

Oftalmologi: Jurnal Kesehatan Mata Indonesia 2026, Volume 8, Number 1.
P-ISSN. 2723-6935, E-ISSN. 2541-4283
Received: 7 April 2026; Revised: 24 April 2026; Accepted: 27 April 2026

When the Blind Eye Gets Red: Bilateral Conjunctivitis in Absolute Glaucoma with Phthisis Bulbi Sinistra

Daniel Pradana Andrian Wicaksono¹, Eny Tjahjani Permatasari²

¹Medical Faculty of Universitas Kristen Duta Wacana, Yogyakarta, Daerah Istimewa Yogyakarta, Indonesia

²RS Mata dr. YAP, Yogyakarta, Daerah Istimewa Yogyakarta, Indonesia

*Correspondence: Daniel Pradana Andrian Wicaksono, danielpradana0@gmail.com

The work is licensed under a Creative Commons Attribution License (CC BY-SA 4.0)

How to Cite:

Wicaksono, D. P. A., & Permatasari, E. T. (2026). When the Blind Eye Gets Red: Bilateral Conjunctivitis in Absolute Glaucoma with Phthisis Bulbi Sinistra. *Oftalmologi: Jurnal Kesehatan Mata Indonesia*, 8(1), 30–35. <https://doi.org/10.11594/ojkmi.v8i1.101>

ABSTRACT

Introduction: Glaucoma is one of the leading causes of irreversible blindness worldwide, affecting more than 76 million people globally, with numbers projected to increase. In Indonesia, glaucoma accounts for approximately 13–15% of blindness cases, with many patients presenting at an advanced stage. End-stage glaucoma may progress to phthisis bulbi and is often associated with ocular surface impairment. Conjunctivitis in this setting presents a diagnostic challenge as it may be caused by infectious or non-infectious mechanisms.

Case Report: A patient presented with bilateral red eyes for one week, accompanied by epiphora and foreign body sensation without purulent discharge. Examination revealed advanced glaucoma in the right eye with elevated intraocular pressure, and absolute glaucoma with phthisis bulbi in the left eye with no light perception. Both eyes showed conjunctival hyperemia without signs of acute infection.

Discussion: Conjunctivitis in end-stage glaucoma is often associated with ocular surface disease due to chronic inflammation, tear film instability, and long-term use of topical medications. This condition may mimic infection, requiring careful clinical evaluation to avoid misdiagnosis and inappropriate treatment.

Conclusion: Conjunctivitis in end-stage glaucoma is not always infectious but is frequently part of ocular surface disease. Management should be individualized, focusing on patient comfort and quality of life.

Keywords: conjunctivitis, glaucoma, ocular surface disease, phthisis bulbi, red eye

ABSTRAK

Pendahuluan: Glaukoma merupakan salah satu penyebab utama kebutaan permanen di dunia dengan lebih dari 76 juta penderita secara global dan diperkirakan terus meningkat. Di Indonesia, glaukoma menyumbang sekitar 13–15% kasus kebutaan, dengan banyak pasien datang pada stadium lanjut. Pada stadium akhir, glaukoma dapat berkembang menjadi phthisis bulbi serta menyebabkan gangguan permukaan okular. Konjungtivitis pada kondisi ini sering menjadi tantangan diagnostik karena dapat bersifat infeksi maupun non-infeksi.

Laporan Kasus: Seorang pasien datang dengan keluhan mata merah bilateral selama satu minggu, disertai epifora dan sensasi mengganjal tanpa sekret purulen. Pemeriksaan menunjukkan glaukoma lanjut pada mata kanan dengan peningkatan tekanan intraokular, serta glaukoma absolut dengan phthisis bulbi pada mata kiri tanpa persepsi cahaya. Kedua mata menunjukkan hiperemia konjungtiva tanpa tanda infeksi akut.

Pembahasan: Konjungtivitis pada glaukoma stadium akhir sering berkaitan dengan ocular surface disease akibat inflamasi kronis, ketidakstabilan tear film, serta penggunaan jangka panjang obat topikal. Kondisi ini dapat menyerupai infeksi sehingga memerlukan evaluasi klinis yang cermat untuk menghindari kesalahan diagnosis dan terapi.

Kesimpulan: Konjungtivitis pada glaukoma stadium akhir tidak selalu disebabkan oleh infeksi, melainkan sering merupakan bagian dari ocular surface disease. Penatalaksanaan harus bersifat individual dengan fokus pada kenyamanan dan kualitas hidup pasien.

Kata kunci: glaukoma, konjungtivitis, mata merah, ocular surface disease, phthisis bulbi

INTRODUCTION

Glaucoma remains one of the leading causes of irreversible blindness worldwide, affecting an estimated more than 76 million people globally, with projections reaching over 110 million by 2040.¹ The burden is particularly high in developing countries, where late diagnosis and limited access to care contribute to disease progression. In Indonesia, glaucoma is among the top causes of blindness, accounting for approximately 13–15% of total blindness cases, with many patients presenting at an advanced stage.² Regional data, including reports from Yogyakarta and surrounding areas, also indicate a similar trend, where patients often seek medical attention only after significant visual deterioration has occurred.

Glaucoma is characterized by progressive optic neuropathy that leads to irreversible visual field loss.³ In its end stage, known as absolute glaucoma, the eye loses all visual function and is often associated with persistent pain, elevated intraocular pressure, and progressive structural damage.⁴ In long-standing cases, this condition may progress to phthisis bulbi, a state in which the globe becomes shrunken and disorganized due to severe atrophy. These end-stage conditions not only impair vision but also compromise the integrity of the ocular surface.

Conjunctivitis, defined as inflammation of the conjunctiva, is a common cause of red eye and may result from infectious or non-infectious etiologies.⁵ However, the occurrence of conjunctivitis in eyes with absolute glaucoma and phthisis bulbi presents a unique clinical challenge. Ocular surface instability, tear film abnormalities, and chronic inflammation in such conditions may mimic or coexist with infection, complicating diagnosis and management.⁶ Additionally, clinical evaluation is often hindered by altered

ocular anatomy and limited patient visual feedback.

This case report aims to describe an uncommon presentation of bilateral conjunctivitis in a patient with absolute glaucoma and phthisis bulbi sinistra, highlighting the importance of comprehensive clinical assessment to guide appropriate management and improve patient comfort while preventing further complications.

CASE REPORT

A patient came with the main complaint of both eyes being red for approximately 1 week before the examination, accompanied by epiphora and a feeling of discomfort like something is stuck. The complaints are felt continuously, without significant purulent discharge. The patient also complained of intermittent mild pain in the right eye. The medical history shows that the left eye has experienced progressive vision loss for several years until it can no longer see at all. The patient has a long history of uncontrolled glaucoma and had been using topical anti-glaucoma medications for a prolonged period; however, adherence to treatment was irregular. Detailed information regarding the specific agents and whether the medications were preservative or preservative-free was not clearly documented.

On ophthalmological examination, visual acuity in the right eye was decreased, while the left eye had no light perception. Intraocular pressure in the right eye was elevated, whereas assessment in the left eye was limited due to structural changes consistent with phthisis bulbi. Anterior segment examination of the right eye revealed diffuse conjunctival hyperemia with minimal ciliary injection, a relatively clear cornea without epithelial defect, ulcer, or infiltrate, and a deep, quiet anterior chamber. The right eye demonstrated conjunctival hyperemia with

bluish corneal opacity and poor visualization of intraocular structures (**Image 1**). No purulent or mucopurulent discharge was observed in either eye, reducing the likelihood of bacterial conjunctivitis.

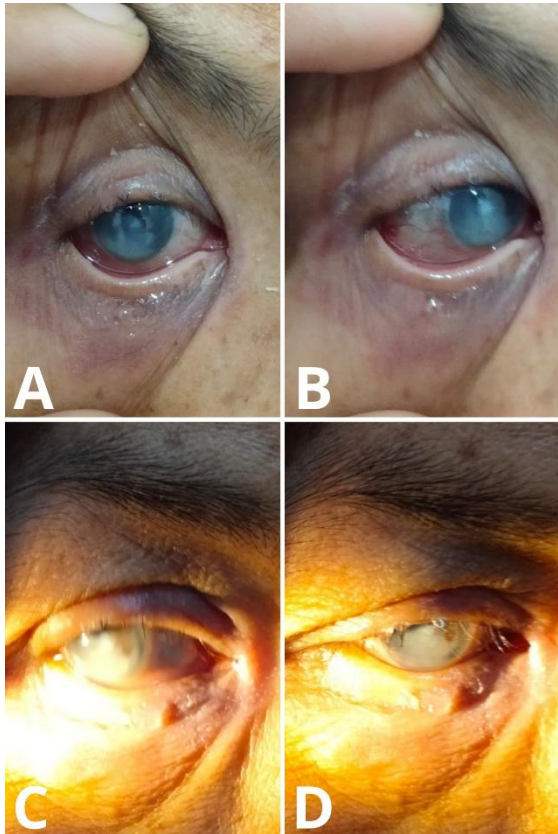


Figure 1. Clinical photographs of the right eye (oculus dextera). (A–B) External examination shows conjunctival hyperemia with bluish corneal opacity. (C–D) Focused illumination demonstrates poor visualization of intraocular structures, consistent with advanced glaucoma

Eversion of the eyelids did not reveal obvious follicles, papillae, or membrane formation. In the left eye, findings were consistent with phthisis bulbi, including a shrunken globe, irregular surface, and corneal opacity. Conjunctival hyperemia with mucosal congestion and marked structural disorganization were observed (**Image 2**). Although formal dry eye evaluation such as Tear Break-Up Time (TBUT), fluorescein staining, and Schirmer test was not performed, tear film instability

was clinically suspected based on epiphora and ocular surface irritation. Lacrimal drainage obstruction was considered unlikely based on the absence of regurgitation or localized swelling; therefore, Anel test was not performed. Microbiological culture was not conducted, as there were no clinical features suggestive of chronic or purulent infection.

The patient had a history of long-term use of topical anti-glaucoma medications; however, detailed information regarding the specific agents and preservative content was not fully available. The chronic use of such medications may have contributed to ocular surface inflammation in this case.

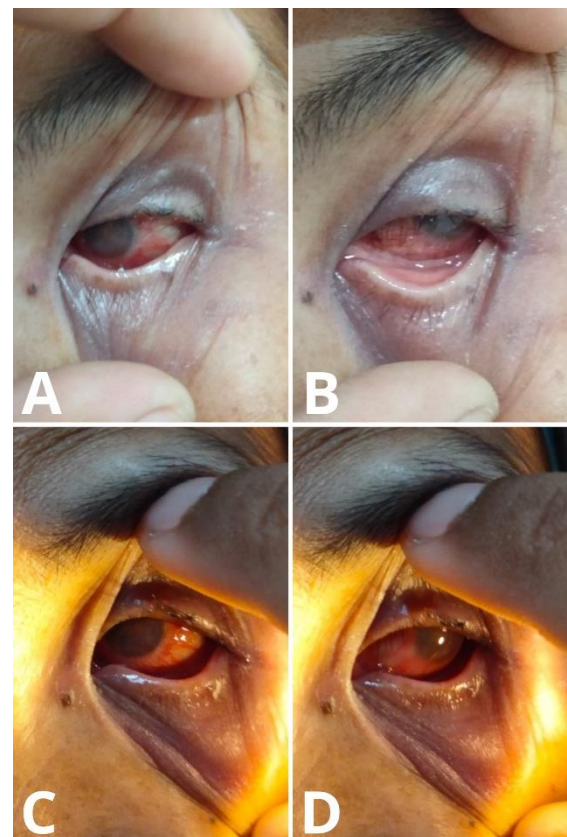


Figure 2. Clinical photographs of the left eye (oculus sinistra). (A–B) Eversion of the lower eyelid reveals conjunctival hyperemia with mucosal congestion. (C–D) Focused illumination demonstrates marked conjunctival injection and poor visualization of intraocular structures, consistent with conjunctivitis in an eye with advanced glaucomatous changes and phthisis bulbi.

An interesting finding in this case is the presence of bilateral conjunctivitis in eyes with very different conditions, namely the right eye with advanced glaucoma and the left eye with absolute glaucoma that has progressed to phthisis bulbi. This shows that ocular surface inflammation is not only caused by infection but also by tear film instability, chronic exposure, and ocular surface dysfunction in end-stage disease conditions.

Based on the anamnesis and clinical examination, the patient was diagnosed with bilateral conjunctivitis in absolute glaucoma with phthisis bulbi of the left eye. Management was provided conservatively with a focus on symptomatic relief, including preservative-free artificial tears and a mild topical corticosteroid (fluorometholone 0.1%) administered twice daily to reduce ocular surface inflammation. Education was provided regarding eye hygiene, avoiding irritants, and the importance of regular check-ups, especially to maintain the visual function of the right eye.

During the post-therapy evaluation after 2 weeks of treatment, the patient reported significant improvement with reduced redness, epiphora, and discomfort. Re-examination showed a decrease in conjunctival hyperemia in both eyes without signs of secondary infection. This case emphasizes the importance of a holistic approach in assessing red eye in end-stage glaucoma conditions, where the main therapeutic goal is not only the eradication of the cause but also the improvement of the patient's quality of life and comfort.

DISCUSSION

This case represents a complex and uncommon clinical scenario involving bilateral conjunctivitis in the setting of markedly asymmetric ocular pathology: advanced glaucoma in the right eye and absolute glaucoma complicated by phthisis

bulbi in the left eye. Glaucoma is a chronic, progressive optic neuropathy characterized by retinal ganglion cell loss and optic nerve head damage, most commonly associated with elevated intraocular pressure (IOP).⁷ In its terminal stage, absolute glaucoma is defined by complete and irreversible vision loss, often accompanied by persistent ocular pain and severe structural deterioration. Prolonged disease progression, especially when inadequately controlled, may lead to phthisis bulbi, a condition characterized by a shrunken, disorganized globe with extensive fibrosis, atrophy, and intraocular architectural collapse.⁷

The coexistence of conjunctivitis in both eyes within this context highlights an important diagnostic and pathophysiological consideration. While conjunctivitis is most frequently attributed to infectious etiologies, the absence of purulent discharge and the bilateral nature of symptoms in this patient suggest a predominantly non-infectious origin.⁸ Ocular surface disease (OSD) is increasingly recognized as a significant comorbidity in patients with chronic glaucoma.⁹ Long-term use of topical antiglaucoma medications, particularly those containing preservatives such as benzalkonium chloride (BAK), has been shown to induce cytotoxic effects on the ocular surface epithelium.¹⁰ These effects include disruption of epithelial tight junctions, apoptosis of conjunctival goblet cells, decreased mucin production, and chronic subclinical inflammation.

Tear film instability plays a central role in the pathogenesis of ocular surface inflammation in such patients. The tear film, composed of lipid, aqueous, and mucin layers, is essential for maintaining ocular surface homeostasis.¹¹ In glaucoma patients, especially those on chronic topical therapy, alterations in tear composition and reduced tear breakup time lead to increased tear osmolarity and inflammatory

mediator release.¹² This results in a self-perpetuating cycle of ocular surface damage, characterized by epithelial breakdown, increased friction during blinking, and reflex hypersecretion of tears, clinically presenting as epiphora despite underlying dryness.

In the left eye, the presence of pthisis bulbi further exacerbates ocular surface dysfunction. Structural disorganization of the globe leads to abnormal eyelid-globe apposition, impaired tear distribution, and reduced blink efficiency.¹³ Additionally, corneal opacity and stromal degeneration limit normal light transmission and reflex pathways, contributing to reduced protective mechanisms. Chronic low-grade inflammation within the pthisical eye may also contribute to persistent conjunctival hyperemia. These factors collectively predispose the eye to a state of chronic irritation that can mimic or coexist with conjunctivitis.¹⁴

The findings observed in **Image 1** and **Image 2** support this pathophysiological framework. In the right eye, conjunctival hyperemia with relatively preserved corneal clarity and anterior chamber depth suggests active ocular surface inflammation in the setting of advanced glaucoma. In contrast, the left eye demonstrates hallmark features of pthisis bulbi, including corneal opacity, globe shrinkage, and poor visualization of intraocular structures, indicating end-stage structural damage.¹⁵ The conjunctival congestion observed in both eyes is therefore more consistent with inflammatory ocular surface disease rather than acute infection.¹⁶

An important clinical implication of this case is the need for careful differentiation between infectious and non-infectious conjunctivitis in patients with chronic ocular disease.¹⁷ Misdiagnosis may lead to inappropriate use of topical antibiotics, which not only provides limited benefit but may further disrupt the ocular surface

microbiome and exacerbate epithelial toxicity. Instead, management should be directed toward restoring ocular surface homeostasis.¹⁸ In this case, the use of artificial tears aimed to improve tear film stability, dilute inflammatory mediators, and reduce mechanical friction. The addition of mild anti-inflammatory agents helps suppress surface inflammation and break the cycle of irritation.¹⁹

Another critical aspect is the disparity in visual prognosis between both eyes. The right eye retains some degree of visual potential and thus requires close monitoring and optimal IOP control to prevent further progression. In contrast, the left eye has no visual potential, and management is primarily palliative, focusing on patient comfort and prevention of complications such as chronic pain or secondary infection. This highlights the importance of individualized treatment strategies based on the functional status of each eye.

CONCLUSION

This case shows a rare and complicated case of bilateral conjunctivitis in eyes with very different end-stage glaucomatous conditions: advanced glaucoma in the right eye and absolute glaucoma with pthisis bulbi in the left eye. The results show that conjunctival inflammation in these patients isn't always caused by an infection. Instead, it is often linked to ocular surface disease caused by tear film instability, chronic inflammation, and long-term use of topical antiglaucoma medications.

A thorough clinical assessment is necessary to distinguish between infectious and non-infectious causes, as misdiagnosis can result in inadequate treatment. In this instance, conservative therapy focused on reestablishing ocular surface homeostasis utilizing artificial tears and mild anti-inflammatory agents demonstrated efficacy in alleviating symptoms and enhancing patient comfort.

It is essential that management be tailored to the visual potential of each eye. To keep the remaining vision in the right eye, it needs to be closely watched. The left eye, on the other hand, needs a palliative approach that focuses on comfort. This case emphasizes the necessity of a comprehensive and patient-centered approach in the management of red eye in advanced glaucoma to enhance quality of life and avert additional complications.

REFERENCES

1. Qi T, Liu H, Fröhn L, Löw K, Cursiefen C, Prokosch V. Understanding glaucoma: why it remains a leading cause of blindness worldwide. *Klin Monbl Augenheilkd.* 2025;242(7):712.
2. Iskandar F, Oktariana VD, Aziza Y. A case of mixed mechanism glaucoma: diagnostic and management challenges. *Med J Indones.* 2025.
3. Dietze J, Blair K, Zeppieri M, Havens SJ. Glaucoma. *StatPearls.* 2024.
4. Tripathy K, Zeppieri M. Open angle glaucoma. In: *Ferri's Clinical Advisor* 2025. 2024:473.e21–473.e23.
5. Hashmi MF, Gurnani B, Benson S. Conjunctivitis. *StatPearls.* 2024:1–9.
6. Takamura Y, Pniakowska Z, Kurys N, Pietruszewska H, Przybylak A, Jurowski P. Dry eye disease: from mechanisms to management and future directions. *J Clin Med.* 2026;15(7):2535.
7. Macanian J, Sharma SC. Pathogenesis of glaucoma. *Encyclopedia.* 2022;2(4):1803–1810.
8. Azari AA, Arabi A. Conjunctivitis: a systematic review. *J Ophthalmic Vis Res.* 2020;15(3):372.
9. Mahoney MJ, Bekibele R, Notermann SL, Reuter TG, Borman-Shoap EC. Pediatric conjunctivitis: a review of clinical manifestations, diagnosis, and management. *Children.* 2023;10(5):808.
10. Zheng X, Shawky M, Ogando DG, Nishiyama M, Ibrahim AS, Srinivas SP. Effects of benzalkonium chloride on the barrier function of human corneal epithelial cells. *Transl Vis Sci Technol.* 2025;14(11):16.
11. Chang AY, Purt B. Biochemistry, tear film. *StatPearls.* 2023.
12. Kawahara A. Treatment of dry eye disease in Asia: strategies for short tear film breakup time-type DED. *Pharmaceutics.* 2023;15(11):2591.
13. Shah SS, Patel BC. Enophthalmos. In: *Master Techniques in Ophthalmic Surgery.* 2025:769.
14. Chigbu DI, Karbach NJ, Abu SL, Hehar NK. Cytokines in allergic conjunctivitis: unraveling their pathophysiological roles. *Life (Basel).* 2024;14(3):350.
15. Li G, Akpek EK, Ahmad S. Glaucoma and ocular surface disease: more than meets the eye. *Clin Ophthalmol.* 2022;16:3641.
16. Li G, Akpek EK, Ahmad S. Glaucoma and ocular surface disease: more than meets the eye. *Clin Ophthalmol.* 2022;16:3641–3649.
17. Kim MS, Tauber J. Clinical considerations and recommended diagnostic algorithm for the differential diagnosis of conjunctivitis: a clinical practice review. *Ann Eye Sci.* 2025;10(1).
18. Tariq F, Hehar NK, Chigbu DGI. The ocular surface microbiome in homeostasis and dysbiosis. *Microorganisms.* 2025;13(9):1992.
19. Kuchar E, Karlikowska-Skwarnik M, Wawrzuta D. Anti-inflammatory therapy of infections. *Encyclopedia of Infection and Immunity.* 2022;4:791.