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Case Report

Deferring Angioplasty and Stenting based on Natural Progression in Severe Middle Cerebral Artery Stenosis: An Observation of Two Cases

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Introduction: Severe middle cerebral artery (MCA) stenosis, as one of the intracranial atherosclerotic diseases, is a major cause of ischemic stroke. The role and optimal timing of interventions, such as angioplasty and stenting, remain subjects of debate, particularly due to the variable natural disease progression. While some cases improve spontaneously, others progress to total occlusion, necessitating comprehensive evaluation of individualized treatment approaches. **Cases:** This case series presents two patients with severe MCA stenosis, each of whom followed a distinct clinical courses. The first patient had 88% stenosis in the M1 segment of the right MCA (NIHSS 3, mRS 2), which spontaneously improved to 57% within days (NIHSS 2, mRS 2), with enhanced distal flow, leading to the decision to defer angioplasty and stenting. In contrast, the second patient initially had severe left MCA stenosis (NIHSS 10, mRS 4), which progressed to total occlusion within three months (NIHSS 10, mRS 4), also resulting in deferred intervention. Serial cerebral digital subtraction angiography (DSA) facilitated the observation of vascular evolution and collateral circulation, guiding decisions about intervention. **Conclusion:** The clinical outcomes of MCA stenosis range widely, from spontaneous resolution to progressive occlusion. Close serial imaging observation of collateral circulation and natural progression is essential for guiding decisions about angioplasty and stenting.

Keywords: Angioplasty and stenting, Collateral circulation, Intracranial atherosclerotic stenosis, Ischemic stroke, Middle cerebral artery stenosis

Highlights
- MCA stenosis may remodel spontaneously or progress to complete occlusion
- Serial imaging observing progression spots high-risk cases, guiding intervention.

Introduction

Middle cerebral artery (MCA) stenosis is a life-threatening condition with a high incidence and severe neurological consequences that frequently leading to an ischemic stroke.[1] Even with optimal medical therapy and risk factor management, there are no clear guidelines for predicting whether MCA stenosis cases would improve spontaneously or progress to total occlusion. Current guidelines, such as the SAMMPRIS trial and its follow-up studies, the Chinese Stroke Association (CSA) 2019 Guidelines, and the American Heart Association/American Stroke Association (AHA/ASA) 2019 Guidelines, all emphasize aggressive medical management as the cornerstone of treatment for symptomatic intracranial atherosclerotic stenosis (ICAS).[2] Despite these established guideline-based recommendations, the optimal timing and necessity of angiplasty and stenting

interventions continue to be debated.[3] This case report highlights the importance of observing the natural progression of severe MCA stenosis, implications for individualized decision-making regarding intervention timing.

Case

Case 1

A 53-year-old woman presented with acute-onset left-sided weakness, facial asymmetry, dysarthria, and dizziness. She had a history of type 2 diabetes mellitus (HbA1c 7.1%), but no previous cerebrovascular events. Neurological examination confirmed left central facial and lingual palsy with mild motor deficits (MRC scale 5/4 in both upper and lower limbs), indicating a stroke (NIHSS 3 and mRS 2).

A head CT angiography revealed a 0.61 cm occlusion in the M1 segment of the right MCA with collateral circulation. Additional findings included agenesis of the right posterior communicating artery, hypoplasia of the left posterior communicating artery, and subacute-chronic thromboembolic infarction in the right MCA territory. Lacunar infarcts were also found in the right corona radiata, caudate nucleus, and anterior peri-cornu of the left lateral ventricle.

Cerebral DSA with devices of introducer sheath 5F and vertebral catheter 5F on day five after stroke showed severe stenosis in the right MCA segment M1 (88%) with a 0.19 mm stenotic lumen, a 1.62 mm distal lumen, and a 3.36 mm stenotic length (Mori type A). A saccular aneurysm was identified in the left supraclinoid internal carotid artery (ICA, neck: 2.52 mm, dome: 3.31 × 4.14 mm).

Daily preventive therapy began with ticagrelor 180 mg, aspirin 80, and atorvastatin 40 mg. Angioplasty with stenting and aneurysm coiling was planned for day 12. A follow-up cerebral DSA revealed spontaneous stenosis improvement (57%, Mori type A) with better distal flow (Figure 1). The aneurysm was coiled during this period (90% packing, modified Raymond-Roy Class II) (Figure 2). Stroke prevention therapy was continued. Clinical symptoms slightly improved (NIHSS improved from 3 to 2), but the patient's overall functional disability remained unchanged (mRS 2) due to residual minor deficits. Angioplasty was deferred due to spontaneous improvement in stenosis severity, distal flow improvement, and clinical stability.

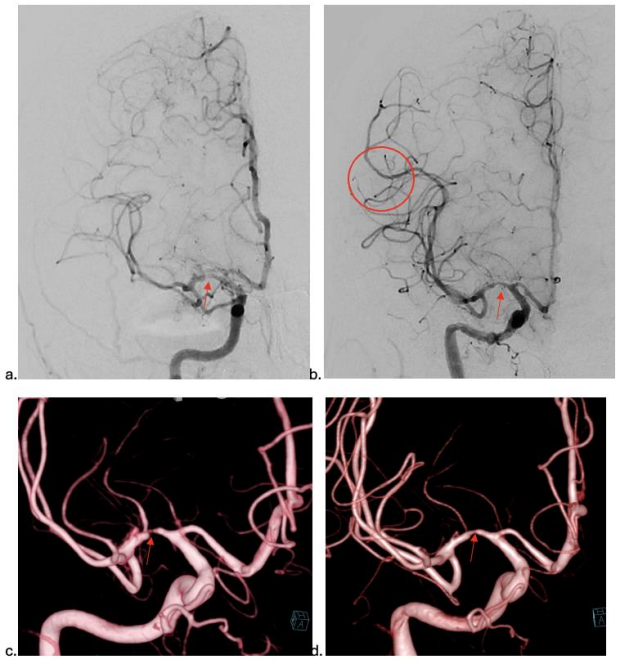

Figure 1. Comparison of cerebral angiography of case 1, injection of right internal carotid artery in 2D view (a, b) and 3D view (c, d) angiogram. Comparing the first cerebral angiogram (a, c) to the follow up cerebral angiogram (b, d) five days later, the stenosis in the right MCA (red arrow) was found to have improved 88% to 57% severity. Improved distal flow MCA also was observed in follow-up angiography (red circle).

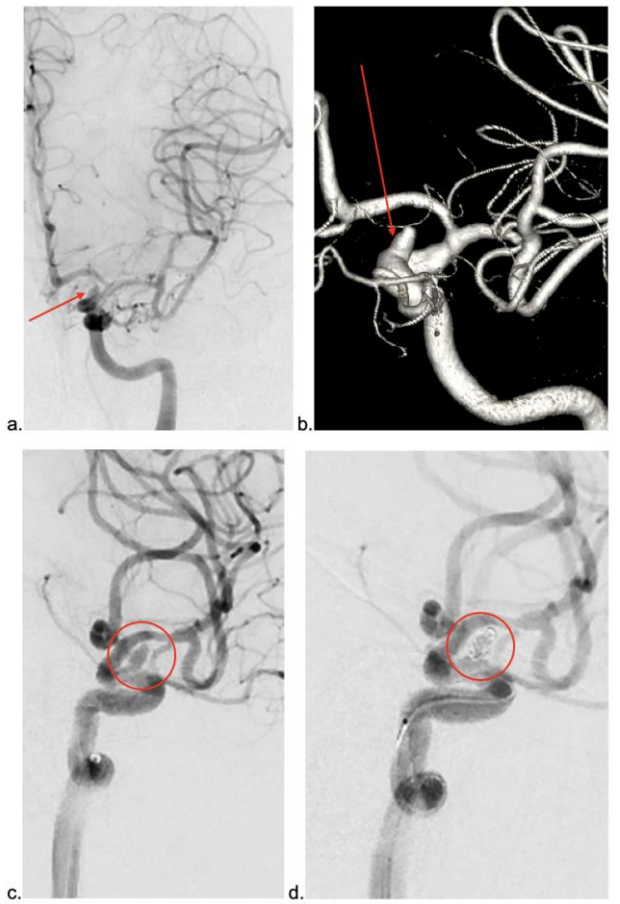

Figure 2. Left ICA injection 2D and 3D cerebral DSA images (a, b) show saccular aneurysm in the left supraclinoid ICA (red arrow) and coiling with 90% packing coil, neck remnant according to modified Raymond-Roy Class II (red circle).

Case 2

A 78-year-old woman experienced sudden-onset right-sided weakness, sensory deficits, facial asymmetry,

and dysarthria upon waking up. She had a history of stroke but no diabetes or hypertension. Neurological examination revealed severe hemiparesis (MRC 1/5 in the upper limb, 2/5 in the lower limb) as well as central facial and lingual palsy, suggesting left MCA stroke (NIHSS 10, mRS 4).

A non-contrast head CT at admission showed subacute ischemic infarction in the left putamen and corona radiata. The initial head MRA indicated significant stenosis of the left MCA segment M1. Cilostazol 100 mg, aspirin 80 mg, and atorvastatin 20 mg were started to optimize medical therapy and reduce the risk of stroke recurrence . Due to a delayed referral process, cerebral DSA was conducted two months after stroke onset and revealed approximately 90% stenosis of left MCA segment M1, with inadequate distal flow compensated by collaterals from the ipsilateral ACA and PCA (Figure 3).

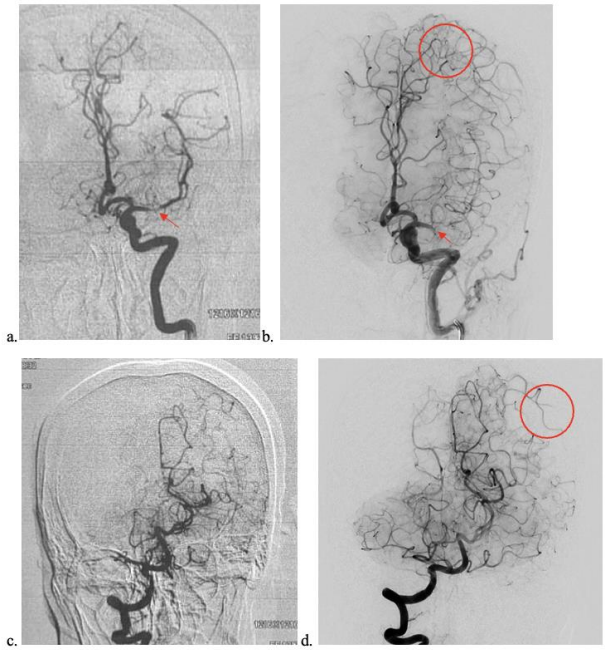

Figure 3. Comparison of cerebral angiography of case 2, 2D view injection of left internal carotid artery (a, b) and right vertebral artery (c, d). Initial angiography identified severe stenosis of the left MCA segment M1 with inadequate distal flow (a), and minimal collateral circulation from posterior (c). Follow-up angiography showed a significant progression of the disease, leading to total occlusion of the left MCA (b). There were collateral circulations (red circles) from the ipsilateral anterior cerebral artery (b) and posterior cerebral artery (d).

Three months post-stroke onset, angioplasty with stenting was attempted at a referral hospital using an 8F sheath to aid navigation. However, intra-procedural angiography revealed complete occlusion of the left MCA segment M1. Angioplasty was postponed because intervention was no longer feasible due to total occlusion and the established collateral circulation. Cilostazol, aspirin, and atorvastatin were continued as stroke prevention therapy. The patient's neurological status remained severely impaired and unchanged (NIHSS 10, mRS 4), reflecting persistent inadequate cerebral perfusion despite collateral circulation.

Discussion

Severe stenosis of the MCA segment M1 affects up to 12.9% of stroke patients, primarily as a result of atherosclerosis.[4] Current guideline-based management of ICAS includes rapid diagnosis, intravenous thrombolysis, and thrombectomy for large-vessel occlusion strokes.[2] Antiplatelet therapy and statins are essential for preventing recurrence, while stenting may be considered in cases of symptomatic ICAS with over 70% stenosis to improve perfusion and outcomes.[5] Advanced imaging, including cerebral DSA, remains essential for assessing ischemia severity.

Differences in Baseline Characteristics of Vascular Status

Cerebral angiography was performed at different time points in the two cases. In the first case, the initial cerebral DSA was performed during the acute phase of ischemic stroke at a type A hospital, with follow-up angiography conducted during the subacute phase, specifically in the second week of hospitalization. In the second case, the initial angiography was performed two months after stroke, at a type B hospital. Subsequent evaluation and intervention planning were performed a month later at a type A referral hospital. This case highlights the significant impact of regional regulatory differences in healthcare access on clinical decision-making.

These cases illustrate variations in vascular status, collateral flow, and MCA stenosis progression on angiography. Case 1 showed severe (88%) stenosis in the right MCA segment M1 (Mori Type A) with spontaneous luminal expansion (55%), and improved distal circulation, justifying conservative management. In contrast, Case 2 showed progressive stenosis in the left MCA segment M1, culminating in total occlusion with inadequate distal flow. Although collateral circulation partially compensated, it was insufficient to maintain optimal function.[6] The progression to total occlusion made unviable. These findings emphasize the importance of analyzing collateral flow in guiding decisions between conservative management and intervention.[7]

Evolution and Involution Pathophysiology

Atherosclerotic stenosis progresses through inflammation, endothelial dysfunction, smooth muscle proliferation, and hemodynamic alterations, leading to luminal narrowing.[8] Stenosis may evolve into occlusion or regress via plaque stabilization, depending on plaque composition, shear stress, and systemic factors.[9]

Vulnerable plaques with a large lipid core and thin fibrous cap are prone to rupture, accelerating occlusion. Hypertension, diabetes, hyperlipidaemia, and smoking contribute to atherosclerosis, while risk modification and medical therapy may stabilize plaques.

In Case 1, the stenosis evolution initially worsened at first due to endothelial swelling and inflammation, but then improved through thrombus dissolution and enhanced distal flow, demonstrating spontaneous recanalization.[6,7] Conservative management led to functional recovery, minimizing the need for intervention.

Conversely, Case 2 exhibited stenosis involution. Conservative management for two months after the stroke did not result in revascularization; instead, total occlusion occurred. Despite sufficient collateral circulation, progressive lipid accumulation, hemodynamic stress, and ongoing thromboembolic activity ultimately result in complete vascular occlusion. While insufficient collateral circulation increased ischemic burden, and endothelial dysfunction promoted thrombosis, precluding revascularization.[10]

These two cases illustrate the dynamic evolution and involution of cerebrovascular pathology, emphasizing the necessity of vigilant monitoring and individualized treatment strategies.[11]

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Decision Management of Intracranial Atherosclerosis Stenosis

The past, present, and future management of ICAS remain questionable. Current guidelines emphasize aggressive medical therapy as the primary treatment approach, including a 90-day course of dual antiplatelet therapy, to mitigate the risk of recurrent stroke in patients with symptomatic severe ICAS (70–99% stenosis). Dual antiplatelet therapy, along with intensive management of vascular risk factors, is strongly recommended.[12,13] Despite advances in symptomatic ICAS treatment, the risk of stroke recurrence remains high in severe cases.

Urgent angioplasty is considered in cases of recurrent ischemic events despite optimal medical management and significant neurological impairment.[3] Priority in decision-making should be accorded to high-risk patients who need aggressive endovascular treatment or when medical management proves most effective. If we are late in deciding an occlusion, but there is a possibility of improvement, ICAS still needs therapeutic guidance and further research.

This case series highlights the progression of severe MCA stenosis through serial cerebral angiography observations. Careful imaging evaluation of luminal patency and collateral circulation was crucial in guiding individualized decision regarding intervention. Diagnostic variability, including subtle differences in imaging parameters, may contribute to perceived changes in stenosis severity. Further studies are needed to refine treatment algorithms for different stenosis severities and optimize management strategies.[3]

Conclusion

Middle cerebral artery stenosis exhibits clinical variability, ranging from spontaneous resolution to progressive occlusion. Serial observations of stenosis severity and collateral circulation status on cerebral DSA are essential for guiding individualized decisions regarding the necessity and optimal timing of angioplasty and stenting interventions.

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None

Conflict of Interest
The authors declare no conflict of interest related to this case report.

Patient consent for publication
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Author contribution
Vita Kusuma Rahmawati: Investigation, Writing–Original Draft, Writing–Review and Editing. **Achmad Firdaus Sani:** Conceptualization, Investigation, Writing–Review and Editing. **Dedy Kurniawan:** Conceptualization, Investigation, Writing–Review and Editing. **Muh. Wildan Yahya:** Investigation, Writing–Review and Editing. **Faishol Hamdani:** Investigation, Writing–Review and Editing

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