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Ocular Toxoplasmosis: Bridging Clinical Diagnosis and Evidence-Based Management

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Abstract

Background: Ocular toxoplasmosis is one of the most common infectious causes of posterior uveitis and may lead to recurrent inflammation and visual impairment. It most often presents as necrotizing retinochoroiditis, but the clinical picture depends on lesion location and immune status.

Aim: This review summarizes current evidence on clinical manifestations, diagnosis and treatment of ocular toxoplasmosis.

Material and methods: A narrative review of PubMed and Scopus publications from 2002–2025 was performed, focusing on clinical presentation, diagnostic methods and therapeutic management.

Results: Ocular toxoplasmosis usually presents as focal retinochoroiditis with vitritis and, less commonly, anterior uveitis. Immunocompromised patients may develop bilateral, multifocal or extensive lesions. Diagnosis is primarily clinical and may be supported by serology, multimodal imaging, PCR testing and assessment of intraocular antibody production in uncertain cases. Conventional therapy with pyrimethamine, sulfadiazine and corticosteroids remains effective, while trimethoprim-sulfamethoxazole offers comparable efficacy with a more favorable safety profile. Intravitreal clindamycin with dexamethasone may be considered when systemic treatment is contraindicated.

Conclusions: Early diagnosis and individualized treatment are essential to reduce irreversible retinal damage. Current therapies control active inflammation but do not eradicate latent infection or reliably prevent recurrence.

Keywords: ocular toxoplasmosis, retinochoroiditis, posterior uveitis, *Toxoplasma gondii*, trimethoprim-sulfamethoxazole.

1. Introduction

Ocular toxoplasmosis (OT) is a disease caused by infection with *Toxoplasma gondii*, a ubiquitous obligate intracellular parasite that may be acquired both prenatally and postnatally. It is estimated that approximately one-third of the global population is chronically infected, although the prevalence of ocular toxoplasmosis varies depending on geographic region, climate, dietary habits, and socioeconomic factors [1,2].

Among infectious causes of posterior uveitis, OT remains one of the conditions most frequently encountered in everyday ophthalmic practice [3]. Clinically, the disease most commonly manifests as focal necrotizing retinochoroiditis, occasionally associated with anterior uveitis. In congenital cases, lesions may occur bilaterally [4]. Ocular symptoms depend on lesion localization and include decreased visual acuity, floaters, blurred vision, eye redness, and photophobia. An important characteristic of OT is its recurrent nature [1]. However, it should be emphasized that the disease may also remain asymptomatic [1,3].

Diagnosis is primarily based on clinical presentation, supported by imaging techniques and the detection of antibodies directed against parasite antigens or DNA. Analysis of intraocular fluids, including polymerase chain reaction (PCR) testing and assessment of intraocular antibody production using the Goldmann–Witmer coefficient (GWC), is reserved for atypical or diagnostically challenging cases [5].

Available treatment strategies aim to limit parasite replication during the acute phase and disease recurrence; however, they do not eliminate latent forms of the parasite or fully prevent future relapses [1,3]. Conventional therapy for ocular toxoplasmosis includes a combination of pyrimethamine, sulfadiazine, and corticosteroids. Nevertheless, trimethoprim-sulfamethoxazole is currently more commonly preferred in clinical practice [3,4].

2. Pathophysiology and Routes of Infection in Ocular Toxoplasmosis

Toxoplasma gondii is an obligate intracellular parasite whose definitive hosts are felids, while humans serve as intermediate hosts. Infection most commonly occurs through the oral ingestion of oocysts present in contaminated water or food, or tissue cysts contained in raw or undercooked meat [6]. Vertical transmission during pregnancy is also possible [7]. Following entry into the host organism, the parasite transforms into the tachyzoite stage, which is responsible for rapid replication and hematogenous dissemination to target organs, including the retina. *T. gondii* may reach the eye through the retinal vascular endothelium via several mechanisms, including transport within leukocytes or direct transmigration across the endothelium. The host immune response limits the acute phase of infection, leading to the

transformation of tachyzoites into bradyzoites and the formation of tissue cysts, which may remain latent throughout life [8]. Disease reactivation most commonly occurs following cyst rupture and parasite release, resulting in recurrent inflammation, typically within or adjacent to pre-existing retinochoroidal scars [9]. Furthermore, the presence of the parasite in the bloodstream and its ability to circulate throughout the body indicate that hematogenous spread plays a key role both in the primary ocular infection and in disease reactivation [10].

3. Clinical Manifestations

Ocular toxoplasmosis may develop at any age; however, it most commonly occurs during the second to fourth decades of life. Symptoms reported by patients include decreased vision, floaters, photophobia, and ocular redness [11].

In immunocompetent individuals, ocular toxoplasmosis most commonly presents as focal necrotizing retinitis accompanied by choroidal involvement (retinochoroiditis). Active lesions appear as whitish foci with indistinct borders, most frequently located adjacent to pre-existing retinochoroidal scars [9]. Retinal inflammation is usually accompanied by vitreous involvement (vitritis) and frequently by anterior uveitis. Vitritis is typically most pronounced in the immediate vicinity of the active retinochoroidal lesion. In severe cases, epiretinal membranes and vitreoretinal traction may develop in the area of the lesion. The severity of anterior segment inflammation may vary considerably, ranging from the absence of inflammatory cells in the anterior chamber to severe inflammation capable of obscuring posterior segment involvement. If diagnosis is delayed, prolonged inflammation within the anterior chamber may lead to permanent posterior synechiae [12].

In immunocompromised patients, the disease course is usually more aggressive and may present atypically, with multifocal or bilateral lesions, extensive inflammatory foci, or optic disc involvement. Toxoplasmic neuroretinitis is characterized by optic disc edema preceding the development of a macular star. Optic nerve involvement may result in significant visual field defects as well as impaired color vision [13,14].

Children with congenital toxoplasmosis may present with cataracts. Lens opacification is believed to develop secondary to inflammatory changes involving the posterior segment of the eye. Initially, the disease process affects the retina and choroid; subsequently, inflammation may extend to the anterior segment, leading to iridocyclitis and, consequently, cataract formation as a secondary complication [15].

Other complications of ocular toxoplasmosis include secondary glaucoma, band keratopathy, cystoid macular edema, retinal detachment, choroidal neovascularization, periphlebitis, and toxoplasmic scleritis [1].

4. Diagnosis

The diagnosis of ocular toxoplasmosis is primarily based on clinical presentation, which in typical cases may be sufficient to establish the diagnosis. However, in atypical or ambiguous cases, confirmation with additional diagnostic tests is required. Initial evaluation includes serological testing of serum for *Toxoplasma gondii*-specific antibodies (IgM and IgG), using screening methods such as enzyme-linked immunosorbent assay (ELISA) or chemiluminescent immunoassay (CLIA) [16]. IgM and IgG antibodies appear in the serum within 1–2 weeks after infection. In clinical practice, IgG antibodies are usually assessed first, followed by IgM testing if the IgG result is positive. The absence of IgG antibodies in an immunocompetent patient virtually excludes the diagnosis of toxoplasmosis. The presence of IgM antibodies suggests an early stage of infection; however, due to their prolonged persistence, they do not constitute definitive evidence of recent infection. In cases of inconclusive results, repeat testing after 2–3 weeks is recommended. Pregnancy represents a particularly important clinical situation in which the presence of IgM antibodies may indicate acute infection in both the mother and the fetus, requiring urgent diagnostic evaluation and specialist consultation [17]. A positive IgG result in the absence of clinical symptoms most commonly indicates latent infection and is of particular importance in immunosuppressed patients, including individuals with HIV infection and transplant recipients, who are at risk of disease reactivation. In this patient population, the serological response may be undetectable; therefore, in cases of strong clinical suspicion, assessment of intraocular antibody production is required. For this purpose, the Goldmann–Witmer coefficient (GWC), which compares the level of specific IgG antibodies in the aqueous humor and serum, is used. A value ≥ 3 is considered indicative of intraocular antibody production. GWC testing demonstrates higher sensitivity than real-time PCR. Polymerase chain reaction (PCR) enables direct detection of *Toxoplasma gondii* DNA in intraocular fluids and is characterized by high specificity; however, its sensitivity remains limited. This method is particularly useful in elderly patients, in cases of severe inflammation or extensive retinochoroidal lesions, and during the early stages of the disease [16].

In recent years, multimodal imaging has become an important adjunct to clinical examination and laboratory testing. Spectral-domain optical coherence tomography (SD-OCT) enables the assessment of retinal and vitreoretinal lesions, monitoring of inflammatory activity,

and detection of complications such as epiretinal membranes, posterior vitreous detachment, and retinal atrophy. Fluorescein angiography and fundus autofluorescence constitute valuable adjuncts to clinical examination, particularly in atypical presentations and during disease monitoring [16,18].

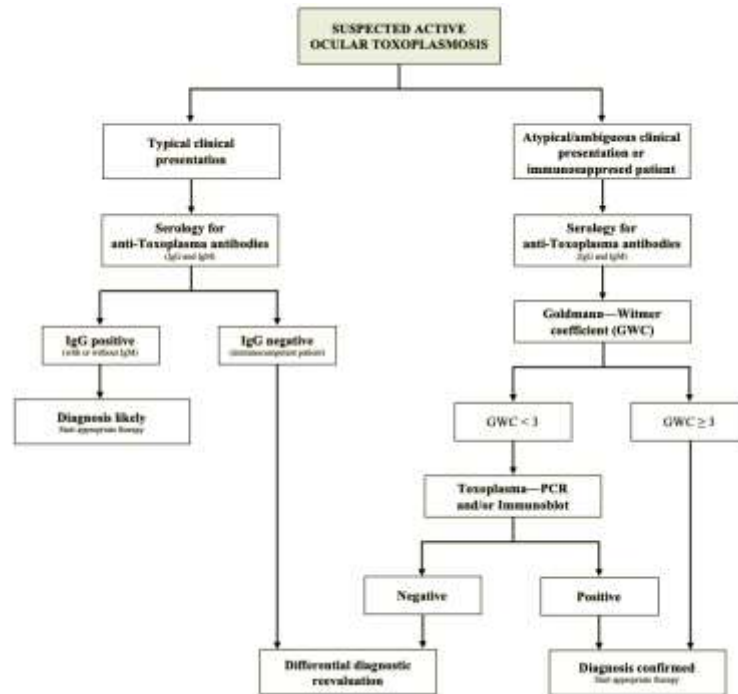


Figure 1. Algorithm for clinically tailored laboratory diagnosis of ocular toxoplasmosis.

5. Treatment

Currently, no effective therapy for ocular toxoplasmosis is capable of completely eradicating *Toxoplasma gondii* infection. The treatment of ocular toxoplasmosis aims to eliminate active infection, reduce inflammation, and prevent vision-threatening complications. In immunocompetent patients presenting with small peripheral lesions and a mild disease course, whether treatment is always necessary remains a matter of debate, as such lesions may undergo spontaneous resolution within 1–2 months. Nevertheless, treatment is recommended in most cases, as it has been shown to accelerate lesion healing and reduce the extent of retinal damage. Absolute indications for immediate treatment include lesions located near the macula or optic disc, severe vitritis, involvement of the only functioning eye, and the presence of immunosuppression [19].

5.1. Conventional Treatment

Therapeutic practices vary considerably between centers, and there is still no universally accepted treatment approach for ocular toxoplasmosis. The classic “triple-drug therapy,” used in most centers, consists of pyrimethamine, sulfadiazine and corticosteroids administered in

combination with folinic acid. Pyrimethamine is typically initiated with a loading dose of 50–100 mg, followed by a maintenance dose of 25–50 mg daily. Sulfadiazine is administered at a dose of 1–1.5 g four times daily. Prednisone is usually introduced 1–3 days after the initiation of antiparasitic therapy in order to avoid premature suppression of the host immune response before adequate parasite control is achieved. It is generally administered at a dose of approximately 20–40 mg per day and subsequently tapered over 2–6 weeks depending on the clinical response. Folinic acid, at a dose of 10–15 mg, is administered 2–3 times weekly to reduce pyrimethamine-related toxicity. However, this treatment regimen requires close monitoring due to the risk of adverse effects, including hematological abnormalities (leukopenia and thrombocytopenia), gastrointestinal symptoms, and cutaneous reactions. More severe hypersensitivity reactions, such as toxic epidermal necrolysis and Stevens–Johnson syndrome, have also been reported [11,20]. Weekly complete blood count monitoring is required throughout treatment [21–23].

5.2. Alternative Treatment Approaches for Ocular Toxoplasmosis

An alternative first-line treatment regimen is trimethoprim-sulfamethoxazole (TMP-SMX), administered as one tablet containing 160 mg of trimethoprim and 800 mg of sulfamethoxazole twice daily. This medication is widely used in the treatment of ocular toxoplasmosis due to its favorable safety profile, broad availability, and relatively low cost [20]. A randomized clinical trial demonstrated that a 6-week course of TMP-SMX combined with corticosteroids resulted in a similar reduction in lesion size and inflammatory activity compared with the conventional regimen consisting of pyrimethamine, sulfadiazine, and corticosteroids, while also being associated with fewer adverse effects [24]. Another alternative therapeutic option involves the addition of clindamycin to the conventional triple-drug regimen, thereby creating a so-called “quadruple therapy,” which has been shown to improve visual acuity and/or reduce intraocular inflammation [9].

5.3. Local Treatment

Intravitreal injection therapy for ocular toxoplasmosis represents an alternative to systemic treatment, particularly in patients with drug intolerance or contraindications to systemic therapy. Direct administration of medication into the vitreous cavity allows bypassing the blood-retinal barrier and achieving high intraocular drug concentrations at the site of infection while minimizing systemic adverse effects [22,25]. The most commonly used regimen consists of intravitreal administration of 1 mg of clindamycin combined with 400 µg of dexamethasone,

typically delivered in 1–3 injections at intervals of 1–2 weeks [25,26]. Despite its favorable safety profile, this therapeutic approach requires close monitoring due to the potential risk of complications such as retinal damage, endophthalmitis, and lens injury [11,23].

5.4. Treatment During Pregnancy

The treatment of toxoplasmosis during pregnancy depends on the timing of maternal infection and the presence of fetal infection. In cases of recent maternal infection without confirmed fetal involvement, spiramycin is administered orally at a dose of 1 g every 8 hours throughout pregnancy to reduce the risk of transplacental transmission. In cases of confirmed fetal infection, combination therapy consisting of pyrimethamine, sulfadiazine, and folinic acid is initiated. Pyrimethamine is typically administered at a dose of 20–50 mg daily in 1–2 divided doses, together with sulfadiazine at approximately 1 g every 6 hours and folinic acid at 5 mg daily. This therapeutic regimen reduces the risk of severe fetal complications; however, it should not be used during the first trimester because of its teratogenic potential and hematological toxicity. In selected cases, alternative treatment regimens may be considered, including TMP-SMX at a dose of 160/800 mg every 12 hours, azithromycin at a dose of 250 mg every 6 hours, or clindamycin at a dose of 300 mg every 6 hours. The choice of therapy should take gestational age into account [27,28].

Treatment	Drugs	Dosage	Main characteristics
Conventional therapy	Pyrimethamine + Sulfadiazine + Prednisone + Folinic acid	Pyrimethamine 50–100 mg loading dose followed by 25–50 mg daily; sulfadiazine 1–1.5 g four times daily; prednisone 20–40 mg daily; folinic acid 10–15 mg 2–3 times weekly	Effective but associated with hematologic toxicity and need for monitoring
TMP-SMX regimen	Trimethoprim-Sulfamethoxazole	160 mg/800 mg (one tablet) twice daily	Favorable safety profile, broad availability, and relatively low cost
Intravitreal therapy	Clindamycin + Dexamethasone	1 mg of Clindamycin combined with 400 µg of Dexamethasone delivered in 1–3 injections at intervals of 1–2 weeks	Reduced systemic adverse effects, but risk of injection-related complications
Pregnancy	Spiramycin	1 g every 8 hours	Reduces fetal transmission risk; the choice of therapy

Pyrimethamine + Sulfadiazine + Folinic acid	Pyrimethamine 20–50 mg daily in 1– 2 divided doses; Sulfadiazine 1 g every 6 hours; Folinic acid 5 mg daily	should take gestational age into account
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Table 1. Summary of selected treatment strategies for ocular toxoplasmosis

6. Conclusion

Despite substantial progress in diagnostic imaging and pharmacological management, ocular toxoplasmosis continues to pose a significant clinical challenge because of its recurrent nature and potential for irreversible retinal damage. The infection may remain asymptomatic; however, clinical manifestations in symptomatic patients are diverse and nonspecific. The selection of appropriate diagnostic methods and interpretation of the obtained results may vary depending on the patient's clinical category, such as immunocompetent individuals or immunocompromised patients.

Clinical evaluation plays a central role in diagnosis and is supported by laboratory testing, including antibody detection and the increasingly utilized polymerase chain reaction (PCR) method, which enables direct identification of the parasite's genetic material. Modern imaging techniques, particularly multimodal imaging, allow precise monitoring of disease progression, assessment of treatment efficacy, and early detection of lesions that may not be visible during clinical examination.

Despite the effectiveness of currently available therapies in managing active lesions, disease recurrence remains a significant clinical challenge. Conventional triple-drug therapy consisting of pyrimethamine, sulfadiazine, and corticosteroids is effective; however, alternative therapeutic approaches, such as TMP-SMX therapy and intravitreal clindamycin with dexamethasone injections, have also demonstrated efficacy. The combination of TMP-SMX is currently a commonly used therapeutic alternative due to its better tolerability and faster visual improvement.

Future studies should focus not only on controlling active inflammation but also on identifying strategies capable of reducing long-term recurrence rates.

Disclosure

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and edited the content as necessary and accept full responsibility for the substantive content of the publication.

References

1. de-la-Torre A, Mejía-Salgado G, Eraghi AT, Pleyer U. Age and ocular toxoplasmosis: a narrative review. *FEMS Microbes*. 2025;6:xtaf002. <https://doi.org/10.1093/femsmc/xtaf002>
2. Greigert V, Bittich-Fahmi F, Pfaff AW. Pathophysiology of ocular toxoplasmosis: facts and open questions. *PLoS Negl Trop Dis*. 2020;14(12):e0008905. <https://doi.org/10.1371/journal.pntd.0008905>
3. Casoy J, Nascimento H, Silva LMP, Fernández-Zamora Y, Muccioli C, Dias JRO, Nóbrega MJ, Nóbrega HAJ, Zummo J, Belfort R Jr. Effectiveness of treatments for ocular toxoplasmosis. *Ocul Immunol Inflamm*. 2020;28(2):249-255. <https://doi.org/10.1080/09273948.2019.1569242>
4. Alshalan HA, Alyousif NA, Jannadi R, Al-Dhibi H. Clinical profile and outcomes of patients with toxoplasmic retinochoroiditis: a tertiary care center experience. *Clin Ophthalmol*. 2025;19:3583-3593. <https://doi.org/10.2147/OPHTH.S552531>
5. Montoya JG. Laboratory diagnosis of *Toxoplasma gondii* infection and toxoplasmosis. *J Infect Dis*. 2002;185 Suppl 1:S73-S82. <https://doi.org/10.1086/338827>
6. Sanchez SG, Besteiro S. The pathogenicity and virulence of *Toxoplasma gondii*. *Virulence*. 2021;12(1):3095-3114. <https://doi.org/10.1080/21505594.2021.2012346>
7. Smith JR, Ashander LM, Arruda SL, Cordeiro CA, Lie S, Rochet E, Belfort R Jr, Furtado JM. Pathogenesis of ocular toxoplasmosis. *Prog Retin Eye Res*. 2021;81:100882. <https://doi.org/10.1016/j.preteyeres.2020.100882>
8. Rodriguez Fernandez V, Casini G, Bruschi F. Ocular toxoplasmosis: mechanisms of retinal infection and experimental models. *Parasitologia*. 2021;1(2):50-60. <https://doi.org/10.3390/parasitologia1020007>
9. Kalogeropoulos D, Sakkas H, Mohammed B, Vartholomatos G, Malamos K, Sreekantam S, Kanavaros P, Kalogeropoulos C. Ocular toxoplasmosis: a review of the current diagnostic and therapeutic approaches. *Int Ophthalmol*. 2022;42(1):295-321. <https://doi.org/10.1007/s10792-021-01994-9>
10. Ramírez-Flores CJ, Mondragón-Flores R. Comprehensive analysis of *Toxoplasma gondii* migration routes and tissue dissemination in the host. *PLoS Negl Trop Dis*. 2025;19(7):e0013369. <https://doi.org/10.1371/journal.pntd.0013369>

11. Goh EJH, Putera I, La Distia Nora R, Mahendradas P, Biswas J, Chee SP, Testi I, Pavesio CE, Curi ALL, Vasconcelos-Santos DV, Arora A, Gupta V, de-la-Torre A, Agrawal R. Ocular toxoplasmosis. *Ocul Immunol Inflamm*. 2023;31(7):1342-1361. <https://doi.org/10.1080/09273948.2022.2117705>
12. Delair E, Latkany P, Noble AG, Rabiah P, McLeod R, Brézin A. Clinical manifestations of ocular toxoplasmosis. *Ocul Immunol Inflamm*. 2011;19(2):91-102. <https://doi.org/10.3109/09273948.2011.564068>
13. Kabedi NN, Mwanza JC. Neuroretinitis and juxtapapillary retinochoroiditis as atypical presentations of ocular toxoplasmosis. *Int Med Case Rep J*. 2021;14:657-661. <https://doi.org/10.2147/IMCRJ.S332147>
14. Hsu C, Uwaydat SH, Chacko JG. Toxoplasma neuroretinitis. *Case Rep Ophthalmol*. 2022;13(3):751-755. <https://doi.org/10.1159/000526682>
15. Randhawa HS, Randhawa J, More A, Jain A. A rare case of unilateral fetal cataract and coincidental polydactyly in congenital toxoplasmosis. *Cureus*. 2024;16(5):e61058. <https://doi.org/10.7759/cureus.61058>
16. Gerges TK. Ocular toxoplasmosis: an update on diagnosis, multimodal imaging and therapy. In: *Infectious Eye Diseases - Recent Advances in Diagnosis and Treatment*. London: IntechOpen; 2021. <https://doi.org/10.5772/intechopen.96752>
17. Villard O, Cimon B, L'Ollivier C, Fricker-Hidalgo H, Godineau N, Houze S, Paris L, Pelloux H, Villena I, Candolfi E. Serological diagnosis of *Toxoplasma gondii* infection: recommendations from the French National Reference Center for Toxoplasmosis. *Diagn Microbiol Infect Dis*. 2016;84(1):22-33. <https://doi.org/10.1016/j.diagmicrobio.2015.09.009>
18. Ozgonul C, Besirli CG. Recent developments in the diagnosis and treatment of ocular toxoplasmosis. *Ophthalmic Res*. 2017;57(1):1-12. <https://doi.org/10.1159/000449169>
19. Bosch-Driessen LH, Verbraak FD, Suttorp-Schulten MS, van Ruyven RL, Klok AM, Hoyng CB, Rothova A. A prospective, randomized trial of pyrimethamine and azithromycin versus pyrimethamine and sulfadiazine for the treatment of ocular toxoplasmosis. *Am J Ophthalmol*. 2002;134(1):34-40. [https://doi.org/10.1016/S0002-9394\(02\)01537-4](https://doi.org/10.1016/S0002-9394(02)01537-4)
20. Casado FC, Samper AD. Sulfonamide allergy and alternative treatments in ocular toxoplasmosis. *Rom J Ophthalmol*. 2025;69(2):147-157. <https://doi.org/10.22336/rjo.2025.25>
21. Miyagaki M, Zong Y, Yang M, Zhang J, Zou Y, Ohno-Matsui K, Kamoi K. Ocular toxoplasmosis: advances in *Toxoplasma gondii* biology, clinical manifestations, diagnostics, and therapy. *Pathogens*. 2024;13(10):898. <https://doi.org/10.3390/pathogens13100898>

22. Chen Z, Cheng S, Chen X, Zhang Z, Du Y. New advances in immune mechanism and treatment during ocular toxoplasmosis. *Front Immunol.* 2024;15:1403025. <https://doi.org/10.3389/fimmu.2024.1403025>
23. Melo LA, Paiva MRB, Fernandes-Cunha GM, Silva-Cunha A, Mol MPG, Fialho SL. Clinical outcomes of intravitreal treatment for ocular toxoplasmosis: systematic review and meta-analysis. *Rev Soc Bras Med Trop.* 2023;56:e05522022. <https://doi.org/10.1590/0037-8682-0552-2022>
24. Soheilian M, Sadoughi MM, Ghajarnia M, Dehghan MH, Yazdani S, Behboudi H, Anisian A, Peyman GA. Prospective randomized trial of trimethoprim/sulfamethoxazole versus pyrimethamine and sulfadiazine in the treatment of ocular toxoplasmosis. *Ophthalmology.* 2005;112(11):1876-1882. <https://doi.org/10.1016/j.ophtha.2005.05.025>
25. Rogaczewska M, Stopa M. Intravitreal clindamycin in the treatment of ocular toxoplasmosis: a report of two cases. *Klin Ocz.* 2021;123(1):42-45. <https://doi.org/10.5114/ko.2021.104752>
26. Zamora YF, Arantes T, Reis FA, Garcia CR, Saraceno JJ, Belfort R Jr, Muccioli C. Local treatment of toxoplasmic retinochoroiditis with intravitreal clindamycin and dexamethasone. *Arq Bras Oftalmol.* 2015;78(4):216-219. <https://doi.org/10.5935/0004-2749.20150056>
27. Artiaga JCM, Azarcon CP, Levina FD, Bromeo AJ, Mesina BVQ, Arcinue CA. Considerations in the management of ocular toxoplasmosis in pregnancy: a review of literature. *Eye (Lond).* 2024;38(7):1262-1268. <https://doi.org/10.1038/s41433-023-02916-y>
28. Cortés JA, Roncancio Á, Uribe LG, Cortés-Luna CF, Montoya JG. Approach to ocular toxoplasmosis including pregnant women. *Curr Opin Infect Dis.* 2019;32(5):426-434. <https://doi.org/10.1097/QCO.0000000000000577>