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PRESCRIPTION PATTERN OF DRUGS USED INPATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE IN HYDERABAD METROPOLITAN

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ABSTRACT

Chronic obstructive pulmonary disease (COPD) is a group of lung diseases characterized by chronic increase in resistance of airway outflow, i.e. airway obstruction. The obstruction can happen in the level of the airway, from proximal airway trachea and larger bronchi, to distal terminal and respiratory bronchioles in varying degrees. Clinically, COPD shows a decreased expiratory flow rate, reflecting decreased maximal airflow rates during expiration (forced expiratory volume at one second [FEV1] overthe forced ventilator capacity less than 0.7). The main underlying pathogenesis of airway obstruction is narrowed airways and/or loss of elastic recoil. Common entities in COPD include emphysema, chronic bronchitis, asthma, and bronchi ectasis. COPD is the third leading cause o death and affects >10 million persons in the United States. It is also a disease of increasing public health importance around the world.The goal of drug therapy includes an attempt to improve lung function or slow the loss of lung function, and to prevent exacerbations. Most medications for COPD are administered by inhalation. Standard therapy consists of inhaled bronchodilators, either β-agonists or antimuscarinics (anticholinergics), and ICS. Oral agents, used less commonly, include methylxanthines (e.g., theophylline), phosphodiesterase- 4 inhibitors (e.g., roflumilast), and corticosteroids (prednisone or prednisolone). The aim of this survey is to determine whether Rational drug therapy is being practiced in patients with the air flow obstruction and COPD associated symptoms.

INTRODUCTION

- ***Chronic obstructive pulmonary disease (COPD)*** is a general name for the chronic airflow obstruction that develops most often as a result of chronic tobacco smoking, but also after exposure to biomass fuels. It is defined as a disease state characterized by air flow limitation that is not fully reversible and that is usually progressive and is associated with a chronic enhanced inflammatory response in the airways and lungs to noxious particles and gases[1].
- COPD includes.
- Emphysema, an anatomically defined condition characterized by destruction and enlargement of the lung alveoli;
- Chronic Bronchitis, a clinically defined condition with chronic cough and phlegm; and small airways disease, a condition in which small bronchioles are narrowed.

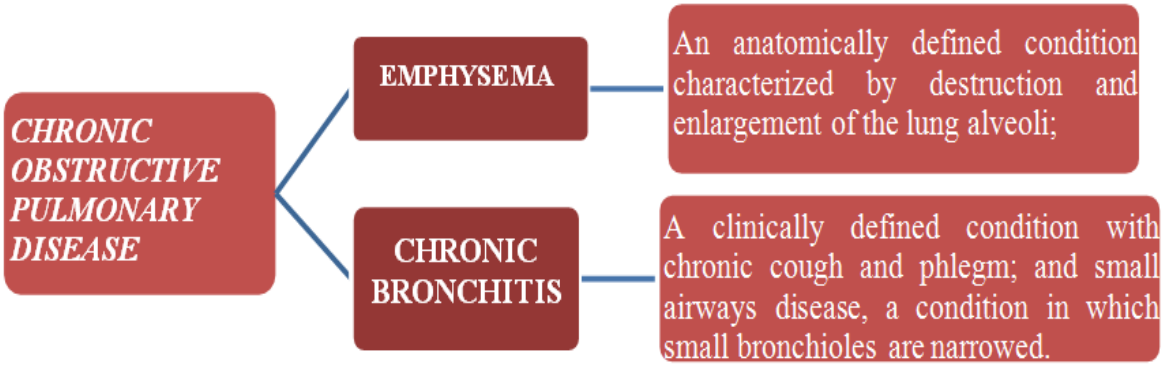

Figure 1: Chronic obstructive pulmonary disease forms.

COPD is present only if chronic air flow obstruction occurs; chronic bronchitis without chronic air flow obstruction is not included within COPD [2]. It (COPD) is a group of lungdiseases characterized by chronic increase in resistance of airway outflow, i.e. airway obstruction.

- The obstruction can happen in the level of the airway, from proximal airway trachea and larger bronchi, to distal terminal and respiratory bronchioles in varying degrees.
- Clinically, COPD shows a decreased expiratory flow rate, reflecting decreased maximal airflow rates during expiration (forced expiratory volume at one second [FEV1] over the forced ventilator capacity less than 0.7).
- Common entities in COPD include emphysema, chronic bronchitis, asthma, and bronchi ectasis.
- Most commonly associated with inhalational exposure to tobacco smoke and biomass fuels, this disease has been traditionally categorized into two distinct subsets: chronic bronchitis and emphysema.
- Chronic bronchitis is defined as a chronic productive cough for three consecutive months in two consecutive years with alternative etiologies having been ruled out first.
- Emphysema is defined as abnormal enlargement of airspaces distal to the terminal bronchioles that is associated with destruction of the airway walls in the absence of fibrosis.[3]
- COPD is a major health problem worldwide. Its prevalence is being recognized increasingly in countries at all levels of development. An ever-increasing number of smokers and an expanding number of elderly people are major factors in the surge in the worldwide prevalence of COPD.[1]

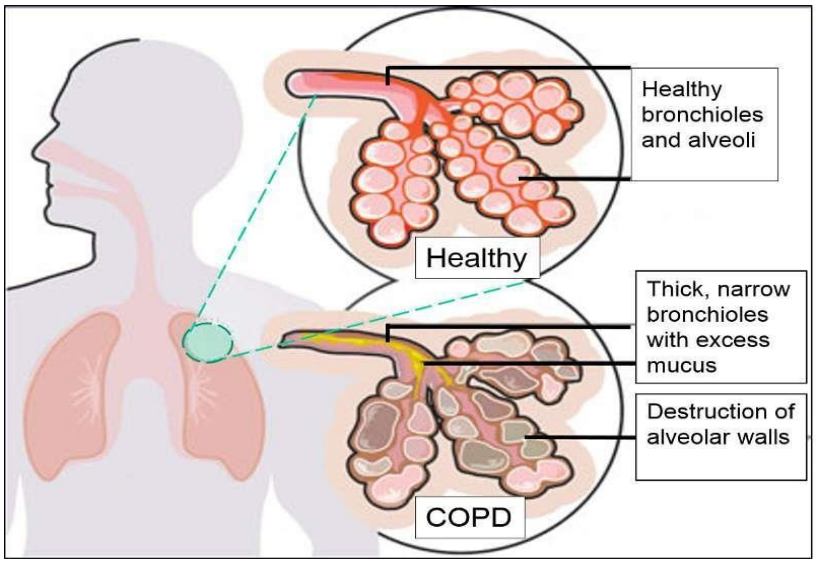

Figure 2: Diagram of Normal and Copd Affected Lungs.

According to criteria set by the Global Initiative for Obstructive Lung Disease, airflowobstruction is present when there is a reduction of the postbronchodilator FEV1/FVC ratio below 0.7 and its severity is graded by the percentage of the postbronchodilator FEV1 of the predictednormal FEV1.[1]

Severity Stage	Postbronchodilator FEV_1
1: Mild	$FEV_1 \geq 80\%$ predicted
2: Moderate	$50\% \geq FEV_1 < 80\%$ predicted
3: Severe	$30\% \geq FEV_1 < 50\%$ predicted
4: Very severe	$FEV_1 < 30\%$ predicted

Source: Modified with permission from Vestbo J, Hurd SS, Augusti AG, et al. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease. Am J Respir Crit Care Med. 2013;187(4):347–365.

Figure 3: Classifications of Airflow Limitation in Patients with Fev1/ Fev < 0.7

Epidemiology

COPD is a major health problem worldwide. Its prevalence is being recognized increasingly in countries at all levels of development. An ever-increasing number of smokers and an expanding number of elderly people are major factors in the surge in the worldwide prevalence of COPD.[1] Often under recognized and under diagnosed, COPD is a major source of morbidity and mortality worldwide. While COPD affects people from all walks of life, the reliance on spirometry to diagnose airflow obstruction has limited its diagnosis.[3] Not only a lethal disease, COPD is also associated with significant morbidity and health care utilization. Due to its association with exposure to tobacco smoke and biomass fuel, as well as advanced age, COPD is often accompanied with a myriad of systemic illnesses, including cardiovascular disease and diabetes mellitus, that significantly impact productivity and quality of life.[3]

Risk Factors

Despite its widespread prevalence, there is limited causal evidence to associate risk factors with the development of COPD. Much of the information derived comes from large epidemiological cross-sectional studies that provide associations but do not prove causation. In developed countries, smoking tobacco is the predominant risk factor. However, never-smokers also develop COPD and women predominate in this cohort. In places where solid fuels are burned, indoor air pollution is probably the dominant risk factor. While smoking is the most widely recognized risk factor, the causes of COPD are myriad, including both developmental and environmental risk factors, many of which are modifiable.

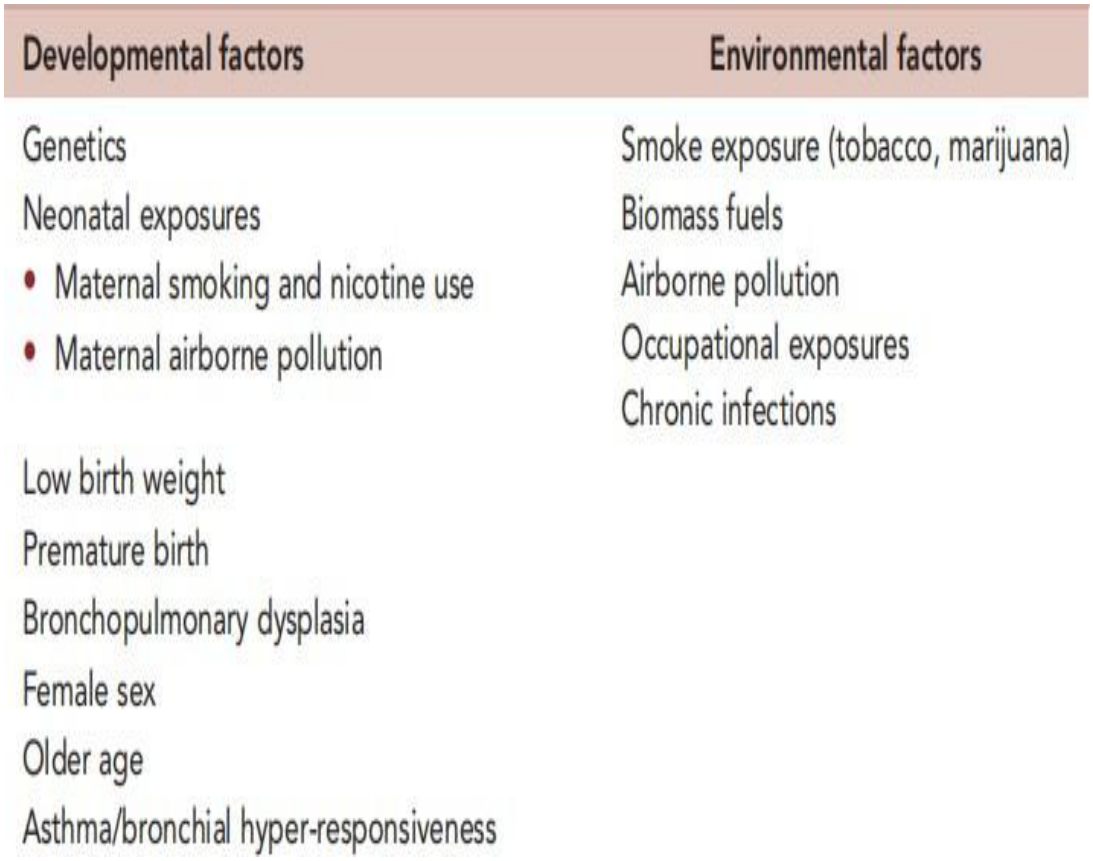

Figure 4: Risk factors for development of airflow obstruction.

Symptoms of Chronic Obstructive Pulmonary Disease

The clinical manifestations of COPD are

- Labored breathing and
- Shortness of breath,
- Cough,
- Hypoxemia, and
- Excessive mucous production.

Once the fundamental changes of COPD, they are irreversible, but individuals with COPD will intermittently experience acute exacerbations during episodes of lung infection. These episodes can be treated, but the underlying pathology remains unchanged.[4]

Though a breakdown of COPD into emphysema and chronic bronchitis is helpful, typically patients have features and findings of each and cannot be simply classified.

Table 1: Symptoms Associated With Copd.

EMPHYSEMA	*CHRONIC BRONCHITIS*
• Pink puffer" – type A • Severe constant dyspnea/tachypnia("puffing"): • Likely related to increasing end- expiratory volume (decreased recoil),making each breath less efficient. • Patients use accessory muscles (tripodposition) and breath faster (hyperventilation) to compensate for feeling of inadequate ventilation. • Mild cough: Irritation of the smallerairway can lead to the production ofcough. • Non cyanotic ("pink") • no hypoxemia. • Thin/cachexic: Loss of skeletal muscle and subcutaneous fat due to inadequate oral intake as well as high levels of inflammatory cytokines (TNF-α) that cause such wasting. • Diminished breath sounds onauscultation: • Hyperinflation of alveoli and destruction of alveolar architecturecauses decreased airway resistance.	• "Blue bloater" – type B • Copious sputum production: High amount of sputum produced by thegoblet cells. • Cough: Irritation of the cough receptors, by the mucous, in thesmaller and the large airways. • Cyanotic ("blue"): The mismatched V/Q defect leads to inadequate oxygenation of the blood; most prominent in the lipsand the nail beds. • Volume overload ("bloater"): Mostlikely from from the right ventricular (RV) failure, known as cor pulmonale. • Wheezy on auscultation: Due to airway obstruction. Compared to asthma, there is less bronchospasm and more mucus/hypertrophy in COPD. • Rhonchi is a gurgling sound that may be heard due to mucus hypersecretion in the airways.

Etiology of chronic obstructive pulmonary disease

Many factors contribute to the development of COPD, including genetic factors like alpha1- antitrypsin deficiency, occupational exposures to dusts and chemicals, pollution, respiratory infections in childhood and cigarette smoke.[5]

Table 2: Etiology of Chronic Obstructive Pulmonary Disease.

ETIOLOGY	*MECHANISM OF ACTION*
1. CigaretzteSmoke	• **Presence of smoke particles in the lungs leads to an inflammatory response with increased macrophage and neutrophil infiltration into the lungs.** • **These immune cells release cytokines, chemokines and elastases, which damages the lung parenchyma over time.**
2. Occupational Exposure TO Dust/Chemicals	▪ **Etiology unclear, however, is hypothesized to be a similarinflammatory response that damages the alveoli**
3. Alpha-1 antitrypsindeficiency	▪ **Alpha-1 antitrypsin is a serine protease inhibitor(SERPIN) secreted by the liver into the blood which inhibits the enzyme neutrophil elastase from damaging the lung tissue. Deficiency of this alpha-1 antitrypsin leads to unopposed elasteolysis (destruction of the elastin fibersin alveolar walls) and development of early emphysema. This is the protease-antiprotease** hypothesis **of emphysema development.**

Pathogenesis of Copd

Air flow limitation, the major physiologic change in COPD, can result from both small airway obstruction and emphysema. As described below, small airways may become narrowed by cells (hyperplasia and accumulation), mucus, and Fibrosis. Of note, activation of transforming growth factor β (GF-β) contributes to airway fibrosis, while lack of GF-β may contribute to parenchymal inflammation and emphysema[5].

Table 3: Pathogenesis Involved In Copd.

PATHOGENESIS	
EMPHYSEMA	**CHRONIC BRONCHITIS**
▪ The inflammatory response, mediated by neutrophils, macrophages and CD8+ T-cells, release inflammatory mediators and enzymes that damage the lung parenchyma. Proteases like elastase and matrix metalloproteinases (MMPs) released by these inflammatory cells break down the connective tissue of the alveolar walls and the septae. ▪ A loss of elastic recoil leads to diminished expiratory flow rates, air trapping and airway collapsing.	▪ Mucous gland enlargement, goblet cell hyperplasia and mucociliary dysfunction occur in larger airways, causing excessive mucus production and build-up reducing the airway lumen. ▪ Although these pathological changes inthe large airways, it appears thatthe major site of increased airway resistance is the small airways (≤2mm). Fibrosis and smooth musclehypertrophy may occur along with excess mucus production and cellularinfiltration in the peripheral airways.

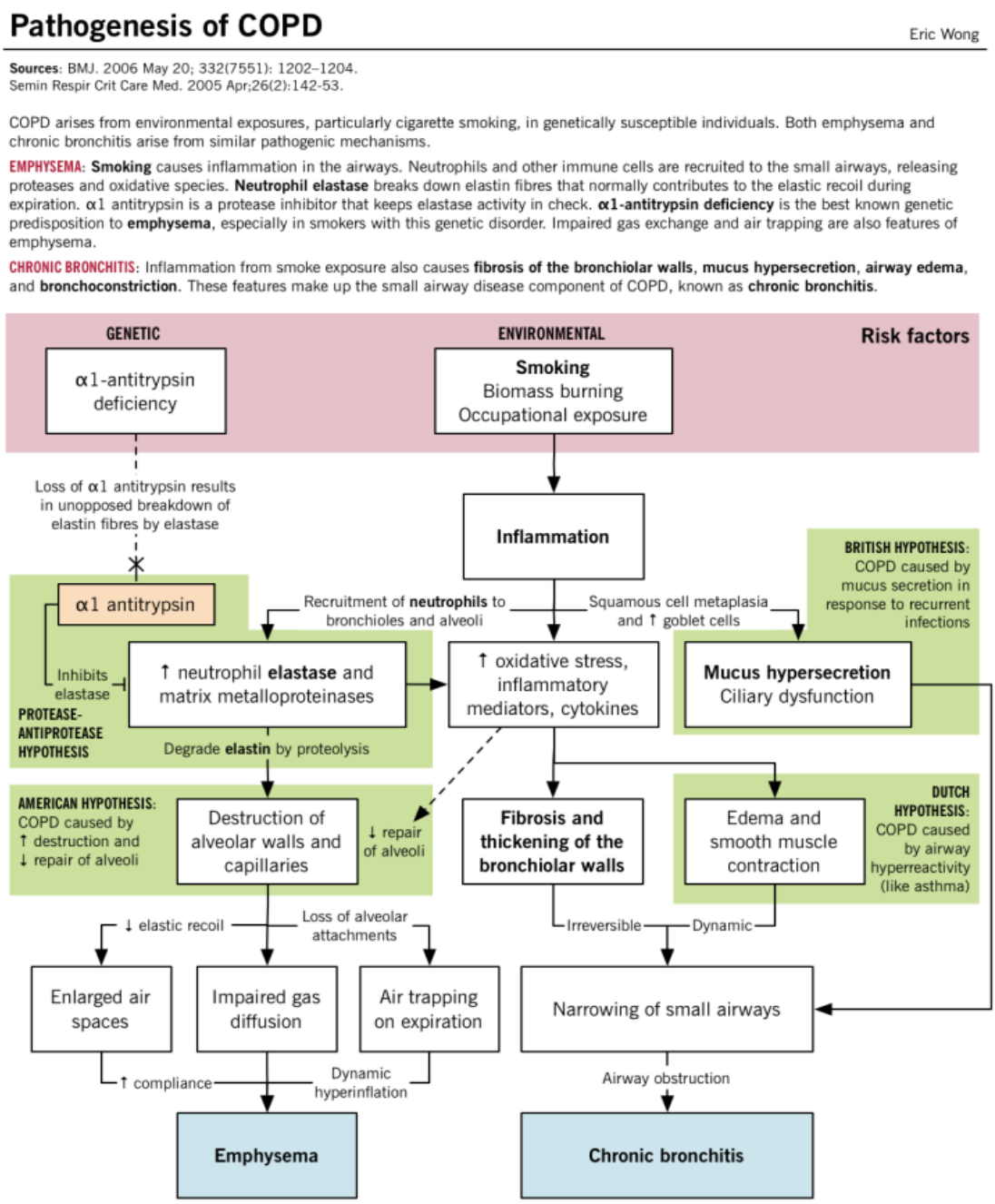

Figure 5: Flow chart of pathogenesis of copd.

Pathophysiology of Copd

COPD is a clinical description of two distinct pathologic disease processes: emphysema and chronic bronchitis. These two entities share similar risk factors and are often found to coexist in patients, providing a spectrum of disease.[3]

A persistent reduction in FEV1/FVC is the defining physiological feature of COPD.

1. Increased airway resistance,
2. Increased residual volume (RV),
3. Increased RV/total lung capacity ratio (RV/TLC),
4. Decreased inspiratory capacity,
5. Decreased maximum voluntary ventilation (MVV),
6. Abnormal distribution of ventilation, and
7. Ventilation–perfusion mismatching are also typical physiological features[1].

Persistent reduction in forced expiratory flow rates is the most typical finding in COPD. Increases in the residua volume and the residua volume/total lung capacity ratio, non-uniform distribution of ventilation, and ventilation-perfusion mismatching also occurs.[2]

Airflow Obstruction

- The low FEV1 and low FEV1/FVC in COPD are not reversible with inhaled bronchodilators, although small improvements are common, especially if responsiveness is tested with both ipratropium and albuterol. Thus, COPD differs from asthma, in which inhaled bronchodilators can

produce large improvements in FEV1.

- The FEV1 is the result of the balance between the elastic recoil of the lungs promoting expiratory flow and the resistance of the airways that limits flow during performance of an FVC.
- In normal lungs, as well as in lungs affected by COPD, maximal expiratory flow diminishes as the lungs empty because the lung parenchyma provides progressively less elastic recoil and the cross-sectional area of the airways falls leading to an increase in airway resistance.
- There is wide variability in COPD in the relationships between FEV1, exercise tolerance, and quality of life.
- Variability also extends to the relationship between the FEV1 and alveolar gas exchange. However, the PaO2 and oxygen saturation usually remain near normal until the FEV1 hasdecreased to about half of the predicted normal while elevation of the PaCO2 seldom occurs until the FEV1 is less than about one-fourth of the predicted. Thus, other causes of hypoxemia or an elevated PaCO2, such as the obesity hypoventilation syndrome, should be considered in patients with abnormal arterial blood gases and only mild to moderate COPD.
- Similarly, pulmonary hypertension and right ventricular failure do not occur unless COPD is severe and associated with chronic hypoxemia (PaO2 <55 mmHg).
- Diastolic dysfunction is common in the general population where COPD is prevalent, and should be considered when pulmonary hypertension is discrepant with COPD severity.

Abnormal Distribution of Ventilation and Ventilation–Perfusion Mismatching:

- Abnormal distribution of ventilation results from the heterogeneity of the pathologic process affecting airways and lung parenchyma.
- This heterogeneous ventilation results in ventilation–perfusion mismatching that is characteristic of COPD.
- Abnormality in the distribution of ventilation is evident in the pattern of nitrogen washout during breathing of 100% oxygen.
- The nitrogen washout is delayed because of regions that are poorly ventilated, and the shape of the nitrogen washout curve reflects compartments with different washout rates due to regional differences in compliance and airway resistance.
- Radioisotopic ventilation scanning with 133xenon also reveals regional heterogeneity of ventilation in COPD, but can also demonstrate the ability of airway mucus to trap xenon tracer.
- The multiple inert gas elimination technique (MIGET), which enables quantification of the ventilation–perfusion profile, has demonstrated different ventilation–perfusion patterns among patients with advanced COPD.

Hyperinflation

- Increases in total lung capacity, functional residual capacity (FRC), residual volume, and the residual volume to total lung capacity ratio (RV/TLC) are common in COPD.
- These abnormalities may be beneficial in that they help to preserve expiratory airflow by increasing lung elastic recoil and the cross-sectional areas of airway lumens.
- However, they have adverse effects.
- They displace the diaphragm into a flattened position causing a number of adverseeffects.
- In addition, they put the thoracic cage at a mechanical disadvantage so that inspirationrequires work rather than being passively assisted by the elastic recoil of the chest wall.
- These abnormalities of increased lung volume may increase further with exertion because reductions in airflow in diseased lungs reduce expiratory volume during rapid breathing.
- This phenomenon, called *DYNAMIC HYPERINFLATION*, adds to the workload on the inspiratory muscles while further reducing their mechanical advantage.
- Dynamic hyperinflation is an important mechanism of dyspnea with exertion in COPD

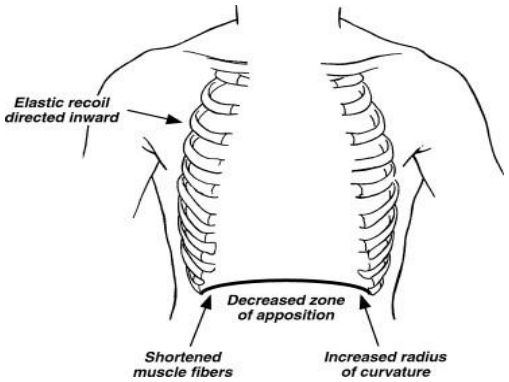

Figure 6: Effect of Hyperinflation in Copd.

Dyspnea

- People with COPD typically seek medical care because shortness of breath limits theiractivities and quality of life.
- Dyspnea is seldom a complaint until the FEV1 has fallen below about 60% of predicted.
- However, the correlation between FEV1 and exercise limitation is not strong.
- Some individuals are relatively free of dyspnea despite a severely reduced FEV1.
- Commonly, the discomfort associated with breathing is associated with inspiration ratherthan expiration.
- Measurement of dyspnea has proven complicated. A number of indices are in use.
- An increased sense of effort relating to the pressures needed from the respiratory muscles relative to their maximum pressure-generating capacity is thought to

be an important factor in causing the dyspnea associated with COPD.

Large (Central) and Small (Peripheral) Airways

- The large airways, which include the trachea and first generations of the bronchi, are a major site of inflammation and mucus hypersecretion.
- This results from anincrease in numbers (hyperplasia) and enlargement (hypertrophy) of the submucosal glands and mucus-producing goblet cells within the surface epithelium.
- Overproduction of mucus in the large airways results in a chronic productive cough, asobserved in chronic bronchitis, but this does not have a major impact on airflow limitation.
- Mucus hyper secretion coupled with impaired ciliary function reduces mucociliary clearance, increases the accumulation of secretions, and enhances the risk of bacterial colonization in an otherwise sterile environment.
- As a result, recurrent infections often occur owing to the inability to clear pathogens.

Inflammatory Cells and Mediators in Chronic Obstructive Pulmonary Disease(COPD)

NEUTROPHILS AND THEIR RESPONSE:
▪ ↑ in sputum of normal smokers. ▪ Further ↑ in COPD and related to disease severity. ▪ Few neutrophils are seen in tissue. ▪ May be important in mucus hypersecretion and through release of proteases
MACROPHAGES AND THEIR RESPONSE:
▪ Greatly ↑ numbers in airway lumen, lung parenchyma,and bronchoalveolar lavagefluid. ▪ Derived from blood monocytes that differentiate within lung tissue. ▪ Produce increased inflammatory mediators and proteases in patients with COPD inresponse to cigarette smoke and may show defective phagocytosis
T-LYMPHOCYTES AND THEIR RESPONSE:
▪ Both CD4+ and CD8+ cells are increased in the airway wall and lung parenchyma, with ↑CD8+:CD4+ ratio. ↑CD8+ T cells (Tc1) and Th1 cells that secrete interferon-γ and express the chemokine receptor CXCR3. ▪ CD8+ cells may be cytotoxic to alveolar cells, contributing to their destruction.
EOSINOPHILS AND THEIR RESPONSE:
▪ ↑ eosinophil proteins in sputum and ▪ ↑ eosinophils in airway wall during exacerbations.
Mediators
CHEMOTACTIC FACTORS AND THEIR REPONSE:
▪ Lipid mediators: e.g., leukotriene-B4 (LTB4) attracts neutrophils and Tlymphocytes. ▪ Chemokines: e.g., interleukin-8 (IL-8) attracts neutrophils and monocytes.
PROINFLAMMATORY CYTOKINES AND THEIR RESPONSE:
▪ Cytokines, including tumor necrosis factor-α (TNF-α),IL-1β, and IL-6 amplify the inflammatory process and may contribute to some of the systemic effects of COPD
GROWTH FACTORS AND THEIR RESPONSE:
▪ E.g., transforming growth factor-β (TGF-β) may induce fibrosis in small airways

Table 4: Inflammatory Response Mediators And Cells In Copd *Parenchymal Destruction*

- The terminal bronchioles lead directly to the alveolar ducts and sacs, the major site where gasexchange occurs.
- Emphysema is characterized by the destructive loss of alveolar walls and enlargement of theterminal airspaces, resulting in a loss of gas-exchanging surface area.
- In advanced cases, large, balloon-shaped bullous lesions may develop. Rupture of these bullae (pneumothorax) can lead to collapse of lung segments (pneumothorax).

Significant Comorbid Illness

- In the later stages of disease, chronic hypoxemia causes persistent vasoconstriction in the lung vascular bed, particularly the small pulmonary arteries.
- This can result in permanent structural alteration of the blood vessels, causing intimal hyperplasia and smooth muscle hypertrophy
- As COPD progresses, additional systemic consequences can arise, including:
- cachexia,
- skeletal muscle dysfunction,
- osteoporosis,
- depression, and
- anemia.
- The cause of these additional systemic disease

processes is not entirely clear but likely involves the dynamic interplay of progressive respiratory dysfunction, lung and systemic inflammation, side effects from medication use, and physical debilitation.

- In summary, although COPD is primarily a disease of the large and small airways and adjacent alveolar structures, it also includes important systemic consequences.
- The clinical consequences of the morphologic and pathophysiologic alterations include progressive dyspnea on exertion, chronic cough and sputum production, increased risk for respiratory infections, deconditioning, and an overall reduction in quality of life.

As chronic obstructive pulmonary disease has 2 major implications i.e., emphysema and chronic bronchitis the pathophysiology in both the conditions are summarised below

Table 5: Copd Associated Pathophysiological SymptomsIn Emphysema And Chronic Bronchitis.

PATHOPHYSIOLOGY	
EMPHYSEMA	*CHRONIC BRONCHITIS*
• **Parenchymal destruction:** Recurrent damage to the alveoli eventually leads toseptal destruction along with the capillary bed also. • **Matched V/Q defect**: Since both the terminal bronchioles and alveoli along with the capillary bed have been destroyed, a matched defect exists between the ventilation and perfusion; areas of low ventilation also have poor perfusion. • **Mild hypoxia:** Despite the "matched" V/Q defect, overtime hyperventilation develops and cardiac output (CO) drops which leads to areas of poor blood flow in relatively well oxygenated areas. Due to this poor CO, the rest of the bodysuffers from tissue hypoxia. • **Cachexia:** At the pulmonary level, the low CO leads to pulmonary cachexia; which induces weight loss and muscle wasting. This gives these patients the characteristic "**pink-puffer**" appearance	• **Smallairway inflammation:** Mechanisms discussed above lead to inflammation in the smaller bronchioles and mucus secretions further narrow the airway lumen. Despite this,the parenchyma are relatively less damaged. • **V/Q mismatch**: The physiologic responseleads to a drop in ventilation and compensation with the rise in CO. Increased perfusion in the areas of poor ventilation takes place eventually causing hypoxia and secondary polycythemia. • **Severe hypoxia and hypercarbia**: Chronic V/Q mismatch leads to decreased oxygenation/deoxygenation of the blood resulting in hypoxemia and increased CO2 retention (respiratory acidosis ensues). • **Pulmonary hypertension and cor pulmonale:** Chronic hypercapnia and respiratory acidosis lead to arterial vasoconstriction in the lungs. With the retrograde pressure build-up, the right ventricular pressures continue to rise and eventually causing RV failure. Otherwise,known as **cor pulmonale.**

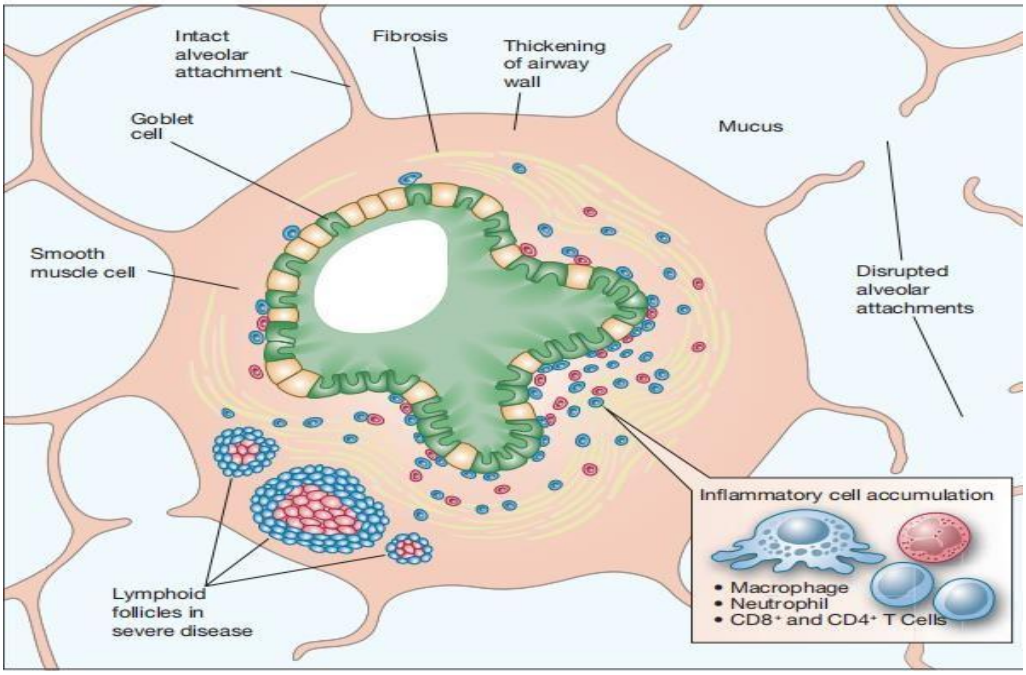

Figure 7: Pathologic Lesions in small airways in copd. Multiple abnormalities lead to partial obstruction of the lumen and altered shape and mechanical properties of the airways.

Diagnosis of chronic obstructive pulmonary disease

- The diagnosis of COPD is based on the presence of risk factors (generally smoking), clinical symptoms, and airflow obstruction on spirometric testing.
- Generally, individuals with COPD present in the sixth decade of life (or later) with symptoms of cough, wheeze, or dyspnea on exertion.
- Patients usually have at least a 10-packyear history (e.g., averaging one pack of cigarettes a day for 10 years) of cigarette smoking.
- Because the severity of COPD is related to the cumulative exposure to cigarette smoke, patients with more severe disease are likely to be older with a heavier smoking history.
- Cough and sputum production may be present for many years before airflow limitation develops, but not everyone with those symptoms will develop COPD. Dyspnea on exertion may not be present until the sixth or seventh decade.

Physical Examination

- Patients with early COPD may not exhibit any changes.
- Later, objective findings include the presence of a barrel chest (defined as an increase in the antero-posterior diameter of the chest), rales (defined as intermittent, nonmusical, brief crackle sounds), rhonchi (defined as continuous, musical high- or low-pitched sounds), prolonged expiratory phase, and cyanosis.
- Symptomatic patients may present with decreased breath sounds, wheezes, or slight rales on auscultation.
- In advanced disease, cyanosis, edema, intercostal retractions, and pursed lip breathing may be present.

Laboratory Findings

- Spirometry is the gold standard measurement in assessing and monitoring obstructive lung disease and is required for a diagnosis of COPD.
- Pulmonary function testing shows air flow obstruction with a reduction in FEV1 and FEV1/FVC
- In the evaluation of COPD, spirometry testing should be performed according to published guidelines, when the patient's condition is stable and after administration of two to four puffs (90mcg/puff) of albuterol by metered-dose inhaler (MDI).
- With worsening of disease severity lung volumes may increase, resulting in an increase in Total Lung Capacity, Functional Residual Capacity, and Residual volume.
- In patients with emphysema, the diffusing capacity may be reduced, reflecting the lung parenchymal destruction chracteristics of the disease.
- The degree of airflow obstruction is an important prognostic factor in COPD and is the basis for the Global Initiative for Lung Disease (GOLD) severity classification.

GOLD CRITERIA FOR SEVERITY OF AIRFLOW OBSTRUCTION IN COPD

GOLD STAGE	SEVERITY	SPIROMETRY
I	Mild	$FEV_1/FVC < 0.7$ and $FEV_1 \geq 80\%$ predicted
II	Moderate	$FEV_1/FVC < 0.7$ and $FEV_1 \geq 50\%$ but $< 80\%$ predicted
III	Severe	$FEV_1/FVC < 0.7$ and $FEV_1 \geq 30\%$ but $< 50\%$ predicted
IV	Very severe	$FEV_1/FVC < 0.7$ and $FEV_1 < 30\%$ predicted

Abbreviations: COPD, chronic obstructive pulmonary disease; GOLD, Global Initiative for Lung Disease.
Source: From the Global Strategy for Diagnosis, Management and Prevention of COPD 2014, © Global Initiative for Chronic Obstructive Lung Disease (GOLD), all rights reserved. Available from http://www.goldcopd.org.

Figure 8: Gold criteria for severity of airflowobstruction in copd.

Diagnostic Tests

- A plain chest X-ray is a useful means of excluding other diseases or diagnosing advanced emphysematous lesions and airway lesions, but it is not suitable for detecting early stage lesions.
- High-resolution computed tomography (HRCT) can

be effective as a means of earlydetection of emphysematous lesions.
- Emphysematous lesions appear as ill-defined low attenuation areas (LAA) on HRCT images, and thus can be distinguished from normal lung.
- HRCT is capable of detecting airway wall thickening.
- Assessment of emphysematous lesions and airway lesions based on HRCT images is also useful in phenotype classification of COPD.[7]

Prevention of chronic obstructive pulmonary disease

Therapeutic options for patients with COPD include the following:

- **Smoking cessation**

Has the greatest influence in improving the natural history of COPD. All patients who smoke should be strongly encouraged to quit. Pharmacotherapies for smoking cessation include the following

- **Nicotine Replacement Products**

Nicotine replacement therapy in any form (nicotine gum, inhaler, nasal spray, transdermal patch, sublingual tablet, or lozenge) is helpful in reducing the desire to smoke.

- **Smoking Prevention**

Efforts to encourage comprehensive tobacco-control policies and programs that have clear, consistent, and repeated nonsmoking messages have been developed by the U.S. Public Health Services and other health-care organizations.

Coordinated efforts to work with government officials to pass legislation to establish smoke-free schools, public facilities, and work environments and encourage patients to keep smoke-free homes are important and ongoing.

- **Occupational Exposure**

Primary prevention is emphasized by the elimination or reduction of exposure to harmful substances in the workplace.

Indoor and Outdoor Air Pollution

Avoidance of indoor air pollution from burning biomass fuel for cooking and heating in poorly ventilated dwellings is stressed. COPD patients are encouraged to monitor public announcementsof air quality and to avoid vigorous exercise outdoors or to remain indoors during pollution episodes.

Physical Activity

COPD patients should be informed of the benefits from regular exercise. Exercise conditioning programs are an integral part of pulmonary rehabilitation.

General Management of Chronic Obstructive Pulmonary Disease

- The overarching goals of COPD management involve two main principles.
- First, to reduce symptoms (i.e., relieve symptoms, improve exercise tolerance, and improve health status); and
- second, to reduce risk (i.e., prevent disease progression, prevent and treat exacerbations, and reduce mortality)
- Unfortunately, the only interventions that have been proven to reduce mortality in COPD are smoking cessation, oxygen therapy for patients with severe hypoxemia at rest, and lung volume reduction surgery for very select patients with advanced emphysema.
- As such, many of the interventions are aimed at alleviating symptoms and maximizing quality of life.
- Immunizations provide protection against serious illness and death in patients with COPD.
- The efficacy, benefit, and cost-effectiveness of vaccination against influenza among this population are significant.
- In addition, the pneumococcal polysaccharide vaccine is recommended to all patients with COPD, as well as, in adult patients through 64 years of age who smoke cigarettes or have asthma.

Non-Pharmacological Treatment

- *Pulmonary rehabilitation* is an exercise-based program aimed at maximizing the patient's functional status and quality of life. Multiple studies have now documented the beneficial effects of pulmonary rehabilitation, particularly with respect to improved exercise tolerance and alleviation of dyspnea.
- In addition, cost-effective analysis suggests that pulmonary rehabilitation programs are very cost-effective.
- Pulmonary rehabilitation programs are multidisciplinary programs typically running for 6to 12 weeks, two to three sessions per week.
- A number of interventions are used, including breathing retraining, psychosocial counseling, education, dietary counseling, and airway clearance techniques for patients with chronic sputum production.
- Arm strengthening and arm endurance exercises are important because patients with COPD commonly have excessive dyspnea when using their upper extremities.
- The most important component of a pulmonary rehabilitation program is lower extremity endurance training, often using a treadmill or bicycle ergometer.
- Because the large muscle groups have diminished oxidative capacity, likely related to de-conditioning and chronic inflammation, patients with COPD convert to anaerobic metabolism at low levels of exercise.
- This leads to increased lactate production for a given level of activity and, subsequently, increased CO2 production.
- Lower extremity endurance training can

significantly improve mitochondrial oxidative capacity in patients with COPD.

Pharmacotherapy

The fundamental goals of medication use are to prevent or control symptoms, reduce the frequency and severity of exacerbations, and improve both health status and exercise tolerance. The currently available medications used to treat COPD, however, do not alter the natural course of this condition; therefore, pharmacotherapy should be individualized for each patient and focused on symptom management to improve quality of life.

Drugs Used In Treatment Of Copd

1. **Bronchodilators:** short-acting and long-acting β2-agonists, short-acting and long-acting anticholinergic agents, and theophylline, methylxanthines.
2. **β-Adrenergic Agonists**

i) Short-acting beta agonists (SABAs) include albuterol (salbutamol), levalbuterol, terbutaline, and fenoterol.

ii) Long-acting β-agonists (LABAs) include Salmeterol, Formoterol, Arformoterol, Indicaterol, Vilanterol.

3. **Anti-muscaranics**

i) Short-acting muscarinic antagonists (SAMAs) include ipratropium and oxitropium

ii) Long-acting muscarinic antagonists (LAMAs) include tiotropium and aclidinium.

4. **Methylxanthines**

i) This class includes Theophylline.

5. **Phosphodiesterase-4 Inhibitors**

i) Roflumilast, Cilomilast

6. **Corticosteroids**

i) Inhaled corticosteroids

ii) Systemic corticoids

7. **Mucolytics and Antioxidants**

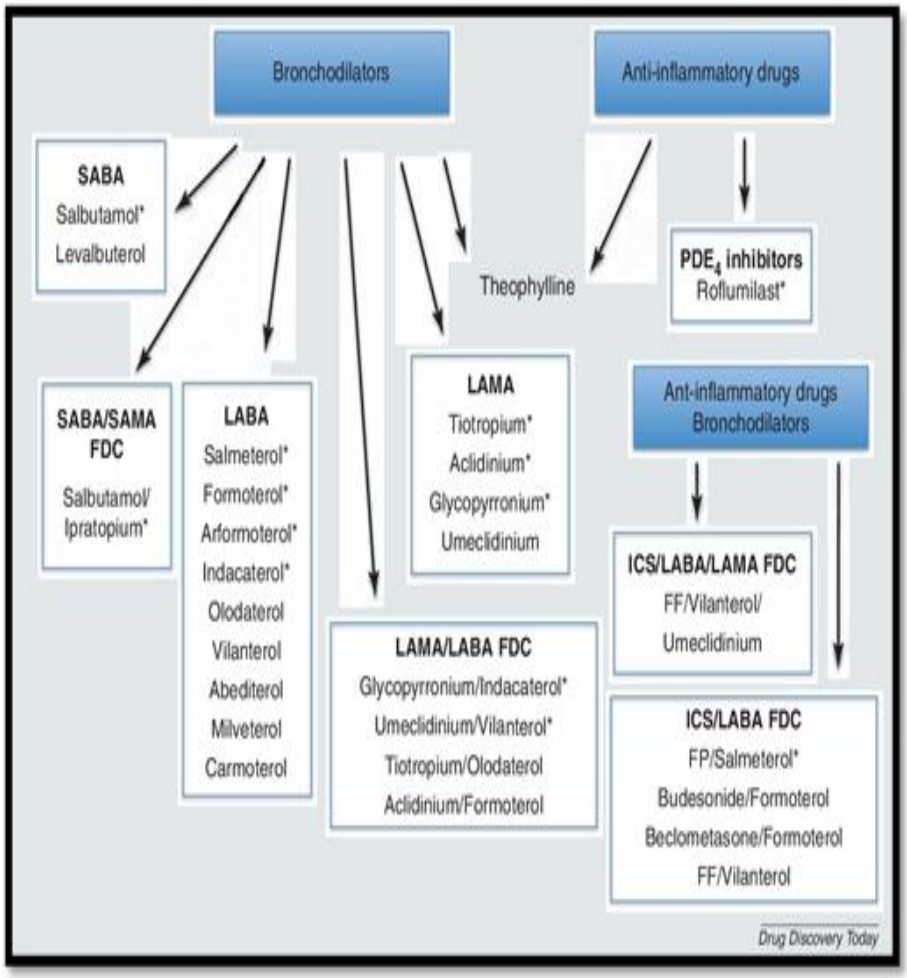

Figure 9: Pharmacotherapy of Copd.

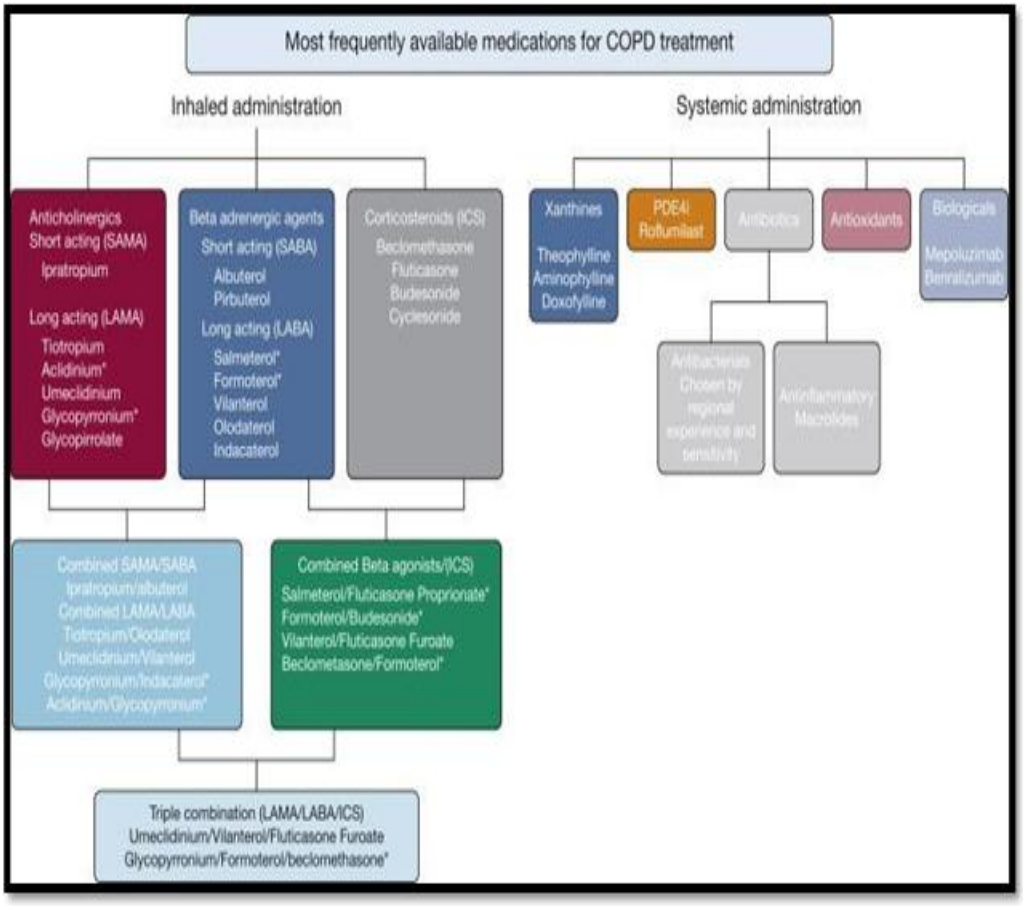

Figure 10: Medications For Copd.

Bronchodilators

1. Drugs that relieve bronchial obstruction by reducing bronchial smooth muscle contraction are called bronchodilators. Usually, they improve spirometric values reflecting obstruction such as FEV1.
2. These compounds generally improve also emptying of the lungs and reduce air trapping (dynamic hyperinflation/restriction) both at rest and during exercise.
3. These effects cannot be predicted based on the ability of the particular compound to improve FEV1.[10-11]
4. The dose–response effect of all bronchodilators at the currently used doses is relatively flat, which means that a small increase (e.g. doubling) in the dose is not expected to produce a vast increase in the bronchodilatory action.[8,9]
5. The adverse effects are generally dose-related. Increase in the dose of short-acting inhaled β2-agonist and anticholinergic, especially when given nebulized, may relieve subjective dyspnoea in acute setting during an exacerbation of COPD but may not help asa long-term therapy.

Short- And Long-Acting B2-Agonists (Saba, Laba)

The main beneficial effect of β2-agonists is the reduction of bronchial smooth muscle contraction that leads to relief of bronchial obstruction. The duration of the effect of short-acting β2-agonists is usually 3–6 hr. Short-acting β2-agonist used either as-needed or regularly reduce symptoms of COPD and improve lung function.[12]

Short- And Long-Acting Anticholinergics (Sama, Lama)

- Anticholinergic compounds block muscarinic receptors (M1, M2 and M3), thus antagonizing acetylcholine-induced bronchial smooth muscle contraction.
- The duration of the effect of short-acting anticholinergic (ipratropium) is usually somewhat longer (even up to 8 hr) than that of the short-acting β2-agonists (3–6 hr), but starts more slowly.[8,9]
- The effect of long-acting anticholinergics lasts either 12 hr (aclidinium) or approximately 24 hr (glycopyrronium, tiotropium or umeclidinium). Of these, tiotropium has been most extensively studied and used.
- The bronchodilatory action of aclidinium and glycopyrronium starts sooner than that of tiotropium.
- Tiotropium improves lung function and quality of life and reduces symptoms and exacerbations of COPD (A).[13]
- In contrast, tiotropium does not affect the progression of the disease as judged by the annual

decline in FEV1.[14]

- Tiotropium may be more effective than salmeterol in reducing exacerbations of COPD.[15]
- Both aclidinium and glycopyrronium have been shown to induce bronchodilation, improve lung function and quality of life and reduce the need for rescue medication 16-17, and their efficacy roughly equals to that of tiotropium. Aclidinium, glycopyrronium and umeclidinium have been shown to reduce COPD exacerbations in studies lasting up to 1 year 18-19, but long-term studies lasting more than 1 year, similar to those made with tiotropium , are still lacking.

Inhaled Glucocorticoids

- In the treatment of asthma, the therapeutic and adverse effects of ICS depend on the doseused.[20]
- Instead, in the treatment of COPD, the dose dependency of the therapeutic and adverse effects of ICS is not known.[21-22]
- In long-term trials, only moderate and high doses of ICS have been used
- Regular long-term (>6 months) therapy with ICS in COPD reduces exacerbations and slows down the decline in the quality of life.
- Generally, patients with mild disease and without previous exacerbation history do not benefit from ICS
- The response to ICS in COPD cannot be foretold from the response to oral glucocorticoids or by measuring hyper-reactivity or response to bronchodilators (bronchodilator test in spirometry)
- Discontinuation of ICS may precipitate exacerbation of the disease in some patients with COPD but may be safely performed in others to decrease risk of long-term adverse effects.
- ICS alone do not affect mortality due to COPD or the rate of decline of lung function (annual FEV1 decline)

Prescription Pattern of Drugs In Patients With Chronic Obstructive Pulmonary Disease In Hyderabad Metropolitan

Aim of Study

To analyze the prescriptions for rational prescribing to the patients suffering from chronic obstructive disease in Hyderabad Metropolitan.

Objective of Study

To describe rational drug prescribing in general practice for a COPD patients, using patients age, sex, encounter and the occurrence of some predefined inappropriate drug prescribing.

LITERATURE REVIEW

Rational use of Drugs

- WHO defined the rational prescribing of drugs as "patients receive medications appropriateto their clinical needs, in doses that meet their own individual requirements, for an adequate period of time, and at the lowest cost to them and their community".
- More than 50% of all medicines worldwide are prescribed, dispensed or sold inappropriately and 50% of patients fail to take them correctly. It has been estimated that fifty percent of the medicines being used in the country (India), either on prescription or in over the counter salesare inappropriately or irrationally used. The irrational use of medicines has an adverse impacton the outcome of therapy and cost, and may cause adverse reactions or negative psychosocial impacts.
- During the past few years, the pharmacy profession has expanded significantly in terms of professional services delivery and now has been recognized as an important professional in the multidisciplinary provision of health care
- But the pharmacists are in a strong position to promote the rational use of medicines because of their extensive knowledge of medicines and good communication skills. The WHO has recommended a special role for pharmacists, particularly in Quality Assurance and in the safeand effectiveadministration of medicines

The Concept Of Rational Drug Use

The aim of any medicine management system is to deliver the right medicine to the patient who needs the medicine and the steps of selection, procurement, and distribution are necessary precursors to the rational use of medicines.

The Conference of Experts on the Rational Use of Drugs, convened by the World Health Organization (WHO) in Nairobi in 1985, defined rational use as follows:

"The rational use of drugs requires that patient receive medications appropriate to their clinical needs, in doses that meet their own individual requirements, for and adequate period of time, and at the lowest cost to them and their community."[23]

Reasons For Irrational Use of Drugs

There are several factors that influence the irrational use of medicines. Some of majorreasons are listed below:

1. Lack of information
2. Faulty & inadequate training & education
3. Poor communication between health professional & patient
4. Lack of diagnostic facilities/Uncertainty of diagnosis
5. Demand from the patient
6. Defective drug supply system & ineffective drug regulation
7. Promotional activities of pharmaceutical Industries

Role Of Pharmacist In Promoting Rational Drug Use

In terms of modern health care delivery, studies have shown that engaging multidisciplinary expertise is one of

the goals for achieving ultimate population health. Although the pharmacy profession is recognized for its Importance as a health care provider in many developed countries, in most developing countries it is still underutilized. Though all health care provider and the public are involved in the rational use of medicine, the WHO has recommended a special role for pharmacists, particularly in Quality Assurance and in the safe and effective administration of medicines. Pharmacists are in a strong position to promote therational use of medicine because of their extensive knowledge of medicines and good communication skills. Pharmacists, particularly those in community pharmacies, play a key role in instructing patients in the correct use of medicines. Pharmacists have the following important role to play in promoting rational use of medicines.

A. Medicine procurement

The selection and range of medicines to be recommended by the pharmacist to clients can be based on the essential medicines concept. The escalating cost of medicines is a majo0r problem in India. Pharmacists should recommend affordable and quality medicines.

- Procure the most cost effective medicines in right quantities
- Select reliable suppliers of high quality products
- Select reliable suppliers of high quality products
- Ensure timely delivery
- Achieve lowest possible total cost

B. Inventory control

Pharmacies should maintain adequate stocks of essential medicines (including those that are not often required, but are critical and life saving) and minimize 'out of stock' Situations by adopting good inventory control measures.

C. Information and Education (Health Care Providers)

The pharmacist should interact with other healthcare providers and promote rational medicines in spite of pressure to do so otherwise, from both patients and company representatives. They must also inform clients about policy changes, specific warnings/ banning of medicines, non-availability of medicines, and medicines related problems. Any adverse drug reactions noted should be reported to appropriate monitoring (Pharmacovigilance) centers in the region or in the country.

D. Public Education

- Patient or consumer education plays an important role in promoting rational use of medicines.The pharmacist can play a key role in patient education to promote the rational use of medicines.
- The pharmacist can perform the following functions:-
- Educate the community about rational medicines,
- Hazardous and useless medicines, and alternative remedies, using patient information leaflets, posters, slide shows, public meetings, articles and letters in the local newspapers, publication in local languages, etc
- Form an action committee of concerned groups and individuals to create awareness ofthe situation, and bring about changes.
- Encourage local health workers to recommend medicines only when necessary, & to
- suggest appropriate home remedies for trivial complaints
- Help facilitate monitoring of continued sales of banned medicines.
- Educate the community about the irrationality of certain medicines[23].

Steps to Improve Rational Drug Prescribing

- ***Step 1***: Identify the patient's problem based on symptoms and recognize the needfor action.
- ***Step 2***: Diagnosis of the disease: identify underline cause and motivating factors.This may bespecific as in infectious disease or non-specific.
- ***Step 3***: List possible intervention or treatment: this may be non drug treatment or drug treatment. Drug must be chosen from different alternatives based on efficacy. Convenience and safety of drugs including, drug interactions and high risk group of patients.
- ***Step 4***: Start the treatment by writing an accurate and complete prescription e.g. name of drug with dosage forms, dosage schedule and total duration of the treatment.
- ***Step 5***: Give proper information: instruction and warning regarding the treatment.
- E.g. side effects (ADR's), dosage schedule and dangers or risk of stopping the therapy suddenly.
- ***Step 6***: Monitor the treatment to check, if the particular treatment has solved the patient's problem. It may be: Passive monitoring- done by the patient by himself. Explain him what to do if thetreatment is not effective or if too many side effects occurs.
- Active monitoring done by physician and he make an appointment to check the response of the treatment.

Rational Prescribing for Chronic Obstructive PulmonaryDisease

The complexity of COPD treatment has increased over the last 5 years mainly because of the proliferation of drugs and delivery devices.

Principles of Rational Therapy

The treatment for COPD should

- Be sustained: COPD is associated with fixed structural changes within the lungs which produce persistent physiological abnormalities. Treatment that mitigates these problems intermittently is likely to be less effective than treatment that covers the 24-h day as was seen with bronchodilatordrugs[24]
- Be safely administered: in general this means that

the inhaled route is preferred given the lower total dose of drug required to be effective. This is not possible for all drug classes, for example, phosphodiesterase IV inhibitors which are ineffective when given by inhalation[25].

- Be complementary in their actions when combined: this usually involves drug with a different mechanism of action and either a common target, for example, β-agonist and antimuscarinic bronchodilators or different targets that are at least additive in effect, for example, long-acting bronchodilators and inhaled corticosteroids (ICS). To date the choice of inhaler does not seem to influence the efficacy or safety of treatment. Initial concerns about tiotropium therapy via soft mist inhalers have been addressed.
- It is possible that the need to take treatment less often improves adherence, although has yet to be conclusively established in COPD.
- Optimize the risk–benefit to patients. Although COPD treatment is generally very safe, it is reasonable to select therapy with a lower risk profile provided it is equally efficacious in other ways.
- Apply previous knowledge when selecting therapy: there is already consensus that some drugs should not be used as monotherapy, for example, ICS or PDE-IV inhibitors used as the only maintenance treatment.
- Rational therapy for any disease, not least COPD must be evidence based, and there is no shortage of clinical trial data defining the efficacy of therapy across a range of end points. Sadly that is not matched by reliable effectiveness data in so called 'real world' settings but given this limitation, we can begin to see some potentially useful approaches to drug therapy. The GOLD approach is a good starting point. For the patient with abnormal spirometry but relatively infrequent symptoms, short-acting inhaled bronchodilators would normally suffice (although good data about the frequency of this clinical presentation are lacking). Certainly, bronchodilators can improve lung mechanics but not necessarily exercise endurance even in mildCOPD[26].
- For patients with more persistent symptoms and especially breathlessness and exercise impairment monotherapy with a LAMA is a good starting point. A LABA might be considered if there are contraindications to LAMA use or the patient wishes to try a different drug. Combiningthe two drugs in a single inhaler is a potential but more expensive option for those with troublesome symptoms, but until better evidence for effectiveness becomes available, this should not be first line therapy. Patients of this type commonly have cardiovascular comorbidities.
- For the patient with severe disease, marked dyspnoea and exacerbations either LABA/ICS or LAMA can be used initially although the LAMA or LAMA/LABA would be preferred initially if there is a history of one or more episodes of pneumonia. Commonly whichever treatment has not been selected is then added in and if the patient still exacerbates or has been hospitalized then it is worth considering roflumilast where it is available.
- This is one scheme which adheres to the principals of rational therapy already outlined. Several others are possible and individual clinicians viewing the evidence may well draw different conclusions from it. Many other factors will play a role in the final decision including considerations of convenience, the complexity of other therapies to be managed and cost

Prescription Writting

- Prescription is one of the most important therapeutic transaction between physician and patient .The word prescription‟ derive from pre (before) and script‟ (writing written) which denotes that it is an order that must be written down before or for the prescription and administration of drug. Commonly the term prescription is used to mean an order to take certain medication.
- A prescription (Rx) is defined as a health care program implemented by a physician in the form of instruction that govern the plan care for an individual patient.
- The prescription symbol (Rx) currently in use is an ancient symbol which was established centuries ago. It signifies the specific latin verb recipe of the medication and the direction for taking it. The essence of a good prescription writing is to ensure that the prescriber is able to understand exactly which drug formulation and dosage to dispense, and the patient has clear written instructions for self-administration of the prescribed drug.
- Prescribing is also used to describe certain activities which include delivery of medicines and devices. It is used to describe written information provided for patients or any advice. It generally does not matter if you write the generic or the brand name here unless youspecifically want to prescribe the brand name. After you write the medication name, you need to tell the pharmacist the desiredstrength.

Type of Prescribing

There are two types of prescribing based on approach of prescriber, one is Rational (Appropriate) and another approach is irrational (Inappropriate) prescribing

Methodology

Study Design: In different clinics and hospitals of Hyderabad a prospective and observational study was undertaken.

Study criteria: The prescriptions of 25 Chronic Obstructive Pulmonary Disease suffering patients were collected from different clinics and hospitals. This study was based on the collection of prescriptions of patients to know the prescribing pattern of drugs used to treat COPD in the patients. The study includes the data collected from the prescriptions of patients of different

years of age group of both the genders.
To determine the prescribing pattern, a basic indicator of drugs were selected. The information of patient characteristics (gender, age and disease), drugs prescribed (dosage form, dose, nature of drug, frequency of administration, route of administration)

Data Source: The prescriptions of Chronic Obstructive Pulmonary Disease patients from different clinics and hospitals of Hyderabad were used for the study. These prescriptions of patients of different age group were collected randomly to avoid bias.

Data Management: Each prescription was analysed, the drugs and their therapeutic agents were recorded. The percentage of generic names of the drugs, the percentage of prescribed drugs encountered and the route of administration based prescribing pattern of the drugs were specifically recorded and grouped into their respective classes. The results were presented by means of statistical methods.

Statistical Analysis: The data obtained was statistically analyzed for all the above parameters. The results obtained were represented in numbers, percentage and graphs using MS Excel. They were computed using pie chart, bar diagram and in tabular form.

Materials study Design
A retrospective study on rational drug prescribing pattern of drugs used to treat COPD was carried out using prescriptions issued to the copd suffering patients, attending the outpatient and inpatient department of various hospitals and clinics of Hyderabad as follow. The prescriptions were collected for a period of 3 months.

1. Care Hospital, Banjara Hills.
2. Owaisi hospital & Research Centre, Kanchanbagh
3. Princess Esra hospital,
4. Sunshine hospital, Gachibowli road, Hyderabad
5. Yashoda hospital, Malakpet, Hyderabad.
6. Art center government chest hospital, Mehdipatnam, Hyderabad.
7. Dr.Rafi's City Chest Clinic, Mehdipatnam, Hyderabad.

Sample Size and Collection
A total of 25 prescriptions for patients suffering from COPD of all age groups emanating outpatient and inpatient departments of hospitals and clinics were consequently selected.

Procedure for Calculating Prescribing Indicators
Average number of drugs per encounter was calculated by dividing the total number of drugs prescribed by the number of prescriptions surveyed. Percentage of encounters with a drug and inhalers prescription was calculated by dividing the number of patient encounters during which a drug or inhaler was prescribed by the total number of encounters surveyed, which was then multiplied by 100 respectively. Percentage of drugs prescribed to different age groups was determined by dividing the number of productsprescribed from the essential drug list by the total number of drugsprescribed, and then multiplied by 100.

RESULTS

A retrospective study was performed to determine the drug prescribing patterns and to determine if prescribing could be rationalised without treatment failure. Following arethe results acquired by the study:

1) Gender
Totally 25 prescriptions collected on a random basis were analyzed. Of the 25 prescriptions 10 were male and 15 were female patients. (The reasons for the greater susceptibility of women to developing COPD with small airway disease predominance are largely unknown. The airways of females are relatively smaller than those of males for the same lung volume, so there may be a greater concentration of tobacco smoke per unit area of small airway surface).

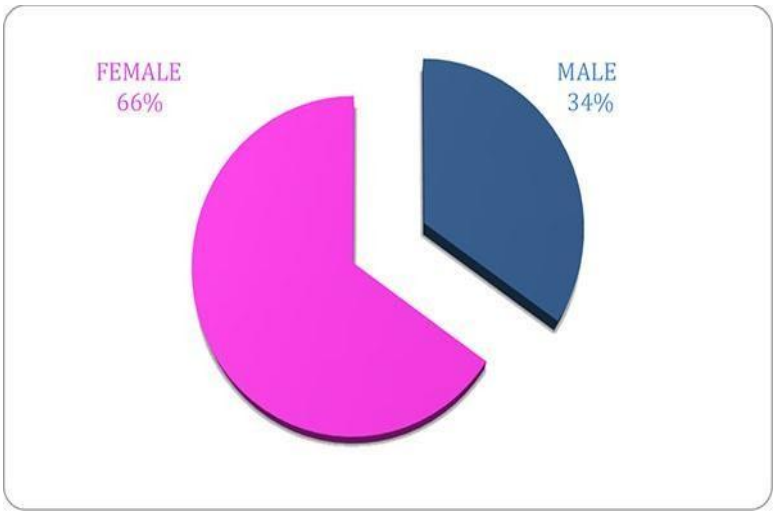

Figure 11: Pie Diagram Showing % Of Copd Patients (N = 25) InTerms Of Gender.

2) AGE

On analysis it was found that 6 patients (19%) were under 45-50 age group each, 3patients(9%) were under 51-55 age group, 10 patients (40%) were under 56-60 age group, 8 patients (23%) under 61-65 age group, 4 patients (16%) were under 66 –70 and 1 patient (4%) under 71-75.

Demographic Characteristics of the COPD Patients (N = 25) in years.

Table 6: Demographic features of COPD patients.

AGE (YEARS)	Number of Prescriptions	Percentage %
45-50	6	19
51-55	3	9
56-60	10	40
61-65	8	23
66-70	4	16
71-75	1	4
Total	**25**	

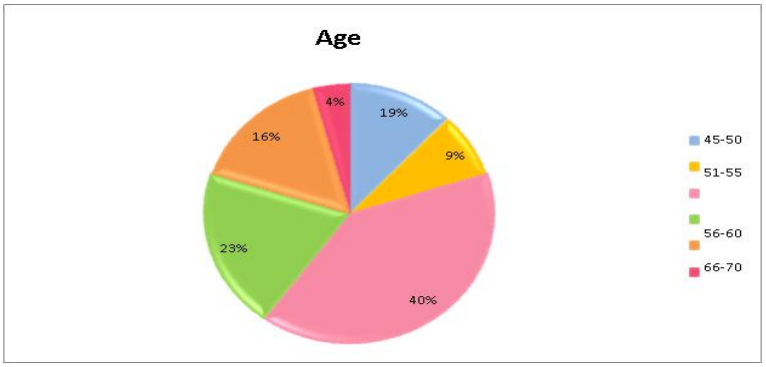

Figure 12: Pie Diagram Showing The Copd Patients (N = 25) In Terms Of Age (Years).

3) Generic and brand name

All the 25 (100%) prescriptions were mixed which constituted of bothgeneric and brandnames.

Table 7: Drugs Prescribed In Patients And TheirPercentage.

Name of the drug	Number of prescription	Percentage %
Antibiotics	20	80
Short-acting beta 2agonist	11	44
Anticholinergic agent	13	52
Inhaled corticosteroids	5	20
Methylxanthines	6	24
Systemic corticosteroids	3	12
Anti-histaminics	1	4
Leucotriene receptorantagonist	1	4
Long-acting beta 2 agonist	6	24

Charecteristics of study population n=25

Table 8: Charecteristics of Population

Variables	Mean (SD) or percentage
Females	56.36
Males	43.63
Age (in years)	66.9
Active smokers	71.42
Ex-smokers	17.85
Passive smokers	73.21
Quality of life scores	
10-20	12.72
21-30	56.36
31-40	30.90

Drug utilization of antibiotics in COPD patients.

Table 9: Drug Utilization of Antibiotics in Copd.

Type of antibiotic	Percentage
Amoxycillin- clavulanic acid	35.71
Ceftriaxone	35.71
Azithromycin	17.85
Levofloxacin	17.85
Cefpodoxime	8.9
Amikacin	7.14
Cefixime	5.35
Metronidazole	3.57
Clindamycin	1.78
Doxocycline	1.78
Gentamicin	1.78

Table 10: Core Drug Use Indicators.

Core drug use indicators	
Average number of drugs per prescription	7 drugs
Percentage of drugs prescribed by genericname	07.38%
Percentage of encounters resulting inprescription of anantibiotic	88.70%
Percentage of encounters resulting inprescription of an injection	55.30%
Percentage of drugs prescribed fromessential drugs list orformulary	43.90%

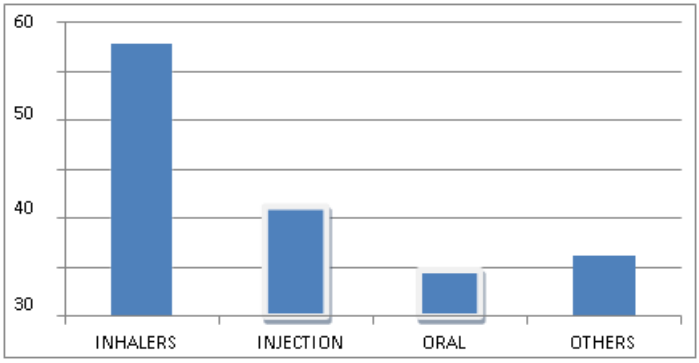

Table 11: Dosage Forms Used And Its Percentage.

Summarising the Drug Therapy Prescribed

- Majority of the patients were receiving either oral or inhalational β-agonists. Inhalational short-acting beta-2 agonists salbutamol and levosalbutamol were prescribed in accordance with the GOLD guidelines, which quickly relieves the symptoms of patients.
- Other long acting beta-agonists like formetrol (26.78%) were also prescribed. Systemic beta 2 agonists terbutaline was also prescribed.
- Parenteral steroids were used in 25% of the patients which constitutedhydrocortisone. Steroid inhalers were used in 67.85% of patients with all of them receiving budesonide. These are in accordance with the GOLD guidelines, as on one hand, steroids decrease the recovery time and hospital stay andat the same time improve lung function. However, anotherstudy showed more use of parenteral steroids than inhalational ones.

DISCUSSION

- Based on the data of the present study, COPD was found to be more common among females rather than males. It might be due to the fact that as the prevalence of smoking in females has grown progressively. Indeed, the increased morbidity and mortality from COPD during the last few decades is partly driven by their increase in women.
- People of age group 61-70 years were found to be more affected.
- The age distribution supported the evidence of the previous studies that only those above 40 years of age were admitted during the period of the study.
- Most of the patients in the present study had co-morbid conditions (n=86; 76.78%).
- The most commonly observed co-morbid conditions were related to GIT followed by cardiovascular system.
- Many patients were on Anti- depressants
- About 10.71% patients were on DOT (directly observed therapy) for tuberculosis. These findings are almost similar to the result of previous study in which 69.8% patients had co-morbid conditions.
- In our study, all the patients were receiving combination therapy.

- This prescribing trend may be attributed to achieve thegoals of COPD therapy including minimization of symptoms, preventing recurrent exacerbations, reducing the need for hospitalizations and maintaining the pulmonary function.
- Out of the patients receiving methylxanthines, 64.28% were receiving hydroxyethyl theophylline (dyphylline) and the remaining aminophylline. As per the GOLD (global initiative for obstructive lung disease) criteria and reviews of cochrane, patient presenting with COPD is to be initially managed with β-agonists followed by anticholinergics.
- Due to the alleged adverse effects associated with methylxanthines, their role remains controversial. In this study, the use of methylxanthines was found to be high and the usage of anticholinergics was on the lower side.
- This might be due to easy availability of the methylxanthines and relative non availability of anticholinergics in the hospital of study.
- Majority of the patients were receiving either oral or inhalational β-agonists. Inhalational short-acting beta-2 agonists salbutamol and levosalbutamol (58.9%) were prescribed in accordance with the GOLD guidelines, which quickly relieves the symptoms of patients. Other long acting beta-agonists like formetrol (26.78%) were also prescribed. Systemic beta 2 agonists terbutaline (62.5%) was also prescribed. Parenteral steroids were used in 25% of the patients which constituted hydrocortisone. Steroid inhalers were used in 67.85% of patients with all of them receiving budesonide. These are in accordance with the GOLD guidelines, as on one hand, steroids decrease the recovery time and hospital stay andat the same time improve lung function. However, anotherstudy showed more use of parenteral steroids than inhalational ones.
- Anticholinergics were used in 46.42% of patients in which 92% were on ipratropium.
- Their use is supported by the previous study in which addition of tiotropiumresulted in a significant reduction in exacerbation frequency.
- However, in our study, short-acting anti- muscarinic agent ipratropium was used more frequently than the long–acting tiotropium.

CONCLUSION

- Through this study on COPD patients, it was found that in present scenario female patients are more being affected by copd than male patients.
- The majority of the drugs prescribed were in accordance with the global initiative for chronic obstructive lung disease (GOLD)guidelines.
- In this study, the major risk factor of the disease was found to be smoking, so patients should be counselled to quit smoking and use of masks can be promoted to prevent inhaling of noxious particles.
- Influenza vaccination should be considered for all patients of COPD and Pneumococcal vaccination recommended for patients ≥65 years.
- The drugs were prescribed in accordance with the respective GOLD criteria and questions were answered regarding the prescribing of syrups and it was responded as a laxative medicine for treating constipation and use of injection Pan for treating Acidity and serofol inhaler to treat chest tightness and wheezing, cough etc.
- However the mean number of drugs per encounter was high, which increased the total cost as well as drug interactions which can lead to adverse reactions and finally an irrational prescribing pattern, so the prescribers and the pharmacist must be cautious while prescribing and dispensing the medications with minimum possible ADR's to patients and cost-effectiveness and awareness programs must be called on for the patients regarding safe use of medications, non-pharmacological therapies by modification of lifestyle pattern, and also Seminars and Programs which help the prescribers and Pharmacist to detect and assess the drug therapy related issues.

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