**A DISSERTATION**

**ON**

# "STUDY ON CARDIAC TROPONIN I LEVELS IN ACUTE COPD EXACERBATION VERSUS STABLE COPD PATIENTS"

Submitted to

**THE TAMILNADU DR. M.G.R UNIVERSITY**
**CHENNAI**

In partial fulfilment of the regulations
for the award of

**M.D DEGREE IN GENERAL MEDICINE**
BRANCH I


**GOVERNMENT MOHAN KUMARAMANGALAM**

**MEDICAL COLLEGE AND HOSPITAL , SALEM.**

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***"To acknowledge is to know gratitude and gratitude is a valuable duty which ought to be paid to valuable people in a lifetime"***

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Ethical committee Meeting held on 30.07.2014 at 12 noon in the Dean's Chamber, Government Mohan kumaramangalam Medical College Hospital, Salem 01, The following members attended the meeting.

**MEMBERS.**

1. Dr.N. MOHAN MS., FICS., FAIS., FMMC.,Dean, Member secretary ECIRB
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8. Mr.S.SUBRAMANIAM, B.Sc.,C.A., Chartered accountant, External Lay person.

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| 6. | **Dr. C.Nirimala Devi** Final year MD (GM) Post graduate student, GMKMCH.salem-30 | **STUDY ON CARDIAC TROPONIN I LEVELS IN ACUTE COPD EXACERBATION VS STABLE COPD PATIENTS** | **Dr S.Ramasamy, MD., Professor, Department of General Medicine** | **Approved** |

The Ethical Committee examined the studies in detail and is pleased to accord Ethical Committee approval for the above Post Graduate of this college to carry out the studies with the following conditions.

1. She should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution or government.
2. She should inform the institutional Ethical committee in case of any change of study or procedure site.
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# LIST OF ABBREVIATIONS USED

| | | |
|---|---|---|
| **AF** | **:** | Atrial Fibrillation |
| **A-a D O2** | : | Alveolar Arterial Diffusion Of Oxygen |
| **APC** | **:** | Atrial Premature Contraction |
| **BLVR** | **:** | Bronchoscopy Lung Volume Reduction |
| **BODE** | **:** | Body Mass Index, Obstruction, Dyspnoea, Exercise capacity |
| **CAT** | **:** | COPD Assessment Test |
| **COPD** | **:** | Chronic Obstructive Pulmonary Disease |
| **CHF** | **:** | Congestive Cardiac Failure |
| **CRP** | **:** | C Reactive Protein |
| **DALY** | **:** | Disability Adjusted Life Years |
| **ECG** | **:** | Electrocardiogram |
| **ECHO** | **:** | Echocardiography |
| **FEV1** | **:** | Forced Expiratory Volume in One second |
| **FVC** | **:** | Forced Vital Capacity |
| **GOLD** | **:** | Global Initiative for Chronic Obstructive Lung Disease |
| **GCS** | **:** | Glascow Coma Scale |
| **HR** | **:** | Heart Rate |
| **ICU** | **:** | Intensive Care Unit |

| | | |
|---|---|---|
| **IHD** | **:** | Ischaemic Heart Disease |
| **IUGR** | **:** | Intra Uterine Growth Retardation |
| **LVD** | **:** | Left Ventricular Dysfunction |
| **LVRS** | **:** | Lung Volume Reduction Surgery |
| **mMrc** | **:** | modified Medical Research Council |
| **PHT** | **:** | Pulmonary Hypertension |
| **PaCO2** | : | Partial Pressure of CO2 in arterial blood |
| **PaO2** | **:** | Partial Pressure of O2 in arterial blood |
| **PASP** | **:** | Pulmonary Artery Systolic Pressure |
| **NIV** | **:** | Non Invasive Ventilation |
| **RA** | **:** | Right Atrium |
| **RTI** | : | Respiratory Tract Infections |
| **RV** | **:** | Right Ventricle |
| **RR** | **:** | Respiratory Rate |
| **SBP** | **:** | Systolic Blood Pressure |
| **SD** | **:** | Standard Deviation |
| **VPC** | **:** | Ventricular Premature Contraction |
| **WHO** | **:** | World Health Organization |

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# ABSTRACT

**BACKGROUND:**

COPD is a major cause of morbidity and mortality. It is the third leading and cardiovascular disease per se is high among COPD patients but the irony is that it often goes unnoticed because of nonspecific signs . The aim of the study was to find out the association of cardiac Troponin I levels in acute exacerbation COPD and its prognostic significance.

**OBJECTIVES:**

To evaluate the incidence of cardiac troponin I levels in acute exacerbation COPD patients admitted in our hospital ICU & Medical wards and to study the association of need for ventilator support , duration of hospital stay , and in hospital mortality as well as morbidity .

**METHODS:**

The study was a prospective study carried out in one year duration in our hospital Government Mohan Kumaramangalam Medical College and Hospital Salem, Tamil Nadu, India ,and blood levels of cardiac troponin I levels were obtained within 24 hours . A level above 0.017 microgram/ml was taken as positive. The following data was also recorded such as age , sex , smoking habits , tobacco Usage, comorbid conditions , clinical signs and symptoms , and investigations like ABG, ECG, ECHO, X Ray chest, PaSP, LVef, PEFR, FEV1/FVC . Statistical analysis was done and analyzed and tabulated.

**RESULTS:**

Among the 60 patients 30 were assigned in the acute exacerbation COPD group and 30 were in the stable COPD group. In acute group males were 23 and females were 7 and in stable group 23 males were there and females were 7 in number .Cardiac Troponin I was positive in 7 cases in the acute group whose percentage falls on 22 % , none of the stable cases were proved positive .Thus this **proves statistical significant association between cardiac troponin I positivity and need for ventilator support ,duration of hospital stay,** PaSP, PEFR , FEV1/FVC, ABG , abnormal ECG , ECHO , poor LVef , thereby **causing increased cardiovascular morbidity as well as mortality**

**CONCLUSION:**

Cardiac troponin I was significantly positive in subset of patients who were in acute group . These patients had longer duration of COPD, higher incidence of Ischaemic Heart Disease , higher need for ventilator support ( that too noninvasive ventilation) , increased ICU stay , and death among the trop I positive patients was found to be statistically significant . Thus the aim and the objectives of the study of trop I as a prognostic marker in acute COPD is met with .

**Key words :**

**COPD Exacerbation ,Troponin I , Cardiovascular Morbidity, Mortality.**

| SN.NO | LIST OF FIGURES | PAGE NO. |
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## INTRODUCTION:

Chronic obstructive pulmonary disease (COPD) is the third leading cause of death worldwide , and also causes significant morbidity resulting in economic and social burden which is substantial and is significantly increasing with time [3].

In 1990 COPD was the twelfth leading cause of disability adjusted life years (DALY ) lost in the world which corresponds to 2.1% of the total deaths . According to the varied studies and meta analysis COPD has projected from twelfth to third leading cause of DALYs lost in 2013 world wide [1].

According to WHO 65 million people globally suffer from moderate to severe COPD , of which 4.5 million deaths have occurred world wide because of COPD and its complications , and more than 90% of COPD deaths occurring in low and middle income countries because of poor socio economic status , changing age structure , life style modification which includes smoking and exposure to environmental pollutants [2] .

Smoking is the most important and an independent risk factor for COPD. Other non conventional forms of tobacco usage like hookah , beedi or chillum and chewing tobacco quid has been present in women also . The prevalence of COPD is proposed to have a progressively increasing course during the forthcoming decades due to changing life patterns.

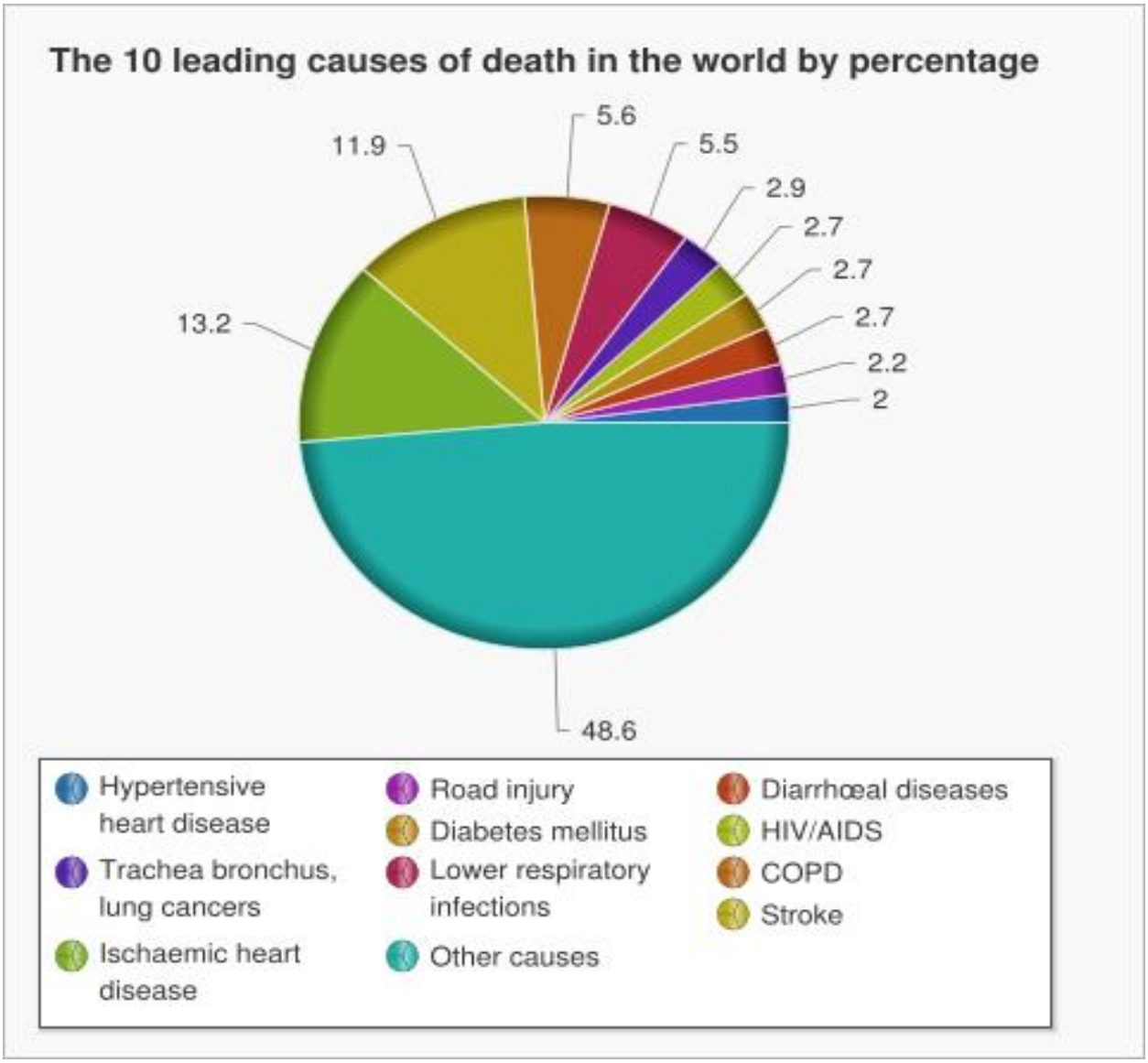
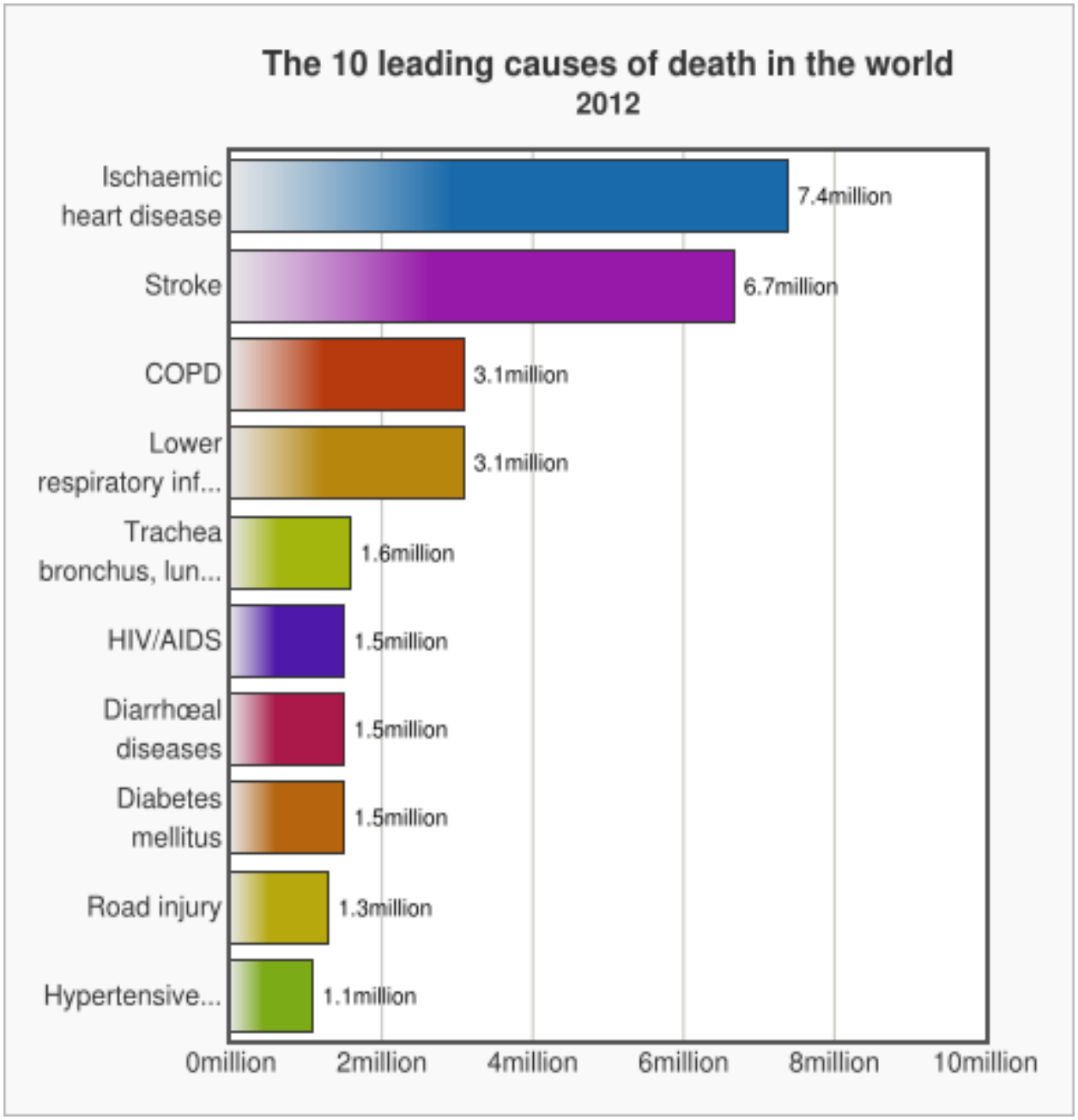



**Figure 1: percentage of deaths attributed to individual diseases**

**Figure 2: Top 10 Leading causes of death worldwide (WHO STATISTICS)**



## INDIAN SCENARIO:

Crude estimates suggests there are 30 million COPD patients in India. Of these 7% contribute to COPD deaths and 3% DALY lost[6]. There are very few studies in India regarding prevalence of COPD, but the available studies show a wide range from 3 to 7 percent, and it is also the fourth leading cause of death in men. The prevalence in women is about 3.5%.

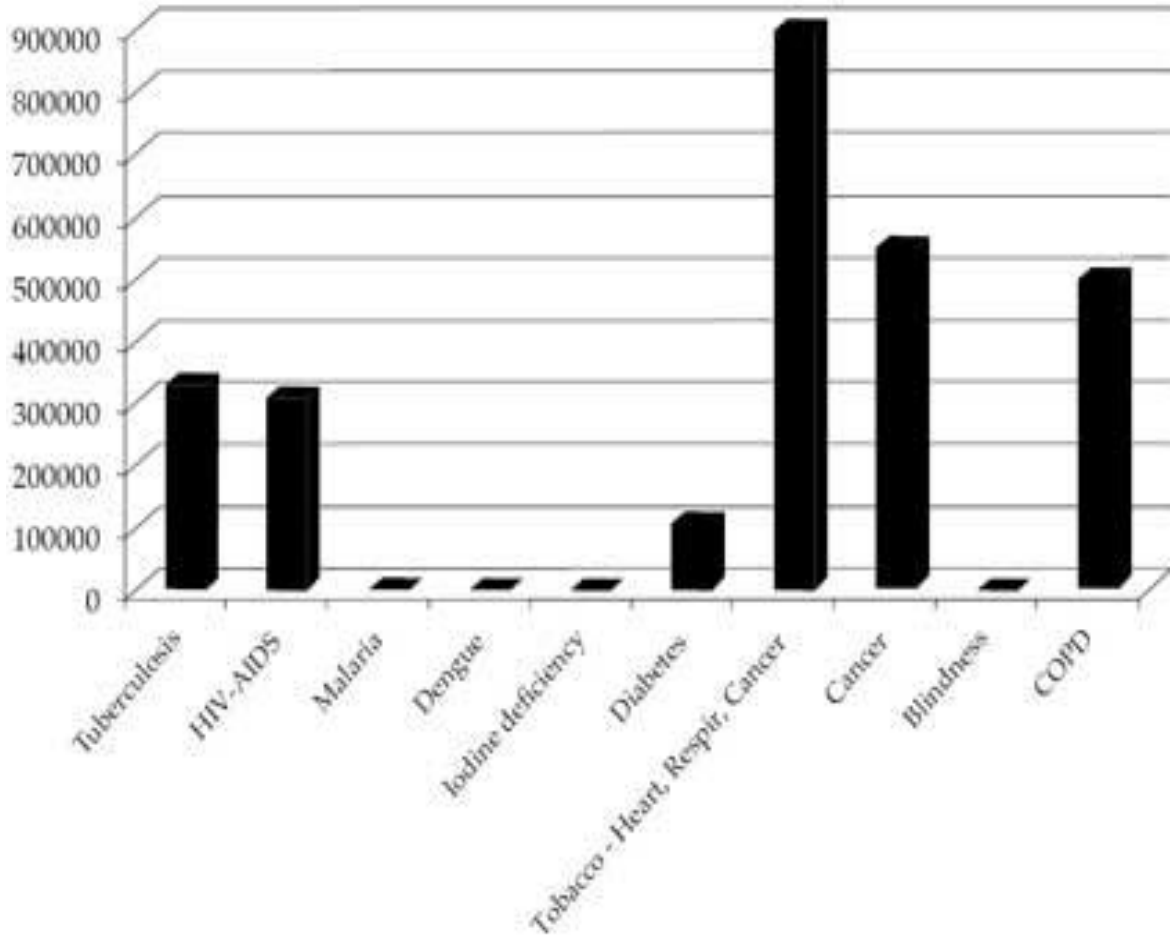



**Figure 3: mortality figures due to diseases covered under national health program**

**COPD AND CARDIOVASCULAR MORBIDITY:**

Cardiovascular compromise is frequently associated with COPD , particularly in acute exacerbations and is found to be a significant cause for morbidity and mortality in addition to the risk factors mentioned above.

Tools for detecting changes in cardiovascular homeostasis can be met with ECG , ECHOCARDIOGRAM as well as Cardiac Biomarkers .

**TROPONIN I** [52,53].

The importance of Troponin I levels in acute exacerbation of COPD causing cardiovascular morbidity has been but a new discovery .This study was undertaken to compare the levels of Troponin I in acute exacerbation of COPD Vs Stable COPD patients as matched controls [45] .

**AIMS OF THE STUDY :**

1) To find out the incidence of cardiac Troponin I levels elevation in acute exacerbation vs stable COPD patients .
2) To evaluate the association of elevated cardiac troponin I levels with
    a) Cardiovascular morbidity ,
    b) Need for ventilation ( both invasive and non invasive ) ,
    c) Length of hospital stay ,
    d) Mortality .

**OBJECTIVES OF THE STUDY:**

The study was done to find out the association between cardiac biomarker Troponin I in acute exacerbation vs stable COPD patients and its relation with cardiovascular morbidity and mortality thereby finding out the significance of the study in relation to social and economic burden by increasing the need for ventilation and duration of hospital stay .

# REVIEW OF LITERATURE :

## HISTORY OF COPD :

* The word emphysema dates way back to early 17 th century.

* ***Bonet*** first described voluminous lungs in 1679.

* ***Balle*** illustrated emphhysematous lungs in cadaveric dissection .

* ***Badham*** described the word catarrh to refer chronic cough , mucus hypersecretion . He described chronic bronchitis and bronchiolitis [3].

* ***Laennec*** invented stethoscope and describe emphysema (1821) in **His Treatise of diseases of the chest.**

* ***Osler*** believed emphysema was caused by excessive pressure in Alveoli [5].

* ***Gaensler*** introduced the concept of air velocity index and later the FVC which is the foundation of FEV1/FVC .

* ***Ronald Christie*** described physical signs of emphysema , chronic bronchitis and asthma .

* ***Dickerson Richards*** , Nobel laureate wrote about Cor Pulmonale .

* ***Reuben cherniack*** described respiratory acidosis in COPD patients.

* ***John Hutchison*** invented Spirometer in 1846 [4] .

* ***Menelee and Callaway*** described PFT in COPD patients .

* ***Mc Crinn and Paul D White*** (1931) were first to use the term **"Acute cor pulmonale" .**[7]

* ***Kountz and Alexander*** (1934) by their research in COPD observed that majority of the patients went in for cardiovascular morbidity [5].
* **Barach and Bickerman** (1956) edited the ***first textbook Pulmonary Emphysema*** and where the early champions of treatment for COPD.
* ***Prof. Setsuro Ebashi*** (1963) first described **TROPONIN [8].**
* ***William Briscoe and Nash*** (1965) first coined the term COPD in a discussion held at ***9 th ASPEN Emphysema Conference*** [4,6].

## DEFINTIONS:

### CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD) :

COPD is defined as a common preventable and treatable disease and is characterised by airflow limitation that is usually progressive and associated with an enhanced chronic inflammatory response in the airways and the lungs to the noxious particles or gases [1].

The above definition was give by WHO & The Global Initiative For Chronic Obstructive Lung Disease **(GOLD ),** a project initiated by the National Heart Lung and Blood Institute **(NHLBI) .**

### SUBTYPES:

1) CHRONIC BRONCHITIS , 2) EMPHYSEMA .

The other entities that produce obstructive lung diseases are Bronchial Asthma , Bronchiectasis , Genetic conditions like Immotile Cilia syndrome , Cystic Fibrosis , Hypogammaglobulinemia etc..

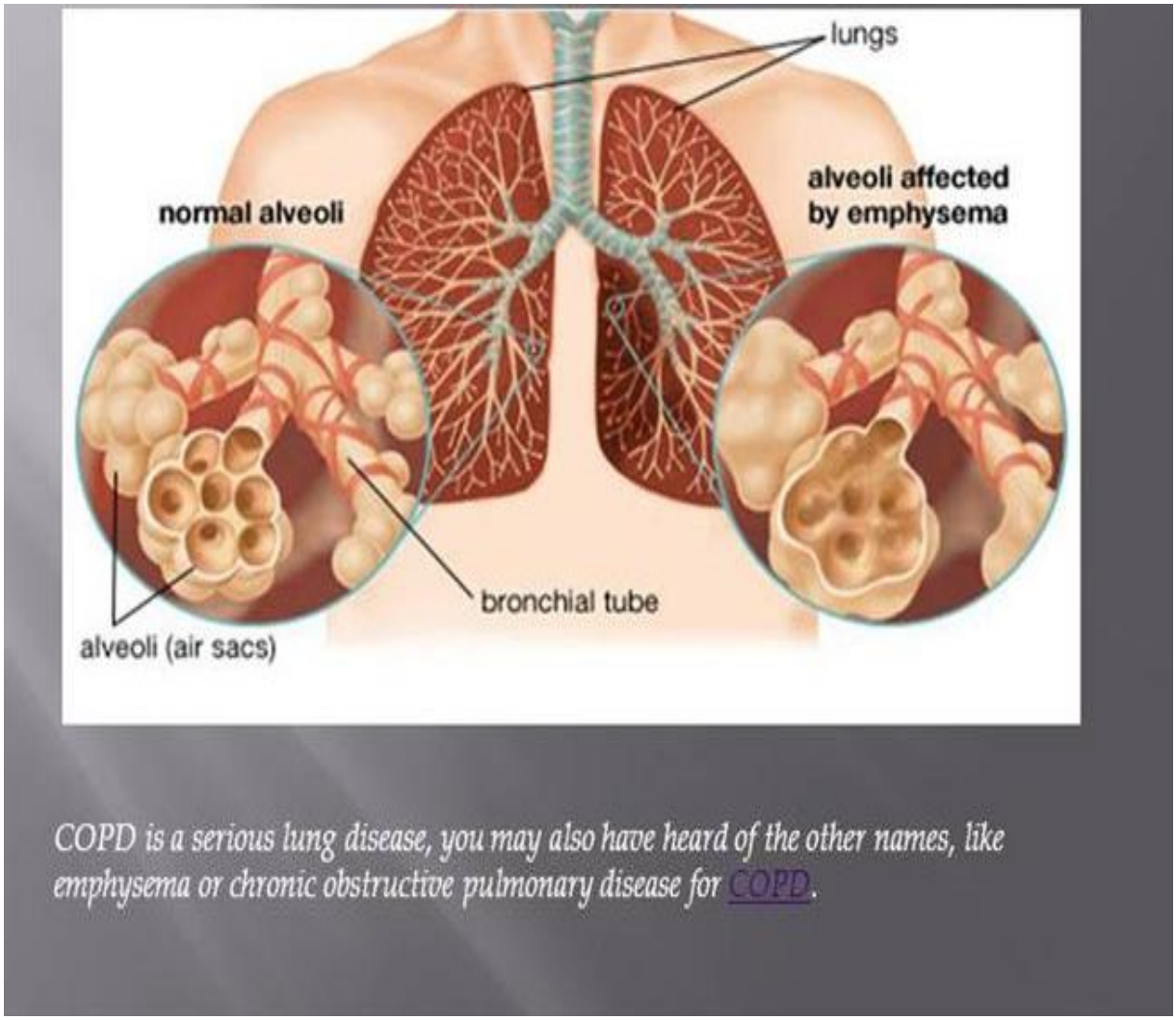
### CHRONIC BRONCHITIS:

Chronic Bronchitis is a clinical diagnosis defined by excessive secretion of bronchial mucus and is manifested by daily productive cough for 3 months or more in atleast two consecutive years [9].

### EMPHYSEMA:

Emphysema is a pathologic diagnosis and is defined by abnormal permanent enlargement of airspaces distal to the terminal bronchiole , with destruction of their walls and without obvious fibrosis [9].

**Figure 4 : Picture representing the destruction of airspaces in emphysema**



## DIAGNOSIS OF COPD :

The diagnosis of COPD (**according to GOLD criteria**) should be considered in any patient who has the following :

1) Cough ,
2) Expectoration ,
3) Shortness of breath or
4) History of exposure to risk factors for the disease .

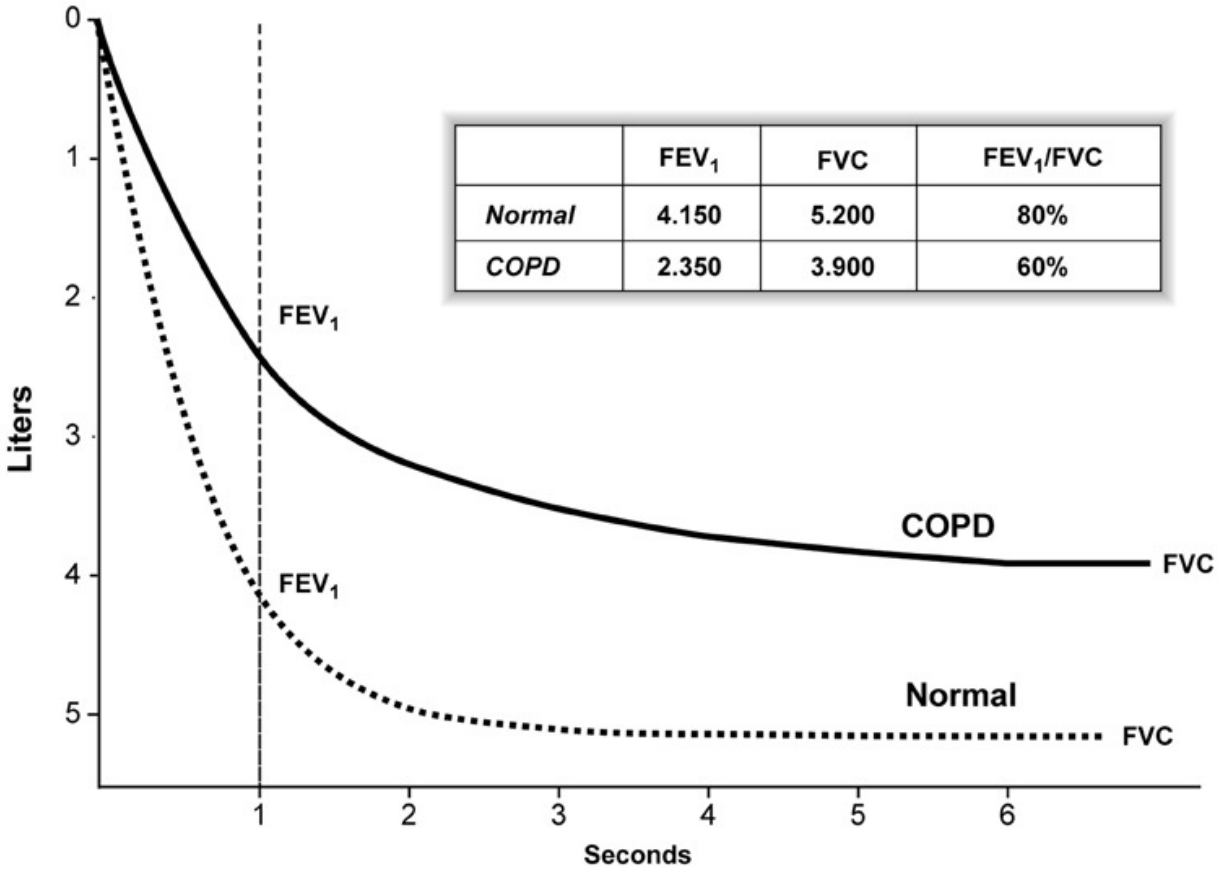
The diagnosis requires [33]

a) Spirometry ;

b) Post – bronchodilator forced expiratory volume in one second ( FEV1) / forced vital capacity (FVC) .--- FEV1/FVC $\leq$ 0.7 confirms the presence of airflow limitation that is not fully reversible .

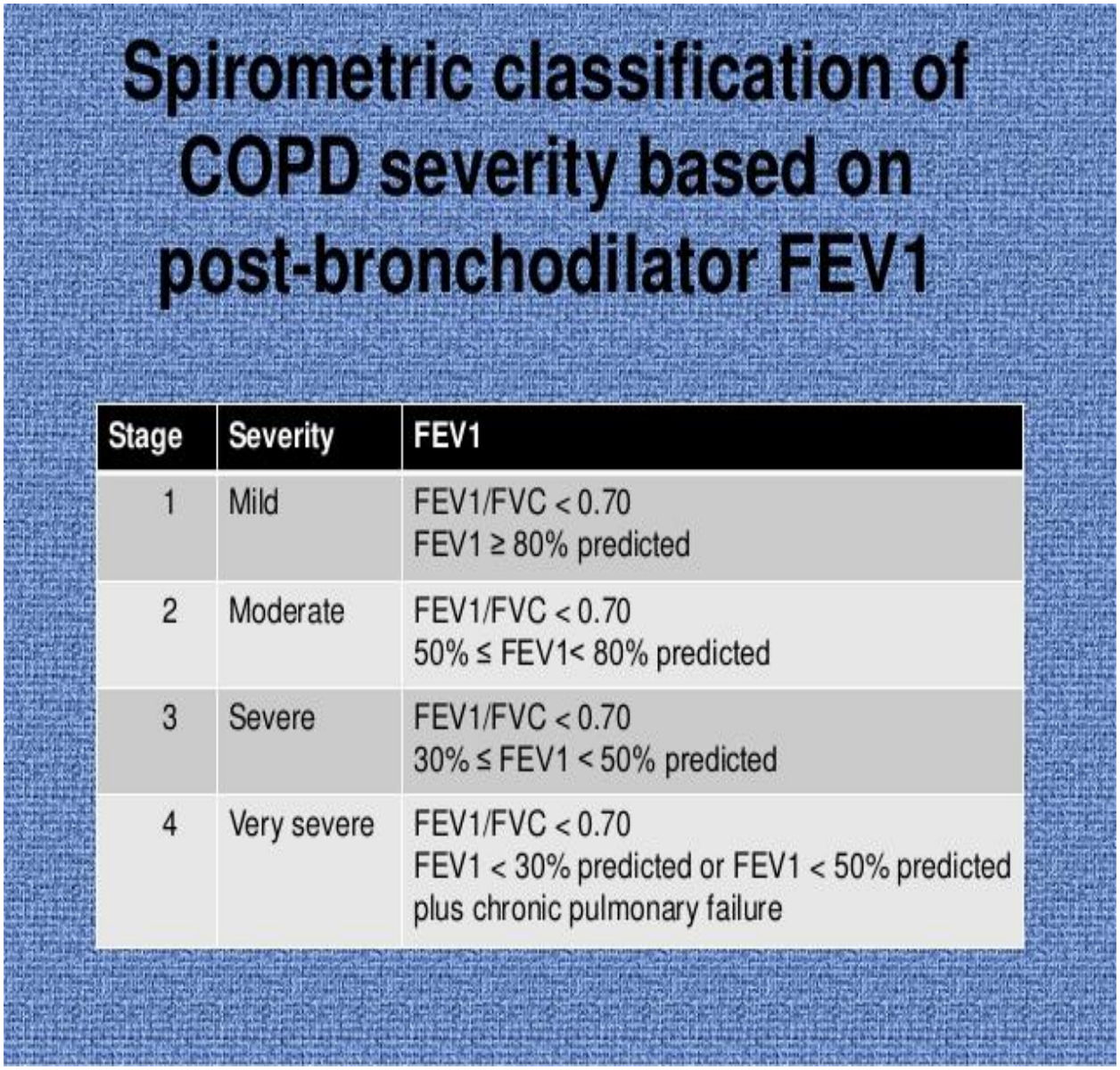
**Figure : 5 HAND HELD SPIROMETER**

**FIGURE 6 : GRAPHICAL REPRESENTATION OF NORMAL AND COPD PATIENTS BASED ON SPIROMETRY :**




| | $FEV_1$ | FVC | $FEV_1$/FVC |
|---|---|---|---|
| *Normal* | 4.150 | 5.200 | 80% |
| *COPD* | 2.350 | 3.900 | 60% |

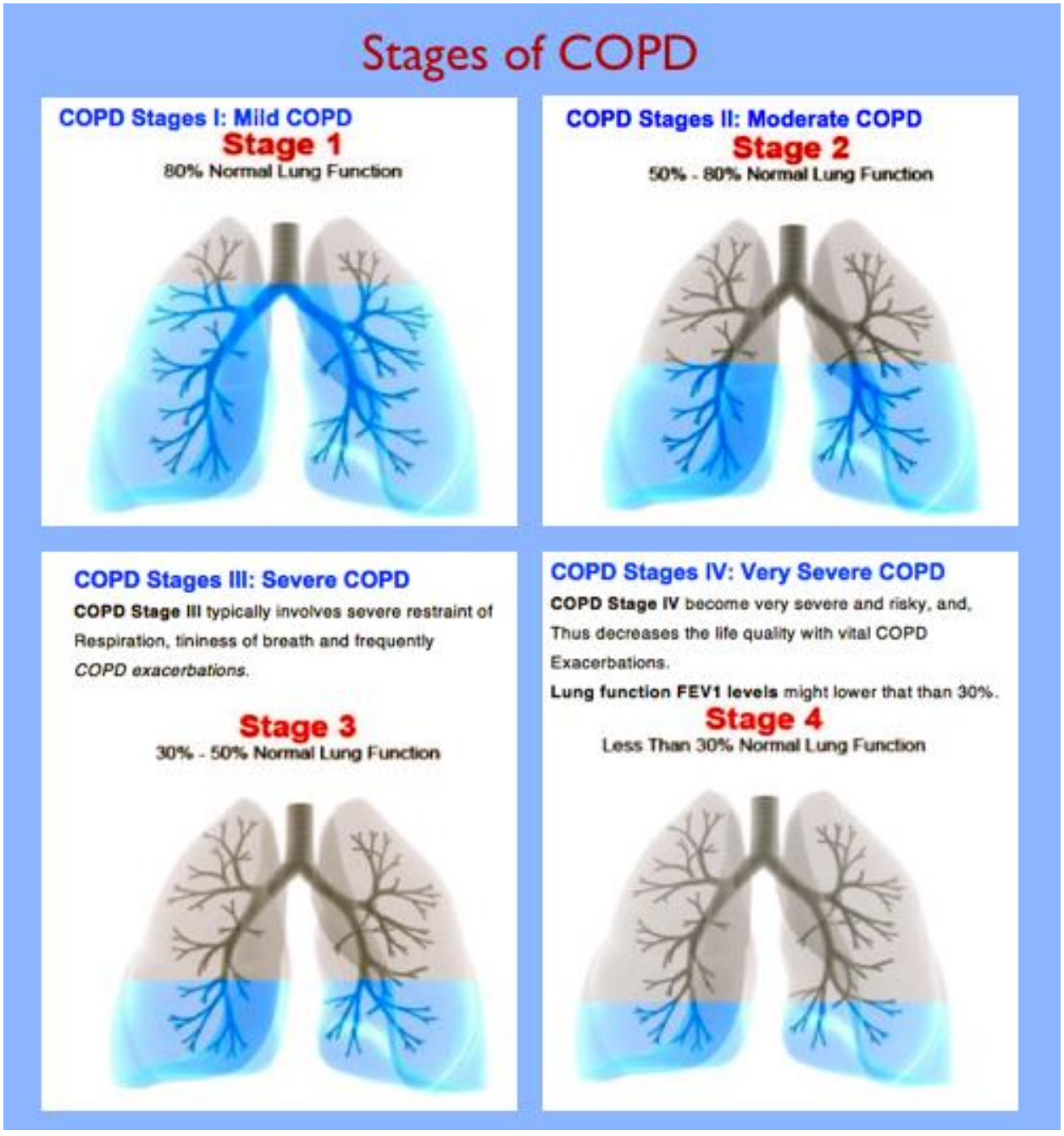
**Figure 7: GOLD classification of COPD severity**




**FEV1** - ***Forced Expiratory volume in 1 second*.**

**FVC** - ***Forced Vital Capacity*.**

**Figure 8: Figure showing the stages of COPD on the basis of lung function**



## EPIDEMIOLOGY:[1,10,11]

COPD is the third leading cause of death worldwide and also causes significant morbidity resulting in social and economic burden .

The incidence , prevalence , morbidity and mortality vary across different countries depending upon age , sex , race , co morbid conditions and the burden of COPD continues to increase in the forth coming decades due to exposure to risk factors like smoking , tobacco use and life expectancy of people and the personal habits and habitats of the individual .

WHO estimates that globally 65 million people have moderate to severe COPD . More than 3.5 million people died of COPD in 2009 , which corresponds to 5.6% of all deaths globally . Almost 80 - 90% of COPD deaths occur in low and middle income countries . Total deaths attributed to COPD are projected to increase by more than 30% in the next 10 years unless urgent action is taken to reduce the risk factors , especially tobacco use .

**RISK FACTORS THAT INCREASE THE RISK OF COPD :**

**Cigarette smoking** remains the single most risk factor for COPD , the evidence that non smokers do develop COPD , presenting with chronic airflow limitation suggests the combined role of factors like genetic susceptibility and exposure to irritant gases and environmental pollutants .

**INDIAN SCENARIO :** [12 ,13]

Crude estimates suggests there are 30 million COPD patients in India . Of these 7% contribute to COPD deaths and 3% DALY lost [1 ,11]. There are very few studies in India regarding prevalence of COPD , but the ones available show a wide range from 3 to 7 percent , and also the third leading cause of death . The prevalence of COPD in women is 7.5% .

**CLASSIFICATION OF RISK FACTORS:** [1 ,14]

Risk factors for COPD can be broadly classified under two heads such as Modifiable and Non modifiable risk factors .

**NON MODIFIABLE RISK FACTORS (Related with Host)**

1) Age
2) Sex
3) Race
4) Genetic factors

**Age :** 15% Develop progressively disabling symptoms in the 4 th or 5 th decade of life .

**Sex :** Male gender are at increased risk . In meta analysis the pooled Global prevalance was 9.8% in men and 5.6% in women . According to **BOLD STUDY** the prevalence is 11.8% and 8.5% in women .

**Race :** Asian , African and middle east are more prone .

**Genetic factors :** Airway hypereactivity , ***IgE sensitivity , atopy , alpha - 1 Anti trypsin deficiency , cystic fibrosis*** , bronchial asthma are some of the genetic factors involved in development of COPD . Single genes such as the gene encoding ***matrix metalloproteinase 12 (MMP12)*** have been associated with decline in lung function .

**MODIFIABLE RISK FACTORS :**

**( Related with exposure , habits and habitat )**

1) Smoking Habits ,
2) Tobacco use ,
3) Occupation ,
4) Socioeconomic Status ,
5) Environmental pollutants ,
6) Perinatal events ,
7 ) Recurrent RTI ,
8 ) Diet .

**Smoking:**

**Cigarette smoking is the single most commonly associated risk** factor in developing COPD . Smokers have increased lung function abnormalities chronic cough , breathlessness on exertion , recurrent RTI and

a gradual decrease in FVC as well as FEV1 and routed to greater prevalance of COPD and its exacerbation as well .

The risk is strongly related to [37]

a) Number of cigarettes smoked ,

b) Age of starting to smoke ,

c) Smoking habits ( deep inhalation , number of puffs , nicotine and tar content , length of cigarettes ) ,

d) Beedis contain more hydrocarbons ,

e) King size filter cigarettes deliver more tar and nicotine .

**Smoking in pregnancy :** This causes a significant increase in incidence of IUGR thereby affecting lung maturity as well as a defective immune system.

**Tobacco Usage :**

Other modes of usage of tobacco like pipe , cigar , water pipe , and tobacco chewing also predisposes an individual to develop COPD . Passive exposure to cigarrete smoke also poses a risk .

**Occupation :**

These constitute a small proportion due to organic and inorganic dusts , chemical agents , exposure to asbestos , arsenic , chromates , polycyclic aromatic hydrocarbons , nickel bearing dusts and fumes in their work places.

**Socio economic status:**

Over crowding , absence of cross ventilation and lack of awareness regarding personal hygiene , predisposes to COPD .

**Environmental pollutants :** [15]

1) **Indoor pollutants :** Almost 3.75 billion people use biomass , coal and firewood for their daily cooking and household works . This account for the prevalence of COPD in non smokers especially women in Asia , Africa and Middle east .

2) **Outdoor pollutants :** Air pollution resulting from combustion of Fossil fuel , emission of fumes from silencers from motor vehicles in crowded cities contribute under this head .

**Perinatal Events:**

This includes birth asphyxia , respiratory distress syndrome requiring prolonged oxygen supplementation thereby resulting in broncho pulmonary dysplasia which predisposes to pulmonary function compromises .

**Recurrent RTI:**

Recurrent respiratory tract infections in the childhood has been associated with reduced lung function and increased respiratory compromises in adulthood .

In India **Tuberculosis and its sequale** has been found to be an important risk factor for development of COPD.

## PATHOLOGY OF COPD :[17,18]

***History:***

The first credible production of emphysema was desribed by ***Gross*** in 1964 who instilled pancreatic extracts ( papain ) into the guinea pig alveoli to cause subsequent destruction of alveoli resulting in hyperinflation **. *Laurel and Ericsson*** in 1963 in Swedon described the proteolytic damage of elastin the forerunner of Protease and Antiprotease therapy in alpha 1 antitrypsin deficiency causing emphysema .

**Pathogenesis:**

Research has been focussed on the release of ***neutrophil elastase*** and its role in destruction of lung elastin . Another role is played by ***matrix metalloproteinases ( MMPs )*** produced by macrophages which are activated by inflammatory mediators triggered by cigarette smoke and result in recruitment of neutrophils and other inflammatory cells into the lung alveoli leading to destruction of elastin and other connective tissue elements present in the lungs as time advances . These collectively leads to loss of elastic recoil and destruction of alveolar structures .

**Role of Oxidative stress :**

Cigarette smoke induces oxidative stress in the lung alveoli including the oxidative inactivation of antiproteases allowing the expression of various proinflammatory genes , which promotes cytokine production and consequtive destruction of alveoli . Finally apoptosis of pneumocytes and endothelial cells occur that explains the permanent destruction of alveoli .

**PATHOPHYSIOLOGY:**[19]

Pathological changes characteristic of COPD are found in the proximal airways , peripheral airways , lung parenchyma , and pulmonary vasculature .

**Airways** :

Airways abnormalities in COPD include chronic inflammation , Goblet cells hyperplasia , mucus gland hyperplasia as well as hypertrophy , fibrosis , narrowing and reduction in the number of alveoli , and alveolar collapse due to the loss of elastic recoil leading to alveolar destruction and emphysema .

**Lung parenchyma :**

Emphysema causes destruction of structures distal to the terminal bronchiole , which comprises the respiratory bronchiole , alveolar ducts & sacs and alveoli collectively termed the acinus , the associated capillaries and interstitium therby leading to permanent fibrosis to the lung parenchyma .

**Pulmonary vasculature :**

The pulmonary vasculature undergoes a varied disruption of its nomenclature like intimal hyperplasia , bronchial smooth muscle hypertrophy and hyperplasia and prolonged hypoxia and hypoperfusion which finally leads to vasoconstriction of the small pulmonary arteries and arterioles .

**Physiological abnormalities in COPD :**[19]

- Hypersecretion of mucus ,
- Limitation of airflow and air trapping leading to hyperinflation ,
- Abnormalities in gas exchange and ventilation perfusion mismatch ,
- Respiratory failure and pulmonary hypertension ,
- Systemic effects of inflammation and skeletal muscle wasting further reduce exercise intolerance thereby worsening the prognosis .

**Symptoms & Signs :**

**Initial presentation :**

1) Cough with expectoration

2) Shortness of breath – insidious in onset , gradually progressive .

As the disease progresses , two symptom pattern emerges which may be referred historically as ***pink puffers ( chronic bronchitis )*** and ***blue bloaters ( emphysema ) .***

Clinical course may involve other factors such as central control of ventilation and concomittant sleep disorder breathing .Most of the initial presentations goes unnoticed because of its milder course and easy control with drugs and contribute to stable COPD cases .

**Late presentation :**

During later stages of COPD patient may present with Pneumonia , Pulmonary hypertension , Cor pulmonale and Chronic respiratory failure .

**ACUTE EXACERBATION :**

The ***hallmark of COPD*** is the exacerbation of symptoms which includes

1) Symptoms beyond normal day to day variation ,

2) Increased dyspnoea ,

3) Increased severity of cough and its frequency ,

4) Increased amount of sputum ,

5) Change in the characteristics of sputum ,

6) Altered mental status ( deteriorating GCS )

These **exacerbations are usually precipitated by infections** ( commonly **Viral** than Bacterial ) as well as environmental factors of course COPD exacerbations widely vary in severity which require a change in regular therapy.

**ASSESSMENT OF DISEASE** : [1]

COPD assessment is done to determine the disease severity , its exacerbations and its triggers , seasonal variation , hospital inpatient admissions , morbidity and mortality , which has a greater impact on the socioeconomic burden of people eventually and causing loss of effective work days leading on to greater loss of DALYs , as well as a guide to treatment and therapeutic options .

The **Gold guidelines** are based upon a combination of

1) Patient's ***symptoms***
2) ***Number of exacerbations*** in a specified period of time
3) ***Spirometric assessment of FEV1*** to determine the exacerbation risk & Therapy in the form of

a) Need of oxygen supplementation ,

b) IV antibiotics ,

c) Bronchodilators ( both iv and inhalations ).

Symptom severity is assessed using

**1)** Modified Medical Research Council ( mMRC ) .

**2)** COPD assessment test ( CAT ) .

**Modified Medical Research Council ( mMRC ) dyspnoea scale :**

**Grade 0 :** No Breathlessness except on strenuous exercise .

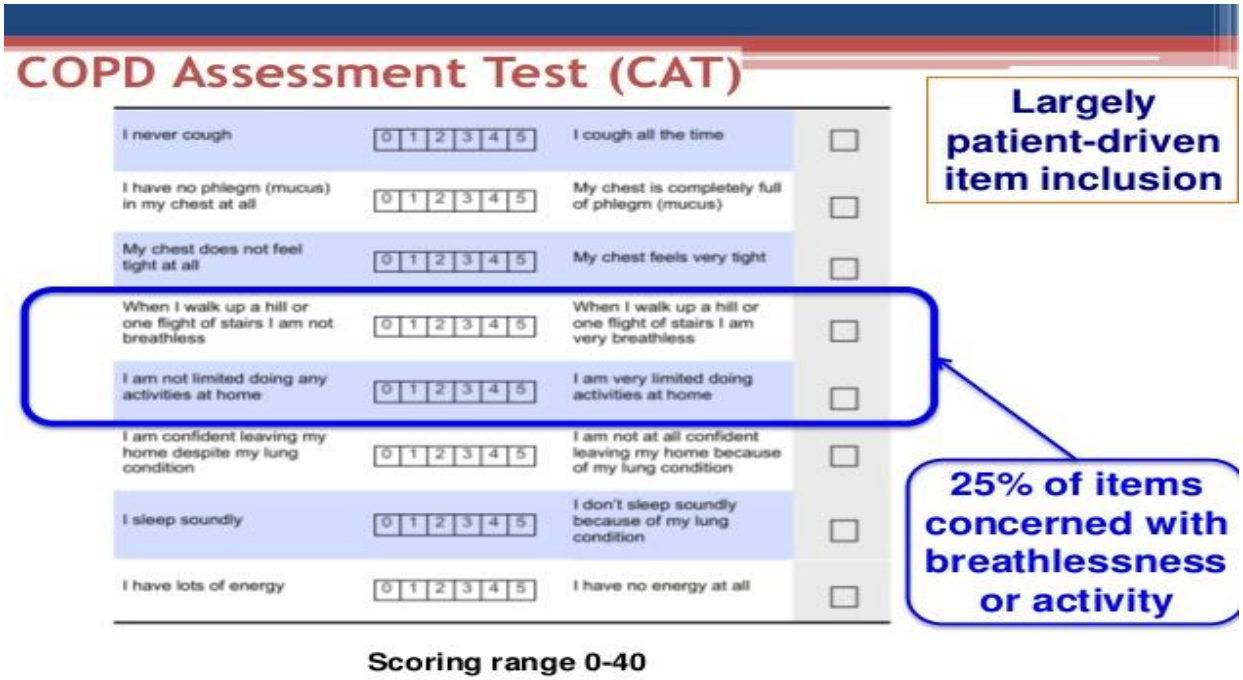
**Grade 1:**Breathlessness when hurrying on a level or walking up a slight hill

**Grade 2 :** Walks slower than contemporaries on level ground because of breathlessness or has to stop for breath when walking at own pace .

**Grade 3 :** Stop for breath after walking every 100 m or after a few minutes on level ground .

**Grade 4:** Too breathless to leave the house or breathless on dressing or Undressing .

**Figure 9: COPD Assesment Test (CAT ) :**




**The GOLD classification is based on** [36]

The number of exacerbations in the previous 12 months can be made use in predicting future risk of complications .

1) A history of zero or one exacerbation in the past 12 months suggests a low future risk of exacerbations
2) A history of two or more exacerbations suggest a high future risk .
3) Post bronchodilator therapy FEV1 .

These three components are collectively grouped under :

**Patient Group A** :

Low Risk , Less Symptoms Typically GOLD 1 or GOLD 2

( Mild or Moderate airflow limitation ) and / or 0-1 exacerbation per year

and **mMRC grade 0-1 or CAT score < 10**

**Patient Group B** :

Low Risk , More Symptoms Typically GOLD 1 or GOLD 2

( Mild or Moderate airflow limitation ) and / or 0-1 exacerbation per year

And **mMRC grade ≥ 2 or CAT score ≥ 10**

**Patient Group C** :

High Risk , Less Symptoms Typically GOLD 3 or GOLD 4

( Severe or Very Severe airflow limitation ) and / or ≥ 2 exacerbations per

year and **mMRC grade 0-1 or CAT score < 10**

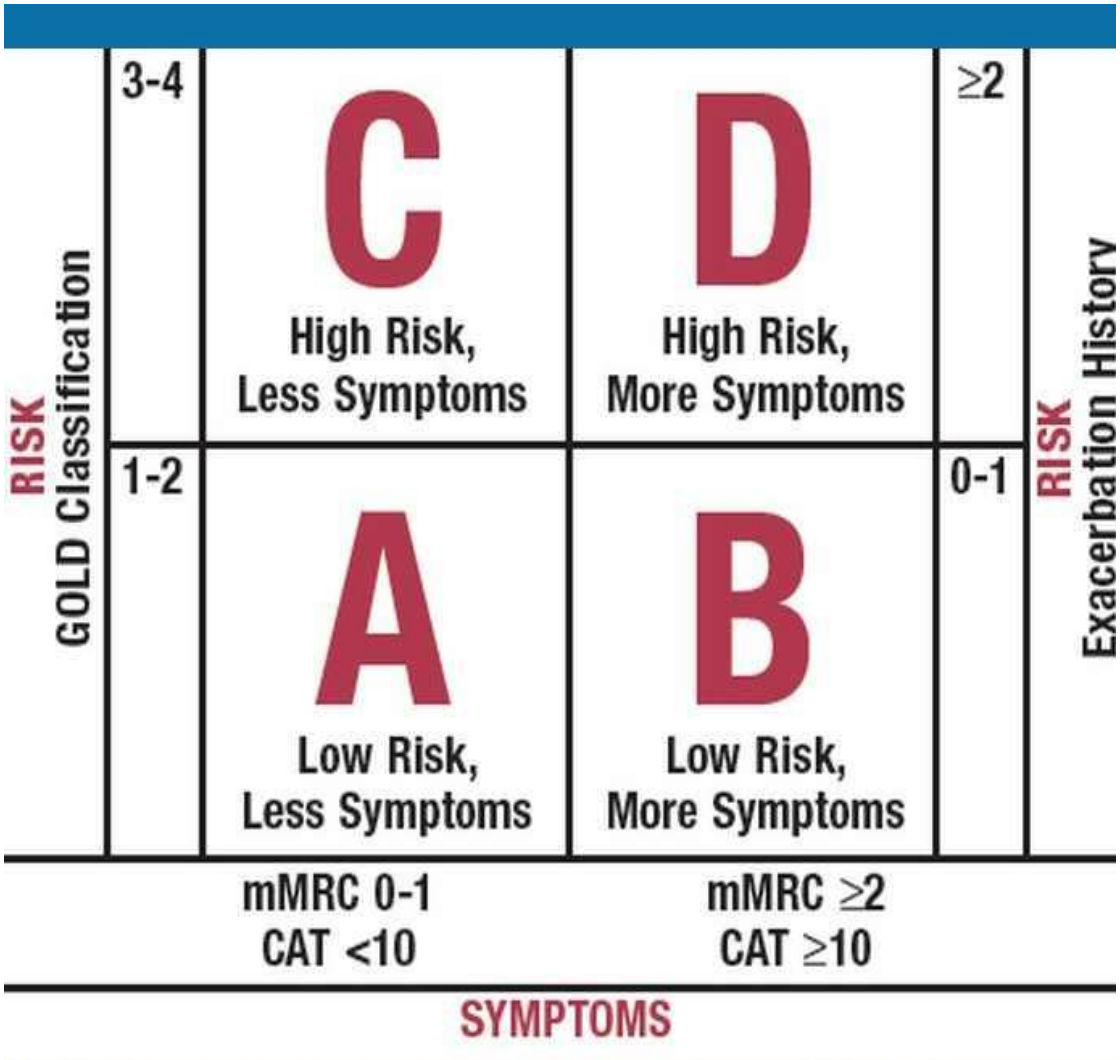
**Patient Group D :**

High Risk , More Symptoms Typically GOLD 3 or GOLD 4

( Severe or Very Severe airflow limitation ) and / or ≥ 2 exacerbations

per year and **mMRC grade ≥ 2 or CAT score ≥ 10 .**

**FIGURE 10 : GOLD CLASSIFICATION OF SEVERITY**



## LABORATORY FINDINGS :

**Based on physiology :**

1) **SPIROMETRY:**

The severity of COPD can be assessed by Spirometry which provides objective information about pulmonary functions and can predict the response to therapy . ***Initial abnormalities in PFT*** [19] ***is only reduced Mid Expiratory Flow Rate*** . Later it progresses to reductions in FEV1 there by leading to reduced FEV1/FVC ratio . Further during endstage FVC is markedly reduced , Residual Volume (RV) is increased thereby Total Lung Capacity (TLC) increases . The ratio of RV/TLC increase clearly indicates air trapping especially in emphysema .

2) **ARTERIAL BLOOD GAS:**

Early stages show an increase in A-a Do2 .

Later stages show hypoxemia and hypercapnia as evidenced by a decrease in po2 and increase in pco2 , and thereby leading to respiratory acidosis which is compensated by metabolic alkalosis , which when untreated progress to respiratory failure (Type 2) .

**Based on Microbiology :**

3) **SPUTUM EXAMINATION :**

This is non specific test for acute exacerbation but sputum may reveal organisms like *Streptococcus pneumonia , Hemophilus influenza , Klebseilla species* in Indian population .

(western data reveals sputum positive for *Moraxella catarrhallis* ) in Culture or by gram staining .

**4) ELECTROCARDIOGRAM :**

ECG may show different patterns which ranges from

a) Sinus tachycardia

b) P Pulmonale

c) Tall tented T waves

d) Rarely supraventricular arrhythmias

e) Multifocal atrial tachycardias

f) Atrial flutter & Atrial fibrillation

g) Right Bundle Branch Block

h) Right Axis Deviation

**IMAGING :**

**a) CHEST X RAY :**

Radiograph of patients with chronic bronchitis without any obvious exacerbations show non specific changes like peribronchial and perivascular markings . Patients with Emphysema show typical bilateral hyperinflation with flattening of diaphragm with tubular heart shadow and peripheral pruning of blood vessels suggesting pulmonary arterial hypertension .

**Figure 11: ECG patterns in COPD**

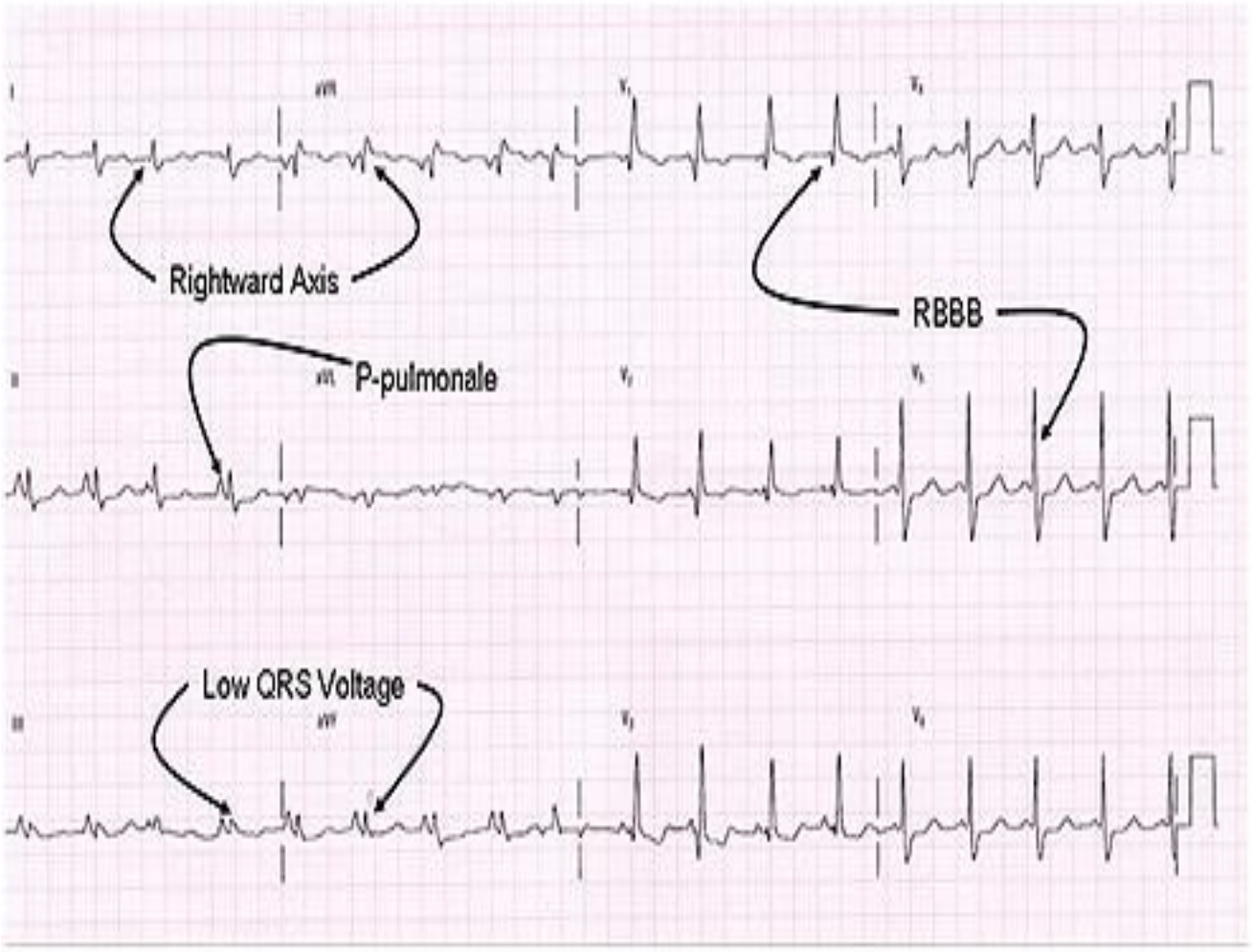



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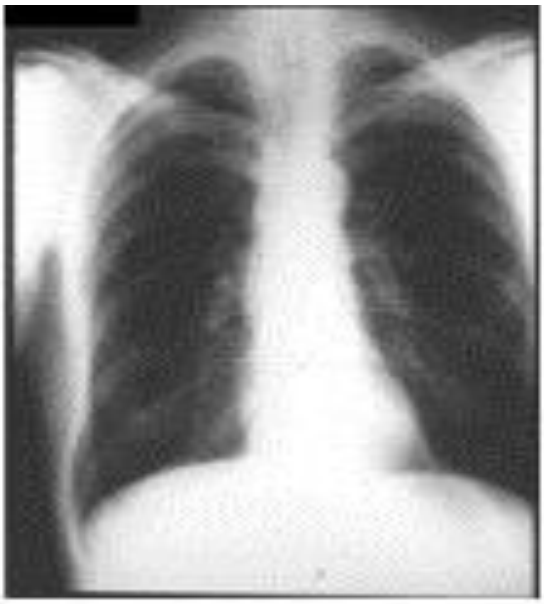
**Figure : 12 XRAY picture of emphysema**



COPD
Note narrow, vertical heart
and low, flat hemidiaphragms

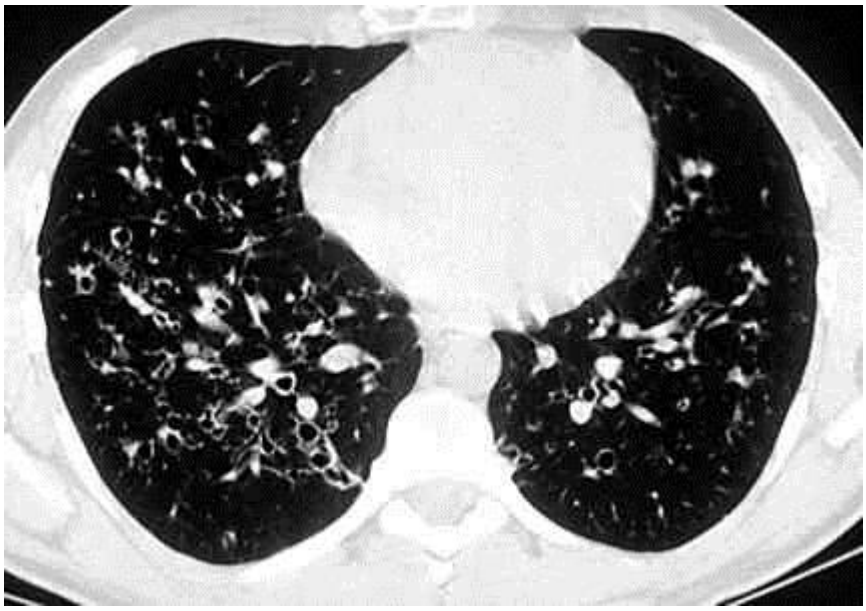
**2 ) CT CHEST :**

**HRCT *(high resolution)*** is more sensitive and specific diagnostic tool in COPD . Pulmonary hypertension may be evidenced by enlargement of central pulmonary arteries .

C **) ECHOCARDIOGRAPHY :**

ECHO provides an estimate of ***pulmonary artery pressure (Pasp)*** and chamber dilatation ( RA/RV Cor pulmonale ) and predicts heart failure both *systolic and diastolic dysfunction* . **The LV ejection fraction** can also be determined .

**Figure 13: COPD changes in HRCT chest**



**Dorsal on the right the dilated bronchi are visualized along their axis**

**Prevention of COPD may be studied under different heads ,**

****Primordial Prevention :**[37]

This is the single most important issue wherein the disease can be prevented before the occurrence or the emergence of risk factors, this may be difficult to be aimed at , but regarding COPD if primordial prevention is achieved , almost all the modifiable risk factors in the development of COPD can be met with . This can be achieved by mass health education .

****Primary Prevention**:

This is aimed at pre-pathogenesis phase which can be achieved by

a) Population ( mass ) strategy .

b) High risk strategy .

(smokers , miners , drillers , other occupational exposures)

This is action taken prior to onset of the disease which includes the elimination of all risk factors involved in the development of COPD .

**A ) Health promotion**

**B ) Specific protection**

are the measures followed here.

**Secondary Prevention** :

This is achieved by actions which " halts the progression of the disease (COPD) '' which has already occurred .

**A) Early diagnosis** , **B) Prompt treatment**, is done to prevent exacerbations.

**Tertiary Prevention** : This is aimed at late pathogenesis phase , includes A ) Disability limitation , & B ) Rehablitation .

**Relapse** :

Relapse may be defined as whenever the exacerbation definition is met within 28 days of previous exacerbation .

**Legislative measures :** [22] Restrictive measures include

a) Heavy taxation ,

b) Control of sales promotion ,

c) Banning of advertisements of tobacco products ,

d) Banning of smoking in public and work places,

e) Sales restriction .

The Government Of India has provided legislative support to Anti – smoking campaign by enforcement of

1) **" The Cigarettes Act : (1975) "**[37]

( regulation of production , supply and distribution ) which came into force from April 1st 1976 .

2) **" The Cigarettes & Other Tobacco Products Act : (2003) "**

( prohibition of advertisement and regulation of trade and commerce , production , supply and distribution )

## EXACERBATIONS : [21]

Acute COPD exacerbation is defined as "An event in the natural course of the disease which is characterized by a change in the patient's baseline dyspnea , cough , and or sputum that is beyond normal day - to – day variations , and is acute in onset , and may warrant a change in regular medication in a patient with underlying COPD." The chronic and progressive course of COPD is explained by its exacerbations and remitting course .[1 ,21]

On an average COPD patients have exacerbations of 1.3 to 1.5 times per yr.

## BURDEN OF COPD EXACERBATION [28] :

COPD exacerbations do cause serious and deleterious effects to the Quality of Life in terms of

a) loss of effective work days

b) Increased effort intolerance

c) Decreased apetite

d) Increased need for hospitalizations

e) Increased number of DALYs

Thereby increasing the social and economic burden which leads to poor quality of life and may also lead to permanent loss of pulmonary function

which is a vicious cycle causing further morbidity. COPD exacerbations needing inpatient admissions account for a major portion of loss of economy for a developing country like India .

**Prevalence Of COPD Exacerbations :**[25]

COPD exacerbations is approximately quantified to more than 20% to 23% of hospital admissions per year in India land approximately 35% of those require admission to an Intensive Care Unit [24].

**AETIOLOGY OF ACUTE EXACERBATION [22] :**

Pulmonary infections are the foremost cause of an acute Exacerbation and can be grouped under

1) **Viral Infections :**

Commonest - Influenza , rhino , respiratory syncitial virus and adeno viruses .

2) **Bacterial Infections :**

Streptococcus pneumonia ( Pneumococcus ) , H.influenza , Klebsiella species ( Moraxella catarrhalis in western world ) are attributed to bacterial causes .

3) **Others :**

**a) Sinus** infections ,

**b) Environmental pollutants** : Both indoor and outdoor pollutants,

**c) Heart failure :**Acute pulmonary edema (acute LV failure)

**d) Pulmonary embolism :** Blood clots to the lungs may trigger .

**e) Pneumothorax :** Due to rupture of staphylococcal bullae , subpleural blebs . About 30% to 35% the etiology remains unidentified because the clear trigger or the precipitating factor goes unnoticed .

**CARDIOVASCULAR MORBIDITY [30 ,31]:**

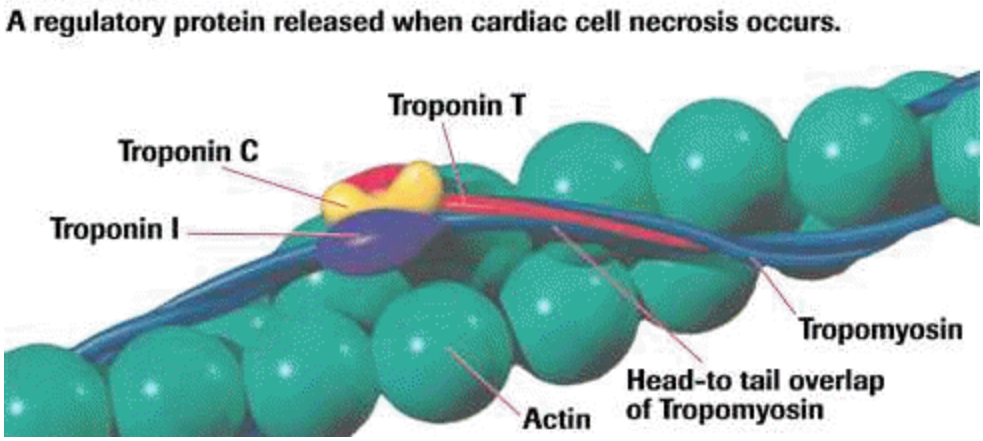
The left ventricular dysfunction poses a great risk for an acute exacerbation and vice versa also holds good evidence . This provides a vicious cycle in increasing the cardiac burden . This can be presented even if the patient doesn't have cor pulmonale .

Prompt identification of an acute exacerbation and its appropriate treatment may significantly influence the outcome of the disease thereby decreasing the cardiovascular morbidity as well as mortality .

**TROPONIN I [53]:**

Cardiac troponins especially TROPONIN I which can be measured by a simple test as a biomarker is widely available and can help us identify the patients with acute exacerbation and thereby predicting the need for hospitalization and intensive care which is the main aim of the study. GOLD endorses that "A biomarker or a panel of biomarkers would be made available in the near future which focuses on accurate etiologic diagnosis" .

**Figure 14: Electron microscopic picture of cardiac cell**




**Troponin I** has three isoforms of which two are present in skeletal muscle and one in the cardiac muscle .The cardiac isoform has a molecular weight of 24,000 Da , larger than other isoforms since it contains 32 amino acids extra in N terminal peptide end [46] .

The cardiac troponin exists in 2 populations inside the cell . The majority of troponin is bound within the cardiac apparatus within the contractile system small amount is free in the cytosol about 2 -4 % [46,47] .Damage to the cardiac myocyte and cardiac injury occurs in acute exacerbation of COPD along with loss of membrane integrity ,immediate release of free cytosolic troponin I into the circulation followed by release of structurally bound troponin resulting in sustained elevation [47,48] .

**ASSESSMENT :**[19]

The following classification is a crude one based on subjective symptoms and clinical signs easy for choosing treatment at the field level and rank the clinical relevance of the episode and its outcome .

**Level I**: Home based treatment .

**Level II:** Inpatient admission.

.

**Level III:** Intensive care requirement ( patients with respiratory failure )

**HISTORY:**

A relevant and detailed history from the patient which includes

a) Symptom analysis

b) Duration of symptoms

c) Worsening of symptoms

d) Appearance of new symptoms

e) Number of previous episodes of exacerbation

f) Number of previous hospitalizations (need for o2 and ventilation)

g) Comorbid conditions

h) Sedentary lifestyle

i) Personal habits ( smoking , tobacco usage )

j) Habitat ( dwelling place related to environmental pollutants )

k) Socioeconomic status ( overcrowding ) .

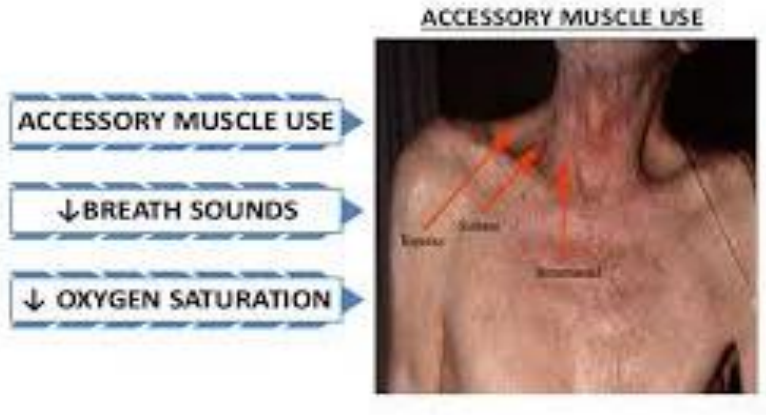
**Symptoms and Signs of COPD Exacerbation.**[22]

Symptoms and Signs can be classified under the following systems :

**Respiratory System [22]:**

1) Cough with expectoration ( increased frequency , consistency )
2) Characteristics of sputum ( alteration i.e thick tenacious , colour change, Foul smelling , increased volume)
3) Breathlessness (on exertion or at rest )
4) Wheeze
5) Fever

**Figure 15 :Patient with acute exacerbation of COPD**




**Cardiovascular symptoms :**

1) Chest tightness
2) Perspiration
3) Effort intolerance
4) Fatiguablity

5) Tachycardia ( palpitation )

6) Cyanosis ( in extremes of severity )

7) Crepitations and Gallop rhythm

8) Muffled heart sounds

**MUSCULOSKELETAL SYMPTOMS:**

1) Decreased exercise tolerance

2) Loss of apetite

3) Loss of weight and systemic malaise .

**PSYCHIATRIC SYMPTOMS** :

1) Insomnia

2) Confusion

3) Disorientation

4) Depression

5) Anhedonia

6) Apprehension

7) Anxiety .

**CENTRAL NERVOUS SYSTEM :**

1) Altered higher mental functions

2) Reduced bulk in chronic cases

3) Language disabilities

4) Perceptual motor functioning

5) Memory dysfunction and

6) Attention deficit

***Signs of Severity in exacerbations* : [1,35 ,42]**

a) **Accessory muscles** of respiration over activity

b) **Cyanosis** - newer onset or worsening ( **VA/Q mismatch** )

c) Paradoxical movements of chest wall (**respiratory failure)**

d) Bilateral pitting pedal edema ( **corpulmonale** )

e) Hemodynamic compromise or instability ( **shock** )

f) **A silent chest** on auscultation (severe bronchospasm)

g) **Mental status deterioration** (GCS ) due to hypoxemia

The risk of death from an acute exacerbation increases with the presence of comorbidities , development of respiratory acidosis , and the need for assisted ventilation either invasive or noninvasive . [36]

## POTENTIAL DANGER SIGNS OF COPD FOR HOSPITALISATION:[22]

1) Severe underlying COPD

2) Sudden increase in intensity and frequency of symptoms , (dyspnoea )

3) Onset of newer physical signs (e.g., central cyanosis , pedal edema)

4) Failure of medical therapy given initially

5) Varied comorbidities (e.g., CCF , arrhythmias , AF or atrial ectopics )

6) Exacerbations occurring frequently in shorter time span

7) Older age group

8) Inadequate financial and social support .

## TREATMENT OF EXACERBATIONS :

### Treatment for hospitalized patient :[28]

The ***three main goals*** in the treatment for exacerbations of COPD are

*To decrease the morbidity & reduce the impact of the present episode.

* To prevent the individual in developing subsequent exacerbations.

* To improve lung functions.

### TREATMENT OPTIONS :

An acute exacerbation COPD patient can be managed depending upon

a) The severity of exacerbation

b) The underlying disease

c ) The presentation of signs and symptoms

d) The duration of the present episode

Depending upon the above mentioned factors exacerbations can be managed on an outpatient basis or in an inpatient setting [26].

More than 70% to 80% of exacerbations can be managed on an outpatient basis with pharmacologic drug therapies which includes

1) Bronchodilators
2) Corticosteroids ,
3) Antibiotics ,
4) Oxygen therapy.

**BRONCHODILATORS :**[22 ,28.]

Bronchodilators remain the central stay in the management . The **inhaled route** is the ideal one preferred . There are different agents ( delivery devices ) for the usage of drugs . Choice of the delivery device should be left to the patient's preference .

Bronchodilators improve the symptoms , exercise tolerance , and overall health . Aggressiveness of therapy should be based on patient's severity . They are grouped under ***three heads***

***1) BETA 2 AGONISTS :*** a) short acting B2 agonists ,

b) long acting B2 agonists.

***2) ANTICHOLINERGICS :*** a) Ipratropium bromide ,

b) Tiotropium bromide .

***3)METHYL XANTHINES :*** a) Theophylline ,

b) Aminophylline ,

c) Doxophylline ,

d) Hydroxyethyl theophylline .

1)**BETA 2 AGONISTS :**

***Mechanism Of Action :*** Beta 2 receptor stimulation present in bronchial smooth muscle producing bronchodilation .

Increased cAMP results in decreased mast cell mediators release .

**Short acting beta 2 agon ists** :

1) Salbutamol
2) Albuterol
3) Metaproteranol
4) Terbutaline .

**Dosage :** Typical doses are 2 to 4 puffs (100 to 200 mcg ) every 6 hrs . They are less expensive , more rapid onset of action , and greater patient satisfaction . Combined action of anticholinergic and b2 agonist provides bronchodilation but does not improve dyspnoea .

**Side effects :** Tachycardia , palpitation , tremors , restlessness , hypokalemia , nervousness , rarely ankle edema .

**Long acting beta 2 agonists :**

1) Salmeterol
2) Bambuterol
3) Formoterol

These drugs are used once a day thereby more helpful in prophylaxis than in acute attacks , best indicated in nocturnal exacerbations . Much more helpful for abolishing frequent episodes of exacerbations .

## 2. ANTICHOLINERGICS : [28]

**Ipratropium bromide:** This is the most commonly prescribed drug which is * an anticholinergic ,

* short duration of action ,

* absence of sympathomimetic side effects .

***Mechanism of action* :** Blocking M3 receptor mediated cholinergic constrictor tone causing bronchodilation . Acts primarily on larger airways.

**Dosage :** 40 - 80 mcg by inhalation 2-4 times a day .

**Duration of action** : Short acting (4 to 6 hrs)

The above mentioned properties make ipratropium superior to other b2 agonists in decreasing dyspnoea and improves FEV1. The parasympathetic tone is the major reversible factor in COPD and hence these are more useful in COPD than in bronchial asthma .

**B) Tiotropium bromide :** Long acting (24 hrs) . Highly selective action on M1 & M3 muscarinic receptors . Dosage is 18 mcg inhalation OD .

Both the anticholinergics are not absorbed from respiratory and G.I Mucosa and has exhibited highly selective bronchial action

These are **bronchodilators of choice in COPD.**

**DELIVERY DEVICES [47] :**

**1) MDI :** metered dose inhaler which deliver same quantity of drug / puff.

**2) ROTAHALER :** rotacaps with inhaler device .

**3) MDI with face mask and spacer device** : in children who cannot be trained to use direct inhaler devices .

**Methylxanthines :**

***Mechanism of action :***

a) Release of calcium from sarcoplasmic reticulum

b) Inhibition of phosphodiesterase (PDE) which degrades cyclic nucleotides

c) Blockade of adenosine receptors ( major smooth muscle contractor )

**Side Effects :**

Headache , nervousness , nausea , narrow therapeutic margin of safety .

Rapid i.v injections result in precordial pain , syncope , even sudden deaths cause attributed to rapid fall in BP , ventricular arrhythmias , or asystole .

**Methyl xanthines cause**

- Bronchodilation ,
- Inhibition of release of inflammatory mediators ,
- Eosinophil apoptosis ,
- Improved mucociliary clearance & Respiratory drive stimulation

**ANTIBIOTICS :** Initiated in acute exacerbations whenever there is [33]

a) a change in their sputum characteristics

b) foul smelling sputum

c) voluminous sputum

d) purulent sputum

e) associated fever

f) chest discomfort . Choice based on local bacterial sensitivity and resistance patterns .

**Oral antibiotics**

* Doxycycline 100 mg bd,

* Amoxicillin / clavulanate 875/125 mg bd,

* Cefpodoxime 200 mg bd,

* **Respiratory fluoroquinolones** Ciprofloxacin 500 mg bd,

* Levofloxacin 500 mg od,

* Moxifloxacin 400 mg od.

**Intravenous Antibiotics** : These are used in case of severe infections . If Pseudomonas spp. or other Enterobactereaces spp . are suspected , consider combination therapy with antipseudomonal penicillins like

* Piperacillin + Tazobactam 4.5 gm iv $6^{th}$ hrly

* Levofloxacin 750 mg iv od

* Moxifloxacin 400 mg iv od

* Ceftrioxone 1 gm iv bd

* Cefotaxime 1 gm iv tds

* Ceftazidime 1 gm iv tds

If aspiration pneumonitis is suspected ***anaerobic coverage*** with Metronidazole 500 mg iv tds or Inj .Clindamycin 600 mg OD is given. ***Atypical organism*** coverage is by Clarithromycin 250 mg – 500 mg BD The duration of antibiotics is 5 to 7 days usually. In case of sputum Positivity 2 weeks course is needed.

In acute exacerbation of COPD particularly in patients requiring Ventilatory support the chances of ***Ventilator Associated Pneumonia (VAP)*** & ***Nosocomial pneumonia*** is high . These patients due to the associated comorbid conditions are at a higher risk of developing the same .

These sort of infections prolong the stay of the patient in the hospital and increase the duration of ventilator support and are caused by highly resistant organisms , requiring higher antibiotic usage . In India the improper usage ( both dose and duration ) of antibiotics leads to emergence of resistant strains which are difficult to control with conventional drugs .

1) Carbapenems – imepenem , meropenem , doripenem

2) Glycylcyclines - Tigecycline

3) Colistin – both inhalational and intravenously

**CORTICOSTEROIDS :**

**1) SYSTEMIC :**

a) Hydrocortisone

b) Prednisolone

**2) INHALATIONAL :**

a) Budesonide

b) Fluticasone

c) Flunisolide

d) Ciclesonide

***Mechanism of Action :***

***** reducing bronchial hyperreactivity [33],

* decreasing mucosal edema

* suppression of inflammatory response

* improving airflow, and influence airway remodeling,

* retarding disease progression , causing reduction in number of episodes of exacerbation . A shorter course of 1 to 3 weeks is sufficient , mild exacerbations is not be beneficial with steroids (oral steroids )however stable COPD doesn't require steroids .

**OXYGEN THERAPY :**

Supplemental oxygen therapy via nasal prongs and nasal mask titrated to maintain Sao2 between 90% to 94% or Pao2 between 60 - 70 mm Hg.

**MECHANICAL VENTILATION : Types** [29]

1) Invasive and 2) Noninvasive

**Indications:**

**A) Clinical criteria :**

*Severe dyspnea , with clinical signs suggestive of respiratory failure ,

*increased work of breathing , use of accessory muscles of respiration,

*paradoxical motion of the abdomen,

*shallow respiration,

* retraction of the intercostal and subcostal spaces

**B) Laboratory criteria :**

***ABG** [48] showing

a) Severe Respiratory acidosis (arterial pH $\leq$ 7.35 )

b) Severe hypercapnea ( PaCO2 $\geq$ 6.0 kPa, 45 mm Hg)

c) Severe hypoxemia

***Chest x ray** – showing signs of heart failure , ARDS , pulmonary hypertension.

**Non invasive ventilation :**[29]

**HISTORY :**

A Prototype was developed by ***Dalziel in 1832 , this led to the Drinker –Shaw iron lung in 1928.***

In 1931 ***EMERSON*** was the first to device NIV in respiratory failure for polio victims .

It refers to the method of ventilation without using an artificial invasive airway . The use of NIV has reduced the requirement of intubating the trachea with an endotracheal tube considered invasive used in home setting and in ICU

1) **Bilevel positive airway pressure (BiPAP)** -is a pressure support With two different strengths of continous postive airway pressure
2) **Proportional - Assist Ventilation : (PAV)**
3) **Continuous Positive Airway Pressure : (CPAP)**

**Delivery devices :**[45]

Orofacial and nasal masks,

Mouth pieces ,

Nasal pillows,

Total face masks ,

Helmet devices etc.

**INDICATIONS :** [47 ,50]

a) Cooperative patients (not agitated , aggressive , or poor GC)

b) Breathlessness (mild to moderate , but not in respiratory failure)

c) Tachypnoea (RR > 24/ min)

d) Accessory muscles of respiration overactivity,

e) Hypercapneic respiratory acidosis ( pH range from 7.10 to 7.35)

f) Hypoxemia (PaO2 / FIO2 < 200 mm Hg)

g) Potentially reversible causes of hypoxemia .

**CONTRAINDICATIONS :**

a) Comatose patient

b) Cardiac arrest

c) Respiratory arrest

d) Hemodynamic instability

e) Acute MI

f) GI bleeding

g) Unable to protect airway (inability to cough ,impaired gag , Depressed sensorium , lethargy , status epilepticus )

h) Upper airway obstruction (extrinsic and intrinsic compression) that comprises tumours , lymph node enlargement ,

i) Angioedema .

**Figure 16 : Patient on NIV (Non Invasive Ventilation )**

**Figure 17 :Patient on Invasive Ventilation**

**Invasive ventilation:** [29]

Endotracheal tube is used to intubate the trachea and connected to Mechanical ventilator and various modes are opted like

a) **CMV/IPPV** mode – (Controlled mandatory Ventilation)

The ventilator delivers the set rate at the set intervals with the set Tidal volume without taking into consideration the efforts of the patient and hence leads to asynchrony and requires more sedation .

b) **SIMV** mode – (Synchronised Intermittent Mandatory Ventilation)

This is a better mode of ventilation which takes into account the patients efforts and assists them and hence the tolerance is better

c) **ACMV** mode - (Assisted Control Mode of Ventilation)

All the efforts of the patient are assisted in this mode and hence the risk of barotrauma is very high

d) **CPAP mode** : (continous airway pressure )

The weaning mode or the best tolerated mode where the ventilator supports only the patients efforts and hence no barotrauma

**NEWER METHODS OF VENTILATION** [52]**:**

e) **APRV** (airway pressure release ventilation)

f) **NAVA** ( Neurally adjusted ventilator assist)

**Indications for Invasive Mechanical Ventilation :** [36]

1)Intolerance to NIV & NIV failure

2)Cardiac arrest or respiratory arrest or both

3)Loss of consciousness or deterioratating mental status

4)Gasping for air

5)Psychomotor agitation inadequately controlled by sedation

6) Massive aspiration (mendelson's syndrome)

7) Poor gag reflex leading to inability to cough out respiratory secretions

8)Heart rate < 60 per min or > 120 per min with a low GCS

9) Hemodynamic instability (shock with cold and clammy peripheries) irresponsiveness to Crystalloids , vasoactive drugs (inotropes and vasopressors)

** Ventricular arrhythmia

** Severe hypoxemia and hypoperfusion in patients unable to tolerate NIV

**RISK FACTORS REDUCTION:**

**Cessation of Smoking** [22], is the sword in abolishing COPD development . The single most important and cost effective intervention is avoidance of tobacco usage in any form inorder to reduce the risk of developing COPD and retard its progression .

**Anti smoking campaigns** [35] are held to create awareness among people , media is actively ensuring the stoppage & avoidance of smoking.

**Legislations** exist to initiate public health even to reduce passive smoking among nonsmokers by banning smoking and using any forms of tobacco in public places.

**Occupational hazards** which imposes a greater risk in work places in developing COPD even among nonsmokers can be reduced by decreasing continous exposure to organic dusts and chemicals , altered work patterns , providing protective face masks , avoidance of combustion of biogas and fossil fuels [42].

Reducing the risk from outdoor and indoor air pollutants is feasible and requires a combination of public policy and protective steps taken by individual .

• Bronchodilators are prescribed on and as-needed or on a regular basis to prevent disease progression or reduction of symptoms .

**Other Pharmacologic Treatments** : [47]

**VACCINES:** a) **Influenza** vaccination

b) **Pneumococcal polysaccharide** vaccine provide certain benefits in halting disease progression as well as decrease the number of exacerbations by decreasing the triggers (viral and bacterial)

**NON PHARMACOLOGIC THERAPY:**

Pulmonary rehabilitation as a measure of tertiary prevention vary widely but a comprehensive & holistic approach is aimed at by programs includes

a) Exercise training especially breathing exercises ,

b) Graded aerobics ,

c) Regular walking (20 min a day)

d) Cycling

e) Smoking cessation ,

f) Nutrition counseling ,

g) Health education ,

h) Life style modification . etc

**Oxygen Therapy :** Long – term O2 therapy is necessary for patients with 1) PaO2 below 55 mm of Hg SpO2 below 88%, with or without hypercapnia (PCO2 – more than 45 mm of Hg) confirmed by ABG twice during a 3 week period ;

2) PaO2 between 55 to 60 mm of Hg , SpO2 of more than 88% if there is Evidence of pedal edema , pulmonary hypertension , depicting congestive cardiac failure, polycythemia (PCV > 55%).

**VENTILATORY SUPPORT :** [29]

- Non – invasive ventilation (NIV) is used in patients with stable COPD. The combination of NIV with long - term oxygen therapy either Continuous or intermittent may be used in a selected group of patients, especially in those with increased daytime hypercapnia
- Invasive Mechanical Ventilation : used in acute episodes in ICU settings.

**Surgical Treatments**:

- **Lung Volume Reduction Surgery (LVRS)** [45]**:**

LVRS is a procedure in which diseased parts of the lung is resected to reduce hyperinflation , making respiratory muscles more effective pressure generators by improving their mechanical efficiency and

by improving pliability . LVRS increases the elastic recoil and improves peak expiratory flow rates .Prolonged survival rate and and an increased 5 yr survival rates have been ensured with LVRS on a long term followup.

**• Bronchoscopic Lung Volume Reduction (BLVR) :** [45]

It has been predicted to result in modest improvement in pulmonary function , exercise tolerance , at the cost of more frequent exacerbations of COPD , pneumonia , sepsis and hemoptysis after implantation. The optimal technique and selection of patients for BLVR needs to be mastered.

**• Lung Transplantation** [49] **:**

In very few patients with very severe COPD , lung transplantation has been shown to improve quality of life (socioeconomic improvement especially in younger individuals with severe COPD) and functional capacity thereby increasing the life expectancy leading on to growth of economy. However the limitations are the lack of donor organs and its availability and cost criterias.

**Criteria for referral for lung transplantation:**

1) COPD with a **BODE** index more than 5.

2) Recommended criteria for listing include a BODE index of 7-10 and at least 1 of the following :

a) H/O of exacerbation associated with hypercapnia [$PaCO_2$ > 45- 50 mm Hg]

b) pulmonary hypertension,

c) cor pulmonale ,

d) FEV1 < 20% predicted with either DLCO < 20%

e) homogenous pattern of emphysema.

**BULLECTOMY:** [51]

Removal of a large bulla that does not participate in active gas exchange or diffusion is done so as to decompresse the adjacent normal lung parenchyma and facilitating better lung functions.

| | 1 (mild) | 2 (moderate) | 3 (severe) | 4 (very severe) |
|---|---|---|---|---|
| $FEV_1$:FVC | <0·70 | <0·70 | <0·70 | <0·70 |
| $FEV_1$ | ≥80% of predicted | 50–80% of predicted | 30–50% of predicted | <30% of predicted or <50% of predicted plus chronic respiratory failure |
| Treatment | Influenza vaccination and short-acting bronchodilator* when needed | Influenza vaccination, short-acting and ≥1 long-acting bronchodilator* when needed; consider respiratory rehabilitation | Influenza vaccination and short-acting and ≥1 long-acting bronchodilator* when needed, inhaled glucocorticosteroid if repeated exacerbations; consider respiratory rehabilitation | Influenza vaccination and short-acting and ≥1 long-acting bronchodilator* when needed, inhaled glucocorticosteroid if repeated exacerbations, long-term oxygen if chronic respiratory failure occurs; consider respiratory rehabilitation and surgery |

GOLD=Global Initiative on Obstructive Lung Disease. *$\beta_2$ agonists or anticholinergics.

*Table:* Therapy at each stage of chronic obstructive pulmonary disease, by GOLD stage[1]

**Figure : 18 Therapy In Each Gold Stage Of Severity**

**SETTINGS :**

This was a descriptive study done on the 60 patients in Government Mohan Kumaramangalam Medical College And Hospital , Salem , TamilNadu . All participants were adults more than 30 yrs of age ,both sex A written informed consent was taken prior to inclusion in the study.

**PATIENTS :**

Out of the 60 patients 30 were in acute exacerbations with varied stages of severity admitted in our hospital medical wards and ICU and further 30 patients who were termed to be stable attending Chest clinic OP Department were evaluated.

**INCLUSION CRITERIA :**

1)All COPD patients both acute (admitted in ICU and medical wards) and stable patients (attending chest clinic OP).

2) Patients who were willing to participate in the study.

**EXCLUSION CRITERIA :**

**Patients with**

1) H/O CAHD,
2) chronic systemic illness like CKD , DCLD .
3) marked hemodynamic instability.
4) cardiac arrest on admission.

5) uncontrolled diabetes or hypertension.

6) Pregnancy,

7) prior H/O lung surgeries , genetic disorders (cystic fibrosis , alpha 1 Antitrypsin deficiency ) causing lung function compromise.

**PERIOD OF STUDY :**

**August 2014 to August 2015.**

**DATA COLLECTION METHODS :**

All patients were interviewed and examined by the investigator . A proforma which was used is enclosed in the annexure . A detailed history, symtoms , vitals RR , BP, detailed respiratory system examination was done.

**Investigations**:

1) ECG ,

2) ECHOCARDIOGRAM,

3) Xray chest,

4) PaSp ,

5) Cardiac TROPONIN-I ,

Duration of hospital stay, need for ICU care , ventilator support have been studied and stable cases attending the OP were also subjected the same.

**STATISTICAL METHODS:**

Tabulation and statistical analysis were performed using Microsoft Excel And SPSS v.17.0 software . Numerical data were summarized by measures of central tendency : mean and standard deviation . Qualitatative data was analysed with descriptive statistics & a two way univariate analysis were were used for comparing the study variables . If $p$ –value $< 0.05$ at 95% confidence intervals its taken as statistical significance.

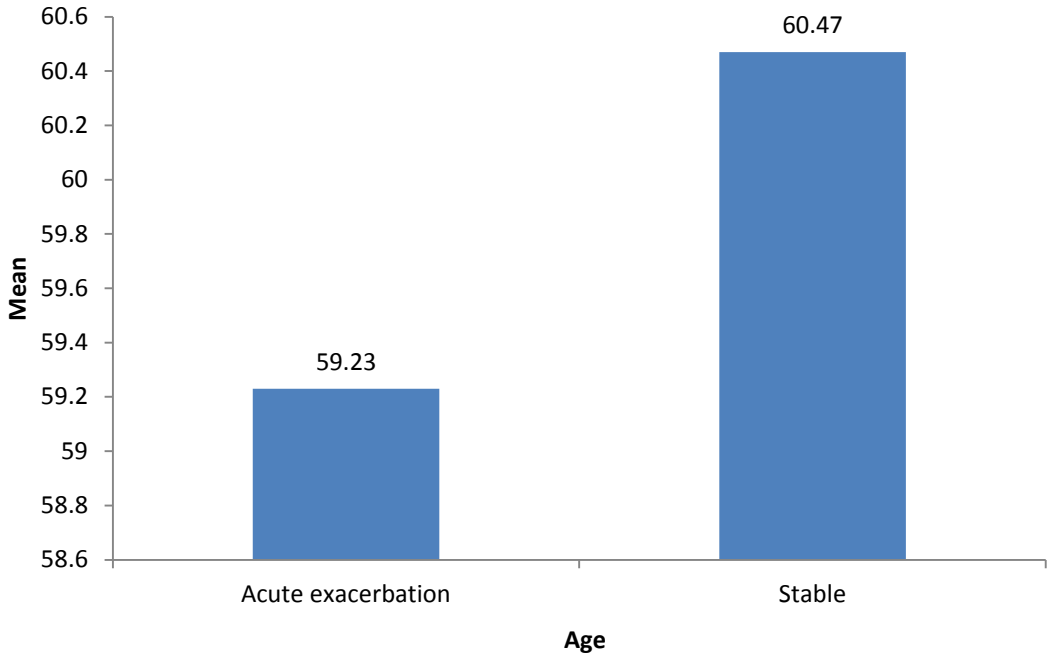
Cardiac troponin I levels were positive in acute exacerbation cases with increased cardiovascular morbidity & mortality was estimated by using ***Conditional logistic regression*** and adjusted for its covariates . Age , smoking duration , RR , BP, (both systolic and diastolic) , ECG , ECHO , PaSp, Pulmonary function tests (PEFR & FEV1/FVC ), Troponin I were analysed as important covariates .

Chi Square Test and Student T Test were performed and the results were analysed based on various covariates mentioned above and compared between the stable COPD patients and acute COPD exacerbation groups and $p$ value was determined which showed statistical significance and tabulated as follows .

## TABLE 1 :AGE COMPARISON BETWEEN TWO GROUPS

| | GROUP | N | Mean | SD | t | p |
|---|---|---|---|---|---|---|
| **Age** | **Acute exacerbation** | 30 | 59.23 | 8.56 | 0.53 | 0.600 |
| | **stable** | 30 | 60.47 | 9.50 | | |

**Figure :19 Showing Age Comparison Between Two Groups**




The **age of patients included in the study ranged from 40 to 80 years** .

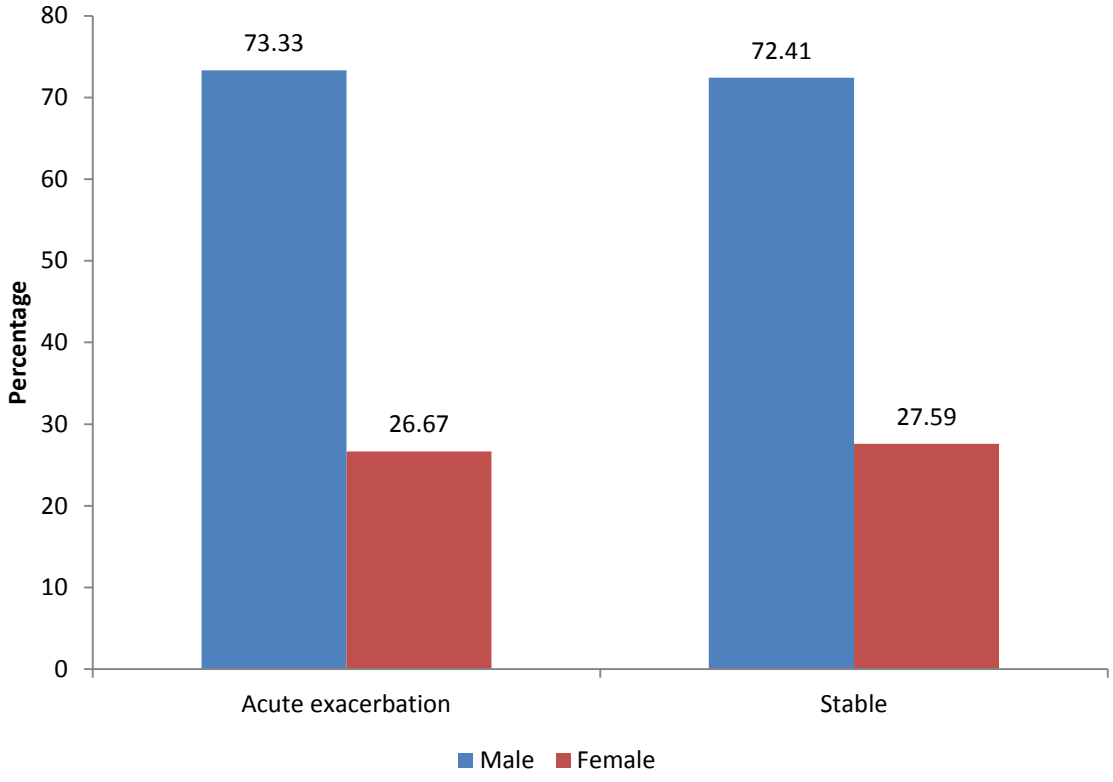
The mean age in acute group was 59.23 with a standard deviation of 8.56

The mean age in stable group was 60.47 with a standard deviation of 9.5

## TABLE 2 : COMPARISON OF SEX IN BOTH GROUPS

| Sex | Group | | | | Total | | Chi square | p |
|---|---|---|---|---|---|---|---|---|
| | Acute exacerbation | | Stable | | N | % | | |
| | N | % | N | % | | | | |
| **Male** | 22 | 73.33 | 21 | 72.41 | 43 | 72.88 | 0.01 | 0.937 |
| **Female** | 8 | 26.67 | 8 | 27.59 | 16 | 27.12 | | |
| **Total** | 30 | 100.00 | 29 | 100.00 | 59 | 100.00 | | |

**Figure 20 : Showing Sex Comparison Between Two Groups**

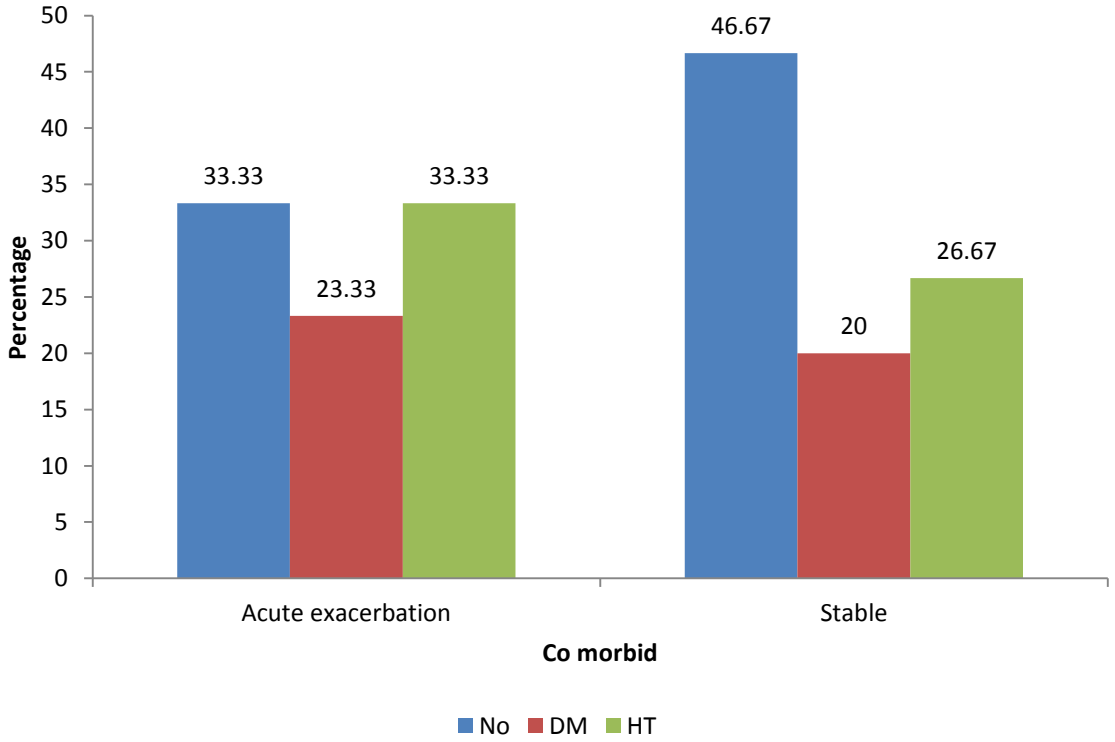



Study showed **increased prevalence of COPD in men than women** .

## TABLE 3 : COMPARISON OF COMORBIDITIES IN BOTH THE GROUPS

| Co morbid | Group | | | | Total | | Chi square | p |
|---|---|---|---|---|---|---|---|---|
| | Acute exacerbation | | Stable | | N | % | | |
| | N | % | N | % | | | | |
| **No** | 10 | 33.33 | 14 | 46.67 | 24 | 40.00 | 1.17 | 0.761 |
| **DM** | 7 | 23.33 | 6 | 20.00 | 13 | 21.67 | | |
| **HT** | 10 | 33.33 | 8 | 26.67 | 18 | 30.00 | | |
| **HT/DM** | 3 | 10.00 | 2 | 6.67 | 5 | 8.33 | | |
| **Total** | 30 | 100.00 | 29 | 100.00 | 59 | 100.00 | | |

**Figure 21 : Comparison Of Comorbid Conditions In Two Groups**

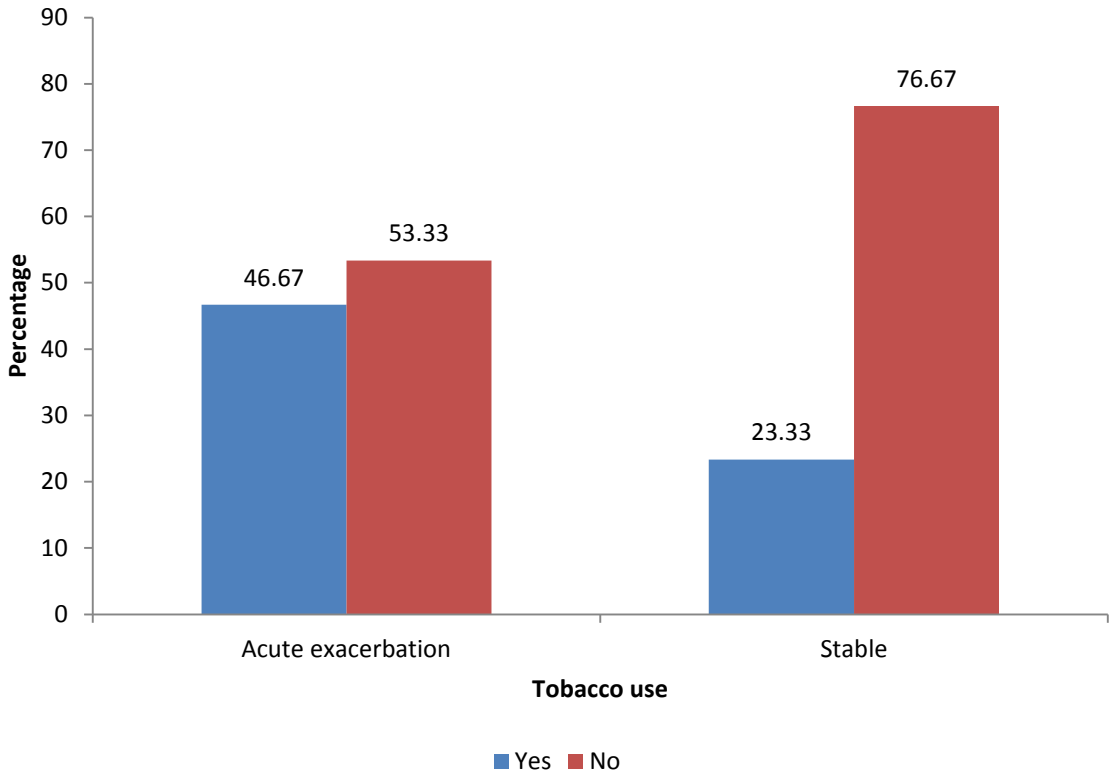



The existence of **Diabetes and Hypertension is almost the same** in both the groups and they don't contribute to acute exacerbations .

## TABLE 4 : COMPARISON OF TOBACCO USAGE

| Tobacco use | Group | | | | Total | | Chi square | p |
|---|---|---|---|---|---|---|---|---|
| | Acute exacerbation | | Stable | | N | % | | |
| | N | % | N | % | | | | |
| Yes | 14 | 46.67 | 7 | 23.33 | 21 | 35.00 | 3.59 | 0.058 |
| No | 16 | 53.33 | 23 | 76.67 | 39 | 65.00 | | |
| Total | 30 | 100.00 | 29 | 100.00 | 59 | 100.00 | | |

**Figure 22 : Comparison Of Tobacco Usage in the Two Groups**

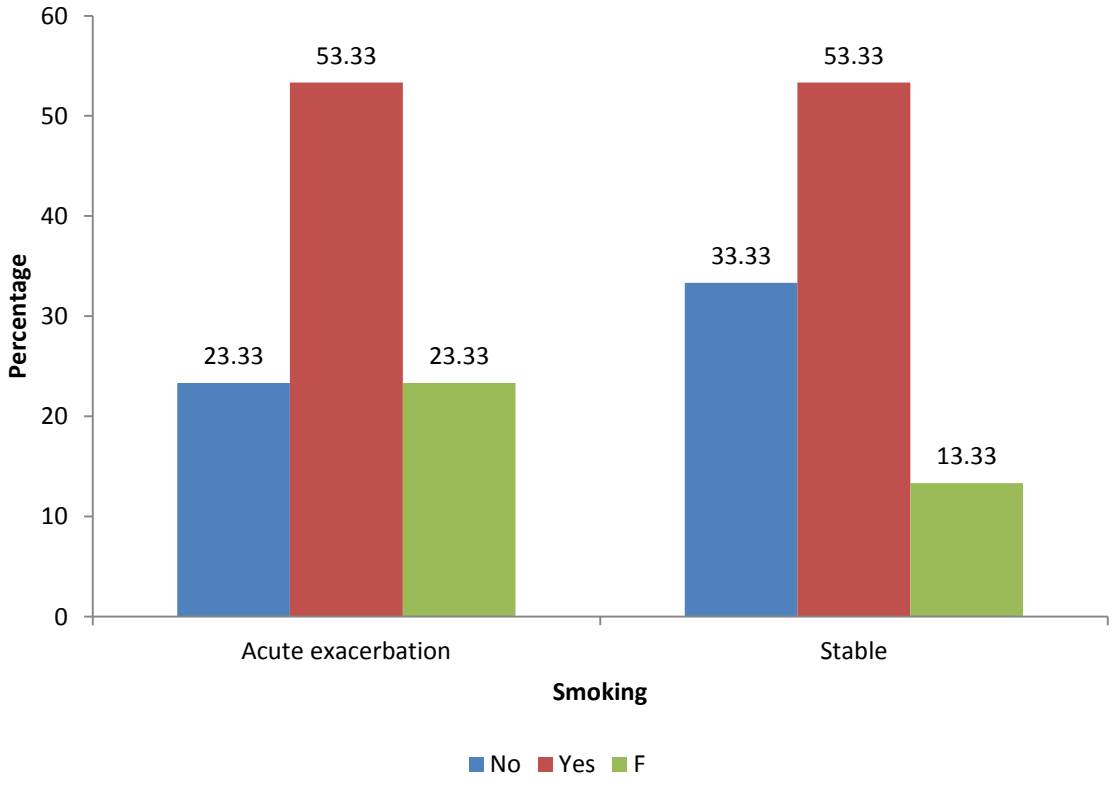



Study showed **increased morbidity in acute exacerbation** group with **response to tobacco usage** when compared to stable group whose habit of tobacco usage is less or who have quit tobacco .

## TABLE 5 : COMPARISON OF SMOKING IN BOTH GROUPS

| Smoking | Group | | | | Total | | Chi square | p |
|---|---|---|---|---|---|---|---|---|
| | Acute exacerbation | | Stable | | N | % | | |
| | N | % | N | % | | | | |
| **No** | 7 | 23.33 | 10 | 33.33 | 17 | 28.33 | 1.35 | 0.510 |
| **Yes** | 16 | 53.33 | 16 | 53.33 | 32 | 53.33 | | |
| **F** | 7 | 23.33 | 4 | 13.33 | 11 | 18.33 | | |
| **Total** | 30 | 100.00 | 29 | 100.00 | 59 | 100.00 | | |

**Figure 23 : Comparison of Smoking**

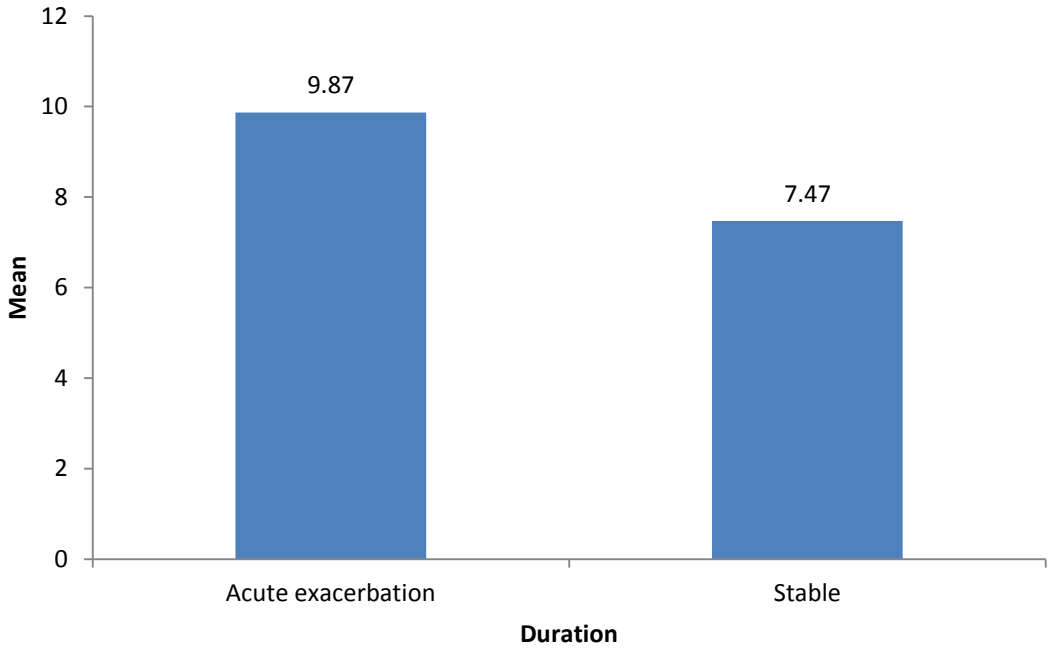



Study showed **direct correlation of smoking risk in COPD in men in both groups equally** , with slight decline in stable group in females

**TABLE 6 : COMPARISON OF DURATION OF DISEASE**

| | GROUP | N | Mean | SD | t | P |
|---|---|---|---|---|---|---|
| **DURATION** | **Acute exacerbation** | 30 | 9.87 | 5.17 | 2.00 | 0.05* |
| | **Stable** | 30 | 7.47 | 4.06 | | |

**Figure 24 : Duration of Disease in Both Groups**

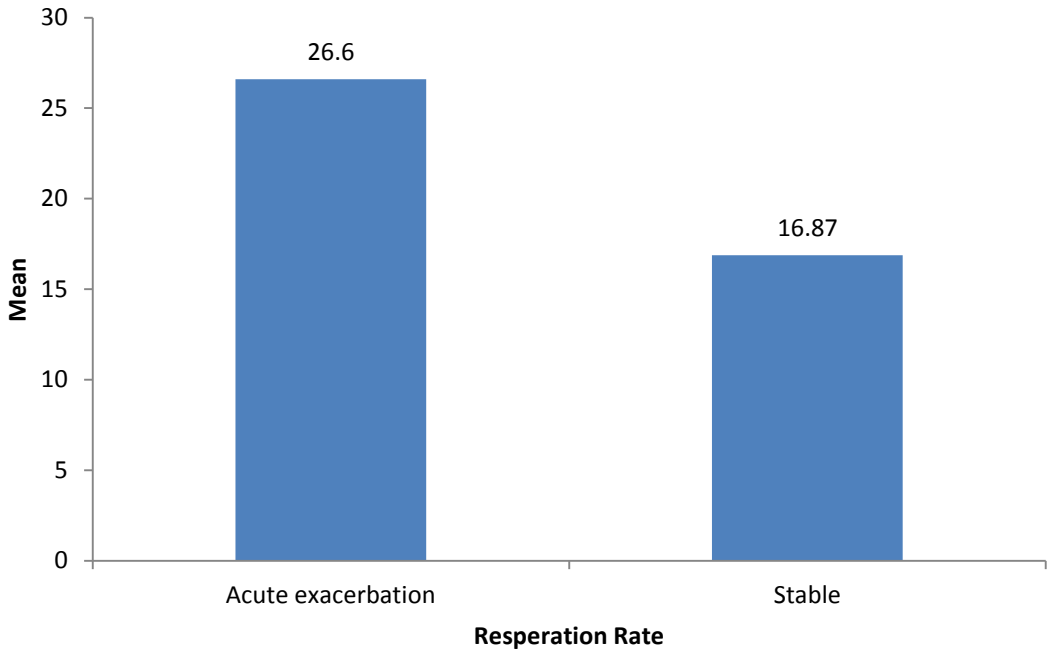



The **duration of disease is prolonged in acute** group than stable group which endorses that most of the at risk COPD patients who have milder symptoms don't require drugs and are no longer diagnosed as COPD .

**TABLE 7 : COMPARISON OF RESPIRATORY RATE**

| | GROUP | N | Mean | SD | t | P |
|---|---|---|---|---|---|---|
| **Respiratory Rate** | **Acute exacerbation** | 30 | 26.60 | 3.33 | 14.64 | < 0.001** |
| | **Stable** | 30 | 16.87 | 1.48 | | |

**Figure 25 : Respiratory Rate Variation In Both Groups**

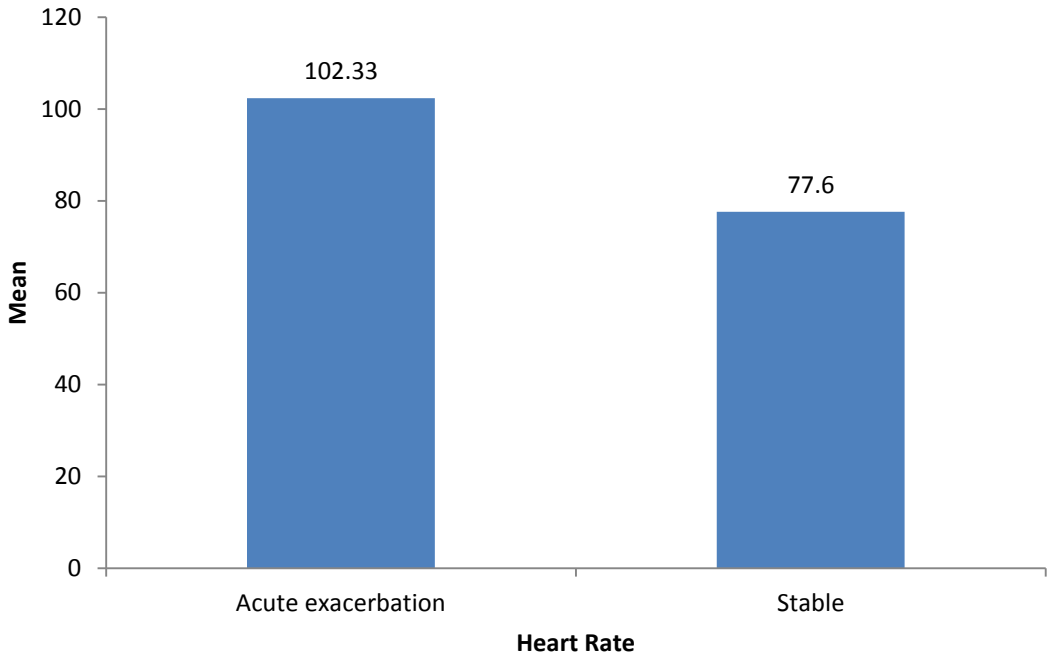



The **respiratory rate in acute group is higher** with a mean of 26.60 and a SD of 3.33 than the stable group with a mean of 16.87 and a SD of 1.48 with a *p* value of 0.001 which is **statistically significant** .

## TABLE 8 :COMPARISON OF HEART RATE IN BOTH THE GROUPS

| | GROUP | N | Mean | SD | t | P |
|---|---|---|---|---|---|---|
| **Heart Rate** | **Acute exacerbation** | 30 | 102.33 | 8.93 | 11.80 | <0.001** |
| | **Stable** | 30 | 77.6 | 7.21 | | |

**Figure 26 : Heart Rate Variablity In Both the Groups**

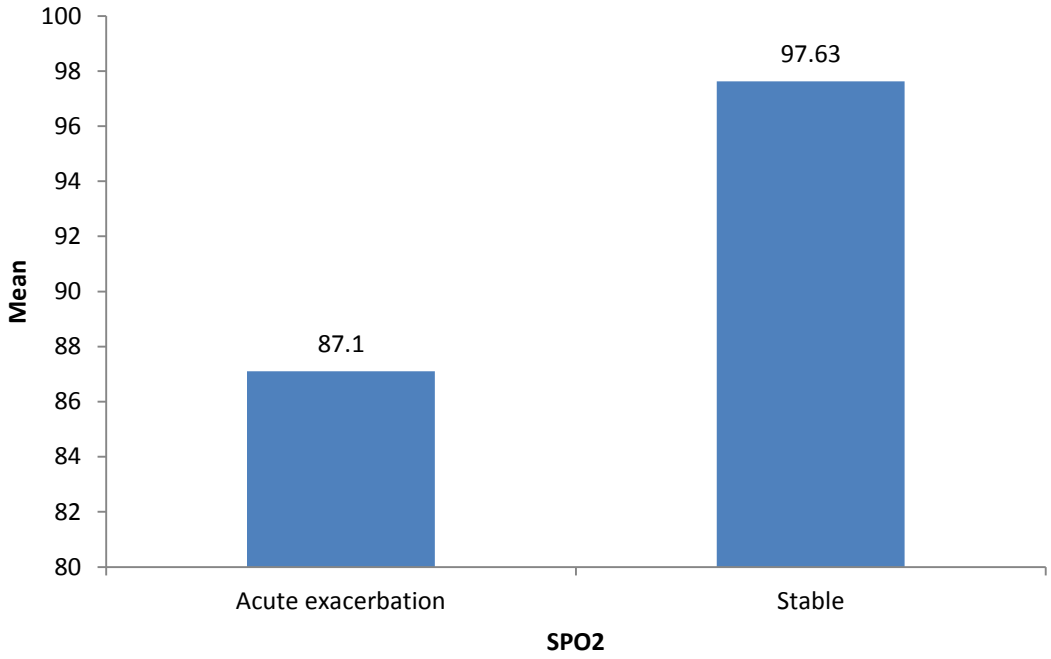



On comparison **acute group patients have more tachycardia** than stable Group .

## TABLE 9 : COMPARISON OF OXYGEN SATURATION

| | GROUP | N | Mean | SD | t | P |
|---|---|---|---|---|---|---|
| **Spo2** | **Acute exacerbation** | 30 | 87.10 | 8.84 | 6.43 | <0.001** |
| | **Stable** | 30 | 97.63 | 1.56 | | |

**Figure 27 : SpO2 Comparison in Both Groups**



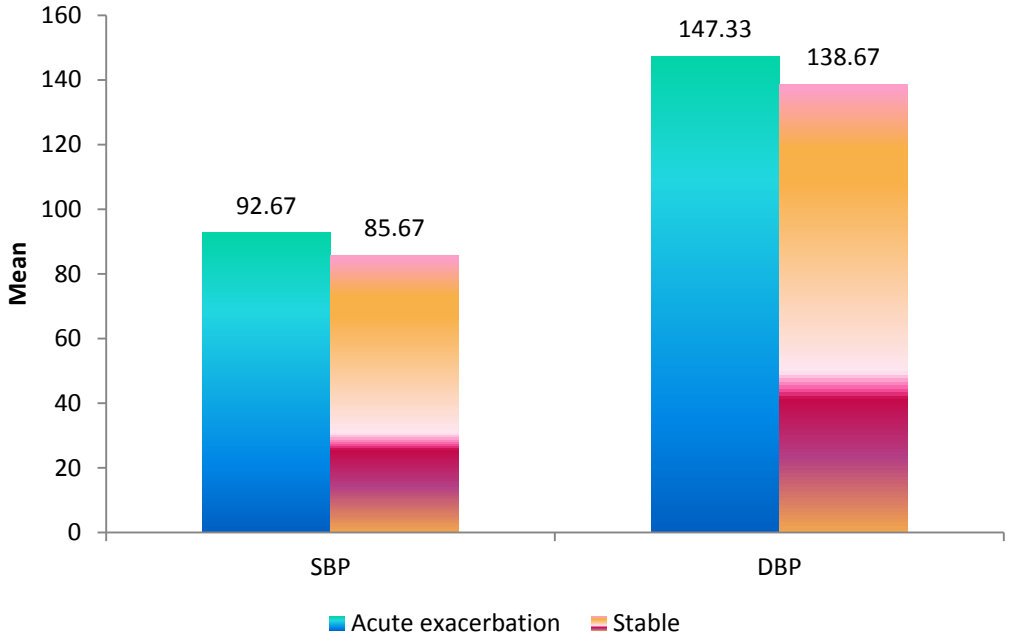

**Oxygen saturation** as measured by SpO2 is **significantly lower in acute** group than stable group .

## TABLE 10 : COMPARISON OF BLOOD PRESSURE

| | GROUP | N | Mean | SD | t | P |
|---|---|---|---|---|---|---|
| **Systolic BP** | **Acute exacerbation** | 30 | 147.33 | 16.39 | 2.20 | 0.032* |
| | **Stable** | 30 | 138.67 | 14.08 | | |

| | GROUP | N | Mean | SD | t | P |
|---|---|---|---|---|---|---|
| **Diastolic BP** | **Acute exacerbation** | 30 | 92.67 | 11.72 | 2.38 | 0.021* |
| | **Stable** | 30 | 85.67 | 11.04 | | |

**Figure 28 : SBP & DBP Variation in Both Groups**

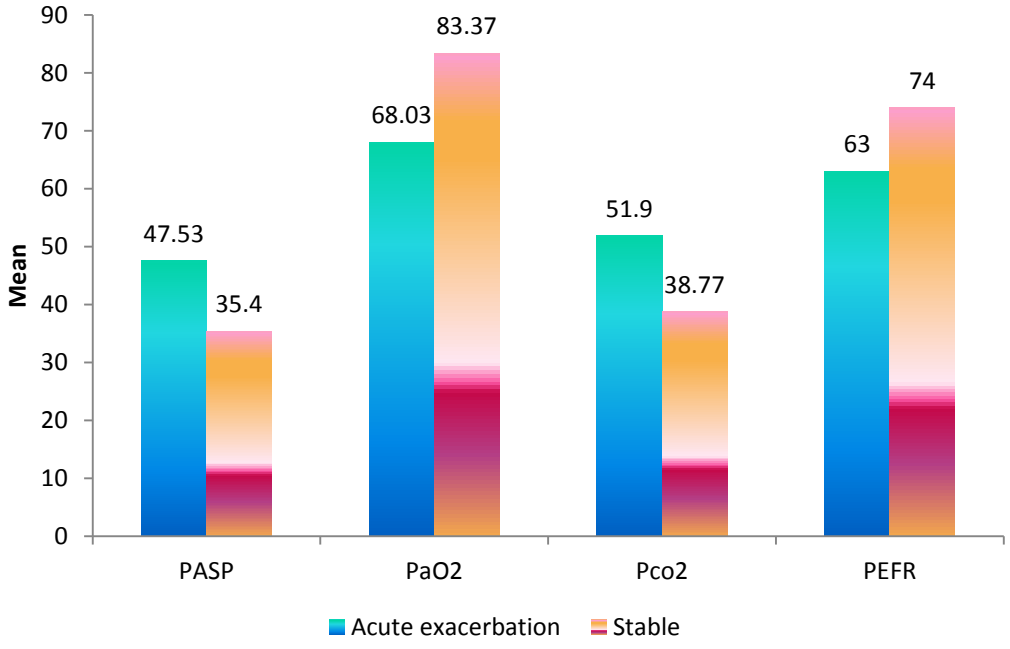



Comparison of **blood pressure doesn't contribute to statistical significance except for it was higher in acute group than stable group .**

## TABLE 11 : COMPARISON OF PaSP, PaO2 , PaCO2 , PEFR

| | GROUP | N | Mean | SD | t | P |
|---|---|---|---|---|---|---|
| **Pasp** | **Acute exacerbation** | 30 | 47.53 | 8.16 | 7.09 | <0.001** |
| | **Stable** | 30 | 35.40 | 4.62 | | |
| **PaO2** | **Acute exacerbation** | 30 | 68.03 | 8.81 | 7.84 | <0.001** |
| | **Stable** | 30 | 83.37 | 6.11 | | |
| **PaCO2** | **Acute exacerbation** | 30 | 51.90 | 8.92 | 7.61 | <0.001** |
| | **Stable** | 30 | 38.77 | 3.11 | | |
| **PEFR** | **Acute exacerbation** | 30 | 63.00 | 7.50 | 6.69 | <0.001** |
| | **Stable** | 30 | 74.00 | 4.98 | | |

**Figure 29 : Comparison of PaSP , PaO2 , PCO2 , PEFR**

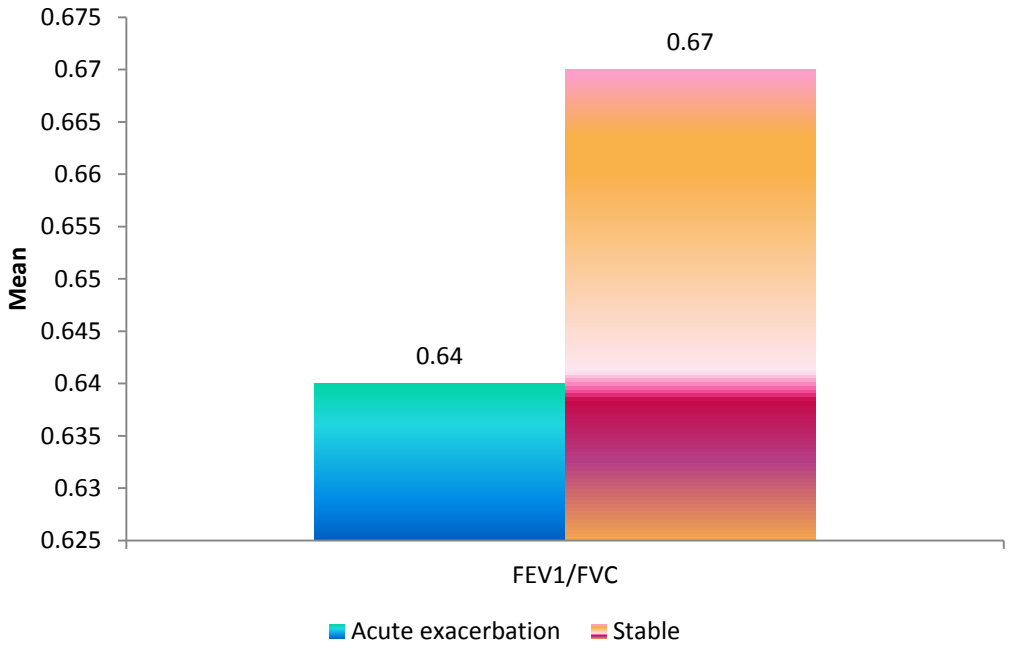


## TABLE 12 : COMPARISON FEV1/FVC IN BOTH THE GROUPS

| | GROUP | N | Mean | SD | t | P |
|---|---|---|---|---|---|---|
| **FEV1/FVC** | **Acute exacerbation** | 30 | 0.64 | 0.06 | 1.91 | 0.061 |
| | **Stable** | 30 | 0.67 | 0.03 | | |

| | GROUP | N | Mean | SD | t | P |
|---|---|---|---|---|---|---|
| **HOSPITAL STAY** | **Acute exacerbation** | 30 | 7.47 | 3.15 | - | - |
| | **Stable** | 30 | nil | nil | | |

**Figure 30 :FEV1/FVC Ratio in Both the Groups**

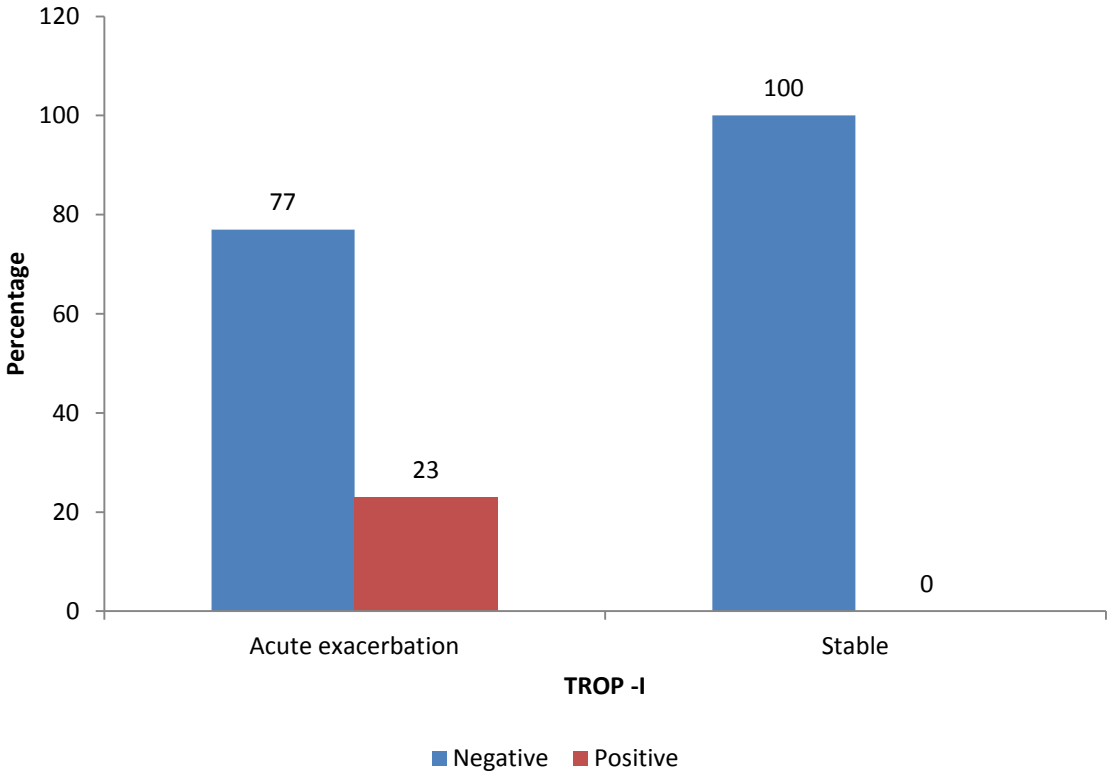



The ratio of **FEV1/FVC is lower in acute exacerbation** group than stable group and is **statistically significant** .

## TABLE 13 :COMPARISON OF TROPONIN –I IN BOTH GROUPS

| Group | TROP -I | | | | Total | Chi square | p |
|---|---|---|---|---|---|---|---|
| | Negative | | Positive | | | | |
| | N | % | N | % | | | |
| Acute exacerbation | 23 | 77 | 7 | 23 | 30 | 7.92 | 0.005** |
| Stable | 30 | 100 | - | - | 30 | | |
| Total | 53 | 88 | 7 | 12 | 60 | | |

**Figure 31 : Comparison of Troponin I Positivity Between The Groups**



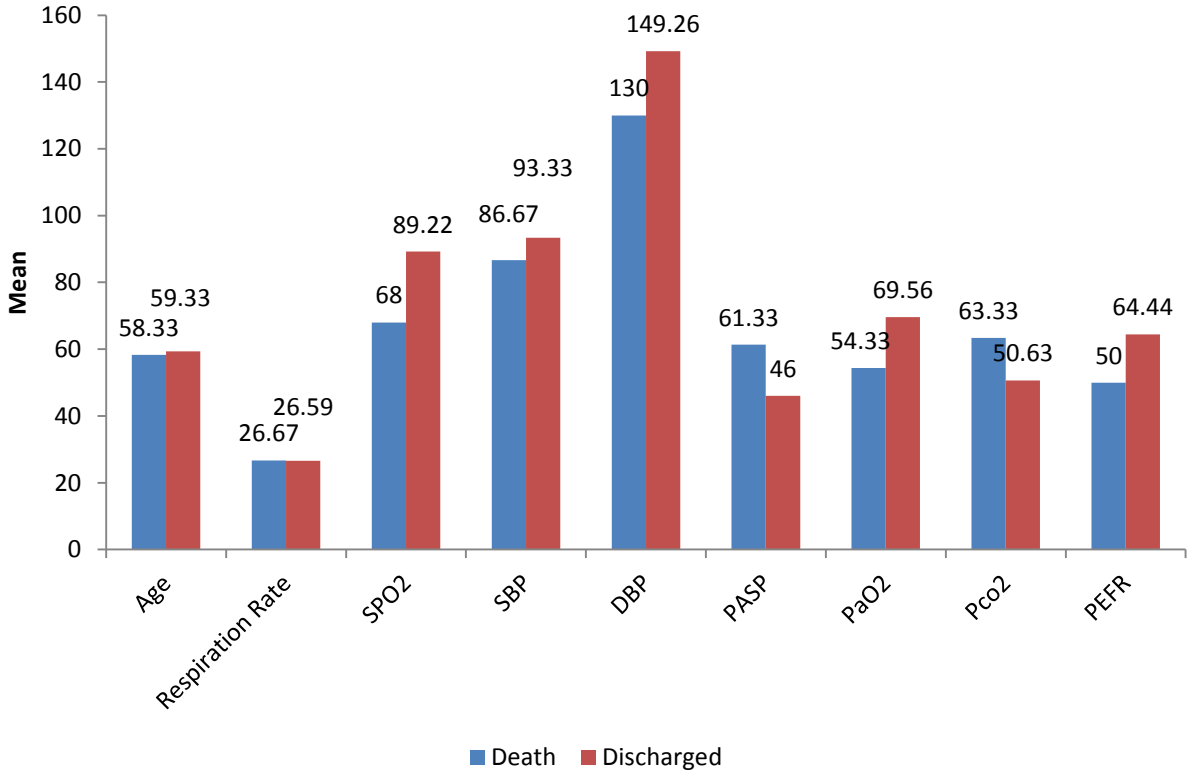

**Troponin I was positive in acute group and is statistically significant**

## TABLE 14 :

## COMPARISON OF ALL VARIABLES BETWEEN THOSE DISCHARGED AND DIED IN ACUTE EXACERBATION GROUP

| | Outcome | N | Mean | SD | t | p |
|---|---|---|---|---|---|---|
| **Age** | Death | 3 | 58.33 | 18.93 | 0.19 | 0.852 |
| | Discharged | 27 | 59.33 | 7.36 | | |
| **Duration** | Death | 3 | 12.33 | 11.02 | 0.87 | 0.393 |
| | Discharged | 27 | 9.59 | 4.44 | | |
| **Respiration Rate** | Death | 3 | 26.67 | 4.16 | 0.04 | 0.972 |
| | Discharged | 27 | 26.59 | 3.32 | | |
| **Heart Rate** | Death | 3 | 114.67 | 9.02 | 2.80 | 0.009** |
| | Discharged | 27 | 100.96 | 7.95 | | |
| **SPO2** | Death | 3 | 68.00 | 2.00 | 5.69 | < 0.001** |
| | Discharged | 27 | 89.22 | 6.33 | | |
| **SBP** | Death | 3 | 86.67 | 15.28 | 0.93 | 0.359 |
| | Discharged | 27 | 93.33 | 11.44 | | |
| **DBP** | Death | 3 | 130.00 | 17.32 | 2.03 | 0.052 |
| | Discharged | 27 | 149.26 | 15.42 | | |
| **PASP** | Death | 3 | 61.33 | 4.16 | 3.71 | 0.001** |
| | Discharged | 27 | 46.00 | 6.96 | | |
| **PaO2** | Death | 3 | 54.33 | 10.69 | 3.28 | 0.003** |
| | Discharged | 27 | 69.56 | 7.32 | | |
| **Pco2** | Death | 3 | 63.33 | 2.31 | 2.55 | 0.016* |
| | Discharged | 27 | 50.63 | 8.46 | | |
| **PEFR** | Death | 3 | 50.00 | 0.00 | 3.85 | 0.001** |
| | Discharged | 27 | 64.44 | 6.41 | | |
| **FEV1/FVC** | Death | 3 | 0.55 | 0.05 | 3.52 | 0.001** |
| | Discharged | 27 | 0.65 | 0.05 | | |
| **Hospital stay** | Death | 3 | 7.00 | 2.65 | 0.27 | 0.792 |
| | Discharged | 27 | 7.52 | 3.24 | | |

**Figure 32 :Bar Diagram showing Comparison of All Variables in Both Groups**




The study showed a **statistically significant association** of the following variables like **heart rate , PaSP , PaCO2 , which was higher in acute group** than stable group , and the variables like **SpO2 , PaO2 , PEFR , FEV1/FVC were lower in acute group** which also exhibited a statistical significance .

**TABLE 15 :**
**COMPARISON OF RISK FACTORS IN ACUTE EXACERBATION**

| | | Outcome | | | | Total | Chi square | p |
|---|---|---|---|---|---|---|---|---|
| | | Death | | Discharged | | | | |
| | | N | % | N | % | | | |
| Sex | Male | 1 | 4.55 | 21 | 95.45 | 22 | 2.73 | 0.099 |
| | Female | 2 | 25.00 | 6 | 75.00 | 8 | | |
| Tobacco use | Yes | 1 | 7.14 | 13 | 92.86 | 14 | 0.24 | 0.626 |
| | No | 2 | 12.50 | 14 | 87.50 | 16 | | |
| Smoking | No | 2 | 28.57 | 5 | 71.43 | 7 | 4.60 | 0.100 |
| | Yes | | | 16 | 100.00 | 16 | | |
| | Former smoker | 1 | 14.29 | 6 | 85.71 | 7 | | |
| Co morbid | No | 3 | 30.00 | 7 | 70.00 | 10 | 6.67 | 0.083 |
| | DM | | | 7 | 100.00 | 7 | | |
| | HT | | | 10 | 100.00 | 10 | | |
| | HT/DM | | | 3 | 100.00 | 3 | | |
| Total | | 3 | 10.00 | 27 | 90.00 | 30 | | |

**The above mentioned risk factors like smoking , tobacco usage , comorbid** conditions, which were included in the study **were increased in acute exacerbation group** that predisposes to further exacerbations .

**Male gender is itself a risk factor** for COPD .

**Figure 33 : Comparison of Smoking in Death & Discharged Patients**

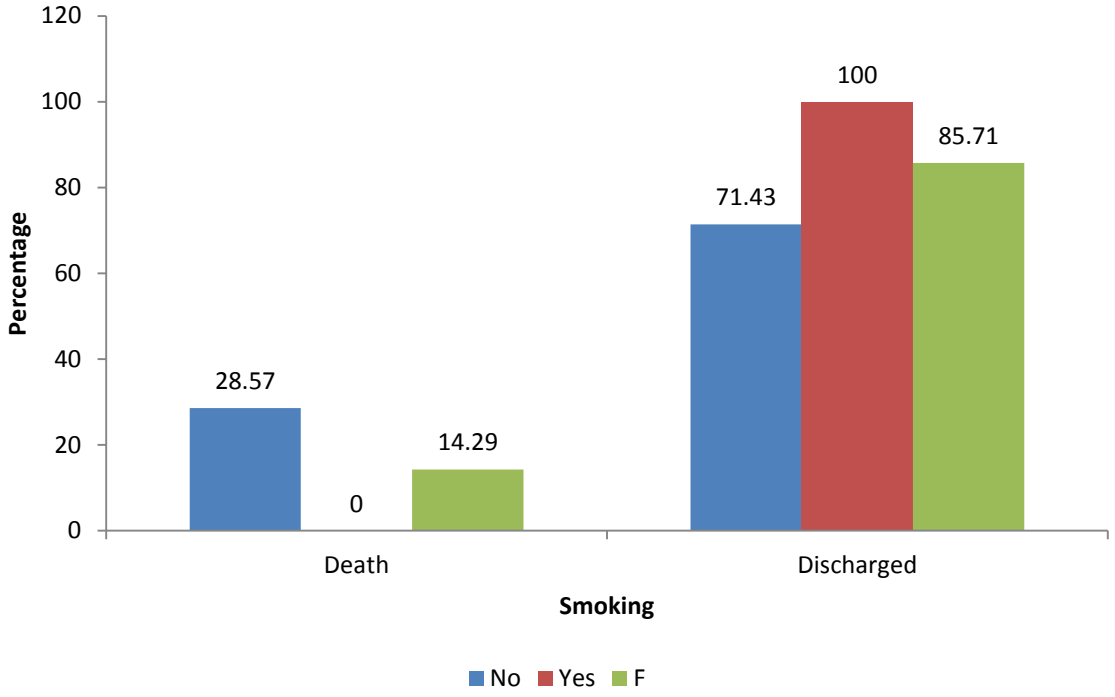
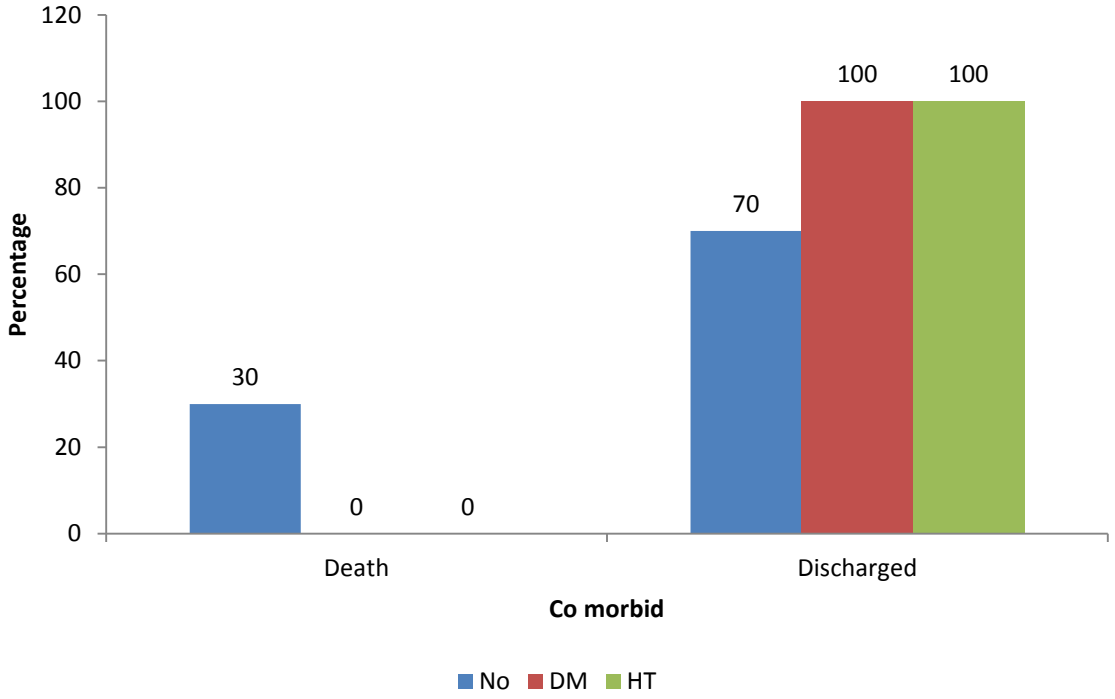


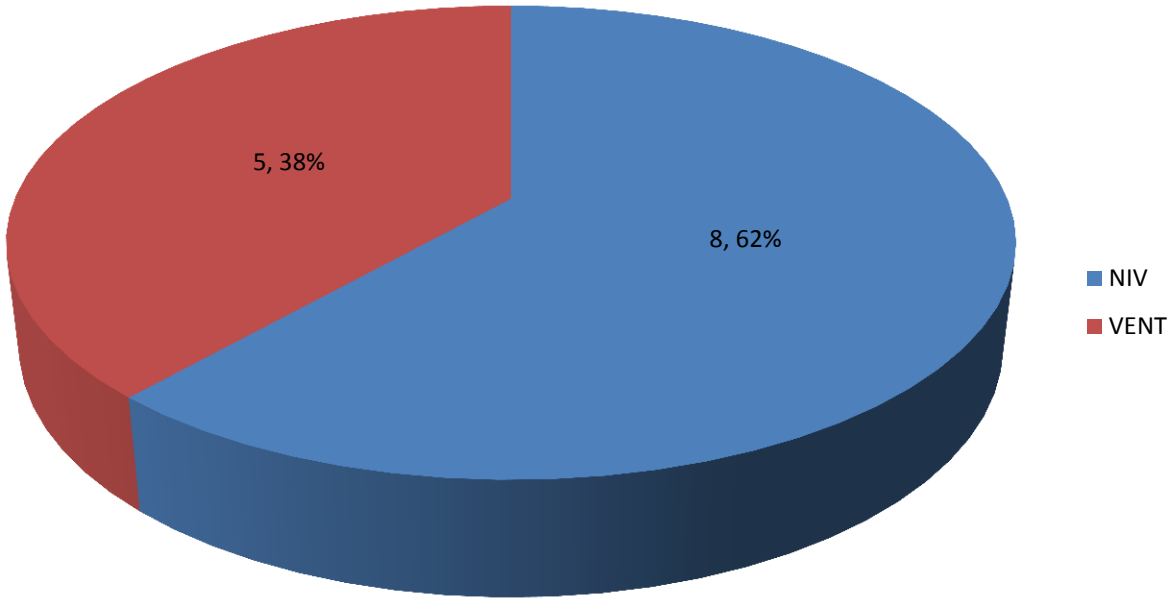

**Figure 34 : Comparison of Comorbidities in Death & Discharged Patients**



## TABLE 16 :COMPARISON BETWEEN TYPE OF VENTILATION USED

| Type of ventilation | Frequency | Percent | Valid Percent |
|---|---|---|---|
| **NIV** | 8 | 27 | 62 |
| **VENT** | 5 | 17 | 38 |
| **Total** | 13 | 43 | 100 |
| **System** | 17 | 57 | |
| **Total** | 30 | 100 | |

**Figure 35 : Type Of Ventilation Used**

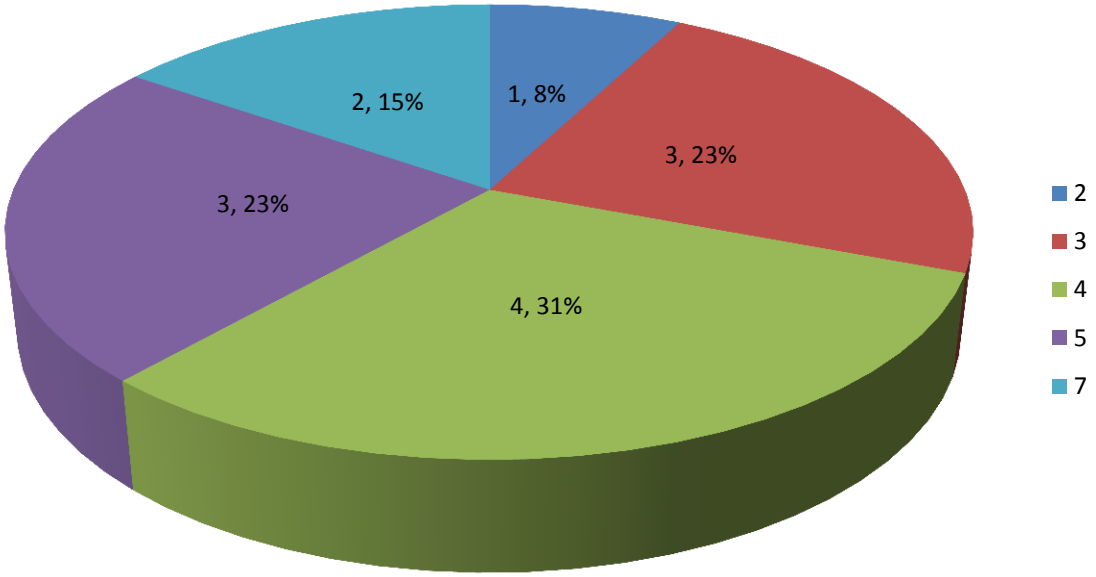



**NIV (non invasive ventilation ) is more frequently needed than invasive** ventilation which increases the patient morbidity and prolongs the length of hospital stay and decreases the chance of ventilator associated pneumonia as well as nosocomial infections which further leads to deterioration of acute exacerbation .

## Table 17 : Percentage Of Patients On The Basis Of Ventilator Days:

| Days on ventilation | Frequency | Percent | Valid Percent |
|---|---|---|---|
| **2** | 1 | 3 | 8 |
| **3** | 3 | 10 | 23 |
| **4** | 4 | 13 | 31 |
| **5** | 3 | 10 | 23 |
| **7** | 2 | 7 | 15 |
| **Total** | 13 | 43 | 100 |
| **System** | 17 | 57 | |
| | 30 | 100 | |

**Figure 36 : Pie Chart Showing Ventilator Dependant Days**

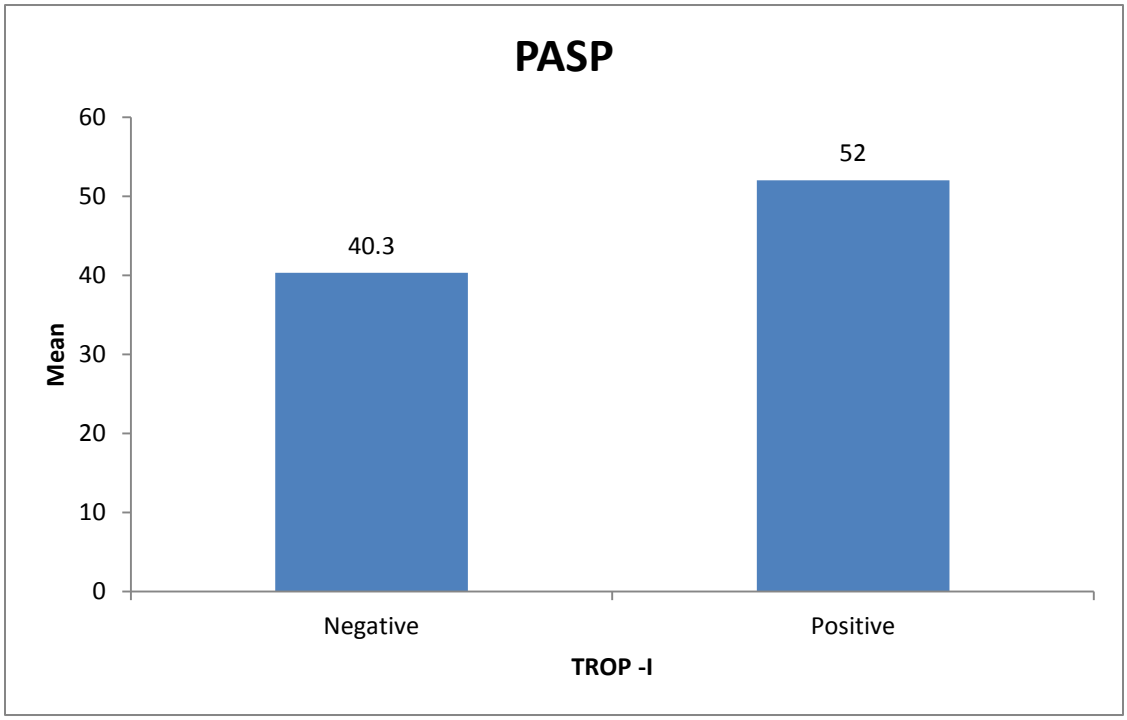



Out of the 60 patients 21 required ventilation support and their percentage dependant days are shown above .

## TABLE 18 : COMPARISON OF TROPONIN I POSITIVITY & PASP

| | TROP -I | N | Mean | SD | t | p |
|---|---|---|---|---|---|---|
| **Pasp** | **Negative** | 24 | 40.3 | 8.23 | 3.27 | 0.002** |
| | **Positive** | 6 | 52 | 9.23 | | |

**Figure 37 : Bar Diagram Showing PaSP & TROPONIN I positivity**

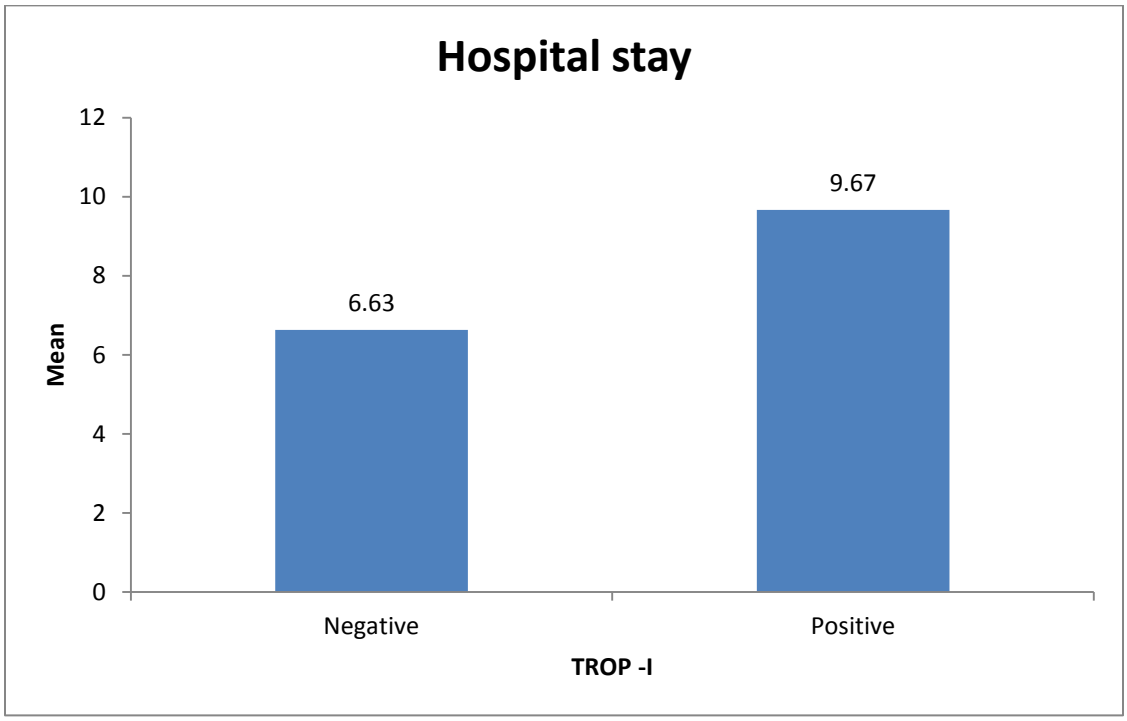



**In patients with troponin I positivity the pulmonary artery Pressures were higher compared to patients with negative Troponin I levels.**

**TABLE 19 : COMPARISON OF TROPONIN I POSITIVITY & DURATION OF HOSPIAL STAY**

| | TROP -I | N | Mean | SD | t | p |
|---|---|---|---|---|---|---|
| **Hospital stay** | **Negative** | 24 | 6.63 | 2.84 | 2.34 | 0.027** |
| | **Positive** | 6 | 9.67 | 2.88 | | |

**Figure 38 : Comparison of TROPONIN I positivity & hospital stay**

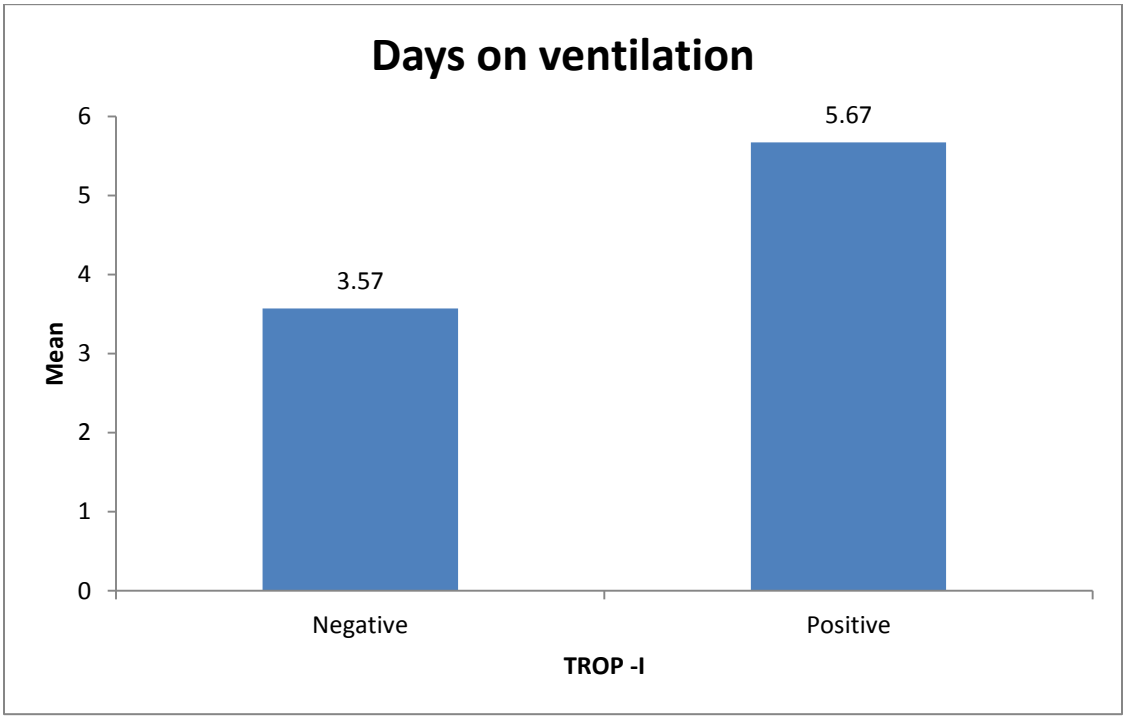



**Mean duration of hospital stay is higher in patients with Troponin I Positivity and is statistically significant.**

## TABLE 20 :TROPONIN I POSITIVITY & DURATION OF VENTILATION

| | TROP -I | N | Mean | SD | t | p |
|---|---|---|---|---|---|---|
| **Days on ventilation** | **Negative** | 7 | 3.57 | 0.98 | 2.86 | 0.015* |
| | **Positive** | 6 | 5.67 | 1.63 | | |

**Figure 39 : Troponin I positivity & Ventilation Dependant Days**

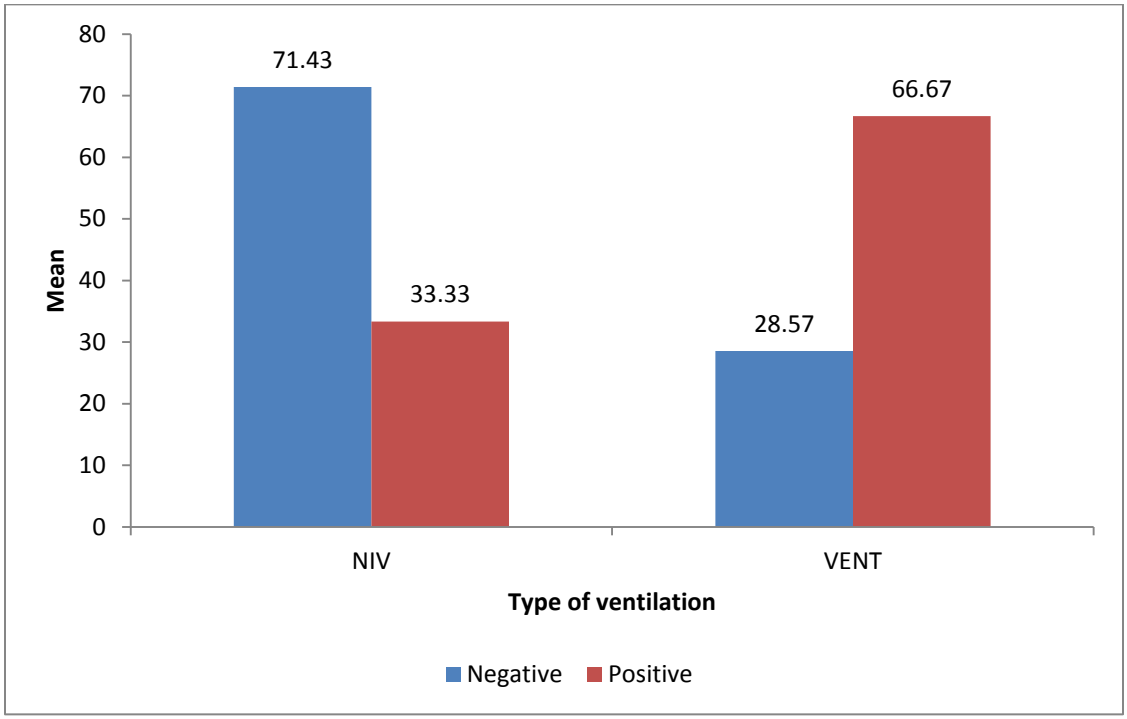



The study showed that **the mean duration of ventilation in Troponin I positive patients with COPD exacerbation is higher and Is statistically significant**

## TABLE 21 :TYPE OF VENTILATION IN TROPONIN I POSITIVE PATIENTS

| TROP -I | Type of ventilation | | | | Total |
|---|---|---|---|---|---|
| | NIV | | VENT | | |
| | N | % | N | % | |
| **Negative** | 5 | 71.43 | 2 | 28.57 | 7 |
| **Positive** | 2 | 33.33 | 4 | 66.67 | 6 |
| **Total** | 7 | 53.85 | 6 | 46.15 | 13 |

**Figure 40 : Troponin I positivity & Type of Ventilation Used**

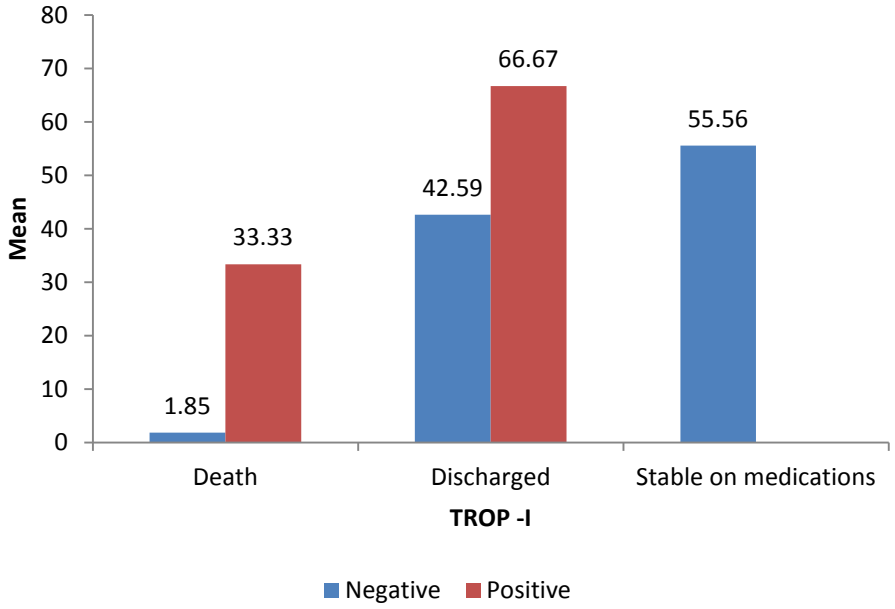



**Troponin I positive patients needed invasive ventilation more than NIV .**

## TABLE 22 :TROPONIN I POSITIVITY & ITS SIGNIFICANCE IN THE OUTCOME OF THE PATIENT

| TROP -I | Outcome | | | | | | Total | Chi square | p |
|---|---|---|---|---|---|---|---|---|---|
| | Death | | Discharged | | Stable on medications | | | | |
| | N | % | N | % | N | % | | | |
| **Negative** | 1 | 1.85 | 23 | 42.59 | 30 | 55.56 | 54 | 14.73 | 0.001** |
| **Positive** | 2 | 33.33 | 4 | 66.67 | | | 6 | | |
| **Total** | 3 | 5 | 27 | 45 | 30 | 50 | 60 | | |

**Figure 41 : Comparison of Outcomes In Troponin Positive Patients**




In the **acute exacerbation of COPD GROUP** the relationship Between **Troponin I positivity** and **death** is directly related and **Is statistically significant.**

# DISCUSSION

The study group comprised of a total of 60 patients out of which 30 patients were in acute exacerbation group who were admitted in medical wards and intensive care unit and the remaining 30 patients were selected randomly from the out patient department who were attending the chest clinic and were assigned to be the stable group.In acute group out of the 30 , 22 patients were male and 8 were female and in the stable group out of the 30 ,8 were female and 22 were male inorder to avoid gender bias and to eliminate the bias based on confounding factor variable like smoking and tobacco use.

Both the groups were matched for age and gender. Both the groups were subjected to the same set of questionnaire by means of a consistant proforma that is attached at the end of the study along with certain investigations , lab tests , imaging (X RAY chest ) ,ECG , ECHO ,ABG , PFT (PEFR ,FEV1/FVC) , PaSP , need for ventilation ,duration of hospital stay and the outcome was analysed on a statistical basis and results were obtained and tabulated .

According to the study and after analysis using statistical method the following data is obtained and based on that the aims and objectives mentioned are studied .

The patients selected were in the age ranging from 40 to 80 years ,with a mean age of 59.23 in the acute group and 60.47 in the stable group. Smoking and tobacco usage as risk factors were significantly associated with both groups , and among the comorbidconditions like hypertension and diabetes except for that it is increased in acute episodes than in the stable group , was not statistically significant .Vitals were unstable in acute exacerbation group like heart rate , respiratory rate ,with a decline in the arterial oxygen saturation as measured by SpO2 .

**The pulmonary pressures (PaSP ) and**

**PaCO2 , were increased** whereas

**PO2 , PEFR , were decreased in acute** group as against stable and was **statistically significant with a *p* value < 0.005** . Chamber dilatations and LV dysfunction as measured by **left ventricular ejection** fraction and **ECG abnormalities** were present in **acute group** and explains the **cardiovascular morbidity** thereby contributing to increased number of **hospital stay ,oxygen dependant days and also leading to mortality . FEV1/FVC ratio was significantly reduced in acute group** .Coming to the main aim of the study is that the association of **TROPONIN I positivity** from venipuncture ( blood ) done on both the groups none of the stable cases came out to be positive whereas , in acute group 7 cases out of 30 were positive which had **significant risk in the outcome** as evidenced by

increased need for oxygen both on a quantitative and qualitative basis , increased need for mechanical ventilation i.e. more patients went through NIV rathor than invasive ventilation with an endotracheal intubation , more hospitalised days had a significant association based on statistical analysis with a ***p*** value <0.005 .

In the **acute group 3 deaths occurred because of cardiorespiratory arrest that too in the Troponin I positive cases which was again significant**. Thus the aim and the objectives of the study is met with.

Thus cardiac TROPONIN I was positive in 23.33 % which is having statistical significance in causing cardiovascular morbidity and mortality , and it was comparable with other studies done in other parts of the world like

** Baillard et al ,

** Martin et al ,

** Hoiseth et al,

** Chang et al which has comparable evidence consistent with our study .

**LIMITATIONS OF THE STUDY :**

The patients were not followed up for prolonged periods in terms of further cardiovascular compromises if they had any .

Arterial blood gas analysis was delayed in considerable number of patients because ofsevere dyspnoea and hypoxemia and time was taken for initial stabilisation of the patient that was the cause of delay in ABG .

The number of patients were 60 totally which was a meager one when compared to other studies .

# SUMMARY

The study group consisted of a total of 60 subjects ,30 subjects in the acute exacerbation group ,30 subjects were in the stable group. Average that is mean age in acute group was 59.23 ,and mean age in stable group was 60.47 .All were age and sex matched .

Blood levels of Troponin I levels was studied in all the 60 subjects and were studied for cardiovascular morbidity and mortality by varied covariates like history of smoking or tobacco usage and its duration with vitals and imaging along with ECG and ECHO, PFT, and the results of the study showed increased need of oxygen , artificial ventilation ,number of hospital admission days , in the acute group especially who were Troponin I positive .

**COMPOSITE ANALYSIS :**

Of various parameters studied in relation with **Troponin I Positivity correlating with cardiovascular morbidity and mortality** shows a **statistical significance with a *p* value < 0.05 .**

# CONCLUSION

** Cardiac TROPONIN I was positive in a significant number of patients out of the small subset of patients i.e. a total of 60.

**Smoking and tobacco usage was an independent risk factor of COPD .

**Troponin I positivity predicted the increased need for ICU stay ,and hospitalization .

**Further cardiovascular burden was evaluated under the heads of vitals , imaging ,echocardiography ,ECG ,PaSP ,PaCO2 ,PO2 ,and all the variables proved longer duration of COPD , increased prevalence of IHD .

**Comorbid conditions like diabetes ,hypertension did not either have any significance in acute exacerbation or contributed to mortality according to the study except it was elevated in acute group .

**Mortality and ventilator necessity was more in acute group in particular Troponin I positive patients which proved statistical significance .

**Altogether the socioeconomic burden is dependant on the risk factor reduction , a pollution free atmosphere , prompt identification of triggers of COPD ,effective treatment , thereby preventing acute exacerbations and decreasing the cardiovascular morbidity and mortality heading on to the goal of **" HEALTH FOR ALL ".**

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# PROFORMA

**Name :** **Age :** **Sex :**

**Address :** **IP Number :**

**Chief Complaints :** **Yes** **No** **Duration**

**1)Breathlessness :**

**2)Cough :**

**3)Expectoration :**

**4)Hemoptysis :**

**5)Fever :**

**6)Chest pain :**

**7)Drug intake :**

**Past History :**

**Family History :**

**Personal History :**

**** H/O Smoking :**

**(Duration & number )**

**** H/O Tobacco use :**

**** H/O Alcohol intake :**

**Treatment History :**

**General Examination :**

**Pulse Rate :** **Blood Pressure :**

**Respiratory Rate :** **Temperature :**

**System Examination :**

****Respiratory System :**

**Trachea :** **Accessory Muscles :**

**Rales And Rhonchi :** **Bronchial Breathing :**

**Tactile Fremitus :** **Vocal Fremitus :**

**Vocal Resonance :** **Adventitious Sounds :**

****Cardiovascular System : (heart sounds & murmurs)**

****Abdomen :**

****Central Nervous System :**

**INVESTIGATIONS :**

**1)Complete Haemogram ,**

**2)Renal Function Tests (urea ,creatinine ),**

**3)Electrolytes ,**

**4)Urine Complete (albumin ,sugar ,deposits ),**

**5)TROPONIN I Levels ,**

**6)ECG ,**

**7)ECHOCARDIOGRAM (PaSP , LVef ,Chamber dilatation)**

**8)Chest X Ray PA view,**

**9)Pulmonary Function Tests (PEFR ,FEV1/FVC )**

**10)Arterial Blood Gas analysis & SpO2**

| S.NO | Name | OP NO | age | sex | tobacco use | smoker | co morb | duration | RR/min | HR/min | spo2 | BP | pasp |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | periasamy | 7412 | 70 | m | no | f | HT | 20 | 18 | 66 | 96 | 150/90 | 40 |
| 2 | saravanan | 6747 | 48 | m | no | s | nil | 8 | 17 | 76 | 98 | 130/80 | 28 |
| 3 | palani | 54621 | 72 | m | NO | S | HT/DM | 10 | 18 | 72 | 96 | 140/80 | 34 |
| 4 | lakshmi | 64286 | 60 |  | YES | N | DM | 9 | 16 | 76 | 95 | 130/80 | 35 |
| 5 | mohd.omar | 426 | 75 | f | no | N | HT | 12 | 20 | 86 | 96 | 160/100 | 32 |
| 6 | sarasu | 740 | 65 | f | NO | N | NIL | 10 | 16 | 80 | 98 | 130/90 | 35 |
| 7 | marimuthu | 68217 | 57 | m | NO | S | NIL | 4 | 16 | 78 | 99 | 130/80 | 41 |
| 8 | kumar | 74186 | 50 | m | YES | S | NIL | 2 | 18 | 74 | 100 | 120/80 | 38 |
| 9 | govindhasamy | 7618 | 50 | m | no | S | DM | 1 | 18 | 68 | 100 | 130/80 | 36 |
| 10 | sasikala | 9100 | 44 | f | no | N | NIL | 3 | 16 | 78 | 100 | 130/90 | 35 |
| 11 | chidambaram | 1021 | 75 | m | NO | F | HT/DM | 11 | 20 | 96 | 95 | 150/90 | 42 |
| 12 | durairaj | 10674 | 41 | m | YES | S | NIL | 3 | 14 | 80 | 99 | 130/80 | 30 |
| 13 | vijaya | 17426 | 65 | f | NO | N | HT | 9 | 16 | 76 | 98 | 150/90 | 40 |
| 14 | gunasekaran | 1967 | 52 | m | YES | S | NIL | 8 | 16 | 70 | 98 | 130/80 | 32 |
| 15 | Palanisamy | 20672 | 68 | m | NO | S | DM | 7 | 18 | 74 | 96 | 140/90 | 38 |
| 16 | rajagounder | 62471 | 61 | m | NO | S | HT | 8 | 16 | 76 | 96 | 160/90 | 34 |
| 17 | ponnan | 667 | 67 | m | YES | S | NIL | 4 | 16 | 70 | 98 | 140/70 | 28 |
| 18 | periasamy | 7216 | 55 | m | no | N | NIL | 3 | 14 | 86 | 99 | 130/90 | 32 |
| 19 | chinnapappa | 86764 | 58 | f | no | N | HT | 9 | 15 | 84 | 97 | 160/100 | 33 |
| 20 | ravindran | 94182 | 61 | m | no | F | DM | 8 | 18 | 74 | 96 | 130/70 | 40 |
| 21 | arumugam | 18216 | 62 | m | YES | S | NIL | 10 | 16 | 70 | 99 | 140/80 | 36 |
| 22 | murugan | 67427 | 68 | m | no | S | HT | 12 | 18 | 72 | 96 | 160/110 | 38 |
| 23 | rajammal | 7104 | 70 | f | no | N | DM | 14 | 16 | 78 | 98 | 170/90 | 30 |
| 24 | prabakaran | 9172 | 55 | m | no | S | NIL | 5 | 16 | 84 | 99 | 140/70 | 32 |
| 25 | ramasamy | 2167 | 73 | m | no | F | HT | 7 | 18 | 88 | 97 | 140/100 | 39 |
| 26 | kulandhai | 74218 | 70 | f | no | N | DM | 8 | 18 | 90 | 97 | 130/90 | 40 |
| 27 | perumal | 86714 | 65 | m | YES | S | NIL | 5 | 16 | 86 | 98 | 120/70 | 42 |
| 28 | saravanan | 45161 | 57 | m | no | S | NIL | 5 | 18 | 70 | 99 | 130/80 | 30 |
| 29 | jayakodi | 5674 | 48 | f | no | N | NIL | 3 | 18 | 72 | 100 | 110/70 | 28 |
| 30 | subramani | 78792 | 52 | m | no | S | HT | 6 | 16 | 78 | 96 | 150/110 | 44 |
|  |  |  |  |  |  |  |  |  |  |  |  |  |  |
|  |  |  |  |  |  |  |  |  |  |  |  |  |  |

| Name | chambers | xray | ecg | po2 | pc02 | TROP-I | PEFR | FEV1/FVC | VENT DAYS | HOSP STAY | OUT COME | Column1 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| periasamy | DIL.RA RV | Emphysema | LVH | 76 | 44 | NEG | 70 | 0.7 | NIL | STABLE on medications | | |
| saravanan | N | N | NSR | 84 | 38 | NEG | 80 | 0.68 | NIL | STABLE on medications | | |
| palani | N | BHI | LVH | 80 | 40 | NEG | 80 | 0.66 | NIL | STABLE on medications | | |
| lakshmi | N | BHI | NSR | 78 | 37 | NEG | 70 | 0.67 | NIL | STABLE on medications | | |
| mohd.omar | N | BHI | NSR | 86 | 41 | NEG | 80 | 0.7 | NIL | STABLE on medications | | |
| sarasu | N | BHI | NSR | 76 | 38 | NEG | 70 | 0.7 | NIL | STABLE on medications | | |
| marimuthu | DIL.RA RV | N | TALL T V2 V4 | 92 | 36 | NEG | 70 | 0.68 | NIL | STABLE on medications | | |
| kumar | N | N | NSR | 90 | 40 | NEG | 80 | 0.68 | NIL | STABLE on medications | | |
| govindhasamy | N | N | NSR | 91 | 32 | NEG | 70 | 0.66 | NIL | STABLE on medications | | |
| sasikala | N | N | NSR | 94 | 36 | NEG | 70 | 0.65 | NIL | STABLE on medications | | |
| chidambaram | IL RA,RV,LV | Emphysema | LVH | 80 | 35 | NEG | 80 | 0.64 | NIL | STABLE on medications | | |
| durairaj | N | N | NSR | 88 | 40 | NEG | 80 | 0.66 | NIL | STABLE on medications | | |
| vijaya | DIL, RA,RV | Emphysema | LVH | 78 | 38 | NEG | 70 | 0.68 | NIL | STABLE on medications | | |
| gunasekaran | N | N | TALL T V2 V5 | 82 | 40 | NEG | 70 | 0.7 | NIL | STABLE on medications | | |
| Palanisamy | N | BHI | NSR | 80 | 38 | NEG | 70 | 0.68 | NIL | STABLE on medications | | |
| rajagounder | N | BHI | LVH | 86 | 44 | NEG | 70 | 0.6 | NIL | STABLE on medications | | |
| ponnan | N | N | NSR | 82 | 41 | NEG | 80 | 0.7 | NIL | STABLE on medications | | |
| periasamy | N | N | NSR | 78 | 39 | NEG | 80 | 0.63 | NIL | STABLE on medications | | |
| chinnapappa | DIL ,RA,RV | Emphysema | LVH | 84 | 38 | NEG | 70 | 0.7 | NIL | STABLE on medications | | |
| ravindran | DIL,RA,RV | BHI | NSR | 66 | 44 | NEG | 70 | 0.68 | NIL | STABLE on medications | | |
| arumugam | N | BHI | NSR | 86 | 36 | NEG | 80 | 0.62 | NIL | STABLE on medications | | |
| murugan | LVD | Emphysema | NSR | 80 | 40 | NEG | 70 | 0.7 | NIL | STABLE on medications | | |
| rajammal | N | BHI | TALL T V2 V4 | 78 | 31 | NEG | 80 | 0.61 | NIL | STABLE on medications | | |
| prabakaran | N | N | NSR | 84 | 38 | NEG | 70 | 0.68 | NIL | STABLE on medications | | |
| ramasamy | N | N | TALL T V2 V5 | 88 | 39 | NEG | 70 | 0.65 | NIL | STABLE on medications | | |
| kulandhai | DIL,RA,RV | N | NSR | 80 | 42 | NEG | 70 | 0.7 | NIL | STABLE on medications | | |
| perumal | LVD | BHI | LVH | 84 | 40 | NEG | 80 | 0.62 | NIL | STABLE on medications | | |
| saravanan | N | N | NSR | 90 | 36 | NEG | 80 | 0.63 | NIL | STABLE on medications | | |
| jayakodi | N | N | NSR | 92 | 42 | NEG | 70 | 0.7 | NIL | STABLE on medications | | |
| subramani | GRADE 1 DD | BHI | LVH | 88 | 40 | NEG | 70 | 0.65 | NIL | STABLE on medications | | |
| | | | | | | | | | | | | |
| | | | | | | | | | | | | |

| S.NO | Name | ip no | age | sex | tobacco use | smoking | co morbid | Duration | RR | HR | SPO2 | BP |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Andigounder60142 | | 70 | m | yes | s | HT/DM | 20 | 28 | 96 | 88 | 150/100 |
| 2 | marippan | 100924 | 42 | m | yes | s | HT | 10 | 24 | 98 | 94 | 170/110 |
| 3 | ramasamy | 101012 | 70 | m | no | s | HT | 25 | 32 | 102 | 84 | 160/100 |
| 4 | kannappan | 101021 | 60 | m | yes | f | DM | 5 | 26 | 106 | 96 | 130/70 |
| 5 | karupatti | 105734 | 65 | m | no | s | NO | 8 | 28 | 108 | 94 | 120/80 |
| 6 | chinnaponnu | 71480 | 45 | f | yes | n | NO | 7 | 22 | 106 | 68 | 110/70 |
| 7 | manickkammal | 20505 | 60 | f | no | n | HT | 12 | 24 | 98 | 84 | 150/100 |
| 8 | asokan | 40293 | 59 | m | yes | s | DM | 8 | 30 | 88 | 96 | 140/80 |
| 9 | akbar basha | 103760 | 55 | m | yes | f | DM | 7 | 32 | 92 | 84 | 160/90 |
| 10 | thangaraj | 38560 | 62 | m | no | s | NO | 9 | 28 | 94 | 92 | 140/100 |
| 11 | venkatachalam | 40094 | 70 | m | yes | s | NO | 12 | 24 | 98 | 92 | 120/70 |
| 12 | chandrappan | 40456 | 55 | m | no | f | NO | 11 | 26 | 112 | 96 | 130/100 |
| 13 | thavamani | 43144 | 60 | f | no | s | HT | 9 | 28 | 116 | 96 | 160/90 |
| 14 | chinraj | 45836 | 60 | m | no | f | HT | 8 | 30 | 102 | 78 | 170/110 |
| 15 | ranganathan | 46534 | 70 | m | yes | s | DM | 14 | 32 | 92 | 90 | 150/90 |
| 16 | kamala | 42210 | 50 | f | no | n | NO | 5 | 28 | 124 | 66 | 140/90 |
| 17 | palanisamy | 18001 | 55 | m | yes | s | HT | 4 | 22 | 96 | 90 | 160/100 |
| 18 | valarmathi | 57096 | 48 | f | no | n | HT | 8 | 22 | 96 | 98 | 160/100 |
| 19 | rajarathinam | 109854 | 54 | m | no | s | DM | 5 | 24 | 106 | 72 | 140/90 |
| 20 | paapa | 57504 | 55 | f | no | n | HT | 6 | 28 | 110 | 95 | 150/110 |
| 21 | sadaiyan | 1216 | 70 | m | no | f | HT/DM | 11 | 30 | 102 | 90 | 170/90 |
| 22 | allimuthu | 9720 | 65 | m | yes | s | NO | 9 | 22 | 116 | 85 | 180/100 |
| 23 | kandhammal | 54454 | 55 | f | no | n | DM | 8 | 26 | 92 | 92 | 140/80 |
| 24 | chinnasamy | 30592 | 60 | m | yes | f | NO | 9 | 28 | 98 | 86 | 130/80 |
| 25 | nainamalai | 107152 | 80 | m | no | f | NO | 25 | 30 | 114 | 70 | 140/100 |
| 26 | nallathambi | 568 | 52 | m | yes | s | HT | 6 | 22 | 90 | 92 | 150/90 |
| 27 | muthumani | 39294 | 65 | f | yes | n | HT/DM | 7 | 22 | 102 | 94 | 160/100 |
| 28 | chandran | 596 | 49 | m | no | s | HT | 8 | 24 | 112 | 85 | 150/110 |
| 29 | raveendran | 2476 | 61 | m | yes | s | NO | 11 | 26 | 108 | 86 | 140/90 |
| 30 | govindharaj | 60322 | 55 | m | no | s | DM | 9 | 30 | 96 | 80 | 150/90 |

| Name | XRAY | PASP | CHAMBERS | ECG | PaO2 | Pco2 | TROP -I | PEFR | FEV1/FVC | DAYS ON NIV/VENT | HOSPITAL STAY | OUTCOME |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Andigounder60142 | emphysema | 52 | RA RV DIL | LVH | 62 | 48 | NEG | 70 | 0.69 | - | 7 | DISCHARGED |
| marippan | BHI | 47 | LVD | ST | 70 | 45 | NEG | 70 | 0.71 | - | 5 | DISCHARGED |
| ramasamy | BHI | 41 | LVD | Tall t v2 v4 | 68 | 57 | **POSITIVE** | 60 | 0.59 | NIV,3 | 10 | DISCHARGED |
| kannappan | Consolidation R lung | 39 | Grade 1 dd | ST | 82 | 42 | NEG | 70 | 0.72 | - | 3 | DISCHARGED |
| karupatti | normal | 35 | normal | ST | 84 | 48 | NEG | 70 | 0.67 | - | 5 | DISCHARGED |
| chinnaponnu | BHI | 60 | ra rv dil,LVD | LVH,ST | 52 | 62 | **POSITIVE** | 50 | 0.6 | VENT. 5 | 10 | **DEATH** |
| manickkammal | Inc.BVM | 43 | mod LVD | ST | 60 | 60 | **POSITIVE** | 50 | 0.58 | NIV,4 | 12 | DISCHARGED |
| asokan | BHI | 51 | RA RV DIL | NSR | 78 | 48 | NEG | 60 | 0.61 | - | 4 | DISCHARGED |
| akbar basha | BHI | 58 | LVD | NSR | 62 | 57 | NEG | 60 | 0.57 | NIV,2 | 5 | DISCHARGED |
| thangaraj | normal | 57 | RA RV DIL | TALL T V2 V5 | 80 | 42 | NEG | 70 | 0.61 | - | 4 | DISCHARGED |
| venkatachalam | BHI | 42 | LVD | LVH | 72 | 38 | NEG | 70 | 0.66 | - | 7 | DISCHARGED |
| chandrappan | emphysema | 44 | normal | ST | 74 | 46 | NEG | 60 | 0.67 | - | 8 | DISCHARGED |
| thavamani | Pneumonitis R LL | 49 | normal | ST | 70 | 45 | NEG | 60 | 0.65 | - | 6 | DISCHARGED |
| chinraj | BHI | 50 | LVD | ST | 60 | 61 | **POSITIVE** | 60 | 0.6 | NIV,7 | 13 | DISCHARGED |
| ranganathan | normal | 51 | LVD | NSR | 68 | 60 | NEG | 70 | 0.7 | - | 5 | DISCHARGED |
| kamala | CONSOLIDATION L LUNG | 58 | LVD | ST | 45 | 66 | NEG | 50 | 0.52 | VENT,4 | 6 | **DEATH** |
| palanisamy | Inc.BVM | 44 | RA RV DIL | NSR | 72 | 42 | NEG | 60 | 0.62 | - | 5 | DISCHARGED |
| valarmathi | BHI | 46 | LVD | NSR,LVH | 80 | 42 | NEG | 60 | 0.61 | - | 7 | DISCHARGED |
| rajarathinam | Inc.BVM | 48 | RA RV DIL | ST | 54 | 52 | **POSITIVE** | 50 | 0.58 | VENT,7 | 12 | DISCHARGED |
| paapa | normal | 50 | SEVERE LVD | ST | 72 | 44 | NEG | 60 | 0.63 | - | 8 | DISCHARGED |
| sadaiyan | BHI | 32 | normal | ST | 66 | 54 | NEG | 70 | 0.72 | - | 4 | DISCHARGED |
| allimuthu | emphysema | 44 | normal | ST | 68 | 58 | NEG | 70 | 0.71 | NIV,4 | 15 | DISCHARGED |
| kandhammal | BHI | 42 | normal | NSR,LVH | 70 | 42 | NEG | 70 | 0.68 | - | 7 | DISCHARGED |
| chinnasamy | Inc.BVM | 32 | normal | NSR, | 66 | 46 | NEG | 70 | 0.67 | NIV,5 | 11 | DISCHARGED |
| nainamalai | BHI | 66 | RA RV DIL | TALL T V2 V5 | 66 | 62 | **POSITIVE** | 50 | 0.52 | VENT,5 | 5 | **DEATH** |
| nallathambi | normal | 43 | RA RV DIL | NSR | 72 | 41 | NEG | 70 | 0.74 | - | 4 | DISCHARGED |
| muthumani | BHI | 45 | normal | ST | 74 | 58 | NEG | 70 | 0.71 | - | 7 | DISCHARGED |
| chandran | Inc.BVM | 47 | LVD | ST | 60 | 57 | **POSITIVE** | 60 | 0.61 | NIV,4 | 8 | DISCHARGED |
| raveendran | Inc.BVM | 50 | LVD | TALL T V2V5 | 68 | 66 | NEG | 70 | 0.69 | NIV,3 | 9 | DISCHARGED |
| govindharaj | Pneumonitis R LL | 60 | RA RV DIL | ST | 66 | 68 | NEG | 60 | 0.67 | VENT,3 | 12 | DISCHARGED |

# LEGEND TO MASTER CHART :

| | | |
|---|---|---|
| **IP No | – | In Patient identification number |
| **OP No | – | Out Patient identification number |
| **T use | – | Tobacco usage |
| **CMorb | – | Comorbid conditions |
| **Dur | – | Duration in years |
| **RR | – | Respiratory rate |
| **PR | – | Pulse rate |
| **SpO2 | – | Saturation of O2 with Pulseoximeter |
| **BP | – | Blood Pressure in mm of Hg |
| **CXR | – | Chest X Ray PA view |
| **BHI | – | Bilateral Hyperinflation |
| **BVM | – | Broncho Vascular Markings |
| **Pn | – | Pneumonitis |
| **Cn | – | Consolidation |
| **E | – | Emphysema |
| **PaSP | – | Pulmonary artery systolic pressure |
| **N | – | Normal |
| **LVD | – | Left Ventricular Dysfunction |
| **DD | – | Diastolic Dysfunction |

| | | |
|---|---|---|
| **RA | – | Right Atrium |
| **RV | – | Right Ventricle |
| **LV | – | Left Ventricle |
| **LVH | – | Left Ventricular Hypertrophy |
| **ST | – | Sinus Tachycardia |
| **NSR | – | Normal Sinus Rhythm |
| **PaCO2 | – | Partial pressure of Carbon dioxide |
| **PO2 | – | Partial pressure of oxygen |
| **PEFR | – | Peak Expiratory Flow Rate |
| **NIV | – | Non Invasive Ventilation |
| **MV | – | Mechanical Ventilation (Invasive) |
| **HT | – | Hypertension |
| **DM | – | Diabetes Melleitus |
| **Y | – | Yes |
| **N | – | No |