Intracranial atherosclerosis is a major cause of ischemic stroke and accounts for 8% to 15% of strokes caused by cerebral atherosclerosis, depending on the population studied (1, 2). Sometimes patients with symptomatic intracranial atherosclerotic stenosis are insufficiently protected against ischemic stroke despite medication with platelet aggregation inhibitors. However, the treatment of patients with intracranial atherosclerosis lesions remains controversial. The results of endovascular revascularization of intracranial vessels with balloon- or stent-assisted angioplasty or stent have been tempered by the increased risk of stroke (3). The availability of recently introduced flexible stents, the development of potent platelet aggregation inhibitors, and increasing evidence from experimental and clinical studies of intracranial stents have improved the endovascular management of ischemic intracranial cerebrovascular disease.

Complications from intracranial artery angioplasty and stenting (IAS) include cerebral infarction, intracranial hemorrhage caused by reperfusion injury, and contrast-induced renal failure. Most cerebral infarctions are directly related to the endovascular procedure, involving the ipsilateral vascular territory (4). This ischemic complication results from distal embolization, vessel dissection, and acute vessel occlusion secondary to dissection or platelet aggregation.

We present two cases with unexpected infarctions involving territories unrelated to IAS procedures.

**CASE 1**

A 60-year-old man presented with right hemiparesis (Grade III) beginning one week prior to admission. He had a history of hypertension, which had been treated with medication. MRI upon admission revealed multiple fragmented acute infarctions in the left centrum ovale and corpus callosum. MR angiography demonstrated severe stenosis of the left middle cerebral
artery (MCA) and anterior cerebral artery (ACA). He was conservatively treated with anti-platelet therapy. After being admitted to our hospital, he reported weakness in his right extremities was deteriorating at times. Diffusion weighted MRI (DWI) performed after symptom progression demonstrated new diffusion-restrictive lesions in the left MCA territory, including the internal border zone, insular cortex, and temporal lobe. Digital subtraction angiography (DSA) showed long segmental severe stenosis of the left MCA M1 with an irregular contour. The left ACA also showed severe stenosis at the proximal A3 segment. There was no evidence of a cardiac embolus and results of hemorheology and coagulation function tests were unremarkable. Given the continuous deterioration of clinical symptoms despite medical treatment, we chose to implement endovascular recanalization therapy. Balloon angioplasty was recommended as the appropriate endovascular method, because the diameter of the non-pathologic segment of the left MCA was lower than 2.5 mm.

Under local anesthesia, percutaneous access was obtained via the right femoral artery. Bolus intravenous injection of heparin (5,000 IU) was administered to prevent a thromboembolic event during the endovascular procedure. A 6 Fr guiding catheter (Cordis Envoy; Johnson and Johnson Medical, Miami Lakes, FL, USA) was positioned in the distal cervical segment of the left internal carotid artery (ICA), proximal to the petrous portion. The stenotic segment was crossed with a 0.014 inch guidewire with the tip positioned in the insular portion of the middle cerebral artery to ensure maximal support and allow tracking of the balloon. Then the stenosis was dilated with a 1.5 × 15 mm coronary balloon catheter (RyuJin balloon; Terumo Corp., Tokyo, Japan) at 7 atm for 20 seconds two times. A control angiogram just after intervention showed an effectively widened Lt MCA caliber and good circulation of the distal branches (Fig. 1).

After 30 minutes, DSA was done to rule out restenosis or in-stent thrombus. Mild recoiling of the stenotic segment after angioplasty was noticed but stenotic...
diameter was over 50% and the contour of the lumen became smooth. The stenosis of the right ACA did not change from initial angiographic findings. During and after the procedure, arterial blood pressure was restrictively controlled under a normal value by administration of a mixed alpha/beta adrenergic antagonist to prevent hyperperfusion.

Six hours after the procedure, a neurologic exam showed progression of left lower extremity weakness. Emergent DWI showed an acute infarction had not developed within the MCA territory but within the ACA territory. Because there was no change in the ACA stenosis between pre- and post-procedural angiography and the main endovascular manipulation occurred in the MCA, we suggest that decreased blood pressure after the periprocedural period resulted in hemodynamic infarction in the left ACA territory.

CASE 2

A 65-year-old man developed right lower extremity weakness 1 month before admission to our hospital. These symptoms progressed and motor aphasia later developed. He had a history of right ACA territorial infarction 2 months before admission. Antihypertensive medication was given to control blood pressure 2 months ago. MRI obtained at admission showed a subacute infarction in the subcortical white matter of the left MCA territory as well as an old focal cortical infarction in the right medial precentral gyrus. MR angiography demonstrated severe stenosis of the clinoid segment of the left ICA and occlusion of the proximal A3 segment of the right ACA. DSA revealed that the left clinoid ICA was severely narrowed (90%)

![Fig. 2. A 65-year-old man with ischemic infarction and severe stenosis of the left supraclinoid ICA. Frontal projection of the left carotid angiography before stenting (A) reveals severe stenosis of left supraclinoid ICA. Frontal projection of right carotid angiography (B) shows the occlusion of the right ACA (arrow) and collaterals from peripheral branches of the right MCA. Oblique view of stent during deployment in the left intracranial ICA (C) shows the fully expanded stent. Left internal carotid angiography after angioplasty (D) shows complete recanalization of the left supraclinoid ICA. Diffusion weighted MRI 48 hours after the endovascular procedure (E) demonstrates a new infarction in the right ACA territory (infracts in left MCA territory have presented before stenting).](image)
and cerebral blood flow to the MCA was delayed. The right ACA A3 segment was occluded and the right ACA territory supplied by pial collaterals of the right MCA and PCA. The left MCA territory showed a moderately decreased vascular reserve on brain SPECT (Fig. 2). Because the patient had suffered repeated ischemic stroke attacks after initial antiplatelet therapy 2 months prior, we elected to perform primary stent-assisted angioplasty.

We administered 75 mg of clopidogrel and 325 mg of aspirin per day starting 3 days before intervention. The patient underwent cerebral angioplasty and stent insertion under general anesthesia. During general anesthesia, blood pressure was decreased lower than non-analgesic status. A 6 Fr guiding catheter (Cordis Envoy; Johnson and Johnson Medical) was selectively placed via a femoral arterial route into the left distal petrous ICA. Heparin (5,000 U) was administered intravenously to avoid thrombus formation during the procedure. A 0.014 inch guidewire was navigated across the stenosis. A balloon-expandable coronary stent (Flexmaster F1 coronary stent; Abbott Vascular Devices, Ulestraten, NL, USA) with a 2.5 mm diameter and 12 mm length was guided over the wire and was deployed by inflating to 9 atm for 15 seconds. The lesion dilated easily, and the stenosis was reduced from 90% to 0%. A control angiogram 30 minutes after stent deployment demonstrated no in-stent stenosis or distal thrombosis. The patient did not present any other symptoms after the procedure. However, DWI obtained 24 hours after the procedure revealed a new acute ischemic lesion within the right ACA territory. Because the right ICA had not been manipulated, we suggest that the right ACA infarction was a hemodynamic infarction due to decreased blood pressure since general analgesic period. The patient was discharged after 8 days without significant complications.

**DISCUSSION**

The natural history of intracranial stenoses has not been studied as well as that of extracranial stenoses, even though intracranial stenoses have a higher rate of strokes. Recently, IAS has been performed in patients refractory to medical treatment, and the usefulness of angioplasty in the treatment of arteriosclerotic stenoses of the intracranial arteries has been well documented (3, 5, 6). However, compared with extracranial vessels, angioplasty of intracranial vessels has a higher complication rate. Therefore, application of IAS depends on both the risks of the untreated disease and the safety and effectiveness of the therapeutic procedure. In our hospital, indication has been applied restrictively to select patients for elective IAS. Patients were included if they had one of the following three conditions: (a) recurrent symptoms indicative of intracranial artery stenosis that were unresponsive to therapeutic doses of aspirin, ticlopidine, clopidogrel, or warfarin; (b) recurrent neurologic symptoms with significant perfusion problems confirmed by a decreased perfusion reserve on single photon emission computed tomography (SPECT); and (c) infarction at the border zone of the MCA region related to hemodynamic insufficiency.

Hyperperfusion syndrome is a rare but well described and potentially devastating complication after carotid endarterectomy or angioplasty and stenting. It is characterized by the development of clinical symptoms as a result of rapidly increased cerebral blood flow in excess of that required to meet metabolic demands. The capillary bed beyond the stenosis may be prone to perfusion breakthrough bleeding after an increase of blood flow to an area with impaired autoregulation. Because chronic cerebral ischemia induced by severe stenosis of the intracranial artery leads to impaired autoregulation of the peripheral vessels, IAS still has a risk for hyperperfusion syndrome (7, 8). We need to understand the mechanisms that promote this complication and determine the precise clinical and radiological risk factors in order to avoid it. After successful recanalization, strict blood pressure management with avoidance of hypertension is necessary to prevent hyperperfusion syndrome. Post-interventional heparinization should be avoided for the same reason, but reversal of heparin given during intervention is not recommended due to the increased risks of stent thrombosis.

Ischemic stroke is a major complication of intracranial endovascular therapy. IAS has problems with abrupt closure owing to complications such as intimal damage, elastic recoil, and in-stent thrombosis (5, 9, 10). Theses problems result in ischemic infarction in the territory of the treated vessel. There have been a number of reports on development of silent, small embolic infarctions in the contralateral and posterior circulation, as well as in the ipsilateral circulation, identified on follow-up DWI after carotid stenting. These findings suggest that the most likely sources of embolic material are catheters and guide wires that dislodge atheromatous material from the aorta.

Contrary to the usual pathophysiology of ischemic stroke induced by IAS, ischemic strokes in these cases resulted from hemodynamic insufficiency. These two
patients had pre-existent peripheral intracranial arterial steno-occlusive lesions that were remote from the endovascular manipulating vessels. Both patients were also treated for blood pressure control to prevent hyperperfusion syndrome after the periprocedural period. Because these patients showed impaired autoregulation on acetazolamide-challenged SPECT, we had to take measures to minimize the risk of hyperperfusion syndrome after recanalization of the intracranial arteries. However, strictly controlling blood pressure to prevent hyperperfusion syndrome triggered hemodynamic infarction of other vascular territories. As in these cases, neurointerventionalists should be aware of the development of remote hemodynamic infarctions related to decreased blood pressure in territories not manipulated during IAS procedures.

When intracranial artery angioplasty is performed on a patient with multiple intracranial arterial steno-occlusive lesions, neurointerventionalists should consider that excessive decreases in blood pressure during the periprocedural period may result in the hemodynamic infarction of co-existent stenotic vessels regardless of endovascular manipulation.

References