Central and branch retinal artery occlusion

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INTRODUCTION

Central retinal artery occlusion (CRAO) and branch retinal artery occlusion (BRAO) present with acute, painless loss of monocular vision. These disorders are considered a form of stroke, with a similar clinical approach and management; the clinician attempts to treat the acute event, find the source of the vascular occlusion, and prevent further vascular events from occurring [1].

Issues related to central and branch retinal artery occlusion will be reviewed here. Other ocular ischemic syndromes are discussed separately. (See "Amaurosis fugax (transient monocular or binocular visual loss)".)

VASCULAR ANATOMY

Blood flowing to the retina travels through blood vessels that are subject to varying environments and external pressures [2]. The central retinal artery arises from the ophthalmic artery, itself a branch of the internal carotid artery. The ophthalmic artery departs from the intracranial portion of the carotid artery at a sharp angle [3], after which it travels intracranially for a short distance before entering the orbit, usually with the optic nerve. Once entering the orbit, the central retinal artery leaves the ophthalmic artery to enter the cerebral spinal fluid space and then travel within the optic nerve, after which it enters the eye, where it is subjected to intraocular pressure.

The central retinal artery supplies the inner retina and the surface of the optic nerve. In approximately 15 percent of individuals, it is assisted by a branch of the ciliary circulation, the

cilioretinal artery, which may supply a portion of the retina, including the macula. This allows for preservation of vision in some patients with CRAO.

The physiology of central retinal artery function is poorly understood, but it is primarily autoregulated [4]. This means that under a range of systemic mean arterial pressures, ocular blood flow remains constant. (See "Moderate to severe hypertensive retinopathy and hypertensive encephalopathy in adults", section on 'Mechanisms of vascular injury'.)

EPIDEMIOLOGY

CRAO is a rare event with an incidence of approximately 1 to 10 in 100,000 [5,6]. Symptomatic BRAO is even less common.

Demographic characteristics of patients with CRAO and BRAO are consistent with those seen for other vascular disorders. The mean age of patients is between 60 and 65 years [7-9]. More than 90 percent of patients with CRAO and BRAO are over the age of 40 years, although individuals as young as 12 years have been affected [7,9-11]. Men are somewhat more commonly affected than women. In some but not all series, hypertension, smoking, and diabetes are more prevalent among patients with retinal artery occlusion compared with controls [9,12,13]. Patients presenting with CRAO often have a previously undiagnosed vascular risk factor (eg, hyperlipidemia, hypertension) [14].

ETIOLOGY

Central and branch retinal artery occlusions share a similar list of potential etiologies. Case series that attempt to define a distribution of etiologies are small and/or include a heterogeneous case mix of amaurosis and asymptomatic retinal emboli in addition to CRAO and BRAO. In addition, diagnostic evaluations are not standardized and vary between studies.

The distribution of etiologies is also influenced by the patient's age. Carotid artery atherosclerosis is the most common etiology for retinal artery occlusion overall but is relatively unusual in patients under 40, in whom cardiogenic embolism is the most likely etiology [15,16]. In patients over 70 years of age, giant cell arteritis (GCA) is more likely than in younger patients. Ethnicity may also be relevant; as an example, carotid artery occlusive disease is most prevalent among Caucasians [17].

Determining the underlying etiology is important to preventing recurrent CRAO and BRAO and other vascular complications. (See 'Diagnosis of etiology' below.)

Carotid artery atherosclerosis — Atherosclerotic disease of the ipsilateral carotid artery is the most common cause of retinal artery occlusion. The reported prevalence of significant (>70 percent) carotid artery stenosis among patients with CRAO or BRAO is 10 to 40 percent in most case series [13,17-28].

The diagnosis of high-grade carotid disease in the setting of retinal artery occlusion is important because of the risk for future stroke and other vascular events. Compared with cerebral ischemia, retinal ischemia has been reported to carry a lower risk for stroke in the setting of high-grade carotid disease (>70 percent stenosis) [29]. However, these case series generally also include transient monocular visual loss and/or asymptomatic retinal emboli, which might be expected to have a more benign prognosis than CRAO or BRAO. In one report, three of seven patients with CRAO and high-grade carotid disease had a subsequent stroke over 4.5 years of follow-up [23]. This suggests that CRAO may be similar to cerebral infarction in predicting future stroke risk in the setting of high-grade carotid stenosis.

Carotid endarterectomy is likely to significantly reduce the risk of future stroke in patients with retinal artery occlusion and high-grade carotid stenosis, although this subgroup has not been specifically studied [29]. (See "Management of symptomatic carotid atherosclerotic disease", section on 'Factors influencing benefit and risk'.)

Cardiogenic embolism — A cardiogenic embolic source (table 1) is second to carotid disease in causing retinal artery occlusion. In some series, this is found in as many as 48 percent of patients (range 2 to 48) [13,18,26]. The prevalence of an underlying cardiac source is influenced by several factors:

- Cardiogenic embolism is more likely to underlie retinal artery occlusion in younger patients (<40 years), in whom it may cause up to half of the cases with an identified origin [30,31]
- The likelihood of a cardiac source for retinal artery occlusion increases significantly if there is a suggestive medical history, such as atrial fibrillation [32-34], infectious endocarditis, rheumatic heart disease, cardiac valvular disease, myocardial infarction, congenital heart disease, intravenous drug use, cardiac tumor, or a cardiac murmur [27,35].
- Visualization of retinal emboli on funduscopic examination is not more frequent among patients with a cardiogenic embolism compared with those with an alternative or unknown etiology [19,36]

Although often considered unlikely, a cardiogenic embolic source is important to identify because chronic anticoagulation may be recommended to prevent more serious events. A

detailed review of cardiogenic sources of cerebrovascular emboli is presented elsewhere. (See "Stroke: Etiology, classification, and epidemiology", section on 'Embolism' and "Echocardiography in detection of cardiac and aortic sources of systemic embolism".)

Small artery disease — In addition to distal emboli, local atheroma within the central retinal artery at the lamina cribrosa may cause arterial occlusion [37]. Small artery disease is often the presumptive etiology in older patients with diabetes and/or hypertension and no other demonstrated cause for retinal artery occlusion. (See "Lacunar infarcts".)

Other vascular disease — In addition to atherosclerotic carotid artery disease, other vascular disease involving the carotid, ophthalmic, or retinal arteries can lead to symptomatic retinal artery occlusion. These include:

- Carotid artery dissection [28,38,39]
- Fibromuscular dysplasia [11]
- Radiation injury of the carotid or retinal arteries [25,40,41]
- Moyamoya disease [42]
- Fabry disease [43]
- Intra-arterial thrombosis [28]

Retinal artery occlusion has also been attributed to vasospasm and migraine, but these are diagnoses of exclusion [25,44].

Giant cell arteritis — Approximately 2 percent of older adult patients with CRAO have underlying GCA. Vision loss in GCA is usually due to ischemic optic neuropathy; however, 10 percent of patients who lose vision from GCA do so on the basis of CRAO [18,45,46]. While an unusual cause of CRAO, it is important to diagnose GCA because of its implications for prognosis and treatment. GCA should be strongly considered as a potential cause of CRAO in any patient over the age of 50 years who does not have visible retinal emboli. (See "Clinical manifestations of giant cell arteritis".)

Other inflammatory disease — A variety of autoimmune diseases can produce a vasculitis that can lead to retinal artery occlusion. The presentation of vision loss is similar to other etiologies; however, retinal emboli are not seen on funduscopic examination in these patients.

- Susac syndrome – Susac syndrome is a rare vasculitic disorder characterized by vision loss due to BRAO and accompanied by sensorineural hearing loss and a subacute encephalopathy. (See "Primary angiitis of the central nervous system in adults", section on 'Alternative diagnoses'.)

- Other – Systemic lupus erythematosus, polyarteritis nodosa, sarcoidosis, eosinophilic granulomatosis with polyangiitis (Churg-Strauss), and granulomatosis with polyangiitis are other disorders that have been associated with retinal artery occlusion [25,47-52]. In such patients, retinal artery occlusion typically occurs in the setting of an established diagnosis of the underlying autoimmune disease.

Hematologic disease — A number of hematologic conditions have been associated with retinal artery occlusion:

- Sickle cell disease can cause arterial occlusion or stenosis [53]. (See "Acute stroke (ischemic and hemorrhagic) in children and adults with sickle cell disease".)

- Hypercoagulable states associated with retinal artery occlusion include antiphospholipid syndrome [54], factor V Leiden mutation [55-57], protein S deficiency [58,59], and protein C deficiency [60].

- Leukemia and lymphoma have been associated with retinal artery occlusion due to either hypercoagulopathy or a hyperviscosity syndrome [25,61,62].

Rare causes

- Infection can cause a secondary vasculitis of the ophthalmic and/or retinal artery. Pathogens include fungi (particularly mucormycosis), viruses (varicella), cat scratch disease, and toxoplasmosis [63-66].

- CRAO may complicate ocular surgery as well as ocular or retrobulbar injections of steroids or other drugs, presumably by direct injury to the central retinal artery [67]. CRAO may also occur after endoscopic sinus surgery, usually as a result of orbital compartment syndrome [68].

- Cerebral angiography, carotid endarterectomy, and coiling of intracranial aneurysms can lead to thromboembolism and retinal artery occlusion [69-75].

- Pressure placed on the eye by a headrest was believed to cause CRAO in two cases after back surgery [76,77].

- One patient developed CRAO following vitrectomy with intraocular gas tamponade and subsequent high-altitude travel [78].

- Other rare causes of CRAO include inadvertent injection of talc or other embolic material [69,79], fat embolism [80], and amniotic fluid embolism [81].

- Case reports have also described CRAO following intravitreal injections of anti-VEGF agents (ranibizumab and bevacizumab) for age-related macular degeneration [82,83].
- CRAO after chiropractic manipulation of the neck has also been reported [84].
- At least one case report identified BRAO secondary to head and neck radiation therapy [85].

ACUTE CLINICAL FEATURES

Central retinal artery occlusion

- **Presenting symptoms** – Most patients with CRAO develop acute and profound loss of vision in one eye that is usually painless. Occasionally, CRAO is preceded by transient monocular blindness or there is a stuttering or fluctuating course [86]. Rarely, the initial event is heralded by flashing lights. CRAO almost never occurs in both eyes simultaneously, but it may occur sequentially [18].
- **Vision loss** – The vision loss is severe, usually leaving no more than a small temporal island of vision. Most affected patients can see only hand motions and rarely can count fingers. However, visual acuity may be normal in the approximately 15 percent of patients who have a cilioretinal artery feeding the macular region. These patients have a small central area of visual field remaining. In one study of 107 CRAOs, 28 (26 percent) showed some degree of macular sparing due to patent cilioretinal arteries [87]. However, only 17 had sparing of the fovea and therefore visual acuity.
- **Pupillary function** – A complete or relative afferent pupillary defect occurs regardless of the presence of macular sparing. (See "Approach to the patient with anisocoria (unequal pupil size)", section on 'Examination of the pupil'.)
- **Funduscopic examination** – On funduscopic examination, ischemic retinal whitening is usually seen immediately after an occlusion of the central retinal artery. A "cherry red spot" appears in the macula, where the retina is thinner and the retinal pigment epithelium and choroidal vasculature can be seen more easily as the ischemic retina becomes less translucent (picture 1). In patients who have macular sparing, funduscopic examination shows the ischemic retinal whitening clearly surrounding the area of preserved cilioretinal circulation (picture 2).

  Retinal emboli can be seen in the fundus of 11 to 40 percent of patients with CRAO [19,24,88,89]. The embolic material may appear as iridescent, shiny cholesterol plaques

(picture 3); gray/white platelet plugs (picture 4); or bright, white calcium fragments. Visible retinal emboli are not more frequent in patients with an identified embolic source in the heart or ipsilateral carotid artery compared with those with another or unidentified etiology [19].

In the acute phase, the blood column in both the arteries and the veins can become segmented with separation of serum from Rouleau stacking of the red corpuscles leading to a "box-carring" appearance of the blood column.

Branch retinal artery occlusion — Patients with BRAO typically complain of monocular visual loss, which may be restricted to just part of the visual field. At presentation, less than half of patients with BRAO have impaired visual acuity [7]. The acute funduscopic examination in BRAO demonstrates a sectoral pattern of retinal opacification. Retinal emboli are more frequently seen than with CRAO, being present in up to two-thirds of patients [24,30,89,90].

Associated symptoms — Associated clinical findings, when present, may suggest a cause. Symptoms and signs of vascular and hematologic disease should be specifically solicited. As examples, contralateral sensory or motor symptoms suggest carotid artery disease, headache may suggest giant cell arteritis (GCA) in an older person, and neck pain with recent cervical trauma may indicate a carotid artery dissection.

Optical coherence tomography — Optical coherence tomography (OCT) is not routinely available or performed in this setting. Case series report that OCT shows thickening of ischemic areas of the inner retina soon after central or branch retinal artery occlusion, with subsequent thinning that is apparent after a few weeks [91-93]. The outer retinal layers are spared. In one case series, a prominent middle limiting membrane sign (seen as a hyperreflective line in the inner layer of the outer plexiform layer on OCT) correlated with early (within the first month) ischemic damage [94].

DIAGNOSIS

A diagnosis of retinal artery occlusion rarely requires confirmatory testing, although fluorescein angiography can be helpful in unusual cases, for example when the acute findings of ischemic retinal whitening or reduced blood flow are not clearly present. In the acute phase, fluorescein angiography shows slowed or absent filling of the central retinal artery with normal filling of the choroid. If the choroid is also affected, giant cell arteritis (GCA) should be considered, especially in an older adult patient [95].

The erythrocyte sedimentation rate (ESR) and serum C-reactive protein (CRP) should be measured in all individuals over age 50 with CRAO and no visible retinal emboli to exclude GCA. Although GCA is relatively rare in this setting, acute treatment with corticosteroids improves visual outcomes (see 'Giant cell arteritis' above). If the ESR or serum CRP is elevated or if the clinical setting is very suggestive, the patient should be evaluated for GCA and possible treatment with high-dose corticosteroids. (See "Diagnosis of giant cell arteritis" and "Treatment of giant cell arteritis".)

Other diagnostic testing in patients with retinal artery occlusion is focused on determining the underlying etiology. These are generally not required for acute management decisions. (See 'Diagnosis of etiology' below.)

ACUTE TREATMENT

Central retinal artery occlusion — CRAO is an ocular emergency. Before any other investigation, attempts to recover vision in the affected eye should be made or at least considered.

Given the poor prognosis of visual recovery after CRAO, some attempt at treatment is usually made if the patient presents early, within the first 24 hours. Unfortunately, there are no clinical trials to determine if any treatment is effective. Some patients do recover spontaneously, and apparently successful maneuvers to recover vision may reflect this phenomenon [96].

A 2013 review of treatment options for CRAO indicated that in order to be effective, treatment should be given within six hours of ischemia onset, regardless of the method utilized [97]. Within this time frame, earlier is likely better. Animal studies suggest that restoration of blood flow within 90 to 100 minutes leads to no retinal injury, while occlusions persisting for longer than 240 minutes produce massive irreversible damage [97-100].

Revascularization

- **Intravenous thrombolysis** – We agree with the American Heart Association statement that intravenous tissue plasminogen activator (tPA) "may be a reasonable treatment for patients with CRAO after a discussion of the benefits and risks with the patient or surrogate" [1]. The basis for this statement includes observational evidence that suggests a substantial benefit in terms of visual recovery; however, physicians may choose not to use thrombolysis in this setting, preferring to await the results of clinical trials in progress, given the known risks of this therapy.

Such therapy should be administered within 4.5 hours of symptom onset (or last known well) and no contraindications should be excluded. These exclusion criteria are those that increase the risk of intracranial or hemorrhagic stroke and are similar to those for treatment of acute ischemic stroke (table 2).

Alteplase is delivered via intravenous infusion (0.9 mg/kg with 10 percent given over one minute and the remainder over one hour).

In a meta-analysis of observational studies, 67 patients with acute CRAO treated with alteplase within 4.5 hours were more likely to recover visual acuity compared with those not treated with lytic therapy (37.3 versus 17.7 percent) [101]. Three patients experienced an intracranial hemorrhage, one of which was asymptomatic. Similarly, a previous meta-analysis of observational studies found a higher rate of improved vision among patients treated with any form of intravenous thrombolytic therapy within 4.5 hours compared with untreated patients (50 versus 17.7 percent) [102]. Clinical trials, which are ongoing, are needed to confirm these results [103].

Challenges limiting the administration of tPA in this setting include the timely presentation of the patient and the availability of urgent ophthalmology consult and/or lack of confidence in the diagnosis of CRAO. In one United States database study, tPA was administered to 5.8 percent of patients admitted with CRAO compared with 8 percent of those with acute ischemic stroke [9].

- **Intra-arterial thrombolysis** – Intra-arterial thrombolysis may have a theoretical advantage over intravenous treatment [101,104-107]. More clinical trial results are awaited to determine whether intra-arterial thrombolysis should be a recommended treatment for CRAO [108-110].

  A meta-analysis of retrospective, nonrandomized studies published in 2000 reported a potential benefit for this treatment when compared with the natural history of CRAO, with 15 percent of patients achieving normal vision and 27 percent achieving 20/40 visual acuity or better [107]. While serious complications are a concern with this treatment, only 6 of the 100 cases reviewed had a complication, none with permanent morbidity. Subsequent reports have largely echoed this experience [111-117]. However, a randomized controlled trial compared intra-arterial thrombolysis with recombinant tPA to conservative therapy in 84 patients with CRAO within 20 hours (mean 12 hours) of symptom onset and found no difference in visual outcomes between the two groups [118]. Intracerebral hemorrhage occurred in two of the treated patients. The long delay to treatment in this study may have contributed to the findings. Finally, a subsequently published retrospective study found

intra-arterial thrombolysis to be more effective than standard treatment in patients with incomplete CRAO; outcomes were similar for patients with subtotal or total CRAO [119].

- **Surgical techniques** – Surgical revascularization techniques are under investigation [120]. In two case reports, patients with CRAO underwent central retinal arteriotomy with a stylet during vitreous surgery or with laser therapy, disrupting the obstruction and producing a good visual outcome [121-123].

Other therapies — In individuals who are not candidates for thrombolysis and present with a CRAO less than 24 hours old, one can perform anterior chamber paracentesis. Anterior chamber paracentesis, using either a Graefe knife or a syringe, causes a sudden drop in intraocular pressure [124,125]. This may dislodge an embolus and/or increase ocular perfusion.

Other treatment options for patients not eligible either for revascularization or anterior chamber paracentesis include:

- **Ocular massage**, whereby intermittent pressure is applied to the eye. Aqueous outflow is increased with pressure, and retinal perfusion should improve once the digital pressure is relieved [126]. These maneuvers may also dislodge the embolism.

- **Reduction of intraocular pressure** may also be accomplished by intravenous acetazolamide, intravenous mannitol, or topical pressure-lowering drops.

- **Vasodilator medications** used to improve ocular blood flow have included pentoxifylline, nitroglycerin, and isosorbide dinitrate [5,43,44,127,128].

- **Hyperbaric oxygen therapy**, to maintain oxygenation of the retina pending reperfusion, has been used to preserve vision with mixed results in small series of patients [129-132]. Several case series suggest that hyperbaric oxygen may improve visual outcome in CRAO [133-135]. However, its use is limited because it is labor intensive to deploy and has limited availability.

  In normal physiology, >50 percent of the retinal oxygen supply is derived from passive diffusion from the choroidal circulation [30], whereas with hyperbaric oxygenation, it is as high as 97 percent [136].

  Hyperbaric oxygen may provide benefit as a temporizing measure while definitive reperfusion is pursued, although it is not felt to promote reperfusion itself. It is associated with a low risk of systemic complications, and intracranial or systemic hemorrhage rates are not increased [77]. One case report describes a successful outcome after concurrent use of hyperbaric oxygen and tPA for CRAO [137].

- **Carbogen**, a mixture of 95 percent oxygen and 5 percent carbon dioxide, can be provided in an attempt to induce vasodilation and improve oxygenation. However, this is difficult to obtain in most hospitals on an urgent basis, and published reports are not supportive of its efficacy [96,125].

Branch retinal artery occlusion — Given the less dire prognosis for BRAO, acute treatments with uncertain efficacy are generally not provided. Clinical efforts instead focus on determining the underlying etiology and preventing future vascular events. (See 'Secondary prevention' below.)

Patients with giant cell arteritis — An important exception is the use of corticosteroids in the rare patient with retinal artery occlusion secondary to giant cell arteritis (GCA) [138]. In these patients, prompt initiation of corticosteroids improves the prognosis for vision in both the affected and the unaffected eye. (See "Treatment of giant cell arteritis".)

PROGNOSIS AND SECONDARY PREVENTION

Clinical course — In most cases of CRAO, apparently normal perfusion is restored within hours or days, and blood flow may then appear normal on ophthalmoscopy or fluorescein angiography.

The ischemic retinal whitening can last for days before the retina returns to a normal appearance. If there is concomitant involvement of the choroidal circulation, pigmentary changes under the retina may develop after many weeks or months. Over time, the optic nerve turns pale due to loss of the normal translucent appearance of the axon bundles, which are replaced by more reflective glial tissue. The retinal nerve fiber layer gradually becomes thin. The retinal blood vessels may remain attenuated or return to their normal size.

The prognosis varies with the site of retinal artery occlusion. Most patients (80 percent) with BRAO recover normal vision [7], while spontaneous clinical improvement from CRAO is rare. In experimental studies, the retina can survive for only 90 to 100 minutes after ligation of the central retinal artery [98-100].

Visual acuity at presentation usually predicts final visual acuity in patients with CRAO. While there are reports of patients who have recovered vision days after apparent CRAO [139], most severely affected patients are left only with a temporal island of vision that allows for hand motions or counting fingers [124]. Among a number of case series, only a minority of patients (less than 20 percent) had any meaningful visual return [87,102,140].

Late ocular complications — A neovascular response may occur in patients with CRAO when enough retina survives under hypoxic conditions. Neovascularization typically becomes apparent after two or three months [98,141,142]. It may involve the retina, or worse, the iris and anterior chamber angle [143].

Subsequent complications include vitreous hemorrhage and neovascular glaucoma. Vitreous hemorrhage is unusual, occurring in less than 2 percent of patients. Neovascular glaucoma has been reported to occur in up to 18 percent of cases and can be severe and refractory to treatment [13,141,144,145]. Enucleation may be required if glaucoma causes unbearable pain. (See "Open-angle glaucoma: Epidemiology, clinical presentation, and diagnosis".)

Future vascular event risk — Older case series report that patients with retinal artery occlusion, particularly CRAO, are at risk of cardiovascular as well as cerebrovascular events and have reduced life expectancy [18,88,146-148].

Risks and modification of risks are etiology specific. Those with unidentified etiology after extensive diagnostic work-up have a good prognosis with regard to future vascular events [8,18,26,149].

Secondary prevention

Diagnosis of etiology — A suggestive medical history may tailor the diagnostic work-up to the most likely etiology. In most cases, testing will include:

- **Carotid artery imaging** – A carotid duplex ultrasound, cervical magnetic resonance imaging, or computed tomographic angiography will, in most cases, be the first test ordered, as carotid atherosclerosis is the most common cause of CRAO and BRAO. The presence of visible emboli on funduscopic examination is not predictive of a hemodynamically significant carotid artery stenosis [19]. (See "Evaluation of carotid artery stenosis".)

- **Exclusion of giant cell arteritis** – Giant cell arteritis (GCA) should be excluded in all patients with CRAO who are over 50 years in age and have no visible retinal embolus. This should be done emergently on presentation. (See 'Diagnosis' above.)

- **Cardiac evaluation** – Cardiogenic embolism should be ruled out in patients in whom carotid disease has been excluded (table 1). Testing may include Holter monitoring and echocardiography [150]. The details of this evaluation are discussed elsewhere. (See "Overview of the evaluation of stroke", section on 'Cardiac evaluation'.)

The yield of cardiac testing is low in this setting [8,35]. In one case series, while 48 of 104 individuals with either CRAO or BRAO had abnormalities on transthoracic echocardiography, in only 12 were the findings felt to warrant long-term anticoagulation, suggesting that in the other 36, the findings may not have been clinically relevant [36]. Similar results are reported in other case series [31,35,151]. A higher rate of abnormalities (72 percent) is reported in patients who undergo transesophageal echography [150,152]. However, most of these additional findings represent entities in which the association with embolism and indications for anticoagulation are uncertain (eg, mitral valve prolapse, aortic arch atheroma). In most cases of isolated retinal artery occlusion, a transthoracic echocardiogram is sufficient to screen for structural cardiac disease that requires anticoagulation.

A baseline electrocardiogram (ECG) should also be included in the evaluation of these patients, as cardiac morbidity and mortality in patients with retinal artery occlusion is significant [18,88,146,147].

- **Hypercoagulable testing** – Hypercoagulable testing should be performed in individuals who have suggestive histories (prior thrombosis, miscarriage, or family history), as well as in individuals with an otherwise negative work-up [153]. (See "Evaluating adult patients with established venous thromboembolism for acquired and inherited risk factors", section on 'Hypercoagulable tests'.)

A significant number of cases (up to 20 percent in older and 50 to 67 percent in younger patients) have no identified etiology after extensive diagnostic work-up [8,18,26].

Treatment — Long-term management of these patients focuses on preventing recurrent vascular events and is cause specific. Patients with a cardiac source of emboli will often be managed with chronic anticoagulation, and patients with high-grade carotid disease will likely undergo carotid endarterectomy. Patients with an uncertain etiology should be treated with antiplatelet therapy and atherosclerosis risk factor modification, if applicable.

Secondary prevention of vascular events in these conditions is discussed in detail separately. (See "Long-term antithrombotic therapy for the secondary prevention of ischemic stroke" and "Overview of secondary prevention of ischemic stroke" and "Atrial fibrillation in adults: Use of oral anticoagulants" and "Management of symptomatic carotid atherosclerotic disease" and "Overview of secondary prevention for specific causes of ischemic stroke and transient ischemic attack".)

SUMMARY AND RECOMMENDATIONS

- **Epidemiology and etiologies** – Symptomatic retinal artery occlusion is a rare clinical event.

  Etiologic mechanisms overlap with those of ischemic stroke. Carotid artery atherosclerosis is the most common etiology and carries a high risk of future cerebral infarction. Cardiogenic embolism is the most likely etiology in young patients. Other causes include clotting disorders, vasculitis, and other vascular diseases. (See 'Etiology' above.)

  Giant cell arteritis (GCA) is an infrequent cause of central retinal artery occlusion (CRAO) in older patients but is important to diagnose because prompt treatment can improve visual outcome. (See 'Diagnosis' above.)

- **Clinical presentation and diagnosis** – CRAO usually presents with severe, profound vision loss in one eye, while branch retinal artery occlusion (BRAO) presents with incomplete monocular vision loss, in which visual acuity is not usually severely affected, if at all.

  Funduscopic examination reveals an ischemic retina, which affects the entire retina in CRAO (picture 1) and a sector of the retina in BRAO. (See 'Acute clinical features' above.)

  Confirmatory testing is rarely required; fluorescein angiography can be helpful in unusual cases.

  The erythrocyte sedimentation rate (ESR) and serum C-reactive protein (CRP) should be measured in all individuals over age 50 years with CRAO to evaluate for possible GCA. (See "Diagnosis of giant cell arteritis".)

- **Acute management**

  - CRAO has a poor prognosis for spontaneous recovery of vision. However, no treatments currently available have been proven to improve visual outcomes. (See 'Acute treatment' above.)

    Intravenous tissue plasminogen activator (tPA) is a reasonable treatment for patients with CRAO who present within 4.5 hours, after a discussion of the benefits and risks. Limited observational evidence suggests that intravenous tPA may offer a substantial benefit in terms of visual recovery. However, physicians may choose to not to use thrombolysis in this setting, preferring to await the results of clinical trials in progress, given the known risks of this therapy. (See 'Revascularization' above.)

- BRAO has a much better prognosis, with 80 percent of patients recovering normal vision. Acute treatments are generally not offered. (See 'Branch retinal artery occlusion' above.)

- Patients with retinal artery occlusion secondary to GCA should receive prompt initiation of corticosteroids, which improves the prognosis for vision in both the affected and the unaffected eye. (See "Treatment of giant cell arteritis".)

- **Subsequent evaluation** – Diagnostic work-up of CRAO and BRAO focuses on identifying the underlying etiology in order to institute appropriate secondary prevention measures to reduce the risk of future vascular events.

  Unless the etiology is apparent, we recommend performing a carotid imaging study and an ECG. Other tests (echocardiography, coagulation tests) are often performed. (See 'Diagnosis of etiology' above.)

- **Long-term management** – Treatment to prevent vascular events is cause specific. For patients with an uncertain etiology of retinal artery occlusion, we recommend atherosclerosis risk factor modification and treatment with an antiplatelet agent are appropriate. (See 'Secondary prevention' above and "Prevention of cardiovascular disease events in those with established disease (secondary prevention) or at very high risk".)

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