# AMAUROSIS FUGAX – AN ALARM SIGNAL FOR CEREBRAL-VASCULAR ACCIDENT

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***Abstract:*** *Amaurosis fugax consists of partial or total loss of sight (temporary or short-lived), representing quite frequently a retinal transient ischemic accident. The sudden loss of sight for seconds or minutes maximum is a symptom which is mostly ignored and considered insignificant by the patients, but which can have very serious consequences. Precocious tracking and the right treatment of the cause of the disease may not only save the patient's sight, but sometimes, even his life. In the etiology of the disease are frequently impugned vascular factors (carotid and ophthalmic artery atherosclerosis, atherosclerotic or cardiac embolism), homodynamic factors (diminution of the perfusion pressure in the central artery of the retina in ipsilateral carotid stenosis with deficient collateral circulation through the Willis polygon, retinal vasospasm), immunological and inflammatory diseases, ATH, iatrogenic causes (CAET, carotid stenting). The diagnosis imposes, beside a detailed ophthalmologic examination, laboratory analysis and specific cardio-vascular exploration (GKE, cardiac and cervical-cerebral vascular Doppler ultrasound, cerebral TC/NMR with angiography sequence, angiofloralretinography, classical angiography). The treatment (which can be drug or surgical) addresses exclusively to the cause, fugitive amaurosis being a criterion for CAET in case of severe symptomatic carotid stenosis.*

***Cuvinte cheie:*** *acuitate vizuală, accident vascular*

***Rezumat:*** *Amauroza fugace constă în pierderea parţială sau completă a vederii (temporară şi de scurtă durată), reprezentând frecvent un accident ischemic tranzitor retinian. De cele mai multe ori, dispariţia bruscă a vederii pentru secunde sau maxim minute, este un simptom ignorat şi considerat neimportant de către pacienţi, dar care poate avea consecinţe extrem de grave. Depistarea precoce şi tratarea corectă a cauzei nu numai că poate salva vederea pacientului, dar uneori - chiar şi viaţa acestuia. În etiologia bolii sunt incriminaţi frecvent factori vasculari (ateroscleroza carotidiană şi a arterei oftalmice, embolia de cauză aterosclerotică sau cardiacă ), factori hemodinamici (scaderea presiunii de perfuzie în artera centrală a retinei în stenoze carotidiene ipsilaterale cu circulaţie colaterală deficitară prin poligonul Willis, vasospasmul retinian), boli inflamatorii şi imunologice, HTA, cauze iatrogene (TEAC, stentarea carotidiană). Diagnosticul impune pe langă un examen oftalmologic amănunţit, analize de laborator si explorări cardio-vasculare specifice (EKG, ecografie Doppler vasculară cervico-cerebrală şi cardiacă, CT/RMN cerebral cu secventa de angiografie, angioflororetinografie, angiografie clasică). Tratamentul (care poate fi medicamentos sau chirurgical) se adresează exclusiv cauzei, amauroza fugace fiind criteriu pentru TEAC în caz de stenoză carotidiană severă simptomatică.*

## INTRODUCTION

Amaurosis fugax or the transient loss of sight represents the sudden loss (partial or total) of sight, which has occurred at one or both eyes, which typically lasts for seconds or minutes, after which it comes back to normal. A lot of patients ignore these passing losses of sight and do not consult an ophthalmologist or they go to the ophthalmologist very late when they have completely lost the sight by the appearance of the retinal vascular accident. Not being given a diagnosis or treated on time, the patients having amaurosis fugax may suffer very serious consequences, such as the loss of sight at the respective eye, cerebral vascular stoke or acute myocardial heart attack. The word *amaurosis c*omes from Greek and it means *to darken*, this term being used to describe the loss of sight of any kind. The word *fugax* comes also from Greek and it means *fugitive*. Together, these two terms represents today a serious symptom of a vast pathology. Detailed amaurosis, detailed neurological and ophthalmologic examination, together with specific and systemic tests, allow tracking the main cause and a quick start of the treatment. The most frequent causes of amaurosis fugax are: circulatory (ischemic), ocular or neurological, the circulatory ones (embolic or hypo perfusion) being, by far, the most involved in the occurrence of amaurosis fugax.

Retinal ischemia (or visual nerve ischemia) is very frequently mentioned in the occurrence of amaurosis fugax. The causes of ischemia are multiple: carotid atherosclerosis, or ophthalmic artery atherosclerosis, sever carotid stenosis and ophthalmic artery stenosis (with hypo perfusion phenomena), cardiac embolism, hyper blood clotting, systemic hypo perfusion (hypo volemia, heart failure, arrhythmia, and severe anaemia), arthritis with gigantic cells (vasculitis which manifests itself through amaurosis fugax, anterior ischemic visual neuropathy and even plosion of the central retinal artery), retinal vasospasm, migraine, drug taking.

The visual affections that are involved in the differential diagnosis of amaurosis fugax are various: the syndrome of dry eye, irritation, keratitis, assault of intermittent glaucoma, drusen buried at the head of the visual nerve, hind

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vitreous discoloration, retinal disruption, orbital tumors, intraocular bleeding, retinal vasospasm etc. that is why the detailed ophthalmologic examination is essential for al the patients that have amaurosis fugax.

Among the neurological causes it is worth mentioning visual neuritis (frequently occurring in the case of demyelinating disease such as multiple sclerosis), papillary edema, phenomena of compression of the visual nerve (through tumors, thyroid ophthalmopathy, traumatism), migraines, SEL, etc.

**Positive diagnosis**

The first step, extremely important, in settling the positive diagnosis and then the etiological one, is the history of the disease. Amaurosis fugax is a frequency in the ophthalmologic practice, rather described by the patient (who comes to the ophthalmologist after the respective episode has disappeared) than being materialized at the moment of the examination. That is a reason for which, in such circumstances, the history is extremely important, insisting on disorders of visual ability (whether it is mono or binocular, total or partial, one or more episodes, associated visual symptoms) and on known visual or systemic affections. The answer to all these questions guides the physician towards the possible etiology.

Although it is impossible many times, it is very important to determine if the visual disorder was *monocular* or *binocular*. Monocular amaurosis fugax may be due to an ischemia inside the ipsilateral carotid artery or due to a previous disease of the visual chiasm (at the eyeball or the visual nerve). Whereas bilateral amaurosis fugax indicates a previous process, involving visual chiasm, visual tract or radiations and even visual cortex.

Another important element in history is the duration of the visual disorder, which again can guide us towards its etiology. Thromboembolic episodes (carotid or of other origin), typically lasts for 1-15 minutes (they exceed an hour rarely). Visual disorders which accompany papillary edema are very short, by mere seconds, whereas those that accompany the classic migraine typically last from 10 to 30 minutes.

**The description** of the visual disorder is another useful element for settling the etiological diagnosis. The negative visual phenomena – the fogginess of the image or the total loss of sight (total or partial) – are specific for retinal ischemia. A very suggestive element of retinal ischemia is the *curtain* or *shadow* which descends over the eyeshot (it may rarely occur from down to up). The positive visual phenomena such as photopsia or scintillations – are very suggestive for migraines. There have been described too a few precipitant factors of amaurosis fugax. Regarding carotid stenosis, the change of the neck posture or position, by a passing diminution of the blood flow through artery, may cause amaurosis fugax. Regarding severe carotid stenosis, the visual disorder may sometimes occur post fed, which is made possible by vascular theft. Regarding the same severe carotid stenosis (as well as in the arthritis with gigantic cells), exposure to strong light may induce the occurrence of amaurosis fugax (the so-called retinal claudication).

In the orbital formations, the movement of the eyeballs in a certain direction may cause the reduction of the blood flow of the retina, choroids or the visual nerve, and implicitly, amaurosis fugax. In demyelinating diseases that affect the visual nerve, it is very frequently met transient loss of sight (or aggravation of visual ability) after a hot bath or physical exercise (Uhthoff phenomena).

*Personal pathological antecedents* of the patient must always be mentioned. Older age, the history of diabetes, arterial hypertension or dyslipidemia are suggestive for atherosclerotic cardiovascular disease. At the same time, a well-known cardiac disorder may indicate a thromboembolic phenomenon. Whereas a family history of migraine syndrome may lead us to a migraine. The studies have shown that orthostatic hypotension at diabetes patients may cause a reduction of the blood flow in the visual artery with about 100% when they suddenly move from supine. That is how each diabetic retinopathy can be associated with amaurosis fugax.

The symptoms associated to amaurosis fugax are important indicators in the differential diagnosis. For instance, most of the thromboembolic phenomena are not painful. That is why, the vision disorder associated to a periorbital pain or to a headache indicates a possible migraine, an assault of glaucoma or of gigantic cells arthritis. At that, the association between amaurosis fugax and contra lateral hemiplegia is a great indicator of a severe occlusive carotid disease.

Detailed history must be compulsory correlated with the data obtained in the complete ophthalmologic examination (which contains testing visual ability, biomicroscopic examination of the preceding visual pole, measuring intraocular tension, examination of the fund eyes = testing the eyeshot).

**Specific investigations**

Laboratory tests (hemoleucogram and complete lipid gram together with other biochemical tests, coagulogram, possibly thyroid hormonal tests, HSV and RCP) are part of the evaluation of the patients with amaurosis fugax. At patients with monocular amaurosis are to be made some noninvasive tests to evaluate carotid and ophthalmic circulation – Doppler carotid or angio TC or angio NMR. Internal carotid ateromatosis with stenosis at this level is the most frequently involved in amaurosis fugax of circulatory nature. When amaurosis fugax is binocular, then it must be done a CT examination or cerebral NMR, insisting upon occipital lobe and upon the route of the visual nerve. The GKE and echocardiography must be done to all the patients whose history and clinical evaluation suggest the possibility of the existence of cardiogenic embolism or heartbeat disorder.

**Ways of treatment**

After establishing the positive diagnosis and the etiological diagnosis, the treatment of the main cause is started.

Visual affections will be treated by the ophthalmologist.

Vascular and cardio embolic causes, once they have been diagnosed, will get specific treatment to prevent cardiac and arterial embolism, anticlotting, antigregging, statin. Carotid stenosis, depending on the degree of stenosis and location, will be treated conservatorily through medicines or they will make carotid angioplasty with stent or CAET in specialized centers of vascular surgery. There will be identified and treated the vascular risk factors.

Tumor pathology is the privilege of neurosurgery and possibly to oncology.

The cooperation between the ophthalmologist, neurologist, neurosurgeon and vascular surgeon is essential for saving the sight and for preventing sever damage, life threatening, to amaurosis fugax patients.

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