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A Case Study of Amaurosis Fugax with Likely Subsequent Central Retinal Artery Occlusion

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Introduction

Amaurosis fugax is a type of transient ischemic attack (TIA) characterized by acute, painless monocular vision loss. Depending on the severity of ischemic impairment, ocular complications such as central retinal artery occlusions (CRAO) or branch retinal artery occlusions (BRAO) may develop and cause permanent vision loss. An episode of amaurosis fugax must be thoroughly assessed as it has been proven to be an indication of imminent cardiovascular and cerebrovascular events. This not only highlights the potential risks of amaurosis fugax but also its etiology, clinical presentations, and methods of management.

Case Presentation

A 74-year-old Causasian male with a medical history of prior strokes and cardiovascular risk factors initially presents at the Palo Alto VA urgent care with acute amaurosis fugax symptoms in the right eye. Examination findings are largely unremarkable save for a potential Hollenhorst plaque seen on Optos. He underwent right sided transfemoral carotid artery stenting less than two weeks after his initial visit. After being lost to follow up for over two months, his condition rapidly progressed likely due to a subsequent CRAO and he developed an ocular sequelae of neovascular glaucoma (NVG). These events were closely associated in time with an episode of ischemic stroke soon after his first follow up.

Discussion

Patients experiencing amaurosis fugax must undergo a complete vascular diagnostic workup. In cases that are caused by significant stenosis of the carotid bifurcation, a carotid endarterectomy (CEA) is highly recommended. However, the presenting patient was managed with carotid artery stenting. There are variable risks and benefits to both procedures, but neither can completely eliminate the threat of impending stroke. If left untreated, amaurosis fugax can lead to vascular-occlusive events such as CRAO. NVG is a potential, albeit uncommon complication that may develop and is managed by anti-vascular endothelial growth factor (VEGF) therapy and panretinal photocoagulation (PRP). Treatment of amaurosis fugax is directed at stroke prevention.

Conclusion

Amaurosis fugax should be recognized as a medical emergency as it signifies a potential cerebrovascular event. It is essential to place immediate referrals to stroke centers and appropriate medical professionals as well as monitor closely for ocular complications.

Introduction

Amaurosis fugax is defined as sudden, painless monocular vision loss usually lasting between 2 to 30 minutes.[1] Although uncommon, some may experience episodes lasting for several hours.[1, 2] It is typically seen in older patients with vascular risk factors such as hypertension, hypercholesterolemia, smoking, and heart disease.[1, 3] Patients often describe the event as a darkening or shadow hindering part of their visual field that typically self-resolves, and there may be 1 or multiple episodes.[2] This most commonly occurs as a result of an occlusion or stenosis of the internal carotid artery (ICA) with thromboembolic origins.[1] Depending on the severity of retinal hypoperfusion, patients may also present with concurrent or subsequent retinal occlusive disease. These conditions require careful ophthalmological and physical examination as they place patients at a high risk for cardiovascular and cerebrovascular events in the future.

Case Presentation

Initial Visit: 10/17/24

Patient JG, a 74-year-old Caucasian male, presented to Palo Alto VA urgent care with a complaint of sudden, painless, transient, monocular vision loss in the right eye (OD). He had experienced 5 episodes since that morning, each lasting from 5 minutes to 1 hour. His blood pressure at the time of admittance was 143/89 mmHg and he denied symptoms of headaches, scalp tenderness, and jaw claudication. He also denied any recent onset of flashes, floaters, and curtain veiling. His ocular health history detailed previous trauma from battery acid in the left eye (OS) and cataract surgery in both eyes (OU). A review of his medical history revealed a myriad of vascular risk factors. The patient had previously suffered a stroke in 2017 in which he intermittently lost right-sided function and a non-ST-elevation myocardial infarction (NSTEMI) in 2015. He was a heavy smoker and drinker, smoking 1 pack of cigarettes every day for the past 60 years and consuming approximately 12 beers per week. The only medication on file was Naproxen for his knees and back. His family history was unremarkable for glaucoma, macular degeneration, or other ocular diseases.

Visual acuities showed that he was light perception during transient episodes of vision loss but otherwise saw 20/200 with pinhole improvement to 20/60 OD. The left eye saw a baseline of 20/30-2. Pupils and extraocular muscle testing appeared normal, and there was no afferent pupillary defect (APD) noted. Confrontation visual fields (CVF) of the right eye showed intact central and right lower sectors, but constriction in all other quadrants. Each eye was instilled with 1 drop of Fluress in preparation for Goldmann tonometry. The intraocular pressure (IOP) measured 16mmHg OU. Slit lamp evaluation of the anterior segment was largely unremarkable. Lids and lashes were clear OU, conjunctiva and sclera were white and quiet OU, cornea had punctate epithelial erosions worse OS than OD, anterior chamber was deep and quiet OU, and the iris was round and reactive OU. The patient was pseudophakic OU with grade 2 posterior capsular opacification OU.

The patient consented to 1 drop of each topical dilation medication, tropicamide 1% and phenylephrine 2.5%, being instilled in each eye. Both eyes had a cup-to-disc ratio of 0.4 round with pink sharp margins, temporal peripapillary atrophy, and absence of disc edema or hyperemia. The macula was flat with no cherry red spot OU and the vessels were of normal caliber and non-tortuous OU. There was a posterior vitreous detachment (PVD) OD but OS was clear. The peripheral retina was clear of breaks, tears, detachments, retinal pallor, intraretinal/subretinal hemorrhages, and cotton wool spots OU. An Optos image and optical coherence tomography (OCT) of the macula was obtained for photo documentation and to evaluate for macular edema/atrophy. Upon further inspection on Optos, there was a potential Hollenhorst plaque in the superior branch arteriole OD that had not been seen earlier on the

dilated fundus examination. Macular OCT confirmed that there was normal foveal contour without intraretinal/subretinal hemorrhages OU. It also showed an area of potential drusen versus prominent choroidal vasculature temporally OD.

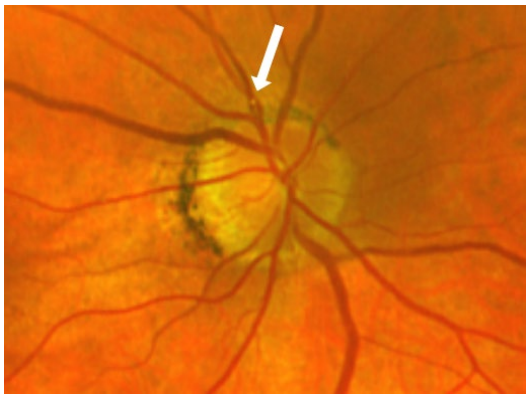


Figure 1: A close-up image of the optic nerve in the right eye shows a potential Hollenhorst plaque (indicated with the white arrow) in the first bifurcation of the superior arcade. No intra/subretinal hemorrhages, cherry red spot, or retinal pallor was observed.

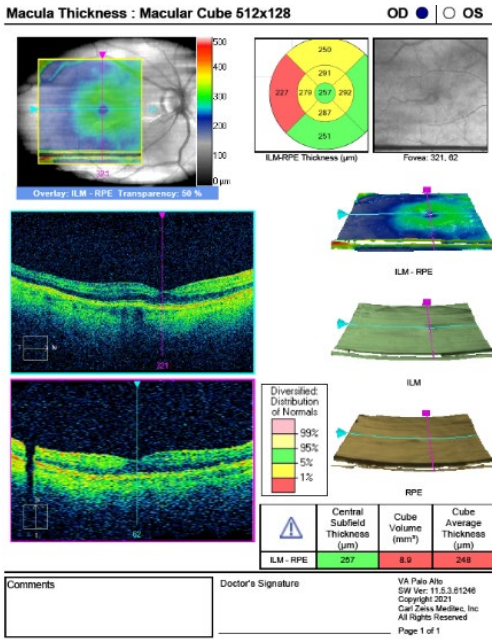


Figure 2: Spectralis macular OCT of the right eye depicts a normal foveal contour without intraretinal/subretinal hemorrhages. There is a potential drusen versus prominent choroidal vasculature temporally, which is represented by the slight deviation at the level of the RPE.

Differential Diagnoses

1. Amaurosis fugax

The nature of the patient's painless, monocular temporary vision loss along with its brief period of duration suggested that this was the top differential. However, it was essential to rule out all other possible etiologies before drawing this conclusion.

2. Central/branch retinal artery occlusion (CRAO/BRAO)

The central retinal artery supplies blood to the inner layers of the retina. An occlusion of this site leads to ischemic insult of retinal tissues. This is commonly seen in the form of retinal whitening with a cherry red spot, arteriolar attenuation, and segmented "box-carring" in vessels.[4] The presence of a retinal embolus may be observed in up to 40% of patients.[5] JG's posterior pole examination was unremarkable for most of the classic funduscopic signs, but the appearance of a Hollenhorst plaque highly suggested that he was at threat of an impending CRAO.

3. Central/branch retinal vein occlusion (CRVO/BRVO)

In contrast, the central retinal vein drains blood from the retina. Therefore, a blockage at this site causes the vein to burst and leak fluid into the retina. This can be observed by extensive retinal hemorrhages, dilated tortuous vessels, hard exudates, and cotton wool spots of ischemia.[6] However, JG's retina was clear with non-tortuous vessels of normal caliber.

4. Giant cell arteritis (GCA)

GCA is a chronic, idiopathic inflammation of medium and large arteries. It primarily involves the carotid artery, the major vessel supplying ICA which branches off into the ophthalmic artery. As a result, it is also one of the major etiologies of CRAO. The distinct signs of GCA include headache, jaw claudication, and scalp tenderness.[7] The patient had a painless onset of vision loss and denied all of these symptoms during case history.

5. Ocular ischemic syndrome (OIS)

OIS is caused by chronic hypoperfusion to the entire arterial blood supply. It is also commonly due to atherosclerosis of the carotid artery. Anterior segment signs include iris neovascularization (NVI) which can lead to neovascular glaucoma. In the posterior segment, one may be able to view narrowed arteries, dot blot retinal hemorrhages in the mid periphery, and neovascularization of the disc. It is also common to observe hypotony secondary to ischemic damage of the ciliary body, which limits the production of aqueous fluid.[8] The patient did not display these findings at this visit, placing it lower on the list of differentials.

6. Papilledema

Vision loss in papilledema occurs secondary to swelling and progressive loss of the optic nerve fibers.[9] This is a response to increased intracranial pressure and can be seen as bilateral swelling of the optic disc, absence of spontaneous venous pulsations, and relative sparing of the papillomacular bundle. It is often associated with headaches, diplopia, and tinnitus.[10] These signs and symptoms were not observed in the patient, so papilledema was ruled out.

A same-day stroke work-up with magnetic resonance imaging (MRI), transthoracic echocardiogram (TTE), carotid duplex, and neurology consult was recommended. Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) testing was also ordered to rule out GCA. He was instructed to perform ocular massages to promote blood flow and break up clots. The physician initiated maximum topical therapy of latanoprost, timolol, brimonidine, and dorzolamide to lower IOP to decrease the likelihood of CRAO/BRAO.

The MRI revealed multiple small infarcts within the right front and temporal lobes, likely a result of residual damage from the stroke in 2017. The MRA showed severe narrowing at origin of right ICA near 70%, but this grading was confounded by the small luminal size. A carotid ultrasonography demonstrated <50% stenosis of the right and left ICA. There were no abnormal findings on TTE and all serology testing was found to be within reference ranges. Unfortunately, the lipid panel, metabolic panel, and CRP tests were unable to be performed since the sample collected could not be stabilized.

Follow-up was originally scheduled for 1 week, but after neurology reevaluated the patient the next day, his vision had returned to baseline. He was directed to discontinue glaucoma drops and return with ophthalmology in 1 month. On October 28, 2024, 11 days after his urgent care visit, he underwent right sided transfemoral carotid artery stenting.

Follow-Up #1 (1/9/25)

The patient was lost to ophthalmology follow-up and returned 3 months later with a report of several more episodes of amaurosis fugax after surgery during approximately mid-November followed by a constant scotoma OD. According to the patient, he did not come in sooner since he had difficulties making an appointment. He had started taking aspirin 81mg, atorvastatin 40mg for stroke prevention, and varenicline 1mg to aid in smoking cessation. When asked about the glaucoma drops prescribed by the previous provider, he claimed that he had never received any of the medications. Visual acuity OD had decreased to count fingers at 2 feet but OS remained at 20/30. An ophthalmic technician worked up the patient and measured IOPs with Tonopen. Repeated measurements on the right eye showed 25, 26, and 47 mmHg and 16 mmHg in the left eye. It was re-measured yet again with Goldmann, confirming 26 mmHg OD and 16 mmHg OS. There was a new onset of 2+ APD OD, but a normal CVF had been restored.

Ocular health examination revealed significant changes in the right eye compared to the patient's initial presentation. A drop of proparacaine 0.5% was instilled in each eye and a Zeis gonioscopy lens was placed to examine the anterior chamber angle. There was development of sparse rubeotic vessels and hemorrhage circumferentially. This correlated with the appearance of rubeotic vessels at the margin of the pupil especially at 6-9 o'clock. All other anterior segment structure findings were stable to the prior visit. The patient agreed to be dilated for better view of the posterior segment. The optic nerve showed pallor and temporal neovascularization of the optic disc (NVD) without edema or hyperemia. The macula remained flat with no cherry red spot and the vessels at a normal ratio. While the presence of neovascularization was clear evidence of previous retinal ischemia, there were no sectors of retinal pallor, intraretinal/subretinal hemorrhages, or cotton wool spots. Ocular health of the left eye was similar to the initial presentation.

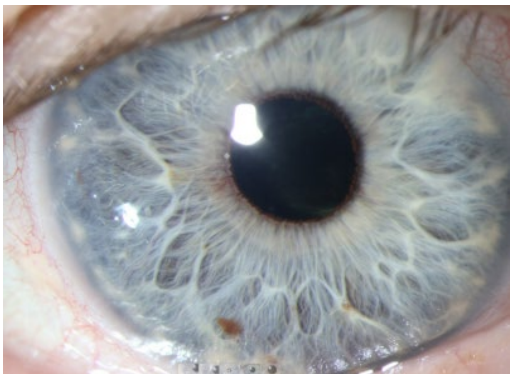
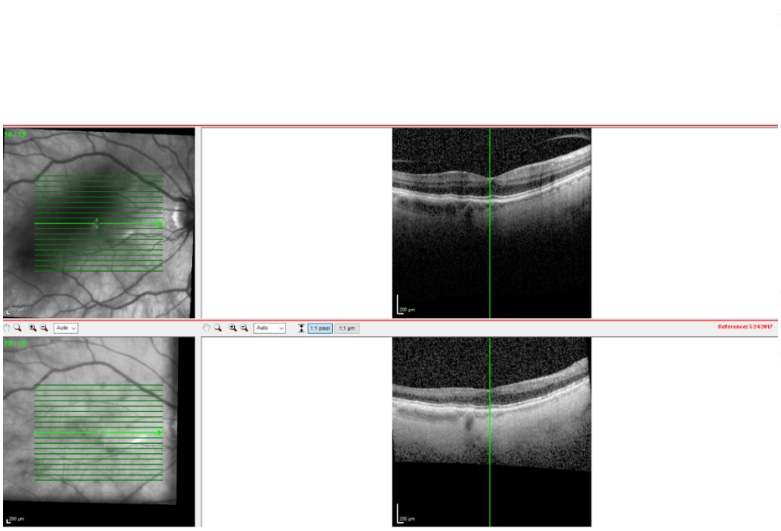


Figure 3: An external photo taken of the right eye highlights the rubeotic vessels at the 6-9 o'clock pupillary margins. The vessels extend outward into the iridocorneal angle, correlating with the neovascularization of the angle (NVA) seen on gonioscopy.

Additional diagnostic testing OD showed a loss of foveal contour with marked diffuse inner retinal thinning on macular OCT. The baseline retinal nerve fiber layer (RNFL) OCT was outside normal limits, with the superior nasal and nasal sectors exhibiting the floor effect. This is an indication that RNFL in these areas are so thin the machine is unable to further measure its structural damage. A second Optos photo was obtained for comparison. At this stage, the

Hollenhorst plaque had cleared but it captured the presence of temporal NVD and optic nerve pallor. He was diagnosed with neovascular glaucoma likely secondary to post CRAO OD and would follow-up with a retinal specialist for anti-VEGF therapy and PRP. In light of elevated IOP OD, he was instructed to instill latanoprost 0.005% in both eyes every night before bed until his next appointment.



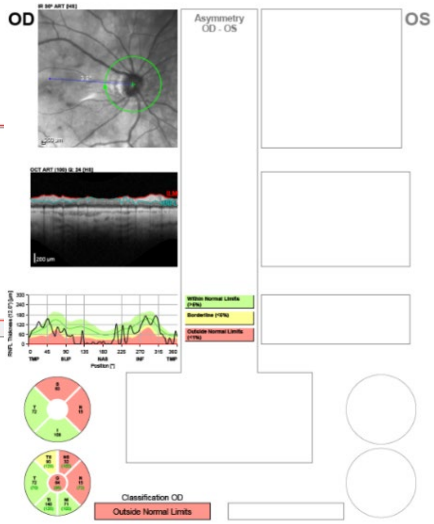


Figure 4 (left)**:** A comparison of the macular OCT of the right eye shows a loss of foveal contour (bottom) vs his initial visit (top). There was also marked thinning of the inner retinal layers.
Figure 5 (right)**:** A baseline RNFL demonstrated severe thinning of retinal fibers, especially in the global, superior nasal, and nasal sectors which were exhibiting floor effect. The superior temporal sector showed borderline thinning.

Three hours after his appointment, he called the telephone triage line with a complaint of sudden loss of muscle strength of his right leg and arm, although it should have been noted as left based on review of all subsequent notes. The triage nurse advised him to hang up and dial 911 immediately, which the patient obliged. He was hospitalized for 3 days with improvement after medical management. A repeat stroke work-up was done and he was reevaluated by neurology. Based on the updated MRI results, they diagnosed him with a minor stroke due to right ICA re-thrombosis post stenting. The plan was to recanalize the right ICA stent and start dual antiplatelet therapy (DAPT) for the next 3 months with initiation of a high intensity statin.

Follow Up #2 (1/14/25)

The patient returned to clinic for follow up with the retinal specialist. His medication list had been updated to reflect the addition of latanoprost and clopidogrel 75mg. His entering visual acuities were CF at 3 feet OD and 20/25- OS. The IOPs in the right eye were measured at 29, 25, and 26 mmHg and 13 mmHg in the left eye. He was noted to have grade 2 NVI on slit lamp evaluation. Upon dilation, the right eye's posterior segment showed mild pallor of the optic nerve and rare microaneurysms and hemorrhages in the macula. There was no emboli seen. Macular and RNFL OCT were repeated with similar findings to the prior examination. The patient was given the risks, benefits, and alternatives to intravitreal injection of ranibizumab-nuna (Byooviz) for treatment of neovascular glaucoma. After expressing informed consent, he was anesthetized with a subconjunctival injection of Lidocaine 2% then injected intraocularly with Byooviz 0.5mg using a 30-gauge needle in the right eye. He was given post-injection precautions and would return to clinic in 4-5 weeks for dilation and PRP OD.

Discussion

Amaurosis fugax is most commonly seen in patients 50 and older and with known risk factors for cardiovascular disease, such as diabetes, tobacco usage, obesity, and hypertension.[1, 2, 3] It is often referred to interchangeably as a TIA, although there is a small, yet distinct difference in the use of terminology. A TIA is defined as a transient loss of neurological function due to ischemia to the brain, spinal cord, or retina. The involvement of the retina is more specifically known as amaurosis fugax.[11] An episode of amaurosis fugax is described as sudden, painless monocular vision loss lasting between 2 to 30 minutes.[1] It is crucial for the clinician to be on alert for these symptoms as they are harbingers of ischemic stroke, which is estimated to be as high as 15-30% at 3 months.[12] Another study showed that there is a significant number of TIA patients who will experience a stroke within the first 2-7 days of the incident event.[13]

Its prevalence varies on its accompanying ocular vascular occlusive disorder. Hayreh et al[3] compiled a study showing its prevalence alongside an array of ocular occlusive conditions and determined that it was seen 12.18% with CRAO, 14.20% with BRAO, 15.38% with OIS, and 32.4% with GCA. Thromboembolism arising from the ipsilateral carotid artery is the most common source, attributing to nearly 20% of amaurosis fugax cases.[1, 3] Less observed mechanisms include retinal arterial vasospasm, hypoperfusion, retinal vein occlusion, compressive tumors, optic disc edema, and papilledema.[11] The American Heart Association/American Stroke Association (AHA/ASA) considers TIAs and consequently amaurosis fugax as a transient episode of neurological dysfunction without an associated infarction.[14] As a result, these patients are at a high risk of stroke and must undergo a complete vascular diagnostic workup.

A full amaurosis fugax evaluation must include a detailed case history, dilated ocular health examination, and diagnostic testing. Specifying visual symptoms, duration of vision loss, speed of onset and recovery, monocularity/binocularity, and associated neurological symptoms is in helpful in narrowing down differential diagnoses.[11] Clinicians should also assign a score using the National Institutes of Health Stroke Scale to determine if there are any neurological defects associated with stroke.[15] Ophthalmoscopy may reveal a refractile, yellow particle of cholesterol lodged within a bifurcation of the retinal arteries, known as a Hollenhorst plaque.[5] Nonreflective lesions suggest compositions of fibrin, platelets, calcium, or other material.[5] However, most dilated fundus exams in amaurosis fugax appear normal.[11] Nevertheless, it is vital to treat these conditions as a medical emergency and perform a stroke work up even in the absence of overt signs of damage.

Mbonde et al[11] released the most current guidelines on management of amaurosis fugax and TIAs in 2022. After careful clinical assessment, brain and neck magnetic resonance imaging and angiography (MRI and MRA) with diffusion-weighted imaging and head and neck computed tomography and angiography (CT and CTA) is recommended to search for possible infarcts. Baseline electrocardiogram is used to evaluate for atrial fibrillation and a transthoracic echocardiogram (TTE) identifies sources of cardiac emboli. Carotid doppler imaging analyzes the patency of the carotid arteries, allowing doctors to select patients who may be candidates for carotid endarterectomy (CEA) or stenting. Additional serology tests such as complete blood count, basic coagulation studies, hemoglobin A1C, lipid profile, and urine drug screening are helpful to identify vascular risk factors. In patients 50 years or older, erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) tests should be ordered to rule out GCA.

Mbonde's guidelines recommend CEA for patients presenting with symptoms of amaurosis fugax with significant stenosis of the carotid bifurcation. This is a surgical procedure during which calcified plaque buildup is removed from the carotid artery.[16] The proposed methods of management are divided into two treatment arms, one for symptomatic extracranial carotid

artery atherosclerosis and one for symptomatic intracranial large artery atherosclerosis. In the former, patients who have experienced an episode of TIA/amaurosis fugax within the past 6 months with ipsilateral severe (70-99%) carotid artery stenosis, are strongly advised CEA within 2 weeks of the incidence to lower the risk of subsequent stroke. It is still recommended moderate (50-69%) carotid artery stenosis if the perioperative morbidity and mortality is less than 6%. Since the risk of periprocedural stroke is higher with stenting, CEA is the preferred over surgical procedure in those 70 years or older.[11] But recall in JG's case, he underwent a transfemoral carotid artery stent. This decision is thought to be due to the severe narrowing of his right ICA and small luminal size. Placing a stent would likely enlarge the lumen and allow proper blood flow. Treatment for symptomatic intracranial atherosclerosis is aimed at stroke prevention. Patients are started on DAPT combination of clopidogrel 75mg and aspirin 325mg daily for 90 days after the TIA which is then followed by single antiplatelet therapy (SAPT). They are also started on a high intensity statin to lower LDL cholesterol to less than 70 mg/dL. Additional general measures include maintaining blood pressure at a systolic of 140 mmHg and lifestyle changes such as adoption of a mediterranean diet, smoking and alcohol cessation, and weight management.[11]

Although CEA and carotid artery stenting (CAS) reduce the risk of cerebrovascular events, it does not completely eliminate the threat. This is clearly evident with JG's case, as he suffered permanent vision loss and a minor stroke secondary to ICA re-thrombosis despite having undergone stenting. One study identified 5 major risk factors associated with ischemic or hemorrhagic stroke within 1 month post CAS placement: patients 80 years or older, symptomatic with TIA/stroke symptoms, a procedure within 2 weeks of symptoms, chronic renal failure, and diabetes mellitus.[17] Our case study encompassed two of these features since the patient presented with amaurosis fugax and stroke symptoms and had his procedure 11 days after his initial visit. Yet another study evaluated the causes of procedural strokes in patients treated with carotid artery stenting or endarterectomies within 30 days of revascularization.[18] Their research revealed that 97% of strokes associated with CAS were the result of infarction and involved the ipsilateral ICA. The 3 most common etiologies were carotid embolic, haemodynamic, and thrombosis or occlusion of the carotid artery.[18] Interestingly, more patients in the CAS branch had postprocedural strokes than in the CEA branch regardless of the mechanisms observed. Carotid embolic circumstances usually resulted in early postprocedural strokes as plaques were more likely to be dislodged during surgical intervention. Prolonged uncontrolled hypertension in patients with reestablishment of blood flow to previously ischemic cerebral tissue can lead to reperfusion syndrome, an inflammatory process leading to further damage to cells.[19] This further supports the need for strict control of blood pressure. This case study supported the researcher's third mechanism of action, as he had a re-thrombosis of the ipsilateral ICA and required recanalization of the stent.

Ocular complications following amaurosis fugax require prompt medical attention. In this case study, the patient likely developed a subsequent CRAO. While amaurosis fugax is defined by transient, monocular vision loss, CRAO is diagnosed by permanent, monocular vision loss due to compromise of central retinal artery blood flow.[4, 20] Its pathophysiology can be further divided into two different entities: nonarteritic and arteritic. More than 90% of cases arise from nonarteritic origins and are most commonly caused by ipsilateral carotid artery atherosclerosis, which is not dissimilar to the etiology of amaurosis fugax.[4] Arteritic CRAO is usually caused by GCA, however other vasculitic disorders have also been noted.[7] It is essential to conduct the proper laboratory tests to rule out GCA to treat the underlying etiology. Prognosis is typically better in the presence of a cilioretinal artery since it is supplied by the choriocapillaris. Extensive periods of ischemia leaves the retinal ganglion cells susceptible to damage. In one study investigating the survival time of the retina following CRAO in monkeys, researchers reported no detectable damage after 97 minutes, but extensive irreversible damage after about 240 minutes.[21] Though the retinal anatomy of a monkey cannot be directly applied to humans, it still

suggests that we have a small window of time before vision loss is irrevocable. There is a lack of conclusive evidence supporting successful treatments of acute CRAOs, in part due to the condition's time sensitive nature and difficulty with patient enrollment.[20]

It has been well documented that NVG is a known ocular complication seen with CRVO, but while uncommon, it can also be induced in CRAO. The incidence of NVG in CRAO ranges from 2-17%.[21] This incidence is thought to be lower because the retinal ischemia is so widespread that there is no viable tissue left to produce VEGF. There is a quicker onset of NVG after CRAO (5-9 weeks) as compared to CRVO (12-24 weeks).[22, 23] The NVI seen during JG's first follow up (about 12 weeks) was already diffusely spread within the iridocorneal angle, indicating that its onset was likely closer to the CRAO timeline detailed in literature. Some researchers suggest that this rarity is associated with an underlying atherosclerotic carotid artery disease[24] while others have theorized that it is due to failure of retinal reperfusion.[25] Regardless of its disease mechanism, PRP is the gold standard of initial treatment to prevent further growth of ocular neovascularization.[25]

Conclusion

Amaurosis fugax is a condition defined by sudden, transient, painless, monocular vision loss. It is a harbinger of cerebrovascular and cardiovascular events and should be regarded as a medical emergency. The presentation of amaurosis fugax should prompt clinicians to conduct a detailed case history, ocular health examination, and careful assessment of diagnostic serology and imaging testing. These patients are to be admitted to the emergency department and stroke center for urgent medical attention, then followed by referrals in cardiology and neurology. Eye care professionals should also follow up closely for possible ocular complications in the interim. In summary, the treatment and management of these cases are focused on minimizing the risk of stroke.

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