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Amaurosis Fugax: Diagnostic and Therapeutic Aspects

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Abstract:

Amaurosis Fugax: Diagnostic and Therapeutic Aspects ■ The three to eight-year follow-up study of 34 patients with unilateral amaurosis fugax (AF) suggests the following points: (1) A significant number of frequent unilateral AF patients suffer from small ulcerative plaques or irregular stenoses without any bruit in the neck or major changes in ophthalmodynamometry (ODM). The most important diagnostic test in these cases is angiography. (2) Surgery in patients with frequent unilateral AF seems to be more successful than anticoagulation. (3) No treatment cannot be considered a policy of "benign neglect" and may result in serious complications. (4) Cholesterol plaques, carotid bruits, and abnormal ODM are suggestive of carotid artery disease in unilateral AF. However, the absence of these signs is not helpful in differentiating carotid artery from cardiac origin of the emboli. (5) Heart disease may be the main etiological factor in a few patients with AF. It plays a major role in mortality regardless of the type of treatment.

Additional Key Words carotid endarterectomy antiplatelet aggregation visual loss cholesterol plaques aspirin ulcerative plaques anticoagulation

Introduction

 \Box In 1875 Gowers¹ reported a patient with retinal embolus and hemiplegia. This first report of amaurosis fugax was in a patient who had heart disease. In 1905 Chiari² suggested carotid artery origin for retinal emboli. Hunt³ (1914) pointed to the relationship between amaurosis fugax and contralateral hemiplegia, and carotid artery disease. Fisher⁴ (1952) pointed out the importance of carotid artery disease in amaurosis fugax (AF) and contralateral hemiplegia. He demonstrated the migrating white plugs⁵ in the retina originating from the carotid artery, or through its collaterals.⁶ Since then three types of emboli have been reported to be present with unilateral AF: cholesterol plaques,7,8 white plugs consisting of fibrin platelet masses,^{9, 10} and large white emboli originating from the heart and resulting in more severe damage to the retina.¹¹⁻¹³

Denny-Brown^{14, 15} emphasized the role of changes in systemic blood pressure in AF. This seems to be more important in bilateral AF in the presence of vertebrobasilar artery disease¹⁴⁻¹⁶ than in unilateral AF.

This is a report of 34 unilateral AF patients

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followed from three to eight years, emphasizing the diagnostic and therapeutic aspects of this condition. It demonstrates the importance of carotid ulcerative plaques in the majority of patients. In addition, the etiological and prognostic role of heart disease is emphasized.

Methods

A diagnostic and therapeutic study of patients who had amaurosis fugax (AF) was carried out. AF was defined as a painless, transient (a few seconds to a few minutes) visual loss which was noted to be unilateral and only occasionally bilateral. Transient attacks of blindness due to migraine, transient visual obscuration due to papilledema, and cortical blindness were excluded from this study.

Thirty-four patients have been followed from three to eight years. The symptoms consisted of a sudden onset of a "shutter closing," a "curtain dropping," dimming of vision, or a scintillating scotoma. After a few seconds to a few minutes, the vision would return. The attacks would vary in frequency from a few a month to several times a day.

Results

Complete neurological workup and angiographical studies revealed that carotid artery disease was the most likely cause in 32 patients. Carotid bifurcation ulcerative plaques were demonstrated in 18 patients, stenosis in 12 patients, and carotid occlusion in two patients (fig. 1). In addition, two of the patients with carotid stenosis had bilateral carotid stenosis, and in two, the stenosis was in the intracranial carotid but proximal to the origin of the ophthalmic artery. Carotid artery stenosis was defined as narrowing of

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Ulcerative, rough, and smooth plaques seen on angiography of AF patients.

the arterial lumen by at least 50%, and occlusion as obstruction of the flow of contrast of the cervical internal carotid regardless of its collateral flow pattern. Ulcerative plaques were diagnosed when irregularity of the contrast borders occurred usually with delayed segmental washout of contrast as well as the welldefined "crater" in the plaque (fig. 1). A plaque was considered irregular when the borders of contrast were undulating to a minimal degree without a definable segmental area of penetration into the plaque. All carotid bifurcations were visualized in at least two views (lateral and oblique), frequently with additional frontal and varying oblique projections. It is recognized, however, that the radiological diagnosis of

TABLE 1

Comparison of Clinical Manifestations With Angiographical Findings in 34 Patients Suffering From Amaurosis Fugax

Unitateral AF (34 cases)	Carotid alcerative plaques (18 cases)	Carotid stenosis* (12 cases)	Carotid occiusion (2 cases)	Heart disease with no carotid lesion (2 cases)	Totel
Cholesterol plaques	4	2			6
White plugs with field defect				1	1
Migrating white plaques		2			2
lpsilateral carotid bruit	2	8			10
Ipsilateral low ODM	1	5	1		7
Contralateral low ODM	1		1		2

*Bilateral carotid stenosis in two cases.

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FIGURE 2

Funduscopic findings in AF patients: left top and bottom: "white plugs" in two patients with rough plaques at carotid bifurcation. Right top: cholesterol plaques in a patient with carotid bifurcation ulcerative plaque. Right bottom: retinal infarction with field defect in a patient who died of heart attack. Autopsy showed ventricular mural thrombosis, and no carotid disease.

ulceration is only 80% to 85% accurate.^{17, 18} Controversy continues as to whether irregular plaques may be a source for cerebral emboli.^{17, 18}

In two patients no carotid disease was demonstrated. One patient suffered from a recent myocardial infarction, and the other had aortic valvular stenosis.

The age range was 49 to 77 years; 21 patients were male, and 13 were female. The correlation of carotid bruit and ophthalmodynamometric (ODM) changes with the angiographical findings is recorded in table 1. The ulcerative plaque group (fig. 1), which consisted of a majority of the patients, was less likely to have a carotid bruit or abnormal ODM. The carotid stenosis patients, on the other hand, were more likely to demonstrate a unilateral carotid bruit as well as an abnormal ODM. Contralateral carotid bruit was heard in six patients. In general, there was a low correlation between the bruits over the carotid artery and angiographical findings¹⁹ (table 1). However, bruits were more commonly heard among the unilateral AF than the bilateral AF patients (two out of ten of the latter group).

Three types of emboli were noted in the retinal examination of these patients. Six patients showed cholesterol plaques (fig. 2); four patients demonstrated ulcerative plaques in the carotid bifurcation, and the other two demonstrated carotid stenosis. One patient showed a white plug with field defect. This patient suffered from a recent myocardial infarction, and had no evidence of carotid artery disease (fig. 2). Two patients who had carotid artery stenosis revealed migrating white plugs. In two other patients (both of whom had carotid stenosis) central retinal artery thrombosis was seen.

The ODM difference between two eyes was measured in 18 of the AF patients, with a difference of 35/15 gm as compared to the same study in ten other

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TABLE 2

Results of Treatment in 34 Patients With Amaurosis Fugax (Three to Eight Year Follow-Up)

Unilateral AF 3-8 yr. follow-up (34 cases)	Dead	Hemiplegic	Blind	No change	Improved
Surgical treatment (17 cases)	1 (MI)	2			14
Anticoagulation treatment (9 cases)			2	1	6
Aspirin treatment* (5 cases)	1 (MI)		1	4	1
Sulfinpyrazone treatment† (5 cases)		4	1	4	1

*Three patients treated with aspirin later had surgery, and one had to be transferred to coumadin treatment group due to persistent symptoms.

[†]Two patients treated with sulfinpyrazone later had surgery, and one had to be transferred to coumadin treatment group due to persistent symptoms.

MI = myocardial infarct.

transient ischemic attack patients who showed a difference of only 7.5/5.5 gm between the two eyes.

The results of treatment are summarized in table 2. The treatment was decided according to the patient's general condition, angiographical findings, and the clinical judgment regarding surgical treatment or anticoagulation. In general, if the patient refused surgical treatment, anticoagulation would be tried unless there were reasons to contraindicate anticoagulation. Three to eight-year follow-up study of these patients revealed that the best result was obtained in the surgically treated group (table 2), and the next best result was in the anticoagulation group. Anticoagulation, in the form of heparin, was more effective than coumadin therapy with attacks of AF, while the patients were treated with heparin as compared to coumadin therapy. There were approximately 40% less attacks of AF per month in the coumadin group compared to the "no treatment" group. The "no treatment" group consisted of the patients who were not treated with heparin, coumadin or surgery. In the anticoagulation group of patients, the anticoagulation consisted of heparin therapy for 48 hours, followed by coumadin therapy as long-term treatment.

Two patients in this study died from myocardial infarction. Altogether seven of these patients had a

history of heart disease, and the five surviving patients continued to have attacks of angina or overt coronary artery insufficiency before and after treatment for amaurosis fugax.

The results of treatment were compared with those of Ehrenfeld et al.²⁰ and Marshall et al.²¹ (table 3). It was concluded that unilateral amaurosis fugax needs to be approached aggressively from a therapeutic standpoint. The major cause of death was myocardial infarction.

Discussion

This study is a review of our experience with AF. It does not pretend to be a prospective, double-blind study. Obviously, an ideal human study of aspects of cerebrovascular disease is hampered by logical lack of willingness on the side of the clinician to enter a highrisk patient into a double-blind study which entails such risky treatments as surgery or anticoagulation. Moreover, a randomized medical study could not be accomplished because of the poor response of many patients to anticoagulation, or antiplatelet aggregation agents which necessitated the transfer of such patients to a surgical group (table 2).

Antiplatelet aggregation therapy²²⁻²⁶ sounds promising, but more experience is needed before

TABLE 3

Comparative Results in Patients With Amaurosis Fugax

	Ehrenfeld et al. (1966), %	Marshall et al. (1968), %	Hooshmand et al. (1974), %
Carotid disease	100	55	94.1
Appropriate bruit	65	20	29.4
Morbidity, no treatment	100	16	100
Morbidity, anticoagulation	Ś	Ś	22.2
Morbidity, surgery	6	Ś	11.8
Mortality	Ś	23.7	5.8

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definite conclusions are drawn. Our limited experience with five patients treated with aspirin (600 mg per day) and five patients treated with sulfinpyrazone (800 mg per day) followed from three to five years (table 2) has been unimpressive. For this reason, we have classified as the "no treatment" group any patient who did not undergo surgery or anticoagulation in forms of heparin and coumadin. As seen in tables 2 and 3, the morbidity is so high in the "no treatment" group that no treatment cannot be considered any longer as a "policy of benign neglect."

Even though the major cause of mortality in these patients seems to be due to arteriosclerotic heart disease, the morbidity among the survivors can be influenced by a more aggressive therapeutic approach to this disease (tables 2 and 3).

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