CENTRAL RETINAL ARTERY OCCLUSION

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The central retinal artery branches off the ophthalmic artery which in turn branches off the internal carotid artery. The central retinal artery is vital because it supplies blood to the inner two-thirds of the retina. If the central retinal artery becomes occluded, there will be a sudden painless loss of vision in that eye.

Central retinal artery occlusion (CRAO) is generally due to an embolism including platelet-fibrin, cholesterol, or calcium plaque. The emboli break off vessel walls or cardiac valves and travel downstream until the vessel becomes too small, and the emboli or plaque simply gets stuck. The entire retina, except the fovea (center of the macula), will become pale and swollen. The hallmark of CRAO is the cherry red spot. The cherry red spot is termed so because the central fovea still appears reddish. This is present because the fovea gets its blood supply from the choriocapillaris, not the central retinal artery. In some cases, approximately 20% of the population, there is a branch of the ciliary circulation called the cilio-retinal artery. The cilio-retinal artery supplies the retina between the fovea and the optic nerve, which includes the papillomacular bundle. When this artery is present, central vision may be preserved due to that small portion of viable macula. This occurrence is known as cilio-retinal sparing.

Central retinal artery occlusion is seen in men more than women. Although the primary age group for CRAO is usually early to mid sixties, it does occur in younger individuals as well. Race and ethnicity have no bearing on this condition. When CRAO is seen in an elderly patient,
it is primarily due to diabetes, hypertension, cholesterol or calcium plaques, tobacco abuse, or giant cell arteritis. When it is found in younger patients, inflammatory disease such as lupus and syphilis are generally the cause.

Signs and symptoms of central retinal artery occlusion include sudden painless unilateral vision loss. The pupil will respond poorly to direct light but will constrict briskly when the other eye is illuminated. This is a sign of relative afferent pupillary defect. A complete visual field defect will also suggest CRAO. Typically, the arteries are attenuated and may even appear bloodless. An embolic obstruction may also be visible. The most common variant is the refractile yellow cholesterol embolus (Hollenhorst Plaque), which is thought to most commonly originate from atherosclerotic disease in the carotid arteries. However, the central retinal artery itself may slowly develop an atherosclerotic plaque. Calcium deposits are less commonly seen, but tend to be larger. They are dull white in appearance and tend to break off of calcific cardiac valves.

Rubeosis iridis and neovascular glaucoma are complications that may follow central retinal artery occlusion. When rubeosis is seen in association with CRAO the possibility of simultaneous ipsilateral carotid artery obstruction should also be considered. Neovascularization of the optic disc and retina may also occur, although the incidence appears to be lower than neovascularization of the iris.

Immediate treatment is indicated if occlusion has occurred within 24 hours of patient presentation. It is important to reduce the intraocular pressure (IOP) quickly thereby improving perfusion pressure. This goal is accomplished by using the same medications as those used to treat glaucoma. This helps to increase oxygen delivery to the hypoxic retina. Paracentesis of the
anterior chamber rapidly lowers IOP and may dislodge an embolus and allow it to move downstream. Yag laser may also be used to remove the offending embolus. Treatments for CRAO rarely improve the visual acuity. The remaining vision ranges from count fingers to light perception in 90% of cases. When an embolus is present, prognosis is even poorer. Some patients are unable to even perceive light.

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