January 2013

Craniocerebral injuries in war against terrorism --- a contemporary series from Pakistan

Muhammad-Umair Bashir

Muhammad-Zubair Tahir
Aga Khan University, muhammad.zubair@aku.edu

Ehsan Bari
Aga Khan University, ehsan.bari@aku.edu

Sehreen Mumtaz

Craniocerebral injuries in war against terrorism — a contemporary series from Pakistan

Muhammad Umair Bashir, Muhammad Zubair Tahir, Ehsan Bari, Sehreen Mumtaz*

【Abstract】**Objective:** Terrorism-related bomb attacks on civilian population have increased dramatically over the last decade. Craniocerebral injuries secondary to improvised explosive devices have not been widely reported in the context of unarmored civilians. This series intends to report the spectrum of these injuries secondary to suicidal and implanted bombs as encountered at the Aga Khan University Hospital, Pakistan (AKUH). Further, a few pertinent management guidelines have also been discussed.

Methods: The hospital database and clinical coding during a 5-year period were examined for head injuries secondary to terrorism-associated blasts. In addition to patient demographics, data analysis for our series included initial Glasgow Coma Scale, presenting neurological complaints, associated non-neurological injuries, management (conservative or operative) to associated complications, and discharge neurological status.

Results: A total of 16 patients were included in this series. Among them 9 were victims of suicidal blasts while 7 were exposed to implanted devices. The patients presented with diverse patterns of injury secondary to a variety of shrapnel. A follow-up record was available for 12 of the 16 patients (mean follow-up: 7.8 months), with most patients having no active complaints.

Conclusion: The results of this series show that civilian victims of suicidal and improvised bombings present with a wide range of neurological symptoms and injury patterns, which often differ from the neurological injuries incurred by military personnel in similar situations, and thereby often require individualized care.

Key words: ***Brain injuries; Craniocerebral trauma; Decompressive craniectomy***

Chin J Traumatol 2013;16(3):149-157

Terrorism-related bomb attacks on civilian population have increased dramatically over the last decade. Although bomb-related head injuries have been historically associated with combat troops and battlefield trauma, militant and terrorist outfits now ruthlessly target urban civilian populations with a variety of improvised explosive devices (IEDs). The wide varieties in IEDs, coupled with the advent of suicide tactics, have given rise to a spectrum of craniocerebral injuries. The brain injuries sustained by the victims of these well-planned attacks are often associated with the lethal effects of both penetrating trauma and blast injury.

Between 2007 and 2009, more than 5 500 people have been killed in suicidal and other attacks on civilians in Pakistan. In 2011 alone, there have been 334 terrorism-related bomb attacks in the country, with a distressing outcome of 659 deaths and 1 585 injuries.[1] Whilst a number of these attacks involved suicidal bombers with explosive-laden vests or belts, many of the casualties were also secondary to rudimentary road-side bombs. Most of these improvised explosive vests or implanted devices were found to be packed with ball bearings, nails, bolts, screws and other objects that served as shrapnel to maximize the injuries.[2] This resulted in unique mechanisms of injury to the skull and brain that have not been previously reported in literature in the context of unarmored civilians.

We report here a series of penetrating and blast-related craniocerebral injuries secondary to suicidal and implanted bombs, followed by a discussion of the patterns of injury, management, complications and follow-up of this unique subset of patients.

METHODS

Karachi is a major metropolitan city with a population of 13 million. In spite of numerous terrorism-related

DOI: 10.3760/cma.j.issn.1008-1275.2013.03.005

Department of Neurosurgery, Aga Khan University Hospital, Karachi, Pakistan (Bashir MU, Tahir MZ, Bari E, Mumtaz S)

*Corresponding author: Tel: 92-2134864764, Email: ehsanbari12@gmail.com

attacks over the years, an organized mass casualty service system does not exist in Pakistan. The distribution of casualties therefore becomes a difficult task in the absence of effective triage and communication between the scene of the incident and the hospitals.[3] In most instances, the distribution of the victims of an attack is determined by the distance to one of the hospitals designated by the government as a trauma center.[4]

Aga Khan University Hospital (AKUH) is a private tertiary hospital, with a commitment to provide trauma care. About 50% of trauma patients to AKUH are transferred from government hospitals and other rural health care centers. Most of these patients have had some kind of initial management and stabilization. The hospital database and clinical coding for a 5-year period were examined for head injuries secondary to terrorism-associated blasts. In this study, we included patients who were both transferred from other centers and directly through the hospital emergency services. A multidisciplinary trauma team was involved in the management of these patients, but we only selected those who had significant neurotrauma to warrant a neurosurgical review. A detailed review of charts and medical records of these patients was performed using a standardized proforma. Those patients who suffered non-terrorism-related head trauma or had incomplete/missing records were excluded from the series.

In addition to patient demographics, data analysis for our series included the mechanism of injury (suicidal vs implanted device), initial Glasgow Coma Scale (GCS), presenting neurological complaints, associated non-neurological injuries, management to associated complications, and discharge neurological status. The baseline neuroimaging was also reviewed to assess skull fractures, the site of the penetration, the presence and location of shrapnel, inter- and extra-axial hemorrhage and associated findings. Follow-up data, if available, were also analyzed, with emphasis on neurologic sequelae.

RESULTS

A total of 16 patients (14 males and 2 females) were included in this series with a mean age of 29 years, ranging from 4-58 years. Among them 9 patients were victims of suicidal blasts while 7 were exposed to implanted devices. The majority of the patients were transferred from other hospitals or trauma centers (*n*=8). Most of the transferred patients were relatively stable upon arrival since they had received some form of initial stabilization at other centers.

The average GCS at presentation was 12 with the lowest value of 6. Five patients underwent emergent intubation upon presentation. Among them 4 were assessed to have unstable vitals with a GCS of 8 or less while one patient had extensive maxillofacial trauma with a compromised airway. The clinical presentation was variable, with drowsiness and headache being the most common complaints. Other neurologic deficits included hemiparesis, visual blurring, vertigo and seizures.

All patients underwent head CT scans for damage assessment. Skull fractures were identified in 13 of the 16 patients, with suicidal blast victims being the clear majority. Among the 13 patients, 3 were basilar fractures, while the rest were the depressed type involving different parts of the skull. Moreover, 6 fractures of the skull were found to be comminuted, with bone fragments driven into the dura/brain due to the force of the impact. A large number of patients (*n*=13) had penetrating injuries. CT scans and intraoperative assessment of these patients revealed bone fragments and/or various kinds of shrapnel lodged in different parts of the brain. The brain parenchymal injury patterns ranged from significant intracranial hemorrhages to small contusions, with the latter being the most consistent finding on CT imaging.

A total of 11 patients underwent neurosurgical interventions while 5 patients were managed conservatively after routine care of their scalp lacerations. Of the 11 patients who needed surgery, 2 required only superficial wound debridement, 7 had a craniotomy for evacuation of hematomas or any accessible foreign bodies and the rest underwent a decompressive craniectomy (DC). The initial step in all penetrating injuries was wound exploration followed by debridement. All the patients who needed a craniotomy were further managed by a primary duraplasty with pericranial fascia. Two patients (S1 and M7) required a DC for massive brain swelling and raised intracranial pressure (ICP). The decision to perform a DC on S1 was taken intraoperatively after observing significant cerebral edema during the craniotomy. However, M7 had indirect evidence of raised ICP on his initial CT scans and hence an early preoperative decision was made to proceed with a DC (Table 1).

Table 1. General data of patients

Patient ID	Age (yrs)/ Gender	GCS at presentation	Location of fracture	Pattern of injury	Type of foreign body	Foreign body location	ICU stay (d)	Immediate complication	GOS at discharge	Follow-up duration (mon)	Late complications
S1	37/M	13	Basilar	Contusions/edema	-	-	15	-	4	6	CSF leak/seizures
S2	56/M	15	Parietal	Hematoma	Bone pieces	Parietal	0	-	5	3	-
S3	5/M	15	Frontal	Contusions	Shrapnel	Frontal	0	Wound infection	5	8	-
S4	29/M	15	Basilar/sinuses	Contusions	Pellets	Midline cortical	0	-	5	Not available	-
S5	12/F	8	Frontal/sinuses	IVH/contusions/ edema/ICH	Shrapnel	Ethmoidal cells	2	-	3	12	-
S6	35/M	6	Frontoparietal	Contusions/edema	Bone pieces/ shrapnel	Frontoparietal	2	-	3	12	-
S7	30/M	13	Frontal	Contusions/edema	Bone pieces/ shrapnel	Frontal	0	-	5	12	-
S8	4/M	15	Temporoparietal	Contusions/SDH	Shrapnel	Parietal	0	-	5	6	Headache/blackouts
S9	30/M	15	Frontal	Contusions	Nail	Parietooccipital	0	-	3	12	-
M1	35/M	15	-	Contusion/edema	Bone pieces	Frontal	0	CSF leak	5	12	-
M2	40/M	11	-	Infarct	-	-	0	-	4	LAMA	-
M3	58/M	12	Basilar/temporal	Contusion	Shrapnel	Orbit	0	Urinary incontinence	4	Not available	-
M4	40/M	8	-	Contusions/edema/ SAH/SDH	-	-	3	-	5	2	-
M5	14/M	12	Temporal	Contusions	Bone pieces/ shrapnel	Deep cortical	2	-	5	8	-
M6	28/M	11	Frontal	Contusions	-	-	0	-	5	Not available	-
M7	45/M	8	Temporal	Edema/contusions	Bone pieces	Temporal	5	Septic shock/wound infection	3	1	Hydrocephalus, (GOS 2)

-: None; GOS: Glasgow Outcome Scale; CSF: cerebrospinal fluid; IVH: intraventricular hemorrhage; ICH: intracranial hemorrhage; SDH: subdural hematoma; SAH: subarachnoid hemorrhage; LAMA: leave against medical advice.

The average length of stay in the intensive care unit was 2 days, followed by 3 days in the special care unit. All patients managed operatively received pre- and post-operative antibiotics. Four patients who were managed conservatively also received prophylactic antibiotics. Mannitol was used to lower ICP in 6 patients, including one from the conservative group. Prophylactic antiepileptic drugs (AEDs) were used in all patients for a minimum of 1 week. Those patients who either presented with seizures or had seizure episodes during their hospital stay were continued on AEDs after discharge for at least 1 week.

Nine of the 16 patients had a discharge GOS of 5, 3 patients had a GOS of 4 and 4 patients had a GOS of 3. There were no mortalities. Generally, victims of suicidal blasts had a lower GOS at discharge compared to the other group. Two patients (S3 and M7) developed superficial wound infections during their hospital stay. A review of their record revealed significantly contaminated wounds on initial presentation. Also two patients (S1 and M1) developed CSF leaks, and one of them (M1) was managed conservatively while another (S1) required an endonasal endoscopic repair for his post-traumatic CSF leak. A follow-up record was available for 12 of the 16 patients (mean follow-up: 7.8 months), with most patients having no active complaints. However, one patient (M7) developed a noncommunicating hydrocephalus after a DC. He was subsequently managed by a ventriculoperitoneal (VP) shunt and cranioplasty. A young patient (4 years old) was reported to have developed headaches, episodes of fainting and behavioral changes. Follow-up imaging for this patient was found to be normal. He was subsequently referred to a neurologist and a child psychiatrist.

DISCUSSION

Terrorist attacks directed against civilian populations have emerged as a significant threat worldwide, especially in Pakistan where the health infrastructure and emergency services are already overwhelmed. The situation becomes worse when terrorism involves suicidal tactics, allowing the fanatic unchecked infiltration, close approximation and maximum injury to unprepared civilians.[5] The absence of body armor and helmets in the civilian population worsens the morbidity and mortality of this type of trauma.

Blast-associated head trauma and its neurologic manifestations in civilians remain a complicated and a relatively obscure aspect of neurotrauma.[6,7] The mechanisms of injury include primary overpressure events, initial penetrating shrapnel injuries followed by secondary fragmentation wounds, tertiary blunt injuries and deceleration forces, all contributing to a diverse spectrum of injuries.[6,8,9] In addition to the explosive content of the device, the presence of augmenting objects such as pellets, nuts, bolts, nails, ball bearings and other shrapnels further complicates the picture (Figures 1 and 2). The most interesting aspect of this type of trauma is therefore the diversity of shrapnel leading to peculiar brain parenchymal injury.

Injury patterns

Exposure to a bomb blast creates a range of injury severities ranging from mild concussive effects to fatal injuries.[10] People in close vicinity of the blasts often have overwhelming fatal injuries with a grim prognosis. The victims who are fortunate enough to be farther often survive the initial blast. However, they later present with a unique pattern of cranial injuries.[11]

Traumatic brain injury (TBI) has classically been divided into two types: penetrating and closed, with both having a distinct pattern of parenchymal injury. Blast-associated TBI, however, incorporates aspects from both types of TBI.[8] As the blast waves are transmitted to the skull and its contents, brain motion and deceleration can lead to hemorrhages, cerebral contusions and diffuse axonal injury with all associated with the closed TBI.[12] Following the initial pressure changes and acceleration-deceleration of the head, victims of terrorist blasts are further exposed to a variety of shrapnel, fractured skull bone fragments, and secondary missiles in form of body parts. The foreign bodies lead to destruction and disruption of neurons, glial cells and fiber tracts, producing hemorrhage, infarcts, cerebral edema and necrotic tissue along the path of travel.[8,9] All of these patterns of injury were observed in our series of patients, ranging from mild injuries to prominent intra- and extra-axial hemorrhage, contusions and cerebral edema. Further, in addition to the classical closed TBI injury patterns, we encountered a variety of shrapnel and bone fragments in some of our patients, producing visually impressive patterns (Figures 1 and 2).

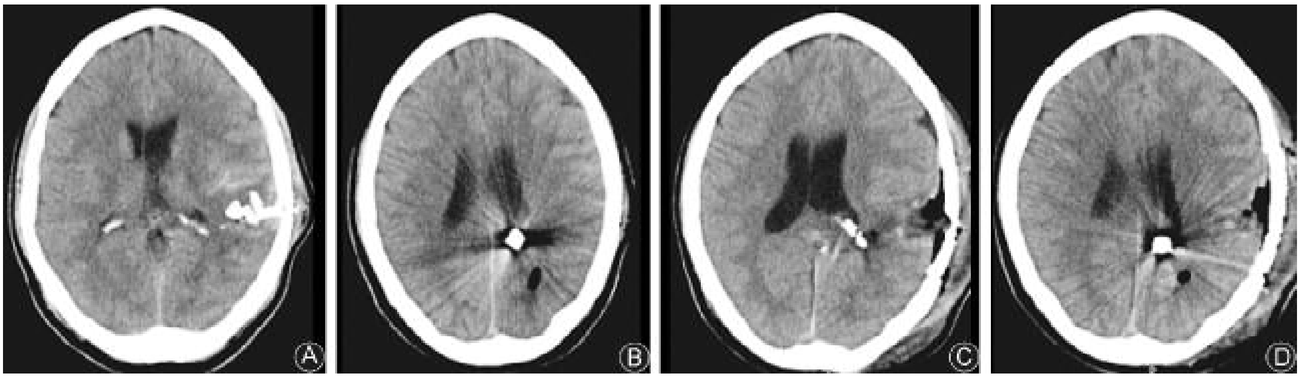

Figure 1. Axial CT scan of the brain after exposure to an implanted bomb blast, showing left parietal fracture, traumatic subarachnoid hemorrhage and secondary intraparenchymal bone fragments (**A**). Another axial view of the same patient showing nut-bolt seated close to lateral ventricle (**B**). Postoperative CT scans showing removal of superficial bone fragments, while deep seated fragments and shrapnel left behind (**C** and **D**).

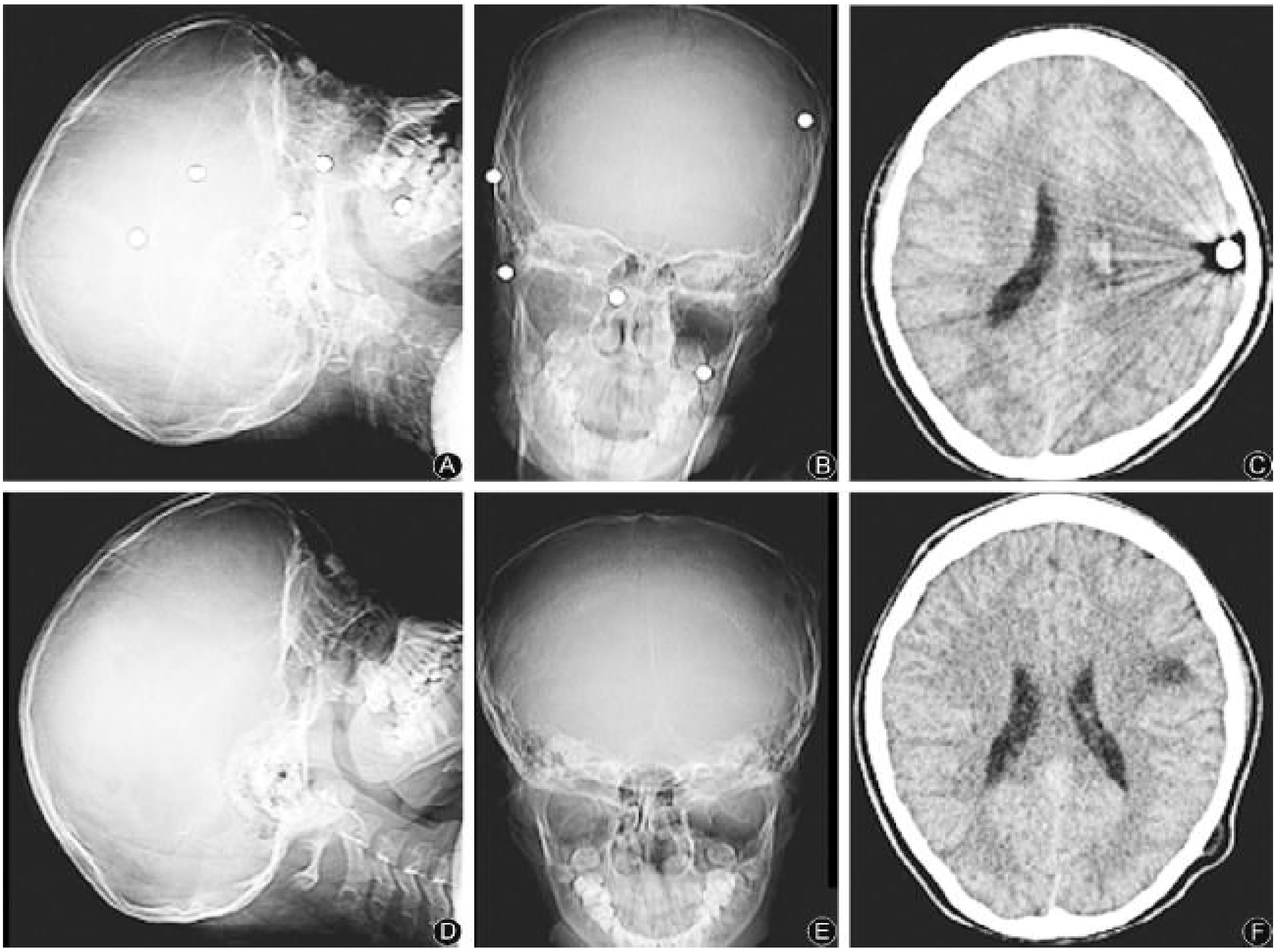

Figure 2. Preoperative CT scanograms showing ball bearings used in a suicidal bombing (**A** and **B**). Axial CT brain showing ball bearings in left parietal region with brain edema and intraventricular hemorrhage (**C**). Postoperative pictures after removal of ball bearings (**D**, **E** and **F**).

Neurological presentation and imaging

The neurological presentation after craniocerebral trauma includes mild concussive symptoms as well as specific neurologic deficits based on the extent of injury.[6] A majority of the patients in our series had received some forms of initial stabilization and resuscitation at other centers and hence their GCS at presentation was not an accurate reflection of the severity of the initial injury. Despite the presence of penetrating brain injuries in many of the patients, most of them presented with non-specific headaches, drowsiness and vertigo. However, a few patients presented with specific neurological complaints of visual blurring and seizures. Such variable presentation is consistent with the concussive signs and symptoms secondary to bomb blasts described in the literature.[6,8,13] However, blast-associated TBI can be deceptively benign early and specific neuroimaging is necessary to discover the precise extent of injuries and institute appropriate operative intervention.[14]

Imaging for head trauma is often based on the resource availability in many primary health care centers in Pakistan. Hence, some of the patients were just assessed with plain skull X-rays prior to coming to AKUH. However, axial CT scan is generally agreed to be the most useful primary imaging investigation. In addition to the assessment of entry site, fragmentation, and missile tracts, CT imaging provides an indication of intracranial pressure.[15] All of our patients underwent immediate CT imaging to assess damage. Thirteen patients were identified with skull fractures. The CT scans were also able to identify the presence of penetrating bodies and associated parenchymal injury in all of the patients. Moreover, the effacement of the ventricles and the patency of basal cisterns gave us an indirect estimate of the ICP, allowing us to decide on an early intervention plan.[16]

In addition to routine CT imaging, four vessel angiography is important in the detection of vascular injuries and pseudoaneurysms in penetrating brain trauma.[8,17,18] However, none of our patients were assessed with an angiogram for vascular trauma due to cost restraints. However, there was no delayed presentation with SAH or ICH in any of our patients.

Clinical management

The guidelines for management of blast-associated TBI originate from protocols determined initially by military trauma experts and have been tailored for use mostly in combat scenarios.[9,19,20] These guidelines emphasize prehospital care, initial stabilization and resuscitation followed by GCS assessment and then appropriate triage for further care.[9,20] This is followed by hospital protocols to ensure early injury assessment, adequate cerebral perfusion and oxygenation with an attempt to prevent posttrauma seizures and infection. However, Pakistan lacks such a coordinated infrastructure for prehospital management and even protocol-based hospital management in many government centers.[3] Most of the patients in this series did not have any definitive neurotrauma care prior to coming to AKUH.

Important issues to attend to after initial assessment and stabilization include maintaining adequate oxygenation, controlling ICP and ensuring proper cerebral perfusion.[9] Intracranial hypertension is common after blast-induced TBI.[8] Ideally, this dictates the use of ICP monitors to adequately monitor and control ICP in developed countries. However, such monitors are an expensive entity in our setting, with most of our ICP assessment relying on CT imaging and clinical examinations. In this context, our management of raised ICP in TBI patients involves standard medical therapy with osmotic agents, followed by intubation and brief period of hyperventilation, if indicated, with a low threshold of progressing to a DC if the patient worsens medical management. In this series of patients, an early DC (within 6 hours of admission) was performed in two patients. In one of these patients (M7), the indication for a DC was based upon indirect findings of increased ICP (midline shift, obliteration of ventricles and cisterns). In another patient (S1), the decision for a DC was made intraoperatively after observing significant brain swelling (Figure 3). A number of recent studies confirm that a DC performed early in patients with severe traumatic brain injury yields a better outcome compared to delayed surgical intervention or to medical therapy.[21-26]

Debridement and antibiotics

Infection control in blast-induced TBI encompasses two aspects of management: wound debridement in case of penetrating trauma and antibiotic prophylaxis. All the patients with significant penetrating injuries in our series underwent wound exploration, debridement and removal of any accessible foreign bodies. The decision with regards to removal of shrapnel, debris, or bone fragments and the extent of resection of the injury tract has evolved over the decades, with most recent recommendations suggesting that aggressive removal of all fragments is unnecessary, but removal of gross contamination with debridement of large injury tracts is beneficial.[27,28] In conformance with these suggestions, we removed any accessible fragments, but those that were deep seated and otherwise inaccessible were left in place.

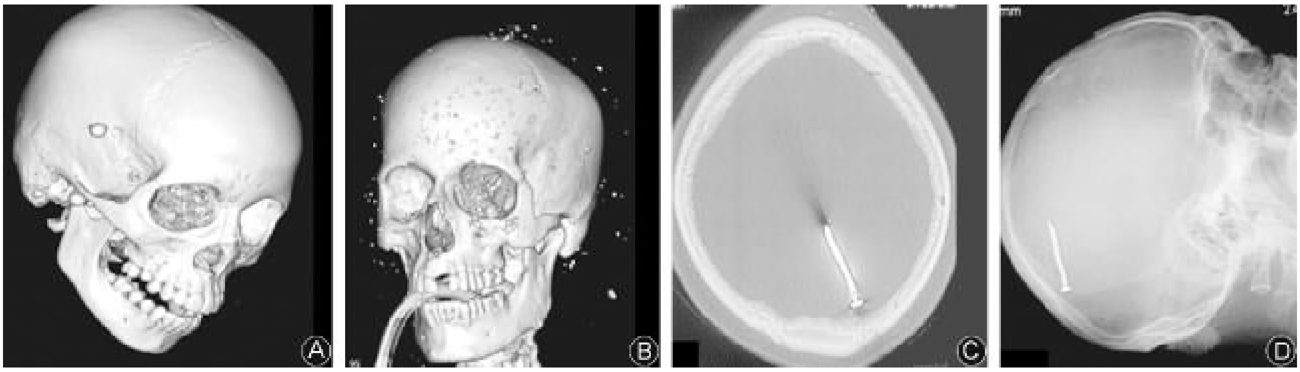

Figure 3. A 3D reconstructed CT of skull showing ball bearing in right temporal region (**A**). Another 3D reconstructed CT skull showing blast of pallets involving the head and face (**B**). Skull X-rays in anteroposterior (**C**) and lateral (**D**) views showing an intracranial nail used in an explosive device by a suicide bomber.

All the patients in our series who had a head injury secondary to bomb blast requiring surgical debridement received preoperative antibiotics (cephazolin 2 g), which were continued postoperatively for 1 week. However, those patients with air sinus wounds and/or CSF leaks received an additional regimen for anaerobic (metronidazole) and gram-negative coverage (gentamycin). The use of prophylactic antibiotics in blast-associated penetrating brain injury is consistent with the recommendations of several military-based studies that report a significantly lower incidence of meningitis and brain abscesses after routine prophylactic antibiotics.[29-32] Although the results of our series are only subject to minimal interpretation due to the study design and limited follow-up, no brain abscesses or other central nervous system infections have been reported so far in 12 of the 16 patients who had a follow-up, in spite of permanently lodged cortical foreign bodies in some of them.

Seizure prophylaxis

Severe TBI has been associated with seizures that often complicate acute trauma management and subsequent rehabilitation. Although still controversial, most centers incorporate early prophylactic treatment with phenytoin, beginning with an IV loading dose, to decrease the risk of posttraumatic seizures occurring within the first seven days.[33] Prophylactic AEDs were used in all patients for 1 week. Patients who presented with seizures or had seizure episodes during the hospital admission were continued on AEDs after discharge. However, one patient (S1) was reported to have developed delayed seizures two months after the incident in spite of seizure prophylaxis therapy. A review of this patient's initial imaging revealed extensive temporoparietal contusions with associated depressed skull fractures. Perhaps, for such patients with severe TBI, anti-epileptic drugs should be used for a longer duration. However, further studies are needed to look at the appropriate dosage and duration for seizure prevention.

Morbidity

In addition to the more discernible complications such as CSF leaks, posttraumatic seizures and wound infections, blast-related TBI has significant neuropsychological and psychiatric morbidity.[8,33-36] This coupled with the possibility of permanent neurological disability, has a devastating socioeconomic backlash with a lasting impact on the patients, their families and the society. Military victims of blast-associated TBI are considered a casualty of war and governments continue to provide them economic support and rehabilitation. However, most civilian victims of terrorist bomb blasts are left to deal with their injuries on their own.

Most of the patients in our series recovered adequately after acute management of their trauma. However, only 12 of the 16 patients had a follow-up visit with a mean of 7.8 months. One patient developed late onset posttraumatic seizures and associated CSF rhinorrhea, while another patient was reported to have developed headaches, episodes of fainting and behavioral changes. One patient who required a DC developed communicating hydrocephalus. He was managed with a VP shunt and cranioplasty (Figure 4). None of our patients underwent neuropsychological testing at the time of acute management or thereafter to determine psychiatric morbidity. Therefore, a spectrum of disorders including acute stress disorders, PTSD, depression and other post-concussive syndromes reported in literature could not be assessed.

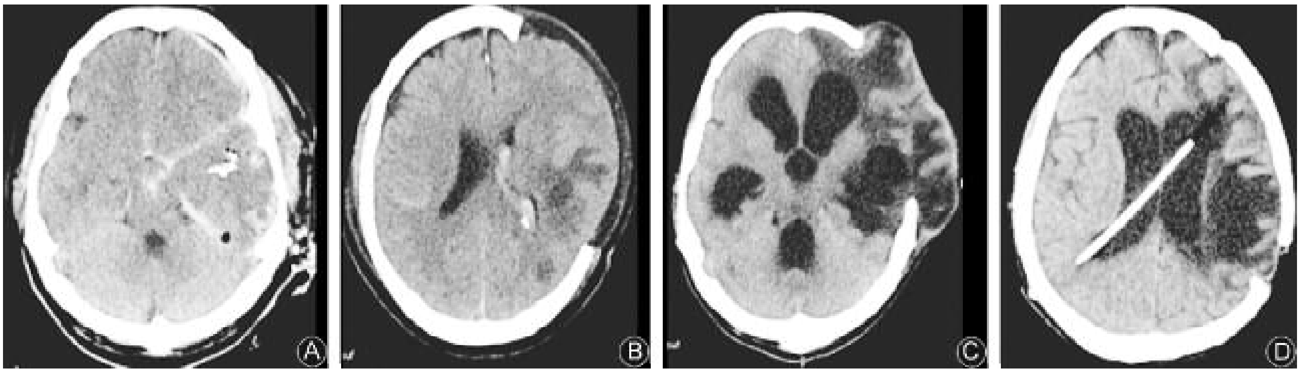

Figure 4. Axial CT of brain showing a left temporal contusion, a traumatic SAH, effacement of basal cisterns and bone pieces in a victim of an implanted explosive device (**A**). Postoperative CT scanogram of the same patient after wound debridement and a decompressive craniectomy (**B**). Brain CT 4 weeks after surgery showing a communicating hydrocephalus (**C**). Brain CT after insertion of VP shunt and cranioplasty (**D**).

This series is by no means a true reflection of the morbidity and mortality due to terrorist bomb attacks in Pakistan. Most of our patients were referred for specialized neurological care after they were assessed to be salvageable at primary trauma centers. However, there are many others brought in government-designated trauma centers that are only considered for palliative care due to limited resources and lack of expertise (personal communication). Future neurotrauma protocols and interventions need to target such patients in order to make a significant contribution to humanity.

In conclusion, blast-induced TBI is historically a casualty of war, which now sadly becomes a civilian disease with diverse problems. Pakistan, like other resource-deprived countries, lacks the resources and infrastructure to effectively deal with these patients. Victims of suicidal and improvised bombings present with a wide range of neurological symptoms and often require individualized care. Future prospective studies are warranted in this group of patients to improve our understanding and thereby influence clinical outcome.

REFERENCES

1. Fatalities in terrorist violence in Pakistan 2003-2012. [2011-6-30]. http://www.satp.org/satporgtp/countries/pakistan/database/casualties.htm#.

2. Kluger Y. Bomb explosions in acts of terrorism-detonation, wound ballistics, triage and medical concerns. Isr Med Assoc J 2003;5(4):235-40.

3. Raftery KA. Emergency medicine in southern Pakistan. Ann Emerg Med 1996;27(1):79-83.

4. Mehdi IJ. Emergency medicine in Pakistan. Ann Emerg Med 1996;27(1):84-8.

5. Lucci EB. Civilian preparedness and counter-terrorism: conventional weapons. Surg Clin North Am 2006;86(3):579-600.

6. Hicks RR, Fertig SJ, Desrocher RE, et al. Neurological effects of blast injury. J Trauma 2010;68(5):1257-63.

7. Taber KH, Warden DL, Hurley RA. Blast-related traumatic brain injury: what is known? J Neuropsychiatry Clin Neurosci 2006;18(2):141-5.

8. Ling G, Bandak F, Armonda R, et al. Explosive blast neurotrauma. J Neurotrauma 2009;26(6):815-25.

9. Ling GS, Ecklund JM. Traumatic brain injury in modern war. Curr Opin Anaesthesiol 2011;24(2):124-30.

10. Ropper A. Brain injuries from blasts. N Engl J Med 2011; 364(22):2156-7.

11. Finkel MF. The neurological consequences of explosives. J Neurol Sci 2006;249(1):63-7.

12. Traumatic brain injury. In: Bandak FA, Eppinger RH, Ommaya AK, ed. Traumatic brain injury: bioscience and mechanics. New York: Mary Ann Liebert Inc., 1996:265-76.

13. Belanger HG, Kretzmer T, Yoash-Gantz R, et al. Cognitive sequelae of blast-related versus other mechanisms of brain trauma. J Int Neuropsychol Soc 2009;15(1):1-8.

14. Bochicchio GV, Lumpkins K, O' Connor J, et al. Blast injury in a civilian trauma setting is associated with a delay in diagnosis of traumatic brain injury. Am Surg 2008;74(3):267-70.

15. Kim JJ, Gean AD. Imaging for the diagnosis and management of traumatic brain injury. Neurotherapeutics 2011;8(1):39-53.

16. Brain Trauma Foundation, American Association of Neurological Surgeons, Congress of Neurological Surgeons. Guidelines for the management of severe traumatic brain injury. J Neurotrauma 2007;24 Suppl 1:S1-106.

17. Armonda RA, Bell RS, Vo AH, et al. Wartime traumatic cerebral vasospasm: recent review of combat casualties. Neurosurgery 2006;59(6):1215-25.

18. Al-Mefty O, Holoubi A, Fox JL. Value of angiography in

cerebral nail-gun injuries. AJNR Am J Neuroradiol 1986;7(1):164-5.

19. Okie S. Traumatic brain injury in the war zone. N Engl J Med 2005;352(20):2043-7.

20. Warden D. Military TBI during the Iraq and Afghanistan wars. J Head Trauma Rehabil 2006;21(5):398-402.

21. Gower DJ, Lee KS, McWhorter JM. Role of subtemporal decompression in severe closed head injury. Neurosurgery 1988; 23(4):417-22.

22. Whitfield P, Guazzo E. ICP reduction following decompressive craniectomy. Stroke 1995;26(6):1125-6.

23. Guerra WK, Gaab MR, Dietz H, et al. Surgical decompression for traumatic brain swelling: indications and results. J Neurosurg 1999;90(2):187-96.

24. Clifton GL, Miller ER, Choi SC, et al. Lack of effect of induction of hypothermia after acute brain injury. N Engl J Med 2001;344(8):556-63.

25. Narayan RK. Hypothermia for traumatic brain injury-a good idea proved ineffective. N Engl J Med 2001;344(8):602-3.

26. Polin RS, Shaffrey ME, Bogaev CA, et al. Decompressive bifrontal craniectomy in the treatment of severe refractory posttraumatic cerebral edema. Neurosurgery 1997;41(1):84-92.

27. Carey ME. Analysis of wounds incurred by U.S. Army Seventh Corps personnel treated in Corps hospitals during Operation Desert Storm, February 20 to March 10, 1991. J Trauma 1996;40(3 Suppl):S165-9.

28. Carey ME, Joseph AS, Morris WJ, et al. Brain wounds and their treatment in VII Corps during Operation Desert Storm, February 20 to April 15, 1991. Mil Med 1998;163(9):581-6.

29. Antibiotic prophylaxis for penetrating brain injury. J Trauma 2001;51(2 Suppl):S34-40.

30. Aarabi B, Taghipour M, Alibaii E, et al. Central nervous system infections after military missile head wounds. Neurosurgery 1998;42(3):500-7.

31. Rish BL, Caveness WF, Dillon JD, et al. Analysis of brain abscess after penetrating craniocerebral injuries in Vietnam. Neurosurgery 1981;9(5):535-41.

32. Taha JM, Haddad FS, Brown JA. Intracranial infection after missile injuries to the brain: report of 30 cases from the Lebanese conflict. Neurosurgery 1991;29(6):864-8.

33. Chang BS, Lowenstein DH. Practice parameter: antiepileptic drug prophylaxis in severe traumatic brain injury: report of the Quality Standards Subcommittee of the American Academy of Neurology. Neurology 2003;60(1):10-6.

34. Kashluba S, Hanks RA, Casey JE, et al. Neuropsychologic and functional outcome after complicated mild traumatic brain injury. Arch Phys Med Rehabil 2008;89(5):904-11.

35. Reeves RR, Panguluri RL. Neuropsychiatric complications of traumatic brain injury. J Psychosoc Nurs Ment Health Serv 2011;49(3):42-50.

36. Kim E, Lauterbach EC, Reeve A, et al. Neuropsychiatric complications of traumatic brain injury: a critical review of the literature (a report by the ANPA Committee on Research). J Neuropsychiatry Clin Neurosci 2007;19(2):106-27.

(Received August 3, 2012)

Edited by DONG Min