

Correlation of Bronchoscopic Score and Fluid Requirements in Burn Patients with Inhalation Injury

Thesis

*Submitted for Partial Fulfillment of M.D. Degree
in Plastic & Reconstructive Surgery*

By

Ebrahim Mohammed Amin Abdel Gwad
M.B.B.CH., M.Sc.

Under Supervision of

Prof. Ayman Abo El Makarem Shaker

*Prof. of Plastic & Reconstructive Surgery
Faculty of Medicine – Ain Shams University*

Prof. Sahar Kamal Mohammed Abo EL Ela

*Prof. of Anesthesia and Intensive Care Unit
Faculty of Medicine – Ain Shams University*

Dr. Karim Khalil EL Lamie

*Ass. Prof. of Plastic & Reconstructive Surgery
Faculty of Medicine – Ain Shams University*

Dr. Ahmed Fathi El Sherif

*Ass. Prof. of Plastic & Reconstructive Surgery
Faculty of Medicine – Ain Shams University*

**Faculty of Medicine
Ain Shams University
2015**

Contents

Page No.

List of Tables	I
List of Figures	III
List of Abbreviations	V
Introduction	1
Aim of the Work	4
Review of Literature:	
• Pathophysiology of thermal injury	5
• Airway management	36
• Burn shock resuscitation	50
Patients and Methods	69
Results	79
Discussion	96
Summary and conclusion	108
References	110
Arabic Summary	—

List of Tables

<i>Table No.</i>	<i>Title</i>	<i>Page No.</i>
1	Systemic response to thermal injury	5
2	Origin of selected toxin compounds	30
3	Bronchoscopic criteria used to grade inhalation injury	34
4	Different types of fluid used in burn resuscitation	51
5	Different resuscitation formula used in burn management	53
6	Traditional criteria for adequate fluid resuscitation.	62
7	Modern resuscitation endpoints	64
8	Selected characteristics of the study participants	79
9	Gender and degree of inhalation burns in the study participants	80
10	Amount of fluid expected to be given by Parkland formula and actual fluid given in the 1 st 48 hours	83
11	Comparison between the degree of inhalation burn and selected demographic and clinical data	84

12	The number of predictor used in each of the model to determine the fluid volume in the first day with the F ratio and statistical significance.	85
13	Coefficient of determination of each of the used models for predicting the needed fluid volume in the first day with the statistical significance of the change of each model from the first model with only one predictor	87
14	Regression coefficients of the five models used to predict the volume of fluids needed in the first day	89
15	The number of predictor used in each of the model to determine the fluid volume in the second day day with the F ratio and statistical significance	91
16	Coefficient of determination of each of the used models for predicting the needed fluid volume in the second day with the statistical significance of the change of each model from the first model with only one predictor	91
17	Regression coefficients of the five models used to predict the volume of fluids needed in the second day	92

List of Figures

<i>Figure No.</i>	<i>Title</i>	<i>Page No.</i>
1	Immune response to thermal burn injury.	8
2	Hemoglobin is converted rapidly to carboxyhemoglobin in the presence of carbon monoxide	27
3	Carboxyhemoglobin-induced changes in the oxygen-hemoglobin dissociation curve.	28
4	Bronchoscope and bronchoscopy equipment.	32
5	Protocol for fluid resuscitation of adult burn patients	61
6	a) Bronchoscope (Pentax Medical Company), Burn unit, Ain shams university b) Power supply of bronchoscopy (Pentax Medical Company), Burn unit, Ain shams university c) Cleaning brushes for endoscope channel (Pentax Medical Company), Burn unit, Ain shams university d) Plastic connector Burn unit, Ain shams university	73
7	Bronchoscopic finding of 28 years old male patient 20% TBSA burn showing erythema and secretion (right side) suggestive of 1 st degree inhalation injury. Burn unit, Ain Shams University.	81

8	Bronchoscopic finding of 23 years old male patient 28% TBSA burn showing erythema and secretion soot and pitecheal hemorrhage suggestive of 2nd degree inhalation injury. Burn unit, Ain Shams University.	81
9	Bronchoscopic finding showing charring of respiratory mucosa, soot, erythema and secretion suggestive of 3 rd degree inhalation injury.	82
10	Bronchoscopic finding showing charring of respiratory mucosa, multiple ulcerations, soot, bleeding and blood clots suggestive of 4th degree inhalation injury.	82
11	Follow up bronchoscopic finding of 28 years old male patient 20% TBSA burn previously diagnosed as had 1 st degree inhalation injury show decrease in the amount of secretion and relieve of hyperemia with restoration of normal appearance of respiratory mucosa. Burn unit, Ain Shams University.	94
12	Follow up bronchoscopic finding of 20years old female patient 20% TBSA burn previously diagnosed as had 2 nd degree inhalation injury show persistance of secretion and hyperemia with new appearance of mucus casts. Burn unit, Ain Shams University.	94

List of Abbreviations

ABG	Arterial Blood Gases
AIS	Abbreviated Injury Score
ARDS	Acute Respiratory Distress Syndrome
ARF	Acute Renal Failure
BAL	Bronchoalveolar Lavage
CBC	Complete Blood Count
CO	Carbon Monoxide
COX	Cyclo-oxygenase Enzyme
CT scan	Computed Tomography scan
ER	Emergency Room
GFR	Glomerular Filtration Rate
GSH	Glutathione
HbC	Carboxy hemoglobin
I:E	Inspiratory/Expiratory
IL-1	Interleukin 1
IL-6	Interleukin-6
iNOS	Inducible Nitric Oxide Synthase
ITBV	Intrathoracic Blood Volume
NIH	National Institute of Health
NO	Nitric Oxide

OR	Operative Room
PEEP	Positive End Expiratory Pressure
PGE2	Prostaglandin E2
TBSA	Total Body Surface Area
Th-2	T helper cell
TNF	Tumor Necrosis Factor
UAO	Upper Airway Obstruction
VILI	Ventilator Induced Lung Injury

INTRODUCTION

Inhalation injuries contribute significantly to the morbidity and mortality of both burned children and adults (**Palmieri, 2009**).

As the lungs are believed to be the first organ to undergo failure in dying burn patients (**Carr et al., 2009**), it is not surprising that this have been extensively investigated, not only in animal models but also in clinical studies. Despite the determinants of inhalation injury the most important pathophysiology of this increased mortality has not been well explained. Many possible mechanisms have been discussed in the literature (**Yen et al., 2000**).

It was thought that inhalation of burn products, as well as respiratory tract thermal injury can impair surfactant production. This can lead to a pro-inflammatory cytokine cascade, resulting in altered integrity of the capillary membrane and activation of oxygen radicals and cytokines. Moreover, inhalation of smoke can release thromboxane, a vasoconstrictor, which can increase pulmonary artery pressure (**Reper et al., 2002**).

Inhalation injury is diagnosed according to the following three aspects. The first is the **history** (i.e., burn in a closed space). The second is **clinical finding** (i.e., burn of the face, carbonaceous sputum and others). The last one is **laboratory**

and imaging results (including arterial blood gas analysis, chest X-ray or computed tomography (CT) and bronchoscopic examination) **(Yang, 1995).**

Bronchoscopy is the standard criterion for diagnosis of smoke inhalation injury. This procedure examines the airways from the oropharynx to the lobar bronchi. Erythema, charring, deposition of soot, edema, and/or mucosal ulceration may be present. Impending airway obstruction may be inferred, and intubation may be facilitated by this technique. Diagnostic accuracy is reported to be 86%. Studies have shown up to a 96% correlation between bronchoscopic findings and the triad of closed-space smoke exposure, carboxy hemoglobin (HbC) levels of 10% or greater and carbonaceous sputum **(Koljonen et al., 2007)**.

After inhalation injury, there is a marked increase in bronchial blood flow, which results in pulmonary edema. In a bovine smoke inhalation model, airway blood flow increases eight fold or more in the main stem bronchi after the injury, whereas cardiac output, and thus blood flow to the peripheral tissues, remains relatively unchanged **(Abdi et al., 1991)**. Bronchial blood flow enters into the pulmonary vasculature through various bronchopulmonary anastomoses. It has been suggested that the bronchial circulation plays a significant role in the spread of injury from the airway to the parenchyma. Investigation of the effect of bronchial artery ligation or ethanol injection after inhalation injury in sheep demonstrated a

decrease in gas exchange [$\text{PaO}_2/\text{FiO}_2$ (P/F) ratio], an increase in lung lymph flow, and the lung wet/dry weight ratio were all improved by these bronchial artery occlusion techniques (**Sakurai et al., 1998**). Therefore, the conclusion that the bronchial circulation contributes to edema formation in the lung that occurs after acute lung injury caused by smoke inhalation injury (**Efimova et al., 2000**).

Scheulen and Munster in 1982 found a 37 percent increase in fluid requirements when inhalation injury was present; **Paul et al., in 1982** found that fluid requirement in patients with inhalation injury was 5.76 ml/kg per percentage of total body surface area burned (TBSA) (about 44percent increase above the usual formula), whereas patients without pulmonary damage required fluid volumes almost exactly equal to the guidelines of the Parkland formula (4 ml Ringer's lactate solution per kilogram of body weight per percentage of TBSA burned), however, no one has proposed a specific modification of resuscitation formulas to accommodate the presence of inhalation injury.

Although bronchoscopic score reflect the severity of underlying inhalation injury, we could not find any study that determine the amount of increased fluid resuscitation in burned patient with inhalation injury and correlate it with the bronchoscopic findings.

AIM OF THE WORK

The aim of this study is to correlate bronchoscopic score with fluid requirements in burn patients with inhalation injury in order to decrease risk of pulmonary edema due to excess fluid intake.

(1)

PATHOPHYSIOLOGY OF THERMAL INJURY

The local and systemic inflammatory response to thermal injury is extremely complex, resulting in both local burn tissue damage and deleterious systemic effects on all other organ system distant from the burn area itself (**table 1**). Although the inflammation is initiated almost immediately after the burn injury, the systemic response progresses with time, usually peaking 5 to 7 days after the burn injury. Much of the local and certainly the majority of the distant changes are caused by inflammatory mediators (**Baris et al., 2004**).

Table (1): Systemic response to thermal injury (Baris et al., 2004)

Cardiovascular system	Excretory system	Respiratory system	Gastrointestinal system
Acute (hypovolemia) phase: • ↓ blood flow • ↓ cardiac output • ↑ capillary permeability • ↑ peripheral vascular resistance Hypermetabolic phase: • ↑ blood flow • edema formation • cardiac arrhythmias • myocardial infarction • myocardial dysfunction/cardiac instability (↑ end-diastolic volume and ↓ right ventricular ejection fraction)	Acute (hypovolemia) phase: • ↓ renal blood flow • ↓ GFR Hypermetabolic phase: • ↑ renal blood flow • ↑ GFR • impaired tubular functions • acute renal failure	• hypoxemia • pulmonary hypertension • ↑ airway resistance • ↓ pulmonary compliance	• adynamic ileus • gastric dilatation • delay in gastric emptying • gastrointestinal hemorrhage • ↑ gastric secretions • ↑ ulcer incidence • ↓ intestinal & colonic motility • ↓ mesenteric blood flow • ↓ nutrient absorption • bacterial translocation • hepatic injury

Thermal injury initiates systemic inflammatory reactions producing burn toxins and oxygen radicals and finally leads to peroxidation. The relationship between the amount of products of oxidative metabolism and natural scavengers of free radicals determines the outcome of local and distant tissue damage and further organ failure in burn injuries **(Cetinkale et al., 1997)**. The injured tissue initiates an inflammation-induced hyperdynamic, hypermetabolic state that can lead to severe progressive distant organs failure **(Baris et al., 2004)**.

Immune Response

Severe thermal injury induces an immunosuppressed state that predisposes patients to subsequent sepsis and multiple organ failure, which are the major causes of morbidity and mortality in burn patients **(Baue et al., 1998)**. A growing body of evidence suggests that the activation of a pro-inflammatory cascade after burn injury is responsible for the development of immune dysfunction, susceptibility to sepsis, and multiple organ failure **(Meakins, 1990)**. Moreover, thermal injury increases the macrophage activity, thereby increasing the productive capacity for the pro-inflammatory mediators **(MacMicking et al., 1997)**. There have been several reports indicating that circulating levels of IL-1 β , IL-6 and TNF- α are increased in patients with burn injury **(figure 1) (Yamada et al., 2000)**.

The immunological response to thermal injury is a depression in both the first and second lines of defense. The epidermis of the skin becomes damaged, allowing microbial invasion; while the coagulated skin and exudates of the patient create an ideal environment for microbial growth (**Moran and Munster, 1987**). A local burn trauma leads to neutrophil infiltration in the wound site, as well as in the remote organs, the liver and intestines (**Jahovic et al., 2004**).