AN OCULAR SIGN OF CARDIOVASCULAR DISEASE


Hollenhorst plaques may indicate serious systemic concerns. Here's what to do if you find them on routine examination.

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Retinal emboli are opacities that can be identified in retinal arterioles.[1] Although these emboli can result from fat, sepsis, or talc, they most commonly stem from vascular disease.[2] Retinal emboli are often detected incidentally on routine dilated ophthalmoscopic examination (Figure 1); are thought to arise from carotid arteries, coronary arteries, or cardiac valves; and typically appear transient.[1,2] The composition of the embolus, rather than its location, is what is important when it comes to migration and resolution. Up to 90% of retinal emboli migrate peripherally or disappear over time.[2]

The estimated prevalence of retinal emboli in the general population is 1.3%, and the estimated incidence is 2.9%.[1] Approximately 80% of retinal emboli originating from vascular disease are composed of cholesterol, followed by platelet-fibrin, and, lastly, calcium.[1] Risk factors for retinal emboli include older age, male sex, hypertension, total cholesterol, coronary artery disease, and a history of coronary artery bypass grafting. The strongest risk factor for retinal emboli, however, is smoking.[1]

Multiple studies suggest patients with retinal emboli have an increased risk of stroke-related, all-cause, and possibly cardiovascular mortality.[3,4] This article focuses on one particular type of emboli: cholesterol plaque, as it is the most common type seen during an optometric examination.

CHOLESTEROL PLAQUE

A Hollenhorst plaque, first described by Robert Hollenhorst, MD, in 1961, is a type of embolus formed from cholesterol deposits and typically originates from the ipsilateral carotid artery.[5]

These plaques appear as yellow, refractile emboli that become lodged at an arterial bifurcation.

A Hollenhorst plaque can dislodge on its own and may not produce any clinical signs or symptoms; however, in some cases, retinal ischemia may occur if the embolus does not dislodge and blocks perfusion of retinal blood in that area.[6] Symptoms may include sudden, painless, unilateral loss of vision, although visual acuity varies, depending on the location of the Hollenhorst plaque. There are many instances in which the presence of Hollenhorst plaque does not produce any symptoms. A retrospective analysis of The Blue Mountains Eye Study (n = 3,583) found a prevalence of retinal emboli in 1.4% of participants, all of whom were asymptomatic.[7]

The Beaver Dam Eye Study demonstrated that patients with emboli tended to be older; had higher total serum cholesterol levels; and had a longer history of smoking, angina, and coronary artery bypass surgery.[8] They also found that patients with retinal emboli were 2.4-times more likely to die from a stroke compared with their age-matched controls over a 7-year follow-up period.

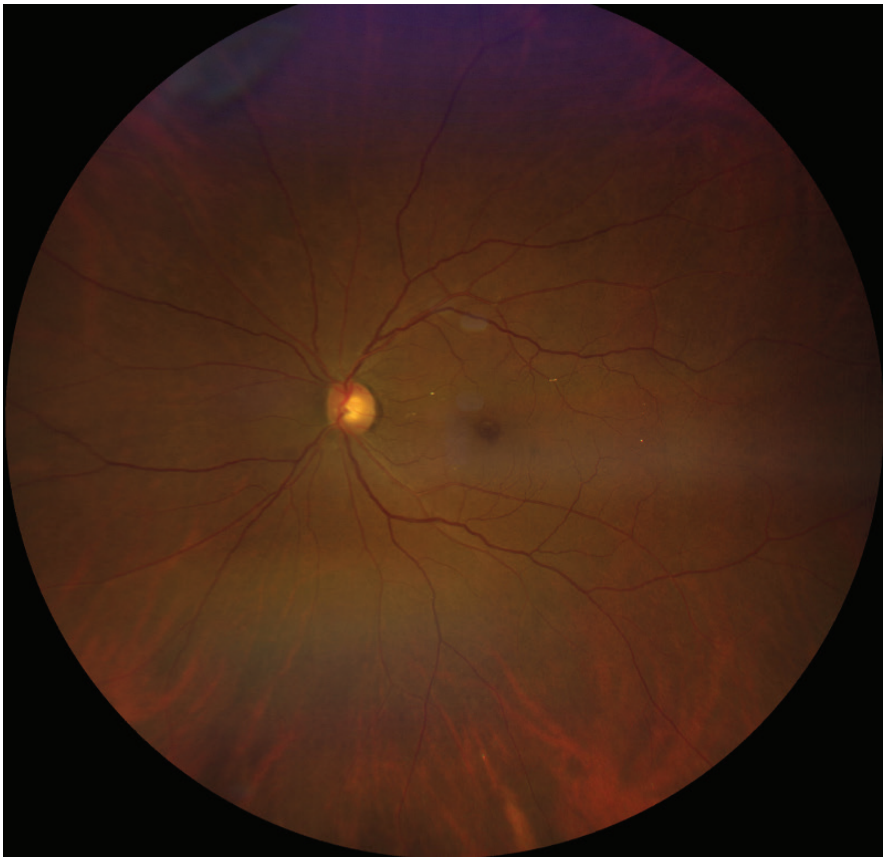

Figure 1. Fundus photo showing multiple retinal emboli.

SYMPTOMATIC HOLLENHORST PLAQUES

There is a consensus among clinicians that urgent workup is indicated if a patient is found to have symptomatic Hollenhorst plaques.[9] In addition to unilateral vision loss, patients may also experience amaurosis fugax, total loss of vision, or visual scotoma. Potential neurologic symptoms include headaches; numbness in the face, arms, legs, or one side of the body; dizziness; difficulties with balance or speaking; and fainting.

As Hollenhorst plaques are common characteristics of an artery occlusion, the presence of retinal ischemia or infarction warrants urgent management. A systematic review by Fallico et al concluded that 30% of patients with acute central retinal artery occlusion presented an acute cerebral ischemia on MRI.[10] This high rate supports the suggestion of prompt referral to the emergency department for neurologic assessment in such cases.

AT A GLANCE

- Risk factors for retinal emboli include older age, male sex, hypertension, total cholesterol, coronary artery disease, a history of coronary artery bypass grafting, and, most significantly, smoking.
- A Hollenhorst plaque is an embolus formed from cholesterol deposits; retinal ischemia can occur if the embolus does not dislodge and blocks perfusion of retinal blood in the area.
- The risk of cerebrovascular events with asymptomatic retinal emboli is as high as 12%.

ASYMPTOMATIC HOLLENHORST PLAQUES

A review by Ghoneim et al evaluated studies that included follow-up for patients with asymptomatic retinal emboli and found the risk of cerebrovascular events to be as high as 12%.[8] Significant carotid artery stenosis, defined as greater than 50%, was found to be present

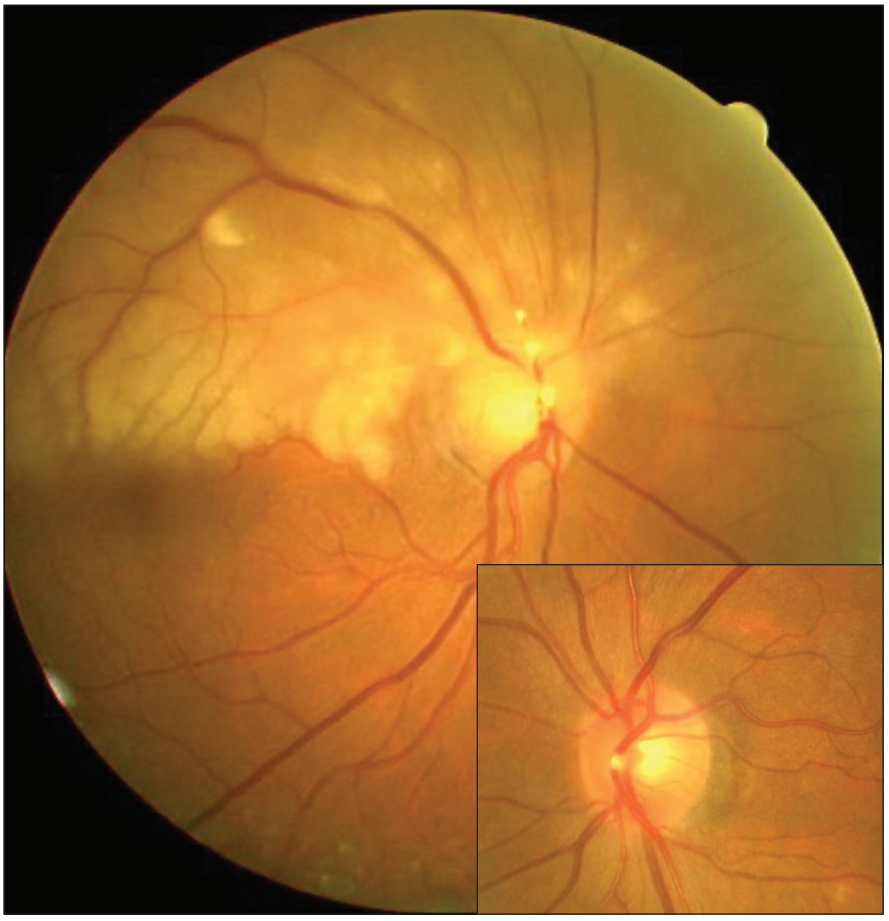

Figure 2. Fundus photo of a branch retinal artery occlusion with multiple plaques within the superior arteriole. A posterior pole embolus found at the bifurcation of the optic nerve vasculature in the same patient (Inset).

in up to 22% of patients with asymptomatic retinal emboli.

Of note, studies that defined significant carotid stenosis as greater than 70% have reported a prevalence of 9% to 22% in asymptomatic patients.[1] These findings suggest asymptomatic patients may still be at risk of serious systemic cardiovascular disease.

DIAGNOSTIC IMAGING AND ANCILLARY OCULAR TESTING

Carotid doppler ultrasound is a noninvasive test to evaluate the internal and external carotid arteries by measuring blood flow velocity and plaque formation. Further testing may also include transesophageal echocardiography to rule out a calcific plaque, computed tomographic angiography (CTA), and/or magnetic resonance angiography, which determines the degree of carotid stenosis. CTA can also help identify the presence of a plaque in other cervical or intracranial vessels.

There is debate about the usefulness of diagnostic carotid doppler ultrasound studies in asymptomatic patients with Hollenhorst plaques. A Hollenhorst plaque finding alone without symptoms may be a marker of a past embolic event and a poor predictor of future events.[11] Nevertheless, there is good evidence to advocate for the use of carotid doppler ultrasound in asymptomatic patients; a study by Bakri et al demonstrated that Hollenhorst plaques are a marker of significant carotid disease, regardless of symptoms.[12] Because acute cerebral infarctions frequently accompany findings of Hollenhorst plaques, diffuse-weighted MRI is also recommended to help identify the cause of the infarction.[13]

Data on the effectiveness of diagnostic testing in these patients varies widely in the literature not only with regard to the prevalence of carotid disease, but also in the way that the results are reported.[1] Specifically, there is a variance in how "significant" disease is defined; in studies that do not specify the degree of carotid stenosis, up to 95% of patients with retinal emboli are estimated to have some carotid disease.[1] Regardless of disease severity, sending out for a carotid workup is a safe and justified approach to managing these patients.

Visual field testing may be performed to assess for a defect consistent with the location of a retinal embolus if a blockage has occurred within the posterior pole. Even in asymptomatic patients, an associated defect found on a visual field test may point to a previous branch retinal arterial occlusion (Figure 2, Inset).

Fundus photography should be performed to document the type of embolus found and its position, monitor for future migration, record any retinal or optic nerve changes, and help identify the presence of new emboli at future visits.[2]

One may also consider performing carotid auscultation of the ipsilateral carotid artery with a stethoscope. Although a negative sign for a bruit does not rule out carotid stenosis, a positive sign can indicate a higher risk for this finding.[5] It should be made clear that carotid auscultation does not replace the need for carotid ultrasound, which should always be performed to confirm the diagnosis.

TREATMENT

Due to the lack of evidence in random controlled trials, there is no standard treatment approach for patients with Hollenhorst plaques. Treatment decisions are based on a variety of factors, including symptoms, stenosis severity, age, comorbidities, and smoking history. Medical intervention, such as antiplatelet therapy, has been shown to reduce the 5-year stroke rate by 50% in asymptomatic patients.[14] The use of cholesterol-lowering medications

has shown to lower the risk of cardiovascular and cerebrovascular events.[15] The North American Symptomatic Carotid Endarterectomy Trial showed surgical intervention was more effective than medical therapy in patients with at least 70% stenosis.[16] Carotid endarterectomy and carotid angioplasty were also found to be safe and effective in treating carotid artery stenosis.[16]

EDUCATION AND REFERRAL

Optometrists are often on the frontline in addressing significant carotid artery disease in patients with Hollenhorst plaques. Reviewing the medical history and assessing for ocular and systemic symptoms is crucial to making appropriate referrals for carotid doppler ultrasound, CTA, or magnetic resonance angiography. At the very least, findings of Hollenhorst plaques should be communicated to the patient's primary care or managing physician. Patients should also be educated on ocular and nonocular symptoms of ischemic attack and be advised to report to their local emergency department immediately if such symptoms occur.

UNDERSTAND THE RISKS

Further research is needed to make effective conclusions on treatment strategies for patients who are asymptomatic. Regardless of their symptoms (or lack of), it is important that we educate our patients on the serious links between Hollenhorst plaques and cerebrovascular and cardiac disease, and the need for referral and prompt testing. ■

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