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Respiratory failure and pneumosepsis secondary to airway inhalation injury – a case report of successful management

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Abstract

Smoke inhalation injury is a major determinant of morbidity and mortality in burn and closed-space fire incidents. Its clinical course is often biphasic and unpredictable, with early airway edema followed by delayed parenchymal injury and infectious complications. Diagnosis is primarily clinical, supported by bronchoscopy and imaging. We reported a young female with initially mild symptoms who rapidly deteriorated to respiratory failure and pneumosepsis requiring critical care.

A previously healthy 22-year-old woman presented two hours after accidental closed-space smoke exposure with tachypnea and bilateral wheeze but preserved oxygen saturation and a normal chest radiograph. Carbon monoxide (CO) oximetry confirmed elevated carboxyhemoglobin, which normalized with high-flow oxygen. Despite initial stability on high-flow nasal cannula and corticosteroids, she developed abrupt desaturation with radiographic right-lung collapse. Urgent fiberoptic laryngoscopy excluded upper-airway burns; she required invasive mechanical ventilation. Bronchoscopy revealed extensive soot deposition. Within 48 hours, she developed fever and evolving bilateral consolidations with diffuse ground-glass opacities on a computed tomography (CT) scan. Bronchoalveolar lavage culture grew Acinetobacter, treated as ventilator-associated pneumonia. With broad-spectrum antibiotics, steroids, bronchodilators, and physiotherapy, she improved and was successfully extubated and discharged on room air.

This case illustrates the deceptive latency and rapid deterioration typical of inhalation injury despite a benign initial examination. Smoke toxicity induced airway cast formation, immunological dysfunction, and predisposition to early pneumonia. Conventional pulse oximetry was unreliable due to interference from carboxyhemoglobin, mandating CO oximetry. Chest imaging might remain normal initially; bronchoscopy was valuable both for diagnosis and for therapy. Inhalation airway injury warrants mandatory 24–48-hour intensive monitoring, even in apparently mild presentations. Early bronchoscopy, aggressive sepsis surveillance, and multidisciplinary critical care are essential to prevent rapid progression to respiratory failure and pneumosepsis, as illustrated by this successfully managed case.

Keywords: Bronchoscopy, airway inhalation injury, burns, respiratory failure, sepsis, soot particles.

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Introduction

Inhalation injury or smoke inhalation injury is a life-threatening illness. The manifestation depends on the type of exposure, duration, and patient factors. The effects can be direct injury to the airway and systemic toxicity. (1) Pulmonary manifestations of the disease include airway edema, atelectasis, pneumonia, pneumonitis, sepsis, and acute respiratory distress syndrome. Clinical symptoms usually seen in practice are persistent cough, wheeze, melanoptysis (soot-containing secretions), breathlessness, and hemoptysis. (2) Here, we describe a young fe-

male presenting with mild symptoms that quickly progressed to respiratory failure and sepsis requiring invasive mechanical ventilation.

Case presentation

A 22-year-old female, with no known comorbidities, came with an alleged history of accidental exposure to inhalation smoke trapped in a closed room for 30 minutes at her workplace 2 hours before arrival at the hospital. She presented with difficulty in breathing since the exposure.

On arrival, she was alert and afebrile. On examination, she had tachypnea with a respiratory rate of 28 per minute with peripheral oxygen saturation (SpO2) 98% on room air and extensive bilateral wheeze. Hemodynamics were normal. Lab investigations showed mild hypoxemia. Chest X-ray and computed tomography (CT) of the thorax showed normal lung parenchyma. Routine investigations were normal except for an increased white blood cell count of 18,640/µl. Presenting carboxyhemoglobin was elevated in carbon monoxide (CO) oximetry done in the Emergency Room, which normalised after a few hours of high-flow oxygen (repeat carboxyhemoglobin was 0.5)

She was shifted to the Respiratory Intensive Care Unit for observation, started on high-flow nasal cannula (HFNC), intravenous (IV) steroids, and bronchodilator nebulisation. Suddenly, the patient desaturated to 64% on HFNC. Repeat chest X-ray showed right lung collapse and consolidation (**Figure 1**). A plan for invasive mechanical ventilation was discussed with the family. Upper airway examination for burn injury was ruled out urgently by the ear, nose, and throat (ENT) surgeons with bedside fibreoptic laryngoscopy. The patient was intubated and started on mechanical ventilation. Bronchoscopy was done, and bronchial wash from bilateral lower lobes showed soot deposits (**Figure 2**). The patient was continued on broad-spectrum antibiotics and supportive care.

The patient developed a fever after 48 hours on invasive ventilation. Repeat CT thorax showed bilateral lower lobe consolidation and diffuse ground glass opacities in the bilateral upper lobes. A repeat bronchoscopy was performed using non-video bronchoscopy, and bronchoalveolar lavage (BAL) was obtained. BAL bacterial culture showed Acinetobacter, for which antibiotics were escalated as per sensitivity results. The patient was treated with IV antibiotics, nebulisation, and IV steroids. Subsequently, the patient improved and was weaned off the ventilator. She was discharged on room air after completing a course of antibiotics, chest and limb physiotherapy in the ward, and other supportive care. After a few days, the patient came to the Outpatient Department for review with complete resolution of symptoms, and the chest X-ray repeated showed normal lung parenchyma (**Figure 3**).

Discussion

Smoke inhalation injury is a common occurrence in fire accidents, and it is an individual factor as a predictor of mortality. (1) The severity of injury is influenced by the source of the fire, the size of the smoke particles, exposure in an enclosed space, the duration of exposure, and gas solubility. (2) The injury can affect the upper airway, the tracheobronchial tree, or the lung parenchyma.

Upper airway injuries are predominantly caused by heat and can affect airway management. Tracheobronchial injuries result from inhaled chemicals, which trigger an inflammatory cascade that leads to exudation of proteins and the formation of airway casts, resulting in lung collapse. Parenchymal injury is delayed based on the severity of the exposure. Parenchymal injury also leads to alveolar collapse, which can further be complicated by pneumonia. Pneumonia pathogenesis is not solely due to obstruction but also to dysfunction of immune cells induced by inhaled smoke. (3)

Systemic toxicity is determined by two gases: carbon monoxide and hydrogen cyanide. The former should be suspected and treated with supplementary high-flow oxygen in conscious patients, or with mechanical ventilation with 100% oxygen in comatose patients, until it is excluded by normal carboxyhemoglobin levels. The latter gas exposure is difficult to confirm by laboratory testing and, therefore, has been advised to treat with an antidote in the event of high suspicion. (2,4)

An important point to be borne in mind during the management of such patients is that the oximeter cannot be relied upon for saturation, as it cannot differentiate between oxyhemoglobin and carboxyhemoglobin; hence, CO oximetric measurement of oxyhemoglobin must be sought.

A chest radiograph must be obtained at presentation, but it has low sensitivity for diagnosing inhalation injury. If the radiograph shows any opacities, it indicates a severe injury and a poor prognosis. (5) Computed tomography imaging has been studied in two small populations and has been postulated to predict the severity of lung injury and total number of ventilation days by airway the wall thickness-to-diameter ratio (T/D ratio). (6,7)

Bronchoscopy can aid in diagnosis and help remove carbon soot particles from the airway to prevent progression of inflammation.

Management includes airway management, inva-

sive mechanical ventilation if profound respiratory failure, sepsis identification and treatment, bronchodilators, mucolytics, heparin, and supportive care. (2)

Conclusion

Airway Inhalation injury can lead to life-threatening consequences if not managed promptly. The initial presentation could be misleading, and hence, monitoring for 24–48 hours is crucial in management. Manifestations of the injury can range from mild symptoms to life-threatening respiratory dysfunction and sepsis. Multidisciplinary management of the condition is the cornerstone of the treatment.

Figure 1. Chest X-ray showing right lower zone collapse and consolidation

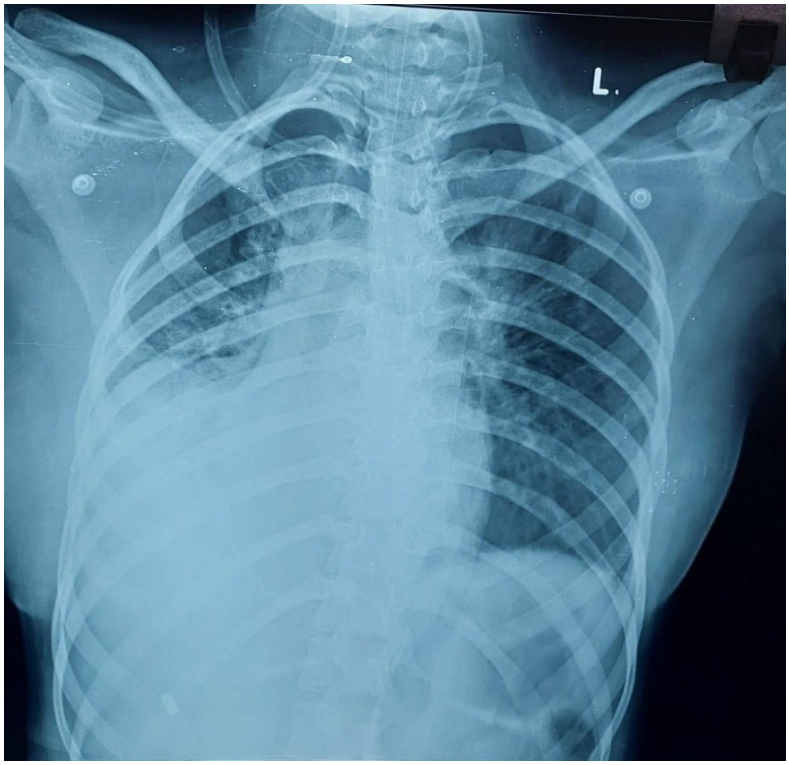

Figure 2. Soot particles that were seen in the bronchoscopy sample were collected in the container

Figure 3. Chest X-ray during follow-up showed complete resolution of parenchymal opacities

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