CAROTID ATHEROSCLEROTIC DISEASE most commonly manifests itself in two ways: as the clinical syndrome of transient monocular blindness (or amaurosis fугаx), and by the finding of embolic material in retinal arterioles. These two ocular manifestations are widely recognized. Fisher1 reviewed a variety of clinical settings in which transient monocular blindness occurs and presented six cases in which transient monocular blindness preceded the development of a contralateral hemiplegia. Transient monocular blindness in patients in the stroke-aged population subsequently became a common indication for arteriography and surgical correction of atherosclerotic lesions in the extracranial carotid system. Fisher subsequently described the fundus findings in a patient during an episode of transient monocular blindness and reported an intravascular column of white material, presumably composed of fibrin-platelet material, which moved quickly through the retinal arterial tree to disappear with return of the patient’s vision.2 Since these fibrin-platelet emboli are present only briefly during the patient’s symptoms, it is uncommon for them to be observed. Cholesterol emboli, however, tend to lodge at retinal arterial bifurcations where they may persist for long periods of time, allowing their detection during funduscopic examination. Since cholesterol emboli often do not occlude the arteriole, there may be no associated visual dysfunction and the embolus may be an isolated finding in an asymptomatic patient. Nevertheless, their importance as a sign of carotid atherosclerosis was demonstrated in a series of articles by Hollenhorst3-5 and their presence also became a widely recognized indication for evaluation of the carotid system and surgical correction of presumably responsible atherosclerotic lesions in the cervical carotid arteries.

Other ocular effects of ischemic cerebrovascular atherosclerosis have been well described but are less familiar to many clinicians. These include venous stasis retinopathy and its sequel, ischemic oculopathy. The recent development of sophisticated vascular reconstructive procedures warrants the reassessment of these ocular ischemic processes and their potential for treatment. Like amaurosis fugax, these ischemic ocular changes provide an indication of insufficient blood flow in the carotid system. The subsequent diagnostic and therapeutic considerations are guided by two possible goals: the prevention of cerebral stroke and the preservation of normal ocular function.

Clinical Findings in Patients With Venous Stasis Retinopathy and Ischemic Oculopathy

Ischemic oculopathy consists of a progressive series of pathological changes which begin in the posterior segment of the eye. It begins with a retinopathy and, if unchecked, ends with pathological changes throughout the eye and neovascular glaucoma. When the ischemic changes are limited to the posterior segment of the eye, the condition is usually termed “venous stasis retinopathy”.6 If progression occurs with more extensive involvement of the eye, then the condition is termed ischemic oculopathy.7

Venous stasis retinopathy had been recognized as a manifestation of aortic arch syndromes8-10 for some years prior to 1963 when Hedges11 and Kearns and Hollenhorst12 called attention to the association of venous stasis retinopathy and atherosclerotic carotid occlusive disease. The earliest changes of venous stasis retinopathy consist of microaneurysms and small dot-and-blot intraretinal hemorrhages along with occasional nerve fiber layer splinter hemorrhages. These findings are most prominent beginning at the vascular arcades and extending throughout the midperiphery of the fundus. More severe ischemia produces dilatation and darkening of the retinal veins, often with marked

SUMMARY Venous stasis retinopathy and ischemic oculopathy are ocular manifestations of ischemia in the distribution of the carotid artery. While not as common as transient monocular blindness or retinal arterial emboli, they are readily recognizable and indicate the presence of severe, often bilateral, carotid occlusive disease. Patterns of occlusion vary but usually include complete occlusion of at least one common or internal carotid artery, often accompanied by occlusion or narrowing in the opposite carotid system. The ocular findings in venous stasis retinopathy and ischemic oculopathy indicate ongoing ocular ischemia and may progress to intractable neovascular glaucoma. Therapy, individualized for the specific pattern of occlusive changes, may be directed toward prevention of stroke or may be indicated primarily for the reversal of ocular ischemia and prevention of blindness secondary to neovascular glaucoma.


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COMMENTS, OPINIONS, AND REVIEWS

Chronic Ocular Ischemia And Carotid Vascular Disease

JOHN E. CARTER, M.D.

SUMMARY Venous stasis retinopathy and ischemic oculopathy are ocular manifestations of ischemia in the distribution of the carotid artery. While not as common as transient monocular blindness or retinal arterial emboli, they are readily recognizable and indicate the presence of severe, often bilateral, carotid occlusive disease. Patterns of occlusion vary but usually include complete occlusion of at least one common or internal carotid artery, often accompanied by occlusion or narrowing in the opposite carotid system. The ocular findings in venous stasis retinopathy and ischemic oculopathy indicate ongoing ocular ischemia and may progress to intractable neovascular glaucoma. Therapy, individualized for the specific pattern of occlusive changes, may be directed toward prevention of stroke or may be indicated primarily for the reversal of ocular ischemia and prevention of blindness secondary to neovascular glaucoma.

irregularity of caliber of the major retinal veins. There may be mild swelling of the optic disc (fig. 1). Still more severe ischemia may produce pallor or a gray cast to the macula due to retinal edema, similar to that seen in central retinal artery occlusion. While visual impairment should always coexist with retinal edema, the remainder of the signs of venous stasis retinopathy may be present with normal visual function. Venous stasis retinopathy may be seen in patients with a recent stroke, with ongoing transient monocular or cerebral ischemic attacks, with known cerebrovascular disease in the past but no recent symptoms, with symptoms limited to mild visual loss, or occasionally as an incidental finding.

Ischemic oculopathy describes additional changes which take place in the anterior segment of the eye if the ischemic state persists. These findings were also recognized earlier in aortic arch syndromes. The most important aspect of ischemic oculopathy is the endpoint, neovascular glaucoma. Smith initially called attention to the association of neovascular glaucoma with occlusion of the internal carotid artery. The spectrum of changes seen in ischemic oculopathy were subsequently described in detail by Hoefnagels and Knox. The abnormalities include episcleral vascular congestion, a cloudy cornea, cells and flare in the anterior chamber, and neovascularization of the iris (fig. 2). The pupil is often mid-dilated and reacts sluggishly or not at all to either direct or consensual stimulation. Intraocular pressure is usually elevated but may be normal or even decreased. In most cases venous stasis retinopathy continues to be present, but occasionally the retinal arteries and veins may become markedly attenuated with visibly sluggish blood flow through the veins. Optic atrophy may develop.

Patients with venous stasis retinopathy or ischemic oculopathy invariably have a marked reduction of the central retinal arterial pressure as measured by ophthalmodynamometry. Often the pressure is so low that touching the eye produces pulsation in the central retinal artery and the pressure can not be measured. In some cases diastolic pressure in the central retinal artery is so low that spontaneous pulsations of the central retinal artery are present.

Ischemic pain is a feature in some patients with venous stasis retinopathy or ischemic oculopathy. It is characterized as a constant aching over the orbit, upper face and temple and may be worse when the patient is upright. This ischemic pain is not related to glaucoma which may also produce pain in patients with ischemic oculopathy. Five percent of patients at the Mayo Clinic have chronic ischemic pain associated with occlusion of the internal carotid artery. Other series do not distinguish between ischemic pain and pain due to glaucoma. Because of its eminent treatability, temporal arteritis should be excluded with an erythrocyte sedimentation rate when this pain is a feature of the patient’s history.

Pathogenesis of Ischemic Oculopathy

With transient monocular blindness the ischemic state lasts only a few seconds or minutes, with complete restoration of circulation and of retinal function. In a typical retinal arterial occlusion, the obstruction to blood flow may be brief or permanent, but is severe enough to produce infarction of the retina. Once infarction has occurred and the necrotic tissue has been removed by phagocytosis, an ischemic state no longer exists. There is no viable but ischemic tissue in which to generate an ischemic response. Venous stasis retinopathy appears to represent a state of “idling ischemia” with viable but ischemic tissue. If the ocular ischemia persists, the retinopathy progressively worsens and neovascular changes develop in the retina and optic disc followed by neovascularization of the iris. Neovascularization of the iris eventually produces a fibrovascular membrane which obstructs the outflow of aqueous through the anterior chamber angle and causes glaucoma. Iris atrophy also develops.

A similar series of events occurs in patients with diabetic retinopathy and in some patients with central retinal vein occlusion. In both of these conditions, the primary process is believed to occur in the retina where ischemia results in the elaboration of a diffusible angiogenesis factor. Iris neovascularization develops when this factor circulates to the anterior chamber. Ablation of retinal tissue by panretinal photocoagulation decreases the oxygen requirements and produces a relative decrease in the ischemic state. If panretinal photocoagulation is performed prior to the development of the fibrovascular membrane and angle closure, neovascular changes in the retina and iris may resolve and neovascular glaucoma may be prevented. All of the anterior segment changes in diabetes and central retinal vein occlusion may be explained by the ischemic processes occurring in the retina. There is evidence that the neovascular changes occurring in ischemic oculopathy are also secondary to the posterior segment ischemia rather than being a direct effect of ischemia on the anterior segment. However, there is also evidence of a direct effect of ischemia on the anterior segment of the eye in ischemic oculopathy which is not seen in other types of neovascular glaucoma. Some patients with fully developed ischemic oculopathy and complete angle closure nevertheless have normal or low intraocular pressure. Decreased aqueous production and ocular hypotony have been described in experimental and therapeutic ligation of the common carotid artery, presumably due to ischemia of the ciliary body. Additional evidence that ciliary body ischemia is responsible for normal or low intraocular pressures in patients with complete angle closure due to ischemic oculopathy is the fact that these patients may develop elevated intraocular pressures following surgical bypass or repair of obstructive lesions in the carotid circulation.

Ischemic Oculopathy and Diabetes

Venous stasis retinopathy resembles diabetic retinopathy and this similarity may contribute to the low diagnostic rate for venous stasis retinopathy. However, there are features which distinguish the two processes (table 1). Diabetic retinopathy is seldom unilateral and the retinopathy is most concentrated in the
FIGURE 1. Early and advanced venous stasis retinopathy. A-C were taken one month after occlusion of the ipsilateral common carotid artery. Despite a subjective sense of visual blurring, the visual function was normal. A. There is mild swelling of the optic disc. The veins are dilated and show segmental constriction. In addition to the several large hemorrhages, a number of small punctate hemorrhages can be seen along the retinal veins. B. The macular region shows minimal involvement. The white punctate lesions are macular drusen, an incidental finding. C. Superior to the disc along the vascular arcade the venous changes and small perivenous hemorrhages continue to be present. D-F were taken in the same patient six months after the occlusion and show progression of the retinopathy. D. The optic disc and nasal retina. The mild disc swelling seen originally is improved and the dilation and segmental constriction of the major retinal veins is still present but less marked. There has been a marked increase in the number of intraretinal hemorrhages. E. The macular region has a few hemorrhages which increase in number approaching the vascular arcade until they are as numerous as noted in the nasal retina. F. Superior to the optic disc, the venous engorgement continues to be prominent although the segmental constriction is less notable. Again, there has been a dramatic increase in the number of intraretinal hemorrhages. All changes resolved following panretinal photocoagulation.22

(Photography courtesy of Mr. Roy Wilson, reproduced by permission from the Annals of Ophthalmology 16: 573, 1984.)
Vascular Status of Patients with Ocular Ischemia

In their original description, Kearns and Hollenhorst found venous stasis retinopathy in 4% of 600 patients presenting with transient or fixed deficits attributed to atherosclerotic carotid vascular disease. In some of the examples cited, the venous stasis retinopathy was relatively mild, consisting of scattered microaneurysms and capillary dilatation in one segment of the retina. Arteriographic studies were limited. The thorough studies of the pathological anatomy of the entire extracranial cerebrovascular system which are routine today were not commonly done at that time. A subsequent report by Kearns et al of patients with arteriographically demonstrated occlusion of the internal carotid artery found venous stasis retinopathy in 11 of 60 patients with unilateral internal carotid artery occlusion and in 1 of 12 patients with bilateral occlusions, some of whom had progressed to fully developed ischemic oculopathy. These reports clearly demonstrated the association between venous stasis retinopathy and atherosclerotic cerebrovascular disease, but they indicate it is not a particularly common finding. Nevertheless, when found in a patient with classic transient monocular blindness, cerebral transient ischemic attacks or stroke, the presence of venous stasis retinopathy or ischemic oculopathy confirms the already suspected cerebrovascular disease.

A more important consideration may be the value of venous stasis retinopathy and ischemic oculopathy as a marker for cerebrovascular disease when it is found incidentally or in patients whose complaints are not those classically thought of as pointing to cerebrovascular disease. In this manner it is similar to the incidental finding of cholesterol plaques in the retinal arteries. The numerous earlier reports of ischemic oculopathy in patients with aortic arch syndrome, and the relative paucity of cases due to atherosclerotic carotid vascular disease, suggests that cases demonstrating persistent ocular ischemia will have major and perhaps multiple-vessel occlusive disease. The literature contains case descriptions or tabular data with arteriography of 30 non-diabetic patients with venous stasis retinopathy or ischemic oculopathy and 11 diabetic patients with asymmetrical ocular findings leading to the diagnosis of carotid occlusive disease. Table 2 presents a compilation of the presenting complaints, findings and arteriographic findings in the 30 non-diabetic patients. Table 3 summarizes the arteriographic findings for these patients as well as for the diabetic patients with asymmetric ocular disease due to venous stasis retinopathy or ischemic oculopathy. Venous stasis retinopathy may occur in only 4% of patients with carotid occlusive disease and only 16–18% of patients with complete occlusion of an internal carotid artery. When present, however, venous stasis retinopathy and ischemic oculopathy indicate with a very high probability that major cervical carotid occlusive disease is present.

**Diagnostic and Therapeutic Approach to Patients With Ischemic Ocular Disease**

The evaluation and therapy of patients with venous stasis retinopathy should be a cooperative effort be-
between the ophthalmologist and the physician skilled in the management of cerebrovascular disease. Venous stasis retinopathy has a characteristic appearance which can be mimicked by three things: diabetic retinopathy, partial central retinal vein occlusion, and a retinopathy produced by hyperviscosity states such as polycythemia vera, lymphoma, leukemia, sickle cell disease, and dysproteinemias. Diabetic retinopathy should be distinguished from venous stasis retinopathy by the features noted earlier. The hyperviscosity states may closely mimic venous stasis retinopathy but segmentation of veins is not characteristic, it is usually bilateral, and blood studies should provide a diagnosis. The characteristic fundus appearance of a florid central retinal vein occlusion (figure 3) should not be confused with venous stasis retinopathy. More recently a situation in which venous outflow from the eye is only partially obstructed has been recognized and noted to present a funduscopic picture very similar to venous stasis retinopathy. In distinguishing between partial central retinal vein occlusion and the venous stasis retinopathy of carotid occlusive disease, Kearns states that segmentatin of the veins is characteristic of venous stasis retinopathy while disc edema is characteristic of central retinal vein occlusion. Low retinal artery pressure is the most specific distinguishing feature. Because of the severe degree of occlusive disease asso-

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CCA = common carotid artery; ICA = internal carotid artery.
citated with venous stasis retinopathy, noninvasive carotid vascular studies such as ophthalmodynamometry, doppler, echography, or ocularplethysmography should be very reliable in distinguishing between partial central retinal vein occlusion and venous stasis retinopathy.

The ischemic changes in the anterior segment of the eye are less specific. Gartner and Henkind noted that iris neovascularization was associated with 41 different entities, most commonly central retinal vein occlusion, diabetic retinopathy, uveitis, and open angle glaucoma but also including systemic lupus erythematosus, sickle cell disease, retinal detachment and giant cell arteritis. Carotid insufficiency was responsible for only 8% of patients with iris neovascularization in Hoskins’ series, but it was still the fourth most common cause after central retinal vein occlusion, diabetes, and uveitis. In evaluating patients referred with unilateral neovascular glaucoma unassociated with central retinal vein occlusion, retinal detachment or intraocular tumor, Brown et al found that 11 of 12 were caused by major carotid artery occlusive disease. Five were diabetic but had unilateral retinopathy. In the absence of a primary ophthalmic cause for neovascular changes, unilateral neovascularization of the retina or iris strongly suggests the diagnosis of carotid artery occlusive disease. A careful examination in conjunction with the ophthalmologist and routine laboratory studies should exclude other causes for ocular neovascularization and allow appropriate cerebrovascular evaluation with arteriography. Intravenous digital subtraction angiography, which is rapidly becoming available on an outpatient basis, should provide sufficient information to establish a diagnosis in many cases.

While the presence of venous stasis retinopathy or ischemic oculopathy clearly indicates major carotid occlusive disease, the appropriate therapeutic intervention is less certain. As in the case of amaurosis fugax and retinal artery emboli, therapeutic considerations may be directed toward preventing stroke or toward preserving normal visual function.

In the case of preventing stroke, the difficulty lies in the degree of occlusive disease present at the time of the diagnosis. There would be little argument about performing endarterectomy on a stenotic lesion on the symptomatic side, but this is seldom the situation. Although still a point of some controversy, the conventional wisdom in cerebrovascular disease is to “operate on the symptomatic side.” Venous stasis retinopathy and ischemic oculopathy may be exceptions. The frequent occurrence in these patients of major bilateral carotid occlusive disease and symptoms and signs limited to the eye may indicate that intracranial blood flow is being distributed via whatever primary or collateral pathways are available at the expense of the ocular circulation. If a major source of intracranial blood flow is from the external carotid artery via maxillary and superficial temporal arteries to the ophthalmic artery, blood flow through the ophthalmic artery is reversed and the ocular circulation may be a cul-de-sac with minimal blood flow. Huckman and Haas provided angiographic evidence of such an “ophthalmic artery steal” in two patients with ischemic oculopathy. Kearns et al also described such a patient with venous stasis changes and labeled this is “collateral flow retinopathy.” Due to complex collateral pathways, the “symptomatic circulation” may be some combination of both carotid circulations or even the carotid circulations and the vertebrobasilar circulation. After reviewing Table 2 it seems reasonable to consider endarterectomy on a significant stenosis of the internal carotid artery even though it is on the opposite side, or on a significant stenosis of an external carotid if arteriography indicates that it is a major remaining source of intracranial blood flow. Extracranial-intracranial (EC-IC) bypass may also be considered if the cervical carotid disease consists of internal carotid artery occlusion and no additional stenoses which are decreasing intracerebral blood flow. EC-IC bypass has intuitive appeal in that it is directed at the primary problem, that is, decreased cerebral blood flow. However, it must be recognized that no controlled study has been done regarding the efficacy of EC-IC bypass in cerebrovascular disease. Some patients may have lesions which are not operable, such as an occlusion of the common carotid artery on one side with normal findings or an occlusion of the internal carotid artery on the opposite side. An initial trial of medical management for cerebral ischemic symptoms would appear to be the most logical approach to such a patient. Probably the most accurate statement regarding stroke prevention therapy in patients with venous stasis retinopathy or ischemic oculopathy is that each case must be individualized because of the variety of stenoses and occlusions which are possible.

The same statement is even more true when considering therapy directed at the ocular ischemia. In evaluating any therapy, the natural history of the disease must be considered. In the original report of venous stasis retinopathy by Hedges, the patient’s acuity and visual fields returned to normal and there was resolution of all retinal changes except for a few hemorrhages despite an unsuccessful endarterectomy on a
complete occlusion of the internal carotid artery. This case suggests that venous stasis retinopathy may be an acute, transient process which resolves with development of collateral circulation, and parallels the spontaneous resolution of retinopathy seen after carotid ligation in most of Swan and Raaf's patients. Other patients have a progressive increase in the retinopathy over a period of several months before developing neovascularization of the disc and peripheral retina and finally iris neovascularization. Therapy directed solely at the ocular ischemia might therefore be delayed if the onset of venous stasis retinopathy is recent as identified by the recent sudden onset of ischemic cerebral symptoms. In the remainder of the cases of venous stasis retinopathy, therapy can probably be initiated early. The presence of any neovascular changes indicates a chronic process and argues for immediate intervention. Iris neovascularization may regress with therapy, but this must occur prior to closure of the angle by the fibrovascular membrane if neovascular glaucoma is to be prevented.

Because most patients with venous stasis retinopathy or ischemic ocularopathy undergo vascular surgery directed at protecting the brain, there has been considerable experience with the effect of carotid endarterectomy and EC-IC bypass on these ocular conditions. Both surgical procedures have produced documented resolution of established venous stasis retinopathy and ocular neovascularization. Resolution of ischemic pain, a primary indication for surgery in some patients, following EC-IC bypass further supports the efficacy of this procedure. Despite the intuitive appeal and anecdotal efficacy, cerebral revascularization procedures do have important associated morbidity and mortality which must be considered in choosing a therapeutic approach.

Panretinal photocoagulation provides an alternative therapy for ocular ischemia induced by carotid occlusive disease. Retinal photocoagulation is standard therapy for conditions such as diabetic retinopathy and central retinal vein occlusion in which retinal ischemia results in neovascularization. Regression of the retinopathy and neovascularization of the optic disc and iris after panretinal photocoagulation has also been reported in one case caused by carotid occlusive disease. In some cases, such as complete occlusion of the common carotid artery, retinal photocoagulation may be the only readily available therapeutic approach. In other cases, such as a completed stroke and occlusion of the internal carotid artery but with established collateral flow to intracranial circulation, retinal photocoagulation may offer a low risk alternative for patients in whom the risk of future stroke is considered low or in whom major surgery is undesirable.

No therapy is uniformly effective in correcting the ocular ischemic process. Retinal photocoagulation has been used in one patient medically unable to undergo surgery with only a temporary benefit. Surgery has produced a transient increase in ocular neovascular changes and sustained intraocular pressure elevations. One patient had persistent venous engorge-

ment and tortuosity five months after EC-IC bypass. Some variability in the ocular response to EC-IC bypass should be expected since only slight improvement of retinal artery pressure occurs following that procedure. Only additional experience and careful follow-up studies will determine if there is a group of patients who will need adjunctive therapy with panretinal photocoagulation for venous stasis retinopathy or early iris neovascularization which persists or progresses despite revascularization procedures. Patients presenting with significant visual loss and marked ocular changes (retinal edema, cells and flare in the anterior chamber, elevated intraocular pressure) seldom improve with therapy. Kiser et al reported recovery of vision after very aggressive initial therapy aimed at decreasing ocular pressure, followed by arteriography and EC-IC bypass. The patient had been symptomatic for only two weeks.

Conclusion

Venous stasis retinopathy and ischemic ocularopathy are less familiar ocular manifestations of cerebrovascular disease than transient monocular blindness and incidentally discovered cholesterol emboli. They are, however, readily recognizable and highly specific for major, often bilateral, carotid occlusive disease. In addition to warning of potential cerebrovascular insufficiency, these conditions indicate ongoing ocular ischemia which has the potential for progressing to intractable neovascular glaucoma. If therapy is undertaken prior to closure of the anterior chamber angle, retinal and iris neovascularization may both resolve and glaucoma may be prevented and vision can be preserved.

The many combinations of stenosis and occlusion which are possible require individualization of therapy on a case-by-case basis. Some general guidelines may be applicable to situations in which ocular ischemia is present.

1. Ocular ischemia, with or without cerebral ischemia, and a tight stenotic lesion: endarterectomy.
2. Ocular ischemia discovered on evaluation for acute stroke, and with occlusion of the internal carotid artery: observe patient for progression or spontaneous resolution of venous stasis retinopathy. If progression occurs, EC-IC bypass can be considered. Panretinal photocoagulation provides an alternative therapy directed specifically at ocular ischemia.
3. Ocular ischemia, with or without cerebral ischemia, and with occlusion of the ipsilateral common carotid artery: panretinal photocoagulation should be the initial therapy. Intermittent cerebral ischemic symptoms in this situation should probably also be treated initially by medical means.

A careful assessment of flow patterns during angiography may be useful in determining the therapy for other patterns of occlusive disease.
References

Chronic ocular ischemia and carotid vascular disease.

J E Carter

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