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Alternative Treatment of Recurrent Ocular Toxoplasmosis in Pregnant Women – Case Report

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Abstract: **Introduction:** Ocular toxoplasmosis, caused by the protozoan *Toxoplasma gondii*, often presents as an inflammatory lesion involving the choroid and retina. In many countries, it represents the most common cause of posterior uveitis. One of the typical forms is recurrent, unilateral, necrotizing retinitis with secondary choroiditis adjacent to a pigmented retinochoroidal scar, with or without involvement of the vitreous body. Standard treatment involves oral administration of pyrimethamine and sulfadiazine in combination with glucocorticosteroids. Other agents that may be used in treatment include azithromycin, trimethoprim–sulfamethoxazole, clindamycin, and atovaquone. However, the considerable toxicity of these drugs and their systemic combinations necessitates the search for alternative therapeutic approaches. One such option is local treatment in the form of intravitreal injections in cases of ocular reactivation of infection.
Material, methods, and results: The present report outlines an effective diagnostic and therapeutic process in a 28-year-old patient in the second trimester of pregnancy with reactivation of a *Toxoplasma gondii*–related scar located along the papillomacular bundle, with partial involvement of the macula.
Conclusions: Intravitreal injection of 1 mg of clindamycin and 400 μg of dexamethasone is an acceptable and safe alternative to standard treatment. This approach is associated with greater patient convenience and a safer systemic adverse-effect profile. The treatment is effective and, in certain patient groups – including pregnant women – may be the only viable option.

Key words: ocular toxoplasmosis, uveitis, *Toxoplasma gondii*, intravitreal antibiotic therapy.

Introduction

Toxoplasmosis is one of the most widespread parasitic infections and zoonotic diseases worldwide [1, 2]. It is caused by the protozoan *Toxoplasma gondii* – an intracellular parasite that exists in three life forms: oocysts (the resistant form present in the feces of the definitive host), bradyzoites (encysted forms found in the tissues of intermediate hosts), and tachyzoites (the active, proliferating form responsible for the inflammatory response). The definitive host of the parasite is the cat, which excretes oocysts in its feces, while intermediate hosts may include mice, birds, livestock, or humans [1, 3].

Routes of infection include ingestion of food or water contaminated with oocysts, consumption of undercooked meat from intermediate hosts leading to infestation with bradyzoites (tissue cysts), hematogenous transmission from a pregnant woman in the active phase of disease through the placenta to the fetus, as well as organ transplantation or blood transfusion [3].

Ocular toxoplasmosis typically presents as unilateral, recurrent, necrotizing retinitis with secondary choroiditis adjacent to a pigmented scar, accompanied by vitritis and vasculitis. Ocular involvement in congenital infection may remain undiagnosed until the incidental detection of characteristic retinochoroidal scars. More than half of quiescent retinal lesions originate from infection acquired after birth [4, 5]. Age and immunological status influence both the occurrence and the severity of the inflammatory process [6].

Case report

A 28-year-old patient at 18 weeks of pregnancy presented to the Ophthalmology Department due to decreased visual acuity in the right eye (OD) persisting for one week. Her medical history revealed no ocular trauma, surgery, or disease, and no chronic systemic conditions.

On admission, BCVA in the OD was 0.16 (left-sided optotypes), and visual acuity in the left eye (OS) was 1.0 with correction. Intraocular pressure was within normal limits. The anterior segment of both eyes was unremarkable. Fundus examination of the OD revealed an inflammatory focus in the papillomacular bundle, characterized by swollen, bright, elevated retina, optic disc edema, and vitreous exudation (Figs. 1–3). The OS showed no abnormalities.

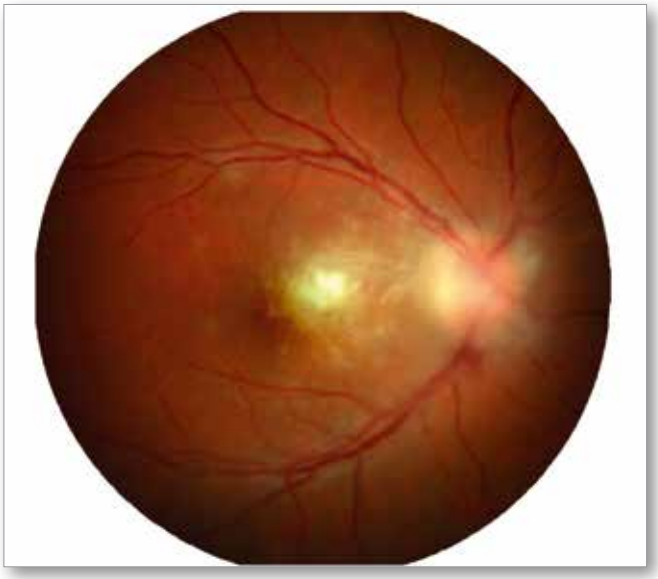

Fig. 1. Fundus photograph on admission.

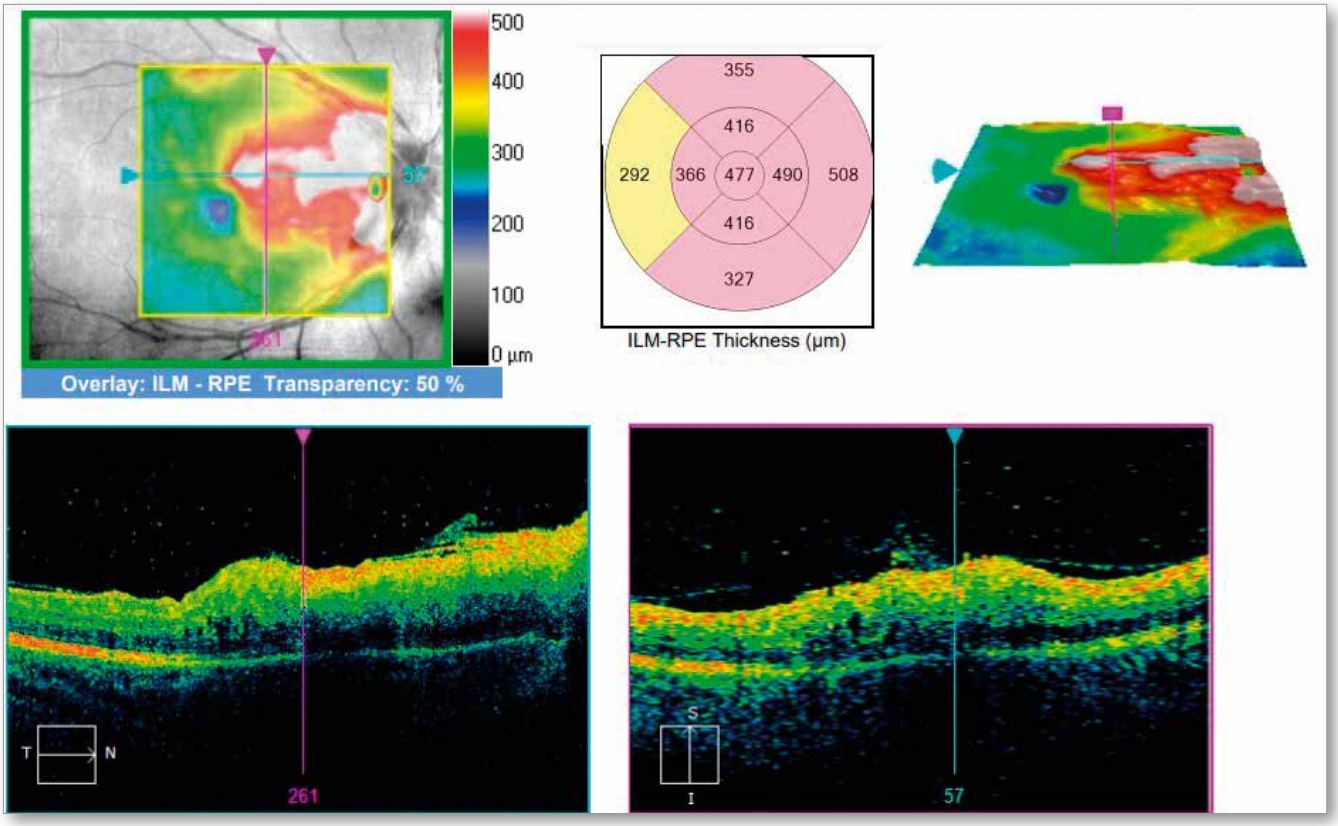

Fig. 2. Macular OCT – thickening and blurring of the inner retinal layers, with vitreous inflammatory reaction.

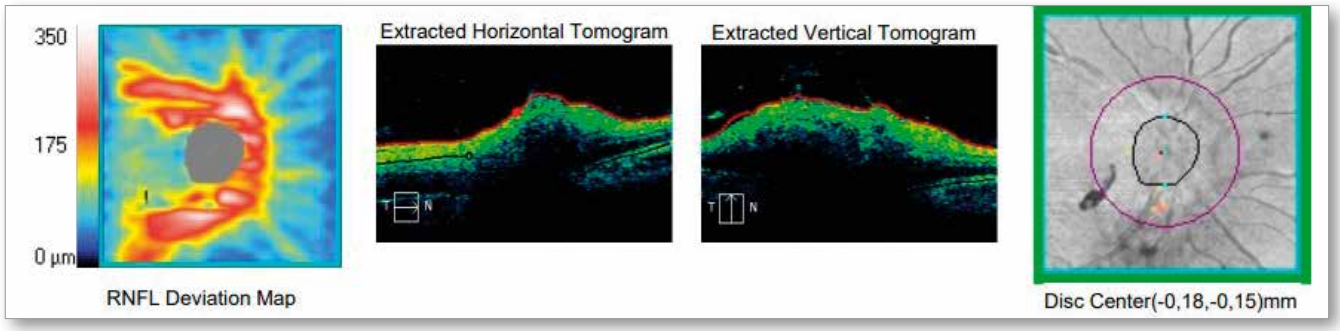

Fig. 3. OCT of the optic disc – edema.

Laboratory tests revealed no abnormalities in complete blood count, ESR, or C-reactive protein. *Toxoplasma gondii* IgG antibodies were present at 20.8 IU/mL with an avidity of 83%, while IgM antibodies were absent (index 0.07).

Based on the clinical presentation and immunological tests, retinitis and optic nerve inflammation due to reactivation of a pre-existing *Toxoplasma gondii* scar were diagnosed.

Considering the patient's pregnancy, the potential adverse effects of orally administered drugs, the limited availability of targeted systemic therapy, and the efficacy of intravitreal treatment in infection reactivation (comparable to triple therapy), the decision was made to initiate local treatment.

In the operating room, under direct visualization with a surgical microscope, an intravitreal injection of 1 mg clindamycin (0.1 mL) and 400 μg dexamethasone (0.1 mL) was administered using a single puncture. Directly after the procedure, light perception was assessed. Initial improvement in both reported symptoms and objective parameters could be observed as early as the third day after the procedure. Complete resolution of the inflammatory condition occurred three weeks after the initiation of therapy (Figs. 4, 5).

One and a half months postpartum (seven months after the first episode of reactivated inflammation), the inflammatory process in the scar reappeared. BCVA in the right eye was 0.16, with the presence of floaters in the visual field.

Given the therapeutic success achieved with the initial intravitreal injection, the same treatment approach was adopted as in the first episode.

Two injections of 1 mg clindamycin and 400 μg dexamethasone, spaced three weeks apart, were required to resolve the inflammation and stabilize the local condition (Fig. 6).

At present, after three years of follow-up, no recurrence of inflammation has been noted. No adverse effects from the therapy were noted.

Discussion

Ocular toxoplasmosis accounts for as many as 20–40% of all cases of posterior uveitis. Clinical manifestations include reduced visual acuity, floaters, and photophobia. On ocular examination, findings may include diffuse anterior uveitis, a fluffy focus of retinochoroiditis adjacent to a pigmented scar, vitritis of varying severity, classic "headlight in the fog" sign, vasculitis, and optic disc oedema [7–10].

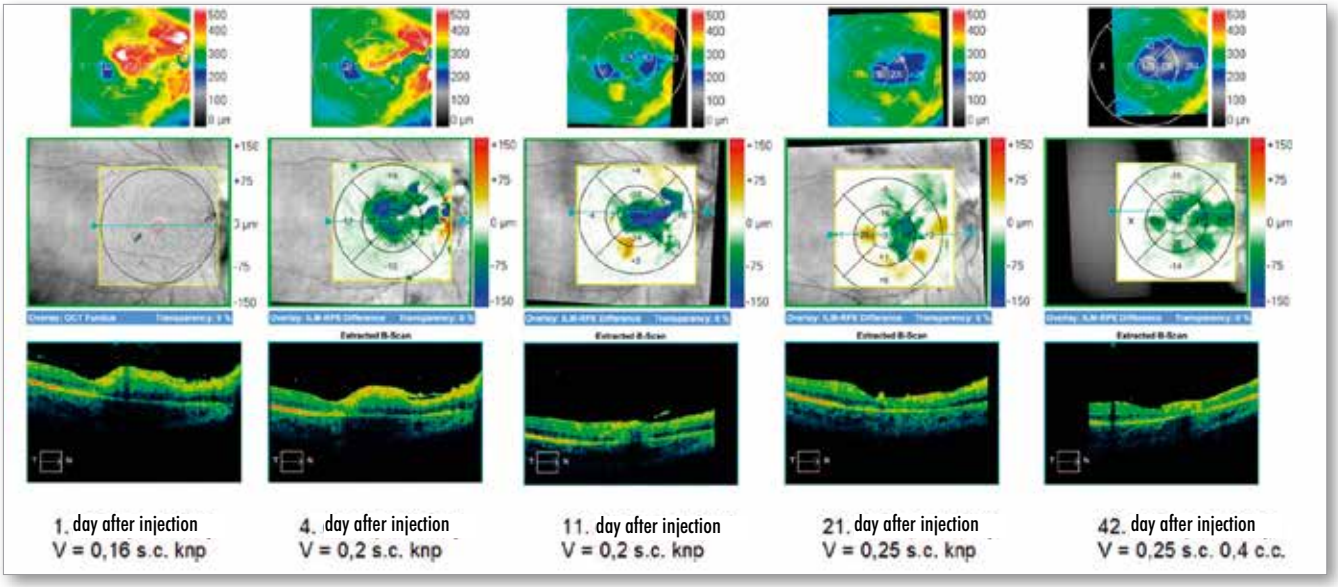

Fig. 4. Change in macular morphology during the healing process after intravitreal injection.

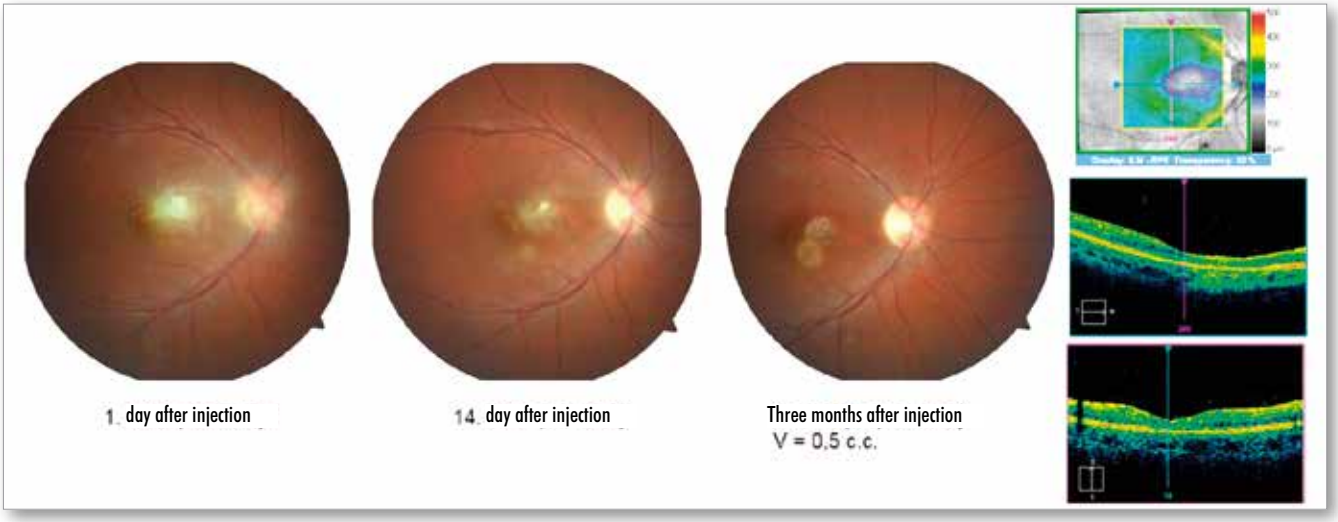

Fig. 5. Change in fundus appearance during the healing process after intravitreal injection.

Pregnancy may trigger reactivation of the infection in the mother, and the disease may be resistant to treatment [11]. Unlike primary infection, recurrent toxoplasmic retinochoroiditis during pregnancy poses minimal risk to the fetus. In the majority of cases, the infection resolves spontaneously [12].

Absolute indications for treatment include disease course threatening vision, with involvement of the macula, the papillomacular bundle, the optic disc, or major vessels, as well as cases of severe vitritis and in immunocompromised individuals [1, 13, 14].

Standard treatment involves oral administration of pyrimethamine and sulfadiazine in combination with glucocorticosteroids. Other agents that may be used in treatment include azithromycin, trimethoprim–sulfamethoxazole, clindamycin, and atovaquone [15].

The considerable systemic toxicity of oral drug combinations has prompted interest in alternative treatment methods. Administration of pyrimethamine requires weekly blood count monitoring and concurrent supplementation of folinic acid to prevent leukopenia and thrombocytopenia. Sulfadiazine may cause severe allergic reactions, which in some cases can be life-threatening, such as Stevens–Johnson syndrome or toxic epidermal necrolysis. Trimethoprim–sulfamethoxazole may lead to hepatic and renal injury and also exhibits teratogenic effects [1].

One of the alternative therapeutic approaches is local intravitreal treatment. Administration of 1 mg clindamycin along with 400 μg dexamethasone has been shown to be as effective as conventional triple therapy in cases of infection reactivation [16]. Clindamycin monotherapy may also be effective [17–19].

By bypassing the blood–retina barrier, this approach ensures high local drug concentrations within the vitreous body. The therapy is safer in terms of systemic adverse-effect profile and, moreover, is associated with greater patient convenience, fewer follow-up visits and hematological tests required [1]. It enables a faster local therapeutic response, which is particularly relevant in cases posing a threat to the macula [20].

It should be emphasized, however, that local therapy alone is insufficient in immunosuppressed patients, in pregnant women with primary infection, and in congenital toxoplasmosis [21].

Conclusions

Intravitreal injection of 1 mg clindamycin combined with 400 μg dexamethasone represents an effective and safe alternative to conventional therapy. In ocular reactivation of infection, this approach achieves high intraocular concentrations of therapeutic agents in the vitreous and retina while minimizing systemic adverse effects. Such local therapy offers a clear advantage over oral

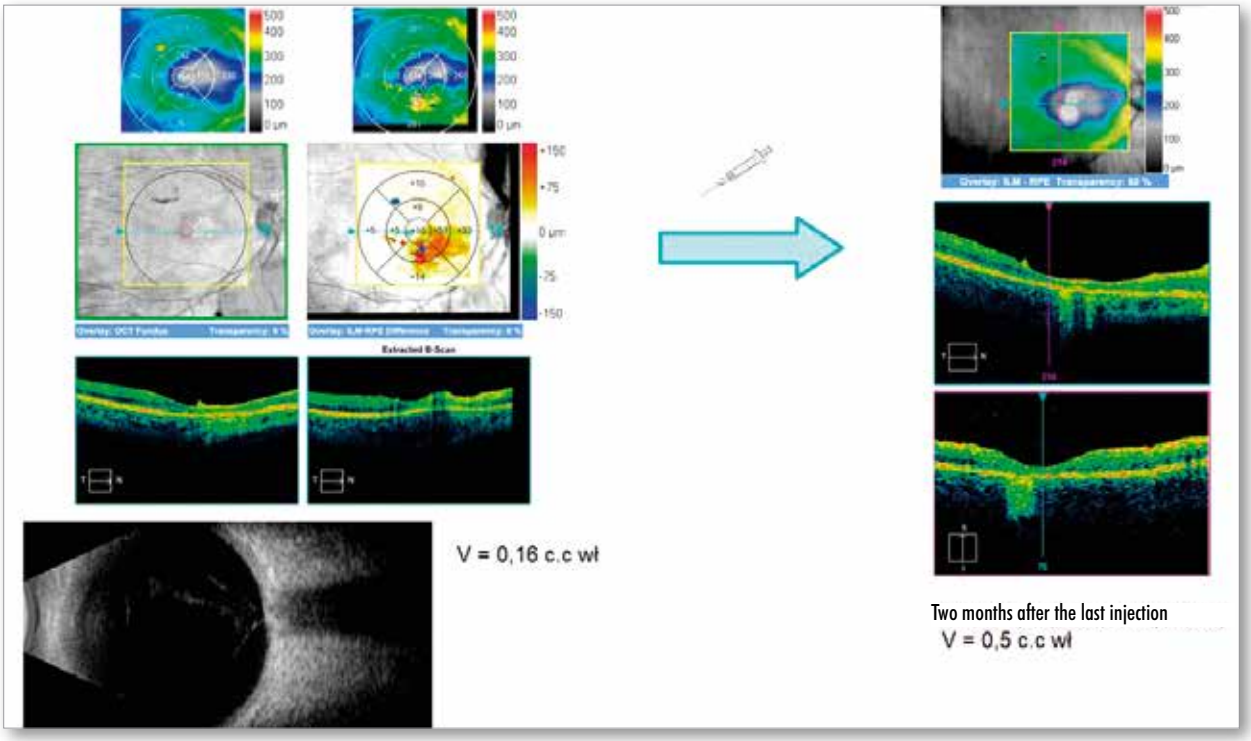

Fig. 6. Change in macular morphology during the healing process after intravitreal injection.

treatment and is particularly valuable for selected patient groups, including pregnant women. Management of recurrent disease during pregnancy should be individualized, and therapy undertaken only when absolute indications are present.

Disclosure
Conflict of interests: none declared
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