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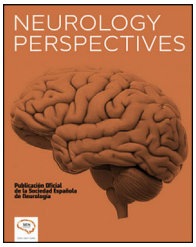

REVIEW

Autoimmune skin diseases in multiple sclerosis

M. Roncero Riesco[a], **A. Cabanillas Cabral**[a], **Y. El Berdei Montero**[b,*]

[a] *Servicio de Dermatología, Complejo Asistencial Universitario de Salamanca, Spain*
[b] *Servicio de Neurología, Complejo Asistencial Universitario de Salamanca, Spain*

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Abstract
Introduction: Autoimmune dermatological diseases have a prevalence greater than 2% in the general population, sometimes as a primary disorder and other times within a context of systemic involvement. Comorbidity with multiple sclerosis (MS) has been described, particularly in the case of psoriasis and bullous pemphigoid, and to a lesser extent in pemphigus vulgaris and other autoimmune skin diseases.
Development: Psoriasis is the autoimmune skin disease for which the most evidence is available on this association, with increased risk in patients with MS. Both disorders probably have common pathophysiological mechanisms. The joint treatment of both diseases will depend on the degree of activity of each one, but in general, it is recommended for patients with MS and psoriasis to avoid interferons, teriflunomide, and anti-CD20 monoclonal antibodies, whereas fumarates and S1P receptor antagonists are recommended. TNF-α inhibitors are formally contraindicated in MS. In the case of bullous pemphigoid, pemphigus vulgaris, and other less common autoimmune dermatological diseases, the relationship with MS is not so clearly established, although an association between the first 2 and neurological diseases, including MS, has been described. Treatment is based on corticotherapy, and classic immunosuppressants or rituximab may be combined, which represent an alternative for joint treatment.
Conclusions: Comorbidity between MS and autoimmune dermatological disorders, and especially psoriasis, requires a joint approach, avoiding treatments that may aggravate one or the other disorders.

* Corresponding author.
E-mail address: yberdei@saludcastillayleon.es (Y. El Berdei Montero).

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PALABRAS CLAVE
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Penfigoide

Patología cutánea autoinmune en esclerosis múltiple

Resumen
Introducción: Las enfermedades dermatológicas autoinmunes presentan una prevalencia superior al 2% en la población general, a veces como patología primaria y otras dentro de un contexto de afectación sistémica. La comorbilidad con la esclerosis múltiple (EM) se ha descrito especialmente en el caso de la psoriasis y del penfigoide ampolloso, y en menor medida en el pénfig. vulgar y otras enfermedades cutáneas autoinmunes.
Desarrollo: La psoriasis es la patología en la que existe más evidencia de asociación, con incremento del riesgo en los pacientes con EM. Probablemente ambos trastornos comparten mecanismos fisiopatogénicos. El tratamiento conjunto de ambas enfermedades dependerá del grado de actividad de cada una de ellas, pero en general se recomienda evitar en los pacientes con EM y psoriasis los interferones, la teriflunomida y los anticuerpos monoclonales antiCD20 y se recomiendan los fumaratos o los antagonistas del receptor de S1P. Los fármacos antiTNF están formalmente contraindicados en la EM. En el caso del penfigoide ampolloso, el pénfig. vulgar y otras enfermedades dermatológicas autoinmunes menos frecuentes la relación con la EM no está tan claramente establecida, aunque sí se ha descrito asociación entre los dos primeros con enfermedades neurológicas, entre ellas la EM. La base del tratamiento es la corticoterapia, pudiendo asociarse inmunosupresores clásicos o rituximab, que suponen una alternativa para el tratamiento conjunto.
Conclusiones: La comorbilidad entre la EM y patologías dermatológicas autoinmunes, especialmente en el caso de la psoriasis, supone la necesidad de realizar un abordaje conjunto, evitando los tratamientos que puedan agravar uno u otro trastorno.

Introduction

The study and clinical practice of dermatology includes a wide variety of inflammatory disorders, some of which are of autoimmune origin. These include diseases with primary cutaneous expression, as well as others in which skin involvement is one of a series of signs of systemic involvement. Comorbidity with multiple sclerosis (MS) is particularly frequent in psoriasis and bullous pemphigoid, and less common in pemphigus vulgaris and other autoimmune skin diseases.

Psoriasis

Psoriasis is an autoimmune disease associated with a significant polygenic burden, whose classic lesion consists of reddish plaques associated with thick scaling and well-defined edges; extension varies according to severity (Fig. 1). Prevalence in the general population is approximately 2%. The disease presents a broad variety of triggers and exacerbating factors, as well as various associations with other immune-mediated disorders, including MS. Numerous studies in the literature have analysed the relationship between psoriasis and MS, with the majority finding an association between the 2 diseases. A recent meta-analysis reported a hazard ratio (HR) of 1.92 (95% confidence interval [CI], 1.32–2.80) for the risk of psoriasis in patients with MS.[1]

Several pathophysiological factors common to both disorders have been described. Adaptive immunity based on the Th1 and Th17 responses plays a fundamental role, with the production of different proinflammatory cytokines. In psoriasis, a key role is played by tumour necrosis factor

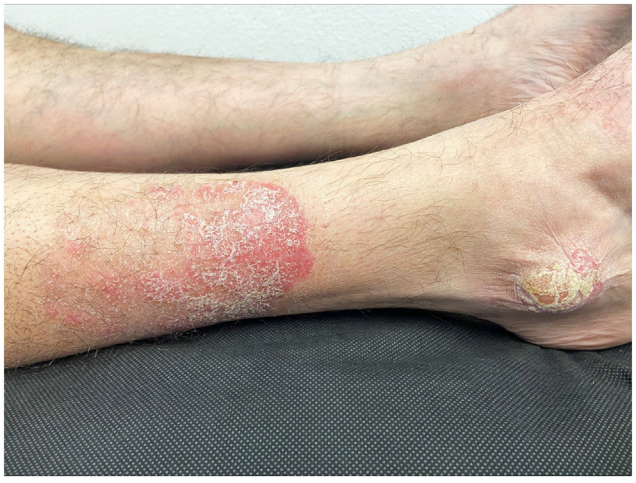

Figure 1 Plaque psoriasis in a patient with multiple sclerosis, who was receiving no treatment for either disease. The patient also presents Koebner phenomenon (plaque affecting the maleolus externus due to pressure).

alpha (TNF-α), as well as interleukins (IL) 17 and 23, which in turn represent important therapeutic targets.[2]

Some genetic polymorphisms have also been found to increase an individual's likelihood of developing both MS and psoriasis; genes encoding human leukocyte antigen proteins, such as HLA-DRB1 (which has an important role in susceptibility to psoriatic arthritis), as well as IL-12B, are particularly relevant.[3,4] In addition to endogenous factors, environmental factors can also jointly influence the pathogenesis of both diseases; the most relevant example is vitamin D deficiency, although this is controversial as supplementation has not been shown to clearly improve prognosis in either disease.[5]

Therapeutic considerations

According to management guidelines and the main treatment algorithms for psoriasis, and particularly if biological treatment is indicated, TNF-α inhibitors are formally contraindicated due to the potential to trigger relapse activity in demyelinating diseases (both MS and peripheral neuropathies).[6] Symptomatic relapses after treatment onset have been described after starting all drugs from this family used in the treatment of psoriasis (golimumab, certolizumab, infliximab, adalimumab, and etanercept); therefore, this can be considered an adverse class effect. The majority of adverse events occur in the first year after treatment onset, and approximately two-thirds of patients do not completely recover the affected functions despite withdrawal of the drug responsible. The European guidelines for the management of psoriasis extend this contraindication to patients with first-degree relatives with any type of demyelinating disorder. Such other biological agents as ustekinumab (an IL-12/IL-23 inhibitor), IL-17 inhibitors (secukinumab, brodalumab, ixekizumab), and selective IL-23 inhibitors (guselkumab, tildrakizumab, risankizumab) appear to be safer in this context.

Although biological therapy currently represents one of the central pillars of psoriasis management, the broad therapeutic arsenal available to dermatologists also includes such traditional treatments as ciclosporin, methotrexate, and dimethyl fumarate. Although the summary of product characteristics for ciclosporin describes some forms of neurotoxicity among the possible adverse effects, both this drug and methotrexate are appropriate options, and their use is not associated with poorer disease control in MS.[7]

However, dimethyl fumarate is considered the optimal drug for joint treatment of both diseases, in cases in which MS activity permits this approach, as it is a moderate-efficacy drug that is indicated for relapsing–remitting forms. Its action mechanism is poorly understood, but favours the differentiation of T-cells to produce Th2 cytokines, reducing activation of the Th1 and Th17 immune pathways.[8] It should be noted that the therapeutic dose may differ between MS and psoriasis.

Another moderate-efficacy drug for MS is teriflunomide, the active metabolite of the prodrug leflunomide, which is approved for the treatment of psoriatic arthritis, and has shown benefits for skin lesions in some cases. However, at least 3 cases have been published since 2017 of patients who developed psoriasis during treatment with teriflunomide; therefore, the drug has a relative contraindication for the treatment of both diseases, when these co-present.[9]

Regarding glatiramer acetate, there is no published evidence that the drug has beneficial or harmful effects in psoriasis. On the other hand, interferon beta is not recommended, as it has been reported to exacerbate and even to trigger psoriasis in some studies.[10]

In cases where the clinical characteristics of multiple sclerosis require treatment escalation or initial treatment with high- or very high–efficacy drugs, it should be noted that sphingosine 1-phosphate receptor antagonists, and particularly ponesimod, are reported to have benefits in psoriasis, with a phase II clinical trial for treatment of psoriasis with and without arthritis reporting good efficacy and safety results at doses of 20 and 40 mg/day.[11] Anecdotally, positive results are reported in small groups of patients with psoriasis treated with cladribine, although with a different treatment regimen than that used in MS. The evidence on natalizumab and alemtuzumab is even more controversial, with several studies reporting contradictory results. The literature includes cases of atypical forms of psoriasis, such as palmoplantar pustulosis, in patients receiving natalizumab treatment.[12] However, other authors have reported cases of patients with psoriasis and MS whose cutaneous symptoms improved after the onset of natalizumab treatment, with several patients not needing any additional treatment for psoriasis during the follow-up period.[13] One possible explanation for the improvement in psoriasis in these patients may be the action mechanism of the drug: it inhibits subunit alpha 4 of various integrins, hindering lymphocyte circulation in the central nervous system. However, this also prevents the migration of these cells to the skin, and therefore the onset of the mechanisms triggering hyperplasia of the keratinocytes responsible for psoriatic lesions. In the light of these contradictory findings, natalizumab also has a relative contraindication in the treatment of MS with comorbid psoriasis.

Very high–efficacy treatments for MS include the anti-CD20 antibodies ofatumumab and ocrelizumab, as well as rituximab (often used off-label for MS treatment). Ocrelizumab is also the first treatment to be approved for active and progressive forms of MS. Increased prevalence of psoriasis and concomitant MS is reported among patients receiving anti-CD20 treatment.[14] These drugs achieve B-cell depletion; though this does not directly entail potentiation of T-cell activity, their action mechanism is able to induce interferon beta in patients receiving the drug. It has been suggested that the greater concentration of this cytokine may be responsible for the increase in cases of psoriasis among patients receiving these treatments, similar to the cases observed in patients receiving interferon directly as a disease-modifying treatment. Nonetheless, although this group of drugs should be used with caution in patients with psoriasis, the final treatment decision must be made on an individual basis, considering the risk/benefit balance as a function of the activity and severity of both diseases and all other relevant factors (MS phenotype, JC virus serostatus, intention to become pregnant, etc).

Drugs with greater efficacy in treating psoriasis include the monoclonal antibodies secukinumab and ustekinumab, with reports of successful joint treatment of both diseases for both drugs.[15] Secukinumab is a fully human monoclonal

antibody that selectively binds to IL-17, inhibiting its interaction with its receptor. In psoriasis, it can be used in monotherapy or in combination with methotrexate or dimethyl fumarate; in MS, a phase II clinical trial has presented robust radiological evidence of its efficacy[16]; therefore, combined use of these drugs may be considered in patients with more aggressive disease.

Table 1 summarises the possible therapeutic alternatives and precautions to be taken regarding the different disease-modifying drugs for MS with comorbid psoriasis.

Bullous pemphigoid

Bullous pemphigoid is an autoimmune disease that typically appears in old age, predominantly in patients older than 70 years, with incidence increasing after the age of 90 years. The characteristic lesion is a reddish oedematous plaque with associated tense bullae, typically without mucosal involvement (although this varies between clinical variants). With an incidence of approximately 21.7 cases per million population, it represents the most frequent autoimmune blistering skin disease in the general population.[17]

This disease presents an important association with several neurological disorders or different aetiologies, such as Parkinson's disease, dementia, epilepsy, and cerebrovascular disease. However, the strongest association appears to be with MS, according to several studies. The relative risk of developing MS is 12.40 (95% CI, 6.64–23.17) in patients with bullous pemphigoid, compared to 4.93 (95% CI, 3.62–6.70) for the above-mentioned neurological diseases.[18] In the majority of cases, neurological disease is present prior to the development of the blistering cutaneous disorder. In fact, one study reports the history of neurological disease before diagnosis of bullous pemphigoid in approximately 55% of patients developing the disease.[19] Some drugs, such as TNF-α inhibitors, have been reported to trigger the appearance of both diseases.[20]

Although the relationship between these 2 nosological entities has not been fully clarified, some hypotheses have been proposed. Antibodies targeting BPAG1 (BP180 antigen) and BPAG2 (BP230 antigen) play a fundamental role in bullous pemphigoid pathogenesis. While these antigens are largely present in collagen XVII in the desmosomes of basal keratinocytes, enabling them to anchor to the basement membrane of the dermoepidermal junction, they are also present in central nervous system tissues. However, anti-BPAG1 antibodies target different epitopes in MS and in bullous pemphigoid.[21] Certain genetic factors, such as various isoforms of HLA-DRB1, are also reported to increase susceptibility to bullous pemphigoid, as well as psoriasis and MS.[22]

Overall, there seems to be a need for further research to clarify the relationship between MS and other neurological disorders and bullous pemphigoid, with particular emphasis on the aetiopathogenic reasons for the copresence of these diseases in a single patient.

Therapeutic considerations:

The treatment of bullous pemphigoid is based on systemic corticotherapy, which may be complemented by classical immunosuppressants; some of these, such as azathioprine, methotrexate, and mycophenolate mofetil, may in turn have certain benefits for MS. Regarding refractory bullous pemphigoid, case reports and retrospective studies have been published of safe and efficacious treatment with rituximab, administered alone or in combination with other treatments including classical immunosuppressants and intravenous immunoglobulins. Despite this, and unlike pemphigus vulgaris, bullous pemphigoid is an off-label indication for the drug.[23] Anecdotally, a single case was reported of a patient with MS and bullous pemphigoid who presented a complete response to treatment with dimethyl fumarate.[24]

Multiple other drugs have been reported to trigger or to increase susceptibility to bullous pemphigoid, although this association has not been reported for disease-modifying therapies for MS.

Pemphigus vulgaris

Pemphigus vulgaris is the second most frequent autoimmune blistering skin disease, after bullous pemphigoid, with an incidence rate of 0.5–8 cases per million person-years in Spain.[25] This disease typically appears at younger ages than bullous pemphigoid: it may appear in any age group, typically in the fourth/fifth decade of life; in this respect, it presents a greater epidemiological similarity with MS than

Table 1 Summary of the recommended treatments for multiple sclerosis in patients with comorbid psoriasis. DMT: disease-modifying therapy.

	DMT of choice	Alternative DMT	Contradictory or lack of evidence	Caution required
Moderate-efficacy DMTs	Dimethyl fumarate 240 mg, 2 times/day	Methotrexate* 7.5 mg/week	Glatiramer acetate 20 mg/24 h 40 mg, 3 times/week	Interferon beta Teriflunomide
High–/very high–efficacy DMTs	Ponesimod 20 mg/day	Fingolimod 0.5 mg/day	Ozanimod/siponimod	Ocrelizumab
			Natalizumab	Ofatumumab
	Secukinumab 300 mg/month*	Cladribine 3.5 mg/kg over 2 years	Alemtuzumab	Rituximab*

* Off-label indication.

does bullous pemphigoid, which predominantly manifests in elderly patients.

Though there are numerous variants of the disease, the most frequent clinical form is characterised by flaccid bullae with serous content and positive Nikolsky sign, leaving areas of denuded skin. It typically presents with predominant mucosal involvement, which has a considerable impact on the patient quality of life, with painful erosions in the mouth and throat.

A recent study analysing the relationship between pemphigus vulgaris and neurological disease observed a statistically significant association between the former and such disorders as Parkinson's disease, dementia, and epilepsy. However, the association with MS was not statistically significant, probably due to the small sample size, with an odds ratio of 1.67 (95% CI, 0.36–8.20).[26] The authors of that paper proposed some hypotheses about the association between the 2 entities. As in the case of bullous pemphigoid, some of the proteins targeted by the antibodies responsible for pemphigus vulgaris (primarily desmoglein 1) present isoforms that are expressed in the central nervous system, specifically in the corpus callosum and in the plasma membrane of some oligodendrocytes. Therefore, cross-reactivity between the cutaneous and neurological isoforms may play a role in this association.

Regarding treatment, in more complex cases that are generally refractory to corticotherapy, rituximab (an anti-CD20 drug) has been suggested as an ideal drug with a good response rate, and may be useful in controlling both diseases when these co-present in a single patient.[27]

Other autoimmune skin diseases

No statistically significant association has been established between MS and other autoimmune skin diseases (principally blistering skin diseases: pemphigus vulgaris, gestational pemphigoid, pemphigus foliaceus, mucous membrane pemphigoid, and epidermolysis bullosa acquisita). However, these diseases have been associated with other neurological diseases, such as dementia, epilepsy, stroke, and Parkinson's disease.

Regarding autoimmune diseases that cause erythema and desquamation, only 3 cases have been reported of lichen ruber planus in patients with MS; therefore, a significant association between the 2 diseases could not be established.[28]

Cases have also been published of MS associated with sclerodermiform disease, although the relationship between these entities has not been clearly demonstrated. It has been suggested that interferon treatment for MS may play some role. A recent article reported a patient with MS and concomitant hepatitis C virus infection, who was receiving injections of IFNβ-1b to control the former disease, and who developed morphea plaques at the injection site.[29]

Finally, some drugs used to treat MS have been implicated in the development of systemic lupus erythematosus (SLE) with subcutaneous manifestations. The specific drugs described include natalizumab, teriflunomide, and IFN. Furthermore, cases have been described of MS associated with SLE of non-pharmacological origin.[30] These patients typically present mild SLE, with predominantly cutaneous and musculoskeletal presentation.

Conflicts of interest

The authors have no conflicts of interest to declare in relation to this article.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.neurop.2025.100200.

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