Solar retinopathy related to antidepressant use in a patient with major depressive disorder: a case report

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This case report is a unique case of solar retinopathy following antidepressant-induced mydriasis and highlights the need for comprehensive ophthalmic evaluation in patients treated with medications having mydriatic effects. A 49-year-old female patient who had received long-term antidepressant therapy presented with bilateral visual impairment after prolonged sun exposure. Fundoscopy confirmed solar retinopathy, which was attributed to drug-induced mydriasis. Medication adjustments and sun protection strategies led to full visual recovery, underscoring the importance of interdisciplinary awareness. This case emphasizes the need for routine ophthalmic evaluation of patients prescribed antidepressants with reported ocular side effects.

Keywords: Antidepressive agents; Depression; Mydriasis; Solar retinopathy

Introduction

Depression is a common mental disorder that affects millions of individuals worldwide and often requires pharmacological treatment to manage symptoms and improve quality of life. Antidepressants are the cornerstone of therapy for many patients and a range of medications alleviate depressive symptoms by acting on neurotransmitter systems. One of the widely recognized side effects of certain antidepressant classes, such as selective serotonin reuptake inhibitors (SSRIs) and tricyclic antidepressants (TCAs), is mydriasis, a condition characterized by pupil dilation [1].

Mydriasis can increase the risk of ocular complications, including solar retinopathy, a photic retinal injury resulting from direct exposure to sunlight. Solar retinopathy is traditionally associated with eclipse viewing or sun-gazing practices [2-4]. If left untreated, this condition can lead to permanent visual impairment. Although mydriasis is often considered a benign side effect of drug therapy, its potential to exacerbate retinal vulnerability to solar damage is not commonly recognized.

This study describes a unique case in which mydriasis induced by long-term antidepressant use was identified as a contributing factor to solar retinopathy. This case emphasizes the significance of prompt and comprehensive ophthalmic assessment in patients presenting with vision complaints who are under treatment with medications capable of inducing mydriasis. The interplay between psychiatric medications and ocular health presents a complex challenge, necessitating heightened awareness among mental health professionals and patients. In this context, I explored the mechanisms by which antidepressant-induced mydriasis may increase retinal susceptibility to solar damage, discussed the diagnostic tools used to identify the early stages of solar retinopathy, and reviewed the management strategies employed to mitigate visual loss and promote retinal recovery.

Integrating my findings, I aim to provide a broader understand-
ing of the intertwined relationship between mental health management and ocular safety and propose guidelines for clinicians to follow when prescribing medications with known ocular side effects.

Case

Ethical statements: This study was approved by the Institutional Review Board (IRB) of Yeungnam University Hospital (IRB No. YUMC 2024-02-015). Written informed consent from the patient was waived by the IRB.

A 49-year-old female patient with a medical history of major depressive disorder for approximately 10 years was referred for retinal evaluation after experiencing bilateral blurred vision. Three days before her presentation, she spent an extended period of approximately 3 hours reading the Bible in the sunlit yard of a Catholic church. For the last 6 months, the patient had been undergoing treatment with a pharmacological regimen for her depressive disorder, which included escitalopram (20 mg), nortriptyline (30 mg), clonazepam (1.5 mg), flunitrazepam (1 mg), trazodone (50 mg), and lorazepam (3 mg) daily. The patient’s medical history was unremarkable other than depression, and her pharmaceutical regimen was exclusively psychotropic medications. Moreover, she had no history of ophthalmic diseases such as glaucoma. On examination, her refractive error was mild +0.50 diopters in both eyes and essentially within the normal range. The best corrected visual acuity (BCVA) was reduced to 20/50 in both eyes. Pupillary examination revealed mid-dilated pupils with a diameter of 6.0 mm in each eye, exceeding the typical average of 3 mm to 4 mm. Intraocular pressures were within normal limits, and anterior segment examination was unremarkable. However, upon fundoscopic examination, bilateral foveal lesions characterized as small, round, whitish-yellow spots were detected (Fig. 1) in conjunction with paracentral scotoma identified by automated visual field testing. The distinctive features observed during the fundoscopic examination unequivocally established the presence of solar retinopathy. Using spectral-domain optical coherence tomography (SD-OCT), I observed distinctive retinal impairments characterized by rod-shaped hyperreflective lesions involving all retinal layers at the foveal region in both eyes, along with a subtle decrease in the reflective profile of the retinal pigment epithelium at the fovea in the left eye (Fig. 2). The vitreoretinal interfaces remained intact in both eyes. Furthermore, the central foveal thickness was within the normal spectrum, measuring 262 μm in the right eye and 271 μm in the left eye.

To mitigate the risk of additional retinal injury, I implemented interventions that focused on sun protection and patient education. The patient was advised against sun gazing and instructed to use protective measures outdoors, including wearing sunglasses and a hat. Regular follow-up appointments were scheduled to monitor the patient’s condition closely.

Considering the patient’s ophthalmologic diagnosis, the psychiatric medication regimen was carefully adjusted. The escitalopram dose was halved from 20 mg to 10 mg, and nortriptyline was discontinued. To preemptively address any potential resurgence of depressive symptoms, the patient was monitored more frequently. Additionally, a psychotherapeutic strategy was employed to pro-

Fig. 1. The color fundus photography of the (A) right eye and (B) left eye shows a small round, whitish-yellow spot-like lesion (arrows).
vide reassurance and support while thoroughly explaining the ocular condition to minimize anxiety and foster understanding. This comprehensive approach ensured a balance between managing the patient’s mental health and safeguarding against further ocular injury.

At the 3-week follow-up, fundoscopic examination revealed complete resolution of the previously observed small, round, whitish-yellow spot-like lesions in both eyes. Bilateral blurred vision, which was the patient’s primary complaint, improved. Six months after the initial presentation, substantial improvement in BCVA was noted, with recovery to 20/25 in the right eye and 20/20 in the left eye. The paracentral scotoma, initially detected on automated visual field testing, resolved in both eyes. SD-OCT imaging demonstrated a hyperreflective external limiting membrane adjacent to the former lesion sites with focal defects at the inner and outer photoreceptor segments, which are indicative of microscopic sequelae of the retinal tissue. The patient’s depressive symptoms did not worsen and remained stable throughout the follow-up period.

Discussion

In solar retinopathy, retinal damage occurs owing to exposure to solar radiation, which can induce photomechanical, photothermal, or photochemical destruction of retinal pigment cells. This damage is primarily caused by high-energy ultraviolet (UV)-A radiation and shorter wavelengths of visible light, leading to the generation of reactive oxygen species and subsequent oxidative damage [5,6]. Previous studies have highlighted the association between solar retinopathy and various factors such as a history of sun exposure during religious activities, solar eclipses, sunbathing, and mental disturbances resulting from drug intoxication or schizophrenia [7,8].

Sun gazing has been linked to retinal damage, particularly in cases of mental disorders [2-4]. Although my patient denied direct sun gazing, it is worth noting that she engaged in activities that included reading and praying outdoors on a sunny day for an extended time, namely 3 hours. This behavior may have contributed to a heightened concentration of light energy delivered to the macula, potentially exacerbating the risk of retinal damage.

The induction of mydriasis has been associated with the administration of SSRIs. These side effects can be attributed to the diverse pharmacological actions of SSRIs, including serotonergic modulation (via 5-HT7 receptors), noradrenergic influences, and anticholinergic activity. The impact of serotonin on 5-HT7 receptors triggers adenylate cyclase activation, leading to dual effects contingent on the receptor location: (1) relaxation of the pupil sphincter, resulting in passive mydriasis, and (2) direct elevation of intraocular pressure by augmenting aqueous humor formation through effects on the iris ciliary body complex [9]. Human studies have specifically indicated that significant pupillary dilation is induced by SSRIs, including sertraline [10], paroxetine [11], fluoxetine [12], and citalopram [13].

TCAs are associated with mydriasis and photosensitivity [14]. When mydriasis occurs in an individual who does not develop tolerance to the impact of TCAs on the ocular smooth muscles, it can lead to blurred vision and/or presbyopia (disturbance of near vision). TCA-induced mydriasis typically results in nonsevere and transient visual disturbances. Cycloplegia is the paralysis of the ciliary muscles. Mydriasis and cycloplegia appear to be primarily attributable to the anticholinergic action of TCAs. Although other mechanisms cannot be excluded, the blockade of noradrenaline.
uptake and α-adrenergic receptors is important, and both conditions tend to improve with time [9].

TCAs such as imipramine, desipramine [15,16], amitriptyline [17], and clomipramine [18], have been reported to photosensitize the skin and induce skin pigmentation. While their ocular effects have not been extensively documented, it is prudent to cautiously suggest that the photosensitizing effect of TCAs creates a conducive environment for solar retinopathy in the eyes of individuals already experiencing mydriasis.

Escitalopram has been reported to induce reversible mydriasis, a phenomenon that resolves upon discontinuation of the therapy [13,19,20]. Similarly, nortriptyline is associated with mydriasis and skin photosensitivity [14,19]. The occurrence of mydriasis induced by both SSRIs and TCAs poses a risk for solar retinopathy in my patient. In clinical practice, SSRIs are often selected as first-line pharmacological treatments for depression, as was the case with my patient who was treated with the maximum therapeutic dose of escitalopram. In contrast, TCAs are not commonly used as primary treatments because of their side effects. Therapeutic doses of nortriptyline typically start at 75 mg, with a maximum dosage of approximately 150 mg. However, in this case, the patient was prescribed a much lower dose of 30 mg, utilized solely at bedtime to aid sleep. Notably, mydriasis associated with TCAs has predominantly been reported after high-dose administration. Despite the potential of both antidepressants to cause mydriasis, the primary agent implicated in this effect was escitalopram, as a reduction in its dosage alone ameliorated the patient’s symptoms.

Benzodiazepines can also induce pupil dilation because of their ability to relax sphincter pupillae and their mild anticholinergic properties [19]. However, the mydriatic effects of benzodiazepines are generally considered benign in clinical practice. The patient’s symptoms of mydriasis improved after solely reducing the dosage of escitalopram and maintaining the other medications, which suggests a significant causative role for the SSRIs. Nevertheless, it is conceivable that the benzodiazepines, clonazepam, lorazepam, and flunitrazepam, administered at low doses, may have incrementally contributed to increasing the patient’s risk.

In summary, a combination of risk factors, including extended outdoor activity with intense sun exposure, drug-induced mydriasis, mild hyperopic refractive error, and medications with photosensitizing potential, contribute to retinopathy in this patient. Despite these risks, the patient recovered fully while continuing antidepressant therapy, highlighting the absence of clear treatment guidelines for solar retinopathy. Although steroids are a therapeutic option, their use involves a risk of central serous chorioretinopathy, emphasizing that preventative strategies remain the optimal treatment modality. Managing the risk factors associated with mydriasis and reducing the susceptibility of the retina to UV exposure, in conjunction with preventive measures, may lead to patient recovery.

Preventative measures for patients who are at risk, including individuals who are young and emmetropic, or those exposed to above-average solar radiation, are crucial. Strategies, such as avoiding direct sunlight, wearing protective eyewear, and using UV-blocking windowpanes, are recommended.

If a patient experiences ocular discomfort after prolonged light exposure while taking antidepressants, it may be necessary to discuss the potential reduction or discontinuation of psychotropic medications that can induce mydriasis and increase the risk of retinal injury due to UV exposure.

Ophthalmological assessments can be performed before and throughout antidepressant treatment. Although the changes in the eye induced by these medications can be subtle and undetectable without specialized equipment, regular examinations can help identify and mitigate the risk of ocular complications.

This report provides a novel account of solar retinopathy in a patient with depression and underscores the importance of interdisciplinary awareness. By prioritizing early prevention and patient education, serious and irreversible ocular injuries can be avoided. This report reinforces the need for vigilance and proactive health care practices when psychiatric and ocular health intersect.

Article information

Conflicts of interest
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