CASE REPORT

RETINAL SEQUELAE OF PRIMARY OCULAR BLAST INJURIES

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Abstract

Primary ocular blast injury is an uncommon and disputed phenomenon. As personal ballistic protection of the head and torso improves for soldiers, increasing numbers of injuries to the unprotected areas such as the face and eyes may be expected; similarly the increase in the use of improvised explosive devices by insurgent terrorists in Iraq is increasing the number of primary blast injuries being seen by the Defence Medical Services. We report a rare case of primary blast injury to the eye and briefly discuss the literature on the subject.

Introduction

Ocular blast injury is usually a fragment effect and any primary blast damage is overshadowed by the secondary effects: indeed, it is still debated as to whether primary blast injury to the eye truly exists or whether all blast damage is of a secondary nature. We report a case of primary blast injury of the right eye sustained from an improvised explosive device (IED) in Afghanistan, whose later vitreo-retinal care took place in our unit.

Case Report

A 43 year old male soldier was injured by a pipe-bomb blast in Afghanistan. The IED exploded four metres away from his relatively protected position within an armoured Landrover; his head was leaning directly against the Landrover's right hand door pillar on the same side as the blast. He suffered multiple injuries including a ruptured right tympanic membrane, a right orbital floor fracture, a maxillary antral haematoma, facial lacerations and contusions to his right upper arm and leg. There was no specific mention of ocular injury or visual disturbance.

Treatment of his non-ocular injuries and initial eye examination were performed in Afghanistan and he was then transferred to our unit 5 days after the injury. His visual acuity had not changed over this time at counting fingers, right and 6/5, left. He had right traumatic mydriasis and a vitreous haemorrhage. The globe was noted to be intact with no penetrating injury or rupture.

After seven days, his visual acuity improved to 6/24 on the right and 6/5 on the left, with no afferent pupillary defect and intraocular pressures of 18mmHg and 16mmHg on the right and left respectively. His optician's report of three months prior to the injury gave his visual acuity as 6/5 on each side. On fundoscopy the right retina had areas of pre-existing lattice degeneration, an area of superotemporal commotio retinae with a tear within it and an inferior vitreous haemorrhage. Breaks were noted in Bruch's membrane in the macular region (lacquer cracks) (Figure 1), confirmed on fluorescein angiography (Figure 2). The left eye appeared normal with no lacquer cracks.

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The patient received argon laser retinopexy around the retinal tear.

Three months later the visual acuity was right, 6/18 left, 6/5 and the commotio retinae had resolved to leave an area of retinal atrophy. The clinical picture had not changed when reviewed one year, at which stage he was discharged from ophthalmic follow up.

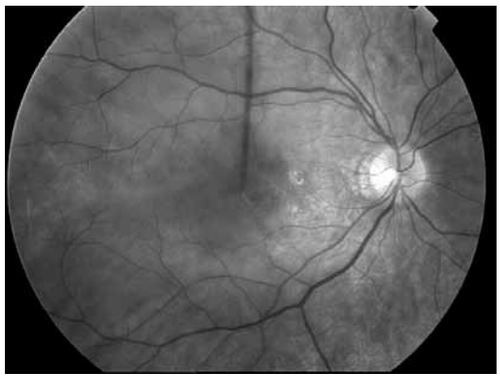

Figure 1: Fundus photograph demonstrating the changes on the right macula.

Figure 2: Fundus fluorescein angiogram, demonstrating the hyperfluorescence of the breaks to Bruch's membrane.

Discussion

An explosion generates a near instantaneous overpressure that travels through the air at a speed faster than sound and which is responsible for primary blast injury (PBI). The classification of blast injuries is outlined in Table 1. Short duration shock waves

Table 1 Classification of blast effects (2)

Primary	Interaction of the blast wave with the body
Secondary	The effects of energized fragments from either the bomb or the environment
Tertiary	The effects of the blast wind including bodily displacement and structural collapse
Quaternary	All other effects including burns, psychological effects and inhalantional injury

with a high peak dynamic overpressure are coupled into the body and propagated as stress waves depositing energy at the interface of tissues of different densities. Injury is then caused directly by the pressure differential across delicate membranes, by spalling and by imploding the incident tissues (1)

Modern theories of the pathogenesis of PBI were formulated after the Second World War, based largely on the results of German research (3). Though this research was not specific to the eye, ophthalmologists concerned with the care of high explosive ocular injuries attributed several cases to PBI (4). It is rare to find a case of ocular PBI without any evidence of a concomitant secondary blast injury from debris and this may be due to the relative lack of marked tissue density boundaries encountered as a blast wave traverses the eye (1).

Bellows was the first to categorise primary ocular blast injuries. There were 13 cases due solely to a primary blast injury. The visual outcome was poor with only 4 achieving a visual acuity of 6/9 or more. Injuries to the optic nerve, choroid and retina carried a worse prognosis (5). The most recent report in 1992 was of a man who suffered a hyphaema and iridocorneal angle recession with no other injury after exposure to a terrorist bomb (6). Our case was protected from secondary injuries by the door pillar of his vehicle.

Body armour against primary and secondary blast injuries is increasingly worn on the battlefield to protect the torso, but the face and eyes are left relatively exposed for reasons of comfort and improved vision. As increased body protection makes an explosion more survivable, then it is likely that more cases of major eye injury will survive to be treated. In a similar vein, if ocular protection against fragment injuries is worn the chance of a SBI is greatly reduced (7), although it offers little in the way of protection against blast waves.

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