Retinal Emboli from Vascular Disease: A Brief Review

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Abstract
Retinal emboli have various distinguishing presentations. It is important to be able to differentiate the characteristics of retinal emboli, such as their appearance and location, in order to determine the origin of the embolus. This determination is key to the management of the ocular and systemic health of the patient. In this paper we will review how the identification of each of these retinal emboli will aid in the management of these patients.

Introduction
While retinal emboli can originate from a variety of sources including talc, fat and sepsis, they most commonly stem from vascular disease. Patients with retinal emboli from vascular disease should undergo an embolic work-up and an evaluation for cardiovascular risk factors though the timing and choice of treatment depends on the patient’s presentation. Herein, we review the presentation and management of retinal emboli associated with vascular disease.

Clinical Presentation
Retinal emboli from vascular disease have a prevalence of 1.3% in patients 43-86 years of age. Patients with retinal emboli may be asymptomatic or symptomatic. Most retinal emboli (up to 85%) are asymptomatic and are usually found on routine dilated exams. Patients with symptomatic visible retinal emboli have a retinal artery occlusion (RAO) and present with sudden, painless loss of vision in one eye; the vision loss may be total, altitudinal or sectoral depending on the location of the embolus. A visible retinal embolus is present in 60-70% of RAOs.

A RAO can be classified based on the location of the occlusion in the retinal vasculature. There are two primary types of RAO: branch retinal artery occlusion (BRAO) and central retinal artery occlusion (CRAO). Relative to a CRAO, a BRAO is less likely to affect the patient’s central vision. A BRAO often occurs distal to the lamina cribrosa with associated retinal whitening of the affected tissue. Patients with symptomatic BRAOs usually have emboli in the temporal arteries; patients may be asymptomatic if the occlusion is small or occurs outside the posterior pole.

An embolic CRAO is caused by an embolus proximal to the lamina cribrosa with decreased perfusion to most of the retina; it will typically manifest as complete retinal whitening with a cherry red spot in the macula and vision loss to 20/400 or worse. A cilioretinal artery, which is present in 20-50% of the population, may preserve central vision in select patients with CRAO; however, the location and amount of retina supplied by the cilioretinal artery varies among patients.

Types of Emboli
There are three main types of retinal emboli associated with vascular disease: cholesterol, platelet-fibrin, and calcific (see Table 1, Image 1 and 2). They typically stem from either carotid artery disease or cardiac valvular disease. Retinal emboli are often transient, and the composition of the embolus rather than the location matters more when it comes to the migration and resolution on subsequent visits. Overall, 66-90% of retinal emboli migrate peripherally or disappear.

Diagnostic Testing
Both ophthalmic and systemic testing are important in patients with retinal emboli. Serial fundus photography can document the type of embolus and its position, monitor for embolus migration, record optic nerve and retinal changes, and verify the presence of new emboli at future visits. Formal visual field testing may show a defect consistent with the location of retinal embolus if a blockage has occurred within the posterior pole; even if a patient is asymptomatic, an associated defect on visual field may point to a previous subclinical RAO rather than an asymptomatic retinal embolus.
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Valvular heart disease is the most common source of calcific emboli. 6,8 Hence, the importance of a cardiac workup—including echocardiogram, electrocardiogram, and ambulatory cardiac rhythm monitoring—with cardiac consultation as needed. Neurological consultation is indicated if the patient has signs or symptoms of transient ischemic attack or stroke. 9,13

Patients with asymptomatic and symptomatic retinal emboli have a 18-33% and 21-55% prevalence, respectively, of significant ICA stenosis. 1,7,8,15

Carotid artery stenosis greater than 50% measured with carotid Doppler ultrasound, magnetic resonance angiography or computed tomography angiography of the neck is considered clinically significant and indicates the need for anti-platelet therapy to reduce the risk of embolic stroke. 6,9,13-14

Carotid endarterectomy (CEA) is recommended in symptomatic patients with 50-99% stenosis and in asymptomatic with 70-99% stenosis. 14,16-17

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With the assistance of the PCP, patients with retinal emboli from vascular disease should be assessed for modifiable vascular risk factors such as hypertension, diabetes mellitus, hyperlipidemia, and tobacco use and, if present, these conditions should be optimally treated.

**Management**

While there is no proven treatment for an embolic RAO, patients with RAO should be immediately referred to a stroke center (preferably a Primary Stroke Center18) or an emergency department for an embolic work-up. 6,8-13 Patients

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**Table I. Characteristics of retinal emboli from vascular disease**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Cholesterol</th>
<th>Platelet-Fibrin</th>
<th>Calcific</th>
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</thead>
<tbody>
<tr>
<td>Percentage of Retinal Emboli</td>
<td>40-80%</td>
<td>15-35%</td>
<td>8-12%</td>
</tr>
<tr>
<td>Appearance</td>
<td>Small, yellow, hyper-reflective circular lesion within arteries 6,8,11</td>
<td>Dull, gray-white lesion seen segmentally along arteries 6,11</td>
<td>Dull or chalky, yellow-white circular lesion within arteries 6,11</td>
</tr>
<tr>
<td>Common Retinal Locations</td>
<td>Distal to the optic nerve; within an artery of an equal caliber or at a bifurcation 6,11,13</td>
<td>Anywhere along retinal arteries typically involving a bifurcation 6,11</td>
<td>Proximal to the first bifurcation (often within optic nerve) 6,14,11</td>
</tr>
<tr>
<td>Most Common Source</td>
<td>Internal carotid artery 6,8,11,13</td>
<td>Internal carotid artery 6,8,11</td>
<td>Cardiac valvular disease 6,8</td>
</tr>
<tr>
<td>Other Sources</td>
<td>Cardiac valvular disease 6,8</td>
<td>Cardiac valvular disease 6,8</td>
<td>Internal carotid artery 6,8</td>
</tr>
<tr>
<td>Movement</td>
<td>Half of the time 6,8</td>
<td>Almost always 6,8</td>
<td>Rare 6,8</td>
</tr>
</tbody>
</table>

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**Figure 1.** Calcific retinal embolus within the superior arcade at the level of the optic nerve (Courtesy of Neuro-Ophthalmology Virtual Education Library [NOVEL] via North American Neuro-Ophthalmology Association [NANOS])

**Figure 2.** Cholesterol retinal embolus within a peripheral retinal artery (Courtesy of Neuro-Ophthalmology Virtual Education Library [NOVEL] via North American Neuro-Ophthalmology Association [NANOS])

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with acute RAOs have up to a 25% risk of stroke within 4 weeks of onset.12,19,20 The embolic work-up for a patient with asymptomatic retinal embolus can be performed less urgently on an outpatient basis.12,13 While asymptomatic emboli do not threaten vision, the incidence of stroke is significantly higher in patients with compared to those without emboli.11,13

Patients with retinal emboli from vascular disease are at higher risk of mortality from all causes with an increased incidence of stroke-related death compared to patients without a retinal embolus over 5-10 years.9,6,17,12 Myocardial infarct (MI) is still the leading cause of death in patients with a retinal embolus, however, the rate of death from MI is the same when compared to patients without retinal emboli present.8,9,12 These trends underscore the importance of coordinating care with the patient’s PCP to ensure that patients with retinal emboli receive appropriate medical and/or surgical therapy as indicated.

**Conclusion**

Patients with acute, symptomatic retinal emboli from vascular disease are at high risk for a stroke and warrant an immediate referral to the nearest emergency department for further management.11,12,13 All patients with retinal emboli from vascular disease require appropriate diagnostic testing to identify and manage vascular risk factors, including potential embolic sources.

**References**