ACCSCIENCE PUBLISHING

Advanced Neurology

ORIGINAL RESEARCH ARTICLE

Myographic evidence of polymyositis and dermatomyositis in COVID-19 patients

Nareen Haikaz Hasrat[1], Haithem Jawad Kadhum[1], Zaineb Adil Yakob[1], Ali Raheem Hashim[2], and Hassan Ala Farid[3]*

[1]Department of Physiology, College of Medicine, University of Basrah, Basrah, Iraq

[2]Department of Medicine, College of Medicine, University of Basrah, Basrah, Iraq

[3]Institute of Medical and Biomedical Education, St. George's University of London, London, United Kingdom

Abstract

Idiopathic inflammatory myopathies, commonly known as myositis, are a diverse group of disorders defined clinically by persistent muscle weakness and reduced muscle endurance, as well as inflammatory cell infiltrates inside the muscle tissue. Myositis as a complication of coronavirus disease 2019 (COVID-19) has been described in an increasing number of reports. An analytical and cross-sectional study was undertaken in Basrah to analyze nerve conduction studies (NCS) and electromyographic (EMG) data in a COVID-19-affected patient. During the evaluation of 2240 patients, three cases of myositis were reported among the COVID-19 population, two of them with new clinical and EMG evidence of inflammatory myositis after the onset of COVID-19 infection, and one patient had a history of polymyositis before the COVID-19 pandemic, but a relapse was triggered by COVID-19, resulting in respiratory failure and death. The study found that the prevalence of myositis among the COVID-19 population was equal to 0.22%, which is 44 times higher than the prevalence of myositis (0.005%) worldwide before the onset of COVID-19 ($P < 0.001$).

Keywords: Inflammatory myopathy; Polymyositis; Dermatomyositis; COVID-19

***Corresponding author:**
Hassan Ala Farid
(hfarid@sgul.ac.uk)

Citation: Hasrat NH, Kadhum HJ, Yakob ZA, *et al.*, 2023, Myographic evidence of polymyositis and dermatomyositis in COVID-19 patients. *Adv Neuro*. https://doi.org/10.36922/an.378

Received: February 15, 2023

Accepted: May 2, 2023

Published Online: May 25, 2023

1. Introduction

The coronavirus disease 2019 (COVID-19) pandemic was caused by a novel coronavirus, namely, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), which rapidly spread from China to all continents. The disease has spread throughout the globe, culminating in an ongoing epidemic[1]. Acute infection with the new coronavirus that causes COVID-19 disease results in a variety of clinical symptoms, including various neurological abnormalities[2]. Peripheral nervous system symptoms, such as peripheral neuropathies, are often recorded in the medical literature, primarily as Guillain–Barré syndrome (GBS)[3].

Idiopathic inflammatory myopathies, often known as myositis, are a heterogeneous collection of illnesses characterized by chronic muscular weakness and decreased muscle endurance, as well as inflammatory cell infiltrations inside the muscle tissue. Variations in clinical and histological features have led to their classification as polymyositis (PM), dermatomyositis (DM), and inclusion body myositis (IBM)[4]. Along with the presence of

autoantibodies, the frequent occurrence of inflammatory cell infiltrates with a high frequency of T-cells in muscle tissue indicates that these are autoimmune illnesses. Additional organs are commonly affected, including the skin in DM and the lungs, heart, joints, and gastrointestinal tract in both PM and DM, demonstrating that these are systemic autoimmune illnesses. The most commonly accessible enzymatic assays for verifying skeletal muscle involvement are serum level measurements for creatine phosphokinase (CPK), lactate dehydrogenase, aspartate transaminase, and alanine transaminase[4]. Certain EMG alterations, including the size, shape, and recruitment pattern of the motor unit's potential, may distinguish between necrosis and denervation; however, no EMG abnormalities are specific to myositis[4].

In COVID-19-infected individuals, myalgia and an asymptomatic CPK increase are common, but they do not correlate with clinical, electrodiagnostic, or histologic markers of muscle damage, nor with the severity of the underlying infection, nor do they predict the start of myopathy. Myopathy has been described as a clinical and laboratory diagnosis in 0.5% to 3.1% of COVID-19 patients[5].

Myositis as a complication of COVID-19 has been documented in increasing studies. Individuals with proximal myopathy, significant bulbar involvement, and DM-like symptoms, including amyopathic DM and interstitial lung disease, were included. In the majority of cases, myositis was identified based on clinical presentation and laboratory findings (e.g., CPK elevation and, where available, myositis-specific autoantibodies) within the context of a molecular diagnosis of a viral (e.g., SARS-CoV-2) infection. In a small percentage of instances, muscle biopsies and/or muscle MRIs confirmed the diagnosis. Electron microscopy ruled out direct viral invasions as the pathophysiological cause of muscle harm in one instance. After muscle injury, inflammation, and compartment syndrome, severe limb ischemia was associated with COVID-19-induced hypercoagulation[6].

The present study aims to estimate the burden of inflammatory myopathies after COVID-19 depending on clinical and electrophysiological diagnosis; additionally, it aims to describe the characteristics of myositis related to COVID-19.

2. Materials and methods

2.1. Participants

In Basra, a multicentric, analytical, and cross-sectional survey was carried out to examine nerve conduction studies (NCS) and electromyographic (EMG) data from a COVID-19-affected patient[7]. Between July 1, 2021, and January 1, 2022, 2240 patients attended the neurology unit at Basrah Teaching Hospital, neurophysiology outpatient clinics at Al-Sadr Teaching Hospital, and Basrah Specialized Children's Hospital were analyzed.

The participants were separated into two groups. The first group (1344) consists of patients who had a confirmed COVID-19 infection in the previous year and were diagnosed using the European Center for Disease Prevention and Control (ECDC)'s criteria, including a positive polymerase chain reaction (PCR) and/or chest computed tomography (CT) demonstrating bilateral peripheral ground-glass opacities in the presence of strong clinical, serological, or epidemiological suspicion[8]. The second group of 896 individuals comprises those who have never been infected with COVID-19.

2.2. Method

A consultant neurologist did a comprehensive history taking with medical and neurological tests, and a full evaluation of the patient's history during the period of COVID-19 infection was undertaken. A specialized neurophysiologist conducted a thorough electrodiagnostic examination consisting of nerve conduction studies and needle electromyography to detect the presence or absence of the features suggestive of inflammatory myopathies, including normal motor nerve conduction studies in most cases (especially if it involved the proximal muscles only) and normal sensory nerve conduction studies in all of the cases. In the needle EMG, the motor unit action potentials (MUAPs) become short in duration, small in amplitude, and polyphasic with early recruitment of the MUAPs. Denervating features in the form of spontaneous activity (fibrillations and positive sharp waves) might be recorded in cases of inflammatory myopathies[9].

2.3. Inclusion criteria

The survey was carried out on all patients who attended the three above-mentioned centers during the 6-month period. The inclusion criteria for participant eligibility include:

(i) All patients aged over 18 years of both genders.
(ii) Whether or not they were diagnosed with COVID-19 infection in the past year. The COVID-19 diagnosis is based on the previous history of COVID-19 that was diagnosed according to the COVID-19 case definition from the criteria of the ECDC, which defines the following categories:
    - Confirmed case of COVID-19: The diagnosis of COVID-19 is made by either a positive PCR or a serological test (positive immunoglobulin "IgM" or "IgG" or viral antigen).

- Probable case of COVID-19: clinical suspicion based on the presence of fever, cough, and dyspnea with or without malaise, plus either a positive typical radiological finding on chest CT demonstrating peripheral bilateral ground-glass opacities or an epidemiological suspicion based on confirmed infection in relatives or family members in close contact with the patient.

(iii) All cases of myositis and DM that were diagnosed based on clinical suggestion and validated electrophysiologically by needle EMG.

2.4. Exclusion criteria

The data from the patients with the following criteria were excluded from the study:

(i) The childhood and adolescent age groups (below 18 years).

(ii) Those with no available nerve conduction study and EMG reports confirming myositis.

2.5. Statistical analysis

Clinical and electrophysiological parameters of the patients were recorded. The Statistical Package for the Social Sciences (SPSS) version 26 software from IBM Corporation was used to analyze the study's results. Before being analyzed using Fisher's exact test, the qualitative data was transformed into a percentage. The observed percentage from our research is compared to the overall population using a one-proportion Z-test. *P*-value of less than or equal to 0.05 is statistically significant, while *P*-value of less than or equal to 0.001 is highly statistically significant.

3. Results

3.1. The association between COVID-19 and inflammatory myopathies

The total number of patients who attended neurology and neurophysiology clinics in Basrah over a 6-month period equals 2240 (2344 had a history of COVID-19 and 896 had no history of COVID-19). The study found that the prevalence of myositis among the COVID-19 population was equal to 0.22%. Moreover, none of the non-COVID-19 population is documented to have myositis, but this is not statistically significant ($P > 0.05$) (Table 1).

3.2. Demographic characteristics of the patients

The demographic characteristics of the three patients are described in Table 2. One of the patients reported having a history of PM and was medicated on azathioprine and steroids before the onset of COVID-19.

3.3. The COVID-19 characteristics of the patients

The three patients were confirmed to have a history of COVID-19 by PCR and chest CT. The COVID-19 characteristics are described in Table 3. Two of the cases had a severe COVID-19 infection with cytokine storm, which was moderate in the other. None of the three cases had a history of COVID-19 or other recent vaccination or infection.

Table 1. The association between COVID-19 and inflammatory myopathies

Diagnosis	History of COVID-19	No history of COVID-19	Total	*P*-value*
Inflammatory myopathies	3 (0.22%)	0 (0.00%)	3	0.2797
No inflammatory myopathies	1341 (99.78%)	896 (100%)	2237	
Total	1344	896	2240	

*Fisher's exact test.

Table 2. Demographic characteristics of the patients

Demographic characteristics	Patient 1	Patient 2	Patient 3
Age/years	34	53	40
Gender	Female	Male	Male
Residency	Urban	Urban	Rural
Chronic illnesses	None	Hypertension	Polymyositis

Table 3. The COVID-19 characteristics of the patients

COVID-19 characteristics	Patient 1	Patient 2	Patient 3
The severity of COVID-19	Moderate	Severe	Severe
The occurrence of cytokine storm	None	Present	Present
The history of hospitalization	Not hospitalized	ICU >30 days	ICU for 1 week
Oxygen saturation	>93%	<70%	<70%
PCR test	Positive	Positive	Positive
Lung involvement by chest CT scan	15%	50%	55%
Serum ferritin (mcg/L)	670	1650	2100
C-reactive protein (mg/L)	21	66	95
Lactate dehydrogenase (U/L)	-	430	980
Interleukin-6 (pg/mL)	-	19	88
Neutrophil to lymphocyte ratio (N/L ratio)	3.1	3.7	4.9
History of COVID-19 vaccination	Not vaccinated	Not vaccinated	Not vaccinated

Abbreviations: PCR: Polymerase chain reaction; CT: Computed tomography; ICR: Intensive care unit.

3.4. The clinical and neurological characteristics of the patients

One of the cases had been diagnosed as DM with a negative initial paraneoplastic screen and positive anti-Jo-1 antibodies, anti-mitochondrial M2 antibodies, and anti-exosome antibodies (PM/SCL 100). In contrast, the two others have been diagnosed with PM, one of them had a negative autoimmune workup, and the other had only positive anti-nuclear antibodies (ANA) with no other available antibodies workup. The details of the neurological history and examination are clarified in Table 4.

3.5. The electrophysiological characteristics of the patients

The nerve conduction studies of the three patients were reported to be normal, apart from a mild axonal pattern of damage affecting the motor nerves of the third patient. Table 5 describes the EMG features of the three patients who were diagnosed with inflammatory myositis, which show myopathic MUAPs, especially in proximal muscles, including the deltoid and vastus medialis muscles, with polyphasia and early recruitment of MUAPs.

4. Discussion

In the post-COVID-19 period, the occurrence of PM is unusual, with very few instances reported[10]. In patients who develop nonspecific symptoms after recovering from COVID-19, it is essential to consider all possible causes, including PM. According to research, PM is an autoimmune disorder induced by a virus and/or an inflammatory condition, although the precise etiology is unclear[11].

In addition, since the beginning of the COVID-19 pandemic, the prevalence of DM has increased dramatically[12-14]. For instance, the rate of new cases of juvenile DM admitted to a tertiary care hospital in Iran between February 2020 and February 2021 was approximately four times that of the preceding decade[13]. Moreover, it was reported that DM patients suffered numerous relapses during the COVID-19 pandemic[15]. The disease is caused by a combination of genetic

Table 4. The clinical and neurological characteristics of the patients

Neurological characteristics	Patient 1	Patient 2	Patient 3
Diagnosis	Dermatomyositis	Polymyositis	Polymyositis
Creatinine kinase	6588 U/L	5170 U/L	7322 U/L
Autoimmune screen	Anti Jo-1 AMA M2 PM/Scl 100	Negative	ANA positive
Paraneoplastic screen	Negative	Negative	Negative
Time of onset of neurological complaints in relation to COVID-19	1 week	3 weeks	5 days
Limb weakness	Proximal upper and lower limbs	Proximal upper and lower limbs	Proximal and distal upper and lower limbs
Sensory features	None	None	None
Sphincter dysfunction	None	None	None
Dysphagia/Dysarthria	None	None	Present
Dyspnea	Mild	Severe	Severe
Skin lesion	Present	None	None
Facial asymmetry	None	None	None
Muscle wasting	None	None	Present
Muscle fasciculation	None	None	None
Tone	Normal	Decrease	Decrease
Power *	Grade 4	Grade 4	Grade 0
Reflexes	Present	Present	Decreased
Coordination	Normal	Normal	Not assessed
Sensory exam	Normal	Normal	Normal

Abbreviations: MRC: Medical research council; AMA: Anti-mitochondrial M2 antibodies; PM/Scl 100: Anti-exosome antibodies. *Power assessment was depend on MRC grading which is a universal grading system for power.

Table 5. The needle EMG parameters of the patients

Patient 1 (Dermatomyositis)				
Findings	**Deltoid**	**FDI**	**VM**	**TA**
Spontaneous activity	Fibrillations/positive sharp waves	Not detected	Fibrillations	Fibrillations
MUAP duration	Short	Normal	Short	Normal
MUAP amplitude	Small	Normal	Small	Normal
Recruitment	Early	Normal	Early	Normal
Number of phases	Polyphasic	Occasional Polyphasic	Occasional polyphasic	Normal
Patient 2 (Polymyositis)				
Findings	**Deltoid**	**FDI**	**VM**	**TA**
Spontaneous activity	Not detected	Not detected	Fibrillations/positive sharp waves	Not detected
MUAP duration	Short	Normal	Short	Normal
MUAP amplitude	Small	Normal	Small	Normal
Recruitment	Early	Normal	Early	Normal
Number of phases	Polyphasic	Normal	Polyphasic	Normal
Patient 3 (Polymyositis)				
Findings	**Deltoid**	**FDI**	**VM**	**TA**
Spontaneous activity	Fibrillations	Not detected	Not detected	Not detected
MUAP duration	Short	Normal	Short	Normal
MUAP amplitude	Normal	Normal	Small	Normal
Recruitment	Early	Normal	Early	Normal
Number of phases	Occasional Polyphasic	Occasional Polyphasic	Polyphasic	Normal

Abbreviations: MUAP: Motor unit action potential; DL: Deltoid; FDI: First dorsal interosseous; VM: Vastus medialis; TA: Tibialis anterior.

predisposition and environmental factors that generate an immunological disruption that is poorly recognized[16]. Whether complement fixation or antibody-dependent activation of the interferon pathway initiates that the inflammatory cascade is still a matter of debate[17].

In the initial phase of this study, our main concern was to detect the GBS cases related to COVID-19, estimate its prevalence, and describe its clinical and electrophysiological characteristics. During the GBS case collection period, however, we discovered three cases of inflammatory myopathies among the COVID-19 patients: two out of three cases, one was a middle-aged man who was diagnosed clinically and electrophysiologically with PM after a severe COVID-19 infection, and the other was a young lady with DM who was also suspected clinically and confirmed electrophysiologically after a moderate COVID-19 infection. A thorough survey was done for those two cases to find any association with trigger factors, autoimmune disease, or paraneoplastic syndrome but failed to yield any conclusion; hence, the post-COVID-19 inflammatory myositis was suggested. On the other hand, the present study reports only one case of PM in the COVID-19 population, which was already diagnosed before the onset of COVID-19 infection, and the patient died after a severe COVID-19 infection thereafter, which triggered a relapse and led to respiratory failure and death. It is worth mentioning that the occurrence of myositis in the current sample is 44 times higher than the prevalence of myositis worldwide, which is equal to 0.005%[18] ($P < 0.001$, 95% confidence interval = 0.1979 – 0.2421).

Ten further instances of PM and DM after COVID-19 infection had been recorded; at the time, this article was written. The United States reported four of these instances, whereas Europe and Asia recorded four and two, respectively[17,19]. Table 6 outlines the clinical characteristics of these ten instances.

The correlation between viral infections and their ability to trigger autoimmune disease is well-established in the medical literature; nevertheless, the precise mechanism behind this association is likely complicated and unknown. Several investigations have shown the existence of autoantibodies in COVID-19 patients. Furthermore, the COVID-19 genome includes 28 human proteins with similar sections to COVID-19 peptides, which may accelerate the formation of autoantibodies[20]. Inflammation produced by COVID-19 is an additional potential autoimmunogenic mechanism. COVID-19 is particularly dangerous, not due to the virus itself but due to the inflammatory response

Table 6. Characteristics of myositis cases related to COVID-19

Item	Description
Diagnosis	Six confirmed cases of polymyositis and four cases of dermatomycosis
Gender	Six were women, and four were men
Age	The minimum and maximum ages were 23 and 77, respectively, with a mean age of 55.6 years
COVID-19 history	Nine patients had positive PCR and one patient had a positive serological test
COVID-19 severity	Critical in one case, severe in four cases, moderate in one case, mild in two cases, and in two cases, the severities were not reported
Relapse	Three cases relapsed after COVID-19 and seven cases got a new diagnosis
Clinical features	All patients presented with lower and upper limb proximal muscle weakness; three reported myalgia; two reported dysphagia; three reported a typical rash; one reported respiratory muscle weakness; and one had cardiac involvement
Immunological tests	Anti-nuclear antibodies were positive in three cases, autoantibodies were negative in five cases. Anti-MI 2b was positive in one patient; anti-Ro/SSA antibodies were positive in one patient; and an anti-Smith antibody was positive in one patient
CPK	Markedly elevated in nine cases, and three reported mild elevation
EMG	Two patients had myopathic potential, one was normal, and for the other seven patients, an EMG was not done
Outcome	One patient died, and the other 11 improved on either steroids alone (4 patients) or a combination of steroids and immunomodulators

Abbreviations: CPK: Creatine phosphokinase; EMG: Electromyography; PCR: Polymerase chain reaction.

it triggers. Early tissue damage caused by COVID-19 increases the production of proinflammatory cytokines, which attract more inflammatory cells and increase inflammatory reactants[21]. Repeat and amplification of this process result in what is often referred to as a "cytokine storm" or a systemic inflammatory response described as macrophage activation syndrome or secondary hemophagocytic lymphohistiocytosis[22]. COVID-19's potential to predispose the body to immunological hyperactivity allows us to predict the development of post-COVID-related autoimmune disorders[10].

The causal link between COVID-19 and symptoms of nervous system problems has been determined purely based on their co-occurrence in time. Two patterns have been documented: (a) neurological complications occurring together with COVID-19 symptoms and suggesting a direct viral mechanism ("parainfectious" hypothesis), such as neuroinvasion; (b) neurological complications developing after the initial infectious symptoms and supporting indirect mechanisms ("postinfectious" hypothesis), likely immune-mediated[6].

Based on the *in vivo* and *in vitro* neuroinvasive capacities of SARS-CoV and MERS-CoV, with which the etiological agent of COVID-19 (i.e., SARS-CoV-2) shares 79.5% and 50% gene homology, respectively, the capacity of SARS-CoV-2 to invade the nervous system has been hypothesized[23]. Given the early onset of anosmia and ageusia, one idea is that olfactory, trigeminal, or gustative terminals may serve as entry points for the virus, which may then propagate to the central nervous system (CNS) through retrograde axonal transport and trans-synaptic transfer[24].

Lower cranial nerves might be additional entry routes, resulting in early involvement of the lower brain stem and perhaps explaining some specific characteristics of COVID-19, such as hypoxia out of proportion to dyspnea and the frequency of syncope[25]. Alternative methods of neuroinvasion applicable to both the CNS and PNS include entrance by circulating immune cells, infection of the vascular endothelium, and passing the blood-brain barrier or blood-nerve barrier[6].

The "postinfectious" immune-mediated hypothesis is supported by the fact that COVID-19 induces a proinflammatory state due to the production of several cytokines, including IL1, IL6, and TNF, in addition to the subsequent activation of immune cells[24].

Furthermore, the gene-level biology of this phenomenon is still poorly understood[26], despite the extensive study conducted in the SARS-CoV-2 area. In addition, the viral infection of endothelial cells leads to the breakdown of the blood–brain barrier, which induces acute inflammation and causes neuronal injury and disruption of neurogenesis. In addition, these proinflammatory cytokines may influence vascular remodeling, resulting in the loss of vascular wall integrity, which may lead to intraparenchymal bleeding. Finally, cytokine storming is an established risk factor for coagulopathy, which may trigger a stroke[27,28].

5. Conclusion

COVID-19 could present with a wide variety of neurological complications, including PM and DM, and the prevalence of these disorders increased noticeably after the pandemic. It is important to highlight such disorders and increase awareness about them as probable sequelae of COVID-19 in order not to misdiagnose them, which might lead to serious complications, including respiratory failure and death.

Acknowledgments

The authors thank Dr. Lana A. Kadhum for her active efforts in the data collection and sample recruitment.

Funding

There was no funding from any society or company; the researchers funded their own research.

Conflict of interest

The authors declare that they have no competing interests.

Author contributions

Conceptualization: Nareen Haikaz Hasrat, Hassan Ala Farid

Investigation: Nareen Haikaz Hasrat, Zaineb Adil Yakob

Supervision: Ali Raheem Hashim, Haithem Jawad Kadhum

Writing – original draft: Nareen Haikaz Hasrat, Hassan Ala Farid

Writing – review & editing: Ali Raheem Hashim, Haithem Jawad Kadhum, Zaineb Adil Yakob.

Ethics approval and consent to participate

The Basrah Health Directorate granted ethical approval (No. 911) on December 13, 2021, and the Basrah University College of Medicine's ethics committee granted approval (No. 7/9/6292) on December 12, 2021. Patients who participated in this study provided written informed consent.

Consent for publication

Written informed consent was obtained from the patients for publication.

Availability of data

The datasets used and/or analyzed during the present study are available from the corresponding author on reasonable request.

References

1. Hashim AR, Kadhum HJ, Farid HA, 2022, The pattern of Covid 19 among hospitalized athletics patient: A comparative study from Basrah city, Southern Iraq. *J Res Med Dent Sci*, 10: 36–40.
2. Farid HA, Hashim AR, Hasrat NH, *et al*., 2021, The neurological manifestations of COVID-19 and their relationship with the disease severity in hospitalized patients: A single-centered, cross-sectional study on patients with COVID-19 from Basra, Iraq. *Multicult Educ*, 7: 391.

   https://doi.org/10.5281/zenodo.4730889
3. Hasrat NH, Kadhum HJ, Hashim AR, *et al*., 2022, Neurographic evidence of inflammatory polyneuropathies in Peri-COVID-19 circumstances and their relationship with acute disease severity and inflammatory storm. *Cureus*, 14: e23517.

   https://doi.org/10.7759/cureus.23517
4. Lundberg IE, Miller FW, Tjärnlund A, *et al*., 2016, Diagnosis and classification of idiopathic inflammatory myopathies. *J Intern Med*, 280: 39–51.

   https://doi.org/10.1111/joim.12524
5. Romero-SánchezCM,Díaz-MarotoI,Fernández-DíazE,*et al*., 2020. Neurologic manifestations in hospitalized patients with COVID-19: The ALBACOVID registry. *Neurology*, 95: e1060–e1070.

   https://doi.org/10.1212/WNL.0000000000009937
6. Taga A, Lauria G, 2022, COVID-19 and the peripheral nervous system. A 2-year review from the pandemic to the vaccine era. *J Peripher Nerv Syst*, 27: 4–30.

   https://doi.org/10.1111/jns.12482
7. Hasrat NH, Kadhum HJ, Hashim AR, *et al*., 2022, Neurophysiological changes by nerve conduction study and electromyography in acute and long-term COVID-19 circumstances: A comparative study protocol and review. *J Xian Shiyou Univ*, 18: 241–249.
8. Case Definition for Coronavirus Disease, 2019, (COVID-19), as of 3 December 2020. European Centre for Disease Prevention and Control. Available from: https://www.ecdc.europa.eu/en/covid-19/surveillance/case-definition [Last accessed on 2022 Jun 12].
9. Preston D, Shapiro B, 2020, Electromyography and Neuromuscular Disorders: Clinical-Electrophysiologic Correlations. 4th ed. Amsterdam: Elsevier Saunders. Available from: https://www.elsevier.com/books/electromyography-and-neuromuscular-disorders/978-0-323-66180-5 [Last accessed on 2022 Dec 10].
10. Anthony S, Phrathep DD, El-Husari A, *et al*., 2022, Post-COVID-19 polymyositis: A case report. *Cureus*, 14: e30991. https://10.7759/cureus.30991
11. Hunter K, Lyon MG, 2012, Evaluation and management of polymyositis. *Indian J Dermatol*, 57: 371–374.

    https://doi.org/10.4103/0019-5154.100479
12. Gokhale Y, Patankar A, Holla U, *et al*., 2020, Dermatomyositis during COVID-19 pandemic (A case series): Is there a cause effect relationship? *J Assoc Physicians India*, 68: 20–24.
13. Movahedi N, Ziaee V, 2021, COVID-19 and myositis; true dermatomyositis or prolonged post viral myositis? *Pediatr Rheumatol Online J*, 19: 86.

    https://doi.org/10.1186/s12969-021-00570-w
14. Saud A, Naveen R, Aggarwal R, *et al*., 2021, COVID-19 and myositis: What we know so far. *Curr Rheumatol Rep*, 23: 63.

https://doi.org/10.1007/s11926-021-01023-9

15. Naveen R, Sundaram TG, Agarwal V, *et al*., 2021, Teleconsultation experience with the idiopathic inflammatory myopathies: A prospective observational cohort study during the COVID-19 pandemic. *Rheumatol Int*, 41: 67–76.

    https://doi.org/10.1007/s00296-020-04737-8

16. Venalis P, Lundberg IE, 2014, Immune mechanisms in polymyositis and dermatomyositis and potential targets for therapy. *Rheumatology* (*Oxford*), 53: 397–405.

    https://doi.org/10.1093/rheumatology/ket279

17. Albakri AM, Subki AH, Albeity A, *et al*., 2022, Dermatomyositis flare after a COVID-19 infection successfully treated with rituximab: A case report and literature review. *J Inflamm Res*, 15: 6047–6053.

    https://doi.org/10.2147/JIR.S369477

18. Cheeti A, Brent LH, Panginikkod S, 2022, Autoimmune myopathies. In: StatPearls. Treasure Island, FL: StatPearls Publishing.

19. Amin S, Rahim F, Noor M, *et al*., 2022, Polymyositis: The comet tail after COVID-19. *Cureus*, 14: e26453.

    https://doi.org/10.7759/cureus.26453

20. Gracia-Ramos AE, Martin-Nares E, Hernández-Molina G, 2021 New onset of autoimmune diseases following COVID-19 diagnosis. *Cells*, 10: 3592.

    https://doi.org/10.3390/cells10123592

21. Ochani R, Asad A, Yasmin F, *et al*., 2021, COVID-19 pandemic: From origins to outcomes. A comprehensive review of viral pathogenesis, clinical manifestations, diagnostic evaluation, and management. *Infez Med*, 29: 20–36.

22. Anka AU, Tahir MI, Abubakar SD, *et al*., 2021, Coronavirus disease 2019 (COVID-19): An overview of the immunopathology, serological diagnosis and management. *Scand J Immunol*, 93: e12998.

    https://doi.org/10.1111/sji.12998

23. Li YC, Bai WZ, Hashikawa T, 2020, The neuroinvasive potential of SARS-CoV2 may play a role in the respiratory failure of COVID-19 patients. *J Med Virol*, 92: 552–555.

    https://doi.org/10.1002/jmv.25728

24. Zubair AS, McAlpine LS, Gardin T, *et al*., 2020, Neuropathogenesis and neurologic manifestations of the coronaviruses in the age of coronavirus disease 2019: A review. *JAMA Neurol*, 77: 1018–1027.

    https://doi.org/10.1001%2Fjamaneurol.2020.2065

25. Canetta C, Accordino S, Buscarini E, *et al*., 2020, Syncope at SARS-CoV-2 onset. *Auton Neurosci*, 229: 102734.

    https://doi.org/10.1016%2Fj.autneu.2020.102734

26. Batchu S, Diaz MJ, Tran JT, *et al*., 2023, Spatial mapping of genes implicated in SARS-CoV-2 neuroinvasion to dorsolateral prefrontal cortex gray matter. *COVID*, 3: 82–89.

    https://doi.org/10.3390/covid3010005

27. Sizemore G, Lucke-Wold B, Small C, 2022, Review of SARS-COV-2 systemic impact: Building the case for sepsis via virus in the circulatory system. *SM J Neurol Disord Stroke*, 6: 7.

28. Small C, Mehkri Y, Panther E, *et al*., 2020, Coronavirus disease-2019 and stroke: Pathophysiology and management. *Can J Neurol Sci*, 28: 1–8.

    https://doi.org/10.1017/cjn.2022.267