COMMENTARY ARTICLE

COPD comorbidities and systemic symptoms

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ABSTRACT

COPD is characterised by an abnormal/excessive inflammatory response of the lungs to respiratory contaminants, primarily cigarette smoke.

Apart from the classic pulmonary pathology of COPD (i.e. chronic bronchitis and emphysema), many effects happening outside the lungs, the so-called systemic effects of COPD, have recently been reported. These impacts are clinically significant because they affect the disease's categorization and therapy.

The following systemic impacts of chronic obstructive pulmonary disease are discussed in this : 1) systemic inflammation; 2) dietary problems and weight loss; 3) skeletal muscle failure; and 4) additional systemic consequences The potential causes and clinical implications for each of these are examined, as well as areas that require further research.

Key Words: *Emphysema; Inflammation; Chronic Bronchitis*

INTRODUCTION

According to the definition of the European Respiratory Society(ERS), chronic obstructive pulmonary disease a disor- a disorder characterised by reduced maximum expiratory flow and slow forced emptying of the lungs due to varying combinations of diseases of the airways and emphysema [1]. This definition, as well as those published by many other societies and organisations [2] focuses exclusively on the lungs. Thus, it is not surprising that, in the staging and prognosis of the disease, only pulmonary variables such as forced expiratory volume in one second (FEV1) or arterial oxygen tension (Pa,O2) have been considered and that current therapy targets almost exclusively the lungs [3]; and coexisting cardiovascular illness were the five clusters found and designated based on the major clinical features, comorbidities, and mortality risk. A trained regularised discriminant model analysis was used to validate the clusters in an independent cohort.

This is beginning to change, as evidence from recent studies shows that COPD is frequently associated with significant extrapulmonary abnormalities, known as "systemic effects of COPD." There is a growing awareness that these systemic impacts are clinically relevant and can help with disease knowledge and therapy. The increased work of breathing and dynamic hyperinflation that arise from the airflow limitation characteristic of COPD have historically been explained by the increased labour of breathing and dynamic hyperinflation that come from the airflow limitation characteristic of COPD.

Several recent investigations, however, have convincingly demonstrated that skeletal muscular dysfunction (SMD) is frequently a major factor to activity limitation in these patients. This is beginning to change, as evidence from recent studies shows that COPD is frequently associated with significant extrapulmonary abnormalities, known as "systemic effects of COPD." There is a growing awareness that these systemic impacts are clinically relevant and can help with disease knowledge and therapy. The increased work of breathing and dynamic hyperinflation that arise from the airflow limitation characteristic of COPD have historically been explained by the increased labour of breathing and dynamic hyperinflation that come from the airflow limitation characteristic of COPD. Several recent investigations, however, have convincingly demonstrated that Skeletal Muscular Dysfunction (SMD) is frequently a major factor to activity limitation in these patients. The discovery of SMD as a major systemic effect of COPD has sparked a significant increase in interest in skeletal muscle physiology in COPD patients, resulting in a better understanding of the role of exercise training and rehabilitation programmes in the clinical management of these patients. Weight loss, another systemic impact of COPD, has been shown in previous studies to be a negative prognostic factor in these patients, even when other prognostic indicators based on the degree of pulmonary dysfunction, such as FEV1 or Pa,O2, are used. Furthermore, unlike alterations in FEV1 and/or Pa, O2, weight loss in COPD.

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Is reversible with appropriate management, and, whenever this happens, the prognosis improves[4]. Weight loss and SMD have been identified as systemic impacts of COPD, emphasising the significance of dietary support, typically in combination with exercise programmes, for improving quality of life and prognosis in these patients. Finally, several recent studies have conclusively demonstrated that COPD is associated not only with an abnormal inflammatory response of the lung parenchyma , but also with evidence of systemic inflammation, such as systemic oxidative stress, activation of circulating inflammatory cells, and increased levels of proinflammatory cytokin.

Systematic Inflammation

A primary pathogenic mechanism in COPD is an excessive/ inadequate inflammatory response of the lungs to a variety of toxic inhaled chemicals or particles (mainly cigarette smoke). The lung inflammatory response is characterised by: 1) increased numbers of neutrophils, macrophages, and T-lymphocytes with a CD8+ predominance; 2) increased concentrations of proinflammatory cytokines, such as leukotriene B4, interleukin (IL)-8, and tumour necrosis factor (TNF)-, among others; and 3) evidence of oxidative stress caused by inhalation of oxidants (tobacco smoke)[5]. The activated inflammatory cells similar inflammatory alterations, such as signs of oxidative stress, the presence of activated inflammatory cells, and higher plasma levels of proinflammatory cytokines, can be observed in the systemic circulation of these patients[6]. This notion is crucial to comprehending COPD's systemic consequences[7].

Systemic stress

All functional or structural changes generated by reactive oxygen species (ROS) are referred to as oxidative stress. Because of their short half-life, measuring ROS directly in vivo is difficult . As a result, determining ROS levels requires proof of biological effects or fingerprints. In nonsmokers, healthy smokers, and COPD patients, Rahman et al. 19 determined the Trolox-equivalent antioxidant capacity and levels of lipid peroxidation products in plasma as indices, or fingerprints, of overall oxidative stress during both clinically stable periods and exacerbations of the disease. Several studies have found changes in various circulating inflammatory cells, such as neutrophils and lymphocytes, in COPD patients, albeit the former has been investigated more extensively. He discovered that neutrophils from COPD patients had increased chemotaxis and extracellular proteolysis. In another investigation, found that COPD patients' circulating neutrophils produced more ROS, or "respiratory burst," than nonsmokers or healthy smokers, both at rest and after stimulation in vitro. The same authors found that patients with persistent COPD had higher levels of expression of many surface adhesion molecules, including Mac-1 (CD11b), in circulating neutrophils than healthy controls . Surprisingly, this difference vanished with illness flare-ups, implying neutrophil sequestration. The pulmonary inflammatory response in COPD is characterised by a low CD4+/CD8+ ratio. Because the bulk of research addressing this topic evaluated peripheral T-cell subsets in smoking and nonsmoking people and did not include patients with COPD, it is unclear if this aberration is replicated in the systemic circulation in these patients.

He found no significant differences in the total number of T-lymphocytes and T-cell subsets in light or moderatesmokers compared to nonsmokers, but he did find that in heavy smokers, the number of circulating CD8+ T-cells increased while the number of CD4+ cells decreased; interestingly, these differences disappeared 6 weeks after smoking cessation.

Increased levels of proinflammatory cytokines in the blood

Several investigations have found that individuals with COPD had higher levels of circulating cytokines and acute phase reactants in their peripheral circulation[8]. TNF-, its receptors (TNFR-55 and TNFR-75), IL-6, IL-8, C-reactive protein, lipopolysaccharide-binding protein, Fas, and Fas ligand are among the abnormalities. These anomalies were detected in patients who were clinically stable, but they were more prominent during illness exacerbations. It's worth noting that during the Southeast Asian haze of 1997, a cytokine profile that included higher levels of IL-6, IL-1, and granulocyte-macrophage colony-stimulating factor (GM-CSF) was recently described in healthy persons.

Weight loss and nutritional deficiencies

Weight loss in COPD is mostly caused by the loss of skeletal muscle mass, with fat mass loss contributing to a lesser amount. However, even if there is no clinically significant weight loss, changes in body composition can occur in COPD. The utilisation of sophisticated equipment, such as dual-energy X-ray absorption or bioelectrical impedance tests, is required to identify these more subtle changes. We were able to reveal substantial changes in body composition (lean mass, fat mass, and bone mineral content) between patients with COPD and healthy volunteers, as well as COPD patients with predominantly chronic bronchitis and COPD patients with mostly emphysema, using this technology. The causes of these dietary anomalies are unknown. As previously stated, decreased calorie intake does not appear to be a common occurrence in these patients, except during periods of disease exacerbation. Most COPD patients, on the other hand, have a higher basal metabolic rate, and because this increased metabolic demand is not met by a corresponding increase in caloric intake, weight loss occurs. The reason for the higher basal metabolic rate is still unknown. It has traditionally been explained by an increased oxygen consumption ($V'O2$) of the respiratory muscles as a result of the disease's increased work of breathing.

Patients with COPD frequently complain of dyspnoea and exercise intolerance as a result of a pathological rate of loss in lung function with age. The idea that exercise intolerance in COPD was caused by dyspnoea, which was caused by increased labour of breathing due to airflow restriction, was initially questioned by Killian and colleagues, who demonstrated that many COPD patients cease exercising due to leg weariness rather than dyspnoea. This was most likely the first hint that skeletal muscle was aberrant in COPD, and it sparked a lot of interest in the subject. SMD is frequent in COPD patients, according to several studies, and it plays a key role in restricting their exercise capacity and quality of life[8].

COPD patients generally adopt a sedentary lifestyle due to shortness of breath during exertion. Physical inactivity results in a net loss of muscle mass, a reduction in muscle force generating capacity, and a decrease in fatigue resistance. Sedentarism is believed to be a majorcontributor to SMD, as exercise training improves muscular function in COPD patients. However, total normalisation of muscle physiology is rarely accomplished following rehabilitation, and several biochemical anomalies detected in muscles are unlikely to be explained by physical inactivity[9].

Several observations point to tissue hypoxia as a possible pathogenic factor in the development of SMD in COPD patients. For starters, prolonged hypoxia lowers the expression of myosin heavy chain isoforms and suppresses protein synthesis in muscle cells. Second, healthy people lose muscle mass when they are at a high altitude (hypobaric hypoxia). Finally, structural (reduction of type I fibres) and functional upregulation of mitochondrial cytochrome oxidase changes in skeletal muscle from individuals with COPD and chronic respiratory failure are proportionate to the severity of arterial hypoxiaemia. If tissue hypoxia is a pathogenic factor in COPD, domiciliary oxygen therapy may help to reduce SMD. This possibility should be investigated further.

Systemic inflammation is believed to play a role in the pathogenesis of SMD in COPD patients. COPD patients have higher plasma levels of a range of proinflammatory cytokines, including TNF-, as previously mentioned. Furthermore, circulating monocytes from these patients release more TNF- in vitro than healthy controls 45, and multiple studies have recently demonstrated higher plasma concentrations of soluble TNF- receptors. TNF- has a variety of effects on muscle cells . TNF stimulates the transcription factor nuclear factor-B and degrades myosin heavy chains through the ubiquitin/proteasome complex (U/P) 90 in differentiated myocites examined in vitro.