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CASE REPORT **OPEN ACCESS**

SPINAL CORD INFARCTION AS A RARE NEUROLOGICAL COMPLICATION OF ANTERIOR STEMI FOLLOWING FIBRINOLYTIC THERAPY: A CASE REPORT

Novia Heriza*, Subandi

Correspondence: *noviaheriza.nh@gmail.com*
Department of Neurology, Faculty of Medicine, Universitas Sebelas Maret, Surakarta, Indonesia

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ABSTRACT

Background: Spinal cord infarction (SCI) is a rare cause of acute neurological deficits and an exceedingly uncommon embolic complication of acute myocardial infarction (AMI). The primary mechanism involves cardioembolism originating from a left ventricular (LV) mural thrombus. We report a rare case of SCI presenting as acute paraplegia following fibrinolysis for anterior ST-elevation myocardial infarction (STEMI).
Case: A 53-year-old man presented with typical chest pain. Electrocardiography demonstrated anterior STEMI, and fibrinolytic therapy with streptokinase was administered immediately. Six hours later, he developed sudden bilateral lower-limb weakness, sensory loss from the toes to the T10 level, and urinary retention. Neurological examination showed complete paraplegia (motor strength 0/0) with loss of pain and temperature sensation below T10, while vibration and proprioception were preserved, consistent with Anterior Spinal Artery Syndrome (ASAS). Spinal magnetic resonance imaging revealed T1-hypointense and T2-hyperintense lesions in the anterior horn at the T10 level, confirming anterior spinal artery infarction.
Discussion: Acute paraplegia after fibrinolysis was strongly suspected to result from an embolic shower caused by destabilization of an LV mural thrombus, leading to occlusion of the radiculomedullary artery and subsequent ASAS. Because neurological recovery is often limited, prompt anticoagulation and early rehabilitation are essential to optimize outcomes.
Conclusion: SCI is a rare but serious complication of anterior STEMI associated with LV thrombus formation, which may cause cardiogenic embolism through the artery of Adamkiewicz. Clinicians should maintain a high index of suspicion for spinal cord stroke in post-STEMI patients, particularly after fibrinolytic therapy, to facilitate timely diagnosis and management.

Keywords: anterior STEMI, artery of Adamkiewicz, fibrinolysis, rare case, spinal cord infarction

Introduction

Spinal cord infarction (SCI) is an exceedingly rare clinical entity, with an estimated prevalence accounting for less than 1% to 2% of all ischemic stroke cases globally.[1,2] This incidence rate stands in stark contrast to the high prevalence of cerebral strokes, which frequently occur as a primary complication of cardiogenic embolism following an anterior STEMI.[3] The rarity of spinal cord embolization compared to cerebral involvement is attributed to unique anatomical barriers, particularly the minute caliber of the blood vessels and the acute branching angles of the spinal arteries from the aorta.[4] The branching angles of the intercostal and radicular arteries, which tend to be perpendicular to the descending aorta, mechanically impede large embolic fragments from entering the spinal circulation unlike the blood flow pathways to the cerebral circulation, which are more linear and high-pressure. Consequently, the clinical manifestation of SCI resulting from cardioembolism is an exceptionally rare phenomenon that necessitates a high degree of clinical suspicion in patients presenting with potent cardiogenic risk factors.

Anterior-wall ST-segment elevation myocardial infarction (STEMI) is consistently identified as the highest risk factor for cardioembolic complications compared to other infarct locations.[5] Anatomically, this condition results from the occlusion of the left anterior descending (LAD) coronary artery, leading to extensive transmural damage in the apex and the anterior wall of the left ventricle.[6] Such massive myocardial damage triggers severe segmental contractility impairment, manifesting as akinesia or the development of a left ventricular aneurysm.[6] These structural alterations create an abnormal kinetic substrate, where the loss of cardiac pump synchronization at the apex induces significant disruptions in intraventricular blood flow dynamics.[7]

Case Report

A 53-year-old male presented with a diagnosis of Anterior STEMI and inferior paraplegia. Five days after admission, the patient experienced a myocardial infarction and received fibrinolytic therapy four hours after onset. Six hours after fibrinolysis, the patient complained of numbness, tingling (paresthesia), and sudden weakness in both lower extremities, originating from the umbilicus downward. The weakness was progressive; initially, the patient maintained minimal movement, but it developed into total paralysis within 24 hours. The patient denied any history of severe back pain, spinal trauma, fever, or seizures. There was no prior history of hypertension, diabetes mellitus, or malignancy, with heart disease being newly diagnosed during this episode.

Cranial nerve examination and higher cortical functions showed no abnormalities. Neurological status revealed UMN-type inferior paraplegia with motor strength of 0/0, normal physiological reflexes (+1), and negative pathological reflexes. Sensory examination demonstrated hypoesthesia and paresthesia consistent with the T10-T11 dermatome levels. Autonomic function currently requires a dwelling catheter, although sensations for voiding and defecation were reported to be intact. No tenderness was found over the vertebral column, and signs of radicular irritation (Laseque and Kernig) were negative. Based on these clinical manifestations, the working diagnosis was suspected SCI at the T10-T11 segment, occurring post-cardiac thromboembolic event or other systemic vascular complications. Cardiographic evaluation via transthoracic echocardiography revealed left ventricular dilatation with significantly reduced ejection fraction and akinesia in the apical and anterior segments. Subsequently, spinal Magnetic Resonance Imaging (MRI) on T2-weighted sequences showed localized intramedullary hyperintensity in the anterior horns of the spinal cord at the thoracic 10 segment, producing the classic "owl's eye appearance" on axial sections. This radiologically confirms an infarction in the territory of the Anterior Spinal Artery without extrinsic mechanical compression. These findings are pivotal in the diagnosis of Anterior Spinal Artery Syndrome and provide a clear illustration of a rare yet significant spinal cord infarction.

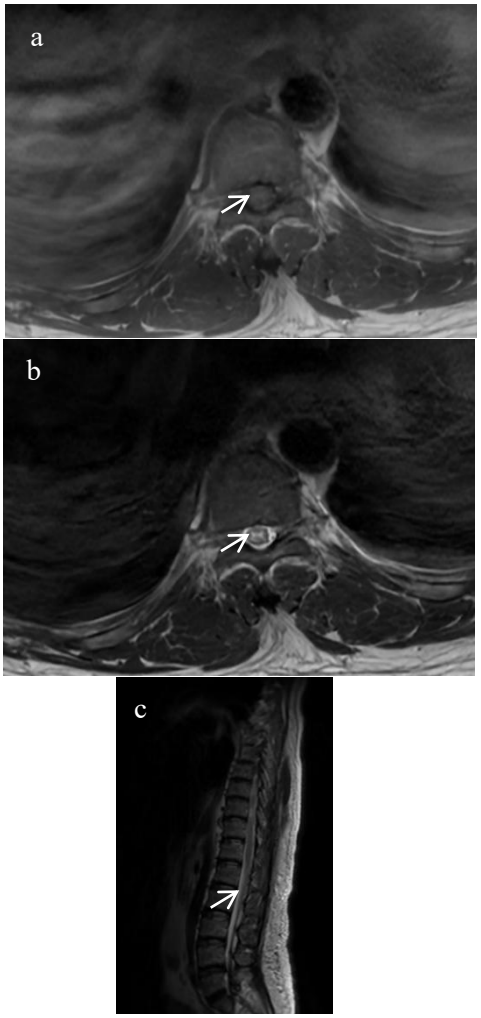

Figure 1. (a) Findings on contrast-enhanced thoracic MRI: axial T1-weighted sequence demonstrates intramedullary hypointensity. (b) Findings on contrast-enhanced thoracic MRI: T2-weighted sequence reveals intramedullary hyperintensity, producing the characteristic "owl's eye appearance". (c) Findings on contrast-enhanced thoracic MRI: Ssagital T1-weighted sequence demonstrates intramedullary hypointensity (white arrow)

Discussion

A crucial aspect of this case is the potential paradoxical role of fibrinolytic therapy in triggering systemic embolization. Based on several case reports, it is hypothesized that fibrinolytic agents may induce instability in pre-existing mural thrombi by partially dissolving the surface fibrin matrix, thereby triggering

fragmentation.[10,18] This phenomenon, often described as an "embolic shower," allows dislodged thrombus fragments to circulate systemically immediately after partial lysis occurs.[2] The temporal relationship between the medical intervention and the onset of symptoms serves as primary supporting evidence, where the rapid onset of neurological symptoms within hours after fibrinolysis strongly supports the hypothesis of therapy-induced embolization.[11] In this patient, the emergence of acute paraplegia shortly after fibrinolytic administration provides a strong clinical clue that mural thrombus destabilization due to chemical lysis was likely the primary triggering mechanism for the spinal cord infarction.[12]

The mechanism of mural thrombus formation in this condition follows Virchow's Triad, where blood stasis acts as the primary determinant alongside endocardial injury and a systemic hypercoagulable state.[7,17] Stagnant blood flow within akinetic areas or aneurysmal sacs facilitates progressive fibrin deposition and platelet aggregation.[13] Mural thrombi formed on damaged endocardial surfaces tend to be unstable and carry a high risk of fragmentation[4]. Cardioembolism occurs when these thrombus fragments are released into the systemic circulation, frequently resulting in embolic ischemic strokes and worsening both functional prognosis and patient mortality.[14]

Systemic embolization originating from an intracardiac thrombus is a rare but well-documented etiology of ischemic spinal cord infarction.[11] These embolic fragments can migrate from the left ventricle through the descending aorta and potentially occlude the great anterior radiculomedullary artery (Artery of Adamkiewicz),[1] which provides the dominant blood supply to the anterior two-thirds of the spinal cord, particularly in the T9 to L2 thoracolumbar segments.[12] Occlusion of this critical artery leads to blood flow disruption in the Anterior Spinal Artery (ASA), clinically triggering Anterior Spinal Artery Syndrome (ASAS), characterized by sudden paraplegia and sphincter dysfunction.[5] Although the incidence of cardiogenic emboli to spinal vessels is significantly lower than to cerebral vessels, this mechanism remains a primary consideration in patients with significant pro-thrombotic cardiac conditions.[2]

The patient's clinical presentation, marked by the sudden onset of paraplegia and severe sensory impairment, strongly points toward a diagnosis of ASAS.[1] This syndrome results from ischemia in the anterior two-thirds of the spinal cord, a territory exclusively supplied by the ASA.[11] The classic finding of loss of pain and temperature sensation, with preserved proprioception and vibration (joint position sense), reflects damage to the spinothalamic tracts and anterior horns, while the posterior columns remain sufficiently perfused by the intact posterior spinal arteries.[5] The acute motor deficits and dissociative sensory impairment pattern in this case anatomically reinforce the diagnosis of a spinal cord infarction localized within the ASA vascular territory.[12] Correlating these specific clinical manifestations with the vascular anatomical distribution provides a robust diagnostic foundation for concluding an occlusion of the anterior spinal arterial supply.[2]

Clinical management in this patient presents a significant dilemma; the initiation of systemic anticoagulation is essential to prevent recurrent embolic events from the left ventricular thrombus, yet it simultaneously carries a high risk of triggering post-fibrinolytic bleeding complications.[4,19] The decision to start anticoagulant therapy must involve a precise balance between the risk of further systemic embolic stroke and the potential for hemorrhagic transformation within the newly infarcted spinal cord area. In line with current medical literature,[7] clinical outcomes for ischemic SCI consistently show a poorer motor recovery prognosis compared to cerebral strokes, with the majority of patients retaining permanent neurological deficits.[11] Given the limitations of spontaneous neurological recovery in SCI, clinical protocols emphasize the urgency of early and aggressive medical rehabilitation to facilitate neuroplasticity and prevent secondary complications such as contractures and pressure ulcers.[1] A multidisciplinary approach integrating meticulous vascular management with intensive physiotherapy is the primary determinant in optimizing the patient's functional independence, even though total anatomical motor recovery is often not achieved.[2]

Several clinical and laboratory markers have been identified to predict a higher risk of such systemic embolization. From an echocardiographic standpoint, thrombi characterized by high mobility, protrusion into the left ventricle (LV) cavity, and a pedunculated shape carry a significantly elevated embolic risk compared to flat, laminated thrombi. Clinically, a severely depressed left ventricular ejection fraction (LVEF) (<30–35%), anterior myocardial infarction, and delayed presentation to reperfusion therapy further compound this risk. Moreover, laboratory findings such as a rapid, disproportionate spike in D-dimer levels shortly after fibrinolysis, alongside elevated inflammatory markers (e.g., high-sensitivity C-reactive protein and high neutrophil-to-lymphocyte ratio), may reflect intense thrombus destabilization and ongoing subclinical fragmentation, serving as potential surrogates for an impending embolic event.[15] Given the very narrow time window between fibrinolytic administration and the onset of paraplegia in a patient

with sudden spinal vascular impairment, the mechanism of thrombus fragmentation triggered by mechanical and chemical changes becomes the most rational explanation compared to the natural progression of spinal microvascular disease itself, as microvascular disorders typically develop more slowly in a clinical setting.[3,16]

Conclusion

It is imperative for clinicians to maintain heightened clinical vigilance regarding the emergence of new neurological deficits in patients following an acute myocardial infarction. The onset of acute paraplegia after an anterior STEMI must be meticulously investigated, as this condition may indicate a catastrophic embolic event within the spinal cord vasculature.[20] Although the incidence of SCI is significantly lower than that of cerebral stroke, it must be promptly considered as a primary differential diagnosis to prevent delays in management and to initiate appropriate neuroprotective strategies. Emphasizing this awareness is crucial in daily clinical practice, where early detection of spinal ischemia in patients with a high risk of left ventricular thrombus can significantly determine the long-term functional prognosis and the patient's overall quality of life.

Declaration of Interests

The author declares that there are no conflicts of interest regarding the research described in this case report. No financial support was received for this study.

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