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Chronic Obstructive Pulmonary Disease: How Can It Be Assessed and Managed?

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ABSTRACT: Chronic obstructive pulmonary disease (COPD) is a serious health problem characterized by constriction in the air passages that results from different etiologic factors. The problems of COPD include narrowing of the small air passages due to inflammation and destruction of the walls result from obstructive bronchiolitis as well as emphysema— this causes air entrapment leading to difficulty in breathing especially upon physical activity. It is a frequent source of morbidity in the world. The consequence of COPD may be mortality, disability and morbidity, in addition to causing high financial and social burnouts. To overcome the complications resulted from COPD, efficient methods of diagnosis and management should be established. The evaluation of COPD is performed by clinical examination, in addition to the indication provided by spirometry. Blood WBC number (especially eosinophil count) are crucial markers for the diagnosis, classifying, and complications of COPD. The classical of frequently used drugs in COPD include bronchodilators and corticosteroids. In this review, we highlight the main causes, diagnosis, pathophysiological aspects and treatment strategies for COPD.

1. INTRODUCTION

COPD is a widespread and manageable condition. It progresses because the airflow limitation and tissue damage gradually increase (see: figure 1.) . Structural changes take place in the lungs due to continuous inflammation caused by harmful particles or gases— typically smoke from cigarettes, which leads this disease to a degenerative state by narrowing airways as well as reducing lung recoil. The illness frequently shows itself through a cough, difficulty breathing, and phlegm production; people can experience no symptoms at all or go into respiratory failure (Singh et al., 2019).

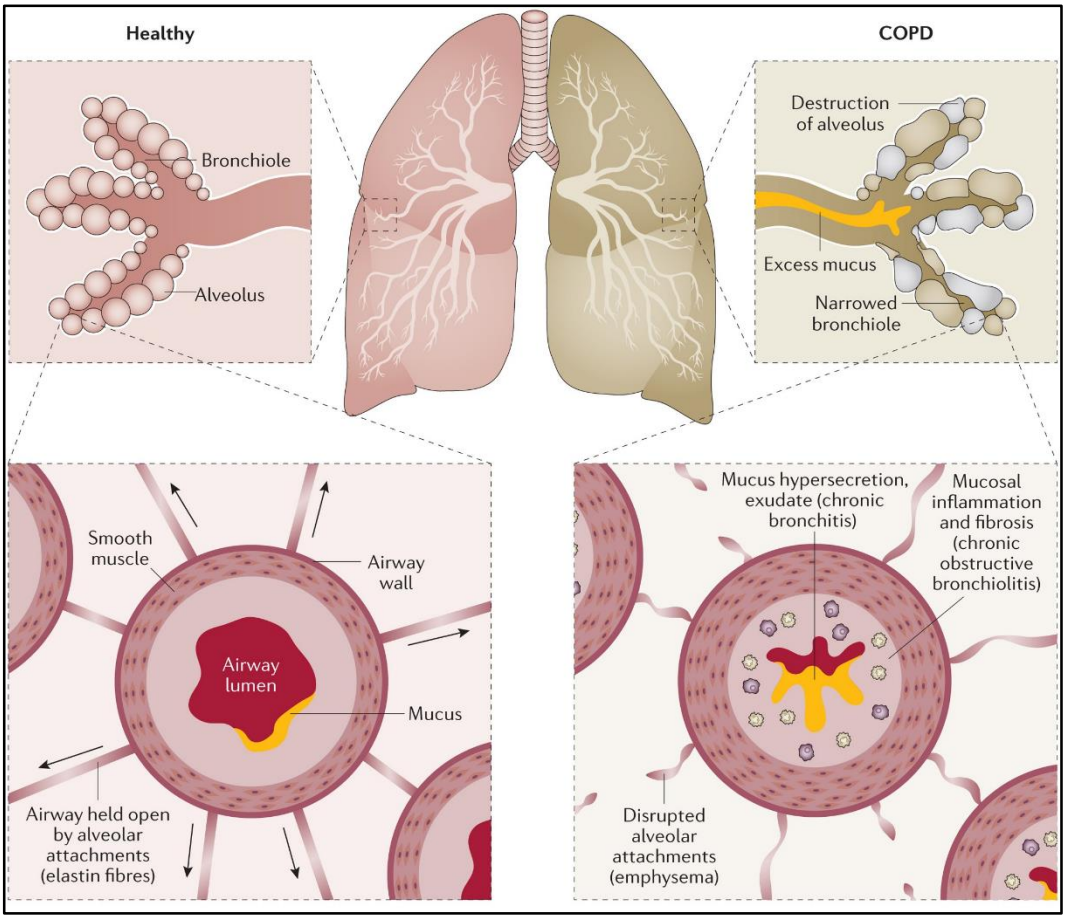

Figure 1. Mechanism of bronchial obstructions during COPD (Barnes et al., 2015)

Chronic obstructive pulmonary disease mainly occurs among smokers and individuals over 40 years old. The older a person gets, the higher the likelihood of having COPD as it stands third among most causes of morbidity and mortality globally. In 2015, the global prevalence of COPD was estimated to be 174 million people with about 3.2 million deaths recorded worldwide. However, this figure is likely an underestimate given that many cases of COPD are not diagnosed (GBD 2015 CRDC, 2017).

COPD does not often appear until adulthood and it is mostly noted during the cold months which are in winter. The common symptoms that patients typically complain about include dyspnea — that is chronic and progressively worsening — sputum and cough formation. Wheezing plus thorax tenderness may also be found although most cases have a smoking history; some do not smoke but they should be asked about exposure to second-hand smoke, occupational and environmental exposures, and family history after COPD has been confirmed. Other queries should be made on previous exacerbations and nighttime awakenings as well as inhaler usage; effects of the disease on activity level should also be determined. The patient's past medical history for other diseases such as asthma or allergies should also be taken into account. Childhood respiratory infections might implicate alpha-1 antitrypsin deficiency if liver disease or basilar emphysema is identified along with a family history of emphysema; all these could increase suspicion for this deficiency when COPD is accompanied with dyspnea, cough — even wheezing: these are the usual signs of COPD flares. (Moszura et al., 2014).

COPD doesn't come alone; it brings a host of other conditions along with it. And recently, the numbers of these co-existing companions have been evaluated. Interestingly, dyspnea isn't just about difficulty in breathing— it's also closely linked to psychological issues like anxiety. When your muscles don't function well, blame inflammation and oxidative stress for their woes; but remember that respiratory muscle satellite cells are there to help repair any damage done. Think of respiratory rehabilitation and physiotherapy as the yin to pharmacological treatment's yang— they must complement each other in an individualized patient approach. Acute exacerbations knocking at your door? Be prepared to fight off 75% caused by infections; knowing sputum characteristics and keeping an eye out for Pseudomonads can make or break antibiotic therapy. But more questions surface: can we unearth markers that reveal where these infections stem from? What prognostic factors loom ahead, or how do short stay pneumology units fit into this jigsaw puzzle scenario? The debate continues on COPD treatments' variability — why haven't suitable international clinical guidelines found their way to us yet? (López-Campos et al., 2009).

Chronic obstructive pulmonary disease (COPD) manifests itself through airway obstruction, which is difficult to reverse and typically diagnosed using spirometry. It involves small airway obstruction — also known as chronic obstructive bronchiolitis —

and emphysema that causes air trapping leading to dyspnea that occurs on exertion. Provision of oxygen plays a significant role in decreasing COPD mortality worldwide; however, there are also other issues surrounding the disease. Misdiagnosis and misclassification prevail due to limited clinical capacity on COPD: this compromises effective use of spirometry, an essential tool for diagnosis (Adeloye et al., 2022).

DIAGNOSIS

Spirometric confirmation of post-bronchodilator airflow obstruction and evaluation of respiratory symptoms are the basic requirements for COPD diagnosis. The symptoms include dyspnea, cough, sputum production and exercise limitation. A five-step assessment is needed to make a definite diagnosis of COPD which consists of: 1/ risk assessment by inhalation, 2/ evaluation of symptoms, 3/ pulmonary physiologic investigations, 4/ diagnostic blood tests and 5/ image scanning (.

Amidst the challenges that arise due to different case definitions and diagnostic criteria, the disparity in approaches to clinical as well as epidemiological studies between regions have been highlighted in previous works; these discrepancies are largely why there is so much uncertainty surrounding the prevalence of COPD. For instance, a study conducted in 2015 using data from 27 states revealed that over 80% of identified COPD cases through spirometry were undiagnosed— this was especially prevalent in sub-Saharan Africa among young adults and men, and individuals who had never smoked. Another study showed that between one-third and two-thirds of physician-diagnosed COPD cases did not actually have the disease even after spirometry and further clinical evaluation; many health care providers were likely to reverse a last COPD identification upon reevaluating patients, which underscores how misdiagnosis can impede understanding of the disease yet it forms part of a high burden-related issue for COPD, hampering response systems within health care across different sectors (Lamprecht et al., 2015).

International respiratory organizations have currently made the suggestion to take the lower limit of normal and standardized residuals as the basis for diagnosing airflow narrowing and COPD even though using a fixed ratio less than 0.7 is easier, and at the same time it predicts many important clinical outcomes in a strong way. Eosinophil count is still one of the key COPD biomarkers; although we now have a better understanding of normal eosinophil levels in COPD, it is also essential to be aware of what they are typically within the wider population (Calverley & Walker, 2023).

SYMPTOMS

COPD dances its way onto the evaluation stage for those with symptoms or who are at risk. But when the curtains fall, it is spirometry that confirms the diagnosis. Other players in this diagnostic drama may include a 6-minute walk test, lab work, and imagery studies. PFT takes a pivotal role in COPD diagnosis, staging and surveillance — where spirometry plays its part both pre- and post-bronchodilator. The bronchodilators stepping into this performance could be SABAs or short-acting anticholinergics alone or hand in hand: their presence helping unveil COPD through FEV1/FVC < 0.7 ratio (Vall Camell et al., 2019).

Laboratory testing typically involves conducting a full blood count, which aids in detecting infection as well as conditions such as anemia and polycythemia. Along with these tests, it is also important to check the levels of Alpha-1 antitrypsin for other causes related to COPD. Among the various aspects that these blood leukocyte counts can reveal include eosinophil count— an important biomarker which contributes towards onset classification and even exacerbation situations surrounding COPD. While understanding causal relationships between these different factors are essential towards coming up with effective strategies for managing COPD, certain details still remain unclear: for instance, whether the elevation in neutrophil number is trigerred by COPD onset or FEV1/FVC decline. As such, we can consider eosinophil instead of neutrophil as a therapeutic target that would help in preventing occurrence/exacerbation of COPD since indications do not support use of Neutrophils — hence any future treatment approach being taken should be non-neutrophil targeted, especially if dealing with cases related to neutrophilic COPD in future (Han et al., 2022).

Radiographic imaging is composed of a chest x-ray and computed tomography (CT). While chest x-rays can reveal hyperinflation and diaphragm flattening, CT is more specific for identifying bronchiectasis in the case of chronic bronchitis. Additionally, CT imaging can be helpful for detecting malignancy or visualizing anatomy prior to surgical procedures among COPD patients: it shows centrilobular emphysema mainly localized at the upper lobes if present plus bullae with subpleural location (Shaker et al., 2007).

An exacerbation of COPD denotes an acute escalation of respiratory issues. Typically, the severity is assessed based on Anthonisen's model which takes into account three major factors determining the level: worsening dyspnea, sputum volume, and purulence. A mild exacerbation can be identified if one of these symptoms co-occurs with any of the following: increased wheezing or cough, unexplained fever, upper respiratory infection within 5 days, or an abnormal rise in heart or respiratory rate from baseline. Moderate and severe exacerbations hinge on the manifestation of two or all three symptoms respectively. Additionally, patients might present with acute respiratory failure along with physical indications such as hypoxemia and hypercapnia — this calls for arterial blood gas analysis, chest imaging, and pulse oximetry without delay (Decramer et al., 2012).

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RISK FACTORS

COPD is one of the leading causes of morbidity, mortality, and medical care consumption across the globe. The development of COPD has been mainly associated with exposure to inhaled particles that lead to harm— among which tobacco smoke and other pollutants are common examples. Yet the wider spectrum of determinants that aggravate the risk for developing and advancing COPD throughout life's course are progressively acknowledged (Christenson et al., 2022).

COPD was traditionally believed to be resulted from smoking tobacco. But over the past decade, the recognition of non-smoking-related risk factors for COPD has emerged as an important issue and continues to grow with evidence on burden, risk factors, and clinical presentation in non-smokers. Nearly half of COPD cases worldwide do not result from tobacco but rather from other non-tobacco-related risk factors that differ across regions such as air pollution, occupational exposures, asthma infection through smoke or other causes, environmental diseases and low socioeconomic status. Additionally, hindered lung growth during childhood — due to various early life exposures — plays a role in COPD development through different mechanisms including oxidative stress (Yang et al., 2022).

PATHOPHYSIOLOGY

COPD does not just involve inflammation. The disease affects not only the airways but also lung parenchyma and pulmonary vasculature. Oxidative stress is a critical aspect of the disease process, as are imbalances between protease and antiprotease activity levels. Emphysema is actually just one part of COPD; it refers specifically to structural changes where alveolar air sacs are destroyed because an irritant such as smoke triggers an inflammatory response involving neutrophils plus macrophages that release a host of mediators (including oxidants and proteases) causing destruction (specifically elastin). With loss of elastic recoil due to this destruction mechanism there will be airflow limitation, but also consider other aspects that would lead into comorbidities— including features only seen among never-smokers presenting with COPD such as mild chronic respiratory symptoms despite having little or no emphysema (Singh, et al., 2019; Yang et al., 2022).

Obstruction of airways and the inflammatory response are contributors to decreased FEV1, while airflow limitation is a result of tissue destruction leading to impairment in gas exchange— elements that define the pathophysiology of COPD. Acute exacerbations are typical in COPD and have identifiable triggers such as bacterial or viral pneumonia environmental irritants; it promotes further inflammation which can be difficult to manage without corticosteroids plus bronchodilators (Parker et al., 2005).

During percutaneous angioplasty, it is typically the norm to use a balloon that has a diameter 0.1–0.2 cm larger than the pre-stenotic aorta. There have been suggestions by some researchers to assess stenosis distensibility before implanting the endoprosthesis by using low pressure (less than 4 atm) to inflate the balloon; this is because in highly fibrotic stenosis, the endoprosthesis may not completely dilate upon the first attempt. However, if it does grow— additional balloon inflations can be performed to further dilate the endoprosthesis despite complications— excellent instant cure ratio is seen (96.9% even among low weight children). An interesting study carried out in 2021 among patients under 20 kg revealed that 18% of complications related to femoral artery injury were actually due to interventional procedures being most frequent (Boe et al., 2021).

MANAGEMENT

In COPD, the major objectives of therapy are symptom management, enhancement in life quality while minimizing occurrences of mortality and exacerbations. The non-pharmacological approach comprises two main components: smoking cessation and pulmonary rehabilitation. There are six classes of medications that are commonly used for COPD which include antibiotics, phosphodiesterase-4 (PDE4) inhibitors, systemic glucocorticoids, inhaled corticosteroids (ICS), methylxanthines, antimuscarinics, and beta2-agonists (Agarwal et al., 2023).

The medications regularly used in COPD belong to several classes. Bronchodilators (methylxanthines, antimuscarinics, beta2-agonists), inhaled corticosteroids (ICS), systemic glucocorticoids, phosphodiesterase-4 (PDE4) inhibitors, and antibiotics make the list. Beta2-agonists act by easing the tension of smooth muscle in air passages; commonly prescribed are short-acting beta2-agonists (SABA) and long-acting beta2-agonists (LABA). SABAs offer instant improvement when used as required while LABAs are intended for continuous maintenance therapy. Antimuscarinics function by inhibiting bronchoconstriction at the smooth muscle M3 muscarinic receptors— thus short-acting antimuscarinic agents (SAMA), like SABAs, act quickly upon use and are recommended on an as-needed basis; whereas long-acting antimuscarinic agents (LAMA), such as LABAs, are meant for maintenance therapy (Melani., 2015).

Methylxanthines are also used in a regular way— after LABA or LAMA treatment as an adjunctive relief. Methylxanthines act by producing mild bronchodilation due to the relaxation of smooth muscles in airways whose mechanism of action is still not clear but may be through inhibition of phosphodiesterase (PDE) III and IV. Theophylline is one of the most common methylxanthines: when used with salmeterol, it has shown to exhibit significantly higher enhancement in FEV1 than salmeterol alone. Inhaled corticosteroids are typically used with LABAs plus LAMAs to reduce inflammatory response: actually, a combination of ICS and LABA has been demonstrated to have more useful effects than each drug alone. A heightened chance for pneumonia is a warning that should be heeded by doctors and patients, especially in cases where ICS is the chosen treatment. Long-term use of

oral glucocorticoids is contraindicated due to their host of adverse effects; these should only be used for the control of acute exacerbations and not indiscriminately (Nannini et al., 2021).

CONCLUSIONS

A lung disease — characterized by limitation of airflow due to abnormal inflammatory response of the lungs — is what chronic obstructive pulmonary disease (COPD) is. It is a major cause of death globally; hence, it must be detected and managed early enough. COPD is diagnosed based on the demonstration of poorly reversible airway obstruction on spirometry. Blood eosinophil counts are important biomarkers that help in onset determination and classification as well as exacerbation prediction for COPD. Medications used broadly in COPD can be divided into two: bronchodilators, corticosteroids.

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