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CERTIFICATE

This is to certify that this dissertation entitled **"STUDY ON CHRONIC OBSTRUCTIVE PULMONARY DISEASES"** is the bonafide record work done by **Dr. R. RAVISHANKAR,** submitted as partial fulfillment for the requirements of M.D. Degree Examinations, General Medicine (Branch I) to be held in March 2007.

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CONTENTS

INTRODUCTION

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Chronic Obstructive Pulmonary Diseases (COPD) has been defined by the Global Initiative for chronic obstructive lung disease (GOLD) as “a disease stage characterized by air flow limitation that is not fully reversible” [1]

Chronic Obstructive Pulmonary Diseases includes (i) Emphysema defined as the permanent abnormal distension of the air spaces distal to the terminal bronchioles accompanied by destruction of their wall without fibrosis. (ii) Chronic Bronchitis defined on the presence of Chronic productive cough on most days for 3 months in each of two consecutive years. (iii) Small airway disease [2] in which small bronchioles are narrowed.

Excluded from this definition is Bronchial Asthma [3] chronic Bronchitis and emphysema were frequently coexist since they share common etiological factors and after many years chronic bronchitis get complicated by emphysema.

GOLD estimates suggested that Chronic Obstructive Pulmonary Diseases, sixth most common cause of death world wide at present, will be the third most common cause of death world wide by 2020. [1]

In India Chronic Obstructive Pulmonary Diseases is the second most common lung disorder after pulmonary tuberculosis. [19]

This disease is frequently seen in middle-aged subjects. Chronic Obstructive Pulmonary Diseases affects male more frequently than females because of smoking. [22]

It is equally prevalent in rural and urban areas. [19]

Increased smoking habits among younger people, increasing urbanization, increasing automobiles and emergence of industries leading to Air pollution that has definite impact on the epidemiology of Chronic Obstructive Pulmonary Diseases.

Low birth weight, malnutrition, recurrent respiratory infection in childhood also predisposed to Chronic Obstructive Pulmonary Diseases in future. [24]

Spirometry is the most robust test of airflow limitation in patient with Chronic Obstructive Pulmonary Diseases. [3]

A low FEV_1 (FEV_1 < 80%) with FEV_1 / FVC ratio < 0.7 and un < 15% reversibility of airflow obstruction to broncho dialators is the diagnostic criteria for COPD. [2]

(FEV_1 – Forced Expiratory Volume in 1 sec. FVC – Forced Vital Capacity)

This study, conducted at Thanjavur Medical College during the period of 2005 – 06 is first of its kind in this region, try to find out the age and sex incidence of Chronic Obstructive Pulmonary Diseases and incidence of various risk factor.

Further more this study is to elucidate the relationship of clinical symptom and spirometry abnormalities and also the incidence of right heart failure in Chronic Obstructive Pulmonary Diseases.

AIM OF THE STUDY

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1. To study the age and sex distribution in Chronic Obstructive Pulmonary Diseases.
2. To study the incidence of various risk factors in Chronic Obstructive Pulmonary Diseases.
3. To correlate the clinical symptoms with spirometry abnormalities in Chronic Obstructive Pulmonary Diseases.
4. To study the clinical incidence of Right Heart failure in Chronic Obstructive Pulmonary Diseases.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

From the time of Laennec., et al through the first half of 20th century, mechanical explanations of Chronic Obstructive Pulmonary Diseases dominate. The importance of cigarette smoking was not appreciated at that time. But the observation “Chronic lung disease may cause heart failure even on other wise normal heart” was made at that time.

As early as in 1905 OPIE, et al suggested that enzymes and anti enzymes imbalance determines the risk of emphysema. [3]

In 1934 Kountz and Alexander concluded that “It appears that heart is affected in most cases with emphysema” [3]

In late 1950 Liebow et al proposed the vascular atrophy model of emphysema.

In 1956 the Medial Research Council in its journal used the term chronic bronchitis to describe conditions with chronic cough with expectoration of bronchial mucus hyper secretion. When other causes like TB, PT Bronchiectasis were excluded.

In 1959 Higgins established the clear relationship between "Smoking and persistent cough and sputum production.

In 1960's Gross and associates leads to the proteinase and antiproteinase hypothesis in emphysema observed in α_1 antitrypsin deficiency.

In late 1960 Owen and Campell et al observed the pathological changes in airways due to cigarette smoking.

In 1971 Boushy SF et al., studied 108 patients of Chronic Obstructive Pulmonary Diseases. The ECG was correlated with pulmonary function Test and haemodynamic data.

In 1972 Benjamin Burrows et al, done a seven year study of 50 cases of Chronic Obstructive Pulmonary Diseases whose cardiac status had been evaluated by cardiac catheterization when they were stable, showed that their longevity was inversely related with pulmonary vascular resistance.

In 1973 Bougly and Colleagues published a series of papers on prognosis factors in Chronic Obstructive Pulmonary Diseases and

prognosis values of lung functions test in Chronic Obstructive Pulmonary Diseases.

In 1981 Matthay et al listed the electro cardio graphic changes chracteristic of RV and RA enlargement in COPD and corpulmonale.

In 1987 Danchin N et al., used 2D echocardiography to study right side cardiac parameter and RV hypertrophy and showed the correlation between Echo cardiography changes and clinical symptoms.

In 1990 Migures M et al showed that usefulness of pulsed Doppler Echocardiography to detect pulmonary artery hypertension in patients with COPD.

EPIDEMIOLOGY

Chronic Obstructive Pulmonary Diseases is responsible for considerable morbidity and mortality in the community especially among the older people. As these disorder has a protracted course spanning over several years to decades, place a major burden to the resources of health care system.

COPD is of considerable public health importance as these diseases are to a great extent preventable.

Exacerbation of the COPD occurs more during winter season.

PREVALENCE IN INDIA

Chronic Obstructive Pulmonary Diseases is the second most common lung disorder after pulmonary tuberculosis.

Incidence are Higher in males due to higher prevalence of smoking. It is a disease of middle aged and elderly people, less common below the age of 35 years.

Studies from North India, reported that the prevalence of chronic bronchitis was as high as 16% in people above 40 years from rural areas. Prevalence is more in North India than South India due to seasonal variability, particularly extremes of climate in North India.

A study by Bhattacharya et al showed prevelance of chronic bronchitis in rural population aged more than 30 years was 57 / 1000 there was a male prepondence which was higher with increasing age and pack years of smoking.

PREVALENCE IN WESTERN COUNTRIES:

In USA, it is estimated that they are > 16 million case of COPD. About 14 million with chronic bronchitis and around 2 million with emphysema. Male : Female ratio ranges from 4 – 6% / 1 – 3%.

In UK, a prospective study involving 40,000 medical practitioners, showed that death rate from chronic bronchitis was higher in smokers and increased with amount smoked.

AETIOLOGY

Chronic Obstructive Pulmonary Diseases is characterized by a reduced Forced Expiratory Volume in 1 second (FEV_1) and an

accelerated rate of decline of FEV_1. The reduction in FEV_1 can occur by any of three pathways.

1. Impaired childhood growth and development, with a lower peak in early adulthood and a normal rate of decline with aging eg. Early childhood infection and passive smoke exposure.
2. Normal growth and development with premature peak but normal subsequent decline eg: astma and passive smoking.
3. Normal growth and development and peak, with accelerated decline eg.: active smoking and to a lesser extent environmental exposures.

RISK FACTORS:

Smoking

Cigarette smoking is the most commonly identified correlate with chronic bronchitis during life. Pipe and Cigar smokers have a higher mortality and morbidity rates for COPD shows a dose response relationship with the number of pack-years of tobacco consumed. The British Thoracic Society guidelines suggest that most patients with COPD have at least 20 pack years smoking history. An average cigarette smoker have high annual rate of decline in FEV_1 of about 50ml, which is nearly double the average value of 30ml in nonsmokers.

In nonsmokers the decline in FEV_1 begins at 30-35 years of age and this may occur earlier in smokers. Stopping cigarette smoking does not produce a substantial improvement in FEV_1 but the subsequent rate of decline is decreased.

Prolonged cigarette smoking impairs respiratory epithelial ciliary movement, inhibits function of alveolar macrohages and leads to hyperplasia and hypertrophy of mucus secreting glands. Cigarette smoke also inhibits anti-proteases and causes polymorphonuclear leukocytes to release proteolytic enzymes. Smoking is associated with increased airway responsiveness, which is associated with more rapid progression in patients with COPD. Obstruction of small airway is the earliest demonstrable mechanical defect in a smoker.

Air Pollution

Incidence and mortality rates of both chronic bronchitis and emphysema may be higher in industrialized urban areas. Exacerbation of bronchitis are clearly related to periods of heavy pollution with sulfur dioxide and particulate matter.

In developing countries like India traditional cooking fuels such as wood, cow dung cake, etc., along with poorly ventilated houses are significant risk factors for chronic bronchitis.

Socio Economic Status

Hrubec et al found a strong association between socio economic status based on occupation and respiratory symptoms in a study of twins. In so many studies it was observed that an inverse relation between percapita income and obstructive lung disease.

Occupation

Chronic bronchitis is more prevalent in workers who engage in occupation exposing them to either inorganic or organic dusts, or to noxious gases. Surveys have found an accelerated decline in lung function in such workers eg, workers in plastic plants to exposure to Toluene disouganate etc. Exposure to cadmium can increase the chance of development of emphysema and hence COPD.

Recurrent Respiratory Infections

Frequency of acute respiratory illness are higher in patients with chronic bronchitis. Epidemiological studies however implicate recurrent respiratory illness as one of the major factors associated with etiology as well as the progression of chronic airway obstruction.

Airway hyper responsiveness and atopy:

Even though airway hyper responsiveness is a future of asthma many patient with COPD also share this feature but less than 15% of reversibility of obstruction to bronchodilators.

Growth And Nutrition

Studies have shown that nutrition may affect both the growth and decline in ventilatory function. There is also some evidence that server viral pneumonia early in life may lead to chronic obstruction, particularly in small airways.

GENETIC FACTORS:

α_1, Antitrypsin Deficiency

α_1 AntiTrypsin (α_1AT) is a polymorphic glycoprotein responsible for the majority of anti-protease activity in the serum, whose synthesis is governed by a gene on 14q 32 chromosome. The most commonest deficient allele termed ZZ (Cor Pi^{ZZ} Phenotypel) results from a single amino acid substitution 342Glu $\rightarrow$ Lys, which causes spontaneous polymerization of the polypeptide, markedly impairing its release into circulation from the liver.

It is commonly seen among people from European descent 1:2000 to 1:7000 people, rate in people from African and Asian lineage.

α_1 AT deficiency accounts for 2% of observed cases of emphysema. Patients present with premature development of enphysema chronic bronchitis or bronchiectasis. The patient usually presents with cough and dyspnea in the forth decade. Nearly 80% had a family history of lung disease with autosomal recessive inheritance.

The average decline of FEV_1 is 100-130ml / yr for smokers and 50 to 80ml / yr for ex-smokers of lifetime nonsmokers.

Pathologically panacinar emphysema predominates and radiographically changes are most marked in lower lobes. Tobacco smoking is an extremely important cofactor for development of disease in α_1 AT deficiency.

These patients are also at increased risk of hepatic cirrhosis.

PATHOLOGY

The pathologic changes of COPD involves large and small airways and the terminal respiratory unit.

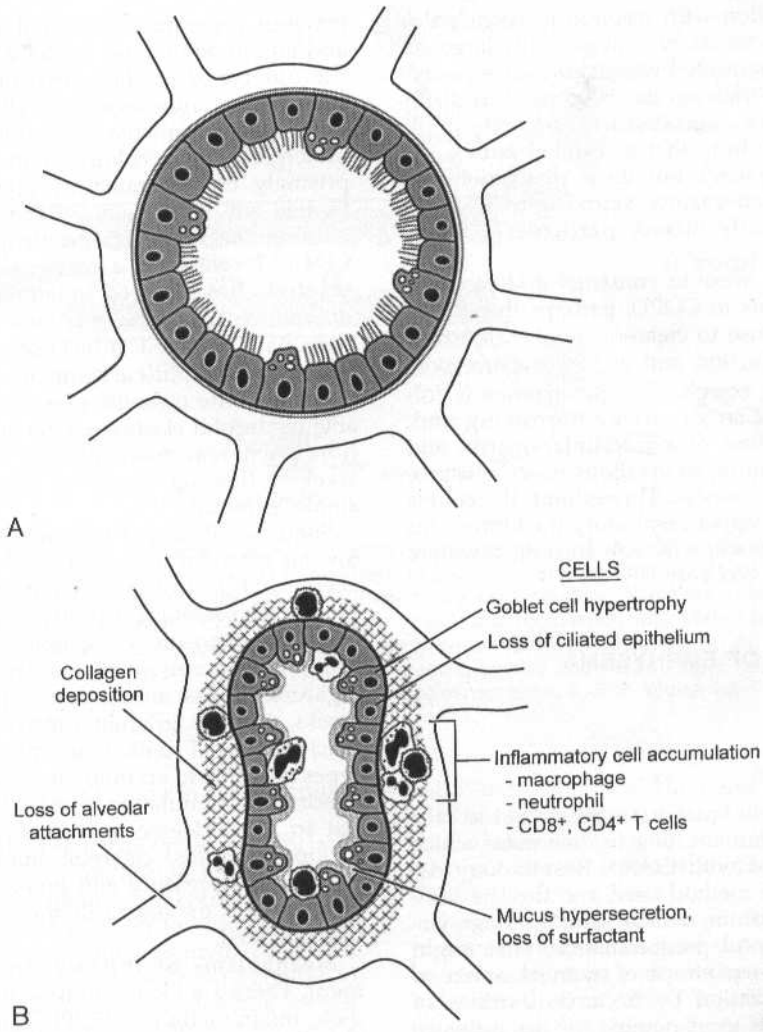


Figure 2. Mechanisms of small airway obstruction in chronic obstructive pulmonary disease (COPD). In contrast to a normal small airway (< 2 mm) *(A)*, note changes in small airways in COPD *(B)*. There are cellular changes including goblet cell hypertrophy, loss of ciliated epithelial cells, and accumulation of inflammatory cells. Mucus hypersecretion and loss of surfactant may also predispose to airway narrowing. Extracellular matrix remodeling is complex with both collagen deposition causing airway obstruction and loss of elastic fibers and hence alveolar attachments that tether open the airways.

Small airways are the major sites of airflow limitation. Small airways show a variety of lesions narrowing their lumina, including goblet cell hyperplasia, mucosal and submucosal inflammatory cells, edema, peribronchial fibrosis, intraluminal mucus plugs and increased smooth muscle.

In large cartilaginous airways chronic bronchitis is associated with hypertrophy of submucosal mucus producing glands. Quantitation of this anatomic change is known as Reid index is based on thickness of submucosal glands to that of bronchial wall. In patients with chronic bronchitis it is 0.44 ± 0.09 otherwise normally 0.52 ± 0.08.

Emphysema begins as an increase in the number and sizes of alveolar fenestrate and results in eventual destruction of alveolar septae and their attachments to terminal and respiratory bronchioles. With centriacinar emphysema the distention and destruction are mainly limited to the respiratory bronchioles with relatively less changes peripherally in the acinus. Panacinar emphysema involves both central and peripheral portions of acinus.

PATHOGENESIS OF EMPHYSEMA

Chronic exposure to cigarette smokes, fumes and dust may lead to inflammatory cell recruitment within the terminal air space of the lungs. Theses cells release elastolytic proteinases that damage the extra cellular matrix of the lung.

In effective luner of other extra cellular matrix components of result in emphysema.

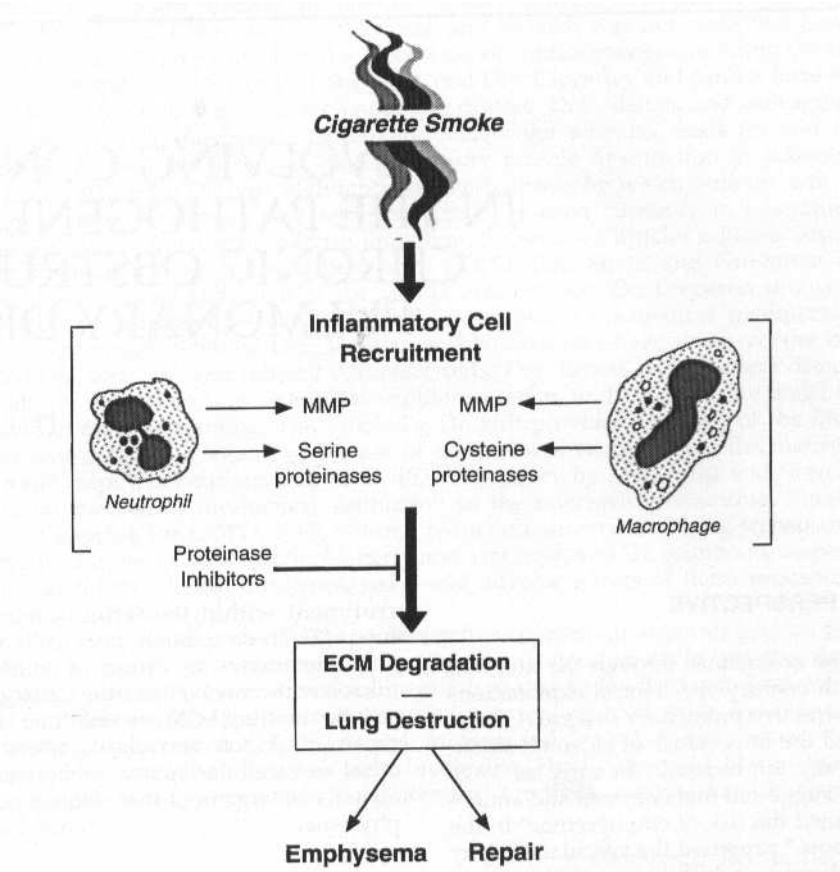


Figure 1. Pathogenesis of emphysema. On long-term exposure to cigarette smoke, inflammatory cells are recruited to the lung, they release proteinases in excess of inhibitors, and if repair is abnormal, this leads to airspace destruction and enlargement or emphysema. MMP = matrix metalloproteinase; ECM = extracellular matrix.

PATHOPHYSIOLOGY

Airflow limitation:

Airflow limitation and increased airway resistance may be caused by loss of elastic recoil during passive exhalation due to emphysema, by increased collapsibility of small airways through loss of radial traction on airways, or to increased resistance due to intrinsic narrowing of small airways.

Hyperinflation:

The residual volume and the functional residual capacity (FRC) are almost higher than normal. In addition prolongation of expiration is associated with obstruction which would lead to dynamic increase in FRC (dynamic hyperinflation).

Dynamic hyperinflation contributes additionally to discomfort associated with air flow obstruction by flattening the diaphragm fiber length and a perpendicular insertion with the lower ribs.

Impaired Gas Exchange:

Maldistribution of inspired air and blood flow is always present. When the mismatching is severe, impairment of gas exchange is reflected in the abnormalities of arterial blood gases. Small airway narrowing causes a decrease in ventilation of their distal alveolar acini. When the alveolar capillaries remain intact, this results in mismatch of ventilation and perfusion leading to mild or moderate hypoxemia.

Pulmonary Circulation in COPD

Pulmonary arterial hypertension develops late in the course of COPD. With the development of hypoxemia and usually hypercapnia. It is the major cardiovascular complication of COPD and is associated with the development of right ventricular hypertrophy (cor pulmonale) and with poor prognosis.

Factors contributing to the development of pulmonary artery hypertension in COPD

(i) ABNORMAL BLOOD GAS TENSIONS

Hypoxemia:

In COPD there is a negative correlation between oxygen saturation of the blood and pulmonary artery pressure. Hypoxemia is known to be a potent arteriolar constrictor in the pulmonary circulation. As the severity of disease progresses in COPD there is more arterial desaturation correlation with an increase in pulmonary artery pressure. Exacerbation of COPD with hypoxemia are associated with acute worsening of pulmonary hypertension. Pulmonary artery pressure (Ppa) can also increase acutely during the episodes of hypoxemia that occur during rapid eye movement of sleep, and it has been suggested that recurrent nocturnal pulmonary hypertension can result in pathologic changes in pulmonary vessels and fixed hypertenison.

Hypercapnea:

In patients with COPD there is a positive correlation between arterial CO_2 pressure ($PaCO_2$) and pulmonary artery pressure. The mechanism could be a change in lung mechanics due to hyperventilation induced by hypercapnea or the potentiation of hypoxic pulmonarly vascoconstriction.

Acidemia:

Hypoxia and acidemia act synergistically to produce pulmonary vasoconstriction in patients with COPD. Thus for a given oxygen saturation, the mean Ppa is higher with increasing arterial hydrogen ion concentration.

(ii) EFFECTS OF ABNORMAL PULMONARY MECHANICS

Changes in airway resistance may augment pulmonary vascular resistance and increase in pulmonary artery pressure, correlating with decrease in FEV_1.

(iii) EFFECTS OF INCREASED CARDIAC OUTPUT

In patients with COPD (in whom the vascular bed may be reduced) even small increase in flow that occurs during exercise may increase pulmonary artery pressure.

(iv) EFFECTS OF BLOOD VISCOSITY

Polycythemia can develop secondary to chronic hyposemia in COPD patients, this contributes to blood viscosity which also adds up to the pulmonary arterial hypertension.

(v) ROLE OF PULMONARY ENDOTHELIUM

There is increasing evidence that endothelial dysfunction is an underlying factor in development of pulmonary artery hypertension. This may result in reduction in nitric oxide synthesis or release in response to hypoxemia.

Thus the putative role of nitric oxide in preventing an excessive rise in pulmonary vascular tone, as a result of stimuli such as hypoxemia, may be lost in COPD. It has also been suggested that nitric oxide may have an inhibitory effect on cell proliferation in the pulmonary vessel walls that therefore has a role in preventing the vascular remodeling that occurs in hypoxic COPD. Circulating levels of endothelin – have been found to be increased in patients who have emphysema and pulmonary hypertension.

PATHOLOGY

Changes in pulmonary circulation occurs characteristically in the peripheral arteries in patients with COPD. An early is the increase intimal thickness in small pulmonary arteries that occur due to accumulation of smooth muscles that are laid down longitudinally along the length of the vessel. Medical hypertrophy in muscular pulmonary vessels, has also been reported in patients of COPD who develop sustained pulmonary arterial hypertension. Pulmonary thromboses may also occur in patients with COPD with may be secondary to peripheral airway inflammation.

Thus structural changes rather than simply hyposic vasconstriction is the major factory in the development of sustained pulmonary hypertension in patients with COPD.

PULMONARY HAEMODYNAMICS IN COPD

Pulmonary haemodynamics in patients with COPD depend on the stage of disease. In patients with mild COPD without severe hypoxemia or hypercapnia, pulmonary arterial pressure is usually normal or only slightly elevated when measured at rest but may rise abnormally during exercises.

Cardiac output is normal, as are right atrial and right ventricular end diastolic pressure. As airflow limitation and arterial blood gas abnormalities, particularly when chronic hypoxemia and hypercapnia are present, pulmonary hypertension develops at rest and worsens on exercise. However in patients with severe COPD when the measurements are made in a clinically stable the pulmonary artery pressure is only modestly elevated.

CONSEQUENCES OF PULMONARY HYPERTENSION IN COPD

Chronic bronchitis and emphysema usually coexist pathologically. Those patients with either predominant chronic bronchitis or emphysema from a minority at the either ends of the disease spectrum of COPD.

The blue and bloated type also know as type B or nonfighter was though to characterize the bronchial type of disease.

These patients had hypoxemia, hypercapnea and secondary polycythemia, they developed pulmonary hypertension relatively early in the course of disease. Right ventricular hypertrophy / cor pulmonale ensure and repeated episodes of right heart failure occurred.

In contrast the pink and puffing variety also known as type A or fighter, were though to represent the emphysematous patients characterized by severe breathlessness, but with preservation of blood gas values and thus no pulmonary hypertension, at least until the later stages of disease.

It was now known that the degree of mucous gland hypertrophy indicative of chronic bronchitis was similar whatsoever the clinical pattern and that more than 50% of patients with blue and bloated clinical pattern had severe emphysema.

THE NATURAL HISTORY OF UNTREATED PULMONARY HYPERTENSION IN COPD

The progression of pulmonary hypertension in COPD is slow. The elevation in pulmonary artery pressure is small and it rarely reaches the levels of primary pulmonary hypertension. Weitzenblum and colleagues found a change of 3mm Hg in the pulmonary artery pressure per year and 33% of patients showed a rise greater than 5mm Hg over 5 years. In these patients hypoxemia and hypercapnia also progressed.

Inspite of the slow progression the presence of pulmonary artery hypertension implies a poor prognosis.

One study shows that the 4 years survival rate was 40% in patients with pulmonary hypertenison compared to 72% in patients whose pulmonary artery pressure was normal (less than 20mg Hg).

The other variable which correlates significally with survival are PaO_2, $PaCO_2$, FEV_1 and the presence of peripheral oedema. Thus although studies have shown an association between the presence of pulmonary hypertension and the prognosis of COPD, pulmonary hypertension may simply be a reflection of the severity of the disease.

COR PULMONALE

Cor pulmonale is defined as right ventricular hypertrophy and dilatation secondary to pulmonary hypertension caused by disease of the lung parenchyma and / or pulmonary vasculature, unrelated to both sides of heart.

The prevalence of right ventricular hypertrophy in a large European study showed that in increased as air flow limitation worsened in patients with COPD, being present in 40% of patients when, FEV_1 was < 1.0L and in 70% when FEV_1 falls to 0.6L. The prevalence of cor pulmonale is also higher in patients with hypercapnia hypoxemia and polycthemia.

Right ventricular contractility is maintained in patients with COPD when their condition is clinically stable, even in the presence of increased pulmonary arterial pressure. But in patients with respiratory failure and peripheral oedema, the pulmonary arterial pressure is greater than that studied in stable state and the right ventricular contractility is decreased.

There is increasing evidence that the oedema which develops late in the course of the disease in patients with COPD may not be entirely due to right ventricular failure. The key factor leading to changes in salt and water balance in patients with COPD is the development of hypoxemia and hypercapnia. The most consistent factor in renal function in patients with hypoxic COPD, particularly those with oedema is a reduction in renal blood flow. Hypercapnia reduces renal blood flow through catecholamine release and vial a nerually mediated action. Arginine vasopressin levels may be inappropriately high in patients with COPD. There is also evidence of activiation of rennine-angiotensin-aldosterone axis. Thus a complex interaction between pulmonary haemodynamics and changes in salt, water and hormonal homeostasis occurs in patients with hypoxic and hypercapnic COPD leading to peripheral oedema.

CLINICAL FEATURES

Symptoms

The characteristic symptoms of COPD are breathlessness on exertion, mostly accompanied by wheeze and cough, which is often but not invariably productive. Most patients have a smoking history of at least 20 pack years before symptoms develop, commonly in the fifth decade.

Breathlessness is the symptom that causes most disability and in associated with loss of lung function over time. It is usually first noticed on climbing hills or stairs. The appearance of breathlessness indicates moderate to severe impairment of airway function. By the time patient seeks medial help FEV_1 has usually fallen to 1-1.5lt in an average male. However, when the FEV_1 has fallen to 30% or less of the predicted values breathlessness is usually present on minimal exertion.

In most patients with COPD cough precedes the onset of breathlessness. Sputum is usually mucoid in character, but becoming purulent during exacerbations. Volume is generally small and less than 60ml per day. As COPD progresses exacerbations becomes more frequent and severe.

Wheezing is present but is neither specific nor indicates the severity of obstruction.

Weight loss and anorexia are signs of severe COPD.

Physical Signs

These are not specific to the disease and depend on degree of air flow limitation and over inflation. In early disease the only abnormal findings is wheeze on forced expiration and a forced expiratory time prolonged beyond 6 seconds with more advanced disease the breathing pattern is characteristic with a prolonged expiratory phase. Some patients adopting pursed-lip breathing on expiration which may reduce expiratory airway collapse. The use of accessory muscles of respiration particularly stemomastoid is seen in advanced disease. These patients adopt the position of leaning forward, supporting themselves with their aims to fix the shoulder girdle and allowing the use of pectoralis and latissimus dorsi to increase chest wall movements. (The tripod position).

In later stages the chest is often barell shaped, an increased anterior posterior diameter, horizontal ribs, prominence of the sternal angle and wide subcostal ange. An inspiratory tracheal tug may be detected. The horizontal position of diaphragm also acts to pull on the lower ribs during inspiration (Hoover's sign).

On percussion there is decreased cardiac and hepatic dullness, indicating over inflation.

Breath sounds may have a prolonged expiratory phase or may be uniformly diminished.

RADIOLOGY

Chest X-ray:

There are no specific features on plan chest-X-ray for chronic bronchitis. The features usually described are for emphysema.

Bronchial wall thickening seen as parallel line opacities on plain chest X-ray has been described in chronic bronchitis.

Radiographic signs for emphysema are:

- Share-sheath treeches.
- Low flattened diaphragm: The border of the diaphragm in the midclavicular line below the seventh rib.
- Height of patients lung being greater than 29.9 cm.
- An obtuse costophrenic angle.
- Reduction in size and number of pulmonary vessels particularly in periphery of lung.
- Heart shadow is vertical and narrow.
- In lateral film increase in the retrosternal airspace.

Computed tomography:

Has greater sensitivity and specificity than plain chest X-ray for emphysema but is rarely necessary except for diagnosis of bronchiectasis and evaluation of bullous lung disease.

SPIROMETRY

Because of imprecisions of clinical findings, objective evaluation of presence, severity and reversibility of airflow obstruction is essential in the diagnostic evaluation of COPD.

Spirometry is themost robust test of airflow limitation in patients with COPD.

Forced expiratory volume in one second (FEV_1) is recommended as the measurement of choice in COPD because

- FEV_1 is reproducible and objective measurement.
- It is simple and relatively quick to measure and can be measured at all stages of disease.
- The forced expiratory maneuver records not only FEV_1 but also FVC and FEV_1 / FVC ratio less than 70% is diagnostic of airway obstruction.
- Studies have shown FEV_1 predicts future morbidity and mortality.
- Serial measurement provides evidence of disease progression.

Global initiative for chronic obstructive lung disease (GOLD) group has reclassified COPD in June 2003 on spirometric values.

FLOW VOLUME LOOPS

Expiratory flow at 75% or 50% of vital capacity have been used as a measure of airflow limitation and provide complementary information to the usual volume time plot. There are problems with the reproducibility of these measurements and hence not preferred for routine clinical use.

Reversibility to bronchodilators

Reversibility tests are important because

1. To help distinguish those patients with marked reversibility (at least 12% or 200ml of FEV_1) who have underlying asthma.
2. To aid with future management.
3. The FEV_1 after bronchodilator is the best predictor of survival.

It is usually recommended that the response to bronchodilator be assessed either using repeated doses from metered dose inhaler or via the nebulised route.

GAS TRANSFER FOR CARBON MONOXIDE

Gas transfer for carbon monoxide values are below normal in many patients with COPD and although there is a relationship between gas transfer and microscopic emphysema the severity of emphysema in an individual patient cannot be predicted from this.

ARTERIAL BLOOD GAS ANALYSIS

Measurement of arterial blood gas is essential in patients with COPD to confirm the degree of hypoxaemia and hypercapnia and in acute exacerbation to determine the hydrogen ion concentration.

OTHER TESTS

Patients with pulmonary hypertension or cor pulmonale with normal day time blood gases should be evaluated for nocturnal desaturation by overnight oximetry.

α_1 AT level are not routinely needed but should be obtained for chronic airflow obstruction in non smokers, as well as COPD patients with bronchiectasis, cirrhosis without apparent risks, premature or bibasilar emphysema in patients under 50 years with unremitting disease and in individuals with family history of AT deficiency.

METHOD OF ASSESSING CARDIAC FUNCTION IN PATIENTS WITH COPD

One of the major difficulties in assessing pulmonary haemodynamics and right ventricular function is the need to measure pressure and flow which involves the use of invasive techniques such as cardiac catheterizaiton.

More recently non invasive technique have been used to assess patients with COPD, these include radiography, electrocardiography, echocardiography, radionucleide ventriculography and magnetic resonance imaging.

CLINICAL ASSESSMENT

Clinical examination is relatively insensitive as a means of detecting pulmonary hypertension or right ventricular dysfunction in patients with COPD, as clinical signs are often obscured by hyperinflation of the chest.

Physical signs that indicate the presence of pulmonary hypertension are accentuated pulmonary component of S_2 early systolic click, an early diastolic murmur of pulmonary regurgitation etc.

Jugular venous pressure are difficult to assess in patients with COPD because of large swings in intrathoracic pressure.

Peripheral oedema can be due to other causes (such as hypoalbuminemia).

A systolic left parasternal heave indicates right ventricular hypertrophy, extra heart sounds and murmur of tricuspid regurgitation suggests right ventricular dysfunction, but these are again modified by hyperinflation.

CHEST X-RAY

The presence of pulmonary arterial hypertension in patients with COPD has been shown to relate to the width of right descending pulmonary artery ≥ 20mm. In addition high value for hilar cardiothoracic ratio was 95% sensitive and 100% specific for the presence of pulmonary hypertension in patients with COPD.

These may be useful as an initial screening test for the presence of pulmonary hypertension, they cannot be used to predict the level of pulmonary artery pressure in individual patients.

ELECTROCARDIOGRAPHY

The voluminous lungs have an insulating effect and there by diminishing the electrical transmission of electrical potentials to the electrodes. The heart descends to a lower position in the thorox due to lowering of diaphragm, altering the position of heart relative to the conventional electrodes.

The common electro cardiographic abnormalities in COPD and COPD with right heart involvement [10]

- Decreased magnitude of electro cardiographic deflections.
- P-waves with right atrial enlargement p-pulmonale i.e., tall peaked P waves in II, III and AVF (P value > 2.5mm)
- QRS abnormalities: Right axis deviationi and QRS > 90^0. At times with extreme north west QRS axis there is the $S_1S_2S_3$ syndrome. In precordial leads there is a general loss of R wave amplitude in all precordial leads. With right ventricular hypertrophy R/S amplitude in $V_6 < 1$. Tendency for incomplete right bundle branch block.

Electrocardiography appears to be specific but has a low sensitivity of picking up right ventricular hypertrophy.

ECHOCARDIOGRAPHY

Because of the deficiencies of clinical examination in detecting pulmonary artery hypertension in patients who have COPD and because pulmonary artery pressure (Ppa) is such as good predictor of prognosis in these individuals, a number of attempts have been made to develop noninvasive methods to estimate it. Echocardiographic measurements of systolic and diastolic and pulmonary pressures have been shown to correlate with Ppa measured by catheterizaiton studies. [11]

The most useful and accurate method of estimating pulmonary artery pressure in patients with chronic obstructive pulmonary disease is systolic transtricuspid gradient calculated from tricuspid regurgitation detected by continuous wave Doppler echocardiography. Continuous wave Doppler determination of tricuspid regurgitation jet velocity and application of modified Bernoulli's equation ($TG = 4V^2$, in which V is the velocity of tricuspid regurgitation jet and TG is the systolic right ventricular to right atrial pressure gradient across the tricuspid valve) permits reliable estimation of pulmonary artery pressure is low. Several studies have shown correlation between echocardiographic measurements of pulmonary artery pressure and pressures measured at cardiac catherterization.

Two dimensional echocardiography can be used to assess right ventricular dimensions and wall thickenings and hence to detect right ventricular volume over load in patients with COPD.

Echocardiography can again be used to assess progression of disease or response to treatment by serial measurements of pulmonary artery pressure and right heart parameters.

MANAGEMENT OF COPD

I. Non Pharmacological measures.

II. Pharmacological measures.

I. Non Pharmacological Measures

Health education: it can play a role in improving stills, ability to cope with illness.

i. Smoking cessation.
ii. Basic information about COPD.
iii. Self management skills.

II. Pharmacological Measures

i. Nicotine replacement therapy in the form of gum, transdermal patch or inhaler is helpful in quitting smoking.

ii. The use of bupropion, a noradrenergic antidepressant, is associated with better abstinence rates.

iii. Bronchodilators are the main stay in the management of COPD.

iv. Inhaled therapy preferred.

v. Choice between β_2 agonist, anticholinergic theophylline or combination theraphy depends on availability and individual response in terms of symptom relief and side effects.

vi. Bronchodilators are prescribed on as needed, or on a regular basis to prevent or reduce symptoms depending on stage.

vii. Long acting bronchodilators are more effective and convenient.

viii. Combining bronchodilators improve efficacy and decrease the risk of side effects compared to increasing the dose of a single bronchodilator.

GLUCOCORTICOSTERIODS

Inhaled glucosteroids are appropriate for symptomatic COPD patients with an $FEV_1 < 50\%$ predicted (Stage III & IV).

Present guidelines recommend a trial of 6 weeks to 3 months with inhaled steroids to identify patients who may benefit from long term inhalation steroid therapy. Long term treatment with oral steroids is not recommended in COPD.

HOME OXYGEN THERAPY (HOT)

Long term oxygen administration (> 15 hours per day) to patients with chronic respiratory failure has been shown to increase survival.

In Nocturnal oxygen treatment trial it has been shown a reduction in pulmonary arterial pressure on exercise after 6 months with either continous or nocturnal oxygen therapy. This study also showed that survival over a period of 8 years was related to the decrease in mean pulmonary arterial pressure during the first 6 months of treatment.

Long term oxygen therapy is introduced in stage IV who have

- PaO_2 at or below 55 mmHg or SaO_2 at or below 88% with or without hypercapnia or
- PaO_2 between 55 mmHg and 60 mmHg if there is evidence of pulmonary hypertension, peripheral oedema suggesting congestive heart failure, or polycythemia (Haematocrit > 55%).

Home oxygen therapy can be supplied in the form of compressed gas cylinders or oxygen concentrators. Oxygen concentrators are most convenient and cost effective.

OTHER PHARAMACOLOGIC TREATMENT

α_1 ANTITRYPSIN AUGMENTATION THERAPY:

Young patients with severe α_1 AT deficiency and established emphysema may be candidates for the same.

ANTIBIOTICS:

Recommended only for exacerbation of Chronic Obstructive Pulmonary Diseases.

VACCINES:

Influenza vaccines can reduce serious illness and death in elderly Chronic Obstructive Pulmonary Diseases patients by about 50%.

MATERIALS AND METHODS

MATERIALS AND METHODS

This study was conducted at Department of Medicine and Thoracic Medicine, Thanjavur Medical College during the period of 2005 – 06.

TOTAL NUMBER OF PATIENTS IN THIS STUDY: -

Number of patients in this study is 60 cases.

INCLUSION CRITERIA: -

The cases in this study have following characters:

(i) Cases between the age group of 30 – 80 years of both sexes.

(ii) These cases having the symptoms suggestive of chronic airway obstruction like cough, cough with expectoration of sputum of more than 2 years duration, dyspnoea, and with (or) without swelling of both legs.

(iii) Cases in whom clinical diagnosis of COPD was made.

(iv) All the cases were subjected to spirometry and the presence of COPD was confirmed by post bronchodilator spirometry values of

i. $FEV_1 < 80\%$.

ii. $FEV_1 / FVC < 0.7$.

iii. Reversibility of obstruction < 15%.

(FEV_1 – Forced Expiratory Volume in 1 sec. FVC – Forced Vital Capacity)

EXCLUSION CRITERIA: -

Case with history of the following diseases were excluded;

(i) Bronchial Ashma. (ii) Pulmonary Tuberculosis.

(iv) Suppurative lung disease. (iv) Systemic Hypertension.

(v) CAHD. (vi) Primary Pulmonary Hypertension.

(vii) Valvular Heart disease. (viii) Sleep Apnoea syndrome.

PROCEDURE:

With above inclusion and exclusion criteria a proforma was prepared to meet the objectives of the study.

GEOGRAPHIC DISTRIBUTION:

Patients were from Thanjavur Town, Pattukottai, and rural areas of Thanjavur, Pattukottai and Ariyalur districts.

All the patients were subjected as follows;

1) Detailed History.

2) Smoking History. i) Age at which smoking was started.

ii) Pack – years was calculated by formula.

$$\text{Pack Year} = \frac{\text{No. of Cigarettes smoked / day}}{20} \times \text{No. of years of smoking}$$

3) General examination and examination of Respiratory system, and other systems.

4) Examination specifically for signs of right heart failure like raised JVP, congestive hepatomegaly and pedal edema.
5) Conventional 12 lead Electro cardiography was taken for ECG changes of COPD and Right Heart failure.
6) X-ray chest PA view and left lateral view.
7) Spirometry: - Spirometry was performed when the patient was clinically stable.

Test was performed with the patient comfortably seated, with clothes loosened. The patients were instructed to take a deep inspiration then close the lips around the mouth piece and blow out as hard and fast as possible, following deep inspiration.

Volume was obtained on the vertical axis of recording paper and time on the horizontal axis. The curve which was obtained is referred to as forced vital capacity curve.

Forced Vital Capacity (FVC) is the volume of air that can be forcibly exhaled (as fast as possible) after a maximal inspiration. It is expressed in litres.

Forced Expiratory Volume in one second (FEV_1)

It is defined as the volume of air expelled in the first second, from the start of maximum expiratory effort of the forced vital capacity. It is expressed in litres or percentage of predicted.

Forced expiratory volume in one second as a percentage of forced vital capacity (FEV_1 / FVC)

It is the percentage of forced vital capacity which is expelled in the first one second of maximal expiratory effort.

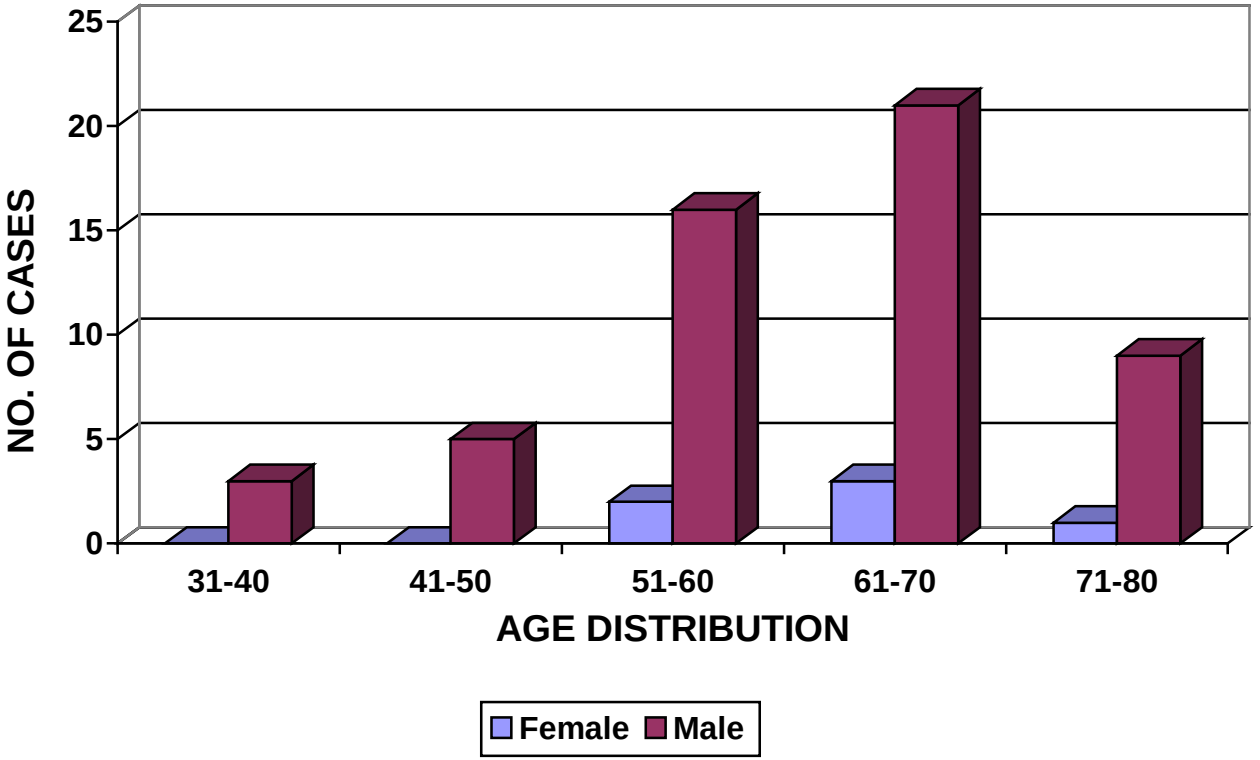
By using spirometry results patients were classified based on GOLD staging of COPD. Severity of clinical symptoms were correlated with GOLD stagning of COPD.

Pack Years of smoking was compared with severity of COPD.

Patients who showed clinical sign of right heart failure where subjected to Echo cardiography for confirmation. In echocardiography Mean maximum TR velocity was recorded in mt/sec and inserted into the modified Bernoulli's equation ($4v^2$), thus calculating the trans tricuspid pressure gradient (TTPG) and the pulmonary artery pressure.

RESULTS
AND
OBSERVATION

BAR DIAGRAM SHOWING AGE DISTRIBUTION OF THE CASES



RESULTS AND OBSERVATION

The results collected from this study was tabulated into different variates and incidence of each variate was calculated in percentage.

Table – 1

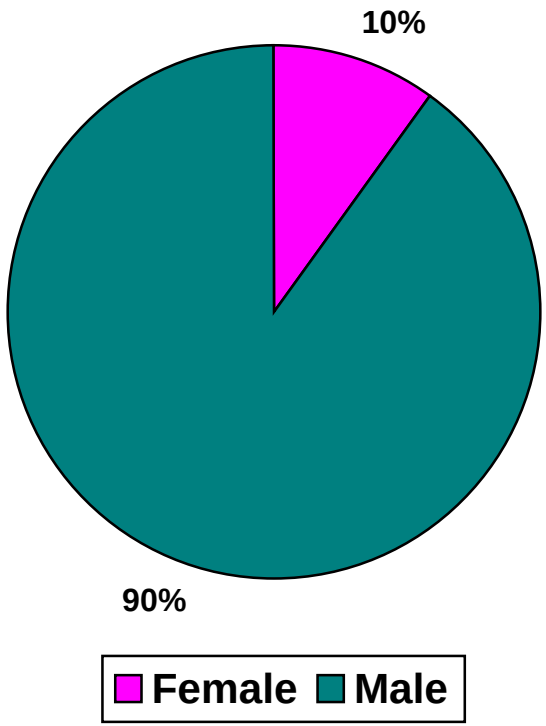
Age in Years	Male	Percentage	Female	Percentage	Total	Percentage
31-40	3	5.5%	-	0%	3	5%
41-50	5	9.2%	-	0%	5	8.3%
51-60	16	29.6%	2	33.3%	18	30%
61-70	21	38.8%	3	50%	24	40%
71-80	9	16.6%	1	16.6%	10	16.6%
Total	**54**	**100**	**6**	**100**	**60**	**100**

From the above table it is observed that the majority of cases among males were between 61 – 70 years of age constituting 38.8% and the minimum number of cases were in the age group of 31 – 40 constituting 5.5%.

Among females the majority of cases were in the age group of 61 – 70 years constituting 40% and nil cases were observed in the age group of 31 – 50 years.

Both sexes put together the maximum cases were in the age group of 61 – 70 years constituting 40% of total cases and minimum cases were observed in the age group of 31 – 40 years which constituted 5% of total cases.

PIE DIAGRAM SHOWING SEX DISTRIBUTION OF THE CASES



Sex distribution:

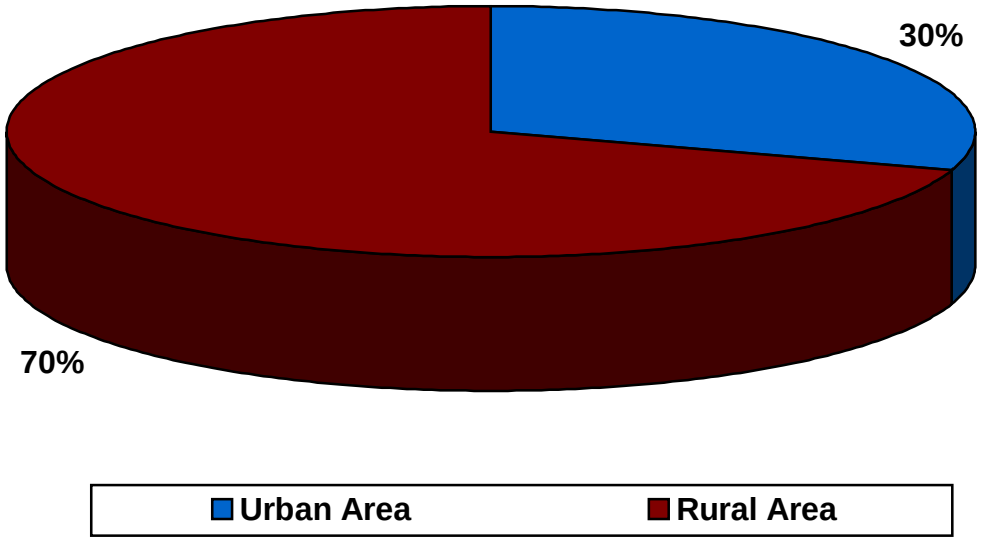
Table – 2

SEX	No. of cases	Percentage
Male	54	90%
Female	6	10%
Total	**60**	**100%**

From the above table, it is observed that the majority of the patients in this present study were belong to male sex.

The male to female ratio was 9:1.

PIE DIAGRAM SHOWING GEOGRAPHIC DISTRIBUTION OF CASES



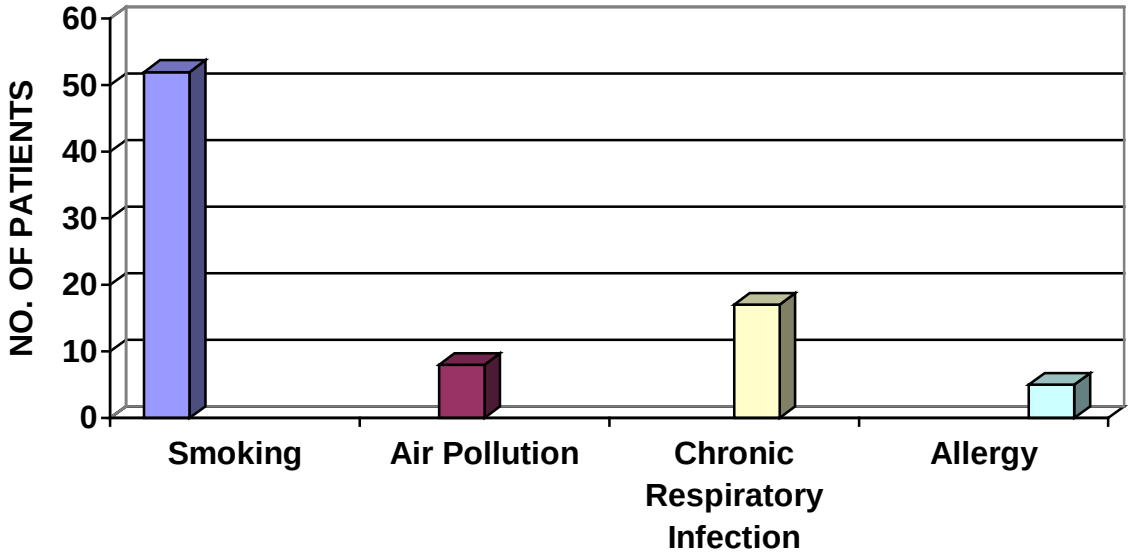
GEOGRAPHICAL DISTRIBUTION:

Table – 3

Area	No. of cases	Male	Female	Percentage
Rural	42	37	5	70%
Urban	18	17	1	30%
Total	60	54	6	100%

It is observed from the above table 42 out of 60 cases were from the rural areas constituting 70% of the total cases. 18 cases from the urban area constituted only 30% of the total.

BAR DIAGRAM SHOWING RISKFACTORS IN CASES



RISK FACTORS

Table – 4

Risk factor		Male	%	Female	%	Total	Percentage
Smoking	Active	50	92.5%			52	86.6%
	Passive			2*	33.3%		
Air pollution		6	11.1%	2	33.3%	8	13.3%
Chronic Respiratory Infection		14	25.9%	3	50%	17	28.3%
Allergy		4	7.4%	1	16.6%	5	8.3%

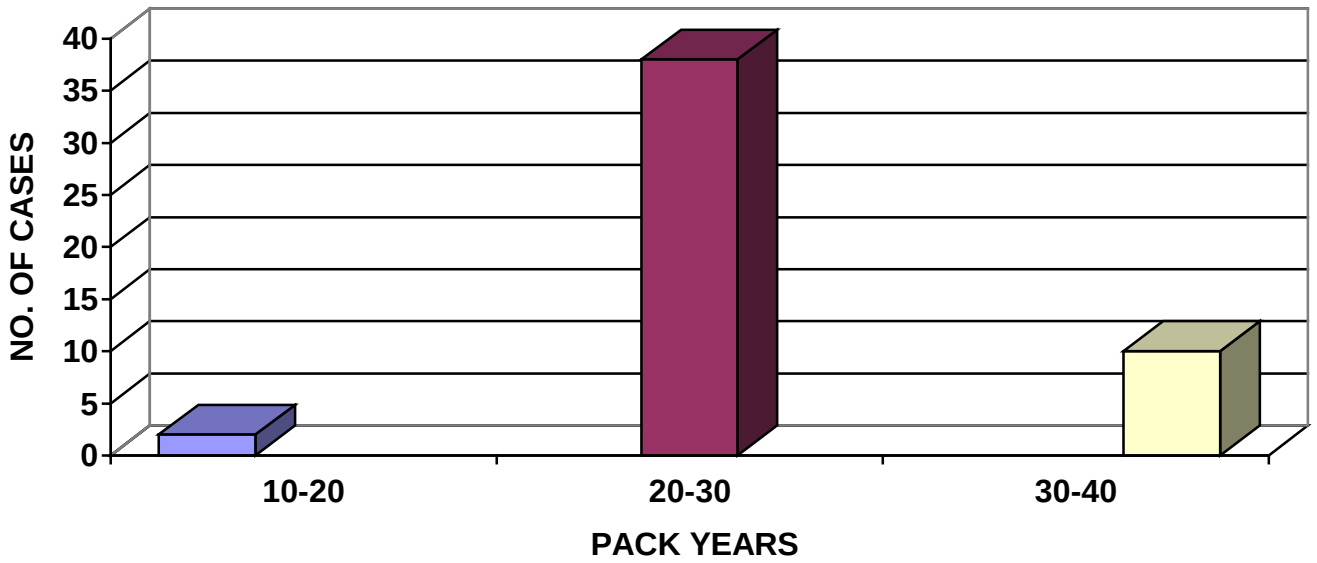
(* Life partners of this two female patients were heavy smokers more than 20 pack-years of smoking).

From the above table it was noted that major risk factor for COPD in males constituted 92.5% in males and 86.6% of total cases.

Chronic respiratory inspection which constituted 50% of risk factor in females and 28.3% of total cases.

Air pollution constituted 13.3% of risk factor followed by allergy with constituted 8.3%.

BAR DIAGRAM SHOWING NO. OF CASES IN PACK YEAR OF SMOKING



INTENSITY OF SMOKING

Table – 5

Pack year	No. of patients	Percentage
10 – 20	2	4%
20 – 30	38	76%
30 – 40	10	20%

From the above table it can be observed that the majority of patients had more than 20 pack years of smoking which constituted 96% of total cases.

All the patients in this table were active smokers and all the patients were male.

DURATION OF DISEASE

Table – 6

Disease Duration	Males	Females	Total	Percentage
2 – 5 years	2	-	2	3.3
6 –10 years	4	-	4	6.6
11 – 15 years	19	3	22	36.6
16 – 20 years	23	2	25	41.6
> 20 years	6	1	7	11.6
Total	**54**	**6**	**60**	**100%**

From the above table it is observed that majority of patients had more than 10 years duration of disease.

Maximum number of cases were seen 16 – 20 years duration of disease constituting 41.6% of cases.

DISTRIBUTION OF SYMPTOMS

Table – 7

Symptoms	No of patients	Percentage
Cough	60	100%
Cough with expectoration of sputum	56	93.3%
Wheeze	46	76.6%
Breathlessness	52	86.6%
Swelling both legs	5	8.3%

From the above table it is noted that all the patients in this study had cough. Cough was the major symptom constituted 100% in this study.

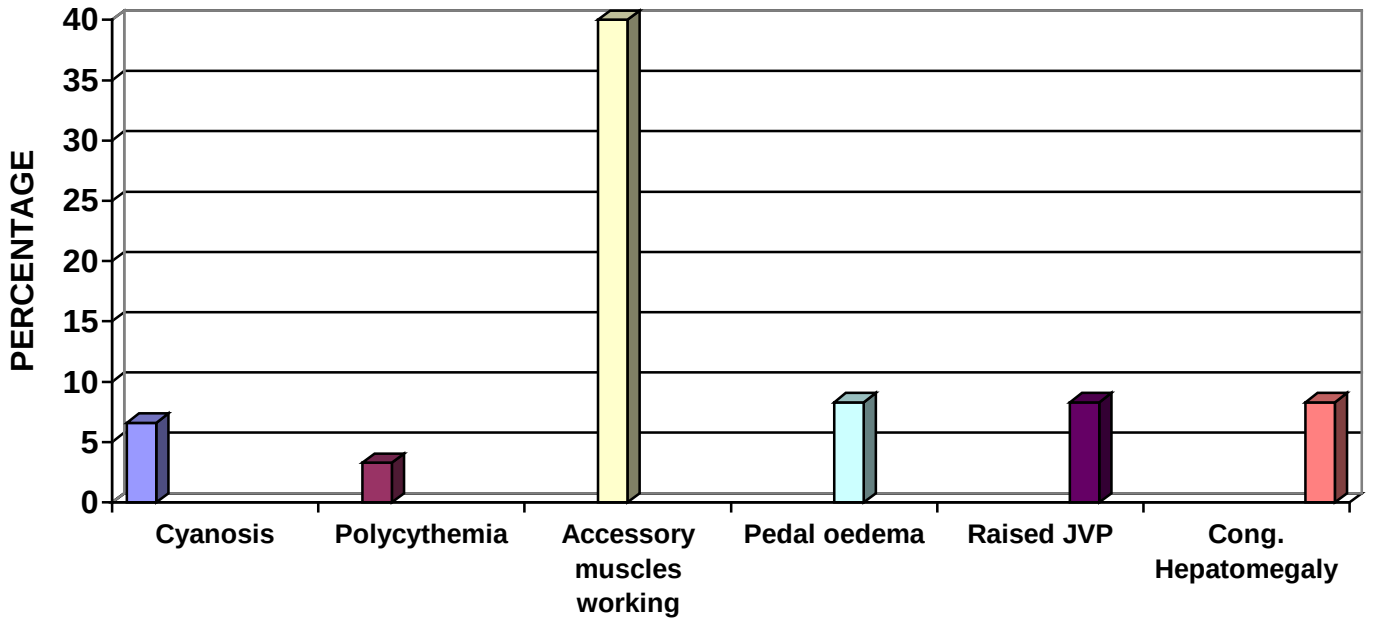
Cough with expectoration of sputum was present in 93.3% of cases.

Breathlessness which constituted 86.6% of cases.

Wheeze which constituted 76.6% of cases.

Swelling of both legs were observed in 8.3% of cases.

BAR DIAGRAM SHOWING CLINICAL SIGN IN PERCENTAGE



DISTRIBUTION OF CLINICAL SIGNS

Table – 8

Clinical signs	No. of patients	Male	Female	Percentage
Cyanosis	4	3	1	6.6%
Polycythemia	2	1	1	3.3%
Accessory muscles working	24	20	4	40%
Pedal oedema	5	4	1	8.3%
Raised JVP	5	4	1	8.3%
Congestive Hepatomegaly	5	4	1	8.3%

From the above table it is observed that active accessory muscles of respiration (inter costal in drawing) was the major clinical sign observed in 40% of the cases.

In this study polycythemia was observed in 3.3% of cases.

Pedal oedema, Raised JVP and Congestive Hepatomegaly were observed in 8.3% of cases.

DISTRIBUTION OF RADIOLOGICAL FINDINGS

Table – 9

Chest x-ray	No. of patients	Percentage
Low flattened diaphragm with hyperinflated lungs	39	65
Obtuse costo phrenic angle	30	50
Reduction in no and size of pulmonary vessels in periphery	12	20
Normal	12	20

From the study it is observed that Low flattened diaphragm with hyperinflated lungs was the major radiological feature present in 65% of cases.

50% of cases were shown obtuse costo phrenic angle in chest x-ray.

20% of the patients in this study had Normal x-ray chest.

DISTRIBUTION OF ECG CHANGES IN VARIOUS STAGES OF COPD

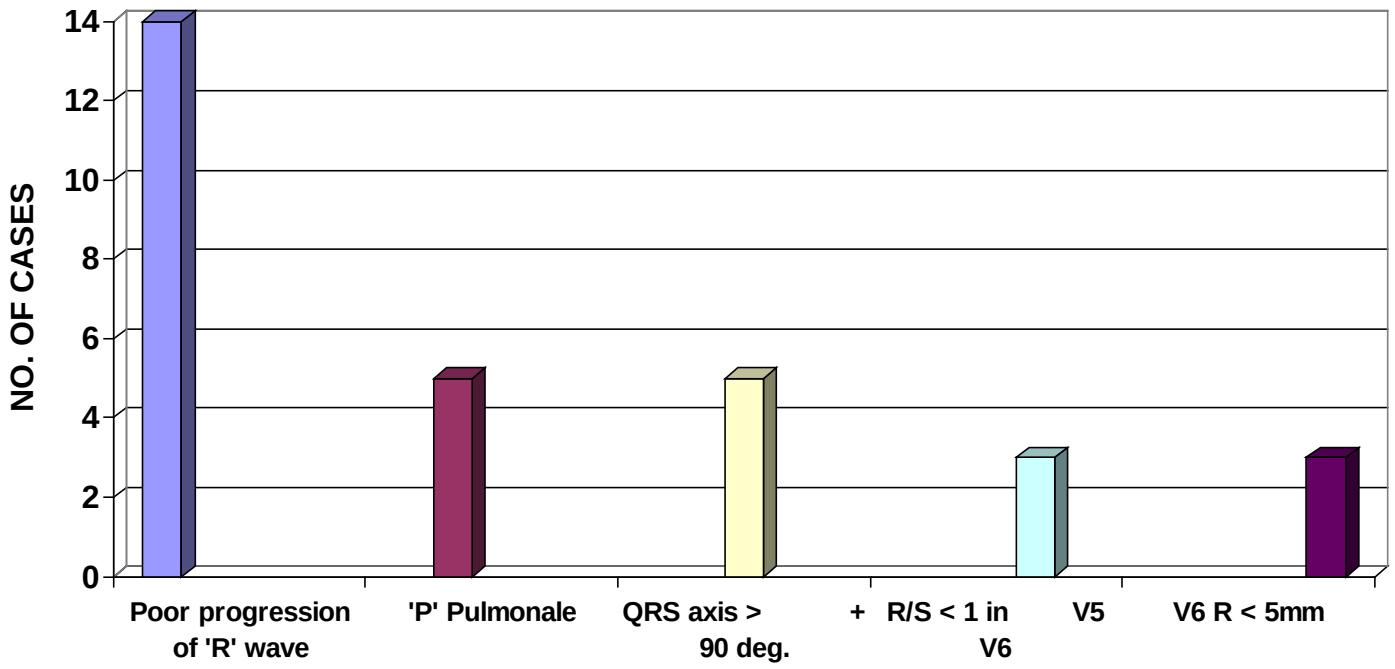
Table – 10

Stage of COPD (*GOLD criteria*)	**ECG changes**					
	Poor 'R' Wave progression in $V_1 – V_6$	**'P' Pulmonale**	**QRS > + 90^0 in frontal plane**	**R/S < 1 in V_5 V_6**	**V_6 < 5 mm**	**Total**
Stage I	-	-	-	-	-	-
Stage II	3	-	-	-	-	3
Stage III	5	0	0	-	-	5
Stage IV	6	5	5	3	3	22
Total	**14**	**5**	**5**	**3**	**3**	**30**

From the above table it is observed that more number of ECG changes were seen in patients in Stage IV of COPD.

There was no ECG changes observed in patients with Stage I of COPD.

BAR DIAGRAM SHOWING ECG CHANGES IN COPD CASES



DISTRIBUTION OF ECG CHANGES

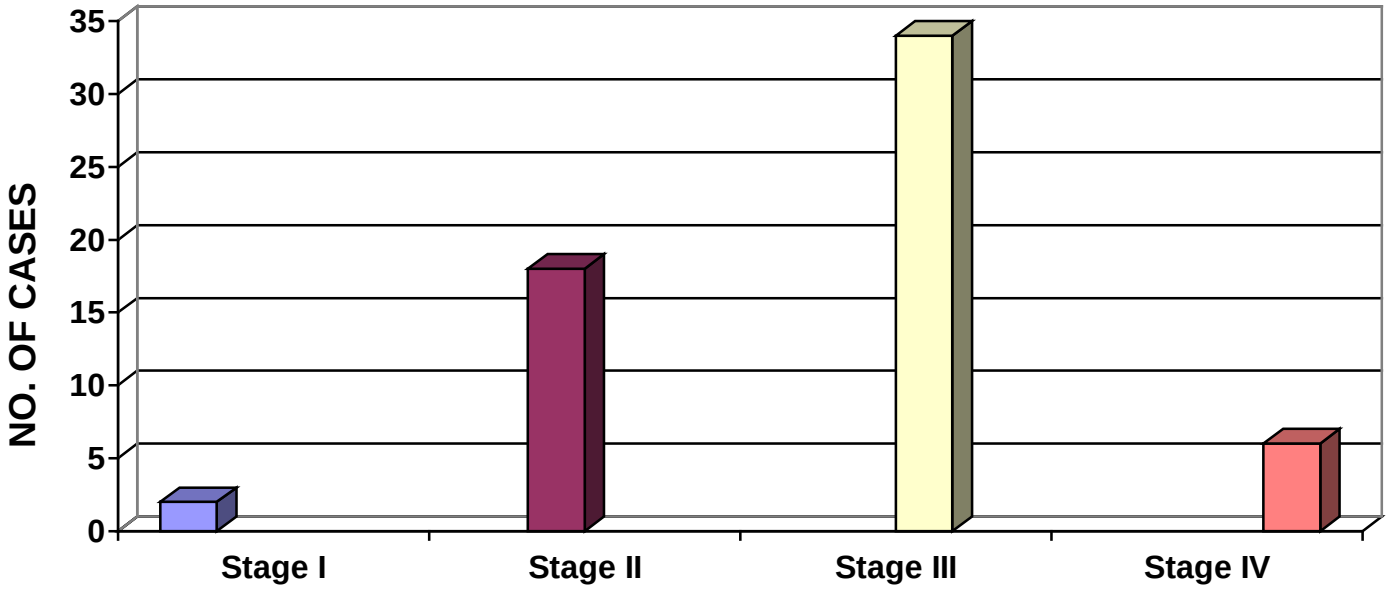
Table – 11

ECG changes	Male	Female	Total	Percentage
Poor 'R' Wave progression in $V_1 - V_6$	12	2	14	23.3%
'P' Pulmonale	4	1	5	8.3%
QRS > + 90^0 in frontal plane	4	1	5	8.3%
R/S < 1 in V_5 V_6	2	1	3	5%
V_6 < 5 mm	2	1	3	5%

From the above table it is observed that most frequent ECG abnormality in this study was poor progression of 'R' wave which was observed in 23.3% of cases.

8.3% of cases showed evidence of cor pulmonale like 'P' Pulmonale and right axis deviation.

BAR DIAGRAM SHOWING GOLD STAGE OF COPD CASES IN PERCENTAGE



DISTRIBUTION OF CASES IN GOLD STAGING OF COPD

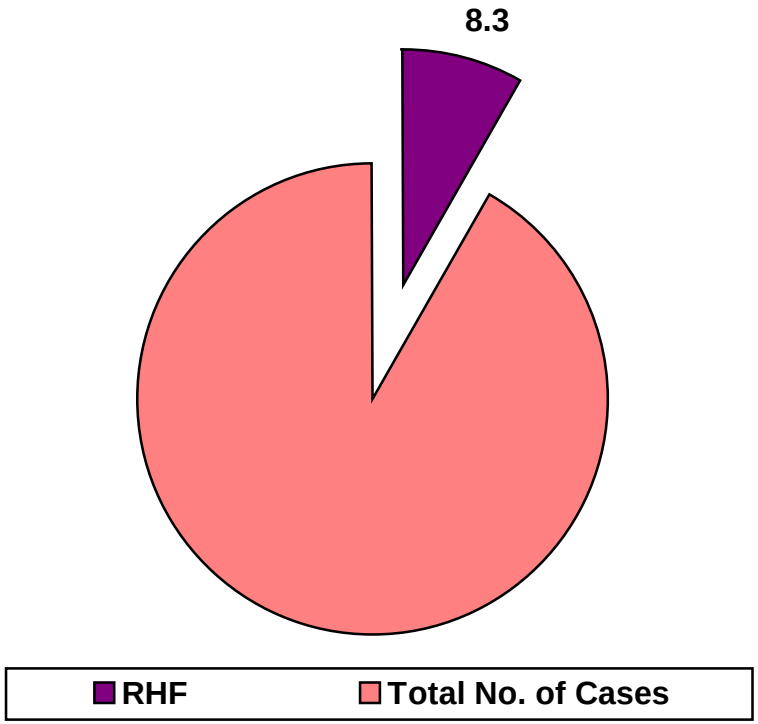
Table – 12

GOLD stage of COPD	Severity	No. of cases			Percentage
		Male	Female	Total	
Stage I $FEV_1 \geq 80\%$	Mild	1	1	2	3.33%
Stage II Fev_1 50% – 80%	Moderate	17	1	18	30%
Stage III FEV_1 30% – 50%	Severe	31	3	34	56.6%
Stage IV FEV_1 < 30%	Very Severe	5	1	6	10%

From the above table, majority of the patients in the study were Gold Stage III of COPD showing severe airflow obstruction (FEV_1 30% - 50%).

10% of the patients showed FEV_1 < 30% very severe air flow obstruction belonged to Stage IV of COPD.

PIE DIAGRAM SHOWING CLINICAL SIGNS OF RHF IN COPD CASES IN PERCENTAGE



DISTRIBUTION OF RIGHT HEART FAILURE IN COPD

Table – 13

Total No. of patients			Patient with clinical signs of Right Heart Failure			Percentage
Male	**Female**	**Total**	**Male**	**Female**	**Total**	
54	6	60	4	1	5	8.33%

From the above table it is observed that 5 cases having the clinical features of right heart failure constituting 8.33% of total.

All the 8.33% are also proved by echocardiography.

DISTRIBUTION OF RIGHT HEART FAILURE IN DURATION OF DISEASE

Table - 14

Duration of disease	No. of cases	
	Male	**Female**
2 – 5 years	-	-
6 –10 years	-	-
11 – 15 years	-	-
16 – 20 years	1	-
> 20 years	3	1
Total	4	1

From the above table it is observed more number of patients with right heart failure having disease more than 20 years of duration constituting 80% of the total cases of right heart failure in COPD.

DISCUSSION

DISCUSSION

1. Total number of cases were 60 in this study.

AGE AND SEX:

2. In the present study maximum number of cases were in the age group of 61-70 years. This coincides well with the various study as follows,

   i) Wig K.L Guleira K. S et al 1964 study showed maximum number of cases in 55-65 years. [20]

   ii) Higham MA Dawson et al at 2001 showed maximum number cases in 61-70years. [52]

   iii) Suzzane Hurd et al showed maximum number of cases in 60-70 years. [4]

3. In this study, it was noted the incidence of COPD was higher in males than females with male to female ratio of 9:1.

   Male cases accounted for 90% of the total cases in this study.

   This finding also coincides with the following studies,

   1. Trivedi H.S. et al study showed 80% of cases were male. [46]
   2. Mieguere et al showed 93% of cases were male. [44]

GEOGRAPHICAL DISTRIBUTION:

In this study it is observed that 70% of patients were from rural areas and 30% were from urban areas.

The study by Bhattacharya S.N. et al, 1975, also made similar observation. [21]

Anderson et al, 1963, made similar observation in his study. [54]

In spite of heavy Air pollution, the urban areas contributed only 30% of the cases in this study.

RISK FACTOR:

Among the various risk factors Smoking is the major risk factor accounted for 87% of the causative risk factor in this present study.

Ashley F, Kannel WB et al, 1975, made similar observation. [54]

Burrows et al, 1979, made similar observation. [25]

Intensity of smoking was expressed in pack-years in this study. As the number of pack-years more than 20, increased predisposition to COPD was observed in many studies.

In this study, most of the patients were 20-30 pack-years of smoking constituting 96% of the total.

Higgins ITT et al, 1959, observed in his study 86% patients were more than 30 pack-years of smoking. [55]

Chronic respiratory infection which constituted 28.3% was the second major risk factor observed in this study, followed by Air Pollution and Allergy.

Burrows B et al, 1977, also made similar observation. In his study Chronic respiratory infection constituted 35%. [56]

Air pollution constituted 13.3% of risk factor and Allergy 8.3% of risk factors. [24]

DURATION OF DISEASES:

In this present study of most of the cases were above 10 years of duration of the disease.

Barnes BJ et al, 1999, in his study observed 55% had more than 15 years. [57]

Symptoms:

In this study cough and cough with expectoration of sputum was observed in 100% and 93% of cases respectively

Burrows et al, 1979, made in his study 96% of patients had cough with exportation of sputum. [25]

Dyspnoea was observed in 86.6% cases.

Altose MD et al, 1985, observed 90% of cases had dyspnoea. [58]

Physical signs:

This study observed acting accessory muscles of respiration was the major physical sign observed in 40% of cases.

Polycythemia (Secondary to hypoxia) was observed in only 3.3% of cases.

Pedal oedema raised JVP and congestive hepatomegaly observered in 8.3% of cases.

Chest X-ray PA view:

In this present study most common radiological finding in chest X-ray PA view was hyper inflated lungs with low flattened diaphragm which constituted 65% of cases.

Thurlbeck WM et al, 1970, observed in his study 78% of the patients showed radiological evidence of emphysema with chronic bronchitis. [59]

Normal X-ray chest was observed in 20% of cases.

Electrocardiography:

By electrocardiography poor progression of 'R' Wave was the most frequent abnormality detected in this present study constituted 23.3%.

8.3% of cases showed 'P' pulmonale and QRS axis > + 90^0

R/S < 1 in V_5 -V_6 observed in 5% of cases.

R wave in V_6 < 5 mm was observed in 3.3% of cases.

Boushy SF et al., 1971, in his study observed that 'P' pulmonale and QRS axis > + 90^0 were the major ECG changes present in 12.5% of the patient. [10]

It was observed from this study more number of cases with ECG changes were seen in Stage IV COPD. Which denoted that as the severity of COPD increases, ECG changes also increases.

SPIROMETRY:

In the study most number of patients were in GOLD Stage III COPD which constituted 56.6% of cases. The study by Higham et al [52] showed that majority of patients were in Stage III (BTS Scheme for COPD) constituted 57 – 58% of cases.

Renzeti AD et al, 1966 observed 76% of his cases belong to moderate to severe stages of COPD. [49]

Right Heart Failure:

In this study it is observed that 8.3% of cases showed clinical evidence of right heart failure. All the patients who showed the clinical evidence of right heart failure were subjected to echocardiography and confirmed the presence of right heart failure.

Mattay R et al, 1981, observed that 12.5% of his cases were showed evidence of cor pulmonale. [16]

SUMMARY

In this study on Chronic Obstructive Pulmonary Diseases the following facts were observed.

COPD is the disease of aged as evidenced by majority of patients in the present study were belong to the age group of 50 – 80 years.

COPD has male predominance as evidenced by 9:1 ratio of Male to Female due to high prevalence of smoking habits observed in males.

Cigarette smoking was the major risk factor for COPD in this study.

Cough / Cough with expectoration of sputum was the major clinical symptom observed in this study.

Acting accessory muscles of respiration with pursed lip breathing was the major clinical sign observed in this study.

Spirometry is the mandatory investigation to diagnose and assess the severity of COPD.

Most number of cases had severe airway obstruction which was not reversible.

High flattened diaphragm and hyper lucent lungs were the most common chest x-ray finding observed in this study.

Poor progression of 'R' wave in chest leads, P Pulmonale, QRS > + 90^0, R wave in V_6 < 5mm and R/S < 1 in V_5 V_6 were the ECG changes observed in this study.

The clinical incidence of Right heart failure in COPD in this study was 5% which was confirmed by ECHO.

CONCLUSION

CONCLUSION

1. Chronic Obstructive Pulmonary Diseases is a preventable disease as smoking is the major risk factor for Chronic Obstructive Pulmonary Diseases.
2. Spirometry is mandatory to diagnose and assess the severity of Chronic Obstructive Pulmonary Diseases FEV_1 was the single most important parameter in spirometry to diagnose Chronic Obstructive Pulmonary Diseases along with the less than 15% of reversibility of airflow obstruction to bronchodilators.
3. Severity of Chronic Obstructive Pulmonary Diseases has direct relation with incidence of ECG changes in Chronic Obstructive Pulmonary Diseases.
4. Clinical signs of right heart failure in Chronic Obstructive Pulmonary Diseases were effective in screening the patients for cor pulmonale.
5. Right heart failure denotes severity and duration of Chronic Obstructive Pulmonary Diseases.

PROFORMA

PROFORMA

STUDY ON COPD

NAME: **AGE:** **SEX: M/F**

ADDRESS: **I.P / O.P. NO.:**

OCCUPATION: **ECONOMY CLASS:** High

Middle

Low

PRESENTING SYMPTOMS: **DURATION**

- Cough
- Cough with expectoration of sputum
- Breathlessness
- Wheeze
- Chest pain
- Fever
- Haemoptysis
- Both Legs Swelling
- Others (Specify)

RISK FACTORS AND TRIGGERS:

- Smoking
- Air Pollution like, Smoke / Fumes / Dust
- Recurrent respiratory infection.
- Atopy and Allergy

SMOKING HISTORY: ♦ Active ♦ Passive

Active: ♦ Cigarette / Ciger / Beedi

♦ Age at smoking started.

♦ Intensity of smoking in Pack-years.

Passive: ♦ Father / Husband / Son / Other (Specify Relation)

♦ Pack-year of smoking

Family History: (A) Bronchial asthma **(B)** COPD

OTHER DISEASES:

♦ Pulmonary TB ♦ CAHD ♦ HT ♦DM ♦ Valvular Heart disease

GENERAL EXAMINATION:

Clinical signs:

♦ Anemia ♦ Polycythemia ♦ Cyanosis

♦ Accessory muscles of respiration active / normal

♦ Pursed lip breathing present / absent

♦ Lymphnodes ♦ Pedal oedema ♦ JVP

Vital signs:

PR: BP: RR:

Examination of systems:

RS: Inspection

Palpation

Percussion

Auscultation

CVS:

P/A: Congestive hepatomegaly present / absent

CNS:

INVESTIGATIONS:

- Urine **i)** Alb. **ii)** Sugar
- Blood **i)** TC **ii)** DC **iii)** Hb% **iv)** ESR
- Blood Sugar

  Urea
- Chest X-ray i) PA View ii) Left Lateral view
- Spirometry

GOLD Stage	**FEV_1**	**FEV_1 / FVC**
Stage I		
Stage II		
Stage III		
Stage IV		

- ECG i) Normal

  ii) Poor progression of 'R' wave in chest leads

  iii) 'P' Pulmonale

  iv) QRS > + 90^0

  v) R / S in V_5 - V_6 < 1

  vi) V_6 R < 5mm
- Echo:

BIBLIOGRAPHY

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1. Global initiative for chronic obstructive pulmonary disease, 2003. *Global obstructive lung disease*. NHLBI / WHO report. NIH Publication, No. 2701.

2. John J. Reilly Jr. Edwin K. Silver man, Steven D Shapiro, Chronic obstructive pulmonary disease" Chapter 242, Pg 1547 – 57, in *Harrison's Principles of Internal Medicine* Volume II by Eugene Brauwald, Anthony S Fauci, Dennis L Kasper.

3. William MacNee "Chronic Bronchitis and emphysema". *Crofton and Douglass Respiratory Diseases* Chapter 616, edited by Anthony Seaton, Douglas Seaton, fifth edition, Blackwell Sciences, Volume – 1, 650.

4. Suzanne Hurd, Ph.D. 2000. "The impact of COPD on lung health world wide, epidemiology and incidence:, *Chest*, Vol. 117 : 1S – 4S.

5. Scott W, and Gravin. 1941. *American Heart Journal*, 22:50.

6. I.T.T. Higgins. 1959. "Tobacco smoking, respiratory symptoms and ventilatory capacity", *British Medical Journal*, 325 – 29.

7. Benjamin Burrows, Richard H. Earle. 1669. "Course and prognosis of chronic obstructive lung disease", *The New England Journal of Medicine*, Vol. 280 – 8, Pg. 297 – 404.

8. Christer Larson. 1978. "Natural history and life expectancy in severe alpha – 1- antitryprin deficiency Piz", *Acta Med Scand*, 204 : pg. 345 – 351.

9. *Medical Research Council*. 1965. "Definition and classification of chronic bronchitis for clinical and epidemiological purpose", Lancent, 1: 775.

10. Boushy SF et al., 1971. "Electrocardiogram in chronic obstructive pulmonary disease". *Amer Rev Resp Disease*, 104 : 1067 – 70.

11. Benjamin Burrows et al., 1972. "Patterns of cardiovascular dysfunction in chronic obstructive lung disease", *The New England Journal of Medicine,* Vol. 286, No. 17 : Pg. 912 – 917.

12. Boushy SF et al., 1964. "The prognostic values of pulmonary function test in emphysema", *Am Rev Resp Disea*, 90 : 553.

13. Boushy SF et al., 1964. "Factors affecting prognosis in emphysema", *Dis Chest*, 45 : 402.

14. Boushy SF et al., 1973. "Prognosis in chronic obstructive pulmonary disease", *Am Rev Resp Dis,* 108 : 1373.

15. Boushy SF et al., 1977, "Haemodynamic changes in chronic obstructive pulmonary disease", *Chest*, 72 – 5, Pg. 565 – 570.

16. Mattay R. et al., 1981. "Cardiovascular performance in chronic obstructive pulmonary disease – Symposium on obstructive lung disease", *Medical Clinics of North America*, 65(3) : 489.

17. Yamaoks AT et al, Study on electrocardiogram in lung diseases, 1967.

18. Srider GL. et at., 1985. "The definition of emphysema. Report of a National Heart, Lung and Blood Institute, Division of Lung Diseases, Workshop", *Am Rev Resp Dis*, 132 : 182.

19. Sharma S.K. 2003. 'Chronic obstructive lung disease" API text book of medicine, Siddarth N. Shah, 7th edition, Chapter 6, Pg. 297.

20. Wig K.L, Guleira K.S. et al., 1964. "Certain clinical and epidemiological aspects of chronic bronchitis as seen in Northern India", *Indian Journal of Chest Diseases*, 6 : 183 – 94.

21. Bhattacharya SN. et al., 1975. "Chronic bronchitis in rural population", *Indian J Chest* Dis, 1 : 17.

22. Doll R, Peto R. 1976. "Mortality in relation to smoking : 10 years observation in British doctors', *Brit. Med*. J, 280 : 967.

23. United States Department of Health and Human Services, Public Health Service 1990 "The Health Benefits of Smoking Cessation. A Report of the Surgeon General", Washington DC, Government Printing Press.

24. Beaty TH, Mankes HA, Cohen BH, et al: Risk Factors associated with longitudinal changes in pulmonary function. 1984.

25. Burrows B. et al., 1979. "Quantitative relationship between cigarette smoking and ventilatory function", *AM Rev Resp Dis*, 115 : 195.

26. British Thoracic Society, 1997 Guidelines for the management of chronic obstructive pulmonary disease, Thorax : 52 (Suppl 5).

27. Chetty K.G. et al., 1982. "Identification of pulmonary hypertension in chronic obstructive pulmonary disease form routine chest radiographs", Am Rev Resp Dis, 126 : 338 – 41.

28. Leo Schmroth. 1997. "An introduction to electrocardiography" Edit Colin Schamroth, Seventh Ed., 'Emphysema chronic obstructive airways disease", *Blackwell Science*, Chap 20, Pg. 233.

29. Masuyma T. et al., 1986. "Continous wave Doppler echocardiographic detection of pulmonary regurgitation and its application to non-invasive estimation of pulmonary artery pressure", Circulations : 74 : 484 – 92.

30. Noctural Oxygen treatment trial group, 1980. “Continuous or nocturnal oxygen therapy in hypoxemic chronic obstructive pulmonary disease : A clinical trial”, *Ann Intern Med*, 93 : 391.

31. Harvey R.M. et al., 1951. “A influence of chronic pulmonary disease on heart and circulation”, *Am J Med*, 10 : 719 – 38.

32. Fowler N.O. et al., 1952. “The cardiac output in chronic cor pulmonale”, *Circulation,* 6 : 888 – 893.

33. Weitzenblu E. et al., 1978. “Clinical functional and pulmonary haemodynamic course of patients with chronic obstructive pulmonary disease followed up over 3 years”, *Respiration,* 36:1.

34. Aber GM. Et al., 1963. “Inter relationships between renal and cardiac function and respiratory gas exchange in obstructive airway disease”, *Clin Sic*, 25 : 159 – 170.

35. Enson Y. et al., 1968. “The influence of hydrongen ion concentration and hypoxia on the pulmonary circulation”, *J Clin Invest,* 43 : 1146 – 62.

36. Haris P. et al., 1968. “The influence of airway resistance and alveolar pressure on the pulmonary vascular resistance in chronic bronchitis”, *Cardiovasc Res,* 2 : 84 – 94.

37. Schrijen F. et al., 1978. “Pulmonary and systemic hemodynamic evolution in chronic bronchitis”, *Am Rev Respi Dis,* 117 : 25.

38. Mincada S. et al., 1987. “Prostacyclin and endothelial derived relaxing factor : biological interaction eds. Thombosis and Haemostasis”, Belgium: University Press Leuven, 597 – 618.

39. Yamakani T. et al., 1997. “Arterial endothelial – 1 level in pulmonary emphysema and interstitial lung disease. Relation with pulmonary hypertension during exercise”, *Eur Respir Jr*, 10 : 2055.

40. Wilkinson M. et al., 1988. “A pathophysiological study of 10 cases of hypoxic cor pulmonale”, *Q.J. Med*, 66 : 65 – 85.

41. Mahler DA. et al., 1984. “Right ventricular performance and central circulatory haemodynamics during upright exercise in patients with chronic obstructive pulmonary disease”, Am Rev of Resdp Dis, 130 : 722.

42. Cooper CB. Et al., 1987. “Twelve year clinical study of patients with hypoxic cor pulmonale given long term domiciliary oxygen therapy”, Thorax, 42 ; 105 – 110.

43. Boushy SF. et al., 1964. “Factors affecting prognosis in emphysema’, Respiratory Diseases, 45 : 402 – 411.

44. Michel Miguer’s et al, 1990. “Doppler Echocardiography in the diagnosis of pulmonary hypertension in COPD”, *Chest*, Pg. 280-285.

45. Simpson T. et al., 1968. “Chronic bronchitis and emphysema with special reference to prognosis”, *British Journal of Diseases of the Chest,*62, 57 – 59.

46. Trivedi HS, Joshi MN. et al, 1992. “Echocardiography and pulmonary artery pressure : Correlation in chronic obstructive pulmonary disease”, J. Post Grad Med, Vol. 38 (1) 24 – 6.

47. Diener CF, Burrows B. 1975. “Further observations on the course and prognosis of chronic obstructive pulmonary disease”, *Am Rev Resp Dis,* 111 ; 719 – 724.

48. Valleric V Mclaughlin, Stuart Rich. 2001. “Cor pulmonale” E-Braunwald Eds. Heart Disease, A Textbook of Cardiovascular Medicine. W.B. Saunders Company, Chap 54 ; Pg. 1936.

49. Renzeti AD. et al., 1966. “The Veterans Administration Cooperative Study of Pulmonary Function, 111, Mortality in

relation to respiratory function in COPD", *AM J Med*, 41 : 115 – 9.

50. Mitchell R.S. et al., 1964. "Chronic obstructive bronchopulmonary disease. IV, The clinical and physiological differential of chronicbronchitis and emphysema", *Am J Med Sci*, 247 : 513 – 517.

51. MacNee W. et al., 1997 "The pathogenesis of peripheral oedema in COPD", *Clin Pulm Med*, 4 : 309.

52. Higham M.A. Dawson D. et al., 2001. "Utility of Echocardiography in Assessment of Pulmonary hypertension secondary to COPD", European Respiratory Journal. Vol. 17 (3), 350 – 355.

53. Anderson et al, Chiliwack respiratory survey, 1963. The prevalence of respiratory disease in a rural Canadian town.

54. Ashely F, Kannel WB et al, Pulmonary function: Relation to aging, cigarette habit, and moratality: The Framingham Study, 1975.

55. Higgins ITT et al, Tobacco smoking, respiratory symptoms, and ventilatory capacity: Studies on random samples of the population, 1959.

56. Burrows B et al, The relationship of childhood respiratory illness to adult obstructive airways disease, 1977.

57. Barnes BJ et al, Medical progress in COPD, 1999. Thorax 54:245.

58. Altose MD et al, Assessment and management of breathlessness, 1985.

59. Thurlbeck WM et al, Chronic obstructive lung disease, A comparison between clinical, roentgenologic, function and

morphological criteria in chronic bronchitis, asthma and bronchiectasis, 1970.

KEY TO MASTER CHART

i. **P / Y –** Pack Years

ii. **C.R.I. –** Chronic Respiratory Infection

iii. **SYMPTOMS**

a – Cough

b – Cough with expectoration of sputum

c – Wheeze

d – Breathlessness

e – Both Legs swelling

iv. **SIGNS**

a – Cyanosis

b – Polycythemia

c - Accessory muscles working

d - Pedal oedema

e - Raised JVP

f - Congestive Hepatomegaly

v. **ECG CHANGES**

a – Poor ‘R’ Wave progression in $V_1 – V_6$

b - ‘P’Pulmonale

c - QRS > + 90^0 in frontal plane

d - R/S < 1 in V_5 V_6

e - V_6 < 5 mm

MASTER CHART

S. No.	I.P. / O.P. No.	NAME	AGE	SEX	RISK FACTORS					SYMPTOMS					SIGNS						R/U	SPIROMETRY FINDINGS				DISEASE DURATION	ECG CHANGES						FEATURES OF RHF
					SMOKING		C.R.I.	ALLERGY	AIR POLLUTION	a	b	c	d	e	a	b	c	d	e	f		I	II	III	IV		a	b	c	d	e	NORMAL	
					ACTIVE in P / Y	PAS-SIVE																											
1	178838	R. Krishnasamy	43	M	-		-	+	-	+	-	-	-	-	-	-	-	-	-	-	U		+			11						+	
2	178846	M. Rengaraj	69	M	32.5		-	-	-	+	+	+	+	-	-	-	+	-	-	-	R			+		17						+	
3	178854	K. Duraisamy	70	M	37.5		+	-	-	+	+	+	+	-	-	-	-	-	-	-	U			+		20	+					-	
4	178862	R. Dhamodaran	58	M	20.5		-	-	-	+	+	+	+	-	-	-	-	-	-	-	R		+			12						+	
5	178870	S. Kannusamy	75	M	28.5		+	-	-	+	+	+	+	-	-	-	+	-	-	-	R			+		22	+					-	
6	178878	A. Nagammal	65	F			+	-	-	+	+	-	+	-	-	-	-	-	-	-	R			+		15						+	
7	178886	P. Arunachalam	67	M	24.5		-	-	-	+	+	+	+	-	-	-	-	-	-	-	R			+		16						+	
8	178892	A. Ameersulthan	55	M	22.5		-	-	-	+	+	+	+	-	-	-	+	-	-	-	U			+		14						+	
9	178900	M. Natesan	66	M	23.5		-	-	-	+	+	-	+	-	-	-	-	-	-	-	R			+		16						+	
10	178837	S. Rathinam	69	M	35		-	-	-	+	+	+	+	-	-	-	-	-	-	-	R			+		19						+	
11	178845	K. Nagarajan	55	M	25.5		-	-	+	+	+	+	+	-	-	-	-	-	-	-	R		+			15						+	
12	178653	A. Vaithiyanathan	67	M	27.5		-	-	-	+	+	+	+	-	-	-	+	-	-	-	R			+		17						+	
13	178761	V. Dhanapal	56	M	26.5		-	-	-	+	+	+	+	-	-	-	-	-	-	-	U		+			13						+	
14	178869	R. Asai thambi	68	M	28		-	-	-	+	+	-	+	-	-	-	-	-	-	-	R			+		18						+	
15	179677	C. Abdul Raghman	57	M	27.5		-	-	-	+	+	+	-	-	-	-	-	-	-	-	U		+			17						+	
16	178633	S. Vijaya	68	M			+	-	-	+	+	+	+	-	-	-	+	-	-	-	R			+		16						+	
17	179741	M. Arivalagan	61	M	22		-	+	-	+	-	+	-	-	-	-	-	-	-	-	R			+		13						+	
18	178849	K. Darman	51	M	21.5		-	-	-	+	+	-	-	-	-	-	-	-	-	-	R		+			12						+	
19	179957	C. Srinivasan	41	M	-		-	-	-	+	+	+	+	-	-	-	+	-	-	-	U			+		16						+	
20	178665	C. Jayakumar	52	M	22.5		-	-	-	+	+	+	+	-	-	-	-	-	-	-	R		+			14						+	
21	179773	L. Karuppusamy	77	M	39		+	-	-	+	+	-	+	+	+	-	+	+	+	+	R				+	25	+	+	+	+	+	-	+
22	178881	S. Selvaraj	63	M	23.5		-	-	-	+	+	+	+	-	-	-	-	-	-	-	U			+		18						+	
23	179987	I. Pandiyan	64	M	26		-	-	-	+	+	+	+	-	-	-	-	-	-	-	U			+		17						+	
24	178695	M. Kulanjinathan	71	M	29		+	-	-	+	+	+	+	-	-	+	+	-	-	-	R				+	19	+					-	
25	178732	L. Mohan Kumar	53	M	22.5		-	-	-	+	+	+	+	-	-	-	-	-	-	-	R		+			13	+					-	
26	178840	C. Rajammal	59	F		+	+	-	-	+	+	-	+	-	-	-	-	-	-	-	R			+		18						+	
27	178948	V. Raju Chettiyar	79	M	38.5		-	-	-	+	+	+	+	+	-	+	+	+	+	+	R				+	23	+	+	+	+	+	-	+
28	178656	A. Anvar Basha	65	M	26.5		-	-	-	+	+	+	+	-	-	-	-	-	-	-	R			+		20						+	
29	178764	K. Ramaiyan	42	M	22		+	-	-	+	+	-	+	-	-	-	+	-	-	-	U			+		6						+	
30	179872	R. Periyasamy	72	M	28		-	-	-	+	+	+	+	-	-	-	-	-	-	-	R			+		17	+					-	

S. No.	I.P. / O.P. No.	NAME	AGE	SEX	RISK FACTORS					SYMPTOMS					SIGNS						R/U	SPIROMETRY FINDINGS				DISEASE DURATION	ECG CHANGES						FEATURES OF RHF
					SMOKING		C.R.I.	ALLERGY	AIR POLLUTION	a	b	c	d	e	a	b	c	d	e	f		I	II	III	IV		a	b	c	d	e	NORMAL	
					ACTIVE in P / Y	PAS-SIVE																											
31	179785	M. Mohammed Safi	66	M	25		-	+	+	+	+	+	+	-	-	-	+	-	-	-	U			+		15						+	
32	178891	R. Appa Durai	77	M	25.5		+	-	-	+	+	+	+	-	-	-	-	-	-	-	R			+		16	+					-	
33	178999	S. Chinnakannu	47	M	26		-	-	-	+	+	+	+	-	-	-	-	-	-	-	U		+			12						+	
34	179636	X. Johnson	66	M	25.5		+	-	-	+	+	+	+	-	-	-	+	-	-	-	R			+		18	+					-	
35	178744	G.P. Ashokan	55	M	25		-	-	+	+	+	+	+	-	-	-	-	-	-	-	U			+		14						+	
36	179852	K. Shanthi	55	F			-	+	+	+	-	+	-	-	-	-	-	-	-	-	U	+				13						+	
37	178960	R. Sundar	46	M	-		-	-	+	+	+	+	+	-	-	-	+	-	-	-	R			+		17						+	
38	179668	S. Gopalan	68	M	28		-	-	-	+	+	+	+	-	-	-	-	-	-	-	U			+		16						+	
39	178776	M. Durairaj	35	M	-		-	+	-	+	+	+	+	-	-	-	-	-	-	-	R		+			12						+	
40	178884	S. Nagappan	63	M	20		-	-	-	+	+	-	-	-	-	-	+	-	-	-	R			+		12						+	
41	178990	U. Murugesan	72	M	34.5		-	-	-	+	+	+	+	+	+	-	+	+	+	+	R				+	21	+	+	+	-	-	-	+
42	179698	S. Mani	63	M	-		-	-	-	+	+	+	+	-	-	-	-	-	-	-	R			+		18						+	
43	178735	R. Venkatraman	62	M	35		+	-	-	+	+	+	+	-	-	-	+	-	-	-	R			+		15						+	
44	179845	P. Govindh	34	M	14		-	-	+	+	+	+	-	-	-	-	-	-	-	-	R	+				3						+	
45	179951	V. Kathamuthu	36	M	16		-	-	-	+	+	+	+	-	-	-	-	-	-	-	R		+			4						+	
46	178959	K. Lakshmi	73	F		+	+	-	-	+	+	-	+	+	+	-	+	+	+	+	R				+	21	+	+	+	+	+	-	+
47	179667	S. Arumugam	62	M	32.5		+	-	-	+	+	+	+	-	-	-	-	-	-	-	R		+			7						+	
48	179775	K. Thangavel	52	M	26		-	-	-	+	-	+	+	-	-	-	-	-	-	-	U		+			7						+	
49	178883	M. Sowndarrajan	66	M	25		-	-	-	+	+	+	+	-	-	-	+	-	-	-	R			+		17	+					-	
50	179989	R. Tamilarasi	53	F			-	-	+	+	+	+	+	-	-	-	-	-	-	-	R		+			11						+	
51	178695	Mohammed younis	52	M	24		-	-	-	+	+	+	+	-	-	-	+	-	-	-	R			+		12						+	
52	179734	P. Karupaiya	55	M	23		-	-	-	+	+	-	+	-	-	-	+	-	-	-	R			+		14						+	
53	179842	R. Rasu	74	M	35		+	-	+	+	+	+	+	-	-	-	+	-	-	-	U			+		25	+					-	
54	179950	M. Kolangi	76	M	30		+	-	-	+	+	-	+	+	+	-	+	+	+	+	R				+	24	+	+	+	-	-	-	+
55	179558	M. Ayyasamy	63	M	24		-	-	-	+	+	+	+	-	-	-	+	-	-	-	R			+		15						+	
56	179766	S. Kannan	55	M	27		-	-	-	+	+	-	+	-	-	-	-	-	-	-	U		+			16						+	
57	178874	M. Kandhasamy	56	M	26.5		+	-	-	+	+	-	+	-	-	-	-	-	-	-	R		+			19						+	
58	179982	R. Samikannu	64	M	23		-	-	-	+	+	+	+	-	-	-	+	-	-	-	R			+		18						+	
59	179688	P. Selvam	56	M	28		-	-	-	+	+	+	+	-	-	-	-	-	-	-	U		+			8						+	
60	179996	S. Raman	57	M	24.5		+	-	-	+	+	+	+	-	-	-	-	-	-	-	R		+			12						+	