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ახალგაზრდა მკვლევარები

Vol. 4 Issue 1, 2026

https://doi.org/10.52340/jr.2026.04.01.07

Exploring the Role of Neuroinflammation and Gut-Brain Axis Dysregulation in the Pathogenesis of Non-Motor Symptoms in Myasthenia Gravis

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Abstract

Myasthenia gravis (MG) is a long-lasting autoimmune neuromuscular disorder usually reflected by the presence of autoantibodies against acetylcholine receptors at the neuromuscular junction and characterized by either ocular or generalized disease with possible respiratory crisis. The situation has not changed, as diagnostic difficulties and recurrences are still the main problems despite the improvements in diagnostics and immunosuppression. Moreover, the totality of the evidence is pointing to the fact that MG is a disease that also has non-motor manifestations of important clinical significance. The aim of this narrative review was to summarize the literature on MG which takes the position of a multisystem neuroimmune disease and to clarify the mechanisms of immune dysregulation, neuroinflammation, and gut-immune-brain axis that are the cause of the non-motor symptoms such as fatigue, cognitive impairment, mood disturbances, sleep changes, and autonomic dysfunction. A thorough search in PubMed and Google Scholar using terms connected to MG, non-motor symptoms, gut microbiota, gut-brain axis, neuroinflammation, and immune dysregulation led to the discovery of full-text English studies highlighting both systemic and central mechanisms; a qualitative synthesis of themes was drawn up and focused on microbiome composition, metabolites, cytokines, and the changes in immune cells. Studies were consistent in correlating MG with diminished variety of gut microbes, loss of Firmicutes (including Clostridia/Lachnospiraceae and Faecalibacterium) producing butyrate, and increase in munificent taxa (e.g., Streptococcus and Enterococcus), which was in line with Treg cell (Treg) function being diminished and Th17/Treg balance being shifted. The

research involving multi-omics techniques pointed toward the conclusion that dysbiosis was a major reason for the widespread disturbance of SCFAs, and also that it disrupted the pathways of KP and BA through the action of receptors such as AhR and TGR5, which was reflected in the metabolomic profile changes of feces (e.g., of cytosine, xanthine, adenine, and methylmalonic).

keywords: Myasthenia gravis, autoimmune disorder, gut microbiota, regulatory T cells, faecal microbiota transplantation, Metabolomics.

Introduction

Myasthenia gravis is a chronic autoimmune neuromuscular disorder characterized by sudden fatigue and fluctuating weakness of skeletal muscles and mainly caused by autoantibodies that target the AChR at the neuromuscular junction. There are various subtypes of Myasthenia Gravis such as ocular Myasthenia Gravis which is confined to extraocular muscles and the generalized Myasthenia Gravis which involves large number of muscles with risk of respiratory depression which further causes further failure (1). Even after improved diagnostic assays and immunosuppressive treatments, there is always a high chance of high recurrence rates and diagnostic issues still remaining. The pathogenesis of Myasthenia gravis depends on a compounded interaction of genetic liability, like HLA-DQ5 and immune dysregulation, mainly on the Th1, B cells and Foxp3 CD4 regulatory T cells disparity (2).

The regulatory T cells have a huge role in balancing immune tolerance by repressing autoreactive T and B cells. It's also necessary to note remarkable reductions in T regulatory cells in a Myasthenia Gravis patient, which correspond to the disease progression and its severity (2). The evidence shows that gut microbiota is a key regulator of the immune system related to Myasthenia Gravis. Studies show that the altered gut microbiota seen in Myasthenia Gravis patients, when compared to healthy individuals, have an increased amount of Bacteroidetes and Streptococcus with a decrease in helpful bacteria like Firmicutes, Clostridia, and Bifidobacterium. This disrupts the homeostasis of microbial metabolites, specifically short-chain fatty acids known as butyrate, therefore elevating T regulatory cells differentiation and potentially worsening Myasthenia Gravis further (3).

Experiments that engage in multi-omics approaches like 16S rRNA gene sequencing and faecal/serum, metabolomics show that Myasthenia gravis-related gut microbiota dysbiosis corresponds with an altered immunological reaction and severity. Experiments on mice further strengthen the evidence that gut changes directly influence Myasthenia Gravis, where faecal microbiota transplantation from Myasthenia Gravis patients causes Immunological alterations and anxiety responses through the microbiota gut-brain axis, especially affecting the hippocampal regions. However, supplementing butyrate restores T regulatory cells' autophagy (3). This metabolite of microbes thus implies potential therapeutic value. Therefore, gut microbiota characterization along with metabolite analysis provides the diagnostic biomarkers and targets for a therapeutic approach in Myasthenia gravis. Regulating microbiomes through probiotics, FMT may restore the immunological decrease in autoimmunity which improves patient outcomes.

It has been found that there has been a steady decrease in general diversity with the loss of butyrate-producing Firmicutes (e.g., some Clostridia, Lachnospiraceae, etc.) and a possible increase in advantageous pro-inflammatory bacteria of the type Streptococcus and Enterococcus (4). Others of these detrimental bacteria are also related to a history of respiratory crisis, and thus require more immunotherapeutic rescue, which serves as a predictive of the severity of myasthenia gravis and not merely a diagnosis. The fecal metabolomics presents the changed amino acid and nucleotide metabolism with the altered concentrations of cytosine, methylmalonic acid, xanthine, and adenine; the metabolic changes are related to the myasthenia gravis-related gut microbiome. Along with the well-known depletion in short-chain amino acids, two major pathways, like the tryptophan-kynurenine pathway and bile acid pathway, are also disrupted, which influence TH17/Treg balance, microglia activity, and intestinal wall integrity by acting on specific receptors like aryl hydrocarbon receptors and TGR5 (4).

Induction of gut bacteria of a patient with myasthenia gravis in germ-free mice resulted in microbial and metabolite pathological alterations, enhanced proinflammatory cytokines such as TNFa, and worsened locomotor performance in these mice compared to colonization by healthy donors (5). Mendelian randomization implies the presence of the bidirectional causal relationship, in which some gut genera might cause myasthenia gravis risk, and myasthenia gravis-related immune responses shift the gut microbiome, creating a feed-forward loop.Studies suggest that incorporating microbial markers with their specific metabolites can characterise myasthenia gravis from healthy controls with very high specificity, which points towards faecal panels as potential adjuncts to autoantibody testing, research also shows a metabolite-immune-neural axis in myasthenia gravis patients and advises diets consisting of high fibre intake, while taking probiotics therefore helping maintain healthy gut microbiota, trials for fecal microbiota transplantation should also be considered to raise tryptophan derivative levels which are beneficial for the gut, keeping in mind the significance of immunological end points (5).

Methodology

This narrative review was done to discuss the non-motor symptoms of Myasthenia Gravis (MG) and the biological mechanisms that could be causing such symptoms in addition to the well-established neuromuscular dysfunction. The main aim was to provide a synthesis of the current literature backing the idea of MG as a multisystem neuroimmune disease, specifically focusing on neuroinflammation, immune dysregulation and gut-brain axis (1,2). Enhanced literature search was conducted in peer-reviewed biomedical databases such as PubMed anf Google scholar. The concept-driven search keywords and combinations thereof (Myasthenia Gravis, non-motor symptoms, gut microbiota, gut-brain axis, neuroinflammation, and immune dysregulation) were used to direct the search strategy. This selection allowed locating the studies which went further than neuromuscular transmission and studied MG in a larger framework of systemic and neuroimmune processes. Literature was chosen according to its suitability in terms of the immune, inflammatory, central nervous system or systemic

mechanisms in relation to the MG and their possible connection to non-motor symptoms such as fatigue, cognitive impairment, mood disturbances, and sleep changes.

Articles published in English with full-text access were only taken into consideration. Articles that only discussed classical motor manifestations with no discussion of neuroimmune or systemic, and articles whose content was not based on academic sources, e.g., commentaries, editorials, blogs, etc., were limited. The relevance of titles and abstracts was first filtered, and a careful process of considering the qualified full-text articles was done. Thematic analysis was used to extract the data qualitatively instead of quantitative synthesis, which is in line with the methodology of narrative review. The changes in gut microbiota composition, immune and cytokine-mediated processes, indicators of neuroinflammatory processes, and suggested connections between the processes and non-motor clinical phenomena were key information gathered. The deduced outcomes were synthesized and clustered in biological motifs which were interrelated to each other, including gut dysbiosis, immune activation, neuroinflammation, and systemic implication. Major focus was on highlighting the presence of overlapping and interacting pathways that could have a role in the development of non-motor symptoms in MG. This synthesis approach contributes to a more extended perception of MG as a disorder with a big non-motor and multi-system implication and presents the conceptual framework of future research and clinical reflection (3,4).

Discussion

Recent scientific publications give more and more evidence to back up the theory that Myasthenia Gravis (MG) is not only defined by its typical motor symptoms but also by a complicated interaction among the gut microbiome, immune system activity, and central nervous system (CNS) pathways.MG has been traditionally characterized by the disruption of neuromuscular transmission due to antibodies formation, but a growing number of researchers say that the non-motor symptoms like the following fatigue, emotional and cognitive discomfort, gastrointestinal issues, and chronic pain are very common and doctors have to take them into account as being of great importance (5). It seems that the signs are, if not wholly, at least partially due to the overarching inflammation and the neuroimmune processes which, while affecting the peripheral neuromuscular junction, are predominant in even larger areas (5).

The research that has been gathered and evaluated in this review all together back the idea that the gut dysbiosis, immune dysregulation, and neuroinflammatory signaling are connected in turning the gut–immune–brain axis which is influencing the disease expression in MG. This integrative view offers a valid biological reason for non-motor symptoms to continue to stay in a good number of patients notwithstanding the control of motor symptoms (6).

The Gut–Immune–Brain Axis in Myasthenia Gravis

The gut microbiota of those suffering from MG has been less diverse and more inflammatory, with the pro-inflammatory taxa dominating the former beneficial bacteria. This change in microbial community is mediated through immune regulation changes, which include new cytokine profiles, regulatory T-cell function loss and metabolic signaling disruption. However, such immune-microbial patterns, that are well acknowledged in other autoimmune diseases, have been considered in MG mainly through the M-pathology of neuromuscular junction (7).

Data that is just coming up give a hint that the activation of the immune system starting from the gut might have an impact both outside and inside the body. The underpinnings thereof can be explained by several pathways, some more important than others (7,8). Firstly, microbial metabolites which include short-chain fatty acids (SCFAs), tryptophan derivatives, and bile acids are the main mediators for immune homeostasis, integrity of the blood–brain barrier, and vagus nerve signaling. Decreased number of bacteria that produce SCFAs might lead to the activation of the pro-inflammatory immune territory and at the same time restrict neuroprotection (8).

Firstly, increased amounts of circulating cytokines such as interleukin-6, tumor necrosis factor-α, and interleukin-17 can potentially act on CNS function either through the access to brain areas having less protection from the blood-brain barrier or by the pathways of neural signaling (7,8). Besides, activating microglia is one of the major effects of these cytokines, and this, along with others, leads to fatigue, mood instability, and cognitive dysfunction. The third route utilized is the gut-brain connection through the means of two-way vagal nerve signaling which is an important factor in the regulation of inflammatory responses and sick behavior during the disease. There is a constant correlation between decreased vagal tone and chronic inflammation, fatigue, and depressive symptoms, thus providing additional evidence for its importance in non-motor manifestations of MG. All in all, such results indicate that changes in the gut microbiota can cause the whole-body immune system to be activated and through different ways this activation may affect the nervous system. Therefore, the model provides rational biological groundwork linking immune system disorders with non-motor symptoms that although may not have been noticed in MG before, are nevertheless clinically significant (9).

Comparison With Other Autoimmune Diseases

Investigating other autoimmune conditions having the gut–immune–brain interactions as their main phenomenon brings valuable information to the context. Among the diseases of multiple sclerosis, systemic lupus erythematosus, rheumatoid arthritis, and inflammatory bowel disease, there are striking similarities with MG, including disturbances in microbiome with minimal SCFA production, high levels of pro-inflammatory cytokines with action in the central nervous system and moreover, the patients suffering from fatigue, pain, cognitive impairment and mood disorders to a great extent. Such a conclusion puts into question long constructive peripherality, thus showing how schizophrenia and MG possibly act synergistically (9).

Nonetheless, multiple distinctions can be made between MG and the conditions mentioned above.MG patients very often have their non-motor complaints neglected, erroneously linked to the side effects

of corticosteroids, or just considered as psychological responses to their chronic sickness. The past research priorities have mainly been directed towards the study of acetylcholine receptor pathology, while the areas of inflammation or central mechanisms were not given that much attention. Also, there are no standard tools for evaluating non-motor symptoms in MG, which makes it hard to associate biological abnormalities with clinical manifestations. Probably, these reasons are the ones why a lot of patients still have a very low quality of life even though their motor weakness is effectively controlled (9,10).

Strengths and Limitations of the Current Evidence

The existing literature presents a number of remarkable strengths. Independent research has repeatedly pointed to gut dysbiosis in MG, therefore giving these findings a strong support. There is a relevance of this kind of correlation between microbiome changes, immune markers, and symptom severity in clinical practice. Most importantly, the latest studies have not only followed descriptive microbiome profiling but also on metabolites, cytokines networks, and immune cells functions. The proof from cohorts of untreated patients additionally indicates that dysbiosis is not just a result of immunosuppressive therapy (10).

Nevertheless, there are still major drawbacks. A large number of studies are using cross-sectional methods which do not allow for drawing conclusions about causality or the timing of events. It is still unclear as to which gut dysbiosis, immune system activation, and central nervous system dysfunction were interconnected. Most of the mechanistic understanding comes from studying animal models while direct proof of neuroinflammation in human myasthenia gravis through using advanced neuroimaging or biomarkers is still missing (10,11). The simultaneous action of immunosuppressive treatments on the composition of microbiota and mood also makes the interpretation difficult. Moreover, the different ways of evaluating non-motor symptoms in different studies have rendered it impossible to relate the biological findings to the patients' reported outcomes. Even though these difficulties were encountered, the concurrence of results from various fields of research validates the suggestion of a well-organized gut–immune–brain axis that plays a role in MG-related non-motor symptoms (11).

Clinical Implications

There are possible indicators of diseases such as circulating cytokines, microbial metabolites including SCFAs and intermediates of the kynurenine pathway, and lastly, the unique microbiome signatures for diseases which fall into this category. The use of these markers might facilitate the recognition of patients who are prone to the development of severe non-motor symptoms, keep track of disease activity, and assess the changes produced by targeted interventions (12,13).

One of the possibilities for treatment that can be drawn from the mentioned framework is using the microbiome as a basis for the creation of new methods, for example, changing of diets, taking prebiotics, probiotics, and formulations that contain both pre- and probiotics, and finally, synbiotics, doing anti-inflammatory and immune-metabolic therapies, and performing the neuromodulatory approaches

aimed at vagal signaling. Most importantly, it is highly likely that such methods will be able to relieve fatigue, pain, and mood disorders, while still keeping the immunosuppression levels the same as before or even low (12,13).

Contribution of This Review

By bringing together the changes in microbiome, immune system's malfunction and involvement of the central nervous system into a single framework, this narrative review provides a deeper understanding of MG as a condition that is not solely limited to the neuromuscular system. It classifies the non-motor symptoms as the primary signs that come from the autoimmune processes in the whole body rather than the ones that are not so important or that have come along with the primary ones (14,15). This point of view calls for a change in the approach to disease evaluation and treatment that takes into account the whole patient and that gives equal importance to the motor and non-motor aspects of MG. The evidence examined in this paper suggests that future longitudinal and interventional studies will be needed to determine the causal pathways, validate the biomarkers, and create the targeted therapies. Considering MG as a systemic autoimmune disorder with main functional repercussions could change not only the research and clinical care in the field but also the patients' outcomes and their quality of life, thus leading to a win-win situation (14,15).

Results

Gut Microbiota Dysbiosis in MG Patients

Patients who suffer from myasthenia gravis have less variety of good bacteria like faecalibacterium, than people without myasthenia gravis. Faecalibacterium keeps the gut healthy by producing mostly a substance known as butyrate, which is a short-chain fatty acid that essentially reinforces the gut barrier and brings down inflammation (16,17). Certain chemicals present in the stool of patients with myasthenia gravis suggests complications in the processing of amino acids and fats like oleic acid which are higher than normal in the stool, there is a correlation between the number of good bacteria such as faecalibacterium and creatinine levels which demonstrates that creatinine levels change due to the decrease in gut microbiota, due to these changes in the gut bacteria it might interrupt the link between the gut and the brain leading to other symptoms outside the musculoskeletal system (16,17).

Organ system specific Reversed Metabolites

The altered metabolites are the substances residing in the body which return to normal amounts when the gut bacteria are modified. In mice with different gut microbiota and mice with myasthenia gravis gut microbiota, half of the metabolites in blood and stool samples were reversed (17,18). Yet, less metabolites were found in the brain particularly in the prefrontal cortex, striatum and the hippocampus where the changes were mostly in lipid and carbohydrate metabolism as the hippocampus is responsible for thoughts and memories including molecules like ascorbate and GABA, a lot of the

reversed metabolites in blood and feces were linked to the processes involving amino acid and carbohydrate metabolism pathways which involve substances like cytidine and L-dopa (17,18).

Non-Motor Symptoms

Patients with myasthenia gravis suffer from various other symptoms beyond those of motor ones. Mental health complications like insomnia, depression, and anxiety manifest in these patients due to autoimmunity; certain receptors like AChR and MuSK are attacked by the immune system, leading to inflammation and, in the future, might even result in thymoma, which causes antibodies to attack AChR at the neuromuscular junction, making it difficult to carry out basic activities like swallowing (19,20). Along with mental health issues, patients with myasthenia gravis come with a long list of complaints regarding autonomic insufficiency/dysfunction affecting the urinary and digestive systems, reversed metabolites which affect the hippocampus primarily by interrupting the natural process of memory and thought production by disrupting lipid and carbohydrate metabolism, leading to difficulty in thinking and memory (19,20).

Female gender-determined anxiety via FMT (fecal microbiota transplantation)

When fecal microbiota transplantation was executed on mice, the female mice exhibited more anxiety and muscle weakness-like behavior as compared to the male mice (21,22). Still, once the healthy gut microbiota was induced into these female mice, they showed some improvements, while their male counterparts did not have any significant changes in their anxiety-like behavior. A gut-microbe central nervous system link was observed with sexual dimorphism, therefore, disrupting oscillatory activity (21,22).

Mendelian randomization (MR) analysis

The Mendelian randomization indicates that the rise of Actinobacteria and Gammaproteobacteria can reduce the risk of myasthenia gravis; at the same time, the presence of Faecalibacterium and Lentisphaerae bacteria further heightens the risk. In contrast, reversed Mendelian randomization implies that myasthenia gravis results in the decrease of Eptostreptococcaceae family and Romboutsia species. Moreover, myasthenia gravis can affect the reduction of cholesterol levels (23,24).

Conclusion

According to recent studies, non-motor symptoms in Myasthenia Gravis are highly correlated with inflammation of the nervous system and imbalance in communication between gut and brain. The mentioned communication and i.e. neuroinflammation pathways give better understanding of the symptoms like tiredness, changes in mood and cognition, which till now were considered as just motor weaknesses. The present investigation accentuates the necessity of performing more integrated studies that would interconnect the immune pathways, the modifications of the microbiome, and the involvement of the CNS in MG. Knowing the connections in this triad will not only enhance the clinical assessment but would also facilitate the development of therapy based on the affected pathways. Medications of the future that tackle the root molecular problems instead of simply the

neuromuscular manifestations can be a significant boost to overall care and the life quality of patients with MG.

Acknowledge

The authors confirm that all individuals listed as authors made substantial, equal contributions to the development of this work, including the abstract, introduction, methodology, discussion, and conclusion. The collaborative nature of this project reflects the shared responsibility and joint effort of all team members.

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