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ORIGINAL PAPER

Clinical manifestation and prognosis of active ocular toxoplasmosis in Iran

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Abstract Toxoplasmosis is the most common cause of posterior uveitis in the world. This study described the clinical characteristics and visual outcome of 193 patients with ocular toxoplasmosis at Feiz Hospital (Isfahan, Iran) during the last six years. The setting and design used was a retrospective non-comparative observational case series. In this study, 193 patients with ocular toxoplasmosis (111 female, 82 male) were enrolled. The distribution of symptoms and fundoscopic findings were studied. The most-reported chief complaint was blurred vision in 96 % (184 patients) and floaters in 13.47 % (25 patients) of cases and most frequent clinical manifestations were chorioretinitis 98.48 % (190 patients), macular scars 50.7 % (98 patients), and atrophic optic papilla two (1.03 %) patients. Primary retinal lesions were observed in 16 (8.2 %) and combination of active lesions and old retinochoroidal scars in 177 (91.7 %) of the patients. Retinal detachment occurred in 11 (5.69 %) patients. Bilateral involvement was found in 27 % of patients. Blindness was 0.05 % after treatment. Recurrence rate was 14.5 %. In conclusion, ocular toxoplasmosis substantially varies among patients with different age, gender, site of lesion and other factors. Suddenly blurred vision, floater, and pain could be caused by *Toxoplasma gondii*. Flashing, may necessitate a more precise peripheral fundus examination.

Keywords Ocular toxoplasmosis · Manifestation · Complication

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Introduction

Toxoplasmosis is the most common cause of posterior uveitis in the world and has the potential to cause blindness and visual impairment in children and young adults due to posterior segment abnormality [1].

Signs and symptoms of ocular toxoplasmosis vary with age. Children are generally referred to an ophthalmologist complaining of decreased visual acuity, strabismus, nystagmus, leukocoria, choroidal coloboma and microphthalmia [2].

The typical complaints in ocular toxoplasmosis, in adolescents and adults, are blurred vision, floaters and sometimes pain, and photophobia and conjunctival hyperemia if the anterior segment is involved. The most common cause of visual loss in ocular toxoplasmosis is a macular scar, but causes of substantial visual loss in ocular toxoplasmosis include dragging of the macula secondary to peripheral lesion, retinal detachment, macular edema, optic atrophy, cataract, glaucoma, opacification of the media, amblyopia and phthisis.

Evidence from clinic-based follow-up studies suggests that retinochoroidal lesions occur in over 80 % of people with congenital toxoplasmosis [3–5].

Despite years of extensive research, the development of novel antitoxoplasmic drugs, and preventive measures directed toward this ubiquitous infection, many basic questions about ocular toxoplasmosis remain unanswered, and curative therapy is not available.

The purpose of this study was to determine in a large series of patients the clinical features of ocular toxoplasmosis infection and some complications of ocular toxoplasmosis, and to introduce two new ones not hitherto encountered in the literature, and, if possible, to identify a profile of patients at risk of visual loss. It was hoped that the result would be useful for comparison with previous studies outside Iran.

Materials and methods

This study was a descriptive retrospective study. The data were collected from the Uveitis Subdivision and the Vitreo-retina Subdivision of Feiz Hospital medical practice. We conducted a retrospective analysis of the medical records of 1,000 consecutive patients with uveitis who had consulted our department between 2003 and 2009, to identify patients with the diagnosis of ocular toxoplasmosis. The study protocol was reviewed and approved by the Clinical Research Ethics Committee of the Iran University of Isfahan. In this study we included 193 consecutive patients with active lesions of ocular toxoplasmosis (first attack and/or recurrence) and excluded eight patients with an asymptomatic retinochoroidal scar compatible with the diagnosis of ocular toxoplasmosis who were not examined during the active stage of the disease. Diagnosis was established by clinical characteristics consistent with retinochoroiditis (foci of retinal necrosis), in the absence of other identifiable diseases, by one ophthalmologist working in retina and vitreous, and by laboratory examinations.

The clinical diagnosis of ocular toxoplasmosis was based on criteria formulated by Holland et al. [6]. Active ocular toxoplasmosis was defined by the presence of an active creamy-white focal retinal lesion eventually resulting in hyperpigmented retinochoroidal scars in either eye. Primary ocular toxoplasmosis was defined as an active creamy-white focal retinal lesion together with specific immunoglobulin (Ig) M antibodies. Recurrent ocular toxoplasmosis was defined as an active retinochoroidal lesion in the presence of old pigmented retinochoroidal scars in either eye. Central lesions were defined as lesions located within the large vascular arcades. The laboratory examinations were IgG and IgM for toxoplasmosis in patients' blood sera.

Serological criteria for the acute phase of systemic infection with *Toxoplasma gondii* included the presence of specific IgM antibodies. All patients with primary lesions were examined for the presence of IgM and for comparison purposes this assay was also requested in some patients with recurrent lesions or in the quiescent phase. The chronic phase of systemic infection was defined as positive IgG antibodies (any positive titer) without IgM antibodies. *T. gondii* serology was performed routinely in all cases by ELISA IgG and IgM commercial assays (Euroimmun). Congenital infection was defined by the presence of a positive PCR assay on amniotic fluid during pregnancy or as ocular symptoms at birth accompanied or not by neurological symptoms (micro- or macrocephaly or cerebral calcifications) with a persistent IgG anti-*Toxoplasma* at 12 months of life, and acquired infection was defined in adults (>18 years of age) with a specific IgM-positive test. Patients under 18 years of age and with IgM were considered to be of uncertain origin because some patients with confirmed congenital toxoplasmosis have persistent specific IgM-positive tests up to 10 years of age [7].

For the purpose of this study, retinochoroidal lesions were subdivided into three groups: smaller than disc, equal to disc and larger than disc.

Patients with acquired immunodeficiency syndrome (AIDS) were excluded, because it may affect clinical presentation of the disease. Chest X-ray, fluorescent treponemal antibody, complete blood cell count, C-reactive protein, erythrocyte sedimentation rate, Lyme and *Bartonella* assays and purified protein derivative (tuberculin) tests were performed in patients without typical presentation.

Legal blindness was defined as the best-corrected visual acuity of the affected eye equal to or less than 20/200 [8–10].

Visual outcome is given by the final optimal visual acuity (not the worst visual acuity at any visit). Data included age, sex, occupation, chief complaint, visual acuity, clinical features, bilateral involvement,

therapeutic outcome and complications. All of the data were tabulated and analyzed descriptively and statistically.

Data were analyzed by SPSS v.13 (SPSS Inc., Chicago, IL, USA). Chi-squared tests and *t*-tests were used for the quantitative and qualitative data, respectively. $P < 0.05$ was considered statistically significant.

Results

In the preceding 6 years, there were 193 ocular toxoplasmosis cases, consisting of 82 (43 %) males and 111 (57 %) females. The age range of patients at first presentation to the ophthalmologist with an active ocular toxoplasmosis lesion was 2–65 years with a mean age of 24.6 years, but mostly in the 2nd and 3rd decades ($n = 104$). The mean age was 23.4 years in men and 25.1 years in women. Subclinical (quiet) retinochoroidal scars in addition to active lesions were observed in 177 of 193 patients (91.7 %) at the time of first presentation with active ocular toxoplasmosis. These old scars were located in the eye with the active lesion ($n = 130$) and in the contralateral eye ($n = 47$). No previous scars were observed in 16 of 193 (8.2 %) patients. In 94.3 % (182) of the patients, ocular toxoplasmosis became manifest before the age of 40 years (in 67.8 % between 15 and 35 years).

At the end of follow-up, bilateral disease was present in 53 (27 %) patients. Patients with primary ocular toxoplasmosis were older than those who were first seen with a combination of active lesions and old scars ($P = 0.03$). Acquired infection was documented in 16 (8.2 %) patients and infection of uncertain origin in 177 (91.7 %) patients.

Serological characteristics of the acute phase of systemic infection were present in 35 (18.1 %) patients. Serological characteristics of the chronic phase of systemic infection were present in 158 (81.9 %) patients.

The presenting symptom was blurred vision in 181 patients (93.78 %), floater in 25 patients (13.4.0 %) and pain in two patients (1.03 %) (Table 1).

Abnormality was commonly found in the posterior segment, especially in the macular area. Sixteen of the chorioretinitis cases were young adults, 19–23 years old, who were assumed to have acquired toxoplasmosis; they had a history of fever, and presented with regional lymphadenopathy.

Table 1 Chief complaints of ocular toxoplasmosis (193 cases)

Symptom(s)	Total	%
Blurred vision	184	96
Floaters	25	13.47
Pain	2	1.03

Table 2 Ocular toxoplasmosis manifestations in the posterior segment (193 cases)

Clinical features		Total	%
Typical (retinitis on border of scar)		177	91.7
Atypical (retinitis without scar)	Neuroretinitis	1	0.51
	Papillitis	2	1.03
	Retinitis without scar	13	6.7

Table 3 Characteristics of retinal lesions in ocular toxoplasmosis (affected eyes) location of retinal lesions (n = 193 eyes)

Locatin	Number	%
Within vascular arcades (central)	124	64.2
Macular	98	50.7
Extramacular	26	13.5
Peripheral	62	32.1
Both central and peripheral	7	3.6
Extensive retinal lesions (larger than optic disc diameters) at presentation	76	39.3
Macular	98	50.7
Adjacent to optic nerve other central location	13	6.7

Among the abnormalities that threatened vision were neuroretinitis, papillitis, optic nerve atrophy, macular scar and juxtapapillaris scar. Ocular manifestations of ocular toxoplasmosis are summarized in Tables 2 and 3. In 124 patients (64.2 %) the retinal lesion was central; 62 patients had a peripheral lesion (32.1 %).

Associated clinical characteristics during attacks of ocular toxoplasmosis are given in Table 4. Intraocular inflammation was more severe in patients with acquired or primary ocular toxoplasmosis and eight patients had anterior uveitis without associated active retinal lesions. Multiple active lesions, which occurred simultaneously during the same attack, were noted in 22 patients.

Table 4 Clinical characteristics of patients with ocular toxoplasmosis

Total	Number	%
Anterior chamber reaction	73	37.8
Keratic precipitates	63	32.6
Fine keratic precipitates	55	28.49
Mutton fat keratic precipitates	8	4.1
Papillitis	2	1.03
Vitritis	191	98.9
Vascular sheatening	140	72.5
Vein and arterial sheatening	28	38.3
Arterial sheatening	74	38.3
Vein sheatening	38	19.6

All patients received a specific therapy with pyrimethamine, trisulfa or steroid. Improvement of visual acuity occurred especially in acute or relapsed cases and those involving the macula or optic nerve papilla.

Of 193 ocular toxoplasmosis patients who were followed for more than 5 years, 28 (14.5 %) developed one or more recurrences: 26 (92.8 %) patients had one recurrence and two patients had two recurrences. Recurrent disease in both eyes developed in two of 28 (7.1 %) patients. The percentage of patients with recurrences increased with follow-up time.

Complications developed in 26 of 193 (13.47 %) patients, whereby five (2.5 %) required at least one (intra)ocular surgical procedure. Retinal detachment occurred in 11 (5.69 %) patients. Pars plana detachment occurred in one case and was treated with cryopexy and prophylaxis. In three patients cystoid macular edema was obvious on fundus slit lamp examination during active attacks, and four patients developed cellophane maculopathy and macular pucker (Table 5).

Table 5 Ocular complications caused by congenital toxoplasmosis (n = 193)

Complications	Total	%
Regmatogenic retinal detachment	5	2.5
Serous retinal detachment	7	3.62
Vitreous hemorrhage	1	0.51
Pars plana detachment	1	0.51
Cystoid macular edema	3	1.55
Macular pucker	4	2.07
Large macular scar	4	2.07
Optic nerve atrophy	2	1.03

Table 6 Visual impact of ocular toxoplasmosis. Visual acuity frequency (WHO criteria) (n = 193)

Vision	n	%
Good vision >7/10	100	51.8
Visual impairment (6/10–<20/200)	74	38.33
Blindness (<20/200)	19	9.8

Blindness in one eye developed in 19 of 193 (21 %) patients before treatment and one eye became legally blind after treatment. Legal blindness was caused predominantly by the macular location of the retinal lesion in 13 of 19 (68.4 %) patients, retinal detachment in four (21 %) patients and optic nerve atrophy in two (10.5 %) patients (Table 6).

Discussion

This study describes the clinical features and visual outcome of ocular toxoplasmosis in a large series of patients.

In our study, the mean age at first presentation with symptomatic ocular toxoplasmosis was 24.6 years, and 67.8 % of the cases became manifest between 15 and 35 years, which is similar to the mean age reported previously [11, 12]. However, because quiescent subclinical retinochoroidal scars were already present in most patients (177 of 193; 91.7 %) at the time of the first clinical presentation with active ocular toxoplasmosis, the exact age at onset of ocular toxoplasmosis remains unknown for most patients. The presence of inactive retinal scars at the time of first presentation with active lesions has already been noted [11, 13, 14].

As in previous studies [15, 16], elderly patients had larger lesions. This was attributed to the possible decline of cell-mediated immunity in the elderly [17, 18].

In our study, ocular toxoplasmosis was more prevalent in females (57.0 %), which is similar to some previous studies [15, 19]. The frequency of 18.1 % of a positive IgM assay was lower than that reported previously of 37 % for patients during the acute phase of ocular toxoplasmosis in Iran by the highly-sensitive assay ISAGA [20, 21]. This could be partly explained by the different commercial assays used.

The exact location of the lesions in ocular toxoplasmosis was not reported in most of the previous studies [11, 12, 22]. Macular lesions were found in 50.7 % of the patients, which is in agreement with earlier studies [13, 23], and most patients (67.8 %) had an ocular lesion in the posterior pole, which was in agreement with other authors [24, 25].

We found papillitis (an atypical presentation of ocular toxoplasmosis) in 1 % of cases, which is lower than the 5 and 7 % reported in Turkey and Colombia [7, 26].

Bilateral involvement in ocular toxoplasmosis varies between 22 and 40 % [11, 14, 27–30], which is consistent with the 27 % found in this series. If we accept that bilateral involvement is more prevalent in congenital cases, we can conclude that our series includes a large number of acquired toxoplasmosis cases [31].

The most common symptom was a decrease in visual acuity (93.78 % of patients), caused by macular lesion, optic papilla, juxtapapillaris, the presence of vitreous cells and inflammation in the anterior segment [32].

Eight patients had anterior uveitis without associated active retinal lesions; all already had old retinal scars. Anterior uveitis could be caused by the parasite itself or because small chorioretinal lesions were not detected at ophthalmoscopy [33, 34]. There were two patients with chorioretinitis without frank vitritis (1 %), which could be explained by a low inflammatory reaction in these cases.

The 21 % frequency of definitive unilateral blindness is similar to that reported in the Netherlands [35] and is due to the macular location of the retinal lesions and retinal detachment, which is consistent with the literature [36, 37].

The most common complication found in ocular toxoplasmosis was serous retinal detachment (3.6 %) due to severe inflammatory reaction and lesions larger than the optic disc, and could be managed with anti-inflammatory agents. Other complications were vitreal bleeding and epiretinal membrane but no phthisis bulbi was found. Tractional ciliary body detachment and associated hypotony is an uncommon complication of toxoplasmosis. One case with pars plana detachment due to capsule contraction syndrome was found, which has not been reported in other studies. It has been associated with various eye diseases including pseudoexfoliation, pars planitis, low grade vitritis, high myopia, retinitis pigmentosa, and myotonic dystrophy. Elevated intraocular pressure [36] was also not noted in this study and the incidence of retinal detachment was 2.5 %. Two patients had retinal detachment and retinal tear in active ocular toxoplasmosis with severe intraocular inflammation and three patients had retinal tear after inflammation recovery.

The impact on visual acuity of toxoplasmosis infection was significant and 100 cases (51.8 %) before treatment and 136 cases (70.4 %) after treatment had good vision, which means that almost 29.6 % of cases had visual disturbances. The study showed that the visual prognosis was worse for one patient after treatment, and there were seven cases of non-rhegmatogenous retinal detachment without surgical therapy.

The development of recurrent disease in our study increased with follow-up time, being 14.5 % for those followed for at least 6 years; most attacks occurred between the ages of 15 and 45 years (mean age = 23.78). For that reason, routine examination every six months during the vulnerable age is necessary. In the literature, recurrence rates of 40–78 % were reported [11, 27, 38, 39]. Short-term treatment with antiparasitic drugs may reduce the recurrence rates [40–44].

However, the follow-up time in these studies was highly variable, which might explain the discrepancies with our data. The presence of different *T. gondii* strains associated with differences in pathogenicity and sensitivity to therapies cannot be ruled out.

To summarize, nearly one quarter of our patients with ocular toxoplasmosis developed legal blindness in at least one eye, and 14.5 % of the patients on long-term follow-up developed recurrences, which occurred predominantly in eyes with old scars.

Despite the limitations of the retrospective nature of this study, we have provided an up-to-date description of the clinical manifestations, course, and prognosis of ocular toxoplasmosis in a large series of patients. These data might be of value when advising and treating patients with this ocular infection and are crucial for future prospective studies.

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