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CASE REPORT

Idiopathic Recurrent Branch Retinal Arterial Occlusion in a Young Adult

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ABSTRACT: A case of bilateral, idiopathic, and recurrent branch retinal arterial occlusions occurring in an otherwise healthy 36-year-old woman is presented. The diagnosis of idiopathic recurrent branch retinal arterial occlusion was made after extensive medical investigations failed to find a cause for the occlusions. The differential diagnosis of retinal arterial occlusion in the young is discussed. (Optom Vis Sci 1998;75:11–16)

Key Words: Eales' disease, retinal artery occlusion, retinal arteriolitis, Susac's syndrome

A syndrome has been described of idiopathic, bilateral, recurrent branch retinal arterial occlusions that are believed to be due to a multifocal retinal arteriolitis.[1, 2] This rare disorder occurs in otherwise healthy middle-aged individuals, and may be associated with tinnitus, hearing loss, and vertigo. Central vision is usually spared. Neovascularization of the retina, optic disc, or iris develops in 25% of affected eyes with potentially devastating visual consequences.[2, 3] Because there is considerable overlap between the clinical features of this condition and Eales' disease, it is suspected that patients with idiopathic recurrent branch retinal arterial occlusion may constitute one group of patients normally lumped into the general category of Eales' disease.[1] We present the findings of a case and discuss the differential diagnosis of retinal arterial occlusion in the young.

REPORT OF A CASE

A 36-year-old white female presented to a local emergency room on 9 May 1993 with the complaint that a focal headache on the left side of her head radiated to the entire left side of the head over the course of the previous 4 days. She also was experiencing a visual disturbance in her left eye, as if "someone had just taken a picture of her with a flash bulb." Additionally, she reported intermittent numbness of her left hand. Medical examination by the attending physician was normal and Fiorinal with codeine was prescribed for possible migraine headache. She was instructed to follow-up with her primary care physician in 2 days if symptoms persisted.

She presented to our eye care department 5 days later with symptoms of a "gray haze" in the vision of her left eye of 2 weeks duration. Best corrected vision was 6/6 (20/20) in each eye. Pupil reflexes were intact, and without an afferent defect. Ocular motility was normal. Tonometry was 13 mm Hg in each eye. Slitlamp examination was unremarkable. Ophthalmoscopy through dilated pupils was normal for the right eye, but a region of retinal edema and opacification was present along the course of the inferior-temporal artery in the left eye (Fig. 1). There was no apparent embolus. Additionally, there was a region of retinal edema nasal to the disc in this eye, possibly a resolving infarct. The cup-to-disc ratio was 0.1 in each eye, and the optic discs were pink and non-edematous. A diagnosis of branch retinal artery occlusion was made. Negative studies at this time included duplex carotid Doppler ultrasonography, two-dimension echocardiography, complete blood count, rheumatoid factor, rapid plasma reagin, fasting glucose, and antinuclear antibody. The retinal lesions had resolved at follow-up examination 1 month later.

The patient returned for a routine eye examination 2 years later on 24 March 1995. The patient had remained asymptomatic since her last eye examination. She was taking Naprosyn for a muscle strain injury, but was otherwise well. She had a history of head and neck aches and paresthesias in her arms after an automobile accident in 1992. She reported a positive paternal family history of glaucoma. Best corrected vision was 6/6 (20/20) in each eye. Tonometry readings were 19 mm Hg in each eye. Pupil reflexes, ocular motility, and biomicroscopy were normal. Ophthalmoscopy of the right eye was unremarkable; the left eye had a sclerosed blood vessel nasal to the optic disc in the region of the previously observed retinal edema, but was otherwise normal. The cup-to-disc ratio was 0.1 in each eye. Laboratory testing to rule out vasculitis was undertaken again at this time, including Lyme antibody. All tests were negative. Perimetry revealed superior-nasal and inferior-nasal defects in the right eye and two scotomas in the left eye, one inferior-nasal and the other just superior-temporal to fixation (Fig. 2). The field defects were thought to represent vision loss from asymptomatic retinal infarctions, but to rule out occult

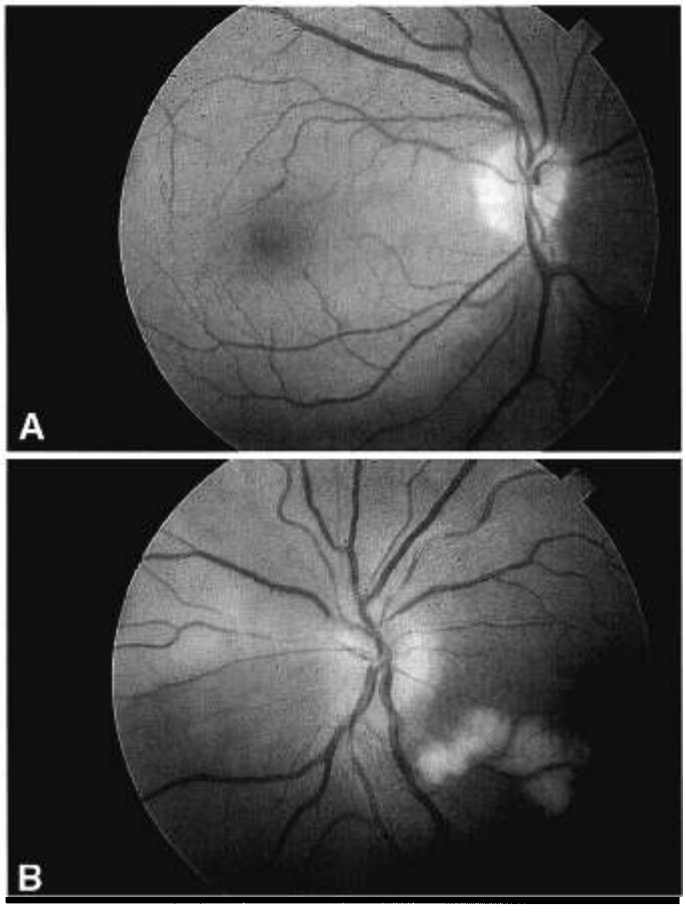

FIGURE 1.

A: Normal right fundus. B: Left inferior temporal branch retinal artery occlusion without visible embolus. Retinal edema nasal to disc suggestive of previous asymptomatic infarct.

glaucoma the patient underwent serial tonometry. Seven readings were obtained at various times of day. No reading exceeded 19 mm Hg.

The patient presented to our clinic again on 28 June 1996 with symptoms of constant "flash bulb glare" in the vision of her left eye of 1 day duration. Visual acuity was 6/6 (20/20) and 6/7.5 (20/25) in the right and left eyes, respectively. Tonometry was 14 mm Hg OD and 12 mm Hg OS. Pupil reflexes were intact and without an afferent defect. Ophthalmoscopy of the right eye was unremarkable. The left eye had a region of retinal edema along the superior temporal vascular arcade. There was no embolus, and the occlusion clearly did not occur at a vessel bifurcation. The patient was referred to a retinologist and an internist for further evaluation. The retinologist examined the patient and performed a fluorescein angiogram. He concurred with our diagnosis of idiopathic branch retinal artery occlusion and informed the patient that there is no known cause or treatment for this condition. The internist performed a medical examination and reviewed her laboratory findings. The patient is obese (238 lbs) but otherwise in good health; specifically, there was no evidence of vasculitis, synovitis, or diabetes. There was no hypertension. She does not take oral contraceptives and had undergone tubal ligation. The patient was placed on a low-fat diet, daily baby aspirin, and told to follow-up in 3 months.

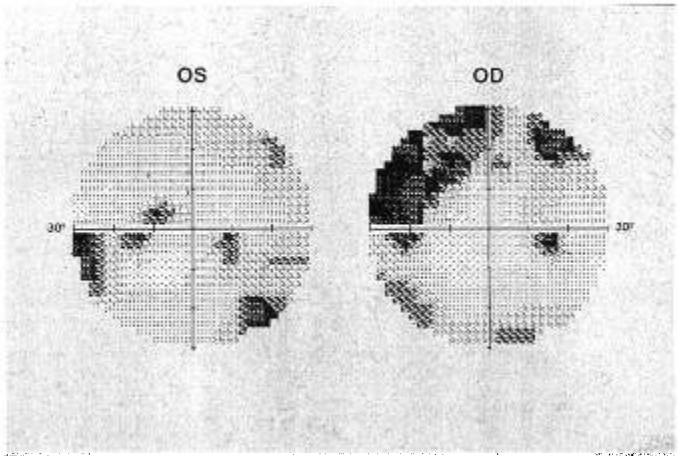

FIGURE 2.

Humphrey 24–2 gray-scale perimetric findings after the patient's first symptomatic occlusive episode reveals a scotoma superior-temporal to fixation in the left eye. This is consistent with the observed occlusion of the inferior temporal artery of this eye. Other scotomas in each eye may represent vision loss from asymptomatic occlusions.

The patient returned approximately 1 week later on 10 July 1996 with complaints of light flashes in the right eye. Visual acuity was 6/6 (20/20) in each eye. Tonometry was 15 mm Hg in each eye. The anterior segments and pupils were normal. Ophthalmoscopy of the right eye revealed an occlusion of the superior temporal artery of the right eye. There was no embolus. There was residual retinal edema along the course of the superior temporal artery in the left eye from the previous occlusion (Fig. 3). Perimetry documented progression of the field loss in each eye (Fig. 4). The patient was again examined by an internal medicine specialist, who repeated most of the previous laboratory studies including the following: amylase, thyroid stimulating hormone, glycohemoglobin, blood chemistry, urinalysis, erythrocyte sedimentation rate, hepatitis A and B antibodies, and Sjogren's syndrome antibodies. All were normal, except the sedimentation rate, which was slightly elevated at 27 mm/h (normal range: 0 to 20), and cholesterol, which was 212 mg/dl (normal range: less than 199). An audiometric screening revealed moderately severe hearing loss for frequencies between 750 to 6000 Hz. The patient was referred to rheumatology and audiometry.

The rheumatologic physical examination was normal. There was no family history of rheumatory arthritis, lupus, or thromboembolic disease. The patient recounted a long-standing complaint of headache, greater on the left side, after a motor vehicle accident in 1992, but denied jaw claudication. A left temporal artery biopsy failed to find evidence of giant cell arteritis. Negative studies at this time included activated partial thromboplastin time, prothrombin time, anticardiolipin, hepatitis C antibodies, cryoglobulins, compliment C3 and C4, lupus anticoagulant, chest X-ray, echocardiogram, and bilateral carotid Doppler ultrasound.

Formal audiometric evaluation disclosed moderately severe sensorineural hearing loss at middle and high frequencies for both ears (Fig. 5). The patient was then examined by an otolaryngologist. The findings were completely normal and the patient was referred for a hearing aid fitting.

Currently, the patient remains asymptomatic and free from oc-

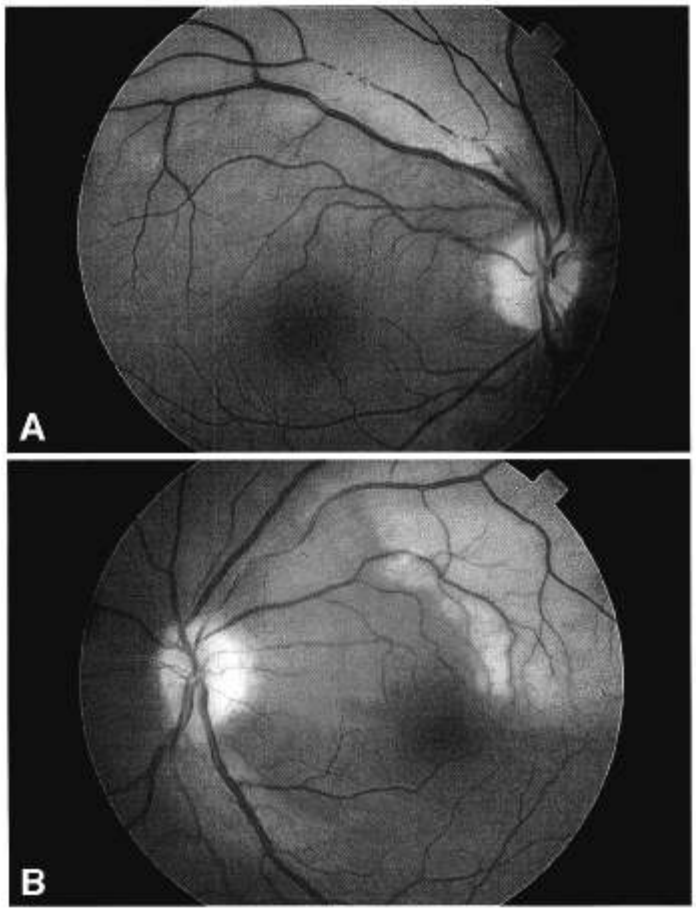

FIGURE 3.

A: Acute occlusion of a branch of the right superior temporal artery with ischemic retinal whitening and white periarterial plaques along the occluded vessel. B: Appearance of left fundus approximately 1 week after occlusion of the superior temporal artery. Note that there is now white periarterial sheathing of the arteriole nasal to the optic disc.

ular complications. She has not developed any ocular neovascularization, but continues to be monitored for it.

DISCUSSION

Retinal arterial occlusions are rare in young people. Fewer than 10% of all patients that suffer retinal arterial occlusion are under 30 years of age.[4] Unlike older adults, where approximately 75% of cases are caused by embolization from atheromatous plaques of the carotid artery,[5] retinal arterial occlusions in younger patients are associated with a diverse assortment of other predisposing conditions including migraine, coagulation abnormalities, cardiac disease, and trauma[4,6] (Table 1). Often, more than one predisposing disease state can be found in affected individuals. It has been suggested that multiple risk factors may act additively or synergistically to produce arterial occlusions in susceptible children and young adults.[4,7] When trauma-related occlusions are excluded, young women are affected twice as often as young men.[6] One explanation is that arterial occlusive disease may be related to hormonal influences and the hypercoagulable states induced by pregnancy or oral contraceptives.

Drug abuse is a frequent cause of arterial occlusion in young

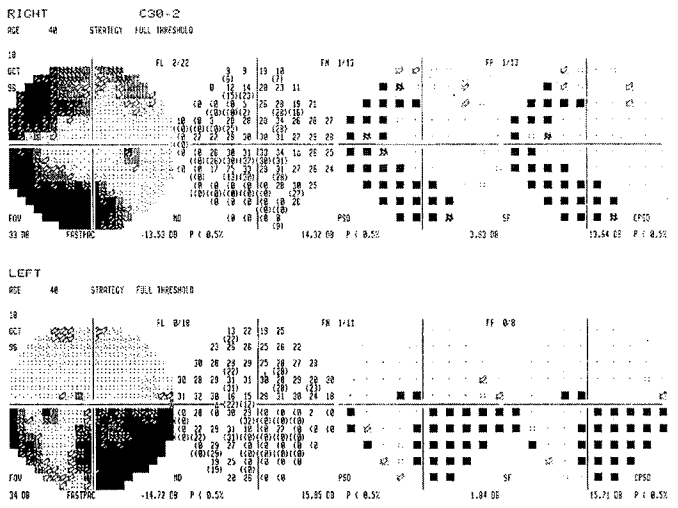

FIGURE 4.

Humphrey 24–2 perimetry documents progression of visual field loss in each eye when compared to Fig. 2.

people. Exogenous emboli such as talc may be introduced into the blood stream by intravenous drug abuse.[8] The term talc retinopathy is used to describe the characteristic appearance of multiple glistening particles lodged at arterial bifurcations in both eyes. Drug abuse has also been associated with nonembolic retinal arterial occlusions. Cocaine and methamphetamine abuse have been reported to produce central retinal arterial occlusion, presumably secondary to arterial spasm or severe transient hypertension.[9–11]

Medical evaluation of patients with retinal arterial occlusion is directed at identifying associated systemic disease[12] (Table 2, Fig. 6). Treatment is then aimed at any associated systemic condition; control of vaso-occlusive risk factors, such as cigarette smoking, oral contraceptive use, and obesity; and long-term follow-up for ocular complications such as neovascularization. Most clinicians do not treat branch retinal arterial occlusion.[13] Treatments that have been attempted for acute embolic branch retinal arterial occlusion include anterior chamber paracentesis and ocular massage. Medical lowering of intraocular pressure with systemic acetazolamide or topical timolol maleate may encourage passage of the embolus. Paper bag rebreathing, carbogen (95% carbon dioxide, 5% oxygen) inhalation, and calcium channel blockers produce vasodilation that may also facilitate passage of the embolus. Experimental surgical therapies for resistant emboli are laser photodisruption[14] and surgical embolectomy.[15] Prophylactic treatments include anticoagulation with warfarin, or antiplatelet therapy with aspirin or dipyridamole. There are, to date, no published studies to

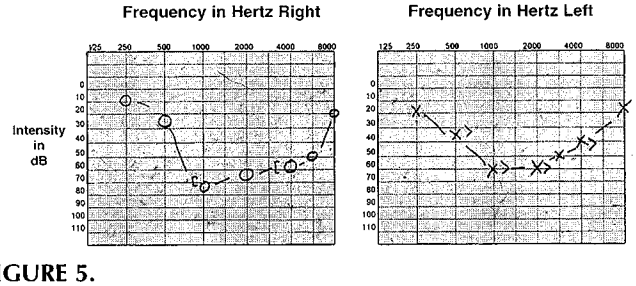

FIGURE 5.

Audiogram documents moderately severe bilateral hearing loss.

TABLE 1.
Differential diagnosis of retinal arterial obstruction in the young.[a]

Embolic
atrial myxoma
cardiac valvular disease (mitral valve prolapse, rheumatic)
premature atherosclerosis (hyperhomocysteinemia)
talc retinopathy
iatrogenic (corticosteroid injection)
Purtscher's-like retinopathy (leukoembolization)
lipid embolism (pancreatitis)
air embolism (decompression sickness, postsurgical)
Thrombosis
sickle cell hemoglobinopathy
oral contraceptives
fibromuscular dysplasia
Fabry's disease
pregnancy/postpartum
lupus anticoagulant
coagulopathies (primary anti-phospholipid antibody syndrome)
homocystinuria
radiation retinopathy
congenital prepapillary arterial loop
Inflammatory Endarteritis
systemic lupus erythematosis
polyarteritis nodosa
Behçet's syndrome
Lyme disease
idiopathic recurrent branch retinal arterial occlusion
varicella
Arterial Spasm
migraine
cocaine/methamphetamine abuse
vasospastic amaurosis fugax (Raynaud's-like phenomenon)
trauma
Vascular Narrowing from Extravascular Disease
orbital disease (fracture, cellulitis, tumor)
optic nerve disease (disc drusen, papilledema, tumor)
retinal disease (retinitis, chorioretinitis)
orbital mucormycosis
retrobulbar injection
retrobulbar hematoma
Hydrostatic
increased intraocular pressure
systemic hypotension
severance of the central retinal artery (traumatic, surgical)

[a] This table is not an exhaustive list of every possible etiology.

TABLE 2.
Proposed laboratory work-up of retinal arterial obstruction in the young.

Blood Dyscrasia Studies	
complete blood count with platelets	
sickle screen (black race)	
Coagulation Studies	
prothrombin time	
partial thromboplastin time	
lupus anticoagulant	
additional coagulation tests that may be indicated when there is a personal or family history of thromboembolic disease	protein C protein S antithrombin III antiphospholipid antibodies cryoglobulins
Vasculitis Studies	
erythrocyte sedimentation rate	
antinuclear antibodies	
rapid plasma reagin	
rheumatoid factor	
chest X-ray	
temporal artery biopsy (if exam suggests giant cell arteritis)	
Metabolic Studies	
fasting blood glucose	
lipid profile	
serum electrolytes	
urinalysis	

suggest that any treatment produces better outcomes than no treatment at all.[13, 16]

Our patient experienced multiple, bilateral branch retinal arterial occlusions without evidence of systemic disease, despite extensive medical investigations. The only positive laboratory findings were a mildly elevated erythrocyte sedimentation rate and high serum cholesterol. The patient's obesity and hypercholesterolemia placed her at risk for developing systemic arterial hypertension, a known risk factor for the development of retinal arterial occlusions,[12] but at no time was the patient's blood pressure recorded as elevated. No definitive cause for our patient's elevated sedimenta-

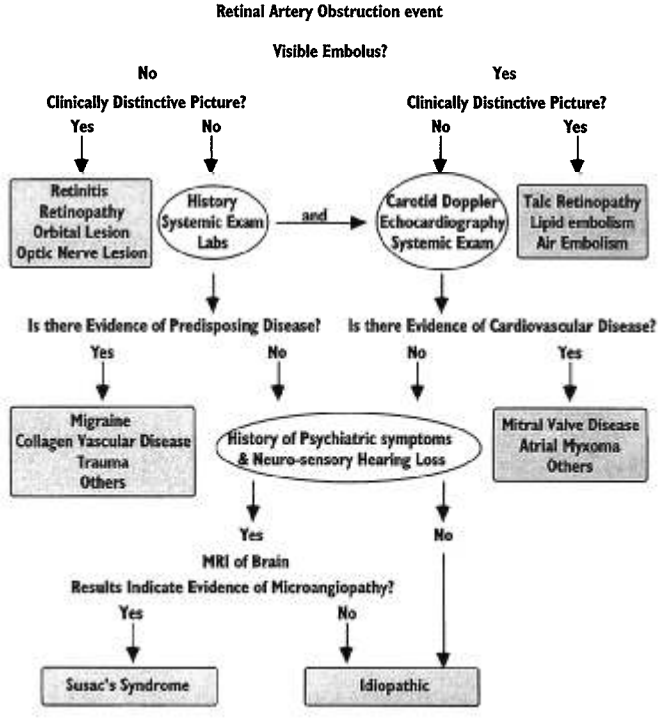

FIGURE 6.
Proposed work-up of retinal arterial obstructions in the young.

tion rate was uncovered. In Johnson et al.'s series of 16 patients with idiopathic recurrent branch retinal arterial occlusion, one case is reported as having a "mild elevation" of the erythrocyte sedimentation rate.[2] We did not test for protein C, protein S, and antithrombin III deficiencies[17] or hyperhomocysteinemia.[18] This is in keeping with current recommendations that it is cost-effective to limit testing for natural anticoagulant deficiency states to patients with histories of recurrent, familial, or juvenile venous thromboembolism.[2, 17] Our patient did not fit into any of these high-risk groups. Similarly, we elected not to test this patient for hyperhomocysteinemia because there was no clinical evidence of arteriosclerosis or thromboembolism.[19]

The diagnostic criteria for idiopathic recurrent branch retinal arterial occlusion are: (1) two or more symptomatic episodes of branch retinal artery occlusion; (2) absence of retinal emboli on dilated fundus examination; and (3) no evidence of systemic vasculitis or connective tissue disease.[2] Our patient meets each of these criteria. Because there is considerable overlap between the clinical features of this condition and Eales' disease, it is suspected that patients with idiopathic recurrent branch retinal arterial occlusion may constitute one group of patients normally lumped into the general category of Eales' disease.[1]

In this disorder, the arterial occlusions may occur at or away from bifurcations. The obstruction is associated with ischemic whitening of the retina, an absence of visible emboli, and arterial staining on fluorescein angiography.[1] Sheathing and multiple periarterial yellow-white plaques often develop along the obstructed artery and may remain permanently. Mild vitreous cells develop in about one-third of cases.[2] Gass et al. suggested that a focal retinal arteritis, perhaps caused by precipitation of immune complexes along the arterial wall, is responsible for the occlusions.[1] Subclinical viral infection has been proposed as initiating the disorder.[2]

In Johnson et al.'s series of 16 patients with idiopathic recurrent branch retinal arterial occlusion, 7 were women and the median age at onset of symptoms was 46 years (range 25 to 65 years).[2] All had evidence of bilateral ocular involvement. Over a mean follow-up of 9 years, each patient experienced an average of 4.9 occlusions (range 2 to 10). Only three eyes (9%) lost two or more lines of Snellen visual acuity from foveal ischemia. However, nine eyes (28%) had central and/or extensive peripheral visual field loss. Eight eyes (25%) developed neovascular complications, including neovascular glaucoma. Eight patients (50%) had nonvisual neurologic symptoms (vestibulo-auditory and focal neurologic) around the time of their retinal arterial occlusions. Treatment trials with aspirin, warfarin sodium crystalline, and prednisone were of little or no value in preventing new occlusions. However, two patients who began taking prednisone within 48 h of the onset of an occlusion reported an unconfirmed reduction in the size of their scotoma. No patient developed significant systemic thromboembolic disease during the follow-up period.

Our patient had significant bilateral sensorineural hearing loss. This is consistent with the clinical picture of idiopathic recurrent branch retinal arterial occlusion.[2] It is noteworthy that she did not experience tinnitus in association with her retinal arterial occlusions. Six patients in Johnson et al.'s series experienced vestibulo-auditory symptoms (tinnitus, vertigo, and/or hearing loss), four of whom developed mild to moderate permanent hearing loss. Only one of the four patients had hearing loss without tinnitus. Our patient had a history of left-sided headache and parasthesias in her arms. This had been attributed to a motor vehicle accident in which she suffered contusions and muscle strain injuries to her neck, left arm, and right knee. Five patients in Johnson et al.'s series reported transient focal motor or sensory symptoms, generally involving the face and upper extremities.

At initial presentation our patient had ophthalmoscopic and perimetric evidence of prior arterial occlusions. It is well-known that if a branch arterial occlusion occurs remote from the fovea it may go unnoticed by the patient.[16] It is not unreasonable to expect that some patients that develop idiopathic recurrent branch retinal artery occlusions will suffer one or more asymptomatic episodes before presenting clinically with a symptomatic occlusion. This had lead Gass et al. to suggest that biomicroscopic and angiographic evidence of this syndrome be sought in patients that present with unexplained visual field loss.[1]

The incidence of ocular neovascularization in patients with idiopathic recurrent branch retinal arterial occlusion has been estimated at 25% (8/32 eyes).[2] This is similar to the incidence after acute central retinal artery obstruction, which has been reported by one recent prospective study as 21.2%.[20] In patients with acute central retinal artery obstruction, secondary neovascularization may appear as early as 12 days to as late as 15 weeks after the occlusion[20]; comparable data have not been published for patients with idiopathic recurrent branch retinal arterial occlusion. Of the eight eyes in Johnson et al.'s series that developed neovascularization, it involved the retina in six, the optic disc in four, and the iris in two.[2] Patients with ocular neovascularization secondary to retinal arterial occlusion should be treated with panretinal photocoagulation to minimize the risk of neovascular glaucoma, vitreous hemorrhage, and tractional retinal detachment.[3, 20] It has been recommended that patients with central retinal artery occlusion be followed closely for a minimum of 3 to 4 months after diagnosis for the development of neovascular complications.[20] This recommendation could be extended to patients with idiopathic recurrent branch retinal artery occlusion. However, given the recurrent nature of this disease, patients should be reminded at each office visit of the need to be monitored regularly irrespective of symptoms.

Idiopathic recurrent branch retinal arterial occlusion may be etiologically related to Susac's syndrome, a neuropsychiatric disorder affecting young women characterized by a triad of microangiopathy of the brain, retina, and inner ear.[21] This disease primarily occurs in women of childbearing age, but rare cases in young men have been reported.[22, 23] The encephalopathy is associated with ataxia, memory loss, and personality changes. Usually, but not always, symptoms resulting from the encephalopathy precede the onset of visual and hearing disturbances. Notis et al.[24] described an unusual case of Susac's syndrome in which the presenting sign was a branch retinal arterial occlusion. Theirs is only the second such reported case. Patients without clinical evidence of encephalopathy at the time of their initial symptomatic arterial occlusion appear to have little risk of progressing to Susac's syndrome at some later date. Our patient never developed any evidence of encephalopathy. Magnetic resonance imaging will reveal numerous small infarcts in the gray and white matter that may be misinterpreted as being due to multiple sclerosis. Infarctions in the cochlea produce a bilateral hearing loss that is greatest for low to moderate frequencies. Tinnitus is common and may be the presenting symptom.

Jerk nystagmus is an occasional finding. The branch retinal artery occlusions associated with Susac's syndrome are indistinguishable from those associated with idiopathic recurrent branch retinal arterial occlusion. Susac's syndrome is self-limiting, with a spontaneous remission after approximately 2 years. Most patients, however, will suffer a permanent mild to moderate dementia. Permanent vision and hearing impairment is also common, but frank blindness and deafness are rare. The etiology is unknown. Patients have been treated with antiplatelet (aspirin), calcium antagonist (nifedipine), and immunosuppressive (steroid and cyclophosphamide) medications, but it is unclear if any treatment is effective in limiting the progression of the disorder. It has been suggested that idiopathic recurrent retinal arterial occlusion is a forme fruste of Susac's syndrome.[1, 21]

CONCLUSIONS

Any patient who develops branch retinal arterial occlusion should have a thorough medical evaluation to exclude embolic and collagen vascular disease. Investigations may include carotid Doppler ultrasonography, echocardiography, complete blood count with platelets, prothrombin time, partial thromboplastin time, lupus anticoagulant, erythrocyte sedimentation rate, antinuclear antibodies, rapid plasma reagin, rheumatoid factor, fasting blood glucose, and lipid profile. Additional coagulation tests may be indicated when there is a personal or family history of thromboembolic disease. More common causes of retinal arterial obstruction in the young include emboli from cardiac valvular disease, atrial myxoma, or intravenous drug abuse; thrombosis secondary to pregnancy, oral contraceptive use, or coagulopathy; arteritis due to systemic lupus erythematosis; arterial spasm secondary to migraine, drug abuse, or trauma; vascular compromise from orbital, optic nerve, or retinal disease; and traumatic severance of the central retinal artery. Idiopathic recurrent branch retinal arterial occlusion becomes the diagnosis of exclusion if such evaluations fail to find evidence of disease, particularly if these patients show ophthalmoscopic and angiographic signs of segmental arteriolitis.[1] The long-term visual and systemic prognosis is good.[2] However, these patients require long-term follow-up for neovascular complications.[3]

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