The impact of SARS-CoV-2 on neuromuscular disorders

Meng-Ju Wu[1], Yuan-Ting Sun[1,2]

Abstract

The emergence of SARS-CoV-2 has profoundly impacted global society and various aspects of human life. While the pandemic has resulted in disruptions and challenges, it has also accelerated scientific research on viruses and immunology, leading to remarkable progress in vaccine technology and immunization strategies. This review examines the impact of SARS-CoV-2 on pre-existing neuromuscular disorders, and neuromuscular events following SARS-CoV-2 infection, including immune-mediated and critical illness status-related disorders. Furthermore, the review discusses the relationship between SARS-CoV-2 vaccination and neuromuscular complications. The findings highlight the need for further research and understanding to improve patient outcomes.

Keywords: SARS-CoV-2, neuromuscular diseases, vaccine.

Acta Neurol Taiwan 2023;32:88-99

The emergence of SARS-CoV-2 over the past three years has undeniably reshaped the global landscape and profoundly affected various aspects of human life. The consequential measures, such as social distancing and restriction lockdown, have disrupted daily routines and prompted a significant societal shift. Nevertheless, amidst these challenging circumstances, the pandemic accelerates scientific research on viruses and immunology. Moreover, the urgency to develop effective vaccines against SARS-CoV-2 has propelled remarkable progress in vaccine technology and immunization strategies. The unexpected benefits arising from the global ordeal with SARS-CoV-2 should not be overlooked. This review focused on the impact of SARS-CoV-2 and vaccines against SARS-CoV-2 on neuromuscular disorders.

SARS-CoV-2 and neuromuscular disorders

The temporal relationship between neuromuscular disorders and SARS-CoV-2 infection can be described in three patterns. Firstly, in a pre-existing neuromuscular disorder, SARS-CoV-2 infection may trigger an exacerbation during the course[1,2]. Secondly, a monophasic neuromuscular disorder can appear in a naïve individual, such as Guillain Barré syndrome (GBS), immune-mediated myositis, critical illness polyneuropathy (CIP), or critical illness myopathy (CIM). Thirdly, a new neuromuscular disorder appears in a susceptible individual or a subclinical disorder has accelerated to a clinical status.

From the [1]Department of Neurology, National Cheng Kung University Hospital, College of Medicine, National Cheng Kung University, Tainan 704, Taiwan; [2]Department of Medical Genomics, National Cheng Kung University Hospital, College of Medicine, National Cheng Kung University, Tainan 704, Taiwan.
Received June 17, 2023. Accepted June 19, 2023.

Correspondence to: Yuan-Ting Sun, Department of Neurology, College of Medicine, National Cheng Kung University, 1 University Road, Tainan 701, Taiwan.
E-mail: ytsun@mail.ncku.edu.tw

The impact of SARS-CoV-2 in patients with existing neuromuscular disorders

Patients with neuromuscular disorders comprised a group of rare diseases, most often of genetic or autoimmune origin. They are considered at a high risk of developing severe complications with SARS-CoV-2 as they can be under immune therapy or subject to cardiac or respiratory insufficiency. In the early pandemic, French Health Care Network for Rare Neuromuscular Diseases published a prevalence of symptomatic SARS-CoV-2 estimated at 0.0017[2]. The hospitalization rate in medical and intensive care units (ICU) was 0.27 and 0.14. SARS-CoV-2 caused worsening symptoms and death in 31% and 11% of this group of patients. Among the 58% of infected patients who received home care, SARS-CoV-2 showed no impact on their neuromuscular disorders. The risk factors for worse outcomes in SARS-CoV-2 infection included comorbid hypertension, diabetes, and severe disease status before the infection, which were identical to that of general populations[3,4].

To particularly consider the effects of immune therapy, myasthenia gravis (MG) might be at a greater risk of worse outcomes than otherwise healthy people because of an immunocompromised state related to immunotherapy and possible respiratory and bulbar muscular weakness. However, discontinuing immunotherapy increases the risk of relapse as well. Although the SARS-CoV-2 infection rate in MG patients was equivalent to that of the general population[5], infection is a well-known trigger of symptom exacerbation for MG. The need for data to answer the critical question was quickly recognized, and a registry was created to capture high-quality information on SARS-CoV-2 Associated Risks and Effects in Myasthenia Gravis (CARE-MG). The preliminary data by December 2020 showed that worsening MG requiring rescue therapy (e.g., intravenous immunoglobulin, plasma exchange, or steroids) in the setting of SARS-CoV-2 was reported in 40% of patients. Complete recovery or discharge to home was reported in 43% of patients, whereas 24% of patients died due to SARS-CoV-2[6]. Subsequent retrospective studies and systematic reviews suggested that SARS-CoV-2-infected MG patients were frequently hospitalized and had a higher mortality rate than the general population with SARS-CoV-2[7-11]. In addition, patients with a pre-existing diagnosis of MG who were hospitalized for SARS-CoV-2 infection had severe disease courses[12]. However, there are systematic reviews that disapprove this notion[13].

Monophasic neuromuscular event after SARS-CoV-2 infection

The neuromuscular manifestations after SARS-CoV-2 infection are commonly mentioned in patients without neuromuscular disorders[14]. In a prospective multicenter observational study that included 4491 hospitalized severe SARS-CoV-2 infected patients, the prevalence of neuropathy and myopathy is around 0.8% and 0.5%. Many of the cases of neuropathy and myopathy were attributed to critical illness. Furthermore, there was a significant association between the severity of illness markers (intubation, SOFA scores, acute renal failure) and the occurrence of neurologic disorders, suggesting that critical illness itself may have contributed to neurologic complications[14].

Neuromuscular events not attributed to the critical illness status also can be found in this pandemic, such as newly developed MG provoked by SARS-CoV-2 infection[15-17], GBS after infection[17-24], and myositis concurrently or subsequently to the SARS-CoV-2 infection[25-28].

Immune-mediated monophasic event: focusing on myopathy

Muscular symptoms are commonly seen in viral infections, such as influenza, coxsackie virus B, Epstein-Barr virus (EBV), and coronal virus[29,30]. In several pandemic coronavirus events, including SARS-CoV-1, Middle East respiratory syndrome coronavirus (MERS), and the recent SARS-CoV-2 pandemic, various proportions of patients (ranging from 30%-70%) reported muscular symptoms, including myalgia and weakness[31-35].

In 2003, there was an outbreak of SARS-CoV-1 in Hong Kong and Taiwan[36]. The report from Hong Kong showed that creatine kinase (CK) levels and lactate dehydrogenase (LDH) were elevated in 32.1% and 71% of patients, respectively. None of the patients with elevated CK levels had abnormal values for CK-MB or troponin T, indicating that the source of CK was unlikely to be cardiac muscle[36]. A postmortem case series showed focal myofiber necrosis with macrophage infiltration.

Regenerative fibers were scanty. In situ hybridization, viral cultures, and examination for viral particles under electron microscopy (EM) revealed negative results[37]. The authors suggested that focal myofiber necrosis is possibly immune-mediated. However, because of the lack of staining for major histocompatibility complex-1 (MHC-I) or myxovirus resistance protein A (MxA), and the small case numbers of muscle pathology, the report could not conclude pathomechanism of SARS-related myopathy. Critical illness myopathy and superimposed steroid myopathy may also play an important role in SARS-CoV-1.

MERS outbroke ten years ago. The muscle pathology in the postmortem study showed atrophic and myopathic myofibre changes, splitting of the muscle fibers, and lymphohistiocytic inflammatory infiltration consisting of CD68+ macrophages, CD4+, and CD8+ lymphocytes. The numbers of inflammatory cells were more abundant in atrophic myofibres. Ultrastructural EM study identified MERS-CoV particles in macrophages infiltrating the muscle but not in the muscle fibers[38].

In this SARS-CoV-2 pandemic, a cross-sectional study analyzed 73435 infected patients and found an increased hazard ratio and excess burden in muscle weakness[39]. Early studies suggested that roughly 30% - 60% of infected patients experienced myalgia[40-43], and the combination of myalgia and increased levels of CK was more pronounced in critically ill patients demanding intensive care support than in mildly affected individuals [44-46] and was associated with higher mortality rates[46,47].

The possibility that the virus invades skeletal muscle has been considered because muscle expresses angiotensin-converting enzyme 2, a cell surface receptor utilized by SARS-CoV-1 and SARS-CoV-2 for host cell entry[48]. In one study that examined the diaphragm muscle obtained from 26 consecutive autopsies of critically ill COVID-19–infected patients who died, SARS-CoV-2 RNA was found in the muscle in 4 cases (15.4%)[49]. At the same time, several case reports included biopsies that advocated a dermatomyositis-like phenotype[27,50-52]. A negative SARS-CoV-2 IHC staining in muscle was reported in two independent postmortem studies which argued against this hypothesis[41,53]. In these two histopathology studies, patients with myositis had CD68+, CD4+, and/or CD8+ histiocytes and T cells in the muscles. Upregulated MHC-1 in the early phase of the disease of non-necrotic/non-regenerating muscle fibers was evident in all patients with myositis or necrotizing myopathy. Concomitant upregulation of MHC-II on myofibers in later stages indicated the involvement of skeletal muscle in the immune response against SARS-CoV-2. Type 2 fiber atrophy was observed in all patients. Compared with cardiac muscle, inflammatory changes were more pronounced in skeletal muscles than in cardiac muscles. Abnormal MxA immunostaining was presented in the capillaries, indicating a type I interferon signature, rather than sarcolemma. In all cases, IHC stainings for SARS-CoV-2 nucleocapsid and antibodies against SARS-CoV-2 spike protein were negative. No viral particles were found by EM.

Critical illness status-related monophasic event

Patients with SARS-CoV-2 infection who develop severe respiratory failure, need ventilation assistance, experience prolonged ICU stay, and suffer from long-term immobility tend to develop CIP or CIM[42,54,55]. According to the PANDEMIC registry in Germany, 20% of SARS-COV-2-positive ICU patients developed neuromuscular disorders[56]. CIP/CIM accounted for 86.25% of all neuromuscular disorders[56]. Although the mortality rate was 50% of intubated patients with CIP/CIM[43], CIP/CIM had the lowest influence on mortality (OR: 0.21, 95% CI 0.08-0.58) compared to acute ischemic stroke (OR: 3.89, 95% CI 1.85-8.17)[56].

The development of CIP/CIM in patients with SARS-CoV-2 is multifactorial[44-46]. The characteristic finding in CIM is the loss of myosin, the motor protein responsible for initiating muscle contraction[57,58]. Myosin is especially vulnerable to high-dose corticosteroids. Contrary to CIM, decreased levels of local axonal survival factors, increased vascular permeability, and voltage-gated sodium channel dysfunction[59] can finally result in CIP[60]. Although evidence of direct SARS-CoV-2 invasion to the skeletal muscle is inconclusive[49,51,61,62], an indirect inflammatory process combined with patients' comorbidities, high-dose steroids, and use of neuromuscular blocking agents appears to be a more plausible explanation[63].

Another study in Italy included 44 patients diagnosed with acute respiratory distress syndrome (ARDS) (23 patients were SARS-CoV-2 positive)[64]. The mortality

rate was 33% in the entire CIP/CIM cohort, regardless of the SARS-CoV-2 status[64]. CIP/CIM was diagnosed in 65% of SARS-CoV-2-positive patients and 71% of SARS-CoV-2-negative patients[64]. The comorbidities, CK levels, LDH levels, length of ICU stay, and the outcome at ICU discharge between the groups with or without SARS-CoV-2 were not statistically different[64]. Therefore, early recognition of CIP/CIM followed by an individualized management strategy is essential to improve ICU discharge outcomes[65-67].

Newly developed chronic neuromuscular disorders after SARS-CoV-2 infection

Jacob et al. summarized the influence of clinically major viruses on different kinds of neuromuscular disorders. In the acute setting, most of the virus families are found to be associated with GBS[68]. In the chronic status, SARS-CoV-2 and influenza viruses are closely linked to myopathic conditions. SARS-CoV-2 virus, Zika virus, and West Nile virus also share similar associations with MG[68]. For the rest of the viruses, chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) is associated with EBV and Hepatitis B virus. Motor neuron disorders are related to the human immunodeficiency virus, human T-lymphotropic virus, enteroviruses, and Coxsackievirus B3 virus[68]. In line with the hypothesis of autoimmunity secondary to viral infection, newly developed chronic neuromuscular disorders will become an important issue for neuromuscular specialists.

SARS-CoV-2 Vaccinations and neuromuscular disorders

Thanks to the emergence of SARS-CoV-2, vaccines have been developed rapidly, leading to a swift accumulation of information regarding neuromuscular complications associated with vaccination. The most common neuromuscular disorders, GBS, brachial neuritis (BN), and MG, are summarized below.

Guillain-Barré syndrome

GBS is one of the most common neurological complications following SARS-CoV-2 vaccines. However, the incidence of SARS-CoV-2 vaccine-induced GBS is smaller than GBS in general (0.81 to 1.91 cases per 100,000 person-years)[69-72]. In addition, SARS-CoV-2 vaccine-induced GBS is less frequent than SARS-CoV-2 infection-induced GBS[73-76]. Compared with influenza vaccines, SARS-CoV-2 vaccines appeared to have fewer cases of neurological complications, including GBS[77]. A retrospective study in the United States demonstrated that the GBS reporting rate was 3.29 per one million doses in Ad26.COV.2.S (Janssen), 0.29 in BNT162b2 (Pfizer-BioNTech), and 0.35 in mRNA-1273 (Moderna)[78]. In Mexico, the overall incidence was 1.19 per one million doses, in which the incidence was 3.86 per one million doses in Ad26.COV2-S and 1.92 in BNT162b2[79]. In the United Kingdom, the prospective surveillance study showed that 67 out of 70 patients with vaccine-induced GBS had received ChAdOx1 (Oxford-AstraZeneca), while the remaining three patients had received BNT162b2[80]. A case of ChAdOx1-induced GBS was also reported in a Taiwanese study[81]. In South Korea, the prospective study showed overall incidence was 1.42 per million doses, in which the incidence of GBS was 4.49 per million doses from vector-based vaccines and 0.80 from mRNA-based vaccines[82]. Based on the aforementioned studies, mRNA-based vaccines were least likely to induce GBS. Recurrent GBS due to vaccine is rare, and the exact incidence related to the SARS-CoV-2 vaccine remains uncertain[83-86].

Among the SARS-CoV-2 vaccination-related GBS, sensorimotor and pure motor variants were the most common clinical presentations, followed by cranial nerve involvement, paraparesis, and Miller-Fisher syndrome[78-80,82,87-89]. Demyelination was the predominant electrodiagnostic finding[79,80,82,90,91]. The presence of antiganglioside antibodies was extremely rare[74,80,82]. About 13% to 30% of vaccine-induced GBS required mechanical ventilation[79,82,92], and the mortality rate ranged from 2% to 9%[80,82,91,93]. Most patients responded to the conventional treatment, but the total recovery rate was generally low[79,80,92,94,95].

Brachial neuritis

The clinical presentation of BN, also known as neuralgic amyotrophy or Parsonage-Turner syndrome, is the sudden onset of severe unilateral pain over the shoulder girdle followed by weakness and muscle atrophy in the following weeks[96]. Hypoesthesia or paresthesia is

usually present over the involved nerves[96]. Several cases of BN have been reported after receiving vaccinations against SARS-CoV-2[97-115]. BN was more likely to develop in patients who received mRNA-based vaccines than adenovirus vector-based vaccines, and the risk was similar to those who received the influenza vaccine[97,98,116]. The time from the vaccination to symptom onset varies greatly, but two weeks on average, regardless of the vaccine type[97,98,100,103,104]. BN mostly occurs at the location where patients received the injection[97,102]. Some studies observed that patients with BN may have enhancement of brachial plexus and/or cervical lymphadenopathy on the Magnetic Resonance Imaging study[97,110,112-114,116]. Prior study revealed that complete remission of BN was roughly 60% in one year and up to 90% in three years[117,118]. Twenty-five percent of SARS-CoV-2 vaccine-induced BN had remission within two months[97].

Myasthenia gravis

Pre-existing MG with SARS-CoV-2 vaccines

Vaccinated MG patients were less likely to have COVID-19 infection (hazard ratio, 0.43; 95% CI 0.30 – 0.60)[119]. Overall, SARS-CoV-2 vaccines are safe for MG patients[120]. The exacerbations of MG due to vaccination were reported. In a cohort of 113 MG patients who received three doses of the BioNTech SARS-CoV-2 vaccine in Italy, only 8 (7.1%) had an exacerbation[121]. In another cohort of 160 MG patients in Israel, 150 MG patients had received BNT162b2 mRNA vaccines, and 8 (5.3%) had confirmed exacerbation after vaccination[122]. Among them, the exacerbation occurred mostly after the second dose (53.8%), and the time from vaccination to symptom onset varied greatly (0.5 to 30 days)[122]. In a cohort with 343 MG patients in Japan, only 3 (0.87%) patients reported exacerbation[123].

MG-related immunotherapies and SARS-CoV-2 vaccines

Immunosuppressive therapies may interfere with the effect of SARS-CoV-2 vaccines, especially for cell-depleting agents. Spike-IgG positivity was seen in 88.77% of patients after vaccination[124]. However, patients receiving combined therapy had a lower seroconversion ratio and T-cell responses[124-127]. In two case reports when one patient receiving mycophenolate and prednisolone and the other one receiving mycophenolate, eculizumab, and prednisolone, both patients had undetectable SARS-CoV-2 antibody levels after two doses of vaccines[128,129]. Temporarily discontinuing mycophenolate or eculizumab before the next dose increased the efficacy of vaccines[128-130]. In a small prospective study, Damato et al. observed that spike-IgG seroconversion only occurred in one of three patients with rituximab therapy after SARS-CoV-2 vaccination[131].

New onset MG and SARS-CoV-2 vaccines

Case reports on the new onset MG associated with SARS-CoV-2 vaccination have increased since 2020[132-146]. In these reports, 83% of the patients were male, and 58% were > 60 years old at onset (ranging from 28 to 91 years old). The median time of symptom onset was six days (ranging from 1 to 28 days). Among all vaccine types, 50% received BNT162b2, 29% with AstraZeneca, 17% with mRNA1273, and 4% with Covishield vaccines. Most patients (54%) had initial symptoms after the first dose of the vaccine. The distribution of ocular type MG and generalized type MG was both 42%, followed by oculo-bulbar type (16%). These findings were comparable to a literature review conducted by Virgilio et al[136]. The exact causality between SARS-CoV-2 vaccination and MG is still under debate[147]. A plausible explanation may be the combination of molecular mimicry between the acetylcholine receptor and vaccine antigen and the bystander effect from T-cell activation[146]. Although these patients were responsive to standard treatment, whether this new onset MG follows similar long-term clinical courses will be an interesting topic in the post-COVID-19 era.

CONCLUSION

In individuals with neuromuscular disorders, the infection can trigger exacerbation in pre-existing conditions, particularly in patients with MG who are immunocompromised related to immunotherapy and possible respiratory and bulbar muscular weakness. The outcome severity depends on comorbidities and disease status before the infection. The SARS-CoV-2 infection also leads to immune-mediated neuromuscular events such as GBS and myositis. SARS-CoV-2-related myositis is currently thought of as a type I interferon-mediated

myopathy. The incidence of CIP/CIM in SARS-CoV-2-positive patients who had ARDS was similar to patients with ARDS secondary to other causes. Moreover, SARS-CoV-2 may cause the development of new neuromuscular disorders in susceptible individuals which warrants more research on the subsequent clinical course.

Regarding the association between SARS-CoV-2 vaccinations and neuromuscular complications, the incidence of vaccine-induced GBS is lower than SARS-CoV-2-related GBS. The incidence of SARS-CoV-2 vaccine-induced BN is also lower than BN indued by influenza vaccine. Overall, this review underscores the importance of ongoing scientific investigation and clinical management to optimize outcomes for patients with neuromuscular disorders in the context of SARS-CoV-2.

REFERENCE

1. Manji H, Carr AS, Brownlee WJ, Lunn MP. Neurology in the time of COVID-19. J Neurol Neurosurg Psychiatry. Jun 2020;91(6):568-570.
2. Pisella LI, Fernandes S, Sole G, et al. A multicenter cross-sectional French study of the impact of COVID-19 on neuromuscular diseases. Orphanet journal of rare diseases. Oct 26 2021;16(1):450.
3. Ben Fredj S, Ghammem R, Zammit N, et al. Risk factors for severe Covid-19 breakthrough infections: an observational longitudinal study. BMC infectious diseases. Nov 28 2022;22(1).
4. Gao YD, Ding M, Dong X, et al. Risk factors for severe and critically ill COVID-19 patients: A review. Allergy. Feb 2021;76(2):428-455.
5. Businaro P, Vaghi G, Marchioni E, et al. COVID-19 in patients with myasthenia gravis: Epidemiology and disease course. Muscle Nerve. Aug 2021;64(2):206-211.
6. Muppidi S, Guptill JT, Jacob S, et al. COVID-19-associated risks and effects in myasthenia gravis (CARE-MG). The Lancet. Neurology. Dec 2020; 19(12):970-971.
7. Wu Z, McGoogan JM. Characteristics of and Important Lessons From the Coronavirus Disease 2019 (COVID-19) Outbreak in China: Summary of a Report of 72 314 Cases From the Chinese Center for Disease Control and Prevention. Jama. Apr 7 2020;323(13):1239-1242.
8. Rodrigues CL, de Freitas HC, Lima PRO, et al. Myasthenia gravis exacerbation and myasthenic crisis associated with COVID-19: case series and literature review. Neurol Sci. Apr 2022;43(4):2271-2276.
9. Digala LP, Prasanna S, Rao P, Qureshi AI, Govindarajan R. Impact of COVID-19 infection among myasthenia gravis patients- a Cerner Real-World Data(TM) study. BMC Neurol. Jan 27 2022; 22(1):38.
10. Saied Z, Rachdi A, Thamlaoui S, et al. Myasthenia gravis and COVID-19: A case series and comparison with literature. Acta Neurol Scand. Sep 2021;144(3): 334-340.
11. Tugasworo D, Kurnianto A, Retnaningsih, Andhitara Y, Ardhini R, Budiman J. The relationship between myasthenia gravis and COVID-19: a systematic review. The Egyptian journal of neurology, psychiatry and neurosurgery. 2022;58(1):83.
12. Camelo-Filho AE, Silva AMS, Estephan EP, et al. Myasthenia Gravis and COVID-19: Clinical Characteristics and Outcomes. Frontiers in neurology. 2020;11:1053.
13. Abbas AS, Hardy N, Ghozy S, et al. Characteristics, treatment, and outcomes of Myasthenia Gravis in COVID-19 patients: A systematic review. Clin Neurol Neurosurg. Feb 2022;213:107140.
14. Frontera JA, Sabadia S, Lalchan R, et al. A Prospective Study of Neurologic Disorders in Hospitalized Patients With COVID-19 in New York City. Neurology. Jan 26 2021;96(4):e575-e586.
15. Shah SMI, Yasmin F, Memon RS, et al. COVID-19 and myasthenia gravis: A review of neurological implications of the SARS-COV-2. Brain Behav. Dec 2022;12(12):e2789.
16. Sriwastava S, Tandon M, Kataria S, Daimee M, Sultan S. New onset of ocular myasthenia gravis in a patient with COVID-19: a novel case report and literature review. J Neurol. Aug 2021;268(8):2690-2696.
17. Tereshko Y, Gigli GL, Pez S, De Pellegrin A, Valente M. New-onset Myasthenia Gravis after SARS-CoV-2 infection: case report and literature review. J Neurol. Feb 2023;270(2):601-609.
18. Aladawi M, Elfil M, Abu-Esheh B, et al. Guillain Barre Syndrome as a Complication of COVID-19:

A Systematic Review. Can J Neurol Sci. Jan 2022;49(1):38-48.
19. Ivan AP, Odajiu I, Popescu BO, Davidescu EI. COVID-19 Associated Guillain-Barre Syndrome: A Report of Nine New Cases and a Review of the Literature. Medicina (Kaunas). Jul 22 2022;58(8).
20. Elzouki AN, Osman MAM, Ahmed MAE, et al. COVID-19 infection presented as Guillain-Barre Syndrome: Report of two new cases and review of 116 reported cases and case series. Travel Med Infect Dis. Nov-Dec 2021;44:102169.
21. Qureshi Z, Kandhi S, Prasai N, Altaf F, Dhallu M. COVID-19-Associated Autoimmune Disease: A Rare First Case Report of Acute Motor Axonal Neuropathy Variant of Guillain-Barre Syndrome in a Woman Patient in New York City. Cureus. Feb 2022;14(2):e22290.
22. Korem S, Gandhi H, Dayag DB. Guillain-Barre syndrome associated with COVID-19 disease. BMJ Case Rep. Sep 21 2020;13(9).
23. Sheikh AB, Chourasia PK, Javed N, et al. Association of Guillain-Barre syndrome with COVID-19 infection: An updated systematic review. J Neuroimmunol. Jun 15 2021;355:577577.
24. Abrams RMC, Kim BD, Markantone DM, et al. Severe rapidly progressive Guillain-Barre syndrome in the setting of acute COVID-19 disease. J Neurovirol. Oct 2020;26(5):797-799.
25. Saud A, Naveen R, Aggarwal R, Gupta L. COVID-19 and Myositis: What We Know So Far. Current rheumatology reports. Aug 2021;23(8).
26. Hannah JR, Ali SS, Nagra D, et al. Skeletal muscles and Covid-19: a systematic review of rhabdomyolysis and myositis in SARS-CoV-2 infection. Clinical and experimental rheumatology. Feb 2022;40(2):329-338.
27. Tanboon J, Nishino I. COVID-19-associated myositis may be dermatomyositis. Muscle & nerve. Jan 2021;63(1):E9-E10.
28. Movahedi N, Ziaee V. COVID-19 and myositis; true dermatomyositis or prolonged post viral myositis? Pediatr Rheumatol. Jun 10 2021;19(1).
29. Crum-Cianflone NF. Bacterial, fungal, parasitic, and viral myositis. Clinical microbiology reviews. Jul 2008;21(3):473-494.
30. Lamabadusuriya SP, Witharana N, Preethimala LD. Viral myositis caused by Epstein-Barr virus (EB virus) in children. The Ceylon medical journal. Mar 2002;47(1):38.
31. Paliwal VK, Garg RK, Gupta A, Tejan N. Neuromuscular presentations in patients with COVID-19. Neurol Sci. Nov 2020;41(11):3039-3056.
32. Flores-Silva FD, Garcia-Grimshaw M, Valdes-Ferrer SI, et al. Neurologic manifestations in hospitalized patients with COVID-19 in Mexico City. PloS one. Apr 8 2021;16(4).
33. Tuzun S, Keles A, Okutan D, Yildiran T, Palamar D. Assessment of musculoskeletal pain, fatigue and grip strength in hospitalized patients with COVID-19. Eur J Phys Rehab Med. Aug 2021;57(4):653-662.
34. Aschman T, Stenzel W. COVID-19 associated myopathy. Current opinion in neurology. Oct 2022; 35(5):622-628.
35. Hsueh SJ, Lee MJ, Chen HS, Chang KC. Myopathy associated with COVID-19. J Formos Med Assoc. Mar 2021;120(3):1022-1024.
36. Lee N, Hui D, Wu A, et al. A major outbreak of severe acute respiratory syndrome in Hong Kong. N Engl J Med. May 15 2003;348(20):1986-1994.
37. Leung TW, Wong KS, Hui AC, et al. Myopathic changes associated with severe acute respiratory syndrome - A postmortem case series. Arch Neurol-Chicago. Jul 2005;62(7):1113-1117.
38. Alsaad KO, Hajeer AH, Al Balwi M, et al. Histopathology of Middle East respiratory syndrome coronovirus (MERS-CoV) infection - clinicopathological and ultrastructural study. Histopathology. Feb 2018;72(3):516-524.
39. Al-Aly Z, Xie Y, Bowe B. High-dimensional characterization of post-acute sequelae of COVID-19. Nature. Jun 2021;594(7862):259-264.
40. Lechien JR, Chiesa-Estomba CM, Place S, et al. Clinical and epidemiological characteristics of 1420 European patients with mild-to-moderate coronavirus disease 2019. J Intern Med. Sep 2020;288(3):335-344.
41. Suh J, Mukerji SS, Collens SI, et al. Skeletal Muscle and Peripheral Nerve Histopathology in COVID-19. Neurology. Aug 24 2021;97(8):e849-e858.
42. Abenza-Abildua MJ, Ramirez-Prieto MT, Moreno-Zabaleta R, et al. Neurological complications in critical patients with COVID-19. Neurologia (Engl

Ed). Nov-Dec 2020;35(9):621-627.
43. Hameed S, Khan AF, Khan S. Electrodiagnostic findings in COVID-19 patients: A single center experience. Clin Neurophysiol. Dec 2021;132(12): 3019-3024.
44. Friedrich O, Reid MB, Van den Berghe G, et al. The Sick and the Weak: Neuropathies/Myopathies in the Critically Ill. Physiol Rev. Jul 2015;95(3):1025-1109.
45. Bagnato S, Boccagni C, Marino G, Prestandrea C, D'Agostino T, Rubino F. Critical illness myopathy after COVID-19. Int J Infect Dis. Oct 2020;99:276-278.
46. Crisafulli O, Baroscelli M, Grattarola L, Tansini G, Zampella C, D'Antona G. Case report: Personalized adapted motor activity in a COVID-19 patient complicated by critical illness polyneuropathy and myopathy. Front Physiol. 2022;13:1035255.
47. Aschman T, Stenzel W. COVID-19 associated myopathy. Curr Opin Neurol. Oct 1 2022;35(5):622-628.
48. Lu R, Zhao X, Li J, et al. Genomic characterisation and epidemiology of 2019 novel coronavirus: implications for virus origins and receptor binding. Lancet. Feb 22 2020;395(10224):565-574.
49. Shi Z, de Vries HJ, Vlaar APJ, et al. Diaphragm Pathology in Critically Ill Patients With COVID-19 and Postmortem Findings From 3 Medical Centers. JAMA internal medicine. Jan 1 2021;181(1):122-124.
50. Ramani SL, Samet J, Franz CK, et al. Musculoskeletal involvement of COVID-19: review of imaging. Skeletal radiology. Sep 2021;50(9):1763-1773.
51. Manzano GS, Woods JK, Amato AA. Covid-19-Associated Myopathy Caused by Type I Interferonopathy. The New England journal of medicine. Dec 10 2020;383(24):2389-2390.
52. Zhang H, Charmchi Z, Seidman RJ, Anziska Y, Velayudhan V, Perk J. COVID-19-associated myositis with severe proximal and bulbar weakness. Muscle & nerve. Sep 2020;62(3):E57-E60.
53. Aschman T, Schneider J, Greuel S, et al. Association Between SARS-CoV-2 Infection and Immune-Mediated Myopathy in Patients Who Have Died. JAMA neurology. Aug 1 2021;78(8):948-960.
54. Tankisi H. Critical illness myopathy and polyneuropathy in Covid-19: Is it a distinct entity? Clin Neurophysiol. Jul 2021;132(7):1716-1717.
55. Nasuelli NA, Pettinaroli R, Godi L, et al. Critical illness neuro-myopathy (CINM) and focal amyotrophy in intensive care unit (ICU) patients with SARS-CoV-2: a case series. Neurol Sci. Mar 2021; 42(3):1119-1121.
56. Dimitriadis K, Meis J, Neugebauer H, et al. Neurologic manifestations of COVID-19 in critically ill patients: results of the prospective multicenter registry PANDEMIC. Crit Care. Jul 16 2022;26(1): 217.
57. McClafferty B, Umer I, Fye G, et al. Approach to critical illness myopathy and polyneuropathy in the older SARS-CoV-2 patients. J Clin Neurosci. Sep 2020;79:241-245.
58. Cacciani N, Skarlen A, Wen Y, et al. A prospective clinical study on the mechanisms underlying critical illness myopathy-A time-course approach. J Cachexia Sarcopenia Muscle. Dec 2022;13(6):2669-2682.
59. Fenzi F, Latronico N, Refatti N, Rizzuto N. Enhanced expression of E-selectin on the vascular endothelium of peripheral nerve in critically ill patients with neuromuscular disorders. Acta Neuropathol. Jul 2003; 106(1):75-82.
60. Cheung K, Rathbone A, Melanson M, Trier J, Ritsma BR, Allen MD. Pathophysiology and management of critical illness polyneuropathy and myopathy. J Appl Physiol (1985). May 1 2021;130(5):1479-1489.
61. Madia F, Merico B, Primiano G, Cutuli SL, De Pascale G, Servidei S. Acute myopathic quadriplegia in patients with COVID-19 in the intensive care unit. Neurology. Sep 15 2020;95(11):492-494.
62. Dodig D, Tarnopolsky MA, Margeta M, Gordon K, Fritzler MJ, Lu JQ. COVID-19-Associated Critical Illness Myopathy with Direct Viral Effects. Ann Neurol. Apr 2022;91(4):568-574.
63. Cabanes-Martinez L, Villadoniga M, Gonzalez-Rodriguez L, et al. Neuromuscular involvement in COVID-19 critically ill patients. Clin Neurophysiol. Dec 2020;131(12):2809-2816.
64. Scarpino M, Bonizzoli M, Lazzeri C, et al. Electrodiagnostic findings in patients with non-COVID-19- and COVID-19-related acute respiratory distress syndrome. Acta Neurol Scand. Aug 2021; 144(2):161-169.

65. Rodriguez B, Branca M, Gutt-Will M, et al. Development and early diagnosis of critical illness myopathy in COVID-19 associated acute respiratory distress syndrome. J Cachexia Sarcopenia Muscle. Jun 2022;13(3):1883-1895.
66. Zupanc A, Vidmar G, Majdic N, Novak P. Health-related quality-of-life during rehabilitation in patients with critical illness neuropathy/myopathy after severe coronavirus disease 2019. Int J Rehabil Res. Mar 1 2023;46(1):53-60.
67. Intiso D, Marco Centra A, Giordano A, Santamato A, Amoruso L, Di Rienzo F. Critical Illness Polyneuropathy and Functional Outcome in Subjects with Covid-19: Report on Four Patients and a Scoping Review of the Literature. J Rehabil Med. Apr 7 2022;54:jrm00257.
68. Jacob S, Kapadia R, Soule T, et al. Neuromuscular Complications of SARS-CoV-2 and Other Viral Infections. Front Neurol. 2022;13:914411.
69. Shahrizaila N, Lehmann HC, Kuwabara S. Guillain-Barre syndrome. Lancet. Mar 27 2021;397(10280): 1214-1228.
70. Lee H, Heo N, Kwon D, Ha J. Deciphering changes in the incidence of the Guillain-Barre syndrome during the COVID-19 pandemic: a nationwide time-series correlation study. BMJ Neurol Open. 2022;4(2):e000378.
71. Jaffry M, Mostafa F, Mandava K, et al. No significant increase in Guillain-Barre syndrome after COVID-19 vaccination in adults: A vaccine adverse event reporting system study. Vaccine. Sep 22 2022;40(40):5791-5797.
72. Hafsteinsdottir B, Dalemo E, Eliasdottir O, Olafsson E, Axelsson M. Decreased Incidence of Guillain-Barre Syndrome during the COVID-19 Pandemic: A Retrospective Population-Based Study. Neuroepidemiology. 2023;57(1):1-6.
73. Finsterer J, Matovu D, Scorza FA. SARS-CoV-2 vaccinations reduce the prevalence of post-COVID Guillain-Barre syndrome. Clinics (Sao Paulo). 2022;77:100064.
74. Keh RYS, Scanlon S, Datta-Nemdharry P, et al. COVID-19 vaccination and Guillain-Barre syndrome: analyses using the National Immunoglobulin Database. Brain. Feb 13 2023;146(2):739-748.
75. Patone M, Handunnetthi L, Saatci D, et al. Neurological complications after first dose of COVID-19 vaccines and SARS-CoV-2 infection. Nat Med. Dec 2021;27(12):2144-2153.
76. Li X, Raventos B, Roel E, et al. Association between covid-19 vaccination, SARS-CoV-2 infection, and risk of immune mediated neurological events: population based cohort and self-controlled case series analysis. BMJ. Mar 16 2022;376:e068373.
77. Kim MS, Jung SY, Ahn JG, et al. Comparative safety of mRNA COVID-19 vaccines to influenza vaccines: A pharmacovigilance analysis using WHO international database. J Med Virol. Mar 2022; 94(3):1085-1095.
78. Abara WE, Gee J, Marquez P, et al. Reports of Guillain-Barre Syndrome After COVID-19 Vaccination in the United States. JAMA Netw Open. Feb 1 2023;6(2):e2253845.
79. Garcia-Grimshaw M, Galnares-Olalde JA, Bello-Chavolla OY, et al. Incidence of Guillain-Barre syndrome following SARS-CoV-2 immunization: Analysis of a nationwide registry of recipients of 81 million doses of seven vaccines. Eur J Neurol. Nov 2022;29(11):3368-3379.
80. Tamborska AA, Singh B, Leonhard SE, et al. Guillain-Barre syndrome following SARS-CoV-2 vaccination in the UK: a prospective surveillance study. BMJ Neurol Open. 2022;4(2):e000309.
81. Shao SC, Wang CH, Chang KC, Hung MJ, Chen HY, Liao SC. Guillain-Barre Syndrome Associated with COVID-19 Vaccination. Emerg Infect Dis. Dec 2021;27(12):3175-3178.
82. Ha J, Park S, Kang H, et al. Real-world data on the incidence and risk of Guillain-Barre syndrome following SARS-CoV-2 vaccination: a prospective surveillance study. Sci Rep. Mar 7 2023;13(1):3773.
83. Ling L, Bagshaw SM, Villeneuve PM. Guillain-Barre syndrome after SARS-CoV-2 vaccination in a patient with previous vaccine-associated Guillain-Barre syndrome. CMAJ. Nov 22 2021;193(46): E1766-E1769.
84. Baars AE, Kuitwaard K, de Koning LC, et al. SARS-CoV-2 Vaccination Safety in Guillain-Barre Syndrome, Chronic Inflammatory Demyelinating Polyneuropathy, and Multifocal Motor Neuropathy.

Neurology. Jan 10 2023;100(2):e182-e191.
85. Shapiro Ben David S, Potasman I, Rahamim-Cohen D. Rate of Recurrent Guillain-Barre Syndrome After mRNA COVID-19 Vaccine BNT162b2. JAMA Neurol. Nov 1 2021;78(11):1409-1411.
86. Bellucci M, Germano F, Grisanti S, et al. Case Report: Post-COVID-19 Vaccine Recurrence of Guillain-Barre Syndrome Following an Antecedent Parainfectious COVID-19-Related GBS. Front Immunol. 2022;13:894872.
87. Liang H, Cao Y, Zhong W, Ma Z, Liu J, Chen H. Miller-Fisher syndrome and Guillain-Barre syndrome overlap syndrome following inactivated COVID-19 vaccine: Case report and scope review. Hum Vaccin Immunother. Nov 30 2022;18(6):2125753.
88. Siddiqi AR, Khan T, Tahir MJ, Asghar MS, Islam MS, Yousaf Z. Miller Fisher syndrome after COVID-19 vaccination: Case report and review of literature. Medicine (Baltimore). May 20 2022;101(20):e29333.
89. Castiglione JI, Crespo JM, Lecchini L, et al. Bilateral facial palsy with paresthesias, variant of Guillain-Barre syndrome following COVID-19 vaccine: A case series of 9 patients. Neuromuscul Disord. Jul 2022;32(7):572-574.
90. Germano F, Bellucci M, Grisanti S, et al. COVID-19 vaccine-related Guillain-Barre syndrome in the Liguria region of Italy: A multicenter case series. J Neurol Sci. Sep 15 2022;440:120330.
91. Taga A, Lauria G. COVID-19 and the peripheral nervous system. A 2-year review from the pandemic to the vaccine era. J Peripher Nerv Syst. Mar 2022; 27(1):4-30.
92. Woo EJ, Mba-Jonas A, Dimova RB, Alimchandani M, Zinderman CE, Nair N. Association of Receipt of the Ad26.COV2.S COVID-19 Vaccine With Presumptive Guillain-Barre Syndrome, February-July 2021. JAMA. Oct 26 2021;326(16):1606-1613.
93. Chalela JA, Andrews C, Bashmakov A, Kapoor N, Snelgrove D. Reports of Guillain-Barre Syndrome Following COVID-19 Vaccination in the USA: An Analysis of the VAERS Database. J Clin Neurol. Mar 2023;19(2):179-185.
94. Chua SKK, Soh QY, Saffari SE, Tan EK. Prognosis of Guillain-Barre Syndrome Linked to COVID-19 Vaccination. Brain Sci. May 30 2022;12(6).
95. Finsterer J, Scorza FA, Scorza CA. Post SARS-CoV-2 vaccination Guillain-Barre syndrome in 19 patients. Clinics (Sao Paulo). 2021;76:e3286.
96. van Alfen N, van Engelen BG. The clinical spectrum of neuralgic amyotrophy in 246 cases. Brain. Feb 2006;129(Pt 2):438-450.
97. Min YG, Kim JE, Hwang JY, Shin JY, Sung JJ, Hong YH. Parsonage-Turner syndrome following COVID-19 vaccination. J Neurol Neurosurg Psychiatry. Apr 6 2022;93(11):1231-1232.
98. Kim JE, Park J, Min YG, Hong YH, Song TJ. Associations of neuralgic amyotrophy with COVID-19 vaccination: Disproportionality analysis using the World Health Organization pharmacovigilance database. Muscle Nerve. Dec 2022;66(6):766-770.
99. Civardi C, Delconte C, Pisano F, Collini A, Geda C. Isolated musculocutaneous involvement in neuralgic amyotrophy associated with SARS-CoV2 vaccination. Neurol Sci. Jun 2022;43(6):3515-3517.
100. Amjad MA, Hamid Z, Patel Y, et al. COVID-19 Vaccine-Induced Parsonage-Turner Syndrome: A Case Report and Literature Review. Cureus. May 2022;14(5):e25493.
101. Lakkireddy M, Sathu S, Kumar R, Madhu Latha K, Maley DK. Parsonage-Turner Syndrome Following Covishield (AstraZeneca ChAdOx1 nCoV-19) Vaccination: A Case Report. Cureus. Aug 2022;14(8):e27867.
102. Meixedo S, Correia M, Machado Lima A, Carneiro I. Parsonage-Turner Syndrome Post-COVID-19 Oxford/AstraZeneca Vaccine Inoculation: A Case Report and Brief Literature Review. Cureus. Feb 2023;15(2):e34710.
103. Bernheimer JH, Gasbarro G. Parsonage Turner Syndrome Following Vaccination With mRNA-1273 SARS-CoV-2 Vaccine. J Clin Neuromuscul Dis. Jun 1 2022;23(4):229-230.
104. Crespo Burillo JA, Loriente Martinez C, Garcia Arguedas C, Mora Pueyo FJ. Amyotrophic neuralgia secondary to Vaxzevri (AstraZeneca) COVID-19 vaccine. Neurologia (Engl Ed). Sep 2021;36(7):571-572.
105. James J, Johnson J, Jose J. Neuralgic Amyotrophy After ChAdOx1 nCoV-19 COVID-19 Vaccination. J Clin Neuromuscul Dis. Dec 1 2022;24(2):112-113.

106. Flikkema K, Brossy K. Parsonage-Turner Syndrome After COVID-19 Vaccination: A Case Report. JBJS Case Connect. Dec 22 2021;11(4).
107. Mahajan S, Zhang F, Mahajan A, Zimnowodzki S. Parsonage Turner syndrome after COVID-19 vaccination. Muscle Nerve. Jul 2021;64(1):E3-E4.
108. Coffman JR, Randolph AC, Somerson JS. Parsonage-Turner Syndrome After SARS-CoV-2 BNT162b2 Vaccine: A Case Report. JBJS Case Connect. Sep 24 2021;11(3).
109. Diaz-Segarra N, Edmond A, Gilbert C, McKay O, Kloepping C, Yonclas P. Painless idiopathic neuralgic amyotrophy after COVID-19 vaccination: A case report. PM R. Jul 2022;14(7):889-891.
110. Queler SC, Towbin AJ, Milani C, Whang J, Sneag DB. Parsonage-Turner Syndrome Following COVID-19 Vaccination: MR Neurography. Radiology. Jan 2022;302(1):84-87.
111. Vitturi BK, Grandis M, Beltramini S, et al. Parsonage-Turner syndrome following coronavirus disease 2019 immunization with ChAdOx1-S vaccine: a case report and review of the literature. J Med Case Rep. Dec 13 2021;15(1):589.
112. Chua MMJ, Hayes MT, Cosgrove R. Parsonage-Turner syndrome following COVID-19 vaccination and review of the literature. Surg Neurol Int. 2022; 13:152.
113. Oncel A, Coskun E. Parsonage-Turner syndrome after SARS-CoV-2 vaccination: A case report. Turk J Phys Med Rehabil. Sep 2022;68(3):418-421.
114. Ng GJ, Chiew YR, Kong Y, Koh JS. Neuralgic amyotrophy in COVID-19 infection and after vaccination. Ann Acad Med Singap. Jun 2022;51(6): 376-377.
115. Cassart EM, Vilas DR, Abe R, Cavanilles-Walker JM. Parsonage-Turner Syndrome After COVID-19 Vaccination in a Child. J Am Acad Orthop Surg Glob Res Rev. Mar 1 2023;7(3).
116. Koh JS, Goh Y, Tan BY, et al. Neuralgic amyotrophy following COVID-19 mRNA vaccination. QJM. Nov 5 2021;114(7):503-505.
117. Tsairis P, Dyck PJ, Mulder DW. Natural history of brachial plexus neuropathy. Report on 99 patients. Arch Neurol. Aug 1972;27(2):109-117.
118. Van Eijk JJ, Groothuis JT, Van Alfen N. Neuralgic amyotrophy: An update on diagnosis, pathophysiology, and treatment. Muscle Nerve. Mar 2016;53(3):337-350.
119. Alcantara M, Koh M, Park AL, Bril V, Barnett C. Outcomes of COVID-19 Infection and Vaccination Among Individuals With Myasthenia Gravis. JAMA Netw Open. Apr 3 2023;6(4):e239834.
120. Breiner A, Bourque PR. Vaccination against COVID-19 does not lead to exacerbation in patients with myasthenia gravis. Muscle Nerve. Jan 2023; 67(1):1-2.
121. Trinchillo A, Esposito M, Habetswallner F, Tuccillo F, De Martino BM. COVID19 vaccine in myasthenia gravis patients: safety and possible predictors of disease exacerbation. Neurol Sci. Feb 2023;44(2):447-450.
122. Doron A, Piura Y, Vigiser I, et al. BNT162b2 mRNA COVID-19 vaccine three-dose safety and risk of COVID-19 in patients with myasthenia gravis during the alpha, delta, and omicron waves. J Neurol. Dec 2022;269(12):6193-6201.
123. Ishizuchi K, Takizawa T, Sekiguchi K, et al. Flare of myasthenia gravis induced by COVID-19 vaccines. J Neurol Sci. May 15 2022;436:120225.
124. Reyes-Leiva D, Lopez-Contreras J, Moga E, et al. Immune Response and Safety of SARS-CoV-2 mRNA-1273 Vaccine in Patients With Myasthenia Gravis. Neurol Neuroimmunol Neuroinflamm. Jul 2022;9(4).
125. Ruan Z, Tang Y, Li C, et al. COVID-19 Vaccination in Patients with Myasthenia Gravis: A Single-Center Case Series. Vaccines (Basel). Sep 29 2021;9(10).
126. Peric S, Rankovic M, Bozovic I, et al. COVID-19 infection and vaccination against SARS-CoV-2 in myasthenia gravis. Acta Neurol Belg. Apr 2023; 123(2):529-536.
127. Lotan I, Hellmann MA, Friedman Y, Stiebel-Kalish H, Steiner I, Wilf-Yarkoni A. Early safety and tolerability profile of the BNT162b2 COVID-19 vaccine in myasthenia gravis. Neuromuscul Disord. Mar 2022;32(3):230-235.
128. Golding B, Lee Y, Golding H, Khurana S. Pause in immunosuppressive treatment results in improved immune response to SARS-CoV-2 vaccine in autoimmune patient: a case report. Ann Rheum Dis.

Oct 2021;80(10):1359-1361.
129. McNeish BL, Colletti RB, Hehir MK. Safety and Immunogenicity of Additional SARS-CoV-2 Vaccinations in a Patient With Myasthenia Gravis on Mycophenolate: A Case Report. J Clin Neuromuscul Dis. Dec 1 2022;24(2):113-114.
130. Plymate LC, Pepper G, Krist MP, Koelle DM. Immunogenicity of repeat COVID-19 mRNA vaccinations in a patient with myasthenia gravis receiving mycophenolate, prednisone, and eculizumab. J Transl Autoimmun. 2021;4:100114.
131. Damato V, Spagni G, Monte G, et al. Immunological response after SARS-CoV-2 infection and mRNA vaccines in patients with myasthenia gravis treated with Rituximab. Neuromuscul Disord. Mar 2023;33(3):288-294.
132. Chavez A, Pougnier C. A Case of COVID-19 Vaccine Associated New Diagnosis Myasthenia Gravis. J Prim Care Community Health. Jan-Dec 2021;12:21501327211051933.
133. Maher DI, Hogarty D, Ben Artsi E. Acute onset ocular myasthenia gravis after vaccination with the Oxford-AstraZeneca COVID-19 vaccine. Orbit. May 2 2022:1-5.
134. Croitoru CG, Cuciureanu DI, Prutianu I, Cianga P. Autoimmune myasthenia gravis after COVID-19 in a triple vaccinated patient. Arch Clin Cases. 2022;9(3):104-107.
135. Galassi G, Rispoli V, Iori E, Ariatti A, Marchioni A. Coincidental Onset of Ocular Myasthenia Gravis Following ChAdOx1 n-CoV-19 Vaccine against Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2). Isr Med Assoc J. Jan 2022;24(1):9-10.
136. Virgilio E, Tondo G, Montabone C, Comi C. COVID-19 Vaccination and Late-Onset Myasthenia Gravis: A New Case Report and Review of the Literature. Int J Environ Res Public Health. Dec 27 2022;20(1).
137. Lee MA, Lee C, Park JH, Lee JH. Early-Onset Myasthenia Gravis Following COVID-19 Vaccination. J Korean Med Sci. Mar 14 2022;37(10):e50.
138. Poli K, Poli S, Ziemann U. Multiple Autoimmune Syndromes Including Acute Disseminated Encephalomyelitis, Myasthenia Gravis, and Thyroiditis Following Messenger Ribonucleic Acid-Based COVID-19 Vaccination: A Case Report. Front Neurol. 2022;13:913515.
139. Hoshina Y, Sowers C, Baker V. Myasthenia Gravis Presenting after Administration of the mRNA-1273 Vaccine. Eur J Case Rep Intern Med. 2022;9(7):003439.
140. Kang MC, Park KA, Min JH, Oh SY. Myasthenia gravis with ocular symptoms following a ChAdOx1 nCoV-19 vaccination: A case report. Am J Ophthalmol Case Rep. Sep 2022;27:101620.
141. Huang BD, Hsueh HW, Yang SH, Lin CW. New-Onset Myasthenia Gravis After ChAdOx1 nCOV-19 Vaccine Inoculation. J Neuroophthalmol. Mar 24 2022.
142. Slavin E, Fitzig J, Neubert C, Garcia-Lopez F, Cuevas-Trisan R. New-Onset Myasthenia Gravis Confirmed by Electrodiagnostic Studies After a Third Dose of SARS-CoV-2 mRNA-1273 Vaccine. Am J Phys Med Rehabil. Dec 1 2022;101(12):e176-e179.
143. Abicic A, Sitas B, Adamec I, Bilic E, Habek M. New-Onset Ocular Myasthenia Gravis After Booster Dose of COVID-19 Vaccine. Cureus. Jul 2022;14(7):e27213.
144. Fanella G, Baiata C, Candeloro E, et al. New-onset myasthenia gravis after mRNA SARS-CoV-2 vaccination: a case series. Neurol Sci. Oct 2022;43(10):5799-5802.
145. Ozenc B, Odabasi Z. New-Onset Myasthenia Gravis Following COVID-19 Vaccination. Ann Indian Acad Neurol. Nov-Dec 2022;25(6):1224-1225.
146. Ramdas S, Hum RM, Price A, et al. SARS-CoV-2 vaccination and new-onset myasthenia gravis: A report of 7 cases and review of the literature. Neuromuscul Disord. Oct 2022;32(10):785-789.
147. Mirmosayyeb O, Moases Ghaffary E, Mazdak M, Bagheri Z, Bagherieh S, Shaygannejad V. Is Myasthenia Gravis a Real Complication of the COVID-19 Vaccine? A Case Report-Based Systematic Review. Can J Infect Dis Med Microbiol. 2022;2022:5009450.