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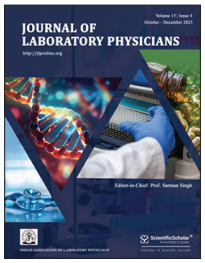

Case Report

Primary blast lung injury

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ABSTRACT

In an era where terrorism and conflict between countries are increasing, our knowledge about the impact of various weaponry including bomb blasts on the human body is of utmost importance. Undoubtedly, the immediate effect would be a surgical casualty. However, understanding the effect on other organ systems helps us manage effectively to reduce morbidity and mortality. Here, we present a middle-aged soldier with polytrauma and primary lung injury after an improvised explosive device blast. After autopsy, histology of the lung showed alveolar rupture, intra-alveolar hemorrhage, denudation of alveolar, bronchial and bronchiolar epithelium, collapse of alveoli, compression of bronchi, bronchioles, and air embolism. He was managed in the intensive care unit with mechanical ventilation and died of refractory shock. Although the pathogenesis, diagnosis, and management of primary lung injury due to blast explosion have already been studied, the autopsy and histopathological features are explained in detail in this article.

Keywords: Acute lung injury, Blast injuries, Pathology, Polytrauma, War-related injuries

INTRODUCTION

Blast injury refers to the injury that occurs by disruption and compression of tissues on exposure to a blast wave.[1] When the explosive detonates, it expands into a gas that displaces the surrounding air, creating a shock wave with high pressure known as a primary blast wave, which directly affects air-filled organs, particularly the lungs. Primary blast lung injury is defined as "radiological and clinical evidence of acute lung injury occurring within 12 h of exposure and not due to secondary or tertiary injury."[2] Usually, the patient develops cough,[3] dyspnea followed by apnea, persistent hypotension, and bradycardia as an autonomic response to primary blast injury.[4] Clinical presentation can be complicated by acute respiratory distress syndrome (ARDS), pneumothorax, hemothorax, and pulmonary embolism depending on the blast wave severity. Diagnosis is made with chest X-ray and supportive care with mechanical ventilation is given to these patients. The incidence of primary blast lung injury is >90% when an explosion happens in a closed space.[2] Research, especially animal experiments on the physics of blast waves and the pathophysiology, diagnosis, and management of primary blast lung injury are well documented. However, human blast lung injury, especially histopathology, is not well studied. We took this as an opportunity to study the histopathological features of blast lung injury and correlate them with its pathophysiology.

CASE REPORT

After an improvised explosive device (IED) blast, a 52-year-old soldier suffered trauma to his extremities. After stabilization in a nearby clinic, he was airlifted and arrived at our hospital

approximately 6 hours after the blast explosion. At the time of the presentation, he was not conscious (Glasgow coma scale E1V1M2), and bilateral pupils were normal sized and reactive to light. On examination, extensive crush injury of bilateral lower limbs, punctured wound, and friction burn over the extremities were present. There were no injuries seen in the chest or abdomen. Extended-focused assessment with sonography in trauma showed no remarkable findings. Because of extensive injuries to his extremities, X-rays of all extremities, head and neck, and chest were ordered which showed fractures in the left distal tibia, left calcaneum, right distal ulna, and left thumb. There were no fractures over the ribs or spine observed. Chest radiograph shows showed diffuse bilateral ground glass opacities predominantly in the middle and lower zones suggestive of pulmonary edema or alveolar hemorrhage [Figure 1a] and did not show any features of hemo/pneumothorax. Because of continuous desaturation, he was given mechanical ventilation in volume-controlled ventilation mode with positive end-expiratory pressure 6, tidal volume 450 mL, plateau 22, and respiratory rate 18/min. His vitals were oxygen saturation at 98%, pulse at 94/min, and blood pressure at 70/40mmHg. Because of hypotension, he was transfused with 1L crystalloids, and subsequently, the massive hemorrhage protocol (blood products 4/4/4) was followed. However, he developed refractory hypotension, and recurrent hyperkalemia (6.4–8.3) with metabolic acidosis and eventually succumbed to death the next day.

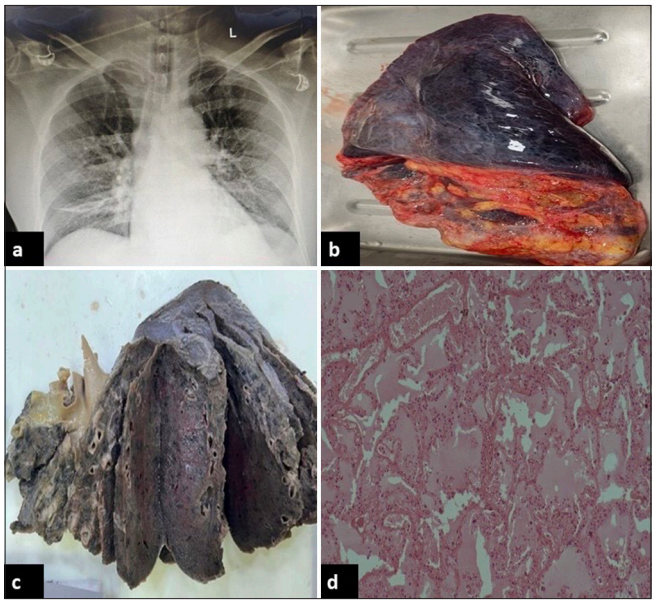

Figure 1: (a) Chest radiograph shows diffuse bilateral ground glass opacities predominantly in the middle and lower zones. (b) Gross image sowing enlarged boggy right lung (during autopsy). (c) Cut surface shows dilated air spaces in the periphery of lung (after 24 h of fixation with 10% Formalin). (d) Hematoxylin and eosin stained section (×100) showing pulmonary edema - transudative fluid filled alveolar spaces with thickened alveolar septa and congested capillaries

Autopsy presentation

The deceased was a well-built and moderately nourished male. External examination, multiple abrasions, contusions, and subcutaneous deep puncture lacerations and friction burns involving the upper and lower limbs; charring and singeing of surrounding hair, multiple avulsed lacerations over the forearm, and thumb suggestive of blast injuries were noted. The chest and abdomen did not show any signs of injury externally. Multiple comminuted fractures and splinter injuries over the shaft of right ulna, first metacarpal of left hand, proximal and distal phalanx of left thumb, shaft of left tibia, and fibula bone and left calcaneal bone were present. On internal examination, the right lung was found to be posteriorly adherent to the chest wall. Both the lungs were congested, heavy, edematous, and firm in consistency at multiple sites [Figure 1b]. On the cut section, blood mixed with frothy fluid was seen coming out of both the lungs consistent with blast lung injury. The right and the left lungs weighed 1020 g (Mean 450 g) and 740 g (Mean 375 g), respectively. After formalin fixation, on serial slicing, multiple air spaces of 0.5–1 cm were noted in the periphery [Figure 1c]. On microscopy, sections taken from bilateral lower lobes showed fluid-filled air spaces with thickened alveolar septa and congested capillaries along with leakage of fluid into the interstitium suggesting intra-alveolar and interstitial edema. [Figure 1d] sections taken from bilateral upper lobes showed open alveolar spaces with ruptured alveolar septa, especially near the pleura [Figure 2a] which was seen grossly as well. Intra-alveolar hemorrhage was seen focally [Figure 2b]. Desquamated bronchial epithelium [Figure 2c] and alveolar macrophages and soot particles [Figure 2d] were seen in the alveolar lumen. The bronchial lumen was intact with denuded bronchial lining epithelium [Figure 3a]. Focal inflammation with dilated capillaries along the submucosa of the bronchus was noted. The pulmonary interstitium showed inflammation [Figure 3b] along with a few soot particles. There were multiple bronchioles with compressed lumen and disrupted walls as evidenced by disorganized smooth muscle fibers [Figure 4a] along the wall. Multiple dilated and congested pulmonary vessels, few vessels with ruptured walls, and few thrombosed pulmonary veins were seen [Figure 4b]. All the features are suggestive of acute lung injury due to pulmonary barotrauma.

DISCUSSION

An explosion can cause injuries to almost all the people in the area. The severity of injury varies from visceral organ injury to fractures, burns, and death.[5] It depends on (i)

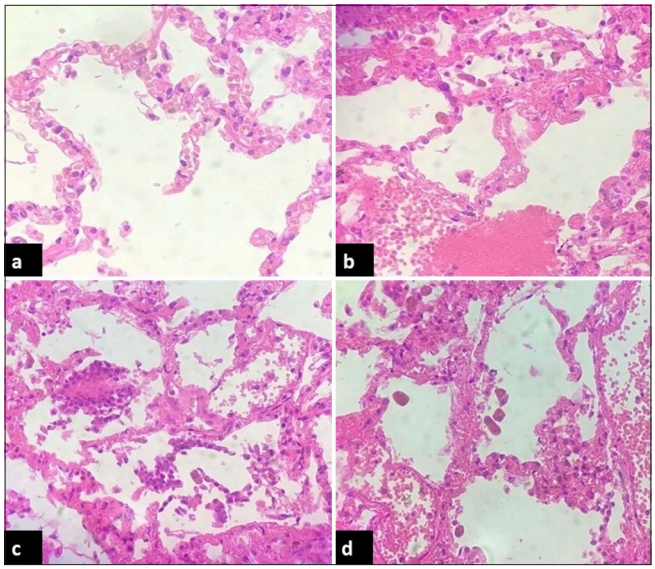

Figure 2: Hematoxylin and eosin stained photomicrographs of alveoli (a) delicate and ruptured alveolar septa lined by attenuated pneumocytes and dilated capillaries (×400). (b) Intra-alveolar hemorrhage (×400). (c) Desquamated bronchial lining seen in alveolar lumen (×400). (d) Inhaled soot particles in the alveolar lumen (×400).

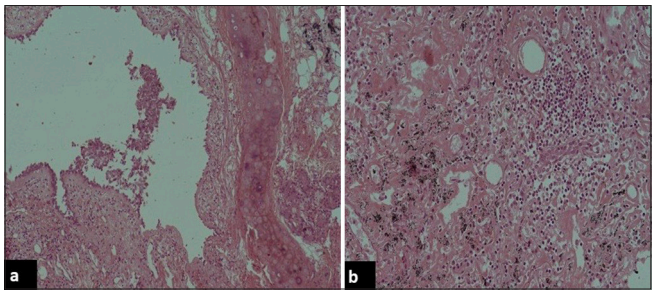

Figure 3: (a) Hematoxylin and eosin stained section (×200) showing desquamated bronchial lining epithelium in the lumen. (b) Hematoxylin and eosin stained section (×200) showing Infiltration of lymphocytes in the interstitium consistent with interstitial inflammation.

the explosion (nuclear, chemical, or mechanical), (ii) the amount of kinetic energy released in a short duration of time (low-order explosive or high-order explosives), (iii) the distance between the victim and the explosion, and (iv) the location of the blast (indoor or outdoor).[6] Blast injury refers to complex physical trauma as a result of direct or indirect exposure to an explosion. Depending on the mechanism of injury, blast injuries are classified into primary (direct injury by primary blast wave), secondary (when struck by sharp objects), tertiary (thrown out and impacted by other objects), quaternary (inhalation of toxic gases), and quinary (exposure to radiological and biological residues).[6] When the explosion happens, it expands into gas, displacing the surrounding air, and creating a shock wave with high pressure known as a

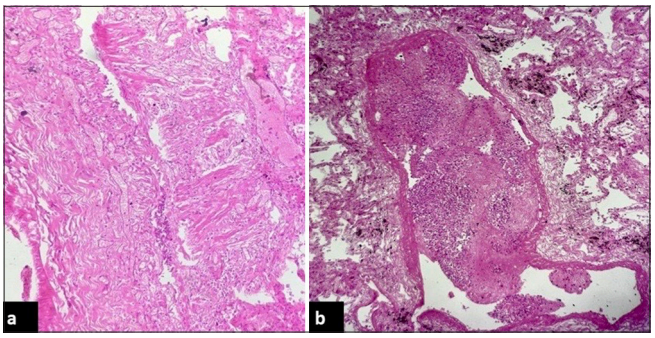

Figure 4: (a) Hematoxylin and eosin stained section (×100) showing Collapsed bronchiolar lumen with disrupted wall seen as discontinuity in the smooth muscle fibers and denuded lining epithelium. (b) Hematoxylin and eosin stained section (×200) showing thrombus in pulmonary vein.

primary blast wave. It has an effect similar to blunt trauma on internal organs.[7] Primary blast injuries occur when the high-pressure blast waves created by an explosion crush the body and cause internal injuries. The primary blast wave directly affects the air-filled organs such as the lungs, eyes, ears, brain, and gastrointestinal tract. Our case presented features of primary blast lung injury.

Explosives are substances that contain large amounts of energy in chemical bonds. Most commercial explosives are organic compounds that contain -NO_2, -ONO_2, and -$NHNO_2$ which release gases such as nitroglycerin, trinitrotoluene (TNT), high melting explosive (HMX), and nitrocellulose in an explosion.[8] Based on the rate of combustion, they are classified into high-order explosives (detonate-explode) or low-order explosives[6] (deflagrate-burn rapidly), namely, high-order chemical explosives are as follows: (1) Military explosives: C4, royal demolition explosive (RDX), and pentaerythritol tetranitrate (PETN), (2) Commercial explosives: type of body cavity bomb (ANAL), ammonium nitrate fuel oil (ANFO), dynamite, and urea nitrate, and (3) improvised explosives: home made peroxide explosive (HMTD) and triacetone triperoxide (TATP). Low-order explosives are gunpowder, petroleum-based bombs, and pipe bombs. An IED that comes under the high-order explosive is homemade and is extremely sensitive to heat, shock, and friction. They are common in asymmetric warfare and terrorism.[9] In our case, the patient developed a primary lung injury after an IED explosion.

As blast explosion affects various organ systems simultaneously, the morbidity and mortality of primary blast lung injury alone is difficult to assess. However, during the war in Afghanistan, it was reported that 6–11% of military casualties sustain primary blast lung injury after an explosion.[2] The incidence of primary blast lung injury is >90% when the attack occurs in a closed space. Among those who die of a blast explosion, there is an 80% incidence of

primary blast lung injury.[10] It is the only autopsy finding in 17% of them.[11]

Tsokos *et al*. reported a case series on the histological, immunohistochemical, and ultrastructure of primary blast lung injury with microscopic features such as alveolar rupture, thinning of septum, and enlargement of alveolar spaces along with circumscribed subpleural, intra-alveolar hemorrhage, and cuff like perivascular hemorrhages in the interstitium in all the cases.[3] Congestion of the pulmonary artery, arterioles, veins, venules, and alveolar capillaries was seen in half of them. A few cases had eosinophilic intra-alveolar deposits, alveolar and interstitial edema, venous air embolism, pulmonary bone marrow embolism, and soot aspiration. While correlating the histopathological features with the prognosis, it was found that the intensity of pulmonary edema is positively correlated with the survival time. The remaining histological features did not show any correlation with age, explosiveness, and distance between the victim and the site of the explosion. There was a debate on whether air embolism is caused by blast injury or mechanical ventilation. As pulmonary fat embolism can be seen after blunt trauma, it is significant as it precipitates ARDS and increases mortality.[3] The histological features noted in our case are following this study.

There are many proposed mechanisms of lung injury after an explosion. In the biochemical mechanism of lung injury, alveolar rupture causes intra-alveolar hemorrhage, and lysis of red blood cells which leads to the release of free radicals resulting in oxidative damage, and activation of an inflammatory cascade accelerating lung injury.[12] However, there are controversies about whether it causes lung injury or occurs as a result of lung injury.[13,14]

Kirkman and Watts described the characterized response to primary blast injury. The primary blast wave causes lung injury resulting in the rupture of alveolar capillaries leading to intraparenchymal hemorrhage and extravasation of fluid resulting in interstitial pulmonary edema.[4] After 3 h of blast injury, WBCs are recruited in the hemorrhagic areas which peak at 12 h.[15] This increases the myeloperoxidase activity, causing excessive oxidative stress and inflammation.[16] Only intraparenchymal hemorrhage and pulmonary edema without endothelial damage are seen within 12 h of primary blast lung injury.[17] Pulmonary gas exchange gets affected and the patient clinically presents with hypoxia and hypercarbia. Damage to type I pneumocytes begins after 12 h and injury to endothelial cells occurs after 24 h of the explosion. The role of inflammatory mediators in primary blast lung injury is further documented by animal studies where administration of N acetyl cysteine[18] and haemin (activates haem oxygenase-1)[19] increases the survival time of blast-exposed rats by suppressing inflammation. In our case, histological evidence of pulmonary edema damaged alveolar epithelium, and endothelium as evidenced by thinned-out alveolar septa and pulmonary vein thrombus are seen.

Studies have shown that thoracic blast injury causes cardiorespiratory response characterized by transient hypercoagulability and triad of bradycardia,[20] and persistent hypotension[21-24] apnea mediated by the autonomic nervous system. The proposed theory is blast lung injury activates pulmonary afferent C fibers mediating vagal reflex which results in bradycardia and apnea.[25] The release of nitric oxide from damaged endothelial cells, decreased vascular resistance and bradycardia leads to persistent hypotension. This theory is established in the cases of isolated thoracic blast injuries with persistent hypotension. Although our patient had persistent hypotension, the absence of bradycardia attributes it to multiple fractures.

In our attempt to correlate histology with blast pathophysiology, the complex physics involved in the development of lung injury after an explosion needs to be understood. Pulmonary barotrauma is a tissue injury caused by a pressure-related change in a body compartment gas volume in the air-containing areas of the body. When there is a difference in pressure between a gas situated inside the body and the surrounding gas, there will be an expansion of gas within the alveoli which causes over-distension and alveolar damage. During the explosion, a blast wave is formed, which is nothing but high-pressured gas. On inhalation of compressed air, increased pressure on alveoli has an impact on its lining which results in inflammatory changes. When the pressure decreases, volume expands, which causes the over distension and rupture of alveoli along with the surrounding blood vessels.[26] The alveoli will be flooded with blood and debris and collapse eventually. Depending on the location of the alveolar rupture, air leaks out and moves toward the mediastinum causing pneumomediastinum and pneumopericardium. Air may move toward the head and neck causing subcutaneous emphysema or track along the esophagus causing pneumoperitoneum. Rupture of the peripherally located alveoli can cause pneumothorax, tension pneumothorax, or pneumatocoele formation. Air may get trapped in the interstitium to form pulmonary interstitial emphysema. Alveolar air may leak into the capillaries or central circulation and result in the formation of air emboli which can be life threatening.[27] The speed of transmission of these high amplitude waves through the lung does not allow energized gas to escape along the airway, resulting in a collection of air bubbles into veins causing venous air embolism. It can also cause injury to the airways, including the trachea, bronchi, and bronchioles. This can result in epithelial disruption [Figure 3a], mucosal ulceration, and peribronchial inflammation.[28] The blast wave can cause damage to pulmonary blood vessels including capillaries, arterioles, and venules, resulting in hemorrhage [Figure 2b], thrombosis [Figure 4b], and disruption of the pulmonary vascular bed, contributing to impaired oxygen delivery and pulmonary hypertension. When the pressurized gas compresses the lungs

from outside lung volume falls below the residual volume, this causes mucosal edema, vascular engorgement, and pulmonary edema [Figure 1d].[29] Inhaled smoke, toxic gases, or soot particles [Figure 2d] can cause chemical pneumonitis, airway injury, and respiratory compromise characterized by diffuse alveolar damage with features such as alveolar hemorrhage, edema, and hyaline membrane formation. This condition can rapidly progress to ARDS.[30]

The histological features of primary blast lung injury help us to understand the blast pathophysiology. Causalities with primary blast lung injury require supportive care with mechanical ventilation in the intensive care unit. The ventilatory strategy should be a low-volume open lung approach with the use of high PEEP for ARDS and low PEEP for bronchopleural fistula and pneumatocele.[31] Since primary blast lung injury accompanies polytrauma in this case, the combination of blast injury and hemorrhagic shock is life-threatening as the thoracic blast injury itself causes persistent hypotension. Damage control resuscitation must be expedited in this kind of scenario. In the long-term, the survivors have excellent recovery, being asymptomatic with normal exercise tolerance and normal lung function tests.[32]

CONCLUSIONS

As blast injury of the lungs is a relatively common accompaniment to polytrauma in causalities exposed to blast, histopathological features of blast lung injury help us to understand its pathophysiology better, which will, in turn, lead to the effective management of these patients.

Author contributions: RR: Histopathology reporting and writing the manuscript; DPB: Conducted the autopsy, collected gross for histology; AbS: Histopathology reporting; ArS: Conceptualized the original idea, reporting and reviewed the final manuscript; SL: Reviewed autopsy and the manuscript.

Ethical approval: The Institutional Review Board approval is not required as the case report is based on a medicolegal autopsy conducted at our institution.

Declaration of patient consent: As the subject was deceased and the autopsy was conducted on legal grounds, informed consent was not applicable.

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