CASE REPORT

Active toxoplasma chorioretinitis in immunocompromised patients: a case series

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ABSTRACT

Toxoplasma chorioretinitis (TC) can exhibit atypical features in immunocompromised patients including bilaterality, extensive spread, multifocal presentation, large areas of retinal necrosis without adjacent retinal scarring, and diffuse necrotizing retinitis resembling the viral retinitis that may cause confusion in the differential diagnosis. The aim of this study was to present the clinical features of four eyes of three immunocompromised patients with active toxoplasma chorioretinitis. Two of the patients were female and one, male. Two patients had hematological malignancies and the remaining patient was under adalimumab treatment for ankylosing spondylitis. Visual complaints began 10 days to four months prior to TC diagnosis. All four eyes had mild-to-moderate anterior chamber cells together with severe vitritis on slit-lamp examination while there were solitary chorioretinitis lesions on fundoscopy. Despite all patients were negative for anti-toxoplasma immunoglobulin M, all were positive for immunoglobulin G. All three patients were successfully treated with a combined treatment of systemic and intravitreal anti-toxoplasmic drugs. Clinicians should be cautious for the possible toxoplasma chorioretinitis besides the other infectious entities when a new uveitis episode is detected in an immunosuppressed patient in order to avoid misdiagnosis and thereby wrong treatment.

KEYWORDS: Anti-toxoplasmic therapy; immunosuppression; ocular toxoplasmosis; toxoplasma chorioretinitis; uveitis

INTRODUCTION

In immunocompetent individuals, initially, *Toxoplasma gondii (T. Gondii)* often causes a subclinical infection and is effectively controlled by the host's immune system ending up with a chronic latent infection [1,2]. *Toxoplasma gondii* remains dormant in the retina of an individual with a healthy immune system, but it has the potential to reactivate and induce granulomatous inflammation [3].

The diagnosis of toxoplasma chorioretinitis (TC) is established by the presence of focal or paucifocal necrotizing retinitis along with evidence of infection with *T. Gondii* (positive polymerase chain reaction [PCR] for *T. Gondii* from an intraocular specimen; or positive serum immunoglobulin [Ig] M antibodies against *T. Gondii*) or the presence of characteristic clinical ocular features (hyperpigmented and/or atrophic chorioretinal scars and round/oval retinitis lesions or recurrent acute/episodic course) according to the Standardization in Uveitis Nomenclature (SUN) criteria [4].

In this case series, we presented the clinical features of TC in four eyes of three immunocompromised patients who were successfully treated with a combination of systemic and intravitreal therapies.

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MATERIALS AND METHODS

Medical records and imaging data of three immuno compromised patients who were diagnosed to have TC were retrospectively reviewed between January 2022 and November 2023.

Each patient underwent a complete ophthalmic evaluation. Best-corrected visual acuity (BCVA) was assessed using the Snellen chart, and intraocular pressure (IOP) was measured with Goldmann applanation tonometry. Crystalline lens status was determined according to the Emery–Little classification [5]. Vitritis was graded according to the Miami scoring system [6]. Color fundus images were captured using VISCUAM 500 (Carl Zeiss Meditec, Jena, Germany) or DRI OCT Triton (Triton, TOPCON Inc, Tokyo, Japan), and the spectral-domain optical coherence tomography (OCT) images were acquired using Heidelberg Spectralis (Heidelberg Engineering, Heidelberg, Germany) whenever deemed necessary. All clinical descriptions adhered to the SUN criteria [7].

Adjunctive intravitreal clindamycin, ganciclovir, and/or dexamethasone injections were given under topical anesthesia in the operating room for at least three minutes following the 5% povidone-iodine instillation whenever deemed necessary. The injections were administered through the superotemporal quadrant, four mm posterior to the limbus as all the patients were phakic. Following the procedure, a topical antibiotic was prescribed four times a day for a week.

CASE PRESENTATIONS

Case 1

A 63-year-old man was referred to our department with the floaters and discomfort of three months' duration in his left eye. Eight years ago, the patient was diagnosed with myelodysplastic syndrome and five months ago with acute myeloid leukemia. He received seven treatment cycles of decitabine and 29 treatment cycles of azacitidine.

On ophthalmological examination, his BCVA was 8/10 in his right eye and 6/10 in his left eye. Intraocular pressures were 9 mmHg in both eyes. Biomicroscopically there was +2 nuclear sclerosis in the right eye and +2 anterior chamber cells with +2 nuclear sclerosis in the left eye. While the right fundus appeared normal, there was a grade 6 vitritis together with a yellowish white two disc-diameter sized chorioretinal lesion at the inferior temporal fundus with fuzzy borders (Figure 1A). Transfoveal spectral-domain OCT section depicted a normal foveal contour together with the vitritis related hyperreflective posterior vitreous dots corresponding to the inflammatory cells. Thickened posterior hyaloid, epiretinal membrane (ERM) formation, disorganized retinal layers, and focally thickened choroid were noted on the spectral-domain OCT section passing through the chorioretinitis lesion (Figure 1B). A full infectious work-up was carried out. Serological tests revealed a positive result for Toxoplasma IgG (IgG, 68.4 UI/mL), while IgM was negative. A diagnosis of left TC was established, and systemic treatment (peroral trimethoprim + sulfamethoxazole [TMP-SMZ] 160/800 mg twice a day for 12 weeks and azithromycin 500 mg once daily for two weeks) and topical treatment (prednisolone acetate 1% twelve times daily, tropicamide 0.5% three times daily) were initiated.

Topical steroid treatment was gradually tapered. Intraocular pressure did not elevate during the treatment. Three months later, fundus examination revealed a marked reduction in vitritis, and the area of chorioretinitis was healed with

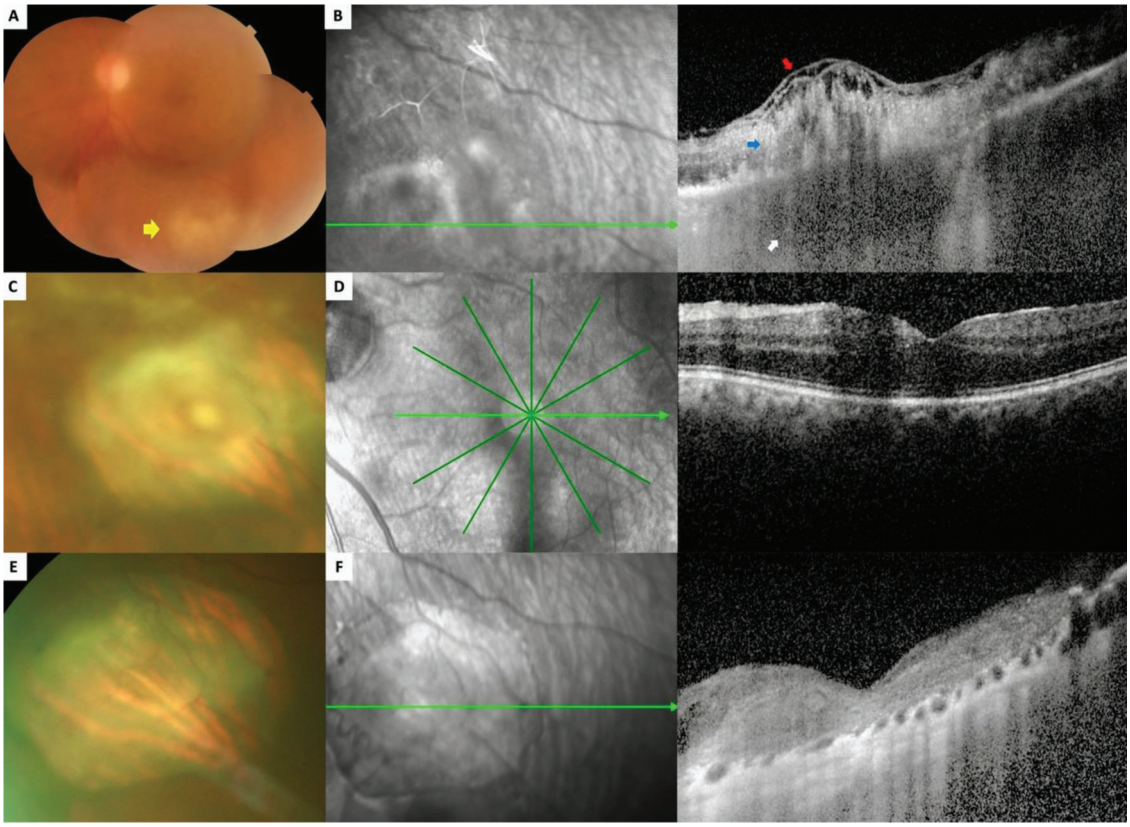

Fig. 1. Case 1, Left eye. At admission; Color fundus picture (A) revealing a yellowish white chorioretinitis lesion with fluffy borders, measuring approximately two optic disc diameters inferior to the lower temporal vascular arcade (yellow arrow), along with a severe vitritis. Spectral-domain optical coherence tomography (SD-OCT) section passing through the chorioretinitis area (B) depicting an epiretinal membrane formation, disorganized retinal layers (blue arrow), thickened posterior hyaloid (red arrow) and focally thickened choroid (white arrow). Five months later, the appearance of recurrent chorioretinitis and severe vitritis on color fundus picture (C) and macular SD-OCT revealing the normal foveal contour together with the signs of vitritis and mild epiretinal membrane formation (D). Ten months after the diagnosis; color fundus picture demonstrating the healed chorioretinitis scar at the inferior fundus with no signs of vitritis (E). Spectral-domain OCT section transversing the healed chorioretinitis area delineating the scarring formation and chorioretinal atrophy (F).

a scar. The patient was recommended to keep on prophylactic TMP-SMZ 160/800 mg once a day three days a week, and topical loteprednol etabonate 0.5% once daily for a month.

Five months after the diagnosis, the patient re-complained of floaters in his left eye. Dilated fundoscopy of the left eye revealed that vitreous was very hazy and there was an active edge adjacent to the previous chorioretinitis lesion at the inferior fundus (Figure 1C). Macular spectral-domain OCT revealed a normal foveal contour, mild ERM, and hyperreflective posterior vitreous dots (Figure 1D). Due to recurrence, peroral TMP-SMZ 160/800 mg twice a day was administered again for six weeks, and in addition, intravitreal 1 mg/0.1 ml clindamycin injection was also given. Vitritis was regressed following the treatment and he was advised to take TMP-SMZ 160/800 mg once a day for three more weeks.

At the last visit, 10 months after the diagnosis, his BCVA was 7/10 in the left eye. The anterior segment and IOP were normal. There was no sign of vitritis and the chorioretinitis area looked scarred in the left eye (Figure 1E). Spectral-domain OCT section passing through the lesion depicted the thinned and scarred choroid and retina (Figure 1F).

Case 2

A 57-year-old woman was consulted to us with bilateral gradual painless visual deterioration of 10 days' duration. She was diagnosed to have acute myeloid leukemia a year ago and underwent an allogenic bone marrow transplant five months ago. She received seven courses of systemic cytarabine, three courses of daunorubicin, and three courses of azacitidine.

On examination, BCVA was 1/10 in both eyes. Slit-lamp examination revealed bilateral clear cornea, +2 anterior chamber cells, and +2 nuclear sclerosis. IOP was 9 mmHg in OD and 10 mmHg in OS. Approximately two optic disc diameter-sized yellowish white bilateral asymmetrical chorioretinitis areas with blurry borders were observed on dilated fundus examination at the upper temporal fundus with a grade 4 vitritis (Figure 2A, B). Optical coherence tomography depicted a normal foveal contour and vitritis-related hyperreflective particles at the posterior vitreous cortex. Unfortunately, no OCT section passing through the lesions could be obtained. A full infectious work-up was carried out. Serological tests demonstrated a positive anti-Toxoplasma IgG at a level of 63.9 IU/mL, whereas IgM tested as negative. Anti-cytomegalovirus (anti-CMV) IgM and IgG titers were positive at a level of 1.95 UI/ml and 217 UI/ml, respectively. As the clinical findings might be related to TC or CMV retinitis; bilateral humor aqueous and vitreous samples were acquired, and bilateral intravitreal 1 mg/0.1 ml clindamycin and 2 mg/0.1 ml ganciclovir injections were performed. However, viral PCR test was negative together with the negative bacterial and fungal cultures. Unfortunately, PCR testing for *T. gondii* could not be done as this test was not performed at our hospital. In light of these findings, peroral azithromycin 500 mg once daily for two weeks and TMP-SMZ 160/800 mg twice daily for three weeks were commenced together with the topical loteprednol etabonate % 0.5 three times daily.

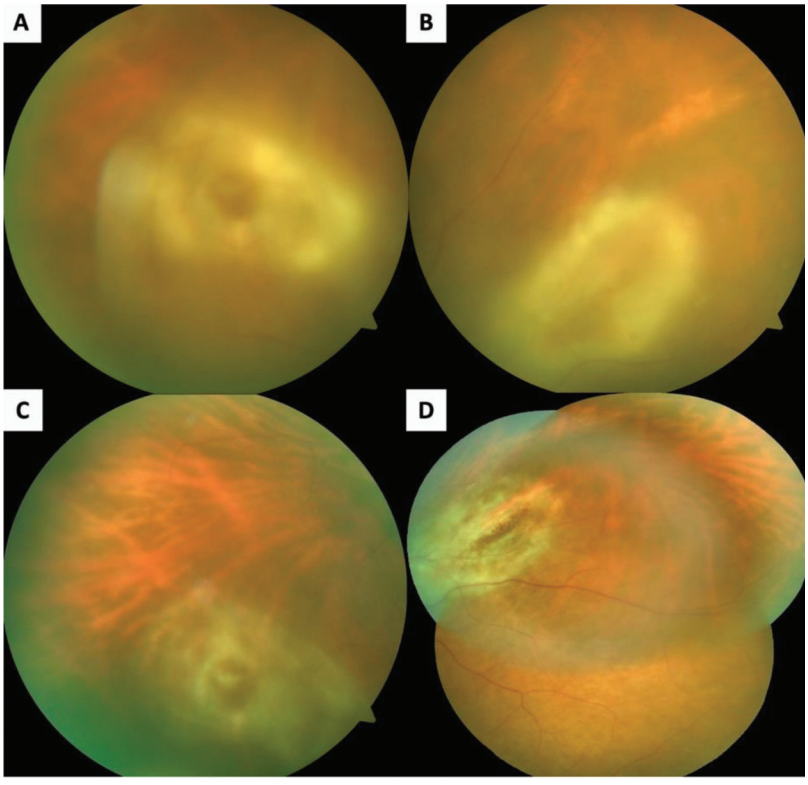

Fig. 2. Case 2. At admission; approximately two optic disc diameters-wide yellowish white chorioretinitis areas with blurry borders on color fundus pictures of the right (A) and the left (B) eyes close to the upper temporal vascular arcade in association with a severe vitritis. Five months after the diagnosis, color fundus pictures showing the chorioretinal scars with sharp borders (C, right eye; D, left eye).

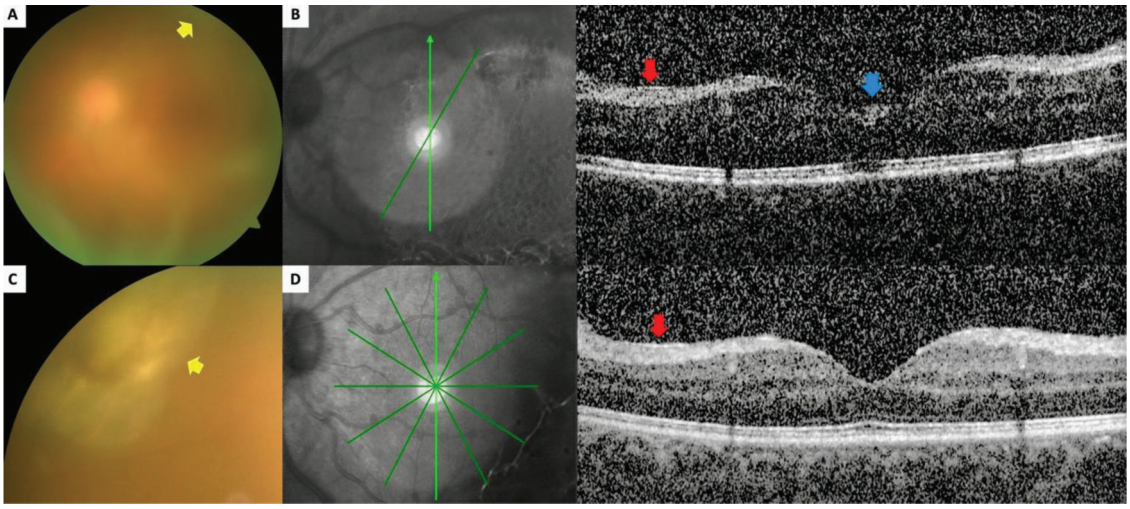

Fig. 3. Case 3, Left eye. At admission, color fundus picture (A) depicting the severe vitritis along with a yellow focal chorioretinitis area, measuring approximately two optic disc diameters, located just superotemporal to the upper temporal vascular arcade. Macular spectral-domain optical coherence tomography (SD-OCT) (B) showing the formation of epiretinal membrane (ERM) (red arrow) with normal foveal contour, a hyperreflective aggregated particle at the posterior vitreous area (blue arrow) and severe vitritis. Five months after the diagnosis; vitritis was regressed, chorioretinitis size was diminished and the borders of the chorioretinitis area had sharpened on the color fundus picture (yellow arrow) (C); while macular SD-OCT depicting the normal foveal contour and a mild formation of ERM (red arrow) (D).

As the patient could not continue oral TMP-SMZ due to its gastrointestinal side effects, two more bilateral intravitreal injections of 1 mg/0.1 ml clindamycin were administered one week and one month after the diagnosis. Topical steroid treatment was gradually tapered and stopped.

At the last visit, five months after the diagnosis, the patient's BCVA was 1/10 in OD and 2/10 in OS. Slit-lamp examination was unremarkable except for +2 nuclear sclerosis and the IOP was 14 mmHg in both eyes. There were bilateral chorioretinal scars with well-demarcated borders with markedly diminished vitreal haze (Figure 2C, D).

Case 3

A 52-year-old woman was presented with a left gradual painless visual decrease of four months' duration. Fourteen years ago, she received the diagnosis of ankylosing spondylitis. She was on peroral 500 mg/day of salazopyrin, 4 mg/day of prednisolone, and subcutaneous 40 mg/two weekly adalimumab treatment.

Her BCVA was 10/10 in the right eye, and hand motions in the left eye. Biomicroscopically while right anterior segment was normal slit-lamp examination revealed granulomatous keratic precipitates and +3 anterior chamber cells in the left eye. Intraocular pressure was 14 mmHg in OD and 15 mmHg in OS. The right fundus was normal whereas there was a yellow focal chorioretinitis area of two optic disc diameters size at the superior temporal fundus together with a grade 7 vitritis in the left eye (Figure 3A). Macular spectral-domain OCT depicted a vitreomacular adhesion with a normal foveal contour in OD, while there was ERM, hyperreflective aggregated particles at the posterior vitreous cortex related to severe vitritis in OS (Figure 3B). Unfortunately, an OCT section transversing the chorioretinitis area could not be obtained due to vitritis and the lesion location. A full infectious work-up was carried out. The serological tests revealed anti-Toxoplasma IgG positivity (40.9 UI/mL). Anti-Toxoplasma IgM was negative. The diagnosis of a left TC was established. She was put on peroral TMP-SMZ 180/600 mg twice daily and azithromycin 500 mg once daily together with the prednisolone acetate 1% drops twelve times daily, and tropicamide 0.5% drops three times daily. Adalimumab treatment was stopped due to active TC upon consultation with the patient's rheumatologist.

The patient was treated with peroral TMP-SMZ 160/800 mg for five months and azithromycin 500 mg for two months daily and received two left intravitreal injections of 1 mg/0.1 ml clindamycin + 0.4 mg/0.1 ml dexamethasone during the five-month follow-up due to longstanding vitritis. We attempted to obtain a vitreous sample for gram staining, cultures, and PCR testing with a 27-gauge needle at the time of the first intravitreal injection, but we could not obtain enough material due to density of the vitreous. At the last visit, five months after the diagnosis; left BCVA was 4/10, slit-lamp examination was unremarkable, and IOP was 13 mmHg. The vitreous haze lessened, and the lesion appeared atrophic with sharp borders (Figure 3C). Macular spectral-domain OCT depicted normal foveal contour in OS (Figure 3D).

Additional immunosuppressive therapy is going to be discussed with her rheumatologist if a systemic immunosuppressive therapy deemed requisite in the future.

DISCUSSION

The diagnosis of TC primarily depends on the visualization of characteristic lesions upon dilated fundoscopy, notably the chorioretinitis. However, the diagnosis can be challenging in immunocompromised patients; as TC can exhibit atypical features such as bilaterality, extensive spread, multifocal presentation, large areas of retinal necrosis without adjacent retinal scarring, neuroretinitis, punctate outer retinitis, and multifocal diffuse necrotizing retinitis resembling the viral retinitis [8-10]. Polymerase chain reaction testing of ocular fluids serves as a rapid and reliable tool if available, particularly valuable for the prompt diagnosis of

TC, especially in patients presenting with atypical clinical features [11].

Several authors described immunocompromised cases with TC and the publications that included at least two cases of ocular toxoplasmosis in immunocompromised patients are summarized in Table-1 [12-15].

Biancardi et al. reported a 38-year-old woman with Crohn's disease and TC with a positive serology [1]. There were multiple necrotizing lesions of yellow-white retinitis with indistinct borders involving the nasal and superior retina together with a severe vitritis in her left eye. She was already on subcutaneous adalimumab (40 mg every two weeks) and oral azathioprine (2.5 mg/kg/day). The authors preferred oral TMP-SMZ due to its convenient posology with low side effects.

Garg et al. reported a 14-year-old girl with a history of acute lymphoid leukemia and two allogeneic bone marrow transplants who experienced blurry vision in her right eye during the disease course [16]. There was a small inferotemporal diffuse granular white deep retinal lesion and a superonasal retinal white lesion along the vascular arcade that was thought to be CMV retinitis. However, the retinal lesions had progressed despite the systemic and intravitreal ganciclovir treatment. Subsequently, anterior chamber fluid was tested for *T. gondii* PCR that yielded a positive result. Additionally, the patient's previous anti-Toxoplasma IgG serology was positive. She was treated with a single intravitreal 1 mg/0.1 ml clindamycin injection and a 14-week course of peroral sulfadiazine 3 gr/day.

Differentiating between TC and CMV retinitis can be challenging in immunocompromised patients. Moreover, co-infection of *T. gondii* and CMV may also occur in this group of patients. Galvan Ramirez et al. described a 7-year-old boy who underwent liver transplantation due to end-stage cirrhosis secondary to type IV glycogenosis [17]. The patient was presented with icterus, high transaminases levels, and poor general health status 11 months after the surgery together with two white retinal lesions and multiple scars in his left eye, suggestive of CMV retinitis and TC. He was tested positive for both anti-CMV IgM and IgG, as well as anti-Toxoplasma IgM and IgG. His treatment regimen included pyrimethamine, sulfadiazine, folinic acid, prednisolone, and ganciclovir that was followed by a prophylactic TMP-SMZ therapy. Despite the resolution of active chorioretinal lesions with residual chorioretinal scars, new bilateral lesions suggestive of CMV retinitis developed 12 months later. Then, the patient was re-treated successfully with ganciclovir by the authors. In our Case 2, we obtained intraocular fluid samples for viral PCR analysis and employed intravitreal clindamycin and ganciclovir at the same setting as the clinical presentation could be related to either *T. gondii* or viruses or could be even a co-infection at the admission. As the viral PCR panel turned out negative, then we carried out the treatment only for toxoplasmosis.

Walkden et al. described an 86-year-old woman with rheumatoid arthritis using 40 mg/two weeks adalimumab who experienced reduced vision in her right eye [18]. Mild non-granulomatous anterior uveitis, significant vitritis, and a large area of necrotizing retinitis at the superotemporal retina were detected. Presence of *T. gondii* was proven with the aqueous humor PCR test. Anti-Toxoplasma IgM serology was also positive. The authors treated the patient with a four-week course of TMP-SMZ (160/800 mg twice daily) and clindamycin (300 mg four times daily) together with rapidly tapered oral prednisone. They emphasized the importance of prompt intraocular sampling and PCR analysis in patients with immunosuppressive states, as the clinical findings of TC might be associated with atypical presentations. We were only able to perform aqueous humor PCR analysis in Case 2 to rule out viral etiology, as the PCR testing for *T. gondii* was not available at our hospital.

Besides systemic immunosuppression, local immunosuppressive status could also facilitate TC occurrence. Crosson et al. reported three patients with unilateral TC mimicking severe acute retinal necrosis who had previously received local corticosteroid treatment prior to the diagnosis [19]. Two patients received subtenon triamcinolone acetonide injection and one intravitreal triamcinolone acetonide injection. The authors concluded that local corticosteroid injection might trigger or exacerbate TC, leading to a fulminant type of retinal necrosis and thereby severe vision loss. Oray et al. reviewed the records of nine patients who experienced fulminant TC due to misdiagnosis related to systemic or intravitreal corticosteroid (CS) therapy [20]. Four patients were treated solely with systemic CS therapy; three, received a combination of systemic and intravitreal CS therapy; one, underwent a subtenon CS injection; and the remaining patient was treated with intravitreal triamcinolone and dexamethasone injections administered two weeks apart. Systemic CS treatment was tapered and/or stopped and the patients received oral anti-toxoplasmic drugs (pyrimethamine, clindamycin, azithromycin, TMP-SMZ) in various combinations. Intravitreal injection of clindamycin was performed in three patients; at the time of pars plana vitrectomy in two, and after two weeks of systemic therapy in one patient. Pars plana vitrectomy was performed in two patients to remove residual depot CS immediately after the diagnosis. The authors concluded that initial misdiagnosis and mistreatment led to an immunosuppressive local status, ending up in an aggressive disease course and serious complications.

The systemic treatment options for TC include sulfadiazine, pyrimethamine with folinic acid, clindamycin, minocycline, TMP-SMZ, and azithromycin. However, systemic use of all these drugs possesses mild-to-moderate side effects. It is reported that 26% of immunocompetent and 40% of immunocompromised patients discontinued the abovementioned systemic treatment due to their side effects [21,22]. Intravitreal injection of clindamycin is as a reliable and relatively safe alternative for the treatment of TC. Theoretically, intravitreal route provides a higher drug concentration in the vitreous and can be potentially advantageous, especially in refractory cases [23]. However, clinicians should not forget that intravitreal clindamycin injection can be fraught with macular infarction [24]. We preferred to employ a combination of systemic and intravitreal therapy in Case 1 and Case 3, as the clinical picture did not improve enough despite the ongoing systemic antimicrobial treatment. We also utilized the combination approach in Case 2 as the patient experienced side effects and couldn't tolerate peroral TMP-SMZ.

CONCLUSION

Infectious uveitis in immunocompromised patients can be a serious challenge for the clinicians as the differential diagnosis can be extremely troublesome. Our case series

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Table 1. Studies that included at least two cases of ocular toxoplasmosis in immunocompromised patients.

First Author (Reference Number)	Cases	Age / Sex	Laterality	Cause of Immunosuppression	Systemic Treatment	Complications
Lassoued et al. (12)	Case 1	43 / M	LE	Anti-TNF treatment for RA	Pyrimethamine, sulfadiazine, and folinic acid for 6 weeks	None
	Case 2	40 / F	LE	Anti-TNF treatment for RA	Pyrimethamine, sulfadiazine, and folinic acid	None
Holland et al. (13)	Case 1	38 / M	RE	AIDS	Oral pyrimethamine (50 mg daily), oral sulfadiazine (2.5 g twice daily), and intravenous dexamethasone (10 mg every six hours)	Rhegmatogenous retinal detachment Left
	Case 2	44 / F	BE	AIDS	Oral sulfadiazine (1 g four times daily) and clindamycin (300 mg four times daily) and oral pyrimethamine (25 mg daily)	rhegmatogenous retinal detachment
	Case 3	28 / M	BE	AIDS	Diagnosed with autopsy	None
	Case 4	46 / M	BE	AIDS	Intravenous dexamethasone (6 mg every six hours for seven days) and oral pyrimethamine (25 mg twice daily) and tetracycline (250 mg four times daily), and folinic acid (5 mg daily)	None
	Case 5	29 / M	RE	AIDS	Spiramycin (1 g four times daily)	None
	Case 6	42 / M	LE	AIDS	Oral pyrimethamine (50 mg daily) and sulfadiazine (2.5 g daily) for seven weeks	None
	Case 7	23 / M	BE	AIDS	Oral pyrimethamine (25 mg twice daily), sulfadiazine (1 g four times daily), and folinic acid (5 mg every other day) for one month and then oral tetracycline therapy (250 mg four times daily) for five months due to reactivation	A large retinal tear in the LE
	Case 8	45 / M	LE	AIDS	Oral pyrimethamine (25 mg daily) and sulfadiazine (2 g four times daily) for three months	None
Elkins et al. (14)	Case 1	44 / F	RE	Systemic steroid treatment for SLE	Oral pyrimethamine (25 mg once weekly) and sulfadiazine (500 mg 4 times daily). The sulfadiazine was later switched to clindamycin (750 mg 4 times daily) because of side effects. Six weeks after the pyrimethamine was discontinued; c1indamycin (150 mg 4 times daily) was maintained for seven months.	None
	Case 2	36 / M	RE	Chronic myelogenous leukemia, graft-versus-host disease	Oral trimethoprim (160 mg) /sulfamethoxazole (800 mg) twice daily for three weeks	None
	Case 3	38 / M	LE	Chronic myelogenous leukemia, graft-versus-host disease	Oral pyrimethamine (100 mg daily) and oral clindamycin (400 mg 3 times daily) for 8 weeks	None
	Case 4	44 / M	RE	AIDS	Oral pyrimethamine (50 mg daily after a 200-mg loading dose) and oral sulfadiazine (4 g daily). The sulfadiazine was replaced with oral c1indamycin (600 mg three times daily) because of intolerance and oral tetracycline (500 mg 3 times daily)	None
	Case 5	35 / M	RE	AIDS	Oral pyrimethamine 50 mg daily for 6 weeks and then oral pyrimethamine 25 mg daily	None
Conrath et al. (15)	Case 1	53 / M	BE	Cardiac transplantation	Oral pyrimethamine and sulfadiazine	None
	Case 2	25 / F	LE	Cardiac transplantation	Pyrimethamine and sulfadiazine for 6 weeks.	None
	Case 3	41 / M	LE	Cardiac transplantation	Pyrimethamine and sulfadiazine were administered full dose for 1 week, then for 4 weeks at half dose associated with alkaline hydration by mouth because of mild renal failure	Epiretinal membrane

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Table 1. Continued.

First Author (Reference Number)	Cases	Age / Sex	Laterality	Cause of Immunosuppression	Systemic Treatment	Complications
	Case 4	55 / M	RE	Cardiac transplantation	Pyrimethamine and sulfadiazine for 6 weeks.	None
Kayabaşı et al. (Present case series)	Case 1	63 / M	LE	Myelodysplastic syndrome, acute myeloid leukemia, stem cell transplant	Oral 160/800 mg trimethoprim + sulfamethoxazole twice a day for four weeks, oral azithromycin 500 mg once daily for two weeks, a single intravitreal 1 mg/0.1 ml clindamycin injection.	Epiretinal membrane
	Case 2	57 / F	BE	Acute myeloid leukemia, allogenic bone marrow transplant	Oral azithromycin 500 mg once daily for two weeks, three times of bilateral intravitreal injections of 1 mg/0.1 ml clindamycin.	None
	Case 3	52 / F	LE	Anti-TNF and salazopyrin treatment for AS	Oral 160/800 mg trimethoprim + sulfamethoxazole twice a day for five months, oral 500 mg/day azithromycin for two months, two times of left intravitreal injections of 1 mg/0.1 ml clindamycin + 0.4 mg/0.1 ml dexamethasone	Epiretinal membrane

AIDS; acquired immunodeficiency syndrome, AS; ankylosing spondylitis, BE; both eyes, F; female, LE; left eye, M; male, RA; rheumatoid arthritis, RE; right eye, SLE; systemic lupus erythematosus, TNF; tumor necrosis factor.

emphasizes the possibility of atypical presentations and diagnostic difficulties in immunocompromised patients with TC. A high index of suspicion for possible TC is a must in immunosuppressed patients with mild to severe posterior uveitis to establish the correct diagnosis promptly and employ the appropriate treatment.

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Declaration of Conflicting Interests

The authors declare that there is no conflict of interest.

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Informed Consent Statement

Written informed consent was obtained from the patients for publication.

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