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RECURRENT BILATERAL ANTERIOR UVEITIS IN PSORIATIC SPONDYLOARTHROPATHY WITH LATENT TUBERCULOSIS- A CASE REPORT

Ophthalmology

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ABSTRACT

Uveitis is a multifactorial intraocular inflammatory disorder that may arise from autoimmune, infectious, or idiopathic causes, often presenting diagnostic and therapeutic challenges when overlapping etiologies coexist. This report describes a 35-year-old male with psoriatic spondyloarthropathy and newly diagnosed latent tuberculosis, who experienced recurrent episodes of ocular redness, photophobia, and blurred vision for over a decade. Examination revealed anterior chamber inflammation with posterior synechiae and mild vitreous spillover. Interferon-gamma release assay (Quantiferon-TB Gold) was positive, confirming latent tuberculosis. Treatment with topical corticosteroids, cycloplegics, and isoniazid led to favorable visual and symptomatic outcomes. The case highlights the need for comprehensive systemic evaluation in patients presenting with recurrent uveitis to differentiate between autoimmune and infectious triggers.

KEYWORDS

Uveitis, Psoriatic Spondyloarthropathy, Latent Tuberculosis, Interferon Gamma Release Assay

INTRODUCTION

Uveitis is a sight-threatening intraocular inflammation that may arise from infectious, autoimmune, or idiopathic causes. The anatomical classification includes anterior, intermediate, posterior, and panuveitis. In immunocompetent individuals, autoimmune and non-infectious etiologies predominate, whereas in immuno-compromised patients, opportunistic infections such as tuberculosis are more common. The coexistence of immune-mediated systemic disease (psoriatic spondyloarthropathy) with latent tuberculosis infection poses a therapeutic challenge, as both immunosuppression and infection control need to be balanced.

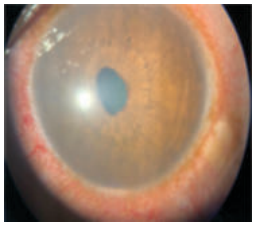
CASE REPORT

A 35-year-old male patient presented with complaints of blurring of vision, redness, and photophobia in the left eye of one week's duration. The patient reported recurrent, alternating episodes of similar ocular symptoms affecting both eyes over the preceding ten years. The systemic history was significant for psoriatic spondyloarthropathy, diagnosed in 2015, for which the patient was receiving leflunomide 10 mg once daily along with folic acid 5 mg once daily. In addition, the patient was a known case of type 2 diabetes mellitus, controlled on metformin 500 mg once daily.

On ophthalmic examination, the best-corrected visual acuity in the left eye was 6/6p. The anterior segment demonstrated endothelial dusting of the cornea with plasmoid aqueous humor and 2+ anterior chamber cells. Posterior synechiae were observed in both eyes. Fundus evaluation revealed vitreous cells graded 1+, suggestive of posterior spillover. Intraocular pressure and routine hematological parameters were within normal limits.

The patient was initiated on topical therapy with difluprednate eye drops six times daily and homatropine three times daily in the affected eye. Laboratory investigations showed a positive Quantiferon-TB Gold assay, confirming latent tuberculosis infection. Based on this finding, the patient was commenced on isoniazid 300 mg once daily for a planned six-month course.

On follow-up, the patient demonstrated stable visual acuity with symptomatic improvement and resolution of anterior chamber and vitreous inflammation. The topical corticosteroid regimen was tapered in a stepwise manner, while systemic medications were continued.



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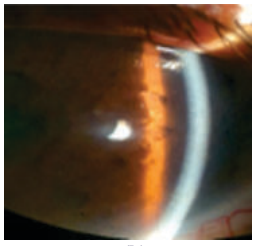

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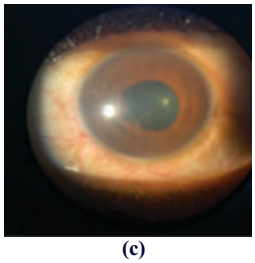

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Figure 1: Ocular findings of anterior uveitis with (a) Ciliary congestion (b) Plasmoid aqueous with cells and (c) Posterior synechiae

DISCUSSION

Determining the etiology of recurrent anterior uveitis in patients with systemic immune disease is often challenging, since clinical phenotypes overlap and infectious and non-infectious triggers may coexist. In spondyloarthritis (SpA)-including psoriatic spondyloarthropathy-uveitis typically presents as acute, unilateral, recurrent, non-granulomatous anterior uveitis [1,2]. Although the frequency of ocular involvement is lower in psoriatic arthritis compared to ankylosing spondylitis, the clinical pattern and propensity for recurrence are similar, and flares may cluster with systemic disease activity [3]. Recognition of this "signature phenotype" is clinically useful when constructing a differential in a patient with psoriatic disease and recurrent eye inflammation.

Tuberculosis (TB) adds a second critical dimension to the diagnostic process. Ocular TB may result either from (1) direct infection with viable mycobacteria, or (2) immune-mediated inflammation triggered by non-viable antigens or systemic immune responses [4,5]. This mechanistic dichotomy explains why some patients respond to corticosteroid therapy alone, while others require full anti-tubercular therapy (ATT). Interferon-gamma release assays (IGRAs) provide supportive evidence of prior TB infection but do not confirm causality for uveitis; interpretation must be integrated with clinical phenotype and chest imaging [6].

Consensus guidance provides a framework for decision-making. The Collaborative Ocular Tuberculosis Study (COTS) group recommends ATT initiation in select uveitis phenotypes when corroborative evidence of TB infection is present, with thresholds varying according to endemicity and recurrence status [7]. For recurrent anterior uveitis, many experts favor ATT if immunologic testing (IGRA/TST) is positive and imaging suggests previous TB, whereas stricter criteria apply for a first episode [8]. Parallel recommendations from respiratory societies emphasize routine LTBI screening before initiating or escalating immunosuppression, especially in TB-endemic regions, to minimize reactivation risk [9].

Management remains debated when only latent TB infection (LTBI) is detected. Observational series describe several approaches: (a) conventional multidrug ATT, either alone or combined with immunosuppressants; (b) immunosuppression with isoniazid preventive therapy when LTBI is identified in patients requiring biologics or high-dose steroids [10,11]. Our patient's favorable response to isoniazid monotherapy, in combination with topical corticosteroids, supports the hypothesis of immune-mediated inflammation rather than direct infection. Nonetheless, evidence remains heterogeneous, and some cohorts advocate conventional ATT in presumed TB-related uveitis [12]. Decisions should therefore be individualized, considering infectious-disease input, regional epidemiology, and close monitoring.

Topical management also plays a key role. Difluprednate 0.05% has been shown in randomized trials to be non-inferior to prednisolone acetate 1% for endogenous anterior uveitis, with some evidence of earlier resolution at certain time points [13]. However, the known risk of intraocular pressure elevation necessitates careful surveillance. Its use as an intensive topical regimen during acute flares, as in our patient, is thus supported by current evidence.

Overall, this case highlights three practice points: (1) in psoriatic spondyloarthropathy, recurrent non-granulomatous anterior uveitis should prompt structured evaluation for systemic drivers; (2) LTBI screening is essential prior to immunosuppression in endemic regions; and (3) therapy should be tailored to phenotype and TB probability, ranging from topical corticosteroids and cycloplegia, to preventive isoniazid with immunosuppression, or full ATT when consensus criteria are met. Close collaboration between ophthalmology, rheumatology, and infectious disease specialists remains key to preventing recurrences and preserving vision.

CONCLUSION

Uveitis in immunocompromised patients demands a thorough systemic evaluation to differentiate between autoimmune and infectious etiologies. In patients with latent tuberculosis, early initiation of anti-tubercular therapy alongside ocular management ensures better visual outcomes. Our case underlines the need for a multidisciplinary approach in managing recurrent anterior uveitis with overlapping systemic associations.

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