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Case Report

Probable Ocular Syphilis Presenting as Bilateral Panuveitis with Retinal Vasculitis in an HIV-Positive Patient with Concurrent Tuberculous Lymphadenitis: A Case Report

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ABSTRACT

Ocular inflammation in people living with HIV presents diagnostic challenges when multiple opportunistic infections coexist. Attribution of ocular pathology becomes critical when microbiologic certainty is incomplete. A man in his 20s with newly diagnosed HIV presented with progressive bilateral visual loss. Examination revealed dense vitritis, optic disc edema, and retinal vasculitis consistent with panuveitis. Syphilis serology was reactive (RPR 1:8; TPHA positive). CSF demonstrated lymphocytic pleocytosis with positive FTA-ABS. Concurrent lymph node biopsy confirmed tuberculous lymphadenitis. Weight-based antitubercular therapy was initiated, followed by intravenous aqueous penicillin G (24 million units/day for 14 days). Oral prednisolone (1 mg/kg/day) was introduced after antimicrobial coverage and tapered over six weeks. Antiretroviral therapy with tenofovir, lamivudine, and efavirenz was started two weeks after ATT initiation. Visual acuity improved from counting fingers/6-60 to 6-24/6-18 within two weeks, with sustained recovery at six months and declining RPR titers. This case highlights the complexity of diagnostic attribution in HIV-associated uveitis and supports neurosyphilis-based therapy in vision-threatening ocular syphilis despite non-reactive CSF-VDRL.

1. Introduction

Ocular inflammation in people living with human immunodeficiency virus (HIV) frequently reflects overlapping opportunistic infections, creating diagnostic and therapeutic uncertainty. Attribution of intraocular pathology becomes particularly challenging when microbiologic confirmation is incomplete.

Ocular syphilis may occur despite normal cerebrospinal fluid (CSF) indices and a non-reactive CSF-VDRL. Current treatment guidelines therefore recommend management using neurosyphilis regimens when clinical suspicion is high [1, 2]. Conversely, tuberculosis is a recognized infectious cause of uveitis; however, extrapulmonary disease alone does not establish ocular involvement, which requires phenotype correlation [3].

We present a diagnostic attribution challenge in an HIV-positive patient with concurrent microbiologically confirmed tuberculous lymphadenitis and vision-threatening panuveitis in whom therapeutic prioritization required probability-based clinical reasoning. Detailed therapeutic sequencing and outcome documentation are critical in such attribution challenges.

2. Case Presentation

A man in his 20s with newly diagnosed human immunodeficiency virus (HIV) infection, not yet initiated on antiretroviral therapy, presented with progressive painless bilateral visual blurring over one month. He also reported unintentional weight loss without fever, cough, or neurological symptoms.

He had no prior history of tuberculosis, opportunistic infections, sexually transmitted infections, or immunosuppressive therapy.

2.1. Clinical Examination

Best-corrected visual acuity was counting fingers at 2 meters in the right eye and 6/60 in the left eye. Pupillary reactions were preserved without relative afferent pupillary defect.

Anterior segment details, including anterior chamber activity and intraocular pressure, were not fully documented due to limited visualization from dense vitritis. No gross anterior inflammation or hypopyon was noted.

Posterior segment examination revealed dense bilateral vitritis, optic disc edema, and retinal perivasculitis. No necrotizing hemorrhagic lesions suggestive of cytomegalovirus retinitis or focal chorioretinal granulomas were identified within examination limits. Intraocular pressure measured by applanation tonometry was within normal limits in both eyes. Anterior chamber examination demonstrated minimal cellular reaction without hypopyon. Lens status was clear bilaterally. Posterior segment inflammation was graded using the Standardization of Uveitis Nomenclature (SUN) criteria, demonstrating vitreous haze consistent with grade 3 inflammation, limiting detailed retinal visualization.

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Neurological examination was normal. Systemic evaluation revealed a firm, mobile, non-tender posterior cervical lymph node measuring approximately 2 cm.

2.2. Initial Clinical Impression

In the setting of HIV infection with moderate immunosuppression, bilateral panuveitis with retinal vasculitis raised concern for infectious etiologies, including ocular syphilis, tubercular uveitis, viral retinitis, and toxoplasmosis, warranting comprehensive diagnostic evaluation.

2.3. Investigations

A comprehensive laboratory and systemic evaluation was undertaken to determine the etiology of intraocular inflammation in the setting of newly diagnosed HIV infection.

Syphilis serology was reactive, with a rapid plasma reagin (RPR) titer of 1:8 and a positive Treponema pallidum hemagglutination assay. HIV viral load was 75,000 copies/mL, and CD4 count was 281 cells/μL.

Lumbar puncture demonstrated cerebrospinal fluid (CSF) lymphocytic pleocytosis (12 cells/μL) with normal glucose and protein levels. CSF-VDRL was non-reactive, while CSF fluorescent treponemal antibody absorption (FTA-ABS) testing was positive. CSF treponemal positivity was interpreted as supportive rather than definitive evidence of neurosyphilis when considered alongside clinical and serologic findings [2, 4].

Excisional biopsy of the cervical lymph node revealed caseating granulomatous inflammation with acid-fast bacilli. Xpert MTB/RIF assay confirmed Mycobacterium tuberculosis without rifampicin resistance, establishing microbiologically confirmed tuberculous lymphadenitis. Chest radiography showed no pulmonary involvement.

Baseline hematologic and liver function testing was obtained prior to therapy initiation.

Multimodal ophthalmic imaging and ocular fluid polymerase chain reaction testing were not performed due to dense vitritis and urgent need for empiric therapy, and this was acknowledged as a diagnostic limitation.

Serologic and molecular testing for alternative infectious etiologies of dense vitritis in the setting of HIV infection was undertaken. Toxoplasma gondii IgG and IgM antibodies were negative. Cytomegalovirus serology and polymerase chain reaction testing were non-reactive. Herpes simplex virus and varicella zoster virus serologic testing were also negative.

Ocular fluid sampling for polymerase chain reaction testing was not performed due to dense vitritis, which limited safe sampling, and the urgency of initiating empiric antimicrobial therapy. This represents a diagnostic limitation.

2.4. Diagnostic Attribution

The principal diagnostic challenge was attributing the intraocular inflammatory phenotype to a dominant infectious etiology in the presence of two concurrently confirmed systemic infections: serologically supported ocular syphilis and microbiologically confirmed tuberculous lymphadenitis.

Tubercular uveitis was considered, given the patient's systemic disease and immunocompromised status. However, ocular tuberculosis demonstrates a heterogeneous spectrum, including choroidal granulomas, serpiginous-like choroiditis, occlusive retinal vasculitis, and multifocal chorioretinitis [3]. Within examination limits, these hallmark phenotypes were not observed.

Instead, the dominant findings of dense bilateral vitritis, optic disc edema, and retinal vasculitis are well described in ocular syphilis, particularly in people living with HIV [1, 5].

Attribution to ocular syphilis was further supported by reactive serum serology and CSF findings supportive of neurosyphilis.

Clinical improvement following intravenous penicillin therapy, occurring prior to the expected response timeline of antitubercular therapy, provided additional supportive evidence. However, this response was interpreted cautiously, given the concurrent administration of corticosteroids and antitubercular therapy.

In the absence of ocular fluid PCR testing and multimodal imaging, a mixed infectious process cannot be definitively excluded. Integrating phenotype, serology, CSF findings, and therapeutic response, ocular syphilis was considered the most probable dominant etiology.

2.5. Treatment

Given concurrent systemic infections with vision-threatening ocular inflammation, a multidisciplinary treatment strategy was implemented in coordination with ophthalmology and infectious disease specialists. Baseline hematologic profile, renal function testing, and liver function tests were obtained prior to therapy initiation. Liver enzymes were monitored periodically during treatment to evaluate for drug-induced hepatotoxicity. The patient was counseled regarding symptoms of hepatitis and drug intolerance.

2.6. Antitubercular Therapy

Standard first-line antitubercular therapy (ATT) was initiated promptly following microbiologic confirmation of tuberculous lymphadenitis. The regimen comprised isoniazid, rifampicin, pyrazinamide, and ethambutol with pyridoxine supplementation, in accordance with national and World Health Organization treatment guidelines [3, 6].

Weight-based first-line antitubercular therapy was initiated using isoniazid (5 mg/kg/day), rifampicin (10 mg/kg/day), pyrazinamide (25 mg/kg/day), and ethambutol (15 mg/kg/day) with pyridoxine supplementation.

The intended treatment duration comprised a two-month intensive phase (HRZE) followed by a four-month continuation phase (HR), in accordance with national tuberculosis control program and World Health Organization guidelines.

Baseline liver function testing and hematologic parameters were obtained prior to therapy initiation to facilitate monitoring for drug-related toxicity. The patient was counseled regarding adherence and potential adverse effects, particularly hepatotoxicity and ethambutol-associated optic neuropathy.

Where feasible, monitoring for ethambutol toxicity included clinical assessment of visual acuity and color vision during follow-up visits, recognizing the limitations imposed by pre-existing severe visual impairment at presentation. Given the risk of ethambutol-associated optic neuropathy, baseline visual acuity and color vision assessment were performed where feasible. Serial visual acuity monitoring was undertaken during follow-up visits. Formal automated perimetry could not be reliably performed due to severe baseline visual impairment and dense vitritis at presentation.

2.7. Neurosyphilis / Ocular Syphilis Therapy

Given the high clinical suspicion for ocular syphilis and CSF findings supportive of neurosyphilis, treatment was administered using a

neurosyphilis regimen irrespective of CSF-VDRL reactivity, in alignment with current treatment recommendations [2, 4, 6].

Intravenous aqueous crystalline penicillin G was administered at a dose of 24 million units per day, delivered in divided doses, for a total duration of 14 days. This was followed by intramuscular benzathine penicillin G administered weekly for three weeks to address potential concurrent latent syphilis infection, acknowledging that evidence supporting post-neurosyphilis benzathine therapy remains variable [2].

2.8. Corticosteroid Therapy

Systemic corticosteroids were introduced after initiation of antimicrobial therapy due to the presence of vision-threatening intraocular inflammation. The decision to initiate steroids was individualized and undertaken under ophthalmologic supervision.

Steroid therapy aimed to mitigate inflammatory ocular damage while antimicrobial therapy targeted the underlying infectious drivers. The timing of steroid initiation after antimicrobial coverage was intended to reduce the risk of infection exacerbation, particularly in the context of concurrent tuberculosis.

The absence of high-quality evidence supporting routine corticosteroid use in ocular syphilis was recognized, and therapy was employed on a risk – benefit basis given the severity of visual compromise. Systemic corticosteroid therapy was initiated due to vision-threatening intraocular inflammation. Oral prednisolone was commenced at a dose of 1 mg/kg/day beginning approximately 48 hours after initiation of intravenous penicillin therapy and four days after initiation of antitubercular therapy. Steroids were tapered gradually over six weeks based on clinical response.

Initiation after antimicrobial coverage was intended to mitigate the risk of exacerbating underlying infectious processes, particularly tuberculosis. Corticosteroid therapy was recognized as a potential confounder in interpreting the early inflammatory response.

2.9. Antiretroviral Therapy and IRIS Considerations

Antiretroviral therapy (ART) was initiated two weeks after commencement of antitubercular therapy. This timing strategy was selected to reduce the risk of immune reconstitution inflammatory syndrome (IRIS), particularly given the presence of active tuberculosis.

The ART regimen comprised tenofovir disoproxil fumarate (300 mg once daily), lamivudine (300 mg once daily), and efavirenz (600 mg once daily). Efavirenz was selected due to pharmacokinetic compatibility with rifampicin-based therapy, thereby minimizing drug – drug interactions while maintaining virologic efficacy.

2.10. Therapeutic Sequencing Summary

The sequence of therapeutic interventions was as follows:

1. Initiation of antitubercular therapy following lymph node biopsy confirmation.
2. Initiation of intravenous penicillin G for ocular syphilis/neurosyphilis.
3. Introduction of systemic corticosteroids after antimicrobial coverage.
4. Initiation of antiretroviral therapy two weeks after starting ATT.

This structured sequencing enabled infection-directed therapy while mitigating inflammatory ocular injury and minimizing IRIS risk. **(Table 1)** summarizes the therapeutic sequence followed by the treating physicians of the patient.

2.11. Outcome and follow-up

Clinical improvement was observed within one week of initiating intravenous penicillin therapy, with a reduction in vitritis and optic disc edema on examination. Although multimodal imaging and standardized vitreous haze grading were unavailable, clinical inflammatory regression was evident.

Visual acuity improved from counting fingers at 2 meters in the right eye and 6/60 in the left eye to 6/24 and 6/18, respectively, at two weeks. Vision remained stable at six-month follow-up with resolution of intraocular inflammation.

Systemic response included regression of cervical lymphadenopathy, HIV viral load suppression following antiretroviral therapy, rising CD4 counts, and declining RPR titers consistent with treatment response.

Baseline rapid plasma reagin titer was 1:8. Follow-up titers demonstrated serologic decline consistent with treatment response, although intermediate serial titers were not consistently available.

No major drug-related adverse effects were documented during follow-up.

3. Discussion

This case derives its clinical significance not merely from the coexistence of multiple opportunistic infections, but also from the diagnostic challenge posed by overlapping systemic and ocular pathology in an immunocompromised host. In individuals living with HIV, determining the dominant etiology of intraocular inflammation is critical because delays in pathogen-directed therapy can result in irreversible visual loss.

Ocular syphilis represents a protean manifestation of Treponema pallidum infection and may involve any segment of the eye. Panuveitis, vitritis-predominant inflammation, optic disc edema, and retinal vasculitis are well-described phenotypes, particularly among people living with HIV [1, 5]. Importantly, current treatment recommendations advise that ocular syphilis be managed using neurosyphilis regimens irrespective of cerebrospinal fluid (CSF) findings when clinical suspicion is high [2, 4, 7, 8].

In this context, lumbar puncture was pursued not solely to establish neurosyphilis but to provide supportive diagnostic information and exclude alternative central nervous system pathology. CSF-VDRL, while highly specific, lacks sensitivity. Conversely, CSF treponemal assays, such as fluorescent treponemal antibody absorption (FTA-ABS), are highly sensitive but less specific. Therefore, positive CSF treponemal testing is best interpreted as supportive rather than definitive evidence of neurosyphilis when considered in isolation and must be contextualized within clinical and serologic findings [2, 4, 7, 8].

3.1. Tuberculosis and Ocular Attribution

Tuberculosis is a recognized cause of uveitis, particularly in endemic settings and among immunocompromised individuals. However, ocular tuberculosis encompasses a heterogeneous clinical spectrum, including choroidal granulomas, serpiginous-like choroiditis, occlusive retinal vasculitis, and multifocal chorioretinitis.

Although retinal vasculitis and panuveitis can occur in ocular tuberculosis, the absence of focal choroidal or granulomatous lesions within the limits of examination reduced the probability of tuberculosis as the dominant driver of inflammation in this case. Importantly, extrapulmonary tuberculosis alone does not establish ocular involvement without phenotype correlation [3].

Table 1: Clinical Timeline of Diagnostic Evaluation and Therapeutic Interventions

Timepoint	Clinical Events	Investigations / Findings	Therapeutic Interventions	Clinical Response
1 month prior to presentation	Progressive bilateral visual blurring; weight loss	—	—	Vision decline
Day 0 (Presentation)	Severe visual impairment; dense vitritis; disc edema; retinal vasculitis	HIV diagnosed; RPR reactive; CD4 281 cells/μL	—	Active inflammation
Day 2–3	Cervical lymph node evaluation	Biopsy: caseating granulomas; AFB positive; GeneXpert MTB detected	—	—
Day 5	Lumbar puncture performed	CSF lymphocytic pleocytosis; CSF FTA-ABS positive; CSF-VDRL nonreactive	—	—
Day 6	Systemic infection confirmed	Tuberculous lymphadenitis established	Antitubercular therapy initiated (HRZE)	—
Day 8	Ocular syphilis treatment initiated	—	IV aqueous penicillin G started	—
Day 10–12	Persistent inflammation	—	Systemic corticosteroids initiated	—
Week 1 after penicillin	Early ocular improvement	Clinical reduction in vitritis and disc edema	Continued therapy	Vision improving
Week 2	Follow-up exam	Improved visual acuity	ART initiated	Continued recovery
6 months	Long-term follow-up	HIV viral suppression; rising CD4; declining RPR titers	Maintenance ART	Stable vision; resolved inflammation

ART, antiretroviral therapy; AFB, acid-fast bacilli; CSF, cerebrospinal fluid; FTA-ABS, fluorescent treponemal antibody absorption test; HRZE, isoniazid, rifampin, pyrazinamide, ethambutol; RPR, rapid plasma reagin.

3.2. Role of Therapeutic Response

Early clinical improvement following initiation of intravenous penicillin therapy provided additional supportive evidence favoring syphilitic predominance. Reduction in vitritis and disc edema was observed within a timeframe more consistent with antimicrobial response to Treponema pallidum than with antitubercular therapy.

However, therapeutic response must be interpreted cautiously. Concurrent administration of systemic corticosteroids and antitubercular therapy represents a potential confounder. Consequently, treatment response was considered supportive rather than confirmatory evidence of diagnostic attribution.

3.3. ART Timing and IRIS Considerations

Antiretroviral therapy was initiated two weeks after commencement of antitubercular therapy to mitigate the risk of immune reconstitution inflammatory syndrome (IRIS). In patients with moderate immunosuppression (CD4 >200 cells/μL), short delays in ART initiation are considered reasonable to balance virologic control against inflammatory complications, particularly in the presence of active tuberculosis. Careful regimen selection is required to account for rifampicin-associated drug – drug interactions.

3.4. Diagnostic Limitations

Several diagnostic limitations warrant acknowledgment.

Multimodal ophthalmic imaging, including optical coherence tomography and fluorescein angiography, was not available at baseline due to dense vitritis. Ocular fluid sampling with polymerase chain reaction testing for viral pathogens and Toxoplasma gondii was not performed. Consequently, alternative infectious etiologies cannot be excluded with absolute certainty.

Similarly, standardized inflammation grading and serial imaging correlation were unavailable, limiting objective quantification of the inflammatory response.

These constraints reflect real-world diagnostic challenges in resource-limited settings but introduce residual uncertainty in etiologic attribution.

Multimodal ophthalmic imaging, including optical coherence tomography and fluorescein angiography, was not feasible at baseline due to dense vitritis and limited visualization.

3.5. Clinical Implications

This case underscores the importance of probability-based therapeutic prioritization in vision-threatening uveitis when microbiologic certainty is incomplete. In such scenarios, integrating ocular phenotype, systemic investigations, serologic data, and response trajectory enables rational treatment sequencing while minimizing irreversible visual morbidity.

4. Conclusions and Learning Point

Diagnostic attribution, rather than the coexistence of infections alone, represents the central clinical challenge in HIV-associated uveitis when multiple opportunistic pathogens are identified.

- Ocular syphilis should be treated using neurosyphilis regimens in vision-threatening presentations, even in the presence of a non-reactive CSF-VDRL, particularly when CSF treponemal testing and serum serology are supportive.
- Microbiologically confirmed extrapulmonary tuberculosis does not establish ocular tuberculosis without concordant ocular phenotypes.
- Therapeutic response may provide supportive diagnostic insight in complex infectious uveitis but should not be interpreted as confirmatory proof, particularly when adjunctive corticosteroids and concurrent antimicrobial therapies are administered.
- Probability-based treatment prioritization is essential in preventing irreversible visual loss when microbiologic certainty is incomplete.
- Limitation: Definitive exclusion of alternative infectious etiologies, including ocular tuberculosis and viral retinitis, was constrained by the absence of ocular fluid PCR testing and multimodal ophthalmic imaging.

Conflicts of Interest

The authors declare no competing interests that could have influenced the objectivity or outcome of this research.

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Informed Consent

Informed consent was obtained from the patient in written form.

Large Language Model

The authors declare that generative artificial intelligence (AI) tools (ChatGPT, OpenAI) were used solely to assist in language refinement, grammatical editing, and structural organization during manuscript preparation. All clinical content, interpretations, and conclusions were developed and verified by the authors, who take full responsibility for the integrity and accuracy of the work.

Authors Contributions

All authors contributed substantially to this work. RPS conceptualized the case report, supervised clinical management, and critically reviewed the manuscript for important intellectual content. RT collected and interpreted clinical data, performed the literature review, and drafted the initial version of the manuscript. PDS structured and refined the manuscript, ensured accurate reporting of case details and references, and finalized the content for submission. All authors approved the final manuscript and agree to be accountable for all aspects of the work.

Data Availability

The data supporting the findings of this case report are not publicly available in order to protect patient privacy and confidentiality. Relevant de-identified clinical information may be made available from the corresponding author upon reasonable request, subject to ethical and institutional considerations.

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