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CASE REPORT

A case of ocular toxoplasmosis presenting with neuroretinitis

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Abstract

A 33-year-old female patient, who followed up in an external center with the diagnosis of optic neuritis 2 years ago, had complaints of decreased vision and headache for 1 week. In our examination, visual acuity was counting fingers from 2 m in the right eye and 1.0 in the left eye with a Snellen chart. The bilateral anterior segment was normal in the slit-lamp examination. Color vision was 0/12 in the right eye and 12/12 in the left eye. In dilated fundus examination, optic nerve head edema was present in the right eye, while the optic nerve, macula, and retina of the left eye were normal. In the visual field, an inferior arcuate visual field defect was observed in the right eye. Anti-Toxoplasma immunoglobulin M resulted in 1240 IU/mL (positive) and immunoglobulin G 90.5 IU/mL (positive). Optical coherence tomography showed pigment epithelial detachment adjacent to the optic disc. Trimethoprim/sulfamethoxazole 800/160 mg 2 × 1, azithromycin 1000 mg loading, followed by 500 mg 1 × 1 (1 week) was started. On the 3rd day of the treatment, a prednisolone 1 mg/kg/day weekly reduction regimen was started. There was a macular star appearance with hard exudates in the macula with a rapid recovery with treatment. At the 6th-month follow-up, visual acuity was 0.5 in the right eye and 1.0 in the left eye, while the anterior segment slit-lamp examination was normal. In dilated fundus examination, the temporal part of the optic disc was pale and macular hard exudates were present in the right eye; and the left was normal.

Keywords: Macular star; neuroretinitis; ocular toxoplasmosis; Toxoplasma gondii.

In human beings and warm-blooded animals, *Toxoplasma gondii* is a mandatory intracellular parasite which remains persistent in contaminated hosts for life.^[1] We usually see the infection progressing asymptotically; in addition, the protozoa can cause toxoplasmosis which is defined as a disease. *T. gondii*, which is one of the most common causes of posterior uveitis, appears as acquired or congenital.^[2] Ocular toxoplasmosis is a condition that can lead to blindness, presenting with focal necrotizing chorioretinitis typically seen adjacent to the focus of atrophic chorioretinitis. Less frequently, clinical manifestations include optic

neuropathy, neuroretinitis, retinal vasculitis, retinal detachment, punctate outer retinal toxoplasmosis, and pigmentary retinopathy.^[3,4] It is aimed to share a case of ocular toxoplasmosis presenting with neuroretinitis.

CASE REPORT

A 33-year-old female patient was admitted to our clinic in March 2021 with complaints of decreased vision and headache for 1 week. It was learned that she was followed up in an external center with the same complaints and a diagnosis of optic neuritis 2 years ago. In the ophthal-



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Fig. 1. Severe optic disc edema in the right eye of the patient in the color fundus photograph at the first admission.

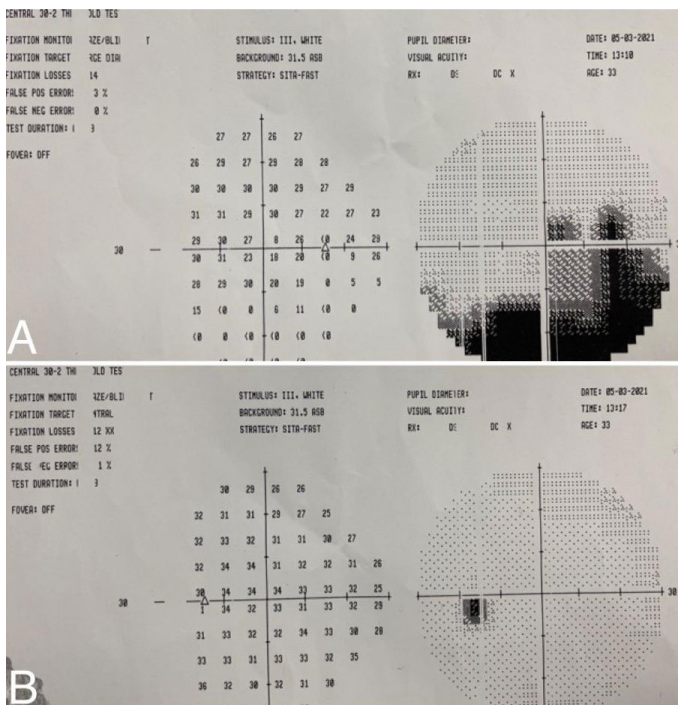


Fig. 2. Visual field test of the patients right and left eyes. (a) An inferior arcuate visual field defect in the right eye. (b) Normal visual field test in the left eye.

mological evaluation, visual acuity was counting fingers from 2 m in the right eye and 1.0 in the left eye, using the Snellen chart. The bilateral anterior segment was normal in the slit-lamp examination. Color vision was 0/12 in the right eye and 12/12 in the left eye. In dilated fundus examination, optic nerve head edema was present in the right eye (Fig.1), while the optic nerve, macula, and retina of the left eye were normal. In the visual field, an inferior arcuate visual field defect was observed in the right eye (Fig. 2). In

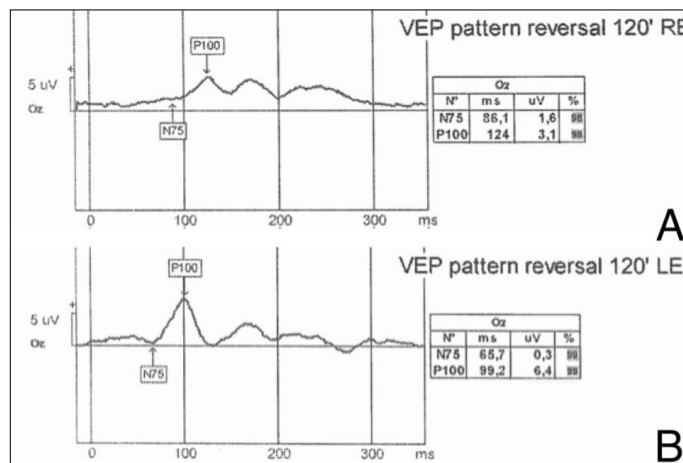


Fig. 3. Visual evoked potential of the patients right and left eyes. (a) A normal response was obtained with the 120' pattern; p100 wave latency was normal and a low amplitude was observed in the right eye. (b) Responses with a 120 pattern in the left eye were within normal limits.

a visual evoked potential test; a normal response was obtained with the 120' pattern; p100 wave latency was normal and low amplitude was observed in the right eye; while responses with a 120' pattern in the left eye were within normal limits (Fig. 3).

Complete blood count, liver and kidney function tests, sedimentation, C-reactive protein, chest X-ray, serum angiotensin converting enzyme, VDRL, Borrelia burgdorferi polymerase chain reaction, Bartonella henselae immunoglobulin G (IgG), anti-Toxoplasma immunoglobulin M (IgM)-IgG, and brain-orbital-cervical magnetic resonance imaging (MRI) were requested. While anti-Toxoplasma IgM results in 1240 IU/mL (positive) and IgG 90.5 IU/mL (positive), in orbital MRI; a 1–2 mm thick lesion with no pathological contrast enhancement was observed on the posterior wall of the bulbus oculi in the right eye, adjacent to the choroid, which was hypointense in T2A series compared to the vitreous. There was no finding to support the differential diagnosis of neuroretinitis in other examination results for etiological factors.

Optical coherence tomography showed pigment epithelial detachment adjacent to the optic disc (Fig. 4). Trimethoprim/sulfamethoxazole 800/160 mg 2 × 1, azithromycin 1000 mg loading, followed by 500 mg 1 × 1 (1 week), was started. On the 3rd day of the treatment, a prednisolone 1 mg/kg/day weekly reduction regimen was started. There was a macular star appearance with hard exudates in the macula with a rapid recovery with treatment (Fig. 5). At the 6th-month follow-up, visual acuity was 0.5 in the right eye and 1.0 in the left eye, while the anterior segment slit-lamp examination was normal. In dilated fundus examination,

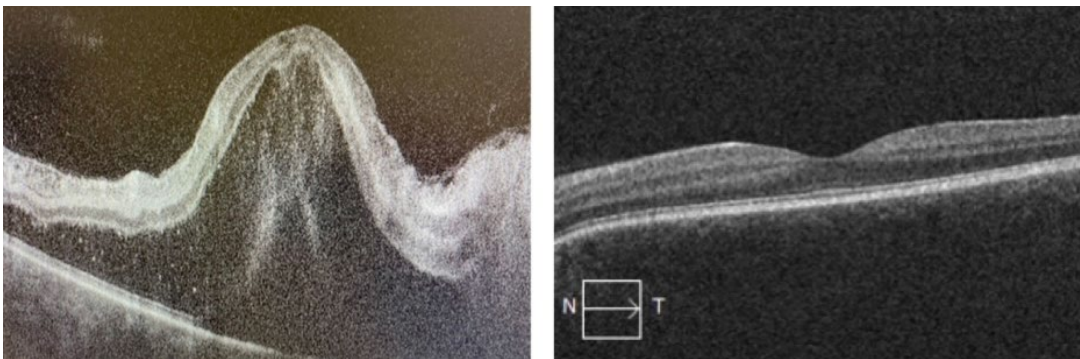


Fig. 4. Optical coherence tomography of the patient at the time of admission. While pigment epithelial detachment and subretinal fluid accumulation were observed adjacent to the optic disc in the right eye; the left eye was normal.



Fig. 5. Color fundus photographs and red-free images of the patient. As a result of rapid recovery with treatment, the appearance of a macular star with hard exudates is seen in the right eye.

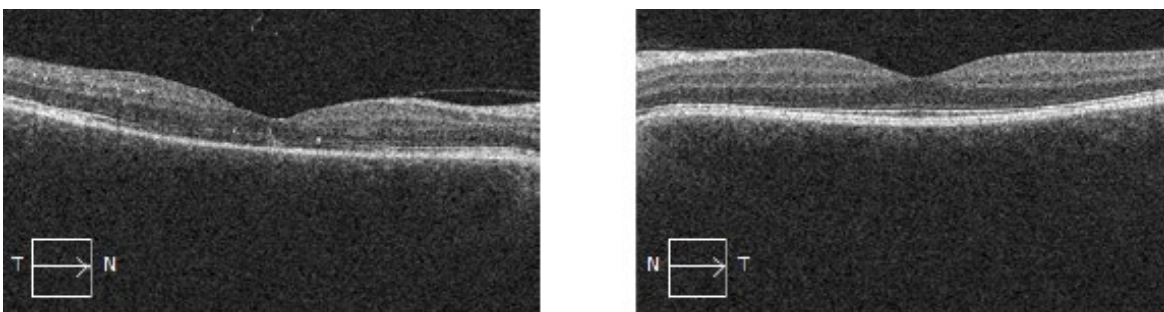


Fig. 6. Optical coherence tomography of the patient at the 6th-month follow-up. The pigment epithelial detachment and subretinal fluid accumulation in the right eye completely regressed.

the temporal part of the optic disc was pale and macular hard exudates were present in the right eye; and the left was normal. The pigment epithelial detachment and sub-retinal fluid accumulation in the right eye completely regressed in optical coherence tomography at the 6th-month follow-up (Fig. 6).

Discussion

Lesions in many different forms due to *Toxoplasma* have been defined. The most common are destructive lesions, punctate internal retinal lesions, and areas of active retinitis.^[5-7] Neuroretinitis is a condition often seen in young adults, characterized by optic nerve head edema, star-shaped hard exudate in the macula, and vitreous inflammation resulting in unilateral rapid vision loss.^[8] There are a wide variety of factors that cause neuroretinitis, which are basically divided into two as infectious and non-infectious.^[9] In non-infectious neuroretinitis, the patient usually has an underlying systemic or inflammatory disease.^[10] In addition, the factors causing infectious neuroretinitis are bacterial (*Bartonella* species, *Rickettsia rickettsii*, *Mycobacterium tuberculosis*, *Salmonella*, and *Syphilis*), viral (*Varicella*, *Herpes simplex*, *Herpes zoster*, *Dengue*, *Influenza A*, *Hepatitis B*, *Epstein-Barr*, and *Coxsackie B*), fungal (*Coccidioidomycosis* *Histoplasmosis*, *Actinomycosis*), and parasitic factors like *Toxoplasmosis*.^[11] Severe hypertension, diabetes mellitus, and retinal vascular diseases in which disc swelling and macular exudate can be seen confused with neuroretinitis. Therefore, the patient's anamnesis should be taken in detail and should be considered among the differential diagnoses.

Perrotta et al. shared a 6-year-old case with bilateral neuroretinitis due to ocular toxoplasmosis. While the diagnosis was suspected in the patient who had only bilateral optic nerve head edema in the first examination, the appearance of a macular star led to the diagnosis. It has been stated that the absence of anterior segment findings and existing chorioretinitis scar may lead to the misdiagnosis of idiopathic neuroretinitis.^[12] Similarly, neuroretinitis due to ocular toxoplasmosis without a previous scar was mentioned in the case report of 2 cases of Küçükerdönmez et al.^[13] As in our case, retinal pigment epithelial detachment was observed adjacent to the disc in both cases. *Toxoplasma* neuroretinitis was diagnosed with a high anti-*Toxoplasma* antibody level and a good response to the treatment given, and the importance of serology in neuroretinitis cases was emphasized. In the review of Vasconcelos-Santos, it was stated that high anti-*Toxoplasma* IgG and IgM titers were significant in terms of newly acquired toxoplasmosis.^[14]

In the retrospective review of the results of 19 ocular toxoplasmosis cases by Yazici et al., it was mentioned that the combination of trimethoprim/sulfamethoxazole and azithromycin is a safe treatment option as it increases visual acuity, decreases inflammation, and decreases recurrence rates.^[15] While we observed rapid recovery with this combination in our case, we did not record any recurrence in the 6-month follow-up.

Hamurcu et al. reported that three cases of ocular toxoplasmosis have optic neuritis and juxtapapillary chorioretinitis.^[16] All three patients applied with similar complaints and their only positive findings were anti-*Toxoplasma* IgG positivity. Caused by ocular toxoplasmosis in patients, it was accepted that juxtapapillary chorioretinitis and accompanying optic neuritis developed, and three patients were treated with Trimethoprim/sulfamethoxazole and systemic corticosteroids, then clinical improvement was observed in those three patient.

In our patient, papilledema was accompanied by macular edema. In these cases, if macular edema is overlooked and neuroretinitis is missed; considering idiopathic optic neuritis, there is a risk of only steroid treatment. In addition, it can be thought that the decrease in vision experienced by our patient 2 years ago was also an attack due to neuroretinitis. In such cases, it should be considered that *Toxoplasma* infection may be the cause of papillitis and appropriate treatment should be given.

Conclusion

Even if there is no chorioretinitis scar in patients with optic nerve head edema, *Toxoplasma* serology should be checked. Ocular toxoplasmosis should not be forgotten in the differential diagnosis of neuroretinitis. In the treatment of ocular toxoplasmosis, corticosteroids should be included in the treatment plan when the optic disc or macula is threatened. On the other hand, giving steroids alone or local steroid applications can cause vision-threatening results. Differential diagnosis of neuroretinitis is vital, steroid treatment should be given safely under the umbrella of antiprotozoal therapy. Infectious etiologies should be investigated while preparing the treatment plan for neuroretinitis patients.

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