Case Report

Duloxetine-induced Optic Neuropathy: A Rare Case Report

Priti Yadav*, Neelima Mehrotra, Kunwar Gaurav Singh, Manu Saini

Department of Ophthalmology, Shri Ram Murti Smarak Institute of Medical Sciences, Bareilly, Uttar Pradesh, India.

Abstract

Cataract is a major cause of blindness worldwide, with a higher prevalence in developing countries, especially in India. The onset of cataracts at a younger age, known as presenile cataract, is of concern due to its multifactorial etiology, including genetic, environmental, and behavioral factors. The present study aimed to investigate the factors responsible for presenile cataracts and their visual outcomes in younger adults undergoing cataract surgery.

Keywords: Duloxetine-induced, Optic neuropathy, Case Report.

Introduction

Duloxetine is a serotonin and norepinephrine reuptake inhibitor (SNRI). It is FDA-approved for use in the treatment of generalized anxiety disorder, major depressive disorder, fibromyalgia, and chronic musculoskeletal pain. Off-label use includes chemotherapy-induced peripheral neuropathy and stress urinary incontinence. In addition to its general SNRI properties, duloxetine also increases dopamine levels, specifically in the prefrontal cortex.[1]

The adverse effects of duloxetine are similar to traditional SSRIs. The most common adverse effect of duloxetine is nausea, which is generally associated with discontinuation. Despite its regular use, duloxetine has been linked to a small number of ocular adverse events, including cycloplegia, dry eye cataract formation, and angle closure glaucoma.[2]

Case Presentation

A 21-year-old, male patient, a student by profession, came to ophthalmology opd with the presenting complaints of diminution of vision from both eyes for 1 month, which was gradually progressive and painless. He denied recent eye trauma or infection. The patient reported that he had a history of burning sensation in bilateral feet and hands for 6 months, for which he took pregalin D 75/20 mg (Pregabalin and duloxetine) twice daily. There was no significant family history.

On examination, the Snellen chart test for best corrected visual acuity was 5/60 OD (oculus dexter) and 3/60 OS (oculus sinister), color vision was abnormal by Ishihara chart, and intraocular pressure was 16 mmHg OU.

On slilt-lamp examination, both eyelids were normal in position. The conjunctiva was white and quiet in both eyes. Pupils were regular, rounded and reactive, with clear cornea, clear lens, deep and quiet anterior chamber, and normal iris without rubeosis in both eyes (Figure 1).

Posterior segment examination of both eyes revealed a clear vitreous, normal optic disc, normal macula contour without edema, and normal appearance of the peripheral retina without tears, breaks, holes, or mass (Figure 2).

Visual field test results demonstrated some constriction in both eyes.

His ocular coherence tomography (OCT) picture showed that retinal nerve fiber layer thickness was 108 microns in OD and 113 microns in OS [Figure 3].

The patient was thoroughly examined for diminution of vision but no other causative factor could be associated except the treatment with duloxetine and pregabalin. Duloxetine was stopped immediately and asked the patient to review it after 1-week. On 1st follow-up visit, the patient reported partial recovery of his best corrected visual acuity of OD 6/36 and 6/60 OS. The patient was switched to pregabalin only as advised by his neurophysician.

Address for correspondence: Priti Yadav,
Department of Ophthalmology, Shri Ram Murti Smarak Institute of Medical Sciences, Bareilly, Uttar Pradesh, India
E-mail: preetiyadav0703@gmail.com

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His blood investigations were within normal limits, which included fasting blood sugar, complete blood count with ESR, C-reactive protein, liver function test, renal function test, and antinuclear antibodies (ANA). MRI brain and orbit scan were normal. The patient was also advised for a nerve conduction test.

On 2nd follow-up visit after 2 weeks his best corrected visual acuity of OU was 6/12, and on 3rd follow-up after the 4th week, the best corrected visual acuity of OU was 6/9.

Discussion

In this case, we highlight a rare case of duloxetine-induced diminution of vision. In our case, the patient had no history of any eye diseases before the duloxetine-pregabalin administration. He had no complaints about his vision. Normal neurological examination and the gradual recovery after the cessation of duloxetine supported the idea that the diminution of vision was an adverse reaction induced by duloxetine. The exact pathophysiology of duloxetine-induced optic neuropathy is unknown. However, Costagliola *et al.* described a mechanism for vasospasm in the optic nerve, postulating that increased plasma serotonin levels may be a factor in the development of optic nerve perfusion disorders.[3] In individuals with atherosclerotica, the susceptibility becomes higher due to serotonin-enhancing platelet aggregation on the atheromata of ocular arteries.[4]

Eye pain, blurring of vision and cycloplegia are possible visual side effects of duloxetine. Rare side effects of SNRIs have been reported: one of a forty-four-year-old female who experienced unilateral vision loss due to retrobulbar optic neuritis, and the other of an 81 year/old female who developed bilateral acute angle closure two days after also commencing duloxetine.[5,6]

Conclusion

Recovery of visual acuity is rare following nonvasculitic ischemic optic neuropathy, and therefore, the question of toxic optic neuropathy is open. The implication of presenting this case is to create awareness amongst doctors prescribing duloxetine to counsel patients and to be vigilant regarding the possibility of the development of ocular side effects.

References

1. Cipriani A, Koesters M, Furukawa TA, Nosè M, Purgato M, Omori IM, Trespidi C, Barbui C. Duloxetine versus other anti-depressive agents for depression. *Cochrane Database Syst Rev.* 2012 Oct 17;10:CD006533.
2. Chen HY, Lin CL, Lai SW, et al. Association of selective serotonin reuptake inhibitor use and acute angle-closure glaucoma. *J Clin Psychiatry.* 2016;77:e692–696.
3. Costagliola C, Parmeggiani F, Semeraro F, Sebastiani A. Selective serotonin reuptake inhibitors: a review of its effects on intraocular pressure. *Curr Neuropharmacol.* 2008;6:293–310.
4. Hayreh SS. Retinal and optic nerve head ischemic disorders and atherosclerosis: role of serotonin. *Prog Retin Eye Res.* 1999;18(2):191–221.
5. Bicer T, Kosker M, Celikay O, et al. A case of retrobulbar optic neuritis caused by duloxetine. *Cutan Ocul Toxicol.* 2016;35:251–253.
6. Shifera AS, Leoncavallo A, Sherwood M. Probable association of an attack of bilateral acute angle-closure glaucoma with duloxetine. *Ann Pharmacother.* 2014;48:936–939.