

Ocular Toxoplasmosis in the Real World: A Case Series with Serological Correlation and Treatment Outcomes

VAIGANDLA LASYA¹, DEEPASHRI MUTALIK², MOUNESH PATIL³, ZARAFSHAN JAMADAR⁴, VIVEK WANI⁵

ABSTRACT

Ocular toxoplasmosis is recognised as the most common cause of posterior uveitis worldwide, which leads to substantial visual impairment when clinicians fail to identify and treat the condition adequately. This case series presents the clinical symptoms, serological profiles and treatment results of eight patients who had ocular toxoplasmosis and received treatment. The study subjects displayed ocular symptoms and they showed multiple eye conditions which included anterior segment involvement, vitritis, focal retinitis and retinochoroiditis. Optical Coherence Tomography (OCT) was performed to detect structural defects which appeared in the retinal tissue. All participants in the study showed positive results for *Toxoplasma gondii* IgG antibodies. The subjects in the study showed positive results for multiple co-infections which included Cytomegalovirus (CMV) and Herpes Simplex Virus (HSV), rubella and tuberculosis. The treatment plan included a course of oral co-trimoxazole which continued into maintenance therapy while some patients received oral prednisolone together with intravitreal clindamycin treatment. The study participants achieved better visual acuity after the treatment. The subject group showed visual acuity results between 6/6 and 6/36. There were no side-effects which affected the study participants. The study presents different clinical symptoms which ocular toxoplasmosis causes and shows how co-infections can complicate the diagnosis of ocular toxoplasmosis and demonstrates how treatment methods work against ocular toxoplasmosis.

Keywords: Clindamycin, Retinochoroiditis, Serologic tests, *Toxoplasma gondii*, Uveitis

INTRODUCTION

Ocular toxoplasmosis is widely recognised as the leading cause of posterior uveitis globally and can result in substantial visual impairment if not identified and treated promptly [1,2]. The disease results from infection by the intracellular protozoan parasite *Toxoplasma gondii*, which can cause recurrent episodes of necrotising retinochoroiditis showing a “headlight in the fog” appearance and variable anterior segment inflammation [3,4]. Clinical manifestations vary widely, ranging from mild focal retinitis to severe inflammation with complications such as retinal detachment and optic neuropathy [3]. There have been significant advances in various diagnostic modalities, such as serological testing and ocular imaging. But challenges remain in correlating clinical findings with serological markers and in improving treatment strategies to improve visual outcomes [5].

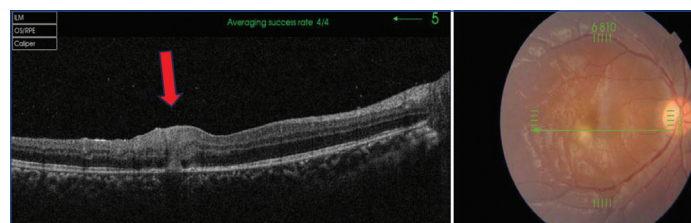
The objective of the study was to provide an overview of the clinical presentation, serological profiles, and treatment responses observed in patients diagnosed with ocular toxoplasmosis at a tertiary hospital. By analysing at these parameters, one can expand the understanding of the disease's diverse presentations and thereby provide evidence-based management approaches. The findings will add to the knowledge on ocular toxoplasmosis and emphasise the importance of combining clinical and laboratory data to provide individualised treatment regimens and enhance patient outcomes.

CASE SERIES

Case 1

An 11-year-old male presented with diminution of vision. There was no other previous medical or surgical history. Visual acuity was found to be 6/18 in the right eye. On anterior segment examination, Anterior Vitreous (AV) cells were noted, indicating intraocular inflammation. Fundus evaluation of the right eye revealed a retinitis scar located inferotemporal to the fovea [Table/Fig-1a]. The left eye showed a pigmented lesion in the macular area accompanied by a white scar situated below it [Table/Fig-1b]. Serological investigations

were positive for *Toxoplasma gondii* IgG, confirming exposure to the infectious agent. The patient was treated with oral co-trimoxazole and prednisolone, along with intravitreal clindamycin injections. Following treatment, the final visual acuity improved to 6/9 in the affected eye.



[Table/Fig-1a]: Figure showing superficial infiltration with hyperreflectivity and destruction of retinal structure with shadowing (red arrow).

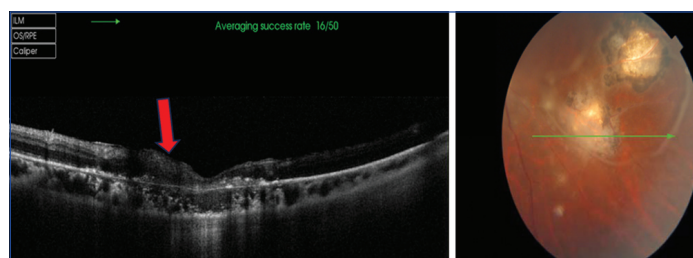


[Table/Fig-1b]: Retinal hyporeflexive space and white area shows hyperreflectivity corresponding to lesion (yellow arrow).

Case 2

A 25-year-old male presented with diminution of vision. There was no relevant medical or surgical history. Visual acuity was found to be 6/9 in the right eye. Anterior segment examination revealed the presence of AV cells, indicating active intraocular inflammation. Fundus evaluation of the right eye showed retinochoroiditis located superonasally, accompanied by vitritis [Table/Fig-2]. Serological investigations were positive for *Toxoplasma gondii* IgG, as well as Rubella IgG, CMV IgG, and HSV IgG, suggesting prior exposure to multiple infectious agents. The patient was managed with oral

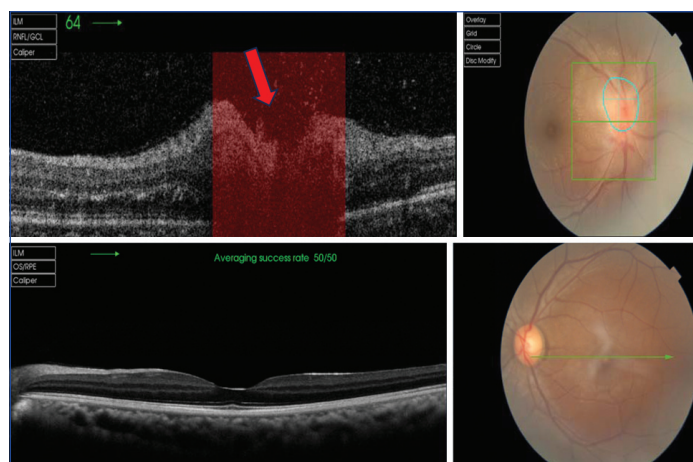
co-trimoxazole and prednisolone, supplemented by intravitreal clindamycin injections. Following treatment, the final visual acuity improved to 6/6 in the affected eye, indicating a favourable response.



[Table/Fig-2]: Figure showing thinning of retinal layers and increased hyperreflectivity of choroid due to Retinal Pigment Epithelium (RPE) loss (red arrow).

Case 3

A 38-year-old male presented with diminution of vision. There was no relevant surgical or medical history. The visual acuity was found to be 6/9 in the right eye. Fundus examination revealed blurred optic disc margins with a few retinal haemorrhages located at the superior border of the right eye [Table/Fig-3]. Serological investigations were positive for *Toxoplasma gondii* IgG, indicating prior exposure to the infectious agent. A Mantoux test was also performed and found to be reactive, while MRI of the brain showed no abnormalities. The patient was treated with oral co-trimoxazole, and following therapy, the final visual acuity improved to 6/6 in the affected eye, demonstrating a favourable clinical response.



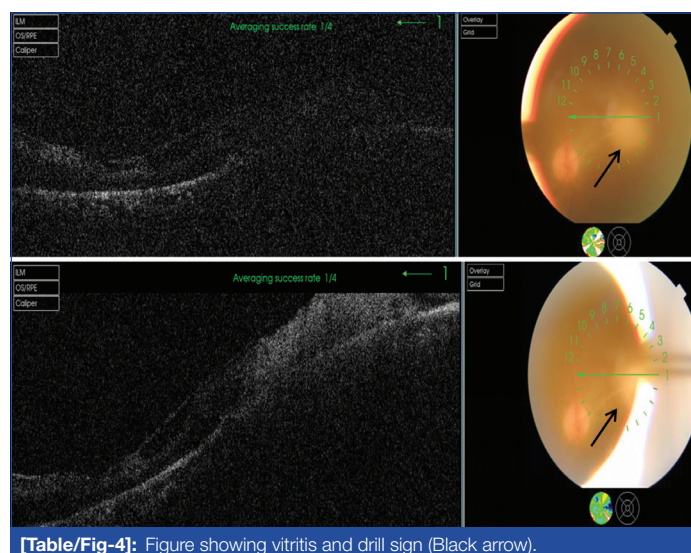
[Table/Fig-3]: Figure showing hyperreflectivity of Vitreoretinal Interface (VRI) and disorganising of retinal layers surrounding optic nerve (red arrow).

Case 4

A 47-year-old male presented with diminished vision. There was no relevant medical or surgical history. The visual acuity was found to be of counting fingers at two metres in the right eye. Anterior segment examination revealed Keratic Precipitates (KP's), Anterior Chamber (AC) cells, AV cells, and Posterior Subcapsular Cataract (PSC). Vitritis was present, and fundus evaluation showed a white lesion superonasally consistent with retinitis and pigmentary changes around the optic disc in the right eye [Table/Fig-4]. Serological tests were positive for *Toxoplasma gondii* IgG, CMV IgG, and HSV IgG. The patient received oral co-trimoxazole and prednisolone, supplemented with intravitreal clindamycin injections. Final visual acuity improved to 6/12, indicating a positive treatment response.

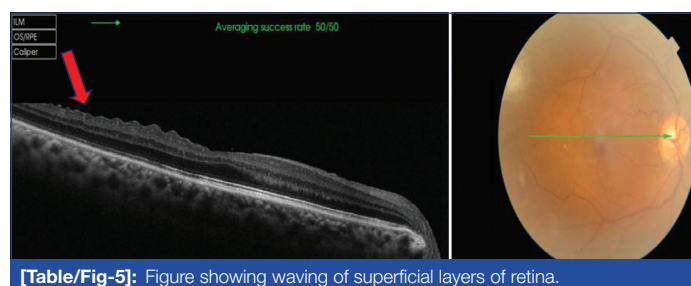
Case 5

A 50-year-old female presented with diminished vision. She was a known case of type 2 diabetes mellitus. The visual acuity was 6/9 (pinhole) in the right eye. The anterior segment examination revealed AC cells, and vitritis was present. Retinal findings included a white lesion superotemporally consistent with retinitis [Table/Fig-5]. Serological investigations were positive for *Toxoplasma gondii* IgG, CMV IgG, HSV IgG, and tuberculosis markers. The patient was



[Table/Fig-4]: Figure showing vitritis and drill sign (Black arrow).

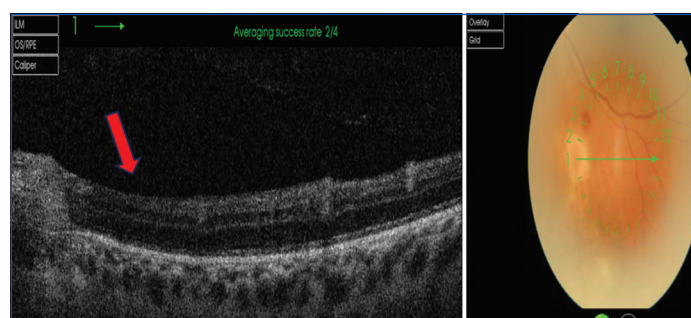
treated with oral co-trimoxazole and received intravitreal clindamycin. Final visual acuity improved to 6/6, indicating a positive treatment response.



[Table/Fig-5]: Figure showing waving of superficial layers of retina.

Case 6

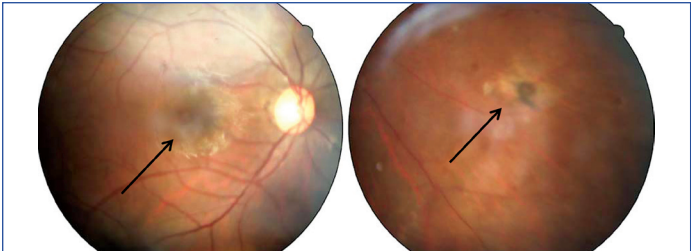
A 52-year-old male presented with headache and diminished vision. The initial visual acuity was 6/36 in the left eye. Anterior segment examination showed KP's and AV cells, with vitritis present. Retinal findings included a white lesion inferotemporally (retinitis), along with hard exudates and haemorrhages [Table/Fig-6]. Serology was positive for *Toxoplasma gondii* IgG, Rubella, and CMV. Treatment consisted of oral co-trimoxazole and prednisolone, with intravitreal clindamycin administered. Final visual acuity improved to 6/18.



[Table/Fig-6]: Figure showing hyperreflectivity corresponding to lesion.

Case 7

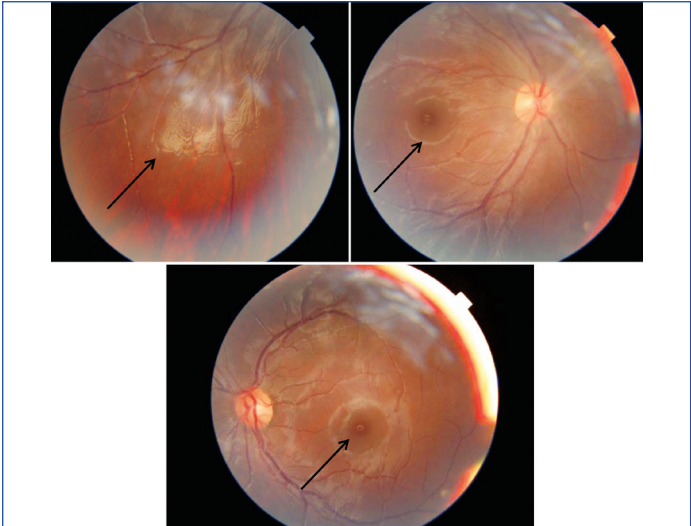
A 26-year-old male presented with diminished vision. There was no relevant medical history. The visual acuity was found to be counting fingers at three metres in the left eye and 6/6 (pinhole) in the right eye. Anterior segment signs included granulomatous KP's and AC cells in the left eye; the left eye was hazy due to vitritis, while the right eye showed a healed chorioretinal scar. Vitritis was present. Fundus evaluation indicated acute granulomatous panuveitis secondary to toxoplasmosis in the left eye and a healed chorioretinal scar in the right eye [Table/Fig-7]. Serology was positive for *Toxoplasma gondii* IgG only. The patient was treated with oral co-trimoxazole and prednisolone, and received intravitreal clindamycin. Final visual acuity in the left eye was 6/36, showing improvement.



[Table/Fig-7]: Figure showing hyperechoic lesions, suggestive of vitritis (Black arrow).

Case 8

A 17-year-old female presented with headache and diminished vision. There was no relevant medical history. The visual acuity was found to be 6/9 (pinhole) in the right eye. Anterior segment examination revealed few KP's, and vitritis was present [Table/Fig-8]. The right eye showed toxoplasmic neuroretinitis on fundus examination, with normal MRI brain findings. Serology was positive for Toxoplasma gondii IgG. Treatment included oral co-trimoxazole and prednisolone, along with intravitreal clindamycin injections. Final visual acuity improved to 6/6 (pinhole), indicating a favourable outcome.



[Table/Fig-8]: Figure showing presence of KP's and vitritis.

All patients had improvement in the Best Corrected Visual Acuity (BCVA) following treatment. Final visual acuities ranged between 6/6 and 6/36. No significant adverse events were seen during the

follow-up period. These findings point towards the clinical diversity of ocular toxoplasmosis, the presence of co-infections, and the effectiveness of combined systemic and intravitreal antimicrobial therapy in improving visual outcomes.

The summary of all the patients is presented in [Table/Fig-9,10].

DISCUSSION

This case series emphasises the clinical heterogeneity inherent in ocular toxoplasmosis, as evidenced by the varied presentations including anterior segment inflammation, retinitis, retinochoroiditis, and vitritis. Clinical observations do not correlate well with serological findings, and current treatment modalities require further refinement to achieve improved visual outcomes [6]. In this study majority of the participants had either reduced visual acuity or blindness as a result of this infection. A study by Arruda S et al., among 344 eyes revealed that 41 (30.8%) individuals, experienced blindness (6/60 or 20/200) following the condition and 33.9% had some degree of visual impairment [7].

The OCT findings of retinal structural disruption and hyperreflectivity align with active inflammatory lesions, reflecting the severity and extent of retinal involvement. Similar findings were reported by Damian I et al., [8]. Serologically, all patients demonstrated reactive Toxoplasma gondii IgG antibodies, confirming the infective etiology, while the detection of co-infections with CMV, HSV, rubella, and tuberculosis markers highlights the complexity of immune interactions and potential diagnostic challenges in endemic settings. The sensitivity of IgG assays for detecting T. gondii was 100% across all evaluated tests, indicating excellent ability to correctly identify positive cases [9]. Specificity for IgG assays in previous studies showed more variation, ranging from 96.7% to 100.0% [10,11]. For IgM assays, sensitivity varied more widely, from 80.0% to 100.0%, while specificity remained consistently high, between 99.4% and 100% [9,12].

The treatment protocol combined systemic oral co-trimoxazole along with oral corticosteroids in selected cases and intravitreal clindamycin injections in the majority of patients. This combination proved effective in achieving visual improvement among the patients. The patient presenting with optic nerve head swelling responded well to oral co-trimoxazole alone. This suggested that personalised therapy based on clinical severity and lesion location can improve outcomes. The absence of significant adverse events further supports the safety of this combined therapeutic approach as reported in previous literature [13].

Case no.	Age/Sex	Eye	Initial VA	Anterior segment findings	Vitritis	Retinal findings
1	11 y/male	Right Left	6/18	Anterior Vitreous (AV) cells +	Absent	Focal retinitis patch inferotemporal to fovea (retinitis) in right eye and pigmented lesion in macula and white scar below it in left eye
2	25 y/male	Right	6/9	Anterior Vitreous (AV) cells +	Present	Superonasally white lesion (retinochoroiditis) in right eye
3	38 y/male	Right	6/9	Not significant	Absent	Optic disc margins blurred; superior border has few retinal haemorrhages in right eye
4	47 y/male	Right	CF 2 mtr	KP's +, AC cells +, AV cells +, PSC +	Present	White lesion superonasally (retinitis) and pigmentary changes around disc in right eye
5	50 y/female	Right	6/9 (P)	AC cells +	Present	Superotemporally white lesion (retinitis)
6	52 y/male	Left	6/36	KP's +, AV cells +	Present	Inferotemporally white lesion (retinitis), hard exudates +, haemorrhages +
7	26 y/male	Left	LE: CF 3 mtr RE: 6/6 (P)	Granulomatous KP's +, AC cells +; LE – Hazy due to vitritis; RE – Healed chorioretinal scar	Present	LE: Acute granulomatous panuveitis secondary to toxoplasmosis; RE: Healed chorioretinal scar secondary to toxoplasmosis
8	17 y/female	Right	6/9 (P)	Few KP's present	Present	RE: Toxoplasmic neuroretinitis MRI Brain - normal

[Table/Fig-9]: Characteristics of the study population.

VA: Visual acuity; KP: Keratic precipitates; AC: Anterior chamber; AV: Anterior vitreous; PSC: Posterior subcapsular cataract; CF: Counting fingers; P: Pinhole; LE: Left eye; RE: Right eye; MRI: Magnetic resonance imaging

Case no.	Age/Sex	Serological investigations (Reactive)	Treatment	Intravitreal clindamycin	Final visual acuity	Improved visual acuity
1	11 y/male	Toxoplasma gondii IgG	T. Co-trimoxazole, T. Prednisolone	Given	6/9	Yes
2	25 y/male	Toxoplasma gondii IgG, Rubella IgG, CMV IgG, HSV IgG	T. Co-trimoxazole, T. Prednisolone	Given	6/6 (P)	Yes

3	38 y/male	Toxoplasma gondii IgG, Mantoux test	T. Co-trimoxazole	Not given	6/6 (P)	Yes
4	47 y/male	Toxoplasma gondii IgG, CMV IgG, HSV IgG	T. Co-trimoxazole, T. Prednisolone	Given	6/12	Yes
5	50 y/female	Toxoplasma gondii IgG, CMV IgG, HSV IgG, TB	T. Co-trimoxazole	Given	6/6	Yes
6	52 y/male	Toxoplasma gondii IgG, Rubella, CMV	T. Co-trimoxazole, T. Prednisolone	Given	6/18	Yes
7	26 y/male	Toxoplasma gondii IgG	T. Co-trimoxazole, T. Prednisolone	Given	6/36	Yes
8	17 y/female	Toxoplasma gondii IgG	T. Co-trimoxazole, T. Prednisolone	Given	6/6 (p)	Yes

[Table/Fig-10]: Characteristics of the study population continued.
IgG: Immunoglobulin G; CMV: Cytomegalovirus; HSV: Herpes simplex virus; TB: Tuberculosis; P: Pinhole

The findings in the current study are similar to the previous studies that emphasises the importance of lesion location, host immune status, and early initiation of targeted therapy in determining prognosis [14,15]. The presence of co-infections may affect the disease course and response to treatment [16]. Thus, serological examination to rule out co-infections is a necessity. Regular monitoring through clinical and imaging modalities such as OCT is crucial for assessing treatment response and detecting complications early.

This study focuses on the need for combined clinical and laboratory assessment to guide individualised management strategies in ocular toxoplasmosis, aiming to preserve visual function and prevent long-term sequelae.

CONCLUSION(S)

Ocular toxoplasmosis shows a wide range of fundus manifestations, leading to diagnostic challenges. Early recognition and timely initiation of targeted therapy are important in achieving the desired visual outcomes. Regular monitoring and follow-up are also necessary to detect disease progression, complications, and recurrences. Although many cases resolve spontaneously with residual scarring, ocular toxoplasmosis remains an important cause of preventable visual impairment worldwide.

REFERENCES

[1] Goh EJH, Putera I, La Distia Nora R, Mahendradas P, Biswas J, Chee SP, et al. Ocular toxoplasmosis. *Ocul Immunol Inflamm.* 2023;31(7):1342-61. Doi: 10.1080/09273948.2022.2117705.

[2] Kalogeropoulos D, Sakkas H, Mohammed B, Vartholomatos G, Malamos K, Sreekantam S, et al. Ocular toxoplasmosis: A review of the current diagnostic and therapeutic approaches. *Int Ophthalmol.* 2022;42(1):295-321. Doi: 10.1007/s10792-021-01994-9.

[3] Butler NJ, Furtado JM, Winthrop KL, Smith JR. Ocular toxoplasmosis II: Clinical features, pathology and management. *Clin Exp Ophthalmol.* 2013;41(1):95-108. Doi: 10.1111/j.1442-9071.2012.02838.x.

[4] Chen Z, Cheng S, Chen X, Zhang Z, Du Y. New advances in immune mechanism and treatment during ocular toxoplasmosis. *Front Immunol.* 2024;15. Doi: 10.3389/fimmu.2024.1403025.

[5] Smith JR, Cunningham ET. Atypical presentations of ocular toxoplasmosis. *Curr Opin Ophthalmol.* 2002;13(6):387-92. Doi: 10.1097/00055735-200212000-00008.

[6] Miyagaki M, Zong Y, Yang M, Zhang J, Zou Y, Ohno-Matsui K, et al. Ocular toxoplasmosis: Advances in toxoplasma gondii biology, clinical manifestations, diagnostics, and therapy. *Pathogens.* 2024;13(10):898. Doi: 10.3390/pathogens13100898.

[7] Arruda S, Vieira BR, Garcia DM, Araújo M, Simões M, Moreto R, et al. Clinical manifestations and visual outcomes associated with ocular toxoplasmosis in a Brazilian population. *Sci Rep.* 2021;11(1):3137. Doi: 10.1038/s41598-021-82830-z.

[8] Damian I, Pop A, Groza A, Miserocchi E, Nicoră SD. Structural retinal analysis in toxoplasmic retinochoroiditis: OCT follow-up with three-dimensional reconstruction. *Diagnostics.* 2025;15(23):3091. Doi: 10.3390/diagnostics15233091.

[9] Robert-Gangneux F, Guegan H. Anti-Toxoplasma IgG assays: What performances for what purpose? A systematic review. *Parasite.* 2021;28:39. Doi: 10.1051/parasite/2021035.

[10] Holec-Gąsior L, Solowińska K. IgG Avidity test as a tool for discrimination between recent and distant toxoplasma gondii infection—current status of studies. *Antibodies.* 2022;11(3):52. Doi: 10.3390/antib11030052.

[11] Ikuta K, Kanno R, Bessho T, Koshizuka T, Suzutani T. Evaluation of Toxoplasma gondii IgG avidity assays through a comparison of IgM serostatus. *Diagn Microbiol Infect Dis.* 2023;105(4):115901. Doi: 10.1016/j.diagmicrobio.2023.115901.

[12] Solowińska K, Holec-Gąsior L. IgM antibody detection as a diagnostic marker for acute toxoplasmosis: Current status of studies and main limitations. *Antibodies.* 2025;14(2):44. Doi: 10.3390/antib14020044.

[13] Patel A, Kelgaonkar A, Kaza H, Tyagi M, Murthy S, Pathengay A, et al. Recent advances in diagnosis and treatment of infectious uveitis prevalent in Asia-Pacific region. *Asia Pac J Ophthalmol.* 2021;10(1):99-108. Doi: 10.1097/APO.0000000000000367.

[14] Nabi W, Ben Amor H, Zina S, Kadri A, Khochali S, Abroug N, et al. Swept-source optical coherence tomography angiography in active and scarred toxoplasmic retinochoroiditis. *Ocul Immunol Inflamm.* 2023;31(7):1479-85. Doi: 10.1080/09273948.2022.2097096.

[15] Zicarelli F, Pichi F, Parrulli S, Oldani M, Mapelli C, Riva A, et al. Acute posterior ocular toxoplasmosis: An optical coherence tomography angiography and dye angiography study. *Ocul Immunol Inflamm.* 2022;30(3):541-45. Doi: 10.1080/09273948.2021.1977831.

[16] Galván-Ramírez M de la L, Soto-Hernández EE, Bojórquez-Pérez R. Coinfection of Toxoplasma gondii and other microorganisms: A systematic review and meta-analysis. *Microorganisms.* 2024;12(10):2106. Doi: 10.3390/microorganisms12102106.

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