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Case Report

A Case Report and Literature Review of New-Onset Myasthenia Gravis after COVID-19 Infection

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Abstract: Myasthenia gravis (MG) is an autoimmune disorder affecting the neuromuscular junction caused by a B-cell-mediated, T-cell-dependent immunologic attack at the end plate of the postsynaptic membrane. Attack on muscle acetylcholine receptors (AChR) of the postsynaptic membrane due to the AChR, muscle-specific tyrosine kinase, or lipoprotein receptor-related peptide 4 antibodies lead to symptoms of painless, fluctuating weakness of muscle groups and often begins with ocular signs and symptoms. Coronavirus disease 2019 (COVID-19) is an acute respiratory disease caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), a novel coronavirus closely related to SARS-CoV. Serious neurologic complications are infrequent and diverse with reported cases of stroke, encephalitis/meningitis, Guillain-Barré syndrome, acute disseminated encephalomyelitis, ataxia, and unspecified limb weakness. MG is a rarely reported sequela of COVID-19 infection. To date, there are 15 reported cases of post-COVID-19 MG. In this article, we present a case of post-COVID-19 MG and a concise review of other reported cases. An 83-year-old Caucasian male with a medical history of atrial fibrillation status post-ablation and non-ischemic cardiomyopathy was initially admitted for COVID-19 pneumonia. He was treated with remdesivir, convalescent plasma, and supplemental oxygen therapy but did not require invasive mechanical intubation. One month after discharge, he started experiencing fatigue with muscle weakness and progressive dyspnea. He progressed to develop dysphonia, especially at the end of the day. After extensive workup, he was diagnosed with MG with a positive antibody against the AChR. The chronological events of developing slowly worsening muscular weakness after recovering from COVID-19 infection and positive AChR antibody led to the diagnosis of post-COVID-19 new-onset MG. Post-COVID-19 fatigue, long-term use of steroids, and intensive care unit-related physical deconditioning can be confounders in the clinical presentation of post-COVID-19 new-onset MG. Careful history-taking and meticulous assessment of chronological events are needed to diagnose this rare entity.

Keywords: Myasthenia gravis (MG), COVID-19, encephalitis/meningitis, Guillain-Barré syndrome, acute disseminated encephalomyelitis, ataxia, and unspecified limb weakness.

INTRODUCTION

As the coronavirus disease 2019 (COVID-19) pandemic has started stabilizing, the secondary complications and long-term sequelae of the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection have begun to surface [1]. It is evident that the morbidity of SARS-CoV-2 infection extends beyond the phase of acute respiratory illness and may affect any organ besides the respiratory system, including the cardiovascular, hematological, and nervous systems [2]. Dysgeusia, anosmia, chronic headaches, hemorrhagic or ischemic strokes, encephalitis/meningitis, encephalopathy, Guillain-Barré syndrome, acute disseminated encephalomyelitis (ADEM), ataxia, neuropathy, and unspecified limb weakness have been reported as post-COVID-19 neurological complications [3, 4]. New-onset myasthenia gravis (MG) is a rarely reported neuromuscular complication of SARS-CoV-2 infection. MG is an autoimmune disorder affecting the neuromuscular junction caused by a B-cell-mediated, T-cell-dependent immunologic attack at the end plate of the postsynaptic membrane [5]. Attack on muscle acetylcholine receptors (AChR) of the postsynaptic membrane due to the acetylcholine AChR,

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muscle-specific tyrosine kinase (MuSK), or lipoprotein receptor-related peptide 4 (LRP4) antibodies lead to symptoms of painless, fluctuating weakness of muscle groups and often begins with ocular signs and symptoms [3].

Diagnosis of MG may be difficult in the post-infectious phase of COVID-19. Symptoms of chronic fatigue and generalized muscular weakness are frequently seen as a part of the long COVID-19 sequela. Furthermore, prolonged duration of hospital stays, mechanical ventilation, limited mobility with isolation, and steroid use can contribute to neuromuscular weakness. A detailed history and a precise time frame of symptom onset play a role in raising suspicion for MG. The literature review showed only 15 reported cases of post-COVID-19 new-onset MG. We present a case of post-COVID-19 new-onset MG in an elderly male with no previous history of neuromuscular or autoimmune disorder.

CASE PRESENTATION

A 73-year-old male with a medical history of atrial fibrillation status post-ablation, non-ischemic cardiomyopathy, and moderate mitral regurgitation was admitted to the intensive care unit (ICU) in December 2020 with acute respiratory failure due to COVID-19 pneumonia. He was treated with heated high-flow oxygen, remdesivir, and convalescent plasma therapy and was hospitalized for 17 days. He was unvaccinated against SARS-CoV-2 at the time of the initial infection. A month after discharge, he started experiencing significant fatigue with muscle weakness and progressive dyspnea. Workup for these symptoms was initially pursued in February 2021. This included an echocardiogram showing moderate-to-severe mitral regurgitation and a chest X-ray which showed ill-defined opacities in the upper lung zones suggestive of atelectasis versus viral pneumonia. A follow-up computed tomography (CT) of the chest showed mild emphysematous changes with prominent interstitial markings in the right upper and middle lobe with no consolidation, pleural effusion, and pulmonary fibrosis. At this time, his dyspnea was attributed to worsening mitral regurgitation. However, his fatigue continued to progress. Due to a history of atrial fibrillation, a loop recorder was implanted which did not show any increased atrial fibrillation burden contributing to his fatigue. His generalized weakness continued to progress over the next few months, and his coworkers reported that his speech would progressively become incomprehensible throughout the day. In September 2021, he was admitted again with acute hypoxemic respiratory failure requiring intubation and mechanical ventilation. He was suspected of having aspiration pneumonia and possibly angiotensin receptor inhibitor-induced angioedema. He was treated with broad-spectrum antibiotics and methylprednisolone 80 mg. Thereafter, methylprednisolone was switched to prednisone 40 mg daily, which was tapered over the course of five days. He was weaned off the ventilator and extubated. After the completion of his taper, his dysarthria and dysphagia worsened. C1 esterase inhibitor function returned normal (result: 83%, normal: >67%).

A brief course of prednisone (40 mg/40 mg/30 mg/30 mg/20 mg/20 mg/10 mg/10 mg over eight days) was started by his primary care provider, and he was referred urgently to a neurologist. Symptoms again improved with prednisone but returned upon completion. His neurologist started him on prednisone 60 mg every other day and pyridostigmine 30 mg daily for residual weakness. However, the patient could not tolerate pyridostigmine, resulting in its discontinuation. The patient was started on azathioprine 50 mg twice daily, and the dose of the steroid was reduced every two weeks. A CT of the chest ruled out underlying thymoma. His symptoms of fatigue improved while on azathioprine treatment.

His MG was well controlled on azathioprine. However, he developed worsening heart failure and a decline in renal function within a year. Approximately one year after his MG diagnosis, he developed a bilateral periprosthetic joint infection with *Streptococcus oralis*/*mitis* bacteremia. He passed away from multiorgan failure.

DISCUSSION

Post-COVID-19, new-onset neurological autoimmune diseases are being increasingly recognized and reported [6-9]. MG is an autoimmune condition that is caused by autoantibodies directed toward the postsynaptic AChR on neuromuscular junctions [10, 11]. Blockade of the AChR on the postsynaptic membrane causes a decrease in action potential generation resulting in muscular weakness. It is a relatively rare disease with a prevalence of 50-125 cases per million [12].

The presentation and prognosis of COVID-19 in patients with pre-existing MG have been published since the onset of the COVID-19 pandemic [13-18]. Both new-onset MG post-SARS-CoV-2 infection [10, 11, 19, 20] and post-vaccination are sparsely reported, and the pathophysiology is currently unclear [21]. The first case of post-COVID-19 new-onset MG with ocular manifestations in a 21-year-old female was reported by Huber *et al.*, in 2020 [11]. Based on reported cases, there is no clear expected latent period between the onset of COVID-19 infection and MG symptoms. Karimi *et al*., in their case series, reported a 10-30-day gap between the initial COVID-19 infection and the onset of MG symptoms [22]. We performed a comprehensive review of such reported cases and summarize the findings. On a review of the reported cases, the gap between acute COVID-19 infection and the onset of MG symptoms ranged between five days and eight weeks. In our patient, the first MG symptoms started about four weeks after acute COVID-19 infection.

Interestingly, post-infectious MG is a known entity and has been reported after Epstein-Barr virus [29], human immunodeficiency virus [30, 31], viral pharyngitis [32], West Nile virus [33], and varicella [12] infections. Although the exact mechanism of post-viral MG remains unclear, molecular mimicry between the AChR and viral antigenic proteins has been speculated [32]. The latent period between COVID-19 symptoms and the onset of MG symptoms in the reported cases thus far supports this hypothesis [34]. One study in 1985 showed cross-reactivity between AChR and proteins of the bacteria *Escherichia coli*, *Proteus vulgaris*, and *Klebsiella pneumonia* [35]. Another study in 1989 showed cross-reactivity between the alpha subunit of AChR and a homologous domain on herpes simplex virus glycoprotein D [36]. The above studies have helped elucidate a possible association between infections and MG, at least in a subset of cases.

The pathogenesis of COVID-19 infection includes immune dysregulation. Like other viral infections, clinical autoimmunity may be induced due to a breakdown in immune tolerance. Liu *et al*., have reported multiple autoimmune antibodies that have been reported positive in patients with COVID-19 infection [37]. Other studies have also alluded to a similar theory due to the depletion of T and B cells in the setting of elevated inflammatory cytokines in patients with MG after a COVID-19 infection [24, 25, 27]. The most proposed mechanism for post-COVID-19 MG is molecular mimicry between SARS-CoV-2 proteins and AChR, supported by elevated acetylcholine receptor antibodies in most cases, including our case [11].

Close to 90% of MG patients have antibodies against the AChR [12]. A subset of the remaining 10% test positive for anti-MuSK antibodies. Those with anti-MuSK antibodies present with severe bulbar symptoms and respiratory failure [12]. The anti-AChR-positive MG cases are more commonly associated with other autoimmune disorders and usually have a good response to pyridostigmine. The anti-MuSK-positive MG cases are rarely associated with autoimmune disorders, pyridostigmine is less effective, and often requires steroids and steroid-sparing agents [27]. The anti-AChR antibodies are either IgG1 or IgG3 and are known to activate the complement system, thus leading to the formation of a membrane attack complex while anti-MuSK antibody is predominantly IgG4 class and not associated with complement activation [5]. Among the 16 reported cases including ours, there are only two cases that were anti-MuSK antibody positive and anti-AChR negative [1, 27]. Due to the fundamental differences in the development of the two different antibodies, anti-AChR and anti-MuSK, the hypothesis of the breakdown of immune self-tolerance becomes more plausible over molecular mimicry [27].

From our literature review, there were two patients who were found to have thymoma [22, 25]. In the patient reported by Karimi *et al*., it was postulated that the SARS-CoV-2 infection itself or the use of azithromycin may have unmasked a pre-existing latent MG [22]. Bhandarwar *et al*., reported an interesting case of post-COVID-19 MG where the patient developed symptoms of muscular weakness two months after COVID-19 infection and was found to have a new thymoma on CT chest imaging when compared to a CT chest from two months earlier. The patient had a complete resolution of symptoms after the thymoma was surgically resected [25]. This is the only case where there is evidence of thymoma developing after the COVID-19 infection.

Anti-cholinesterase inhibitors are the first line for symptom management in generalized MG. However, most patients require immunosuppression directed against underlying immune dysregulation eventually during their disease course [38]. So far, there are only two reported post-COVID-19 MG cases with positive anti-MuSK antibodies, and both patients required the use of azathioprine. Our patient was positive for anti-AChR antibodies, could not tolerate pyridostigmine, but responded well to steroids. However, he became steroid dependent, and, therefore, azathioprine needed to be used as a steroid-sparing agent.

The incidence of anti-AChR-associated MG has been found to have a bimodal age of presentation [5]. The early-onset MG is described in those below 40 years of age, with 60% of cases being diagnosed between the ages of 20 and 40 years. The second peak incidence occurs after 70 years of age. The prognosis is poorer in late-onset MG. These individuals have temporary relief with the use of acetylcholinesterase inhibitor treatment, and, therefore, portend side effects with the need for long-term steroid use [39]. Interestingly, our patient had a very late presentation, with symptoms starting at the age of 82 years. To date, our patient is the oldest reported case in the literature. The temporal relation of events in our patient's history strongly points toward a post-COVID-19 etiology of MG. Our patient had good functional capacity prior to the COVID-19 infection, making de novo MG less likely. Moreover, our patient had no personal or family history of autoimmune or musculoskeletal disease.

CONCLUSIONS

The correct diagnosis of our patient was delayed by a few months as muscular weakness in elderly patients might be difficult to evaluate in the post-COVID-19 infection phase, especially in the presence of comorbidities. Therefore, a high index of suspicion along with a low threshold for testing is required in individuals with difficulty in weaning off ventilators. This should especially be sought out in those in whom the degree of clinical disease does not correlate with image findings not consistent with severe COVID-19 disease. It is important to remember that post-COVID-19 fatigue, long-term use of steroids, and ICU-related physical

deconditioning are easy confounders and can delay the diagnosis as in our patient. As a result, we suspect that underdiagnosis of this condition is plausible which warrants increasing awareness. Further studies will be needed to help elucidate the definitive pathophysiology of this condition.

Competing Interests: The authors declare that they have no known competing interests or personal relationships that could have appeared to influence the work reported in this paper.

Authors' Contributions:
Youssef Haouas, Abdelhak TISSIR and Zakaria ALLAL: Drafting of manuscript
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Chafik ELKETTANI ELHAMDI and Lahoucine BAROU: Final approval

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