Case Report

Blinded by the Unseen: An Uncommon Posterior Pole Variant of Ocular Toxocariasis in an Adult without Pet Exposure

Swati Singh*, Niraj Kumar Yadav, Shivalika Thakur, Swati Agarwal

Dr KNS Memorial Institute of Medical Sciences, Barabanki, Uttar Pradesh, India

Abstract

Background: Ocular toxocariasis (OT) is an infrequent parasitic infection typically affecting children, with posterior pole involvement being particularly uncommon. Adult presentations without a clear history of pet exposure complicate early recognition and clinical suspicion.

Case Presentation: We present the case of a 31-year-old woman from Barabanki, Uttar Pradesh, with unilateral ocular pain, photophobia, and progressive visual disturbance over one month. Prior empirical therapy was ineffective. Ophthalmic evaluation revealed a large elevated whitish posterior pole lesion with surrounding pigmentary changes, vitreoretinal traction bands, mild disc hyperemia, and distortion of the retinal vasculature, characteristic of posterior pole OT. Her right eye remained unaffected. Systemic and ocular history were unremarkable for risk factors such as cat/dog exposure.

Conclusion: This case highlights the diagnostic challenge of adult-onset posterior pole OT in the absence of typical epidemiologic clues. Recognition of hallmark fundus features, supported by targeted serology and imaging, is essential for preventing irreversible visual consequences.

Keywords: Ocular toxocariasis, Posterior pole granuloma, Vitreoretinal traction, Adult-onset toxocariasis, Case report.

Introduction

Ocular toxocariasis (OT) is an uncommon but vision-threatening parasitic infection caused by the migration of *Toxocara canis* or *T. cati* larvae into ocular tissues. Classically regarded as a pediatric disease, OT has increasingly been reported in adults, expanding both its epidemiological scope and clinical variability.[1-3] Human infection typically results from ingestion of embryonated *Toxocara* eggs through contaminated soil, food, or fomites, although many affected adults do not recall direct exposure to dogs or cats, reflecting the complexity of environmental transmission routes.[3,4] Once in the bloodstream, larvae may localize to the retina or vitreous, eliciting granulomatous inflammation, tractional changes, or chronic vitritis. Clinical manifestations of OT vary widely, with three major phenotypes described: peripheral granuloma, posterior pole granuloma, and chronic endophthalmitis.[5] Of these, posterior pole ocular toxocariasis (PPOT) is considered particularly severe due to its proximity to the macula and optic nerve, often resulting in significant visual morbidity. Posterior pole lesions typically present as elevated white granulomas accompanied by vitreoretinal traction bands, retinal folds, and vascular distortion, features that may mimic retinoblastoma, toxoplasmosis, tuberculosis, or neoplastic lesions, complicating early diagnosis.[6-8] Recent imaging advancements, including spectral-domain optical coherence tomography (SD-OCT), have improved characterization of PPOT, revealing subretinal hyper-reflective masses, retinal pigment epithelium (RPE) disruption, and tractional elements that aid diagnostic differentiation.[9,10] The growing number of reported adult cases underscores an evolving understanding of OT's epidemiology. Large cohort analyses demonstrate that adults may exhibit more insidious disease, with macular involvement, vitreoretinal fibrosis, and delayed presentation contributing to poorer visual outcomes.[3,11] In a landmark study of 101 adult OT patients, Ahn et al. identified posterior

Address for correspondence: Swati Singh
Dr KNS Memorial Institute of Medical Sciences, Barabanki, Uttar Pradesh, India
E-mail: swatisinghss12345@gmail.com

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pole granulomas as a significant predictor of long-term visual impairment.[3] Similarly, Despreaux et al. observed that macular edema and tractional complications were prominent in adult cohorts, highlighting the importance of early recognition and targeted intervention.[11] Despite these insights, PPOT in adults remains a diagnostic challenge, particularly when classical risk factors such as pet exposure or systemic eosinophilia are absent. This case report contributes to existing literature by documenting a rare adult presentation of posterior pole toxocariasis with characteristic tractional features but without any identifiable exposure history. Enhanced awareness of such atypical presentations is essential to avoid misdiagnosis and prevent irreversible structural and functional damage.

Case Report

This case describes an unusual presentation of adult-onset posterior pole ocular toxocariasis in a 31-year-old woman without identifiable exposure to typical vectors such as cats or dogs, the most common reservoirs for Toxocara species. The patient reported a progressive one-month history of left-eye pain, photophobia, and headaches, for which she had previously received empirical treatment including albendazole and topical anti-inflammatory and antimicrobial agents. Despite this early therapy, her symptoms persisted, prompting further evaluation at the reporting institution.

Her systemic and ocular history was otherwise unremarkable. She wore spectacles inconsistently and had no history of ocular trauma, surgery, allergic disease, systemic infections, or chronic medical conditions. Importantly, she denied contact with pets, highlighting the diagnostic complexity of toxocariasis in settings where environmental exposure may occur without direct animal interaction.

Clinical examination revealed best-corrected visual acuity of 6/36 in the left eye, significantly reduced compared to the right eye (6/6). Anterior segment findings were normal bilaterally, ruling out active anterior uveitis or keratitis. However, fundoscopic evaluation of the left eye uncovered striking posterior segment abnormalities: a large elevated whitish lesion at the posterior pole, surrounded by pigmentary alterations, accompanied by a prominent vitreoretinal traction band, retinal folds, mild optic disc hyperemia, and distortion of adjacent retinal vasculature. These features correspond closely with the classic but relatively rare phenotype of posterior pole granuloma, a hallmark of ocular toxocariasis.

The right eye remained entirely normal, consistent with the g nature of the disease.

This constellation of findings, posterior pole granuloma, vitreoretinal traction, and absence of systemic or anterior segment involvement, illustrates a distinct clinical pattern that can easily mimic infectious, inflammatory, or neoplastic retinal conditions, including toxoplasmosis, tuberculosis, and even retinoblastoma in younger patients. The delay in symptom resolution despite initial anthelmintic therapy underscores the potential for persistent inflammation or structural sequelae once tractional changes have developed. The case ultimately emphasizes the importance of recognizing posterior pole ocular toxocariasis as a diagnostic possibility even in adults without classical risk factors, particularly in endemic regions. Early identification of its characteristic fundus features is essential to initiate appropriate medical and, when needed, surgical intervention to prevent irreversible macular damage and permanent visual impairment.

Discussion

Ocular toxocariasis (OT) remains an important but frequently underdiagnosed cause of posterior uveitis, particularly when presenting in atypical demographic or clinical contexts. Although classically described in children, a growing body of epidemiological data indicates that OT in adults is more common than previously appreciated, and may follow a more insidious clinical course with greater risk for visual morbidity.[1-3] The present case involving a 31-year-old woman without pet exposure exemplifies the diagnostic complexity of adult-onset posterior pole ocular toxocariasis (PPOT), a variant that can masquerade as diverse inflammatory, infectious, and neoplastic retinal diseases.

Adult OT differs from pediatric disease in both presentation and evolution. While children more commonly demonstrate peripheral granulomas or chronic endophthalmitis, adults frequently present with posterior pole granulomas, vitreoretinal traction, or macular involvement, findings that directly threaten central vision.[1,3] Ahn et al., in a landmark cohort of 101 adults, found that posterior pole lesions were significantly associated with poorer long-term visual outcomes, especially when complicated by traction, macular edema, or retinal distortion.[3] Similarly, Despreaux et al. reported that adult OT often demonstrates prolonged inflammatory activity, macular edema, and tractional complications that can persist despite appropriate therapy.[11]

The fundoscopic features observed in this case, namely a large posterior pole granuloma with pigment disturbance, retinal folds, and a vitreoretinal traction band, align closely with classical imaging descriptions of PPOT.[5,6,9] Such lesions result from localized inflammation induced by larval migration or death, stimulating granuloma formation and reactive gliosis. The presence of traction bands, as documented here, reflects fibrocellular proliferation along vitreous scaffolds, a phenomenon well-documented in both OCT-based studies and histopathologic analyses.[7,9,10] These structural alterations can persist even after the inflammatory phase subsides, accounting for the partial but incomplete visual recovery often reported in adult cases. A critical diagnostic challenge is the absence of typical epidemiological risk factors in many adult patients. Several studies report that a substantial proportion of adults with OT deny pet exposure, suggesting alternative transmission routes via contaminated soil, unwashed produce, or poor sanitation.[4,11] This emphasizes the need for clinicians, particularly in endemic regions, to maintain a high index of suspicion when encountering solitary posterior pole lesions accompanied by vitreoretinal traction, even without corroborating history.

Posterior pole OT frequently mimics other retinal pathologies. Retinoblastoma (in children), toxoplasmosis, sarcoidosis, tuberculosis, and even intraocular lymphoma may present with overlapping features such as elevated retinal lesions, vitreous haze, or peripapillary inflammation.[6,12] Thus, multimodal imaging, including OCT and fluorescein angiography, plays a crucial role in differentiating OT, revealing characteristic hyperreflective granulomas, RPE disruption, and tractional changes.[9,10] When serology and imaging are integrated with clinical features, diagnosis becomes far more reliable. Despite the theoretical benefits of early anthelmintic therapy, clinical outcomes in adult PPOT remain variable. Inflammatory sequelae, including epiretinal membrane formation and vitreoretinal traction, may develop regardless of parasitic eradication.[3,11,13] Therefore, early recognition, before tractional changes mature, is essential to preserving visual function. Studies have proposed combining albendazole with corticosteroids to minimize inflammatory damage, though evidence remains heterogeneous.[1,11] The significance of rapid identification and timely management in sight-threatening ocular disease is further supported by the work of Yadav *et al* (2025), whose clinical research emphasizes the critical role of early intervention in preventing irreversible structural damage. In a recent BMJ Case Report, Yadav and colleagues demonstrated how prompt recognition and treatment of a rare spontaneous scleral melt in a patient with Marfan syndrome resulted in successful preservation of ocular integrity, underscoring parallels with the present case, where delayed diagnosis of posterior pole ocular toxocariasis could have led to permanent macular injury if unmanaged.[14]

Figure 1 highlights the findings from the systematic evaluation performed to identify potential sources of granulomatous disease. The axial HRCT chest image demonstrates a small, well-defined calcified granuloma measuring 5.8 × 4.7 mm, situated in the posterior segment of the right upper lobe. Its dense central calcification and sharply demarcated borders are characteristic of a healed, inactive granulomatous lesion, most commonly associated with prior subclinical infections such as tuberculosis or certain fungal exposures. The remaining lung parenchyma appears normal, with no evidence of active infiltration, cavitation, nodularity, or lymphadenopathy. Although incidental, this radiologic finding suggests the possibility of prior systemic granulomatous exposure, thereby complementing the ocular diagnosis and reinforcing the value of a comprehensive systemic assessment in cases of unexplained ocular inflammation.

Figure 2 illustrates the hallmark posterior segment findings noted at ocular presentation. The fundus photograph reveals a large, elevated yellow-white granulomatous lesion at the posterior pole, accompanied by chorioretinal scarring, pigmentary alterations, retinal folds, and prominent vitreoretinal traction. These features strongly support the diagnosis of ocular toxocariasis, reflecting an inflammatory response to larval migration or degenerating parasitic

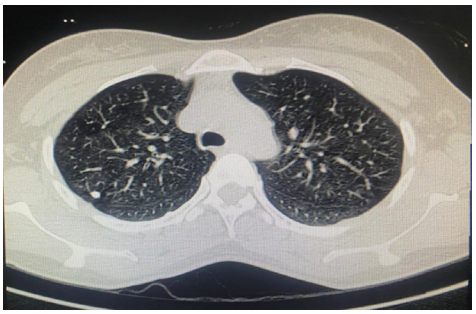

Figure 1: Axial high-resolution CT (HRCT) chest image demonstrating a small, well-defined calcified granuloma, measuring 5.8 × 4.7 mm, situated in the posterior segment of the right upper lobe. The lesion appears densely calcified with sharp margins, consistent with a healed, benign post-inflammatory granuloma. Surrounding lung parenchyma shows no evidence of active infiltrates, consolidation, cavitation, or lymphadenopathy, supporting the interpretation of a remote, inactive granulomatous process

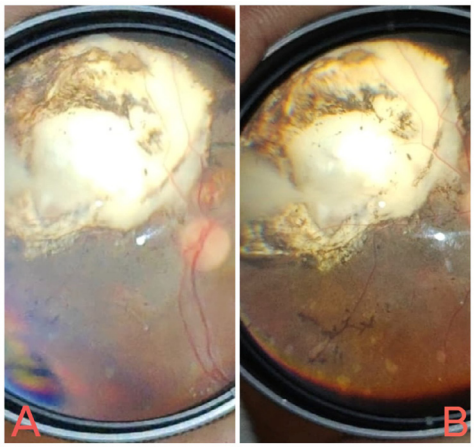

Figure 2: A) Fundus photograph demonstrating a large, elevated yellow-white granulomatous lesion at the posterior pole with surrounding chorioretinal scarring, pigmentary disruption, and prominent vitreoretinal traction, findings characteristic of ocular toxocariasis. B) Follow-up image one week after initiation of treatment showing marked clinico-investigative improvement, including sharper delineation of lesion margins and reduction in the fluffy hyper-reflective inflammatory areas, indicating partial resolution of active inflammation

remnants. The irregular, fluffy borders and tractional elements indicate an active or subacute inflammatory phase contributing to structural distortion of the macular region. One week after initiation of therapy, follow-up imaging demonstrates marked clinico-investigative improvement, including sharper delineation of lesion margins, reduction in fluffy exudative areas, and early resolution of localized inflammatory changes. This rapid response underscores the importance of timely diagnosis and intervention in preventing progressive tractional changes and irreversible visual impairment. Such outcomes align with broader ophthalmic principles emphasizing that vigilant assessment and early therapeutic action significantly improve prognosis across inflammatory, infectious, and degenerative retinal diseases. Collectively, these findings reinforce several important considerations. First, adult-onset

posterior pole ocular toxocariasis (PPOT) should remain a differential diagnosis for any solitary posterior pole lesion, even in the absence of traditional exposure history. Second, vitreoretinal traction serves as a hallmark of chronicity and carries meaningful prognostic implications. Third, early identification of characteristic ocular imaging features, supported by systemic evaluation when appropriate, facilitates prompt management and may prevent irreversible macular injury. Ultimately, this case contributes meaningfully to the existing literature by documenting an uncommon yet clinically significant presentation of PPOT in an adult without clear risk factors, highlighting the ongoing need for clinician awareness and high diagnostic suspicion in atypical retinal inflammatory presentations.

Conclusion

This case underscores the importance of maintaining a broad differential diagnosis when evaluating solitary posterior pole lesions, particularly in adults who lack classic epidemiologic risk factors for *Toxocara* exposure. The patient's presentation, marked by a posterior pole granuloma, vitreoretinal traction, and preserved anterior segment, highlights the subtle yet distinctive features of adult-onset ocular toxocariasis, a condition that is frequently overlooked or misattributed to more common retinal inflammatory or infectious diseases. Early recognition of these hallmark fundoscopic and structural findings is crucial, as delayed diagnosis increases the risk of irreversible macular damage and long-term visual impairment. Furthermore, this case reinforces that environmental transmission remains a significant route of infection and that adult patients, even in the absence of pet contact, are not exempt from disease. By documenting this uncommon presentation, the report contributes meaningful insight into the expanding clinical spectrum of ocular toxocariasis and emphasizes the ongoing need for clinician awareness, timely multimodal imaging, and early therapeutic intervention to optimize visual outcomes.

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Declaration

The authors affirm that this manuscript represents original work and has not been published previously nor is it under consideration for publication elsewhere. All authors have read and approved the final version of the manuscript.

Ethical Considerations

Written informed consent was obtained from the patient for publication of this case report and any accompanying clinical images. All procedures adhered to the ethical principles outlined in the Declaration of Helsinki. Institutional ethics committee approval was not required for single-patient case reports according to local guidelines.

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Conflict of Interest

The authors declare that there are no conflicts of interest regarding the publication of this case report.

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