Treatment of central retinal artery occlusions

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To the Editor:

As a practicing retinal specialist for the last 28 years, I read the guidelines for the treatment of central retinal artery occlusion (CRAO)(1) with interest. There are two major issues of concern: diagnosis and cause.

• Making the correct ophthalmic diagnosis

Acute visual loss is when the known duration of visual loss is short, to be distinguished from when the patient accidentally discovers a longer-standing visual loss. The elderly patient (the group most affected by CRAO), is frequently unaware of the loss of vision in one eye, until they happen to close the other eye (i.e., a foreign body in the “good” eye). They date the loss of vision to the time they closed the eye with better vision. The patient is then seen as an emergency with “acute” visual loss, but retinal examination reveals the visual loss to be long-standing.

The “acuteness” or “chronicity” of visual loss may be difficult to assess without ophthalmic examination experience. It may also be difficult to perform a retinal examination through a miotic, undilated pupil in an elderly patient with cataracts in the emergency room setting.

Many conditions associated with visual loss also exhibit cotton-wool spots (soft exudates) which may be mistaken for the retinal edema seen in arteriolar occlusion. The differential diagnosis includes: pre-proliferative diabetic retinopathy with macular edema; epimacular membrane and vitreomacular traction (the whitish posterior pole sheen may simulate retinal edema); shallow retinal detachment; retinoschisis (a splitting of the retina) extending into the posterior pole; commotio retinae (caused by an ocular contusion); Purtscher’s retinopathy (from a crush injury), malignant hypertension, collagen diseases, hematologic conditions, Hodgkin’s disease, infective conditions, etc.

These diagnoses are sometimes difficult to make, in the best of circumstances, more so in a busy emergency room.

The most common cause of visual loss in younger patients is diabetic retinopathy, and in the elderly it is age-related macular degeneration. Vascular occlusion, whether branch retinal vein or artery occlusion, or central retinal vein or artery occlusion, is far less common. CRAO is an infrequent cause of visual loss, occurring in only 0.85 per 100,000 persons per year. (2) Not surprisingly, the EAGLE Study (3), a 16-center randomized controlled prospective trial comparing two treatments for CRAO, has enrolled only 47 patients with CRAO in three years.

• Determining the cause of the CRAO.

Two causes of CRAO, giant cell arteritis and emboli from carotid artery stenosis, may result in further morbidity and even death. While treating visual loss in one eye is important, it should not be at the risk of severe visual loss in the other eye (as in giant cell arteritis) or cerebral stroke (as in emboli from significant carotid stenosis). The risk versus benefits of the treatment of one eye must be put in the perspective of the patient’s life.

Giant cell arteritis

This most common primary vasculitis of adults in the Western world is an immunopathogenic process that results in luminal occlusion as well as a generalized inflammatory response. Three common clinical subtypes have been defined: a systemic inflammatory syndrome, which may include myalgias, weight loss, night sweats and fever of unknown origin; a cranial arteritis including visual loss; and a large-vessel vasculitis, which may include aortic rupture, aortic dissection and death (4,5,6).
Retinal embolism and risk of stroke

Pfaffenbach et al. (7) reported a case-series of 208 patients with retinal emboli. Fifteen percent of this group died within one year, and 54% died within seven years. Cerebral strokes or transient ischemic attacks were present in 63% before or at the time of baseline examination. Bruno et al. (8) studied the risk of stroke in 70 patients with retinal emboli as compared to a 70-person control group. With a 3.4-year follow-up, there was a tenfold increase in the annual stroke rate (8.5% versus 0.8% per year in cases versus controls, p = 0.002). (This may actually represent an underestimation of the stroke risk, as patients with retinal emboli and other additional cardiovascular risk factors, i.e., hypertension and cigarette smoking, may be more likely to die.)

The patient presenting to the emergency room with truly acute visual loss may be placed on supplemental oxygen prior to ophthalmology consultation. While a negative result (no visual improvement) is not diagnostic, a positive result (visual improvement) may serve as a provocative test that HBO2 may be beneficial. Following the examination, the ophthalmologist will refer the patient for HBO2, if appropriate. While the visual results may not be as good as if the patient had been immediately placed in a hyperbaric chamber, the potential benefit does not justify the risk.

The UHMS has recently contacted other specialty societies, including the American Academy of Ophthalmology and the American Society of Retina Specialists, to inform them of the CRAO indication for HBO2. This opportunity — specialists of one society treating the patients of another — must be tempered with the natural human tendency and appropriateness of each group to be understandably “protective” of their individual expertise. Treating the wrong patient with the wrong therapy carries risk that neither group would accept, the legal community would frown on and the insurance companies would not pay for.

Recent evidence (9) seems to indicate that there may still be visual improvement if HBO2 treatment is begun after what should be a minimal delay to establish the correct course of action. Once the patient has been referred for HBO2, that patient is under the care of the hyperbaric physician. Whether higher, lower or a variation in atmospheric pressure results in the maximal visual improvement is unknown at this time. More is not necessarily better — e.g., the platelet effect of low-dose aspirin versus higher-dose aspirin. The “correct” dosage is the most “beneficial” pressure and will be determined by further study. The hyperbaric physician, not the ophthalmologist, will make that decision.

Physicians, regardless of their specialty, must work in a complementary fashion according to their training and experience to provide the patient with the best possible result.
References


Response to Treatment of central retinal artery occlusions

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To the editor:

As a practicing academic emergency medicine physician and hyperbaricist, I (along with my co-author, Dr. Frank Butler, who has contributed to this response) am very pleased with the interest and passion exhibited from the ophthalmology community. We feel our biggest hurdle here is getting the word out there – time is vision, and hyperbaric oxygen therapy (HBO2) can save vision if administered before irreversible ischemia has occurred.

While we understand the critical importance of making the correct diagnosis and the treatment of concomitant systemic diseases that may contribute to the development of central retinal artery occlusion (CRAO), we do not feel that treatment should be delayed to accomplish this. As little as one or two hours of delay may make the difference in whether or not the patient’s vision is restored (1,3,9,10). The major differential diagnosis of acute painless vision loss includes: CRAO, central retinal vein occlusion (CRVO), vitreous hemorrhage, retinal detachment, ischemic optic neuropathies, amaurosis fugax, and posterior circulation cerebral vascular accidents (2). None of these is as crucially time-sensitive as is CRAO. Also, none of these would be harmed by HBO2 initiated prior to an ophthalmologist’s exam, and there are case reports actually showing improvement in CRVO and ischemic optic neuropathies treated with HBOT (3,4,5,6,7,8,11,12).

The emergency physician is capable of differentiating CRAO from retinal detachment or vitreous hemorrhage in the majority of cases, often based on the quality of visual complaints, coupled with fundoscopic findings. Additionally, the information provided in the discussion of HBO2 for acute painless loss of vision in the 2008 paper “HBOT and the Eye,” provides some suggested exclusion criteria that include: age under 40, duration of visual loss greater than 24 hours, a history or recent eye surgery or eye trauma, and a history of floaters of photopsias (3). These exclusion criteria will help to exclude many patients whose loss of vision is likely due to eye disorders that are not known to respond to HBO2. These recommendations also call for evaluation of the patient by an ophthalmologist as soon as feasible, either before or immediately after the HBO2.

We plan to expand the discussion of systemic disease work-up for patients with CRAO in the next update of the UHMS Hyperbaric Oxygen Committee Report. We had assumed this would be done as part of basic practice in either emergency medicine or ophthalmology prior to the consultation of a hyperbaricist, but we have had a couple of requests for clarification, so we will elucidate this in the next edition. We never intended to suggest that work-up for concomitant disease was unnecessary. Thank you to Dr. Weiss and others for helping us improve communication and understanding among our specialties so that we may save vision whenever possible.
References

1. Murphy-Lavoie H, Butler FK, and Hagan CE. Hyperbaric oxygen therapy in the management of central retinal artery occlusion.” In, Gesell L, ed; Hyperbaric Oxygen Therapy Committee Report 2009; Durham; Undersea and Hyperbaric Medical Society; 2009.


