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Posterior Scleritis With Secondary Uveal Effusion Unmasking Peripapillary Pachychoroid Syndrome

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Short Report

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Abstract

Purpose

To describe a case of posterior scleritis with secondary uveal effusion in which treatment of the underlying inflammatory process unmasked a coexisting peripapillary pachychoroid syndrome (PPS).

Methods

Report of a 71-year-old patient presenting with decreased visual acuity (20/100) and periocular pain in the right eye (OD). Examination included best-corrected visual acuity (BCVA), slit-lamp and dilated fundus examination, spectral-domain optical coherence tomography (SD-OCT), fundus autofluorescence (FAF), fluorescein angiography (FA), indocyanine green angiography (ICGA), and contrast-enhanced orbital magnetic resonance imaging (MRI). Laboratory testing was performed. High-dose intravenous methylprednisolone was followed by an oral taper, as well as systemic immunosuppression with subcutaneous methotrexate.

Results

At presentation, OD showed disc edema, macular serous detachment, and four-quadrant choroidal detachments; the left eye (OS) was asymptomatic with the exception of choroidal folds. MRI demonstrated posterior scleral enhancement and confirmed choroidal detachments. FA showed mottled hyperfluorescence without vasculitis; ICGA showed no choriocapillaritis or stromal granulomas. Following intravenous corticosteroids there was symptomatic improvement, BCVA increased to 20/32, and choroidal detachments resolved. Three weeks later, despite resolution of subretinal fluid, bilateral peripapillary intraretinal fluid/schisis developed. Considering short, hyperopic ocular anatomy, choroidal folds, peripapillary thickening, and steroid exposure, PPS within the pachychoroid spectrum was considered. Gradual corticosteroid de-escalation led to bilateral fluid resolution and final BCVA of 20/25 OD 20/20 OS.

Conclusion

Posterior scleritis may coexist with PPS, and treatment with corticosteroids may worsen pachychoroid syndrome. Multimodal imaging plays a critical role in distinguishing posterior scleritis from granulomatous choroiditis and is essential in leading to a prompt diagnosis and treatment.

Introduction

Choroidal detachment involve a wide range of inflammatory, neoplastic or infectious, etiologies. Posterior scleritis is a recognized entity, although it implies a diagnostic challenge due to its clinical

overlap with disorders such as Vogt–Koyanagi–Harada disease, sympathetic ophthalmia or even choroidal metastasis. On the other hand, pachychoroid spectrum disorders—including peripapillary pachychoroid syndrome (PPS)—may present with peripapillary intraretinal fluid or schisis which are characteristically very steroid-sensitive. We report a patient with posterior scleritis presenting with secondary uveal effusion, in whom subsequent bilateral peripapillary fluid during corticosteroid therapy led to the recognition of underlying PPS. The coexistence of inflammatory scleral disease and pachychoroid spectrum disorders has been rarely reported, despite potential therapeutic interferences.

Report of a case.

A 71-year-old man with a history of uneventful bilateral cataract surgery, and medical history notable for diabetes, hypertension, dyslipidemia presented with two weeks of decreased vision and periocular pain in the right eye (OD). Best corrected visual acuity (BCVA) was 20/100 OD and 20/20 in the left eye (OS). Mild anterior chamber inflammation was noted in OD; intraocular pressures (IOP) were normal in both eyes (OU). Fundus examination of the OD showed optic nerve edema and a macular serous detachment, as well as four-quadrant choroidal detachments (Fig. 1A). OS was asymptomatic but showed choroidal folds, which had been documented during previous routine examinations in OU. Macular optic coherence tomography (OCT) of OD confirmed a serous macular detachment with peripapillary intraretinal fluid (IRF) (Fig. 1B); OS was notable for the presence of a pigment epithelial detachment (PED) in the inferior macular region (Fig. 1C-D). Fundus autofluorescence (FAF) displayed areas of patchy hyperautofluorescence which could correspond to the subretinal fluid (SRF) in the OD and to the PED in OS. Fluorescein angiography (FA) revealed mottled hyperfluorescence but without evidence of vasculitis or arterial occlusions; indocyanine green angiography (ICGA) showed no choriocapillaritis or stromal nodules. B-scan ultrasonography confirmed choroidal thickening greater than 2 mm in OD, consistent with posterior scleritis. Orbital MRI demonstrated posterior scleral enhancement and confirmed the presence of choroidal detachments (Fig. 2A-B). Laboratory testing was unremarkable except for a positive interferon-γ release assay and HLA-B27. Chest X-ray was within normal limits. Assuming posterior scleritis with secondary uveal effusion, intravenous methylprednisolone pulses (500mg for 3 days) were administered and transitioned to oral taper. At one week, the patient reported symptomatic improvement; BCVA increased to 20/32 in OD. Macular OCT displayed resolution of the serous retinal detachment and reduction of the IRF.

At three weeks, despite complete resolution of SRF in OD, the patient reported new visual disturbances in OS, with a confirmed BCVA of 20/32 OD and 20/25 OS. OCT demonstrated increased peripapillary IRF/schisis along the papillomacular bundle in OU (Fig. 3A-B). Retrospective review of preoperative biometry disclosed short axial lengths (21.40 mm (OD) and 21.32 mm (OS)), with high-power posterior chamber intraocular lenses (+ 27.5D OD, + 28.0D OS), consistent with hyperopic, short eyes. In the context of baseline choroidal folds, peripapillary choroidal thickening with a chronic peripapillary PED, and recent high dose systemic corticosteroid exposure, these findings were suggestive of PPS within the pachychoroid spectrum.

Given the transient increase in peripapillary IRF in AU and the initial inflammatory presentation, methotrexate was introduced (as a steroid-sparing agent) to reduce the risk of posterior scleritis recurrence, while corticosteroids were de-escalated cautiously to mitigate pachychoroid activity. Over subsequent visits, peripapillary IRF/schisis regressed bilaterally. Final BCVA was 20/25 OD and 20/20 OS, with slight persistence of peripapillary IRF in OAU, without associated visual symptoms or further progression on follow-up imaging (Fig. 4A-B)

Discussion

Posterior scleritis is an uncommon inflammatory disorder of the sclera, accounting for fewer than 10% of all scleritis cases and frequently posing a diagnostic challenge due to its variable clinical manifestations[1,2]. Unlike anterior scleritis, which presents with characteristic external inflammatory signs, posterior scleritis tends to manifest through nonspecific symptoms such as deep orbital pain, decreased vision, or serous retinal changes[3,4]. Its clinical and imaging spectrum is broad and can mimic disorders such as Vogt–Koyanagi–Harada disease, sympathetic ophthalmia, infectious etiologies, and even metastatic disease[5,6]. Although exudative retinal detachment is a recognized manifestation, the occurrence of extensive choroidal detachments is exceedingly rare; few case reports describe posterior scleritis presenting with four-quadrant choroidal detachments, a presentation that may closely resemble malignant or infiltrative choroidopathies.

The pathophysiological basis for extensive choroidal detachments in this setting warrants consideration, particularly in the context of our patient's anatomical predisposition. In eyes with short axial length, inherent scleral thickening reduces transscleral fluid egress and may predispose to vortex vein compression, thereby impairing choroidal venous drainage at baseline. When posterior scleritis supervenes, active scleral inflammation further increases choroidal vascular permeability and exacerbates outflow obstruction through additional scleral thickening and inflammatory mediator release. This dual mechanism — augmented fluid production coupled with restricted lymphatic and venous drainage — disrupts the equilibrium of the suprachoroidal space, facilitating fluid accumulation and, ultimately, choroidal detachment. This structural predisposition may explain why four-quadrant choroidal detachments, though rare in posterior scleritis overall, may occur preferentially in hyperopic, short-axial-length eyes — the same anatomical phenotype that underlies pachychoroid spectrum disorders.

Although intraocular pressure was within normal limits bilaterally, we acknowledge that normal IOP does not entirely exclude anterior rotation of the ciliary body, as concurrent increases in uveoscleral outflow mediated by inflammatory prostaglandins may mask angle narrowing. Ultrasound biomicroscopy or anterior-segment OCT was not performed in this case, which represents a limitation; however, the absence of anterior chamber shallowing, the presence of posterior scleral enhancement on MRI, and the prompt response to systemic corticosteroids collectively supported a posterior inflammatory etiology rather than a primary angle-closure mechanism.

In the present case, the combination of periocular pain, exudative macular detachment with four-quadrant choroidal detachments, and posterior scleral enhancement on contrast-enhanced orbital MRI was strongly suggestive of posterior scleritis. B-scan ultrasonography demonstrated posterior scleral thickening consistent with scleritis; however, as adequate image documentation could not be obtained, contrast-enhanced orbital MRI was performed to confirm the diagnosis and characterize the extent of scleral involvement. Multimodal angiographic evaluation, including FA and ICGA, helped exclude more aggressive conditions sharing a similar presentation — FA demonstrating mottled hyperfluorescence without vasculitis, and ICGA revealing no choriocapillaritis or stromal granulomas, effectively reducing the likelihood of granulomatous choroiditis. Once infectious etiologies had been excluded, systemic corticosteroids were initiated.

The initial response to intravenous corticosteroids reflected effective suppression of the acute inflammatory component of posterior scleritis, with marked clinical and symptomatic improvement at one week prompting de-escalation of steroid therapy. However, the subsequent development of bilateral peripapillary intraretinal fluid (IRF) at three weeks — paradoxically during active corticosteroid therapy — requires mechanistic explanation. The worsening of pachychoroid spectrum disorders under corticosteroid exposure is not immediate and follows a temporal sequence driven by transcriptional reprogramming, hormonal feedback, and tissue remodeling. In the first week, suppression of the hypothalamic-pituitary-adrenal axis begins, with progressive shifts in the ratio of mineralocorticoid to glucocorticoid receptor activity within ocular tissues. By the second week, corticosteroid-induced transcriptional changes increase choroidal vascular permeability and impair retinal pigment epithelium barrier function. From the third week onward, progressive fluid accumulation exceeds the compensatory capacity of the outer blood-retina barrier, resulting in clinically manifest IRF — precisely as observed in our patient. Careful review of prior ancillary testing at this stage revealed the underlying predisposition: short, hyperopic eyes with preexisting choroidal folds and a chronic peripapillary pigment epithelial detachment, consistent with peripapillary pachychoroid syndrome (PPS) — a recognized entity within the pachychoroid spectrum particularly vulnerable to corticosteroid exposure[7,8], and sharing the same anatomical substrate already implicated in the choroidal detachments earlier in the clinical course.

A comprehensive differential diagnosis was considered at both stages. Neoplastic and infiltrative etiologies — including uveal metastasis, circumscribed choroidal hemangioma, intraocular lymphoma, and diffuse uveal melanocytic proliferation — were deemed unlikely given the absence of a focal mass on enhanced-depth OCT or MRI, the lack of leopard-spot pigmentation on widefield angiography, and the absence of vitritis or subretinal lesions. Drug-related uveal effusion secondary to recent sulfonamide exposure was also considered; however, normal IOP, markedly unilateral presentation, and the presence of periocular pain with posterior scleral enhancement argued against this diagnosis, as did the subsequent bilateral peripapillary manifestations. Vascular entities such as carotid-cavernous fistula were excluded based on normal IOP and the absence of corkscrew episcleral vessels or anterior segment congestion.

The positive interferon-γ release assay warranted consideration of tuberculous choroiditis; however, the clinical phenotype lacked choroidal granulomas, occlusive vasculitis, or necrotizing retinitis, and the angiographic profile was not consistent with this diagnosis. Management of latent tuberculosis was therefore deemed sufficient without antituberculous therapy.

Subfoveal choroidal thickness may serve as a useful activity biomarker in posterior scleritis and may help differentiate inflammatory exudation from pachychoroid-driven fluid — the latter being predominantly localized to the peripapillary region and characteristically exacerbated by corticosteroid exposure[11,12]. This distinction has direct therapeutic implications, as illustrated by the present case: corticosteroids, the mainstay of posterior scleritis treatment, may simultaneously exacerbate underlying pachychoroid disease. Methotrexate was therefore introduced as a steroid-sparing immunomodulator, with the dual aim of minimizing scleritis recurrence risk and enabling cautious corticosteroid de-escalation — a strategy that ultimately led to anatomical restitution and visual recovery.

Clinically, this case emphasizes that the appearance of peripapillary IRF during corticosteroid treatment should prompt reconsideration of the underlying mechanism, particularly in hyperopic eyes with pre-existing pachychoroid features. Recognizing this coexistence early is therapeutically critical: it shifts management from escalating immunosuppression toward a steroid-sparing strategy, avoiding iatrogenic worsening while preserving control of the inflammatory disease.

Declarations

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Declaration of Interest Statement

The authors declare no conflicts of interest related to this work.

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Ethics and Consent to Participate

Ethics approval was not required for this case report as it describes routine clinical care. Verbal informed consent was obtained from the patient for the publication of this case report. No personally identifiable information is included in this manuscript or its accompanying images.

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Figures

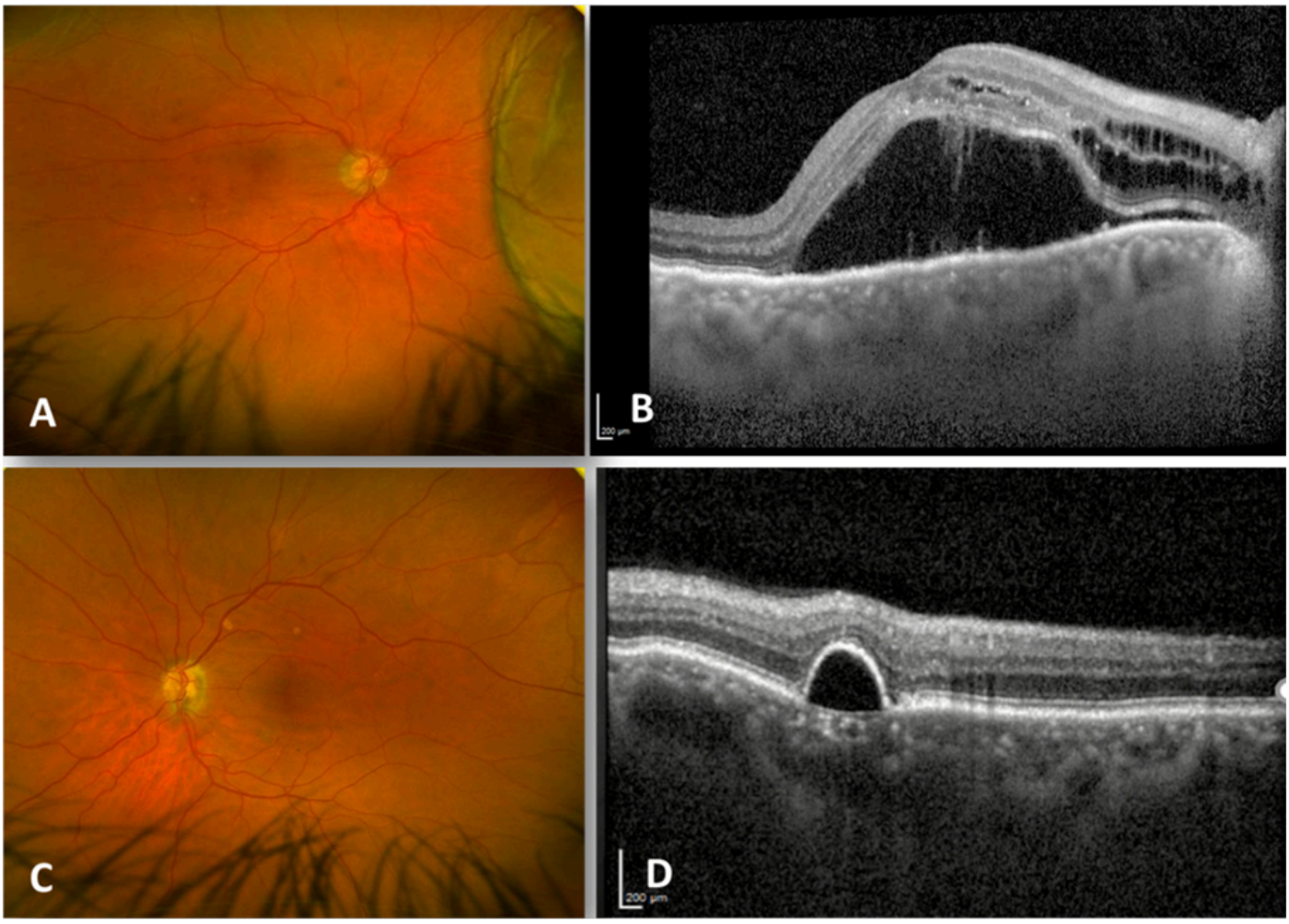


Figure 1

Baseline multimodal imaging at presentation. (A) Ultra-widefield color fundus photograph of the right eye (OD) showing optic disc edema with associated serous macular detachment and choroidal detachment (B) Spectral-domain OCT through the macula demonstrating a large serous retinal detachment with underlying choroidal thickening and associated intraretinal fluid. (C) Ultra-widefield color fundus photograph of the left eye (OS), asymptomatic at presentation, showing pre-existing choroidal folds in the peripapillary region. (D) OCT of OS revealing a chronic peripapillary pigment epithelial detachment without subretinal fluid, consistent with baseline pachychoroid-related changes.

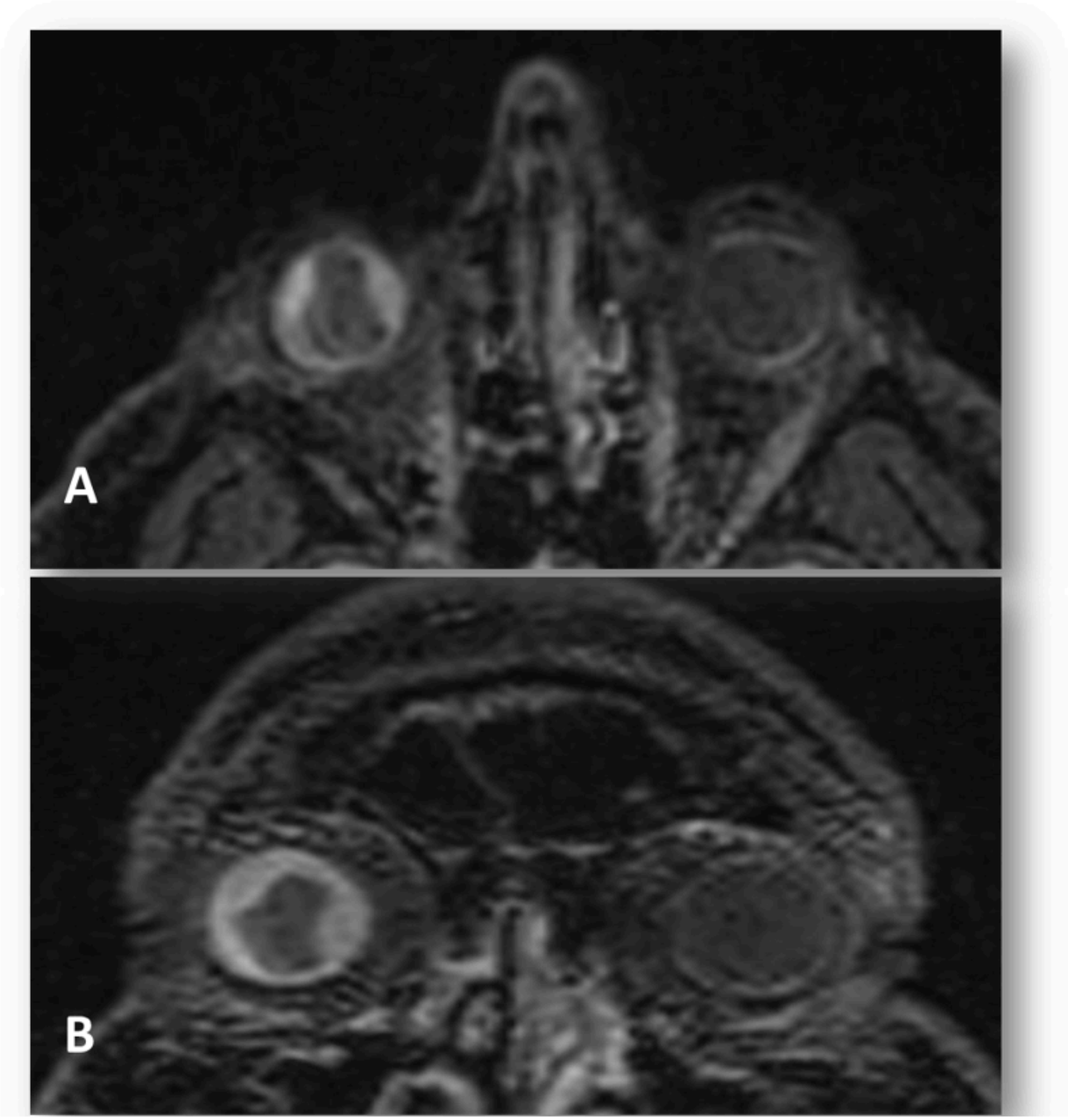


Figure 2

Orbital magnetic resonance imaging at presentation. (A) Axial contrast-enhanced T1-weighted MRI showing posterior scleral thickening and enhancement in the right eye (OD), consistent with active posterior scleritis. (B) Axial contrast-enhanced T1-weighted MRI at a slightly inferior level confirming posterior scleral enhancement with associated choroidal detachments, supporting an inflammatory scleral etiology.



Figure 3

Peripapillary OCT findings during follow-up. (A) Spectral-domain OCT through the peripapillary region of the right eye (OD) demonstrating intraretinal fluid with schitic changes along the papillomacular bundle following corticosteroid treatment. (B) Corresponding peripapillary OCT of the left eye (OS) showing similar intraretinal fluid/schisis, revealing bilateral involvement consistent with peripapillary pachychoroid syndrome.

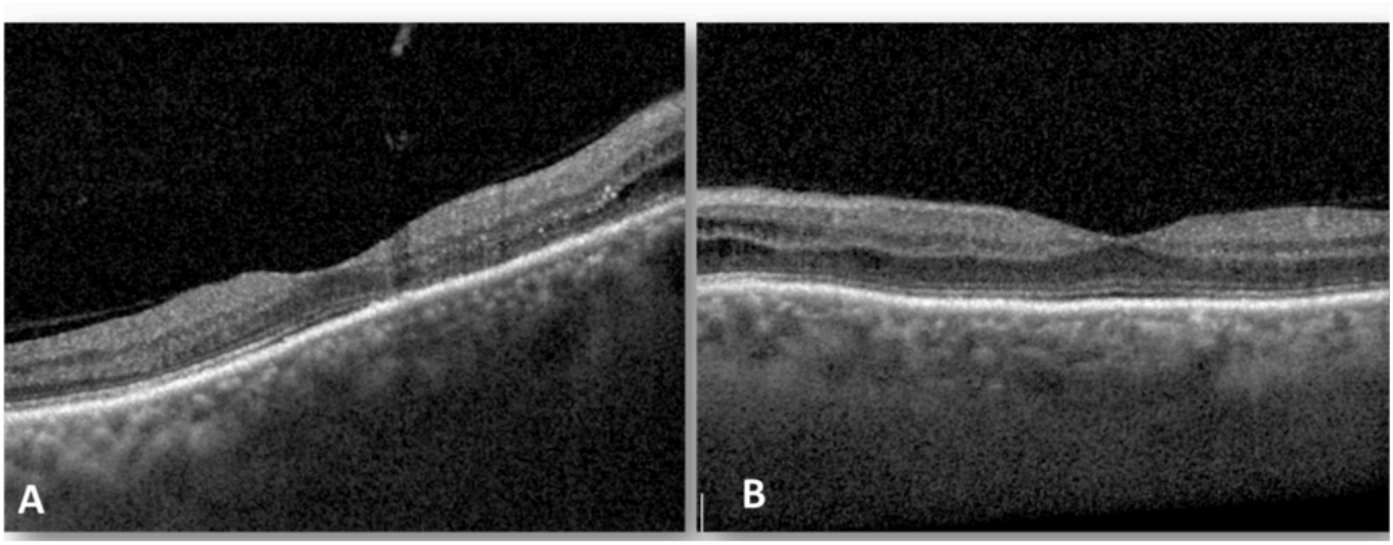


Figure 4

Resolution after corticosteroid de-escalation. (A) Peripapillary spectral-domain OCT of the right eye (OD) showing near-complete resolution of intraretinal fluid and schitic changes following gradual corticosteroid tapering. (B) Corresponding peripapillary OCT of the left eye (OS) demonstrating complete anatomical restitution, with restoration of normal retinal contour.