Middle Cerebral Artery Stenosis: Endovascular and Surgical Options

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ABSTRACT

Atherosclerotic middle cerebral artery stenosis is a rare but potentially devastating cause of cerebral ischemia and stroke. While medical management remains the mainstay for stroke prevention, surgical and/or endovascular intervention is indicated in selected patients. This article reviews the role of surgery and endovascular techniques in the treatment of middle cerebral artery stenosis based on its natural history, pathophysiology, and prognosis when treated medically.

KEYWORDS: Middle cerebral artery stenosis, surgery, bypass, stenting, endovascular, angioplasty

Atherosclerotic disease of the middle cerebral artery (MCA) is a relatively rare condition, occurring in only 7 to 8% of patients presenting with stroke in the MCA distribution.¹⁻³ Subsequent MCA stenosis or occlusion may lead to transient ischemic attacks (TIAs) or frank stroke, thus prompting the need for intervention. This article reviews the surgical and endovascular management options for atherosclerotic MCA stenosis based on its natural history, pathophysiology, and prognosis after medical treatment.

NATURAL HISTORY

Intracranial atherosclerotic disease is usually found in the internal carotid artery (ICA) siphon, the

main trunk of the MCA, the distal vertebral artery, the vertebrobasilar junction, and/or the midportion of the basilar artery. The basilar artery is the most common site, harboring 8% of all brachiocephalic lesions.⁴ In the anterior circulation, intracavernous lesions predominate, followed by petrous and supraclinoid lesions.⁵ Although intracranial stenosis is consistently associated with a high stroke rate, its natural history has not been studied as well as that of extracranial stenoses.^{6,7} However, during an average follow-up of 3.9 years in a study of patients with intracranial arteriosclerotic disease, 12.1% of patients experienced TIAs and 15.2% had a stroke. The event rate was 27.3%.

Patients with intracranial disease are more likely to present with an unheralded stroke.⁸ The risk of stroke in patients with intracranial stenosis is

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high,^{9–11} among which the stroke risk associated with MCA stenosis is at least 8% per year.^{10,12} The incidence of stroke in patients with MCA stenosis is higher than that in patients with extracranial carotid stenosis.^{8,12}

In the extracranial-intracranial (EC-IC) Bypass Trial, the annual ipsilateral stroke rate of patients with symptomatic MCA stenosis randomized to medical therapy was 7.8% and their overall stroke rate was 9.5%.^{10,12} The most common presentation was a stroke without a warning TIA; only about a third of patients had a warning TIA before stroke.^{10,12} In another study, patients with TIAs had a high incidence of stroke within a few months of symptom onset.^{13,14} In the EC-IC Bypass Trial, patients with severe MCA stenosis were among those with the worst outcomes.

Patients with symptomatic intracranial atherosclerotic disease tend to be younger than those with extracranial carotid disease.¹⁵ Compared with Caucasians, the incidence of ischemic stroke related to MCA stenosis is higher among Asians, Hispanics, Blacks, and especially in the Chinese population.^{6,8,13,16–18} In the EC-IC Bypass study, 58% of the enrolled Asian population were also eligible for the MCA atherosclerosis study compared with 34% of the Blacks and 18% of the Caucasians.^{10,12}

PATHOPHYSIOLOGY

MCA occlusive disease may lead to an ischemic event via three mechanisms: (1) deep lacunar infarcts that develop when the exiting branch of the lenticulostriate perforating artery is trapped within the thromboatheroma; (2) development of atheromatous ulceration with thrombosis and subsequent distal embolization; and (3) hemispheric hypoperfusion caused by significant MCA obstruction and inadequate collateralization.^{1,6,19–22} None of these mechanisms are mutually exclusive, and a given lesion can cause neurologic sequelae by any or all three. In fact, some researchers have postulated that hypoperfusion may potentiate the effect of

distal emboli. Hypoperfusion decreases blood flow and pressure, precluding adequate clearance of distal emboli in an already poorly perfused area.⁶

Unfortunately, scant literature has addressed the relative frequency with which each of these three mechanisms is responsible for subsequent neurologic sequelae in MCA stenosis. In 22 patients with MCA stenosis and acute strokes, 46% were of the deep lacunar type, whereas the remaining 54% were of the distal hemispheric type.¹⁹ The latter group was not divided into embolic or hypoperfusion subgroups. Both Segura and colleagues²⁰ and Wong and associates⁶ used transcranial Doppler ultrasonography (TCD) to evaluate symptomatic MCA stenosis patients: 36% and 33% of patients, respectively, demonstrated evidence of microemboli distal to the stenosis. Finally, Mohr et al¹ evaluated the data from Hinton and associates²² and concluded that 13 of 16 patients with symptomatic MCA stenosis demonstrated clinical evidence of hemodynamic insufficiency. Based on these few studies, it is difficult to state that one mechanism is the most common cause of neurologic dysfunction in MCA stenosis. However, it appears safe to conclude that each mechanism is a significant contributor to the development of stroke.

The mechanism by which MCA stenosis becomes symptomatic is an important factor in choosing a management strategy. Typically, anti-thrombotic or antiplatelet agents are the initial treatment of choice for patients with symptomatic MCA stenosis. These medications are well suited for the treatment of embolic disease and perhaps thrombotic occlusion of lenticulostriate perforators. However, it is difficult to imagine how they can effectively treat hemodynamic insufficiency related to atherosclerosis.

REVIEW OF MEDICAL MANAGEMENT

The current paradigm for the management of both symptomatic and asymptomatic MCA stenosis suggests that most patients should be treated medically

with either antiplatelet or anticoagulant agents at first diagnosis. Several studies have evaluated the prognosis of medically treated MCA stenosis. Of 50 patients with asymptomatic MCA stenosis, 42 of whom were on antiplatelet or anticoagulant therapy, none had suffered an acute neurologic event at their 1-year follow-up.²³ These findings suggest that asymptomatic MCA stenosis is a relatively benign disease that should be managed medically.

In studies on the prognosis of medically treated symptomatic MCA stenosis, 14 to 33% of patients subsequently experienced a neurological event in the distribution of the MCA (Table 1).^{6,10,17,24,25} Wong and colleagues⁶ and Arenillas and associates¹⁷ followed their medically treated symptomatic stenoses clinically and with TCD. In the former study, only 9% of patients demonstrated stenosis progression on TCD compared with 32% in the latter. Both studies found that progression of stenosis was associated with a 35 to 75% risk of a subsequent adverse neurological event. Arenillas and associates also found that patients on anticoagulants had a 7% likelihood of experiencing a subsequent event, whereas the risk of those on antiplatelet agents was 39%.¹⁷ The finding

suggests that anticoagulation therapy may be superior to antiplatelet agents.

In a large prospective study by the EC-IC Bypass Study Group, patients with radiographically demonstrated anterior circulation stenosis who suffered a sentinel neurologic event (TIA or cerebrovascular accident) in a corresponding distribution were randomized to medical or surgical management.¹⁰ In patients with MCA stenosis and occlusion, there was no significant difference in the subsequent development of neurologic events after medical (23%) or surgical (29%) treatment. The study group concluded that patients with symptomatic intracranial stenoses (including MCA stenosis) should be treated medically.

These studies suggest that most asymptomatic and symptomatic patients with MCA stenosis should be treated medically. However, medical management alone is insufficient in three subsets of symptomatic patients: (1) those who develop neurologic events while on medical therapy; (2) those who develop radiographically demonstrable, progressively worsening MCA stenosis that significantly increases the likelihood of a subsequent neurologic event; and (3) those with hypoperfusion related to MCA stenosis in whom antithrombotic

Table 1 Summary of Studies Evaluating Medical Management of Symptomatic MCA Stenosis

Reference	No. Patients	F/U (yrs)	No Further Neurological Events (%)	Radiographic Progression of Stenosis (%)	Comments
Arenillas et al, 2001 ¹⁷	40	2.2	80	32.5	7% of patients on anticoagulants had further events, 28% of those on antiplatelet therapy; radiographic progression associated with 46% event rate
Becker et al, 1996 ²⁴	8	4.2	100	Not done	Young patients only (32 to 56 years)
Corston et al, 1984 ²⁵	21	6.8	67	Not done	Small subset of patients including 2/7 (40%) patients who had neurological events were not medically treated
EC-IC Study Group, 1985 ¹⁰	138	4.7	77	Not done	Results for CVAs only (no TIAs); subgroup analysis of patients with severe MCA stenosis or occlusion
Wong et al, 2001 ⁶	143	0.5	86	9.6	Radiographic progression associated with 38.5% event rate

F/U, follow-up; EC-IC, extracranial-intracranial; CVA, cerebrovascular accident; TIA, transient ischemic attack; MCA, middle cerebral artery.

medications are unlikely to provide protection against potential neurologic sequelae. Surgical or endovascular intervention is usually indicated for these three subsets of patients.

SURGICAL MANAGEMENT

Indications

The role of surgery in the treatment of symptomatic MCA stenosis has changed significantly since publication of the results of the EC-IC Bypass Trial and with the continued evolution of endovascular techniques. EC-IC bypass was once a common treatment for multiple conditions, including MCA stenosis. Consequently, the technique became well developed and has a long track record. In experienced hands, it is associated with a high patency rate and low complication rate. In the EC-IC Bypass Trial, however, surgery provided no benefit over medical therapy.¹⁰ Thereafter, the use of bypass to manage symptomatic MCA stenosis declined dramatically. Nonetheless, bypass remains an appealing and effective treatment option for a subset of patients with symptomatic MCA stenosis. The key to its success lies in identifying the appropriate indications for surgery.

The EC-IC bypass group included all patients with symptomatic MCA stenosis regardless of the pathophysiology underlying each patient's ischemia (e.g., distal embolization, thrombotic perforator occlusion, or hypoperfusion). That is, patients with hemodynamic insufficiency were not analyzed separately. Intuitively, this condition is unlikely to benefit significantly from medications alone. Consequently, revascularization is indicated when hemodynamic insufficiency is a contributing or the sole factor underlying MCA stenosis symptoms refractory to medical management.²⁶⁻²⁹ Asymptomatic patients should not be considered for surgery regardless of etiology.

Patients in whom hypoperfusion is suspected as the sole reason for symptoms should be considered

for early revascularization at or near the time of diagnosis; a brief trial of medical management may be warranted. Conversely, those in whom thromboembolism is thought to be the source of symptoms should be managed medically. Patients with a mixed picture should undergo a trial of medical management because hypoperfusion can potentiate the effects of embolization. If the emboli are prevented medically, symptoms may resolve. Patients in the latter subgroup who fail medical management should be considered for revascularization. Finally, it is unlikely that surgery is indicated when medications fail in patients with MCA stenosis whose symptoms are entirely caused by thromboembolism without hypoperfusion. Hypothetically, it seems that bypass will not benefit these patients unless proximal occlusion is performed to eliminate the source of embolism. This situation appears ideal for endovascular management.

Evaluation of Hemodynamic Insufficiency

Various modalities are available to determine the presence of hemodynamic failure. We use computed tomographic (CT) perfusion with and without acetazolamide.³⁰ Other options for evaluation are positron emission tomography (PET), stable xenon CT, and single photon emission CT (SPECT). Findings suggestive of hemodynamic insufficiency are relative hypoperfusion lateralized to the area of the stenosis and symptoms, prolonged mean transit time in the territory of the stenosis, and worsened perfusion on provocative testing with an acetazolamide challenge. Each technique identifies regions of the brain with a compromised ability to increase blood flow to meet metabolic demand, otherwise known as stage I hemodynamic failure. Stage II hemodynamic failure, which is defined as increased oxygen extraction, can only be identified on PET.

Four-vessel cerebral angiography is imperative for all patients with MCA stenosis for whom revascularization is being considered. Angiography not only allows evaluation of the stenotic lesion but also provides a gross estimate of collateralization in

the MCA distribution. Furthermore, the superficial temporal artery (STA) can be evaluated should revascularization be necessary.

Selection of Surgical Technique

Once hemodynamic insufficiency has been related to MCA stenosis, the surgeon must choose the surgical revascularization technique. Options include high-flow EC-IC bypass procedures using vein or arterial graft from the proximal ICA to the M1 or M2 segment of the MCA, STA-MCA bypass, or nonanastomotic bypasses, which include encephaloduroarteriomyosynangiosis (EDAMS) and encephaloduroarteriosynangiosis (EDAS). The technical details of each procedure are discussed elsewhere. Any sutured bypass is a technically demanding procedure. Before it is attempted in the operating room, the bypass procedure should be practiced in a microvascular laboratory until a high level of proficiency has been achieved.

Technologies aimed at evaluating patient-specific arterial hemodynamic characteristics are on the horizon.^{31,32} At present, however, there is no objective method by which to determine the type of bypass needed by a given patient. Rather, the surgeon must rely on subjective evaluation of multiple factors: the nature and severity of the patient's symptoms, the degree of collateralization and caliber of recipient vessels on angiography, and the extent to which hemodynamic insufficiency is demonstrated on functional imaging. Experience is vital in making such subjective decisions.

In most patients with MCA stenosis, the procedure selected for surgical revascularization is the STA-MCA bypass.^{33,34} There are, however, exceptions. Patients with frequent and profound symptoms with evidence of marked insufficiency and few collaterals may benefit from a high-flow bypass. In the case of poor arterial recipients for direct anastomotic bypass with a moyamoya-like pattern, EDAS or EDAMS is indicated. When prior surgery or a small caliber renders the ipsilateral STA unavailable, a bonnet bypass can be performed.³⁵ In this procedure, the contralateral

STA is sutured to an intervening vein or arterial graft to span the vertex to the ipsilateral hemisphere.

Surgical Outcomes

Many studies support the feasibility and efficacy of surgical revascularization. Studies evaluating intracranial arterial occlusive disease without respect to the specific vessel affected or degree of hypoperfusion have shown that 78 to 90% of patients who fail medical therapy and subsequently undergo anastomotic revascularization (mostly STA-MCA) have no further ischemic events.^{36–38} The rate of major morbidity in this group has ranged from 3 to 5% while bypass patency rates have ranged from 90 to 97%.^{36–38}

Most data on nonanastomotic bypasses (e.g., EDAS, EDAMS) are from the literature on moyamoya disease. This may be a less morbid subgroup, but studies suggest that nonanastomotic techniques are clinically successful in 75 to 90% of patients and neovascularity can be seen on angiography in 84 to 92% of cases.^{39–43} Time, however, is needed for the neovascularization to mature. Therefore, short-term neurologic events can occur.³⁹

Andrews and colleagues⁴⁴ reported the only large series that specifically evaluated EC-IC bypass in 65 patients with MCA stenosis or occlusion. After bypass, 88% of their patients suffered no further neurologic events. Sixty-three patients underwent STA-MCA bypass while occipital-MCA bypasses were performed in the remaining two. The surgical morbidity and mortality rates were 9.2% and 1.5%, respectively, and the rate of permanent morbidity was 3.0%. At late follow-up the bypass patency rate was 100%. The investigated cohort was treated between 1972 and 1983. Consequently, none of the patients were evaluated with functional cerebral blood flow (CBF) studies.

Recently, several studies have evaluated the results of bypass in patients with intracranial arterial occlusive disease in whom hemodynamic compromise was demonstrated on preoperative CBF (Table 2) studies.^{26,27,45–49} The type and extent of

Table 2 Summary of CBF Studies Evaluating Surgical Outcomes of Patients with Intracranial Arterial Stenosis

Reference	No. Patients	F/U (yrs)	No Further Neurological Events after Bypass (%)	Graft Patency (%)	Morbidity (%)	Postop CBF	Comments
Anderson et al, 1992 ⁴⁹	13	1.2	100	100	NA	All had increased CBF and reserve	Focused on CBF studies; few clinical data
Holzschuh et al, 1991 ⁴⁷	18	5.4	89	94	NA	No change in CBF; all had increased reserve	Focused on CBF studies; few clinical data
McCormick et al, 1991 ⁴⁶	19	1.2	72.2	94	27	Not done	Patients only had CBF preop; reserve not evaluated
Powers et al, 1989 ⁴⁸	29	1	87.5	NA	7	Only performed in 3 patients	<i>Criticism:</i> Case control study showed no benefit of surgery; however, only 10 patients had absent reserve on preop CBF studies
Schick et al, 1996 ²⁷	47	5.6	78	79	27	75% had increased reserve	<i>Criticism:</i> Only 36% had altered reserve preop; many patients had evidence of embolic source of symptoms
Schmiedek et al, 1994 ²⁶	28	3	82	96	14	No change in CBF; all had increased reserve	NA
Vorstrup et al, 1986 ⁴⁵	18	0.33	94	87	NA	89% had no change in CBF; all had increased reserve	Short follow-up of immediate CBF outcomes; only 2 patients had preop vascular steal

CBF, cerebral blood flow; MCA, middle cerebral artery; F/U, follow-up; NA, not available.

blood-flow evaluations vary considerably from study to study as do the lengths of follow-up. However, 72 to 94% of patients suffered no further neurological events after bypass, and the rate of surgical morbidity ranged from 7 to 27%. As demonstrated by the presence of vascular steal when acetazolamide was administered, a key predictor of clinical success was the absence of cerebrovascular reserve on CBF studies. Bypass patency rates with variable follow-up times ranged from 79 to 100%.

A subset of this same group of studies compared pre- and postoperative blood flow in patients who underwent a bypass (Table 1). Some studies found increases in both postoperative CBF and cerebrovascular reserve in most or in a specific subset of patients.^{27,45,49} Others demonstrated no change in CBF but found a significant increase in cerebrovascular reserve.^{26,45,47} Although the results of postoperative blood flow studies vary across studies, some element of cerebrovascular dynamics improved after bypass. Again, the common thread appeared to be improvement in cerebrovascular reserve. Parenthetically, the authors of one study concluded that preoperative hemodynamic evaluation is not helpful in identifying patients in need of bypass,⁴⁸ and a second concluded that "few patients benefit . . . from EC/IC bypass."²⁷ Their comments are beyond the scope of this discussion, but Schmiedek and colleagues pointed out the weaknesses of both studies and refuted the authors' conclusions.^{26,50,51}

Based on the results of these studies and on anecdotal experience, we believe that candidates for bypass are patients with medically refractory, symptomatic MCA stenosis, in whom blood flow studies demonstrate evidence of altered vascular reserve as suggested by the presence of vascular steal. In experienced hands, bypass effectively reduces the risk of further ischemic events with an acceptable risk of surgical complications in this highly morbid subgroup. Ideally, a prospective, randomized trial should be conducted to validate the role of bypass in patients with intracranial arterial occlusive disease.

Future Considerations

Surgical revascularization has been the mainstay of treatment for medically refractory MCA stenosis, but the appeal of endovascular techniques is increasing. As technology evolves and endovascular efficacy increases, this modality will likely become the treatment of choice for patients who fail medical therapy. Indeed, we routinely refer our MCA stenosis patients in need of intervention to the endovascular service for treatment. We reserve bypass for those who fail or are not amenable to endovascular treatment.

ENDOVASCULAR MANAGEMENT

Indications

The goal of patient selection is to identify the population that will most benefit from the endovascular treatment of M1 stenosis with an acceptable complication rate compared with the risk of the stroke. Reports on the efficacy of endovascular therapy for this disease have identified the following criteria⁵²⁻⁵⁷: (1) patients with recurrent low-flow TIAs or nondisabling ischemic stroke in the MCA distribution; (2) disease refractory to medical therapy (i.e., persistence despite a therapeutic dosage of antiplatelet or anticoagulation therapy); (3) corresponding M1 stenosis equivalent to a 50% or greater reduction in diameter on angiography⁵⁶; (4) significant perfusion problems in MCA regions as evidenced by decreased perfusion reserve on SPECT; and (5) infarction at the border zone of the MCA region considered related to hemodynamic insufficiency.⁵⁷

The criteria used to exclude patients from studies of endovascular treatment of MCA stenosis are as follows: (1) presence of significant atherosclerotic lesions in the arteries proximal to the MCA; (2) emboligenic heart disease; (3) coexistent severe stenosis or occlusion of M2 or M3; (4) stroke within 6 weeks; and (5) total occlusive disease.

Additional exclusion criteria are the presence of a tumor or arteriovenous malformation, severe disability related to stroke or dementia, coexistent ipsilateral ICA stenosis, moyamoya disease, or vasculitis.⁵⁶

Classification

In the endovascular treatment of MCA disease, several factors determine technical and clinical outcomes. Jiang et al studied stenting of the MCA for atherosclerotic disease and proposed that (1) technical success relates to access, (2) the risk of complications and restenosis relates to the morphology of the lesion, and (3) the risk of occlusion of larger branches is related to the location of the lesion. Based on these factors, the authors introduced the LMA (location, morphology, and access) classification for assessing M1 lesions (Table 3).⁵⁶

Lesions are divided into seven groups based on their location: type A, prebifurcation lesion; type B, postbifurcation lesion; type C, lesion across the nonstenotic ostium of its branch; type D, lesion across a stenotic ostium of its branch; type E, ostium lesions of a branch alone; type F, combinations of lesions of the prebifurcation and its small branch ostium; and type N, nonbifurcation lesions.

The morphological classification was based on a system proposed by Mori and colleagues⁵⁴ in their series of patients treated with balloon angioplasty for stenotic disease of the MCA. The researchers subdivided their patients into three groups based on the angiographic characteristics of their lesions. Type A were short (5 mm or less), concentric or moderately eccentric lesions that were less than totally occlusive. Type B were tubular (5 to 10 mm), extremely eccentric or totally occluded lesions less than 3 months old. Type C were diffuse (more than 10 mm), extremely angulated (> 90 degrees) lesions with excessive tortuosity of the proximal segment or totally occluded lesions, and 3 months old or older.

Access was measured as mild-to-moderate tortuosity and smooth access (Group I); severe

tortuosity, irregular arterial wall, or both (Group II); or excessively severe tortuosity (Group III).

Balloon Angioplasty

The use of percutaneous balloon angioplasty for the treatment of atherosclerotic arterial stenosis has gained popularity.⁵⁸ As early as 1980, the technique was proposed as a therapeutic approach for symptomatic patients with intracranial arterial stenosis.⁵⁹ It is a promising treatment for patients with ongoing cerebral ischemic events despite standard medical therapy.⁵²⁻⁵⁴ See Figure 1.

Typically, patients are treated via a transfemoral approach under general anesthesia. Heparinization is achieved by administering a bolus dose and is not reversed after treatment. A microballoon is inserted coaxially along a microguidewire and can be introduced primarily or after a microcatheter exchange. The diameter of the balloon ranges from 2 to 2.5 mm, and its length ranges from 10 to 13 mm. The balloon is inflated slowly to its nominal pressure. After percutaneous transluminal angioplasty (PTA), selective angiography is performed to determine the degree of dilatation and whether dissection has occurred and to evaluate the intracranial circulation for signs of distal embolization.

In a review of their 9-year experience treating intracranial stenosis with balloon angioplasty, Connors and Wojak described the evolution of the technique for intracranial stenting.⁶⁰ Their study was divided into how patients were treated early, in the middle, and currently in their experience. They highlighted the rate and extent of balloon expansion as key factors that changed during their experience. Initially, the balloon expanded rapidly and its size approximated that of the vessel, first being smaller, then being slightly oversized. They then began to use undersized balloons, which were inflated slowly over several minutes. The incidence of dissection decreased from 50% and 75% in the early and middle part of their experience to 14% in their current subgroups. Extremely slow balloon inflation

Table 3 Location, Morphology, and Access Classification of MCA Disease⁵⁶

Lesion Type	Location	Classification	Morphology	Classification	Access
A	Prebifurcation	A	< 5 mm long, concentric or moderately eccentric, smooth stenosis	I	Mild-to-moderate tortuosity, smooth access
B	Postbifurcation	B	5–10 mm long, extremely eccentric, or angulated (> 45%), or irregular stenosis, or total occlusion (< 3 months old)	II	Severe tortuosity, irregular arterial wall, or both
C	Lesion across nonstenotic ostium of its branch	C	10 mm long, extremely angulated (> 90%) stenosis, or total occlusion (> 3 months old), or with a number of neovasculatures around	III	Excessively severe tortuosity
D	Lesion across stenotic ostium of its branch		N/A		N/A
E	Ostium lesion of branch alone		N/A		N/A
F	Combined lesions of prebifurcation and its small branch ostium		N/A		N/A
N	Nonbifurcation lesion				

MCA, middle cerebral artery; N/A, not applicable.

Figure 1 (A) Admission left internal carotid artery (ICA) anteroposterior (AP) angiogram in a 54-year-old man who experienced the acute onset of aphasia caused by thrombosis of M1 atherosclerotic stenosis. (B) Immediate post-procedural left ICA AP angiogram after intra-arterial administration of t-PA and angioplasty shows partial restoration of flow. Note the distal filling of the middle cerebral artery. The patient's symptoms resolved immediately after the procedure. (C) Six-month follow-up computed tomographic angiogram confirms complete resolution of the left M1 stenosis and restoration of flow.

combined with undersized balloons decreased intimal damage, acute platelet/thrombus deposition, and acute closure.

Technical success may be defined as 50% or less residual stenosis.^{55,61} Angiographic results may be suboptimal, but overdilatation is a danger when performing angioplasty of intracranial vessels. Underdilatation prevents potential vessel rupture, and an angiographic cure is unnecessary for clinical success.^{58,60}

Many series have analyzed the safety and efficacy of angioplasty of the MCA, although the number of patients has usually been small.⁵²⁻⁵⁵ The series by Mori et al⁵⁴ included all intracranial vessels. The rates of technical success were 92%, 86%, and 33%, respectively, for Type A, B, and C lesions. The risk of fatal or nonfatal ischemic stroke

or of requiring ipsilateral bypass surgery was 8%, 26%, and 87%, respectively, in these three groups. At the 1-year follow-up, the rate of recurrent stenosis was 0%, 33%, and 100% for Types A, B, and C, respectively.

Touho treated nine patients with MCA angioplasty. Six patients had M1 lesions and three had M2 lesions.⁶² Two patients with stenosis in the M1 segment suffered worsening hemiparesis after PTA, which improved to better than before the procedure within 1 month. Takis and colleagues⁶³ treated a patient with 90% stenosis of the M1 segment with angioplasty. The patient developed symptomatic vasospasm that failed to respond to treatment with intra-arterial papaverine or alteplase. Overdilatation is associated with the risk of both vessel rupture and vasospasm. Tsai et al⁶⁴ minimized the risk of

these complications by underdilating the stenosis. Normal flow was restored when the vessel was restored to 50% of its normal caliber. In the report by Suh and others,⁵⁵ there were no significant complications during or after angioplasty in 10 patients. The results were attributed to the slight underdilatation of the stenotic M1 and the choice of the most suitable microballoon for each patient.

In the study by Mori and researchers,⁵⁴ angioplasty of intracranial vessels had a higher complication rate compared with extracranial vessels. The pitfalls of angioplasty of the MCA are similar to those associated with angioplasty of other small vessels. The most common complications are dissection, acute closure, elastic recoil and restenosis, and stroke.^{52,54,63,65,66} The inherent risks of vessel dissection, distal embolization, and vessel rupture have limited the widespread use of intracranial balloon angioplasty despite reports of increased patency rates and good outcomes.^{53,54} Given the small size of the MCA and the area of the brain that is fed, abrupt closure often cannot be tolerated, resulting in stroke or death.

Stenting

The use of stents is a well-established technique for the endovascular treatment of atherosclerotic lesions in the coronary and peripheral arteries. Stents have reduced the complications often associated with balloon angioplasty in other vascular beds. The advantages of stent-assisted angioplasty include exclusion of the plaque and regions of dissection from the vessel lumen and prevention of vessel recoil and rupture.⁶⁷ Huang and colleagues⁶⁸ demonstrated the efficacy of stenting coronary vessels. Of 43 lesions with coronary vessels with a caliber of less than 2.5 mm, 76% of patients remained symptom free or had patent target sites after coronary stent placement.

Stenting minimizes the risk of acute closure caused by compression of an intimal flap and by entrapment of plaque material between the stent and vessel wall. The stent can act as a barrier to

platelet aggregation of plaque. By providing additional wall support, the risk of vessel rupture is also reduced. Although no evidence has demonstrated the superiority of stent placement over angioplasty alone in the intracranial circulation, available data indicate that it is superior for coronary vessels of similar caliber to the intracranial vessels. Stenting is a better tool for vascular recanalization than PTA. In theory, stenting should treat dissection and reduce the incidence of vessel recoil and restenosis associated with PTA alone.

Stent placement for intracranial stenosis developed because most long-term outcomes of balloon angioplasty alone have been less than satisfactory.^{54,59,63,65,66} Previously, the limiting factor of the deployment of stents in the intracranial circulation was difficulty tracking the stents into the vessels.^{16,56,58} The carotid siphon was considered the limit for intravascular stents in the anterior circulation.⁶⁹

A new generation of more flexible and compact coronary stents that can be deployed in the major intracranial arteries has now been developed.^{54,70–73} The availability of more flexible stents and the development of potent antiplatelet inhibitors have led to the application of stents in the management of ischemic intracranial cerebrovascular disease.⁷⁴

In 2000, Gomez and associates⁵⁸ first reported the technique of stenting the MCA. Since then three other series have been reported.^{16,56,57} Gomez et al⁵⁸ predilated the lesion to facilitate the crossing and deployment of the stent, to test the response of the vessel to balloon expansion, to assess the trackability of the interventional equipment, and to gauge the effect of balloon inflation on the patient. They emphasized that stent-assisted angioplasty should provide sufficient perfusion to reduce ischemic symptoms, not to provide an angiographic cure.

Kim and colleagues⁵⁷ attempted stent-assisted angioplasty of symptomatic high-grade stenosis on the proximal portion of the MCA in 14 patients. Stenting was successfully performed in eight patients without serious complications; it

was unsuccessful in two patients because the curve of the ICA siphon was tortuous. Four patients had complications. Two had an arterial rupture (one was rescued by an additional stent and balloon tamponade; the other died), one had thrombotic occlusion, and one had a distal thrombosis. Although the rate of restenosis was better than that for PTA as reported previously, the complication rate of stent-assisted angioplasty was higher than the known annual risk of ipsilateral stroke. Kim and coworkers concluded that the technique is worthwhile in the prevention of secondary ischemic stroke in patients who have suffered stroke and is potentially effective but also hazardous in patients with recurrent TIAs related to MCA stenosis. They emphasized the danger of balloon overdilatation in the intracranial circulation and stressed the importance of underdilatation because the muscularis and adventitial layers of the arterial wall are insubstantial and the risk of perforation is elevated.^{55,57} The balloon was inflated to a maximum pressure of 6 atm.

Jiang et al have reported the largest series of patients (42 stenoses in 40 patients) treated with stenting for M1 stenosis.⁵⁶ The technical success rate of stenting was 97.6% for the lesions and 97.5% for the 40 patients. The mortality rate was 2.5% and the perioperative complication rate was 10%. Thirty-eight patients remained free of TIAs or stroke during the median follow-up of 10 months. The complication rate was 10%. Three patients suffered intracranial hemorrhage, which the authors postulated might have been related to the use of double stents in tandem, to prior damage of the thinner wall of the MCA by the distal end of the first stent while delivering the second stent, or to perforation of the artery distal to the lesion by the microguidewire. Two of three patients with double stents in tandem experienced intracranial hemorrhage.

Several strategies have been proposed to prevent intracranial hemorrhage or subsequent patient deterioration: blood pressure strictly maintained below 120/80 mm Hg, a CT scan of the brain immediately after stenting to detect early hemorrhage so as to avoid continued anticoagulation and

antiplatelet therapy, gentle manipulation of the microguidewire, deliberate use of double stents in tandem, and avoidance of additional vascular dilator agents that may increase the risk of hemorrhage. Once intracranial hemorrhage is detected, blood pressure must be strictly controlled below 110/70 mm Hg, heparinization is reversed with protamine, and emergent salvage is performed.

No randomized controlled trials have compared the efficacy of balloon angioplasty or stent-assisted angioplasty with other treatment modalities. Although stent-assisted angioplasty of MCA stenosis decreases the incidence of some complications associated with simple balloon angioplasty, there are technical limitations. In one study, the risk of stroke or death was higher than the expected natural history of the disease. However, the field of endovascular neurosurgery is evolving rapidly. As the safety of treating small intracranial vessels improves, the indications for endovascular treatment of intracranial stenosis will expand.

CONCLUSION

Current management of symptomatic MCA stenosis requires an initial trial of antiplatelet or anticoagulant therapy. Patients who fail medical therapy and demonstrate evidence of hemodynamic insufficiency are candidates for surgical or endovascular intervention. EC-IC bypass has been the mainstay of treatment for such patients, but endovascular techniques are rapidly becoming an equally appealing treatment option. As techniques evolve and options expand, treatment of this complex condition will continue to improve.

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