Juvenile myasthenia gravis: a case series review

Miastenia Gravis Juvenil: revisão de série de casos

Lucas Garcia Marcelino[1], Flávia Nardes[2]

[1]Pediatrician, Resident Physician in Child Neurology at IPPMG/UFRJ, Rio de Janeiro/RJ - Brasil;
[2]MD, PhD, Child Neurologist at IPPMG/UFRJ, Assistant Professor at UFRJ Medical School, Rio de Janeiro/RJ - Brasil.

ABSTRACT

Juvenile myasthenia gravis (JMG) is an autoimmune disease of the neuromuscular junction that affects children and adolescents under 18 years of age and can pose life risk. The clinical characteristics in Brazilian children have been barely described. A descriptive and retrospective study was carried out on 2024 to outline the clinical profile of patients with JMG at a neuromuscular disease center in Rio de Janeiro. Among the 11 eligible patients, there was a predominance of females (72.7%), with initial clinical manifestations at 5 years old. The most commonly observed initial form of disease presentation was ocular (45,5%), followed by bulbar form (27,3%), myasthenic crisis (18,2%) and muscular form (9,1%). The most frequent signs and symptoms through follow-up were ptosis (100%), muscle weakness (54,5%), dysphagia (45,4%), dysphonia (18,1%) and facial paresis (18,1%). Patients with ocular-onset disease experienced no disease progression, remaining with strictly ocular symptoms. In contrast, those with bulbar-onset developed limb weakness, and the single muscular-onset case developed ocular involvement, as all patients did at some point. Most patients had positive anti-acetylcholine receptor antibodies (72,7%) and none had anti-Musk antibodies. All patients used a cholinesterase inhibitor and only one (9%) did not require associated corticosteroids for disease improvement. Six patients (54.5%) had never had a myasthenic crisis, while 5 patients (45.5%) had at least one. Among those who did, all responded to human immunoglobulin and corticosteroid pulse therapy. Approximately one third (36,3%) required other immunosuppressants to control exacerbations. The majority have a normal thymus (72,7%), with thymectomy indicated for only one patient. Asthma is the most prevalent autoimmune comorbidity (27,2%), as is anxiety as a neuropsychiatric comorbidity (27,2%). At our neuromuscular disease center in Brazil, our patient profile is very similar to the descriptions made so far of patients with JMG.

Key-words: Myasthenia Gravis; Child Care; Cholinesterase Inhibitors; Intravenous Immunoglobulin; Thymectomy.

RESUMO

A miastenia gravis juvenil (MGJ) é uma doença autoimune da junção neuromuscular que afeta crianças e adolescentes menores de 18 anos e pode representar risco de vida. As características clínicas em crianças brasileiras são pouco descritas. Um estudo descritivo e retrospectivo foi realizado em 2024 para traçar o perfil clínico de pacientes com MGJ em um centro de doenças neuromusculares no Rio de Janeiro. Entre os 11 pacientes elegíveis, houve predomínio do sexo feminino (72,7%), com manifestações clínicas iniciais aos 5 anos de idade. A forma inicial de apresentação da doença mais comumente observada foi a ocular (45,5%), seguida da forma bulbar (27,3%), crise miastênica (18,2%) e forma muscular (9,1%). Os sinais e sintomas mais frequentes durante o acompanhamento foram ptose palpebral (100%), fraqueza muscular (54,5%), disfagia (45,4%), disfonia (18,1%) e paresia facial (18,1%). Pacientes com forma de de início ocular não apresentaram progressão da doença, permanecendo com sintomas estritamente oculares. Em contraste, aqueles com início bulbar desenvolveram fraqueza nos membros, e o único caso de início muscular desenvolveu envolvimento ocular, como todos os pacientes em algum momento. A maioria dos pacientes apresentou anticorpos antirreceptor de acetilcolina positivos (72,7%) e nenhum apresentou anticorpos anti-Musk. Todos os pacientes usaram um inibidor de colinesterase e apenas um (9%) não necessitou de corticosteroides associados para melhora da doença. Seis pacientes (54,5%) nunca tiveram uma crise miastênica, enquanto 5 pacientes (45,5%) tiveram pelo menos uma. Entre aqueles que tiveram, todos responderam à imunoglobulina humana e pulsoterapia com corticosteroides. Aproximadamente um terço (36,3%) necessitou de outros imunossupressores para controlar as exacerbações. A maioria tem timo normal (72,7%), com timectomia indicada para apenas um paciente. A asma é a comorbidade autoimune mais prevalente (27,2%), assim como a ansiedade como comorbidade neuropsiquiátrica (27,2%). Em nosso centro de doenças neuromusculares no Brasil, nosso perfil de pacientes é muito semelhante às descrições feitas até agora de pacientes com JMG.

Palavras-chave: Miastenia Gravis; Cuidado da Criança; Inibidores da Colinesterase; Imunoglobulina intravenosa; Timectomia;

ARTICLE INFO

DOI: https://doi.org/10.46979/rbn.v61i3.68336

CORRESPONDENCE AUTHOR
Lucas Garcia Marcelino, email: lgarciamarcelino@gmail.com

Institution: Instituto de Puericultura e Pediatria Martagão Gesteira - Universidade Federal do Rio de Janeiro (IPPMG - UFRJ)

Declaration of conflict of interest: The authors declare no conflict of interest.

Funding: The article is funded by the authors, with no external sources.

INTRODUCTION

Myasthenia Gravis (MG) is the most common autoimmune disease of the neuromuscular junction[1]. It usually has available treatments, but it is a disease with considerable morbidity and mortality. Juvenile Myasthenia Gravis (JMG) refers to the manifestations of MG in patients under 18 years of age, accounting for 10-20% of total cases. Myasthenic symptoms are explained by transmission defects at the neuromuscular junction, presenting in varied clinical spectra[2]. In mild forms, it can affect only the ocular muscles, with ptosis and double vision. Most cases present muscle fatigue and limb weakness, and may or may not evolve with dysphonia, dysphagia and respiratory muscle fatigue in myasthenic crisis, presenting a serious risk to the child's life.

Treatment is individualized and scaled according to the severity of the symptoms reported. Involves acetylcholine reuptake inhibitors, immunosuppressive drugs, immunoglobulin infusion and procedures such as plasmapheresis and thymectomy[3,4]. There is no universally accepted standard treatment for JMG in children. At our service, we utilize the Clinical Protocol and Therapeutic Guidelines (PCDT)[5] from the Brazilian Ministry of Health, which is essential in the treatment of JMG, as it guides therapeutic decisions and ensures standardized and effective access to treatment options. The presence of comorbidities related to other autoimmune diseases and/or derived from treatment constitutes an important prognostic factor for these patients[6].

Despite being a clinically recognized entity, its rarity in the pediatric population means we know little about the clinical features of these patients. In a large and ethnically diverse population like Brazil, it is important to develop a profile of our children. There is a need to understand whether our population is similar to that observed in other groups, as this impacts follow-up and could lead us to a more refined disease management guideline for Brazilian children.

OBJECTIVE

JMG is a subgroup of a rare childhood disease, with its own specificities. Given the limited number of updated references in descriptive studies, the objective of this article is to describe the clinical characteristics and follow up of children with JMG at neuromuscular disease center in Rio de Janeiro - Brazil. We present the clinical profile of JMG in terms of initial symptoms and signs, diagnosis, laboratory alterations, associated comorbidities, frequency of myasthenic crises and the different treatments with respective responses. These patients with JMG were followed up at the neuromuscular diseases center of Instituto de Puericultura e Pediatria Martagão Gesteira (IPPMG) at Universidade Federal do Rio de Janeiro (UFRJ). We will perform a descriptive comparison between our group of patients and the information available in the databases through a non-systematic review of the medical-scientific literature.

MATERIALS AND METHODS

We have conducted a retrospective, descriptive study of patients with JMG of the past 20 years (2004-2024), the period of establishment of a clinical follow up formulary for neuromuscular diseases at our reference center. This study was approved by the Institutional Review Board of IPPMG-UFRJ, CAEE number 76695423.5.0000.5264, complying with all applicable ethical standards. Our criteria of inclusion were patients from age 0 - 18 years, regularly followed up, with chronic myasthenic manifestations and who presented a positive therapeutic test with the use of cholinesterase inhibitors. Patients with symptoms explained by mitochondrial myopathies, muscular dystrophies and congenital myopathy were excluded. Clinical variables were collected from the formulary at the hospital's medical records. It was not possible for all patients to undergo laboratory, imaging and pathological assessments, since this was not a prospective longitudinal study. Numerical variables included current age and age at onset of symptoms. Categorical variables included sex, onset presentation, symptoms through follow up, antibody positivity, results of neurophysiological tests, thymus evaluation through CT scan, treatments performed by the patient, occurrence of myasthenic crises and associated comorbidities. All assessments analyzed in this study were performed in hospitals affiliated with the Brazilian Public Healthcare System. Information was later transferred to electronic spreadsheets and presented as measures of central tendency and dispersion. Based on the results found, we present a descriptive view or our cases together with a brief literature review. Eleven children and adolescents aged between 0 and 18 years met the inclusion criteria. Three patients initially included had limb weakness but were excluded because of other diagnoses.

RESULTS

We recovered 11 cases that met the inclusion criteria, between 2009 and 2024. Currently, the average age of patients being followed is 9,7 years, with a standard deviation of 4,3 years. The average age of symptom onset is 5,1 years, with a standard deviation of 3,7 years. The majority (72,7%) of patients in our group are female, the remaining (27,3%) are man. The most frequent initial presentation was ocular (45.5%, n=5), followed by bulbar (27.3%, n=3), myasthenic crisis (18.2%, n=2), and muscular forms (9%, n=1). The variability of muscle groups affected at the onset of symptoms determines the clinical presentation. Patients with symptoms exclusively restricted to the eye muscles have the ocular form, while those who present solely with limb weakness have the muscular form.

Those who present with dysphonia and/or dysphagia combined have the bulbar form. It is also possible for the onset of the condition to begin with a myasthenic crisis.

After the onset of the condition, until the expected remission with treatment, it is possible that some patients will experience other symptoms in addition to the initial ones. Ptosis stands out in all patients as the most prevalent sign and symptom presented though follow up (100%, n=11), mainly unilateral but also bilateral. Half had musculoskeletal symptoms such as weakness in the upper and lower limbs (54,5%, n=6). Dysphagia is also common (45,4%, n=5), although other symptoms like dysphonia and facial paresis were less observed (18,1%, n = 2). All patients who began with the ocular form of symptoms remained with only ocular symptoms, without progression. Patients with the bulbar-onset form also presented with symptoms of limb weakness throughout follow-up. The one patient with the muscular-onset form developed ocular symptoms. See Table 1 for the detailed variables results described above.

Table 1. Clinical Profile

Variables	Mode	Relative Frequency	Absolute Frequency
Sex	Feminine	Feminine: 72,7%	Feminine: 8/11
		Masculine: 27,3%	Masculine: 3/11
Onset presentation	Ocular	Ocular: 45,5%	Ocular: 5 /11
		Bulbar: 27,3%	Bulbar: 3/11
		Myasthenic crisis: 18,2%	Myasthenic crisis: 2/11
		Muscular: 9,1%	Muscular: 1/11
Follow up signs and symptoms	Ptosis	Ptosis: 100%	Ptosis: 11/11
		Weakness: 54,5%	Weakness: 6/11
		Dysphagia: 45,4%	Dysphagia: 5/11
		Dysphonia: 18,1%	Dysphonia: 2/11
		Facial paresis: 18,1%	Facial paresis: 2/11

Asthma appears as the most frequent autoimmune comorbidity, in 3 patients (27.2%) and in 1 patient (9.1%) there was acanthosis nigricans, suggestive of insulin resistance. Among the neuropsychiatric comorbidities, 3 patients had anxiety (27.2%) and 1 had depression (9.1%). One patient had autism spectrum disorder (9.1%) and 2 patients also had epilepsy (18.2%).

Antibodies testing were performed on all patients, however, it was not possible to find the results of one patient. Amongst the 10 patients with results available, 8 had positive antibodies (72.7%), all of which were anti-acetylcholine receptor (Anti-AchR) antibodies. Two were seronegative for this antibody (18.1%). Among the AchR-positive patients, two presented initially with ocular form, three with bulbar form, one with muscular form, and one opened with a myasthenic crisis. Between AchR-negative patients, one started with the ocular form and another with a myasthenic crisis. All patients (100%) were seronegative for anti-muscle-specific tyrosine kinase (Anti-Musk) antibodies.

All patients went through thymus evaluation through computed tomography of the chest, except for one. It showed that 8 patients (72.7%) had a normal-sized thymus, 1 patient (9%) had thymic atrophy and 1 patient (9%) had thymic hyperplasia. This last patient it was the only one who underwent thymectomy, at 10 years of age. Electromyography was not performed in 9 patients, while 1 patient showed a decremental response and 1 patient showed a myopathic pattern.

All patients (100%) used an acetylcholine degradation inhibitor, pyridostigmine, to treat JMG. Ten patients (90.9%) received corticosteroids at immunosuppressive doses, encompassing all forms of initial presentation. The patient who did not received corticosteroids presented with a purely ocular form. Among the prescribed corticosteroids, 8 patients (80%) used prednisone/prednisolone and 2 patients (20%) used deflazacort. Only 1 patient (9.1%) used pyridostigmine as monotherapy and it was a patient with pure ocular form. Four patients (36.3%) required another type of immunosuppressor at some point due the side effects of chronic use of corticosteroids for symptom improvement, with azathioprine being the chosen treatment. Other treatments can be used, with 2 patients (18,1%) using other salbutamol as adjuvant therapy.

Six patients (54.5%) had never had a myasthenic crisis, while 5 patients (45.5%) had already suffered from acute crises at least once since diagnosis. Between the patients who had a myasthenic crisis, one had the ocular opening form, two had the bulbar form while two others had an opening in crisis. Of the patients who never experienced crisis, four presented with the ocular form, one with the muscular form, and one with the bulbar form.

All patients who had already suffered crises currently have one crisis per year or less, after optimization of treatment. Among the hospital treatments available for myasthenic crises in our institution for these patients, 3 patients (60%) used human immunoglobulin, 2 underwent pulse therapy with methylprednisolone (40%) and 1 required plasmapheresis (20%). It was not possible to ascertain the crisis treatment proposed for 1 patient.

DISCUSSION

The clinical characteristics of JMG in this Brazilian pediatric population were similar to ones reported worldwide. MG has a bimodal behavior regarding the age of initial symptoms in women, with peaks at the ages of 30 and 50 years. In adult men, the older the age, the higher the incidence, especially after 60 years. In our group of patients, the prevalence of females was observed, which is predicted

by the literature, since in diagnoses under 40 years of age, the ratio of women:men is 3:1[7]. The most affected pediatric age group, as in other autoimmune diseases, are children aged 9 years and older, usually pre-adolescents or adolescents[8]. In our service, the average age at onset of the disease is 5 years and the average age among patients is 9 years. The average age at onset of cases seen in a recent meta-analysis is approximately 7 years[8]. This discrepancy is probably explained by the fact that we are a regional reference center, with a capillarity that allows access to earlier cases.

Fluctuating muscle fatigue is one of the most specific symptoms of MG, mainly affecting the ocular muscles[9]. In our case series, we observed the pattern already demonstrated in North American, Central American and European populations of predominance of the pure ocular form and ocular symptoms present in all cases[10–12]. The frequency of ocular myasthenia is usually inversely proportional to the age at onset of the condition[10,13]. Bulbar symptoms appear more frequently than described, however, this does not translate into a greater number of patients initiating the condition already in myasthenic crisis. Generalization of symptoms with myasthenic crisis is usually common in post-pubertal patients[14]. It is observed that the older the child at the age at onset of the condition, the greater the risk of myasthenic crisis, which is reflected in the behavior of patients followed. Thus, JMG is considered to be a disease with a relatively benign course, although a national case series[9] observed up to 61% of patients with severe forms of the disease, with up to 44.4% of these patients presenting myasthenic crisis. In a comparative analysis with the profile of our service, we see a less severe progression due to the younger age group of our patients, with earlier diagnoses and a larger window of opportunity for administration of acetylcholine reuptake inhibitors, which help to inhibit the progression of the disease in most cases[4].

In addition to the physical examination, antibody testing is part of the investigation of the disease, the most common being the anti-acetylcholine receptor antibody (Anti-AchR). A positivity for this antibody is seen in up to 72-86% of patients, and the proportionality of titers of this antibody is not related to factors that worsen the disease[15]. In patients seronegative for Anti-AchR, up to 40% positivity for Anti-Musk can be observed[16]. A small group of patients may be seronegative for both antibodies, and these should be retested over time because there is a chance of seroconversion. Seropositive patients usually present severe forms of the disease, and it is not surprising that this antibody is less common in the pediatric population, including our group, where JMG most often manifests as a mild, ocular form.

The frequency of thymomas in the pediatric population with myasthenia is low, but thymectomy should always be considered in patients with difficult-to-control disease or in severe forms, regardless of whether there is thymic alteration or not[17]. Among the alterations, thymic hyperplasia is the most common, especially on post pubertal patients[18], and thymic atrophy the rarest[8]. Due to the role of this lymphoid organ in the course of the disease, all children with JMG should undergo imaging examination. Although most of our patients underwent thymus evaluation, few were indicated for thymectomy, which was performed in only one patient and with good results. Because our age group of patients is still just a few years into puberty, it is unlikely that there would be a clear indication, given that the general trend in non-ocular forms is for worsening after puberty[19].

As an autoimmune disease, MG may be associated with other concomitant autoimmune disorders, contributing to the prognosis and adherence to treatment of these patients. The most common autoimmune diseases associated with MG in adults are thyroid disease (hypothyroidism and hyperthyroidism), rheumatoid arthritis, and systemic lupus erythematosus (SLE)[8]. A similar pattern is observed in the pediatric population, with thyroid disease being extremely common, and psoriasis and SLE being less prevalent[8]. Given the age of our patients, it is unlikely that they will present comorbidities during this period, and screening for these most common diseases should be part of our therapeutic plan for early diagnosis and intervention. Being less studied in the literature in association with JMG, we also observed neuropsychiatric comorbidities and concluded that the older the patient and the severity of the symptoms, the greater the chances of the pediatric patient developing emotional disorders. This increases the patient's medicalization and consequently adherence to treatment, influencing their prognosis.

A myasthenic crisis is characterized by an acute worsening of muscle weakness that may require ventilatory support and may be triggered by infections, surgeries and procedures, or medications[4] that impair transmission at the neuromuscular junction. Crises require treatment in hospital units for monitoring and advanced support. Among the hospital treatments commonly used to manage myasthenic crises, the use of intravenous immunoglobulin (IVIG), pulse therapy with methylprednisolone, and plasmapheresis stand out. All of our patients responded well to myasthenic treatment. There is no consensus on the factors that lead a patient to develop a myasthenic crisis, especially among patients who migrate from the ocular form to other presentations. Some systematic reviews correlate positivity for anti-AchR antibodies and female gender with a greater risk factor[20], which our patient group appears to be somewhat consistent with, given that most myasthenic crises occurred in female patients with positive serology.

The gold standard treatment of MG and JMG is based on the use of anticholinesterase drugs and immunosuppressants[21,22]. Symptom management is usually

done with pyridostigmine, but early use of immunosuppressants is considered when good control is not achieved or when the initial clinical symptoms are serious. Oral prednisolone/prednisone is used at an immunosuppressive dose initially, with appropriate prevention and monitoring of side effects. Since most of our children maintained the pure ocular form, they would be expected to be able to control symptoms with acetylcholine reuptake inhibitors alone. However, as demonstrated in other centers, even in this milder form, corticosteroids are often necessary to achieve optimal clinical control[23]. Second-line therapies with other immunosuppressants must be considered if there is no response to steroids, or it is impossible to reduce to a reasonable minimum effective dose, or if side effects are intolerable[3,4]. Azathioprine usually is the first choice because of larger experience in JMG, although is possible to utilize mycophenolate, cyclosporine, cyclophosphamide or tacrolimus[3,24]. Other classes of medications have recently emerged as a new therapeutic strategy. They inhibit complement pathways pathophysiological linked to the process of myasthenic antibody formation[25].

Limitations of this study are the small number of patients for more robust results; the lack of electroneuromyography study, antibodies results and thymus evaluation for all patients; and the difficulty in capturing patients with purely ocular myasthenia gravis because these cases are generally followed up by ophthalmologists only.

CONCLUSION

At our neuromuscular diseases center in Brazil, our patient profile is very similar to the descriptions made so far of patients with JMG. The understanding that our population is similar to that observed in other groups reinforces the applicability of national and international guidelines for treatment and follow-up, and improves our knowledge for conducting new cases.

REFERENCES

1. McGrogan A, Sneddon S, De Vries CS. The Incidence of Myasthenia Gravis: A Systematic Literature Review. Neuroepidemiology. v.34, n.3, p.171–83, 2010.
2. Ha JC, Richman DP. Myasthenia gravis and related disorders: Pathology and molecular pathogenesis. Biochim Biophys Acta BBA - Mol Basis Dis. Apr; v.185, n.4, p.651–7, 2015.
3. O'Connell K, Ramdas S, Palace J. Management of Juvenile Myasthenia Gravis. Front Neurol. July, v. 24, n.11, p.743, 2020.
4. Munot P, Robb SA, Niks EH, Palace J, Munot P, Palace J, et al. 242nd ENMC International Workshop: Diagnosis and management of juvenile myasthenia gravis Hoofddorp, the Netherlands, 1–3 March 2019. Neuromuscul Disord. Mar; v.30, n.3, p.254–64, 2020.
5. PCDT Miastenia Gravis, 2022. PORTARIA CONJUNTA Nº 11, DE 23 DE MAIO DE 2022. 2022. Disponível em: https://www.gov.br/conitec/pt-br/midias/protocolos/copy_of_20220530_PORTAL_PCDT_Miastenia_Gravis.pdf.Acesso em: 28 setembro de 2025.
6. Lin Y, Kuang Q, Li H, Liang B, Lu J, Jiang Q, et al. Outcome and clinical features in juvenile myasthenia gravis: A systematic review and meta-analysis. Front Neurol. Mar, v.8, n.14, p.1119294, 2023.
7. Gilhus NE. Myasthenia gravis and congenital myasthenic syndromes. Handb Clin Neurol. v.195, p.635–52, 2023.
8. Dresser L, Wlodarski R, Rezania K, Soliven B. Myasthenia Gravis: Epidemiology, Pathophysiology and Clinical Manifestations. J Clin Med. May v.10, n.11, p.2235, 2021.
9. Morita MDPA, Gabbai AA, Oliveira ASB, Penn AS. Myasthenia gravis in children: analysis of 18 patients. Arq Neuropsiquiatr. Sept; v.59, n.3B, p.681–5, 2001.
10. Melbourne Chambers R, Forrester S, Gray R, Tapper J, Trotman H. Myasthenia gravis in Jamaican children: a 12-year institutional review. Paediatr Int Child Health. Feb; v.32, n.1, p.47–50, 2012.
11. Lu M, Reisch J, Iannaccone S, Batley K. Juvenile Myasthenia Gravis in North Texas: Clinical Features, Treatment Response, and Outcomes. Pediatr Neurol. July; v.156, p.10–4, 2024.
12. Vissing J, Atula S, Savolainen M, Mehtälä J, Mehkri L, Olesen TB, et al. Epidemiology of myasthenia gravis in Denmark, Finland and Sweden: a population-based observational study. J Neurol Neurosurg Psychiatry. Sept; v.95, n.10, p.919–26, 2024.
13. Keovilayhong S, Mulliez A, Feral L, Chenaf C, Clavelou P, Moisset X, et al. Epidemiology of myasthenia gravis in France: Incidence, prevalence, and comorbidities based on national healthcare insurance claims data. Rev Neurol (Paris). May; v.18, n.5, p. 451–8, 2024.
14. Zhou J, Nilius S, Pilipczuk O, Scowcroft A, Tarancón T, Tennigkeit F, et al. Treatment Patterns and Disease Burden of Juvenile Myasthenia Gravis in the United States: A Cohort Study Using Health Care Claims Databases. Neurology. July; v.105, n.1, p. e213736, 2025.
15. Anlar B, Şenbil N, Köse G, Değerliyurt A. Serological follow-up in juvenile myasthenia: clinical and acetylcholine receptor antibody status of patients followed for at least 2 years. Neuromuscul Disord. May; v.15, n.5, p.355–7, 2005.
16. Zhou L, McConville J, Chaudhry V, Adams RN, Skolasky RL, Vincent A, et al. Clinical comparison of muscle-specific tyrosine kinase (MuSK) antibody-positive and -negative myasthenic patients. Muscle Nerve. July; v.30, n.1, p.55–60, 2004.
17. Ng WC, Hartley L. Effectiveness of thymectomy in juvenile myasthenia gravis and clinical characteristics associated with better outcomes. Neuromuscul Disord. Nov; v.3, n.11, p.1113–23, 2021.
18. Truffault F, Auger L, Dragin N, Vilquin JT, Fadel E, Thomas De Montpreville V, et al. Comparison of juvenile and adult myasthenia gravis in a French cohort with focus on thymic histology. Sci Rep. June v.14, n.1, p.13955, 2024.
19. Özsoy Ö, Cinleti T, Günay Ç, Sarıkaya Uzan G, Giray Bozkaya Ö, Çağlayan AO, et al. Genetic, serological and clinical evaluation of childhood myasthenia syndromes- single center subgroup analysis experience in Turkey. Acta Neurol Belg. Dec; v.123, n.6, p.2325–35, 2023.
20. Fang CEH, Bokre D, Wong SH. Clinical Characteristics Associated With Secondary Generalization in Patients With Ocular Myasthenia Gravis: A Systematic Review and Meta-analysis. Neurology [Internet]. 2023 Oct 17 [cited 2025 Sept 23];101(16). Disponível em: https://www.neurology.org/doi/10.1212/WNL.0000000000207642. Acesso em: 01 de outubro de 2025.
21. Della Marina A, Trippe H, Lutz S, Schara U. Juvenile Myasthenia Gravis: Recommendations for Diagnostic Approaches and Treatment. Neuropediatrics. Jan 27; v.45, n.02, p.075–83, 2014.
22. Ionita CM, Acsadi G. Management of Juvenile Myasthenia Gravis. Pediatr Neurol. Feb; v.48, n.2, p.95–104, 2013.
23. Kessi M, Tang Y, Chen B, Wang G, Zhang C, He F, et al. Pediatric Ocular Myasthenia Gravis: Single-Center Experience. Pediatr Neurol. Apr, v.153, p.137–43, 2024.

24. Jiang A, Hu Q, Wang Z, Wu F. Efficacy and Safety of Tacrolimus Therapy in Patients With Juvenile Myasthenia Gravis: A Single-Arm Meta-Analysis. Pediatr Neurol. May; v.166, p.32–8, 2025.
25. Wang Y, Nao J, Duan Y, Li Z, Feng J. Therapeutic strategies targeting complement in myasthenia gravis patients. J Neurol. Aug; v.272, n.8, p.489, 2025.