

Blast-induced injury of air-containing organs

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ALTHOUGH TERRORISM has been present for years, the number of serious terror incidents more than tripled in recent years. Access to information on the Internet has facilitated tremendous technological advancement in terrorists' destructive capabilities. This underscores the importance of appropriate education within the medical system and the need for careful advanced planning to cope with terror attacks.

Terrorists use various instruments, including explosives and chemical or biological weapons. Because of the controls on access to stockpiles of nuclear, biological and chemical weapons, conventional explosives are the most common tools for terrorist attacks. The number of lives lost, the number of people injured, and the damage to infrastructure resulting from bombings are orders of magnitude higher than those caused by chemical or biological incidents. This contradicts the common perception that unconventional weapons are more dangerous than explosives.

Because of the increasing risk of terrorist attacks, physicians must become familiar with the characteristics of contemporary explosive devices and the spectrum of injuries inflicted by blasts and explosions.^{1,2}

The mechanics of explosions

The damage caused by explosions is mediated primarily through air pressure changes, which follow complex

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Abstract

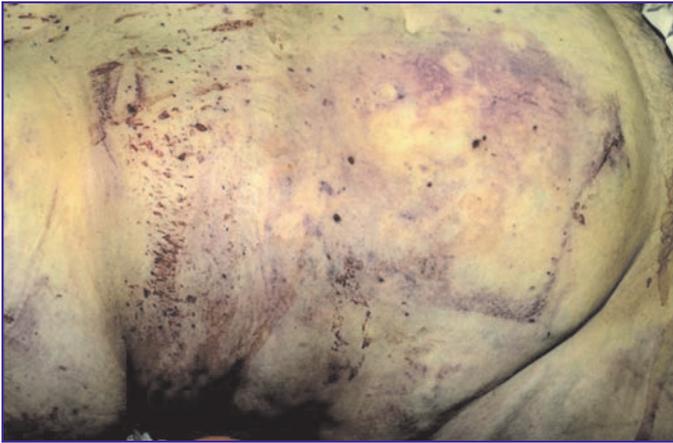
- ◆ Explosions produce blast injury through four different mechanisms: primary, the direct result of pressure; secondary, the result of projectiles; tertiary, resulting from high speed winds; and quaternary, mainly burns and asphyxia.
- ◆ Air-containing organs (the ear, lungs and digestive tract) are the first injured by the primary blast mechanism.
- ◆ Tympanic membrane injury is most common. However, it is not a reliable indicator of the blast exposure.
- ◆ Pulmonary blast injury is caused by the pressure differential across the alveolar–capillary interface, and presents a combination of haemorrhage, pulmonary contusion, pneumothorax, haemothorax, pneumomediastinum, and subcutaneous emphysema. Most of its survivors have excellent long-term outcomes.
- ◆ The large intestine is the most commonly injured part of the digestive tract. Not all sections of blast-contused bowel require resection and anastomosis.
- ◆ The complexity and delayed nature of some blast injuries requires a high index of suspicion for early diagnosis and successful treatment of casualties.

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nonlinear physics. The initial shock wave, called the positive pressure wave or the blast wave, comprises an almost instantaneous rise of air pressure emanating from the blast. A wave of negative pressure immediately follows, as air rushes in to fill the void caused by the initial pressure wave. In general, damage produced by blast waves decreases exponentially with distance from source.

When an explosion occurs indoors, standing waves and enhanced pressure differentials occur because of the additive effects of reflections or reverberations of the waves from walls and other rigid objects. Explosions in confined spaces are associated with very high mortality.^{2,3}

Explosives are categorised as low-grade or high-grade, differentiated by the speed of their combustion. Low-grade explosives burn rapidly. Examples are black powder, smokeless gunpowder, and rocket propellants. Nitro-starch, nitrocellulose and fireworks are also low-grade explosives. High-grade explosives, on the other hand, combust nearly instantaneously. Although more stable than low-grade explosives, they detonate on exposure to shock or similar impulses. The archetypes of high-grade explosives are nitroglycerine, ammonium nitrate and trinitrotoluene (TNT). Other high-grade explosives include



The effect of the blast wave on the abdominal wall. Notice the skin abrasion and subcutaneous haemorrhage.

Amatol 80/20, cyclotrimethylenetrinitramine (also known as RDX), pentaerythritol tetranitrate (PETN), and dynamite. Lead styphnate and lead azide are members of a separate class of very sensitive high-grade explosives.

Types of blast injury

The effects of blasts can be categorised as primary, the direct result of pressure; secondary, the result of projectiles; tertiary, resulting from high speed winds; and quaternary, mainly burns and asphyxia. Recently, a quinary blast effect from exposure to toxic substances has been described.

The types of injuries caused by a blast depend on whether the blast occurs in open air or within a confined space. Injuries can be aggravated by the collapse of buildings and structures, increasing wounding and mortality.

Primary blast injuries

Primary blast injury is caused by barotrauma, and it mostly involves air-filled organs and air-liquid interfaces. Such organs are damaged by dynamic pressure changes at tissue-density interfaces (eg, air-fluid). Rupture of the tympanic membranes, pulmonary damage and air embolism, and rupture of hollow viscera are the most important forms of primary blast injury.^{4,5}

Secondary blast injuries

Bombs may contain metallic particles such as nails, bolts or other projectiles. These and the disintegrated casing are designed to increase the wounding potential of the bomb. Such fragments are sometimes incorrectly called shrapnel after Henry Shrapnel, who developed specific military

antipersonnel munitions based on the same principle. The secondary blast effect of penetrating injuries is the leading cause of death and injury from bombs.

Tertiary blast injuries

The blast wind, which follows the wave of high pressure, causes tertiary blast injuries. These occur when the body strikes fixed objects. Any body part may be affected, and fractures, traumatic amputations, and open and closed brain injuries occur. This wind can also cause structural collapse and fragmentation of buildings and vehicles, and increases mortality through crush injuries and entrapment. Structural collapse and large airborne fragments lead to crush injuries and extensive blunt trauma.

The crush syndrome in victims of structural collapse reflects the metabolic derangement resulting from damage to muscle tissues and subsequent rhabdomyolysis and release of myoglobin and its nephrotoxic metabolites.

Compartment syndrome is also common. This results from the compression that a damaged oedematous muscle exerts within its inelastic sheath. Such confined swelling promotes local ischaemia, which initiates a positive feedback mechanism of swelling, increased compartment pressures, decreased tissue perfusion, and further ischaemia. Although compartment syndrome usually involves extremities, it may occasionally involve the buttocks and abdominal musculature such as the rectus sheath.^{6,7}

Quaternary blast injuries

Quaternary injuries include burns (chemical or thermal) and exposure to radiation. Conventional explosives generally do not cause primary fires because most of the available oxygen is exhausted during the explosion, but fires are sometimes encountered. Heat burns and other quaternary injuries predominated in victims of the Pentagon attack on 11 September 2001, in which terrorists crashed a fuel-laden aircraft. Incendiary bombs, like those used for bombing cities during World War II, can cause widespread fires and destruction. Napalm, used in incendiary bombs, consists of powdered aluminium soap or similar compounds that gelatinise or thicken oil and fuel, and so increase adherence and burning time. Nuclear explosions also produce considerable primary thermal effects.

After incendiary bomb attacks, the number of burn injuries should be ascertained as early as possible and alternative national burn management resources should be alerted, because a large number of burn victims can quickly overwhelm local medical resources.

Quinary blast injuries

In patients involved in a terrorist attack in the city of Tel Aviv, we noticed a unique early hyperinflammatory state

that did not correlate with the complexity of the sustained injury. It was postulated that this unique haemodynamic behaviour indicated the presence of toxic substances absorbed through wounds or by inhalation. This early hyperinflammation was not present in injured victims distant from the centre of the explosion or among patients who had no skin injuries. Toxic substances embedded in the bomb and metabolites of the explosive substance could explain this unique clinical observation.⁸



Eardrum perforation induced by blast.

Blast injury of air-containing organs

Because air is easily compressible and water is not, gas-containing organs, especially the middle ear, lungs, and digestive tract, are most susceptible to blast injury. The tympanic membrane is injured most frequently, followed by the lung. Viscera (stomach, intestine and colon) are less frequently involved. Organs such as the eyes, limbs and the central nervous system may also be injured, but are beyond the scope of this review, which focuses on typical blast injuries to the ear, lung and digestive tract.

The tympanic membrane and the inner ear

The tympanic membrane is the structure most frequently injured by blasts. This happens even at low pressure, and increased pressures as small as 5 psi (34.5 kPa) can cause tympanic membrane trauma.⁹

Temporary neurapraxia in the ear's receptor organs, manifested by deafness, tinnitus, and vertigo, characterises eardrum rupture. If dynamic overpressures are high enough, the ossicles of the middle ear can be dislocated.^{9,10}

Rupture of the tympanic membrane does not require specific treatment or hospitalisation. Patients should be instructed not to put anything in the affected ear and should be referred to ear, nose and throat specialists for follow-up care. Neomycin (a component of many otic solutions and suspensions) is ototoxic and is contraindicated in cases of tympanic membrane perforation.

Most tympanic membrane perforations heal spontaneously,¹¹ but complications such as ossicle disruption, cholesteatoma formation, and development of perilymphatic fistulae are possible. About a third of patients with tympanic membrane perforations have permanent hearing loss, mainly due to inner ear injury.¹²

Eardrum injury was considered a predictor of other blast injuries, but a recent Israeli series of 647 civilian victims of terrorist bombings has contradicted this traditional belief. Of 142 patients in that study who were initially diagnosed with isolated eardrum perforation and who were well enough to be discharged, none developed later manifesta-

tions of other blast injuries. Furthermore, 18 patients with pulmonary blast injuries had no eardrum perforation. Thus, isolated eardrum perforation in survivors of explosions is not a reliable marker of concealed pulmonary blast injury or of poor prognosis.¹³ It has been postulated that cerumen-filled auditory canals and elastic tympanic membranes in younger patients are relatively resistant to blast, and therefore eardrum injury is not a sensitive indicator of blast exposure.

Blast lung injury

The lung is the organ second most susceptible to primary blast injury. Pressure differentials across the alveolar-capillary interface can cause disruption, haemorrhage, pulmonary contusion, pneumothorax, haemothorax, pneumomediastinum, and subcutaneous emphysema.¹⁴

The mechanics involved in blast-induced lung injury are complex. The high-velocity pressure waves that propagate through the body cause pressure differentials at the interfaces of tissues of different densities. These pressure differences lead to the rupture of alveolar septa and capillary walls. The resulting diffuse interstitial haemorrhage around vessels within the lung parenchyma is properly regarded as potentially life-threatening intrapulmonary bleeding. In rare cases, immediate surgery may be required for haemorrhage control.¹⁵ These haemorrhages may result either from pressure differentials (a primary blast injury) or from shearing forces applied to the lung parenchyma when it is decelerated against the chest wall (a tertiary blast injury).

At the biochemical level, changes such as depletion of pulmonary antioxidant reserves result in free-radical-mediated oxidative stress. The accumulation of lipid peroxidation products contributes to the blast-induced lung injury, and ruptured erythrocytes release haemoglobin, further amplifying the oxidative stress.¹⁶

The pathological features of human blast lung injury were established by an autopsy study.¹⁷ They were characterised by two main mechanisms: mechanical overdistension of alveoli, and diffuse microscopic haemorrhages. Alveolar ruptures typify the first mechanism, with thinning of alveolar septa and enlargement of alveolar spaces. Haemorrhage, the second mechanism, may be observed in the subpleural, intra-alveolar, and perivascular (cuff-like) areas. A direct correlation was found between proximity to the centre of detonation and the severity of these pathological manifestations.¹⁷

Air embolism is a well-recognised consequence of blast lung injury. It is one of the major factors leading to cardiac dysfunction and immediate death after blast wave exposure.¹⁸ Whether air embolism is caused by mechanical

ventilation of blast victims is still debated, but in an autopsy study where immediate casualties were examined, evidence for air embolism was found in almost 50%, even though none had been mechanically ventilated.¹⁷ It was proposed that disruption of the alveolar septa and the interstitial vessel walls, allowing air into the adjacent pulmonary veins, is the mechanism for this pathology.

Pulmonary fat embolism is a finding of clinical importance in survivors of blast trauma, because it can lead to the development of acute respiratory distress syndrome (ARDS), and significantly affects clinical outcomes.¹⁹

Blast-induced pulmonary injuries are life-threatening, with immediate onset of pulmonary oedema carrying a grave prognosis. Pulmonary barotrauma is the most common fatal primary blast injury, and the most common critical injury in people close to the blast centre. ARDS may result from direct lung injury or from shock due to other injuries.

Pulmonary blast injury produces a unique cardiovascular response of decreased heart rate, stroke volume and cardiac index, with failure of the normal reflex increase in systemic vascular resistance. This cardiovascular derangement occurs within seconds,²⁰ and can cause death even in the absence of any demonstrable physical injury.²⁰ If not fatal, recovery usually occurs in 15 minutes to 3 hours; however, impaired pulmonary performance can last for hours or days.

Acute gas embolism as a form of pulmonary barotrauma most commonly occludes blood vessels in the coronary system, brain or spinal cord. The resulting neurological symptoms must be differentiated from the direct effects of possible brain trauma.²¹

Signs of blast lung, characterised by the clinical triad of apnoea, bradycardia, and hypotension, are usually present at the time of initial evaluation, but they have been reported as developing as late as 48 hours after an explosion. Blast lung should be suspected in any casualty with dyspnoea, cough, haemoptysis, or chest pain following blast exposure.²²

In general, managing blast lung injury is similar to caring for pulmonary contusion, which requires judicious fluid administration to ensure tissue perfusion without volume overload. All patients with blast lung injury should receive supplemental high flow oxygen sufficient to prevent hypoxaemia. Impending airway compromise, secondary oedema, injury, or massive haemoptysis requires immediate intervention to secure the airway. Patients with massive haemoptysis or significant air leaks may benefit from selective bronchial intubation. Clinical evidence or suspicion of haemothorax or pneumothorax warrants prompt decompression. If ventilatory failure is imminent or has occurred, patients should be intubated. In pulmonary blast injury, positive pressure ventilation and positive end expiratory pressures should be avoided because of the risk

of pulmonary alveolar rupture and subsequent formation of air emboli.

However, mechanical ventilation often cannot be avoided. Because of the heterogeneous pulmonary compliance that characterises the blast lung, localised over-inflation of the more compliant lung segments occurs with high ventilatory pressures. Whenever possible, the tidal volume should be reduced to limit the peak inspiratory pressure and to minimise further, ventilator-induced, lung barotrauma injury. If necessary, permissive hypercapnoea ventilation should be considered. In cases of air embolism, high flow oxygen should be administered and the patient should be placed in a prone, semi-left lateral, or left lateral position. Patients with air emboli should be transferred to a hyperbaric chamber.

There are no definitive guidelines for observation, admission, or discharge of patients with possible blast lung injury. Patients so diagnosed may require complex management and should be admitted to an intensive care unit. Asymptomatic patients with findings merely suggesting this complex injury should be observed elsewhere in hospital. In general, patients with normal chest radiographs and arterial blood gases, and without complaints that suggest blast lung injury, may be considered for discharge after 4–6 hours of observation.²² Data on the short- and long-term outcomes of patients with blast lung injury are limited. However, in a study conducted on survivors 1 year after injury, none had pulmonary complaints, all had normal physical examinations and chest radiographs, and most had normal lung function tests.²³

The digestive tract

Gas-containing sections of the gastrointestinal tract are most vulnerable to primary blast effect. Gastrointestinal tract blast injuries are rare compared with injury to ears and lung; up to 1.2% of patients exposed to bomb explosions will suffer gastrointestinal tract blast injury.²⁴

This injury can cause immediate bowel perforation, haemorrhage ranging from small petechiae to large haematomas, mesenteric tear and ischaemia. Solid organs are rarely affected. Blast abdominal injury should be suspected in anyone exposed to an explosion who then develops abdominal pain, nausea, vomiting, haematemesis, rectal pain, tenesmus, testicular pain, unexplained hypovolaemia, or any other findings suggestive of an acute abdomen. Clinical findings may be absent until the onset of complications.

The colon is the gastrointestinal structure most frequently affected by primary blast injury, as it contains a significant amount of air. Less frequently, small intestine injury is an immediate result of a blast. Delayed rupture or perforation can result from mesenteric ischemia or infarct. These injuries are difficult to detect initially.²⁵

The question of bowel contusion management is still controversial. In most cases, bowel contusion is regarded as a dynamic injury that can lead to perforation of the damaged organ. Previous studies have suggested that small bowel and colonic contusions larger than 10 mm in diameter are at high risk for delayed perforation. Accordingly, bowel resection, with or without anastomosis, was performed in all cases. Recently, a pig blast injury model has shown that, following identification at laparotomy, the number of small bowel contusions requiring excision could be reduced from 86% to 60% with conservative management of small bowel contusions smaller than 15 mm in diameter. Similarly, excision of colonic contusions could be reduced from 73% to 27% if colonic contusions of less than 20 mm were managed conservatively.²⁶

Summary

Conventional bomb explosions are likely to remain the primary tool of terrorists, and the medical consequences are likely to increase in incidence and distribution. Knowledge of the mechanisms of blast injury, the associated pathophysiology, the clinical diagnosis and recommended management of the resulting complex trauma are the keys for helping casualties. The air-containing organs are extremely vulnerable to the unique primary blast effect of bombs. Familiarity with the effects that explosions exert on these organs, their consequences and treatment, together with a high index of suspicion, are crucial for medical professionals to obtain the best outcome for terrorists' victims.

Competing interests

None identified.

References

1. DePalma RG, Burris DG, Champion HR, Hodgson MJ. Blast injuries. *N Engl J Med* 2005; 352: 1335-1342.
2. Leibovici D, Gofrit ON, Stein M, et al. Blast injuries: bus versus open-air bombings — a comparative study of injuries in survivors of open-air versus confined-space explosions. *J Trauma* 1996; 41: 1030-1035.
3. Kluger Y, Peleg K, Daniel-Aharonson L, et al. The special injury pattern in terrorist bombings. *J Am Coll Surg* 2004; 199: 875-879.
4. Guy RJ, Glover MA, Cripps NP. The pathophysiology of primary blast injury and its implications for treatment. Part I: the thorax. *J R Nav Med Serv* 1998; 84: 79-86.

5. Cripps NP, Glover MA, Guy RJ. The pathophysiology of primary blast injury and its implications for treatment. Part II: the auditory structures and abdomen. *J R Nav Med Serv* 1999; 85: 13-24.
6. O'Mara MS, Semins H, Hathaway D, Caushaj PF. Abdominal compartment syndrome as a consequence of rectus sheath hematoma. *Am Surg* 2003; 69: 975-977.
7. Brumback RJ. Traumatic rupture of the superior gluteal artery, without fracture of the pelvis, causing compartment syndrome of the buttock. A case report. *J Bone Joint Surg Am* 1990; 72: 134-137.
8. Kluger Y, Nimrod A, Biderman P, et al. The quinary pattern of blast injury. *J Emerg Manage*. In press.
9. Patterson JH Jr, Hamernik RP. Blast overpressure induced structural and functional changes in the auditory system. *Toxicology* 1997; 121: 29-40.
10. Deguine C, Pulec JL. Traumatic dislocation of the incus. *Ear Nose Throat J* 1995; 74: 800.
11. Sprem N, Branica S, Dawidowsky K. Tympanoplasty after war blast lesions of the eardrum: retrospective study. *Croat Med J* 2001; 42: 642-645.
12. Mrena R, Paakkonen R, Back L, et al. Otologic consequences of blast exposure: a Finnish case study of a shopping mall bomb explosion. *Acta Otolaryngol* 2004; 124: 946-952.
13. Leibovici D, Gofrit ON, Shapira SC. Eardrum perforation in explosion survivors: is it a marker of pulmonary blast injury? *Ann Emerg Med* 1999; 34: 168-172.
14. Mellor SG, Cooper GJ. Analysis of 828 servicemen killed or injured by explosion in Northern Ireland 1970-84: the Hostile Action Casualty System. *Br J Surg* 1989; 76: 1006-1010.
15. Levinsky L, Vidne B, Nudelman I, et al. Thoracic injuries in the Yom Kippur war. Experience in a base hospital. *Isr J Med Sci* 1975; 11: 275-280.
16. Elsayed NM, Gorbunov NV, Kagan VE. A proposed biochemical mechanism involving hemoglobin for blast overpressure-induced injury. *Toxicology* 1997; 121: 81-90.
17. Tsokos M, Paulsen F, Petri S, et al. Histologic, immunohistochemical, and ultrastructural findings in human blast lung injury. *Am J Respir Crit Care Med* 2003; 168: 549-555.
18. Argyros GJ. Management of primary blast injury. *Toxicology* 1997; 121: 105-115.
19. Elmaraghy AW, Aksenov S, Byrick RJ, et al. Pathophysiological effect of fat embolism in a canine model of pulmonary contusion. *J Bone Joint Surg Am* 1999; 81: 1155-1164.
20. Irwin RJ, Lerner MR, Bealer JF, et al. Cardiopulmonary physiology of primary blast injury. *J Trauma* 1997; 43: 650-655.
21. Dedouit F, Otal P, Loubes-Lacroix F, et al. Post-traumatic venous and systemic air embolism associated with spinal epidural emphysema: Multi-slice computed tomography diagnosis. *Forensic Sci Int* 2005; doi:10.1016/j.forsciint.2005.07.012.
22. Avidan V, Hersch M, Armon Y, et al. Blast lung injury: clinical manifestations, treatment, and outcome. *Am J Surg* 2005; 190: 927-931.
23. Hirshberg B, Oppenheim-Eden A, Pizov R, et al. Recovery from blast lung injury: one-year follow-up. *Chest* 1999; 116: 1683-1688.
24. Paran H, Neufeld D, Shwartz I, et al. Perforation of the terminal ileum induced by blast injury: delayed diagnosis or delayed perforation? *J Trauma* 1996; 40: 472-475.
25. Tatic V, Ignjatovic D, Jevtic M, et al. Morphologic characteristics of primary nonperforative intestinal blast injuries in rats and their evolution to secondary perforations. *J Trauma* 1996; 40 (3 Suppl): S94-S99.
26. Cripps NP, Cooper GJ. Risk of late perforation in intestinal contusions caused by explosive blast. *Br J Surg* 1997; 84: 1298-1303.

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