Visual Aspects of Extracranial Internal Carotid Artery Disease

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The visual symptoms of ipsilateral internal extracranial carotid disease are sudden transient monocular visual loss (amaurosis fugax); sudden persistent visual loss and ischemic eye pain.

The funduscopic signs include a normal fundus, a visible retinal embolus, occlusion of the central retinal artery (CRA) or branch retinal artery, venous stasis low-pressure retinopathy, anterior ischemic optic neuropathy (AION) and hypertensive retinal signs and arcus senilis restricted to the nonoccluded side.

Monocular Amaurosis Fugax

Amaurosis fugax is a major visual symptom that may herald stroke. The four types of monocular amaurosis fugax are 1, due to transient retinal ischemia; 2, due to retinal vascular insufficiency; 3, due to angiospasm; and 4, idiopathic.

Amaurosis Fugax Type 1

The visual disturbance in amaurosis fugax type 1 is a sudden attack of partial or complete transient monocular blindness (TMB), lasting seconds to minutes, with total recovery. Partial visual impairment may be described by the patient as a grayout, as an ascending or descending curtain (altitudinal hemianopia, 20/35 cases), or as a blind moving sideways across the eye (hemianopia, 6/35 cases). Occasionally, the patient will describe moving tracks of light. Ipsilateral headache is rare (2/35 cases) (1).

In 1952, Fisher (2) drew attention to the association of amaurosis fugax of this brevity, with contralateral hemiplegia. Since then, amaurosis fugax has come to be regarded as one variety of carotid artery distribution transient ischemic attack (TIA). Like other TIAs, this symptom means the patient has an above average risk of stroke. By extrapolation from data for TIAs in general, the risk of stroke and/or death is probably about 10%
5. Vision in Extracranial Carotid Artery Disease

per annum (3), but death more often is due to ischemic heart disease than to stroke (4). In a recent study (5), cerebral vascular symptoms (TIA or stroke) occurred in 22 patients (43%) either before or after amaurosis fugax. Of 44 patients with no cerebral ischemic symptoms before the onset of amaurosis fugax, 15 (34%) subsequently had TIAs or strokes. Yet, the neurologic prognosis for the patient who has isolated amaurosis fugax still remains difficult to determine on the basis of clinical findings.

The majority of attacks of amaurosis fugax type 1 are due to embolism of fresh thrombus into the ophthalmic circulation from a source in the carotid artery, notably, the bifurcation of the common carotid artery in the neck or from the aorta or heart. Careful funduscopv looking for retinal emboli or other vascular changes (eg, central retinal or branch artery occlusion), ischemic optic neuropathy, or asymmetric hypertensive retinopathy is the most important examination to perform next to auscultation of the neck for a carotid artery bruit. The retinal microcirculation is visualized best if the pupil is dilated with 10% phenylephrine (Neosynephrine) eye drops after the visual acuity is measured and the pupillary response recorded. The ophthalmoscopic appearance of a retinal embolus can provide specific information about the embolic material and its possible source.

Bright, yellowish, glinting lipid emboli (Hollenhorst plaques) are the most common emboli seen in the eye. They are associated mostly with atheromatous changes of the ipsilateral carotid artery or aortic arch disease (6,7). The embolic material has been confirmed to be cholesterol.

Calcific emboli are characteristically matte-white, nonscintillating and somewhat wider than the blood column. Calcific emboli may be dislodged by the surgical manipulation of calcified heart valves at the time of valvulotomy or may occur spontaneously from rheumatic valvular vegetation.

Some of the circulating microemboli that pass through the retina, so-called migrant, pale emboli, are believed to be composed of platelets. Their occurrence is associated with thrombocytosis. The emboli that occur after myocardial infarction fall into the category of fibrin plugs. They are especially frequent in patients who have neurologic complications after open heart surgery. The heart lesions traditionally associated with thromboembolism have been myocardial infarction producing mural thrombus; mitral stenosis, with or without atrial fibrillation; and vegetative valvular lesions, bacterial or nonbacterial. Previously unrecognized potential sources of emboli have come to light: mitral-annulus calcification may be the origin of cerebral and retinal emboli in the elderly (8–10), and the prolapsing mitral valve may be the source in younger patients (11–13). In the case of embolic seeding from a myxoma of the heart, the histopathology of the embolus is like that of the original tumor (14).

Clinical experience has shown that the risk of permanent visual loss in patients with amaurosis fugax type 1 is relatively low, perhaps 3% per annum. In one series only 11% of patients with CRA occlusion and 18%