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CASE REPORT

Spinal Cord Infarction as an Atypical Thromboembolic Complication of First-Diagnosed Atrial Fibrillation

Georgios Tyrikos[1,*], Paschalis Karakasis[1,2,*], Vasileios Nevras[1], Nikolaos Zelevaris[1], Chrysa Nalmpantidou[3], Georgia Gkounti[1]

[1]Cardiology Department, G. Gennimatas Hospital, Thessaloniki, Greece; [2]Second Department of Cardiology, Aristotle University of Thessaloniki, General Hospital Hippokration, Thessaloniki, Greece; [3]Radiology Department, G. Gennimatas Hospital, Thessaloniki, Greece

*These authors contributed equally to this work

Correspondence: Paschalis Karakasis, Second Department of Cardiology, Aristotle University of Thessaloniki, General Hospital Hippokration, Thessaloniki, Greece, Email pakar15@hotmail.com

Background: Atrial fibrillation (AF) is the most common sustained arrhythmia and a major contributor to systemic thromboembolism, typically manifesting as ischemic stroke or peripheral arterial occlusion. Although spinal cord infarction (SCI) represents a rare form of ischemic injury, accounting for only 1–2% of all stroke events, its consequences are often devastating. The pathophysiological link between AF and SCI is poorly recognized, particularly in the setting of concurrent pulmonary embolism and hemodynamic instability.

Case Summary: We report the case of a 91-year-old woman with chronic comorbidities who presented with first-diagnosed, asymptomatic AF and was found to have a submassive pulmonary embolism. Following initiation of anticoagulation, she developed acute flaccid paraplegia and hypotension. Spinal magnetic resonance imaging revealed a longitudinally extensive anterior-predominant T2 hyperintensity from T6 to the conus medullaris, consistent with acute anterior spinal artery territory infarction. Despite aggressive hemodynamic support, the patient's condition deteriorated and she died shortly thereafter.

Conclusion: SCI is a rare but catastrophic complication of AF. In patients with AF—especially those with concomitant venous thromboembolism or hypotension—new-onset paraplegia should prompt urgent spinal MRI to exclude ischemic myelopathy. Early recognition may guide supportive strategies, although prognosis remains poor in extensive thoracolumbar infarctions.

Keywords: atrial fibrillation, spinal cord infarction, thromboembolism, cardiovascular imaging, magnetic resonance imaging, pulmonary embolism

Introduction

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia, affecting millions worldwide and posing a major public health challenge due to its association with thromboembolism, heart failure, and mortality.[1] Of importance, up to 30% of AF cases are asymptomatic, yet silent AF confers a thromboembolic risk comparable to symptomatic presentations, underscoring the need for proactive detection, risk stratification and effective intervention.[2,3] AF is a well-established risk factor for thromboembolism, most commonly presenting as ischemic stroke or systemic arterial embolism.[4] In contrast, spinal cord infarction (SCI) represents a rare manifestation of embolic disease, accounting for only 1–2% of all ischemic events, yet it is associated with considerable morbidity and mortality.[5–7] Diagnosis is challenging because symptom onset is abrupt while early neurological signs can be non-specific; timely access to definitive imaging therefore determines diagnostic certainty.[8]

Spinal cord infarction (SCI) is an uncommon cause of acute myelopathy, accounting for a small fraction of central nervous system ischemic events, yet it is associated with substantial long-term disability.[9,10] Recent cohort studies and systematic reviews have shown that the most frequent etiologies include aortic surgery and aortic pathology, followed by non-iatrogenic causes such as systemic hypotension, chronic vascular risk factors, and presumed cardioembolism, while a substantial proportion of cases

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remain idiopathic despite extensive work-up.[11,12] The anterior spinal artery (ASA) supplies the anterior two-thirds of the cord, and its occlusion or critical hypoperfusion typically results in anterior cord syndrome, characterized by acute paraplegia or paraparesis with dissociated sensory loss and relative sparing of dorsal column modalities, often accompanied by sphincter dysfunction. Within this spectrum, AF represents an important but likely under-recognized contributor to SCI: AF is a major source of cardioembolic thrombi, and although embolic events most commonly involve the cerebral circulation, case series and observational data suggest that emboli may rarely lodge in radiculomedullary arteries or the ASA, leading to spinal cord ischemia.[9] In addition, systemic hypotension or concomitant pulmonary embolism may further compromise cord perfusion in watershed territories, amplifying the vulnerability of the thoracic cord.[13]

In the milieu of first-diagnosed AF with concomitant pulmonary embolism and intermittent hypotension, an interplay between distal arterial embolization and systemic hypoperfusion is plausible.[14] We report a case of MRI-confirmed SCI shortly after AF diagnosis and submassive pulmonary embolism, using it to delineate practical diagnostic thresholds, discuss mechanistic nuances relevant to cardiology, and emphasize therapeutic priorities in a condition for which disease-modifying treatments remain limited.

Case Presentation

A 91-year-old woman with heart failure with preserved ejection fraction, chronic kidney disease, hypertension, and hyperuricemia presented with a three-day history of chest pain. Long-term medications included furosemide, spironolactone, empagliflozin, and allopurinol. On arrival she was comfortable and hemodynamically stable The electrocardiogram demonstrated first-diagnosed AF with left bundle branch block, without AF-related symptoms. Laboratory testing showed elevated D-dimer levels and a mild troponin rise. Computed tomography pulmonary angiography revealed a submassive embolus involving the left main pulmonary artery without radiographic features of right heart strain. Transthoracic echocardiography reported mildly reduced left ventricular ejection fraction with preserved right ventricular function and no significant valvular disease. Oral anticoagulation with dabigatran was commenced, and symptoms initially improved. On the third hospital day, she developed the sudden onset of bilateral lower-extremity paralysis accompanied by hypotension, with systolic blood pressure in the mid-90s mmHg. There was no back pain, trauma, or prodromal febrile illness. Neurological examination suggested a spinal level myelopathy with flaccid paraplegia and impaired pinprick and temperature sensation below the umbilicus, raising concern for anterior cord involvement. Emergent spinal MRI was obtained. Axial T2-weighted MRI at the T9–T10 level demonstrated symmetric anterior horn hyperintensity with the classic "owl's eye" appearance,[13] consistent with anterior spinal artery territory ischemia (Figure 1). Corresponding post-contrast T1-weighted images did not show abnormal enhancement, vessel malformations, or compressive lesions (Figure 2). Sagittal STIR and T2-weighted sequences revealed a longitudinally extensive, anteriorly predominant intramedullary T2 hyperintensity with mild cord swelling extending from T8 to T11 (Figure 3), in keeping with an acute ischemic insult of the anterior cord. Diffusion-weighted imaging was not available at the time of scanning; however, the clinical–radiographic pattern was considered highly characteristic of acute SCI.[8] Hemodynamic support with norepinephrine and dobutamine was instituted to improve spinal cord perfusion pressure while carefully maintaining oxygenation and hemoglobin targets. Despite escalation of vasoactive therapy, progressive instability ensued and the patient died shortly thereafter.

Discussion

This case broadens the recognized thromboembolic manifestations of AF to encompass spinal cord infarction—an infrequent and often underappreciated entity, particularly when neurological deficits emerge below the level of the brain and are not immediately attributed to a central embolic source. The temporal sequence—first-diagnosed AF and submassive pulmonary embolism treated with anticoagulation, followed by paraplegia and hypotension—supports a dual mechanism. Embolic occlusion of the anterior spinal artery or its radiculomedullary feeders may precipitate infarction of the spinal cord, while concomitant systemic hypotension—especially in older patients with impaired autoregulatory reserve—can exacerbate vulnerability in watershed zones, converting marginal perfusion into critical ischemia. The thoracolumbar cord is especially vulnerable because ASA flow beyond the midthoracic region depends on a small number of radiculomedullary arteries, most notably the artery of Adamkiewicz, which typically arises between T8 and L2; interruption or underperfusion in this region may lead to extensive ischemia.[15] Although paradoxical embolism is theoretically possible in the setting of venous thromboembolism, there was no clinical suggestion of a right-to-left shunt; thus, the mechanistic weight here likely rests on arterial embolism from AF and concomitant hypoperfusion.

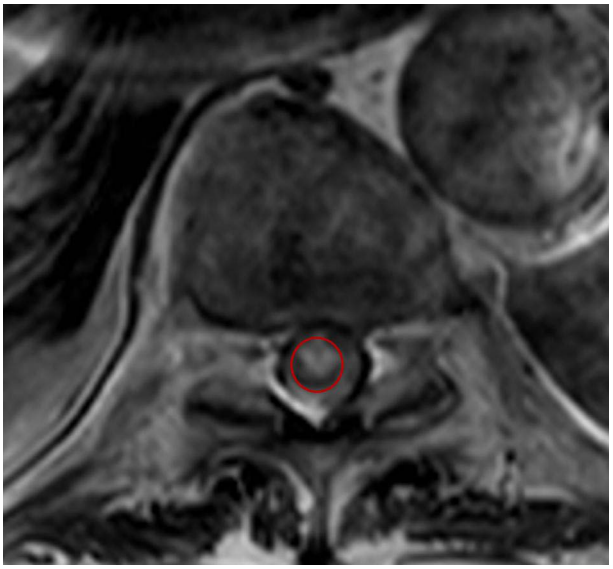

Figure 1 Axial T2-weighted MRI of the lumbar spine demonstrating the classic "owl's eye" appearance, with bilateral, symmetric T2 hyperintensities in the anterior horn regions, highlighted within the red circle.

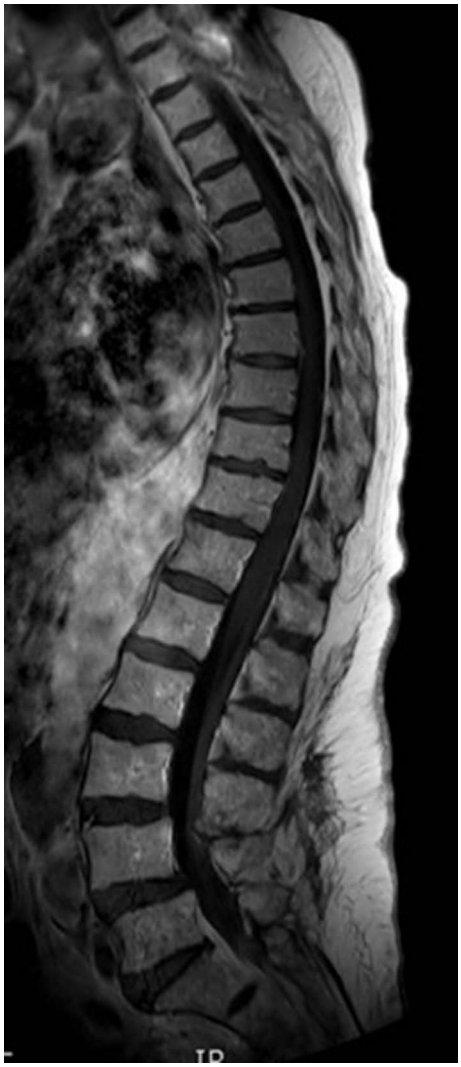

Figure 2 Post-contrast T1-weighted MRI following intravenous contrast administration demonstrates no abnormal contrast enhancement.

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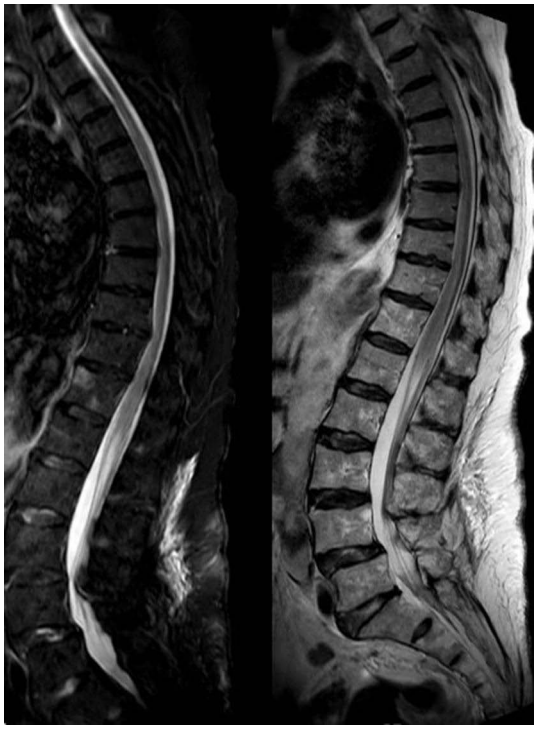

Figure 3 Sagittal short tau inversion recovery (STIR, left) and T2-weighted (right) MRI of the spine demonstrate a longitudinally extensive, centrally located intramedullary T2/STIR hyperintense lesion with associated mild spinal cord expansion.

The diagnostic pathway in suspected SCI requires both a low threshold for imaging and alertness to mimics. The differential diagnosis includes compressive myelopathy (tumor, epidural abscess, hematoma), transverse myelitis, demyelination, metabolic myelopathy, and peripheral neuropathic processes.[12] From a diagnostic standpoint, the principal differentials for this acute thoracic myelopathy included spinal cord infarction, transverse myelitis, demyelinating disorders, and compressive myelopathies. Several features favored a vascular etiology consistent with SCI: the abrupt onset and rapid progression of paraplegia, the "owl's eye" pattern of bilateral anterior horn involvement on axial T2-weighted imaging, and the longitudinally oriented T2/STIR hyperintensity with mild cord expansion and anterior predominance, all in the context of newly diagnosed AF, submassive pulmonary embolism, and intermittent hypotension. In contrast, transverse myelitis and other inflammatory myelopathies typically present with a more subacute time course, often accompanied by prodromal systemic symptoms, elevated inflammatory markers, and cerebrospinal fluid pleocytosis or oligoclonal bands, none of which were evident in our patient. Demyelinating disorders such as multiple sclerosis or neuromyelitis optica spectrum disorder would be expected to show either multifocal lesions disseminated in space and time, characteristic periventricular or optic pathway involvement, or longitudinally extensive cord lesions with distinct enhancement patterns, together with disease-specific serological or CSF abnormalities; these features were absent. Compressive etiologies, including epidural hematoma, neoplasm, or degenerative canal stenosis, were excluded by MRI, which demonstrated no extrinsic mass lesion, epidural collection, or significant spinal cord compression. Taken together, the clinical tempo, laboratory profile, and spinal MRI characteristics strongly support the diagnosis of anterior spinal artery territory infarction rather than inflammatory, demyelinating, or compressive myelopathy.

In anticoagulated patients, epidural hematoma is a key concern; however, the absence of compressive pathology on MRI alongside a longitudinally extensive, anterior-predominant T2 hyperintensity strongly favors ischemia. Classical radiological signs include the "owl's eyes" appearance on axial T2 from bilateral anterior horn involvement and, when available, diffusion restriction with low apparent diffusion coefficient in the acute phase.[12] Gadolinium enhancement is variable and typically a subacute feature. Importantly, early MRI can be nondiagnostic; repeating MRI or adding diffusion sequences after several hours may unmask changes.[12] These criteria are consistent with proposed diagnostic frameworks for spontaneous SCI, which integrate clinical timing, neuroanatomical localization, and characteristic MRI patterns.

Therapeutic options for acute SCI remain limited and largely supportive.[16] Unlike large-vessel cerebral occlusion, there is no established endovascular reperfusion strategy for spontaneous ASA occlusion, and randomized evidence is lacking.[16] Management therefore focuses on restoration and maintenance of adequate spinal cord perfusion pressure, avoidance of hypotension and hypoxemia, temperature control, and optimization of hemoglobin and cardiorespiratory status.[16] While recommendations from traumatic spinal cord injury contexts suggest targeting mean arterial pressures in the 85–90 mmHg range for the first week, extrapolation to ischemic etiologies is empiric and should be individualized.[16] Corticosteroids have no proven role in ischemic myelopathy and may be harmful.[16] Antithrombotic management in elderly patients with chronic kidney disease and recent pulmonary embolism requires careful balancing of embolic and bleeding risks, recognizing that reversal of established neurologic injury is not currently achievable.

Against this backdrop of therapeutic uncertainty, our case underscores a rare and easily overlooked manifestation of AF-related thromboembolism—spinal cord infarction—in the setting of first-diagnosed AF. While current guidelines provide clear recommendations for stroke prevention in non-valvular AF, they do not specifically address thromboembolic events involving the spinal cord, leaving a substantial evidence gap regarding the optimal antithrombotic strategy in this context.[17] In our patient, once intracranial and intraspinal hemorrhage had been reasonably excluded and no contraindications were present, we elected to initiate therapeutic oral anticoagulation with dabigatran, in line with contemporary practice for acute thromboembolic events in newly diagnosed non-valvular AF. The initial presentation was subacute, without hemodynamic instability or rapidly progressive neurological deficit, and there is no robust evidence supporting routine use of intravenous unfractionated heparin in spinal cord ischemia, whereas more aggressive parenteral anticoagulation may increase the risk of hemorrhagic transformation within the cord. On this basis, the choice of a DOAC represented a deliberate balance between effective thromboembolic protection and bleeding risk. Nonetheless, given the rarity of AF-associated spinal cord infarction, the optimal intensity and route of anticoagulation remain uncertain, and more intensive parenteral regimens might be considered on an individualized basis in patients with ongoing cord ischemia or clinical deterioration.[18]

Of note, prognosis correlates with infarct length, completeness of the motor deficit, early autonomic involvement, and hemodynamic instability.[13] Longitudinally extensive infarctions spanning multiple thoracolumbar segments, as in this case, portend poor functional recovery even when patients survive the acute hospitalization.[10] Survivors benefit from early, intensive rehabilitation, albeit with a high likelihood of persistent motor and sphincter dysfunction. From a cardiology perspective, this case reinforces the importance of vigilant bedside assessment for atypical thromboembolism in AF. Although CHA_2DS_2-VA and HAS-BLED frameworks remain central to long-term decision-making,[17] new or rapidly progressive bilateral leg weakness, acute areflexia, or dissociated sensory loss in an AF patient—particularly in the setting of venous thromboembolism or hypotension—should trigger urgent spinal MRI and early multidisciplinary coordination among cardiology, neurology, neuroradiology, and critical care.

Conclusion

SCI represents a rare but devastating thromboembolic manifestation of AF, for which evidence-based, disease-specific therapies are currently lacking. In patients with AF who develop acute paraplegia—particularly in the setting of pulmonary embolism, hypotension, or other systemic embolic phenomena—clinicians should maintain a high index of suspicion, promptly obtain spinal MRI with diffusion-weighted sequences when available, and institute perfusion-directed supportive care. Our case highlights the central role of multimodality imaging in establishing the diagnosis, the need for individualized antithrombotic decision-making in the absence of clear guideline recommendations, and the importance of early recognition to optimize neurological and systemic outcomes in this vulnerable population.

Data Sharing Statement

No data were generated in this study.

Patient Consent

Written informed consent for publication, including all relevant clinical details and imaging, was obtained from the patient's next of kin. Approval from the institutional review board was not required for this single-patient case report in accordance with local ethical guidelines.

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Disclosure

The authors report no conflicts of interest in this work.

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