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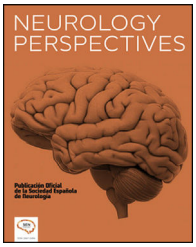

REVIEW

Other autoimmune diseases and multiple sclerosis

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Treatment;
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Abstract
Introduction: This study explores the association between multiple sclerosis (MS) and other autoimmune diseases, including myasthenia gravis (MG), autoimmune encephalitis (AE), and demyelinating polyneuropathies such as combined central and peripheral demyelination (CCPD), chronic inflammatory demyelinating polyneuropathy (CIDP), and Guillain-Barré syndrome (GBS).
Development: For each association, we discuss epidemiological data, clinical features, and therapeutic management strategies. The prevalence of MG is higher in patients with MS than in the general population. Certain MS treatments, such as alemtuzumab, may increase the risk of developing MG or AE. Among the most suitable therapeutic options for patients with coexisting MG or AE are azathioprine and rituximab. Antibody-mediated AE associated with MS is managed similarly to AE unrelated to MS. The association between MS and CIDP contributes to neurological disability and is likely underdiagnosed.
Conclusions: The coexistence of MS with other autoimmune diseases presents significant diagnostic and therapeutic challenges. Awareness of the therapeutic options available for each association is essential in order to identify the most appropriate approach in each case. Early recognition and adequate management of these comorbidities may improve clinical outcomes and enhance patients' quality of life.

PALABRAS CLAVE
Esclerosis múltiple;
Miastenia gravis;
Encefalitis autoinmune;

Otras enfermedades autoinmunes y esclerosis múltiple

Resumen
Introducción: Este artículo analiza la relación entre la esclerosis múltiple (EM) y otras condiciones autoinmunes como la miastenia gravis, la encefalitis autoinmune (EA) y las

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Polineuropatía desmielinizante;
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polineuropatías desmielinizantes (desmielinización combinada central y periférica, polineuropatia crónica desmielinizante idiopática (CIDP) y síndrome de Guillain-Barré).
Desarrollo: Se discuten aspectos tanto epidemiológicos como clínicos y de manejo terapéutico para cada una de estas asociaciones. La prevalencia de MG es mayor en pacientes con EM que en la población general. Algunos tratamientos para la EM, como el alemtuzumab, pueden favorecer la aparición de MG y EA. Probablemente las opciones terapéuticas más adecuadas en pacientes con EM y MG sean la azatioprina y el rituximab. El tratamiento de la EA mediada por anticuerpos asociada a EM no difiere de la EA no asociada a EM. La asociación de la CIDP con la EM contribuye a la discapacidad neurológica y probablemente esté infradiagnosticada.
Conclusiones: La coexistencia de EM con otras enfermedades autoinmunes implica desafíos diagnósticos y terapéuticos. Es importante conocer las alternativas de manejo terapéutico de ambas entidades con el objeto de encontrar la alternativa terapéutica más adecuada para cada asociación. El reconocimiento y el manejo adecuado de estas asociaciones pueden mejorar los resultados clínicos y la calidad de vida de los pacientes.

Multiple sclerosis and myastenia gravis

Epidemiological association

Several studies suggest that the co-occurrence of myasthenia gravis (MG) and multiple sclerosis (MS) is more frequent than would be expected by chance alone. In a cohort from British Columbia, eight patients were identified as having both MS and MG, whereas the expected number based on population estimates would be fewer than one.[1] A recent population-based study conducted in the United Kingdom on autoimmune diseases reported that the risk of developing MG in patients with MS is four times higher than in the general population (incidence rate ratio [IRR]: 4.0 [2.3–6.9]).[2]

Regarding the potential influence of treatments used in MS, the onset of MG has been described after treatment of MS with interferon beta (IFN-β) (3 patients), glatiramer acetate (one patient), and alemtuzumab (one patient).[3] The contribution of these therapies to MG development remains unclear, although the emergence of MG after alemtuzumab treatment is not unexpected, given the well-documented increased risk of secondary autoimmune disorders associated with this drug.[4]

From the perspective of MG, associations have been suggested between the disease and different central nervous system (CNS) autoimmune diseases, such as MS, neuromyelitis optica spectrum disorder (NMOSD), anti–NMDA receptor encephalitis, and Morvan syndrome.[5] The association with Morvan syndrome would be explained by the relationship of both disorders with the presence of thymoma. It is likely that pathogenic autoantibodies play a role in MG, NMOSD, anti-NMDA receptor encephalitis, and Morvan syndrome. However, current evidence suggests this is not the case for MS.

Treatment

Three aspects of treatment should be considered: *1)* treatment for MS relapses and myasthenic crises; *2)* disease-modifying treatment; and *3)* symptomatic treatment.

Treatment of MS relapses and myasthenic crises

In the management of myasthenic crises, treatments such as corticosteroids, intravenous immunoglobulins (IVIG), and plasmapheresis are commonly used and do not appear to exert a detrimental effect on MS. Similarly, neither corticosteroids nor plasmapheresis used for the treatment of MS relapses have been shown to adversely affect MG. The use of intravenous methylprednisolone boluses during myasthenic crises remains controversial; although they appear to be more effective than oral prednisone, some reports suggest an increased risk of steroid-induced myopathy in critically ill patients.[6]

Disease-modifying treatment

Maintenance immunotherapy for MG typically relies on corticosteroids; if an adequate response is not achieved, first-line steroid-sparing agents such as azathioprine or mycophenolate mofetil are commonly employed. In refractory cases, several alternative therapies are available, including rituximab (RTX), eculizumab, methotrexate, tacrolimus, and cyclophosphamide.[7,8]

Both azathioprine and mycophenolate mofetil have been used in the treatment of MS as well.[9,10] Azathioprine is formally indicated for the management of both MS and MG.[11] Another therapeutic option is rituximab (RTX); although not officially approved for either indication in its summary of product characteristics, substantial evidence supports its efficacy in both MG and MS.[12] RTX appears to be particularly effective in MG associated with anti–muscle-specific kinase (MuSK) antibodies. Data on the efficacy of other immunosuppressive agents used in MG for the treatment of MS remain limited.

The use of alemtuzumab in patients with coexisting MS and MG is generally discouraged due to the elevated risk of secondary autoimmunity. Similarly, interferon beta and glatiramer acetate should likely be avoided, given reports of MG onset following their administration in patients with MS.

Symptomatic treatment

Certain drugs used for the symptomatic management of MS may exacerbate MG and should be avoided whenever

Table 1 Treatment for patients with multiple sclerosis and myasthenia gravis.

Treatment for MS relapses and myasthenic crises	
Treatment of choice	• The treatment recommended for each clinical situation (MS relapse or myasthenic crisis) will be used.
Disease-modifying treatments	
Immunotherapy of choice	• Rituximab • Azathioprine
Symptomatic treatment	
Safe treatment	• Fampridine
Use with caution	• Tricyclic antidepressants • Antibiotics: aminoglycosides, macrolides, fluoroquinolones • Baclofen • Benzodiazepines • Beta-blockers • Carbamazepine • Gabapentin/pregabalin • Metamizole
Contraindicated	• Botulinum toxin (spasticity)

MS: multiple sclerosis.

possible. Table 1 summarizes the preferred treatment options for patients with both MS and MG, as well as symptomatic MS therapies that are either contraindicated or potentially aggravating in the context of MG.

Multiple sclerosis and autoimmune encephalitis

Epidemiological association

The term autoimmune encephalitis (AE) refers to a group of immune-mediated brain disorders that are frequently associated with antibodies, some of them pathogenic, against neuronal or glial proteins.[13] More than 20 different types of AE have been described to date. The incidence of AE is similar to that of infectious encephalitis and is estimated at 1.2 cases per 100,000 person-years, with a prevalence of approximately 13.7 cases per 100,000 population.[14]

The most common form of AE is anti–N-methyl-D-aspartate receptor (anti-NMDAR) encephalitis. Clinical manifestations vary by age: seizures, abnormal movements, insomnia, and irritability are more commonly observed in children, while psychosis and behavioral disturbances are more frequent in adults. As the disease progresses, adults are also more likely than children to develop memory impairment and hypoventilation, and less likely to exhibit focal neurological deficits or speech and movement disorders.[15]

A literature review identified 16 reported cases of patients diagnosed with both MS and anti-NMDAR encephalitis.[16] In five of these cases, symptoms of anti-NMDAR encephalitis preceded the onset of MS, whereas in the remaining eleven, MS was diagnosed prior to the development of anti-NMDAR encephalitis. Notably, none of the cases involving both conditions were associated with tumors, in contrast to the general population of women with anti-NMDAR encephalitis, in whom over half present with an underlying ovarian teratoma.

Patients with anti-NMDAR encephalitis may exhibit characteristic demyelinating lesions. In a cohort of 691 patients with anti-NMDAR encephalitis, 3.3% presented extensive or multifocal lesions on T2-weighted FLAIR sequences. Half of these patients experienced at least one demyelinating event separated in time from the anti-NMDAR encephalitis episode. The other half, however, presented with concurrent clinical symptoms and MRI findings consistent with both conditions. These mixed phenotypes suggest the simultaneous activation of two distinct immune mechanisms, a hypothesis further supported by the detection of antibodies against NMDAR, aquaporin-4 (AQP4), or myelin oligodendrocyte glycoprotein (MOG) in 7 of the 11 cases.[17]

Several cases of autoimmune encephalitis (AE) have been reported following treatment with alemtuzumab in patients with MS. The clinical characteristics of these cases are summarized in Table 2.[18–22]

Treatment

Antibody-mediated AE associated with MS is treated in the same way as cases not associated with MS. First-line treatment includes corticosteroids, intravenous immunoglobulins (IVIG), and plasmapheresis. In cases refractory to these therapies, second-line agents such as rituximab and cyclophosphamide are commonly employed.[13,23]

Paraneoplastic AE typically follows a progressive course, at least until treatment of the underlying neoplasm is initiated. Moreover, relapses following tumor treatment are rare. In contrast, patients with AE associated with antineuronal cell-surface antibodies are more likely to experience relapses, occurring in approximately 10%–35% of cases. The decision to initiate long-term immunosuppressive therapy depends on multiple factors, including the presence or absence of an underlying neoplasm, the severity of the initial presentation, the type of associated antibody (if any), and individual patient factors such as treatment tolerance and the potential risks of sustained immunosuppression. In cases of a second episode, the initiation of long-term immunosuppressive therapy is generally less controversial. When indicated, azathioprine, mycophenolate mofetil, or rituximab (RTX) are commonly considered for maintenance treatment.

As sudden discontinuation of immunotherapy is believed to be associated with a high probability of recurrences, several bridging therapy strategies have been proposed. The most common approach involves initiating oral prednisone at a dose of 1–2 mg/kg/day, followed by a gradual taper over weeks or months after the introduction of long-term immunosuppressive therapy, when indicated. An alternative strategy is the periodic administration of maintenance intravenous immunoglobulin (IVIG).[22]

Because the immunosuppressive effects of azathioprine and mycophenolate mofetil typically take several months to manifest, it is recommended to continue corticosteroid therapy for at least 3–6 months following the initiation of long-term immunosuppression. When rituximab (RTX) is selected for long-term therapy, some authors also advocate maintaining oral prednisone during the initial months of RTX treatment to prevent relapses associated with its onset, in line with recommendations established for neuromyelitis optica spectrum disorder (NMOSD).[24]

Table 2 Autoimmune encephalitis after alemtuzumab treatment in patients with MS.

Year of publication	Sex	Age	Time after ALT	Immunological study	Treatment	Outcome
2019[18]	W	45	21 months	Anti-Glu3R antibodies	IVIG	Resolved
2019[19]	W	20	18 months	Negative	IVMP, IVIG, plasmapheresis Ocrelizumab	Resolved
2019[22]	W	20	19 months	Negative	IVMP, IVIG, plasmapheresis	Resolved
2020[20]	M	68	28 months	Anti-GABA-A antibodies	IVMP, IVIG	Resolved
2023[21]	W	22	6 months	Anti-GABA-A antibodies	IVMP, IVIG, plasmapheresis, RTX, ciclosporin	Resolved

ALT: alemtuzumab; IVIG: intravenous immunoglobulins; IVMP: intravenous methylprednisolone; M: man; RTX: rituximab; W: woman.

In refractory cases of AE, alternative therapies such as natalizumab (NTZ), intrathecal methotrexate, bortezomib, daratumumab, anakinra, tofacitinib, and basiliximab have been employed.[25] A phase II clinical trial of NTZ was conducted in patients with AE associated with anti-Hu antibodies. Approximately half of the patients demonstrated a positive response, and NTZ was considered to be as effective as conventional immunotherapy.[26]

Given this context, it appears reasonable to consider the use of anti-CD20 therapies (rituximab or ocrelizumab) as bridging and/or maintenance strategies when clinically indicated. The use of natalizumab (NTZ) may be considered in cases of refractory AE, particularly when associated with anti-Hu antibodies.

In a review of published cases of anti-NMDAR encephalitis associated with MS, additional immunosuppressive agents used included teriflunomide, mycophenolate mofetil, mitoxantrone, and bortezomib. Clinical outcomes were considered favorable in 9 of the 16 reported cases.[16]

A particular scenario is AE occurring as a complication of alemtuzumab treatment in patients with MS. In such cases, early initiation of anti-CD20 therapy (rituximab or ocrelizumab) has been proposed following inadequate response or relapse after initial treatment with corticosteroids, plasmapheresis, and intravenous immunoglobulins.

Combined central and peripheral demyelination

Epidemiological association

Although recognized for many years, the co-occurrence of demyelinating disorders affecting both the central and peripheral nervous systems remains rare. The term combined central and peripheral demyelination (CCPD) is currently used to describe this entity. However, this designation encompasses both acute and chronic or relapsing presentations and likely includes a spectrum of disorders with distinct pathogenic mechanisms.

This designation should be distinguished from entities with a well-defined classification in which concurrent involvement of the central and peripheral nervous systems is frequent. These entities include anti-GQ1b syndrome, immune checkpoint-related toxicity, anti-GFAP astrocytopathy, MOG antibody–associated disease, some paraneoplastic syndromes, POEMS syndrome, some types of histiocytosis, neurological complications of connective tissue diseases, and some forms of systemic vasculitis.[27–31]

Approximately, one-third of cases of CCPD follow a monophasic course, often preceded by a history of infection or vaccination in the preceding weeks.[32–34] This subset of patients, with a presumed post-infectious or post-vaccinal etiology, may represent a distinct nosological group. Therefore, Therefore, the subsequent discussion will focus on CCPD cases with a chronic or relapsing course, which currently lack sufficient differentiation to be universally recognized as a distinct etiopathogenic entity.

An association is known to exist between CIDP and MS. CIDP is the most frequent immune-mediated demyelinating neuropathy. The authors of a series from the Mayo Clinic identified 133 patients with MS and polyneuropathy (PNP), 11 of whom were diagnosed with MS and CIDP.[35] The onset of CIDP has been described as a complication of autologous haematopoietic stem cell transplantation in patients with MS.[36] The presence of PNP in patients with MS contributes to neurological disability and is probably underdiagnosed due to the overlapping of symptoms; however, abolished or diminished tendon reflexes constitute a clinical sign that may lead us to suspect CIDP in a patient with MS. This finding was observed in all patients with MS and CIDP. Another sign that may raise suspicion of CIDP in a patient with MS is elevated CSF protein levels and the presence of spinal root and cranial nerve hypertrophy in imaging studies. The possibility of associated PNP should be considered in all patients with MS and hyporeflexia or areflexia. Diagnosis of CIDP is important because it is a treatable disease and additional immunotherapy (for example, intravenous immunoglobulins) may improve neurological disability and quality of life.

A subset of patients with CIDP present antibodies targeting nodal or paranodal proteins, including neurofascin-155, neurofascin-186, contactin-1, and contactin-associated protein 1 (CASPR1). Notably, neurofascin-155, contactin-1, and CASPR1 are also expressed in the central nervous system (CNS). These antibodies are typically of the IgG4 subclass.[37] Subclinical CNS demyelination is not uncommon in patients with neurofascin-155 IgG–associated CIDP. It has been proposed that the presence of CNS demyelination in CIDP may be indicative of underlying anti–neurofascin-155 antibodies. In one of the three available serum samples from the Mayo Clinic series of patients with coexisting MS and CIDP, antibodies against neurofascin-155 were detected, supporting the hypothesis of a shared pathogenic mechanism involving both glial and Schwann cells.[35] A Japanese study identified CNS demyelination in 8% of patients with CIDP positive for anti–neurofascin-155 antibodies.[38] These

antibodies have been reported in approximately 10% of patients with MS and in up to 86% of those with combined central and peripheral demyelination (CCPD).[39] Further supporting CNS involvement, visual evoked potential (VEP) abnormalities are more frequently observed in patients with anti–neurofascin-155 antibody–positive CIDP (70%–80%) compared to the overall CIDP population (40%–50%).[40]

Treatment

Patients with anti–neurofascin-155 IgG antibody–associated CIDP present poorer response to IVIG, and the use of RTX should be considered; the effectiveness of this treatment has been demonstrated in cases refractory to conventional immunotherapies.[40,41] The use of fingolimod for the treatment of CIDP has not shown clinical benefit.[42] For the management of CCPD, treatment strategies have included corticosteroids, IVIG, plasmapheresis, and RTX.[43] Ocrelizumab may also represent a therapeutic option, as it appears to be effective in some patients with CIDP and CCPD.[44]

Multiple sclerosis and Guillain-Barré syndrome

Epidemiological association

Clinical cases of patients with MS who subsequently develop Guillain-Barré syndrome (GBS) have been reported.[45,46] A population-based study conducted in Iran, involving 3,522 patients with MS (2,716 women and 806 men), identified seven individuals (six women and one man) with a confirmed diagnosis of GBS. In six cases (five women and one man), MS developed after the diagnosis of GBS, while in one case, GBS occurred three years after the diagnosis of MS.[47] A case–control study conducted in the United States, based on health insurance data, found a stronger-than-expected association between MS and GBS, with an odds ratio of 5.0 (95% CI, 1.6–15.4).[48] It has been suggested that this association may be linked to a shared pathogenesis, possibly involving Epstein-Barr virus (EBV) infection; however, the literature to date includes only isolated case reports.[49]

Treatment

Treatment of GBS is based on the use of IVIG and plasmapheresis. There is no evidence suggesting that these treatments may negatively affect the course of MS.

Patient informed consent

No patient data are reported.

Ethical considerations

Not applicable.

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None.

Conflicts of interest

None.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.neurop.2025.100196.

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