A rare spinal cord infarction caused by aortic mural thrombosis in a patient with cancer and the associated diagnostic challenges due to it mimicking spinal cord compression: A case report

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Abstract. Patients with malignancy are at an increased risk of arterial thrombosis; however, spinal cord infarction (SCI) secondary to aortic mural thrombosis is rare. The present report describes a case of SCI in a 75-year-old man with cancer of unknown primary undergoing chemotherapy and immune checkpoint inhibitor (ICI) therapy. The patient presented with acute bilateral lower limb weakness and bladder-rectal dysfunction. Although spinal cord compression was initially suspected due to multiple vertebral metastases, contrast-enhanced magnetic resonance imaging revealed no evidence of compression or leptomeningeal dissemination. Diffusion-weighted imaging demonstrated hyperintensity with reduced apparent diffusion coefficient values in the spinal cord, consistent with SCI. Immune-related adverse event-associated myelitis was excluded based on cerebrospinal fluid analysis. Contrast-enhanced computed tomography revealed a large aortic mural thrombus, which was considered the most likely etiology of SCI after exclusion of alternative causes. Anticoagulation therapy combined with early rehabilitation led to partial neurological recovery. This case underscores the importance of considering SCI in the differential diagnosis of acute neurological deficits in patients with malignancy receiving ICI therapy.

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Abbreviations: CT, computed tomography; CUP, cancer of unknown primary; ICI, immune checkpoint inhibitor; MRI, magnetic resonance imaging; SCI, spinal cord infarction

Key words: SCI, aortic mural thrombosis, malignancy

Introduction

Patients with malignancy have an increased risk of both venous and arterial thrombosis. The risk of venous thromboembolism in cancer patients has been reported to be approximately nine-fold higher than that in the general population (1). Similarly, patients with cancer are at an elevated risk of arterial thromboembolism (2). Retrospective studies have shown that aortic thrombosis is associated with malignancy in approximately 20% of cases (3).

Aortic thrombosis can lead to a wide range of embolic events, among which spinal cord infarction (SCI) is rare but potentially devastating. SCI often results in irreversible neurological deficits, making prompt recognition and management crucial. Clinically, presentations such as bilateral lower limb weakness and bladder-rectal dysfunction can mimic spinal cord compression-a recognized oncologic emergency, making differential diagnosis particularly challenging.

To our knowledge, SCI caused by aortic mural thrombosis in patients with malignancy is exceedingly rare. Herein, we report a case of SCI secondary to aortic mural thrombosis in a patient receiving chemotherapy for cancer of unknown primary (CUP). This case highlights the diverse clinical manifestations of arterial thrombosis in cancer patients and underscores the importance of including SCI in the differential diagnosis of acute neurological symptoms resembling spinal cord compression.

Case report

A 75-year-old man was diagnosed with CUP with multiple bone metastases involving the scapula, clavicle, sternum, ribs, ilium, ischium, and lumbar vertebrae in June 2024. He was diagnosed and treated at the Department of Medical Oncology, Nara Medical University Hospital (Kashihara, Japan). The Department of Medical Oncology is clinically and organizationally integrated with the Department of Cancer

Genomics and Medical Oncology, Nara Medical University, where the treating physicians are primarily affiliated. Bone biopsy revealed a malignant lesion composed in part of loosely arranged bundles of spindle-shaped cells. Although consistent with malignancy, no definite line of differentiation was identified. Sarcomatoid carcinoma and soft tissue sarcoma were considered in the differential diagnosis; however, a definitive diagnosis could not be established based on histology alone. Given the possibility that the tumor represented either a sarcoma or CUP, treatment with doxorubicin was initiated, and the patient received six courses beginning in June 2024. As the disease progressed, nivolumab therapy, approved in Japan for CUP, was initiated in December 2024.

In August 2025, the patient presented urgently with acute bilateral lower limb weakness and inability to walk. He was admitted to the same institution. On admission, manual muscle testing showed 3/5 strength in the iliopsoas, hamstring, and quadriceps muscles bilaterally, while the tibialis anterior and gastrocnemius muscles were preserved at 5/5. Deep tendon reflexes of the lower limbs were decreased. He was unable to urinate or defecate independently, indicating bladder and rectal dysfunction.

Given his known bone metastases, spinal cord compression was initially suspected. However, whole-spine contrast-enhanced magnetic resonance imaging (MRI) revealed multiple vertebral metastases but no evidence of cord compression or leptomeningeal dissemination (Fig. 1). Diffusion-weighted imaging showed high signal intensity with reduced apparent diffusion coefficient values in the spinal cord from thoracic vertebrae 11 to 12 (Th11-Th12), raising suspicion of SCI (Fig. 2). Because he was receiving nivolumab, immune-related adverse event (irAE) myelitis was also considered. Cerebrospinal fluid analysis, however, showed no pleocytosis, no protein elevation, normal glucose, and normal opening pressure, making irAE myelitis unlikely. Based on these findings, SCI was diagnosed.

Subsequent contrast-enhanced computed tomography (CT) revealed a large aortic mural thrombus (Fig. 3), which was considered the most likely etiology of SCI after exclusion of alternative causes. The D-dimer level at presentation was 1.6 μg/ml (reference range, 0-1.0 μg/ml); previous measurements ranged from 0.8 to 1.8 μg/ml, showing no significant elevation from baseline. Routine coagulation studies showed a prothrombin time-international normalized ratio (PT-INR) of 1.10 (reference range, 0.8-1.15) and an activated partial thromboplastin time (APTT) of 40 sec (reference range, 24-39 sec), without clinically significant abnormalities. Fibrinogen was mildly elevated at 473 mg/dl (reference range, 200-400 mg/dl). Heparin anticoagulation therapy was initiated, and early rehabilitation began during hospitalization. By hospital Day 3, lower limb motor function had improved sufficiently to allow ambulation with a walker. However, by hospital Day 9, he developed difficulty maintaining a kneeling posture, and no further neurological improvement was observed. A repeat spinal MRI performed following this deterioration demonstrated no apparent extension of the infarcted area or new findings suggestive of progressive spinal cord injury. Although infarct progression was considered unlikely, the patient's overall condition deteriorated because of advanced malignancy and declining performance status. Therefore, the decision to transition to best supportive care was based on his overall clinical condition rather than neurological status alone. He was discharged home on hospital Day 19 under best supportive care. The latest follow-up was performed in September 2025 at the time of discharge, when persistent lower extremity neurological deficits remained without further improvement.

Discussion

The most frequent causes of SCI are idiopathic or related to aortic surgery. Other reported etiologies include aortic dissection, fibrocartilaginous embolism, systemic atherosclerosis, systemic hypotension, hypercoagulable states, cardioembolism, and vasculitis (4-6).

In the present case, imaging suggested that the SCI was caused by an aortic mural thrombus. However, transthoracic and transesophageal echocardiography were not performed; therefore, occult cardioembolic sources such as nonbacterial thrombotic endocarditis, intracardiac thrombus, or patent foramen ovale could not be completely excluded. Consequently, the causal relationship between the aortic mural thrombus and SCI cannot be definitively established and should be regarded as a clinical inference based on imaging findings and exclusion of alternative etiologies. Alternative etiologies were also considered. The patient had no documented episodes of systemic hypotension or hemodynamic instability preceding symptom onset, making spinal cord ischemia due to hypoperfusion unlikely. In addition, routine coagulation studies did not support disseminated intravascular coagulation (DIC), with no clinically significant abnormalities in PT-INR or APTT and only mild elevation of fibrinogen, which was considered attributable to the underlying malignancy and inflammatory status. Although a cancer-associated hypercoagulable state may have contributed to thrombus formation, there was no clinical or radiological evidence specifically suggesting overt DIC. Furthermore, tumor embolism was considered unlikely because contrast-enhanced CT demonstrated a large aortic mural thrombus that provided a plausible embolic source, and no pathological or imaging findings suggested tumor embolization. Although small-vessel disease and other embolic sources cannot be completely excluded, the imaging findings and the presence of a large aortic mural thrombus were considered more consistent with an embolic mechanism originating from the aorta. Nevertheless, contrast-enhanced CT demonstrated a large mural thrombus extending from the aortic arch to the descending thoracic aorta. Furthermore, the anatomical distribution of the thrombus was considered compatible with a potential source of embolization to the spinal cord circulation, supporting aortic mural thrombosis as the most likely etiology in this case. Patients with malignancy are known to have an increased risk of arterial thrombosis compared with the general population (2). Although additional testing for lupus anticoagulant and anticardiolipin antibodies was not performed, routine coagulation studies showed no clinically significant abnormalities in PT-INR or APTT. Fibrinogen was mildly elevated (473 mg/dl), which was considered attributable to the underlying malignancy and inflammatory status rather than a specific hypercoagulable disorder. Notably, the D-dimer level at SCI onset was not substantially elevated compared with previous measurements. This may reflect the

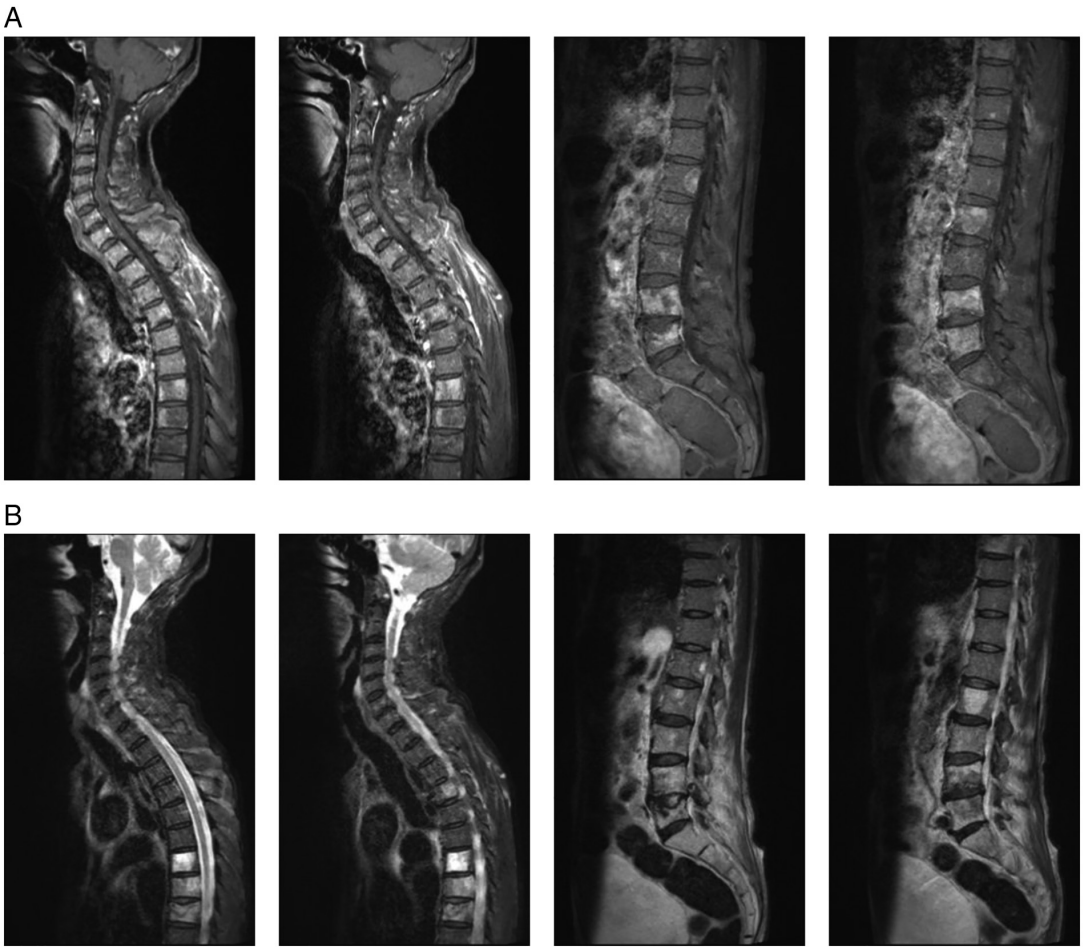

Figure 1. Whole-spine magnetic resonance imaging demonstrating multiple vertebral metastases. (A) Contrast-enhanced T1-weighted image with fat suppression. (B) Fat-suppressed T2-weighted image. Multiple hyperintense bone lesions are visible on fat-suppressed T2-weighted images, which demonstrate contrast enhancement on T1-weighted images, consistent with bone metastases.

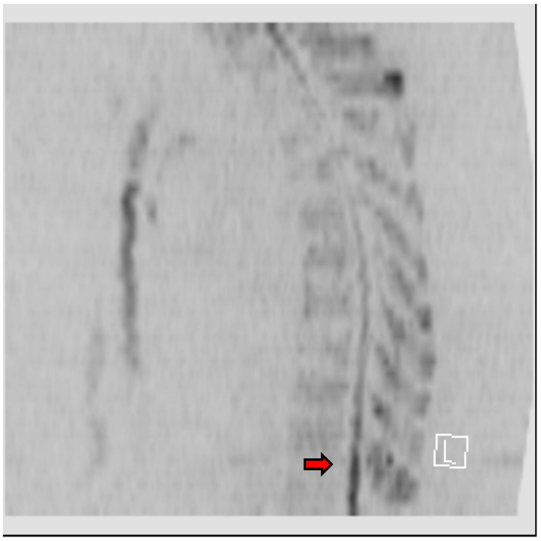

Figure 2. Inverted grayscale display of a diffusion-weighted image showing hyperintensity in the spinal cord from thoracic vertebra 11 to the inferior margin of the field of view at thoracic vertebra 12 level (red arrow), consistent with spinal cord infarction.

patient's pre-existing cancer-associated hypercoagulable state, as D-dimer levels had remained persistently elevated before SCI onset. Consequently, any additional increase associated with SCI may have been relatively small and difficult to distinguish from baseline variation. Furthermore, D-dimer levels do not necessarily correlate with the extent or clinical severity of arterial thrombosis. Furthermore, emerging evidence indicates that patients receiving immune checkpoint inhibitors (ICIs) may have a higher risk of arterial thrombosis than those not treated with ICIs (7). The exact mechanisms underlying ICI-associated thrombosis remain incompletely understood. However, several potential mechanisms have been proposed, including enhanced T-cell activation, endothelial inflammation, accelerated atherosclerosis, and neutrophil extracellular trap (NET)-mediated thrombogenesis (8). In particular, PD-1 plays an important role in suppressing pro-atherogenic T-cell responses, and PD-1 blockade may exacerbate vascular inflammation by releasing these inhibitory signals, thereby promoting thrombus formation (8). Although causality cannot be established in the present case, these mechanisms may have contributed to the development and progressive enlargement of the aortic mural thrombus observed after nivolumab initiation. Although a definitive causal relationship cannot be established in this case, follow-up contrast-enhanced CT after initiation of nivolumab demonstrated the emergence and progressive enlargement of an aortic mural thrombus over time (Fig. 4). Evidence regarding aortic thrombosis in cancer patients remains limited; a small retrospective

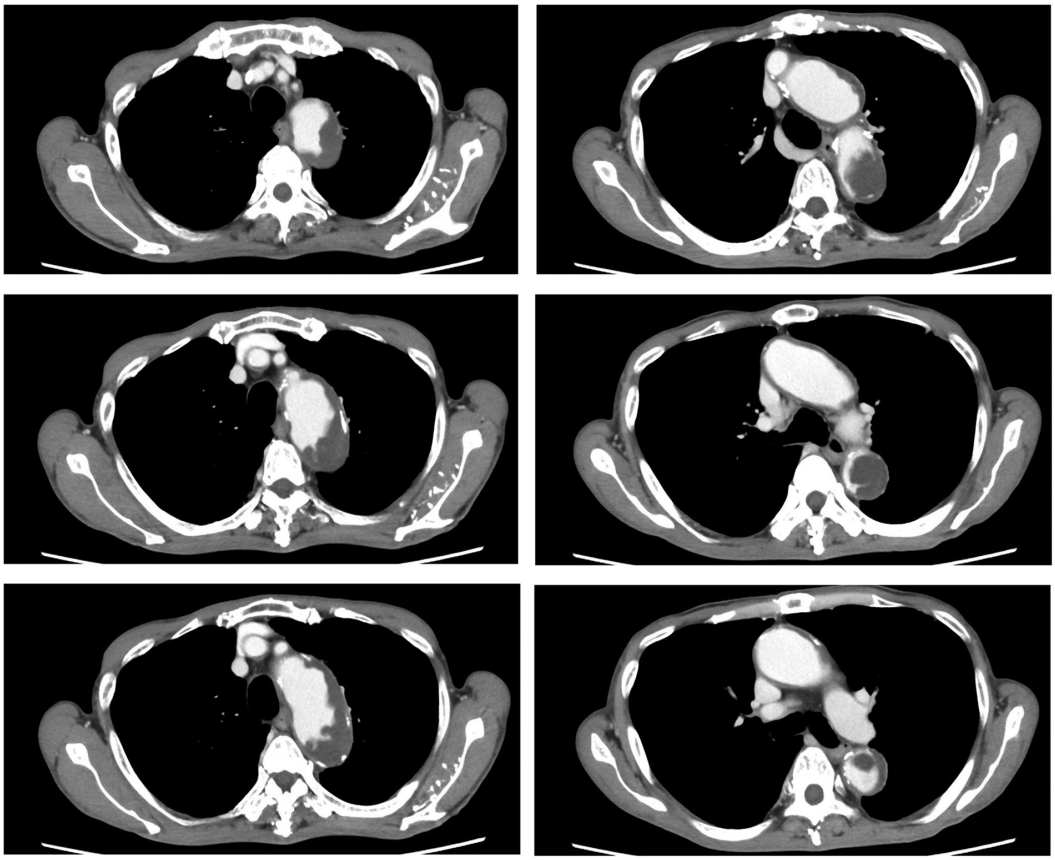

Figure 3. Contrast-enhanced computed tomography demonstrating an aortic mural thrombus extending from the aortic arch to the descending aorta.

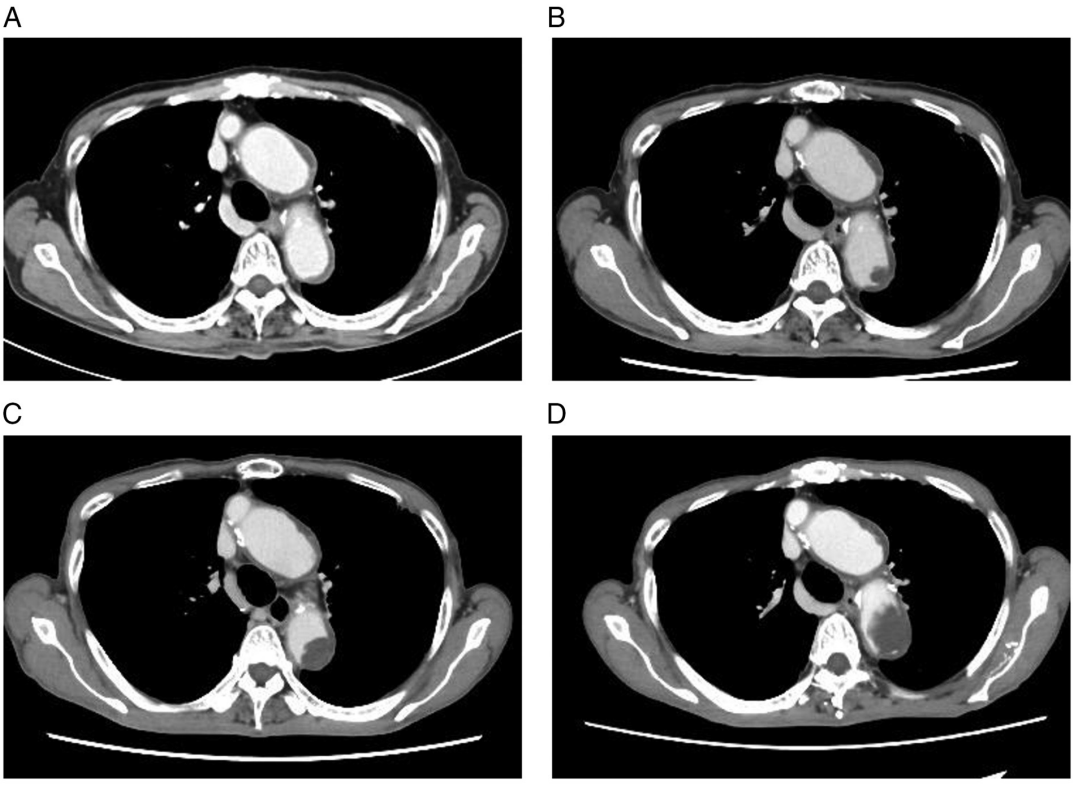

Figure 4. Serial contrast-enhanced CT images showing the development and progression of an aortic mural thrombus. (A) A CT obtained in November 2024, prior to nivolumab initiation, showing no evidence of aortic mural thrombus. (B) A CT obtained in February 2025, at the first evaluation after nivolumab initiation, demonstrating a newly appeared aortic mural thrombus. (C) A CT obtained in May 2025, the second follow-up after nivolumab initiation, showing interval enlargement of the thrombus. (D) A CT obtained in August 2025, after the onset of spinal cord infarction, demonstrating further progression of the thrombus. CT, computed tomography.

study reported that 3 of 13 patients with primary aortic mural thrombus had concomitant malignancy (3). While several cases of SCI due to aortic thrombosis have been reported (9,10), SCI caused by aortic mural thrombosis in patients with malignancy is exceedingly rare. Reports on cancer-associated SCI are limited, but a recent systematic review indicated a poor prognosis, with a median survival of approximately 4 months (11). In our patient, early initiation of physical therapy led to rapid neurological recovery, allowing ambulation with a walker within several days. However, the patient subsequently developed recurrent generalized weakness and a decline in performance status, ultimately necessitating a transition to best supportive care. Repeat spinal MRI was performed after neurological deterioration following the initial recovery and demonstrated no apparent extension of the infarcted area or other radiological findings suggestive of progressive spinal cord injury. Therefore, infarct progression was considered unlikely to explain the subsequent neurological deterioration. Nevertheless, secondary neuronal injury or other factors contributing to the recurrent neurological deficits cannot be completely excluded. Another limitation of the present study is the incomplete imaging characterization of the aortic mural thrombus. Although contrast-enhanced CT demonstrated a large mural thrombus extending from the aortic arch to the descending thoracic aorta, precise measurement of its maximum diameter and length was difficult because the lesion exhibited an elongated mural configuration with irregular and continuous extension along the aortic wall rather than a discrete mass. In addition, thrombus mobility and classification as pedunculated or sessile could not be reliably assessed using static contrast-enhanced CT alone, and dynamic imaging modalities such as transesophageal echocardiography or cine imaging were not performed. Furthermore, the relationship between the thrombus and the ostia of spinal feeding arteries, including the artery of Adamkiewicz, could not be evaluated because these vessels were not clearly visualized on routine contrast-enhanced CT. Therefore, interpretation of the embolic potential and anatomical relationship of the thrombus is limited by the available imaging data. Importantly, the decision to transition to best supportive care was based not only on neurological status but also on the patient's overall clinical deterioration and progressive decline in performance status associated with advanced malignancy. Treatment options for aortic mural thrombus include anticoagulation therapy, stent graft placement, and surgical intervention (3). In this case, stent graft insertion or surgery was deemed high risk due to the potential for further embolic events, and conservative management with heparin alone was chosen. Given the rarity of asymptomatic aortic mural thrombosis in patients with malignancy receiving ICIs and the limited available evidence, no established guidelines currently exist regarding screening or prophylactic treatment in this setting. Therefore, routine prophylactic anticoagulation solely on the basis of ICI therapy cannot be recommended. If an asymptomatic aortic mural thrombus is incidentally identified, management should be individualized based on thromboembolic risk, bleeding risk, thrombus characteristics, cancer status, and overall prognosis. Although anticoagulation therapy may be considered in selected high-risk patients, the optimal agent and treatment duration remain uncertain and warrant further investigation. SCI in cancer patients may therefore severely limit further oncologic treatment and negatively impact overall prognosis.

In this case, SCI occurred during nivolumab therapy for CUP. Given the presence of vertebral metastases, spinal cord compression was initially suspected. After imaging excluded compression and leptomeningeal dissemination, irAE myelitis was also considered. Cerebrospinal fluid analysis, however, revealed normal cell counts, protein, and glucose, effectively ruling out irAE myelitis. The final diagnosis of SCI was established based on diffusion-weighted imaging findings on MRI, neurological examination, and identification of an aortic mural thrombus on contrast-enhanced CT.

Although SCI is rare, it can mimic other urgent neurological conditions. Clinicians evaluating acute neurological deficits in patients receiving ICIs should consider not only spinal cord compression and irAE-related myelitis but also SCI in the differential diagnosis to ensure timely recognition and management.

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Availability of data and materials

The data generated in the present study may be requested from the corresponding author.

Authors' contributions

NH conceptualized the study. NH, SO, RS, YK, YY, MO, MK, HN, MI, TM, YN, MaiT and MasT participated in the diagnosis and clinical management of the patient. Acquisition and interpretation of data were performed by NH, SO, RS, YK, YY, MO, MK, HN, MI, TM, YN, MaiT and MasT. Writing the original draft was undertaken by NH, and writing, reviewing and editing of the manuscript were carried out by NH and MasT. NH and MasT confirm the authenticity of all the raw data. All authors read and approved the final manuscript, and agreed to be accountable for all aspects of the research in ensuring that the accuracy or integrity of any part of the work is appropriately investigated and resolved.

Ethics approval and consent to participate

Not applicable.

Patient consent for publication

Written informed consent was obtained from the patient for the publication of this case report and the accompanying images. The patient was informed that all identifying information would be removed to ensure anonymity.

Competing interests

The authors declare that they have no competing interests.

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