Case Report

Unilateral Absolute Open-Angle Glaucoma with Ocular Surface Disease in Juvenile Systemic Lupus Erythematosus: A Case Report

PJO – Official Journal of Ophthalmological Society of Pakistan

Chandra Prabaswara[1], Evelyn Komaratih[2], Yulia Primitasari[3]
Ismi Zuhria[4]
[1-4]Dr. Soetomo General Academic Hospital and Medical Faculty Universitas Airlangga, Indonesia

ABSTRACT

Systemic lupus erythematosus (SLE) is an autoimmune condition with many heterogeneous ocular manifestations while glaucoma is a rare sight-threatening disease, particularly in juvenile cases. We present a case involving a 15-year-old girl with juvenile-onset SLE and vasculitis, developed unilateral absolute open-angle glaucoma with recurrent ocular surface disease. The patient presented with no light perception in the left eye, and severely elevated intraocular pressure (IOP) despite maximum medical therapy with her right eye unaffected. Anterior segment and retinal imaging demonstrated advanced open-angle glaucoma. The affected eye developed bullous keratopathy, recurrent hyphema, hemosiderosis, blepharitis, keratitis, and band keratopathy. Cyclocryotherapy was planned but deferred after the affected eye became painless. This is an unusual presentation of unilateral absolute glaucoma secondary to juvenile SLE, likely due to inflammatory and steroid-related effects, highlighting the need for multidisciplinary care to assist detection to avoid irreversible vision loss.

Keywords: Systemic Lupus Erythematosus, Glaucoma, Secondary Open-Angle Glaucoma, Corneal Diseases, Dry Eye Syndromes, Child / Adolescent.

How to Cite this Article: Prabaswara C, Komaratih E, Primitasari Y, Zuhria I. Unilateral Absolute Open-Angle Glaucoma with Ocular Surface Disease in Juvenile Systemic Lupus Erythematosus: A Case Report. 2026;42(3):348-352 **Doi:10.36351/pjo.v42i3.2347**

Correspondence: Evelyn Komaratih
Dr. Soetomo General Academic Hospital and Medical Faculty Universitas Airlangga, Indonesia
Email: evelyn.komaratih@fk.unair.ac.id

Received: January 10, 2026
Revised: April 20, 2026
Accepted: June 24, 2026

INTRODUCTION

Systemic lupus erythematosus (SLE) is a long-term autoimmune condition where the immune system attacks its own tissues, leading to formation of autoantibodies and the buildup of immune complexes, which cause inflammation in multiple organs and systems. Ocular involvement is present in about one-third of patients and can affect almost every part of the eye, such as the conjunctiva, sclera, retina, choroid, and optic nerve.[1,2] Common manifestations include keratoconjunctivitis sicca, episcleritis, retinal vasculitis, and optic neuropathy, while glaucoma remains an uncommon but sight-threatening complication.[3]

In juvenile-onset SLE (JSLE), ocular manifestations are generally more aggressive and associated with higher systemic disease activity compared to adults.[2] The pathophysiology of secondary glaucoma in lupus involves multiple mechanisms including chronic uveal inflammation, immune complex deposition in the trabecular meshwork, corticosteroid-induced trabecular dysfunction, and vascular compromise leading to impaired aqueous outflow.[1,4] Although corticosteroids remain the cornerstone of SLE therapy, they may cause steroid-induced ocular hypertension and glaucoma, especially in children and genetically

predisposed individuals.[5,6] Such secondary open-angle glaucoma may progress silently to end-stage absolute glaucoma if not recognized early.

Reports of absolute glaucoma in pediatric or juvenile SLE are extremely rare. We report a case of a 15-year-old female with JSLE and vasculitis, the patient developed unilateral absolute open-angle glaucoma with recurrent ocular surface disease. This case report emphasizes the importance of routine eye examinations in children with SLE and the importance of a multi-disciplinary approach to preserve eyesight.

Case Presentation

A 15-year-old girl who was diagnosed with SLE and vasculitis presented to ophthalmology clinic with gradual loss of vision in her left eye. Her systemic course was notable for recurrent lupus flares since early 2024, presenting with fever, polyarthralgia, alopecia, malaise, and vasculitis. A positive family history was documented, as her elder sibling was also diagnosed with SLE. She had been under rheumatology care and was maintained on long-term immunosuppressive therapy, including corticosteroids and mycophenolate sodium (Myfortic), with partial control of systemic disease. Serial laboratory tests showed consistently positive results for antinuclear antibodies (ANA) and anti–double stranded DNA antibodies, along with low levels of complement components C3 and C4. Evidence of lupus nephritis was present in the form of intermittent elevations of serum creatinine and blood urea nitrogen, as well as proteinuria. Hematologic abnormalities included mild anemia during flares and occasional leukocytosis.

Eye examination in October 2024 found that her best-corrected visual acuity (BCVA) was 6/6 in the right eye, but she had no light perception in the left eye. The right eye showed no unusual findings, but the left eye had unequal pupil size, with a dilated and slow-reacting pupil, and signs of retinopathy. The IOP was 17 mmHg in the right eye and 33 mmHg in the left eye. She was started on topical timolol 0.5% and lubricants. Despite maximally tolerated topical therapy with timolol, Brinzolamide (Glopac), and corticosteroid drops, the IOP remained uncontrolled, reaching up to 46.7 mmHg. By November 2024, slit-lamp examination revealed conjunctival injection, a deep anterior chamber, mid-dilated pupil, and tractional retinal detachment in the left eye. In March 2025, she reported ocular discomfort in the same eye, which showed conjunctival hyperemia, minimal corneal edema, and glaucomatous optic neuropathy, while right eye remained stable with preserved vision and IOP control.

During follow-up, the left eye deteriorated. Initially, the vision was 6/6 in the right eye and doubtful light perception in the left eye with IOP of 16 mmHg and 5 mmHg, respectively. Left eye revealed bullous keratopathy, hyphema, hemosiderosis, a maximally dilated non-reactive pupil, and a cataractous lens with no fundus view (Figure 1). Conservative management was given. Two weeks later, she visited the emergency room with redness, tearing, burning, and difficulty in opening left eye. Vision in left eye was No light perception with IOP of 37 mmHg. There was eyelid edema, conjunctival hyperemia, bullous and band keratopathy, hyphema with clot, and cataract. USG B-scan is shown in Figure 2. She was managed medically and counseled for cyclocryotherapy with close follow-up.

By mid-2025, the left eye had progressed to a painful blind eye with IOP of >40 mmHg, accompanied by bullous keratopathy, hyphema, and hemosiderosis. Although cyclocryotherapy was recommended as a palliative measure for pain control, during subsequent reviews, the patient reported spontaneous reduction in ocular pain and improved comfort. The right eye remained stable with BCVA 6/6 and IOP ~15 mmHg on topical timolol.

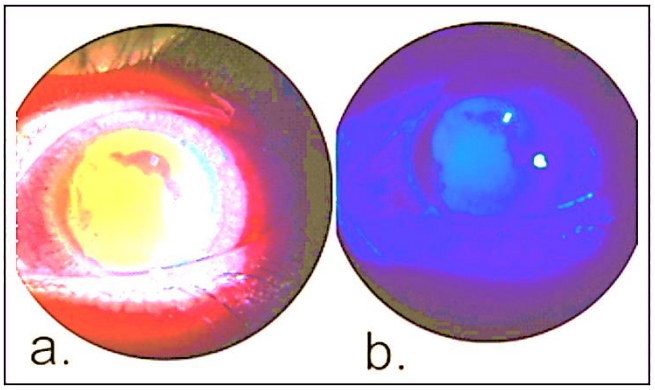

Figure 1: Slit-lamp biomicroscopy of the left eye. (a) Diffuse illumination shows corneal edema with bullous changes, central stromal infiltrates, hypopyon, conjunctival and pericorneal hyperemia, and coagulum with hemosiderosis in the anterior chamber (Van Herick grade II). Lens opacity. (b) Fluorescein staining with a cobalt blue filter demonstrates multiple punctate epithelial defects, diffusely located in the corneal epithelium.

Optical coherence tomography (OCT) and anterior segment (Figure 3) imaging in May 2025 showed

average RNFL thickness of 105 µm, preserved macular contour, and cup–disc ratio of 0.49, consistent with intact neuroretinal rim in the right eye. Anterior chamber depth was 2.72 mm with central corneal thickness of 577 µm and open angles. ONH angiography demonstrated normal radial peripapillary capillary perfusion (47–49%). In contrast, OS had a markedly thickened cornea (694 µm) and open angles, but no usable RNFL or optic nerve head values due to media opacity and end-stage glaucoma.

In August 2025, the patient remained stable under rheumatology care. The right eye had well controlled IOP and good vision. The left eye, though blind but without pain.

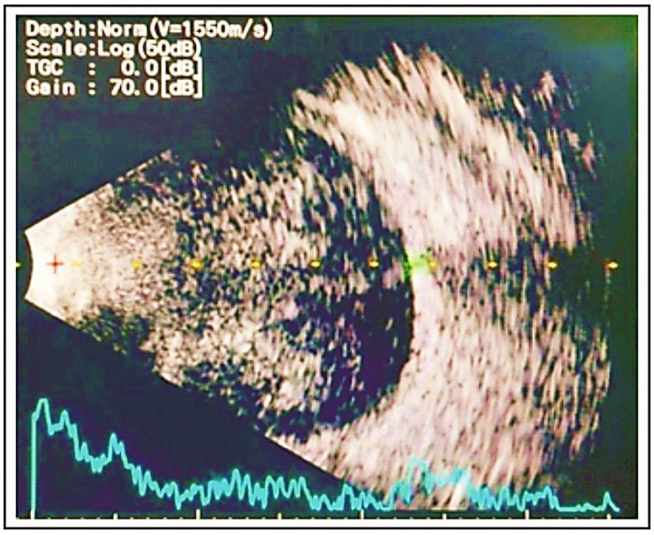

Figure 2: B-scan ultrasonography of the left eye. Diffuse echogenicity with low mobility in vitreous, filling the entire vitreous cavity. Echospike intensity 30–60%. Detachment of retina. Images compatible with vitritis, differential diagnosis vitreous hemorrhage.

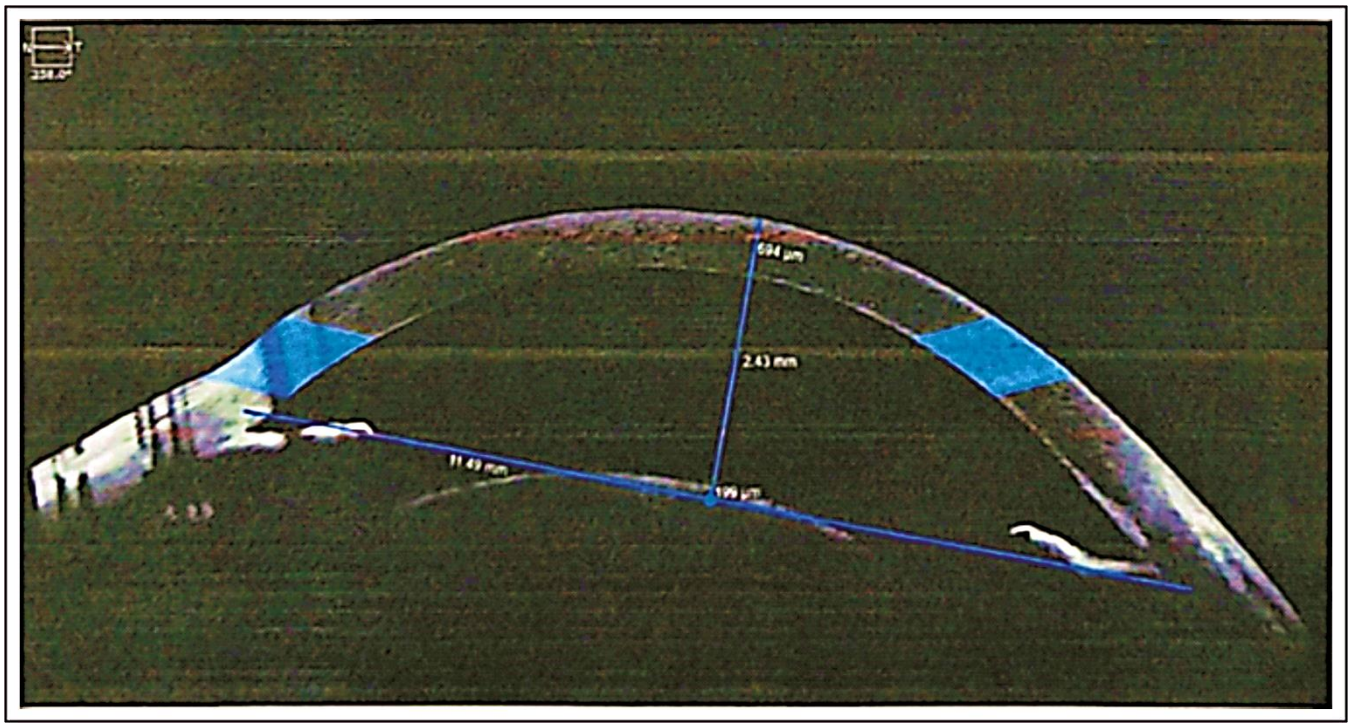

Figure 3: Anterior Segment Optical Coherence Tomography (AS-OCT) of the left eye.

DISCUSSION

The case highlights a rare presentation of unilateral absolute glaucoma with recurrent ocular surface complications in a 15-year-old female with JSLE. Ocular involvement in SLE occurs in up to one-third of patients, most frequently presenting as keratoconjunctivitis sicca, retinal vasculitis, and optic neuropathy.[1,3]However, secondary open angle glaucoma leading to absolute glaucoma in a JSLE pediatric population is still exceptionally rare. Case report strengths include detailed longitudinal ophthalmic records, multimodal imaging (anterior segment OCT and OCT RNFL), and correlation with systemic disease activity. The main limitation is the

absence of histopathologic confirmation and limited systemic immunological data, which are inherent to case-based observational studies.

The pathogenesis of ocular hypertension and glaucoma in JSLE may involve multiple mechanisms: immune complex deposition in the trabecular meshwork, steroid-induced trabecular dysfunction, or chronic inflammation of anterior segment structures.[7,8] In our patient, long-term corticosteroid therapy and recurrent anterior uveitis episodes likely acted synergistically to induce irreversible optic nerve damage, consistent with steroid-induced glaucoma mechanisms described in literature.[5-7] The open-angle configuration demonstrated on anterior segment OCT further supports a secondary open-angle pathophysiology rather than inflammatory angle closure.

Ocular surface complications including bullous keratopathy, hyphema, hemosiderosis, and band keratopathy may develop in end-stage glaucomatous eyes due to chronic epithelial stress, hypoxia, and altered corneal metabolism. Similar corneal findings have been observed in chronic ocular inflammation associated with lupus keratopathy and dry eye disease.[3,8] Furthermore, the development of ocular surface inflammation during follow-up aligns with the literature describing the interplay between systemic inflammation and local immune dysregulation in lupus-related keratopathy.[2]

Although most ocular SLE lesions are bilateral, glaucoma was unilateral. Nikolaidou et al, found that between 18% and 44% of ocular JSLE lesions are unilateral at presentation.[2] However, in this case, asymmetric local steroid exposure and asymmetric microvascular injuries may have caused unilateral trabeculum damage. This does not exclude a systemic etiology.

The treatment of absolute glaucoma in lupus patients is challenging. Cyclodestructive procedures such as cyclocryotherapy are performed in a palliative setting for pain relief.[1] However, in this case, the procedure was not performed as the ocular pain responded adequately to medical management per the guidelines of avoiding destructive procedures where comfort and cosmesis is preserved. The management of the patient thereafter involved surface protection with lubricants, treating any infection and counselling the patient. This is in keeping with the paradigm shift of conservatism in the management of end-stage pediatric glaucoma.

CONCLUSION

Unilateral absolute glaucoma secondary to juvenile SLE is rare but associated with high morbidity. Steroid-induced IOP elevation should be diagnosed early and corticosteroids tapered to avoid bilateral irreversible blindness. Even after control of IOP, ocular surface changes may persist. A multidisciplinary approach and long-term followup is needed.

Funding: This study was not funded by any organization.

Patient's Consent: Researchers followed the guidelines set forth in the Declaration of Helsinki.

Conflict of Interest: Authors declared no conflict of interest.

REFERENCES

1. **Silpa-Archa S, Lee JJ, Foster CS.** Ocular manifestations in systemic lupus erythematosus. Br J Ophthalmol. 2016;**100(1):**135-141. Doi:10.1136/bjophthalmol-2015-306629
2. **Nikolaidou A, Gianni T, Sandali A, Toumasis P, Benekos K, Tsina E.** Ocular manifestations of juvenile systemic lupus erythematosus: a systematic review. Eye (Lond). 2025;**39(6):**1056-1069. Doi:10.1038/s41433-025-03664-x
3. **Palejwala NV, Walia HS, Yeh S.** Ocular manifestations of systemic lupus erythematosus: a review of the literature. Autoimmune Dis. 2012;**2012:**290898. Doi:10.1155/2012/290898
4. **Wang X, Xie H, Yi Y, Zhou J, Yang H, Li J.** Clinical research of lupus retinopathy: quantitative analysis of retinal vessels by optical coherence tomography angiography in patients with systemic lupus erythematosus. Diagnostics (Basel). 2023;**13(20):**3222. Doi:10.3390/diagnostics13203222
5. **Roberti G, Oddone F, Agnifili L, Katsanos A, Michelessi M, Mastropasqua L, et al.** Steroid-induced glaucoma: Epidemiology, pathophysiology, and clinical management. Surv Ophthalmol. 2020;**65(4):**458-472. Doi: 10.1016/j.survophthal.2020.01.002.
6. **Lai HY, Lai IC, Fang PC, Hsiao CC, Hsiao YT.** Steroid-induced ocular hypertension in a pediatric patient with acute lymphoblastic leukemia: a case report. Children (Basel). 2022;**9(3):**440. Doi:10.3390/children9030440
7. **Sihota R, Konkal VL, Dada T, Agarwal HC, Singh R.** Prospective, long-term evaluation of steroid-induced glaucoma. Eye (Lond). 2008;**22(1):**26-30. Doi:10.1038/sj.eye.6702474

8. **Kedia N, Theillac V, Paez-Escamilla M, Indermill C, Gallagher DS, Adam R, et al.** The full range of ophthalmological clinical manifestations in systemic lupus erythematosus. Front Ophthalmol (Lausanne). 2023;**2:**1055766. Doi: 10.3389/fopht.2022.1055766.

Authors Designation and Contribution

Chandra Prabaswara; General Practician: *Design, Literature Search, Data Acquisition, Manuscript Preparation, Manuscript Editing.*

Evelyn Komaratih; Consultant Ophthalmologist: *Concepts, Design, Data Acquisition, Manuscript Review.*

Yulia Primitasari; Consultant Ophthalmologist: *Concepts, Design, Data Acquisition, Manuscript Review.*

Ismi Zuhria; Consultant Ophthalmologist: *Design, Literature Search, Manuscript Preparation, Manuscript Review.*