Paradoxical Acute Angle Closure Following Topiramate Discontinuation: A Case Report

Carolyn Young[1†] and Tatiana Franco[2]

[1]Geisinger College of Health Sciences, Scranton, PA 18509
[†]Doctor of Medicine Program
[2]Division of Ophthalmology, Geisinger, Pittston, PA 18505
Correspondence: cyoung01@som.geisinger.edu

Abstract

Background: Topiramate is a widely prescribed antiepileptic medication increasingly used for off-label indications such as migraine prevention, weight loss, and mood stabilization. While topiramate-induced acute angle closure (AAC) is rare, it has become more frequently recognized due to its expanding use. This condition is typically associated with ciliochoroidal effusion and anterior displacement of the lens-iris diaphragm. Although most cases occur shortly after therapy initiation, emerging reports suggest angle compromise may also occur after abrupt drug discontinuation.

Methods: We describe 2 cases of bilateral AAC in female patients without prior history of glaucoma. Case 1 presented 2 days after discontinuing topiramate, while Case 2 developed symptoms during active treatment. Both cases were evaluated with slit lamp examination, gonioscopy, tonometry, optical coherence tomography (OCT), and ultra-widefield fundus imaging.

Results: Case 1 showed shallow anterior chambers, optic disc cupping, and normal intraocular pressure (IOP) despite recent drug withdrawal. Case 2 demonstrated acute vision loss, significant myopic shift (>5.00 D), and IOP elevation up to 40 mmHg. Anterior segment OCT revealed quadrant-specific angle narrowing. Both patients improved with discontinuation of topiramate and appropriate medical therapy, including aqueous suppressants and cycloplegics. Anatomical normalization and refractive recovery were documented over follow-up.

Conclusion: These cases broaden the known spectrum of topiramate-induced angle closure by highlighting both paradoxical post-discontinuation and classic acute presentations. Clinicians should maintain a high index of suspicion for AAC in patients with recent or current topiramate exposure, regardless of IOP. Early recognition and ophthalmology referral are essential to prevent permanent vision loss.

Introduction

Topiramate is a sulfamate-substituted monosaccharide with multiple mechanisms of action, including enhancement of GABA activity, inhibition of voltage-gated sodium channels, and mild carbonic anhydrase inhibition. It is FDA-approved for epilepsy and migraine prophylaxis, but its use has expanded significantly in recent years due to off-label applications in psychiatric disorders, weight loss, and chronic pain management (1–3). As its clinical use broadens, reports of rare but vision-threatening ocular side effects—including acute angle closure and myopic shift—have become more prevalent (4).

Topiramate-induced angle closure is a secondary, non-pupillary block mechanism that differs fundamentally from primary angle closure glaucoma. Rather than iris bombe or anatomical crowding, the underlying pathology involves ciliochoroidal effusion and anterior rotation of the ciliary body, which displaces the lens-iris diaphragm forward (4–5). This leads to anterior chamber shallowing, appositional angle closure, and refractive changes such as sudden-onset myopia. While intraocular pressure is often elevated, it may be normal or only mildly increased, contributing to diagnostic uncertainty (6).

Most cases occur within the first two weeks of initiating topiramate therapy and present with acute bilateral visual blurring, eye pain, halos, and headache (7). Prompt recognition and drug cessation typically result in rapid symptom resolution and reversal of anatomical changes. However, emerging reports describe more atypical presentations, including cases occurring after a missed dose or after abrupt discontinuation of the drug (8).

These paradoxical variants can be more subtle, sometimes presenting without pain, without elevated intraocular pressure, or with delayed onset—raising the risk of misdiagnosis or inappropriate treatment.

In this report, we present 2 cases that illustrate the clinical spectrum of topiramate-induced angle closure. The first case involves a paradoxical subacute presentation following abrupt drug discontinuation, in which visual symptoms developed despite normal intraocular pressures and a lack of ocular pain. The second case represents a more classic acute presentation shortly after therapy initiation, with bilateral myopic shift, angle narrowing, and elevated intraocular pressure (IOP). Together, these cases underscore the importance of recognizing variable presentations of topiramate-induced angle closure, and they highlight the role of detailed medication history, multimodal imaging, and timely management in preventing permanent vision loss.

Case Presentation

Case 1

A 37-year-old female with no significant ophthalmic history presented to the Emergency Department (ED) at Geisinger Wyoming Valley Medical Center in October 2023 with bilateral photopsia, visual disturbances described as halos and flare-like patterns around lights, and a dull right-sided headache. The patient also noted a sudden shift in her vision, characterized by increasing difficulty with distance focus. These symptoms developed 2 days after abrupt discontinuation of topiramate, which she had initiated 4 days earlier for migraine prophylaxis and weight management. She denied any history of glaucoma, ocular trauma, autoimmune disease, or systemic inflammatory conditions.

Initial visual acuity was significantly reduced to 20/300 in the right eye (OD) and 20/150 in the left eye (OS), improving to 20/100 OD and 20/70 OS with pinhole correction. Intraocular pressures (IOP) measured by Goldman applanation tonometry were within normal limits: 20 mmHg OD and 18 mmHg OS. Pupils were equal, round, and reactive to light with no afferent pupillary defect. Extraocular motility was full, and visual fields were intact to confrontation. Slit-lamp examination revealed normal lids and lashes, clear corneas with no staining on fluorescein testing, and bilaterally shallow anterior chambers without cell or flare. The irides were round and reactive, and the lenses were clear. Gonioscopy revealed closed angles in all quadrants bilaterally, with no visible trabecular meshwork or angle structures. Fundoscopic examination revealed large optic nerves with cup-to-disc ratios of 0.8 OD and 0.9 OS. The peripheral retina was notable for lattice degeneration in the right eye; maculae and vasculature appeared normal bilaterally.

A broad differential diagnosis was considered. Primary acute angle-closure glaucoma (AACG) was initially suspected due to the shallow anterior chambers and optic nerve cupping; however, this diagnosis was less likely in the absence of elevated IOP, ocular pain, corneal edema, or mid-dilated fixed pupils. The bilateral and symmetric nature of the presentation further favored a secondary angle-closure mechanism. Optic neuritis was also considered given the visual complaints and headache but was ruled out due to the absence of pain with eye movement, preserved color vision, intact visual fields, and normal optic disc appearance aside from cupping. Posterior uveitis was considered but deemed unlikely due to the absence of vitreous inflammation or retinal lesions. Retinal pathology such as central serous chorioretinopathy was also considered but lacked corresponding macular findings on examination.

Ancillary imaging was obtained to further characterize the ocular findings. Optical coherence tomography (OCT) of the retinal nerve fiber layer (RNFL) demonstrated normal thickness across all quadrants in both eyes, with no evidence of glaucomatous damage. Macular OCT revealed preserved foveal contour and intact ganglion cell–inner plexiform layer (GC-IPL) thickness, with no signs of cystoid macular edema, subretinal fluid, or retinal layer disruption (Figure 1). Ultra-widefield fundus imaging demonstrated peripapillary nerve fiber layer striations and suspected vitreoretinal traction: the right eye exhibited localized bundle loss, while the left eye showed retinal striae suggestive of mechanical stress (Figures 2 and 3). Anterior segment OCT revealed significant narrowing and iridocorneal apposition across multiple quadrants bilaterally, confirming anatomical angle closure despite the normal IOP (Figure 4). Standard automated perimetry (Humphrey 24-2) conducted 3 months later showed mild functional asymmetry, with the right eye demonstrating a glaucoma hemifield test result of "Outside Normal Limits" and the left eye "Within Normal Limits." Test reliability was limited by a high rate of false positives, though the findings were

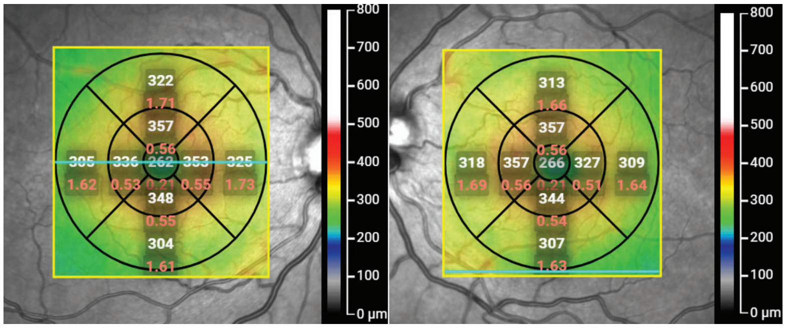

Figure 1. Macular optical coherence tomography (OCT) ganglion cell analysis for Case 1, demonstrating preserved ganglion cell-inner plexiform layer (GC-IPL) thickness in both eyes. The values fall within expected ranges, with no significant thinning or asymmetry. These findings support the absence of structural retinal ganglion cell damage despite the acute angle closure episode.

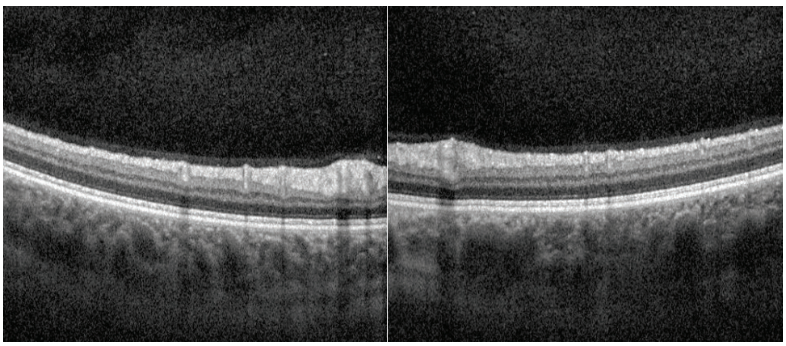

Figure 2. Spectral-domain optical coherence tomography (SD-OCT) horizontal macular scans for Case 1, demonstrating intact foveal contour and preserved retinal architecture in both eyes. No subretinal or intraretinal fluid, macular edema, or outer retinal disruption is observed, supporting structural recovery following the resolution of topiramate-induced acute angle closure.

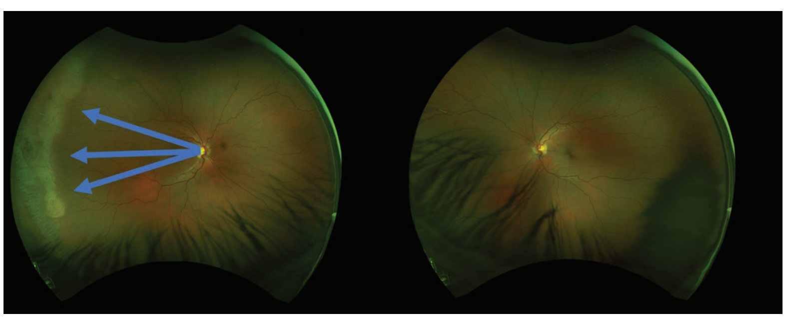

Figure 3. Case 1 widefield fundus imaging of both eyes obtained via Optomap ultra-widefield retinal imaging. The left image (OD) demonstrates retinal nerve fiber layer (RNFL) striations with blue arrows indicating the direction of nerve fiber bundle loss. The right image (OS) reveals retinal striae suggestive of vitreoretinal traction or mechanical stress.

consistent with mild visual field loss corresponding to the observed optic nerve cupping.

Additional systemic evaluation was conducted to investigate potential non-ophthalmic contributors. Bloodwork revealed elevated inflammatory markers, including a C-reactive protein of 75 mg/L and a white blood cell count of 14.98 $\times 10^9$/L. Magnetic resonance imaging (MRI) of the brain demonstrated an enlarged empty sella, but no demyelinating lesions, mass effect, or signs of elevated intracranial pressure. There was no evidence of central nervous system inflammation or compressive pathology. Given the elevated inflammatory markers, the patient was referred to rheumatology and hematology for further evaluation, though no definitive systemic diagnosis was established.

In the context of recent topiramate use followed by abrupt discontinuation, the bilateral angle closure, shallow anterior chambers, myopic shift, and confirmatory imaging findings supported the diagnosis of paradoxical topiramate-induced angle closure. The normal IOP and absence of acute pain or corneal changes suggested a more insidious course. The anatomical findings were consistent with ciliochoroidal effusion leading to anterior displacement of the ciliary body and forward displacement of the lens-iris diaphragm. This mechanism is well described in topiramate-associated ocular syndromes and distinguishes it from primary angle-closure glaucoma. The patient's symptoms gradually improved with conservative management, including close ophthalmologic monitoring and permanent discontinuation of the medication. No surgical intervention or topical therapy was required.

Case 2

A 43-year-old female presented to the ED in March 2025, with acute bilateral vision loss, excessive tearing, and ocular irritation that began approximately 12 hours prior to evaluation. She denied eye pain, photophobia, trauma, or contact lens use. Her ocular history was notable only for bilateral laser-assisted in situ keratomileusis (LASIK) approximately 20 years prior. One week before presentation, she had initiated topiramate 25 mg daily for weight gain attributed to prior psychotropic medication use. She reported that she had not taken her full dose the previous day.

At presentation, uncorrected visual acuity was reduced to 20/100 in both eyes. Pupils were equal and reactive without relative afferent defect. Extraocular motility was intact. Slit-lamp examination revealed moderate conjunctival injection and chemosis but no corneal epithelial defect or anterior chamber inflammation. Applanation tonometry revealed markedly elevated intraocular pressures, measured at 39 mmHg OD and 40 mmHg OS. Anterior chambers were shallow in both eyes. Based on the acute, bilateral nature of the symptoms, elevated IOP, and recent topiramate exposure, the leading diagnosis was secondary angle-closure glaucoma, likely medication-induced.

Initial management included cessation of topiramate and initiation of topical therapy consisting of brimonidine tartrate 0.2% and timolol 0.5% twice daily, prednisolone acetate 1% three times daily, and cyclopentolate once daily. On follow-up the next day, uncorrected visual acuity had decreased to 20/250 OD and 20/300 OS but improved with pinhole to 20/50 OU. Pupils remained reactive and extraocular movements were preserved. Slit-lamp examination revealed LASIK flaps bilaterally, clear corneas, and persistent shallow anterior chambers. Chemosis had improved. Intraocular pressures had decreased to 18 mmHg in both eyes. Gonioscopy demonstrated closed angles in all four quadrants bilaterally, with no visible angle structures.

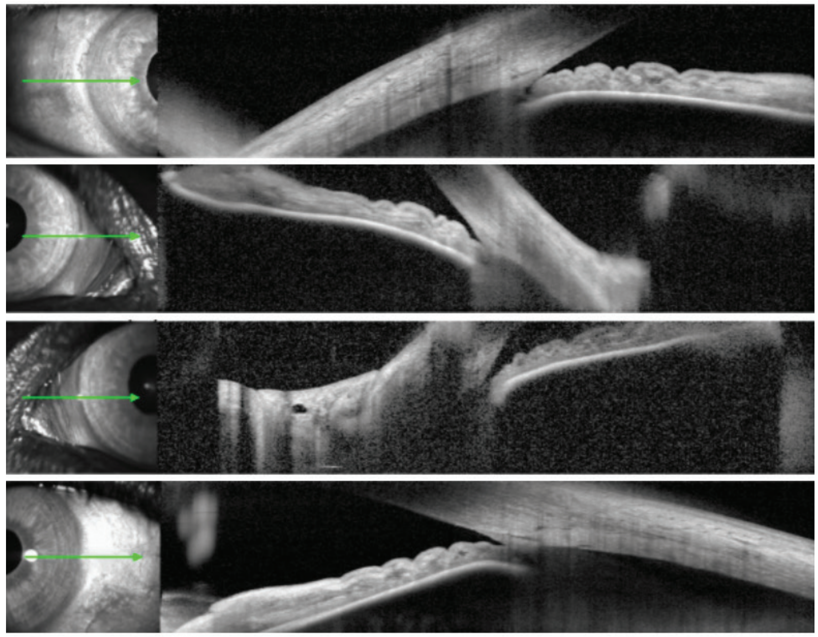

Figure 4. Case 1 anterior segment optical coherence tomography (AS-OCT), showing cross-sectional views of the iridocorneal angle in multiple quadrants. Images reveal significant narrowing of the anterior chamber angles with variable degrees of iridocorneal apposition, consistent with topiramate-associated angle compromise. These findings align with the clinical diagnosis of paradoxical angle closure following drug discontinuation and demonstrate residual anatomical crowding despite normalization of intraocular pressure.

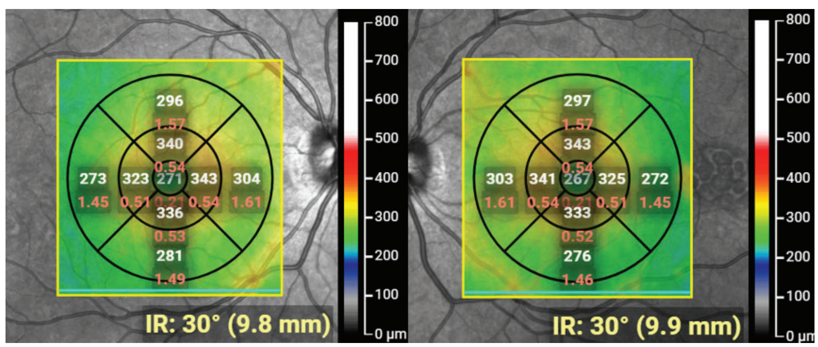

Figure 5. Macular optical coherence tomography (OCT) ganglion cell analysis for Case 2, revealing preserved ganglion cell-inner plexiform layer (GC-IPL) thickness bilaterally. No focal or diffuse thinning is observed, and measurements remain within expected ranges despite prior angle closure. These findings suggest that, while RNFL thinning was borderline, the central macular ganglion cell layers were structurally intact at the time of imaging.

Multimodal imaging was obtained to further characterize the anatomical changes and evaluate for possible glaucomatous damage. Optical coherence tomography (OCT) of the retinal nerve fiber layer demonstrated borderline thinning, most pronounced in the nasal quadrants of both eyes, which may have been artifact or related to axial elongation. Macular OCT revealed preserved ganglion cell-inner plexiform layer (GC-IPL) thickness bilaterally, with no evidence of central ganglion cell loss or retinal disruption (Figure 5). Spectral-domain OCT of the macula confirmed intact foveal contour and normal lamination of retinal layers, with no signs of macular edema or subretinal fluid (Figure 6). These findings supported the absence of irreversible retinal or optic nerve damage at the time of imaging.

Ultra-widefield fundus photography captured during early follow-up demonstrated grade 3 peripheral lattice degeneration in both eyes and normal-appearing optic nerves without edema, hemorrhage, or

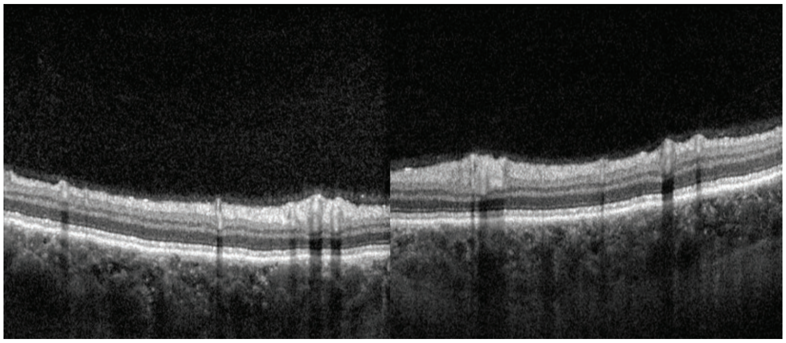

Figure 6. Spectral-domain optical coherence tomography (SD-OCT) horizontal macular scans for Case 2, showing preserved foveal contour and normal lamination of retinal layers in both eyes. There is no evidence of macular edema, subretinal fluid, or disruption of the outer retinal layers, consistent with structural integrity following topiramate-associated angle closure.

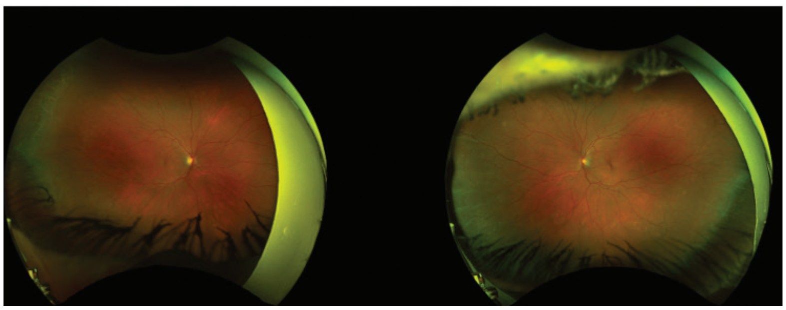

Figure 7. Case 2 ultra-widefield fundus imaging (Optos) of both eyes obtained during follow-up. The left image (OD) and right image (OS) demonstrate clear visualization of the posterior pole with no optic disc edema or hemorrhage. Peripheral lattice degeneration (grade 3+) is visible in both eyes, most prominently inferotemporally. The nerves appear within normal limits.

pallor (Figure 7). These peripheral retinal findings were considered incidental and unrelated to the acute presentation.

Anterior segment OCT (AS-OCT) provided detailed anatomical confirmation of the angle closure. Imaging showed significant quadrant-to-quadrant variability in angle configuration. In the right eye, iridocorneal touch and anterior segment crowding were evident both temporally and nasally, consistent with ciliochoroidal effusion and forward displacement of the lens-iris diaphragm. In contrast, the left eye demonstrated more open angles in the corresponding quadrants (Figure 8). These findings confirmed an asymmetric but bilateral anatomical predisposition to non-pupillary block angle closure, aligning with known mechanisms of topiramate-induced ocular changes.

Over the next 48 hours, intraocular pressures normalized to 8 mmHg bilaterally. Manifest refraction revealed a pronounced myopic shift: −5.75 D OD and −5.50 D OS, consistent with anterior displacement of the lens-iris diaphragm. Visual acuity stabilized and slit-lamp examination remained unchanged with shallow anterior chambers but no evidence of inflammation. No corneal edema, uveitis, or optic disc edema was observed throughout the course. Over the following week, the patient's refractive error showed progressive improvement. One week after presentation, manifest refraction revealed a mild hyperopic shift, and by two weeks, the refraction had returned to near-emmetropic values (−0.25 D OD and plano OS), indicating resolution of the ciliochoroidal effusion. Intraocular pressures remained within normal limits.

Alternative diagnoses were considered, including primary angle-closure glaucoma, LASIK-related ectasia or flap complications, and uveitic glaucoma. However, the acute bilateral presentation, absence of corneal or anterior segment inflammation, recent exposure to topiramate, and dynamic anatomical findings on AS-OCT collectively favored a diagnosis of secondary angle closure due to topiramate-induced ciliochoroidal effusion. The rapid normalization of IOP and resolution of refractive error following medication cessation and topical therapy further supported this conclusion.

This case illustrates a classic presentation of topiramate-associated acute angle closure, with bilateral IOP elevation, marked myopic shift, and anterior segment crowding. Multimodal imaging — including RNFL and macular OCT, widefield fundus photography, and anterior segment OCT — was essential for confirming the diagnosis, ruling out glaucomatous progression, and monitoring therapeutic response. The patient recovered fully with prompt medical intervention and discontinuation of the offending agent, avoiding long-term structural or visual sequelae.

Discussion

These 2 cases highlight the diagnostic complexity and clinical variability of topiramate-induced angle closure, a rare but increasingly recognized ocular complication of a widely prescribed systemic

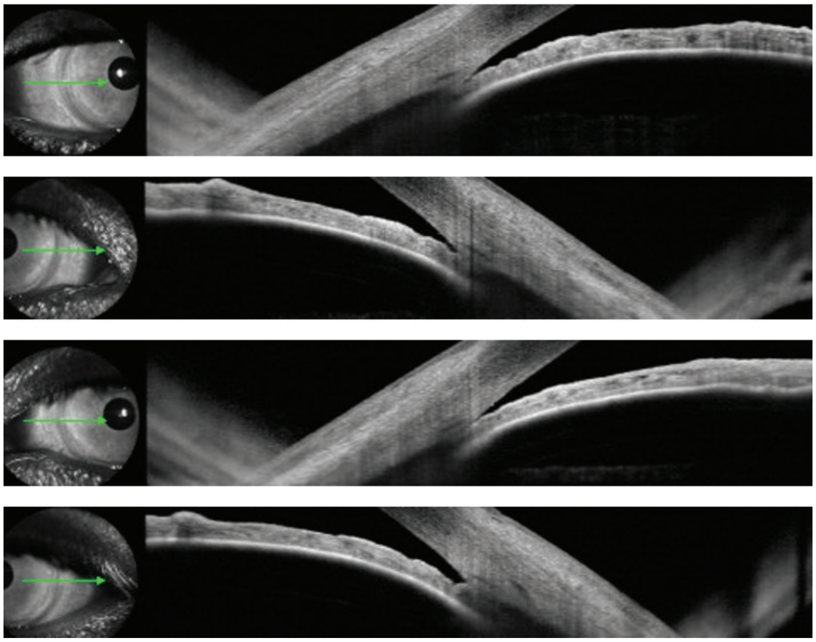

Figure 8. Case 2 anterior segment optical coherence tomography (AS-OCT) for temporal and nasal angles in the right eye shown in the top 2 images above, each with corresponding localization images. Bottom 2 images show AS-OCT for nasal and temporal angles in the left eye similarly with corresponding localization images. The green scan lines indicate the orientation of each cut. Images reveal geographical-dependent variability in angle anatomy: the right eye images (top and second images) show iridocorneal touch and anterior segment crowding, consistent with angle closure; the temporal and nasal quadrants for the left eye (third and fourth images) demonstrate more open angles with visible separation between the iris and cornea. This asymmetry underscores the importance of multi-region imaging when assessing angle status following topiramate discontinuation.

medication. Topiramate is used for epilepsy, migraine prevention, weight loss, and mood stabilization, with expanding off-label use contributing to a rise in associated adverse events (1, 5). While acute angle closure associated with topiramate is typically described as an early complication of treatment, often occurring within 7 to 21 days of initiation, the cases presented here demonstrate that clinically significant anatomical compromise may also occur after abrupt discontinuation or when dosing is inconsistent (7, 8).

Topiramate-induced angle closure is mechanistically distinct from primary angle closure glaucoma. Rather than being caused by pupillary block, the condition arises from ciliochoroidal effusion and anterior displacement of the lens-iris diaphragm, leading to anterior chamber shallowing and secondary angle narrowing (6, 7). This non-pupillary block mechanism is thought to be related in part to topiramate's weak inhibition of carbonic anhydrase isoenzymes (CA-II and CA-IV), which alters aqueous dynamics and increases uveal permeability (5). These changes are often bilateral and may not consistently produce elevated intraocular pressure (IOP), making diagnosis challenging when patients present without classic signs of acute glaucoma.

In Case 1, the patient developed shallow anterior chambers, optic nerve cupping, and subjective visual changes 2 days after discontinuing topiramate. Notably, her IOP remained within normal limits throughout the course. These findings underscore that topiramate-induced angle closure may persist beyond active drug use due to the medication's extended half-life and wide tissue distribution, particularly in individuals with altered renal function (1, 2, 4). The absence of pain or corneal edema further complicated diagnosis, demonstrating the need for high clinical suspicion and detailed anatomical assessment even in atypical presentations.

Case 2 presented more classically, with acute bilateral blurry vision, markedly elevated IOP, and a myopic shift greater than –5.50 diopters. The patient had initiated topiramate one week earlier and had reduced her dose the day before symptom onset. Slit-lamp examination showed chemosis and shallow anterior chambers, and gonioscopy revealed complete angle closure in both eyes. The rapid clinical improvement following discontinuation of the drug and initiation of topical aqueous suppressants and cycloplegics further supported the diagnosis. This case highlights the more well-recognized end of the spectrum, where the diagnosis is clearer and treatment outcomes are typically favorable if prompt therapy is initiated (1, 7).

These 2 cases therefore represent different but complementary patterns of topiramate-induced angle closure: one paradoxical and subacute, occurring after discontinuation with normal IOP, and the other acute and classic, presenting during active therapy with elevated IOP and dramatic refractive shift. Both scenarios support the current understanding that ciliochoroidal effusion, rather than pupillary block, is the underlying mechanism—resulting in anterior segment crowding and angle compromise without necessarily involving intraocular inflammation (5, 6).

Management of topiramate-induced angle closure differs from primary angle closure. Laser peripheral iridotomy is ineffective, as it does not address the non-pupillary block pathophysiology. Treatment instead focuses on cessation of the offending agent, reduction of aqueous production, and posterior displacement of the lens-iris diaphragm using cycloplegics (7). Topical corticosteroids may be beneficial in cases with inflammatory features, though their role remains less well defined. In both cases presented here, prompt recognition and medical management led to complete resolution of symptoms and anatomical recovery, avoiding the need for surgical intervention.

The existing literature supports a rising incidence of topiramate-related ocular adverse events, particularly as the drug is prescribed more broadly across neurology, psychiatry, and primary care (1, 5, 7). Many prescribers may be unaware of this rare but reversible complication, increasing the risk of delayed diagnosis. According to a systematic review by Abtahi et al., nearly all documented cases of topiramate-induced angle closure occurred within 2–3 weeks of therapy initiation, but late and post-discontinuation presentations may be underreported (7). Additionally, as shown in our first case, angle closure may occur even in the absence of elevated IOP or overt pain —factors that could easily lead to missed or delayed ophthalmology referral if clinicians rely solely on classic glaucoma signs.

Therefore, clinicians in all specialties should maintain a high index of suspicion in patients presenting with acute vision changes, halos, or myopic shift while on or recently off topiramate therapy. Comprehensive ocular evaluation — including slit-lamp examination, gonioscopy, and multimodal imaging when available — is essential to confirm the diagnosis and avoid irreversible visual loss. In both of our cases, early recognition and appropriate management resulted in complete visual and anatomical recovery, underscoring the importance of timely intervention.

Conclusion

Topiramate-induced angle closure represents a distinct and often underrecognized form of secondary angle-closure glaucoma, characterized by ciliochoroidal effusion, anterior displacement of the lens-iris diaphragm, and resultant anterior chamber shallowing without pupillary block (7). The cases presented here underscore the broad clinical spectrum of this condition — ranging from acute presentations with elevated intraocular pressure (IOP) and pain to subacute or asymptomatic cases occurring even after medication discontinuation.

In Case 1, a patient developed bilateral angle narrowing and visual disturbances several days after abrupt topiramate cessation yet had normal IOP and minimal discomfort. This aligns with emerging literature showing that topiramate-induced ocular changes may persist beyond active use due to prolonged drug half-life and slow redistribution from ocular tissues (8, 13). Notably, Tran et al. described a similar paradoxical presentation of angle closure following drug withdrawal, emphasizing the importance of considering recent topiramate exposure even when therapy has been stopped (8). Moreover, Jalali et al. and Abtahi et al. both highlight that topiramate-induced angle closure is non-pupillary in nature and may not respond to traditional interventions such as laser iridotomy (7, 11).

In contrast, Case 2 exemplifies a more classic presentation: acute bilateral blurry vision, myopic shift, chemosis, and elevated IOP during active topiramate use. This presentation corresponds closely to the majority of cases described in systematic reviews and pharmacovigilance reports, including the original cases reported by Fraunfelder et al (9). However, even within such typical cases, variability exists. As described by Goyal et al., some patients present with rapidly reversible anatomical changes, while others may demonstrate lingering symptoms or asymmetric involvement despite bilateral exposure (12).

Both cases reinforce that the underlying mechanism involves ciliochoroidal effusion — distinct from pupillary block — leading to anterior displacement of the ciliary body and shallowing of the anterior chamber (7, 9). The topiramate package insert also acknowledges angle-closure glaucoma as a rare but serious adverse effect, typically occurring within the first month of use, though exact mechanisms remain incompletely understood (13). In both of our cases, optical coherence tomography (OCT), gonioscopy, and careful refractive assessment were instrumental in confirming the diagnosis and guiding treatment.

Management centers on immediate discontinuation of the drug, use of aqueous suppressants, and cycloplegics to posteriorly rotate the lens-iris diaphragm (7, 9). Corticosteroids may be added to reduce ciliary body edema, although evidence remains limited to case reports and small series (11, 12). Importantly, laser peripheral iridotomy is ineffective, and surgical intervention is rarely required if diagnosis is timely (7). In both patients, appropriate medical management resulted in full anatomical and functional recovery.

Recent literature emphasizes that demographic risk factors for topiramate-induced angle closure differ from those of primary angle closure. Patients are often younger, female, and without hyperopia or crowded anterior segments (7, 11). Additionally, several reports, including Mansoor et al., describe atypical cases involving prolonged myopic shift and delayed resolution, highlighting the need for extended monitoring even after symptom improvement (10).

Despite its low incidence, the expanding use of topiramate across neurology, psychiatry, and primary care underscores the need for broader awareness of its ocular complications (7, 9). Clinicians should maintain a high index of suspicion when evaluating patients presenting with acute or subacute visual symptoms—including blurred vision, halos, or refractive changes—particularly if they have recently initiated or discontinued topiramate. Early ophthalmologic referral is essential, as prompt intervention can reverse symptoms and prevent permanent vision loss.

Disclosures

None

Acknowledgements

None

References

1. Fraunfelder FW, Fraunfelder FT, Keates EU. Topiramate-associated acute, bilateral, secondary angle-closure glaucoma. *Ophthalmology*. 2004;111(1):109–111.
2. Shank RP, Gardocki JF, Vaught JL, et al. Topiramate: preclinical evaluation of a structurally novel anticonvulsant. *Epilepsia*. 1994;35(2):450–460.
3. Maryanoff BE, Nortey SO, Gardocki JF, Shank RP, Dodgson SP. Anticonvulsant O-alkyl sulfamates: 2,3:4,5-bis-O-(1-methylethylidene)-ß-D-fructopyranose sulfamate and related compounds. *J Med Chem*. 1987;30(5):880–887.
4. Vega D, Maalouf NM, Sakhaee K. Increased propensity for calcium phosphate kidney stones with topiramate use. *Expert Opin Drug Saf*. 2007;6(5):547–557.
5. Choi J, Yoon D, Park M, Joung KI, Shin JY. Topiramate-related adverse events: pattern and signals in the Korea Adverse Event Reporting System, 2010–2017. *Medicine (Baltimore)*. 2020;99(42):e22669.
6. Etminan M, Maberley D, Mikelberg FS. Use of topiramate and risk of glaucoma: a case-control study. Am J Ophthalmol. 2012;153(5):827–830.
7. Abtahi MA, Abtahi SH, Fazel F, et al. Topiramate and the vision: a systematic review. *Clin Ophthalmol*. 2012;6:117–131. doi:10.2147/OPTH.S27695.
8. Tran HV, Ravinet E, Schnyder C, Reichhart M, Guex-Crosier Y. Blood–brain barrier disruption associated with topiramate-induced angle-closure glaucoma of acute onset. *Klin Monbl Augenheilkd*. 2006;223(5):425–427.
9. Quist TS, Fortenbach CR, Miller MA, Bettis DI. Topiramate-induced angle closure. EyeRounds.org. Published 2019. Accessed May 21, 2025. https://webeye.ophth.uiowa.edu/eyeforum/cases/283-topiramate-induced-angle-closure.htm
10. Eggebrecht MS, Sriram S, Tamhankar MA. Topiramate-associated bilateral angle-closure glaucoma: a case series and review. *Indian J Ophthalmol*. 2024;72(4):852–855.
11. Gidal BE, Clark AM, Anders B, Gilliam F. The application of half-life in clinical decision making: comparison of the pharmacokinetics of extended-release topiramate (USL255) and immediate-release topiramate. Epilepsy Res. 2017;129:26–32. doi:10.1016/j.eplepsyres.2016.10.020
12. Guier CP, Gokhale NS, Walters GJ. Topiramate-induced bilateral acute angle-closure glaucoma. *Optometry*. 2004;75(7):431–437.
13. Lobo AM, Sobrin L, Papaliodis GN, Buxton DF, Kaiser PK. Topiramate-induced acute-onset bilateral myopia and angle-closure glaucoma. *Am J Ophthalmol*. 2004;137(1):193–195.