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CASE REPORT

Exceptionally delayed sympathetic ophthalmia diagnosed by multimodal imaging: A 36-year latency case report

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Abstract

Sympathetic ophthalmia (SO) is a rare bilateral granulomatous uveitis that typically appears weeks to months after penetrating ocular trauma; presentations decades later are exceptional. A 40-year-old man developed blurred vision in his left eye thirty-six years after childhood penetrating ocular trauma to the right eye. Best-corrected visual acuity was no light perception in the right eye and counting fingers at two meters in the left eye. Multimodal imaging revealed diffuse choroidal thickening as well as subfoveal serous detachment. Laboratory work-up excluded infectious and malignant etiologies. High-dose intravenous methylprednisolone, followed by oral prednisone and azathioprine, led to the resolution of subretinal fluid and improvement to 20/20 at 6 months, sustained over 18 months. This case underscores the lifelong risk of SO after ocular injury and illustrates the diagnostic value of multimodal imaging in detecting choroidal inflammation, guiding treatment response monitoring, and long-term management.

Keywords: Sympathetic ophthalmia; optical coherence tomography; multimodal imaging; uveitis; ocular trauma.

SO is a rare bilateral granulomatous panuveitis that may appear weeks to decades after penetrating ocular injury or surgery.[1] Latencies beyond 30 years are exceptional and often delay diagnosis because clinicians and patients underestimate the lifelong risk.[2] Modern multimodal imaging (combining fluorescein angiography, enhanced-depth imaging OCT (EDI-OCT), and OCT angiography (OCT-A)) allows non-invasive visualization of choroidal inflammation and objective tracking of treatment response.[3]

We report a 40-year-old man who developed acute SO 36 years after childhood trauma to the right eye. Prompt image-guided immunosuppression led to full visual recovery and sustained remission over 18 months. This case highlights the need for lifelong vigilance in monocular patients and illustrates how quantitative imaging endpoints refine the management of very-late-onset SO.

Case Report

A 40-year-old Iranian man presented with a 3-day history of progressive visual blurring, photophobia, and mild ocular discomfort in his left eye. His ophthalmic history was significant for penetrating corneoscleral trauma to the right eye at age 4, resulting in permanent visual loss.

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He denied recent ocular trauma, intraocular surgery, or systemic illness.

On presentation, best-corrected visual acuity (BCVA) measured no light perception in the right eye and counting fingers at 2 meters in the left eye. Intraocular pressure (IOP) was 14 mmHg bilaterally by Goldmann applanation tonometry. Slit-lamp examination of the traumatized right eye revealed a vascularized leucomatous cornea with mutton-fat keratic precipitates and a mature white cataract. B-scan ultrasonography confirmed retinal attachment. The left eye demonstrated granulomatous anterior uveitis with mutton-fat keratic precipitates, 1+ anterior chamber cells, and 1+ vitreous cells. Dilated fundus examination of the left eye revealed multiple dome-shaped serous retinal detachments involving the macula. Additionally, an inferotemporal yellow-white placoid lesion with characteristic radial chorioretinal folds was observed (Fig. 1a–b).

Spectral-domain OCT revealed extensive multilobular serous retinal detachment with subretinal fibrin deposits and central retinal thickness (CRT) of 560 µm (Fig. 2a). En-face OCT-A using an 8 × 8 mm scan pattern demonstrated confluent choriocapillaris flow voids throughout the posterior pole. Mid-phase fluorescein angiography showed the pathognomonic pattern of multiple pinpoint retinal pigment epithelium (RPE) leaks with subsequent lobular pooling in areas of serous detachment. Blue-light fundus autofluorescence (FAF) imaging revealed a crescent-shaped parafoveal hyper-autofluorescent plaque (Fig. 3a–b).

All systemic and uveitis work-up, including laboratory tests, chest radiography, and brain MRI, was unremarkable.

Based on the clinical presentation and imaging findings consistent with SO, immunosuppressive therapy was initiated promptly. The treatment regimen consisted of intravenous methylprednisolone (1 g/day) for three days, followed by oral prednisone (60 mg/day, 1 mg/kg) with subsequent tapering. Steroid-sparing therapy with oral azathioprine (150 mg/day, 2 mg/kg) was initiated after confirming normal thiopurine methyltransferase activity. Topical therapy included prednisolone acetate 1% every 6 hours and cyclopentolate 1% twice daily for 4 weeks.

Rapid clinical improvement was observed within the first week, with BCVA improving to 20/60 and CRT decreasing by 32%. By week 4, complete resolution of serous retinal detachment occurred, and OCT-A demonstrated near-complete choriocapillaris reperfusion, allowing prednisone reduction to 20 mg daily.

At the three-month follow-up, best-corrected visual acuity had improved to 20/25, and multimodal imaging confirmed

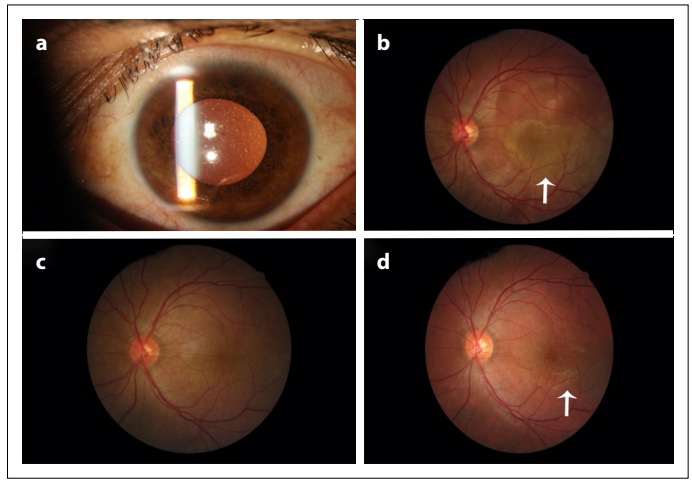

Fig. 1. Clinical course of the sympathizing eye. **(a)** Presentation: mutton-fat keratic precipitates, clear media. **(b)** Exudative macular detachment with inferotemporal subretinal patch (white arrow) and retinal folds. **(c)** Week 4: fluid resolved, macula reattached, mild pigment mottling. **(d)** Month 6: quiet fundus; small inferotemporal retinal scar (white arrow), no recurrent subretinal fluid or active fibrovascular tissue.

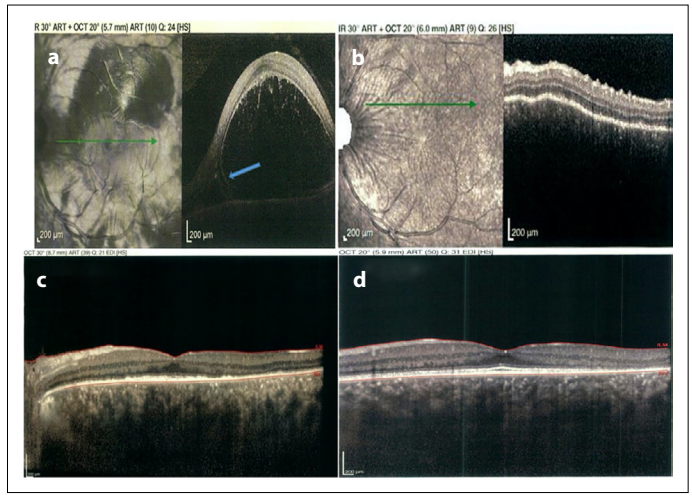

Fig. 2. Spectral-domain OCT evolution on horizontal foveal scans. **(a)** Baseline: multifocal serous detachment with thick subretinal fluid and a fibrinous septum (blue arrow); undulating RPE, apparent choroidal thickening. **(b)** Week 1: partial fluid resorption, chorioretinal folds. **(c)** Week 4: retina reattached; temporal ellipsoid zone (EZ) loss. **(d)** Month 6: attachment stable; central EZ recovered, temporal EZ thinning, and outer nuclear attenuation, matching persistent FAF hyperautofluorescence.

complete anatomical restoration. Visual acuity stabilized at 20/20 by month 6 and remained unchanged through 18 months of follow-up. Despite vascular normalization, a parafoveal hyper-autofluorescent plaque persisted on FAF imaging (Figure 3d), representing the "persistence of memory" phenomenon. Serial OCT-A and near-infrared reflectance imaging confirmed the absence of recurrent inflammatory activity or fluid accumulation (Fig. 4c, 3c–e).

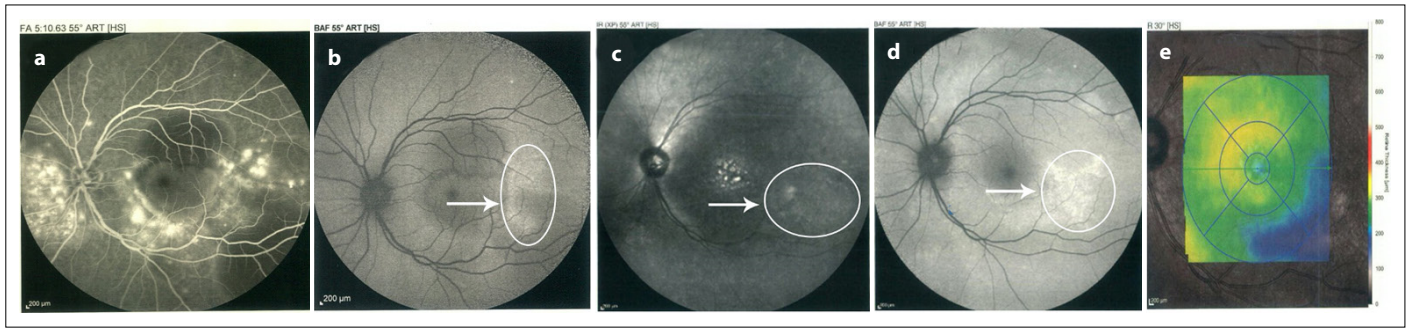

Fig. 3. Multimodal imaging baseline and 6-month follow-up. **(a)** Baseline mid-phase FA: multiple pinpoint RPE leaks with lobular pooling. **(b)** Baseline FAF: parafoveal hyperautofluorescent crescent. **(c)** Month 6 near-infrared reflectance: residual hyperreflective dots. **(d)** Month 6 FAF: mottled hyperautofluorescent patch showing "persistence of memory" lesion. **(e)** Month 6 thickness map: focal inferotemporal thinning (205 µm).

The traumatized right eye remained quiescent throughout the treatment period, negating the need for enucleation. A detailed clinical timeline, therapeutic interventions, and treatment outcomes are comprehensively outlined in Table 1.

Discussion

SO is an autoimmune bilateral granulomatous uveitis triggered by penetrating trauma or surgery to one eye. The release of retinal and uveal antigens during the initial injury triggers a T cell–mediated delayed hypersensitivity response. The time from injury to disease onset can vary greatly, ranging from just a few days to as long as 66 years.[4] Delays exceeding 30 years, as observed in our patient, account for approximately 3–4% of reported cases, underscoring the critical need for lifelong counseling in monocular patients.[5,6]

Historically, fluorescein angiography showing multiple pinpoint leaks was considered pathognomonic for SO, but multimodal imaging now refines both diagnosis and follow-up monitoring. EDI-OCT and OCT-A objectively quantify inflammatory exudation and choriocapillaris non-perfusion.[7] In our case, CRT decreased by 32% within one week of treatment initiation, and OCT-A flow deficits demonstrated reperfusion by week 4, enabling safe steroid tapering. Serial CRT measurements may therefore serve as an accessible biomarker of disease activity, complementing qualitative angiographic patterns.

Fundus autofluorescence completed the structural-functional assessment. A parafoveal hyper-autofluorescent plaque persisted at 6 months despite vascular normalization (Figure 3b,d), replicating the "persistence of memory" lesion first described by Wilkins et al.[8] Fundus autofluorescence provides a map of cumulative photoreceptor or retinal pigment epithelium (RPE) damage, while OCT angiography reflects ongoing inflammatory activity. This dual-metric strategy has been advocated by Parchand et al. to refine tapering decisions and avoid overtreatment.[9]

Corticosteroids remain the first-line therapy for SO. However, prolonged high-dose exposure increases the risks of cataract formation, secondary glaucoma, osteoporosis, and systemic toxicity. To minimize steroid-related adverse effects, immunomodulatory agents such as azathioprine, methotrexate, or mycophenolate mofetil are commonly introduced early in the treatment course.[9,10] Azathioprine was selected over mycophenolate mofetil because both agents demonstrate comparable efficacy in SO,[5] yet azathioprine is substantially more cost-effective,[9] can be monitored with routine thiopurine methyltransferase testing in our clinical setting, and has an extensive safety profile in our healthcare environment. Biologic agents such as adalimumab may be considered for refractory cases, although their use is often limited by cost and availability. The differential diagnosis included Vogt-Koyanagi-Harada (VKH) disease, posterior scleritis, idiopathic uveal effusion, sarcoidosis, syphilis, and primary intraocular lymphoma.

The primary alternative diagnosis was VKH disease. Several clinical features favored SO over VKH: documented penetrating ocular trauma, absence of VKH-related systemic manifestations (alopecia, vitiligo, dysacusis, headache), normal neuroimaging, and characteristic histological patterns. SO demonstrates epithelioid granulomas clustered around uveal melanocytes with Dalen-Fuchs nodules, whereas VKH exhibits a more diffuse choroidal infiltrate and, in chronic stages, a characteristic sunset-glow fundus appearance.[2,9] Posterior scleritis was excluded by the absence of significant ocular pain and lack of T-sign on B-scan ultrasonography, while normal axial length and pars plana configuration excluded idiopathic uveal effusion syndrome.

Our report is limited by its single-case design and 18-month follow-up period; extended observation is necessary to confirm sustained remission and successful drug-sparing therapeutic taper. Nonetheless, this case offers three

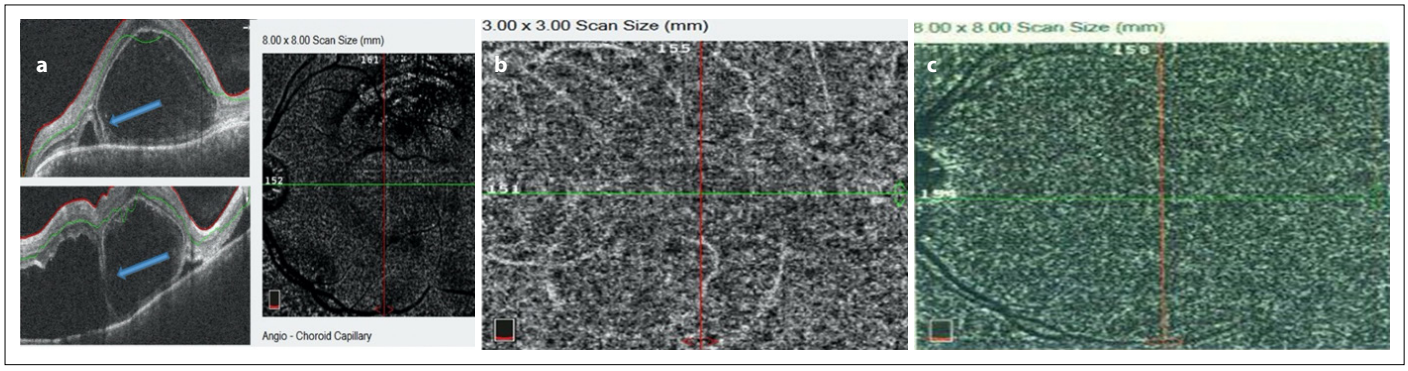

Fig. 4. OCT-angiography of the choriocapillaris. **(a)** Baseline 8 × 8 mm slab shows confluent macular and peripapillary flow voids. **(b)** Week 4, 3 × 3 mm scan demonstrates near-complete reperfusion; only scattered pinpoint deficits remain. **(c)** Month 6, repeat 8 × 8 mm imaging reveals a homogeneous granular flow signal with no new voids.

Table 1. Summary of diagnostic findings, therapeutic interventions, and clinical course during an 18-month follow-up

Time Point	Clinical Event	Intervention	Outcome
Day 0 (Presentation)	3-day history of blurred vision; BCVA counting fingers at 2 m (OS); CRT 560 µm on OCT; extensive choroidal flow voids on OCT-A	Intravenous methylprednisolone 1g daily for 3 days, transitioned to oral prednisone 60mg daily (tapered); azathioprine 150mg daily; topical prednisolone acetate 1% q6h and cyclopentolate 1% bid	Initiation of systemic and local immunosuppressive therapy
Week 1	BCVA improved to 20/60 (OS); CRT reduced by 32%	Continuation of initial regimen	Early anatomical and functional response
Week 4	BCVA 20/40 (OS); near-complete cho-roidal reperfusion on OCT-A	Prednisone reduced to20 mg daily; azathioprine continued	Gradual steroid taper enabled by imaging response
Month 3	BCVA 20/25 (OS); complete retinal reattachment; no subretinal fluid	Ongoing steroid taper; azathioprine maintenance	Transition to mainte-nance phase
Month 6	BCVA 20/20 (OS); stable multimodal imaging findings	Prednisone ≤5mg daily; azathioprine maintained	Clinical remission achieved
Month 18	BCVA 20/20 (OS); no relapse or recurrence	Discontinuation of prednisone; ongoing azathioprine maintenance	Sustained remission without recurrence

BCVA, best-corrected visual acuity; OD, right eye; OS, left eye; OCT, optical coherence tomography; CRT, central retinal thickness; OCT-A, optical coherence tomography angiography.

practical clinical lessons:[1] very late-onset SO remains a significant threat decades after initial injury, necessitating lifelong vigilance,[2] quantitative CRT measurements combined with OCT-A can guide immunosuppressive therapy more objectively than clinical grading alone, and[3] early combination immunosuppressive therapy can achieve rapid visual recovery without resorting to enucleation of the affected eye.

The exceptional 36-year latency period in our patient emphasizes the importance of patient education and long-term ophthalmologic surveillance in individuals with a history of penetrating ocular trauma. Healthcare providers

must maintain high clinical suspicion for SO development, regardless of the interval since initial injury.

Conclusion

This case emphasizes that SO remains a lifelong threat following penetrating ocular trauma, capable of manifesting decades after the initial injury. The integration of contemporary multimodal imaging techniques has transformed both diagnostic capabilities and therapeutic monitoring, providing objective quantitative measures that optimize immunosuppressive management. Despite an extraordinary 36-year latency period, prompt image-guided intervention achieved complete visual recovery, demonstrating that excellent outcomes remain attainable even in exceptionally delayed presentations. These findings reinforce the critical importance of sustained clinical vigilance and patient education in individuals with a history of penetrating ocular trauma.

Ethics Statement and Patient Consent

Written informed consent for the publication of anonymized clinical data and images was obtained from the patient in accordance with the Declaration of Helsinki (2013 revision). According to the regulations of the Tehran University of Medical Sciences institutional review board, single-patient case reports do not require formal ethics committee approval.

Informed Consent: Written informed consent was obtained from the patient for the preparation of this work.

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