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Acute Angle Closure in an Eye with Dense Vitreous Hemorrhage and Retinal Detachment in the Setting of Neovascular Age-Related Macular Degeneration

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Introduction

Acute angle closure (AAC) is an ophthalmic emergency that occurs when obstruction of aqueous humor outflow leads to a sudden and pronounced increase in intraocular pressure. Patients typically present with an actue episode of unilateral blurred, red and painful eye. Most cases are primary and occur in eyes with predisposing anterior-segment anatomical features, particularly a shallow anterior chamber and a narrow iridocorneal angle.[1] Nevertheless, a wide variety of secondary causes should be considered when the clinical scenario is atypical. These mechanisms include medication-induced changes, neovascularization, and posterior-segment expansion that displaces the lens–iris diaphragm anteriorly, producing appositional angle closure.[2,3]

Herein, we report a case of a 74-year-old patient who presented to the ophthalmic emergency department with an acutely painful eye, no light perception, and diffuse shallowing of the anterior chamber in the affected eye. B-scan ultrasonography revealed findings suggestive of a superotemporal retinal detachment and dense vitreous hemorrhage throughout the vitreous cavity. Although these posterior-segment abnormalities were observed concurrently with acute angle closure, the mechanism responsible for the marked anterior chamber shallowing could not be established. This case highlights the value of ocular ultrasonography in evaluating atypical acute angle closure and the importance of distinguishing associated posterior-segment findings from a demonstrated cause.

Case Report

A 74-year-old male presented with a 7-day history of severe right ocular pain and sudden vision loss, accompanied by ipsilateral hemicranial headache, nausea, and vomiting. There was no history of diabetes,

anticoagulant use, known bleeding disorders, recent ocular trauma, or a Valsalva-associated event. His ocular history included bilateral neovascular age-related macular degeneration (nAMD) treated with multiple intravitreal anti–vascular endothelial growth factor injections approximately 7–8 years earlier. However, he subsequently discontinued follow-up.

Before being transferred to our care**,** he had been hospitalized locally and treated with intravenous mannitol 20%, oral acetazolamide 250 mg, fixed-combination brimonidine/timolol twice daily, and brinzolamide 1% three times daily. Despite treatment, the right ocular pain persisted.

On admission, visual acuity in the right eye was no light perception, and intraocular pressure (IOP) was 58 mmHg. Slit-lamp examination revealed nearly 360° conjunctival injection and chemosis (Figure 1A), diffuse corneal edema, and an extremely shallow anterior chamber (Figure 1B). The lens–iris diaphragm was markedly displaced anteriorly, nearly apposing the posterior corneal surface (Figure 1B). The lens showed grade 3 brown nuclear opalescence without intumescence, total white opacity, or clinically evident subluxation, and the pupil was mid-dilated and nonreactive; fundus examination was limited due to the patient's severe ocular pain, difficulty opening the eyelids, and significant corneal edema. Only a view of the dense vitreous hemorrhage could be observed via slit-lamp biomicroscopy, while the optic disc and macula were completely obscured and unobservable. No iris neovascularization was observed. Initial gonioscopy of the right eye was deferred because severe ocular pain, eyelid and conjunctival swelling, and corneal edema, prevented adequate exposure of the globe and reliable angle examination. In the left eye, visual acuity was counting fingers at 1 m, IOP was 7 mmHg, and the anterior chamber was moderately deep with a wide-open angle on gonioscopy (Figure 1C). A cataract, macular scar, and well-defined optic disc margins were also noted in the left eye.

The patient was treated with intravenous mannitol 20%, oral acetazolamide 500 mg twice daily, fixed-combination brimonidine/timolol twice daily, and brinzolamide 1% three times daily in the right eye. Despite maximal pressure-lowering therapy, the IOP of the right eye remained 58 mmHg, and the eye remained painful.

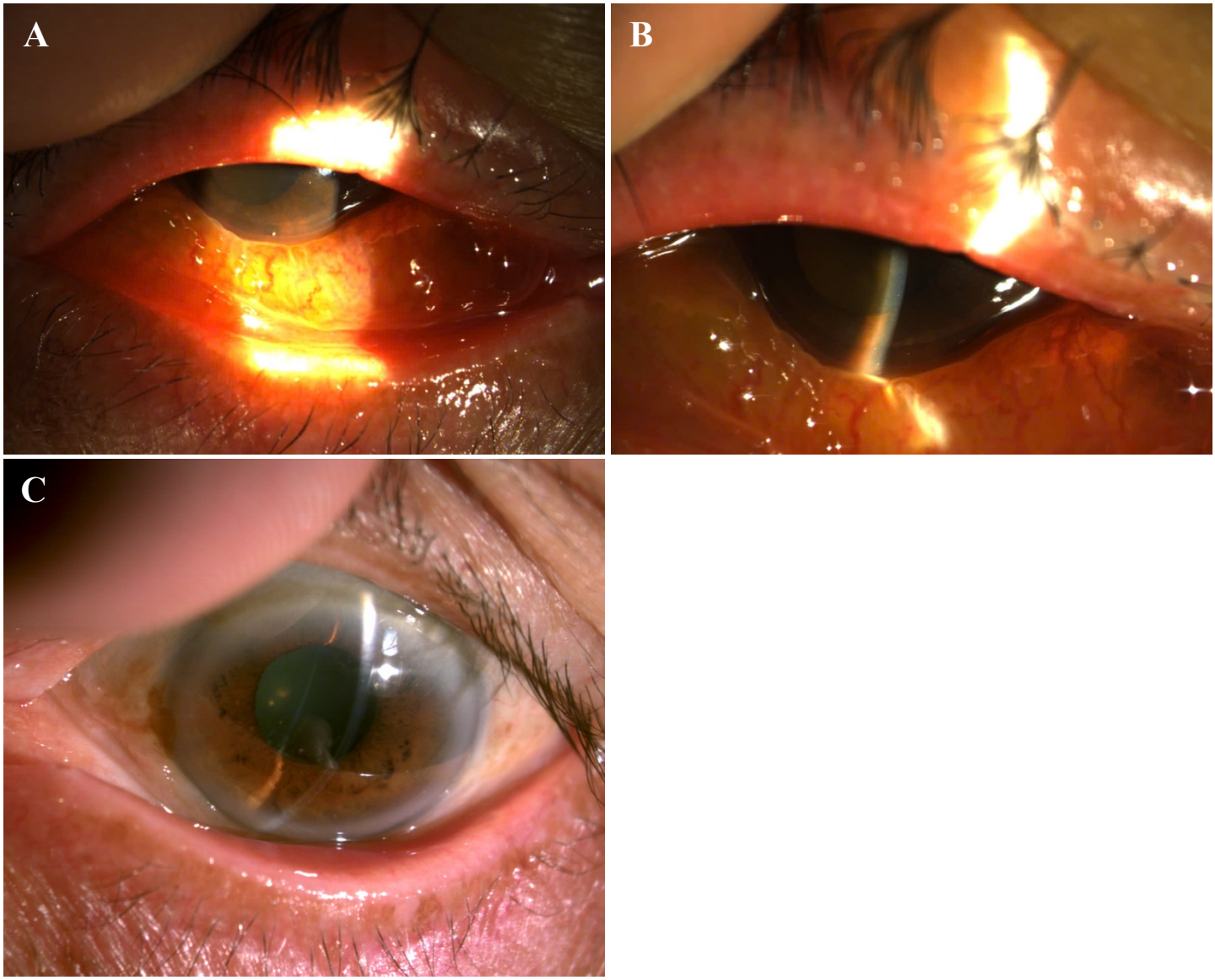


Figure 1. Slit-lamp examination at presentation. A. The right eye shows diffuse conjunctival injection and chemosis. B. An extremely shallow anterior chamber in the right eye, with lens–iris diaphragm displaced anteriorly. C. The left eye shows a moderately deep anterior chamber without conjunctival injection or corneal edema and an open-angle on gonioscopy.

B-scan ultrasonography was performed the following morning. Dynamic examination showed findings suggestive of a superotemporal retinal detachment (Figure 2A) together with diffuse, clumped, low-reflective echoes of limited mobility throughout the vitreous cavity, consistent with dense vitreous hemorrhage (Figure 2B). Baseline systemic laboratory parameters of the patient, including a complete blood count, coagulation profile, and blood glucose levels, were within normal limits.

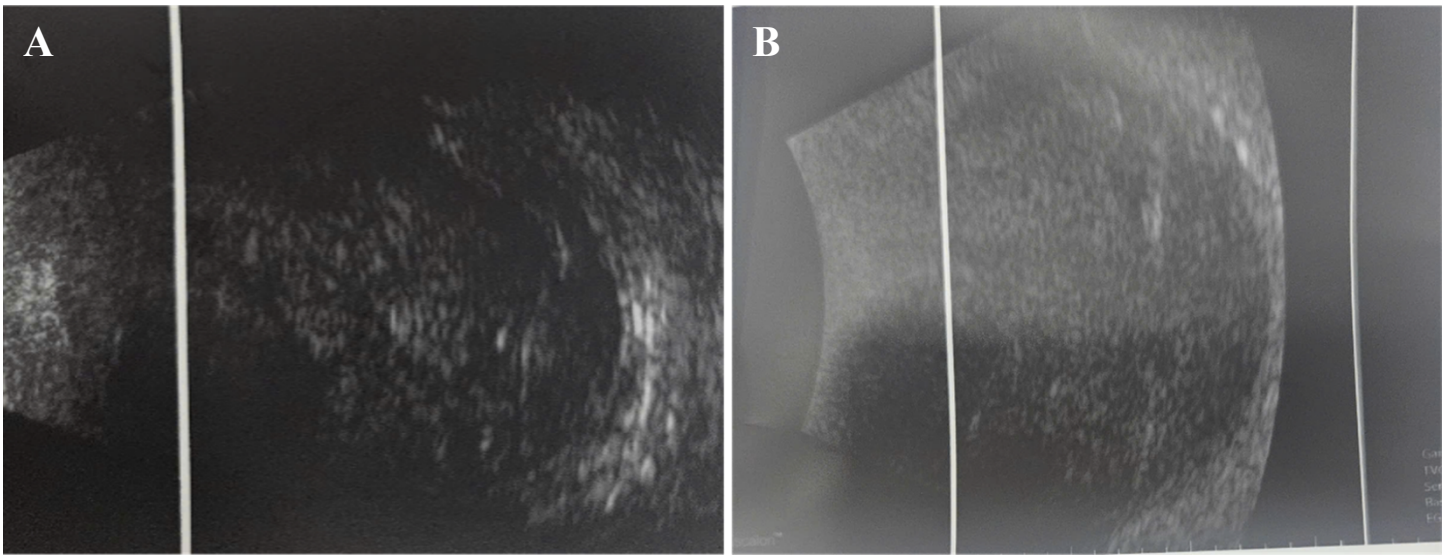


Figure 2. B-scan ultrasonography of the right eye. A. Temporal retinal detachment was noted. B. Dense vitreous hemorrhage occupying the entire vitreous cavity.

Additional treatment included oral methylprednisolone 16 mg daily, topical prednisolone acetate 1% six times daily, and atropine 1% twice daily in the right eye. After corneal clearing and improved eye opening, gonioscopy demonstrated 360° angle closure. Pars plana vitrectomy was considered but was not pursued because the affected eye had no light perception, the potential for visual recovery was considered negligible, and the patient and his family preferred management focused on pain relief and globe preservation. Because the IOP and ocular pain remained refractory to maximal medical therapy, transscleral diode cyclophotocoagulation was performed. The same medical regimen was continued after cyclophotocoagulation. After more than 12 days of hospitalization, right ocular pain was markedly reduced, although visual acuity remained no light perception and IOP was 60 mmHg. Other ophthalmic findings were unchanged (Figures 3A-B). Once adequate pain relief was achieved, the patient was discharged.

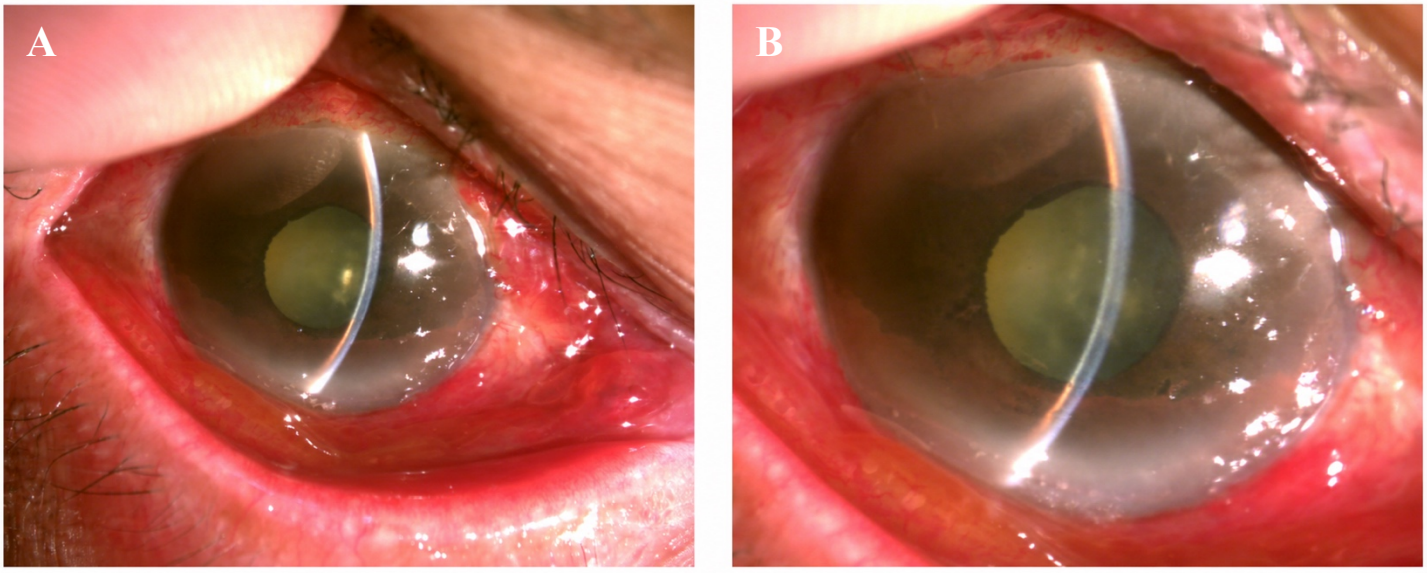


Figure 3. Slit-lamp examination of the right eye 12 days after cyclophotocoagulation. A. The conjunctiva has less swelling, and the corneal edema has improved. B. The anterior chamber remained extremely shallow, with marked anterior displacement of the lens–iris diaphragm nearly apposing the posterior corneal surface.

Discussion

AAC most commonly results from primary pupillary block; however, alternative mechanisms should be considered in atypical presentations.[1] In this patient, acute ocular pain, markedly elevated intraocular pressure, no light perception, and diffuse anterior chamber shallowing—with the lens–iris diaphragm nearly apposing the posterior corneal surface—raised concern for a secondary mechanism. No iris neovascularization was observed, and the open angle in the fellow eye made bilateral primary anatomic predisposition less likely. Gonioscopy confirming 360° angle closure was obtained after corneal clearing. B-scan ultrasonography showed dense vitreous hemorrhage throughout the vitreous cavity and findings suggestive of a superotemporal retinal detachment. These posterior-segment findings were present in the eye with acute angle closure; however, their causal relationship to the marked anterior displacement of the lens–iris diaphragm remains unproven.

Secondary AAC has been reported in association with massive subretinal or suprachoroidal hemorrhage and, more rarely, dense vitreous hemorrhage.[4] Reported predisposing factors include nAMD, polypoidal choroidal vasculopathy, retinal vascular abnormalities, systemic hypertension, anticoagulant use, and coagulopathy.[5] In our patient, active neovascular disease in the affected eye could not be directly confirmed because posterior-segment visualization was obscured and optical coherence tomography (OCT) or angiographic assessment was unavailable. Nevertheless, given the previous history of nAMD and the absence of trauma, diabetes, anticoagulant use, a Valsalva trigger, or laboratory evidence of coagulopathy, nAMD-related hemorrhage was considered the most likely, although presumptive, etiology.

Massive posterior-segment hemorrhage, particularly subretinal or suprachoroidal hemorrhage, has been reported to produce anterior displacement of the lens–iris diaphragm and secondary angle closure.[4-6] B-scan ultrasonography can help identify posterior-segment abnormalities in atypical presentations.[7] In the present case, the available images suggested a relatively shallow retinal detachment and showed vitreous hemorrhage,

but did not demonstrate sufficient posterior-segment expansion to account for the degree of anterior chamber shallowing. Therefore, the concurrent vitreous hemorrhage and suspected retinal detachment should be described as associated findings rather than established causes of the acute angle closure.

The mechanism of angle closure in this case remains undetermined. The available B-scan images were digitized from paper printouts obtained using an older-generation ultrasound device. Although the original dynamic examination was interpreted as showing dense vitreous hemorrhage and findings suggestive of retinal detachment, the quality of the archived static images limits independent assessment. No choroidal detachment was identified on the available images. However, the available evaluation cannot reliably exclude an occult ciliochoroidal effusion or other posterior-pushing process, and neither the vitreous hemorrhage nor the suspected shallow retinal detachment independently explains the nearly flat anterior chamber. Slit-lamp examination showed grade 3 nuclear opalescence without clinically evident lens intumescence, total white opacity, or subluxation, making a phacomorphic component less likely on clinical examination. Ultrasound biomicroscopy and anterior-segment OCT were not performed; consequently, the configuration of the ciliary body and anterior segment could not be characterized fully, and alternative mechanisms, including a lens-related contribution, cannot be definitively excluded. Given the absence of light perception and the palliative treatment goal, additional imaging was not expected to alter immediate management.

Uveal melanoma may also present with subretinal or vitreous hemorrhage and secondary angle closure.[8,9] On B-scan ultrasonography, it may appear as a dome- or mushroom-shaped intraocular mass, while computed tomography or magnetic resonance imaging can provide further characterization. Although no intraocular mass was identified in our patient, the absence of advanced orbital imaging or histopathologic examination prevented its definitive exclusion.

Alternative reported etiologies of acute angle closure associated with vitreous hemorrhage include Valsalva-induced hemorrhage and anti-coagulation bleeding.[6,9] In our patient, however, the onset was entirely spontaneous. The patient had no history of anticoagulant therapy or underlying coagulopathy, which was further supported by normal baseline hematological parameters.

Management of hemorrhage-associated secondary AAC remains challenging because of its rarity and variable clinical course.[6,10] Medical intraocular pressure–lowering therapy may be insufficient in this condition when angle closure is driven by a posterior-pushing mechanism.[5,9,11] Systemic and topical corticosteroids may also be used to control inflammation and pain.[6]

Consequently, commonly employed interventions include cyclophotocoagulation.[11] A few authors have reported attempts at surgical drainage of hemorrhage with anterior chamber reformation;[9] however, outcomes have been limited, with only a single reported case achieving visual acuity of 6/24 after drainage.[9] If these measures fail to relieve pain, enucleation remains the final therapeutic option.[5,8]

In our case, the patient was initially treated with maximal medical therapy for IOP reduction and both systemic and topical corticosteroid therapy. Because visual acuity was no light perception and the likelihood of visual recovery was extremely poor, pars plana vitrectomy was considered but was not pursued. Furthermore, given the extremely high intraocular pressure, any major intraocular surgery would expose the patient to severe ocular complications such as spontaneous choroidal hemorrhage, while offering negligible visual benefits. Additionally, there remained a high risk of recurrent vitreous hemorrhage from fragile neovascularization associated with nAMD. Weighing these heavy surgical hazards and the risk of re-bleeding against the minimal visual gain, subsequently the cyclophotocoagulation was performed for pain control and globe preservation,

which was also aligned with the patient's treatment preferences. Although the IOP was 60 mmHg at discharge, ocular pain was substantially relieved.

Conclusion

This case describes acute angle closure occurring in an eye with dense vitreous hemorrhage and ultrasonographic findings suggestive of retinal detachment in a patient with a history of neovascular age-related macular degeneration. Although these findings occurred together, a causal relationship was not established, and the mechanism responsible for the profound anterior chamber shallowing remains uncertain. B-scan ultrasonography is valuable for identifying coexisting posterior-segment abnormalities in atypical presentations. When visual potential is absent, management may appropriately prioritize pain relief and globe preservation. In this patient, cyclophotocoagulation substantially reduced ocular pain despite persistently elevated intraocular pressure.

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Statement of Ethics

This case report adheres to patient confidentiality and ethical principles in accordance with the guidelines of the Declaration of Helsinki and relevant local regulations.

Patient Consent and Privacy

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Conflict of Interest Statement

The authors have no conflict of interest to disclose.

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