JOURNAL OF INTERNATIONAL CRISIS AND RISK COMMUNICATION RESEARCH
ISSN: 2576-0017
2024, VOL 7, NO S8

Multidisciplinary Approach to Chronic Obstructive Pulmonary Disease (COPD) Management

Omar Baker Banamah[1*], Batool Abbas Almishkab[2], Nada Faiez Alshanbari[3], Shimaa Fahad Alradadi[4], Usama Ibrahem Ibrahem Badr[5], Salwa Ahmed Osman[6], Hamed Saeed Alghamdi[3], Ibrahim Ali Alhussain[7], Fatimah Hassan Muhanna[1], Afraa Abdulrahman Aljahdali[8], Abdullah Saeed Alzaki[1], Sultan Saeed Alharbi[8], Sarah Mohammed Alnassar[9], Marwah Thani Hawsawi[10], Mayssan Hussein Ali Almalki[11]

1. King Abdulaziz Medical City, National Guard, Health Affairs – Jeddah, Saudi Arabia
2. Dammam Medical Complex, 5343 King Khalid Dammam 32245, Dammam 32245, Saudi Arabia
3. King Abdulaziz Hospital, Al Mahjar, Jeddah 22421, Saudi Arabia
4. Al-Noor Specialist Hospital, 3rd Ring Rd, Al Hijrah, Makkah 24241, Saudi Arabia
5. Qatif Central Hospital - Ministry of Health-Qatif, Eastern Province, Dhahran Jubail Branch Rd, Al Iskan, Al Qatif 32654, Saudi Arabia
6. Sudan International University, GJ32+W3F, Khartoum, Sudan
7. Al Naeriyah General Hospital, Al Faisaliyah, Nairyah 37487, Saudi Arabia
8. King Fahad Hospital Al Hofuf, Alhufayrah, Al Hofuf 36441, Saudi Arabia
9. Jeddah Second Health Cluster, Alamal Plaza Hail Street PO Box 6659, Jeddah, Saudi Arabia
10. Ibn Sina National College for Medical Studies, Al Mahjar, Jeddah 22421, Saudi Arabia
11. King Abdullah Medical Complex, Prince Nayef Street، Northern Abhor, Jeddah 23816, Saudi Arabia

Corresponding Author Email: Dr_omarbb@hotmail.com

Abstract:
Background: A multidisciplinary approach to Chronic Obstructive Pulmonary Disease (COPD) management is essential for optimizing patient outcomes, as it integrates various therapeutic strategies tailored to individual needs. This approach combines pharmacological treatments with non-pharmacological interventions, addressing the disease's respiratory and systemic aspects. Integrating different healthcare professionals and personalized care plans has been shown to improve lung function, reduce exacerbations, and enhance the quality of life for COPD patients. **Objective:** An overview of COPD etiology, pathophysiology, and management. **Methods:** The PUBMED And Google Scholar Search Engines Were the Main Databases Used for The Search Process, With Articles Collected From 1970 To 2024.**Conclusion:**COPD is a common and severe respiratory disorder with far-reaching health, economic, and social effects, particularly growing in developing areas. Its main cause is cigarette smoking, alongside environmental and genetic factors, emphasizing the importance of awareness and early intervention for better patient care. The condition leads to chronic inflammation and airflow limitations, resulting in symptoms like breathlessness and cough, which require thorough diagnosis and tailored treatment. A holistic management strategy, including pharmacological and non-pharmacological approaches, is vital for improving patients' overall quality of life.
Keywords: Etiology, Risk Factor, Pathophysiology, Manifestation, Diagnosis, Management, COPD.

Introduction:

Chronic Obstructive Pulmonary Disease (COPD) is a significant global health issue defined by the Global initiative for chronic Obstructive Lung Disease (GOLD) as a common preventable and treatable disease characterized by persistent airflow limitation, which is usually progressive and associated with an enhanced chronic inflammatory response of the airways and lungs to noxious particles or gases(1). This condition primarily encompasses chronic bronchitis and emphysema [Figure 1], leading to a heterogeneous group of respiratory ailments that result in airflow obstruction that is not fully reversible(2). The prevalence of COPD is alarmingly high, with estimates suggesting that it affects between 4-6% to 10-25% of the adult population globally. Since 2010, COPD has been recognized as the third leading cause of death worldwide, with approximately 200,000 to 300,000 deaths occurring annually in Europe alone(1). The disease's increasing prevalence is particularly concerning in developing countries, where awareness and diagnostic capabilities may be limited. Moreover, The prognosis for individuals diagnosed with COPD is serious; about half of those diagnosed die within ten years, underscoring the disease's severity and the urgent need for effective management strategies(2, 3).

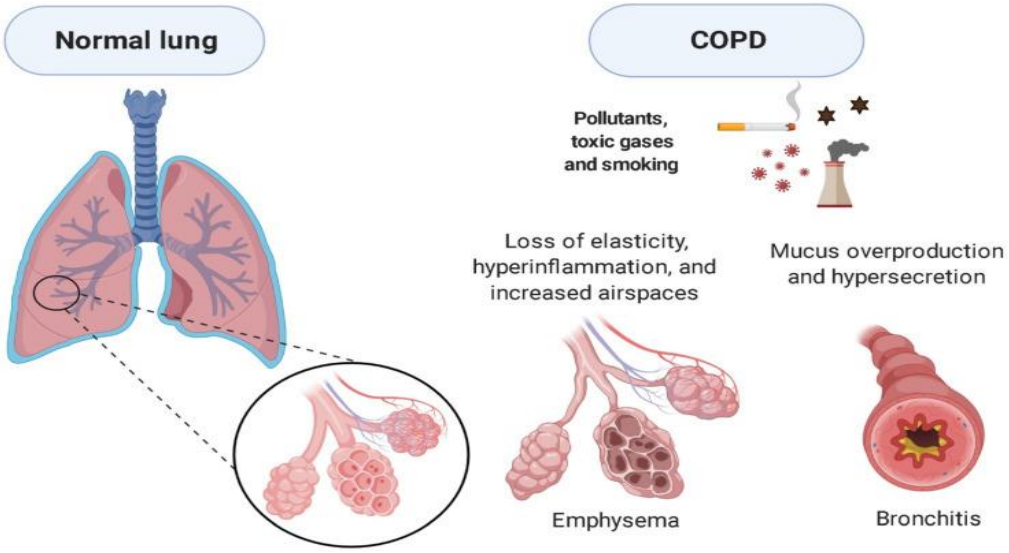

Figure (1): Distinct morphological observed between a healthy lung and an affected one by COPD(4).

COPD is a complex condition with a multifactorial etiology, primarily driven by environmental and genetic risk factors. The most significant risk factor for developing COPD is cigarette smoking, which is responsible for the majority of cases due to its detrimental effects on lung tissue. Smoking not only initiates the disease but also exacerbates its progression, making smoking cessation a critical therapeutic approach for affected individuals. In addition to active smoking, other forms of tobacco exposure, such as environmental tobacco smoke, biomass smoke, and occupational exposures, contribute to the risk of COPD, albeit to a lesser extent. Biomass smoke, often encountered in developing countries, is particularly relevant for populations relying on solid fuels for cooking and heating. (5-7)Furthermore, occupational exposures to dust, chemicals, and air pollution are recognized as significant environmental risk factors, especially when exposure is intense or prolonged(8). Genetic factors also play a role in the etiology of COPD. A notable genetic risk factor is alpha 1-antitrypsin deficiency, a rare genetic syndrome that can lead to early onset COPD in susceptible individuals(6). Family and twin studies suggest that genetic predispositions may interact with environmental exposures, influencing the variability in disease development among individuals(5, 6, 8).

Pathophysiology of COPD

COPD characterized by progressive airflow limitation that is not fully reversible. The pathophysiology of COPD involves a combination of chronic inflammation, tissue destruction, and structural changes in the lungs, as mentioned earlier due to exposure to noxious particles, most notably from cigarette smoke [Figure2](9). At the core of COPD's pathophysiology is chronic inflammation, which leads to the remodeling and narrowing of small airways. This inflammation is marked by an increase in neutrophils and macrophages, which contribute to the inflammatory response(9, 10). The persistent inflammatory state results in small airway inflammation and fibrosis, further exacerbating airflow obstruction(11). The combination of small airway disease, known as obstructive bronchiolitis, and parenchymal destruction, particularly emphysema, leads to significant airflow limitation and symptoms such as dyspnea and chronic cough. Tissue destruction is a hallmark of emphysema, where the alveoli are damaged, leading to a loss of elastic recoil and the collapse of small airways during expiration(7, 10, 11). This destruction is driven by an imbalance between proteases and antiproteases, which normally regulate tissue integrity, and an imbalance between oxidants and antioxidants, contributing to oxidative stress in the lungs. The resulting oxidative stress amplifies the inflammatory response, leading to further tissue damage and impaired repair mechanisms. (11)The pathological changes in COPD also include increased resistance to airflow and air trapping due to the loss of alveolar attachments to the small airways, which decreases lung compliance(9, 10).

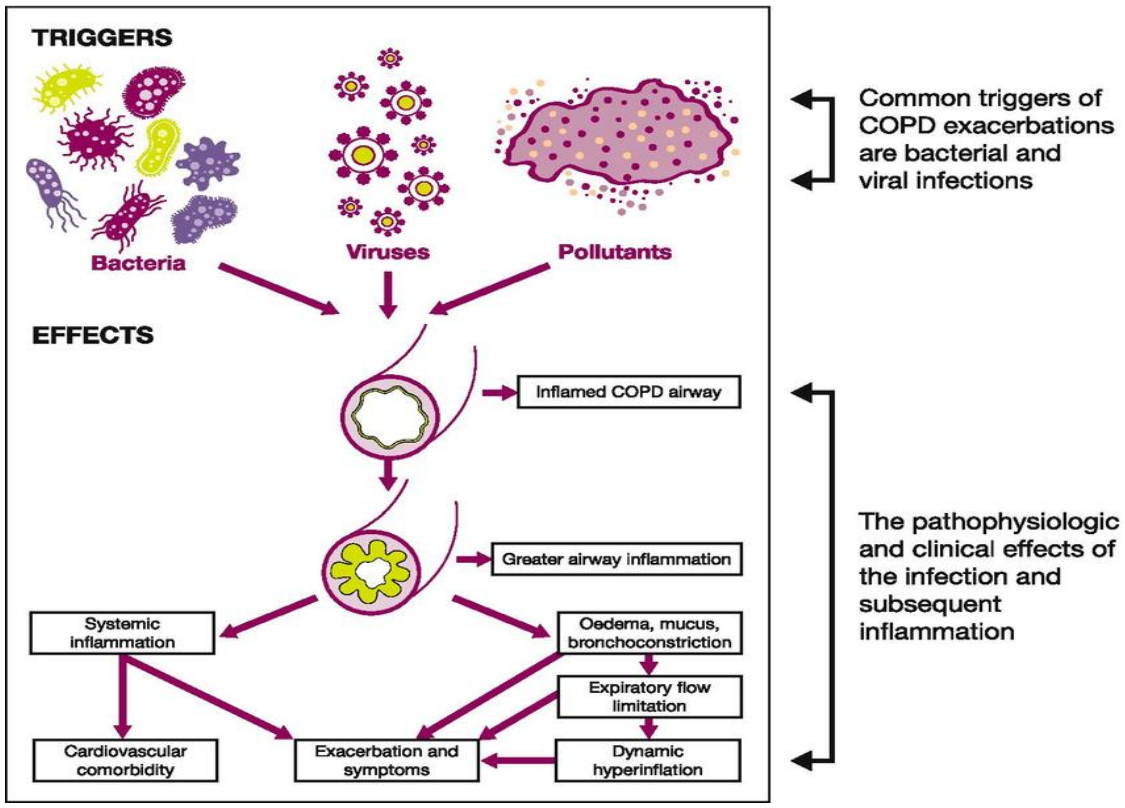

Figure (2): The pathophysiology of COPD exacerbations.

Clinical Manifestations of COPD

COPD is characterized by a range of symptoms and complications that significantly impact patients' quality of life. The most common symptoms include increased breathlessness and productive cough, particularly pronounced during acute exacerbations of the disease. These exacerbations are clinically recognized as episodes requiring more intensive treatment due to the worsening of these symptoms. Shortness of breath during physical activities is also a prevalent symptom, contributing to the limitations in daily functioning that many COPD patients face(12). In addition to breathlessness, patients often experience dyspnea, a subjective feeling of difficulty in breathing that can be exacerbated during sleep. This symptom is particularly concerning as it can lead to sleep disturbances, resulting in morning fatigue and early awakenings, further complicating the patient's overall health status(13). Complications arising from COPD are severe and multifaceted. One of the most critical complications is hypoxemia, which results from impaired gas exchange in the lungs. This condition can lead to elevated pulmonary pressures and, in severe cases, right ventricular overload and failure(13). The risk of exacerbations not only increases the annual decline in lung function but also correlates with a higher mortality rate among patients. The exacerbations are associated with significant morbidity and can lead to hospitalizations, which further strain healthcare resources (12). Moreover, the complications of COPD extend beyond respiratory issues. Patients often face an increased risk of comorbidities, including cardiovascular disease, osteoporosis, and neuro-psychiatric complications, all of which can exacerbate the overall burden of the disease (12). The interplay between these symptoms and complications underscores the importance of effective management strategies aimed at reducing the frequency of exacerbations and improving patients' health status.

Diagnosis of COPD

Diagnosing COPD involves a systematic approach that combines clinical suspicion, patient history, physical examination, and objective testing. The process begins with clinical suspicion, particularly in patients aged 35 years or older who present with relevant respiratory symptoms such as chronic cough, increased sputum production, or progressive dyspnea.A thorough patient history is essential, focusing on respiratory symptoms and risk factors, especially a history of smoking, which is the leading risk factor for COPD(14, 15). Once clinical suspicion is established, the next step is to confirm the diagnosis through spirometry. This objective test measures lung function, specifically the forced expiratory volume in one second (FEV1) and forced vital capacity (FVC). A FEV1/FVC ratio of less than 70 percent that is incompletely reversible with bronchodilator administration is indicative of COPD(14).

Spirometry is crucial as it provides confirmatory evidence for the diagnosis, distinguishing COPD from other respiratory conditions, such as asthma, which can present with similar symptoms. In addition to spirometry, appropriate examination findings are necessary to support the diagnosis (14, 15). Physicians should look for signs of respiratory distress and assess lung function through physical examination. While spirometry is the gold standard for diagnosis, imaging techniques such as computed tomography (CT) can also play a role, particularly in identifying emphysema, a common component of COPD. CT is superior to chest radiography in detecting mild and moderate emphysema, with detection rates exceeding 90%. High-resolution computed tomography (HRCT) can further enhance the identification of small areas of emphysema, although it may miss very small lesions(16).

The GOLD criteria for COPD classification [Figure 3] provide a comprehensive framework for categorizing patients based on their symptoms, exacerbation history, and spirometry measurements. This system is pivotal for guiding treatment decisions and predicting patient outcomes. The GOLD classification divides COPD patients into four categories: A, B, C, and D. Category A includes patients with low symptoms and low risk of exacerbations, while Category B consists of patients with more symptoms but still low risk. Conversely, Category C encompasses those with fewer symptoms but a high risk of exacerbations, and Category D includes patients who experience both high symptoms and high risk of exacerbations(17). To determine these categories, the GOLD guidelines recommend a multifactorial assessment that includes the COPD Assessment Test (CAT) [Table1] score and the modified Medical Research Council Dyspnoea scale (mMRC) [Figure 2]. Specifically, a CAT score of ≥10 or an mMRC score of ≥2 indicates a higher symptom burden, which is crucial for classifying patients into the appropriate categories. Additionally, the history of exacerbations in the past year is assessed, distinguishing between patients with zero or one exacerbation versus those with two or more. This classification further refines the assessment by integrating the degree of airflow limitation into the overall evaluation of COPD severity(18).The importance of the GOLD criteria lies in their ability to facilitate a more nuanced understanding of COPD, moving beyond simple airflow obstruction to encompass symptom burden and exacerbation risk. This multifaceted approach allows for better patient stratification and tailored management strategies, ultimately improving patient outcomes(17).

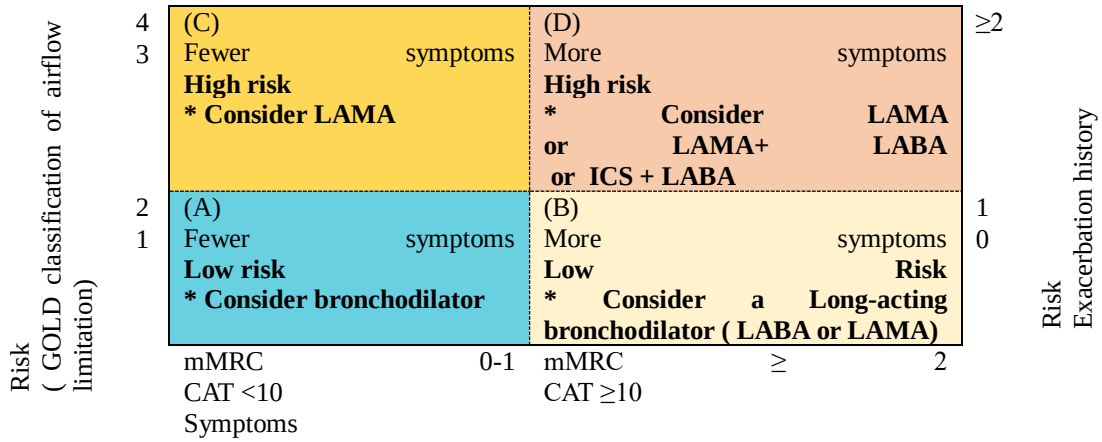

Figure (3): Gold criteria for COPD classification and pharmacological management.

Table (1): COPD Assessment Test (CAT)(19).

I never cough.	**0**	**1**	**2**	**3**	**4**	**5**	**I cough all the time.**
I have no phlegm (mucus) in my chest at all.	**0**	**1**	**2**	**3**	**4**	**5**	**My chest full of phlegm (mucus).**
My chest does not feel tight at all.	**0**	**1**	**2**	**3**	**4**	**5**	**My chest feels very tight.**
When I walk up a hill or one flight of stairs, I am not breathless.	**0**	**1**	**2**	**3**	**4**	**5**	**When I walk up a hill or one flight of stairs, I am very breathless.**
I am not limited to doing any activities at home.	**0**	**1**	**2**	**3**	**4**	**5**	**I am very limited doing any activities at home.**
I am confident leaving my home despite my lung condition.	**0**	**1**	**2**	**3**	**4**	**5**	**I am not at all confident leaving my home because of my lung condition.**
I sleep soundly.	**0**	**1**	**2**	**3**	**4**	**5**	**I don't sleep soundly because of my lung condition.**
I have lots of energy.	**0**	**1**	**2**	**3**	**4**	**5**	**I have no energy at all.**

Table (2): modified Medical Research Council Dyspnea scale (mMRC)(20).

Grade of Dyspnea	Description of breathlessness
0	I only get breathless with strenuous exercise.
1	I get short of breath when hurrying on level ground or walking up a slight hill.
2	On level ground, I walk slower than people of the same age because of breathlessness, or I have to stop for breath when walking at my own pace on the level.
3	I stop for breath after walking 100 yards or after a few minutes on level ground.
4	I am too breathless to leave the house, or I am breathless when dressing.

Pharmacological Management of COPD

The pharmacological management of COPD is multifaceted, focusing on alleviating symptoms, preventing exacerbations, and improving overall quality of life. The primary classes of medications used include bronchodilators, inhaled corticosteroids (ICS), and combination therapies. Bronchodilators, which include Long-Acting Beta-2 Agonists (LABA) and Long-Acting Muscarinic Antagonists (LAMA), are essential in managing COPD. LABAs are recommended to improve breathing and reduce symptoms of dyspnea and exercise intolerance, while LAMAs enhance bronchodilation when used in combination with LABAs, providing a synergistic effect that is often more effective than monotherapy(21, 22)The combination of these agents is particularly beneficial for patients who do not achieve satisfactory symptom control with a single bronchodilator(23). Inhaled corticosteroids play a crucial role in reducing airway inflammation, especially in patients with a history of frequent exacerbations. While ICS can decrease the rate of exacerbations and improve lung function, their use must be carefully considered due to potential side effects, such as pneumonia and oropharyngeal candidiasis(24, 25).The efficacy of ICS is often enhanced when combined with LABAs, leading to improved outcomes in terms of exacerbation frequency and quality of life. For patients with more severe symptoms or frequent exacerbations, triple therapy, which includes ICS, LABA, and LAMA, is recommended. This approach has been shown to reduce hospitalizations for acute exacerbations compared to monotherapy with long-acting bronchodilators. However, the potential for overprescribing triple therapy raises concerns about unnecessary healthcare costs and the need for careful patient assessment(26). Additionally, patient-centered care is vital in the pharmacological management of COPD. Tailoring treatment to individual patient needs, preferences, and comorbidities can significantly enhance treatment adherence and outcomes(27) This approach ensures that patients are actively involved in their care, which is essential for effectively managing a chronic condition like COPD.

According to Gold's criteria for COPD management, patients in Group A are advised to undergo treatment with either a short-acting or a long-acting bronchodilator to alleviate breathlessness. The initial treatment approach for those in Group B involves using a LABA or LAMA. There is no definitive evidence indicating a preference for one class of inhaled long-acting bronchodilators over the other, thus making the selection potentially individualized based on patient needs. For individuals in Group C, a LAMA is recommended as the primary option. Research has demonstrated that LAMAs are more effective than LABAs in preventing exacerbations. Patients classified in Group D may receive treatment with a LAMA, as it aids in both managing breathlessness and preventing exacerbations. If a patient experiences significant symptoms, characterized by increased dyspnea and/or limitations in exercise, a combination therapy of a LAMA and a LABA may be warranted. The recommended treatment for patients in Group D who exhibit eosinophil counts of ≥300 cells per microliter or possess a history of asthma is an inhaled corticosteroid (ICS) with a LABA. An additional update to the guidelines indicates that the effectiveness and benefits of lung function improvement with ICS usage are diminished in heavy or current smokers compared to light or former smokers; nonetheless, this does not warrant the avoidance of ICS in these patient populations.(28, 29)

Non-pharmacological Management of COPD

Non-pharmacological management of COPD is essential for improving patients' quality of life and mitigating disease progression. This approach encompasses a variety of strategies, including pulmonary rehabilitation, breathing techniques, oxygen therapy, nutritional support, and psychosocial interventions. Pulmonary rehabilitation is a structured program that combines exercise training, education, and support, aimed at enhancing both physical and emotional well-being in COPD patients. It is particularly effective in addressing common comorbidities such as anxiety and depression, which are often overlooked in this population. The program typically includes tailored exercise regimens that improve exercise tolerance, respiratory muscle strength, and overall health. By breaking the cycle of worsening breathlessness and reduced physical activity, pulmonary rehabilitation plays a crucial role in restoring patients to an optimally functioning state. Breathing techniques, such as pursed-lip and diaphragmatic

breathing, are also vital in managing breathlessness and improving lung function(30-32). These methods empower patients to control their symptoms more effectively, enhancing their ability to engage in daily activities. Oxygen therapy and supplementation are critical for patients with low blood oxygen levels, as they alleviate symptoms and improve exercise capacity(32). This strategy, along with the reduction of metabolic demands, helps optimize tissue oxygenation and overall management of COPD(33). Tailoring these interventions to the severity of the disease is essential for maximizing their effectiveness. Nutritional support is another key component, particularly for patients experiencing involuntary weight loss or undernutrition. Dietary interventions can enhance exercise tolerance and overall health, further contributing to improved quality of life(30). Regular physical activity, encouraged through structured programs, is vital for maintaining muscle strength and functional capacity in COPD patients. (30)Moreover, education and self-management strategies empower patients to recognize symptoms and understand treatment options, fostering a sense of control over their condition. Psychosocial support, including counseling and support groups, addresses the emotional challenges associated with living with COPD, helping to reduce anxiety and depression.

Conclusion:

COPD is a common and severe respiratory disorder with far-reaching health, economic, and social effects, particularly growing in developing areas. Its main cause is cigarette smoking, alongside environmental and genetic factors, emphasizing the importance of awareness and early intervention for better patient care. The condition leads to chronic inflammation and airflow limitations, resulting in symptoms like breathlessness and cough, which require thorough diagnosis and tailored treatment. A holistic management strategy, including pharmacological and non-pharmacological approaches, is vital for improving patients' overall quality of life.

Author Contributions

All authors contributed significantly by gathering information and searching the literature for the essay, even if the corresponding author was in charge of the initial writers' work. Every author participated in the manuscript's critical assessment, took ownership of every section, and gave their approval to the final draft.

Acknowledgments

The authors acknowledge and are very grateful to the publishers and open access databases such as Pubmed, Medline, DOAJ, Embase, and others whose works provided the literature needed to complete this article.

Conflict of Interest

The authors declare they don’t have any conflict of interest.

Ethical Approval

Not Applicable

References:

1. Lahousse L. Epidemiology of Comorbidities in Chronic Obstructive Pulmonary Disease. 2014.
2. Wier LM, Elixhauser A, Pfuntner A, Au DH. Overview of hospitalizations among patients with COPD, 2008. 2011.
3. Umoh VA. An overview of chronic obstructive pulmonary disease (COPD): epidemiology and pathogenesis. The Nigerian Health Journal. 2012;12(3):55-64.
4. Rodrigues SO, Cunha C, Soares GMV, Silva PL, Silva AR, Gonçalves-de-Albuquerque CF. Mechanisms, Pathophysiology and Currently Proposed Treatments of Chronic Obstructive Pulmonary Disease. Pharmaceuticals (Basel). 2021;14(10).
5. Paré P, Sandford A. Genetic risk factors of chronic obstructive pulmonary disease. Swiss medical weekly. 2002;132(0304):27-37.
6. Muro S. Cigarette smoking is the most important causal factor for developing chronic obstructive pulmonary disease (COPD). Nihon rinsho Japanese Journal of Clinical Medicine. 2011;69(10):1735-40.
7. Ullmer, Solèr, Perruchoud. Etiology, diagnosis and therapy of COPD. Therapeutische Umschau. 1999;56(3):125-30.
8. Chiba H, Abe S. The environmental risk factors for COPD--tobacco smoke, air pollution, chemicals. Nihon rinsho Japanese Journal of Clinical Medicine. 2003;61(12):2101-6.
9. Russell R, Ford P, Barnes P, Russell R, Ford P, Barnes P. Epidemiology, risk factors and pathophysiology. Managing COPD. 2011:7-28.
10. Maestrelli P. [Pathophysiology of chronic obstructive pulmonary disease]. Ann Ist Super Sanita. 2003;39(4):495-506.
11. MacNee W. Pathology, pathogenesis, and pathophysiology. Bmj. 2006;332(7551):1202-4.

12. Chhabra SK, Dash DJ. Acute exacerbations of chronic obstructive pulmonary disease: causes and impacts. Indian J Chest Dis Allied Sci. 2014;56(2):93-104.
13. Urbano F, Mohsenin V. Chronic obstructive pulmonary disease and sleep: the interaction. Panminerva medica. 2006;48(4):223-30.
14. Stephens MB, Yew KS. Diagnosis of chronic obstructive pulmonary disease. American family physician. 2008;78(1):87-92.
15. Currie GP, Legge JS. ABC of chronic obstructive pulmonary disease. Diagnosis. Bmj. 2006;332(7552):1261-3.
16. Sanders C. The radiographic diagnosis of emphysema. Radiologic Clinics of North America. 1991;29(5):1019-30.
17. Kon SS, Canavan JL, Nolan CM, Jones SE, Clark AL, Polkey MI, et al. The clinical chronic obstructive pulmonary disease questionnaire: cut point for GOLD 2013 classification. American Journal of Respiratory and Critical Care Medicine. 2014;189(2):227-8.
18. Mullerova H, Locantore N, Jones P. GOLD assessment of COPD patients: Impact of symptoms assessment choice. Eur Respiratory Soc; 2012.
19. Jones PW, Harding G, Berry P, Wiklund I, Chen WH, Kline Leidy N. Development and first validation of the COPD Assessment Test. Eur Respir J. 2009;34(3):648-54.
20. JA BM, Martinez TY, FP LG, de Castro Pereira CA. Dyspnea scales as a measure of health-related quality of life in patients with idiopathic pulmonary fibrosis. Medical science monitor: international medical journal of experimental and clinical research. 2002;8(6):CR405-10.
21. Kew KM, Dahri K. Long-acting muscarinic antagonists (LAMA) added to combination long-acting beta2-agonists and inhaled corticosteroids (LABA/ICS) versus LABA/ICS for adults with asthma. Cochrane Database of Systematic Reviews. 2016(1).
22. Anderson DE, Kew KM, Boyter AC. Long-acting muscarinic antagonists (LAMA) added to inhaled corticosteroids (ICS) versus the same dose of ICS alone for adults with asthma. Cochrane Database of Systematic Reviews. 2015(8).
23. Dong Y-H, Hsu C-L, Li Y-Y, Chang C-H, Lai M-S. Bronchodilators use in patients with COPD. International Journal of Chronic Obstructive Pulmonary Disease. 2015:1769-79.
24. Gillissen A. Medikamentöse Therapie der COPD. In: Lingner H, Schwartz F-W, Schultz K, editors. Volkskrankheit Asthma/COPD: Bestandsaufnahme und Perspektiven. Berlin, Heidelberg: Springer Berlin Heidelberg; 2007. p. 249-55.
25. Yang IA, Clarke MS, Sim EHA, Fong KM. Inhaled corticosteroids for stable chronic obstructive pulmonary disease. Cochrane Database of Systematic Reviews. 2012(7).
26. Simeone JC, Luthra R, Kaila S, Pan X, Bhagnani TD, Liu J, et al. Initiation of triple therapy maintenance treatment among patients with COPD in the US. International journal of chronic obstructive pulmonary disease. 2016:73-83.
27. Bhoomadevi M, Ganesh M, Panchanatham N. Designing a conceptual model for patient-centered care–a patient perspective. Res J Sci IT Manag. 2014;3(5):20-4.
28. Vogelmeier C, Hederer B, Glaab T, Schmidt H, Rutten-van Mölken MP, Beeh KM, et al. Tiotropium versus salmeterol for the prevention of exacerbations of COPD. New England Journal of Medicine. 2011;364(12):1093-103.
29. Decramer ML, Chapman KR, Dahl R, Frith P, Devouassoux G, Fritscher C, et al. Once-daily indacaterol versus tiotropium for patients with severe chronic obstructive pulmonary disease (INVIGORATE): a randomised, blinded, parallel-group study. The lancet Respiratory medicine. 2013;1(7):524-33.
30. Currie GP, Douglas JG. Non-pharmacological management. BMJ. 2006;332(7554):1379-81.
31. Heslop K. Non-pharmacological treatment of anxiety and depression in COPD. Nurse Prescribing. 2014;12(1):43-7.
32. Clini E, Costi S, Lodi S, Rossi G. Non-pharmacological treatment for chronic obstructive pulmonary disease. Medical Science Monitor: International Medical Journal of Experimental and Clinical Research. 2003;9(12):RA300-5.
33. Golish JA, Ahmad M. Management of COPD. Postgraduate Medicine. 1977;62(1):131-6.