

Early Recognition and Timely Cataract Surgery Are Critical to Prevent Irreversible Optic Nerve Damage in Acute Primary Angle Closure

Errorry Ramadiansyah¹, Rika Agustanti¹

¹Department of Ophthalmology, Rumah Sakit Mata Masyarakat Jawa Timur, Surabaya, Indonesia

Correspondence: **Errorry Ramadiansyah**: Jl. Ketintang Baru Selatan I No.1, Surabaya, Indonesia; erroryramadiansyah21@gmail.com

ABSTRACT

Acute primary angle closure represents an ophthalmic emergency, and this forms the basis of the introduction of the case, as the condition can cause a sudden and severe rise in intraocular pressure leading to rapid and irreversible optic nerve damage when diagnosis and treatment are delayed. The objective of this report is to describe the clinical presentation, diagnostic evaluation, and management of acute primary angle closure occurring together with an immature senile cataract in a patient treated at Mata Masyarakat Hospital, Surabaya, Indonesia. The methods applied in this case involve a descriptive case report approach that outlines the patient's clinical course, examination findings, and therapeutic interventions. The results of the clinical evaluation showed that the patient experienced progressive visual decline, ocular pain, redness, headache, and halos in the left eye. Examination revealed markedly elevated intraocular pressure of 49.5 mmHg, corneal edema, a shallow anterior chamber, a mid-dilated pupil, glaukomflecken, and thinning of the retinal nerve fiber layer with neuroretinal rim loss. The diagnosis of acute primary angle closure with an immature senile cataract was established, while the fellow eye was categorized as a primary angle closure suspect. After surgical management, intraocular pressure decreased and visual acuity improved partially, although structural optic nerve damage remained irreversible. In conclusion, this case illustrates the multifactorial nature of angle-closure disease and emphasizes the importance of early recognition, multimodal assessment, and timely surgical intervention to prevent permanent glaucomatous optic neuropathy.

Keywords: angle closure glaucoma; intraocular pressure; cataract; optic nerve diseases; phacoemulsification

INTRODUCTION

Acute primary angle closure (APAC) glaucoma represents one of the most critical ocular emergencies encountered in clinical ophthalmology, characterized by a sudden, marked, and often dramatic elevation of intraocular pressure (IOP) that can rapidly threaten visual function if not promptly recognized and treated [1]. The abrupt nature of this condition means that irreversible optic nerve damage may occur within a very short time window, making APAC a true ophthalmic emergency requiring immediate intervention. From a global perspective, glaucoma remains a major cause of irreversible blindness, affecting an estimated 76 million individuals worldwide, with projections suggesting that this number may rise to 112 million by 2040 as populations age and life expectancy increases [2]. Within this global burden, angle-closure glaucoma accounts for approximately 10–20% of all glaucoma cases, yet contributes disproportionately to glaucoma-related visual disability, particularly in Asian populations where anatomical predispositions such as shallow anterior chambers, thicker lenses, and narrow angles are more prevalent [2]. These anatomical characteristics, combined with demographic patterns such as older age distribution and higher prevalence of hyperopia, make APAC a condition of heightened relevance in Asian regions, including Southeast Asia, where population-level susceptibility amplifies the clinical and public health significance of angle-closure disease.

The pathophysiology of APAC involves an abrupt obstruction of aqueous humor outflow at the anterior chamber angle, most commonly precipitated by pupillary block or lens-related mechanisms. In pupillary block, resistance to aqueous flow from the posterior to the anterior chamber increases, causing forward bowing of the peripheral iris and subsequent angle closure. Lens-related mechanisms, including increased lens thickness or anterior lens position, may further narrow the anterior chamber angle and predispose individuals to acute attacks. This obstruction leads to a rapid and substantial elevation in IOP, often reaching levels above 40–60 mmHg, resulting in corneal edema, optic nerve ischemia, and accelerated retinal ganglion cell loss. Clinically, patients typically present with severe ocular pain, blurred vision, halos around lights, conjunctival hyperemia, nausea, vomiting, and mid-dilated pupils; symptoms that reflect both ocular and systemic autonomic responses to extreme IOP elevation. Without timely intervention, APAC can cause permanent optic nerve damage within hours to days, underscoring the urgency of early recognition and rapid therapeutic action.

The presence of coexisting cataract further narrows the anterior chamber angle and may exacerbate the acute attack, complicating both diagnosis and management by intensifying pupillary block and reducing visualization during examination [3]. Cataract progression, particularly in older adults, contributes to lens enlargement and anterior displacement, thereby increasing the risk of angle closure. In many cases, cataract extraction not only restores visual clarity but also deepens the anterior chamber and widens the angle, providing both therapeutic and preventive benefits in APAC management.

Although APAC is well described in the literature, real-world clinical encounters often reveal substantial variability in presentation. Some patients may present with atypical symptoms, mild pain, or intermittent episodes of blurred vision that precede a full-blown attack. Delayed health-seeking behavior, limited access to ophthalmic care, and coexisting lens opacity may obscure early recognition and hinder appropriate intervention. These challenges highlight the importance of reporting individual cases to emphasize diagnostic complexity and refine management strategies in diverse clinical settings [3]. Furthermore, despite its well-recognized features, APAC may present with confounding comorbidities, subtle anatomical variations, or delayed onset that complicate clinical decision-making. For example, patients with plateau iris configuration or phacomorphic tendencies may exhibit overlapping features that require careful differentiation.

Comprehensive assessment integrating slit-lamp examination, tonometry, gonioscopy, and optical coherence tomography (OCT) is essential to identify underlying anatomical contributors and determine the urgency and sequence of therapeutic interventions [4]. Slit-lamp examination allows evaluation of corneal edema, anterior chamber depth, and pupillary abnormalities. Tonometry provides immediate confirmation of elevated IOP. Gonioscopy remains the gold standard for assessing angle status, although it may be challenging during acute attacks due to corneal edema. OCT offers high-resolution imaging of the anterior segment and optic nerve head, enabling clinicians to quantify structural damage and monitor disease progression. In addition, ultrasound biomicroscopy (UBM) may be useful in evaluating ciliary body position, lens vault, and plateau iris configuration, especially in complex or recurrent cases.

Given the rapid progression and potential severity of APAC, timely intervention is critical. Initial management typically includes topical and systemic IOP-lowering medications, hyperosmotic agents, and anti-inflammatory therapy. Definitive treatment often involves laser peripheral iridotomy (LPI) to relieve pupillary block, although its effectiveness may be limited in cases with significant lens-related mechanisms. In such situations, early lens extraction through phacoemulsification may provide both immediate and long-term benefits by deepening the anterior chamber and widening the angle. Surgical intervention is particularly important when cataract contributes significantly to angle narrowing or when optic nerve damage is already evident.

This case report aims to describe the clinical presentation, diagnostic evaluation, and management of a 57-year-old male with acute primary angle closure complicated by an immature senile cataract. By detailing the patient's clinical course, this report underscores the importance of early recognition, multimodal assessment, and timely surgical intervention in preventing irreversible glaucomatous optic neuropathy. The case also highlights the need for heightened clinical vigilance, especially in populations with anatomical predispositions to angle-closure disease, and reinforces the value of integrating structural imaging, gonioscopic evaluation, and lens assessment in guiding therapeutic decisions.

METHODS

This study employed a descriptive case report design to comprehensively document the clinical course, diagnostic evaluation, and management of a patient with acute primary angle closure glaucoma. The case was managed at Mata Masyarakat Hospital, Surabaya, Indonesia, during the period encompassing the patient's initial presentation, acute management, surgical intervention, and postoperative follow-up. All observations and clinical data were collected in real time throughout the continuum of care, ensuring accurate representation of the patient's condition and treatment outcomes.

The subject of this report was a 57-year-old male who presented with symptoms consistent with acute primary angle closure. Data collection involved direct and systematic clinical examination conducted by ophthalmologists on duty. This included detailed history taking to identify symptom onset, progression, and associated systemic manifestations; measurement of visual acuity using standardized Snellen charts; and intraocular pressure assessment using Goldmann applanation tonometry. Slit-lamp biomicroscopy was performed to evaluate corneal clarity, anterior chamber depth, pupillary characteristics, and the presence of glaukomflecken. Gonioscopy was conducted to assess the angle configuration and confirm angle closure, while fundoscopy allowed evaluation of the optic nerve head, including neuroretinal rim status and cup-to-disc ratio.

Advanced imaging modalities were utilized to enhance diagnostic accuracy. Optical coherence tomography was performed to assess anterior chamber configuration, quantify retinal nerve fiber layer thickness, and document structural optic nerve changes. These imaging findings were integrated with clinical observations to establish a comprehensive diagnostic profile. All diagnostic procedures adhered to standard ophthalmic practice guidelines and were conducted using calibrated instruments.

Therapeutic decisions, including administration of intraocular pressure-lowering medications, anti-inflammatory agents, and hyperosmotic therapy were made according to established clinical protocols for acute primary angle closure. Following stabilization of intraocular pressure, the patient underwent phacoemulsification with intraocular lens implantation as definitive management, given the presence of an immature senile cataract contributing to anterior chamber narrowing. No experimental treatments or investigational drugs were used at any stage of care. All surgical procedures were performed by experienced ophthalmic surgeons following standard operative protocols.

Clinical findings, imaging results, intraoperative observations, and postoperative outcomes were systematically documented in the patient's medical record. Data analysis for this case report was descriptive in nature, focusing on temporal progression of symptoms, changes in intraocular pressure, structural optic nerve parameters, and functional visual outcomes. The analysis aimed to illustrate the relationship between anatomical predisposition, acute pathophysiology, and therapeutic response in the context of angle-closure disease.

RESULTS

A 57-year-old male was referred from a secondary hospital with a three-week history of progressive vision loss in the left eye. During this period, he noted gradually worsening visual acuity accompanied by ocular pain, redness, headache, and rainbow-colored halos around lights. He denied nausea or vomiting. He also reported frequent stumbling and bumping into objects, suggesting impaired peripheral vision. The right eye was asymptomatic, with no visual disturbance or discomfort.

The patient had no known systemic diseases such as diabetes mellitus or hypertension and denied any history of ocular trauma. He was a presbyopic reader using plus lenses. Family history was notable for progressive bilateral vision loss in an older sibling. He also reported frequent use of traditional herbal medicines and supplements.

Before referral, the patient had been treated with Timolol Maleate, Tobramycin-Dexamethasone combination drops, and oral Acetazolamide by the secondary facility. On arrival, his blood pressure measured 158/83 mmHg, and the remainder of the general physical examination was unremarkable.

Based on Figure 1, Table 1, Table 2 and Table 3, ophthalmic evaluation revealed reduced visual acuity in the left eye, with conjunctival hyperemia, corneal edema, a shallow anterior chamber, and a mid-dilated pupil. Glaukomflecken was observed on slit-lamp examination.

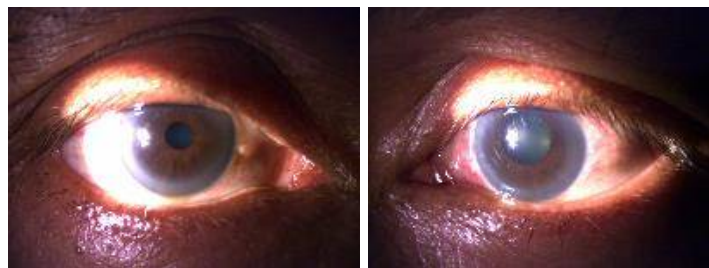


Figure 1. Clinical features of the patient's right and left eyes

Table 1. Ophthalmological status

OD	Examination	OS
6/7.5 ph - 15.0	Visus TIO	3/60 ph - 49.5

Table 2. Examination of the anterior segment of the eye

OD	Anterior segment	OS
Mass (-), edema (-)	Palpebra	Mass (-), edema (-)
Hiperemi (-)	Conjungtiva	Hiperemi (+)
Edema (-)	Cornea	Edema (+)
Shallow, VH II	Front eye chamber	Shallow, VH I - II
Radier	Iris	Radier
Round, RC (+)	Pupil	Mid dilatation, decreased RC
Clear	Lens	Cloudy, Glaukomflecken (+)

Based on Figure 2, Figure 3 and Figure 4, optical coherence tomography (OCT) demonstrated retinal nerve fiber layer (RNFL) thinning in the left eye and normal RNFL thickness in the right eye. Optic nerve head assessment showed neuroretinal rim thinning with an enlarged cup-to-disc ratio on the affected side. Based on these findings, the patient was diagnosed with acute primary angle closure (OS), immature senile cataract (OS), and primary angle closure suspect (OD). He was treated with Timolol Maleate 0.5% (2x/day OS), Tobramycin–Dexamethasone (6x/day OS), Sodium–Potassium chloride drops (6x/day OU), oral Acetazolamide (3x/day), oral Potassium chloride (1x/day), and scheduled for phacoemulsification with intraocular lens implantation (OS).

Table 3. Examination of the posterior segment of the eye

OD	Posterior segment	OS
Positive	Reflex fundus	Positive
Firm borders (+), normal colors (+), C/D Ratio 0.5, lying (-), bayoneting (-), nasalization (-)	Papil nervus II	C/D Ratio 0.6 – 0.7, GON (+)
Bleeding (-), exudate (-)	Retina	Bleeding (-), exudate (-)
Positive	Reflex macula	Positive

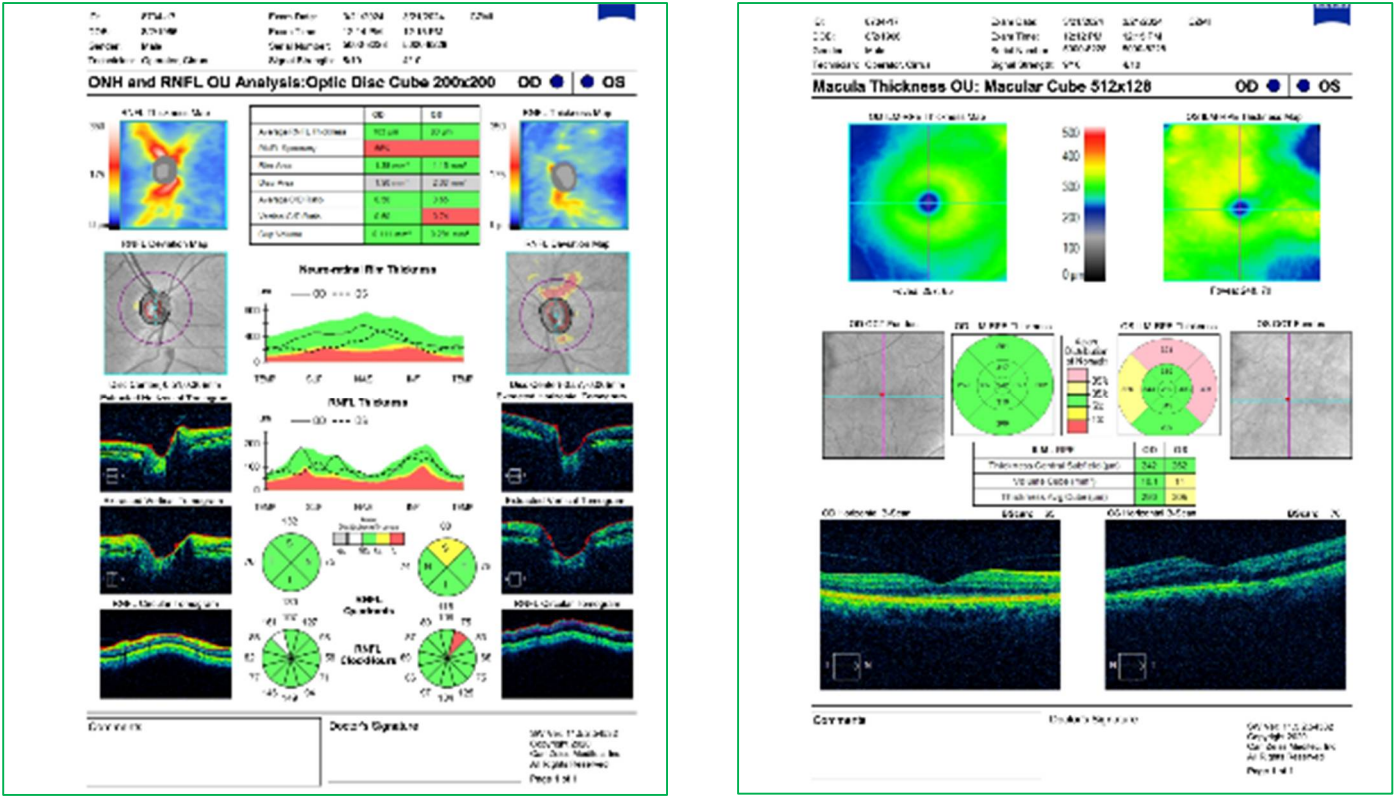


Figure 2. OCT examination

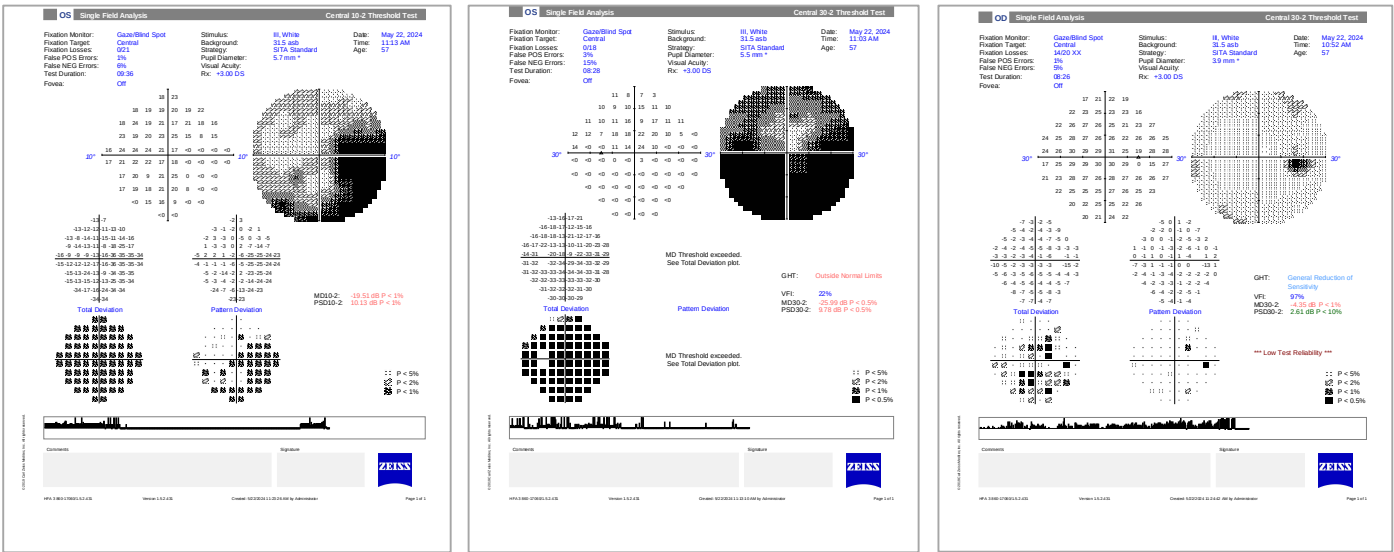


Figure 3. HFA Examination

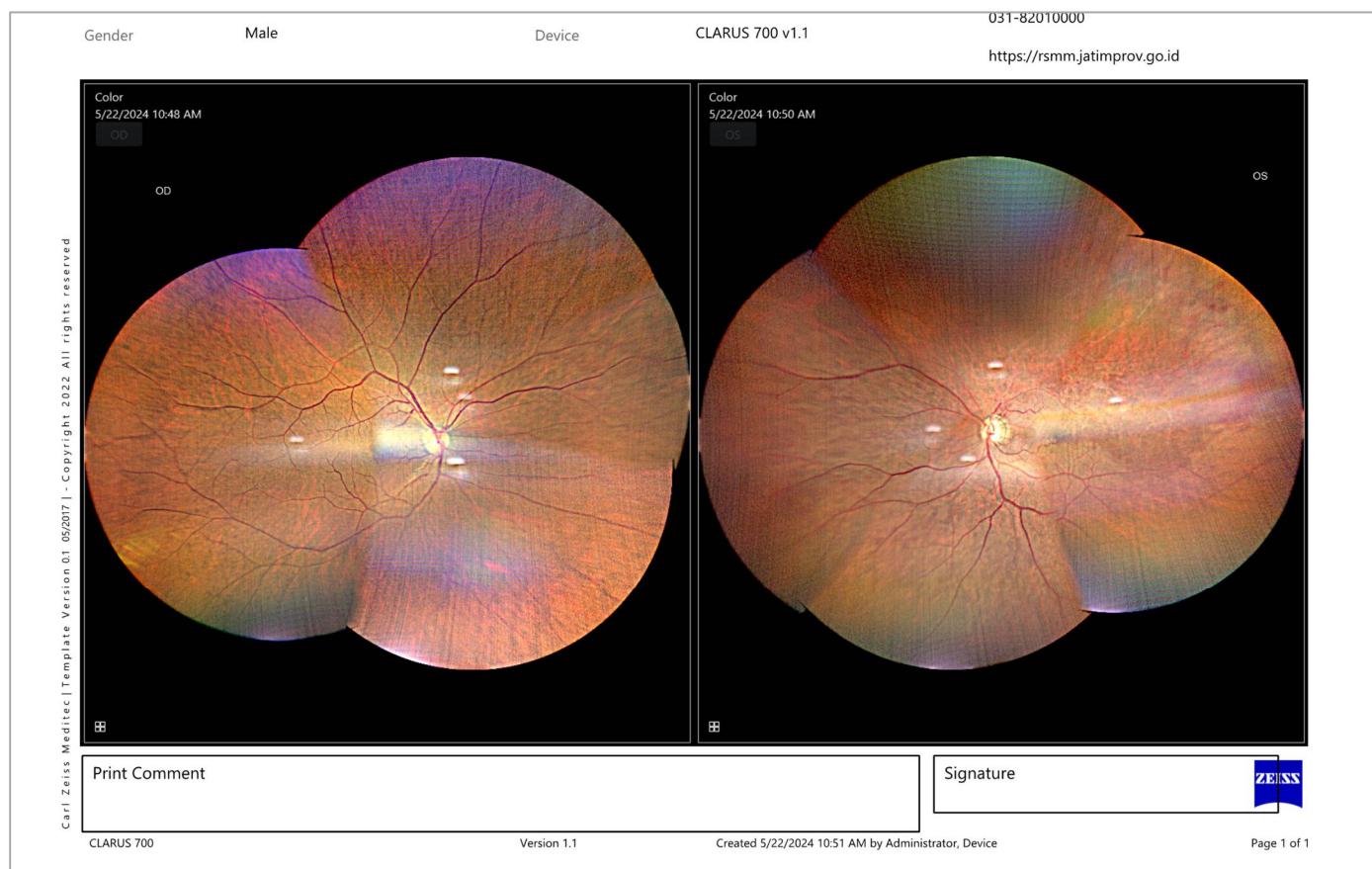


Figure 4. Fundus examination

Following surgery, postoperative follow-up was conducted, and the corresponding examination findings were recorded (Figure 5, Table 4, Table 5 and Table 6).

DISCUSSION

The clinical presentation of this 57-year-old male illustrates the typical yet inherently complex features of acute primary angle closure (APAC), a condition known for its abrupt onset and rapid progression toward irreversible optic nerve damage. The patient's constellation of symptoms; progressive vision loss, ocular pain, conjunctival hyperemia, corneal edema, and halos aligns closely with the classical manifestations of acute angle-closure glaucoma. Although nausea and vomiting are frequently reported in severe APAC due to trigeminal and vagal autonomic activation, their absence in this case highlights the variability of systemic manifestations among individuals and reinforces that APAC cannot be excluded solely based on the absence of systemic symptoms. The patient's difficulty navigating his environment and frequent stumbling further reflect functional impairment resulting from reduced peripheral vision, a phenomenon often associated with early optic nerve compromise and visual field constriction.

These functional deficits correspond with structural abnormalities documented through optical coherence tomography (OCT), which demonstrated retinal nerve fiber layer (RNFL) thinning and neuroretinal rim loss. Such findings are hallmark indicators of glaucomatous optic neuropathy, where elevated intraocular pressure (IOP) induces mechanical deformation of the lamina cribrosa, axonal compression, and subsequent retinal ganglion cell apoptosis. The presence of glaukomflecken; pathognomonic anterior lens epithelial necrosis further supports the diagnosis of APAC and indicates that the patient experienced a sufficiently severe IOP spike to cause ischemic damage to the lens epithelium.

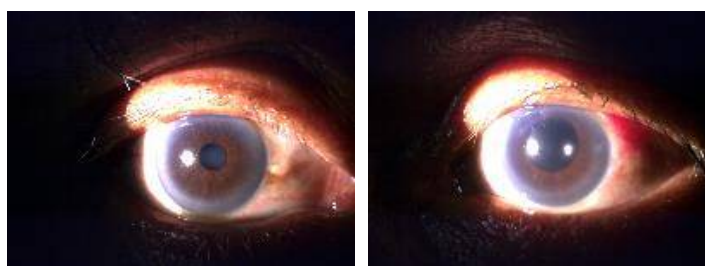


Figure 5. Clinical features of the patient's right and left eyes post operation

Table 4. Post operation ophthalmological status

OD	Examination	OS
6/12	Visus	4/60
15.3	TIO	52.3

Table 5. Post Operation Examination of the anterior segment of the eye

OD	Anterior segment	OS
Mass (-), edema (-)	Palpebra	Mass (-), edema (-)
Hiperemi (-)	Conjungtiva	Hiperemi (+)
Edema (-)	Cornea	Edema (+)
Shallow, VH II	Front eye chamber	Shallow, VH I - II
Radier	Iris	Radier
Round, RC(+)	Pupil	Mid dilatation, decreased RC
Clear	Lens	Clear, IOL (+)

Table 6. Post Operation Examination of the posterior segment of the eye

OD	Posterior segment	OS
Positive	Reflex fundus	Positive
C/D Ratio 0.5, GON (-)	Papil nervus II	C/D Ratio 0.7 – 0.8, GON (+)
Bleeding (-), exudate (-)	Retina	Bleeding (-), exudate (-)
Positive	Reflex macula	Positive

A positive family history of vision loss in an older sibling raises the possibility of hereditary susceptibility to glaucoma. Genetic factors have increasingly been recognized as contributors to angle-closure mechanisms, with variations in loci associated with anterior chamber morphology, iris thickness, and trabecular meshwork resistance implicated in predisposing individuals to angle-closure disease. Although genetic testing was not performed in this case, the patient's presentation underscores the importance of family screening and early ophthalmic evaluation among individuals with similar symptoms or anatomical predispositions [5]. In populations with high prevalence of angle-closure disease, such as those in East and Southeast Asia, family history may serve as an important risk stratification tool.

The diagnosis of APAC in this patient was supported by hallmark clinical findings, including markedly elevated intraocular pressure (49.5 mmHg), a shallow anterior chamber, a mid-dilated nonreactive pupil, glaukomflecken, and an increased cup-to-disc ratio. OCT imaging further corroborated these findings by demonstrating RNFL thinning, neuroretinal rim reduction, and a vertical cup-to-disc ratio of 0.74. These structural changes are consistent with the established pathophysiology of glaucomatous damage, where elevated IOP leads to optic nerve head deformation, lamina cribrosa displacement, and progressive retinal ganglion cell loss [6]. The combination of clinical and imaging findings provides a robust diagnostic foundation, confirming both acute and chronic components of optic nerve compromise.

A key aspect of this case is the differential diagnosis, particularly distinguishing APAC from phacomorphic glaucoma, a secondary angle-closure mechanism caused by intumescence of a cataractous lens. Phacomorphic glaucoma typically presents with progressive lens thickening, anterior displacement of the iris, pupillary block, and elevated IOP. Although this patient exhibited an immature senile cataract that may have contributed to angle narrowing, the acute symptom onset, classical mid-dilated pupil, presence of glaukomflecken, and gonioscopic findings were more consistent with primary rather than secondary angle-closure mechanisms. Nonetheless, the cataract likely exacerbated anterior segment crowding, reinforcing the importance of lens extraction after initial IOP control [6]. This interplay between primary and lens-related mechanisms is well documented in Asian populations, where biometric predispositions amplify the risk of angle closure.

Recent literature strengthens these interpretations. Sun et al. (2023) demonstrated that RNFL thinning, larger cup-to-disc ratios, and more pronounced visual field defects are characteristic structural features of primary angle-closure glaucoma, aligning closely with this patient's OCT findings [7]. Updated epidemiologic data from Kitnarong et al. (2024) also confirm that APAC remains highly prevalent in Asian populations due to biometric predispositions such as shallow anterior chambers, thicker lenses, and shorter axial lengths, consistent with the anatomical factors observed in this case [8]. A 2023 analysis of spectral-domain OCT angiography (OCTA) revealed that reduced peripapillary vessel density and RNFL thinning are strongly associated with angle-closure severity, providing objective markers for distinguishing primary angle-closure mechanisms from other forms of secondary IOP elevation [9]. These imaging biomarkers enhance diagnostic precision and support early intervention strategies.

Management in this case aligns with contemporary clinical evidence. Initial treatment with Timolol Maleate, Tobramycin–Dexamethasone, and oral Acetazolamide was appropriate for acute IOP reduction and inflammation control. However, persistent elevation of IOP and structural optic nerve compromise necessitated surgical intervention. Recent systematic reviews, including a 2024 analysis by Sharif et al., demonstrate that phacoemulsification provides greater long-term IOP stability, deeper anterior chambers, and a reduced risk of recurrent angle closure compared with laser peripheral iridotomy, reinforcing the appropriateness of cataract extraction in this case [10]. Phacoemulsification not only removes the lens contributing to anterior segment crowding but also widens the angle and reduces pupillary block, offering both therapeutic and preventive benefits. Despite timely intervention, structural damage to the optic nerve can continue to progress [11-21], especially after prolonged or severe acute attacks, as noted in contemporary longitudinal studies.

Overall, this case highlights the multifactorial nature of angle-closure disease, the interplay between anatomical predisposition and lens changes, and the critical importance of early detection, multimodal imaging, and timely surgical intervention in preventing irreversible glaucomatous optic neuropathy. The integration of clinical findings, imaging biomarkers, and evidence-based management underscores the need for heightened vigilance in populations at risk and reinforces the value of comprehensive ophthalmic evaluation in acute presentations.

Limitation

This case report has several limitations inherent to single-patient observations. First, as an isolated clinical case, the findings may not fully represent the broad spectrum of presentations, anatomical variations, or disease severity encountered in acute primary angle closure, particularly in diverse populations with varying biometric profiles and genetic backgrounds. The clinical course described here reflects one individual's experience and may differ substantially from patients with milder symptoms, atypical manifestations, or more advanced structural compromise.

Second, although detailed clinical examinations, multimodal imaging results, and postoperative findings were documented comprehensively, the report does not include biometric parameters such as axial length, lens thickness, anterior chamber depth, anterior chamber volume, or angle width. These measurements could have provided additional insight into the anatomical contributors to angle closure and strengthened the interpretation of predisposing factors. The absence of ultrasound biomicroscopy or anterior segment OCT further limits the ability to characterize subtle anterior segment crowding or plateau iris configuration.

Third, the follow-up period was limited, preventing assessment of long-term intraocular pressure behavior, structural stability of the optic nerve head, and functional visual field recovery after treatment. Longitudinal monitoring is essential in angle-closure disease, as progressive optic neuropathy may continue even after successful IOP reduction, particularly in cases with severe initial attacks. Without extended follow-up, the durability of postoperative outcomes and the risk of recurrent angle closure cannot be fully evaluated.

Fourth, genetic evaluation was not performed, despite the presence of a positive family history of vision loss. This limits exploration of hereditary contributions, including potential genetic variants associated with anterior chamber morphology, iris thickness, or trabecular meshwork resistance. Incorporating genetic analysis could have provided a deeper understanding of familial predisposition and informed recommendations for screening among first-degree relatives.

Finally, as with all case reports, the findings cannot be generalized to broader populations or used to infer causal relationships. These factors restrict the ability to evaluate prolonged disease progression but do not diminish the clinical relevance of the acute findings described, particularly in highlighting the importance of early recognition, multimodal assessment, and timely intervention in preventing irreversible glaucomatous optic neuropathy.

CONCLUSION

This case demonstrates how acute primary angle closure can rapidly threaten visual function and highlights the importance of early recognition, comprehensive evaluation, and timely intervention. The coexistence of an immature senile cataract contributed to anterior segment crowding and

reinforced the need for cataract extraction as a definitive component of management after initial intraocular pressure control. Prompt treatment helped prevent further glaucomatous progression, although structural damage persisted due to delayed presentation. This case underscores the clinical importance of rapid diagnosis, multimodal assessment, and appropriate surgical planning in achieving optimal outcomes for patients presenting with acute angle-closure disease.

Ethical consideration, competing interest and source of funding

- The case report was approved by the ethics committee and informed consent was obtained from the patient for publication, ensuring confidentiality and the right to withdraw at any time.
- There is nothing to declare
- Source of funding is authors.

REFERENCES

1. Ezinne NE, Shittu O, Ekemiri KK, et al. Visual impairment and blindness among patients at Nigeria Army Eye Centre, Bonny Cantonment Lagos, Nigeria. *Healthc Switz*. 2022;10(1):1–8.
2. Allison K, Patel D, Alabi O. Epidemiology of glaucoma: the past, present, and predictions for the future. *Cureus*. 2020 Nov 24;12(11).
3. Weinreb RN, Aung T, Medeiros FA. The pathophysiology and treatment of glaucoma: A review. *Jama*. 2014;311(1):1901–1911.
4. Nüßle S, Reinhard T, Lübke J. Acute closed-angle glaucoma - An ophthalmological emergency. *Dtsch Arzteblatt Int*. 2021;118(1):771–780.
5. Wiggs JL, Pasquale LR. Genetics of glaucoma. *Hum Mol Genet*. 2017;26(1):R21–R27.
6. Tanna AP, Boland MV, Ciacconi JA, Krishnan C, Lin SC, Medeiros FA. Basic and clinical science course, section 10 glaucoma. *American Academy of Ophthalmology*; 2022.
7. Sun JA, Yuan M, Johnson GE, et al. Comparison of structural and functional features in primary angle closure and open angle glaucomas. *J Glaucoma*. 2024;33(1):254–261.
8. Kitnarong N, Phongsuphan T, Tanratanavong T. Incidence of acute angle closure in patients with primary angle closure without prophylactic iridotomy during pharmacological mydriasis for cataract surgery. *Medicine (Baltimore)*. 2024;103(1):e41020.
9. Jiang W, Jiang N, Liu G-B, et al. Structural measurements and vessel density of spectral-domain optical coherence tomography in early, moderate, and severe primary angle-closure glaucoma. *Int J Ophthalmol*. 2023;16(1):1100–1109.
10. Sharif RK, Aljahdali FF, Aljabri HM, et al. Phacoemulsification versus laser peripheral iridotomy for treating primary angle closure glaucoma: A systematic review and meta-analysis. *Clin Ophthalmol*. 2024;18(1):1023–1032.
11. Miller NR, Tsai RK. Optic neuropathies: current and future strategies for optic nerve protection and repair. *International Journal of Molecular Sciences*. 2023 Apr 10;24(8):6977.
12. Cen LP, Park KK, So KF. Optic nerve diseases and regeneration: How far are we from the promised land?. *Clinical & Experimental Ophthalmology*. 2023 Aug;51(6):627–41.
13. Gokoffski KK, Peng M, Alas B, Lam P. Neuro-protection and neuro-regeneration of the optic nerve: recent advances and future directions. *Current Opinion in Neurology*. 2020 Feb;33(1):93.
14. Morrison JC, Johnson EC, Cepurna W, Jia L. Understanding mechanisms of pressure-induced optic nerve damage. *Progress in retinal and eye research*. 2005 Mar 1;24(2):217–40.
15. Fague L, Liu YA, Marsh-Armstrong N. The basic science of optic nerve regeneration. *Annals of Translational Medicine*. 2021 Aug;9(15):1276.
16. Liu ZG, Zhou LY, Sun YQ, Ma YH, Liu CM, Zhang BY. Unlocking the potential for optic nerve regeneration over long distances: a multi-therapeutic intervention. *Frontiers in Neurology*. 2025 Jan 9;15:1526973.
17. Benowitz LI, He Z, Goldberg JL. Reaching the brain: Advances in optic nerve regeneration. *Experimental Neurology*. 2017 Jan 1;287:365–73.
18. Coleman-Belin J, Harris A, Chen B, Zhou J, Ciulla T, Verticchio A, Antman G, Chang M, Siesky B. Aging effects on optic nerve neurodegeneration. *International Journal of Molecular Sciences*. 2023 Jan 29;24(3):2573.
19. Williams PR, Benowitz LI, Goldberg JL, He Z. Axon regeneration in the mammalian optic nerve. *Annual Review of Vision Science*. 2020 Sep 15;6(1):195–213.
20. Biousse V, Danesh-Meyer HV, Saindane AM, Lamirel C, Newman NJ. Imaging of the optic nerve: technological advances and future prospects. *The Lancet Neurology*. 2022 Dec 1;21(12):1135–50.
21. Shin HJ, Costello F. Imaging the optic nerve with optical coherence tomography. *Eye*. 2024 Aug;38(12):2365–79.