

Intravitreal Dexamethasone Implantation in A Patient with Persistent Macular Edema After Closed Globe Trauma

*Israrcı Maküla Ödemi Olan
Bir Hastada İntravitreal
Deksametazon İmplantasyonu*

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ABSTRACT

Commotio retinae, is mainly due to the pathologic changes of the outer retina and especially photoreceptors and a real macular edema and extracellular fluid accumulation rarely associate to commotio retinae. In this case report, we performed an intravitreal Ozurdex® implant to an eye with persistent macular edema after a blunt trauma. We observed a dramatic improvement in visual acuity and macular edema after intravitreal Ozurdex® implantation. The dramatic improvement in our case report shows that more prospective studies are warranted to learn more information about the importance of inflammation and vascular changes in the pathogenesis of the commotio retinae.

Key Words: Commotio retinae, persistent macular edema, intravitreal dexamethasone implant.

ÖZ

Kommosyo retina, esas olarak dış retina ve özellikle fotoreseptörlerdeki patolojik değişikliklere bağlı oluşur ve gerçek bir maküla ödemi ve ekstraselüler sıvı birikimi nadiren oluşur. Bu olgu sunumunda, künt travma sonrası ısrarcı macula ödemi olan bir olguya intravitreal Ozurdex® implantasyonu uygulandı. Olgu sunumumuzdaki dramatik düzelme, kommosyo retinanın patogenezindeki inflamatuvar ve vasküler değişiklikler hakkında daha çok prospektif çalışma yapılması gerektiğini göstermektedir.

Anahtar Kelimeler: Kommosyo retina, ısrarcı macula ödemi, intravitreal deksametazon implant..

INTRODUCTION

Macular edema is one of the leading cause of vision loss among patients with retinal vein occlusion, diabetic retinopathy, and posterior-segment inflammatory diseases.¹⁻³ Corticosteroids possess anti-inflammatory and antiangiogenic properties that make them an attractive therapeutic option for a variety of posterior segment diseases.^{4,5}

Given the short half-life of dexamethasone in the vitreous cavity, the crystalline form of a lipophilic corticosteroid, triamcinolone acetonide, gained widespread use for treatment of macular edema.^{6,7} To address this drawback, a biodegradable dexamethasone drug delivery system was developed and acquired by Allergan (Ozurdex®, Allergan, Irvine, CA, USA). Ozurdex® was designed to provide sustained delivery of 700 µg of preservative-free dexamethasone to the vitreous cavity and retina, and was recently approved by the United States Food and Drug Administration (FDA) for the treatment of macular edema associated with retinal vein occlusion, as well as for noninfectious posterior uveitis.^{8,9} Ozurdex has also an approval for diabetic macular edema by FDA in the USA in September 2014.

The relation between the deformation of the globe under high-speed impact and the existence of retinal lesions has been extensively explored.¹⁰ Commotio retinae, may be observed in patients involved in blunt eye traumas. In commotio retinae, structural changes have been observed in the outer layers with breakage of connecting cilia of rods and cones.^{11,12} The fundoscopic findings may be normal or are often subtle, including a gray swelling of the fovea accompanied by retention of the foveal depression.

Here we report a case that had a persistent macular edema after a closed globe trauma which was treated with Ozurdex® implant.

CASE REPORT

A 30-year-old man had acute reduction of visual acuity in the left eye following a blunt eye trauma with a steel rope.

The patient was present 1 day after the trauma. On presentation, best corrected visual acuity (BCVA) were 20/20 in the right eye and 20/100 in the left eye. Slit-lamp examination was normal except a minimal anterior chamber reaction in the left eye, intraocular pressure (IOP) was 14 mmHg in the right eye, 12 mmHg in the left eye. Fundus examination was normal in right eye, and there was a mild irregularity in the foveal region of the left eye (Figure 1). There was no reaction in the vitreous. Peripheral fundus examination revealed no hemorrhage or retinal tear bilaterally. Optic nerves of both eyes were normal and there was no relative afferent pupil defect.

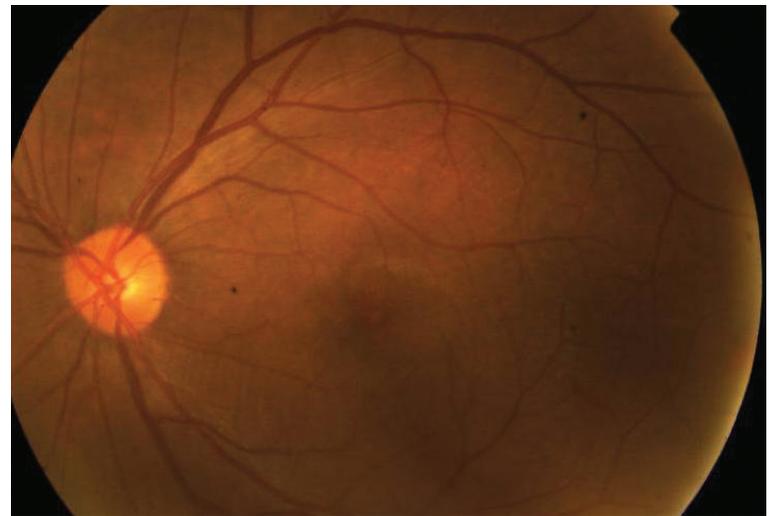


Figure 1: Colour fundus photograph of the left eye shows a mild irregularity at the foveal region of the left eye.

Fundus fluorescein angiography was normal in the right eye, whereas there was an increasing fluorescein leakage in the left eye (Figure 2a,b). In optical coherence tomography (OCT) there was no remarkable finding in the right eye but OCT findings included a thickened outer retina and diffuse macular edema in the left eye (Figure 3). The central foveal thickness was recorded as 215 µm and 450 µm in the right and left eye, respectively. We decided to observe the patient for spontaneous resolution of macular edema. There was no improvement in BCVA or macular edema at 2 months after the trauma in the left eye.

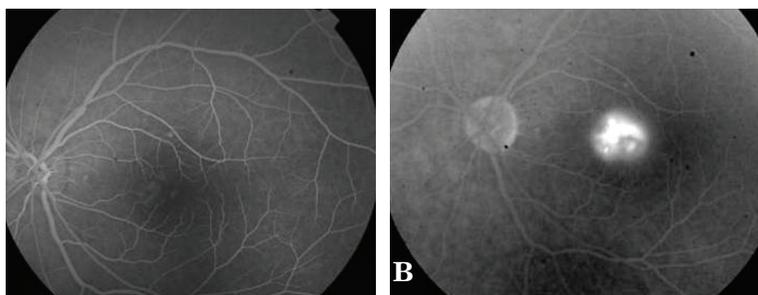


Figure 2a,b: Fundus fluorescein angiography demonstrates fluorescein leakage in early arteriovenous phase (a). Increasing fluorescein leakage in the late venous phase of the fundus fluorescein angiography in the left eye (b).

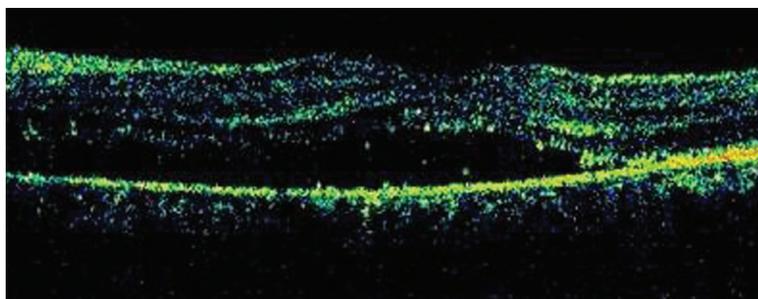


Figure 3: Optical coherence tomography of the left eye at the first examination shows an intraretinal and subretinal fluid accumulation.

At the 4th visit which was performed 2 months after the trauma an intravitreal Ozurdex[®] implantation was performed under subconjunctival anesthesia. At day 12 after implantation BCVA was 20/20, central macular thickness was 235 μm on OCT, IOP was 18 mmHg. One month after the implantation VA was 20/20, central macular thickness was 235 μm on OCT and IOP was 16 mmHg. The improvement of BCVA and macular edema were consistent at the last visit which was performed 6 months after the implantation (Figure 4a,b). There was no significant adverse effect such as uncontrolled IOP increase or cataract formation during this short follow-up.

In this case report, we performed an off-label usage of intravitreal Ozurdex[®] implantation. Written informed consent was taken from the patient. As Ozurdex[®] implantation is not in the permission list of the off-label drugs of Ministry of Health we did not take any further permission from Ministry of Health.

DISCUSSION

Damage to the macula is one of the most important causes of vision loss after eye trauma. Yu et al.,¹³ reported a study to describe the characteristics of macular lesions after eye trauma using Spectral Domain-OCT (SD-OCT). In the 24 eyes examined, a variety of lesions was demonstrated: intraretinal hemorrhage in 3 eyes; epiretinal membrane in 6 eyes; foveal detachment in 1 eye, and scar formation in 2 eyes (8.3%) combined with epiretinal membrane and diffuse macular edema or macular distortion; retinal pigment epithelium/choroidal rupture combined with choroidal neovascularization formation was identified in 2 eyes, macular hole in 4 eyes, photoreceptor inner/outer surface change or loss in 1 eye and fovea/whole macular atrophy in 8 eyes of which some cases had multiple lesions.

There have been a few histological studies into why a whitish coloration occurs in the retina after a blunt trauma.¹⁴⁻¹⁶ Berlin suggested that the whitish coloration might occur due to extracellular edema.¹⁷ In recent reports however, extracellular edemas were not observed.¹⁴⁻¹⁶ The disruption or fragmentation of the photoreceptor outer segment of the retina is the most common finding in histological studies of commotio retinae.¹⁴⁻¹⁶ Recently, OCT has been applied to the pathophysiological analysis of commotio retina, resulting in confirmation of previous histological studies of the abnormality of the retina outer segment layer.^{18,19} Although rare, macular edema is one of the possible pathology after blunt trauma. Park et al.,²⁰ reported on the anatomical and functional changes to the macula in nine patients suffering from commotio retina not accompanied by any other types of traumatic retinopathy. Only 3 out of the 9 injured eyes showed abnormal findings in SD-OCT images such as discontinuity of the inner/outer segment junction or abnormal hyper-reflectivity from the inner/outer segment junction and retinal pigment epithelium lines in the macula. During follow-up of 26 days, the commotio retinae resolved in all 9 eyes. The changes to the outer retinal region detected in 3 patients by SD-OCT were also resolved. Ahn et al.,²¹ investigated the morphologic characteristics of macular commotio retina using SD-OCT in 49 patients and developed a grading system for traumatic photoreceptor damage. They observed 4 distinct photoreceptor morphologic features: increase in inner segment/outer segment (IS-OS) junction reflectivity with

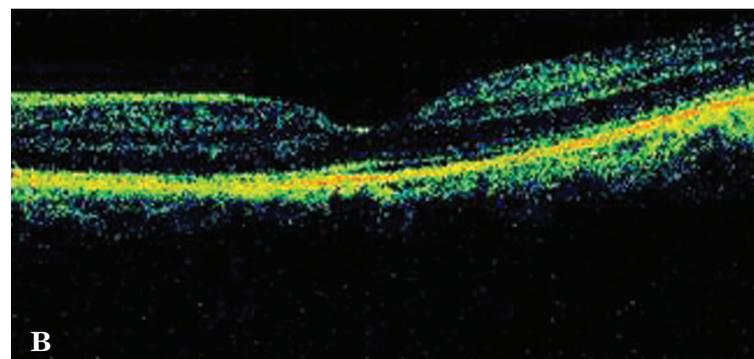
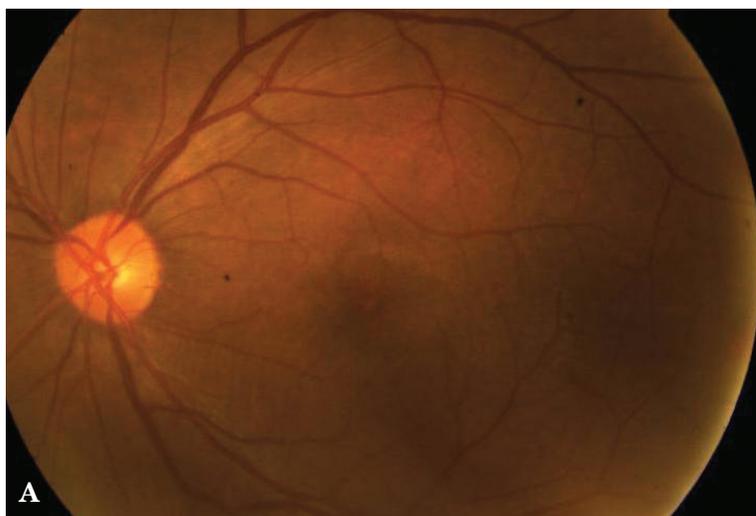


Figure 4a,b: Optical coherence topography of the left eye at 6th month after the intravitreal Ozurdex® implantation demonstrate a flat fovea (a). Colour fundus photograph of the left eye at the last examination (b).

the disappearance of the thin hyporeflective optical space in 8 patients (grade 1), cone outer segment tips line (COST) defect only in 5 patients (grade 2), COST and IS-OS junction defects in 16 patients (grade 3), and COST, IS-OS junction, and external lamina defects in 20 patients (grade 4). They concluded that eyes with higher grades at baseline had significantly worse visual and anatomic outcomes. Blanch et al.,²² reported the prognosis of 34 patients with macular commotio retinae and 58 patients with extramacular commotion retinae. They found a recovery of visual acuity in 74% of the patients with macular commotion retinae and in 95% of the patients with extramacular commotion retinae. Oladiwura et al.,²³ reported the OCT findings of a patient with commotion retinae due to blunt ocular trauma. They reported that the OCT showed outer photoreceptor segment disruption, retinal pigment epithelium inter-digitation, and intra-retinal edema of the outer nuclear layer. In our case report, the BCVA was not improved and the macular edema did not resolve within 2 months. Although, the main pathologic process inside the retinal tissue is in the outer part of the retina and this pathology mostly resolve spontaneously, we believe that an ophthalmologist should always consider that the retina is not an isolated tissue and the surrounding tissues might affect the retina after a blunt trauma.

Steroid therapy for commotio retinae was mentioned in a case report previously.²⁴ Mendes et al.,²⁴ reported a case of commotio retinae due to blunt trauma who successfully treated with high-dose pulse steroid therapy and consequent intravitreal triamcinolone injection.

The dramatic response to an intravitreal Ozurdex® implant might be the possible sign of the inflammatory process and vascular changes of the retina or the surrounding choroidea.

To the best of our knowledge, this is the first case report about the effect of Ozurdex® implant in the eye had persistent macular edema due to blunt trauma.

In conclusion, although it is very well known that the main problem is in the outer segment of the photoreceptors and a real extracellular fluid accumulation is a very rare associated finding in commotio retinae, our case demonstrates that some unknown parts of the pathogenesis of commotio retinae should be clarified with more prospective studies.

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