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A CASE OF SEVERE INHALATIONAL THERMAL INJURIES

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Abstract *Exposure of a patient to super-heated gases, incomplete combustion of toxic products should always raise suspicion of airway burns. A meticulous history including temperature, duration of exposure and composition of the inhaled gases is of paramount importance. The goals of initial management of the airway burns should be to protect the airway patency and prevention of associated hypoxic damage.*

DL Direct Laryngoscopy VAP- Ventilator Associated Pneumonia; BAL-Broncho Alveolar Lavage; FOB- Fiber Optic Bronchoscopy; FASTHUG- Feeding, Analgesia, Sedation, Thrombo-prophylaxis, Ulcer-prophylaxis, Glycemic control; CO Carbon monoxide.

Introduction

Diagnosing airway burn depends on high degree of suspicion, clinical examination, and laboratory testing. Ominous signs of burns including but not restricted to darkened or reddened oral and/or nasal mucosa, singed eyebrows or nasal hairs, hoarse voice should alert against a possible airway involvement. Progressive difficulty in securing the airway of burns patients is mentioned in literature hence an aggressive approach is needed to decrease failure. An early tracheostomy in such patients decreases need of sedation, while providing better toileting of the respiratory tract.

I. Case

The present case is an adult male who sustained burn injury while igniting the paper products using an inflammable liquid in a high altitude area with pre-existing hypoxic conditions.Initial examination revealed only superficial burn wounds (Fig.1). Other than the hoarseness of voice there were no ominous signs. Since there was a history of inhalation of hot fumes and progressive hoarseness of voice DL was performed which showed the evidence of supra-Glottic edema. In view of these findings, we secured the airway using surgical tracheostomy under General anaesthesia. Peri operative period was uneventful, and patient was shifted to the ICU on mechanical ventilator with lung protective strategies. We started with 6 hourly nebulization of bronchodilators and intravenous antibiotics post-operatively. Following first 48 hours the patient developed tachypnea, ventilator asynchrony and fall in saturation. A diagnostic fiber FOB revealed crusting of the entire airway up to tertiary bronchioles. (Fig. 2,3,4,5,)

The patient was started on nebulization with Colistin and LMWH, bronchodilators. BAL was performed.

48 hourly for next 10 days while following FASTHUG [1] and VAP bundles [2]. After a period of 07 days and successful weaning trial patient was shifted to ward on 10th day post injury. (Fig 9)

PICTORIAL DEPICTION OF SEQUENCE OF EVENTS

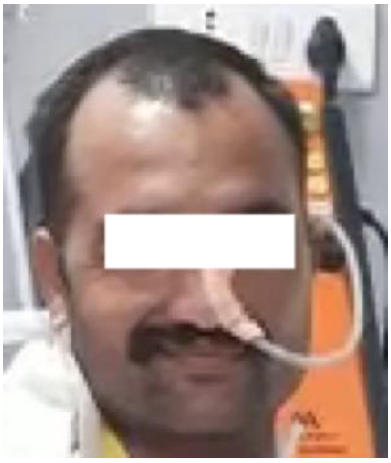

Fig 1: Initial presentation

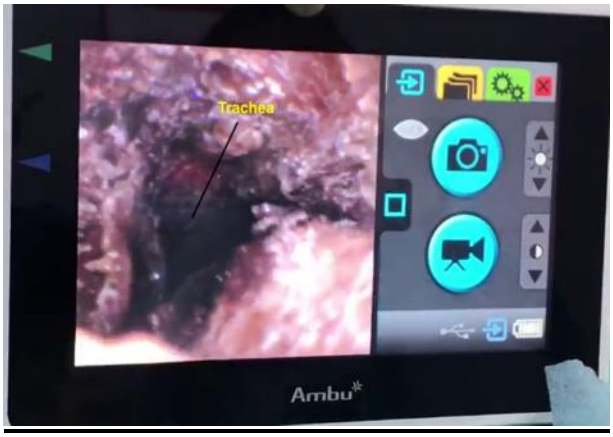

Fig 2. (Burnt Trachea)

Fig 3: Rt.& Lt Main Bronchus

Fig 4: Carina

Fig 5: Right. Secondary Bronchus

Fig 6: Left. Secondary Bronchus

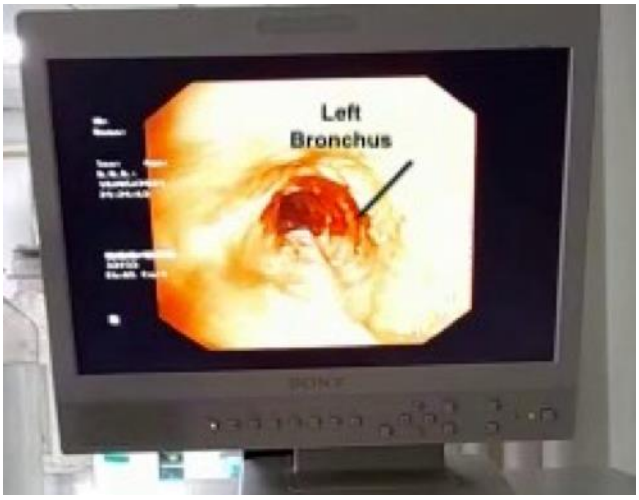

Fig 7. Cleared Left Bronchus

Fig 8. Cleared Right Bronchus

Fig 9 Patient shifted to ward

The case we present is an interesting since he had virtually no involvement of any major facial burns. FOB showed profound airway burns hence we recommend use 48-hourly bronchoscopy mandatory in all airway burns.

I. Discussion

AETIOLOGY OF ARIWAY BURNS

Gaseous compounds [5]

Carbon monoxide: CO reduces the oxygen carrying capacity of blood resulting in tissue hypoxia even though the PaO_2 values remain within normal ranges by various mechanisms including but not restricted to following [6]

(i) Reduced oxygen carrying capacity of blood due to high affinity to haemoglobin.

Leftward shift of the ODC

Binding of CO to cytochrome A_3

Binding to myoglobin, causing myocardial and skeletal muscle dysfunction.

Sulphur dioxide: When oxidized it forms strong acids which causes lower-airways injury

Nitrogen di oxide: It has limited solubility in water but can be carried by particles leading to damage of respiratory epithelial membranes.

Chlorine: Once dissolved in water on the mucosal surface, it forms strong acids.

Particulate matter [7]

Partially combusted particulate matter is a source of infection while causing physical obstruction to the airway

Severe inflammatory response [8]

Following the initial insult there is release of potent inflammatory mediators including but not restricted to free radicals, super oxide ions, interleukins, histamine, prostaglandin, bradykinin. IL-1, IL-6 and TNF which cause peroxidation of the outer lipid bilayer of the cells leading to apoptosis.

Airway injury above the larynx

- The damage to the respiratory mucosa is due to Heat, Oxygen deficiency and toxins.

Airway injury below the larynx

- ***Chemical trachea-bronchitis****:* The inhalation of incomplete products of combustion leads to pulmonary inflammatory response. Necrotic cell debris, particulate matter and proteinaceous fluid accumulate in bronchial tree, leading to cast formation and atelectasis.

II. Conclusion

We recommend that apart from initial history taking and physical examination of the patient an early DL is strongly recommended for the assessment of the airway. In the present case, we realized that the absence of facial burns doesn't rule out the possibility of airway involvement. An early fiber optic bronchoscopy is mandatory for assessment of the infra Glottic structures in all the burn patients who are on mechanical ventilator.

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