

Archives of Clinical Case Reports

Review Article

Ocular Toxoplasmosis in Immunocompetent Adults: A Review of Published Cases

Jankauskas R^{1*}, Toth R² and Juric I³

¹Department of Ophthalmology, Lithuanian University of Health Sciences, Kaunas, Lithuania

²Department of Infectious Diseases, Semmelweis University, Budapest, Hungary

³Department of Clinical Microbiology, University of Zagreb, Zagreb, Croatia

*Address for Correspondence: Ruta Jankauskas, Department of Ophthalmology, Lithuanian University of Health Sciences, Kaunas, Lithuania; E-mail: ruta.jankauskas@lsmu.edu

Received: 26 January 2026; Accepted: 6 May 2026; Published: 10 August 2026; DOI: 10.5281/zenodo.22279550

Citation of this article: Jankauskas R, Toth R, Juric I (2026) Ocular Toxoplasmosis in Immunocompetent Adults: A Review of Published Cases. Arch Clin Case Rep, 9(1): 01-08.

Copyright: © 2026 Ruta Jankauskas, et al. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Abstract

Background: Ocular toxoplasmosis is a leading cause of infectious posterior uveitis, but published evidence specific to immunocompetent adults derives largely from regional cohorts, outbreak reports and small trials.

Objective: To review published case reports, case series, cohort studies and trials of ocular toxoplasmosis in immunocompetent adults.

Methods: Narrative review of case reports, case series, observational cohorts, randomised trials and systematic reviews.

Findings: Disease burden varies markedly by region, with waterborne and foodborne outbreaks producing clusters of acquired retinochoroiditis and strain genotype influencing severity. Aqueous humour testing (Goldmann-Witmer coefficient, immunoblot, PCR) supports diagnosis, but sensitivity depends on lesion typicality and timing. Randomised trials show trimethoprim-sulfamethoxazole, pyrimethamine-sulfadiazine and intravitreal clindamycin-dexamethasone produce broadly equivalent outcomes; prophylaxis reduces recurrence during treatment, but this benefit narrows by ten years.

Conclusions: Management should be individualised given the limited superiority evidence among regimens, weighing corticosteroid timing and prophylaxis duration against modest but real risks documented in the literature.

Keywords: Ocular toxoplasmosis; Retinochoroiditis; Posterior uveitis; Toxoplasma gondii; Immunocompetent

Abstract

Background: Ocular toxoplasmosis is a leading cause of infectious posterior uveitis, but published evidence specific to immunocompetent adults derives largely from regional cohorts, outbreak reports and small trials.

Objective: To review published case reports, case series, cohort studies and trials of ocular toxoplasmosis in immunocompetent adults. **Methods:** Narrative review of case reports, case series, observational cohorts, randomised trials and systematic reviews. **Findings:** Disease burden varies markedly by region, with waterborne and foodborne outbreaks producing clusters of acquired retinochoroiditis and strain genotype influencing severity. Aqueous humour testing (Goldmann-Witmer coefficient, immunoblot, PCR) supports diagnosis, but sensitivity depends on lesion typicality and timing. Randomised trials show trimethoprim-sulfamethoxazole,

pyrimethamine-sulfadiazine and intravitreal clindamycin-dexamethasone produce broadly equivalent outcomes; prophylaxis reduces recurrence during treatment, but this benefit narrows by ten years. **Conclusions:** Management should be individualised given the limited superiority evidence among regimens, weighing corticosteroid timing and prophylaxis duration against modest but real risks documented in the literature.

Keywords: ocular toxoplasmosis; retinochoroiditis; posterior uveitis; *Toxoplasma gondii*; immunocompetent

Introduction

Toxoplasma gondii is among the commonest identifiable causes of posterior uveitis globally, producing focal necrotising retinochoroiditis that can recur throughout life. Most cases are traditionally attributed to reactivation of congenitally acquired infection, but an accumulating body of published work documents acute, postnatally acquired disease in immunocompetent adults, sometimes in outbreak clusters linked to contaminated water or food [1,2]. This distinction matters clinically: acquired disease

in an adult without a childhood diagnosis of toxoplasmosis can present as a solitary lesion with no antecedent scar, mimicking other infectious or inflammatory chorioretinitides, and its recognition depends on a combination of epidemiological history, serology and, increasingly, molecular testing of intraocular fluid.

This is a narrative review, not a report of the authors' own patients: it surveys published case reports, case series, cohort studies, randomised trials and systematic reviews addressing ocular toxoplasmosis specifically in immunocompetent adults, organised around epidemiology, pathophysiology, clinical presentation, diagnosis, management (including recurrence prophylaxis) and outcomes. Where the underlying literature includes congenital cohorts or mixed immune-status populations, this is stated explicitly rather than extrapolated silently to immunocompetent adults.

Epidemiology

Seroprevalence and ocular disease burden vary widely by geography. An early Brazilian cohort reported an unusually high prevalence of ocular toxoplasmosis in southern Brazil, implicating a heavily contaminated local environment [3], a pattern later corroborated by global reassessments describing marked regional variation in incidence and severity [4]. Point-source outbreaks illustrate acquired disease directly: a municipal drinking-water outbreak in British Columbia produced confirmed acute toxoplasmosis in exposed residents [1], and a related report documented multiple cases of acquired toxoplasmic retinitis presenting within that outbreak [2]. In a Serbian uveitis referral centre, toxoplasmosis accounted for 59 of 457 uveitis patients (12.9%), the third leading identifiable cause, with recurrent disease (64%) outnumbering first-episode presentations (36%) [5]. A large Brazilian series of 262 subjects (344 eyes) found active disease in just over half, again predominantly recurrent, with HIV co-infection in

only 3.8% — underscoring that most published ocular disease occurs in immunocompetent hosts [6]. A comprehensive review of clinical features and prognosis further situates these regional cohorts within the wider global picture [7]. Taken together, these sources indicate that toxoplasmosis is not a rare or marginal cause of uveitis in adults with normal immune function; rather, in the settings where it has been systematically counted, it ranks among the leading identifiable aetiologies, and a meaningful minority of cases represent recently acquired infection rather than late reactivation of childhood disease.

Pathophysiology

Ocular disease follows tachyzoite invasion of retinal tissue with cyst formation, chronic latency, and periodic reactivation producing focal necrotising retinochoroiditis adjacent to pre-existing scars, or, in postnatally acquired disease, without any antecedent scar. Strain genotype appears to influence severity: Brazilian ocular isolates show unusual abundance of atypical, non-clonal genotypes distinguishable from the clonal types dominant elsewhere, with the majority of genotyped Brazilian strains sharing polymorphisms not seen in classical lineages [8]. Comparative congenital cohorts illustrate the clinical consequence of such strain diversity: Brazilian children had substantially higher rates of chorioretinal lesions by four years of age and worse visual outcomes than European children, with a hazard ratio for lesion development exceeding five [9]. Although this comparison derives from congenital rather than adult-acquired disease, it supports a broader inference that regional strain virulence, not host immune status alone, shapes disease severity, and it offers one explanation for why acquired disease in Brazilian and other Latin American cohorts tends to appear more aggressive than the disease traditionally described in European case series. The immune response itself also contributes to tissue damage: retinochoroiditis is thought to reflect not only direct

parasite-mediated cytolysis but also a host inflammatory reaction to released antigen, which is the pathophysiological rationale usually offered for adjunctive corticosteroid use, notwithstanding the absence of controlled trial evidence isolating that benefit (see Management, below).

Clinical Presentation

Presentation ranges from asymptomatic peripheral scarring to sight-threatening macular or juxtapapillary lesions. In a non-endemic uveitis service, 16 patients with primary acquired infection and ocular involvement had a mean age of 53 years; most (69%) had a single retinitis focus without a pre-existing scar, and just over half reported systemic symptoms [10]. Published individual cases add texture to these cohort figures: a 69-year-old immunocompetent traveller developed bilateral necrotising chorioretinitis without prior scarring three weeks after a South American cruise, with vitreous and aqueous PCR both positive, illustrating recently acquired rather than reactivated disease [11]. A recent case series of punctate outer retinal toxoplasmosis described eight patients with predominantly unilateral, often perifoveal lesions and variable baseline acuity from 20/40 to counting fingers, highlighting an atypical, deep retinal presentation that can mimic other white-dot syndromes [12]. In the large Brazilian series, typical lesion morphology predominated (96.6%), and roughly one in three eyes active at presentation had visual acuity worse than 20/400 [6]. The heterogeneity across these sources is instructive: presentation age, laterality, presence of a pre-existing scar and baseline acuity all vary widely even within single case series, so no single vignette should be treated as archetypal. Systemic symptoms such as malaise, lymphadenopathy or low-grade fever accompany a substantial minority of acquired cases and, when present alongside a compatible fundus lesion, should raise suspicion for recently acquired rather than reactivated infection [10].

Table 1: Published cases of ocular toxoplasmosis in immunocompetent adults reviewed here [12,11,27]; N = 8 individual cases.

First author, year	Age/s ex	Lesion location/size	VA at presentation	Treatment regimen	Outcome /recurrence
Paquenti n-Jimenez 2025, case 1	70s/F	Macular, macular hole	20/800	TMP-SMX + clindamycin + prednisone taper, 2mo	20/200 at 14 months
Paquenti n-Jimenez 2025, case 2	40s/F	Not stated; epiretinal membrane	Counting fingers	TMP-SMX + clindamycin + prednisone, 2mo; retreated	20/40
Paquenti n-Jimenez 2025, case 3	40s/M	Not stated	20/100	TMP-SMX + clindamycin + prednisone, 3mo	Worsened to 20/400
Paquenti n-Jimenez 2025, case 5	30s/F	Reactivation adjacent to old scar	20/60	TMP-SMX + clindamycin + prednisone, 2mo	20/40, residual scotoma
Paquenti n-Jimenez 2025, case 6	30s/F	Perifoveal, macular oedema	20/40	TMP-SMX + clindamycin + prednisone 2mo + topical	20/20 at 2 months
Paquenti n-Jimenez 2025, case 8	30s/F	Not stated; old scars	20/100	TMP-SMX + clindamycin + prednisone + topical, 3mo	20/30 at 4 months
Seers 2021	69/M	Bilateral, no pre-existing scars	0.9 logMAR OD/0.5 logMAR OS	TMP-SMX 3mo + azithromycin + prednisolone	Inactive bilaterally at 6 months
Lin 2022	42/M	Superonasal, not stated	20/20	SMZ-TMP + IV dexamethasone; escalated to TMP-SMX + prednisolone; vitrectomy	Final 20/80 after retinal detachment

Diagnostic Approach

Diagnosis rests on characteristic fundus appearance supported, where atypical or diagnosis is uncertain, by intraocular fluid analysis. In typical disease, immunoblot and the Goldmann-Witmer coefficient each achieve roughly 81% sensitivity, with PCR alone lower at 38%, though combining all three approaches raises sensitivity

to 97% [13]. This ordering reverses in atypical retinochoroiditis, where PCR (55%) outperforms the Goldmann-Witmer coefficient (45%) and immunoblot (53%), particularly in larger acute lesions, with combined testing again improving yield [14]. An earlier series similarly found combined Goldmann-Witmer coefficient and immunoblot testing more sensitive (71%) than either alone [15]. Timing matters: aqueous testing within three weeks of symptom onset confirmed only 73% of episodes on first sampling, with local antibody production positive in just 57%, values that both improved substantially on repeat sampling [16]. These findings collectively argue for repeat or combined aqueous testing when initial results are negative but clinical suspicion remains high. In practice, the choice among Goldmann-Witmer coefficient, immunoblot and PCR should be informed by lesion appearance rather than applied uniformly: for a typical peripheral lesion adjacent to an old scar, serology-based local antibody testing carries most of the diagnostic weight, whereas for an atypical, large or macular lesion without a clear scar, molecular testing assumes greater relative importance. Serum IgG and IgM remain useful to establish exposure and to distinguish likely recent from remote infection, but a positive serum IgG alone confirms only prior exposure, not that the current retinal lesion is toxoplasmic, given the high background seroprevalence in many populations.

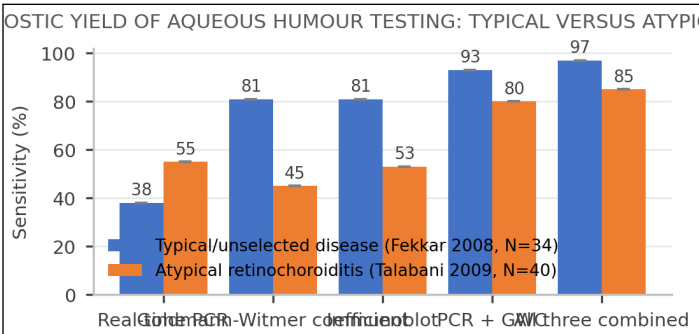


Figure 1: Sensitivity of aqueous humour testing for typical [13] versus atypical [14] ocular toxoplasmosis: PCR outperforms the Goldmann-Witmer coefficient (GWC) in atypical disease, while GWC and immunoblot outperform PCR in typical disease; combined testing improves yield in both settings.

Management, Including Recurrence Prophylaxis

Classic pyrimethamine-sulfadiazine-corticosteroid triple therapy remains widely used, but randomised evidence for its superiority over alternatives is limited. A single-blind trial of 59 patients found trimethoprim-sulfamethoxazole produced retinal lesion reduction (61%) statistically equivalent to pyrimethamine-sulfadiazine (59%), with comparable visual outcomes, adverse effects and 24-month recurrence [17]. A separate randomised comparison of intravitreal clindamycin-dexamethasone against oral therapy in 66 patients likewise found equivalent visual, lesion-size and inflammatory outcomes, with numerically similar recurrence (12.5% versus 14.7%) and fewer systemic adverse effects in the intravitreal arm [18]. A further trial compared pyrimethamine-azithromycin against pyrimethamine-sulfadiazine, though its comparative results were not independently retrievable for this review [19]. A Cochrane systematic review addressed antibiotics versus no treatment for toxoplasma retinochoroiditis, underscoring how limited the controlled evidence base remains even for this basic question [20]. A Colombian cost-effectiveness analysis modelled trimethoprim-sulfamethoxazole as the most cost-effective first-line option among four regimens, at roughly 86% modelled effectiveness and under US\$57 per course, though this effectiveness figure is a model input drawn from prior literature rather than a newly observed cure rate [21]. Global reassessments of disease manifestations and management synthesise this trial landscape alongside broader clinical practice [22]. Reading across these trials, a consistent pattern emerges: every randomised comparison with verifiable results in this literature found the regimens compared to be statistically equivalent on lesion reduction, visual outcome and recurrence, rather than establishing that any one option is superior. This is a materially different evidence base from the confidence with which triple therapy is sometimes presented in textbooks and guidelines, and it leaves genuine room for

regimen choice to be guided by toxicity profile, cost, route of administration and patient preference rather than by demonstrated comparative efficacy.

Table 2: Randomised trial evidence for treatment and prophylaxis in ocular toxoplasmosis [17,18,24,25,23,26].				
Comparison	N	Primary result	Recurrence	Source
TMP-SMX vs pyrimethamine+sulfadiazine	59	Lesion reduction 61% vs 59%, not significant	Equivalent at 24 months	soheilian2005
Intravitreal clindamycin+dexamethasone vs oral therapy	66	Equivalent BCVA, lesion size, inflammation	12.5% vs 14.7%, p=0.54	baharivan2013
TMP-SMX prophylaxis vs placebo, up to 5 years	Not stated	0% vs 26.1% recurrence	Sustained benefit	felix2014
TMP-SMX prophylaxis vs placebo, 6 years	Not stated	1.4% vs 27.5% recurrence	Sustained benefit	felix2020
Intermittent TMP-SMX prophylaxis, 10-year extension	~38	37.3% vs 38.6% recurrence	Benefit largely gone	silveira2002 (via keshavarz2026)

Recurrence prophylaxis with intermittent trimethoprim-sulfamethoxazole reduced recurrences in an early Brazilian trial [23], and later randomised follow-up of a placebo-controlled trial reported markedly lower recurrence on active treatment through several years [24], sustained through six years of follow-up [25]. However, a recent review tabulating this trial evidence alongside extended follow-up of the original intermittent-prophylaxis cohort found that the protective benefit substantially narrows by ten years, when treated and untreated recurrence rates converge to within a few percentage points, against a pooled treatment-limiting adverse-event rate of roughly 8% across prophylaxis studies [26]. This trajectory argues against reflexive indefinite prophylaxis in immunocompetent adults and for individualised, time-limited use, reserved for patients with vision-threatening recurrences, macula-involving disease, or a documented pattern of frequent reactivation, rather than applied as a default after a single episode.

Corticosteroid use alongside antimicrobials is near-universal but supported by no controlled trial isolating its effect; one published case documented clinical deterioration, including retinal necrosis and detachment, following early systemic corticosteroid administration in an immunocompetent patient, prompting the authors to caution against early steroid use before infection control is established [27]. Because corticosteroid was co-administered in both arms of the trimethoprim-sulfamethoxazole trial [17] and in the oral arm of the intravitreal-versus-oral trial [18], neither can isolate the steroid's own contribution to outcome; the field's practice of near-universal adjunctive corticosteroid therefore rests on pathophysiological plausibility and long clinical tradition rather than on controlled comparison, and the published deterioration case is a caution, not proof of harm, that should prompt more deliberate timing of steroid initiation relative to antimicrobial control of the infection.

Outcomes and Prognosis

Visual outcomes depend heavily on lesion location and activity at presentation. In the large Brazilian series, eyes active at presentation had substantially worse baseline acuity than inactive eyes, and macular location, atypical appearance and complications each independently predicted poor outcome; overall reactivation occurred at 0.091 episodes per eye-year, nearly doubling in eyes active at enrolment [6]. The Serbian cohort similarly reported legal blindness in 24% of affected eyes, with central lesion location predicting worse prognosis [5]. Individual published cases illustrate both favourable and poor trajectories: several punctate outer retinal toxoplasmosis cases regained good acuity with treatment, while others progressed despite therapy [12], and the traveller case achieved near-full bilateral recovery with prompt combination therapy [11]. Bilateral involvement, though less common than unilateral disease, carries a disproportionate functional burden when it occurs:

among subjects with bilateral disease and final measurements in the large Brazilian series, a meaningful minority remained bilaterally blind despite treatment [6], reinforcing that visual prognosis should be communicated cautiously even after apparently successful antimicrobial therapy, particularly when the initial lesion is macular, large, or associated with vitreous haemorrhage or retinal detachment as in the corticosteroid-associated case above [27].

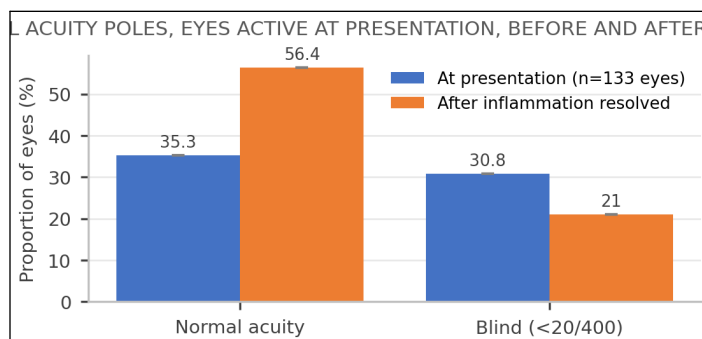


Figure 2: Visual acuity poles among eyes active at presentation (n=133), before and after resolution of inflammation [6]. Intermediate acuity bands were not separately reported after resolution and are omitted rather than interpolated.

Conclusion

Published evidence in immunocompetent adults describes a disease with markedly variable severity, driven by regional strain diversity, lesion location and timing of care. Diagnostic aqueous testing performs best when combined and repeated. Randomised trials to date show equivalence, not superiority, among the principal treatment regimens, and long-term prophylaxis trades an early recurrence-reduction benefit against adverse effects and a benefit that appears to fade over a decade. Corticosteroid timing and prophylaxis duration remain the most consequential unresolved management questions, warranting individualised decisions until better-powered controlled evidence emerges.

References

1. Bowie, W.R., King, A.S., Werker, D.H., Isaac-Renton, J.L., Bell, A., Eng, S.B., et al. (1997) Outbreak of toxoplasmosis associated

- with municipal drinking water. *The Lancet*, 350(9072): 173-177.
2. Burnett, A.J. (1998) Multiple cases of acquired toxoplasmosis retinitis presenting in an outbreak. *Ophthalmology*, 105(6): 1032-1037.
 3. Glasner, P.D., Silveira, C., Kruszon-Moran, D., Martins, M.C., Burnier, M., Silveira, S., et al. (1992) An Unusually High Prevalence of Ocular Toxoplasmosis in Southern Brazil. *American Journal of Ophthalmology*, 114(2): 136-144.
 4. Holland, G.N. (2003) Ocular toxoplasmosis: a global reassessment. Part I: epidemiology and course of disease. *American Journal of Ophthalmology*, 136(6): 973-988.
 5. Kovacevic-Pavicevic, D., Radosavljevic, A., Ilic, A., Kovacevic, I., Djurkovic-Djakovic, O. (2012) Clinical pattern of ocular toxoplasmosis treated in a referral centre in Serbia. *Eye*, 26(5): 723-728.
 6. Arruda, S., Vieira, B.R., Garcia, D.M., Araujo, M., Simoes, M., Moreto, R., et al. (2021) Clinical manifestations and visual outcomes associated with ocular toxoplasmosis in a Brazilian population. *Scientific Reports*, 11(1): 3137.
 7. Bosch-Driessen, L.E.H., Berendschot, T.T.J.M., Ongkosuwito, J.V., Rothova, A. (2002) Ocular toxoplasmosis. *Ophthalmology*, 109(5): 869-878.
 8. Khan, A., Jordan, C., Muccioli, C., Vallochi, A.L., Rizzo, L.V., Belfort, R., et al. (2006) Genetic Divergence of *Toxoplasma gondii* Strains Associated with Ocular Toxoplasmosis, Brazil. *Emerging Infectious Diseases*, 12(6): 942-949.
 9. Gilbert, R.E., Freeman, K., Lago, E.G., Bahia-Oliveira, L.M.G., Tan, H.K., Wallon, M., et al. (2008) Ocular Sequelae of Congenital Toxoplasmosis in Brazil Compared with Europe. *PLoS Neglected Tropical Diseases*, 2(8): e277.
 10. Bhikoo, R., Damato, E.M., Guest, S., Sims, J. (2019) Primary Ocular Toxoplasmosis Presenting to Uveitis Services in a Non-endemic Setting. *Korean Journal of Ophthalmology*, 33(6): 514-519.
 11. Seers, T., Myneni, J., Chaudhry, N.L., Ugarte, M. (2021) Bilateral ocular toxoplasmosis in a returning traveller: age and route of infection as potential risk factors. *BMJ Case Reports*, 14(1): e237068.
 12. Paquentin-Jimenez, R., Rivera-Sempertegui, R., Concha-Del-Rio, L.E. (2025) Small but threatening: Punctate Outer Retinal Toxoplasmosis (PORT), a case series report. *PLoS Neglected Tropical Diseases*, 19(4): e0013013.
 13. Fekkar, A., Bodaghi, B., Touafek, F., Le Hoang, P., Mazier, D., Paris, L. (2008) Comparison of Immunoblotting, Calculation of the Goldmann-Witmer Coefficient, and Real-Time PCR Using Aqueous Humor Samples for Diagnosis of Ocular Toxoplasmosis. *Journal of Clinical Microbiology*, 46(6): 1965-1967.
 14. Talabani, H., Asseraf, M., Yera, H., Delair, E., Ancelle, T., Thulliez, P., et al. (2009) Contributions of Immunoblotting, Real-Time PCR, and the Goldmann-Witmer Coefficient to Diagnosis of Atypical Toxoplasmic Retinochoroiditis. *Journal of Clinical Microbiology*, 47(7): 2131-2135.
 15. Robert-Gangneux, F., Binisti, P., Antonetti, D., Brezin, A., Yera, H., Dupouy-Camet, J. (2004) Usefulness of Immunoblotting and Goldmann-Witmer Coefficient for Biological Diagnosis of Toxoplasmic Retinochoroiditis. *European Journal of Clinical Microbiology and Infectious Diseases*, 23(1): 34-38.
 16. Garweg, J.G., Jacquier, P., Boehnke, M. (2000) Early Aqueous Humor Analysis in Patients with Human Ocular Toxoplasmosis. *Journal of Clinical Microbiology*, 38(3): 996-1001.
 17. Soheilian, M., Sadoughi, M.-M., Ghajarnia, M., Dehghan, M.H., Yazdani, S., Behboudi, H., et al. (2005) Prospective randomized trial of trimethoprim/sulfamethoxazole versus pyrimethamine and sulfadiazine in the treatment of ocular toxoplasmosis. *Ophthalmology*, 112(11): 1876-1882.
 18. Baharivand, N., Mahdavi-fard, A., Fouladi, R.F. (2013) Intravitreal clindamycin plus dexamethasone versus classic oral therapy in toxoplasmic retinochoroiditis: a prospective

- randomized clinical trial. International Ophthalmology, 33(1): 39-46.
19. Bosch-Driessen, L.H., Verbraak, F.D., Suttorp-Schulten, M.S.A., van Ruyven, R.L.J., Klok, A.M., Hoyng, C.B., et al. (2002) A prospective, randomized trial of pyrimethamine and azithromycin vs pyrimethamine and sulfadiazine for the treatment of ocular toxoplasmosis. American Journal of Ophthalmology, 134(1): 34-40.
20. Pradhan, E., Bhandari, S., Gilbert, R.E., Stanford, M. (2016) Antibiotics versus no treatment for toxoplasma retinochoroiditis. Cochrane Database of Systematic Reviews, 2016(5): CD002218.
21. Alvarez-Garcia, V., Rubio-Romero, L., Maldonado, M.A., Gomez-Suarez, M., de-la-Torre, A. (2021) Ocular toxoplasmosis in immunocompetent adults: current cost-effectiveness of four treatment regimens in Colombia. Heliyon, 7(11): e08265.
22. Holland, G.N. (2004) Ocular toxoplasmosis: a global reassessment. Part II: disease manifestations and management. American Journal of Ophthalmology, 137(1): 1-17.
23. Silveira, C., Belfort, R., Muccioli, C., Holland, G.N., Victora, C.G., Horta, B.L., et al. (2002) The effect of long-term intermittent trimethoprim/sulfamethoxazole treatment on recurrences of toxoplasmic retinochoroiditis. American Journal of Ophthalmology, 134(1): 41-46.
24. Fernandes Felix, J.P., Lira, R.P.C., Zacchia, R.S., Toribio, J.M., Nascimento, M.A., Arieta, C.E.L. (2014) Trimethoprim-Sulfamethoxazole Versus Placebo to Reduce the Risk of Recurrences of Toxoplasma gondii Retinochoroiditis: Randomized Controlled Clinical Trial. American Journal of Ophthalmology, 157(4): 762-766.e1.
25. Fernandes Felix, J.P., Lira, R.P.C., Grubenmacher, A.T., Assis Filho, H.L.G., Cosimo, A.B., Nascimento, M.A., et al. (2020) Long-term Results of Trimethoprim-Sulfamethoxazole Versus Placebo to Reduce the Risk of Recurrent Toxoplasma gondii Retinochoroiditis. American Journal of Ophthalmology, 213: 195-202.
26. Keshavarz, T., Furtado, J.M., Smith, J.R. (2026) Antimicrobial Drug Prophylaxis for Recurrent Ocular Toxoplasmosis. Pathogens, 15(4): 388.
27. Lin, H.-Y., Lee, W.-J.A. (2022) The Role of Corticosteroids in Treating Acute Ocular Toxoplasmosis in an Immunocompetent Patient: A Case Report. Frontiers in Medicine, 9: 843050.