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Olanzapine-Induced Acute Angle Closure

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Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
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Patient: Male, 59-year-old
Final Diagnosis: Acute angle closure
Symptoms: Pain • red eye
Medication: —
Clinical Procedure: —
Specialty: Ophthalmology

Objective: Unusual clinical course

Background: Drug-induced acute angle closure glaucoma is an uncommon ocular emergency that may follow the administration of certain topical and systemic medications. Acute angle closure can be triggered by various classes of drugs, including adrenergic agonists, anticholinergics, and serotonergic medications. Here, we report a rare case of drug-induced acute angle closure glaucoma secondary to olanzapine.

Case Report: A 59-year-old male patient of Arabian Peninsula descent, known to have schizophrenia, presented to our Emergency Department with a 3-day history of right ocular pain and decrease in vision. He was started recently on olanzapine 5 mg once daily by his psychiatrist 1 week prior to the onset of his symptoms. The diagnosis of drug-induced pupillary block was made based on clinical and radiological findings. The patient was started on topical and systemic IOP-lowering agents. A therapeutic Nd: YAG laser peripheral iridotomy for the right eye was performed. On follow-up, his symptoms alleviated and clinical examination showed significant improvement.

Conclusions: The reported case highlights the importance of systemic medical history in secondary acute angle closure glaucoma. Physicians from other specialties should be aware of drugs triggering pupillary block and therefore be able to educate patients about symptoms of acute angle closure glaucoma.

Keywords: Antipsychotic Agents • Glaucoma • Olanzapine

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/934432

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Background

Drug-induced acute angle closure glaucoma (ACCG) is an uncommon ocular emergency that may follow the administration of certain topical and systemic medications. Patients with AAC present with acute severe ocular pain, headache, blurry vision and seeing halos around lights, nausea, and vomiting. Known risk factors of AACG include history of AACG in the other eye, Asian ethnicity, family history, female sex, hyperopia, narrow angles, short axial length, and advanced age [1]. When the pupil dilates (like in dim light conditions, emotions, or adverse effects to medications), the contact between the lens and the iris increases, thus the angle gets shallower, which prevents the aqueous outflow to the trabecular meshwork, resulting in an acute angle closure (AAC) attack. If the AAC attack resulted in optic nerve damage or a visual field defect, the term AACG is appropriate. Generally, AAC can be triggered by various classes of drugs that affect the pupil and cause it to dilate, especially in predisposed patients. These include adrenergic agonists, anticholinergics, and serotonergic medications [2]. Physicians should be aware of their potentially sight-threatening adverse effects and inform their patients to visit the ophthalmology ER whenever they experience AAC symptoms. Here, we report a rare case of drug-induced acute angle closure glaucoma secondary to olanzapine.

Case Report

A 59-year-old Saudi male patient, known to have schizophrenia, presented to our ophthalmology emergency room (ER) reporting right ocular pain associated with decreased vision for the last 3 days. One week prior to his presentation, the patient had been started by his psychiatrist on olanzapine (5 mg) once a day. The patient denied taking any other medication. There was no history of eye trauma or family history of glaucoma. On physical examination, visual acuity was 6/200 and 20/30 in the right and left eye, respectively. The intraocular pressure (IOP) was 51 mmHg in the right eye and 16 mmHg in the left eye. Slit lamp biomicroscopy examination of the right eye showed an injected conjunctiva, microcystic corneal edema, mid-dilated non-reactive pupil, shallow anterior chamber along with iris bombe, and a healthy optic nerve (**Figure 1**). Examination of the fellow eye was unremarkable. Gonioscopic examination revealed an appositionally closed angle in the right eye and a narrow angle in the left eye. Ultrasound biomicroscopy confirmed the presence of a shallow AC with 360-degree angle closure (**Figure 2**). The patient was started on topical IOP-lowering agents (timolol maleate 0.5%, brimonidine tartrate 0.15%) and oral systemic acetazolamide. Pilocarpine 2% was used to constrict the pupil, followed by a therapeutic Nd: YAG laser peripheral iridotomy (LPI) for the right eye and a prophylactic LPI for the left eye.

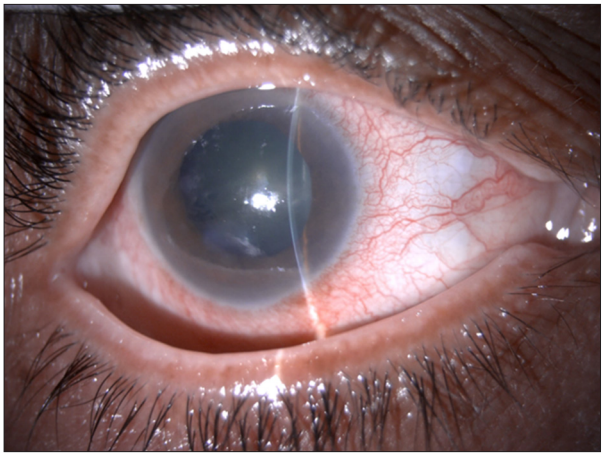

Figure 1. An external photo with a slit beam of the right eye demonstrating hyperemic conjunctiva, microcystic corneal edema, mid-dilated pupil, shallow anterior chamber (AC) with iris bombe, and early nuclear sclerosis.

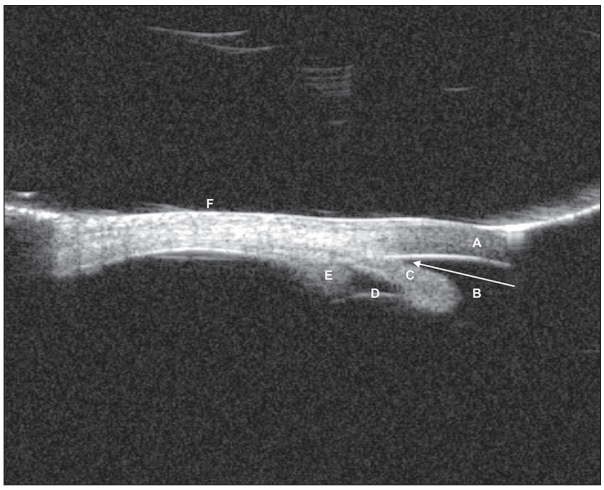

Figure 2. Ultrasound biomicroscopy of the right eye showing closed angle (at the arrow) 360 degrees, with no evidence of choroidal effusion. **A:** Cornea, **B:** Anterior Chamber, **C:** Iris, **D:** Anterior Lenticular Surface, **E:** Ciliary Body, **F:** Anterior Scleral Wall.

On the next day, the patient's symptoms and signs improved significantly. Visual acuity improved to 20/30 and IOP dropped to 20 mmHg in the right eye. The development of pupillary block shortly after the administration of a drug with anti-cholinergic properties, such as olanzapine, led to the diagnosis of drug-induced pupillary lock.

Discussion

Unlike serotonin-specific reuptake inhibitors and tricyclic antidepressants, atypical antipsychotics are rarely associated with

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angle closure glaucoma [3]. Here, we a describe case of acute angle closure triggered by olanzapine. Olanzapine, an atypical or a second-generation antipsychotic agent, acts through a combination of dopamine and serotonin type 2 antagonism, along with a weak anti-cholinergic effect. Anti-cholinergic-related systemic adverse effects include, dry mouth, constipation, urinary problems, and palpitations [4]. The pupillary block induced is explained by the weak anti-cholinergic effects associated with olanzapine [5].

Drug-induced AAC may be caused by 2 main mechanisms: pupillary block, or anterior displacement of the lens-iris diaphragm without pupillary block. Sympathomimetic agents like α1- and β2-adrenergic agonists cause pupillary dilation by acting on the dilator pupillae muscle, leading to thickening of the iris base and crowding of the iridocorneal angle, which can precipitate AAC in predisposed patients [6]. Anticholinergic agents such as tropicamide, atropine, homatropine, and cyclopentolate can also lead to mydriasis and AAC by relaxing the ciliary muscles [7]. Cholinergic agents such as pilocarpine constrict the pupil and increase the conventional outflow of the aqueous through the trabecular meshwork. Sulfa-based drugs like topiramate can cause supraciliary effusion, forward rotation of the ciliary body, and edema, relaxation of the zonules, and thickening of the lens, leading to narrowing of the AC and the development of AAC [2]. Management depends on the underlying mechanism of drug-induced AAC. Laser peripheral iridotomy is required in cases of drug-induced pupillary block, while immediate discontinuation of the triggering medication is required in topiramate-induced acute angle closure, along with topical corticosteroids and cycloplegic drops [7].

Conclusions

In summary, drug-induced AAC is an uncommon ocular emergency that may follow the administration of certain topical and systemic medications, including atypical antipsychotics. Patients started on drugs capable of inducing AAC should be instructed to visit the ophthalmology emergency department whenever they experience symptoms of AAC. Physicians, including ophthalmologists and psychiatrists, should be aware of drugs triggering pupillary block and therefore be able to educate patients about symptoms of AAC.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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