Foveal Regeneration after Treatment of Acute Foveal Toxoplasmic Chorioretinitis

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Brief report

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Abstract

Purpose: To report a patient with impaired vision due to foveal involvement of toxoplasmic chorioretinitis, who was successfully treated with intravitreal and oral therapy and led to successful visual and anatomic recovery.

Case presentation: A thirty two-year-old man presented with three-day history of gradually decreasing visual acuity, redness, pain and photophobia of the right eye. Anterior chamber cellular reaction, vitritis and a white retinochoroiditis patch with adjacent retinal vasculitis in the fovea was suggestive of the toxoplasmic chorioretinitis. He was treated with intravitreal Clindamycin and Dexamethason injection followed by six-week regimen of Azithromycine, Trimethoprim-Sulfamethoxazole, and Prednisolone. In serial optical coherence tomography imaging, retinitis patch changed to cavitary foveal destruction. Fovea was regenerated gradually, and visual acuity was concurrently improved from counting finger 3m to 20/25.

Conclusion: In foveal toxoplasmic chorioretinitis lesions, timely treatment is associated with retinal regeneration and visual improvement.

Introduction

Toxoplasmic chorioretinitis caused by Toxoplasma gondii is a potential cause of posterior uveitis that could lead to blindness. It occurs in the posterior pole in more than 50% of cases.[1] Symptoms of ocular toxoplasmosis usually include a unilateral decrease in vision with floaters, accompanied by signs of anterior uveitis, and around one fifth of patients have increased intraocular pressure.[2] In the posterior segment, the condition classically appears as focal, grayish-white retinitis with overlying moderate vitreous inflammation, often adjacent to a pigmented chorioretinal scar.[2] The diagnosis of toxoplasma chorioretinitis is clinical in most instances. Visual loss caused by ocular toxoplasmosis arises from complications due to structural changes and the effects of associated intraocular inflammation and scar formation.[2] As the focal lesion of necrosis of the retina and choroid heals, a permanent punched-out chorioretinal scar will develop. Due to the high prevalence of the lesion occurring at the posterior pole, it is considered as a cause of severe vision loss.[2] The inflammation process itself may create inadvertent complications that can lead to permanent conditions if left unattended in a timely manner.[3]

Here we report a patient with impaired vision due to foveal involvement of toxoplasmic chorioretinitis, who was successfully treated with intravitreal antibiotic and corticosteroid injection followed by six-week oral therapy and had near complete visual recovery.

Case Presentation

A thirty two-year-old man presented with three-day history of decreased visual acuity, redness, pain and photophobia of the right eye. Best corrected visual acuity (BCVA) was counting finger 3 meters in the right

eye and 20/20 in the left eye. Relative afferent pupilary defect was absent. Intraocular pressure was within normal limits. In slit lamp biomicroscopy of the right eye, conjunctival injection and 3 + anterior chamber reaction was found. Lids, cornea and lens were normal. Fundus examination revealed vitritis, media haziness, and a round, fluffy, white retinochoroiditis patch, about one-disc diameter in size, in the foveal area associated with edema and retinal vasculitis. Left eye examination was normal. These findings were compatible with the diagnosis of ocular toxoplasmic chorioretinitis in the active phase. The clinical suspicion of toxoplasmosis was confirmed by positive serology tests of anti-toxoplasma IgG and IgM antibodies. Foveal involvement was documented with enhanced-depth imaging (EDI) optical coherence tomography (OCT). At first, EDI-OCT image showed foveal retinal thickening and edema besides choroidal thickening and congestion. Treatment was initiated immediately with intravitreal injection of Dexamethason (0.4 mg/0.1 mL) and Clindamycin (1.0 mg/0.1 mL) in the right eye. It was followed by oral Azithromycine (250 mg/day) and trimethoprim-sulfamethoxazole (160 mg/800 mg twice daily), topical Betamethasone (every four hours) and Homatropine (twice a day) for up to six weeks. Oral Prednisolone (50 mg/day) was added to the above regimen from the fourth day of treatment.

One week after starting the treatment, retinal and choroidal thickening subsided and cavitary retinal changes attributable to retinal destruction developed in EDI-OCT. Fovea was regenerated gradually and foveal retinal layers integrity which had been lost, returned progressively. Simultaneously with these anatomical changes, the right eye vision also improved significantly to 20 /25.

Discussion

Herein, we report a patient with foveal toxoplasmic chorioretinitis that showed anatomical regeneration of the fovea with intravitreal and oral antibiotics and corticosteroid. Response to treatment and control of infection and inflammation was associated with the reformation of the foveal pit and restoration of the layered structure of the tissue. While macular toxoplasmic chorioretinitis are usually associated with scar formation and severe visual reduction, our patient's visual acuity was well recovered, most probably due to foveal regeneration.

Different modes of foveal regeneration were proposed in spontaneous or surgical closure of macular hole: 1) regular regeneration resulted in the formation of a fovea which contained a foveola and photoreceptors in the center; 2) irregular regeneration resulted in the formation of a fovea which did not contain a foveola and central photoreceptors and was filled by a tissue formed by Müller and retinal pigment epithelial cells.[4] Because the fovea is free of astrocytes[5], the closure is likely mediated by Müller cells. Concentric contraction of the Müller cell processes envelops the photoreceptor cells in the outer nuclear layer and at the external limiting membrane and result in the centripetal movement of photoreceptor cells.[4] The irregular regeneration was mediated by proliferation of Müller and retinal pigment epithelial cells.[6,7] It was suggested that closure of small macular holes and the subsequent reconstruction of the normal foveal structure are mediated by active mechanisms of Müller cells, without cell proliferation, that resemble those involved in ontogenetic foveal development.[7] We believe that

regeneration of fovea in our patient may be related to Müller cells, like what happens in macular hole repair.

Histopathologic findings in an eye with active toxoplasmic retinochoroiditis show cell infiltration and edema, initially in the internal retina and then in the vitreous and choroid. As a result of the infiltration, there is a disorganization of the retinal layers.[8,9] This hystopathological feature can be correlated to the spectral domain optical coherence tomography (SD-OCT) findings of hyper-reflectivity and increased thickness of retina at the lesion site.[10] Some of the SD-OCT findings in reactivation of ocular toxoplasmosis have been described previously which included increased reflectivity in the inner retina, shadowing of the outer retina layers, thickened and detached posterior hyaloid with irregular hyperreflective formations.[11–13]Alwassia A. et al described the progression of retinitis in a case with acute ocular toxoplasmosis using SD-OCT. In this patient, the development of cystic spaces in the area of previous hyperreflectivity possibly represents the progression from retinitis to liquefactive necrosis as a result of inflammation. The retina in this case began a healing process as the gap within the retina caused by necrosis became smaller.[14] Our patient also initially underwent cavitary changes, possibly due to liquefactive necrosis, and then the healing process began with fluid absorption. Finally, the layered structure of the tissue restored and the foveal pit reformed.

In conclusion, we believe that although toxoplasmic chorioretinitis lesions often heal with atrophy and scar, early aggressive treatment with both intravitreal and systemic antibiotics and corticosteroid appears to prevent scar formation and allows retinal layer regeneration.

Abbreviations

BCVA: best corrected visual acuity

EDI-OCT: enhanced depth imaging optical coherent tomography

SD-OCT: spectral domain optical coherence tomography

Declarations

Ethics Approval:

Not Applicable

Consent for publication:

Written consent for images and data publication and identifying clinical details was obtained from the patient.

Availability of data and material:

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing interests:

The authors declare that they have no competing interests

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Authors' contributions:

All the authors contributed significantly to this report, and all authors agree to be accountable for all aspects of the work. All authors read and approved the final manuscript.

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Figures

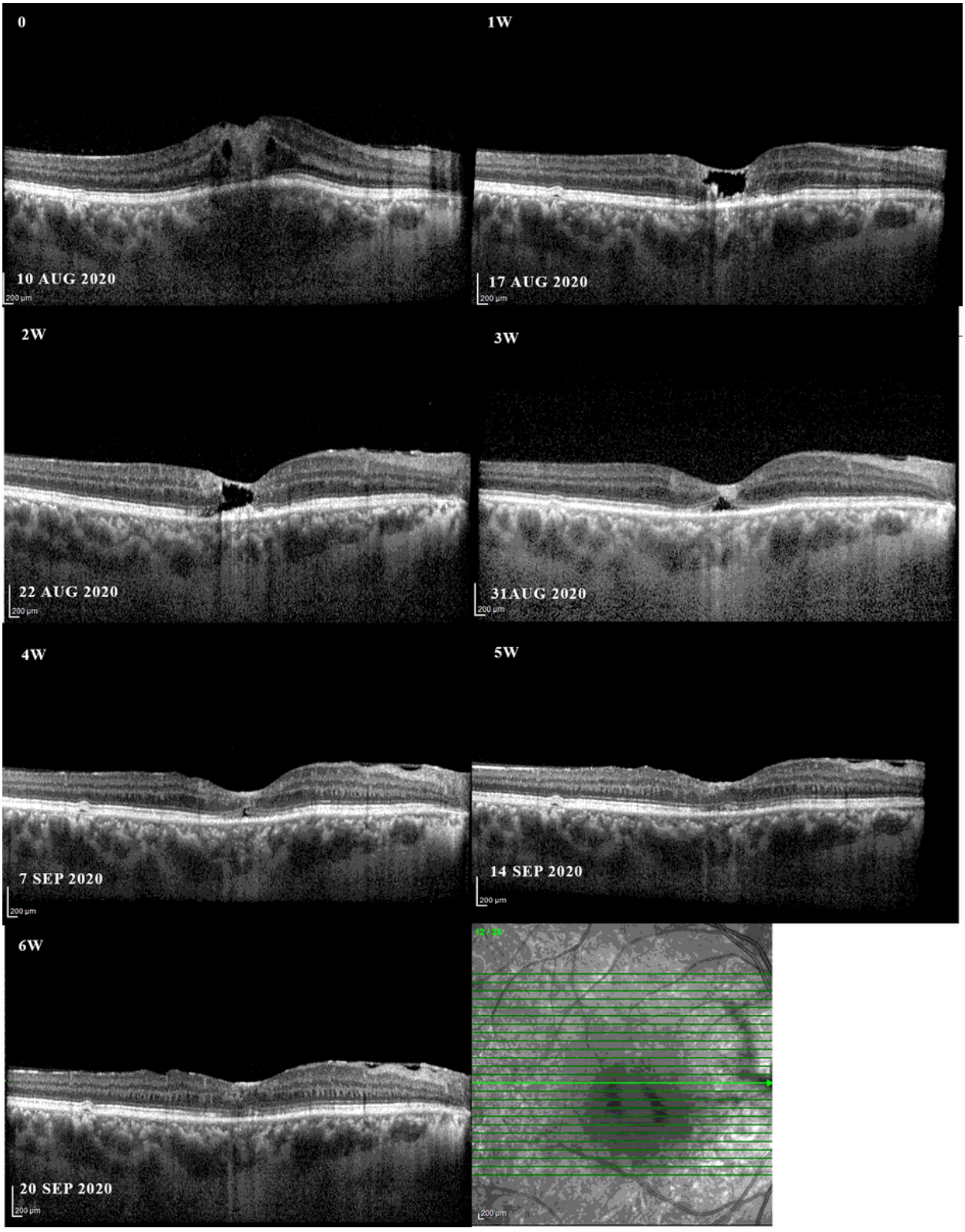

Figure 1

The optical coherence tomographical images (linear horizontal scans through the fovea and parafovea) of the right eye. The weeks after the first examination (0) are indicated above the images. The orientations of the scans are shown at the right bottom side.