IPSILATERAL OPHTHALMIC ARTERY STENOSIS IN AMAUROSIS FUGAX: A CASE REPORT

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ABSTRACT

Introduction: Amaurosis fugax is caused by an abrupt reduction of blood flow to the retina. Determining the etiology of amaurosis fugax should ensure proper management.

Case Report: A 47-year-old female patient complaint about the first episode of sudden vision loss in her right eye and was referred to our hospital. The vision loss resolved spontaneously, however, we found a 3 mm long stenosis at her right ophthalmic artery during magnetic resonance angiography. She had clinical histories of untreated hypertension and dyslipidemia. Transient ischemic attack (TIA) was suspected and unfractionated heparin, aspirin, antihypertensive agent, and statin were given. Treatments were maintained, the symptoms had not recurred in the following 6 months after the event.

Discussion: Stenosis of the ophthalmic artery is very rare. It occurs in approximately 2% of patient suffering from amaurosis fugax. In our case, stenosis of the right ophthalmic artery due to thromboembolism might cause retinal ischemia leading to a transient visual loss.

Conclusion: This case suggests stenosis of ophthalmic artery as the cause of amaurosis fugax.

Keywords: amaurosis fugax, ipsilateral, ophthalmic artery, stenosis


INTRODUCTION

Amaurosis fugax describes monocular or binocular loss of vision, typically lasting seconds to minutes.1,2 This symptom is generally caused by a manifestation of thromboembolic event in which atherosclerotic debris originating in the carotid artery or the aortic arch temporarily disrupts blood flow in the branch or central retinal arteriolar network.2,3 Immediately treating a patient with amaurosis fugax in whom aortic or carotid atherosclerosis is detected with an anti-platelet agent and a statin are consistent established guidelines.4 Migraine, retinal detachment, optic disc drusen or papilledema, glaucoma, emboli from thoracic arteries or external carotid, mitral valve prolapse, hypercoagulable state, giant cell arteritis (GCA), vasospasm, postural hypotension, acute sharp pain, nose-blowing, and sensitivity to cold, however, can also be the cause of amaurosis fugax.4-5 Therefore, proper management depends on the underlying cause.

CASE REPORT

A 47-year-old female patient presented with the first episode of sudden vision loss in her right eye for approximately 30 minutes, prior to reaching the hospital. She experienced no headache. She had clinical histories...
of hypertension and dyslipidemia, and was not under medical therapy. She was fully alert, and visual acuity of both eyes had returned to 20/20 by the time of initial clinical examination. Her blood pressure was 158/77 mmHg. Slit-lamp and ophthalmoscopy examination findings were unremarkable (Figure 1). Intraocular pressure (IOP) in the right eye and left eye was 19.7 mmHg and 18.0 mmHg respectively, and was measured with applanation tonometry. Laboratory studies demonstrated high levels of total cholesterol (222 mg/dL), low-density lipoprotein/LDL (139 mg/dL), triglycerides (169 mg/dL), and normal level of high-density lipoprotein/HDL (55 mg/dL). Coagulation parameters including d-dimer, fibrinogen, and platelet aggregation studies were within normal limits. Other neurological and hematological parameters were within normal ranges. Magnetic resonance angiography revealed a 3 mm long stenosis at the right ophthalmic artery (Figure 2 and 3). Optical coherence tomography (OCT) examination was within normal limits (Figure 4 and 5). The amaurosis fugax resolved spontaneously. We treated her case as retinal transient ischemic attack (TIA), and

Figure 1. Normal color fundus photograph of right eye (A) and left eye (B).

Figure 2. MR angiography, blue arrow demonstrated a 3mm-long stenosis at the right ophthalmic artery (axial image).
she received unfractionated heparin during hospitalization. Aspirin, candesartan, and rosuvastatin regimens were initiated promptly.

**DISCUSSION**

Braat et al.\(^6\) reported a patient with isolated ophthalmic artery stenosis and one episode of amaurosis fugax. During the episode, fundoscopy revealed a cholesterol crystal in the lower temporal branch of the central retinal artery with hemorrhage of the macula. Micro-embolism of the ophthalmic artery seemed to cause amaurosis fugax. During the episode, fundoscopy revealed a cholesterol crystal in the lower temporal branch of the central retinal artery with hemorrhage of the macula. Micro-embolism of the ophthalmic artery seemed to cause amaurosis fugax.

![Figure 3. MR angiography, blue arrow demonstrated a 3mm-long stenosis at the right ophthalmic artery (sagittal image).](image)

![Figure 4. Normal Optical Coherence Tomography (OCT) of the right eye (OD)](image)
fugax, and anticoagulation was maintained successfully for preventing attacks. Nakajima et al. postulated that high frequency of amaurosis fugax attacks in the ophthalmic artery stenosis could be explained by platelet hyperaggregability since aspirin proved effective in preventing attacks. Cerebral angiography may show focal stenosis at the origin of the central retinal artery or ophthalmic artery. Stenosis of the ophthalmic artery is very rare. According to a previous study, the prevalence of ophthalmic artery stenosis is approximately 2% in patients suffering from amaurosis fugax.5

In our case, we did not perform the visual field examination because our hospital did not have the device. The patient’s ophthalmic examination finding was unremarkable, and despite the absence of significant pathologic finding of the right internal carotid on MRA, we found stenosis of right ophthalmic artery due to thromboembolism which might cause ischemia of the retina, leading to transient visual loss. Amaurosis fugax and TIA are clinical conditions with shared etiology that both increase the risk of ischemic stroke, as the patient had several risk factors, notably dyslipidemia and hypertension. The treatment for retinal TIA is similar to that of cerebral TIA9, therefore unfractionated heparin and aspirin were administered along with candesartan and rosuvastatin to manage the underlying conditions.

TIA suspected patients should be evaluated as soon as possible after an event. If retinal TIA is present within 72 hours, American Heart Association recommends admission to a hospital setting. For longer presentation, between 3 to 7 days, hospitalization is still recommended if the patient already had a known untreated source of ischemia (carotid stenosis, atrial fibrillation).10 The assessment of vascular risk factors is most important. Patient should be assessed for hypertension, dyslipidemia, diabetes mellitus,

Figure 5. Normal optical Coherence Tomography (OCT) of the right eye (OS)
cardiac disease, and tobacco use. Laboratory studies, electrocardiogram, and neuroimaging studies should be done. Treatment first aimed at controlling and treating the underlying vascular risk factors.

**CONCLUSION**

Amaurosis fugax is caused by an abrupt reduction of blood flow to the retina, with multiple possible etiologies. This case suggests stenosis of ophthalmic artery as the cause of amaurosis fugax. Anticoagulant, antiplatelet, angiotensin receptor blocker, and statin were given and treatment for underlying conditions was maintained. The patient remains under treatment for her hypertension and dyslipidemia, and symptoms had not recurred in the following 6 months after the initial (and only) event.

**REFERENCES**


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