# DISSERTATION ON

## PROGNOSTIC VALUE OF DIFFERENT SCORING MODELS IN PATIENTS WITH MULTIPLE ORGAN DYSFUNCTION SYNDROME ASSOCIATED WITH ACUTE COPD EXACERBATION

*Submitted in partial fulfillment of the regulations*
*for the award of the degree of*

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## DEPARTMENT OF GERIATRIC MEDICINE
## MADRAS MEDICAL COLLEGE
## CHENNAI – 600003
## MAY 2019

# CERTIFICATE

This is to certify that the dissertation titled **"PROGNOSTIC VALUE OF DIFFERENT SCORING MODELS IN PATIENTS WITH MULTIPLE ORGAN DYSFUNCTION SYNDROME ASSOCIATED WITH ACUTE COPD EXACERBATION"** is the bonafide work done by **Dr. GANESAN.C**, Post Graduate Student, Department of Geriatric Medicine, Madras Medical College, Chennai – 600003, in partial fulfilment of the University rules and regulations for the award of **MD DEGREE** in **GERIATRIC MEDICINE BRANCH – XVI**, under our guidance and supervision, for the examination to be held on **May 2019**.

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# DECLARATION

I solemnly declare that this dissertation titled **"PROGNOSTIC VALUE OF DIFFERENT SCORING MODELS IN PATIENTS WITH MULTIPLE ORGAN DYSFUNCTION SYNDROME ASSOCIATED WITH ACUTE COPD EXACERBATION"** was done by me at Madras Medical College, Chennai – 600003, during the period of July 2017 to June 2018 under the guidance and supervision of **Prof. Dr.G.S.SHANTHI, M.D. (Geriatrics),** to be submitted to The Tamilnadu Dr. M.G.R. Medical University, towards the partial fulfilment of requirements for the award of **MD DEGREE IN GERIATRIC MEDICINE BRANCH – XVI**.

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# ABBREVIATION

| | |
|---|---|
| US | United States |
| COPD | Chronic obstructive pulmonary disease |
| MODS | Multiple organ dysfunction syndrome |
| SOFA | Sepsis related organ failure assessment |
| APACHE II | Acute physiology And chronic health evaluation |
| WHO | World health organization |
| GOLD | Global obstructive lung disease |
| NCMH | National commission on macroeconomic and health |
| DALY | Disability adjusted life year |
| EAG | Empowered action group |
| FEV1 | Forced expiratory volume in one second |
| ETS | Environmental tobacco smoking |
| AATD | Alpha 1 antitrypsin deficiency |
| MMP | Matrix metalloproteinase -12 |
| FVC | Forced vital capacity |
| HHIP | Hedgehog interacting proteins |
| HIV | Human immunodeficiency virus |
| TH1 | T helper type 1 |
| TH17 | T helper type 17 |
| Tc1 | T cytotoxic 1 |
| ILC3 | Innate lymphoid cells 3 |
| IgA | Immunoglobulin A |
| VA/Q | Ventilation perfusion ratio |
| MMRC | Modified medical research council |
| AFL | Severity of airflow limitation |
| CVD | Cardiovascular disease |
| tPA | tissue plasminogen activator |
| PAI | Plasminogen activator inhibitor |
| vWF | Von willebrand factor |
| BODE | BMI, airflow obstruction, Dyspnea, Exercise capacity |
| BMI | Body Mass Index |
| ACCP | American college of clinical pharmacy |
| SCCM | Society of critical care medicine |
| GABA | Gamma –Aminobutyric Acid |

| | |
|---|---|
| RBC | Red blood cell |
| ARDS | Acute respiratory distress syndrome |
| AKI | Acute kidney injury |
| CNS | Central nervous system |
| TT | Thrombin time |
| aPTT | activated Partial thromboplastic time |
| DIC | Disseminated intravascular coagulation |
| PARs | Protease activated receptor |
| SIRS | Systemic inflammatory response syndrome |
| TNF | Tumor Necrosis factor |
| IL | Interleukins |
| PAF | Platelet activating factor |
| $LTB_4$ | Leukotriene $B_4$ |
| PGE | Prostaglandin E |
| $TxA_2$ | Thromboxane $–A_2$ |
| PCA | Procoagulant activity |
| $PGI_2$ | Prostaglandin $I_2$ |
| CARS | Compensatory anti- inflammatory response syndrome |
| DNA | Deoxyribo Nucleic Acid |
| NTEs | Neutrophil Extracellular traps |
| ICU | Intensive care unit |
| GCS | Glasgow coma scale |
| GICU | Geriatric intensive care unit |
| IMCU | Intensive medical care unit |
| PE | Pulmonary embolism |
| CBC | Complete blood count |
| RFT | Renal functional test |
| LFT | Liver functional test |
| ABG | Arterial blood gas |
| ROC | Receiver operating characteristic curve |
| AUC | Area under curve |
| CI | Confidence interval |
| PaO2 | Partial pressure of oxygen |
| Fio2 | Fraction of inspired oxygen |
| MAP | Mean systolic Arterial pressure |

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# INTRODUCTION

# INTRODUCTION

At a faster rate, the global population is growing old, owing to decreased fertility, improving health and longevity. Remarkably, there is great rise in elderly population at never before seen rate. 562 million people (8%) constituted the elderly population > 65 years, when world population reached 7 billion in 2012. 3 years later the elderly population rose by 0.5% i.e., 617 million constituting 8.5% of total population (figure 1).[1, 2]

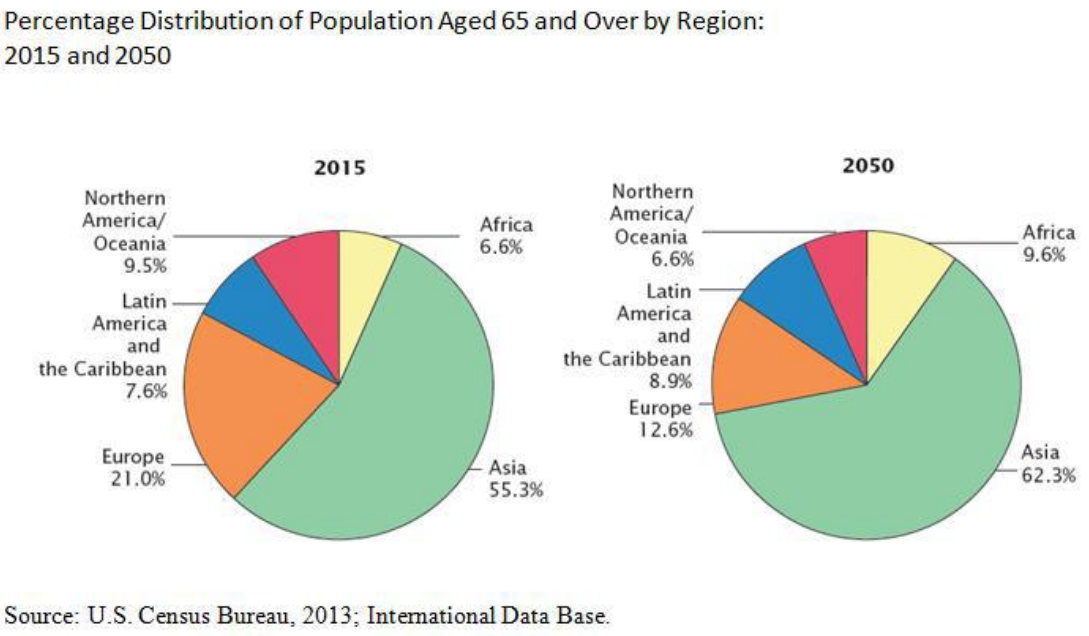



**FIGURE 1: P**ercentage distribution of population aged 65 and over by region: 2015 and 2050.

As per census 2011, India's total population was 1210.9 million with elderly above 60 years contributes 8.6% of total population i.e. around 103.9 million of elderly population 51.1 million are elderly males (8.2%) and 52.8 million are elderly females (9%) with

respect to residence, the proportion of rural elderly population was 8.8%. In urban areas it was 8.1 percent (table 1).[4, 16]

| Census | Person | Male | Female | Rural | Urban |
|---|---|---|---|---|---|
| 1961 | 5.6 | 5.5 | 5.8 | 5.8 | 4.7 |
| 1971 | 6.0 | 5.9 | 6.0 | 6.2 | 5.0 |
| 1981 | 6.5 | 6.4 | 6.6 | 6.8 | 5.4 |
| 1991 | 6.8 | 6.7 | 6.8 | 7.1 | 5.7 |
| 2001 | 7.4 | 7.1 | 7.8 | 7.7 | 6.7 |
| 2011 | 8.6 | 8.2 | 9.0 | 8.8 | 8.1 |

**TABLE 1:** Percentage share of elderly population aged 60 and above to total population.

Inspite of overwhelming global geriatric population, due to latest advanced treatment modalities and elderly health schemes have caused a remarkable raise in life expectancy of elderly people. Yet, Chronic Obstructive Pulmonary Disease (COPD) is one among the leading causes of death of elderly patients in the last decade. The burden of COPD is expected to increase in forthcoming decades due to sustained exposure to COPD risk factors by the elderly patients.[8]

Chronic obstructive pulmonary disease [COPD] is a prevalent disease and a major cause of morbidity in elderly patients worldwide Severe exacerbation of COPD has been associated with increased mortality, which has been attributed to complex underlying disease that are more likely to progress into multiple organ dysfunction syndrome [MODS]

due to their association with inflammatory mediators. COPD is one of the leading cause of death in urban area.[8]

The economic burden of COPD increased due to repeated hospitalization and increased length of hospital stay. COPD is a multiorgan disorder, the past decade assessment of COPD have demonstrated that COPD is associated with multiple non-pulmonary manifestation that contributes to remarkable increase in morbidity and mortality.[11]

Acute exacerbation of COPD with systemic consequence of multiple organ disorder syndrome (MODS) is characterized by more than one organ failing especially during critical illness. In order to assess the severity and predict the outcome of elderly people who have been admitted due to COPD with MODS, several scoring models have been proposed in the previous literature. Among the numerable scoring models, the Sepsis related Organ Failure Assessment (SOFA) and Acute Physiology And Chronic Health Evaluation II (APACHE II) are the widely used prognosis scoring scales. This study compare and contrast the above mentioned two scoring models and analyze the superior, time and cost effective model in predicting the prognosis of patients affected by COPD with MODS.[6]

# AIMS AND OBJECTIVES

# AIM

To assess the prognostic value of different scoring models in elderly patients with multiple organ dysfunction syndrome associated with acute COPD exacerbation.

# OBJECTIVES

* To compare the predictive value of APACHEII and SOFA scores in elderly patients with multiple organ dysfunction syndrome associated with acute COPD exacerbation.

* To find out which score is highly predictable in elderly patients with multiple organ dysfunction associated with acute COPD exacerbation.

# REVIEW OF LITERATURE

# REVIEW OF LITERATURE

## DISEASES OF THE OLD AGE:

Age-related diseases are illnesses and conditions that occur more frequently in people as they get older, meaning age is a significant risk factor. The leading causes of death among adults over the age of 65 are also the most common causes of death among the population as a whole. Many of these conditions are also highly preventable and treatable. It is important to understand these diseases, know when and where to get treatment, and know how you can live with them to help prolong life and health. Here are the top causes of death for adults over the age of 65[7]

* Heart Disease
* Cancer
* Chronic Obstructive Pulmonary Disease (COPD)
* Cerebrovascular Disease (Stroke)
* Diabetes
* Pneumonia and Influenza
* Accidents
* Acute nephritis
* Septicemia
* Alzheimer's disease

**COPD (Chronic Pulmonary Obstructive Disease):**

Chronic obstructive pulmonary disease [COPD] is a prevalent disease and a major cause of morbidity in elderly patients worldwide Severe exacerbation of COPD has been associated with increased mortality, which has been attributed to complex underlying disease that are more likely to progress into multiple organ dysfunction syndrome [MODS] due to their association with inflammatory mediators. COPD is one of the leading cause of death in urban area.[8]

Chronic obstructive pulmonary disease (COPD) and asthma are the two commonest causes of adult airflow obstruction. Asthma is differentiated from COPD by the variability of the airflow obstruction (i.e. exacerbations) while COPD is defined as "a chronic slowly progressive disease characterized by airflow obstruction that does not change markedly over several months."

Presently COPD has aroused considerable concern in the medical and scientific communities due to its poor prognosis and the growing substantial burden, the disease imposes on the healthcare system. The problem of COPD is expected to increase in near future due to nonstop exposure to risk factors causing COPD.

Chronic Obstructive Pulmonary Disease (COPD) is a major public health problem. In 2020, COPD is projected to rank fifth worldwide in term of burden of disease and third in term of mortality. Although COPD has received increasing attention from the medical

community in recent years, it is still relatively unknown or ignored by the public as well as public health and government officials.[9]

In 1998, the WHO along with the US National Heart, Lung and Blood institute have designed the Global Initiative for Chronic Obstructive Lung Disease (GOLD) to bring more attention to the management and prevention of COPD. Among the important objectives of GOLD are to increase awareness of COPD and to help the millions of people who suffer from this disease and die prematurely from it or its complications. [8]

Thus, COPD is a common preventable disease which is characterized by continual airflow limitation that is usually progressive and accompanied with an enhanced chronic inflammatory response in the airways and the lung to noxious particles or gases. Exacerbations and comorbidities contribute to the overall prognosis in the patients affected with COPD.[8]

**BURDEN OF COPD:**

COPD incidence, morbidity, and mortality may differ among different countries and across different groups within countries. COPD is the result of cumulative exposures over decades. Often, the occurrence of COPD is directly interrelated to the frequency of tobacco smoking, other than cigarrete smoking, outdoor, occupational and indoor air pollution due to burning of wood and other biomass fuels may also have remarkable role in development

of COPD. The burden of COPD is projected to increase in the fore coming decades due to continued exposure to COPD risk factors and the aging of the world's population.[10, 11]

**PREVALENCE:**

Even though the prevalence of COPD data show remarkable variation due to differences in survey methods, analytical, and investigative approaches, the data from National Commission on Macroeconomics and Health (NCMH) has recognized India as one of the countries furthermost affected by COPD. The Commission reported that about 17 million Indians suffering from COPD in 2006, and has suggested that the affected people score may reach 22 million by 2016. According to NCMH estimates, COPD is more predominant in the rural areas of India compared to the urban areas of the country.[4]

**GEOGRAPHICAL DISTRIBUTION OF COPD IN GLOBAL POPULATION:**

Approximately 2.7 million deaths from chronic obstructive pulmonary disease (COPD) occurred in 2000, half of them in the Western Pacific Region, with the majority of these occurring in China. About 400,000 deaths occur each year from COPD in industrialized countries.

Age-standardized DALY rates due to COPD in 2015 were estimated to exceed 2000 per 100 000 people in Papua New Guinea, India, Lesotho, and Nepal. Rates below 300 per 100 000 people were seen in some countries in high-income Asia Pacific, central Europe,

north Africa and Middle East, the Caribbean, western Europe, and Andean Latin America (figure 2).[5]

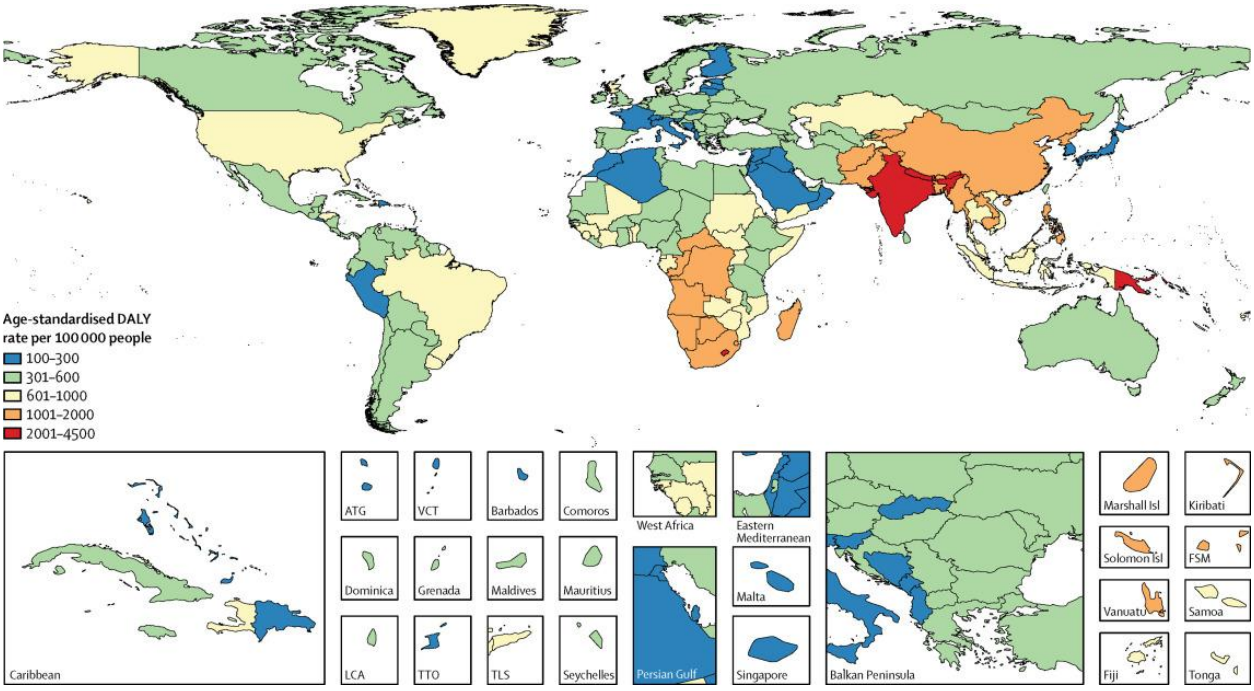



**FIGURE 2:** Age-standardized DALY rate per 100 000 people due to COPD by country, both sexes, 2015
DALYs=disability-adjusted life years. COPD=chronic obstructive pulmonary disease. ATG=Antigua and Barbuda. FSM=Federated States of Micronesia. Isl=islands. LCA=Saint Lucia. TLS=Timor-Leste. TTO=Trinidad and Tobago. VCT=Saint Vincent and the Grenadines.

## GEOGRAPHICAL DISTRIBUTION OF COPD IN INDIAN POPULATION:

In a study conducted by Hossain MM et al, 2018 revealed the range of prevalence of COPD in different states. The prevalence ranged between 2 to 22% among the men and 1.2 to 19% among women in different population-based studies across India. It has became the fourth leading cause of years of life lost in Empowered Action Group (EAG) States

including Bihar, Jharkhand, Madhya Pradesh, Chhattisgarh, Odisha, Rajasthan, Uttar Pradesh and Uttarakhand. Also, COPD ranked seventh among the North-East States including Assam, Mizoram, Arunachal Pradesh, Meghalaya, Nagaland, Tripura, Sikkim and Manipur. Among the remaining states of India, COPD ranked fourth among all causes of years of life lost. In this varying range of disease burden, the highest rate of death from COPD was nine times the lowest rate among all the states (figure 3).[13 14]

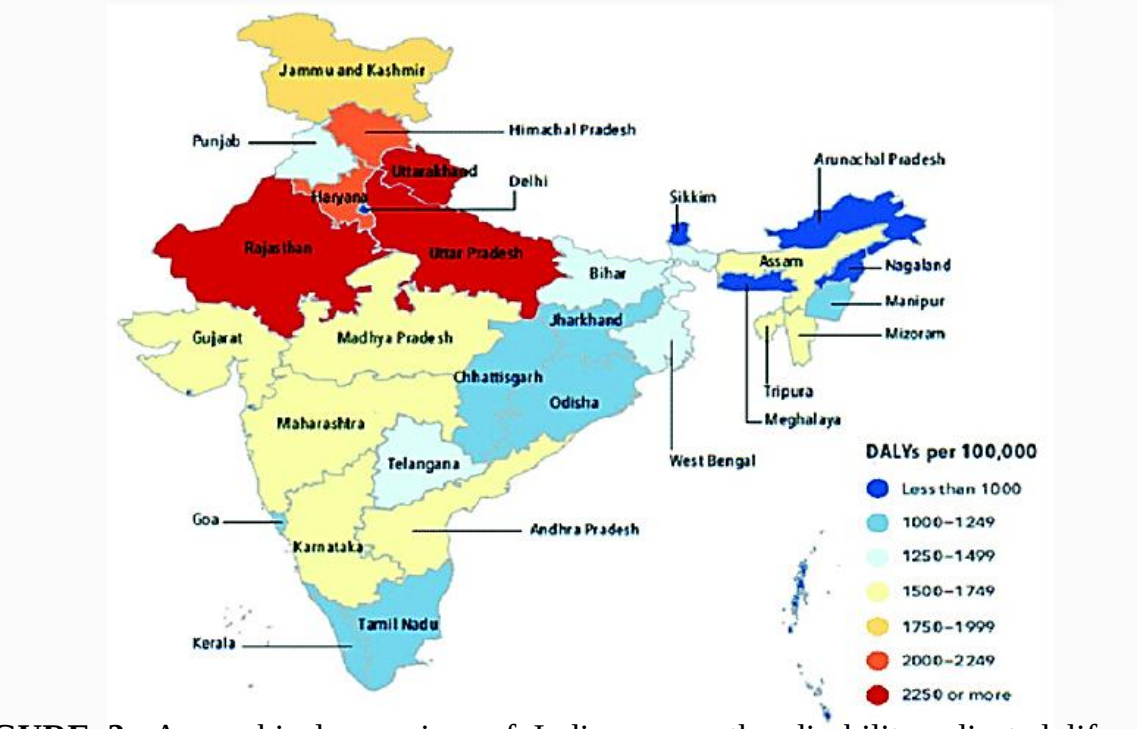



**FIGURE 3:** A graphical overview of India as per the disability adjusted life years (DALYs) rate due to COPD.

## ECONOMIC BURDEN OF COPD:

Treating COPD and associated comorbidities require institutional resources and frequent hospitalization which can be costly for the individuals as well as the health system. The rate of hospitalization can be four times among elderly aged more than 65 years

compared to younger patients. The mean length of hospital stays for COPD ranges from 4.5 to 16 days in normal to intensive care. [8]

The economic burden of COPD was estimated in Crores of Rupees (1 Crore =10 Million). As per these estimates the current estimated burden of COPD for India is 35,000 Crore Rs. or 350,000 Million Rs. (Rs. 350 Billion). This is likely to reach a staggering 48,000 Crore Rs. (Rs. 480 Billion) in next five years (figure 4). If, however the medical community were to adhere to standardized national and international treatment guidelines this cost could drastically come down.[10, 11]

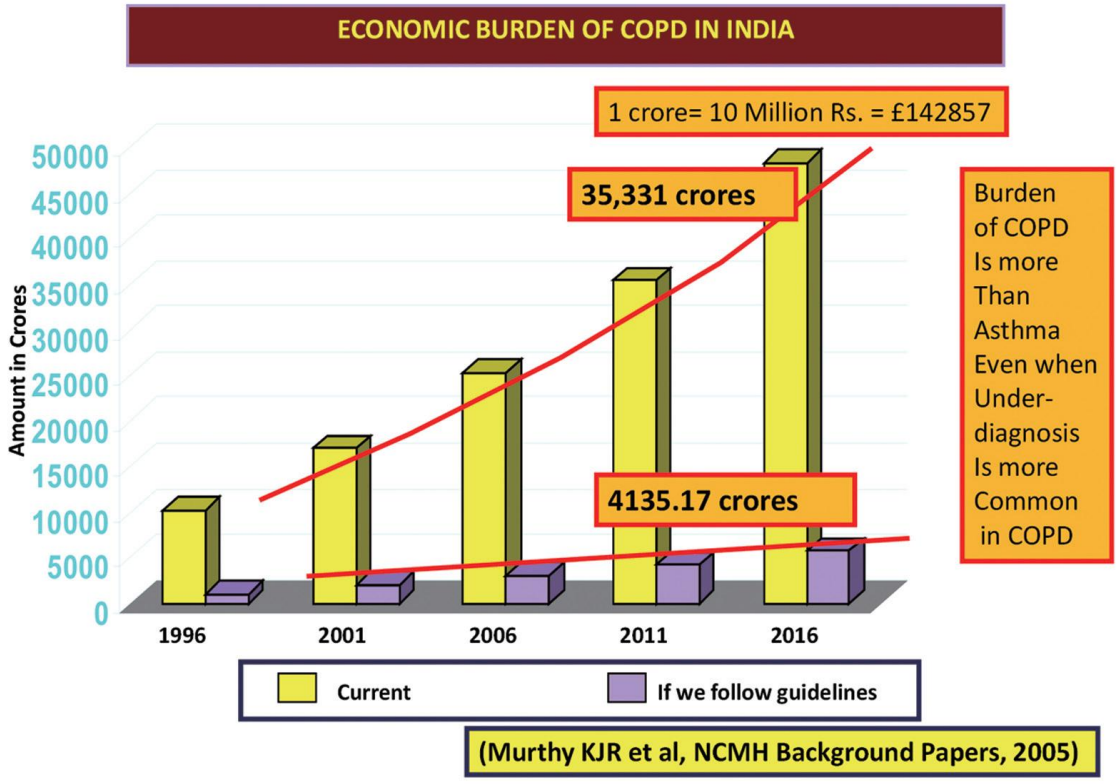



**FIGURE 4:** Graphical presentation of economic burden of COPD for the year 1996 to 2016.

**MORTALITY:**

Under-recognition and under-diagnosis of COPD still affect the accuracy of mortality data with COPD often listed as a contributory cause of death or omitted from the death certificate entirely. The Global Burden of Disease Study projected that COPD, could be the sixth leading cause of death in 1990, will turn out to be third by 2020; a newer projection appraised COPD will be the fourth foremost cause of death in 2030. The mortality rate of COPD patients is noted to be expanding due to widespread of smoking habit (figure 5).[8]

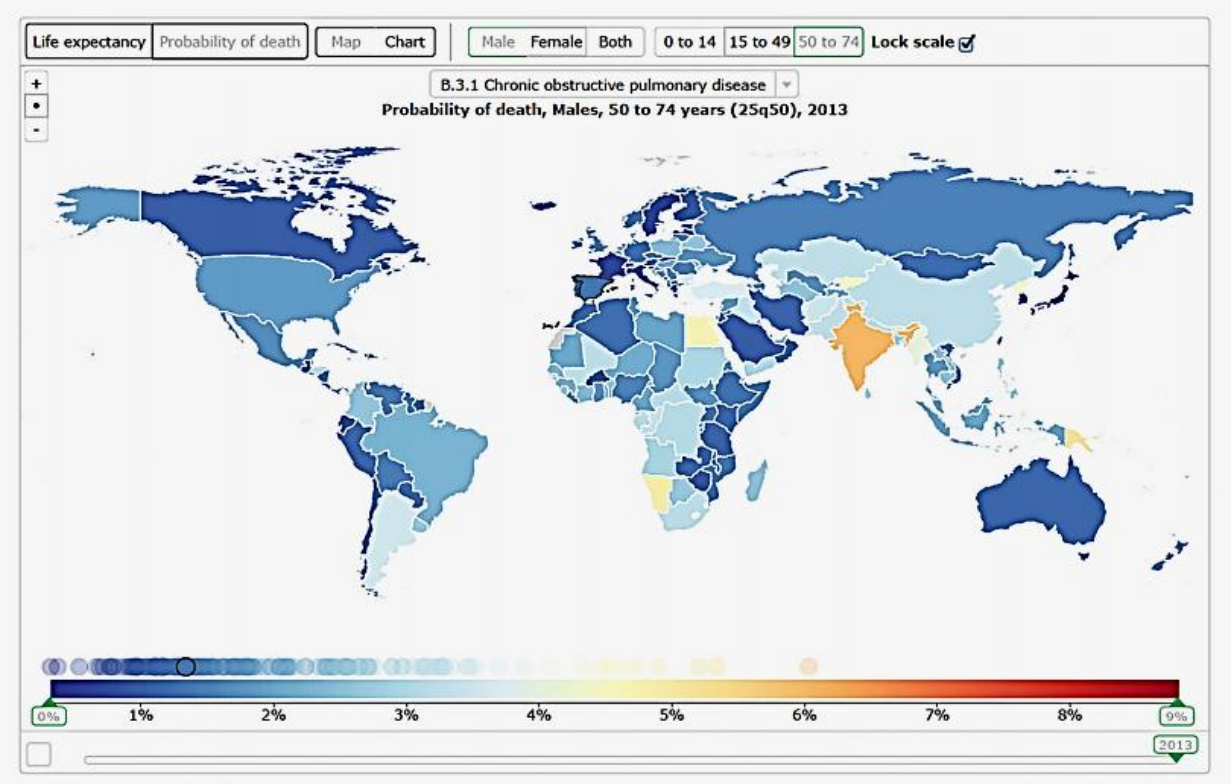



**FIGURE 5:** It shows that the probability of a given individual to die of COPD in his/her lifetime is actually higher in India or some African countries than in the any western country nowadays, despite a lower prevalence of COPD.

## FACTORS THAT INFLUENCE DISEASE DEVELOPMENT AND PROGRESSION OF COPD:

### Cigarette smoking:

Although cigarette smoking is the major COPD risk factor, previous literatures have identified that nonsmokers may also develop COPD. Besides, among people with the same smoking history, all smokers are not eventually affected by COPD which is still unclear but most probably due to differences in genetic backgrounds and other exposures.

Across the world, cigarette smoking is the most frequently faced risk factor for COPD. Other types of tobacco (e.g., pipe, cigar, water pipe) and marijuana can also contribute to COPD. Cigarette smokers have a greater chance to develop respiratory symptoms and lung function disorders leading to gradual decline in FEV1, and high COPD mortality rate than nonsmokers.[8, 12, 17 & 18.]

### Passive smoking or Environmental Tobacco Smoke (ETS):

Passive exposure to cigarette smoke (also known as environmental tobacco smoke or ETS) may also contribute to respiratory diseases and COPD by increasing the total burden of inhaled particles and gases in lung. Smoking during pregnancy may also be teratogenic by affecting lung growth and development of the fetus in utero and possibly having an ill-control over the immune system of the fetus.

According to Hagstad et al, 2014 in his study of relationship between ETS and COPD in lifelong never smokers he concluded that the prevalence of COPD is markedly higher among subjects reporting ETS both at work and at home, with the highest prevalence

among those reporting current exposure at home in combination with both at previous and current work (figure 6 and 7).[19]

**FIGURE 6:** Picture depicting the indoor air pollution.

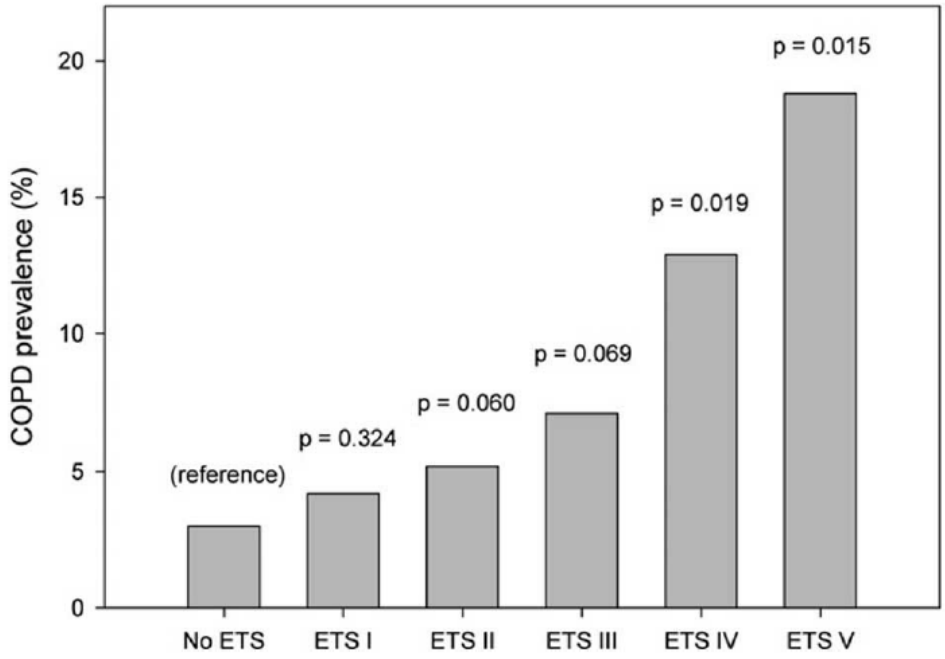



**FIGURE 7:** Prevalence of COPD according to GOLD (Global Initiative for Chronic Obstructive Lung Disease) by ETS exposure among never smoking subjects aged _ 65 years old. ETS exposure was characterized as no ETS exposure, ETS I (ever at home), ETS II (at previous work), ETS III (at both previous and current work), ETS I (ever at home and at both previous and current work), ETS V (current at home and at both previous and current work).

Exposure of humans to occupational hazardous agents such as biochemical agents, fumes, organic and inorganic dusts play a crucial role in occurrence of COPD. Indoor pollution can be caused by burning of wood, Degradation of human and animal dung, crop residues, gases released while burning coals and poorly functioning stoves, may lead to very high levels of indoor air pollution. Evidence continues to grow that indoor pollution from biomass cooking and heating in poorly ventilated dwellings is an important risk factor for COPD. Billions of people worldwide use biomass and coal as their main source of energy for cooking, heating, and other household needs, so the population at risk of developing COPD worldwide is very high.[21]

**Occupational dusts and chemicals:**

Long-term exposure to industrial dust, chemicals, and gases can irritate and inflame the airways and lungs. This increases your risk of developing COPD. People exposed to dust and chemical vapors, such as coal miners, grain handlers, and metal molders, have a greater likelihood of developing COPD. One survey in the United States found that the fraction of COPD attributed to work was estimated at 19.2 percent overall, and 31.1 percent among those who had never smoked.[22]

**Genetic factors:**

Severe hereditary alpha -1 antitrypsin deficiency (AATD) is documented as a major contributing genetic risk factor for COPD. Although AATD involves only a small part of world population, it explains the interaction between the genes and environment exposures

that predispose a human being to COPD. Gene encoding for matrix metalloproteinase -12 (MMP-12) aid to decline the lung function. Several genome –wide association studies have linked genetic loci with COPD (or FEV1 or FEV1/FVC as the phenotype) including markers near the alpha-nicotinic acetyl choline receptors, hedgehog interacting proteins (HHIP) and several others are directly responsible for COPD.[2]

**Age:**

Aging changes in respiratory system is one of the risk factor for COPD. Aging of the airways and parenchyma mimics some of the structural changes associated with COPD (figure 8).[7, 48]

**Socioeconomic status:**

Lower socioeconomic status is associated with increased risk of developing COPD. The components of poverty such as exposures to indoor and outdoor air pollutions, crowding, poor nutrition, infection and other factors related to low socioeconomic status may contribute the risk of developing COPD.[7, 48]

**Asthma and airway hyperactivity:**

Asthma might be a hazardous factor for the development of chronic airflow limitation and COPD. In a longitudinal cohort study of Tucson Epidemiological study of Airway Obstructive Disease, revealed patient with asthma are at a 12-fold higher risk of developing COPD compared to those without asthma. Airway hyper-responsiveness in the

absence of diagnosis of asthma can be also considered as independent predictor of COPD and respiratory mortality in population studies.[26]

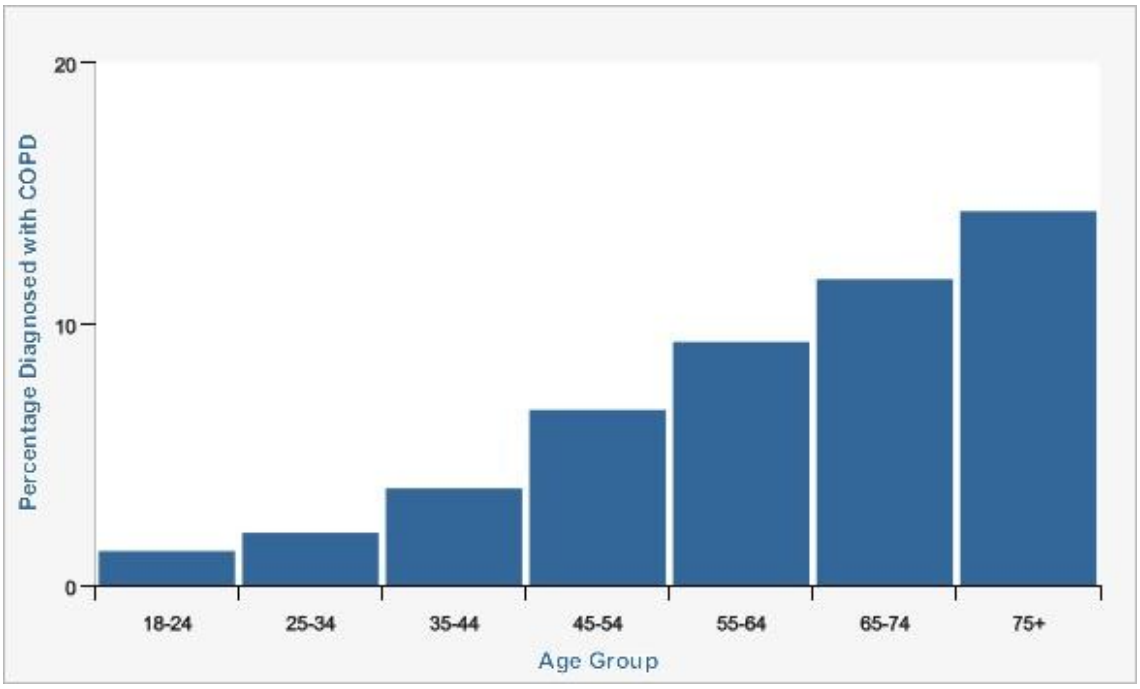



**FIGURE 8:** Graphical representation of increased risk of developing COPD with increase in age.

**Chronic bronchitis:**

In a study conducted by Felcher and colleagues, chronic bronchitis was not associated with an accelerated decline in lung function. However, subsequent studies have observed an association between mucous hypersecretion and increased FEV1 decline, and in younger adult who smoke, the presence of chronic bronchitis has been associated with an increased likelihood of developing COPD. Chronic bronchitis has also been associated with an increased risk in the total number as well as severity of exacerbation.

**Infection:**

Recurrent childhood respiratory tract infection has been related with reduced lung function and increased respiratory symptoms in adult hood. Susceptibility to infections plays a role in exacerbation of COPD but the effect on disease development is unclear. Tuberculosis is also been identified as a risk factor for COPD and a potential comorbidity.[8]

**PATHOLOGY, PATHOGENESIS AND PATHOPHYSIOLOGY:**

Inhaled particles (from cigarette smoke or other sources) cause lung inflammation, a normal response that appears to be modified in individuals who develop COPD. The chronic lung inflammatory response may bring out parenchymal tissue destruction (resulting in emphysema), and interrupt normal repair and defense mechanisms (causing small airway fibrosis), which in turn lead to air trapping and progressive airflow limitation.

**PATHOLOGY:**

Chronic inflammatory changes with increased numbers of inflammatory cell types, and structural changes resulting from repeated injury and repair are found in the airways, lung parenchyma, and pulmonary vasculature of patients with COPD. In general, these changes increase with disease severity and persist despite smoking cessation. Systemic inflammation may be present and play a role in the multiple comorbidity conditions found in patients with COPD.[23]

## PATHOGENESIS:

The above mentioned pathological changes appears to be an enhancement of the normal, physiological, inflammatory response of the respiratory tract to chronic irritants. The mechanisms for this amplified inflammation in COPD are not yet understood but may be genetically determined. Patients can clearly develop COPD without smoking, but the nature of the inflammatory response in these patients is unknown. Oxidative stress and an excess of proteinases are likely to further modify lung inflammation. Together these mechanism lead to characteristic pathological changes in COPD. Lung inflammation persists after smoking cessation through unknown mechanisms, although auto-antigens and persistent microorganisms may play a role (figure 9).[25]

### Oxidative stress:

Oxidative stress may be an important amplifying mechanism of COPD. These oxidants are generated by cigarette smoke, other inhaled particulates and released from activated inflammatory cells such as macrophages and neutrophils. There may be reduction in endogenous anti-oxidants in COPD patients as a result of reduction in levels of transcription factor Nrf2 that regulate many antioxidant genes. Biomarkers of oxidative stress (e.g. Hydrogen peroxide, 8-isoprostane) are increased in the exhaled breath condensate, sputum and systemic circulation of COPD patients. Oxidative stress is further increased by further exacerbations.[26]

PATHOGENESIS OF CHRONIC BRONCHITIS AND EMPHYSEMA

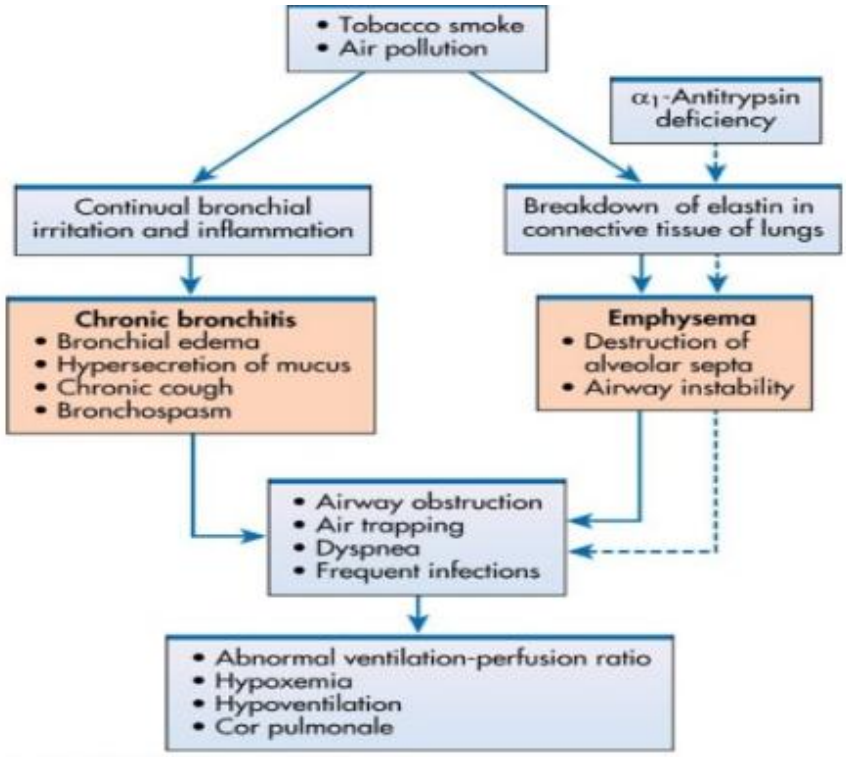



**FIGURE 9:** Flowchart explaining the risk factors and pathogenesis of chronic bronchitis and emphysema and pathological changes occurring in the respiratory system.

**Protease-antiprotease imbalance:**

Imbalance in the protease that break down the connective tissue components in the lung of the COPD patients and the antiprotease that counterbalance this action of protease is noted in the lung of COPD patients. The increased levels of several protease, derived from inflammatory cells and epithelial cells have been observed in COPD patients. There is increasing evidence that this protease leads to destruction of elastin, a major connective tissue component in lung parenchyma, is believed to be an important feature of emphysema.[25]

**Inflammatory cells:**

Increased number of macrophages, neutrophils and lymphocytes that include Tc1, Th1, Th17 and ILC3 cells are seen in the peripheral airways, pulmonary vasculature and lung parenchyma of COPD patients. All of these inflammatory cells together with epithelial cells and other structural cells release multiple inflammatory mediators. A recent study suggest that local IgA deficiency is associated with bacterial translocation, small airway inflammation and airway remodeling.[26]

**Inflammatory mediators:**

The wide variety of inflammatory mediators that have been shown to be increased in COPD patients attract inflammatory cells from the circulation (chemotactic factors), amplify the inflammation process (proinflammatory cytokines) and induce structural changes (growth factors).[26]

**Peribronchiolar and interstitial fibrosis:**

An excessive production of growth factors may be found in smokers or those with preceding airway inflammation who have COPD. Inflammation may precede the development of Peribronchiolar fibrosis and interstitial opacities have been reported in patients with COPD or those who are asymptomatic smokers. The repeated injury of the lung airway wall itself may lead to excessive production of muscle and fibrous tissue. This may be a contributory factor for the development of small airway limitation and eventually the obliteration that may precede the development of emphysema.[26]

## PATHOPHYSIOLOGY:

### Airflow Limitation and Air Trapping:

Inflammation and narrowing of peripheral airways leads to decreased FEV1. Parenchymal destruction due to emphysema also contributes to airflow limitation due to reduced elastic recoil. In combination, both progressively lead to gas trapping during expiration, resulting in hyperinflation.[23]

### Gas Exchange Abnormalities:

Gas exchange abnormalities may result in hypoxemia and hypercapnia, and have several mechanisms in COPD. The main one is VA/Q abnormalities. Reduced ventilatory drive may lead to carbon dioxide retention, particularly when combined with reduced ventilation.[28]

### Mucus Hypersecretion:

Mucus hypersecretion, resulting in a chronic productive cough, is a feature of chronic bronchitis and is not necessarily associated with airflow limitation. Conversely, not all patients with COPD have symptomatic mucus hypersecretion. When present, it is due to an increased number of goblet cells and enlarged sub mucosal glands in response to chronic airway irritation.[26]

**Pulmonary Hypertension:**

Pulmonary hypertension may develop late in the course of COPD. It can be due to hypoxic vasoconstriction of small pulmonary arteries, eventually resulting in structural changes that include intimal hyperplasia and later smooth muscle hypertrophy/hyperplasia, and/or loss of pulmonary capillary bed due to emphysema. In pulmonary vessels an inflammatory response similar to that seen in the airways (and evidence of endothelial dysfunction) has been identified. Severe pulmonary hypertension may lead to right ventricular hypertrophy and eventually to right-side cardiac failure.[26, 28]

**DIAGNOSIS AND ASSESSMENT:**

**Medical History:**

A detailed medical, environmental, family history should be taken to assess the occurrence COPD: [2]

• Exposure to risk factors

• Past medical history

• Family history of COPD or other chronic respiratory disease

• Pattern of symptom development

• History of exacerbations or previous hospitalizations for respiratory disorder

• Presence of comorbidities

• Impact of disease on patient's life

• Social and family support available to the patient

• Possibilities for reducing risk factors, especially smoking cessation[49]

## CLINICAL DIAGNOSIS:

A clinical finding of COPD should be watchful in any patient who has dyspnea, chronic cough and/or sputum production, and/or a history of contact to risk factors for the disease (figure 10).[49]

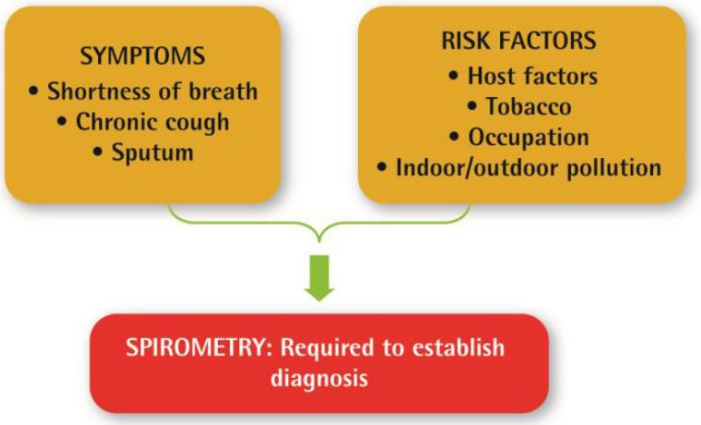



**FIGURE 10:** Pathway for clinical diagnosis of COPD.

## SPIROMETRY:

Spirometry is mandatory to make the clinical diagnosis of COPD. The presence of a post bronchodilator FEV1/FVC < 0.70 confirms the presence of persistent chronic airflow limitation and thus of COPD. The spirometric criterion for airflow limitation remains a post-bronchodilator fixed ratio of FEV1/FVC < 0.70. This criterion is simple, independent of reference values, and has been used in numerous clinical trials forming the evidence base from which most of our treatment recommendations are drawn. Diagnostic simplicity and consistency are key for the busy non specialist clinician. While post-bronchodilator spirometry is required for the diagnosis and assessment of severity of COPD, the degree of

reversibility of airflow limitation (e.g., measuring FEV1 before and after bronchodilator or corticosteroids) is no longer recommended (figure 11).[24, 48]

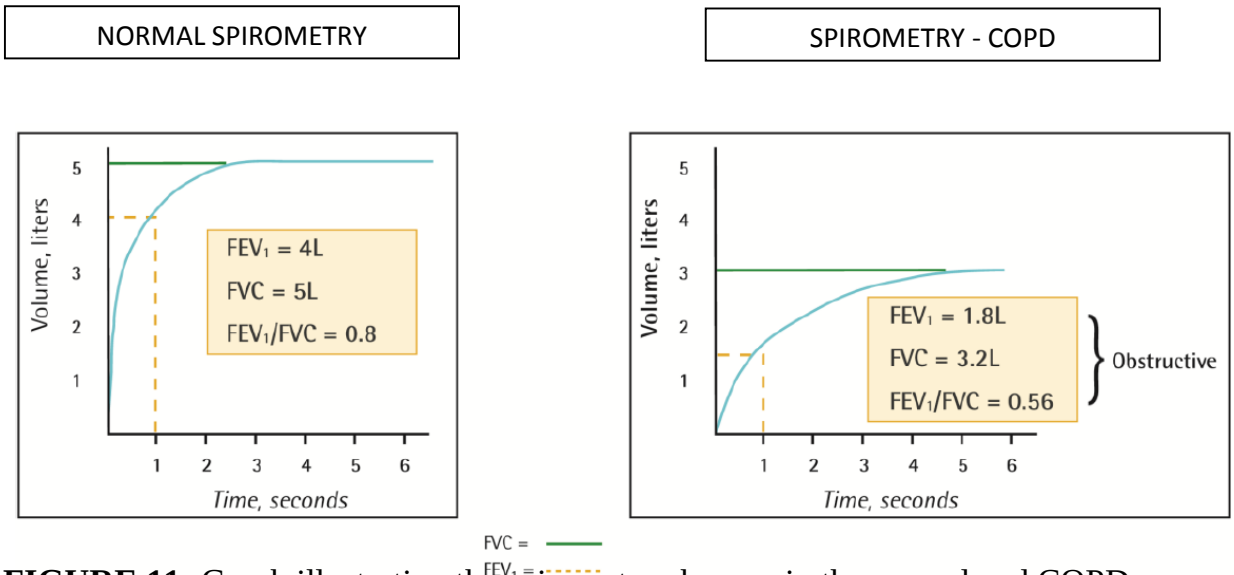



**FIGURE 11:** Graph illustrating the spirometry changes in the normal and COPD cases.

**ASSESSMENT:**

The objectives of COPD assessment are to determine the level of chronic airflow limitation, its influence on patient's health condition and the risk of exacerbation.

**GOLD Classification of severity of airflow limitation:**

The GOLD staging of airflow limitation and severity in COPD is shown in table 2. Exact spirometric cut-points are used for purposes of simplicity. Spirometry should be done after the administration of sufficient dose of at least one short-acting inhaled bronchodilator in order to diminish variability. It should be noted that there is only a weak association between FEV1, signs and symptoms, impairment of a patient's health status.[2]

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

**TABLE 2:** GOLD classification of airflow limitation severity in COPD.

**Assessment of symptoms:**

The Assessment of COPD was observed as a disease mainly characterized by breathlessness. A simple measure of breathlessness such as Modified British Medical Council (mMRC) grading from zero to four (table 3).[49]

**THE MODIFIED MEDICAL RESEARCH COUNCIL (MMRC) DYSPNOEA SCALE**

| GRADING OF DYSPNOEA | DESCRIPTION |
|---|---|
| 0 | Not troubled by breathlessness except on strenuous exercise. |
| 1 | Shortness of breath when hurrying on the level or walking up a slight hill. |
| 2 | Walks slower than people of the same age on the level because of breathlessness or has to stop for breath when walking at own pace on the level. |
| 3 | Stops for breath after walking about 100 meter or after a few minutes on the level. |
| 4 | Too breathless to leave the house or breathless when dressing or undressing |

**TABLE 3:** Table illustrating the Modified Medical Research Council (mMRC) dyspnea assessment scale.

## COPD EXACERBATION:

## DEFINITION OF EXACERBATION:

An exacerbation of COPD is defined as "an event in the natural course of the disease characterized by a change in the patient's baseline dyspnea, cough, and/or sputum and beyond the normal day-to-day variations the symptoms may be noted to occur with acute onset and may permit a change in regular medication in a patient with underlying COPD"[29].

Exacerbations of COPD are of major global importance. They have a profound and long lasting effect on patients, resulting in poor health status; they may accelerate the progression of the disease; and they account for a large proportion of the increasing healthcare spending on COPD. Exacerbations are an important outcome, not only because they pose a considerable economic burden but more importantly because repeated exacerbations of COPD lead to deteriorating health-related quality of life and, when associated with ventilatory failure, to premature death.[29]

There is still debate about how exacerbations should be defined and graded, and their mechanisms are poorly understood. The major causal agents are either bacteria or viral infections, or a combination of the two. Noninfective causes include air pollution and pulmonary embolus but, in some patients, no cause is identified. Exacerbations represent an increase in the inflammation that is present in the stable state, with increased numbers of inflammatory cells (particularly neutrophils), cytokines, chemokines and proteases in the airways, and increased concentrations of certain cytokines and C-reactive protein in the blood. There are presently no reliable biomarkers with which to predict exacerbations.[29]

## CAUSES OF EXACERBATION OF COPD:

The most common causes are bacteria, viruses and environmental agents account for the vast majority of episodes of exacerbation (figure 12). In a recent study of patients admitted to hospital with severe exacerbations, 78% of patients had evidence of either viral or bacterial infection. Other patients had a non-infective cause.[8, 29]

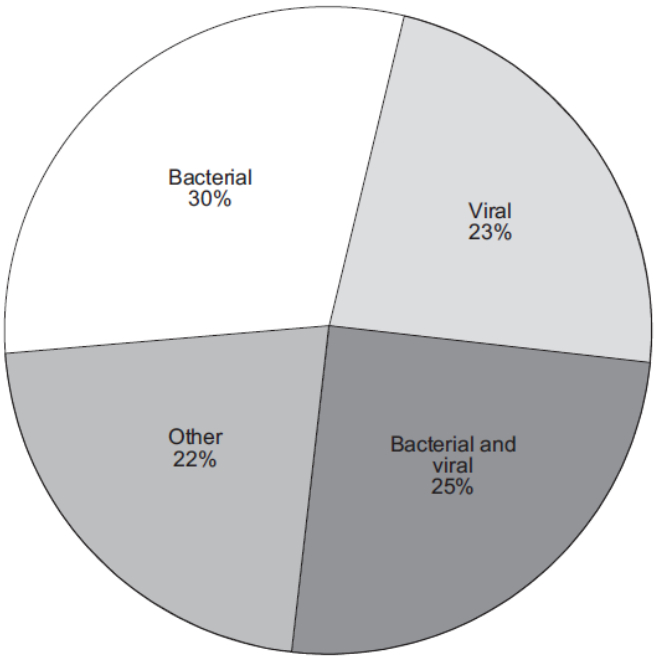



**FIGURE 12:** Pie chart depicting the causative agents of COPD exacerbation.

| Causal mechanism | Common | Less common |
|---|---|---|
| **Bacteria** | *Haemophilus influenzae* | Pseudomonas |
| | *Streptococcus pneumoniaie* | |
| | *Moraxella catarrhalis* | |
| **Virus** | Rhinovirus | Influenza A and B |
| | Respiratory syncytial virus | Parainfluenza virus |
| | | Coronavirus |
| | | Adenovirus |
| **Atypical organisms** | | *Mycoplasma pneumoniae* |
| | | *Chlamydia pneumoniae* |
| **Noninfective** | Air pollution (particulates, ozone) | Pulmonary embolus |
| | Cold temperatures | Congestive cardiac failure |

**TABLE 4:** Table listing out the Causes of chronic obstructive pulmonary disease.

## CLASSIFICATION OF EXACERBATION:

The exacerbation of COPD can be classified as mild, moderate and severe. Severe exacerbation may also be associated with acute respiratory failure. This exacerbation of COPD coexisting with chronic diseases of respiratory system such as asthuma, pneumonia, bronchiectasis, interstitial lung disease and pneumothorax. The COPD exacerbation may also accompany non-respiratory diseases such as hypertension, heart failure, ischemic heart disease, pulmonary embolism, stroke and depression.[8, 29]

## CLASSIFICATION OF COPD EXACERBATION SEVERITY:

- **MILD:** Treated with short acting bronchodilators (SABAs).
- **MODERATE:** Treated with short acting bronchodilators plus antibiotics and/or oral corticosteroids.
- **SEVERE:** (Patient requires hospitalization or visits the emergency room). Severe exacerbations may also be associated with acute respiratory failure.

## ASSESSMENT OF SYSTEMIC MANIFESTATION OF COPD:

Chronic Obstructive Pulmonary Disease is a Multi-Organ Disorder, over the past decade have demonstrated that COPD is associated with multiple nonpulmonary manifestations that contribute significantly to its morbidity and mortality. These associated processes include cardiac and cerebrovascular, oncologic, musculoskeletal, hematologic, psychological, and endocrine effects. The major causes of death in individuals with early

COPD (measured by severity of airflow limitation (AFL)) are lung cancer and cardiac disease. During acute exacerbations of COPD, the leading causes of death are [30]

- Heart failure (37.2%)
- Pneumonia (27.9%)
- Pulmonary hypertension (20.9%)
- Respiratory failure (14%)

The respiratory morbidity and mortality among those with COPD are declining but nonrespiratory morbidity and mortality are increasing. These improvements in respiratory mortality may be due to better pharmacologic and nonpharmacologic management of COPD. The increase in nonrespiratory related mortality suggests that greater identification and management of nonpulmonary processes associated with COPD may be warranted to improve the longevity and health of individuals with COPD.[30]

**SYSTEMIC MANIFESTATION OF COPD**:

The main systemic consequences such as cardiovascular disease, cognitive decline, osteoporosis, muscle dysfunction, anemia, cancer, cachexia and depression may develop in cases of COPD (figure 13).[30]

**CARDIOVASCULAR DISORDERS IN COPD:**

In patients with mild to moderate COPD (forced expiratory volume in one second, FEV1, >60% of predicted), cardiovascular events are the leading cause of hospitalization

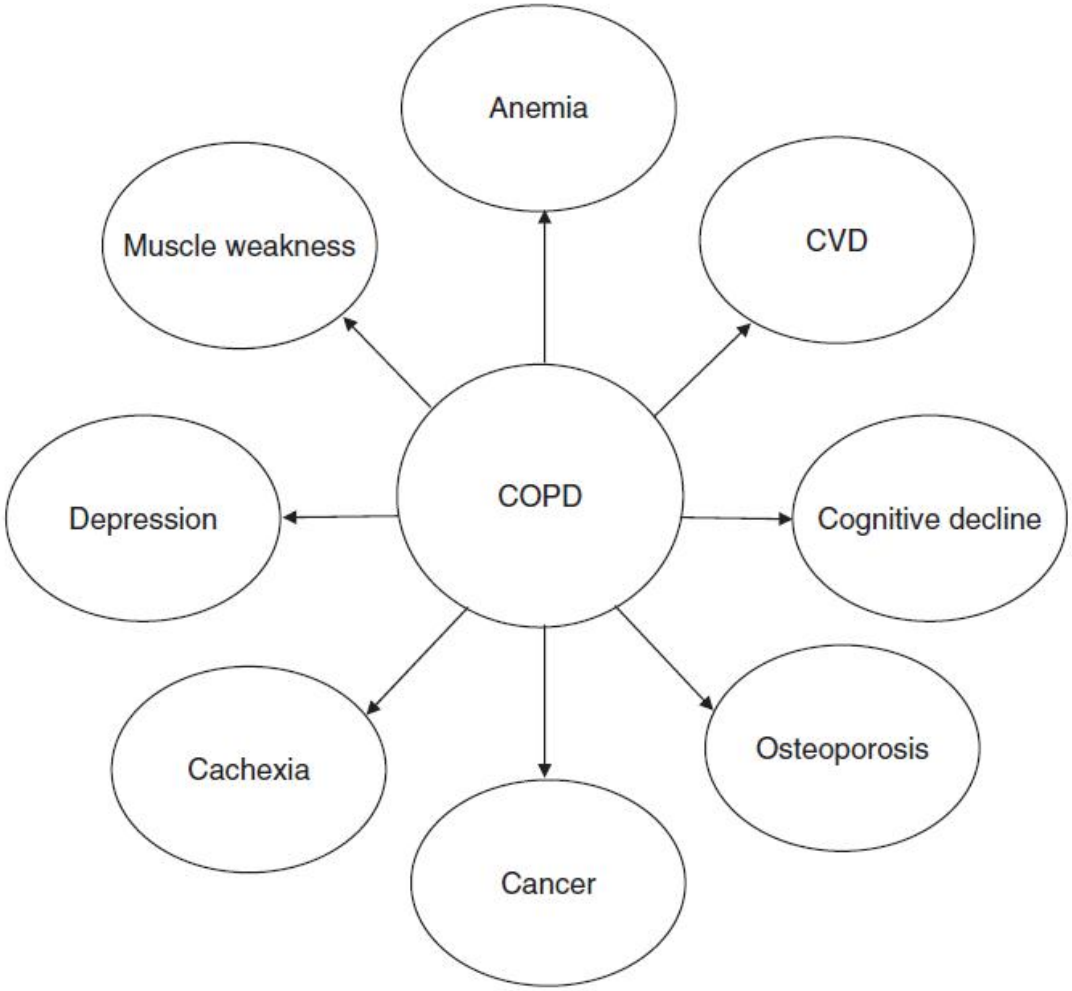



**FIGURE 13:** The Systemic Manifestations of Chronic Obstructive Pulmonary Disease (COPD). Patients with COPD are at increased risk of developing cardiovascular disease (CVD), cancer, osteoporosis, peripheral muscle Weakness, cognitive decline, anemia, cachexia and many other conditions. These systemic conditions contribute significantly to the morbidity and mortality of COPD patients

and the second leading cause of mortality. Among patients with Global Initiative for Chronic Obstructive Lung Disease (GOLD) stages 0 to 2 disease (i.e. FEV1 >50% of predicted), cardiovascular disorders account for approximately 50% of all hospitalizations and nearly a third of all deaths. In more advanced disease, cardiovascular events account

for 20–25% of all deaths in COPD. Sin and colleagues examined data from The First National Health and Nutritional, Examination Survey and showed that subjects in the lowest quintile of FEV1 had over three times the risk of cardiovascular mortality compared to those with the best lung function (figure 14).[31]

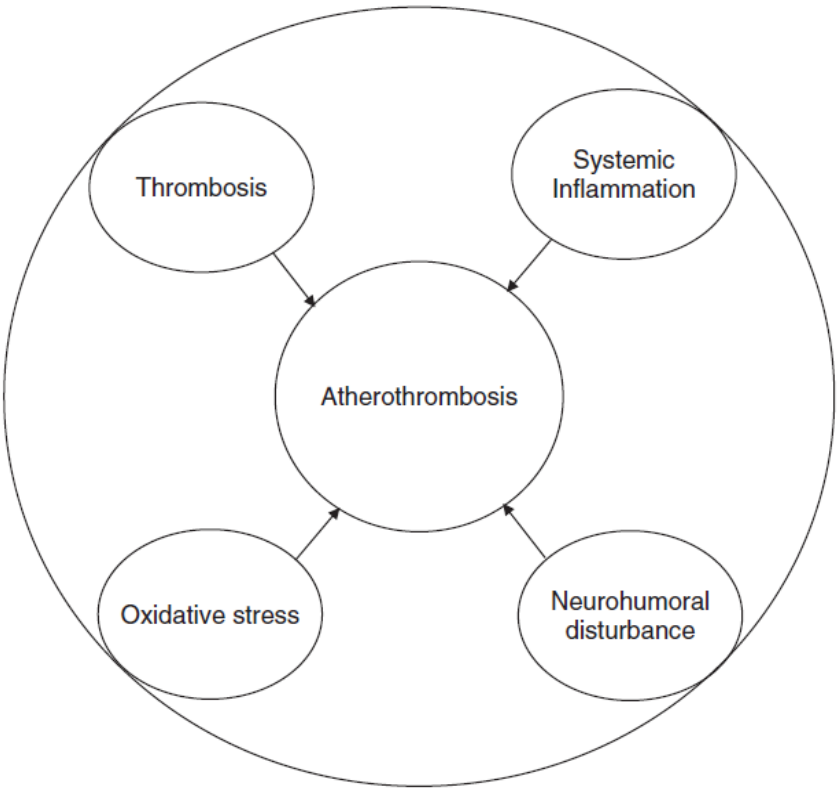



**FIGURE 14:** The Potential Mechanisms by which COPD Patients are at Increased Risk of Cardiovascular Disease .Patients with COPD (especially in moderate to severe disease) demonstrate systemic inflammation, increased oxidative stress, neurohumoral disturbances, and increased thrombotic tendency. All of these factors are mechanistically linked with increased risk of cardiovascular disease in the general community.

**SYSTEMIC INFLAMMATION:**

The first relates to inflammation. It is postulated that in COPD, persistent pulmonary inflammation promotes the release of pro-inflammatory chemokines and cytokines into the

circulation. These mediators then stimulate various end organs including the liver, adipose tissues, and the bone marrow to release excessive amounts of acute-phase proteins, inflammatory cells, and secondary cytokines into the general circulation, resulting in a state of persistent low-grade systemic inflammation. The systemic inflammation in turn adversely impacts the blood vessels, contributing to plaque formation and, in certain cases, to plaque instability and rupture.[31]

**THROMBOSIS:**

Hemostasis and thrombotic pathways may also play relevant roles in COPD and ischemic heart disease. Increased expression of vWF and/or $\alpha 2\beta 1$ integrin might therefore increase the risk for cardiovascular disease. COPD patients have increased circulating levels of thrombin, tissue plasminogen activator– plasminogen activator inhibitor (tPA–PAI) complex, and $\beta$-thromboglobulin, a marker of platelet activation. Increases in the circulating levels of thrombotic factors may be expected to elevate the risk for cardiovascular disease.[31]

**NEUROHUMORAL DISTURBANCES:**

Excess sympathetic nervous activity is significantly related to cardiovascular disease. The intensity of the sympathetic nervous activity was inversely related to the patients' oxyhemoglobin saturation. Supplemental oxygen attenuated (but did not normalize) the sympathetic nervous activity.[31]

## OXIDATIVE STRESS:

Oxidative stress induces endothelial dysfunction. Oxygen-derived free radicals such as superoxide anions impair endothelial vasomotor function. Oxidative stress can impair vasodilation, endothelial cell growth, and promote plaque build-up and rupture. As COPD patients have perturbed oxidant/anti-oxidant balance in favor of oxidative stress, it is plausible that the excess oxidant load could contribute to the development and progression of atherosclerosis and cardiovascular events.[31]

## OTHER SYSTEMIC MANIFESTATION OF COPD:

Other systemic manifestations such as skeletal muscle dysfunction, anemia, cachexia, cancer, depression, cognitive dysfunction and osteoporosis.[30]

## ASSESSMENT OF SYSTEMIC CONSEQUENCE OF COPD ON MORTALITY:

In the past, COPD mortality research has focused on measurements of lung function, especially FEV1. Although FEV1 correlates with survival in COPD, the relationship is rather weak probably because FEV1 does not fully capture the extra-pulmonary manifestations of COPD. The BMI, airflow obstruction, dyspnea, and exercise capacity (BODE) index is an integrated scale that captures respiratory function, cardiovascular fitness, nutritional status, and skeletal muscle performance of COPD patients. The BODE index is a multidimensional instrument that is derived from BMI, FEV1, modified Medical Research Council dyspnea score and 6-minute walk distance. Overall, the BODE index is

much better than FEV1 alone in predicting risk of all-cause and respiratory-cause specific mortality.[8, 30]

## MULTIPLE ORGAN DYSFUNCTION SYNDROME:

The term MODS was introduced in the 1991 ACCP-SCCM Harmony Conference replacing the term "multiple organ failure" because MODS stresses the continuum of organ dysfunction rather than just its result. MODS was defined as ‘‘the presence of altered organ function in an acutely ill patient such that homeostasis cannot be maintained without intervention”. Numerous age related changes occur in the different organ systems of elderly patients. When these aged patients have an attack of COPD, it may cause worsening of the organ function and may ultimately lead to multiple organ dysfunction syndrome (MODS).[33]

## AGE CHANGES IN ELDERLY:

Aging process is an inevitable change that occurs in elderly persons. These age changes occur in the respiratory, cardiovascular, renal, hepatic, hematological and central nervous system. Any acute illness or stress can aggravate and worsen these age related changes in different body systems. Even though the age changes occur in the above mentioned body systems the average life expectancy has improved in the past decade due to health schemes, advanced medical treatments and care provided.[7]

**Respiratory system:**

Aging changes that occur in the respiratory system of elderly persons are:

- Loss of elasticity of lung parenchyma and thoracic wall
- Increased mucosal degeneration, decreased ciliary action, decreased phagocytic activity
- Collagenous replacement of smooth muscles in bronchioles.
- Alveolar air is not exchanged as well with tidal air.
- Decreased forced vital capacity and FEV1.[7]

**Cardiovascular system:**

Age changes seen in the cardiovascular system are as follows:

- Reduction in maximum cardiac output.
- Changes in the activities of nodal and conducting cells
- Replacement of damaged cardiac cells by scar tissue.
- Progressive changes in blood vessels that restrict coronary circulation.
- Changes related to atherosclerosis.
- Reduced β-adrenergic receptor and baroreceptor sensitivity.
- Sclerosis of atrial and mitral valves
- Left ventricular and atrial hypertrophy.[7]

**Renal system:**

Age related changes observed in renal system of elderly patients are as follows:

- Decreased in kidney mass, blood flow and glomerular filtration rate (GFR)
- Decreased number of glomeruli
- Glomerulosclerosis and hyaline arteriosclerosis.
- Tubular atrophy and interstitial fibrosis.[7]

**Hepatobiliary system:**

Changes related to aging seen in the Hepatobiliary system in the elderly patients:

- Gradual decrease in size and weight of the liver, and blood flow.
- Reduced drug metabolism
- Decline in drug clearance capability.
- Decreased gall bladder contraction after a meal
- Increased prevalence of gall stones.[7]

**Hematology:**

Hematological changes on aging:

- Decreased bone marrow reserve.
- Decreased total blood volume
- Decreased RBC production
- Decreased erythropoietin production

- WBC count remains stable except for the lymphocyte count that decreases on aging.[7]

**Central nervous system:**

Age related changes in the central nervous system in the elderly patients are as follows:

- Decline in brain mass and cerebral blood flow.
- Neuronal loss in cortex, midbrain, brainstem, thalamus and dorsal horn neuron.
- Decreased neurotransmitters such as catecholamines, acetyl choline, GABA, serotonin.[7]

**SEPSIS RELATED ORGAN FUNCTION CHANGES:**

Presently, MODS is defined as a clinical syndrome considered by the development of progressive and potentially reversible change of physiological dysfunction in 2 or more organs systems that is induced by a variety of acute insults, including (but not limited to) sepsis. MODS has conventionally been defined in terms of involvement of six organ systems in the order of [33, 34]

1) Pulmonary

2) Hepatic

3) Renal

4) Cardiovascular

5) Central nervous system

6) Haematologic systems.

**PULMONARY:**

The Endothelial injury in the pulmonary vasculature leads to disturbed capillary blood flow and improved microvascular permeability, causing in interstitial and alveolar edema. Neutrophil entrapment inside the pulmonary microcirculation recruits and amplifies the injury to alveolar capillary membranes. Acute lung injury and acute respiratory distress syndrome (ARDS) are frequent manifestations of these effects. Indeed, sepsis and pneumonia are the most common causes of ARDS.[33,34]

**HEPATIC:**

As a consequence of the role the liver plays in host defense, the abnormal synthetic function caused by liver dysfunction can contribute to both the initiation and development of sepsis. The reticuloendothelial system of the liver performances as a first line of defense in clearing bacteria and their products, liver dysfunction leads to spillover of these products into systemic circulation.[33, 34]

Liver failure (‘Shocked liver”) can be manifested by elevations in liver enzymes and bilirubin coagulation defects and failure to excrete toxins such as ammonia which lead to worsening encephalopathy.[33, 34]

**CARDIOVASCULAR SYSTEM:**

The major cardiovascular changes occurring in MODS are: Decreased cardiac output and index, and hemodynamic changes such as increased systemic vascular resistance, right arterial pressure, left ventricular stroke work index and decreased oxygen delivery and consumption by the cardiac tissues.[33]

**RENAL:**

Acute kidney injury [AKI] often accompanies sepsis. Different etiologies for AKI have been reported, and the cause is typically thought to be multifactorial .The mechanism of AKI is complex but likely involves a decrease in effective intravascular volume resulting from systemic hypotension, direct renal vasoconstriction ,release of cytokines ,and activation of neutrophils by endotoxins and other peptides, which contribute to renal injury. Still most animal studies shows that renal blood flow is increased, not decreased, in sepsis, though associated with impaired tubular function and a lack of significant histologic evidence of tubular injury.[33, 34]

**CENTRAL NERVOUS SYSTEM:**

Involvement of central nervous system [CNS] in sepsis produces encephalopathy and peripheral neuropathy. The pathogenesis is poorly defined but is probably related to systemic hypotension, which can lead to brain hypoperfusion.[33, 34]

## HAEMATOLOGY:

Subclinical coagulopathy, signaled by a mild elevation of the thrombin time [TT] or activated partial thromboplastic time [aPTT] or a moderate reduction in the platelet count, is extremely common: however, overt disseminated intravascular coagulation [DIC] may also develop, protease activated receptors [PARs]. Especially PAR 1, form the molecular link between coagulation and inflammation: PAR1 exerts cytoprotective effects when stimulated by activated protein C or low dose thrombin but exerts disruptive effects on endothelial cell barrier function when activated by high dose thrombin.

As per the current understanding, MODS also includes derangements of the endocrine, metabolic, immunologic and gastrointestinal systems, which were not originally included in the description of the syndrome. The early accepted concept of "organ system involvement" regarding the above described six systems have been developed. However, the addition of derangements of other systems such as endocrine, metabolic, immunologic and gastrointestinal systems have not emerged and these have been variously defined.[32]

## PATHOPHYSIOLOGY OF MULTIPLE ORGAN DYSFUNCTION:

Multiple organ dysfunction syndrome (MODS) is caused by an overwhelming, uncontrolled systemic inflammatory response that is activated by a number of hostile stimuli including sepsis, hypovolemic shock, and severe trauma resulting in massive tissue injury. The indiscriminate activation of the inflammatory response due to these insults causes loss of the host's ability to localize the inflammation to the focus of the problem,

leading to systemic inflammation and severe host tissue damage and subsequent MODS. While the major players, namely: [33]

- Neutrophils
- Macrophages
- Endotoxin
- Cytokines
- Oxidants

are known to be aiding in the disease processes responsible for the pathogenesis of MODS.

The lung has been a major focus of research. As a marker organ for the sequence of events. That occur throughout the host during a systemic response to injury. The lung is sensitive to the inflammatory insult and can be monitored closely using precise methods to detect pulmonary dysfunction. Endotoxin, ischemia/ reperfusion insults, and other inflammation mediated causes of acute lung injury begin with a massive cellular inflammatory infiltration of neutrophils within 4 to 6 hours, monocytes within 24 hours, and lymphocytes within 48 hours. The monocyte, once present within the tissue, transforms into a long-lived tissue-fixed macrophage that plays a critical role in perpetuating the systemic inflammatory response syndrome (SIRS) by producing a cascade of proinflammatory mediators such as shown in table 5 & figure 15.[33]

| *Cytokine* | *Abbreviation* |
|---|---|
| Tumor necrosis factor | TNF |
| Interleukins | IL-1<br>IL-8<br>IL-8<br>IL-10 |
| Platelet activating factor | PAF |
| Leukotriene-$B_4$ | $LTB_4$ |
| Prostaglandin E | PGE |
| Thromboxane-$A_2$ | $TxA_2$ |
| Procoagulant activity | PCA |
| Prostacyclin | $PGI_2$ |

**TABLE 5:** Immunomodulatory cytokines expressed by macrophages.

Thus neutrophils, endotoxin, macrophages, cytokines, and other toxic by-products of neutrophils and macrophages play a vital role in the pathogenesis of multiple organ dysfunction syndrome (MODS).[33]

## KEY PATHOGENETIC MECHANISMS:

The key concepts regarding the pathogenetic mechanisms underlying the evolution of MODS include: [33, 34]

Stage 1: Local response

Stage 2: Initial systemic response

Stage 3: Massive systemic inflammation

Stage 4: Excessive immunosuppression

Stage 5: Immunologic dissonance.

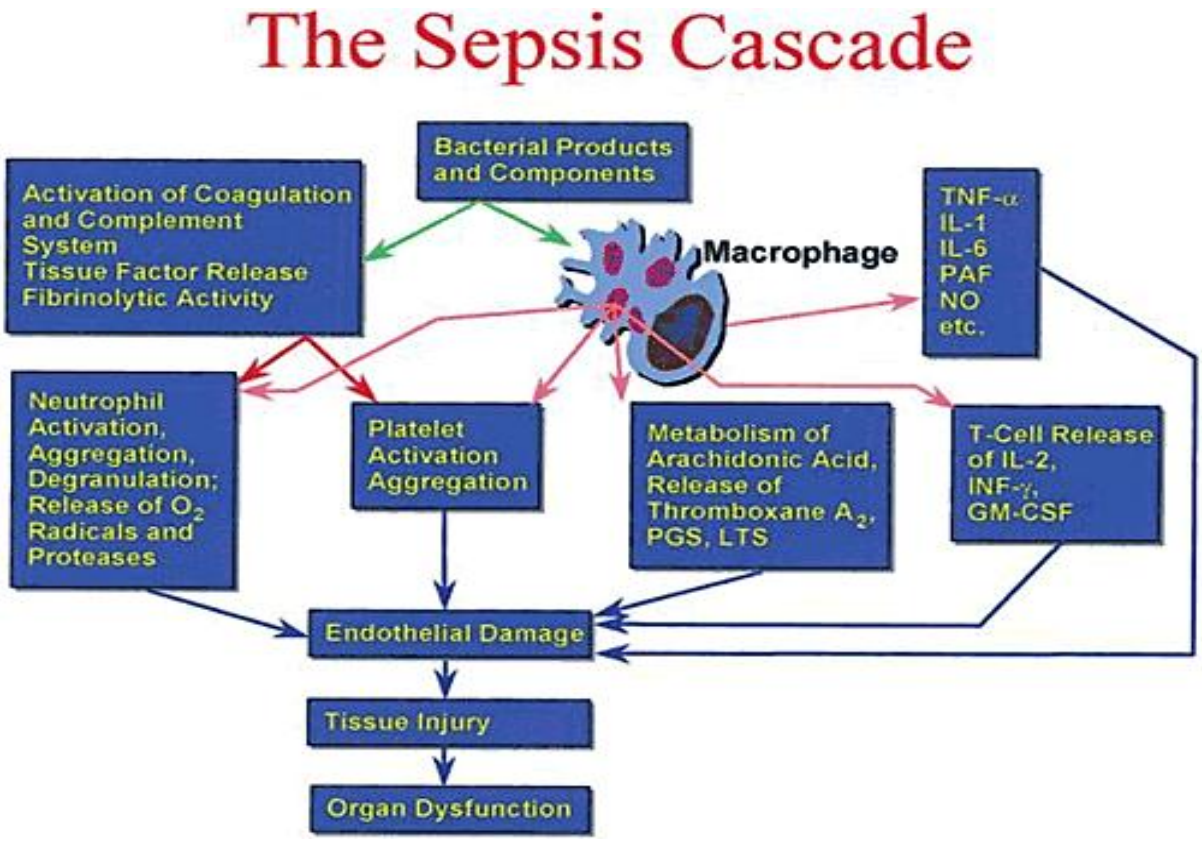



**FIGURE 15:** Flowchart illustrating the pathogenesis of MODS.

**A New Theory:**

The body is designed to compensate for any assault. Its defenses include macrophages and their products, such as tumor necrosis factor; interleukin-1, interleukin-6, and interleukin-8; neutrophils and the products of their degranulation; platelets and the coagulation factors formed on their surfaces; derivatives of arachidonic acid; T and B lymphocytes and their products; and many other substances. Following figure 16 depicts how these agents work together to overcome a severe assault and, paradoxically, how they can cause SIRS and MODS.[33]

**Stage 1:**

Begins at a site of local injury or infection. Proinflammatory mediators are released locally to promote wound healing and to combat foreign organisms or antigens. Anti-inflammatory mediators are then released to down regulate this process. If the original insult is small and the patient is healthy, homeostasis will be quickly restored.

**Stage 2:**

Occurs if local defense mechanisms are insufficient to correct the local injury or eliminate the local infection. Through various mechanisms, proinflammatory mediators are released into the systemic circulation; these recruit additional cells to the local area of injury. Systemic release of anti-inflammatory mediators follows soon thereafter; under normal circumstances, these mediators ameliorate the proinflammatory reaction and restore homeostasis.[33]

**Stage 3:**

Occurs if the systemic release of proinflammatory mediators is massive or if the anti-inflammatory reaction is insufficient to permit down regulation. It is at this stage that most patients have symptoms of the systemic inflammatory response syndrome (SIRS), as well as incipient evidence of the multiple organ dysfunction syndrome (MODS).[33]

**Stage 4:**

It can be represented by excessive systemic levels of anti-inflammatory mediators that develop as a response to a massive proinflammatory response; however, these levels can also develop de novo. Patients with a stage 4 compensatory anti-inflammatory response syndrome (CARS) response have marked immunosuppression and thus are at increased risk for infection. If the body can reestablish homeostasis after stage 3 or 4, the patient may survive.[33]

**Stage 5:**

It is the final stage of MODS. At this stage of immunologic dissonance, the balance between pro- and anti-inflammatory mediators has been lost. Some patients may have persistent, massive inflammation; others may have ongoing immunosuppression and secondary infections. Still others may oscillate between periods of inflammation and immunosuppression.

Various hypotheses such as oxygen delivery hypothesis, gut-origin hypothesis, two-hit hypothesis, mitochondrial dysfunction hypothesis have been postulated to explain the genesis of MODS. However, they have been disappointing when translated into clinical therapies for MODS.[33]

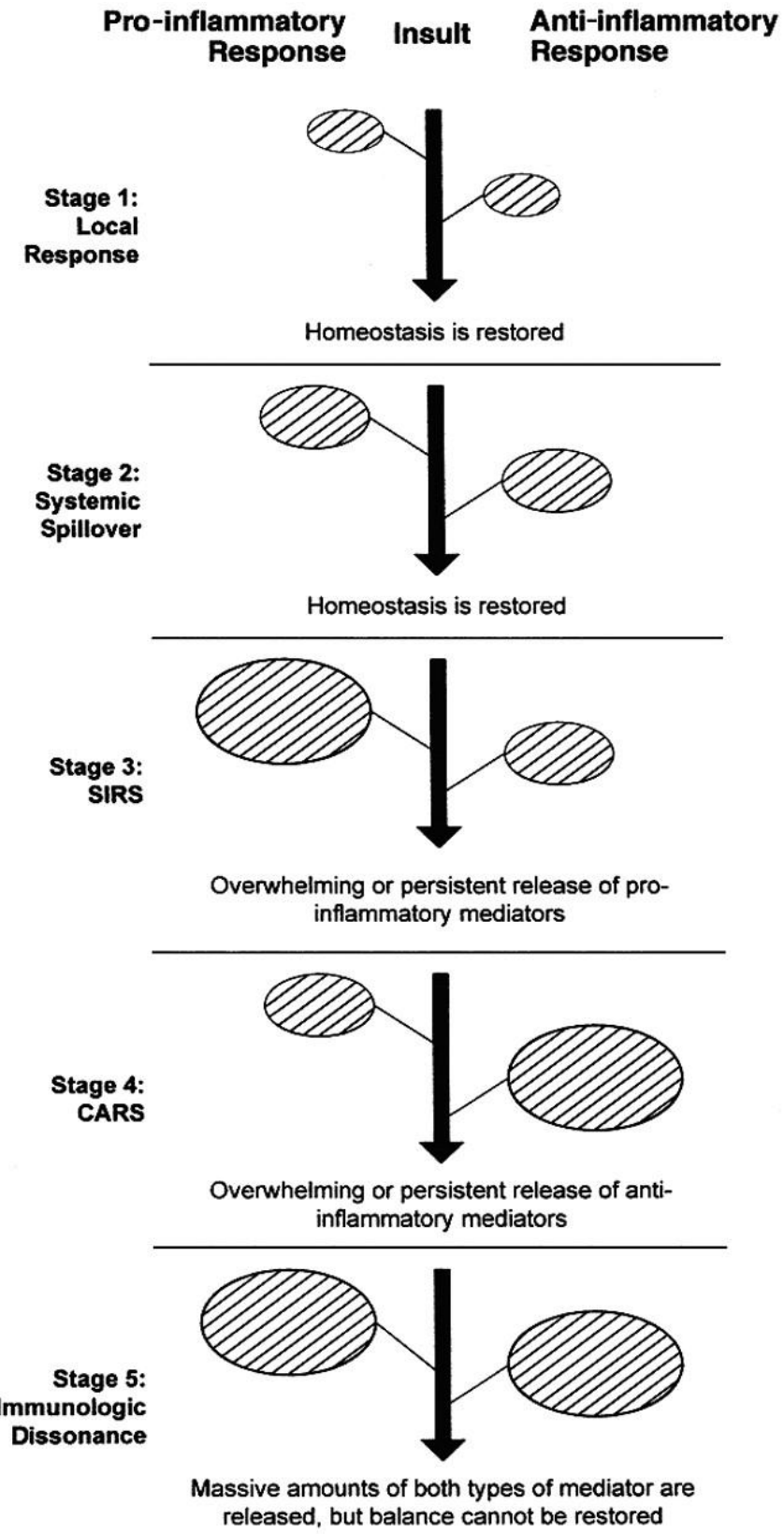



**FIGURE 16:** Flowchart depicting evolution of initial response to MODS.

**HYPOTHESIS OF MODS:**

Literature reveals three hypothesis for the development of MODS. They are

i) Gut Hypothesis

ii) Two-hit hypothesis

iii) Mitochondrial dysfunction [57]

**Gut hypothesis:**

This hypothesis was proposed by Deitch to explain MODS in critically ill patients. This hypothesis stats that due to splanchnic hypo perfusion and the subsequent mucosal ischemia there are structural changes and alterations in cellular function. This results in increased gut permeability, changed immune function of the gut and increased translocation of bacteria. Liver dysfunction leads to toxins escaping into the systemic circulation and activating an immune response. This results in tissue injury and organ dysfunction (figure 17). [57]

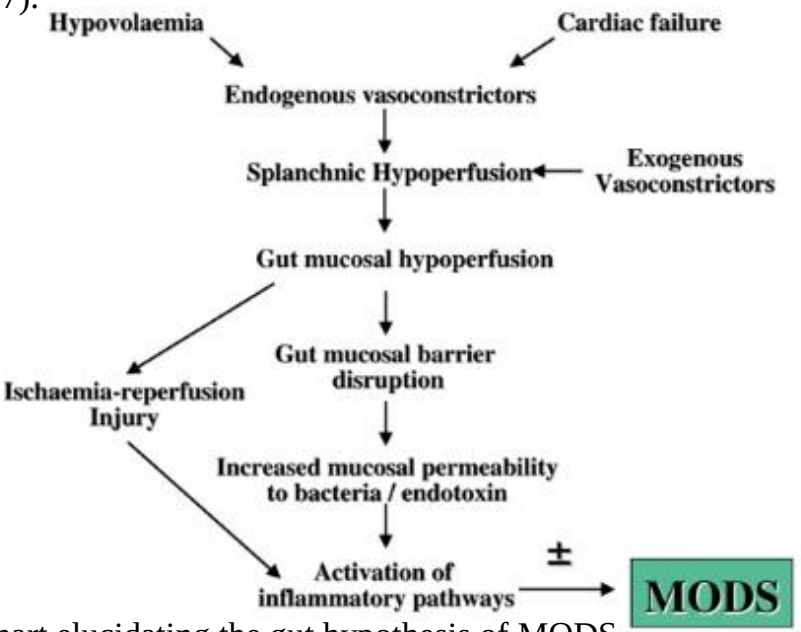



**FIGURE 17:** Flowchart elucidating the gut hypothesis of MODS.

**Two-hit hypothesis:**

In this hypothesis, the first hit occurs after sepsis or trauma activates neutrophils, macrophages, and the cytokine cascade that leads to lung injury and other organ damage. Blocking neutrophil adherence and migration may serve to inhibit initial organ damage and prevent the inflammation "priming" that occurs prior to a second hit. The second hit activates tissue-fixed macrophages to produce inflammatory mediators that sustain SIRS and eventually lead to MODS and death. Cyclic hypovolemia-induced ischemia/reperfusion, nosocomial pneumonia, endotoxemia, or release of toxic by-products from injured tissue can cause this second hit (figure 18).[58]

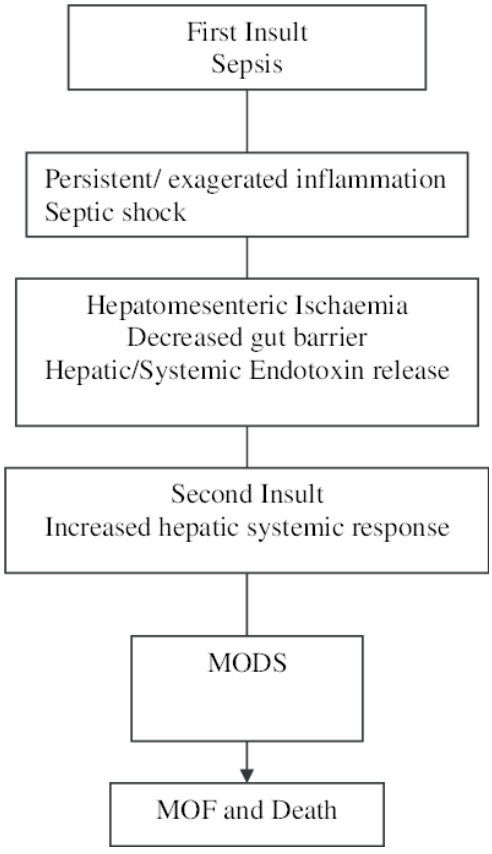



**FIGURE 18:** Flowchart explaining the two-hit hypothesis of MODS.

**Mitochondrial DNA hypothesis:**

According to findings of Professor Zsolt Balohh et al, the mitochondrial DNA is the leading cause of severe inflammation due to a massive amount of mitochondrial DNA that leaks into the blood stream due to cell death of patients that survived major trauma. Mitochondrial DNA resembles bacterial DNA. If bacteria triggers leukocytes, mitochondrial DNA may do the same. When confronted with bacteria, white blood cells, or neutrophil granulocytes, behave like predatory spiders. They spit out a web, or net, to trap the invaders, then hit them with a deadly oxidative blast, forming neutrophil extracellular traps (NETs). This results in catastrophic immune response leading to multiple organ dysfunction syndrome (figure 19).[58]

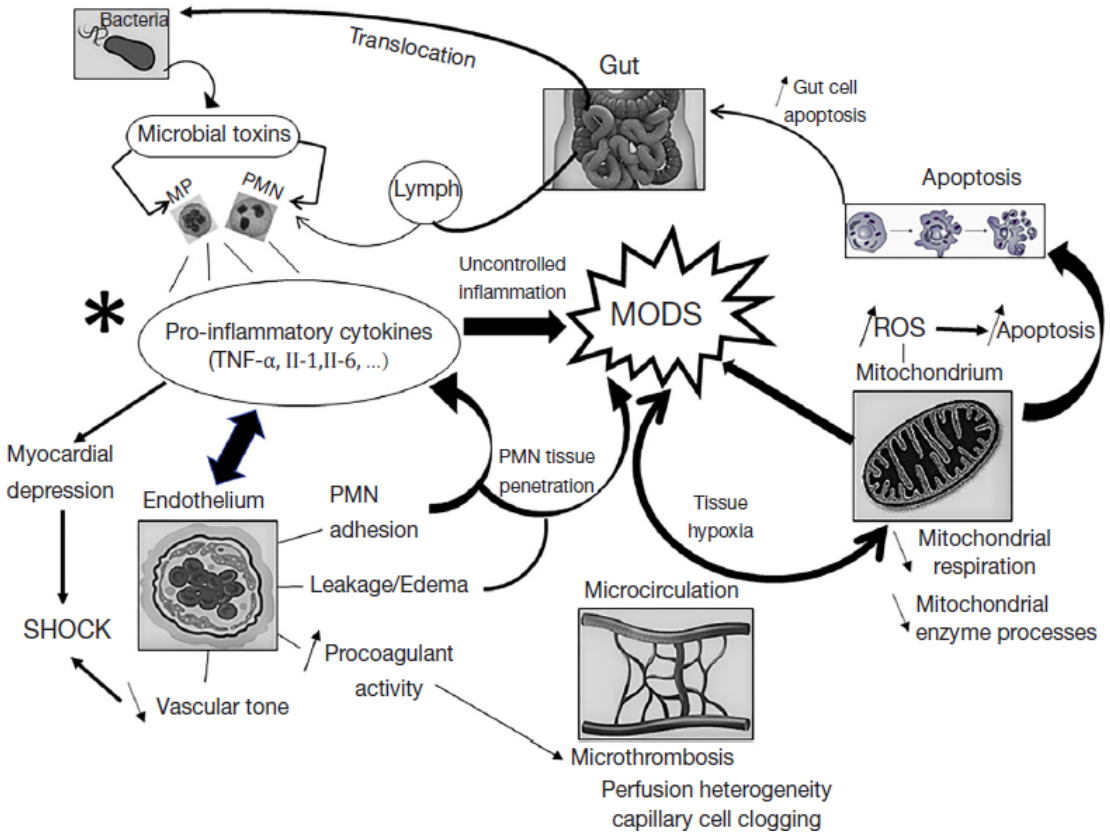



**FIGURE 19:** Flowchart depicting all three hypothesis involved in the development of MODS.

**APACHE (acute physiology and chronic health evaluation):**

Severity- of- illness classification system began in 1978 with the specific goal of developing a measure for use in describing groups of intensive care unit (ICU) patients and evaluating their care. ICU's receive patients with a wide variety of diagnoses and severity of illness, and it is difficult for one ICU physician to precisely describe his case mix to another. Diagnoses are necessary but not sufficient.[37]

Because APACHE was designed for the evaluation of efficacy of medical treatment, the timing, quality, and type of data collected have been different than in research principally oriented toward hospital reimbursement. The most important difference is that all of the severity and diagnostic data have been collected early in the course of each patient's hospital stay, within 24 hours of ICU admission, rather than after hospital discharge. In medical research terminology, this has been a prospective observational study, not retrospective chart review.[37]

The APACHE score is the best –known and most widely used score with good calibration and discrimination. The original APACHE score was developed in 1981 to classify groups of patients according to the severity of illness and divided into two sections: **Physiology score** to assess the degree of acute illness and **Preadmission evaluation** to

determine the chronic health status of the patient. The original APACHE score consists of 34 physiologic measures (0-4).[38]

The APACHE II scoring system was released in 1985. The APACHE II was based on 12 of the most commonly measured physiologic measures included in the original APACHE system. The APACHE II scoring system is measured during the first 24 hours of ICU admission with a maximum score of 71. A score of 25 represents a predicted mortality of 50% and a score of over 35 represents a predicted mortality of 80%. The APACHE II score is sum of:

- Acute physiology score
- Age
- Chronic health score [38]

**Acute physiological score:**

The acute physiology score include 12 variables include vital signs (heart rate, mean blood pressure, respiratory rate, temperature, and Glasgow coma score), variables derived from routine venous blood tests (hematocrit and white blood cell count, serum potassium, serum sodium, and serum creatinine),and 2 variables derived from arterial blood gas tests (serum pH and Pao2) (table 6 & 7).[38]

| Physiologic Variable | High Abnormal Range | | | | | | Low Abnormal Range | | |
|---|---|---|---|---|---|---|---|---|---|
| | +4 | +3 | +2 | +1 | 0 | +1 | +2 | +3 | +4 |
| Rectal Temp (°C) | ≥41 | 39-40.9 | | 38.5-38.9 | 36-38.4 | 34-35.9 | 32-33.9 | 30-31.9 | ≤29.9 |
| Mean Arterial Pressure (mmHg) | ≥160 | 130-159 | 110-129 | | 70-109 | | 50-69 | | ≤49 |
| Heart Rate | ≥100 | 140-179 | 110-139 | | 70-109 | | 50-69 | 40-54 | ≤39 |
| Respiratory Rate | ≥50 | 35-49 | | 25-34 | 12-24 | 10-11 | 6-9 | | ≤5 |
| Oxygenatation<br>a) $FIO_2 \geq 0.5$ record A-a$DO_2$<br>b) $FIO_2 < 0.5$ record $PaO_2$ | ≥500 | 350-499 | 200-349 | | <200<br>$PO_2$>70 | $PO_2$ 61-70 | | $PO_2$ 55-60 | $PO_2$<55 |
| Arterial pH | ≥7.7 | 7.6-7.69 | | 7.5-7.59 | 7.33-7.49 | | 7.25-7.32 | 7.15-7.24 | <7.15 |
| $HCO_3$ (mEq/l) | ≥52 | 41-51.9 | | 32-40.9 | 22-31.9 | | 18-21.9 | 15-17.9 | <15 |
| K (mEq/l) | ≥7 | 6-6.9 | | 5.5-5.9 | 3.5-5.4 | 3-3.4 | 2.5-2.9 | | <2.5 |
| Na (mEq/l) | ≥100 | 160-179 | 155-159 | 150-154 | 130-149 | | 120-129 | 111-119 | ≤110 |
| S. Creat (mqm/dl) | ≥3.5 | 2-3.4 | 1.5-1.9 | | 0.6-1.4 | | <0.6 | | |
| Hematocrit (%) | ≥60 | | 50-59.9 | 46-49.9 | 30.45.9 | | 20-29.9 | | <20 |
| TLC ($10^3$/cc) | ≥40 | | 20-39.9 | 15-19.9 | 3-14.9 | | 1-2.9 | | <1 |
| GCS | | | | | | | | | |

**TABLE 6:** Physiological variables of APACHE II score.

| BEHAVIOR | RESPONSE | SCORE |
|---|---|---|
| Eye opening response | Spontaneously | 4 |
| | To speech | 3 |
| | To pain | 2 |
| | No response | 1 |
| Best verbal response | Oriented to time, place, and person | 5 |
| | Confused | 4 |
| | Inappropriate words | 3 |
| | Incomprehensible sounds | 2 |
| | No response | 1 |
| Best motor response | Obeys commands | 6 |
| | Moves to localized pain | 5 |
| | Flexion withdrawal from pain | 4 |
| | Abnormal flexion (decorticate) | 3 |
| | Abnormal extension (decerebrate) | 2 |
| | No response | 1 |
| Total score: | *Best response* | 15 |
| | *Comatose client* | 8 or less |
| | *Totally unresponsive* | 3 |

| | | | |
|---|---|---|---|
| 15 – 0 | 12 – 3 | 9 – 6 | 6 -- 9 |
| 14 – 1 | 11 – 4 | 8 – 7 | 5 -- 10 |
| 13 – 2 | 10 – 5 | 7 – 8 | 4 – 11 |
| | 3 – 12 | | |

**TABLE 7:** Table explaining the assessing scores of Glasgow coma scale and its corresponding score given in APACHE II scoring system.

**Age score:**

The aging score for APACHE II scoring are:

* <44 – 0
* 45 -54 --- 2
* 55 – 64 --- 3
* 65 – 74 --- 5
* ≥ 75 --- 6

**Chronic health condition:**

The chronic health condition scores contributing the APACHE II scoring system are:

| HISTORY OF SEVERE ORGAN INSUFFICIENCY | POINTS |
|---|---|
| Non-operative patients | 5 |
| Emergency post-operative patients | 5 |
| Elective post-operative patients | 2 |

**APACHE III SCORE:**

The APACHE III scoring system released in 1991, it was developed with the objectives of improved statistical power, ability to predict the individual patient outcome and identify the factors in ICU that influence outcome variations but it is far more complex than the two previous scoring system.[39]

**APACHE IV SCORE:**

The APACHE IV scoring system was published in 2006. It is more complex as it contains 142 variables. It is a web-based calculations. It is developed and validated in ICUs of USA only.

Therefore because of the reliability, simplicity and credibility of APACHE II score, it is widely used in ICUs for assessing the severity and predicting the prognosis of the patients admitted.[39]

**SEPSIS RELATED ORGAN FAILURE ASSESSMENT (SOFA):**

The Sequential Organ Failure Assessment (SOFA) score is a scoring system that assesses the performance of several organ systems in the body (neurologic, blood, liver, kidney, and blood pressure/hemodynamics) and assigns a score based on the data obtained in each category. The higher the SOFA score, the higher the likely mortality (figure 20).[41]

**Limitations:**

Because SOFA was designed to look at populations, and not individual patients, it cannot accurately predict which patients will survive when the mortality rate is high (i.e., if mortality is 90%, which 10 patients will survive) or which patients will die if the mortality rate is low. Some of the factors used in scoring can be difficult to assess depending on the care being provided (e.g., it is difficult to assess a level of coma when a patient is receiving sedatives) and some of the medications listed are no longer used routinely (e.g., low dose dopamine or dobutamine).[42]

| SOFA score | 0 | 1 | 2 | 3 | 4 |
|---|---|---|---|---|---|
| **Respirationa** $PaO_2/FIO_2$ (mm Hg) $SaO_2/FIO_2$ | >400 | <400<br>221–301 | <300<br>142–220 | <200<br>67–141 | <100<br><67 |
| **Coagulation** Platelets $10^3/mm^3$ | >150 | <150 | <100 | <50 | <20 |
| **Liver** Bilirubin (mg/dL) | <1.2 | 1.2–1.9 | 2.0–5.9 | 6.0–11.9 | >12.0 |
| **Cardiovascular**[b] Hypotension | No hypotension | MAP <70 | Dopamine </=5 or dobutamine (any) | Dopamine >5 or norepinephrine </=0.1 | Dopamine >15 or norepinephrine >0.1 |
| **CNS** Glasgow Coma Score | 15 | 13–14 | 10–12 | 6–9 | <6 |
| **Renal** Creatinine (mg/dL) or urine output (mL/d) | <1.2 | 1.2–1.9 | 2.0–3.4 | 3.5–4.9 or <500 | >5.0 or <200 |

**FIGURE 20**: SOFA scoring criteria in assessing six vital organ functions.

**QUICK SOFA SCORE:**

The Quick SOFA Score (quick SOFA or qSOFA) was introduced by the Sepsis-3 group in February 2016 as a simplified version of the SOFA Score as an initial way to identify patients at high risk for poor outcome with an infection. The SIRS Criteria definitions of sepsis are being replaced as they were found to possess too many limitations; the "current use of 2 or more SIRS criteria to identify sepsis was unanimously considered by the task force to be unhelpful." The qSOFA simplifies the SOFA score drastically by only including its 3 clinical criteria and by including "any altered mentation" instead of requiring a GCS ≤13. qSOFA can easily and quickly be repeated serially on patients (table 8).[44]

| Assessment | qSOFA score |
|---|---|
| Low blood pressure (SBP ≤ 100 mmHg) | 1 |
| High respiratory rate (≥ 22 breaths/min) | 1 |
| Altered mentation (GCS <=13) | 1 |

**TABLE 8:** Quick SOFA scoring system assessing three vital organs.

The score ranges from 0 to 3 points. The presence of 2 or more qSOFA points near the onset of infection was associated with a greater risk of death or prolonged intensive care unit stay. These are outcomes that are more common in infected patients who may be septic than those with uncomplicated infection. Based upon these findings, the Third International Consensus Definitions for Sepsis recommends qSOFA as a simple prompt to identify infected patients outside the ICU who are likely to be septic.

**DIFFERENCE BETWEEN APACHE AND SOFA SCORE:**

| FEATURE | APACHE | SOFA |
|---|---|---|
| Basis | Three factors that influence outcome in critically ill patients<br>1. Chronic background disease<br>2. Patient reserve<br>3. Severity of acute illness | Degree of organ dysfunction is related to acute illness(initially based on sepsis related organ dysfunction but later validated for organ dysfunction not related to sepsis) |
| Score | Physiological variable, chronic health conditions, emergency / elective admissions, and post-operative / non-operative admissions | Defined score (1-4) for each of six organ systems<br>1.Respiratory<br>2. CVS<br>3. CNS<br>4. Renal<br>5. Coagulation and<br>6. Liver |
| **Scoring duration** | Based on the most abnormal measurements in the first 24 hours of ICU stay | Daily scoring of individual and composite scores possible during course of ICU stay |
| **Population outcome comparison** | Standardized mortality ratios can be used for large patient populations | No predicted mortality algorithm. In general, higher SOFA score is associated with worse outcome. Treatment effects on SOFA |
| **Individual patient outcomes** | Not possible to predict individual patient outcome or response to therapy | Response of organ dysfunction to therapy can be followed over time |

# MATERIALS AND METHODS

# MATERIALS AND METHODS

## SETTING:

Older persons aged 60 and above admitted in Geriatric Intensive Care Unit (GICU) and Intensive Medical Care Unit (IMCU) in the Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai.

## ETHICAL COMMITTEE APPROVAL:

Ethical committee clearance obtained from the Institutional Ethical Committee of Madras Medical College as per the meeting held in June 2017.

## STUDY DESIGN:

This is a cross – sectional observational study.

## STUDY PERIOD:

July 2017 to June 2018 for a period of one year.

## CONSENT:

Consent was obtained from all patients who participated in the study.

**STUDY POPULATION:**

100 elderly patients aged 60 years and above admitted with Acute Exacerbation of COPD in Geriatric Intensive Care Unit (GICU) and Intensive Medical Care Unit (IMCU) in the Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai.

**INCLUSION CRITERIA:**

* Should be admitted to the hospital with history of acute exacerbation of COPD with any organ dysfunction.
* The patient developed MODS during hospitalization, the definition of which was proposed by American college of chest physician [ACCP].

**EXCLUSION CRITERIA:**

* Patients with Acute respiratory distress syndrome (ARDS)
* Patients suffering massive Pulmonary Embolism (PE)
* Patients diagnosed with Cancer such as [Lung, GIT…]
* Patients with respiratory failure due to reason other than COPD.

**DETAILS OF THE STUDY:**

Persons aged 60 years and above admitted with Acute Exacerbation of COPD in geriatric intensive care unit [GICU], and Intensive medical care unit [IMCU] are included in the study. Persons who are giving informed written consent for the study are selected as per inclusion and exclusion criteria. The baseline data of 100 patients (CBC, RFT, LFT, ABG, GCS and serum electrolytes) collected as per proforma, the length of hospital stay, number of failing organ during hospitalization and number of exacerbations occurred in the past 6 months or one year were calculated and then by applying prognostic scoring system of SOFA and APACHE II and thereby categorizing them as survivors and non-survivors.

**STATISTICAL ANALYSIS:**

- Data were expressed as the mean ± standard deviation (SD) (normal distribution) or as the median (interquartile range for non-normal distributions) for continuous variables and as percentages for categorical variables.
- Student's *t*-test was used to compare normally distributed data between two groups (SOFA & APACHE II).
- Pearson correlation was used to evaluate the correlation between two groups (SOFA & APACHE II).
- The qualitative results were evaluated using the chi-square test.

* The predictive values of the scoring systems were analyzed using a receiver operating characteristic (ROC) curve, and the sensitivity, specificity, area under the curve (AUC) and 95% confidence intervals (95% CI) of each scoring system were calculated.

# RESULTS

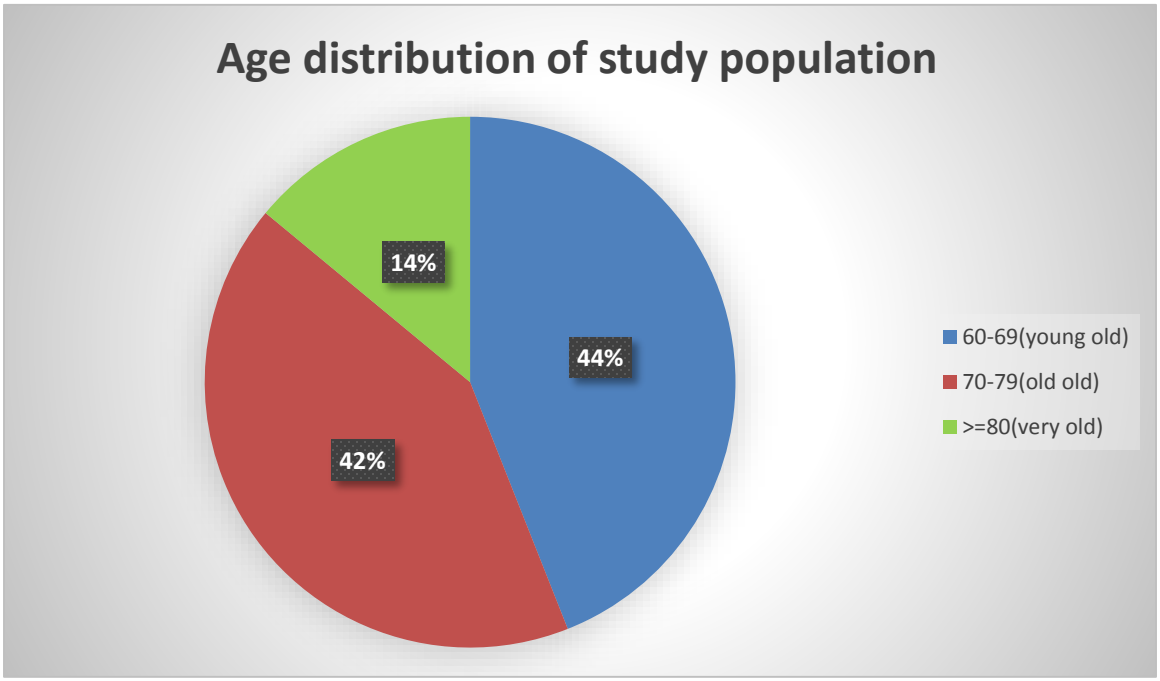
# RESULTS

A cross-sectional and observational study was conducted in a population of 100 patients who are aged 60 and above and were admitted with Acute Exacerbation o COPD in the Geriatric Intensive Care Unit (GICU) of the Department of Geriatric Medicine and Intensive Medical Care Unit (IMCU) of the Department of General Medicine with a diagnosis of acute exacerbation of COPD, with any organ failure at the time of admission and developed Multiple Organ Dysfunction Syndrome (MODS) during hospitalization. Patient selection was done by convenience sampling. The study was conducted for a period of one year (July 2017 – June 2018).

In this study, the study population were categorized into young old, old-old and very old comprising of 44 patients, 42 patients and 14 patients respectively in each group. Among the study population 66 patients were male and 34 patients were female. The patients were also categorized based on the number of exacerbation of COPD prior to hospitalization, number of failing organs at the time of admission and developed during the hospital stay and the length of hospital stay of the patients. The general outcome and prognosis among different categories were assessed using prognostic scoring models such as SOFA and APACHE II score. One third of the study population had severe COPD associated with MODS and suffered death. Among the non-survivors the most failing organs were cardiac and respiratory systems.

**AGE WISE DISTRIBUTION OF STUDY POPUATION:**

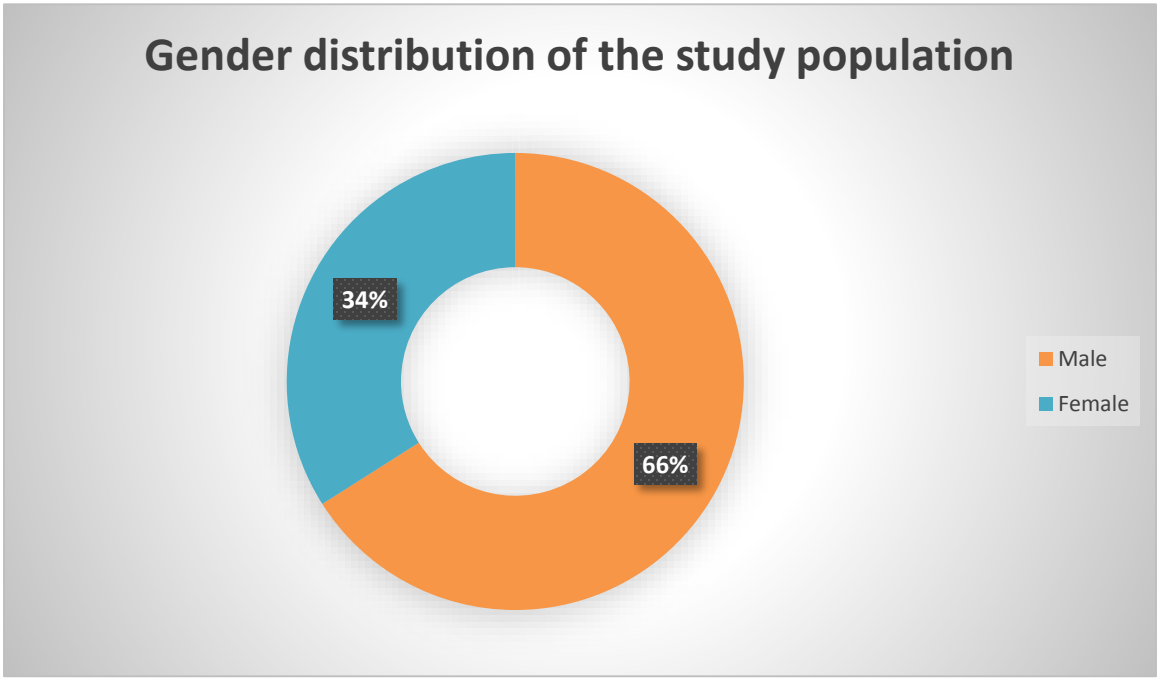
| Age Group | Frequency | Percentage |
|---|---|---|
| 60-69(young old) | 44 | 44 |
| 70-79(old old) | 42 | 42 |
| >=80(very old) | 14 | 14 |
| Total | 100 | 100 |




Of the 100 subjects studied, 44 patients were in the age group of 60-69 years, 42 patients were in the age group of 70-79 years and 14 patients were in the age group of more than 80 years of age.

**GENDER DISTRIBUTION AND SMOKING HISTORY OF THE STUDY POPULATION:**

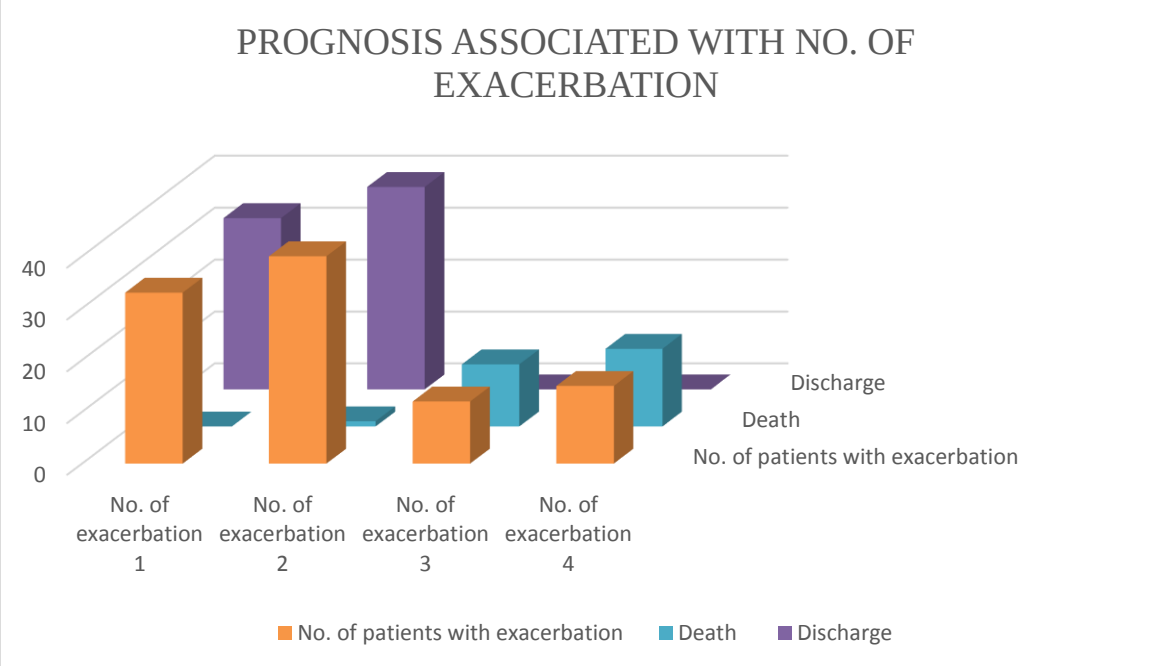
| Gender | Frequency | Smokers | Non- smokers |
|---|---|---|---|
| Male | 66 | 66 | - |
| Female | 34 | - | 34 |
| Total | 100 | 66 | 34 |




Of the 100 study population 66 patients were male and 34 patients were female. On analyzing the personal history of the patients all 66 males included in the study were smokers and all 34 females were non-smokers.

**NUMBER OF EXACERBATION AMONG THE STUDY POPULATION:**

| No. of exacerbation | No. of patients with exacerbation | Death | Discharge |
|---|---|---|---|
| 1 | 33 | - | 33 |
| 2 | 40 | 1 | 39 |
| 3 | 12 | 12 | - |
| 4 | 15 | 15 | - |
| Total | 100 | 28 | 72 |

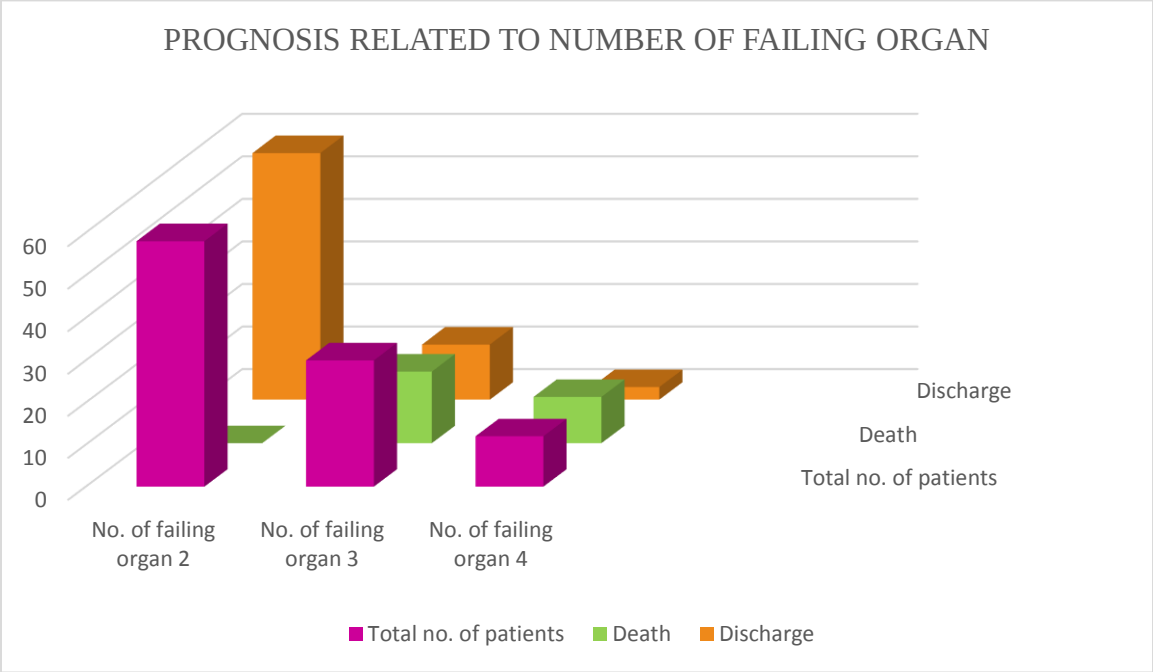



Among the 100 study population, 33 patients had 1 exacerbation during the period of 6months or 1 year before hospitalization, 40 patients had 2 exacerbation, 12 patients had 3 exacerbation and 15 patients had 4 exacerbation. The patients who has 3 or 4 exacerbation

had a worst prognosis and all expired. Among the patients with 1 exacerbation there was no death and among the patients with 2 exacerbation suffered 1 death. Therefore increase in number of exacerbation turned down the prognosis.

**NUM BER OF FAILING ORGAN:**

| No. of failing organ | Total no. of patients | Death | Discharge |
|---|---|---|---|
| 2 | 58 | 0 | 58 |
| 3 | 30 | 17 | 13 |
| 4 | 12 | 11 | 1 |
| Total | 100 | 28 | 72 |




Among the 100 study population, 58 patients had 2 organ failure, 30 patients had 3 organ failure and 12 patients had 4 organ failure. Among the patients with 2 organ failure no death was noted. Amidst the patients with 3 and 4 organ failure, 17 and 12 death was

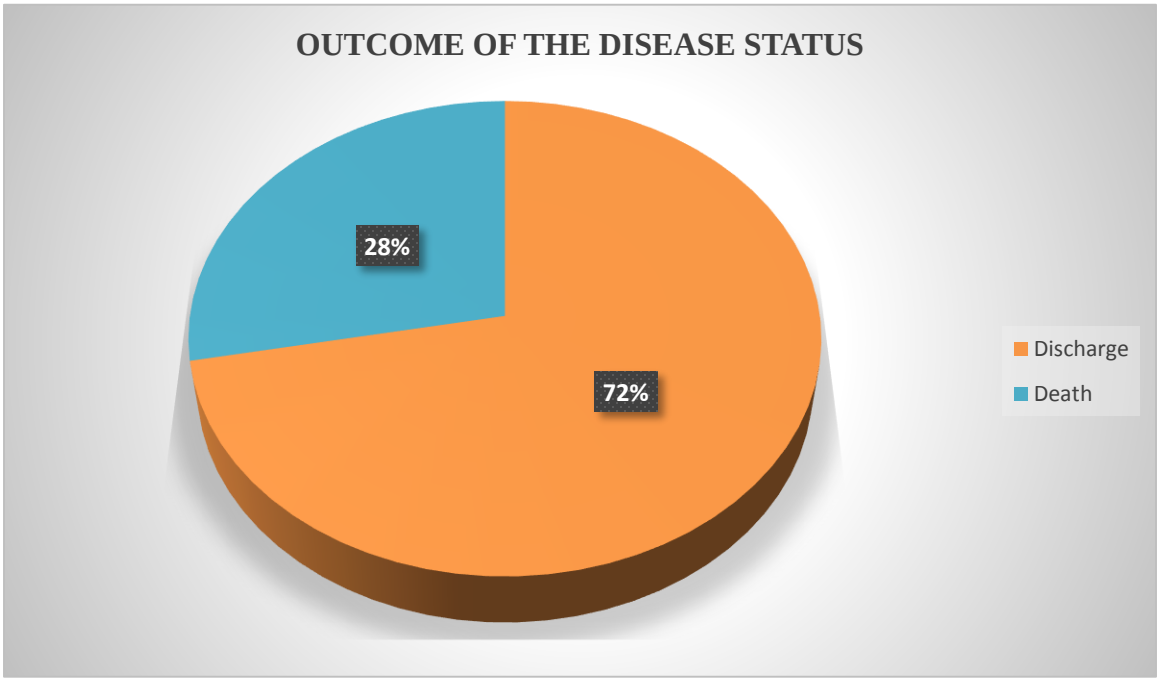
noticed respectively. When there is increase in number of failing organs more death was observed.

**CORRELATION BETWEEN LENGTH OF STAY AND OUTCOME OF THE PATIENTS:**

**Group Statistics**

| | Outcome | N | Mean | Std. Deviation | Std. Error Mean | t value |
|---|---|---|---|---|---|---|
| Length of stay | Discharge | 72 | 12.6389 | 2.12501 | .25043 | 8.710** |
| | Death | 28 | 8.7500 | 1.64711 | .31127 | |

**p<0.001




This table computes the outcome of the disease status to the length of stay of the hospitalized patients. The table shows that the average length of stay of 72 patients who

were discharged had a mean hospital stay of 13 days, whereas shorter length of hospital stay with the mean of 9 days was noticed in patients who expired. According to the statistical analysis, this relationship between the length of hospital stay and outcome of the disease status was found statistically significant (P<0.0010).

## CALCULATION OF MEAN VALUE OF DAY 3 SOFA AND APACHE II SCORE ALONG WITH THEIR SENSITIVITY AND SPECIFICITY:

| Coordinates of the Curve | | | |
|---|---|---|---|
| Test Result Variable(s) | Positive if Greater Than or Equal To[a] | Sensitivity | 1 - Specificity |
| Sofa score on day 3 | 1.0000 | 1.000 | 1.000 |
| | 2.5000 | .893 | .792 |
| | 3.5000 | .821 | .514 |
| | 4.5000 | .786 | .333 |
| | 5.5000 | .786 | .278 |
| | 6.5000 | .786 | .208 |
| | **7.5000** | **.786** | **.194** |
| | 8.5000 | .750 | .167 |
| | 10.5000 | .750 | .125 |
| | 12.5000 | .643 | .083 |
| | 13.5000 | .571 | .083 |
| | 14.5000 | .464 | .083 |
| | 15.5000 | .250 | .069 |
| | 16.5000 | .107 | .014 |
| | 18.0000 | .036 | .000 |
| | 20.0000 | .000 | .000 |

| | | | |
|---|---|---|---|
| | 11.0000 | 1.000 | 1.000 |
| | 12.5000 | .964 | .986 |
| | 13.5000 | .929 | .986 |
| | 14.5000 | .893 | .847 |
| | 15.5000 | .857 | .722 |
| | 16.5000 | .821 | .611 |
| | 17.5000 | .786 | .458 |
| | 18.5000 | .750 | .403 |
| | 19.5000 | .714 | .333 |
| | 20.5000 | .714 | .306 |
| | 21.5000 | .679 | .236 |
| Apache II score on 3 day | **22.5000** | **.607** | **.194** |
| | 24.5000 | .607 | .167 |
| | 29.5000 | .571 | .167 |
| | 34.0000 | .536 | .167 |
| | 37.5000 | .500 | .153 |
| | 40.5000 | .464 | .139 |
| | 41.5000 | .393 | .111 |
| | 43.5000 | .357 | .111 |
| | 45.5000 | .321 | .097 |
| | 46.5000 | .214 | .069 |
| | 47.5000 | .143 | .069 |
| | 49.0000 | .071 | .069 |
| | 51.0000 | .000 | .000 |

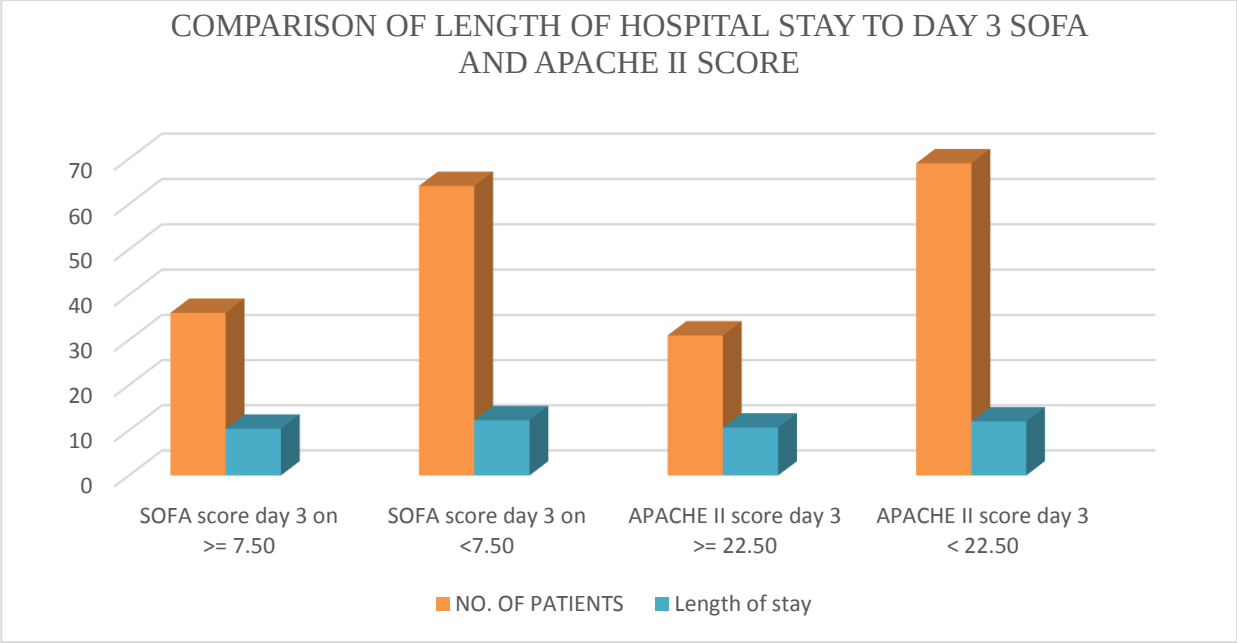
The above table computes the mean predictive value for the SOFA score to discriminate the prognosis of the patients is 7.50 which had high sensitivity and specificity. In the same manner, the mean predictive value for the APACHE II score to calculate the prognosis of the patients is 22.50 and the value had high sensitivity and specificity.

**COMPARISON OF LENGTH OF STAY FOR SOFA AND APACHE SCORE:**

**Comparison of length of stay for sofa and apache score for survivors and non survivors (100 Cases):**

| Independent Samples Test | | | | | | | |
|---|---|---|---|---|---|---|---|
| | Apache II score on 3 day | N | Mean | Std. Deviation | Std. Error Mean | t value | p value |
| length_of_stay | >= 22.50 | 31 | 10.6129 | 2.90606 | .52194 | 2.422* | 0.017 |
| | < 22.50 | 69 | 11.9710 | 2.44330 | .29414 | | |

| Independent Samples Test | | | | | | | |
|---|---|---|---|---|---|---|---|
| | Sofa score on day 3 | N | Mean | Std. Deviation | Std. Error Mean | t value | p value |
| length_of_stay | >= 7.50 | 36 | 10.3333 | 2.75681 | .45947 | 3.641** | p<0.001 |
| | < 7.50 | 64 | 12.2344 | 2.35529 | .29441 | | |

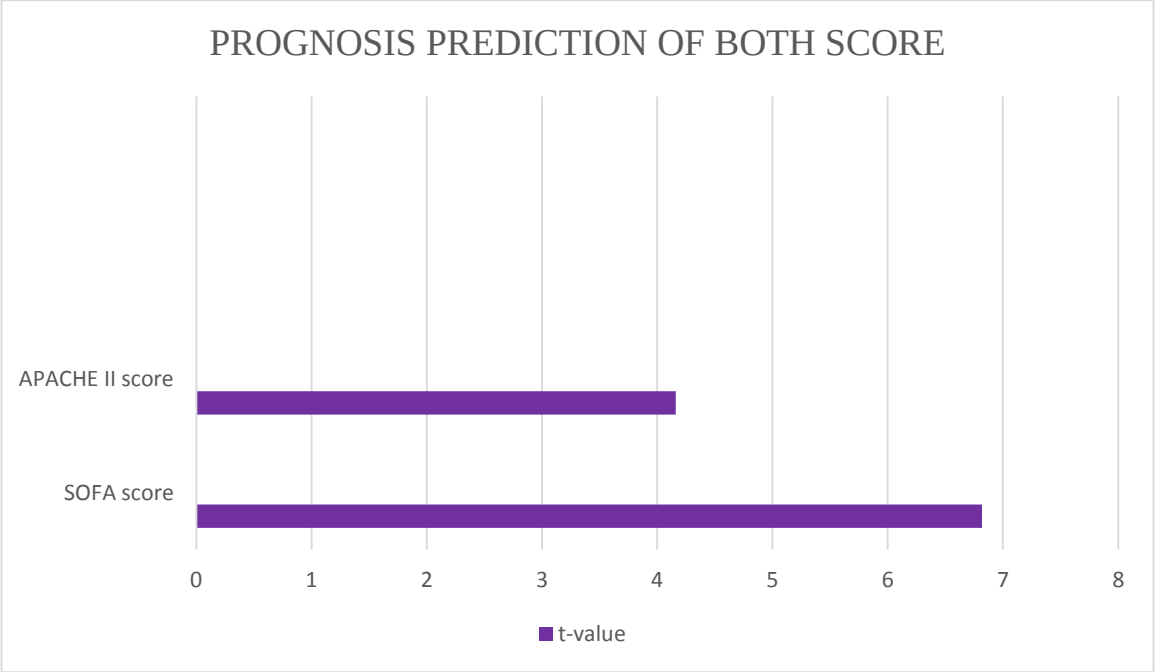



This comparison between the length of hospital stay to the day 3 final score of SOFA and APACHE II score indicates that in case of SOFA score 7.50 was taken as an average score. Patients with SOFA score value more than or equal to 7.50 had a worst prognosis. Therefore their length of stay in hospital was 10± 2 days. Whereas who had the score below 7.50 had a better prognosis and their hospital stay of about 12± 2 days which was little longer compared to the patients with the SOFA score > 7.50 and the difference was statistically significant with the P value <0.001 when compared to APACHE II .

In the similar manner, the average day 3 APACHE II overall score was calculated as 22.50. The patients who had the score more than or equal to 22.50 suffered multiple organ failure and had a reduced number of hospital stay of about 10 ±2 days due to worst prognosis. Whereas those patients who had score below 22.50 had a better prognosis and had a lengthy stay in hospital of about 11± 2 days.

## PROGNOSTIC PREDICTION OF SOFA AND APACHE II SCORE:

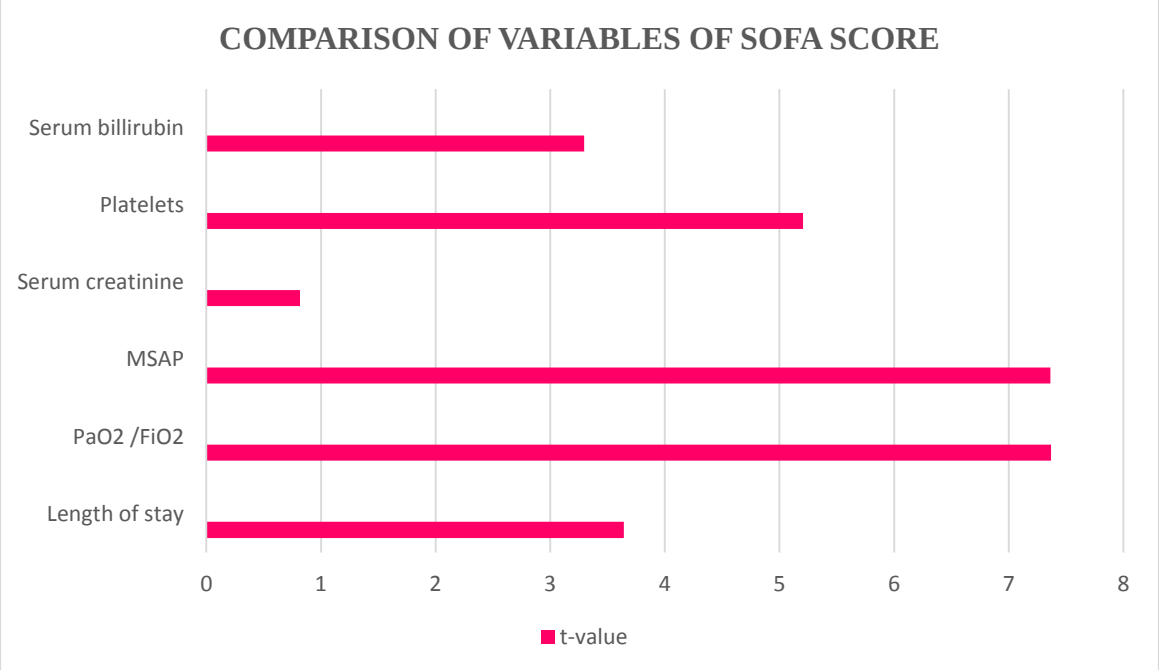
| Group Statistics | | | | | | |
|---|---|---|---|---|---|---|
| | Outcome | N | Mean | Std. Deviation | Std. Error Mean | t VALUE |
| Sofa score on day 3 | Discharge | 72 | 5.1944 | 4.06856 | .47948 | 6.817** |
| | Death | 28 | 11.9643 | 5.35054 | 1.01116 | |
| Apache II score on 3 day | Discharge | 72 | 21.8056 | 11.05765 | 1.30316 | 4.160** |
| | Death | 28 | 32.8571 | 13.95685 | 2.63760 | |




This table and graph calculates the effectiveness of the scores in predicting the prognosis of the patients with acute exacerbation of COPD. Among the two scores, SOFA score which assess six essential body systems predicts the prognosis of the patients accurately with a t-value of 6.817 compared to the APACHE II score which has a t-value of 4.160. The APACHE II includes numerous variables and are difficult to calculate.

## COMPARISION OF PREDICTIVE VALUES OF DIFFERENT VARIABLES OF SOFA SCORE:

| COMPARISON OF VARIABLES FOR SOFA SCORE | | | | | | | |
|---|---|---|---|---|---|---|---|
| Sofa score on day 3 | | N | Mean | Std. Deviation | Std. Error Mean | t value | p value |
| Length of stay | >= 7.50 | 36 | 10.33 | 2.76 | 0.46 | 3.641** | p<0.001 |
| | < 7.50 | 64 | 12.23 | 2.36 | 0.29 | | |
| $PaO_2$ / $FiO_2$ | >= 7.50 | 36 | 214.22 | 136.24 | 22.71 | 7.370** | p<0.001 |
| | < 7.50 | 64 | 391.75 | 102.38 | 12.80 | | |
| Mean Systolic arterial pressure | >= 7.50 | 36 | 74.17 | 19.48 | 3.25 | 7.362** | p<0.001 |
| | < 7.50 | 64 | 107.66 | 23.04 | 2.88 | | |
| Serum Creatinine | >= 7.50 | 36 | 2.56 | 1.03 | 0.17 | 0.817 | 0.416 |
| | < 7.50 | 64 | 2.40 | 0.94 | 0.12 | | |
| Platelets | >= 7.50 | 36 | 153.69 | 77.10 | 12.85 | 5.204** | p<0.001 |
| | < 7.50 | 64 | 260.65 | 108.80 | 13.60 | | |
| Serum Bilirubin | >= 7.50 | 36 | 2.31 | 1.40 | 0.23 | 3.297** | p<0.001 |
| | < 7.50 | 64 | 1.39 | 1.31 | 0.16 | | |

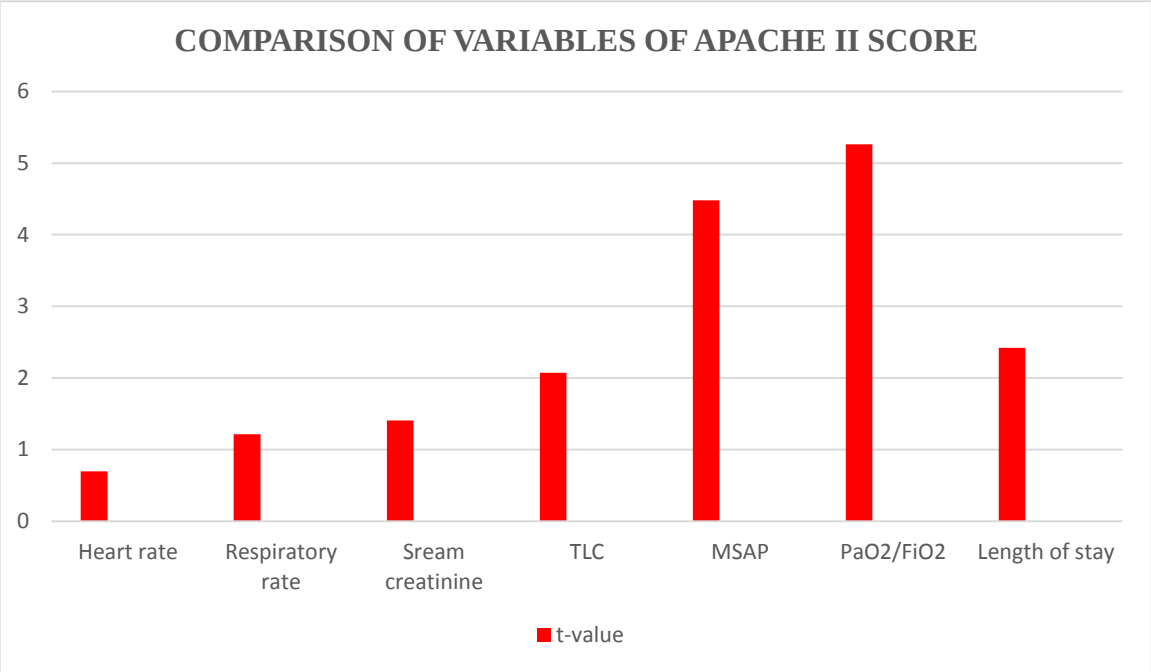


The above picturized table and graph assess the different variables used in the prognosis assessing SOFA score. The statistical analysis states that among the multiple variables of SOFA score, assessment of length of stay, PaO2/ FiO2, Mean arterial systemic pressure, platelets and Serum bilirubin were significantly predicting the prognosis.

## COMPARISON OF PREDICTIVE VALUES OF DIFFERENT SCORES OF APACHE II SCORE:

| COMPARISON OF VARIABLES FOR APACHE II SCORE | | | | | | | |
|---|---|---|---|---|---|---|---|
| Apache II score on 3 day | | N | Mean | Std. Deviation | Std. Error Mean | | |
| Length of stay | >= 22.50 | 31 | 10.61 | 2.91 | 0.52 | 2.422* | .017 |
| | < 22.50 | 69 | 11.97 | 2.44 | 0.29 | | |
| Heart rate | >= 22.50 | 31 | 115.77 | 6.60 | 1.18 | 0.696 | .488 |
| | < 22.50 | 69 | 114.77 | 6.73 | 0.81 | | |
| Respiratory rate | >= 22.50 | 31 | 34.45 | 2.81 | 0.51 | 1.213 | .228 |
| | < 22.50 | 69 | 33.54 | 3.75 | 0.45 | | |
| Serum Creatinine | >= 22.50 | 31 | 2.12 | 0.62 | 0.11 | 1.404 | .163 |
| | < 22.50 | 69 | 2.45 | 1.25 | 0.15 | | |
| Total leucocyte count | >= 22.50 | 31 | 18138.71 | 3539.65 | 635.74 | 2.071* | .041 |
| | < 22.50 | 69 | 15992.75 | 5249.97 | 632.02 | | |
| PaO2/FiO2 | >= 22.50 | 31 | 227.94 | 144.99 | 26.04 | 5.261** | p<0.001 |
| | < 22.50 | 69 | 372.72 | 118.63 | 14.28 | | |
| MSAP | >= 22.50 | 31 | 79.03 | 26.25 | 4.71 | 4.479** | p<0.001 |
| | < 22.50 | 69 | 103.04 | 24.12 | 2.90 | | |
| Serum Creatine | >= 22.50 | 31 | 2.54 | 0.91 | 0.16 | 0.578 | .565 |
| | < 22.50 | 69 | 2.42 | 1.00 | 0.12 | | |

The table and graph indicates the different variables used in assessing the predictive value of APACHE II score. The statistical analysis of different variables of APACHE II score states that among the 13 variables, only the PaO2/FiO2 and Mean systemic arterial pressures plays a significant role in predicting the prognosis of the patients and are statistically significant.



**CORRELATION OF THE DIFFERENT VARIABLES OF THE SOFA AND APACHE II SCORE ALONG WITH THE LENGTH OF HOSPITAL STAY:**

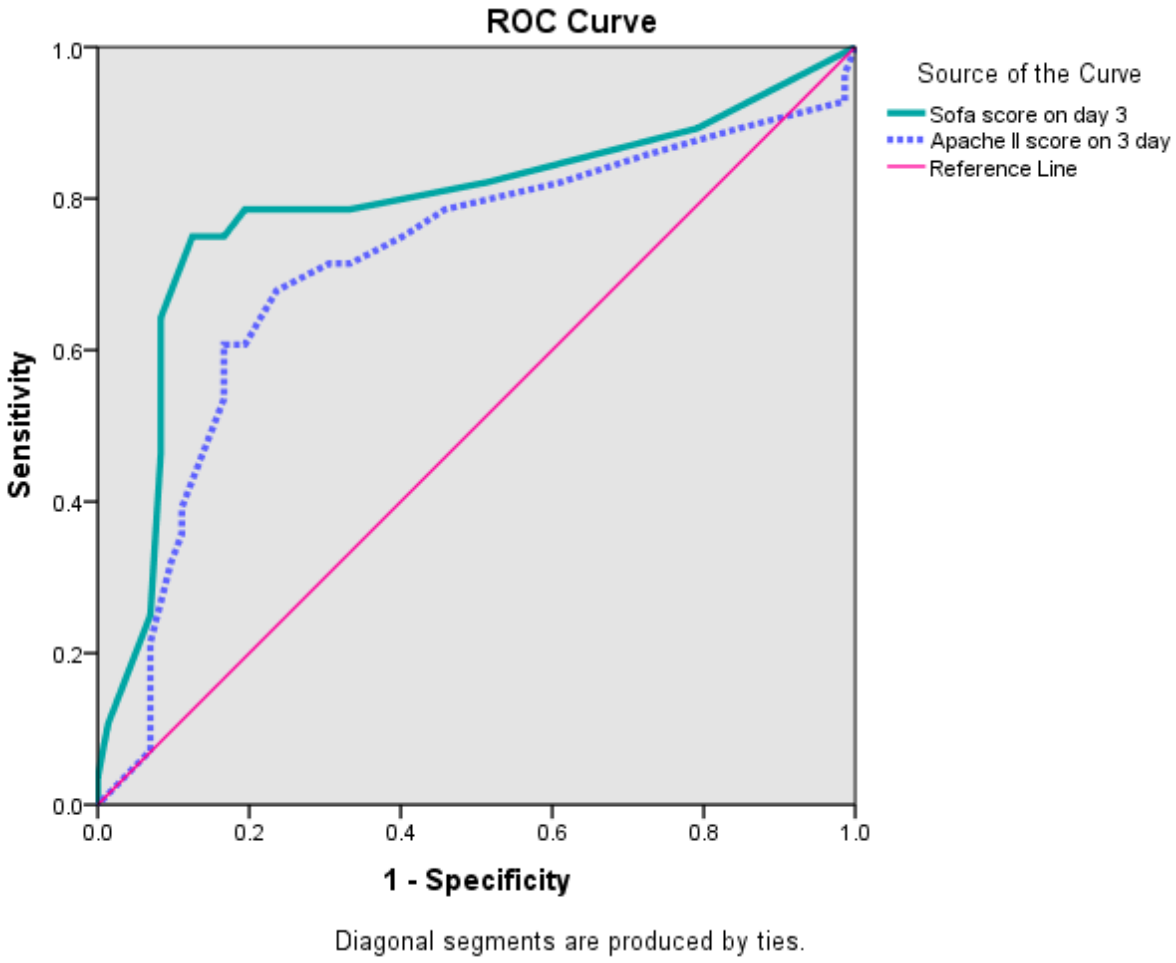
| | | LHS | HR | RR | Sr. creatinine | TLC | Platelets | Sr. Bilirubin | MSAP | PaO2/FiO2 |
|---|---|---|---|---|---|---|---|---|---|---|
| Sofa score on day 3 | Pearson Correlation | -.320** | .020 | .226* | -.013 | .097 | -.508** | .380** | -.668** | -.186 |
| | P value | .001 | .841 | .024 | .901 | .338 | .000 | .000 | .000 | .064 |
| Apache II score on 3 day | Pearson Correlation | -.222* | .093 | .121 | -.125 | .210* | -.349** | .301** | -.379** | -.229* |
| | P value | .027 | .356 | .230 | .216 | .036 | .000 | .002 | .000 | .022 |
| length_of_stay | Pearson Correlation | 1 | -.187 | -.259** | .199* | -.042 | -.013 | .096 | .113 | .270** |
| | P value | | .063 | .009 | .047 | .679 | .895 | .340 | .262 | .007 |

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

The table depicts the Pearson correlation of different variable used in assessing the prognosis of acute exacerbation of COPD patients with MODS. The statistical analysis states that assessment of respiratory rate, platelet count, serum bilirubin, mean systolic arterial pressure and PaO2/FiO2 ratio which has a statistically significant p-value (<0.05) plays a critical role in diagnosing the present status of the patient and thereby predicting the prognosis of the case.

**ROC CURVE:**




Diagonal segments are produced by ties.

**Area Under the Curve**

| Test Result Variable(s) | Area | Std. Error[a] | Asymptotic Sig.[b] | Asymptotic 95% Confidence Interval | |
|---|---|---|---|---|---|
| | | | | Lower Bound | Upper Bound |
| Sofa score on day 3 | .787 | .059 | .000 | .670 | .903 |
| Apache II score on 3 day | .711 | .063 | .001 | .587 | .835 |

The Area under the Curve i.e., AUC. It is used in classification analysis in order to determine which of the used models predicts the classes best. An example of its application is ROC curves.

A Receiver Operating Characteristic curve, i.e., ROC curve, is a graphical plot that illustrates the diagnostic ability of a binary classifier system as its discrimination threshold is varied. The ROC curve is created by plotting the true positive rate against the false positive rate at various threshold settings. The receiver operating characteristic (ROC) curve is a plot of test sensitivity as the y coordinate versus its 1-specificity or false positive rate (FPR) as the x coordinate, is an effective method of evaluating the performance of diagnostic tests.

Though apache and sofa predicts outcome of Acute Exacerbation of COPD with MODS, when compared to APACHE II score (71%) (With Confidence interval of 59%-84%) SOFA (79%) with Confidence interval (67% – 90%) is best predictor for outcome.

# DISCUSSION

# DISCUSSION

Our study is a hospital based cross- sectional and observational study of assessing the prognostic value of APACHE II and SOFA score in acute exacerbation of Chronic Obstructive Pulmonary Disease (COPD) with Multiple Organ Dysfunction Syndrome (MODS).

In this study, 100 patients of acute exacerbation of COPD with any organ dysfunction were chosen from the patients admitted in the intensive care unit with COPD based on inclusion and exclusion criteria.

Patients were assessed by the prognostic scoring systems of COPD such as SOFA and APACHE II score based on different variables such as number of exacerbation, rectal temperature, mean arterial pressure, heart rate, respiratory rate, oxygen saturation, PaO2/ FiO2, arterial pH, HCo3, Serum electrolytes, Serum bilirubin, Serum creatinine, Platelets, Hematocrit, Total leucocyte count, Glasgow coma scale, age score and chronic health point that are calculated in the on the day 1 and day 3 and average total scores were calculated. Both the scores were statistically analyzed by independent sample test, Pearson Chi square test, Pearson correlation, Receiver Operating Characteristic (ROC) curve and Area under Curve (AUC) and the best score was identified for predicting the prognosis of patients with acute exacerbation of Chronic Obstructive Pulmonary Disease (COPD) with Multiple Organ Dysfunction Syndrome (MODS).

Among the 100 study population, 66 percentages were contributed by males and 34 percentages were by females. Males were affected more than females probably due to higher exposure to toxic substances of tobacco products such as cigarette smoking and other types of tobacco (e.g., pipe, cigar, water pipe and marijuana). In the study, it was interestingly noted that 34 percentages of the affected patients were female inspite of not being a smoker or tobacco user. This can be attributed to the reason that female are affected by passive smoke exposure due to cooking using biomass fuels, straw and firewood in rural areas and the exhausted gases from them contain sulphur dioxide act as a critical factor for development of COPD in females.

Of the 100 subjects studied, 44 percentage of patients lie in the age limit of 60-69 years who were categorized as "young old", 42 percentages in the age group of 70-79 years who were categorized as "old – old" and 14 percentage were in the age group of more than 80 years of age who were categorized as "very old". The lesser number of patients in 80 above age group is due to overall lesser contribution by them to the total population and also due to lack of seeking medical attention or hospitalization. Increase in death rate or non-survivors were noted in patients within the age group of very old i.e. above 80 years is due to the inability of the patients to revive from the effect of acute exacerbation of COPD and accompanying MODS. Therefore age can be considered as one of the risk factor for COPD. Similar to the previous study conducted by Xiao K et al. in 2014, this study also demonstrates that aged patients had a poor prognosis and all the 14 patients in the category of very old were not able to survive the disease severity and expired.[6]

Among the 100 study population, 33 patients had 1 exacerbation during the period of 6months or 1year before hospitalization, 40 patients had 2 exacerbation, 12 patients had 3 exacerbation and 15 patients had 4 exacerbation. The patients who has 3 or 4 exacerbation had a worst prognosis and all expired. Among the patients with 1 exacerbation there was no death and among the patients with 2 exacerbation suffered 1 death. Therefore increase in number of exacerbation turned down the prognosis. This findings of this study goes in hand with the similar previous study of Xiao K et al. suggesting that the number of previous exacerbation in the past, is one of the predicting factor for prognosis of COPD with high number of exacerbation correlated with higher mortality rate.[6]

Among the 100 study population, patients who were diagnosed to have more than two organ failure suffered high mortality rate than patients who had less than two organ failure was noted in the study. A previous similar study shows that the number of failing organs were higher in non-survivor patients compared to the survivors.

Among the study population, 28% patients expired during the hospital stay and 72% patients survived and were free of Acute Exacerbation, therefore they were discharged. In this study, among the non-survivors the most badly affected organ system was cardiovascular system and respiratory system and this synchronized with previous study of Xiao K et al. Few other similar studies conducted by Huiart L et al., in 2005, Rutten FH et al., in 2005 and MacDonald MI et al., in 2016 concluded that cardiovascular system is the most affected organ in the Multiple Organ Dysfunction Syndrome associated with COPD

exacerbation and this cardiovascular disease is the greater cause of mortality in patients of COPD even more than the lung disease itself.[58, 59 & 61]

In this study, among the non-survivors and survivors the Acute Kidney Injury (AKI) did not have any predictive value in the prognosis of the Multiple Organ Dysfunction Syndrome associated with COPD exacerbation. This observation of this study is in contrast to the other two studies conducted by Xiao K et al., in 2014 and Barakat MF et al., in 2015.[6,60]

In the present study, the mean score of SOFA and APACHE II scoring systems were calculated based on the average day 1 and day 3 score of different variables used in the scoring system. The prognostic prediction value of the both the score were considered as a median value of the SOFA score which was about 7.5 and APACHE II score was about 22.5. These values were considered as a mean value which decides the prognosis of the patients. The SOFA score value below or equal to 7.5 and the APACHE II score below or equal to 22.5 had a less mortality rate and had a better prognosis and were successfully treated and got free of Acute Exacerbation of COPD.

Length of the hospital stay was also considered as a prognostic factor in predicting the outcome of the disease because in patients who had a SOFA score above 7.5 and APACHE II score above 22.5 had a lesser days of hospital stay due to worst prognosis, increased mortality rate and early expiry of the patients and the difference was statistically significant with the SOFA score P value $< 0.001$ when compared to APACHE II.

Among the different variables used in assessing the SOFA and APACHE II score, variables such as PaO2/FiO2 ratio, respiratory rate, mean systolic arterial pressure (MSAP) and platelets count and serum bilirubin were identified to play a critical role in predicting the prognosis of the patients with acute exacerbation of COPD with MODS and the difference caused by them were statistically significant. This result was in harmony with the similar former study of Xiao K et al.[6]

In this study, among the two scoring systems the prediction of prognosis of Chronic Obstructive Pulmonary Disease (COPD) with Multiple Organ Dysfunction Syndrome (MODS), the SOFA score has high sensitivity and specificity in predicting the prognosis than the APACHE II score. It is because that the SOFA score assess six internal organ functions and the scoring system is easy to calculate than APACHE II scoring model which has numerous variables and are difficult, lengthy and bothersome to calculate. This result of the study coincides with the earlier study conducted by Xiao K et al., in 2014.[6]

# CONCLUSION

# CONCLUSION

* This study shows that SOFA scores determined at the onset of MODS in elderly patients with COPD were a reliable predictor of the prognosis.

* The exacerbation history, number of failing organs, and the SOFA score were risk factors of a poor prognosis, and the exacerbation history could also make an effective prediction of the outcome of COPD.

* The cardiovascular disease is the major cause of poor prognosis in patients with MODS associated with COPD. Therefore early diagnosis of cardiovascular diseases and its appropriate periodic follow-up and adequate intervention prevents the high mortality rate of patients with COPD associated with MODS.

* Among the multiple scoring models, SOFA score is the best predictor of prognosis. It is easy to calculate, cost effective and less time consuming. Therefore, early diagnosis and effective treatment can be done with the help of SOFA score.

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# ANNEXURES

**PROFORMA**

Name: Age/Sex: GM no:

Occupation: Literacy:

Address:

Complaints:

| | | | |
|---|---|---|---|
| Breathlessness | Chronic cough with expectoration | PND | Orthopenia |
| Chest pain | Syncope | Palpitation | Abdominal Distension |
| Decreased urine output | Leg swelling | Bleeding diathesis | Constipation |

PAST HISTORY:

| | | |
|---|---|---|
| Hypertension | Diabetes mellitus | PTB/BA |
| Hypothyroidism | CAD | CKD |
| CLD | CVA | Others: No of exacerbation/year |

MEDICATIONS:

PERSONAL HISTORY:

Smoking Alcoholic chronic exposure to firewood smoke Diet

EXAMINATION:

| | | | | |
|---|---|---|---|---|
| Built | Pallor | Icterus | Cyanosis | Clubbing |
| Edema | Lymphadenopathy | Height | Weight | BMI |

VITALS:

| | | | |
|---|---|---|---|
| Pulse rate: | BP: Sitting-<br>Lying- | Resp Rate: | Heart rate: |
| Temp: | SPO2: | Urine output/day: | MAP: |

**GLASGOW COMA SCALE SCORE:**

**SCORING SYSTEM:**

**SOFA SCORE- Sepsis related Organ Failure Assessment**

| **VARIABLES / POINTS** | **1** | **2** | **3** | **4** | **1ST DAY** | **3RD DAY** | **DIS** |
|---|---|---|---|---|---|---|---|
| Glasgow coma score | 13-14 | 10-12 | 6-9 | <6 | | | |
| Pulmonary: $PaO_2$/ $FiO_2$ | < 400 | <200 | <200 with respiratory support | < 100 with respiratory support | | | |
| Cardiology: MSAP (mm Hg) | <70 | Dopamine ≤5 Or Dobutamine whatever dose | Doamine >5 Or Adrenaline ≤ 0.1 Or Noradrenaline ≤0.1 | Dopamine > 15 Or Adrenaline >0.1 Noradrenaline >0.1 | | | |
| Renal: blood creatinine (mg/dl) | 1.2-1.9 | 2.0-3.4 | 3.5-4.9 | > 5 | | | |
| Haematology: Platelets $10^3$/L | <150 | <100 | <50 | <20 | | | |
| Hepatic: Serum Bilirubin (mg/dl) | 1.2-1.9 | 2.0-5.9 | 6.0-11.9 | >12 | | | |

TOTAL SCORE:

**A] APACHE II Score**:

| Physiologic Variable | +4 | +3 | +2 | +1 | 0 | +1 | +2 | +3 | +4 | 1st | 3rd | Dis |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Rectal temp | >41 | 39-40.9 | | 38.5-38.9 | 36-38.4 | 34-35.9 | 32-33.9 | 30-31.9 | <29 | | | |
| MAP | >160 | 130-159 | 110-129 | | 70-109 | | 50-69 | | <49 | | | |
| HR | >100 | 140-179 | 110-139 | | 70-109 | | 50-69 | | <49 | | | |
| RR | >50 | 35-49 | | 25-34 | 12-24 | 10-11 | 6-9 | | <5 | | | |
| Oxygenation $Fio_2$ >0.5 [A-a$DO_2$ ] $Fio_2$ <0.5Pao2 | >500 | 350-499 | 200-349 | | <200 <70 | 61-70 | | 55-60 | <55 | | | |
| Arterial $P_H$ | >7.7 | 7.6-7.69 | | 7.5-7.59 | 7.33-7.49 | | 7.25-7.32 | 7.15-7.24 | <7.15 | | | |
| $HCO_3$[mEq/l] | >52 | 41-51.9 | | 32-40.9 | 22-39.9 | | 18-21.9 | 15-17.9 | <7.15 | | | |
| K[mEq/l] | >7 | 6-6.9 | | 5.5-5.9 | 3.5-5.4 | 3-3.4 | 2.5-2.9 | | ,2.5 | | | |
| Na[mEq/l] | >160 | 160-179 | 155-159 | 150-154 | 130-149 | | 120-129 | 111-119 | <110 | | | |
| S.crea [mqm/dl] | >3.5 | 2-3.4 | 15-1.9 | | 0.6-1.4 | | <0.5 | | | | | |
| Hematocrit [%] | >60 | | 50-59.9 | 46-49.9 | 30-45.9 | | 20-29.9 | | <20 | | | |
| TLC 103/cc | >40 | | 20-39.9 | 15-19.9 | 3-14.9 | | 1-2.9 | | <1 | | | |

High abnormal Range Low abnormal Range Day

GCS:15=0,14=1,13=2,12=3,11=4,10=5,9=6,8=7,7=8,6=9,5=10,4=11,3=12.

B] Age score :< 44=0, 45-54=2, 55—64=3, 65-74=5,>75=6

C] Chronic health point: Non-operative patients=5

Emergency post-operative patients=5

Elective post-operative patients=2

Total APACHE II Score=A+B+C Outcome:

Mechanical ventilation:

Length of ICU stay [days]: Outcome:

# INFORMATION SHEET

We are conducting a study titled *PROGNOSTIC VALUE OF SOFA SCORE IN PATIENTS WITH MULTIPLE ORGAN DYSFUNCTION SYNDROME ASSOCIATED WITH ACUTE COPD EXACERBATION* among patients admitting in Rajiv Gandhi Government General Hospital, Chennai and for that your specimen may be valuable to us.

The purpose of this study is to assess the predictive value of SOFA score for critically ill patients with COPD.

We are selecting certain cases and if you are found eligible, we may be using your blood and urine samples to do special studies which in any way do not affect your final report or management.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of investigator

Date:

Place: Chennai. Signature of participant

**PATIENT CONSENT FORM**

Study Detail : PROGNOSTIC VALUE OF SOFA SCORE IN PATIENTS WITH MULTIPLE ORGAN DYSFUNCTION SYNDROME ASSOCIATED WITH ACUTE COPD EXACERBATION**.**
Study Centre : Rajiv Gandhi Govt. General Hospital, Chennai.
Patient's Name :
Patient's Age :
Identification No.:

Patient may check (✓) these boxes:

a) I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction. ☐

b) I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ☐

c) I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ☐

d) I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or wellbeing or any unexpected or unusual symptoms. ☐

e) I hereby consent to participate in this study. ☐

f) I hereby give permission to undergo complete clinical examination and hematological tests. ☐

Signature / Thumb Impression Signature of Investigator

Patient's Name & Address:

Study Investigator's Name:
**Dr. C. GANESAN.**

# INSTITUTIONAL ETHICS COMMITTEE
# MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
Fax: 011 25363970

## CERTIFICATE OF APPROVAL

To

Dr.C.Ganesan
I Year PG in MD Geriatrics
Department of Geriatric Medicine
Madras Medical College
Chennai 600 003

Dear Dr.C.Ganesan,

The Institutional Ethics Committee has considered your request and approved your study titled "**PROGNOSTIC VALUE OF DIFFERENT SCORING MODELS IN PATIENTS WITH MULTIPLE ORGAN DYSFUNCTION SYNDROME ASSOCIATED WITH ACUTE COPD EXACERBATION " - NO.20062017(A)**

The following members of Ethics Committee were present in the meeting hold on **20.06.2017** conducted at Madras Medical College, Chennai 3

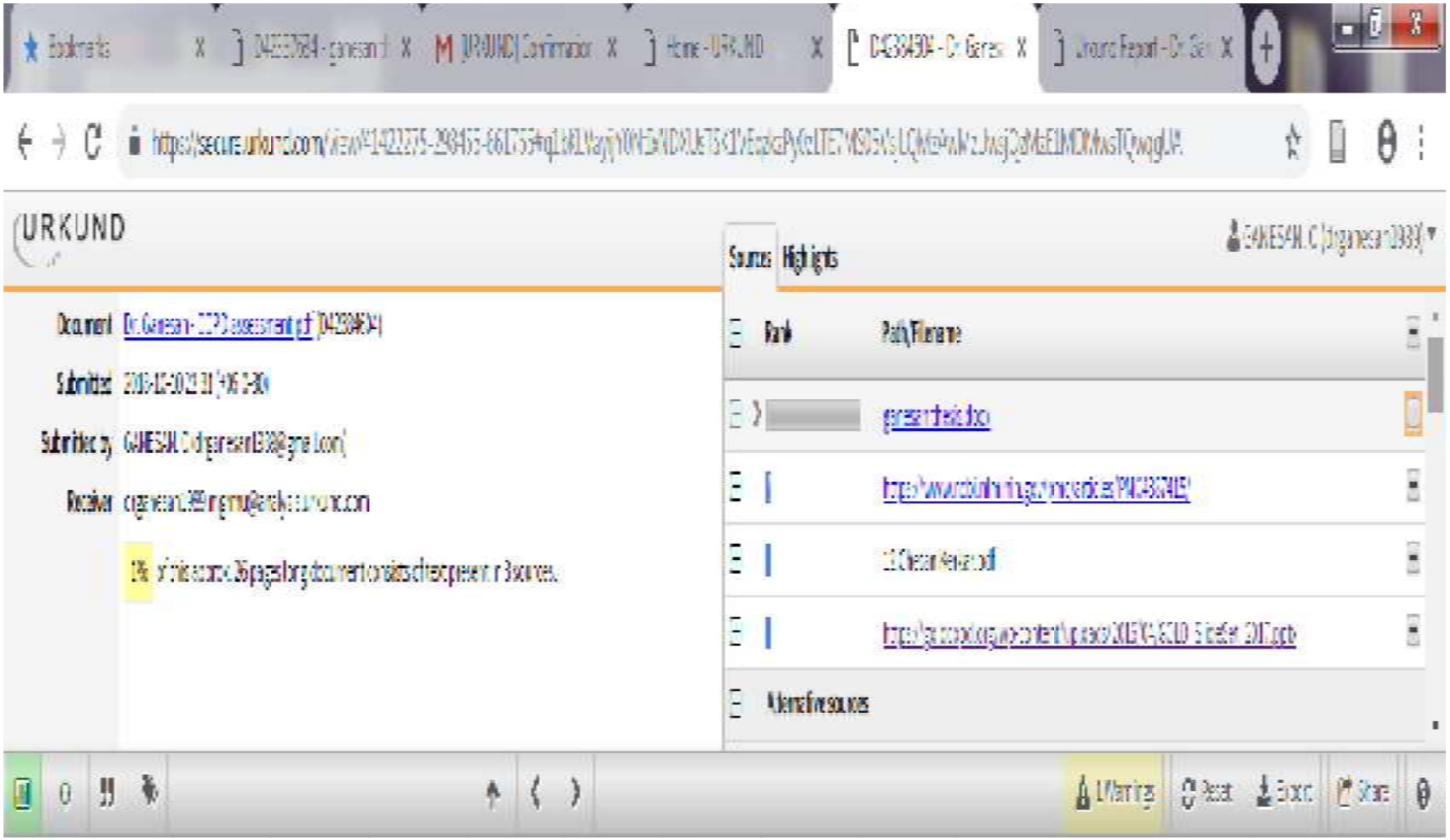
| | |
|---|---|
| 1. Prof.Dr.C.Rajendran, MD., | :Chairperson |
| 2. Prof.R.Narayana Babu,MD.,DCH., MMC,Ch-3 | : Deputy Chairperson |
| 3. Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3 | :Member Secretary |
| 4. Prof.S.Mayilvahanan,MD,Director,Inst. of Int.Med,MMC, Ch-3 | : Member |
| 5. Prof.A.Pandiya Raj,Director, Inst. of Gen.Surgery,MMC | : Member |
| 6. Prof.Rema Chandramohan,Prof.of Paediatrics,ICH,Chennai | : Member |
| 7. Prof. Susila, Director, Inst. of Pharmacology,MMC,Ch-3 | : Member |
| 8.Thiru S.Govindasamy, BA.,BL,High Court,Chennai | : Lawyer |
| 9.Tmt.Arnold Saulina, MA.,MSW., | :Social Scientist |
| 10.Tmt.J.Rajalakshmi, JAO,MMC, Ch-3 | : Lay Person |

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary – Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003


URKUND
Sources
Highlights
Document
Submitted
Submitted by
Receiver
Match
Path/Filename
Alternative sources

URKUND

# Urkund Analysis Result

| | |
|---|---|
| **Analysed Document:** | Dr. Ganesan - COPD assessment.pdf (D42384604) |
| **Submitted:** | 10/10/2018 7:01:00 PM |
| **Submitted By:** | drganesan1989@gmail.com |
| **Significance:** | 1 % |

## Sources included in the report:

12 Chetan Kerkar.pdf (D17227652)
https://goldcopd.org/wp-content/uploads/2016/04/GOLD_SlideSet_2017.pptx
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4387415/

## Instances where selected sources appear:

3

# MASTER CHART

## APACHE II SCORE

| S.NO | NAME | AGE | GENDER | HABIT OF SMOKING | NO. OF EXACERBATON | RECTAL TEMP | MAP | HR | RR | OXYGENATION FiO2<0.5 & FiO2 >0.5 | ARTERIAL Ph | HCo3 [mEq/L] | K mEq/L | Na mEq/L | S.Crea mgm/dl | HEMATOCRIT % | TLC 103/cc | GCS | AGE SCORE | CHP | T. APACHE II day 3 SCORE | T. APACHE II day 1 score | OUTCOME |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Arumugam | 65 | M | Yes | 1 | 38.6 | 105 | 92 | 26 | 92 | 7.35 | 18 | 5.2 | 139 | 5.2 | 30 | 13500 | 14 | 5 | 5 | 17 | 38 | DISCHARGED |
| 2 | Lakshmi | 68 | F | No | 2 | 38.5 | 100 | 98 | 28 | 89 | 7.28 | 28 | 3.8 | 132 | 1.2 | 41 | 13100 | 14 | 5 | 5 | 15 | 23 | DISCHARGED |
| 3 | Kanagammal | 61 | F | No | 1 | 39.3 | 80 | 102 | 26 | 86 | 7.34 | 32 | 4.5 | 135 | 2.142 | 42 | 12100 | 12 | 3 | 5 | 17 | 27 | DISCHARGED |
| 4 | Salomi | 68 | F | No | 3 | 40.9 | 60 | 55 | 17 | 267 | 7.09 | 35 | 4.1 | 142 | 1.9 | 49 | 22100 | 4 | 5 | 5 | 35 | 33 | DEATH ON 8 DAY |
| 5 | Kandasamy | 63 | M | Yes | 1 | 38.6 | 80 | 86 | 22 | 88 | 7.45 | 26 | 5.1 | 140 | 1.6 | 46 | 16100 | 15 | 3 | 5 | 12 | 23 | DISCHARGED |
| 6 | Perumal | 76 | M | Yes | 2 | 39.9 | 80 | 96 | 28 | 84 | 7.45 | 28 | 4 | 135 | 1.6 | 42 | 15300 | 14 | 6 | 5 | 16 | 31 | DISCHARGED |
| 7 | Mariyamma | 64 | F | No | 2 | 40.9 | 60 | 116 | 14 | 310 | 7.18 | 12 | 5.6 | 128 | 3.5 | 30 | 20200 | 6 | 3 | 5 | 46 | 40 | DEATH ON 11 DAY |
| 8 | Subramaniyan | 78 | M | Yes | 3 | 39.5 | 60 | 112 | 14 | 298 | 7.15 | 36 | 4.5 | 135 | 5.8 | 29 | 18000 | 6 | 6 | 5 | 40 | 36 | DEATH 0N 7 DAY |
| 9 | Murugan | 85 | M | Yes | 1 | 38.5 | 100 | 98 | 28 | 89 | 7.35 | 26 | 4.3 | 136 | 1.6 | 42 | 12000 | 13 | 6 | 5 | 20 | 39 | DISCHARGED |
| 10 | Malliga | 62 | F | No | 2 | 38.5 | 100 | 98 | 28 | 92 | 7.42 | 28 | 4.2 | 142 | 1.9 | 40 | 11000 | 13 | 3 | 5 | 16 | 33 | DISCHARGED |
| 11 | Krishnan | 73 | M | Yes | 1 | 38.5 | 120 | 100 | 22 | 90 | 7.35 | 26 | 4.6 | 142 | 1.6 | 41 | 9000 | 11 | 5 | 5 | 18 | 25 | DISCHARGED |
| 12 | Munusamy | 75 | M | Yes | 2 | 38.5 | 130 | 99 | 24 | 88 | 7.34 | 30 | 4.5 | 135 | 3.8 | 34 | 11000 | 11 | 6 | 5 | 23 | 31 | DISCHARGED |
| 13 | Kannan | 68 | M | Yes | 2 | 39 | 100 | 99 | 22 | 84 | 7.35 | 32 | 4.5 | 132 | 1.2 | 29 | 12000 | 13 | 5 | 5 | 18 | 27 | DISCHARGED |
| 14 | Lakshmi | 66 | F | No | 4 | 40.5 | 50 | 140 | 16 | 288 | 7.14 | 42 | 5.6 | 111 | 2.2 | 38 | 18500 | 5 | 5 | 5 | 46 | 32 | DEATH ON 9 DAY |
| 15 | Arumugam | 72 | M | Yes | 2 | 38.5 | 100 | 109 | 22 | 92 | 7.35 | 28 | 4.8 | 134 | 2.5 | 32 | 10100 | 14 | 5 | 5 | 15 | 32 | DISCHARGED |
| 16 | Elumalai | 80 | M | Yes | 2 | 38.5 | 100 | 96 | 24 | 87 | 7.35 | 28 | 4.2 | 129 | 2.1 | 35 | 14000 | 12 | 6 | 5 | 21 | 32 | DISCHARGED |
| 17 | Chellammal | 69 | F | No | 3 | 40.5 | 60 | 142 | 14 | 324 | 7.12 | 38 | 5.4 | 128 | 2.2 | 22 | 22000 | 5 | 5 | 5 | 41 | 30 | DEATH ON 8 DAY |

| | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 18 | Kuppusamy | 69 | M | Yes | 2 | 38.5 | 130 | 98 | 28 | 92 | 7.42 | 26 | 4.4 | 132 | 1.4 | 42 | 45000 | 14 | 5 | 5 | 15 | 25 | DISCHARGED |
| 19 | Saroja | 82 | F | No | 1 | 38.4 | 120 | 99 | 26 | 88 | 7.44 | 24 | 3.6 | 142 | 2.2 | 26 | 10000 | 13 | 6 | 5 | 22 | 36 | DISCHARGED |
| 20 | Ramasamy | 72 | M | Yes | 2 | 38.4 | 120 | 102 | 26 | 92 | 7.42 | 26 | 3.8 | 133 | 1.4 | 39 | 12000 | 14 | 5 | 5 | 14 | 29 | DISCHARGED |
| 21 | Padmavathy | 80 | F | No | 3 | 38.5 | 100 | 96 | 24 | 87 | 7.35 | 28 | 4.2 | 129 | 2.1 | 35 | 14000 | 12 | 6 | 5 | 21 | 26 | DEATH ON 10 DAY |
| 22 | Rathinam | 69 | F | No | 1 | 40.5 | 60 | 142 | 14 | 324 | 7.12 | 38 | 5.4 | 128 | 2.2 | 22 | 22000 | 5 | 5 | 5 | 41 | 32 | DISCHARGED |
| 23 | Ramanujam | 69 | M | Yes | 3 | 38.5 | 130 | 98 | 28 | 92 | 7.42 | 26 | 4.4 | 132 | 1.4 | 42 | 45000 | 14 | 5 | 5 | 15 | 29 | DEATH ON 9 DAY |
| 24 | Sundar | 82 | M | Yes | 1 | 38.4 | 120 | 99 | 26 | 88 | 7.44 | 24 | 3.6 | 142 | 2.2 | 26 | 10000 | 13 | 6 | 5 | 22 | 29 | DISCHARGED |
| 25 | Lakshmi | 72 | F | No | 2 | 38.4 | 120 | 102 | 26 | 92 | 7.42 | 26 | 3.8 | 133 | 1.4 | 39 | 12000 | 14 | 5 | 5 | 14 | 25 | DISCHARGED |
| 26 | Parvathy | 68 | F | No | 2 | 37.5 | 130 | 99 | 24 | 88 | 7.45 | 28 | 4.4 | 136 | 3.2 | 41 | 12000 | 14 | 5 | 5 | 17 | 28 | DISCHARGED |
| 27 | Kalimuthu | 82 | M | Yes | 2 | 41.1 | 70 | 132 | 16 | 256 | 7.1 | 10 | 5.5 | 138 | 2.7 | 27 | 25300 | 6 | 6 | 5 | 45 | 36 | DISCHARGED |
| 28 | Arputhammal | 69 | F | No | 2 | 38.1 | 140 | 92 | 26 | 88 | 7.41 | 28 | 3.9 | 133 | 1.4 | 38 | 12000 | 14 | 5 | 5 | 15 | 34 | DISCHARGED |
| 29 | Thirunavukarasu | 78 | M | Yes | 1 | 38.2 | 120 | 106 | 28 | 92 | 7.42 | 26 | 3.7 | 148 | 3.5 | 38 | 12300 | 14 | 6 | 5 | 19 | 34 | DISCHARGED |
| 30 | Ramuthai | 72 | F | No | 1 | 38.4 | 130 | 102 | 24 | 86 | 7.45 | 28 | 2.8 | 138 | 2.4 | 35 | 5600 | 14 | 5 | 5 | 19 | 31 | DISCHARGED |
| 31 | Alagappan | 82 | M | Yes | 4 | 42.5 | 60 | 140 | 14 | 355 | 7.12 | 42 | 5.8 | 111 | 2.2 | 29 | 18500 | 5 | 6 | 5 | 50 | 37 | DEATH ON 11 DAY |
| 32 | Rajammal | 70 | F | No | 2 | 38.5 | 105 | 109 | 24 | 88 | 7.35 | 28 | 4.7 | 134 | 2.6 | 32 | 10600 | 14 | 5 | 5 | 14 | 33 | DISCHARGED |
| 33 | Sarala | 77 | F | No | 3 | 41.5 | 60 | 132 | 14 | 264 | 7.1 | 10 | 4.2 | 132 | 2.9 | 22 | 240000 | 4 | 6 | 5 | 47 | 40 | DEATH ON 7 DAY |
| 34 | Kandasamy | 78 | M | Yes | 1 | 38.2 | 100 | 102 | 24 | 86 | 7.41 | 28 | 5.1 | 140 | 1.5 | 46 | 16000 | 14 | 6 | 5 | 16 | 28 | DISCHARGED |
| 35 | Nandagopal | 66 | M | Yes | 1 | 38.5 | 100 | 92 | 28 | 84 | 7.4 | 27 | 4.5 | 135 | 1.8 | 42 | 15000 | 14 | 5 | 5 | 16 | 30 | DISCHARGED |
| 36 | Narayanan | 66 | M | Yes | 2 | 41.5 | 60 | 140 | 14 | 364 | 7.12 | 9 | 5.6 | 117 | 2.5 | 26 | 18500 | 5 | 5 | 5 | 50 | 26 | DISCHARGED |
| 37 | Lalitha | 62 | F | No | 4 | 38.5 | 140 | 101 | 28 | 88 | 7.41 | 28 | 3.9 | 133 | 1.4 | 38 | 14000 | 14 | 3 | 5 | 13 | 40 | DEATH 0ON 8DAY |
| 38 | Namimabeebe | 69 | F | No | 2 | 37.5 | 110 | 99 | 28 | 92 | 7.42 | 26 | 4.4 | 132 | 1.4 | 44 | 12000 | 14 | 5 | 5 | 14 | 25 | DISCHARGED |
| 39 | Durai | 39 | M | Yes | 1 | 37.4 | 110 | 104 | 24 | 84 | 7.44 | 28 | 4.2 | 138 | 4.1 | 40 | 12000 | 14 | 5 | 5 | 17 | 28 | DISCHARGED |
| 40 | Babu | 40 | M | Yes | 3 | 41.5 | 60 | 140 | 14 | 274 | 7.21 | 38 | 5.4 | 119 | 2.8 | 20 | 22100 | 5 | 6 | 5 | 42 | 31 | DEATH ON 12 DAY |

| 41 | Ramamoorthy | 66 | M | Yes | 2 | 37.5 | 110 | 98 | 26 | 86 | 7.42 | 24 | 4.4 | 132 | 2 | 33 | 4500 | 14 | 5 | 5 | 17 | 26 | DISCHARGED |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 42 | Parvathy | 78 | F | No | 1 | 37.4 | 120 | 102 | 28 | 88 | 7.44 | 28 | 4.4 | 136 | 3.2 | 41 | 12000 | 14 | 5 | 5 | 16 | 29 | DISCHARGED |
| 43 | Natarajan | 72 | M | Yes | 4 | 41.2 | 60 | 142 | 16 | 298 | 7.1 | 12 | 3.6 | 122 | 3.7 | 20 | 21000 | 5 | 5 | 5 | 50 | 35 | DEATH ON 7 DAY |
| 44 | Aathilakshmi | 68 | F | No | 2 | 37.4 | 120 | 102 | 24 | 92 | 7.44 | 28 | 4.2 | 136 | 3.8 | 32 | 12000 | 14 | 5 | 5 | 17 | 27 | DISCHARGED |
| 45 | Ramamoorthy | 76 | M | Yes | 1 | 38.2 | 90 | 122 | 24 | 88 | 7.4 | 28 | 3.2 | 140 | 1.5 | 46 | 14300 | 14 | 6 | 5 | 19 | 33 | DISCHARGED |
| 46 | Sundaram | 78 | M | Yes | 3 | 41 | 60 | 126 | 14 | 312 | 7.15 | 10 | 4 | 135 | 2.3 | 29 | 18000 | 6 | 6 | 5 | 48 | 44 | DEATH ON 8 DAY |
| 47 | Nagammal | 68 | F | No | 1 | 38.2 | 100 | 108 | 24 | 92 | 7.35 | 28 | 4.7 | 136 | 2.8 | 32 | 11000 | 14 | 5 | 5 | 14 | 33 | DISCHARGED |
| 48 | Murugan | 83 | M | Yes | 2 | 38.7 | 110 | 98 | 25 | 88 | 7.42 | 28 | 2.6 | 138 | 2.5 | 38 | 21000 | 14 | 6 | 5 | 21 | 34 | DISCHARGED |
| 49 | Selvaraj | 76 | M | Yes | 4 | 41.2 | 70 | 132 | 14 | 288 | 7.01 | 10 | 3 | 139 | 2.2 | 28 | 200000 | 6 | 6 | 5 | 46 | 25 | DEATH ON 7 DAY |
| 50 | Padmini | 78 | F | No | 2 | 38.5 | 120 | 102 | 24 | 92 | 7.45 | 28 | 4.4 | 136 | 3.2 | 41 | 12000 | 14 | 65 | 5 | 18 | 29 | DISCHARGED |
| 51 | Jacob Immanuel | 80 | M | Yes | 2 | 38.5 | 100 | 96 | 24 | 87 | 7.35 | 28 | 4.2 | 129 | 2.1 | 35 | 14000 | 12 | 6 | 5 | 21 | 28 | DISCHARGED |
| 52 | Ramani | 69 | F | No | 1 | 40.5 | 60 | 142 | 14 | 324 | 7.12 | 38 | 5.4 | 128 | 2.2 | 22 | 22000 | 5 | 5 | 5 | 41 | 36 | DISCHARGED |
| 53 | Krishnan | 69 | M | Yes | 1 | 38.5 | 130 | 98 | 28 | 92 | 7.42 | 26 | 4.4 | 132 | 1.4 | 42 | 45000 | 14 | 5 | 5 | 15 | 34 | DISCHARGED |
| 54 | Ragavan | 82 | M | Yes | 2 | 38.4 | 120 | 99 | 26 | 88 | 7.44 | 24 | 3.6 | 142 | 2.2 | 26 | 10000 | 13 | 6 | 5 | 22 | 34 | DISCHARGED |
| 55 | Sarguna pandian | 72 | M | Yes | 1 | 38.4 | 120 | 102 | 26 | 92 | 7.42 | 26 | 3.8 | 133 | 1.4 | 39 | 12000 | 14 | 5 | 5 | 14 | 31 | DISCHARGED |
| 56 | Muthukumarasamy | 72 | M | Yes | 1 | 38.4 | 120 | 102 | 26 | 92 | 7.42 | 26 | 3.8 | 133 | 1.4 | 39 | 12000 | 14 | 5 | 5 | 14 | 26 | DISCHARGED |
| 57 | Chandra | 68 | F | No | 1 | 37.5 | 130 | 99 | 24 | 88 | 7.45 | 28 | 4.4 | 136 | 3.2 | 41 | 12000 | 14 | 5 | 5 | 17 | 29 | DISCHARGED |
| 58 | Jayaraman | 82 | M | Yes | 4 | 41.1 | 70 | 132 | 16 | 256 | 7.1 | 10 | 5.5 | 138 | 2.7 | 27 | 25300 | 6 | 6 | 5 | 45 | 35 | DEATH ON 11 DAY |
| 59 | Kajamoideen | 69 | M | Yes | 2 | 38.1 | 140 | 92 | 26 | 88 | 7.41 | 28 | 3.9 | 133 | 1.4 | 38 | 12000 | 14 | 5 | 5 | 15 | 27 | DISCHARGED |
| 60 | Janakiraman | 78 | M | Yes | 3 | 38.2 | 120 | 106 | 28 | 92 | 7.42 | 26 | 3.7 | 148 | 3.5 | 38 | 12300 | 14 | 6 | 5 | 19 | 37 | DEATH ON 8 DAY |
| 61 | Vasantha | 66 | F | No | 1 | 37.5 | 110 | 98 | 26 | 86 | 7.42 | 24 | 4.4 | 132 | 2 | 33 | 4500 | 14 | 5 | 5 | 17 | 33 | DISCHARGED |
| 62 | Sangeetha | 78 | F | No | 4 | 37.4 | 120 | 102 | 28 | 88 | 7.44 | 28 | 4.4 | 136 | 3.2 | 41 | 12000 | 14 | 5 | 5 | 16 | 40 | DEATH ON 7 DAY |
| 63 | Baby | 72 | F | No | 2 | 41.2 | 60 | 142 | 16 | 298 | 7.1 | 12 | 3.6 | 122 | 3.7 | 20 | 21000 | 5 | 5 | 5 | 50 | 28 | DISCHARGED |

| | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 64 | Abdul malik | 68 | M | Yes | 1 | 37.4 | 120 | 102 | 24 | 92 | 7.44 | 28 | 4.2 | 136 | 3.8 | 32 | 12000 | 14 | 5 | 5 | 17 | 30 | DISCHARGED |
| 65 | Glory | 76 | F | No | 2 | 38.2 | 90 | 122 | 24 | 88 | 7.4 | 28 | 3.2 | 140 | 1.5 | 46 | 14300 | 14 | 6 | 5 | 19 | 33 | DISCHARGED |
| 66 | Sreenivasan | 78 | M | Yes | 4 | 41 | 60 | 126 | 14 | 312 | 7.15 | 10 | 4 | 135 | 2.3 | 29 | 18000 | 6 | 6 | 5 | 48 | 44 | DEATH ON 9 DAY |
| 67 | Devaki | 68 | F | No | 2 | 38.2 | 100 | 108 | 24 | 92 | 7.35 | 28 | 4.7 | 136 | 2.8 | 32 | 11000 | 14 | 5 | 5 | 14 | 33 | DISCHARGED |
| 68 | Kajitha | 83 | F | No | 1 | 38.7 | 110 | 98 | 25 | 88 | 7.42 | 28 | 2.6 | 138 | 2.5 | 38 | 21000 | 14 | 6 | 5 | 21 | 34 | DISCHARGED |
| 69 | Sanjeev | 65 | M | Yes | 4 | 38.6 | 105 | 92 | 26 | 92 | 7.35 | 18 | 5.2 | 139 | 5.2 | 30 | 13500 | 14 | 5 | 5 | 17 | 25 | DEATH ON 7 DAY |
| 70 | Rukmini | 68 | F | No | 2 | 38.5 | 100 | 98 | 28 | 89 | 7.28 | 28 | 3.8 | 132 | 1.2 | 41 | 13100 | 14 | 5 | 5 | 15 | 38 | DISCHARGED |
| 71 | Mumthaj | 61 | F | No | 2 | 39.3 | 80 | 102 | 26 | 86 | 7.34 | 32 | 4.5 | 135 | 2.142 | 42 | 12100 | 12 | 3 | 5 | 17 | 23 | DISCHARGED |
| 72 | Kuppan | 68 | M | Yes | 1 | 40.9 | 60 | 55 | 17 | 267 | 7.09 | 35 | 4.1 | 142 | 1.9 | 49 | 22100 | 4 | 5 | 5 | 35 | 27 | DISCHARGED |
| 73 | Esther | 63 | F | No | 3 | 38.6 | 80 | 86 | 22 | 88 | 7.45 | 26 | 5.1 | 140 | 1.6 | 46 | 16100 | 15 | 3 | 5 | 12 | 33 | DEATH ON 11 DAY |
| 74 | Ellammal | 76 | F | No | 1 | 39.9 | 80 | 96 | 28 | 84 | 7.45 | 28 | 4 | 135 | 1.6 | 42 | 15300 | 14 | 6 | 5 | 16 | 23 | DISCHARGED |
| 75 | Palani | 64 | M | Yes | 1 | 40.9 | 60 | 116 | 14 | 310 | 7.18 | 12 | 5.6 | 128 | 3.5 | 30 | 20200 | 6 | 3 | 5 | 46 | 25 | DISCHARGED |
| 76 | Pattammal | 78 | F | No | 2 | 39.5 | 60 | 112 | 14 | 298 | 7.15 | 36 | 4.5 | 135 | 5.8 | 29 | 18000 | 6 | 6 | 5 | 40 | 31 | DISCHARGED |
| 77 | Raju | 85 | M | Yes | 2 | 38.5 | 100 | 98 | 28 | 89 | 7.35 | 26 | 4.3 | 136 | 1.6 | 42 | 12000 | 13 | 6 | 5 | 20 | 27 | DISCHARGED |
| 78 | Valliamma | 62 | F | No | 4 | 38.5 | 100 | 98 | 28 | 92 | 7.42 | 28 | 4.2 | 142 | 1.9 | 40 | 11000 | 13 | 3 | 5 | 16 | 32 | DEATH ON 9 DAY |
| 79 | Neelavathy | 73 | F | No | 2 | 38.5 | 120 | 100 | 22 | 90 | 7.35 | 26 | 4.6 | 142 | 1.6 | 41 | 9000 | 11 | 5 | 5 | 18 | 32 | DISCHARGED |
| 80 | Masilamani | 75 | M | Yes | 1 | 38.5 | 130 | 99 | 24 | 88 | 7.34 | 30 | 4.5 | 135 | 3.8 | 34 | 11000 | 11 | 6 | 5 | 23 | 32 | DISCHARGED |
| 81 | Radha | 68 | F | No | 4 | 39 | 100 | 99 | 22 | 84 | 7.35 | 32 | 4.5 | 132 | 1.2 | 29 | 12000 | 13 | 5 | 5 | 18 | 30 | DEATH ON 11 DAY |
| 82 | Thagamani | 66 | F | No | 2 | 40.5 | 50 | 140 | 16 | 288 | 7.14 | 42 | 5.6 | 111 | 2.2 | 38 | 18500 | 5 | 5 | 5 | 46 | 25 | DISCHARGED |
| 83 | Bommi | 72 | F | No | 2 | 38.5 | 100 | 109 | 22 | 92 | 7.35 | 28 | 4.8 | 134 | 2.5 | 32 | 10100 | 14 | 5 | 5 | 15 | 36 | DISCHARGED |
| 84 | Kaanjana | 80 | F | No | 2 | 38.5 | 100 | 96 | 24 | 87 | 7.35 | 28 | 4.2 | 129 | 2.1 | 35 | 14000 | 12 | 6 | 5 | 21 | 29 | DISCHARGED |
| 85 | Somasundaram | 69 | M | Yes | 4 | 40.5 | 60 | 142 | 14 | 324 | 7.12 | 38 | 5.4 | 128 | 2.2 | 22 | 22000 | 5 | 5 | 5 | 41 | 26 | DEATH ON 8 DAY |
| 86 | Boopathiyammal | 69 | F | No | 2 | 38.5 | 130 | 98 | 28 | 92 | 7.42 | 26 | 4.4 | 132 | 1.4 | 42 | 45000 | 14 | 5 | 5 | 15 | 32 | DISCHARGED |

| | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 87 | Vishvanathan | 82 | M | Yes | 3 | 38.4 | 120 | 99 | 26 | 88 | 7.44 | 24 | 3.6 | 142 | 2.2 | 26 | 10000 | 13 | 6 | 5 | 22 | 29 | DEATH ON 6 DAY |
| 88 | Sivakumar | 72 | M | Yes | 2 | 38.4 | 120 | 102 | 26 | 92 | 7.42 | 26 | 3.8 | 133 | 1.4 | 39 | 12000 | 14 | 5 | 5 | 14 | 29 | DISCHARGED |
| 89 | Masthana | 72 | F | No | 1 | 38.4 | 130 | 102 | 24 | 86 | 7.45 | 28 | 2.8 | 138 | 2.4 | 35 | 5600 | 14 | 5 | 5 | 19 | 25 | DISCHARGED |
| 90 | Subbiah | 82 | M | Yes | 1 | 42.5 | 60 | 140 | 14 | 355 | 7.12 | 42 | 5.8 | 111 | 2.2 | 29 | 18500 | 5 | 6 | 5 | 50 | 31 | DISCHARGED |
| 91 | Krishnaveni | 70 | F | No | 4 | 38.5 | 105 | 109 | 24 | 88 | 7.35 | 28 | 4.7 | 134 | 2.6 | 32 | 10600 | 14 | 5 | 5 | 14 | 40 | DEATH ON 8 DAY |
| 92 | Muthysamy | 77 | M | Yes | 3 | 41.5 | 60 | 132 | 14 | 264 | 7.1 | 10 | 4.2 | 132 | 2.9 | 22 | 240000 | 4 | 6 | 5 | 47 | 36 | DEAH 0N 9DAY |
| 93 | Karupasamy | 78 | M | Yes | 2 | 38.2 | 100 | 102 | 24 | 86 | 7.41 | 28 | 5.1 | 140 | 1.5 | 46 | 16000 | 14 | 6 | 5 | 16 | 39 | DISCHARGED |
| 94 | Duraiselvam | 66 | M | Yes | 1 | 38.5 | 100 | 92 | 28 | 84 | 7.4 | 27 | 4.5 | 135 | 1.8 | 42 | 15000 | 14 | 5 | 5 | 16 | 33 | DISCHARGED |
| 95 | Rajeshwari | 66 | F | No | 1 | 41.5 | 60 | 140 | 14 | 364 | 7.12 | 9 | 5.6 | 117 | 2.5 | 26 | 18500 | 5 | 5 | 5 | 50 | 26 | DISCHARGED |
| 96 | Raman | 62 | M | Yes | 4 | 38.5 | 140 | 101 | 28 | 88 | 7.41 | 28 | 3.9 | 133 | 1.4 | 38 | 14000 | 14 | 3 | 5 | 13 | 40 | DEATH ON 8 DAY |
| 97 | Amutha | 69 | F | No | 2 | 37.5 | 110 | 99 | 28 | 92 | 7.42 | 26 | 4.4 | 132 | 1.4 | 44 | 12000 | 14 | 5 | 5 | 14 | 25 | DISCHARGED |
| 98 | Nadesan | 39 | M | Yes | 2 | 37.4 | 110 | 104 | 24 | 84 | 7.44 | 28 | 4.2 | 138 | 4.1 | 40 | 12000 | 14 | 5 | 5 | 17 | 28 | DISCHARGED |
| 99 | Annamma | 40 | F | No | 4 | 41.5 | 60 | 140 | 14 | 274 | 7.21 | 38 | 5.4 | 119 | 2.8 | 20 | 22100 | 5 | 6 | 5 | 42 | 31 | DEATH ON 11 DAY |
| 100 | Nagarajan | 72 | M | Yes | 1 | 41.2 | 60 | 142 | 16 | 298 | 7.1 | 12 | 3.6 | 122 | 3.7 | 20 | 21000 | 5 | 5 | 5 | 50 | 29 | DISCHARGED |

# SOFA SCORE

| S.No | Name | AGE | age group | GENDER | HABIT OF SMOKING | NO OF EXACERBATON | GCS | Pao2Fio2 | MSAP | Sr. Creatine | Platelets103L | Sr. Bilirubin | T. SOFA score day 1 | GCS1 | Pao2Fio21 | MSAP1 | Sr. Creatine1 | Platelets | Sr. Bilirubin1 | T. SOFA Score day 3 | Outcome |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Arumugam | 65.00 | 60-70 Years | M | Yes | 1.00 | 14.00 | 352.00 | 130.00 | 8.80 | 283.00 | 2.20 | 9.00 | 14.00 | 452.00 | 105.00 | 5.20 | 387.00 | 0.80 | 5.00 | Discharge |
| 2 | Lakshmi | 68.00 | 60-70 Years | F | No | 2.00 | 12.00 | 194.00 | 90.00 | 2.10 | 352.00 | 2.20 | 8.00 | 14.00 | 422.00 | 100.00 | 1.20 | 323.00 | 1.00 | 2.00 | Discharge |
| 3 | Kanagammal | 61.00 | 60-70 Years | F | No | 1.00 | 12.00 | 188.00 | 105.00 | 2.90 | 245.00 | 2.40 | 8.00 | 14.00 | 410.00 | 80.00 | 2.10 | 321.00 | 1.60 | 4.00 | Discharge |
| 4 | Salomi | 68.00 | 60-70 Years | F | No | 3.00 | 6.00 | 78.00 | 80.00 | 1.20 | 212.00 | 1.40 | 10.00 | 4.00 | 100.00 | 60.00 | 1.90 | 179.00 | 2.00 | 14.00 | Death |
| 5 | Kandasamy | 63.00 | 60-70 Years | M | Yes | 1.00 | 14.00 | 200.00 | 120.00 | 1.80 | 185.00 | 2.00 | 6.00 | 14.00 | 442.00 | 80.00 | 1.60 | 220.00 | 0.80 | 2.00 | Discharge |
| 6 | Perumal | 76.00 | 71-80 Years | M | Yes | 2.00 | 14.00 | 352.00 | 130.00 | 8.80 | 283.00 | 2.20 | 9.00 | 14.00 | 482.00 | 80.00 | 1.60 | 175.00 | 1.60 | 3.00 | Discharge |
| 7 | Mariyamma | 62.00 | 60-70 Years | F | No | 2.00 | 12.00 | 194.00 | 90.00 | 2.10 | 352.00 | 2.20 | 8.00 | 5.00 | 98.00 | 60.00 | 3.50 | 120.00 | 1.50 | 16.00 | Death |
| 8 | Subramaniyan | 78.00 | 71-80 Years | M | Yes | 3.00 | 12.00 | 188.00 | 105.00 | 2.90 | 245.00 | 2.40 | 8.00 | 6.00 | 94.00 | 60.00 | 5.80 | 96.00 | 2.50 | 19.00 | Death |
| 9 | Murugan | 85.00 | Above 80 Years | M | Yes | 1.00 | 6.00 | 78.00 | 80.00 | 1.20 | 212.00 | 1.40 | 10.00 | 11.00 | 450.00 | 100.00 | 1.60 | 186.00 | 2.50 | 8.00 | Discharge |
| 10 | Malliga | 64.00 | 60-70 Years | F | No | 2.00 | 14.00 | 352.00 | 130.00 | 8.80 | 283.00 | 2.20 | 9.00 | 11.00 | 446.00 | 100.00 | 1.90 | 253.00 | 1.90 | 9.00 | Discharge |
| 11 | Krishnan | 73.00 | 71-80 Years | M | Yes | 1.00 | 12.00 | 194.00 | 90.00 | 2.10 | 352.00 | 2.20 | 8.00 | 11.00 | 456.00 | 120.00 | 1.60 | 314.00 | 1.50 | 4.00 | Discharge |
| 12 | Munusamy | 75.00 | 71-80 Years | M | Yes | 2.00 | 12.00 | 188.00 | 105.00 | 2.90 | 245.00 | 2.40 | 8.00 | 11.00 | 400.00 | 130.00 | 3.80 | 212.00 | 1.40 | 4.00 | Discharge |
| 13 | Kannan | 68.00 | 60-70 Years | M | Yes | 2.00 | 6.00 | 78.00 | 80.00 | 1.20 | 212.00 | 1.40 | 10.00 | 13.00 | 414.00 | 100.00 | 1.20 | 252.00 | 0.80 | 2.00 | Discharge |
| 14 | Lakshmi | 72.00 | 71-80 Years | F | No | 4.00 | 14.00 | 200.00 | 120.00 | 1.80 | 185.00 | 2.00 | 6.00 | 5.00 | 98.00 | 50.00 | 2.20 | 94.00 | 2.20 | 17.00 | Death |
| 15 | Arumugam | 72.00 | 71-80 Years | M | Yes | 2.00 | 10.00 | 424.00 | 100.00 | 3.40 | 193.00 | 1.40 | 6.00 | 14.00 | 442.00 | 100.00 | 2.50 | 251.00 | 0.80 | 3.00 | Discharge |
| 16 | Elumalai | 80.00 | 71-80 Years | M | Yes | 2.00 | 8.00 | 456.00 | 100.00 | 1.80 | 173.00 | 1.40 | 5.00 | 12.00 | 412.00 | 100.00 | 2.10 | 223.00 | 1.40 | 5.00 | Discharge |
| 17 | Chellammal | 69.00 | 60-70 Years | F | No | 3.00 | 9.00 | 196.00 | 90.00 | 1.40 | 144.00 | 1.60 | 8.00 | 5.00 | 146.00 | 60.00 | 2.20 | 98.00 | 1.80 | 13.00 | Death |
| 18 | Kuppusamy | 69.00 | 60-70 Years | M | Yes | 2.00 | 12.00 | 422.00 | 120.00 | 1.90 | 155.00 | 1.20 | 4.00 | 14.00 | 424.00 | 130.00 | 1.40 | 222.00 | 0.80 | 2.00 | Discharge |
| 19 | Saroja | 82.00 | Above 80 Years | F | No | 1.00 | 10.00 | 368.00 | 110.00 | 1.80 | 162.00 | 1.40 | 5.00 | 13.00 | 468.00 | 120.00 | 2.20 | 263.00 | 1.20 | 4.00 | Discharge |
| 20 | Ramasamy | 72.00 | 71-80 Years | M | Yes | 2.00 | 12.00 | 422.00 | 110.00 | 3.20 | 160.00 | 1.60 | 6.00 | 14.00 | 432.00 | 120.00 | 1.40 | 282.00 | 1.20 | 2.00 | Discharge |
| 21 | Padmavathy | 74.00 | 71-80 Years | F | No | 3.00 | 9.00 | 342.00 | 120.00 | 1.80 | 140.00 | 1.40 | 6.00 | 5.00 | 146.00 | 80.00 | 3.20 | 90.00 | 1.60 | 16.00 | Death |
| 22 | Rathinam | 68.00 | 60-70 Years | F | No | 1.00 | 10.00 | 324.00 | 140.00 | 3.20 | 168.00 | 1.20 | 6.00 | 14.00 | 422.00 | 140.00 | 2.40 | 202.00 | 0.80 | 3.00 | Discharge |
| 23 | Ramanujam | 82.00 | Above 80 Years | M | Yes | 3.00 | 10.00 | 378.00 | 100.00 | 1.90 | 175.00 | 0.80 | 4.00 | 7.00 | 126.00 | 70.00 | 2.80 | 132.00 | 1.40 | 12.00 | Death |
| 24 | Sundar | 69.00 | 60-70 Years | M | Yes | 1.00 | 10.00 | 322.00 | 130.00 | 1.90 | 108.00 | 1.20 | 5.00 | 12.00 | 472.00 | 130.00 | 1.40 | 353.00 | 0.80 | 3.00 | Discharge |
| 25 | Lakshmi | 78.00 | 71-80 Years | F | No | 2.00 | 12.00 | 368.00 | 100.00 | 2.50 | 262.00 | 1.20 | 5.00 | 9.00 | 166.00 | 70.00 | 2.70 | 180.00 | 2.40 | 12.00 | Discharge |
| 26 | Parvathy | 68.00 | 60-70 Years | F | No | 2.00 | 10.00 | 398.00 | 130.00 | 2.40 | 262.00 | 1.20 | 6.00 | 14.00 | 410.00 | 130.00 | 3.20 | 353.00 | 0.80 | 3.00 | Discharge |
| 27 | Kalimuthu | 82.00 | Above 80 Years | M | Yes | 2.00 | 9.00 | 342.00 | 90.00 | 2.20 | 234.00 | 1.20 | 4.00 | 6.00 | 124.00 | 70.00 | 2.70 | 32.00 | 6.20 | 15.00 | Discharge |
| 28 | Arputhammal | 69.00 | 60-70 Years | F | No | 2.00 | 11.00 | 346.00 | 130.00 | 1.90 | 222.00 | 0.90 | 6.00 | 14.00 | 410.00 | 140.00 | 1.40 | 288.00 | 0.90 | 2.00 | Discharge |
| 29 | Thirunavukarasu | 78.00 | 71-80 Years | M | Yes | 1.00 | 10.00 | 322.00 | 110.00 | 2.40 | 212.00 | 1.20 | 6.00 | 14.00 | 420.00 | 120.00 | 3.50 | 457.00 | 0.90 | 4.00 | Discharge |

| 30 | Ramuthai | 72.00 | 71-80 Years | F | No | 1.00 | 10.00 | 364.00 | 130.00 | 1.90 | 169.00 | 1.20 | 5.00 | 14.00 | 400.00 | 130.00 | 2.40 | 282.00 | 0.80 | 3.00 | Discharge |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 31 | Alagappan | 82.00 | Above 80 Years | M | Yes | 4.00 | 9.00 | 196.00 | 60.00 | 2.50 | 175.00 | 1.20 | 8.00 | 5.00 | 98.00 | 60.00 | 2.20 | 92.00 | 2.00 | 17.00 | Death |
| 32 | Rajammal | 70.00 | 60-70 Years | F | No | 2.00 | 9.00 | 364.00 | 100.00 | 3.40 | 180.00 | 1.20 | 6.00 | 14.00 | 422.00 | 105.00 | 2.60 | 251.00 | 0.80 | 3.00 | Discharge |
| 33 | Sarala | 77.00 | 71-80 Years | F | No | 3.00 | 9.00 | 296.00 | 80.00 | 1.90 | 168.00 | 1.80 | 5.00 | 4.00 | 168.00 | 60.00 | 2.90 | 132.00 | 1.80 | 15.00 | Death |
| 34 | Kandasamy | 78.00 | 71-80 Years | M | Yes | 1.00 | 12.00 | 342.00 | 100.00 | 1.80 | 262.00 | 1.20 | 5.00 | 14.00 | 412.00 | 100.00 | 1.50 | 352.00 | 0.80 | 2.00 | Discharge |
| 35 | Nandagopal | 66.00 | 60-70 Years | M | Yes | 1.00 | 12.00 | 366.00 | 90.00 | 2.00 | 188.00 | 1.20 | 6.00 | 14.00 | 446.00 | 100.00 | 1.80 | 1.80 | 1.00 | 2.00 | Discharge |
| 36 | Narayanan | 69.00 | 60-70 Years | M | Yes | 2.00 | 11.00 | 298.00 | 120.00 | 2.20 | 178.00 | 2.20 | 7.00 | 5.00 | 184.00 | 60.00 | 2.50 | 98.00 | 2.20 | 16.00 | Discharge |
| 37 | Lalitha | 66.00 | 60-70 Years | F | No | 4.00 | 9.00 | 298.00 | 60.00 | 1.90 | 188.00 | 1.20 | 7.00 | 14.00 | 468.00 | 140.00 | 1.40 | 350.00 | 1.10 | 2.00 | Death |
| 38 | Namimabeebe | 69.00 | 60-70 Years | F | No | 2.00 | 12.00 | 394.00 | 120.00 | 1.80 | 282.00 | 1.20 | 5.00 | 14.00 | 414.00 | 110.00 | 1.40 | 351.00 | 0.80 | 2.00 | Discharge |
| 39 | Durai | 68.00 | 60-70 Years | M | Yes | 1.00 | 10.00 | 344.00 | 110.00 | 3.80 | 186.00 | 1.10 | 7.00 | 14.00 | 442.00 | 110.00 | 4.10 | 322.00 | 0.60 | 4.00 | Discharge |
| 40 | Babu | 79.00 | 71-80 Years | M | Yes | 3.00 | 9.00 | 192.00 | 90.00 | 1.40 | 163.00 | 1.80 | 7.00 | 9.00 | 166.00 | 70.00 | 2.70 | 180.00 | 2.40 | 12.00 | Death |
| 41 | Ramamoorthy | 66.00 | 60-70 Years | M | Yes | 2.00 | 11.00 | 324.00 | 110.00 | 1.90 | 186.00 | 1.80 | 5.00 | 14.00 | 410.00 | 130.00 | 3.20 | 353.00 | 0.80 | 3.00 | Discharge |
| 42 | Parvathy | 78.00 | 71-80 Years | F | No | 1.00 | 10.00 | 364.00 | 120.00 | 2.40 | 192.00 | 1.80 | 6.00 | 6.00 | 124.00 | 70.00 | 2.70 | 32.00 | 6.20 | 15.00 | Discharge |
| 43 | Natarajan | 72.00 | 71-80 Years | M | Yes | 4.00 | 9.00 | 294.00 | 80.00 | 3.80 | 163.00 | 1.20 | 8.00 | 5.00 | 144.00 | 60.00 | 3.70 | 110.00 | 2.00 | 15.00 | Death |
| 44 | Aathilakshmi | 68.00 | 60-70 Years | F | No | 2.00 | 10.00 | 392.00 | 120.00 | 2.40 | 268.00 | 1.20 | 6.00 | 14.00 | 412.00 | 120.00 | 3.80 | 482.00 | 0.80 | 4.00 | Discharge |
| 45 | Ramamoorthy | 76.00 | 71-80 Years | M | Yes | 1.00 | 12.00 | 324.00 | 100.00 | 2.40 | 168.00 | 1.40 | 6.00 | 14.00 | 406.00 | 90.00 | 1.50 | 272.00 | 1.00 | 2.00 | Discharge |
| 46 | Sundaram | 78.00 | 71-80 Years | M | Yes | 3.00 | 8.00 | 164.00 | 60.00 | 1.70 | 160.00 | 2.60 | 9.00 | 6.00 | 98.00 | 60.00 | 2.30 | 111.00 | 4.80 | 15.00 | Death |
| 47 | Nagammal | 68.00 | 60-70 Years | F | No | 1.00 | 9.00 | 356.00 | 100.00 | 3.20 | 268.00 | 1.20 | 7.00 | 14.00 | 428.00 | 100.00 | 2.80 | 352.00 | 0.80 | 3.00 | Discharge |
| 48 | Murugan | 82.00 | Above 80 Years | M | Yes | 2.00 | 10.00 | 320.00 | 110.00 | 1.90 | 144.00 | 1.10 | 6.00 | 14.00 | 412.00 | 110.00 | 2.50 | 189.00 | 0.90 | 3.00 | Discharge |
| 49 | Selvaraj | 76.00 | 71-80 Years | M | Yes | 4.00 | 12.00 | 188.00 | 80.00 | 1.80 | 112.00 | 4.20 | 8.00 | 9.00 | 198.00 | 70.00 | 2.20 | 96.00 | 6.30 | 16.00 | Death |
| 50 | Padmini | 76.00 | 71-80 Years | F | No | 2.00 | 10.00 | 364.00 | 110.00 | 2.40 | 182.00 | 1.20 | 6.00 | 14.00 | 404.00 | 120.00 | 3.20 | 321.00 | 0.80 | 3.00 | Discharge |
| 51 | Jacob Immanuel | 68.00 | 60-70 Years | M | Yes | 2.00 | 14.00 | 352.00 | 130.00 | 8.80 | 283.00 | 2.20 | 9.00 | 14.00 | 410.00 | 130.00 | 3.20 | 353.00 | 0.80 | 3.00 | Discharge |
| 52 | Ramani | 82.00 | Above 80 Years | F | No | 1.00 | 12.00 | 194.00 | 90.00 | 2.10 | 352.00 | 2.20 | 8.00 | 6.00 | 124.00 | 70.00 | 2.70 | 32.00 | 6.20 | 15.00 | Discharge |
| 53 | Krishnan | 69.00 | 60-70 Years | M | Yes | 1.00 | 12.00 | 188.00 | 105.00 | 2.90 | 245.00 | 2.40 | 8.00 | 14.00 | 410.00 | 140.00 | 1.40 | 288.00 | 0.90 | 2.00 | Discharge |
| 54 | Ragavan | 78.00 | 71-80 Years | M | Yes | 2.00 | 6.00 | 78.00 | 80.00 | 1.20 | 212.00 | 1.40 | 10.00 | 14.00 | 420.00 | 120.00 | 3.50 | 457.00 | 0.90 | 4.00 | Discharge |
| 55 | Sarguna pandian | 72.00 | 71-80 Years | M | Yes | 1.00 | 14.00 | 200.00 | 120.00 | 1.80 | 185.00 | 2.00 | 6.00 | 14.00 | 400.00 | 130.00 | 2.40 | 282.00 | 0.80 | 3.00 | Discharge |
| 56 | Muthukumarasam | 66.00 | 60-70 Years | M | Yes | 1.00 | 12.00 | 422.00 | 110.00 | 3.20 | 160.00 | 1.60 | 6.00 | 5.00 | 184.00 | 60.00 | 2.50 | 98.00 | 2.20 | 16.00 | Discharge |
| 57 | Chandra | 78.00 | 71-80 Years | F | No | 1.00 | 9.00 | 342.00 | 120.00 | 1.80 | 140.00 | 1.40 | 6.00 | 14.00 | 468.00 | 140.00 | 1.40 | 350.00 | 1.10 | 2.00 | Discharge |
| 58 | Jayaraman | 72.00 | 71-80 Years | M | Yes | 4.00 | 10.00 | 324.00 | 140.00 | 3.20 | 168.00 | 1.20 | 6.00 | 14.00 | 414.00 | 110.00 | 1.40 | 351.00 | 0.80 | 2.00 | Death |
| 59 | Kajamoideen | 68.00 | 60-70 Years | M | Yes | 2.00 | 10.00 | 378.00 | 100.00 | 1.90 | 175.00 | 0.80 | 4.00 | 14.00 | 442.00 | 110.00 | 4.10 | 322.00 | 0.60 | 4.00 | Discharge |
| 60 | Janakiraman | 82.00 | Above 80 Years | M | Yes | 3.00 | 10.00 | 322.00 | 130.00 | 1.90 | 108.00 | 1.20 | 5.00 | 5.00 | 188.00 | 60.00 | 2.80 | 132.00 | 1.60 | 14.00 | Death |
| 61 | Vasantha | 70.00 | 60-70 Years | F | No | 1.00 | 10.00 | 398.00 | 130.00 | 2.40 | 262.00 | 1.20 | 6.00 | 14.00 | 466.00 | 110.00 | 2.00 | 272.00 | 1.60 | 4.00 | Discharge |
| 62 | Sangeetha | 77.00 | 71-80 Years | F | No | 4.00 | 9.00 | 342.00 | 90.00 | 2.20 | 234.00 | 1.20 | 4.00 | 9.00 | 166.00 | 70.00 | 2.70 | 180.00 | 2.40 | 12.00 | Death |
| 63 | Baby | 78.00 | 71-80 Years | F | No | 2.00 | 11.00 | 346.00 | 130.00 | 1.90 | 222.00 | 0.90 | 6.00 | 14.00 | 410.00 | 130.00 | 3.20 | 353.00 | 0.80 | 3.00 | Discharge |
| 64 | Abdul malik | 66.00 | 60-70 Years | M | Yes | 1.00 | 10.00 | 322.00 | 110.00 | 2.40 | 212.00 | 1.20 | 6.00 | 6.00 | 124.00 | 70.00 | 2.70 | 32.00 | 6.20 | 15.00 | Discharge |
| 65 | Glory | 76.00 | 71-80 Years | F | No | 2.00 | 14.00 | 352.00 | 130.00 | 8.80 | 283.00 | 2.20 | 9.00 | 14.00 | 452.00 | 105.00 | 5.20 | 387.00 | 0.80 | 5.00 | Discharge |

| 66 | Sreenivasan | 78.00 | 71-80 Years | M | Yes | 4.00 | 12.00 | 194.00 | 90.00 | 2.10 | 352.00 | 2.20 | 8.00 | 14.00 | 422.00 | 100.00 | 1.20 | 323.00 | 1.00 | 2.00 | Death |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 67 | Devaki | 68.00 | 60-70 Years | F | No | 2.00 | 12.00 | 188.00 | 105.00 | 2.90 | 245.00 | 2.40 | 8.00 | 14.00 | 410.00 | 80.00 | 2.10 | 321.00 | 1.60 | 4.00 | Discharge |
| 68 | Kajitha | 82.00 | Above 80 Years | F | No | 1.00 | 6.00 | 78.00 | 80.00 | 1.20 | 212.00 | 1.40 | 10.00 | 4.00 | 100.00 | 60.00 | 1.90 | 179.00 | 2.00 | 14.00 | Discharge |
| 69 | Sanjeev | 76.00 | 71-80 Years | M | Yes | 4.00 | 14.00 | 200.00 | 120.00 | 1.80 | 185.00 | 2.00 | 6.00 | 14.00 | 442.00 | 80.00 | 1.60 | 220.00 | 0.80 | 2.00 | Death |
| 70 | Rukmini | 65.00 | 60-70 Years | F | No | 2.00 | 10.00 | 424.00 | 100.00 | 3.40 | 193.00 | 1.40 | 6.00 | 5.00 | 146.00 | 80.00 | 3.20 | 90.00 | 1.60 | 16.00 | Discharge |
| 71 | Mumthaj | 68.00 | 60-70 Years | F | No | 2.00 | 8.00 | 456.00 | 100.00 | 1.80 | 173.00 | 1.40 | 5.00 | 14.00 | 422.00 | 140.00 | 2.40 | 202.00 | 0.80 | 3.00 | Discharge |
| 72 | Kuppan | 61.00 | 60-70 Years | M | Yes | 1.00 | 9.00 | 196.00 | 90.00 | 1.40 | 144.00 | 1.60 | 8.00 | 7.00 | 126.00 | 70.00 | 2.80 | 132.00 | 1.40 | 12.00 | Discharge |
| 73 | Esther | 68.00 | 60-70 Years | F | No | 3.00 | 12.00 | 422.00 | 120.00 | 1.90 | 155.00 | 1.20 | 4.00 | 12.00 | 472.00 | 130.00 | 1.40 | 353.00 | 0.80 | 3.00 | Death |
| 74 | Ellammal | 63.00 | 60-70 Years | F | No | 1.00 | 10.00 | 368.00 | 110.00 | 1.80 | 162.00 | 1.40 | 5.00 | 9.00 | 166.00 | 70.00 | 2.70 | 180.00 | 2.40 | 12.00 | Discharge |
| 75 | Palani | 73.00 | 71-80 Years | M | Yes | 1.00 | 12.00 | 366.00 | 90.00 | 2.00 | 188.00 | 1.20 | 6.00 | 14.00 | 482.00 | 80.00 | 1.60 | 175.00 | 1.60 | 3.00 | Discharge |
| 76 | Pattammal | 75.00 | 71-80 Years | F | No | 2.00 | 11.00 | 298.00 | 120.00 | 2.20 | 178.00 | 2.20 | 7.00 | 5.00 | 98.00 | 60.00 | 3.50 | 120.00 | 1.50 | 16.00 | Discharge |
| 77 | Raju | 68.00 | 60-70 Years | M | Yes | 2.00 | 9.00 | 298.00 | 60.00 | 1.90 | 188.00 | 1.20 | 7.00 | 6.00 | 94.00 | 60.00 | 5.80 | 96.00 | 2.50 | 19.00 | Discharge |
| 78 | Valliamma | 72.00 | 71-80 Years | F | No | 4.00 | 12.00 | 394.00 | 120.00 | 1.80 | 282.00 | 1.20 | 5.00 | 11.00 | 450.00 | 100.00 | 1.60 | 186.00 | 2.50 | 8.00 | Death |
| 79 | Neelavathy | 72.00 | 71-80 Years | F | No | 2.00 | 10.00 | 344.00 | 110.00 | 3.80 | 186.00 | 1.10 | 7.00 | 11.00 | 446.00 | 100.00 | 1.90 | 253.00 | 1.90 | 9.00 | Discharge |
| 80 | Masilamani | 80.00 | 71-80 Years | M | Yes | 1.00 | 14.00 | 352.00 | 130.00 | 8.80 | 283.00 | 2.20 | 9.00 | 9.00 | 166.00 | 70.00 | 2.70 | 180.00 | 2.40 | 12.00 | Discharge |
| 81 | Radha | 69.00 | 60-70 Years | F | No | 4.00 | 12.00 | 194.00 | 90.00 | 2.10 | 352.00 | 2.20 | 8.00 | 14.00 | 410.00 | 130.00 | 3.20 | 353.00 | 0.80 | 3.00 | Death |
| 82 | Thagamani | 69.00 | 60-70 Years | F | No | 2.00 | 12.00 | 188.00 | 105.00 | 2.90 | 245.00 | 2.40 | 8.00 | 6.00 | 124.00 | 70.00 | 2.70 | 32.00 | 6.20 | 15.00 | Discharge |
| 83 | Bommi | 82.00 | Above 80 Years | F | No | 2.00 | 6.00 | 78.00 | 80.00 | 1.20 | 212.00 | 1.40 | 10.00 | 9.00 | 198.00 | 70.00 | 2.20 | 96.00 | 6.30 | 16.00 | Discharge |
| 84 | Kaanjana | 72.00 | 71-80 Years | F | No | 2.00 | 14.00 | 200.00 | 120.00 | 1.80 | 185.00 | 2.00 | 6.00 | 14.00 | 404.00 | 120.00 | 3.20 | 321.00 | 0.80 | 3.00 | Discharge |
| 85 | Somasundaram | 74.00 | 71-80 Years | M | Yes | 4.00 | 12.00 | 422.00 | 110.00 | 3.20 | 160.00 | 1.60 | 6.00 | 11.00 | 456.00 | 120.00 | 1.60 | 314.00 | 1.50 | 4.00 | Death |
| 86 | Boopathiyammal | 68.00 | 60-70 Years | F | No | 2.00 | 9.00 | 342.00 | 120.00 | 1.80 | 140.00 | 1.40 | 6.00 | 11.00 | 400.00 | 130.00 | 3.80 | 212.00 | 1.40 | 4.00 | Discharge |
| 87 | Vishvanathan | 82.00 | Above 80 Years | M | Yes | 3.00 | 10.00 | 324.00 | 140.00 | 3.20 | 168.00 | 1.20 | 6.00 | 13.00 | 414.00 | 100.00 | 1.20 | 252.00 | 0.80 | 2.00 | Death |
| 88 | Sivakumar | 69.00 | 60-70 Years | M | Yes | 2.00 | 10.00 | 378.00 | 100.00 | 1.90 | 175.00 | 0.80 | 4.00 | 5.00 | 98.00 | 50.00 | 2.20 | 94.00 | 2.20 | 17.00 | Discharge |
| 89 | Masthana | 78.00 | 71-80 Years | F | No | 1.00 | 10.00 | 322.00 | 130.00 | 1.90 | 108.00 | 1.20 | 5.00 | 14.00 | 442.00 | 100.00 | 2.50 | 251.00 | 0.80 | 3.00 | Discharge |
| 90 | Subbiah | 76.00 | 71-80 Years | M | Yes | 1.00 | 10.00 | 364.00 | 130.00 | 1.90 | 169.00 | 1.20 | 5.00 | 5.00 | 98.00 | 60.00 | 2.20 | 92.00 | 2.00 | 17.00 | Discharge |
| 91 | Krishnaveni | 62.00 | 60-70 Years | F | No | 4.00 | 9.00 | 196.00 | 60.00 | 2.50 | 175.00 | 1.20 | 8.00 | 14.00 | 422.00 | 105.00 | 2.60 | 251.00 | 0.80 | 3.00 | Death |
| 92 | Muthysamy | 78.00 | 71-80 Years | M | Yes | 3.00 | 9.00 | 364.00 | 100.00 | 3.40 | 180.00 | 1.20 | 6.00 | 4.00 | 168.00 | 60.00 | 2.90 | 132.00 | 1.80 | 15.00 | Death |
| 93 | Karupasamy | 85.00 | Above 80 Years | M | Yes | 2.00 | 9.00 | 296.00 | 80.00 | 1.90 | 168.00 | 1.80 | 5.00 | 14.00 | 412.00 | 100.00 | 1.50 | 352.00 | 0.80 | 2.00 | Discharge |
| 94 | Duraiselvam | 64.00 | 60-70 Years | M | Yes | 1.00 | 12.00 | 342.00 | 100.00 | 1.80 | 262.00 | 1.20 | 5.00 | 14.00 | 446.00 | 100.00 | 1.80 | 1.80 | 1.00 | 2.00 | Discharge |
| 95 | Rajeshwari | 69.00 | 60-70 Years | F | No | 1.00 | 9.00 | 192.00 | 90.00 | 1.40 | 163.00 | 1.80 | 7.00 | 12.00 | 412.00 | 100.00 | 2.10 | 223.00 | 1.40 | 5.00 | Discharge |
| 96 | Raman | 66.00 | 60-70 Years | M | Yes | 4.00 | 11.00 | 324.00 | 110.00 | 1.90 | 186.00 | 1.80 | 5.00 | 5.00 | 146.00 | 60.00 | 2.20 | 98.00 | 1.80 | 13.00 | Death |
| 97 | Amutha | 69.00 | 60-70 Years | F | No | 2.00 | 10.00 | 364.00 | 120.00 | 2.40 | 192.00 | 1.80 | 6.00 | 14.00 | 424.00 | 130.00 | 1.40 | 222.00 | 0.80 | 2.00 | Discharge |
| 98 | Nadesan | 68.00 | 60-70 Years | M | Yes | 2.00 | 9.00 | 294.00 | 80.00 | 3.80 | 163.00 | 1.20 | 8.00 | 13.00 | 468.00 | 120.00 | 2.20 | 263.00 | 1.20 | 4.00 | Discharge |
| 99 | Annamma | 79.00 | 71-80 Years | F | No | 4.00 | 10.00 | 392.00 | 120.00 | 2.40 | 268.00 | 1.20 | 6.00 | 14.00 | 432.00 | 120.00 | 1.40 | 282.00 | 1.20 | 2.00 | Death |
| 100 | Nagarajan | 76.00 | 71-80 Years | M | Yes | 1.00 | 9.00 | 196.00 | 90.00 | 1.40 | 144.00 | 1.60 | 8.00 | 11.00 | 450.00 | 100.00 | 1.60 | 186.00 | 2.50 | 8.00 | Discharge |