified as the underlying etiology. This case highlights the importance of keeping this in the differential diagnosis of when a patient presents with progressive neurological symptoms so that timely diagnosis may be made.

CONFLICT OF INTEREST

Declared none.

ACKNOWLEDGEMENTS

Declared none.

REFERENCES

- [1] Davies BM, Mowforth OD, Smith EK, Kotter MR. Degenerative cervical myelopathy. *BMJ* 2018; 360: k186. doi: 10.1136/bmj.k186.
- [2] Oh T, Lafage R, Lafage V, et al. Comparing quality of life in cervical spondylotic myelopathy with other chronic debilitating diseases using the short form survey 36-health survey. *World Neurosurg* 2017; 106: 699-706. doi: 10.1016/j.wneu.2016.12.124.
- [3] Mora-Boga R, Vázquez-Muñíos O, Pérttega-Díaz S, et al. Neurological recovery after traumatic spinal cord injury: Prognostic value of magnetic resonance. *Spinal Cord* 2022; 60(6): 533-9. doi: 10.1038/s41393-022-00759-0.
- [4] Iyer A, Azad TD, Tharin S. Cervical spondylotic myelopathy. *Clin Spine Surg* 2016; 29(10): 408-14. doi: 10.1097/BSD.0000000000000397.

- [5] Tracy JA, Bartleson JD. Cervical spondylotic myelopathy. *Neurologist* 2010; 16(3): 176-87. doi: 10.1097/NRL.0b013e-3181da3a29.
- [6] Behrbalk E, Salame K, Regev GJ, Keynan O, Boszczyk B, Lidar Z. Delayed diagnosis of cervical spondylotic myelopathy by primary care physicians. *Neurosurg Focus* 2013; 35(1): E1. doi: 10.3171/2013.3.FOCUS1374.
- [7] Broers MC, Bunschoten C, Nieboer D, Lingsma HF, Jacobs BC. Incidence and prevalence of chronic inflammatory demyelinating polyradiculoneuropathy: A systematic review and meta-analysis. *Neuroepidemiology* 2019; 52(3-4): 161-72. doi: 10.1159/000494291.
- [8] Hafsteinsdottir B, Olafsson E. Incidence and natural history of idiopathic chronic inflammatory demyelinating polyneuropathy: A population-based study in iceland. *Eur Neurol* 2016; 75(5-6): 263-8. doi: 10.1159/000445884.
- [9] Bunschoten C, Jacobs BC, Van den Bergh PYK, Cornblath DR, van Doorn PA. Progress in diagnosis and treatment of chronic inflammatory demyelinating polyradiculoneuropathy. *Lancet Neurol* 2019; 18(8): 784-94. doi: 10.1016/S1474-4422(19)30144-9.

Received: March 26, 2023

Revised: September 14, 2023

Accepted: September 14, 2023