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BURN INHALATION INJURY TREATMENT

Evidence Based Medicine Guideline

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SUMMARY

The presence of facial burns, soot to face or airways, hoarse voice, cough, difficulty managing respiratory secretions, altered level of consciousness, and increased work of breathing all support a diagnosis of smoke inhalation injury. Toxic chemical airway injury can result from exposure to carbon monoxide and is treated with administration of 100% oxygen. Cyanide toxicity may also be suspected in patients found unresponsive at the scene or with altered level of consciousness or hemodynamically instability. This can be treated presumptively with administration of hydroxocobalamin at the scene or in the emergency department. The diagnosis of burn inhalation injury is based on clinical findings and direct airway visualization via bronchoscopy performed within 4 hours of admission. When inhalation injury is detected, aggressive pulmonary hygiene, airway protection, and ventilatory support are required and burn inhalation treatment including aerosolized heparin, N-acetylcysteine, and albuterol should be initiated and continued for 7 days post-inhalation injury.

RECOMMENDATIONS

- Level 1
 - > None
- Level 2
- None
- Level 3
 - In patients with suspected inhalational injuries, bronchoscopy should be performed within 24 hours of admission
 - The use of aerosolized heparin, N-acetylcysteine, and albuterol significantly improves survival in burn inhalation injury
 - ➤ Hydroxycobalamin (Cyanokit[™]) should be considered in burn injuries sustained in an enclosed space with suspected cyanide toxicity AND one or more of the following criteria:
 - Hypotension without clear etiology
 - Altered mental status or seizure
 - Cardiopulmonary arrest
 - Patients with confirmed smoke inhalation injury and any of the following factors should receive 3 days of inhaled tobramycin (300 mg nebulized twice daily), starting on day 3 of mechanical ventilation (if expected to remain intubated):
 - Age > 60 years
 - TBSA > 20%
 - Initial PaO₂/FiO₂ < 300</p>
 - Initial carboxyhemoglobin >10%

INTRODUCTION

Burn inhalation injury can result from inhalation of smoke, steam, hot gases, or liquids. These substances may contain both superheated and toxic products of incomplete combustion. Such injuries may occur either supraglottic

LEVEL OF RECOMMENDATION DEFINITIONS

- Level 1: Supported by multiple, prospective randomized clinical trials or strong prospective, non-randomized evidence if randomized testing is inappropriate.
- Level 2: Supported by prospective data or a preponderance of strong retrospective evidence.
- Level 3: Supported by retrospective data or expert opinion.

DISCLAIMER: These guidelines were prepared by the Department of Surgical Education, Orlando Regional Medical Center. They are intended to serve as a general statement regarding appropriate patient care practices based on the medical literature and clinical expertise at the time of development. They should not be considered to be accepted protocol or policy, nor are intended to replace clinical judgment or dictate care of individual patients.

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or subglottic. Thermal injury is typically isolated to the upper airway due to reflex closure of the larynx and can cause significant airway swelling and obstruction (1,2). Lower airway injuries result from prolonged exposure to smoke. Chemical inhalation injuries can affect the entire respiratory tract leading to damage of epithelial and capillary endothelial cells. Intubation should be performed in patients with respiratory distress or concern for airway edema, as swelling can worsen during fluid resuscitation (3).

INITIAL EVALUATION

Upon arrival to a trauma/burn center, patients with burn injuries should be screened for inhalation injury. Physical examination should include evaluation for signs of respiratory distress, stridor, voice changes, nasal/oral soot, singed facial hair, significant facial burns, and edema. In suspected inhalation injury, patients should be placed on 100% oxygen by non-rebreather mask and, if indicated, intubated for airway protection. If burn inhalation is suspected, bronchoscopy should be performed upon admission during the initial evaluation and the degree of injury should be noted using the inhalation injury grading system (Figure 1). Though no universally accepted grading system exists for smoke inhalation injuries, it is generally accepted that bronchoscopic findings of erythema, edema, soot, and tissue damage support the presence and indicate the severity of inhalation injury. Findings on chest radiography are often delayed, however, admission chest x-ray is important to establish a baseline for comparison and potential diagnosis of underlying conditions (1,4,5).

Grade	Class	Description
0	No Injury	Absence of carbonaceous deposits, erythema, edema, bronchorrhea, or obstruction
1	Mild Injury	Minor or patchy areas of carbonaceous deposits, erythema, edema, bronchorrhea, or obstruction
2	Moderate Injury	Moderate degree of carbonaceous deposits, erythema, edema, bronchorrhea, or obstruction
3	Severe Injury	Severe inflammation with friability, copious carbonaceous deposits, bronchorrhea or obstruction
4	Massive Injury	Evidence of extensive mucosal sloughing tissue necrosis, endoluminal obstruction

Figure 1: Inhalation Grading System (4,5)

CARBON MONOXIDE POISONING

Patients should be suspected of suffering inhalation injury if the fire occurred in an enclosed space, such as an automobile or building. Findings of soot on the face, in the mouth, or in the nose support exposure to smoke. Upon initial evaluation of patients suspected of inhalation injury, a co-oximetry arterial blood gas (ABG) with carboxyhemoglobin measurement should be performed to evaluate for elevated carbon monoxide levels. The affinity of carbon monoxide for hemoglobin is 200 times greater than that of oxygen, reducing hemoglobin oxygen carrying capacity and decreasing oxygen delivery to tissues. Patients with carbon monoxide poisoning may present with symptoms of poor oxygenation (headache, dizziness, nausea, altered mental status), but normal pulse oximetry. Treatment for carbon monoxide poisoning is mainly supportive with administration of 100% oxygen by non-rebreather mask to reduce the degree of carboxyhemoglobin and promote excretion of carbon monoxide (1-3). Hyperbaric oxygen therapy has been shown to reduce the risk of long-term cognitive complications. However, there is uncertain availability of emergent hyperbaric therapy and significant risks involved in placing critically ill burn patients in a chamber during acute resuscitation (6).

CYANIDE TOXICITY

Cyanide toxicity results from inhibition of the cytochrome oxidase system (6). Smoke from house fires can place patients at risk for exposure to cyanide, produced from fumes of a variety of household items including plastics. Fires within cars, recreational vehicles, boats, airplanes, and industrial spaces may have a higher propensity for

cyanide toxicity due to the prevalence of plastic. In cases of suspected cyanide toxicity, the use of intravenous hydroxycobalamin (Cyanokit[®]) is indicated. Treatment should be initiated based on clinical suspicion for suspected cyanide exposure and hemodynamic instability (unrelated to another cause), altered mental status, seizure, respiratory or cardiac arrest (2,6).

The starting dose of hydroxycobalamin for adults is 5 grams administered by IV infusion over 15 minutes. A second dose of 5 grams IV may be administered depending upon the response and clinical severity of the poisoning (7). Acute renal failure with acute tubular necrosis, renal impairment, and calcium oxalate crystals are potential side effects of hydroxocobalamin. Therefore, if patients have received hydroxycobalamin, do not administer ascorbic acid infusions or low dose ascorbic acid, as patients are at a higher risk for calcium oxalate nephropathy (7,8).

TREATMENT

The inflammatory response initiated by inhalation airway injury is theorized to propagate endothelial damage and vascular permeability leading to release of coagulation factors. This response is supplemented by extrinsic fibrin formation and deposition in the alveoli, promoting cast formation and shunting.

Burn inhalation treatments should be implemented in patients with documented inhalational injuries. Various studies have been performed to evaluate treatment regimens. The use of aerosolized heparin, N- acetylcysteine, and albuterol have been shown to significantly improve survival (9,10). The following burn injury inhalation protocol is recommended:

- N-acetylcysteine (20% aerosolized solution) 3 ml
- Heparin aerosolized solution (10,000 unit(s)/mL) 1 ml
- Albuterol sulfate aerosolized solution (2.5 mg/0.5 mL) 0.5 ml

All three medications are combined by respiratory therapy and administered by inhalation treatment every four hours for the first seven days post-inhalation injury. This inhalation protocol continues even after extubation in patients who initially required mechanical ventilation (9,10).

VENTILLATOR-ASSOCIATED PNEUMONIA PROPHYLAXIS

Inhalation injuries complicated by pneumonia lead to increased mortality. A retrospective analysis of 129 patients treated for inhalation injury over a 10-year timeframe was conducted to identify factors that contribute significantly to the development of pneumonia. The 5 factors independently associated with development of pneumonia were: age > 60 years, TBSA > 20%, bronchoscope grade 3 or 4 injury, initial $PaO_2/FiO_2 < 300$, and initial carboxyhemoglobin level > 10%. A predictive scoring system was proposed with each factor assigned a score of 1. Patients with a score > 2 were deemed at high risk of developing pneumonia (11). The incidence of ventilator-associated pneumonia peaks at day 7 of intubation

A multi-center, double-blind, randomized, controlled study conducted in France sought to evaluate the impact of prophylactic use of inhaled amikacin on the development of ventilator-associated pneumonia in patients who had undergone invasive mechanical support for at least 72 hours. A total of 847 critically ill patients (417 assigned to amikacin group, and 430 to the placebo group) were included in the analysis. The amikacin group received nebulized amikacin daily for 3 days (starting 72 hours after ventilation), and the primary outcome was first episode of ventilator-associated pneumonia during 28 days of follow-up. The amikacin group saw significantly less infection-related ventilator-associated complications compared to the placebo group (18% vs. 26% [hazard ratio 0.66; 95% CI, 0.50 to 0.89]). Additionally, there were no differences in the measured safety outcomes between the 2 groups (12). While the trial used inhaled amikacin, inhaled tobramycin (300 mg nebulized twice daily) has a similar safety/efficacy profile and is the preferred inhaled aminoglycoside at this institution. The process of using inhaled aminoglycosides in select patients at high risk of developing pneumonia following smoke inhalation injury, offers a low-risk intervention with the potential to significantly reduce the development of pneumonia in these patients (11,12).

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