

Post-COVID-19 acquired myasthenia gravis: A review of reported cases

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ABSTRACT

Introduction: The emergence of cases of myasthenia gravis (MG) following SARS-CoV-2 infection suggests a potential triggering role of COVID-19 in the onset of this autoimmune disorder.

Objective: This literature review aims to synthesize the available evidence on the clinical, paraclinical, therapeutic, and outcome features of post-COVID MG to better characterize this emerging nosological entity.

Literature Review: Reported cases of MG occurring after COVID-19 describe both localized and generalized forms, typically appearing within weeks after infection. Autoimmune testing frequently reveals antibodies against acetylcholine receptors (anti-AChR), suggesting a virus-triggered or virus-amplified autoimmune mechanism. Patients present with variable degrees of muscle fatigability, ptosis, diplopia, or generalized weakness, independent of the initial severity of COVID-19. Management follows standard MG treatment protocols, including cholinesterase inhibitors, immunotherapy, and, in selected cases, thymectomy, with generally favorable outcomes when diagnosis is timely and treatment is appropriate.

Conclusion: Post-COVID myasthenia appears to be an emerging clinical entity, potentially distinct in its demographic, immunological, and outcome characteristics. Early recognition of this syndrome is crucial for initiating appropriate therapy, particularly in patients experiencing persistent fatigue after SARS-CoV-2 infection. More studies are needed to elucidate underlying mechanisms and optimize management strategies.

Keywords: Myasthenia gravis, COVID-19, Post-COVID fatigue, Autoimmunity, Anti-AChR antibodies.

INTRODUCTION

Myasthenia gravis (MG) is an autoimmune disorder of the neuromuscular junction characterized by fluctuating skeletal muscle weakness resulting from autoantibodies targeting postsynaptic structures, most commonly the acetylcholine receptor (AChR), but also muscle-specific kinase (MuSK) and low-density lipoprotein receptor–related protein 4 (LRP4).^{1,2} Additional autoantigens, including agrin, titin, and ryanodine

receptors, have also been described, highlighting the immunological heterogeneity of the disease. This serological diversity, together with variability in clinical presentation (ocular versus generalized forms), age at onset, and thymic abnormalities, underpins the multi-dimensional classification of MG.

Since the emergence of the COVID-19 pandemic in 2019,³ increasing attention has been directed toward the relationship between viral infections and autoimmune diseases. A large retrospective cohort study involving more than 3–8 million individuals in the United States demonstrated a significantly increased risk of several autoimmune conditions following SARS-CoV-2 infection.⁴ Concurrently, multiple reports have described newly diagnosed cases of MG after

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COVID-19, suggesting that viral infection may act as a trigger or unmasking factor for an autoimmune response targeting the neuromuscular junction.

In this context, we conducted a literature review focusing on post-COVID myasthenia gravis, synthesizing data from published reports. The aim of this analysis was to examine the clinical, serological, and radiological features particularly thymic status as well as the clinical course of these post-infectious cases. This synthesis seeks to determine whether a distinct clinical phenotype, specific immunological profiles, or differences in therapeutic response may characterise this emerging subgroup of MG.

GENERAL OVERVIEW

Available evidence on post-COVID-19 myasthenia gravis remains limited and is largely derived from case reports and small case series. To ensure the reliability and comparability of the analysed data, strict inclusion criteria were applied, restricting the analysis to newly diagnosed MG cases occurring after a confirmed SARS-CoV-2 infection (table 1).^{5–21} This review highlights several key epidemiological, clinical, diagnostic, and outcome-related characteristics of post-COVID-19 MG.

Published cases of post-COVID-19 myasthenia gravis (MG) demonstrate a global distribution, with reports from Europe, Asia, and North America,^{5–21} indicating that the association with SARS-CoV-2 is not confined to a specific region or viral variant. No clear sex predominance is apparent. Patient ages range from 19 to 83 years;^{15,16} men generally align with classical MG epidemiology (60–70 years), whereas affected women tend to be older than expected for early-onset forms.^{17–19}

All cases confirmed SARS-CoV-2 infection by RT-PCR or serology, supporting a post-infectious autoimmune mechanism. However, assessment of COVID-19 severity is limited by heterogeneous reporting; disease is often described qualitatively as “mild” “moderate” or “severe” without standardized clinical metrics. Thoracic imaging is inconsistently documented, with reported findings typically including ground-glass opacities.^{6,7,14,15,20,21} The lack of quantitative evaluation of pulmonary involvement limits the ability to explore

correlations between systemic inflammation and neuromuscular autoimmunity.

SARS-CoV-2 AND THE ONSET OF MYASTHENIA GRAVIS: EVIDENCE OF ASSOCIATION

Viral infections are established triggers of autoimmunity,²² and SARS-CoV-2 has emerged as a potential initiator or exacerbator of autoimmune disorders,²³ particularly in genetically susceptible individuals.²⁴ Proposed mechanisms include molecular mimicry between viral proteins especially the spike protein and self-antigens, leading to activation of autoreactive T and B lymphocytes;^{25–26} a systemic inflammatory response with cytokine release promoting bystander activation of autoreactive lymphocytes;²⁷ tissue damage releasing self-antigens and driving epitope spreading;²⁸ and persistent viral stimulation predisposing to chronic autoimmunity.²⁹

Beyond exacerbations of pre-existing disease, de novo autoimmune disorders have been reported post-COVID-19, including systemic lupus erythematosus, antiphospholipid syndrome,³⁰ multiple sclerosis, Guillain-Barré syndrome,³¹ rheumatoid arthritis, Miller Fisher syndrome,³² Kawasaki disease,³³ and recently, myasthenia gravis.^{34,35} Personal or family histories of autoimmunity were infrequently documented in post-COVID MG cases,¹⁸ limiting assessment of baseline susceptibility. Nonetheless, epidemiological studies support a genetic contribution: in a North American multicenter cohort, 5.6% of MG patients reported a family history of MG, and >20% reported a first-degree relative with another autoimmune disease, especially in early-onset forms.³⁶ Earlier reports also demonstrated familial clustering and HLA associations (HLA-B8, HLA-DR3).³⁷

Data on vaccination status are sparse. Post-vaccination MG, mostly after mRNA vaccines, generally presents with rapid, often generalized onset and responds well to immunomodulatory therapy.^{38–40} These events do not compromise the overall benefit-risk profile of COVID-19 vaccination but warrant vigilance in patients with known or suspected autoimmune predisposition. Incomplete reporting of acute COVID-19 treatments further limits evaluation of iatrogenic contributors. Azithromycin, widely used

Table 1. Clinical, Diagnostic, and Outcome Characteristics of Newly Diagnosed Myasthenia Gravis Following Covid-19 Infection

Reference	Sex	Age	Delay (days)	Clinical Presentation	Clinical Form	Auto Antibodies	RNS / SFEMG	Thymus CT	Treatment	Outcome
Huber et al., 2020 (5), Germany	F	21	~14	Ptosis, diplopia, dysarthria	Ocular initially, then progressive	Anti-AChR +	Normal RNS	NR	IVIg + pyridostigmine + thymectomy	Improvement
Restivo et al., 2020 (6), Italy – Case 1	M	64	5	Diplopia followed by generalized weakness	Ocular then generalized	Anti-AChR +	Facial/ulnar decrement >50%	Normal	Pyridostigmine + corticosteroids	Improvement
Restivo et al., 2020 (6), Italy – Case 2	M	58	7	Diplopia, dysphagia, generalized weakness	Generalized	Anti-AChR +	Facial and ulnar decrement 52% / 21%	Normal	IVIg	Partial remission
Restivo et al., 2020 (6), Italy – Case 3	F	71	5	Ptosis, diplopia, then acute respiratory failure	Severe generalized	Anti-AChR +	Ulnar decrement 56%	Normal	Plasmapheresis	Improvement after extubation
Sriwastava et al., 2021 (7), India	F	65	11	Bilateral ptosis, diplopia	Ocular	Anti-AChR +	RNS positive (>10%)	Normal	NR	Improvement
Karimi et al., 2021 (8), Iran – Case 1	M	61	42	Ptosis, diplopia, dysphagia, dysphonia, proximal weakness, dyspnea	Generalized	Anti-AChR +	decrement 10–40%	Thymoma	Pyridostigmine + corticosteroids + plasmapheresis; planned thymectomy	Improvement
Karimi et al., 2021 (8), Iran – Case 2	M	57	7	Ptosis, diplopia, dysphagia, generalized fatigue	Generalized	Anti-AChR +	NR	Normal	Pyridostigmine + corticosteroids	Improvement
Karimi et al., 2021 (8), Iran – Case 3	F	38	28	Ptosis, diplopia, dysphagia, fatigue	Generalized	Anti-AChR +	NR	Normal	Pyridostigmine + corticosteroids	Improvement
Bhandarwar et al., 2021 (9), India	M	61	60	Ptosis, diplopia, dysphagia, fatigue	Generalized	Anti-AChR +	NR	NR	Pyridostigmine + plasmapheresis + thymectomy	Improvement
Muhammed et al., 2021 (10), UK	F	24	28	Bilateral ptosis, diplopia, dysphagia, muscle weakness	Localized then generalized	Anti-AChR – / Anti-MuSK +	RNS +; SFEMG +	Normal	NR	Improvement
Assini et al., 2021 (11), Italy	M	77	56	Ptosis, diplopia, dysphagia, dysphonia	Oculobulbar	Anti-AChR – / Anti-MuSK +	RNS +	Normal	NR	Improvement
Muralidhar Reddy et al., 2021 (12), India	M	65	42	Generalized weakness, dysphagia	Generalized	Anti-AChR +	Facial/ accessory decrement	Normal	IVIg + prednisolone + pyridostigmine	Improvement

(continued)

Table 1. Clinical, Diagnostic, and Outcome Characteristics of Newly Diagnosed Myasthenia Gravis Following Covid-19 Infection (Continued)

Reference	Sex	Age (years)	Delay (days)	Clinical Presentation	Clinical Form	Auto Antibodies	RNS / SFEMG	Thymus CT	Treatment	Outcome
Jögi et al., 2022 (13), Estonia	M	75	30	Respiratory weakness followed by diffuse motor weakness	Generalized	Anti-AChR +	RNS + ; SFEMG +	Normal	NR	Improvement
Taheri et al., 2022 (14), Iran	F	35	15	Bilateral ptosis and diplopia	Ocular	Anti-AChR +	SFEMG +	Normal	Pyridostigmine	Recovery
Chatterjee et al., 2022 (15), India	M	83	30	Muscle weakness, dyspnea, dysphonia	Generalized	Anti-AChR +	RNS + (10–40%)	Normal	Pyridostigmine + corticosteroids	Improvement
Tereshko et al., 2023 (16), Italy	F	19	13	Oculobulbar symptoms progressing to generalized MG and myasthenic crisis	Localized then generalized	Anti-AChR +	RNS + ; SFEMG +	Normal	Thymectomy	Improvement
Popescu et al., 2023 (17), France	F	77	5	Unilateral ptosis	Ocular	Anti-AChR +	orbicularis RNS normal	Normal	NR	Recovery
De Giglio et al., 2023 (18), Italy	M	74	~30	Severe diplopia	Ocular + Graves' disease	Anti-AChR + ; Anti-TSHR +	NR	NR	NR	Improvement
Hiraoka et al., 2025 (19), Japan	F	78	14–30	Neck and upper limb weakness	Generalized	Anti-AChR + ; Anti-MuSK +	RNS decrement ~20%	NR	IVIg + corticosteroids + tacrolimus	Improvement
Minca et al., 2024 (20), Romania	F	30	NR	Myasthenic crisis	Severe generalized	Anti-AChR +	NR	Normal	Pyridostigmine + corticosteroids	Improvement
Feiz et al., 2025 (21), USA	M	81	~120	Dysarthria, diplopia, dyspnea, oropharyngeal dysphagia	Generalized	Anti-AChR +	NR	Normal	Pyridostigmine + corticosteroids + IVIG	Improvement

AChR: acetylcholine receptor; CTS: corticosteroids; IVIG: intravenous immunoglobulins; MuSK: muscle-specific kinase; NR: not reported; MG: myasthenia gravis; CT: computed tomography; RNS: repetitive nerve stimulation; SFEMG: single-fiber electromyography.

early in the pandemic, is recognized for exacerbating pre-existing MG and triggering myasthenic crises.^{41,42}

Taken together, current evidence supports that SARS-CoV-2 infection can trigger or unmask MG in genetically predisposed individuals through molecular mimicry, systemic inflammation, and possibly treatment-related factors. These findings underscore the need for careful post-COVID clinical monitoring, particularly in patients with personal or familial autoimmune risk, irrespective of vaccination status.

POST-SARS-CoV-2 MYASTHENIA GRAVIS: PHENOTYPE AND MANAGEMENT

Analysis of reported cases of post-SARS-CoV-2 myasthenia gravis (MG) reveals a predominance of generalized forms, often presenting without an initial ocular phase.^{6,8,9,12,13} Purely ocular forms, limited to ptosis and/or diplopia, are infrequent,^{5,7,14,17,18} although some cases show secondary generalization,^{6,10,16} oculo-bulbar involvement,¹¹ or acute myasthenic crises requiring intensive care.^{16,20} Early absence of classic signs such as ptosis, diplopia, or dysphagia complicates diagnosis, as weakness is frequently attributed to post-viral fatigue, hyperventilation syndrome, or critical illness myopathy.⁴³⁻⁴⁴ Recognition of fluctuating, exertion-dependent weakness remains key to suspecting MG.

The latency between COVID-19 infection and MG onset varies from days to months,^{6,21} consistent with a post-infectious immunological mechanism. Women tend to develop MG earlier, whereas men exhibit more variable and sometimes delayed onset,^{6,21} possibly reflecting sex-specific differences in post-viral immune responses. Younger patients also present earlier, whereas older individuals show prolonged latency, potentially due to immunosenescence or diagnostic delay.

Disease severity appears influenced by initial COVID-19 severity and treatment. Mild ambulatory COVID-19 often precedes MG with shorter latency,¹⁶ whereas severe cases, especially those receiving immunomodulators, may demonstrate delayed onset.¹⁵ Longer latency correlates with generalized and more severe MG, predominantly in older men,²¹ contrasting with early-onset, mild ocular forms typically seen in younger women.

Thymic assessment using thoracic computed tomography (CT) or magnetic resonance imaging (MRI) remains central to the etiological workup.⁴⁵ Overall, the incidence of thymoma in myasthenia gravis is low, according to the literature,⁴⁶ and thymomas are rarely observed in post-COVID-19 myasthenia.^{8,9} MRI may enhance detection, providing higher sensitivity and specificity compared with CT.⁴⁷

Electrophysiological studies, including repetitive nerve stimulation (RNS) and single-fiber electromyography (SFEMG), are essential diagnostic tools. Autoantibody profiling remains a cornerstone: anti-AChR antibodies are common (85% in generalized forms), while anti-MuSK antibodies occur in 40% of anti-AChR-negative generalized cases.⁴⁸⁻⁵⁰ Post-COVID MG cases largely mirror these patterns, though favorable outcomes have been observed in some MuSK-positive patients.^{10,11} Rare triple-seronegative forms continue to pose diagnostic challenges.⁵¹

Treatment mirrors standard MG management, combining pyridostigmine, intravenous immunoglobulins (IVIG), corticosteroids,^{52,53} and thymectomy when indicated.⁵⁴ Notably, post-COVID MG appears to have a more favorable prognosis than idiopathic forms, particularly when diagnosed early and managed promptly.

Overall, post-COVID MG constitutes a distinct clinical phenotype, characterized by rapid generalization, variable latency, and generally good response to established therapies. Early recognition, serological assessment, and personalized management are critical to optimize outcomes in this emerging post-infectious entity.

CLINICAL IMPLICATIONS AND FUTURE PERSPECTIVES

The review highlights several important considerations for clinical practice. First, the establishment of standardized clinical registries is essential to investigate risk factors, the potential contribution of COVID-19 vaccination or therapies, and to clarify underlying immunopathogenic mechanisms. Early recognition of post-COVID-19 myasthenia gravis (MG) requires heightened clinician awareness, as diagnostic delays are common due to overlap with post-viral fatigue. A systematic diagnostic approach including

electrophysiological studies and serological testing is therefore critical.

Second, the frequent absence of thymic abnormalities challenges conventional diagnostic algorithms, emphasizing the central role of autoantibody profiling in this context. The generally favorable prognosis observed in reported cases suggests that post-COVID MG may constitute a distinct subgroup with unique clinical and immunological characteristics.

Finally, prospective longitudinal studies are needed to delineate the natural history of post-COVID MG, refine diagnostic and therapeutic strategies, and identify predictors of favorable outcomes. Long-term follow-up will be crucial to evaluate remission durability, the risk of relapse, and to guide personalized management strategies in this emerging post-infectious autoimmune entity.

CONCLUSION

The COVID-19 pandemic has reshaped diagnostic approaches by unmasking latent autoimmune disorders. Although rare, myasthenia gravis (MG) should be considered in the differential diagnosis of unexplained post-viral fatigue. Initial COVID-19 severity does not reliably predict subsequent MG, which may develop even after mild or ambulatory infection. Overall prognosis is generally favorable when diagnosis is prompt and management appropriate. Diagnostic delay remains a major clinical challenge, particularly given the heterogeneity of presentations and potential differential diagnoses.

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