# Secondary Glaucoma in Patient with A Mature Cataract: A Case Report

**Ni Made Savita Medha Sumantra[1], Faozan[2]**
[1]Tarumanagara University, Jakarta, Indonesia
[2]Bhayangkara Hospital Ophthalmology Specialist, Semarang, Indonesia
*E-mail: savitamedha11@gmail.com, zan.fao@gmail.com*

**Article Info**
**Article History**
Received: 2025-01-10
Revised: 2025-02-20
Published: 2025-03-11

**Keywords:**
*Secondary Glaucoma;*
*Lens-Induced Glaucoma;*
*Mature Cataract;*
*Acute Glaucoma.*

**Abstract**
According to WHO estimates, there were roughly 60,7 million cases of glaucoma in 2010 and will be 79,4 million cases by 2020. A cataract audit at the Sultanah Nur Zahirah Hospital, Kuala Terengganu, discovered that lens-related glaucoma affected 1.08% of cataract cases. This lens abnormality was a secondary glaucoma in which the crystalline lens had a role in the process causing elevated intraocular pressure (IOP). Secondary glaucoma is a glaucoma that occurs because of other underlying conditions. Glaucoma can be acute or chronic. Since acute glaucoma can result in lifelong blindness, it is a medical emergency that needs to be treated very away Understanding the warning signs and symptoms is crucial to preventing glaucoma-related lifelong blindness. A case of secondary glaucoma was presented in a 73 year old man who had ODS mature cataract and underwent OS phacoemulsification 1 day ago, now complaining of sudden redness and pain in the right eye accompanied by nausea and foggy vision. Vision OD 1/300. Ocular examination found narrow vision, hyperemic bulb conjunctive OD, conjunctive injection OD, corneal epithelial edema OD, shallow anterior chamber OD, curvy lens OD, mid-dilated pupil, shadow test negative, pupil reflex that cannot be evaluated, and IOL OS positive. A tonometry test was carried out and found IOP OD 30 mmHg, OS 13 mmHg. Fundoscope direct OD could not be assessed due to stiffness on the lens.

**Artikel Info**
**Sejarah Artikel**
Diterima: 2025-01-10
Direvisi: 2025-02-20
Dipublikasi: 2025-03-11

**Kata kunci:**
*Glaukoma Sekunder;*
*Glaukoma Diinduksi Lensa;*
*Katarak Matur;*
*Glaukoma Akut.*

**Abstrak**
WHO memperkirakan penderita glaukoma di dunia mencapai sekitar 60,7 juta (2010) dan akan menjadi 79,4 juta (2020). Audit kasus katarak di Rumah Sakit Sultanah Nur Zahirah, Kuala Terengganu, menemukan bahwa 1,08% kasus katarak mengalami glaukoma akibat lensa, Glaukoma yang terjadi akibat kelainan lensa adalah glaukoma sekunder di mana lensa kristalin terlibat dalam mekanisme peningkatan tekanan intraokular (TIO). Glaukoma yang disebabkan karena adanya penyakit yang mendasarinya dikenal dengan glaukoma sekunder. Glaukoma dapat berupa akut ataupun kronik. Glaukoma akut merupakan suatu kegawatdaruratan medis yang harus segera karena dapat menyebabkan kebutaan permanen. Pentingnya mengetahui tanda dan gejala untuk menghindari kebutaan permanen akibat glaukoma. Dipresentasikan sebuah kasus glaukoma sekunder pada laki-laki usia 73 tahun yang mengalami katarak matur ODS dan telah dilakukan fakoemulsifikasi OS 1 hari yang lalu, kini mengeluhkan adanya kemerahan dan nyeri tiba-tiba pada mata kanan disertai mual dan pandangan berkabut. Visus OD 1/300. Pemeriksaan okular ditemukan menyempitnya lapang pandang, hiperemis konjungtiva bulbi OD, injeksi konjungtiva OD, edema korena OD, bilik mata kanan depan dangkal, lensa keruh OD, *shadow test* negatif, *mid-dilated pupil*, refleks pupil tidak dapat dinilai, IOL OS (+). Dilakukan tes tonometri ditemukan TIO OD 30 mmHg, OS 13 mmHg. funduskopi direk OD tidak dapat dinilai karena kekeruhan pada lensa.

## I. INTRODUCTION

Secondary glaucoma is a type of glaucoma that occurs because of other underlying conditions. The crystalline lens plays a role in the mechanism of elevated intraocular pressure (IOP) in secondary glaucoma, which is a type of glaucoma caused by abnormalities in the lens. Glaucoma can occur both acutely and chronically, and there are four distinct variants: phacolytic, lens particle, phacoantigenic, and phacomorphic (Laurenti K et all., 2016). Since acute glaucoma can result in lifelong blindness, it is a medical issue that requires prompt attention (AAO, 2024). Acute glaucoma originates from the sudden obstruction of the eye's drainage system as a result of the closure of the angle formed by the cornea and iris. Pupil constriction is often regarded as the primary factor contributing to

acute glaucoma, accounting for around 90% of cases (Babak K et all., 2023).

Glaucoma ranks as the second leading cause of blindness worldwide, following cataracts. WHO predicts that the global prevalence of glaucoma will increase from approximately 60.7 million in 2010 to 79.4 million in 2020. An audit conducted at Sultanah Nur Zahirah Hospital in Kuala Terengganu revealed that 1.08% of cataract cases were associated with lens-related glaucoma. Among these cases, Women made up the majority (57%) and those aged 70–79 years old were the most affected (44.30%) (Mohd Azmi A et all., 2022). A study of lens-induced glaucoma in rural areas found that compared to phacolytic glaucoma, phacomorphic glaucoma was more prevalent. (30 cases vs. 13 cases, or 30.23%) (Anitha SM et all., 2017).

In 2019 "Glaucoma Situation in Indonesia" report, the Ministry of Health of the Republic of Indonesia estimates that the number of glaucoma patients worldwide will reach 76 million by 2020, an increase of 25.6% from 60.5 million in the previous 10 years. According to official data in Indonesia, about 0.46% (about 4-5 people per 1,000 in habitants) have glaucoma (Pusat Data dan Informasi Kemenkes RI, 2019; Ministry of Health of the Republic of Indonesia, 2022). A high incidence of blindness due to glaucoma and cataracts will affect the quality of life due to a person's loss of vision. Therefore, early detection and treatment are needed as soon as possible to prevent permanent blindness caused by glaucoma, especially in patients with cataract.

## II. METHOD

A day ago, a 73-year-old man came to Bhayangkara Hospital with a red right eye complaint and sudden pain and nausea, accompanied by blurred vision in both eyes that had deteriorated in the last week. Initially, the blurred vision felt like a fog in the right eye a month ago and did not interfere with activity. However, for the last two weeks it has been felt in both eyes, with swelling in the right eye. The complaints are perceived to interfere with the patient's activity, the vision feels cloudy and constant, heavily tired during the day, the patient feels bleak when seeing sunlight, and the complaint improves at night. Since last week, patient have complained about the cloudy vision that increasingly interferes with his activity. The local clinic then refers the patient to Bhayangkara Hospital. The patient was diagnosed with cataracts in both eyes and is scheduled to undergo surgery on the left eye first. In addition, patient was given eye drops to treat swelling in the right eye, and the swelling complaints improved. Since 1 day ago, the patient complained of redness in the right eye and pain that arose suddenly in the right eye, continuously, VAS 5/10, the pain was not heightened or relieved by any factor accompanied by nausea. The patient has been using glasses for the past month to address complaints of blurred vision, but the use of these glasses did not alleviate the symptoms. For the last 20 years, patient have had the habit of frequently drinking a cup of warm sweet tea and smoking as many as four cigarettes a day. A history of hypertension, diabetes mellitus, heart disease, eye trauma, routine medication, and allergies were denied. A family history of similar complaints is denied, the patient's wife has diabetes mellitus and has died.

According to the current physical examination, the general condition appears to be moderate, vital signs within normal limits. Visual examination found OD 1/300, OS 0.3 F2. During the physical examination of the right eyes, we found narrowed field of vision, hyperemic bulb conjunctiva, conjunctive injection, corneal edema, shallow anterior chamber, an inability to assess the direct and indirect light reflex, mid-dilated pupil, curvy lens, shadow test negative and IOL OS positive shown in Figure 1.

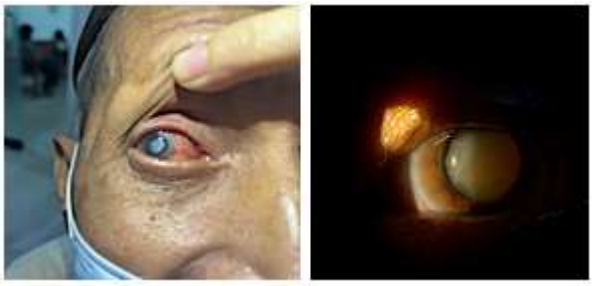

**Figure 1**. Patient's ocular examinations OD (a) without slitlamp; (b) with slitlamp showed hyperemic bulb conjunctiva, conjunctive injection, corneal edema, shallow anterior chamber, an inability to assess the direct and indirect light reflex, mid-dilated pupil, curvy lens, shadow test negative

The tonometric examination obtained IOP OD 30 mmHg, OS 13 mmHg. The fundoscope examination could not be evaluated at the direct OD, but the OS revealed a round, firmly bounded optic disc with yellow redness, a ratio of C/D 0.3, a ratio of A/V 2/3, retina within normal limits, and a positive macular reflex.

Based on the history and examinations carried out, the patient was diagnosed with secondary

glaucoma OD et causa cataract senilis mature OD. Implementation was made of administration of acetazolamide 1x250 mg, KSR 1x600 mg, timolol 0.5% 2x1 gtt OD, prednisolone acetate eye drops 1% 2 gtt/day. Patient is also planned to undergo OD phacoemulsification after glaucoma-related complaints improve and patient IOP OD decreases.

## III. RESULT AND DISCUSSION

Glaucoma is an eye disease characterised by increased IOP, constricted optical discs, and a narrowed field of vision. The normal limit of IOP falls between 15-20 mmHg. In addition, high IOP causes retinal blood vessel disorders that can cause retinal metabolism disruption, followed by the death of the eye nerve. Retinal function will be disrupted. As time passes, the victim will become completely blind (The Indonesian Association of Eye Specialists, 2002). The higher the IOP, the sooner the retinal nerve fibres get damaged. In some cases, normal IOP can cause damage to optic nerve fibers. (Normal tension glaucoma). IOP in glaucoma has nothing to do with a person's blood pressure (Suhardjo SU et all., 2007).

Understanding the pathophysiology of glaucoma requires knowing the physiology of aqueous humor (AH). AH is composed of ~98.7% water and ~1.3% solids. The solids found in AH are organic substances (such as albumin, glucose, globulin, lactate, urea, and pyruvate) and inorganic substances (like sodium, potassium, calcium, chloride, magnesium, phosphate, and bicarbonate) (Ganong W et all., 2009; Riordan EP et all., 2010). Processes and cellular bodies secrete about 80% of AH through active metabolite processes, depending on the amount of carbonic anhydrase enzyme, while the remaining 20% will be secreted through passive processes of diffusion and ultrafiltration (Schuman JS et all., 2008). The total volume of AH on the front and rear eyelashes is on average 0.2–0.4 mL, and a 1-2% replacement of AH is performed every minute (Lang GK et all., 2006). After AH is produced and is in the rear eye room, the pupil goes to the front eye room and exits through the corner of the front eye room. About 75% through the trabecular meshwork and 25% through the uveoscleral pathway (Sitorius SR et all., 2020). AH plays an important role in providing nutrients, disposing of waste products, and transporting neurotransmitters to the avascular lens and cornea. In addition, AH also plays a role in maintaining the structural integrity of the eyeball by keeping IOP within normal limits (Ganong W et all., 2009).

When the pupil's outflow resistance increases, the pressure on the rear eye chamber will rise. Furthermore, there will be front-facing iris inflammation on the base, as well as stress on the trabecular material. Increased intraocular pressure caused by trabecular obstruction is the cause of primary closed-angle glaucoma. The trabecular form, which consists of loose forms that resemble avascular tissue located between the spurdan schwalbe's scleral lines, is the second type of physiological resistance. Open-angle glaucoma occurs when resistance increases in this place. Absence of abnormalities in the anatomical relationship between the base of the iris, the peripheral cornea, and the trabecular membrane in open-angle glaucoma. Open-angle glaucoma is caused by increased resistance to AH drainage and trabecular tissue congestion (Lang GK et all., 2006).

According to its etiology, glaucoma is classified into four categories, including primary, secondary, absolute and congenital (Table 1) (John FS, 2018).

**Table 1**. Glaucoma Classified According to Etiology by the Vaughan & Asbury's General Ophthalmology

| Glaucoma Classified According to Etiology | | |
|---|---|---|
| Primary Glaucoma | Open angle glaucoma | Primary open angle glaucoma |
| | | *Normal tension glaucoma* |
| | Angle closure glaucoma | |
| Secondary Glaucoma | Pigmentary glaucoma | |
| | Exfoliation syndrome | |
| | Due to lens change | Intumescence, dislucation, phacolytic |
| | Due to uveal tract changes | Uveitis, tumor, ciliary body swelling, posterior synechiae |
| | Iridocorneal endothelial (ICE) syndrome | |
| | Trauma | Peripheral anterior synechiae, hyphema and *angle contusion/recession* |
| | Post-operative | Malignant glaucoma, epithelial downgrowth, peripheral anterior synechiae, following corneal graft surgery or retinal detachment surgery |
| | Neovascular glaucoma | Diabetes mellitus, intraocular tumor and central retinal vein occlusion |
| | Raised episcleral venous pressure | *Struge weber syndrome, carotid cavernous fistula* |
| | Steroid induced | |

| | | |
|---|---|---|
| Absolute Glaucoma | The end result of any uncontrolled glaucoma | |
| Congenital Glaucoma | Primary congenital glaucoma | |
| | Associated with other developmental ocular abnormalities | *Anterior chamber cleavage syndrome,* peter syndrome, axenfeld-rieger syndrome |
| | Associated with extraocular developmental abnormalities | Struge-weber syndrome, congenital rubella, marfan's syndrome, lowe syndrome and neurofibromatosis type 1 |

Increased IOP is a sign of glaucoma, while the presence of other eye diseases or underlying factors (such as tumours, trauma, bleeding, medication, inflammation, and physical and chemical factors) accompanied by an increase in IOP are signs of secondary glaucoma (Suhardjo Su et all., 2007). Glaucoma resulting from lens abnormalities is a type of secondary glaucoma where the mechanism of the rise involves the crystalline lens. IOP. Glaucoma can occur acutely or chronically, and there are four different variants: phacolytic, lens particle, phacoantigenic, and phacomorphic. Later, Epstein's study provided evidence of the role of high-molecular weight soluble lens proteins in causing direct obstruction of the aqueous outlet. While phacomorphic glaucoma occurs because the senile cataract lens can become intumescent, thicken, and cause pupil blockade. This iridolenticular apposition interferes with the flow of aqueous humour from the front chamber to the front eye chamber. This causes the accumulation of fluid in the posterior space, pushing the iris root forward, which can eventually contact the trabecular circuit and cause the corner closure (Laurenti K et all., 2016). The patient is classified as having secondary glaucoma OD as a result of manifestations of other eye diseases such as mature cataract OD. The diagnosis of secondary acute glaucoma can be established based on the history, physical examination, and ocular examination performed. Typically, acute closed-angle glaucoma attacks are unilateral, only 5–10% occur bilaterally. Clinical manifestations and physical examinations of the eye that can be found in patients with acute glaucoma, such as blurred vision or sudden decrease in visual acuity, visible halo surrounding light (coloured like a rainbow, with a central halo coloured green blue and a peripheral halo coloured yellow red), red eyes and very painful, can be accompanied by headache with varying degrees, nausea, vomiting, corneal edema, mid dilated pupil, irregular, negative pupil reflex, shallow anterior chambers, and on the conjunctive bulbs obtained presence (congestive hyperemia, chemosis with silier injection, conjungtive injections) (AAO, 2024; Sitorius SR et all., 2020; The Indonesian Association of Eye Spesialist, 2018). Patient obtained clinical manifestations such as blurred vision and progressive decrease in visual acuity due to cataracts experienced, redness in the right eye, sudden pain with VAS 5/10, nausea, and found physical examination of right eye such as conjunctiva bulbi hyperemic, conjuntive injection, corneal edema, curvy lens, mid dilated pupil, pupil reflex cannot be evaluated, shallow anterior chamber, and shadow test negative.

Eye tests like slit lamp exams, visual field tests, tonometry (IOP > 21 mmHg), direct fundoscope, gonioscopy, ultrasound biomicroscope (UBM), or AC-OCT can help find out if someone has acute glaucoma.

**Table 2**. Criteria diagnostic based on American Academy of Ophthalmology (AAO)

| Criteria Diagnostic According AAO | |
|---|---|
| At least 2 from 3 these symptoms | Ocular/periocular pain |
| | Nausea/vomiting |
| | History of intermiten blurred vision with halo |
| At least 3 from 4 these signs | Conjuntive injection |
| | Corneal epithelial edema |
| | Mid-dilated unreactive pupil |
| | Shallow anterior chamber |
| Should have an increase IOP > 21 mmHg | |

In this patient, there are any symptoms such as ocular pain and nausea (fulfill 2 from 3 symptoms). According to physical examinations there are any signs such as conjungtive injection, cornea edema, mid-dilated pupil with an inability to assess the direct and indirect light pupil reflex and shallow anterior chamber (fulfill 4 from 4 signs). Tonometry examination found IOP OD 30 mmHg. This patient meet the acute glaucoma criteria based on AAO. Acute glaucoma that patient experienced due to other ocular manifestation such as mature cataract OD. The diagnosis of the patient is acute secondary glaucoma.

Acute glaucoma is a medical emergency and should be treated with IOP-lowering drugs that have a rapid onset of action (Table 3). When the IOP has dropped, the patient must undergo a definitive procedure as quickly as possible to prevent recurrence (Khondkaryan A et all., 2013;

Murray D, 2018). The goal of treating acute glaucoma is to preserve the patient's quality of life and ability to see. Management of secondary glaucoma is also carried out for the underlying disease.

**Table 3**. Overview drugs to treat glaucoma

| Overview Drugs Used To Treat Glaucoma | | |
|---|---|---|
| Mechanism of IOP decrease | Drugs | Mechanism of Action |
| Decrease synthesis of aqueous humor | Beta blocker (timolol, betaxolol, carteolol) | Blockage of sympathetic nerve fibers in the ciliary epithelium |
| | Alpha 1 agonists (epinefrin) | Via decrease in cAMP, and additional conjunctival vasoconstriction |
| | Alpha 2 agonists (apraclonidine, brimonidine) | Via decrease in cAMP |
| | *Carbonic anhydrase inhibitor* (acetazolamide, methazolamide, brinzolamide, dorzolamide) | Inhibits the action of *carbonic anhydrase enzyme* |
| Increase aqueous humor outflow | PGF 2$\alpha$ (latanoprost, travaprost, bimatoprost) | Decreases resistance through uveoscleral flow |
| | Direct parasimpatomimetik (pilocarpine, carbachol) | Ciliary muscle contraction and trabecular meshwork opening (into canal of Schlemm) |
| | Indirect arasimpatomimetik (physostigmine, echothiophate) | |

In addition to medications to lower IOP, patients may also be given anti-inflammatory drugs such as topical steroids to reduce inflammatory reactions and corneal edema. If corneal edema is still present, iridoplasty laser surgery can be performed, with or without iridotomy. Furthermore, peripheral iridectomy (laser or surgical) can be performed if IOP has decreased and corneal edema has improved. It may also be considered to perform trabeculectomy with or without an antifibrotic agent if the use of IOP medication does not decline. If the eye condition is calm, cataract extraction can be performed when glaucoma occurs due to cataract disease. To prevent an attack on a healthy eye, a peripheral laser iridectomy or surgical surgery may be considered. All patients with acute glaucoma who have been treated should be re-evaluated (AAO, 2024; Sitorius SR et all., 2020; The Indonesian Association of Eye Spesialist, 2018). This patient received CAI, beta blockers, potassium tablet supplements, and topical steroids. Patient was also planned to undergo OD phacoemulsification after glaucoma-related complaints improved and patient IOP OD decreased.

## IV. CONCLUSION AND SUGGESTION

### A. Conclusion

Secondary glaucoma is a known underlying cause of glaucoma. A variety of factors, including dislocation, intumescence, and phacolytic abnormalities in the lens, can cause secondary glaucoma, leading to an increase in IOP. The process of diagnosing glaucoma involves a variety of methods, including history, physical examinations, and ocular examinations such as gonioscopy, slit lamp examinations, tonometry, visual acuity, and field of vision examinations, as well as direct fundoscope, UBM, and related supporting examinations of the underlying causes. According to the AAO, there are also criteria for diagnosing acute glaucoma. Diagnosis is needed as soon as possible to deal with the underlying disease and prevent secondary glaucoma. Acute glaucoma should be treated immediately to prevent permanent blindness.

### B. Suggestion

There is still limited discussion in this research. A more comprehensive study of secondary glaucoma in patient with a mature cataract is needed as a suggestion for future authors.

## REFERENCES

American Academy of Ophthalmology. Angle Closure Glaucoma. AAO [Internet]. 2013 [cited 2024 Jun 26]. Available from: https://www.aao.org/education/current-insight/angleclosure-glaucoma-19

Anitha, S.M., Ravindra, B., Manjunath, B.H., Meghana, P. (2017). Lens-Induced Galucoma In The Rural Population: Is Cataract Surgery In One Eye Boon or Bane?. *Indian Journal of Clinical and Experimental Ophthalmology, 3*(1), 24-27.

Babak, K., Leila, K. Acute Closed Angle Glaucoma [Updated 2023 Jan 03]. In: StatPearls [Internet]. Treasure Island (FL): *StatPearls Publishing*; 2023 [cited 2024 Jun 26]. Available from:

https://www.ncbi.nlm.nih.gov/books/NBK430857/

Ganong, F.W. (2009). Buku Ajar Fisiologi Kedokteran. Edisi 22. Jakarta: ECG.

Pusat Data dan Informasi Kementerian Kesehatan RI. Situasi Glukoma di Indonesia. InfoDATIN [Internet]. 2019.

John, F.S. (2018). Glaukoma. In: Vaughan & Asbury's General Ophthalmology [Book]. 19th Edition. Lange. McGraw Hill Education. 518-534p.

Kementerian Kesehatan Republik Indonesia. Glaukoma dan Kelainan Refraksi. Kemekes RI [Internet]. 2022.

Khondkaryan, A., Francis, B.A. Angle-Closure Glaucoma. AAO [Internet]. 2013.

Laurenti, K., Salim, S. Lens-Induced Glaucoma: Diagnosis and Management. Ophthalmic Pearls AAO [Internet]. 2016.

Lang, G.K. (2006). *Ophthalmology A Pocket Textbook Atlas 2nd Edition*. Thieme. Stuttgart- New York.

Murray, D. Emergency management: angle-closure glaucoma. Community eye health. 2018; 31(103): p. 64.

Mohd Azmi, A., Che Omar, N. A., & Wan Abdul Halim, W. H. (2022). Lens-Induced Glaucoma: An Audit of Cataract Cases in Hospital Sultanah Nur Zahirah, Kuala Terengganu (HSNZKT). *Cureus*, *14*(3), e22875.

Perhimpunan Dokter Spesialis Mata Indonesia. (2002). Ilmu Penyakit Mata Untuk Dokter Umum dan Mahasiswa Kedokteran. Edisi ke-2. Jakarta: Sagung Seto.

Perhimpunan Dokter Mata Indonesia. (2018). Pedoman Nasional Pelayanan Kedokteran Glaukoma. Jakarta: Badan Penerbit PERDAMI.

Riordan, E.P., Whitcher, J.P. (2010). Vaughan & Asbury Oftalmologi Umum. Edisi 17. Jakarta: EGC.

Schuman, J.S., Christopoulos, V., Dhaliwal, D.K., Kahook, M.Y., Noecker, R.J. (2008). Rapid Diagnosis in Ophthalmology: Lens and Glaucoma. Philadelphia: Mosby Elsevier.

Sitorius, S.R., Sitompul, R., Widyawati, S., Bani, P.A. (2020). Buku Ajar Oftalmologi Fakultas Kedokteran Universitas Indonesia. Edisi 1 Cetakkan ke 2. Jakarta: Universitas Indonesia Publishing.

Suhardjo, S.U., Sasongko, M.B., Anugrahsari, S. (2007). Ilmu Kesehatan Mata. Yogyakarta: Bagian Ilmu Penyakit Mata Fakultas Kedokteran Universitas Gadjah Mada Yogyakarta.