Answer to Ophthaproblem *continued from page 167*

4. Central retinal artery occlusion

Central retinal artery occlusion (CRAO) is a visually debilitating event with a relatively poor visual prognosis.[1] Central retinal artery occlusion results from an abrupt cessation of blood flow through the central retinal artery, causing ischemia to the inner retina. It typically affects individuals older than 50 years of age who have cardiovascular risk factors. Hypertension and diabetes mellitus are frequent comorbidities in CRAO patients, and a history of amaurosis fugax can often be elicited. There are many etiologic factors that might underlie CRAO. Central retinal artery occlusion commonly occurs secondary to a cardioembolic event within a setting of atrial fibrillation or valvular disease or secondary to local carotid artery disease. A cardioembolic embolus is the prevailing cause of CRAO in patients younger than 40 years of age. Giant cell arteritis should be excluded in elderly patients with CRAO.[2]

Typical symptoms of CRAO include sudden, painless, severe, and persisting monocular vision loss. Most affected patients can see hand motions or count fingers at best. A relative afferent pupillary defect is present. Diagnosis of CRAO is based on the fundoscopic examination, which reveals, in the acute stages, an edematous, ground-glass retina with the classic "cherry-red spot." This progresses to diffuse retinal pallor over time. Retinal emboli can be seen in the fundus of the eyeball in 20% to 25% of patients. Attenuation of the retinal vasculature, including segmental irregularities of the retinal arterioles, can be seen and is referred to as "box-carring." Most important, fluorescein angiography shows a noticeably delayed rate of filling of the retinal arterial tree.

Systemic investigations are important for patients with CRAO. At the time of presentation, blood pressure assessment, palpation of the temporal arteries, and auscultation of the heart and carotid arteries are imperative. Patients should also be scheduled for carotid doppler ultrasonography and 2-dimensional echocardiography. Additional blood tests are important in certain situations. A fasting blood glucose test, lipid profile, and a hypercoaguability workup might also be indicated, particularly if patients do not have cardiovascular risk factors or if they are younger than 40 years of age.[2] If giant cell arteritis is suspected upon history or examination, a complete blood count, erythrocyte sedimentation rate, and C-reactive protein level should be obtained. If clinical suspicion is high, these patients should be prescribed high-dose systemic steroids (eg, 1 mg/kg prednisone) and require a subsequent temporal artery biopsy.

Management

The visual outcome after CRAO is poor, with only 6.5% of patients achieving 20/20 or better, 16% achieving 20/40 or better, and 29% achieving 20/200 or better.[3] Outcomes depend on the type and etiology of the CRAO. Further, the presence of a cilioretinal artery, which provides an alternate circulation to the macula, can protect central visual acuity from devastating loss.[3]

A number of treatment modalities for CRAO have been proposed and practised but have been met with limited success. Conventional treatment options include anterior chamber paracentesis, oral acetazolamide, acetylsalicylic acid, ocular massage, carbogen inhalation (95% oxygen, 5% carbon dioxide), and topical intraocular pressure–lowering medications. However, a recent Cochrane meta-analysis concluded that these treatment regimens do not improve the natural course of the disease.[4] Because of the dismal prognosis and low rate of adverse affects from treatment, most physicians prescribe varying combinations of these therapies. As such, managing CRAO remains a therapeutic dilemma.[5]

More recently, the use of tissue plasminogen activator enzymes delivered super-selectively to the ophthalmic artery through an endovascular catheter has shown some promise in improving visual outcomes after CRAO. A recent systematic review of the drug provides encouraging evidence for such treatment; however, the first randomized controlled trial investigating this novel treatment modality is still under way.[1]

Recommendations

Patients with acute CRAO should be urgently referred to an ophthalmologist for prompt assessment and management. These patients require systemic investigations to identify possible underlying etiologic causes, including carotid artery disease, valvular heart disease, and giant cell arteritis.

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Competing interests
None declared

References

1. Noble J, Weizblit N, Baerlocher MO, Eng KT. Intra-arterial thrombolysis for central retinal artery occlusion: a systematic review. *Br J Ophthalmol* 2008;92(5):588-93.
2. Wray SH. The management of acute visual failure. *J Neurol Neurosurg Psychiatry* 1993;56(3):234-40.
3. Hayreh SS, Zimmerman MB. Central retinal artery occlusion: visual outcome. *Am J Ophthalmol* 2005;140(3):376-91.
4. Mueller AJ, Neubauer AS, Schaller U, Kampik A; European Assessment Group for Lysis in the Eye. Evaluation of minimally invasive therapies and rationale for a prospective randomized trial to evaluate selective intra-arterial lysis for clinically complete central retinal artery occlusion. *Arch Ophthalmol* 2003;121(10):1377-81.
5. Hayreh SS. Intra-arterial thrombolysis for central retinal artery occlusion. *Br J Ophthalmol* 2008;92(5):585-7.