# ROLE OF THE DECAF SCORE IN PREDICTING IN HOSPITAL MORTALITY IN ACUTE EXACERBATION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE

**Dissertation submitted in partial fulfillment of the Requirement for the award of the Degree of**

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**THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY**

**CHENNAI, TAMILNADU, INDIA**

**MAY 2022**

# CERTIFICATE FROM THE DEAN

This is to certify that the dissertation entitled **"ROLE OF THE DECAF SCORE IN PREDICTING IN HOSPITAL MORTALITY IN ACUTE EXACERBATION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE"** is the bonafide work of **DR. LEO CLINGTON A** in partial fulfilment of university regulations of the Tamil nadu Dr.M.G.R medical university, Chennai for M.D General Medicine Branch I examination to be held in MAY 2022.

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# DECLARATION

I, Dr. LEO CLINGTON A solemnly declare that this dissertation **"ROLE OF THE DECAF SCORE IN PREDICTING IN HOSPITAL MORTALITY IN ACUTE EXACERBATION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE"** is a bonafide record of work done by me at the department of General medicine, Govt. Rajaji Hospital, Madurai under the guidance Professor of Dr.S.DAVID PRADEEP KUMAR.M.D., Department of general medicine, Madurai Medical college,Madurai. This dissertation is submitted to the Tamil nadu Dr.M.G.R medical university, Chennai in partial fulfillment of the rules and regulations for the award of Doctor of Medicine (M.D.), General Medicine Branch -I; examination to be held in MAY 2022.

Place: Madurai **DR. LEO CLINGTON A**

Date :

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# INTRODUCTION

# INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a preventable and treatable disease that is characterised by persistent airflow limitation which is usually progressive and associated with enhanced chronic inflammatory response in the airways and lung to noxious particles or gases[1].

COPD is the third most common cause of death worldwide after Ischemic heart disease, Cerebrovascular disease. Exacerbations and comorbidities contribute to overall severity in COPD patients[1].

COPD includes emphysema, an anatomically defined condition characterized by destruction of the lung alveoli with air space enlargement; chronic bronchitis, a clinically defined condition with chronic cough and phlegm; and small airway disease, a condition in which small bronchioles are narrowed and reduced in number[4]. The presence of chronic airflow restriction, as shown by spirometry, is required for the classic definition of COPD, which frequently occurs in the context of noxious environmental exposures, most commonly cigarette smoking[5].

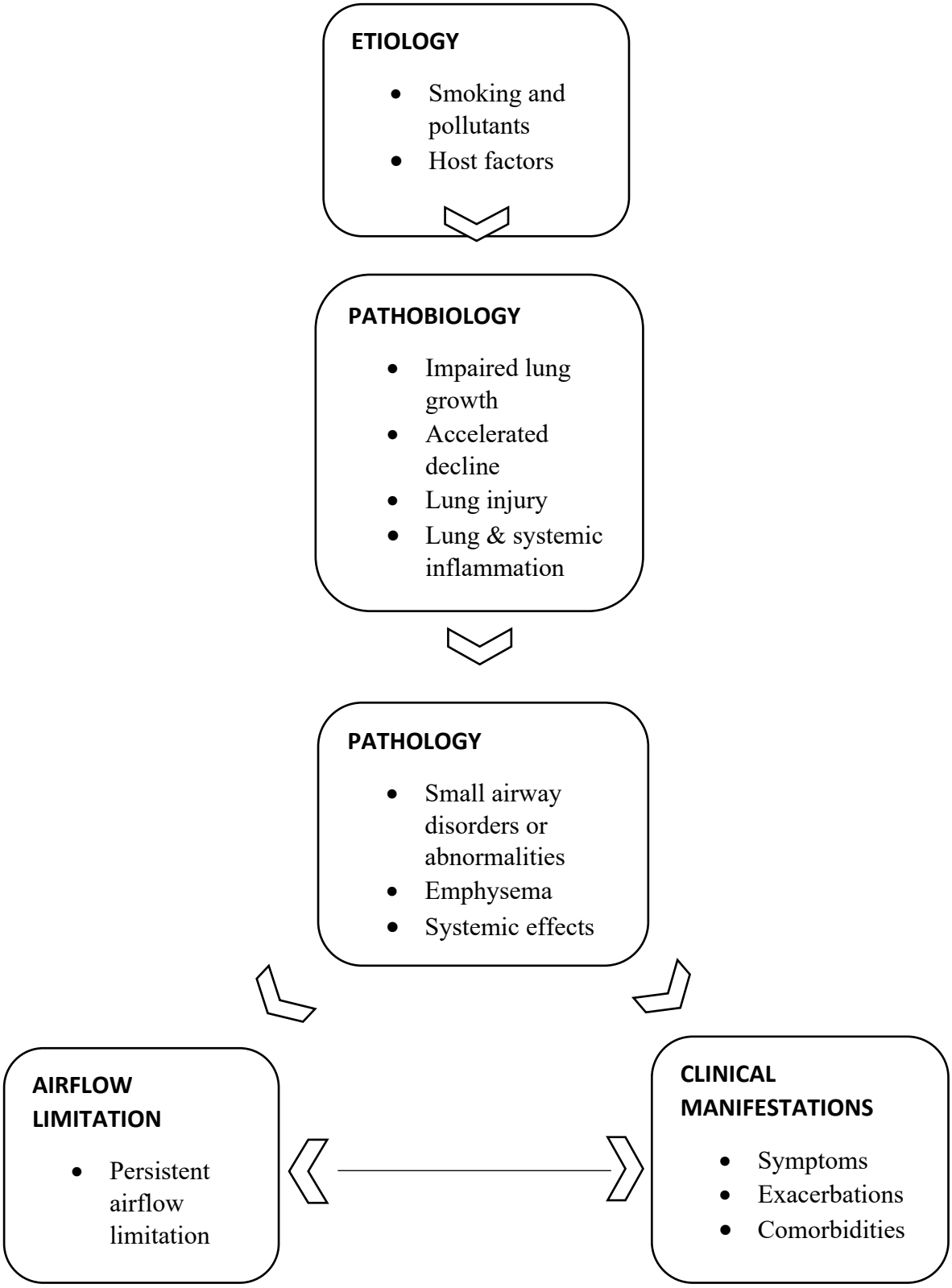



**Fig 1 . Etiology , Pathobiology and Pathology of COPD leading to airflow limitation and clinical manifestations[1]**

## RISK FACTORS OF COPD:

The higher prevalence of COPD among males is largely due to a historically higher incidence of smoking; however, the prevalence of COPD among females is growing as the gender gap in smoking rates has narrowed in the last 50 years. Cigarette smoke can harm large airways, small airways (less than 2 mm in diameter), and alveoli. Long-term exposure to smoke produced by biomass combustion, which is a widespread form of cooking in various countries, including ours, appears to be a substantial risk factor for COPD in women in such countries. The loss of cilia in the airway epithelium caused by cigarette smoke, as well as reduced macrophage phagocytosis, predisposes to bacterial infection with neutrophilia.

The intensity of cigarette smoking is expressed as pack-years (average number of packs of cigarettes smoked / day x the total number of years of smoking) Smoking cessation at a younger age[11] has a higher positive effect than smoking cessation when significant pulmonary function impairments have already occurred[5].

Long after smoking cessation, there is still an exuberant inflammatory response in end-stage lung disease, suggesting that cigarette smoke-induced inflammation both initiates the disease and establishes a

chronic process in susceptible individuals that can continue disease progression even after smoking cessation[2].

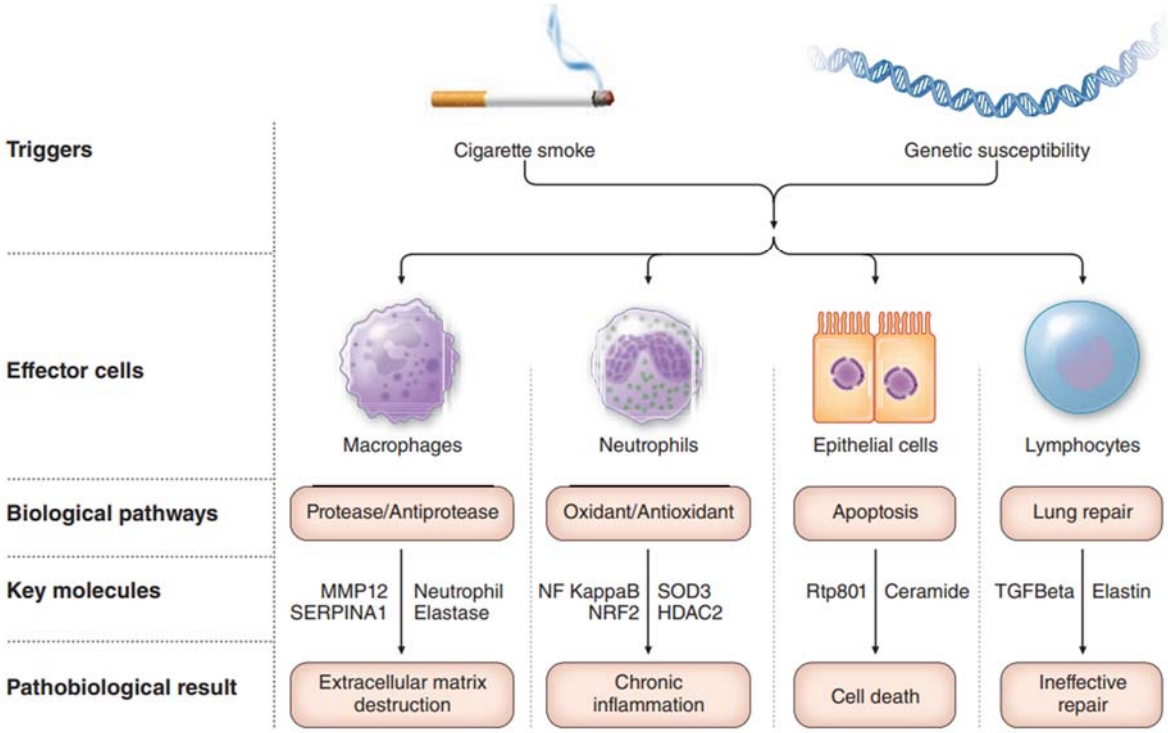



**Fig 2. Pathogenesis of COPD**

**CLINICAL MANIFESTATIONS OF COPD:**

Cough, sputum production, and exertional dyspnea are the three most common symptoms of COPD. Exertional dyspnea, also known as increased effort to breathe, heaviness, air hunger, or gasping, can appear gradually[9]. A careful history focused on typical physical activities and how the patient's ability to perform them has changed is the best way to elicit it.

The physical examination of the lungs in patients with more severe disease reveals a prolonged expiratory phase, which may include expiratory wheezing. Patients with severe airflow obstruction may also use accessory respiratory muscles, sitting in the characteristic "tripod" position to facilitate the actions of the sternocleidomastoid, scalene, and intercostal muscles[4]. Cyanosis, which is visible in the lips and nail beds, may develop in patients. Patients may also develop resting hypoxemia, necessitating the use of supplemental oxygen. Since the introduction of supplemental oxygen therapy, signs of overt right heart failure, known as cor pulmonale, have become less common.

**DIAGNOSIS OF COPD:**

The obstruction of airflow is a defining feature of COPD. Airflow obstruction with a decrease in FEV1 and FEV1 /FVC lung volumes may increase, resulting in an increase in total lung capacity, functional residual capacity, and residual volume, according to pulmonary function testing.

The FEV1/FVC ratio is chronically lowered in patients with COPD-related airflow obstruction. In contrast to asthma, COPD patients' lower FEV1 rarely responds well to inhaled bronchodilators, while improvements of up to 15% are typical.

The one of the important prognostic factor in COPD is degree of airflow obstruction and is the basis for the Global Initiative for Chronic Obstructive Lung Disease (GOLD) spirometric severity classification[1]

| GOLD STAGE | SEVERITY | SPIROMETRY |
|---|---|---|
| I | Mild | FEV1/FVC<0.7 and FEV1≥80% predicted |
| II | Moderate | FEV1/FVC<0.7 and FEV1≥50% but <80% predicted |
| III | Severe | FEV1/FVC<0.7 and FEV1≥30% but <50%predicted |
| IV | Very severe | FEV1/FVC<0.7 and FEV1<30% predicted |

**Table 1. (GOLD) spirometric severity classification**

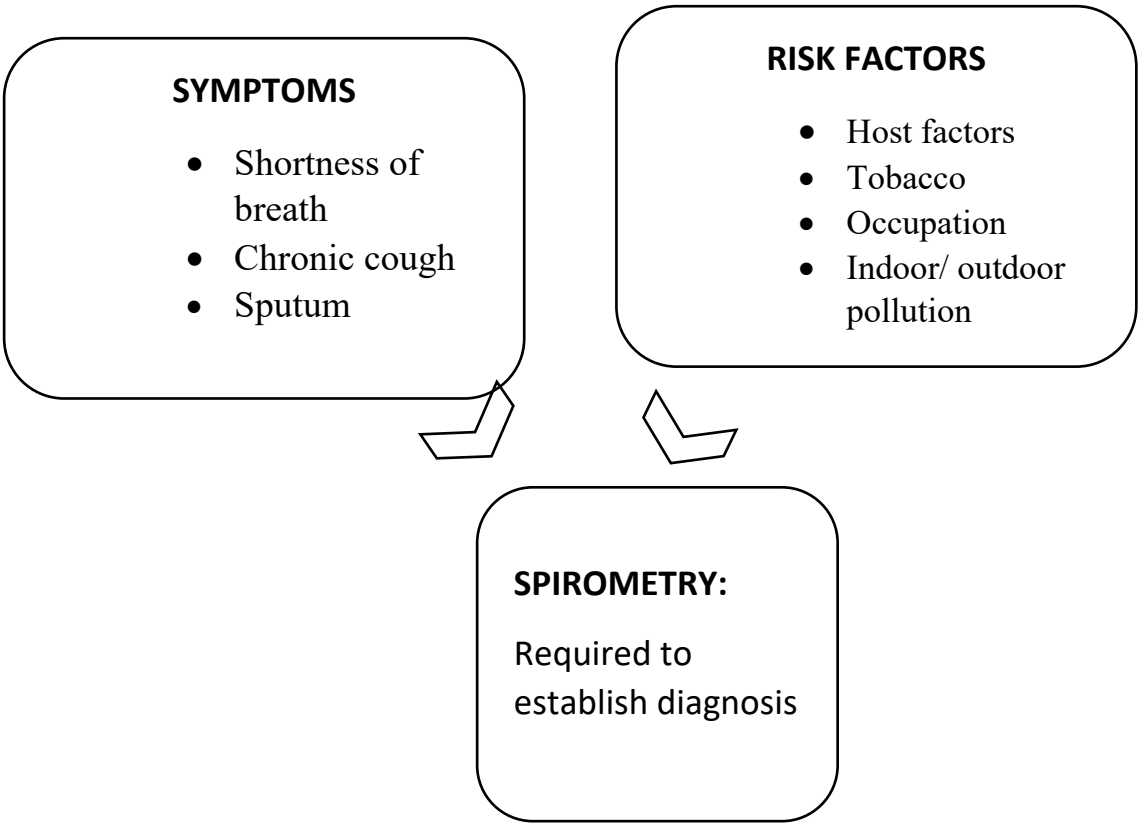



**Fig. 3. Pathways to the diagnosis of COPD[1]**

As COPD progresses, the main symptom is worsening dyspnea on exertion, which has an increasing impact on one's ability to perform vocational or avocational activities. In the most advanced stages, patients become breathless while performing simple daily activities. Exacerbations are becoming more frequent as airflow obstruction worsens. Cachexia, significant weight loss, bitemporal wasting, and diffuse loss of subcutaneous adipose tissue may accompany advanced disease.

Although the presence and severity of respiratory symptoms, exacerbations, emphysema, and hypoxemia[11] are generally correlated with the degree of airflow obstruction, the correlations are far from perfect. As a result, clinical features in each individual patient with COPD should be carefully assessed in order to determine the most appropriate therapies.

A multifactorial index (BODE)[20] incorporating airflow obstruction, exercise performance, dyspnea, and body mass index has been shown to be a better predictor of mortality rate than pulmonary function alone. Recently, the GOLD classification system was expanded to include respiratory symptoms and exacerbation history; these metrics are used to guide COPD treatment.

Arterial blood gas analysis and oximetry may reveal resting or exertional hypoxemia. Arterial blood gases, which measure arterial Pco2 and pH, provide additional information about alveolar ventilation and

acid-base status. The acute pH change with Pco2 is 0.08 units/10 mmHg and the chronic pH change is 0.03 units/10 mmHg. Knowing the arterial pH allows to categorise ventilatory failure, defined as Pco2 >45 mmHg, as acute or chronic, with acute respiratory failure being associated with acidemia[7]. The arterial blood gas is an important part of the evaluation of patients who present with exacerbation symptoms.

**EXACERBATIONS OF COPD :**

Exacerbations are a common occurrence in the course of COPD. Exacerbations are episodes of acute worsening of respiratory symptoms such as increased dyspnea, cough, wheezing, and/or changes in the amount and nature of sputum. Exacerbations become more common as airflow obstruction worsens; patients with severe (FEV<50 % predicted) or very severe airflow obstruction (FEV<30 % predicted) have 1-3 episodes per year on average.

A variety of factors may contribute to the final common pathway of airway inflammation and increased respiratory symptoms that characterise COPD exacerbations. According to studies, acquiring a new strain of bacteria is associated with an increased near-term risk of exacerbation, and bacterial infection/superinfection is involved in more than half of all exacerbations. Approximately one-third of COPD exacerbations are

caused by viral respiratory infections. No specific precipitant can be identified in a significant minority of cases (20–35 %).

The history should include quantification of the degree and change in dyspnea by inquiring about breathlessness during daily activities and the patient's typical activities. The patient should be asked about fever, sputum changes, and other symptoms such as wheezing, nausea, vomiting, diarrhoea, myalgias, and chills. Inquiring about the frequency and severity of previous exacerbations can provide important information; a history of previous hospitalisation is the single greatest risk factor for hospitalisation with an exacerbation.

Patients who have severe underlying COPD, are in moderate or severe distress, or have focal findings should have a chest x-ray or a chest CT scan[5]. Approximately 25% of x-rays in this clinical situation will be abnormal, with pneumonia and congestive heart failure being the most common findings. Patients with advanced COPD, a history of hypercarbia, changes in mental status (confusion, sleepiness), or those in severe distress should have an arterial blood-gas measurement[11]. The presence of hypercarbia, defined as a Pco2 >45 mmHg, has significant treatment implications. In contrast to its utility in the management of asthma exacerbations, measurement of pulmonary function has not been shown to be useful in the diagnosis or management of COPD exacerbations .

The presence of respiratory acidosis and hypercarbia, new or worsening hypoxemia, severe underlying disease, and those whose living situation does not allow for careful observation and the administration of prescribed treatment indicate the need for inpatient treatment of exacerbations.

For that particular hospitalisation, the mortality rate of patients requiring mechanical ventilation is 17–30%. Regardless of whether mechanical ventilation was required, the mortality rate for patients aged >65 admitted to the intensive care unit for treatment doubles over the next year to 60%. Following a COPD hospitalisation, approximately 20% of patients are re-hospitalized within 30 days, and % are hospitalised within a year. In the year following hospital discharge, mortality is around 20%.

Multiple prognostic indices associated with higher death rates in COPD have been investigated, including Forced Expiratory Volume in One Second[12], Patient age[13], Hypoxemia[14], Hypercapnia[15], Comorbidity, Pulmonary hypertension, and Body mass Index[16,17]. Few studies have been conducted to assess prognostic factors in AECOPD patients who are hospitalised. There has been no development of robust clinical tools to aid in management decisions. There are well-established scores, such as the BODE[18] score, that can be used to assess mortality risk in patients with stable COPD. Patients requiring hospitalisation have not been studied

using prognostic tools developed for stable state disease. There has been little prognostic research in exacerbations that necessitate hospitalisation. There appears to be a significant difference in prognostic factors between acute exacerbation and stable COPD. There is a need to identify simple, easy-to-use, and easy-to-obtain predictors of in-hospital mortality. These predictors should also assist in determining the need for post-hospital care.

J Steer et al [19] developed the DECAF score, a simple prognostication tool in acute COPD exacerbation that will aid in deciding location of care, early stepping up of care, and anticipating the need for ventilatory support. It assists the physician in informing relatives and patients about the prognosis and risks associated with exacerbations. As a result, it will aid in directing the most efficient use of resources, reducing mortality and morbidity.

# AIM & OBJECTIVES

# AIM OF THE STUDY

To assess the prognostic effect of the DECAF score in predicting in hospital mortality in acute exacerbation of chronic obstructive pulmonary disease.

# REVIEW OF LITERATURE

# REVIEW OF LITERATURE

**Severity of Dyspnea and Mortality:**

In COPD, the extent of breathlessness is assessed by the Medical Research Council Dyspnea (MRCD) Score. The MRCD score for dyspnea appears to be a better predictor of mortality[20] when Compared to Forced Expiratory Volume in one second (FEV1) . studies regarding MRCD score predictive value in exacerbations that need hospitalization are not adequate. However, few reports shows that higher MRCD scores are predictors of both short and long term mortality. It has been shown that greater 'functional dependence' independently predicts hospital readmission and patients performance status. patient's inability to manage self-care is An important predictor of three month mortality following hospitalization. Long term mortality is higher if there is higher level of functional dependence in those patients who survive up to discharge.

As a result severe disability strongly influences on individual patient care decisions. So the predictive ability of the traditional dyspnea scoring scale improved when dyspnea assessment is combined with a measure of functional dependence.

J Steer et al[21], in a retrospective study defined an enhanced version of MRCD scale – the extended MRC Dyspnea score (eMRCD) , which was

better in categorizing patients at risk of repeated hospitalization when compared to the MRCD scale. They studied a large population of patients with acute exacerbation of COPD. The aptitude of both MRCD and eMRCD scores in predicting in-hospital mortality and early admission were compared.

**Table – 2. Extended Medical Research Council Dyspnea Score[19]**

| **Limitation due to breathlessness** | **MRCD** | **eMRCD** |
|---|---|---|
| Breathless only with strenuous exercise | 1 | |
| Breathless when hurrying on level / walking up a slight hill | 2 | |
| Walks slower than peers, or stops when walking on the flat at own pace | 3 | |
| Stops after walking 100m, or for a few minutes on the level | 4 | |
| Too breathless to leave the house | 5 | - |
| **& independent in washing and / or dressing** | **-** | **5a** |
| **& dependent in washing and dressing** | **-** | **5b** |

In their study with 920 participants, the in hospital death was 96 . The mortality rate for eMRCD 5a and 5b was 17.3% (30/96) and 33.1% (47/96) respectively with (p=0.0012). In the non pneumonic AECOPD, individuals with eMRCD 5b had significantly higher in hospital death rates than those with eMRCD 5a (p=0.048). There was similar association in patients with pneumonic AECOPD .it was not, however, statistically

significant (p=0.069). The eMRCD 5b group had higher 28 day readmission rates compared to the eMRCD 5a group (p=0.044). The ability of MRCD, eMRCD, and CURB-65 to predict short-term mortality was compared using areas under receiving operator characteristics curve. When compared to MRCD (p=0.0012) and CURB-65, eMRCD predicted in-hospital mortality better in the study population (0.019). eMRCD had superior discrimination in the non pneumonic AECOPD group than both MRCD (p=0.057) and CURB-65 (p=0.053), albeit the difference was not statistically significant. eMRCD outperformed CURB-65 when it came to pneumonic exacerbation. The predictive strength of the grading system is increased when the traditional MRCD scale is extended to include a person's functional dependence (eMRCD), according to this study. eMRCD is more accurate in predicting both in-hospital mortality and the requirement for readmission after discharge. This grading method is used to identify a subgroup of patients who are at a greater risk of mortality in the hospital (33.1 % with eMRCD 5b). It's simple to determine the degree of dyspnea in people who need to be admitted to the hospital for AECOPD. The severity of dyspnea is a strong predictor of outcome and gives valuable information that can help with management decisions.

**Eosinopenia and Mortality:**

There are many studies that have examined at eosinophil levels, particularly eosinopenia, as an indicator for infection, inflammation, and bacteremia. These studies have a limited sample size and involve people from a variety of backgrounds. This could have resulted in inconsistent results, impose a significant limitation to their interpretation.

Gil et al[22] investigated the role of eosinophil count in patients with infection in 2003. They found that having a high total leukocyte count (more than 10,000/mm3) and a low eosinophil count (less than 40/mm3) was significantly associated with the occurrence of bacterial infections. Abidi et al[23] afterward studied eosinopenia as a marker of sepsis. They concluded that a low eosinophil count could be used as an indicator of infection in routine medical practice.

In a cohort[24] of 2,311 patients with bacteremia, investigators found that, eosinopenia ($<50/mm^3$) was associated with a 4.77-fold increase in mortality when compared with normal eosinophil count. When confounding factors were eliminated, it was discovered that a persistently low eosinophil count was an independent but robust predictor of mortality.

Abidi et al[23]. evaluated the role of eosinopenia in predicting in-hospital mortality. The study was done in patients admitted in Intensive Care unit , many of them had infection. Eosinopenia was strongly

associated with mortality at 28 days. The hazard ratio in the multivariate analysis was 1.8.

Holland et al[25] studied 66 patients hospitalized withexacerbation of chronic obstructive pulmonary disease. Admission eosinophil count was obtained in all 66 patients. The mortality rate in patients with eosinopenia at baseline and with normal baseline eosinophil count was was 17.4% and 2.4% respectively. When compared to patients with normal eosinophil values, mortality in eosinopenia was significantly higher at p=0.049. Likewise group with eosinopenia had significantly longer duration of hospital stay (8 vs 5 days p=0.005).

According to these authors, low eosinophil count could be utilised as an independent marker of disease severity and prognosis, in addition to commonly used indicators. In animal models, it has been demonstrated that leukocytes are diverted towards the development of polymorphonuclear cells in the presence of acute infection or inflammation, resulting in a low eosinophil count. Eosinopenia occurs as a result of the body's response to an acute infection. This isn't dependent on glucocorticoids from the adrenal glands. Eosinopenia is an independent and useful marker of sepsis in patients who require intensive care. In the setting of an acute COPD exacerbation, a low eosinophil count may be indicative of the severity of the inflammatory response.

**Acute exacerbation of COPD with coexisting pneumonia:**

When a known case of COPD develops typical symptoms of an exacerbation episode due to community acquired pneumonia, the clinician is always unsure whether such an episode should be labelled as AECOPD. Radiographic consolidation is frequently associated with COPD exacerbations. There have always been questions about whether patients with AECOPD and coexisting consolidation should be diagnosed as having AECOPD, with varying practices around the world. Patients with concurrent pneumonia, on the other hand, were not excluded from major national studies of COPD exacerbation and non-invasive ventilation conducted in the United Kingdom. Concurrent pneumonic consolidation occurred in 16 percent and 34.2 percent of the cases in these studies, respectively26,27. Furthermore, conventional chest radiography may be ineffective at detecting parenchymal consolidation. It has been observed that in a significant number of patients who had initially negative radiography, a more detailed evaluation revealed the presence of consolidation[28].

When compared to subjects with non-pneumonic exacerbations, patients with pneumonic exacerbations of COPD had the same socio-demographic profile and severity of underlying disease. The former group, on the other hand, had more severe clinical and physiological

derangement[29]. The severity of airway obstruction and pathogens involved are similar in pneumonic and non-pneumonic exacerbations.

Exacerbations of pneumonia should not be treated as pneumonia alone, but rather as a complication of the AECOPD. As a result, these patients will require continuous low flow oxygen, parenteral steroids, and nebulized bronchodilators. Noninvasive ventilation should be administered if hypercapnic respiratory failure occurs. This suggests that coexisting pneumonia aids in the identification of patients with severe acute illness. It does not signify a different disease process[30].

**Pneumonic and Non pneumonic acute exacerbations of COPD**

David Lieberman et al[31] carried out a study in a tertiary medical care centre in southern Israel. There were 23 hospitalizations for pneumonic acute exacerbation of COPD (PNAE) and 217 hospitalizations for non pneumonic exacerbation of COPD (NPAE). Patients with community-acquired pneumonia were also included in the AECOPD diagnosis for the reasons listed below.

1. The clinical features of the patients were consistent with the accepted criteria for AECOPD. Only radiographic examination can reveal the presence of pneumonia to the clinician. It is not rational to eliminate the diagnosis of acute COPD exacerbation in such patients. These

patients are more likely to have a pneumonic exacerbation. This emphasizes the significance of the combination of events.

2. Most of the patients with AECOPD are managed on an out- patient setup. In such a scenario, chest radiographs are not routinely obtained. Hence elimination of patients with community acquired pneumonia is not practically possible. They showed that compared to NPAE, patients with pneumonia had higher rates of hypoxemia, higher rates of hospitalisation(P=0.004), higher rates of sudden onset (P=0.005). Patients with PNAE also had higher rates of ICU admission (P=0.006), intubation(p=0.01), in-hospital death (P=0.007) and longer duration of stay in hospital(P=0.001). In PNAE, Viral and pneumococcal etiologies are more common.

J Steer et al[21] found that co-existing pneumonia is common in COPD individuals suffering an acute exacerbation. It is also associated with a high mortality rate. When AECOPD and pneumonia occur simultaneously, the mortality rate is higher than when pneumonia occurs alone. The study included 920 patients in total. Patients with pneumonic AECOPD had a longer hospital stay (7 days) than patients with non-pneumonic AECOPD (6 days, $p<0.001$).

Non-pneumonic AECOPD (36/621) had a 5.8 % in-hospital death rate, whereas PNAE (60/299) had a 20.1 % in-hospital death rate. The 28-day readmission rate for pneumonic AECOPD was 19.5 % , while the rate for non-pneumonic AECOPD was 18 % . In terms of readmission rates, there was no statistical difference (P=0.62).

CURB-65 is frequently used to classify patients with pAECOPD. In these patients, it is frequently used to guide treatment. However, the data show that CURB-65 is not an accurate predictor of mortality risk in this population. When statistically analysed, CURB-65 performs moderately, with an AUROC of 0.661. eMRCD performed better in predicting short-term (p=0.017) and post-hospital follow-up moratlity (p=0.040) moratlity (p=0.040). Recent research has found that CURB-65 is a good predictor of in-hospital mortality in patients with non-pneumonic AECOPD. According to the findings of the above study, eMRCD outperformed CURB-65 in all patients.

**Acidemia and mortality :**

In patients with AECOPD, the prevalence of hypercapnic respiratory failure ranges from 16 to 35 percent, with overall mortality ranging from 35 to 43 percent 32. On hospital admission, hypoxia and hypercapnia were both common conditions. In AECOPD1, respiratory acidosis (arterial pH 7.35 and/or PCO2 6.0kPa, 45 mm hg) is an indication

for ventilator support. Noninvasive (via nasal or facial mask) or invasive ventilation can be used (by oro-tracheal tube or tracheostomy). Acute respiratory acidosis is reduced by mechanical ventilation. It reduces tachypnea, work of breathing, the severity of dyspnea, and the length of hospital stay.

Karin H. Groenewegen et al[33] studied a total of 171 AECOPD patients. The in-hospital death rate was 8%. At one year of follow-up, the death rate was 23%. In-hospital mortality was comparable at 6% for patients requiring intensive care management. However, the 1-year follow-up mortality rate in patients admitted to the ICU for respiratory failure was significantly higher, at 35%. To identify independent predictors of survival, the multivariate Cox proportional hazards model was used. Age, gender, FEV1, PO2, PCO2, body mass index, long-term use of oral corticosteroids, comorbidity index, and hospital readmissions were all variables in the regression model. The use of oral glucocorticosteroids for maintenance (relative risk [RR], 5.07; 95 percent CI, 2.03 to 12.64), PCO2 (RR, 1.17; 95 percent CI, 1.01 to 1.38), and age (RR, 1.07; 95 percent CI, 1.01 to 1.12) were all independently related to mortality. When the characteristics of ICU patients and non-ICU patients were compared, they discovered that ICU patients had higher PCO2 and lower pH values.

Chronic alveolar hypoventilation causes hypercapnia. As a result, hypercapnia reflects the severity of the respiratory disease. As a consequence, patients with elevated pCO2 have a poor prognosis when compared to patients without elevated pCO2. Patients with chronic hypercapnia made up a considerable proportion of the study population.

In one study[34], Patients with severe COPD who had at least one hospitalization for hypercapnic respiratory failure were compared to patients who had been treated for unresectable non-small cell lung cancer. According to the findings of this study, COPD patients had significantly less ability to perform daily activities, as well as lower physical, social, and emotional functioning than patients with non-small cell lung cancer. This study confirms that COPD patients are not receiving the necessary palliative care.

J Steer et al[19] in their study found that arterial pH was statistically lower in patients who died in the hospital versus those who survived until discharge (pH 7.3 odds ratio (95 percent CI) 2.68(1.41 – 5.09) p=0.003).

**Atrial fibrillation and COPD:**

COPD is accompanying with increased risk of cardiac arrhythmias. The major causes of arrhythmias in COPD are hypoxemia[35], acidosis[36], corpulmonale[37], coexisting ischemic heartdisease[38]. The severity of the

disease determines the type and risk of arrhythmias in COPD patients. The most common arrhythmias that occur during exacerbations are supraventricular tachycardias. Even in patients with stable COPD, the incidence of cardiac arrhythmias is high.

Atrial fibrillation (AF) is commonly seen in patients hospitalised for exacerbations. By far the most common arrhythmia in the elderly is atrial fibrillation (AF).

P.Buch et al[39] analyzed data from 13,430 men and women. The participants were taken from the Copenhagen City Heart Study. There had been no previous myocardial infarctions in any of the subjects. After 5 years, a re-examination was performed to look for new arrhythmias. Multivariate analyses with cardiopulmonary risk factor adjustment were used. 290 cases of AF were diagnosed at the time of hospitalisation (2.20 percent ). At the 5-year follow-up, there were 62 new cases of AF (0.58 percent ). After adjusting for gender, age, smoking, blood pressure, diabetes, and body mass index, the risk of new AF at re-examination was 1.8 times higher for FEV1 between 60–80 percent of predicted versus FEV1 > 80 percent. When additional adjustments for education, diuretic treatment, and activity-related chest pain were made, the risk of hospitalisation for AF was 1.3-times higher for FEV1 between 60–80 percent and 1.8-times higher for FEV1<60% compared with FEV1 ≥80%.

The authors came to the conclusion that decreased lung function is an independent predictor of incident atrial fibrillation.

The mechanism underlying the link between reduced lung function and AF is unclear. Recent findings40 show that ectopic beats that cause AF frequently originate in the walls of the pulmonary veins. It's possible that changes in gas composition or pulmonary hypertension triggered this. Because the relationship was also found in subjects with mild to moderately reduced FEV1, hypoxia and cor pulmonale could only account for a portion of this effect. Reduced lung function has been shown to be an independent predictor of IHD and stroke, and it is possible that the biological mechanism for the development of AF is linked to atherosclerosis via a common pathway of vascular and airway disease development, such as fetal or early childhood exposure[41].

**Factors Predicting outcome in acute exacerbation of COPD:**

Roche et al[30] studied COPD exacerbation patients visiting emergency department.

They encountered simple, easily accessible, but strong predictors of in-hospital mortality and the need for post-hospital care. They found three simple clinical criteria that were significant predictors of in-hospital mortality.

In 2001, The American College of Physicians and The American Society of Internal Medicine shown an evidence based process[42].Only 11 studies had been shown to identify predictors of in-hospital mortality

None of the studies offered a simple prediction tool that helps in management decision making.

John Steer et al[19], established a strong clinical prediction tool. He studied at a large group of COPD patients who were admitted to the hospital due to exacerbations. A total of 920 patients were enlisted from various geographical locations. Socio-demographic and clinical information was gathered. They wanted to create a simple but effective prognostic tool. The five major categorical variables were selected, and relative weights were assigned based on the regression coefficient. As a result, the DECAF score was created, which stands for Dyspnea, Eosinopenia, Consolidation, Acidemia, and Atrial Fibrillation. The DECAF score outperformed other predictive instruments in AECOPD, such as the Acute Physiology And Chronic Health Evaluation II prognostic index, the COPD and Asthma Physiology Score, and the BAP 65 score, in predicting in-hospital death. For predicting in-hospital mortality, the area under the DECAF score ROC curve was 0.86 (95 percent CI– 0.82 to 0.89).

# MATERIALS & METHODS

# METHODOLOGY

## STUDY POPULATION:

50 Patients getting admitted to Government Rajaji Hospital, Madurai (GRH , Madurai) with symptoms of acute exacerbation of Chronic Obstructive Pulmonary Disease (COPD) were selected..

## INCLUSION CRITERIA:

- patients aged 35 years or older
- a primary clinical diagnosis of AECOPD, spirometry consistent with airflow obstruction (FEV1/forced vital capacity <0.70)
- a smoking history of ≥10 cigarette packs per year
- admission from the primary residence

## EXCLUSION CRITERIA:

- Patients in whom the primary reason for admission was other than acute exacerbation of COPD were excluded from the study. Hence patients with the following diseases were excluded from our study

➢ Bronchial Asthma-acute exacerbation

➢ Bronchiectasis-infective exacerbation

➢ Interstitial Lung Diseases-exacerbation

- Lung cancer
- Pneumothorax
- Congestive cardiac failure
- Acute on chronic decompensated liver disease
- Acute on chronic decompensated renal disease
- Psychiatric illness

- survival-limiting comorbidity (e.g., metastatic malignancy),
- Patients with domiciliary ventilation,
- Patients who are treated with oral corticosteroids in the week prior to this admission
- Previous inclusion in the study.

**STUDY CENTRES:**

The study was conducted at tertiary care centre Government Rajaji Hospital, Madurai (GRH , Madurai).

**STUDY DESIGN:**

- The study was a prospective study.
- No specific intervention was carried out

- No specific method of randomisation was used. Consecutive patients admitted with the diagnosis of acute exacerbation of COPD during the study period were included in the study.

- No controls were used in the study

**STUDY PERIOD:**

6 months January 2021 – June 2021

**DATA COLLECTION:**

Samples will be collected from 50 COPD patients with acute exacerbation admitted in Govt Rajaji Hospital Madurai during the study period of 6 months

All patients included in the study are subjected on admission to the followings

1. Thorough medical history including Socio-demographic and comorbidity details

2. Full clinical examination (general and local examination).

3. Routine laboratory investigations:

- Complete blood count

- Liver function tests

- Renal function tests
- Serum electrolytes
- Arterial blood gas analysis
- Plain Chest X- ray
- Electrocardiogram

## METHODOLOGY

50 patients admitted with primary diagnosis of AECOPD were included. Patients were scored according to the DECAF scoring system – Dyspnea, Eosinopenia, Consolidation, Acidemia and atrial Fibrillation. The patients were followed during the entire hospital stay. The clinical outcome was recorded. The role of DECAF score in predicting in hospital mortality was analyzed.

**Table – 3. DECAF Score**

| Variable | Score |
|---|---|
| Dyspnea<br>eMRCD 5a<br>eMRCD 5b | <br>1<br>2 |
| Eosinopenia (<50cells/mm3) | 1 |
| Consolidation | 1 |
| Acidemia (pH <7.3) | 1 |
| Atrial fibrillation | 1 |
| Total score | 6 |

DECAF: Dyspnea according to eMRCD, extended MRC dyspnea, Eosinopenia, Consolidation, Acidemia and atrial Fibrillation;.

The clinical outcome was categorised as

a. Survivors
b. Non survivors

**ANTICIPATED OUTCOME:**

- The DECAF Score show promise for risk stratification of patients hospitalized with AECOPD
- The death rates for each grade of the DECAF Score suggest the following risk categories:

- **DECAF 0–1 ('low risk')**
- **DECAF 2 ('moderate risk')**
- **DECAF 3–6 ('high risk')**

DECAF scores of zero to one are strong predictors for survival, and DECAF scores of four to six are strong predictors of mortality

**COLLABORATING DEPARTMENTS:**

Department of Thoracic medicine,GRH

Department of Radiology,GRH

Department of Biochemistry,GRH

**ETHICAL CLEARANCE:** Obtained

**CONSENT:** Informed written consent from relatives

**ANALYSIS:** Statistical analysis will be performed using appropriate tests required according to data collected.

**CONFLICT OF INTEREST:** nil

**FINANCIAL SUPPORT:** Self

**PARTICIPANTS:** 50 COPD patients with acute exacerbation admitted in Govt Rajaji Hospital Madurai.

**PROFORMA**

Name:

Age / Sex:

In patient No:

Occupation:

**Presenting complaints:**

H/O Difficulty in breathing

H/O cough with expectoration

H/O wheezing

H/O chest pain

H/O fever

H/O swelling of both legs

H/O loss of appetite

H/O loss of weight

**Past History:**

H/o COPD, DM, HT, CKD, CAD, CCF, CLD, TUBERCULOSIS, MALIGNANCY

**Personal History:**

H/o smoking,

H/o alcoholism

H/o sleep, diet

**Family History:**

H/o similar illness in family members

**General Examination:**

Consciousness

Pallor

Jaundice

Clubbing

Lymphadenopathy

Cyanosis

Pedal edema

**Examination of Oral cavity:**

**Vitals:**

PR

BP

RR

SpO2

**Systemic examination:**

CVS:

RS:

ABDOMEN:

CNS:

**Laboratory investigations:**

- Complete blood count
- Liver function tests
- Renal function tests
- Serum electrolytes
- Arterial blood gas analysis
- Plain Chest X- ray
- Electrocardiogram

**DECAF Score:**

**STATISTICAL ANALYSIS:**

Statistical analysis of the collected data was done using the SPSS softwares. Continuous data with normal distribution was specified as mean with standard deviation. Categorical data were specified as frequency with percentage. For to compare frequency between groups Fisher's exact test was used. Significance of correlation inbetween variables was evaluated using p value. if the p value was less than 0.05 the correlation was considered to be statistically significant.

# RESULTS & OBSERVATIONS

# RESULTS

**Age distribution:**

A total of 50 patients were included in our study who are fulfilled our selection methods, inclusion and exclusion criteria. In our study age group of our patients ranged from 38 to 81. The mean age of the study population was 56.2 with a standard Deviation of 9.45.

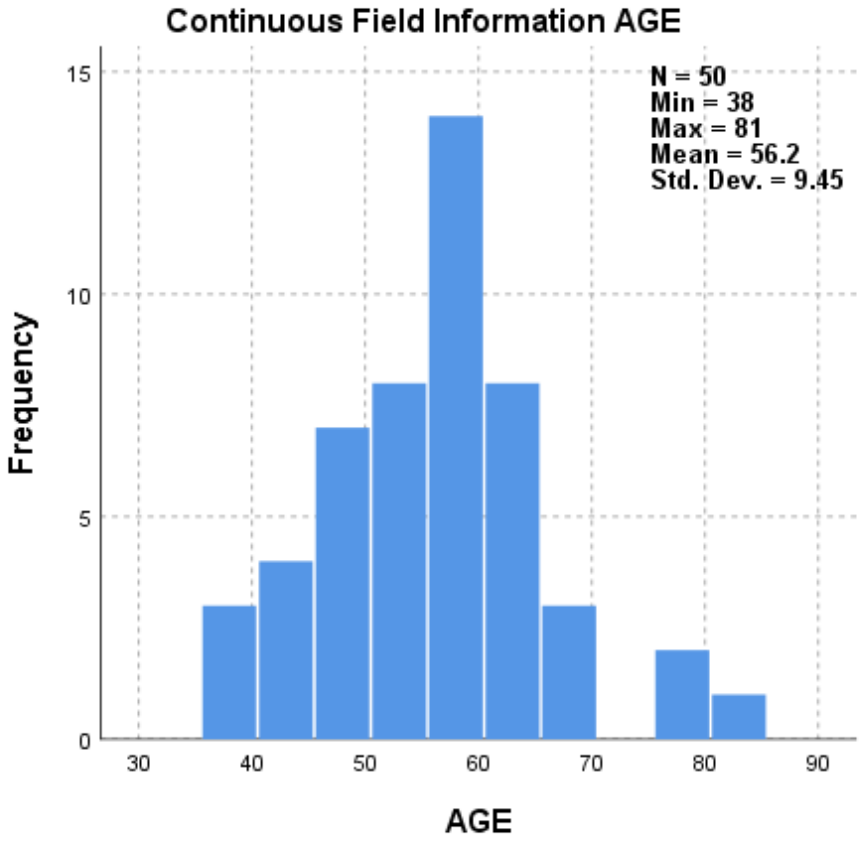
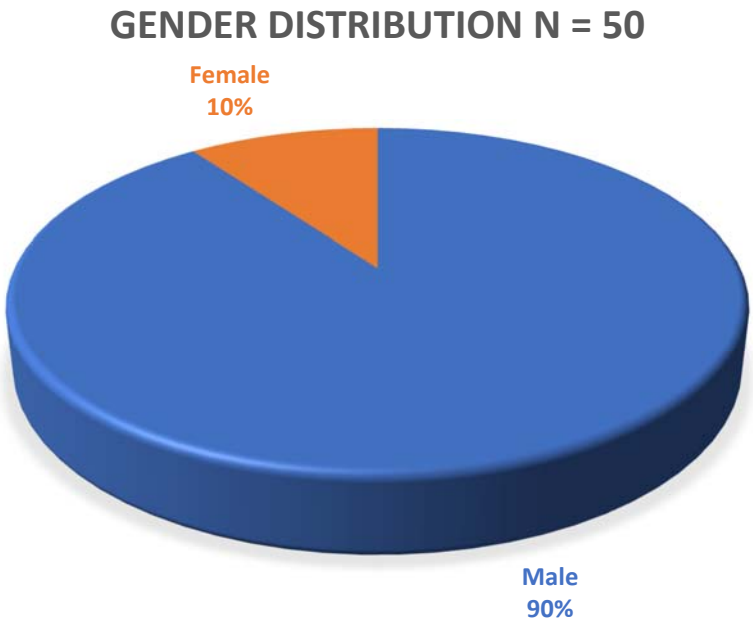



**Fig. 4: Age Distribution**

**Gender distribution:**

Out of the 50 patients in the study, 45 are male and 5 are female. Thus males accounted for 90% of our study population while females accounted for 10%.

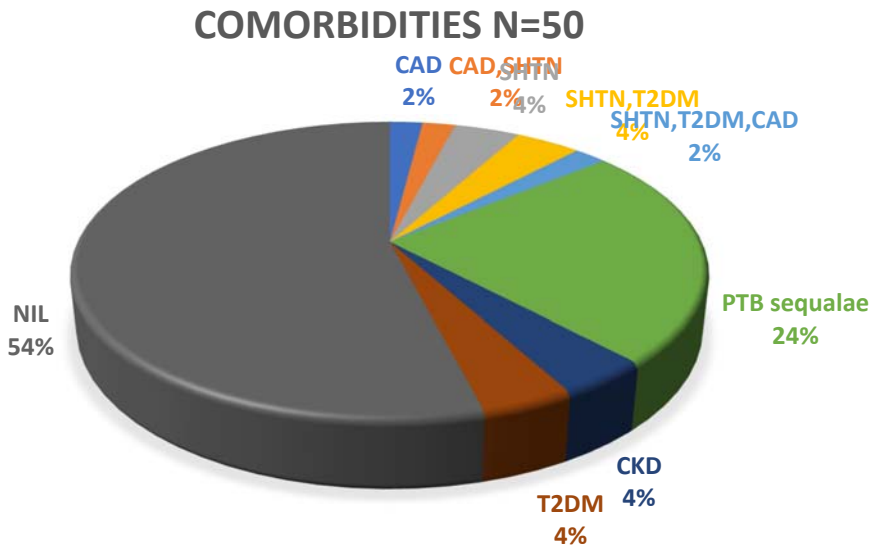



| Gender | Male | Female |
|---|---|---|
| No | 45 | 5 |

**Fig. 5: Gender Distribution**

**Comorbidity:**

In the study population, 27 did not have any comorbid illness. The most common comorbidity among the study population is Pulmonary Tuberculosis Sequalae.

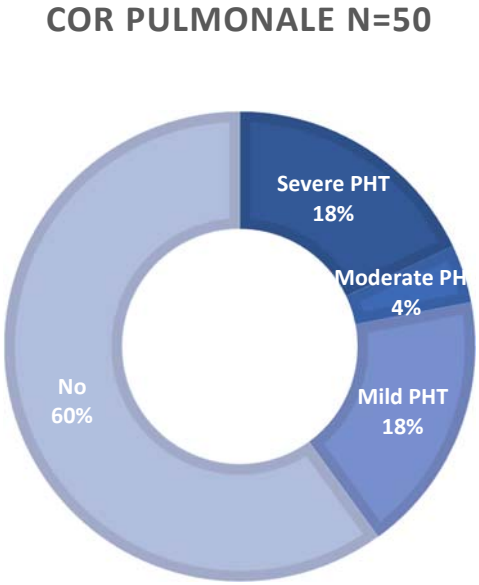



| Comorbidity | No |
|---|---|
| Coronary artery disease | 1 |
| Coronary artery disease and Systemic hypertension | 1 |
| Systemic hypertension | 2 |
| Systemic hypertension and Diabetes mellitus | 2 |
| Diabetes mellitus and Systemic hypertension and coronary artery disease | 1 |
| PTB sequalae | 12 |
| Chronic kidney disease | 2 |
| Diabetes mellitus | 2 |
| Nil | 27 |
| Total | 50 |

**Fig. 6 : Comorbidities**

**Presence of cor pulmonale:**

Out of 50 patients included in the study 20 (40%) patients had corpulmonale as evidenced on echo. Out of them 9 (18%) patients had mild pulmonary hypertension (PHT), 2 (4%) patients had moderate PHT, 9 (18%) patients had severe PHT.

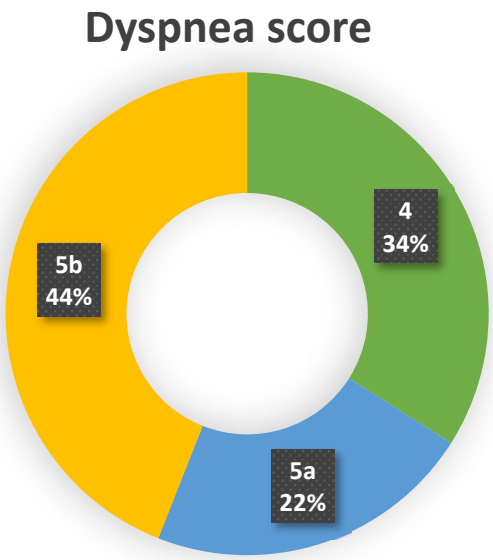



| Cor pulmonale | No |
|---|---|
| Severe PHT | 9 |
| Moderate PHT | 2 |
| Mild PHT | 9 |
| No | 30 |
| Total | 50 |

**Fig. 7 : Cor pulmonale**

**Dyspnea grading:**

The patients in the study were graded according to the extended Medical Research Council score. Accordingly, 17 patients had eMRC grade 4, 11 patients had eMRC grade 5a and 22 patients had a score of 5b. In terms of percentage, the distribution of patients in grades 4, 5a and 5b was 34, 22 and 44 respectively.

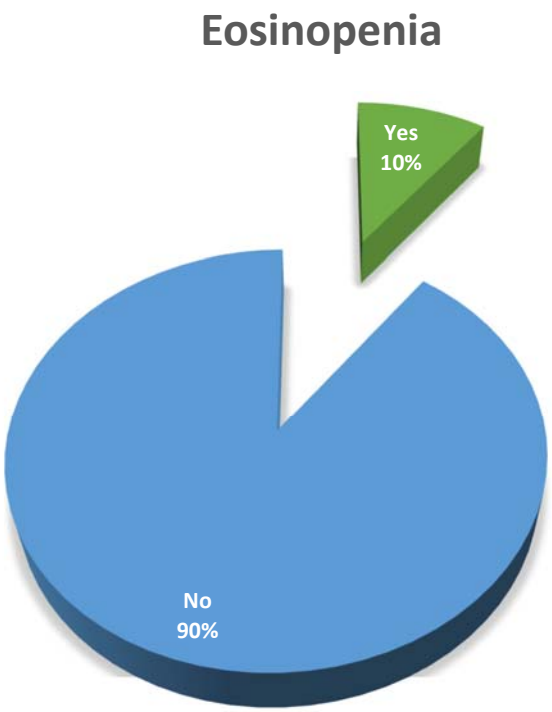



| Dyspnea grade eMRC | No |
|---|---|
| 4 | 17 |
| 5a | 11 |
| 5b | 22 |

**Fig. 8 : Dyspnea Score**

**Presence of Eosinopenia:**

Eosinopenia was defined as an absolute eosinophil count of less than 50/mm3. 5 out of 50 patients had eosinopenia. Hence 10% of the study population had low eosinophil count.

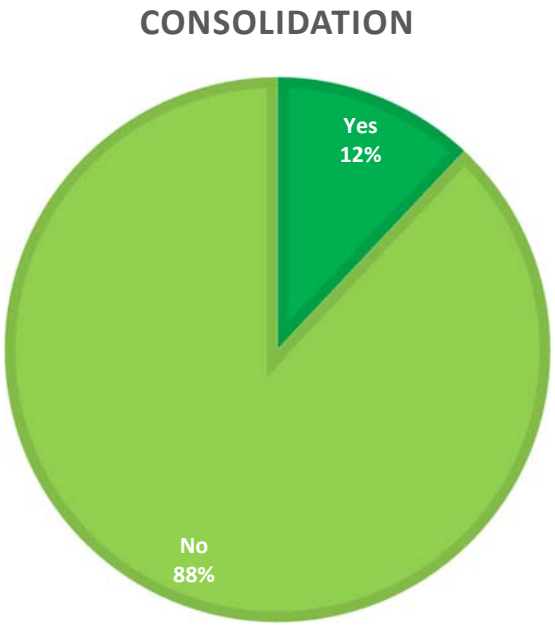



| Eosinopenia | No |
|---|---|
| Yes | 5 |
| No | 45 |

**Fig. 9 : Eosinopenia**

**Presence of Consolidation:**

Assessment of chest radiographs of patients at admission to confirm the presence of consolidation was done. Accordingly 6 (12%) patients had consolidation on chest radiograph.

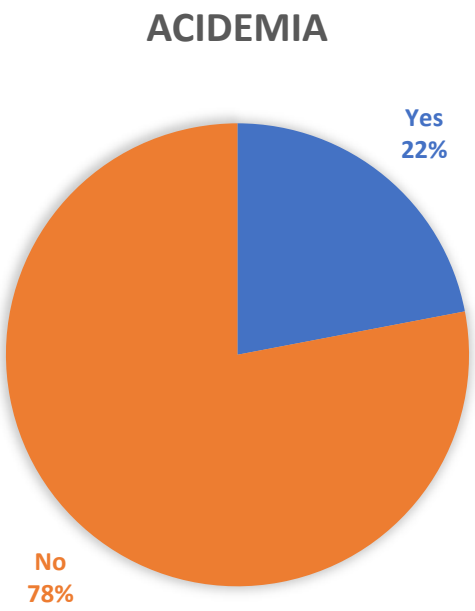



| Consolidation | No |
|---|---|
| Yes | 6 |
| No | 44 |

**Fig. 10 : Consolidation**

**Presence of Acidemia:**

Acidemia is defined as the presence of arterial blood gas pH<7.30. 11 patients (22%) had acidemia.

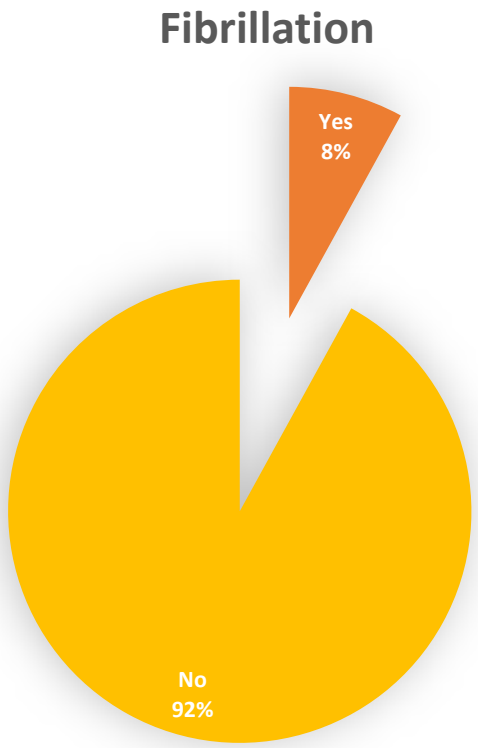



| Acidemia | No |
|---|---|
| Yes | 11 |
| No | 39 |

**Fig. 11 : Acidemia**

**Presence of Fibrillation:**

Presence of atrial fibrillation was confirmed with the presence of admission electrocardiogram. Accordingly 4 (8%) patients had atrial fibrillation, while the remaining 46 did not have fibrillation.




| Atrial Fibrillation | No |
|---|---|
| Yes | 4 |
| No | 46 |

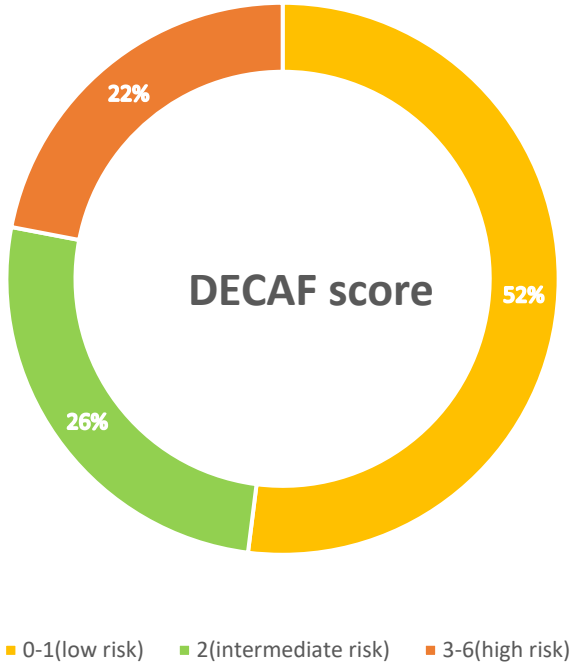
**Fig. 12 : Atrial Fibrillation**

**THE DECAF SCORE:**

Each patient was scored using DECAF score – where dyspnea eMRC grade 5a gets 1 point, dyspnea eMRC grade 5b gets 2 points, others parameters, namely Eosinopenia, Consolidation, Acidemia, atrial Fibrillation get 1 point each.

We divided the population into three groups namely low risk, intermediate risk and high risk with the groups getting DECAF score of 0-1, 2 and 3-6 respectively.

26 patients had a DECAF score between 0-1, 13patients had a DECAF score of 2 and 11 patients had a DECAF score between 3-6. In terms of percentage this is 52%, 26% and 22% respectively.

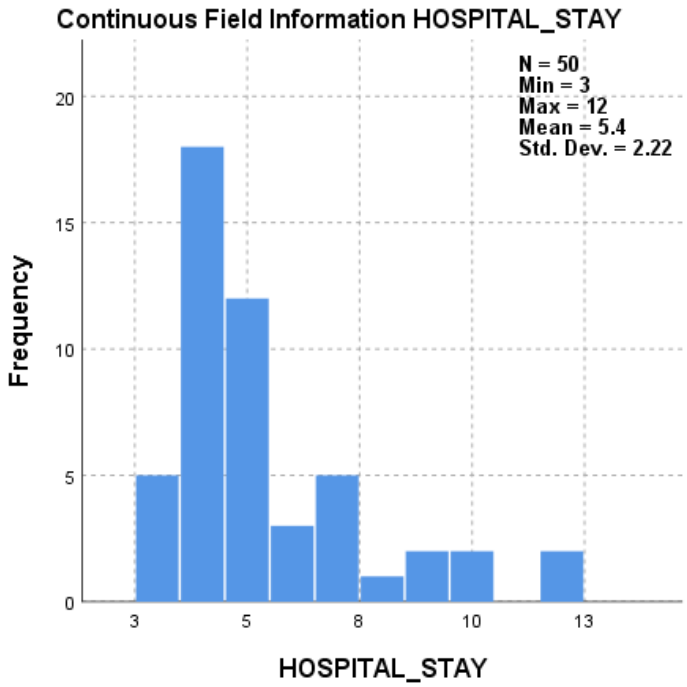



| DECAF score | No |
|---|---|
| 0-1(low risk) | 26 |
| 2(intermediate risk) | 13 |
| 3-6(high risk) | 11 |

**Fig. 13 : DECAF score**

**Duration of hospital stay :**

The average duration of hospital stay for the study group was 5 days. The shortest length of hospital stay was 3 days and the longest length of hospital stay was 12 days.

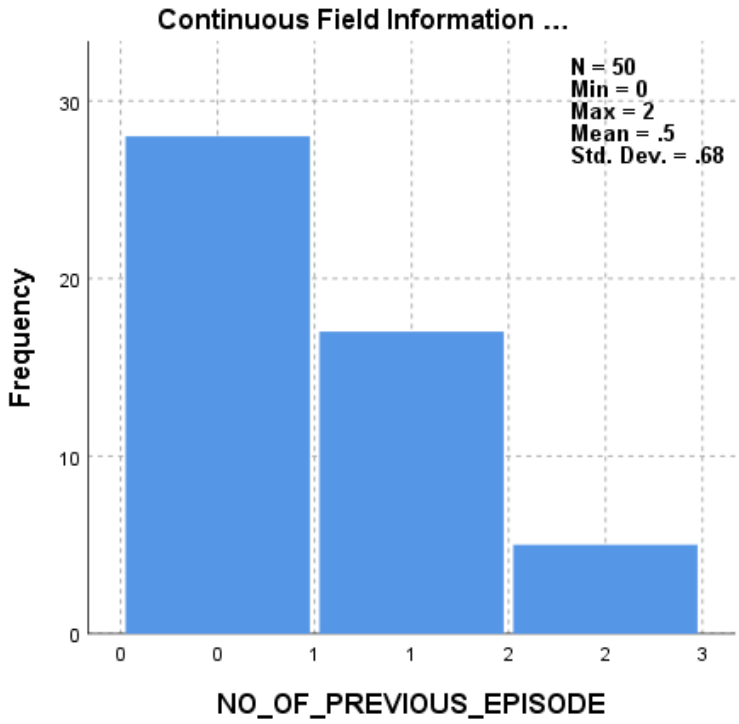



| Hospital stay | days |
|---|---|
| Avg duration | 5 |
| Shortest | 3 |
| longest | 12 |

**Fig. 14 : Hospital Stay**

**Number of previous episodes:**

Regarding number of pervious episodes of acute exacerbation mean episode was 0.54 and standard deviation was 0.68.minimum episode was 0 and maximum episode was 2.

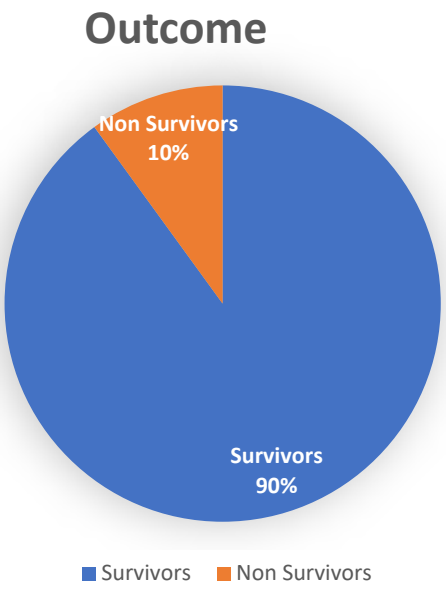



| Number of previous episodes | Episodes |
|---|---|
| Avg | 0.5 |
| min | 0 |
| max | 2 |

**Fig. 15 : Number of previous episodes**

**Outcome:**

The mortality rate for the study population was 5 out of 50 (10%). 45 patients were improved, clinically defined as subjective sense of improvement and objective improvement in dyspnea scoring they were categorised under survivors , 5 patients died in the hospital.




| Outcome | No |
|---|---|
| Survivors | 45 |
| Non Survivors | 5 |

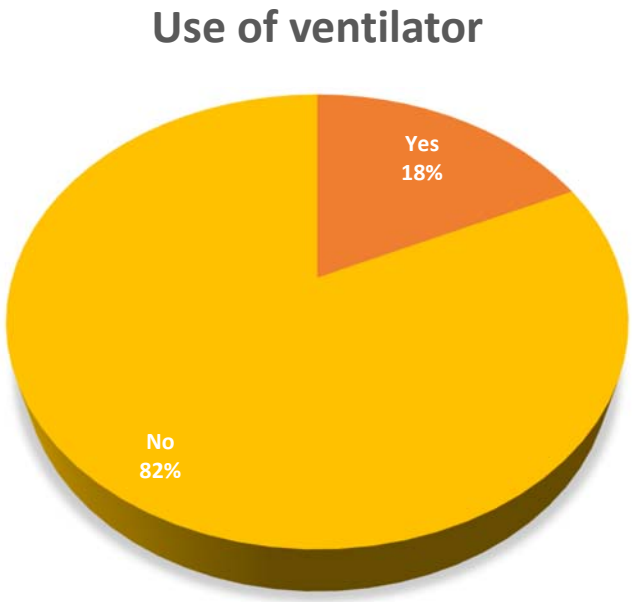
**Fig. 16 : Outcome**

**Use of ventilator:**

Out of the 50 patients 9(18%) were put on ventilator,

5 were puton Non-invasive ventilation and

4 were put on Invasive ventilation.




| Use of ventilator | No |
|---|---|
| Yes | 9 |
| No | 41 |

**Fig. 17 : Use of Ventilator**

**Age and outcome:**

When age was taken as scattered variables, older patients showed higher mortality. However, there is no statistically significant association between the age and outcome, P≥0.05(P=0.059).

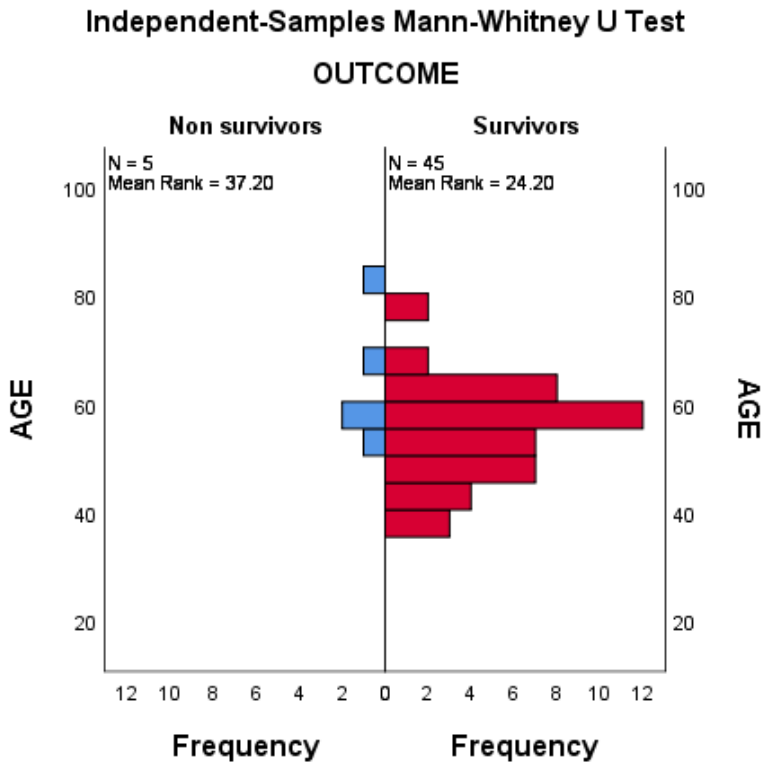



**Table – 4. Age Vs Outcome**

**Hypothesis Test Summary**

| Null Hypothesis | Test | Significance |
|---|---|---|
| The distribution of AGE is the same across categories of OUTCOME. | Independent-Samples Mann-Whitney U Test | .059[a] |

**Gender and outcome:**

The mortality among female patients in the study is 1 out of 5 (20%). The moratlity among the male patients is 4 out of 45 (8.9%).There is no significant association between gender and outcome. This could be attributed to the very low number of female participants in the study.

**Table – 5. Gender Vs Outcome**

| Sex | Non survivors | | survivors | | P value |
|---|---|---|---|---|---|
| Female | 1 | 20.0% | 4 | 8.9% | 0.423 |
| Male | 4 | 80.0% | 41 | 91.1% | |

**Chi square test; not significant**

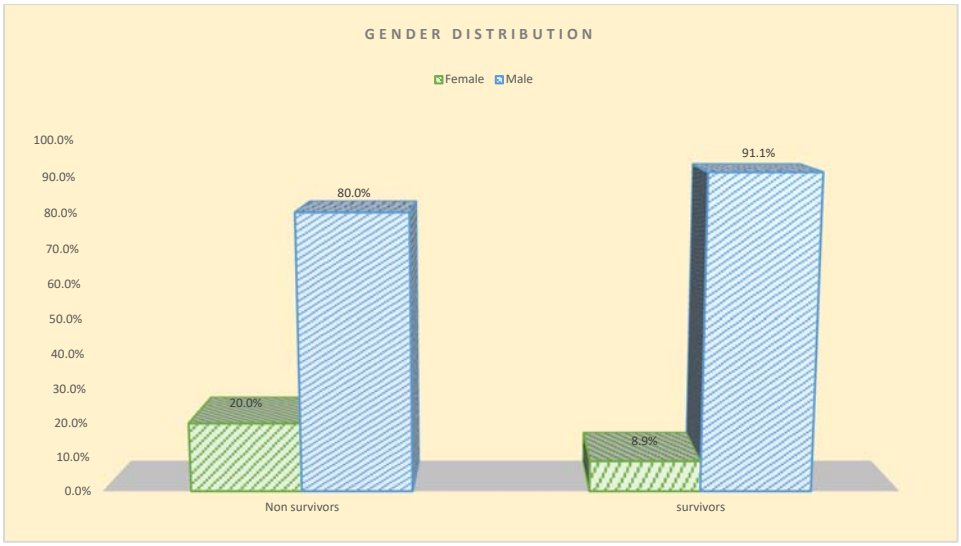



**Fig. 18 Gender vs Outcome**

There is no association between gender and outcome.

**Impact of cor pulmonale on outcome:**

The mortality among patients with cor pulmonale is 25% ( 5/20). The mortality among patients without cor pulmonale is 0% (0/30). Among the study population, patients having cor pulmonale and pulmonary hypertension had higher mortality. Patients who did not have PHT were survived. This association between cor pulmonale and outcome is statistically significant at p=0.001.

**Table – 6. Cor Pulmonale Vs Outcome**

| Cor pulmonale | Non survivors | | survivors | | P value |
|---|---|---|---|---|---|
| MILD PHTN | 0 | | 9 | 20.0% | |
| MODERATE PHTN | 1 | 20.0% | 1 | 2.2% | 0.001* |
| SEVERE PHTN | 4 | 80.0% | 5 | 11.1% | |
| NO | 0 | | 30 | 66.7% | |

**Chi square test; shows (*p<0.05)**

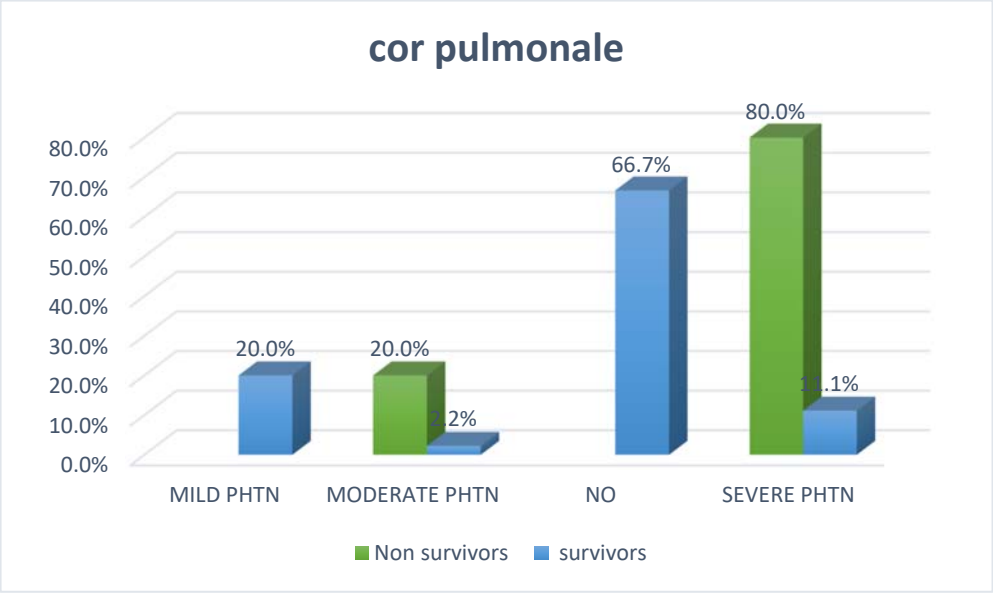



**Fig. 19 : Cor pulmonale and outcome**

**Grade of dyspnea and outcome:**

In patients getting admitted with AECOPD, as the eMRC dysnea grade increases, the mortality increases. All the patients in the score of 4 and 5a were survived. Mortality is predominantly seen in eMRC 5b group. The mortality rate among eMRC 5b is 5 out of 22(22.7%). The relation is statistically significant at p=0.029.

**Table – 7. DYSPNEA Vs Outcome**

| DYSPNEA * OUTCOME crosstab | | | OUTCOME | | Total |
|---|---|---|---|---|---|
| | | | Non survivors | Survivors | |
| DYSPNEA | 4 | Count | 0 | 17 | 17 |
| | | % within OUTCOME | 0.0% | 37.8% | 34.0% |
| | 5a | Count | 0 | 11 | 11 |
| | | % within OUTCOME | 0.0% | 24.4% | 22.0% |
| | 5b | Count | 5 | 17 | 22 |
| | | % within OUTCOME | 100.0% | 37.8% | 44.0% |
| Total | | Count | 5 | 45 | 50 |
| | | % within OUTCOME | 100.0% | 100.0% | 100.0% |

**Chi-Square Tests**

| DYSPNEA * OUTCOME crosstab | Value | df | Asymptotic Significance (2-sided) |
|---|---|---|---|
| Pearson Chi-Square | 7.071[a] | 2 | .029 |
| Likelihood Ratio | 8.926 | 2 | .012 |
| N of Valid Cases | 50 | | |

a. 3 cells (50.0%) have expected count less than
5. The minimum expected count is 1.10.

**Eosinopenia and outcome:**

The mortality among patients with eosinopenia is 25% ( 3/5). The mortality among patients without eosinopenia is 4.4% (2/45). Among the study population, patients having eosinopenia had higher mortality. This association between eosinopenia and outcome is statistically significant at p=0.005.

**Consolidation and Outcome:**

3 out of the 5 (60%) patients in the Non survivors group had chest X- ray features of consolidation. Out of 45 patients who survived 3 (6.6%) had consolidation. Presence of consolidation is associated with higher mortality. This association is statistically significant at p=0.009.

**Acidemia and outcome:**

All 5 (100%) patients with in-hospital mortality had arterial blood pH <7.30. 6 out of 45(13.3%) patients who survived had acidemia. Presence of acidemia is associated with higher in-hospital mortality. This relation is statistically significant at p=0.001.

**Atrial fibrillation and outcome:**

In the study group 4 patients had atrial fibrillation. All the patients died during the course of treatment. In terms of percentage, 80% of the patients with in-hospital mortality had atrial fibrillation. Presence of atrial fibrillation is associated with higher mortality. By Chi square thisrelation is statistically significant at p=0.001.

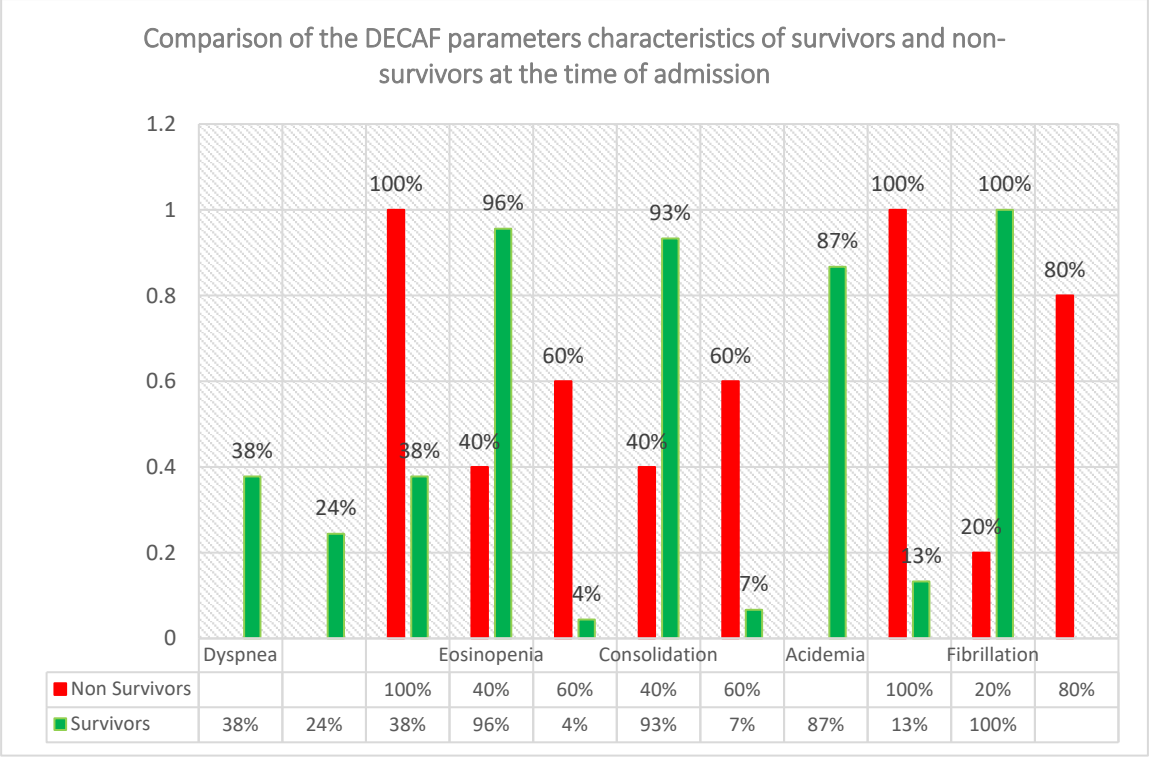



**Fig. 20 Comparison of the DECAF parameters characteristics of survivors and non-survivors at the time of admission**

| | | Non survivors | | Survivors | | p value |
|---|---|---|---|---|---|---|
| | | N | % | N | % | |
| Dyspnea | 4 | 0 | | 17 | 37.80% | 0.029* |
| | 5a | 0 | | 11 | 24.40% | |
| | 5b | 5 | 100.00% | 17 | 37.80% | |
| Eosinopenia | Absent | 2 | 40.00% | 43 | 95.60% | 0.005* |
| | present | 3 | 60.00% | 2 | 4.40% | |
| Consolidation | Absent | 2 | 40.00% | 42 | 93.30% | 0.009* |
| | present | 3 | 60.00% | 3 | 6.70% | |
| Acidemia | Absent | 0 | | 39 | 86.70% | 0.001* |
| | present | 5 | 100.00% | 6 | 13.30% | |
| Fibrillation | Absent | 1 | 20.00% | 45 | 100.00% | 0.001* |
| | present | 4 | 80.00% | 0 | | |

**Fisher exact test; shows (*p<0.05)**

**Table – 8. Comparison of the DECAF parameters characteristics of survivors and non-survivors at the time of admission**

**The DECAF score and outcome:**

The DECAF score comprising the five variables – Dyspnea, Eosinopenia, Consolidation, Acidemia, atrial Fibrillation is strongly associated with outcome. There is no mortality in the in patients with DECAF score between 0-2(low and intermediate risk group). The mortality rate for patients getting score of 3 and above(high risk group) is 5 out of 11. In terms of percentage this is 45.4%. The higher is the DECAF score , the higher is the mortality. This relation is statistically significant at p=0.001.

**Table – 9. DECAF Score Vs Outcome**

| Crosstabulation DECAF__SCORE * OUTCOME | | | OUTCOME | | Total |
|---|---|---|---|---|---|
| | | | Non survivors | Survivors | |
| DECAF_ _SCORE | 0 | Count | 0 | 17 | 17 |
| | | % within OUTCOME | 0.0% | 37.8% | 34.0% |
| | 1 | Count | 0 | 9 | 9 |
| | | % within OUTCOME | 0.0% | 20.0% | 18.0% |
| | 2 | Count | 0 | 13 | 13 |
| | | % within OUTCOME | 0.0% | 28.9% | 26.0% |
| | 3 | Count | 0 | 5 | 5 |
| | | % within OUTCOME | 0.0% | 11.1% | 10.0% |
| | 4 | Count | 0 | 1 | 1 |
| | | % within OUTCOME | 0.0% | 2.2% | 2.0% |
| | 5 | Count | 5 | 0 | 5 |
| | | % within OUTCOME | 100.0% | 0.0% | 10.0% |
| Total | | Count | 5 | 45 | 50 |
| | | % within OUTCOME | 100.0% | 100.0% | 100.0% |

**Chi-Square Tests**

| DECAF score *OUTCOME | Value | df | Asymptotic Significance (2-sided) |
|---|---|---|---|
| Pearson Chi-Square | 50.000[a] | 5 | .000 |
| Likelihood Ratio | 32.508 | 5 | .000 |
| Linear-by-Linear Association | 26.060 | 1 | .000 |
| N of Valid Cases | 50 | | |

a. 9 cells (75.0%) have expected count less than 5. The minimum expected count is .10.

**Table – 10. DECAF Score Grade Vs Outcome**

| Crosstabulation DECAF_score_grade * OUTCOME | | | OUTCOME | | Total |
|---|---|---|---|---|---|
| | | | Non survivors | Survivors | |
| DECAF_score_grade | Low risk | Count | 0 | 26 | 26 |
| | | % within OUTCOME | 0.0% | 57.8% | 52.0% |
| | Moderate risk | Count | 0 | 13 | 13 |
| | | % within OUTCOME | 0.0% | 28.9% | 26.0% |
| | High risk | Count | 5 | 6 | 11 |
| | | % within OUTCOME | 100.0% | 13.3% | 22.0% |
| Total | | Count | 5 | 45 | 50 |
| | | % within OUTCOME | 100.0% | 100.0% | 100.0% |

a. 3 cells (50.0%) have expected count less than 5. The minimum expected count is 1.10.

**Chi-Square Tests**

| DECAF_score_grade * OUTCOME | Value | df | Asymptotic Significance (2-sided) |
|---|---|---|---|
| Pearson Chi-Square | 19.697[a] | 2 | .000 |
| Likelihood Ratio | 17.350 | 2 | .000 |
| Linear-by-Linear Association | 14.156 | 1 | .000 |
| N of Valid Cases | 50 | | |

**DECAF score**

| | Non survivors | | survivors | | |
|---|---|---|---|---|---|
| 0 | 0 | | 17 | 37.8% | |
| 1 | 0 | | 9 | 20.0% | 0.001 |
| 2 | 0 | | 13 | 28.9% | |
| 3 | 0 | | 5 | 11.1% | |
| 4 | 0 | | 1 | 2.2% | |
| 5 | 5 | 100.0% | 0 | | |

**Fisher exact test test; shows (*p<0.05)**

**DECAF grading**

| | Non survivors | | survivors | | P value |
|---|---|---|---|---|---|
| **Low risk** | 0 | | 26 | 57.8% | |
| **Moderate risk** | 0 | | 13 | 28.9% | 0.001 |
| **High risk** | 5 | 100% | 6 | 13.3% | |

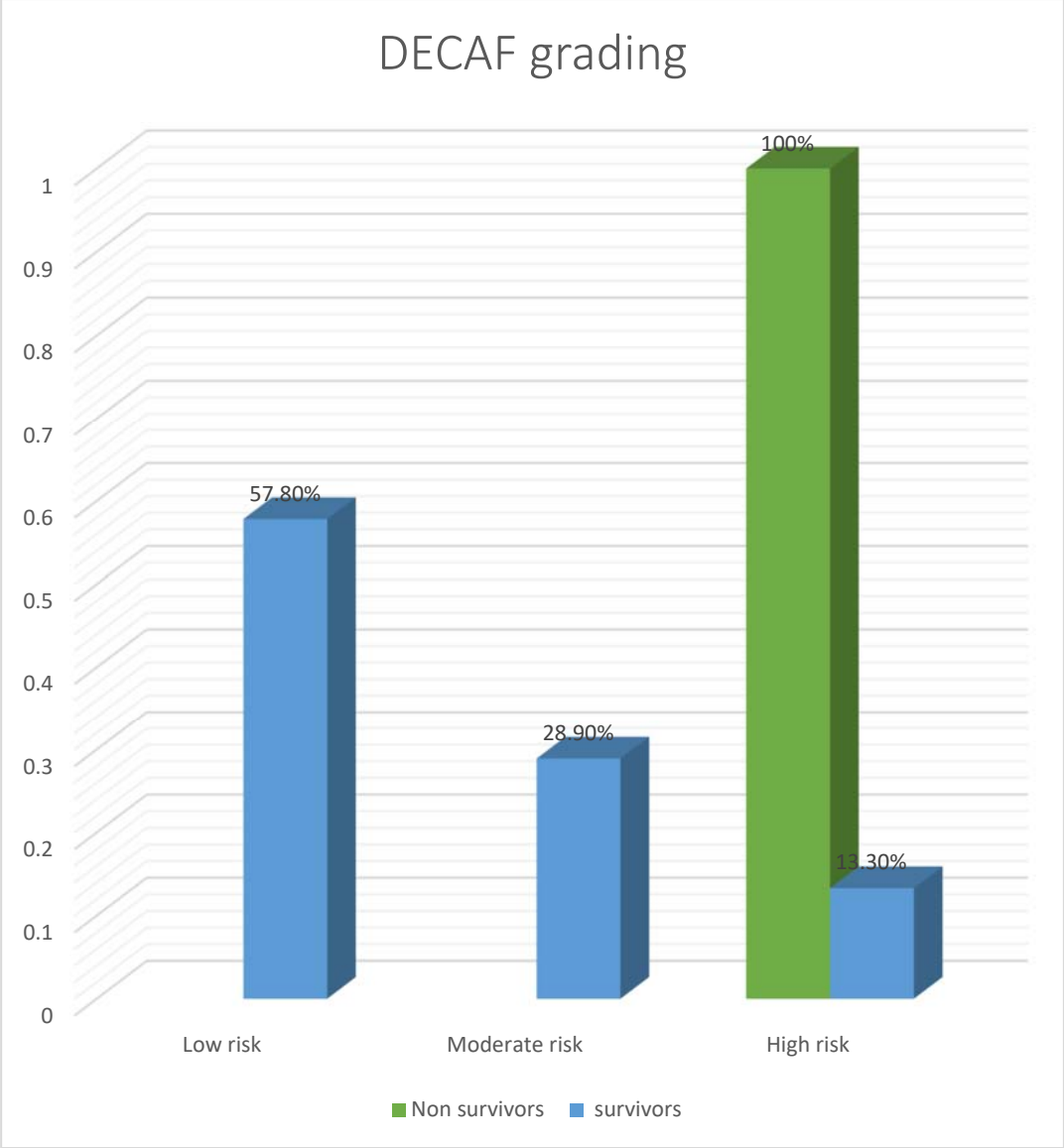



**Fig. 21. DECAF Grading Vs outcome**

Significant association exists between DECAF score and outcome.

**Use of ventilator and mortality:**

The mortality rate among patients who were ventilated and those not ventilated are 100% and 8.9% respectively. There was higher mortality among patients who were ventilated. This relation between use of ventilator and outcome is statistically significant at p=0.001.

**Hospital stay and outcome:**

The average duration of hospital stay for the survivors group was5 days . whereas the average duration of hospital stay for the non survivors group was 9.4 . The higher is the DECAF score the longer is the hospital stay. This association between the DECAF score and in-hospital stay is statistically significant at p=0.01.

**Previous episodes and outcome:**

Regarding number of pervious episodes of acute exacerbation mean episode was 0.54 and standard deviation was 0.68.minimum episode was 0 and maximum episode was 2.

Among non survivors mean episode was 1.4 and survivors was 0.4. so it explained that if the number of previous episode increased means the mortality risk also increased. This association between the previous episodes and in-hospital stay is statistically significant at p=0.007.

**Table 11. Hospital stay & Number of previous episode Vs Outcome**

| | Null Hypothesis | Test | Sig. |
|---|---|---|---|
| 1 | The distribution of HOSPITAL_STAY is the same across categories of OUTCOME. | Independent-Samples Mann-Whitney U Test | .000[a] |
| 2 | The distribution of NO_OF_PREVIOUS_EPISODE is the same across categories of OUTCOME. | Independent-Samples Mann-Whitney U Test | .007[a] |

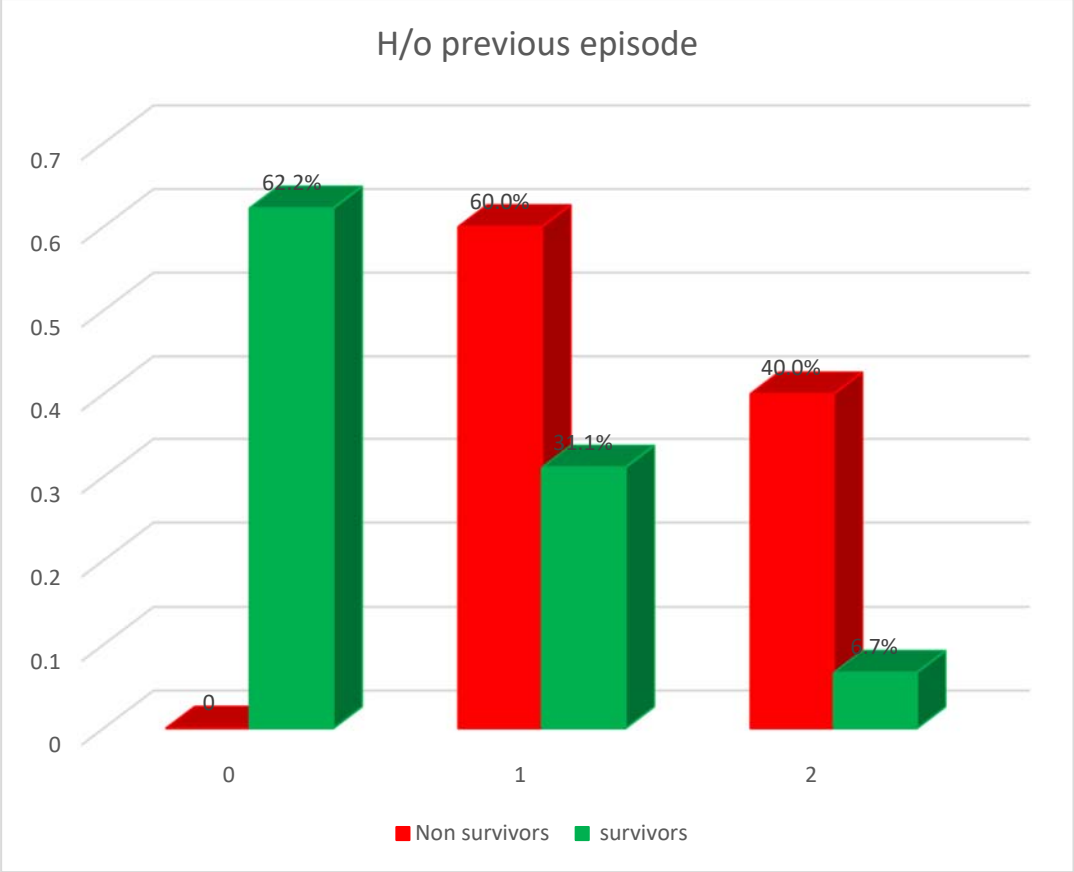



**Fig. 22. H/O Previous episode vs Outcome**

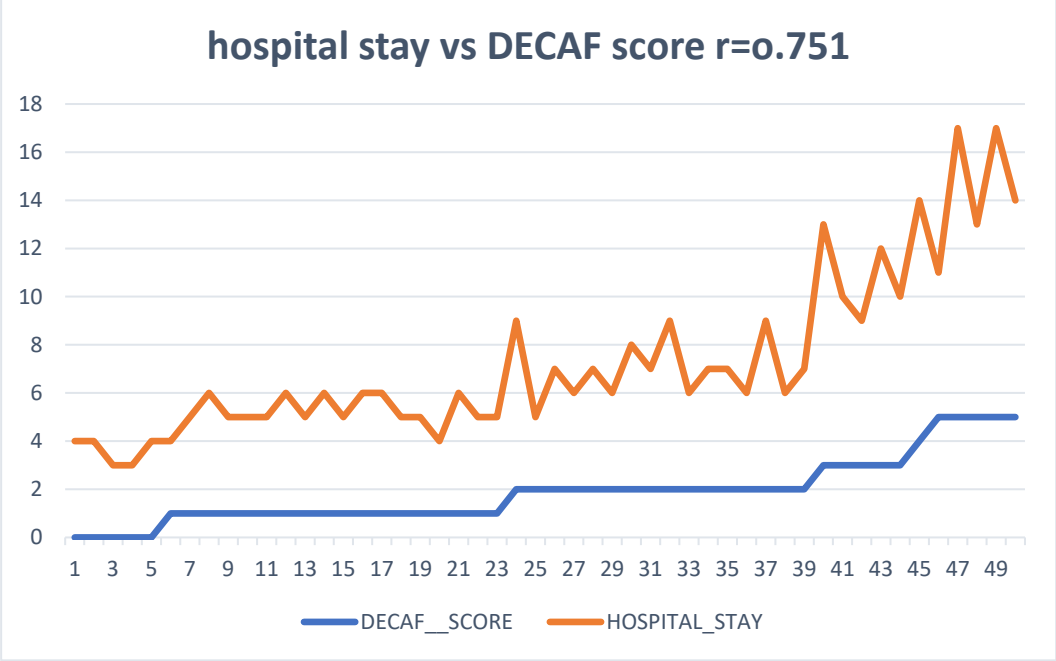



**Fig. 23. Hospital stay Vs DECAF score**

# DISCUSSION

# DISCUSSION

## Clinical Profile of the study population:

A total of 50 patients were included in our study who are fulfilled our selection methods, inclusion and exclusion criteria. In our study age group of our patients ranged from 38 to 81. The mean age of the study population was 56.2 with a standard Deviation of 9.45.

Age distribution shows that we had more patients in older age groups than younger age groups. This is consistent with the fact that age is often listed as a risk factor for COPD[49]. It is unclear if healthy aging as such leads to COPD or if age reflects the sum of cumulative exposures throughout life. Beyond 70 years of age there are fewer patients. It is because of exclusion of patients with other co-morbidities. Since co-morbid illnesses are common with aged population we had this sort of age distribution of patients.

Out of the 50 patients in the study, 45 are male and 5 are female. Thus males accounted for 90% of our study population while females accounted for 10%.This could be attributed to low prevalence of smoking among ladies. This shows that smoking habit may not have entered into our female population as much as in the western literature. Another reason

could be that many female patients with COPD are usually branded as having asthma in our country.

The primary reason for females developing COPD in our country could be attributed to passive smoking, biomass exposure and post tuberculosis.

In the study population, 27 did not have any comorbid illness. The most common comorbidity among the study population is Pulmonary Tuberculosis Sequalae.12 patients had history and radiological features of prior pulmonary tuberculosis. 2 patients had systemic hypertension, 2 patients had chronic kidney disease, 2 patients had Diabetes mellitus 2 patients had both hypertension and diabetes mellitus, 1 patient had coronary artery disease, 1 patient had coronary artery disease and Systemic hypertension. 1 patient had Diabetes mellitus and Systemic hypertension and coronary artery disease. This is consistent with the finding that tuberculosis has been found to be risk factor for COPD[1]. In addition tuberculosis is a potential comorbidity in COPD patients. Severe respiratory infections have been associated with reduced lung function.

Cor pulmonale is classically defined as “hypertrophy of the right ventricle resulting from diseases affecting the function and/or structure of the lungs except when these pulmonary alterations are the result of diseases that primarily affect the left side of the heart”[51]. Out of 50 patients

included in the study 20 (40%) patients had cor pulmonale as evidenced on echo. Out of them 9 (18%) patients had mild pulmonary hypertension (PHT), 2 (4%) patients had moderate PHT, 9 (18%) patients had severe PHT. This is consistent with other studies that have reported prevalence varying considerably from 20%–91%[52,53] depending on the definition of pulmonary hypertension, the severity of lung disease in the group studied and the method of measuring the pulmonary artery pressure (PAP). During an exacerbation of COPD, PAP may rise by as much as 20 mm Hg and return to its baseline after recovery[53]. Pulmonary hypertension in COPD has been considered to be the result of hypoxic pulmonary vasoconstriction, polycythemia and destruction of the pulmonary vascular bed by emphysema. Recently, it has been recognized that hyperinflation and endothelial dysfunction also play a role in the pathogenesis of Pulmonary hypertension.

MRC dyspnea scale is used for dyspnea grading because it is simple and allows patients to indicate the level of breathlessness. Extended Medical Research Council (eMRC) is used since it includes functional dependence as well. The patients in the study were graded according to the extended Medical Research Council score.

Accordingly, 17 patients had eMRC grade 4, 11 patients had eMRC grade 5a and 22 patients had a score of 5b. In terms of percentage, the

distribution of patients in grades 4, 5a and 5b was 34, 22 and 44 respectively.

Since patients were admitted with acute exacerbation of COPD most of them had dyspnea at rest, grade 5 which was again subdivided into 5a and 5b based on functional dependence. The patients with grade 4 dyspnea, though they did not have dyspnea at rest, they were admitted for AECOPD with increased quantity and purulency of sputum.

Eosinopenia was defined as an absolute eosinophil count of less than $50/mm^3$. 5 out of 50 patients had eosinopenia. Hence 10% of the study population had low eosinophil count. The incidence of eosinopenia is comparatively lower in our study population compared to western literature. It has been shown in previous studies that eosinopenia accompanies the response to acute infection and inflammation[22]. Thus in AECOPD eosinopenia may reflect severity of accompanying acute inflammatory response.

Assessment of chest radiographs of patients at admission to confirm the presence of consolidation was done. Accordingly 6 (12%) patients had consolidation on chest radiograph. This is lower when compare to other studies which shows, prevalence of 32.5% of consolidation in patients with AECOPD was reported by J Steer et al[19] in his study of 920 patients. In two UK national audits[26,27], consolidation was reported in 16% of all

admissions and in 34% of patients requiring ventilator assistance. Many a times the cause for an acute exacerbation of COPD is infectious and related to viralor bacterial infection.

Out of 50 patients, 11 (22%) had acidemia.This is lower with various studies that have reported a prevalence between 25% to 53%[19,21]. In a study involving consecutive patients admitted with AECOPD over one and half year period in UK, the incidence of acidemia was 27.9%.

Respiratory acidosis in COPD is secondary to hypoventilation. It includes multiple mechanisms including decreased responsiveness to hypoxia and hypercapnia, increased ventilation-perfusion mismatch leading to increased dead space ventilation and decreased diaphragmatic function due to fatigue and hyperinflation.

Among the study population 4 (8%) patients had atrial fibrillation, while the remaining 46 did not have fibrillation. This incidence is lower compared to other studies which have reported an occurrence of above 12%[39]. Acidemia, drugs and corpulmonale contribute to occurrence of arrythmias in COPD patients. It is postulated that ectopic beats initiating atrial fibrillation originate in the walls of pulmonary veins and it could be triggered by changes in gas composition[38].

The average duration of hospital stay for the study group was 5 days. The shortest length of hospital stay was 3 days and the longest length of hospital stay was 12 days.

In the study by Ying et al[54] in Oslo involving 590 patients admitted with AECOPD, the median length of hospital was 6 days. Various studies have illustrated wide range of hospital stay between 3-11days.

The mortality rate for the study population was 5 out of 50 (10%). 45 patients were improved, clinically defined as subjective sense of improvement and objective improvement in dyspnea scoring they were categorised under survivors, 5 patients died in the hospital. This study confirms the findings of previous studies. Karin H Groenewegen et al[33], in a study of 171 patients admitted with AECOPD showed the mortality rate during hospital stay was 8%, increasing to 23% after 1 year of follow-up. In the study byJ Steer et al[19] the in-hospital mortality rate was 10.4% (96/920) . In the study by Connors et al[55] the in-hospital mortality was 11%.

Out of the 50 patients 9 (18%) were put on ventilator, 5 were puton Non-invasive ventilation and 4 were put on Invasive ventilation.. This is slightly higher when compared with the previous studies that have reported ventilator use between 8 to 12% in patients getting admitted with AECOPD[32,33]. Regarding number of pervious episodes of acute

exacerbation mean episode was 0.54 and standard deviation was 0.68.minimum episode was 0 and maximum episode was 2.

**Factors influencing in-hospital prognosis:**

When age was taken as scattered variables, older patients showed higher mortality. However, there is no statistically significant association between the age and outcome, P≥0.05(P=0.059).

In the study group, the use of ventilator increases as the age advances. The use of ventilator is more with older patients. In the study population the average number of hospital stay is 5 days. It is seen that as age advances the duration of hospital stay increases with older people having the need to stay longer. These findings show that younger patients have milder forms of the disease compared to older patients. This could be attributed to decline in lung function as age advances and presence of comorbid illness which are more in older patients.

The mortality among female patients in the study is 1 out of 5 (20%). The moratlity among the male patients is 4 out of 45 (8.9%).There is no significant association between gender and outcome. This could be attributed to the very low number of female participants in the study. Male patients had longer in-hospital stay compared to female patients. 8 out of 45 (17.8%) male patients were put on ventilator whereas 1 out of 5 (20%)

female patients were ventilated. There is no statistically significant gender difference in the use of ventilator among the study population. This agrees with the findings by S Yu et al[56] and Cooper et al[57] who showed that gender was not an independent risk factor for short or long term prognosis in acute exacerbation of COPD.

The mortality among patients with cor pulmonale is 25% ( 5/20). The mortality among patients without cor pulmonale is 0% (0/30). Among the study population, patients having cor pulmonale and pulmonary hypertension had higher mortality. Patients who did not have PHT were survived. This association between cor pulmonale and outcome is statistically significant at p=0.001. There was increased need for usage of ventilators in patients with pulmonary hypertension compared to patients without PHT. Most of the patients without PHT didn't require ventilator. Patients with corpulmonale had a longer duration of hospital stay. This finding agrees with various studies by JJ Soler et al[58], M Oswald et al[59] which have showed that corpulmonale is an adverse prognostic variable in AECOPD.

In patients getting admitted with AECOPD, as the eMRC dysnea grade increases, the mortality increases. All the patients in the score of 4 and 5a were survived. Mortality is predominantly seen in eMRC 5b group. The mortality rate among eMRC 5b is 5 out of 22(22.7%). The relation is

statistically significant at p=0.029.With increasing grade of dyspnea there is increased usage of ventilator support. None of the patients in the score of 4 and 5a required ventilator whereas 81.8% in the dyspnea score group of 5b required ventilator. This association is statistically significant at p=0.000. With increasing grade ofdyspnea, the in-hospital stay for patients admitted with AECOPD increases . This association is statistically significant at p=0.000. In the study by E Steer et al[21] the in hospital mortality rate for eMRCD 5b patients was 33.1%. The findings of the present study are consistent with previous studies[19,21] which have showed that severity of dyspnea is strongly associated with in-hospital mortality. By combining MRCD scale with a person's ability to manage personal care (eMRCD) the predictive value of dyspnea scoring is improved.

The mortality among patients with eosinopenia is 25% ( 3/5). The mortality among patients without eosinopenia is 4.4% (2/45). Among the study population, patients having eosinopenia had higher mortality. This association between eosinopenia and outcome is statistically significant at p=0.005.

The finding of our study is consistent with other studies by Holland et al[25] andJ steer et al[19] that have showed eosinopenia to be a significant prognostic factor in AECOPD.

3 out of the 5 (60%) patients in the Non survivors group had chest X- ray features of consolidation. Out of 45 patients who survived 3 (6.6%) had consolidation. Presence of consolidation is associated with higher mortality. This association is statistically significant at p=0.009.This is comparable to previous studies by Liebermann et al, J Steer et al who have shown that mortality among pneumonia associated AECOPD is more than non pneumonic AECOPD. Community acquired pneumonia is common among patients hospitalised with AECOPD and usually causes the exacerbation to have more severe clinical and laboratory parameters.

All 5 (100%) patients with in-hospital mortality had arterial blood pH <7.30. 6 out of 45(13.3%) patients who survived had acidemia. Presence of acidemia is associated with higher in-hospital mortality. This relation is statistically significant at p=0.001.According to previous studies[56], the frequency of hypercapnic respiratory failure in patients with AECOPD varies from 16-35% with overall mortality of 35-43%[5]. Our study has shown higher mortality among hypercapnic patients compared to previous studies.

The level of hypercapnia, suggestive of chronic alveolar hypoventilation, reflects the severity of the underlying respiratory condition, and Patients with chronic hypercapnia, who comprised the

majority of our study population, have a worse prognosis than patients with normoventilation.

In the study group 4 patients had atrial fibrillation. All the patients died during the course of treatment. In terms of percentage, 80% of the patients with in-hospital mortality had atrial fibrillation. Presence of atrial fibrillation is associated with higher mortality. By Chi square thisrelation is statistically significant at p=0.001.

This is in accordance with study by J Steer et al[19] in which 26% of patients with in hospital mortalityhad atrial fibrillation. Our study shown higher mortality when compared to Previous studies. And Previous studies have shown that occurrence of atrial fibrillation is associated with poor prognosis.

The average hospital stay for study population was 5 days. Longer in- hospital stay was associated with poor prognosis. There was significant relationship between duration of hospital stay and outcome. There are no clinical trials that have evaluated the optimal duration of treatment in AECOPD. Our study shows that prolonged hospital stay is associated with poor outcome.

The mortality rate among patients who were ventilated and those not ventilated are 100% and 8.9% respectively. There was higher mortality among patients who were ventilated. This relation between use of

ventilator and outcome is statistically significant at p=0.001.

Patients who were put on ventilator had longer hospital stay, compared to patients not ventilated. Out of the 50 patients 9(18%) were put on ventilator, 5 were puton Non-invasive ventilation and 4 were put on Invasive ventilation.Use of ventilator improves acute respiratory acidosis, decreases respiratory rate, work of breathing, severity of breathlessness. However the higher incidence of mortality and longer hospital stay in patients put on ventilator may be attributed to the fact that weaning or discontinuation from mechanical ventilation can be difficult and hazardous in patients with COPD[1]. In patients on ventilatorysupport there is higher incidence of ventilator associated pneumonia,barotraumas and need for longer antibiotic usage. This study highlights the need for trial of non invasive ventilation even in conditions where invasive ventilation is generally indicated. NIV can reduce the complications associated with intubation like ventilator associatedpneumonia.

**The use of DECAF Score in assessing in-hospital prognosis**:

Out of 50 patients studied, 26 patients had a DECAF score between 0-1 (low risk), 13 patients had a DECAF score of 2 (intermediate risk) and 11 patients had a DECAF score between 3-6(high risk). In terms of percentage this is 52%, 26% and 22% respectively. This is consistent with the study by J.Steer et al[19] , in which the low risk group comprised 53.5% of the study population, intermediate risk group comprised 24.5%of the

study population, high risk group comprised 22%of the study population. This shows that in a given population getting admitted with AECOPD, low risk group out number the high risk patients.This may be due to the fact that these patients approach health care facilities early during the course of exacerbation. The DECAF scorecomprising the five variables – Dyspnea, Eosinopenia, Consolidation, Acidemia, atrial Fibrillation is strongly associated with outcome. There is no mortality in the in patients with DECAF score between 0-2. The mortality rate for patients getting score of 3 and above is 5 out of 11. In terms of percentage this is 45.4%. The higher is the DECAF score , the higher is the mortality. This relation is statistically significant at p=0.001. Our study agrees with the findings by J Steer et al[19]. In their study involving 920 AECOPD patients, the strongest five categorical variables strongly associated with in-hospital mortality were selected and the DECAF score devised. They reported that in DECAF 0-1 the in- hospital mortality was 1.4%, in DECAF 2 mortality was 8.4%. and in DECAF 3-6 the mortality was 34.6%. As the DECAF score increases use of ventilator increases. In the low and intermediate risk group (DECAF 0-2) out of 39 no one was ventilated. In the high risk group 9 out of 11persons were ventilated. In terms of percentage this is 0% and 81.8% in the low-intermediate risk group and high risk group respectively. This association is statistically significant at p=0.000. The average duration of hospital stay for the low to intermediate risk group (DECAF 0-2) was

5 days . whereas the average duration of hospital stay for the high risk group (DECAF 3-6) is 9.4 . The higher is the DECAF score the longer is the hospital stay. This association between the DECAF score and in-hospital stay is statistically significant at p=0.000. To our knowledge there are no previous studies that have evaluated the association between DECAF score and need for ventilator use or duration of hospital stay. Regarding number of pervious episodes of acute exacerbation mean episode was 0.54 and standard deviation was 0.68 minimum episode was 0 and maximum episode was 2.

Among non survivors mean episode was 1.4 and survivors was 0.4. so it explained that if the number of previous episode increased means the mortality risk also increased. This association between the previous episodes and in-hospital stay is statistically significant at p=0.007.

**Factors related to DECAF score:**

The number of patients less than 60 years of age in the low, intermediate and high risk groups are 19, 11 and 4 respectively. The number of patients more than 60 years of age in the low, intermediate and high risk groups are 7, 2 and 7 respectively. There is no significant relation between age and DECAF score. The number of female patients in low, intermediate and high risk groups are 1, 3 and 1 respectively. In terms of percentage this is 20, 60 and 20 respectively. The number of male patients

in low, intermediate and high risk groups are 25, 10 and 10 respectively. In terms of percentage this is 55.5, 22.2 and 22.2 respectively. There was no significant association between DECAF score and gender.

In patients with dyspnea grade 4 all 17 patients were in low risk group. In patients with dyspnea grade 5a the number of patients in low and intermediate groups are 9 and 2 respectively. In patients with dyspnea grade 5a, No patients belongs to high risk group. In patients with dyspnea grade 5b the number of patients in intermediate and high risk groups are 11 and 11 respectively. No patients belongs to low risk group In patients with dyspnea grade 5b. Patients having a dyspnea grade of 4 and 5a had better prognosis than patients having a grade 5b. Patients with higher grade of dyspnea according to eMRC had higher DECAF score. This association is statistically significant p=0.029. In patients with cor pulmonale the number of patients in low, intermediate and high risk groups are 7 , 3 and 10 respectively. In terms of percentage this is 41.2 , 11.7 and 58.8 respectively. In patients without cor pulmonale, the number of patients in low, intermediate and high risk groups are 19 , 10 and 1 respectively. In terms of percentage this is 63.3 , 33.3 and 3.3 respectively. Patients with cor pulmonale had higher DECAF score. Most of the patients without pulmonary hypertension had a score of 0 to 2 withgood prognosis. This association between DECAF score and cor pulmonale is statistically

significant at p=0.001. Hence higher dyspnea grade and presence of corpulmonale may be taken as indirect markers of higher DECAF score.

In patients with previous episodes of exacerbation, number of patients in low, intermediate and high risk groups are 9, 5 and 8 respectively. In terms of percentage this is 41, 22.7 and 36.3 respectively. In patients without previous episodes of exacerbation, the number of patients in low, intermediate and high risk groups are 17, 8 and 3 respectively. In terms of percentage this is 61, 28 and 11 respectively. Patients with previous episodes of exacerbation had higher DECAF score. Most of the patients without previous episodes of exacerbation had a score of 0 to 2 withgood prognosis. This association between DECAF score and previous episodes of exacerbation statistically significant at p=0.007. Hence higher dyspnea grade and presence of previous episodes of exacerbation may be taken as indirect markers of higher DECAF score.

# CONCLUSION

# CONCLUSION

- The present study shows that the DECAF score, which includes five parameters such as dyspnea, eosinopenia, consolidation, acidemia, and atrial fibrillation, is strongly associated with outcome in patients admitted with an acute exacerbation of COPD.
- Based on DECAF score these patients are classified into low risk (DECAF 0-1), intermediate risk (DECAF-2) and high risk (DECAF 3-6).
- The higher the DECAF score, the higher is the mortality, the longer is the hospital stay and the need for use of ventilator is higher.
- Presence of cor pulmonale and previous episodes of exacerbation can be considered as a surrogate marker of higher DECAF score.

The DECAF score is a simple, effective and quick evaluation of clinical parameters that predict mortality in patients admitted with acute exacerbation of Chronic Obstructive Pulmonary Disease. This scoring system incorporates routinely available indices. and it effectively stratifies patients admitted with AECOPD into mortality risk categories.

Hence measuring the DECAF score at the time of admission in AECOPD helps in decision regarding

1. Prompt care escalation at an early stage
2. Deciding the location of care – Intensive care unit or ward
3. Identifying whether or not a ventilator is required.
4. Deciding on end-of-life care
5. Assists the physician in informing the patient and families about the prognosis and short-term risks associated with exacerbation.

# LIMITATIONS

# LIMITATIONS

**The major limitations of our study are:**

1. There is a lack of post-hospital follow-up data, which may be required for validation of the prognostic features identified in this study.
2. The number of female patients enrolled within the study was much lower than the expected. However since consecutive patients were recruited, this has to be considered as reflection of what occurs in the real life setting.

# ANNEXURES

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# ABBREVIATIONS

| | |
|---|---|
| AECOPD | Acute Exacerbation of Chronic Obstructive Pulmonary Disease |
| AF | Atrial Fibrillation |
| COPD | Chronic Obstructive Pulmonary Disease |
| DECAF | Dyspnea, Eosinopenia, Consolidation, Acidemia,Fibrillation. |
| eMRCD | extended Medical Research Council Dyspnea Score |
| FEV1 | Forced Expiratory Volume in one second |
| FVC | Forced Vital Capacity |
| MRCD | Medical Research Council Dyspnea Score |
| NPAE | Non pneumonic Acute Exacerbation |
| PNAE | Pneumonic Acute Exacerbation |

# ஆராய்ச்சி ஒப்புதல் படிவம்

**ஆராய்ச்சி தலைப்பு :** நாட்பட்ட நுரையீரல் காற்றுக்குழாய் அடைப்பு நோயினால் மருத்துவமனையில் அனுமதிக்கப்படும் நோயாளிகளுக்கு **DECAF Score** கொண்டு நோயின் விளைவுகளை முன்கணிப்பு செய்தல்.

பெயர் தேதி

வயது உள் நோயாளி எண்

பாலினம் ஆராய்ச்சி சேர்க்கை எண்

எனக்கு நாட்பட்ட நுரையீரல் காற்றுக்குழாய் அடைப்பு நோய் உள்ளதையும் அதன் விளைவுகளையும் விவரங்களையும் மருத்துவர் நன்கு தெரிவித்துள்ளார். மேற்கொள்ளப்படும் ஆராய்ச்சியின் விவரங்களையும் அதன் நோக்கங்களையும் முழுமையாக எனக்கு விளக்கப்பட்டுள்ளது. எனக்கு விளக்கப்பட்ட விவரங்களை நன்கு புரிந்துக்கொண்டு இந்த ஆராய்ச்சியில் பங்குக்கொள்வதற்கு சம்மதம் அளிக்கிறேன். இந்த ஆராய்ச்சியிற்காக இரத்த பரிசோதனை, சுருள்படும், மார்பகப்படம் எடுப்பதற்கும் சம்மதம் அளிக்கிறேன்.

இந்த ஆராய்ச்சியில் பிறரின் நிர்பந்தம் இன்றி என் சொந்த விருப்பத்தின் பேரில் நான் பங்கு பெறுகிறேன் மற்றும் நான் இந்த ஆராய்ச்சியிலிருந்து எந்நேரமும் பின்வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்துக்கொண்டேன். நான் என்னுடைய சுய நினைவுடன் மற்றும் முழு சுதந்திரத்துடன் இந்த மருத்துவ ஆராய்ச்சியில் என்னை சேர்த்துக்கொள்ள சம்மதிக்கிறேன்.

கையொப்பம்.

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# CERTIFICATE - II

This is to certify that this dissertation titled **"ROLE OF THE DECAF SCORE IN PREDICTING IN HOSPITAL MORTALITY IN ACUTE EXACERBATION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE"** is the bonafide work of the candidate **DR. LEO CLINGTON A** with registration number **201911109** for the award of M.D., in the branch of **GENERAL MEDICINE**. I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows 11 percentage of plagiarism in the dissertation.

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**CDSCO:Reg.No.ECR/1365/Inst/TN/2020 &**
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| | | |
|---|---|---|
| **Study Title** | : | Role of the DECAF Score in predicting in hospital mortality in Acute Exacerbation of Chronic Obstructive Pulmonary Disease |
| **Principal Investigator** | : | Dr.A.Leo Clington |
| **Designation** | : | PG in MD., General Medicine (2019-2022) |
| **Guide** | : | Dr.David Pradeep kumar, MD., (G.M)<br>Professor of General Medicine |
| **Department** | : | Department of General Medicine,<br>Government Rajaji Hospital & Madurai Medical College,<br>Madurai |

The request for an approval from the Institutional Ethics Committee (IEC) was considered on the IEC meeting held on **22.06.2021** at GRH Auditorium, Govt. Rajaji Hospital, Madurai at 10.00 A.M

The Members of the committee, the Secretary and the Chairman are pleased to inform you that your proposed project mentioned above is **Approved.**

You should inform the IEC in case of any changes in study procedure, methodology, sample size investigation, Investigator or guide or any other changes.

1. You should not deviate from the area of work for which you had applied for ethical clearance.

2. You should inform the IEC immediately, in case of any adverse events or serious adverse reactions. If encountered during from study.

3. You should abide to the rules and regulations of the institution(s)

4. You should complete the work within the specific period and if any extension is required, you should apply for the permission again for extension period.

5. You should submit the summary of the work to the ethical committee on completion of the study.

**MEMBER SECRETARY,**
IEC, Madurai Medical College,
Madurai
Dr.K.RAADHIKA, M.D(Pharm)
Associate Professor
Member Secretary
IEC - Madurai Medical College
Madurai.

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Madurai
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Madurai

# MASTER CHART

| NAME | AGE | SEX | COMORBIDITY | COR_PULMONALE | DYSPNEA | EOSINOPENIA | CONSOLIDATION | ACIDEMIA | FIBRILLATION | DECAF_score_grade | DECAF__SCORE | HOSPITAL_STAY | OUTCOME | USE_OF_VENTILATOR | NO_OF_PREVIOUS_EPISODE |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| SEKAR | 38 | M | NIL | NO | 4 | NO | NO | NO | NO | LOW RISK | 0 | 4 | 1 | NO | 1 |
| GOVINDARAJ | 56 | M | NIL | NO | 4 | NO | NO | NO | NO | LOW RISK | 0 | 4 | 1 | NO | 0 |
| BALU | 38 | M | NIL | NO | 4 | NO | NO | NO | NO | LOW RISK | 0 | 3 | 1 | NO | 0 |
| NAVANEETAHN | 48 | M | CAD,SHTN | NO | 4 | NO | NO | NO | NO | LOW RISK | 0 | 3 | 1 | NO | 1 |
| SARAVANAN | 44 | M | NIL | MILD PHTN | 4 | NO | NO | NO | NO | LOW RISK | 0 | 4 | 1 | NO | 0 |
| AROKIYASAMY | 59 | M | PTB SEQUALE | MILD PHTN | 4 | NO | NO | NO | NO | LOW RISK | 0 | 3 | 1 | NO | 0 |
| SENTHILKUMAR | 51 | M | NIL | NO | 4 | NO | NO | NO | NO | LOW RISK | 0 | 4 | 1 | NO | 0 |
| ANNAMALAI | 45 | M | CKD | NO | 4 | NO | NO | NO | NO | LOW RISK | 0 | 5 | 1 | NO | 0 |
| GANESAN | 50 | M | NIL | NO | 4 | NO | NO | NO | NO | LOW RISK | 0 | 4 | 1 | NO | 0 |
| ALAGUPANDI | 53 | M | NIL | NO | 4 | NO | NO | NO | NO | LOW RISK | 0 | 4 | 1 | NO | 0 |
| SELVAGANESH | 62 | M | NIL | MILD PHTN | 4 | NO | NO | NO | NO | LOW RISK | 0 | 4 | 1 | NO | 1 |
| MUTHUKUMAR | 53 | M | NIL | NO | 4 | NO | NO | NO | NO | LOW RISK | 0 | 5 | 1 | NO | 0 |
| PANDI | 62 | M | PTB SEQUALE | MILD PHTN | 4 | NO | NO | NO | NO | LOW RISK | 0 | 4 | 1 | NO | 1 |
| IYAPPAN | 61 | M | NIL | NO | 4 | NO | NO | NO | NO | LOW RISK | 0 | 5 | 1 | NO | 0 |
| MOORTHY | 48 | M | PTB SEQUALE | NO | 4 | NO | NO | NO | NO | LOW RISK | 0 | 4 | 1 | NO | 0 |
| MOHAMMED ISMAIL | 39 | M | NIL | NO | 4 | NO | NO | NO | NO | LOW RISK | 0 | 5 | 1 | NO | 0 |
| ALAGAMMAL | 61 | F | NIL | NO | 4 | NO | NO | NO | NO | LOW RISK | 0 | 5 | 1 | NO | 0 |
| VELU PILLAI | 63 | M | NIL | NO | 5a | NO | NO | NO | NO | LOW RISK | 1 | 4 | 1 | NO | 1 |
| BALACHANDAR | 52 | M | NIL | NO | 5a | NO | NO | NO | NO | LOW RISK | 1 | 4 | 1 | NO | 0 |
| MUNISAMY | 65 | M | PTB SEQUALE | SEVERE PHTN | 5a | NO | NO | NO | NO | LOW RISK | 1 | 3 | 1 | NO | 1 |
| RAMKUMAR | 43 | M | NIL | NO | 5a | NO | NO | NO | NO | LOW RISK | 1 | 5 | 1 | NO | 0 |
| SELVAM | 47 | M | NIL | NO | 5a | NO | NO | NO | NO | LOW RISK | 1 | 4 | 1 | NO | 0 |
| ARUMUGAM | 58 | M | CAD | MILD PHTN | 5a | NO | NO | NO | NO | LOW RISK | 1 | 4 | 1 | NO | 1 |
| RAJKUMAR | 50 | M | NIL | NO | 5b | NO | NO | NO | NO | INTERMEDIATE RISK | 2 | 7 | 1 | NO | 0 |
| POONGOTHAI | 66 | F | PTB SEQUALE | MILD PHTN | 5b | NO | NO | NO | NO | INTERMEDIATE RISK | 2 | 3 | 1 | NO | 0 |
| SAKTHIVEL | 48 | M | NIL | NO | 5b | NO | NO | NO | NO | INTERMEDIATE RISK | 2 | 5 | 1 | NO | 0 |
| BEER MOHAMMED | 57 | M | SHTN | NO | 5a | YES | NO | NO | NO | INTERMEDIATE RISK | 2 | 4 | 1 | NO | 1 |
| SIVASAMY | 54 | M | NIL | NO | 5a | NO | NO | NO | NO | LOW RISK | 1 | 5 | 1 | NO | 0 |
| JEYARAJ | 67 | M | PTB SEQUALE | MILD PHTN | 5a | NO | NO | NO | NO | LOW RISK | 1 | 4 | 1 | NO | 1 |
| SHIEK DAWOOD | 60 | M | SHTN | NO | 5a | NO | NO | NO | NO | LOW RISK | 1 | 6 | 1 | NO | 1 |
| SUDHAGAR | 45 | M | NIL | NO | 5a | YES | NO | NO | NO | LOW RISK | 2 | 5 | 1 | NO | 0 |
| GOPAL | 52 | M | T2DM | NO | 5b | NO | YES | NO | NO | INTERMEDIATE RISK | 2 | 7 | 1 | NO | 0 |
| AMSAVALLI | 60 | F | NIL | NO | 5b | NO | NO | NO | NO | INTERMEDIATE RISK | 2 | 4 | 1 | NO | 2 |
| RAJASEKAR | 57 | M | NIL | NO | 5b | NO | NO | NO | NO | INTERMEDIATE RISK | 2 | 5 | 1 | NO | 0 |
| PONVANNAN | 57 | M | NIL | NO | 5b | NO | NO | NO | NO | INTERMEDIATE RISK | 2 | 5 | 1 | NO | 1 |
| KARUPASAMY | 58 | M | CKD | SEVERE PHTN | 5b | NO | NO | NO | NO | INTERMEDIATE RISK | 2 | 4 | 1 | NO | 2 |
| LAKSHMI | 58 | F | T2DM | NO | 5b | NO | YES | NO | NO | INTERMEDIATE RISK | 2 | 7 | 1 | NO | 0 |
| KRISHNAN | 50 | M | NIL | MILD PHTN | 5b | NO | NO | NO | NO | INTERMEDIATE RISK | 2 | 4 | 1 | NO | 0 |
| CHELLAIYA | 55 | M | NIL | NO | 5b | NO | NO | NO | NO | INTERMEDIATE RISK | 2 | 5 | 1 | NO | 1 |
| KARUPPAIYA | 61 | M | NIL | SEVERE PHTN | 5b | NO | NO | YES | NO | HIGH RISK | 3 | 10 | 1 | YES | 2 |
| SUBRAMANIAN | 62 | M | NIL | MILD PHTN | 5b | NO | NO | YES | NO | HIGH RISK | 3 | 7 | 1 | NO | 0 |
| MUNIYAN | 58 | M | PTB SEQUALE | SEVERE PHTN | 5b | NO | NO | YES | NO | HIGH RISK | 3 | 6 | 1 | YES | 0 |
| IRULANDI | 79 | M | PTB SEQUALE | MODERATE PHTN | 5b | NO | NO | YES | NO | HIGH RISK | 3 | 9 | 1 | YES | 1 |
| AANDIYAPAN | 78 | M | PTB SEQUALE | SEVERE PHTN | 5b | NO | NO | YES | NO | HIGH RISK | 3 | 7 | 1 | YES | 1 |
| DURAISAMY | 57 | M | T2DM, SHTN | NO | 5b | NO | YES | YES | NO | HIGH RISK | 4 | 10 | 1 | NO | 0 |
| KASI MUTHU | 55 | M | T2DM,SHTN,CAD | SEVERE PHTN | 5b | YES | YES | YES | NO | HIGH RISK | 5 | 6 | 0 | YES | 1 |
| PERAMAIYAN | 69 | M | PTB SEQUALE | SEVERE PHTN | 5b | NO | YES | YES | YES | HIGH RISK | 5 | 12 | 0 | YES | 2 |
| PARAMESHWARI | 59 | F | SHTN,T2DM | MODERATE PHTN | 5b | NO | YES | YES | YES | HIGH RISK | 5 | 8 | 0 | YES | 1 |
| SAHUL HAMMED | 60 | M | PTB SEQUALE | SEVERE PHTN | 5b | YES | NO | YES | YES | HIGH RISK | 5 | 12 | 0 | YES | 1 |
| RAMAIYA | 81 | M | PTB SEQUALE | SEVERE PHTN | 5b | YES | NO | YES | YES | HIGH RISK | 5 | 9 | 0 | YES | 2 |