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ACUTE VIRAL-INDUCED MYOPATHY FOLLOWING COVID-19

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ABSTRACT

Background: Coronavirus disease 2019 (COVID-19) has been associated with myopathies that may arise from immune dysregulation, autoimmunity, or possible direct viral injury, though the exact mechanisms remain unclear. Recognizing these cases is clinically important, as they can resemble primary autoimmune myopathies but may follow a different course and prognosis.

Case report: We report a 59-year-old previously healthy woman who developed acute myopathy shortly after COVID-19. She presented with diffuse myalgia, fatigue, exertional dyspnoea, dizziness, fine motor difficulties, and urinary incontinence. Neurological examination showed preserved reflexes, no sensory deficits, and normal cerebrospinal fluid, excluding Guillain–Barré syndrome and transverse myelitis. Laboratory tests revealed elevated lactate dehydrogenase (616 U/l; ref. 105–205), alanine aminotransferase (239 U/l; ref. <70), and plasma myoglobin (60–74 µg/l; ref. <45). The myositis antibody panel was negative except for isolated CHD4 (anti-Mi-2) positivity, deemed non-specific. She was treated with intravenous fluids, paracetamol, ibuprofen, and physiotherapy. Symptoms resolved within 1 week, and she was discharged on day 10. At 6-month follow-up, she remained fully recovered without recurrence.

Conclusion: This case illustrates that COVID-19 can cause acute, self-limiting viral myopathy in otherwise healthy individuals, emphasizing the need for clinical awareness and supportive management.

KEYWORDS

SARS-CoV-2, COVID-19, viral infection, myopathy

LEARNING POINTS

- Coronavirus disease 2019 can cause acute, self-limiting viral myopathy that mimics autoimmune myositis with positive anti-Mi-2 even in previously healthy individuals.
- Careful exclusion of neuropathic and autoimmune causes prevents misdiagnosis, overtreatment, and avoiding unnecessary invasive tests.
- Supportive management with hydration, analgesia, and physiotherapy leads to full recovery.

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INTRODUCTION

Coronavirus disease 2019 (COVID-19) has been associated with a wide spectrum of complications, ranging from respiratory failure and multiorgan involvement to autoimmune phenomena[1]. Among the less common but increasingly recognized manifestations are coronavirus disease 2019 (COVID-19)–induced or -associated myopathies, typically presenting with muscle pain, weakness, and elevated muscle enzyme levels[1,2].

The underlying pathogenesis appears multifactorial, however; immune dysregulation and infection-triggered autoimmunity are thought to play a central role, as illustrated by reports of new-onset dermatomyositis and necrotizing autoimmune myopathy following severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection[1,2]. Similar cases have also been described after COVID-19 vaccination, suggesting that both natural infection and vaccine-induced immune activation may precipitate clinically significant muscle disease[3]. Direct viral injury has also been proposed. Muscle biopsy studies have revealed myofiber necrosis with limited inflammation and occasional viral particles, consistent with a combination of direct cytopathic effects and secondary immune-mediated damage[4]. Taken together, these findings suggest that COVID-19–associated myopathy exists along a clinical spectrum, from mild, self-limiting muscle involvement to severe immune-mediated inflammatory myositis[5]. However, diagnosis and management of such cases remain clinically challenging.

Diagnosis of such cases is clinically important, as they can mimic primary autoimmune myopathies but often follow a distinct course and prognosis. By presenting this case, we aim to contribute to the growing body of evidence, highlight the diagnostic challenges, and support clinicians in managing this rare but relevant complication.

CASE DESCRIPTION

The patient was a 59-year-old woman, previously healthy and physically active, with no history of regular medication use, herbal remedies, or family history of neuromuscular disease. She developed upper respiratory tract symptoms and tested positive for SARS-CoV-2 at home using a rapid antigen test. Shortly thereafter, she developed progressive fatigue, diffuse myalgia, dyspnoea on exertion, dizziness, and difficulty performing fine motor tasks such as writing and using a phone. She also reported urinary incontinence.

Over the next few days, her muscle weakness worsened. On day 4, she presented to the emergency department of our hospital with difficulty getting up from a chair, climbing stairs, and carrying objects, accompanied by shortness of breath. On examination, she was alert and oriented, with normal vital signs apart from tachycardia. Physical examinations revealed diffuse muscle tenderness and pronounced proximal weakness in both upper and lower extremities, while reflexes and sensation remained intact. The patient had received four doses of the Pfizer-BioNTech SARS-CoV-2 vaccine, the last administered 1 year before admission.

Laboratory tests confirmed COVID-19 and demonstrated elevated creatine kinase (CK) (1,370 U/l), alanine aminotransferase (239 U/l), and lactate dehydrogenase (LDH) (616 U/l) *(Table 1)*. Lumbar puncture was unremarkable, with no signs of inflammation or protein elevation. Chest X-ray revealed no remarkable changes *(Fig. 1)* and an electrocardiogram showed sinus tachycardia *(Fig. 2)*.

The differential diagnoses considered included Guillain–Barré syndrome (excluded by preserved reflexes and normal cerebrospinal fluid), transverse myelitis (excluded by normal cerebrospinal fluid and absence of upper motor neuron signs), medication-induced myopathy such as statin or corticosteroid-related (excluded due to absence of relevant drug exposure), and autoimmune inflammatory myopathies (excluded by negative autoantibody testing and lack of characteristic features).

A multidisciplinary team of infectious disease specialists, neurologists, and rheumatologists concluded that the patient had acute post-viral myopathy secondary to COVID-19. Management included intravenous fluids (a total of 3 litres comprising normal saline and Ringer's lactate during admission), analgesia with paracetamol 1 g four times

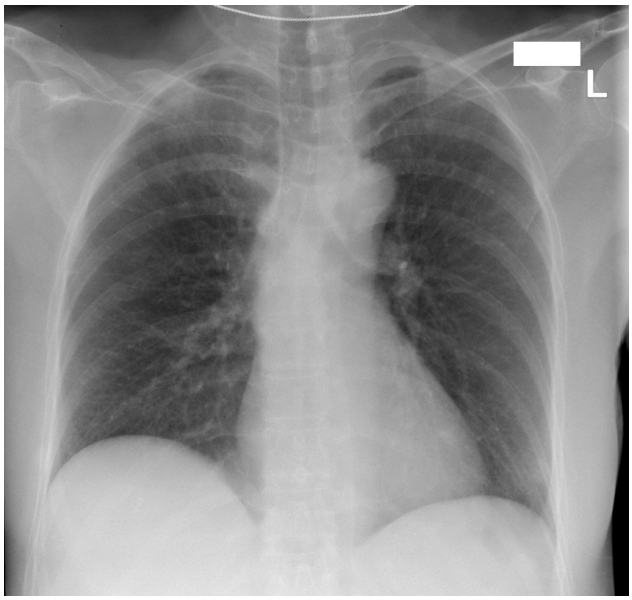


Figure 1. Chest X-ray showing clear lung fields and a normal cardiac silhouette.

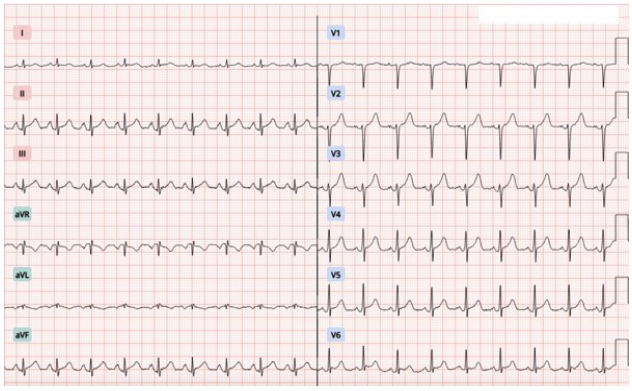


Figure 2. Twelve-lead electrocardiogram demonstrating sinus tachycardia.

Laboratory examination		Result	Reference range (adults)
Complete blood count (CBC)	White blood cells (WBC) (×10^9/l)	8	3.5–8.8
	Platelets (×10^9/l)	134	145–390
	Red blood cells (RBC) (×10^{12}/l)	5.34	4.3–5.7 (men)
	Lymphocytes (×109/l)	0.98	1.1–3.5
	Monocytes (×10^9/l)	0.93	0.2–0.8
	Neutrophils (×10^9/l)	5.98	1.6–6.1
	Eosinophils (×10^9/l)	0.01	0.04–0.5
	Basophils (×10^9/l)	0.01	0–0.1
	Immature granulocytes (Meta/Myelo/Promyelo) (×10^9/l)	0.04	0
	Haemoglobin (g/dl)	12.6 (7.8 mmol/l)	11.8–15.3 (7.3-9.5 mmol/l)
Liver and pancreas tests	Alanine aminotransferase (ALT) (U/l)	239	<70
	Alkaline phosphatase (ALP) (U/l)	105	35–105
	Lactate dehydrogenase (LDH) (U/l)	616	105–205
	Amylase (U/l)	59	15–100
Cerebrospinal fluid (CSF) analysis	Appearance	Clear	Clear
	Glucose (mmol/l)	4.3	2.2–4.4
	Protein (g/l)	0.25	0.15–0.45
	Lactate (mmol/l)	1.9	1.2–2.1
	Red blood cells (/µl)	~300	0
	White blood cells (mono) (/µl)	<10	0–5
	White blood cells (poly) (/µl)	<10	0–5
Urine analysis	Acetoacetate (mmol/l)	<0.5	<0.5
	Nitrite test	Negative	Negative
	Erythrocytes (/µl)	10	0–5
	Glucose (mmol/l)	<5.5	Negative
	Leukocytes (/µl)	<15	0–10
	pH	5.5	5.0–7.5
	Protein (g/l)	<0.15	<0.15
Thyroid	Thyroid stimulating hormone (TSH) (IU/l)	1.8	0.4–4.8
Markers of muscle injury	Myoglobin (plasma) (µg/l)	60–74	<45
	Creatine kinase (CK) (U/l)	1,370	25–200
Autoantibody panel	Positive	Chromodomain-helicase-DNA (CHD4, anti-Mi-2) antibody	
	Negative	PCNA antibody; 52 kDa Ro protein IgG; double-stranded DNA IgG; DNA topoisomerase I (Scl-70) IgG; EXOSC10 IgG (PM-Scl100); EXOSC9 IgG (PM-Scl75); fibrillarin IgG; histidyl-tRNA ligase (Jo-1) IgG; major centromere B IgG; Mi-2 alpha IgG; ribosomal protein IgG; RNA polymerase III IgG; Sjögren syndrome antibodies (SSA, SSB); Smith IgG; U1 snRNP IgG; cN1A IgG; glycyl-tRNA synthetase IgG (EJ); HMG-CoA reductase IgG; MDA-5 IgG; MORC3 IgG; polymyositis antibodies (Ku, PL-12, PL-7, SRP); SAE1 IgG; and TIF1-gamma IgG. The only positive finding was chromodomain-helicase-DNA (CHD4, anti-Mi-2) antibody.	
Infection panel	Positive	SARS-CoV-2	
	Negative	Influenza A&B, Hepatitis A virus antibodies; hepatitis B core antibody; hepatitis B e antigen; hepatitis B surface antigen; hepatitis E virus antibodies; and leptospira sp. antibodies. Hepatitis B surface antibody was 0.9 IU/l, and hepatitis B DNA was not detected.	

Abbreviations: DNA, deoxyribonucleic acid; CHD4, chromo-domain-helicase-DNA; PCNA, proliferating cell nuclear antigen; tRNA, transfer ribonucleic acid; Scl-70, DNA topoisomerase I autoantibody; PM-Scl, Polymyositis/scleroderma autoantigen; SSA/SSB, Sjögren's-syndrome-related antigen A/B; snRNP, small nuclear ribonucleoprotein; HMG-CoA, hydroxymethylglutaryl-CoA; SRP, signal recognition particle; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2.

Table 1. Laboratory test results.

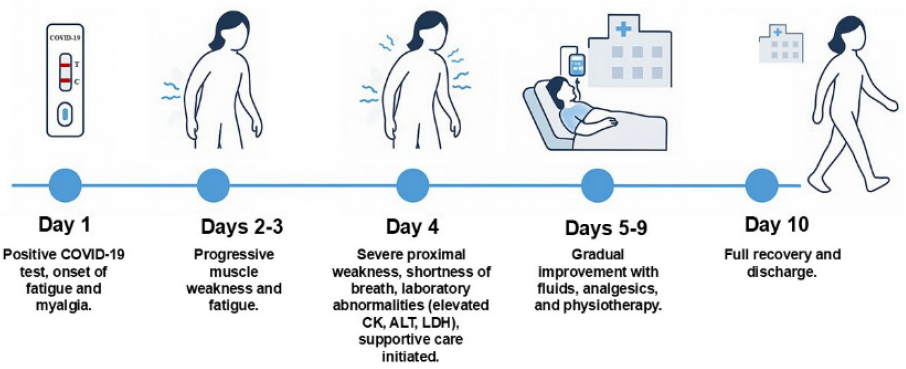


Figure 3. Timeline of the patient's illness and recovery, showing onset of COVID-19 symptoms, development of myopathy, initiation of treatment (intravenous fluids, analgesics, physiotherapy), and gradual clinical improvement leading to discharge and full recovery by day 10, with no recurrence at 3-month follow-up.

daily and ibuprofen 400 mg three times daily, and initiation of early physiotherapy. Her symptoms gradually improved, with resolution of myalgia and restoration of muscle strength within one week. By day 10, she was discharged in near-complete recovery and able to resume her daily activities *(Fig. 3)*.

The patient was followed for 6 months. She returned to work and resumed horse riding 4–5 times per week, as well as fitness training 3 times per week.

DISCUSSION

Viral infections have long been recognized as triggers of acute myopathies and Influenza A and B, enteroviruses such as Coxsackie virus and Echo virus, human immunodeficiency virus, Epstein–Barr virus, cytomegalovirus, and parvovirus B19 are known causes of viral associated myopathies[6]. These precedents highlight that SARS-CoV-2 fits into a broader pattern of viral agents capable of inducing muscle disease[7].

COVID-19-associated myopathy has been described in several forms, including dermatomyositis[1], autoimmune necrotizing myositis[2], and post-vaccination myositis[3]. These heterogeneous presentations suggest complex mechanisms involving immune dysregulation, cytokine-mediated muscle damage, microvascular injury, and possibly direct viral invasion[3]. Histopathological studies have demonstrated myofiber necrosis, perivascular inflammation, and capillary injury in affected patients[4], supporting this multifactorial pathogenesis.

The clinical spectrum ranges from severe immune-mediated myopathies requiring immunosuppressive treatment to mild, self-limiting viral myositis that responds to supportive care[1,2]. Our case belongs to the latter category, with acute but transient muscle weakness, elevated LDH and myoglobin, and full recovery after intravenous fluids, analgesics, and physiotherapy. Importantly, preserved reflexes, normal cerebrospinal fluid, and absence of sensory deficits helped exclude Guillain–Barré syndrome, transverse myelitis, and other neuropathic processes. The autoantibody panel was negative for myositis-specific and myositis-associated antibodies, except for an isolated positivity for chromodomain-helicase-deoxyribonucleic acid (CHD4, anti-Mi-2). While anti-Mi-2 is typically linked to dermatomyositis with characteristic cutaneous features, these were absent, and the short, self-limiting clinical course contrasted with the chronic autoimmune pattern usually associated with dermatomyositis. In a published case, Plavsic et al. described a 36-year-old woman who developed severe autoimmune myopathy after COVID-19 pneumonia, with markedly elevated CK, persistent anti-Mi-2 and anti-Ro52 positivity, and a chronic course requiring corticosteroids, methotrexate, and intravenous immunoglobulin[8]. Taken together, these cases highlight the heterogeneity of COVID-19–associated muscle involvement, ranging from transient viral-induced myopathy to persistent autoimmune disease.

In our case, the advanced diagnostic tools such as magnetic resonance imaging, electromyography, or muscle biopsy were not performed, because the clinical and laboratory features were sufficient to establish the diagnosis. This pragmatic approach reflects real-world settings, where extensive neuromuscular workup may not be required if the clinical course is benign and self-limiting.

Although rare, COVID-19 can lead to acute myopathy even in individuals with no prior neuromuscular disease. This case illustrates the importance of clinical recognition, exclusion of alternative diagnoses, and the potential for full recovery with supportive therapy. Clinicians should remain alert to muscular manifestations of COVID-19, particularly when patients present with unexplained muscle weakness and pain during or shortly after infection. Furthermore, viral infections, including COVID-19, should be considered as differential diagnosis when patients present with signs and symptoms of myopathy. Sharing such experiences broadens awareness, facilitates timely diagnosis, and may improve outcomes in future patients.

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