Editorial

The modified DECAF score: A critical tool in copd patient assessment

Rinkle Gemnani[1]*

[1]Dept. of Medicine, Jawahar Lal Nehru Medical College, Datta Meghe Institute of Medical Sciences (Deemed to be University), Wardha, Maharashtra, India

ARTICLE INFO

A gradual chronic illness, COPD is marked by an irreversible deterioration in health, exercise capacity, and respiratory function.[1] Exacerbations of symptoms, which differ in intensity and frequency across patients as well as during an individual's illness, disrupt this underlying condition. These flare-ups are significant due to their immediate implications on health status, morbidity, and mortality as well as their long-term effects on an individual's life. One of the most significant factors influencing health-related quality of life is the frequency of exacerbations.[2] According to a study, there were more than 250 million COPD patients worldwide in 2016.[3] Between 1990 and 2015, the global prevalence of COPD grew by 44.2%. A systematic review and meta-analysis by Halbert et al. found that the combined prevalence of COPD from 37 studies was 7.6%, while the combined prevalence from 26 spirometry estimates was 8.9%.[4]

Moreover, estimates from 123 worldwide studies' meta-analyses revealed that 11.4% of adults 30 years of age and older had COPD[5]. 63.9 million disability-adjusted life years (DALYs) were attributed to COPD worldwide in 2015. In addition to DALYs, COPD affects the health-related quality of life for its sufferers. Patients with COPD experience a steady reduction in their physical performance as a result of increased symptoms, a decline in lung function, and the presence of risk factors as the disease advances.[6,7]

COPD not only increases the burden of the illness but also increases the cost of care due to direct and indirect medical costs. Working-age individuals (18–65 years old) with COPD incur higher direct and indirect costs and use more resources.[8]

Prolonged exposure to toxic chemicals or particles causes COPD. Worldwide, smoking cigarettes is the leading cause of COPD. Alpha-1 antitrypsin deficiency (AATD), exposure to the environment, and secondhand smoke are possible additional causes.[9] Interestingly, the onset of adult COPD appears to be influenced by childhood exposure to cigarette smoke. Research suggests that tobacco exposure during pregnancy and early childhood significantly increases the risk of developing COPD later in life, as indicated by a systematic review encompassing 16 studies involving 69,365 participants.[10]

Another significant, avoidable cause of outdoor air pollution and a contributing factor to COPD is work-related exposure. Approximately 15–20% of the risk of COPD is linked to these occupational exposures; among those who do not smoke, a considerable percentage (26–53%) of COPD cases are caused by numerous hazards such as dust, fumes, gases, vapors, and secondhand smoke. Studies like the Swiss Cohort Study on Air Pollution and Adult Lung and Heart Diseases have shown a strong correlation between high occupational exposure levels and a higher risk of COPD.[11]

The COPD mortality rate in 2016 for men aged 45 to 64 stood at 22.91 per 100,000 per year, whereas for women, the

* Corresponding author.
E-mail address: rinklegemnani42@gmail.com (R. Gemnani).

figure was 10.26. A recent study reveals that after the age of 65, mortality rates are 403.71 for women and 568.76 for men per 100,000 individuals annually. Similarly, in India, another recent study found a COPD prevalence of 7.3% in men and 4.8% in women aged 45 to 49, and 28.6% in men and 17.6% in women aged 65 to 69. [12,13]

When COPD is suspected, spirometry is employed to confirm the diagnosis. However, there is insufficient evidence to support the routine use of spirometry screening in asymptomatic individuals. Diagnosing COPD early requires a high level of suspicion. Individuals with airflow limitation indicative of COPD are characterized by a forced vital capacity (FVC) ratio of less than 70% and a forced expiratory volume in one second (FEV1) less than 80% of the predicted value post-bronchodilator administration. Since the FEV1/FVC ratio naturally declines with age, it is crucial to compare results to age-specific norms before confirming the diagnosis.

Developed to predict mortality risk in patients with COPD, the Modified Dyspnea, Eosinopenia, Consolidation, Acidemia, and Frequency of hospital stay (in last one year) (DECAF) score is a risk stratification measure that may be readily used at the bedside to direct therapy, such as hospital admission or at-home treatment for low-risk patients. The indices used by the modified DECAF score are regularly provided upon admission. The extended Medical Research Council Dyspnea score is used to quantify stable state dyspnea, which is the strongest of the five predictors included in the score. This score is better than other prognostic scores because it is a precise and simple-to-score predictor for patients with COPD. The majority of patients with COPD may be accurately classified as low risk by the modified DECAF score, and the risk stratification of the modified DECAF score in the high-risk population increases dramatically with an increase in the cut-off value.

This score is a strong predictor of hospital mortality from COPD exacerbation. Patients might be categorized into low-risk (scores 0–2), intermediate-risk (scores 3-5), and high-risk (scores 4–6) groups based on their Modified DECAF scores. The length of hospital stay rises as there is a rise in the modified DECAF scores indicating that it can be useful in differentiating between patients who can safely be dismissed early and those who will require longer hospital stays. To determine which patients can be managed in a general ward and which ones require intensive care and ventilator support, the doctor can also use the modified DECAF score to guide treatment. Validity, usefulness, and practicality are all important components of a clinical prediction tool. The modified DECAF score is a useful and straightforward tool to compute with a few straightforward questions and standard research thus being a reliable method to predict an outcome in patients with COPD.

Conflict of Interest

None.

References

1. Kanner RE, Anthonisen NR, Connett JE. Lower respiratory illnesses promote FEV(1) decline in current smokers but not ex-smokers with mild chronic obstructive pulmonary disease: results from the lung health study. *Am J Respir Crit Care Med*. 2001;164(3):358–64.
2. Talwar D, Kumar S, Acharya S, Talwar D, Jain S, Acharya N, et al. Implication of chronic obstructive pulmonary disease on the severity and outcome of covid-19 in rural India; Is COPD the hidden culprit amongst the havoc: A cross-sectional study. *Med Sci*. 2022;26(122):131.
3. Halbert RJ, Natoli JL, Gano A, Badamgarav E, Buist AS, Mannino DM, et al. Global burden of COPD: systematic review and meta-analysis. *Eur Respir J*. 2006;28(3):523–55.
4. Adeloye D, Chua S, Lee C, Basquill C, Papana A, Theodoratou E. Global and regional estimates of COPD prevalence: Systematic review and meta-analysis. *J Glob Health*. 2015;5(2):20415.
5. Dransfield MT, Kunisaki KM, Strand MJ, Anzueto A, Bhatt SP, Bowler RP. Acute Exacerbations and Lung Function Loss in Smokers with and without Chronic Obstructive Pulmonary Disease. *Am J Respir Crit Care Med*. 2017;195(3):324–54.
6. Hurst JR, Vestbo J, Anzueto A, Locantore N, Müllerova H, Singer RT. Susceptibility to Exacerbation in Chronic Obstructive Pulmonary Disease. *N Engl J Med*. 2010;363(12):1128–66.
7. Han MK, Kazerooni EA, Lynch DA, Liu LX, Murray S, Curtis JL, et al. Chronic Obstructive Pulmonary Disease Exacerbations in the COPDGene Study: Associated Radiologic Phenotypes. *Radiology*. 2011;261(1):274–82.
8. Tayde PS, Kumar S, Tayde PS. Chronic obstructive pulmonary disease in the elderly: evaluation and management. *Eur J Intern Med*. 2013;25(4):320–8.
9. Bhatt SP, Kim Y, Lutz SM, Harrington KF, Hokanson JE. Smoking duration alone provides stronger risk estimates of chronic obstructive pulmonary disease than pack-years. *Thorax*. 2018;73(5):414–35.
10. Doiron D, Hoogh D, Hensch KP, Fortier N, Cai I. Air pollution, lung function, and COPD: results from the population-based UK Biobank study. *Eur Respir J*. 2019;54(1):1802140.
11. Salvi S, Kumar GA, Dhaliwal RS, Paulson K, Agrawal A, Koul PA, et al. The burden of chronic respiratory diseases and their heterogeneity across the states of India: The Global Burden of Disease Study 1990-2016. . *Lancet Glob Health*. 2018;6(12):1363–74.
12. Wang C, Xu J, Yang L, Xu Y, Zhang X, Bai C. Prevalence and risk factors of chronic obstructive pulmonary disease in China (the China Pulmonary Health [CPH] study): a national cross-sectional study. *The Lancet*. 2018;391(10131):1706–23.
13. Sharma S, Khan K, Yadav GS, Koolwal S. A study of modified DECAF score in predicting hospital outcomes in patients of acute exacerbation of chronic obstructive pulmonary disease at SMS Medical College, Jaipur. . *Adv Hum Biolo*. 2020;10:171–6.

Author biography

Rinkle Gemnani, Postgraduate Resident