Blast injuries to the lung: epidemiology and management

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Lung injury is frequently a component of the polytrauma sustained by military personnel surviving blast on the battlefield. This article describes a case series of the military casualties admitted to University Hospital Birmingham’s critical care services (role 4 facility), during the period 1 July 2008 to 15 January 2010. Of the 135 casualties admitted, 107 (79.2%) were injured by explosive devices. Plain chest films taken soon after arrival in the role 4 facility were reviewed in 96 of the 107 patients. In 55 (57.3%) films a tracheal tube was present. One or more radiological abnormalities was present in 66 (68.75%) of the films. Five patients met the consensus criteria for the definition of adult respiratory distress syndrome (ARDS). The majority of casualties with blast-related lung injury were successfully managed with conventional ventilatory support employing a lung protective strategy; only a small minority received non-conventional support at any time in the form of high-frequency oscillatory ventilation. Of those casualties who survived to be received by the role 4 facility, none subsequently died as a consequence of lung injury.

Keywords: blast injuries; blast lung; radiology of blast lung injury; conventional mechanical ventilation in blast lung injury; HFOV in blast lung injury

1. INTRODUCTION

Medical support for UK military personnel is provided in 4 tiers or ‘roles’; in-field physician-delivered first-aid (role 1), in-field medical post (role 2), deployed field hospitals (role 3) and UK-based specialist hospital care (role 4). In the past, role 4 was provided by a combination of military and National Health Service hospitals, but with the rationalization of the Armed Forces medical services over the last 20 years all acute trauma has been admitted to the University Hospital Birmingham’s (UHB) NHS Foundation Trust (Selly Oak site) since 2001, where care is delivered by both military and NHS personnel. Military casualties surviving to role 3 receive world-class resuscitation and stabilization, including imaging and surgery as required, and those requiring further hospitalization are returned to the UK as soon as it is clinically safe to do so. Repatriation is coordinated by the Royal Air Force’s tactical medical wing and effected by their ‘critical care air support teams’, that are able to provide advanced critical care support during transfer in one of the Royal Air Force’s specially equipped C-17 Globemasters. Most of the patients evacuated in this way represent intensive care unit (ICU) to ICU transfers and in the period between 1 July 2008 and 15 January 2010 a total of 135 patients were admitted to UHB’s critical care services, of which 107 (79.2%) were injured by explosive devices, mostly improvised explosive devices (IEDs).

2. BLAST INJURIES

The origin of the mixture of powdered charcoal, sulphur and potassium nitrate known as black powder, or gunpowder, is commonly attributed to China in the period between the eleventh and thirteenth centuries but may have arisen independently at about the same time in the Middle East and Europe. Blast injuries have, therefore, been a feature of human activity for almost 1000 years. Injuries from explosions arise in a number of ways. In temporal order these include tissue damage from; the blast shock wave (primary blast injury), material propelled into the casualty (secondary), the casualty propelled against other objects (tertiary), heat, chemicals and toxins delivered by the device (quaternary) and finally the systemic inflammatory response provoked in the host (quinary) [1]. Injury from the shock wave is thought to arise from three distinct mechanisms—spallation, implosion and inertia. Spallation arises at the interface between media of high (tissue) and low (gas) densities where the high-density material has nothing to which it can transfer the kinetic energy received, causing it to accelerate like the last ball in a Newton’s cradle. Implosion damage is caused by the rapid compression and expansion of gas-filled tissues, originally postulated in 1812 [2]; while inertial damage is generated by shearing between tissues of different densities that are subject to differential

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acceleration by the shock wave. The key roles of density contrasts and gas in the mechanism of primary blast injury explain why the lungs are particularly susceptible to damage. The first formal descriptions of blast lung were made in air-raid victims of the second world war [3], but shock wave injuries were alluded to in the descriptions of injuries ascribed to the ‘wind of a [cannon] ball’ by Gilbert Blane, Physician to the Fleet, in 1785 [4]. More recently, our understanding of this condition has been informed by terrorist activity in the Middle East [5–8], the UK [9,10] and Europe [11,12].

3. BLAST LUNG

Fatal blast lung injury (BLI) can be sustained in the absence of any other external signs of trauma, thoracic [13] or otherwise [3,14–16]. Our series derives from 517 blast casualties, of which 95 (18.4%) were immediately fatal and 13 of the 412 surviving to role 3 (3.1%) died before they could be evacuated to the UK (figure 1). In a series arising from the battle of Monte Cassino in spring 1944 evidence of BLI was found in 34.5 per cent of a series of 87 autopsies performed in soldiers who died with no external evidence of thoracic injury [13], while diffuse pulmonary contusions were found in 47 per cent of the fatalities in Northern Ireland in the period between 1969 and 1974 [10]. The incidence of blast lung injury in fatalities in this series awaits the results of autopsy investigations.

The clinical diagnosis of blast lung is based on context, clinical symptoms and radiology. Symptoms may include respiratory distress, restlessness, and in some cases haemoptysis, associated with cyanosis and hypoxaemia. In some patients symptoms may be significantly delayed [3]. With respect to radiological features most of the available data are based on plain chest films [5,17–19] rather than computerized tomography (CT). Typical findings described to date include unilateral or bilateral focal opacities, diffuse unilateral or bilateral loss of lung translucency which, if unilateral, may be associated with reduced rib-expansion, and radiological evidence of barotrauma. The latter may include pneumothorax, pneumomediastinum, pneumopericardium, surgical emphysema, interstitial emphysema and haemothorax secondary to pulmonary parenchymal lacerations. Abnormalities on plain chest films have been reported in 52 per cent [17] to 91.7 per cent [5] of patients whereas respiratory symptoms are only reported in 22 per cent [17] to 50 per cent [5]. In the setting of this series of cases symptoms of chest pain and dyspnoea were not recorded and would, in any case, have been impossible to disentangle from similar symptoms arising from other injuries, shock or, in the case of patients who were unconscious or had severe head injuries, could not have been elicited. For example, in this series of 107 patients, 39 patients suffered one or more traumatic limb amputations, 20 suffered severe head injuries, and three suffered both. Symptoms would thus have been unreliable as a marker of blast lung in 62 (57.9%) cases. Radiological examinations were performed in all patients, but were not available on UHB’s picture archiving and communications system (PACS) in 19 (17.7%) cases. In the 88 patients for whom role 3 pulmonary imaging was available, 21 (23.9%) only had plain chest films taken, 11 (12.5%) only had CT and 56 (63.6%) had both. Most patients that had both examinations (87.5%) had plain chest films taken 1.6 (0.74 to 3.6) h prior to CT, the remaining eight patients had their chest films taken 4 (1.25 to 9.65) h after their CT . Overall, the two examinations were performed a median of 1.8 (0.75 to 4.2) h apart. Forty-two (47.7%) patients had a tracheal tube in place at the time of the first radiological examination (table 1).

4. INITIAL RADIOGRAPHIC EXAMINATION

Plain chest radiographs were abnormal in 48 of the 69 cases (69.5%) in which it was the first examination. The commonest abnormality was diffuse loss of translucency present in 43 (89.6%) and as a single
abnormality in 34 (70.8%). Focal opacities were present in 13 (27.1%), usually in combination with diffuse loss of translucency in 10 (20.8%, two of these also associated with mediastinal displacement), although in three cases (6.25%) this was the single abnormality. Mediastinal displacement was apparent in seven (14.6%) cases, but never as an isolated finding. One patient’s film showed the deep sulcus sign in association with mediastinal displacement. Thoracic CT was abnormal in 11 of the 19 cases (57.9%) in which it was the first examination, with abnormalities detected including pulmonary contusion in 10 (30.5%), surgical emphysema in five (26.3%), pneumothorax in four (21.0%), haemothorax in four (21.0%), and pneumomediastinum in three (15.8%).

Overall, pulmonary abnormalities were detected radiographically in 59 of the 88 patients (67%) for whom role 3 radiology was available and given that these examinations were performed relatively early in the process of resuscitation it is probable that these changes were blast-related, rather than the consequence of trauma-related acute respiratory distress syndrome (ARDS).

5. ROLE 4

Demographic data for the 107 casualties who were evacuated to role 4 is presented in table 2. No plain chest radiogram was performed on arrival in the UK in 11 patients. Three of these patients had head injuries and spent 84.6, 171.1 and 102.5 h, respectively, on intensive care; the remaining eight patients spent a median of 11.1 h on intensive care (range 1.5–49.9). One of these patients died within 2 h of ICU admission, the other 10 all survived to hospital discharge.

Plain chest films were taken in the remaining 96 patients within a median of 3 (1.6–10.7) h of admission to the ICU. In 55 (57.3%) a tracheal tube was present. One or more radiological abnormalities was present in 66 (68.75%) of the films. Not surprisingly, the number of abnormalities on plain chest radiographs was loosely associated with the average PF ratio during first 24 h and duration of mechanical ventilation.

Table 3. Relationship between plain chest radiology, PF ratio during first 24 h and duration of mechanical ventilation.

<table>
<thead>
<tr>
<th>number of radiological abnormalities on plain chest films taken on admission to role 4 intensive care</th>
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<tr>
<td>1</td>
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<tr>
<td>PF ratio (kPa)</td>
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<td>days of ventilation</td>
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6. VENTILATORY MANAGEMENT

Lung protective ventilation is the cornerstone of ventilatory management in the intubated blast injured casualties evacuated to role 4. Given that the vast majority of these casualties do not have isolated lung injuries, this forms only one component of their care, alongside management of their wounds and other injuries. Beyond an initial, brief (2–4 days), requirement for slightly more aggressive respiratory support in a minority, in the majority, the presence of blast lung injury is almost an epiphenomenon and has little impact on the casualties’ care, in that their need for respiratory support, particularly its duration, is determined by other factors. In others, its presence has more important effects and on occasions dominates the clinical picture; of the 107 casualties in this series, two were formerly referred to us for urgent consideration of extracorporeal membrane oxygenation (ECMO) although on both occasions this proved unnecessary, each being successfully managed with...
high-frequency oscillatory ventilation (HFOV) from the time of their arrival. This contrasts to blast lung in civilian terrorist events where the incidence of severe respiratory distress is higher, a phenomenon that may reflect the impact of explosions in enclosed spaces.

The principles of the lung protective conventional ventilation that we employ are based on the ARDS network protocol [21], with low tidal volumes (6 to 8 ml kg bw⁻¹), the application of PEEP and limitation of plateau pressures to less than 30 cm H₂O. Permissive hypercapnia is deliberately variably pursued dependent on the presence or absence of concerns around cerebral perfusion/raised intracranial pressure related to other injuries. In general, in the absence of concerns and metabolic acidosis, our target pH is 7.25–7.3. When concerns are present, particularly, where the trade-off between potential ventilator-induced lung injury and the need for control of PaCO₂ are critical, we have used intracranial pressure monitoring to help guide our decisions. We recognize that the use of PEEP in the management of blast lung injury is controversial; while a detailed review of our PEEP selection in this series is beyond the remit of this paper, we have in general adopted the ARDS network approach and have to date avoided complications associated with air- leaks. This is in contrast to our management of lung injury consequent to penetrating and ballistic injury where we have adopted a more conservative approach to the application of PEEP.

In most cases, conventional lung protective ventilation as outlined above has proved more than adequate for the respiratory support of the blast injured casualties in this series. In a small proportion of casualties, predominantly in the presence of ongoing sepsis with a profound systemic inflammatory response syndrome response where lung injury is likely to reflect more than just the consequences of blast, we have instituted HFOV as rescue ventilation when conventional ventilation is failing. Failure of conventional ventilation in our hands has predominantly been on the basis of failure of oxygenation (defined as a requirement for an FiO₂ more than 0.6 for 4 or more hours when being expertly ventilated). In each event HFOV has proved highly effective and successful.

When using HFOV in this setting, either as rescue or ab initio, we have conceptually seen it as an extension of the concept of lung protective ventilation, and have aimed to exploit permissive hypercapnia to the same degree, accepting similar targets as we would during conventional ventilation. Where practical, we have also used controlled cuff deflation of the endotracheal tube to further enhance lung protection by reducing the need to drive CO₂ clearance through changes in frequency and amplitude. In those casualties with the most profound lung injury receiving HFOV, hypercapnia rather than problems with oxygenation has presented challenges; to date when this has arisen active cooling, following the exclusion of a partially occluded endotracheal tube and detailed review of the oscillatory parameters, has proven effective. Contingency planning, should these measures have proved ineffective, has included the use of tris-hydroxymethyl aminomethane (THAM), extra corporeal CO₂ removal and ECMO, but fortunately none has been required to date.

7. CONCLUSIONS

Blast lung injury appears to be a relatively common accompaniment to the polytrauma experienced by military casualties exposed to blast. In this series, of those casualties who survived to be received by the role 4 facility, none subsequently died as a consequence of lung injury. In our experience the majority of casualties with blast-related lung injury have been very successfully managed with conventional ventilatory support employing a lung protective strategy with only a small minority requiring non-conventional support in the form of HFOV.

The Royal Centre for Defence Medicine has funded the audit of this patient series.

ENDNOTES

1 When a ball then passes close...there is, in the first place, a great addition to the pressure...from the condensation of the air: as soon as the ball is passed, this pressure, with a great part of the atmosphere, is taken off; the consequence of which is a sudden expansion of all the fluids in the stomach and the blood in its blood vessels, and the rupture of both.'.

2 Calculated as the arterial partial pressure of oxygen (PaO₂) divided by the fractional inspired oxygen concentration (FiO₂).

3 Increased fractional inspired oxygen concentration, higher positive end-expiratory pressure (PEEP), and increase in the inspiratory to expiratory ratio.

REFERENCES


