Amaurosis Fugax Associated with Ophthalmic Artery Stenosis: Clinical Simulation of Carotid Artery Disease

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SUMMARY A 72 year old woman complained of transient loss of vision in the left eye. She had undergone a left carotid endarterectomy 10 years previously. Reduced ophthalmic artery pressure was found on non-invasive carotid artery testing and cerebral angiography was performed. No lesion was evident in the carotid artery, but significant ophthalmic artery stenosis was identified. We report this case as showing the occurrence of amaurosis fugax in association with ophthalmic artery stenosis.

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TRANIENT MONOCULAR BLINDNESS (amaurosis fugax) in older patients is a hallmark of atherosclerotic disease in the carotid artery. The etiology is believed to be primarily from emboli from ulcerated plaques, but can also occur with reduction in flow through a markedly stenotic carotid vessel. Occasionally, no cause is found in the carotid artery. Local disease in the retinal vasculature usually produces permanent visual loss with retinal infarction. We report a 72-year-old woman who had undergone left carotid endarterectomy in 1969 for transient weakness of the right side and in 1979 developed transient loss of vision in the left eye. Oculoplethysmography revealed reduced ophthalmic artery pressure in the left eye. Angiography showed no evidence of left carotid disease, but did demonstrate occlusive disease in the left ophthalmic artery. This mimicked occlusive disease of the carotid artery both clinically and on non-invasive testing.

Patient Report

A 72-year-old right-handed woman complained of 2 episodes of transient loss of vision in the left eye 5 days apart. Ten years prior to this, she had had 2 episodes of transient weakness of the right leg associated with speech deficit and confusion. An aortic arch arteriogram revealed marked stenosis of the left internal carotid artery and carotid endarterectomy was performed.

On this admission, the patient was alert and oriented. She complained of memory deficit, but this was not detected on objective testing. There was some dysprosody of speech, but no errors were made and there was no dysnomia. Pupillary reflexes, visual fields and extracocular motions were within normal limits. There were 2 small hemorrhages in the left fundus near the upper nasal border of the disc and one linear hemorrhage at the inferior temporal border. Motions of face, tongue, jaw and palate were normal. There was mild weakness of the right hand. Deep tendon reflexes were 2+ and symmetric bilaterally. There was decreased graphesthesia to written numbers on the right hand and some extinction of the right hand to touch on double simultaneous stimulation. Pin prick, vibration and position sense were intact.

Investigations

Because of the recent onset of amaurosis fugax, the clinical impression was recurrence of atherosclerotic disease in the left carotid artery. Non-invasive carotid artery testing was performed. Oculoplethysmography by the method of Gee showed ophthalmic artery pressure of greater than 110 mm Hg on the right and 100 mm Hg on the left with a brachial blood pressure of 180/110 (fig. 1A). Supraorbital Doppler examination showed antegrade flow bilaterally, but no augmentation on superficial temporal compression (fig. 1B). This was interpreted as being suggestive of hemodynamically significant reduction of pressure in the carotid artery circulation on the left.

Computerized axial tomography showed multiple old infarcts in the left cerebral hemisphere with ipsilateral ventricular displacement and prominent sulci. An aortic arch arteriogram was performed, revealing a normal aortic arch, carotid arteries and vertebral arteries, but prominent stenosis of the left ophthalmic artery with delayed filling (fig. 2).

Discussion

Transient loss of vision in one eye has been associated with a wide variety of illnesses, including migraine, Raynaud's disease, arteritis and papilledema. However, in the older age group, it is predominantly due to atherosclerotic disease and is thought to be related to temporary occlusion of the retinal vessels by emboli from atheromatous plaques in the carotid artery. This probable etiology is based on the observations by Fisher of white streaks in the retinal vessels during attacks of amaurosis fugax with
AMAUROSIS FUGAX AND OPHTHALMIC STENOSIS/Weinberger et al.

Distal dilatation and beading of the arteries and veins. Hollenhorst observed bright plaques at bifurcations of retinal vessels during and following attacks of amaurosis fugax. However, Gerstenfeld reported similar white streaks in a 30-year-old patient undergoing an attack of amaurosis fugax who had an internal carotid occlusion above the origin of the ophthalmic artery and no evidence of atherosclerotic disease in vessels proximal to the ophthalmic on angiography. Therefore, it is possible that these white streaks may occur without embolic cause. However, several authors have observed amaurosis fugax in patients with completed internal carotid occlusion who have had embolic retinal episodes, possibly through retrograde flow into the ophthalmic artery from the external carotid.

Others have witnessed amaurosis fugax on what appeared to be an ischemic basis. Fisher observed the fundus of 2 patients having amaurosis fugax who did not have white streaks. They both had decreased ophthalmic artery pressure due to ligation of the carotid artery in one man and the aortic arch syndrome in one woman. Sundt et al. have observed retinal ischemia in patients with transient non-embolic flow alterations and in patients with orthostatic cerebral hypoperfusion without systemic decrease in blood pressure. Dyll et al., photographed the fundus of a man during an episode of amaurosis fugax who had roughening and narrowing in the uncoiled siphon of the carotid artery just proximal to the origin of the ophthalmic artery. No white streaks were seen; only generalized pallor and narrowing of the vessels. David has reported a patient with a similar lesion in the carotid siphon with amaurosis fugax, possibly on an embolic basis. Cogan found central retinal artery occlusion in 6 of 25 patients with amaurosis fugax and no evidence of carotid artery disease though most patients with central retinal artery occlusion have permanent visual deficits. Therefore, it appears that amaurosis fugax can occur with decreased carotid perfusion pressure or local ischemia as well as from embolic sources. Nevertheless, there is a high correlation between amaurosis fugax and carotid artery disease. Marshall and Meadows found carotid bruits in 17 of 80 patients with amaurosis fugax and carotid lesions on angiography in 16 of 27 patients.

In our patient, ophthalmic artery stenosis was associated with 2 episodes of amaurosis fugax, but no permanent visual loss. The symptoms could have been due to focal ischemia or to autoembolization into the retinal circulation from the ophthalmic artery. Since hemorrhages were observed in the retina a few days following the attacks, embolization may be more likely.

Ophthalmic artery stenosis is an uncommon finding on cerebral angiography and has not previously been associated with amaurosis fugax. Gross et al. reported ophthalmic artery stenosis as a cause of false positive pulse delay as measured by oculoplethysmography in 2 of 287 patients studied arteriographically. They did not describe whether the patients had any visual symptoms. This figure gives some range of the incidence of ophthalmic artery stenosis in patients with cerebrovascular disease. In our patient, the oculoplethysmographic indication of hemodynamic abnormality suggested significant carotid artery disease and prompted concern of renewed stenosis of the left carotid artery. This study was performed by the Gee type of oculoplethysmography in which the actual ophthalmic artery pressure is measured.

This report is of note not only as showing another cause of misleading noninvasive carotid artery testing; it also points out that transient ischemic symptoms
FIGURE 2. Left common carotid angiograms in lateral projections. A) The distal common carotid artery and proximal segments of internal carotid and external carotid arteries are well opacified. No significant stenosis is noted. B) Subtraction film of early arterial phase. The left ophthalmic artery is filled poorly. C) Arterial phase one second later. Approximate 50% stenosis is noted at the origin of the left ophthalmic artery (arrows).
generally attributed to emboli from large proximal vessels such as the carotid artery can also arise from atherosclerotic disease in the distal cerebral vasculature.

References

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