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A Clinical Study of Prognostic Determinants And Outcomes of Acute Respiratory Distress Syndrome

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Abstract: *Acute Respiratory Distress Syndrome occur in response to a variety of insults and are characterised by the development of non cardiogenic pulmonary edema, impaired gas exchange and bilateral infiltrates on the chest radiograph. Acute Lung Injury and ARDS are the major causes of acute respiratory failure that are associated with high mortality and morbidity. Present study aims to analyse the prognostic determinants and outcomes of acute respiratory distress syndrome (ARDS).Patients that fulfilled the Berlin definition for ARDS and who were mechanically ventilated for more than a twenty four hour period were selected for the study and were analysed. PaO2/FIO2 is a strong predictor outcome of mortality in ARDS. Clinical scores – SOFA, maxSOFA, SAPS II and LIS had a statistically significant association with mortality and hence are validated methods to predict outcome of ARDS. CRP and Procalcitonin are not significant predictors.*

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I. Introduction

Acute Respiratory Distress Syndrome occur in response to a variety of insults and are characterised by the development of non cardiogenic pulmonary edema, impaired gas exchange and bilateral infiltrates on the chest radiograph. Acute Lung Injury and ARDS are the major causes of acute respiratory failure that are associated with high mortality and morbidity.An understanding of the basic clinical epidemiology of a disease—its incidence, diagnosis, etiologic and prognostic factors, relevant disease subsets, mortality, and long-term outcomes—is essential to caring for patients with the disease and for designing studies to evaluate potential therapies. The first definition of ARDS dates to Ashbaugh & colleagues in1967[1], followed by the American-European consensus conferences definition in1994.[2]The AECC guidelines have been challenged over the years in several studies since the assessment of oxygenation defect does not require standardized ventilatory support.[3] The hypoxaemia criterion (pao_2 fio_2<200mm of hg) can be markedly effected by patients ventilation settings[4]

Recently a new consensus definition of ARDS , the Berlin definition of has been published.[5] A new definition of ARDS maintains a link to the 1994 definition with diagnostic criteria timing, chest imaging, origin of edema & hypoxemia. There are a few key modifications (oxygenation, timing of acute onset, chest x-ray and wedge pressure criterion) in the Berlin definition as compared with the AECC definition.

American European Consensus Conference Criteria for Acute Lung Injury (ALI) and the Acute Respiratory Distress Syndrome (ARDS)

Clinical Variable	Criteria for Acute Lung Injury	Criteria for Acute Respiratory Distress Syndrome
Onset	Acute	Acute
Hypoxemia	$Pa_{O_2}/FI_{O_2} \leq$ 300mm Hg	$Pa_{O_2}/FI_{O_2} \leq$ 200 mmHg
Chest radiograph	Bilateral infiltrates consistent with pulmonary edema	Bilateral infiltrates consistent with pulmonary edema
Noncardiac cause	No clinical evidence of left atrial hypertension or, if measured, pulmonary artery occlusion pressure ≤ 18mmHg	No clinical evidence of left atrial hypertension or, if measured, pulmonary artery occlusion pressure ≤ 18 mmHg

Table 1: aecc guidelines for ali and ards.

II. Methodology

This is a prospective study conducted in the intensive care units of our department during 2015-2017. Patients that fulfilled the Berlin definition for ARDS and who were mechanically ventilated for more than a

twenty four hour period were selected for the study. Patients with heart failure either on clinical examination or on echocardiography were excluded from the study. A total of seventy two patients were enrolled during the study period. A relevant history was noted and a physical examination was performed on enrolment of the patient into the study. Data was recorded on the day of diagnosis ARDS and every twenty four hours thereafter. Baseline clinical data and demographics were noted. Patients were scored on Day 0 of diagnosis of ARDS using the SAPS II system, SOFA score and Lung Injury Score. Baseline investigations including a haemogram, renal and liver function tests, serum electrolytes coagulation profile, arterial blood gases, relevant cultures and serology for an etiological diagnosis were sent. Chest radiographs were ordered on the day of diagnosis and at periodic intervals to look for worsening or improvement. Serum Procalcitonin and C Reactive protein levels were sent within forty eight hours of diagnosis of ARDS. A two dimensional echocardiography was performed when deemed necessary to rule out myocardial dysfunction. Patients were followed up every twenty four hours to record vital parameters, new onset organ dysfunction, arterial blood gases and use of ionotropes. In the event of more than one value for any parameter in a 24 hour period, the most abnormal value was taken into consideration. The patients were followed up till death or discharge from the ICU.

III. Results

Of the 72 patients included in the study, 61% were males and average age of the group was 42.6 years (28-64 years). The most common etiological factor for ARDS in our study was LRTI(48%) followed by Sepsis (41%). Eighty eight percent of the cases of ARDS in this study were secondary to infectious causes. twenty one patients grew organisms on cultures. Three patients were diagnosed with Plasmodium Falciparum Malaria on blood smear examination andone patient was diagnosed with Dengue.

Variable	Mean values (SD)		P value
	Non **Survivors**	**Survivors**	
PEEP	8.9	5.8	<0.001
MAP (mmHg)	82	89	0.3
Ph	7.19	7.43	0.5
S.Bicarbonate (mEq/L)	21.4	25.9	0.086
PaO2/FiO2	91.9	165.9	<0.001
Hematocrit	31.9	33.2	0.5
WBC count (thousand/mm3)	11.8	9.7	0.3
Platelet count (lakhs/mm3)	1.56	2.4	0.02
S.Creatinine (mg/dl)	1.23	0.9	0.05
S.Bilirubin (mg/dl)	2.0	1.2	0.14
S.Albumin (g/dl)	2.5	2.8	0.06
No of blood products transfused	3.7	1.1	0.03
Procalcitonin	9.7	1.7	0.3
SOFA	8.39(32.0)	4.24(1.6)	<0.001
MaxSOFA	9.97(4.1)	4.41(1.8)	<0.001
SAPS II	32.61(13.2)	19.94(6.8)	<0.001
LIS	2.73(0.4)	1.83(0.2)	<0.001

Table 1 : Difference In The Baseline Physiological And Laboratory Parameters Between Survivors And Non Survivors

It was noted that the percentage of non survivors were higher in classes with lower albumin. The mean Albumin among non survivors was lower than that of non survivors, 2.5 versus 2.8 g/dl (p-0.06). Patients were divided into classes with increasing SAPS II scores. It was observed that greater the score, higher was the mortality in that group. (p value - <0.001). It was observed that amongst patients with higher SOFA scores, the mortality was greater compared to those with lower values on the scoring system. Based on the LIS, patients were divided into those that has mild to moderate and severe lung injury. All the patients who recovered fell into the former category. Two thirds of the patients that succumbed to the illness had severe lung injury scores. Of the 72 patients enrolled in the study, 43 patients succumbed to their illness – the mortality being 60%.

Mean (days)	**Non Survivors**	**Survivors**
Length of ICU Stay	7.56	10.12
Duration of Ventilation	5.78	5.45

Table 2: Mean duration of ICU stay and ventilation between survivors and Non Survivors

IV. Discussion

The commonest cause of direct injury is pulmonary infection (48%of total cases) and Aspiration (4%of total cases).While systemic sepsis(42%of total cases) was the commonest among indirect causes. Indirect causes include severe non thoracic trauma (2% of total cases) and post abdominal surgery (4%of total cases). The results were similar with findings in other studies.[6] In our study a higher proportion of patients having moderate and severe ARDS had an infectious etiology. Pneumonia is the commonest underlying condition of ARDS. Streptococcal pneumonia, Staphylococcus aureus, Mycoplasma, Coxiella and gram negative bacilli are the most common etiological agents of pulmonary infections that cause ARDS and the spectrum of isolated pathogens are similar in America and Europe.[7,8] The etiology of pneumonia may vary depending on the geographical area and that the microbiology of one third of cases may remain unidentified. Since ventilator associated pneumonia (VAP) may complicate the course of ARDS that required mechanical ventilation [7]Some primary causes of ARDS may have been unidentified in the cases of VAP seen in our study.

Acute respiratory failure and ARDS are one of the commonest complications of H1N1 infection requiring ICU care. Incidence of ARDS among patients infected with H1N1 admitted into the ICU was 65.4% according to one study.[9] Kumar et al in 2012, described 32 patients with ARDS and 20 cases succumbing to illness.[10]Only one case was diagnosed as H1N1 in our study. Dengue is a major seasonal health problem in tropical countries, but the incidence of ARDS is very low, albeit with high mortality.[11] Pulmonary manifestations of dengue infection such as pleural effusion and pneumonitis are common as a part of polyserositis in dengue.[12]One case of ARDS was due to dengue fever and she had survived. Wang et al evaluated 606 dengue patients and reported an incidence of 1.8% ARDS in their study.[11]

The parameters that had a statastically significant association with mortality include PEEP, PaO2/FiO2, Serum Bicarbonate, Platelet count, serum creatinine. A prescription of inotropes and number of blood products transfused. The clinical scores –Lung Injury Score, SAPSII, and SOFA had a significant association with mortality.The PaO2/FiO2 ratio at the onset of ARDS does not predict clinical outcome but may be more useful after the first day of ARDS. A persistently low PaO2/FiO2 ratio is associated with worst outcomes and may be a marker of failure to respond to conventional therapy.[13.] Hemodynamic profile of ARDS was studied by P.squara and his collegues.[14] ·They had concluded that PaO2/FiO2 ratio at admission was an independent predictor of survival, however an analysis performed by Kraft and his collegues[15] showed that statistical comparisons of the PaO2/FiO2 ratio of survivors and non survivors were not significant on the first day of ARDS. Our results were consistent with the former study.Serum levels of procalcitonin have been reported to be elevated in sepsis. PCT, is a protein of 116 amino acids of known sequence. Although it is unclear at present which tissue is responsible for PCT liberation during inflammation, it does not seem to be thyroid gland. In sepsis, PCT levels increase seven fold until they are several thousand fold greater, and on admission this increase often correlates with the severity of the condition and with subsequent mortality.[16] A variety of studies and reviews have shown the superior diagnostic accuracy of PCT as compared with other parameters for the diagnosis of sepsis, independent of origin of infection. Whereas the increase of other inflammatory markers such as CRP is attenuated by immunosuppressive medication such as corticosteroid, the diagnostic accuracy of PCT remains unaffected. In addition PCT seems to have a slight advantage over CRP because of its earlier increase upon infection and a better negative predictive value, as shown for example in children with fever of unknown origin and adult patients with systemic inflammatory response in critical illness, trauma,and surgery.[16] C reactive protein (CRP) is a 21-kd protein that is synthesized primarily in the liver and found in blood plasma. Originally extracted from the blood of patients with pneumonia, CRP was the first of the acute-phase reactant proteins to be discovered. Plasma CRP levels undergo a rapid and robust rise in response to inflammatory stimuli. Because of this phenomenon, plasma levels of CRP have long been considered to be an important biomarker for detecting the presence of systemic inflammation. Measurement of CRP level has been shown to have prognostic and/or diagnostic value in a large number of disease states, including sepsis, pneumonia, appendicitis, coronary artery disease (CAD), stroke, diabetes, and rheumatic disease, among others. In most cases, higher CRP levels have been associated with adverse outcomes. However, a recent study conducted by Bajwa et al demonstrated improved outcomes in ARDS with higher CRP levels[17].

It was observed that the no of units of blood products transfused were significantly higher in the group of patients that died compared to those that recovered (p - 0.03). A study conducted by Michelle Ng Gong had noted a similar association[18].This apparent increase in mortality, amongst patients transfused larger volumes of blood products, may be either because this group has a greater degree of organ dysfunction thus necessitating transfusions or; may indicate that the transfusions have contributed to the lung injury. To test the latter hypothesis, the correlation between the number of transfusions and the LIS was calculated and was found to be statistically insignificant. (p– 0.1).

V. Conclusion

PaO2/FIO2 is a strong predictor outcome of mortality in ARDS. Clinical scores – SOFA, maxSOFA, SAPS II and LIS had a statistically significant association with mortality and hence are validated methods to predict outcome of ARDS. CRP and Procalcitonin are not significant predictors.

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