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REVIEW

Predictors of mortality in COPD

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Summary
Chronic obstructive pulmonary disease (COPD) is a major cause of morbidity and mortality in adults. Although FEV_1 remains the most important physiologic indicator of the severity of airflow obstruction in COPD, its predictive value for mortality is weak when it is higher than 50% of predicted. Furthermore, other easily obtainable clinical variables predict mortality better than the FEV_1 in COPD patients with a wide range of airflow limitation. Chief among these predictors are functional dyspnea, exercise capacity, and the body mass index (BMI), although emerging research suggests a potential role for biomarker profiles in outcome predictions. The validated multidimensional BMI (B), degree of airflow obstruction as expressed by the FEV_1 (O), dyspnea with the modified medical research council (D), and exercise (E) measured with the 6 min walk or BODE index encompasses the predictive validity of the best of these variables into a single surrogate measure of disease severity and survival. This article reviews these predictors of mortality in COPD.

Contents

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Chronic obstructive pulmonary disease (COPD) is now the fourth leading cause of death worldwide[1] and it will become the third leading cause of death worldwide by 2020.[2,3] This increasing mortality is attributed to the smoking epidemic and the aging of the world population. The prevalence of COPD has increased as mortality from heart attacks and strokes, both of which share a common risk factor with COPD (ie, cigarette smoking), has decreased.[4] In 2004, the United States ranked third highest among 18 industrialized countries in COPD mortality for females and fourth highest for males.[5]

Although the hallmark feature of COPD is airflow limitation resulting from chronic bronchitis or emphysema, COPD is often accompanied by multiple comorbidities that lead to a spiral of decline, with an increased risk for mortality. The extent to which specific comorbidities and symptoms may be used to predict COPD mortality is unclear. This article reviews potential predictors of mortality in COPD.

Forced expiratory volume in 1 second

COPD is a complex disease with many inflammatory pathways that initiate and potentiate the disease process (Fig. 1). Neutrophils, macrophages and CD8 + T-lymphocytes are the key inflammatory cell types involved in COPD.

These cells release the reactive oxygen species (ROS), chemokines (e.g. interleukin [IL]-8), cytokines (e.g. tumor necrosis factor [TNF]-α) and proteases (e.g. neutrophil elastase and matrix metalloproteinase) that are instrumental in producing a chronic inflammatory state.[6,7]

The ongoing inflammatory process leads to enlargement of the alveolar spaces, fibrosis, destruction of the lung parenchyma, loss of elasticity and small airways obstruction (obstructive bronchiolitis). Mucus hypersecretion is a prominent feature of COPD. In contrast to asthma, airway hyper-responsiveness is not a commonly prevalent feature of COPD.[6,7] Notably, histological studies reveal that even

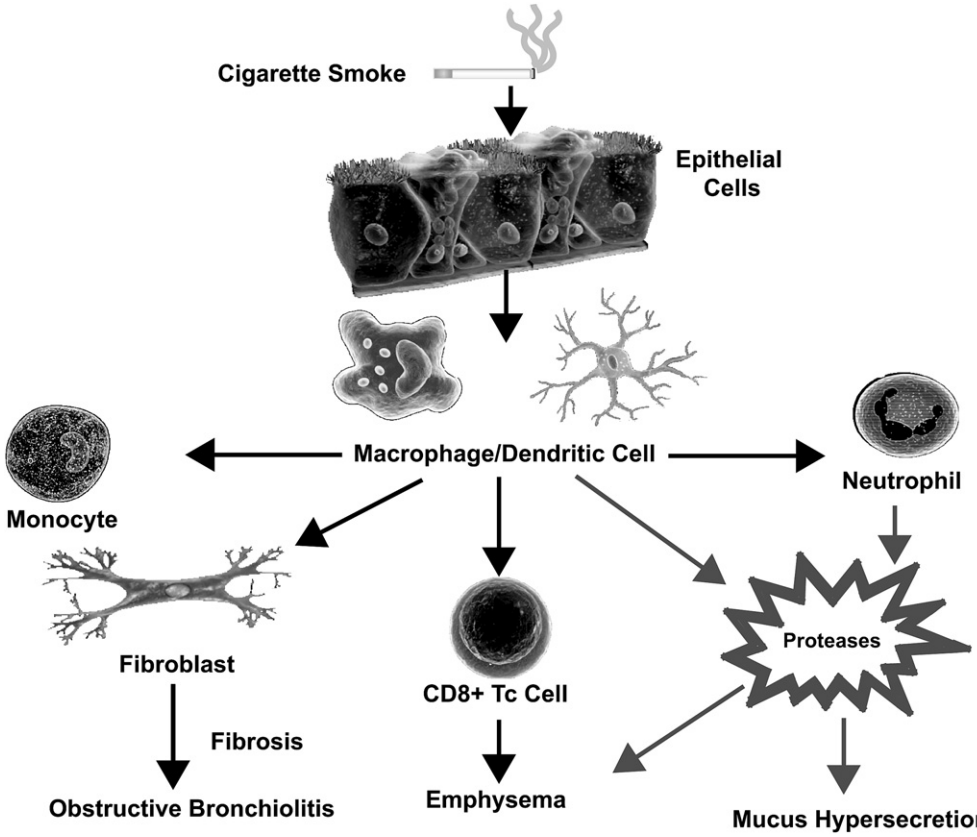

Figure 1 COPD Results from Inflammation. Legend: Airway inflammation in COPD is characterized by a neutrophilic inflammation with increased numbers of macrophages and CD8 + T-lymphocytes. These cells release the reactive oxygen species (ROS), chemokines (e.g. interleukin [IL]-8), cytokines (e.g. tumor necrosis factor [TNF]-α) and proteases (e.g. neutrophil elastase and matrix metalloproteinase) that are instrumental in producing a chronic inflammatory state.[6,7] Source: Barnes, Hansel. Lancet 2004;364:985.[6] (no permissions required).