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Case report

Endovascular recanalization for subacute symptomatic intracranial arterial occlusion: A report of two cases

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ABSTRACT

Background: We hope to illustrate the feasibility of endovascular recanalization for intracranial symptomatic arterial occlusion in the subacute period without adjunctive stenting.
Case description: Two patients presented with recurrent transient ischemic attacks due to subacute occlusion of intracranial vessels (left MCA M1 and basilar artery). Both had pressor-dependent ischemic symptoms referable to the occluded artery. Sustained angiographic antegrade flow was achieved following angioplasty of our first patient's occluded basilar artery. Excellent angiographic perfusion of our second patient's left hemisphere was achieved following angioplasty of her occluded M1. A Maverick 2 mm × 9 mm balloon was employed in both cases, neither requiring chemical thrombolysis.
Conclusion: Symptoms in both patients abated and they were weaned off pressors within 24 h, underscoring the potential of angioplasty to treat fluctuating, pressor-dependent cerebral ischemia from subacute intracranial arterial occlusion. At most recent follow-up, both patients were neurologically intact, denying any transient neurologic events occurring in the interim.

1. Introduction

Recently, the feasibility of endovascular recanalization for extracranial carotid occlusion has been shown with promising results [1–3]. One small case series [4] and case reports [5–7] have demonstrated successful recanalization of chronically occluded intracranial carotid, MCA and vertebral arteries. However, to our knowledge, intracranial endovascular recanalization of symptomatic intracranial occlusion in the subacute period (beyond the 8 h window) has not been well documented in the literature [8]. Attempts to treat acutely symptomatic severe stenosis or occlusion by EC-IC bypass procedures have proven disappointing [9,10] and most cases are therefore deemed inappropriate for revascularization [11–13]. While the Carotid Occlusion Surgery Study (COSS) may reveal the efficacy of EC-IC bypass for the subset of patients with stage II hemodynamic failure and symptomatic severe stenosis or occlusion [14], an endovascular alternative may prove efficacious for a broader spectrum of patients. Furthermore, this approach addresses the specific problem at hand – the occlusion – allowing for direct correction of hemodynamic insufficiency. We present two unique cases, an in situ mid basilar thrombosis at the site of an atherosclerotic plaque, and an embolic M1 MCA occlusion, which both demonstrated immediate and lasting clinical response to angioplasty beyond the acute period.

2. Case 1

2.1. History and examination

A 64-year-old Caucasian male smoker with type 2 diabetes, hypertension, and congestive heart failure underwent aortic valve replacement and closure of a patent foramen ovale. He had a history additionally notable for prior carotid endarterectomy, coronary artery bypass graft, peripheral vascular disease, and renal transplantation.

On the fifth post-operative day, he suffered three separate 5-min episodes of right-sided upper extremity paresis, diplopia, vertigo, and dysphasia, precipitated by sitting upright. Due to a suspicion of heparin-induced thrombocytopenia earlier in his course, the patient was started on an argatroban drip for presumed transient ischemic attacks. Despite this measure, the patient had two additional episodes in the following 2 days, the second including additional paresis of the right lower extremity. Neurological exam

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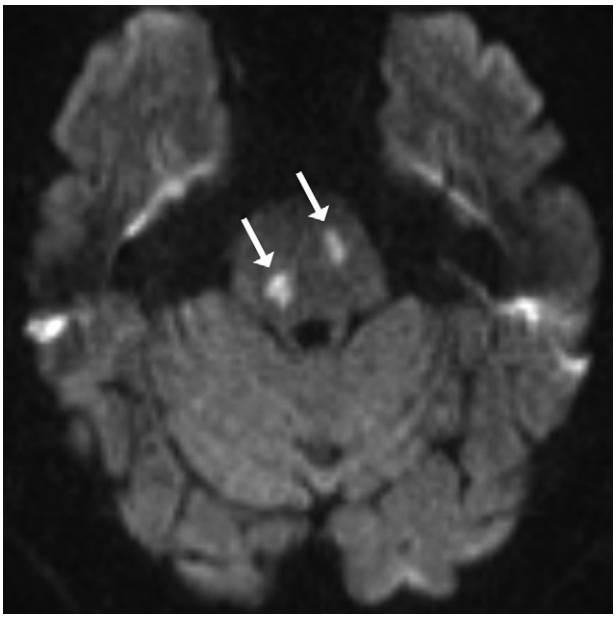

Fig. 1. DWI shows two punctate foci of diffusion restriction consistent with pontine infarcts (arrows).

at this time confirmed dysphasia, a partial left third nerve palsy, and a 4/5 right-sided hemiparesis. Brain MRI showed acute multifocal pontine infarction (Fig. 1). MRA showed severe left V4 and critical mid-basilar artery stenosis, the latter demonstrating a "string sign" suggesting residual patency (Fig. 2). The right vertebral artery ended in the right PICA. MR perfusion imaging revealed compromised perfusion in the vertebrobasilar territory with prolonged MTT in the left pons and cerebellum.

The patient's systolic blood pressure was maintained between 150 and 180 mm Hg with phenylephrine, alleviating his symptoms. Given the patient's initially unstable course despite anticoagulation, the severity of the stenoses and significant MR perfusion abnormality, the risk of subsequent events was deemed extremely high. Angiography was thus performed on the ninth day (from his first TIA) with the intent to angioplasty and/or stent the critically stenosed basilar artery.

2.2. Endovascular treatment

Under general anesthesia, left vertebral artery catheterization was initially performed, confirming severe irregular stenosis of V4 at the PICA origin (Fig. 3). Under magnification, no antegrade flow was visible through an apparently occluded mid-basilar artery. However, delayed retrograde filling of the basilar tip via tenuous left PICA to AICA pial collaterals was appreciated (Fig. 4). The microcatheter was advanced into the basilar artery over a Synchro 2 Soft guidewire and passed easily through the occluded segment. Angiography performed in the distal basilar artery showed the distal aspect of the stenosis located at the level of patent AICA vessels. Antegrade flow through the basilar stenosis was appreciated on angiography via the vertebral artery guide catheter at this time.

The basilar artery was then angioplastied by three inflations of a Maverick 2 mm × 9 mm balloon. Angiography subsequently showed significant improvement in hemodynamic flow through the basilar artery with residual moderate stenosis and a small intimal dissection. Subsequent angioplasty of the left V4 segment was performed very gently (two balloon inflations) due to the risk of PICA Occlusion by dislodged plaque. The degree of V4 stenosis improved modestly from greater than 90% to 70% with a visible

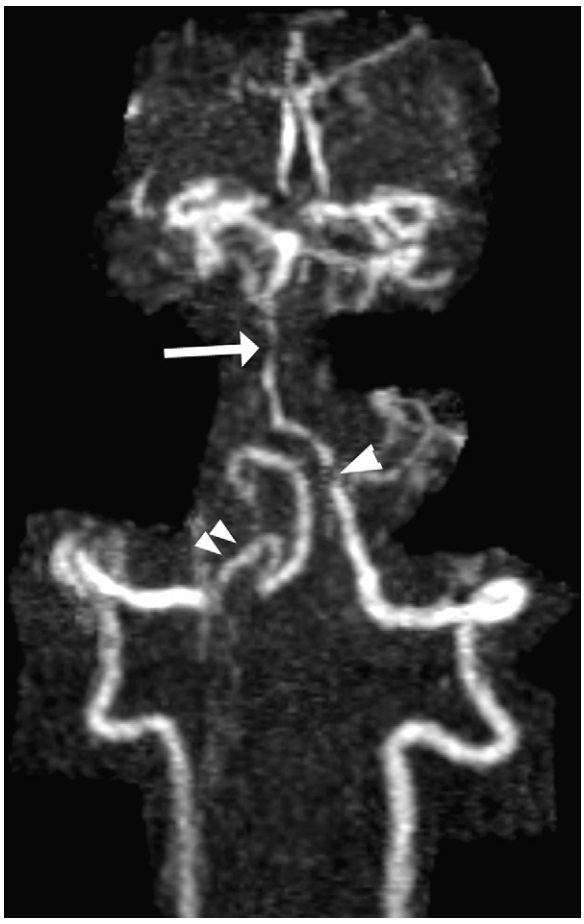

Fig. 2. Contrast enhanced MRA reveals severe stenosis of the left V4 vertebral artery (arrowhead). Focal signal drop-out in the mid basilar artery with patent vessel distally suggests a critical stenosis or "string-sign". Right vertebral artery contributes only to the right PICA (double arrowheads).

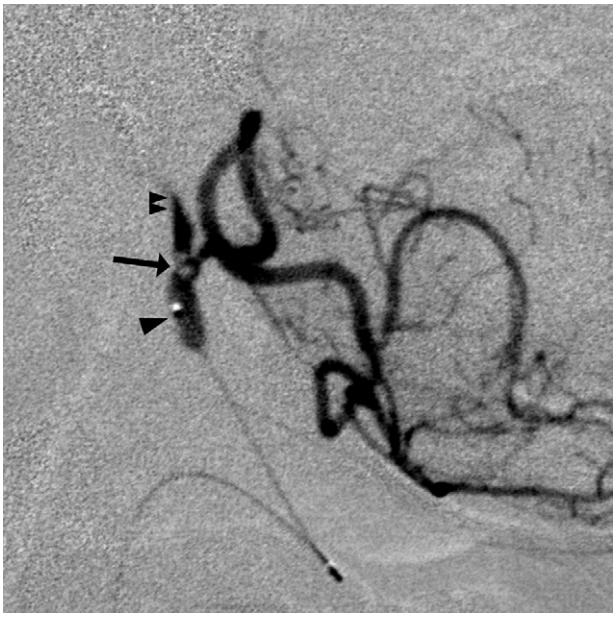

Fig. 3. Microcatheter angiography with catheter tip in the left V4 vertebral artery (arrowhead). Severely stenotic plaque (arrow) causes hold-up of contrast at the vetebrobasilar junction (double arrowheads).

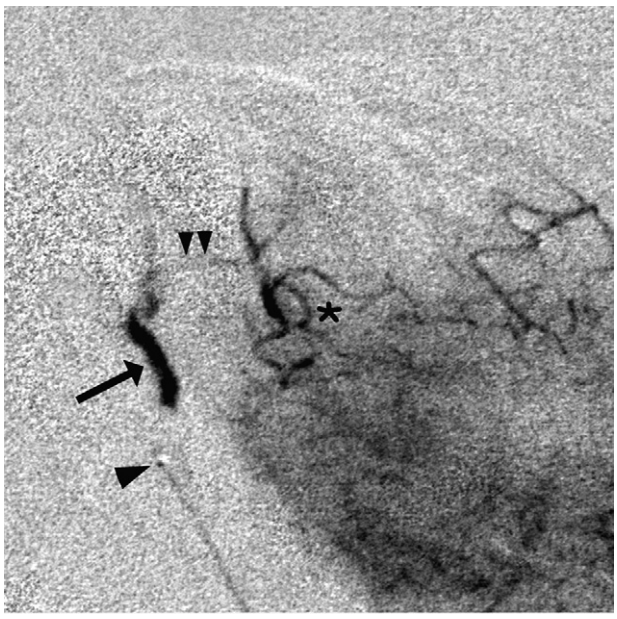

Fig. 4. Later phase of angiography demonstrates mid basilar stasis of contrast (arrow), PICA to AICA pial collaterals (*) and retrograde flow through the Left AICA into the mid basilar artery. Microcatheter tip in the left vertebral artery (arrowhead).

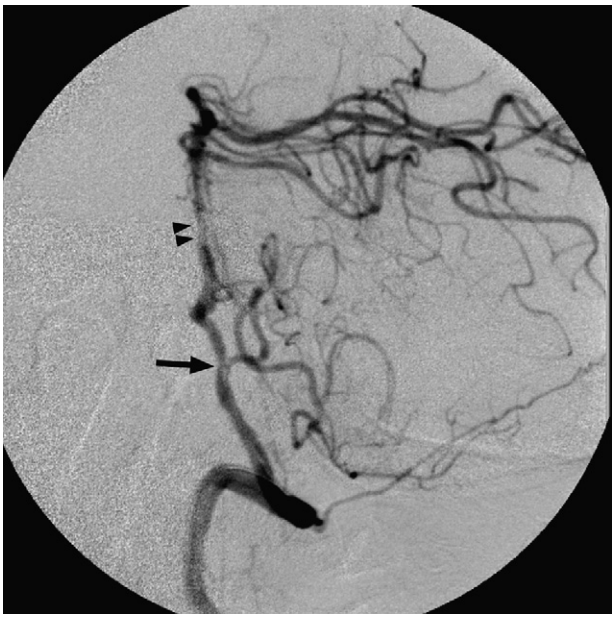

Fig. 5. Post-angioplasty angiography shows moderate residual left V4 (arrow) and mid basilar (arrowheads) stenoses.

increase in flow (Fig. 5). Restoration of normal antegrade vertebrobasilar hemodynamics was reflected by the cessation of pial collateral flow post-angioplasty.

2.3. Post-operative course

The patient had an uneventful recovery from general anesthesia. Systolic blood pressure was maintained between 100 and 120 mm Hg to help prevent reperfusion injury, and the patient was started on aspirin 81 mg and clopidogrel 75 mg daily on the first post-interventional day. MRA performed on the second day following intervention demonstrated a small basilar dissection (Fig. 6A), but overall significantly improved caliber of the basilar and distal left vertebral arteries. MRP demonstrated normalized, symmetrical mean transit time in the posterior fossa. After 1 month, the patient's aspirin dose was increased to 325 mg and clopidogrel was ceased. Compared to MRA performed on the second post-interventional day (Fig. 6A), 4 month follow-up MRA did not demonstrate significant restenosis (Fig. 6B). At most recent follow-up (5 months post-intervention), the patient was neurologically intact, denying the occurrence of any transient neurologic events in the interim.

3. Case 2

3.1. History and examination

An 80-year-old nonsmoking Caucasian female with a history of hypertension and dyslipidemia awoke with right-sided hemiparesis and aphasia and presented to our institution 6 h later

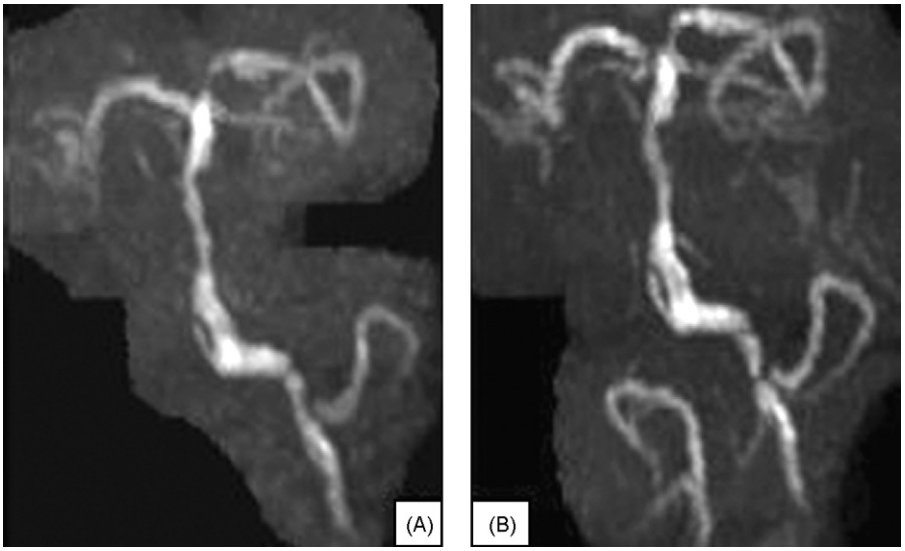

Fig. 6. MRA performed on the second post-interventional day (A) demonstrates residual severe focal stenosis of the V4 segment of the left vertebral artery just after the origin of the left PICA, a small focal dissection at the vertebrobasilar junction, and residual moderate to severe stenosis of the mid-basilar artery. 4 months follow-up MRA (B) does not demonstrate significant restenosis.

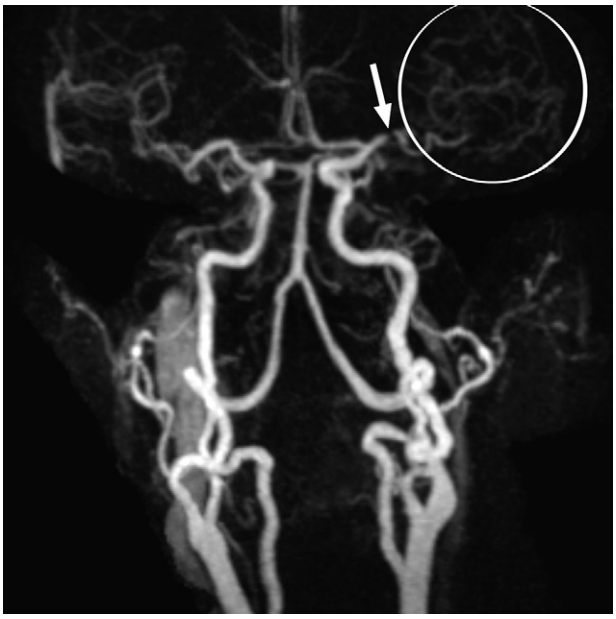

Fig. 7. DWI shows acute infarcts in the left deep white matter watershed distribution.

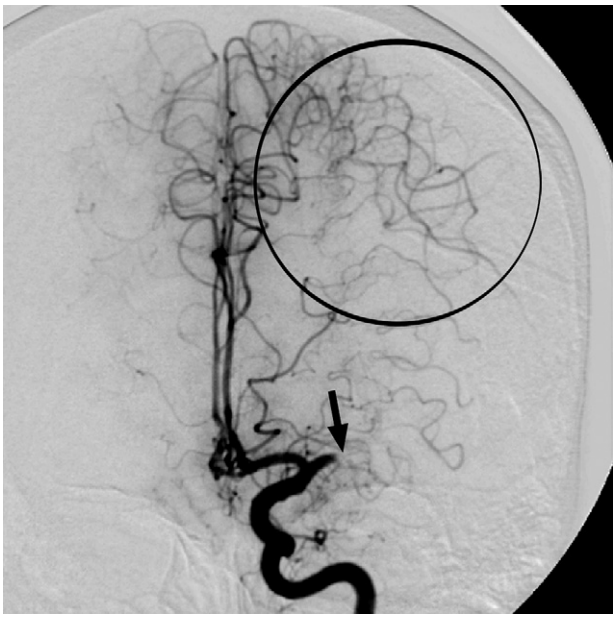

Fig. 9. Angiography confirms occlusion of the proximal left M1 MCA. Note pial collaterals extending from the right ACA territory (circle).

that morning. Initial CT demonstrated hypoattenuation in the left frontal periventricular and subcortical white matter with MR diffusion restriction confirming a chain of infarcts in a left MCA watershed distribution (Fig. 7). MRA demonstrated a short segmental occlusion of the left M1 MCA with distal vascular enhancement reflecting collateral flow (Fig. 8). Symptoms waxed and waned over the course of the day with best neurological exam demonstrating dysnomia and 4+/5 right-sided hemiparesis. As she presented outside the therapeutic window for acute thrombolysis, she was treated medically with aspirin 325 mg and a 300 mg loading dose of clopidogrel. After a transient episode of global aphasia very late in her first admission day, the patient was kept flat with permissive hypertension to systolic pressures of 170 mm Hg. Given these significant deficits and the need for hypertensive therapy to control her symptoms, she was transferred to the endovascular suite that evening (16 h after awakening) for revascularization.

3.2. Endovascular treatment

Under general anesthesia, angiography demonstrated an occluded left M1 segment (Fig. 9). This was traversed with a Maverick 2 mm × 9 mm balloon microcatheter over an Agility-soft microguidewire beyond the M1 occlusion confirming distal patency. The balloon was then drawn back to the point of occlusion and angioplasty was performed. Subsequent angiography

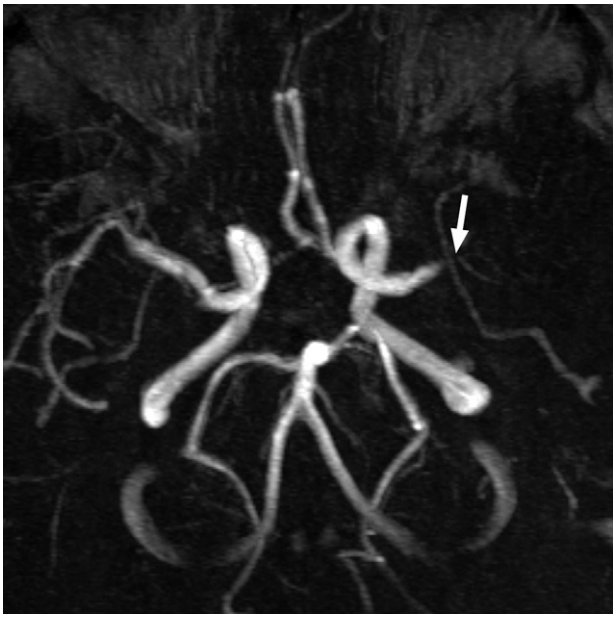

Fig. 8. Contrast enhanced MRA reveals focal occlusion of the left M1 MCA. Note enhancement of patent left MCA branches due to pial collateral flow (circle).

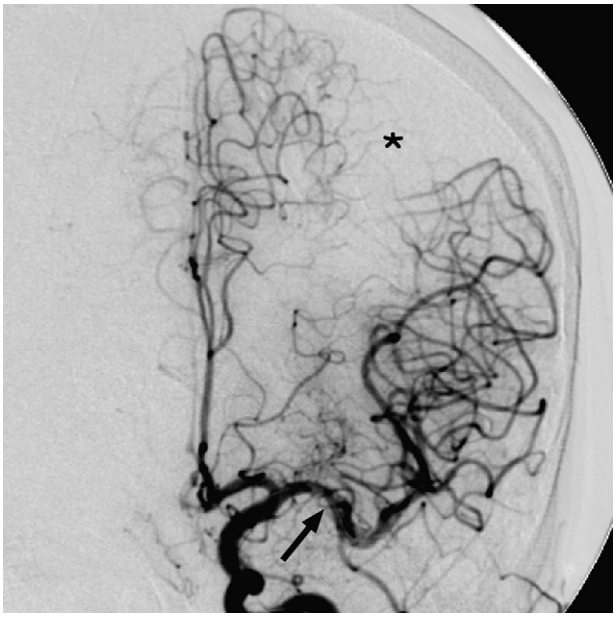

Fig. 10. Post-angioplasty angiography shows near complete patency of the M1 MCA. Right ACA pial collaterals are no longer prominent (*) due to restored antegrade flow in the left MCA territory.

demonstrated restored antegrade flow to the MCA territory via the M1 segment. Additional angioplasty led to resolution of residual irregular stenosis in the proximal M1 segment with a final angiogram demonstrating overall excellent perfusion to the patient's left hemisphere (Fig. 10).

3.3. Post-operative course

The patient had an uneventful recovery from general anesthesia with an unremarkable post-procedural CT scan of her brain and a repeat MRI demonstrating no new foci of diffusion restriction. Post-revascularization, her blood pressure was maintained between 120 and 140 mm Hg via labetalol drip to help prevent reperfusion injury. Remarkably, the patient's speech and strength improved to her baseline by the first post-procedural day. Prior to discharge 5 days later, her statin dose was increased, and she was sent home on daily aspirin 81 mg and clopidogrel 75 mg for 1 month, with subsequent cessation of clopidogrel and increase of aspirin dose to 325 mg daily. At most recent follow-up (3 months post-intervention), the patient was neurologically intact, denying the occurrence of any transient neurologic events in the interim.

4. Discussion

Mechanistically, the combined effects of thromboembolism and impaired hemodynamic flow from atherosclerotic occlusive disease may lead to ischemic stroke [15]. To improve flow, extracranial–intracranial arterial bypass has been employed, though overall results have proven disappointing in randomized trials [9,10]. In contrast, endovascular recanalization of occluded cervical carotid arteries has been used with impressive results [1–3]. While recanalization of occluded intracranial arteries has been employed successfully for acute intracranial occlusion [16–18], MR perfusion/diffusion mismatch may become one reliable predictor of the potential for tissue salvage in lieu of time from symptom onset [19]. In our first case, perfusion-imaging findings indicated a significant volume of tissue at risk, encouraging intervention. In both cases, standard conservative therapy employing anti-platelet agents and anticoagulants was ineffective, with symptoms ameliorated only by IV vasopressor therapy. We felt that the degree of hypoperfusion and the severity of the stenosis (e.g. occlusion) merited revascularization, although it was possible in both cases that collaterals might ultimately have developed that would allow the weaning of pressors.

Like Ahuja et al. [20] and Nakatsuka et al. [21], in our first case, we avoided aggressive dilatation of the basilar artery owing to its relatively deficient medial and adventitial layers. Likewise, only a modest angioplasty of the left V4 segment was undertaken to minimize the risk of a PICA occlusion.

In our cases, we were compelled to attempt endovascular recanalization beyond the acute phase due to unstable symptoms clearly related to cerebral hypoperfusion, with perfusion imaging showing significant volumes of tissue at risk. Two case reports have demonstrated successful recanalization in the subacute phase. In one, the patient's symptoms abated 2 days prior to angioplasty and stenting of his occluded proximal intracranial carotid artery was performed as a prophylactic measure due to significant contralateral ICA stenosis [22]. In the other case, a Gianturco-Roubin-2 coronary stent was required after unsuccessful attempts at chemical thrombolysis (750,000 U of urokinase) and angioplasty of a lower basilar occlusion causing recurrent, transient locked in syndrome [8]. The patient recovered neurologically but died as a result of cardiogenic shock and sepsis within 1 month.

In our first case, we initially diagnosed a critical intracranial stenosis and proceeded on the basis of a WASID-based estimation of a 22.5% annual rate of stroke for 75–99% stenosis [23]. At the time of angiography, however, the lesion was occlusive and although the reported mortality rate of untreated acute basilar occlusion exceeds 70% [24–26], the natural history of a subacute lesion is less certain. Reports by Brandt et al. [27] and Cross et al. [28] suggest that proximal or midbasilar occlusion portend a poorer prognosis as they are more likely, as in our case, to be thrombotic as opposed to embolic, the former being less amenable to lysis due to a fixed underlying lesion. This risk of recurrence may be reduced by the deployment of a stent, as performed by Phatouros et al. [8]. Given recent work by Levy et al. demonstrating decreased morbidity following staged stenting in lieu of direct or conventional stenting for severe atherosclerotic vertebrobasilar disease [29], we planned only to stent in a subsequent procedure if significant restenosis occurred. However, like Mori et al. [4], our case demonstrated sufficient and maintained recanalization following angioplasty without adjunctive stenting.

Most cases of successful angioplasty for non-occlusive intracranial stenosis often include adjunctive stent placement [11–13,30]. Additionally, the literature on basilar occlusion typically addresses the efficacy of intra-arterial thrombolysis in the acute setting [31,32]. In this report, we demonstrate adequate, sustained antegrade flow following angioplasty without intra-arterial thrombolysis or adjunctive stenting for symptomatic, subacute complete thrombosis of intracranial vessels, one in the posterior circulation (basilar) and the other in the anterior circulation (M1). Future attempts may employ the MERCI device or further cultivate cerebral protection techniques [6,17,18,22,33–36]. We hope that these cases will stimulate debate in the use of endovascular therapy for the restoration of patency of subacutely occluded intracranial vessels.

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