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Bilateral Idiopathic Posterior Scleritis: A Case Report

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Abstract

Background: A case of bilateral idiopathic posterior scleritis in a patient who developed headache, hypermetropic refractive shift, and bilateral choroidal folds, supported by multimodal ophthalmic imaging. A 52-year-old woman presented with a two-week history of headache and difficulty reading, and was found to have bilateral choroidal folds. She had initially been treated for sinusitis by her general practitioner with oral antibiotics, but did not improve and was referred to our ophthalmology outpatient department. Bilateral posterior scleritis was diagnosed based on B-scan ultrasonography, optical coherence tomography (OCT), and fluorescein angiography findings. An idiopathic aetiology was confirmed after a negative systemic workup. Oral prednisolone resulted in complete resolution of the choroidal folds and full reversal of the hypermetropic shift, with best-corrected visual acuity (BCVA) maintained at 20/20.
Conclusion: Bilateral posterior scleritis is uncommon and easily misdiagnosed. A combination of B-scan ultrasonography, OCT, and fluorescein angiography confirmed the diagnosis in this case, and oral prednisolone produced a complete response.

Keywords

Posterior Scleritis, Choroidal Folds, Multimodal Imaging, Corticosteroids, Hypermetropic Shift

1. Introduction

Posterior scleritis is an uncommon and frequently unrecognised inflammatory disorder of the posterior segment, and its presentation can be highly variable. Patients may have ocular pain, headache, visual disturbance, and in some cases, present with anterior segment involvement; one series reported anterior uveitis in around 55% of cases [1] [2]. Posterior segment signs include optic disc swelling,

choroidal folds, serous retinal detachment, and occasionally subretinal exudates or subretinal masses [1] [2].

The condition is most commonly seen in women between the third and sixth decades of life, and accounts for approximately 2% - 12% of all cases of scleritis [3] [4]. The aetiology may be idiopathic, infectious, traumatic, or associated with systemic autoimmune disease, which is identified in approximately 10% - 30% of cases in different series [3] [5].

The bilateral form is less common than the unilateral form, with a reported incidence of around 3% - 17%, and it is particularly challenging to diagnose [5]. It may mimic a choroidal tumor, and it must be distinguished from other, more common inflammatory disorders of the posterior segment [5] [6].

The diagnosis depends on multimodal imaging. B-scan ultrasonography typically shows posterior scleral thickening and fluid in Tenon's space, producing the well-known "T-sign", sometimes with distension of the optic nerve sheath. Optical coherence tomography (OCT) is useful for measuring scleral and choroidal thicknesses and for monitoring response, and MRI may be required when retrobulbar pathology needs to be excluded [3] [6].

Because of the risk of visual loss, management may involve both ophthalmology and rheumatology, particularly when a systemic association is suspected [6]. Most patients respond to systemic corticosteroids, either oral prednisolone or intravenous methylprednisolone, and in special situations, topical corticosteroids and periocular triamcinolone injections may be used in selected cases [7].

2. Case Presentation

A 52-year-old woman presented to our ophthalmology outpatient department with a two-week history of severe left-sided headache, ocular pain, and progressive visual disturbance, especially regarding near vision, which was a new issue following her complaint.

Her general practitioner treated her empirically for sinusitis with oral antibiotics, but her symptoms did not improve, and she was referred for ophthalmological assessment.

In addition to the headache, she reported difficulty in reading, photophobia, and intermittent blurring. She denied experiencing flashes, floaters, or diplopia. Her medical and ocular history was unremarkable.

3. Clinical Examination

The best-corrected visual acuity (BCVA) was 20/20 in each eye, but with a hypermetropic shift of approximately +1.50 D bilaterally on autorefraction. Her current refraction was $+0.50/-0.50 \times 90^{\circ}$ in the right eye (OD) and $+1.00/-0.75 \times 165^{\circ}$ in the left eye (OS), compared with a baseline of $-1.00/-0.50 \times 90^{\circ}$ (OD) and $-0.50/-0.75 \times 165^{\circ}$ (OS), obtained from her previous spectacle prescription, dated approximately six months before this presentation. Both refractions were obtained by subjective refraction and autorefraction, allowing a direct comparison.

Intraocular pressure measured using Goldmann applanation tonometry was 12 mmHg in the right eye and 10 mmHg in the left eye. The conjunctiva and episclera were normal in both eyes, with no chemosis or tenderness on palpation. Ocular motility was unremarkable, and no proptosis was observed. The corneae were clear, and there was no anterior segment inflammation. The pupils were round and reactive to light, with no relative afferent pupillary defect (RAPD). Only mild age-related changes were observed in the lenses.

On dilated fundus examination, the optic discs were bilaterally well perfused, with a cup-to-disc ratio of 0.1. A choroidal nevus was noted superonasal to the left optic disc, and dull foveal reflexes in both maculae were associated with clearly visible choroidal folds. There were no signs of retinal hemorrhages, exudates, retinal vasculitis, or vitritis.

4. Investigations

Multimodal imaging was performed to characterize the posterior segment findings. Macular OCT (DRI OCT Triton Plus, Topcon) demonstrated mildly increased choroidal thickness with undulation of the retinal pigment epithelium, consistent with the choroidal folds in both eyes (**Figure 1**). No significant subretinal fluid, intraretinal fluid, or cystoid macular edema was observed, and the inner retinal layers were intact.

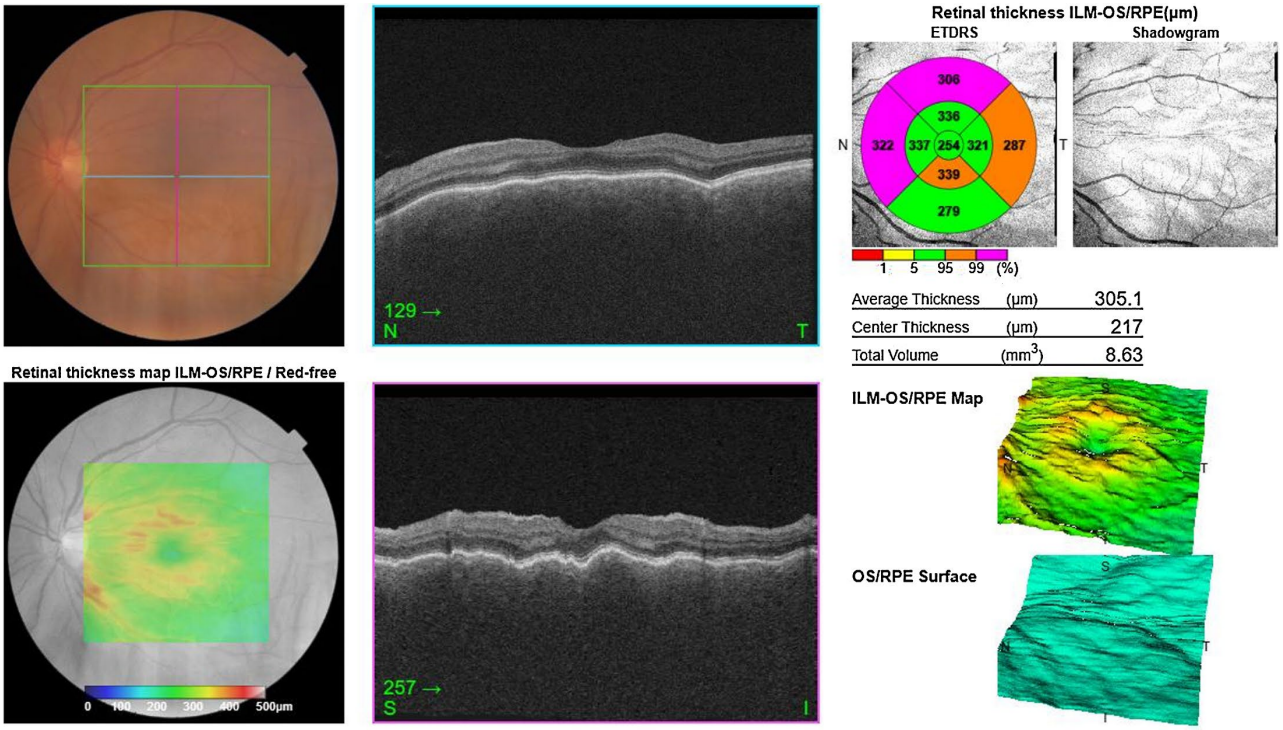
Wide-field color fundus photography (ZEISS CLARUS 700) confirmed the bilateral choroidal folds in the macular area (**Figure 2**). Fluorescein angiography showed bilateral choroidal folds and late optic disc hyperfluorescence ("hot disc") in both eyes, without obvious vascular leakage elsewhere at the posterior pole (**Figure 3**). Automated perimetry (Oculus Twinfield, program 30-2) demonstrated bilateral enlargement of the blind spot with mild reduction in paracentral sensitivity; no dense scotoma involving fixation was identified, consistent with the preserved BCVA of 20/20 in both eyes throughout the case (**Figure 4**).

B-scan ultrasonography demonstrated diffuse posterior scleral thickening with a positive T-sign, representing fluid in Tenon's space, in both eyes (**Figure 5**).

A comprehensive systemic workup was performed to identify infectious and autoimmune causes. The QuantiFERON-TB Gold test result was negative, effectively excluding tuberculous scleritis. Borrelia (Lyme) serology showed negative IgG and a borderline weak-positive IgM by ELISA, which was not confirmed by immunoblotting, making active borreliosis unlikely. Serology for HIV-1 and HIV-2 was negative. Varicella-zoster virus, Epstein-Barr virus, and cytomegalovirus IgG were positive, consistent with past infection only. Serum angiotensin-converting enzyme (ACE), cytoplasmic anti-neutrophil cytoplasmic antibodies (c-ANCA), and rheumatoid factor levels were within normal limits. The complete blood count, liver function tests, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) levels were unremarkable.

Chest radiography revealed a small (5 mm) pulmonary granuloma at the left apex, with no pulmonary infiltrates or evidence of active infection. Cranial CT

excluded retrobulbar pathology and showed no signs of sinusitis.



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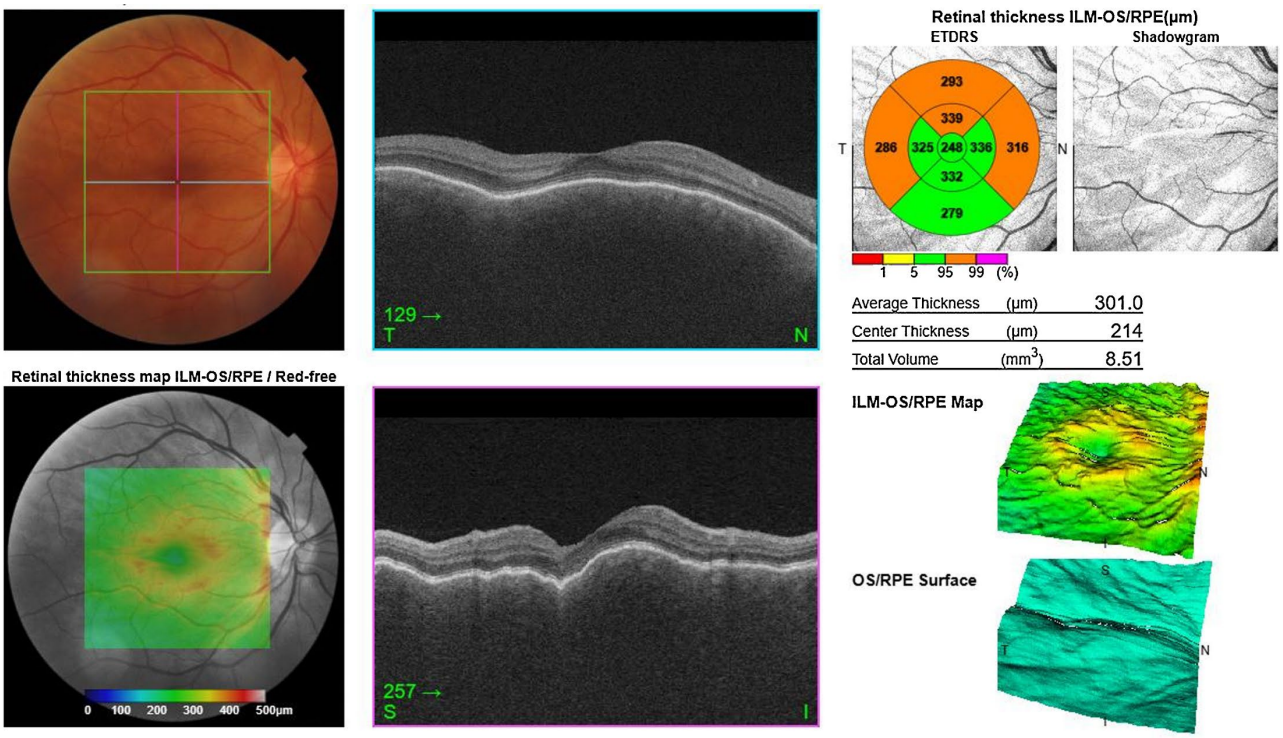

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Figure 1. OCT shows choroidal thickening with folds. No significant subretinal fluid or cystoid macular edema (CME), inner retinal layers were intact.

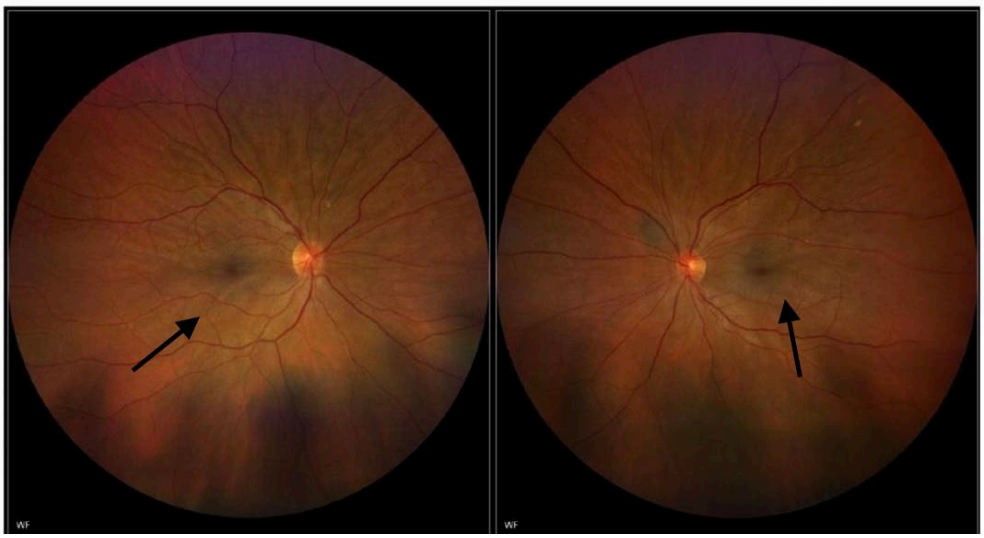


Figure 2. Wide-field colour fundus photography confirmed bilateral choroidal folds.

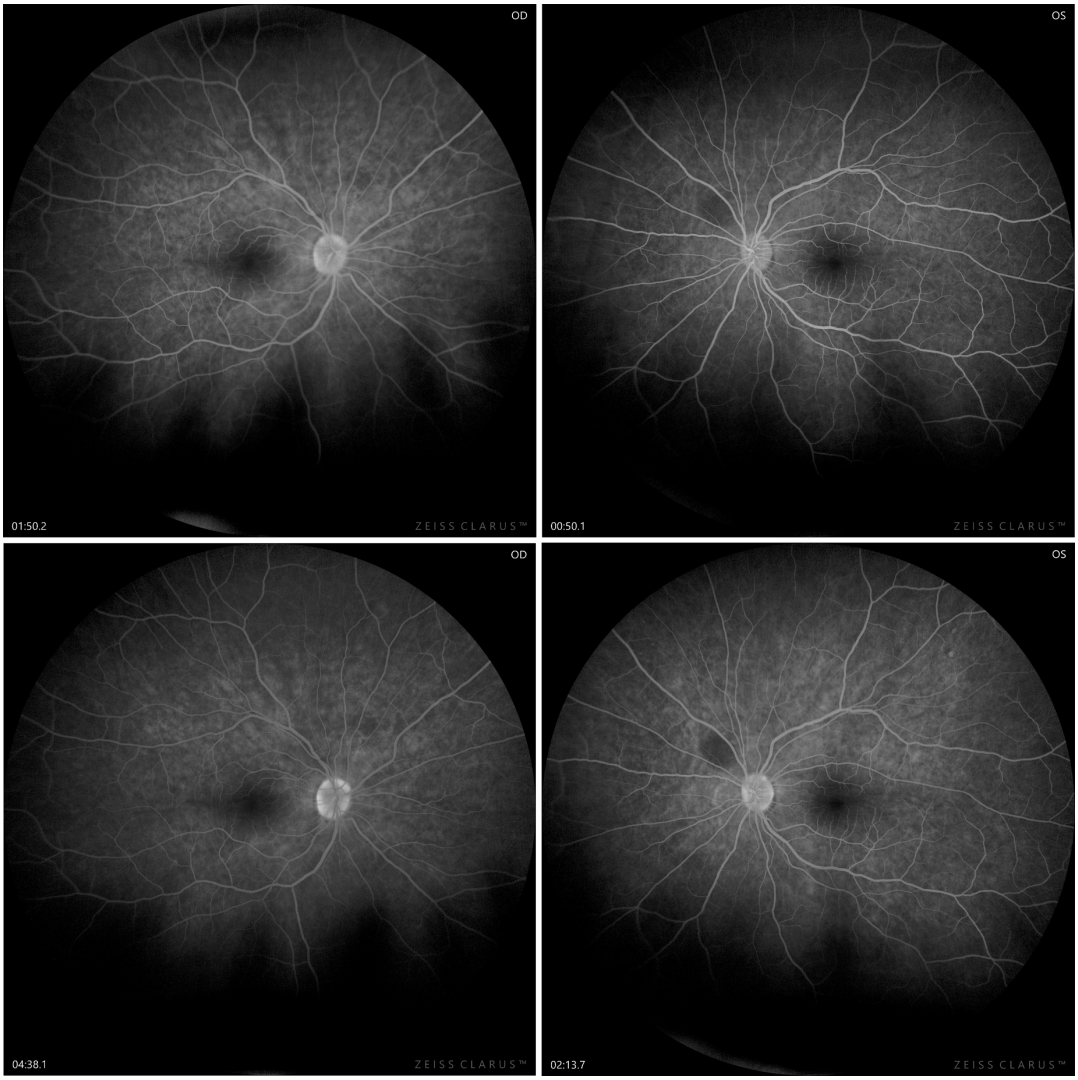
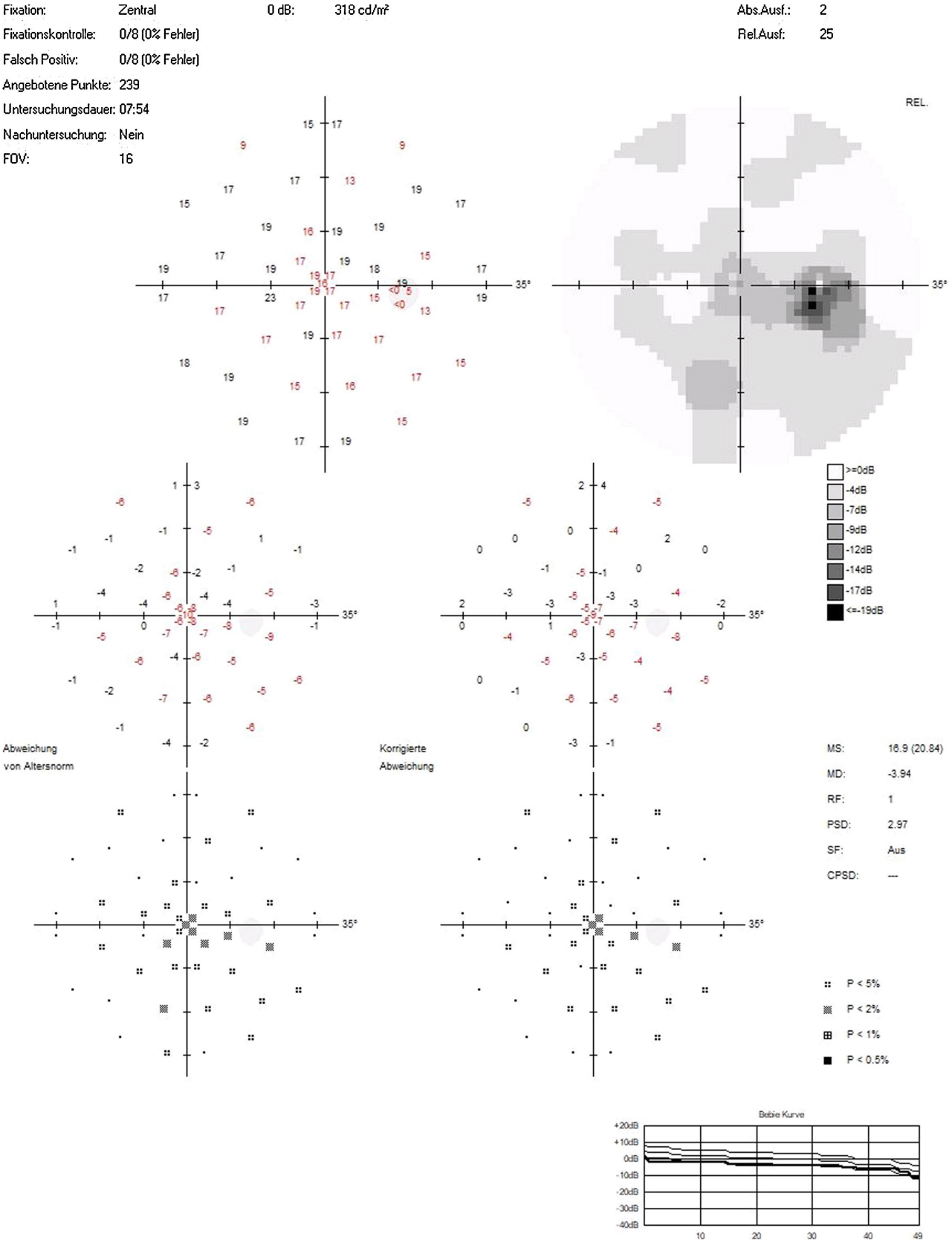


Figure 3. Fluorescein angiography, bilateral choroidal folds and late optic disc hyperfluorescence ("hot disc").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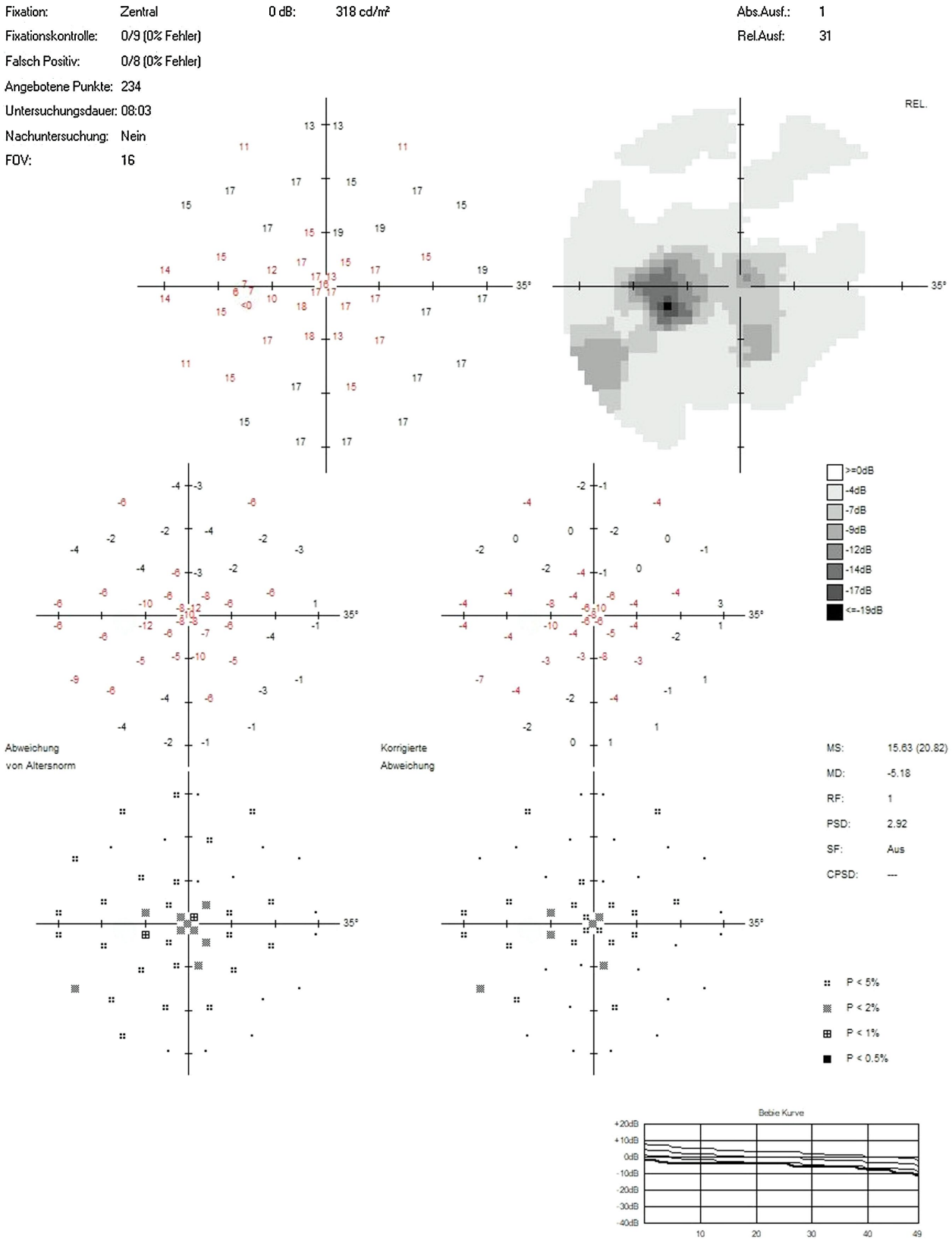

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Figure 4. Automated perimetry (Oculus Twinfield, programme 30-2), bilateral enlargement of the blind spot.

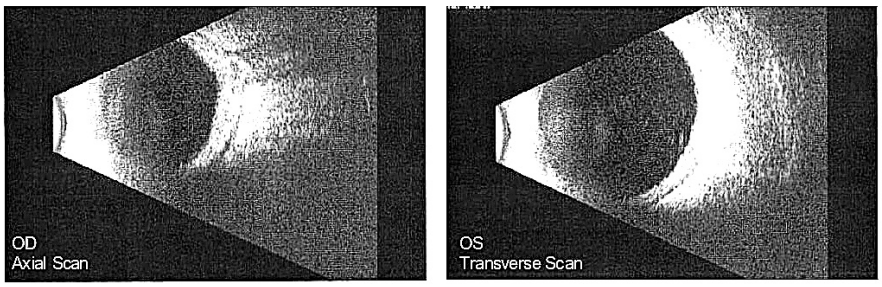


Figure 5. B-scan, posterior scleral thickening with a positive T-sign.

5. Diagnosis and Differential Diagnosis

Based on the clinical features and imaging findings, bilateral choroidal folds, posterior scleral thickening with a positive T-sign, late optic disc hyperfluorescence, and a hypermetropic refractive shift revealed a diagnosis of bilateral posterior scleritis.

Although chest radiography demonstrated a small 5-mm apical pulmonary granuloma, this was considered an incidental finding because there was no clinical, laboratory, or microbiological evidence of active granulomatous, infectious, or systemic autoimmune disease. The negative QuantiFERON-TB test, normal serum ACE and inflammatory markers, negative autoimmune serology, and absence of systemic features supported classification of the case as idiopathic.

The principal differential diagnoses included posterior uveitis, choroidal tumour (in particular amelanotic choroidal melanoma or metastasis), idiopathic orbital inflammation, sarcoidosis-associated scleritis, tuberculous choroiditis, and Vogt-Koyanagi-Harada disease. The absence of intraocular inflammation, symmetrical bilateral findings, negative serology, and a characteristic B-scan appearance argued against these alternatives. Cluster headache was considered briefly because of the retro-orbital pain, but the bilateral ocular signs, peripapillary field changes, and imaging findings made this diagnosis unlikely [8].

6. Management

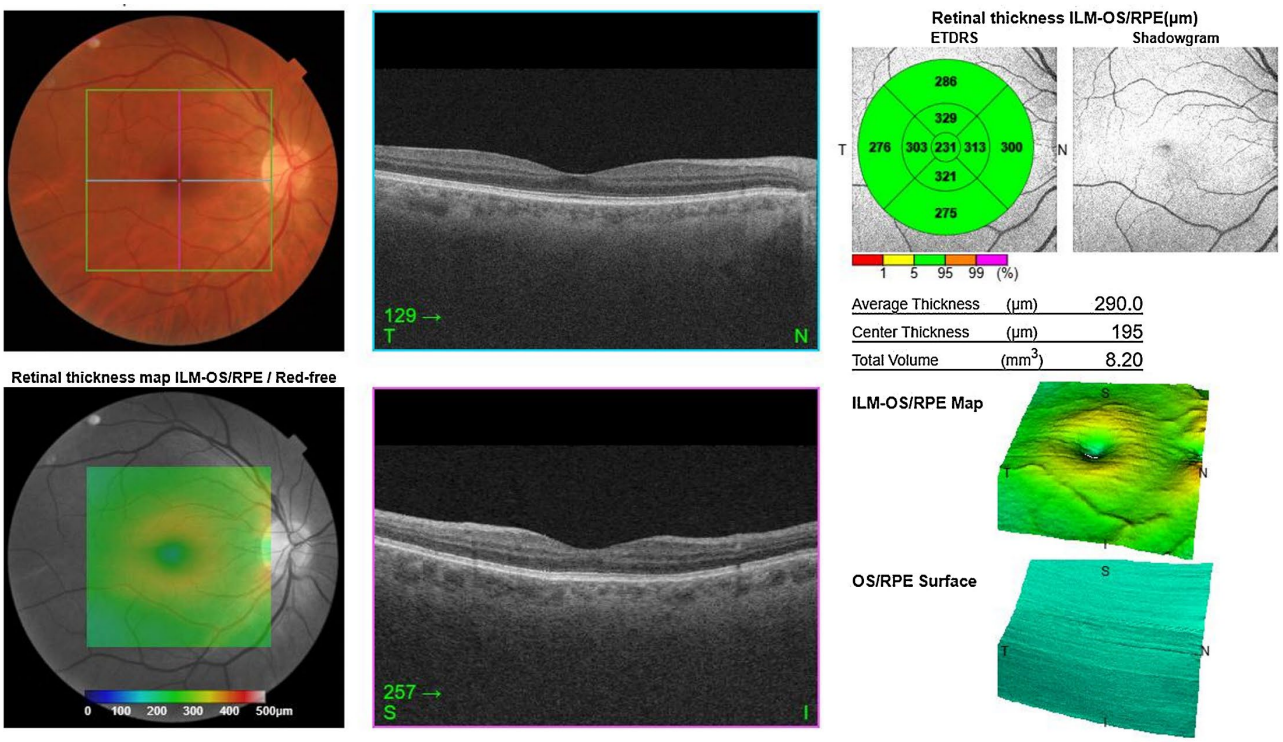
After systemic workup, oral prednisolone was started at 1 mg/kg/day (approximately 80 mg daily for the patient's body weight). Blood pressure and capillary blood glucose levels were monitored regularly. Pantoprazole 40 mg once daily was prescribed for gastric protection, and because the patient had received only a partial course of antibiotics from her general practitioner for presumed sinusitis, oral cefuroxime 250 mg twice daily was continued for one further week as a precautionary measure while high-dose systemic corticosteroids were commenced, pending final confirmation of the negative infectious and imaging workup. It was stopped once cranial CT had excluded active sinusitis and the infectious screen was confirmed negative.

At the first follow-up one week later, the patient reported partial improvement in ocular pain and vision. Upon further questioning, it became clear that she had been taking only 20 mg of prednisolone daily, rather than the prescribed 80 mg,

because of a misunderstanding of the dosing instructions. The plan was clarified, and the patient was reviewed again one week later. At that visit, the patient had improved substantially, and the choroidal folds had resolved and her refraction had returned to baseline. A tapering schedule was started, and the prednisolone dose was reduced by 10 mg per week. Her primary care physician was informed of the diagnosis, and a rheumatology referral and follow-up were nevertheless arranged as a precaution to monitor for any future evidence of an underlying systemic disorder, and for longer-term assessment; however, the outcome was not available at the time of manuscript preparation.

7. Outcome and Follow-Up

At the four-week follow-up, the patient was asymptomatic. The BCVA was 20/20 in both eyes. Fundus examination and macular OCT confirmed the complete resolution of the choroidal folds (Figure 6). Corticosteroid tapering was continued without incident. Follow-up was arranged at three-monthly intervals for one year, and BCVA, refraction, and macular OCT were monitored at each visit. The complete clinical course, from symptom onset to long-term follow-up, is summarized, as shown in Table 1.



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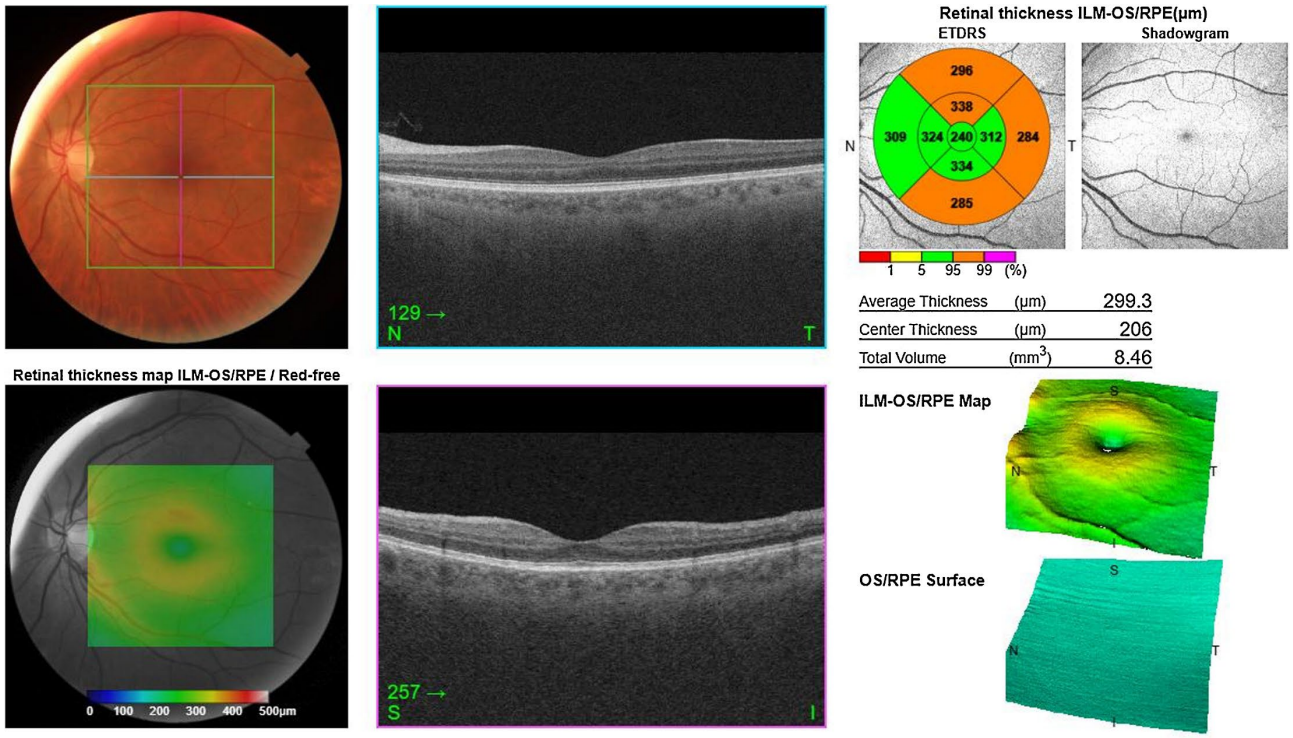

(b)

Figure 6. Macular OCT, complete resolution of the choroidal folds.

Table 1. Clinical timeline.

Stage	Timing (as stated in manuscript)	Detail
Onset of Symptom	At Day 0	Onset of left-sided headache, ocular pain, and progressive near-vision difficulty
GP assessment and treatment	within 2-weeks	Empirical treatment for presumed sinusitis with oral antibiotics; no improvement
Referral to ophthalmology	~2 weeks after symptoms onset	Referred after failure to improve with GP treatment
Ophthalmological Assessment & Multimodal Imaging	~2 weeks after symptoms onset	BCVA 20/20 OU; hypermetropic shift +1.50 D bilaterally; bilateral choroidal folds on examination, OCT, wide-field photography, FA, B-scan, perimetry
Systemic workup	Within one week after the ophthalmological assessment	QuantiFERON-TB, Borrelia serology, HIV, ACE, c-ANCA, RF, CBC/LFT/ESR/CRP, CXR, cranial CT
Steroid initiation	After the systemic workup	Oral prednisolone 1 mg/kg/day (~80 mg/day) started; pantoprazole and cefuroxime co-prescribed
First follow-up	1 week after starting prednisolone	Partial improvement in pain/vision reported; dosing error discovered—patient had taken only 20 mg/day instead of 80 mg/day dosing clarified and corrected
Second follow-up	1 week after the dose correction (≈2 weeks after starting prednisolone)	Substantial improvement; choroidal folds resolved; refraction returned to baseline; tapering (−10 mg/week) begun

Continued

Rheumatology referral/GP notified	At the second follow-up	Primary care physician informed; rheumatology referral made—outcome not available at the time of manuscript preparation
Four-week follow-up	4 weeks after starting prednisolone.	Asymptomatic; BCVA 20/20 OU; complete resolution of choroidal folds confirmed on fundus exam and OCT
Long-term follow-up	Ongoing, 3-monthly for 1 year	Monitored BCVA, refraction, and macular OCT at each visit

8. Discussion

Two aspects of this case are worth discussing in detail. First, the initial misdiagnosis of sinusitis was not unreasonable based on symptoms alone, such as headache, ocular pain, and visual blurring, with no obvious external signs of ocular inflammation; however, it cost the patient two weeks. Posterior scleritis is known to present with non-specific symptoms and is misdiagnosed as a result [5] [6], and a low threshold for ophthalmological referral is justified when ocular pain fails to settle with treatment of the suspected cause.

Second, a hypermetropic shift of approximately +1.50 D bilaterally was, in retrospect, the most useful early sign. This is explained by the anterior displacement of the retina secondary to posterior scleral and choroidal thickening, which shortens the effective axial length. It also reversed completely once the choroidal folds resolved upon treatment, which made it a useful objective marker of disease activity in this patient. Refractive change of this kind is easy to overlook in routine clinical assessment but is worth asking about specifically in any patient with new-onset reading difficulty and ocular pain.

Posterior scleritis is often associated with systemic autoimmune diseases, including rheumatoid arthritis, granulomatosis with polyangiitis (Wegener's disease), systemic lupus erythematosus, and relapsing polychondritis [9], although a substantial proportion of cases remain idiopathic after thorough investigation [10]. Reported series suggest that systemic disease is identified in approximately 17% - 37% of patients [5], and bilateral disease in particular is often idiopathic, although associations with tuberculosis and giant cell arteritis have also been described [2] [3]. In this patient, a negative QuantiFERON-TB test, normal inflammatory markers, and the absence of clinical or serological evidence of autoimmune disease supported an idiopathic diagnosis. Small apical pulmonary granuloma on chest radiography didn't indicate active disease in the absence of supporting findings; however, it is a reasonable trigger for continued surveillance.

The diagnosis was confirmed by combining several imaging modalities rather than by any single test. B-scan ultrasonography is often regarded as the most useful single investigation, but the T-sign is not pathognomonic; González-López *et al.* reported signs in approximately 11% of bilateral cases, which limits its sen-

sitivity [11]. OCT contributed to quantifying choroidal thickening and excluding subretinal fluid and cystoid macular edema, while fluorescein angiography demonstrated the characteristic late optic disc hyperfluorescence. MRI was not performed in this case because the ultrasound and OCT findings, together with the negative cranial CT, were considered sufficient; however, MRI would have added orbital soft-tissue detail and would be appropriate in less clear-cut cases [6].

The therapeutic response of this patient was rapid and complete. Oral prednisolone alone has been reported to be effective in only approximately 22% of patients with bilateral posterior scleritis, and intravenous methylprednisolone is often required to control inflammation and prevent relapse [11]. The favorable response here is probably explained by the early initiation of treatment, the absence of underlying systemic disease, and relatively mild structural changes on OCT (no subretinal fluid).

The partial improvement reported during the first week, while the patient was inadvertently taking only 25% of the intended dose, cannot be reliably attributed to a specific pharmacological effect of the lower dose; it may equally reflect early, non-specific symptom fluctuation over a short observation period. The substantially greater and more complete improvement seen only after the intended dose of 1 mg/kg/day was reached is consistent with the established recommendation for adequate initial corticosteroid dosing in posterior scleritis, and this case should not be taken as evidence that reduced doses are sufficient for disease control.

This study had several limitations. In a single-case study, the findings cannot be generalized.

Long-term follow-up beyond the planned 12 months is necessary to confirm sustained remission and detect any late-emerging systemic association. MRI was not performed; although the diagnosis was secure based on the available evidence, future similar cases would benefit from this additional modality. Finally, the label "idiopathic" reflects the limits of currently available investigations rather than a definitive etiological conclusion.

9. Conclusion

The hypermetropic refractive shift in this patient was approximately +1.50 D bilaterally; it was the most useful early sign, and it reversed completely once the choroidal folds were resolved on treatment. Combining B-scan ultrasonography, OCT, and fluorescein angiography made the diagnosis secure; no single imaging modality would have been sufficient. The systemic workup was negative, and the patient responded fully to oral prednisolone, although the small apical pulmonary granuloma on chest radiography was a reasonable trigger for continued surveillance. Posterior scleritis is worth keeping in mind for any patient presenting with ocular pain and headache that does not settle with treatment of more common diagnoses.

Funding Statement

No funding was received for this study.

Data Access Statement

No data are available for sharing. Data sharing is not applicable to this study.

Author Contributions

Abdelhamid Ghunaim, Mohammed Allawi, and Ali Zimmermann contributed to the conception, literature review, drafting, and final approval of the manuscript.

Ethics Approval

Ethical approval was not required for this single-patient case report, in accordance with local institutional policy, as no experimental intervention was performed and all data were collected during routine clinical care.

Consent for Publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying clinical images.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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