Section: Neurosurgery

Case Series

EVALUATING THE SAFETY OF INTRACRANIAL CAROTID ARTERY STENTING: OUR EXPERIENCE

Nidhi Roy[1], Ajay Sharma[2], Nakul Rathore[3], Akshay Pol[1], Abu Shahma[1], Pandurang Barve[3], D.K. Tyagi[4]

[1]*Senior Resident, Department of Neurosurgery, Topiwala National Medical College, Mumbai, Maharashtra, India*
[2]*Senior Resident, Department of Neurosurgery, UCMS and GTB Hospital, Delhi, India*
[3]*Assistant Professor, Department of Neurosurgery, Topiwala National Medical College, Mumbai, Maharashtra, India*
[4]*Professor and Head, Department of Neurosurgery, Topiwala National Medical College, Mumbai, Maharashtra, India.*

Received *: 03/01/2026*
Received in revised form *: 08/02/2026*
Accepted *: 26/02/2026*

Corresponding Author:
Dr. Nidhi Roy,
Senior Resident, Department of Neurosurgery, Topiwala National Medical College, Mumbai, Maharashtra, India.
Email: docnidhink@gmail.com

DOI: 10.70034/ijmedph.2026.2.39

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (1); 221-228

ABSTRACT

Intracranial atherosclerotic disease is a major cause of ischemic stroke in Asian populations, and while aggressive medical therapy remains the standard of care, a subset of patients with severe intracranial stenosis continues to experience recurrent ischemic events. This retrospective case series evaluates the safety and short-term outcomes of intracranial carotid artery stenting in five medically refractory patients with cavernous ICA stenosis, including four patients with right-sided disease presenting with recurrent TIAs and one patient with left-sided stenosis presenting with persistent headache and dizziness. All patients underwent detailed clinical and angiographic evaluation, followed by endovascular treatment using balloon angioplasty and/or intracranial stenting under standardized institutional protocols. The degree of stenosis ranged from 68% to 90%, and technical success was achieved in all cases with satisfactory luminal expansion and restoration of antegrade flow. No peri-procedural complications, including stroke, haemorrhage, or death, were observed. All patients demonstrated complete resolution of presenting ischemic or haemodynamic symptoms and remained free of recurrent transient ischemic attacks or stroke during 3–6 months of follow-up. These findings suggest that intracranial carotid artery stenting can be performed safely in carefully selected, medically refractory patients when undertaken in experienced centres, although larger prospective studies with longer follow-up are required to define its long-term role.

Keywords: Intracranial atherosclerotic disease, Cavernous internal carotid artery, Intracranial carotid artery stenting, Endovascular treatment, Ischemic stroke.

INTRODUCTION

Stroke is a major public health concern and a leading cause of death and long-term disability worldwide.[1] Intracranial atherosclerotic disease (ICAD) contributes to roughly 10% of ischemic strokes in Western populations but up to 30–50% in Asians.[2] In India, ICAD is responsible for nearly one-third of ischemic strokes, highlighting its significant regional impact.[3]

Maximal medical management is the first-line therapy for symptomatic intracranial stenosis, comprising dual antiplatelets (aspirin and clopidogrel), high-intensity statins, strict blood pressure and glycaemic control, and lifestyle modifications.[4] The WASID (Warfarin–Aspirin Symptomatic Intracranial Disease) trial showed warfarin offered no advantage over aspirin and posed greater risks, establishing antiplatelets as the standard of care.[5] Subsequent trials, including SAMMPRIS (Stenting vs. Aggressive Medical Management for Preventing Recurrent Stroke in Intracranial Stenosis), demonstrated that intensive medical therapy significantly reduced stroke recurrence and outperformed stenting due to lower peri-procedural risks and effective risk factor control.[4,5] Thus, current guidelines recommend aggressive medical management, particularly for anterior circulation lesions, over routine endovascular intervention in unselected patients.[6]

Despite optimal medical therapy, some patients with high-grade intracranial stenosis continue to

experience recurrent transient ischaemic attacks/ strokes, prompting consideration of endovascular options like percutaneous transluminal angioplasty and stenting.[4] Although recurrent transient ischemic attacks are the most common clinical presentation, high-grade intracranial stenosis may also manifest with haemodynamic symptoms such as persistent headache, dizziness, or imbalance due to chronic cerebral hypoperfusion.[7] However, trials such as SAMMPRIS, VISSIT (Vitesse Intracranial Stent Study for Ischemic Therapy), and CASSISS (China Angioplasty & Stenting for Symptomatic Intracranial Severe Stenosis) have shown no added benefit of stenting over medical therapy and reported higher peri-procedural risks.[6,8] The 2024 CASSISS follow-up confirmed similar long-term stroke rates (approximately 15%) between stenting and medical groups.[8] As a result, routine stenting is not recommended and should be limited to select patients with severe (≥70%) stenosis unresponsive to medical therapy.[4,6]

Recent evidence suggests that endovascular treatment may carry lower risks in carefully selected patients. The WEAVE (Wingspan Stent System post-market surveillance) and WOVEN (12-month follow-up of WEAVE) studies showed significantly lower complication rates (roughly 8.5% stroke or death at 1 year) when stenting was limited to on-label, medically refractory cases with proper timing post-stroke.[2] These findings, along with advancements in stent technology and procedural protocols support the potential for safe outcomes in experienced centres.[2] However, no randomized trial has yet confirmed a definitive benefit, thus endovascular treatment for ICAD remains an individualized decision weighed against its inherent risks.[2]

In regions with a high burden of ICAD like India, reducing recurrent stroke risk remains challenging when medical therapy alone is insufficient. High-volume tertiary centres in India report performing a substantial number of intracranial angioplasty and stenting procedures annually.[3] Clinicians in high-volume centres have noted that certain patients with severe intracranial carotid stenosis and recurrent ischemic events may not be suitable for prolonged medical management alone due to the urgency of their condition.[3] In this context, our institution implemented a cautious yet proactive strategy targeting a well-defined subset of patients. In the present case series, we evaluate the safety and feasibility of intracranial carotid artery stenting in symptomatic patients at a tertiary care centre in India.

MATERIALS AND METHODS

The present retrospective case series was conducted in the Department of Neurosurgery at BYL Nair Hospital, Mumbai, India.

Institutional records were reviewed to identify patients who had undergone intracranial carotid artery angioplasty and stenting for symptomatic cavernous internal carotid artery (ICA) stenosis during the study period. Patients were retrospectively included if they had angiographically confirmed cavernous ICA stenosis ≥50% with either recurrent ischemic symptoms or persistent haemodynamic symptoms attributable to the stenotic lesion, despite optimal medical therapy. Patients who had undergone the procedure for non-atherosclerotic intracranial vasculopathies, those with incomplete clinical or angiographic data, or those in whom the procedure was contraindicated due to severe comorbid conditions or inability to receive antiplatelet therapy were excluded from analysis. Five cases fulfilled these criteria, and were selected for the study.

Prior to intervention, all patients underwent detailed clinical assessment, including neurological examination and evaluation of vascular risk factors. Diagnostic digital subtraction angiography (DSA) was performed in all cases to confirm the site and severity of stenosis, assess lesion morphology, and evaluate distal cerebral circulation. The degree of stenosis was quantified using standard angiographic measurement techniques.

Endovascular procedures were carried out under standardized institutional protocols. Vascular access was obtained through the femoral artery. Following diagnostic angiography, balloon angioplasty was performed where required, followed by intracranial stenting across the stenotic segment of the cavernous ICA. Selection of balloon size and stent type was individualized based on vessel diameter and lesion characteristics. Technical success was defined as successful placement of the stent with adequate luminal expansion and restoration of antegrade flow, without immediate procedural complications.

All patients received dual antiplatelet therapy according to institutional protocol, initiated pre-procedurally and continued post-intervention. Dual antiplatelet therapy was continued for at least 3–6 months post-procedure, followed by lifelong single antiplatelet therapy. Standard peri-procedural anticoagulation measures and continuous hemodynamic monitoring were maintained during the procedure and in the immediate postoperative period.

Clinical, radiological, procedural, and outcome data were retrospectively collected from patient medical records and angiographic archives.

Case 1

A 53-year-old male was referred to our tertiary care centre with recurrent transient ischemic attacks (TIA) over a 3-month period, characterized by episodic left-sided facial numbness and upper-limb weakness lasting 10–15 minutes followed by complete spontaneous resolution. The frequency had increased to two to three episodes per week in the past two weeks despite ongoing medical therapy. He was a known hypertensive for 6 years, well controlled on amlodipine 5 mg once daily, and had dyslipidaemia treated with atorvastatin 40 mg HS.

He was a current smoker with a 20-pack-year history. There was no history of diabetes mellitus, coronary artery disease, atrial fibrillation, vasculitis, connective tissue disease, or prior intracranial haemorrhage.

On examination, the patient was conscious and oriented with stable vital parameters (blood pressure 138/84 mmHg, heart rate 76 beats/min). Neurological examination was unremarkable with no focal deficits (NIHSS score 0), and systemic examination was normal. Despite more than six weeks of dual antiplatelet therapy (aspirin 75 mg/day and clopidogrel 75 mg/day), high-intensity statin therapy, and risk-factor modification, the patient continued to experience recurrent ischemic symptoms, consistent with medically refractory intracranial atherosclerotic disease.

Baseline laboratory investigations were within normal limits, including haemoglobin 14.1 g/dL, platelet count 2.4 × 10^5/µL, serum creatinine 0.9 mg/dL, LDL cholesterol 92 mg/dL, and HbA1c 5.6%. Electrocardiography and transthoracic echocardiography showed no cardioembolic source. Brain magnetic resonance imaging (MRI) revealed no acute infarction, while time-of-flight MR angiography suggested significant stenosis of the right cavernous internal carotid artery. Digital subtraction angiography (DSA) confirmed a focal, eccentric 72% atherosclerotic stenosis of the right cavernous ICA (NASCET criteria), with preserved distal circulation and no evidence of dissection or thrombus.

Following multidisciplinary discussion and informed consent, endovascular revascularisation was performed under conscious sedation and with continuous haemodynamic monitoring. After femoral arterial access (right femoral artery) and systemic heparinisation (ACT 250–300 s), the lesion was crossed, balloon angioplasty was performed, and an appropriately sized intracranial stent was placed. Post-procedural angiography demonstrated good stent apposition, significant luminal gain, and restoration of brisk antegrade flow without complications [Figure 1].

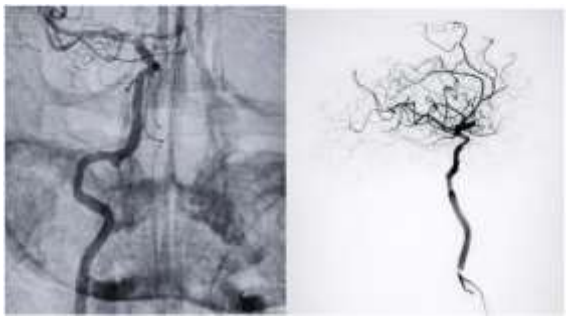

Figure 1: Pre- and post-procedure digital subtraction angiography showing right cavernous ICA stenosis with satisfactory post-stenting luminal restoration.

The post-procedural course was uneventful. Neurological examination remained normal, and a 24-hour non-contrast CT scan showed no haemorrhage. The patient was discharged on dual antiplatelet and statin therapy on post-procedure day three.

At 3- and 6-month follow-up, the patient remained asymptomatic with complete resolution of TIAs and no recurrent ischemic events.

Case 2

A 52-year-old male presented with recurrent transient neurological deficits over two months, manifesting as sudden-onset left upper-limb weakness with transient speech difficulty. Each episode lasted 5–10 minutes followed by complete resolution. The frequency of events had increased despite ongoing medical therapy, prompting referral for further evaluation. He was a known hypertensive for 8 years managed with telmisartan 40 mg once daily, and had type 2 diabetes mellitus for 5 years managed with metformin 500 mg twice daily. He was a smoker with a 15-pack-year history, but quit one year back. There was no past history of stroke, myocardial infarction, or peripheral arterial disease.

On admission, the patient was alert and oriented with stable vital parameters (blood pressure 142/86 mmHg, heart rate 80 beats/min). Neurological examination revealed no focal deficits (NIHSS score 0), and systemic examination was unremarkable. He had been on dual antiplatelet therapy (aspirin 75 mg/day and clopidogrel 75 mg/day) for over one month, along with high-intensity statin therapy (rosuvastatin 40 mg/day), with optimised glycaemic and blood pressure control. Despite strict compliance, he continued to experience recurrent ischemic symptoms, consistent with failure of aggressive medical management.

Laboratory investigations were within acceptable limits, including haemoglobin 13.6 g/dL, platelet count 2.6 × 10^5/µL, serum creatinine 1.0 mg/dL, fasting blood glucose 118 mg/dL, HbA1c 6.8%, and LDL cholesterol 88 mg/dL. Electrocardiography showed normal sinus rhythm, and transthoracic echocardiography revealed no intracardiac thrombus or structural abnormality. MRI Brain demonstrated no acute diffusion restriction, while MR angiography revealed significant luminal narrowing of the right intracranial internal carotid artery, predominantly involving the cavernous segment. DSA confirmed a 73% concentric atherosclerotic stenosis of the right cavernous ICA. The lesion was relatively short in length with smooth margins, without evidence of ulceration or intraluminal thrombus. Distal flow was maintained, though delayed, with adequate opacification of distal ICA branches.

After multidisciplinary discussion and informed consent, the patient underwent primary intracranial stenting under conscious sedation with continuous neurological and haemodynamic monitoring. Vascular access was obtained via the right femoral artery, and systemic anticoagulation was achieved using intravenous heparin. A guiding catheter was navigated into the cervical ICA, and the lesion was carefully crossed using a microcatheter–microwire

system. Given the favourable lesion morphology and vessel calibre, direct intracranial stent placement was performed without prior balloon angioplasty. Post-procedural angiography demonstrated optimal stent expansion with restoration of normal luminal diameter and brisk antegrade flow, without evidence of distal embolization, dissection, or perforation [Figure 2].

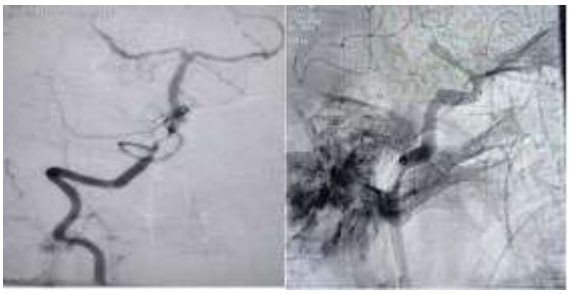

Figure 2: Angiographic images demonstrating pre-procedure stenosis and post-stenting vessel patency.

The post-procedural course was uneventful. Neurological status remained intact, and a 24-hour non-contrast CT scan showed no haemorrhage. The patient was discharged on post-procedure day three on dual antiplatelet and statin therapy. At 3 and 6-month follow-up, the patient reported complete resolution of transient ischemic episodes with no recurrent neurological events. He remained compliant with medical therapy and lifestyle modifications, and no delayed complications were noted.

Case 3

A 54-year-old male presented with recurrent transient ischemic episodes over past six weeks, characterized by sudden-onset left-sided limb weakness and transient slurring of speech lasting 5–20 minutes, with complete neurological recovery thereafter. Episodes occurred intermittently, often during physical exertion, and persisted despite medical therapy. He had a 9-year history of hypertension treated with atenolol 50 mg once daily and dyslipidaemia managed with atorvastatin 40 mg HS. The patient was overweight (BMI 29.6 kg/m²) with a sedentary lifestyle. He reported loud snoring with excessive daytime somnolence, suggestive of undiagnosed obstructive sleep apnoea. There was no history of diabetes mellitus, atrial fibrillation, ischemic heart disease, or prior stroke. He was a smoker with a 20-pack-year history, but had quit three years back. The patient had been on dual antiplatelet therapy (aspirin 75 mg/day and clopidogrel 75 mg/day) along with high-intensity statin therapy for more than four weeks. Blood pressure optimisation and lifestyle modification were reinforced. Despite compliance with aggressive medical management, he continued to experience recurrent ischemic symptoms, indicating failure of optimal medical therapy.

On admission, vital signs were stable (blood pressure 140/84 mmHg, heart rate 72 beats/min). Neurological examination was normal with no focal deficits (NIHSS score 0), and systemic examination was unremarkable. Despite more than four weeks of dual antiplatelet therapy (aspirin 75 mg/day and clopidogrel 75 mg/day) and high-intensity statin therapy (atorvastatin 80 mg/day), along with blood pressure optimisation and lifestyle modification, the patient continued to experience recurrent ischemic symptoms.

Laboratory investigations were within acceptable limits, including haemoglobin 14.0 g/dL, platelet count 2.5 × 10^5/µL, serum creatinine 0.9 mg/dL, LDL cholesterol 108 mg/dL, triglycerides 186 mg/dL, and fasting blood glucose 102 mg/dL. Electrocardiography and transthoracic echocardiography revealed no cardioembolic source. Brain MRI showed no acute infarction, while MR angiography demonstrated significant narrowing of the right intracranial internal carotid artery involving the cavernous segment. DSA confirmed a 68% stenosis of the right cavernous internal carotid artery, consistent with a focal atherosclerotic lesion. Distal intracranial circulation was preserved, although delayed, suggesting haemodynamic compromise in the setting of recurrent symptoms.

Following multidisciplinary discussion and informed consent, endovascular revascularisation was performed under conscious sedation and continuous neurological and haemodynamic monitoring. Femoral arterial access was obtained, and systemic anticoagulation was achieved with intravenous heparin. Following careful lesion crossing, balloon angioplasty was performed to optimise luminal diameter, followed by intracranial stent placement across the stenotic cavernous ICA segment. Post-procedural angiography demonstrated adequate stent expansion, significant luminal gain, and restoration of antegrade flow, without angiographic evidence of vessel dissection, distal embolization, or perforation [Figure 3].

The post-procedural course was uneventful. Neurological status remained intact, and a follow-up non-contrast CT scan showed no haemorrhagic or ischemic complications. The patient was discharged in stable condition. At follow-up, he reported complete resolution of transient ischemic symptoms with no recurrent neurological events. He was advised regarding weight reduction, increased physical activity, and further evaluation for sleep-disordered breathing.

Figure 3: Pre-intervention and post-intervention angiograms showing improved vessel calibre.

Case 4

A 61-year-old male was referred with recurrent transient ischemic attacks over past four months, presenting as sudden-onset left-sided weakness with transient dysarthria and facial deviation lasting 10–20 minutes and then resolving completely. Over the preceding month, episode frequency increased to nearly three per week despite medical therapy. He had a 12-year history of type 2 diabetes mellitus and a 10-year history of hypertension, both on irregular treatment (metformin 500 mg twice daily and amlodipine 5 mg once daily). He was an alcoholic (60–80 g/day for >15 years). He was also a smoker with a 20-pack-year history. There was no prior history of stroke, myocardial infarction, or peripheral vascular disease.

On admission, the patient was alert and oriented with blood pressure 146/88 mmHg, heart rate 82 beats/min, respiratory rate 18/min, and oxygen saturation of 96% on room air. Neurological examination was normal with no focal deficits (NIHSS score 0), and systemic examination was unremarkable. Despite treatment with dual antiplatelet therapy (aspirin 75 mg/day, clopidogrel 75 mg/day) and high-intensity statin therapy (atorvastatin 40 mg/day), along with advice on risk-factor modification, the patient continued to experience recurrent ischemic events, consistent with medical therapy–refractory intracranial atherosclerotic disease.

Laboratory investigations showed haemoglobin 13.2 g/dL, platelet count 2.1 × 10^5/µL, serum creatinine 1.1 mg/dL, fasting blood glucose 132 mg/dL, HbA1c 7.4%, and LDL cholesterol 102 mg/dL. Liver function tests were normal. Electrocardiography and transthoracic echocardiography revealed no cardioembolic source. MRI brain did not show any acute infarction, while MR angiography revealed critical narrowing of the right intracranial internal carotid artery. DSA demonstrated a severe (approximately 90%) stenosis of the right cavernous segment of the internal carotid artery, with marked luminal compromise and delayed distal flow. The lesion was long-segment, concentric, and atherosclerotic in appearance, without evidence of dissection or intraluminal thrombus.

Given the severity of stenosis, recurrent symptoms, and failure of aggressive medical therapy, the patient was selected for endovascular revascularisation following multidisciplinary discussion. The procedure was performed under conscious sedation with continuous neurological and haemodynamic monitoring. Femoral arterial access was obtained, and systemic anticoagulation was achieved with intravenous heparin. A guiding catheter was advanced into the cervical ICA, and the lesion was carefully crossed using a microcatheter–microwire system. Considering the critical stenosis and vessel characteristics, primary intracranial stenting was performed without prior balloon angioplasty to minimise the risk of plaque disruption and distal embolization. Post-deployment angiography demonstrated excellent stent expansion with restoration of antegrade flow, with no evidence of vessel perforation, dissection, or distal embolic phenomena [Figure 4]. The patient tolerated the procedure well and remained neurologically intact throughout the peri-procedural period. He was monitored in the neuro-intensive care unit for 48 hours. Dual antiplatelet therapy was continued, and strict blood pressure and glycaemic control were ensured. The patient was counselled extensively regarding alcohol cessation and lifestyle modification.

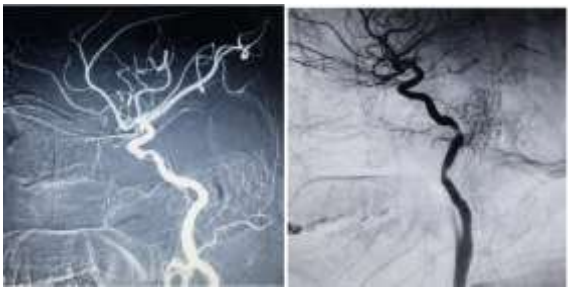

Figure 4: Angiographic images showing critical stenosis with satisfactory flow restoration following stent deployment.

A non-contrast CT scan of the brain performed 24 hours post-procedure showed no evidence of intracranial haemorrhage or new ischemic changes. The patient was discharged in stable condition on the fourth post-procedural day. At follow-up, the patient reported complete resolution of transient ischemic symptoms, with no further TIAs or stroke events. He remained neurologically stable and compliant with prescribed medications.

Case 5

A 59-year-old male presented to our tertiary care centre with a history of persistent headache and episodic dizziness for approximately two months. The headache was described as dull, diffuse, and intermittent, without features of migraine, visual aura, or associated vomiting. The episodes of dizziness were non-rotatory, often precipitated by sudden head movements, and occasionally accompanied by a sensation of imbalance. There was no history of focal neurological deficits, loss of consciousness, seizures, or prior cerebrovascular events. Also, the patient had no known medical comorbidities. He was a non-smoker and denied alcohol consumption. There was no family history of premature cardiovascular or cerebrovascular disease. The patient led an active lifestyle and was not on any long-term medications at presentation.

On examination, the patient was conscious, alert, and oriented. Vital parameters were within normal limits, with a blood pressure 128/80 mmHg, heart rate 70 beats/min, respiratory rate 16/min, and oxygen saturation 99% on room air. Neurological examination revealed no focal motor, sensory, or cranial nerve deficits (NIHSS score: 0). Cerebellar signs were absent, and gait examination was normal.

Systemic examination did not reveal any abnormality.

The patient initially underwent conservative management due to the absence of focal neurological deficits and any atypical symptoms. He was started on antiplatelet therapy (aspirin 75 mg/day) and statin therapy (atorvastatin 40 mg/day) following preliminary imaging suggestive of intracranial arterial narrowing. Despite medical therapy and symptomatic management over several weeks, the patient continued to experience persistent headaches and recurrent dizziness. Further evaluation was done.

Routine laboratory investigations were within normal limits, including haemoglobin 14.4 g/dL, platelet count 2.7 × 10^5/µL, serum creatinine 0.9 mg/dL, fasting blood glucose 98 mg/dL, and lipid profile within reference ranges. ECG showed normal sinus rhythm, and transthoracic echocardiography did not identify any cardiac source of embolism. MRI brain revealed no acute or chronic ischemic lesions. However, MR angiography demonstrated significant luminal narrowing of the left intracranial internal carotid artery, involving the cavernous segment. Subsequent DSA confirmed a high-grade (approximately 85%) stenosis of the left cavernous internal carotid artery. The lesion was concentric and atherosclerotic in appearance, without evidence of dissection or intraluminal thrombus. Distal flow was preserved but appeared delayed, suggesting haemodynamic compromise. The clinical presentation was attributed to haemodynamic insufficiency secondary to critical left cavernous internal carotid artery stenosis.

Given failure of conservative medical therapy and angiographic evidence of haemodynamic compromise, the patient was considered medically refractory. In view of the severity of stenosis and persistent symptoms, the patient was considered for endovascular revascularisation.

After obtaining written informed consent, the procedure was performed under conscious sedation with continuous haemodynamic and neurological monitoring. Vascular access was obtained via the femoral artery, and systemic anticoagulation was achieved with intravenous heparin. A guiding catheter was positioned in the cervical ICA, and the lesion was carefully crossed using a micro guidewire. Intracranial stenting was performed across the stenotic cavernous ICA segment. Post-procedural angiography demonstrated excellent stent expansion with restoration of normal luminal calibre and brisk antegrade flow, without evidence of vessel dissection, perforation, or distal embolization [Figure 5].

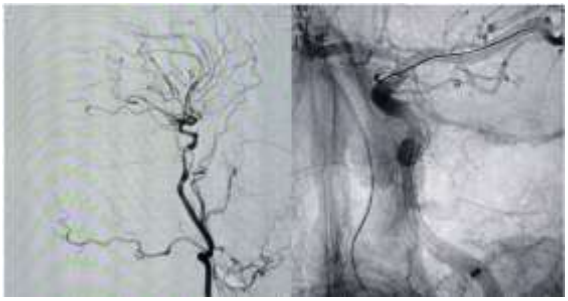

Figure 5: Angiographic images showing critical stenosis with satisfactory flow restoration after stenting.

The patient tolerated the procedure well, with no intra-procedural or immediate post-procedural complications. Neurological examination remained normal throughout the hospital stay. A non-contrast CT scan of the brain obtained 24 hours after the procedure showed no intracranial haemorrhage or ischemic changes.

The patient was discharged in stable condition on dual antiplatelet therapy and statin therapy. At subsequent follow-up, he reported significant improvement in headache and complete resolution of dizziness, with no new neurological symptoms.

DISCUSSION

In our case series of five patients with symptomatic cavernous ICA stenosis, intracranial stenting was performed without any peri-procedural complications, and all patients remained symptom-free at 3–6 months follow-up. This favourable short-term outcome contrasts with early randomized trials like SAMMPRIS and VISSIT, which reported high complication rates with stenting. Chimowitz et al. (4,9) (SAMMPRIS) found a 30-day stroke or death rate of 14.7% in the stenting arm versus 5.8% with medical therapy (p=0.002), leading to a 1-year primary endpoint incidence of 20.0% vs 12.2% (p=0.009). Similarly, Zaidat et al. (10) (VISSIT) halted enrolment early after observing 30-day stroke/TIA rates of 24.1% in the stent group compared to 9.4% with medical management (p=0.05), along with significantly higher 12-month stroke/TIA in the stent arm (36.2% vs 15.1%, p=0.02). Both trials concluded that adding intracranial stenting to aggressive medical therapy provided no benefit and in fact increased early risks.[9,10] These findings established intensive medical management as the standard first-line treatment and raised concerns that the peri-procedural hazards of stenting outweighed any potential reduction in recurrent stroke in unselected patients.[9,10]

More recent evidence from the CASSISS trial aligns with the concept that routine stenting does not improve outcomes over medical therapy. Gao et al,[11] randomized 380 patients and found no significant difference in stroke or death rates between stenting plus medical therapy and medical therapy alone. The 1-year risk of stroke or death was

8.0% with stenting vs 7.2% with medical treatment (p=0.82).[11] Even with longer follow-up, outcomes remained equivalent. The 3-year stroke rates in the target artery were 11.3% vs 11.2% in both groups (p>0.99).[11] While the stenting arm showed a trend toward higher 3-year mortality (4.4% vs 1.3%; p=0.08), this difference was not statistically significant.[11] Importantly, CASSISS incorporated refined selection criteria (severe 70–99% stenosis, exclusion of recent perforator strokes, and waiting ≥3 weeks post-symptom onset to intervene) in an attempt to improve safety (11). Nonetheless, the neutral results reinforced prior trial evidence that intracranial stenting offers no clear advantage in preventing strokes when compared to best medical therapy.[11] Current guidelines have accordingly advised against routine use of intracranial stents for stroke prevention, except in carefully selected cases of severe stenosis with recurrent ischemia despite optimal medical treatment.[4,6,12] Our patient selection reflects this viewpoint of reserving stenting for medically refractory, high-risk scenarios.

The striking absence of complications in our series underscores the impact of strict adherence to proper patient selection and timing. All procedures in our series were performed beyond the hyperacute phase, under strict protocol with experienced operators. This approach is supported by prior observations that delaying intervention until a patient's condition stabilizes reduces peri-procedural risk. Suh et al,[13] reported an overall 10% 6-month stroke/death rate after intracranial angioplasty and stenting, but the risk was highly dependent on symptom stability. Patients treated in a stable phase (with no rapid fluctuations in symptoms) had only a 4.1% adverse event rate, whereas those stented during an unstable period of ongoing neurological decline suffered a 25.9% complication rate (p=0.004). This emphasizes that careful timing is critical for safety. In the FDA-mandated WEAVE registry, which enrolled 152 on-label patients, Alexander et al,[14] achieved a remarkably low 72-hour stroke, bleed, or death rate of 2.6%. All WEAVE patients had ≥70% ICAD lesions, at least two prior strokes (including one on medical therapy), and underwent stenting ≥8 days after the last stroke. This stringent selection and use of experienced interventionalists resulted in a peri-procedural safety profile far better than that seen in SAMMPRIS or VISSIT. Our zero peri-procedural event rate is in line with such findings, suggesting that in a controlled setting with appropriate precautions, intracranial stenting can be performed with minimal immediate risk.

Beyond the peri-procedural period, the short-term efficacy observed in our patients (no recurrent TIA or stroke in up to 6 months) is encouraging, though longer follow-up is needed to confirm durability. It is worth noting that in the WEAVE/WOVEN cohort, the low early risk was maintained over time. The WOVEN study – a 12-month follow-up of WEAVE – reported an 8.5% cumulative rate of stroke or death at 1 year among on-label stented patients (15). Of 129 patients followed, only seven experienced any stroke during the year (six minor, one major) and there were no post-procedural deaths.[15] Including the 72-hour events, Alexander et al,[15] documented a total 1-year stroke/death rate of 8.5%, which represents a substantial improvement over the outcomes of earlier trials. In fact, post-hoc analyses suggest that when stenting is successful without early complications, subsequent stroke rates approach those of medical therapy. An editorial by Kim,[2] highlighted that between 1 month and 1 year, the incidence of ischemic events in properly selected stented patients (≈5.4%) was comparable to the SAMMPRIS medical arm (~6.5%). Thus, the key to unlocking potential benefit from stenting lies in eliminating the excess upfront risk. Our findings add to this i.e. by avoiding any peri-procedural strokes, our patients could fully realize the protective effect of revascularization, remaining symptom-free in the months following stent placement.

Despite these encouraging results in our study, it is essential to understand that multiple randomized trials (SAMMPRIS, VISSIT, CASSISS) have failed to show a superiority of stenting over aggressive medical management in reducing strokes, largely due to the procedural risks involved.[9,10] Our zero-complication outcome likely reflects both the small sample size and the high level of expertise and caution applied; it may not be generalizable to all settings. Furthermore, our follow-up of 3–6 months, while reassuring, is short. Thus, it remains to be seen whether the excellent outcomes in our five cases persist over years. We also acknowledge that our series predominantly involved right-sided cavernous ICA lesions, with one case of left-sided cavernous ICA stenosis, which tend to be more accessible and possibly safer for stenting than lesions in certain other locations (e.g. vertebrobasilar or middle cerebral arteries).[13] This may limit direct comparison to trials that included various arterial territories. Nonetheless, the cavernous ICA is an anatomically challenging segment, and the absence of any cranial nerve palsies, perforator strokes, or distal emboli in our patients is noteworthy and speaks to the procedural care taken.

CONCLUSION

This case series highlights that intracranial carotid artery stenting, when restricted to carefully selected patients with symptomatic cavernous ICA stenosis refractory to optimal medical therapy, can be performed safely with favourable short-term outcomes. All patients achieved successful revascularization without peri-procedural complications and remained free from recurrent ischemic or haemodynamic symptoms during follow-up. These results reinforce the importance of

stringent patient selection, appropriate timing beyond the acute phase, and execution in experienced high-volume centres. While aggressive medical management remains the cornerstone of treatment, intracranial stenting may serve as a viable option in select high-risk patients. Larger prospective studies with longer follow-up are required to validate durability and define its precise role in contemporary practice.

REFERENCES

1. Feigin VL, Brainin M, Norrving B, Martins SO, Pandian J, Lindsay P, et al. World Stroke Organization: Global Stroke Fact Sheet 2025. Int J Stroke. 2025 Feb;20(2):132–44.
2. Kim DJ. Intracranial Stenting; the Current Landscape. Neurointervention. 2021 Feb 24;16(1):2–5.
3. Bell J. BRAIN 2024: ICAD confronted as globally relevant yet under-addressed stroke aetiology [Internet]. NeuroNews International. 2024 [cited 2026 Jan 22]. Available from: https://neuronewsinternational.com/brain-2024-icad-confronted-as-globally-relevant-yet-under-addressed-stroke-aetiology/
4. Chimowitz MI, Lynn MJ, Derdeyn CP, Turan TN, Fiorella D, Lane BF, et al. Stenting versus Aggressive Medical Therapy for Intracranial Arterial Stenosis. N Engl J Med. 2011 Sep 15;365(11):993–1003.
5. Chimowitz MI, Lynn MJ, Howlett-Smith H, Stern BJ, Hertzberg VS, Frankel MR, et al. Comparison of warfarin and aspirin for symptomatic intracranial arterial stenosis. N Engl J Med. 2005 Mar 31;352(13):1305–16.
6. Turan TN, Zaidat OO, Gronseth GS, Chimowitz MI, Culebras A, Furlan AJ, et al. Stroke Prevention in Symptomatic Large Artery Intracranial Atherosclerosis Practice Advisory. Neurology. 2022 Mar 22;98(12):486–98.
7. Nouh A, Remke J, Ruland S. Ischemic Posterior Circulation Stroke: A Review of Anatomy, Clinical Presentations, Diagnosis, and Current Management. Front Neurol [Internet]. 2014 Apr 7 [cited 2026 Feb 16];5. Available from: https://www.frontiersin.org/journals/neurology/articles/10.3389/fneur.2014.00030/full
8. Bell J. Seven-year data provide “compelling evidence” that intracranial stenting does not confer benefits over medical therapy [Internet]. NeuroNews International. 2024 [cited 2026 Jan 22]. Available from: https://neuronewsinternational.com/seven-year-data-provide-compelling-evidence-that-intracranial-stenting-does-not-confer-benefits-over-medical-therapy/
9. Derdeyn CP, Fiorella D, Lynn MJ, Turan TN, Lane BF, Janis LS. Intracranial Angioplasty and Stenting: Current SAMMPRIS results and future directions. Stroke. 2013 Jun;44(6 0 1):10.1161/STROKEAHA.111.000370.
10. Zaidat OO, Fitzsimmons BF, Woodward BK, Wang Z, Killer-Oberpfalzer M, Wakhloo A, 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 Mar 24;313(12):1240–8.
11. Gao P, Wang T, Wang D, Liebeskind DS, Shi H, Li T, 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 Aug 9;328(6):534–42.
12. Kleindorfer DO, Towfighi A, Chaturvedi S, Cockroft KM, Gutierrez J, Lombardi-Hill D, et al. 2021 Guideline for the Prevention of Stroke in Patients With Stroke and Transient Ischemic Attack: A Guideline From the American Heart Association/American Stroke Association. Stroke. 2021 Jul;52(7):e364–467.
13. Suh DC, Kim JK, Choi JW, Choi BS, Pyun HW, Choi YJ, et al. Intracranial stenting of severe symptomatic intracranial stenosis: results of 100 consecutive patients. AJNR Am J Neuroradiol. 2008 Apr;29(4):781–5.
14. Alexander MJ, Zauner A, Chaloupka JC, Baxter B, Callison RC, Gupta R, et al. WEAVE Trial: Final Results in 152 On-Label Patients. Stroke. 2019 Apr;50(4):889–94.
15. Alexander MJ, Zauner A, Gupta R, Alshekhlee A, Fraser JF, Toth G, et al. The WOVEN trial: Wingspan One-year Vascular Events and Neurologic Outcomes. J.