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CASE REPORT

# Posterior nodular scleritis associated with presumed ocular tuberculosis: two case reports

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## ABSTRACT

**Introduction:** Posterior nodular scleritis is an uncommon form of scleral inflammation that represents a diagnostic challenge, as it mimics malignant lesions, potentially leading to misdiagnosis and inadequate treatment.

**Methodology**: Two case reports of posterior nodular scleritis, focusing on its clinical characteristics and multimodal evaluation.

**Results:** Case 1 describes a 40-year-old woman with visual scotoma, headache, and periocular pain in the left eye for 2 weeks, presenting with whitish subretinal lesions and macular elevation on the fundoscopic exam. Ultrasound revealed temporal parietal nodular thickening with subretinal and subtenon fluid compatible with posterior nodular scleritis. Etiological investigation revealed a positive tuberculin skin test with a normal chest CT scan, and specific antituberculosis treatment was instituted in conjunction with systemic corticosteroid therapy. The patient progressed with complete regression of the lesions and preservation of vision. Case report 2 describes a 56-year-old diabetic woman with progressive visual loss and pain in her left eye for 2 months. Anterior biomicroscopy revealed conjunctival hyperemia, granulomatous keratic precipitates, and cataract, while fundoscopy showed proliferative diabetic retinopathy. Ultrasound revealed superior nasal nodular thickening and diffuse subtenon fluid. Etiological investigation showed a positive tuberculin skin test with a normal chest CT scan, and she received antituberculosis and anti-inflammatory therapy. Despite therapy, she developed macular atrophy with permanent visual impairment.

**Discussion:** Both cases represent posterior nodular scleritis associated with presumed ocular tuberculosis. The presumptive diagnosis of ocular tuberculosis was based on a triad of compatible clinical-radiological findings, a positive tuberculin skin test, and a favorable response to treatment. Multimodal evaluation including B-scan ultrasound was essential for differential diagnosis with intraocular tumors. Combined treatment proved effectiveness in controlling inflammation in both cases.

**Conclusion:** Recognizing ocular tuberculosis as the etiology of posterior nodular scleritis is essential in endemic settings, particularly where there is a positive tuberculin skin test and compatible clinical and radiological findings. Multimodal evaluation including ocular ultrasound allows for accurate diagnosis and differentiation from tumorous lesions. Visual prognosis remains variable depending on early diagnosis and severity of initial inflammation, reinforcing the importance of timely detection and treatment.

## Introduction

Scleritis is characterized by inflammation of the sclera, and it may also affect adjacent ocular and periocular tissues.[1] Anatomically, scleritis is classified as anterior when it affects the portion of the sclera located anterior to ora serrata; and posterior, when it involves the region posterior to it.[2,3] Posterior scleritis, that correspond to approximately 10% of all scleritis cases, is subdivided into diffuse and nodular, differentiated by the extent of scleral inflammatory involvement: in diffuse forms, global inflammation of the ocular wall is observed, while in nodular forms the inflammation is restricted to a part of the sclera, resulting in the formation of scleral nodules.[4,5] Posterior nodular scleritis presents as a significant diagnostic challenge because it mimics malignant lesions, which can lead to misdiagnosis, inadequate treatment and permanent vision lost.[4] This anatomical differentiation is important since the etiologies, clinical manifestations, treatment approaches and visual outcomes are different among these entities.[3] The diagnosis of scleritis and its etiology is based on the correlation between clinical, laboratory, and imaging findings, with ocular ultrasonography being one of the most frequently used modalities, particularly in cases with posterior involvement.[2] Etiologically, scleritis is broadly categorized into non-infectious and infectious causes, the former accounting for the majority of cases. Although uncommon, infectious scleritis remains clinically relevant due to its distinct therapeutic implications.[6] Among infectious etiologies, ocular tuberculosis represents a rare but important cause, particularly in tuberculosis-endemic regions. It may manifest as anterior or posterior scleritis, in both diffuse and nodular forms.[7,8] However, microbiological confirmation is often not feasible in cases of isolated ocular involvement, and the diagnosis frequently relies on a presumptive approach based on compatible clinical findings, epidemiological context, and therapeutic response. Despite isolated reports in the literature, posterior nodular scleritis associated with presumed ocular tuberculosis remains sparsely documented.[8] Here, we present two cases of posterior nodular scleritis associated with presumable ocular tuberculosis, analyzing their clinical characteristics and multimodal assessment, as well as comparing them with data published in the literature.

## Case Report

CASE 1

A 40-year-old female patient, hypertensive, with no relevant past ophthalmological history, presented to the emergency service complaining of a visual scotoma in her left eye, associated with ipsilateral headache and periocular pain, which worsened with eye movement, symptoms reportedly started 2 weeks before the first evaluation. The patient denied other symptoms, as well as recent travel, contact with sick people, or exposure to animals.

Initial ophthalmological examination revealed visual acuity of 20/20 bilaterally, with an unaltered anterior segment. Fundoscopy of the left eye revealed whitish subretinal lesions in the temporal, nasal, and inferior perifoveal regions, as well as macular elevation (Figure 1.A). The right eye showed no alterations. Optical coherence tomography demonstrated hyperreflectivity and irregularity of the outer retina in the areas corresponding to the lesions visible in the retinography, as well as elevations in the choroid (Figure 1.B).

Ocular ultrasound (10 MHz transducer, transpalpebral) demonstrated mild vitreous opacities and the presence of nodular parietal thickening in the temporal region, with moderate internal reflectivity, associated with subtenon and subretinal fluid, consistent with posterior nodular scleritis. Adjacent macular thickening was also observed, probably secondary to local inflammatory involvement .

Etiological investigation revealed a tuberculin skin test of 21 mm (reactive), chest tomography without significant alterations, and absence of other possible infectious or rheumatological causes. Given the clinical picture associated with a reactive tuberculin skin test and considering the brazilian epidemiology, the hypothesis of posterior nodular scleritis secondary to presumed ocular tuberculosis was raised, and specific

treatment was instituted consisting of rifampicin, isoniazid, pyrazinamide, and ethambutol for 2 months, followed by rifampicin and isoniazid for 7 months (total of 9 months), associated with oral corticosteroid therapy in a tapering scheme during 4 months.

After treatment, the patient maintained visual acuity in the left eye of 20/20, with regression of the subretinal lesions and resolution of macular elevation, presenting only some areas of pigmentary alteration (Figure 2A). Optical coherence tomography demonstrated a regression in the previously visualized choroidal thickening, with a small residual area of outer retinal interruption (Figure 2B).

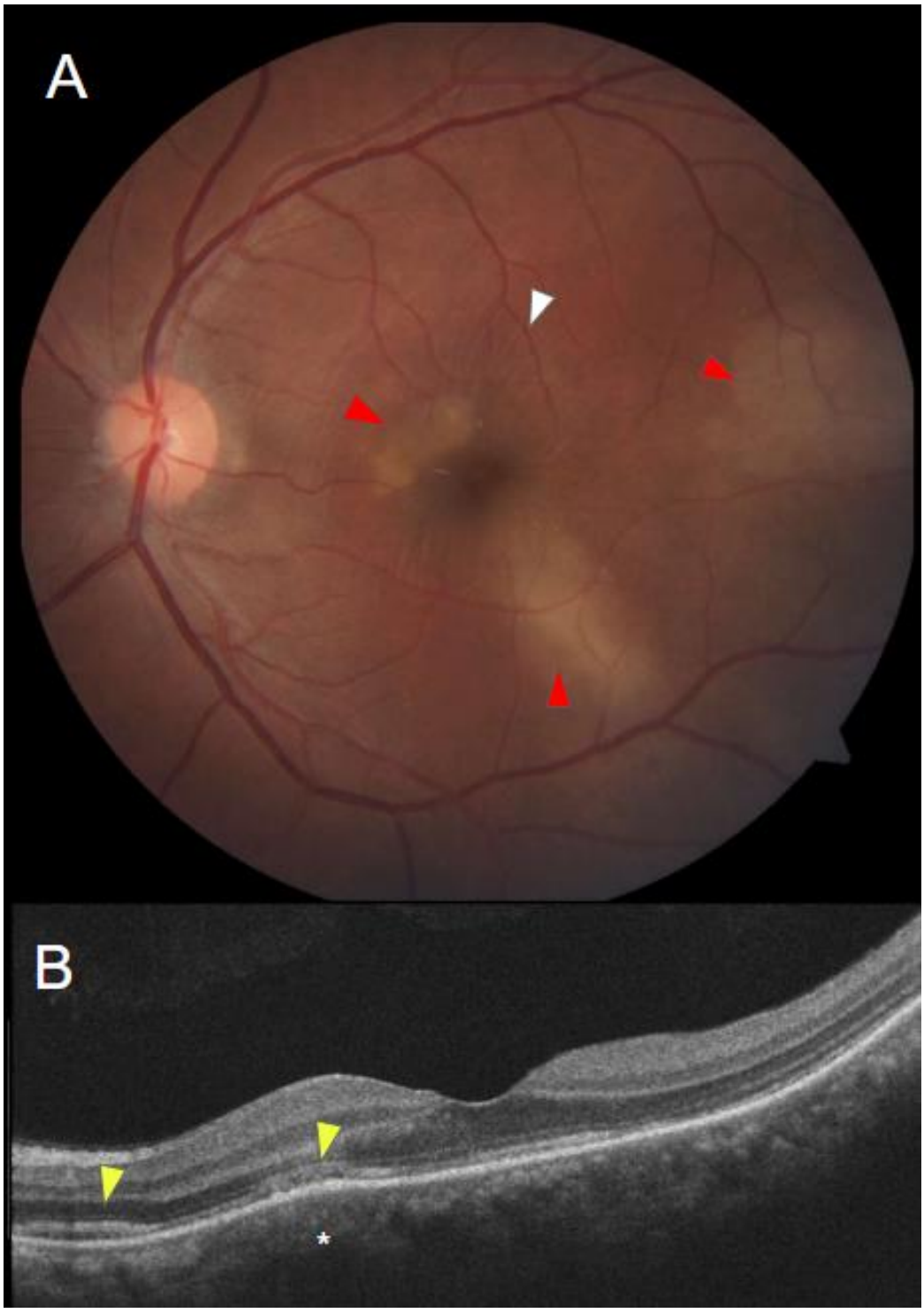


**Figure 1.** Pre-treatment evaluation of the left eye. (A) Retinography showing diffuse whitish subretinal lesions (red arrowheads) and macular elevation (white arrowhead). (B) Horizontal OCT scan demonstrating choroidal elevation (white asterisk) and outer retinal hyperreflectivity and irregularity (yellow arrowheads).

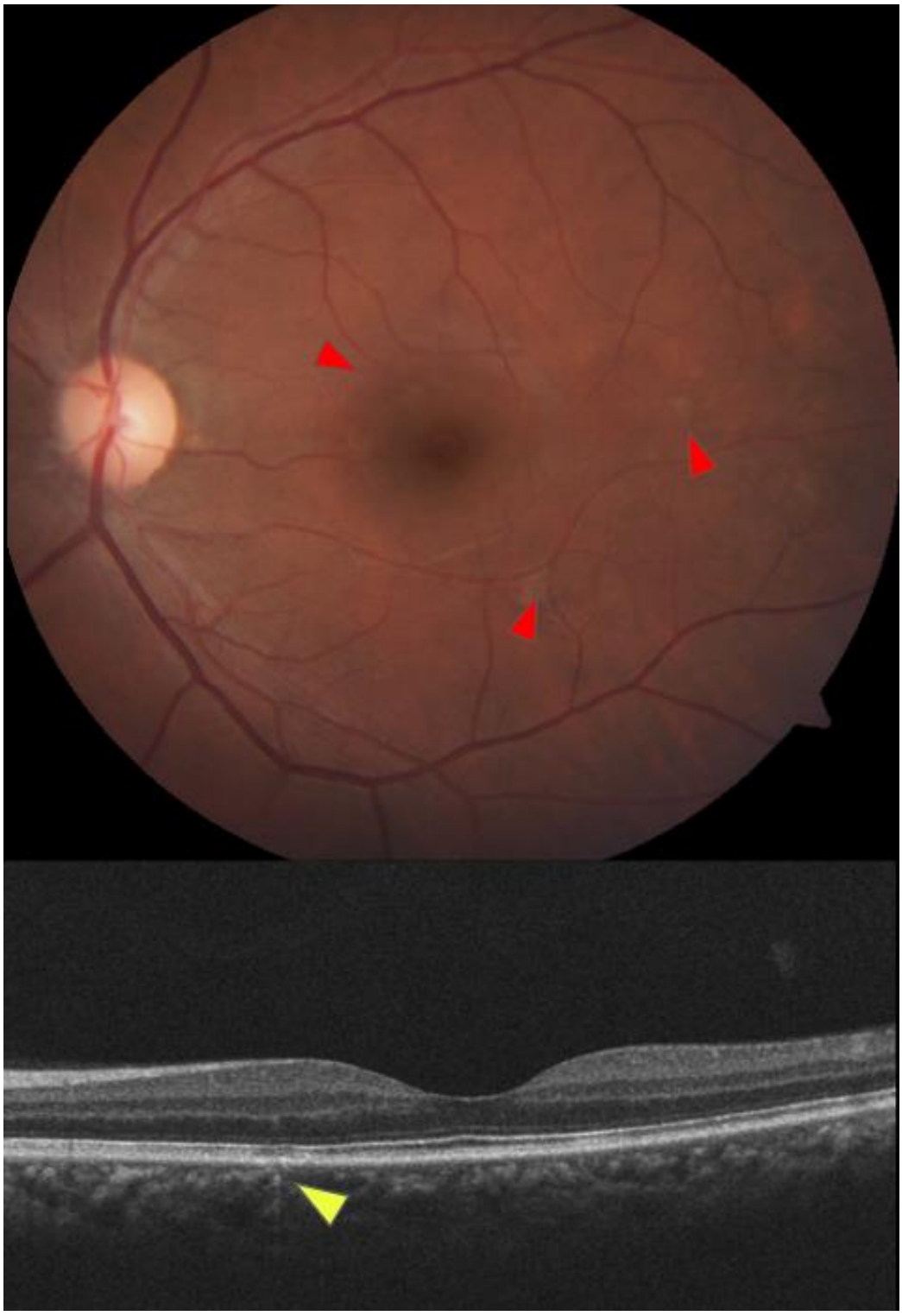

**Figure 2.** Post-treatment left eye. (A) Retinography showing regression of subretinal lesions and resolution of macular elevation, with residual pigmentary changes (red arrowheads). (B) Horizontal OCT demonstrating reduced choroidal thickening and a small residual outer retinal disruption (yellow arrowhead).

B-scan ultrasound performed after treatment demonstrated regression of the temporal nodular thickening, as well as resolution of the subretinal and subtenon fluid (Figure 3).

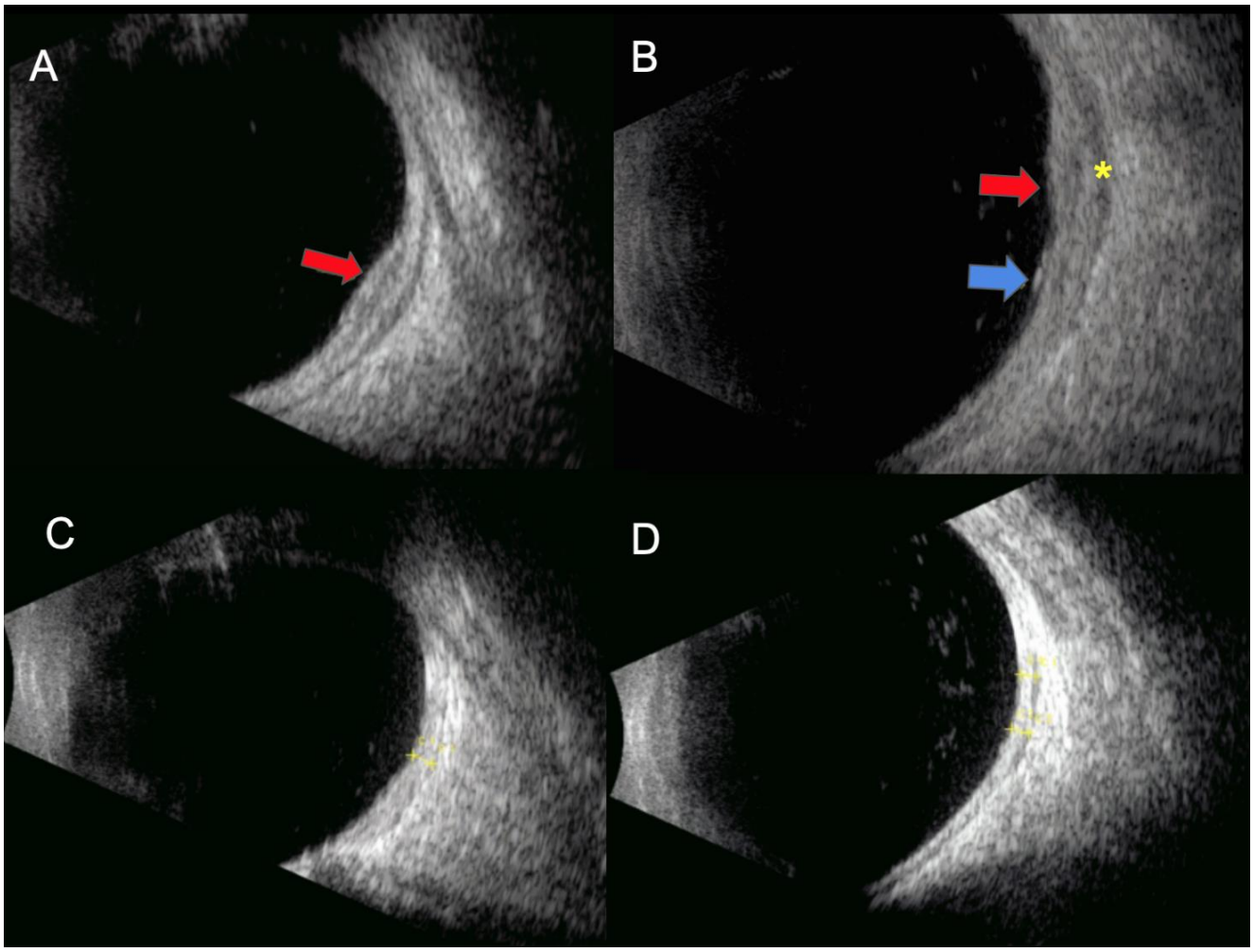


**Figure 3.** B-scan ultrasound of the left eye, pre (A,B) and post-treatment (C,D). (A,B) Temporal scans showing nodular parietal thickening (red arrow) with adjacent subretinal fluid (blue arrow) and subtenon fluid (yellow asterisk). (C,D) Regression of nodular thickening and resolution of subretinal and subtenon fluid.

After the end of the systemic therapy scheme, there was complete improvement of the pain, without recurrence of the inflammatory condition during a follow-up period of more than 12 months, reinforcing the diagnosis of presumed ocular tuberculosis.

CASE 2

A 56-year-old female patient, diabetic and hypertensive, with no relevant past ophthalmological history, presented to the emergency service complaining of reduced visual acuity and pain in her left eye that had started approximately 2 months prior to the first evaluation. The patient denied other symptoms, as well as recent travel, contact with sick people, or exposure to animals.

On initial ophthalmological examination, visual acuity in the right eye was 20/30 and 20/70 in the left eye. Anterior biomicroscopy revealed mild conjunctival hyperemia, granulomatous keratic precipitates, moderate chamber reaction, and cataract in the left eye, with no alterations in the right eye. Fundoscopy showed proliferative diabetic retinopathy in both eyes, without inflammatory signs. Patient began panretinal photocoagulation for the treatment of proliferative diabetic retinopathy and underwent etiological investigation of the ocular inflammatory condition, which revealed a tuberculin skin test of 11 mm (reactive), chest tomography without significant alterations, and the absence of other possible infectious or rheumatological etiologies.

During follow-up, the patient experienced worsening of the visual acuity and pain in the left eye, without alterations in the right eye. In a new evaluation, she presented visual acuity of hand movements in the left eye, anterior biomicroscopy showed nasal conjunctival hyperemia, and fundoscopy revealed intense vitreitis,

preventing adequate evaluation of the posterior segment.

Given the association between intense ocular pain, worsening inflammation, and media opacity, it was decided to perform ocular ultrasound of the left eye. The examination revealed vitreous and subhyaloid inflammatory processes, nodular parietal thickening in the peripheral region of the superior nasal meridian, as well as subtenon fluid in all meridians, more intense in the nasal portion, compatible with posterior nodular scleritis .

Given the diagnosis of granulomatous panuveitis associated with posterior nodular scleritis, associated with a reactive tuberculin skin test, the hypothesis of presumed ocular tuberculosis was raised, and the patient started specific systemic treatment consisting of rifampicin, isoniazid, pyrazinamide, and ethambutol for 2 months, followed by rifampicin and isoniazid for 7 months (total of 9 months), associated with oral corticosteroid therapy in a tapering scheme for 5 months.

Despite therapy, there was progression of cataract and worsening of media opacity. Following phacoemulsification, macular atrophy was evidenced, presumably secondary to intense and prolonged ocular inflammation, resulting in permanent visual impairment. The patient has been followed up for more than a year, with no signs of inflammatory reactivation after the end of specific therapy. Post-treatment ultrasound did not show the previously described superior nasal scleral focal thickening, but demonstrated thickened hyaloid partially detached, associated with vitreous and subhyaloid residual processes, as well as irregularity and thickening of the posterior pole wall (Figure 4).

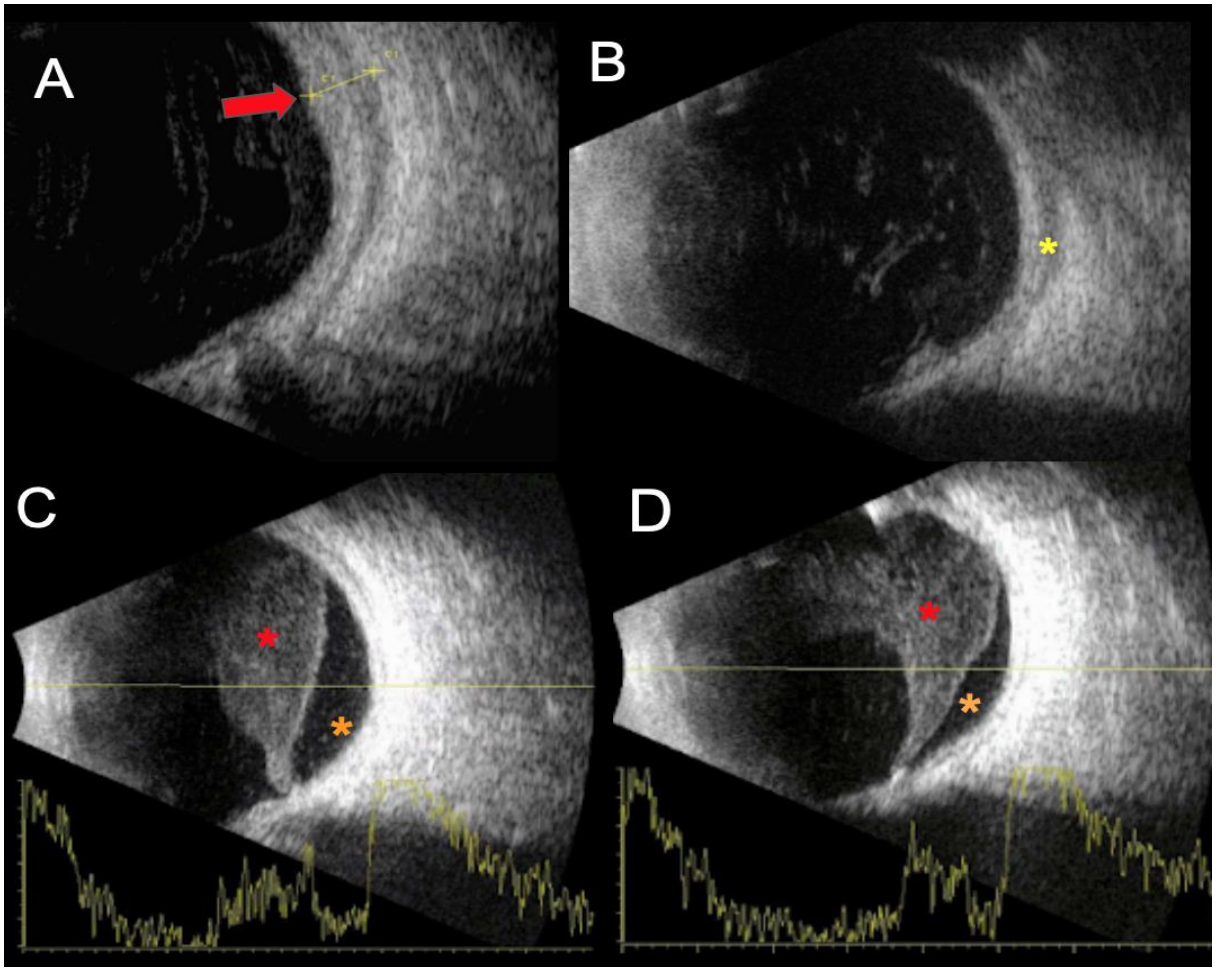


**Figure 4.** B-scan ultrasound of the left eye, pre (A,B) and post-treatment (C,D).(A) Superior nasal and (B) nasal longitudinal scans showing nodular parietal thickening (red arrow) and subtenon fluid (yellow asterisk); (C,D) nasal scans demonstrating residual vitreous (red asterisk) and subhyaloid (orange asterisk) processes with regression of nodular thickening.

## Discussion

Epidemiologically, posterior nodular scleritis is the rarest subtype of scleritis [9,10], predominantly affecting middle-aged women (average age 50 years)[5,9,11], with no predilection for ethnicity. Our two cases are concordant: both patients were women, aged 40 and 56 years, respectively, coinciding with the age range described in the literature.

Etiologically, most of the cases described are idiopathic (70-80%) [9,10,14]. In secondary cases, the main associations include autoimmune conditions such as systemic lupus erythematosus[9], rheumatoid arthritis[11], giant cell arteritis[15], polyarteritis nodosa[16] and sarcoidosis[17]. Other reported causes are inflammatory bowel disease[11], tuberculosis[8] and drugs[18]. Although ocular tuberculosis presents multiple clinical manifestations, posterior nodular scleritis is a rare but recognized form[8,19]. In Brazil, where tuberculosis is endemic, it should be considered in the differential diagnosis. The diagnosis is clinically presumed given the difficulty to isolate *Mycobacterium tuberculosis* in cases where there is only ocular involvement[20]. In both of our cases, ocular tuberculosis was considered as the etiology due to the triad of compatible clinical-radiological picture, positive tuberculin skin test and inflammatory response favorable to antituberculosis treatment.

Clinically, posterior nodular scleritis is typically unilateral[14,21], although sequential bilateral involvement has been reported [10]. The main symptoms include ocular pain that worsens with movement, although its absence does not exclude the diagnosis[9,11,18]. Reduced visual acuity is common, ranging from mild to severe depending on the degree of inflammation and extent of serous retinal detachment[9], but unchanged visual acuity is reported in about one third of cases[14]. Additional symptoms include headache, photopsias, and diplopia[12,18]. On ophthalmological examination, posterior nodular scleritis usually manifests as a well-demarcated, amelanotic subretinal elevation, frequently accompanied by subretinal fluid and choroidal/retinal folds[5,9,14,15]. Other fundoscopic findings include optic disc edema, retinal pigment epithelium changes, vasculitis, and vitritis[11,22]. Anterior segment involvement may occur in approximately half of the cases, manifesting as ciliary injection, chemosis, anterior chamber reaction, and anterior scleritis[5]. In our first patient, fundoscopic evaluation was possible, revealing typical findings, while the second, due to dense vitreitis, required ultrasound for diagnosis, demonstrating the importance of multimodality in severe inflammatory cases.

Multimodal assessment is essential for diagnostic clarification. In the context of scleritis, ocular ultrasonography is the main ancillary exam and plays a central role in performing differential diagnosis[12]. The main ultrasonographic findings of posterior nodular scleritis are a localized scleral thickening, dome-shaped or sessile, unilobulated, with medium-high homogeneous internal reflectivity, which may be associated with subtenon fluid and subretinal fluid near the nodular thickening[11]. Optical coherence tomography demonstrates choroidal elevation with overlying retinal thickening, associated with subretinal fluid and outer retinal changes[11,13,23]. In the two reported cases, ancillary exams demonstrated findings consistent with what is described in the literature. Magnetic resonance imaging also assists in diagnostic clarification, typically demonstrating an iso- or hypointense mass, with enhancement after contrast injection. However, cases with intense subretinal or subtenon fluid accumulation may present as hyperintense lesions[24]. Our patients underwent only chest imaging investigation, aiming to rule out pulmonary disease concomitant with ocular involvement.

The main differential diagnosis are intraocular tumors, especially choroidal melanoma[25], and this differentiation is essential to avoid invasive approaches in eyes with treatable inflammatory conditions[14]. Typically, choroidal melanomas manifest as a pigmented subretinal lesion, without pain or associated ocular inflammatory signs[26], presenting on ocular ultrasound as a dome or mushroom shaped lesion, with medium/low internal reflectivity and choroidal excavation, as well as absence of subtenon

fluid[9, 10, 21]. Another important differential diagnosis are choroidal metastases, which tend to be bilateral and multifocal, without inflammatory signs, presenting on ocular ultrasound with more extensive subretinal fluid and a plateau shape[14]. Finally, a satisfactory response to anti-inflammatory treatment reinforces an inflammatory etiology over a tumoral one. In both of our cases, the clinical and imaging characteristics presented are more consistent with an inflammatory rather than a tumoral etiology, which was confirmed by the treatment and follow-up of the patients.

Treatment of posterior nodular scleritis is based on two pillars: controlling the inflammatory process and managing the underlying cause, when present. Systemic corticosteroid therapy, oral or intravenous, is the main anti-inflammatory agent used in these cases, initially in high doses, followed by slow gradual reduction. The association with local corticosteroids is also reported, seeking to avoid the systemic side effects of systemic corticosteroid therapy, as well as faster control of ocular inflammation. Other agents whose use has been reported are non-steroidal anti-inflammatory drugs, both in monotherapy in mild cases and in combination with corticosteroids[9,11,14]. Approximately one-third of cases require the association of immunosuppressive agents for long-term disease control[14]. Pain tends to respond rapidly to anti-inflammatory therapy, and visual acuity improves as the inflammatory process and subretinal fluid decrease[9,10]. Scleral nodulation presents variable response in the literature, with reports of both total anatomical resolution[4,9,27,28] but also partial maintenance of a scleral nodule, despite improvement in symptomatology[14]. In our two cases, specific therapy was necessary, given the hypothesis of association with presumed ocular tuberculosis, with concomitant use of systemic corticosteroid therapy, resulting in effective inflammatory control and regression of the scleral nodule.

The prognosis for this clinical entity is generally favorable, with low recurrence rates and good visual recovery in most treated patients. Different from the anterior nodular scleritis, which can resolve the inflammation and cause a thinning of the sclera[29], the posterior nodular scleritis usually show regression of the nodule without scleral alterations[14]. Complications such as retinal pigment epithelium changes, maculopathy, glaucoma, cataracts, and epiretinal membrane have been reported.[5,9,11,14,16], Our two cases illustrate the clinical spectrum and variable prognosis of posterior nodular scleritis. The first case presented a favorable evolution with complete visual preservation, while the second resulted in macular atrophy and permanent visual impairment, despite the therapy instituted. Several factors may explain these divergent outcomes: the first case presented with early diagnosis, moderate initial inflammation, and absence of other ocular comorbidities, while the second case had a longer history, with more severe initial inflammation and concomitant proliferative diabetic retinopathy. This set of factors may have contributed to a worse visual outcome in the second case, despite treatment.

## Conclusion

Posterior nodular scleritis is an uncommon entity that can mimic intraocular tumors, and its recognition is essential to avoid unnecessary interventions. To this end, clinical aspects as well as multimodal evaluation should be considered to establish a robust diagnostic basis, allowing for the institution of appropriate therapy.

The two cases reported in this study highlight that ocular tuberculosis should be considered among the possible etiological diagnoses in the presence of posterior nodular scleritis, within a compatible clinical-epidemiological context. In these cases, adequate inflammatory control and specific therapy are fundamental, although the visual prognosis remains variable depending on factors such as early diagnosis and severity of the initial inflammation.

## Conflicts of interest:

The authors have no conflicts of interest to declare.

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