Annals of Clinical and Analytical Medicine

Case Report

Atypical presumed ocular histoplasmosis syndrome: A Case Report

Atypical ocular histoplasmosis syndrome

Kardelen Taş[1], Burak Turgut[1]

[1] Department of Ophthalmology, Faculty of Medicine, Çanakkale Onsekiz Mart University, Çanakkale, Türkiye.

Abstract

Introduction: Presumed Ocular Histoplasmosis Syndrome (POHS) is a rare eye condition characterized by peripapillary atrophy, chorioretinal scars, and possible choroidal neovascularization (CNV). Although more common in endemic regions such as the United States, it can occur in non-endemic areas. This report presents a case from Türkiye, highlighting the diagnostic challenges of POHS.

Case Presentation: A 36-year-old male with no history of living in endemic areas presented with multiple peripheral chorioretinal scars in the left eye. Visual acuity was normal, with no intraocular fluid or CNV. Optical coherence tomography (OCT) and fundus fluorescein angiography confirmed POHS. His history included mitral valve prolapse, with no systemic evidence of histoplasmosis. Infectious disease tests were negative; histoplasmin testing was unavailable.

Conclusion: POHS can occur in non-endemic areas, and diagnosis requires thorough ocular examination and exclusion of other causes. Regular CNV monitoring is recommended in asymptomatic patients, especially with retinal scars of unknown etiology.

Keywords

ocular histoplasmosis syndrome, chorioretinal scars, fundus fluorescein angiography, choroidal neovascularization, OCT

DOI: 10.4328/ACAM.23023 Ann Clin Anal Med 2026;17(10):1120-1122
Received: 10.12.2025 Accepted: 14.04.2026 Published Online: 09.04.2026 Printed: 01.10.2026
Corresponding Author: Kardelen Taş, Department of Ophthalmology, Faculty of Medicine, Çanakkale Onsekiz Mart University, Çanakkale, Türkiye.
E-mail: kardelentas05@gmail.com **P:** +90 286 263 14 84
Corresponding Author ORCID: https://orcid.org/0009-0003-3847-1887
Other Authors ORCID: Burak Turgut, https://orcid.org/0000-0002-5393-0557

Introduction

Presumed Ocular Histoplasmosis Syndrome (POHS) is a rare condition typically characterized by atrophic chorioretinal scars, peripapillary atrophy, and absence of vitritis, following infection with the yeast form of Histoplasma capsulatum.[1] POHS is asymptomatic until complications such as choroidal neovascularization (CNV) or disciform scars develop. It is endemic to the river valleys of states such as Ohio and Mississippi in the United States. Our goal is to present a case of this rare disease, which is not endemic in Türkiye, in a patient referred to our clinic from an external center due to multiple chorioretinal scars in the peripheral retina.

Case Presentation

A 36-year-old male patient presented to an external clinic with a 1-week history of left eye irritation. Upon dilated fundus examination, multiple chorioretinal scars were observed, and the patient was referred to our clinic. The patient's refractive error was measured at -1.25 (-0.25, 140) in the right eye and -0.75 (0, 0) in the left eye. His visual acuity was 20/20 bilaterally. Intraocular pressure was 13 mmHg in the right eye and 17 mmHg in the left. Biomicroscopic examination revealed clear corneas and quiet anterior chambers. The patient was phakic bilaterally with clear vitreous. Dilated fundus examination showed bilateral peripapillary atrophy (Figure 1). In the macula, the right eye appeared normal, while the left eye showed whitish-yellow retinal pigment epithelium (RPE) atrophy adjacent to the superior temporal arcade. Multiple round creamy-white chorioretinal scars were seen bilaterally, outside the arcades (Figure 2). Optical coherence tomography (OCT) showed no irregularities in the inner and outer retinal layers, and no CNV or fluid accumulation was observed. Fundus fluorescein angiography revealed mid-peripheral hyperfluorescence and window-defect staining in the late phase, consistent with lesions. Additionally, in the left macula, a hyperfluorescent area near the superior temporal arcade showed no leakage in the late phase, suggesting RPE atrophy rather than active CNV (Figure 3).

The patient had a known history of mitral valve prolapse but was not on any medications. He had no travel history to endemic areas for histoplasmosis, but he had visited Georgia, Ukraine, and Belarus. He denied exposure to pets or tick bites. There was no relevant family history. The patient had no systemic symptoms of histoplasmosis, including fever, night sweats, hepatosplenomegaly, cough, or dyspnea.

To investigate potential infectious causes, the patient was consulted with the departments of infectious diseases, dermatology, and pulmonology. The results of the purified protein derivative (PPD) test were negative, and serological tests for syphilis, brucellosis, rubella, toxoplasmosis, cytomegalovirus, human immunodeficiency virus, hepatitis A virus, hepatitis B virus, angiotensin-converting enzyme, and antineutrophil cytoplasmic antibody were also negative. Routine laboratory tests, including a complete blood count, chemistry panel, and urinalysis, showed only elevated cholesterol levels. Histoplasmin skin antigen testing could not be performed due to the unavailability of kits in our hospital.

Ethical Approval

Ethical approval was not required.

This case report is presented in accordance with the CARE guidelines.

Discussion

POHS is a common multifocal chorioretinal disorder characterized by peripapillary atrophy, chorioretinal scars, and possible CNV. It is believed to be secondary to an infection caused by the dimorphic fungus Histoplasma capsulatum.[1] However, since no direct causative link has ever been established between H. capsulatum and POHS, the condition is referred to as "presumed" ocular histoplasmosis syndrome.[2] While the disease is commonly observed in endemic regions, cases have also been reported in non-endemic areas.[3] It is possible that a different pathogen could cause a similar syndrome in non-endemic areas.[1,3] While new evidence suggests a direct link between H. capsulatum and POHS, reports have also documented cases of POHS in the absence of seropositivity to H. capsulatum.[4]

The disease occurs equally in both genders, predominantly affecting adults between the ages of 20 and 50. Clinical findings typically do not include anterior chamber or vitreous inflammation. In the early stages, patients may be asymptomatic or present with visual symptoms, such as chorioretinal lesions in the mid-periphery or a choroidal neovascular membrane (CNVM) in the macula, leading to central vision loss.[1] The exact mechanism of CNVM is unclear, but it is hypothesized that chronic inflammatory reactions to fungal antigens may alter the blood-retinal barrier and trigger the release of angiogenic factors like VEGF.[5] Although some cases of POHS have shown positive histoplasmin skin antigen tests, no gold-standard diagnostic test currently exists for POHS.[6] Since POHS is not an active infectious disease, antifungal therapy is not recommended.[7] Treatment options include laser photocoagulation, vitreoretinal surgery, photodynamic therapy (PDT), corticosteroids, and anti-VEGF therapy. PDT and submacular surgery have emerged as promising treatment modalities, while traditional laser therapy is less preferred due to the risk of inducing new scotomas. Additionally, growing evidence supports the effectiveness of anti-VEGF therapies.[8]

Our case was consistent with the literature, as the patient was a 36-year-old male with no significant medical history apart from mitral valve prolapse. He had no prior history of living in endemic regions. Ophthalmic examination revealed bilateral normal visual acuity, clear anterior chambers and vitreous, with peripapillary atrophy and multiple creamy-white round chorioretinal scars in the mid-periphery. OCT showed no evidence of CNVM or fluid, and fundus fluorescein angiography demonstrated bilateral peripapillary atrophy and lesions with late-phase window defects. The RPE atrophy in the left macula was deemed unrelated to POHS and was likely due to scar tissue from a prior inflammatory process.

The patient was advised to undergo regular Amsler grid testing for early detection of CNVM. He was instructed to seek immediate evaluation if experiencing metamorphopsia or decreased visual acuity. Otherwise, routine follow-up every four months was recommended.

Since this is a case report, ethical approval was not required. However, the patient provided informed consent for the use of their medical data and images.

Limitations

Lack of Histoplasmin Skin Test: Histoplasmin skin testing was not performed due to the unavailability of test kits at our hospital, which would have provided further diagnostic confirmation.

Absence of Long-Term Follow-Up Data: The patient is still under regular follow-up; however, the case report does not include long-term follow-up data to assess disease progression or the potential development of CNV.

Conclusion

POHS can present in non-endemic countries, either asymptomatically or with central vision loss due to CNVM. Asymptomatic patients should be monitored regularly for the development of CNVM, and appropriate treatment options (anti-VEGF, photodynamic therapy, etc.) should be considered in cases of CNVM. These cases can be challenging to diagnose, so a thorough fundus examination is essential. When POHS is suspected, a travel history to endemic areas should be explored, but it is important to keep in mind that the disease can also occur in non-endemic regions. The possibility of POHS should be considered in patients with retinal scars of unknown etiology.

Declarations

Ethics Declarations

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the principles of the 1964 Declaration of Helsinki and its later amendments. Formal ethical approval was not required in accordance with institutional guidelines.

Animal and Human Rights Statement

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Declaration of Helsinki and its later amendments, or comparable ethical standards.

Informed Consent

Written informed consent was obtained from the patient for the publication of this case report and accompanying images. Patient confidentiality and anonymity were strictly preserved.

Data Availability

The data supporting this case report are available from the corresponding author upon reasonable request, subject to patient confidentiality and privacy considerations.

Conflict of Interest

The authors declare no conflict of interest.

Funding

None.

Author Contributions (CRediT Taxonomy)

Conceptualization: K.T.
Methodology: B.T.
Software: B.T.
Validation: B.T.
Formal analysis: K.T.
Investigation: K.T.
Resources: K.T.
Data curation: K.T.
Writing – original draft: K.T.
Writing – review & editing: B.T.
Visualization: K.T.
Supervision: B.T.
Project administration: B.T.

AI Usage Disclosure

The authors declare that no AI-assisted technologies were used.

Abbreviations

CARE: Case report guidelines
CNV: Choroidal neovascularization
CNVM: Choroidal neovascular membrane
OCT: Optical coherence tomography
PDT: Photodynamic therapy
POHS: Presumed ocular histoplasmosis syndrome
PPD: Purified protein derivative
RPE: Retinal pigment epithelium
VEGF: Vascular endothelial growth factor

References

1. *Diaz RI, Sigler EJ, Rafieetary MR, Calzada JI. Ocular histoplasmosis syndrome. Surv Ophthalmol. 2015;60(4):279-295. doi:10.1016/j.survophthal.2015.02.005*

2. *Macaron N, El Orra S, Orra M, et al. Presumed ocular histoplasmosis syndrome in the Middle East: a case report. Int J Clin Res. 2021;2(1):1-6. doi:10.38179/ijcr.v2i1.24*

3. *Amaro MH, Muccioli C, Abreu MT. Ocular histoplasmosis-like syndrome: a report from a nonendemic area. Arq Bras Oftalmol. 2007;70:577-580. doi:10.1590/s0004-27492007000400003*

4. *Prasad AG, Van Gelder RN. Presumed ocular histoplasmosis syndrome. Curr Opin Ophthalmol. 2005;16(6):364-368. doi:10.1097/01.icu.0000188321.61101.93*

5. *Krichene MA, Hassina S, Tebbay N, et al. Presumed ocular histoplasmosis syndrome with choroidal neovascularization: case report. Sch J Med Case Rep. 2024;5:894-898. doi:10.36347/sjmcr.2024.v12i05.083*

6. *Parnell JR, Jampol LM, Yannuzzi LA, et al. Differentiation between presumed ocular histoplasmosis syndrome and multifocal choroiditis with panuveitis based on morphology of photographed fundus lesions and fluorescein angiography. Arch Ophthalmol. 2001;119(2):208-212.*

7. *Giles CL, Falls HF. Further evaluation of amphotericin B therapy in presumed histoplasmosis chorioretinitis. Am J Ophthalmol. 1961;51:588-598. doi:10.1016/0002-9394(61)91600-2*

8. *Toussaint BW, Kitchens JW, Marcus DM, et al. Intravitreal aflibercept injection for choroidal neovascularization due to presumed ocular histoplasmosis syndrome: the HANDLE study. Retina. 2018;38(4):755-763.*

How to Cite

Kardelen Taş, Burak Turgut. Atypical presumed ocular histoplasmosis syndrome: A Case Report. doi:10.4328/ACAM.23023