



Isolated Bilateral Macular Edema due to Le Fort Type I and Mandibular Fracture: A Case Report

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Abstract

We report the diagnosis and follow-up process of a case who had bilateral macular edema after blunt facial trauma. A 36-year-old male patient with Le Fort type I and mandibular fracture without direct ocular trauma referred to the ophthalmology clinic. Visual acuity was 0.1 in both eyes according to Snellen chart. Ocular examination was normal except bilateral macular edema. The patient did not have any prior systemic or neurological diseases. The patient did not have cotton-wool spots, retinal hemorrhage, or Purtscher flecken in the fundus examination. He used topical 0.1% nepafenac solution for I month. Visual acuity returned to normal after complete resolution of the macular edema at I month and did not recur in the follow-up. We think that this case may be an isolated macular edema due to facial trauma or an atypical presentation of Purtscher retinopathy. Although facial fractures and trauma may cause Purtscher retinopathy with involvement of different retinal structures, the findings in this case suggest that isolated involvement of macula can also occur in these injuries.

Keywords: Facial trauma, le fort fracture, macular edema, purtscher retinopathy

Introduction

Blunt facial trauma and fractures of the face can cause severe ocular complications (1). The fractures of the mid-face due to blunt trauma are classified according to Le Fort classification system which is described in 1901 and still used due to functional clinical significance (2-4). Le Fort type I fracture frequently occurs due to trauma to the lower maxillary rim. In this fracture, the maxilla is separated from the rest of facial bones in a horizontal plane above the roots of teeth and the hard palate (4). Cranial trauma without direct injury to the eye can cause Purtscher retinopathy which is

commonly associated with Purtscher flecken, cotton-wool spots, and intraretinal hemorrhages (5). Similar retinal changes can be seen in other trauma, systemic conditions such as acute pancreatitis, fat embolism syndrome, renal failure, and autoimmune disorders. This condition is named as "Purtscher-like retinopathy" (6). In this case report, we present a case with Le Fort type I fracture and mandibular fracture without direct ocular trauma who developed bilateral macular edema, but lack the typical findings such as cotton-wool spots and intraretinal hemorrhages seen in Purtscher retinopathy.

How to cite this article: Ramazan Zor K, Kucuk E, Samur Salbas C, Yildirim Bicer G. Isolated Bilateral Macular Edema due to Le Fort Type I and Mandibular Fracture: A Case Report. Beyoglu Eye J 2022; 7(2): 150-153.

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Submitted Date: December 16, 2021 Accepted Date: April 17, 2022 Available Online Date: May 27, 2022

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Case Report

A 36-year-old male patient who had blunt trauma to the face in a work accident and diagnosed as having Le Fort type I fracture and mandibular fracture referred to the ophthalmology clinic due to complaint of blurred vision. The patient was examined on the same day approximately 6 h after the accident. The patient told that he did not have a direct blow to his eyes and there was not any evidence of direct trauma to the ocular and periocular tissues. He also reported that he did not have any ocular disease before the accident and his vision had been quite well. He noticed blurred vision after the trauma. He had no known systemic disease such as diabetes mellitus, hypertension, or neurological diseases. The routine blood test results were also normal. The patient was ambulatory and external examination of the eye and periocular tissues were normal. The best-corrected visual acuity (BCVA) was 0.1 in both eyes. The pupils were reactive, there were no anisocoria or relative afferent pupillary defect. The biomicroscopic evaluation of the anterior segment was normal. Intraocular pressures were 12 mmHg in the right eye and 14 mmHg in the left eye. The fundus examination revealed macular edema with loss of foveal reflex but there were no cotton wool spots, intraretinal hemorrhages, or disk edema (Fig. 1). The optical coherence tomography (OCT) scan performed, and it revealed bilateral macular edema (Fig. 2). There was no sign of fracture in or near the optic canal, optic nerve injury, or retrobulbar hematoma in cranial and orbital CT scans (Fig. 3). The patient used topical nepafenac ophthalmic solution 0.1% 3 times a day for 1 month. At the end of 2 weeks, the BCVA was 0.9 in the right eye and 0.7 in the left eye. The OCT scan showed disappearance of macular edema and normal foveal contour. Visual field testing and MRI were performed and there was no finding suggestive of optic nerve injury. At the end of 1st month, the BCVA was 1.0 in the right eye and 0.9 in the left eye and the foveal contour was normal in OCT scans of both eyes (Fig. 4). At the end of 3rd month, the BCVA was 1.0 in both eyes and the macular OCT was also normal. The visual acuity remained stable and macular edema did not recur in the I-year follow-up. This study received approval from the Ethics Committee of Niğde Ömer Halisdemir University. The study was performed according to the tenets of Declaration of Helsinki and written and verbal informed consent was obtained from the patient.

Discussion

This patient we presented had bilateral macular edema after le Fort type I fracture and mandibular fracture without direct ocular injury. The anterior segment and ocular fundus examination did not reveal any other abnormality in this patient. Head trauma and associated fractures of the facial bones are one of the frequent causes of Purtscher retinopathy (3,7). Vascular endothelial damage and embolic occlusion

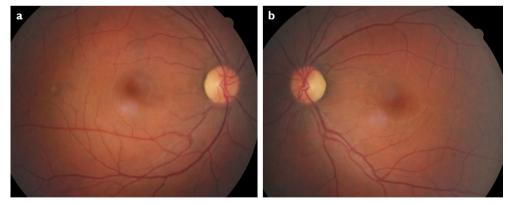


Figure 1. Fundus photographs of the right (a) and left (b) eye at the 1st day after trauma.

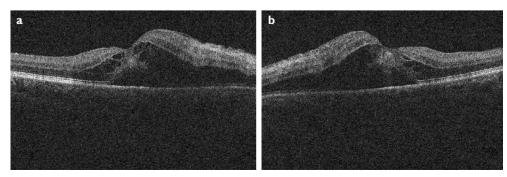


Figure 2. Macula OCT of the right (a) and left (b) eyes at the first day after trauma



Figure 3. Computed tomography (CT) scan of the patient at the first day after trauma.

of the pre-capillary arterioles were thought to be the underlying mechanisms in the pathogenesis of Purtscher retinopathy (6,8). The diagnosis of this disease is mostly clinical together with the history of etiological factors (5,6). Miguel et al. proposed a set of diagnostic criteria for Purtscher retinopathy which include Purtscher flecken, retinal hemorrhages, cotton-wool spots, an explanatory etiology, and an investigation compatible with diagnosis of Purtscher retinopathy (5). They stated that at least three of these five criteria is necessary for the diagnosis of Purtscher retinopathy. This patient we presented lacks the typical features of Purtscher retinopathy such as Purtscher flecken, cotton-wool spots, and retinal hemorrhages. Apparently, it does not conform to the criteria presented by Miguel et al. The prevalence of macular edema was reported to be 22% in patients with Purtscher retinopathy (5). Localized endothelial damage, complement activation, coagulation, and occlusion of capillaries by emboli of leucocytes and fibrin were proposed as the mechanism underlying macular edema and the other retinal changes seen after trauma (8,9). The lack of fundus fluorescein angiography of the patient in the first presentation of the patient limits our discussion of pathophysiology of macular edema in this case, but we think that similar factors may be the cause of edema in this case. There are also reports describing macular edema in Purtscher-like retinopathy, but all of these cases had typical findings of Purtscher retinopathy such as Purtscher flecken, cotton-wool spots, and retinal hemorrhage (8). Our case differs from them by lack of typical findings of Purtscher retinopathy. We think that this case we presented may be an atypical presentation of Purtscher retinopathy or it may be isolated macular edema due to facial trauma and fractures of facial bones.

A similar case was presented by Sigona et al.(9) The case in their report had reversible macular edema after a traffic accident. The authors reported that the patient did not have any ocular, head, torso, or limb injuries which are different from our case. Similar to our case, the patient used a topical non-steroidal anti-inflammatory drug and the isolated bilateral macular edema resolved in 2 weeks which is confirmed by OCT and did not recur in 1-year follow-up. The authors concluded that rapid bilateral visual loss may occur after indirect trauma due to macular edema which has a good prognosis for visual recovery.

Male gender and trauma were reported to be good prognostic factors for visual recovery after Purtscher retinopathy (5). Similarly, despite the prominent macular edema at the initial examination in this male patient, the BCVA returned to almost normal after resolution of the macular edema as determined by OCT. There is no standard treatment for traumatic retinopathies (5,6). Mostly, observation and treatment of underlying pathology is suggested. Nepafenac, a non-steroidal anti-inflammatory drug is used in the prophylaxis and treatment of retinal edema due to various retinal diseases (10). Since the only finding that could explain the visual loss in this patient was macular edema, we observed

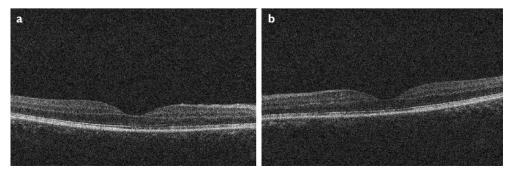


Figure 4. Macula OCT of the right (a) and left (b) eyes at the Ist month.

the patient with topical nepafenac, in this case. The macular edema resolved and BCVA returned to normal without additional treatment.

The lack of fundus fluorescein angiography is a limitation of the study. We did not perform fundus fluorescein angiography in the acute setting, and it became unnecessary in the follow-up due rapid resolution and lack of recurrence of the macular edema. This also limited our discussion of pathophysiology of macular edema in this case.

We presented a case who had reversible macular edema after le Fort type I fracture and mandibular fracture without direct ocular trauma. This case may be an isolated macular edema due to indirect trauma or an atypical presentation of Purtscher retinopathy. Blunt facial trauma may cause isolated macular edema and central visual loss.

Disclosures

Informed consent: Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.

Peer-review: Externally peer-reviewed.

Conflict of Interest: None declared.

Authorship Contributions: Concept – E.K., K.R.Z., G.Y.B., Ç.S.S.; Design – E.K., K.R.Z., G.Y.B., Ç.S.S.; Supervision – E.K., K.R.Z., G.Y.B., Ç.S.S.; Materials – G.Y.B.; Data collection and/or processing – E.K., K.R.Z.; Analysis and/or interpretation – E.K., G.Y.B.; Literature search – Ç.S.S.; Writing – K.R.Z.; Critical review – G.Y.B.

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