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Toxoplasmic Chorioretinitis: Clinical and Therapeutic Aspects: About 16 Cases

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Abstract: Chorioretinitis is the most common form of uveitis of infectious origin (congenital or the incidence remains limited to about 2% of infected patients, it is the first cause of posterior uveitis in France. a series of 927 patients. The diagnosis of ocular toxoplasmosis is essentially clinical**.** The evolution is towards healing, the prognosis depends on the location of the lesions: severe in case of macular involvement and favorable in case of peripheral impairment**.** retrospective study conducted in the ophthalmology department B on a series of 16 cases over a period of 9 years (January 2008-October 2016). The average age is 23.5 years old with extremes of 12 to 35 years.It has a slight female predominance. The attack is unilateral in the majority of patients (87.5%). The average consultation time was 5 days, the decrease in visual acuity was the most frequent sign (100%), myodesopsies (50%), visual fog (25%), redness and eye pain (18%) of cases. Fundus examination revealed chorioretinal foci active in all patients (100%), scarring in 62% of cases, active hyalitis (37%) and retinal serous detachment (37%) with papilledema. in 18% of cases and finally thyndall of CA in only 6% of patients. Angiography was performed in all our patients, the toxoplasmic serology was positive in 87.5%, the PCA was performed in 02 patients with a coefficient of desmonts that is> 3 in one patient and not significant in another. 56% of patients were placed on pyrimethamine + sulfadiazine; it was replaced by azithromycin in 2 patients, sulfamethoxazol + trimethoprim in 25% and one patient under azithromycin. Corticosteroids were associated 48 hours after the start of treatment in 75% of cases. Complete healing of all foci between 4 to 6 weeks. Ocular toxoplasmosis is the leading cause of retinochoroiditis of infectious origin. It is sometimes difficult to differentiate between a congenital and acquired form, this is much more common than we thought. The diagnosis is based on the clinic for typical cases, for atypical cases and severe forms extensively (AIDS, hemopathy, immunodepression ...), the diagnosis is confirmed by the search for a local production of antitoxoplasmic AC or the search for antigens by PCR in aqueous humor or vitreous.It also poses therapeutic difficulties: heavy treatment in the long run that does not prevent frequent recurrence. The visual prognosis of toxoplasmic chorioretinitis depends on the seat and the quality of treatment; relapses are frequent during the evolution. A vaccine is being tested to prevent brain malformations and ocular lesions related to toxoplasmosis.

Keywords: Chorioretinitis, toxoplasmosis, serology, active focus, medical treatment, recurrence.

INTRODUCTION

Ocular toxoplasmosis is the leading cause of posterior uveitis of infectious origin and the most common etiology of retinochoroiditis. The infectious agent is the toxoplasma gondii[1].

The diagnosis is essentially clinical and presumed by the discovery of a lesion suggestive of the fundus: whitish and oedematous active retinochoroiditis lesions are distinguished from cicatricial, pigmented or atrophic lesions, active lesions result in contiguity, hyalitis and sometimes anterior uveitis of varying intensity. In case of doubt, the search for a local production of antitoxoplasmic antibodies in the aqueous humor taken by anterior chamber puncture can be proposed. The prognosis of the attack lies in its location on the one hand, and in its propensity for recurrence on the other hand: a macular or papillary focus will immediately affect visual acuity irreversibly, which is not the case of a peripheral focus [1-2].

The objectives of the treatment are: to reduce the duration of the inflammatory phase, to activate the chorioretinal cicatrization and to prevent possible

recurrences. These facts prompted us to undertake a retrospective study of 16 cases of toxoplasmic retinochoroiditis, we will reveal the clinical and paraclinical aspects as well as the therapeutic modalities and the visual prognosis.

Patients and methods

This is a retrospective study conducted in the ophthalmology B department of the flap specialty hospital, over a period of 9 years, from 2008 to 2016, involving 16 cases of ocular toxoplasmosis (toxoplasmic retinochoroiditis). Each patient benefited:

- Detailed interrogation specifying the reason for consultation, the epidemiological data and the antecedents.
- An ophthalmological and general clinical examination.
- And lastly, an etiological assessment, which reveals elements favoring ocular toxoplasmosis.

The diagnosis of toxoplasmic retinochoroiditis was selected according to several criteria:

- Frequency of toxoplasmosis: 1st cause of posterior infectious uveitis
- Aspect of the fundus of de eye and angiographic focus.
- Atropho-pigmented scars of the Fundus of the eye.
- Toxoplasmic serology.

RESULTS

- The average age in our series is 23.5 years with extremes of 12 to 35 years. The most affected age group is 15 to 25 years, or 63%, with the 26 to 35 age group accounting for 31%.
- 9 women for 7 men: a slight feminine predominance

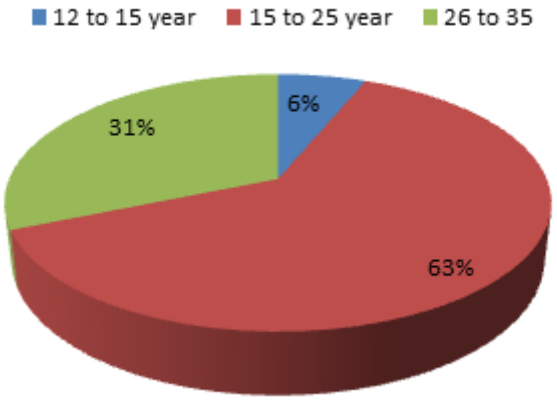

Fig-1: Patients by age group

The average consultation time is 5 days, the decrease of visual acuity was the most frequent sign: in 100% of cases, myodopsia 50%, visual 25% blur, redness and eye pain were reported in 18% of cases.

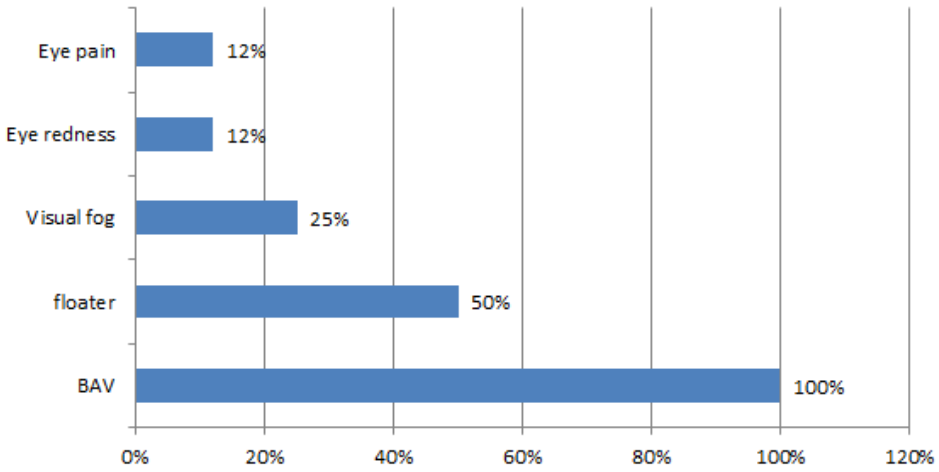

Fig-2: revealing functional signs

- Ophthalmological examination revealed unilateral involvement in the majority of patients (87.5%), the bilateral form in 12.5%.
- For initial visual acuity: 25% of patients had collapsed acuity <1/10 versus 62.5% with acuity between 2/10 and 5/10, and only 12% presented with acuity exceeding 5/10.

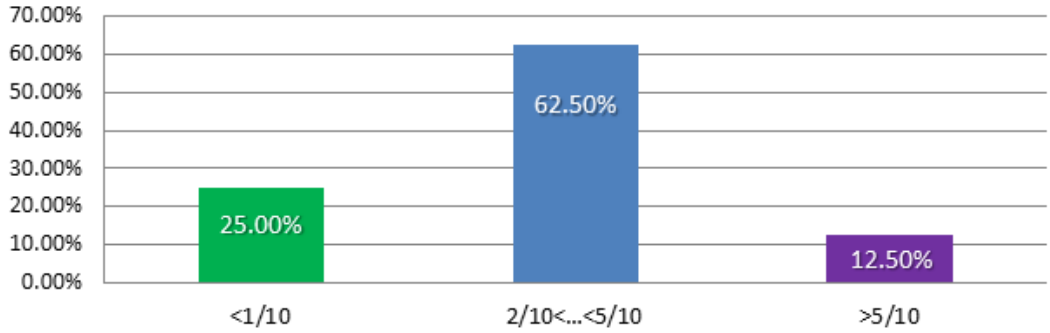

Fig-3: initial visual acuity of patients

The fundus examination found active chorioretinal foci in all patients, scar tissue in 62%, active hyalitis in retinal serous detachment in 37%, papilledema in 18%, and finally thyndall in only 6% of patients.

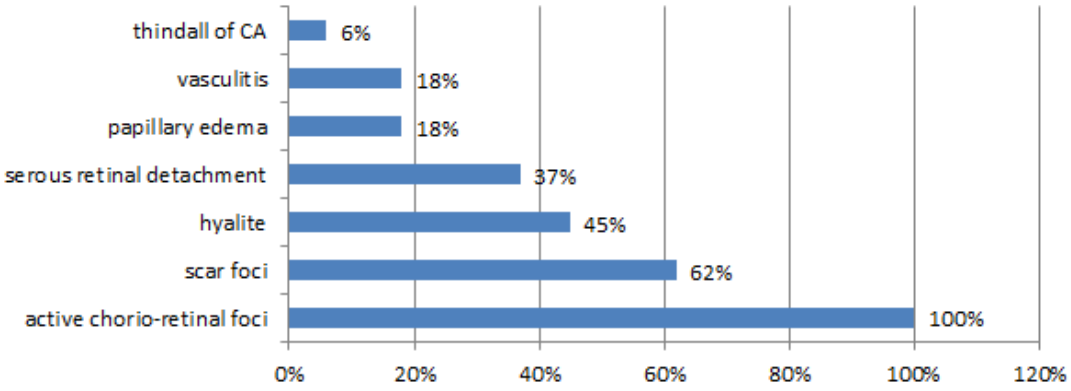

Fig-4: Data from the fundus

Angiography was performed in all our patients, it showed active lesions characterized by a hyperfluorescence beginning peripherally and progressing centripetally during the angiographic sequence, pigmented scarring lesions cause a persistent mask effect.

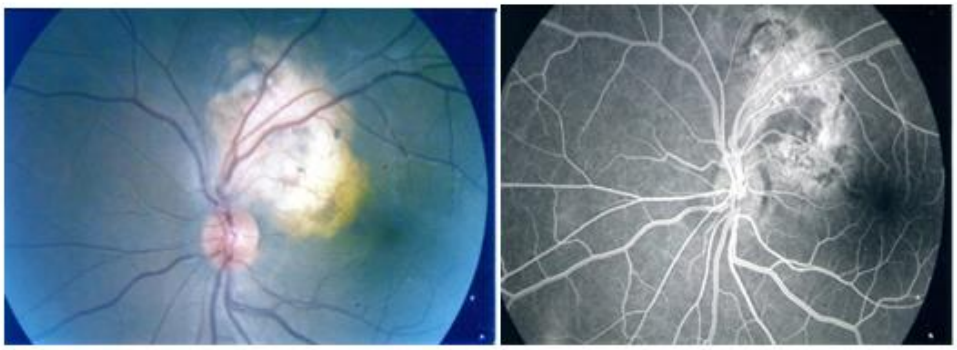

Fig-5: Large focu, juxta papillary whitish in contact with a cicatricial focus

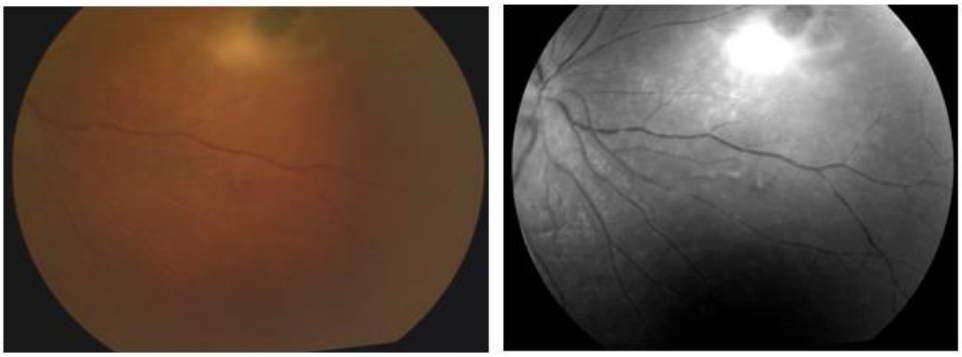

Fig-6: Peripheral active focus in contact with a cicatricial focus

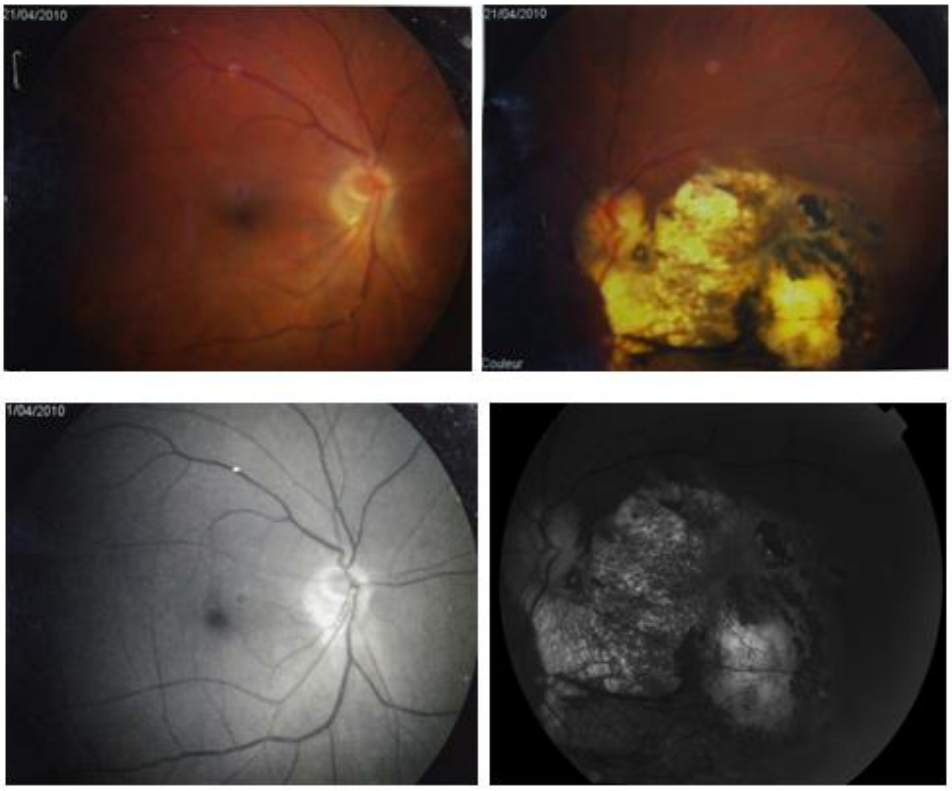

Fig-7 bilateral toxoplasmosis in a 29-year-old patient, right eye: small temporal infra-papillary focus. Left eye: cicatricial lesion of toxoplasmosis taking the whole posterior poll

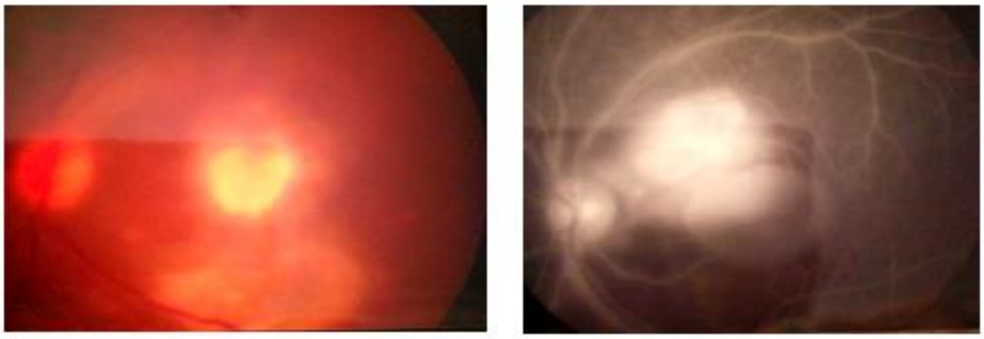

Fig-8: Active supra-macular focus associated with a large macular serous detachment in a 22-year-old patient

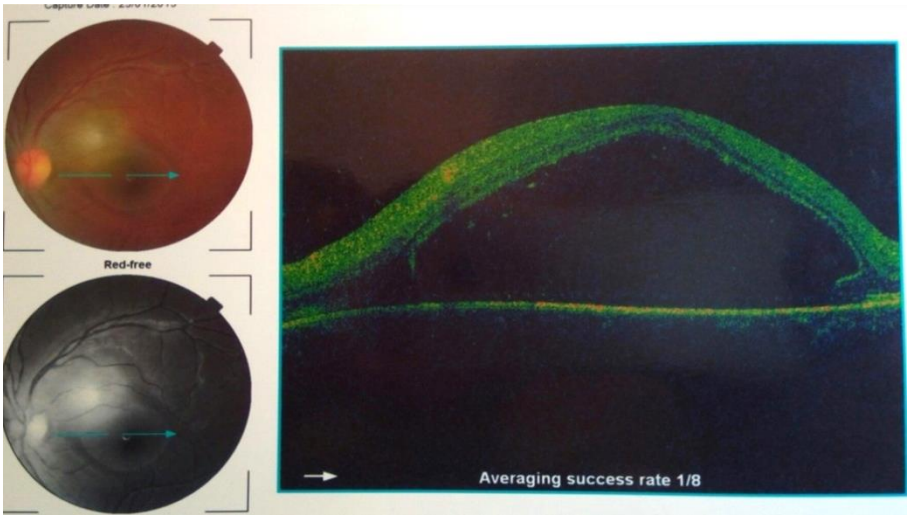

Fig-9: Aspect of serous macular detachment with OCT in the same patient

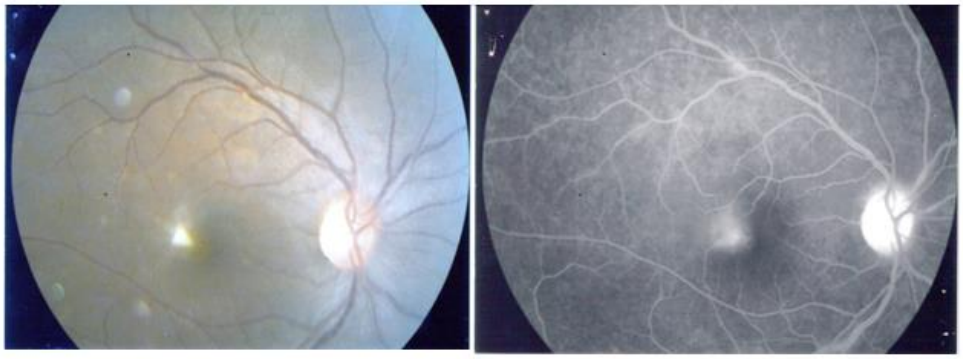

Fig-10: Small active macular focus with papillary retention at late time

Toxoplasmic serology was positive in 14 of our patients. The anterior chamber was performed in 02 patients with a coefficient of dependence that was> 3 in one patient and not significant in the other.

With the most treatment, the most patients were treated with azithromycin in 2 patients because of an allergy, 5 patients were treated with ATB alone: sulfamethoxazol trimethoprim (4 cases) and one case under azithromycin alone. Corticosteroids were administered by the general route 48 hours after the start of treatment in 12 patients or 75%. Complete healing of all patients was achieved between 4 to 6 weeks, with minimal side effects noted in only 2 patients.

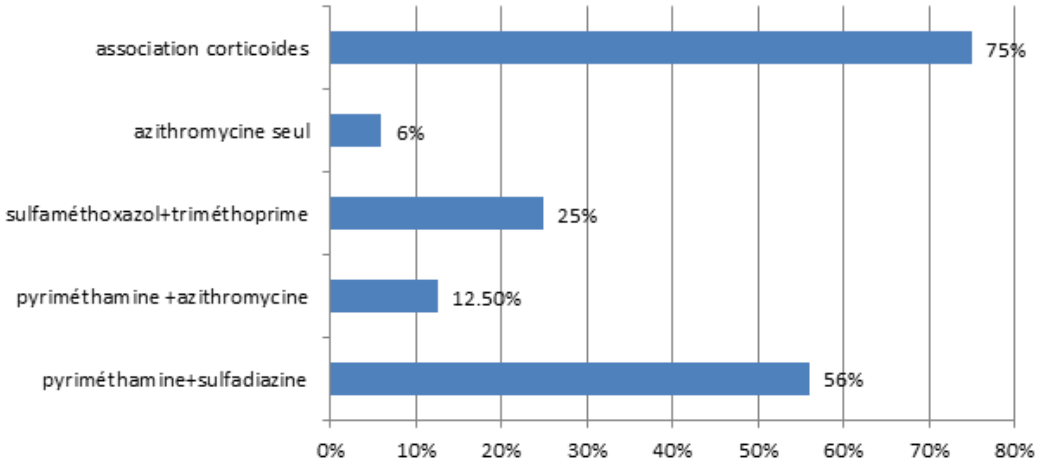

Fig-11: The different treatments used

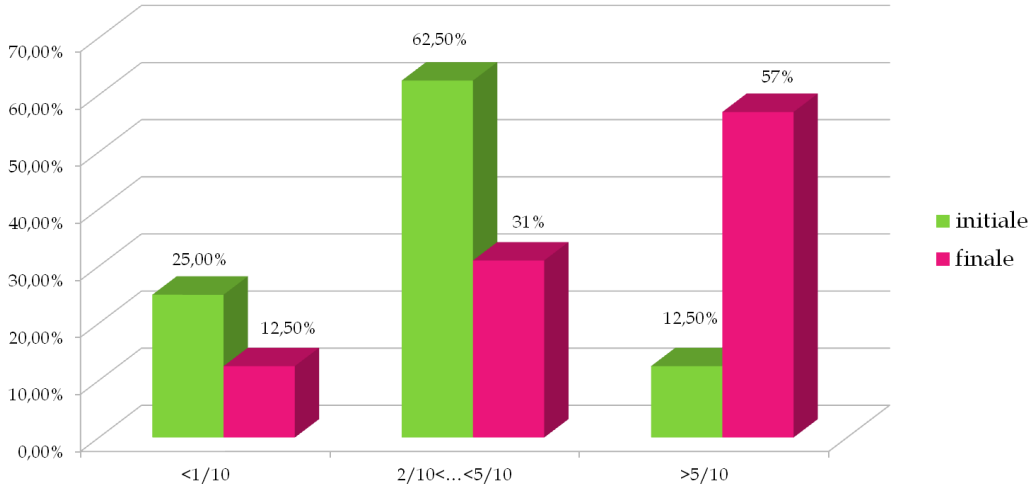

Fig-12: initial visual acuity vs final visual acuity

There was an increase in visual acuity in 10 patients or 62.5%: in the group with an Acuity less than 1/10: we went from 25% to 12.5%, in the group between 2/10 and 5/10: it went from 62% to 31% and finally 57% of the patients could have a greater than 5/10% instead of 12.5%.

With respect to evolution, there were 2 relapses after a follow-up of 18 and 24 months.

DISCUSSION

Ocular toxoplasmosis is a parazitosis toxoplasma gondii, it is a common disease, since it represents the first etiology of infectious retinochoroiditis: 30% of the world population is infected, its incidence is very high in some countries: in Brazil, its incidence is greater than 20% with 1 in 100 people with unilateral blindness, and 1 in 500 people with bilateral blindness. Its seroprevalence is variable: in Brazil 70%, in France 50%, in the United States 20%. In our context, it represented 19.7% of all infectious retinochoroiditis[1].

It occurs in the vast majority of cases following the reactivation of an outbreak of congenital toxoplasmosis, according to a European study reported by kope and kloostermann, 70% of neonates with congenital infection show chorioretinitis scars compatible with toxoplasmosis after a follow-up of 16 years, with 1 to 2% who had severe ocular involvement. The acquired form should not be discarded, it is much more frequent than thought, and this is demonstrated during a toxoplasmosis epidemic in Canada by drinking water contamination: we had 20% of ocular lesions associated systemic signs of toxoplasmosis. in our series, it was a congenital form reactivation in (14 cases) or 88%, whereas the acquired form is represented by only 2 cases with absence of cicatricial foci and presence of IgM ++ antibodies[1-3].

Congenital toxoplasmosis, severe for the fetus, ocular manifestations can shift sometimes several years after maternofetal infection. Acquired toxoplasmosis: often subclinical, the chorioretinal focus is unique, of variable size, located in the middle periphery often accompanied by a medium vitreous disorder.

The toxoplasmosis of the immunocompromised is responsible for only 1 to 2% of ocular infections, these foci can appear either isolated or in the vicinity of scarred chorioretinal lesions, or in the form of a fresh granuloma [4].

For the typical forms of toxoplasmic retinochoroiditis, the diagnosis is mainly clinical and angiographic, the serology lacks specificity but its negativation completely eliminates the diagnosis of toxoplasmosis, it also confirms a primary infection with high levels of IgM and IGAs detectable 7 days after infection, or by doubling IgG at 3 weeks intervals. In atypical forms, such as bilateral, multifocal, extensive necrotizing retinitis, or active focus without adjacent wound scars, the diagnosis can be confirmed by finding local antibody production in the aqueous humor by calculating the Desmont which corresponds to the ratio IgG anti-Toxoplasma gondi / total IgG in the aqueous humor on the ratio IgG anti-Toxoplasma gondi / total IgG in the blood when this ratio is greater than 3. When this ratio is less than 2, the production antibody is not demonstrated, without ocular toxoplasmosis being eliminated. A value between 2 and 3 of the Desmonts coefficient is doubtful for asserting a local production of antibodies **(5)** In difficult cases, Toxoplasma Gondi antigens can be detected by PCR in the aqueous humor or vitreous, this search is positive in 30% of ocular toxoplasmoses with a positive coefficient of desmonts[5, 6].

Disseminated severe form is mostly described in immunocompromised patients (with AIDS, hematologic malignancies or iatrogenic). In

immunocompetent patients, this form is relatively rare, there are less than 50 documented cases in the literature, its severity is especially related to the age of the host and the virulence of the strain of toxoplasma gondi.

In our series, only one case of severe toxoplasmosis in an immunocompromised patient has been reported with active, multiple, and peripheral lesions.

Regarding the treatment, it is necessary to know if the injury induced by Toxoplasma requires treatment or not based on the following criteria:

- An injury in the temporal arch.
- A lesion reaching the optic nerve or threatening a large retinal vessel.
- A lesion causing significant bleeding.

A lesion inducing a significant inflammatory vitreous response with a decrease in visual acuity (two lines minimum)[7].

A relative indication may be the case of multiple recurrences that develop vitreous condensation that can lead to retinal detachment.

The treatment of ocular toxoplasmosis is based on a dual therapy: pyrimethamine (0.5 to 10 mg/kg/ day) and Sulfadiasine (50 to 80 mg / kg / day), combined with folate supplementation, sulfadiazine may be replaced by clindamycin if allergy, or azithromycin.

The marketed combination sulfamethoxazol + trimethoprim is used by some practitioners because it has shown efficacy comparable to that of antiparazitics. In our series, 25% of patients received this treatment with a favorable evolution.

Subconjunctival injections of clindamycin have also been approved for the treatment of ocular toxoplosmosis, avoiding all the undesirable effects of systemic treatment, namely sd lyell, agranilocytosis, pseudomembranous colitis [7, 8].

Spiramycin has also shown its effectiveness in the treatment of infantile forms. A general corticotherapy administered 48 hours after the treatment has for essential purpose, the limitation of the vitreous or perilesional inflammatory reaction. The duration of the treatment must be according to the evolution. On average, the healing time of an active outbreak is about 3 to 4 weeks.

Of all the molecules available for the treatment of ocular toxoplasmosis, none is able to act beyond the acute thrust, and no treatment has been able to prevent recurrence despite the hopefuls for atovaquone, which has shown in vitro a clear efficacy against encysted forms compared to other molecules, but this result is not confirmed in humans**[**9].

Regarding the evolution, several studies were interested in the different therapeutic protocols and their recurrence rate, these rates are difficult to compare because of differences in duration of follow-up, nevertheless, clindamycin is orally or subconjunctivally , seems to give the best therapeutic results[10, 11].

Study	Molecul	Recidivism Rate	Duration Of Treatment
Canamucio *et al*	pyriméthamine +sulfadiazine	13%	1-28 months
Rhotova *et al*	pyrithémanie +azithromycine	27%	20 months
Lakhampal *et al*	clindamycine orally	7,70%	3 years
Jeddi *et al*	clindamycine in sub conjunctive	6%	8 à 24 months
colin and harie	clindamycine in sub conjunctive	7%	2-24months
Our series	pyriméthamine + sulfadiazine	12,50%	3-24months

Comparative table of different therapeutic protocols and their recurrence rates according to lliterature

The visual prognosis of the disease depends on the location of the attack as well as the quality of the treatment:

- Central forms: macular involvement or recurrence, with a risk of permanent loss of central vision
- Juxtapapillary forms: focus near the papilla creates a crescent-shaped campimetric deficit.

CONCLUSION

Ocular toxoplasmosis is a condition that threatens visual acuity. The localization of the lesions is the essential determinant of the therapeutic decision, since it is a heavy enamelled treatment of several side effects of variable severity.

Relapses are frequent, estimated 2 to 3 times during the life of an infected person: 2.7 episodes during the evolution of the disease.

Recently, a vaccine against ocular toxoplasmosis was developed by Vitam Fero, it is being patented. This vaccine has already shown its effectiveness in the prevention of congenital malformations due to toxoplasmosis in the animal species tested.

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