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Ocular Toxoplasmosis: Diagnosis and Management in low resource country – A case Series

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Abstract

Ocular Toxoplasmosis is the commonest cause for infective posterior Uveitis. It is potentially a blinding disease with a progressive and relapsing course. The incidence of ocular infection is very high, most of which are subclinical. Hence its important to highlight this blinding disease, which may escape detection and to emphasize its preventive measures so as to prevent the visual disability arising from it .Though PCR of vitreous fluid may be confirmatory but in Low resource countrylikeNepalwheremanyofthecentreshasverylimitedornoaccesstoPCR test ,Clinical Finding and Ancillary tests like OCT is of great help to come to a diagnosis supplement by Toxoplasma Serology. We are describing few straightforward and few challenging cases of Ocular toxoplasma.

Keywords: Toxoplasmosis, OCT, Retinochoroiditis, Posterior Uveitis.

Introduction

Ocular Toxoplasmosis represents the most common cause of infectious retinochoroiditis in children and adult. It is caused by the obligate intracellular parasite Toxoplasma gondii.1 The typical presentation of ocular toxoplasmosis is characterized by focalretinitis adjacent to ahealedchorioretinal scar and vitreous inflammation.2 In addition to this , atypical form of Ocular toxoplasmosis may exits in the form of retinal Vasculitis, Punctate Outer Retinal Toxoplasmosis(PORT),Optic neuropathy, Retinal Detachment and Scleritis.3Classic Clinical findings ,ancillary test like Optical Coherence Tomography (OCT) through lesion and Serology (IgM/IgG) are the most important triad used to diagnose Ocular Toxoplasma as there is almost poor access to Ocular fluid PCR which is almost hundred percent specificity for Ocular

toxoplasma.4 Here I have described three cases with different perspectives.

Case 1: (Clinically looking toxoplasma with positive Serology)

16 years old female ,with history of mild Blurring of Vision RE for 3 weeks .She is Otherwise healthy with no history of contact with pets. Her Left Eye was normal and RE Unaided visual acuity RE 6/24 and corrected to 6/6 with refraction. Anterior Segment showed Fine diffuse non granulomatous Keratic precipitates with3+Cellsin Anterior chamber. Vitreous show2+Haze.Fundus Examination(Fig 1a) show Focal Retinitis of approximately 1 DD above and adjacent to the optic disc (jensen's jaxtapapillary retinochoroiditis) with overlying vitritis and arteritis kyrieleis.

OCT (Fig 1b) through the lesion shows Vitritis, Detached and Taught Posterior Hyaloid, Cells beneath post hyaloids (often termedas posterior Keratic Precipitates) Full Thickness Retinal involvement and Choroidal Elevation.

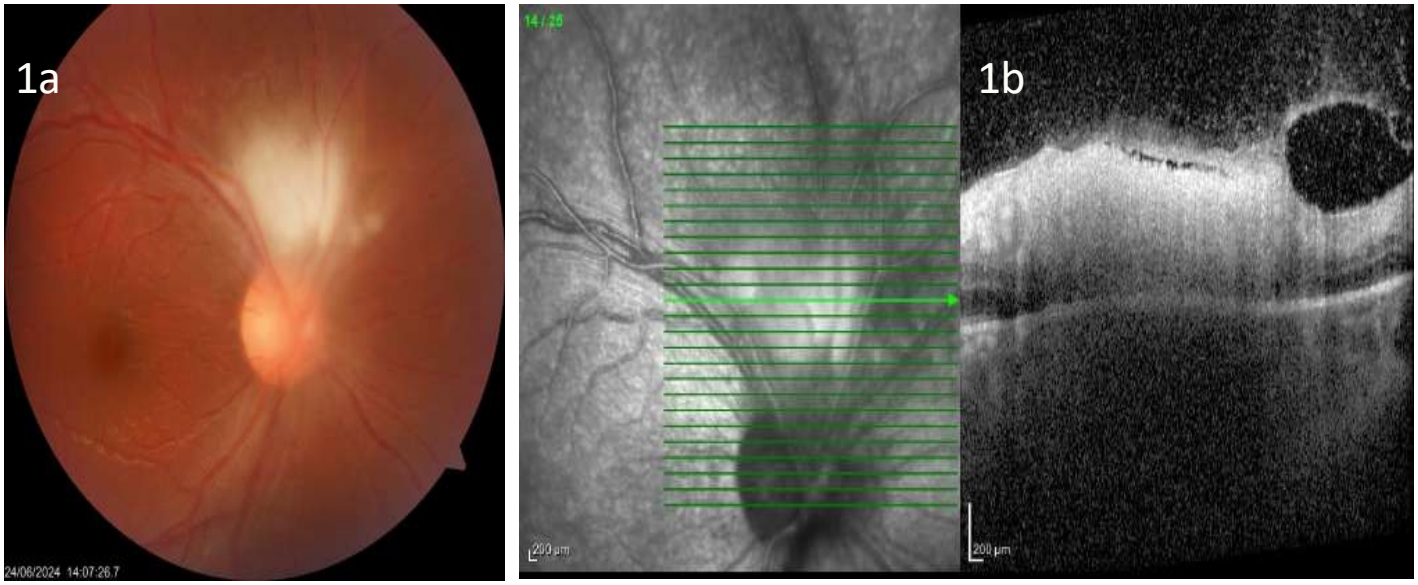

Fig1a: typical Jenson's jaxta papillary retinochoroid it is lesion and Fig1b: OCT through the lesion. Note the detached posterior hyaloid, full thickness retinitis and choroidal elevation suggestive of active retinochoroiditis

Lab investigation Toxoplasma IgM was negative where Toxoplasma IgG was positive with high titre(179.6 IU/ml) .

Patient was Treated with Intravitreal Clindamycin with Dexamethasone as the lesion was near to optic disc , Oral Trimethoprim 160 mg +Sufamethoxazole 800 mg combination (TMP-SMX) for 6 weeks with with oral steroid 1mg/kg from 3rd day. On just 2 week follow up there was remarkable improvement in Anterior chamber inflammation and marked healing of the lesion (Fig 1c)

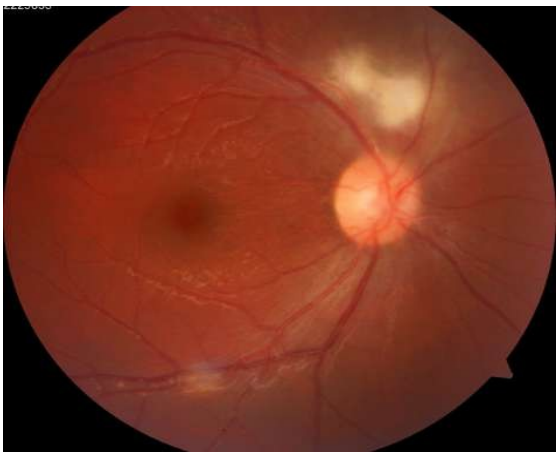

Fig1c: Healing of active retinochoroiditis with resolving over lyingvitritis

Case 2 (Clinically looking Toxoplasma but NegativeToxoplasma Serology)

17 year /Male, otherwise healthy presented with Blurring of vision in RE 15 days. His Visual Acuity RE was Hand Motion Close to Face no and normal LE. Right Eye was Non Congested ,clear cornea, with 2+ cells in Anterior chamber.Vitreoushaze3+.Fund us showed Focal Retinitis of half disc diameter size with overlying vitritis giving Classic Headlight in Fog appliance (Fig 2a) above opticdisc.OCT through Lesion shows detached posterior hyaloid and full thickness Retinitis.

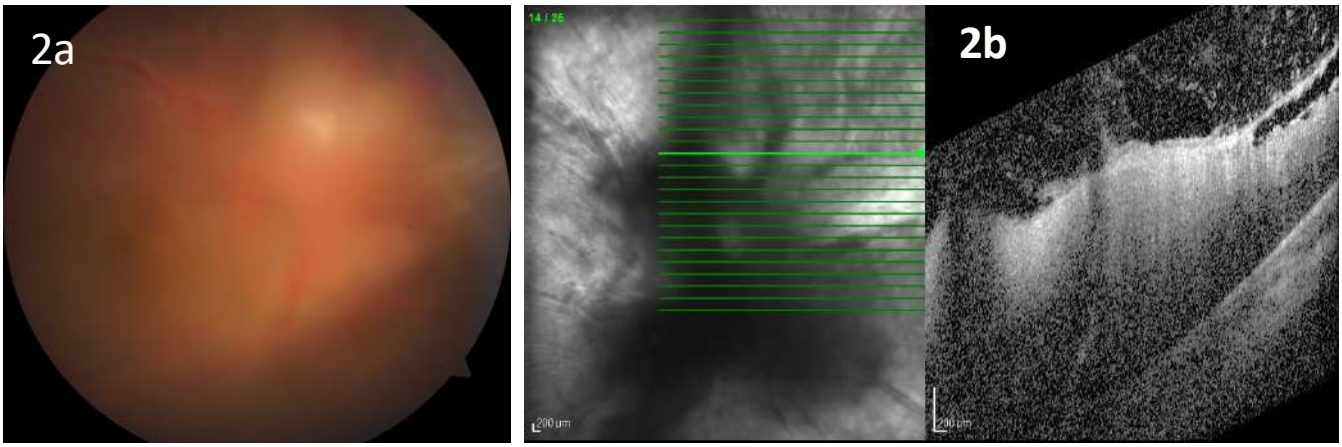

Fig 2a: typical lretinitis lesion and Fig 2b:OCT through the lesion. Note the detached posterior hyaloid, full thickness retinitis

Lab investigation showed Toxoplasma IgM negative and surprisingly Toxoplasma IgG was also negative Using chemiluminescence Immunoassay (CLIA)

As we always believe that Clinical phenotype is more important than Lab so we treated with Antitoxoplasma with same regimen as in our first case with Additionally Intravitral Clindamycin and Dexamethasone for this case. There was gradual clearing of vitritis and restoration of almost normal vision In subsequent follow up visit .Vision improved to 6/9 from HM at presentation just within three weeks time (Fig 2c, 2d)

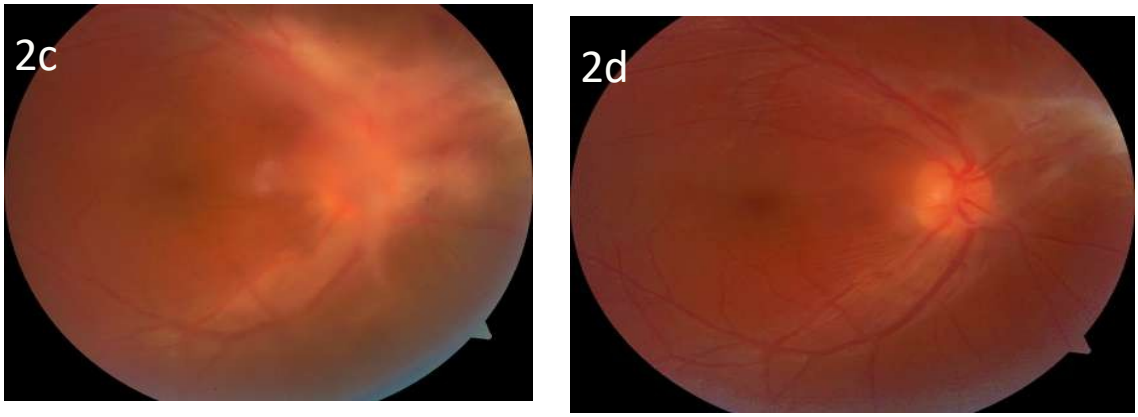

Fig 2c & 2d: Gradual clearing of vitritis with better visualization of retina and resolution of retinitis.

Case3:Clinically doesn't look like to xoplasma butturned out to be toxoplasma (Atypical Toxoplasmosis)

23 y/ otherwise healthy female presented with sudden blurring of vision LE for six days. Her Right eye washad 6/6 vision with normal anterior and posterior segment.

LE was non congested with,Visual acuity 6/18 no improvement with refraction, granulomatous Keratic precipitates, AC cells 3+ ,Vitreous haze 1+.

Fundus picture showed generalized sheathing of major retinal vessels, Roth spots and Focal white Patch like cotton wool spot of approximately 1 Disc diameter along the superior arcade. (Fig 3a).

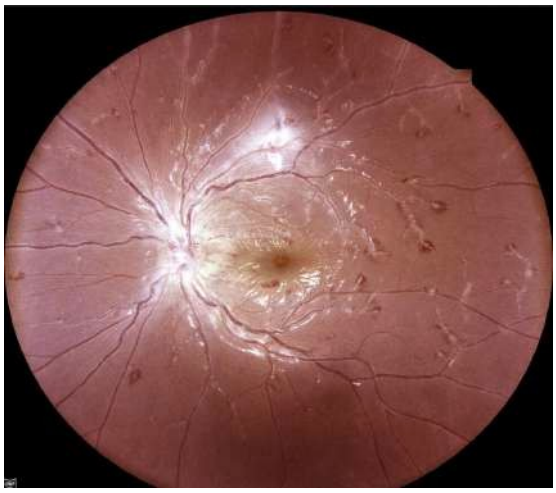

Fig 3a: Sheathing of major retinal vessel, Roth Spotand Whitish lesion in superotemporalarcade

With these findings we kept systemic vasculitis, HIV and Blood Dyscrasias as differentials and sent investigation for the same but all investigations were negative .After 1 week of Intense topical steroid her inflammation didn't reduce. Wethendid OCT through thelesion (Fig3b) which gave more cluestoward toxoplasmosis.

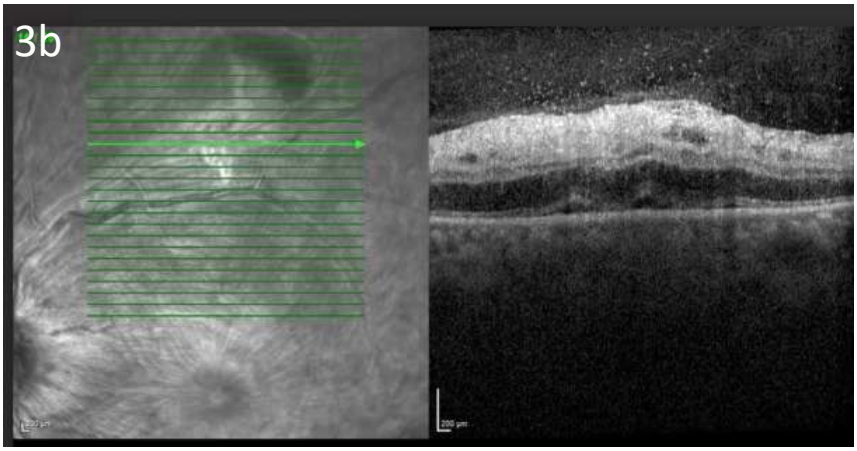

Fig 3b: OCT through lesion shows Focalvitritis and full thickness necrotisingretinitis

Toxoplasma serology was sent which surprisingly showed very high titre of IgG.Intravitreal Clindamycin with dexamethasone was injected and patient was started on oral Antitoxoplasma and oral steroid in the same way as our previous case 1 & 2.Within 1 week allvasculitis and Roth spots resolved completely and the whitish focal lesion gradually resolved with improvement in vision and anterior segment inflammation too in third (Fig 3c) and Fourth week (Fig 3d)

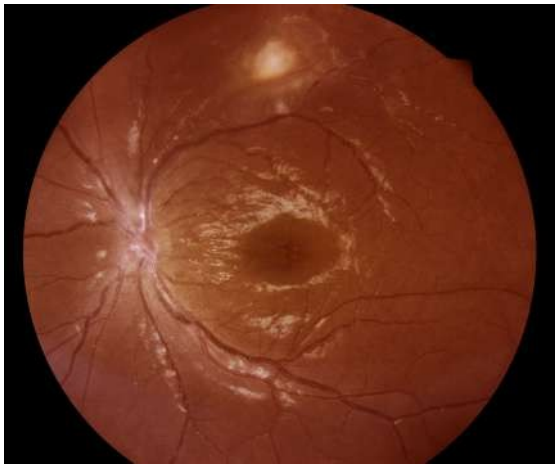

Fig 3c: Healing lesion after1week of treatment

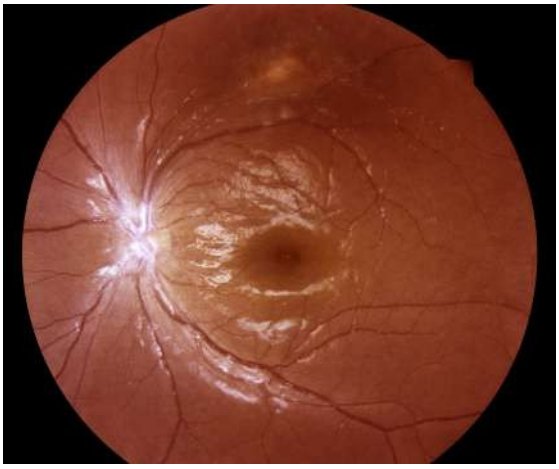

Fig 3d: Healed lesion after 3 week of treatment

Discussion

The diagnosis of ocular toxoplasmosis can be challenging. Clinical exam, serology and PCR are frequently used to diagnose suspected ocular toxoplasmosis.[5] In low resource country like Nepal, most of the time we have to depend only on Clinical findings as OCT and PCR facilities are not available in many centers. During acute or active phase, differential diagnosis with other types of infectious (toxocariasis, viral-induced necrotizing retinopathies, syphilis, diffuse unilateral subacute neuroretinitis, and endophthalmitis), non infectious (Behçet's disease, multifocal choroiditis, panuveitis, serpiginouschoroiditis, punctuate inner choroidopathy) and ocular conditions (primary intraocular lymphoma) is necessary.[6] OCT is a great non invasive tool to differentiate among such confusing differentials . Severe vitritis over the lesion, thickened and detached posterior hyoid ,Hyper-reflective dots underneath the detached hyaloid (termed as posterior Keratic precipitates), Full thickness retinal involvement, precipitate

along Internal limiting membrane causing mirror image, stalagmite like protrusion from ILM, Choroidal elevation and back showing in choroid are the major clues in OCT.[7]

In our first and second case we had almost classical lesion of toxoplasmosis though there was no severe vitritis like headlight in fog appearance and nearby old healed lesion in first case. The first case is often described as Jensen's jaxtapapillary retinochoroiditis .This type of presentation is well described by Young hun park et al.[8]

In our Second case though the lesion and finding were of typical Toxoplasmosis but negative IgG toxoplasma serology leads to a dilemma . Monias et al[9] has described a case of seronegative Ocular toxoplasma inimmunocompetent patient where they have mentioned that serum IgG will be peak on eighth week of infection .The possible Hypothetical explanation for IgG negativity of our case might me that the test was performed before eight week whereas in their case it was performed twice twelve months apart but still was negative.

Our third casehad very confusing clinical picture which falls in category of A typical Toxoplasmosis. Immuno competent status, generalized retinal Vasculitis made us to think on the line of systemic vasculitis but all the systemic vasculitis work-up was negative. Multiple Roth's spot arrowed us toward possible blood dyscrasias and the work up was also negative . Normal ESR and OCT through the focal lesion finally directed us one step close to Ocular Toxoplasmosis.

we empirically injected intravitral clindamyc in without dexamethasone ,as we were still not sure about etiology and ordered IgG too .The positive response to intravitral Clindamycin and very high titre of IgG Toxoplasma almost confirm the diagnosis.

In all our three cases we treated with Classical Triple therapy (Sulfamethoxazole 800 mg+ Trimethoprim 160 mg combination) for 6 week and oral steroid 1 mg/kg body weight 24-48 hour after starting anti toxoplasma therapy as described by Kengadhevietal.[10] We injected intravitral Clindamycin with dexamethas one in our first two cases as the first was near to optic disc and second was in macula (Zone 1) as described by Seyedeh et al.[11] Dexamethas one limits the inflammation which is equally important to prevent retinal complications .In our Third case of atypical ocular toxoplasmosis we inject only clindamycin intravitreally without dexamethas one as we were not sure about other infective entity of retinitis like viral or fungal. than toxoplasma .

Conclusion

Ocular toxoplasmosisis that commonest form of infectious posterior Uveitis. Typical Phenotype and Noninvasive test like OCT is almost confirm to diagnosis. Positive toxoplasma serology is supportive but Negative serology doest exclude infection .There may be atypical cases which may create dilemma where PCR of ocular fluidis helpful.

Source of Support

Nil

Conflict of Interest

None

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