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Case Report and Literature Review: A Severe Case of Blast-Related Traumatic Brain Injury

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Abstract

Blast-related traumatic brain injuries (bTBIs), once considered the signature wound of wars, have increasingly affected civilian populations due to the rise in terrorist attacks and industrial accidents. These injuries are complex, resulting from a combination of primary blast effects, secondary projectiles, tertiary impacts, and quaternary injuries from burns and toxic gas inhalation. Understanding the clinical presentation, management strategies, and outcomes of bTBIs is essential for enhancing patient care and improving prognosis. We report a case of industrial-related severe bTBI with opened depressed skull fracture and intracranial hematoma. The patient underwent decompressive craniectomy and evacuation of clot but postoperatively had a stormy recovery and multiple complications. He eventually succumbed due to his complications. This underscores the complexity of bTBIs and highlights the importance of a multidisciplinary approach in the management of bTBIs. Further research is needed to optimize treatment protocols and rehabilitation strategies for individuals with bTBIs.

Keywords

- case report
- blast-related traumatic brain injury
- decompressive craniectomy
- Glasgow Coma Scale
- industrial blast
- industrial injury
- multidisciplinary approach
- oxidative stress

Case Report

Mr. H, a 60-year-old Chinese gentleman, was brought into the accident and emergency department for alleged industrial injury. He was initially sent from a railway construction site to a district hospital, which then referred him to us. He was identified as a construction worker working for the East Coast Rail Link (ECRL) project in Pahang, Malaysia. According to the witness, he was found near a blasting site where explosives were used to blow up solid rock for the construction of railway tracks. When he was found, he was buried by a landslide of sands and rocks. The other workers removed him from the landslide and noticed he was not responding coherently, with multiple puncture wounds on his body. He was then rushed to the nearest district hospital.

In the district hospital, his Glasgow Coma Scale (GCS) on arrival was E4V4M5 with bilateral equal and reactive pupils. Blood pressure was 76/45 mm Hg, heart rate was 105/min, and saturation was 86% under room air. It was immediately evident that there were multiple small size puncture wounds over the scalp, face, arms, trunk, and abdomen. There was a larger puncture wound over the right parietal scalp and three larger ones on the right chest. Primary survey revealed a right pneumothorax. A chest tube was inserted immediately and he was resuscitated adequately with crystalloids and received transfusion of 1 pint packed red cell. Chest X-ray showed right second to sixth rib fracture with hemopneumothorax. Post-Chest tube insertion, his GCS was noted to be E1V2M4. He was intubated in the district hospital for airway protection and transported to our center for urgent computed tomography (CT) of the brain and further neurosurgical management.

We attended to him when he arrived at our hospital and noted his GCS on arrival to be E1VtM1 under sedation. He

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was on a cervical collar and ventilated by a ventilator; recorded vital signs were the following: blood pressure 124/88 mm Hg, heart rate 101/min, and saturation of 100%. His right and left pupils were 8 mm and 1 mm, respectively. There were multiple small to large puncture wounds over his head, face, and neck with foreign bodies embedded within. Notably, three separate large puncture wounds were found on the right lateral aspect of his head—with one on the right parietal/posterior most about 3×3 cm in size, appearing punched out with an island of skin loss, and protruding fractured bone fragments ►**Fig. 1**. The scalp also appeared to be covered in soot and dirt-like material.

There were also multiple puncture wounds of varying sizes all over the body and upper limbs as shown in ►**Fig. 2**. His chest tube was functioning and once he was stabilized, an urgent CT of the brain and cervical spine was done. CT of the brain showed a right temporal and parietal comminuted skull fracture with intraparenchymal bleed of about 20 mL in volume. Multiple small foreign bodies were seen lodged in the scalp and within the fracture site. There was the presence of cerebral edema and midline shift ►**Figs. 3** and **4**; hence, he was prepared for urgent right decompressive craniectomy and clot evacuation. We anticipated a certain degree of difficulty in having a good approximation of the scalp in view of the 3×3 cm punched-out skin loss. The plastic surgery team was consulted prior to surgery. The plastic surgeons decided to provide assistance for scalp wound closure while at the same time conducting wound exploration, debridement, and removal of foreign bodies.

Diagnosis

The patient was diagnosed with severe blast-related traumatic brain injury (bTBI).

Operative Procedure

His head was cleaned thoroughly with chlorhexidine shampoo prior to surgery. Any dirt, debris, and foreign bodies were removed. The right decompressive craniectomy incision will incorporate the preexisting scalp wound. Trauma flap was designed starting at the zygomatic arch less than 1 cm anterior to the tragus, then proceed superiorly, and then curve posteriorly at the level of top of the pinna; about 4 cm behind the pinna, it was taken superiorly incorporating the open skull fracture wound. The incision extended to about 2 cm ipsilateral to the midline and curved anteriorly to end behind the frontal hairline. After raising the scalp, the pericranium was harvested for fascia-duraplasty. Craniectomy was done by connecting 4 burr holes, one each at McCarthy's keyhole, frontal burr hole, temporal burr hole, and parietal burr hole. Bone opening was large and beyond 12 cm in diameter. The bone flap was removed in a single large piece with comminuted fragments still attached to it. Some remaining comminuted bone fragments over the right parietal was removed using forceps pieces by pieces. The bones were discarded and returned to the patient's next of kin in view of gross contamination.

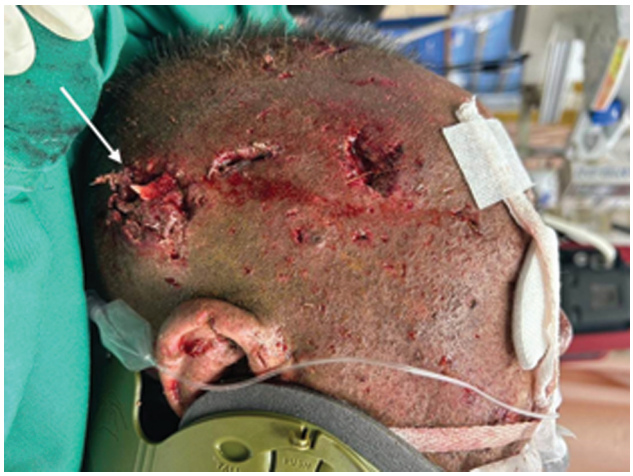

Fig. 1 Right lateral view of Mr. H's head. Note the multiple puncture wounds on his scalp and face. Laceration wounds can also be seen on his ear. Fragments of fractured skull can be seen protruding from the wound (*arrow*).

Upon removal of the bone flap, it was evident that the right parietal dura has been breached and blood clot could be seen pushing its way upward through the dura defect. The temporal bone and part of the sphenoid wing were rongeured to the base of the middle cranial fossa for adequate decompression. The dura was then opened with the base facing the middle skull base and releasing incisions were done circumferentially up to the bone margins. The clot from the parietal burst lobe was then removed with warm saline flush and gentle suction. Bleeding from the brain parenchyma was secured with Surgicel (oxidized cellulose). Small cortical contusions could also be seen in the frontal region and was left undisturbed. Post decompression and clot evacuation, the brain appeared mildly swollen but pulsatile.

Expansile fasciaduraplasty was done using the pericranium and tacking suture was applied just enough to fixate it in place.

A subgaleal drain was inserted below the temporalis muscle, and the temporalis muscle, subcutaneous tissue, and scalp were closed in layers. During scalp closure, the area of skin defect that was debrided appeared gapping and the plastic surgeon was able to close the scalp after flap transposition. Prior to ending the surgery, both neurosurgery and plastic surgery doctors attempted to remove as much foreign bodies embedded within the scalp and skin as possible. Multiple interrupted sutures were applied to areas where debridement was done during the removal of foreign bodies ►**Fig. 5**.

Progress

Postsurgery he was shifted to the intensive care unit (ICU) for cerebral resuscitation. Analgesia, antiepileptics, and antibiotics were given regularly. He required high inotropic support during the first 2 days and subsequently was able to taper off. Clinically, while under cerebral resuscitation, his pupils measured 7 mm in the right eye and 1 mm in the left eye postsurgery. The craniectomy site was swollen but soft,

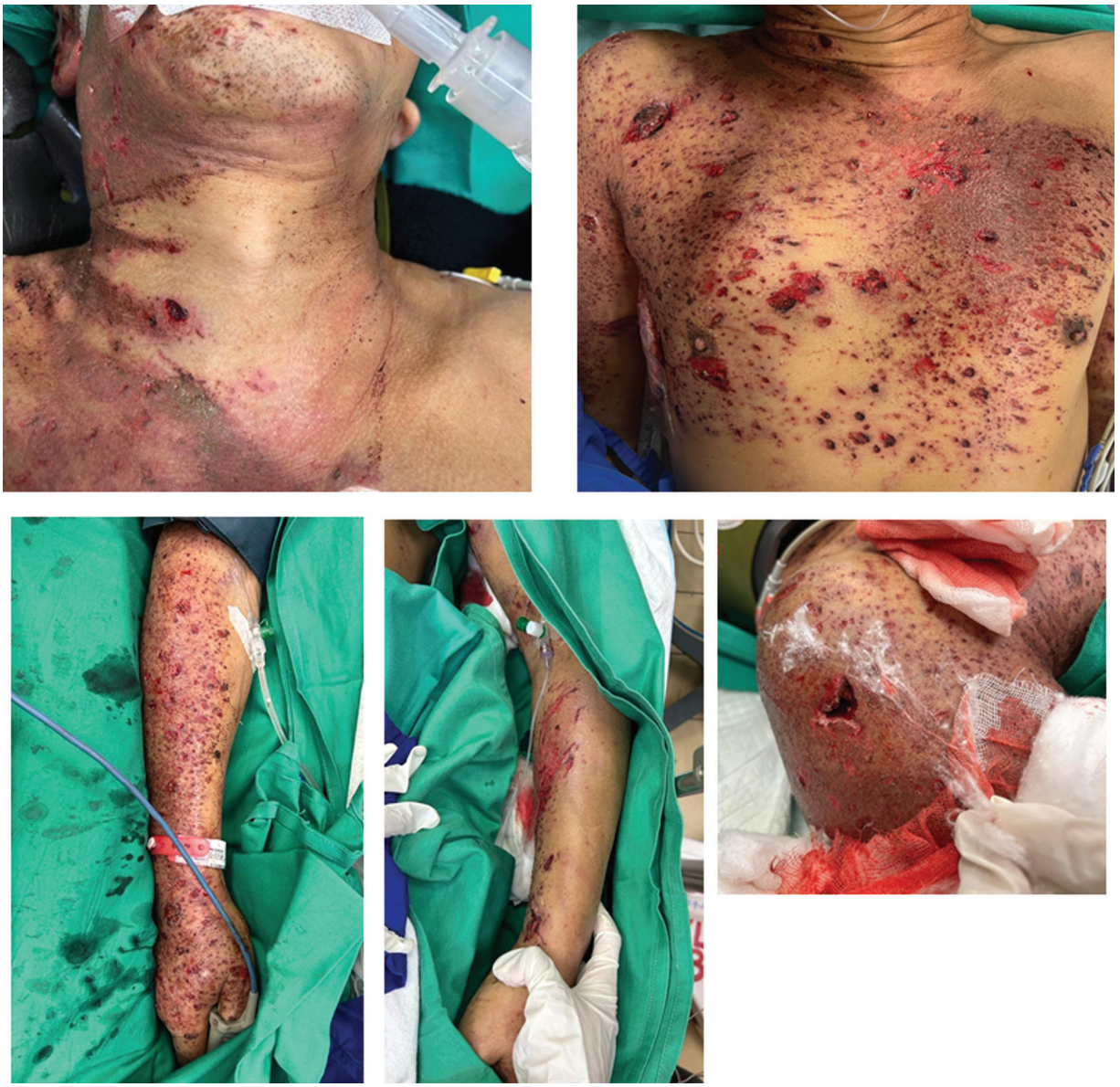

Fig. 2 Multiple small puncture wounds over the neck, body, and arms with areas covered in soot, which can be likely attributed to burn injury.

not tense. A repeat CT of the brain on day 2 postsurgery showed extracalvarial herniation of the right hemisphere, improving midline shift, and evacuated clot over the right parietal region. Unfortunately, there were also new evolution of intraventricular bleed and intraparenchymal bleed ►**Fig. 6**. This clot was left undisturbed.

On day 3 after surgery, we noticed necrosis over the topmost part of the scalp flap. His recovery was poor with a GCS of E1VtM2. The plastic surgeons decided to treat the scalp necrosis conservatively via dressing and prevention of infection. A tracheostomy tube was inserted for weaning off ventilator and tracheal toileting at 1 week after surgery. He managed to wean off ventilator support and moved out of the ICU. There were multiple bouts of fever due to nosocomial infection. He eventually developed sepsis secondary to surgical site infection with concurrent hospital-acquired pneumonia and succumbed to his illness 1.5 months after the trauma.

Discussion

Traumatic brain injuries (TBIs) are categorized into mild, moderate, or severe based on the GCS, which assesses injury severity by evaluating eye, verbal, and motor responses. A GCS score of 13 to 15 indicates a mild TBI, 9 to 12 a moderate TBI, and 3 to 8 a severe TBI. Although bTBIs are still relatively rare in Malaysia, they have become increasingly common globally over the past decade, especially in military and civilian contexts.[1] Historically, bTBI was primarily associated with warzones and considered a signature wound of combat. However, it is now recognized as a growing concern among civilians, particularly in areas impacted by conflict, terrorism, or the use of explosive devices. Several factors contribute to the rising incidence of bTBI among civilians, including the following:

- *Terrorist activities and armed conflicts*: The prevalence of urban warfare, armed protests, and terrorist attacks in

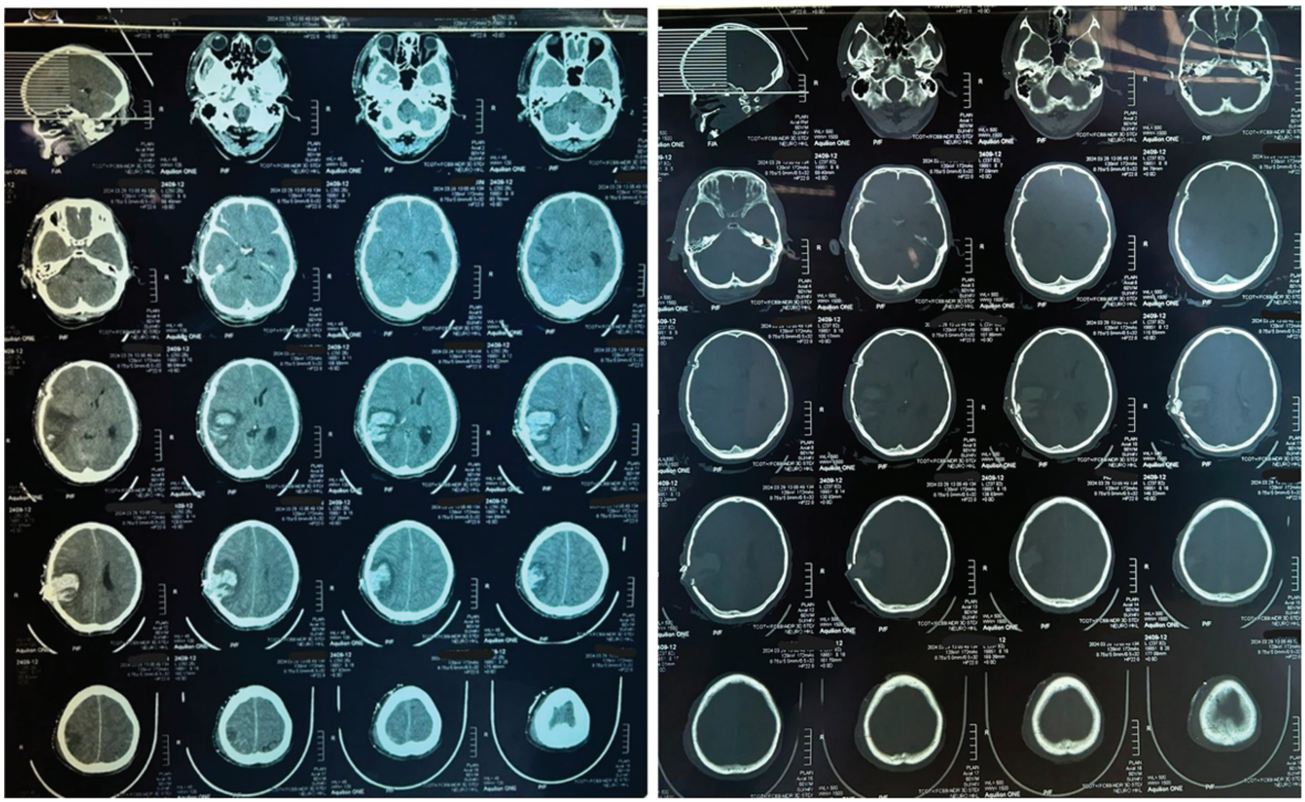

Fig. 3 Computed tomography (CT) of the brain of Mr. H showing a right temporal and parietal bleed with areas of comminuted skull fractures and multiple foreign bodies.

densely populated areas has increased the likelihood of civilians being exposed to blasts. The risk of exposure to explosive devices, such as improvised explosive devices (IEDs) or vehicle-borne improvised explosive devices (VBIEDs), is higher in these scenarios, leading to civilian casualties and injuries, including bTBI.

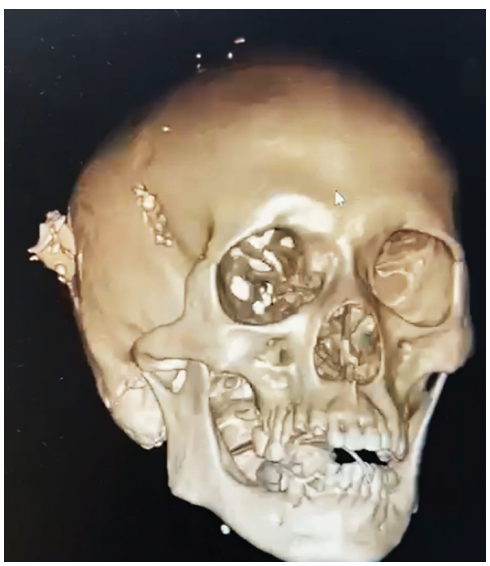

Fig. 4 Computed tomography (CT) reconstruction of the bone window showing two areas of comminuted skull fracture, one at right temporal and another larger one at the right parietal. Multiple foreign bodies can be seen above the skull.

- *Industrial accidents*: In industrial settings where explosives are used or stored, accidents can occur, resulting in explosions and blast injuries among workers and nearby civilians. Inadequate or compromised safety protocols can particularly lead to bTBI in these environments.
- *Accidental explosions*: Accidental explosions, such as those resulting from gas leaks and firework mishaps, can also cause bTBI among civilians. These incidents may happen during celebrations, construction activities, or industrial operations, underscoring the need for vigilance and safety measures.

It is important to understand that explosives are classified as high-order explosives (HE) or low-order explosives (LE). HE generate a supersonic over-pressurization shock wave, with examples including TNT, C-4, Semtex, nitroglycerin, dynamite, and ammonium nitrate fuel oil (ANFO). In contrast, LE create a subsonic explosion and lack the over-pressurization wave of HE. Examples of LE include pipe bombs, gunpowder, and most pure petroleum-based bombs like Molotov cocktails.

The Centers for Disease Control and Prevention (CDC) divides the mechanism of bTBI into four stages as seen in ►**Table 1**, which highlights the different characteristics and consequent pathologies caused by an explosion.[2–4]

- **Primary injury** (unique to HE): Initial damage to the brain that occurs when the supersonic blast wave generated by the detonation impact traverses the head and brain. Explosion/detonation creates a transient pressure wave

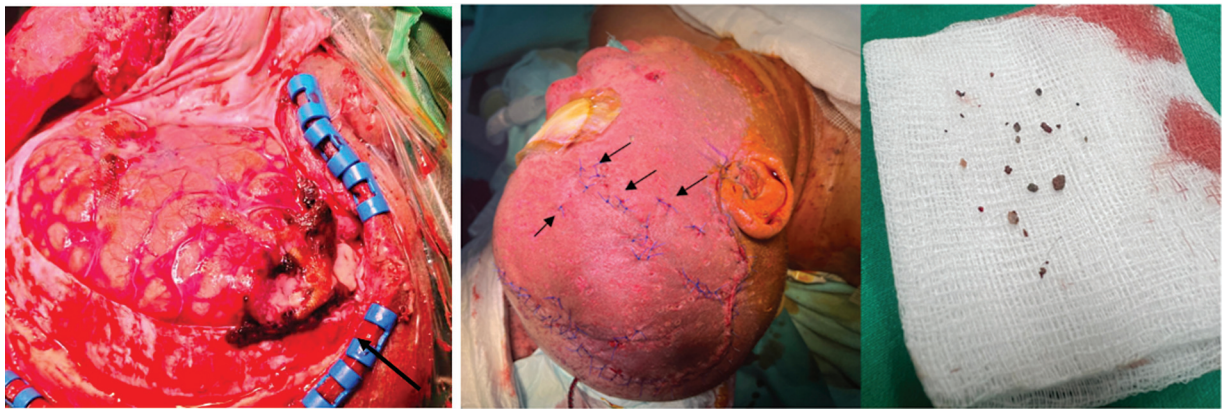

Fig. 5 Left: Intraoperative image showing the area of parietal burst lobe (*arrow*) after craniotomy. Middle: Postoperative surgical wound of the patient. Note the area of defect was closed using mattress suture to prevent breakdown. Multiple small puncture areas are also seen on the scalp. Areas where there are gapping postdebridement were sutured with interrupted sutures (*arrows*). Right: Picture showing the amount of small foreign bodies (sands and debris) found within a single pin hole puncture site that was removed after exploration and debridement.

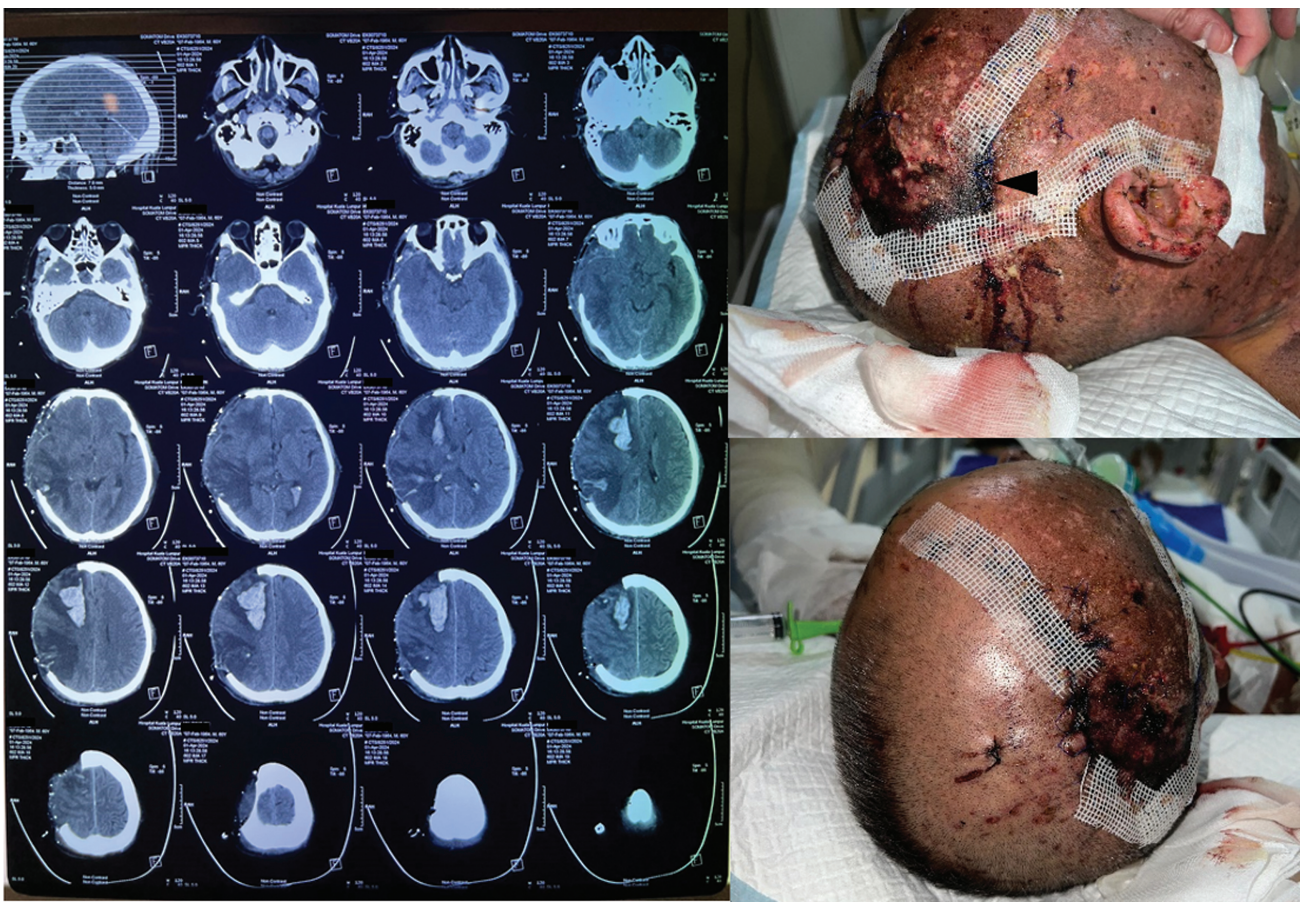

Fig. 6 Left: Repeated computed tomography (CT) of the brain postsurgery shows transcalvarial herniation, a well-evacuated right parietal contusional bleed with new bleeding seen over the right frontal and intraventricular bleed. Right: Scalp flap necrosis over the topmost region surrounding part of the surgical scar and laceration wound (*arrowhead*).

disturbance that travels through a medium like air or water. This pressure wave travels faster than the characteristic wave speed of the host medium (air or water), and compresses to become over-pressured blast waves. ►**Fig. 7** The blast wave is followed by pressure decay with varying peak amplitudes. An explosive blast wave induces localized particle motion in the medium it is passing through. Any object, such as the head in the path of a blast wave will experience forces that will move it and deform it. This could lead to blast lung, tympanic membrane rupture, abdominal injuries, and cerebral concussion as seen in ►**Table 1**.

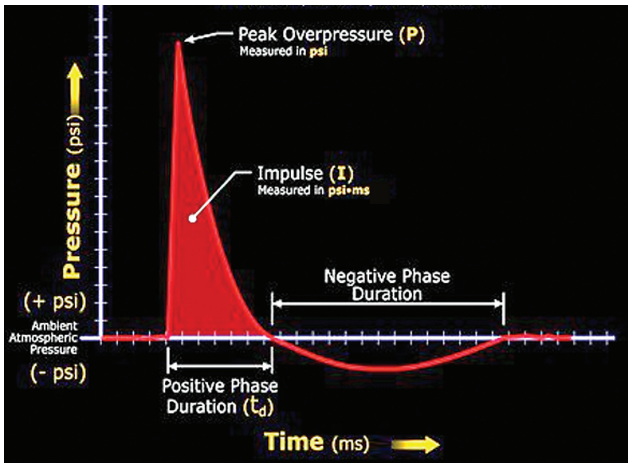

Fig. 7 A Friedlander curve shows the profile of an ideal blast wave where there is a sudden rapid rise in pressure followed by a "vacuum" duration.[2]

- **Secondary injury:** Injuries result from objects propelled by the explosion, which can include fragments of projectile from the weapon casing as well as debris from the surrounding environment. Low-velocity projectiles, or projectiles with relatively lower kinetic energy, may penetrate the skull but cause little underlying parenchymal damage, whereas very high-velocity, or high kinetic energy, projectiles can cause massive tissue damage from a large secondary cavity. The cavity creates large enough volumes that can be of such pressure that skull fracture occurs with fragments expanding in an outward displacement fashion.
- **Tertiary injury:** Injuries sustained when the force of the explosive blast propels the patient, causing them to collide with objects like walls or the ground. This impact can lead to head injuries, including skull base fractures and injuries to the middle fossa, temporal bone, or frontal sinus. These patients exhibit symptoms like otorrhea (ear discharge) or rhinorrhea (nose discharge). Additionally, other injuries that may be sustained include traumatic amputations, crush injuries, and bone fractures (►**Table 1**).
- **Quaternary injury:** This is an injury secondary to other products or chemicals related to the explosive device, such as burns or toxic fume inhalation. Any complications of injuries such as asthma and rhabdomyolysis also fall into this category (►**Table 1**).

An individual may be subject to repeated blast exposures from the same incident as reflections of the blast wave bounce off solid objects, akin to an acoustic echo. As of now, there is inadequate evidence to endorse a singular injury mechanism for all cases. Nevertheless, multiple mechanisms as described above have been validated through previous research. In our patient, the hemopneumothorax was likely due to primary blast lung injury or a tertiary injury resulting from collision, causing associated rib fractures. The secondary injury due to

Table 1 Mechanisms of blast injury, its characteristics, and possible type of injuries

Category	Characteristics	Body part affected	Type of injuries
Primary	Unique to HE, results from the impact of the over-pressurization wave with body surfaces	Gas-filled structures are most susceptible: lungs, GI tract, and middle ear	• Blast lung (pulmonary, barotrauma); hemothorax, pneumothorax, pulmonary contusion, and hemorrhage • Tympanic membrane rupture and middle ear damage (ossicular disruption), cochlear damage • Abdominal hemorrhage and perforation (bowel perforation, ruptured liver or spleen) • Globe (eye) rupture • Concussion (TBI without physical signs of head injury)
Secondary	Results from flying debris and bomb fragments	Any body part may be affected	• Penetrating ballistic (fragmentation) or blunt injuries • Eye penetration • Foreign bodies
Tertiary	Results from individuals being thrown by the blast wind	Any body part may be affected	• Fracture and traumatic amputation • Crush injuries • Closed and open traumatic brain injury (TBI) • Spinal cord injury • Cardiac contusion
Quaternary	All explosion-related injuries, illnesses, or diseases not due to primary, secondary, or tertiary mechanisms Includes exacerbation and complications	Any body part may be affected	• Burns • Asthma, COPD, or other breathing problems from dust, smoke, or toxic fumes • Angina • Renal failure due to rhabdomyolysis

Abbreviation: COPD, chronic obstructive pulmonary disease; GI, gastrointestinal; HE, high-order explosives.
Source: Adapted from Centers for Disease Control and Prevention (U.S.).[5]

projectiles was particularly evident, as multiple puncture wounds were observed throughout, along with an outward-displaced bone fracture.

Effect of Blast Wave on the Brain in bTBI

Several mechanisms have been proposed to explain the effect of blast waves in bTBI. The acceleration mechanism theory states that an overpressure wave created by an explosive device causes macroscopic translational and rotational acceleration of the brain, resulting in compression and shearing of brain tissue. Additionally, the motion of the brain within the cranium can produce coup and counter-coup injuries, leading to contusions, lacerations, subdural hematomas, or inducing diffuse micro-hemorrhage and axonal injuries.

The direct transmission mechanism theory claims that when a pressure wave transmits through the brain, some of its energy is absorbed by structures within the brain. These include brain parenchyma, blood vessels, and circulating cerebrospinal fluid (CSF). CSF and tissues of different densities can reverberate within the skull, resulting in the shearing and rupture of tissues and blood vessels. The transition from pressurized to negative pressure can produce cavitation, a process where implosion effects lead to the expansion of dissolved gases, which subsequently burst, releasing kinetic energy that damages surrounding tissue.

A more controversial mechanism, called vascular surge, hypothesizes that the transmission of the pressure wave through the thorax causes a volumetric blood surge, increasing blood pressure within the cerebral vasculature to the extent that the blood–brain barrier (BBB) is breached, capillaries rupture, and hemorrhage becomes probable. This hypothesis is supported by animal models in which pigs, rats, or rabbits are exposed to experimental whole-body or thorax-only blast injuries.[4]

Clinical Effect of bTBI

Clinically, bTBI patient's condition may be as mild as a brief period of confusion to as severe as a coma or death. bTBI is presently categorized similarly as conventional TBI. Mild bTBI is associated with brief ($<$30 minutes) loss of consciousness or awareness and a GCS score of 13 to 15. Mild bTBI may lead to prominent sensory and neurocognitive impairments, which usually improve within 3 months after the incident. There may be complaints of confusion, amnesia, as well as other symptoms such as difficulty concentrating, mood alteration, sleep disturbance, and anxiety. Clinical manifestations observed in mild bTBI often include visual impairments, affecting a significant proportion of patients, ranging from 40 to 68% with photosensitivity, and decreased visual acuity. Walsh et al showed at least one visual field defect (scatter scotoma, hemianopia, quadrantanopia, central scotoma, and constricted) in 64% of eyes examined in patients with mild bTBI. Other visual impairments reported include oculomotor dysfunction, floaters, pain, diplopia, and reading difficulty.[6] The cellular effects of blast wave are much smaller than the resolution of conventional CT imaging. Hence, mild bTBI is typically unremarkable on brain CT. However, if abnormalities are present, they usually demonstrate subtle abnormalities such as mild edema, petechial hemorrhages, and/or small surface contusions.[1,2]

For severe bTBI, patients present with reduced level of consciousness (GCS $\leq$8) and require urgent assessment, stabilization, and resuscitation based on trauma protocol. This is seen in the case we presented here. Primary assessment includes airway, breathing, circulation, and GCS scores. Many of these patients require medical care and stabilization while still on the field prior to transportation. It is crucial to prevent secondary brain insult, as systolic blood pressure (SBP) below 90 mm Hg or oxygen saturation (SpO_2) below 92% has been associated with increased mortality rates.[1] Patients with severe bTBI should undergo a CT scan as soon as possible to evaluate the brain injuries. CT of the brain may reveal cerebral edema, intracranial hemorrhage, foreign bodies, and skull fractures. Penetrating injuries are reportedly associated with up to 70% of moderate to severe bTBIs as a wide variety of fragments can be propelled by the detonation. Blood in the basilar cisterns suggest a higher degree of injury and carries risk of delayed vasospasm. Vasospasm is often the cause of delayed neurological deterioration and seems to be more common following a blast than other types of TBI, and has led military neurosurgeons to perform decompressive craniectomies more often than commonly done for TBI. Ling et al[2,3] revealed that up to 47% of patients who suffer moderate to severe bTBI may develop cerebral vasospasm. Early transcranial Doppler studies demonstrates that demonstrate that this vasospasm can develop early, often within 48 hours of injury. Vasospasm can also present later in the course of the disease, typically $\geq$10 days after initial injury.

Blast injuries can have severe vascular effects, such as sagittal sinus injury and lacerated cortical arteries. A 35% incidence of pseudoaneurysms was reported in blast-injured patients who underwent cerebral angiography at the National Naval Medical Center.[3,7] These patients usually have unexplained worsening of neurological status or worsening intracranial pressure (ICP) while under monitoring. Blast-related pseudoaneurysms can expand or rupture and require treatment by appropriate endovascular or microsurgical techniques.

Role of Oxidative Stress in bTBI

Subacute or delayed intracranial bleeding can also be seen after bTBI, as seen in this patient. Research suggests that blast activates secondary biochemical mechanisms such as NOX-mediated oxidative stress, which leads to delayed increase in the permeability of BBB. A study by Kuriakose et al found a 10-fold increase in NOX1 expression 4 hours after blast injury in endothelial cells in the frontal cortex, which lasted for a few days postinjury. Oxidative stress causes breakdown of the tight junction complexes connecting adjacent endothelial cells, causing increased permeability of the BBB. In Kuriakose et al's experiment, they successfully used apocynin to prevent the increase in superoxide production and reduce the breakdown of tight junctions, suggesting a possibility to reduce secondary mechanism by means of pharmacological intervention ►**Fig. 8**.[8]

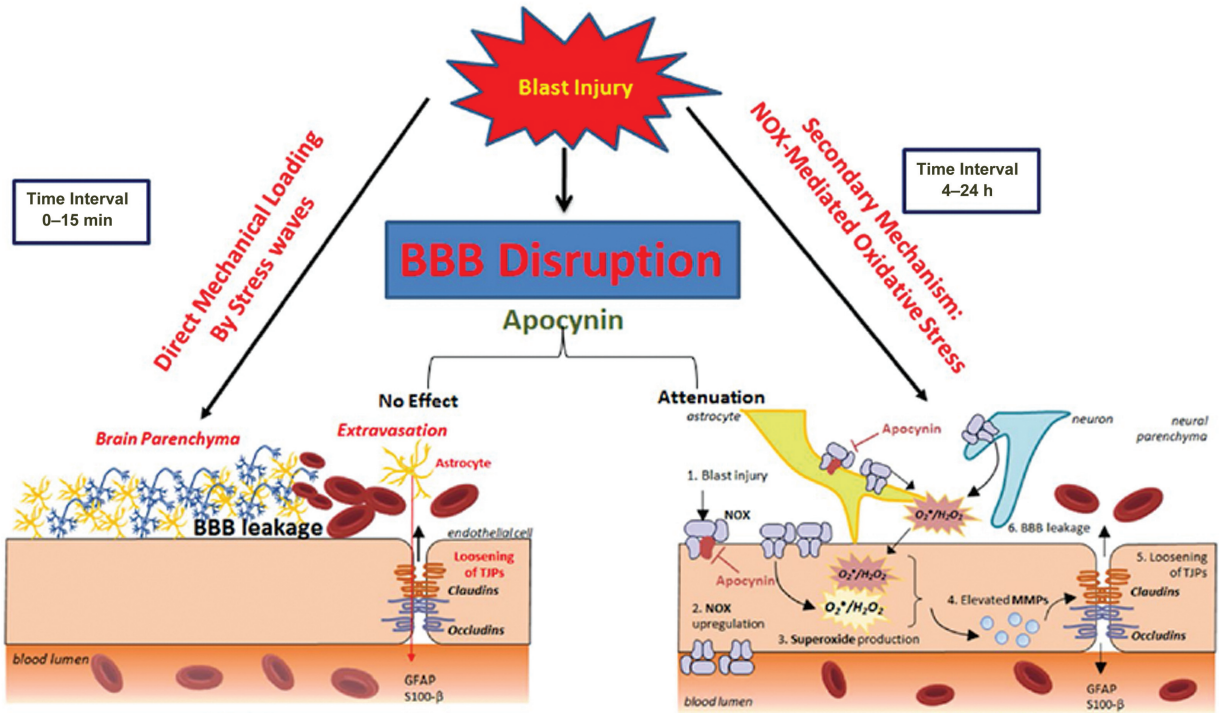

Fig. 8 Primary and secondary events that contribute to blood–brain barrier (BBB) permeability changes following blast traumatic brain injury (TBI). Left: Primary damage caused by direct mechanical loading by blast wave rupturing the vasculature immediately (0–15 minutes) following blast without any biochemical events. Right: Secondary biochemical mechanisms due to oxidative stress that are activated by blast. Oxidative stress derived by the upregulation of NOX on neurovascular endothelial cells (and other neural cells) cause increased superoxide production both within and outside the endothelial cells. This superoxide elevates matrix metalloproteinases (MMPs) production, which breaks down the tight junction complexes, causing increased permeability of the BBB. Use of apocynin attenuates this secondary mechanism. (Adapted from Kuriakose et al.[8])

Management of Severe bTBI

There are no specific guidelines customized for TBIs resulting from blasts. The emergency responder's first responsibility is to protect the patient from further harm so that primary survey of ABCs (airway, breathing, and circulation) can be carried out. After stabilizing the patient, it is important to ascertain the patient is GCS score. The GCS score can be helpful in making triage decisions, particularly in cases of isolated head injury. If the GCS score is low, the patient should be transferred to medical centers where CT scan and neurosurgical specialty are available. Patients suffering from severe bTBI typically present with multiple or complex injuries, all requiring simultaneous management. Hence, it is important to address a life-threatening condition to prevent secondary insults. A head CT scan should be done as soon as possible to identify lesions such as intracranial hemorrhage, skull fracture, cerebral edema, etc. Patients with severe bTBI will likely require measures such as airway protection, mechanical ventilation, neurosurgical procedures, ICP monitoring, and specialized neurological care in an ICU.

Increased ICP is common in severe bTBI. Some patients have also been noted to develop delayed increase in ICP by as much as 14 to 21 days after the injury. Armonda et al attribute this to delayed vasospasm.[7] Intravenous hypertonic saline can be used for temporary reduction of ICP in acute situation or given via infusion for refractory intracranial hypertension. For continuous intravenous infusion, 3% sodium chloride (NaCl) is the fluid concentration of choice. The benefit of hypertonic saline is that serum osmolality can be increased without decreasing the intravascular volume. This is critical as most blast injury patients suffer from a certain degree of hemorrhagic shock.

Decompressive craniectomy is a life-saving procedure that relieves the pressure on the swollen brain and allows the surgeon to remove the offending cause such as depressed skull fracture, intraparenchymal bleeding, subdural hemorrhage, etc. An ICP monitoring probe or a ventricular catheter can be inserted during the surgery for objective measurement of ICP. If foreign bodies (debris, shrapnel, etc.) are present within the surgical region, current practice is to remove any accessible fragments that can be safely debrided; but deep or subcortical fragments that are inaccessible are left in place. Studies based on other conflicts have shown that aggressive removal of all fragments is unnecessary, but removal of gross contamination with debridement of large injury tracts is beneficial.[2,3]

Recovery from severe bTBI is typically prolonged and often incomplete. Drawing from civilian TBI data, it is anticipated that a considerable portion of severe bTBI patients may not survive beyond 1 year postinjury. A 2018 study focusing on the southern Thailand conflict with bTBI found that a subfalcine herniation (midline shift) greater than 5 mm,

basal cistern effacement, and coagulopathy on admission are associated with a poor outcome after treatment.[9]

Conclusion

Blast injuries are not confined to the battlefield. They should be considered for any victim exposed to an explosive force. A thorough understanding of the mechanisms behind blast injuries can provide the attending neurosurgeon a better understanding of the type of bTBI. Nevertheless, the management of this kind of trauma may be challenging, especially for severe bTBI occurring in the civilian setting. In the present report, we describe the case of a 60-year-old man with industrial accidental severe bTBI who presented in a comatose state with a right parietal open skull fracture and intraparenchymal bleed who eventually succumbed to the blast-related quaternary insults of scalp necrosis and infections 1.5 months after surgery.

Funding
None.

Conflict of Interest
None declared.

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