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Case Report

Lung involvement in accident victims: report of three cases

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ABSTRACT

Lungs are target organ for damage during accidents and contribute significantly to the mortality of the victims. Here we present three different types of accidental injury to the lungs leading to respiratory failure and their varied presentations.

Keywords: Blunt trauma, Burn injuries, Drowning, Lung contusion

INTRODUCTION

Lung is a target organ for damage in most of the accidents. Lungs are involved either directly or indirectly. Lung involvement often contributes to mortality by asphyxiation, hypoxaemia or hemorrhage. Survivors are at the risk of long term morbidity due to lung damage.

Thoracic trauma accounts for 25% of death due to road accidents. About 70% of patients with polytrauma had major thoracic injuries.[1] Blunt trauma of chest occurs when chest is compressed between two hard objects or due to deceleration force when moving body hit against a hard surface. Lung contusion is seen in 30-75% of patients with significant blunt trauma.[2]

Fire injuries to the lungs and airways from smoke inhalation are often less apparent and may not present until 24-36 hours after exposure. Approximately 10-30% of all burns admissions had smoke inhalation injury. Smoke or toxic fumes was partly or wholly the cause of death in 53% of fatalities in burn victims.[3]

Drowning is yet another important form of accidents. This is the sixth leading cause of accidental death for people of all ages and the second leading cause of death for children aged 1-14 years.[4] The target organ of submersion injury is the lung. Aspiration of as little as 1-3 mL/kg of fluid leads to significant impairment of gas exchange. Injury to other systems is largely secondary to hypoxia and ischemic acidosis.

Here we present three cases where lung is severely involved in accidents leading to respiratory failure. With early identification of the type and magnitude of lung injury all the three patients were saved. This report is to highlight three important ways of accidental injury to the lungs and their clinical presentation.

CASE REPORT

Case 1

30-year-old female was involved in road traffic accident and admitted with fractures of both bones of both legs. After 8 hours of admission she had tachypnea and fall in oxygen saturation. Saturation maintained between 85-90% with 4 liters of oxygen. Pao2 was 124 mmHg on oxygen with mild alkalosis. Clinically she was tachypnoec. There was no evidence of chest wall injury. Lungs on auscultation showed coarse crackles in the interscapular and infrascapular area on both sides. She remembers being thrown out of the car and falling on the road during collision. There was mild leukocytosis.

Blood sugar and Blood urea were within normal limits. She was put on antibiotics and other supportive care including noninvasive ventilation. Her saturation tended to fall when NIV support was withdrawn. Systemic corticosteroid was started as an anti-inflammatory measure. By 3 days her clinical status stabilized and saturation was maintained at 94% on 2 L oxygen. Her ABG parameters on 4th day were PaO2 78 mmHg, PaCO2 36 mmHg, pH 7.34 and HCO3 34. X-ray and CT showed resolution of shadows and she was shifted to the ward.

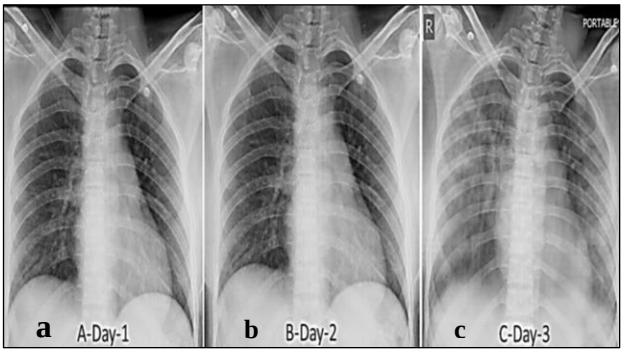

Figure 1a, 1b and 1c: X-ray of the patient at admission (a) and within the next 48 hours (b and c).

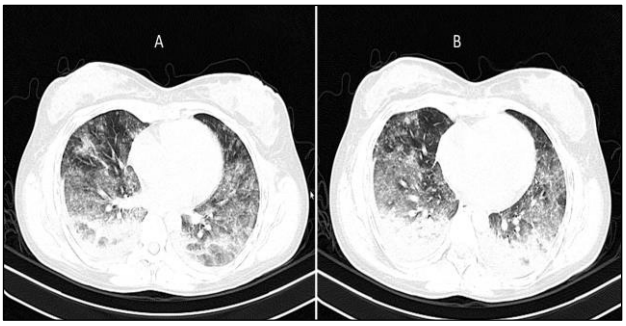

Figure 2: HRCT thorax showing non-segmental, sub pleural opacity in the paravertebral gutter on both sides with less dense shadows towards the deeper parenchyma.

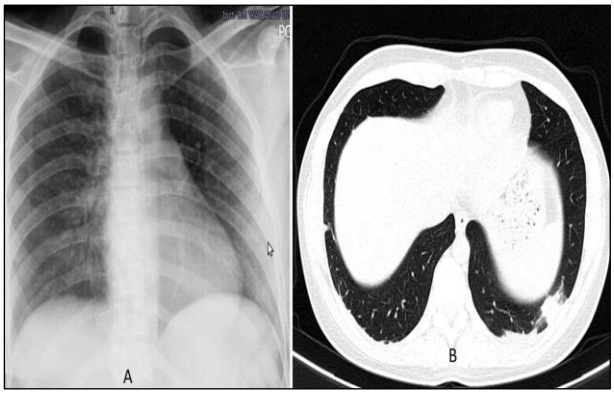

Figure 3A and 3B: X-ray taken on the 8th day (A) and CT thorax taken on the 14th day (B) show resolution.

Case 2

42-year-old male patient was brought to the Emergency Department as a referral case from King Fahad Hospital, Dammam, with history of 45% flame burns and pulmonary injury. He was treated there with wound care, antibiotics, and mechanical ventilation for 3 months. He was weaned off from ventilator and referred to India. On arrival, patient was conscious, oriented and vitals were stable. He had scars of superficial burns on face, anterior chest wall, abdomen and upper limbs. There was a deep ulcer on the left arm with slough.

Tracheostomy tube was in situ and the patient was having dyspnea on minimal exertion. He was detected to be a diabetic recently. His oxygen saturation at rest on room air was 78% with a PaO2 of 54 mmHg and normal pH and PaCO2. Routine investigations were done, which showed anemia, pus cells in urine and poor glycemic control.

His routine investigation showed a Glycated Hb (HbA1c): 5.8%, Fasting Blood Sugar (FBS): 209.0 mg/dL, HIV 1+2 CARD: negative, HCV (CARD): negative; Bleeding time (BT): 2.0 minutes Clotting Time (CT): 4.30 minutes, Haemoglobin (HB): 7.9 gm/dl, HBsAg Card: negative, Total WBC Count (TC): 7060.0 /µl, Basophils: 00 % , Monocytes: 04 %, Eosinophils: 03 %, Lymphocytes: 36%, Neutrophils: 57%, MCV : 84.1 fl, MCH: 26.8 pg, I Ca: 1.152 mmol/L, Potassium: 4.6 mmol/L, Sodium: 139.0 mmol/L, Serum creatinine: 1.6 mg/dL, and Urea: 59.0 mg/dl.

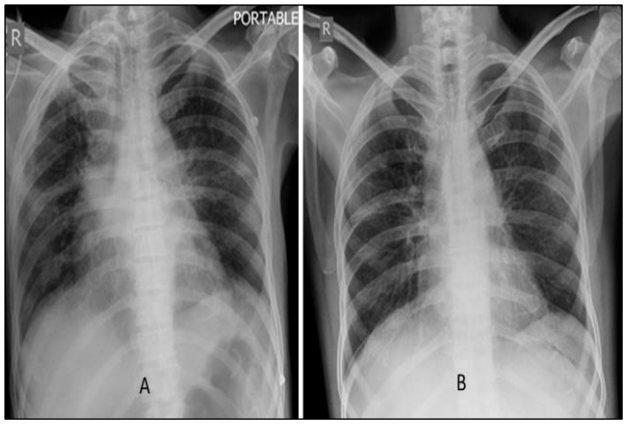

Figure 4A and 4B: X-Rays of the patient on admission (A) and after 9 days of treatment (B).

His X-ray chest showed diffuse non-homogenous infiltrate bilaterally. CT thorax showed features of residual pulmonary infection with fibrotic changes involving apical and anterior segment of bilateral upper lobes, medial segment of right middle lobe and inferior lingular segment of lingular lobe; Segmental bronchopneumonia in apical segments of bilateral lower lobes and tiny nodular opacities seen in bilateral lower lobes of lung. Sputum culture yielded growth of

Klebsiella species sensitive to Cefoperazone-Sulbactam, Amikacin, Aztreonam, Netilmicin, Gentamicin, Meropenem and Imipenem; and Pseudomonas species sensitive to Gentamicin, Ciprofloxacin, Piperacillin-Tazobactam, Amikacin and Tobramycin.

He was treated with antibiotics, wound cleaning, skin grafting and oxygen supplementation. He had some residual shadows in the x-ray suggestive of damage due to thermal injury and a short course of oral steroid was given. Patient improved and oxygen saturation reached 86% without supplemental oxygen.

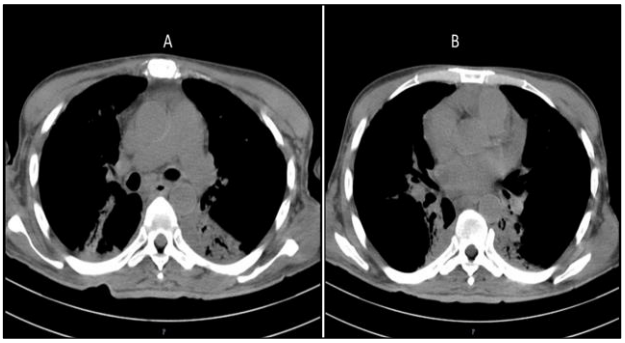

Figure 5: CT thorax axial window showing segmental collapse, fibrosis and bronchiectasis.

Case 3

A 15-year-old male student on a picnic with his friends fell into a lake while taking photographs. After initial resuscitation at the site he was brought to the ED in a conscious state with oxygen saturation at 65%. He was put on NIV and supplemental oxygen at 6 L/minute and shifted to ICU. His ABG showed a PaO2 of 54 mmHg, PaCO2 of 32 mmHg, pH of 7.24 and HCO3 24. There was no history of convulsions and there was no neurological deficit. His X-Ray and HRCT Thorax showed diffuse alveolar infiltrates. ECG and EEG were normal. Blood parameters were within normal limits. By the fourth day his respiratory rate, SaO2, ABG and X-ray chest were found to be within normal limits.

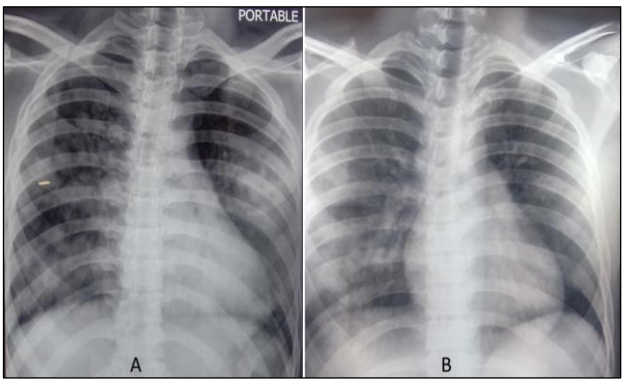

Figure 6a and 6b: X-ray chest of the patient on admission (A) and after 4 days (B).

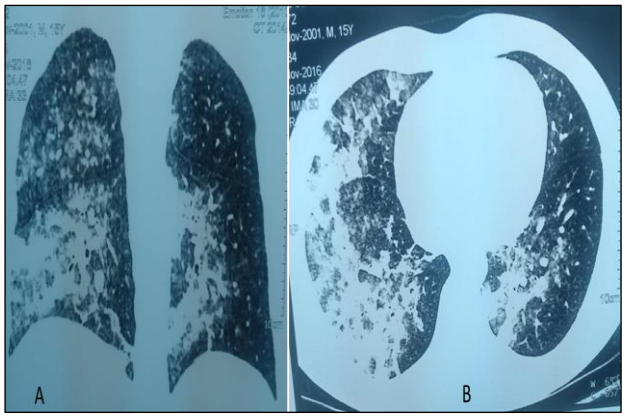

Figure 7: HRCT thorax of the patient, both coronal view (A) and axial view (B) showing alveolar infiltrates.

DISCUSSION

Road traffic accidents represent the most common cause of major thoracic injury among patients attending emergency department (ED).[1] Several factors are associated with a higher risk of thoracic injury such as speed of the vehicle, not using seatbelt, extensive damage to the vehicle and steering wheel deformity.[5,6] Thoracic trauma accounts for 25% of death due to road accidents. Blunt trauma of chest occurs when chest is compressed between two hard objects or due to deceleration force when moving body hit against a hard surface. Lung contusion is seen in 30-75% of patients with significant blunt trauma. The risk of lung contusion appears to correlate with severity of crash and the proximity of the site where the patient hits.[7] Lung damage leads to ventilation-perfusion inequalities and decreased lung compliance. The possible mechanisms for contusion are the alveolar tearing or shearing at the gas-liquid interface. Interior of the lung may be pulped even in the presence of intact visceral pleura from transmitted forces or massive variation in intrathoracic pressure during the impact. In lung contusion, there will be vertical line of sub pleural bruising in the paravertebral gutter. Micro-hemorrhage may occur and large amount of blood is leaked to alveolar space compromising ventilation. Lung often shows areas of bleeding under the pleura which may be due to contusion or due to aspiration of blood from other damaged areas of the lung. This is frequently associated with rib fractures. Hemoptysis is usually an accompanying symptom. Lung contusions generally develop over the first 24 hours and resolve in about one week.

X-ray chest shows irregular, nonlobular opacification of the pulmonary parenchyma which is the diagnostic hallmark. Chest CT will clearly delineate the lesion. Contusion may become visible in X-ray and CT over the first 24-48 hours after trauma as bleeding and edema into lung tissues progress. CT scan is the sensitive investigation compared to plain X-ray in detecting contusion, that too as early as 6 hours of injury. It shows

dense non-segmental, sub pleural opacity on the side of impact and the lesion becoming less dense and nonhomogeneous towards the deeper parenchyma. This finding is characteristic of pulmonary contusion. CT scanning also helps determine the size of a contusion, which is useful in determining whether a patient needs mechanical ventilation. A larger volume of contused lung on CT scan is associated with an increased likelihood that ventilation will be needed. CT scans also help differentiate between contusion and pulmonary hematoma.

Inhalation injury resulting from fire is one of the leading causes of death.[3] Inhalation injury is common following burn injury and increases in incidence with the size of the burn injury and age of the patient. Injuries to the lungs and airways from smoke generated from fire are less apparent initially and may not present until 24-36 hours after exposure. Inhalation injury refers to damage to the airways or lung parenchyma from heat, smoke, or chemical irritants entering the respiratory system during inspiration.[8] Severe external burns to the chest also can induce parenchymal damage. It is reported that 10-30% of all burns admissions had smoke inhalation injury.[9] Gas, smoke or toxic fume inhalation is the cause of death in 53% of fatalities in fire accidents. A further 19% were due to external burns in addition to inhalation of gas or smoke.[3] Pulmonary complications following burns and inhalation injury are responsible for up to 77 percent of the deaths, among which the majority are due to carbon monoxide poisoning.[10] In addition, inhalation injury has been shown to be an independent predictor of mortality in burn patients. Risks are increased by being in a confined space, duration of exposure, substances being burned that may emit various poisons and pre-existing respiratory disease.

There are three basic ways by which lung damage occurs in fire accidents. Heat causes thermal damage, gases cause asphyxiation and there may be irritation of the lungs or airways by smoke or fumes. Irritation of the lungs and airways will incite an inflammatory response with bronchospasm and an outpouring of fluid. Subglottic stenosis, bronchiectasis, pulmonary oedema and atelectasis can occur. Injury to the lung parenchyma is characterized by atelectasis and alveolar collapse resulting in increased transvascular fluid flux, a decrease in surfactant, and a loss of hypoxic vasoconstriction and therefore impaired oxygenation. Furthermore, a severe imbalance in alveolar hemostasis and decreased antifibrinolytic activity, with massive fibrin deposition in the airways, causes a ventilation-perfusion mismatch.[10] Airway obstruction and atelectasis leads to pneumonia. The risk for pneumonia is increased due to impaired function of alveolar macrophages, polymorphonuclear leukocytes, and mucociliary clearance mechanisms.[11,12]

Chest radiography is typically obtained in the initial evaluation of the injured patient but has low sensitivity for inhalation injury. Most patients with inhalation injury have a normal chest radiograph at presentation and presence of pulmonary opacities on initial chest films has been implicated as a marker of severe injury and a poor prognosis.[10,13,14] Computed tomography of the chest may be helpful as an early predictor of smoke inhalation severity based on airway wall thickness.[15,16] Chest computed tomography (CT) scans may show ground-glass opacities in a peribronchial distribution and/or patchy peribronchial consolidations. These findings may be present on CT scan as early as a few hours after inhalation injury.

Drowning is the sixth leading cause of accidental death for people of all ages and the second leading cause of death for children aged 1-14 years, after motor vehicle collisions.[17] Drowning is a process resulting in primary respiratory impairment from submersion in a liquid medium and the outcomes were described to death, morbidity, or no morbidity.[18] This replaces the earlier terms like near-drowning and non-fatal drowning. Every year, drowning accounts for at least 500,000 deaths worldwide and nonfatal events may occur several hundred times as frequently as reported drowning deaths.[19,20] The age distribution of submersion injury is bimodal. The first peak occurs among children less than five years of age who are inadequately supervised. The second age peak is seen among males between 15 and 25 years old, and these episodes tend to occur at rivers, lakes, and beaches.

The target organ of submersion injury is the lung. Asphyxia leads to relaxation of the airway, which permits the lungs to take in water in many individuals. Aspiration of as little as 1-3 mL/kg of fluid results in significant impairment of gas exchange. Injury to other systems is largely secondary to hypoxia and ischemic acidosis. Destruction of surfactant produces alveolar instability, atelectasis, and decreased compliance, with marked ventilation/perfusion (V/Q) mismatching. Post-obstructive pulmonary edema following laryngospasm and neurogenic pulmonary edema may also occur. ARDS from altered surfactant effect and neurogenic pulmonary edema often complicate management. Pneumonia is a rare consequence of submersion injury and is more common with submersion in stagnant warm and fresh water. Pulmonary insufficiency can develop insidiously or rapidly; signs and symptoms include shortness of breath, crackles, and wheezing.

The chest radiograph or computed tomography at presentation can vary from normal to localized, perihilar, or diffuse pulmonary edema. Chest radiography may detect evidence of aspiration, pulmonary edema, or segmental atelectasis suggesting the presence of foreign bodies. In symptomatic patients who do not require immediate intubation, supplemental oxygen should be provided to maintain the SpO2 above 94 percent. In addition, noninvasive positive pressure ventilation via CPAP (continuous positive airway pressure) or BIPAP (bi-level positive airway pressure) can improve

oxygenation and decrease ventilation-perfusion mismatch. Intubation and mechanical ventilation is indicated when there are signs of neurologic deterioration, inability to maintain a PaO2 above 60 mmHg or oxygen saturation (SpO2) above 90 percent despite supplemental oxygen, and PaCO2 above 50 mmHg.

Earlier studies reported survival rates as high as 75 percent, with approximately 6 percent suffering a residual neurologic deficit. The factors associated with a poor prognosis are duration of submersion >5 minutes, time to effective basic life support >10 minutes, resuscitation duration >25 minutes, age >14 years, Glasgow coma scale <5, persistent apnea and arterial blood pH <7.1 20.

CONCLUSION

In this report, we present three different types of accidents where lungs were severely affected and presented with acute respiratory failure. Lung is the target organ in most of the accidents, involved either directly or indirectly. Clinical manifestations vary from mild injury with bronchospasm and pneumonitis to acute respiratory distress syndrome. Timely resuscitative measures at the site of accidents, early assessment to ascertain the type and severity of injury and prompt treatment will help in complete recovery. However severe injury and secondary infection may leave some residual damage such as collapse, fibrosis and bronchiectasis.

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