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CASE REPORT

Post-COVID-19 generalized myasthenia gravis: a case report and literature review

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ABSTRACT

The emergence of myasthenia gravis (MG) cases following SARS-CoV-2 infection suggests a potential triggering role of COVID-19 in the development of this autoimmune disorder. A 33-year-old woman with a family history of autoimmune disease developed generalized MG five days after a mild COVID-19 infection. Initial symptoms included progressive, fatigable limb weakness occurring after the resolution of respiratory manifestations. Electrophysiological studies demonstrated impaired neuromuscular transmission on repetitive nerve stimulation, and serum anti–acetylcholine receptor (AChR) antibodies were positive, confirming the diagnosis of generalized MG. The early presentation initially raised the differential diagnosis of post-COVID fatigue syndrome. Psychological assessment revealed mild-to-moderate anxiety and depressive symptoms (Hospital Anxiety and Depression Scale scores: anxiety 12/21; depression 11/21), which may have contributed to delayed recognition of the neuromuscular disorder. The patient was treated with pyridostigmine, followed by intravenous immunoglobulins, resulting in marked clinical improvement. She subsequently underwent thymectomy. At 12-month follow-up, she remained asymptomatic, with no evidence of recurrence. Post-COVID-19 MG appears to represent an emerging autoimmune neurological entity with distinct clinical and immunological features. Early recognition is essential in patients presenting with persistent, fluctuating weakness after SARS-CoV-2 infection. Further longitudinal studies are needed to clarify the underlying immunopathogenic mechanisms and long-term outcomes of this condition.

Keywords: Myasthenia gravis, COVID-19, post-COVID fatigue, autoimmunity, anti-AChR antibodies, thymectomy.

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1. INTRODUCTION

Myasthenia gravis (MG) is an autoimmune disorder of the neuromuscular junction characterized by fluctuating weakness due to autoantibodies against postsynaptic targets, mainly the acetylcholine receptor (AChR), and less frequently MuSK and LRP4 (1,2). Additional antigens (agrin, titin, ryanodine receptor) reflect its immunological heterogeneity, supporting a multidimensional classification based on serology, clinical phenotype, age at onset, and thymic status.

Since 2019, SARS-CoV-2 has been implicated in immune dysregulation and post-infectious autoimmunity through mechanisms including molecular mimicry, bystander activation, and epitope spreading. Large cohort data have shown an increased incidence of autoimmune diseases after COVID-19 (3), and several cases of new-onset MG have been reported, suggesting a potential triggering or unmasking role. Although causality remains unproven, these observations support a link between SARS-CoV-2 infection and autoimmune neuromuscular disorders.

In this context, we report a case of MG occurring shortly after SARS-CoV-2 infection.

2. OBSERVATION

A 33-year-old woman, a university lecturer with no significant personal medical history, had a family history of Sjögren syndrome (mother) and colorectal cancer (grandmother). She had received an inactivated SARS-CoV-2 vaccine with a single booster one year earlier. She developed a PCR-confirmed COVID-19, initially presenting with dry cough, low-grade fever, asthenia, and anorexia. On admission, vital signs showed a respiratory rate of 25/min, heart rate 85 bpm, oxygen saturation 96% on room air, and blood pressure 100/70 mmHg. Physical examination, including pulmonary auscultation, was unremarkable. Chest computed tomography revealed ground-glass opacities involving approximately 10% of the lung parenchyma, without severity features. Laboratory tests showed leukocytosis (15,000/mm³), marked lymphopenia (730/mm³), thrombocytopenia (97,000/mm³), mildly elevated transaminases (2× normal), increased lactate dehydrogenase (685 U/L), and D-dimers (600 ng/mL).

She was managed as an outpatient with dexamethasone (5 days) and amoxicillin–clavulanate (7 days), with improvement in respiratory symptoms but persistent asthenia. On day 5, she reported progressive muscle fatigue, predominantly affecting the upper limbs and worsening at the end of the day; rest and vitamin C supplementation were advised.

Three weeks later, despite overall improvement, she complained of persistent and worsening fatigability, with difficulty climbing stairs and intermittent palpitations. Cardiopulmonary examination remained normal. The Nijmegen questionnaire was negative (20/64), while the Hospital Anxiety and Depression Scale showed scores of 12 (anxiety) and 11 (depression). Follow-up investigations showed normalization of laboratory parameters except for mild persistent lymphopenia (1,100/mm³) (table 1). Arterial blood gases, spirometry, electrocardiogram, and transthoracic echocardiography were normal. The 6-minute walk test revealed a reduced performance (37% of predicted), with early termination at 3 minutes due to lower limb muscle weakness. Follow-up chest CT showed complete resolution of pulmonary lesions and a normal thymic region.

Table 1. Evolution of biological, respiratory, and cardiac functional parameters between the acute phase of COVID-19 infection and the post-COVID-19 phase.

Examination	Parameter	Unit	Reference values	Acute phase (COVID-19 infection)	Post-COVID phase (3 weeks later)
Hematology	WBC	$10^3/mm^3$	04 -10	15	7.8
	Lymphocytes	$10^3/mm^3$	1.5 - 4	0,97 (lymphopenia)	1,1 (mild lymphopenia)
	RBC	$10^6/mm^3$	3,6-5,8	3.98	4,8
	Hb	g/dl	12-16	13,2	12,9
	Plateles	$10^3/mm^3$	150 - 400	97 000 (thrombo-cytopenia)	129 000
	AST	UI/L	< 40	89	39
	ALT	UI/L	< 40	98	40
	LDH	UI/L	< 480	685 (elevated)	356
	D-dimers	ng/mL	< 500	600 (slightly elevated)	430
	Urea	g/l	0,15-0,45	0,29	0,30
	Creatinine	mg/l	07-14	12	12
	Glucose	g/dl	0,70-1,10	1,00	0,86
Arterial blood gases	ph		7,35-7,45	NF	7.38
	PaO_2	mmHg	> 85	NF	93,4
	$PaCO_2$	mmHg	35-45	NF	42,3
	HCO_3^-	mmol/L	22-28	NF	24,9
	SaO_2	%	95-100	NF	98,1
Spirometry	FVC	L	3,56	NF	3,56 (108% predicted)
	FEV_1	L/s	3,29	NF	3,63 (106% predicted)
	TI =(FEV_1/FVC)	%	83	NF	104
Functional test	6MWT distance	meters	688,76	NF	254,8 (37% predicted)

WBC: white blood cells; RBC: red blood cells; Hb: hemoglobin; AST: aspartate aminotransferase; ALT: alanine aminotransferase; LDH: lactate dehydrogenase; PaO_2: arterial partial pressure of oxygen; $PaCO_2$: arterial partial pressure of carbon dioxide; HCO_3^-: bicarbonates; SaO_2: arterial oxygen saturation; FVC: forced vital capacity; FEV_1: forced expiratory volume in one second; TI: Tiffeneau index (FEV_1/FVC); 6MWT: six-minute walk test; NF: not performed.

Electromyography demonstrated impaired neuromuscular transmission, and anti–acetylcholine receptor antibodies were positive, supporting the diagnosis of myasthenia gravis. Repetitive nerve stimulation further supported this diagnosis; however, some technical parameters, particularly the stimulation frequency and the exact decrement percentage, were not available in the archived electrophysiological report. MG was diagnosed. The patient was treated with pyridostigmine followed by two courses of intravenous

immunoglobulins, with marked clinical improvement. Exercise tolerance improved (6-minute walk distance: 70% predicted). She subsequently underwent prophylactic thymectomy without complications. The patient remained asymptomatic with sustained remission at 12-month follow-up.

3. DISCUSSION

Available data on post–COVID-19 myasthenia gravis (MG) remain limited and are largely derived from isolated case reports. SARS-CoV-2 is increasingly recognized as a potential trigger or exacerbating factor for autoimmune diseases (4), particularly in genetically predisposed individuals. Proposed mechanisms include molecular mimicry between viral proteins, especially the spike protein and self-antigens, leading to activation of autoreactive T and B cells and loss of immune tolerance (5,6). In addition, the infection induces a systemic inflammatory response with cytokine release, promoting polyclonal lymphocyte activation (bystander activation) (7), exposure of self-antigens through tissue damage, and epitope spreading (8). Persistent immune stimulation may further promote chronic autoimmunity in susceptible individuals (9).

Clinical observations have reported the emergence of de novo autoimmune diseases following COVID-19, including systemic lupus erythematosus, antiphospholipid syndrome (10), multiple sclerosis, Guillain–Barré syndrome (11), rheumatoid arthritis, Miller Fisher syndrome (12), Kawasaki disease (13), and more recently MG (14,15).

In our case, the patient had a family history of Sjögren's syndrome. Although personal or familial autoimmune history is inconsistently reported in post-COVID MG (16,17), these findings suggest a possible immunogenetic susceptibility. Shared HLA haplotypes, particularly HLA-DR3 and HLA-B8, have been associated with both Sjögren syndrome and MG, supporting the hypothesis that SARS-CoV-2 may trigger MG in genetically predisposed individuals. This is consistent with epidemiological data demonstrating a hereditary contribution to MG (18), with up to 30% of patients reporting a family history of autoimmune disease, often through the maternal lineage (19). Overall, these observations support a multifactorial predisposition involving interactions between genetic background and environmental triggers such as viral infections.

Data on treatments administered during acute COVID-19 are often incomplete, limiting assessment of potential iatrogenic contributors. Some drugs may exacerbate MG; notably, azithromycin has been associated with worsening or triggering myasthenic symptoms (20, 21). In our case, azithromycin was not used; the patient received amoxicillin–clavulanate, which has no known myasthenic effect. These elements support a role of SARS-CoV-2 in breaking immune tolerance, particularly in predisposed individuals, and highlight the need for close post-COVID follow-up, especially in patients with autoimmune backgrounds.

Published cases suggest a predominance of generalized MG after SARS-CoV-2 infection (22) (23-26), whereas purely ocular or oculobulbar forms are less frequent and may secondarily generalize (16, 27-30). Some cases show rapid progression from an initial ocular presentation to generalized disease (22,31,32). In contrast, severe myasthenic crises requiring mechanical ventilation and intensive care remain rare (31,33). In our patient, the absence of ptosis, diplopia, or dysphagia initially delayed diagnosis. Fluctuating limb weakness was initially attributed to post-COVID conditions, including post-viral fatigue syndrome (34), hyperventilation syndrome (excluded), or critical illness myopathy (unlikely). The fluctuating, fatigable nature of weakness ultimately suggested MG, confirmed by electrophysiology and anti-AChR antibody positivity.

The interval between COVID-19 and MG onset varies widely (5-120 days; mean ~28 days) (17, 22), consistent with a post-infectious immune mechanism. Our patient presented early (day 5), within the reported range. Shorter delays are often observed after mild infections, whereas longer latencies (>60 days) have been reported after severe COVID-19, possibly influenced by immunomodulatory treatments or diagnostic delays (17,33).

Thymic imaging is essential to exclude thymoma, with chest CT as the first-line modality (35). However, thymoma remains relatively uncommon and is more frequent in older patients and generalized MG (36). In our case, repeated imaging showed no thymic abnormality. Notably, normal imaging does not exclude MG, particularly in seropositive patients. Electrophysiological studies remain central: repetitive nerve stimulation is frequently abnormal, and single-fiber electromyography is the most sensitive test. In our case, electrophysiology confirmed impaired neuromuscular transmission.

Serologically, anti-AChR antibodies are detected in 85% of generalized MG and 50% of ocular forms (37), while anti-MuSK antibodies are found in 40% of AChR-negative generalized MG (38). MuSK-positive MG is typically more severe (39), although post-COVID cases may show milder courses. Anti-AChR antibodies predominate in reported post-COVID MG cases, as in our patient, with rare MuSK-positive or double-seropositive forms (32,40,41). Triple-seronegative MG remains rare and diagnostically challenging (42).

Mortality in MG has been estimated at ~3.5% at one year, higher in older patients and males (43). Among patients with pre-existing MG and COVID-19, mortality ranges from 4.9% to 11% (44,45), mainly related to respiratory complications and comorbidities rather

than MG itself (46). Immunosuppressive therapies, particularly corticosteroids, azathioprine, and rituximab, may worsen outcomes (47). In contrast, no deaths have been reported in newly diagnosed post-COVID MG, which appears to follow a more favorable course, even in cases with myasthenic crisis.

Treatment strategies include pyridostigmine, intravenous immunoglobulins, corticosteroids, and thymectomy when indicated, with generally favorable outcomes (33). Thymectomy is recommended in generalized AChR-positive MG and has demonstrated long-term benefit (48).

Our case illustrates this profile, with insidious onset, no respiratory failure, rapid response to IVIG, and favorable outcome after thymectomy. Prospective longitudinal studies are needed to better define the natural history, optimize management, and identify predictors of outcome. Long-term follow-up will be essential to assess remission durability and relapse risk.

4. CONCLUSION

The present case supports the growing evidence that SARS-CoV-2 infection may precipitate or unmask autoimmune neuromuscular disorders such as MG in predisposed individuals. The very early onset after infection, association with familial autoimmunity, and favorable response to conventional MG therapies suggest a distinct post-infectious phenotype. Clinicians should maintain a high index of suspicion in patients presenting with persistent fluctuating weakness after COVID-19, particularly when symptoms are initially attributed to post-viral fatigue or psychological distress. Early diagnosis and multidisciplinary management remain essential to optimize outcomes and prevent delayed treatment.

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