Intravitreal Bevacizumab for the Treatment of Retinal Neovascularization Secondary to Ocular Toxoplasmosis

Oküler Toksoplazmozise Sekonder Retinal Neovaskülarizasyon Tedavisinde İntravitreal Bevacizumab

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ABSTRACT Our aim was to present a case of ocular toxoplasmosis with retinal neovascularization treated with intravitreal bevacizumab. A 21-year-old healthy man had admitted to our Eye Clinic with complaints of floaters and blurred vision in the right eye (OD) for one year. On examination, visual acuity was 20/200 in the right eye and 200/200 in the left eye (OS). A few active inflammatory cells were seen in the vitreous (1+ cell), and a high IgG anti-Toxoplasmosis titer was detected. Fundus examination revealed bilateral chorioretinal scars in posterior poles and peripheral retinal neovascularization in the superotemporal region of the right eye. After administration of intravitreal bevacizumab (1.25 mg in 0.05 ml OD) the peripheral retinal neovascularization regressed. In summary, intravitreal bevacizumab may be an effective alternative option in the management of retinal neovascularization secondary to ocular toxoplasmosis.

Key Words: Bevacizumab; toxoplasmosis, ocular; retinal neovascularization

ÖZET Bu yazıda amacımız periferik neovaskularizasyon gelişen toksoplasma retinokoroiditli bir olguda bevacizumab tedavisinin etkinliğini sunmaktır. Yirmi bir yaşında erkek hasta bir yıldır sağ gözde bulanık görme ve uçuşma şikâyetleri ile başvurdu. Sağ makulada ve sol temporal retinada retinokoroidit skarları, sağ göz üst temporal periferde aktif neovaskularizasyon, vitreusta 1+ hücre ve yüksek serum Toksoplazmozis IgG titresi tespit edildi. Olgunun sağ gözüne intravitreal bevacizumab (1,25 mg/0,05 ml) enjeksiyonu uygulandı ve periferik neovaskularizasyonda gerileme görüldü. Oküler toksoplazmozisli olgulardaki retinal neovaskülarizasyonlarda intravitreal bevacizumab uygulaması etkin, alternatif bir tedavi seçeneği olabilir.

Anahtar Kelimeler: Bevacizumap; toksoplazmozis, göz; retina yeni damarlanması

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Ocular toxoplasmosis is one of the most common causes of severe visual loss in young people, accounting for 30-50% of all posterior uveitis cases.[1] Ocular involvement can be a result of acquired infection or, more commonly, a recurrence of the congenital form of the disease. Recent reports indicate that acquired infections may be an important cause of ocular disease than previously thought.[2] Atypical manifestations of ocular toxoplasmosis include retro bulbar neuritis, pars planitis, papillitis, neuroretinitis, and retinal or choroidal neovascularizations (CNV).[3]

Recent reports have demonstrated short-term efficacy and safety of intravitreal injection of bevacizumab in the treatment of choroid neovascularization due to age-related macular degeneration, myopia, and angioid streaks.[4,5] To our knowledge, no reports on the intravitreal bevacizumab

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administration for the treatment of retinal neovascularization caused by ocular toxoplasmosis are available. In this report, we presented a case of retinal neovascularization secondary to ocular toxoplasmosis and it's, effectively treatment with intravitreal bevacizumab.

CASE REPORT

A 21-year-old man was admitted to the ophthalmology clinic with floaters and reduced, blurred vision which had persisted for one year. His best corrected visual acuity was 20/200 in the right eye and 200/200 in the left eye. Biomicroscopy of the anterior segments were normal except for presence of few active inflammatory cells (grade 1+) in the vitreous. Intraocular pressures for the right and left eyes were 14 and 17 mmHg, respectively. Fundoscopic evaluation revealed atrophic chorioretinal scars in the macula of both eyes and peripheral retinal neovascularization in superotemporal region of the right eye (Figure 1). On fundus flourocein angiography, increased leakage was observed in the late phase which was due to retinal neovascularization secondary to ocular toxoplasmosis. After three months, the leakage decreased due to regression of neovascularization (Figure 2 on right and left). There was positive IgG anti-Toxoplasmosis titer in laboratory report. The patient was treated with trimethoprim/sulfamethoxazole (160 mg/800 mg, twice a day), and clindamycin (300 mg, four times a day) for 6 weeks. Oral corticosteroid therapy (0.5 mg/kg/day prednisolone equivalent) was initiated 48 h after the administration of antiparasitic treatment, and was discontinued before antiparasitic treatment was terminated.

Peripheral neovascularization evolved by the 3rd month of follow-up, and at this time after giving his written informed consent, the patient received a single intravitreal injection of bevacizumab (1.25 mg/0.1ml). At the 6th month visit, neovascularization seemed regressed (Figure 2b). An additional laser treatment (532 nm) was applied to protect retina from retinal breaks. In the last visit of the patient, the neovascularization seemed to be regressed more and his visual acuity remained stable with no active cells in vitreous cavity (Figure 3).

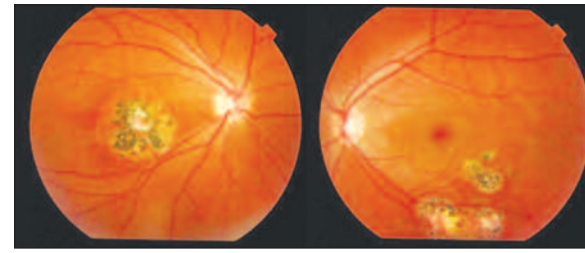

FIGURE 1: Old lesions of toxoplasmosis retinochoroiditis in the posterior poles of both eyes.
(See for colored form http://tipbilimleri.turkiyeklinikleri.com/)

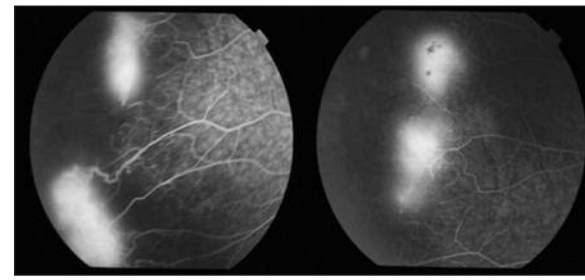

FIGURE 2: The leakage of retinal neovascularization in the superotemporal region of the right eye (on left) intravitreal application, the leakage decreased due to regression of neovascularization on 6th months (at right).

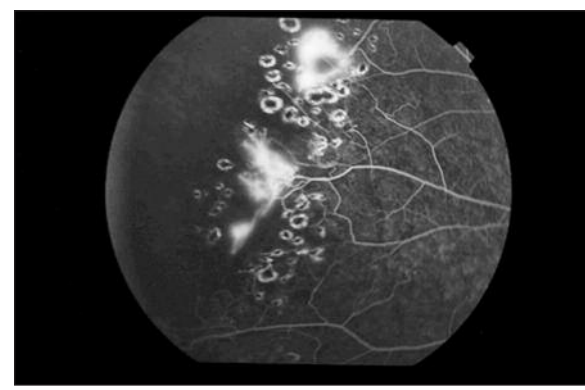

FIGURE 3: After protective laser treatment, fibrotic membrane remnant of peripheral neovascularization.

DISCUSSION

Toxoplasmosis retinochoroiditis is unilateral in 72-83% of reported cases.[4] A prospective longitudinal study showed that lesions were located in the macula in 54% of the eyes examined, and extramacular peripheral scars were detected in 58% of the eyes.[6] The primary active intraocular manifestation of toxoplasmosis is retinitis. The lesion begins in the superficial layers of the retina, and then affects the retinal pigment epithelium and choroids.[7] As we

observed in our case, ocular toxoplasmosis is seen unilaterally and then involves the posterior pole of the other eye. Adolescents and young adults complain of blurred vision, floaters and sometimes pain and photophobia, and they present with conjunctival hyperemia if the anterior segment is involved.

The most common complications of ocular toxoplasmosis is visual loss due to macular or optic nerve lesions. Chronic iridocyclitis, cataract, secondary glaucoma, cystoids, macular edema, choroid neovascularization and retinal detachment are other complications of the disease.[8] Recent treatment options for CNV due to ocular toxoplasmosis include laser photocoagulation, surgery, corticosteroids and verteporfin as well as their combinations.[9,10] In our case, intravitreal bevacizumab was applied to retinal neovascularization as an alternative treatment when previous studies in literature were taken into consideration.[11,12]

In conclusion, ocular toxoplasmosis frequently affects the macula and seriously impairs visual acuity. Patients with ocular toxoplasmosis should be followed and monitored for late atypical complications including retinal neovascularization due to retinal ischemia. In our case, peripheral retinal neovascularization was disclosed and regressed via anti-vascular endothelial growth factor (VEGF). Intravitreal bevacizumab has the advantage of being cost-effective when compared to other VEGF inhibitors and photodynamic therapy. However, its long-term efficacy and safety in the treatment of retinal neovascularization secondary to ocular toxoplasmosis require further investigations.

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