Dermatology Practical & Conceptual

Targeted Therapies and Pharmacologic Advances in Mucous Membrane Pemphigoid: A Comprehensive Review

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ABSTRACT

Introduction: Mucous membrane pemphigoid (MMP) is a rare autoimmune bullous dermatosis which predominantly affects the mucous membranes and, occasionally, the skin. The exact pathogenesis of MMP remains unclear and should be considered as a unique phenomenon, which involves the formation of subepithelial blisters and fibrosis.

Objectives: This narrative review aimed to summarize the pharmacological agents which showed efficacy in the management of MMP but that are not included in the guidelines.

Methods: We conducted a search on Google Scholar, PubMed, and the Web of Science databases concerning articles published in English on the management of MMP between January 2000 and February 2025; all the sourced articles were full-text reviewed.

Results: We included 13 articles. The studied pharmacological agents are classified as immunosuppressive agents (leflunomide, sirolimus, daclizumab) and biologics (daclizumab, dupilumab, omalizumab, bevacizumab, aflibercept, cenegermin); the immunosuppressant leflunomide and the antimalarial agent hydroxychloroquine are also classified as disease-modifying antirheumatic drugs. Other pharmacological agents (colchicine, corticotropin, varenicline, lifitegrast) exert miscellaneous mechanisms.

Conclusion: Considering the severity of the condition, progressive fibrosis, and resistance to therapy, more research is required in relation to the pathogenesis of MMP and the efficacy and safety profile of novel pharmacological options. Pharmacological agents should provide the achievement and maintenance of remission with minimal adverse effects. A broader spectrum of pharmacological agents will allow a personalized approach and more alternatives, in particular for recalcitrant cases, failure of the previous therapy, and in patients with MMP and malignancy.

Introduction

Mucous membrane pemphigoid (MMP) is a rare autoimmune bullous dermatosis (AIBD) which predominantly affects mucous membranes and the skin. Generally, MMP has a late onset, and the mean age of patients ranges from 60 to 80 years; the association between the condition and HLA-DQB1*0301, HLA-DRB1*1101, HLA-DQA1*0505 alleles has been notes noted.

The pathogenesis of MMP remains unclear and involves the formation of subepithelial blisters and fibrosis. The formation of blisters is linked to the production of antibodies (IgG and/or IgA) against the basement membrane proteins BP230 and BP180, α6 and β4 integrin subunits, laminin-332, laminin-6, type VII collagen. The activation of fibroblasts and hyperproduction of collagen may be associated with the dysregulation of immunity, excessive release of pro-inflammatory molecules, and proteolytic enzymes [1]. Transforming growth factor beta (TGF-β) plays a major role in fibrosis, and an increased expression of TGF-β and alpha-smooth muscle actin were observed in conjunctiva samples taken from patients with MMP [2].

MMP develops through three distinct phases: injury, acute inflammation and proliferation, and fibrosis. Subepithelial tense blisters precede erosions and scarring. Histologically, the separation of the epithelium from the underlying connective tissue, leukocyte infiltrates, granulation tissue, and fibrosis may be seen. Direct immunofluorescence demonstrates peculiar linear depositions of immunoglobulins (IgG and/or IgA) and/or complement C3 along the basal membrane; indirect immunofluorescence (salt-split) and immunoserological tests detect pathogenic autoantibodies [1].

Since 2002, several guidelines have evolved on the management of MMP. The First International Consensus on MMP [3] and the guidelines of the Brazilian Society of Dermatology [4] and the Japanese Dermatological Association [5] classify patients as low risk (oral and/or cutaneous lesions) or high risk (ocular, pharyngeal, laryngeal, esophageal, and/or genital involvement), with adapted treatment options depending on the risk category; the guidelines of the French Society of Dermatology [6] are based on the location and disease severity. The European Guidelines (S3) [7] and the S2k guideline [8] offer lines of therapy, alternative combinations, and treatment options, depending on the clinical course, location, and response. Given the risk of disease progression and complications, early initiation of immunosuppressive therapy is of vital importance; in severe cases, surgical treatment is considered. Nonetheless, there is no gold standard for treatment.

This narrative review aimed to summarize the pharmacological agents which showed efficacy in the management of MMP but that are not included in the earlier published guidelines.

We conducted a search on Google Scholar, PubMed, and the Web of Science databases concerning articles published in English on the management of MMP between January 2000 and February 2025, using the search terms “mucous membrane pemphigoid”, “cicatricial pemphigoid”, “Brunsting-Perry pemphigoid”, “management”, “guidelines”. All the sourced articles were full-text reviewed. The criteria for inclusion of the articles were publication in English and the use of pharmacological options not included in the published guidelines.

Drug Review

We included 13 articles in English in which the diagnosis of MMP was verified and the data on the capacity to induce disease control and improvement and the absence of reported severe adverse effects of the suggested pharmacological options were available (Table 1).

The studied pharmacological agents are classified as immunosuppressive agents (leflunomide, sirolimus, daclizumab) and biologics (daclizumab, dupilumab, omalizumab, bevacizumab, aflibercept, cenegermin); the immunosuppressant leflunomide and the antimalarial agent hydroxychloroquine are also classified as disease-modifying antirheumatic drugs. Other groups of pharmacological agents (colchicine, corticotropin, varenicline, lifitegrast) exert miscellaneous mechanisms, which are specified bellow.

Interleukin (IL)-2 is a pro-inflammatory cytokine, also known as T cell growth factor; positive expression of IL-2 is observed in T cells, infiltrating the MMP-affected conjunctiva [9]. Daclizumab, a monoclonal anti-CD25 antibody, binds to the human IL-2 receptor (IL-2Rα) in the IL-2Rα

Table 1. Pharmacological Agents Reported in the Management of Mucous Membrane Pemphigoid.

Drug	Drug class	Condition	No. of reported patients	No. of patients with improvement	References
Omalizumab	Monoclonal anti-IgE antibody	MMP	5	3	Alexandre et al. (2022)
Daclizumab	Monoclonal anti-CD25 antibody / IL-2R antagonist	OcMMP	1	1	Papaliodis et al. (2003)
Dupilumab	IL-4/IL-13-inhibitor	MMP BPP	1 1	1 1	Wang et al. (2024) Raef et al. (2021)
Cenegermin	rhNGF	OcMMP	1 1	1 1	Abbot et al. (2024) Surico et al. (2024)
Colchicine	Anti-mitotic agent	OrMMP MMP	6 12	5 8	Fribourg et al. (2024) Chaidemenos et al. (2011)
Leflunomide	Pyrimidine synthesis inhibitor	OcMMP MMP	1 1	1 1	Smichowski et al. (2020) Boedeker et al. (2003)
Hydroxychloroquine	Antimalarial agent	OcMMP BPP	1 1	1 1	Morel et al. (2022) Guerrero et al. (2025)
Corticotropin	Adrenocorticotropic hormone formulation	OcMMP	15	9	Sharon et al. (2022)
Varenicline	Selective nAChR partial agonist	OcMMP	4	1	Abbot et al. (2024)
Lifitegrast	ICAM-1 and LFA-1 antagonist	OcMMP	10	2	Abbot et al. (2024)

ectodomain, the binding site of IL-2 to immune cells, and inhibits B cell proliferation and IL-2-mediated T cell activation. Papaliodis et al. (2003) reported one patient diagnosed with ocular MMP (OcMMP) and successfully treated with daclizumab [10].

An increased expression of IL-4 was noted in conjunctival sections of patients with OcMMP and in isolated fibroblasts; IL-4 may exert a modulatory effect on macrophages via inducing m-CSF as well as uncontrolled extracellular matrix accumulation [11]. An elevated stromal expression of IL-13 is seen in active OcMMP, suggesting profibrotic and pro-inflammatory effects on human conjunctival fibroblasts [12]. Dupilumab, a recombinant fully human IgG4 monoclonal antibody, suppresses inflammatory signaling via binding to the IL-4Rα subunit of IL-4 and IL-13 receptor complexes. Dupilumab combined with systemic corticosteroids was reported as effective in a patient with multisite MMP (oral, genital, and cutaneous involvement) [13] and Brunsting-Perry pemphigoid [14].

The circulation of sera IgE autoantibodies against the BM components [15,16] as well as linear IgE depositions along BM on immunofluorescence [15,17] have been observed in patients with MMP. Omalizumab is a humanized monoclonal anti-IgE antibody which neutralizes total serum IgE via forming interactions with IgE Cε3 domains outside the FcεRI-binding site and inhibiting FcεRI and CD23 binding. Omalizumab was reported to induce complete remission in three out of 5 patients with MMP (with oral, laryngeal, and genital MMP-related lesions) [18].

Neutrophils are present in inflammatory infiltrates at the acute stage and persist in MMP-affected tissues even when inflammation is visually resolved [9,19], and the involvement of other mechanisms (i.e., neutrophil recruitment) has been suggested in the pathogenesis of MMP [20]. Colchicine, a bioactive plant alkaloid derived from *Colchicum autumnale*, is commonly used in gout flares, familial Mediterranean fever, Behçet's disease, psoriasis, Sweet's syndrome, and erythema nodosum [21]. Colchicine inhibits neutrophil recruitment and disrupts the NLRP3 inflammasome assembly in neutrophils and monocytes through the inhibition of cellular P2X7 receptors; the suppression of β-tubulin polymerization and synthesis of microtubules is related to anti-mitotic properties [22]. Colchicine has been offered as a second-line option in oral MMP [23]; earlier colchicine was also suggested as a first-line corticosteroid-sparing agent in MMP [24]. Adverse effects (AEs) of colchicine include cardiomyopathy, cytopenia, elevation of hepatic transaminases, maculopapular rash, and alopecia [21].

Hydroxychloroquine, an antimalarial and antirheumatic agent, exerts anti-inflammatory action via the disruption of antigen presentation in the background of intracellular pH alteration, processing of antigenic protein, and formation of the alpha and beta chains of major histocompatibility complex class II proteins, reducing T cell stimulation and immune responses [25]. Hydroxychloroquine has been reported to induce disease control in MMP [26] and Brunsting-Perry pemphigoid [27]; however, retinopathy, as well as cardiac and auditory toxicity [28] and dermatological AEs, in particular in autoimmune conditions [29], are linked to hydroxychloroquine.

Leflunomide is an isoxazole derivative and pyrimidine synthesis inhibitor used in MMP. The drug inhibits the mitochondrial enzyme dihydroorotate dehydrogenase, disrupting de novo synthesis of pyrimidine ribonucleotide uridine monophosphate, required for the progression from G1 to S phase in activated lymphocytes; these cytostatic and anti-proliferative effects may contribute to disease control in patients with MMP [30,31]. Diarrhea, elevation of liver enzymes, rash, and alopecia are the most frequent AEs of leflunomide in rheumatoid arthritis [32].

Melanocortins exert anti-inflammatory effects via the stimulation of glucocorticoid-dependent and glucocorticoid-independent pathways, reducing the release of pro-inflammatory cytokines and ROS. Repository corticotropin injection (RCI) was earlier offered for systemic lupus erythematosus, given the reduction of B cell proliferation (possibly, via suppressing the activity of IL-6 and B-cell-activating factor of the tumor necrosis factor (TNF) family signaling) [33]. Furthermore, RCI has been suggested as a safe and well-tolerated alternative or adjunctive option for patients with severe and refractory OcMMP, inducing improvement in 60% of cases; no serious AE has been reported [34].

The recombinant human nerve growth factor (rhNGF) cenegermin improves corneal healing via binding to the tyrosine kinase receptor TrkA and to p75NTR, promoting neuronal and epithelial cell growth. Abbot et al. (2024) reported the resolution of MMP-associated large, non-healing corneal defect in two patients treated with cenegermin [35,36], but Surico et al. (2024) highlighted patient monitoring due to the formation of corneal superficial plaque linked to posttreatment epithelial hyperplasia [36].

Sirolimus, a mammalian target of rapamycin (m-TOR) inhibitor, suppresses cytokine and growth factor receptor signaling and T cell progression from G1 to S [30]. Sirolimus was reported in combined and rotation therapy in OcMMP [37]. On the other hand, sirolimus-induced bullous pemphigoid was reported in two renal transplant recipients [38]; consequently, sirolimus should be employed with caution in autoimmune skin blistering diseases (AIBDs).

Vascular endothelial growth factor (VEGF) is involved in immune responses and proliferation, exerting signaling by binding to tyrosine kinase receptors, and bevacizumab and aflibercept neutralize VEGF via forming interactions with the receptor-binding regions. Subconjunctival and intravitreal injections of bevacizumab and aflibercept [39,40] have been reported in OcMMP; in the mentioned cases, these agents did not alter the severity of the disease course. However, hypothetically, given their capacity to suppress angiogenesis and tissue remodeling, these agents may represent an opportunity to improve quality of life of patients with MMP as adjuvant therapy, although clinicians must be aware of drug-induced MMP [1].

Dry eye disease is frequently associated with OcMMP. Varenicline, a selective nicotinic acetylcholine receptor (nAChR) partial antagonist, induces the basal tear film formation by the lacrimal gland via the stimulation of the trigeminal parasympathetic nerve pathway [41]. Lifitegrast binds to the integrin lymphocyte function-associated antigen-1, preventing its interaction with the intercellular adhesion molecule-1, blocking the formation of immunological synapse, T cell activation, and suppressing inflammatory cascades [42]. These agents are employed in dry eye disease; however, their efficacy was limited in OcMMP [35].

Mucous Membrane Pemphigoid and Malignancy

In MMP, malignancy is reported in 15.5% of cases [43], while in patients with sera anti-laminin 332 antibodies (anti-laminin-332 MMP), the malignancy rate is 21–21.8% [43,44]. Laminin-332 is a structural glycoprotein of BM which is involved in extracellular matrix formation, cell proliferation and migration; its elevation has been observed in some tumors and may trigger autoimmune reactions and cross-reactivity with BM proteins [45,46]; thus, various studies highlight an elevated malignancy risk in anti-laminin-332 MMP [44,47]. Another issue is MMP induced by malignancy treatment (e.g., radiation [48] and immune checkpoint inhibitors [49]).

The current guidelines do not contain sections on the management of anti-laminin-332 MMP or of MMP in patients with a history of previous or concurrent malignancy, and the selection of therapy depends on lesion location and severity of the disease course. The use of immunosuppressive agents raises concerns over tumorigenesis risks [43,50,51], and several studies warn about elevated malignancy risks [52]. On the other hand, malignancy was detected before establishing the diagnosis of MMP in 47.1% of patients [43], and immunosuppressive agents have also been reported to have antineoplastic effects [51,53,54,55]. Corticosteroids remain among the most frequently indicated drugs in oncological conditions [56]. In paraneoplastic pemphigus, another autoimmune blistering disease, the guidelines indicate systemic

corticosteroids and steroid-paring agents: cyclophosphamide, azathioprine, mycophenolate mofetil, rituximab, intravenous immunoglobulin (IVIG) [57,58]. The latter two were recently reported in anti-laminin-332 MMP [59,60,61], while cyclophosphamide is associated with bladder malignancy [62]. The use of mycophenolate mofetil raises concerns [52] but may also be associated with lower cancer risks [52,63]. Curiously, in inflammatory bowel disease, thiopurines (azathioprine) should be withdrawn in patients with active cancer, and the combination of thiopurines and TNF-alpha inhibitors is linked to a higher lymphoma risk, which should be considered before the therapy selection [64].

The pharmacological agents discussed in this review may also be of interest in the management of MMP and malignancy.

The antitumor effect of leflunomide has been confirmed in myeloma (NCT02509052) and will be examined in other clinical trials (NCT06540937, NCT04997993, NCT03709446, NCT04508790, NCT06229340, NCT06454383); the evaluation of the effect of hydroxychloroquine in melanoma and various cancer types in different regimes is planned for clinical trials (NCT04464759, NCT05647330, NCT0632 8387, NCT07061717, NCT06949982, NCT05518110, NCT06229340, NCT06408298, NCT05733000, NCT0584 3188, NCT04841148).

The expression of CD25 is observed in activated T cells and some malignancies; regulatory T lymphocytes (Tregs) constitute the tumor microenvironment, contributing to drug resistance via the suppression of immune surveillance [65]. Before its global withdrawal, daclizumab was reported in combination with chemotherapy and as an option to overcome the Tregs-mediated drug resistance [66,67]. Developing pharmacological agents which target CD25 may be of use for the management of both malignancy and MMP.

Given the link between the PI3K/mTOR signaling pathway and genetic mutations [51,68], sirolimus is a curious option.

Colchicine exerts anti-mitotic properties [69] and has been associated with a lower risk of colorectal cancer in immune-mediated inflammatory diseases [70].

Dupilumab is generally considered safe, but caution is required in patients with cutaneous T-cell lymphoma [71,72]. However, the initially incorrect diagnosis of atopic dermatitis was suggested as an explanation for the poor prognosis in such cases [52]. Patient monitoring is recommended for cenegermin, given its growth factor properties [73]. The anti-angiogenic agent bevacizumab is used in combination with immune and chemotherapy, but patient selection criteria should be developed due to the risk of hemorrhage and to high mortality rates [74].

The presence of IgE antibodies to BM proteins (e.g., laminin-332 [17]) were earlier reported in MMP, suggesting the potential of omalizumab; additionally, omalizumab can be employed in desensitization to chemotherapeutic agents [75].

The rarity of MMP and its comorbidity with malignancy complicates the collection of further data on the association between MMP and malignant tumors and the impact of immunosuppressive and other pharmacological agents on malignancy risks.

Future Perspectives

Considering that the mechanisms of action of the abovementioned agents are directed against the earlier discussed or hypothesized components of the pathogenesis of MMP and its complications, the studied drugs possess the potential for additional research concerning their efficacy and safety profile.

The analysis of the guidelines shows the susceptibility to therapeutic novelties. Azathioprine, cyclophosphamide, and mitomycin C were included in the First International Consensus on MMP, while mycophenolate mofetil, rituximab, intravenous immunoglobulin, ciclosporin A, and etanercept were introduced by the guidelines of the French Society of Dermatology; a broader term "TNF-inhibitors" was later included in the S3 and S2k guidelines, Janus kinase inhibitors were indicated by the S2k guidelines for severe and recalcitrant cases. Corticosteroids are the most frequently employed immunosuppressive agents owning to their availability and efficiency. However, the unacceptance of long-term use of systemic corticosteroids due to severe AEs (e.g., aseptic hip necrosis, uncontrolled diabetes mellitus, and hypertension) was earlier highlighted in patients diagnosed with OcMMP [76], and there is a wide range of both topical and systemic steroid-sparing immunosuppressants, which are included in the guidelines and which exert effects through various pathways, reduce proliferation of immune cells, and release of pathogenic autoantibodies, with the underlying risk of complications, therapy resistance, and recalcitrant disease course. The search for new options with minimal AEs continues, and time will show which pharmacological agents will be included in new guidelines; some options will remain outside the list of recommended agents (e.g., thalidomide), while others may fade away.

Furthermore, in preclinical studies, additional pharmacological targets have been proposed.

In a murine model of anti-laminin-332 MMP the inhibition of phosphodiesterase-4, responsible for cell signaling, resulted in the improvement of oral lesions [77].

Phosphatidylinositol-3-kinase-δ (PI3Kδ) is involved in cutaneous blistering following leukocyte activation after Fc gamma receptor-dependent binding to the skin-located immune complexes and subsequent production of proteases and ROS. Parsaclisib, a PI3Kδ-selective inhibitor, showed the amelioration of cutaneous lesions in the MMP-induced mouse model [78]. Additionally, laminin-332 is linked to

malignancy development through the activation of PI3K and the Rho family of small GTPases [45,46], with decreased signaling of the latter after the administration of IVIG demonstrated by Murthy et al. (2024) [20]. Thus, the role of these pathways should be further clarified in MMP.

MMP-associated fibrosis may be linked to the aldehyde dehydrogenase/retinoic acid (ALDH/RA)-mediated dendritic cell paracrine effect and activation of a pro-fibrotic phenotype of fibroblasts. Patzelt et al. (2021) concluded that the ALDH inhibitor disulfiram does not reduce fibrosis in ocular and oral lesions in MMP-induced mice but improved cutaneous lesions [79]. However, earlier disulfiram effectively alleviated fibrotic changes in human OcMMP fibroblasts and in a mouse model [80].

In patients with MMP, high serum levels of IL-1 [81] and IL-5 [82] and an elevated expression of IL-6, IL-12, IL-17 [83], and IL-5 mRNA [84] in biopsy samples were reported. IL-1, IL-5, IL-6, IL-12, and IL-17 inhibitors are employed in dermatology; IL-5, IL-12/23, and IL-17 inhibitors were used in bullous pemphigoid [85,86]. Nonetheless, these agents occasionally paradoxically induce BP [87,88]. Moreover, complement inhibitors have been suggested for BP [85,86]; no report on the use of these agents in MMP was found.

Summarizing the above-mentioned data, a broader spectrum of pharmacological agents will allow a personalized approach and more alternatives, in particular for recalcitrant cases and failure of previous therapy. Moreover, given the participation of mechanisms other than the formation of autoantibodies in the pathogenesis of MMP [20], it is advisable to collect more data on the impact of cytokines, complement, proteases, immune cells, and other agents in search of solutions to avoid severe complications and overcome therapy resistance.

Conclusion

We present the analysis of pharmacological agents used in the management of MMP but that are not included in earlier published guidelines. Considering the severity of the condition, progressive fibrosis, and resistance to therapy, more research is required in relation to the pathogenesis of MMP and the efficacy and safety profile of novel pharmacological options. Pharmacological agents should provide the achievement and maintenance of remission with minimal adverse effects. A broader spectrum of pharmacological agents will allow a personalized approach and more alternatives, in particular for recalcitrant cases, failure of the previous therapy, and patients with MMP and malignancy.

Abbreviations: Recombinant human nerve growth factor (rhNGF); Nicotinic acetylcholine receptor (nAChR); Lymphocyte function-associated antigen-1 (LFA-1); Intercellular adhesion molecule-1 (ICAM-1); Interleukin-2 receptor (IL-2R); IL-4 receptor alpha chain (IL-4Rα); Adrenocorticotropic hormone (ACTH); Tropomyosin receptor kinase A (TrkA); Nerve growth factor receptor (p75NTR); Mucous membrane pemphigoid (MMP); Ocular mucous membrane pemphigoid (OcMMP); Oral mucous membrane pemphigoid (OrMMP); Brunsting-Perry pemphigoid (BPP).

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