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Prognostic factors in the Acute respiratory distress syndrome/Acute lung injury (ALI)

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ABSTRACT

Introduction: Despite improvements in critical care, acute respiratory distress syndrome (ARDS) remains a devastating clinical problem with high rates of morbidity and mortality.
Aim of study were To identify the different risk factors responsible for ARDS/ALI, To study the lung functions in ARDS/ALI survivors, To analyze the effect of treatment strategies on lung functions in survivor,To study different scoring systems.

Materials and methods:
This was a prospective cohort (analytical epidemiology) study conducted on Human Subjects. All the survived cases of ARDS/ALI attending tertiary health care center over a period of 6 months were enroll in the study. All patients were screened for the diagnostic criteria of ARDS according to American European consensus definition. Patients with diagnosis of ARDS/ALI having age more than 12 years and willing to take part in the study and given consent were recruited. Patients having contraindication for Pulmonary function test, history of recent myocardial infarction and active hemoptysis were excluded from the study.

ISSN:0975-3583,0976-2833 VOL13,ISSUE04,2022

Result: In our study, we had a total of 70 patients, out of which 50 survived. The risk factors for developing ALI/ARDS were pneumonia, tropical diseases, postoperative sepsis, poly trauma, tuberculosis, malignancy, poisoning and neurological disorder.

Conclusion: Pneumonia and tropical diseases are the common risk factor for the ARDS/ALI. The presence of co-morbid conditions also affects the outcome of ALI/ARDS patients. MODS of >4, LIs >2 and APACHE II >2 had associated with higher mortality

Keywords: Acute respiratory distress syndrome, Simplified Acute Physiology Score, National Institutes of Health.

INTRODUCTION

The acute respiratory distress syndrome (ARDS) is a devastating syndrome of pulmonary inflammation and edema manifested clinically by the acute onset of bilateral infiltrates on chest radiograph, and arterial hypoxemia. [1] Despite advances in the care of critically ill patients, both short and long-term mortality rates of ARDS remain high.[2] Currently, the only therapy that has been definitively proven to reduce mortality in ARDS is a lower tidal volume ventilator strategy.[3] However, other therapies have been shown to reduce mortality in a subset of patients with ARDS. For example, application of prone positioning in patients with severe ARDS (defined for this study as a ratio of the partial pressure of arterial oxygen to the fraction of inspired oxygen [PaO2/FiO2] of less than 150 mm Hg) significantly decreased 28-day and 90-day mortality. [4] Thus, a better understanding of prognostic factors in ARDS is crucial for facilitating risk stratification and developing new therapeutic interventions that aim to improve clinical outcomes. [5]

There have been a number of large studies over the past several decades that have explored clinical risk factors for hospital and short-term mortality in ARDS. Clinical predictors that were consistent across multiple studies include older age, worse physiologic severity of illness (as measured by severity scores such as the Acute Physiology and Chronic Health Evaluation (APACHE) or Simplified Acute Physiology Score (SAPS)), shock on hospital admission, arterial pH less than 7.30, liver disease, early air leak, immunosuppression, triggering risk factor, and right ventricular dysfunction.[6] It is important to note that these studies were all done before the era of lower tidal volume ventilation. In more recent studies done in clinical trial patient populations after the widespread implementation of low tidal volume ventilation, severity of illness as measured by APACHE II or APACHE III has remained a robust clinical predictor of mortality as have age, and the presence of non-pulmonary organ failures.[7]

In regards to effects of race and ethnicity on mortality from ARDS, a retrospective cohort study from the National Institutes of Health (NIH) ARDS Network revealed that African American and Hispanic patients have a higher mortality from ARDS than Caucasian patients.[8] The

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mechanisms that underlie this racial disparity in mortality are unknown; one proposed factor is related to the high incidence of a mutation in the promoter region of the Duffy antigen/receptor for chemokines (Darc) gene in those of African ancestry that leads to low levels of erythrocyte Duffy antigen, reduced chemokine binding and potentially to higher levels of circulating chemokines such as IL-8.[9] In a large multicenter study, Lemos-Filho LB et al. found that AfricanAmerican patients were less likely to develop ARDS than Caucasians even after adjustment for clinical predictors despite having higher severity of illness at presentation.[10] In the same study, there was no difference in ARDS mortality by sex or race after adjustment for covariates [11].

Aim of study were to identify the different risk factors responsible for ARDS/ALI. To study the lung functions in ARDS/ALI survivors.To analyze the effect of treatment strategies on lung functions in Survivor.To study different scoring systems, MODS (Multiple Organ Dysfunction Score), SOFA (Sequential Organ Function Assessment), LIS (Lung Injury Severity) and APACE II (Acute Physiological, age and Chronic Health Evaluation II) in ALI/ARDS.

MATERIALS AND METHODS

This was a prospective cohort (analytical epidemiology) study conducted on Human Subjects. All the survived cases of ARDS/ALI attending tertiary health care center over a period of 6 months were enroll in the study. All patients were screened for the diagnostic criteria of ARDS according to American European consensus definition. Patients with diagnosis of ARDS/ALI having age more than 12 years and willing to take part in the study and given consent were recruited. Patients having contraindication for Pulmonary function test, history of recent myocardial infarction and active hemoptysis were excluded from the study.

Baseline evaluation included recording of demographic details, medical history, general and systemic examination, and laboratory investigations, which included completehaemogram, PFT (using Jaeger PFT machine), DLCO and chest X-ray. Further investigations included a peripheral smear for malarial parasite and tri-dot test for leptospirosis. A coagulation profile including levels of APTT (Activated Partial Thromboplastic time) and PT (prothrombin Time) was recorded. The severity of the illness was measured by the Acute Physiology and Chronic Health Evaluation (APACHE) Scrore, Multiple Organ Dysfunction score (MODS) lung injury score (LIS) and Sequential Organ Dysfunction Assessment (SOFA score). These scores were calculated on admission to our intensive care unit.

RESULTS

In our study, we had 50 patients.

Table 1: Distribution of gender between alive patients and dead patients

Gender	Alive patients in no.	Dead patients in no.
Male	32	14
Female	18	6

ISSN:0975-3583,0976-2833 VOL13,ISSUE04,2022

Total	50	20

Table 2: Distribution of age groups between alive patients and dead patients

Age (in years)	Alive patients in no.	Dead patients in no.
<25	15	4
25-60	25	14
>60	10	2
Total	50	20

In our study, most of the patients were 25-60 years i.e., 25 out of 50, followed by <25 years, i.e., 15 out of 50 alive patients in number.

Table 3: Smoker and Alcoholic of ARDS/ALI (both survivors and non-survivors)

Parameter		Alive patients in no.	Dead patients in no.
Smoker	Yes	9	7
	No	41	13
Alcoholic	Yes	11	7
	No	39	13

Table 4: Diagnosis of ARDS/ALI (both survivors and non-survivors)

Diagnosis	Alive patients in no.	Dead patients in no.
Pneumonia	22	11
Post-operative sepsis	10	3
Poly trauma	9	3
Neurological Disorder	2	1
TB	2	1
Poisoning	2	0
Tropical diseases	3	1

The risk factors for developing ALI/ARDS were pneumonia (33 patients), postoperative sepsis (13 patients), poly trauma (12 patients), tuberculosis (3 patients), poisoning (2 patients) and neurological disorder (3 patients).Tropical diseases (4 patients).

Table 5: Sign and symptoms of patients with ARDS/ALI (both survivors and non-survivors)

Sign and Symptoms		Alive patients in no.	Dead patients in no.
Time since onset	<10 days	28	15
	>10days	22	5
Fever	No	15	4
	<8days	35	16
Cough	No	16	2
	<8days	29	16

ISSN:0975-3583,0976-2833 VOL13,ISSUE04,2022

	>8days	5	2
Breathlessness	No	10	3
	<8days	31	14
	>8days	9	3
Bleeding	Yes	11	4
	No	36	16
	Epistaxis	3	0
Respiratory disease	COPD	3	10
	Byssinosis	3	4
	Bronchiectasis	3	1
	Bulla	0	0
	Nil	41	5
Temperature O^{o}C		37.96	37.13
Pulse (/min)		107	104
Respiratory rate (/min)		34	37
Glasgow coma scale		14	12
Pallor	Yes	19	6
	No	31	14
Cyanosis	Yes	12	3
	No	38	17
Skin Rash	Yes	42	15
	No	8	5
DIC	Yes	11	4
	No	39	16
Liver dysfunction	Absent	10	11
	Present	40	9
Renal dysfunction	Absent	21	8
	Present	29	12
P/F ratio		173	119

Table 6: Blood investigation of patients with ARDS/ALI (both survivors and non-survivors)

Investigation		**Alive patients in no.**	**Dead patients in no.**
Platelets in lacks		2.6	1.9
PT/APTT	Normal	3	1
	Deranged	47	19

ISSN:0975-3583,0976-2833 VOL13,ISSUE04,2022

Table 7: Clinical details of patients with ARDS/ALI (both survivors and non-survivors)

Parameter		Alive patients in no.	Dead patients in no.
SOFA	<5	30	18
	>5	20	2
MODS	<4	33	16
	>4	17	4
LIS	<2	19	3
	>2	31	17
APACHE II	<12	27	15
	>12	23	5
Steroids taken	Yes	6	1
	No	44	19
Days on ventilator		2	6

DISCUSSION:

Acute respiratory distress syndrome (ARDS) refers to the syndrome of lung injury characterized by dyspnea, severe hypoxemia, decreased lung compliance, and diffuse bilateral pulmonary infiltrates. Family physicians can play an essential role in the early recognition of ARDS and contribute to the multispecialty team required to manage this life-threatening condition. This article reviews the current understanding of the pathophysiology, management, and prognosis of ARDS.[12]

Originally referred to as traumatic wet lung, shock lung, or congestive atelectasis, ARDS was recognized in 1967 when the clinical, physiologic, radiographic, and pathologic abnormalities that were unique to a group of 12 patients were described, distinguishing them from other cases in a series of 272 patients treated for respiratory failure.[13] A National Institutes of Health (NIH) panel initially estimated an incidence of 75 per 100,000 persons per year, but subsequent prospective studies give a range of 12.6 to 18 per 100,000 persons annually.[14] Controversy still exists about the correct incidence because of differing criteria used to define ARDS. The prevalence of ARDS is reported to be between 15 and 18 percent of all ventilated patients.4 while a mortality rate greater than 50 percent is reported in the majority of clinical investigations performed between 1979 and 1994, more recent studies show a decline in mortality to be between 32 and 45 percent. [15]

Before 1992, the acronym ARDS represented the adult respiratory distress syndrome. The American-European Consensus Committee on ARDS standardized the definition7 in 1994 and renamed it acute rather than adult respiratory distress syndrome because it occurs at all ages. The term acute lung injury (ALI) was also introduced at that time. The committee recommended that ALI be defined as “a syndrome of inflammation and increased permeability that is

ISSN:0975-3583,0976-2833 VOL13,ISSUE04,2022

associated with a constellation of clinical, radiologic, and physiologic abnormalities that cannot be explained by, but may coexist with, left atrial or pulmonary capillary hypertension. [16]

Exclusion of left atria hypertension as the primary cause of hypoxemia is critical to this definition, and measurement of pulmonary capillary wedge pressure may be necessary. The distinction between ALI and ARDS is the degree of hypoxemia, defined by the ratio of arterial oxygen tension to fractional inspired oxygen concentration (PaO2/FIO2). ALI is defined by a ratio less than 300 mm Hg, and 200 mm Hg or less is required for ARDS.[17]

In ARDS, the injured lung is believed to go through three phases: exudative, proliferative, and fibrotic, but the course of each phase and the overall disease progression is variable. In the exudative phase, damage to the alveolar epithelium and vascular endothelium produces leakage of water, protein, and inflammatory and red blood cells into the interstitium and alveolar lumen. These changes are induced by a complex interplay of proinflammatory and anti-inflammatory mediators.[18]

CONCLUSION

Pneumonia and tropical diseases are the common risk factor for the ARDS/ALI. The presence of co-morbid conditions also affects the outcome of ALI/ARDS patients. MODS of >4, LIs >2 and APACHE II >2 had associated with higher mortality.

Conflicts of Interest: None declared.
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ISSN:0975-3583,0976-2833 VOL13,ISSUE04,2022

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