ZITA MEDICAL MANAGEMENT

www.hsmc.gr/en/pharmakeftiki-journal/

PHARMAKEFTIKI, 37, 2S, 2025 | 442-445 REVIEW

https://doi.org/10.60988/p.v37i2S.251

Psoriasis: a short review of its pathophysiology and causes

Ghufran Abd Omran Abdulridha[1,*], Sarah Kassab Shandaway Al-Zamali[2], Mohammed Sabah Otaiwi[3], Hiyam Adnan Ali[2], Marwa Saad Mohammed[4]

[1]Department of Clinical Chemistry, Hammurabi College of Medicine, University of Babylon, Hillah, Iraq
[2]Department of Medical Microbiology, Hammurabi College of Medicine, University of Babylon, Hillah, Iraq
[3]College of Basic Education, University of Babylon, Hillah, Iraq
[4]Department of Chemistry, College of Science, Mustansiriyah University, Baghdad, Iraq

KEY WORDS:
psoriasis; plaque psoriasis; types of psoriasis; topical treatments; systemic treatment

ARTICLE INFO:
Received: January 11, 2025
Revised: February 18, 2025
Accepted: February 20, 2025
Available online: October 10, 2025

*** CORRESPONDING AUTHOR:**
Ghufran Abd Omran Abdulridha, Department of Clinical Chemistry, Hammurabi College of Medicine, University of Babylon, Hillah, Iraq; e-mail: ghufranabdamrin1990@gmail.com; ham297.qufran.abad@uobabylon.edu.iq

ABSTRACT

Psoriasis is a chronic autoimmune inflammatory condition characterized by abnormal keratinocyte metabolism. Its development is closely associated with immune system dysfunction and aberrant activation. This common skin disorder is marked by the formation of clearly defined, scaly, erythematous plaques, typically affecting the knees, elbows, scalp, and trunk. While there is no cure for psoriasis, it is believed to be an immune-mediated disease in which skin cells proliferate more rapidly than normal. The pathogenesis of psoriasis involves multiple factors, including genetic predisposition, lipoprotein-2, galectin-3, fractalkine, vaspin, human neutrophilic peptides, T-cells, dendritic cells, and antimicrobial peptides. Four forms of psoriasis exist, each with distinct clinical manifestations and pathological features: erythrodermic, pustular, chronic plaque, and articular psoriasis. Plaque psoriasis is the most common type, characterized by dry, itchy scales covering raised areas of skin. Although currently incurable, psoriasis treatment should focus on minimizing patients' physical and psychological distress through early intervention, identification and prevention of associated multimorbidity, lifestyle modifications, and a personalized treatment approach. This short review summarizes recent progress in understanding psoriasis pathophysiology and its causes, aiming to improve our comprehension of the disease.

1. Introduction

Psoriasis is a prevalent, chronic, and multifaceted inflammatory skin condition that is associated with various comorbidities. It is charac-

PHARMAKEFTIKI, 37, 2S, 2025 | 442-445

terized by immune cell infiltration into the dermis and epidermal hyperplasia[1]. Psoriasis is a systemic disorder that affects not just the skin but also the liver, joints, and kidneys. It has been demonstrated that psoriasis can raise the incidence of various inflammatory conditions. Treatment for psoriasis may lessen the occurrence of these comorbidities, as the psoriasis-associated high levels of pro-inflammatory cytokines can be carried by the blood to other systems, thereby resulting in inflammation and subclinical inflammation[2]. Moreover, psoriasis lesions are known to be triggered or made worse by a variety of conditions.

An Iraqi study, conducted at a private dermatology clinic in Samawa City and the Al Hussein Teaching Hospital, has found that the most common type of psoriasis among children aged 6 months to 14 years is the plaque psoriasis, followed by guttate and scalp psoriasis[3]. The study has also indicated a higher prevalence of psoriasis in females than in males[3]. Childhood psoriasis differs from adult psoriasis, necessitating specific consideration in order to address potential long-term psychological issues related to the disease. This short review provides an overview of recent progress in our understanding of the psoriasis pathophysiology and its causes, aiming to enhance our comprehension of the disease processes involved.

2. Epidemiology of psoriasis

Patients with psoriasis have been found to have greater rates of mental health issues, such as anxiety, depression, and suicidal thoughts. Psoriasis can affect individuals of any age. However, its onset appears to follow a bimodal distribution, with peaks between the ages of 20 and 30 and again between 50 and 60. A retrospective study conducted in the outpatient clinic of the Baquba Teaching Hospital in Diyala has examined the prevalence of psoriasis in Iraq[4]. Of the 10,964 outpatients with skin conditions, 220 (2%) were diagnosed with psoriasis, of which 102 (46%) were female and 118 (54%) were male[4]. An earlier study has also indicated that the IL20RA gene contributes to genetic susceptibility to psoriasis within the Iraqi population[5].

3. Causes of psoriasis

Several risk factors contribute to the development of psoriasis, including genetic predisposition, age, sex, stress, obesity, and bacterial infections.

4. Pathophysiology of psoriasis

Psoriasis is a complex, chronic inflammatory skin condition primarily driven by immune system dysfunction. It is characterized by abnormal differentiation, hyperproliferation of keratinocytes, and angiogenesis. The development of psoriasis involves multiple interacting factors, including immune system dysfunction, genetic predisposition, and environmental triggers. Increased DNA synthesis and a markedly lower rate of epidermal turnover are indicators of hyperproliferation. Reduced expression of some keratins (1 and 10) and increased expression of others (6 and 16), which are typically expressed in differentiating skin, are characteristics of abnormal keratinocyte differentiation[1,6].

4.1. Immune system participation

Psoriasis is mostly a T lymphocyte-mediated condition, in which skin inflammation and keratinocyte hyperproliferation are caused by the activation of pathogenic T lymphocytes and innate immune cells. Although B lymphocytes have long been underappreciated in the pathogenesis of psoriasis, new research has shown their significance in inflammatory skin disorders[6].

4.2. Genetic factors

Psoriasis more commonly follows a polygenic inheritance pattern. Research on affected families has identified chromosomal regions, known as “PSORS” (psoriasis susceptibility loci), that are associated with the disease’s development[7]. While twelve such regions have been identified, the *PSORS1* region is the most significant, accounting for 35%–50% of

PHARMAKEFTIKI, 37, 2S, 2025 | 442-445

hereditary psoriasis cases. This region also contains *HLA-Cw6* (human leukocyte antigen C); the first gene linked to psoriasis, which has been detected in 10.5% to 77.2% of patients[8]. Monozygotic twins are two to three times more likely to have psoriasis than dizygotic twins, and first- and second-degree relatives of those who have the condition are more likely to get it themselves. *TNFAIP3*, *IL23R*, and *HLA-Cw6* are among the many genes that raise an individual's risk of psoriasis[8].

5. Symptoms, clinical presentation, and diagnosis

Common symptoms of psoriasis include skin scaling, erythema (redness), itching, fatigue, burning, bleeding, and swelling. Psoriasis presents in five clinical types: (i) plaque psoriasis (also known as "psoriasis vulgaris", is the most prevalent kind, making up between 80% and 90% of all cases), (ii) flexural psoriasis (also known as "inverse psoriasis", is characterized by intertriginous areas that are affected by mildly erosive erythematous plaques and patches), (iii) pustular psoriasis (the occurrence of many sterile pustules that coalesce is a characteristic of this type of psoriasis; it may be widespread or isolated; palmoplantar pustular psoriasis and acrodermatitis continua of Hallopeau are two different localized subtypes), (iv) erythrodermic psoriasis (1% to 2.5% of psoriasis patients have erythrodermic psoriasis, which is one of the rarest and most severe types; it is characterized by significant morbidity), and (v) psoriatic arthritis (20% to 30% of people suffering from psoriasis also have psoriatic arthritis; a chronic, genetically-determined musculoskeletal condition that frequently appears a few years after the onset of psoriasis)[2,9].

The majority of psoriasis diagnoses are clinical; 80% to 90% of people with psoriasis have chronic plaque psoriasis, which is the most prevalent clinical type. Erythematous, symmetrical, well-defined plaques covered in silvery scales are the hallmark of classic plaque psoriasis. Although plaques can develop anywhere on the body, the scalp, trunk, buttocks, and extremities are where they are most commonly encountered. There may also be nail involvement, occasionally without plaques present. Itchy or painful lesions are possible. Clinical presentation is used in order to make the diagnosis[9].

6. Treatment and management of psoriasis

The UK National Institute for Health and Care Excellence recommendations list topical therapy, phototherapy, and systemic treatment as possibilities for treating psoriasis. Topical, systemic, and phototherapeutic are the basic categories into which these treatments can be divided. When psoriasis is mild, topical treatment is usually advised. Psoriasis is typically treated with corticosteroids, biologic medicines, methotrexate, cyclosporine, acitretin, calcineurin inhibitors, vitamin D3 analogs, and phototherapy[9].

6.1. Topical therapies

The first-line treatment for mild, localized psoriasis is topical therapy. It can lower the percentage of the body surface area that is impacted by plaque lesions, as well as their quantity and thickness. Despite the current availability of biologic medications and apremilast for the treatment of psoriasis, the ongoing need for new therapies remains, particularly in order to tailor treatment approaches based on individual patient clinical characteristics and comorbidities, and to achieve a sustained therapeutic response[9].

6.2. Systemic therapies

Poor tolerability and cumulative toxicity are the main drawbacks of using traditional systemic psoriasis therapies for an extended period of time. Examples include liver damage from methotrexate, kidney toxicity from cyclosporine, and skin carcinogenesis from phototherapy (especially psoralens with ultraviolet A). A biological drug called "etanercept" has been authorized for the treatment of psoriasis[10].

7. Conclusion

PHARMAKEFTIKI, 37, 2S, 2025 | 442-445

Psoriasis is a skin condition that can reduce life expectancy due to its significant negative impact on patients' quality of life and its increased risk of various comorbidities. Psoriasis also has a substantial socioeconomic impact on society. A better understanding of its pathophysiology could lead to the development of more effective treatments.

Acknowledgements

None.

Conflicts of interest

None exist.

ORCIDs

0009-0009-2454-0267 (G.A.O. Abdulridha); 0000-0002-4950-132X (S.K.S. Al-Zamali); 0009-0005-5036-451X (M.S. Otaiwi); 0000-0002-5028-4510 (H.A. Ali); 0009-0006-1323-5505 (M.S. Mohammed)

References

1. Zhou X., Chen Y., Cui L., Shi Y., Guo C. Advances in the pathogenesis of psoriasis: from keratinocyte perspective. *Cell Death Dis.* 13(1), 81, 2022. DOI: 10.1038/s41419-022-04523-3
2. Cao L., Lu L., Yu Y., Zhou H., Lin B. Clinical characteristics of patients with a family history of psoriasis: an observational epidemiological study in Chinese Han population. *Front. Med. (Lausanne)* 11, 1455953, 2024. DOI: 10.3389/fmed.2024.1455953
3. Abdul-Hussein A.A., Hussain F.E. Childhood psoriasis: a clinical and epidemiological study in Samawa city. *J. Pak. Assoc. Dermatol.* 30(2), 267–270, 2020.
4. Murad A.A.K., Hussien W.H. Incidence of psoriasis in patients with different skin diseases in Baquba city. *Diyala J. Med.* 12(1), 25–28, 2017.
5. Razzaq M.S.A., Al-Saadi M.A.K., Ahmed M.M., Naji A.T. Assessment of genetic variations associated with susceptibility to psoriasis among Iraqi population. *Karbala J. Med.* 8(1), 2049–2055 (2015).
6. Vičić M., Kaštelan M., Brajac I., Sotošek V., Massari L.P. Current concepts of psoriasis immunopathogenesis. *Int. J. Mol. Sci.* 22(21), 11574, 2021. DOI: 10.3390/ijms222111574
7. Ellinghaus E., Stuart P.E., Ellinghaus D., Nair R.P., Debrus S., Raelson J.V., *et al.* Genome-wide meta-analysis of psoriatic arthritis identifies susceptibility locus at REL. *J. Invest. Dermatol.* 132(4), 1133–1140, 2012. DOI: 10.1038/jid.2011.415
8. Dand N., Mahil S.K., Capon F., Smith C.H., Simpson M.A., Barker J.N. Psoriasis and genetics. *Acta Derm. Venereol.* 100(3), adv00030, 2020. DOI: 10.2340/00015555-3384
9. Elmets C.A., Leonardi C.L., Davis D.M.R., Gelfand J.M., Lichten J., Mehta N.N., *et al.* Joint AAD-NPF guidelines of care for the management and treatment of psoriasis with awareness and attention to comorbidities. *J. Am. Acad. Dermatol.* 80(4), 1073–1113, 2019. DOI: 10.1016/j.jaad.2018.11.058
10. Kuba R.H., Al-Qadhi B.N., Fadheel B.M. Effect of the biological drug etanercept on tumor necrosis factor-α levels in psoriatic patients. *Iraqi J. Sci.* 59(2C), 998–1005, 2018.

HOW TO CITE:
Abdulridha G.A.O., Al-Zamali S.K.S., Otaiwi M.S., Ali H.A., Mohammed M.S. Psoriasis: a short review of its pathophysiology and causes. *Pharmakeftiki* 37(2s), 442-445, 2025. https://doi.org/10.60988/p.v37i2S.251