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Review Article

Psoriasis treatment: a literature review

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ABSTRACT

Psoriasis is a chronic skin disease that affects millions of people worldwide. Its etiology is not yet fully understood, but it is thought to be related to a combination of genetic, immunological and environmental factors. In this literature review article, several relevant scientific studies and publications will be analyzed to provide an overview of psoriasis treatment. Aspects such as epidemiology, the significance of the disease, the theoretical framework that includes definition, diagnosis, treatment and complications will be addressed, and conclusions based on the available evidence will be presented.

Keywords: Psoriasis, treatment, chronic skin disease

INTRODUCTION

Psoriasis is a chronic, recurring skin disease that affects millions of people worldwide. It is characterized by the presence of inflammatory lesions on the skin, in the form of scaly, reddened and itchy plaques. This disease can have a significant impact on the quality of life of patients, both physically and psychologically.[1,2]

The exact etiology of psoriasis has yet to be fully elucidated, but it is thought to be the result of a complex interaction between genetic, immunological and environmental factors. A genetic predisposition has been observed, as those with a family history of psoriasis are at an increased risk of developing the disease. In addition, an immunological component has been identified in its pathogenesis, in which the immune system plays a fundamental role in the activation and maintenance of the skin inflammation characteristic of the disease.[3,4]

Psoriasis can manifest in various clinical forms, the most common being plaque psoriasis, which presents as well-defined lesions with silvery scales and underlying erythema. These lesions are usually located in areas such as the elbows, knees, scalp and lumbar region. However, psoriasis can affect any part of the body, including the nails, palms, soles of the feet, and genitals.[5]

The epidemiology of psoriasis shows variable prevalence worldwide, with estimates ranging from 2% to 3% of the world's population. In addition, a geographical variation has been observed, with a greater incidence in colder climates. The disease can manifest itself at any age, although it usually appears in the second and third decade of life, with a peak incidence in the fourth and fifth decade.[6]

The significance of psoriasis goes beyond its cutaneous impact, since an association with various systemic comorbidities has been demonstrated. Psoriatic arthritis, a form of chronic inflammatory arthritis, affects approximately 30% of psoriasis patients and can lead to joint pain and functional disability. In addition, psoriasis has been linked to an increased risk of developing cardiovascular diseases, such as coronary artery disease, hypertension, and diabetes. This disease can also have a

significant impact on patients' mental health, with an increased risk of depression and anxiety.[7,8]

TREATMENT OF PSORIASIS

Physiopathology

The pathophysiology of psoriasis involves a complex interaction between genetic, immunological and environmental components. It is considered an autoimmune disease in which the individual's immune system triggers a chronic, exaggerated inflammatory response in the skin.[3,4]

At the genetic level, it has been shown that there is a family predisposition to develop psoriasis. Several genes associated with the disease have been identified, such as the PSORS1 gene, which is present in a high percentage of familial cases of psoriasis. These genes influence the functioning of the immune system and the inflammatory response.[9]

The inflammatory process in psoriasis begins with the activation of immune system cells, such as T lymphocytes. Under normal conditions, T lymphocytes help defend the body against infection and disease. However, in psoriasis, these T lymphocytes are abnormally activated and release inflammatory mediators, such as cytokines and chemokines.[10]

The main cytokine involved in the pathogenesis of psoriasis is tumor necrosis factor-alpha (TNF-α). This cytokine plays a key role in the inflammation and excessive cell proliferation seen in psoriasis. In addition, an increase in the production of other cytokines, such as interleukin-17 (IL-17), interleukin-23 (IL-23) and interleukin-22 (IL-22), which are also involved in the inflammatory response and cell hyperproliferation, has been observed.[10]

The imbalance in the regulation of these cytokines and the abnormal immune response lead to the activation of skin cells, such as keratinocytes. Keratinocytes are the main cells of the epidermis and are involved in the formation of the skin barrier. In psoriasis, keratinocytes proliferate excessively and accumulate in the outermost layer of the skin, forming the scaly plaques characteristic of the disease. This hyperproliferation of keratinocytes is mediated by the interaction between inflammatory cytokines and receptors present on the surface of these cells.[10]

Treatment

The main objective of psoriasis treatment is to relieve symptoms, control inflammation and improve the quality of life of patients. Treatment options vary depending on the severity of the disease, the extent of the lesions, and the individual response of the patient.[11]

Topical treatments

Topical treatments are applied directly to skin lesions and are widely used in mild to moderate cases of psoriasis. Topical corticosteroids are one of the most common and effective treatments, as they reduce inflammation and cell proliferation. Other topical treatments include vitamin D analogues, such as calcipotriol, which help normalize the growth of skin cells, and vitamin A analogues (topical retinoids), which reduce inflammation and scaling.[12]

Phototherapeutic therapy

Phototherapeutic therapy, also known as phototherapy, involves controlled exposure to ultraviolet (UV) light to treat psoriasis. Narrowband UVB radiation and UVA radiation combined with photosensitizers (PUVA therapy) are commonly used. These therapies reduce inflammation and slow cell proliferation in the affected skin.[13]

Systemic therapy

When psoriasis is more severe or does not respond adequately to topical treatments, systemic therapies may be employed. These medications are administered orally or injectably and act systemically to suppress the immune response and reduce inflammation. Some systemic therapy options include:[14]

Methotrexate: It is an immunosuppressive drug that reduces inflammation and cell proliferation.

Acitretin: It is an oral retinoid that normalizes the growth of skin cells and reduces flaking.

Cyclosporine: It is an immunosuppressant that inhibits the immune response and inflammation.

Phosphodiesterase-4 inhibitors: These drugs reduce inflammation and cell proliferation by inhibiting a specific enzyme.

Biological therapy

In recent years, highly effective biological therapies have been developed for the treatment of moderate to severe psoriasis. These biologic drugs target specific molecules and cells involved in inflammation and cell proliferation in psoriasis. Tumor necrosis factor-alpha (TNF-α) inhibitors and interleukin inhibitors (IL) are examples of biologic therapies used in psoriasis. These medications have been shown to be highly effective in controlling symptoms and improving patients' quality of life.[15]

DISCUSSION

Psoriasis is a chronic skin disease that affects millions of people worldwide. Despite advances in our understanding of its pathophysiology and the development of various

treatment options, it remains a clinical challenge due to its chronic and recurrent nature.[1-4]

The pathophysiology of psoriasis involves a complex interaction between genetic, immunological and environmental factors. Genetic predisposition plays an important role, as it has been shown that there are several genes associated with the disease. The PSORS1 gene, for example, has been identified in a high percentage of familial cases of psoriasis. These genes influence the immune response and regulation of inflammation in the skin.[3,4]

At the immune level, psoriasis is characterized by an abnormal immune response, in which T lymphocytes and dendritic cells play a crucial role. Exaggerated activation of T lymphocytes leads to the release of inflammatory cytokines, such as TNF-α, IL-17, IL-23 and IL-22. These cytokines trigger a cascade of inflammatory events and promote the hyperproliferation of keratinocytes in the skin. Keratinocytes, in turn, produce more inflammatory cytokines, creating a self-amplifying cycle of inflammation and cell proliferation.[10]

Knowledge of the pathophysiology of psoriasis has led to the development of various therapeutic approaches. The treatment of psoriasis is based on the principle of controlling inflammation and reducing excessive cell proliferation in the skin. Topical treatments, such as corticosteroids, vitamin D analogues, and topical retinoids, are effective in mild to moderate cases by reducing local inflammation and normalizing skin cell growth.[11,12]

However, in more severe cases or when topical treatments are not sufficient, systemic therapy is required. These treatments act at the systemic level to suppress the immune response and reduce widespread inflammation in the body. Methotrexate, acitretin, cyclosporine, and phosphodiesterase-4 inhibitors are examples of systemic therapies used in psoriasis. These drugs have different mechanisms of action, but all aim to reduce inflammation and normalize cell proliferation in the affected skin.[13,14]

In recent years, biological therapies have revolutionized the treatment of psoriasis. These biologic drugs, such as TNF-α inhibitors and IL inhibitors, target specific molecules and cells involved in inflammation and cell proliferation in psoriasis. These biological therapies have been shown to be highly effective in controlling symptoms and improving patients' quality of life. However, their cost and limited availability can be obstacles for many patients.[15]

CONCLUSION

In conclusion, psoriasis is a complex and chronic disease that involves a complex interaction between genetic, immunological and environmental factors. Although significant progress has been made in understanding its pathophysiology and developing treatment options, it remains a clinical challenge. Topical treatments, phototherapeutic therapy, systemic therapy, and biological therapy are used at different levels of disease severity. However, more research is needed to improve our understanding of the disease and develop more effective and accessible treatment options for psoriasis patients.

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REFERENCES

1. Richard MA, Paul C, Nijsten T, Gisondi P, Salavastru C, Taieb C, et al. Prevalence of most common skin diseases in Europe: a population-based study. J Euro Acad Dermatol Venereol. 2022;36(7):1088-96.
2. Strober B, Thaçi D, Sofen H, Kircik L, Gordon KB, Foley P, et al. Treatment of plaque psoriasis with deucravacitinib (POETYK PSO-2 study): a plain language summary. Immunotherapy. 2023.
3. Otsuka A, Nomura T, Rerknimitr P, Seidel JA, Honda T, Kabashima K. The interplay between genetic and environmental factors in the pathogenesis of atopic dermatitis. Immunological Rev. 2017;278(1):246-62.
4. Campalani E, Barker JNWN. The clinical genetics of psoriasis. Current Genomics, 2005;6(1):51-60.
5. Johnson MAN, Armstrong AW. Clinical and histologic diagnostic guidelines for psoriasis: a critical review. Clinical Rev Allergy Immunol. 2013;44:166-72.
6. Pezzolo E, Cazzaniga S, Colombo P, Chatenoud L, Naldi L. Psoriasis incidence and lifetime prevalence: suggestion for a higher mortality rate in older age-classes among psoriatic patients compared to the general population in Italy. Acta dermato-venereologica. 2019;99(4):400-3.
7. Kaushik SB, Lebwohl MG. Psoriasis: which therapy for which patient: psoriasis comorbidities and preferred systemic agents. J Am Acad Dermatol. 2019; 80(1):27-40.
8. Kimball AB, Wu EQ, Guérin A, Andrew PY, Tsaneva M, Gupta SR, et al. Risks of developing psychiatric disorders in pediatric patients with psoriasis. J Am Acad Dermatol. 2012;67(4):651-7.
9. Reich K, Hüffmeier U, König IR, Lascorz J, Lohmann J, Wendler J, et al. TNF polymorphisms in psoriasis: association of psoriatic arthritis with the promoter polymorphism TNF*-857 independent of the PSORS1 risk allele. Arthritis Rheumatism, 2017;56(6):2056-64.
10. Lin ZM, Ma M, Li H, Qi Q, Liu YT, Yan YX, et al. Topical administration of reversible SAHH inhibitor ameliorates imiquimod-induced psoriasis-like skin lesions in mice via suppression of TNF-α/IFN-γ-induced inflammatory response in keratinocytes and

T cell-derived IL-17. Pharmacological Res. 2018;129, 443-52.
11. Mrowietz U. Implementing treatment goals for successful long-term management of psoriasis. J Euro Acad Dermatol Venereol. 2012;26:12-20.
12. Stein L. Clinical studies of a new vehicle formulation for topical corticosteroids in the treatment of psoriasis. J Am Acad Dermatol. 2005;53(1):S39-S49.
13. Hönigsmann H. Phototherapy for psoriasis. Clin Exp Dermatol. 2001;26(4):343-50.
14. Mrowietz U, Kragballe K, Reich K, Spuls P, Griffiths CEM, Nast A, et al. Definition of treatment goals for moderate to severe psoriasis: a European consensus. Arch Dermatol Res. 2011;303:1-10.
15. Campa M, Mansouri B, Warren R, Menter A. A review of biologic therapies targeting IL-23 and IL-17 for use in moderate-to-severe plaque psoriasis. Dermatol Therapy. 2016;6:1-12.

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