Occurence, diagnosis and treatment of chronic obstructive pulmonary disease.

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Introduction

Although some patients' lung-function trajectories are milder, the major pathophysiology of COPD is irreversible obstruction of the airway with progressive lung function decline, especially in patients with continuous exposure to risk factors such as cigarette smoke, biomass smoke exposure, and air pollution. As a result, it's critical to slow the deterioration of lung function in individuals who don't fulfil the diagnostic criteria for COPD but are at risk of progressing to overt COPD (pre-COPD). Furthermore, early detection of preclinical COPD in individuals with fixed obstruction on spirometry but no or very moderate symptoms before clinically significant deterioration, such as reduction in lung function, development of symptoms, or acute exacerbation, may be critical.

Early life disadvantages are barriers to proper lung development or alveolar formation from conception through puberty. The formation of bronchi begins with the onset of lung buds on days 21–28 of pregnancy and continues with dichotomous and lateral branching of lung epithelium to form the final structure of the bronchial tree on week's 3–17 of pregnancy (embryonic, pseudoglandular stage). At gestational weeks 16–26 (canalicular stage), bronchial epithelial cells develop into ciliated, nonciliated, and secretory cells, followed by the saccular stage, when terminal acinar tubules branch and the air space expands from gestational week 24 until delivery.

Early life disadvantages that may obstruct lung development, resulting in a reduction in lung function at a young age. Smoking by mothers during pregnancy, low birthweight, and premature delivery have all been linked to deteriorated lung function. Smoking at a young age or passive smoking, asthma, a history of respiratory infection, and chronic bronchitis are all linked to an increased risk of lung function impairment from infancy through adolescence. These drawbacks in early life make it difficult to obtain adequate peak lung function, which is normally reached around the age of 20. Lung function tends to plateau or slightly grow from the early twenties to the mid-thirties. Tobacco smoke exposure shortens this time and may result in early deterioration of lung function [1].

Smoking is not only linked to the above-mentioned early-life problems, but it also hastens the decrease of lung function in people of all ages. The extent to which smoking reduces lung function varies by individual, and this can be linked to a variety of factors such as genetic predisposition, sex, and ethnicity. As a result, only a small percentage of smokers develop COPD throughout the course of their lives. Furthermore, stopping smoking reduces smokers' steeper lung trajectories to a gradient similar to non-smokers.

Risk factors

The greatest significant risk factor for COPD is smoking. The negative effects of tobacco smoking on lung function start as early as the embryonic stage, when the mother is exposed to it [2]. Spirometry revealed a lower expiratory flow volume in 8863 non-smoking children aged 8–12 years who were exposed to mother smoking. Furthermore, a 21-year follow-up of a longitudinal prospective research found a significant link between mother smoking during pregnancy and a decline in FEV1 in male patients. Secondhand smoking exposure, primarily from parents, from infancy through young adulthood is also a significant risk factor for adult obstructive lung disease. Finally, the most important risk factor for COPD is tobacco smoking, which has been well documented from childhood to old life.

Other risk variables, including as childhood infections or respiratory disorders like asthma, biomass smoke exposure, air pollution exposure, and occupational exposures, are equally relevant, but are sometimes disregarded in the evaluation of COPD patients. Non-smoking-related COPD accounts for 25–45 percent of COPD patients, which is significantly more than what doctors often estimate.

Because of the pathophysiological differences between COPD and asthma, most large-scale COPD studies or randomised controlled trials have excluded individuals with asthma. Because the aetiology of asthma is mostly chronic allergic airway inflammation with airway variable, including asthma patients in the COPD arm may introduce severe bias. However, compared to lung function in people without asthma, a 15-year follow-up of lung function in asthma patients revealed a significant decline in FEV1. Furthermore, individuals with a history of early or late asthma have a 10–20-fold increased risk of airway obstruction, and these patients may develop to early COPD, according to prior research [3].

Airway hyperresponsiveness is the most major risk factor for COPD, accounting for 15–17% of new cases in young people after smoking. According to the ATS roundtable criteria and

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modified Spanish COPD recommendations, patients with a history of asthma who acquire COPD later in life are classified as asthma–COPD overlap (ACO). These people are more symptomatic, have more exacerbations, and are more likely to be hospitalised. The burning of biomass fuel, to which nearly 3 billion people are exposed globally, may be one of the most important non-tobacco risk factors, with the proportion being larger in developing nations, where it may harm more than 80% of the population. This level of exposure is nearly three times that of cigarette smoking, and because biomass fuel use is higher in underdeveloped countries, COPD caused by biomass smoke exposure may have been underestimated.

When compared to patients with smoking-related COPD, these biomass smoke exposure-related COPD patients are more likely to be female, with improved lung function but similar symptoms, exercise capacity, and quality of life (QOL). Furthermore, earlier research has demonstrated that COPD caused by biomass smoke exposure mostly affects the airways, with less emphysematous alterations in the lungs than COPD caused by smoking. In individuals with suspected early COPD, genetic variables should be considered. Cutis laxa, Marfan syndrome, and Ehlers-Danlos syndrome, which all include emphysema, lung blebs, and pneumothorax, may be linked to COPD [4]. Furthermore, several genetic variants, such as CHRNA3/5, HHIP, and FAM13A, have been linked to a hereditary risk of COPD. Further research is required before these findings may be used in clinical practise.

Physiological Tests

The most essential physiological test in COPD patients is the pulmonary function test (PFT), which verifies airway blockage and quantifies the degree of airflow restriction. In the criterion established by Martinez et al., evidence of rapid FEV1 decrease >60 mL/year is one of the selected conditions for diagnosis of early COPD. The 60 mL/year criterion was calculated by almost double the typical fall in FEV1 in nonsmokers, which is 25–30 mL/year. Several features of the spirometry test, in addition to FEV1, may help predict people who are in the early stages of COPD. Although forced expiratory flow between 25% and 75% of vital capacity (FEF25–75) has been linked to minor airway blockage, it has only been used in a limited number of studies owing to subject variability. FEF25–75, on the other hand, should be reconsidered in the early identification of COPD since early alterations in the pathophysiology of COPD occur mostly in the small conducting airways. Lung hyperinflation and air trapping are common symptoms of COPD, which can occur in both severe and moderate forms. The ratio of residual volume to total lung capacity (RV/TLC) has been found in studies to be useful in the diagnosis and evaluation of early COPD. Pre-COPD and preclinical COPD may have elevated RV/TLC, which predicts poor lung function and a greater bronchodilator response. As will be detailed in a subsequent section, radiologically assessed RV/TLC has also been explored. Furthermore, as the respiratory rate rises in response to activity, lung hyperinflation may become more pronounced, leading in exertional dyspnea [5].

Conclusion

Early COPD is gaining popularity as researchers seek to better understand the pathophysiological and clinical changes that occur in the early stages of the disease, as well as to identify at-risk or undiagnosed COPD patients before they develop overt COPD and to provide the most effective treatment for preventing disease progression and alleviating clinical deterioration. Previously, early COPD was defined differently, and it was frequently based on minor airway obstruction, which is similar to mild COPD.

References

1. Guerra S, Martinez FD. The complex beginnings of chronic obstructive pulmonary disease. Am J Respir Crit Care Med. 2020;201(16):641-42.
2. Wijnant SRA, De Roos E, Kavousi M, et al. Trajectory and mortality of preserved ratio impaired spirometry: The Rotterdam Study. Eur Respir J. 2020;55(1):1901217.
3. Grant T, Brigham EP, McCormack MC. Childhood origins of adult lung disease as opportunities for prevention. J Allergy Clin Immunol Pract. 2020;8(3):849-58.
4. Çolak Y, Nordestgaard BG, Vestbo J, et al. Prognostic significance of chronic respiratory symptoms in individuals with normal spirometry. Eur Respir J. 2019;54(3):1900734.
5. Rennard SI, Drummond MB. Early chronic obstructive pulmonary disease: Definition, assessment, and prevention. Lancet. 2015;385(9979):1778-88.

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