EDITORIAL

Paradigm Shift in Chronic Obstructive Pulmonary Disease Management: Global Initiative for Chronic Obstructive Lung Disease 2023

Anuj K Pandey[1], Surya Kant[2], Ajay K Verma[3]

Indian Journal of Respiratory Care (2023): 10.5005/jp-journals-11010-1060

The Global Initiative for Chronic Obstructive Lung Disease (GOLD) was structured in 1997 and has released chronic obstructive pulmonary disease (COPD) guidelines annually since 2001. The GOLD update 2023 was released on 14th November 2022; it was the fifth major update of COPD guidelines.[1] Here, we have tried to compile the main changes of the GOLD report 2023, especially in the management of COPD.

A new update has come up with a new way to describe COPD as "a heterogeneous lung condition characterized by chronic respiratory symptoms (dyspnea, cough, sputum production, and exacerbations) due to abnormalities of the airways (bronchitis, bronchiolitis) and/or alveoli (emphysema) that cause persistent and often progressive airflow obstruction."[1,2] Chronic bronchitis is characterized by the presence of a persistent cough and sputum expectoration on a daily basis for a time period.[1] In addition, the new update included a taxonomy (six etiotypes) for COPD—(1) COPD-G, genetically determined COPD; (2) COPD-D, abnormal lung development COPD; (3) environmental COPD (COPD-C, cigarette smoking COPD, COPD-P, biomass, and pollution exposure COPD; (4) COPD-I, infection-related COPD; (5) COPD-A, COPD and asthma; and (6) COPD-U, unknown cause-associated COPD.[3]

As per a new update, 10.3% (8.2–12.9%) is the overall prevalence of COPD, worldwide.[1,4] GOLD's 2023 guideline says that COPD is spreading quickly around the world because more people are smoking in low- and middle-income countries (LMICs) and more older people live in rich countries. At present, three million people worldwide die every year due to COPD. Mortality due to COPD is increasing because smoking is prevalent in poor countries and there is a huge population of older people in rich countries; due to this it is estimated that by 2060, death due to COPD could exceed 5.4.[5] Often, in both smokers and in nonsmokers, COPD percentage is higher in smokers than the nonsmokers. Smoking is a well-established risk factor associated with COPD. Estimates suggest that smoking accounts for 70% of COPD cases in developed countries, compared to 30–40% in LMICs. In the world, three billion people rely on biomass fuels (cow dung cake, coal, wood, gas stoves, etc.) for cooking, heating, and other purposes; this population is at higher risk of COPD. This report concludes that nearly 50% of smoking and 50% of nonsmoking factors are contributors to COPD.[1]

The COPD is caused by intricate, cumulative, as well as gene-environment (GE) interactions for a lifetime (T) that can harm the lungs and/or change typical development and/or aging.[1,6] Further investigations are required to understand the connections and interactions between the host's genetic background and various environmental risk variables over a lifetime, dubbed "GETomics."[1] Smoking tobacco, inhalation of harmful gaseous particles from household and air pollution (indoor and outdoor) are the primary environmental exposures leading to COPD. Additionally, several environmental and host factors, *viz.* aberrant lung development and accelerated lung ageing could contribute. One of the most common genetic risk factors for COPD is a mutation in the *SERPINA1* gene which is responsible for α-1 antitrypsin deficiency.[7]

Patients with breathlessness, chronic cough, sputum, history of lower respiratory tract infections, and/or a history of exposure to risk factors for the disease should be evaluated for COPD; however, a postbronchodilator forced expiratory volume in one second (FEV_1/FVC) of <0.7 is primal for COPD diagnosis. Albeit, using a fixed FEV_1/FVC ratio (<0.7) to characterise airway restriction may result in over and underdiagnosis of disease in the elders, particularly in mild diseases. Some people may have respiratory symptoms; anatomical lung lesions (like emphysema) and physiological abnormalities (including low and normal FEV_1, gas trapping, hyperinflation, decreased lung diffusing capacity, and/or fast FEV_1 decline) without having airflow obstruction. This is possible because some people have a genetic predisposition to develop respiratory diseases. These individuals have been given the diagnosis of "pre-COPD."[8] People who have a normal ratio but aberrant spirometry are said to have "preserved ratio impaired spirometry" (PRISm). The pre-COPD or PRISm have an increased risk of acquiring airflow restriction with time, but this is not true in all cases. The early COPD examination seeks to ascertain the degree of airflow obstruction, COPD's effect on the patient's health, and the likelihood of future events (for example exacerbations, hospitalizations, and mortality) in order to direct treatment.

[1–3]Department of Respiratory Medicine, King George's Medical University, Lucknow, Uttar Pradesh, India

Corresponding Author: Ajay K Verma, Department of Respiratory Medicine, King George's Medical University, Lucknow, Uttar Pradesh, India, Phone: +919919788862, e-mail: ajaykumarverma@kgmcindia.edu

How to cite this article: Pandey AK, Kant S, Verma AK. Paradigm Shift in Chronic Obstructive Pulmonary Disease Management: Global Initiative for Chronic Obstructive Lung Disease 2023. Indian J Respir Care 2023;12(3):211–213.

Source of support: Nil

Conflict of interest: None

When spirometry confirms a diagnosis of COPD, further evaluation must focus on four basic factors to guide treatment—(1) severity of airflow limitation (mild, moderate, severe, and very severe); (2) current symptoms (Modified Medical Research Council Dyspnea Scale, Chronic Respiratory Questionnaire, St. George's Respiratory Questionnaire, COPD Assessment Test Score, COPD Control Questionnaire); (3) history of exacerbations (moderate and/or severe); and (4) additional disorders (heart disease, muscle weakness, metabolic syndrome, bone loss, psychological issues, and lung cancer).

The 2011 revision of the GOLD included an evaluation of symptoms, severity of airflow restriction, and exacerbation history, which replaces the GOLD 2007 (dependent on only spirometric grading) and opens the path to combined COPD assessment.[9] The GOLD 2017 report (ABCD assessment tool) used both the frequency of prior exacerbations and the degree of airflow obstruction to gauge exacerbation risk. The degree of airflow restriction was eventually eliminated from the GOLD 2023 classification due to its less accurate ability to predict outcomes and guide treatment choices at the individual level (compared to the population level), as well as the complexity of its usage by physicians. The GOLD 2023 report suggests the ABCD combined assessment acknowledges the therapeutic importance of exacerbations, regardless of the severity of the patient's symptoms. "A" and "B" groups remain the same, but to emphasize the clinical importance of exacerbations, the "C" and "D" groups have been integrated into one group called "E."[1] Here, it is important to consider that the GOLD 2023 document recognizes that this categorization needs to be supported by relevant clinical studies. In situations where there is a clear discrepancy between the degree of airflow obstruction and symptoms, thorough evaluation is primal toward a better understanding of lung mechanics and structure and comorbidities that may affect patient symptoms.

All "group A" patients should receive bronchodilators (short- or long-acting bronchodilators) for dyspnea. Long-acting bronchodilators are preferred if available and cheap, except for occasional dyspnea. If beneficial, it should be continued. Treatment for "group B" patients begins with a combination of long-acting beta agonist (LABA) and long-acting muscarinic antagonist (LAMA). If a LABA + LAMA combination isn't acceptable, there are no indications to prescribe one type of long-acting bronchodilators (LABA or LAMA) for early symptom reduction in "group B" patients. If these patients have associated diseases that increase their symptoms and impair their prognosis; these should be managed according to standard recommendations. Group E patients should start with LABA + LAMA. COPD patients shouldn't use LABA + ICS. LABA + LAMA + ICS is recommended over LABA + ICS if an ICS is indicated. LABA + LAMA + ICS in group E is practically recommended when the eosinophil count is ≥300 cells/L (ICS affects exacerbation prevention based on blood eosinophil levels). GOLD 2023 recommends using it for those with a high eosinophil count (≥300 cells/L) because there is no direct data on starting triple therapy in newly diagnosed patients. In these situations, ICS is required. In summary, patients in "group A" were advised to use bronchodilators, patients in "group B" were advised to use LABA + LAMA, and patients in "group E" were advised to use LABA + LAMA (or LABA + LAMA + ICS if the blood eosinophil count was ≥300 cells/L). Further, following up on the principles of first reviewing (symptoms, dyspnea, and exacerbation) and assessing (inhaler usage technique and its adherence, pulmonary rehabilitation, etc.), then adjusting (escalation, de-escalation, changing the inhaler device and/or molecule) if necessary, should guide pharmacological management.

An exacerbation of COPD is defined as a worsening of dyspnea, cough, and/or sputum in <14 days, which may be accompanied by tachypnea and/or tachycardia and is often caused by an infection, pollution, or other injury to the airways.[10] Possible indicators for hospitalization should be considered in cases of severe symptoms such as high respiration, lowered oxygen saturation, ineffective initial treatment, drowsiness, acute respiratory failure, the onset of new physical signs, serious comorbidities, inadequate home care, etc.

Patients with elevated mortality risk are identified by a number of factors, including FEV_1, exercise tolerance as measured by peak oxygen consumption or walking distance, weight loss, as well as decreased arterial oxygenation. Body mass, obstruction, dyspnea, and exercise index (BODE index) are useful in better predicting overall survival.

The new guideline offers advice on inhalation therapy.[1] Patient understanding and satisfaction are required when prescribing inhaler devices. Briefly, devices should not be changed frequently until clinically needed. Correct usage of devices is paramount in delivering the medication; hence, proper individual training and education on how to use devices is fundamental. If the patient can make forceful and deep breaths, a dry-powder inhaler (DPI) should be prescribed; metered dose-inhalers (MDI) should be used if the patient can make slow and deep inhalations with and without spaces/valved holding chamber (VHC); if patients are unable to use MDI, then soft mist inhaler (SMI) or DPI nebulizers should be considered.[11]

Vaccines against influenza, COVID-19, pneumonia, whooping cough, and shingles are recommended for patients with stable COPD.[1] Lung cancer is one of the major causes of death in COPD. People with COPD who get it from smoking get a low-dose computed tomography scan once a year, it is not the case in nonsmoking COPD.[12] This approach can result in better treatment for COPD and lung cancer. In COPD patients, bone diseases, depression, and anxiety disorders are frequently overlooked. These disorders should be adequately investigated and treated in COPD patients. Furthermore, it is imperative to provide treatment for comorbidities (like cardiovascular disease, diabetes, periodontitis, obstructive sleep apnea, etc.) while receiving COPD treatment. In the 2023 update, telerehabilitation and COVID-19-related guidelines[13] were also documented.

Since ancient times, tobacco smoking has been a well-known risk factor for COPD. A new report updates that 50% tobacco and 50% non-tobacco-related (air pollution, biomass fuel exposure, etc.) risk factors are common in COPD. It can begin early in life and affect young people and the identification of prior conditions (such as pre-COPD and PRISm) offers new opportunities for early detection, prevention, and initiation of suitable therapy. The amended standards advise practitioners to diagnose COPD patients earlier than they do now. The latest guidelines recommend starting therapies before patients get very unwell. Treatment routes have been modified and streamlined. Some COPD medications are now indicated for earlier disease stages to prevent exacerbations. Shifting from ABCD to ABE management of combined treatment of COPD is altogether termed a paradigm shift in the overall management of COPD. Underdiagnosis and misdiagnosis in COPD lead to inadequate treatment. To overcome it early diagnosis and personalized medication are indispensable. Multiple areas need more research and explanation, including appropriate therapies for distinct etiotypes, blood eosinophil cutoffs for determining choices about treatment, and triple therapy following COPD admissions

to the hospital. Many evidence-based recommendations were included in the new report, in addition to expert opinion and research-based ones that must be proven in the future.

Acknowledgments

We thank to Scientific Committee of GOLD for publishing GOLD Report 2023. The authors are thankful to the Indian Council of Medical Research (ICMR, New Delhi) for providing a fellowship to AKP (No. 3/1/2/1(Env)/2018-NCD-I, dated 19/12/2018).

Orcid

Anuj K Pandey https://orcid.org/0000-0002-7403-9800
Surya Kant https://orcid.org/0000-0001-7520-5404
Ajay K Verma https://orcid.org/0000-0002-2973-1793

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