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ORIGINAL ARTICLE

Fulminant Ocular Toxoplasmosis: The Hazards of Corticosteroid Monotherapy

Merih Oray, MD[1], Pinar Cakar Ozdal, MD[2], Zafer Cebeci, MD[1], Nur Kir, MD[1], and Ilknur Tugal-Tutkun, MD[1]

[1]*Department of Ophthalmology, Istanbul Faculty of Medicine, Istanbul University, Istanbul, Turkey and* [2]*Ulucanlar Eye Education and Training Hospital Ophthalmology Clinic, Ankara, Turkey*

ABSTRACT

Purpose: To describe fulminant toxoplasma retinochoroiditis induced by corticosteroid monotherapy.

Methods: Clinical records of nine patients were reviewed.

Results: All patients (five female, four male; aged 15–64 years) had been misdiagnosed as unilateral non-infectious uveitis and given systemic and/or local corticosteroid injections elsewhere. Mean disease duration before referral was 105.6 ± 71 (45–240) days. Visual acuity at presentation was <20/200 in six eyes. Average lesion size was 6.6 disc areas in eight eyes and all four quadrants were involved in one. Toxoplasma DNA was detected in eight tested eyes. Mean duration of anti-toxoplasmic therapy was 92.5 ± 37.1 days. Three eyes developed rhegmatogenous retinal detachment. Four patients underwent pars plana vitrectomy. Final visual acuity was <20/200 in five eyes.

Conclusions: Iatrogenic immunosuppression due to initial misdiagnosis may lead to an aggressive course and serious complications of ocular toxoplasmosis, a potentially self-limiting infection.

Keywords: Anti-toxoplasmic therapy, corticosteroid, necrotizing retinitis, ocular toxoplasmosis, retinochoroiditis

INTRODUCTION

Ocular toxoplasmosis is the most common cause of infectious posterior uveitis. The prevalence and clinical course of the disease varies significantly in different regions of the world, depending on the climate, socioeconomic factors, eating, and hygiene habits.[1–8] It represents the most common infectious cause of posterior uveitis cases seen at referral centers in Turkey.[9]

In immunocompetent patients, typical clinical presentation of ocular toxoplasmosis is characterized by a unilateral well-defined focus of retinochoroiditis, usually at the border of a pre-existing pigmented retinochoroidal scar and overlying vitritis.[1–5] Patients may also present with a primary active lesion without any visible scar in either eye. We have previously reported that 17.4% of 109 patients with active ocular toxoplasmosis presented without a pre-existing scar.[6] In such cases, its differential diagnosis from other infectious and non-infectious causes of posterior uveitis may be difficult. Clinical diagnosis and treatment may be even more challenging in immunocompromised patients who may present with atypical features such as bilateral involvement, large areas of retinal necrosis, and/or multifocal retinochoroiditis.[10–15].

We herein report the clinical features, diagnostic approach, and treatment results in nine patients who presented with fulminant toxoplasma retinochoroiditis following corticosteroid (CS) monotherapy administered elsewhere.

PATIENTS AND METHODS

Ocular toxoplasmosis was diagnosed in 118 patients who presented at the Uveitis Service, Istanbul University, Istanbul Faculty of Medicine, Department of Ophthalmology between February 2010 and July

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Correspondence: Ilknur Tugal-Tutkun, Istanbul Tip Fakultesi, Goz Hastaliklari A.D., 34390 Capa, Istanbul, Turkey. E-mail: itutkun@yahoo.com
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2014; and 32 patients who presented at Ulucanlar Eye Education and Training Hospital Ophthalmology Clinic between February 2012 and December 2014. Nine consecutive patients, five at the former and four at the latter referral center, who had been diagnosed with fulminant toxoplasma retinochoroiditis as a result of local and/or systemic immunosuppression due to CS monotherapy, were included in the study.

The research followed the tenets of the Declaration of Helsinki. The retrospective study protocol was approved by the Ethics Committee of Istanbul University, Istanbul Faculty of Medicine. An informed consent was obtained before all diagnostic and therapeutic procedures.

In this retrospective study, the clinical records of patients were reviewed for demographic data, ocular and medical history, ocular findings, color fundus photographs, laboratory results, and treatment modalities.

A complete ocular examination was performed at each visit, including best-corrected Snellen visual acuity, slit-lamp biomicroscopy, tonometry, and indirect ophthalmoscopy. Anterior chamber flare was measured by laser flare photometer (Kowa FC-2000, Kowa Company Ltd., Electronics and Optics Division, Tokyo, Japan) at the Uveitis Service, Istanbul Faculty of Medicine. A clinical grading of flare was done in patients seen at Ulucanlar Eye Education and Training Hospital. Standardization of uveitis nomenclature criteria was used for clinical grading of anterior chamber cells, flare, and vitreous haze.[16] Color fundus photographs were taken at the first visit and during follow-up. The area of retinochoroiditis was estimated based on a comparison with the surface area of the optic disc.

The aqueous humor (six patients) and vitreous samples (two patients) were submitted for real-time polymerase chain reaction (PCR) to detect *T. gondii* DNA to confirm the diagnosis of ocular toxoplasmosis in all and viral DNA to exclude viral necrotizing retinitis in five of them. Additional laboratory work-up included blood chemistry, complete blood count, and serum serology for IgM and IgG anti-toxoplasma antibodies, human immunodeficiency virus (HIV) antibodies as well as specific and nonspecific tests for syphilis, chest X-ray, and QuantiFERON® TB Gold test (Cellestis Inc. Valencia, CA, USA).

RESULTS

Demographic features and previous diagnoses and treatment are shown in Table 1. Five patients were female and four were male. There was only one patient in the pediatric age group and two patients were older than 60 years.

The mean duration between onset of ocular symptoms and presentation to us was 105.6 ± 71 (45–240) days. All patients had unilateral active retinochoroiditis. Three patients had concomitant systemic diseases, including non-Hodgkin lymphoma in remission (Patient 5) and rheumatoid arthritis (Patients 4 and 7). One of them (Patient 4) had ongoing immunosuppressive therapy (methotrexate) combined with low-dose oral CS for the treatment of her systemic disease. All patients had been previously misdiagnosed elsewhere. Seven patients had received high-dose systemic CS (20–80 mg/day of prednisolone or equivalent for 1–6 weeks) and were still under systemic CS therapy (prednisolone 10–80 mg/day) at presentation to us. Five patients had received periocular or intravitreal CS injections. One of them had an intravitreal dexamethasone implant, which had been placed 2 weeks after an intravitreal triamcinolone acetonide (IVTA) injection.

Ocular findings at presentation and results of ocular fluid PCR analysis are shown in Table 2. Best-corrected visual acuity (BCVA) at presentation was <20/200 in six eyes and between 20/200 and 20/40 in three eyes.

TABLE 1. Demographic features and previous diagnoses and treatment in nine patients who presented with fulminant ocular toxoplasmosis.

Patient no.	Gender/age	Concomitant disease/treatment	Ocular symptom duration (days)	Previous diagnosis	Previous treatment employed elsewhere
1	M/36	None	150	Autoimmune uveitis	Oral CS, IVTA, intravitreal bevacizumab injection
2	F/22	None	45	Autoimmune uveitis	Oral CS
3	F/15	None	120	Behçet uveitis	Oral CS, IVTA, azathioprine
4	F/61	RA/MTX+CS	240	ARN	Oral CS, Oral acyclovir
5	M/44	NHL/None	45	Autoimmune uveitis	Subtenon's CS injection
6	M/44	None	60	Behçet uveitis	Oral CS
7	F/64	RA/None	180	Autoimmune uveitis	Oral CS
8	M/22	None	60	Autoimmune uveitis	Oral CS, IVTA, intravitreal bevacizumab injection, subtenon's CS injection
9	F/33	None	50	Autoimmune uveitis	IVTA, intravitreal dexamethasone implant

M, male; F, female; RA, rheumatoid arthritis; MTX, methotrexate; CS, corticosteroid; NHL, non-Hodgkin lymphoma; ARN, acute retinal necrosis; IVTA, intravitreal triamcinolone acetonide.

TABLE 2. Ocular findings at presentation and results of ocular fluid PCR analysis in nine patients with fulminant ocular toxoplasmosis.

Patient no./Eye	Visual acuity (Snellen)	Anterior chamber cells	Anterior chamber flare (Photon/ms)	IOP (mmHg)	Vitreous haze grading	Anterior vitreous cells	Number of foci of active retinochoroiditis	Size (disc area)/location of retinochoroiditis	Pre-existing scar	PCR (number of copies of toxoplasma DNA)
1/OS	LP	3+	63.6	26	1+	3+	Extensive in 4 quadrants	Extensive in 4 quadrants	No	Toxoplasma DNA + (786 000)
2/OD	CF at 1 meter (20/800)	2+	1+*	13	2+	2+	1	7/posterior pole	No	Toxoplasma DNA + (17 200)
3/OS	CF at 1 meter (20/800)	4+	2+*	16	3+	3+	1	15/posterior pole	Yes (fellow eye)	Toxoplasma DNA + (1.8×10^7)
4/OD	20/50	3+	48	16	2+	3+	2	1 and 15/peripheral	No	Toxoplasma DNA + (12 600)
5/OD	20/200	4+	79.1	15	2+	3+	2	2 and 3/midperipheral	No	Toxoplasma DNA + (13 450)
6/OS	CF at 1 meter (20/800)	2+	1+*	14	3+	2+	1	6/midperipheral	Yes	N/A
7/OD	20/60	4+	53.2	15 (D+T)	1+	2+	2	5 and 6/midperipheral	No	Toxoplasma DNA + (19 800)
8/OS	HM	1+	12.5	13	3+	3+	1	8/posterior pole	Yes (bilateral)	Toxoplasma DNA + (69 900)
9/OD	HM	1+	0+*	17	0.5+	2+	1	5/posterior pole	No	Toxoplasma DNA + (1 770 000)

LP, light perception; CF, counting fingers; HM, hand movement; IOP, intraocular pressure; D+T, dorzolamide+timolol; PCR, polymerase chain reaction; N/A, not available
*Anterior chamber flare clinical grading using standardization of uveitis nomenclature criteria.

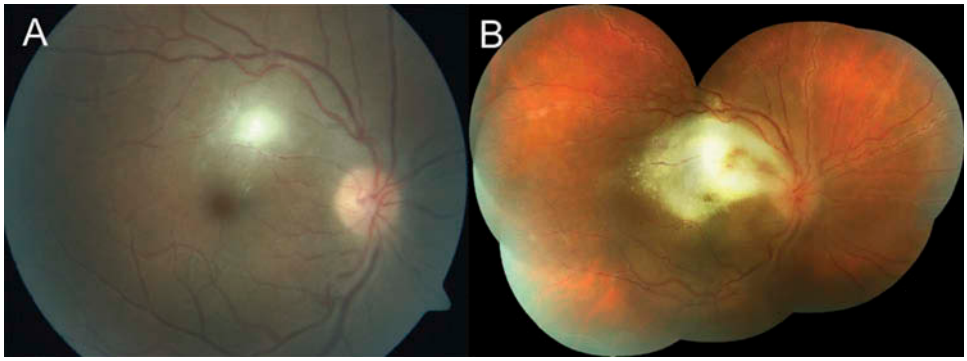

FIGURE 1. Color fundus photograph of Patient 9 with ocular toxoplasmosis at presentation elsewhere shows active retinochoroiditis associated with adjacent periarteriolitis located at the posterior pole (A). Color fundus photograph at presentation to us (30 days after intravitreal triamcinolone acetonide and 16 days after intravitreal dexamethasone implant injections elsewhere) shows significantly enlarged area of retinochoroiditis at the posterior pole together with optic disc inflammation and extensive periphlebitis (B).

All patients had vitritis. Five patients had one, and three patients had two retinochoroiditis foci of up to 15 disc areas, involving the posterior pole in four patients (Figure 1). One patient had extensive retinochoroiditis covering four quadrants (Figure 2). There were three patients with retinochoroidal scars. In only one patient, the active retinochoroiditis was at the border of a pre-existing pigmented retinochoroidal scar. One patient had a scar in the involved eye distant from the active lesion and another scar in the fellow eye. Another patient had a scar only in the fellow eye. All patients had positive serum anti-toxoplasma IgG antibodies (264–1000 IU/mL), but negative IgM antibodies. Complete blood count was within normal limits in all patients. All patients revealed negative serology for syphilis and HIV. Two patients had latent tuberculosis. *Toxoplasma gondii* DNA was detected in the vitreous sample of two and in the aqueous humor of six patients.

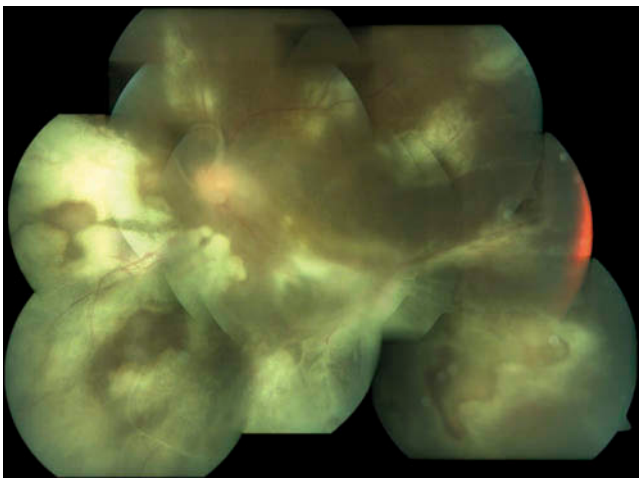

FIGURE 2. Color fundus photograph of Patient 1 with fulminant ocular toxoplasmosis at presentation showing extensive retinochoroiditis covering four quadrants.

Table 3 shows treatments and outcomes. Patients initially received the following anti-toxoplasmic drugs in combination: pyrimethamine (25 mg twice a day), clindamycin (300 mg three times a day), and azithromycin (500 mg once a day) in five patients; and trimethoprim-sulfamethoxazole (160 mg/800 mg twice a day) along with azithromycin (500 mg once a day) in four patients. In two patients, pyrimethamine was switched to trimethoprim-sulfamethoxazole (160 mg/800 mg twice a day) after 25 and 40 days due to leukopenia (Patient 8) and thrombocytopenia (Patient 7), respectively. Clindamycin therapy was stopped in one patient (Patient 7) because of diarrhea. Azithromycin (500 mg once a day) was used during the 1st week of treatment in three patients and administered for 20–80 days in four patients. Clindamycin (300 mg three times a day) was started after 1 month in one patient (Patient 3). Intravitreal clindamycin (1000 µg) injection was done in three patients, at the time of pars plana vitrectomy (PPV) in two (Patients 3 and 9) and after 2 weeks of systemic therapy in one patient (Patient 5). A second injection of clindamycin was given after 1 month in one patient (Patient 3). Mean duration of anti-toxoplasmic therapy was 92.5 ± 37 (60–150) days in eight patients. Patient 9 is still under treatment.

Two patients with recent IVTA or intravitreal dexamethasone implant injections underwent PPV for removal of depot CS immediately after the first visit. Systemic CS was also immediately stopped in six patients, and tapered and continued at a low dose in one patient. Systemic CS treatment was restarted in four patients after 1 week to 3 months. Reinstitution of systemic CS had to be determined on an individual basis, and it was considered only after we had a clinical impression of infection control. Corticosteroid therapy was restarted after 1–3 weeks in three patients for the treatment of accompanying inflammation, especially the vitreous haze. In one patient, CS was administered at 3 months for the treatment of

TABLE 3. Treatment and outcomes in nine patients with fulminant ocular toxoplasmosis.

Patient no.	Anti-toxoplasmic treatment	Duration of anti-toxoplasmic treatment (days)	CS therapy	Visual acuity (Snellen)	Fundus	Complication	Follow-up time (months)
1	P+C+A → P+C	60	Stopped at 1st visit	LP	Total RD	RD (2nd month)	2
2	TS+A → TS	75	Stopped at 1st visit; restarted after 3 weeks	CF at 3 meters (20/250)	Scar formation	None	3
3	TS+A → TS+C Intravitreal C injection (×2)	90	CS and AZA stopped at 1st visit PPV for IVTA removal	HM	Scar formation, subretinal and preretinal fibrosis	Subretinal and preretinal fibrosis (3rd month)	30
4	P+C+A → TS+C → TS	150	Tapered and continued at a low dose (5mg/day)	20/50	Scar formation	RD (4th month)	5
5	P+C+A → P+C Intravitreal C injection	60	Started after 1st week	20/25	Scar formation	Macular lamellar hole (10th month)	12
6	TS +A → TS	60	Stopped at 1st visit; restarted after 2 weeks	20/25	Scar formation	None	8
7	P+C+A → TS	95	Stopped at 1st visit	20/50	Scar formation	None	11
8	P+C+A → TS+C+A → TS+A → TS	150	Stopped at 1st visit; restarted after 3 months	CF at 1 meter (20/800)	Scar formation, subretinal and preretinal fibrosis	RD (2nd month) subretinal and preretinal fibrosis	10
9	TS+A → P Intravitreal C injection	120 days (ongoing treatment)	PPV for intravitreal dexamethasone implant removal	HM	Clinical improvement with ongoing treatment	None	3

P, pyrimethamine; C, clindamycin; A, azithromycin; TS, trimethoprim-sulfamethoxazole; CS, corticosteroid; AZA, azathioprine; PPV, pars plana vitrectomy; IVTA, intravitreal triamcinolone; LP, light perception; CF, counting fingers; HM, hand movement; RD, retinal detachment.

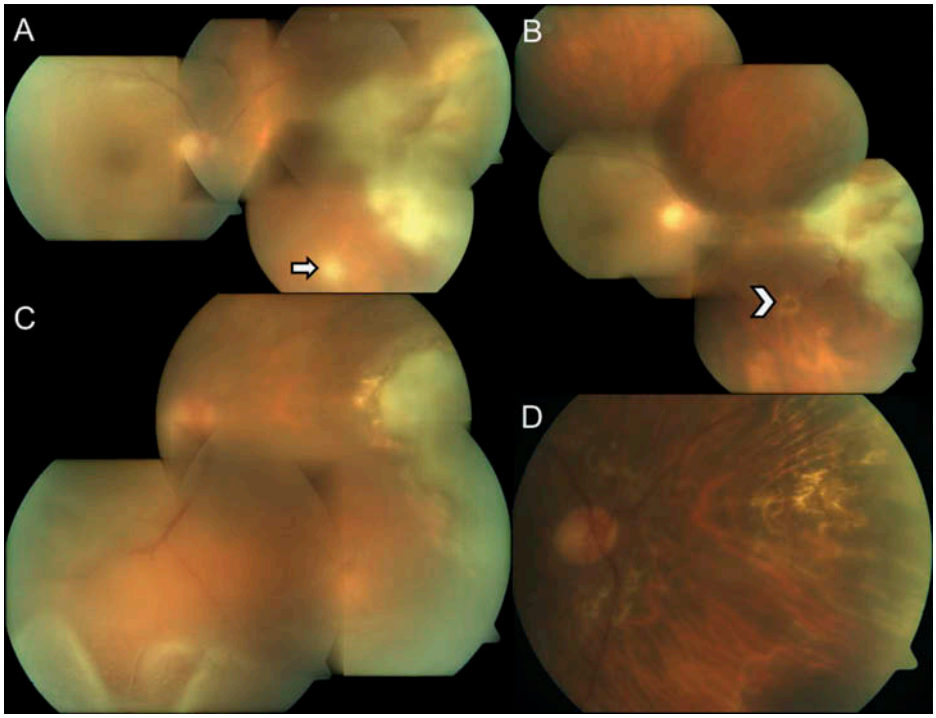

FIGURE 3. Color fundus photograph of Patient 4 with fulminant ocular toxoplasmosis at presentation shows an extensive area of active retinochoroiditis at the nasal quadrant and a satellite active lesion inferiorly (arrow) (A). On the 45th day of treatment the satellite lesion revealed scar formation (arrowhead) and the wide area of retinochoroiditis started to resolve with distinct margins (B). Retinal detachment developed at 4 months (C). After 6 months of anti-toxoplasmic treatment and pars plana vitrectomy with silicone oil injection, a complete scar formation was seen (D).

emergent periphlebitis. After the resolution of active retinochoroiditis, trimethoprim-sulfamethoxazole (160 mg/800 mg three times a week) as prophylactic therapy, was initiated in one patient who had ongoing immunosuppressive treatment for concomitant systemic disease (Patient 4).

Ocular complications during follow-up included retinal detachment in three patients; extensive preretinal and subretinal fibrosis formation in two patients, and macular lamellar hole formation in one patient. Two patients with retinal detachment underwent successful scleral buckling, PPV, and intraocular silicone oil injection (Patients 4 and 8; Figures 3 and 4, respectively). Final BCVA was 20/50–20/20 in four, counting fingers in two, hand movements in two, and light perception in the patient with inoperable retinal detachment.

Patient 8 is now described in more detail.

Patient 8

A 22-year-old male presented with redness, pain, and decreased vision in the left eye having failed to respond to treatments employed elsewhere, including systemic CS therapy, intravitreal bevacizumab, IVTA, and subtenon's depot CS injections during the last 2 months.

The patient's past ocular history included two episodes of uveitis activation in the last 3 years, treated with only topical CS. Color fundus photographs taken elsewhere 1 year prior to presentation to us, revealed three foci of pigmented scars at the macular area in the left eye (Figure 4). He was immunocompetent and had no underlying systemic disease.

At presentation, the patient was still on an oral dose of 16 mg/day methylprednisolone and topical dexamethasone 4 times/daily. He had a BCVA of 1.0 in the right and hand movement in the left eye. Slit-lamp examination findings were within normal limits in the right eye. Left eye had circumciliary congestion, fine keratic precipitates, and 1+ anterior chamber cells, no flare, freely mobile pupil, clear lens, and 3+ cells in the anterior vitreous with 3+ vitreous haze. Intraocular pressure was 14 mmHg in the right and 13 mmHg in the left eye. Laser flaremeter readings were 5.3 photons/ms in the right and 12.5 photons/ms in the left eye. Fundus examination revealed retinochoroiditis scars located inferiorly in both eyes, and dense vitreous haze overlying the posterior pole, disc edema, and active retinochoroiditis involving the entire macula in the left eye (Figure 4).

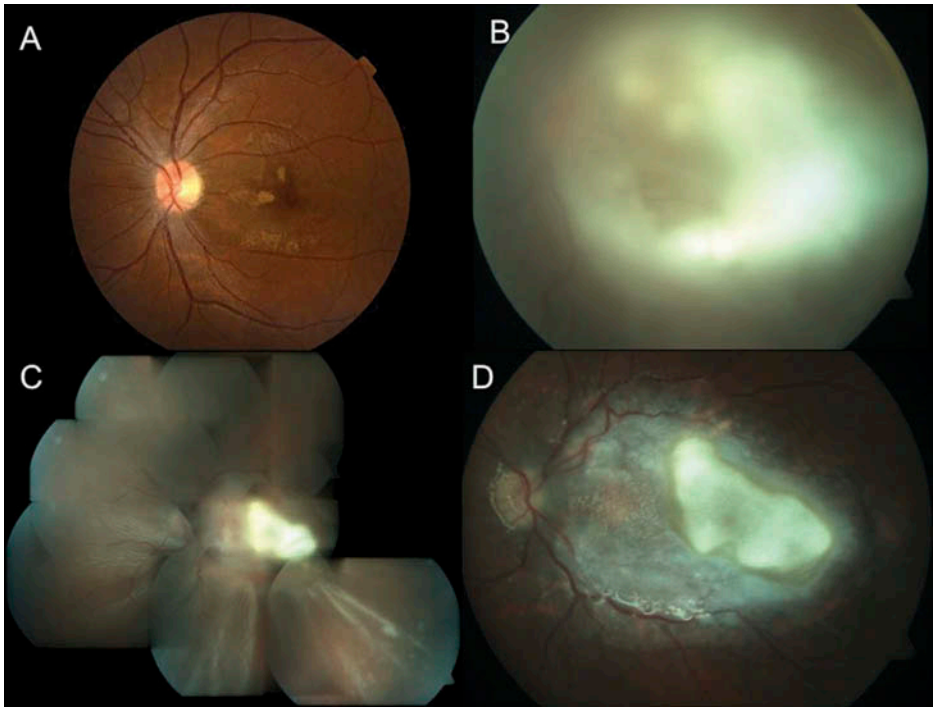

FIGURE 4. Color fundus photograph of Patient 8 with ocular toxoplasmosis taken elsewhere 1 year prior to presentation to us, revealed three foci of pigmented scars at the macular area in the left eye (A). Color fundus photograph at presentation showing 2+ vitreous haze, disc edema, and active retinochoroiditis involving the entire macula in the left eye (B). Retinal detachment developed on the 75th day of the treatment (C). At the time of last visit, 2 months after cessation of the treatment, there was a prominent fibrotic macular scar with pigmented borders and a pale disc (D).

The patient was diagnosed with fulminant toxoplasma retinochoroiditis presumably exacerbated by systemic CS, IVTA, and subtenon's depot CS injections without anti-toxoplasmic coverage. The diagnosis was confirmed by positive aqueous humor PCR for *T. gondii*-DNA. Serum anti-toxoplasmosis IgG antibody titer was high (700 IU/mL) and IgM was negative. Systemic methylprednisolone therapy was stopped immediately and the patient was started on pyrimethamine 25 mg twice daily, clindamycin 300 mg every 8 h, and azithromycin 500 mg once a day. At 25 days after initiation of pyrimethamine, the white blood cell count decreased and it was switched to trimethoprim-sulfamethoxazole (160 mg/800 mg twice a day), which was continued for 125 days. Clindamycin therapy was given for 2 months and azithromycin for 80 days. Lesion size started to decrease 10 days after initiation of anti-toxoplasmic therapy.

Retinal detachment developed on the 75th day of the treatment (Figure 4). The patient then underwent scleral buckling, and PPV with silicone oil placement. Postoperatively, there was pigmentation around the margins of the retinochoroidal lesion, which was surrounded by retinal and subretinal fibrosis. Although there was no activation of retinochoroiditis, emergence of peripheral periphlebitis was observed 20 days after the surgery and oral prednisolone 20 mg/day was added to ongoing anti-toxoplasmic therapy. Oral CS was tapered and stopped in 3 weeks and anti-toxoplasmic therapy was continued for another month. At the time of the last visit, 5 months after cessation of the treatment, there was a prominent fibrotic macular scar with pigmented borders, pale disc (Figure 4), and BCVA of counting fingers at 1 meter.

DISCUSSION

It has long been known that systemic CS monotherapy may induce a fulminant form of ocular toxoplasmosis.[10,17] There are also recent reports of fulminant toxoplasmosis associated with IVTA.[10,18–20] To the best of our knowledge, there has been no previous report of fulminant toxoplasmosis following intravitreal dexamethasone implant. In the present study, all patients had received systemic and/or local CS therapy without proper anti-toxoplasmic coverage due to initial misdiagnosis elsewhere. Subsequently, all had an aggressive disease and the clinical presentation was atypical when we first saw the patients.

After prolonged use of systemic CS and/or presence of intra- or periocular depot CS in the eye, *T. gondii* retinochoroiditis may spread extensively,

making it difficult to distinguish toxoplasmosis infection among the different infectious and non-infectious causes of retinochoroiditis. Especially when peripheral large areas of necrotizing retinochoroiditis are seen as in three patients in this series (Patients 1, 3, and 4), it may mimic viral retinitis.[10,11] One of the patients had indeed been misdiagnosed as necrotizing herpetic retinopathy and treated with an antiviral agent (Patient 4).

Serum serology may be used for confirmation of the clinical diagnosis in typical cases of ocular toxoplasmosis. Papadia et al. [21] reported significant elevation of IgG titers in patients with active disease with a mean antibody level of 147.75 ± 259.4 IU/mL. Patients in the present series had markedly elevated IgG antibody levels ranging from 264 to 1000 IU/mL, which may be associated with the severity of infection. Analysis of intraocular fluids may be required for definitive diagnosis.[10,12,18,22–24] In order to demonstrate intraocular specific antibody synthesis, the Goldmann–Witmer coefficient (GWC) is calculated based on antibody titers in ocular fluid and serum samples. However, this method is not available at our institutions. Therefore, we have performed only PCR analysis of the aqueous humor and vitreous samples in our patients. In fact, in the setting of immunosuppression, patients with infectious posterior uveitis are more likely to yield positive PCR results[12,25,26] and sensitivity of GWC may be reduced.[27,28] Polymerase chain reaction yield is also considered to be high in the setting of large exudative lesions and severe vitritis, as was the case in patients presented here.[1,12,27]

There is no consensus regarding treatment of ocular toxoplasmosis. In two recent meta-analyses no strong evidence has been found to support routine treatment for all immunocompetent patients with active toxoplasmic retinochoroiditis.[29,30] Nevertheless, two surveys conducted in 1991 and 2001 suggest that uveitis specialists have an increased tendency to treat ocular toxoplasmosis.[31,32] There is no consensus regarding the best anti-toxoplasmic regimen, either. A total of 78 responders who participated in the 2001 survey described a total of 24 different regimens utilizing nine anti-toxoplasmic drugs or combinations.[31] The most frequent treatment also known as "classic therapy" consisted of pyrimethamine, sulfadiazine, and prednisone.[31,32] In a few randomized clinical trials various regimens have been shown to have similar efficacy.[33–36]

There is controversy about the route, initiation, and timing of adjunctive CS therapy. Ocular toxoplasmosis patients are usually treated with systemic CS starting simultaneously or 24–72 h after initiation of the antibiotherapy in order to control the inflammatory tissue response and to speed up the clearing of the visual axis.[2,30,31,37,38] Corticosteroids are tapered off before discontinuation of anti-toxoplasmic therapy.[2] There are recent reports suggesting the use of intravitreal dexamethasone injection combined with intravitreal clindamycin, especially in patients where systemic therapy is ineffective, contraindicated, or not tolerated.[33,39,40] However, a case of fulminant toxoplasmic retinochoroiditis has been reported due to IVTA injection, despite simultaneous systemic anti-toxoplasmic therapy.[19]

In the present series, parasite proliferation, rather than inflammation seemed to be the major cause of tissue damage; therefore, all patients received pyrimethamine or trimethoprim-sulfamethoxazole in combination with clindamycin and azithromycin. We did not initiate CS at the beginning of the treatment and even discontinued CS immediately at presentation in most of the patients because of the fulminant infection induced by previous systemic and/or local depot CS injections without anti-toxoplasmic coverage. In two cases with recent IVTA or intravitreal dexamethasone implant administration, PPV was performed to remove the depot CS.

In typical cases of ocular toxoplasmosis, a good response with resolution of inflammation and development of characteristic hyperpigmentation of the lesion can be observed after 4–6 weeks of treatment.[6] However, lesions >1 disc area may persist longer, have a higher rate of complications and vision loss, and require longer treatment period than smaller lesions.[29] Because of the extensive lesion size in most of our cases and the presence of an underlying local and/or systemic immunosuppression, treatment duration was much longer. Furthermore, one patient was placed on long-term prophylactic trimethoprim-sulfamethoxazole regimen because she had to continue low-dose oral CS and immunosuppressive agent for her concomitant systemic disease. Optimum duration of prophylactic treatment is not known.[41,42]

Ocular toxoplasmosis may be associated with vitreoretinal complications including retinal vessel occlusions, vitreous hemorrhage or opacities, epiretinal membrane and macular hole formations, choroidal neovascular membranes, and rhegmatogenous and/or tractional retinal detachments, which may potentially cause severe visual loss and require vitreoretinal surgery.[3,13,43,44] It is well known that this risk is increased in immunocompromised patients with severe ocular disease who have large areas of retinal necrosis and higher risk for retinal breaks.[4] Retinal detachments have been related to the severity of inflammation and may be inevitable in fulminant cases even if adequate treatment is administered as seen in three patients in this series (Patients 1, 4, and 8).[43]

In conclusion, it is well known that CS therapy alone without antiparasitic coverage may lead to an aggressive course of ocular toxoplasmosis, a potentially self-limiting infection. The present series shows that patients may still be misdiagnosed and mistreated even in a country where toxoplasmic retinochoroiditis is the most common infectious cause of posterior

uveitis. With increasing use of intravitreal CS in uveitis practice, an increased awareness of the infectious etiologies is required to prevent catastrophic consequences.

DECLARATION OF INTEREST

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the article.

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