

Case Report

Not peer-reviewed version

---

# Dual Autoimmunity: Myasthenia Gravis and Rheumatoid Arthritis—A Case Report

---

Andreea V. Slevoacă-Grigore , [Alexandra Mincă](#) \* , Dragoş I. Mincă \* , [Claudiu C. Popescu](#) , [Alexandra M. Cristea](#) , [Adina Rusu](#) , [Amalia L. Călinoiu](#)

Posted Date: 17 April 2026

doi: 10.20944/preprints202604.1284.v1

Keywords: myasthenia gravis; rheumatoid arthritis; autoimmune diseases; drug-induced liver cirrhosis



Preprints.org is a free multidisciplinary platform providing preprint service that is dedicated to making early versions of research outputs permanently available and citable. Preprints posted at Preprints.org appear in Web of Science, Crossref, Google Scholar, Scilit, Europe PMC.

Copyright: This open access article is published under a [Creative Commons CC BY 4.0 license](#), which permit the free download, distribution, and reuse, provided that the author and preprint are cited in any reuse.

Disclaimer/Publisher's Note: The statements, opinions, and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of MDPI and/or the editor(s). MDPI and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions, or products referred to in the content.

Case Report

# Dual Autoimmunity: Myasthenia Gravis and Rheumatoid Arthritis—A Case Report

Andreea V. Slevoacă-Grigore <sup>1</sup>, Alexandra Mincă <sup>1,2,\*</sup>, Dragoș I. Mincă <sup>3,4,\*</sup>, Claudiu C. Popescu <sup>3,4</sup>, Alexandra M. Cristea <sup>4,5</sup>, Adina Rusu <sup>1</sup> and Amalia L. Călinoiu <sup>1</sup>

<sup>1</sup> "Prof. Dr. Agrippa Ionescu" Emergency Clinical Hospital, 011356 Bucharest, Romania

<sup>2</sup> Public Health and Management Department, "Carol Davila" University of Medicine and Pharmacy, 020021 Bucharest, Romania

<sup>3</sup> Rheumatology Department, "Carol Davila" University of Medicine and Pharmacy, 020021 Bucharest, Romania

<sup>4</sup> Pneumology I Department, Carol Davila University of Medicine and Pharmacy, 020021 Bucharest, Romania

<sup>5</sup> Marius Nasta Institute of Pneumology, 050159 Bucharest, Romania

\* Correspondence: alexandra.tintea@drd.umfcd.ro (A.M.); dragos.minca@reumatologiedrstoia.ro (D.I.M.)

## Abstract

**Background:** The coexistence of Myasthenia Gravis (MG) and Rheumatoid Arthritis (RA) represents a rare but clinically challenging form of polyautoimmunity, raising interesting questions about shared immunopathogenic mechanisms and the safety of long-term immunomodulatory therapies.

**Methods:** The article describes a case report of a 66-year-old female with a 12-year history of seropositive RA who subsequently developed seropositive MG during long-term exposure to hydroxychloroquine (HCQ) therapy. Following discontinuation of HCQ, methotrexate (MTX) therapy was initiated and stable control of both diseases was temporally obtained. **Results:** Three years later, the patient presented with upper gastrointestinal bleeding and severe microcytic anemia. Further evaluation revealed advanced liver fibrosis (F4) and severe gastropathy, consistent with Child–Pugh class A cirrhosis. Viral, alcoholic, and autoimmune causes of chronic liver disease were excluded. In the absence of alternative etiologies, this was considered possibly associated with MTX therapy, in the context of additional metabolic risk factors, including type 2 diabetes mellitus and increased body mass index. **Conclusions:** The complex interplay between polyautoimmunity and treatment-related toxicity is underscored in this article. Overlapping autoimmune diseases may arise on a shared immunological background, while therapeutic agents may contribute to disease expression or long-term complications. These findings highlight the need for individualized therapeutic strategies and vigilant monitoring, particularly in patients with coexisting metabolic risk factors.

**Keywords:** myasthenia gravis; rheumatoid arthritis; autoimmune diseases; drug-induced liver cirrhosis

## 1. Introduction

Myasthenia gravis (MG) is an acquired autoimmune disease of neuromuscular transmission characterised by fluctuating weakness of skeletal muscles, with variable distribution among muscle groups, involving ocular, bulbar, respiratory, or limb muscles [1]. MG comprises both thymoma-related (paraneoplastic) and non-paraneoplastic subtypes, underscoring the immunological heterogeneity of the disease [2–4]. Most commonly, this pathology is mediated by autoantibodies against the acetylcholine receptor (AChR-Abs), present in approximately 80% of patients, while muscle-specific kinase (MuSK-Abs) and low-density lipoprotein receptor-related protein 4 antibodies (LRP4-abs) are less frequent, being present in 4% and 2% of the cases, involved in reducing receptor density and disrupting postsynaptic membrane structure, impairing neuromuscular transmission

[2,5]. The reported prevalence of MG is 150–200 cases per million, and an annual incidence in Europe ranging from 4.1 to 30 per million [6]. MG shows a bimodal distribution, predominantly affecting women between 15 and 64 years old, while incidence increases in men after the sixth decade of life [7]. Genetic predisposition has also been described in the literature, with first-degree relatives presenting an increased lifetime risk of disease development [5]. Additionally, recent studies have documented cases of new-onset MG precipitated by SARS-CoV-2 infection, suggesting a potential post-infectious or immune-mediated trigger [8–10].

Beyond its primary manifestations, MG is associated with other autoimmune diseases (ADs) in 13–22% of cases. Autoimmune thyroid diseases represent the most frequent association, followed by systemic lupus erythematosus and rheumatoid arthritis (RA) [11–14]. Moreover, thymectomy has been identified as a risk factor for the development of subsequent ADs, possibly reflecting alterations in central immune tolerance and inadequate elimination of autoreactive lymphocyte clones [13,15].

RA is a systemic AD characterized by persistent systemic inflammation and joint destruction [16,17]. Beyond joint involvement, RA patients may present with extra-articular manifestations in various organ systems, including the cardiovascular, renal, pulmonary, gastrointestinal, ocular, dermatologic, and nervous systems [16,18].

The coexistence of RA in patients with MG is relatively uncommon, with an estimated prevalence of 1–4% [19,20]. Meta-analytic data demonstrated an overall prevalence of approximately 3% for this association, findings that are consistent with observations reported in smaller patient cohorts [14,21,22]. Similar to other ADs, patients with RA have an increased risk of developing MG compared with the general population. Epidemiological data indicate that both the prevalence and incidence of MG are approximately 3–4 times higher in RA patients, supporting a non-random association between these two ADs [23]. This association requires significant diagnostic and therapeutic approaches, being challenging, particularly in the context of immunosuppressive treatment and potential drug-related complications. Although methotrexate (MTX), along with rituximab and glucocorticoids, remains one of the preferred, safe and effective treatment options in patients with RA and MG, its role in the development of advanced liver disease remains controversial, as recent studies suggest that hepatotoxicity is multifactorial and influenced by both patient-related factors (for example, type 2 diabetes mellitus, obesity) and cumulative dose [24–26]. This study aims to report a rare case of a 66-year-old female with both RA and MG complicated by probable MTX-associated liver cirrhosis after 3 years of treatment.

This case highlights the complex interplay between two ADs and drug-related toxicity, underscoring the importance of individualized risk assessment and long-term monitoring in patients receiving long-term MTX therapy.

## 2. Case Presentation

A 66-year-old Caucasian female was admitted to the internal medicine department of an emergency hospital from Bucharest, Romania for a 2-week history of vertigo, palpitations, pallor, and lower limb edema, which had been worsening during the preceding 48 h.

The patient had no significant family medical history, but she had a personal complex autoimmune background, including the coexistence of RA and MG. Also, the patient had type 2 diabetes mellitus and hypertension. She was diagnosed with anti-citrullinated protein antibodies (ACPA)-positive RA 12 years earlier, at which time hydroxychloroquine (HCQ) 200 mg/day was initiated. The patient's clinical course remained favorable under this treatment for nine years, when she developed bilateral ptosis, diplopia, and generalized limb weakness. At that time, immunological evaluation revealed an elevated anti-AChR titer (19.15 nmol/L; normal range <0.25 nmol/L), whereas anti-MuSK antibodies were negative, confirming seropositive MG. HCQ was discontinued, and MTX 10 mg/week was introduced, along with pyridostigmine 240 mg/day. Over the following three years, the patient's clinical course remained stable, with no exacerbations of RA or MG. Previous medical records, including abdominal ultrasound examinations performed before the current admission, showed no evidence of liver or splenic pathology.

On presentation to the emergency room, she was conscious and cooperative but in a poor general condition. Physical examination revealed an overweight patient (body mass index 28.3) with pale skin and mucous membranes, tachycardia (heart rate 119 beats/minute), and blood pressure within normal limits (120/60 mmHg). Also, joint stiffness and deformities consistent with longstanding RA were observed. Laboratory investigations revealed severe hypochromic microcytic anemia (Hb- 3.1 g/dL, normal range 10.9-14.3g/dL, WBC 12.0/10<sup>3</sup>/μL, normal range 3.8-11.8/10<sup>3</sup>/μL, RBC- 1.161/10<sup>6</sup>/μL, normal range 3.63-4.92/10<sup>6</sup>/μL, PLT 214/10<sup>3</sup>/μL, normal range 179-408/10<sup>3</sup>/μL, MCV-65.8fL, normal range 75.5-95.3fL, HCT 10.6%, normal range 31.2-41.9%, RDW 22.7%, normal range 12.3-17.7% ), the international normalized ratio (INR) was 1.51, hyperglycemia (177 mg/dL, normal range 74-106mg/dL), increased level of direct bilirubin (0.8mg/dL, normal range 0-0.3mg/dL), alanine aminotransferase, aspartate aminotransferase, gamma-glutamyl transferase, albumin and vitamin B12 serum levels were within normal limits. Given the laboratory findings suggestive of hepatic dysfunction and the patient's long-term exposure to MTX, further evaluation for chronic liver disease was initiated. Viral hepatitis markers (HBV, HCV) were negative. Alcohol consumption was denied, and autoimmune liver diseases (autoimmune hepatitis, primary biliary cholangitis, and primary sclerosing cholangitis) were excluded based on a negative autoimmune panel (antinuclear antibodies, anti-smooth muscle antibodies, anti-mitochondrial antibodies and liver-kidney microsomal type 1 antibodies)

Abdominal ultrasonography revealed splenomegaly (130 × 45mm) and a heterogeneous micro- and macronodular liver appearance. Upper gastrointestinal endoscopy revealed portal hypertensive gastropathy and multiple erosions in the pyloric antrum, some of which showed stigmata of recent bleeding. Transient elastography (FibroScan) confirmed advanced fibrosis (F4). Based on these findings, the patient was diagnosed with upper gastrointestinal bleeding, secondary severe anemia and Child–Pugh class A liver cirrhosis, possibly associated with MTX therapy. Liver biopsy was not performed due to clinical considerations, as the diagnosis was supported by non-invasive and endoscopic findings.

During the hospitalization, the treatment with MTX was discontinued, she received blood transfusions, folic supplement, hypoglycemic medication (consisting of oral antidiabetes agents and insulin), ACE inhibitors and proton pump inhibitors. Subsequently, disease-modifying therapy for RA was initiated with leflunomide, which the patient did not tolerate due to gastrointestinal side effects. Treatment was then switched to sulfasalazine; however, this was discontinued following the development of an allergic reaction. As a result, the patient remained under clinical observation and is currently in remission, with no acute exacerbations of RA. In case of disease reactivation, initiation of biologic therapy will be considered.

### 3. Discussion

A small number of case reports describing the coexistence of RA and MG have been published, while large-scale cohort studies remain lacking, resulting in the absence of well-established management guidelines for patients with both conditions [24,27–35]. Recent genetic and transcriptomic analyses have demonstrated that RA is associated with a significantly increased risk of MG, while also identifying multiple shared genes and immune pathways involved in both diseases [36]. MG and RA are characterized by a breakdown of self-tolerance and the production of pathogenic autoantibodies, although targeting different structures: neuromuscular junction components in MG and synovial antigens in RA [5,37]. CD4+ T-cell activation, cytokine dysregulation, and aberrant antigen presentation have been implicated in both conditions, reinforcing the concept of systemic immune dysregulation rather than organ-specific autoimmunity alone [5,37]. Additional contributing factors include shared genetic susceptibility, particularly within the HLA region, as well as environmental influences that modulate immune responses [5,37]. Therefore, this case contributes to the growing body of evidence suggesting that overlapping ADs may develop on a shared immunological background, although the underlying mechanisms and individual contributing factors remain incompletely understood.

While a causal relationship cannot be established, the coexistence of these two ADs in the present patient could reflect an underlying predisposition to polyautoimmunity, rather than an incidental association.

Both the onset of MG and the exacerbation of pre-existing disease have been reported in association with HCQ use [38–40]. In the present case, the onset of MG manifestations (ptosis, diplopia, and generalized weakness) occurred after nine years of HCQ exposure.

Additionally, glucocorticoids, commonly used in the management of RA, have been reported to transiently worsen or potentially unmask MG, particularly during treatment initiation or dose adjustments [5,41,42]. The patient presented in this case had no recent history of corticosteroid exposure. Overall, the development of MG in this context may be interpreted as a multifactorial process, in which underlying autoimmune susceptibility and potential pharmacological triggers interact.

MTX remains the first-line option in the management of RA [24]; however, its long-term use has been associated with hepatic adverse effects, including hepatic fibrosis and, less commonly, cirrhosis [16,25,26,43,44]. Earlier studies suggested a relatively high risk of progressive liver injury, particularly with cumulative dosing, leading to recommendations for routine liver monitoring [25]. More recent evidence indicates that clinically significant fibrosis is relatively uncommon and that the risk of advanced liver disease is likely multifactorial rather than directly attributable to MTX alone [45]. In particular, components of metabolic syndrome (type 2 diabetes mellitus, obesity) have been consistently identified as important contributors to hepatic fibrosis in patients receiving MTX therapy [25,45].

In this patient, the development of advanced fibrosis (F4) and Child-Pugh A cirrhosis after several years of MTX therapy should therefore be interpreted with caution. Given the presence of additional risk factors, including type 2 diabetes mellitus and overweight status, a multifactorial mechanism of liver injury appears more likely. Consequently, this case supports the need for individualized risk assessment and tailored monitoring strategies in patients receiving long-term MTX therapy, rather than attributing hepatic complications solely to drug exposure.

Also, this case report underscores the complexity of polyautoimmunity and the challenges associated with managing overlapping ADs. The coexistence of RA and MG introduces significant therapeutic dilemmas, particularly when treatments for one condition may adversely affect the other.

Future research should focus on clarifying the immunological links between RA and MG and identifying predictive markers for patients at increased risk of developing multiple ADs. Furthermore, prospective studies are needed to better define the contribution of metabolic risk factors and MTX exposure in the development of liver fibrosis, in order to optimize monitoring strategies and improve long-term patient outcomes.

#### 4. Conclusions

Clinicians should remain vigilant for both ADs and drug-related complications in patients with complex autoimmune profiles. Early recognition of hepatic involvement and tailored monitoring, particularly in patients with individual risk factors such as the coexistence of type 2 diabetes mellitus and increased body weight, may prevent the progression to advanced liver disease.

**Author Contributions:** Conceptualization, A.V.S.G. and A.M.; methodology, A.L.C., C.C.P. and D.I.M.; validation, A.M.C, A.R. and A.M; formal analysis, D.I.M; investigation, A.V.S.G.; resources, A.M; data curation, A.R.; writing—original draft preparation, A.V.S.G; writing—review and editing, A.M.; visualization, A.M.C. and D.I.M.; supervision, A.L.C. and C.C.P; project administration, D.I.M; . All authors have read and agreed to the published version of the manuscript.

**Funding:** This research received no external funding.

**Institutional Review Board Statement:** The study was conducted in accordance with the Declaration of Helsinki. This case report does not require review or approval by the Institutional Review Board.

**Informed Consent Statement:** Informed consent was obtained from the patient involved in the study. Written informed consent has been obtained from the patient to publish this paper.

**Data Availability Statement:** The data presented in this study are available on request from the corresponding author.

**Acknowledgments:** Not applicable.

**Conflicts of Interest:** The authors declare no conflicts of interest.

## Abbreviations

The following abbreviations are used in this manuscript:

ACPA	Anti-citrullinated protein antibodies
AChR-Abs	Anti-acetylcholine receptor antibodies
ADs	Autoimmune diseases
HCQ	Hydroxychloroquine
LPR4-Abs	Low-density lipoprotein receptor-related protein 4 antibodies
MG	Myasthenia gravis
MTX	Methotrexate
MuSK-Abs	Muscle-specific kinase antibodies
RA	Rheumatoid arthritis

## References

1. Dresser, L.; Wlodarski, R.; Rezaia, K.; Soliven, B. Myasthenia Gravis: Epidemiology, Pathophysiology and Clinical Manifestations. *J. Clin. Med.* **2021**, *10*, 2235, doi:10.3390/jcm10112235.
2. Gilhus, N.E.; Verschuuren, J.J. Myasthenia gravis: subgroup classification and therapeutic strategies. *Lancet Neurol.* **2015**, *14*, 1023–1036, [https://doi.org/10.1016/s1474-4422\(15\)00145-3](https://doi.org/10.1016/s1474-4422(15)00145-3).
3. Khateb, M.; Abu Zant, M.; Bsoul, A.; Karny, T.; Yarnitsky, D.; Shelly, S. The Association Between Myasthenia Gravis and Higher Extrathymic Cancer Risk. *Brain Behav.* **2025**, *15*, e70143, <https://doi.org/10.1002/brb3.70143>.
4. Hamoud, B.H.; Georgescu, D.E.; Călinoiu, A.L.; Văcăroiu, I.A.; Georgescu, M.T. Advances in Squamous Cell Carcinoma Antigen and Cervix Cancer Relationship. *Romanian J. Mil. Med.* **2023**, *126*, 10–14, <https://doi.org/10.55453/rjmm.2023.126.1.2>.
5. Gilhus, N.E.; Tzartos, S.; Evoli, A.; Palace, J.; Burns, T.M.; Verschuuren, J.J.G.M. Myasthenia gravis. *Nat. Rev. Dis. Prim.* **2019**, *5*, 30, <https://doi.org/10.1038/s41572-019-0079-y>.
6. Carr, A.S.; Cardwell, C.R.; O McCarron, P.; McConville, J. A systematic review of population based epidemiological studies in Myasthenia Gravis. *BMC Neurol.* **2010**, *10*, 46–46, <https://doi.org/10.1186/1471-2377-10-46>.
7. Deenen, J.C.; Horlings, C.G.; Verschuuren, J.J.; Verbeek, A.L.; van Engelen, B.G. The Epidemiology of Neuromuscular Disorders: A Comprehensive Overview of the Literature. *J. Neuromuscul. Dis.* **2015**, *2*, 73–85, <https://doi.org/10.3233/jnd-140045>.
8. Mincă, A.; I Mincă, D.; Calinoiu, A.L.; Gheorghită, V.; Popescu, C.C.; Rusu, A.; Cristea, A.M.; Mincă, D.G. Myasthenia Gravis Triggered by a COVID-19 Infection: A Case Report and Literature Review. *Cureus* **2024**, *16*, e59538, <https://doi.org/10.7759/cureus.59538>.
9. Hiraoka, Y.; Hosoi, Y.; Tsubata, T.; Yanagida, M.; Otake, Y.; Ito, M. Double-seropositive Myasthenia Gravis Following COVID-19. *Intern. Med.* **2025**, *64*, 1591–1594, <https://doi.org/10.2169/internalmedicine.5027-24>.
10. Tereshko, Y.; Gigli, G.L.; Pez, S.; De Pellegrin, A.; Valente, M. New-onset Myasthenia Gravis after SARS-CoV-2 infection: case report and literature review. *J. Neurol.* **2022**, *270*, 601–609, <https://doi.org/10.1007/s00415-022-11472-6>.
11. Zhu, Y.; Wang, B.; Hao, Y.; Zhu, R. Clinical features of myasthenia gravis with neurological and systemic autoimmune diseases. *Front. Immunol.* **2023**, *14*, 1223322, <https://doi.org/10.3389/fimmu.2023.1223322>.

12. Yeh, J.-H.; Kuo, H.-T.; Chen, H.-J.; Chen, Y.-K.; Chiu, H.-C.; Kao, C.-H. Higher Risk of Myasthenia Gravis in Patients With Thyroid and Allergic Diseases. *Medicine* **2015**, *94*, e835, <https://doi.org/10.1097/md.0000000000000835>.
13. Chang, C.-C.; Lin, T.-M.; Chang, Y.-S.; Chen, W.-S.; Sheu, J.-J.; Chen, Y.-H.; Chen, J.-H. Thymectomy in patients with myasthenia gravis increases the risk of autoimmune rheumatic diseases: a nationwide cohort study. *Rheumatology* **2018**, *58*, 135–143, <https://doi.org/10.1093/rheumatology/key236>.
14. Tamer, S.; Gunes, H.N.G.; Gokcal, E.; Yoldas, T.K. Coexistence of autoimmune diseases and autoantibodies in patients with myasthenia gravis. *Neurol. India* **2016**, *64*, 45–9, <https://doi.org/10.4103/0028-3886.173638>.
15. Berrih-Aknin, S. Myasthenia Gravis: Paradox versus paradigm in autoimmunity. *J. Autoimmun.* **2014**, *52*, 1–28, <https://doi.org/10.1016/j.jaut.2014.05.001>.
16. Smolen, J.S.; Aletaha, D.; McInnes, I.B. Rheumatoid arthritis. *Lancet* **2016**, *388*, 2023–2038; Erratum in *Lancet* **2016**, *388*, 1984.
17. Aletaha, D.; Neogi, T.; Silman, A.J.; Funovits, J.; Felson, D.T.; Bingham, C.O., 3rd; Birnbaum, N.S.; Burmester, G.R.; Bykerk, V.P.; Cohen, M.D.; et al. 2010 Rheumatoid arthritis classification criteria: an American College of Rheumatology/European League Against Rheumatism collaborative initiative. *Ann. Rheum. Dis.* **2010**, *69*, 1580–1588, <https://doi.org/10.1136/ard.2010.138461>.
18. Sahatçiu-Meka, V.; Rexhepi, S.; Manxhuka-Kërliu, S.; Rexhepi, M. Extra-Articular Manifestations of Seronegative and Seropositive Rheumatoid Arthritis. *Bosn. J. Basic Med Sci.* **2010**, *10*, 26–31, <https://doi.org/10.17305/bjbms.2010.2729>.
19. Fang, F.; Sveinsson, O.; Thormar, G.; Granqvist, M.; Askling, J.; Lundberg, I.E.; Ye, W.; Hammarström, L.; Pirskanen, R.; Piehl, F. The autoimmune spectrum of myasthenia gravis: a Swedish population-based study. *J. Intern. Med.* **2014**, *277*, 594–604, <https://doi.org/10.1111/joim.12310>.
20. Nacu, A.; Andersen, J.B.; Lisnic, V.; Owe, J.F.; Gilhus, N.E. Complicating autoimmune diseases in myasthenia gravis: a review. *Autoimmunity* **2015**, *48*, 362–368, <https://doi.org/10.3109/08916934.2015.1030614>.
21. Misra, U.K.; Kalita, J.; Singh, V.K.; Kumar, S. A study of comorbidities in myasthenia gravis. *Acta Neurol. Belg.* **2019**, *120*, 59–64, <https://doi.org/10.1007/s13760-019-01102-w>.
22. Mao, Z.-F.; Yang, L.-X.; Mo, X.-A.; Qin, C.; Lai, Y.-R.; He, N.-Y.; Li, T.; Hackett, M.L. Frequency of Autoimmune Diseases in Myasthenia Gravis: A Systematic Review. *Int. J. Neurosci.* **2010**, *121*, 121–129, <https://doi.org/10.3109/00207454.2010.539307>.
23. Neil Accortt, Mary Anthony, Jennifer Schenfeld, Travis Wheeling, Anna Hassebroek, Cynthia O'Malley, Michael Sprafka: Incidence and Prevalence of Myasthenia Gravis in Rheumatoid Arthritis Patients with and without Treatment Compared with the General Population. *Arthritis & Rheumatology*. 2014, 6.
24. Bixio, R.; Bertelle, D.; Pistillo, F.; Pedrollo, E.; Carletto, A.; Rossini, M.; Viapiana, O. Rheumatoid arthritis and myasthenia gravis: a case-based review of the therapeutic options. *Clin. Rheumatol.* **2022**, *41*, 1247–1254, <https://doi.org/10.1007/s10067-022-06062-w>.
25. Aithal, G.P. Hepatotoxicity Related to Methotrexate. In: *Drug-Induced Liver Disease*. Elsevier; 2013. 593–604. [10.1016/B978-0-12-387817-5.00032-7](https://doi.org/10.1016/B978-0-12-387817-5.00032-7)
26. Kim, T.Y.; Kim, J.Y.; Sohn, J.H.; Lee, H.-S.; Bang, S.-Y.; Kim, Y.; Kim, M.Y.; Jeong, W.K. Assessment of Substantial Liver Fibrosis by Real-time Shear Wave Elastography in Methotrexate-Treated Patients With Rheumatoid Arthritis. *J. Ultrasound Med.* **2015**, *34*, 1621–1630, <https://doi.org/10.7863/ultra.15.14.10035>.
27. Wakata, N.; Nakazato, A.; Sugimoto, H.; Iguchi, H.; Saito, R. A case of myasthenia gravis accompanied by erythema elevatum diutinum and rheumatoid arthritis. *J. Neurol.* **2001**, *248*, 435–436, <https://doi.org/10.1007/s004150170192>.
28. Zis, P.; Argiriadou, V.; Temperikidis, P.P.; Zikou, L.; Tzartos, S.J.; Tavernarakis, A. Parkinson's disease associated with myasthenia gravis and rheumatoid arthritis. *Neurol. Sci.* **2014**, *35*, 797–799, <https://doi.org/10.1007/s10072-014-1660-5>.
29. Fee, D.B.; Kasarskis, E.J. Myasthenia gravis associated with etanercept therapy. *Muscle Nerve* **2009**, *39*, 866–870, <https://doi.org/10.1002/mus.21280>.
30. Pelechas, E.; Memi, T.; Markatseli, T.E.; Voulgari, P.V.; Drosos, A.A. Adalimumab-induced myasthenia gravis: case-based review. *Rheumatol. Int.* **2020**, *40*, 1891–1894, <https://doi.org/10.1007/s00296-020-04587-4>.

31. Kerkeni, S.; Marotte, H.; Miossec, P. Improvement with rituximab in a patient with both rheumatoid arthritis and myasthenia gravis. *Muscle Nerve* **2008**, *38*, 1343–1345, <https://doi.org/10.1002/mus.21047>.
32. Horai, Y.; Honda, M.; Nishino, A.; Nakashima, Y.; Suzuki, T.; Kawashiri, S.-Y.; Ichinose, K.; Tamai, M.; Nakamura, H.; Motomura, M.; et al. Anti-citrullinated Protein Antibody-positive Rheumatoid Arthritis Associated with RS3PE Syndrome-like Symptoms and an Elevated Serum Vascular Endothelial Growth Factor Level in a Patient with Myasthenia Gravis. *Intern. Med.* **2014**, *53*, 895–898, <https://doi.org/10.2169/internalmedicine.53.1897>.
33. Angelucci, E.; Cesarini, M.; Vernia, P. Successful resolution of pneumonia developed in a patient affected by Crohn's disease, rheumatoid arthritis, myasthenia gravis and recurrent uveitis during concomitant treatment with tumour necrosis factor  $\alpha$  inhibitors and conventional immunosuppressive drugs. *Rheumatol. Int.* **2009**, *30*, 977–978, <https://doi.org/10.1007/s00296-009-1012-6>.
34. Novella-Navarro, M.; Salvatierra-Ossorio, J.; Muñoz-Gómez, M.d.M.; Pavo-Blanco, M. Artritis reumatoide y miastenia gravis ocular: efectividad del rituximab en el manejo de ambas enfermedades. *Reum. Clin.* **2018**, *14*, 179–180, <https://doi.org/10.1016/j.reuma.2017.05.006>.
35. Oono, M.; Fujita, Y.; Uchida, N.; Kawai, U.; Fujita-Nakata, M.; Nakanishi, M.; Sanada, M.; Nagayama, S.; Matsui, M. Rheumatoid meningitis developed in patient with stable rheumatoid arthritis and myasthenia gravis—detailed analysis of intracranial inflammation using flow cytometry. *J. Neuroinflammation* **2018**, *15*, 151, <https://doi.org/10.1186/s12974-018-1196-3>.
36. Huang, J.; Wang, L.; Hu, X.; Wang, T.; Zhang, Y. Genetic and molecular underpinnings of the link between rheumatoid arthritis and myasthenia gravis: Insights from GWAS and transcriptomic analyses. *Clin. Rheumatol.* **2025**, *44*, 3399–3417, <https://doi.org/10.1007/s10067-025-07582-x>.
37. McInnes, I.B.; Schett, G. The Pathogenesis of Rheumatoid Arthritis. *N. Engl. J. Med.* **2011**, *365*, 2205–2219, <https://doi.org/10.1056/nejmra1004965>.
38. Bhaskar, S.; Rani, M.F.B.A. MuSK-Myasthenia Gravis Unmasked by Hydroxychloroquine. *Case Rep. Med.* **2022**, *2022*, 1–4, <https://doi.org/10.1155/2022/4802538>.
39. Varan, O.; Kucuk, H.; Tufan, A. Myasthenia gravis due to hydroxychloroquine. *Reumatismo* **2015**, *67*, 125, <https://doi.org/10.4081/reumatismo.2015.849>.
40. Goyal, V.; Elavarasi, A. Hydroxychloroquine and myasthenia gravis-can one take this risk? *Ann. Indian Acad. Neurol.* **2020**, *23*, 360–+, [https://doi.org/10.4103/aian.aian\\_363\\_20](https://doi.org/10.4103/aian.aian_363_20).
41. Mantegazza, R.; Antonini, G.; Gastaldi, M.; Liguori, R.; Maestri, M.; Pegoraro, E.; Polistena, B.; Rodolico, C.; Habetswallner, F. Use of Corticosteroids in Myasthenia Gravis: Expert Opinion for Daily Management. *Neurol. Ther.* **2025**, *14*, 2063–2081, <https://doi.org/10.1007/s40120-025-00796-w>.
42. Kanai, T.; Uzawa, A.; Kawaguchi, N.; Oda, F.; Ozawa, Y.; Himuro, K.; Kuwabara, S. Predictive score for oral corticosteroid-induced initial worsening of seropositive generalized myasthenia gravis. *J. Neurol. Sci.* **2019**, *396*, 8–11, <https://doi.org/10.1016/j.jns.2018.10.018>.
43. Visser, K.; Van Der Heijde, D.M.F.M. Risk and management of liver toxicity during methotrexate treatment in rheumatoid and psoriatic arthritis: a systematic review of the literature. *Clin Exp Rheumatol.* **2010**, *27*, 1017–25.
44. Calinoiu, A.; Guluta, E.-C.; Rusu, A.; Minca, A.; Minca, D.; Tomescu, L.; Gheorghita, V.; Minca, D.G.; Negreanu, L. Accessory renal arteries - a source of hypertension: A case report. *World J. Clin. Cases* **2023**, *11*, 1506–1512, <https://doi.org/10.12998/wjcc.v11.i7.1506>.
45. Atallah, E.; Grove, J.I.; Crooks, C.; Burden-Teh, E.; Abhishek, A.; Moreea, S.; Jordan, K.M.; Ala, A.; Hutchinson, D.; Aspinall, R.J.; et al. Risk of liver fibrosis associated with long-term methotrexate therapy may be overestimated. *J. Hepatol.* **2023**, *78*, 989–997, <https://doi.org/10.1016/j.jhep.2022.12.034>.

**Disclaimer/Publisher's Note:** The statements, opinions and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of MDPI and/or the editor(s). MDPI and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions or products referred to in the content.