WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
SJIF Impact Factor 8.084
Volume 12, Issue 18, 865-889. Research Article ISSN 2277– 7105

ASSESSMENT OF COPD SYMPTOMS AND RESPONSIVENESS OF THERAPY BY USING CLINICAL COPD QUESTIONNAIRE

Dr. Pitesh Gurjar*, Dr. Rajesh Sharma*, Dr. Rahul Gupta, Dr. Rajveer Singh Chopra, Mr. Ashwani Kumar

Pharm D. JNU Campus, Jagatpura Jaipur, Rajasthan India.

Article Received on
29 August 2023,

Revised on 19 Sept. 2023,
Accepted on 10 Oct. 2023

DOI: 10. 20959/wjpr202318-29947

***Corresponding Author**
Dr. Pitesh Gurjar &
Dr. Rajesh Sharma
Pharm D. JNU Campus,
Jagatpura Jaipur, Rajasthan
India.

1. INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a common lung disease in which airflow limitation that is not 100% reversible. This is preventable and manageable. Early diagnose phase by using spirometry and pulse oximeter. Chronic Obstructive Pulmonary disease severity can be assessed by pulmonary function test such as FVC1 (forced vital capacity) and FEV1 (forced expiratory volume).[1,2]

Stages of COPD

1. Mild FVC1 <0.7 and FEV >80%
2. Moderate FVC1 <0.7 and FEV >50%
3. Severe FVC1 <0.7 and FEV >30%

COPD is progressive disease in which decrease the rate of lung function.
COPD Not single disease in which include emphysema & bronchitis.[1,3]

Emphysema is a lung disease that affects the alveoli (tiny air sacs). This illness is more likely to occur in people who smoke or have chronic bronchitis. It can expand the lungs, causing damage to their walls and making breathing difficult.

Bronchitis Chronic inflammation of conduction airway. Patient is having a productive cough for at least 3 months for 2 consecutive years. Causes Smoking (cigarette), cystic fibrosis. Pathogenesis Chronic inflammation - Stimulation of submucosal mucus secreting gland increase secretion of mucus in bronchi, constriction of segmental bronchi and/or proximal bronchioles irreversible fibrosis. Bronchoconstriction decreased radius increased airways resistance. (5,6) Sometimes acute inflammation can be superimposed on top of a chronic inflammation. (6) Chronic obstructive pulmonary disease has mostly occurred person who

have smoker 85% (cigarette, bidi, pipe, cigar), cool mining worker, genetic factor, stone worker, long term air pollution & dust exposure they are risk factor that increase the chance of occurrence of COPD.[7,8]

COPD risk is increase in females who have cooking food on challah and chemin because this area is more air polluted. Some patient in their whole life untouched by COPD, while some are almost completely incapacitated.[8]

COPD increase respiratory symptoms such as dyspnoea, cough, phlegm production, cough and phlegm are also may be in respiratory infection. The main goal in COPD management is stop disease progression & minimise the symptom of dyspnoea & cough.

Smoking cessation and avoid exposure of pollutants is commonly management to stop and slow progression of disease.[9,10]

Oxygen therapy can reduce symptoms of hypoxia in particular patient who suffering with COPD.[11]

Bronchodilator are drug of choice for the patients who suffering from mild to moderate COPD, such as salbutamol and patients who suffering chronic symptoms, use long-acting bronchodilator or beta2 agonist or anticholinergic for good benefits. combination of long-acting bronchodilators is needed is necessary.[12,13]

EPIDEMIOLOGY

The smoking s primary cause of COPD and those how age more than 40 year. COPD is 3rd leading cause of death globally approx. 6% of total death, according to WHO. Prevalence increases with age. In 2015, the prevalence of COPD was 174 million and there were approximately 3.2 million deaths due to COPD worldwide. However, the prevalence is likely to be underestimated due to the underdiagnosis of COPD.[13,14]

PREVALENCE IN INDIA

Chronic Obstructive Pulmonary Diseases is the second most common disease that affect the lung followed by pulmonary tuberculosis.[2,3]

Higher chance of occur in males due to higher prevalence of smoking. COPD is mainly affects to middle aged and elderly people, less common in person how below the age 35

years. The random-effects pooled estimate for the spreading of COPD among the Indian population was 7.4% (95% CI: 5.0%–9.8%).[15,16]

There was becoming heterogeneity between the studies. Heterogeneity test showed I^2 value of 95.5% and $P < 0.001$.[17]

ETIOLOGY

COPD is caused by long term exposure or irritants particles or gases that damage lungs and small airways. Cigarette smoking is one of the most common causes of COPD followed by bidi, cigar, pipe, and some other type of tobacco smoking.[18,19] and other except tobacco smoking are exposure of polluted air, occupational exposure, airway hyperreactivity, asthma, chronic bronchitis, respiratory infection, genetic factor such as antitrypsin deficiency, age and sex, lung growth and development.[20]

RISK FACTORS

The risk factors for COPD include:

- **Smoking**

This is one of the most common risk factors. Up to 85% of people who have COPD smoker or used to smoke.

Long term cigarette smoking damage respiratory epithelial ciliary movement and also effect to function of alveoli their gas exchange and increase the number and size of mucus secreting glands.[22,23]

- **Occupational exposure**

Such as organic and inorganic dust, second hand smoke, air pollution, coal mine worker, stone worker, females who cooking food on fire stove, chemin and exposure of chemical fumes and dusts from the environment or workplace.[21,24]

- **Age**

The chance of occurrence of COPD with the age people are commonly affected who have age greater than 40 years.[24,25]

- **Genetics**

This includes alpha-1 antitrypsin deficiency (AATD) and glutathione S-transferase which is a genetic condition that increase risk of COPD.

Alpha1 Anti Trypsin (Alpha $_1$AT) 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 commonest deficient allele termed ZZ (Cor Pi^{ZZ} Phenotype) results from a single amino acid substitution 342Glu 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.

Alfa$_1$ AT deficiency accounts for 2% of observed cases of emphysema. Patients present with premature development of emphysema chronic bronchitis or bronchiectasis. The patient usually presents with cough and dyspnea in the fourth decade. Nearly 80% had a family history of lung disease with autosomal recessive inheritance.

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

Pathologically pan acinar emphysema predominates and radiographically changes are most marked in lower lobes. Tobacco smoking is an extremely important cofactor for development of disease in alpha$_1$ AT deficiency.[26,27]

- **Asthma**

People who suffering from asthma more risk of developing airflow obstruction that can cause COPD. Then people who don't have asthma, but asthma does not cause always COPD.[28,29]

- **Recurrent Respiratory Infections**

Recurrent acute respiratory infection can reduce the function or activity of alveolar macrophage. higher in patients with chronic bronchitis. Or history of childhood respiratory infection.[30,31]

- **Growth And Nutrition**

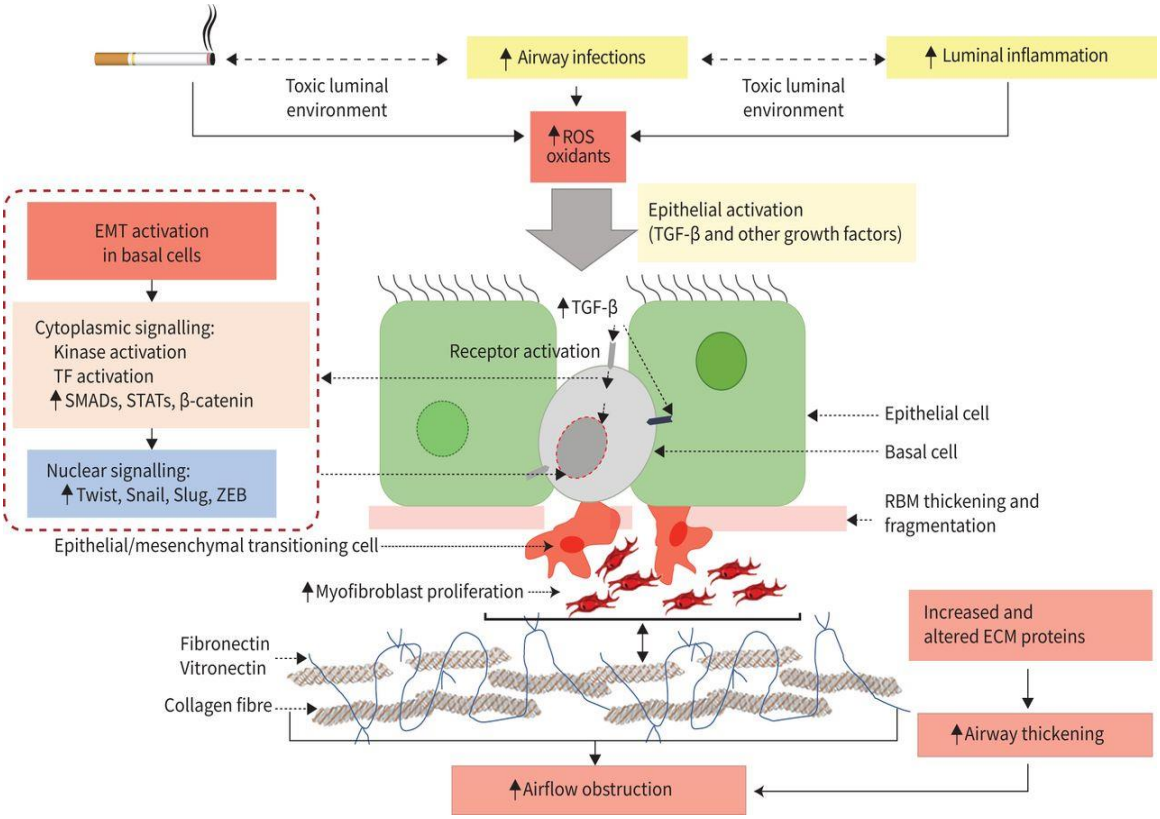
Restudies have shown that nutrition may affect both the growth and decline in ventilatory function. There is also some evidence that server viral pneumonia early in life may lead to chronic obstruction,particularly in small airways**.**[31,32]

PATHOPHYSIOLOGY

It affects not just the airways, but also the pulmonary vasculature and lung tissue. COPD inflammation is frequently described as neutrophils, however macrophages and CD8+ lymphocytes also play important roles. 19 – 21 Inflammatory cells emit a substance called TNF (tumour necrosis factor) is one of many chemical mediators. (TNF-), interleukin 8 (IL-8) and leukotriene B 4 are all examples of cytokines. (LTB 4) are play a important roles.[33,34] When unpleasant particles and gas are inhaled, the body's inflammatory cells and mediators are triggered. Exposure to ambient cigarette smoke is the most common causative factor, but other chronic inhalational exposures, as well as long-term tobacco smoke exposure, can cause similar inflammatory alterations. Cigarette smoke contains oxidants that react with and destroy numerous proteins and lipids, causing cell and tissue damage.[35,36] In addition, oxidants cause inflammation and worsen the protease-antiprotease imbalance in the lungs. Certain enzymes, such as neutrophil elastase, which destroys elastin, a significant component of the lung wall, may become more active or less active as a result of smoking. The inflammatory cells that predominate in COPD and asthma are very different, with neutrophils playing a major role in the latter, and eosinophils and mast cells in the former. Mediators of inflammation also differ between the two conditions, with LTB 4, IL-8, and TNF-α pre-dominating in COPD and LTD 4 and IL-4 dominating in asthma. There is frequently an inflammatory exudate present, which causes an increase in the number and size of goblet cells and mucus glands.[37,38] Ciliary motility is hindered, and mucus output is elevated. There is diffuse airway constriction, which is especially noticeable in smaller peripheral airways. The vascular changes of COPD include a thickening of pulmonary vessels and an increase in pulmonary pressures. In severe COPD, secondary pulmonary hypertension can cause right-sided heart failure. Mucus hypersecretion is present early in the disease and is associated with an increased number and size of mucus producing cells.[39,40]

At rest, thoracic hyperinflation is explained as an increase in the amount of air present in the lungs following exhalation. COPD medication, particularly bronchodilators, can help with airflow restriction.[41] This could explain why patients experience improvement despite just minor improvements in lung function with medication therapy. COPD is a condition in which many pathologic alterations in the lungs disrupt gas exchange and protective processes.[41,42] Dyspnoea and a persistent cough with sputum are two common COPD symptoms. Gas exchange problems cause hypoxemia and/or hypercapnia as the condition advances. In reaction to persistent hypoxemia, some COPD patients lose the capacity to raise the rate or

depth of their breathing. These changes in Pa O2 and Pa CO2 are gradual and take years to manifest.[43,44] As a result, the kidneys compensate by holding bicarbonate, and the pH is usually close to normal. The most prevalent cardiovascular consequence of COPD is pulmonary hypertension. Corpulmonale is characterised by right ventricular hypertrophy as a result of increased pulmonary vascular resistance. COPD patients have significant airway inflammation, but there is also evidence of systemic inflammation.[46,47]



CLINICAL PRESENTATION IN COPD

The patient's cough, sputum output, and dyspnea should all be examined, as well as the patient's history of tobacco smoke exposure and other COPD risk factors. Occupational exposures and the presence of genetic variables, such as AAT deficiency, should also be considered. A FEV:1FVC ratio of less than 70%, which shows airway blockage, is a characteristic of COPD. A bronchodilator challenge is used to assess the reversibility of airflow restriction.[48,49] COPD is characterized by a low peak expiratory flow. Spirometry is used to identify those who could benefit from pharmacotherapy to help them manage their chronic obstructive pulmonary disease. Spirometry can also be used to measure the severity of the condition, as well as the presence of complications and symptoms. Dyspnea is generally the most bothersome symptom for COPD patients. Dyspnea can affect exercise

performance and functional capacity, and it's often linked to depression and anxiety. A low BMI is a systemic complication of chronic COPD and is linked to an increased risk of death.[50,46]

CLINICAL SYMPTOMS

- Prolonged cough.
- Production of sputum.
- Dyspnea by Exposure to Risk Factors.
- Tobacco Exposure.
- α1 -Antitrypsin deficiency.
- Occupational hazards Physical Examination.
- Cyanosis of mucosal membranes.
- Hyperinflated lungs.
- Increased respiratory rate during rest.
- Tachypnea.
- Pursed lips during expiration.
- Use of accessory respiratory muscles Diagnostic Tests.
- Spirometry with reversibility testing.
- Chest Radiograph.
- ABG (not routine)

PHYSICAL EXAMINATION

- Fever.
- Wheezing.
- Decreased breathing sounds.

DIAGNOSTIC TEST

- Sputum for Gram stain and culture
- Radiograph of Chest to evaluates for new filtrates

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 teaches.
- Low flattened diaphragm: The border of the diaphragm in the midclavicular line below the seventh rib.
- Height of patient's lung being greater than 29.9 cm.
- An obtuse costophrenic angle.
- Reduction in size and number of pulmonary vessels particularly inperiphery 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.

PROGNOSIS

If COPD worsens, it becomes a lethal disease, thus advanced directives and end-of-life care options should be considered. The reduction in pulmonary function follows a straight line. The faster the rate of decrease, the worse the FEV 1 was at the time of diagnosis. The rate of decrease in blood gases has not been proved to be a good indicator of illness development. The extent of impairment in the FEV 1 is closely associated with the survival rate of COPD patients. Pulmonary function tests that rapidly drop indicate a dismal prognosis. While ABG values are useful, they lack the predictive value of pulmonary function testing.[31-39]

TREATMENT

General Approach To Treatment

Clinicians should assess and monitor the condition, avoid or reduce exposure to risk factors, stop progression of the disease and reduce the symptoms. These components can be addressed through a variety of nonpharmacologic and pharmacologic approaches, according to the American Journal of Internal Medicine (JIM).[42-44]

NON-PHARMACOLOGICAL TREATMENT

COPD's natural course is respiratory failure, and COPD's natural course is respiratory failure. Clinicians should discuss end-of-life decisions and advanced directives with patients and their families in advance. COPD patients should be educated about their disease and techniques for slowing its course and avoiding complications.

Smoking cessation.[22-27]

Avoid exposure of dust and air pollution.

Oxygen therapy.

Chest physiotherapy.

Vaccination – Pneumococcal vaccine

PHARMACOLOGICAL TREATMENT

There is currently no drug available for the treatment of COPD that has been proved to slow the progression of lung function decrease or to extend life. Chronic treatment with long-acting inhaled beta-agonists, inhaled corticosteroids, or a combination of the two appears to slow the rate of spirometry deterioration. Controlling symptoms (including dyspnea), reducing exacerbations, and improving exercise tolerance and health status are the main goals of medication.[17-18] Early in the course of disease, there is insufficient evidence to support the use of more aggressive medication. Patients respond differently to current medicines; therefore, treatment should be tailored to the person. Inhaled medicines are usually used in COPD pharmacotherapy. There are several delivery devices available (e.g., dry powder inhalers, nebulizers, and ancillary devices such as holding chambers). Common comorbidities in COPD patients can have a substantial impact on the patient's ability to use them. Bronchodilators increase exercise tolerance by relaxing the bronchial smooth, improving lung emptying, reducing thoracic hyperinflation at rest and during activity. Based on the severity of the condition, a stepwise strategy to COPD care has been recommended.[4-9]

BRONCHIODIALATOR

Bronchodilators work by relaxing the smooth muscle of the airway, allowing more air to flow through. Although inhalation therapy is often preferable, there is no obvious benefit to one drug or class over another. In comparison to other groups, COPD patients may find it more challenging to use inhalation devices properly.[2-6]

Short acting beta 2 agonist – albuterol (salbutamol), levalbuterol, and pirbuterol

Short-Acting Anticholinergics – Ipratropium

Long-Acting Inhaled β2-Agonists (LABAs) - salmeterol, formoterol, and arformoterol

Long-Acting Anticholinergics – tiotropium bromide

Combination Anticholinergics and β-Agonists - albuterol and ipratropium

Methylxanthines - Theophylline aminophylline and etophylline

Glucocorticosteriods – Budesonide, methylprednisolone, prednisolone dexamethasone, hydrocortisone and betamethasone.

Antimicrobial Agents – Amoxycillin, ofloxacin, azithromycin and ceftriaxone

2. 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 maycause heart failure even on otherwise 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 2003 Thys van der Molen** concluded that "It appears The CCQ is a self-administered questionnaire specially developed to measure clinical control in patients with COPD. Data support the validity, reliability and responsiveness of this short and easy to administer questionnaire."
- **In 2004 B.R. CELLI** This summary provides a high-level understanding of the complete document, which is easily accessible online (www.copd-ats-ers, www.ersnet.org and www.thoracic.org). This summary makes no mention of the patient document, which cannot be provided in a traditional fashion due to its inherent qualities.
- **In 2008 john F.** COPD will continue to be a major healthcare issue for many years to come. The importance of focusing on smoking cessation will have a significant impact on disease progression. Translation of a more fundamental understanding of the pathophysiologic mechanisms involved into disease-modifying therapies will be required for advancements in treatment.
- **In 2012 Agnieszka Lewko** in OVID, more than 600 manuscripts were retrieved, the majority of which were unrelated or concerned with skeletal and respiratory muscle fatigue. After evaluating abstracts and looking for duplication, 33 publications were found; data from Cochrane reviews and conferences relevant to the subject were extracted and examined.

- **In 2013 Thys van der molen** Using standardized and trustworthy questionnaires like the CCQ, CAT, and MRC to assess the impact of COPD on a regular basis provides a more realistic picture of the disease's burden. The use of these questionnaires in ordinary clinical practice is an effective technique for physicians to gain a better understanding of their patients' health-related quality of life.
- **In late 2013** Based on the data collected, we could outline the profile of patients with COPD, showing characteristics of an elderly population, with multiple comorbidities, suggesting a health-related quality of life lower than expected.
- **In 2014 HAMMAD QURESHI** Exacerbations of COPD are frequently caused by airway infection and are a leading cause of morbidity, health impairment, and mortality. Despite the availability of numerous pharmaceutical and nonpharmacological therapies to avoid exacerbations, the degree of reduction in exacerbation frequency remains limited.
- **In 2018 Christian Viniol** COPD is the third leading cause of death worldwide. Smoking cessation is key to prevent further COPD exacerbations. Influenza and pneumococcal vaccination are recommended. Treatment of hypoxemia and hypercapnia reduce the rate of COPD exacerbations.
- **In 2020 John R. hurst** COPD exacerbations represent a significant clinical problem, with deleterious effects on many aspects of patient health status, such as lung function, quality of life, comorbidities, and mortality. Furthermore, patients may take several weeks to recover following a COPD exacerbation. Improving physician and patient recognition, and increasing awareness of the impact of COPD exacerbations, are key to minimizing detrimental effects. Patient education initiatives are needed to increase their understanding and reporting of COPD exacerbations, allowing swift identification and treatment.

3. AIM AND OBJECTIVE OF THE STUDY

AIM OF THE STUDY

Assessment of COPD symptoms and responsiveness of therapy by using clinical COPD questionnaire.

OBJECTIVES OF THE STUDY

- To improve the patient quality of life.
- To assess severity of dyspnea.

4. MATERIALS AND METHODS

This study was conducted at Department of Respiratory Medicine and General Medicine, JNU Hospital &Medical College, Jaipur, 2022

TOTAL NUMBER OF PATIENTS IN THIS STUDY

Number of patients in this study is 63 cases.

INCLUSION CRITERIA

The cases in this study have following characters.

- Patients above the age of 18.
- Both male and female patients.
- Patients willing to fill the consent form.

EXCLUSION CRITERIA

- Patients with co-morbid conditions such as Hypertension, chronic kidney diseases, etc.
- Pregnant and breastfeeding women
- Patients not willing to give consent.

STUDY DESIGN

Prospective Observational Single cantered

TIME FRAME

Six (6) Month

5. RESULTS

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

Table 1: Age Distribution.

Age in Years	Male	Percentage	Female	Percentage	Total	Percentage
36-45	5	7.93%	1	1.58%	6	9.52%
46-55	18	28.57%	4	6.34%	22	34.92%
56-65	16	25.39%	2	3.17%	18	28.57%
66-75	12	19.04%	0	0%	12	19.04%
>76	5	7.93%	0	0%	5	7.93%
Total	**54**	**88.88**	**6**	**11.09**	**63**	**100**

From the above table it is observed that the majority of cases among males were between 46–55 years of age constituting 28.57%.

Followed by age group 56-65. and the minimum number of cases were in the age group of 36 – 45 constituting 7.93 % and followed by age group > 76.

Among females the majority of cases were in the age group of 46– 55 years constituting 6.34% and followed by age group 56-65. Minimum case was observed in the age group of >66.

Both sexes put together the maximum cases were in the age groupof 46-55 years constituting 34.92% of total cases and followed by age group 56-65. minimum caseswere observed in the age group of > 76 years which constituted 7.93%of total cases.

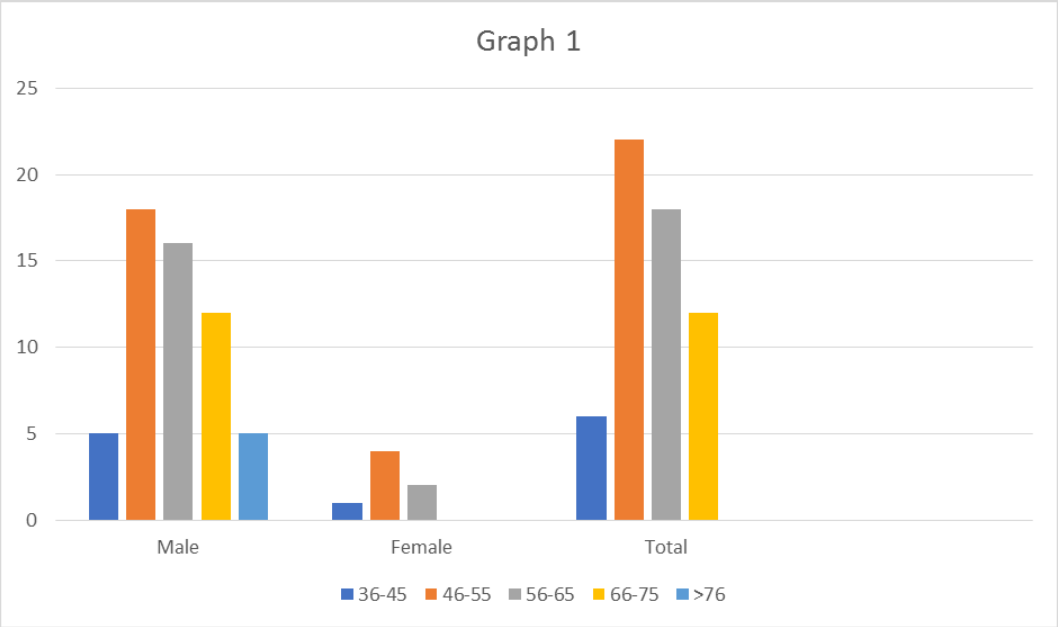


Table 2: Severity of Dyspnea According to S.T. Molen Copd Questionnaire.

Severity	No of Patients	Male	Female	Percentage
Very Mild (0-15)	2	1	1	3.17
Mild (16-30)	24	21	3	38.09
Moderate (31-45)	29	26	3	46.03
Severe (46-60)	8	8	0	12.65

From the above table it is observed that the 46.03 % of cases of dyspnea according to S.T. molen COPD questionnaire suffering moderate stage. Followed by 38.09 % patients are suffering mild stage of dyspnea.

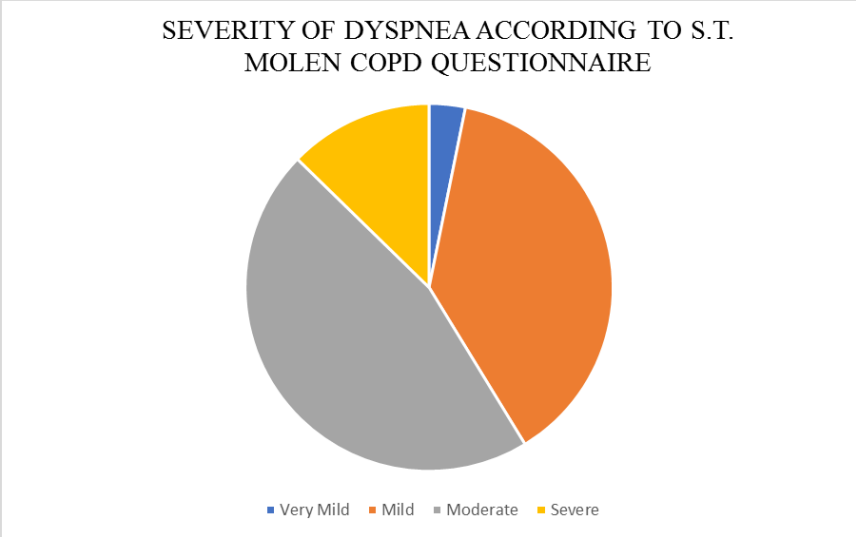


Table 3: Occupational Distribution.

Occupation	No. of patient	Percentage
Farmer	26	41.26
Stone & Coal mine worker	17	26.98
Shopkeeper	7	11.11
House Wife	7	11.11
laborer	6	9.52

From the above table, it is observed that the majority of patients according to Occupational distribution 14.26% of farmer then followed by stone & coal mine worker 26.98%.

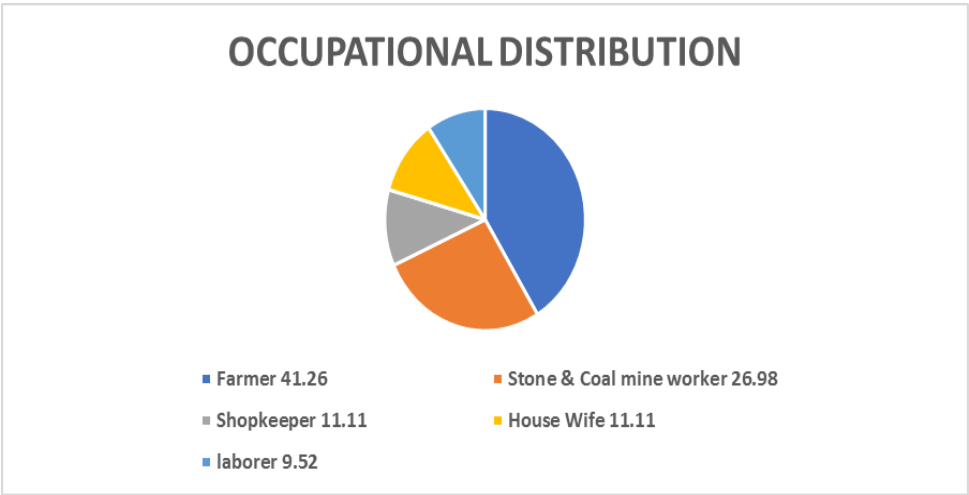


Table 4: Patients Condition.

Condition	No of patients	Percentage
Good	51	80.95
Stable	4	6.3
Poor	8	12.69

In above given table patient condition were show good relief from therapy 80.95% and stable patient is 6.3% & poor improvement by therapy patient were show 12.69%.

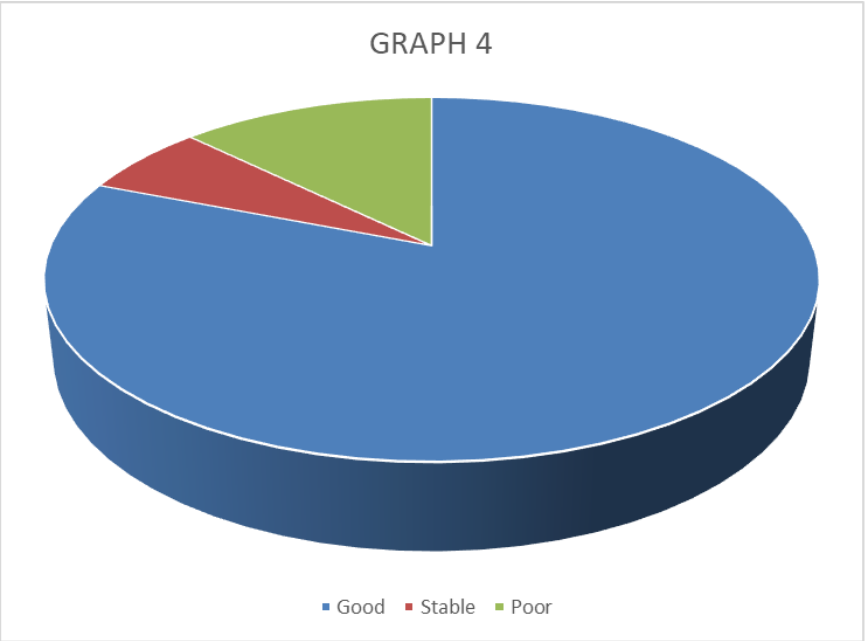


Table 5: Sex Distribution.

SEX	No. of cases	Percentage
Male	56	88.88%
Female	7	11.11%
Total	**63**	**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.

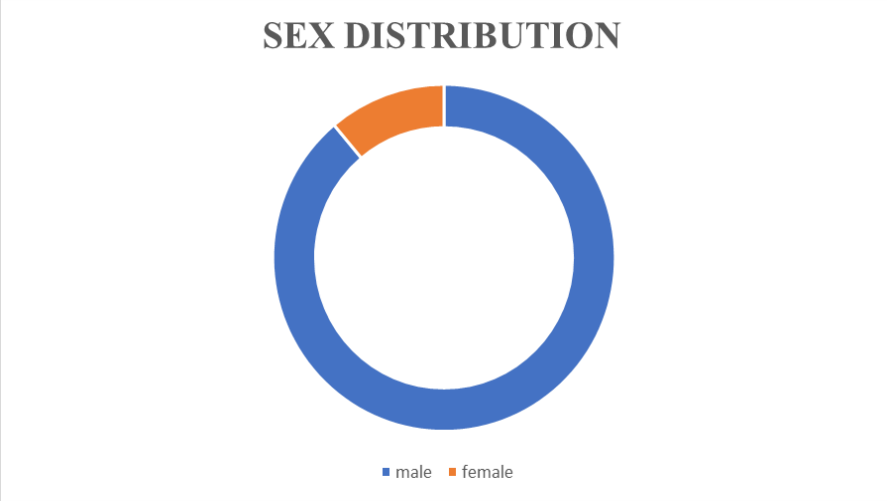
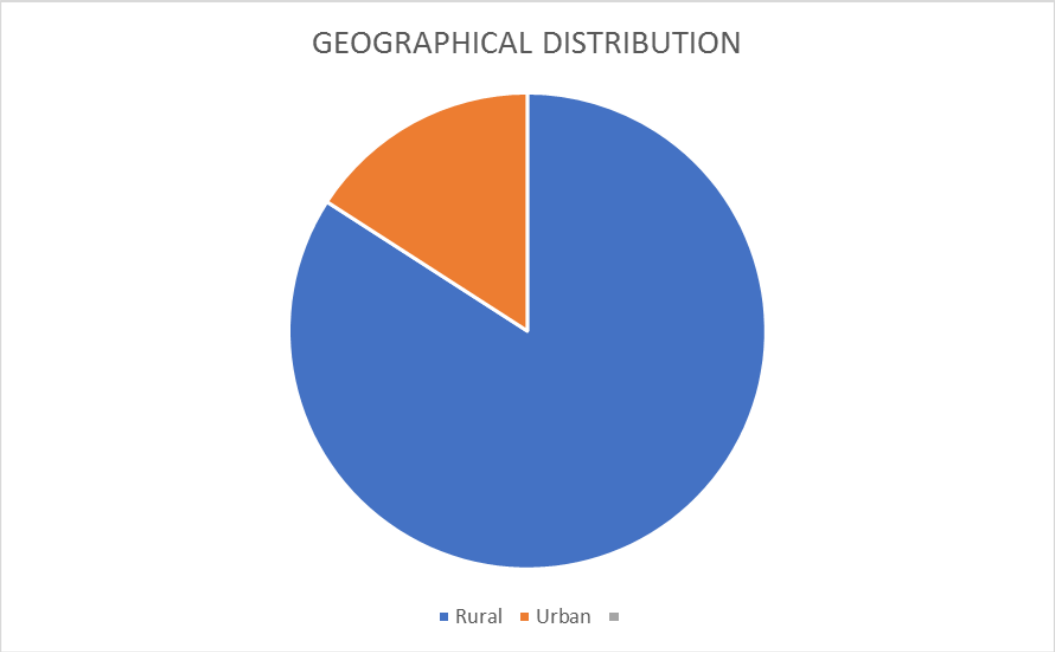


Table 6: Geographical Distribution.

Area	No. of cases	Male	Female	Percentage
Rural	53	46	7	84.12%
Urban	10	10	0	15.88%
Total	63	56	7	100%

From the above table, it is observed that the majority of the patients 84.12% from the rural area and 15.88% patients from urban area in this present study



RISK FACTORS

Table – 7.

Risk factor		Male	%	Female	%	Total	Percentage
Smoking	Active	40	63.5%	1	1.6	41	90.5%
	Passive	16	25.4%	0	0%	16	
Pollution exposure		17	26.9%	7	11.11%	24	38.1%

From the above table it was noted that major risk factor for COPDis smoking in males constituted 88.9% in males and 90.5% of total cases.

Pollution exposure constituted 38.1% of risk factor.

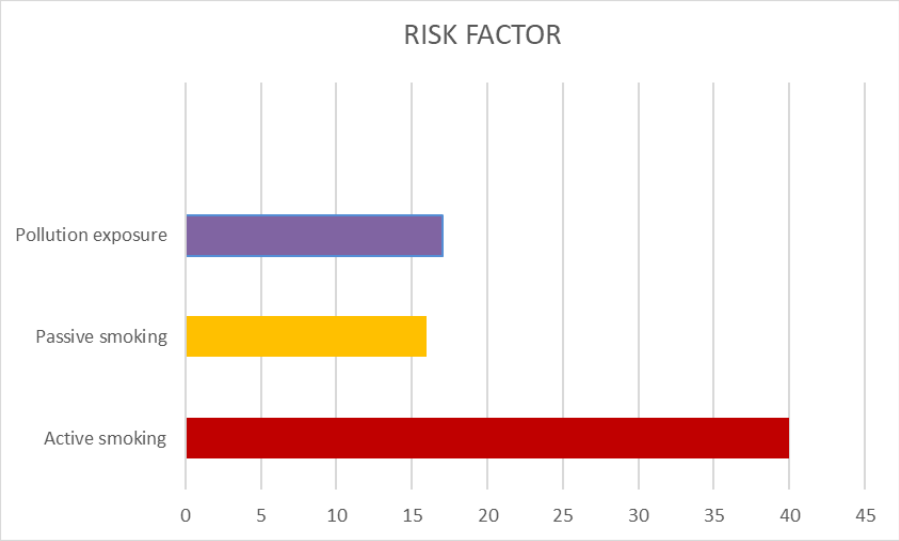


Table 8: Intensity of Smoking.

Duration of smoking (year)	No. of patients	Percentage
<20	23	36.5%
21 – 30	26	41.26%
>31	8	12.69%
Non-Smoker	6	9.52%

From the above table it can be observed that the majority of patients had more than 21-30 pack years of smoking which constituted 41.26%of total cases followed by <20 pack year.

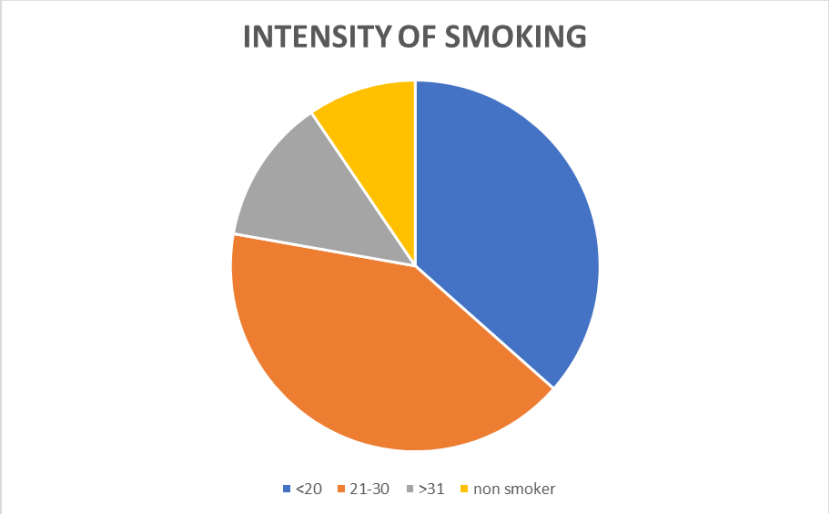


Table 9: Distribution of Copd Symptoms.

Symptoms	No of patients	Percentage
Cough	60	95.23%
Cough with expectoration	51	80.95%
Wheeze	16	25.39%
Breathlessness	59	93.65%

From the given table it is observed that majority of the patients in this study had cough. Cough was the major symptom constituted 95.23% in this study.

Cough with expectoration of sputum was present in 80.95% of cases.

Breathlessness which constituted 93.65% of cases.

Wheeze which constituted 25.39% of cases.

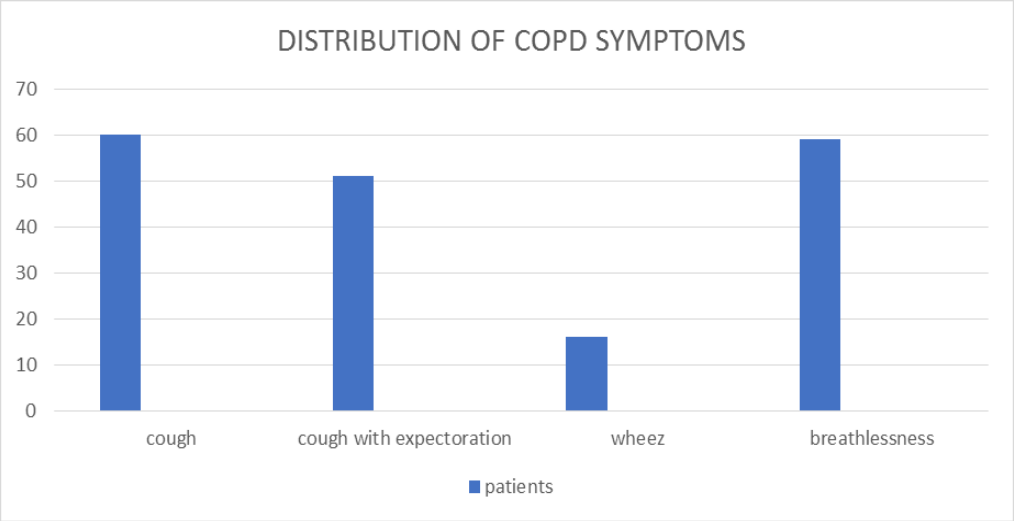


Table 10: Severity of Dyspnea At Addmission By Using Pulse Oximeter Device.

SEVERITY	Total	Male	Female	Percentage
Normal 98-100	0	0		0
96-97 Mild	12	9	3	19.04
91-95 Moderate	49	45	4	77.77
<90 Severe	2	2	0	3.17

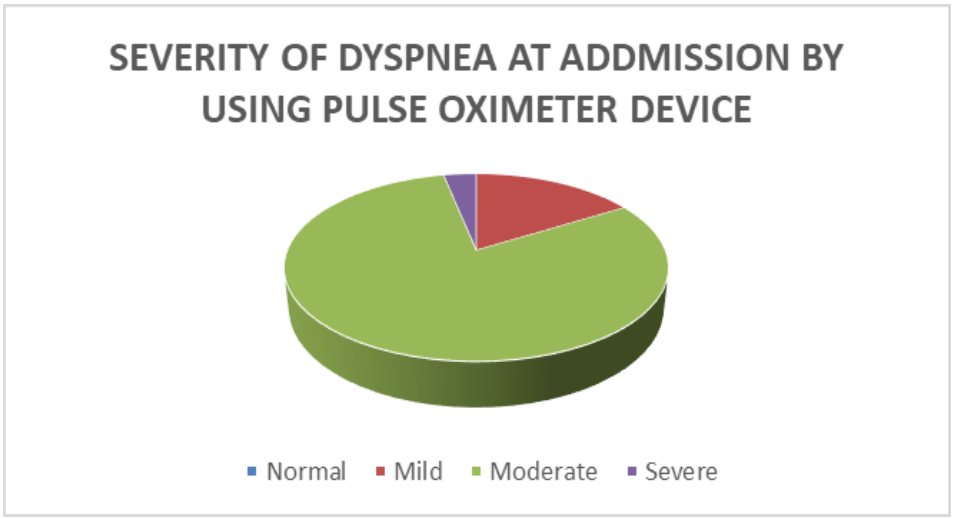


Table 11: Severity of Dyspnea At Discharge By Using Pulse Oximeter.

SEVERITY	Total	Male	Female	Percentage
Normal 98-100	52	45	7	82.53
96-97 Mild	5	5	0	7.93
91-95 Moderate	6	6	0	9.52
<90 Severe	0	0	0	0

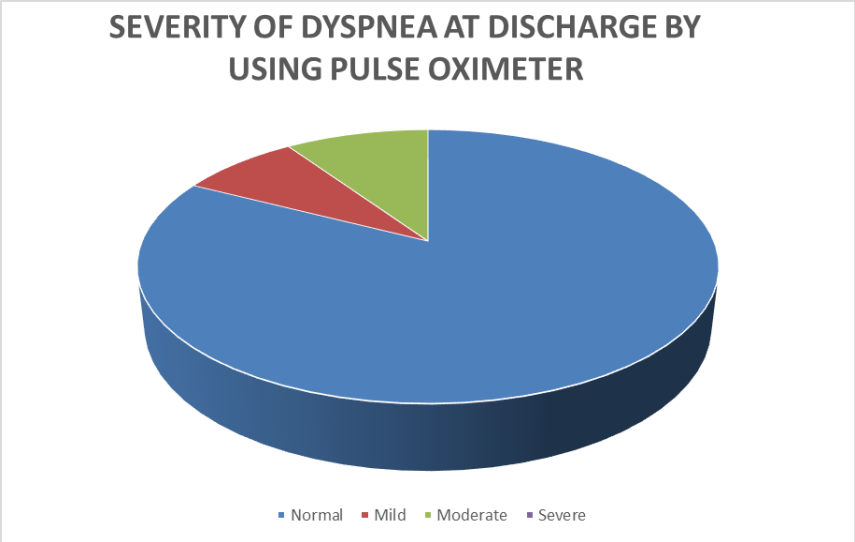


Table 12: Distribution Accordind To Hb% Report.

Severity of Hb	No of Patients	Percentage
Normal	33	52.38
Mild	15	23.8
Moderate	12	19.04
Severe	3	4.76

From the above table it is observed that severity of Hb% 52.38% patients is normal followed by mild no of patients are 23.8%.

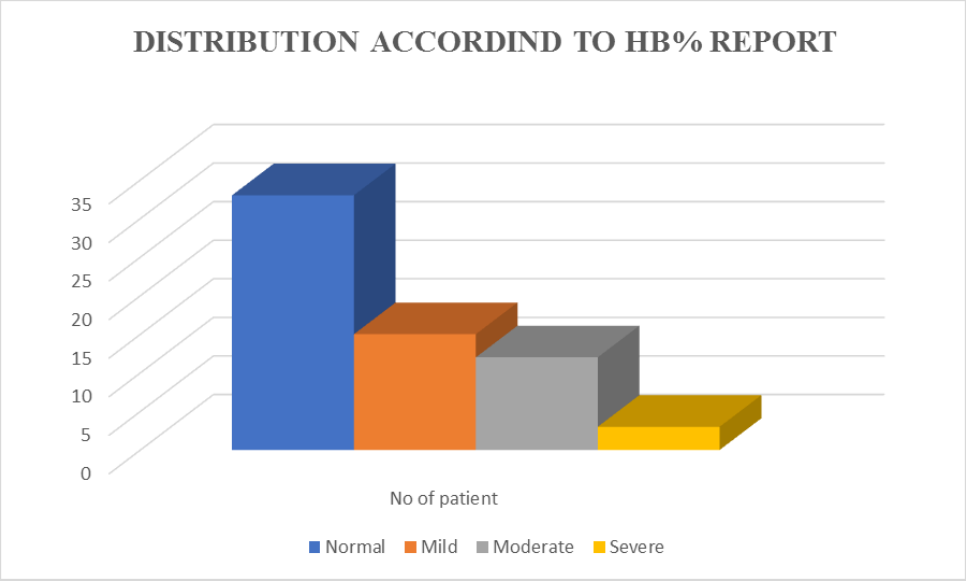


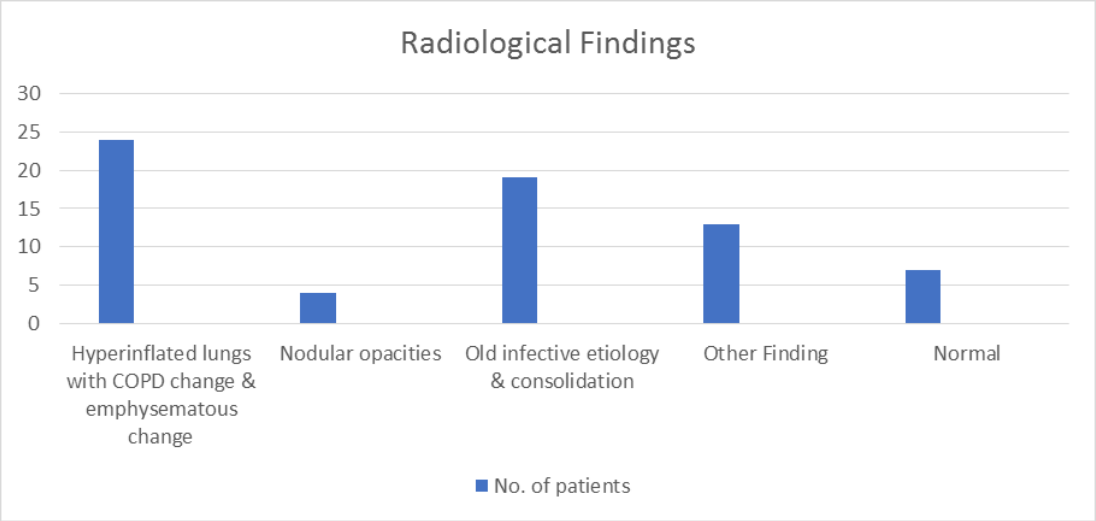
Table 13: Distribution of Radiological Findings.

HRCT Chest	No. of patients	Percentage
Hyperinflated lungs with COPD change & emphysematous change	24	38.19
Nodular opacities	4	6.3
Old infective etiology & consolidation	19	30.15
Other Finding	13	20.63
Normal	7	11.11

From the study it is observed that Hyperinflated lungs with COPD change & emphysematous change was the major radiological feature present in 38.19% ofcases.

30.15% of cases were shown Old infective etiology & consolidation.

Nodular opacities 6.3% of total case. 11.11% of the patients in this radiological study had normal.



6. DISCUSSION

In our study maximum number of cases in the age group between 46-55 years.

In this study, it was noted the incidence of COPD was higher in male than females with male to female ratio of 8.8:1.2. Male cases accounted for 88.88% of the total cases in this study. This finding also coincides with the following studies.

In our study it is observed that 85% of patients were from rural areas and 15% were from urban areas. In spite of heavy Air pollution, the urban areas contributed only15% of the cases in this study. Among the various risk factors in Smoking is the major risk factor is 90% of the causative risk factor in this present study. Most of the patients smoking between 21-30 years. Pollution exposure which constituted 38% was the second major risk factor

observed in this study.

In this study cough and cough with expectoration of sputum was observed in 95% and 80.95% of cases respectively. Dyspnoea was observed in 100% cases.

Dyspnoea observed most of patients 77% in moderate stage.

In this present study most common radiological finding in chest X-ray & HRCT Chest was Hyperinflated lungs with COPD change & emphysematous change which constituted 38.19 % of cases followed by old infective ethology and consolidation constituted 30.15%. Normal X-ray chest was observed in 11.1% of cases.

Severity of dyspnea was observed at the time of admission most patients are suffered from Moderate stage which constituted 77.7% followed by Mild stage which constituted 19%.

SUMMARY

In this study on Chronic Obstructive Pulmonary Diseases thefollowing facts were observed. COPD is the disease of aged as evidenced by majority of patientsin the present study were belong to the age group of 46 – 65 years.

COPD has male predominance as evidenced by 8.8:1.2 ratio of Maleto 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 followed by Dyspnea was the major clinical symptom observed in this study. Most number of cases had Moderate airway obstruction which was manageable & preventable. Hyperinflated lungs with COPD change & emphysematous change were the most common chest x-ray finding observed in this study.

7. CONCLUSION

1. Chronic Obstructive Pulmonary Diseases is a preventable & treatable disease. Smoking is the major risk factor for Chronic Obstructive Pulmonary Diseases. Pulse oximeter is mandatory to diagnose and assess the severity of PaO2 & Chronic Obstructive Pulmonary Diseases, was the singlemost important parameter.
2. Severity of Chronic Obstructive Pulmonary Diseases has direct relation with incidence of PaO2 changes in Chronic Obstructive Pulmonary Diseases.S T Molen COPD

Questionnaire help to assessment Shortness of breath at rest & while performing physical activity and cough, cough with expectoration. S T Molen COPD Questionnaire help to assessment Strenuous physical activity, Moderate physical activity, Daily activity at home and social activities.

9. BIBLIOGRAPHY

1. Mapel DW, Hurley JS, Roblin D, et al. Survival of COPD patients using inhaled corticosteroids and long-acting beta agonists. Respir Med., 2006; 100: 595–609.
2. Calverley PMA, Anderson JA, Celli B, et al. Salmeterol and fluticasone propionate and survival in chronic obstructive pulmonary disease. N Engl J Med., 2007; 356: 775–789.
3. Drummond MB, Dasenbrook EC, Pitz MW, Murphy DJ, Fan E. Inhaled corticosteroids in patients with stable chronic obstructive pulmonary disease: A systematic review and meta-analysis. JAMA, 2008; 300: 2407–2416.
4. Singh S, Amin AV, Loke YK. Long-term use of inhaled corticosteroids and the risk of pneumonia in chronic obstructive pulmonary disease: A meta-analysis. Arch Intern Med., 2009; 169: 219–229.
5. Lipworth BJ. Systemic adverse effects of inhaled corticosteroid therapy: A systematic review and meta-analysis. Arch Intern Med, 1999; 159: 941–955.
6. Global Initiative for Chronic Obstructive Lung Disease. Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Pulmonary Disease. NHLBI/WHO workshop report. Bethesda, MD: National Heart, Lung, and Blood Institute; April 2001. Updated November 2009, http://www.goldcopd.com/.
7. Celli BR, MacNee W, ATS/ERS Task Force. Standards for the diagnosis and treatment of patients with COPD: A summary of the ATS/ERS position paper. Eur Respir J., 2004; 23: 932–946. Full report accessible at http://www.thoracic.org .
8. Rabe KF. Guidelines for chronic obstructive pulmonary disease treatment and issues of implementation. Proc Am Thorac Soc, 2006; 3: 641–644.
9. Specialized Centers of Clinically Oriented Research (SCCOR). In: Chronic Obstructive Pulmonary Disease (COPD). RFA Number: REA-HL-05-008. Bethesda, MD, August 2004.
10. National Center for Health Statistics. National Health Interview Survey. Hyattsville, MD, U.S. Department of Health and Human Services, CDC, NCHS; 2001, www.cdc.gov/nchs/nhis.htm.

11. Mannino DM, Homa DM, Akinbami LJ, et al. Chronic obstructive pulmonary disease surveillance—United States, 1971–2000. Surveillance Summaries, August 2, 2002. MMWR, 2002; 51: 1–16.
12. Wilt TJ, Niewoehner D, Kim C, et al. Use of spirometry for case finding, diagnosis, and management of chronic obstructive pulmonary disease (COPD). Evidence Report #121. Agency for Healthcare Research and Quality. AHRQ Publication 05-E017–1, August 2005.
13. Brown DW, Croft JB, Greenlund KJ, Giles WH. Deaths from Chronic Obstructive Pulmonary Disease—United States, 2000–2005. MMWR, 2008; 57: 1229–1232.
14. Chronic Obstructive Pulmonary Disease: Data Fact Sheet. U.S. Department of Health and Human Services, National Institutes of Health. NHLBI, NIH Publication 03-5529. Bethesda, MD; March 2003.
15. Holguin F, Folch E, Redd SC, Mannino DM. Comorbidity and mortality in COPD-related Hospitalizations in the United States, 1979–2001. Chest, October 2005; 2005–2011.
16. Chronic obstructive pulmonary disease (COPD) Fact sheet. American Lung Association. Feb 2010. Washington DC, available at www.lun gusa.org/lung-disease/COPD/resources/fact-figures/COPD-Fact-Sheet. html. Accessed July 31, 2010.
17. National Institutes of Health. NHLBI Morbidity & Mortality: 2004 Chart Book on Cardiovascular, Lung, and Blood Diseases. U.S. Department of Health Services; May 2004, http://www.nhlbi.nih.gov/ resources/docs/cht-book.htm.
18. Jansson SA, Andersson F, Borg S, Ericsson A, Jonsson E, Lundback B. Costs of COPD in Sweden according to disease severity. Chest, 2002; 122: 1994–2002.
19. The Health Consequences of Smoking: A Report of the Surgeon General. Washington, DC, 2004.
20. Sandford AJ, Silverman EK. Chronic obstructive pulmonary disease. 1: Susceptibility factors for COPD the genotype-environment interaction. Thorax, 2002; 57: 736–741.
21. Carrel RW, Lomas DA. Alpha-1 antitrypsin deficiency: A model for conformational diseases. N Engl J Med., 2002; 346: 45–53.
22. Hunninghake GM, Cho MH, Tesfaigzi Y, et al. MMP12, Lung Function, and COPD in High-Risk Populations. N Engl J Med., 2009; 361: 2599–2608.
23. Barnes PJ. Chronic obstructive pulmonary disease. New Engl J Med., 2000; 343: 269–280.
24. Stockley RA. Neutrophils and the pathogenesis of COPD. Chest., 2002; 121: 151–155S.

25. Hogg JC. Pathophysiology of airflow limitation in chronic obstructive pulmonary disease. Lancet, 2004; 364: 709–721.
26. Hill AT, Bayley D, Stockely RA. The interrelationship of sputum inflammatory markers in patients with chronic bronchitis. Am J Respir Crit Care Med., 1999; 160: 893–898.
27. Ries AL. Impact of chronic obstructive pulmonary disease on quality of life: The role of dyspnea. Am J Med., 2006; 119: S12-S20.
28. MacNee W. Pathophysiology of cor pulmonale in chronic obstructive pulmonary disease. Part 2. Am J Respir Crit Care Med., 1994; 150: 1158–1168.
29. Sin DD, Man SF. Why are patients with chronic obstructive pulmonary disease at increased risk of cardiovascular disease? The potential role of systemic inflammation in chronic obstructive pulmonary disease. Circulation, 2003; 107: 1514–1519.
30. Casaburi R, ZuWallack R. Pulmonary Rehabilitation for Management of Chronic Obstructive Pulmonary Disease. N Engl J Med., 2009; 360: 1329–1335.
31. Rodriguez-Roisin R. Toward a consensus definition for COPD exacerbations. Chest, 2000; 117(2): 398S-401S.
32. Landbo C, Prescott E, Lange P, Vestbo J, Almdal TP. Prognostic value of nutritional status in chronic obstructive pulmonary disease. Am J Respir Crit Care Med., 1999; 160: 1856–1861.
33. Ferris BG. Epidemiology standardization project (American Thoracic Society). Am Rev Respir Dis, 1978; 118: 1–120.
34. Anzueto A. Clinical course of chronic obstructive pulmonary disease: Review of therapeutic interventions. Am J Med., 2006; 119: S46-S53.
35. Mannino DM, Doherty DE, Buist S. Global initiative on obstructive lung disease (GOLD) classification of lung disease and mortality: Findings from the atherosclerosis risk in communities (ARIC) study. Respir Med., 2006; 100: 115–122.
36. Celli BR. The importance of spirometry in COPD and asthma. Chest, 2000; 117: 15S-19S.
37. Andersson F, Borg S, Janson S-A, et al. The costs of exacerbations in chronic obstructive pulmonary disease (COPD). Respir Med., 2002; 96: 700–708.
38. Anthonisen NR, Manfreda J, Warren CPW, et al. Antibiotic therapy in exacerbations of chronic obstructive pulmonary disease. Ann Intern Med., 1987; 106: 196–204.
39. Groenewegen KH, Schols AMW, Wouters EFM. Mortality and mortality-related factors after hospitalization for acute exacerbation of COPD. Chest, 2003; 124: 459–467.

40. Bach PB, Brown C, Gelfand SE, McCrory DC. Management of acute exacerbations of chronic obstructive pulmonary disease: A summary and appraisal of published evidence. Ann Intern Med., 2001; 134: 600–620.
41. Almagro P, Calbo E, Ochoa de Echaguen A, et al. Mortality after hospitalization for COPD. Chest, 2002; 121: 1441–1448.
42. Connors AF, Dawson NV, Thomas C, et al. Outcomes following acute exacerbation of severe chronic obstructive disease. Am J Respir Crit Care Med., 1996; 154: 959–967.
43. Heffner JE, Fahy B, Hilling L, Barbieri C. Outcomes of advanced directive education of pulmonary rehabilitation patients. Am J Respir Crit Care Med., 1997; 155: 1055–1059.
44. Parrott S, Godfrey C, Raw M, et al. Guidance for commissioners on the cost-effectiveness of smoking cessation interventions. Health International Authority. Thorax, 1998; 53(5): S1-S38.
45. Anthonisen NR, Connett JE, Kiley JP, et al. Effects of smoking intervention and the use of an inhaled anticholinergic bronchodilator on the rate of decline in FEV 1 : The Lung Health Study. JAMA, 1994; 272: 1497–1505.
46. Dolovich MB, Ahrens RC, Hess DR, et al. Device selection and outcomes of aerosol therapy: Evidence-based guidelines. Chest., 2005; 127: 335–371.
47. Truitt T, Witko J, Halpern M. Levalbuterol compared to racemic albuterol: efficacy and outcomes in patients hospitalized with COPD or asthma. Chest., 2003; 123: 128–135.
48. Asmus MJ, Hendeles L. Levalbuterol nebulizer solution: Is it worth five times the cost of albuterol? Pharmacotherapy, 2000; 20: 123–129.
49. Datta D, Vitale A, Lahiri B, ZuWallack R. An evaluation of nebulized levalbuterol in stable COPD. Chest., 2003; 124: 844–849.
50. O'Donnel DE, Lam M, Webb KA. Measurement of symptoms, lung hyperinflation, and endurance during exercise in chronic obstructive pulmonary disease. Am J Respir Crit Care Med., 1998; 158: 1557–1565.