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Systemic considerations in bilateral central retinal vein occlusion

Dawn N. Tomasini, O.D., and Bhagya Segu, O.D., M.P.H.

VA Hudson Valley Healthcare System, Optometry Service, Castle Point, New York.

KEYWORDS
Antiphospholipid antibody syndrome;
Central retinal vein occlusion;
Dysproteinemias;
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Systemic lupus erythematosus

Abstract

BACKGROUND: Central retinal vein occlusion (CRVO) is a common cause of visual impairment and can occur at any age. Nonetheless, 90% of patients with CRVO are older than 50 years, and only 10% of CRVO patients are younger than 40 years. Systemic vascular diseases, such as hypertension and diabetes, are common risk factors for the development of CRVO. However, when a patient less than 50 years of age has bilateral and simultaneous central retinal vein occlusions, a hyperviscosity syndrome or inflammatory condition is also suspected.

CASE REPORT: This article presents the case of a 40-year-old man with bilateral ischemic CRVO and the differential diagnoses considered, including systemic lupus erythematosus (SLE), antiphospholipid antibody syndrome (APAS), dysproteinemias, and hyperhomocysteinemia.

CONCLUSION: When a CRVO is observed in a young patient, more obscure underlying etiologies must be explored. As primary care providers, optometrists need to consider common (and atypical) vascular risk factors for vein occlusion to prevent further ocular morbidity and systemic complications.

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Central retinal vein occlusion (CRVO) is a common cause of visual impairment and can occur at any age.[1,2] Nonetheless, 90% of patients with CRVO are older than 50 years, and only 10% of CRVO patients are younger than 40 years.[1,3] The central retinal vein is particularly susceptible to occlusion at the site of the lamina cribrosa, where the central retinal vein and central retinal artery are in close proximity as they exit the eye. Arteriosclerosis of the adjacent central retinal artery may result in compression of the central retinal vein causing the vein to collapse, thereby impeding blood flow. Decreased blood flow results in increased pressure and can be thrombus forming, resulting in a vein occlusion. Alternatively, increased intraocular pressure can accentuate the external pressure difference as the vein exits the eye, resulting in turbulent blood flow predisposing the vein to a thrombotic event.[2]

A CRVO can be classified as nonischemic (perfused) or ischemic (nonperfused) depending on the degree of blockage. Nonischemic CRVO is characterized by less than 10 disc diameters of capillary nonperfusion with fluorescein angiography, visual acuity better than 20/400, tortuosity and engorgement of retinal vessels, mild intraretinal hemorrhages in all 4 quadrants, optic disc edema, and various degrees of macular edema. Ischemic CRVO is characterized by at least 10 disc diameters of retinal capillary nonperfusion with fluorescein angiography, visual acuity worse than 20/400, more extensive retinal hemorrhages in all 4 quadrants, sheathed arteries with gross retinal and macular edema. Visual acuity is generally worse with ischemic CRVO secondary to more extensive macular edema.[4] Furthermore, retinal hypoxia in ischemic CRVO can stimulate neovascularization of the anterior and posterior segment of the eye, often resulting in neovascular glaucoma and retinal detachment.[3]

Corresponding author: Dawn N. Tomasini, O.D., VA Hudson Valley Healthcare System, Optometry Service, Castle Point, New York 12511.
E-mail: dawn.tomasini@va.gov

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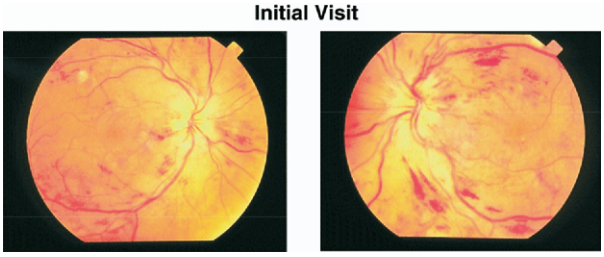

Figure 1 At the initial exam, dilated fundus examination revealed flame-shaped hemorrhages in all 4 quadrants, optic disc edema, and macular edema in both eyes.

Central retinal vein occlusions have been associated strongly with systemic disease. Therefore, when diagnosed, a thorough investigation for an underlying systemic condition must be considered. Necessary testing may include blood work, chest x-ray, carotid ultrasound scan, and even radiologic imaging. The etiology can be quite varied, but age can be helpful in determining the differential diagnosis. Patients older than 50 years usually have common systemic vascular conditions such as hypertension or diabetes that put them at risk for a vascular occlusive event. However, when a CRVO occurs in a patient less than 50 years of age or is bilateral in nature, a hyperviscosity syndrome or inflammatory condition is also suspected.[1]

Case report

A 40-year-old white man presented to the Veterans Administration (VA) optometry clinic with the complaint of decreased vision over the previous 3 weeks, which began in his right eye and progressed to his left eye. The patient's medical history included type I diabetes with insulin treatment of 10 years' duration. He had also been treated for Lyme disease 1.5 years before. His best-corrected visual acuities on presentation were 20/200 in the right eye (O.D.) and 20/400 in the left eye (O.S.). Anterior segment and ocular tensions were normal.

Dilated fundus examination found flame-shaped hemorrhages in all 4 quadrants, optic disc edema, and severe macular edema in both eyes (*see* Figure 1). Based on the examination findings, the patient had bilateral CRVO with macular edema diagnosed. Results of our initial serologic testing confirmed that the patient had poorly controlled diabetes. His hemoglobin A1C, blood urea nitrogen, and serum creatinine levels were all elevated. Increased levels of liver enzymes also indicated a fatty liver likely caused by uncontrolled diabetes. Erythrocyte sedimentation rate (ESR) was also elevated, and his antinuclear antibody (ANA) test result was positive, indicating a possible autoimmune disorder. Results of further testing for anti–double stranded DNA were positive, which strongly supported a diagnosis of systemic lupus erythematosus. His lipid profile confirmed uncontrolled hyperlipidemia with elevated levels of total cholesterol, triglycerides, and low-density lipoproteins (LDL). The complete blood cell count with differential had minor abnormalities including a low red blood cell count possibly indicative of mild anemia. Rapid plasma reagin (RPR), angiotensin-converting enzyme (ACE), and lyme titer were all negative (*see* Table 1). In addition, carotid duplex ultrasound results were normal. The patient was referred for a primary care and hematology consult and scheduled to return in 2 weeks.

By the 2-week follow-up the patient had seen a primary care practitioner (PCP) at the VA hospital and had hypertension diagnosed, for which he was taking lisinopril and furosemide. In addition, the patient was started on atorvastatin for hyperlipidemia. The PCP maintained a sliding scale schedule for the diabetes. The ocular examination findings remained unchanged, and similar retinal findings were reported with the exception of additional cotton-wool spots, more apparent in the left eye than the right eye.

Despite several attempts to contact the patient and the patient's next of kin, the patient was lost to follow-up. The patient returned 3 months later after having had macular laser treatment to the left eye with a private ophthalmologist. Visual acuities were now 20/200+ O.D. and finger counting at 3 feet O.S. Retinal examination showed neovascularization of the disc and persistent macular edema in both eyes, with focal laser scars evident in the left eye (*see* Figure 2). The patient was referred for pan retinal photocoagulation. After treatment, the patient moved to another state to be closer to his family.

The patient returned 4 years later to explore options for visual enhancement. He presented with unaided visual acuities of no light perception in both eyes (OU). However, the patient denied any ocular pain or discomfort and reported good mobility with a walking cane. He reported that pan

Table 1 Initial blood tests

Test	Value	Reference range
Hemoglobin A1c	8.6	4.6-6.5
Urea nitrogen	26 mg/dL	7-18
Serum creatinine	1.0 mg/dL	0.6-1.3
ESR	52	0-10
RPR	Nonreactive	Nonreactive
ACE	53	8-52
Total cholesterol	284	140-200
Triglycerides	180	35-160
HDL	34	29-71
LDL	214	0-130
CHL/HDL	8	3.3-11
ANA	Positive	Negative
LYME	Negative	Negative
DNA AB	Positive	Negative
P-time	13.4	11.5-14.2
INR	1.08	0.8-1.2
PTT	31.8	23.3-36.4

LDL = low-density lipoprotein; HDL = high-density lipoprotein; CHL = combined hyperlipidemia; INR = international normalized ratio; PTT = partial thromboplastin time.

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