



The Red Eye that did not Respond : A Diagnostic Challenge in Scleritis

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Abstract

Scleritis is a severe ocular inflammatory condition that can be either immune-mediated or infectious. Infectious scleritis is relatively uncommon but often mimics immune-mediated disease, leading to delayed diagnosis and inappropriate therapy. We report the case of a 34-year-old female who presented with pain, redness, watering, and discharge in the left eye for 10 days, unresponsive to topical moxifloxacin prescribed for presumed conjunctivitis. Examination revealed localized scleral congestion with a yellowish abscess and overlying conjunctival ulceration. AS-OCT demonstrated scleral edema, dilated vessels, and separation of collagen fibrils. Laboratory tests and microbiological evaluation were negative for organisms. The patient was treated empirically with fortified cefotaxime and tobramycin eye drops, azithromycin ointment, oral doxycycline, and NSAIDs, resulting in rapid improvement and complete resolution within two weeks. This case emphasizes the need for early clinical suspicion and empirical broad-spectrum antibiotics in suspected infectious scleritis, even with negative cultures, and highlights the role of AS-OCT in diagnosis and monitoring.

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Key Messages

Infectious scleritis may closely resemble immune-mediated forms, making diagnosis challenging. Careful clinical examination combined with anterior segment OCT enables early differentiation, guides timely empirical antimicrobial therapy, and aids in monitoring treatment response, thereby preventing vision-threatening complications even in culture-negative cases.

Introduction

Scleritis is a severe inflammatory condition affecting the sclera, the outer protective layer of the eyeball. It can be broadly classified into infective and non-infective types, with the non-infective (immune-mediated) form being more common. Infectious scleritis is relatively rare, accounting for 5%–10% of scleritis cases in developed countries. However, in India, the incidence is significantly higher, contributing to approximately 18%–30% of reported cases.

We report rare a case of culture-negative infectious scleritis presenting as a diffuse anterior scleritis, which responded well to empirical broad-spectrum antibiotic therapy, despite the absence of typical risk factors such as trauma, prior surgery, or systemic disease.

Case History

A 34-year-old female presented with complaints of redness, pain, watering, swelling, and discharge in her left eye for the past 10 days. On elaborating the history, it was found that she was prescribed for presumed conjunctivitis a week earlier on topical moxifloxacin (0.5%) elsewhere. However, there was no improvement in symptoms. There was no history of trauma or previous ocular surgery in the affected eye.

The uncorrected visual acuity was 6/6 in both eyes on Snellen's chart. Slit-lamp examination of the right eye was unremarkable. The left eye showed inferonasal congestion with dilated episcleral and scleral vessels. A localized yellowish elevated lesion, measuring approximately 2 mm × 2 mm, was noted at the 8 o'clock scleral position (figure 1). The overlying conjunctiva appeared ulcerated, confirmed with fluorescein staining, with pus discharge was noted. The rest of the anterior segment was normal. Fundus examination of both eyes were within normal limits.



Figure 1: Left Eye Anterior segment image at presentation

A provisional diagnosis of diffuse infective anterior scleritis was made. Smear samples were sent for Gram stain, KOH mount, and culture and sensitivity. Anterior segment OCT (AS-OCT) imaging of the left eye at the lesion site revealed dilated episcleral and deep scleral vessels, scleral edema, and separation of collagen fibrils (figure 2A).

The patient was started empirically on Fortified cefotaxime (5%) eye drops hourly due to non availability of ceftazidime to cover the gram negative organisms, Tobramycin (0.3%) eye drops hourly (broad spectrum), azithromycin (1%) eye ointment at night and oral doxycycline 100 mg twice daily. Topical and oral NSAIDs were also initiated.

Laboratory investigations revealed elevated white blood cell count, ESR and CRP, borderline blood glucose levels. Gram stain showed presence of pus cells but no organisms, KOH mount and culture did not show any organism. Rheumatoid factor (RF) and anti-nuclear antibody (ANA) were negative. Mantoux test was negative.

The patient was reviewed on the second and fourth day. Her symptoms showed improvement. On examination, conjunctival injection had reduced, and the abscess had significantly subsided by the fourth day.

AS-OCT imaging repeated on day 10 showed restoration of scleral anatomy, with few residual areas of scleral edema (figure 2B).

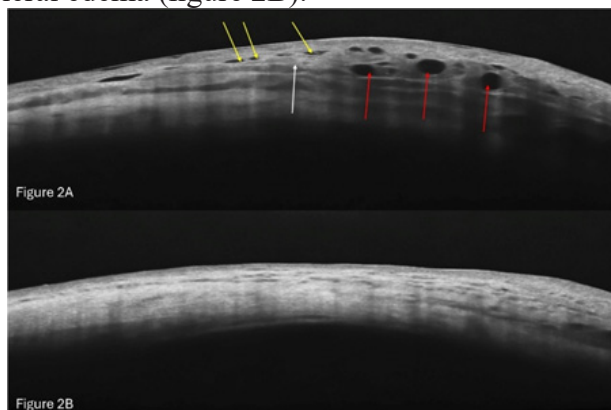


Figure 2A : anterior segment OCT imaging in LE at shows dilated episcleral vessels (yellow arrow), dilated deep scleral vessels (red arrows) and disorganized collagen fibrils (white arrow).

Figure 2B: Anterior segment OCT imaging in LE 10 days after treatment showed restored scleral anatomy.

The patient was followed up at two weeks, showed complete resolution.

Discussion

Infectious scleritis is a rare but serious ocular condition that can be difficult to distinguish from immune-mediated scleritis due to overlapping clinical features. The most commonly isolated bacterial organism is *Pseudomonas aeruginosa*¹, followed by *Staphylococcus*² and *Streptococcus* species². Among fungal etiologies, *Aspergillus flavus* is frequently reported, particularly in tropical climates². In India, tuberculosis accounts for a significant proportion up to 75% of infectious scleritis cases². Other rare but important causes include viral infections such as Herpes simplex virus and Varicella zoster virus, and systemic infections like syphilis, leprosy, and Lyme disease². Cases due to *Acanthamoeba* have also been documented, though infrequently².

One of the main challenges in diagnosing infectious scleritis lies in its clinical similarity to immune-mediated scleritis, particularly in the early stages, leading to delayed or inappropriate treatment. Accurate differentiation is crucial, as the use of corticosteroids in infectious scleritis without concurrent antimicrobial coverage can worsen the infection. Infectious

cases may present as diffuse, nodular, or necrotizing scleritis, and can feature purulent exudate, focal scleral abscesses, or even scleritis associated with hypopyon. Deep episcleral vessel congestion and scleral edema are common findings, but they are not pathognomonic. Therefore, diagnostic confirmation relies heavily on microbiological evaluation through staining, culture, and, when necessary, scleral biopsy². However, cultures may be negative if done by taking swab or scraping alone as in our case. Culture of scleral biopsy sample is preferred choice for pathogen detection. Since, in our case the initial presentation did not show scleral thinning or perforation we did not go for a biopsy.

Several risk factors have been identified in the pathogenesis of infectious scleritis, including direct ocular trauma², previous ocular surgeries¹ (such as cataract, pterygium excision³, glaucoma filtration procedures, strabismus surgery and pars plana vitrectomy⁴). Intraoperative factors like excessive cauterization and the use of antimetabolites³ such as mitomycin C can lead to localized scleral ischemia or melt, increasing susceptibility to infection. Extension from adjacent infectious foci—such as corneal ulcers or endophthalmitis² can also be responsible. Furthermore, long-term topical corticosteroid use⁵, by suppressing local immune responses, poses an additional risk. Interestingly, in our case, the patient had no identifiable risk factors, highlighting that infectious scleritis can occur even in immunocompetent individuals without surgical history or trauma.

In this case, differential diagnoses included immune-mediated nodular scleritis, necrotizing scleritis, and episcleritis. Nodular scleritis was less likely due to the absence of systemic autoimmune disease and bilateral involvement. Necrotizing scleritis was ruled out due to the lack of severe pain or scleral tissue loss. Episcleritis, being superficial and self-limiting, did not match the purulent and deeper lesion seen here. The clinical features most closely aligned with infectious scleritis.

Empirical treatment with fortified broad-spectrum antibiotics is the mainstay of management while awaiting microbiological results. A combination of topical fortified ceftazidime/cefotaxime, tobramycin, and oral doxycycline provides effective coverage against most

gram-positive and gram-negative organisms and also benefits from the anti-collagenase and anti-inflammatory effects of doxycycline. Fortified preparations of cefotaxime can be used in case of non-availability of ceftazidime^{6,7}. Topical fluoroquinolones may serve as adjunct therapy. In our patient, rapid clinical improvement following empirical antibiotic therapy, in the absence of a positive culture, supports a diagnosis of culture-negative infectious scleritis. A comprehensive flow chart on how to approach a case of scleritis has been given in (figure 3).

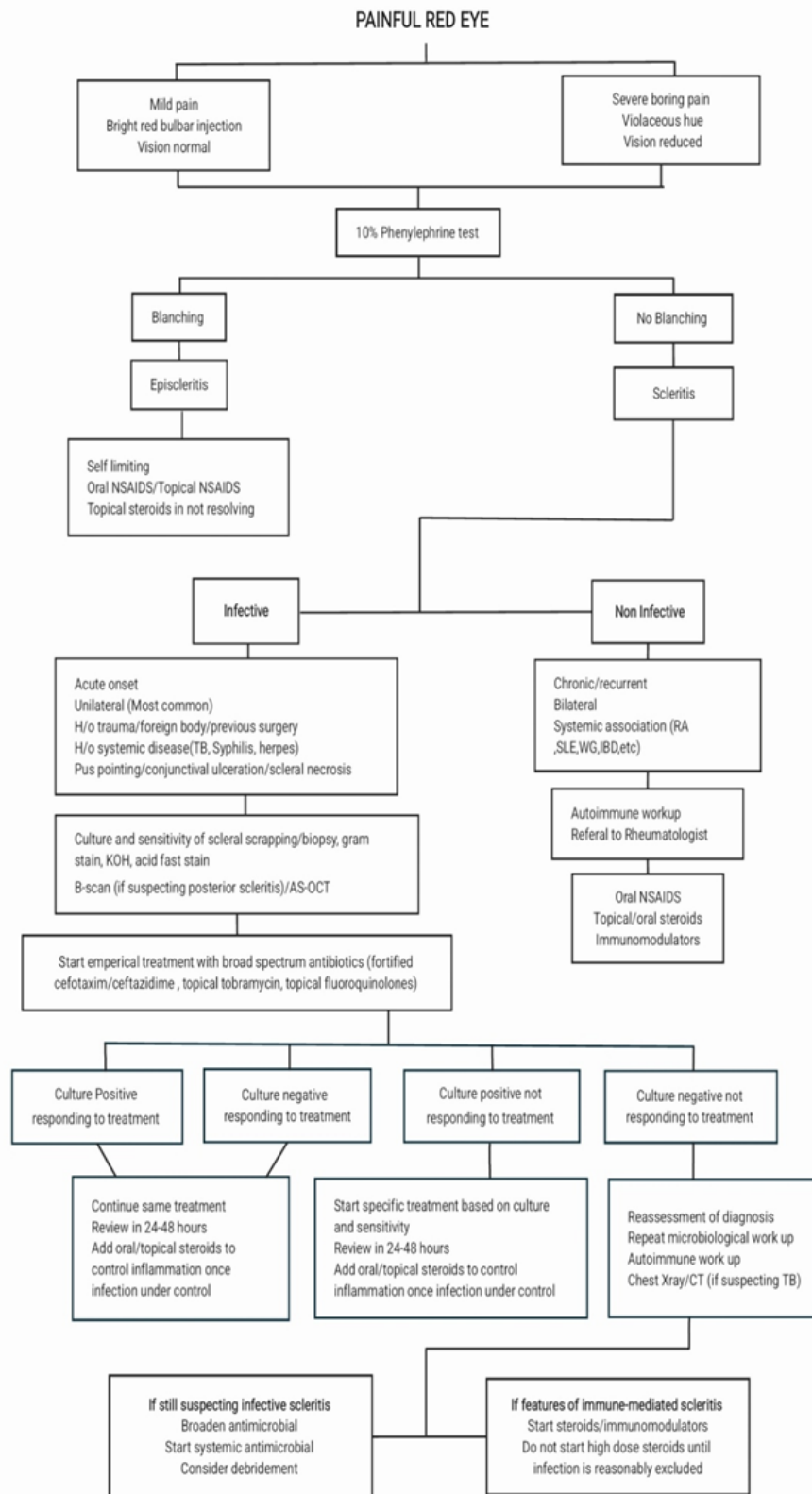


Figure: 3

This case also highlights the role of anterior segment OCT (AS-OCT)⁸ as a non-invasive imaging tool in diagnosis (episcleritis vs scleritis), objective monitoring of scleral thickness/melt and monitoring scleritis which help in accurate diagnosis in initial stage of the disease where differentiation is difficult.

Conclusion

This case underscores the importance of clinical judgment in diagnosing infectious scleritis, especially when microbiological tests fail to isolate an organism. The early stages of infectious and immune-mediated scleritis can appear remarkably similar; however, the management strategies differ significantly. Delayed or inappropriate treatment in infectious cases can lead to severe complications such as scleral perforation, necessitating surgical interventions like enucleation, and resulting in considerable morbidity. Therefore, early clinical suspicion, supported by imaging and empirical antimicrobial therapy, is essential in preventing vision and globe-threatening outcomes. This case highlights the need for ophthalmologists to consider infectious causes even in patients without classic risk factors, and to act promptly to initiate treatment in suspected cases.

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