

Recent Updates in Management of Smoke Inhalation Injury in Burn Victims

An Essay

Submitted for Partial Fulfillment of Master Degree
in General Intensive Care

By

Yasmine Ashraf Ibrahim ElSharnoby
MB.B.CH

Supervised by

Prof. Dr. Raouf Ramzy Gad Allah

*Professor of Anesthesia and Intensive Care
Faculty of Medicine, Ain Shams University*

Dr. Mayar Hassan Elseri

*Assistant Professor of Anesthesia and Intensive Care
Faculty of Medicine, Ain Shams University*

**Faculty of Medicine
Ain Shams University
2017**



First of all, thanks to **Allah** for helping and guiding me in accomplishing this work and for everything else I have.

Words are not sufficient to express my sincerest appreciation and my deepest gratitude to **Prof. Dr. Raouf Ramzy Gad Allah**, Professor of Anesthesia and Intensive Care, Faculty of Medicine, Ain Shams University, for his continuous encouragement, and his precious remarks which guided me to present this work in its proper way, it was indeed an honor to have been supervised by him.

I would like to thank **Dr. Mayar Hassan Elseri**, Assistant Professor of Anesthesia and Intensive Care, Faculty of Medicine, Ain Shams University, for her guidance and suggestions which were of great value to me.

An endless thanks for my family for their support without it, I would never completed this work.

Contents

Page No.

• List of figures	I
• List of Tables	IV
• Abbreviations	V
• Introduction	1
• Aim of the essay	3
• Pathophysiology of smoke inhalation injury	4
• Diagnosis and grading of inhalation injury ...	30
• Complications of inhalation injury	64
• Management of inhalation injury and its updates	73
• Summary	106
• References	109
• Arabic summary	—

LIST OF FIGURES

<i>Fig. No.</i>	<i>Title</i>	<i>Page No.</i>
Figure (1):	Distribution of the irritant gases and the site of injury in the respiratory Tract according to their particle size and water solubility	5
Figure (2):	The Pathogenesis of Inhalation Injury	7
Figure (3):	Temperature distribution in the airway for breathing rate of 20 l/min and inlet temperature of 300°C	10
Figure (4):	Role of nitric oxide (NO) in the pathophysiology of smoke inhalation injury ...	16
Figure (5):	Poly (ADP-ribose) polymerase (PARP) plays a role in smoke inhalation injury	18
Figure (6):	Isolated airway cast	21
Figure (7):	Pathophysiology of acute smoke inhalation injury	23
Figure (8):	Hemoglobin is convertad rapidly to carboxyhemoglobin in the presence of carbon monoxide	26
Figure (9):	Carboxyhemoglobin-induced changes in the oxygen-hemoglobin dissociation curve	27

Figure (10): Fibro-laryngoscopic observations of laryngeal burns	45
Figure (11): (A) First-degree injury. (B) Second-degree injury. Bronchial mucosa with obvious congestion and edema, petechial hemorrhage, and erosion. (C) Third-degree injury	49
Figure (12): Bronchoscopic biopsy of a 67-year-old woman with smoke inhalation injury	50
Figure (13): Example of radiologist's score findings in chest computed tomography scan slice	52
Figure (14): Diffuse Hypodensity of Cerebellum Due to Hypoxic-ischemic Injury	57
Figure (15): A 21-year-old man with inhalation burn in the tracheobronchial wall	71
Figure (16): An algorithm to manage the patients at risk for inhalation injury in the emergency room	77
Figure (17): Coagulation-dependent and coagulation-independent anti-inflammatory effects of antithrombin	92
Figure (18): An algorithm to manage the patients at risk for carbon monoxide poisoning	96

Figure (19): Prehospital algorithm. GCS, Glasgow
Coma Scale 103

Figure (20): Hospital algorithm. CO, carbon monoxide;
CN, cyanide; HOCo, hydroxocobalamin 104

LIST OF TABLES

<i>Table No.</i>	<i>Title</i>	<i>Page No.</i>
Table (1):	Risk factors of inhalation injury	33
Table (2):	Bronchoscopic criteria used to grade inhalation injury	48
Table (3):	Radiologist's scoring table (RADS score) for inhalation injury	51

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	Arginine
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
FiO₂Fraction of inspired oxygen.
FOB.....Fibro optic bronchoscopy
GCS.....Glasgow Coma Scale
HBOHyperbaric oxygen
HCNHydrogen cyanide.
HFPVHigh frequency percussive ventilation
HTVHigh 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
NACN-acetylcysteine
NONitric oxide
NO₂⁻Nitrate
NO₃⁻Nitrite
NBONormobaric oxygen
NOS.....Nitric oxide synthase
NF-κBNuclear factor κB
nNOS.....Neuronal Nitric oxide synthase
iNOS.....Inducible Nitric oxide synthase.
eNOSEndothelial Nitric oxide synthase.

O₂⁻Superoxide
OHCo_b.....Hydroxycobalamin
ONOO-Peroxynitrite
PaO₂Arterial partial pressure of oxygen
PAFPlatelet activating factor
PARsProtease activated receptors
PARPPoly (ADP ribose) polymerase
PEEPPositive end expiratory pressure
PEFR.....Peak expiratory flow rate
RADSHighest radiologist's score
RhATRecombinant human antithrombin
RNSReactive Nitrogen Species
ROS.....Reactive Oxygen Species
SaO₂Oxyhemoglobin saturation.
SII.....Smoke Inhalation Injury
SpO₂Pulse Oximeter Oxygen Saturation
TNF alpha .Tumor necrosis factor alpha
UAOUpper Airway Obstruction
VCAM-1Vascular cell adhesion molecule 1
VDRVolumetric diffusive respiratory mode
VILIVentilator-induced lung injury
V/Q.....Ventilation perfusion
4-DMAP4-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

Introduction

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**).

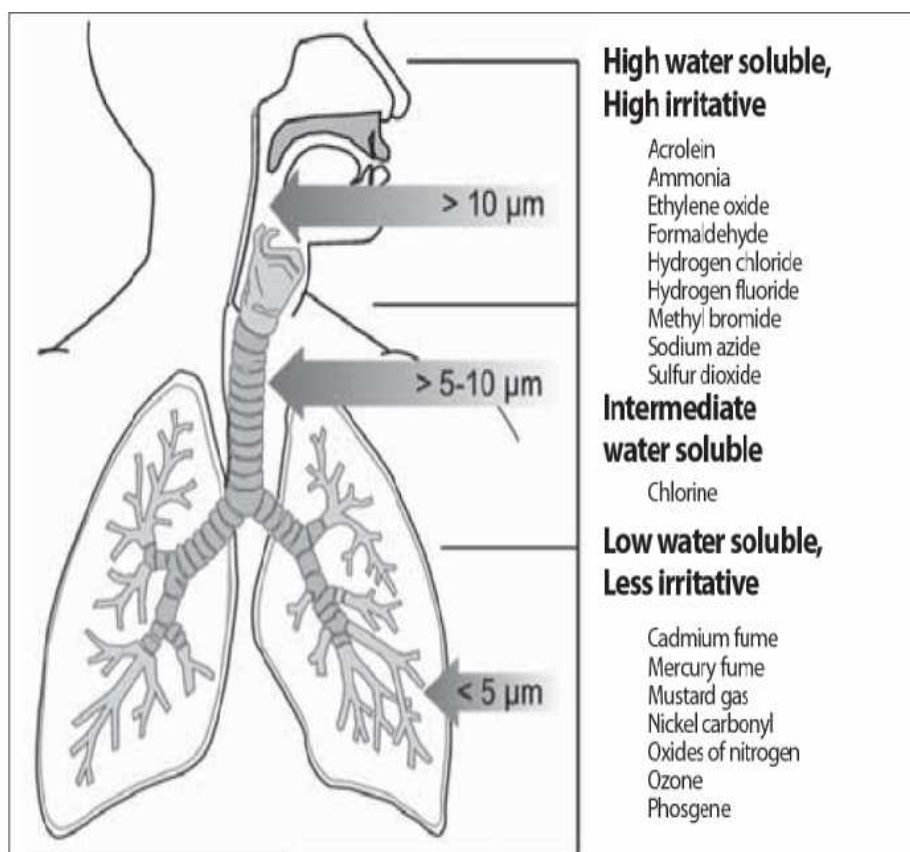


Figure (1): Distribution of the irritant gases and the site of injury in the respiratory Tract according to their particle size and water solubility (**Gorguner et al., 2010**).