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Early recognition and coordinated care in spinal cord infarction recovery

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Abstract

Spinal Cord Infarction (SCI) is a rare condition lacking standardized acute treatment guidelines. We report the case of a 23-year-old woman on estrogen-progestin therapy who presented with sudden-onset paraplegia and sensory loss below L4. Despite a normal initial spinal MRI, clinical suspicion of spinal cord ischemia led to intravenous thrombolysis within 4 hours. Aortic CT angiography excluded dissection. The patient achieved complete neurological recovery. Multidisciplinary emergency collaboration—neurology, emergency medicine, radiology, nursing, and technicians—was essential to the outcome. Follow-up MRI confirmed anterior cord lesions at D11–D12. This case underscores the value of clinical judgment, rapid team-based management, and suggests that IV thrombolysis may be a reasonable option in selected SCI cases, pending further evidence.

Introduction

Spinal Cord Infarction -SCI-(or ischemia/myelopathy) is rare (often < 1 % of all stroke syndromes). Because of the rarity, there are no established guidelines for acute treatment (unlike cerebral ischaemic stroke).[1] Some anecdotal reports suggest that Intravenous Thrombolysis (IVT) may be

feasible in selected cases of acute SCI — albeit off-label and with limited data: a recent review found ~19 cases of SCI treated with IVT.[2] While outcomes are variable, the reported cases had a surprisingly high rate of favourable short-term recovery, and no major hemorrhagic complications reported in that small dataset. Nonetheless, risk/benefit remains unknown: careful patient selection, rapid diagnosis, imaging to exclude bleed/dissection, and multidisciplinary discussion are essential.

Case Report

A 23-year-old previously healthy woman, with no history of smoking, drug abuse, or sickle cell disease, and on combined estrogen-progestin therapy for contraception presented with sudden-onset, painless paraplegia and sensory loss below the L4 dermatome, approximately one hour prior to admission Neurological examination revealed acute paraplegia with a sensory level at L1-L2. Deep tendon reflexes were reduced in the lower limbs, without pathological reflexes at presentation. Pain and temperature sensation were markedly impaired below the T12 level, while vibration and proprioception were relatively preserved. The patient developed acute urinary retention; bladder catheterization drained approximately 1,000 mL of urine. There were no signs of cranial nerve involvement or upper motor neuron signs. An emergent MRI of the entire spine was performed and showed no pathological findings at the time of imaging (Figure 1). A diagnosis of acute ischemic myelopathy (partial ischemia in the T12 distribution of the anterior spinal artery) was suspected. Given the suspicion of spinal cord ischemia, the patient underwent an aortic CT angiography, which was unremarkable and excluded aortic dissection or other vascular pathology. Given the clinical presentation and within a 4-hour window from symptom onset, intravenous thrombolysis with alteplase was administered (thrombolytic dose r-TP of 0,9mg/Kg with door to needle time of 3 hours). The patient experienced complete neurological recovery following thrombolytic therapy. The day after thrombolytic treatment, the urinary catheter was removed, and the patient voided spontaneously on the same day. Moreover, the patient reported only a subjective sensation of heaviness in the right lower limb; no neurological deficits were objectively identified, and gait and activities of daily living were unaffected.

Subsequently, during the hospitalization, tests were carried out to investigate the cause of the spinal cord ischemia. A comprehensive diagnostic work-up was performed during hospitalization to identify the underlying cause of the spinal cord ischemia. This included a complete cardiological assessment (echocardiography, ECG Holter monitoring and transcranial Doppler ultrasound to screen for a patent foramen ovale). A blood sample was obtained for a comprehensive thrombophilia workup: specifically, testing included antiphospholipid antibodies, lupus

anticoagulant, factor V Leiden, prothrombin gene mutation, protein C and protein S, antithrombin III, homocysteine, fibrinogen, coagulation profile. An autoimmune panel (ANA-ENA screening) and a panel for inflammatory myelitis were also performed (including testing for anti-aquaporin-4 and anti-MOG antibodies, as well as investigations of infectious markers (C-reactive protein and procalcitonin). Completion imaging was also performed in the Interventional Neuroradiology unit with Digital Subtraction Angiography (DSA). All investigations yielded normal results.

Follow-up 1,5 T MRI at ten days and three months plus ten days (99 days) after the first follow up of the thoracic/lumbar spinal cord revealed subtle T2/STIR hyperintensities that correspond to smooth diffusion restriction lesions at the D12-L1/ D11–D12 level, with no additional pathological findings (Figure 2). The patient was discharged with a normal neurological examination (Rankin scale score zero). Acetylsalicylic acid therapy was initiated, and estrogen-progestin therapy was discontinued. At both 3- and 6-month follow-up visits, the patient remained asymptomatic, with no recurrence of neurological events.

Discussion

Although Intravenous Thrombolysis (IVT) is an established treatment for acute ischemic stroke of cerebral origin, its application in cases of Spinal Cord Infarction (SCI) remains extremely limited, with only a few reports documented in the literature. This case is noteworthy for the complete neurological recovery observed following IVT in a patient with acute SCI—an outcome that is rare, given the typically poor prognosis associated with spinal cord ischemia.[3] In partial ischemia of the anterior spinal artery territory, especially when the insult is incomplete and/or transient, the presence of reflex cutaneous responses (flexion and rotational RCP) is possible. This is because the segmental spinal reflex arcs may remain intact. Early, even spontaneous, reperfusion can limit neuronal injury and preserve anterior horn cells and interneuronal circuits, allowing reflex activity to be maintained despite involvement of long tracts supplied by the anterior spinal artery.

We emphasize that rapid clinical recognition and immediate multidisciplinary collaboration among the emergency care team are essential. Moreover, treatment administered within a very short therapeutic window may have been critical to achieving the favorable outcome.[4,5]

The diagnosis was primarily clinical, as early MRI did not show pathological findings, underscoring the importance of maintaining a high index of suspicion in similar presentations. Indeed, an early negative MRI does not exclude spinal cord ischemia, as diffusion-weighted imaging may be initially normal in the hyperacute phase,[6,7] with abnormalities becoming evident only on follow-up

imaging; diagnostic confirmation therefore relies on the subsequent demonstration of a lesion consistent with the clinical vascular territory, as occurred in our case with the later identification of a spinal cord lesion at the D11–D12 level. The apparent discrepancy between the clinical sensory levels and the lesion at D11–D12 can be reconciled by the known variability in spinal cord segmental anatomy, where dermatomal sensory manifestations do not always exactly correspond to the vertebral level of the lesion. Sensory fibers may ascend or descend one or two segments within the cord before synapsing, and the spinal cord terminates above the level of the vertebral column, contributing to such mismatches between clinical findings and imaging. Recognition of this anatomical variability is essential to ensure accurate clinical-radiological correlation.[8,9]

This case suggests that spinal cord infarction may be considered analogous to an ESUS (embolic stroke of undetermined source), a concept from cerebral stroke where, after exclusion of large-artery atherosclerosis, cardioembolism, and other known causes, the event is presumed embolic in origin but without an identifiable source. As in our patient, this framework may guide both diagnostic evaluation and secondary prevention strategies, although its direct applicability to spinal cord ischemia remains to be fully established.

Given the initially normal MRI, a structured differential diagnosis was essential. Acute inflammatory myelitis was considered but, as in our case, was less compatible with the acute onset and vascular distribution on follow-up imaging. Demyelinating disorders such as NMOSD- or MOG-associated myelitis can mimic ischemia but usually present with distinct MRI features and clinical progression, requiring targeted serology.[10] Fibrocartilaginous embolism is rare but can present similarly and was considered in our patient based on clinical context.[11] Parainfectious myelitis was considered less likely given the absence of systemic infectious signs, and CSF was not obtained.[12] Functional neurological disorders were also considered when organic causes could not fully explain the presentation. Overall, as demonstrated in our case, careful clinical evaluation and follow-up imaging were crucial to establish the final diagnosis.[6]

While acknowledging the limited evidence base, we suggest that IVT might be considered in selected cases of acute spinal cord ischemia, after careful exclusion of contraindications such as haemorrhage or aortic dissection, and in centres with established experience in thrombolytic therapy. These observations highlight the need for more systematic data collection and potentially prospective trial to better define the role of IVT in spinal cord infarction.

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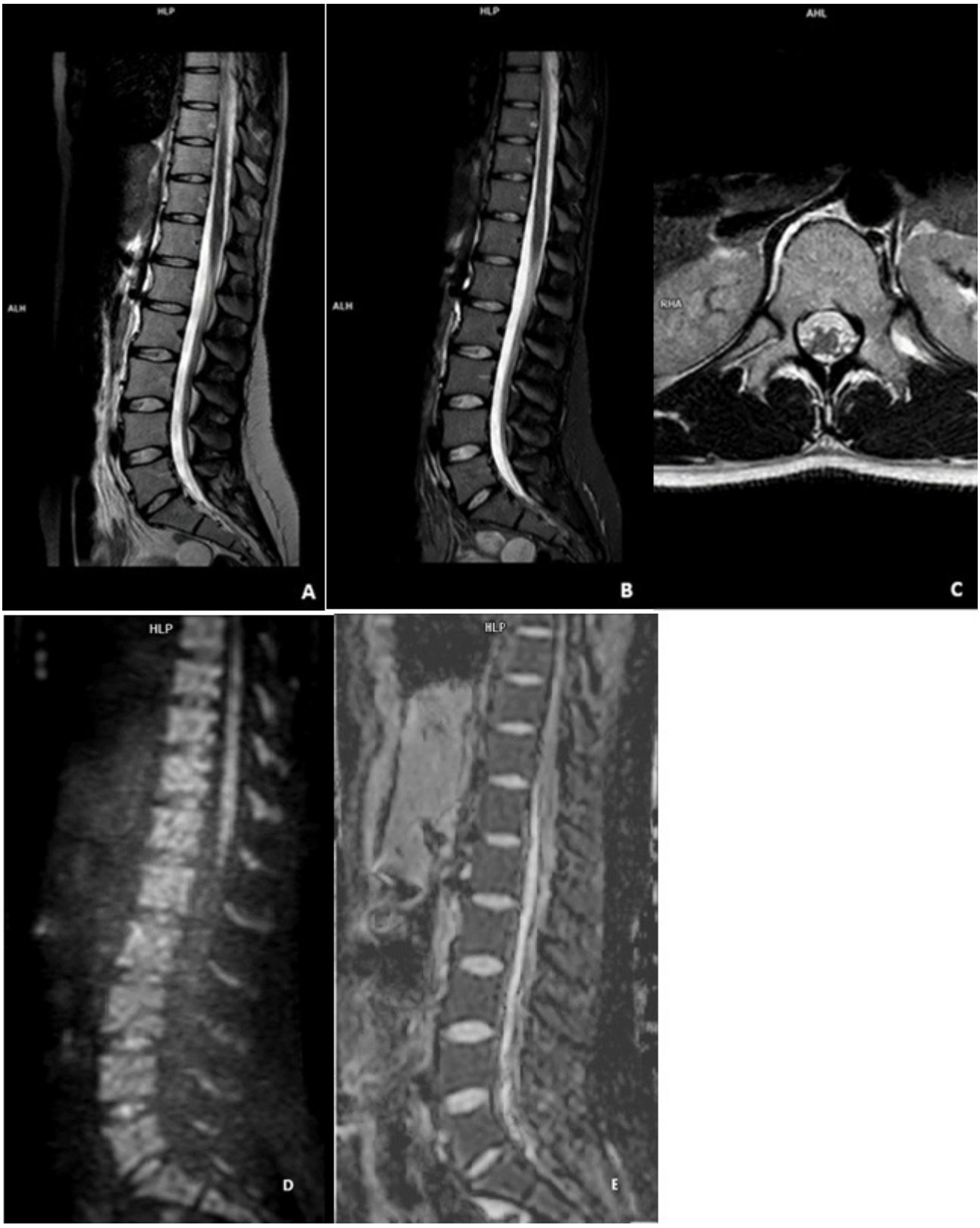


Figure 1. Acute phase. 1,5 T MRI . Sagittal (A) and axial (C) T2 TSE, sagittal STIR (B) , DWI (D), ADC (E) unremarkable. T1 Dixon Images with and without contrast are not shown (no pathological findings).

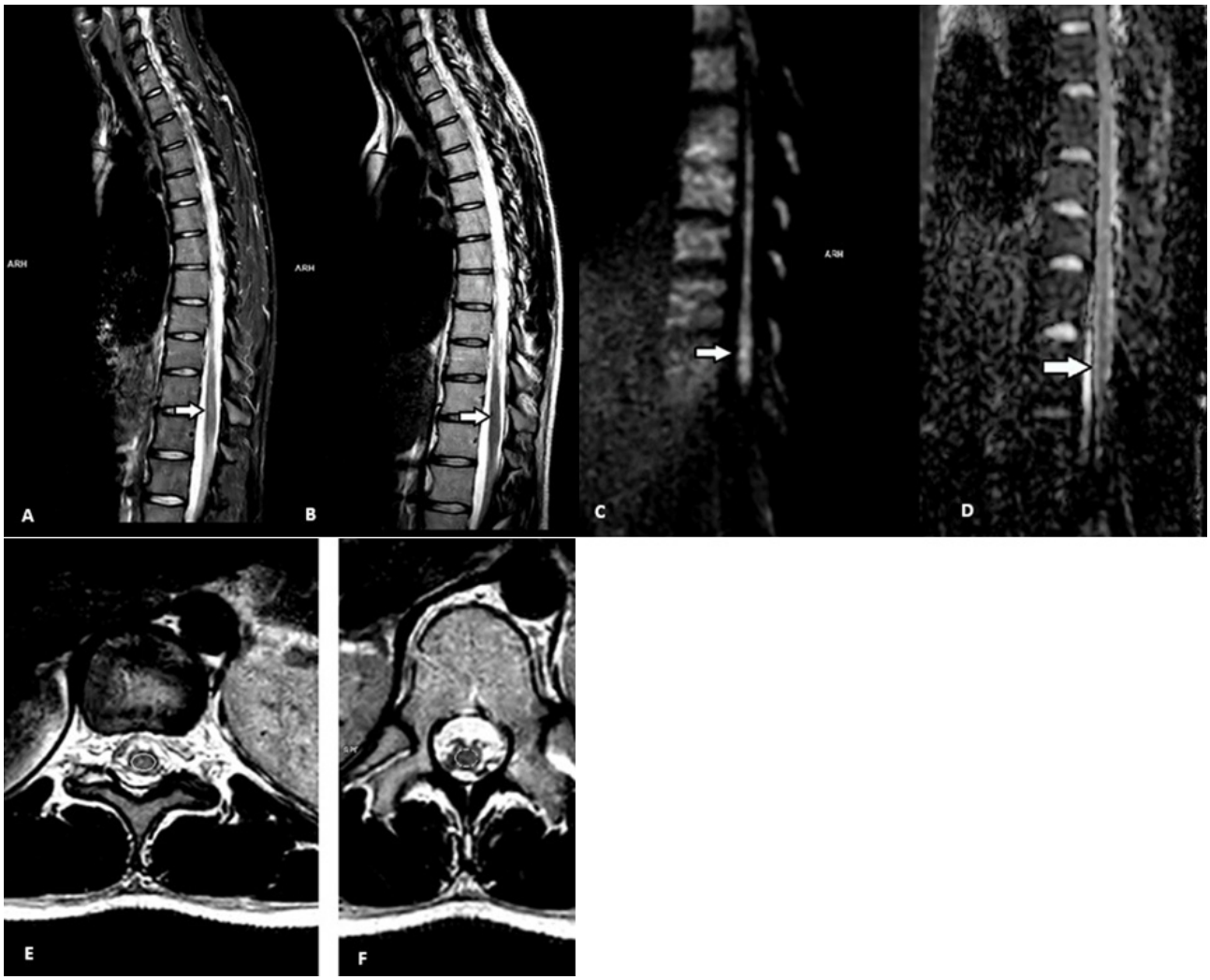
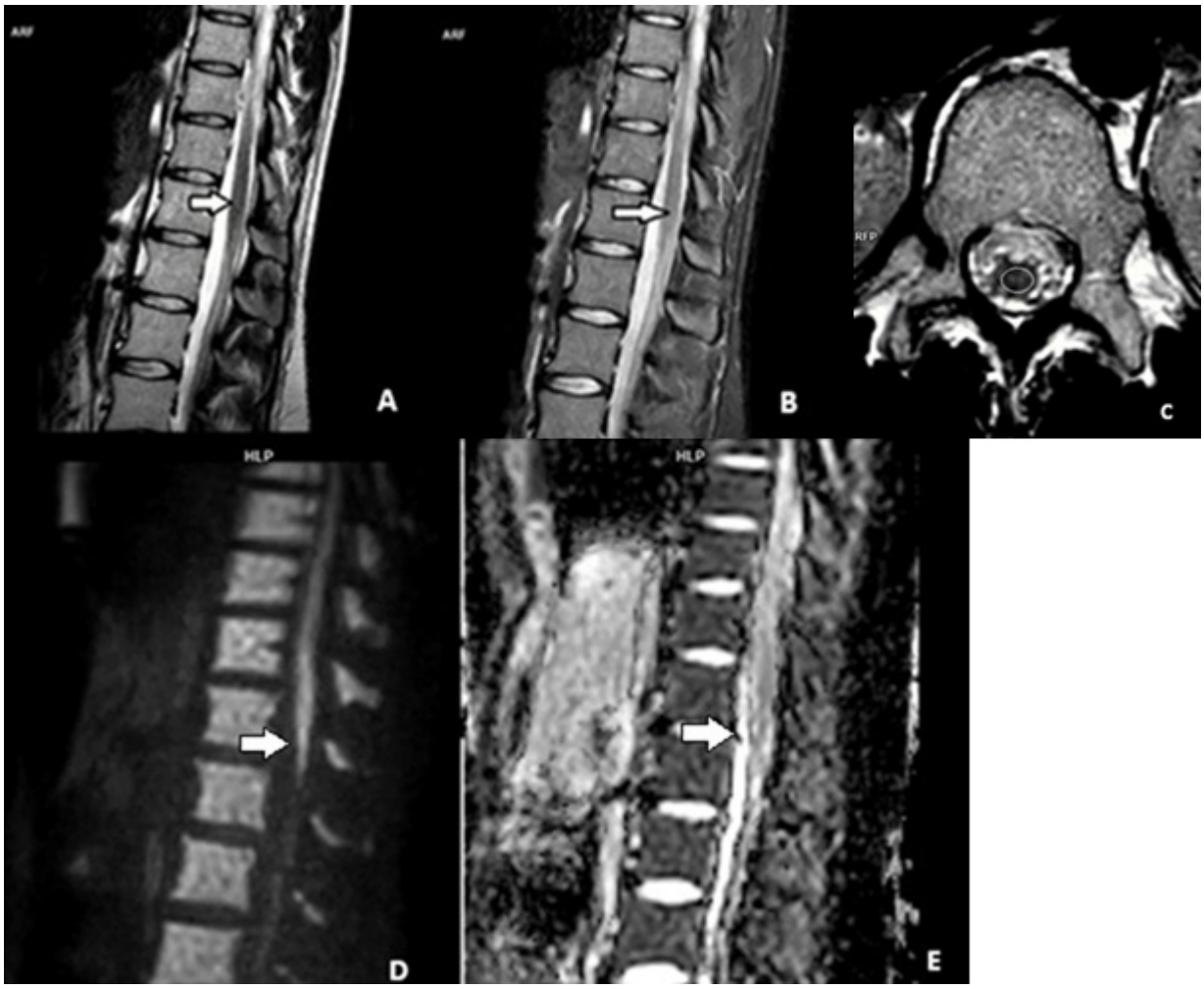


Figure 2. First follow up, ten days later. Sagittal STIR (A) and T2 (B) show subtle linear hyperintensities in the anterior-central part of conus medullaris/D11-D12, corresponding subtle and faded spot of restricted diffusion in sagittal DWI (C)/ADC (D) indicating very limited ischemic changes. Axial T2 TSE one hyperintense spot in central conus medullaris D12/L1 (E) corresponding to central gray matter involvement. D11-D12 in left image (F), two central-anterior hyperintense spot resembling “owl-eyes” in the right image corresponding to anterior horns involvement.



Figures 3. Second follow-up, 3 months and 10 days later. Sagittal T2 TSE (A) /STIR (B) , axial T2 TSE (C) DWI (D) / ADC (E). More fading but similar findings as in figure 2, reflecting natural evolution/ initial gliosis.

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Ethics approval and consent to participate: no ethical committee approval was required for this case report by the Department, because this article does not contain any studies with human participants or animals. Informed consent was obtained from the patient included in this study.

Patient consent for publication: the patient gave her written consent to use her personal data for the publication of this case report and any accompanying images.

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