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The quinary pattern of blast injury

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ABSTRACT

Objective: *Bombing is the primary weapon of global terrorism, and it results in a complicated, multidimensional injury pattern. It induces bodily injuries through the well-documented primary, secondary, tertiary, and quaternary mechanisms of blast. Their effects dictate special medical concern and timely implementation of diagnostic and management strategies. Our objective is to report on new clinical observations of patients admitted to the Tel Aviv Medical Center following a recent terrorist bombing.*

Results: *The explosion injured 27 patients and three died. Four survivors, who had been in close proximity to the explosion as indicated by their eardrum perforation and additional blast injuries, were exposed to the blast wave. They exhibited a unique and immediate hyperinflammatory state, two upon admission to the intensive care unit and two during surgery. This hyperinflammatory state was manifested by hyperpyrexia, sweating, low central venous pressure, and positive fluid balance. This state did not correlate with the complexity of injuries sustained by any of the 67 patients admitted to the intensive care unit after previous bombings.*

Conclusion: *The patients' hyperinflammatory behavior, unrelated to their injury complex and severity of trauma, indicates a new injury pattern in explosions, termed the quinary blast injury pattern. Unconventional materials used in the manufacture of the explosive can partly explain the observed early hyperinflammatory state. Medical personnel caring for blast victims should be aware of this new type of bombing injury.*

Key words: terrorism, blast injury, hyperinflammation

INTRODUCTION

Bombings and explosions are the hideous tool of terrorism. Their simple manufacture and easy concealment ensure that they will remain the main weapon of terrorists. Using suicide bombers, terror groups can reach and explode within crowded areas filled with unprotected civilians. In his Presidential Address in September 2001, President Bush stated that war on terror would not cease until every terrorist group of global reach was found, stopped, and defeated. But since then the number of terrorist bombing attacks has steadily increased, and during 2003 it has reached the level of global epidemic.

In past years at the Rabin Trauma Center, we have experienced the aftermath of a long series of terror attacks. The most complicated trauma victims are those admitted after bombing. Explosions involve more bodily regions than other kinds of trauma and affect the release of greater quantities of cellular mediators.[1] Therefore, increased in-hospital mortality is encountered in these events (6 percent vs. 2 percent for conventional trauma).[2]

Mellor explored the causes of death among victims of bomb explosions and found that 14 percent had disrupted bodies, 39 percent suffered multiple injuries, 11 percent chest injuries alone, 12 percent head injuries alone, and 21 percent combined head

and chest injuries.[3] From this important work it was concluded that the main factor affecting mortality in an explosion is the proximity to the center of detonation. Bodies of victims who had suffered only minor external injuries imparted greater knowledge of the devastating effects of the blast.[4]

The explosive material is composed of trinitrotoluene (TNT) or other explosives from military, commercial, or homemade sources. Detonation is triggered by an electrical current, and the solid explosive is transformed into gas to create a very high-pressure wave of air that emanates radially at the speed of sound. This creates a peak of over-pressure, a shock-front that is only 3 to 5 mm thick, and is followed by the blast wind. For example, 25 kg of TNT produces a peak over-pressure of 150 psi (10.5 kg/cm^2). These pressures last 2 ms and traverse at a speed of 3,000 to 8,000 m/sec. Greater explosives produce longer duration of the front shock wave and more damage results.

There are four injury patterns related to the blast. The primary blast injuries occur from the blast wave passing through the victim. The human body is remarkably resistant to the blast wave, and the tissues will respond according to their composition, with air-containing organs suffering the most. Three putative mechanisms, spalling, implosion, and acceleration and deceleration are associated with the primary blast injury. Spalling occurs as the blast wave passes through organs filled with gas and fluid and generates high velocity bubbles that hit the wall of the organ and damage it. Implosion originates from the blast passing through air-filled organs, initially compressing its air-filled spaces and then inflating them rapidly, damaging the surrounding tissue. Acceleration and deceleration are similar to what is seen in conventional trauma. The hallmarks of the primary blast injuries are eardrum perforation and blast lung injuries. Perforations of hollow abdominal viscera are rare (≥ 1.2 percent). Solid organs are rarely damaged by the blast itself, and acceleration and deceleration mechanisms are the true cause of injury to these organs.

The secondary blast injury results from the casings, debris, and particles added to the bomb that are energized by the wave of air. Metal particles are usually added to the charge to increase the wounding potential. Steel balls, nails, and other sharp particles are the terrorists' favorites. The velocity of these missiles depends on their shape and distance of flight, and they are influenced by yaw and drag, just like classic missiles. Peppering of the skin is the distinctive sign of this ballistic effect. Multiple penetrations of the skin are common and often represent only the tip of the iceberg.

The tertiary blast injury is the result of overpressure. It is responsible for total body destruction in some patients and, for others, can cause amputation of limbs. The blast wind alone can accelerate the body and thrust it against stationary objects.

The quaternary blast injury is related to the thermal effects of the blast. Burns are caused by ignition of flammable materials. Thermal lung injury can develop directly from the very high air temperatures created at the site of the explosion.

Following we report on some unique clinical observations of patients admitted after a recent bombing. Their clinical picture may be attributed to a new, quinary pattern of blast injury.

CASE REPORTS

On April 30, 2003, a suicide bomber exploded in a Tel Aviv nightclub. Twenty-seven patients were evacuated to the Rabin Trauma Center; two of them were dead on arrival. Five patients were rushed to the operating theatre due to life-threatening injuries, where one of them succumbed to his injuries.

All the victims arrived at our hospital 20 to 30 minutes after the event. They were evaluated in the emergency department by trauma teams guided by the medical director and team leaders. Advanced Trauma Life Support (ATLS) protocols and the hospitals' mass casualty protocols were applied.

We present four cases of victims who were very near the center of the explosion (e.g., Patient 4 was one meter away from the suicide bomber). Two patients (Patients 2 and 3) were admitted to the intensive care unit (ICU) one hour after arrival. Patients 1 and 4 had surgical interventions and were admitted to the ICU two and four hours after admission, respectively. In the

Table 1. Clinical details of the patients											
Patient	**Age**	**Injury complex**	**HR (beats/ min)**	**BP (mm Hg)**	**T (°C)**	**Leu (per μl)**	**SVR (dynes · s · cm^{-5})**	**pO_2/F (iO_2)**	**Lung compliance (L · cm H_2O^{-1})**	**ISS**	**AIS**
1	27	Lung, colon, burn 7%	120	90	39	9,000		120	31	33	Lung-4 Skin-1 Colon-4
2	32	Burn 9%	130	110	39	8,700	400	242	28	9	Skin-3
3	24	Lung, burn 3.5%	170	138	39	8,300		138	40	10	Skin-1 Lung-3
4	38	Colon, amputation, burn 7%	125	90	39	11,700		233	43	29	Colon-4 Skin-2 Extremity-3

HR, heart rate; BP, blood pressure; T, temperature; Leu, Leukocytes; SVR, systemic vascular resistance; ISS, Injury Severity Score; AIS, Abbreviated Injury Scale.

immediate post-trauma phase, these four patients developed a unique clinical state that was manifested in increased heart rate, fever, and increased fluid demand, and was unrelated to the severity of their injuries (Table 1).

Patient 1

A healthy 27-year-old male was conscious on admission, hypoxemic and in hemodynamic shock. A left pneumothorax was relieved by tube thoracostomy, and fluid resuscitation was started. He also suffered from first and second degree burns of the face and chest (7 percent) and from bilateral eardrum perforation. A positive diagnostic peritoneal lavage (DPL) resulted in exploratory laparotomy. Colonic wall perforation led to ileocecectomy with anastomosis. No other injuries were found.

Upon termination of the surgical procedure, the patient was hypotensive with wide pulse pressure and signs of peripheral vasodilatation. Despite central venous pressure (CVP) guided aggressive fluid therapy, the patient remained hypotensive. A pulse index contour cardiac output (PICCO) monitor was introduced (Pulsion Medical System, Munich, Germany). The measured cardiac index was 6 lit/min, and the calculated systemic vascular resistance was low (400 mm Hg · dyne · min · lit -1). Vasopressor support with noradrenalin was started and the patient's blood pressure stabilized. No signs of bleeding were noted. Although fluid therapy was at room temperature, the patient's fever rose to 39°C. The white blood cell count was slightly elevated with moderate left shift. The hyperdynamic circulatory state resolved after 24 hours, the cardiac output and systemic vascular resistance normalized, and noradrenalin was withdrawn. All blood and sputum cultures were negative, and the patient's fever gradually decreased over the next four days.

Patient 2

A 32-year-old male was hemodynamically stable upon arrival with Glasgow Coma Score of 15. Physical examination revealed second degree burns of the face and hands, bilateral eardrum perforation, and carbonized blackening around the nostrils and mouth. Preventive intubation was preformed and the patient was admitted to the ICU. He was sedated with midazolam and fentanyl, and ventilated. At this stage, cardiorespiratory parameters, initial laboratory results, chest x-ray, and electrocardiogram were all normal. A few minutes after his admission he had a fever of 39.6°C, tachycardia (>150 beats/min). His blood pressure was 90/40 mm Hg, and wide range pulse pressure was recorded for several hours. No evidence for missed or obscured hemorrhage was found. CVP monitoring was introduced and fluid resuscitation

with crystalloids started. During the following 24 hours, despite aggressive CVP and urinary output-guided fluid resuscitation and a positive fluid balance of 3500 ml, the patient continued to exhibit hyperdynamic circulatory response and hyperpyrexia up to 40.6°C. This hyperdynamic state gradually resolved over the next 48 hours, although fever continued for five days following hospital admission. White blood cell count was normal with slight left shift, which improved within a few days. All blood cultures were negative, careful skin wound inspection revealed no active infection, and sputum cultures resulted in normal upper airway flora.

Patient 3

A 24-year-old male with previous history consistent with mild asthma was admitted with severe hypoxemia due to bilateral lung contusion. He was conscious and hemodynamically stable. Physical examination revealed left hemiparesis, bilateral eardrum perforation, and 3.5 percent second degree burns of the chest. Head computerized tomography demonstrated small subdural and pontine hematomas. On arrival to ICU he was intubated and ventilated. He was hemodynamically stable but hypoxemic, with PaO_2/FiO_2 of 140 and measured lung compliance of 28 L · cm H_2O -1. A few hours later the patient's temperature reached 39.4°C, and the fever continued for the next five days and resolved after three days. The white blood cell count was normal and blood, urine, and sputum cultures were negative.

Patient 4

A healthy 38-year-old male was conscious but hypotensive on admission. His right hand was mangled, and first and second degree burn injuries of the head and neck (7 percent) were noticed. The patient was intubated and underwent emergency exploratory laparotomy due to positive DPL. Laparotomy revealed intraperitoneal blood from a minor liver laceration and right colonic perforation that resulted with right colectomy. The right hand was amputated. Upon termination of the operation, the patient was hemodynamically unstable with wide pulse pressure and peripheral vasodilatation. His hemoglobin level was 10 g/dl. A positive fluid balance of 5500 ml of crystalloids was reached on the first ICU day. His temperature reached 39.4°C a few hours after admission and above 40.0°C on the second day. The hyperdynamic state gradually resolved in the next 72 hours, although the fever continued for seven days. The white blood cell count was normal. Blood cultures were negative and sputum cultures revealed normal flora.

DISCUSSION

Compared to its predecessors, this suicide bombing in a nightclub in the city of Tel Aviv was unique. The explosive belt carried by the suicide bomber was devoid of metal additives and consisted only of explosives. This resulted in pure blast energy emanating from the site of explosion, injuring people crowded in a radius of 20 m from the explosion center. The explosive was estimated to be 1.5 kg of "DETA-sheet." This material is composed of pentaerythritol tetranitrate (PETN), a highly unstable, easily detonative substance, equivalent to 140 percent TNT. It is used in a variety of military and civilian applications but was never used before in a suicide bombing in Israel.

In previous bombings, we have noticed increased mortality among those injured at close proximity to the explosion. This was magnified if the explosion occurred in confined spaces, and even more so in ultra-confined spaces such as buses.[2] The inclusion of metal projectiles embedded in the explosive further increased the injuring potential of the charge, resulting in increased mortality. In the present event described above, the charge was small, metal objects were absent, and the explosion occurred in an open space. Therefore, it resulted in greater patient survival and in lighter injuries at the center of detonation compared to what is usually seen. This enabled us to monitor patients who were in close proximity to the explosion during their immediate hospital stay. Such patients usually do not survive explosions or are so severely injured that their hemodynamic instability can be attributed to the complex injury.

In the patients described above, we have noticed a unique early hyperinflammatory state that did not correlate to the complexity of the sustained injury.

Putatively, this unique hemodynamic behavior may indicate the presence of toxic substances absorbed by the casualties through their injuries or via inhalation. This early hyperinflammatory manifestation was not present in those injured further from the explosion or among patients who suffered no skin injury. This could indicate that the toxic substances were very short-lived, and that their absorption was most probably through the disrupted skin, perhaps in a dose-dependent nature with dose related to the distance from the center of the explosion.

This was the first bombing incident in Israel that we have observed in which PETN was used. Upon its chemical modification for increased stability, the PETN becomes a potent vasodilator. Although we could not demonstrate abnormal levels of nitrates in the patients' sera, this could explain our observation of hyperinflammatory state even among lightly injured patients. Late awareness of the clinical entity resulted in delayed blood sampling and could, therefore, explain the normal serum nitrate levels. The need for special laboratory techniques for measurement of these metabolites also delayed the pursuit of the initiating mediator of hyperinflammation.

The hyperinflammatory state must be differentiated from missed injuries that may express with hemodynamic instability. The use of all investigative means necessary for this differentiation is recommended.

The hyperinflammatory behavior, unrelated to the patients' injury complex and trauma severity, constitutes a new injury pattern in bomb explosions that we term "the quinary blast injury pattern." The use of toxic materials in the manufacture of the explosive, or the use of uncommon explosives, could explain, to some extent, our unique clinical observation. Medical personnel caring for bombing victims should be aware of this pattern of injury and, when this pattern is detected, should direct management to reduce its complications.

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