A Comprehensive Review of the Association between Sarcopenia and COPD

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Abstract. COPD is a complex disease with pulmonary and extrapulmonary manifestations intensively studied due to the numerous pathologic processes involved. Its prevalence is increasing, representing the fourth leading cause of mortality worldwide. Sarcopenia can occur in COPD patients with common risk factors. Sarcopenia is characterized by a decrease in muscle mass and function with consequences on muscle performance. Muscle changes can be measured by different methods: MRI, DXA, BIA, CT or biopsy. The prevalence of sarcopenia has been studied in numerous studies with varying results. This review identifies the main risk factors that contribute to the variable outcomes with a focus on the characteristics of the studied population, the criteria for defining sarcopenia and the methods used to measure muscle mass, strength and physical performance.

Keywords: sarcopenia, pulmonary disease, prevalence, muscle wasting, sedentary lifestyle, frailty, body composition, malnutrition, body mass index.

Abbreviations

COPD – Chronic Obstructive Pulmonary Disease
DXA – Dual-energy X-ray Absorptiometry
BIA – Bioelectrical Impedance Analysis
CT – Computed Tomography
MRI – Magnetic Resonance Imaging
GLIM – the Global Leadership Initiative on Malnutrition

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a severe, complex, multisystem condition primarily caused by cigarette smoking [1], [2], predominantly affecting the elderly population. COPD is a significant public health issue and the fourth leading cause of mortality worldwide [3]. In order to diagnose COPD, it is necessary to perform spirometry both before and after administering a bronchodilator. Obtaining an FEV1/FVC ratio value lower than 0.7 indicates the presence of airway obstruction. To determine the severity of the obstruction, the GOLD classification is used [11]. Pulmonary manifestations of COPD include airflow limitation, resulting from structural changes in the airways and alveoli. In addition to lung-related symptoms, COPD is often associated with several extrapulmonary manifestations, such as osteoporosis, skeletal muscle dysfunction, diabetes, cardiovascular diseases, and sarcopenia [1], [4].

Sarcopenia is another complex condition, recently recognized as a geriatric syndrome, typically seen in elderly patients. There are several committees that have defined sarcopenia. The presence of low muscle mass along with low muscle strength or a decline in physical performance alone is enough to make the diagnosis. All these changes lead to an increased risk of falls, fractures and increased mortality [5], [17]. To assess muscle mass, DXA, MRI, CT and BIA can be used [7]. Sarcopenia can be classified as either primary, occurring due to aging, or secondary, resulting from other associated conditions (COPD, cancer, heart failure) [6].

 DOI: 10.2478/rjim-2026-0003

Importantly, sarcopenia can develop in COPD patients at any age.

Considering that both conditions predominantly affect older adults, studying the association between sarcopenia and COPD is essential for understanding the extent of their co-occurrence, shared underlying mechanisms, and their impact on patient outcomes. Additionally, this association becomes increasingly relevant in the context of an aging population, leading to a higher prevalence of these diseases. The objectives of this review are to determine the prevalence of sarcopenia among COPD patients, identify common risk factors, and explore the shared pathophysiological processes. Understanding these factors can guide the development of targeted treatments aimed at improving prognosis and enhancing the quality of life in affected patients.

MATERIALS AND METHODS

A thorough literature search was conducted using PubMed, Scopus, Web of Science, and Embase, employing the following search terms: sarcopenia OR muscle atrophy, COPD or chronic obstructive pulmonary disease, skeletal muscle dysfunction in COPD, COPD and frailty, pulmonary rehabilitation and sarcopenia. The search was limited to full-text articles published between 2006 and 2024. Studies were selected based on their relevance to the association between sarcopenia and COPD, focusing on human populations and studies that utilized validated diagnostic criteria for both conditions. After careful evaluation of titles, abstracts, and full-texts, a total of 41 studies were included in this review.

INCLUSION AND EXCLUSION CRITERIA

The inclusion criteria were:

– Studies conducted on human subjects aged 18 and older;

– Articles that explicitly addressed the association between sarcopenia and COPD;

– Studies that measured muscle mass, strength, or function using validated assessment tools, such as dual-energy X-ray absorptiometry (DXA) or bioelectrical impedance analysis (BIA), combined with spirometry for COPD diagnosis;

– Randomized controlled trials, cohort studies.

DISCUSSIONS

Both sarcopenia and COPD are complex conditions primarily affecting the elderly and the locomotor system, sharing common etiologies and risk factors. A 2019 meta-analysis demonstrated a sarcopenia prevalence of 21.6% among COPD patients [2], and recent data shows an increase to 27.5%, highlighting a growing trend [7]. The most significant risk factor for COPD is cigarette smoking, which has also been linked to sarcopenia in one study [8]. Factors leading to sarcopenia in patients with COPD include: age, body mass index, smoking, diabetes and severity of lung disease according to the GOLD classification [9].

The association between sarcopenia and COPD has significant negative implications for patient prognosis. This manifests as a lower quality of life, an increased risk of hospitalization, and reduced exercise tolerance [3]. A 2018 study found that COPD patients with sarcopenia exhibit poorer lung function, diminished muscular indicators, and a decline in body composition compared to COPD patients without sarcopenia. This association has been correlated with lower FEV1 (forced expiratory volume in one second), FEV1/FVC (forced vital capacity) ratios, skeletal muscle mass, BMI (body mass index), hemoglobin, and albumin levels. Additionally, the presence of sarcopenia is associated with a higher number of acute COPD exacerbations [3], [10].

Spirometry before and after bronchodilator administration is necessary to diagnose COPD. Obtaining an FEV1/FVC ratio <0.7 after bronchodilator administration is diagnostic of reversible incomplete airway obstruction. After the diagnosis of COPD, it is necessary to determine the severity of the disease using the GOLD classification. The mild form of the disease (GOLD 1) is characterized by a predicted FEV1 ≥80%, the moderate form (GOLD 2) by a predicted FEV1 between 50–80% and the severe form (GOLD 3) by a predicted FEV1 between 30% and 50%. The most severe form, GOLD 4, has an FEV1 <30% of predicted [11].

In COPD patients, one of the extrapulmonary manifestations is the progressive loss of both muscle mass and function, leading to a deterioration in quality of life as the disease advances [2]. Muscle impairment is more pronounced in the lower limbs compared to the upper limbs and does not spare the respiratory [12], [13]. Typically, in the elderly, an increase in the proportion of type 1 muscle fibers is observed. However, in COPD patients, a shift in muscle fiber composition has been demonstrated, with a transition from type 1 to type 2 fibers, which are more susceptible to atrophy-inducing factors [7], [14]. This shift is correlated with disease severity and is more pronounced in women, who present with a smaller cross-sectional area of type II fibers compared to men [15].

A key aspect is that the severity of muscle atrophy increases with the severity of the disease and is worsened by acute exacerbations [13]. As COPD progresses, muscle dysfunction in the limbs, particularly the lower limbs, is associated with reduced exercise capacity, poorer prognosis, and increased mortality [7]. The muscles most affected are those of the thighs, with emphasis on the quadriceps muscle, which exhibits reduced mass and strength, a shift in fiber composition, and progression toward atrophy [12], [16]. One study highlighted that one-third of COPD patients exhibit quadriceps dysfunction even in the early stages of the disease [13]. However, another study demonstrated that more than 50% of patients with severe COPD did not show reduced quadriceps strength, suggesting the existence of variable phenotypes in the muscle response to the disease [12], [16]. Acute exacerbations negatively affect muscles, with long recovery periods – over three months – required for restoring muscle mass and function [17].

Moreover, COPD represents a major risk factor for the development of secondary sarcopenia, while aging is responsible for primary sarcopenia. The two conditions share common risk factors that impact muscle cellular signaling, such as systemic inflammation (the presence of inflammatory cytokines), malnutrition, reduced physical activity, hormonal imbalances, and hypoxia [2], [7]. These factors, acting individually or synergistically, negatively affect muscle tissue, leading to muscle mass reduction and metabolic deterioration, with consequences on muscle repair and regeneration processes [18].

Muscle mass decline begins at age 50, with a 1–2% annual reduction in muscle mass [19]. With aging, the loss of neuromuscular connections, muscle mass, and strength become more pronounced, particularly in type 2 fibers [20].

Systemic inflammation is evidenced by elevated blood levels of inflammatory markers such as TNF-α, C-reactive protein, IL-1, and IL-6, all of which are involved in muscle degradation and inhibition of protein synthesis. During COPD exacerbations, these inflammatory markers increase significantly, accelerating muscle mass decline [7], [21]. Specifically, TNF-α has been associated with motor neuron death and muscle mass loss [19].

Additionally, a 2021 study demonstrated that IL-6 plays a dual role: under normal conditions, it increases during physical exertion, stimulating muscle differentiation, but under pathological conditions, elevated IL-6 levels are associated with reduced quadriceps strength and overall physical capacity [21]. However, there is insufficient research confirming the presence of inflammation in the quadriceps muscle in COPD patients [12]. Another factor involved in muscle atrophy is myostatin, a myokine that inhibits muscle differentiation and growth. Myostatin is elevated in patients with both COPD and sarcopenia, inhibiting muscle regeneration and promoting osteoclast activity, thus favoring the onset of osteoporosis [21]. There are satellite cells involved in muscle regeneration which are considered postnatal muscle stem cells. Their number decreases with aging and the onset of muscle atrophy. Miostatin inhibits the proliferation of satellite cells keeping them in a state of quinscence [22], [23].

Patients with both COPD and sarcopenia frequently present with malnutrition (58.7%), which worsens the prognosis [2]. Malnutrition occurs as a result of an imbalance between energy intake and consumption. This energy imbalance translates into a reduction in lean body mass, which can be measured using methods such as BIA (bioelectrical impedance analysis), DXA (dual-energy X-ray absorptiometry), or skinfold measurements [24]. Malnutrition can be diagnosed using the GLIM criteria, which are divided into two categories: phenotypical and etiological. The phenotypic criteria include unintentional weight loss, low body mass index and muscle mass reduction, while the etiological criteria involve decreased food intake or assimilation as well as inflammation or disease burden. To establish the diagnosis, at least one criterion from each category must be met. A 2022 study, involving 124 participants with moderate and severe COPD, showed that 1 in 10 patients suffered from sarcopenia and concomitant malnutrition, while 1 in 7 had sarcopenia alone. The rate of malnutrition is directly proportional to COPD severity [3], [17]. Another marker of malnutrition is low albuminemia, frequently seen in sarcopenic patients [25].

Muscle mass reduction in COPD patients is not always reflected by a reduced BMI. Approximately 26% of patients with normal BMI present with a low fat-free mass index (FFMI). FFMI is used to estimate muscle mass. FFM and fat mass (FM) can be measured using DXA, BIA, CT, ultrasonography [12]. Both FFMI and BMI are considered predictors of mortality in COPD patients, with FFMI reduction being more common in women [26], [27]. There are two phenotypes of COPD: pink puffer and blue bloater. The “pink puffer” phenotype, describing emaciated patients with emphysematous lung lesions, is more commonly associated with low FFMI, while sarcopenia can occur in both underweight and overweight patients [28], [29].

Physical inactivity is a significant risk factor for the development of sarcopenia, as well as a consequence of it. In patients with Chronic Obstructive Pulmonary Disease (COPD), symptoms such as dyspnea, reduced chest wall compliance, and acute exacerbations severely limit physical activity, leading to the deterioration of both muscle mass and function [7]. The reduction in physical activity and energy expenditure may contribute to weight gain, with consequential effects on the respiratory, cardiovascular, and metabolic systems [17].

As individuals age, hormonal imbalances occur, leading to a decrease in anabolic hormones, including growth hormone, testosterone, thyroid hormone, and insulin-like growth factor (IGF-1). This hormonal decline results in decreased protein synthesis (and consequently muscle mass) and increased muscle degradation [7]. The loss of muscle mass occurs more rapidly in women due to their lower testosterone levels.

Hypoxia increases heart rate and induces peripheral vasoconstriction and oxidative stress, which can result in systemic inflammation and muscle atrophy (oxidative stress alters the integrity of muscle proteins) [4], [7]. A decrease in hemoglobin, an indicator of the nutritional status of the body, is observed in patients with sarcopenia and may lead to muscular hypoxia. The demand for hemoglobin is heightened in older adults, and even a slight reduction can correlate with an increased risk of mortality and hospitalization [10].

A significant cause of muscle dysfunction and atrophy in patients with COPD is the chronic use of corticosteroids as part of their treatment regimen. Changes in quadriceps composition have been observed, including necrotic fibers, increased connective tissue, and variations in muscle fiber sizes [12]. During acute exacerbations, corticosteroid use exacerbates muscle loss [7]. Corticosteroids are also risk factors for the development of osteoporosis, with 38% of patients with COPD presenting concomitantly with osteoporosis [30].

Among the biological processes involved in the pathogenesis of muscle dysfunction are structural abnormalities, muscle remodeling, autophagy, and metabolic disturbances [13]. Autophagy plays a crucial role in maintaining muscle mass, contractile function, and the integrity of muscle fibers [31].

The etiology and mechanisms underlying sarcopenia are not yet fully understood. Given its complex nature, which can lead to progressive disability, early identification of sarcopenia is vital for timely intervention and improved prognosis. A screening method involves the use of the SARC-F questionnaire, which aims to identify functional deficits or its combination with thigh circumference measurement (SARC-calF) for increased sensitivity [32]. Key processes involved in the pathophysiology of sarcopenia include systemic inflammation, mitochondrial dysfunction, imbalances between protein synthesis and degradation, satellite cell damage, and oxidative stress [21], [33].

A 2018 study demonstrated that patients who have both COPD and sarcopenia also exhibit more severe geriatric syndromes, including sleep disorder. This combination acts as a risk factor for COPD exacerbations which can lead to hypoxia and systemic inflammation, further accelerating muscle decline [10].

Numerous studies have sought to determine the prevalence of the association between sarcopenia and COPD within the same patient population, yielding variable results [9]. The variability in sarcopenia prevalence is influenced by several factors, including definitions, cut-off values, population characteristics, and diagnostic methods [34].

Sarcopenia has been defined by various international committees (EWGSOP, IWGS, AWGS, FNIH) using differing criteria, all of which share a common denominator: the reduction of muscle mass and, consequently, skeletal muscle function. In 2019, a new consensus, EWGSOP2, was implemented by EWGSOP [7]. According to EWGSOP, the presence of reduced muscle mass alongside impaired skeletal muscle function or the sole presence of decreased physical performance indicates a diagnosis of sarcopenia [17]. The new criteria define physical performance as a criterion for disease severity [35]. Additionally, new cut-off values have been established. For handgrip strength, the cut-off is now set at 27 kg for men and 16 kg for women, compared to previous values of 30 kg and 20 kg, respectively [36].

Population characteristics are another criterion contributing to variability. A meta-analysis published in 2017 revealed that the prevalence of sarcopenia is higher in Asian populations compared to non-Asian populations, attributed to racial characteristics (body size, cultural background, dietary regimes, quality of life). This finding was corroborated by another meta-analysis in 2020, which reached the same conclusion. Both studies underscore that, in addition to racial characteristics, the cut-off values differ for the two populations, being lower for Asians in both sexes [37], [38].

The methods chosen for measuring muscle mass, strength, and physical performance also contribute to the variability in results. Techniques for identifying muscle mass include DXA, CT, MRI, BIA, and calf circumference measurements. MRI is considered the gold standard for assessing muscle mass due to its accuracy, while CT can be a valuable method as it is frequently used to monitor lung disease progression in COPD patients and can measure pectoralis muscle area. DXA measures lean mass and fat mass and it is more precise than BIA [7]. Some studies have indicated that BIA may underestimate fat mass and overestimate muscle mass in individuals with overweight or obesity [39]. Muscle strength can be measured in the upper limb using the handgrip strength test and in the lower limb through quadriceps strength testing or chair stand tests, with a cut-off of 15 seconds [3], [7]. For physical performance assessment, gait speed (cut-off of 0.8 m/s), the timed up and go test, and the 6-minute walking test can be utilized [40], [41].

The most recent meta-analysis has shown that the incidence of sarcopenia in patients with COPD is 27.5%. The significant differences in prevalence values are attributed to the populations selected for the studies. A meta-analysis from 2019 highlights a higher prevalence of sarcopenia in nursing home residents (66.7%) compared to the general population (7.9%), with a pooled prevalence of 21.6% [2]. Another study from 2017 indicated that the highest prevalence of sarcopenia is found among elderly individuals in rehabilitation units, followed by those in nursing homes [38]. A recent meta-analysis from 2020 analyzed three groups of older adults: those in nursing homes, hospitalized patients, and community-dwelling older adults. The highest prevalence of sarcopenia was observed in nursing home residents, followed by hospitalized individuals. The increased prevalence in nursing home and hospitalized populations may be due to reduced physical activity and malnutrition, as these patients often lack a nutrient-rich diet and are subjected to isolation and depression [37]. The prevalence of sarcopenia increases with age, standing at 5.28% in individuals under 50 years and 21.38% in those over 60, with a frequency of exacerbations at 48% [7]. Muscle dysfunction is exacerbated during exacerbations due to reduced physical activity during hospitalization, intensified dyspnea, nutritional imbalances (including reduced appetite and difficulty feeding), and corticosteroid treatment [12].

CONCLUSIONS

The prevalence of sarcopenia in patients with COPD is increasing and is associated with decreased quality of life, physical performance, lung function and increased risk of hospitalization. Acute exacerbations of COPD may also worsen the patient's prognosis. Given the numerous negative effects of the association of sarcopenia in COPD patients, it is important to perform periodic screening to identify early onset of sarcopenia and start pulmonary rehabilitation.

BPOC este o afecțiune complexă, cu manifestări pulmonare și extrapulmonare intens studiate datorită numeroaselor procese patologice implicate. Prevalența sa este în creștere, reprezentând a patra cauză de mortalitate la nivel mondial. Sarcopenia poate apărea la pacienții cu BPOC, având factori de risc comuni. Aceasta se caracterizează prin scăderea masei și funcției musculare, cu impact asupra performanței musculare. Modificările musculare pot fi evaluate prin diverse metode: IRM, DXA, BIA, CT sau biopsie. Prevalența sarcopeniei a fost studiată în numeroase cercetări, cu rezultate variabile. Această revizuire identifică principalii factori de risc care contribuie la aceste variații, cu accent pe caracteristicile populației studiate, criteriile utilizate pentru definirea sarcopeniei și metodele aplicate pentru evaluarea masei musculare, a forței și a performanței fizice.

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Declaration of interest: The authors declare no conflict of interest.

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Received: 16th April 2025