Clinical and Laboratory Prognosis of Patients at Risk for Traumatic ARDS

Essay

Submitted for Partial Fulfillment of Master Degree in Intensive Care

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بسم الله الرحمن الرحيم

قالوا

سبحانك لا علم لنا
إلا ما علمتنا إنك أنت
العليم الحكيم

صدق الله العظيم

سورة البقرة الآية: ٣٢

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Mohamed Eshohady

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List of Abbreviations

Abb.	Full term
AIS	Abbreviated injury score
ALI	Acute lung injury
Ang-2	Angiopoietin-2
APACHE	Acute Physiology and Chronic Health Evaluation
APRV	Airway pressure release ventilation
ARDS	Acute respiratory distress syndrome
BAL	Broncho alveolar lavage
BALF	Broncho alveolar lavage fluid
CC-16	Clara cell protein
CMV	Cytomegalovirus
CPAP	Continuous positive airway pressure
CRP	C-reactive protein
cTnT	Cardiac troponin T
DAD	Diffuse alveolar damage
DARC	The Duffy antigen/receptor for chemokines
DcR	Decoy receptor
ECMO	Extracorporeal membrane oxygenation
ELF	Epithelial lining fluid
Foxp3	Transcription factor Forkhead box protein 3
GOCA score	Gas exchange, Organ failure, Cause, Associated disease)
HFOV	High-frequency oscillatory ventilation
I: E	Inspiratory: expiratory time ratio
ICUs	Intensive care units
IL	Interleukin
ISS	Injury severity score
KL-6	Mucin-like glycoprotein
LIS	Lung injury score
LOFS	Lung organ failure score
LOVS	The lung open ventilation study
LT	Plasma leukotriene

List of Abbreviations Cont...

Abb.	Full term
NETs	Neutrophil extracellular traps
NIH	National institutes of health
OI	Oxygenation index
PAC	Pulmonary artery catheters
PAI-1	Plasminogen activation inhibitor-1
PaO2/FiO2	Arterial oxygen partial pressure to fractional inspired oxygen
PAP	Pulmonary artery pressure
PCWP	Pulmonary capillary wedge pressure
PEEP	Positive end-expiratory pressure
RAGE	The receptor for advanced glycation end products
RBC	Red blood cells
RVSW/LVSW	Left/right ventricular stroke work
SAPS	Simplified acute physiology score
SE	Standard error
SP	Surfactant proteins
SpO2/FiO2	Oxygen saturation to fraction of inspired oxygen ratio
TBI	Traumatic brain injury
TIIP	Type II pneumocytes
TLR4	Toll-like receptor 4
tPA	Tissue plasminogen activator
Tregs	T-regulatory cells
TTS score	Triple test score
uPA	Urokinase-type plasminogen activator
VD /VT	Pulmonary dead space to tidal volume
VEGF	Vascular endothelial growth factor
VT	Tidal volume
VWF	Von Willebrand factor antigen

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INTRODUCTION

Thoracic trauma occurs in more than 50% of blunt trauma patients, pulmonary contusions are one of the most common injuries, found in 30–75% of these trauma patients.

The occurrence of pulmonary contusions was associated with higher mortality in several studies, especially because it frequently evolved to gas exchange impairment, delayed acute respiratory distress syndrome and/or multi-organ failure, the pathophysiological pathway is the activation of local and systemic inflammatory mechanisms in which innate immunity plays a key role ***(Daurat et al., 2016).***

Pulmonary contusion is well known to evolve towards respiratory failure, typically after a free interval of 24–48 h This injury primes innate immunity through enhanced reactivity of toll-like receptors 4 (TLR4), which induces exaggerated production of pro-inflammatory mediators well described in the early phase of trauma (e.g. interleukin (IL) (IL-1b, IL-6, IL-8) In the case of secondary insults such as infection or surgical aggression, so called ‘‘second hit’’, pro-inflammatory mediators induce both local infiltration of neutrophils and systemic inflammation ***(Daurat et al., 2016).***

This proinflammatory cascade leads to ARDS and multi-organ failure, which are strongly associated with increased morbidity and mortality in thoracic trauma. Accordingly, the management of trauma patients with pulmonary contusion remains challenging, especially because the initial clinical evaluation may underestimate the risk of delayed worsening ***(Godoy et al., 2016).***

It has been well demonstrated that these deleterious mechanisms may appear after a free interval of 24–48 h, consequently, in trauma patients with pulmonary contusion initial assessment may underestimate the gravity of the situation whereas respiratory status may worsen during the hours or days following admission ***(Klein, 2014).***

The main outcome criterion was the occurrence of acute respiratory distress syndrome during the first 10 days of hospitalization for 48 h or more, the international Berlin definition for acute respiratory distress syndrome was used for this purpose ***(Hernu et al., 2013).***

Several scoring systems have been developed to predict the outcome of thoracic trauma. Some, such as the Lung Organ Failure Score (LOFS) ***(Wutzler et al., 2012)*** or the Watkins predictive model determined risk factors associated with acute respiratory distress syndrome ***(Watkins et al., 2012)*** but the Thoracic Trauma Severity score is the only one to account for demographic data such as age or respiratory status (ratio between partial pressure of oxygen in arterial blood and inspired fraction of oxygen [PaO_2/FiO_2 ratio]) in addition to most thoracic injuries (pleural effusions, pulmonary contusions, rib fractures) ***(Pisani et al., 2016).***

Triple test score (TTS) determined on admission was associated with increased occurrence of acute respiratory distress syndrome and mortality in a population of patients with severe thoracic trauma at very high risk of respiratory failure.

The performance of the thoracic trauma severity score for predicting acute respiratory disress syndrome remains therefore to be determined in a less severe trauma population, especially those who had no respiratory distress on admission, these latter patients who are most at risk of being underestimated would benefit from specific prophylactic strategies knowing the specific risk of delayed progression to acute respiratory distress syndrome induced by pulmonary contusions ***(Lee, 2012).***

Early identification of a large number of patients at risk of delayed ARDS depend on these scores especially thoracic trauma severity score and laboratory finding lead to early detection of acute respiratory distress syndrome and guide optimal initial management and preventive measures such as protective mechanical ventilation, restrictive fluid therapy, damage control surgery and strategy of staged interventions ***(Fernandez et al., 2016).***

Aim of the Work

Diagnosis of traumatic patients who are at risk of developing ARDS based on clinical and laboratory findings and their proper management.

IDENTIFICATION AND EPIDEMIOLOGY OF ACUTE RESPIRATORY DISTRESS SYNDROME

Identification

Classic definition:

Acute respiratory distress syndrome is first described in 1967 consists of a clinical triad of respiratory distress, cyanosis that is refractory to oxygen therapy, decrease lung compliance and diffuse pulmonary infiltrates that are evident in a chest radiograph ***(Ashbaugh et al., 1967).***

AECC definition:

In 1994, the American-European Consensus Conference Committee defined ARDS as presence of diffuse pulmonary infiltrates, P/F ratio (P/F ratio of 300 or less indicates ALI, P/F ratio of 200 or less indicates ARDS) and absence of left heart failure ***(Bernard et al., 1994).***

Criticism of AECC definition:

Chest x-Ray: Inter observer reliability is only moderate even when applied by experts ***(Meade et al., 2008).***

Hypoxia: Pao2 /Fio2 ratio is not constant across a range of Fio2 and may vary in response to ventilator setting particularly PEEP ***(Villar et al., 2014).***

Wedge pressure: Patients with ARDS may have an elevated PAWP, often because of transmitted airway pressure and/or vigorous fluid resuscitation ***(Ferguson et al., 2012).***

When AECC criteria are compared with diffuse alveolar damage sensitivity is 84% specificity is 51% ***(Ferguson et al., 2005).***

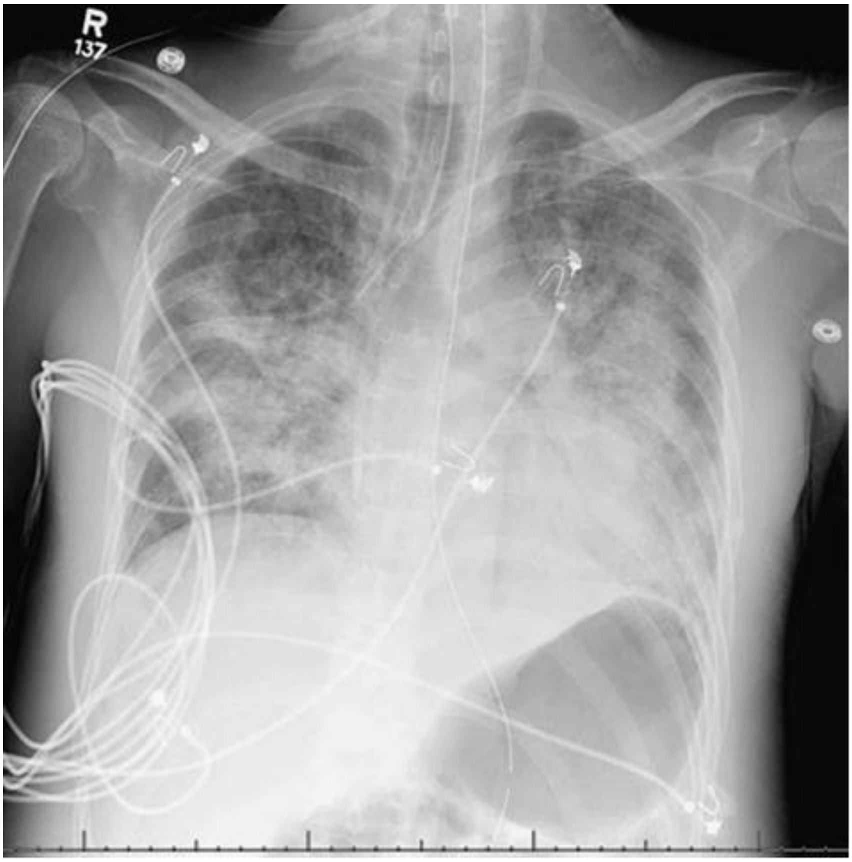

Figure (1): Portable chest radiograph, revealing bilateral pulmonary opacities ***(Meade et al., 2008).***

The berlin definition

ARDS is defined by an acute hypoxemia, a ratio of partial pressure of artrial oxygen to the fraction of inspired oxygen less than or equal to 300 mmHg on positive end-expiratory pressure greater than or equal to 5 cm H2O, together with bilateral infiltration on radiology that is not otherwise explained fully by fluid overload or cardiac failure ***(Fan et al., 2018).***

Table (1): The berlin definition of acute respiratory distress syndrome ***(Force et al., 2012).***

Acute Respiratory Distress Syndrome	
Timing	Within 1 week of a known clinical insult or new or worsening respiratory symptoms.
Chest imaging	Bilateral opacities not fully explained by effusions, lobar/lung collapse, or nodules.
Origin of edema	Respiratory failure not fully explained by cardiac failure or fluid overload Need objective assessment (eg, echocardiography) to exclude hydrostatic edema if no risk factor present.
Oxygenation	Mild PaO2/FIO2 < 300 mm Hg with PEEP ≥ 5 cm H2O.
	Moderate PaO2/FIO2 < 200 mm Hg with PEEP ≥ 5 cm H2O.
	Severe PaO2/FIO2 < 100 mm Hg with PEEP ≥ 5 cm H2O.