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REVIEW

Autoimmune pemphigus: opportunistic infectious complications and comprehensive patient care

Pénfigo autoinmune: complicaciones infecciosas oportunistas y cuidados integrales del paciente

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ABSTRACT

Pemphigus is a rare autoimmune blistering disease characterized by loss of keratinocyte adhesion and the development of blisters and erosions on the skin and mucous membranes. The aim of this narrative review is to describe the clinical and pathophysiological features of pemphigus, analyze opportunistic infectious complications related to immunosuppressive therapy, and summarize recommendations for comprehensive patient care. A structured literature search was performed in SciELO, RedALyC, and LILACS, including original articles, case reports, clinical series, guidelines, and reviews published between 2013 and 2024 in Spanish and Portuguese. The reviewed evidence shows that pemphigus vulgaris and paraneoplastic pemphigus are the variants with the highest mucosal involvement and greater disruption of the cutaneous-mucosal barrier, predisposing patients to bacterial, viral, and fungal superinfections, particularly under long-term immunosuppression. Systemic corticosteroids remain the mainstay of treatment, often combined with steroid-sparing agents and targeted immunotherapy, such as rituximab, according to disease severity. Nursing care plays a key role in skin and mucosal protection, prevention of secondary infections, nutritional support, pain control, and psychosocial assistance. Close coordination of the multidisciplinary team, together with patient and family education, is essential to reduce morbidity and mortality, enhance treatment adherence, and improve quality of life in this chronic and potentially life-threatening disease.

Keywords: Pemphigus Vulgaris; Autoimmune Blistering Diseases; Opportunistic Infections; Immunosuppression; Comprehensive Patient Care.

RESUMEN

El pénfigo es una enfermedad ampollosa autoinmune poco frecuente, caracterizada por la pérdida de adhesión entre queratinocitos y la formación de ampollas y erosiones en piel y mucosas. El objetivo de esta revisión narrativa es describir las características clínicas y fisiopatológicas del pénfigo, analizar las complicaciones infecciosas oportunistas asociadas a la inmunosupresión y sintetizar las recomendaciones para un cuidado integral del paciente. Se realizó una búsqueda bibliográfica estructurada en SciELO, RedALyC y LILACS, incluyendo artículos originales, reportes de caso, series clínicas, guías de manejo y revisiones publicados entre 2013 y 2024 en español y portugués. Los estudios revisados muestran que el pénfigo vulgar y el pénfigo paraneoplásico son las formas con mayor compromiso mucoso y riesgo de deterioro de la barrera cutaneomucosa, lo que favorece la sobreinfección bacteriana, viral y fúngica, especialmente en pacientes bajo tratamiento inmunosupresor prolongado. Los corticoides sistémicos continúan siendo la base terapéutica, complementados con fármacos ahorradores e inmunoterapia dirigida, como rituximab, según la gravedad clínica. El cuidado de enfermería desempeña un papel central en la protección de la piel, la prevención de infecciones, el soporte nutricional, el control del dolor y el acompañamiento psicoemocional. La coordinación del equipo multidisciplinario y la educación del paciente y su familia son esenciales para

reducir la morbimortalidad, mejorar la adherencia terapéutica y optimizar la calidad de vida en esta enfermedad crónica y potencialmente grave.

Palabras clave: Pénfigo Vulgar; Enfermedades Ampollosas Autoinmunes; Infecciones Oportunistas; Inmunosupresión; Cuidado Integral del Paciente.

INTRODUCTION

Pemphigus is a rare autoimmune blistering disease characterized by flaccid skin blisters and mucosal ulcerations, a direct consequence of the loss of adhesion between keratinocytes.[1] This loss of cohesion is due to an immune response directed against desmogleins, calcium-dependent adhesion proteins that form part of desmosomes, structures responsible for maintaining intercellular junctions in the epithelium.[1]

The aim of this article is to analyze the available scientific evidence on opportunistic infectious complications in patients with pemphigus and to describe the pathophysiological, clinical, and therapeutic mechanisms associated with their development.

METHOD

This work corresponds to a narrative review of the literature. To prepare it, a structured bibliographic search was conducted across electronic databases, prioritizing the retrieval of Spanish-language articles available in SciELO, RedALyC, and LILACS. The search was conducted between January and October 2025, using controlled terms and keywords related to pemphigus, immunosuppression, infectious complications, bullous lesions, and immunological treatment. Original articles, case reports, clinical series, management guidelines, and reviews published between 2013 and 2024 that addressed clinical, immunopathological, therapeutic, or infectious complications associated with pemphigus were included. Evidence from Latin American countries was prioritized due to its epidemiological relevance to the regional context.

Duplicate articles, publications without full-text access, reports not related to pemphigus, and studies that exclusively addressed other bullous dermatoses not directly associated with pemphigus were excluded. The information obtained was organized and analyzed comparatively, grouping the findings into thematic categories: clinical classification, pathophysiology, diagnosis, treatment, opportunistic infections, and patient care. Finally, the results were integrated and synthesized to identify common patterns, current therapeutic trends, and gaps in the evidence relevant to clinical practice.

RESULTS AND DISCUSSION

Clinically, the disease presents with recurrent vesicles, painful erosions, or erythematous plaques, most frequently observed in people between the third and fourth decades of life.[2] The estimated incidence ranges from 10 to 22 cases per million inhabitants, while the prevalence is approximately 0,1 to 0,7 per 100 000 inhabitants.[3,4] In the pathophysiology of pemphigus, the desmosome is the central altered structure: it is formed by three main groups of proteins—cadherins, keratins, and desmogleins—that help maintain epithelial mechanical stability.[3] Within the cadherin group, there are two relevant subfamilies, desmocollins and desmogleins, whose interactions mediate intercellular adhesion and explain the clinical variability based on the pattern of autoantibodies present.[4]

Regarding desmoglein distribution, there are three main isoforms, each with distinct expression patterns depending on the type of epithelial tissue. On the one hand, they predominate in the epidermis, while, in contrast, their expression is significantly lower in the mucous membranes. In addition, the desmoglein-3 isoform shows a higher concentration in the basal layers of the epidermis and in deep mucosal epithelia.[5]

Consequently, the typical clinical lesions of pemphigus are intraepidermal blisters resulting from acantholysis. In this context, the diagnosis must be comprehensive, as it is based not only on clinical inspection but also on anatomopathological and immunological studies. Specifically, biopsy of the affected skin and immunofluorescence (ELISA) allow the demonstration of intercellular deposits of IgG—predominantly subclasses 1 and 4—characteristic of this disease.[6]

Likewise, histological findings with hematoxylin-eosin staining usually reveal acantholytic keratinocytes, non-degenerated neutrophils, and a variable number of eosinophils, both inside the pustules and in the superficial dermis.[7]

The choice of research is justified because pemphigus remains a rare disease with high morbidity and mortality due to late diagnosis and complications associated with immunosuppressive treatment. In addition, opportunistic infections are a frequent cause of skin superinfection and systemic complications, affecting the clinical course and quality of life of patients. Therefore, further research is needed to improve early recognition and optimize therapeutic approaches. Finally, Latin American evidence on pemphigus remains limited, which

reinforces the importance of contextualized regional scientific production.

Table 1. Classification of pemphigus, symptoms, causes, and treatment.

Type of pemphigus	Symptoms/Manifestations	Causes/Mechanism	Treatment
Pemphigus vulgar	Flaccid blisters on the oral mucosa, painful erosions, and progressive skin involvement.	IgG against desmoglein 3 ± desmoglein 1.	Systemic corticosteroids + immunosuppressants (mycophenolate/ azathioprine). Rituximab as first-line treatment in moderate to severe cases.
Pemphigus foliaceus	Superficial blisters and crusts. Does not affect mucosa.	IgG against desmoglein 1.	Topical or systemic corticosteroids depending on severity + immunomodulators.
Paraneoplastic pemphigus	Severe mucositis, conjunctivitis, polymorphic skin lesions.	Associated with neoplasia (lymphoma, leukemia, thymoma).	Treatment of neoplasia + immunosuppression; rituximab possible.
Drug-induced pemphigus	Lesions similar to PV or PF after starting medication.	Drug-induced antibodies (captopril, penicillins, rifampicin).	Discontinue the drug; systemic corticosteroids if required.
IgA pemphigus	Pustules and erosions.	Epidermal IgA deposition (anti-desmocollins).	Dapsone ± corticosteroids.

In clinical terms, pemphigus is divided into five main variants: pemphigus vulgaris, pemphigus foliaceus, paraneoplastic pemphigus, drug-induced pemphigus, and IgA pemphigus. However, among these clinical forms, only pemphigus vulgaris (PV) and paraneoplastic pemphigus most frequently involve the mucosa, especially in the oral cavity.[8] On the other hand, the pemphigus vulgaris can involve various epithelial surfaces, such as the genital mucosa, conjunctiva, nasal mucosa, and oral mucosa, the latter being the most common location. In this context, the lesions initially manifest as flaccid blisters that, when ruptured, cause irregular, intensely painful superficial erosions with poorly defined edges and are frequently covered by hemorrhagic scabs. Finally, it is essential to note that, in many cases, oral lesions develop before or during the progressive spread of skin lesions in patients with pemphigus vulgaris.[9]

As for treatment, the first line of therapy continues to be the use of systemic steroids. According to the European guidelines of the Academy of Dermatology, it is recommended to start prednisone in a range of 0,5 to 1,5 mg/kg/day. If clinical activity cannot be controlled in the first two weeks, the dose can be increased to 2 mg/kg/day.[10] Likewise, to reduce the adverse effects associated with prolonged exposure to high doses of corticosteroids, the use of non-steroidal immunomodulators such as azathioprine, mycophenolate mofetil, cyclophosphamide, methotrexate, dapsone, tetracycline, sulfasalazine, chlorambucil, and cyclosporine has been indicated.[10] Furthermore, systematic clinical follow-up is essential to assess therapeutic response and patient evolution.[11]

In this regard, PCR quantification has been used as a complementary tool to estimate immunological activity, as it allows the identification of desmoglein levels in fibroblasts, endothelial cells, and epithelial cells.[12] However, when diagnosis is delayed or the clinical picture cannot be confirmed early on, the determination of desmoglein levels loses its practical usefulness.[13] In addition to the immunological component, relevant biopsychosocial factors must be considered, including emotional impact, chronic pain, and systemic symptoms, which negatively affect the patient's quality of life and their work, social, and family performance.[14]

Similarly, because the oral mucosa is involved, food intake can become painful and limited, predisposing patients to weight loss and malnutrition.[15] Finally, adjuvant therapies have attracted interest to clarify whether sustained B-cell depletion is a key mechanism for maintaining long-term clinical remission, given that, in most cases, corticosteroids remain the predominant therapeutic tool.[16] However, prolonged administration of these drugs can also generate significant adverse effects, which translates into one of the main challenges in the medical management of pemphigus.[17]

Individualized clinical follow-up is an essential part of the therapeutic approach to pemphigus; therefore, the treating physician must document each patient's natural progression and implement periodic monitoring to ensure the efficacy of the initiated treatment.[18] In this regard, patients with localized pemphigus vulgaris tend to have a more favorable prognosis and, in many cases, show a rapid response to immunosuppressive therapy.[19] However, despite this initial response, it has been reported that between 40 % and 80 % of patients experience recurrences within the first 6 to 24 months, which is why strict clinical monitoring is recommended, ideally every 6 months, to maintain clinical remission and avoid flare-ups.[20] Even so, morbidity and mortality rates

remain high even when diagnosis is early and treatment is applied correctly.[21]

Furthermore, recent studies have shown that, as with other autoimmune dermatoses, pemphigus can coexist with or be associated with systemic diseases, underscoring the importance of a comprehensive medical approach.[22] Thus, its onset may be determined by a combination of environmental factors, genetic predisposition, immunological alterations, and, in some cases, stress as a physiological or emotional trigger.[23] However, according to published case series, even appropriately used treatments have not entirely prevented disease recurrence, reinforcing the chronic and unpredictable nature of the clinical picture.[24] Finally, experimental research in mouse models has shown that desmogleins 1, 3, and 4 play a fundamental role in hair follicle adhesion, which could explain the onset of alopecia in certain patients with pemphigus.[25]

The average age of onset of pemphigus is usually in the fourth decade of life. When the condition presents with extensive skin involvement, it may be associated with a poorer clinical prognosis.[26] After starting immunosuppressive treatment, many patients achieve remission within approximately 12,5 weeks, with clinical improvement observed in some cases as early as 6 weeks.[27] Therapies targeting CD20 cells have also been incorporated to induce B-cell depletion and reduce the production of pathogenic autoantibodies.[28] Blistering lesions may be localized or generalized, affecting areas such as the face, abdomen, or extremities.[29]

Furthermore, blisters can exude large volumes of fluid, which can promote electrolyte loss and be potentially fatal in severe cases.[30] Similarly, T lymphocytes actively participate in the immunopathogenesis of the disease through the secretion of proinflammatory cytokines, which has prompted the development of new therapeutic strategies aimed at modulating this response.[31] From an epidemiological point of view, some studies suggest a higher frequency in women than in men, with an average age of onset of around 47 years in women and 45 years in men, respectively,[32] while the choice of treatment will depend mainly on the clinical severity of the patient's condition.[33]

In addition, patient self-care and proper management of lesions are an essential component of a comprehensive therapeutic approach,[34] and it is necessary to identify sociodemographic variables that may influence the clinical course.[35] As with other autoimmune disorders, pemphigus can be associated with depressive symptoms and emotional stress,[36] yet there are no globally established public policy strategies specifically targeting this population.[37] In this scenario, the primary care physician's role is essential to ensure longitudinal follow-up, therapeutic control, and early detection of complications.[37] Finally, it is essential to note that both correct clinical identification and histological findings allow proper classification of pemphigus variants,[38] and that even in previously asymptomatic or remitting patients, episodes of stress can trigger disease reactivation.[39] Characteristic microscopic changes also serve as differential markers to distinguish between different subtypes.[40]

Table 2. Patient care and nursing management in pemphigus

Nursing action	Objective	Suggested interventions
Protection of skin and mucous membranes	Prevent mechanical rupture and new lesions	Gentle hygiene, non-adherent dressings, avoid friction and moisture
Prevention of secondary infections	Reduce risk of opportunistic infections	Monitoring for signs of infection, early notification to the doctor
Nutritional support	Prevent weight loss and malnutrition	Adapt food consistency, adequate hydration
Pain management	Improve tolerance to food intake and rest	Scheduled analgesia and education about painful stimuli
Psycho-emotional support	Reduce associated anxiety and stress	Brief counseling, active listening, support

Opportunistic skin infections are among the main complications associated with pemphigus, particularly in patients who require long-term immunosuppressive regimens to control disease activity.[41] In this context, the deterioration of the skin barrier—secondary to acantholysis and extensive erosions—creates an environment conducive to the penetration of pathogenic microorganisms.[42] At the same time, pharmacological immunosuppression significantly reduces the host's humoral and cellular defense capacity.[43] As a result, secondary bacterial infections caused by Staphylococcus aureus and Streptococcus pyogenes are relatively common. They can progress from impetiginized lesions to cellulitis or even cutaneous sepsis if not treated appropriately.[44]

Likewise, viral infections, especially the reactivation of herpes simplex and varicella-zoster,[45] have been described as an added risk in these patients and may present with more extensive, aggressive clinical forms and a worse prognosis compared to immunocompetent individuals.[46] For all of the above reasons, the clinical

management of pemphigus must include rigorous monitoring of skin condition, early identification of signs of infection, and the application of personalized preventive measures, which may consist of antiviral prophylaxis in cases with a history of recurrence and the use of systemic antibiotics when there is clinical evidence of active bacterial infection.

CONCLUSION

Medical information on pemphigus, an autoimmune disease, was collected, described, and analyzed.

The care of patients with pemphigus requires the active participation of nursing staff due to the fragility of the skin, loss of barrier function, and the high risk of opportunistic infections.[47]

The role of nursing focuses on maintaining skin integrity through gentle hygiene, the use of sterile, non-adherent dressings, monitoring areas of friction, and early identification of inflammatory signs, abnormal exudation, or changes in local coloration that suggest superinfection.[48]

Nurses must also ensure adequate hydration and nutritional support, adapting the consistency and temperature of food when there is mucosal involvement, to reduce pain during intake and prevent weight loss.[49]

Another priority is educating patients and their families, reinforcing self-care strategies aimed at avoiding mechanical trauma to the skin and recognizing triggers such as stress, intercurrent infections, or excessive heat exposure.[50]

Finally, the emotional component must also be addressed as a whole, since physical disability, chronic pain, and visible changes to the body can lead to anxiety, depression, social isolation, and poor therapeutic adherence.[51]

Nursing care, therefore, contributes directly to improving the patient's quality of life and maintaining their clinical stability.[52]

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