



Case Report

FLUCTUATING NEUROLOGICAL DEFICITS IN SEVERE PROXIMAL BASILAR ARTERY STENOSIS: CONSERVATIVE MANAGEMENT OR ENDOVASCULAR ANGIOPLASTY?

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ABSTRACT

Introduction : Symptomatic basilar artery (BA) stenosis is a high-risk cause of posterior circulation ischemia with potential for recurrent events despite optimal medical therapy. The decision between conservative treatment and endovascular angioplasty remains challenging, particularly in patients with fluctuating symptoms.

Case Summary : A 62-year-old man with hypertension, poorly controlled type 2 diabetes mellitus, and prior ischemic stroke presented with a two-month history of oscillopsia, intermittent headache, and transient left-sided weakness. Symptoms progressed to persistent imbalance, diplopia, and gait instability. Brain imaging demonstrated acute infarctions in the left thalamus and right anterior pons, multiple chronic posterior circulation infarcts, occlusion of the right vertebral artery, and severe proximal BA stenosis. Digital subtraction angiography (DSA) confirmed approximately 90% symptomatic proximal BA stenosis with reduced flow to the right anterior inferior cerebellar artery. Despite risk factor control, the patient continued to experience symptoms that significantly interfered with daily activities. Given the high procedural risk, conservative management was chosen with close monitoring. A three-month observation period with repeat DSA was planned to reassess the need for angioplasty.

Discussion : This case highlights the unstable course of severe BA stenosis despite medical therapy. Although angioplasty may improve perfusion, BA interventions carry substantial risk due to brainstem perforators, with perforator infarction from “snow-plow” effect during angioplasty being a major concern, particularly in proximal lesions.

Conclusion : Severe symptomatic BA stenosis presents a challenging therapeutic dilemma. Short-term observation with planned reassessment may be appropriate, but progressive symptoms require close follow-up and early consideration of endovascular treatment.

Keywords: Basilar artery stenosis; conservative management; endovascular angioplasty

INTRODUCTION

Symptomatic intracranial atherosclerotic stenosis (sICAS) is a major cause of ischemic stroke and is associated with a high risk of recurrent cerebrovascular events, particularly in patients with severe

stenosis.¹ Basilar artery (BA) stenosis is especially concerning because compromised posterior circulation perfusion may result in fluctuating neurological deficits, recurrent transient ischemic attacks, or progressive ischemic stroke

involving the brainstem and cerebellum.² Current evidence supports aggressive medical therapy as the first-line treatment for symptomatic ICAS, including antithrombotic therapy and intensive vascular risk factor control. However, some patients continue to deteriorate despite optimal conservative management, raising consideration for endovascular intervention. Although angioplasty with or without stenting may improve cerebral perfusion in selected cases, basilar artery procedures remain technically challenging and carry significant periprocedural risks.³⁻⁶ We report a case of severe proximal basilar artery stenosis presenting with fluctuating neurological deficits, highlighting the clinical dilemma between continued conservative management and endovascular angioplasty in medically refractory posterior circulation ischemia.

CASE REPORT

A 62-year-old man with a history of hypertension, poorly controlled type 2 diabetes mellitus (DM), deep vein thrombosis (DVT), and prior non-hemorrhagic ischemic stroke in 2016 with residual left-sided weakness presented with a

two-month history of oscillopsia, intermittent headache, and transient left-sided weakness. His symptoms progressively worsened into persistent dizziness, imbalance, diplopia, blurred vision, and gait instability, significantly impairing daily activities and occupational performance as a notary due to difficulty reading for prolonged periods.

Initial neurological examination demonstrated mild left central facial nerve paresis without nystagmus. Laboratory evaluation showed poorly controlled diabetes mellitus with HbA1c levels ranging from 9.8-10.5%. The patient had been receiving rivaroxaban 15 mg once daily, atorvastatin 20 mg once daily, candesartan 8 mg once daily, insulin glargine, flunarizine 5 mg once daily, and citicoline 500 mg twice daily.

Initial brain MRI demonstrated multiple acute lacunar infarctions involving the splenium of the corpus callosum and bilateral occipital lobes, accompanied by chronic infarction in the right cerebellar hemisphere and chronic white matter ischemic changes. MRA revealed stenosis of the mid-basilar

artery, right vertebral artery, and left posterior cerebral artery, consistent with intracranial atherosclerotic disease. Despite continuation of conservative therapy, the patient continued to experience recurrent vertigo-like symptoms, headache, and intermittent diplopia over the following months.

Subsequent hospitalization was prompted by worsening dizziness, diplopia, gait instability, and persistent headache. Repeat MRI revealed new acute infarctions in the left thalamus and right anterior pons, with additional chronic posterior circulation infarcts involving the cerebellum, pons, and right cerebral peduncle. Follow-up MRA demonstrated distal occlusion of the right vertebral artery (V4 segment), severe subtotal basilar artery stenosis, and bilateral posterior cerebral artery stenotic-occlusive lesions. Cerebral digital subtraction angiography (DSA) confirmed approximately 90% symptomatic

proximal basilar artery stenosis with reduced flow to the right anterior inferior cerebellar artery (AICA). The patient was subsequently managed with aggressive medical therapy, with the escalation of rivaroxaban to 20 mg once daily, and the continuation of statin therapy, antihypertensive treatment, glycemic control, and supportive rehabilitation. Despite treatment, the patient continued to experience persistent diplopia, oscillopsia, gait instability, and recurrent vertebrobasilar insufficiency symptoms. Given the substantial procedural risk, conservative management with aggressive medical therapy and close clinical monitoring was continued. A three-month observation period with repeat DSA was planned to reassess disease progression and determine the potential need for delayed endovascular angioplasty if symptoms worsened despite optimized medical treatment.

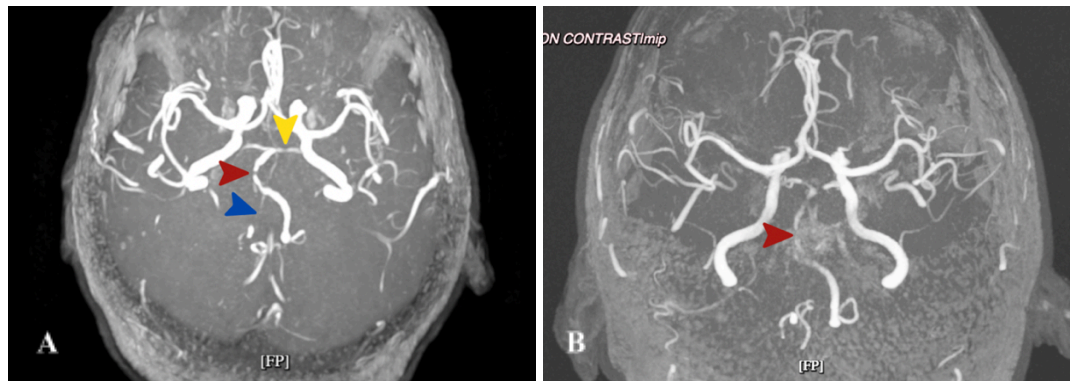


Figure 1. Serial magnetic resonance angiography (MRA) findings demonstrating progression of posterior circulation intracranial atherosclerotic disease. [A. Initial MRA showing stenosis of the basilar artery (red arrowhead), left posterior cerebral artery/P1 segment stenosis (yellow arrowhead), and right vertebral artery stenosis (blue arrowhead). B. Follow-up MRA obtained during hospitalization demonstrating progression to severe subtotal basilar artery stenosis (red arrowhead) with progression of vertebral artery disease to distal occlusion].

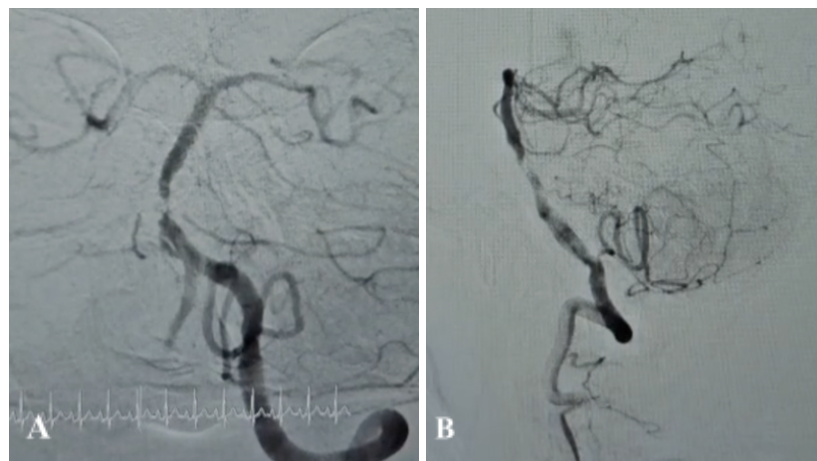


Figure 2. Cerebral digital subtraction angiography (DSA) findings demonstrating approximately 90% symptomatic proximal basilar artery stenosis with reduced flow to the right anterior inferior cerebellar artery. [A. AP view. B. Lateral view.].

DISCUSSION

Severe BA stenosis is associated with a high risk of recurrent vertebrobasilar ischemia because of limited collateral circulation and the dense concentration of pontine perforating arteries.² In the present case, the patient experienced progressive posterior circulation ischemic events manifested by oscillopsia, diplopia, dizziness, gait

instability, and recurrent infarctions involving the occipital lobes, thalamus, pons, and cerebellum. Several vascular risk factors likely contributed to disease progression, including hypertension, poorly controlled DM, and prior ischemic stroke. Persistent hyperglycemia during follow-up may have accelerated endothelial dysfunction, plaque instability, and recurrent

ischemic injury, contributing to ongoing cerebrovascular progression despite treatment.⁷

Aggressive Medical Therapy as First-Line Management

Current evidence supports aggressive medical therapy as the standard first-line treatment for sICAS. The Stenting and Aggressive Medical Management for Preventing Recurrent Stroke in Intracranial Stenosis (SAMMPRIS) trial demonstrated that aggressive medical management was superior to percutaneous transluminal angioplasty and stenting (PTAS), primarily because peri-procedural stroke rates in the stenting arm were substantially higher than anticipated. In SAMMPRIS, the 30-day rate of stroke or death in the PTAS group reached 14.7%, compared with 5.8% in the medical-management group. Many peri-procedural events were attributed to perforator infarctions, while restenosis was reported in approximately 25–30% of patients within six months after PTAS. The inclusion of patients with severe (70–99%) symptomatic stenosis and recent ischemic events likely reflected unstable plaque morphology and increased susceptibility to distal embolization

during intervention.³

Similarly, the Vitesse Intracranial Stent Study for Ischemic Stroke Therapy (VISSIT) trial reported significantly higher rates of stroke and transient ischemic attack (TIA) in the stenting group compared with medical therapy alone at both 30 days and one year. Intracranial hemorrhage within 30 days occurred in 5.8% of patients in the stenting group, whereas no cases were observed in the medical therapy group. In addition, worsening functional outcomes, measured by modified Rankin Scale (mRS), occurred more frequently in the stenting arm (24.1% vs. 11.3%).⁴ Subsequent evidence from the China Angioplasty and Stenting for Symptomatic Intracranial Severe Stenosis (CASSISS) trial further reinforced aggressive medical therapy as the preferred treatment strategy, demonstrating no significant benefit of stenting even when procedures were delayed and performed by experienced operators.⁵

Aggressive medical therapy in these landmark studies consisted of short-term dual antiplatelet therapy (DAPT), intensive vascular

risk-factor modification, and lifestyle intervention. Standard management generally includes aspirin 81-325 mg/day combined with clopidogrel 75 mg/day for the first 90 days, followed by long-term single antiplatelet therapy (SAPT). Intensive lipid lowering with high-intensity statin therapy is recommended to achieve low-density lipoprotein (LDL) levels <70 mg/dL. Blood pressure control should target systolic blood pressure <140 mmHg, or <130 mmHg in patients with DM. Glycemic optimization is also essential, with recommended HbA1c targets <7%. Additional non-pharmacological measures include smoking cessation, weight reduction, regular physical activity, and comprehensive lifestyle modification. Collectively, these measures emphasize the importance of strict secondary stroke prevention in patients with sICAS. Furthermore, transcranial Doppler studies have suggested that DAPT may reduce distal microembolic signals compared with aspirin monotherapy, potentially lowering the risk of recurrent ischemic events.³⁻⁵

Compared with guideline-based aggressive medical management, the present patient was maintained on

long-term rivaroxaban rather than protocol-based DAPT because of a prior history of DVT. This represents a therapeutic dilemma frequently encountered in clinical practice when concomitant thromboembolic disorders coexist. Despite anticoagulation therapy, the patient continued to develop recurrent posterior circulation infarctions and progressive vertebrobasilar symptoms, suggesting that anticoagulation alone may have been insufficient to suppress platelet-mediated thromboembolism associated with severe intracranial atherosclerosis. This observation is consistent with current understanding that thrombotic events in sICAS are predominantly platelet-driven rather than venous thrombin-mediated processes.

Earlier evidence from the Warfarin-Aspirin Symptomatic Intracranial Disease (WASID) trial demonstrated that warfarin was not superior to aspirin for asymptomatic intracranial stenosis and was associated with higher mortality and major hemorrhagic complications.⁸ Consequently, subsequent landmark trials adopted antiplatelet-based aggressive medical therapy rather than anticoagulation strategies.³⁻⁵

Recent observational studies have explored the potential role of direct oral anticoagulants (DOACs), including rivaroxaban, particularly in combination with antiplatelet agents as dual pathway inhibition (DPI). Although preliminary findings suggest a possible reduction in recurrent ischemic events, current evidence remains limited and insufficient to replace guideline-based antiplatelet therapy.^{8,9} Furthermore, combining anticoagulants with antiplatelet agents substantially increases bleeding risk, particularly intracranial hemorrhage, which is especially concerning in elderly patients with extensive cerebrovascular disease.¹⁰

Consideration of Endovascular Angioplasty

The role of endovascular treatment in refractory symptomatic ICAS remains controversial, particularly in basilar artery lesions. One major concern during BA intervention is the “snow-plow” effect, in which balloon inflation or stent deployment displaces atherosclerotic plaque into perforator artery ostia, potentially causing devastating pontine infarction. This mechanism is especially relevant in proximal BA

stenosis because of the high density of pontine perforators.¹¹⁻¹³

More recent evidence suggests that submaximal balloon angioplasty without routine stenting may reduce perforator complications compared with conventional PTAS. The Balloon Angioplasty for Symptomatic Intracranial Artery Stenosis (BASIS) trial was among the first studies to suggest a possible benefit of balloon angioplasty over aggressive medical management in selected refractory symptomatic ICAS cases. Proposed advantages included easier device navigation, shorter procedural duration, improved antegrade flow, and potentially lower risk of the snow-plow effect compared with stent-based techniques. However, arterial dissection remained an important complication, occurring in approximately 14.5% of patients and frequently requiring rescue stenting.¹⁴

Procedural timing also appears critical in determining safety outcomes. Data from Wingspan Stent System Post Market Surveillance (WEAVE) studies and subsequent analyses suggest that intracranial intervention should

ideally be delayed beyond the acute stroke phase because early intervention may increase the risk of plaque disruption, distal embolization, reperfusion injury, and hemorrhagic transformation.¹⁵ Delayed intervention performed several weeks after the most recent ischemic event appears to improve procedural safety.³⁻⁵

In the present case, the patient fulfilled several criteria supporting consideration of angioplasty, including severe symptomatic stenosis (>70%), recurrent posterior circulation ischemic events despite treatment, and progressive vertebrobasilar symptoms.¹⁶ However, the lesion location within the proximal BA, recurrent perforator-territory infarctions, and uncertainty regarding the optimal antithrombotic regimen due to chronic rivaroxaban use substantially increased procedural complexity and risk. Consequently, conservative management with intensive vascular risk-factor control and close radiological surveillance was selected, while delayed

angioplasty remained a potential option should neurological deterioration continue despite optimized medical therapy.

CONCLUSION

Severe symptomatic BA stenosis remains a challenging condition because of its high risk of recurrent vertebrobasilar ischemia and the substantial procedural risk. Although aggressive medical therapy remains the standard first-line treatment, recurrent ischemic events may still occur despite optimal conservative management, particularly in patients requiring chronic anticoagulation. Therefore, short-term observation with intensive risk-factor control and close radiological follow-up may be appropriate, while progressive symptoms should prompt early consideration of endovascular treatment in selected patients. Further studies are needed to clarify the optimal role of anticoagulation in sICAS.

ACKNOWLEDGEMENT

The authors declared no acknowledgement

REFERENCE

1. Turan TN, Zaidat OO, Gronseth GS, et al. Stroke Prevention in Symptomatic Large Artery Intracranial Atherosclerosis Practice Advisory: Report of the AAN Guideline Subcommittee. *Neurology*. 2022;98(12):486-498. doi: 10.1212/WNL.0000000000200030
2. Ausman JI, Liebeskind DS, Gonzalez N, et al. A Review of The Diagnosis and Management of Vertebral Basilar (Posterior) Circulation Disease. *Surg Neurol Int*. 2018;9:106. doi: 10.4103/sni.sni_373_17
3. Chimowitz MI, Lynn MJ, Derdeyn CP, et al. Stenting versus Aggressive Medical Therapy for Intracranial Arterial Stenosis. *N Engl J Med*. 2011;365(11):993-1003. doi: 10.1056/NEJMoa1105335
4. Zaidat OO, Fitzsimmons BF, Woodward BK, et al. Effect of a Balloon-Expandable Intracranial Stent vs Medical Therapy on Risk of Stroke in Patients with Symptomatic Intracranial Stenosis: the VISSIT Randomized Clinical Trial. *JAMA*. 2015;313(12):1240-8. doi: 10.1001/jama.2015.1693
5. Gao P, Wang T, Wang DM, et al. Effect of Stenting Plus Medical Therapy vs Medical Therapy Alone on Risk of Stroke and Death in Patients with Symptomatic Intracranial Stenosis: The CASSISS Randomized Clinical Trial. *JAMA*. 2022;328(6):534-42. doi: 10.1001/jama.2022.12000
6. Kim JH, Cho KC, Kim TM, et al. Endovascular Treatment of Symptomatic Basilar Artery Stenosis. *Neurointervention*. 2023;18(3):166-71. doi: 10.5469/neuroint.2023.00437
7. Venkat P, Chopp M, Chen J. Blood-Brain Barrier Disruption, Vascular Impairment, and Ischemia/Reperfusion Damage in Diabetic Stroke. *J Am Heart Assoc*. 2017;6(6):1-11. doi: 10.1161/JAHA.117.005819
8. Chimowitz MI, Lynn MJ, Howlett-Smith H, et al. Comparison of Warfarin and Aspirin for Symptomatic Intracranial Arterial Stenosis. *N Engl J Med*. 2005;352(13):1305-16. doi: 10.1056/NEJMoa043033
9. Zhang D, Zhao ZY, Qian YW, et al. Low-Dose Rivaroxaban plus Antiplatelet Therapy for Symptomatic Intracranial Atherosclerotic Stenosis: A Prospective Cohort Study. *International Journal of Stroke*. 2026: 17474930261423387. doi: 10.1177/17474930261423387
10. Sridharan K, Sivaramakrishnan G. Hemorrhage Risk Associated with Anticoagulant and Antiplatelet Drug Combinations: Insights from the USFDA Adverse Event Reporting System. *J Clin Med*. 2025;14(17):6262. doi: 10.3390/jcm14176262
11. Du CH, Mang J, Yu SY, et al. Weighing in on the Off-Label Use: Initial Experience of Neuroform EZ Stenting for Intracranial Arterial Stenosis in 45 Patients. *Front Neurol*. 2018;9:852. doi: 10.3389/fneur.2018.00852
12. Huang R, Yang B, Gao F, et al. Association between Basilar Artery Stenosis Features, Vertebral Artery Stenosis and Perforator Stroke after Stenting. *Interventional Neuroradiology*. 2025;31(2):246-54. doi: 10.1177/15910199231164840
13. Wang T, Luo JC, Li TH, et al. Stenting versus Medical Treatment Alone for Symptomatic Intracranial Artery Stenosis: a Preplanned Pooled Individual Patient Data Analysis. *J Neurointerv Surg*. 2025;17(1):1032-39. doi: 10.1136/jnis-2024-022189
14. Sun X, Deng YM, Zhang Y, et al. Balloon Angioplasty vs Medical Management for Intracranial Artery Stenosis: The BASIS Randomized Clinical Trial. *JAMA*. 2024;332(13):1059-69. doi: 10.1001/jama.2024.12829
15. Gupta V, Parthasarathy R, Khan IA. Basilar Artery Stenosis: Technical Tips for Endovascular Revascularization. *Clin Neurol Neurosurg*. 2023;231:107792. doi: 10.1016/j.clineuro.2023.107792
16. Palmisciano P, Hoz SS, Algburi HA, et al. Percutaneous Transluminal Angioplasty and/or Stenting for the Treatment of Basilar Artery Stenosis: a Systematic Review and Meta-Analysis. *Neuroradiology*. 2023;65(6):985-1000. doi: 10.1007/s00234-023-03124-x