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The Trident Sign on Isolated Sarcoidosis-Associated Myelopathy: A Case Report

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Abstract

This report presents a rare case of a possible isolated sarcoidosis-associated myelopathy (SAM) in a 50-year-old woman who exhibited sensory disturbances and gait instability. Cerebrospinal fluid (CSF) analysis revealed inflammatory characteristics without evidence of infectious disease. Computed tomography (CT) scans of the head, chest, abdomen, and pelvis showed no vascular, neoplastic, or systemic granulomatous abnormalities. Magnetic resonance imaging (MRI) of the spine with gadolinium enhancement demonstrated the characteristic trident sign on axial sequences, strongly suggestive of SAM. The patient underwent pulse therapy with methylprednisolone followed by a tapering course of glucocorticoids, resulting in gradual clinical improvement. Although a definitive diagnosis requires histopathological confirmation, this case underscores the importance of MRI recognition patterns in facilitating prompt diagnosis and guiding effective therapeutic management.

Keywords

Neurosarcoidosis, Spinal Cord Involvement, Trident Sign, Neuroimaging, Granulomatous Diseases

1. Introduction

Sarcoidosis is a multisystemic granulomatous disorder characterized by immune-mediated inflammation of unknown etiology, with its clinical manifestations determined by the organ systems involved [1]. Neurosarcoidosis (NS) occurs when the disease affects any component of the nervous system. Central nervous system (CNS)

involvement may lead to diverse manifestations, including cranial neuropathies, hypopituitarism, seizures, myelopathies, and radiculopathies. Peripheral nervous system involvement can manifest as mono- or polyneuropathies and myopathies.

Given the potential risks associated with nervous tissue biopsy, particularly in cases where the disease appears confined to the nervous system, contrast-enhanced MRI has become indispensable in the diagnostic evaluation of suspected NS [2]. To differentiate NS from other pathologies that mimic similar clinical presentations, increasingly specific imaging markers—such as the trident sign described in spinal cord sarcoidosis—are being investigated. Therefore, case reports that correlate imaging findings with clinical suspicion remain essential to improving diagnostic accuracy and management strategies.

2. Case Description

A 50-year-old woman was admitted for evaluation of acute thoracic myelopathy presenting with sudden-onset pain and paresthesia in the left hallux, which ascended progressively to the T1 sensory level over approximately 24 hours. The patient also developed gait disturbance and bowel habit changes, without infectious prodromes or recent vaccination.

Extensive laboratory testing for autoimmune diseases, including antibodies related to antiphospholipid syndrome, Sjögren's syndrome, and other antinuclear antibodies, yielded negative results. Imaging studies—including CT scans of the skull, chest, abdomen, and pelvis—revealed no evidence of lymphoproliferative or granulomatous disease. The only incidental finding was a uterine lesion, later confirmed by biopsy as a submucosal leiomyoma. Aquaporin-4 antibody testing was negative.

CSF analysis demonstrated lymphocytic pleocytosis with normal protein, glucose, and lactate levels, and no evidence of infection. Serum angiotensin-converting enzyme (ACE) levels were elevated.

Thus, based on the compatible clinical picture associated with extensive laboratory and radiological investigation with coherent findings, and given the impossibility of biopsy for histopathological confirmation, the diagnosis of a possible isolated neurosarcoidosis was considered. The neuraxial section of the MRI study in the T1-weighted sequence did not show any spinal cord injury as we can see in **Figure 1(A)**, however, the post-contrast sequence revealed a posterior spinal cord lesion at the C6-C7 intervertebral level (**Figure 1(B)**), hyperintense in the T2-weighted images (**Figure 1(C)**) and hypointense in the T1-weighted sequences, with gadolinium enhancement showing the characteristic trident sign (**Figure 2(A)** and **Figure 2(B)**).

The patient received pulse therapy with methylprednisolone for five days during hospitalization, followed by a gradual reduction in the dose of oral corticosteroids, which led to significant clinical improvement, presenting only bilateral hypoesthesia in the lower limbs. In addition, a reduction in serum ACE levels was observed. In outpatient follow-up, the patient continued the use of azathioprine, initiated during hospitalization, in combination with oral prednisone, presenting

recurrence and worsening of neurological symptoms when prednisone was discontinued.

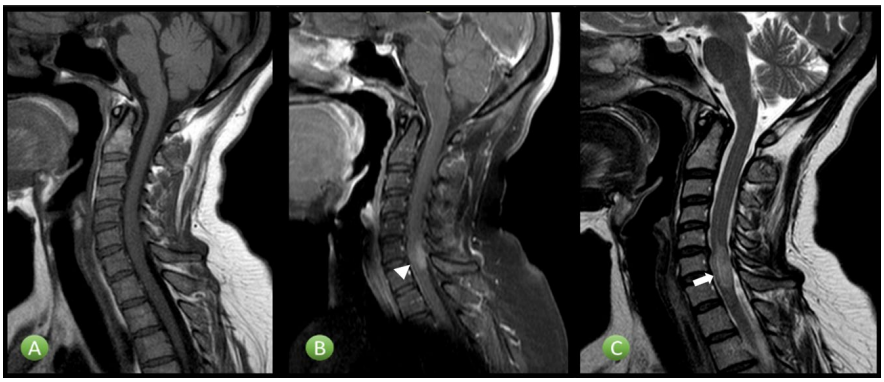

Figure 1. Magnetic resonance image, sagittal section of the cervical spine. (A) T1 weighted without significant changes. (B) Post-contrast magnetic resonance imaging. An enhancing lesion (arrow head) is observed in the posterior aspect of the spinal cord at the C6-C7 level. (C) T2-weighted imaging. Note signal alteration(arrow) in the posterior aspect of the spinal cord, at the level of C6-C7, with a slight expansile effect.

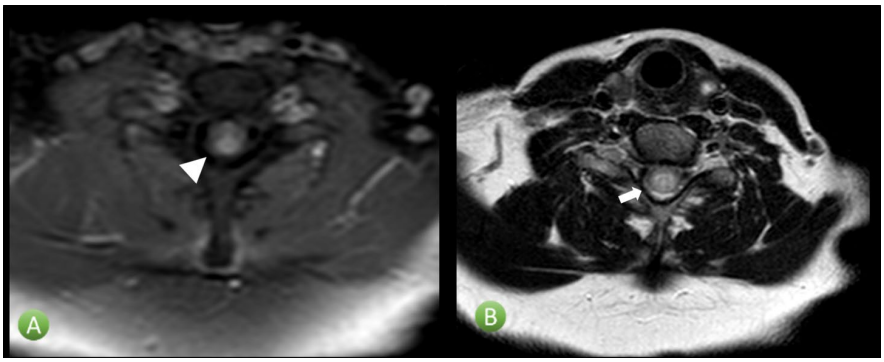

Figure 2. Axial section of the cervical spine. (A) T1-weighted post-contrast magnetic resonance imaging exam, demonstrating the "trident sign" (arrow head). (B) T2-weighted magnetic resonance imaging exam, demonstrating the "trident sign" (arrow).

3. Discussion

This case describes a 50-year-old female patient with acute spinal cord syndrome whose diagnostic workup excluded infectious (viral, fungal, mycobacterial), neoplastic, and environmental causes, including recent vaccination. The presence of the trident sign on axial post-contrast MRI, a hallmark imaging feature in SAM [2] [3], supported the diagnosis.

The clinical presentation aligns with previous literature indicating that neurosarcoidosis is more prevalent in women and that neurological symptoms represent the initial manifestation in up to 52% of cases [4]. Myelopathy occurs in approximately 30% of patients [5], with sensory deficits as the most common initial symptom.

The trident sign, characterized by central canal enhancement communicating with dorsal subpial enhancement, is best visualized on contrast-enhanced MRI and

can help differentiate SAM from idiopathic transverse myelitis [6]-[8]. It forms part of the imaging spectrum of longitudinally extensive transverse myelitis (LETM), typically involving three or more contiguous vertebral segments and most commonly affecting the cervical cord.

The main differential diagnoses for longitudinally extensive transverse myelitis include multiple sclerosis, which presents as partial myelitis usually associated with focal neurological symptoms, as well as typical lesions on MRI; infectious myelitis; idiopathic myelitis; acute disseminated encephalomyelitis (ADEM), a condition more commonly seen in children; anti-MOG antibody-associated disease (MOGAD), which has an acute presentation and positive serology; and neuromyelitis optica spectrum disorder (NMOSD), a longitudinally extensive myelitis whose radiological finding differs from neurosarcoidosis by presenting with a typical ring-shaped, irregular, and peripheral pattern, also showing bright mottled lesions as a highly specific finding. Furthermore, linear dorsal subpial enhancement is rarely seen in NMOSD, but is frequent and persistent in neurosarcoidosis. There are also several other etiologies for longitudinally extensive myelitis, including metabolic, vascular, traumatic, and idiopathic causes [9].

Other imaging patterns described in sarcoid myelitis include short tumefactive myelitis, meningitis/meningoradiculitis, and anterior myelitis adjacent to disc degeneration [5] [10]. Additionally, Boban and Thurnher [11] described a braid-like sign, corresponding to ventral subpial enhancement on sagittal post-contrast T1-weighted images in a case of probable neurosarcoidosis.

CSF findings are generally consistent with inflammatory activity, typically showing mild to moderate lymphocytic pleocytosis. Although protein levels and IgG index may be elevated, these were normal in the present case. No specific CSF biomarker for NS exists; however, hypoglycorrhachia associated with elevated ACE levels has been identified as a distinguishing feature of NS compared to NMOSD and multiple sclerosis [7].

However, it is important to emphasize that despite the use of ACE levels for suspected diagnosis of neurosarcoidosis, this finding has no proven diagnostic or management value, as it may be elevated in other granulomatous, lymphoproliferative, or inflammatory diseases. Thus, even though it helped as a guide during the etiological investigation in this case, its specificity and sensitivity are low [12] [13].

This case has limitations, as a definitive diagnosis requires histopathological confirmation of sarcoid granulomas in neural or extraneural tissue, according to Zajicek's criteria. Nevertheless, spinal cord biopsy carries a significant risk, making SAM a challenging diagnosis that relies heavily on MRI findings, particularly when no systemic disease is available for biopsy.

4. Conclusion

Despite the absence of histopathological confirmation, this case highlights the diagnostic value of combining clinical, laboratory, and imaging data—especially when specific MRI features, such as the trident sign, are present. In scenarios where biopsy is contraindicated or unfeasible, radiologists and clinicians must recognize

these imaging patterns to facilitate timely diagnosis and initiation of appropriate therapy.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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