Losing Weight, Losing Sight: Acute Bilateral Angle-Closure Glaucoma Triggered by Topiramate for Weight Loss

Perda de Peso, Perda de Visão: Glaucoma Agudo de Ângulo Fechado Bilateral Secundário a Topiramato para Perda Ponderal

Marta Vaz Pereira [1], Francisca Maia [1], Marina João [1], Nuno Lopes [1]

[1] Ophthalmology Department, Unidade Local de Saúde de Braga, Braga, Portugal

Recebido/Received: 2024-10-16 | **Aceite/Accepted:** 2026-05-03 | **Published online/Publicado online:** 2026-11-10

© 2026 Oftalmologia. This is an open-access article under the CC BY-NC 4.0. Re-use permitted under CC BY-NC 4.0. No commercial re-use.
© 2026 Oftalmologia. Este é um artigo de acesso aberto sob a licença CC BY-NC 4.0. Reutilização permitida de acordo com CC BY-NC 4.0. Nenhuma reutilização comercial.

DOI: https://doi.org/10.48560/rspo.38268

ABSTRACT

Acute angle-closure glaucoma (AACG) is a sight-threatening emergency, with approximately one-third of cases triggered by medications. The most common mechanism of angle closure is pupillary block. Topiramate, commonly used for migraines and epilepsy, is a rare cause of AACG that occurs due to a different mechanism: choroidal effusion and anterior displacement of the lens-iris diaphragm. This report presents a 22-year-old woman who experienced sudden bilateral blurry vision without pain or redness one week after starting topiramate for weight loss. Her corrected visual acuity was 20/100 bilaterally (-9.00-3.00x180 /-10.50-4.00x180), with a myopic shift (previous refraction: +1.25 -2.25x180 /+2.00 -4.00x180) and intraocular pressure (IOP) of 40/44 mmHg. Gonioscopy revealed 360º iridotrabecular contact, and B-scan ultrasonography confirmed bilateral uveal effusion, leading to the diagnosis of topiramate-induced AACG. Immediate discontinuation of topiramate and treatment with cycloplegics for posterior displacement of the lens-iris diaphragm, aqueous suppressants, and topical steroids resulted in complete resolution, with her visual acuity returning to 20/20 and IOP normalizing to 10 mmHg in both eyes. This case underscores the importance of a thorough medication history in patients with AACG and of recognizing rare secondary causes when features are atypical – such as younger age, bilateral involvement, myopic shift, and absence of predisposing anatomy. Topiramate–induced angle closure, though uncommon, is increasingly reported with its wider use, including in weight-loss regimens. Importantly, it is a potentially reversible condition if promptly diagnosed and with drug discontinuation.

KEYWORDS: Drug-Related Side Effects and Adverse Reactions; Glaucoma, Angle-Closure; Topiramate/adverse effects; Weight Loss/drug effects.

RESUMO

O glaucoma agudo de ângulo fechado (GAAF) é uma emergência ocular ameaçadora da visão, e cerca de 1/3 dos casos são desencadeados por medicamentos. O mecanismo mais comum de encerramento do ângulo é o bloqueio pupilar. O topiramato, habitualmente utilizado no tratamento de enxaquecas e epilepsia, é uma causa rara de GAAF que ocorre por um mecanismo diferente: efusão coroideia e consequente anteriorização do diafragma íris-cristalino. Descrevemos

o caso de uma mulher de 22 anos com visão turva bilateral súbita, sem dor ou rubor ocular, uma semana após iniciar topiramato para emagrecimento. A acuidade visual corrigida era 20/100 bilateralmente (-9,00-3,00x180 / -10,50-4,00x180), com desvio miópico (refração prévia: +1,25 -2,25x180 / +2,00 -4,00x180) e pressão intraocular (PIO) de 40/44 mmHg. A gonioscopia revelou contacto iridotrabecular a 360° e a ecografia modo-B mostrou efusão uveal bilateral, confirmando o diagnóstico de GAAF induzido pelo topiramato. A sua suspensão imediata e o tratamento com cicloplégicos para promover o deslocamento posterior do diafragma íris-cristalino, supressores do humor aquoso e corticoide tópico resultaram numa resolução completa, com recuperação da acuidade visual para 20/20 e normalização da PIO para 10 mmHg em ambos os olhos. Este caso reforça a importância de uma história medicamentosa detalhada em doentes com GAAF e do reconhecimento de causas secundárias raras nos casos atípicos — idade jovem, envolvimento bilateral, desvio miópico e ausência de fatores anatómicos predisponentes. O encerramento do ângulo induzido por topiramato, embora raro, tem sido cada vez mais reportado, dado o seu uso crescente em regimes de emagrecimento. Importa salientar que se trata de uma condição potencialmente reversível se diagnosticada atempadamente e com a suspensão do fármaco.

PALAVRAS-CHAVE: Efeitos Colaterais e Reações Adversas Relacionados a Medicamentos; Glaucoma de Ângulo Fechado; Redução de Peso/efeitos dos fármacos; Topiramato/efeitos adversos.

INTRODUCTION

Acute angle-closure glaucoma (AACG) is a sight-threatening ophthalmic emergency that requires prompt diagnosis and treatment to prevent irreversible optic nerve damage and significant vision loss.[1] This arises from sudden occlusion of the trabecular meshwork by the iris, resulting in elevated intraocular pressure (IOP). It is estimated that one-third of AACG cases are triggered by over-the-counter or prescription medications.[2] Among the culprits in AACG are adrenergic drugs, both cholinergic and anticholinergic agents, several psychiatric medications, anticoagulants and sulfa-based drugs.[2] These agents induce AACG primarily through pupillary block – the most common underlying mechanism of angle closure – secondary to their mydriatic effect. In contrast, some medications, such as topiramate, induce AACG through anterior displacement of the lens–iris diaphragm.[1] Topiramate, a commonly used drug for migraine prevention and as an antiepileptic, is a sulfamate-substituted monosaccharide, structurally unrelated to any other antiepileptic agent.[3] It is known to cause bilateral AACG due to supraciliary effusion and ciliary body edema, which pushes the lens-iris diaphragm forward, leading to a significant myopic shift, up to -17D as described in literature.[1,3] Topiramate's sulfa-derivative structure is believed to play a role in an idiosyncratic non-inflammatory disruption of the blood-aqueous barrier and increased vascular permeability within the ciliary body and choroid that leads to this effusion.[4–6] The pharmacodynamic actions of topiramate (sodium channel inhibition, GABA enhancement, antiglutamatergic effects, and carbonic anhydrase inhibition) are not directly implicated in the pathogenesis of supraciliary effusion, except for weak carbonic anhydrase inhibition, which may contribute to altered fluid dynamics but is not considered the primary cause.[7,8] This reaction typically occurs within two weeks of initiating the medication and all changes are reversible if recognized early and after discontinuation of the drug.[3]

The purpose of this article is to report a case of acute angle-closure glaucoma secondary to topiramate, taken for weight loss, where the patient's only symptom was sudden bilateral blurry vision.

The patient was observed at Hospital de Braga, ULS Braga, Portugal. Given that this report involved negligible risk and only involved the use of existing data records with non-identifiable patient data, the ethics committee of Hospital de Braga waived the need for ethical approval. Informed consent for the publication of clinical information and photography was obtained from the patient. The guidelines outlined in the Declaration of Helsinki were followed.[9]

CASE REPORT

A 22-year-old woman presented to the emergency department complaining of a sudden onset of bilateral blurry vision 4 hours ago. She reported seeing better without her glasses and denied pain or redness. Her medical history included anxiety, for which she was taking fluoxetine. Upon further questioning, she revealed she had started taking topiramate 25 mg one week earlier for weight loss. She had no relevant surgical or ocular history, except for wearing glasses (+1.25 -2.25x180 / +2.00 -4.00x180).

On examination, her corrected distance visual acuity (CDVA) was 20/100 in both eyes. Biomicroscopy revealed very discrete ciliary hyperemia and chemosis, mild corneal edema, a shallow anterior chamber, and a mild anterior chamber reaction with round and reactive pupils (Fig. 1). There were no corneal lesions or opacities identified. Her IOP was elevated, measuring 40 mmHg in the right eye and 44 mmHg in the left eye (Goldmann applanation tonometry). Gonioscopy showed a 360º iridotrabecular contact (Fig. 2), further documented in anterior segment optical coherence tomography (OCT) (Fig. 3). Fundoscopy was unremarkable, with a crowded disc with a normal-appearing macula. Her refraction was -9.00-3.00x180 and

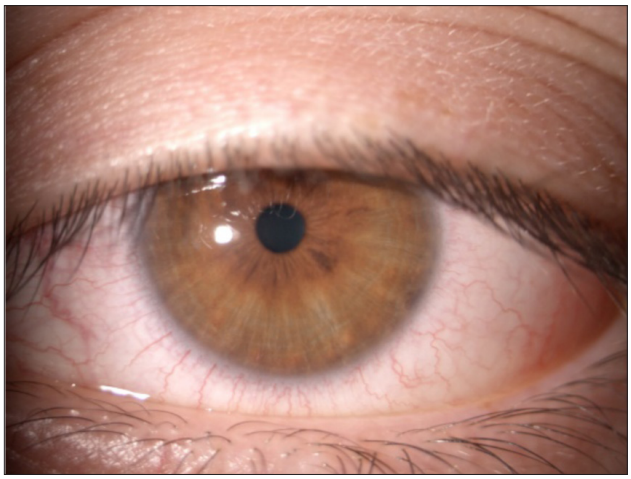


Figure 1. Biomicroscopy photograph showing discrete ciliary hyperemia and chemosis.

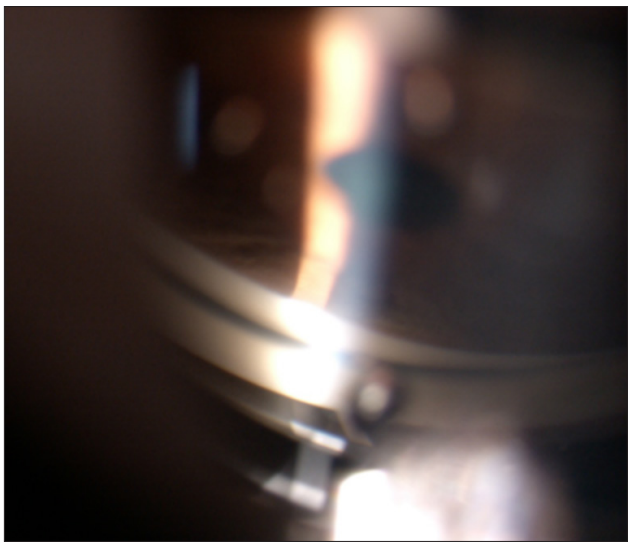


Figure 2. Gonioscopy showing 360º appositional iridotrabecular contact.

-10.50-4.00x180, revealing a significant myopic shift. B-scan ultrasonography showed bilateral choroidal thickening and a fine line of uveal effusion, confirming the diagnosis of topiramate-induced AACG (Fig. 4A). Optical coherence tomography of the macula revealed some hyperreflective spots on the internal limiting membrane bilaterally (Fig. 4B), possibly consistent with mild inflammatory debris or some reactive retinal glial response.

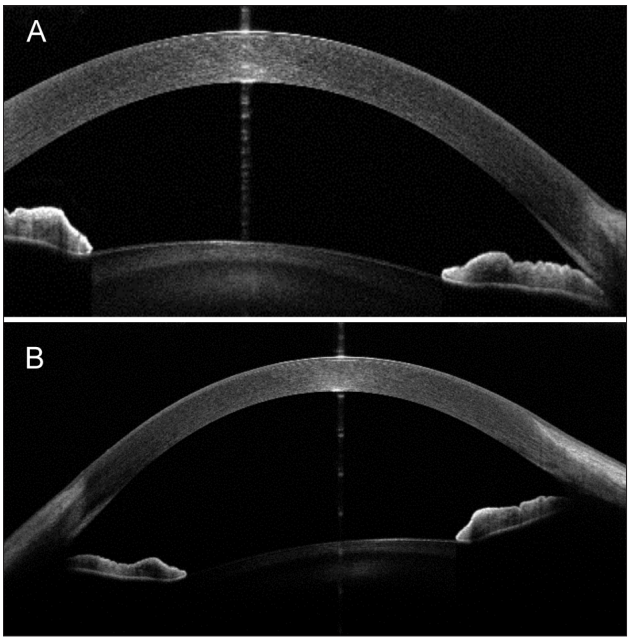


Figure 3. Anterior segment optical coherence tomography revealing a shallow anterior chamber with a closed irido-corneal angle (A) and significant improvement after suspension of topiramate and treatment (B).

The patient immediately discontinued topiramate and was started on cycloplegics, aqueous suppressants, and topical steroids, with significant improvement in four days. With a slow taper of topical steroids and eventual suspension of hypotensive medications, her visual acuity returned to 20/20 bilaterally, with complete reversal of myopic shift (+1.00 -3.75x180 / +1.00 -5.75x180), and her IOP normalized to 10 mmHg in both eyes. The iridocorneal angle opened bilaterally, and the subtle OCT changes resolved completely at 1 month of follow-up. She remained asymptomatic and without complications during follow-up.

DISCUSSION

Primary acute angle-closure glaucoma occurs due to pupillary block in most cases. The rapid rise in intraocular pressure typically leads patients to present with sudden and severe unilateral eye pain or headache, associated with

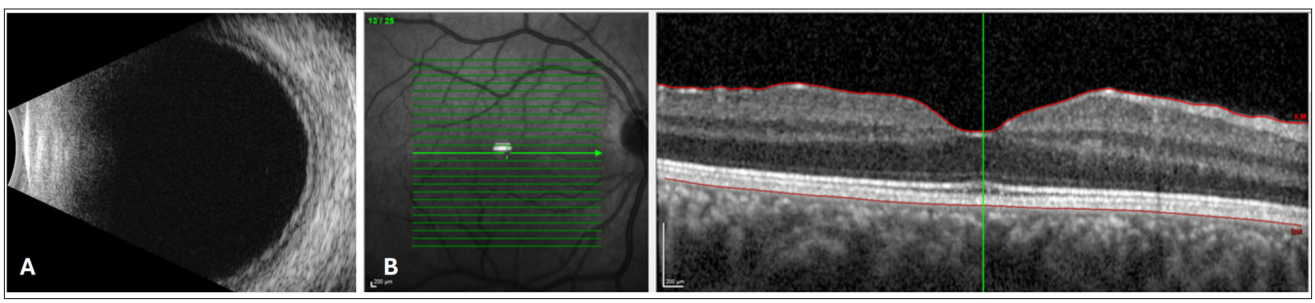


Figure 4. A: B-mode ultrasound imaging demonstrating a fine line of uveal effusion. B: Optic coherence tomography of the macula reveals some hyperreflective spots on the internal limiting membrane.

intense hyperemia, corneal edema, fixed mid-dilated pupil, blurred vision, rainbow-colored halos around bright lights, nausea and vomiting.[10] Risk factors for this type of AACG include increasing age, associated lens thickening[11]; female gender, with a 2 to 4 times higher incidence than males[12]; Southeast Asian and Inuit descent[13]; family history (predisposing anatomic features)[14]; hyperopia; shallow anterior chamber; and finally, medications – over 60 drugs, either topical or systemic, have been associated with increased risk of angle-closure.[15]

Topiramate-associated acute bilateral secondary angle-closure glaucoma was first reported in 2001 by Banta et al, emerging after post-marketing surveillance.[3,16] It is a very rare event, occurring in about 3 out of 100 000 patients undergoing treatment with this drug.[3] The largest case series of topiramate-induced AACG, which included 115 cases, showed most patients developed AACG within the first two weeks of topiramate use, typically presenting with sudden bilateral blurry vision due to the myopic shift, headache, ocular pain and hyperemia, nausea and vomiting, in a variable combination.[3] This acute myopic shift can occur within hours, reaching up to -17 diopters, though full resolution may take weeks.[1,3] In this series, 38% of patients underwent laser or surgical iridectomies, which suggests confusion with acute pupillary block, the main mechanism of primary AAC.[3] Unlike pupillary block-mediated primary angle closure, topiramate, a sulfa-based drug, induces angle closure through ciliochoroidal effusion, leading to ciliary body edema and anterior displacement of the lens-iris diaphragm, therefore, iridotomy is not the solution.[2,3]

Though primarily prescribed for migraines and epilepsy, topiramate is increasingly used off-label as a weight reduction agent due to its appetite-suppressing and satiety-enhancing effects.[3] However, side effects like cognitive impairment, paresthesia, and AACG make it unsuitable as a first-line treatment for obesity.[7]

The patient in this case report presented to the emergency department solely with blurry vision, mainly associated with myopic shift. The absence of ocular pain likely reflected the early stage of angle-closure, despite IOP being 40 mmHg on examination. Bilateral involvement, the young age and absence of cataract, unlike in the majority of primary AACG due to pupillary block, raised suspicion for a systemic or pharmacological etiology. It is important to emphasize that topiramate may induce an isolated myopic shift that presents solely as a bilateral reduction in visual acuity, without associated angle closure or intraocular hypertension.

When suspecting of this entity, an investigation should be carried out with an ocular B-mode ultrasonography, which may detect ciliary effusion, and, if available, with an ultrasound biomicroscopy, which allows more detailed imaging, especially of the structures posterior to the iris.

This case underscores the importance of thorough medical history-taking, including a detailed review of all prescription, over-the-counter medications, and supplements. Physicians should be particularly vigilant when prescribing medications like topiramate, which carry the risk of inducing AACG. High-risk patients, such as those of Southeast Asian descent, the elderly, hyperopic individuals, or phakic patients with narrow angles, should be counselled and closely monitored when starting medications that can trigger AACG. Notably, in the presence of an acute bilateral distance visual acuity reduction, with or without bilateral acute angle closure, topiramate should be considered as a possible cause, and the patient should be specifically questioned about its use. If unrecognized as a drug-related event, serious outcomes such as permanent vision loss can occur.[3] However, with prompt cessation of the offending drug and medical management, IOP normalizes in a period of hours to days, significantly improving all signs and symptoms.[3]

CONTRIBUTORSHIP STATEMENT / DECLARAÇÃO DE CONTRIBUIÇÃO

MVP: Data collection, literature review, and manuscript writing.

FM: Data collection.

MJ and NLP: Review of the final manuscript.

All authors collaborated equally in patient evaluation, diagnosis and management, data collection for the article and discussion of results.

All authors approved the definitive version of the article to be published.

MVP: Recolha de dados, revisão bibliográfica e escrita do manuscrito.

FM: recolha de dados.

MJ e NLP: Revisão do manuscrito final.

Todos os autores colaboraram igualmente na avaliação do doente, diagnóstico e abordagem terapêutica, na recolha de informação para o artigo e discussão de resultados.

Todos os autores aprovaram a versão final do artigo para publicação.

RESPONSABILIDADES ÉTICAS

Conflitos de Interesse: Os autores declaram a inexistência de conflitos de interesse na realização do presente trabalho.

Fontes de Financiamento: Não existiram fontes externas de financiamento para a realização deste artigo.

Confidencialidade dos Dados: Os autores declaram ter seguido os protocolos da sua instituição acerca da publicação dos dados de doentes.

Consentimento: Consentimento do doente para publicação obtido.

Proveniência e Revisão por Pares: Não comissionado; revisão externa por pares.

ETHICAL DISCLOSURES

Conflicts of Interest: The authors have no conflicts of interest to declare.

Financing Support: This work has not received any contribution, grant or scholarship.

Confidentiality of Data: The authors declare that they have followed the protocols of their work center on the publication of patient data.

Patient Consent: Consent for publication was obtained.

Provenance and Peer Review: Not commissioned; externally peer-reviewed.

REFERENCES

1. Medication-Induced Acute Angle-Closure Glaucoma - American Academy of Ophthalmology.[Accessed September 28, 2024] Available at: https://www.aao.org/eyenet/article/medication-induced-acute-angle-closure-glaucoma
2. Lachkar Y, Bouassida W. Drug-induced acute angle closure glaucoma. Curr Opin Ophthalmol. 2007;18:129-33. doi:10.1097/ICU.0b013e32808738d5
3. Fraunfelder FW, Fraunfelder FT, Keates EU. Topiramate-associated acute, bilateral, secondary angle-closure glaucoma. Ophthalmology. 2004;111:109-11. doi:10.1016/j.ophtha.2003.04.004
4. DailyMed - TOPIRAMATE capsule, extended release. [Accessed February 15, 2026] Available at: https://dailymed.nlm.nih.gov/dailymed/drugInfo.cfm?setid=691d276c-9cc1-4d39-828f-0f44d7ef6328
5. Craig JE, Ong TJ, Louis DL, Wells JM. Mechanism of topiramate-induced acute-onset myopia and angle closure glaucoma. Am J Ophthalmol. 2004;137:193-5. doi:10.1016/s0002-9394(03)00774-8
6. Goldberg JL, Lau AG, Fan B, Ford L, Greenberg HE. Is uveitis associated with topiramate use? A cumulative review. Clin Ophthalmol. 2016;10:1467-70. doi:10.2147/OPTH.S104847
7. Pearl NZ, Babin CP, Catalano NT, Blake JC, Ahmadzadeh S, Shekoohi S, et al. Narrative review of topiramate: clinical uses and pharmacological considerations. Adv Ther. 2023;40:3626-38. doi:10.1007/s12325-023-02586-y
8. 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. doi:10.2147/OPTH.S27695
9. Parums DV. Editorial: The 2024 Revision of the Declaration of Helsinki and its Continued Role as a Code of Ethics to Guide Medical Research. Med Sci Monit Int Med J Exp Clin Res. 2024;30:e947428. doi:10.12659/MSM.947428
10. Khazaeni B, Zeppieri M, Khazaeni L. Acute Angle-Closure Glaucoma. In: StatPearls. StatPearls Publishing; 2024. [Accessed October 2, 2024] Available at:http://www.ncbi.nlm.nih.gov/books/NBK430857/
11. Jonas JB, Iribarren R, Nangia V, Sinha A, Pardhi P, Shukla R, et al. Lens position and age: The Central India Eye and Medical Study. Invest Ophthalmol Vis Sci. 2015;56:5309-14. doi:10.1167/iovs.15-16796
12. Park SJ, Park KH, Kim TW, Park BJ. Nationwide incidence of acute angle closure glaucoma in Korea from 2011 to 2015. J Korean Med Sci. 2019;34:e306. doi:10.3346/jkms.2019.34.e306
13. Bourne RRA, Taylor HR, Flaxman SR, Keeffe J, Leasher J, Naidoo K, et al. Number of people blind or visually impaired by glaucoma worldwide and in world regions 1990 - 2010: a meta-analysis. PloS One. 2016;11:e0162229. doi:10.1371/journal.pone.0162229
14. Xu BY, Friedman DS, Foster PJ, Jiang Y, Porporato N, Pardeshi AA, et al. Ocular biometric risk factors for progression of primary angle closure disease: the Zhongshan Angle Closure Prevention Trial. Ophthalmology. 2022;129:267-75. doi:10.1016/j.ophtha.2021.10.003
15. Foster PJ, Luben R, Khawaja AP. Association, Risk, and Causation-Examining the Role of Systemic Medications in the Onset of Acute Angle-Closure Episodes. JAMA Ophthalmol. 2022;140:1064-5. doi:10.1001/jamaophthalmol.2022.3724
16. Banta JT, Hoffman K, Budenz DL, Ceballos E, Greenfield DS. Presumed topiramate-induced bilateral acute angle-closure glaucoma. Am J Ophthalmol. 2001;132:112-4. doi:10.1016/s0002-9394(01)01013-3
17. Kramer CK, Leitão CB, Pinto LC, Canani LH, Azevedo MJ, Gross JL. Efficacy and safety of topiramate on weight loss: a meta-analysis of randomized controlled trials. Obes Rev Off J Int Assoc Study Obes. 2011;12:e338-47. doi:10.1111/j.1467-789X.2010.00846.x



Corresponding Author/
Autor Correspondente:

Marta Vaz Pereira
Hospital de Braga,
Rua das Sete Fontes,
4710-243 Braga, Portugal
E-mail: martavazrxpereira@gmail.com

ORCID: 0009-0005-2782-5904