Recent Updates in Management of Smoke Inhalation Injury in Burn Victims

An Essay

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Abbreviations

ABCDEF....Airway, breathing, circulation, disability, exposure, and fluid resuscitation.

ABG ..... Arterial blood gases

AIS ..... Abbreviated injury score

ALI..... Acute Lung Injury

APRV..... Airway Pressure Release Ventilation

AT ..... Antithrombin

ATLS..... Advanced Trauma Life Support

ARDS ..... Acute respiratory distress syndrome

Arg ..... Argenine

BUN..... Blood urea nitrogen

BWT..... Bronchial wall thickness

CBC..... Complete blood count

CK ..... Creatine kinase

CN ..... Cyanide

CNCob ..... Cyanocobalamin

CNS ..... Central nervous system

CO ..... Carbon monoxide.

COHb..... Carboxyhemoglobin

COPD..... Chronic obstructive pulmonary disease

CT ..... Computed tomography.

ECG ..... Electrocardiogram

ETT ..... Endotracheal tube

Fe2+ ..... Ferrous ion

Fe3+ ..... Ferric ion

FiO_2 ..... Fraction of inspired oxygen.

FOB..... Fibro optic bronchoscopy

GCS..... Glasgow Coma Scale

HBO ..... Hyperbaric oxygen

HCN ..... Hydrogen cyanide.

HFPV ..... High frequency percussive ventilation

HTV ..... High tidal volume ventilation

IDGC/MS .. Isotope-dilution gas chromatography-mass spectrometry

ICAM-1..... Intercellular adhesion molecule 1

IL..... Interleukin

LTV..... Low tidal volume ventilation

MI..... Myocardial infarction

NAC .....N-acetylcysteine

NO ..... Nitric oxide

NO_2^- ..... Nitrate

NO_3^- ..... Nitrite

NBO ..... Normobaric oxygen

NOS..... Nitric oxide synthase

NF-κB ..... Nuclear factor κB

nNOS..... Neuronal Nitric oxide synthase

iNOS..... Inducible Nitric oxide synthase.

eNOS..... Endothelial Nitric oxide synthase.

O_2^- ..... Superoxide

OHCob..... Hydroxycobalamin

ONOO- ..... Peroxynitrite

PaO_2 ..... Arterial partial pressure of oxygen

PAF ..... Platelet activating factor

PARs ..... Protease activated receptors

PARP ..... Poly (ADP ribose) polymerase

PEEP..... Positive end expiratory pressure

PEFR..... Peak expiratory flow rate

RADS ..... Highest radiologist's score

RhAT ..... Recombinant human antithrombin

RNS ..... Reactive Nitrogen Species

ROS..... Reactive Oxygen Species

SaO_2 ..... Oxyhemoglobin saturation.

SII..... Smoke Inhalation Injury

SpO_2 ..... Pulse Oximeter Oxygen Saturation

TNF alpha . Tumor necrosis factor alpha

UAO ..... Upper Airway Obstruction

VCAM-1 .... Vascular cell adhesion molecule 1

VDR ..... Volumetric diffusive respiratory mode

VILI ..... Ventilator-induced lung injury

V/Q..... Ventilation perfusion

4-DMAP 4-dimethylaminophenol

INTRODUCTION

Lung injury resulting from inhalation of smoke or chemical products of combustion continues to be associated with significant morbidity and mortality **(Dries, 2013)**.

Combined with cutaneous burns, inhalation injury increases fluid resuscitation requirements, incidence of pulmonary complications and overall mortality of thermal injury **(Palmier, 2007)**.

Smoke is heterogeneous and unique to each fire; it comprises particulates, respiratory irritants, and systemic toxins as well as heat, all contributing to the pathological insult. Thermal injury below vocal cords is rare because of the effective heat dissipation in the upper airway. Particulate matter is the chief contributor to the pathophysiology of smoke inhalation injury leading to activation of cascade of inflammatory mediators leading to pulmonary edema, mucosal sloughing, cast formation, airway obstruction and ventilation/perfusion mismatch. Systemic toxicity may occur with inhalation of carbon monoxide or cyanide **(Toon, 2010)**.

A variety of factors explain slower progress for improvement in management of inhalation injury. Burned cutaneous tissue may be excised and replaced with skin grafts, but injured pulmonary tissue must merely be

supported and protected from secondary injury. The critically ill burn patient has multiple mechanisms in addition to smoke inhalation that may contribute to lung injury such as sepsis, ventilator-induced lung injury (VILI) or systemic inflammation in response to burns. Thus, inhalation injury has a significant effect on burn patient outcome but is difficult to separate from the contribution of other mechanisms which also affect the lungs **(Sheridan, 2012).**

AIM OF THE ESSAY

This essay aims to highlight smoke inhalation injury and recent updates in its management hoping to improve the outcome of burn victims with smoke inhalation injury.

PATHOPHYSIOLOGY OF SMOKE INHALATION INJURY

Fire victims may be exposed to three potential injuries: burns, trauma and smoke inhalation. However, 60–80% of deaths at the fire scene are attributable to smoke inhalation. Smoke is a heterogeneous compound unique to each fire in both its chemical composition and its toxic features **(Anseeuw et al., 2013).**

Inhalation injury can result from direct local thermal & chemical exposures, immune responses to these factors, systemic effects of inhaled toxins, accrual of endobronchial debris, and secondary infection. Structural fires generate smoke that contains a large variety of chemicals, products of incomplete combustion, and aerosolized debris of widely varying particle sizes **(Sheridan, 2016)**.

Smoke is composed of a gas phase and a particle phase. Particle size and tidal volume determine their distribution in the lung. Physiologically the nasopharynx clears the inspiratory air of the majority of particles with a diameter larger than 5 μm. During a fire, however, victims (both conscious and unconscious) breathe through the mouth owing to nasopharyngeal irritation. As a result, particle deposition in the airway is much greater, causing progressive

cellular injury and severe lung injury. The gas phase causes predominantly proximal airway and local damage, although some long- acting oxidants are able to reach distal lung tissues **(Rehberg et al., 2009)**.

The chief contributor to the pathophysiology of smoke inhalation is particulate matter. Carbonaceous particles (soot) impregnated with a variety of toxins reach the alveolar level suspended in air **(Toon et al., 2010).**

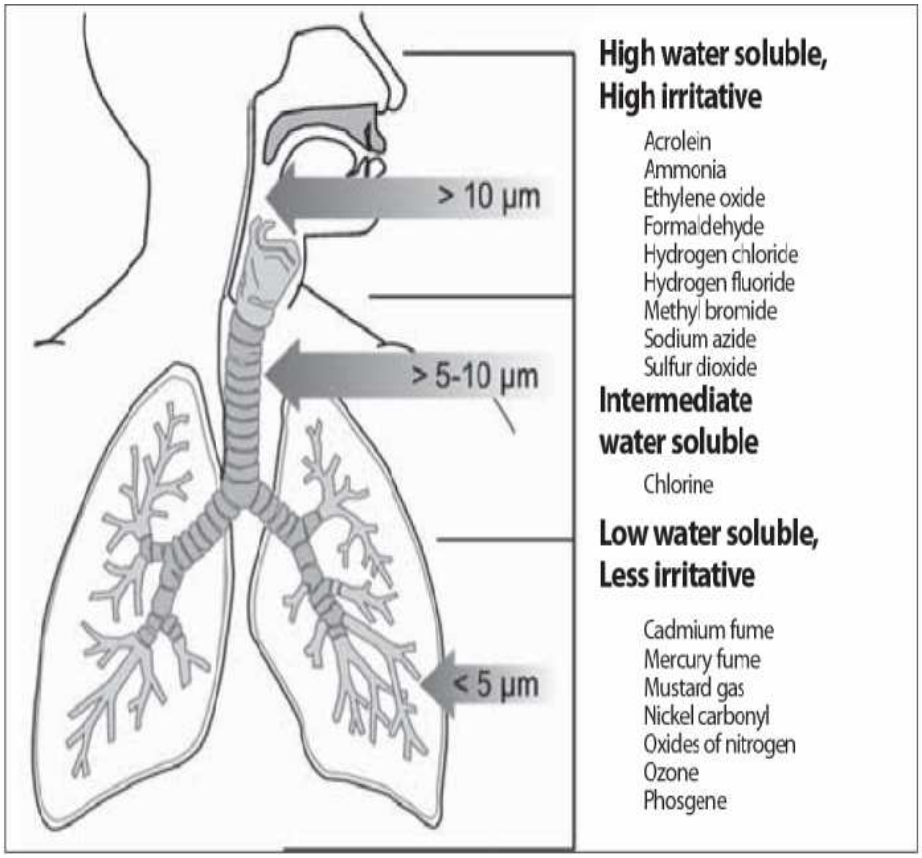

Figure (1): Distribution of the irritant gases and the site of injury in the respiratory Tract according to their particle size and water solubility **(Gorguner et al., 2010)**.