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# Diagnostic and Management Approach to Chronic Obstructive Pulmonary Disease

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***ABSTRACT:****COPD is a significant cause of morbidity and mortality worldwide, with over 300 million people are affected or nearly 5% of world population, and killing 3 million people. Fourth leading cause of death in the United States.Risks factors include tobacco smoking, air pollution, and workplace exposure to dust, chemicals, and fumes, genetic playing a smaller role.Primary risk factor for COPD globally is tobacco smoking.Diagnosis of COPD by lung function after the use of bronchodilators. Diagnosis need to be differentiated from other causes of shortness of breath such as congestive heart failure, pulmonary embolism, pneumonia or pneumothorax. The distinction between asthma and COPD is made on the basis of symptoms, smoking history, and whether airflow limitation is reversible with bronchodilators. Prophylactic antibiotic therapy may have some use for highly selected patients with frequent exacerbations. Treatment and prevention of COPD is smoking cessation.*

***KEY WORDS:*** *Chronic obstructive pulmonary disease (COPD), Chronic obstructive lung disease (COLD), Tobacco smoking, Diagnosis, and Management.*

## I. INTRODUCTION

Chronic obstructive pulmonary disease (COPD), also known as chronic obstructive lung disease (COLD), and chronic obstructive airways disease (COAD), among others, is a type of obstructive lung disease characterized by chronically poor airflow. It typically worsen over time[1].COPD has defined in joint statement of the American Thoracic Society(ATS) and the European Respiratory Society(ERS) as a disease characterized by and diagnosed with spirometeric measurement of air flow limitation that is not fully reversible[2].This same definition is supported by the Global Initiative of Obstructive Lung Disease (GOLD)[3].Most people with chronic bronchitis have COPD[4].Worldwide, COPD affects 329 million people or nearly 5% of the population. In 2012,it ranked as the third-leading cause of death, killing over three million people[5]The number of deaths is projected to increase due to higher smoking rates and an aging population in many countries[6].It resulted in an estimated economic cost of $2.1 trillion in 2012[7].Data from the Centers for Disease Control (CDC) showed in 2005 that COPD was overall the fourth leading cause of death in the United States and ,in some states, had surpassed stroke as the third[8].Importantly, in the United States, men and women contribute equally to the COPD epidemic since the year 2000,COPD mortality has been higher in women[9].Tobacco smoking is the most common cause of COPD, with a number of other factors such as air pollution and genetic playing a smaller role[10].In the developing world, one of the common sources of air pollution is from poorly vented cooking and heating fires. Long term exposure to these irritants causes an inflammatory response in the lungs resulting in narrowing of the small airways and breakdown of the lung tissue known as emphysema [3].The diagnosis is based on poor air airflow as measured by the lung function tests[11].Uniform international standards for the diagnosis of COPD are lacking[11].Bronchial secretions of the lower airways of patients with stable COPD grow bacteria normally found in the nasopharynx of healthy individuals,notably *Streptococcus pneumonia,Haemophilus influenza,Moraxxella catarrhalis* and other oropharyngeal commensal bacteria[12].The major goals of management are to reduce risk factors, manage stable COPD,prevent and treat exacerbation and manage associated illness[3].Long term antibiotics,specifically those from macrolide class such as erythromycin, reduce the frequency of exacerbation [13].Concerns include that antibiotic resistance and hearing problems with azithromycin[13]. Methylxanthine such as theophylline generally cause more harm than benefit, but may be used as second- line agent in those not controlled by other measures[14].The paper reviews the current literature,clinical manifestation,and management of COPD.

## II .HISTORICAL PERSPECTIVE

The word"Emphysema"is derived from the Greek word *emphysan* meaning"inflate"-itself meaning "*in*" *physan*,meaning"*breath blast*"[15].The term chronic bronchitis came into use in 1808,while the term COPD is believed to have first been used in 1965 [16,17].Previously it has been known by a number of different names, including chronic obstructive bronchopulmonary disease, chronic obstructive respiratory disease, chronic airflow obstruction, chronic airflow limitation, chronic obstructive lung disease, nonspecific chronic pulmonary disease, and diffuse obstructive pulmonary syndrome. The term chronic bronchitis and emphysema were formally defined in 1959 at CIBA guest symposium and in 1962 at the American Thoracic Society Committee meeting on Diagnostic Standards[17].Early description of probable emphysema include: in 1679 by T,Bonet of a condition of"Voluminous lungs" and in 1769 by Giovanni Morgagni of lungs which were "turgid particularly from air[17].In 1721 the first drawings of emphysema were made by Ruysh [18].These were followed by with pictures by Matthew Baillie in 1789 and descriptions of the destructive nature of the condition. In 1814 Charles Badham used "catarrh" to describe the cough and excess mucus in chronic bronchitis.Rene Laennec,the physician who invented the stethoscope,and used the term"emphysema" in his book *A treatise on the Diseases of the Chest and Mediate Ausculation*(1837) to describe lungs that did not collapse when he opened the chest during an autopsy.He noted that they did not collapse as usual because they were full of air and the airways were filled with mucus.In 1842, John Hutchinson invented the spirometer, which allowed the measurement of vital capacity of the lungs. However, his spirometer could only measure volume, not airflow.Tiffeneau and Oinelli in 1947 described the principles of measuring airflow[17].In 1953,Dr George L.Waldbott,an American allergist, first described a new disease he named "smoker's respiratory syndrome" in the 1953 Journal of the American Medical Association. This was the first association between tobacco smoking and chronic respiratory disease[19].Early treatments include garlic, cinnamon and ipecac(ipecacuanha), among others[17].Modern treatments were developed during the second half of the 20$^{th}$ century. Evidence supporting the use of steroids in COPD were established in the late 1950s.Brochodialtors came into use in the 1960s following a promising trial of isoprenaline.Further bronchodilators, such as salbutamol, were developed in the 1970s,and the use of LABAs (long-acting beta agonists) in the mid-1990s[20].

## III. PREVALENCE

Globally, as of 2010,COPD affected approximately 329 million people(4.8% of the population)[21].The affects men and women almost equally, as there has been increased tobacco use among women in the developed world[22].The increase in the developing world between 1970 and the 2000s is believed to be related to increasing rates of smoking in this region, an increasing population and an aging population due to less deaths from other causes such as infectious diseases[10].Some developed countries have seen increased rates, some have remained stable and some have seen a decrease in COPD prevalence [10].The global numbers are expected to continue increasing as risk factors remain common and the population continues to get older[23].Between 1990 and 2010 the number of the deaths from COPD decreased slightly from 3.1 million to 2.9 million[24], and became the fourth leading cause of death[10].In 2012 it became the third leading cause as the number of deaths rose again to 3.1 million[5].In some countries, mortality has decreased in men but increased in women[25].This most likely due to rates of smoking in women and men becoming more similar[4].COPD is more common in older people[1].,it affects 34-200 out of 1000 people older than 65 years, depending the population under review[1,26].In England, an estimated 0.84 million people(of 50 million) have a diagnosis of COPD,this translates into approximately one person in 59 receiving a diagnosis of COPD at some point of their lives. In the most socioeconomically deprived parts of the country, one in 32 people were diagnosed with COPD,compared with one in 98 in most affluent areas[27].In the United States approximately 6.3% of the adult population totaling approximately 15 % people have been diagnosed with COPD[28].25 million people may have COPD if currently undiagnosed cases are included[29].In 2011,there were approximately 730,000 hospitalization in the United States for COPD[30].

## IV.CONTRIBUTORY FACTORS

**Smoking**.There are multiple contributory factors of COPD,the primary contributory factor is tobacco smoke, with occupational exposure and pollution from Indoor first being significant causes in some countries.Typically these exposures must occur over several decades before symptoms develop A person's genetic makeup also affects the risks [1].The primary risk factor for COPD globally is tobacco smoking [1].Of those who smoke 20% will get COPD, and those who are lifelong smokers about half will get COPD[31,32].In the Unites States and United Kingdom, of those with COPD,80-95% are either current smokers or previously smoked[31,33,34].The likelihood of developing COPD increases with the total smoke exposure[35].Additionally, women are more susceptible to the harmful effects of smoke than men[34]. In non-smokers secondhand smoke is the cause of about 20% of cases[33].Other types of smoke, such as marijunana, cigar,and water pipe smoke, also confer a risk[1].Women who smoke during pregnancy may increase the risk of COPD in their child[1].

**Air pollution**.Poorly vented cooking fires, often fueled by coal or biomass fuels such as wood or animal dung, lead to indoor air pollution and one of the most common cause of COPD in developing countries[36].These fires are a method of cooking and heating for nearly three billion people with health effects being greater among women due to more exposure[35].They are used as main source of energy in 80% of homes in India, China and sub-Saharan Africa[37].People who live in large cities have higher rate of COPD compared to people who live in rural areas[38].While the urban air pollution is a contributory factor in exacerbations, its overall role as a cause of COPD is unclear[1].Areas with poor outdoor air quality, including that from exhaust gas, generally have higher rates of COPD[37].The overall effect in relation to smoking, however, is believed to be small[1].

**Occupational factors**.Intense and prolonged exposure to workplace dusts, chemicals, and fumes increase the risk of COPD in both smokers and nonsmokers [39].Workplace exposures are believed to be the cause of 10-20% of cases[40].In the United States they are believed to be related to more than 30% of cases among those who have never smoked and probably represent a greater risk in countries without sufficient regulations[1].In number of industries and sources have been implicated, including high levels of dust in coal mining, gold mining, and the cotton textile industry, occupations involving cadmium and isocyanides, and fumes from welding[38,39].Working in the agriculture is also a risk[36].In some professions the risks have been estimated as equivalent to that of half to two packs of cigarettes a day[41].Silica dust exposure can also lead to COPD ,with the risk unrelated to that of silicosis[42].The negative effects of dust exposure and cigarette smoke exposure appear to be additive to possibly more than additive[41].

**Role of Genetics**.Genetics play a role in the development of COPD[1].It is more common in the relatives of those with COPD who smoke than unrelated smokers[1].Currently the only clearly inherited risk factors is alpha1-antitrypsin deficiency(AAT).This risk is particularly high if someone deficient in alpha1-trypsin also smokes[43].It is responsible for about 1-5% of cases[43,44],and the condition is present in 3-4 in 10,000 people[4].Other genetic factors are being investigated[43],of which there are likely to be many[37].

**Role of airway pathology**.Tobacco smoking is the single most important risk factor for developing COPD[2].The Lung Health Study has shown that smoking cessation is the single major intervention that reduces the accelerated decline of $FEV_1$(forced expiratory volume in 1 second) in COPD patients[45].Additional risk factors are occupational dusts and chemicals, environmental tobacco smoke(second hand smoke) and indoor and outdoor air pollution[3].As is the case for lung cancer, not all smokers develop COPD, although the common belief that only105 to 15% of smokers develop airflow limitation has been recently challenged by a 25 year prospective study reporting that the absolute risk of developing COPD is continuous smokers is at least 25%[46].In patients with COPD without obvious risk factors,it is appropriate to consider the presence of a subtle defect in pulmonary host defense or the presence of an immunodeficiency syndrome[47].COPD features pathologic changes induced by combination of airways irritants, microbial infections inflammation and possibly defective regulation of innate and adaptive immunity components[48].

**Miscellaneous factors** .A numbers of other factors are less closely linked to COPD. The risk is greater in those who are poor, although it is not clear if this is due to poverty itself or other risk factors associated with poverty, such as air pollution and malnutrition[1].There is tentative evidence that those with asthma and airway hypersensitivity are at increased risk of COPD[1].Birth factors such as low birth weight may also play a role as do a number of infectious diseases including HIV/AIDS and tuberculosis[1].Respiratory infections such as pneumonia do not appear to increase the risk of COPD, at least in adults[4].

## V. PATHOPHYSIOLOGY

COPD is a type of obstructive lung disease in which chronic incompletely reversible poor airflow(Airflow limitation) and inability to breath out fully(air trapping) exists[10].The poor airflow is the result of breakdown of lung tissue(known as emphysema)and small airways disease known as obstructive bronchiolitis. The relative contributions of these two factors vary between people [1].Severe destruction of small airways can lead to the formation of large air pockets-known as bullae that replace lung tissue. This form of disease is called bullous emphysema[49].COPD develops as a significant and chronic inflammatory response to inhaled irritants[1].Chronic bacterial infections may also add to this inflammatory state[50],The inflammatory cells involved include neutrophils and macrophages two types of white cell. Those who smoke additionally have Tc1 lymphocyte involvement and some with COPD have eosinophil involvement similar to asthma. Part of this cell response is brought on by inflammatory mediators such as chemostatic factors. Other processes involved with lung damage include oxidative stress produced by free radicals in tobacco smoke and released by inflammatory cells, and breakdown of connective tissue of the lung by proteases that are insufficiently inhibited by protease inhibitors. The destruction of connective tissue of the lung is what leads to emphysema, which then contributes to the poor airflow and finally poor absorption and release of respiratory gases[1].General muscle wasting that often occurs in COPD may partly due to inflammatory mediators released by lungs into the blood[1].

Narrowing of the airways occurs due to inflammation and scarring within them.This contributes to the inability to breathe out fully.The greatest reduction in air flow occurs when breathing out, as the pressure in the chest is compressing the airways at this time [51].This results more air from previous breath remaining within the lungs when the next breath is started, resulting in an increase in the total volume of air in the lungs at any given time, a process called hyperinflation or air trapping[51].Hyperinflation from exercise is linked to shortness of breath in COPD,as it is less comfortable to breathe in when the lungs are already partly full[52].Some have a degree of airway hyperresponsiveness to irritants similar to those found in asthma[4].Low oxygen levels and eventually, high carbon dioxide levels in the blood can occur from poor gas exchange due to decreased ventilation from airway obstruction, hyperinflation and reduced desire to breathe[1].During exacerbation, airway inflammation is also increased, resulting in increased hyperinflation, reduced expiratory airflow and worsening of gas transfer. This can also lead to insufficient ventilation, and low oxygen levels [3].Low oxygen levels, if present for a prolonged period, can result in narrowing of the arteries in the lungs, while emphysema leads to breakdown of capillaries in the lungs. Both these changes result in increased blood pressure in the pulmonary arteries, which may cause cor pulmonale[1].

## VI. CLINICAL MANIFESTATION

Clinical manifestation of COPD reflect the various contributions of the two main pathologic processes, small airway remodeling and obstruction on one side and loss of elastic recoil from emphysema on the other side[48].The interplay with additional components such as asthma or airway hypersensitivity also complicates COPD phenotypes,creating additional subsets of COPD patients with different symptoms[3].Advances in understanding the complexity of COPD phenotypes have mandated that the past descriptive classification of "pink puffers"and "blue bloaters "should no longer be used because of their oversimplication. Nevertheless, for patients with COPD,separating the extreme forms as predominantly emphysema or predominantly bronchitis,based on clinical and physiological criteria, remains helpful.Briefly,the emphysema-predominant patient typically has more dyspenia,less sputum, and fewer respiratory infections. The patient is more likely to be of thin habitus, with barrel chest, has diminished breath sounds but clear lungs on auscultation, maintains reasonably normal arterial blood gas levels and has a hyperexpanded appearance of lung fields on chest films.

In contrast, the bronchitis-predominant patient has more productive coughing is prone to infections, maintain body weight or is overweight, has lung crackles and wheezing, and perhaps has right sided heart failure and pedal edema. Lethargy or somnolence can develop and acrocyanosis,polycythemia,and arterial blood gags levels that feature hypoxemia,carbon dioxide retention and academia are often present. Digital clubbing is not a finding in patient with uncomplicated COPD.Differential diagnosis of COPD must include asthma and congestive heart failure, although both may be present as comorbid conditions, bronchiectasis,tuberculosis,obliterative bronchiolitis and diffuse panbronchiolitis[3].Frequent coughing is characteristic of most patients with advanced COPD, causing them to clear their throats repeatedly and expectorate sputum throughout the day. Many patients cough up the largest amount in the morning on arising. Sputum may be viscous, sticky, and purulent, with latter being a sign of bacterial infection. Nasal and sinus congestion and postnasal drip often are associated.Several scenario must be considered in diagnosing patients. At risk individuals may be unaware that their lung function has some airflow obstruction consistent with mild COPD,[3],especially if cough and sputum production are not predominant and, as such, they have unrecognized illness and undiagnosed airway obstruction.

## VII. DIAGNOSIS

The diagnosis of COPD should be considered in anyone over the age of 35 to 40 who has shortness of breath, a chronic cough, sputum production, or frequent winter colds and a history of exposure top risk factors for disease [53].Spirometry is then used to confirm the diagnosis. **Spiromtery** measures the amount of airflow obstruction present and is generally carried out after the use of a bronchodilator, a medication to open up the airways [54].Two main components are measured to make the diagnosis: the forced expiratory volume in one second($FEV_1$),which is the greatest volume of air that can be breathed out in the first second of a breath, and the forced vital capacity (FVC),which is the greatest volume of the air that can be breathed out in a single breath[55].Nomally,75 -80% of the FVC comes out in the first second[55] and a $FEV_1$/FVC ratio of less than 70% in someone with symptoms of COPD defines a person as having the disease[54].Based on these measurements, spirometry would lead to over-diagnosis of COPD in the elderly[54].The National Institute of Health and Care Excellence criteria additionally require a $FEV_1$ of less than 80% of predicted [53].Evidence for using spirometry among those without symptoms in an effort to diagnose the condition earlier is of uncertain effect and is therefore currently not recommended[54].A peak expiratory flow (the maximum speed of expiration),commonly used in asthma, is not sufficient for the diagnosis of COPD[53].The modified British Medical Research Council questionnaire 9mNRC) or **COPD assessment test(CAT)** may be used to determine the severity of symptoms[56].Both the American European guidelines recommend partly basing treatment

recommendations on the $FEV_1$[54].**GOLD guidelines** suggest dividing people into four categories based on symptoms assessment and airflow limitation(e.g.,GOLD 1,mild, GOLD2, moderate, GOLD3, severe,and GOLD4 very severe)[56].Weight loss and muscle weakness, as well as the presence of other diseases, should also be taken into account[56].**Chest X-rays** and complete blood count may be useful, a high resolution computed tomography (CT) scan may show the distribution of emphysema throughout the lungs can be useful[4].An analysis of arterial blood is used to determine the need for oxygen; this recommended in those with an FEV1 less than 35% predicted and peripheral oxygen saturation of less than 92% and those with congestive heart failure[56].People from areas of the world where alpha-1 trypsin deficiency is common should be considered for testing[56].

COPD may need to be differentiated from other causes of shortness of breath such as congestive heart failure, pulmonary embolism, pneumonia or pneumothorax. Many people mistakenly think they have asthma[57].The distinction between asthma and COPD is made on the basis of the symptoms, smoking history, and whether airflow limitation is reversible with bronchodilators at spirometry[58].COPD may also be differentiated from other similar conditions that include tuberculosis, chronic bronchitis and others[4,56].

## VIII. MANAGEMENT

There is no cure for COPD,but symptoms are treatable and its progression can be delayed[59].The major goals of management are to reduce risk factors, manage stable COPD,prevent and treat acute exacerbations, and manage associated illnesses[3].Stopping smoking decreases the risk of death by 18%[10].Other recommendations include influenza vaccination once a year, pneumococcal vaccination once every 5 years[10].Other recommendation include (a)**pulmonary rehabilitation** is a program of exercise [60].(b)**Inhaled bronchodilators** are the primary medication, If long acting bronchodilators are insufficient, then inhaled corticosteroids are typically added[10],With respect to long-acting agents, it is unclear if tiotropium(a long acting anticholinergic)**or long-acting beta agonists(LABs)** are better it may be worth trying each and continuing that worked best[61],short- acting β agonists available including salbutamol(Ventolin)[62].(c) **corticosteroids** are usually inhaled or may be used as tablets. While inhaled corticosteroids (ICS) have not benefit for people with mild COPD ,they decrease acute exacerbations in those with either moderate or severe disease [63].When used in combination with LABs they decrease mortality more than either IVS or LABs alone[64].It unclear if they affect the progression of the disease[10].Long term treatment with steroids tablets is associated with significant side effects[62].(d) **supplement oxygen** is recommended in those with oxygen levels at rest(a partial pressure of oxygen of less than 50-55mmHg or oxygen saturations of less than 88%),it decreases the risk of heart failure and death if used 15 hours per day[62],(e)**acute exacerbations** are typically treated by increasing the usage of short-acting bronchodilators[10].This commonly includes a combination of a short-acting inhaled β agonist and anticholinergic.Nebulizer may be easier for those who are more unwell [65].(f)**Surgery** for those with very severe disease, surgery is sometimes helpful and may include lung transplantation or lung volume reduction surgery[10].

**Therapy considerations**.Overall management should address four areas:(1)maintenance therapy during quiescent intervals designed to optimize daily activity, enhance exercise tolerance, and keep, secretions ,cough ,and wheezing minimal (2)prevention of respiratory infections(3)more intensive treatment when exacerbations occur and(4) selection of appropriate antibiotic therapy for flares of bacterial origin. Prophylactic antibiotic therapy may have some use for highly selected patients who experience frequent exacerbations (four or more annually);current studies are addressing the possibility[66,].Seemungal and colleagues has shown that erythromycin,250 mg daily reduced exacerbation frequency by 35% and increased the mean time to first exacerbation of 182 days[67].Long-term antibiotics, such as erythromycin, reduce the frequency of exacerbation in those who have two or more a year[68]**.Prognosis**.COPD usually gets gradually worse over time and can ultimately results in death. It is estimated that 3% of all disability is related to COPD[69].The proportion of disability from COPD globally has decreased from 1990 to 2010 due to improved indoor air quality primarily in Asia[69].Results of spirometry are good predictor of future progress of the disease but not as good as the BODE(burden of disease estimate) index[4].

## XI. CONCLUSION

Tobacco smoking is the most common cause of COPD, with number of other factors such as air pollution and genetic has minimum role. Uniform international standards for the diagnosis of COPD are lacking. Future research to be directed towards better diagnosis and therapy of COPD. .

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