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Case Report

A rare case of posterior circulation stroke with anterior spinal artery involvement

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ABSTRACT

A 36 year old gentlemen with diabetes mellitus and systemic hypertension with acute onset giddiness with weakness of both upper limbs, developed over a period of several hours with bilateral triceps weakness, wrist dorsiflexion and palmar flexion weakness with no sensory disturbance and cerebellar signs. MRI brain showed left cerebellar infarct, left lateral medullary infarct, non- visualization of left vertebral artery, T2 hyper-intensity of cervical cord from 4th cervical segment to 8th cervical segment, multiple areas are affected because of left vertebral artery occlusion.

Keywords: Anterior spinal artery, Posterior inferior cerebellar artery, Anterior spinal artery infarction

INTRODUCTION

Posterior circulation strokes constitute approximately 20% of ischemic strokes, primarily involving the vertebrobasilar system. These strokes typically present with vertigo, imbalance, unilateral limb weakness, slurred speech, headache, double vision, nausea, and vomiting.[1] Involvement of the anterior spinal artery in the context of posterior circulation stroke is uncommon, as the anterior spinal artery predominantly supplies anterior 2/3rd of the spinal cord, including corticospinal tracts.[4] We present a rare case of anterior spinal artery infarction secondary to left vertebral artery occlusion, which resulted in isolated motor weakness of the bilateral upper limbs.[6] This case highlights importance of detailed clinical and radiological evaluation in identifying atypical stroke presentations.

CASE REPORT

A 36-year-old male with a history of poorly controlled diabetes mellitus for six years and systemic hypertension presented with acute-onset giddiness. The giddiness was spontaneous and not triggered by positional changes, and was not associated with nausea, vomiting, diplopia, or dysarthria. On examination, his blood pressure was markedly elevated at 220/120 mmHg. Intravenous labetalol was administered, which controlled his blood pressure and relieved his giddiness.

Approximately thirty minutes after the resolution of giddiness, the patient noted weakness in his right hand, which began as a reduction in grip strength and progressed distally to proximally. Within fifteen minutes, he developed similar weakness in the left upper limb. Over the next four hours, proximal muscle strength in both upper limbs improved spontaneously, but the weakness persisted distally. Clinical examination revealed bilateral motor weakness characterized by reduced strength in elbow extension and wrist dorsiflexion and flexion. Reflexes were preserved, and there were no sensory deficits, cerebellar signs, or cranial nerve involvement.

The initial clinical suspicion was bilateral cortical stroke due to the motor weakness. However, the absence of sensory involvement or cortical signs made this diagnosis less likely. Imaging was performed to evaluate the cause of the patient's symptoms. MRI of the brain revealed a left

cerebellar infarct and a left lateral medullary infarct. MRA showed non-visualization of the left vertebral artery. As these findings did not fully explain the patient's motor weakness, further imaging was performed. MRI of the cervical spine revealed T2-weighted hyperintensity in the anterior spinal cord from C4 to C8, with axial diffusion-weighted imaging demonstrating the characteristic "snake-eye sign," consistent with anterior spinal artery infarction.

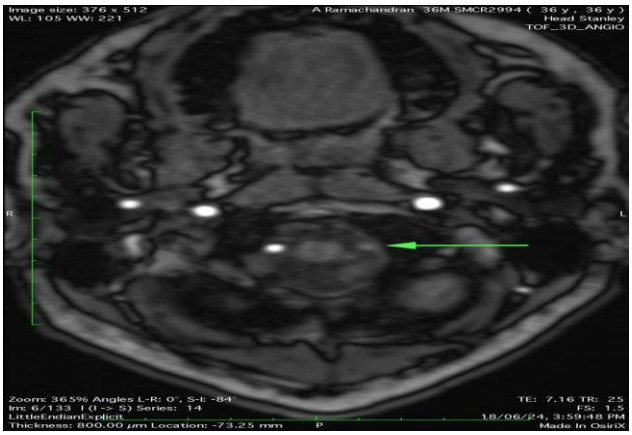

Figure 1: CT angiogram-left vertebral artery not seen.

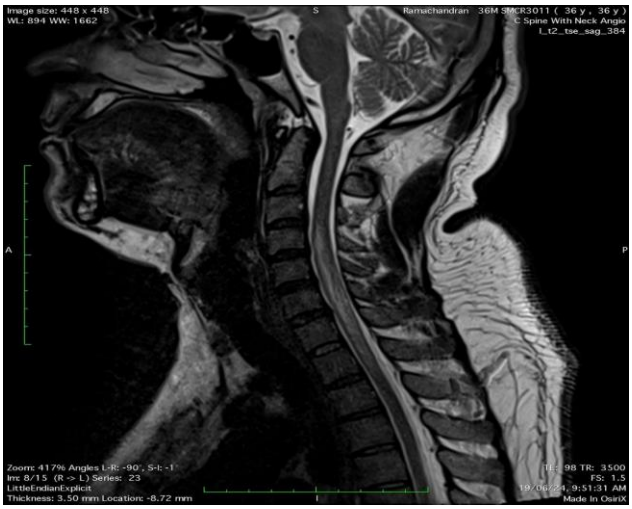

Figure 2: T2 hyperintensity in cervical cord, from C3 to C8.

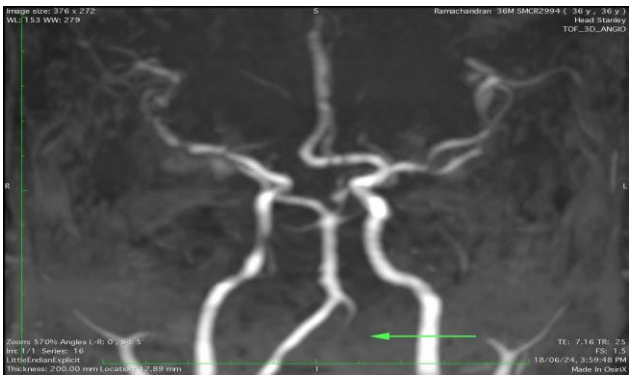

Figure 3: MRA-left vertebral artery is not visualised.

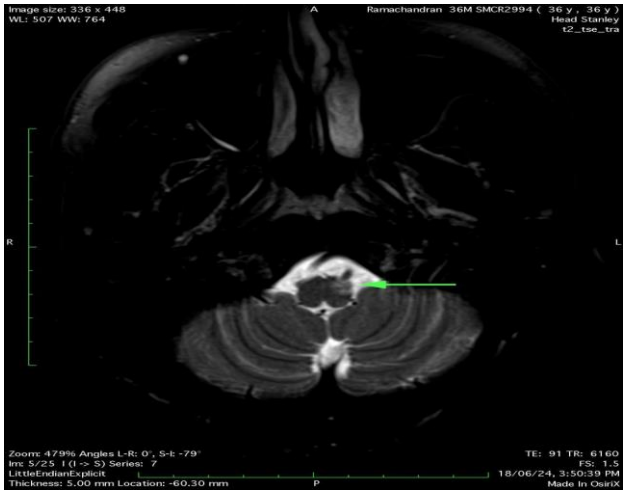

Figure 4: Left lateral medullary infarct.

The findings confirmed a diagnosis of anterior spinal artery syndrome secondary to occlusion of the left vertebral artery. The involvement of the anterior spinal artery in a posterior circulation stroke is rare, particularly with isolated bilateral motor weakness of the upper limbs.

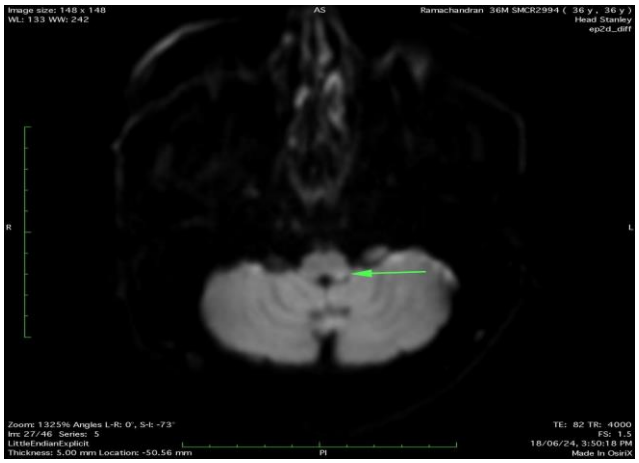

Figure 5: DWI-left lateral medullary infarct.

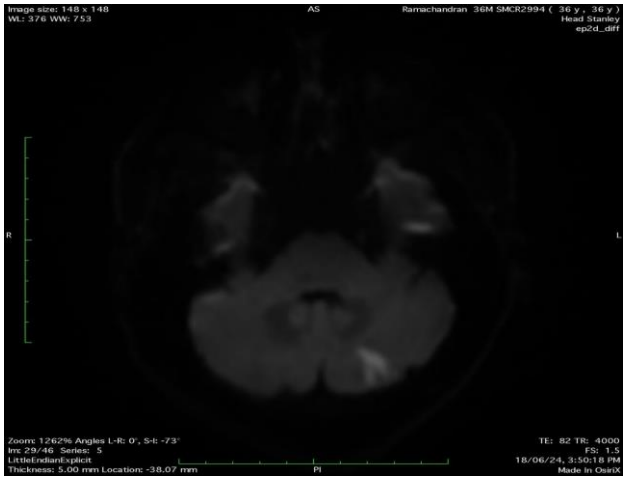

Figure 6: Left cerebellar infarct.

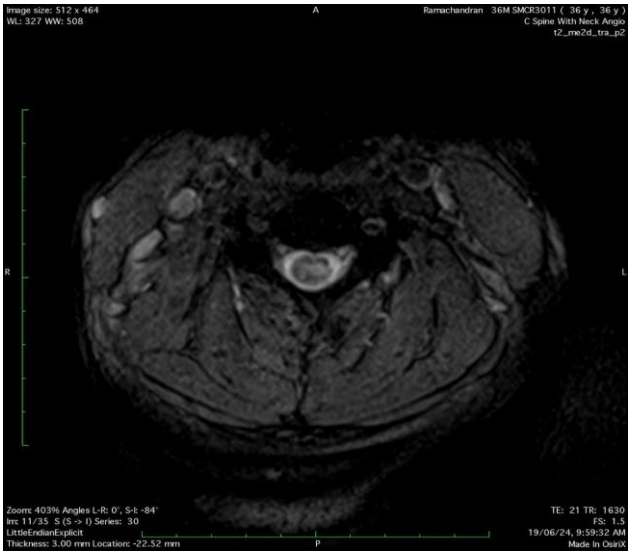

Figure 7: Axial section of cervical cord showing anterior cord involvement.

The patient was treated with dual antiplatelet therapy consisting of aspirin and clopidogrel to prevent further thromboembolic events. High-dose atorvastatin was initiated for lipid control and plaque stabilization. Blood pressure management included intravenous labetalol during the acute phase, followed by oral antihypertensive therapy with amlodipine and losartan. Insulin therapy was started to address long-standing hyperglycemia.

Over the course of his hospital stay, the patient demonstrated significant improvement in motor function. At discharge, he had mild residual weakness in wrist dorsiflexion bilaterally, with no new symptoms or complications. At the three-month follow-up, he had near-complete recovery of motor function and no recurrence of stroke symptoms.

DISCUSSION

Posterior circulation strokes are typically associated with vertigo, imbalance, unilateral limb weakness, slurred speech, headache, double vision, nausea, and vomiting.[1] Lateral medullary syndrome, or Wallenberg syndrome, is a common manifestation, presenting with sensory disturbances, dysphagia, and cranial nerve deficits rather than motor weakness.[2] The presence of isolated motor weakness in our patient suggested an alternative mechanism, prompting further investigation.

Infarction of the anterior spinal artery can result in motor deficits due to involvement of the corticospinal tracts, often accompanied by sensory disturbances if the spinothalamic tracts are also affected.[3] The anterior spinal artery predominantly arises from the vertebral arteries and receives collateral support from radicular arteries at various spinal levels. Occlusion of the vertebral artery disrupts this intricate vascular network, compromising the anterior spinal artery's blood flow[4]. This mechanism explains the coexistence of cerebellar infarction, lateral medullary syndrome, and anterior spinal artery infarction in our patient, a rare but significant pattern that highlights the complexity of posterior circulation strokes. The "snake-eye sign" observed on MRI, characterized by bilateral hyperintensity in the anterior horn regions of the spinal cord on axial diffusion-weighted imaging, is a distinctive feature of anterior spinal artery infarction. While traditionally associated with cervical spondylotic myelopathy or ischemic myelitis, its presence in this case reinforces the role of vascular compromise secondary to vertebral artery occlusion.[5,6] The selective involvement of upper limb motor function in our patient reflects the somatotopic organization of the corticospinal tracts, where fibers innervating the upper limbs are located medially within the cervical spinal cord.

The differential diagnosis of bilateral upper limb weakness includes brachial plexopathy, motor neuron disease, syringomyelia, and central cord syndrome.[7] In this case, the rapid onset, absence of sensory involvement, and characteristic MRI findings strongly supported the diagnosis of anterior spinal artery infarction. This case underscores the importance of a multidisciplinary approach, integrating clinical, radiological, and pathological findings to arrive at an accurate diagnosis.

Management strategies for posterior circulation strokes with anterior spinal artery involvement require an individualized approach. Timely reperfusion remains the cornerstone of acute ischemic stroke management, although it is less commonly achieved in anterior spinal artery infarction due to delayed diagnosis.[8] In our patient, dual antiplatelet therapy with aspirin and clopidogrel was pivotal in preventing further thromboembolic events. High-dose statin therapy not only stabilized atherosclerotic plaques but also exerted pleiotropic effects, reducing inflammation and improving endothelial function.[9] Additionally, strict blood pressure and glycemic control were essential to minimize the risk of recurrent strokes. Rehabilitation plays a crucial role in recovery, particularly in atypical stroke syndromes involving the spinal cord. Early initiation of physiotherapy has been shown to enhance neuroplasticity and improve motor outcomes in patients with spinal ischemia.[10] Our patient's significant improvement by the three-month follow-up demonstrates the potential for favorable outcomes with comprehensive management.

This case highlights the need for heightened clinical suspicion and extensive imaging, including cervical spine MRI, in atypical presentations of posterior circulation strokes. Recognizing and addressing the full spectrum of vascular compromise in such cases can improve diagnostic accuracy and optimize patient outcomes.

CONCLUSION

PICA vessel infarct causes lateral medullary syndrome, motor weakness in pica infarct is uncommon unless the

infarct extends to the medial medulla. Distal symmetrical or asymmetrical distal weakness of upper limbs when present in temporal relation with a pica stroke, anterior spinal artery involvement must be excluded as it is also a branch of vertebral artery. Other causes of distal hand weakness must be evaluated after ruling out anterior spinal artery involvement in cases with upper limb motor weakness.

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