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A Study on Comorbid Conditions and Its Therapeutic Management in Chronic Obstructive Pulmonary Disease

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ABSTRACT:

BACKGROUND: Chronic obstructive pulmonary disease (COPD) is the third leading cause of death worldwide. It is a chronic inflammatory condition in the lungs marked by a lung airflow obstruction that interferes with normal respiration and is not fully reversible. The effect of risk factors like smoking, severe restriction of the airflow, bacterial and viral infections, and comorbid conditions all contribute to COPD.

MATERIALS AND METHODS: This is a prospective observational study conducted in MEDICOVER hospital Hyderabad. Cases of patients who were diagnosed with COPD were collected from inpatients of the Pulmonology department in the duration of 6 months.

RESULTS: A total of 100 cases of COPD were observed. It was predominant in men as compared to women and was found to be more prevalent in patients of age groups 60. Most common comorbidity was Hypertension (25%). Smoking and drinking alcohol were associated with the worsening of the condition. The drugs used for the treatment were Sympathomimetics (7%), Corticosteroids (24%), Bronchodilators (14%), Anticholinergics (15%), Methylxanthines (7%), Antimicrobial Therapy (33%).

CONCLUSION: COPD is one of the most common diseases. The main symptoms include cough, Exacerbations and Shortness of breath. Patients with COPD have other comorbidities as well like cardiovascular, Respiratory, and comorbidities related to liver and kidneys. Other comorbidities were also seen like lung cancer, anaemia, hypothyroid, HTN and DM. The age group most affected was 60. Males were at more risk than *females*. Smoking is one of the major risk factors. The drugs used were Sympathomimetics, Corticosteroids, Bronchodilators, Anticholinergics, Methylxanthines and Antimicrobial Therapy. COPD has significantly greater comorbidities than in other diseases and thus, it has to be taken into consideration in COPD control strategies.

KEY WORD: COPD, Comorbidities, Therapeutic Management

I. INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a chronic inflammatory condition in the lungs that causes obstructed airflow. Symptoms include difficulty breathing, cough, the development of mucus (sputum), and wheezing. Long-term exposure to toxic chemicals or particulate matter, most often due to smoke from cigarettes, induces this. People with COPD have a greater risk of developing heart disease, lung cancer and disorders. The two most common conditions which contribute to COPD are emphysema and chronic bronchitis. [1]

PATHOPHYSIOLOGY [2]

• Chronic inflammatory changes lead to detrimental changes and a lack of chronic airflow. The most common cause of this is exposure to the cigarette smoke.

• Noxious particles and gas inhalation stimulates neutrophils, macrophages, and CD8 + lymphocytes that trigger the chemical mediators, including factor-α, interleukin-8, and B4 tumour necrosis. Inflammatory cells and mediators resulted in extensive damaging changes in airways, pulmonary vasculature, and parenchyma in the lungs.

• There may also be oxidative stress and an imbalance between the aggressive and defensive lung defence mechanisms (proteases and antiproteases). Smoke from cigarettes produced oxidants reacts with proteins and

lipids and causes damage to the tissues. Oxidants also encourage inflammation by inhibiting antiprotease activity, which intensifies protease – antiprotease imbalance.

• The defensive α1-antitrypsin (AAT) antiprotease inhibits protease enzymes like neutrophil Elastase. It targets elastin, a major component of alveolar walls, in the presence of unopposed AAT action. Inherited AAT deficiency raises the risk of early emphysema. Unbalance is associated with increased protease activity or decreased antiprotease activity in the emphysema from cigarette smoking.

• Inflammatory exudation in airways allows the number and size of goblet cells and mucus glands to increase. The secretion of the mucus decreases, and ciliary motility is reduced.The smooth muscle and connective tissue inside the airways are thickened. Chronic inflammation leads to scarring and fibrosis. Diffuse airway narrowing occurs in small peripheral airways and is more prominent.

• Smoking-related COPD typically results in the centrilobular emphysema which primarily affects bronchioles in the respiratory system. In the AAT deficiency, pan lobular emphysema is seen and spreads to the sacs alveolar ducts.

• Vascular changes like thickening of the pulmonary arteries, can lead to pulmonary artery endothelial dysfunction. Later, the structural changes, particularly during exercise, enhances the pulmonary pressure. In extreme COPD secondary pulmonary hypertension leads to right-sided (Cor pulmonale) heart failure.[3]

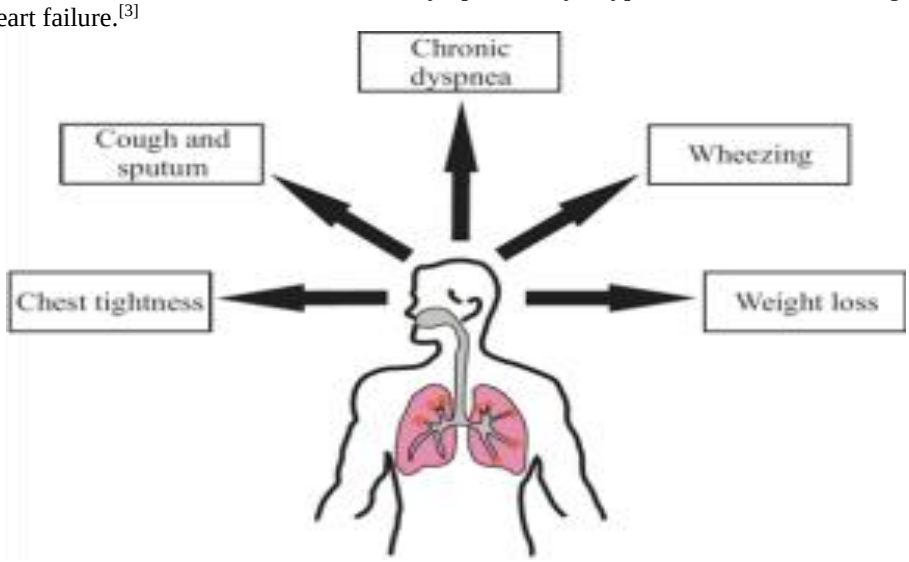


Fig.1 Symptoms of COPD [3]

RISK FACTORS OF COPD

- **Tobacco smoking [1]**

Long-term cigarette smoking is the major risk factor for COPD. The longer a person smokes, the more packs a person smokes, the higher the risk. Pipe smokers, cigar smokers and marijuana users, as well as people exposed to large amounts of second-hand smoke, may be at risk too.

- **Asthma [1]**

People who smoke with asthma are at a greater risk as combining asthma, chronic inflammatory airway disease and smoking raises the risk of COPD even more.

- **Dust and toxins [1]**

Exposure to toxins and dust at workplace. Long-term occupational exposure to chemicals, vapours and dusts can irritate and inflammate the lungs. Exposure to emissions generated through burning of fuel.

- **Infections [4]**

Serious infections of the viral and bacterial lungs in early childhood were associated with decreased lung function and increased respiratory symptoms in adulthood, which lead to COPD growth. In particular, chronic lung infections such as tuberculosis are related to COPD.

- **Age [1]**

COPD progresses slowly over the years so when symptoms begin, most people are at least 40 years old.

- **Genetics [1] [5] [6]**

The rare alpha-1-antitrypsin deficiency in the genetic disorder is the cause of some of the COPD cases. Certain genetic factors would likely make few smokers more vulnerable to the illness. The main genetic risk factor for COPD is extreme alpha 1-antitrypsin (AAT) deficiency. Patients with severe AAT deficiency (most specifically,

Protease Inhibitor [PI] are at a greater risk of developing COPD, particularly when they are smoking. Just 1–2 percent of cases of this condition are clarified by the only known genetic risk factor for COPD, extreme AAT deficiency.

COMORBIDITIES ASSOCIATED WITH COPD

a. CARDIOVASCULAR COMORBIDITIES [7]

Because they have a direct impact on patient safety, Vascular and heart diseases are amongst the most significant comorbidities found in COPD. Coronary heart disease and COPD share the same major risk factor, that is to say smoking. Therefore, the close epidemiological link between the two diseases is unaware. The occurrence of signs of severe chronic bronchitis enhances the risk of mortality by 50 per cent due to a coronary accident. The effect of obstructive airway disease, described by a reduction in the ratio of forced expiratory volume in 1s (FEV1) to (FVC), is less evident, since it only increases the risk of coronary event by 30 percent. Nevertheless, all-cause mortality decreases by 14% for every 10% decline in FEV1, CV mortality increases by 28% and non-fatal accidents increase by 20%.

b. DEPRESSION AND ANXIETY [7]

Both symptoms are widespread in COPD, anxiety and dyspnea are closely related, and depression in COPD is more prevalent than in other chronic conditions. [7].Depression often represents growing comorbidity in the elderly population. Its frequency is 1–5 percent in outpatients, 13 percent in institutionalized patients and 40 percent in resp. failure patients. Yoannes .et al. showed that a significantly higher rate of distress in a population of 96 elderly COPD patients is seen than in stable or otherwise impaired elderly participants.

c. SLEEP DISTURBANCE [7]

Impaired standard of sleep tends to be more prevalent in COPD patients. COPD is an anxiety risk factor with an OR of 1.9 (95 per cent CI 1.5–2.5; $p<0.001$) to a control population. According to Cornick et al. out of a sample of 50 patients with severe COPD, 36% reported difficulty falling asleep, 42% of non-restorative sleep and 76% of over two extended wakefulness cycles a night, while 28% using hypnotics.

d. HYPERTENSION IN COPD [8]

A common complication of COPD is mild-mod pulmonary hypertension; it is associated with increased risks of exacerbations and reduced survivability. During exercise, sleep and exacerbation, pulmonary hypertension typically worsens.

e. DIABETES MELLITUS [9] :

There are well-known risk factors for diabetes, including obesity, lack of exercise, sex, cholesterol levels and high blood pressure. But there is also growing evidence linking COPD to diabetes. Studies suggest that the progression of type 2 diabetes could be responsible for the general inflammation characteristic of COPD. Additionally, certain risks resulting from COPD may justify their relationship with diabetes, such as hypertension, oxygen deficiency (hypoxia), and corticosteroid use to reduce inflammation. In effect, high blood sugar rates often influence lung function, and the risk of developing COPD may potentially increase.

f. HYPOTHYROID [10]

Research indicates that hypothyroidism patients with COPD have a high level of exacerbation but, given this, have a comparable quality of life in patients with normal thyroid function. Directed by Sevinc Sarinc Ulasli (Afyon Kocatepe University, Turkey), the research team examined a group of 44 patients with hypothyroidism, 44 patients with COPD and normal thyroid function, and 40 healthy controls. They found that the patients with hypothyroidism reported slightly more severe exacerbations over the span of 1 year as compared to the patients with euthyroid, at an average of 1.50 vs 0.86.

g. BRONCHIAL ASTHMA [11]

Bronchial asthma and COPD are pulmonary obstructive disorders which have affected millions of people across the globe. Asthma is a severe global health problem which affects an estimated 300 million people. COPD is one of the major causes of chronic morbidity and mortality, and one of the biggest public health problems in the world.

h. URINARY TRACT INFECTIONS [12]

Inhaled LAA, widely used for moderate to extreme COPD, was shown to reduce hospitalizations, visits to emergency rooms, and acute exacerbations due to COPD, but was also correlated with UTI in a previous meta-analysis.

i. CHRONIC KIDNEY DISEASE [13]

CKD is a frequent comorbidity occurring among patients with COPD that affects the mortality and other outcomes of the patients, according to observational results examining the relationship between the two conditions. More often patients with comorbid CKD reported other co-morbidities, including CV and cerebrovascular disease, peripheral artery disease, DM, gout and malignancy. The patients, irrespective of other cardiovascular comorbidities, were at increased mortality risk.

j. ACUTE KIDNEY INJURY [14]

According to the study findings, patients with COPD and AKI had an elevated risk of mortality as compared to those who had COPD alone. Acute kidney damage is used as a indicator of bad outcome in COPD exacerbation patients. It is recommended that patients with AKI as well as COPD exacerbation be classified as patients with higher risk, and therefore more closely monitored, particularly after early hospital discharge. Compared to patients without AKI, those with COPD and AKI had a 1.8-fold enhanced mortality risk after occurrence of the exacerbations during the first 6 months (95 percent CI, 1.61-2.03).

k. **LOWER RESPIRATORY TRACT INFECTIONS [15]**

LRTI's, both acute and chronic, occur with greater frequency in the environment of chronic obstructive pulmonary disease (COPD). Provided that these infections contribute substantially to the patient's clinical path with COPD, they represent a serious COPD comorbidity. Recurring acute infections triggered by bacterial and/or viral pathogens are now specifically related to COPD exacerbations.

l. PNEUMONIA [15] [16]

For people with COPD, pneumonia is especially harmful as it causes an increased risk of respiratory failure. It is when the body either doesn't get enough oxygen or absorbs carbon dioxide with no success. Incidence of COPD pneumonia has gained significant recent attention, as the use of inhaled corticosteroids tends to increase it. The role of chronic infection in COPD pathogenesis is an active field of research with several types of pathogens being likely involved.

m. **ANEMIA [8] [17]**

Chronic disease anemia (CDA), with hemoglobin circulating at consistently low levels, is an immunologic-driven abnormality that occurs in many inflammatory diseases and in chronic heart failure. While "traditionally" synonymous with polycythemia is COPD, the systemic inflammation now recognized as a COPD characteristic makes it a potential cause of ACD. If present in COPD, anemia can exacerbate dyspnea and reduce the tolerance to exercise.The prevalence of anemia in COPD patients ranges from 7.5 to 33 per cent. CDA is possibly the most common form of COPD-related anemia. It is driven by a systemic inflammation mediated by COPD. COPD-anemia is associated with increased utilization of health care services, reduced QOL decreased survival and a higher risk of hospitalization.

n. SEPSIS [18]

Sepsis is a life-threatening condition that is characterized by disruption of the organ, triggered by the host's dysregulated response to the infection. Due to impaired barrier function and the use of corticosteroids COPD patients were found to have a greater risk of sepsis.

o. CEREBRO VASCULAR ACCIDENT [19]

Emerging data shows that a contributing factor for stroke may be the COPD. Smoking is one of the preventable confounders for the risk of COPD and stroke, which leads to fatal accidents and stroke. Patients treated for either condition will receive comprehensive cessation of tobacco education and support both at the hospital and as they transition to their post-discharge residence.

p. PULMONARY KOCH AND COPD [18]

Two major causes of death and morbidity in our country are chronic obstructive pulmonary disease (COPD) and tuberculosis (TB). A few patients with TB experience post tubercular airway disease or COPD associated with TB. This is the most frequently reported relationship.[12] The patients with COPD are also at a higher risk of developing pulmonary TB. The background of TB negatively impacts the long-term trajectory of COPD with early mortality and increased frequency of exacerbations COPD often affects the clinical diagnosis of TB and is also one of the risk factor for increased morbidity and mortality by TB.

q. RESPIRATORY FAILURE [20]

Respiratory failure is a common and serious occurrence and is often associated with extreme COPD exacerbations. Good ventilation / perfusion mismatching with a relative increase in physiological dead space leads to hypercapnia and thus acidosis. This is primarily the product of a change to a fast-shallow respiration pattern and an increase in each breath's dead space / tidal volume ratio. This breathing pattern is the product of adaptive physiological responses that reduce the risk of breathing muscle tiredness and decrease breathlessness.

r. COR PULMONALE [21]

This form of right-sided heart disease can evolve gradually or unexpectedly, and lung disease often triggers it. COPD is one of the most common cause of Cor pulmonary COPD, and is a common cause of heart failure, especially Cor pulmonary disease. COPD is a category of pulmonary diseases including emphysema and chronic bronchitis, which are worsening disorders related to a variety of complications.

s. LUNG CANCER [22]

COPD patients are at an increased risk for both the occurrence of primary lung cancer, as well as poor outcomes following diagnosis and treatment of lung cancer. Because of known impairments in lung function, COPD patients often fail to meet standard tolerance requirements for conclusive surgical lung cancer therapy. [13] The correlation between COPD and lung cancer was documented in various studies and is especially independent of patient age

or level of exposure to tobacco. In people with COPD the incidence of lung cancer is two to five times higher than in smokers without COPD. While the risk of lung cancer has long been identified in COPD patients, improvements in surgical treatment as well as an increased understanding of COPD's lung resection physiology may help improve outcomes for COPD patients and lung cancer patients.

PHARMACOLOGICAL TREATMENT

i.ANTICHOLINERGICS [23] [24]

For their bronchodilatory effects, anticholinergics are used in COPD management. Such agents contradict muscarinic receptors (i.e. subtypes M1, M2, and M3). Blocking these receptors in the airways-smooth muscle inhibits acetylcholine production, which decreases cyclic monophosphate levels of guanosine in order to achieve bronchodilation. In COPD patients, ipratropium bromide and tiotropium bromide are two widely used anticholinergics. These two agents have the most widely reported side effects, including dry mouths, nausea and metallic taste. Tachycardia, blurry vision, urinary retention and constipation are other common side effects.

ii.SYMPATHOMIMETICS [25] [26]

Level one treatment for chronic treatment or COPD is known to be a sympathomimetics. Nevertheless, sympathomimetics remains the first choice of therapy for their rapid initiation of action in acute exacerbations and rescue. The agents include albuterol, levalbuterol, and bitolterol. Beta-2 selective sympathomimetics allow adenosine-3,5'-monophosphate formation to be induced by bronchodilating the enzyme adenyl cyclase. Sympathomimetic pathways such as α-adrenergic agonists, β-adrenergic agonists, and dopamine agonists may be directed (direct medication-receptor interactions) or indirect (medicine-receptor interactions) like MAOIs, COMT inhibitors, release stimulants, and reuptake inhibitors that enhances the rates of catecholamine in endogenous medicines.

Direct Adrenergic agonists : A sympathomimetic effect can be created by direct stimulation of the α and the β-adrenergic receivers. A commonly used β2-agonist is Salbutamol . Phenylephrine, isoproterenol, and dobutamine are other examples.D1 receptor activation is used intravenously by dopamine agonists like fenoldopam for evaluation of hypertensive crisis.

Indirect dopaminergic stimulants, like amphetamine, ephedrine, and propylhexedrine, work by releasing dopamine and norepinephrine and, in other cases, by preventing recovery.

Side effects include thin shaking, normally in the face, the tension of the nerve, tipping, the vasodilation in the periphery, tachycardia sinus, hypokalaemia-salbutamol after heavy doses, hypersensitivity, like bronchospasm paradoxical, diabetic immunity to glucose deficiency.

iii.INHALED BRONCHODILATOR [27]

Inhaled bronchodilators drugs called bronchodilators help relax tight airway muscles. Normally, they are taken via an inhaler or nebulizer. Four to six hours of short-acting bronchodilators last. You just use it when you need it. There are long-acting formulations that you can use every day for continuing symptoms. We last approximately 12 hours [16] [18]

Bronchodilators may be either:

- **Long-acting bronchodilators** [28]

Long-acting –routinely used to monitor asthma and COPD respiration and increase the effectiveness of asthma corticosteroids. Example include salmeterol, formoterol etc.

- **Short acting bronchodilators** [28] [29]

Used as an urgent relief from sudden and unexpected breathing attacks. Examples include albuterol levalbuterol etc.

Side effects include: anxious or nervous sensations, increased heart rate or muscle spasms, displeasure of the abdomen.

iv.CORTICOSTEROIDS [30] [31]

Corticosteroid use in multiple clinical conditions can be used in COPD like the

1. Systemic application for acute exacerbation
2. A Systematic framework for chronically stable COPD
3. Chronically controlled COPD inhalation.

- **Inhaled corticosteroids** [31] :

The most important control agents of asthma and only drug that can effectively alleviate the characteristic inflammation of the airways are by far the inhaled corticosteroids (ICS, which is also known as glucocorticoids, glucocorticoids, and steroids). Because of ICS, the suppression of pulmonary inflammation in COPD is largely ineffective and has a low clinical impact. In both asthma and COPD, ICS are also given with long-acting β2

agonists as a combination inhaler.The short-term application of inhaled corticosteroids results in a vast majority of people's tolerance with little to no side effects. Long-term use can cause oral thrush (fungal infections that grow inside your mouth) to treat a chronic condition, such as asthma.
Examples include budesonide, mometasone, fluticasone.

- **Oral corticosteroids** [32] [33]

These oral medications are normally administered for short-term use, generally for five to seven days. The severity of your symptoms, the strength of the drug, and other factors will depend on your dosage. Normal short-use is also recommended. It makes the long-term use of the drug less likely to be difficult.
Side effects of longer-term oral corticosteroids include: osteoporosis (fragile tissue), high blood pressure (high blood pressure), diabetes, weight gain, increased infection susceptibility, cataracts, and glaucoma (eye disorders), skin dissipation, quick bruises and muscle fatigue.
Examples include: prednisolone, hydrocortisone, prednisone, methylprednisolone.

v.PHOSPHODIESTERASE - 4 INHIBITORS [34] [35] [36]

The risk of exacerbations in patients with serious COPD, combined with a history of aggravation and persistent bronchitis, has been minimized with a selective phosphodiesterase 4 inhibitors. Phosphodiesterase (PDE) inhibitors modulate inflammation of the lungs and induce bronchodilation, rising 3', 5" monophosphate in airway muscle, and inflammatory cell intracellular cyclic adenosine. The only licensed inhibitor for use in chronic obstructive pulmonary disease (COPD), is PDE-4 (PDE4I). Roflumilast. It results clinically primarily in chronically bronchitis patients and recurrent COPD exacerbations. Roflumilast for the use of other COPD drugs as an additional or replacement treatment reduces exacerbations and modestly increases lung function.
Examples of PDE4 inhibitors include apremilast, diazepam, roflumilast, cilomilast.

vi.METHYL XANTHINES [30] [37]

For at least five decades methylxanthines have been eligible for COPD diagnosis and at the same time first-line therapy has been considered. Theophylline is the most popular methylxanthine that can be used in clinical practice in many chronic, stable disease patients. Methyl-xanthines can induce bronchodilation by various pathways, including:
• Activation of phosphodiesterase, thus raising cyclic adenosine monophosphate levels
• Inhibition of Ca+ ion flow into smooth muscle
• Antagonism of Prostaglandin
• Activation of endogenous catecholamines
• Adenosine receptor antagonism suppression of mast cell and leukocyte release mediators.

vii.ANTIBIOTICS

They are given to encounter respiratory infections.

viii.VACCINES [38]

According to a recent report in the International Journal of Chronic Obstructive Pulmonary Disease, patients should be given the pneumococcal conjugate vaccine (PCV) and influenza vaccines in patients with COPD to minimize the risk of exacerbations [63]

- **Influenza (flu) vaccine** [39]

Individuals with COPD should be given an annual vaccine against the flu. Normally, the flu season lasts from October to May and peaks from December to Feb. The flu can be transmitted during the year, however, at any time.

- **Pneumococcal vaccine** [39]

For all adults 65 years of age or older, particularly those with chronic lung conditions like COPD, the two pneumococcal vaccines, PCV13, and PPSV23, are recommended. They are also suggested especially for younger people with COPD. Among other things, these vaccines provide protection against various pneumococcal bacteria which can cause pneumonia.

II. MATERIAL AND METHODS

***Study Design*:** Prospective, open label, observational study
Source of data and materials:
• Patient consent form
• Patient data collection form
• Patient case note/prescription
Study procedure*:*

- Enrolling of patients for study after taking consent form
- Patients will be grouped according to the inclusion and exclusion criteria.

- Demographic details and other details will be recorded by using patient data collection form
- Patient will be interviewed specifically for fulfilment of the purpose of the objectives of this study
- Analysis will be done after the collection of statistical data
- Results will be interpreted after analysis of data
- Finally results will be concluded and impression will be highlighted

Duration of the study: The study was conducted for a period of 6 months.
Place of study: The study was conducted at MEDICOVER HOSPITAL.
Inclusion criteria:

- Inpatients of department of Pulmonology Department diagnosed with Chronic Obstructive Pulmonary Disease.
- Any patient with a chief complaint of SOB and is at a risk of COPD.
- All patients of either gender with COPD attending pulmonology unit.
- Patients above 18 years of age.
- Patients with known COPD.
- Patients with known Comorbidities.

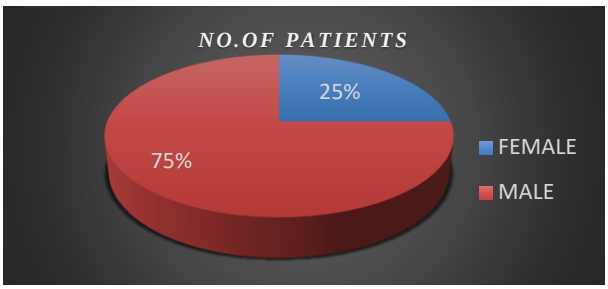
Exclusion criteria:
• Patients who are not willing to give the consent
• Patients who were not diagnosed with COPD
• Pregnant women are not included.
• Patients with psychological disorder.
• Patients with cognitive impairment.

III. RESULT

1. DISTRIBUTION OF PATIENTS BASED ON THE SEX

Table no 1

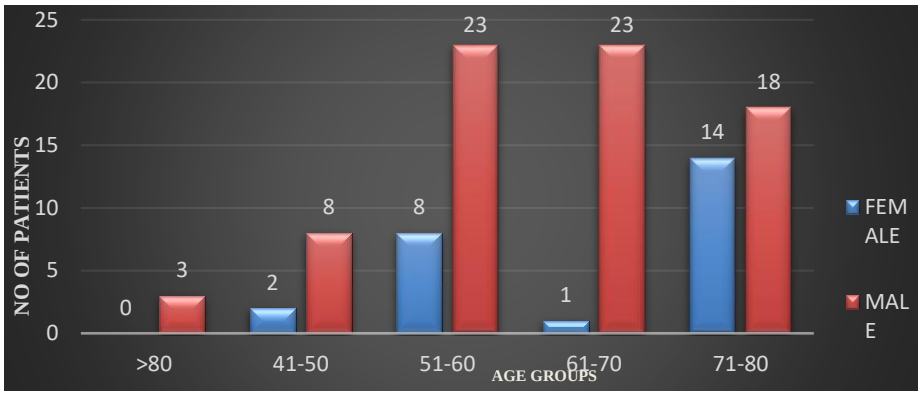
SEX	NO. OF PATIENTS (%)
MALE	75%
FEMALE	25%



2. DISTRIBUTION BASED ON AGE

Table 2.

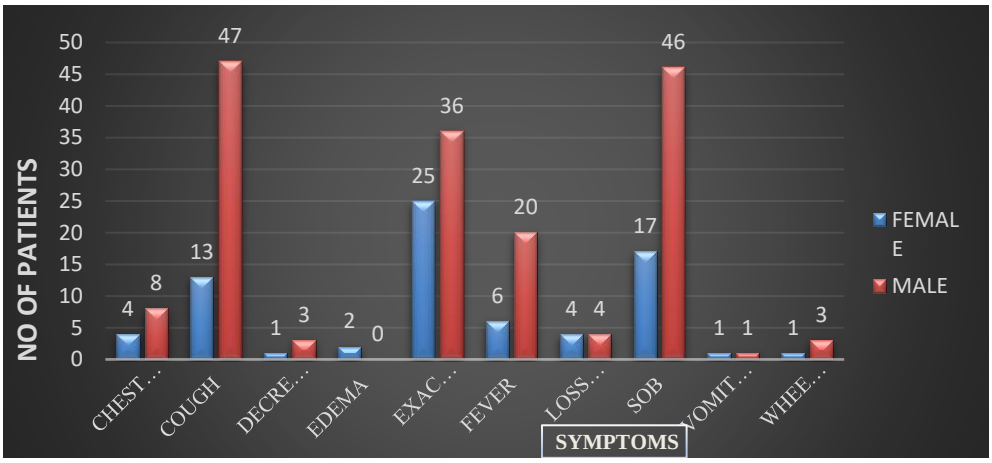
AGE (YRS)	MALE	FEMALE
41-50	8	2
51-60	23	8
61-70	23	1
71-80	18	14
>80	3	0



3. DISTRIBUTION OF PATIENTS BASED ON THE SIGNS AND SYMPTOMS

Table 3

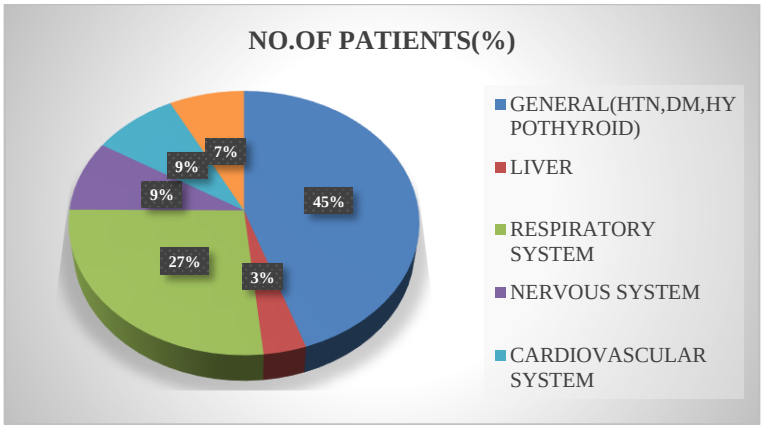
S.NO	SYMPTOMS	MALE	FEMALE
1	COUGH	47	13
2	SOB	46	17
3	EXACERBATIONS	36	25
4	FEVER	20	6
5	CHEST TIGHTNESS	8	4
6	LOSS OF APPETITE	4	4
7	WHEEZING	3	1
8	DECREASED URINE OUTPUT	3	1
9	VOMITING	1	1
10	EDEMA	0	2



4. DISTRIBUTION OF THE PATIENTS BASED ON THE COMORBIDITIES EFFECTING THE SYSTEMS:

Table 4.

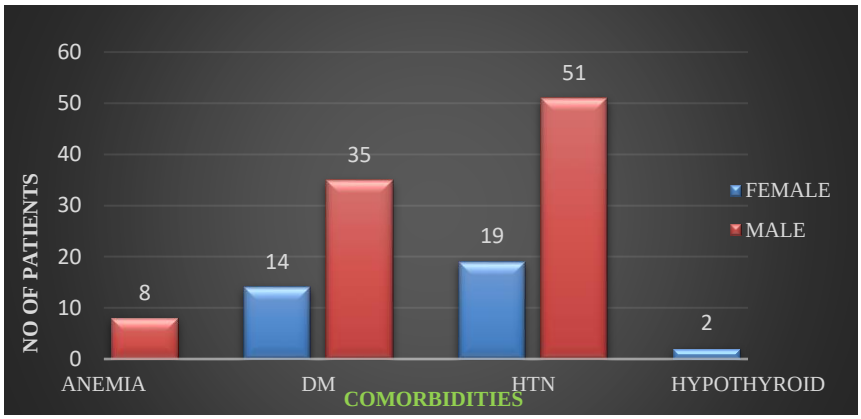
S.NO.	COMORBIDITIES RELATED TO:	NO. OF PATIENTS (%)
1	GENERAL (HTN, DM, HYPOTHYROID)	45%
2	RESPIRATORY SYSTEM	27%
3	LIVER	3%
4	NERVOUS SYSTEM	9%
5	CARDIOVASCULAR SYSTEM	9%
6	RENAL SYSTEM	7%



5. DISTRIBUTION OF PATIENTS BASED ON THE GENERAL COMORBIDITIES:

Table 5.

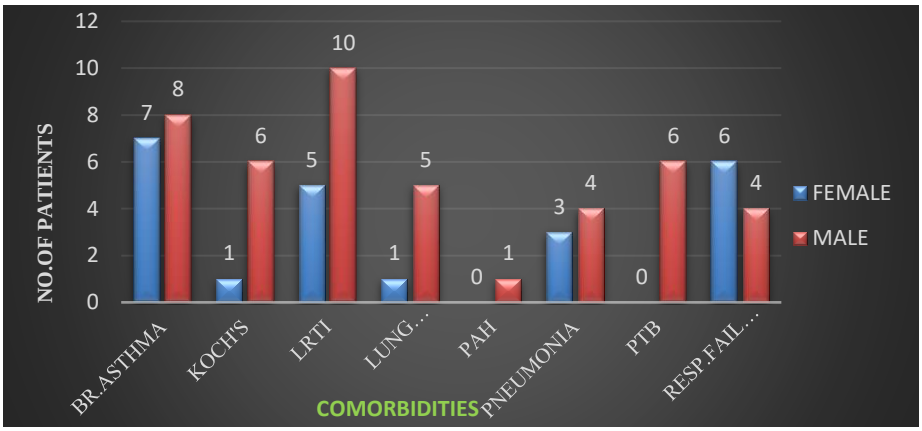
S.NO.	COMORBIDITIES	NO. OF MALES	NO. OF FEMALES
1	HYPERTENSION	51	19
2	DIABETIES MELLITUS	35	14
3	ANEMIA	8	2
4	HYPOTHYROID	3	2



6. **DISTRIBUTION OF PATIENTS BASED ON COMORBIDITIES OF THE RESPIRATORY SYSTEM.**

Table 6.

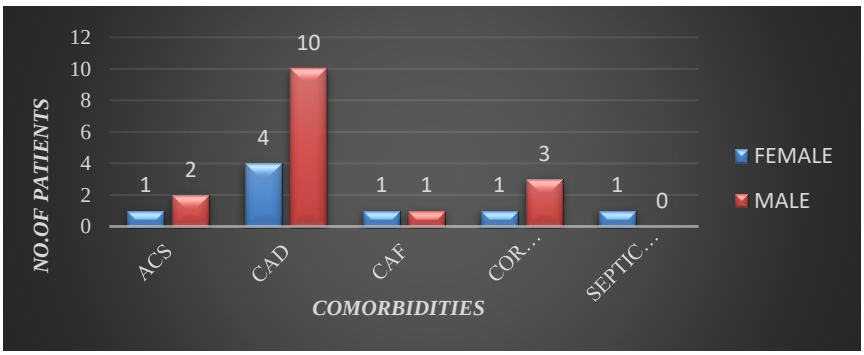
S.NO	COMORBIDITIES	NO. OF MALES	NO. OF FEMALES
1	LRTI	10	5
2	BR. ASTHMA	8	7
3	KOCH'S	6	1
4	PTB	6	0
5	LUNG CANCER	5	1
6	PNEUMONIA	4	3
7	RESP.FAILURE	4	6
8	PAH	1	0



7. DISTRIBUTION OF THE PATIENTS BASED ON THE COMORBIDITIES RELATED TO THE CARDIOVASCULAR SYSTEM

Table 7.

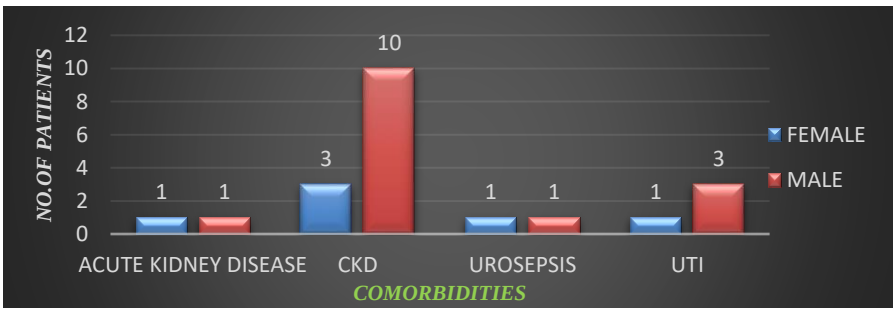
SNO	COMORBIDITIES	MALE	FEMALE
1	CAD	10	4
2	COR PULMONALE	3	1
3	ACUTE CORONARY SYNDROME	2	1
4	CHRONIC ATRIAL FIBRILLATION	1	1
5	SEPTIC SHOCK	0	1



8. DISTRIBUTION OF PATIENTS BASED ON THE COMORBIDITIES RELATED TO THE URINARY SYSTEM

Table 8

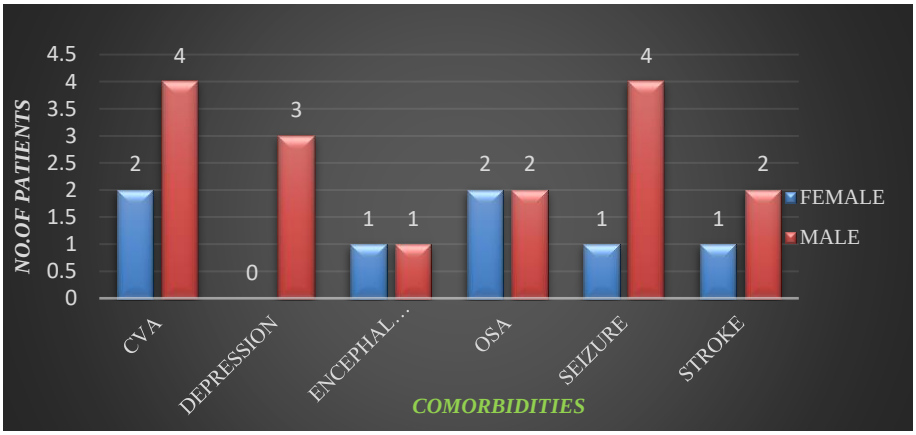
S.NO.	COMORBIDITIES	MALES	FEMALES
1	CKD	10	3
2	UTI	3	1
3	UROSEPSIS	1	1
4	AKD	1	1



9. DISTRIBUTION OF THE PATIENTS BASED ON THE COMORBIDITIES RELATED TO THE NERVOUS SYSTEM.

Table 9.

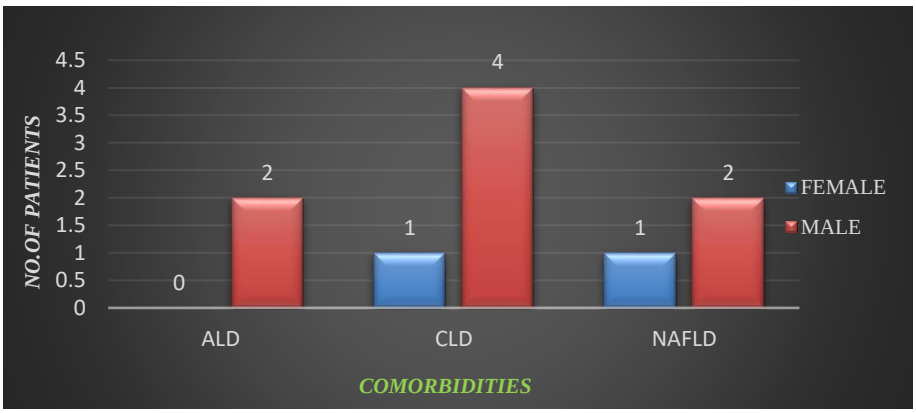
SNO	COMORBIDITIES	MALES	FEMALES
1	CVA	4	2
2	SEIZURE	4	1
3	DEPRESSION	3	0
4	OSA	2	2
5	STROKE	2	1
6	ENCEPHALOPATHY	1	1



10. DISTRIBUTION OF THE PATIENTS BASED ON THE COMORBIDITIES RELATED TO THE LIVER.

Table10.

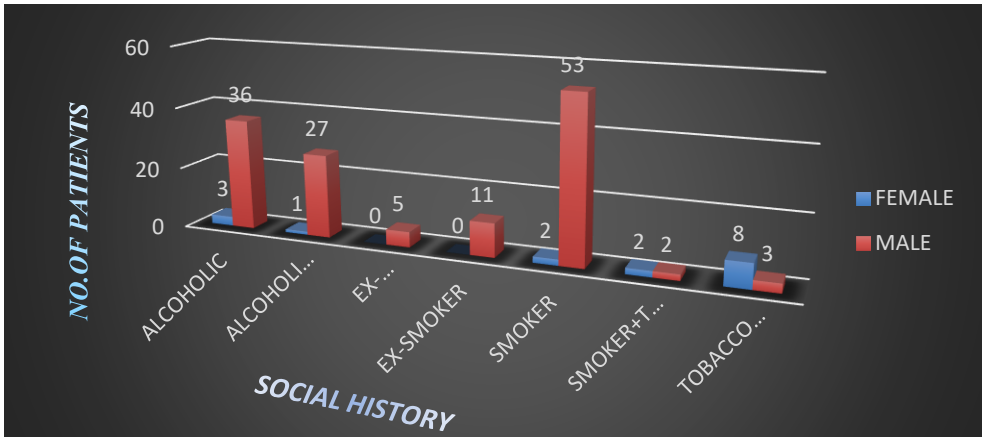
S.NO.	COMORBIDITIES	MALES	FEMALES
1	CLD	4	1
2	ALD	2	0
3	NAFLD	2	1



11. DISTRIBUTION OF THE PATIENTS BASED ON SOCIAL HISTORY

Table 11.

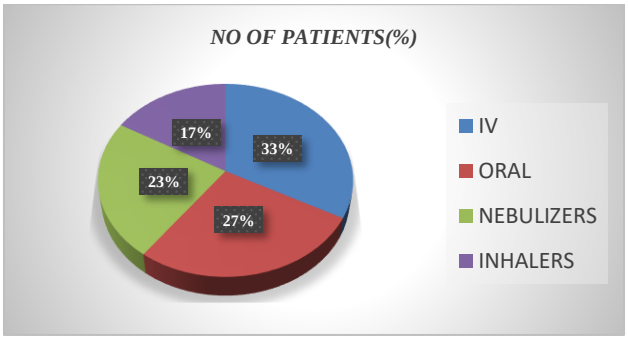
SNO	PERSONAL HISTORY	MALE	FEMALE
1.	SMOKER	53	2
2.	ALCOHOLIC	36	3
3.	ALCOHOLIC+SMOKER	27	1
4.	EX-SMOKER	11	0
5.	EX – ALCOHOLIC	5	0
6.	SMOKER+TOBACCO CHEWER	2	2
7.	TOBACCO CHEWER	3	8



12. ROA OF THE DRUGS USED FOR THE TREATMENT OF COPD

Table 12.

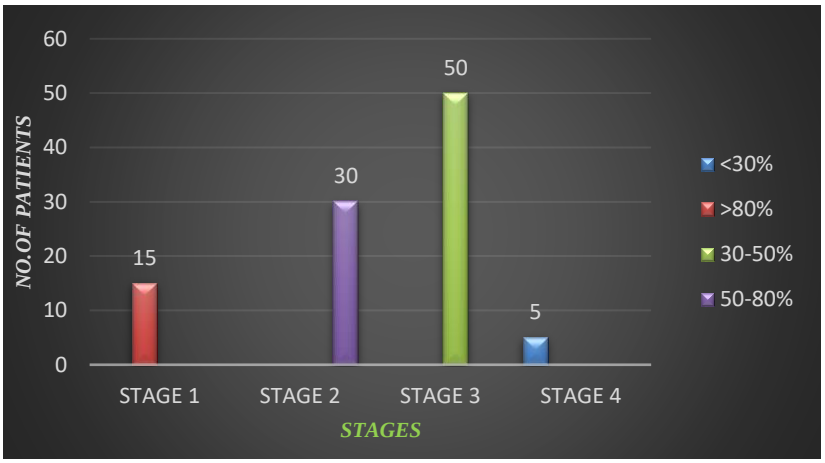
S.NO.	ROA	NO. OF PATIENT'S (%)
1	IV	33%
2	ORAL	27%
3	NEBULIZERS	23%
4	INHALERS	17%



13. DIAGNOSIS:

Table 13.

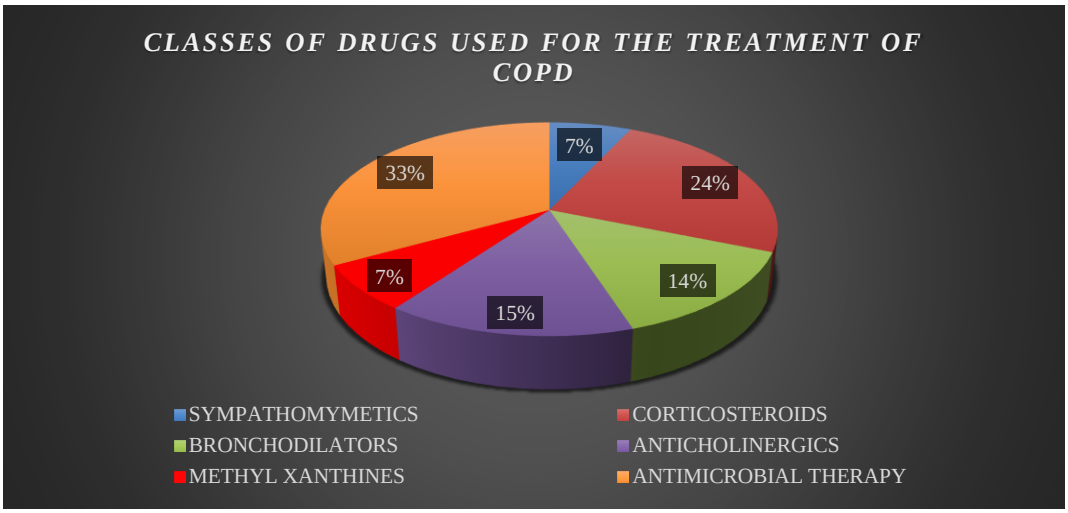
S.NO.	STAGES	% OF PREDICTED FEV1 VALUES	NO. OF PATIENTS
1	MILD	>80%	15
2	MODERATE	50-80%	30
3	SEVERE	30-50%	50
4	VERY SEVERE	<30%	5



14. PHARMACOLOGICAL MANAGEMENT OF COPD

Table 14.

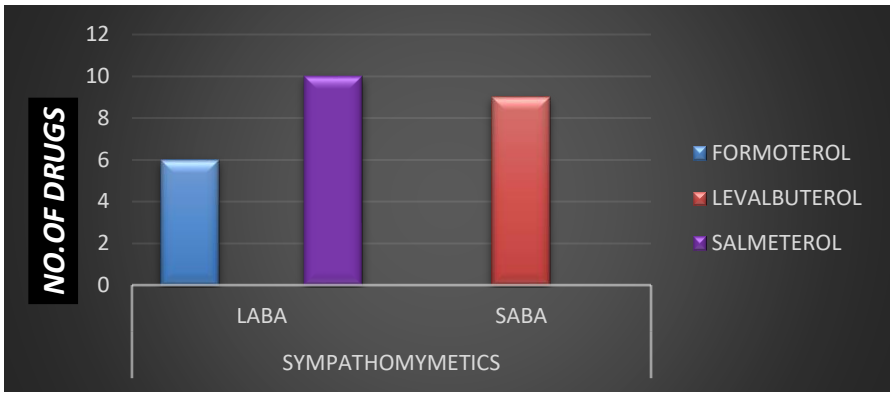
S.NO.	CLASS OF DRUGS	NO. OF DRUGS PRESCRIBED
1	SYMPATHOMYMETICS	25
2	CORTICOSTEROIDS	88
3	BRONCHODILATORS	50
4	ANTICHOLINERGICS	54
5	METHYL XANTHINES	25
6	ANTIMICROBIAL THERAPY	120



14.1. SYMPATHOMIMETICS

Table 14.1

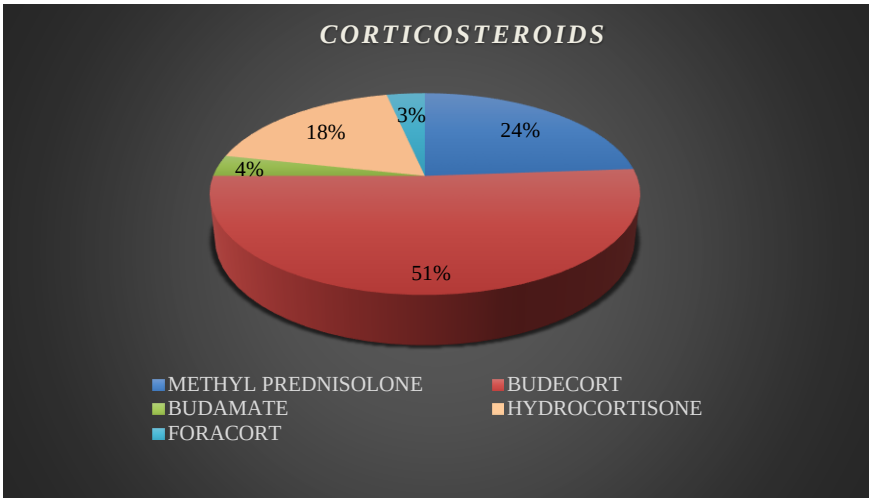
S.NO.	CLASS OF DRUG	DRUGS PRESCRIBED	NO. OF DRUGS
1	SABA	LEVALBUTEROL	9
2	LABA	SALMETEROL	10
		FORMETEROL	6



14.2 CORTICOSTEROIDS

Table 14.2

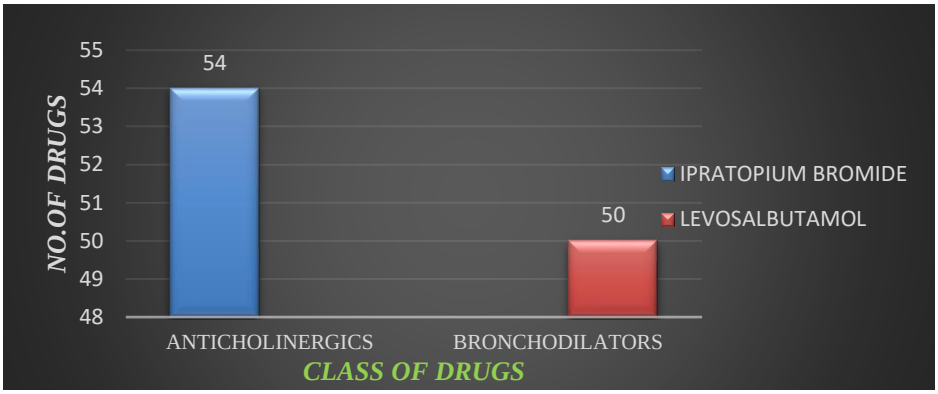
SNO.	DRUGS PRESCRIBED	NO. OF DRUGS (%)
1	BUDECORT	45 (51%)
2	METHYL PREDNISOLONE	21(24%)
3	HYDROCORTISONE	16(18%)
4	FORACORT	3(3%)
5	BUDAMATE	3(4%)



14.3 ANTICHOLINERGICS AND BRONCHODILATORS

Table 14.3

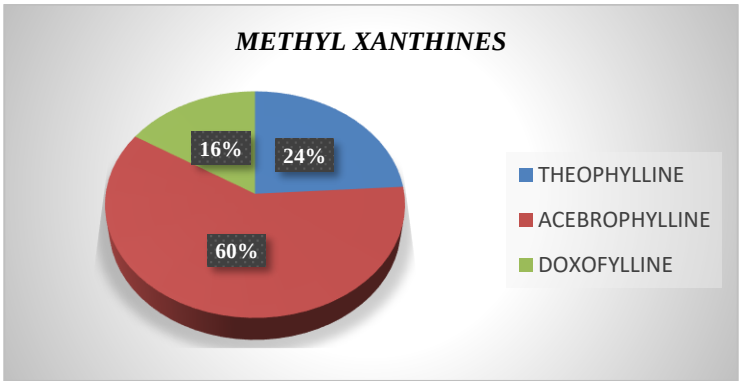
S.NO.	CLASS OF DRUGS	DRUGS GIVEN	NO. OF DRUGS
1	ANTI-CHOLINERGICS	IPRATROPIUM BROMIDE	54
2	BRONCHODILATORS	LEVOSALBUTAMOL	50



14.4 METHYL XANTHINES:

Table 14.4

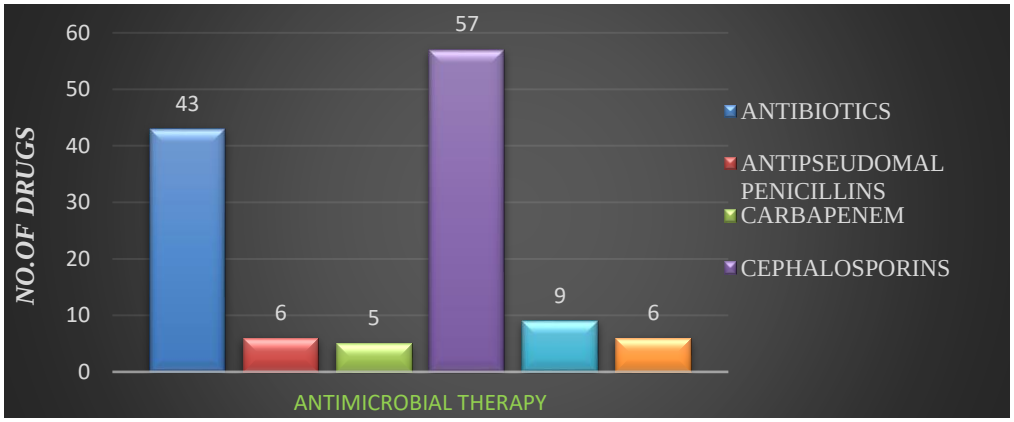
S.NO.	DRUGS PRESCRIBED	NO. OF DRUGS (%)
1	THEOPHYLLINE	24%
2	ACEBROPHYLLINE	60%
3	DOXOFYLLINE	16%



14.5. ANTI-MICROBIAL THERAPY

Table 14.5

S.NO.	CLASS OF DRUGS	NO. OF DRUGS
1	CEPHALOSPORINS	57
2	ANTIPSEUDOMAL PENICILLINS	6
3	CARBAPENEM	5
4	ANTIBIOTICS	43
5	FLOUROQUINOLONE	9
6	MACROLIDES	6



IV. DISCUSSION

In the present 6 months study, out of a total of 100 cases of COPD collected, we observed that allthe cases had at least one or more concomitant multimorbidity or comorbid conditions. Based on a study of patient medical history, the occurrence of comorbidities was reported and classified as separate comorbidities and combined diseases. In our study various comorbidities were associated along with COPD like CVD (9%), Respiratory (27%), liver (3%), renal (7%), neuro (9%), HTN (25%), DM (18%), Depression (1%). Weight loss and anemia were also frequently associated with this. Comparatively, the literature showed that 64.4% were treated for CVD, for diabetes (12.4%), and for depression (8%). This can be due to the lesser no. of participants (100) in this study compared to the other studies (>100). All these conditions potentiate COPD morbidity, resulting in higher hospitalizations and costs of healthcare. These can also cause death, regardless of respiratory failure. Comorbidities affect COPD management and, thus, need to be properly diagnosed and handled.

➢ Our study showed a predominance of male patients. A total of 100 prescriptions were assessed out of which 75% were males and 25% were females. But some studies concluded that COPD is more prevalent in women in developing countries than in men. In this study, we found no association between the frequency of men and COPD. This can be justified by a higher percentage of male participants in this study compared to the females in other studies and hence, higher risk of COPD prevalence. It was also found that females are more likely to be affected by comorbidities as compared to males.

➢ An association between smoking and COPD was found. Smoking is a major risk factor for COPD as it damages the air sacs airways and lining of the lungs. Up to 90% of the cases were as a result of lung damage due to smoking. We also found that the social habits were not only common among males (91%) but also found in females (9%). But comparatively, some studies concluded that it is more common in females as shifting smoking patterns have likely played a part. A systematic analysis of 11 studies found that female smokers have a faster annual decline in FEV1 relative to male smokers, even if they consume fewer cigarettes. Overall, women smokers are around 50 percent more likely than men to develop COPD. In our study the majority of the cases were smokers. Similarly, according to literature, about 75% of people with COPD were current or former smokers.

➢ Our study suggested that age also has an impact on comorbidities as the elderly are more likely to be prone to multiple comorbidities or illness. The average age recorded for males was 63.67 years and for females was 67.04 years showing a distinctive distribution of female patients of older age compared to their male counterparts. The prevalence of COPD was higher in older age groups, and females had higher rates than males.

➢ In our study, the patients were categorized into 4 stages based on their FEV1 values. 15 cases were under the 1st stage i.e. Mild (>80%) , 30 under the 2nd stage i.e. Moderate (50-80%) ,most of the cases i.e. 50 were under the 3rd stage i.e. severe (30-50%) ,and 5 cases were in the last stage /Stage 4 i.e. Very severe (<30%) .

➢ This study shows that the drugs used for the treatment of COPD include Sympathomimetics (7%), Corticosteroids (24%), Bronchodilators (14%), Anticholinergics (15%), Methyl Xanthines (7%), Antimicrobial Therapy (33%). O2 support and ventilation are also given to the patients in severe cases.Thus, smoking abstinence, symptoms management, and treatment of the exacerbations by appropriate pharmacological and non-pharmacological intervention in the initial stages may have a beneficial impact on the effects and progression of the disease, symptoms, exacerbations, as well as quality of life.

V. CONCLUSION

This is a prospective observational study on COPD cases involving patients ranging from 40 – 90 years. Patient scribe notes or case sheets along with Data collection form have been analyzed and utilized after patient consent to gather relevant data including but not limited to which includes demographic data & medication chart, periodic drug-related information followed by 1-1 interviews with the patients over a period of 6 months.

Around 100 cases were taken into account out of which 75% were males and 25% were females. The average age recorded for males was 63.67 years and for females was 67.04 years showing a distinctive distribution of female patients of older age compared to their male counterparts. Based on signs and symptoms patients were having Cough (24.79%), Exacerbations (26.03%), SOB (26.03%), Fever (10.74%), Chest Tightness (4.95%), Loss of Appetite (3.30%), Wheezing (1.65%), Decreased Urine Output (1.65%), Vomiting (0.82%) & Edema (0.82%).

Based on comorbidities in place, the top two offenders were Hypertension (25.27%) and Diabetes Mellitus (17.68%). Breaking down to each subtype for comorbidities observed we had soaring results for General type in Hypertension (70 cases), for Respiratory System in both LRTI and Br. Asthma (15 cases each), for Cardiovascular System in CAD (14 cases), for Urinary System in CKD (13 cases), for Nervous System in CVA (6 cases), For Liver Cases in CLD (5 cases).

Also, based on social history, smokers were the most affected group of individuals susceptible to COPD cases. Routes for drugs administered were IV (33%) followed by Oral (27%), Nebulizers (23%) and Inhalers (17%).

The diagnosis was done and the patients were categorized into 4 stages based on their FEV1 values.

15 cases were under the 1st stage i.e. Mild (>80%) , 30 were under the 2nd stage i.e. Moderate (50-80%), most of the cases i.e. 50 were under the 3rd stage i.e. severe (30-50%) , and 5 cases were in the last stage /Stage 4 i.e. Very severe (<30%) .

Table 14 describes the pharmacological management of COPD where it is evident from the findings that Antimicrobial therapy (33%) toppled among all drug treatments followed by Corticosteroids (24%). Digging deeper into each of the cases we see Sympathomimetics containing SABA like Levalbuterol (9), LABA like Salmeterol (10), and Formoterol (6). Corticosteroids that wereprescribed were as follows: Budecort (45%), Methyl Prednisolone (21%), Hydrocortisone (16%) followed by Foracort and Budamate (3%) respectively. The drugs prescribed under the class Methyl Xanthines were Acebrophylline (24%), theophylline (24%), and doxofylline (16%). The drugs prescribed under Anti Cholinergic's were Ipratropium Bromide (54%) and those under Bronchodilators were Levosalbutamol (50%), Anti-Microbial Therapy (33%).

All in all, it can be concluded that the prevalence of COPD cases is highly dependent on the age factor (>60 years most affected), chronic conditions, comorbidity type, and social habits. Comorbidities are frequently associated with COPD. Therefore, Healthcare workers must actively seek and treat these comorbidities appropriately as it can reduce mortality and hospitalizations. They must be aware to identify and prevent these comorbidities in order to prevent further worsening of the symptoms. They should engage in training sessions to ease the patients on much use of drugs and educational sessions to prevent further cases by educating them on social habits and their effects (Substance Abuse) and enforcing law officials in reducing the social norms affecting the crowd as well as monitoring periodically for ongoing recovery and prevention schemes. The ultimate result will be promising,

promoting a healthier, responsible, and safer society than ever before.20 mg on every other regimen had equal effect when compared to daily dose regimen of atorvastatin 40 mg &rosuvastatin 20mg.

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