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Case Report

Blast-related traumatic brain injury: Report of a severe case and review of the literature

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ABSTRACT

Background: Traumatic brain injury (TBI) is a well-known brain dysfunction commonly encountered in activities such as military combat or collision sports. The etiopathology can vary depending on the context and bomb explosions are becoming increasingly common in war zones, urban terrorist attacks, and civilian criminal feuds. Blast-related TBI may cause the full severity range of neurotrauma, from a mild concussion to severe, penetrating injury. Recent classifications of the pathophysiological mechanisms comprise five factors that reflect the gravity of the experienced trauma and suggest to the clinician different pathways of injury and consequent pathology caused by the explosion.

Case Description: In the present report, the authors describe a case of 26 years old presenting with blast-related severe TBI caused by the detonation of an explosive in an amusement arcade. Surgical decompression to control intracranial pressure and systemic antibiotic treatment to manage and prevent wound infections were the main options available in a civilian hospital.

Conclusion: While numerous studies examined the burden of blast-related brain injuries on service members, few papers have tackled this problem in a civilian setting, where hospitals are not sufficiently equipped, and physicians lack the necessary training. The present case demonstrates the urgent need for evidence-based diagnostic and therapeutic protocols in civilian hospitals that would improve the outcome of such patients.

Keywords: Blast, Civil population, Neurotrauma, Severe traumatic brain injury

INTRODUCTION

Traumatic brain injury (TBI) is characterized by an acute brain injury that occurs as a result of external forces applied to the head. It is estimated that more than 5% of military personnel deployed to Iraq and Afghanistan have suffered from TBI and have exhibited long-term impairment of both cognitive and psychological functions.[18] Blast-related brain injuries are becoming increasingly common as a primary cause of TBI in the military and the civilian setting.[3-15] Nevertheless, despite more than a decade of research, the etiology, treatment, and recovery from such injuries remain poorly understood.

In the present report, a case of 26 years old with blast-related severe TBI presenting with a left frontoparietal fracture and multiple intraparenchymal foreign bodies is described. The

management of such cases still poses various challenges, especially in nonmilitary hospitals, where physicians lack sufficient expertise and training.

CASE REPORT

A 26-year-old man was found unconscious in an amusement arcade following the detonation of an unspecified explosive. On his initial assessment, the emergency physician found a Glasgow Coma Scale (GCS) score of 3, a left frontoparietal lacerated and contused wound extending to the frontosphenoidal suture [Figure 1], hypotension, and tachycardia. Eye examination revealed fixed and dilated pupils bilaterally with dubious light reflexes and intact corneal responses. On general examination, irregular breathing patterns were noticed and an injury severity score of 59 was reported (head or neck: "critical," face: "critical," and external injury: classified as "serious" due to the presence of diffuse burns caused by the detonation). Therefore, the patient was intubated for safe transport and sequentially admitted to the emergency department. Computed tomography (CT) scan of the head and neck showed a right parietal fracture with underlying frontoparietal pneumocephalus [Figure 2]. Further investigation revealed multifragmentary burst fractures of the skull bilaterally accompanied by the presence of intracerebral foreign bodies exhibiting metal density and causing interhemispheric and subarachnoid hemorrhage. After briefly informing the patient's family of the dismal prognosis, the decision to proceed with the reanimation procedures and surgical intervention was taken. Following initial surgical stabilization according to the Advanced Trauma Life Support guidelines, the patient was transferred to the neurosurgical operating room and underwent left frontotemporal decompressive craniotomy with ligation and transection of the anterior third of the superior sagittal sinus, and microscopic resection of intraparenchymal foreign bodies. After surgery, the patient was transferred to the intensive care unit (ICU) for postoperative care. Despite following standard of care bundles to prevent surgical site infection, the administration of 1 g of topical vancomycin in the subgaleal space, and tight glycemic control, he eventually developed signs of infection 3 days after surgery, and a control CT scan of the head showed evidence of abscess formation and wound dehiscence, accompanied by obstructive hydrocephalus [Figure 3]. Revision surgery consisted of an enlargement of the first craniotomy and positioning of a ventriculoperitoneal shunt. During the procedure, a tense brain parenchyma was encountered, and the complete excision of the 3.5 cm abscess located within the left frontoparietal region was performed. Methicillin-resistant *Staphylococcus aureus* was isolated from the culture and the patient was initiated on combined intravenous and intrathecal antibiotic treatment with vancomycin hydrochloride 500 kU intravenously every 6 h and 20 mg vancomycin once a day through lumbar puncture. The patient was subsequently transferred to the ICU where he persisted in a state of coma (GCS: E2M2V2, open eyes in response to pain, extension to painful stimuli, and incomprehensible sounds) with normalized intracranial pressure (ICP) and hemodynamic stability. Three days

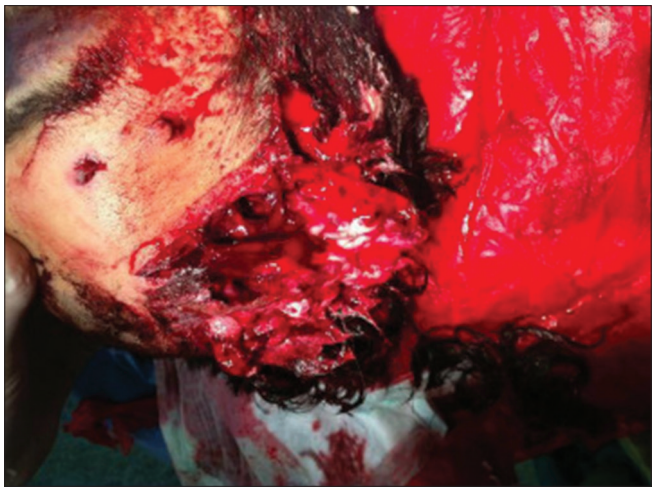

Figure 1: The left frontoparietal lacerated and contused wound extending to the frontosphenoidal suture measuring 6 cm in maximum diameter.

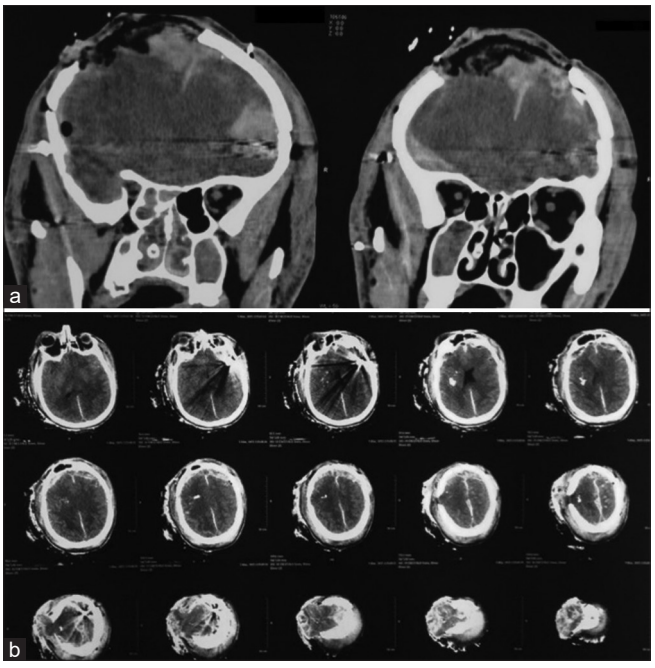

Figure 2: Preoperative CT scans of the head demonstrating (a) a right parietal fracture with underlying frontoparietal pneumocephalus and (b) bilateral multifragmentary burst fractures of the skull accompanied by the presence of intracerebral foreign bodies exhibiting metal density and causing interhemispheric and subarachnoid hemorrhage.

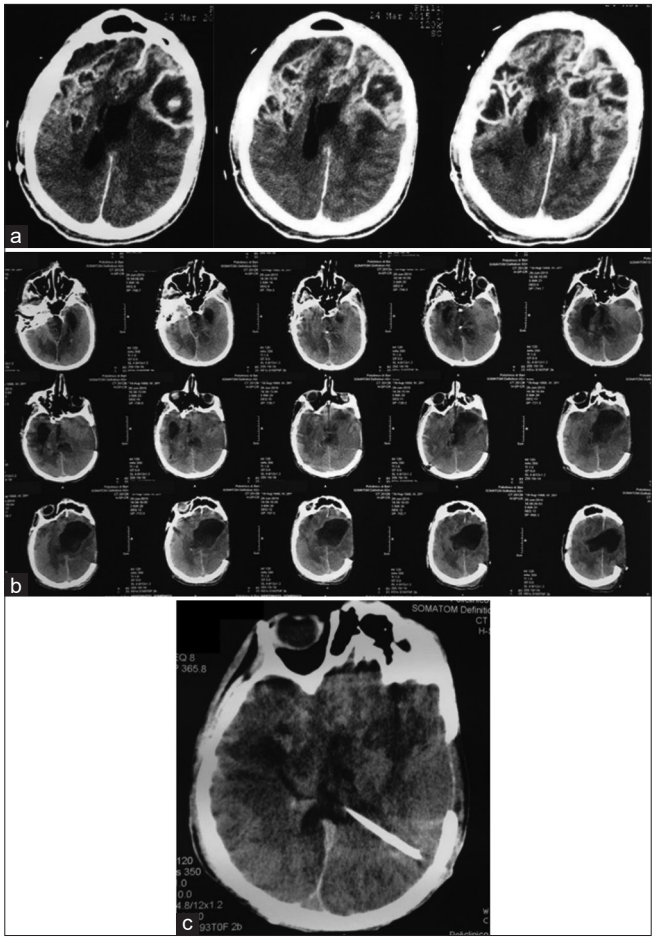

Figure 3: Following the first surgery, the patient developed signs of infection, and a control CT scan of the head showed (a) evidence of abscess formation and wound dehiscence and (b) obstructive hydrocephalus. (c) Subsequent surgical treatment consisted of an enlargement of the first craniotomy and positioning of a ventriculoperitoneal shunt.

later, he was transferred to a neurorehabilitation facility but unfortunately died 1 month after the initial admission. The postmortem examination revealed evidence of basilar and interhemispheric subarachnoid hemorrhage.

DISCUSSION

Definition of a complex entity

TBI is a physical and psychological injury that has been considered a major public health concern for decades, largely associated with motor vehicle crashes, accidental falls, and sports collisions.[3] The pathophysiological mechanisms consist of either structural or functional damage caused by external forces which ultimately determine transient or permanent deficit of different cognitive functions. TBIs can be classified as mild, moderate, or severe, based on the 15-point GCS that defines the severity of the injury as mild (13–15), moderate (9–12), and severe (3–8) with regard to eye, verbal, and motor responses.[21] Recently, explosion-related TBIs have become one of the most frequent injuries in both the military and the civilian setting, leading health-care professionals to identify a new pathophysiological entity, termed blast-induced neurotrauma.[3] Recently, the American Congress of Rehabilitation Medicine has proposed a clinical characterization of blast-induced mild TBI as a brain injury resulting in at least one of loss of consciousness for 30 min or less; posttraumatic amnesia for <24 h; any alteration of the mental state shortly after the accident; or focal neurological deficit(s) that may or may not be transient.[19]

Extent of the burden

In a recent article, Phipps *et al*.[15] have defined explosion-related TBI as one of the signature wounds of war, being strictly associated to battleground's head injury associated with a wide variety of neurological signs, including loss or reduction of consciousness, amnesia, auditory and visual impairment, recurrent headaches, and mental disorders. In an extensive analysis of the medical records of 22.203 personnel from the US Army Special Operations Command who completed assessments for cognitive impairment, postconcussive syndrome, and PTSD symptoms, 2813 (12.7%) had at least one mild TBI and 1476 (6.6%) reported clinical symptoms of PTSD. Four hundred and ten (14.6%) of personnel with mild TBI also referred PTSD symptoms.[8] Demographic analyses disclose the epidemiological relevance that TBI has had in the public health system during the past decades, with an estimated prevalence of 1.4 million Americans being diagnosed every year.[9] In their review of blast-related TBI, Rosenfeld *et al*.[18] showed that among 1.64 million US military personnel deployed to Iraq and Afghanistan between 2001 and 2008, 320.000 suffered from TBI and that women were more likely to report it. This sex-based difference matches the results obtained by Mu *et al*.[13] who found that the menstrual phase affects the TBI outcome, demonstrating that concussions were more frequent in female athletes and that they needed more time to recover from the neurological deficits.

Pathophysiology and clinical aspects of blast-related TBI

Recent classifications of blast-related TBI comprise five factors that reflect the gravity of the experienced trauma and suggest to the clinician various mechanisms of injury and consequent pathology caused by the explosion. The majority of deceases are not to be associated with the explosion's blast wave alone, which most frequently causes ear damage, pulmonary trauma, and concussions. For instance, penetrating injuries are associated with 70% of moderate-to-severe blast-induced TBIs and are extensively

more invalidating, as a consequence of the wide variety of fragments propelled by the detonation that can lead to fatal outcomes. Further analysis reveals also tertiary mechanisms that comprise bone fractures, traumatic amputations, and collapsing of buildings that result in crushing of the body. Moreover, quaternary injuries may result from burns, asphyxia, and exposure to toxic inhalants usually contained within the explosive.[2]

Blast-related mTBI is reported to be associated with impairments in sensory and neurocognitive functions, which tend to be most prominent immediately after the injury and usually diminish 3 months after the accident.[6,10,14,17] The damage secondary to the explosion takes place within microseconds after the blast shock wave passes through brain tissue; its initial cellular effects are much smaller than the resolution offered by conventional CT imagining. In mTBI, the acute day-of-injury CT scan is typically unremarkable but when positive will usually demonstrate subtle abnormalities such as mild edema, petechial hemorrhages, and/or small surface contusions.[20] Among the studies that leveraged imaging, the most commonly reported neurological abnormalities are white matter irregularities, cerebellar damage, thalamic network architectural differences, metabolic activation, diffuse axonal injury, and sensorimotor impairment.[7,22] As for clinical manifestations, visual disabilities are reported in 40–68% of patients with photosensitivity and decreased visual acuity being the most commonly described complaints.[6,10,12,14,16] Walsh *et al*.[24] showed at least one visual field defect (scatter, hemianopia, quadrantanopia, altitudinal, central, and constricted) in 64% of eyes examined in participants with blast-related mTBI. Other visual impairments reported include oculomotor dysfunction, floaters, pain, diplopia, and reading difficulty.[12,16]

Blast-related severe TBI

As for blast-related severe TBI, this condition is characterized by cerebral edema, intracranial hemorrhage, delayed vasospasm, and pseudoaneurysm formation.[1,11]

Clinical management of blast-related severe TBI includes the assessment of the airway, breathing, and circulation and the GCS scores. The patient should undergo a CT scan as soon as possible to evaluate possible brain injuries, such as skull fractures, intracranial hemorrhage, or cerebral edema.

Attention to secondary brain injuries, being SBP < 90 mmHg or SpO_2 < 92%, should be paid during prehospital management, as they both appear to increase mortality. Along with these, preintubation in patients with a GCS score <9 is not recommended, as it may result in excessive overventilation, consequent to transient hypoxia.[23]

Adequate oxygenation in the hospital setting, functional cerebral perfusion, and control of ICP are vital to avoid further damage, given that intracranial hypertension is a common finding in severe TBI.

Blood in the basilar cisterns might suggest a higher severity of the lesion, whereas a subarachnoid hemorrhage, revealed with the CT scan, usually leads to hyperemia and severe edema in the acute period.[11]

Vascular damage, unexplained variations of the neurological status, or sudden alteration observed by ICU monitoring imply the need for angiography or transcranial Doppler, due to the high risk of pseudoaneurysms formation and cerebral vasospasm.[1,11]

Early decompressive craniotomy has been a major factor in the improvement of survival and outcomes for patients with these severe injuries. Extensive unilateral or bilateral decompressive craniotomies are often needed to treat gross brain swelling and increased ICP. Cranioplasty could represent a viable option after the craniotomy; however, a latency of 3 months is recommended following evidence of infection. Ventriculostomy or lumbar drain can be performed if there is evidence of concomitant CSF leaks. Besides surgical treatment, hypertonic saline solutions and mild hypothermia (34–36°C) seem to have a beneficial effect on delayed intracranial hypertension.[11] However, prognosis remains uncertain with better outcomes recorded in the military population with isolated blast-related TBI and penetrating severe TBI when compared with those of a matched civilian population.[5] Furthermore, a study by de la Plata *et al*.[4] found that 16–26 years old and 27–39 years old showed better and faster improvement in functional abilities (e.g., following commands, performing daily activities, and engaging in recreational activities) as compared to individuals over 40 years, suggesting that neuroplasticity plays a role in the recovery process.

Summary of the present case

In the present report, a case of a 26-year-old male with blast-related severe TBI that occurred in a civilian setting is described. On his initial assessment, the patient presented in a comatose state with a left frontoparietal lacerated and contused wound, hypotension, and tachycardia. A head CT scan revealed multifragmentary burst fractures of the skull bilaterally accompanied by the presence of intracerebral foreign bodies, indicating that both primary and secondary explosion-related mechanisms had concurred in the damage. Emergency surgical treatment consisted of a left frontotemporal decompressive craniotomy with ligation and transection of the anterior third of the superior sagittal sinus and microscopic resection of intraparenchymal foreign bodies. Subsequent reoperation was needed as the patient soon exhibited signs of infection and obstructive hydrocephalus, and intravenous and intrathecal antibiotic

treatment was initiated. Unfortunately, the patient showed progressive worsening of the neurological condition and died 1 month after the accident.

CONCLUSION

Blast-related TBI has come to represent a serious concern in both the military and the civilian settings. The effects of a bomb blast on the human brain depend on many factors, such as blast energy, distance from the blast, and body position at the moment of explosion, therefore, resulting in damage of variable gravity. Management of this kind of trauma may be challenging, especially for severe TBI occurring in the civilian setting. In the present report, the authors describe the case of a 26-year-old patient presenting in a comatose state with a left frontoparietal lacerated and contused wound, hypotension, and tachycardia. Notwithstanding surgical and antibiotic emergency treatment, the patient died 1 month after the accident. Therefore, it appears clear that early neurosurgical and pharmacological treatment is crucial to favor the outcome of these patients, although evidence-based treatment algorithms are still needed.

Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

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