Prolonged bilateral lower limb weakness: Is it due to COVID-19 or something else?

Anshuman Srivastava MD, Sandhya J. Kadam MD, Rishika Sharma MD, Archana Reddy Bongurala MD

Abstract

People who experience musculoskeletal symptoms during COVID-19 infection usually have long COVID-19. If a patient has symptoms after 30 days of COVID-19 infection, it is considered long COVID-19. Symptoms like pain, tenderness, and stiffness are common in the neck, back, and shoulders. Symptoms can be related to the body's immune response to the virus. During complicated COVID-19 infection, patients may experience muscle weakness and decreased bone density, leading to osteoporosis attributed to prolonged bed rest and reduced physical activity.

Several disorders can cause bilateral lower limb weakness. Spinal disorders like cord compression caused by a tumor, herniated disc, infarction, cauda equina syndrome, and transverse myelitis can cause bilateral lower limb weakness. Inflammatory conditions like Guillain-Barré syndrome, chronic inflammatory demyelinating polyneuropathy (CIDP), inflammatory myopathy, and other conditions such as amyotrophic lateral sclerosis (ALS), multiple sclerosis, Parkinson's disease, myasthenia gravis, or a pinched nerve can also cause bilateral lower limb weakness. In addition, potassium deficiency, tick paralysis, and leg weakness as a side effect of medication can present with bilateral lower limb weakness.

This case report presents a 59-year-old ambulatory patient with multiple underlying medical conditions who contracted COVID-19 and developed bilateral lower limb weakness, which did not improve even with vigorous physical therapy sessions, creating a challenge to find the underlying cause of weakness.

Keywords: COVID-19, lower limb weakness, musculoskeletal symptoms, creatine kinase, myalgia

Introduction

Lower limb weakness often has a multifactorial etiology. Systemic inflammation, viral infiltration, muscle hypoxia, muscle disuse, malnutrition, and medication side effects can explain the responsible factors. Physical therapy may benefit these patients by reducing local and systemic inflammation.[1]

Corresponding author: **Anshuman Srivastava**
Contact Information: Anshuman.Srivastava87@gmail.com
DOI: 10.12746/swjm.v13i55.1463

Along with other multi-systemic manifestations, musculoskeletal symptoms occur in COVID-19 patients experiencing severe ischemic myalgia. Muscle weakness can be observed in all patients, and the degree of muscle dysfunction is particularly linked to disease activity. Muscular involvement in COVID-19 can be a combination of myalgia, physical fatigue, and functional impairment.[2]

This case report presents a patient who, after contracting COVID-19, developed severe muscle weakness in both lower limbs and has remained wheelchair-dependent for three years following the infection.

Table 1. Lab Results

	COVID	CK U/L	Creat mg/dl	AST U/L	ALT U/L	AlkPO4 IU/L	BUN mg/dl
2 months ago	Positive	Normal	Normal	Normal	Normal	Normal	Normal
At ER presentation	Positive	21310	4.09	1042	473	108	50
Day 1		26980	4.24	999	464	113	47
Day 2		31150	4.29	877	423	114	42
Day 3		35200	4.24	772	367	99	36
Day 4		39560	4.50	797	366	110	35
Day 5		9900	4.84	767	364	122	114
Day 6		33012	5.55	657	339	128	116
Day 7		31500	3.69	483	278	121	116
Day 9		17478	3.37	398	255	123	117
Day 10		7349	3.89	214	166	90	124
Day 11		5742	3.63	182	136	95	147
Day 12		3604	3.81	125	66	84	86
Day 13		2427	3.84	106	44	85	73
Day 14		1851	3.68	99	36	114	88
Day 19		318	1.51	79	33	73	50
Day of discharge			1.66	60	33	85	36
After 5 months		101	1.19	40	25	86	21

CK: creatine kinase, Creat: creatinine, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, ALKPO4: alkaline phosphatase, BUN: blood urea nitrogen.

Case

A 59-year-old man with a past medical history of insulin-dependent diabetes mellitus, hepatitis C, chronic kidney disease (CKD) stage III, anemia of chronic disease, hypertension, chronic pain on methadone, moderate central canal stenosis of the lumbar spine presented to the emergency room (ER) with dark-colored urine, bilateral lower limb weakness, and muscle pain. He had a history of low back pain for a few months, managed with cyclobenzaprine. He was able to ambulate inside and around his house. The patient denied taking any recreational drugs or any changes in his medications. He was on rosuvastatin 10 mg per day for cardiovascular risk. In the ER, the patient reported that he had muscle weakness and the worsening of both lower limb weakness for the past 2 months after contracting COVID-19 infection and started to have dark-colored urine for the past 3 days. On admission, he was found to have high levels of creatinine kinase (CK) at 21,000 IU/ml, elevated liver enzymes, and labs suggesting acute on chronic kidney disease stage III (Table 1). He had urinary retention in the ER, requiring Foley's catheterization. He tested COVID-positive 2 months ago for the first time and continued to test positive multiple times, including during this presentation. His COVID-19 symptoms were mild at the initial presentation, and he did not require hospitalization. He did not receive the COVID-19 vaccine.

In the ER, he was evaluated by a neurosurgeon for worsening bilateral lower limb weakness. Magnetic resonance imaging (MRI) of the lumbar spine suggested multilevel disc disease and facet arthropathy, with multilevel central canal stenosis and neural foraminal narrowing (Figure 1). Neurosurgery suggested that the lower limb weakness was likely attributed to rhabdomyolysis related to an underlying possible medical cause and not to the surgical pathology, so no acute surgical intervention was advised. An MRI of the brain was normal. A nephrologist was consulted for worsening CKD, and the patient was started on IV fluids.

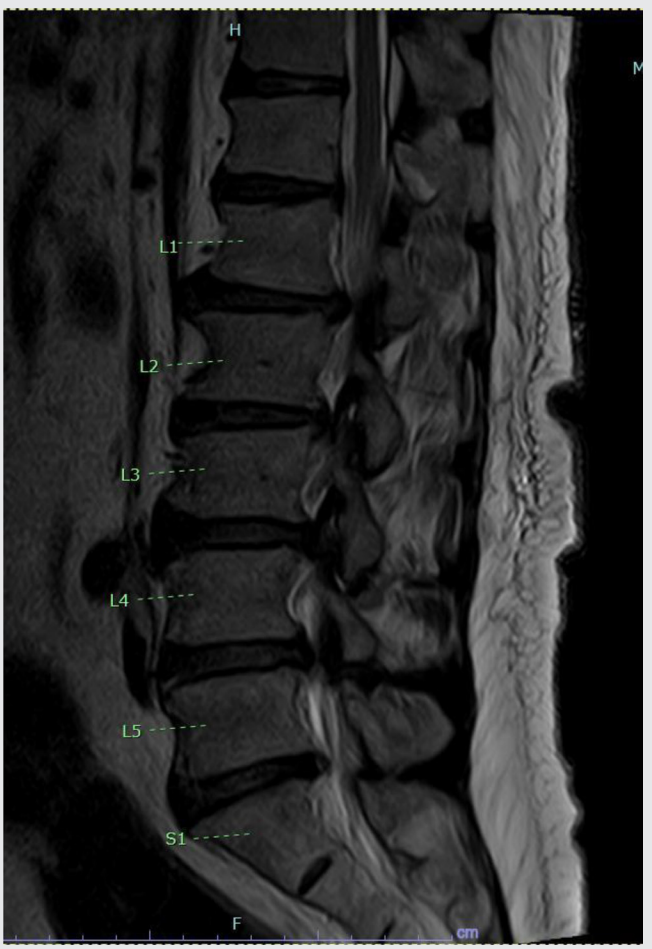

Figure 1. MRI of Lumbar Spine.

The etiology of lower limb weakness was still unclear. A neurologist recommended labs to rule out possible autoimmune causes. Lab tests including antinuclear antibody, Sjögren syndrome antigen A, and Sjögren syndrome antigen B were negative (Table 2). Within 2 days of admission, the patient's renal functions worsened to the point of needing hemodialysis. His hospital stay was also complicated by acute gastrointestinal bleeding, which was managed with esophagogastroduodenoscopy. The statin was discontinued to rule out muscle-related side effects, which did not improve the patient's symptoms.

The patient continued to have severe myopathy and myositis with severe bilateral lower limb weakness and was not able to ambulate. He continued to have elevated transaminase and CK levels. An MRI

Table 2. Autoimmune Labs

	Results	Normal Range
GBMIgG	Negative	6–16 g/dl
MPO Ab	Negative	Less than 400 pmol/L
ANA	Negative	Negative
SSA Ab	6	Less than 25 Units/L
SSA 60	2	0–73 Units/ml
JO-1 Ab	3	Less than 40 AU/ml
SRP Ab	Negative	Negative

Glomerular basement membrane (GBM) antibodies, myeloperoxidase (MPO) antibodies, ANA antinuclear antibodies, SSA antibodies, Sjögren's-syndrome-related antigen A antibodies, SSB Sjögren's Syndrome Antigen B antibodies, JO-1Ab anti-Jo-1 antibodies, Anti-signal recognition particle (SRP).

of the bilateral lower extremity showed diffuse severe edema throughout the thigh musculature bilaterally with associated subcutaneous edema (Figure 2). Due to nonspecific findings on MRI limbs, rhabdomyolysis

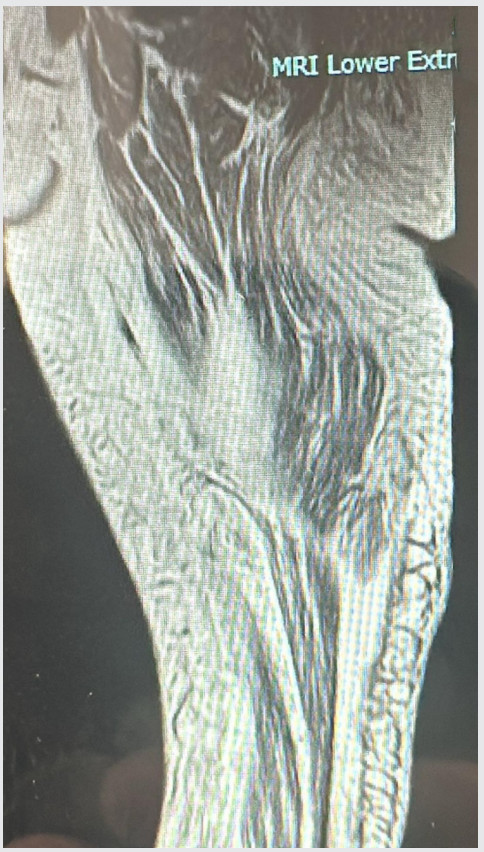

Figure 2. MRI of Lower Extremity.

was considered an important differential diagnosis. There was no improvement in the patient's symptoms, so rheumatology was consulted, who advised to start the patient on prednisone; a muscle biopsy of the right thigh was performed, which showed severe necrotizing myopathy and ultrastructural evidence of coronavirus-like particles. Prednisolone was weaned off once biopsy results were available.

Intravenous fluids, hemodialysis, and supportive management helped the patient normalize all the labs except creatinine kinase. He was started on physical therapy during hospitalization, which did not help much. His creatinine stabilized around 1.5 mg/dl, and he was taken off the hemodialysis. The patient continued to have lower limb weakness and was non-ambulatory and wheelchair bound. He was then discharged home with family care and outpatient physical therapy. He was compliant with his medications and appointments. He was restarted on the rosuvastatin. The patient did follow up with his primary care physician and nephrologist as scheduled. All his labs are at the baseline, with improvement in creatinine kinase but with the same degree of bilateral lower limb weakness, keeping him wheelchair bound.

Discussion

Multiple studies have been done on post-COVID-19 syndrome (PCS), in which patients were found to have symptoms involving musculoskeletal, pulmonary, digestive, and neurological system involvement unrelated to the severity of COVID-19 infection. Symptoms are likely related to long-term antibody responses to infection and high inter-individual variability.[3] According to another study published during the pandemic, most COVID-19 ICU survivors developed acquired limb muscle weakness; around half of the patients continued to have severely limited function after one month of COVID-19 diagnosis.[4]

Muscle weakness related to COVID-19 can be severe, involving the diaphragm, reported in ICU patients whose muscle biopsy showed muscle atrophy without evidence of inflammatory myopathy, making the presence of pre-existing autoimmune myopathy unlikely.[5] Long-term COVID can lead to lower exercise capacity, which could be related to tissue infiltration of amyloid-containing deposits in skeletal muscles. Amyloid deposits can impair peripheral oxygenation, leading to exercise intolerance.[6]

Statins are advised for COVID-19 patients due to their expected benefits, but adverse outcome risks, especially rhabdomyolysis, in hospitalized COVID-19 patients caution clinicians to consider carefully their risks and benefits. Our patient was on statins before contracting the COVID-19 infection. Based on possible side effects, statins were stopped to see if the patient's high CK was related to statins. However, stopping statins did not help to improve CK in our patient.[7]

Long COVID can present with symptoms involving any system in the body. Our patient has persistent bilateral lower limb weakness post-COVID-19 infection that did not improve even 3 years post-illness. Long COVID symptoms could be generalized or specific: malaise, body aches, musculoskeletal pain, prolonged loss of taste and smell, or specific symptoms involving respiratory and cardiovascular systems can persist. Symptoms could depend on demographics, patients' pre-existing conditions, COVID-19 vaccination status, and severity of COVID-19 illness.[8]

Post-acute cardiovascular sequelae of COVID-19 (PASC) are reported as lingering cardiac symptoms. During acute illness, cardiac injury is caused by cytokine release, leading to ischemic and thrombotic complications, resulting in myocardial necrosis. Long-term disability and reduced quality of life can occur due to these complications.[9] Patients with pre-existing cardiac problems may be more vulnerable to having long-term cardiac complications, and thrombotic complications may occur after a few months after COVID-19 infection.[10]

Conclusion

A few case reports are present in the literature that report chronic muscle weakness and muscle necrosis due to COVID-19 infection, but no significant studies are available to guide clinicians. This case report intensifies the need for more studies to establish the correlation between COVID-19 infection and muscle necrosis, chronic muscle fatigue, and weakness.

Acknowledgment

We would like to thank the patient for providing written informed consent to publish this case report.

Article citation: Srivastava A, Kadam SJ, Sharma R, Bongurala AR. Prolonged bilateral lower limb weakness: Is it due to COVID-19 or something else? The Southwest Journal of Medicine 2025;13(55):26–30
From: Department of Family Medicine, Family HealthCare Network, Visalia, CA, USA (AS) Department of Pediatrics, Family HealthCare Network, Visalia, CA, USA (SJK) Department of Pediatrics, Omni Family Health, Bakersfield, CA, USA (RS, ARB)
Submitted: 12/24/2024
Accepted: 4/1/2025
Conflicts of interest: none

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