

Isolated Sensory-motor leg monoparesis in the setting of posterior circulation stroke: A case report

Varghese S, Doctor SP

ABSTRACT

Isolated leg monoparesis due to ischemic stroke is rare, and the lesions have been traditionally localized to the anterior cerebral artery territory, corona radiata or the posterior limb of internal capsule. Any lesion suitably placed along the course of the corticospinal tract can provoke monoparesis, due to its somatotopic organization of nerve fibres. A posterior circulation stroke usually manifests as transient ischemic attacks of headache/dizziness/nausea or a wide-range of symptoms and signs. Very rarely one has ever come across a case of isolated sensory-motor leg monoparesis in the setting of a posterior circulation stroke.

Keywords: isolated leg monoparesis, posterior circulation, stroke

INTRODUCTION

Posterior circulation includes mainly the paired vertebral arteries which form the basilar artery which finally terminates into the paired posterior cerebral arteries and also their branches.¹ The posterior circulation is particularly vulnerable to atherothrombosis like the systemic circulation, but it also has several collateral connections which can open up to connect it to middle cerebral artery and internal and external carotid arteries, thus giving it a wide range of clinical features on occlusion of its different branches.^{2,3} Isolated leg monoparesis has been the subject of discussion in few discrete studies till date, and very few causes have been localized. These have been mainly restricted to small infarcts or hemorrhages in the top of the precentral gyrus or in the corona radiata or uncommonly ascribed to the lateral medullary syndrome. Interestingly, the posterior limb of internal capsule, which is basically supplied by the middle cerebral artery and the internal carotid artery, carries the corticospinal tract fibres as well as the sensory fibres (medial lemniscus and the anterolateral system). Less commonly, the thalamoperforator branches arising from the basilar

artery (posterior circulation) supply the inferior half of the posterior limb of internal capsule.^{4,5}

This rare case depicts one such incidence of isolated sensory-motor leg monoparesis due to complete basilar artery occlusion.

CASE REPORT

A 46 year old man was admitted with sudden onset weakness in the right foot since evening on the day before. He had no other complaints other than inability to walk with his right foot and decreased sensibility at that part. No history of low back pain or fever or any bowel/bladder abnormalities or any shooting pain/tingling sensation in the affected or other extremities. Past history revealed that he was a regular smoker and alcoholic since last many years. Family history was insignificant. Detailed clinical evaluation revealed 1/5 power in right ankle with inability to dorsiflex or plantarflex the foot, along with Babinski's sign positive and decreased pain/temperature/crude touch on the dorsum of foot and mildly on the sole. Vibration and joint position sense was preserved on the right foot. Rest of the neurological examination was absolutely normal. Complete hemogram, renal function,

liver function, thyroid function, chest X-ray, ultrasonography abdomen/pelvis, random blood sugar and lipid levels tests were normal. Vitamin B12, prothrombin time, VDRL and HIV antibody tests were within normal limits. Homocysteine was 32 micromol/litre (mildly high). MRI spine and brain were notably unremarkable while MR angiography of the brain (Fig.1.) showed diffuse narrowing of both vertebral arteries with complete basilar artery block.



Fig.1

We treated the patient with Enoxaparin (0.6 mg subcutaneous, twice a day for 5 days) and oral aspirin 150 mg/day with atorvastatin 40 mg/day oral. We also counselled him for alcohol withdrawal and prescribed oral multivitamin and thiamine supplements. By the 6th day, as he recovered significantly, he was discharged with 4+/5 power and almost normal sensation in the right foot.

DISCUSSION

Complete Basilar artery occlusion is a dreaded neuromedical condition with mortality in some studies of upto 90%.³ But this was a rare

occurrence of pure right leg sensory motor monoparesis and affirms the traditional finding that the clinical manifestations of basilar artery occlusion vary according to the site and nature of vascular compromise and the location of neural ischemia.⁶ The cause of vertebra-basilar thrombosis in this patient could be associated to the vascular effects of smoking and alcoholism as well as mild hyperhomocysteinemia. The sensory and motor affection in the right foot could well be explained by a minor occlusion in the thalamoperforator branches of the basilar artery, which may have been supplying the posterior limb of internal capsule. Another hypothesis is an invisible small infarct in the lower lateral medulla oblongata, in compliance with the somatotropic arrangement of the corticospinal and sensory fibres in the central nervous system. The success of our treatment with enoxaparin confirms the causation of this rare entity.

CONCLUSION

Very few cases have been documented worldwide with isolated leg sensory-motor monoparesis, and hardly any of these has been caused by basilar artery occlusion. This occurrence emphasizes the fascination which neurology imparts to clinical examination even today.

AUTHOR NOTE

Sajit Varghese, 3rd Year P.G. (Corresponding Author), Email: dr_sajit2@yahoo.com
Doctor Shachish P, 1st Year P.G.

Department of Medicine, Smt. N.H. L Municipal Medical College, Ahmedabad, Gujarat

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