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A RARE CASE OF ACTIVE OCULAR TOXOPLASMOSIS – CASE REPORT

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ABSTRACT **Background:** Ocular toxoplasmosis, a potentially blinding and non-curable disease with a progressive and relapsing course, is the most common cause of infectious posterior uveitis. It is caused by the parasite Toxoplasma Gondii. Human infection by toxoplasma gondii may be either acquired or congenital. The principal modes of transmission include ingestion of undercooked, infected meat containing tissue cysts, ingestion of contaminated water, fruit, or vegetables with oocysts. Inadvertent contact with cat feces, cat litter, or soil containing oocysts .Transplacental transmission with primary infection during pregnancy and through blood transfusion or organ transplantation. **Case Presentation:** We report a case of 25-year-old female presented to our OPD with complaints of blurred vision since 15 days. Visual acuity in the right eye was 6/6 and 3/60 in the left eye with normal intraocular pressure in both eyes. Anterior segment examination with the slit lamp biomicroscopy revealed quiet anterior chamber with media clear .Posterior segment evaluation showed media haze due to vitritis of grade 2 with yellow white focal lesion of 1 disc diameter located at macula . Since the disease's progress may lead to recurrence and potential blindness, it must be recognized clinically, and treatment should be started as early as possible, especially in the active period. Our patient was treated with oral trimethoprim (160 mg)/sulfamethoxazole (800 mg) twice daily and topical eye drops of prednisone four times daily in the left eye. Suprachoroidal injection of TA was given .These drugs were prescribed instead of sulfadiazine/Pyrimethamine, which is classical and standard therapy. After 2 weeks, the fundus examination showed the toxoplasmic lesion was significantly decreased in size, and vitreous haze was improved. **Conclusion:** Trimethoprim-Sulfamethoxazole regiments as the alternative option for active ocular toxoplasmosis also shows significant improvement with less adverse effect.

KEYWORDS : retinochoroiditis, toxoplasma, trimethoprim,Sulfamethoxazole

INTRODUCTION

Ocular toxoplasmosis represents the most common cause of infectious retinochoroiditis in many countries, approximately 23-30% of the population worldwide(1,2) and has become the most frequent etiology of posterior uveitis .It is a potentially blinding, progressive, recurring, and non-curable disease caused by the intracellular parasite Toxoplasma gondii. Infection mainly occurs by eating raw or undercooked meat containing tissue cysts of T. gondii or by eating food or drinking water contaminated by the oocysts spread by cats as the definitive hosts. Ocular toxoplasmosis can be congenital, acquired, or recurrent. It may be active or inactive in the form of a scar affecting the posterior fundus. Typical symptoms are blurred vision, floaters, and metamorphopsia(1,2) It mostly presents as focal necrotizing retinitis involving the inner retinal layers appearing as a circular whitish fluffy lesion with surrounding retinal edema, localized or diffuse vitritis, and granulomatous anterior uveitis. In healthy patients, the retinitis heals within 1-4 months of treatment and is replaced with a sharply demarcated atrophic scar with pigmented borders.(2)

CASE PRESENTATION.

A 25-year-old female presented to our OPD with complaints of blurred vision since 15 days. Visual acuity in the right eye was 6/6 and 3/60 in the left eye with normal intraocular pressure in both eyes. Anterior segment examination with the slit lamp biomicroscopy revealed quiet anterior chamber with media clear .Posterior segment evaluation showed media haze due to vitritis of grade 2 with yellow white focal lesion of 1 disc diameter located at macula . Fundus examination of right eye was normal. FFA was done which showed hypofluroscent lesion at macula. Spectral-domain optical coherence tomography (SD-OCT) showed macular Hole stage 2 , vitreomacular traction band and epiretinal membrane. On laboratory examination, the serum titers of IgG antibodies against Toxoplasma gondii was found to be >150 IU/mL (normal <= 4 IU/mL) . Based on the clinical features and laboratory findings, a presumptive diagnosis of toxoplasma retinochoroiditis was made. patient was treated with oral trimethoprim (160 mg)/sulfamethoxazole (800 mg) twice daily and topical eye drops of prednisone four times daily in the left eye. After 2 weeks of treatment, the visual acuity of the left eye was 5/60, and intraocular pressure was stable. And after 4 weeks suprachoroidal injection of TA was given along with topical prednisolone. Vision improved from 5/60 to 6/24 . Fundus examination and photography after 2 weeks demonstrated a decrease in vitreous haze and size of toxoplasmic lesion on FFA. OCT showed vitreomacular traction band , macular hole of stage 2 , epiretinal memebrane. The course of oral trimethoprim/sulfamethoxazole was continued for another 4 weeks and patient was advised to visit the clinic every week for follow ups.

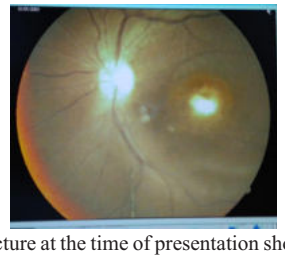


Fig :1 Fundus picture at the time of presentation showed chorioretinal scarring in macular area secondary to toxoplasmosis characterized by yellowish color lesion in centre surrounded by edge of hyperpigmented lesion.

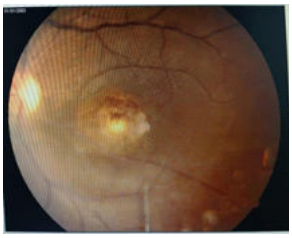


Fig :2. Fundus picture after two weeks of treatment showing decrease in size of lesion

Fig :3. FFA showing hypofluroscent area surrounded by hyperfluroscent area macula

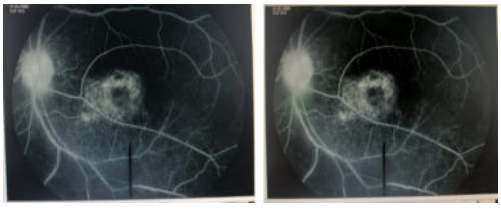
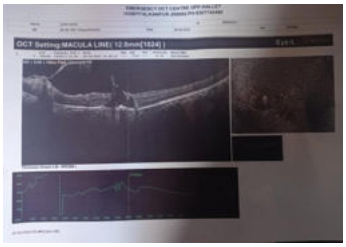


Fig :4. OCT picture showed macular hole of stage 2, vitreomacular traction band and epiretinal membrane.



Discussion

As a potentially blinding disease, ocular Toxoplasmosis must be recognized clinically, and treatment should be started as early as possible, especially in the active period. This case demonstrated the typical findings of ocular toxoplasmosis, which Is focal necrotizing chorioretinitis appearing as a whitish fluffy lesion with vitritis. The typical toxoplasmic lesion is usually near or at the border of an old pigmented and/or atrophic scar (satellite Lesions) and seen at the posterior pole of the fundus (3,4) an active retinochoroidal lesion usually heals within 1-4 months in healthy patients and is replaced with a sharp demarcated atrophic scar with a pigmented border, which resolves from the periphery to the centre(5) Few patients may develop foci of inflammation within or adjacent to the optic nerve head. Severe vitritis gives a "headlight in the fog" appearance(4)

The diagnosis of ocular toxoplasmosis is clinical, based on the medical history and clinical findings on slit-lamp or funduscopy examination that is consistent with Toxoplasma gondii infection of the retina. Various serology tests that are helpful for diagnosing include the Sabin-Feldman dye test, indirect fluorescent antibody test, immunosorbent agglutination assay, and ELISA (6). Positive serologic testing for anti– T gondii IgG or IgM confirms exposure to the parasite. IgG antibodies appear after the first 2 weeks of infection, typically remain detectable for life at variable levels, and cross the placenta. IgM antibodies, however, increase in number early during the acute phase of the infection, typically remain detectable for less than 1 year, and do not cross the placenta (7) In our patient IgG antibodies serum titer against T. gondii was found to be elevated, which confirmed the diagnosis.

The aim of the treatment is to reduce the risk of permanent visual impairment by reducing the size of the scar, the risk of recurrence,the severity and duration of the symptoms. The current suggested treatment options act against the tachyzoite form of Toxoplasma gondii (8). Although there is no consensus regarding the best treatment for ocular toxoplasmosis, classic "triple-drug therapy" of pyrimethamine (loading Dose 50-100 mg; therapeutic dose 25-50 mg/day), Sulfadiazine (therapeutic dose 4x1 g/day), and corticosteroids have been the most commonly used drugs combination(8-10) Alternatively, intravitreal injection of clindamycin and dexamethasone are acceptable as clindamycin is non-toxic to the retina, can cross the ocular barrier, and penetrates cells well (3) . Clindamycin 1,5 grams injection has a half-life of 5-6 days and is given every 1-2 weeks which may increase patient convenience, better systemic side effects, greater drug availability, and fewer follow-up visits (8)

Prevention of Recurrences

Prevention of recurrences of toxoplasmic retinochoroiditis may prevent loss of vision. Patients with paramacular toxoplasmosis may be given antimicrobial prophylaxis to prevent recurrences. Long-term intermittent trimethoprim/sulfamethoxazole prophylactic treatment can prevent or decrease the recurrences of toxoplasmic retinochoroiditis. The treatment consists of the administration of sulfamethoxazole/trimethoprim 800/160 mg 1 tablet every 3 days. (11)

Conclusion

Instead of classic treatment for ocular Toxoplasmosis, Trimethoprim-Sulfamethoxazole regimens as the alternative option for active ocular Toxoplasmosis also shows significant improvement with less adverse effect. Moreover, the standard treatment is expensive, might have major adverse effects, and could not be readily available in Some countries. As presented in this case report , the regiments resulted in a good outcome with significant improvement. But our patient had already developed complications such as macular hole stage 2 ,traction band and epiretinal membrane hence her vision might detoriate in future Being a potentially blinding disease, ocular Toxoplasmosis preventive measures should be taken, such as proper handwashing and satisfactory food hygiene. Newer treatment options need to be effective in preventing and treating the disease with lower doses of drugs, better patient compliance, cost reduction, and fewer adverse effects. Moreover, prophylaxis to the disease needs to be advised, especially for immunocompromised and recurring patients

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