TRANSIENT MONOCULAR VISUAL LOSS (TMVL) AMAUROSIS FUGAX (AF)

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Abstract: *Transient visual loss decrease of vascular cause is monocular and secondary to a brutal and temporary deficit in ocular perfusion by reducing blood flow in the retinal artery, ophthalmic artery or ciliary artery with transient retinal ischemia. It may be followed by permanent decline of view after a major ischemic attack (stroke). The urgent diagnostic evaluation is always required with the rapid establishment of the appropriate treatment.*

Cuvinte cheie: *hipoperfuzie oculară, ischemie retiniană, stroke cerebral hemisferic*

Rezumat: *Scăderea tranzitorie a vederii de cauză vasculară este monoculară şi secundară unui deficit brutal şi temporar de perfuzie oculară prin reducerea fluxului sanguin în artera retiniană, artera oftalmică sau artera ciliară cu ischemia temporară a retinei. Poate fi urmată de scăderea permanentă de vedere după un accident ischemic major (stroke) întotdeauna fiind necesară evaluarea urgentă diagnostică şi instituirea rapidă a unui tratament adecvat.*

The transient visual loss produced by the temporary ciliary and retinal arterial ischemia is monocular and reversible.

Retinal arterial ischemia and progressive optic nerve ischemia (13) can be generated by ocular or general causes:

- closed angle glaucoma in the episodic closure of the angle, when the transient decrease of view can be accompanied by pain and proceeded or associated with light-coloured halos;
- transient increase of the intraocular pressure;
- abnormal transparency of the anterior chamber in hyphaema can cause a variable vision termination (seconds, hours);
- cornea alterations in keratoconus;
- congenital anomalies of the optic disc may give episodic transient decrease of vision on short-term;
- orbital tumours (hemangioma, osteom) can intermittently compress the central retinal artery (CRA) causing AF;
- rarely, the occlusion of the central retinal vein (CRV) can give transient vision disorders, refractive errors in diabetes by increasing blood glucose.

The transient decrease of the visual acuity (VA) may have varying lengths with different clinical manifestations:(2)

- seconds under the form of visual eclipses in: intracranical pressure, optic disc drusen, abnormal optic nerve (ON) when the change of view takes seconds, rarely more than that;
- less than 10 minutes in nonembolic hypoperfusion ischemia (more often than in embolism) under the form of nonspecific positive visual phenomena in the patients with venous congestion and acute carotid dissection;
- variable duration in: ocular migraine (visual aura, bilateral, homonymous topography); glaucoma with subacute closed angle, orbital mass, perioptic meningioma;
- transient decrease of the visual acuity with pain, headache and/or periorbital pain may be present in: homolaterally internal carotid artery dissection, ocular ischemic syndrome, temporal arteritis Horton, retinal migraine (diagnosis of elimination).

The transient visual decrease of vascular cause can be achieved by vascular perfusion deficit in the ophthalmic artery, central retinal artery or branch and inadequate blood flow at the level of the optic nerve (posterior ciliary arteries).

The emboli present in TMVL people come from the atheromatous plaques of the carotid bifurcation, aortic arch.(9)

The atheromatous formations (1) are most commonly located in the common carotid artery bifurcation in the internal and external carotid and in the carotid siphon: hypertension, diabetes, hypercholesterolemia, and smoking, risk factors that should be known and treated.

Cardiac embolism (2) is due to many causes: ventricular aneurysm, wall hyperkinesia, infectious/non-infectious endocarditis, mitral valve disease, myxoma, cardiac arrhythmias (atrial fibrillation, paroxysmal arrhythmias), persistent permeable foramen ovale with right-to-left cardiac shunt.

Chronic secondary ocular hypoperfusion (5,9,13) is accompanied by transient loss of vision - minutes, hours, through the poor regeneration of the visual pigments in the retinal photoreceptors in vasculitis, giant cell arteritis, severe stenosis of the carotid artery, ophthalmic artery stenosis, arc aortic (in severe atheromatosis,Takayasu arteritis) (figure no. 1).

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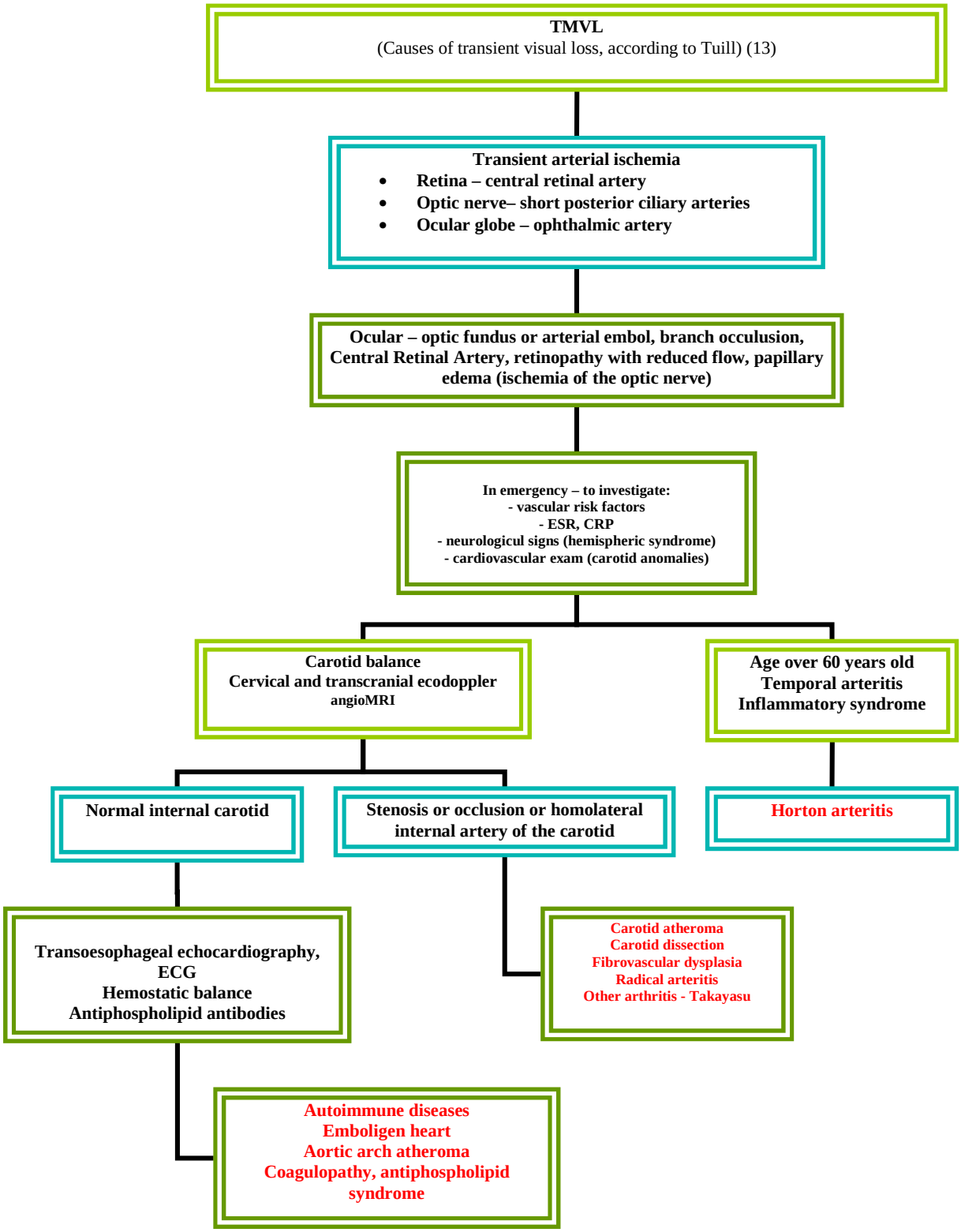
Figure no. 1. Transient monocular visual loss



The transient decrease of view can be accompanied by various clinical signs under various causal conditions:

- Brilliant criss-cross in cortical visual migraine;
- Head pain, sensitive scalp, jaw claudication in ciliary artery occlusion in association with giant cell arteritis;
- Intermittent eye pain leading to angle closure glaucoma or ischemia associated with giant cell arteritis;
- Headache, pains in neck, jaw, forehead in cervical carotid dissection;

- Presyncope in systemic hypotension or in hyperviscosity syndrome;
- Ipsilateral Horner's syndrome in carotid disease, carotid dissection.

Clinically speaking, the transient loss or decrease of view is accompanied by the loss or temporary painless lowering of view, a loss of 4.10 AV can be totally or sectorally unprovoked and unpredictable, which takes seconds, but also hours being followed by the full recovery of the view. Between episodes, the ophthalmological examination is normal or there are pre-existing retinal vascular abnormalities. Rarely, neurological signs can be associated: dysphasia, contralateral hemiparesis, paraesthesia.

The decrease of view in the retinal ischemia is persistent, in 1-2% of the cases with the permanent loss of vision and it is secondary to the central retinal artery or branch occlusion.

The ophthalmoscopic examination of the fundus of eye – the fundus of eye examined after mydriasis may be normal or there may be signs of preexisting ocular disorders, sometimes highlighting the local ocular transient decrease of view: retinal embolism, retinal ischemia, venous stasis retinopathy.

The neurological examination is mandatory.

The positive diagnosis of the transient visual decrease is generally anamnestic and requires comprehensive eye examination in order to highlight disorders associated with visual transient dizziness: papillary stasis, giant cells arteritis, optic disc drusen, migraine.

Carotid diseases (5,13) are the most common cause of fugitive amaurosis in the elderly patients. In these patients, the stroke lasts 2-10 minutes and can be repeated several times a day, being associated with crises of sensorial hemiparetic deficit, aphasia.

The visual recovery may be sectoral or altitudinal.

The evolution of the patient with transient visual loss is different according to etiology and age.(11)

In retinal stroke with the intersection of the central retinal artery or one of the branches, the loss of vision may be permanent through the occlusion of the central retinal artery or branch.

In the hemispheric cerebral stroke, cerebral infarction in the transient ischemic stroke may be accompanied by the transient decrease of vision through the atherosclerotic internal carotid occlusion, sometimes followed by the irreversible loss of vision through the occlusion of the central retinal artery.

The patients with atheromatous internal carotid artery stenosis have a risk over 50% in correlation with the degree of stenosis to develop irreversible loss of sight through retinal stroke and/or brain stroke.

The risk of death in the patients with transient loss of vision and atheromatous carotid stenosis is approximately of 4% per year

Laboratory tests are useful in the diagnosis orientation towards an atherosclerotic process, an important risk factor, but the presence of the positive inflammation tests, increased ESR, CRP, fibrinogen in the elderly, may suggest giant cell arteritis.

The ultrasound examination can individualize the carotid embolism source, cardiac (rarely), carotid dissection or the carotid atheromatosis. Transcranial Doppler assesses the intracranial blood flow in the patients with severe carotid stenosis at risk of stroke. Ophthalmic artery Doppler may show decreased flow in the patients with normal carotid artery, suggesting stenosis or ophthalmic artery occlusion. Transthoracic or transesophageal ultrasound can highlight the embolus source at cardiac level or the aortic arch atheroma.(6)

The treatment in the transient visual loss (2,3) is differentiated according to the cause of the decrease of sight.

The patients should be clinically investigated from the ocular, cardiac, carotid, aortic arch point of view, requiring when needed, prolonged treatment with antiplatelet agents (INR 2.5) - aspirin, clopidogrel for the prevention of stroke in the patients with atrial fibrillation and in the patients at high risk of cardiac embolism and hypercoagulable state. If necessary, antiatherosclerotic statins will be administered.

If vasospasm is suspected, antiaggregants will be administered - antiplatelet agents and calcium channel blockers that may reduce the frequency of attacks, avoiding the vasoconstrictors.

Carotid endarterectomy in the patients with extended internal carotid stenosis who have 70% risk of AF is rarely indicated in the transient visual loss, being indicated only in the patients with high risk of ipsilateral internal carotid stenosis.

Retinal, cerebral stroke prevention, of the cardiovascular events in the patients with AF requires the control of the risk factors: smoking, alcohol consumption, treatment of the coronary artery disease, heart arrhythmias, heart failure, valvulopathies, hyperlipidemia treatment, hyperglycemia treatment.

The transient decrease of view is also present in the ocular ischemic syndrome. The ocular ischemic syndrome (3) represents the reduction of the ocular blood flow through the anterior or posterior artery ischemia, through chronic ocular hypoperfusion secondary to a severe homolateral carotid stenosis brought about by atherosclerosis, being the additional sign of the carotid thrombosis.

The risk factors are: males (60-90 years old), smoking, diabetes, hypertension, coronary heart disease.

Etiologically speaking, ischemic ocular syndrome is caused by: carotid occlusion (most commonly), carotid dissection, temporal arteritis, papilledema in cerebral pseudotumours, stenosis or ophthalmic artery aneurysm, vascular heart abnormalities.

The ocular symptoms are variable and start with: amaurosis fugitive, gradual loss of vision, transient loss of the visual acuity unilaterally, sometimes associated with orbital pain in older people through difficult regeneration of photoreceptors, visual disorders with delayed visual recovery after the exposure to bright light (photostress test).

Ocular signs:(6)

- anterior segment-striae and corneal edema, discreet Tyndale albumin (pseudo-inflammatory ischemic irritation), pupil with less reactive, rubeosis and iris atrophy, neovascular glaucoma, cataract;
- posterior segment - venous dilatation, thinning of the retinal arteries, retinal hemorrhages in the peripheral area sometimes papilledema, exudates in vitreous superficial hemorrhage in the medium periphery of the retina, retinal ischemia nodules, subretinal neovascularisation occurs in 2/3 cases, CME (cystoid macular edema), systolic pulse, cherry red macula.

The positive diagnosis requires full clinical examination, ophthalmodinamometry, carotid Doppler – it shows severe stenosis 80%, angiography; the digital pressure on the eye may produce arterial pulse.

The differential diagnosis (4,11) of the ocular ischemic syndrome is made by:

- edematous obstruction of the central vein of retina that resembles to the presence of unilateral retinal hemorrhages, venous dilatations, nodules and it is

differentiated through normal arterial perfusion and numerous hemorrhages;
- diabetic retinopathy in which hemorrhages and venous dilatations are present, proliferative retinopathy as in the ocular ischemic syndrome and it is distinguished by bilateral touch and hard exudates;
- hypertensive retinopathy is similar to the ocular ischemic syndrome by reducing the arterial calibre and focal constriction, hemorrhages, nodules and it is differentiated by bilateral touch and the absence of venous changes.

Table no. 1. Differential diagnosis of the ocular ischemic syndrome (2,3)

	Ocular ischemic syndrome	**Non-ischemic obstruction of the central vein of the retina**	**Retinal displacement (RD)**
Age	70-80	70-80	Variable
Uni/bilateral	Unilateral 80%	Unilateral	Bilateral
Veins of the eye fundus	Dilated	Tortuous dilated	Dilated
Disc optic	Normal	Edema	Normal
Ophthalmodinamometry	Reduced	Normal	Normal
Retinal hemorrhages	Medium	Medium-severe	Medium moderate
Microaneurysms	In the mean periphery	Variable	Posterior pole
Hard exudates	Absent	Rare	Present
Angiofluorography (AFG) transit time arteries/veins stagnation time in the retinal vessels	Extended Long arterial time	Prolonged Predominant venous time	N Absent

The treatment is controversial (5,10) and requires: topic corticosteroids and mydriatics for the anterior segment complications, intraocular pressure (IOP) decrease in order to reduce the perfusion pressure, carotid surgeries - endarterectomy to avoid an accident and/or death, and in the ocular ischemic syndrome with neovascular glaucoma and subretinal neovascularisation (14) – pan-retinal photocoagulation.

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