Prognostic value of DECAF score in patients with acute exacerbation of COPD – A one year longitudinal study in KLE's Dr.Prabhakar Kore Hospital and Medical Research Centre, Belagavi.

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LIST OF ABBREVIATIONS

AECOPD	–	Acute Exacerbation of Chronic Obstructive Pulmonary Disease
BOLD	–	Burden of Obstructive Lung Disease
WHO	–	World Health Organization
ICMR	–	Indian Council of Medical Research
DECAF	–	Dyspnoea, Eosinopenia, Consolidation, Acidemia, Fibrillation
ATS	–	The American Thoracic Society
ERS	–	Alpha -1 antitrypsin
eMRCD	-	extended Medical Research Council Dyspnoea scoring
ABG	–	Arterial Blood Gas
BMI	–	Body Mass Index
CAT	–	COPD Assessment Test
COPD	–	Chronic Obstructive Pulmonary Disease
FEV1	–	Forced Expiratory Volume in 1 Second
FRC	–	Functional Residual Capacity
FVC	–	Forced Vital Capacity
GOLD	–	Global Initiative for Chronic Obstructive Lung Disease

MMP	–	Matrix Metalloproteinase
ROS	–	Reactive Oxygen Species
RV	–	Residual Volume
SERPINA	–	Serpin peptidase inhibitor, clade A, member 1
TNF Alpha	–	Tumor Necrosis Factor Alpha
TGF Beta	–	Transforming Growth Factor Beta
CURB	-	65 Confusion, Urea, Respiratory rate, Blood pressure, Age >65
APACHE II	–	Acute Physiology and Chronic Health Evaluation II
CAPS	–	COPD and Asthma Physiology Score
AUROC	–	Area Under Receiver Operating Characteristic
eMRCD	–	extended Medical Research Council Dyspnoea Score

ABSTRACT

BACKGROUND AND OBJECTIVES

Chronic Obstructive Pulmonary Disease (COPD) is a preventable, common and treatable disease that is characterized by persistent respiratory symptoms and airflow limitation due to airway and alveolar abnormalities usuallycontributed by significant exposure to noxious particles or gases. More than 30 lakh patients die every year due to COPD. It is the 4th major cause of mortality in the world.

Even though exacerbations of COPD are both very common and often fatal, it is difficult to comment on the outcome of hospitalised patient with AECOPD due to the scarce availability of scoring systems for the same.

A simple prediction tool, using indices commonly available during the time of admission in hospital, can stratify patients hospitalized with AECOPD accurately into clinically relevant risk categories and could, therefore, help clinicians in managing this fatal condition. The DECAF Score (Dyspnoea, Eosinopenia, Consolidation, Acidemia and Atrial Fibrillation Score) uses indices which are routinely available during the time of hospital admission to prognosticate the patients hospitalized with acute exacerbation of COPD. The objective of the present study is to evaluate the prognostic value of DECAF score in patients with acute exacerbation of COPD.

MATERIALS AND METHODS

The study was done on 70 patients, fulfilling inclusion criteria who were admitted in wards or ICU in KLE's Dr. Prabhakar Kore Hospital and MRC, Belagavi over a period of one year from January 2019 to December 2019.Participants were subjected to a detailed evaluation of their present symptoms, co morbidities, history of

smoking and other habits, any other exposure history, past and present history of exacerbations of COPD.

Routine workup for COPD including complete blood count with eosinophil count, ECG,arterial blood gas analysis, chest X-ray was done Dyspnoea of the patient was evaluated by eMRCD(extended Medical Research Council Dyspnoea) grading of 5a and 5b.Based on these investigations and dyspnoea scoring, the decaf score of each individual was obtained.After 5 days of admission, the outcome of patient in terms of mortality or continuation of same line of care or stepping down of care, was noted. The initial DECAF score of patients on admission and prognosis of the patients on the 5^{th} day was correlated. The statistical analysis was done using SPSS version 20.0 software. The chi square test and the multiple logistic regression analysis were performed.

RESULTS:

The mean age of our study population is 64.27 years with 50(71.43%) male patients among 70 patients enrolled in the study. Mean duration of COPD was 7.84 years. 47(67.14%) patients were smokers in our study with a mean pack years of 13.23 years.

Out of 70 patients, 26(37.14%) patients had DECAF score of 0 to 1, 26(37.14%) patients had DECAF score of 2 to 3 and 18(25.71%) patients had a score more than 3.All 26(100%) patients with score 0 to 1 improved, 22(84.26%) out of 26 patients with the score of 2 to 3 improved and only 3(16.67%) out of 15 patients with score more than 3 improved.

This data is statistically significant, with ‘p’ value of 0.0001. This shows that, DECAF score is a significant predictor of outcome. 8 patients in our study died accounting to 11.4 % of the population. All of their scores were more than 4.

Number of exacerbations per year,dyspnoea, consolidation, acidemia, atrial fibrillation and DECAF score in total were good predictors of outcome of AECOPD. Eosinopenia is not a good independent predictor of outcome of AECOPD in our study.

CONCLUSION

In our study, DECAF score is a good predictor of outcome in patients hospitalised with AECOPD (‘p’=0.0001).High DECAF score(more than 3) during the time of admission resulted in bad outcome, in patients with AECOPD. Patients with lower scores on admission had clinically better outcomes, tapered on antibiotics and other supportive treatment. They were discharged after full recovery, indicating good prognosis.

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INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a term used for chronic airflow obstruction which often develops due to tobacco smoking.[1,2] Global Initiative for Chronic Obstructive Lung Disease (GOLD) 2019 defined Chronic Obstructive Pulmonary Disease (COPD) as "A preventable, common and treatable disease that is characterized by persistent respiratory symptoms and airflow limitation due to airway and alveolar abnormalities usually contributed by significant exposure to noxious particles or gases."[2] The burden of COPD is expected to rise in the forthcoming decades, as there is continued exposure to risk factors of COPD. The significant risk factors include cigarette smoke, occupational dust, chemicals, outdoor and indoor air pollution, respiratory infections and ageing.2

More than 30 lakh patients die every year due to COPD. It is the 4th major cause of mortality in the world.[3] According to World Health Organization (WHO), by 2030 COPD will rise to thethird most significant cause of death. Mortality due to COPD is expected to rise by 160% within the next two decades.[4]The deaths in COPD usually occur due to pre-existing co morbidities like diabetes, ischemic heart disease, hypertension, renal disease, depression and osteoporosis rather than the actual pulmonary disease.[5]

"Acute Exacerbation of COPD(AECOPD) is an event which is characterized by the worsening of patient's respiratory symptoms which are beyond the day to day changes and needs change in medication."

AECOPD negatively affects the quality of a patient's life and also the lung function that takes many weeks to recover from. Those requiring hospitalisation due to AECOPD have considerable mortality.[6]

Even though exacerbations of COPD are both very common and often fatal, it is difficult to comment on the outcome of hospitalised patient with AECOPD.[7] The all-cause mortality in COPD patient for three years is 49%.[6]In those patients who require ventilatory support, mortality goes up to 40%. In stable patients, tools like the BODE score(Body-mass index, airflow Obstruction, Dyspnoea, and Exercise)are available to predict the outcome of the patient.[8] Research in predicting the prognosis of the patient with exacerbations of COPD necessitating hospitalization and ICU admission is scarce.[9]

Furthermore, not many prognostic tools used in stable disease were tested in patients who were hospitalised. A simple prediction tool, using indices commonly available during the time of admission in hospital, can stratify patients hospitalized with AECOPD accurately into clinically relevant risk categories and could, therefore, help clinicians in managing this fatal condition. The DECAF Score (Dyspnoea, Eosinopenia, Consolidation, Acidemia and Atrial Fibrillation Score) uses indices which are routinely available during the time of hospital admission to prognosticate the patients hospitalized with acute exacerbation of COPD. This study, focuses on calculating the DECAF score at the time of admission and assessing the outcome of the patient on the 5^{th} day(mean duration of hospital stay of COPD patients). The value of DECAF score is compared with the outcome of the patient and hence, used in assessing the prognosis in AECOPD.[10]

OBJECTIVES

1. To study the prognostic value of DECAF score in patients with acute exacerbation of COPD.

REVIEW OF LITERATURE

DISEASE BURDEN

By the end of 2030, COPD ispredicted to become the 3rd major cause of mortality worldwide. Approximately, 3 million people die due to COPD every year contributing to 6% of all mortalities worldwide.[2]

According to WHO estimates, 65 million people are noted to be suffering from moderate to severe COPD worldwide. Respiratory diseases in general, receive little attention and funding in comparison with other major causes of global morbidity and mortality.[11] The deaths from COPD are projected to increase by more than 30% in the next 10 years, and estimates show that COPD will become the third leading cause of death in 2030.[11] The prevalence of the disease is increasing and now it affects men and women almost equally .The Burden of Obstructive Lung Disease (BOLD) study is an international effort to collect population-based estimates of the prevalence of COPD using standardized methods.[12]

The Indian scenario is not different from the world statistics[13]. Malik in 1986 reported that, the prevalence of COPD in North India is 9.4% in rural males and 3.7% in urban males. Prevalence of COPD was found to be 4.9% in rural females and 1.6% in urban females.[14]

However, a subsequent prevalence study in the same region by Jindal et al, found a decreased prevalence of COPD in rural population and a notable increase in urban population.[15] The COPD prevalence in rural and urban males were found to be 6.2% and 4.2% respectively. Another study conducted in South India by Rayet et al,

found a prevalence of 4.08% among males and 2.55% among females, with male to female ratio of 1.6. [16]

A multi-centric study on the epidemiology of COPD, conducted by Jindal et al, in 2006 with a large population of 35295 subjects found that, COPD is 2.65 times more common in smokers compared to non-smokers.The prevalence of COPD was 5.0% and 3.2% among males and females, respectively. Prevalence among cigarette and bidi smokers was 5.9% and 8.2% respectively.[17]

The INSEARCH study, (Indian Study on Epidemiology of Asthma, Respiratory symptoms and Chronic bronchitis, 2006-2009) which was a multicentric study, funded by Indian Council of Medical Research (ICMR), showed wide variations in the prevalence of chronic bronchitis among the different centres, ranging from 0.61% to 13.54%.[18]

DEFINITION OF COPD

Chronic obstructive pulmonary disease (COPD) and its various aspects have been defined by multiple textbooks and guidelines. Harrison's Principles of Internal Medicine states "COPD includes *emphysema*, an anatomically defined condition characterized by destruction and enlargement of the lung alveoli; *chronic bronchitis*, a clinically defined condition with chronic cough and phlegm; and *small airway disease* , a condition in which small bronchioles are narrowed.[19]

Fishman's Pulmonary Diseases and Disorders states that, "COPD is a common name for the chronic airflow obstruction which develops most often as a result of chronic tobacco smoking." The American Thoracic Society (ATS)/European Respiratory Society (ERS) task force defined COPD as "A preventable and treatable disease state

characterized by airflow limitation which is not fully reversible. The airflow limitation is generally progressive and is associated with an abnormal inflammatory response of the lungs to noxious particles and/or gases, mainly caused by cigarette smoking." Although COPD affects the lungs, it also produces significant systemic consequences. COPD is present only in the setting of chronic airflow obstruction; chronic bronchitis without chronic airflow obstruction isn't included within COPD

According to GOLD guidelines COPD is defined as, "COPD is a common, treatable and preventable disease that is characterized by respiratory symptoms that are persistent and airflow limitation that is produced due to airway and/or alveolar abnormalities which is usually due to significant exposure to noxious particles or gases."[2]

RISK FACTORS

Cigarette smoking is a proven and well-studiedrisk factor in COPD.Other factors predisposing to COPD have also been elucidated.

1. GENE

COPD develops as a result of complex gene-environment interaction.[22] Not all smokers with similar smoking history develop COPD. It depends on the genetic susceptibility of the individual. For over 45 years, we have known that genetic variations in the Alpha-1 AntiTrypsin (AAT) gene,Serpin Peptidase Inhibitor Clade A member 1 (SERPINAI) lead to COPD.[23] Several genes have been postulated to be associated with COPD such as Glutathione STransferase, Tensin 1 (TNS1), Advanced Glycosylation End product-specific Receptor (AGER), 5-HydroxyTryptamine

(serotonin) Receptor 4(HTR4), and ThromboSpondin type 1 Domain Containing 4 (THSD4), metalloproteinase 1 and 2.[24,25]

2. AGE AND GENDER

According to Maceicz et al, age is a one of the significant risk factor for the development of COPD.[26]Earlier, most studies showed that prevalence and mortality were considerably more in men as compared to women. But recent studies, especially those done in developed countries show equal gender distribution.[27] Studies done by Silverman EK et al and Foreman M et al, have shown that susceptibility to tobacco smoke is more in females.[28,29]

3. LUNG GROWTH AND DEVELOPMENT

Any process that affects lung growth and development in utero and during childhood results in reduced maximal FEV1(Forced Expiratory Volume in one second) and have risk of getting COPD in later life.[30]A large study and meta-analysis by Lawler DA et al, confirmed a positive association between birth weight and maximum FEV1 attained in adult life.[31,32]

4. EXPOSURE TO PARTICLES

Cigarette smoking is the most common cause of COPD worldwide. Cigarette smokers have, larger annual rates of decline in FEV1, a higher incidence of lung function decline, more respiratory symptoms and higher mortality rates as compared to non-smokers .[33] Fumes, chemicals, organic as well as inorganic dusts are under appreciated occupational risk factors for COPD.[34-36] Cooking and biofuel heating in poorly ventilated houses are indoor pollutants causing COPD.[36-41] Combustion of

fossil fuel for motor vehicles results in reduced lung functions on prolonged exposure.[42]

5. SOCIOECONOMIC STATUS

Lower the socio-economic status, higher are the chances for developing COPD.[43] This may be due to factors like over-crowding, poor nutrition, infection, indoor and outdoor air pollutants.

6. INFECTIONS

History of respiratory infection in childhood has an effect on lung functions and respiratory symptoms in adulthood.[44,45] Infections play a major part in COPD exacerbations but their role in development of COPD is still unclear.[46,47]

PATHOGENESIS

COPD represents the clinical expression of complex alterations in the structure and function of alveolar tissue and small airways. Small airway narrowing and remodelling along withlung parenchymal destruction with subsequent destruction of the alveolar attachments of the airway leading to emphysema are the two main pathological processes that cause progressive airflow limitation in COPD.[48] The major pathogenic processes leading to this pathway are as follows:-

1. INFLAMMATION
2. PROTEASE-ANTI-PROTEASE IMBALANCE
3. OXIDATIVE STRESS

1. INFLAMMATION

Cigarette smoking and other irritants inhaled in the respiratory tract activates surface macrophages in lungs and airway epithelial cells which release multiple chemotactic mediators, particularly chemokines which attract circulating neutrophils,

monocytes and lymphocytes in the lung. This inflammation persists suggesting that there are self-perpetuating mechanisms although they have not been elucidated.[48]

Epithelial Cells: Activation of the airway epithelial cells causes release of Tumour Necrosis Factor alpha (TNF a), Interleukin-1 (IL-1), Interleukin-6(IL-6), Interleukin-8(IL-8) and Transforming growth factor beta (TGF B) and chemokines like CXCL10 and CXCL11 which in turn cause small airway fibrosisand recruitment of T cells leading to further activation of macrophages and release of matrix metalloproteinase (MMP) and other inflammatory mediators while perpetuating the inflammation. Moreover, defence mechanisms of airway epithelial cells such as production of antioxidants, mucous, antiproteases and defensins are impaired. This worsens the effects of inflammation.[48]

Macrophage: Macrophages play a key role in the pathophysiology of COPD and orchestrate the chronic inflammatory response. Macrophages activated by cigarette smoking release mediators of inflammation like TNFA, CXCL1, CXCL8, reactive oxygen species and secrete elastolytic enzymes including MMP2, MMP9, MMP12 causing pathological destruction.[48]

Neutrophils - Neutrophil recruitment to the airways is due to the effect of direct stimulation on production of granulocytes secondary to smoking and CXCL1 and CXCL8 (factors which are chemotactic to neutrophils) released by the activated macrophages. These recruited neutrophils secrete serine proteases such as neutrophil elastase, cathepsin G, MMP8, MMP9 which cause alveolar destruction.[48]

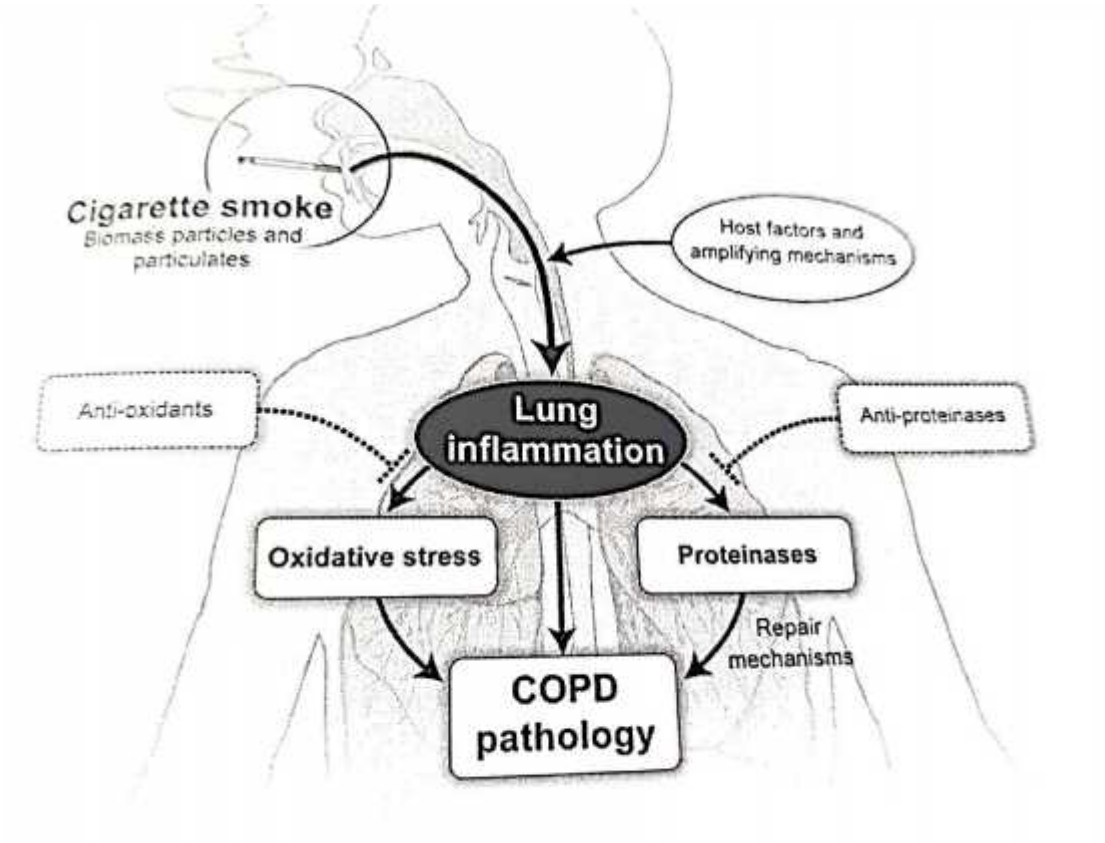


Figure 1: pathogenesis of COPD

2. PROTEASE ANTI-PROTEASE IMBALANCE

Cigarette smoke (and probably other risk factors) along with inflammation can produce oxidative stress that on one hand, primes several inflammatory cells to release proteases such as neutrophil elastase, cathepsin G,MMP 8,MMP 9 and on the other hand, decreases/inactivates several anti-proteases such as SLP1, alpha l anti-trypsin and tissue inhibitors of MMP by oxidation. This protease anti-protease imbalance leads to alveolar destruction.[48]

3. OXIDATIVE STRESS

Oxidative stress is said to occur when reactive oxygen species (ROS) is produced in excess to antioxidant defence mechanisms and result in hazardous effects,

including damage to lipids, proteins and DNA. These in turn increase the inflammation producing further destruction.Moreover, oxidative stress in COPD decreases the antioxidant level due to down regulation of NuclearErythroidFactor 2(Nrf 2), which helps in the increased production of antioxidants such as superoxide dismutase and glutathionine.[48]

PATHOPHYSIOLOGY

According to ATS the symptoms and functional limitations of COPD are " adirect result of lung and airway parenchymal processes which lead to airflow obstruction, increased work of breathing and gas exchange abnormalities. When progressed, these in turn can cause pulmonary hypertension, cor-pulmonale and right heart dysfunction."

1. AIRFLOW LIMITATION AND AIR TRAPPING

Expiratory airflow limitation is the major problem in COPD. Factors causing this are divided into intrinsic and extrinsic factors. Intrinsic factorscause oedema, mucosal thickening, increased secretions of bronchial wall, fibrosis and remodelling.Extrinsic factors produce reduction in elastic tissue support in small airways and reduced dynamic compression of the airways.[21]Other factors like dysfunction of respiratory muscle also limits flow of air in some patients.

Hyperinflation is common in COPD, causing a rise in functional residual capacity (FRC) which is the amount of air that remains in the lungs at the end of tidal exhalation.This leads to trapping of gas and residual volume (RV) rises. This causes increase in the expiratory phase of breathing. It is a major factor causing dyspnoea. The factors that reduce the expiratory time like increased respiratory rate produced by

exercise will increase the FRC.[49]So the lung emptying is ineffective. This is the cause of exercise limitation in patients with COPD and it is termed as dynamic hyperinflation.[49-53]

2. ABNORMALITIES IN GAS EXCHANGE

Gas exchange abnormalities produce hypoxemia as well as hypercapnia. There are many mechanisms for development of gas exchange abnormalities in COPD. There is CO_2 retention secondary to decreased ventilatory drive and increased work of breathing leading to reduced ventilation. Airflow obstruction and hyperinflation exaggerate this process leading to further drop in ventilation.[21]

3. VENTILATORY MUSCLE DYSFUNCTION

There is a mechanical disadvantage in inspiratory muscles due to hyperinflation which limits the generation of force.[54] The contractile apparatus is also affected by nutritional variations, inflammatory response and muscle strength.[55,56] These factors contribute to major exercise limitation.[57,58]

4. PULMONARY HYPERTENSION

It is a major complication of COPD and worsens the prognosis of COPD.[56-63]The causative factor mainly is chronic hypoxia. Endothelial dysfunction, pulmonary capillary destruction are other factors.

COMORBITIES

COPD is primarily a respiratory disease but it has some extrapulmonary manifestations which had not been acknowledged until recently.[64] Different types of studies like epidemiological studies, clinical trials have helped to understand the

importance of comorbidities.[65] The pathophysiology of COPD focuses on systemic inflammation which leads to higher prevalence of metabolic, cardiovascular, skeletal and nutritional disorders that are seen in COPD patients in addition to the coexisting conditions that one would naturally expect.

In the past few years, investigators have analysed that many COPD patients suffer from various other chronic illnesses. These chronic illnesses that are manifested in COPD have been called co-morbidities(i.e coexisting chronic disorders or diseases, regardless of whether the co- morbid condition were or were not directly related to COPD).[66]

The most important study that demonstrated the impact and prognostic role of comorbidities in COPD was undertaken by Antonelli-Incalzi et al. Following the analysis of data from a cohort of 270 patients of AECOPD, the most common comorbid conditions were hypertension (28%), diabetes mellitus (14%) and ischemic heart disease (10%). The median survival was 3.1 years and 228 out of the 270 patients died during the 5 year follow up period.

The prevalence of the various coexisting illnesses varied in the different studies that were conducted. The same could be attributed to differences in methods used to ascertain comorbidities and different population. In spite of these differences there was a routinely high prevalence of coexisting illnesses in patients with COPD. There is evidence that these comorbidities have had a worse impact on COPD patients in terms of quality of life, exacerbation, mortality, social and economic burden.

ASSESSMENT OF SEVERITY OF DISEASE

Goals of COPD assessment is to determine the severity of obstruction of airflow, risk of future exacerbations, hospitaladmissions, health status of patients and death. To attain these goals COPD assessment must consider the following aspects of the disease separately:-

A. The level of patients' symptoms

B. Severity of spirometricabnormalites

C. Risk of exacerbations

D. Presence of co-morbidities

A. SYMPTOM ASSESSMENT

Several validated questionnaires are there for symptom assessment. As per GOLD guidelines[21].MMRC questionnaire (Modified British Medical Research Council questionnaire) and COPD assessment test (CAT) are recommended for the assessment of symptoms. MMRC questionnaire for assessment of breathlessness, relates well to other measures of health status and predict future mortality risk.[68] COPD Assessment Test (CAT) has 8 parameters for impairment of health status and it is scored between 0 to 40.[70] It is at par with St. George questionnaire and is responsive and reliable.[71]

B. SPIROMETRY

Severity of airflow limitation is classified on the basis of post bronchodilator FEVI as per GOLD guidelines.

In patients with $FEV_1/FVC < 0.70$:		
GOLD 1:	Mild	$FEV_1 \geq 80\%$ predicted
GOLD 2:	Moderate	$50\% \leq FEV_1 < 80\%$ predicted
GOLD 3:	Severe	$30\% \leq FEV_1 < 50\%$ predicted
GOLD 4:	Very Severe	$FEV_1 < 30\%$ predicted

Table 1: GOLD staging as per spirometry values

There is only a weak correlation between FEV1, symptoms and impairment of patient's health related quality of life.[72,73] So any category here can have a good or a very poor health status.

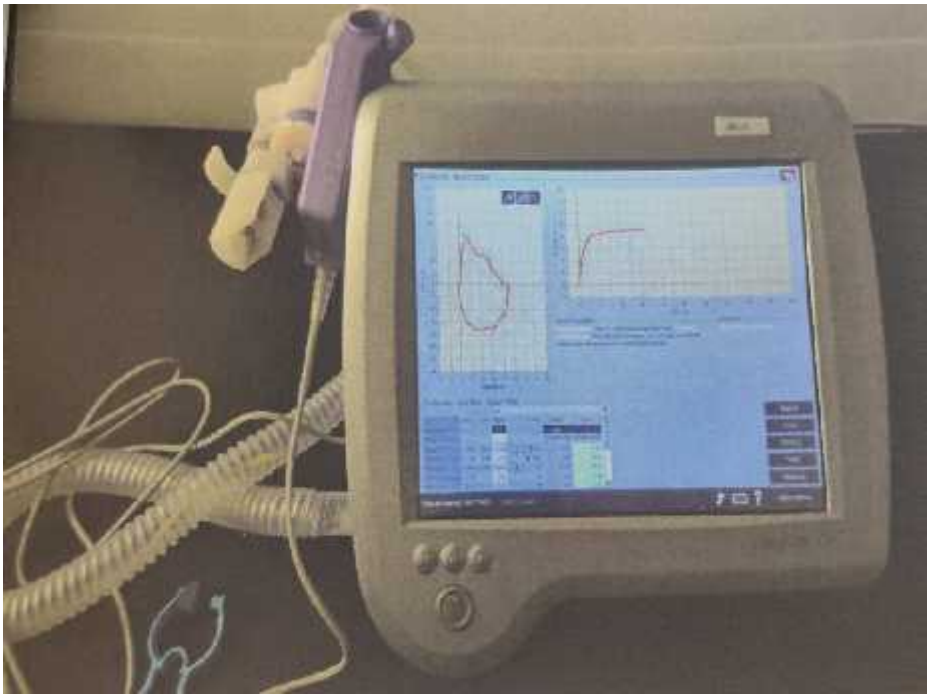


FIGURE 2: Spirometer

C. ASSESSMENT OF RISK OF EXACERBATION

An exacerbation of COPD is defined as "An acute event in the course of the disease characterized by worsening of the patient's respiratory symptoms like increased breathlessness, increased quantity of sputum production and increase in the purulence of sputum, that is beyond normal day to day variation and leads to change in the medication."[74-76] The best predictor of having repeated exacerbations is previous history of treatment for the same events.[67] As the airflow limitation worsens there is more risk for exacerbations, faster loss of FEV1 and death.[21] Instead of an unidirectional approach towards airflow limitation, combining this with assessment of co-morbidities shows the complexity of COPD better.[65,77,78]

ACUTE EXACERBATION OF COPD

"An Exacerbation of Chronic Obstructive Pulmonary Disease (AECOPD) is an event characterized by the worsening of patients respiratory symptoms that is beyond day to day changes and needs a change in medication." It negatively affects the quality of a patient's life taking a long time to recover from, and causing significant mortality. AECOPD accelerates thedeterioration of lung function and has a high socioeconomic cost.

They are classified as:

a. Mild (treatment is with short acting bronchodilators only)
b. Moderate (treatment is with Short Acting Bronchodilators plus antibiotics and/or oral corticosteroids) or
c. Severe (patient requiring hospitalization or the emergency room visit). Severe exacerbations may also be associated with acute respiratory insufficiency.

The main triggering event for exacerbation is viral infections. Aggravating factors may be bacterial infections, dusts, pollution and ambient temperature. Even after one week of exacerbation human rhinovirus which is the causative agent of common cold can be isolated from the respiratory tract of AECOPD patients. Viral infections are seen more in winter, leading to more severe exacerbations and longer hospital stays.[2]

Acute exacerbations of COPD (AECOPD) account for one in eight hospital admissions, and are associated with worsening of symptoms, lung function, health related quality of life and morbidity and mortality risk. In-hospital mortality is reported to be between, 4.4% to 7.7%. The prognosis of the patient cannot be predicted accurately in AECOPD patients who are hospitalised.[80]

PREDICTION OF PROGNOSIS IN AECOPD

A clinically oriented prediction tool, which is developed by cohort study of prospective nature of random admissions, would help in deciding the following factors, namely, a)location of care, b)early escalation of treatment, c)feasibility for early supported hospital discharge and hence reduce morbidity as well as mortality.[81]

In two large national audits from UK, 16% of all admission and 34% patients requiring ventilatory assistance, were in AECOPD.[82,83] Most studies of prognosis in AECOPD requiring hospitalization have not excluded patients with consolidation.[84-88] In stable COPD, the indices for prognostication have been thoroughly studied and investigated and tools predicting morbidity and mortality risk, such as the BODE Score, are well established. However, prognostic research in exacerbations requiring hospitalization and in stable patients is limited.[9]

None of the prognostic indices developed in stable disease have been tested on hospitalized patients with AECOPD. Of the prognostic indices proposed for use in AECOPD requiring hospitalization, most were derived in highly selected population.[89-94]

Presently, for hospitalized patients with AECOPD, the CURB-65 (Confusion, Urea, Respiratory Rate, Blood pressure, Age> 65) is commonly used for assessment of risk and guiding antibiotic therapy. In patients with AECOPD, the use of CURB-65 score is suboptimal as proved by recent studies.[95-97]This again, emphasizes the need for our study with a new tool called the DECAF score.

THE DECAF SCORE

The Dyspnoea, Eosinopenia, Consolidation, Acidaemia and Atrial Fibrillation (DECAF)score was first introduced by Steer et al[81]. The tool is simple to administer at bedside, using indices routinely available on admission. TheDECAF Score is a clinical prediction tool, that can accurately risk stratify all patients with AECOPD. This score has to be assessed and compared with other available scoring systems. Steer et al, in 2012 in the UK, studied a cohort of 920 patients. In the study, the DECAF Score performed drastically better for the prediction of in-hospital mortality.

DECAF score was a better predictor of in- hospital mortality compared to the following scores namely, the APACHE II (Acute Physiology and Chronic Health Evaluation II prognostic index), the CAPS score (COPD and Asthma Physiology Score)and the BAP-65 score(elevated Blood Urea Nitrogen, Altered mental status, Pulse >109/min, Age >65 years), which have all been proposed as useful predictors of outcome in AECOPD.[81,89,90,98,99] DECAF score performed significantly better than APACHE score with AUROC of 0.78, CAPS score with AUROC of 0.68, and BAP-

65 score with AUROC of 0.68. All these findings had a significant 'p' value of <0.001.

Echevarria et al, in 2016 found that, DECAF score was a significantly stronger predictor of in-hospital mortality as well as outcome than CURB-65 for both patients with, as well as without consolidation. The validation of the study done internally and externally found that, the DECAF score was indeed a robust predictor of mortality, in patients hospitalized with AECOPD.[80]

In another study by Nafae et al, the DECAF score showed an excellent discrimination for in-hospital mortality (AUROC 0.83). The DECAF Score performed significantly better for the prediction of in-hospital mortality than the APACHE II prognostic index and the COPD and Asthma Physiology Score (CAPS) which have been proposed as useful predictive instruments in AECOPD. In this study, DECAF was a significantly stronger predictor of in-hospital outcome and mortality, than CURB-65 for a subgroup of patients with radiological consolidation (AUROC - 0.87 vs 0.65, p=0.02).[100]

Zidan et al, studied 100 patients using the DECAF score and found that most of parameters in the DECAF score, dyspnoea (p = 0.001), respiratoryacidosis (p < 0.001) and consolidation (p 0.030), showed a statistically significant value in assessing the mortality and outcome of the patient. Frequency of admissions was found to be the factor most linked to mortality, with a statistically significant value (p < 0.001).[101]

An Indian study conducted by Saranya et al,in a tertiary care centre in South India, included 90 patients of AECOPD. The patients wereprognosticated according to their DECAF score.Out of 90 patients studied, 44 patients had DECAF score

between 0-1, 15 patients had a DECAF score of 2, and 31 patients had a DECAF score between 3-6. In the last group (DECAF 3-6) there was higher mortality, longer hospital stays and increased need for ventilatory support.[102]

There are considerable number of studies to address the value of DECAF score, in predicting the mortality including the one conducted by American Thoracic Society[81].

Following the promising results from the pioneer study, conducted by Steer et al, in 2012,[81] only few studies have been conducted in the past, using DECAF score to predict outcomes in patients hospitalised with AECOPD.

Our study aims to assess the outcome of patients admitted with AECOPD based on DECAF score on admission. The outcome of the patient on 5th day will be assessed and compared to the initial DECAF score on admission of the patient. Thus, DECAF score is used in the study to prognosticate patients presenting with AECOPD.

METHODOLOGY

This study was done in the General medicine department at KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi.

STUDY DESIGN:

Longitudinal study.

STUDY PERIOD:

JANUARY 2019 TO DECEMBER 2019.

SOURCE OF DATA:

The data was collected from all the diagnosed cases of COPD who were admitted in ICU and wards at KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi.

SAMPLE SIZE:

A total of 70 patients were included in the study.

SELECTION CRITERIA:

INCLUSION CRITERIA:

- A clinician made diagnosis of COPD with supporting spirometry or a high probability of the disease (on the basis of clinical history, history of chronic exposure to respiratory irritants, smoking history, physical examination and/or chest x-ray)
- In already known cases of COPD "a change in the patient's baseline dyspnoea, cough, and/or sputum beyond normal day-to-day variations, that is acute in

onset and may warrant a change in regular medication is taken as Acute Exacerbation of COPD" and included in the study.

- Age of the patient more than 18 years.

EXCLUSION CRITERIA:

- Any primary reason for admission other than COPD.
- Patients suffering from bronchial asthma.
- Patients having Metastatic Malignancy.

Patients with a greater number of exacerbations during the period of study were enrolled only once which was at the time of first presentation.

Patient who had exacerbation before the study period were included in our study, only if they had exacerbation at the time of study.

PROCEDURE

All participants, fulfilling inclusion criteria who are admitted in wards or ICU and are willing to participate in the study were enrolled in this study, after taking an informed consent. Participants were subjected to a detailed evaluation of their present symptoms, co morbidities, history of smoking and other habits, any other exposure history, past and present history of exacerbations of COPD.

Routine workup for COPD including complete blood count with eosinophil count was done. ECG was done to look for mainly arrhythmias. Arterial blood gas analysis was done to identify acidosis. Chest X-ray was done to look for consolidation. Dyspnoea of the patient was evaluated by eMRCD(extended Medical Research Council Dyspnoea) grading of 5a and 5b.

MRC Dyspnoea Scores : Traditional (MRCD) & Extended (eMRCD)

Limitation due to breathlessness	MRCD	eMRCD
Breathless only with strenuous exercise	1	
Breathless when hurrying on the level or 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**

Figure 3: Extended MRCD grading

Based on these investigations and dyspnoea scoring, the decaf score of each individual was obtained.

DECAF SCORE is assessed by:

DypnoeaeMRC 5a	1
DypnoeaeMRC 5b	2
Eosinopenia (<0.05 x10^9/l)	1
Consolidation	1
Acidemia(pH <7.3)	1
Atrial fibrillation	1
TOTAL	6

Patients were then scored based on the DECAF score into scores of 0 to 6, with “6” being the worst prognosis and “0” being the best.

After 5 days of admission, the outcome of patient in terms of mortality or continuation of same line of care or stepping down of care, was noted. The initial DECAF score of patients on admission and prognosis of the patients on the 5^{th} day was correlated. Those patients who clinically improved, tapered on antibiotics or other supportive treatment or who were discharged after full recovery were considered to be good prognosis and the rest of the patients were considered as bad prognosis

Following investigations were done:-

1. Complete blood count
2. ECG
3. Chest radiography
4. Arterial blood gas analysis
5. Absolute Eosinophil Count

Ethical clearance

Before the start of the study, the ethical clearance was obtained from the ethical and research committee, Jawaharlal Nehru Medical College, Belagavi.

Informed consent

After explaining the content of the study, written consent (Annexure1) was obtained for participation in those who satisfied the selection criteria.

Statistical analysis

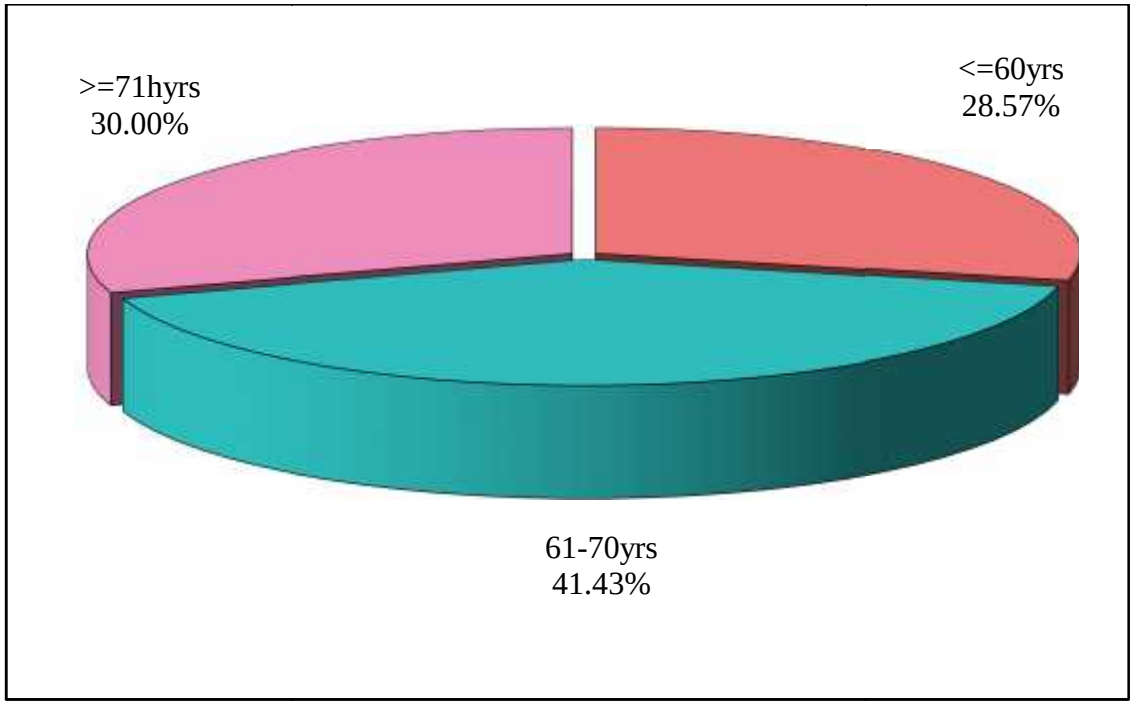
The data obtained was coded and entered into the Microsoft excel spreadsheet. The categorical data were shown as rates, ratios and percentages. Continuous data was expressed as mean ± standard deviation. The categorical data was analysed using Chi Square test of independence. Independent ‘t’ test was used for analysing data which was of continuous type. SPSS software version 20 was used in regression analysis to predict the outcome. A 'p' value of less than 0.05 was considered as statistically significant.

RESULTS

Table2: Age group wise distribution of patients

Age groups	No of patients	% of patients
<=60yrs	20	28.57
61-70yrs	29	41.43
>=71hyrs	21	30.00
Total	70	100.00
Mean age	64.27	
SD age	11.32	

Figure4: Age group wise distribution of patients



From table 2 and figure 4, we observe that 29(41.43%)patients were from age group of 61 to 70 years.

21(30%)patients were in age group of more than 70 years (30%) and the least,

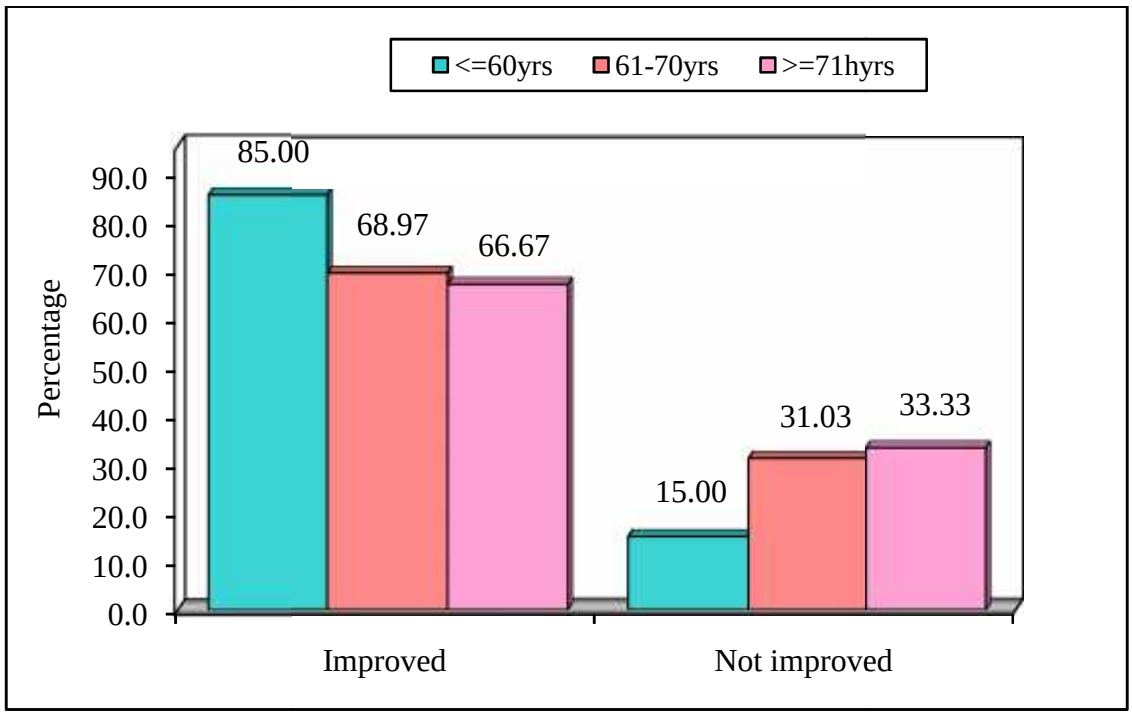
20(28.57%) patients were in age group less than 60 years.

The mean age of the population in our study, is 64.27 years with S.D of 11.32.

Table 3: Comparison of outcome with age groups

Age groups	Improved	%	Not improved	%	Total	%
<=60yrs	17	85.00	3	15.00	20	28.57
61-70yrs	20	68.97	9	31.03	29	41.43
>=71hyrs	14	66.67	7	33.33	21	30.00
Total	51	72.86	19	27.14	70	100.00
Chi-square=2.1200, p=0.3460,NS						

Figure 5: Comparison of outcome with age group



From table3 and figure 5, 17 (85%) out of 20 patients, who were in the age group of less than 60 years, improved.

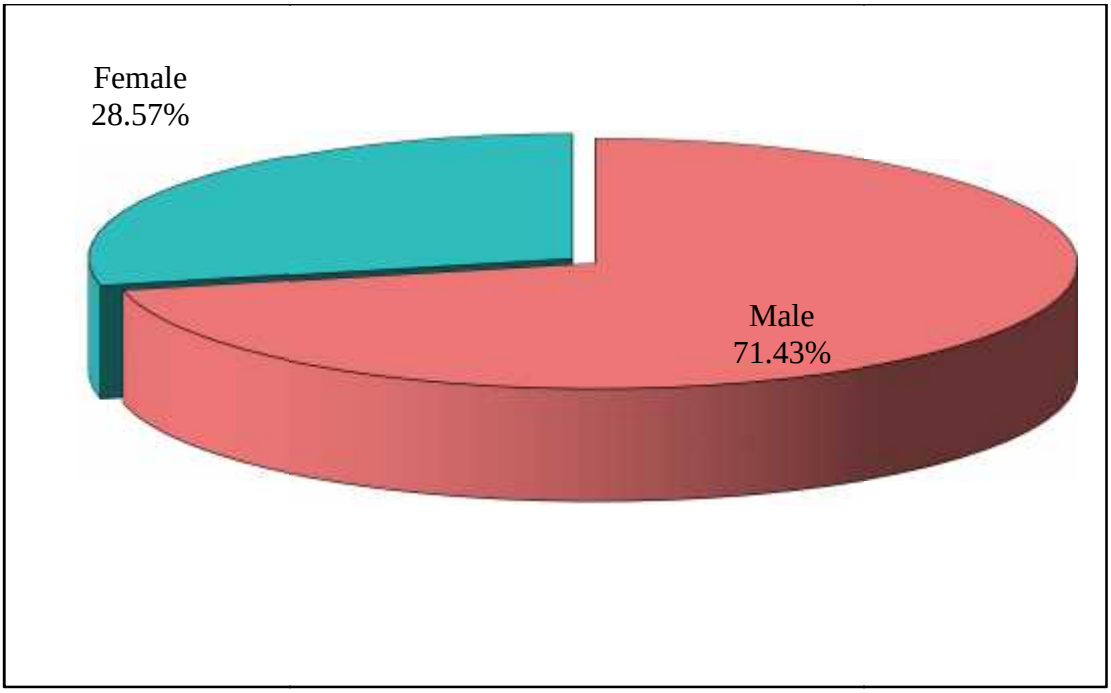
In age group of 61 to 70 years, 20(68.97%) out of 29 patients improved.

In the age group more than 71 years,14 (66.67%)out of21 patientsimproved.

Table4: Gender wise distribution of patients

Gender	No of patients	% of patients
Male	50	71.43
Female	20	28.57
Total	70	100.00

Figure 6: Gender wise distribution of patients.

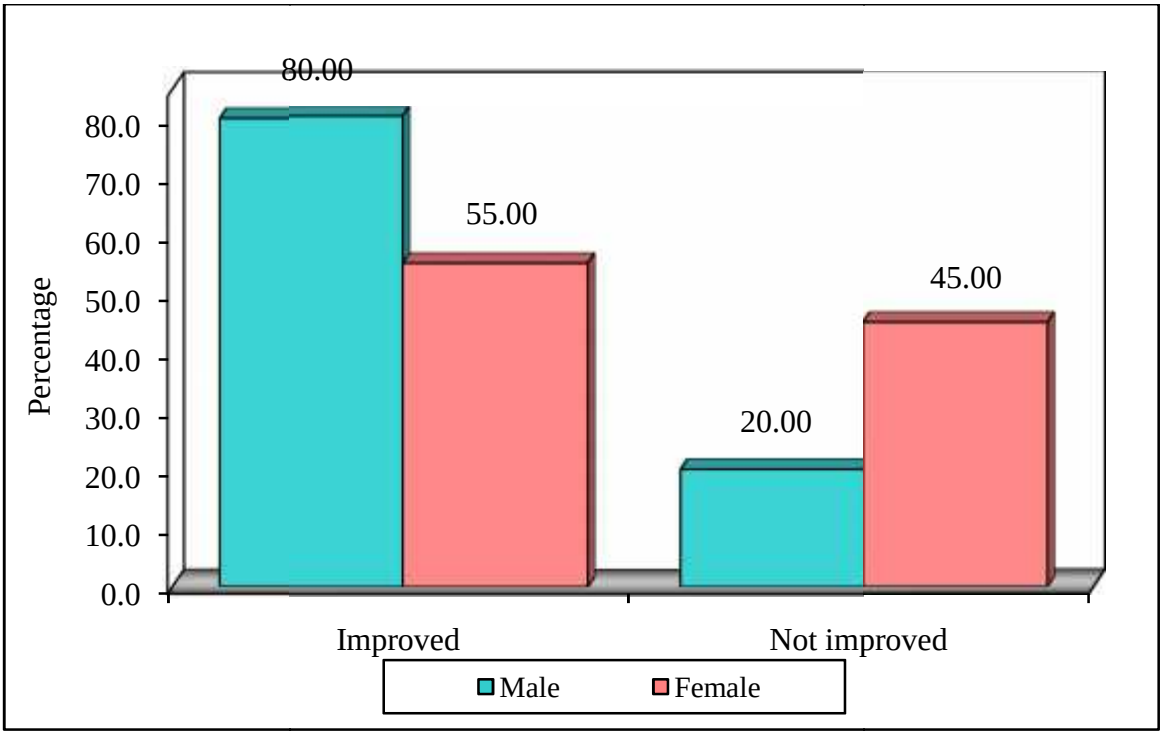


From table 4 and figure 6, 50 (71.4%) patients were male and 20 (28.57%) patients were female.

Table5: Comparison of outcome with gender

Gender	Improved	%	Not improved	%	Total	%
Male	40	80.00	10	20.00	50	71.43
Female	11	55.00	9	45.00	20	28.57
Total	51	72.86	19	27.14	70	100.00
Chi-square=4.5150, p=0.0340,S						

Figure7: Comparison of outcome with gender

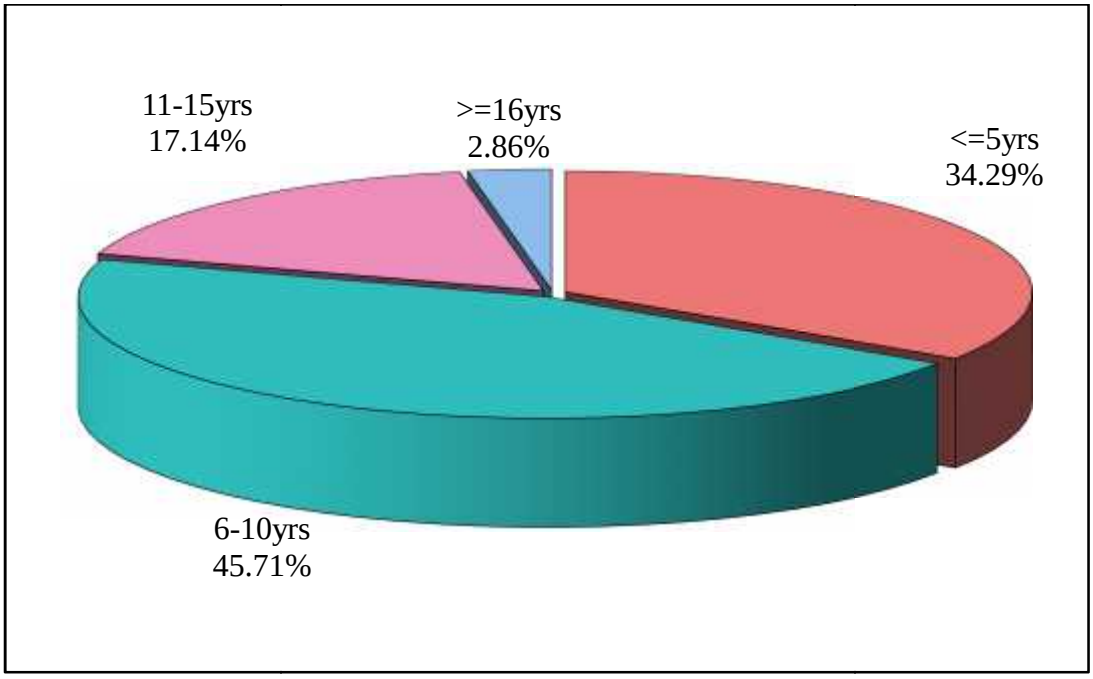


From table 5 and figure 7, 40(80%) out of 50 male patients improved. 11(55%) out of 20 female patients improved in our study.

Table6: Duration of disease wise distribution of patients

Duration of disease	No of patients	% of patients
<=5yrs	24	34.29
6-10yrs	32	45.71
11-15yrs	12	17.14
>=16yrs	2	2.86
Total	70	100.00
Mean	7.84	
SD	4.33	

Figure8: Duration of disease wise distribution of patients



From table 6 and figure 8, mean duration of disease in hospitalised patient is 7.84 years with SD of 4.33.

32 (45.71 %) subjects had COPD for 6 to 10 years, followed by 24 subjects(34.29%) for less than 5 years, 12 subjects(17.14%) for 11 to 15 years and 2 subjects(2.86%) had COPD for more than 16 years.

Table7: Comparison of outcome with Duration of disease

Duration of disease	Improved	%	Not improved	%	Total	%
<=5yrs	21	87.50	3	12.50	24	34.29
6-10yrs	21	65.63	11	34.38	32	45.71
11-15yrs	8	66.67	4	33.33	12	17.14
>=16yrs	1	50.00	1	50.00	2	2.86
Total	51	72.86	19	27.14	70	100.00
Chi-square=4.2090, P=0.2400,NS						

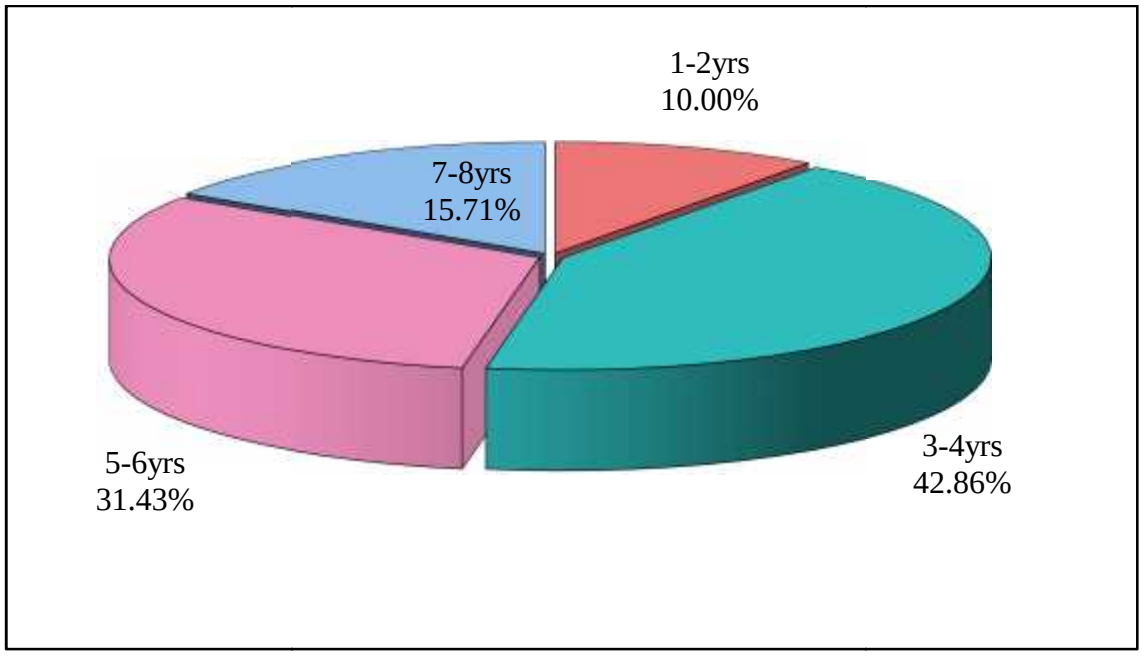
From this table7, 21(87.5%)out of 24 patients with duration of the disease less than 5 years, had good outcome.

The ‘p’ value is 0.2400. Hence, it is statistically not significant.

Table8: Number of exacerbations in past one-year wise distribution of patients.

No of exacerbation in past year	No of patients	% of patients
1-2	7	10.00
3-4	30	42.86
5-6	22	31.43
7-8	11	15.71
Total	70	100.00

Figure9: Number of exacerbations in past one-year wise distribution of patients

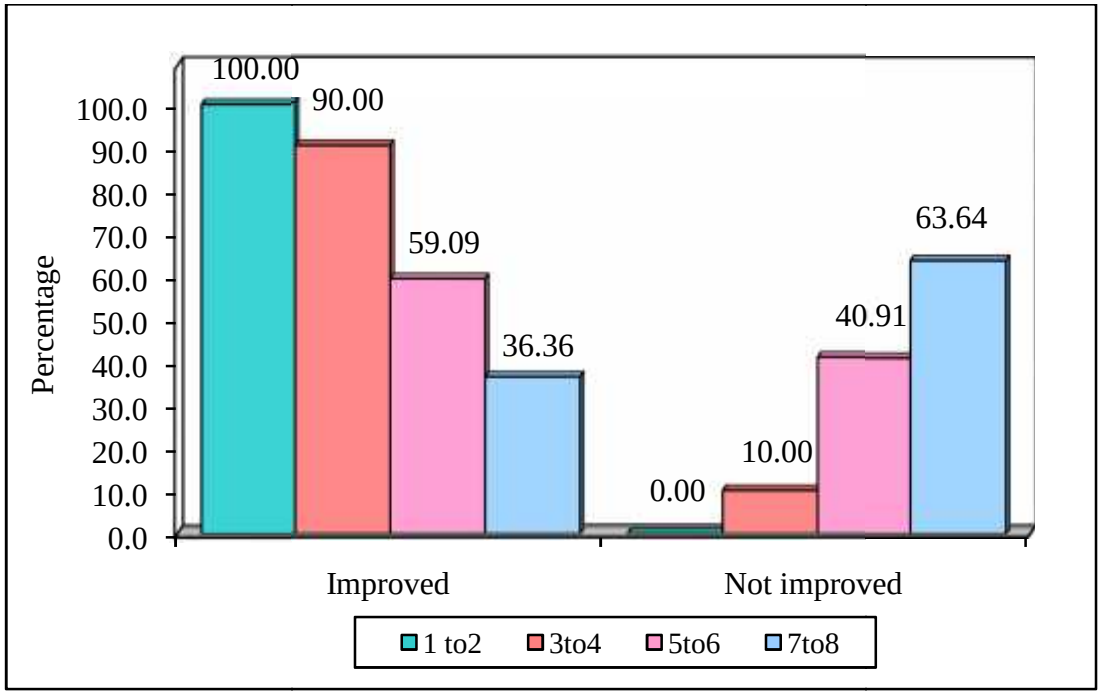


From this table 8 and figure 9, out of 70 patients, 7(10%) patients had 1 to 2 exacerbations in the past one year, 30(42%) patients had 3 to 4 exacerbations, 22 (31.43%) patients had 5 to 6 exacerbations,and 11 (15.71%) patients had 7 to 8 exacerbations in the past one year.

Table9: Comparison of outcome with number of exacerbations in past one year.

No of exacerbation in past year	Improved	%	Not improved	%	Total	%
1-2	7	100.00	0	0.00	7	10.00
3-4	27	90.00	3	10.00	30	42.86
5-6	13	59.09	9	40.91	22	31.43
7-8	4	36.36	7	63.64	11	15.71
Total	51	72.86	19	27.14	70	100.00
Chi-square=16.5822, P=0.0010,S						

Figure10: Comparison of outcome with number of exacerbations in past one year.



From table 9 and figure 10, most of the patients with lesser number of exacerbations had better outcome. Most of the patients had minimum one exacerbation in the past one year.

All patients (100%), with 1 to 2 exacerbations in the past year improved.27(90%) patients with 3 to 4 exacerbations, 13(59.09%) patients with 5 to 6 exacerbations, 4 (36.36%) patients with 7 to 8 exacerbations in the past one year improved.

Table10: Smoking status wise distribution of patients

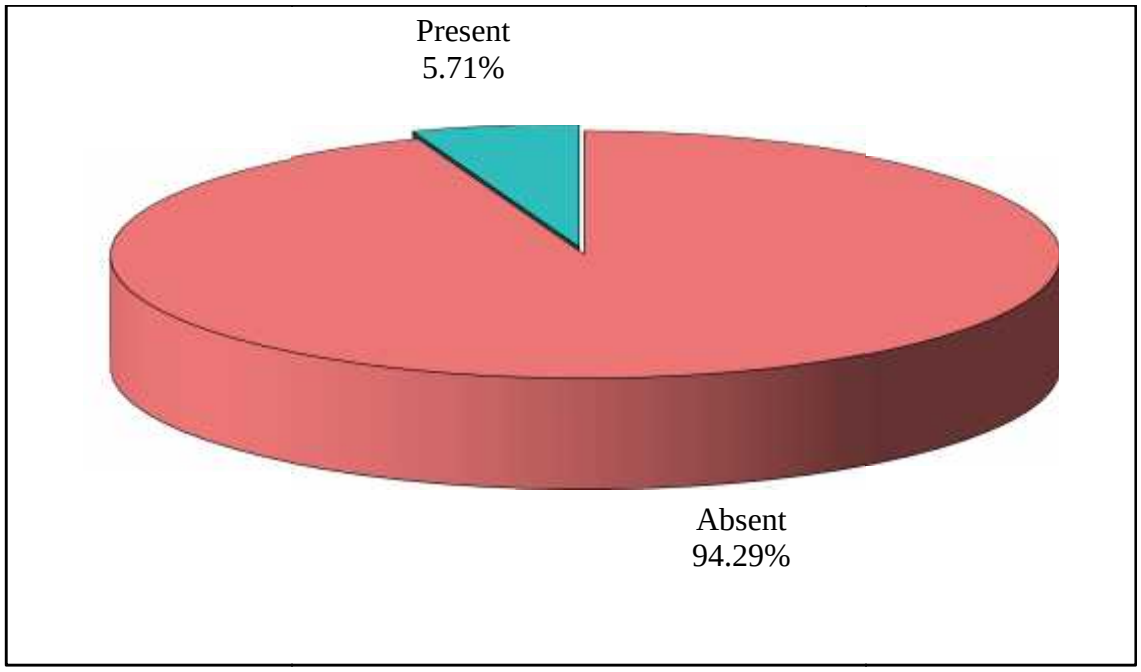
Smoking status	No of patients	% of patients
Absent	23	32.86
Present	47	67.14
Total	70	100.00

From table 10, 47(67.14%) patients smoked and 23(32.8 %) patients did not smoke.

Table11: Other smoke exposure wise distribution of patients

Other smoke exposure wise	No of patients	% of patients
Absent	66	94.29
Present	4	5.71
Total	70	100.00

Figure11: Other smoke exposure wise distribution of patients

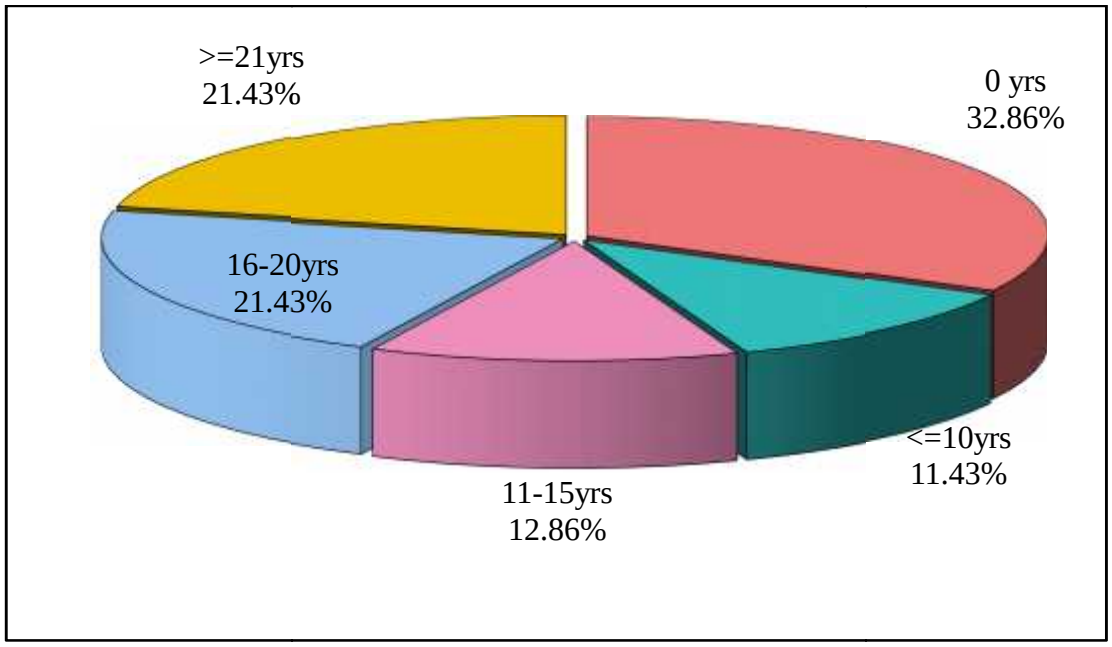


From table 11 and figure 11,4 (5.71%) patients had other smoke exposure, which in our study was biofuel.

Table12: Pack years wise distribution of patients

Pack years	No of patients	% of patients
0 yrs	23	32.86
<=10yrs	8	11.43
11-15yrs	9	12.86
16-20yrs	15	21.43
>=21yrs	15	21.43
Total	70	100.00
Mean	13.23	
SD	12.09	

Figure 12 Pack years wise distribution of patients



From figure 12 and table 12, the mean age of smoking was 13.23 years with a wide standard deviation of 12.09.

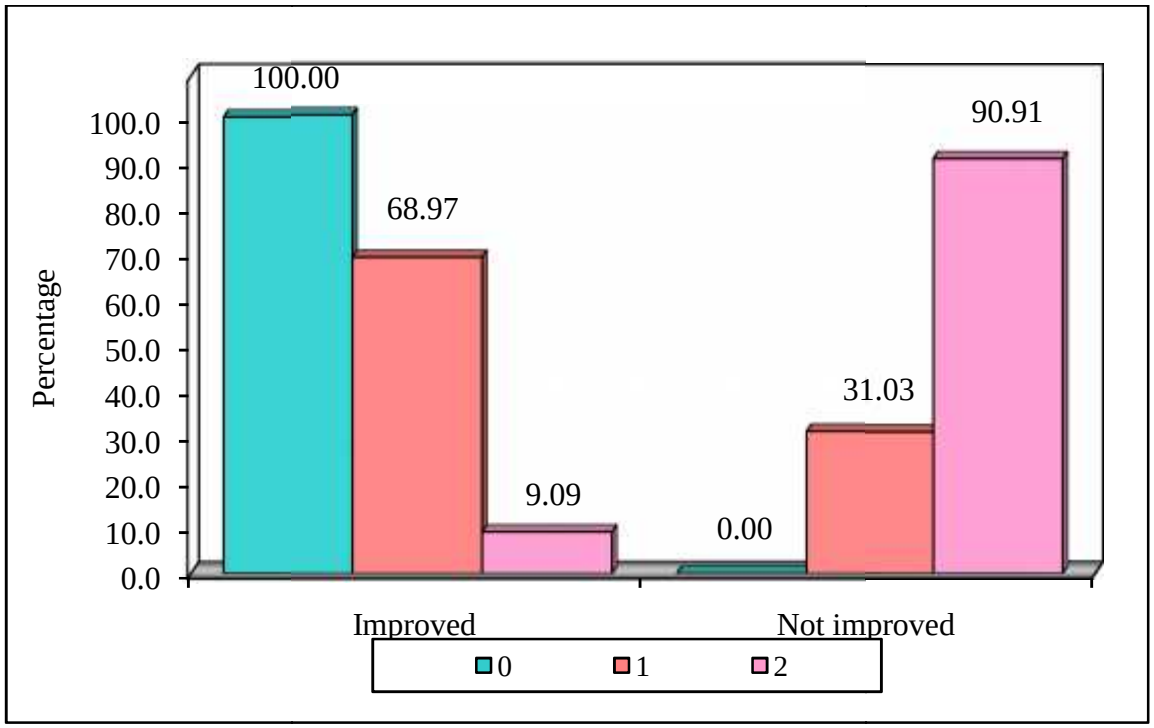
23(32.86%) patients had “0”pack years, 8 (11.43%) patients had less than 10 pack years, 9(12.86%) patients had 11 to 15 pack years.

15(21.43%) patients had pack years of 16 to 20 years and another 15(21.43%) had pack years of more than 20 years.

Table13: Comparison of outcome by dyspnoea

Dyspnoea	Improved	%	Not improved	%	Total	%
0	30	100.00	0	0.00	30	42.86
1	20	68.97	9	31.03	29	41.43
2	1	9.09	10	90.91	11	15.71
Total	51	72.86	19	27.14	70	100.00
Chi-square=34.0160, P=0.0001,S						

Figure13: Comparison of outcome by dyspnoea



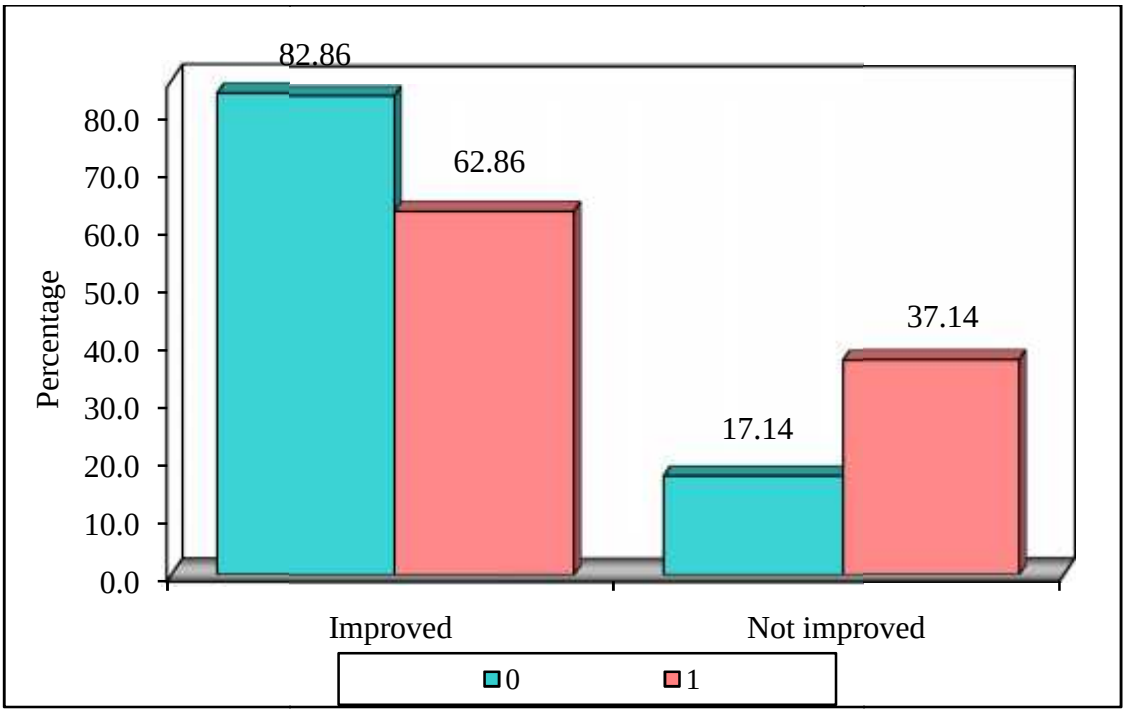
From figure 13 and table 13, 30(42.86%) patients had aeMRCD score of '0' and all (100%) of them improved. 29(41.43%) patients had a eMRCD score of '1' and 20(68.97%) patients improved in this group. 11(15.71%) patients had a eMRCD score of 2 and only 1(9.09%) improved in this group.

With a statistically significant 'p' value of 0.0001, dyspnoea is a significant factor in predicting the outcome of the patient.

Table14: Comparison of outcome by eosinopenia.

Eosinopenia	Improved	%	Not improved	%	Total	%
0	29	82.86	6	17.14	35	50.00
1	22	62.86	13	37.14	35	50.00
Total	51	72.86	19	27.14	70	100.00
Chi-square=3.5400, P=0.0600,NS						

Figure14: Comparison of outcome by eosinopenia.



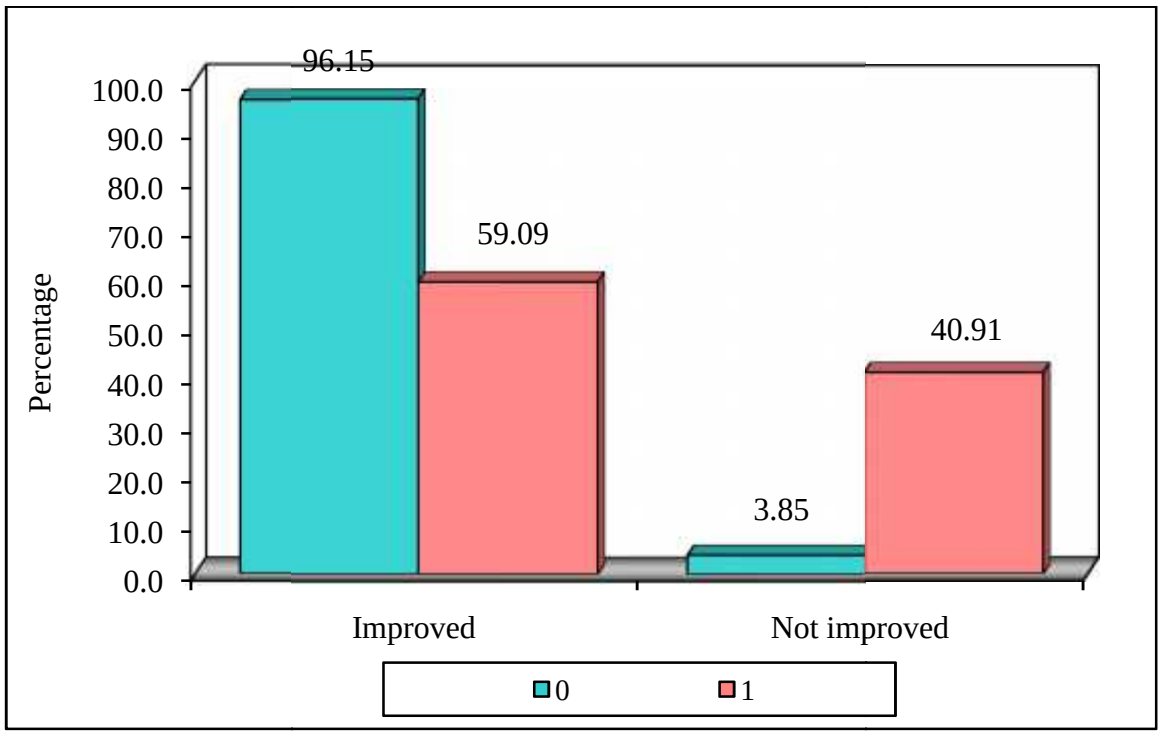
From figure 14 and table 14, 35 (50 %) patients had eosinopenia, out of which 22(62.86 %) improved. Remaining 35(50 %) patients did not have eosinopenia, out of which 29 (82.8 %) patients improved. This data has a 'p' value less than 0.05, hence statistically not significant.

Eosinopenia independently is a poor index for predicting the outcome of the patient in AECOPD.

Table15: Comparison of outcome by consolidation

Consolidation	Improved	%	Not improved	%	Total	%
0	25	96.15	1	3.85	26	37.14
1	26	59.09	18	40.91	44	62.86
Total	51	72.86	19	27.14	70	100.00
Chi-square=11.3520, P=0.0001,S						

Figure15: Comparison of outcome by consolidation



From table 15 and figure 15,. 44(62.86%) patients had consolidation out of which 26(59.09%) patients improved. 26(37.14%) patients had no consolidation,out of which 25(96.15%) patients improved.

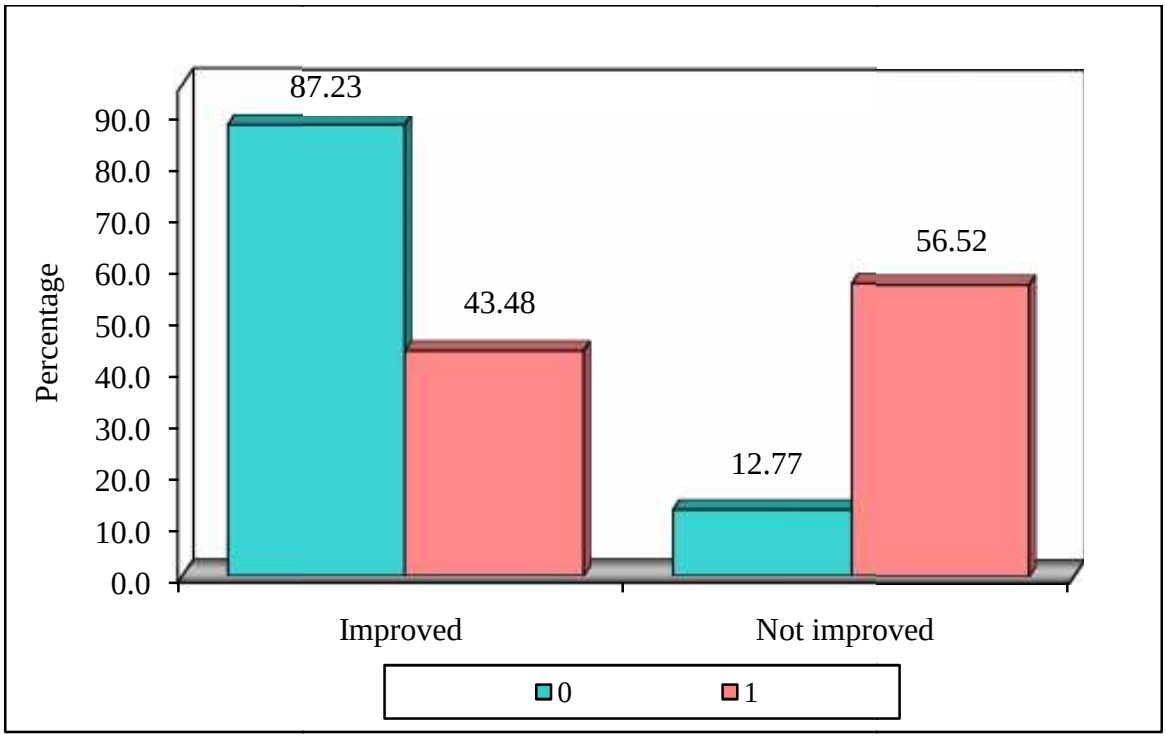
With ‘p’ value of 0.0001, this data is statistically significant.

This shows that consolidation independently is a good predictor of outcome in AECOPD.

Table16: Comparison of outcome by acidemia

Acidemia	Improved	%	Not improved	%	Total	%
0	41	87.23	6	12.77	47	67.14
1	10	43.48	13	56.52	23	32.86
Total	51	72.86	19	27.14	70	100.00
Chi-square=14.9510, P=0.0001,S						

Figure16: Comparison of outcome by acidemia



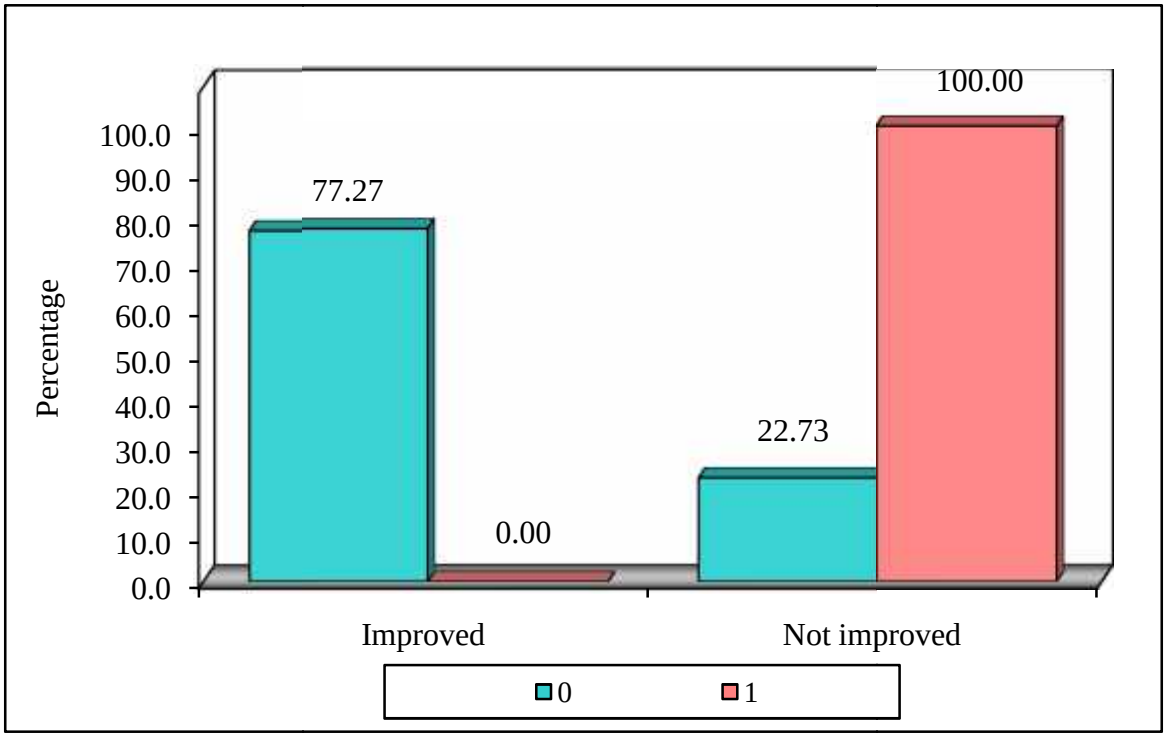
From figure 16 and table 16,23(32.86%) patients had acidemia, out of which 10(43.48%) patients improved. 47(67.14%) patients had no acidemia, out of which 41(87.23) patients improved. These values were statistically significant, with 'p' value of 0.0001.

Thus, acidemia independently is a good predictor of outcome in AECOPD.

Table17: Comparison of outcome by atrial fibrillation

Atrial fibrillation	Improved	%	Not improved	%	Total	%
0	51	77.27	15	22.73	66	94.29
1	0	0.00	4	100.00	4	5.71
Total	51	72.86	19	27.14	70	100.00
Chi-square=11.3880, P=0.0100,S						

Figure17: Comparison of outcome by atrial fibrillation



From table 17 and figure 17, 4 (5.71%) patients had atrial fibrillation and none of the 4 (0%) improved.

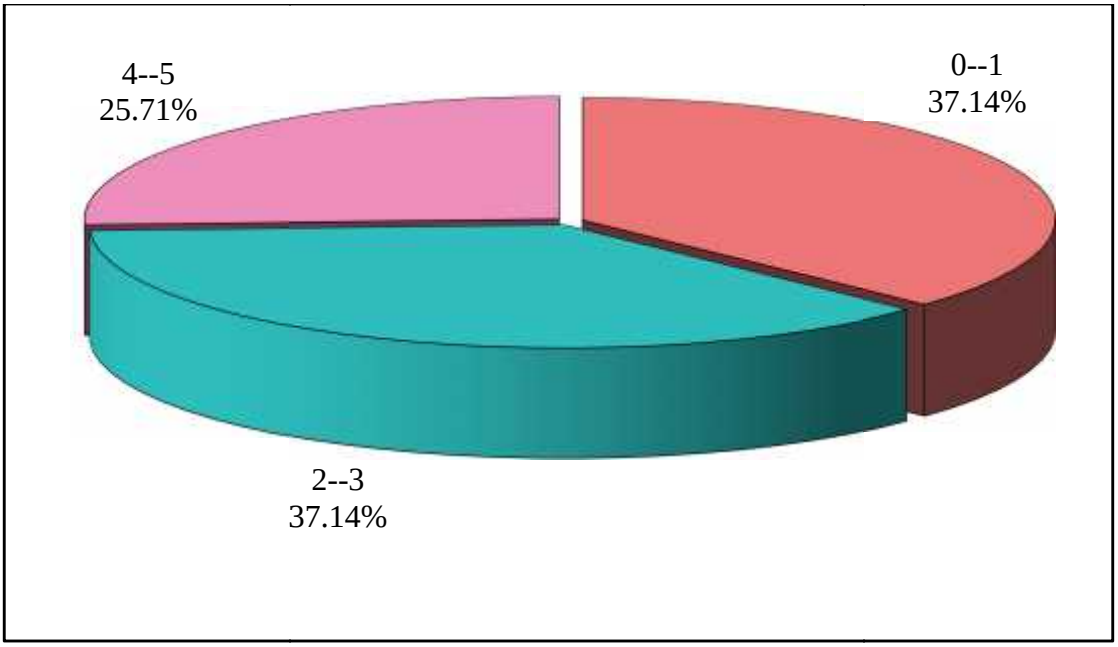
This data is statistically significant, with a ‘p’ value of 0.01.

Thus, atrial fibrillation independently is a good predictor of outcome in AECOPD.

Table18: DECAF score wise distribution of patients

Total DECAF	No of patients	% of patients
0--1	26	37.14
2--3	26	37.14
4--5	18	25.71
Total	70	100.00

Figure18: DECAF score wise distribution of patients

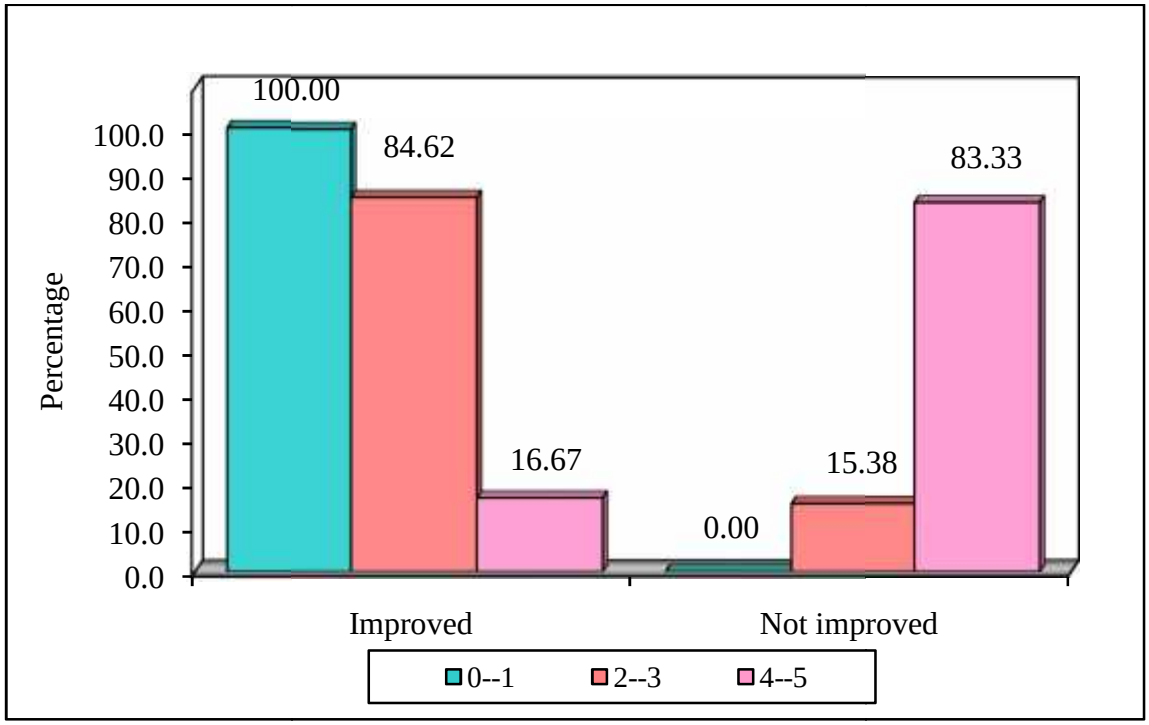


From figure 18 and table 18, 26(37.14%) patients had DECAF score of 0 to 1, 26(37.14%) patients had DECAF score of 2 to 3 and 18(25.71%) patients had a score more than 3.

Table19: Comparison of outcome by total DECAF score

Total DECAF	Improved	%	Not improved	%	Total	%
0-1	26	100.00	0	0.00	26	37.14
2-3	22	84.62	4	15.38	26	37.14
>3	3	16.67	15	83.33	18	25.71
Total	51	72.86	19	27.14	70	100.00
Chi-square=40.243, P=0.0001,S						

Figure19: Comparison of outcome by total DECAF score



From table 19 and figure 19, all 26(100%) patients with score 0 to 1 improved, 22(84.26%) out of 26 patients with the score of 2 to 3 improved and only 3(16.67%) out of 15 patients with score more than 3 improved.

This data is statistically significant with ‘p’ value of 0.0001.

This shows that, DECAF score is a significant predictor of outcome. The lower the DECAF score the better is the outcome of the patient.

Table20: Sensitivity and Specificity of total DECAF

Sensitivity	78.95%
Specificity	94.12%
Positive predictive value	83.33%
Negative predictive value	92.31%
Correctly classified	90.00%

From Table 20, the sensitivity of DECAF score is 78.95 % with a specificity of 94.12 %.

Figure 20: ROC curve

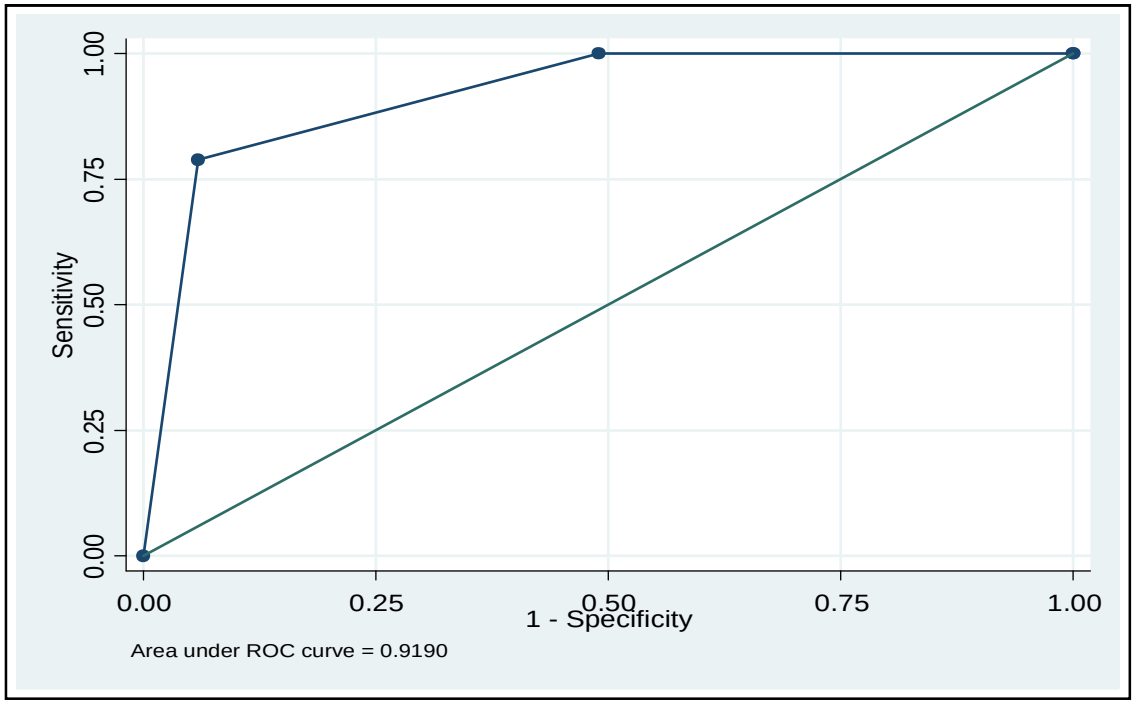
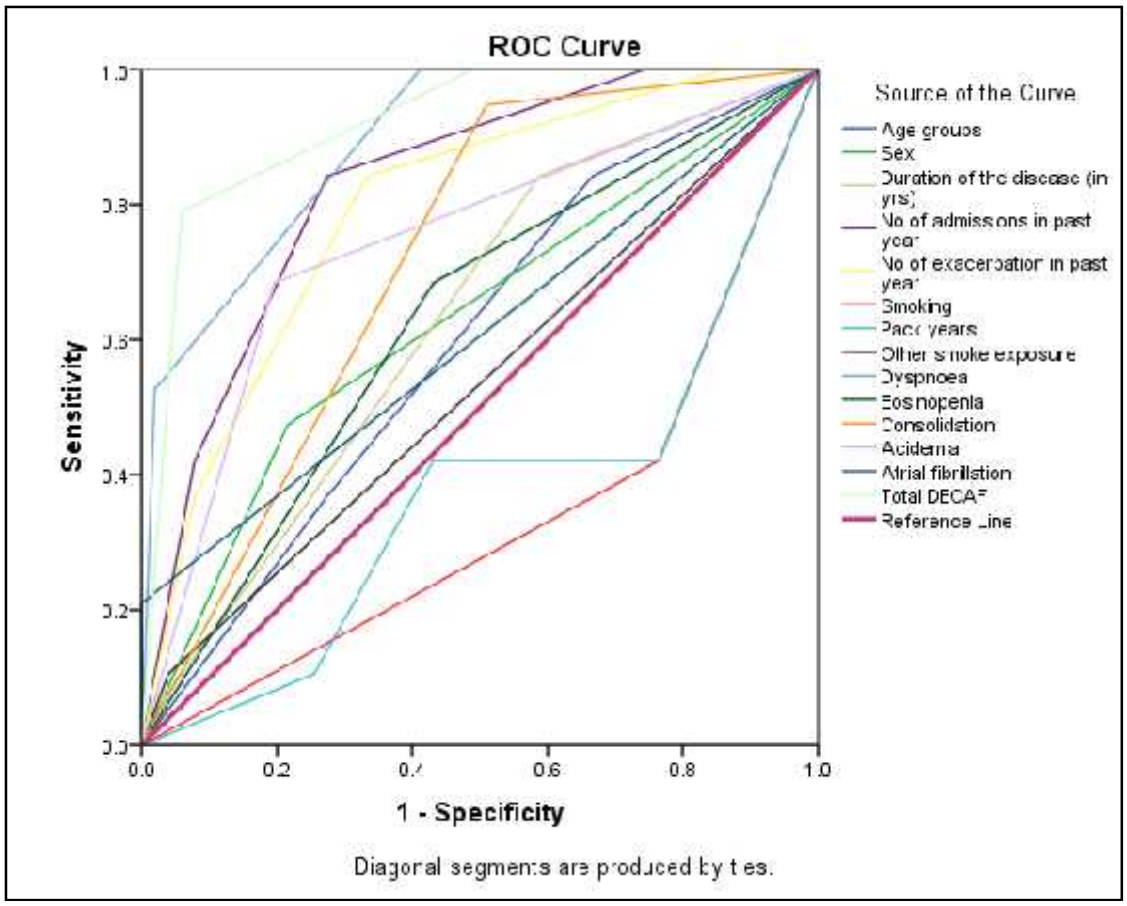


Table21: Area under curve by ROC for multiple variable

Test Result Variable(s)	Area	Std. Error	Asymptotic Sig	Asymptotic 95% Confidence Interval	
				Lower Bound	Upper Bound
Age groups	0.60	0.07	0.2240	0.45	0.74
Sex	0.63	0.08	0.0990	0.48	0.78
Duration of the disease (in yrs)	0.63	0.07	0.0910	0.49	0.77
No of admissions in past year	0.83	0.05	0.0001*	0.73	0.93
No of exacerbation in past year	0.79	0.06	0.0001*	0.68	0.91
Smoking	0.33	0.08	0.0280*	0.18	0.48
Pack years	0.37	0.08	0.0900	0.21	0.52
Other smoke exposure	0.53	0.08	0.6730	0.38	0.69
Dyspnoea	0.89	0.04	0.0001*	0.82	0.97
Eosinopenia	0.63	0.08	0.1060	0.48	0.77
Consolidation	0.72	0.06	0.0050*	0.60	0.84
Acidemia	0.74	0.07	0.0020*	0.61	0.88
Atrial fibrillation	0.61	0.08	0.1780	0.44	0.77
Total DECAF	0.92	0.04	0.0001*	0.85	0.99

*$p<0.05$

Figure21: ROC curve for multiple variable



From table 21 and figure 21, the ROC curve analysis was done for independent variables and for DECAF score in predicting the prognosis of the patient.

Number of exacerbations per year, dyspnoea, consolidation, acidemia, atrial fibrillationand DECAF score in total were good predictors of outcome of AECOPD. Eosinopenia is not a good independent predictor of outcome in our study.

DISCUSSION

COPD represents a significant and growing healthcare concern as a leading cause of morbidity and mortality worldwide. India contributes enormously to COPD burden which is estimated to be amongst the highest in the world.[4]

Every year approximately half a million people die due to this disease in India, which is 4 times more than that of deaths caused by COPD in USA and Europe.[103] The number is steadily rising owing to the increased usage of tobacco smoking and biomass fuel. In patients admitted with acute exacerbation of COPD, a simple prognostic tool that incorporates clinical details and laboratory values which are routinely available on admission and accurately predicts the outcome of the patients is DECAF score.[8]

A total of 70 patients with acute exacerbation of COPD, were enrolled in this study. On admission, complete blood count, chest x-ray, ECG, ABG analysis were recorded for all patients. The severity of dyspnoea at the time of admission was recorded based on the eMRCD scoring for dyspnoea. Based on these details, the DECAF score was assessed for each patient and they were followed up on the 5^{th} day. The outcome of the patient on 5th day was noted. The score with which each patient initially presented and the prognosis of those patients after 5 days were correlated. Those patients who clinically improved, tapered on antibiotics or other supportive treatment or who were discharged after full recovery, were considered to be good prognosis and the rest of the patients were considered as bad prognosis.

AGE DISTRIBUTION and outcome

In our study,we observed that 29(41.43%) patients were in the age group of 61 to 70 years. 21(30%) patients were in the age group of more than 70 years(30%) and the least, 20(28.57%)patients were in age group less than 60 years.

The mean age of the population in our studyis 64.27 years, with S.D of 11.32.The higher age group in our study, was comparable with other studies previously done by Echevarria C et al, Steer J et al, Nafae et al and Yadavilli R et al.[80,81,100,102] However certain studies, like the one done by Ziden et al, had a lower mean age of 46.4 years in patients admitted with AECOPD.[101]

17 (85%) out of 20 patients, who were in the age group of less than 60 years, improved.In the age group of 61 to 70 years, 20(68.97%) out of 29 patients improved. In the age group of more than 71 years, 14 (66.67%)out of 21 patientsimproved. This data shows that, higher age groups are associated with bad outcome. The 'p' value of this is 0.346 and hence, statistically not significant.

Gender distribution and outcomes

There were 50 male(71.43%)and 20 female (28.57%) patients in our study. This data is similar to various prevalence studies which has showed increased prevalence of COPD among men in India. A study done by Nafae et al, had 102 males and 98 females. In Ziden et al's study, there were 58 male patients who represented 58% of total studied cases. 42 female patients were enrolled which represented 42% of total cases. In studies done by Steer J et al, and Scmidt et al, there was a female preponderance of 53.9% and 53% respectively.

In our study 40(80%) out of 50 males improved. 11(55%) out of 20 females improved.

Duration of disease and outcome

In our study, 32 (45.71 %) subjects had COPD for 6 to 10 years, followed by 24 subjects(34.29%) for less than 5 years, 12 subjects(17.14%) for 11 to 15 years and 2 subjects(2.86%) had COPD for more than 16 years.The mean duration of disease in hospitalised patient of AECOPD in our study is 7.84 years with SD of 4.33.

21(87.5%) patients out of 24 patients, with duration of the disease less than 5 years, had good outcome.

The 'p' value is 0.2400. Hence, statistically it was not significant. The duration of illness and its correlation with outcome, has not been studied in any of the studies. If studied in a larger population, it might yield significant results.

Number of exacerbations in past one yearand outcome

Out of 70 patients, 7(10%) patients had 1 to 2 exacerbations in the past one year, 30(42%) patients had 3 to 4 exacerbations, 22 (31.43%) patients had 5 to 6 exacerbations , and 11 (15.71) patients had 7 to 8 exacerbations in the past one year. Hence, most of the study population had at least one exacerbation in the previous one year. This data is similar to all the previous studies, including the pilot study by steer et al.[80,81,100-102] Patients with lesser exacerbation had better outcome.

All patients (100%)with 1 to 2 exacerbations in the past one year improved. 27(90%) patients with 3 to 4 exacerbations, 13(59.09%) patients with 5 to 6 exacerbations and 4 (36.36%) patients with 7 to 8 exacerbations in the past one year improved. This data is statistically significant with 'p' value less than 0.05. Thus, number of exacerbations in the past one year is a good predictor of outcome in hospitalised AECOPD patients.

According to Schmidt et al, the all-cause mortality increases with the number exacerbations in the past one year, which supports the result of our study.[106] Seemungal et al, has observed that exacerbations of COPD lead to a bad quality of life and accounts for the disease burden and mortality.[107]The number of exacerbations in the last one year has an important impact on the outcome of the patients with AECOPD. This is in concordance with the results obtained by Ziden et al, Schmidt et al, where a bigger cohort consisting of 16,647 patients having COPD were involved. [101,106]

Smoking status and other exposure

All the male patients in our study were smokers. All female patients were non-smokers. The social stigmata among Indians where smoking is taboo among females must be the reason for higher prevalence of COPD among males as compared to females.[104]In our study, males with history of smoking were 67.14%. 32.86% of our patients were non-smokers. The mean pack years in our study, was 13.23 years with a standard deviation of 12.06.

4 (5.71%) patients had other smoke exposure, which in our study was biofuel. Jindal SK et al, conducted a population study from India, to estimate COPD burden and its association with smoking. Their study found, COPD to be 2.65 times more common in smokers compared to non-smokers and the prevalence of COPD was 5% and 3.2% among cigarette and bidi smokers respectively.

Dyspnoea and outcome

According to eMRCD grading, eMRCD 5a was given a score of ‘1’ and eMRCD 5b was given a score of ‘2’. Other grades of dyspnoea in eMRCD (no dyspnoea, grade1,grade 2, grade 3 and 4) were considered as score 0.

30(42.86%) patients had a score of '0' and all (100%) of them improved. 29(41.43%) patients had a score of '1',out of which 20(68.97%) patients improved. 11(15.71%) patients had a score of 2,out of which 1(9.09%) improved.

With a statistically significant 'p' value of 0.0001, dyspnoea is a significant factor in predicting the outcome of COPD patients.

Our results are consistent with all studies previously done on DECAF score, including the pilot study done by Steer J et al.[81] Other studies on DECAF scores done by Zidan et al, Nafae et al and Echevarria et al, have reported dyspnoea as a significant predictor of outcome. [80,100,101]

Eosinopenia and outcome.

In our study, 35 (50 %) patients had eosinopenia out of which 22(62.86 %) improved. Remaining 35(50 %)patientsdid not have eosinopenia out of which 29 (82.8 %)patients improved. This data has a 'p' value less than 0.05,hence it was statistically not significant.Eosinopenia independently is a poor index for predicting the outcome of the patient in AECOPD.

This is in concordance with the study done by Ziden et al, where eosinopenia was found to be a poor predictor of mortality and outcome.[101]Saranya et al,conducted a study inSouth India, found that, eosinopenia is poor predictor of outcome in COPD [102] But other studies conducted by Steer et al[81],Nafae et al[81] and Echevarria et al,[80] found eosinopenia to be good predictor of outcome.

Consolidation and outcome

In our study, 44(62.86%) patients had consolidation out of which 26(59.09%) patients improved. 26(37.14%) patients had no consolidation out of which 25(96.15%) patients improved.

With ‘p’ value of 0.0001, this data is statistically significant. This shows that consolidation is a good independent predictor of outcome in AECOPD.

In two UK national audits, consolidation was reported in 16% of all admissions and in 34% of patients requiring ventilator assistance[82,83] Our results reported arein concordance with a similar study done by Steer et al, and also all major studies conducted on DECAF score.[80,81,100,101,102]

ACIDEMIA

23(32.86%) patients has acidemia, out of which 10(43.48%) patients improved. 47(67.14%) patients had no acidemia, out of which 41(87.23) patients improved. These values were statistically significant with ‘p’ value of 0.0001. Thus, acidemia independently is a good predictor of outcome in AECOPD.

All the major studies on DECAF score done by Steer et al, Nafae et al, Echevarria et al, and Ziden et al, have shown acidemia to be significant predictor of outcome. [80,80,100,101]

ATRIAL FIBRILLATION

4(5.71%) patients had atrial fibrillation and none of them improved.This data is statistically significant, with a ‘p’ value of 0.01.Thus, indicating that atrial fibrillation independently is a good predictor of outcome in AECOPD.

Ziden et al, did not find atrial fibrillation to be an independent predictor of outcome of AECOPD.[101] But other studies done by Steer et al, Nafae et al,Echevarria et al, demonstrated atrial fibrillation as an independent predictor of mortality in AECOPD.[80,81,101.]

DECAF SCORE

Out of 70 patients, 26(37.14%) patients had DECAF score of 0 to 1, 26(37.14%) patients had DECAF score of 2 to 3 and 18(25.71%) patients had a score more than 3. All 26(100%) patients with score 0 to 1 improved, 22(84.26%)out of 26 patients with the score of 2 to 3 improved and only 3(16.67%) out of 15 patients with score more than 3 improved.

This data is statistically significant, with 'p' value of 0.0001. This shows that, DECAF score is a significant predictor of outcome. The lower the DECAF score the better is the outcome of the patient with AECOPD.

8 patients in our study died accounting to 11.4 % of the population. All of their scores were more than 4.Higherthe DECAF score, higher is the mortality. This is similar to data reported by J Steer etal.[80]In their study, involving 920 AECOPD patients, a strong association between five categorical variables and in-hospital mortality was established. The same were selected and the DECAF score was devised. Our finding is consistent with other major studies done by Nafae et al,Echevarria et al and Ziden et al, which confirms that DECAF score is a good predictor of prognosis in patients admitted with AECOPD. [81,100,101]

The ROC curve analysis was done for independent variablesand for DECAF score in predicting the prognosis of the patient.

Number of exacerbations per year,dyspnoea, consolidation, acidemia, atrial fibrillation and DECAF score in total were good predictors of outcome of AECOPD. Eosinopenia is not a good independent predictor of outcome in our study.

We observed in our study that, the number of exacerbations in the past one year, dyspnoea, consolidation, acidemia and atrial fibrillation were all good independent predictors of outcome in AECOPD. In our study, DECAF score is a good predictor of outcome in patients hospitalised with AECOPD('p'=0.0001).

LIMITATIONS OF THE STUDY

1. The samples that were selected for our study were from a single centre.
2. The gender distribution in the study was disproportionate, hence cannot establish a relationship which is definite.
3. The DECAF scoring was not compared with the CURB-65 score or other prognostic scoring indices, routinely used in patients having pneumonia, as a cause for the acute exacerbation of COPD.
4. In view of small sample size of 70 patients, further studies are definitely required in order to validate the use of DECAF score,in predicting outcome in AECOPD patients.

STRENGTHS OF THE STUDY

1. This study was a longitudinal study and included all the patients admitted with AECOPD, in a tertiary care hospital for a period of one year.
2. This study utilized a structured analytical tool for the estimation of mortality using the DECAF Score.
3. Very few studies have been conducted internationally using the DECAF score to predict the hospital mortality, among patients with AECOPD. Limited data exists, regarding the role and prognostic value of this robust clinical prediction score.
4. This is one of the very few studies inIndia which evaluated the relationship between DECAF score and the outcome of patients admitted with AECOPD.

CONCLUSION

- The DECAF score is a simple, yet robust predictor of prognosis in patients hospitalised with AECOPD.
- High DECAF score (more than 3) during the time of admission resulted in bad outcome, in patients with AECOPD.
- Patients with lower scores on admission had clinically better outcomes, tapered on antibiotics and other supportive treatment. They were discharged after full recovery, indicating good prognosis.
- The DECAF score can help clinician to predict prognosis by identifying the low risk patients, for treatment at home and for early discharge from hospital. Using the DECAF score we can identify patients with high risk, who need intensivecarefor better outcome.

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ANNEXURE I. ETHICAL CLEARANCE

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 72 **Date: 24/11/2018**

To,

REG NO. Bh0118009
PG student in Medicine,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"PROGNOSTIC VALUE OF DECAF SCORE IN PATIENTS WITH ACUTE EXACERBATION OF COPD – A ONE YEAR CROSS SECTIONAL STUDY IN KLE'S DR. PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELAGAVI"**, is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Arathi Darshan)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

ANNEXURE -II

INFORMED CONSENT

Dear Mr./Mrs./Dr. _____, you are kindly requested to enroll yourself in a research study tilted, **"Prognostic value of DECAF score in patients with acute exacerbation of COPD – A one year cross sectional study in KLE's Dr.Prabbhakar Kore hospital and medical research centre, Belagavi "** being conducted by a post graduate student in M.D. General Medicine and the study will be carried out under the direct supervision and guidance of Dr. , Professor, Department of General Medicine, Jawaharlal Nehru Medical College, Belgaum.

You have been requested to participate in this as you fit into the laid out criteria for a study 'subject'/ participant.

Your participation in study is voluntary. During the study you will be asked some questions and you are supposed to answer to the best of your knowledge. Your decision whether or not to participate in the study will not affect your treatment in any form. If you decide to participate you are free to withdraw at any time.

TITLE OF THE STUDY:

"Prognostic value of DECAF score in patients with acute exacerbation of COPD – A one year cross sectional study in KLE's Dr.Prabbhakar Kore hospital and medical research centre, Belagavi "

PURPOSE OF THE STUDY:

To assess the prognostic value of DECAF score in patients admitted with acute exacerbation of COPD.

PROCEDURES INVOLVED:

If you agree to enroll yourself in my study, you will be interviewed regarding your present, past and family history then you will be clinically examined in detail and investigated accordingly.

Then you will be subjected to a few investigations namely Complete blood counts, ECG ,Chest radiography, Arterial Blood Gas Analysis and Pulmonary Function Tests

RISKS AND BENEFITS:

There are no potential risks involved in this study.

Benefits of taking part in this research:

By taking part in this study, prognostic value of DECAF score in patients admitted with acute exacerbation of COPD can be known

VOLUNTARY PARTICIPATION / WITHDRAWAL FROM THE STUDY:

Taking part in the study is voluntary. You may choose not to enroll yourself in this study and may choose to leave the study anytime in between.

ALTERNATIVES:

Your decision regarding participation in study will not change present or future health care services offered to you at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. You would simply be excluded from the study if you wish to, and all your details shall be kept confidential and you will get the routine line of management.

PRIVACY AND CONFIDENTIALITY:

All data collected or disclosed by you during the course of participation of study, will be kept fully confidential. If however during the course it becomes necessary for the progress of the course to disclose the identity, it would be done so only after your informed & written consent.

The only people to know that you are a research subject are

members of the research team. No information about you will be disclosed to other without your written permission except:

- In emergency to protect your rights AND welfare.
- If required by law.

AUTHORIZATION TO PUBLISH RESULT:

The results of the study may be used to publish an article.When the results of research published or discussed, in a conference, no information will be displayed that would disclose your identity. Any information obtained in connection with this study and that can be identified with you will remain confidential.

FINANCIAL INCENTIVES FOR PARTICIPATION:

No additional costs shall be incurred upon you for the purpose of this study.

It is purely being done with the idea of research and all the cost of study will be borne by the investigator.

COMPENSATION:

In the event that you become injured as a result of taking part in this study, treatment will be offered to you at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum, or you will be given information about where to receive medical care. However, no reimbursement, compensation or free medical care will be given.

QUESTIONS/CONTACT DETAILS:

You shall be free to contact the below mentioned name & addresses anytime during the study period for any clarification or help as you may desire for.

In case of the queries during study or in future you may contact following persons,

J.N.M.C Ethical Committee for Human Research 9448113403	Professor and HEAD OF DEPARTMENT, Dept of General Medicine, JNMC, Belgaum. 9448845883
Investigator, PG in General Medicine, JNMC, Belgaum. 8550851961	

CONSENT FORM

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read this consent form, or it has Been read to me, this consent form and have had all the questions answered

Signature / Left Thumb print of the Participant or legally authorized representative

Participant's name :.....

Signature / Left thumb impression:..... of the participant

Name of the legally authorized representative / guardian :.....

Signature / Left thumb impression :.....

Witness' name :.....

Signature / Left thumb impression :.....

Investigator's name and signature :.....

Date:

Place:

ಕೆ.ಎಲ್.ಇ. ವೈದ್ಯಕೀಯ ಮಹಾವಿದ್ಯಾಲಯ
ಜೆ.ಎನ್. ಮೆಡಿಕಲ್ ಕಾಲೇಜು
ಜನರಲ್ ಮೆಡಿಷಿನ್ ವಿಭಾಗ

ಸಂಶೋಧನಾ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವಿಕೆಗಾಗಿನ ಸಮ್ಮತಿ ಪತ್ರ

ನಾವು ನಿಮ್ಮನ್ನು ಸಂಶೋಧನೆಯಲ್ಲಿ ತೊಡಗಿಸಿಕೊಳ್ಳಲು ಏನಂತಿಸುತ್ತಿದ್ದೇವೆ "ಪ್ರೊಗನೋಸ್ಟಿಕ್ ವ್ಯಾಲು ಆಫ್ DECAF ಸ್ಕೋರ್ ಇನ್ ಪೆಷಂಟ್ ವಿಥ್ ಎಕ್ಷೀವ್ ಎಕ್ಷರಬಿಶನ್ ಆಫ್ COPD" – ಸ್ವಡಿ

ಸ್ನಾತಕೋತ್ತರ ನಡೆಸಿದ, *M.D, F.I.C.P ಪ್ರೊ. ಮೆಡಿಷಿನ ಜನರಲ್ ವಿಭಾಗ, ಜೆ.ಎನ್.ಮೇಡಿಕಲ್ ಕಾಲೇಜು, ಕೆ.ಎಲ್.ಇ. ವಿಶ್ವವಿದ್ಯಾಲಯ, ಬೆಳಗಾವಿ,* ಇವರ ಮಾರ್ಗದರ್ಶದಲ್ಲಿ ನಡೆಸುತ್ತಿದ್ದೇವೆ. ಗೌರವಾನ್ವಿತರೇ ನೀವು ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ಅರ್ಹರಿದ್ದಿರಿ. ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ನಿಮ್ಮ ಪ್ರಸ್ತತ ದೂರು ಬಗ್ಗೆ ಕೆಲವು ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲಾಗುತ್ತದೆ.

ನೀವು ಈ ಅಧ್ಯಯನಕ್ಕಾಗಿ ಅರ್ಹರಾಗಿದ್ದೀರಾ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಮೇಲೆ ಸೂಚಿಸಿರುವ ಜನರಲ್ ಮೆಡಿಷಿನ್ ಮೂಲಕ ಹೇಗೆ ಗುಣವಾಗುವಿರಿ ಎಂಬುದರ ಬಗ್ಗೆ ಅಧ್ಯಯನ ನಡೆಸಲಾಗುವುದು.

ಸಂಶೋಧನೆಯಲ್ಲಿ ನಿಮ್ಮ ಭಾಗವಹಿಸುವಿಕೆ ವೈಯಕ್ತಿಕವಾಗಿದ್ದು ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವುದು ನಿಮ್ಮ ನಿರ್ಧಾರವಾಗಿರುತ್ತದೆ. ಇದರಿಂದ ಜೆ.ಎನ್ ಮೇಡಿಕಲ್ ಕಾಲೇಜಿಗೆ ನಿಮ್ಮಗಿರುವ ಸಂಬಂಧಕ್ಕೆ ಪರಿಣಾಮ ಬೀರುವುದಿಲ್ಲ. ನೀವು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ಸರಿಯಬಹುದು.

ನೀವು ನನ್ನ ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮನ್ನು ತೊಡಗಿಸಿಕೊಳ್ಳಲು ಒಪ್ಪಿದ ನಂತರ ಪ್ರಾಯೋಗಿಕವಾಗಿ ವಿಚಾರಣೆ ಹಾಗೂ ಅದಕ್ಕೆ ತಕ್ಕಂತೆ ತನಿಖೆ, ನಿಮ್ಮ ವರ್ತಮಾನ ಬೂತ ಹಾಗೂ ಕುಟುಂಬದ ಇತಿಹಾಸದ ಬಗ್ಗೆ ಸಂದರ್ಸನ ನಡೆಯಿಸಲಾಗವುದು.

ತಾವುಗಳು ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವಾಗ ಮುನ್ನಚ್ಚರಿಕೆ ಕ್ರಮಗಳನ್ನು ಅನುಸರಿಸಲಾಗುವುದು. ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವುದರಿಂದ ಖಾಯಿಲೆಯ ಸಂಪೂರ್ಣ ಮಾಹಿತಿ ಪಡೆಯಬಹುದು ಇದರಿಂದ ಇದೇ ಖಾಯಿಲೆಯಿಂದ ಬಳಲುತ್ತಿರುವ ಇತರರಿಗೆ ತುಂಬಾ ಸಹಾಯವಾಗುವುದು.

ಈ ಪರೀಕ್ಷೆಯ ವೆಚ್ಚವನ್ನು ಆಸ್ಪತ್ರೆಯ ನಿಯಮದಂತೆ ತಾವೇ ಭರಿಸಬೇಕು. ಆದರೆ ಇದರಲ್ಲಿ ಯಾವುದೇ ಇತರ ವೆಚ್ಚುಗಳು ಇರುವುದಿಲ್ಲ.

ತಾವು ಈ ಪರೀಕ್ಷೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವುದನ್ನು ಸಂಪೂರ್ಣವಾಗಿ ಗೌಪ್ಯವಾಗಿ ಇಡಲಾಗುವುದು.

ತಮ್ಮ ಖಾಯಿಲೆ ಹಾಗೂ ಸಂಶೋಧನೆಯ ವಿವರಗಳನ್ನು ಎಲ್ಲಿಯೂ ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ. ಸಂಶೋಧನೆಯ ವಿವರಗಳನ್ನು ಯಾವುದೇ ಕಾನ್ಫರೇನ್ಸದಲ್ಲಿ ಬಹಿರಂಗಪಡಿಸುವುದಿಲ್ಲ. ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ಆಗುವ ಯಾವುದೇ ವ್ಯತಿರಿಕ್ತ ಪರಿಣಾಮಗಳಿಗೆ ಕೆ.ಎಲ್.ಇ.ಆಸ್ಪತ್ರೆಯ ಮೂಲಕ ಚಿಕಿತ್ಸೆ ನೀಡಲಾಗುವುದು.

ತಮಗೆ ಯಾವುದಾದರು ಸಂಶಯಗಳಿದ್ದಲ್ಲಿ ಅಥವಾ ಹೆಚ್ಚಿನ ಮಾಹಿತಿ ಬೇಕಾಗಿದ್ದಲ್ಲಿ ಈ ಕೆಳಗಿನ ವೈದ್ಯರನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು.

(1) ಸ್ನಾತಕೋತ್ತರ ವಿದ್ಯಾರ್ಥಿ, ಜನರಲ್ ಮೆಡಿಸಿನ ವಿಭಾಗ, ಜೆ.ಎನ್.ಮೇಡಿಕಲ್ ಕಾಲೇಜ, ಬೆಳಗಾವಿ (ಮೋ) 8550851961

(2) [redacted] ಪ್ರೊ. ಮೆಡಿಸಿನ ಜನರಲ್ ವಿಭಾಗ, ಜೆ.ಎನ್.ಮೇಡಿಕಲ್ ಕಾಲೇಜ, ಬೆಳಗಾವಿ

(3) ಡಾ॥ ಎನ್.ಎಸ್.ಮಹಾಂತಶೆಟ್ಟಿ, ಪ್ರಿನ್ಸಿಪಾಲ್ ಜೆ.ಎನ್.ಮೇಡಿಕಲ್ ಕಾಲೇಜ, ಬೆಳಗಾವಿ

(4) ಡಾ॥ ರೂಪಾ ಬೆಲ್ಲದ್, ಚೇರಮನ್, ಎಥಿಕಲ್ ಕಮಿಟಿ ಫಾರ್ ಹುಮ್ಯೂನ್ ಸಬ್ಜೆ ರಿಸರ್ಚ್, ಜೆ.ಎನ್.ಮೇಡಿಕಲ್ ಕಾಲೇಜ, ಬೆಳಗಾವಿ (ಮೋ) 9448113403.

ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ಸ್ವ-ಒಪ್ಪಿಗೆ ಪ್ರಮಾಣ ಪತ್ರ :

ನಾನು ಶ್ರೀ/ಶ್ರೀಮತಿ _____

ಈ ಸಂಶೋಧನೆಯ ಬಗ್ಗೆ ನನ್ನ ಸ್ವಂತ ಭಾಷೆಯಲ್ಲಿ ಸಂಪೂರ್ಣ ವಿವರವಾಗಿ ನನಗೆ ಅರ್ಥವಾಗಿರುತ್ತದೆ. ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ನನಗೆ ಸಂಪೂರ್ಣವಾದ ಒಪ್ಪಿಗೆ ಇರುತ್ತದೆ. ಈ ಸಂಶೋಧನೆಯ ವಿವರಗಳು ಹಾಗೂ ಪರಿಣಾಮಗಳ ಬಗ್ಗೆ ನನಗೆ ಸಂಪೂರ್ಣವಾದ ಮಾಹಿತಿ ಇರುತ್ತದೆ. ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಸ್ವ ಇಚ್ಛೆಯಿಂದ ಪಾಲ್ಗೊಳ್ಳಲು ಬಯಸುತ್ತೇನಂದು ಈ ಮೂಲಕ ದೃಢೀಕರಿಸುತ್ತೇನೆ.

ಭಾಗವಹಿಸುವವರ ಹೆಸರು : _____

ಭಾಗವಹಿಸುವವರ ಸಹಿ : _____

ಭಾಗವಹಿಸುವವರ ಹೆಬ್ಬಟ್ಟಿನ ಗುರುತು : _____

ಸಾಕ್ಷಿದಾರರ ಹೆಸರು : _____

ಸಾಕ್ಷಿದಾರರ ಸಂಬಂಧ : _____

ಸಂಶೋಧಕರ ಹೆಸರು : _____

ಸಂಶೋಧಕರ ಸಹಿ : _____

ಸ್ಥಳ : _____
ದಿನಾಂಕ : _____

के.एल.इ. वैद्यकीय महाविद्यालय
जे.एन. मेडिकल कॉलेज
जनरल मेडिशन विभाग

संशोधन में भाग लेने हेतु सहमति पत्र.

आपको इस संशोधन -" प्रोगनोस्टीक व्यालु आफ "DECAF" स्कोर इन पेशन्ट विथ एक्टीव एक्सरबिशन आफ COPD स्टडि अॅट के. एल. ई. डॉ. प्रभाकर कोरे हॉस्पिटल ऑन्ड एम. आर.सि. बेळगावि स्टडि अॅट" पि.जि. विद्यार्थि". में भाग लेने के लिये निमंत्रित करता हु । ये संशोधन, जे.एन. मेडीकल कालेज बेळगावि. और मार्गदर्शक : M.D, F.I.C.P जनरल मेडिशन विभाग. द्वारा किया जा रहा है ।

मुझे इस संशोधन के बारे मे और इसके फायदे और इसके रिस्क पुरे तरिके से बतादिय गये है ।

मै अपनी मरजी से संशोधन में भाग लेना चाहता हुं और इसके लिये मेरी सहमती है । मैं अपनी मरजी से कभी भी संशोधन में भाग लेने के लिये मना कर सकता हू । मेरे पास संशोधन के बारे मे प्रश्न पुछने के लिये पूरा समय है और इसके लिय मैं कभी भी प्रश्न पुछ सकता हु ।

मेरा साइन / अगूंठा साक्षी है कि मै सहमति पत्र के लिय तैयार हूँ ।

जानखारि केलिये संपर्क करे

१. मुख्य तपासक, पि.जि. विद्यार्थि. जे.एन. वैद्यकीय महाविद्यालय, बेळगावि मो

२. जनरल मेडिशन विभाग जे.एन. वैद्यकीय महाविद्यालय, बेळगावि.-

३. डॉ. एन. एस. माहान्त शेट्टी, मुख्याध्यापक. जे.एन. वैद्यकीय महाविद्यालय, बेळगावि.-

४. डॉ. रूपा बेल्लद चेअरमन, जे.एन. वैद्यकीय महाविद्यालय, संस्थात्मक नीतिशास्त्र समिती -

सहमती पत्र :

मै

इस संशोधन में भाग लेने के लिये पूर्ण सहमति दे रहा हूँ ।

सहभागी का नाम :

हस्ताक्षर :

साक्षीदार :

हस्ताक्षर :

संशोधक :

हस्ताक्षर :

दिनांक :

स्थळ :

के.एल.इ. वैद्यकीय महाविद्यालय
जे.एन. मेडिकल कॉलेज
जनरल मेडिशन विभाग
संशोधन अभ्यास सहभाग संमती

श्री/श्रीमती शीर्षक अभ्यास स्वत: ला नोंदणी करणे विनंती करत आहेत **"प्रोगनोस्टीक व्यालु आफ "DECAF" स्कोर इन पेशन्ट विथ एक्टीव एक्सरबिशन आफ COPD स्टडि ॲट के. एल. ई. डॉ. प्रभाकर कोरे हॉस्पिटल ॲन्ड एम. आर.सि. बेळगावि स्टडि"** पि.जि. **विद्यार्थि.** पदव्युतर द्वारा आयोजित : जनरल मेडिशन विभाग, यांच्या मार्गदर्शक जे.न. वैद्यकीय महाविद्यालय, बेळगावी. करत आहे.

प्रक्रिया सहभाग:

तुम्ही ह्या अध्ययनाला हाजर अहात, व तुमच्या वर उपचार करण्यात येईल.

तुम्ही माझ्या अभ्यासात स्वत: नावनोंदणी सहमत असल्यास, नंतर वैद्यकीय तपशील केली जाईल, आणि त्यानुसार तपास, तुमच्या सध्याच्या गेल्या आणि कुटुंब इतिहास संबंधित मुलाखत घेतली जाईल.

या अभ्यासातील धोके आनि फ़ायदे मला समजावले गेले आहेत.

स्वय्मसेवी सहभाग/काढणे:

सहभाग इछुक आहे. आपण स्वत: ला या अभ्यासात नाही निवडू शकता. रूग्णालयाशी असलेल्या सबंधात काही फरक पडणार नाही. तुम्ही या अभ्यासातून कधीही माघार घेऊ शकता

पर्याय : आपण अभ्यास सहभाग सोडला तरी आपणास व्यवस्थापन नियमा प्रमाणे मिळेल.

गोपनीयता : तुमही दिलेली संशोधनदरम्यानची माहिती ही फक्त संशोधन संघाती लोकांनाच माहित असेल. तुमच्या लेखी परवानगीशिवाय कोणतीच माहिती उघड केली जाणार नाही.

परिणाम छापण्याबाबत : संशोधनच्या परिणामांबाबत चर्चा करताना, तुमची ओळख पटेल अशी माहिती उघड केली जाणार नाही.

नुकसानभरपाई : काहीही आरोग्यिक नुकसान झाल्यास रूग्णालय कोणतीहीं आर्थिक नुकसानभरपाई देण्यास बद्ध नाही.

संशोधनाबाबत काहीही प्रश्न असल्यास संपर्क साधावा :

१. मुख्य तपासक, पि.जि. विद्यार्थि. जे.एन. वैद्यकीय महाविद्यालय, बेळगावि मो नं.

२. जनरल मेडिशन विभाग जे.एन. वैद्यकीय महाविद्यालय, बेळगावि.-

३. डॉ. एन. एस. माहान्त शेट्टी, मुख्याध्यापक. जे.एन. वैद्यकीय महाविद्यालय, बेळगावि.–

४. डॉ. रूपा बेल्लद चेअरमन, जे.एन. वैद्यकीय महाविद्यालय, संस्थात्मक नीतिशास्त्र समिती –

परवानगीचे विधान

मी _____

अभ्यासात भाग घेण्यास तयार आहे. या अभ्यासाबाबत दिलेली सर्व माहिती मला समजली आहे.

भाग घेणार्याचे नाव :

सही अथवा डाव्या हाताचा अँगठा :

साक्षीदाराचे नाव :

साक्षीदाराची सही व तारीख :

स्थळ :

मैं श्री/श्रीमति:_____

ANNEXURE -III

PROFORMA

PROGNOSTIC VALUE OF DECAF SCORE IN PATIENTS WITH ACUTE EXACERBATION OF COPD – A ONE YEAR LONGITUDINAL STUDY IN KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH, BELAGAVI

CASE NO:	I.P. NO.:
NAME:	AGE AND SEX:
ADDRESS:	OCCUPATION:

DATE OF ADMISSION:

PLACE OFADMISSSION: WARD/ ICU STEP IN/ICU STEP DOWN

CHIEF COMPLAINTS **DURATION**

BREATHLESSNESS
COUGH
EXPECTORATION
(MENTION COLOUR AND QUANTITY)
FEVER
WHEEZE

OTHERS IF ANY

MRC GRADE OF DYPNOEA:

PAST HISTORY WITH PAST TREATMENT:

NO OF PREVIOUS ADMISSIONS IN PAST YEAR

NO. OF AECOPD IN PAST YEAR

COMORBIDITIES WITH DURATION

DIABETES MELLITUS
HYPERTENSION
ISCHEMIC HEART DISEASE
CEREBRO VASCULAR ACCIDENT
CHRONIC KIDNEY DISEASE

DURATION OF COPD

TYPE OF VENTILATORY SUPPORT REQUIRED DURING PRESENT ADMISSION: (tick)
INVASIVE MECHANICAL VENTILATION (IMV)
NON INVASIVE VENTILATION (NIV)
NO VENTILATION (NO)

HISTORY OF SMOKING WITH PACK YEARS:

HISTORY OF HOUSEHOLD OR OTHER OCCUPATIONAL EXPOSURE: YES/NO
IF YES SPECIFY:

PHYSICAL EXAMINATION

CYANOSIS- YES/NO

CLUBBING-YES/NO

EDEMA-YES/NO

VITALS:

TEMPERATURE

PULSE

RESPIRATORY RATE

SPO2%

BLOOD PRESSURE

SYSTEMIC EXAMINATION:

R. S. :

C.V.S.:

C.N.S :

P.A. :

ANTIBIOTCS THAT ARE STARTED FOR THE PATIENT AT PRESENTATION:

SUPPORTIVE CARE GIVEN TO THE PATIENT AT PRESENTATION:

SPIROMETRY VALUES:(IF PRESENT)

DECAF SCORE COMPONENTS
SCORE

Dyspnoea with extended MRC grading 5a OR 5b _____

Eosinopenia $<0.05 \times 10^9$ (PRESENT OR ABSENT)

Consolidation on chest X-ray (PRESENT OR ABSENT)

Acidaemia with pH less than 7.3 (PRESENT OR ABSENT)

Atrial fibrillation (PRESENT OR ABSENT)

TOTAL SCORE

AT THE END OF 5 DAYS OF ADMISSION AND CARE

1. Condition of the patient IN TERMS OF SYMPTOMS
2. whether the patient is in

- STEP IN ICU
- STEP DOWN ICU
- EMERGENCY BLOCK
- WARD OF CHOICE
- DISCHARGED
- DIED

3. ANY CHANGE IN ANTIBIOTICS SINCE FIRST DAY OF ADMISSION

BIOCHEMICAL PARAMETERS:

Hemoglobin (G/Dl)

Total Count

Neutrophils

Lymphocytes

Monocytes

Eosinophils

Absolute Eosinophil Count

Platelet count(lakh/ mm^3)

Blood gas analysis

pH

pO2

pCO2

HCO3

O2 saturation

CHEST RADIOGRAPH

CONSOLIDATION PRESENT / ABSENT

ECG

ATRIAL FIBRILLATION: PRESENT/ ABSENT

ANNEXURE -IV

KEY TO MASTER CHART

eMRCD	-	extended Medical Research Council Dyspnoea score
AEC	:	Absolute Eosinophil Count
Exacerbation in POY	:	exacerbations in Past one year.
PYH	:	Pack Year History
NIV	:	Non-Invasive Ventilation
CPAP	:	Continuous Positive Airway Pressure
No	:	No support required
BP	:	Blood pressure
PR	:	Pulse Rate
RR	:	Respiratory Rate
D	:	Dyspnoea
E	:	Eosinopenia
C	:	Consolidation
A	:	Acidemia
F	:	Atrial Fibrillation

SNO	IP NO	AGE	SEX	DURATION OF THE DISEASE	PLACE OF ADMISSION IN THE HOSPITAL	NO. OF AECOPD IN PAST YEAR	SMOKING	PACK YEARS	OTHER SMOKE EXPOSURE	TYPE OF VENTILATION REQUIRED ON ADMISSION	BLOOD PRESSURE	PULSE	TEMPERATURE	RESPIRATORY RATE	SPO2
1	989570	75	MALE	2 YEARS	WARD	2	PRESENT	13.8	ABSENT	NO	110/80	98	AFEBRILE	38	92% IN RA
2	987921	82	FEMALE	10 YEARS	STEP DOWN ICU	4	ABSENT	0	PRESENT	NIV	100/70	96	AFEBRILE	34	80% 4L
3	986465	79	MALE	10YEARS	WARD	4	PRESENT	16.8	ABSENT	NIV	106/70	88	AFEBRILE	27	91% IN RA
4	988447	74	MALE	3YEARS	WARD	3	PRESENT	10	ABSENT	NO	140/90	92	AFEBRILE	34	90% IN RA
5	987404	58	MALE	10YEARS	WARD	7	PRESENT	16	ABSENT	NIV	112/72	89	AFEBRILE	32	88% IN RA
6	972385	51	FEMALE	8YEARS	ICU STEP IN	4	ABSENT	0	ABSENT	NIV	110/60	98	AFEBRILE	36	84% 4LO2
7	988320	65	FEMALE	6YEARS	ICU STEP IN	6	ABSENT	0	ABSENT	NIV	146/92	140	AFEBRILE	43	98% 2LO2
8	987254	75	FEMALE	6YEARS	ICU STEP IN	5	ABSENT	0	ABSENT	NIV	120/80	98	AFEBRILE	34	89%3L02
9	986835	49	FEMALE	8YEARS	STEP DOWN ICU	3	PRESENT	24	PRESENT	NIV	110/70	94	AFEBRILE	33	90% IN RA
10	987443	64	MALE	16YEARS	ICU STEP IN	8	PRESENT	20	ABSENT	NIV	82/56	117	AFEBRILE	36	88%4LO2
11	987922	75	FEMALE	10YEARS	ICU STEP IN	8	ABSENT	0	ABSENT	NIV	90/60	88	AFEBRILE	40	85%IN RA
12	970910	50	FEMALE	4YEARS	ICU STEP IN	5	ABSENT	0	ABSENT	NIV	100/70	98	AFEBRILE	40	92%IN RA
13	987434	55	FEMALE	2 MONTHS	WARD	3	ABSENT	0	ABSENT	NIV	100/70	96	AFEBRILE	98	95%IN RA
14	986756	62	MALE	8 YEARS	ICU STEP IN	2	PRESENT	42	ABSENT	NIV	100/70	102	AFEBRILE	30	96%3LO2
15	1008169	76	FEMALE	12YEARS	ICU STEP IN	4	ABSENT	0	PRESENT	NIV	110/70	98	AFEBRILE	36	82%4LO2
16	1008329	82	MALE	15YEARS	ICU STEP IN	6	PRESENT	15	ABSENT	NIV	100/70	90	AFEBRILE	38	80%4LO2
17	1007112	57	MALE	3YEARS	STEP DOWN ICU	4	PRESENT	12	ABSENT	NIV	130/80	94	AFEBRILE	36	96%4LO2
18	1007309	25	FEMALE	1YEAR	WARD	1	ABSENT	0	ABSENT	NIV	100/70	98	AFEBRILE	30	94%INRA
19	1007616	46	MALE	5YEARS	STEP DOWN ICU	3	ABSENT	0	ABSENT	NIV	90/60	90	AFEBRILE	33	94%INRA
20	1004344	67	FEMALE	12 YEARS	ICU STEP IN	7	ABSENT	0	ABSENT	CPAP	86/70	112	101.4	40	80%INRA
21	1006515	51	FEMALE	7YEARS	ICU STEP IN	3	ABSENT	0	ABSENT	NIV	166/92	100	AFEBRILE	34	88%INRA
22	969121	68	MALE	3YEARS	WARD	3	PRESENT	20	ABSENT	NO	120/70	96	AFEBRILE	30	96%INRA
23	970616	75	MALE	12 YEARS	WARD	3	PRESENT	24	ABSENT	NO	150/100	90	AFEBRILE	32	100%INRA
24	969491	70	MALE	1YEAR	WARD	2	NIL	0	ABSENT	NO	130/80	88	AFEBRILE	32	96%INRA
25	966365	74	MALE	7YEARS	WARD	4	PRESENT	9	ABSENT	NIV	140/80	84	AFEBRILE	31	94%INRA
26	997865	67	MALE	12YEARS	ICU STEP IN	7	PRESENT	17.5	ABSENT	NIV	100/70	106	100	36	80%INRA
27	999424	48	MALE	5YEARS	ICU STEP IN	4	ABSENT	0	ABSENT	IMV	100/70	110	99	40	98%INRA
28	999270	65	MALE	10YEARS	ICU STEP IN	7	ABSENT	0	PRESENT	NIV	100/76	96	AFEBRILE	38	96%6LO2
29	101245	65	FEMALE	10YEARS	TRAUMA	5	ABSENT	0	ABSENT	NIV	110/70	106	101	40	78%INRA
30	995116	72	FEMALE	6YEARS	ICU STEP IN	6	ABSENT	0	ABSENT	CPAP	120/80	106	99	38	78%INRA
31	999412	58	MALE	10YEARS	ICU STEP IN	5	PRESENT	10.5	ABSENT	NIV	96/70	122	AFEBRILE	34	84%INRA
32	971165	40	MALE	2YEARS	WARD	2	PRESENT	10	ABSENT	NO	100/70	82	AFEBRILE	20	90%INRA
33	1006465	46	MALE	15YEARS	STEP DOWN ICU	6	PRESENT	10	ABSENT	NIV	116/74	90	AFEBRILE	36	84%INRA
34	969470	75	FEMALE	10YEARS	WARD	4	ABSENT	0	ABSENT	NO	120/70	90	AFEBRILE	32	80%INRA

35	971758	79	MALE	15YEARS	WARD	6	PRESENT	12.2	ABSENT	NIV	100/70	96	AFEBRILE	28	94%INRA
36	999842	78	MALE	10YEARS	ICU STEP IN	5	PRESENT	30	ABSENT	NIV	160/100	102	AFEBRILE	36	90%4LO2
37	989861	65	MALE	1YEAR	WARD	4	PRESENT	35	ABSENT	NO	110/70	90	AFEBRILE	32	90%INRA
38	1000445	77	MALE	15YEARS	ICU STEP IN	6	PRESENT	20	ABSENT	NIV	100/70	92	AFEBRILE	34	94%4LO2
39	998761	64	MALE	8YEARS	STEP DOWN ICU	3	PRESENT	12.5	ABSENT	NIV	140/90	110	AFEBRILE	38	98%2LO2
40	967529	70	FEMALE	1YEAR	WARD	2	ABSENT	0	ABSENT	NO	160/90	88	AFEBRILE	30	96%INRA
41	923626	43	MALE	12YEARS	ICU STEP IN	8	PRESENT	18	ABSENT	NIV	90/60	106	AFEBRILE	38	94%INRA
42	1007773	65	MALE	8YEARS	ICU STEP IN	7	ABSENT	0	ABSENT	NIV	92/64	88	99	42	86%INRA
43	999804	62	MALE	15YEARS	ICU STEP IN	6	PRESENT	36.8	ABSENT	NIV	100/70	92	100	36	84%INRA
44	1013445	64	MALE	10YEARS	STEP DOWN ICU	7	PRESENT	16	ABSENT	NIV	110/70	88	99.5	32	84%INRA
45	1013871	54	MALE	10YEARS	ICU STEP IN	4	PRESENT	18	ABSENT	NIV	110/70	98	AFEBRILE	30	92%INRA
46	1013844	52	MALE	5YEARS	STEP DOWN ICU	5	PRESENT	15	ABSENT	NIV	100/70	80	AFEBRILE	30	80%INRA
47	1000233	69	MALE	10YEARS	ICU STEP IN	8	PRESENT	16	ABSENT	NIV	100/70	106	100	38	62%INRA
48	999130	50	MALE	4YEARS	STEP DOWN ICU	6	PRESENT	22	ABSENT	NIV	180/90	98	AFEBRILE	36	90%INRA
49	1014192	60	MALE	10YEARS	ICU STEP IN	4	PRESENT	20	ABSENT	NIV	90/60	110	AFEBRILE	36	87%INRA
50	95570	76	FEMALE	6YEARS	ICU STEP IN	6	ABSENT	0	ABSENT	CPAP	100/70	106	AFEBRILE	42	86%ONCPAP
51	964570	75	MALE	9.5YEARS	WARD	5	PRESENT	36	ABSENT	NIV	180/90	94	AFEBRILE	34	80%INRA
52	959959	65	FEMALE	5YEARS	ICU STEP IN	4	ABSENT	0	PRESENT >25Y	CPAP	110/70	111	AFEBRILE	40	96%CPAP
53	963413	65	FEMALE	3YEARS	WARD	3	ABSENT	0	ABSENT	NIV	100/70	101	AFEBRILE	32	96%2LO2
54	961407	65	MALE	6YEARS	STEP DOWN ICU	3	PRESENT	10	ABSENT	NIV	116/76	108	AFEBRILE	36	100%2LO2
55	979418	65	MALE	5YEARS	WARD	2	PRESENT	5	ABSENT	NO	100/70	96	AFEBRILE	30	90%INRA
56	961785	76	MALE	16YEARS	ICU STEP IN	5	PRESENT	40	ABSENT	NIV	160/100	98	AFEBRILE	34	86%6LO2
57	963699	65	MALE	8YEARS	WARD	3	PRESENT	21	PRESENT	NIV	100/70	102	AFEBRILE	32	98%2LO2
58	963911	69	MALE	2YEARS	WARD	4	PRESENT	15.2	ABSENT	NIV	120/80	96	AFEBRILE	32	94%4LO2
59	963986	72	MALE	5YEARS	ICU STEP IN	5	PRESENT	24	ABSENT	NIV	140/90	110	AFEBRILE	38	90%4LO2
60	1013699	78	MALE	5YEARS	ICU STEP IN	5	PRESENT	40	ABSENT	NIV	100/70	120	AFEBRILE	42	80%INRA
61	1014017	72	MALE	10YEARS	STEP DOWN ICU	3	PRESENT	28	ABSENT	CPAP	140/90	116	AFEBRILE	40	82%INRA
62	1010731	51	MALE	7YEARS	WARD	4	PRESENT	15	ABSENT	NIV	100/70	102	AFEBRILE	33	96%INRA
63	999631	45	MALE	4YEARS	ICU STEP IN	4	PRESENT	9.8	ABSENT	NIV	96/70	100	AFEBRILE	34	90%INRA
64	986587	69	MALE	15YEARS	WARD	6	PRESENT	12.2	ABSENT	NIV	100/70	96	AFEBRILE	28	94%INRA
65	942355	65	FEMALE	10YEARS	ICU STEP IN	5	PRESENT	30	ABSENT	NIV	160/100	102	AFEBRILE	36	90%4LO2
66	938310	65	MALE	1YEAR	WARD	4	PRESENT	35	ABSENT	NO	110/70	90	AFEBRILE	32	90%INRA
67	988975	69	MALE	15YEARS	ICU STEP IN	6	PRESENT	20	ABSENT	NIV	100/70	92	AFEBRILE	34	94%4LO2
68	922447	65	MALE	10YEARS	WARD	4	PRESENT	16.8	ABSENT	NIV	106/70	88	AFEBRILE	27	91% IN RA
69	947977	68	MALE	3YEARS	WARD	3	PRESENT	10	ABSENT	NO	140/90	92	AFEBRILE	34	90% IN RA
70	978953	66	MALE	10YEARS	WARD	7	PRESENT	16	ABSENT	NIV	112/72	89	AFEBRILE	32	88% IN RA

RESPIRATORY SYSTEM	EOSINOPHILS	AEC	pH	DYSPNOEA	DYSPNOEA MRC	DYSPNOEA mrc grade5a/5b	EOSINOPENIA	CONSOLIDATION	ACIDEMIA	ATRIAL FIBRILLATION	ON 5 TH DAY PATIENT IS IN	DECAF SCORE	OUTCOME AT THE END OF 5 TH DAY	MORTALITY
RHONCHI PRESENT	4	5000	7.35	PRESENT	GRADE3	0	0	0	0	0	DISCHARGED	0	IMPROVED	NO
RHOCHI PRESENT, LEFT UPPERZONE CREPTS PRESENT	1	1000	7.29	PRESENT	GRADE5A	1	0	1	1	0	STEP DOWN ICU	3	IMPROVED	NO
RHOCHI PRESENT, CREPTS PRESENT IN RIGHT INFRASCPULAR AREA	2	2000	7.39	PRESENT	GRADE3	0	0	1	0	0	WARD	1	IMPROVED	NO
RHONCHI PRESENT	0	0	7.31	PRESENT	GRADE4	0	1	0	0	0	WARD	1	IMPROVED	NO
RHONCHI PRESENT	1	500	7.27	PRESENT	GRADE4	0	1	0	0	0	DISCHARGED	0	IMPROVED	NO
RHONCHI PRESENT	2	200	7.37	PRESENT	GRADE5A	1	0	0	0	0	STEP DOWN ICU	1	IMPROVED	NO
RHONCHI PRESENT,B/L CREPTS PRESENT	0	0	7.39	PRESENT	GRADE5A	1	1	1	0	1	STEP IN ICU	4	WORSENED	YES
RHONCHI PRESENT,B/L CREPTS PRESENT	0	0	7.048	PRESENT	GRADE5A	1	1	1	1	0	DIED	4	DEATH	YES
RHONCHI PRESENT	2	400	7.3	PRESENT	GRADE5A	1	0	1	0	0	WARD	2	IMPROVED	NO
RHOCHI PRESENT,B/LCREPTS PRESENT	0	0	7.3	PRESENT	GRADE5B	2	1	1	0	0	DIED	4	DEATH	YES
RHONCHI PRESENT,LEFT MIDZONE CREPTS PRESENT	0	0	7.25	PRESENT	GRADE5B	2	1	1	1	0	STEP IN ICU	5	WORSENED	NO
WHEEZE PRESENT	0	0	7.27	PRESENT	GRADE5B	2	1	0	1	0	STEP IN ICU	4	WORSENED	NO
WHEEZE PRESENT	39	4400	7.39	PRESENT	GRADE2	0	0	0	0	0	DISCHARGED	0	IMPROVED	NO
WHEEZE PRESENT	0	0	7.21	PRESENT	GRADE5A	1	1	0	1	0	WARD	3	IMPROVED	NO
B/L RHONCHI PRESENT, LEFT UPPERZONE CREPTS PRESENT	4	1	7.21	PRESENT	GRADE5A	1	0	1	1	0	STEP DOWN ICU	3	IMPROVED	NO
B/LRHONCHI PRESENT	2	200	7.24	PRESENT	GRADE4	0	0	1	1	0	WARD	2	IMPROVED	NO
B/L RHONCHI PRESENT	0	0	7.37	PRESENT	GRADE4	0	1	1	0	0	WARD	2	IMPROVED	NO
B/LRHONCHI PRESENT	0	0	7.42	PRESENT	GRADE3	0	1	1	0	0	DISCHARGED	2	IMPROVED	NO
B/LRHONCHI PRESENT,CREPTS PRESENT IN RIGHT INFRAXILLARY AREA	0	0	7.42	PRESENT	GRADE5A	1	1	1	0	0	DISCHARGED	3	IMPROVED	NO
B/LCREPTS PRESENT,B/LRHONCHI PRESENT	1	100	7.39	PRESENT	GRADE5B	2	0	1	0	0	STEP IN ICU	4	WORSENED	YES
CREPTS PRESENT,B/L RHONCHI PRESENT	0	0	7.2	PRESENT	GRADE5A	1	1	1	1	0	STEP DOWN ICU	4	IMPROVED	NO
B/LRHONCHI PRESENT,CREPTS PRESENT	0	0	7.41	PRESENT	GRADE3	0	1	0	0	0	DISCHARGED	1	IMPROVED	NO
B/L RHONCHI PRESENT	2	450	7.34	PRESENT	GRADE4	0	0	0	0	0	DISCHARGED	0	IMPROVED	NO
B/LRHONCHI PRESENT	1	100	7.33	ABSENT	0	0	0	0	0	0	DISCHARGED	0	IMPROVED	NO
B/LWHEEZE PRESENT	6	700	7.37	PRESENT	GRADE5A	1	0	1	0	0	DISCHARGED	0	IMPROVED	NO
B/LCREPTS PRSENT,RHONCHI PRESENT	1	100	7.44	PRESENT	GRADE5A	1	0	1	0	0	WARD	2	IMPROVED	NO
B/L COARSE CREPTS PRESENT,	2	400	7.027	PRESENT	GRADE5B	2	0	1	1	0	STEP IN ICU	4	WORSENED	YES
B/L CREPTS PRESENT	1	100	7.26	PRESENT	GRADE5B	2	0	1	1	0	STEP IN ICU	4	WORSENED	YES
B/LCREPPTS PRESENT	8	700	7.47	PRESENT	GRADE5A	1	0	1	0	0	STEP IN ICU	2	SAME	NO
RIGHT AIR ENTRY DECREASED, B/L CREPTS PRESENT	3	0	7.19	PRESENT	GRADE5B	2	1	1	1	0	STEP IN ICU	5	WORSENED	NO
CREPTS PRESENT,B/L RHONCHI PRESENT	0	0	7.19	PRESENT	GRADE5A	1	1	1	1	0	STEP DOWN ICU	4	IMPROVED	NO
B/LRHONCHI PRESENT	1	125	7.37	PRESENT	GRADE2	0	0	0	0	0	DISCHARGED	0	IMPROVED	NO
B/LRHONCHI PRESENT	**0**	**0**	7.4	PRESENT	GRADE4	0	1	0	0	0	WARD	1	IMPROVED	NO
B/LCREPTS PRESENT,B/LRHONCHI PRESENT	1	150	7.3	PRESENT	GRADE3A	0	0	0	0	0	WARD	0	IMPROVED	NO

B/LRHONCHI	1	100	7.45	PRESENT	GRADE3	0	0	0	0	0	DISCHARGED	0	IMPROVED	NO
LEFT COARSE CREPTS PRESENT,RIGHT CREPTS PRESENT	0	0	7.38	PRESENT	GRADE5A	1	1	1	0	0	STEP IN ICU	3	SAME	NO
B/LRHONCHI PRESENT	0	0	7.37	PRESENT	GRADE4	0	1	0	0	0	DISCHARGED	1	IMPROVED	NO
B/LRHONCHI PRESENT	0	0	7.21	PRESENT	GRADE5B	2	1	1	1	0	STEP IN ICU	5	SAME	NO
B/L CREPTS AND RHONCHI PRESENT	2	200	7.31	PRESENT	GRADE4	0	0	0	0	0	WARD	0	IMPROVED	NO
B/L RHONCHI PRESENT	0	0	7.2	PRESENT	GRADE3	0	1	0	1	0	WARD	2	IMPROVED	NO
B/L RHONCHI PRESENT,B/L CREPTS PRESENT	0	0	7.19	PRESENT	GRADE5A	1	1	1	1	0	STEP IN ICU	4	WORSENED	PU
B/LRHONCHI PRESENT	0	0	7.335	PRESENT	GRADE5B	2	1	1	0	0	STEP IN ICU	4	IMPROVED	NO
B/L RHONCHI PRESENT,CREPTS PRESENT IN B/L INFRASAPULAR AREA	2	200	7.45	PRESENT	GRADE5A	1	0	1	0	0	STEP DOWN ICU	2	IMPROVED	NO
B/L RHONCHI PRESENT,B/L CREPTS PRESENT	0	0	7.28	PRESENT	GRADE5A	1	1	1	1	0	STEP IN ICU	4	WORSENED	NO
B/L RHONCHI PRESENT	0	0	7.27	PRESENT	GRADE5A	1	1	0	1	0	WARD	3	IMPROVED	NO
B/L RHONCHI PRESENT	0	0	7.37	PRESENT	GRADE4	0	1	0	0	0	DISCHARGED	1	IMPROVED	NO
B/LRHONCHI, B/L CREPTS PRESENT	0	0	7.52	PRESENT	GRADE5B	2	1	1	0	1	STEP IN ICU	4	SAME	NO
B/L CREPTS PRESENT,RHONCHI PRESENT	1	100	7.53	PRESENT	GRADE5A	1	0	1	0	0	WARD	2	IMPROVED	NO
B/L CREPTS PRESENT, RHONCHI PRESENT	0	0	7.32	PRESENT	GRADE5A	1	1	1	0	0	STEP DOWN ICU	3	IMPROVED	NO
B/L RHONCHI PRESENT,B/L CREPTS PRESENT	7	700	7.12	PRESENT	GRADE5A	1	0	1	1	0	ICU STEP IN	3	SAME	NO
B/L RHONCHI PRESENT	2	100	7.32	PRESENT	GRADE4	0	0	0	0	0	DISCHARGED	0	IMPROVED	NO
B/L CREPTS PRESENT, RHONCHI PRESENT	0	0	7.22	PRESENT	GRADE5A	1	1	1	1	1	ICU STEP IN	5	SAME	NO
B/L RHONCHI PRESENT	5	400	7.47	PRESENT	GRADE3	0	0	1	0	0	DISCHARGED	1	IMPROVED	NO
B/L RHONCHI PRESENT, CREPTS PRESENT	0	0	7.4	PRESENT	GRADE5A	1	1	1	0	0	WARD	3	IMPROVED	NO
B/L RHONCHI PESENT	0	0	7.38	PRESENT	GRADE3	0	1	0	0	0	DISCHARGED	1	IMPROVED	NO
NCHI PRESENT, B/L CREPTS PRESENT MORE IN RIGHT INFRA AXXILLAR	4	400	7.41	PRESENT	GRADE5A	1	0	1	1	0	STEP DOWN ICU	3	IMPROVED	NO
B/L RHONCHI PRESENT	1	100	7.43	PRESENT	GRADE3	0	0	0	0	0	DISCHARGED	1	IMPROVED	NO
B/L RHONCHI PRESENT	0	0	7.4	PRESENT	GRADE4	0	1	1	0	0	WARD OF CHOICE	2	IMPROVED	NO
B/LCREPTS PRESENT,B/LRHONCHI PRESENT	1	100	7.26	PRESENT	GRADE5A	1	0	1	1	0	WARD OF CHOICE	3	IMPROVED	NO
B/LCREPTS PRESENT,B/LRHONCHI PRESENT	0	0	7.41	PRESENT	GRADE5A	1	1	1	0	0	STEP IN ICU	3	IMPROVED	NO
B/L RHONCHI PRESENT	0	0	7.19	PRESENT	GRADE5A	1	1	1	1	1	ICU STEP IN	5	WORSENED	YES
B/L RHONCHI	1	200	7.44	PRESENT	GRADE4	0	0	0	0	0	DISCHARGED	0	IMPROVED	NO
B/L RHONCHI PRESENT,RHONCHI PRESENT	3	200	7.37	PRESENT	GRADE5A	1	0	1	0	0	STEP DOWN ICU	2	IMPROVED	NO
B/LRHONCHI	0	0	7.45	PRESENT	GRADE3	0	1	0	0	0	DISCHARGED	0	IMPROVED	NO
LEFT COARSE CREPTS PRESENT,RIGHT CREPTS PRESENT	1	100	7.47	PRESENT	GRADE5A	1	0	1	0	0	STEP IN ICU	2	IMPROVED	NO
B/LRHONCHI PRESENT	1	100	7.5	PRESENT	GRADE4	0	0	1	0	0	DISCHARGED	1	IMPROVED	NO
B/LRHONCHI PRESENT	5	600	7.21	PRESENT	GRADE5B	2	0	1	1	0	STEP IN ICU	4	WORSENED	NO
RHOCHI PRESENT, CREPTS PRESENT IN RIGHT INFRASCPULAR AREA	1	125	7.39	PRESENT	GRADE3	0	0	1	0	0	WARD	1	IMPROVED	NO
RHONCHI PRESENT	3	225	7.31	PRESENT	GRADE4	0	0	0	0	0	DISCHARGED	0	IMPROVED	NO
RHONCHI PRESENT	3	500	7.27	PRESENT	GRADE4	0	1	0	0	0	DISCHARGED	0	IMPROVED	NO