CASE REPORT

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Amaurosis Fugax Associated with Stenosis of the Intracranial Internal Carotid Artery : Successful Restoration of Ophthalmic Artery Flow by Stent Placement

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Two patients presented with amaurosis fugax(AMF), despite treatment with antiplatelet drugs and anticoagulants due to previous transient ischemic attacks. Angiography demonstrated severe stenosis in the cavernous and petrous internal carotid artery(ICA) respectively, with reduced flow in the ophthalmic artery(OA). Endovascular stent placement in both patients resulted in normalization of the vessel lumen of the stenotic vessel segments. In addition, complete restoration of OA flow was noted immediately after stenting. Both patients showed no further episode of AMF over a follow-up period of 38 and 23 months respectively. Our clinical and angiographic findings suggest that hemodynamic insufficiency in retinal vasculature caused by a stenosis of the cavernous or petrous ICA can be treated successfully by endovascular stent.

KEY WORDS : Amaurosis fugax · Intracranial stenosis · Vascular hemodynamic insufficiency · Stenting.

Introduction

AMF is mostly attributed to microembolism from atherosclerotic lesions of the ipsilateral internal carotid artery(ICA), or other vascular lesions, including lesion of the aortic arch and heart[1]. Vascular hemodynamic insufficiency caused by ophthalmic artery(OA) stenosis, vasospasm or obstructive lesions of the ICA has been reported as a possible mechanism causing AMF[1,4,10].

Also, hemodynamic retinal vascular insufficiency due to atherosclerotic stenosis of the siphon portion of the ipsilateral ICA has been proposed as an etiology for AMF[9]. However, no effective treatment modality has been reported. We describe two patients with AMF and atherosclerotic stenosis of the petrous or cavernous portion of the ICA with compromised flow in the OA that showed restoration of OA flow after stenting for the stenosis.

This is believed to be the first report showing vascular hemodynamic insufficiency caused by atherosclerotic stenosis of the intracranial ICA can contribute to AMF which can be successfully treated by stent placement.

Case Report

Case 1

A 54-year-old man presented with recurrent, transient right-side monocular blindness accompanied by transient left hemiparesis during exercise over a period of 6 months, despite treatment with Warfarin. He was on Warfarin medication because of transient left hemiparesis, which occurred two years prior to his admission. No cerebral infarction was found, but magnetic resonance imaging and angiography(MRA), performed at time of the admission, showed a stenotic lesion in the right cavernous ICA. A 70% stenosis of the cavernous portion of right the ICA causing reduced flow in the OA was found in the diagnostic angiograms obtained just before stenting (Fig. 1A). The patient received aspirin (300mg per day) and Clopidogrel (75mg per day) orally one week prior to the procedure. Stenting was performed successfully with a 3.5 × 12mm stent (Express; Boston Scientific). Control angiogram (Fig. 1B) immediately after stenting showed reestablishment of the normal caliber in the stenotic portion as well as restoration of flow in the OA. Follow-up angiography performed 19 months

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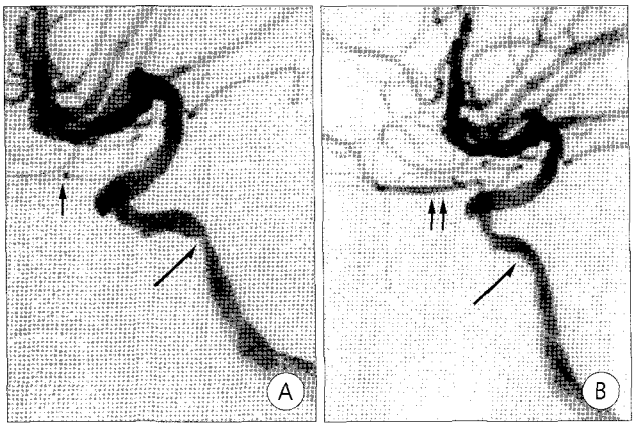

Fig. 1. Case 1. A : Right internal carotid arteriograms (lateral view). The early arterial phase with a 70% stenosis (large arrow) in the cavernous portion of the internal carotid artery and faint filling of the ophthalmic artery (small arrow). B : The post stenting arteriogram revealing normalization of the previously stenosed segment and restoration of flow (double arrow) in the ophthalmic artery.

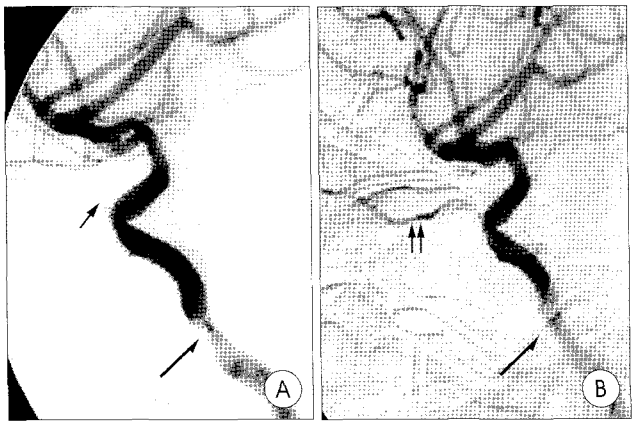

Fig. 2. Case 2. A : Right internal carotid arteriogram (lateral view) shows 75% stenosis (large arrow) of petrous portion and scant flow in the ophthalmic artery (small arrow). B : Arteriogram after stenting shows normalization of the stenosed vessel lumen and restoration of flow in the ophthalmic artery (double arrow).

after the stent placement showed no restenosis of the stented segment and good flow in the OA. The patient remained asymptomatic during a 38-months follow-up period.

Case 2

A 41-year-old man presented with three episodes of right-side AMF, occurring during treatment with aspirin that was taken because of angina pectoris. This symptom occurred when he stood up from sitting position. Funduscopy revealed no abnormal findings on admission. No embolic sources were found in the ipsilateral extracranial carotid artery, aortic arch, or heart, in MRA, carotid angiography and echocardiography examinations. The right ICA angiogram obtained just before stenting revealed a 75% stenosis in the petrous ICA, and reduced flow in the OA (Fig. 2A). The right external carotid artery(ECA) angiogram showed scanty collateral flow to the retina, but no retrograde flow from branches of the ECA to the OA in late arterial phase. Stenting was performed successfully using a 3.5×15mm stent (S670; Medtronic, Minneapolis, MN). The control angiogram (Fig. 2B) obtained immediately after stenting demonstrated normalization of the stenotic vessel lumen and complete restoration of flow in the OA. The patient remained asymptomatic during a 23-months follow-up period.

Discussion

AMF has been considered to be attributed to transient ischemia of the retina caused by various mechanisms, including embolism or vascular insufficiency, vasospasm, states of altered coagulability, and thrombocytosis[1,4-6,9]. Because the therapeutic approach and the prognosis differs depending on the specific cause, accurate knowledge of the mechanism causing AMF in an individual patient is important. Some observations of embolic particles in retinal arteries suggest that embolism is one of the pathogenetic mechanisms of AMF[5]. Terao et al.[9] described hemodynamic retinal vascular insufficiency as an etiology for AMF in patients with atherosclerotic stenosis of the siphon portion of the ipsilateral carotid. However, no evidence has been presented that severe stenosis of the intracranial carotid artery siphon is indeed relevant to the hemodynamic retinal vascular insufficiency. Also, no effective treatment modality has been suggested.

AMF in patients with atheromatous lesions of the ipsilateral ICA is thought to be caused by thromboembolism[2]. In our patients, however, the presence of precipitating factors (exercise, position change), improvement of recurrent symptoms of patients as well as the immediate increase in the previously compromised blood flow in the OA following stenting, indicates that AMF was caused rather by vascular hemodynamic insufficiency than by thromboembolism. Although degree of stenosis in our cases did not seem high enough to cause hemodynamic insufficiently in the OA territory, the stenotic lesion may have diverted blood flow within the ICA proximal to the OA origin, resulting in retinal vascular insufficiency. Thus, we believe that restoration of normal flow in the OA in our cases was caused by normalization of flow pattern in the ICA after stenting. Patients with AMF, associated with atherosclerotic stenosis of the ICA, have a considerable risk of subsequent stroke, although AMF per se has a better prognosis as compared to cerebral transient ischemic attacks(TIAs)[8].

Proper surgical treatment, such as carotid endarterectomy, has been proposed for operable lesions in patients with atherosclerotic stenosis of ICA[3]. Antiplatelet therapy has a favorable effect in the majority of those AMFs presumed to be caused by microthromboembolism formed at the "stump" at

either the extracranial ICA or the OA occlusions[1,7,9]. This medical treatment was ineffective, however, in patients with AMF that was thought to be caused by a hemodynamic mechanism resulting from the stenosis of the intracranial carotid artery, as it was in our patients[9]. Recent advances in intravascular technology and devices, allow easier navigation of stents to the intracranial circulation. Compared with balloon angioplasty, stent placement is more efficacious because it provides better and longer luminal patency by avoiding abrupt luminal closure due to vascular elastic recoil and dissection.

A direct surgical approach for stenosis of the petrocavernous portion of the ICA is not feasible, and the external carotid-internal carotid bypass would not be expected to have a beneficial effect on OA flow. Endovascular treatment with stents is technically not difficult and can affect OA flow directly by normalization of flow pattern in the ICA. We therefore believe that stenting offers a safe, effective, and minimally invasive treatment option for AMF being caused by atherosclerotic stenoses of the petrocavernous portion of the ICA in patients, in whose antithrombotic therapy fails.

Conclusion

Hemodynamic insufficiency in retinal vasculature due to a stenosis of the intracranial ICA is a possible etiology of AMF. Stent placement can be considered a safe and effective treatment option in these patients, in particular when medical treatment with antiplatelets and anticoagulation has failed.

References

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Commentary

Ocular ischemic syndrome(OIS) is defined by a constellation of sign and symptoms and occurs secondary to atherosclerotic carotid artery stenosis of more than 90%, resulting in a 50% reduction in ipsilateral perfusion pressure. Amaurosis fugax is one of the symptom of OIS that include visual loss and pain.

Until recently, carotid endarterectomy was the only option to reverse carotid artery stenosis surgically. Numerous prospective, randomized controlled studies have been performed to evaluate the efficacy and safety of carotid endarterectomy. Two studies, the North American Symptomatic Carotid Endarterectomy Trial and the European Carotid Surgery Trial, showed that symptomatic patients with 70% to 99% extracranial stenosis had between a 38.5% and 65.4% relative risk reduction of developing an ipsilateral cerebral vascular accident.

But total occlusions of the carotid artery and stenotic lesions above the second cervical vertebrae were not amenable to surgical intervention. Carotid artery angioplasty with stenting has been performed in patients with intracranial carotid artery stenosis, resulting in improved cerebral perfusion.

To my knowledge, there has been the first report in the Japanese literature of stenting in the cervical carotid artery for OIS in 2001. And then, in 2004, american ophthalmologists reported three patients with OIS that had been treated with intracranial carotid artery angioplasty and stenting. As authors, I believe that intracranial carotid artery stenting for OIS that is not amenable to surgical intervention is the alternative option.

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