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Review Article

SARS-CoV-2 Sarcopenia: An Update

Ioannis Kourniotis[1,2], George I. Lambrou[2,3,4,5]

[1]*Department of Orthopaedic Surgery, Asklipieio Voulas, Athens, Greece;*
[2]*Postgraduate Program "Metabolic Bone Diseases", National and Kapodistrian University of Athens, Medical School, Athens, Greece;*
[3]*Laboratory for the Research of the Musculoskeletal System "Th. Garofalidis", National and Kapodistrian University of Athens, Medical School, Athens, Greece;*
[4]*Choremeio Research Laboratory, First Department of Pediatrics, School of Medicine, National and Kapodistrian University of Athens, Athens, Greece;*
[5]*University Research Institute of Maternal and Child Health & Precision Medicine, National and Kapodistrian University of Athens, Athens, Greece*

Abstract

Sarcopenia, characterized by the loss of muscle mass and strength, has become a critical concern during the COVID-19 pandemic, influencing patient outcomes such as extended hospital stays, higher mortality, and slower recovery. Factors like chronic inflammation, immune dysfunction, respiratory muscle impairment, and sarcopenic dysphagia contribute to these poor outcomes. COVID-19 exacerbates sarcopenia through mechanisms such as cytokine storms, hormonal imbalances, hypoxia, and mitochondrial dysfunction, all of which accelerate muscle degradation and functional decline. Additionally, the pandemic's impact on physical activity, nutritional status and mental welfare has further worsened sarcopenia, especially in older adults and vulnerable populations. The relationship between COVID-19 and sarcopenia is bidirectional, where muscle loss compromises the immune response, increasing the risk of severe infection, while COVID-19 itself accelerates muscle wasting through inflammation, physical inactivity, malnutrition, and other factors. This interplay heightens vulnerability, prolongs recovery, and worsens overall health outcomes, emphasizing for effective management strategies. Addressing sarcopenia through tailored exercise programs and nutritional interventions is vital for improving long-term recovery and reducing the pandemic's lasting health effects.

Keywords: COVID-19, Cytokine storm, Muscle loss, Respiratory Dysfunction, Sarcopenia

Introduction

Sarcopenia is a syndrome characterized by the progressive loss of muscle mass, strength, and function[1]. Although primarily associated with aging, it can also affect younger individuals[2]. The main factors contributing to age-related muscle loss include impaired protein homeostasis, disruption of proteolytic and autophagic pathways[3,4], mitochondrial dysfunction[5], reduced satellite cells numbers or their impaired ability to proliferate and differentiate[6], increased inflammatory mediators[7] and hormonal changes[8]. Apart from aging, other factors that contribute to sarcopenia include physical inactivity, malnutrition, chronic diseases, endocrine disorders, as well as the effects of medications, prolonged hospitalization, and acute conditions such as infections. Sarcopenia has been linked to significant functional impairment, increased fall risk, disability, reduced quality of life, higher hospitalization rates and increased mortality due to various conditions, including Severe Acute Respiratory Coronavirus 2 (SARS-CoV-2) infection[9,10].

The rapid spread of SARS-CoV-2, the virus responsible for COVID-19, led the World Health Organization to announce a pandemic in March 2020. The COVID-19

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Corresponding author: *George I. Lambrou, National and Kapodistrian University of Athens, 1st Department of Pediatrics, Choremeio Research Laboratory, Thivon & Levadias, 11527, Goudi, Athens, Greece*

E-mail: *glamprou@med.uoa.gr*

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pandemic represents a unique global health crisis, with more than 777 million confirmed cases and over 7 million deaths by 26 January 2025[11]. COVID-19 primarily affects the respiratory system and triggers widespread inflammation, often leading to long-term effects on overall health[12]. Most patients, around 80–90%, experience mild to moderate symptoms, while the remaining 10–20% develop severe complications requiring hospitalization and intensive care. Mortality is especially high in older adults, reaching 20% in those over the age of 80[13]. Several factors increase the risk of poor COVID-19 outcomes, including advanced age, obesity, and comorbidities such as sarcopenia, COPD, diabetes, and cardiovascular disease[14]. In an effort to control the spread of SARS-CoV-2 and prevent the collapse of healthcare systems, health authorities globally enforced various restrictions such as travel bans, quarantines, isolation and social distancing. The SARS-CoV-2 pandemic has profoundly disrupted healthcare systems and daily life, directly impacting public health Beyond the immediate effects of COVID-19 infection, the pandemic and related measures resulted in extended periods of home confinement. This significantly reduced physical activity and increased sedentary behavior, factors known to contribute to muscle loss and physical decline. Additionally, changes in eating habits and heightened psychological stress further elevated the risk of developing sarcopenia[15].

This narrative review explores the association between sarcopenia and COVID-19, focusing on the bidirectional relationship and the pathophysiological mechanisms that link the two conditions. It examines how COVID-19 can contribute to the onset or progression of sarcopenia, as well as how pre-existing sarcopenia can lead to poor COVID-19 outcomes. Additionally, the review discussed the impact of restrictive measures, such as quarantine and lockdowns, on muscle mass, which can further exacerbate sarcopenia. Strategies for managing sarcopenia during and after the pandemic are also explored, emphasizing the importance of nutrition and physical activity muscle loss and improve overall health.

Mechanisms Linking Sarcopenia and COVID-19

Sarcopenia is a significant comorbidity that complicates COVID-19 infection. It has been shown to worsen hospitalization outcomes and the recovery process, increasing the risk of complications. Therefore, it's essential to identify and manage sarcopenia early, particularly in patients dealing with COVID-19, as this can significantly improve their overall prognosis. Even though sarcopenia and COVID-19 follow different pathways, their mechanisms can significantly influence each other. In both conditions, several pathways share similar patterns and common stimuli. Sarcopenia and COVID-19 interact creating a vicious cycle where each condition exacerbates the other. Sarcopenia leads to muscle loss, negatively affecting the body's ability to cope with infections and recover efficiently. At the same time, COVID-19 can worsen sarcopenia, further accelerating the catabolic state. This two-way relationship not only increases the risk of severe outcomes but also impedes the recovery process. Therefore, addressing sarcopenia early, both during and after a COVID-19 infection, is critical for improving patient prognosis and recovery.

Systemic Inflammation

Chronic low-grade inflammation associated with aging (inflammaging) is characterized by increased levels of pro-inflammatory cytokines such as IL-6, TNF-α, and CRP, which play a significant role in the development of sarcopenia[16]. These inflammatory mediators disrupt muscle protein homeostasis by impairing both synthesis and degradation processes, leading to progressive muscle mass loss and reduced strength[17].

A key mechanism linking COVID-19 to sarcopenia is the excessive release of pro-inflammatory cytokines, known as cytokine storm. In severe COVID-19 cases, a hyperactive immune response drives an uncontrolled release of pro-inflammatory cytokines, leading to systemic inflammation and tissue damage[18]. While these cytokines play a vital role in initiating the acute phase response to infection, their overproduction causes systemic inflammation and muscle wasting[19]. This severe immune reaction can rapidly progress to acute respiratory distress syndrome, multi-organ failure, and death. Studies have shown that patients with severe COVID-19 exhibit significantly higher levels of pro-inflammatory cytokines, linking the severity of the disease with the presence of the cytokine storm[20,21].

This hyperinflammatory state has a significant impact on skeletal muscle. The excessive secretion of cytokines accelerates muscle fiber breakdown, stimulates fibroblasts, and leads to fibrosis while inhibiting the function of satellite cells, which are essential for muscle repair and regeneration[22,23]. When cytokine levels remain high for prolonged periods, as seen in chronic inflammatory conditions, persistent muscle loss occurs, a hallmark of sarcopenia. Long-term inflammation also stimulates catabolic pathways, such as the ubiquitin-proteasome system, which degrades muscle proteins and exacerbates muscle atrophy[19,24]. Consequently, muscle loss continues even after the acute phase of infection, complicating recovery and increasing the risk of long-term functional decline.

Another serious consequence of the cytokine storm is the induction of oxidative stress. The excessive production of free radicals during this response damages muscle cells and tissues, further impairing muscle function. Oxidative stress also impairs mitochondrial function, which is essential for energy production in muscle cells, leading to muscle fatigue and functional decline. The combination of systemic inflammation, cytokine-driven muscle atrophy, and oxidative damage creates a vicious cycle that

accelerates muscle loss and significantly delays recovery, particularly in critically ill COVID-19 patients[25].

Given the chronic inflammatory background of sarcopenic patients, they face an even higher risk of severe COVID-19 complications, as inflammaging may amplify the cytokine storm response[26]. This underscores the importance of early interventions to mitigate muscle loss and inflammation, particularly in older adults or those recovering from severe illness.

Immune Dysfunction

Sarcopenia is associated with immune dysfunction, including impaired immune cell proliferation, an altered neutrophil-to-lymphocyte ratio, and disrupted natural killer (NK) cell homeostasis, contributing to aging immune system[27]. This dysfunction is clinically evident in the higher incidence of community and hospital-acquired pneumonia, which shares respiratory complications with COVID-19.

Skeletal muscle acts as an endocrine organ by releasing myokines like IL-15, IL-17, and IL-6, in response to muscle contraction. These myokines contribute to a systemic anti-inflammatory environment and exert specific endocrine effects, that regulate immune responses[27]. In sarcopenia, myokine expression is reduced, particularly IL-15, which is crucial for the activation, proliferation, and survival of immune cells such as NK and CD8+ T-cells. Impaired IL-15 signaling leads to inadequate immune responses against SARS-CoV-2[28]. Changes in myokine levels mainly due to aging, inflammation, and sarcopenia weaken immune function, a phenomenon known as immune aging, which primarily affects the older adults[27]. Additionally, skeletal muscles serve as a reservoir of amino acids like glutamine, which support immune activity and metabolism during infections. Sarcopenic patients have reduced protein mobilization, further compromising their immune response and increasing metabolic stress during infection, explaining their poor response to SARS-CoV-2[29].

Reduced physical activity is associated with weak immune function. For example, sedentary older adults have lower levels of naïve T-cells and higher levels of memory T-cells, indicating a diminished immune response[30]. Regular exercise can enhance immune function, with studies showing that physically active seniors have stronger immune responses to vaccinations[31]. The relationship between muscle mass and myokines is bidirectional—while changes in muscle mass can influence myokines secretion, alterations in myokine levels can also contribute to muscle loss and the onset of sarcopenia. The decrease in physical activity during the COVID-19 pandemic has worsened muscle loss and weakened immune function. This could explain why older adults, who are more vulnerable to sarcopenia and its effects on immunity, faced higher risks of severe COVID-19 outcomes[32].

Viral Myopathy

While SARS-CoV-2 mainly targets respiratory cells, it also impacts skeletal muscles through viral myopathy, where virus directly invades and damages muscle tissue. Although rare, viral particles have been detected in muscle biopsies from deceased COVID-19 patients, indicating that the virus can invade muscle fibers and cause localized damage, supporting the occurrence of virus-induced myopathy in severe cases[33,34]. Skeletal muscle cells express the angiotensin-converting enzyme 2 (ACE2) receptor, which allows viral entry, leading to muscle damage, inflammation, and, in some cases, muscle fiber necrosis.

The mechanism of viral myopathy involves the binding of the SARS-CoV-2 spike protein to ACE2 receptors on skeletal muscle cells. Once the virus gain entry to the muscle cells, it disrupts normal cellular functions, triggering inflammation, necrosis, and apoptosis. Additionally, systemic inflammation further intensifies muscle cell damage, making muscle fibers more susceptible to injury[33]. Muscle biopsies from critically ill COVID-19 patients show signs of muscle fiber degeneration, necrosis, and inflammation, provide further evidence that the SARS-CoV-2 directly damages the skeletal muscles. This muscle injury, combined with the catabolic effects of cytokine release, likely contributes to the rapid onset of sarcopenia seen in hospitalized COVID-19 patients[35,36].

Hypoxia

Given that COVID-19 primarily targets the lungs, respiratory complications are common. As a result, gas exchange is impaired, reducing oxygen delivery to tissues including skeletal muscle. The virus induces hypoxia, a condition marked by insufficient oxygen supply to tissues, and in severe cases respiratory failure. Muscle cells rely on a sufficient oxygen supply to produce ATP, the primary energy source for muscle contractions[37]. Under hypoxic conditions, muscle cells shift to anaerobic metabolism, which is less efficient and leads to lactate accumulation and other metabolic byproducts. This metabolic shift further impairs muscle function[38]. Moreover, hypoxic muscles are deprived of the oxygen necessary for recovery and repair. Chronic hypoxia, particularly in critically ill patients who require mechanical ventilation or experience prolonged respiratory distress, severely restricts oxygen supply to muscles, hindering their recovery and leading to muscle atrophy can lead to muscle atrophy[15,39]. Chronic oxygen deprivation negatively impacts mitochondrial function, which is essential for energy production and muscle fiber regeneration[37]. This impairment can result in reduced muscle function and, eventually, muscle wasting —a hallmark of sarcopenia.

Studies, mainly in animal models, have shown that hypoxia can stimulate muscle protein synthesis. However, this potential benefit is often counteracted by an increased proteolysis, resulting in net muscle loss[40]. Hypoxia is associated with high myostatin levels, a protein that promotes muscle breakdown and serves

as a negative regulator of muscle growth. Additionally, hypoxia alters IGF-1 signaling pathways, shifting from the IGF-1/Akt pathway, which promotes muscle growth, to the IGF-1/ERK pathway, which leads to myogenesis without enhancing muscle fiber differentiation. This shift promotes myoblast proliferation rather than differentiation, further impairs muscles' ability to regenerate and recover despite ongoing myogenesis[41]. The combined effects of chronic hypoxia, reduced physical activity, and limited muscle regeneration create an environment that accelerates sarcopenia in COVID-19 patients. In critically ill patients, muscle loss is further aggravated by prolonged respiratory distress and mechanical ventilation, which significantly contribute to the decline in muscle mass and function.

Mitochondrial Dysfunction

Upon infection with SARS-CoV-2, the release of inflammatory cytokines is activated, leading to oxidative stress. In this condition, the production of reactive oxygen species (ROS) exceeds the body's ability to counteract them, causing damage to muscle cellular structures, impairing muscle cell function. In skeletal muscle, oxidative stress disrupts mitochondrial function, which in turn, increases ROS levels, creating a vicious cycle that further impair muscle function over time and increase atrophy[15,42].

Mitochondrial dysfunction is a critical factor in the development of sarcopenia since mitochondria are essential for producing ATP, the energy needed by muscle cells. When mitochondria are impaired, the result is reduced energy, muscle fatigue, and atrophy[43]. In COVID-19 patients, a combination of inflammation, direct viral damage to muscle tissue, and hypoxia contribute to mitochondrial dysfunction. This dysfunction causes structural and functional changes in mitochondria, including ROS accumulation that damages muscle cells[33]. Mitochondrial DNA is also affected, disrupting the electron transport chain and further reducing ATP production, weakening even more the muscles' ability to contract and regenerate, which accelerates muscle loss[44]. Additionally, during the infection, high ferritin levels disrupt mitochondrial iron homeostasis, forcing a metabolic shift from aerobic to anaerobic pathways, which increases ROS levels and promotes muscle cell apoptosis[33].

The mTORC1 signaling pathway, which is essential for mitochondrial biogenesis and muscle maintenance, is inhibited during COVID-19 infection, further impairing mitochondrial regeneration and contributing to muscle loss[45]. Nutrition also plays a role: a lack of amino acids, especially leucine, disrupts mTORC1 activation and muscle protein synthesis, while pro-inflammatory diet can worsen oxidative stress. Along with reduced physical activity, these factors contribute to sarcopenic obesity, further compounding mitochondrial dysfunction[46].

Hormonal Changes

COVID-19 is associated with significant hormonal changes that contribute to muscle loss and sarcopenia. Studies have shown that COVID-19 increases insulin resistance and impacts basic anabolic hormones involved in muscle maintenance. Specifically, the SARS-COV-2 raises estrogen levels while reducing testosterone, IGF-1, and growth hormone (GH)[47]. Additionally, myostatin, a key hormone that regulates muscle metabolism, is elevated to control muscle cell growth, preserving energy and amino acids for other essential metabolic processes[48]. These hormonal imbalances, combined with inflammation from COVID-19 infection, accelerate muscle loss. Therefore, muscle loss in COVID-19 patients is not solely due to prolonged immobility but also arises from the interaction between increased inflammation and reduced anabolic signaling.

Respiratory Dysregulation

Sarcopenia not only affects overall muscle strength but also plays a crucial role in respiratory muscle dysfunction, which can worsen the outcome of COVID-19 infection. A study conducted on 711 patients hospitalized with moderate to severe COVID-19 explored the relationship between sarcopenia and pulmonary function[49]. It found that sarcopenia was associated with reduced lung function, and respiratory muscle deterioration was observed in 17.4% of patients with sarcopenia. Similar findings have been reported in other populations, where respiratory muscle strength correlated with lower muscle strength, muscle mass, and sarcopenia, negatively impacting pulmonary function[50,51].

The diaphragm, the primary respiratory muscle, can be severely affected by sarcopenia, leading to impaired respiratory function, especially in COVID-19 patients. This muscle dysfunction may contribute to a higher vulnerability to pneumonia. Sarcopenia can decrease the strength and size of diaphragm muscle fibers, resulting in atrophy and difficulty with essential actions like coughing and sneezing, crucial for clearing the airways[52]. Studies show that a thinner and weaker diaphragm is associated with an increased risk of mechanical ventilation or death in COVID-19 patients, highlighting the importance of monitoring diaphragm function with ultrasound in critically ill patients[53].

Malnutrition, Dysphagia, and Sarcopenia

Malnutrition is a significant concern for COVID-19 patients, as many experience appetite loss, taste alterations, and nausea. These symptoms can reduce food intake and, in severe cases, lead to malnutrition, which increases the risk for poor outcomes, particularly among hospitalized COVID-19 patients[54]. Research suggests that dietary patterns are also linked to a higher risk of hospitalization due to COVID-19[55]. Hospitalized COVID-19 patients face high energy demands to sustain metabolic functions and often enter a catabolic state that worsens disease progression and accelerates sarcopenia. This process is further aggravated by

inadequate nutrient intake[56]. Malnutrition depletes essential nutrients with anti-inflammatory and antioxidant properties, promoting muscle breakdown and inflammation[57].

Sarcopenic dysphagia refers to swallowing difficulties caused by sarcopenia that significantly impacts health and quality of life, particularly in older adults[58]. It is associated with high mortality rates among hospitalized older adult patients[59]. Many older COVID-19 patients already have swallowing impairments before infection, and the disease can further exacerbate dysphagia, complicating patient management[60]. Sarcopenic dysphagia is also a recognized risk factor for aspiration pneumonia, a complication that can worsen outcomes in bedridden COVID-19 patients[61]. Approximately 30% of critically ill COVID-19 patients develop dysphagia after extubation, correlating with disease severity and prolonged mechanical ventilation[62]. While the relationship between sarcopenic dysphagia and poor COVID-19 prognosis remains under investigation, it is likely that pre-existing dysphagia heightens the risk of adverse outcomes, emphasizing the need for further research.

COVID-19 Restriction Measures and Sarcopenia

Dietary Changes and Nutritional Deficiencies

The COVID-19 pandemic significantly exacerbated muscle wasting, largely due to disruptions in food supply chains and limited access to fresh, protein-rich foods. During lockdowns, obtaining essential items became more challenging, particularly for older adults and individuals with disabilities[63]. As a result, many individuals turned to processed, calorie-dense foods that lacked nutritional value. This dietary shift led to inadequate protein intake and deficiencies in key micronutrients crucial for muscle health, such as vitamin D, magnesium, and omega-3 fatty acids[64,65].

A study conducted during the pandemic revealed that up to 30% of older adults had a significant reduction in dietary protein intake due to changes in eating habits, putting those already affected by sarcopenia at an even greater risk of muscle loss due to both protein deficiency and reduced physical activity. People with sarcopenia require higher levels of protein to counteract muscle loss, making them particularly vulnerable to the negative effects of this dual challenge[66,67].

Lockdowns and restrictions also led to changes in meal patterns and appetite regulation, with psychological stress and social isolation affecting eating behaviors. Some individuals experienced appetite and weight loss, while others turned to emotional overeating. Disruptions in food access—due to shortages, fewer grocery visits, or fear of infection—further impacted dietary choices and nutrition quality. These shifts, along with changes in appetite, resulted in weight gain or loss. A study found that 30% of participants gained weight, while over 18% lost it. Overweight people were more likely to gain weight, while underweight tended to lose it[68], suggesting that lockdowns intensified preexisting dietary habits.

Weight gain during the pandemic was resulted from an increase in ultra-processed foods, which are often chosen for their long shelf life and as a response to stress-related eating. A study found that 46.1% of participants ate more, particularly comfort foods linked to higher anxiety levels[69]. This increase in meal frequency and consumption of calorie-dense foods negatively impacted muscle mass. Diets high in ultra-processed foods are typically low in nutrients, especially protein, reducing the body's ability to promote muscle protein synthesis. Excessive calorie intake leads to weight gain and fat accumulation, which, in turn, contributes to sarcopenic obesity. This condition, in turn, further limits mobility due to reduced muscle strength and excess weight, while also promoting chronic inflammation, insulin resistance, and metabolic dysfunction, all of which worsen sarcopenia[70,71]. Severe obesity increases the risk of COVID-19 complications, including a higher likelihood of ICU admission[72].

On the other hand, weight loss also has significant negative effects. During the COVID-19 pandemic, food insecurity and financial difficulties led to unintentional weight loss and nutrient deficiencies, especially in older adults. The limited access to groceries, combined with reluctance to shop in person due to safety concerns, resulted in inadequate food intake and malnutrition, accelerating muscle loss by impairing muscle protein synthesis. Since about 25% of the weight lost in young, healthy individuals consists of lean mass, older adults who experienced unintentional weight loss likely faced rapid and severe muscle decline, further compromising their strength, mobility, and overall health[73].

Healthcare Disruptions

The diagnosis and management of sarcopenia were significantly affected by the restrictions on non-emergency medical services and limited access to routine healthcare during the pandemic. Early interventions critical for sarcopenia—such as physiotherapy and nutritional counseling—became largely inaccessible as most outpatient clinics and rehabilitation centers suspended in-person visits. These barriers to healthcare also disproportionately affected individuals with other chronic conditions who were already at higher risk of muscle wasting. The lack of timely medical attention further increased their risk of developing or progressing sarcopenia[39].

A retrospective analysis of healthcare delivery during the COVID-19 pandemic found a sharp decline in musculoskeletal assessments and physiotherapy sessions. These disruptions in care contributed to the progression of sarcopenia, as it often remains asymptomatic in its early stages and is typically diagnosed only when functional decline becomes apparent. Furthermore, early interventions, such as resistance training and dietary modifications, were largely unavailable, increasing the long-term risk of disability[25,37,74].

Although telehealth services were widely adopted as an alternative way of healthcare access, they were partial insufficient to fully replace in-person evaluations of muscle strength, gait, and overall functional capacity. This approach was especially challenging for older adults, many of whom either lacked access to virtual consultations or encountered difficulties with the technology required for remote assessments[75].

Lockdowns and Physical Activity

A significant impact of COVID-19 lockdowns was a drastic reduction in physical activity levels across all age groups. Opportunities for physical activity were limited due to the closure of gyms, leisure centers, and outdoor recreational areas, which resulted in a marked increase in sedentary behavior. As expected, older adults experienced the most substantial decline in mobility. A study assessing physical activity patterns under COVID-19 restrictions found that both step counts and overall movement decreased by more than 40%, with those over the age of 60 years showing the greatest decline[76,77]. Irregular physical activity led to a rapid deterioration in muscle mass and strength.

Prolonged immobility due to hospitalization, isolation, or fear of exposure to SARS-CoV-2 further worsened muscle atrophy. Even after recovery from COVID-19, many patients remained bedridden for extended period of time, exacerbating muscle loss and delaying rehabilitation. In uninfected individuals, restrictions resulted in reduced muscle mechanical loading, leading to decreased muscle protein synthesis and an imbalance between anabolic and catabolic processes. These effects were particularly severe in those with pre-existing mobility issues, as even short periods of inactivity caused a disproportionate muscle loss[78].

Psychological Impact and Sleep Disorders

The psychological impact of COVID-19 pandemic has been particularly significant for older adults and those with chronic conditions like sarcopenia. Chronic stress increases cortisol levels, which in turn accelerate muscle breakdown and impair regeneration, ultimately contributing to muscle weakness and sarcopenia[79]. Various studies have highlighted the negative psychological consequences of quarantine and social isolation, with common symptoms including depression, anxiety, insomnia, and emotional exhaustion[80]. An Italian study found that 57.1% of participants reported poor sleep quality, 32.1% experienced heightened anxiety, and 41.8% worried about the pandemic[81]. Another study recorded post-traumatic stress symptoms in up to 29.5% of participants[82]. Stress-related overeating was also a common response to negative emotions during lockdowns.

The pandemic caused a rise in depression and anxiety, which often correlates with reduced motivation for physical activity and poor eating habits. Additionally, the stress during the pandemic has led to sleep disturbances, which impede muscle recovery. Disrupted sleep patterns, with reduced quality and duration, can hinder the body's ability to regulate anabolic hormones, such as GH and testosterone, both vital for muscle maintenance[79]. Shortened sleep and higher cortisol levels increased muscle degradation[83], while poor sleep also impacts appetite regulation, leading to weight gain and further muscle loss[81]. Even a small reduction in sleep (e.g. from 8 to 6 hours) increases pro-inflammatory cytokines linked to muscle breakdown[83]. Just two nights of only 4 hours of sleep elevate cortisol levels[84], while eight nights with 5-hour sleep reduce testosterone by 10-15%[85]. High cortisol promotes muscle protein degradation, a process worsened by physical inactivity, as seen during lockdowns. Both stress and sleep deprivation contribute to muscle loss by affecting metabolic pathways essential for muscle maintenance. The combination of stress, poor sleep, and reduced physical activity creates a vicious cycle that exacerbating muscle loss, particularly in older adults[86].

Sleep disturbances also affect appetite regulation. In a study, up to 57.1% of participants reported poor sleep[81], while 46.1% increased intake of high-calorie foods[69] during lockdowns. This is partly due to hormonal imbalances: sleep deprivation decreases leptin (which suppresses appetite) and increases ghrelin (which stimulates hunger), leading to stronger cravings for carbohydrate-rich foods[87]. Combined with the wide availability of low-nutrient foods and reduced physical activity, this creates an environment that promotes weight gain, which in turn accelerates muscle loss through metabolic and hormonal disruptions, chronic inflammation, and insulin resistance[88].

Pandemic-related weight loss, when coupled with insufficient sleep, may have worsened muscle wasting, particularly in older adults. Emotional distress and low mood reduced motivation for physical activity, which is critical for muscle maintenance[86]. Sleep deprivation exacerbates stress, creating a vicious cycle of worsening sleep and rising anxiety levels[89].

Vitamin D and Muscle Health

Vitamin D plays a crucial role in skeletal and muscle health by influencing muscle contraction, mitochondrial function, and protein synthesis. Deficiency in vitamin D is associated with reduced muscle mass and strength, a concern that became more prominent during the COVID-19 pandemic due to limited sun exposure from lockdowns and restricted outdoor activities[90]. Vitamin D is essential for calcium homeostasis in muscle cells, ensuring proper contraction and relaxation. Its deficiency may lead to prolonged muscle relaxation and impaired mitochondrial function, reducing muscle efficiency and endurance. Restoring optimal vitamin D levels has been linked to improved mitochondrial oxidative capacity and overall muscle function[91].

Moreover, vitamin D enhances insulin signaling and promotes protein synthesis in muscle cells. *In vitro* studies reveal that vitamin D boosts insulin receptor phosphorylation

and increases vitamin D receptor (VDR) expression, amplifying the effects of insulin and leucine on muscle protein synthesis[92]. Animal studies further support this, showing that VDR deletion in skeletal muscle results in increased fat mass and decreased lean mass, leading to reduced endurance, slower movement speed, and lower grip strength—hallmarks of both acute and chronic sarcopenia[93]. Additionally, vitamin D regulates myostatin. By reducing myostatin expression, vitamin D supports muscle hypertrophy, which may explain its role in maintaining muscle mass and strength in aging people. Vitamin D supplementation, particularly at high doses and for extended periods, has been shown to improve muscle strength, especially in those with pre-existing deficiencies[94]. The benefits are further enhanced when vitamin D intake is combined with adequate protein consumption[95].

Beyond muscle health, vitamin D deficiency is considered a risk factor for COVID-19 susceptibility and severity. Low vitamin D levels have been linked to increased pro-inflammatory cytokines, which contribute to immune dysfunction and worsen COVID-19 outcomes. Older adults, who are more prone to vitamin D deficiency, faced greater risks of both sarcopenia and severe COVID-19 complications during lockdowns[96].

Managing Sarcopenia During and After the COVID-19 Pandemic

The COVID-19 pandemic had a profound impact on physical health. COVID-19 not only has the potential to cause sarcopenia, but sarcopenia itself can also lead to worse outcomes in COVID-19 patients. Several strategies can be employed to combat sarcopenia both during and after a pandemic. These include targeted exercise, proper nutrition, and innovative interventions such as telehealth services, which have gained importance during the pandemic.

Exercise Interventions

Exercise, particularly resistance training, is one of the most effective strategies for managing sarcopenia. For individuals at risk, and especially for COVID-19 survivors, resistance exercises are crucial in rebuilding lost muscle strength and mass. An exercise program should follow the principles of overload, specificity, and progression to maximize benefits. Even a modest routine of one or two low-intensity resistance training sessions per week (about 50% of 1RM) can significantly improve muscle strength and function. For optimal gains, higher intensities (70-85% of 1RM) and more frequent sessions (two to three times a week) may be necessary[97,98]. While resistance training is the cornerstone for muscle mass, aerobic exercise also plays a vital supporting role. Even though less effective for increasing muscle mass, aerobic exercise helps reduce both total and intramuscular fat, which improves muscle function[99]. The combination of resistance training and aerobic exercise is particularly beneficial for improving walking speed, lower limb strength, and body composition, especially in older adults[100]. Moreover, such a combined exercise program helps reduce insulin resistance, a key factor in anabolic resistance and sarcopenia, thus supporting long-term muscle health. For COVID-19 survivors, maintaining physical activity, even during quarantine or isolation, is crucial. Simple, everyday movements such as increasing daily steps, or engaging in household tasks like cleaning or gardening, offer practical, cost-effective ways to maintain physical health.

Nutritional Support

Nutrition plays an essential role in managing sarcopenia, particularly for COVID-19 survivors who may experience muscle wasting due to illness or immobility. Protein supplementation along with resistance training, has proven to be an effective strategy for improving muscle mass and strength. A balanced diet, which includes lean protein sources, whole grains, fruits, vegetables, and healthy fats, can be beneficial in preventing sarcopenia and managing the consequences of COVID-19[101]. Proper nutrition provides essential antioxidants, vitamins, and minerals that strengthen the immune system and overall health, while also offering anti-inflammatory properties that help reduce systemic inflammation. Studies have shown that a high-protein diet, especially when combined with exercise, can significantly enhance muscle health in sarcopenic patients[102]. During COVID-19 infection, ensuring adequate protein intake is essential to reduce muscle protein breakdown and prevent a catabolic state that could worsen sarcopenia. For COVID-19 patients, particularly those requiring intensive care, higher protein intake (1.2 to 2.5 g/kg/day) is recommended to prevent muscle loss and promote recovery[103]. However, further research is needed to determine the optimal source and timing of protein intake, especially in relation to exercise. In addition to protein, vitamin D supplementation is often considered for managing sarcopenia, as it supports muscle function and bone health. While clinical studies on its effectiveness in treating sarcopenia yield mixed results[104], vitamin D remains vital for individuals with sarcopenia and may play a role in enhancing the immune response in COVID-19 patients, potentially reducing inflammation and the severity of the infection[105].

Telemedicine-Based Interventions

Telehealth services have proved to be a valuable tool for encouraging exercise adherence, particularly during the pandemic when in-person sessions were limited. Virtual exercise programs and real-time interactions with trainers, have been successfully integrated into rehabilitation plans for sarcopenia. A 12-week telehealth program resulted in significant improvements in muscle mass and functional capacity for sarcopenic population[106]. Furthermore, these virtual programs help combat social isolation, which became a concern during the pandemic, particularly among older adults. Although access to telemedicine can be challenging due to technological barriers, it remains a promising

alternative for those unable to participate in traditional exercise programs. For COVID-19 survivors, virtual group classes not only provide physical benefits but also contribute to emotional well-being by fostering social interaction[107].

Conclusion

COVID-19 and sarcopenia are closely linked, with the infection impacting muscle health both directly and indirectly. The virus triggers mechanisms like inflammation, oxidative stress, hypoxia, mitochondrial dysfunction, and hormonal imbalances, all of which contribute to muscle degradation. The long-term effects of COVID-19 on the musculoskeletal system are especially significant for older adults and individuals with pre-existing conditions, increasing their susceptibility to sarcopenia. Hospitalized patients, particularly those in ICUs or with prolonged immobility, face an elevated risk of severe muscle atrophy, which can adversely affect their quality of life. Sarcopenia, in turn, complicates the recovery process for COVID-19 patients, as weakened muscles can impair mobility, respiratory function, and overall physical resilience. The loss of muscle mass and strength can lead to increased frailty, longer hospital stays, and a greater need for intensive care, all of which contribute to poorer health outcomes. To address these challenges, managing strategies, including timely nutritional support and physical exercise, are crucial. A better understanding of the mechanisms behind the interaction between SARS-CoV-2 and muscle tissue will enable more effective treatments and preventive measures for sarcopenia in both acute and chronic stages, helping mitigate the functional consequences for vulnerable populations.

Authors' contributions

IK: Drafted the manuscript, reviewed literature. GIL: Proof-edited the manuscript and gave final permission for publication. All authors read and approved the final version of the manuscript.

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