Optometry (2007) 78, 122-128

Optic atrophy after sildenafil use

Joseph W. Sowka, O.D., Maryke N. Neiberg, O.D., and Lori A. Vollmer, O.D.

Nova Southeastern University, College of Optometry, Ft. Lauderdale, Florida.

KEYWORDS
Optic atrophy;
Optic neuropathy;
Nonarteritic anterior ischemic optic neuropathy;
Sildenafil;
Erectile-enhancing medications;
Phosphodiesterase inhibitor

Abstract

BACKGROUND: It has been well-reported that phosphodiesterase-5 (PDE-5) inhibitors, originally investigated for their effect on smooth muscles and now used widely in treatment of erectile dysfunction, can cause mild transient visual disturbances because of their action on inhibiting enzymes involved in retinal transduction. Recently, these medications have been associated with the development of non-arteritic anterior ischemic optic neuropathy (NAAION) with attendant vision loss.

CASE REPORT: An older male patient, previously examined and ocularly healthy, presented asymptomatically with an occult optic neuropathy, not characteristic of NAAION. Neuroimaging and serology failed to reveal any other underlying cause. The patient did, however, report the use of sildenafil during the interval between his previously normal examination and the observation of his optic neuropathy.

CONCLUSIONS: This case details the development of an optic neuropathy with atrophy seemingly associated with the use of sildenafil, although no cause and effect could be conclusively found. This may indicate that medications used in the treatment of erectile dysfunction may be responsible for optic neuropathies other than NAAION.

Optometry 2007;78:122-128

It has been well-reported that phosphodiesterase-5 (PDE-5) inhibitors, originally investigated for their effect on smooth muscles, now used widely as treatment for erectile dysfunction, can cause mild transient visual disturbances because of their action on inhibiting enzymes involved in retinal transduction.[1,2] In clinical trials, abnormal vision was reported in 3% of men taking sildenafil (Viagra; Pfizer, New York, New York). Several reports have recently implicated these medications (PDE-5) in the development of more serious complications such as non-arteritic anterior ischemic optic neuropathy (NAAION) with attendant vision loss.[3-6] Considering that these medications are widely used, it follows that other complications may be reported. It is conceivable that PDE-5 inhibitors are capable of causing optic neuropathies other than NAAION.

Corresponding author: Joseph Sowka, O.D., Nova Southeastern College of Optometry, 3200 S. University Drive, Fort Lauderdale, Florida 33328.

E-mail: jsowka@nova.edu

Case

A 69-year-old white man presented for a comprehensive eye examination with complaints of blur at near with his current glasses. His last examination was 13 months before. He had no complaints with his distance vision and had not noted any abrupt visual changes at either distance or near. He reported no history of ocular disease, injury, or surgery. His family ocular history was significant for glaucoma in his mother and exudative macular degeneration in his father. There was no significant family medical history. The patient was reportedly healthy and not using any medication. Blood pressure was 130/80 mmHg, measured in the right arm and sitting. His medical history was significant only for surgical removal of a thyroglossal duct cyst many years ago. He

1529-1839/07/$ -see front matter
doi:10.1016/j.optm.2007.01.005

reported moderate social consumption of alcohol, but denied tobacco use, having quit smoking 40 years previously. The patient's best-corrected visual acuities were 20/20^{-} in the right eye (O.D.) and 20/20 in the left eye (O.S.) with a low hyperopic astigmatic prescription. His reading difficulty was resolved with an appropriate increase in his bifocal prescription. Confrontation visual fields were full in each eye. Extra ocular muscle testing showed no restrictions. Pupils were round and reactive to light. There was no anisocoria; however, there was a marked (grade 3) relative afferent pupil defect (RAPD) O.S. On color vision testing, the patient was able to correctly identify 14 of 14 Ishihara's pseudo-isochromatic color plates in each eye. He reported minimal color desaturation O.S. but reported approximately a 50% decreased brightness appreciation in his left eye.

Biomicroscopic examination found early nuclear sclerotic changes in the lens of each eye, more significant O.D. than O.S., adequately explaining the slightly reduced acuity O.D. There were rare peripupillary transillumination defects of each iris and rare pigment granules on the corneal endothelium of each eye. There were no midperipheral iris transillumination defects in either eye. There was no clinically apparent pseudoexfoliative material on the crystalline lens of either eye. Intraocular pressure by Goldmann applanation was 20 mmHg O.D. and 23 mmHg O.S. at 3:55 PM. Central corneal thickness was 527 μm O.D. and 541 μm O.S. Gonioscopic evaluation of the right eye found posterior trabecular meshwork for 180° and ciliary body for the remainder 180°. The left anterior chamber angle showed scleral spur for 180° and ciliary body for the remainder 180°. There was flat iris approach and light pigmentation within the trabecular meshwork of each eye. There was no evidence of pigment accumulation on Schwalbe's Line indicative of a Sampaolesi's Line in either eye.

Dilated fundus examination found a cup-to-disc ratio of 0.4/0.4 O.D. and 0.5/0.5 O.S (*see* Figure 1). The left disc was slightly larger than the right, likely accounting for some of the cup-to-disc asymmetry. There was no peripapillary atrophy, hemorrhages, or focal glaucomatous defects of either disc. The right disc neuroretinal rim was pink and perfused. The left disc neuroretinal rim was pale and atrophic. The pallor was sectoral, from 12:00 o'clock to 3:00 o'clock. Neither optic nerve manifested a characteristic crowded or "disc at risk" appearance typically associated with NAAION. The remainder of the fundus examination was unremarkable in both eyes (OU).

Automated threshold visual field analysis of the right eye showed isolated depressions in both hemifields with a small, shallow paracentral scotoma. There were deeper depressions in both hemifields of the left eye. The more significant field loss was in the inferior hemifield, with greater concentration on the pattern deviation in the inferior–temporal quadrant; however, it would not be classified as a relative inferior altitudinal defect or absolute nasal defect commonly seen in NAAION (*see* Figure 2). Scanning laser polarimetry confirmed nerve fiber layer loss primarily superiorly O.S., corresponding to the visual field defect. There were also

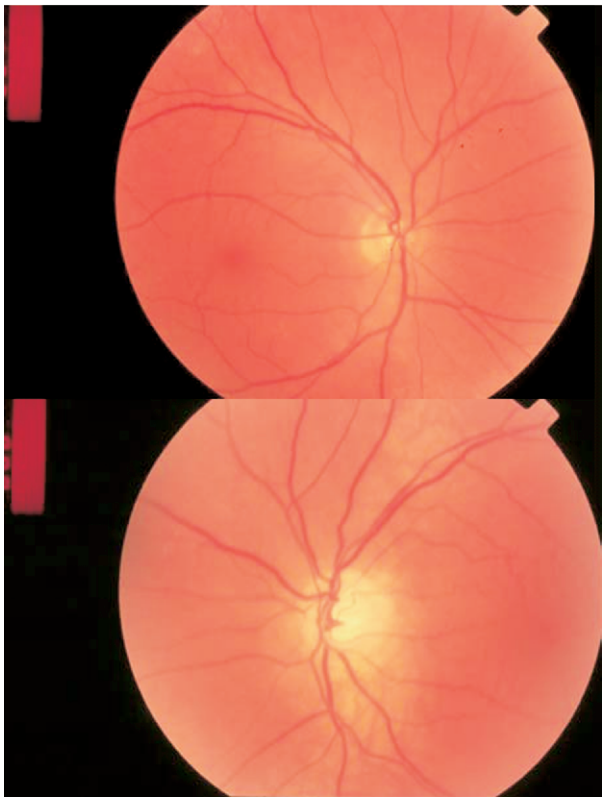

Figure 1 Sectorial disc pallor O.S.

departures from the normative database in the inferior nasal aspect as well, matching the superior hemifield defect; however, there was no appreciable disc pallor corresponding to this area (*see* Figure 3). The asymmetry in the abnormality of the visual field and nerve fiber layer between the 2 eyes, however, was not significant enough to account for an RAPD O.S. from glaucoma (especially one so marked as seen here). Because there was the subjective appearance of sectoral disc pallor, yet some contradictory findings (RAPD, light desaturation, but no loss of acuity or color perception), a visually evoked potential (VEP) was ordered to verify involvement of the optic nerve. A contrast reversing pattern evoked potential was performed and found an increase in the latency of the P100 value (P, positive; 100, latency around 100 milliseconds), as well as a reduction of the amplitude in the left eye compared with the right eye (*see* Figure 4). The difference in the latency of the P100 peak between the 2 eyes of 10.8 ms was clinically significant. The latency period (or peak time) is the time between stimulus onset and the most significant measurable potential. Delays in this value have been reported to occur in persons over the age of 60 years; however, this does not explain the asymmetry in patient response. Diseases of the optic nerve head lead to delays of P100 latency, and in the absence of other explainable etiology, the VEP confirms asymmetry between the eyes and pathologic involvement of the optic nerve of the left eye.

The patient had optic atrophy O.S. of unknown origin. Review of the past several examinations (as recently as 13