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Case Report

Oral Nitroglycerine - Induced Spinal Cord Infarction: A Case Report

Ranadheer R Dande[1]*, Anusha Tipparthi[2], Yashwanth R Sudhini[3], Taruni Suram[4] and Ashok R Polu[5]

[1]*Department of Internal Medicine, Baptist Health Hardin Hospital, Elizabethtown, KY, United States*

[2]*Department of Internal Medicine, Appalachian Regional Healthcare, Harlan, KY, United States*

[3]*Department of Neurology, Rush University Medical Center, Chicago, IL, United States*

[4]*Department of Surgery, Prathima Institute of Medical Sciences, Karimnagar, India*

[5]*Department of Neurology, Fort Sanders Regional Medical Center, Knoxville, TN, United States*

***Corresponding Author:** Ranadheer R Dande, Department of Internal Medicine, Baptist Health Hardin Hospital, Elizabethtown, KY, United States.

Received: August 12, 2024; **Published:** September 04, 2024

Abstract

Spinal cord ischemic strokes are rare. Systemic hypotension resulting in spinal cord ischemic stroke is rare compared to Cerebral ischemic strokes. This case report discusses a 73-year-old male with severe atherosclerotic disease who developed a spinal cord ischemic stroke with extremity weakness after experiencing iatrogenic hypotension from oral nitroglycerine taken for chest pain.

Keywords: *Oral Nitroglycerine; Spinal Cord Infarction; Cerebral Ischemic Strokes*

Background

73 y/o male with a history of severe atherosclerotic disease and risk factors including hypertension, dyslipidemia, and type 2 diabetes mellitus presented to the emergency department (ED) with concerns for dizziness, fall, and extremity weakness. Upon arrival, he was noted to be hypotensive, likely secondary to the nitroglycerine that he took before presenting to the ED for chest discomfort. Further workup showed Spinal cord ischemic stroke contributing to the weakness in the extremities likely due to Nitroglycerine induced systemic hypotension.

Objective of the Study

To educate and report on the rare incidence of spinal cord ischemic stroke due to systemic hypotension from sublingual nitroglycerine.

Case Presentation

A 73-year-old male with a medical history of hypertension, dyslipidemia, type 2 diabetes mellitus, coronary artery disease status post coronary stents and coronary artery bypass graft (CABG), and obstructive sleep apnea on continuous positive airway pressure (CPAP), presented to the emergency department (ED) with chest pain, lightheadedness, extremity weakness, fall. The symptoms began two days before hospital admission after he worked in the yard for an extended period. The patient experienced bilateral shoulder pain radiating down his arms, which he ignored and attributed to the yard work. The following day, the shoulder pain resolved but he experienced chest pain and self-medicated with nitroglycerin. Subsequently, his family noticed he was dizzy, and experienced a fall. They also noted that he was unable to move his right lower extremity and left upper extremity, prompting them to bring him to the hospital.

On day 1 of the hospital presentation, initial triage revealed significant hypotension (blood pressure [BP] 80/40, dropping to 57/32), which improved with fluid administration. A code stroke was called, and the National Institutes of Health Stroke Scale (NIHSS) score of 2 was given for the weakness in the right lower extremity by the ED team. Diagnostic imaging, including a non-contrast computed tomography (CT) head and computed tomography angiography (CTA) of the head and neck, showed no acute hemorrhage, large vessel occlusions, or significant stenosis. CTA of the chest/abdomen was also obtained to rule out aortic dissection and was noted to have an incidental small pulmonary embolism (PE) in the left lower lobe. Despite improvements in left upper extremity weakness transiently and chest pain, he developed urinary retention overnight. On day 2, Neurology was consulted, and the primary team obtained neuroaxis (brain, cervical, thoracic, and lumbar) magnetic resonance imaging (MRI) with and without contrast, which were grossly negative except for moderate cervical spinal canal stenosis without any abnormal cord signal and no acute intracranial abnormalities.

On the Neurology team's exam, based on the Medical Research Council (MRC) scale, he had 5/5 proximal bilateral upper extremity (UE) strength, 3/5 distal bilateral UE strength, 1/5 right lower extremity (RLE) strength throughout, and 2/5 left lower extremity (LLE) strength throughout. By this time, he also developed a sensory level around T8 with decreased sensation to light touch below this level. The patient complained about worsening weakness overnight. We discussed care with radiology and raised concerns for spinal cord ischemia in the setting of systemic hypotension on admission. We requested diffusion-weighted imaging (DWI) and apparent diffusion coefficient (ADC) sequences of the cervical and thoracic cord to rule out ischemia. Later MRIs showed ischemia from C5-6 through the thoracic spine to the conus, leading to a diagnosis of acute spinal cord infarction. The etiology of the stroke was thought to be systemic hypotension. As he had a small PE though asymptomatic with normal oxygen saturations, therapeutic anticoagulation (AC) was started along with aspirin. The rest of the stroke risk factors were managed, and it was recommended to avoid hypotension, maintain normal blood pressures around 120/80, and keep his LDL below 70 and his HbA1c below 6.5. His exam was stable as described above without any further decompensation, and he was eventually discharged with a plan to follow up in the stroke clinic.

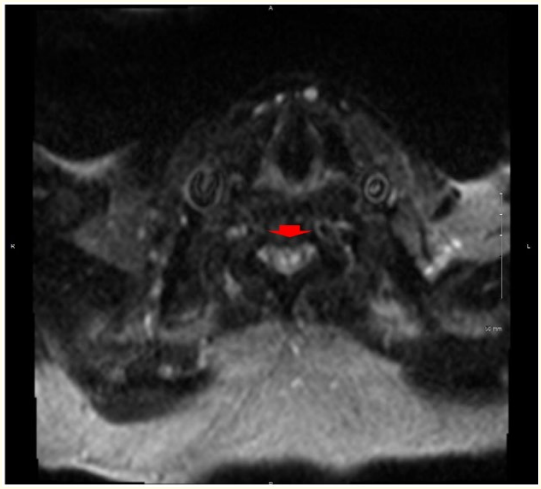

Figure 1: *Diffusion weighted imaging (DWI) sequence in axial section at cervical vertebra C6 level demonstrating hyperintensity in the spinal cord concerning cytotoxic injury from ischemia (Arrow).*

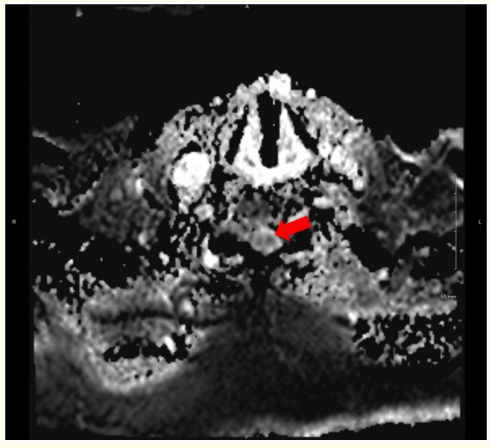

Figure 2: *Apparent diffusion coefficient (ADC) sequence in axial section at cervical vertebra C6 level demonstrating hypointensity, diffusion restriction in the spinal cord concerning for cytotoxic injury from ischemia (Arrow).*

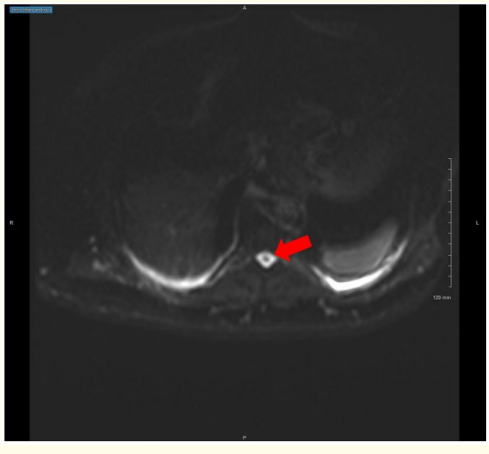

Figure 3: *Diffusion weighted imaging (DWI) sequence in axial section at cervical vertebra T8 level demonstrating hyperintensity in the spinal cord concerning cytotoxic injury from ischemia (Arrow).*

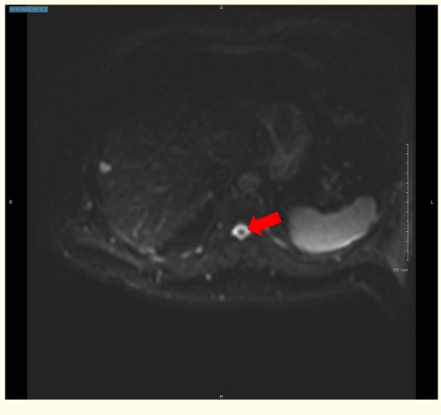

Figure 4: *Diffusion weighted imaging (DWI) sequence in axial section at cervical vertebra T10 level demonstrating hyperintensity in the spinal cord concerning cytotoxic injury from ischemia (Arrow).*

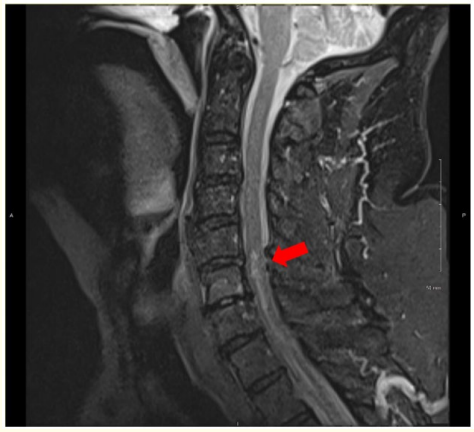

Figure 5: *T2 short-tau inversion recovery STIR sequence in sagittal section demonstrating hyperintensity in the center of the spinal cord from C5-C6 junction and below (Arrow).*

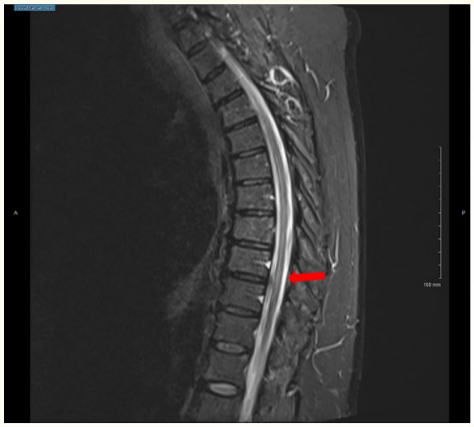

Figure 6: *T2 short-tau inversion recovery STIR sequence in sagittal section demonstrating hyperintensity in the center throughout thoracic spinal cord better seen at T8 & T9 level (Arrow).*

Discussion

Spinal cord ischemic (SCI) strokes are significantly rarer than cerebral ischemic strokes, with an incidence of approximately 1 - 2% [1]. Despite their low incidence, SCI strokes result in substantial morbidity and severely impact patient's quality of life, presenting symptoms ranging from minor weakness to acute paraplegia or quadriplegia, depending on the affected spinal cord level [1].

The pathophysiology of SCI strokes involves hypoperfusion due to various vascular etiologies, such as arterial occlusion or reduced blood flow from thrombosis, embolism, vasculitis, dissection, or atherosclerotic changes. Venous occlusions or systemic hypoperfusion can also contribute to the condition [2,3]. Additionally, spinal and vascular surgeries can be contributing factors to spinal strokes due to potential complications such as arterial dissection or other vascular injuries [4]. Patients typically exhibit symptoms of weakness and/or numbness in bilateral or unilateral extremities and the trunk, without cranial nerve involvement or facial abnormalities commonly seen in

cerebral strokes. Other symptoms can include neck or back pain at the level of spinal ischemia and autonomic dysfunction such as bladder, bowel, or sexual dysfunction [5,6]. Diagnosis is primarily achieved through spinal MRI, particularly using DWI and ADC sequences [7]. Treatment focuses on managing risk factors and utilizing antithrombotic or antiplatelet agents depending on the underlying cause of the ischemic stroke [2].

In our patient, who initially presented with an NIHSS score of 2 for the right lower extremity, a cerebral stroke was high on the differential diagnosis list. Appropriate imaging studies, including a CT head and CTA head and neck, were performed but were unremarkable. Cerebral ischemic stroke is commonly seen in patients experiencing acute episodes of hypotension, particularly those with a history of hypertension and extensive atherosclerotic vascular disease, like our patient. As the patient's symptoms progressed to include weakness in the bilateral upper and lower extremities and sensory abnormalities below the T8 level, the likelihood of a spinal ischemic stroke increased. Given the patient's risk factors and initial presentation with prolonged systemic hypotension, likely secondary to nitroglycerin intake, we were particularly concerned about hypoperfusion-induced spinal ischemic stroke.

Despite an initial negative MRI of the spine with and without contrast, the high clinical suspicion warranted further imaging. Subsequent MRI of the cervical and thoracic regions with DWI and ADC sequences revealed ischemia extending from C5-C6 to the conus, aligning with the neurological exam findings. Although the patient had incidental findings of pulmonary embolism, the extent of ischemia involving the cervical spine down to the conus suggested that systemic hypotension-induced hypoperfusion was the most likely cause of the SCI stroke in this case, a rare complication given the higher incidence of systemic hypotension and shock cases we encounter.

Conclusion

This case highlights the importance of thorough physical examination and clinical diagnosis. Equally important is educating patients about the serious adverse effects and common side effects of medications so they can promptly seek medical attention if they experience adverse signs and symptoms, as seen in our patient, rather than waiting for improvement.

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Volume 16 Issue 9 September 2024