

CERVICAL MYELOPATHY DUE TO CERVICAL DISC HERNIATION-CASE REPORT

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Abstract

Cervical myelopathy is a progressive degenerative disorder resulting from spinal cord compression, most commonly due to spondylotic changes and intervertebral disc herniation. It presents with a wide spectrum of neurological deficits, including walking disturbance, limb weakness, sensory impairment, and upper motor neuron signs. Early recognition is essential, as advanced disease may lead to irreversible neurological damage. We present the case of a 63-year-old woman with progressive quadriparesis, walking impairment, and sensory disturbances following minor trauma. Neurological examination revealed upper motor neuron signs and intrinsic hand muscle atrophy. Magnetic resonance imaging of the cervical spine demonstrated multilevel degenerative disc disease with severe spinal canal stenosis and spinal cord compression, accompanied by intramedullary signal changes consistent with compressive myelopathy. Despite neurosurgical indication for operative treatment, the patient declined surgery and was managed conservatively with physical therapy, resulting in partial clinical improvement. This case highlights the importance of thorough clinical evaluation and neuroimaging in the diagnosis of cervical myelopathy and underscores the need for timely recognition and appropriate management to prevent long-term disability.

Key words: Cervical myelopathy, cervical spondylosis, spinal canal stenosis, spinal cord compression, magnetic resonance imaging

Introduction

Cervical myelopathy (CM) is primarily a degenerative process that results in myelopathic and/or radiculopathic syndromes. [1] It is a clinical syndrome resulting from dysfunction of the spinal cord.[2] Cervical myelopathy due to spondylotic and disc herniation is a disturbance or pathological change in the spinal cord, secondary to degenerative changes in the cervical spine. Compression of the spinal cord can result from a number of stenotic processes, including disc protrusion, degeneration causing spur formation within the canal, ossification of ligaments and connective tissues, hypertrophy of facets, and infoldings of joint capsules.

The four primary components that contribute to the development of cervical spondylotic myelopathy (CSM) are: (1) stenosis of the central spinal canal, (2) focal projections into the canal (spurs, osteophytes, or sequestration of vertebral discs), (3) damage to intrinsic spinal or radicular circulation, and (4) chronic obstruction of cerebrospinal fluid (CSF) flow.[3]

The most common levels for degenerative spinal stenosis and spondylosis in the cervical spine are C5 and C6, due to cervical cord enlargement for the brachial plexus and frequent degenerative changes at these levels.[4] These changes can be associated with compression of the spinal cord or its vascular supply via stenotic processes, causing ischemia and demyelination.[5]

However, cervical myelopathy can theoretically be caused by any disruption of neural pathways in the cervical spine, which may lead to a wide array of presenting symptoms. Associated neurological deficits are more prominent in patients with congenital narrowing of the spinal canal, segmental instability, or disc protrusions.[5] Stenosis is often regarded as present when the sagittal canal diameter is less than 13 mm.[1,6] As much of the degeneration occurs in the lateral vertebral structures, the corresponding lateral columns of the spinal cord are most commonly affected, including the corticospinal tract, the anteromedial portion of the posterior columns, and the nerve roots.

Early symptoms of cervical myelopathy include weakness, diminished dexterity, walking instability, and decreased sensation, while later symptoms include spasticity and bowel and urinary

incontinence.[2] Rare presentations include acute-onset paraplegia, false localizing sciatica-like leg pain, and hypoventilation.[7,8] Upper motor neuron (UMN) signs are typically present below the level of the lesion. Common UMN signs include hypertonia, clonus, Hoffman's sign, and the Babinski response. There are no pathognomonic findings; however, classic neurological examination signs include pathological reflexes such as ankle clonus, Babinski sign, and Hoffman sign.[9] As spinal cord compression progresses, subtle long tract signs may appear, initially in the lower limbs and later involving the upper limbs.

Pathogenesis

The pathogenesis of cervical myelopathy can be described in part by the Kirkaldy-Willis model, which outlines degeneration in three stages: dysfunction, instability, and stability. Phase I describes joint dysfunction resulting from macrotrauma or repetitive microtrauma, which alters joint biomechanics and physiological movement, such as annular tearing or zygapophyseal capsulitis. When the nucleus pulposus becomes involved, segmental control is lost, resulting in dysfunction.

With continued stress, the condition progresses to Phase II, the unstable phase, characterized by excessive joint movement and laxity of posterior structures, including the annulus fibrosus. The intervertebral disc undergoes multiple annular tears, internal disruption, and loss of disc height.[10] Loss of disc height reduces the tensile strength of the ligamentum flavum, causing buckling and canal narrowing. Cartilage degeneration and capsular laxity are also present.

In Phase III, the body attempts to regain stability through osteophyte formation at the vertebral end plates. This increases mechanical load on the zygapophyseal joints, leading to further degeneration and bony overgrowth along the facets and end plates. Excessive bone formation narrows the spinal canal and may cause central or lateral spinal cord compression at the neural foramina.[10,11]

Although degenerative changes are common with aging, they do not always cause symptoms.[12] The prevalence of myelopathy increases with age, particularly in individuals older than 70 years. With aging, the level of involvement shifts cranially, becoming more common at C3–C4, whereas younger patients more frequently exhibit pathology at C5–C6.¹² Cervical radiculopathy, often associated with cervical spinal stenosis, affects approximately 85 per 100,000 individuals annually.[13]

Diagnosis

Diagnosing cervical myelopathy can be challenging, as symptoms and signs are variable and may not always present as a complete clinical picture. Therefore, a detailed medical history, thorough physical examination, and appropriate diagnostic testing are essential. Diagnostic evaluation may include plain radiographs of the cervical spine, magnetic resonance imaging (MRI) of the cervical spine, and electromyoneurography (EMNG) of the upper and lower extremities.

Differential Diagnosis

The differential diagnosis includes amyotrophic lateral sclerosis (ALS)/motor neuron disease (MND), syringomyelia, peripheral neuropathy, spinal cord tumors, multiple sclerosis (MS), and cerebrovascular disease. In MND, sensory deficits are absent, and fasciculations may be observed in the tongue or facial muscles.[14] Electromyography is useful in differentiating these conditions.

MRI is the diagnostic modality of choice for noninvasive evaluation, as it can identify spinal cord compression in CSM, demyelinating plaques in MS, tumors, or syrinx formation in syringomyelia.

Case Report

A 63-year-old female patient presented for neurological examination due to progressive weakness of all extremities, predominantly affecting the lower extremities, resulting in walking difficulties. She also reported tingling in the left hand, initially involving the fourth and fifth fingers, which later spread to the entire hand. Symptom onset occurred approximately five months prior to examination.

The patient reported a fall from a bicycle five months earlier, resulting in a left lateral malleolar fracture treated conservatively. Subsequently, progressive lower limb weakness developed, leading to loss

of independent walking. Sensory symptoms in the left hand followed, and approximately one month later, finger deformity and curvature were noted, more pronounced on the left, accompanied by decreased hand strength and frequent object dropping. Occasional muscle twitching in the left thigh was also reported.

Neurological examination revealed bilaterally hyperactive deep tendon reflexes, predominantly in the lower extremities, with symmetrical polykinetic patellar reflexes progressing to clonus, an expanded reflexogenic zone, and bilaterally positive Babinski signs. Hypotrophy of the thenar, hypothenar, and interosseous muscles of the left hand was observed, with flexion deformity of the first, second, and third fingers. Superficial sensation was intact. There were no sphincter disturbances. Walking was not possible without assistance and demonstrated spastic paraparetic characteristics.

Extensive laboratory investigations were unremarkable. Abdominal ultrasound findings were normal. EMNG demonstrated chronic partial radicular lesions involving the proximal lower motor neuron, with predominant involvement of the right C6 and C8 nerve roots of moderate severity.

MRI of the brain revealed no acute ischemic or hemorrhagic lesions, moderate microangiopathic changes, and global cortical atrophy.

MRI of the cervical spine demonstrated multilevel degenerative changes, including marked disc protrusions from C3–C7 with severe spinal canal stenosis, spinal cord compression, and intramedullary signal changes consistent with compressive myelopathy.



Figure 1. MRI of the cervical spine T1 sequence

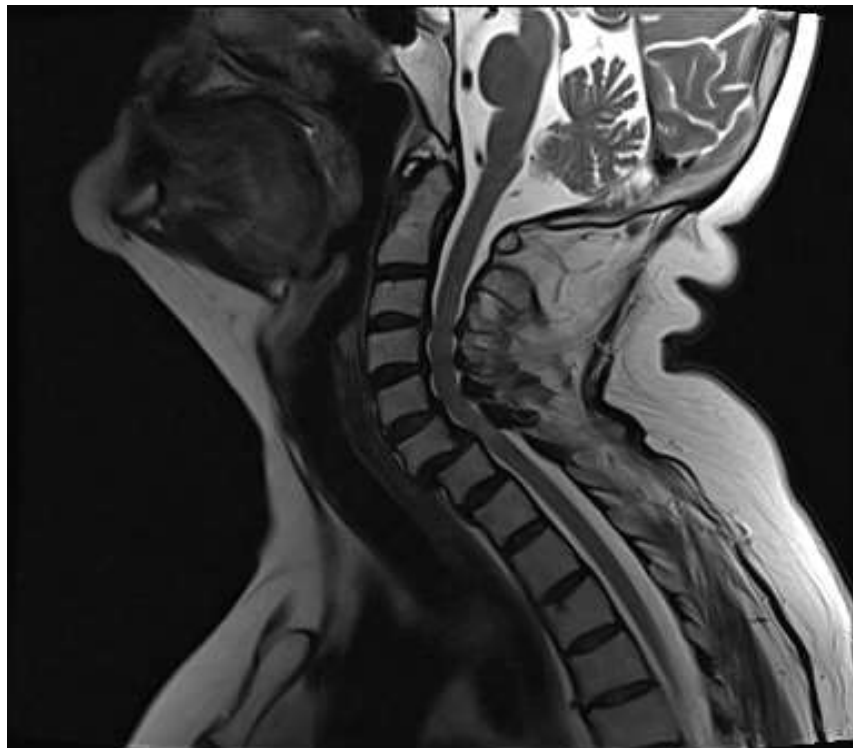


Figure 2. MRI of the cervical spine T2 sequence

The patient declined lumbar puncture and surgical intervention despite neurosurgical recommendation. Conservative management with physical therapy resulted in partial clinical improvement. The patient remains under neurological outpatient follow-up.

Conclusion

This case highlights the critical importance of thorough clinical evaluation in the diagnosis of cervical myelopathy. Early recognition of characteristic neurological signs and timely imaging are essential, as delayed diagnosis or inappropriate management may result in progressive and irreversible neurological deterioration. In severe cases of cervical myelopathy caused by disc herniation, conservative management carries a significant risk of neurological decline and may compromise functional recovery. Prompt differentiation between compressive etiologies requiring surgical decompression and intrinsic spinal cord pathologies amenable to medical treatment is therefore fundamental to optimizing patient outcomes and preventing long-term disability.

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