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Case Report

The "pancake-like" enhancement in cervical spondylotic myelopathy ☆

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ABSTRACT

Cervical spondylotic myelopathy, characterized by chronic spinal cord compression resulting from degenerative spine changes, manifests with a spectrum of neurological and pain symptoms. Despite the complexity of intramedullary spinal cord abnormalities, employing a systematic approach to differential diagnosis, considering factors such as lesion location, cord length, segment involvement, and enhancement pattern, can significantly aid in narrowing down the potential diagnoses, potentially avoiding invasive diagnostic procedures and guiding treatment decisions. This article presents two cases of cervical spondylotic myelopathy characterized by progressive weakness and paraesthesia, exhibiting progressive bilateral upper extremity numbness, tingling, and impaired gait, with cervical myelopathy evident on MRI displaying transverse pancake-like gadolinium enhancement.

Introduction

Cervical spondylotic myelopathy is characterized by the chronic compression of the spinal cord due to degenerative changes in the spine, leading to a diverse range of neurological and pain symptoms. While primarily diagnosed clinically, advanced imaging techniques like magnetic resonance imaging (MRI) enable rapid exploration of potential causes of myelopathies [1]. Intramedullary spinal cord abnormalities are a diagnostic challenge. The radiologist's ability to narrow the differential diagnosis of spinal cord abnormalities has the potential to save patients from invasive diagnostic procedures and guide appropriate management [2].

A rare yet specific MRI finding indicative of spondylotic myelopathy is transverse pancake-like gadolinium enhancement. Identifying this sign expedites treatment initiation and plays a critical role in averting subsequent disability [1].

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Case report

Case 1

A 56-year-old male presented with a three-month history of progressive weakness in his right arm and hand, along with intermittent neck pain. He reported episodes of clumsiness and dropping objects due to decreased grip strength. Clinical examination revealed weakness in the upper extremities with diminished sensation in both hands. MRI of the cervical spine showed evidence of cervical spondylotic changes and degenerative disc disease, with maximum stenosis at the C4-C5 level and intramedullary T2-signal hyperintensity, indicative of spinal cord compression and cervical spondylotic myelopathy (Figs. 1A and B). Post-contrast MRI confirmed the presence of a transverse band of gadolinium enhancement below the site of maximum stenosis, supporting the diagnosis (Figs. 1C and D).

Case 2

A 60-year-old female presented with a 6-month history of progressive numbness and tingling in her lower extremities, along with difficulty walking and frequent falls. She reported no significant neck pain but noted increasing clumsiness in her feet and legs. Clinical examination revealed decreased sensation in the lower extremities, bilateral foot drop, and hyperreflexia in the knees. MRI of the cervical spine showed cervical spondylotic changes and degenerative disc disease, with maximum stenosis at the C6-C7 level and intramedullary T2-signal hyperintensity (Figs. 2A and B). Post-contrast MRI demonstrated a transverse band of gadolinium enhancement

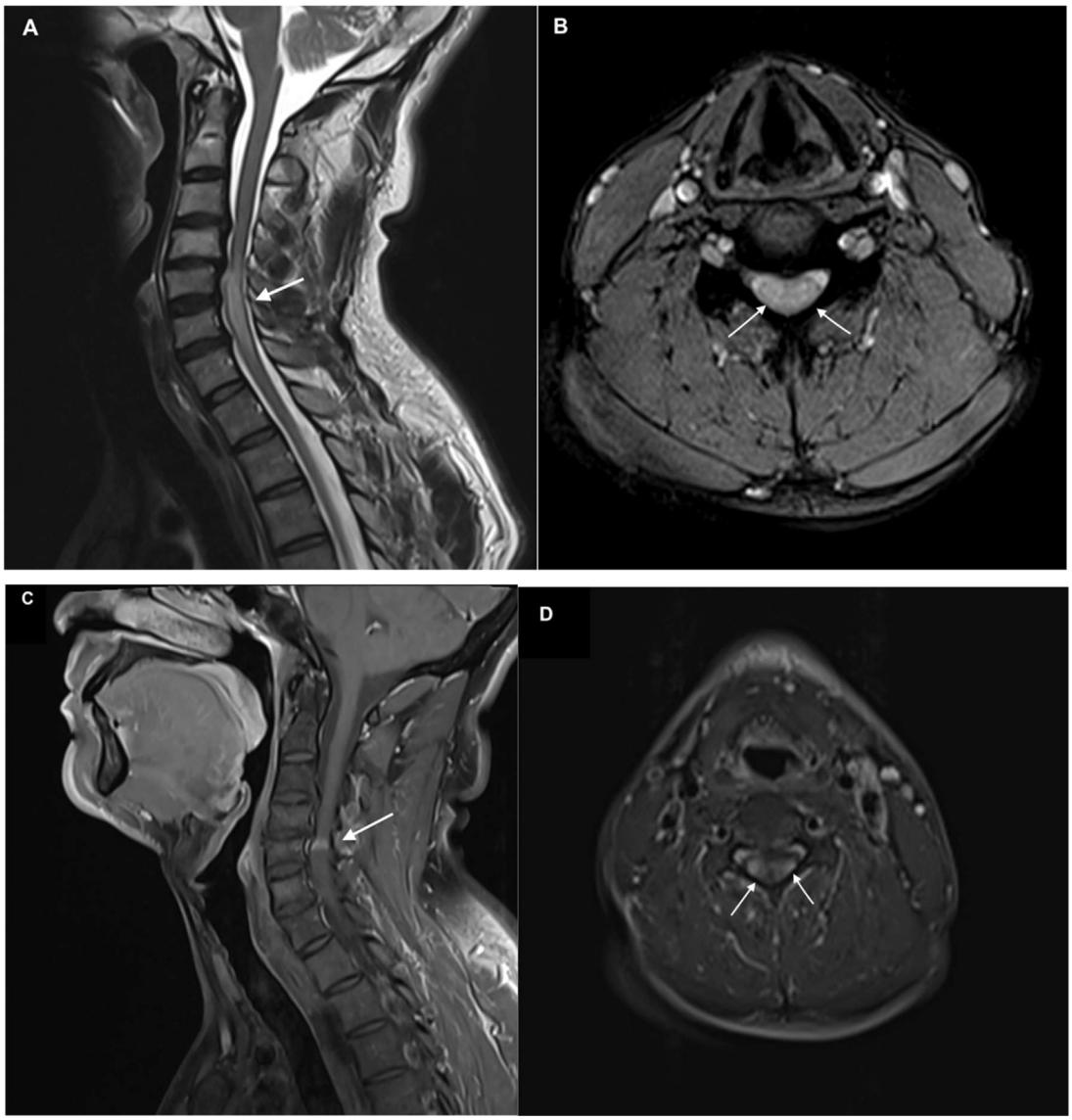

Fig. 1 – Case 1. (A) Sagittal MRI showing cervical spondylosis with spinal cord compression and below the site of maximal stenosis at C4-C5. (B) Axial MERGE images showing circumferential T2 hypersignal at the same level. (C) There is a pancake-like band of enhancement on the post-gadolinium image at at C4-C5. (D) Post contrast T1 FS images demonstrating that the enhancement concerns the lateral aspects of the spine.

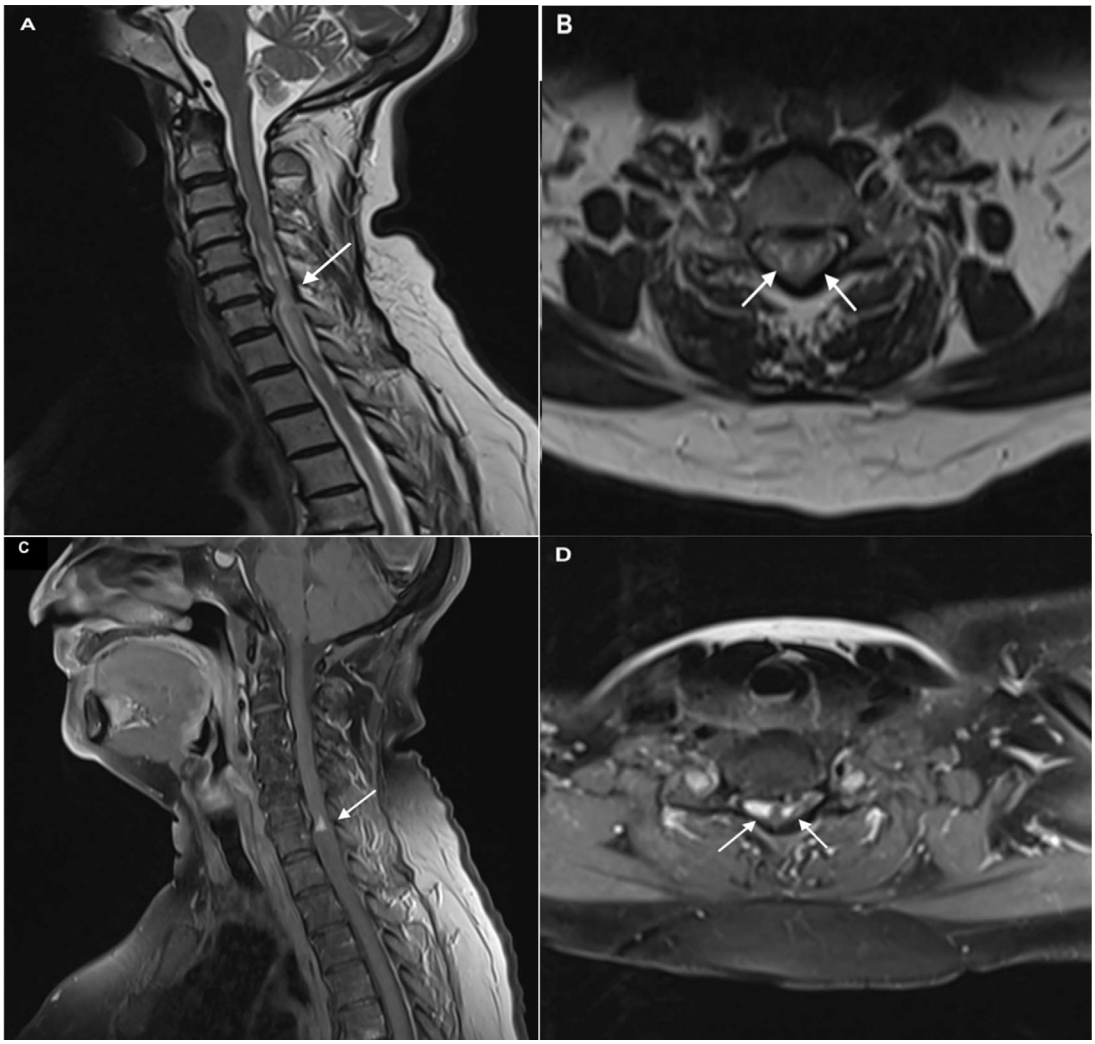

Fig. 2 – Case 2. Sagittal MRI of the cervical spine, showing spondylosis with spinal cord compression and vasogenic edema below the site of maximal stenosis at C6-C7 with intramedullary T2 hyperintensity extending from C5 to D1 (A), predominantly involving the right lateral aspect of the spinal cord as seen on axial images (B). Pancake-like band of enhancement at the same level on the post-gadolinium sagittal T1 image (C). The enhancement on axial slices is located at the bilateral lateral aspect of the spinal cord, but it is predominantly on the right side (D).

below the area of stenosis, consistent with cervical spondylotic myelopathy (Figs. 2C and D).

Discussion

Cervical spondylotic myelopathy (CSM) is a degenerative condition affecting the cervical spine, characterized by the gradual compression of the spinal cord due to age-related changes in the structures surrounding it. Radiographically, CSM manifests as progressive degenerative alterations such as intervertebral disc herniation, osteophyte development, hypertrophy of ligamentum flavum, and narrowing of the spinal canal, collectively contributing to spinal cord compression and resultant neurological impairment. Additionally, the presence of congenital cervical spinal stenosis may heighten the risk of developing cervical spondylotic myelopathy [3].

Patients with cervical spondylotic myelopathy (CSM) may present with neck pain, radiating discomfort to the arms, weakness, numbness, gait disturbances, coordination problems, fine motor skill difficulties, imbalance, and in severe cases, bowel or bladder dysfunction, sensory deficits, and muscle weakness in the lower extremities. Symptoms can worsen over time, underscoring the need for early diagnosis and treatment. The clinical presentation of CSM can be insidious and progressive, varying from person to person, with some individuals undergoing a gradual deterioration, others experiencing periods of stability, and a subset potentially demonstrating signs of improvement as time passes [4,5].

Examinations conducted on cadavers have revealed a distinct progression of lesions in cervical spondylotic myelopathy (CSM), with initial findings of atrophy and neuronal loss

in the anterior horn and intermediate zone, followed by degeneration in the lateral and posterior funiculi. Subsequently, widespread atrophy develops throughout the gray matter, accompanied by significant degeneration in the lateral funiculus. Notably, the presence of thin myelinated fibers in the white matter suggests the occurrence of focal demyelination and remyelination processes in CSM, with the degree of compression correlating with the pathological changes involving both gray and white matter of the spinal cord [5,6].

On MRI scans, intramedullary signal abnormalities in cervical spondylotic myelopathy (CSM) often result from a combination of edema and structural changes, typically appearing as a hyperintense signal on T2-weighted images (T2-WI) in most cases with clinical myelopathy [7]. Less commonly, these abnormalities may manifest as hypointense signals on T1-weighted images (T1-WI). Although post-gadolinium MRI sequences are not standard in the evaluation of cervical degenerative disease, they can provide valuable information regarding the spinal cord parenchyma's integrity, blood-cord barrier, and aid in differential diagnosis of intramedullary lesions. Notably, they may reveal a distinct "pancake-like" enhancement pattern that is characteristic of CSM [8].

The exact mechanism underlying gadolinium enhancement in spondylotic myelopathy (SM) is not fully understood, but it is likely linked to the localized disruption of the blood-spinal cord barrier. This disruption may be triggered by the emergence of new blood vessels in response to injury, potentially leading to a compromised blood-spinal cord barrier. Furthermore, increased vascular permeability resulting from venous hypertension has been proposed as another potential factor contributing to this phenomenon. The presence of scarring in the subarachnoid space could alter the dynamics of cerebrospinal fluid and extracellular fluid flow, potentially leading to the development of edema [8].

Flanagan et al. detailed in their study the "pancake-like" enhancement in cervical spondylotic myelopathy (CSM) and outlined key criteria for clinicians, including a transverse band appearance on sagittal images, localization below the main stenosis point within a spindle-shaped T2 hyperintensity, and circumferential enhancement sparing the gray matter on axial images [8]. Additionally, the enhancement was predominantly noted at the C5 or C6 levels, which are commonly affected areas in spondylotic myelopathy.

It is common to observe persistent enhancement lasting for a year or more after decompressive surgery, with rates as high as 75%. In cases where there is no clinical deterioration, this prolonged enhancement is typically consistent with spondylotic myelopathy (SM). However, any worsening or increase in enhancement seen in the months to years following surgery should prompt consideration of alternative diagnoses or incomplete decompression.

When assessing spinal cord enhancement, it's crucial to explore other potential causes in the differential diagnosis, including demyelinating, metabolic, neoplastic, or vascular etiologies. Spinal cord tumors typically exhibit expansive lesions with heterogeneous signal intensity, sometimes showing enhancement along a long craniocaudal segment. Demyelinating diseases often feature long craniocaudal lesions with diverse enhancement patterns (none, diffuse, patchy, peripheral), occasionally with additional lesions in the spinal cord and brain. Inflammatory or infective conditions typically demonstrate long craniocaudal segments of enhancement. Vascular lesions commonly present with a long craniocaudal extent and varied enhancement patterns (none, mild, patchy, nodular). Acute myelopathies caused by infectious agents should also be considered in the differential diagnosis, although the presence of fever, meningismus, and inflammatory cerebrospinal fluid (CSF) typically prompt investigation into identifying the causative agent.

When differentiating intramedullary enhanced lesions on MRI from pancake-like enhancement in cervical spondylotic myelopathy, various factors such as enhancing patterns, lesion characteristics, distribution, and associated findings need to be considered. The differential diagnosis, including demyelinating, metabolic, neoplastic, or vascular etiologies. For example, multiple sclerosis lesions typically exhibit longitudinally extensive pattern along the spinal cord associated with periventricular and juxtacortical lesions, while neuromyelitis optica lesions often involve longer spinal cord segments and may show central necrosis. Spinal cord tumors like ependymomas present as well-defined lesions with homogeneous enhancement, inflammatory conditions like transverse myelitis may have longitudinally extensive lesions with irregular shapes, and vascular malformations such as AVMs show abnormal vessels and flow voids [2,8].

The treatment can be non-surgical and based on cervical immobilization, steroids, and pain control medications. Potentially beneficial surgical decompression is delayed or deferred. The presence of pancake-like gadolinium enhancement alone should not be the sole basis for recommending surgery. Instead, recommendations for surgical decompression should take into account multiple factors, including the progression of symptoms and signs, the absence of other identifiable causes after thorough evaluation, assessment of the severity of cord compression based on axial T2-weighted images, and a personalized risk-to-benefit assessment for each patient [9].

Conclusion

Identifying a distinct pattern of gadolinium enhancement on sagittal and axial MRI, alongside an intramedullary T2-signal abnormality, in individuals with cervical spondylosis and clinical myelopathy strongly suggests spondylosis as the underlying cause. Recognizing this specific radiological pattern can play a pivotal role in achieving an accurate diagnosis and ruling out other potential causes of myelopathy. Consequently, it may guide treatment strategies, helping to avoid unnecessary interventions.

Patient consent

Written informed consent for the publication of this case report was obtained from both patients.

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