CASE REPORT

DOI: 10.15713/ins.bhj.213

Floating Thrombus – Basilar Artery Occlusion

ABSTRACT

Aim: The treatment of Basilar artery occlusion (BAO) with low NIHSS score requires to be individualized to each patient

Background: Basilar artery occlusion represents a small proportion of strokes worldwide but can have catastrophic presentations. Recent trials for BAO have studied the benefits of endovascular treatment (EVT) over intravenous thrombolysis (IVT) and have concluded that EVT has better mortality and morbidity outcomes. However, the management of BAO with lower NIHSS scores may become risky as the complications of the said treatment may outweigh the benefits.

Case description: We present a 54-year-old lady who presented with dizziness and hemiplegia which resolved within 1 hour. She had hypertension, mild left facial weakness and NIHSS score of 1 on presentation. MRI Brain revealed an acute focal left lateral medullary infarct, and angiogram showed a basilar artery thrombosis with distal basilar artery flow. She was started on dual antiplatelet therapy, low molecular weight heparin and statin. Cerebral DSA revealed a floating thrombus within the basilar artery and left vertebral artery V4 segment stenosis with normal flow in the distal basilar artery. As the patient had mild symptoms, she was discharged on oral anticoagulant and single antiplatelet therapy. She underwent stenting of the Vertebral artery V4 segment, 2 months later as she had recurrent posterior circulation transient ischemic attacks.

Conclusion: All basilar artery occlusions need not require immediate endovascular thrombectomy and may be managed conservatively initially with anti-coagulation and antiplatelet therapy.

Key words: Basilar artery occlusion, thrombus, Thrombectomy

INTRODUCTION

Basilar artery occlusion is the complete or partial blockage of the basilar artery in the posterior circulation. The clinical symptoms can vary from dizziness, vertigo to dysarthria, facial weakness, oculomotor abnormalities or severe motor deficits and "locked in syndrome" in severe cases. Treatment options offered are thrombolysis or mechanical thrombectomy. Recent studies, ATTENTION trial and BAOCHE trial in China have shown better functional outcomes in patients with endovascular thrombectomy in patients with a NIHSS score>10. We want to report a case of basilar artery occlusion and discuss the management.

CASE PRESENTATION

A 54-year-old lady with hypertension presented with a brief episode of dizziness with vomiting which was followed by a transient weakness of the right upper and lower limb. There was no history of seizure, loss of consciousness, headache, vomiting or vision or speech abnormalities. On examination, the patient was alert and conscious with a GCS of 15. She was normotensive and had a regular pulse rhythm. Cranial nerve examination revealed a left mild facial weakness. Tone and power were normal in all 4 limbs. There were no sensory deficits or cerebellar signs. The NIHSS score was 1.

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In view of the episodic weakness and risk factor of hypertension, a vascular cause likely a TIA or a stuttering lacunar infarct was thought of. She was admitted in the ICU for the same.

Diagnostic evaluation

MRI brain stroke protocol showed diffusion restriction in the left lateral medulla with an ADC drop out and no evidence of hemorrhage suggestive of a small focal left lateral medullary infarct and a hyperintense signal in the basilar artery suggestive of a basilar artery thrombus (Figure 1 and 2). The MRA revealed narrowing of the left vertebral artery and decrease in flow signal across the basilar artery from the origin to the mid basilar artery with normal flow across the distal basilar artery (Figure 3).

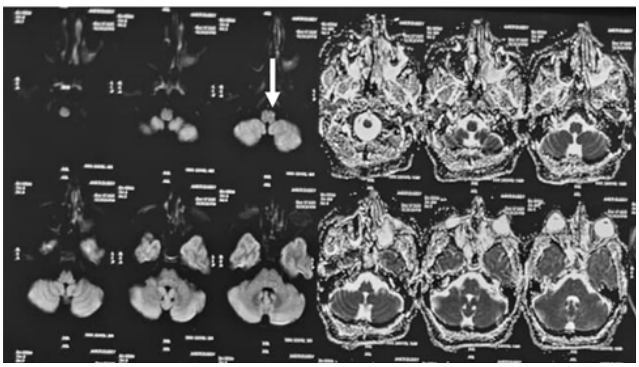

Figure 1: DWI and ADC sequence of brain indicating a focal infarct in the left lateral medulla (solid arrow)

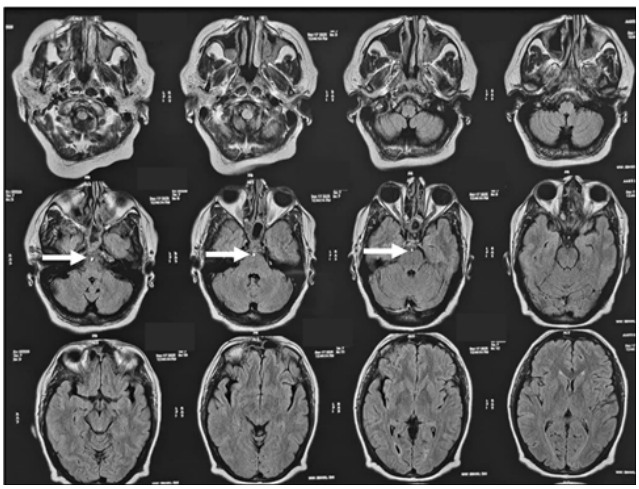

Figure 2: T2 FLAIR sequence reveals hyperintense signal in the basilar artery suggestive of an acute thrombus (solid arrow)

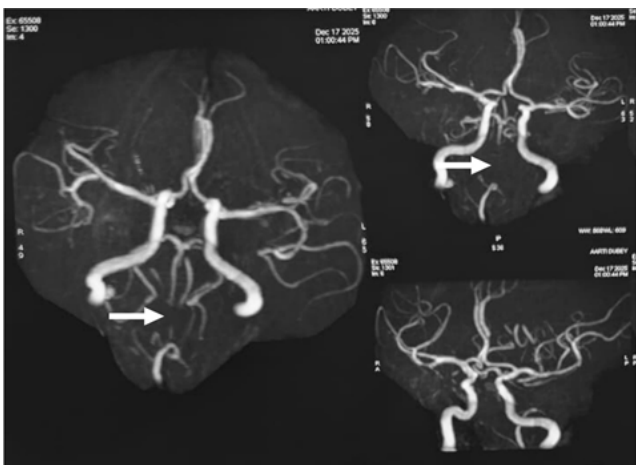

Figure 3: MRA reveals narrowing of the left vertebral artery and decrease in flow signal across the basilar artery from the origin to the mid basilar artery with normal flow across the distal basilar artery (solid arrow)

In view of the presence of thrombus, a cerebral DSA was done which revealed a focal stenosis in the V4 segment of the left vertebral artery with a thrombus within the basilar artery and normal flow in the distal basilar artery and posterior cerebral arteries (Figure 4).

Further risk factor evaluation was done. 2D ECHO showed an ejection fraction of 55%, no regional wall motion abnormalities and no clots. ECG showed a normal sinus rhythm. Glycosylated hemoglobin was 5.6%. Serum Homocysteine was 6 micromol/L. Lipid profile showed LDL 70 mg/dL, HDL 40 mg/dL and total cholesterol of 150 mg/dL.

Platelet sensitivity test was done which showed high inhibition to both aspirin and clopidogrel.

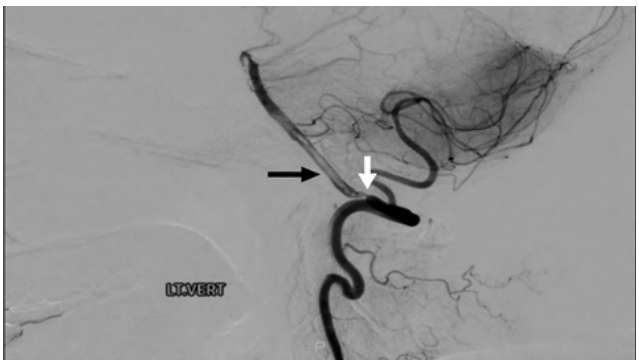

Figure 4: Cerebral angiogram of the left vertebral artery reveals a floating thrombus within the proximal basilar artery (black solid arrow) and a stenosis of the V4 segment of left vertebral artery (white solid arrow).

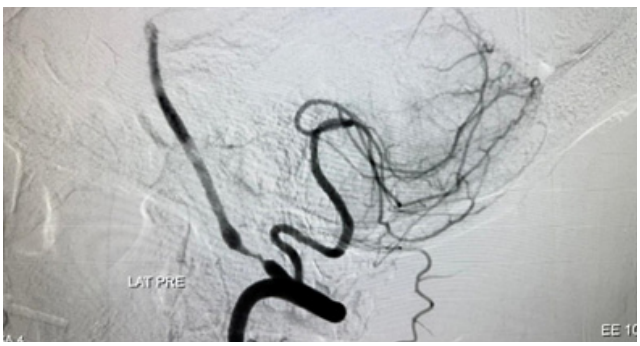

Figure 5: Cerebral angiogram showing a stenosis of the V4 segment of the left vertebral artery.

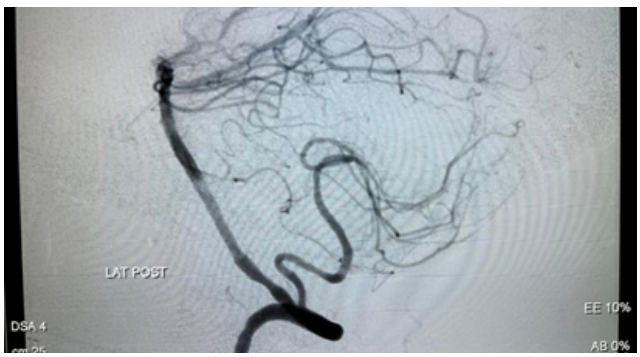

Figure 6: Cerebral angiogram of the left vertebral artery post stent placement.

TREATMENT

As the patient had an acute infarct with a vessel thrombosis, she was treated with dual antiplatelets, low molecular weight heparin and statin for 5 days. In view of a low NIHSS score she was treated conservatively. She was discharged on oral anticoagulant (rivaroxaban), clopidogrel and atorvastatin. She was offered endovascular thrombectomy in the event of a new neurological deficit or worsening of NIHSS score.

On follow up, she developed new symptoms of recurrent vertiginous episodes associated with vomiting after 2 months. There were no neurological deficits. Her repeat MR Angiogram Brain revealed a persistence of the left vertebral artery V4 segment stenosis and resolution of the basilar artery thrombus. She underwent a stent placement in the left vertebral artery. There were no procedure related complications.

DISCUSSION

Basilar artery thrombosis is a life-threatening form of posterior circulation stroke, classically associated with severe neurological deficits and high mortality. However, a subset of patients presents with mild symptoms, reflected by a National Institutes of Health Stroke Scale (NIHSS) score of less than 6.[1] These patients represent a distinct clinical challenge, as the initially benign presentation may belie the potential for rapid neurological deterioration due to brainstem ischemia.

Patients with low NIHSS scores often present with subtle posterior circulation symptoms such as vertigo, diplopia, dysarthria, mild ataxia, or visual disturbances.[2] Despite low clinical severity, vascular imaging frequently demonstrates true large-vessel occlusion of the basilar artery, underscoring the importance of early CT angiography or MR angiography in suspected posterior circulation stroke.[3] Imaging confirmation is essential to guide therapeutic decisions and risk stratification.

Intravenous thrombolysis (IVT) remains a key component of acute medical management in eligible patients presenting within 4.5 hours of symptom onset. Posterior circulation strokes are particularly prone to underestimation by the NIHSS, and reliance on clinical severity alone may fail to capture the risk of subsequent neurological decline.[4] Nevertheless, IVT achieves lower recanalization rates in basilar artery occlusion compared with anterior circulation strokes, especially in the presence of large clot burden or underlying intracranial atherosclerosis.[5]

In patients presenting outside the thrombolysis window or with contraindications to IVT, antithrombotic therapy forms the mainstay of medical management. Aspirin is routinely initiated once intracranial hemorrhage is excluded. In cases of minor non-cardioembolic stroke, short-term dual antiplatelet therapy (DAPT) with aspirin and clopidogrel may be considered to reduce early stroke recurrence, extrapolating evidence from the CHANCE and POINT trials.[6,7] However, data specifically addressing DAPT in basilar artery thrombosis remain limited, and treatment decisions should be individualized.

When basilar artery thrombosis is attributed to a cardioembolic source such as atrial fibrillation, long-term anticoagulation is indicated for secondary prevention. The timing of anticoagulation initiation depends on infarct size and hemorrhagic risk, typically guided by follow-up neuroimaging.[8] In cases related to vertebrobasilar atherosclerotic disease, aggressive medical therapy, including high-intensity statins, blood pressure control, glycemic management, and smoking cessation, is essential to prevent progression and recurrence.[9]

Close neurological monitoring is a critical component of conservative management. Patients with initially mild deficits remain at risk for sudden deterioration due to thrombus propagation or failure of collateral circulation.[10] Admission to a dedicated stroke unit with frequent neurological assessments allows early detection of worsening and timely escalation of care, including consideration of rescue endovascular therapy if indicated, as in the case of our patient.

In conclusion, medical management of basilar artery thrombosis in patients with NIHSS < 6 emphasizes early diagnosis, appropriate reperfusion strategies, tailored antithrombotic therapy, and vigilant monitoring. Although symptoms may be mild at presentation, the underlying pathology carries substantial risk, and individualized, imaging-guided management is essential to prevent catastrophic posterior circulation stroke.

Conclusion: Basilar artery occlusion does not necessitate immediate endovascular thrombectomy as a subset of patients with low NIHSS scores may be managed conservatively and be considered for thrombectomy on escalation of symptoms.

How to cite this article: Kumar Samantha C, Phulsunge Sunetra D, Singh R, Pardasani V. Floating Thrombus – Basilar Artery Occlusion.Bombay Hosp J 2026; 68(1):43-46.

Conflicts of Interest: None. **Source of Support:** None.

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