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Traumatic Maculopathy 6 Months after Injury: A Clinical Case Report

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Key Words

Traumatic maculopathy · Commotio retinae · Intravitreal triamcinolone acetonide

Abstract

Purpose: This study aims to report a case of traumatic maculopathy in a 12-year-old male following blunt trauma in his left eye (LE) who presented 6 months after injury. **Methods:** Retrospective and descriptive case report based on data from clinical records, patient observation and analysis of diagnostic tests. **Results:** A previously healthy, 12-year-old male presented for a routine visit with complaints of a 2-month history of decreased visual acuity in his LE. Six months before the initial visit, he suffered blunt trauma to the LE during a struggle and had no medical observation. At the visit, best-corrected visual acuity (BCVA) in the LE was counting fingers and in the right eye, it was 20/20. Fundus examination of the LE showed a central macular lesion of 1 disc diameter with fibrosis, increased retinal thickness and intraretinal hemorrhage. Optical coherence tomography showed disruption of the inner/outer segment (IS/OS) photoreceptor junction, increased reflectivity, cell infiltration of the retinal wall and retinal pigment epithelium detachment. Retinal thickness was 289 μm at the site of the lesion. A fluorescein angiogram revealed early impregnation and late diffusion. High-dose steroid pulse therapy (intravenous methylprednisolone 500 mg for 3 days and oral prednisolone 30 mg, tapering for 10 days) was done. LE BCVA increased to 20/200, and retinal thickness decreased by 71 μm 1 week after treatment. Off-label intravitreal triamcinolone (IVTA; 0.05 ml/2 mg) was administered 2 weeks after oral treatment in an attempt to achieve additional improvement. Three weeks after IVTA, LE BCVA improved to 20/150 and retinal thickness decreased by 10 μm . Three months after the initial visit, LE BCVA was 20/125 and retinal thickness 208 μm . **Conclusion:** We present a case of commotio retinae caused by an ocular blunt trauma 6 months before, with loss of BCVA. BCVA improved after oral steroids and IVTA. Nevertheless, fibrosis and disruption of the IS/OS junction in the macula limited the gain of BCVA.

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Introduction

Comotio retinae, also known as Berlin's edema, is frequently observed after ocular blunt trauma. It is characterized by transient grey-white retinal coloration and a decrease in best-corrected visual acuity (BCVA). It may be confined to the macula or involve areas of the peripheral retina [1]. Depending on the severity of the trauma, histopathology can reveal areas of disruption of the inner/outer segment (IS/OS) junction, hyperreflectivity of the overlying retina, pigment disorders and retinal atrophy [2]. Extracellular edema and glial swelling have been proposed for the transient grey-white retinal coloration [3]. There may be associated injuries such as retinal hemorrhages (preretinal, intraretinal, subretinal) and choroidal rupture [4]. Patients may have no visual complaints if only the peripheral retina is involved, but they may have severe visual loss when macular lesions are present [2]. Mild cases present transient visual loss and recover spontaneously with minimal sequelae [5], but more severe cases may be associated with permanent visual loss [1, 4, 6]. A study including 30 patients with damage to the posterior pole showed that visual acuity achieved maximum recovery up to 6 months after initial presentation [7, 8]. Optical coherence tomography (OCT) allows visualization of structural abnormalities of the macula and their evolution. The OCT findings usually show disruption of the IS/OS junction and corresponding hyperreflectivity [9], defects at the cone OS tips or damage to the external limiting membrane [10]. It is hypothesized that severe photoreceptor damage leads to worse visual and anatomic outcomes [10].

Case Presentation

A previously healthy, 12-year-old male presented for a routine visit with complaints of a 2-month history of decreased visual acuity in his left eye (LE). Six months before the visit, he suffered blunt trauma to the LE during a struggle, with no medical observation. At the initial visit, BCVA in the LE was counting fingers and in the right eye, it was 20/20. He had neither restricted eye movements nor afferent pupillary defects. Biomicroscopic examination of the anterior segment was normal. Fundus examination of the LE showed a central macular lesion of 1 disc diameter with fibrosis, increased retinal thickness and intraretinal hemorrhage (fig. 1, left). Peripheral retinal examination was normal. Spectralis OCT (Heidelberg Engineering Inc., Heidelberg, Germany) showed disruption of the IS/OS junction with corresponding increased reflectivity, loss of the outer nuclear layer, cell infiltration of the retinal wall and subretinal fibrosis. Retinal thickness was 289 μm at the site of the lesion (fig. 1, right). A fluorescein angiography (FA) revealed early impregnation with no diffusion, increasing with time (fig. 2). With support from Pediatrics, off-label, high-steroid pulse therapy (intravenous methylprednisolone 500 mg for 3 days and oral prednisolone 30 mg, tapering for 10 days) was done, resulting in an increase of LE BCVA to 20/200 and a decrease of retinal thickness by 71 μm to 218 μm 1 week after treatment. In an attempt to achieve additional improvement without undesired systemic effects, off-label intravitreal triamcinolone acetonide (IVTA; 0.05 ml/2 mg) was administered 2 weeks after oral treatment. LE BCVA improved to 20/150, and retinal thickness decreased by 10 μm 3 weeks after IVTA. Three months after initial presentation, LE BCVA was 20/125 and retinal thickness was 208 μm . However, the alterations in fundoscopy and OCT remained (fig. 3).

Discussion

Posterior segment damage after blunt trauma frequently coexists with anterior segment damage [7]. In the present case, there is no data regarding the status of the anterior segment at the time of injury since the patient was observed only 6 months after trauma. Patients with a damaged IS/OS junction are more likely to have persistent photoreceptor defects, with irreversible photoreceptor loss [10]. In our case, development of fibrosis, alterations in the IS/OS junction and loss of the outer nuclear layer that showed on OCT (fig. 1, right; fig. 3) limited the recovery of the retinal lesion remaining without treatment for a long period. From the FA, the authors concluded that there was impregnation but not diffusion (fig. 2). Therefore, the diagnosis was a fibrotic lesion and not a neovascular membrane. The OCT scan showed increased retinal thickness, probably due to posttraumatic inflammatory edema. With support from Pediatrics, off-label, high-dose steroid pulse therapy (intravenous methylprednisolone 500 mg for 3 days and oral prednisolone 30 mg, tapering for 10 days) was done, resulting in an increase of LE BCVA to 20/200 and a decrease of retinal thickness by 71 μm to 218 μm 1 week after treatment. In an attempt to achieve additional improvement without undesired systemic effects, off-label IVTA (0.05 ml/2 mg) was administered 2 weeks after oral treatment, with recovery of BCVA to 20/150 and a decrease of retinal thickness by 10 μm 3 weeks after IVTA. The recovery was most probably due to therapy since 6 months after the initial injury no spontaneous recovery was expected. The fact that this patient has been followed for 3 months after the treatment and that the condition did not worsen seems to indicate a fibrotic lesion rather than a neovascular membrane. A PubMed search was performed using the following terms: commotio retinae and treatment. To our knowledge, no other report with such a delay of treatment and using off-label, high-dose steroid pulse therapy and off-label IVTA in a youngster has previously been published. This case seems to indicate that it is worth treating lesions from trauma without spontaneous resolution, even when they have been progressing for as long as 6 months. Nevertheless, when structural damage occurs, recovery may be limited.

Disclosure Statement

The authors have no proprietary interests, financial support or other conflicts of interest to report.

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Fig. 1. Retinography and OCT of the LE at presentation, revealing a central macular lesion of 1 disc diameter with fibrosis, showing disruption of the IS/OS junction with corresponding increased reflectivity, infiltration of the retinal wall and retinal pigment epithelium detachment. Retinal thickness was 289 μm at the site of the lesion.

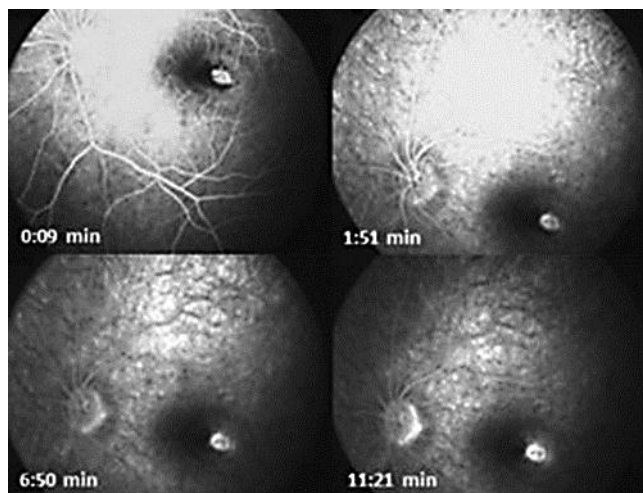


Fig. 2. FA of the LE at presentation, showing impregnation but not diffusion.

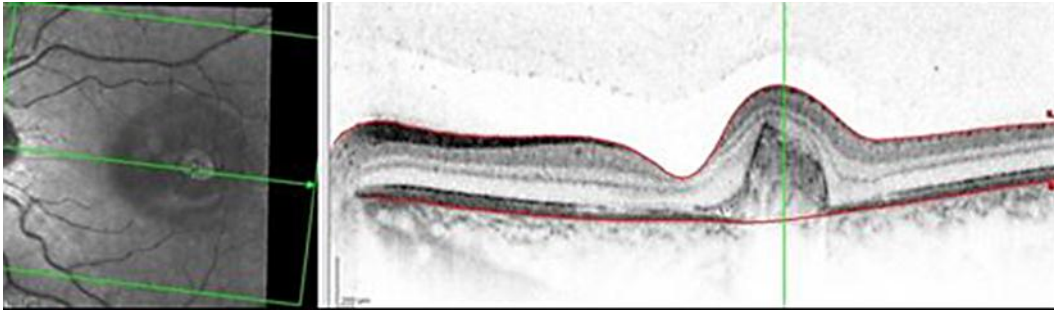


Fig. 3. Last OCT of the LE 3 months after initial presentation, showing a decreased retinal thickness (208 μm).