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CLINICAL PROFILE OF PATIENTS OF OCULAR TOXOPLASMOSIS PRESENTED TO THE TERTIARY HEALTH CARE CENTRE IN BUNDELKHAND REGION

Ophthalmology

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ABSTRACT

Purpose: The aim of this study was to provide a retrospective analysis of the clinical profile of patients of ocular toxoplasmosis in department of Ophthalmology at M.L.B. Medial College, Jhansi. **Materials And Methods:** Retrospective study of 30 patients with 38 eyes of Ocular Toxoplasmosis presented to us in the Department of Ophthalmology at M.L.B. Medial College, Jhansi. **Results:** A total of 38 eyes of 30 patients were studied in which mean age of presentation was 40 years with 8(26.6%) patients having bilateral presentation and rest 22(73.33%) with unilateral presentation. 70% of patients were male and rest 30% female. 44.7% of males and 15.7% had chronic retinochoroiditic scar. 47.5% patient had peripheral scar while 18.4% had macular scar. **Conclusion:** Majority of patients were male with unilateral involvement and peripheral chronic retinochoroiditic scar with hyperpigmented borders and satellite lesions. Patients with active lesions were treated with classic drug therapy.

KEYWORDS

Ocular Toxoplasmosis, Toxoplasma Gondii, Posterior Uveitis, Congenital, Acquired.

INTRODUCTION-

Toxoplasma gondii is a ubiquitous obligate intracellular parasite, which infects both humans and animals [1,2]. Ocular toxoplasmosis is classified as either congenital or acquired infection. During the congenital infection, the foetus is infected via placental bloodstream, whereas during the acquired infection, parasite transfer is mediated typically through the gastrointestinal tract. Postnatal infection is now thought to be a more common cause of ocular toxoplasmosis [3,4,5]Clinical features are quite different from each other in that the congenital form tends to show bilateral macular lesions, which is quite different from typical ocular features which is focal retinitis adjacent to a chorioretinal scar or even without the presence of a scar. Most of acute systemic toxoplasmosis in normal hosts tend to be subclinical, but some may present with mild flu-like symptoms. If parasites reach an eye and they yield a focus of inflammation, the lesion is progressed to retinitis and involves the choroid secondarily. Immune responses of the host appear to induce conversion of the parasitic forms, from tachyzoites to bradyzoites and their encystment[6]. Ocular toxoplasmosis most often presents as a focal necrotizing retinitis. It is generally associated with vitritis and often with anterior uveitis. Less commonly, it may present as a papillitis. The age of the first attack of ocular toxoplasmosis is typically in the second decade and during a long-term follow-up, 5-year recurrence rate was 79%, and some patients had multiple recurrences [7,8]. The severity of anterior uveitis can range from a quiet anterior chamber reaction to an intense anterior uveitis, masking the inflammation of the posterior segment. It can be either granulomatous or non-granulomatous inflammations. Also, intense anterior inflammation may occur secondary to retinochoroiditis, near the ora serrata, which may be missed at initial examinations [9,10].

MATERIALAND METHODS-

This study was a retrospective observational study that involved 38 eyes of 30 patients of Ocular Toxoplasmosis complaining of blurring of vision, photophobia or floaters which was not improved with pinhole. Patients were recruited from the OPD done at the Maharani Laxmi Bai Medical College, Department of Ophthalmology between May 2022 to December 2022. It was performed under the Helsinki Declaration of 1975, as revised in 2000. The necessary from the Ethical and Research Committee was obtained for the study.

Inclusion Criteria

All patients who presented to us with blurring of vision and on both slit lamp and fundus examination had features of ocular toxoplasmosis, later confirmed by serology both by IgG and IgM specific for toxoplasma in patients with doubtful diagnosis.

Exclusion Criteria

Patient with other types of uveitis
Patient with ocular TB
Patient who had corneal opacity.
Patient who had cataract or retinitis pigmentosa
Patient who had diabetic retinopathy or hypertensive retinopathy.
Patient who had any intraocular surgery.
Patient who had any history of trauma.

All patients were subjected to proper history taking, and examination which included Slit lamp examination, Indirect ophthalmoscopy and Fundus picture.

RESULT:

A total of 38 eyes of 30 patients were studied in which mean age of presentation was 40 years with 8(26.6%) patients having bilateral presentation and rest 22(73.33%) with unilateral presentation. 70% of patients were male and rest 30% female.

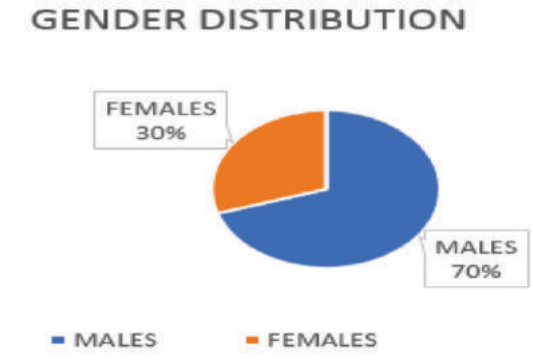

Table 1: Summary Of Various Symptoms In Male And Femaletypes Of Glaucoma

SYMPTOMS	MALE	FEMALE
Posterior uveitis (inactive phase)	17(44.7%)	06(15.7%)
Active posterior uveitis	05(13.1%)	03(7.8%)
Posterior + spillover anterior uveitis	03(7.8%)	01(2.6%)
Associated with vision impairing complications such as macular hole	02(5.2%)	01(2.6%)

Table: Location Of Retinitis At Presentation In Patients With Ocular Toxoplasmosis

LOCATION	n (%)
Macular	07(18.4%)
Adjacent to optic nerve	06(15.8%)
Peripheral	18(47.5%)
Both central and peripheral	07(18.4%)

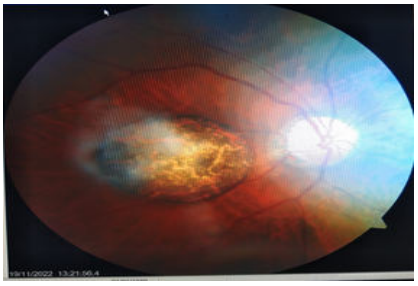

Image 1: Fundus photo of Inactive posterior uveitis in 58 years old male with chronic retinochoroiditis scar with hyperpigmented border present at macula of about 2DD (disc diameter) size with no vitritis.

The main aim of treatment is to stop the multiplication of tachyzoites during episodes of active retinochoroiditis. In patients in whom anterior chamber reaction was present, a topical cycloplegic with topical steroids was advised, and then the steroids were tapered as anterior chamber reaction resolved. In patients with active lesions affecting or near the optic nerve, a lesion that that threatened the major vessel was prescribed tab Pyrimethamine + tab sulfadiazine + tab folinic acid. Patients with inactive retinochoroiditis scar was just advised for follow up every monthly to see for any reactivation.

DISCUSSION:

Ocular toxoplasmosis is the most common cause of posterior uveitis worldwide. It is caused by the protozoa parasite Toxoplasma gondii, and can be acquired congenitally or by ingesting uncooked meat infected with cysts or vegetables and water contaminated by oocysts shed by cats. It is the most common cause of infectious intraocular inflammation. It typically affects the posterior pole of one or both eyes, and the lesions can be solitary, multiple or satellite to a pigmented retinal scar. The retina is the primary site of T. gondii infection in the eye but the choroid, vitreous and anterior chamber is retinochoroiditis i.e. the choroid is secondarily affected, meaning choroidal lesions do not occur in the absence of retinal infection. Active lesions present as grey-white focus of retinal necrosis with adjacent choroiditis, vasculitis (Kyreleis arteriolitis), hemorrhage and associated vitritis. Borders of the active lesion are the periphery towards the center of the lesion, with variable pigmentary changes. Healed lesion will have sharp boundary with no associated vitritis. The mother can transmit toxoplasmosis to the fetus if infected by T. gondii during pregnancy or a few months before conception. The infection can result in visual and hearing loss, mental and psychomotor retardation, seizures, hematological abnormalities, hepatosplenomegaly, or death. Maternal infection during first trimester of gestation has a lower chance of congenital transmission, but more severe consequences for the fetus when compared to the third[11].The diagnosis of Ocular toxoplasmosis is mainly clinical. Serological diagnosis can be done by IgG antibody titre demonstration. Pathological diagnosis of ocular toxoplasmosis can be established by identifying the cysts in biopsies stained with hematoxylin and eosin (H&E).The use of pyrimethamine, sulfadiazine, and corticosteroids is classical therapy for ocular toxoplasmosis and is the most common drug combination used. Patients with active toxoplasmosis may also be treated with trimethoprim-sulfamethoxazole with or without adjunctive clindamycin and prednisone for four to six weeks. In patients with frequent recurrences, long-term intermittent treatment with trimethoprim (160 mg)/sulfamethoxazole (800 mg), one tablet 3 times a week reduced the rate of recurrent toxoplasmic retinochoroiditis from 23.8% to 6.6%[12].

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