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Ophthalmology

An Interesting Case of Acute Chorioretinitis Due to Ocular Toxoplasmosis

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Abstract **Case Report**

Ocular Toxoplasmosis is a recurrent and progressive necrotizing retinitis that can cause blindness. In most cases, it manifests as a localised retinochoroidal lesion and is the most prevalent cause of posterior uveitis worldwide. Ocular infection is relatively common, with the majority of cases being asymptomatic. As a result, it's important to draw attention to this blinding disease that might go undetected, as well as the preventative actions that can be taken to avoid vision impairment. We report a case of a 35 years old female came with complaints of defective vision in both eyes for past 2 months which was acute in onset and painless in nature. There was history of similar episode 1 year back with no history of any treatment taken and there was a history of eating undercooked meat in the past. Right eye fundus showed a typical headlight in the fog appearance and left eye fundus showed exudative sheathing of vessel wall and multiple pigmented old retinochoroidal scars. Blood investigation showed elevated total count and an elevated ESR level. Peripheral smear report suggested dimorphic anemia and thrombocytosis. Antinuclear antobody-positive.Toxoplasm igG antibody- 511.90IU/ml (positive for toxoplasmosis).

Key words: Toxoplasmosis, retinochoroidal, exudate, sheathing.

INTRODUCTION

Ocular toxoplasmosis is a recurrent and progressive necrotizing retinitis that can cause blindness. In most cases, it manifests as a localised retinochoroiditis [1]. World-wide, it is the most common cause of posterior uveitis [2]. It's a zoonotic illness, with the cat serving as the definitive host and humans and other animals serving as intermediate hosts. Toxoplasmosis is a worldwide zoonosis caused by Toxoplasma gondii, parasitic protozoa that are a necessary parasite of cats. Infection is spread through the consumption of oocysts found in cat faeces or bradyzoits found in undercooked meat. It is normally a benign self-limited infection in an immunocompetent host; nevertheless, severe consequences, such as retinochoroidal involvement, have been observed to occur.

CASE REPORT

A 35 years old female came with presenting complaints of defective vision in both eyes (Right eye more than left eye) for past two months. Diminished vision was acute in onset painless in nature. The patient had a similar episode 1 year back but no treatment was done for the previous episode. The patient gave history of frequent eating of undercooked meat. The patient has no history of diabetes, hypertension and tuberculosis. There was no history of any contact with pets or stray animals. Cardiovascular system showed no abnormality, S1 and S2 were normal, no murmurs were noted. Respiratory system examination elicited normal vesicular breath sounds. Abdomen was soft and no organomegaly was noted. No neurological deficiets noted.

Ocular examination is as follows:

Table-1: Visual acuity

	Right eye	Left eye
Vision	1/60	6/24
Vision with pinhole	NIP	6/12

Head posture: Normal
Facial symmetry: normal
Primary gaze position: both eye normal

Table-2: Anterior segment findings

	Right eye	**Left eye**
LID	Normal	Normal
CONJUNCTIVA	Normal	Normal
CORNEA	Clear	Clear
ANTERIOR CHAMBER	Normal depth, No cells, No flares	Normal depth, No cells, No flares
IRIS	Normal colour and pattern	Normal colour and pattern
PUPIL	3mm, Round, Regular, Direct and Indirect light reflex +	3mm, Round, Regular, Direct and Indirect light reflex +
LENS	Clear	Clear

Table-3: Fundus findings

	Right eye	**Left eye**
Fundus	• Media- Hazy suggestive of vitritis. Vitreous inflammatory strands +. • Disc- normal • Vessels - Peripheral vascular tortuosity + • A yellowish white raised lesion with fluffy margin obscuring the underlying vessels superotemporal to disc typically giving **headlight in the fog** appearance suggestive of active retinochoroiditis lesion. • Along the peripheral superotemporal arcade multifold pigmented R-C scar present. • Hard exudates around macula • FR dull	• Media-Minimal haze+ • Vitreous strands at the periphery. • Disc-Normal • Vessels-Exudative sheathing of arterial wall focally at the superotemporal arcade. • Multiple pigmented old retinochoroidal scar present in the peripheral arcades. • Macula-FR dull

Fundus picture showed yellowish white raised lesion with fluffy margin obscuring the underlying vessels superotemporal to disc typically giving head light in fog appearance.

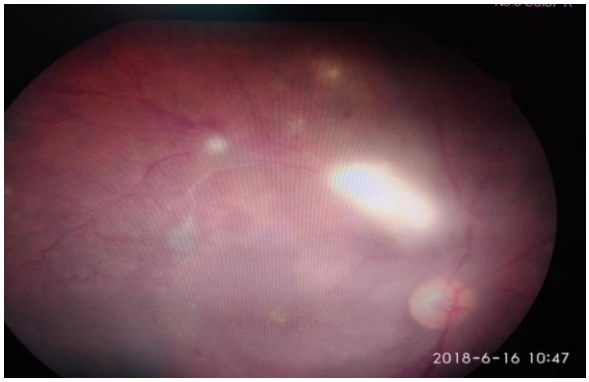


Fig-1: Right eye

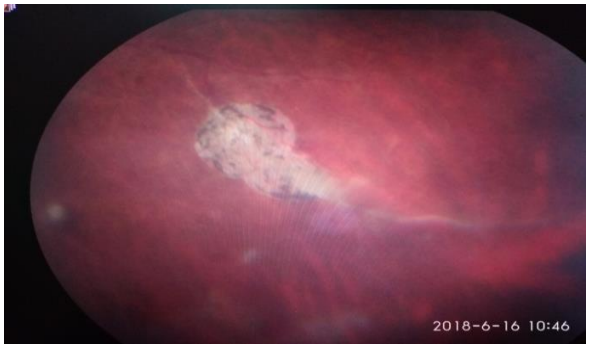


Fig-2: Right eye

Fig-3: Left eye

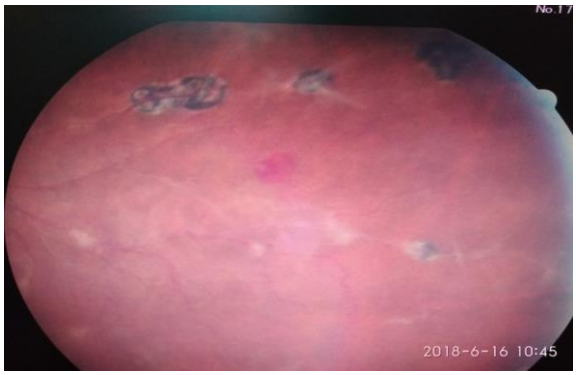


Fig-4: Left eye

Investigation
CBC-Hb-10.00gm%
Total count-54,000cells/mm cube
DC-N86%, L10%, E04%

Platelets-2, 87, 000cells/mm cube
ESR-Elevated
Chest Xray- Normal
SEROLOGY-VDRL-Non-Reactive
HBsAg-Negative
HIV-Non-Reactive
RA factor-Negative
CRP-Negative

Peripheral smear report-suggestive of dimorphic anemia and Thrombocytosis

Anti-nuclear Antibody-Positive

Toxoplasma IgG antibody-511.90IU/ml (Positive for Toxoplasma) (Normal value<9 IU/ml)

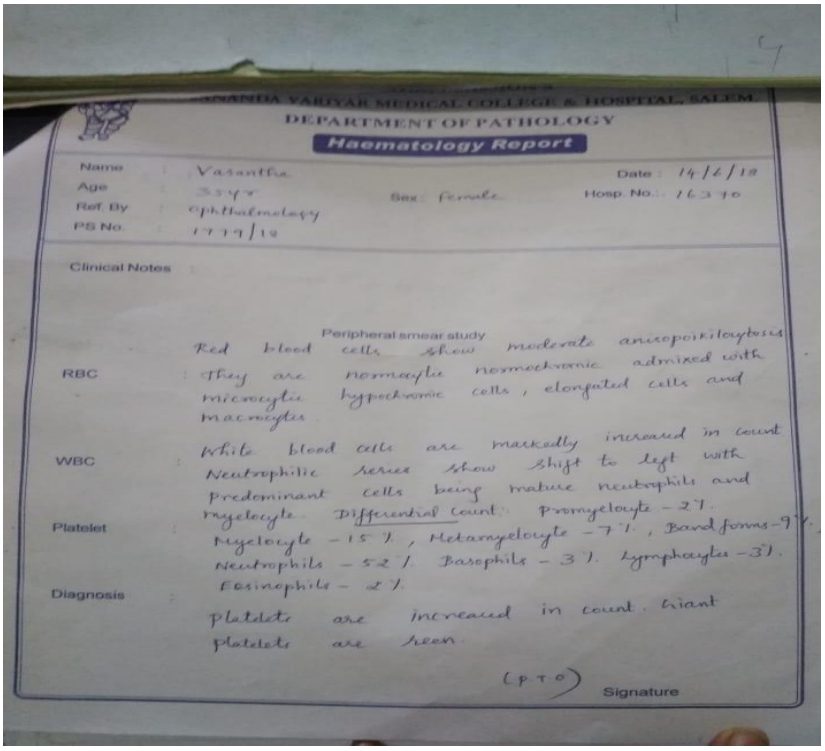


Fig-5: Hematology report

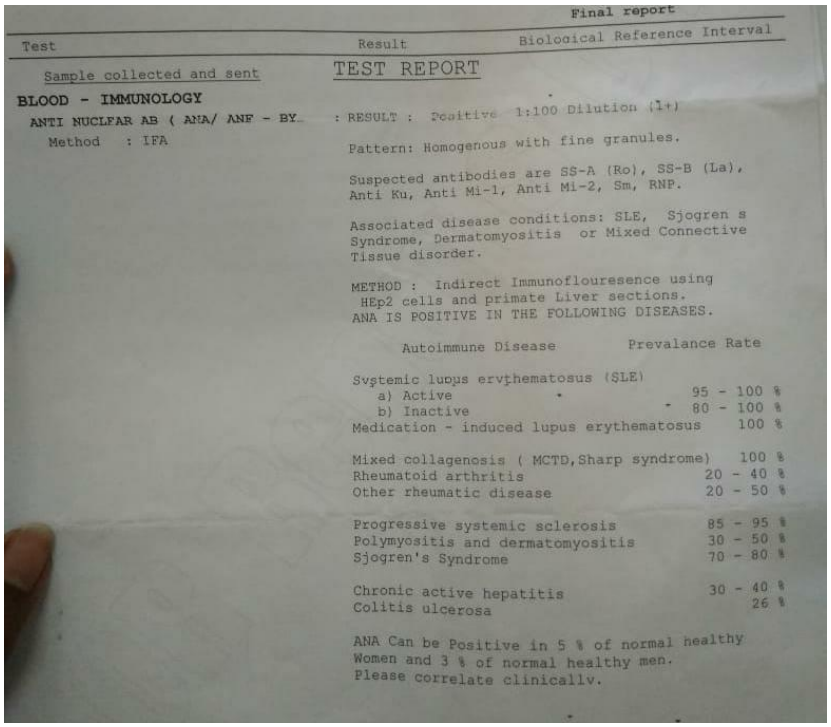
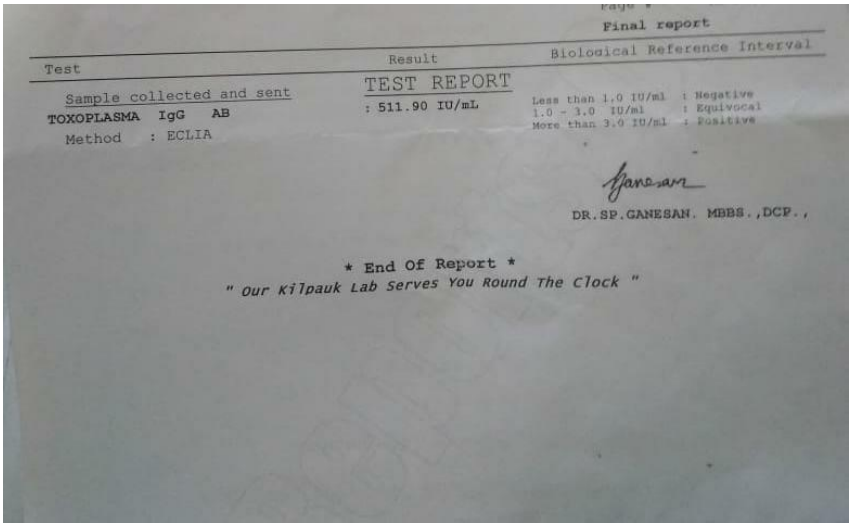


Fig-6: Blood immunology report


Final report

Test	Result	Biological Reference Interval
Sample collected and sent	TEST REPORT	
TOXOPLASMA IgG AB	: 511.90 IU/mL	Less than 1.0 IU/ml : Negative 1.0 - 3.0 IU/ml : Equivocal More than 3.0 IU/ml : Positive
Method : ECLIA		

DR.SP.GANESAN. MBBS.,DCP.,

* End Of Report *

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Fig-7: Toxoplasma IgG AB report

Treatment given:
Patient was started on
1.Tab.Trimethoprim 200mg QID
2.Tab.Sulphadiazine 1g QID
3.Tab.Folvite 5mg BD
4.Tab.B Complex BD
5.Tab.Wysolone 50mg OD started 2 days later and tapered over one month.
6.Tab.Rantac 150mg BD

FOLLOW UP

Patient lesion activity decreased and visual acuity improved after 3 months follow up.

DISCUSSION

Ocular toxoplasmosis is a a recurrent retinochoroiditis caused by the organism Toxoplasma gondii. It represents the most common cause of infectious posterior uveitis worldwide. A unilateral decrease in visual acuity in the most common symptom. A unifocal area of acute onset inflammation adjacent to old chorioretinal scar is pathognomic. It typically presents with quiescent, chorioretinal scars in the inactive phase and a necrotising chorioretinitis with overlying vitritis. (Headlight in the fog) in the active phase.With acquired toxoplasmosis, unilateral lesions are more common. Congenital disease, on the other hand, affects three-quarters of individuals and is more likely to affect the macular region. When the lesion is distinct, the presence of IgG serum antibodies [5] gives a presumptive diagnosis and allows initiation of specific therapy. In doubtful cases, it is possible to detect the parasite DNA by vitreous puncture and PCR for T. gondii. The severe vitritis gives a "headlight in the fog" appearance [4]. The choroid and sclera may become involved secondarily [2]. Anterior uveitis is due to hypersensitivity reaction to the antigen. Sheathing of the retinal vasculature, vascular occlusions and periarterial exudates (kyrieleis arterialitis) at or away from the foci of retinitis may be seen. In healthy patients, the retinitis heals within 1-4 months of treatment and is replaced with a sharply demarcated atrophic scar with pigmented borders as seen in our case. Complications of ocular toxoplasmosis include cataract, secondary glaucoma, band keratopathy, vascular occlusions, scleritis, retinal gliosis, tractional retinal detachment, cystoid macular edema, macular pucker, optic atrophy and choroidal neovascular membrane [3]. Anti-toxoplasma IgG antibodies can persist at high titers for years after acute infection and there is a high prevalence of such antibodies in the general population giving false positive results [5]. The classical "triple drug therapy" with pyrimethamine, sulphadiazine and prednisolone is reserved for lesions involving the macula and optic nerve head, in large destructive lesions, severe vitritis and in any lesion in AIDS patients. Some studies have shown that prophylactic treatment for ocular toxoplasmosis in immunocompetent patients reduces the chances of recurrence [6]. Furthermore, prophylactic treatment is recommended in all patients with inactive toxoplasmic retinochoroiditis undergoing cataract surgery [7]. Since ocular toxoplasmosis is a potentially blinding disease with recurrences, preventive measures should be taken to avoid it.

CONCLUSION

In the present case patient had consumption of undercooked meat which is a cause for ocular toxoplasmosis. Fundus finding showd the typical headlight in the fog appearance suggestive of active retinochoroiditis lesion. A unifocal area of acute onset inflammation adjacent to old chorioretinitis scar was present which is pathognomic of ocular toxoplasmosis. Patient was started on triple drug therapy of trimethoprim, sulphadiazine and corticosteroid and there was a decrease in lesion activity.

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