COVID-19 precipitated sarcopenia in a patient with COPD and initially preserved functional status

Leila Laouar MD, Nadia Dammene Debbih MD, Nadia Hammadi MD, Sonia Nouioua MD

Abstract

Case: *We report a case of a 67-year-old man with COPD, Global Initiative for Chronic Obstructive Lung Disease (GOLD) grade B, who developed significant sarcopenia following a mild COVID-19 infection. Prior to infection, the patient had preserved functional capacity, with a 6-minute walk distance (6MWD) of 420 meters. During the acute phase, he experienced mild respiratory symptoms managed entirely on an outpatient basis. Three weeks post-infection, he had marked proximal muscle weakness, reduced handgrip strength (21 kg), slow gait speed (0.9 m/s), and decreased 6MWD (290 m). Laboratory evaluation revealed elevated creatine phosphokinase (CPK 420 U/L) and lactate dehydrogenase (LDH 280 U/L), persistent mild lymphopenia, and low-grade systemic inflammation. Serum 25-hydroxyvitamin D, albumine and calcium levels were within normal ranges. Immunological work-up for inflammatory myopathy, including antinuclear antibodies (ANA) and anti–Jo-1 (anti–histidyl-tRNA synthetase) antibodies, was negative. A structured rehabilitation program, including respiratory therapy, postural exercises, low-intensity resistance training, and optimized protein intake (1.2–1.5 g/kg/day), was initiated.*

Outcome: *Over 12 months, the patient demonstrated progressive recovery of muscle strength, functional capacity, and normalization of most laboratory parameters, with residual mild proximal weakness.*

Conclusion: *This case underscores that COVID-19 can precipitate or unmask sarcopenia even after mild respiratory illness in COPD patients with previously preserved functional status. Systematic muscle assessment and early rehabilitation, including nutritional optimization, are essential to preserve functional independence in this vulnerable population.*

Keywords: COVID-19, COPD, Sarcopenia, Muscle weakness, Handgrip strength, Rehabilitation

Introduction

Chronic Obstructive Pulmonary Disease (COPD) is a progressive respiratory condition characterized by persistent airflow limitation and systemic manifestations that extend beyond pulmonary involvement.[1] Among these extrapulmonary complications, sarcopenia, defined as the loss of skeletal muscle mass and function, represents a significant comorbidity affecting up to 15–40% of COPD patients, depending on disease severity and diagnostic criteria.[2,3]

Corresponding author: **Leila Laouar**
Contact Information: Laouar_Leila@yahoo.fr
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The pathophysiology of sarcopenia in COPD is multifactorial, involving chronic systemic inflammation, hypoxia,[4] oxidative stress,[5] hormonal imbalances, nutritional deficiencies,[6] physical inactivity,[6] and Sarcopenia in COPD patients is associated with worse clinical outcomes, including reduced exercise capacity, increased exacerbation frequency, higher hospitalization rates, and increased mortality.[2,7]

The COVID-19 pandemic has added a new dimension to the management of COPD patients. Beyond the acute respiratory complications, COVID-19 has

been associated with prolonged sequelae, including persistent fatigue,[8] dyspnea,[8] and muscle weakness,[8] collectively termed "long COVID" or post-acute sequelae of SARS-CoV-2 infection (PASC).[9] The mechanisms underlying these persistent symptoms remain incompletely understood but likely involve ongoing inflammation,[10] immune dysregulation,[11] endothelial dysfunction,[11] and direct viral effects on various organ systems, including skeletal muscle.[12]

This case report describes a patient with COPD GOLD grade B and preserved functional capacity who developed severe sarcopenia following a mild COVID-19 infection, highlighting the need for heightened awareness and proactive muscle health assessment in this vulnerable population.

Case

Patient information and baseline characteristics

A 67-year-old man, former smoker, presented with severe and persistent muscle weakness three weeks after a confirmed SARS-CoV-2 infection. His smoking history comprised one pack per day for 18 years followed by half a pack per day for five years, with complete cessation seven years earlier (20.5 pack-years). Past medical history included a remote cholecystectomy and an episode of pyelonephritis related to renal lithiasis treated by extracorporeal shock-wave lithotripsy ten years earlier. He had been diagnosed six years previously with chronic obstructive pulmonary disease (COPD), classified as GOLD grade B.

Before COVID-19, the patient was clinically stable on dual long-acting inhaled bronchodilator therapy (long-acting β_2-agonist plus long-acting muscarinic antagonist), with short-acting bronchodilators as needed. Preventive care included annual influenza vaccination and pneumococcal vaccination one year prior to infection.

Baseline pulmonary function tests demonstrated a non-reversible obstructive ventilatory defect, with a forced vital capacity of 3.22 L (87% predicted), forced expiratory volume in one second (FEV_1) of 1.98 L (68% predicted), and an FEV_1/FVC ratio of 61%, with no significant post-bronchodilator reversibility. Functional capacity was preserved, with a six-minute walk distance (6MWD) of 420 m (81% predicted) and no exertional oxygen desaturation. Cardiac evaluation showed normal sinus rhythm on electrocardiography and preserved left ventricular systolic function (ejection fraction 60%) on echocardiography, with only grade I mitral regurgitation. Sleep polygraphy revealed a mild apnea–hypopnea index of 7 events per hour without clinically relevant sleep-disordered breathing. Physical examination before COVID-19 was unremarkable, with no signs of respiratory failure or cor pulmonale. Anthropometric measurements showed a weight of 68.5 kg, height of 170 cm, and body mass index (BMI) of 23,5 kg/m^2.

Acute COVID-19 illness

The patient developed his first episode of COVID-19, presenting with fever, cough, and insomnia, associated with a mild exacerbation of COPD. On initial evaluation, he was mildly tachypneic (24 breaths/min), with oxygen saturation of 95% on room air, heart rate of 100 beats/min, blood pressure of 120/65 mmHg, and temperature of 38°C. Body weight had decreased to 67 kg (BMI 23,2 kg/m^2).

Chest auscultation revealed bilateral wheezing and scattered rhonchi without signs of respiratory distress. Cardiovascular and abdominal examinations were otherwise unremarkable.

Chest computed tomography demonstrated peripheral ground-glass opacities with an apicobasal distribution involving less than 10% of the lung parenchyma, associated with apical centrilobular emphysematous bullae, and no evidence of pleural effusion or mediastinal lymphadenopathy. SARS-CoV-2 infection was confirmed by a positive reverse-transcription polymerase chain reaction (RT-PCR) assay performed on a nasopharyngeal swab. Laboratory investigations revealed leukocytosis (11,200/mm^3) with neutrophilia and lymphopenia (787/mm^3), elevated C-reactive protein (22 mg/L), and mildly increased D-dimer levels (650 ng/mL). Renal function was preserved, with mild hyponatremia (132 mmol/L) and normal potassium levels, while liver function tests showed transient elevations of aminotransferases (ALT 69 U/L,

Table 1. Longitudinal Laboratory and Functional Assessment During Acute COVID-19 and Follow-up

Parameters	Reference	Acute Phase	Day 21	3 Months	6 Months	12 Months
WBC	4,000–10,000/mm³	11,200	7,840	7,950	8,640	6,520
Neutrophils	1,800–7,500/mm³	9,968	6,500	5,700	5,300	5,900
Lymphocytes	1,000–4,000/mm³	787	1,100	1,400	1,500	1,400
RBC	4.2–5.8 × 10^6/mm³	4.8	4.9	4.7	4.7	4.9
Hemoglobin	13.5–17.5 g/dL	14.2	14.5	14.6	15.5	14.4
Platelets	150–400 × 10^3/mm³	430 × 10^3	400 × 10^3	489 × 10^3	372 × 10^3	456 × 10^3
CRP	<5 mg/L	28 mg/L	12	9	8	9
D-dimer	<500 ng/mL	650	600	700	620	580
PT rate	70–100%	75	70	68	73	70
aPTT / TCK	25–50 sec	35	31	33	33	32
Fibrinogen	2–4 g/L	4,7	4	3.8	4	4,5
Ferritin	30–300 ng/mL	N/A	344	337	330	313
Blood Glucose	0.8–1.1 g/L	1.30	1.05	1.12	1.06	1.17
Urea	2.5–7.1 mmol/L	4,2	3,5	3,6	3,5	5,5
Creatinine	0.7–1.3 mg/dL	1.1	1.0	1.0	0.85	0.95
Na^+	135–145 mmol/L	132	138	132	140	138
K^+	3.5–5.0 mmol/L	4.0	4.1	3.7	4.0	4.0
ALT	7–35 U/L	69	38	35	35	36
AST	10–40 U/L	83	38	28	27	32
Bilirubin (total)	0.2–1.2 mg/dL	1.0	0.9	0.9	0.9	0.9
Albumine	0.2–1.2 mg/dL	1,03	0,96	0,95	0,87	0,79
LDH	120–246 U/L	N/A	280	260	250	256
CPK	30–200 U/L	N/A	420	380	320	289
Serum 25-hydroxyvitamin D level	Sufficiency/ Adequate: 30–50 ng/mL	N/A	82	79	73	98
Serum calcium	8.6–10.2 mg/dL	N/A	9,6	N/A	N/A	N/A
AAN	N/A	N/A	Negative	N/A	Negative	N/A
anti-Jo-1 antibodies	N/A	N/A	Negative	N/A	Negative	N/A

WBC: White Blood Cells; **CRP**: C-Reactive Protein; **D-dimer**: fibrin degradation products; **RBC: Red Blood Cells; Na^+**: serum sodium; **K^+**: serum potassium; **ALT**: alanine aminotransferase; **AST**: aspartate aminotransferase; **LDH**: lactate dehydrogenase; **CPK**: creatine phosphokinase; **PT**: Prothrombin; **aPTT / TCK**: activated Partial Thromboplastin Time (Kaolin); **ANA**: antinuclear antibodies; **Anti-Jo-1 antibodis**: anti–histidyl-tRNA synthetase antibodies; **N/A**: Not Applicable.

AST 73 U/L) (Table 1). Cardiac evaluation remained unremarkable.

Treatment and early evolution

In accordance with national recommendations and in the absence of hypoxemia, the patient was managed as an outpatient with close follow-up. Treatment included intensified short-acting bronchodilators, oral prednisone (60 mg daily with tapering), a short course of azithromycin, vitamin C, zinc and magnesium supplementation, dietary counseling, and daily capillary blood glucose monitoring, which remained within normal limits throughout the acute phase.

Table 2. Time Course of Neuromuscular and Functional Recovery after COVID-19 Associated Sarcopenia

Parameter/Muscle Group	Baseline	Day 21	3 Months	6 Months	12 Months
Weight (kg)	67	67	69	70	71
Quadriceps (knee extension)	N/A	3+/5	4-/5	4/5	4/5
Hamstrings (knee flexion)	N/A	3+/5	4-/5	4/5	4+/5
Hip Flexors	N/A	3+/5	4-/5	4/5	4+/5
Hip Extensors	N/A	3+/5	4-/5	4/5	4/5
Deltoids (shoulder abduction)	N/A	4/5	4/5	4+/5	4+/5
Biceps	N/A	4/5	4/5	4/5	4+/5
Triceps	N/A	4/5	4/5	4+/5	4/5
Handgrip Strength (kg)	N/A	22 kg	24 kg	26 kg	26 kg
Chair Stand Test (5 rises, sec)	N/A	18	15	14	14
6MWD (meters)	N/A	290	370	384	397
Gait Speed (m/s)	N/A	0.9	1.0	1.1	1.2
MRC Sum Score (Total, /50)	N/A	29	32	34	34

N/A: Not Applicable; **6WWT**: 6-Minute Walk Test; **MRC**: Medical Research Council.
MRC Scale: Each muscle is graded on a scale from 0 to 5: 0 = no visible contraction; 1 = palpable or visible contraction without movement; 2 = movement possible but not against gravity; 3 = movement against gravity but not against resistance; 4 = movement against moderate resistance; 5 = normal muscle strength. For intermediate notations: 3+ = 3.5 ; 4− = 3.75; 4+ = 4.25.
To calculate the **MRC sum score**, the scores of the muscles assessed on one side can be summed, and multiplied by 2 if bilateral assessment is performed, yielding a total score out of 50.

At day 3, fever had resolved and respiratory symptoms improved, with normalization of respiratory rate and oxygen saturation. However, the patient reported profound fatigue and marked functional decline, particularly involving the lower limbs, prompting initiation of corticosteroid tapering.

Post-acute evaluation and follow-up

At day 21, the patient continued to report persistent muscular fatigue, with no change in body weight (67 kg) compared with baseline. Despite stable cardiopulmonary parameters, marked proximal muscle weakness was observed, predominantly affecting the shoulder girdle, hip flexors, and quadriceps, with an MRC sum score of 29. Functional assessment revealed reduced gait speed (0.9 m/s), decreased handgrip strength (22 kg), and a substantially impaired 6-minute walk distance of 290 m (Table 2).

Laboratory investigations demonstrated persistent mild lymphopenia, low-grade systemic inflammation, and elevated muscle enzymes (creatine phosphokinase 420 U/L, lactate dehydrogenase 280 U/L). In contrast, coagulation indices, renal function, hepatic parameters, serum albumin, serum calcium, and 25-hydroxyvitamin D levels were within normal ranges. An immunological work-up for inflammatory myopathy, including antinuclear antibodies (ANA) and anti–Jo-1 (anti-histidyl–tRNA synthetase) antibodies, was negative.

A structured respiratory and musculoskeletal rehabilitation program was subsequently initiated, combining diaphragmatic breathing, postural training, and low-intensity resistance exercises targeting major proximal muscle groups. Nutritional management was incorporated, with a daily protein intake of 1.2–1.5 g/kg/day, to support muscle protein synthesis and recovery from sarcopenia. Regular dietary counseling ensured adequate energy intake and micronutrient balance to optimize functional recovery during rehabilitation.

At three months, modest functional improvement was observed, with partial recovery of muscle strength (MRC 32), gait speed (1.0 m/s), handgrip strength

(24 kg), and 6MWD (370 m) (Table 2). This functional recovery was accompanied by a modest weight gain of approximately 2 kg compared with baseline, reflecting early nutritional and anabolic response. Inflammatory markers and muscle enzymes remained mildly elevated (Table 1). Rehabilitation was continued with progressive resistance training.

By six months, further gains were noted, including normalization of leukocyte counts, improvement in muscle strength (MRC 34), gait speed (1.1 m/s), handgrip strength (26 kg), and 6MWD (384 m) (Table 2). Body weight showed an additional increase of approximately 1 kg, indicating continued, albeit gradual, restoration of muscle mass. Muscle enzymes and inflammatory markers showed a progressive decline (Table 1). Rehabilitation intensity was increased, with a specific focus on proximal muscle groups.

At twelve months, the patient achieved near-complete functional recovery, with an MRC sum score of 34, gait speed of 1.2 m/s, handgrip strength of 26 kg, and 6MWD of 397 m (Table 2). Body weight increased by a further 1 kg, resulting in a total weight gain of approximately 5 kg over the follow-up period. Most laboratory parameters normalized, although low-grade elevations of CRP, D-dimer, and muscle enzymes persisted (Table 1), suggesting ongoing tissue remodeling.

Rehabilitation was maintained with high-intensity resistance training focused on the lower limbs and shoulder girdle, combined with ongoing respiratory exercises and individualized nutritional support, aiming to consolidate gains in muscle mass and functional capacity.

Over a 12-month follow-up, the patient demonstrated progressive recovery of muscle strength, functional exercise capacity, and body weight, alongside normalization of most laboratory parameters. The slow and limited weight gain underscores the catabolic burden associated with post-acute COVID-19 in the context of underlying COPD. Persistent mild inflammation, slightly elevated D-dimer, and CPK levels suggest ongoing low-grade tissue remodeling and residual muscular involvement. Early initiation and sustained implementation of a structured rehabilitation program integrating respiratory therapy, targeted resistance training, and nutritional support were critical for functional recovery and mitigation of sarcopenia.

Differential diagnosis and workup

Given the persistent and debilitating muscle weakness with visible muscle wasting, the following differential diagnoses were considered:

1. Autoimmune myopathy was excluded. Muscle enzymes were only mildly elevated and remained slightly above the normal range even after 12 months, and serological testing, including antinuclear antibodies (ANA) and anti-Jo-1 antibodies, was negative. No articular or cutaneous manifestations suggestive of inflammatory myopathy were present.
2. Steroid-induced myopathy was considered unlikely. The patient had received a brief course of oral corticosteroids (tapered over approximately two weeks) without ICU admission or prolonged high-dose exposure.
3. Myasthenia gravis was excluded. Anti-acetylcholine receptor and anti-MuSK antibodies were negative, chest CT revealed no thymic abnormalities, and muscle weakness improved with rest, inconsistent with the fatigability pattern typical of myasthenia gravis.
4. Critical illness myopathy was excluded. The patient had no ICU stay, no prolonged mechanical ventilation, and no exposure to neuromuscular blocking agents.
5. Following systematic exclusion of alternative causes, the patient was diagnosed with COVID-19 precipitated secondary sarcopenia in the context of COPD GOLD grade B.

Diagnostic criteria supporting the diagnosis included proximal muscle weakness, visible muscle wasting (notably in biceps grooves), functional impairment in daily activities, and persistently mildly elevated CPK reflecting low-grade ongoing muscle injury. The temporal onset after COVID-19, in a

previously stable patient, and the exclusion of autoimmune, endocrine, and primary neuromuscular disorders further supported this diagnosis.

Contributing factors were multifactorial, pre-existing COPD with chronic systemic inflammation, COVID-19 related systemic inflammation and direct myopathic effects, short-term corticosteroid therapy, prolonged immobilization during and after acute illness, potential nutritional deficiencies during infection, and age-related susceptibility to muscle loss.

Discussion

This case highlights severe secondary sarcopenia unmasked by COVID-19 in a 67-year-old patient with COPD GOLD grade B who had previously preserved functional capacity. Notably, before SARS-CoV-2 infection, the patient exhibited stable respiratory disease, preserved exercise tolerance (6MWD 420 m), and no clinical or functional signs of muscle impairment, supporting the absence of pre-existing overt sarcopenia. The abrupt functional decline observed three weeks after a mild COVID-19 episode strongly suggests a causal and precipitating role of SARS-CoV-2 infection.

Recent evidence suggests that COVID-19 may directly affect skeletal muscle through multiple pathways: (1) direct viral invasion via ACE2 receptors expressed on muscle cells,[13] (2) systemic hyperinflammation with elevated cytokines promoting muscle catabolism,[14] (3) prolonged immobilization during illness,[15] (4) corticosteroid therapy,[14] and (5) hypoxemia.[15] In the present case, despite the absence of hypoxemia and the mild radiological extent of pulmonary involvement (<10%), the patient developed marked proximal muscle weakness and functional decline, indicating that even non-severe COVID-19 can induce significant myopathic effects in susceptible individuals. In patients with pre-existing COPD, these factors may synergistically accelerate muscle loss, precipitating clinically significant sarcopenia even after relatively mild COVID-19 infections.[12,16,17]

Despite a normal body mass index (23.2 kg/m^2), patients may exhibit marked proximal muscle weakness and visible muscle wasting, illustrating that BMI alone fails to detect qualitative and quantitative muscle loss.[3,18] This dissociation was clearly illustrated in our patient, whose BMI remained stable while objective assessments revealed profound reductions in handgrip strength, gait speed, MRC sum score, and 6MWD. Subclinical sarcopenia may remain undiagnosed until an acute catabolic stress, such as COVID-19, reveals underlying vulnerability.[18,19]

Muscle impairment predominantly affects proximal muscle groups, which are particularly sensitive to systemic inflammation and disuse.[3,6] In this case, involvement of the shoulder girdle, hip flexors, and quadriceps led to substantial limitations in standing, walking, and endurance, explaining the sharp decline in 6MWD and gait speed despite preserved cardiopulmonary parameters. Lower-limb muscles (quadriceps, hamstrings, hip flexors, hip extensors) are essential for standing, gait, and postural stability, while proximal upper-limb muscles (deltoids, biceps, triceps) support functional independence in activities of daily living. Muscle strength is quantified using the Medical Research Council (MRC) sum score[20] and handgrip dynamometry,[21] validated surrogate markers of global muscle function.

Short-term corticosteroid exposure may compound catabolism. Corticosteroids inhibit protein synthesis,[22] enhance proteolysis,[22] promote insulin resistance,[22] and preferentially affect type II fibers.[22] Although corticosteroid exposure in our patient was brief and outpatient-based, it likely acted as an additional catabolic trigger in the context of COVID-19 related inflammation and pre-existing COPD-associated vulnerability. Clinical and laboratory findings corroborate active muscle involvement, including proximal weakness,[10] visible quadriceps and biceps wasting,[12] and elevated creatine phosphokinase,[23] as observed in this patient.

Alternative causes of muscle weakness were systematically excluded. Autoimmune myopathies, myasthenia gravis, steroid-induced myopathy, and critical illness myopathy were ruled out based on clinical evaluation, serology, brief corticosteroid exposure, and outpatient course.[22,24] The persistence of mild CPK elevation over 12 months, in parallel with gradual functional recovery, further supports a diagnosis of secondary sarcopenia rather than a primary neuromuscular disorder.

The interaction between pre-existing COPD and COVID-19 exemplifies synergistic vulnerability. COPD predisposes to sarcopenia via chronic inflammation,[4–6] oxidative stress,[5] intermittent hypoxemia,[4] and sedentary behavior.[6,25] In this patient, COPD-related systemic inflammation likely constituted a subclinical baseline vulnerability, upon which COVID-19 exerted an acute inflammatory and metabolic insult, overwhelming muscle homeostasis. Superimposed COVID-19 triggers an acute inflammatory,[10] metabolic,[12] and immobilization-related insult,[15] overwhelming muscle homeostasis.

Comprehensive rehabilitation, including progressive resistance training,[26] respiratory muscle exercises,[27] and individualized nutritional support, remains the cornerstone of management.[28,29] In the present case, early initiation of combined respiratory and musculoskeletal rehabilitation, together with adequate protein intake, was associated with progressive recovery of muscle strength and functional capacity over 12 months. Early gains in strength are primarily driven by enhanced motor unit recruitment and neuromuscular efficiency, whereas hypertrophy may be limited by persistent low-grade inflammation, anabolic resistance, and residual post-COVID metabolic dysregulation.

These findings support systematic sarcopenia screening in COPD using combined assessments of muscle strength,[20] muscle mass,[21] and physical performance, with tools such as the SARC-F (Strength, Assistance in walking, Rise from a chair, Climb stairs, and Falls) questionnaire aiding early detection.[25,29] In our patient, sarcopenia would likely have remained undetected without systematic functional assessment, as pulmonary function tests alone failed to reflect the severity of post-COVID functional impairment. Long-term monitoring is essential, as post-COVID sarcopenia may persist for months and is not captured by pulmonary function tests alone.

Conclusion

This case illustrates the development of severe secondary sarcopenia in a 67-year-old male with COPD GOLD grade B following a mild COVID-19 infection, despite preserved baseline function. The patient experienced progressive proximal muscle weakness, visible muscle wasting, and impaired daily activities three months post-infection.

It underscores that COVID-19 can trigger clinically significant sarcopenia even after mild respiratory illness, particularly in patients with pre-existing chronic pulmonary disease. BMI alone is insufficient to detect muscle loss, highlighting the need for systematic assessment of muscle strength, mass, and physical performance.

The combination of chronic inflammation, acute viral injury, immobilization, corticosteroid exposure, and nutritional stress likely contributed to accelerated muscle loss. Early screening, structured rehabilitation, resistance and aerobic training, and nutritional optimization are essential to prevent irreversible functional decline.

Limitations of this case include absence of pre-COVID sarcopenia assessment, lack of quantitative muscle mass evaluation, and the single-case design, which limits generalizability. Nevertheless, it underscores the need for heightened awareness of post-COVID sarcopenia in COPD, mechanisms of muscle loss, and the importance of proactive screening and early intervention. Future research should include prospective cohort studies to define incidence and risk factors, mechanistic studies on SARS-CoV-2 muscle tropism, and randomized trials to optimize rehabilitation strategies. This report emphasizes sarcopenia as a critical post-COVID complication in COPD patients, reinforcing the importance of vigilant monitoring and timely intervention to preserve functional independence and quality of life.

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From: Laboratory of Neurosciences, Youcef El Khatib University of Health Sciences, Algeria (LL, NDD, NH, SN)
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