Optic Disc Traction Syndrome Associated With Central Retinal Vein Occlusion

Shimon Rumelt, MD; Muge Karatas, MD; Joseph Pikkel, MD; Michael Majlin, MD; Avinoam Ophir, MD

Objective: To describe the association between ischemic central retinal vein occlusion (CRVO) and the development of optic disc (vitreopapillary) traction, as verified by optical coherence tomography (OCT).

Methods: In a prospective, noncomparative, observational patient series, 3 women aged 51 to 64 years developed an ischemic type of CRVO. One patient underwent cannulation of the central retinal vein with injection of a tissue plasminogen activator. In each eye, the contour of the optic nerve head could not be accurately detected because of overlying fibrous tissue. Each patient underwent OCT examination 6 to 10 months (average, 8.3 months) after the occlusive event.

Results: Optic disc traction was found by OCT in the 3 patients. In each, the disc was elevated, associated with either incomplete posterior vitreous detachment (2 eyes) or vitreopapillary fibrous membrane (1 eye). Secondary peripapillary retinal traction and macular or retinal detachment developed (“optic disc traction syndrome”). These findings were less marked, or not evident, on both clinical examination and ocular ultrasonography.

Conclusions: Optic disc traction and secondary localized retinal detachment can develop after ischemic CRVO and may contribute to the poor visual acuity. This syndrome should merit special attention before surgery for CRVO is planned. The use of OCT was helpful to diagnose this syndrome.

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fibrous tissue in front of the ONH that obscured its contour for precise evaluation. In each patient, the OCT examination disclosed an inward traction and elevation of the ONH toward the vitreous compartment by a vitreal membrane. After OCT assessment of vitreopapillary traction, the traction became evident clinically in 1 eye (patient 2) and remained suggestive (because of the thick fibrous prepapillary tissue) in the remaining 2 eyes.

**CASE 1**

A 57-year-old woman was referred to our outpatient clinic with a history of CRVO in her left eye of 4 months’ duration. Her medical history showed systemic hypertension and type 2 diabetes mellitus for 18 months. Results of laboratory evaluation, including complete blood cell count with differential count, serum electrolyte and blood homocysteine levels, liver and kidney functions, and lipid profile, were normal except for hyperglycemia and hypertriglyc eridemia. On ocular examination, the patient’s best-corrected visual acuity (BCVA) was 20/20 in the right eye and counting fingers from 0.6 m in the left. The intraocular pressure was 13 mm Hg in the right eye and 12 mm Hg in the left. The left retina showed blot intraretinal hemorrhages in the paracentral, midperipheral, and peripheral retina in all quadrants, as well as peripapillary and preretinal fibrosis (Figure 2A); the right retina appeared normal. Fluorescein angiography demonstrated widespread areas of retinal nonperfusion in the midperiphery and periphery, characteristic of ischemic CRVO. The contour of the ONH could not be accurately evaluated, and ultrasonography showed a small, prepapillary echo.

Two months after initial examination, the patient underwent OCT examination that showed dense vitreopapillary membrane in the left eye, causing nasal traction of the ONH into the vitreous cavity and secondary traction of the peripapillary retina (Figure 2B). A peripapillary hyporeflection space was noted around the retracted disc between the retina and retinal pigment epithelium, corresponding to a serous retinal detachment, which was apparent mainly temporal to the disc. The OCT showed the detached macula to be atrophic, associated with marked cystoid spaces (Figure 2C).

**CASE 2**

A 51-year-old woman had an acute decrease in her visual acuity to counting fingers from 1.2 m in the right eye. Ocular examination of that eye showed engorged peri-

papillary retinal vessels and multiple intraretinal hemorrhages characteristic of CRVO. The anterior segment was normal. The BCVA of the left eye was 20/20, and the anterior and posterior segments were normal.

The patient’s medical history showed systemic hypertension and the intake of hormonal replacement. Fluorescein angiography demonstrated ischemic-type CRVO with widespread areas of capillary nonperfusion. The patient underwent pars plana vitrectomy and cannulation of the central retinal vein with injection of tissue plasminogen activator (tPA) elsewhere, 5 months after the occlusive event. After an uneventful operation, BCVA remained unchanged.

During a 5-month period, a fibrous membrane appeared between the optic disc and the retinal periphery.
At that time, the patient was referred to our outpatient clinic for evaluation. Ultrasonography of the right eye demonstrated a thick membrane in front of the disc with probable adjacent serous retinal detachment; other echoes, corresponding to a detached posterior hyaloid, were evident that extended from the thick membrane to the retinal periphery (Figure 3A). The OCT demonstrated a double-stranded membrane retracting the ONH at its center into the vitreous cavity. A peripapillary serous retinal detachment was noted that extended temporally under the macula (Figure 3B). In addition, large cystoid spaces and advanced thinning of the retina at the papillomacular bundle site were apparent.

CASE 3

A 64-year-old woman complained of an abrupt decrease in vision in her right eye. She had had systemic hypertension and type 2 diabetes mellitus for 14 years and had mild background diabetic retinopathy in each eye. On ocular examination, her BCVA was 20/60 in the right eye and 20/30 in the left eye. The intraocular pressures were 25 and 26 mm Hg, respectively. The anterior segments were normal except for a mild nuclear cataract in each eye. Funduscop>
Optic disc traction developed in 3 women with ischemic CRVO, resulting in a secondary localized retinal detachment ("optic disc traction syndrome"). The serous detachment involved the macula in 2 eyes and the nasal retina in the third eye. The syndrome was detected 6 and 9 months after the CRVO event in 2 previously nonoperated-on eyes and 10 months after the occlusive event (3 months after surgery) in an eye that had undergone pars plana vitrectomy and cannulation of the central retinal vein by tPA. These findings suggest, at least in the 2 non–operated-on eyes, that vitreopapillary traction and its sequelae can be part of the natural course of ischemic CRVO and that this abnormality may appear, or persist, after pars plana vitrectomy and tPA administration for ischemic CRVO.

Optic disc traction showed 2 forms on OCT examination. The first was an eccentric traction on the disc margin (patients 1 and 3), causing nasal or temporal pulling of the disc, respectively, resulting in a detached retina on the opposite side, temporal or nasal to the disc, respectively. The second form appeared as a central, symmetric traction (patient 2), resulting in peripapillary temporal and nasal retinal detachment. On the basis of the ultrasound findings in patients 2 and 3, incomplete posterior vitreous detachment was evident in each. The tissue adjacent to the ONH looked thickened in each, resulting in an OCT appearance of a thick, somewhat elongated membrane. The latter could be due to encroachment and growth of a peripapillary fibrous tissue that developed secondary to the hemorrhagic CRVO insult, over the posterior hyaloid. The adherence of the fibrous tissue overlying the posterior hyaloid to the optic disc could result in optic disc traction on either detachment of the posterior vitreous from the ONH or contraction of that fibrous tissue over the incompletely detached hyaloid. The OCT appearance of these traction membranes more peripherally could not be assessed because of limitations of the device, ie, its use is practical in the current form only for the posterior pole of the eye. The findings may indicate that meticulous removal of the posterior hyaloid should be considered in CRVO surgery.

In this regard, Uchino et al14 conducted a study of 209 healthy eyes with the use of the OCT and found that the human eye may have incomplete or partial posterior vitreous detachment, beginning as early as the fourth decade of life. It initially occurs at the perifovea, slowly extending its range for years, while vitreopapillary adhesion remains the last to separate. Pathologic detachment of the posterior vitreous probably plays a role as the underlying cause of other vitreoretinal disorders, such as idiopathic macular hole8 and vitreomacular traction syndrome.9

The OCT had several advantages in the demonstration of optic disc traction. It allowed definite diagnosis of vitreopapillary traction, serous retinal or macular detachment, and its association with disc elevation, as well as evaluating thickness and configuration of the atrophic macula. These findings were more efficacious and/or definitive than both the clinical examination and ocular ultrasonography. Such diagnoses should affect surgical planning and its prognosis (see the following paragraphs). In patients 1 and 2, macular detachment was detected. A relatively shallow serous retinal detachment nasal to the disc was apparent in patient 3. These findings indicate the importance of early diagnosis of optic disc traction syndrome. Further delay in surgical removal of the contracting membranes could cause further irreversible sequelae because of the increased period of elevation of the detached retina or macula and secondary atrophy of that tissue. In addition, it could possibly cause further elevation of the disc as well as the area and height of the surrounding detached retina or macula and its sequelae. Furthermore, long-standing optic disc traction might result in optic atrophy either by impeding the axoplasmic flow or by changing the laminar flow through the peripapillary blood vessels.3 The latter may decrease the blood flow, create turbulence, and increase the formation of thrombi. A sole traction of the ONH has been described in diabetic retinopathy to be the cause of a further decrease in visual acuity, visual field scotomas, and decreased foveal visual-evoked potentials.2

Several approaches have been proposed to treat ischemic CRVO, including posterior ring section10,11 and tPA therapy, either systemic12 or intravitreal.13 Weiss and Byone14 treated 28 eyes with CRVO with retinal vein tPA injection; 15 (54%) recovered at least 3 lines of visual acuity postoperatively. Opremcak et al15 described radial optic neurotomy, which included pars plana vitrectomy, for the treatment of ischemic CRVO. It is possible that preoperative diagnosis of optic disc traction syndrome and more precise evaluation of the macular status, eg, by OCT, could have provided surgical considerations in the studies mentioned and improved the surgical outcome of these eyes. Lam and Blumenkranz16 recently reported a case of CRVO treated by subretinal peripapillary tPA injection in association with vitreopapillary and epipapillary membrane dissection.

Furthermore, optic disc traction syndrome may be an important prognostic sign for each of the aforementioned surgical procedures, especially if it is longstanding or if macular detachment is involved. Thinning of the perifoveal retina, due to either the occlusive event and/or the optic disc traction, is usually not accurately detected by standard examinations, especially when the retina is markedly hemorrhagic; the degree of thinning may indicate a poorer prognosis for visual improvement postoperatively. Preoperative misdiagnosis of that syndrome, with various levels of macular atrophy, could explain some unexpected large differences in visual outcome after surgery in some of the studies mentioned in the previous paragraphs.

Vitreoretinal traction can be treated by pars plana vitrectomy with removal of the proliferative membranes and the posterior cortical vitreous, and therefore its diagnosis is crucial.1,17 However, surgery for vitreopapillary traction should probably be performed even more cautiously, since removal of the peripapillary membranes or posterior vitreous, which are adherent to the disc, may be accompanied by inadvertent excision of axons that may further compromise visual acuity and visual field.17
Diabetic retinopathy and CRVO are among the retinal vasculopathies that may result in neovascularization and proliferation of a fibrovascular or fibrocellular membrane over the disc. In addition to vitreopapillary traction, other types of traction may appear in the aforementioned vasculopathies.\(^1,2,18-20\) Retinal traction is probably more common than optic disc traction in diabetic eyes, since the leaking vessels are more often retinal than at the disc site. In contrast, optic disc traction may be more apparent in ischemic CRVO than in diabetic cases, since the major leaking abnormality, usually markedly hemorrhagic, occurs at the disc site.

**CONCLUSIONS**

Optic disc traction syndrome may develop after ischemic CRVO due to traction either from detached posterior hyaloid or from vitreopapillary membrane and may be a part of the natural course of the disease. It may contribute to the poor visual outcome, in addition to the underlying abnormality. Optical coherence tomography is a powerful device to demonstrate this abnormality and may be advantageous when planning surgical intervention and evaluating prognosis.

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Corresponding author and reprints: Avinoam Ophir, MD, Department of Ophthalmology, Hillel-Yaffe Medical Center, Hadera, Israel (e-mail: ophthalmology@hillel-yaffe.health.gov.il).

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