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**ORIGINAL RESEARCH PAPER**

# CLINICOETIOLOGICAL PROFILE AND OUTCOME IN ACUTE RESPIRATORY DISTRESS SYNDROME

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**ABSTRACT** : ARDS is caused by diffuse lung injury from many underlying medical and surgical disorders. The lung injury may be direct, as occurs in toxic inhalation, or indirect, as occurs in Sepsis. Common risk factors for ARDS are pneumonia, Sepsis, gastric content, aspiration, trauma, pancreatitis, inhalation injury, burns, non-cardiogenic shock, drug overdose, acute lung injury following massive transfusion (TRALI), drowning Approximately 10%- 15% of all intensive care unit (ICU) admissions involve patients with ARDS.An understanding of basic clinical epidemiology of the disease, its incidence aetiology, diagnosis, Mortality and

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outcome is essential to caring for patients with the disease and for designing studies to potentiate essential therapies. Various clinical studies have been done to know the aetiology and Factors affecting the outcome and treatment strategy. Yet the only. proven therapy to consistently reduce Mortality is a protective Ventilation strategy with low tidal volume and prone ventilation. Despite this ,the present mortality estimates range from 26 % to 58%. Mortality in tropical countries is incredibly high.

This observational study was conducted to know the aetiology of ARDS in Patients in a tertiary care hospital. This was done to recognize ARDS early.

**AIM OF STUDY:**

To study the Etiology for development of ARDS among patients admitted in ICU.

To study outcome in terms of Mortality, duration of mechanical ventilation, length of ICU stay.

To determine the short term outcome in ARDS.

**MATERIALS AND METHODS :** This is a prospective observational study and study was carried out in patients who fulfilled the AECC definition of ARDS and admitted in the Intensive care unit in King George Hospital, Visakhapatnam. The study was conducted between July 2018 and October 2020 for a period of 2 years and two months.

**The Study design is an Observational study.**

**RESULTS AND ANALYSIS :** For Statistical analysis, IBM SPSS version 22 was used.

Fifty patients that met the predefined criteria were enrolled for the study. Analysis of data was performed using the IBM SPSS version 22 software. Pulmonary Infection (48%) followed by Sepsis (42%) were the most common causes for ARDS in this study. 56% of cases were attributable to direct causes, and 94% of cases were secondary to infectious etiologies.

**INTRODUCTION**

ARDS (Acute Respiratory Distress Syndrome) is a rapidly progressive form of acute respiratory failure characterized by refractory hypoxemia and non-cardiogenic pulmonary edem[1]

The Acute Respiratory Distress Syndrome (ARDS)is characterized by pulmonary inflammation and increased pulmonary vascular permeability, which results in non- cardiogenic Pulmonary oedema and refractory hypoxaemia.[2]

ARDS first defined by Ashbaugh and colleagues in 1967 when they described 12 patients with severe acute respiratory failure [3] and since then there have been multiple studies addressing the various clinical aspects of the syndrome, its pathogenesis, risk factors, and treatment American-European Consensus Conference (AECC) published a definition and defined ARDS as the acute onset of hypoxemia (the ratio of the partial pressure of arterial oxygen(Pao2) to the fraction of inspired oxygen (FiO2) ≤200 mmHg), with bilateral infiltrates on frontal chest X-ray, in the absence of left atrial hypertension [4]

The hallmarks of the clinical syndrome are hypoxemia and bilateral. Radiographic opacities five morphological features in the acute phase are lung oedema, inflammation, hyaline membranes, and alveolar haemorrhage (i.e., diffuse alveolar damage) [6]

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ARDS is caused by diffuse lung injury from many underlying medical and surgical disorders. The lung injury may be direct, as occurs in toxic inhalation, or indirect, as occurs in Sepsis.

Common risk factors for ARDS are pneumonia, Sepsis, gastric content, aspiration, trauma, pancreatitis, inhalation injury, burns, non-cardiogenic shock, drug overdose, acute lung injury following massive transfusion (TRALI), drowning [7]

Approximately 10%- 15% of all intensive care unit (ICU admissions involve patients
with ARDS [8-11] ).

An understanding of basic clinical epidemiology of the disease, its incidence aetiology, diagnosis, Mortality and outcome is essential to caring for patients with the disease and for designing studies to potentiate essential therapies. Various clinical studies have been done to know the aetiology and Factors affecting the outcome and treatment strategy.

Yet, the only. proven therapy to consistently reduce Mortality is a protective
Ventilation strategy with low tidal volume and prone ventilation.

Despite this, the present mortality estimates range from 26 % to 58%.

Mortality in tropical countries is incredibly high.

This observational study was conducted to know the aetiology of ARDS in Patients in a tertiary care hospital. This was done to recognize ARDS early.

**AIMS OF THE STUDY**

**AIMS AND OBJECTIVES**

To study the Etiology for development of ARDS among patients admitted in ICU.

To study outcome in terms of Mortality, duration of mechanical ventilation, length of ICU stay. To determine the short term outcome in ARDS.

**REVIEW OF LITERATURE**

American European Consensus Conference (AECC) has defined ARDS in 1994 which included the following- Acute bilateral pulmonary infiltrates, Ratio of Arterial oxygen tension (Pao2) to the fraction of inspired oxygen (Fio2)<200mm Hg, No evidence of Heart failure or volume overload as the principal cause of the pulmonary infiltrates [12].

In 2013, Berlin definition was published which included the following 4 components

1.Acute onset over less than one week,

2-Bilateral opacities consistent with pulmonary oedema must be present and may
be detected on Chest X-ray or CT,

3-PaO2/FiO2 ratio less than 300mm Hg,

4- Respiratory failure not explained by cardiac failure or fluid overload. An objective assessment should be performed (eg.2DECHO) to exclude cardiogenic oedema [13]. The annual incidence of ARDS is 60 cases per 1,00,000 population [14]. Mild ARDS comprises only 25% of patients with ARDS and moderate to severe ARDS comprises 75%.

As per the recent study done in LUNG SAFE trial, the period prevalence of ARDS was 10.4% of ICU admissions [15].

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## PATHOPHYSIOLOGY

The hallmark pathophysiologic feature of ARDS is increased capillary permeability [16]. There is a damage of capillary endothelium and alveolar epithelium with the accumulation of protein-rich fluid inside the alveoli.

## CAUSES

**TABLE – 1 – CAUSES OF ARDS**

| DIRECT LUNG INJURY | INDIRECT LUNG INJURY |
|---|---|
| COMMON CAUSES-<br>PNEUMONIA<br>ASPIRATION OF GASTRIC CONTENTS | COMMON CAUSES-<br>SEPSIS<br>SEVERE TRAUMA WITH SHOCK AND MULTIPLE TRANSFUSIONS |
| LESS COMMON CAUSES-<br>PULMONARY CONTUSION<br>FAT EMBOLI<br>NEAR DROWNING<br>INHALATION INJURY<br>REPERFUSION PULMONARY OEDEMA | LESS COMMON CAUSES-<br>CARDIO-PULMONARY BYPASS<br>DRUG OVERDOSE<br>ACUTE PANCREATITIS<br>TRANSFUSION OF BLOOD PRODUCTS |

## CLINICAL MANIFESTATIONS AND DIAGNOSIS

ARDS is characterized by the development of dyspnea initially, dyspnea on exertion followed by dyspnea at rest. There will be the development of dyspnea and hypoxemia within hours to days of inciting event. Patients developing ARDS are critically ill, often with multisystem organ failure. Typically the illness develops within 12 to 48 hours after the inciting event.

**Diagnosis**- ***Based on Berlin criteria published in 2013***[29] ·

Acute onset over less than one week ·

Bilateral opacities consistent with pulmonary oedema must be present and maybe detected on Chest X-ray or CT. ·

PaO2/FiO2 ratio of less than 300mm Hg. ·

Respiratory failure not explained by fluid overload or cardiac failure.

An objective assessment should be performed (eg.2DECHO) to exclude cardiogenic oedema.

## MANAGEMENT OF ARDS

**Control of causal factor:**

Although ARDS does not have a specific treatment, many of the factors that cause and perpetuate this pathology can be treated or controlled.

**Oxygen Therapy:**

Patients with ARDS present, by definition with significant hypoxemia. Hypoxemia in ARDS is due to intrapulmonary shunting where Unventilated areas due to alveolar oedema, atelectasis and consolidation continue

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to receive blood supply. Hence, Oxygen administration is indicated in the initial phases of acute respiratory failure. But in most of the cases, it is only of temporary symptomatic relief.

With the progression of ARDS, there will be decreased lung compliance increased respiratory muscle work, worsening hypoxemia requiring mechanical ventilation. Mechanical
Ventilation: Mechanical Ventilation is the main supportive modality in ARDS and is indicated in most cases [30]. One of the key point in the treatment of ARDS is the early identification of patients with respiratory compromise, so that mechanical ventilation is initiated before they
develop an extreme state of respiratory failure.

**Indications for Mechanical Ventilation in ARDS are ·**

Clinical data- Dyspnea, tachypnea, accessory muscle fatigue, acidosis. ·
Radiological – Worsening of alveolar infiltrate ·
PaO2 < 50mm Hg, PaCO2- > 50 mm Hg with FiO2 of 50%.

**Ventilation Mode :**

Regardless of ventilation mode used, it is important to emphasize that no one conventional model has been shown to be clinically superior to another in the management of patients with ARDS, as long as principles of protective ventilation are maintained. The most common used Ventilatory modes are Time cycled Volume limited modes –
Synchronized Intermittent Mandatory Ventilation, Continuous Mandatory
Ventilation [31].
In these, the operator determines the exact Tidal volume to be delivered at each mandatory fan cycle. The pressure measurements generated by this volume at the end of inspiration or after
a pause are indicators of pulmonary complacency in ARDS. A peak of inspiratory pressure that increases over Time to given volume generally indicates worsening compliance. A decrease in peak inspiratory pressure generally indicates an improvement in compliance.

**Positive End Expiratory Pressure-**

PEEP In ARDS; dependent lung regions show gravity dense alveolar and interstitial inflammatory infiltration, oedema, cell debris, atelectasis and consolidation, while non-dependent areas are relatively spared [32]. As a result, functioning alveoli are reduced in the dependent regions in relation to non-dependent regions. Thus at each expiration, there will be
the closure of alveoli resulting in alveolar collapse. Then during inspiration, these collapsed alveoli are reopened. Repetitive opening and collapse generates very high forces capable of causing tissue damage- atelectrauma. The main purpose of PEEP is to prevent less compliant
alveoli from collapsing at the end of expiration. High PEEP levels are used in ARDS which may open collapsed alveoli and decrease intrapulmonary shunt with improvement in oxygenation [33], But excessive use of PEEP increases the risk of pneumothorax, adverse hemodynamic effects increased intrathoracic pressure and decreased venous return- preload. At the same time, low PEEP during mechanical ventilation leads to cyclic closure and alveolar reopening and results in atelectrauma. The use of PEEP with an adequate level to maintain pulmonary volume during expiration give adequate outcomes.

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**Low Tidal Volume Ventilation –**

One of the clinical hallmarks of ARDS is decreased respiratory system compliance. This is caused by atelectasis and flooding of alveoli and by increased surface tension at air-fluid interfaces. Initially when traditional tidal volumes of 10-15ml/kg were used in patients with ARDS receiving mechanical ventilation, the airway pressures were raised with over

distention of less affected lung regions.

As a result, there was increased pulmonary vascular permeability, alveolar haemorrhage, intrapulmonary shunt and diffuse radiographic infiltrates. When ventilation with 34 small tidal volume, 6ml/kg and limited airway pressures were used, ventilator-associated lung injury

from overdistention was reduced with a side effect of hypercapnia. A large scale randomized controlled trial sponsored by the National Institute of Health conducted by ARDS network compared conventionally tidal volume ventilation and low volume ventilation and noted that there was a decrease in Mortality with 31% in low volume ventilation

compared to 40% with conventional volume ventilation. Hence it is the low tidal volume 6ml/kg ventilation with adequate high PEEP that minimizes FiO2 and maximizes PaO2 with minimum ventilator-associated lung injury.

**Ventilation associated complications-**

Ventilator-associated lung injury is a common phenomenon due to repeated alveolar overdistention and recurrent alveolar collapse which can be minimized with adequate PEEP and low tidal volume ventilation. Other complications are Ventilator-associated pneumonia, upper airway injuries, sinusitis, tracheomalacia, tracheal stenosis.

## MATERIAL AND METHODS

This is a prospective observational study and study was carried out in patients who fulfilled the AECC definition of ARDS and admitted in the Intensive care unit in King George Hospital, Visakhapatnam. The study was conducted between July 2018 and October 2020 for a period of 2 years and two months. This study has got the approval from IEC, KING GEORGE HOSPITAL, AMC vide Regd No EC/NEW/Inst/KGH/2019/337. SERIAL NO: 96/IEC AMC//OCT/ 2020.

**Inclusion criteria:**

- All patients that fulfilled AECC criteria for ARDS
- Acute onset of bilateral chest infiltrates on chest radiograph
- PaO2/FiO2<300 for ALI,<200 for ARDS
- Absence of left atrial hypertension or cardiac failure (assessed clinically or echocardiographically)

**Exclusion criteria:**

- Evidence of heart failure clinically or echocardiographically.
- Patients under the age of 18 were also excluded.
- Chronic kidney disease with fluid overload states
- Chronic liver disease with fluid overload states
- Those who are not willing to participate in the study.

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## METHODOLOGY

This study was conducted at King George hospital, Visakhapatnam. after taking written informed consent from the patients and after getting Approval from ethical committee. Proformas will be filled up during inclusion of patients which will contain epidemiological information (age, sex, occupation, and place), questionnaires for risk factor evaluation for Acute respiratory distress syndrome, information of Clinical presentation and clinical signs.
The following set of investigations are asked for the patients in the Study.:
Complete hemogram, Renal function tests, Urine analysis, Serum electrolytes ,Chest X-ray,Electrocardiography 12 lead, 2D echocardiography ,CT chest, Viral markers, Blood culture sensitivity, Urine culture sensitivity, Serum amylase and lipase, Arterial blood gas analysis.

## STATISTICAL ANALYSIS:

Data was entered in Microsoft MS Excel, and the analysis done in MS Excel and
SPSS. categorical data is expressed in Proportions. Descriptive analysis was carried out by standard deviation in quantitative variable, proportion and categorical variable. Data is
also represented using appropriate bar diagrams and pie diagrams.
***For Statistical analysis, IBM SPSS version 22 was used.***

## Ethical consideration:

The study evaluated the clinical characteristics and outcomes of patients with ARDS. Prior permission was taken from Institutional Ethics Committee, Andhra Medical College, Visakhapatnam. The evaluation was done by history, physical examination and laboratory investigations. Informed consent was taken from the patient and his/her attenders after
Explaining about the study. Participation was voluntary and “standard care” was provided even if the patient did not agree to participate in the Study.
**There are no conflicts of interest.**

## RESULTS

Fifty patients that met the predefined criteria were enrolled for the study. Analysis of data was performed using the IBM SPSS version 22 software.

**TABLE 2. GENDER DISTRIBUTION**

| | FREQUENCY | PERCENTAGE |
|---|---|---|
| **MALE** | 26 | 52 |
| **FEMALE** | 24 | 48 |
| **TOTAL** | 50 | 100 |

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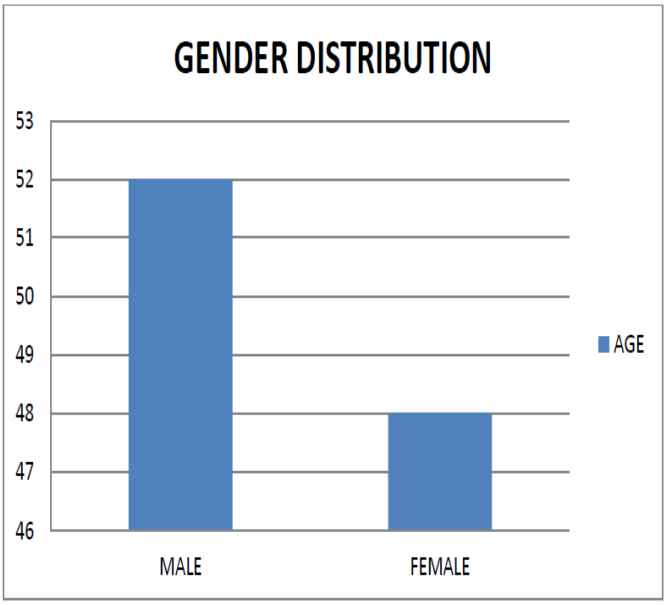
**Figure 1 – Gender Distribution in ARDS**

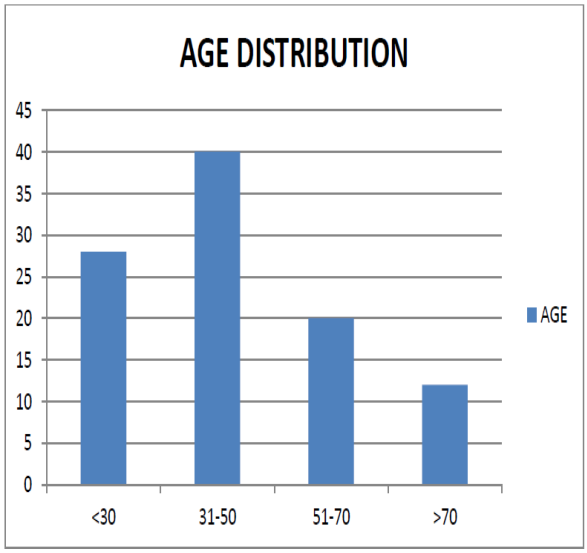



Among 50 patients admitted with ARDS, 26(52%) were male, and 24(48%) were female. ***The male compromised slightly more than half the study group at 52%***, and the rest were female.

**TABLE 3.AGE DISTRIBUTION IN ARDS**

| AGE | FREQUENCY | PERCENTAGE |
|---|---|---|
| **<30** | 14 | 28 |
| **31-50** | 20 | 40 |
| **51-70** | 10 | 20 |
| **>70** | 6 | 6 |

**Figure 2 - AGE DISTRIBUTION IN ARDS**



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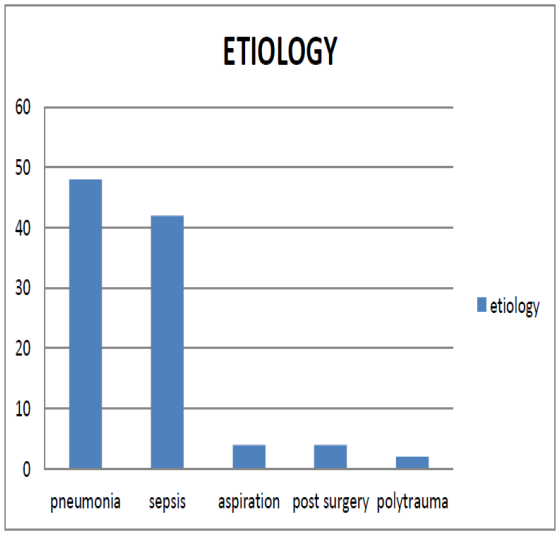
Among 50 patients, 14(28%) were in the age group of <30, 20(40%) were in the age group of 31- 50, 10(20%) were in the age group of 51-70, 6(12%) were in the age group of 66-80.

***The bulk of the patients were within the 32-50 year age at 40 per cent.***

**TABLE 4.ETIOLOGY OF ARDS**

| ETIOLOGY | NUMBER OF CASES | PERCENTAGE OF CASES |
|---|---|---|
| DIRECT | 26 | 52 |
| Pulmonary Infection | 24 | 48 |
| Aspiration | 2 | 4 |
| INDIRECT | 24 | 48 |
| Sepsis | 21 | 42 |
| Severe non-thoracic trauma | 1 | 2 |
| Post abdominal surgery | 2 | 4 |

**Figure 3 – ETIOLOGY OF ARDS**




Among 50 patients, 24(48%) had pneumonia, 21(42%) had sepsis, 2(4%) had Aspiration, 2(4%) had post abdominal surgery, 1(2%) had severe non-thoracic trauma.

***The most common etiological factor for ARDS in our study was pulmonary Infection followed closely by Sepsis.***

**TABLE 5.DISRIBUTION OF CAUSES OF ARDS**

| CAUSE | FREQUENCY | PERCENTAGE |
|---|---|---|
| DIRECT | 26 | 56 |
| INDIRECT | 24 | 44 |

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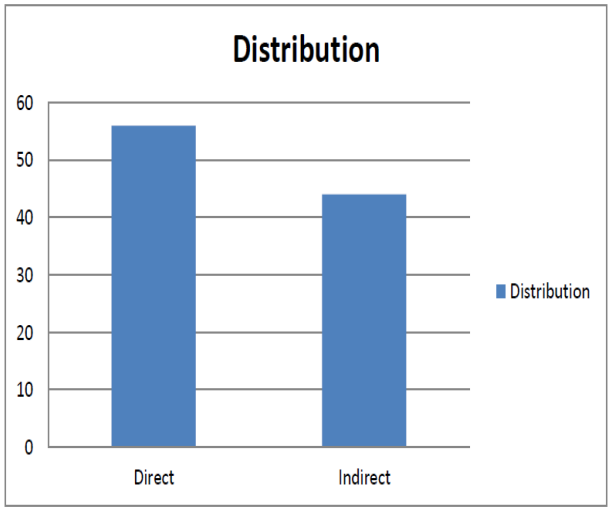
**Figure 4. DISTRIBUTION OF CAUSES OF ARDS**

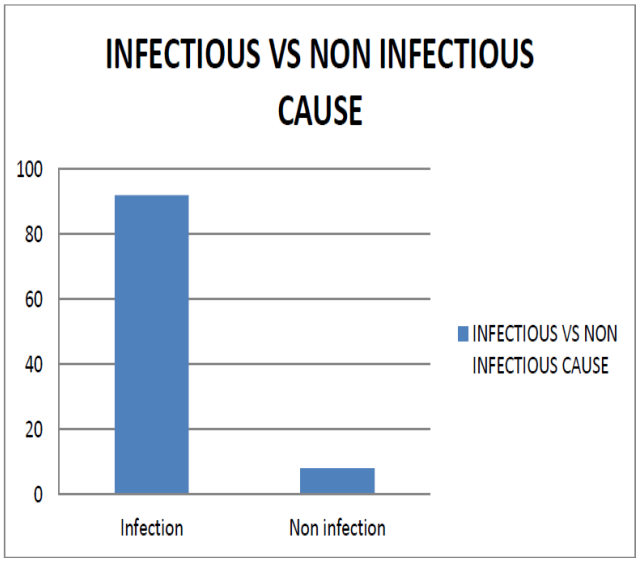



Among 50 patients, 26(56%) attributed to Direct causes and 24(44%) attributed to Indirect causes. In 56% of the patients in the study, ARDS could be attributed to direct causes of predominantly pulmonary Infection.

**TABLE 6.INFECTIOUS VS NON-INFECTIOUS CAUSES OF ARDS**

| CAUSE | FREQUENCY | PERCENTAGE |
|---|---|---|
| INFECTION | 46 | 92 |
| NON INFECTION | 4 | 8 |

**Figure 5. INFECTIOUS VS NON-INFECTIOUS CAUSES OF ARDS**




Among 50 patients,46(92%) cases caused by infectious aetiology and 4(8%) cases caused by non-infectious aetiology. Ninety-two per cent of the cases of Acute Respiratory distress syndrome in this study were secondary to infectious causes.

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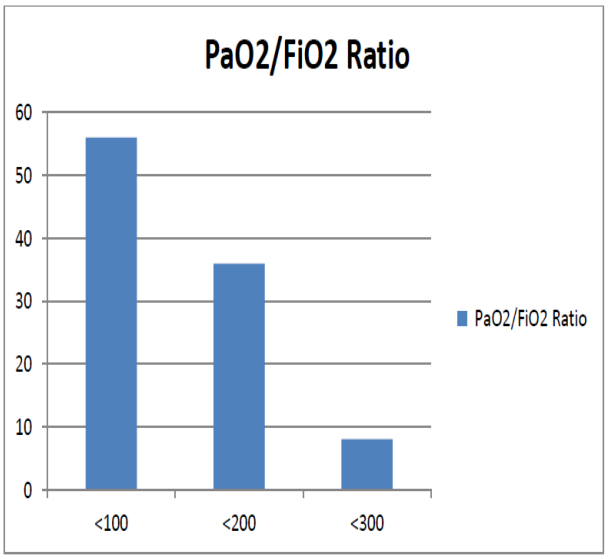
**Table 7 – PaO2/FiO2 ratio percentage in ARDS**

| PaO2/FiO2 | FREQUENCY | PERCENTAGE |
|---|---|---|
| <100mmHg | 28 | 56 |
| <200mmHg | 18 | 36 |
| <300mmHg | 4 | 8 |
| TOTAL | 50 | 100 |

**Figure 6- PaO2/FiO2 ratio percentage in ARDS**

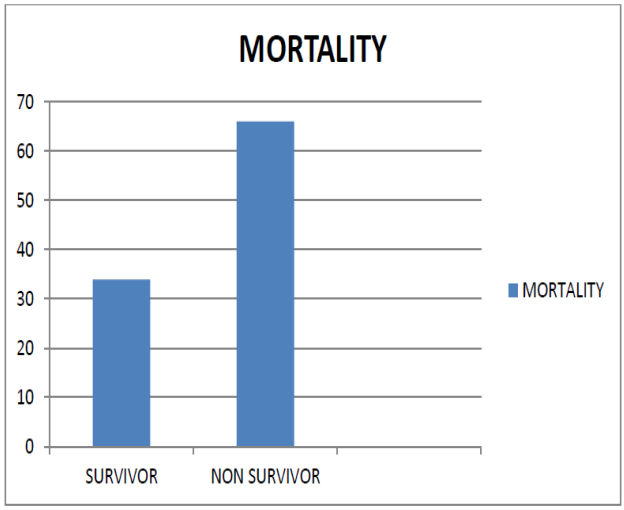



Among 50 patients, 56% had intial PaO2/FiO2 of <100 mmHg, 36% had PaO2/FiO2 of <200, 8% had PaO2/FiO2 of <300 mmHg.

**TABLE 8.MORTALITY PERCENTAGE**

| FINAL OUTCOME | FREQUENCY | PERCENTAGE |
|---|---|---|
| DISCHARGE | 17 | 34 |
| DEATH | 33 | 66 |

**Figure 7. MORTALITY PERCENTAGE**



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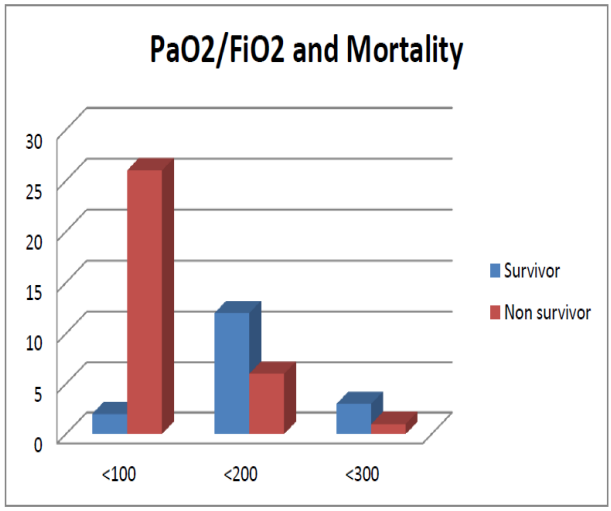
In this study out Of the 50 patients, 33 patients succumbed to their illness-the Mortality being 66 percent.

**TABLE 9.PaO2/FiO2 Ratio and Mortality**

| PaO2/Fio2 | Non-survivors | Survivors | Total |
|---|---|---|---|
| <100 | 26(92.9%) | 2(7.1%) | 28 |
| <200 | 6(33.3%) | 12(66.7%) | 18 |
| <300 | 1(25%) | 3(75%) | 4 |
| Total | 33 | 17 | 50 |

**Figure 8. PaO2/FiO2 Ratio and Mortality**




The above table and chart depict the survival status at different values of the Pao2/FiO2 ratio on admission. There is a trend towards increasing Mortality with lower Pao2/Fio2 values.
(p-value - <0.01 by the Fisher's Exact test)

**TABLE 10.ALBUMIN AND OUTCOME**

| Albumin(g/dl) | Non-survivors | Survivors | Total |
|---|---|---|---|
| <2 | 7(77.8) | 2(22.2) | 9 |
| 2.1-2.5 | 13(81.8) | 3(18.2) | 16 |
| 2.6-3.0 | 8(61.5) | 5(38.5) | 13 |
| >3.1 | 5(41.7) | 7(58.3) | 12 |
| Total | 33 | 17 | 50 |

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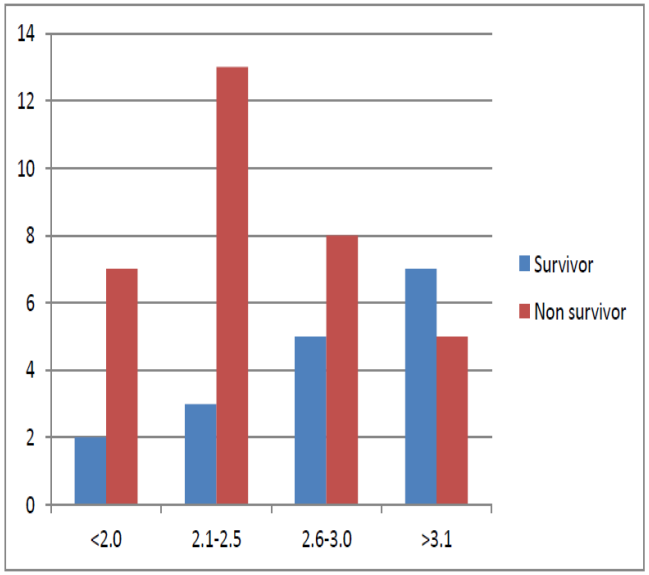
**FIGURE 9.ALBUMIN AND OUTCOME**



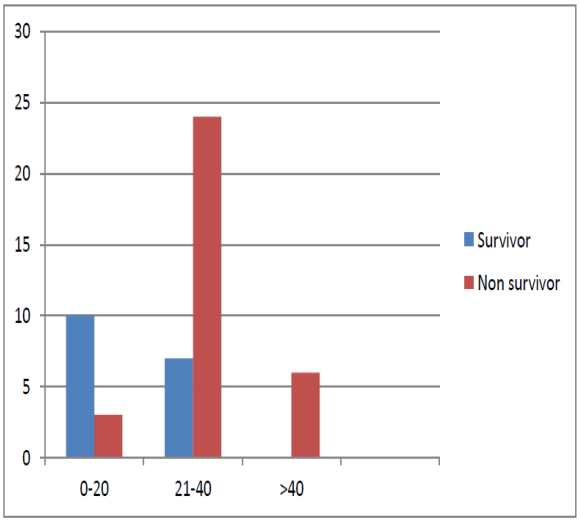

The percentage of non-survivors were higher in classes with lower Albumin. The mean Albumin among non-survivors was lower than of non-survivors,2.5 vs 2.8g/dl(p-0.06).

**SAPS II SCORE AND OUTCOME**

**TABLE 11.SAPS II SCORE AND OUTCOME**

| SAPS II SCORE | Non-survivor | Survivor | Total |
|---|---|---|---|
| 0-20 | 3(23.1%) | 10(76.9%) | 13 |
| 21-40 | 24(77.4%) | 7(22.6%) | 31 |
| >40 | 6(100) | | |
| Total | 33 | 17 | 50 |

**FIGURE 10.SAPS II SCORE AND OUTCOME**



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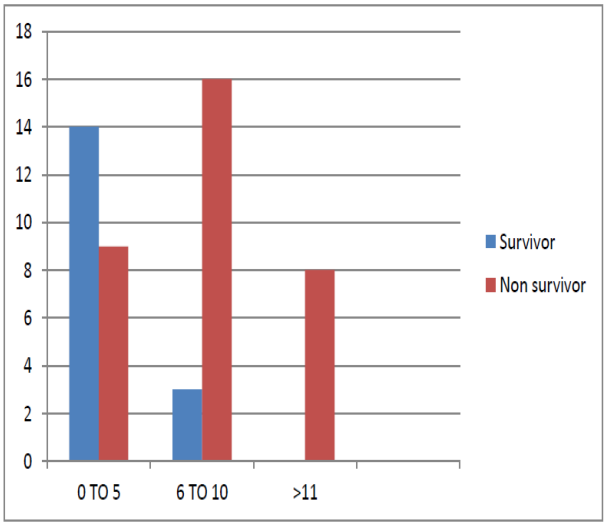
Patients were divided into classes with increasing SAPS II scores. It was observed that the greater the score, the higher was the Mortality in that group.(p-value <0.001).

**SOFA SCORE AND OUTCOME**

**TABLE 12.SOFA SCORE AND OUTCOME**

| SOFA SCORE | Non-survivors | survivors | Total |
|---|---|---|---|
| 0-5 | 9(39.1%) | 14(60.9%) | 23 |
| 6-10 | 16(84.2%) | 3(15.8%) | 19 |
| >11 | 8(100%) | - | 8 |
| Total | 23 | 17 | 50 |

**FIGURE 11.SOFA SCORE AND OUTCOME**




It was observed that amongst patients with higher SOFA scores, the mortality was greater compared o those with lower values on the scoring system(p-value -0.001).

**LUNG INJURY SCORE AND OUTCOME**

**TABLE 13.LUNG INJURY SCORE AND OUTCOME**

| LIS SCORE | Non-survivors | survivors | Total |
|---|---|---|---|
| 0.1-2.5 | 11(29.3%) | 17(60.7%) | 28 |
| >2.5 | 22(100%) | - | 22 |
| Total | 33 | 17 | 50 |

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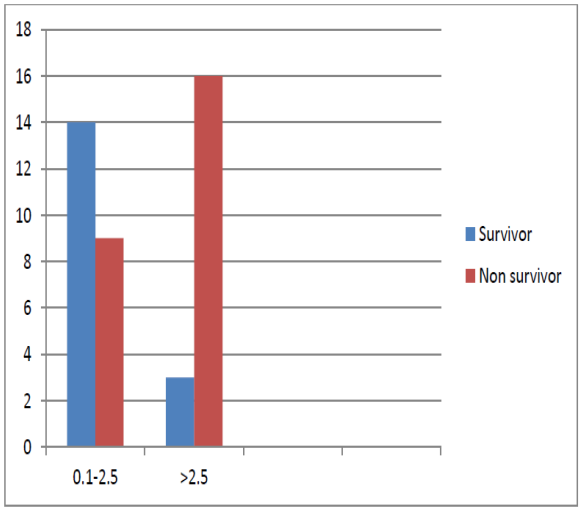
**FIGURE 12 .LUNG INJURY SCORE AND OUTCOME**



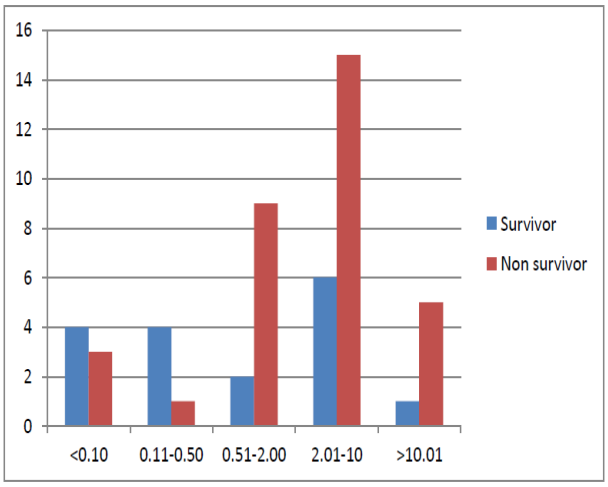

Based on lung injury score, patients were divided into those that have mild to moderate and severe lung injury. All the patients who recovered fell into the former category. two-thirds of patients that succumbed to illness had severe lung injury scores.(p-value-<0.001).

**PROCALCITONIN AND MORTALITY**

**TABLE 14. PROCALCITONIN AND MORTALITY**

| Procalcitonin | Non-survivors | survivors | Total |
|---|---|---|---|
| <0.10 | 3(42.9%) | 4(52.1%) | 7 |
| 0.11-0.50 | 1(20%) | 4(80%) | 5 |
| 0.51-2.00 | 9(81.8%) | 2(18.2%) | 11 |
| 2.01-10 | 15(71.4%) | 6(28.6%) | 21 |
| >10.01 | 5(83.3%) | 1(16.7%) | 6 |
| Total | 33 | 17 | 50 |

**Figure 13. PROCALCITONIN AND MORTALITY**



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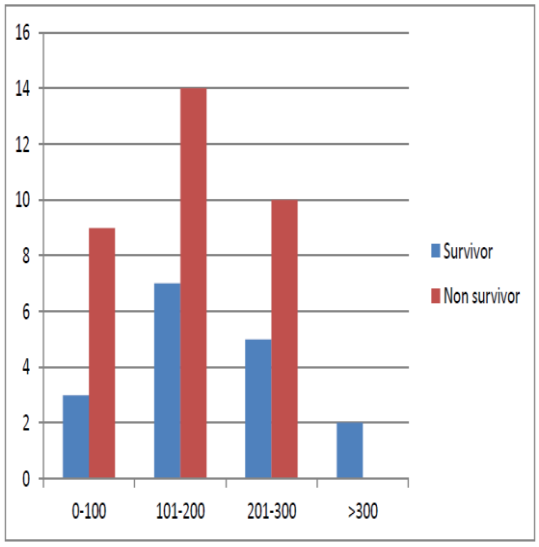
With increasing values of procalcitonin, the percentage of non-survivors in the group increases, suggesting that procalcitonin may be used as a marker to predict prognosis. However, the difference was statistically insignificant(p-<0.069).The mean procalcitonin value amongst the non-survivors and survivors ere 9.9ng/ml and 1.7ng/ml, respectively. (p-0.3).

**CRP DISTRIBUTION AND MORTALITY**

**TABLE 15.CRP AND MORTALITY**

| CRP | Non-survivor | survivors | Total |
|---|---|---|---|
| 0-100 | 9(75%) | 3(25%) | 12 |
| 101-200 | 14(66.7%) | 7(33.3%) | 21 |
| 201-300 | 10(66.7%) | 5(33.3%) | 15 |
| >300 | 0 | 2(100%) | 2 |
| Total | 33 | 17 | 50 |

**FIGURE 14.CRP AND MORTALITY**




It was observed that with increasing values of CRP, the Mortality also increases. However, the observation was statistically insignificant(p-0.2).

**DURATION OF ICU STAY AND VENTILATION**

**TABLE 16.MEAN DURATION OF ICU STAY AND VENTILATION**

| Mean(days) | Non-survivors | Survivors |
|---|---|---|
| Length of ICU stay | 7.42(4.5) | 10.12(3.0) |
| Duration of ventilation | 5.55(4.3) | 5.65(2.2) |

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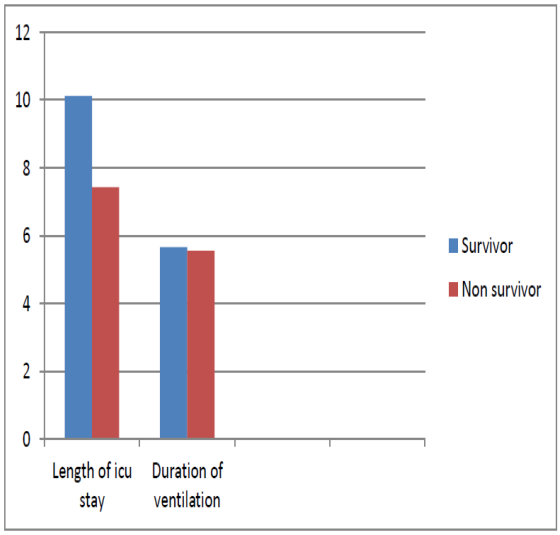
**FIGURE 15.MEAN DURATION OF ICU STAY AND VENTILATION**

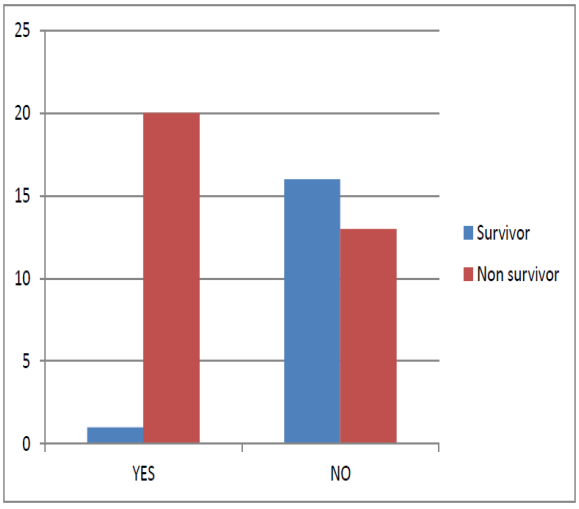



Between survivors and Non-survivors, there was no significant the difference in the length of ICU stay and duration of ventilation.

**TABLE 17.INOTROPIC SUPPORT AND OUTCOME**

| Inotrope support | Non-survivors | survivors | Total |
|---|---|---|---|
| Yes | 20(60.6%) | 1(0.005%) | 21 |
| No | 13(39.4%) | 16(99.5%) | 29 |
| Total | 33 | 17 | 50 |

**FIGURE 16.INOTROPIC SUPPORT AND OUTCOME**




There was a significant increase in the Mortality in patients that were prescribed inotrope support when compared to those who were not. ($p<0.001$).

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**Table 18.The difference in baseline physiological and laboratory parameters between survivors and non-survivors**

| Variable | Non-survivor | Survivor | P-value | Confidence interval |
|---|---|---|---|---|
| age | **43.6(19.0)** | **42.5(14.9)** | **0.8** | **-9.5,11.7** |
| PEEP | **8.6(2.8)** | **5.6(1.1)** | **<0.001** | **1.5,4.4** |
| MAP(mmHg) | **82.7(15.3)** | **87.0(10.3)** | **0.3** | **-12.6,4.0** |
| Ph | **7.16(1.1)** | **7.43(0.12)** | **0.3** | **-0.8,0.2** |
| S.Bicarbonate(meq/L) | **21.7(5.9)** | **25.8(3.5)** | **0.013** | **-7.2,-0.9** |
| Hematocrit | **31.9(8.0)** | **33.2(7.2)** | **0.5** | **-5.9,3.3** |
| WBC(thousand/mm3) | **11.8(8.9)** | **0.7(4.0)** | **0.3** | **2.4,6.7** |
| Platelet count(lakhs/mm3) | **1.6(1.1)** | **2.4(1.2)** | **0.02** | **-1.5,-1.3** |
| S.Creatinine(mg/dl) | **1.3(0.8)** | **0.9(0.3)** | **0.05** | **0,0.8** |
| S.Bilirubin(mg/dl) | **2.0(2.0)** | **1.2(1.2)** | **0.14** | **-0.6,0.02** |
| S.Albumin(g/dl) | **2.5(0.5)** | **2.8(0.6)** | **0.06** | **-0.28,1.9** |
| procalcitonin | **9.8(32.0)** | **1.7(2.8)** | **0.3** | **77.1,107.0** |
| CRP | **145.9(77.1)** | **186.6(107.0)** | **0.11** | **-95.7,10.2** |
| SOFA | **8.39(3.8)** | **4.24(1.6)** | **<0.001** | **2.1,6.1** |
| SAPS II | **32.61** | **19.94(6.8)** | **<0.001** | **0.6,1.1** |
| LIS | **2.73(0.4)** | **1.83(0.2)** | **<0.001** | **5.7,19.5** |

## DISCUSSION

ARDS is an acute form of respiratory failure characterized by refractory hypoxemia and non-cardiogenic pulmonary oedema.In spite of many studies done on aetiology and factors
predicting Mortality, the only proven therapy is effective ventilation strategy. 50 patients admitted to the ICU or shifted from ward to ICU who satisfied the inclusion or exclusion
criteria were enrolled in the study. The study was done from September 2016 to February 2018. All the patients with acute a respiratory failure who satisfy Berlin criteria were identified as
ARDS and they were followed up daily in the ICU and the aetiology and outcome of ARDS in terms of number of days on ventilator, the number of days in ICU and Mortality is noted.

## ETIOLOGY OF ARDS

The primary objective of this study is to study the aetiology of ARDS. As per our study, we found that pneumonia (48%) and Sepsis (42%) were the two commonest etiologies for ARDS.
These findings are consistent with studies done previously from other centres.
A study conducted by vigg et al.43 in Hyderabad had made similar observations with primary pulmonary Infection is the most common cause of ARDS.

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A Prospective observational study was done in Spain under the name ALIEN ( Acute Lung Injury: Epidemiology and Natural history). A total of 255 patients fulfilled the criteria of ARDS
with an incidence of 7.2/ 1,00,000 population per year. Pneumonia comprising 42% by Sepsis 31%, Trauma 9%, Aspiration 8%, Pancreatitis 4%. Mortality was 47%.[44]

A study was done on the prevalence of direct vs indirect aetiology of ARDS by Dr Akash Teja Durbesula, Dr KB Chetan Reddy, Dr Gangaram Usham, Dr Rajesh Kumar Meriga,
Dr T Venkata Krishnan, in Andhra Pradesh. It was a prospective observational study conducted from November 2015 to May 2016. It was observed that ARDS was mostly secondary to infectious causes 92%, and the most common aetiology of ARDS was direct cause 52%, followed by indirect cause 48%.
Pulmonary infections were the common direct cause comprising 48% and Sepsis comprising 42% [45].In this study 56% of the cases are attributed to Direct causes predominantly pulmonary infection and 44% are attributed to indirect causes. Ninety-two per cent of cases of ARDS in this study were secondary to infectious causes.

**GENDER ASSOCIATION WITH MORTALITY**

In our study, 52% of the patients were males, and the rest were females. The mean age of patients enrolled in our study was 43.26 years. There is no statistically significant increase in
mortality was seen with increasing age in our study The other studies also found no statistically significant association between gender and Mortality.
Bhadade et al. [46] had found a mortality rate of 73% in female and 51% in male with a p-value of 0.23 which was not statistically significant. Surendra Sharma et al.[47] found that the mortality rate in male was 61% and 38.9% in the female with p-value 0.7 which was not statistically significant. Surendra Sharma et al.[47] found that the mortality rate in male was 61% and 38.9% in the female with p-value 0.7 which was not statistically significant.

**PaO2/Fio2 association with Mortality**

In our study 92.9% of the patients who expired, had initial PaO2/FiO2 ratio <100mmHg, 33.3% of the expired patients had PaO2/FiO2 ratio of <200 mmHg and 25% of the patients who
expired had PaO2/FiO2 ratio <300 mmHg. The p-value of this is <0.001, which is statistically significant. In the study done by Bhadade et al.[48] mortality was 62.7% in
PaO2/FiO2 ratio of <200 mmHg and 12.5 % in PaO2/FiO2 the ratio of >200 with a p-value of 0.04 which is statistically significant.

In a study done by Bellani G et al [49], Mortality in those with PaO2/FiO2 <300 mmHg was 34.9%, in those with PaO2/FiO2 <200 was 40.3%, PaO2/FiO2 <100 was 46.1% with significant p-value, which is similar to the findings in our study.
In a study done by Lalit et al 50, mortality rate in PaO2/FiO2 <300 was 8.5%, 22.5% in PaO2/FiO2 <200 and 45% in PaO2/FiO2 <100 mmHg with p value <0.005 which was
statistically significiant. Similar to the other studies, we found that initial PaO2/FiO2
ratio < 100 had more Mortality compared to those with values <200 and <300, which was statistically significant and can be considered as a predictor of Mortality.

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**ASSOCIATION OF DURATION OF MECHANICAL VENTILATION WITH MORTALITY**

In our study the average duration of mechanical ventilation in survived/discharged patients is 5.65 days and in the expired, it is 5.55 days. The p values of these are 0.73 and 0.72 respectively which are not statistically significant. In a study done by Akash Teja Durbesula et al [51], the average duration of mechanical ventilation is 5.5 in non-survivors and 5.6 in survivors with p values which were not statistically significant.

Our study also did not find any statistically significant association in the average duration of mechanical ventilation between survivors and non-survivors.

**Association of Length of ICU stay and Mortality**

In our study, the average length of ICU stay in survived /discharged patients is 10.12 days and 7.42 days in non survivors. The p values of these are 0.15 and 0.14 respectively which are not statistically significant. In Akash Teja Durbesula et al [52] study the average length of ICU stay is 7.42 in non-survivors and 10.12 in survivors.

Our study also did not find any statistically significant association in the average duration of ICU stay between survivors and non-survivors In our study the parameters that had a statistically significant association with Mortality include PEEP, Pao2/Fio2, Serum Bicarbonate, platelet count and use of Inotropes. The clinical scores – LIS, SAPS II and SOFA, had a highly
significant association with Mortality.

A study was done by Monchi et al. [53] to identify early predictors of survival in ARDS showed a significant association between Mortality and SAPS II score, serum bicarbonate and use
of Inotropes. our study also indicates increasing Mortality in patients with higher procalcitonin levels were noted in our the study, the association, however, was statistically found similar associations.

In our study, it was noted that the percentage of non-survivors were higher in classes with lower Albumin. The mean Albumin among non-survivors was lower that of survivors 2.5 versus 2.8 g/dl (p-0.06).

A study was done by Maskara et al. conducted in CMC Vellore showed that lower albumin levels are associated with greater mortality and higher lung injury scores [54].

In our study, the mean procalcitonin value amongst the non -survivors and survivors were 9.8ng/ml and 1.7ng/ml(p-0.3).The percentage of Non-survivors increases with an increase in procalcitonin, suggesting that procalcitonin may be used as a marker to predict diagnosis. However, the difference was statistically insignificant(p-0.069).

A prospective observational study was done by Dr Surendra K Sharma, Anunay Gupta, Ashutosh Biswas in AIIMS, Delhi, India from July 2010 to June 2012 which included aetiology, outcome and predictors of Mortality a showed Elevated levels of inflammatory markers such as hsCRP, Procalcitonin are other predictors of Mortality[55].

A study conducted in Taiwan in patients with ARDS secondary to severe community-acquired pneumonia concluded that procalcitonin analyzed wihin 72 hours of the onset of ARDS
predicted Mortality[56].

The Mortality rate in our study is 66%

In an Observational a prospective study was done in Maharashtra by Dr RR Bhadade, RA de Souza, MJ Harde – with 58 cases of ARDS in MICU mortality was 57% [57.]

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A study conducted in RICU in North India noted a mortality rate of 47.8%.[58]
In a review published of 101 cases of ARDS the average mortality was 50%, with reported Mortality varying from 30% to 70%.[59]
No significant difference in Mortality was observed between the patients who developed ARDS secondary to direct and those developed the ARDS as a result of indirect causes.

**Table19.Mortality percentage in various studies.**

| **Study Mortality** | Akash Teja Durbesula et al(41) |
|---|---|
| 62% | ALIEN Study(39) |
| 40% | Lalit Kumar et al(38) |
| 41.8% | |

## CONCLUSIONS

Pulmonary Infection (48%) followed by Sepsis (42%) were the most common causes for ARDS in this study.
56% of cases were attributable to direct causes, and 94% of cases were secondary to infectious etiologies
Factors showing association with Mortality Pao2/Fio2 showed a strong correlation with Mortality.
Clinical scores-SOFA, SAPS II, and LIS had a statistically significant association with Mortality.
CRP and procalcitonin did not show a significant correlation with Mortality
Outcome-mortality in our study was 66%.

## LIMITATIONS

1-Small sample size
2- shorter period of Time
3- The predictors of Mortality other than PaO2/FiO2 ratio and
gender could have been studied more extensively.

## SUMMARY

This was a prospective observational study to identify the distribution of aetiology, the clinical behaviour, the factors affecting the course of illness and the eventual outcome of patients with ARDS amongst the Indian population. During the predefined study period, a total of fifty patients that met the inclusion criteria were enrolled.pulmonary infection(48%) followed by Sepsis (42%) were the most common causes of ARDS in this study. Fifty-six per cent of cases were
attributable to direct causes, and ninety-four per cent of caseswere secondary to infectious etiologies. The initial PaO2/FiO2 ratio <100 mm was found to be associated with higher mortality
in our study, the same has been mentioned in the other studies too. Hence it can be used as a predictor of Mortality.
No gender predisposition has been noticed with Mortality of ARDS which was consistent with other studies.
Various clinical scores SOFA, SAPS II and LIS score showed a strong correlation with Mortality. The Mortality in our study was sixty-two per cent, which is comparable to the figures
reported in the literature.

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**Recommendations** – As Pneumonia and Sepsis, were found to be the most common causes of ARDS, if any of these patients have a new onset/ worsening dyspnea, then ARDS
should be considered and diagnosed using Berlin criteria. Early recognition and initiation of mechanical ventilation will decrease Mortality. Early proning should be considered in all
patients who are difficult to oxygenate adequately.

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