

# Challenges in management of blast injuries in Intensive Care Unit: Case series and review

# Tanvir Samra, Mridula Pawar<sup>1</sup>, Jasvinder Kaur<sup>2</sup>

Blast injuries are rare, but life-threatening medical emergencies. We report the clinical presentation and management of four bomb blast victims admitted in Intensive Care Unit of Trauma center of our hospital in 2011. Three of them had lung injury; hemothorax (2) and pneumothorax (1). Traumatic brain injury was present in only one. Long bone fractures were present in all the victims. Presence of multiple shrapnels was a universal finding. Two blast victims died (day 7 and day 9); cause of death was multi-organ failure and septic shock. Issues relating to complexity of injuries, complications, management, and outcome are discussed. **Keywords:** Blast lung, bomb blast injuries, clinical presentation, outcome



# Introduction

A high intensity bomb blast exploded in Delhi on 7 September 2011. Four of the most severely injured were shifted to Intensive Care Unit (ICU) of our Trauma center after primary resuscitation by a team of advanced trauma life support providers in the emergency department (ED).

## **Case Reports**

#### Case 1

A 34-year-old male with decreased level of consciousness (Glasgow Coma Scale = 6), obstructed breathing, hypotension, right hemothorax and compound fracture in bilateral both bones of lower limbs and right humerus was managed in the ED with tracheal intubation, intercostal drain (ICD) insertion (500 ml of blood) and intravascular administration of crystalloids, colloids, blood products and inotropes (dopamine and noradrenaline).

#### From:

#### Correspondence:

Minimal peritoneal fluid collection was reported on Focused Assessment Sonography in Trauma (FAST); computed tomography (CT) abdomen confirmed right ileal injury. A large (7.2 cm × 3.7 cm) parietal hematoma and fracture right temporal bone was reported on CT head [Figure 1].

He underwent craniotomy and evacuation of hematoma, exploratory laparotomy, wound debridement and application of external fixator of lower limb on day 1 of admission.

Necessary laboratory investigations were conducted and 11 units of whole blood, 19 units of fresh frozen plasma (FFP) and 9 units of platelets were administered to him during his stay in the ICU. Radiological evaluation on day 5 reported cerebral edema (noncontrast computed tomography head) and consolidation in right upper lobe and bilateral lower lobes (chest X-ray [CXR] and CT chest). His clinical condition deteriorated due to development of septic shock and acute respiratory distress syndrome (ARDS). Hemodynamic management based on surviving sepsis guidelines<sup>[1]</sup> and lung protective ventilation<sup>[2]</sup> was instituted (FiO, 100%, pressure controlled ventilation-volume guaranteed, upper limit of pressure [Pimax] =40 cm H<sub>2</sub>O; tidal volume = 350 ml, positive end-expiratory pressure = 16, P/F ratio = 70, lung compliance = 11 ml/cm H<sub>2</sub>O).

Department of Anesthesia and Intensive Care, Lady Hardinge Medical College, <sup>1</sup>Safdarjung Hospital, <sup>2</sup>Department of Anesthesia and Intensive Care, PGIMER and Dr Ram Manohar Lohia Hospital, Baba Kharak Singh Marg, Connaught Place, New Delhi, India

Dr. Tanvir Samra, Department of Anesthesia and Intensive Care, Lady Hardinge Medical College, New Delhi - 110 001, India. E-mail: drtanvirsamra@yahoo.co.in

He had cardiac arrest on the  $7^{th}$  day and could not be revived.

# Case 2

A 58-year-old male presented with traumatic autoamputation of the distal part of both lower limbs and right sided pneumothorax managed by insertion of ICD. He was conscious, hemodynamically stable and maintained airway and oxygenation with ventimask ( $FiO_2 = 50\%$ ). He was operated for above knee amputation of the right limb and below knee amputation of the left limb on day 1. FAST and CT abdomen was normal [Figure 2].

On day 4, he was intubated and administered inotropes (dopamine 15  $\mu$ g/kg/min; noradrenaline 0.15  $\mu$ g/kg/min). Infected amputated stumps and abscess around chest drain insertion site communicating with a pleural space complicated his stay. Persistent right sided pneumothorax [Figure 3] with patchy consolidation in bilateral lower lobes of the lung was reported on repeat CT chest. CT abdomen and two-dimensional Echo were normal.

The patient developed renal failure (serum creatinine = 2.6 mg/dl; serum urea = 100 mg/dl), thrombocytopenia (platelet count =  $30,000/\text{mm}^3$ ) and an increased total leucocyte count (9900 on day  $0-25,000/\text{mm}^3$  on day 9) during his stay in the ICU. He was transfused 18 units of FFP, 9 units of leucodepleted packed cell and 8 units of platelets. Dopamine, noradrenaline, adrenaline and vasopressin in supraphysiological doses could also not maintain his hemodynamic and he expired on day 9.

## Case 3

A 23-year-old male with a brachial artery injury and compound fracture of the right humerus was shifted from ED to operating theatre for repair of the artery and transacted median nerve. Intraoperative course was uneventful except for the need of inotropic support (dopamine  $8 \mu g/kg/min$ ). Total blood products administered in first 24 h were 5 units of whole blood and three units of FFP. He was extubated on day 2 and transferred to ward on day 4. Saphenous vein graft necrosis was managed by redo surgery and hyperbaric oxygen therapy (1.2 ATA; 3 sessions). He was discharged from the hospital on day 15.

### Case 4

A 34-year-old male with right-sided hemothorax and initial ICD volume of 1200 ml of blood was maintained with intermittent application of noninvasive ventilation mask for first 48 h. He also had right sided tibial fracture [Figure 4], right-sided fracture of first proximal phalanx. Four units of whole blood were transfused in first 24 h and patient was discharged on day 4 from the ICU.

# Discussion

In an explosion, there is rapid chemical conversion of solid or liquid into highly pressurized gas that compresses the surrounding air. This generates a pressure pulse which spreads as a blast wave in all directions.

Severity of blast injuries and case fatality is directly related to the type of explosive used and inversely related to the square of the distance from the detonation. Explosives are classified as; "high order" or "low order" explosives.<sup>[3]</sup> In our case nitrate based explosive with traces of pentaerythritol trinitrate was used which is an Research Department explosive (RDX) category of high order explosive.



Figure 1: Computed tomography brain showing right parietal hematoma, fracture temporal bone and multiple metallic foreign bodies in scalp

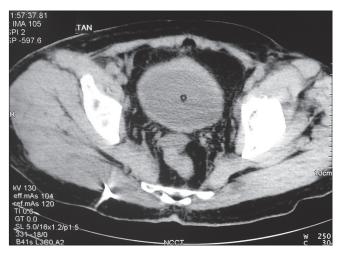


Figure 2: Computed tomography abdomen. Shrapnel in right gluteal region



Figure 3: Computed tomography chest: Persistent Pneumothorax "Fallen Lung Sign" Lung falling away from the hilum

Four categories of blast injuries have been described [Table 1].<sup>[4]</sup> Quinary blast injury pattern (fifth blast injury mechanism) is a hyper inflammatory state manifested as hyperpyrexia, sweating, low central venous pressure and positive fluid balance. It is unrelated to the severity of trauma and secondary to unconventional materials used in the manufacture of an explosive.<sup>[5]</sup>

Role of free radical-mediated oxidative stress in pathogenesis of blast-induced injury is supported by a rat lung model in which blast overpressure caused depletion of pulmonary antioxidant reserves and individual antioxidants (ascorbate, Vitamin E, glutathione) and accumulation of lipid peroxidation products.<sup>[6-8]</sup>

The first victim of our case series died of multiple organ failures; thrombocytopenia and coagulopathy developed on day 3. Head trauma predisposes to the development of coagulopathies. Cause of shock at time of admission can be attributed to combined blood loss from hemothorax and multiple long bone fractures. Source of sepsis in this patient was the ileal perforation. Septic shock, ARDS, renal function impairment and cerebral edema were the main contributors of mortality. Manifestations of peritonitis are usually missed in semiconscious or unconscious patients. This leads to a delay in surgery and hence increase in morbidity. ARDS usually develops within 24-48 h of blast injury.

Persistent right sided pneumothorax, infected amputated stumps, bacteremia, deranged renal function tests, intermittent decrease in platelet counts and septic shock complicated the ICU stay of the second victim in our ICU. High dose of antibiotics based on culture/sensitivity reports, aggressive wound management of amputated limb and strict asepsis could not control the bacteremia.

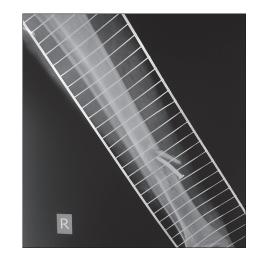


Figure 4: Shrapnel in situ

Outcome was good for the remaining two patients who did not have multi system involvement. Literature is sparse on the ICU management of such patients. Kamauzaman *et al.*<sup>[9]</sup> has reported mortality in a victim of hand grenade blast who was aggressively managed in their ICU for 1 weeks. Lung contusion, lung collapse, sepsis and diabetes insipidus had contributed to his mortality.

Hirshberg *et al.*<sup>[10]</sup> has reported normal lung function tests and complete resolution of the chest radiograph findings in 11 victims of a bus explosion with blast lung injury (BLI) after a follow-up period of 1-year.

Blast lungw is recognized in a victim when respiratory distress is present with either a butterfly appearance or a pneumothorax in CXR. Outcome of fifteen patients with primary BLI resulting from explosions on two civilian buses has been analyzed by Pizov *et al.*<sup>[11]</sup> BLI severity score has been proposed [Table 2].

# Management of patients with blast lung injury

- Supplemental high flow oxygen via noninvasive continuous positive airway pressure or endotracheal tube
- Avoidance of high inspiratory pressure and volume; (upper limit of inspiratory pressures <40 cmH<sub>2</sub>O)
- One lung ventilation in severe hemoptysis or significant air leak (Broncho pleural fistula)
- Chest tubes for pneumothorax and hemothorax
- Hyperbaric oxygen therapy for patients with arterial gas embolism
- Permissive hypercapnia, high-frequency jet ventilation and extracorpeal membrane oxygenation for patients with ARDS

Category	Organs affected
Primary	Gas filled structures
Unique to high explosives,	Lungs
results from the impact of the	Pulmonary contusion
over-pressurization wave with body surfaces	Hemoptysis
	Hemothorax
	Pneumothorax
	Pulmonary pseudocyst
	Age
	Gastrointestinal tract: Abdominal
	hemorrhage and perforation
	Middle ear: TM rupture and middle
	ear damage
	Globe (eye) rupture
	Concussion (traumatic brain injury
	without physical signs of head injury)
Secondary	Any body part may be affected
Results from flying debris and bomb fragments	Trauma to the head, neck, chest,
	abdomen, and extremities in the
	form of penetrating and blunt trauma
	Fractures
	Traumatic amputations
	Soft tissue injuries
Tertiary	Any body part may be affected
Results from individuals being	Fracture and traumatic amputation
thrown by the blast wind	Closed and open brain injury
Quaternary	Any body part may be affected
All explosion-related injuries,	Burns (flash, partial, and full thickness)
illnesses, or diseases not due to	burns (hash, partial, and full thickness)
primary, secondary, or tertiary	
mechanisms	
Includes exacerbation or	Crush injuries
complications of existing	Closed and open brain injury
conditions of existing	Asthma, chronic obstructive pulmonary
	disease, or other breathing problems
	from dust, smoke, or toxic fumes
	, ,
	Angina, hypertension
	Hyperglycemia

,
---

AGE: Arterial gas embolism; TM: Tympanic membrane

Table 2: BLI severity score				
	Severe BLI	Moderate BLI	Mild BLI	
PaO <sub>2</sub> /FiO <sub>2</sub>	<60	60-200	>200	
CXŔ	Massive bilateral infilterates	Bilateral or unilateral infilterates	Localized lung infilterates	
Bronchial pleural fistula	Yes	Yes/no	No	

Mortality with severe BLI was reported to be 75%. BLI: Blast lung injury; CXR: Chest X-ray

 Pneumomediastinum, persistent pneumothorax, subcutaneous emphysema, fallen lung sign (collapse of the lung away from hilum) should raise suspicion of tracheobronchial injury.

Blast injuries lead to severe sepsis/systemic inflammatory response, multiple organ dysfunctions and prolonged critical illness. Recent studies have shown improved outcome with activated protein C, steroid replacement, aggressive control of blood glucose, optimal antimicrobial cover and selective digestive decontamination.<sup>[12]</sup>

Combined crush/blast injury can cause hyperkalemic cardiac arrest which is amplified if patient develops renal failure secondary to myoglobinuria.

Thawed plasma, fresh whole blood, cryoprecipitate and recombinant Factor VIIa is administered to avoid dilution of clotting factor (<50%). Fluid resuscitation should be based on urine output and central venous pressure to ensure tissue perfusion without volume overload.

Damage control surgery (DCS) is indicated when a person sustains an injury of such severity that it impairs their ability to maintain homeostasis. Severe hemorrhage then leads to triad of metabolic acidosis, hypothermia, and increased coagulopathy. This form of surgery puts more emphasis on preventing the above-mentioned trauma triad of death, rather than correcting the anatomy. A major component of the surgery is early recognition of a person who could benefit from it and thus patients are transported to the operating room upon arrival, and resuscitation ensues concurrently with surgery. Goals of DCS are to stop the bleeding, remove contaminants and leave the wound open to avoid abdominal compartment syndrome.

The person is then sent to an ICU for the second phase in which the patient is given a combination of various medications and treatments to help restore a physiologic balance, especially with regards to their temperature, oxygenation, and pH level. Passive rewarming reverses most of the ill effects of the trauma triad. This phase generally lasts no longer than 2 days but varies from patient to patient. If a person's condition fails to improve within the first 24 h, it could mean missed hemorrhage and the need for immediate surgery. Definitive surgical procedures are generally performed in the third phase after patient stabilisation.

Trimodal distribution of death occurs after a blast injury. Very severely injured die on the spot; not much can be done for them. Secondary peak is reduced by managing airway, breathing and circulation. Third peak that occurs days to weeks later and is due to sepsis and multi-organ failure and was the cause of death in our patients. Associated blunt, penetrating and thermal injuries further increase morbidity and mortality of blast victims further research is needed to devise management protocols for blast victims in ICU to minimise physiological dysfunction caused due to multi-organ failure.

## References

 Dellinger RP, Levy MM, Carlet JM, Bion J, Parker MM, Jaeschke R, et al. Surviving sepsis campaign: International guidelines for management of severe sepsis and septic shock: 2008. Crit Care Med 2008;36:296-327.

- Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. The Acute Respiratory Distress Syndrome Network. N Engl J Med 2000;342:1301-8.
- Center for Disease Control and Prevention (CDC). Explosions and blast injuries: A primer for elinicians. Available from: http://www.bt.cdc.gov/ masscasualties/explosions.asp. [Last accessed on 2012 May 11].
- Mellor SG. The pathogenesis of blast injury and its management. Br J Hosp Med 1988;39:536-9.
- Kluger Y, Nimrod A, Biderman P, Mayo A, Sorkin P. The quinary pattern of blast injury. Am J Disaster Med 2007;2:21-5.
- 6. Elsayed NM. Toxicology of blast overpressure. Toxicology 1997;121:1-15.
- Gorbunov NV, Elsayed NM, Kisin ER, Kozlov AV, Kagan VE. Air blast-induced pulmonary oxidative stress: Interplay among hemoglobin, antioxidants, and lipid peroxidation. Am J Physiol 1997;272:L320-34.
- 8. Elsayed NM, Tyurina YY, Tyurin VA, Menshikova EV, Kisin ER,

Kagan VE. Antioxidant depletion, lipid peroxidation, and impairment of calcium transport induced by air-blast overpressure in rat lungs. Exp Lung Res 1996;22:179-200.

- Kamauzaman TH, Ahmad R, Latif KA, Hamzah MS, Kheng CP. Hand grenade blast injuries: An experience in hospital universiti sains Malaysia. Malays J Med Sci 2007;14:58-61.
- Hirshberg B, Oppenheim-Eden A, Pizov R, Sklair-Levi M, Rivkin A, Bardach E, *et al.* Recovery from blast lung injury: One-year follow-up. Chest 1999;116:1683-8.
- Pizov R, Oppenheim-Eden A, Matot I, Weiss YG, Eidelman LA, Rivkind AI, *et al.* Blast lung injury from an explosion on a civilian bus. Chest 1999;115:165-72.
- Lavery GG, Lowry KG. Management of blast injuries and shock lung. Curr Opin Anaesthesiol 2004;17:151-7.

How to cite this article: Samra T, Pawar M, Kaur J. Challenges in management of blast injuries in Intensive! Care Unit: Case series and review. Indian J Crit Care Med 2014;18:814-8.

Source of Support: Nil, Conflict of Interest: None declared.

Announcement

#### iPhone App



A free application to browse and search the journal's content is now available for iPhone/iPad. The application provides "Table of Contents" of the latest issues, which are stored on the device for future offline browsing. Internet connection is required to access the back issues and search facility. The application is Compatible with iPhone, iPod touch, and iPad and Requires iOS 3.1 or later. The application can be downloaded from http://itunes.apple.com/us/app/medknow-journals/ id458064375?ls=1&mt=8. For suggestions and comments do write back to us.