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Management of Acute Retinal Ischemia

Follow the Guidelines!

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Acute retinal arterial ischemia, including vascular transient monocular vision loss (TMVL) and branch (BRAO) and central retinal arterial occlusions (CRAO), are ocular and systemic emergencies requiring immediate diagnosis and treatment. Guidelines recommend the combination of urgent brain magnetic resonance imaging with diffusion-weighted imaging, vascular imaging, and clinical assessment to identify TMVL, BRAO, and CRAO patients at highest risk for recurrent stroke, facilitating early preventive treatments to reduce the risk of subsequent stroke and cardiovascular events. Because the risk of stroke is maximum within the first few days after the onset of visual loss, prompt diagnosis and triage are mandatory. Eye care professionals must make a rapid and accurate diagnosis and recognize the need for timely expert intervention by immediately referring patients with acute retinal arterial ischemia to specialized stroke centers without attempting to perform any further testing themselves. The development of local networks prompting collaboration among optometrists, ophthalmologists, and stroke neurologists should facilitate such evaluations, whether in a rapid-access transient ischemic attack clinic, in an emergency department–observation unit, or with hospitalization, depending on local resources. *Ophthalmology 2018;■:1–11 © 2018 by the American Academy of Ophthalmology*

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Acute retinal arterial ischemic events are classic causes of acute painless monocular vision loss. Transient or permanent occlusion of the central retinal artery or a branch retinal artery reflects acutely impaired blood flow in the anterior cerebral and ocular circulation and is associated with high cerebrovascular and cardiovascular morbidity and mortality.[1–3] Indeed, transient monocular vision loss (TMVL) of vascular origin is a retinal transient ischemic attack (TIA), whereas branch retinal artery occlusion (BRAO) and central retinal artery occlusion (CRAO) result in retinal infarctions, with mechanisms and causes identical to those of acute cerebral infarctions in the territory of the internal carotid artery.[1] Many health professionals and the public consider TIAs benign but regard strokes as serious. These views are incorrect. Strokes and TIAs are on a spectrum of serious conditions involving brain and eye ischemia, just as angina and acute myocardial infarction are part of the continuum of acute coronary syndromes.[4] It is therefore logical to combine vascular TMVL, BRAO, and CRAO as "acute retinal arterial ischemia" and to propose the same systematic management for these 3 entities. Although their respective visual outcomes are different, their overall significance and their systemic and neurologic implications are similar. Vascular TMVL can be compared to a cerebral TIA, whereas BRAO and CRAO are best classified as minor strokes, and all must be managed accordingly. In 2011 and 2013, the American Stroke Association[5] and the American Heart Association (AHA)[6,7] published a consensus statement defining central nervous system infarction (stroke) as "brain, spinal cord, or *retinal* cell death attributable to ischemia, based on neuropathological, neuroimaging, and/or clinical evidence of permanent injury." This statement clearly emphasizes that acute retinal arterial ischemia is a stroke equivalent and represents an ophthalmologic and medical emergency. Numerous excellent publications originating in the ophthalmic literature have helped understand and clarify the spectrum of acute retinal ischemia over the past 40 years. However, although a few authors have emphasized the need to urgently care for these patients with isolated visual loss, there is ample evidence that appropriate care of patients with acute retinal ischemia is too often delayed.[8] The aim of this article is to review the most recent data and recommendations regarding the acute management of patients with TIAs and minor strokes and to propose guideline-compliant strategies applicable to eye care providers who routinely see patients with acute visual loss from retinal arterial ischemic events. The outdated belief that acute retinal ischemia is of less concern than cerebral ischemia (and therefore may not need emergent care) must be revisited. It is time for a change in practice among eye care professionals.

Are Retinal Transient Ischemic Attacks Different From Cerebral Transient Ischemic Attacks?

Several studies[9–18] have reported that the risk of stroke after a retinal TIA is lower than the risk of stroke after a cerebral

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TIA. However, none of these studies concluded that retinal TIAs are benign, and most emphasized that although the risk of subsequent stroke may be lower than expected, the overall risk for cardiovascular events and death was the same as in the population of patients with cerebral TIAs, consistent with shared major vascular risk factors.[10–12,19,20]

What can account for this apparently lower risk of stroke after TMVL? The most likely explanation is that retinal arterial ischemia as the cause of TMVL is overdiagnosed in most large studies, contributing to the seemingly better vascular prognosis after a retinal TIA. It is indeed often extremely difficult to determine the cause of an episode of transient visual loss, and despite a detailed history and ocular examination, a diagnosis of presumed vascular TMVL often remains uncertain.[17,21,22] In our experience, nonvascular ocular causes and migrainous visual aura (often misinterpreted as being monocular by patients) explain many episodes of transient visual loss. Additionally, a number of recurrent isolated episodes of vascular TMVL may be related to central retinal artery vasospasm, which is a local (usually benign) disease not associated with higher cerebrovascular and cardiovascular risk[22] (Fig S1, available at www.aaojournal.org).

Even within the subgroup of patients with internal carotid artery stenosis and related ocular symptoms, there may be different individual responses to cerebral hypoperfusion. Collateral circulation plays a major role in protecting the ipsilateral hemisphere in patients with severe internal carotid artery stenosis. In these patients, flow is often diverted from the eye to the brain via the circle of Willis to maintain cerebral perfusion. Such patients classically have recurrent episodes of TMVL but no cerebral infarction because the brain is perfused at the expense of the eye.[23]

It has also been suggested that very small platelet-fibrin emboli will more readily become manifest when reaching the eye than when reaching the brain. Indeed, it is probable that very small retinal emboli resulting in focal retinal hypoperfusion do result in transient visual symptoms, whereas it is very unlikely that similar small cerebral emboli would produce neurologic symptoms obvious enough to be noticed by patients.[9,24] Additionally, because our eyes are constantly open when we are awake, we are more likely to perceive very brief episodes of visual loss than brief episodes of neurologic dysfunction, allowing us to notice "mini ocular TIAs," with relatively good prognosis. Such a theory would explain the not uncommon occurrence of small, multiple *asymptomatic* cerebral infarctions found acutely on magnetic resonance imaging (MRI) with diffusion-weighted imaging (DWI) sequences (DWI-MRI) in patients with TMVL.[8]

In the North American Symptomatic Carotid Endarterectomy Trial (NASCET),[9] patients with TMVL were more likely to subsequently have retinal infarctions whereas patients with cerebral TIAs were more likely to have cerebral infarctions, with a resultant lower risk of cerebral infarctions after retinal ischemia. This could be explained by the intraluminal streaming phenomenon in which recurrent vascular emboli tend to go to the same arterial branch.[25] It has been theorized that because particles originating from the heart are usually of larger caliber than those commonly originating from carotid atherosclerosis, cardiac emboli will more often reach the brain than the retinal circulation.[24] This is supported by laminar flow in large arteries in which various laminae are directed to different distal vascular beds. Large emboli tend to travel centrally in midstream and reach the most distal vascular bed, whereas small emboli originating from an atherosclerotic carotid wall will remain closer to the wall and be swept into the first arterial bifurcation they encounter (e.g., the ophthalmic artery), similar to the migration of rocks and debris in rivers (the streaming effect).[24–26] Such phenomena may explain why retinal artery occlusions are more commonly caused by carotid disease than by cardiac sources of emboli, as previously suggested in another study comparing risk factors for cerebral versus ocular ischemic events.[26]

Prognosis of Transient Ischemic Attacks

It is well established that TIAs offer an opportunity to initiate treatment that can forestall the onset of permanent disability.[4,27] Major advances in the urgent evaluation of TIA patients and in secondary prevention strategies have resulted in a dramatic decrease in the risk for major stroke after a TIA or minor stroke.[27–30] Previous studies conducted before the early 2000s estimated the risk of stroke and acute coronary syndromes between 12% and 20% during the first 3 months after a TIA or a minor stroke, with a large proportion of these strokes occurring very early after the first events.[31,32] The recent TIAregistry.org project[33] included 4789 TIA patients (including 172 patients with TMVL) who were enrolled over 2.5 years in 61 specialized TIA clinics by experienced stroke specialists. Seventy-five percent of patients were evaluated and treated within 24 hours of symptom onset. The reported rate of stroke and acute coronary syndrome was only 1.5%, 2.1%, 2.8%, 3.7%, and 5.1% at days 2, 7, 30, 90, and 365, respectively. However, this very low risk for recurrent stroke is likely explained by the excellent immediate care received by these patients in specialized stroke centers. Indeed, previous studies relying on rapid assessment of TIA and immediate initiation of aggressive secondary prevention showed that proven management strategies for TIA can reduce the relative risk of subsequent stroke by 80%.[34,35] A meta-analysis from 2007 evaluating the risk for stroke early after TIA[36] demonstrated a wide range of stroke risk among studies, with risks ranging from 0% to 12.8%, and a pooled stroke risk of 3.1% at 2 days (95% confidence interval [CI] 2.0–4.1) and 5.2% at 7 days (95% CI 3.9–6.5). Not surprisingly, the lowest risks (0.6% at 2 days [95% CI 0.0–1.6] and 0.9% at 7 days [95% CI 0–1.9]) were seen in studies of emergency treatment in specialized stroke services, and the highest risks (3.6% at 2 days [95% CI 2.4–4.7] and 11% at 7 days [95% CI 8.6–13.5]) were seen in population-based studies without urgent treatment. The publication of the EXPRESS study[34] and the SOS-TIA study[35] in 2007, both of which showed conclusively that immediate evaluation and treatment of TIA patients (cerebral and retinal TIAs) in specialized

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