CASE REPORT

# Acute angle closure glaucoma secondary to vitreous haemorrhage in neovascular age-related macular degeneration: A rare complication

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## SUMMARY

**Acute angle closure glaucoma (AACG) secondary to vitreous haemorrhage is a rare but devastating complication of neovascular age-related macular degeneration (AMD). We report a visually devastating case of AACG in a 60-year-old gentleman with multiple systemic comorbidities. He initially presented with a central scotoma in the left eye and was diagnosed with bilateral AMD with subretinal fibrosis in the left eye. After being lost to follow up, he returned with a new submacular haemorrhage in the right eye, for which intravitreal anti-vascular endothelial growth factor (VEGF) therapy was planned. However, before treatment initiation, he developed sudden painful vision loss, elevated intraocular pressure (IOP) and signs consistent with secondary AACG. Imaging confirmed a massive vitreous haemorrhage with no evidence of intraocular malignancy. Despite aggressive medical therapy, the patient's vision deteriorated rapidly to no light perception, necessitating cyclodiode laser treatment for pain control. This case illustrates the rare but serious complication of vitreous haemorrhage in neovascular AMD with associated risk factors emphasizing the importance of early recognition and intervention to prevent secondary AACG and permanent vision loss.**

## INTRODUCTION

Age-related macular degeneration (AMD) is a leading cause of severe visual impairment in the elderly.[1] It typically begins with the accumulation of drusen in the macula and may progress to advanced stages, characterized by either neovascular AMD or non-neovascular AMD. In neovascular AMD, abnormal vessel growth often leads to subretinal haemorrhage and other exudative complications. Although vitreous haemorrhage is a known consequence of neovascular AMD, secondary acute angle closure glaucoma (AACG) is an exceedingly rare complication.[2]

In such cases, extensive vitreous haemorrhage can cause forward displacement of the lens–iris diaphragm, resulting in occlusion of the anterior chamber angle and a rapid rise in intraocular pressure (IOP), ultimately precipitating AACG. This acute presentation represents an ophthalmic emergency requiring immediate intervention to prevent irreversible vision loss. Here we report a rare presentation of AACG secondary to vitreous haemorrhage in neovascular AMD, aiming to enhance clinical awareness of this uncommon complication by emphasising its underlying pathophysiology, associated risk factors and the necessity of prompt recognition to avoid diagnostic delay and irreversible vision loss.

## CASE PRESENTATION

A 60-year-old gentleman with a history of intravenous drug use, alcohol consumption and multiple comorbidities including diabetes mellitus, hypertension, hyperlipidaemia, iron deficiency anaemia and hepatitis C presented with a three-month history of central scotoma in the left eye. He denied any metamorphopsia, eye pain or history of trauma. Examination revealed a best corrected visual acuity (BCVA) of 6/9 in the right eye and 6/36 in the left eye.

Fundus examination of both eyes revealed macular drusen and subretinal fibrosis in the left eye without retinal haemorrhage. Optical coherence tomography (OCT) showed drusen in both eyes without subretinal fluid, as well as retinal pigment epithelium (RPE) atrophy in the left eye. These findings were consistent with bilateral AMD, with advanced disease and subretinal fibrosis in the left eye. A one month follow up was advised for monitoring, however the patient did not attend the scheduled visit.

Three months later, the patient returned with complaints of bilateral eye metamorphopsia. Best-corrected visual acuity (BCVA) had declined to 6/24 in the right eye and remained at 6/36 in the left eye. Fundus examination of the right eye revealed a submacular haemorrhage approximately three disc diameters in size, located inferotemporally within the vascular arcade, along with extensive subretinal drusen. The right eye showed no signs of diabetic retinopathy, while the left eye demonstrated mild non-proliferative changes, with no other significant differences from previous findings (Figure 1). OCT demonstrated multiple haemorrhagic PED and subretinal fluid (SRF) in the right eye (Figure 2). The diagnosis of neovascular AMD was made clinically based on funduscopic and OCT findings, as FFA and ICG were not performed for this patient. A treatment plan was initiated,

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**Table 1: Summary of published case report on acute angle closure in neovascular AMD/ PCV**

| Author (year) | Age (year) | Primary pathology | Mechanism of acute angle closure | VA & IOP (mmHg) | Risk factors | B-scan findings | Treatment (besides medication) | Final outcomes |
|---|---|---|---|---|---|---|---|---|
| Chen et al.[6] (2000) (case 1) | 57 | nAMD | Bullous haemorrhagic RD | HM, 67 | DM, HPT, HPL, CVA | Massive subretinal hematoma, bullous haemorrhagic RD | - | Phthisical eye |
| Chen et al.[6] (2000) (case 2) | 78 | nAMD | Diffuse subretinal / choroidal haemorrhage | NPL, 50 | Coronary artery disease (post CABG) | Haemorrhagic RD temporo-inferior | Sclerotomy and blood drainage and AC | Phthisical eye |
| Chen et al.[6] (2000) (case 3) | 55 | nAMD | Haemorrhagic RD | CF, 42 | HPT, HPL, hyperglycaemia | Haemorrhagic RD temporo-inferior | reformation Sclerotomy and drainage | VA HM |
| Chen et al.[6] (2000) (case 4) | 67 | nAMD | Total haemorrhagic RD | Not documented, 70 | DM, HPT, HEP C | Diffuse subretinal haemorrhage with total RD | Sclerotomy and AC reformation | Phthisical eye |
| Baskaran et al.[8] (2017) (case 1) | 67 | PCV | Annular haemorrhagic CD | NPL, 50 | DM, HPT | Annular haemorrhagic CD with “kissing choroids” | CPC diode laser | Phthisical eye |
| Baskaran et al.[8] (2017) (case 2) | 71 | PCV | Annular haemorrhagic CD | NPL, 40 | DM, HPT | Annular haemorrhagic CD with “kissing choroids” | CPC diode laser | Phthisical eye |
| Jersey et al.[4] (2020) | 80 | nAMD | VH | NPL, 74 | Post IVT injection | VH, severe narrowing of anterior chamber angle | - | VA NPL |

nAMD; neovascular age-related macular degeneration; PCV, polypoidal choroidal vasculopathy; DM, diabetes mellitus; HPT, hypertension; HEP C, hepatitis C; HPL, hyperlipidaemia; CVA, cerebrovascular accident; CABG, coronary artery bypass graft; AC, anterior chamber; RD, retinal detachment; VH, vitreous haemorrhage; VA, Visual acuity; CF, counting finger; HM, hand movement; NPL, non-perception to light; CPC, cyclophotocoagulation

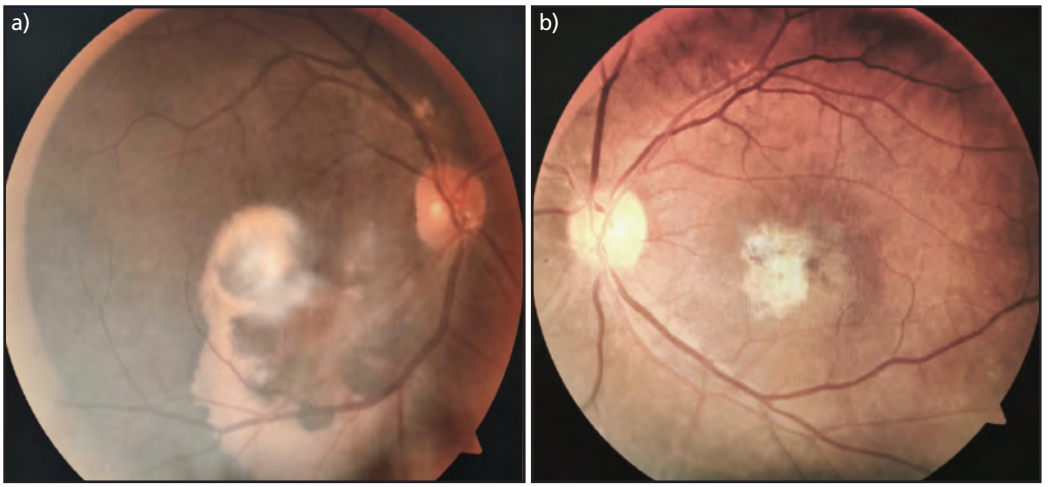


**Fig. 1:** a) Right eye fundus showed a submacular haemorrhage inferotemporally with extensive subretinal drusen
b) Left eye fundus showed macular subretinal fibrosis and dot haemorrhage at inferonasal area

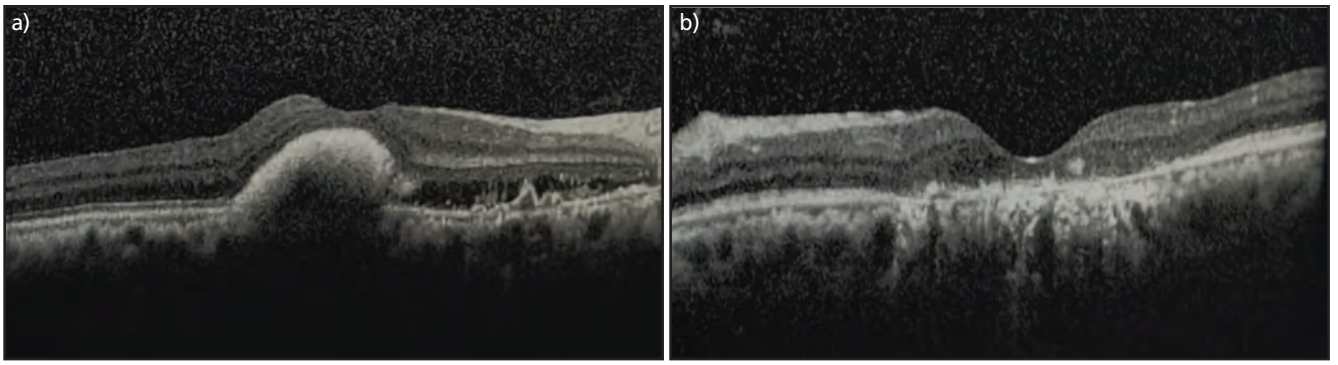


**Fig. 2:** a) Right eye OCT showed haemorrhagic PED, multiple drusen and SRF
b) Left eye OCT showed outer retinal layer atrophy

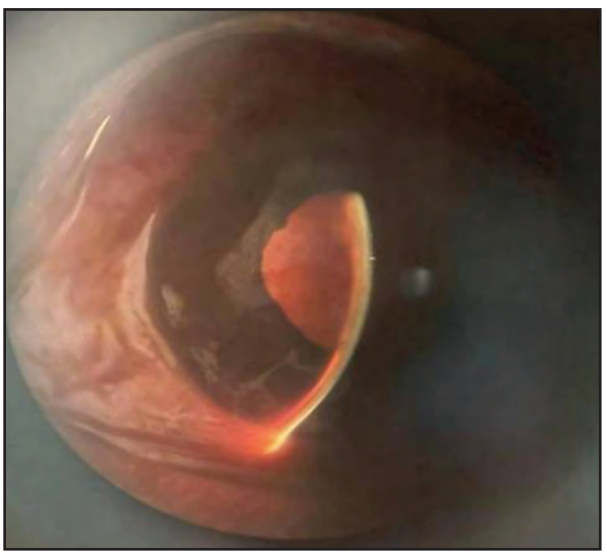

**Fig. 3:** Slit lamp photograph of the anterior segment of the right eye shows a flat anterior chamber with iridolenticular touch to the cornea

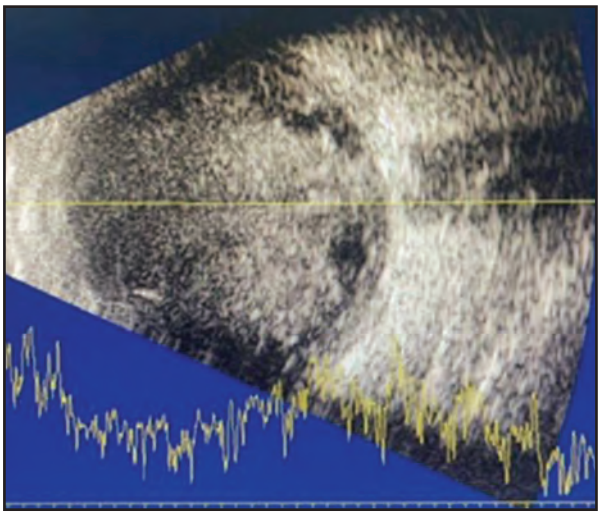

**Fig. 4:** B-scan ultrasound of the right eye shows dense, diffuse vitreous haemorrhage with no evidence of a retrolenticular mass

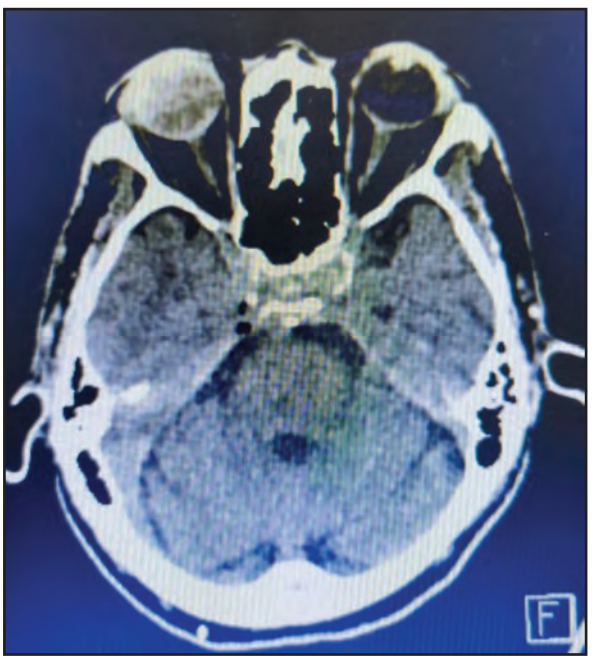

**Fig. 5:** CT scan of the orbit in the axial plane showed a diffuse heterogeneous hyperdensity within the vitreous chamber of the right eye, suggestive of vitreous haemorrhage

consisting of monthly intravitreal anti-vascular endothelial growth factor (VEGF) injections for the right eye over a three month period.

Two months later, the patient was scheduled for his first intravitreal injection. However, one week before the procedure, he developed a three day history of acute onset painful redness in the right eye, progressive visual loss, headache, nausea, and vomiting. He denied any recent ocular trauma or procedures performed outside the institution. On examination, BCVA was hand movement in the right eye and 6/36 in the left eye. A positive relative afferent pupillary defect (RAPD) was noted in the right eye. The cornea was edematous, the anterior chamber was shallow with endothelial touch and the pupil was mid-dilated, fixed and non-reactive (Figure 3). Fundus visualization in the right eye was obscured by a dense, reddish retrolental hue. The left fundus remained unchanged from previous examinations. IOP was markedly elevated at 50 mmHg in the right eye and 14 mmHg in the left eye. Gonioscopy evaluation demonstrated closed angles in the right eye and open angles in the left, with no signs of rubeosis iridis or peripheral anterior synechiae.

B-scan ultrasonography showed a large hyperechogenic shadow in the posterior chamber of the right eye suggestive of vitreous haemorrhage without evidence of an intraocular mass (Figure 4). CT imaging of the orbit ruled out intraocular malignancy (Figure 5). The patient was admitted for IOP control and managed with a combination of a single stat

dose of intravenous acetazolamide 500 mg, oral acetazolamide 250 mg four times daily, topical latanoprost 0.005% at night, topical timolol 0.25% twice daily, topical dorzolamide 2% three times daily, topical brimonidine 0.15% three times daily, topical dexamethasone 0.1% every four hours and oral glycerol syrup 30 mL three times daily during the ward stay. Oral celecoxib 200 mg twice daily and oral paracetamol 1 g four times daily were prescribed for pain control. Despite these interventions, the patient experienced persistent pain and high IOP, rapid progression to no light perception vision over the next day of admission and worsening corneal edema. As the patient's vision had declined to no light perception with no potential for recovery, surgical intervention was not indicated. Transscleral cyclodiode photocoagulation (TSCPC) was performed to control intraocular pressure and alleviate ocular pain, resulting in marked symptomatic improvement. However, the right eye subsequently progressed to phthisis on further follow-up. This case highlights the progression of neovascular AMD with secondary complications, AACG from vitreous haemorrhage causing in poor visual outcomes despite timely interventions.

## DISCUSSION

Massive intraocular haemorrhage leading to AACG is a rare sequela of neovascular AMD and is often associated with at least one predisposing factor, such as oral anticoagulant use, blood dyscrasia, or systemic hypertension.[3] Although the exact mechanisms leading to vitreous haemorrhage in neovascular AMD remain unclear, breakthrough vitreous haemorrhage is reported more frequently in polypoidal choroidal vasculopathy than in exudative AMD.[4]

Previous studies have identified histologically that disciform scars with choroidal neovascularization in AMD as the primary source of haemorrhage in most cases.[3,5] When these abnormal vessels extend into fibrous scars, mechanical stress increases the risk of rupture, producing massive haemorrhages that penetrate all retinal layers and extend into the vitreous. The vitreous haemorrhage causes increased pressure in the posterior chamber, leading to forward displacement of the iris-lens diaphragm. This forward shift results in sufficient iris displacement to obstruct aqueous outflow in the anterior chamber, ultimately culminating in acute angle closure glaucoma.

Unlike suprachoroidal haemorrhage, which often occurs during hypotony, surgery or rarely from anticoagulation, bleeding in neovascular AMD typically follows mechanical disruption of choroidal neovascular membranes and often preceded by submacular haemorrhage.[3,5] Clinical observations support this mechanism. Chen et al., in their follow-up of two cases of neovascular AMD with submacular haemorrhage and a case with a disciform scar reported that severe eye pain and elevated intraocular pressure developed 2–4 weeks after the onset of rapidly progressive visual blurring and visual field defects.[6]

As demonstrated in this case, the patient had a documented submacular haemorrhage prior to the onset of vitreous haemorrhage, during which a course of three consecutive intravitreal injections was planned. This approach is consistent with current evidence indicating that anti-VEGF therapy either alone or in combination with pneumatic displacement using intravitreal tissue plasminogen activator (tPA) is generally preferred for medium-sized submacular haemorrhages.[7] Monotherapy was selected in this instance as it is less invasive, well-tolerated, and effective in suppressing choroidal neovascular membrane activity.[7]

Impaired local haemostasis is a recognized predisposing factor for massive intraocular haemorrhage in macular degeneration, particularly among patients taking anticoagulants such as warfarin or NSAIDs, or those with systemic conditions like diabetes mellitus.[3,6,8] In this case, the vitreous haemorrhage may have been influenced by the patient's systemic comorbidities particularly diabetes mellitus, anaemia and chronic hepatitis C which are associated with increased vascular fragility. Although the patient was not receiving anticoagulant therapy, microvascular compromise due to diabetes and systemic hypertension likely contributed to the haemorrhagic event. Additional systemic risk factors, such as advanced age and arteriolosclerosis, may further increase vascular fragility, rendering vessels more susceptible to mechanical shearing forces.[8] A summary of cases and associated risk factors reported in AACG secondary to neovascular AMD or related pathologies, such as polypoidal choroidal vasculopathy (PCV), is shown in Table I.

Management of AACG secondary to massive subretinal or vitreous haemorrhage remains a significant clinical challenge, often associated with poor outcomes. Medical therapy alone is frequently inadequate for controlling IOP and many cases reported in the literature have required enucleation or cyclodestructive procedures to relieve intractable pain.[5,6,8] In the present case, conservative treatment failed to control IOP, necessitating cyclophotocoagulation for pain management. Although timely surgical interventions such as sclerotomy, haemorrhage drainage or anterior chamber reformation may reduce IOP and provide symptomatic relief, these procedures rarely restore useful vision.[5,6,8] Importantly, the timing of surgical intervention appears to play a critical role in outcomes. Prolonged elevation of IOP can result in irreversible ischemic damage to intraocular tissues and lead to phthisis bulbi. In contrast, early intervention aimed at decompressing the globe and restoring IOP may help preserve ocular structure and alleviate pain, although visual recovery remains limited.[5] Therefore, in cases where IOP is refractory to medical management, prompt surgical consideration is essential not with the aim of restoring vision, but to prevent irreversible structural damage and improve patient comfort.

## CONCLUSION

Acute angle closure glaucoma secondary to vitreous haemorrhage is a rare but vision threatening complication of neovascular AMD. This case highlights the importance of regular follow up, recognition of systemic and ocular risk factors and early intervention to mitigate pain and preserve ocular integrity even when visual recovery is limited.

## ACKNOWLEDGEMENTS
None

## DECLARATIONS
Written consent was obtained from the patient including the use of anonymized medical data and images for the publication of this case report, ensuring compliance with ethical standards.

## CONFLICT OF INTEREST
None declared.

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