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A Traumatic Macular Hole in a Young Soldier after an Explosion Injury

Ke-Hung Chien, Ching-Long Chen, and Jiann-Torng Chen*

Department of Ophthalmology, Tri-Service General Hospital, National Defense Medical Center, Taipei, Taiwan, Republic of China

Although ocular trauma is usually not fatal, it has adverse sequelae. Herein we report a case of a 23-year-old soldier who presented at our emergency room with sudden-onset blurred vision in his left eye (OS) after an explosion injury. His best-corrected visual acuity (BCVA) was 6/6 for the right eye (OD) and 6/60 for the OS. Pulse corticosteroid therapy was administered because of suspected traumatic optic neuropathy. Examination of the fundus three days after admission revealed macular hemorrhage, commotio retinae, and a stage 1-B occult macular hole. He did not recover his visual acuity, and the macular hole persisted for two months. This case emphasizes the need for eye protection when soldiers are exposed to explosions.

Key words: ocular trauma, traumatic macular hole, explosion injury.

INTRODUCTION

A macular hole is a full-thickness retinal defect that involves the fovea and severely impairs central visual acuity. Clinical findings include blurred vision, distortion of vision, and central scotoma. In women, spontaneous macular holes usually occur in the sixth to eighth decade[1]. In men, traumatic macular holes are usually incurred between the ages of 11 years and 30 years. Knapp (1869) attributed traumatic macular holes to direct contusion of the eyeball, but it is now recognized that macular holes are caused by cystoid degeneration of the macula, a secondary effect of ocular trauma. At present, only 9% of macular holes are directly or indirectly associated with ocular trauma. Reports of ocular traumas caused by explosions are rare. Herein we describe a stage 1-B occult macular hole in a soldier who incurred indirect ocular trauma as a result of an explosion.

CASE REPORT

A 23-year-old soldier experienced blurred vision OS immediately after an explosion injury. He was fully conscious during ambulance transport to the hospital and had hypertension (156/90 mmHg), tachycardia (102 beats per minute), and mild tachypnea (22 breaths per minute). However, his oxygen saturation was 98%. Physical examination revealed that there were no bony fractures or painful limitation to the movement of his extremities. He had one laceration about 1.5 cm long over his lateral canthus and swelling with ecchymosis over the left periorbital region. An emergent brain CT scan and a cervical spine X-ray did not reveal any other injuries. Ophthalmologic emergent consultation showed that his uncorrected visual acuity (UCVA) was 6/6 OD and 6/60 OS; his intraocular pressure was 16 mmHg OD and 13 mmHg OS. Slit-lamp biomicroscopy showed an intact eyeball contour, hyphema, corneal edema, severe subconjunctival hemorrhage, and chemosis in the affected eye. There was a relatively mild afferent pupillary defect of the OS. We were unable to perform dilated fundus examination because of vitreous hemorrhage. A B-scan disclosed commotio retinae with vitreous hemorrhage but without retinal detachment. The results of the OD examination were unremarkable. As traumatic optic neuropathy was suspected, we administered pulse corticosteroid therapy and topical cycloplegics, and ordered the patient to rest in a sitting position. Three days later, the hyphema, corneal edema, and vitreous hemorrhage resolved. Visual acuity improved (BCVA = 6/30 OS). Dilated fundus examination and optical coherence tomography (OCT) showed macular hemorrhage, Berlin's edema and a 240×300 μm stage 1-B occult macular hole (Figures 1, 2A, and 2B). After tapering the dose of systemic corticosteroid, we discussed the management of the traumatic macular hole with the patient and decided to proceed

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*Corresponding author: Jiann-Torng Chen, Department of Ophthalmology, Tri-Service General Hospital, ational Defense Medical Center, No. 325, Sec. 2, Cheng-Gong Road, Taipei 114, Taiwan, Republic of China. Tel: +886-2-87927165; Fax: +886-2-87927164; E-mail: jt66chen@ms32.hinet.net

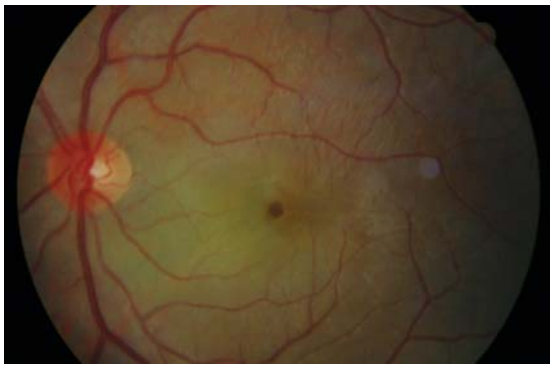

Fig.1 Three days after the explosion injury, color fundus photography showed circular macular hole which is measured about 240×300 μm^2 with focal retinal edema inside the arcades, especially the inferior nasal quadrant.

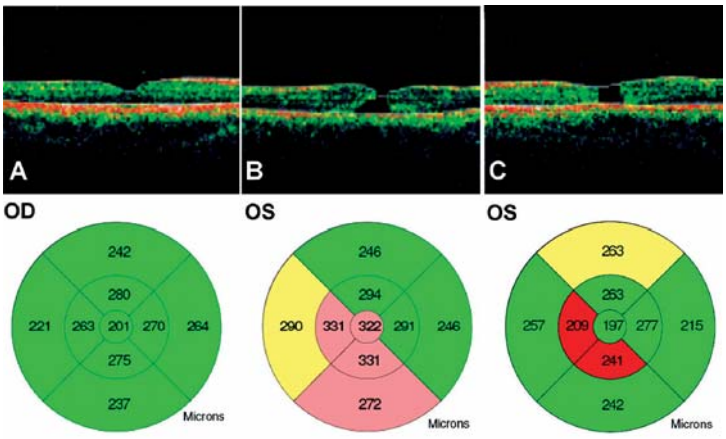

Fig.2 Three days after the explosion injury, OCT disclosed normal foveal thickness (201 μm) OD (2A) and stage 1-B occult macular hole with circumferential retinal edema which demostrated increasing parafoveal thickness (322 μm) OS (2B). Visual acuity was 6/60 OS at that time. Two months later, OCT showed persistent stage 1-B occult macular hole with resolved retinal edema (foveal thickness 197 μm) (2C). The patient improved his visual acuity to 6/20 OS.

with cautious observation. Two months after the injury, the patient's BCVA improved to 6/20 OS. According to OCT, the size of the macular hole had not diminished, but the retinal edema had resolved (foveal thickness 197 μm; Figure 2C).

DISCUSSION

During war or armed conflicts, ocular injuries constitute 5%-13% of all injuries[2]. The majority of ocular injuries are associated with fragmentation of accessories such as glasses. Explosions usually cause severe injury. An explosion results in a blast wave preceded by a positive phase (increased air pressure) and a negative phase (negative air pressure). The most common types of explosion injuries are (1) injury to the lungs, gastrointestinal system, eardrums, or central nervous system, which are caused by the blast wave itself, (2) cuts or tissue penetration caused by flying objects propelled by the wave, (3) injury of part or all of the body caused by the acceleration effect, and (4) thermal injury[3].

In 1869, Knapp observed a macular hole in an ocular trauma patient and attributed all macular holes to ocular trauma[1]. In 1988 and 1995, Gass et al. described a classification system for macular holes based on biomicroscopic and anatomic characteristics. They believed that tangential vitreous traction was the underlying cause of idiopathic macular holes. At present, three mechanisms are thought to cause traumatic macular holes: (1) application of a concussion force to the posterior pole or the equatorial region, resulting in macular rupture; (2) cystoid formation beneath the macula as proposed by Coats; and (3) sudden-onset posterior vitreous detachment.

Traumatic macular holes caused by direct force are widely reported. Blunt injuries incurred during sports, games, and traffic accidents, and by paintball pellets have been described[4]. There are fewer reports of injuries caused by indirect force than by direct force. Only 5%-11% of explosion or blast injury victims who suffer ocular trauma die[5]. Our patient, a 23-year-old male soldier, had ocular trauma caused by an explosion injury. We believe that his injuries were mainly caused by the blast wave and that the macular hole was caused by a rapid change in axial length subsequent to a sudden expansion of air. We observed commotio retinae and macular hemorrhage in the initial dilated examination and cystoid formation beneath the macula during the OCT. In contrast to the irregularly shaped macular hole evident upon vitreous traction, a small, round macular hole of full thickness was observed several days after resolution of the macular hemorrhage. Fluorescent angiography revealed focal leakage from the swollen retina. We believe that the transient compression of the globe changed the axial length and exerted a tangential force on the macula, which altered the permeability of focal retinal vessels and thus induced macular and focal retina edema and cystoid formation. As a thickened fovea was observed during macular OCT, we believe that the round macular hole observed after resolution of the macular hemorrhage was caused by the traction force of the edematous retina.

Stage-I traumatic macula holes are thought to resolve spontaneously, whereas early vitrectomy (within 6 months) is suggested for holes of other stages[6]. Amari et al. conducted a study on 23 eyes and reported that surgery on 22 (96%) eyes was successful and that an improvement in visual acuity of more than two lines occurred in 20 (87%) eyes. He concluded that the prognosis for traumatic macular holes is better than that for spontaneous macular holes. Vitreous surgery should be considered if there is no chorioretinal atrophy in the foveal region. Our patient was kept under observation for a few months. During the follow-up visit, we observed that the macular hole had persisted but there was no vitreous traction or vitreoretinopathy.

CONCLUSION

Most macular holes observed in the early 1920s resulted from ocular trauma incurred during war or conflicts, whereas idiopathic macular holes are the most common type at present. Traumatic macular holes occur mainly as a result of industrial accidents and sports injuries. The few reports on explosion injury have concerned eyelid or eyebrow lacerations and corneal abrasions[5]. In our patient, the macular hole was not accompanied by systemic injuries. The incidence of eye injuries caused by explosions could be lessened by decreased proximity to the area of potential danger and by orientation away from windows or fragile objects.

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