Ryan et al. Int J Ophthalmol Clin Res 2015, 2:5
ISSN: 2378-346X

International Journal of
Ophthalmology and Clinical Research

Research Article: Open Access

The Cornea after Primary Blast Trauma

Denise S Ryan[1]*, Bennett J Oberg[2], Michael J Mines[2], Rose K Sia[1], Keith J Wroblewski[2], Marcus Colyer[2], Dal Chun[3], Eric Weichel[3] and Kraig S Bower[4]

[1]Warfighter Refractive Eye Surgery Program and Research Center at Fort Belvoir, Fort Belvoir Community Hospital, Fort Belvoir, VA, USA

[2]Ophthalmology Service, Walter Reed National Military Medical Center, Bethesda, Maryland, USA

[3]Retina Group of Washington, Greenbelt, Maryland, USA

[4]The Wilmer Eye Institute, Green Spring Station, Baltimore, Maryland, USA

***Corresponding author:** *Denise S Ryan, MS, Warfighter Refractive Eye Surgery Program and Research Center at Fort Belvoir, Fort Belvoir Community Hospital 9300 DeWitt Loop, Fort Belvoir, VA 22060, USA, E-mail: denise.s.ryan.ctr@mail.mil; dryan@hjfresearch.org*

Abstract

Purpose: This study examined the seemingly uninjured eye of soldiers with blast injuries involving the head, face, and fellow eye to obtain insight into primary blast ocular injuries.

Methods: Serial exams were performed on nineteen U.S. Army Soldiers with a combat-related blast injury involving the head, face and either eye for effects of primary blast trauma in the clinically unaffected eye. Outcome measures included corrected distance visual acuity (CDVA), endothelial cell density (ECD), corneal hysteresis (CH), corneal resistance factor (CRF), cornea compensated intraocular pressure (IOPcc), and central corneal thickness (CCT). The Wilcoxon signed ranks test was used to compare initial versus final values.

Results: CDVA improved over the course of follow up: median logMAR -0.04 (-0.12 to 0.18) to -0.12 (-0.18 to 0.12), $p = 0.015$. There were no significant changes for either ECD or CCT over time. Of the 19 patients, 10 had measurements of CH, CRF, IOPcc, and CCT, with no significant changes over time. Median CH and CRF were lower than reported in the literature.

Conclusions: In this case series, ocular health in terms of ECD, IOPcc, and CCT did not change over time after exposure to primary blast injury. CH and CRF were noted to be lower than averages reported in the literature. Direct injury from secondary blast injury remains the most significant threat to the eye but longitudinal studies are recommended to determine the long term prognosis of eyes exposed to blast shock waves.

Keywords

Primary ocular blast injury, Trauma, Cornea, Endothelial cell count

Abbreviations

CCT: Central Corneal Thickness, CDVA: Corrected Distance Visual Acuity, CH: Corneal Hysteresis, CRF: Corneal Resistance Factor, ECD: Endothelial Cell Density, IED: Improvised Explosive Devices, IOPcc: Cornea Compensated Intraocular Pressure, logMAR: Logarithm of The Minimum Angle of Resolution, PBI: Primary Blast Injury, RPG: Rocket Propelled Grenades

Introduction

Insurgents are changing the face of modern warfare by using unconventional tactics and creating increasingly dangerous explosive devices. The use of improvised explosive devices (IEDs), rocket propelled grenades (RPGs), mines, mortars, and suicide vests are exposing a larger number of soldiers and civilians to devastating blasts. These blasts render varying types of injuries characterized as primary, secondary, tertiary or quaternary depending on the mechanism of injury. Multisystem injuries are common due to the damaging force of explosives, and the potential and severity for these are dependent on size, environment, and distance from the explosive source [1-3].

The eye, although less than 1% of the body surface area, is also at risk of injury [1]. Traditionally ocular blast injuries have been characterized as primary or secondary injuries. Secondary blast injuries result from penetration of fragmentary projectiles dispersed by the blast and are the most frequent cause of ocular injuries [1-6]. Ocular trauma due to penetration of the cornea by supersonic debris is well-established, but the effects due to primary blast injuries are less well-known. Primary blast injuries (PBI) result from a pressure differential created by the passage of the blast wave. It is this wave that delivers the primary injury upon its impact with body tissues. The blast wave can interact with the body in two ways causing either stress damage or shear damage [8]. Stress damage occurs at the interface of body tissues of differing densities and waves travelling through differing media may have their path altered by the density gradient. Injury is most severe at the surface and as the wave continues through tissue shedding energy, it causes spall-like stress damage and shearing resulting in tissue disruption [7,9-12]. Transverse shearing waves are responsible for damage such as globe rupture due to compression and deformation [8,9].

Some instances of ocular PBI may go unnoticed due to the morbidity of a blast or the lack of a visually significant injury. A study modeling PBI showed that the structure and shape of the eye as well

ClinMed International Library

Citation: Ryan DS, Oberg BJ, Mines MJ, Sia RK, Wroblewski KJ, et al. (2015) The Cornea after Primary Blast Trauma. Int J Ophthalmol Clin Res 2:040

Received: September 04, 2015: **Accepted:** October 13, 2015: **Published:** October 16, 2015

as the differing interfaces are ideal for propagating the pressure wave. Using the model, structures posterior to the vitreous base were shown to bear higher pressures from PBI compared to the cornea resulting in retinal and optic nerve damage [13]. Vascular and neural damage to the retina may be more easily detected than the cornea through which both incident and rebounding pressure waves traverse. The purpose of this study was to observe the cornea in the seemingly uninjured eyes of Soldiers exposed to a blast for PBI.

Methods

U.S. Army Soldiers with a combat related blast injury enrolled in an ongoing Walter Reed Army Medical Center Institutional Review Board approved study underwent standard anterior segment testing, when possible, at the Ophthalmology service, Walter Reed Army Medical Center, Washington, D.C. Participants in this limited series were included if they had a monocular injury and were serially examined after sustaining a blast injury to the head, face and eye. Data for analysis included age, gender, injury, use of protective eyewear, and duration of follow up. Measurement of corrected distance visual acuity (CDVA) and corneal measurements of the seemingly uninjured eye were analyzed. Corneal evaluation included the following: automated endothelial cell density (ECD), central corneal thickness (CCT), corneal hysteresis (CH), corneal resistance factor (CRF), and cornea compensated intraocular pressure (IOPcc).

Corrected distance visual acuity (CDVA)

CDVA was assessed using a projector-type Snellen chart viewed at a distance equivalent to 20 feet (6 meters). Best optical correction was determined by refractometry using a manual mechanical phoropter (Reichert Ophthalmic Instruments Inc., Depew, NY). Snellen acuity results were converted to logarithm of the minimum angle of resolution (logMAR) for data analysis.

Corneal hysteresis, corneal resistance factor, cornea compensated intraocular pressure (CH, CRF, IOPcc)

CH, CRF, and IOPcc were measured using the ocular response analyzer (ORA, Reichert Ophthalmic Instruments Inc., Depew, NY) at the initial enrollment visit and each subsequent visit. Participants were instructed to fixate on a central light while the ORA's auto-aligning air pulse was delivered. Four waveforms were recorded and the average for each parameter was used for the data analysis.

Central corneal thickness (CCT)

CCT measurements were obtained using the minimum corneal thickness value as measured by the Orbscan II corneal topography (Orbtek, Bausch & Lomb, Salt Lake City, UT) or the pachymetry map using the anterior segment optical coherence tomography (AS OCT) System (Visante, Carl Zeiss Meditec Inc., Dublin, CA).

Endothelial cell density (ECD)

Endothelial cells were imaged and counted using the Confoscan 4 confocal microscope (Nidek Inc., Fremont, CA). ECD scans were performed using a 40x lens and analyzed using the CS4 NAVIS (Nidek Advanced Vision Information System). A region of interest (ROI) was selected to include the area of cells most suitable for automated ECD analysis. Automatic cell counts of selected ROI were manually edited to optimize identification and counting of cells. Data generated included ECD and the morphometric indices of polymegathism (degree of change in cell area), and pleomorphism (variation in cell shape based on percentage of hexagonal cells).

Analysis

Statistical analyses of initial and final results for CDVA, ECD, CCT, CH, CRF, and IOPcc were performed using SPSS version 16.0 for windows (IBM Corp., Armonk, NY). A Wilcoxon signed ranks test was used to compare the initial and final values. A Mann-Whitney test was used to compare the use of eye protection, and associated head or face injury in terms of the change in CDVA, ECD, polymegathism, pleomorphism, CH, CRF, IOPcc, and CCT. A p-value of < 0.05 was considered statistically significant. Data are presented as median and range, unless otherwise indicated.

Results

Nineteen male Soldiers with a mean age of 28.8 ± 8.2 years (range 22 to 50) had measurements taken on average 124 ± 149 days (range 7 to 600) post injury. The follow up visit averaged 425 ± 340 days (range 115 to 1393) post injury. Table 1 presents an overview of injury findings. Blast trauma was caused by IEDs in 79% of patients (15 patients), mortars in 11% (two patients), RPGs in 5% (one patient), and mines in 5% (one patient). The use of eye protection at the time of injury was confirmed by 26% of injured soldiers (five patients), not worn by 42% (eight patients), and unknown in 32% (six patients).

Table 1: Overview of injury findings.

Number	Age	Mechanism of Injury	Eye Protection	Outcome in fellow eye	Head or Face Injury	Initial Visit (days)	Final Visit (days)
1	25	IED*	Unknown	Globe laceration, IOFB†, traumatic cataract	No	46	115
2	31	IED	No	Globe laceration, IOFB, loss of lens	No	74	472
3	24	IED	Unknown	Lid lacerations, traumatic cataract	Yes	267	562
4	27	IED	No	Globe laceration, IOFB	Yes	360	1393
5	38	IED	Unknown	Globe laceration, IOFB, traumatic cataract	Yes	9	969
6	22	RPG‡	No	Globe laceration, IOFB	No	21	296
7	26	IED	Yes	Globe laceration, IOFB, retained corneal foreign bodies, traumatic cataract	Yes	106	193
8	24	IED	No	Globe laceration, IOFB	Yes	163	276
9	23	IED	Yes	Globe laceration, IOFB, traumatic cataract	Yes	77	140
10	48	IED	Yes	Globe laceration, IOFB, loss of lens	Yes	600	926
11	30	IED	Yes	Globe laceration, IOFB, loss of lens	Yes	109	351
12	30	IED	Yes	IOFB	Yes	9	220
13	25	IED	No	Globe laceration, IOFB, traumatic cataract	No	8	248
14	22	IED	No	Globe laceration, traumatic cataract	No	7	197
15	25	IED	Unknown	Rupture, traumatic cataract	Yes	35	539
16	23	IED	Unknown	Globe laceration, IOFB, traumatic cataract	Yes	66	128
17	23	Mortar	No	Globe laceration, IOFB	Yes	181	244
18	31	Mortar	Unknown	Globe laceration, IOFB	Yes	180	270
19	50	Mine	No	Dislocated lens	Yes	42	536

*Improvised Explosive Device

†Intraocular Foreign Body

‡Rocket Propelled Grenade

Table 2: Clinical outcome measures presented as median and range n = 19 eyes; shaded region n = 10.

	Initial	Final	*p*-value
Best Corrected Visual Acuity (LogMAR)	-0.04 (-0.12 to 0.18)	-0.12 (-0.18 to 0.012)	*P* = 0.015
Endothelial cell density (cells/mm^2)	2741 (2078 to 3334)	2674 (1961 to 3514)	*P* = 0.39
Polymegathism (%)	30.0 (25.4 to 38.9)	31.1 (23.7 to 48.1)	*P* = 0.12
Pleomorphism (%)	59.3 (35.7 to 68.9)	56.7 (35.1 to 76.9)	*P* = 0.21
Central Corneal Thickness (μm)	517 (475 to 588)	516 (462 to 642)	*P* = 0.62
Corneal Hysteresis (mmHg)	9.28 (5.90 to 12.73)	9.71 (8.40 to 13.10)	*P* = 0.45
Corneal Resistance Factor (mmHg)	9.60 (6.70 to 12.43)	9.82 (6.18 to 13.20)	*P* = 0.88
Cornea Compensated Intraocular Pressure (mmHg)	16.33 (11.75 to 22.00)	15.24 (6.75 to 19.63)	*P* = 0.14
Anterior Chamber Depth (mm)	3.27 (2.83 to 3.48)	3.27 (2.71 to 3.50)	*P* = 0.51

Table 2 summarizes the clinical characteristics at the initial and final follow up. CDVA improved over the course of follow up: logMAR -0.04 (-0.12 to 0.18) to -0.12 (-0.18 to 0.12), *p = 0.015*. The average number of endothelial cells analyzed in this study was 57 (37-94) at the initial visit and 61 (37-93) at the final visit.

In the subset comparing patients wearing eye protection (+ eye pro; n = 5) to those not wearing eye protection (- eye pro; n = 8), as self-reported, there was a significant difference in the initial CDVA logMAR + eye pro 0.00 (0.00 to 0.11); -eye pro logMAR -0.10 (-0.12 to -0.03) p = 0.003 and final CDVA logMAR + eye pro -0.10 (-0.11 to 0.01); -eye pro logMAR -0.12 (-0.12 to -0.08) p = 0.030. There were no other significant differences at the initial or final visits p > 0.17 when comparing CDVA, ECD polymegathism, pleomorphism, CH, CRF, IOPcc, and CCT between those wearing and not wearing eye protection.

Comparing patients with associated head or face injuries (n = 14) to those without (n = 5), there were no significant differences in initial or final ECD, polymegathism, pleaomorphism, CH, CRF, IOPcc, and CCT p > 0.12. There was a significant difference in initial CDVA p = 0.003 but not final p = 0.11. Comparing the subset of patients seen within ten days of injury (n = 4) to those seen later (n = 15), there were no significant differences in any measures at the initial visit or final visit in CDVA, ECD, polymegathism, pleomorphism, CH, CRF, IOPcc, and CCT *P > 0.10*.

Discussion

The force and injury associated with a blast is substantial. Facial structures, specifically nasal and brow ridges, reflect and focus blast waves amplifying pressure on the eye [14]. Furthermore, orbital geometry and diverse ocular tissue interfaces generate and channel opposing stress waves that contribute to eye damage [13]. We examined corneal morphology to describe the consequences of primary blast in the eyes of patients exposed to blast injury with trauma to the fellow eye.

Variability in morphometry of the cornea may indicate injury and the drastic pressure change from primary blast injury (PBI) has been shown to be capable of disrupting the delicate arrangement of corneal cell layers [7,15-18]. The variation in cell size of normal people is less than 32% while the proportion of normal hexagonal shaped cells is greater than 60% [19]. This study found polymegathism within the normal range at both the initial and final follow up. Pleomorphism, however, was just under the 60% normal range at 59.3% at the initial visit and 56.7% at the final visit. A limitation of the study is the lack of pre-blast measurements which would help differentiate whether the values for polymegathism and pleomorphism found in the study are due to primary blast injury or can otherwise be attributed to sampling error. A study by Walsh et al. emphasized the importance of vision testing before and after deployment to assess the true impact on vision [20].

A few studies have shown the progressive decrease in endothelial cell density (ECD) with age, long-lasting disease, and trauma [15,18,21-25]. Studies by Cockerham et al. [18] found mean ECD was reduced in veterans exposed to blast injury versus age-and refraction-matched controls and some blast injury patients had a reduction in ECD on the side of the blast compared to the fellow eye. Petras et al. [26] also noted injuries on the same side as the blast wave, highlighting the effects of body position with respect to the blast wave [26]. This study found no significant change in ECD from the initial to the final visit up to an average of 425 days after blast injury. However, one of the 19 participants presented with an initial exam ECD lower than the normal range. Longer follow up is recommended to determine if lower ECD is significant in the future.

Biomechanical examination has shown that corneas with low corneal hysteresis (CH) are less capable of absorbing or damping the energy of the air pulse [27]. Furthermore, studies have shown that eyes exhibiting significantly lower than average CH may be at risk of developing corneal disorders [28,29]. Shah et al. [29] suggested that CH may be a useful tool in assessing the progression of corneal disease [29]. This study showed median CH and corneal resistance factor (CRF) were lower than those reported in normal patients [29,30]. Some variability may be attributed to the difficulty in obtaining optimal scans in participants due to trauma in the fellow eye. However, without significant visual complaint, longitudinal follow up would be needed to determine if this decrease in biomechanics becomes significant.

Eye armor has been shown by Bailoor et al. to be effective in reducing blast wave penetration and decreasing the resulting pressure load on the eye [14]. The small subset comparison in this study showed no significant differences in corneal metrics. However, the self-reported use of protective eyewear remains unreliable as a third of participants in this series could not confirm the use of eye armor when injured.

Distance, explosive construct, containment and content, and angle of blast propagation are also factors affecting the degree of blast injury. A prospective, comparative study by Capó-Aponte et al. [31] noted repeated low-level blast exposures may damage ocular structures [31]. Information regarding blast size and explosive content, distance from epicenter, environmental conditions at the blast site (open/closed environment) was unavailable in most cases of this study therefore limiting analysis. In addition to external conditions, a modeling study by Rossi et al. observed pressure and strain rates from a blast and found two pressure patterns: structures outside the orbit attained peak pressure then quickly decayed whilst pressure posterior to the equator was significantly higher and persisted longer resulting in vascular and neural damage [13].

Conclusion

Corrected distance visual acuity did improve statistically during the study which may be attributed to improved systemic health (improved mental status, resolution of associated facial injuries, decreasing narcotic pain management) over the study period. However, in this small cohort of patients, corneal measurements of CH and CRF were observed to be below the averages reported in the literature. These findings emphasize the importance of ongoing follow-up to determine the significance and long-term prognosis of eyes exposed to primary blast as corneal PBI may not be easily detected.

References

1. Thatch AB (2003) Eye injuries associated with terrorist bombings. Ophthalmic care of the Combat Casualty. Borden Institute Army Mil. Publisher Department of the Army. Washington, DC, USA, 421-429.

2. Shuker ST (2008) Mechanism and emergency management of blast eye/orbital injuries. Expert Rev Ophthalmol 3: 229-246.

3. Gawande A (2004) Casualties of war- Military care for the wounded from Iraq and Afghanistan. N Engl J Med 351: 2471-2475.

4. Weichel ED, Colyer MH (2008) Combat ocular trauma and systemic injury. Curr Opin Ophthalmol 19: 519-525.

5. Garner J, Brett SJ (2007) Mechanism of Injury by Explosive Devices. Anesthesiology Clin 25: 147-160.

6. Scott R (2011) The injured Eye. Philos Trans. R Soc Lond B Biol Sci 366: 251-260.

7. Ritenour AD, Baskin TW (2008) Primary blast injury: Update on diagnosis and treatment. Crit Care Med 36: 311-317.

8. Horrocks CL (2001) Blast Injuries: Biophysics, Pathophysiology and Management Principles. J R Army Med Corps 147: 28-40.

9. Beiran I, Miller B (1992). Pure ocular blast injury. Am J Ophthalmol 114: 504-505.

10. Taber KH, Warden DL, Hurley RA (2006) Blast-Related Traumatic Brain Injury: What is Known? J Neurophychiatry Clin Neurosci 18: 141-145.

11. Champion HR, Holcomb JB, Young LA (2009) Injuries from Explosions: Physics, Biophysics, Pathology, and Required Research Focus. J Trauma 66: 1468-1477.

12. Abbotts R, Harrison SE, Cooper GL (2007) Primary Blast Injuries to the Eye: A review of the Evidence. JR Army Med Corps 153: 119-123.

13. Rossi T, Boccassini B, Esposito L, Clemente C, Iossa M, et al. (2012) Primary Blast Injury to the Eye and Orbit: Finite Element Modeling. Invest Ophthalmol 53: 8057-8066.

14. Bailoor S, Bhardwaj R, Nguyen TD (2015) Effectiveness of eye armor during blast loading. Biomech Model Mechanobiol 14: 1227-1237.

15. Lemp MA (1972) Air blast keratopathy. Arch Ophthalmol 88: 575-576.

16. Slingsby JG, Forstot SL (1981) Effect of blunt trauma on the corneal endothelium. Arch Ophthalmol 99: 1041-1043.

17. Cockerham GC, Goodrich GL, Weichel ED, Orcutt JC, Rizzo JF, et al. (2009) Eye and visual function in traumatic brain injury. J Rehabil Res Dev 46: 811-818.

18. Cockerham GC, Lembke S, Rice TA, Wang G, Glynn-Milley C, et al. (2014) Closed-Globe Injuries of the Ocular Surface Associated with Combat Blast Exposure. Ophthalmol 121: 2165-2172.

19. Stein HA, Skatt BJ, Stein RM, Freeman MI (2002) Endothelial response to contact lenses. In: Fitting guide for rigid and soft contact lenses: A practical approach. St Louis, MO: Mosby Inc 501-503.

20. Walsh DV, Capó-Aponte JE, Jorgenson-Wagers K, Temme LA, Goodrich G, et al. (2015) Visual Field Dysfunctions in Warfighters During Different Stages Following Blast and Nonblast mTBI. Mil Med 180: 178-185.

21. Edelhauser HF (2006). The Balance between Corneal Transparency and Edema. Invest Ophthalmol Vis Sci 47: 1755-1767.

22. Bourne WM, McLaren JW (2004) Clinical Responses of the Corneal Endothelium. Exp Eye Res 78: 561-572.

23. Sanchis-Gimeno JA, Lleo-Perez A, Alonso L, Rahhal MS, Martinez Soriano F (2005) Corneal endothelial cell density decreases with age in emmetropic eyes. Histol Histopathol 20: 423-427.

24. Krohn J, Hovding G (2005) The influence of donor age and cause of death on corneal endothelial cell density. Acta Ophthalmol Scand 83: 746-750.

25. Bourne WM (2003) Biology of the corneal endothelium in health and disease. Eye 17: 912-918.

26. Petras JM, Bauman RA, Elsayed NM (1997) Visual System Degeneration induced by blast overpressure. Toxicol 121: 41-49.

27. Dupps WJ (2007) Hysteresis: New mechanospeak for the ophthalmologist. J Cataract Refract Surg 33: 1499-1501.

28. Luce DA (2005) Determining in vivo biomechanical properties of the cornea with an ocular response analyzer. J Cataract Refract Surg 31: 156-162.

29. Shah S, Laiquzzaman M, Bhojwani R, Mantry S, Cunliffe I (2007) Assessment of the Biomechanical Properties of the Cornea with the Ocular Response Analyzer in Normal and Keratoconic Eyes. Invest Ophthalmol Vis Sci 48: 3026-3031.

30. Ortiz D, Piñero D, Shabayek M, Arnalich-Montiel F, Alió J (2007) Corneal biomechanical properties in normal, post-laser in situ keratomileusis, and keratoconic eyes. J Cataract Refract Surg 33: 1371-1375.

31. Capó-Aponte JE, Jurek GM, Walsh DV, Temme LA, Ahroon WA, et al. (2015) Effects of repetitive low-level blast exposure on visual systems and ocular structures. J Rehabil Res Dev 52: 273-290.