Chung Shan Medical Journal 2023; 34:91-94

airiti

Case Report

Acute transient myopia induced by famotidine: A case report and literature review

Chin-Te Huang[1,2,3], Emily Wan-Ju Su[1, 2*]

[1] Department of Ophthalmology, Chung Shan Medical University Hospital, Taichung City, Taiwan;
[2] Department of Ophthalmology, School of Medicine, Chung Shan Medical University, Taichung City, Taiwan.
[3] Department of Optometry, Chung Shan Medical University, Taichung, Taiwan.

Acute drug-induced transient myopia has been associated with various sulfa-containing medications. We present a novel case of famotidine causing acute bilateral myopia in a young woman. A 22-year-old woman presented with bilateral blurry distance vision for two weeks. An ophthalmic exam revealed a significant increase of myopia in both eyes, by -3.25 diopters in the right and -5.50 diopters in the left. Recently added oral famotidine was identified as the causative agent and discontinued promptly. Topical atropine eye drop was prescribed along with cessation of famotidine. The patient's refractive status returned to baseline with the best corrected visual acuity of 1.0 in both eyes at one-month follow-up visit. Early recognition and discontinuation of the offending medication are crucial to preventing permanent visual loss due to drug-induced transient myopia and angle closure glaucoma. Drug-induced myopia should be included in the differential diagnosis when evaluating patients with a significant acute myopic shift. To the best of our knowledge, this is the first reported case of transient myopia induced by famotidine.

Keywords: transient myopia, drug-induced myopia, famotidine.

1. Introduction

Several medications are known to induce transient myopia, with or without elevated intraocular pressure (IOP) and acute angle closure glaucoma (AACG).(1) The most reported causative agents are sulfonamide derivatives,(2) such as topiramate,(3) acetazolamide,(4) and methazolamide (5). In a review article focusing on topiramate-induced myopia and AACG, significant drug-induced myopic shift up to 8 diopters and elevated IOP as high as 60~70 mmHg were reported. (6) Permanent damage to vision is possible if acute angle closure glaucoma is not managed promptly. We now reported the clinical findings of a young female who presented with transient myopia secondary to her newly prescribed medicine, famotidine, a rare known sulfa derivative.

* Corresponding Author: Emily Wan-Ju Su
Address: No. 110, Sec. 1, Jianguo N. Rd., Taichung City, 40201, Taiwan
Tel: +886-4-2473-9595 ext. 34511
E-mail: emilywsu@gmail.com

2. Case report

A 22-year-old female presented with the bilateral progressive worsening vision for two weeks. Near vision appeared to be unchanged. She denied pain, trauma, or other neurological symptoms. Past medical history was only significant for gastroesophageal reflux disease (GERD).

On examination, the patient's corrected vision with her current myopic spectacles was 0.4 in the right eye (OD) and 0.3 in the left eye (OS). However, the best corrected visual acuity reached 1.0 in her bilateral eyes. Autorefraction showed -6.00 diopter OD and -9.00 diopter OS. These were significant deviations from her baseline myopia of -2.75 diopters

airiti

Table 1. Changes of refractive status and intraocular pressure at each visit.

Time	Refraction		IOP (mmHg)		Ophthalmic medication
	OD	OS	OD	OS	
Baseline	-2.75 sph	-3.50 sph			
Initial visit	-6.00 sph	-9.00 sph	22	21	
Week 1	-5.25 - 0.50 X 55	-7.25 - 0.25 X 5	21	19	Topical atropine 1% HS
Week 2	-4.50 - 0.25 X 60	-6.00 sph	20	20	Off Atropine
Week 4	-3.00 sph	-3.50 sph	16.3	17.3	Off Atropine

HS, every night at bedtime; IOP, intraocular pressure; OD, right eye; OS, left eye; sph, spherical refraction.

OD and -3.50 diopters OS. The slit lamp exam was grossly within normal limits with the clear cornea and deep anterior chambers. IOP were 22 mmHg OD and 21 mmHg OS. Retina and optic discs also appeared normal bilaterally.

Upon further questioning, the patient revealed that famotidine 20 mg orally twice daily was added in addition to magnesium oxide 3 weeks ago due to the recent worsening of her acid reflux symptoms. Being a sulfa-containing medication, the recently initiated famotidine was suspected as the cause of her acute myopic shift. The patient was instructed to stop oral famotidine and started on topical 1% atropine eye drops once daily in both eyes. Her refractive error and distant vision improved steadily over time at each follow-up (Table 1). IOP remained normal during the recovery. At one month follow-up, refraction and best corrected visual acuity returned to baseline.

3. Discussion

To the best of the authors' knowledge, this is the first reported case of famotidine-induced transient myopia in the literature. Famotidine is a type 2 histamine receptor antagonist that functions to decrease the amount of acid produced in the stomach. It is commonly prescribed for gastric and duodenal ulcers, erosive esophagitis, and GERD. Its chemical structure, $C_8H_{15}N_7O_2S_3$, contains a sulfonamide that is exposed on the exterior of the molecule. Its most frequently encountered side effects include headache, dizziness, constipation, and diarrhea.(7) Although the exact mechanism is still unclear, acute myopic shift and elevated IOP are believed to be precipitated by the ciliary body edema and the subsequent anterior shift of the iris-lens diaphragm.(8) The resulting anteriorly displaced lens produced a clinical acute myopic change with or without a rise in intraocular pressure. This is most likely an idiosyncratic dose-independent reaction.(3-6) When a patient presents with acute myopia with or without elevated IOP and shallow anterior chamber, drug-induced myopia should be considered. Possible differential diagnoses of sudden onset myopia may include accommodative spasm, uveitis-induced myopia, traumatic cataract, and anterior lens dislocation. Mydriasis, ocular pressure lowering medications, and most importantly, prompt cessation of the offending drug are the main components of the treatment strategy. Some authors also suggested the use of topical or systemic steroids,(5) as inflammation and edema of the ciliary body are proposed as part of the pathophysiology of this condition. Since pupillary block does not play a role in the disease mechanism, interventions such as peripheral iridotomy and miotics are ineffective in this scenario.(3) Topical miotics such as pilocarpine may even worsen the situation by further anterior displacement of the iris-lens diaphragm.(9)

At least three positive rechallenging cases of topiramate-induced AACG have been reported.(10) These patients experienced another attack of AACG and myopic shift at the same dose of topiramate that precipitated the first event. Reinstituting famotidine was not considered in our patient, as alternative treatments were readily available.

Vision recovery is generally favorable in patients with acute drug-induced myopia and AACG. In most

airiti

cases, the elevated IOP typically normalized within days, and the refractive status returned to baseline within 2 to 4 weeks.(10) However, permanent visual field defects and vision loss are possible if the disease is not recognized and treated promptly.(3, 6, 10) A detailed review of medication history is key to accurate diagnosis when the condition is suspected. Further investigation is needed to understand the mechanism behind drug-induced acute transient myopia and AACG.

References

1. Patasova K, Khawaja AP, Tamraz B, Williams KM, Mahroo OA, Freidin M, et al. Association Between Medication-Taking and Refractive Error in a Large General Population-Based Cohort. Invest Ophthalmol Vis Sci. 2021;62(2):15.
2. Panday VA, Rhee DJ. Review of sulfonamide-induced acute myopia and acute bilateral angle-closure glaucoma. Compr Ophthalmol Update. 2007;8(5):271-6.
3. Medeiros FA, Zhang XY, Bernd AS, Weinreb RN. Angle-closure glaucoma associated with ciliary body detachment in patients using topiramate. Arch Ophthalmol. 2003;121(2):282-5.
4. Binder SS, Steele W, Sr. Acute transient myopia associated with use of acetazolamide (diamox). J Am Med Assoc. 1957;165(2):154-5.
5. Kwon SJ, Park DH, Shin JP. Bilateral transient myopia, angle-closure glaucoma, and choroidal detachment induced by methazolamide. Jpn J Ophthalmol. 2012;56(5):515-7.
6. Abtahi MA, Abtahi SH, Fazel F, Roomizadeh P, Etemadifar M, Jenab K, et al. Topiramate and the vision: a systematic review. Clin Ophthalmol. 2012;6:117-31.
7. Campoli-Richards DM, Clissold SP. Famotidine. Pharmacodynamic and pharmacokinetic properties and a preliminary review of its therapeutic use in peptic ulcer disease and Zollinger-Ellison syndrome. Drugs. 1986;32(3):197-221.
8. Krieg PH, Schipper I. Drug-induced ciliary body oedema: a new theory. Eye (Lond). 1996;10 (Pt 1):121-6.
9. Ritch R. The pilocarpine paradox. J Glaucoma. 1996;5(4):225-7.
10. Fraunfelder FW, Fraunfelder FT, Keates EU. Topiramate-associated acute, bilateral, secondary angle-closure glaucoma. Ophthalmology. 2004;111(1):109-11.