Reappraisal of Primary Balloon Angioplasty without Stenting for Patients with Symptomatic Middle Cerebral Artery Stenosis

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Abstract

There is a controversy regarding the safety and efficacy of intracranial stenting. We describe our experience with primary balloon angioplasty without stenting for symptomatic middle cerebral artery (MCA) stenosis. All patients who underwent balloon angioplasty without stenting for MCA stenosis between 1996 and 2010 were retrospectively reviewed. We evaluated technical success rates, degrees of stenosis, and stroke or death within 30 days. Among patients who were followed-up for > 1 year we evaluated latest functional outcomes, stroke recurrence at 1 year, and restenosis. In total 45/47 patients (95.7%) were successfully treated. Average pre- and postprocedure stenosis rates were 79.9% and 39.5%, respectively. Three neurological complications occurred within 30 days: one thromboembolism during the procedure; one lacunar infarction; and one fatal intraparenchymal hemorrhage after the procedure. Stroke or death rate within 30 days was 6.4%. Thirty-three patients were available for follow-up analysis with a mean period of 51.5 months. The combined rate of stroke or death within 30 days and ipsilateral ischemic stroke of the followed-up patients within 1 year beyond 30 days was 9.4%. Restenosis was observed in 26.9% of patients and all remained asymptomatic. In our retrospective series, balloon angioplasty without stenting was a safe, effective modality for symptomatic MCA stenosis. For patients refractory to medical therapy, primary balloon angioplasty may offer a better supplemental treatment option.

Key words: intracranial stenosis, angioplasty, middle cerebral artery, stent

Introduction

The results of the Stenting and Aggressive Medical Management for Preventing Recurrent Stroke in Intracranial Stenosis (SAMMPRIS) trial showed a 30-day stroke or death rate of 14.7% in the stent plus medical treatment arm compared to that of 5.8% in the medical therapy arm.1 Advancements in medical treatment are considerably notable; however, the stroke or death rate in 1 year in the medical treatment arm was approximately 12.2%. Thus, certain high-risk populations may still benefit from endovascular intervention.

The following are the two primary endovascular modalities used for intracranial stenosis: primary balloon angioplasty alone and stenting. Stenting remains a challenging technique when navigating into a tortuous artery and also involves a possible risk of releasing plaques as atheroemboli into the adjacent perforating vessels (snow-plowing effect) or distal cerebral vessels (cheese grater effect). Compared to stenting, primary submaximal angioplasty may have some potential benefits because it is easier to navigate into a tortuous artery and incurs minimal injury to atherosclerotic plaques. Recently, some reports reappraised primary angioplasty without stenting for intracranial stenosis.2,3

In Japan, stenting for intracranial arteries has not been officially approved by the Ministry of Health until 2014, so we have performed balloon angioplasty without stenting for intracranial stenosis. The middle cerebral artery (MCA) is located distal to the carotid siphon and has many perforating arteries. Hence, we believe that primary angioplasty alone is more suitable for this lesion than stenting. In this study, we describe the clinical outcomes of primary balloon angioplasty without stenting for treating symptomatic MCA stenosis in order to compare these with the outcomes of medically-treated MCA stenosis and the stenting arms of the SAMMPRIS trial.
Materials and Methods

I. Patients

We retrospectively evaluated consecutive patients who underwent balloon angioplasty without stenting for atherosclerotic MCA stenosis at our hospital and associated institutions between April 1996 and October 2010. All patients had symptomatic ischemic symptoms because of severe stenosis in the M1 segment of the MCA and had received elective angioplasty in addition to medical therapy. The inclusion criterion for endovascular treatment was angiographically demonstrated MCA stenosis of \( \geq 70\% \) without complete infarction. Exclusion criteria were as follows: lesions > 10 mm in length, extremely eccentric, totally occluded, and extremely angulated (\( \geq 90^\circ \)); patients with complete cerebral infarction in the territory of the target stenosis; and patients with poor physical conditions. For each patient, treatment indications were determined after a discussion with an experienced neuroendovascular surgeon and the physician in charge regarding the necessity and feasibility of endovascular therapy. Patients who underwent angioplasty in acute settings (within 24 h of onset) were excluded from this analysis.

II. Procedures

All patients were administered at least one of the following antiplatelet agents before the procedure: ticlopidine, aspirin, or clopidgrel. Systemic heparinization (5,000 E and 1,000 E per 1 h administration of unfractionated heparin) was initiated after a sheath puncture under local anesthesia, and a 6 F guiding catheter was navigated into the internal carotid artery (ICA). The diameter of the parent vessel was measured from a diagnostic angiogram, and an undersized balloon with a diameter of \(< 80\% \) of the parent vessel was selected. A 0.014-inch guidewire was carefully placed across the lesion to maintain the true lumen of the M2 segment. The balloon catheter was used in navigating the 0.014-inch wire in the first place. However a microcatheter was also used when a safe navigation of the wire was difficult in combination with the balloon catheter itself, because of a tortuous vascular anatomy or a very severe stenosis. The microcatheter was exchanged for a balloon catheter after it crossed the stenotic lesion. When the exchange method was used, a tip of the 0.014-inch wire was shaped to a round form and was placed in a straight portion of distal MCA to avoid a risk of vessel perforation during the procedure. The angioplasty balloon was delivered and positioned to cover the total length of the stenotic lesion. The balloon was inflated at a rate as slow as 1 atmosphere per 10 s to the nominal pressure and was kept inflated for at least 60 s. Stenosis of \(< 50\% \) after treatment was acceptable. When adequate dilatation was not achieved or when elastic recoil of the stenosis occurred, a repeat angioplasty with a higher inflating pressure or with a larger balloon was performed within the range of 80% of the diameter of the parent vessel. When an acute intraluminal thrombus was observed, ozagrel sodium was added intravenously or cilostazol was added through a gastric tube. Intra- and postoperative images of an illustrative case are presented in Fig. 1.

III. Postprocedure management

After the procedures, patients were managed in the intensive care unit or a postoperative observation room. Heparin was naturally deactivated, and antiplatelet therapy was continued. Blood pressure was managed so as not to exceed 160 mmHg during the acute phase using intravenous nicardipine. Patients received rehabilitation therapy, if necessary.

IV. Follow-up policy

At 6 months after treatment, imaging follow-up using digital subtraction angiography (DSA) and/or magnetic resonance imaging (MRI) was performed to evaluate restenosis of the lesion and recurrent

Fig. 1 Case: A 77-year-old man who presented with mild right hemiparesis and aphasia. a: Cerebral angiography showing severe left middle cerebral artery (MCA) stenosis. b: Angiogram of balloon angioplasty. c: Angiogram showing stenosis improvement. d: SPECT showing vascular reactivity improvement in the territory of the left MCA (upper left: control pretreatment, upper right: with acetazolamide (ACZ) loading before treatment lower left: control posttreatment, lower right: with ACZ loading after treatment). e: Angiogram 6 months after the procedure. SPECT: single photon emission computed tomography.
Retreatment indications were as follows: when the patient exhibited recurrent ischemic symptoms due to restenosis of the treated lesion or when asymptomatic restenosis of ≥ 70% was observed and single photon emission computed tomography (SPECT) showed hemodynamic compromise in the territory of the lesion. In actuality, an indication for retreatment for each patient was decided after discussion with an endovascular therapist and the physician in charge.

Data were collected from clinical records, surgical reports, and neuroimages. Technical success was defined by meeting the following two criteria: residual stenosis of < 50% and restoration of blood flow velocity that was apparently delayed before the procedure. We evaluated the success rates of the procedures, the degrees of stenosis before and after treatment, and complications associated with the procedures. Follow-up data were obtained from current medical records for patients with a continuous follow-up or by telephone interviews for patients who were not followed-up at the treating hospital.

To assess long-term follow-up, we evaluated current functional status, stroke recurrence after treatment, incidence of restenosis, and retreatment of the treated lesion. Good functional status was defined as 0–2 on the modified Rankin Scale (mRS). Binary restenosis was defined as > 50% restenosis on follow-up angiography or on magnetic resonance angiography (MRA). These data were collected between September 2010 and August 2013 for the purpose of this study.

The aim of this study was explained to each patient or to the patient’s family, and they all consented to inclusion in this study. All the procedures were conducted in accordance with the Declaration of Helsinki 1964 and its later amendments.

Results

I. Patients and angioplasty procedures

A total of 52 patients with symptomatic MCA stenosis were treated by primary balloon angioplasty during this period. Five patients who were treated in the acute setting (within 24 h of onset) were excluded from this analysis. Thus, 47 patients (men 33; women 14; mean age 65.7 ± 10.7 years; range 26–83 years) were evaluated in this study. The initial symptoms included minor stroke in 35 patients and transient ischemic attack (TIA) in 12 patients. The interval from onset to endovascular treatment was 25.3 days (range, 1–90 days), and 23 patients (47.9%) were treated within 14 days. Clinical characteristics of all patients included in this study are summarized in Table 1. Early in our series, we diverted coronary balloon catheters to intracranial angioplasty and used STEALTH (Target Therapeutics, Fremont, California, USA) for 5 patients, Ranger (Boston Scientific, Natick, Massachusetts, USA) for 2 patients, OpenSail (Abbott, Chicago, Illinois, USA) for 1 patient, and Maverick (Boston Scientific, Natick, Massachusetts, USA) for 4 patients. After late 2002, we used Gateway Balloon Catheter (Stryker, Kalamazoo, Michigan, USA) for 30 patients and Unryu (Kaneka, Osaka) for 5 cases. The balloons were dilated in a slow inflation manner between the nominal and the rated-burst pressure of each balloon catheter within the range of 80% of the diameter of the parent vessel with reference to the durability chart. There was neither a vessel rupture nor vessel perforations during the procedure. No rescue stenting procedure was performed.

II. Immediate results

The technical success rate was 95.7% (45/47 patients). The mean pre- and posttreatment stenosis rates were 79.9% and 39.9%, respectively. One procedure was aborted because the balloon catheter could not pass a tight, tortuous stenosis. This patient was subsequently treated by medical therapy alone. In the second failed case, the lesion was occluded because of arterial dissection after balloon angioplasty. Rescue stenting or emergency bypass surgery was considered; however, we treated this patient conservatively as neurological symptom exaggeration did not appear at the time of occlusion because the leptomeningeal collateral circulation was well developed. Fortunately, recanalization of the MCA with mild residual stenosis was observed at 2 weeks after this procedure.

The combined 30-day stroke or death rate was 6.4% (3/47 patients), which included one thromboembolism during the procedure (2.1%), one intracerebral...
hemispherical lacunar infarction (2.1%), and one ipsilateral lacunar infarction due to occlusion of the perforating artery at 4 days after the procedure (2.1%). The intracerebral hemorrhage occurred in a 73-year-old woman who presented with progressive stroke due to severe right MCA stenosis. Medical therapy could not prevent neurological worsening, and we performed balloon angioplasty 5 days after onset. A first angioplasty using a 1.5-mm balloon restored her delayed blood flow velocity on DSA, and a second angioplasty using a 2-mm balloon successfully added to a gain of < 50% stenosis. Her blood pressure was difficult to moderately control despite intravenous nicardipine injection, and her level of consciousness deteriorated 2 h after the procedure. A computed tomography (CT) scan revealed a huge intraparenchymal hematoma and a threatening cerebral herniation (Fig. 2). Emergency craniotomy and hematoma evacuation were performed, but she died 1 week after this procedure. Immediate results and details of technical failures and stroke or death within 30 days were summarized in Tables 2 and 3.

## III. Follow-up data

Clinical follow-up data of > 1 year was available for 33 patients. Five patients died within the first 12 months. One patient died because of postprocedure intracerebral hemorrhage (as noted above), one because of liver cirrhosis, one committed suicide, one because of lung cancer, and one because of an unknown cause. Nine patients including the two failed cases were lost to follow-up within the initial 1 year and we could not contact them or their family. Flow chart of patients who were treated and followed-up are illustrated in Fig. 3.

The mean follow-up period for the 33 successfully followed-up patients was 51.5 months (range, 13–152 months). Of these, 27 (78.8%) patients had good functional status (mRS, 0–2) at the time of data collection (Fig. 4). Three had a recurrent stroke hemorrhage after the procedure (2.1%), and one ipsilateral lacunar infarction due to occlusion of the perforating artery at 4 days after the procedure (2.1%). The intracerebral hemorrhage occurred in a 73-year-old woman who presented with progressive stroke due to severe right MCA stenosis. Medical therapy could not prevent neurological worsening, and we performed balloon angioplasty 5 days after onset. A first angioplasty using a 1.5-mm balloon restored her delayed blood flow velocity on DSA, and a second angioplasty using a 2-mm balloon successfully added to a gain of < 50% stenosis. Her blood pressure was difficult to moderately control despite intravenous nicardipine injection, and her level of consciousness deteriorated 2 h after the procedure. A computed tomography (CT) scan revealed a huge intraparenchymal hematoma and a threatening cerebral herniation (Fig. 2). Emergency craniotomy and hematoma evacuation were performed, but she died 1 week after this procedure. Immediate results and details of technical failures and stroke or death within 30 days were summarized in Tables 2 and 3.

### Table 2 Immediate results (within 30 days)

<table>
<thead>
<tr>
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<th>N (%)</th>
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<tbody>
<tr>
<td>Technical success</td>
<td>45 (95.7)</td>
</tr>
<tr>
<td>Stenosis</td>
<td></td>
</tr>
<tr>
<td>Pretreatment</td>
<td>79.9%</td>
</tr>
<tr>
<td>Posttreatment</td>
<td>39.5%</td>
</tr>
<tr>
<td>Stroke or death</td>
<td>3 (6.4%)</td>
</tr>
<tr>
<td>Mortality</td>
<td>1 (2.1%)</td>
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</tbody>
</table>

### Table 3 Details of technical failures and stroke or death within 30 days

<table>
<thead>
<tr>
<th></th>
<th>N (%)</th>
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<tbody>
<tr>
<td>Technical failure</td>
<td>2 (4.3%)</td>
</tr>
<tr>
<td>Unsuccessful lesion cross</td>
<td>1 (2.1%)</td>
</tr>
<tr>
<td>Occlusion due to iatrogenic dissection</td>
<td>1 (2.1%)</td>
</tr>
<tr>
<td>Stroke or death within 30 days</td>
<td>3 (6.4%)</td>
</tr>
<tr>
<td>Thromboembolism during the procedure</td>
<td>1 (2.1%)</td>
</tr>
<tr>
<td>Occlusion of the perforating artery</td>
<td>1 (2.1%)</td>
</tr>
<tr>
<td>Intraparenchymal hemorrhage</td>
<td>1 (2.1%)</td>
</tr>
</tbody>
</table>

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**Fig. 2** Case: A 73-year-old woman with fatal postprocedure intraparenchymal hemorrhage. a: DWI showing growing cerebral infarction in the territory of the right middle cerebral artery (MCA); left: on admission, right: 4 days after starting dual antiplatelet therapy. b: Pretreatment SPECT showing decreased cerebral vascular reactivity; left: control pretreatment, right: with acetazolamide (ACZ) loading pretreatment. c: Pretreatment angiogram showing severe right MCA stenosis and delayed distal flow. d: Stenosis improved and distal flow was restored after the procedure. e: CT scan showing a huge intraparenchymal hematoma. CT: computed tomography, SPECT: single photon emission computed tomography.
within 1 year beyond 30 days. These included one with ipsilateral lacunar infarction (10 months after the procedure), one with brainstem infarction (9 months after the procedure), and one with cerebellar hemorrhage (10 months after the procedure). The recurrence rate of ipsilateral ischemic stroke beyond 30 days was 3.0% at 1 year (1/33 followed-up patients). Thus, the observed stroke or death rate within 30 days or ipsilateral ischemic stroke beyond 30 days at 1 year was 9.4%. Imaging follow-up data were available for 26 patients, of which 7 (26.9%) had asymptomatic restenosis. Retreatment was successfully performed for four patients by balloon angioplasty alone and without any procedural complications.

**Discussion**

In this study, we report the outcomes for 47 consecutive patients with symptomatic MCA stenosis who underwent primary balloon angioplasty without stenting. Our technical success rate was 95.7%, and the 30-day stroke or death and mortality rates were 6.4% and 2.1%, respectively. The combined stroke or death rate within 30 days and ipsilateral recurrent stroke within 1 year beyond 30 days was 9.4%. Among the followed-up patients, 78.8% had a good functional status (mRS, 0–2). This study used a retrospective design and only a limited number of patients were available for follow-up analysis. Therefore, direct comparisons with the SAMMPRIS trial are impossible. However, the immediate results in our series were comparatively better than those in the stenting arm (6.4% vs. 14.7%), and our 1-year provisional results were comparable with those in the medical management arm (9.4% vs. 12.2%).

A premature halt to enrollment in the SAMMPRIS trial raised a question regarding the role of endovascular therapy for the management of symptomatic intracranial atherosclerotic disease. As an explanation of this unexpected outcome, two possible reasons have been proposed. The first was the markedly lower stroke rates in the medical management arm compared with those in the preceding Warfarin versus Aspirin for Symptomatic Intracranial Disease (WASID) trial. These successful results were explained on the basis of advances in medical therapy that included two antiplatelet agents, a statin, and antihypertensive therapy and close patient management. However, there was a primary end point of 12.2% (any stroke or death within 30 days after enrollment or a stroke in the territory of the symptomatic artery beyond 30 days) in the aggressive medical management group, and many experts consider that there is still room for endovascular therapy to improve the outcomes of symptomatic intracranial stenosis.

The second reason was a considerably higher 30-day stroke or death rate in the stenting group than that in previous reports. In our opinion, this unexpected result can possibly be explained as a consequence of the following single device system that was used in this trial: the Gateway angioplasty balloon and Wingspan stent.

Recently, a detailed investigation regarding the periprocedural events in the SAMMPRIS trial was published. According to that paper, there were 21 periprocedural ischemic events in the stenting arm; 15 occurred in the perforator territory and 3 were attributed to acute and delayed stent thrombosis. Six of the 13 hemorrhagic events were categorized as procedural subarachnoid hemorrhage (SAH), and 3 of these 6 patients were attributed to wire perforation. These three types of complications (perforator ischemia, stent thrombosis, and wire perforation) may stem from the intrinsic drawbacks of the stenting system. Occlusion of the perforating artery may have been caused by displacement of the atheromatous debris after stenting (snow-plowing effect). Stent thrombosis was one of the complications of stenting. Wire perforation was speculated to be caused by a catheter-exchange technique that was required for navigation with the Wingspan system.

Intracranial angioplasty without stenting has recently been reappraised. In a series of 41 consecutive patients who were treated by primary submaximal angioplasty and without stenting for symptomatic intracranial stenosis, the 30-day event rate was 4.9%, and the 1-year periprocedural and ischemic event-free survival rate was 91%, which was higher than that in the medical (88%) and surgical (77%) arms of SAMMPRIS. Twenty-six of primary angioplasty patients treated between 2006 and 2011 were retrospectively reviewed. The stenosis rate improved from 71.2% to 46.6%, and retreatment was required for only 3.8% of these patients. The 30-day stroke or death rate of 11.5% in this balloon angioplasty series was higher than that in the medical arm of SAMMPRIS but was lower in the stenting arm. The 1-year risk of stroke was comparable to the
medical arm. These results suggest that primary balloon angioplasty is safer than stenting, and it is as effective as aggressive medical therapy, at least at 1 year. These reports also indicated the negative aspects of primary stenting similar to those we described above. However, subgroup analyses between different stenosis locations were not available in those reports.

Different intracranial stenosis locations should be separately discussed. We consider that the MCA is not suitable for primary stenting because it is distally located and involves a perforator-rich zone. The Wingspan system is occasionally undeliverable to distal lesions with an excessive vascular tortuosity. A snow-plowing effect is a possible mechanism for perforator ischemia after stenting and may be disadvantageous, particularly with MCA stenosis. In addition, posttreatment stenosis more commonly develops in the MCA and supraclinoid ICA compared with that in arteries at other locations. We propose primary submaximal angioplasty as the first-line endovascular option for symptomatic MCA stenosis.

There is a dispute regarding whether intracranial stenting causes perforator ischemia. An in vivo study indicated the possibility of ostial narrowing of a perforator in atherosclerotic vessels after stent placement. Leung et al. reported a case series including 23 patients with MCA stenting having no new perforator territory infarction after intracranial stenting; however, treatment timing was not clearly described in that paper. In our experience, most patients with symptomatic intracranial stenosis that is refractory to medical therapy have both embolicigenic and hemodynamic propensities. A non-stabilized fresh atheroma possibly displaces an embolic shower of debris into the perforating or parent arteries when it is pressurized by the sustained radial force of a self-expanding stent. Stent thrombosis is possibly caused when a considerable amount of debris is plowed into the stent lumen through a stent strut. Primary submaximal balloon angioplasty is a theoretically safer modality to gain minimal lumen width in order to get through the acute phase.

Prospective series of primary balloon angioplasty without stenting for symptomatic MCA stenosis do not exist. Retrospective analyses that were reported in the early-to-mid 2000 described clinical results that were equivalent to our series, and the rates of technical success, periprocedural stroke or death, and mortality were 90–91%, 0–6%, and 0–3%, respectively. The rate of asymptomatic restenosis was 20–50%, and ipsilateral ischemic stroke recurrence was not reported in those series.

There are two main disadvantages with primary angioplasty without stenting. One is iatrogenic arterial dissection and the other is inadequate dilatation due to elastic recoil. Iatrogenic arterial dissection was observed in 11/36 patients (30.5%) in a previous balloon angioplasty series, although all these patients remained asymptomatic. If dissection after balloon angioplasty causes arterial occlusion, rescue stenting will be one of the effective countermeasures. Medical therapy is not a promising treatment modality for such cases, although one patient with arterial occlusion in our series was fortunate enough to recover without a subsequent worsening in their condition. Stenting may also provide one treatment option for inadequate dilatation due to elastic recoil after balloon angioplasty, although minimal stenosis improvement provides an adequate treatment effect in most cases. When stenting is required for recurrent stenosis after primary balloon angioplasty, a staged procedure may be reasonable to avoid periprocedural complications.

Intraparenchymal hemorrhage after treatment is an inevitable periprocedural risk of endovascular treatment for intracranial stenosis. This type of hemorrhage occurred in 2–3% of all reported series, and the incidence was equal to that in our series (2.1%). Reperfusion hemorrhage is considered to be the underlying mechanism; however, risk factors and effective prevention strategies have not yet been determined. Although we experienced one fatal case, we consider that submaximal angioplasty and strict control of posttreatment blood pressure may help avoid fatal hemorrhage. Additional use of sedative drugs may be effective if intravenous antihypertensive drugs cannot control blood pressure. In any event, a rigid postprocedure protocol is mandatory after intracranial angioplasty.

Reducing periprocedural complications is the most important issue for endovascular therapy for intracranial stenosis. In our opinion, primary submaximal angioplasty possibly offers a safe additional treatment option to aggressive medical therapy for MCA stenosis. Patient selection is also important. Patients who are at a high risk of stroke recurrence because of medical therapy alone and are concomitantly at a low risk for balloon angioplasty are good candidates for additional endovascular therapy. Patients with progressive symptoms despite complete administration of antiplatelet agents will be valid for adapting the endovascular option. In addition, lesions that are unsuitable for balloon angioplasty should be excluded as endovascular candidates. Mori et al. suggested a classification for intracranial lesions; type A (≤ 5 mm in length and concentric or moderately eccentric lesions less than totally occlusive) yielded favorable clinical outcomes.

Neurol Med Chir (Tokyo) 55, February, 2015
We consider that the balloon angioplasty has several advantages on bypass surgery in treating the stenotic lesion of the MCA. First, we are able to access to the stenotic lesion and start angioplasty more promptly after neurological deterioration of the patient than by bypass surgery. Second, endovascular treatment could be performed without induction of general anesthesia and considered to be less invasive than open surgery. Third, antegrade cerebral blood flow could be kept by angioplasty whereas bypass surgery supplies retrograde cerebral blood flow. Microembolism is thought to be one of the mechanisms of cerebral ischemia in intracranial stenosis, so we consider angioplasty of the lesion may be more essential than bypass surgery in treating the stenotic lesion. If symptoms of the patient are caused by the occlusion of the MCA, bypass surgery may become a preferable option to improve hemodynamic ischemia.

There were several limitations to our study. This was a retrospective study and only limited number of patients was available for follow-up analysis. The rate of recurrent stroke was provisionally calculated; however, if all of the patients who were lost to follow-up had stroke recurrence, our results would considerably differ. In addition, there was no comparison arm for medically treated patients in our study. Only a prospective randomized study will be able to establish the usefulness of primary submaximal angioplasty for symptomatic MCA stenosis and, in our opinion, the future study should target only for patients with recurrent symptoms despite aggressive medical therapy and stenting should be used temperately for this lesion.

Conclusion

In this retrospective analysis, primary angioplasty for symptomatic MCA stenosis was feasible with a low complication rate compared to the stenting group in the SAMMPRIS study. The preventative effects of angioplasty were maintained for a long period among the successfully followed-up patients in our study. In our opinion, for symptomatic MCA stenosis refractory to aggressive medical therapy, angioplasty without stenting is the primary endovascular therapy before performing intracranial stenting. A prospective study will be required to establish the safety and efficacy of this procedure.

Conflicts of Interest Disclosure

The authors have no conflict of interest and have registered online self-reported conflict of interest disclosure statement forms through the website for Japan Neurosurgical Society members.

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