



ETIOLOGY, CLINICAL STAGING AND EVIDENCE-BASED TREATMENT APPROACHES FOR PSORIASIS

Boyniyozova Nigora Otabekovna

Student of General Medicine, Faculty of Medicine,

Termiz University of Economics and Service.

boyniyozovanigora@gmail.com

Author: Chariyev Muzaffar Yo'ldashevich

Orcid: <https://orcid.org/0009-0006-8995-343>

muzaffar86chariyev@gmail.com

Abstract: Psoriasis is a chronic immune-mediated inflammatory skin disease with systemic implications. It is no longer interpreted only as a cosmetic disorder or a simple disturbance of keratinization; contemporary dermatology defines it as a complex inflammatory condition in which genetic predisposition, environmental triggers, dysregulated innate and adaptive immunity, and metabolic comorbidity interact. The methodological basis of the article is a narrative synthesis of dermatology textbooks used in Uzbekistan and current international guidance from dermatology organizations and public health institutions. The results show that plaque psoriasis remains the most common clinical phenotype, but scalp, nail, inverse, guttate, pustular and erythrodermic variants require differentiated clinical thinking. Severity assessment should not rely only on the visible area of lesions; special-site involvement, pruritus, pain, quality-of-life impairment and symptoms of psoriatic arthritis must also be considered. Treatment follows a stepwise model: topical therapy for limited disease, phototherapy for more extensive or resistant cases, and systemic or biologic therapy for moderate-to-severe disease or functionally disabling involvement. The article concludes that effective psoriasis management requires early recognition, careful staging, comorbidity screening and patient-centered long-term control rather than short-term suppression of plaques alone.

Keywords: psoriasis; plaque psoriasis; immune-mediated inflammation; PASI; topical therapy; phototherapy; biologics; psoriatic arthritis; dermatology.

Introduction

Psoriasis is one of the most important chronic dermatoses in clinical dermatology because it combines visible skin lesions, recurrent inflammation, psychosocial burden and systemic comorbidity. In everyday language it is often described as a scaly skin condition, but this description is incomplete. The disease involves accelerated epidermal turnover, abnormal differentiation of keratinocytes, activation of dendritic cells and T lymphocytes, and increased production of inflammatory mediators such as tumor necrosis factor alpha, interleukin 17 and interleukin 23. These mechanisms explain why psoriasis may persist for years, relapse after apparent improvement and respond to targeted immunomodulatory medicines.

The clinical importance of psoriasis is also connected with its prevalence and social effect. The World Health Organization has emphasized that psoriasis can involve the skin, nails and joints, may be painful and stigmatizing, and can negatively influence quality of life. Patients with visible lesions on the face, scalp, hands or genital region may experience embarrassment, reduced social participation and anxiety. In adolescents and young adults, the impact may be especially strong because appearance, peer interaction and self-confidence are central to daily life. Therefore, the goal of medical care is not simply to remove scales; it is to reduce inflammation, prevent complications, support psychological well-being and restore normal participation in education, work and family life.



Etiology is multifactorial. Genetic susceptibility increases the probability of disease, but genetic risk alone does not determine the clinical course. Streptococcal infection may precede guttate psoriasis, mechanical trauma may provoke new lesions through the Koebner phenomenon, and stress, smoking, alcohol misuse, obesity, certain medicines and abrupt withdrawal of systemic corticosteroids may aggravate disease activity. These triggers vary by patient, which means that management must include individualized education and follow-up rather than a universal prescription.

Modern treatment has changed radically during the last two decades. Classical approaches such as emollients, keratolytics, topical corticosteroids, vitamin D analogues and phototherapy remain important. However, systemic agents and biologic medicines have transformed the prognosis of patients with moderate-to-severe psoriasis. The availability of therapies targeting TNF-alpha, IL-17 and IL-23 pathways demonstrates how understanding of pathogenesis has become directly linked with treatment choice. At the same time, high treatment cost, infection risk, laboratory monitoring, pregnancy considerations and access inequality remain serious limitations. For this reason, a rational clinical model must balance efficacy, safety, availability and patient preference.

The purpose of this article is to summarize psoriasis as an etiological, clinical and therapeutic problem in a format appropriate for international publication. The article focuses on the causes and triggers of psoriasis, clinical forms and staging, differential diagnosis, and evidence-based treatment strategies. Special attention is paid to the practical point often missed in student presentations: psoriasis should be assessed as a systemic inflammatory disease with skin expression, not as an isolated cosmetic defect.

Materials and Methods

The second group included international clinical guidance and public health documents on psoriasis assessment and management. The third group included modern scientific summaries concerning immunopathogenesis, comorbidity, systemic treatment and biologic therapy.

The review method was analytical rather than experimental. No patients were enrolled and no personal clinical data were collected. The aim was not to calculate prevalence in a local population but to systematize existing knowledge into a coherent academic article. The inclusion criteria for sources were clinical relevance, educational reliability, and usefulness for explaining etiology, diagnosis, staging or treatment. Preference was given to sources that describe practical decision-making: how to recognize psoriasis, how to grade severity, when topical treatment is enough, when phototherapy is indicated, and when systemic or biologic therapy should be considered.

The synthesis was organized around four core questions. First, which etiological factors and triggers are most important for the development and exacerbation of psoriasis? Second, which clinical forms and stages should be distinguished by a clinician or medical student? Third, which assessment tools and comorbidity checks are necessary before selecting treatment? Fourth, how should treatment be selected according to disease severity, lesion localization, patient age, comorbid conditions and expected safety profile? The findings are presented in descriptive form and supported by tables in order to make the material useful for academic writing, teaching and presentation.

Results

The reviewed literature confirms that psoriasis develops through interaction between inherited susceptibility and external or internal triggers. The immune response is characterized by activation of inflammatory pathways that stimulate keratinocyte proliferation and abnormal epidermal maturation. Clinically, this produces erythematous plaques with sharply defined borders and silvery-white scale, most commonly on extensor surfaces, scalp and lumbosacral region. Nevertheless, the disease is



heterogeneous. Some patients have only a few plaques for many years, while others develop widespread disease, nail involvement, palmoplantar lesions, erythroderma or psoriatic arthritis.

Table 1. Main etiological and triggering factors in psoriasis

Factor group	Clinical meaning	Practical implication
Genetic predisposition	Family history increases susceptibility, especially in early-onset disease.	Ask about relatives with psoriasis or psoriatic arthritis.
Immune dysregulation	T-cell activation and cytokine pathways drive epidermal hyperproliferation.	Explains the use of targeted systemic and biologic therapy.
Infections	Streptococcal infection may precede guttate psoriasis.	Evaluate recent sore throat, fever or acute eruptive onset.
Trauma and Koebner phenomenon	New plaques may appear at sites of scratching, pressure, wounds or irritation.	Educate patients to avoid unnecessary skin injury.
Lifestyle and metabolic factors	Obesity, smoking, alcohol use and stress may worsen severity and relapse risk.	Include lifestyle counselling and comorbidity screening.
Medicines and withdrawal effects	Some drugs and abrupt systemic corticosteroid withdrawal may aggravate psoriasis.	Review medication history before changing treatment.

Clinical staging requires more than naming the disease. A patient with a small plaque on the elbow and a patient with limited but painful genital or palmoplantar psoriasis cannot be judged by body surface area alone. In practice, assessment should combine body surface area, lesion thickness and erythema, scaling, special-site involvement, pruritus, pain, nail disease, functional limitation and quality-of-life impairment. The Psoriasis Area and Severity Index is widely used in research and specialty care, while body surface area and physician global assessment are simpler tools for routine practice.

Table 2. Clinical forms of psoriasis and key diagnostic clues

Clinical form	Typical signs	Common localization	Why it matters
Plaque psoriasis	Well-demarcated erythematous plaques with silver scale	Elbows, knees, scalp, lower back	Most common phenotype; basis for routine staging
Guttate psoriasis	Multiple small drop-like papules	Trunk and proximal limbs	Often follows infection, especially in younger patients
Inverse psoriasis	Smooth red lesions with minimal scale	Axillae, groin, inframammary folds	Easily confused with fungal or irritant dermatitis
Scalp psoriasis	Scale, erythema, itching, sometimes hair shedding from scratching	Hairline, occipital scalp, entire scalp	Visible and socially distressing; treatment vehicle is important



Nail psoriasis	Pitting, onycholysis, subungual hyperkeratosis	Fingernails and toenails	Associated with higher risk of psoriatic arthritis
Pustular psoriasis	Sterile pustules on erythematous skin	Localized palms/soles or generalized	May be severe and requires urgent specialist care
Erythrodermic psoriasis	Diffuse redness and scaling over most skin surface	Generalized	Can be life-threatening; urgent care required

Severity stratification leads directly to treatment choice. Mild psoriasis usually means limited body surface area and no major functional impairment. Moderate disease includes broader involvement or lesions that are resistant to simple topical therapy. Severe disease includes extensive plaques, unstable pustular or erythrodermic disease, major quality-of-life impairment, or involvement of areas that interfere with walking, manual work, sleep, sexual health or social communication. Nail disease and joint symptoms should be actively investigated because untreated psoriatic arthritis may lead to disability.

Table 3. Practical treatment model according to severity and clinical context

Clinical situation	Main treatment direction	Examples of therapy groups	Monitoring focus
Limited plaque psoriasis	Topical treatment and skin care	Emollients, topical corticosteroids, vitamin D analogues, keratolytics	Correct application, local side effects, relapse
Scalp or thick plaques	Vehicle-adapted topical therapy	Solutions, gels, foams, shampoos, keratolytic combinations	Adherence, irritation, cosmetic acceptability
Moderate widespread disease	Phototherapy or systemic therapy after assessment	Narrowband UVB, methotrexate, ciclosporin, acitretin, apremilast	Contraindications, pregnancy status, laboratory safety
Moderate-to-severe persistent disease	Targeted systemic or biologic therapy	TNF-alpha, IL-17, IL-23 pathway inhibitors, tyrosine kinase 2 inhibitor where appropriate	Infection screening, response, adverse events, access
Pustular or erythrodermic disease	Urgent specialist management	Systemic therapy, supportive care, hospital referral when unstable	Temperature, hydration, infection, systemic complications
Joint symptoms	Dermatology-rheumatology coordination	Disease-modifying and biologic therapy selected by joint and skin activity	Pain, swelling, function, imaging when needed



The results of the review also show that patient education is a treatment component, not an optional addition. Many patients stop topical therapy too early, apply corticosteroids incorrectly or expect permanent cure after a short course. A realistic explanation should emphasize that psoriasis is controllable but usually chronic. Patients should know the difference between maintenance care and flare treatment, recognize warning signs such as rapidly spreading redness or joint swelling, and understand why follow-up is necessary even when plaques improve.

Discussion

The clinical management of psoriasis has two frequent weaknesses. The first is underestimation of disease burden when the visible body surface area appears small. A few lesions on the palms, soles, face, scalp, nails or genital region may create more disability than a larger but hidden plaque on the trunk. Therefore, the clinician should not mechanically classify disease only by percentage of body surface involvement. Quality of life and functional localization must be included in the decision. The second weakness is excessive focus on external treatment while ignoring systemic associations. Psoriasis is linked with psoriatic arthritis, metabolic syndrome, obesity, hypertension, dyslipidemia and cardiovascular risk. This does not mean that every patient will develop these conditions, but it does mean that screening and preventive counselling are clinically justified.

From an etiological perspective, psoriasis demonstrates the modern concept of gene-environment-immune interaction. Classical Uzbek dermatology textbooks describe the morphology and clinical course of psoriasis in terms that remain useful for teaching: plaques, scaling, chronic recurrence and characteristic localization. International guidelines add a more detailed framework for severity scoring, comorbidity screening and evidence-based selection of therapy. Combining these two traditions is productive. Local medical education provides strong descriptive diagnosis, while contemporary international sources strengthen the logic of staged management and targeted therapy.

Topical therapy remains the foundation for mild disease. Emollients reduce dryness and improve barrier function. Topical corticosteroids reduce inflammation, but their potency and duration must be matched to body site because thin skin areas are more vulnerable to atrophy and irritation. Vitamin D analogues help regulate keratinocyte proliferation and are commonly used alone or in combination. Keratolytics such as salicylic acid may help remove thick scale, especially on the scalp or plaques with heavy hyperkeratosis. The main failure mode of topical therapy is not only weak medicine; it is poor adherence caused by greasy texture, slow response, inconvenient application or fear of side effects. Treatment vehicles should therefore be chosen according to localization and patient routine.

Phototherapy, especially narrowband UVB, is an important option for patients with more widespread plaques or insufficient response to topical therapy. It can be effective and avoids some systemic drug toxicities, but it requires regular visits, equipment access and monitoring for burns or cumulative ultraviolet exposure. In many regions, these logistical requirements limit real-world use. This is a practical tradeoff: phototherapy may be medically appropriate but inaccessible for patients living far from specialized centers.

Systemic conventional therapy is considered when disease is moderate to severe, disabling or resistant. Methotrexate is widely used because it can help both skin and joint disease, but it requires laboratory monitoring and attention to liver, blood and pregnancy-related risks. Ciclosporin may act rapidly, but kidney function and blood pressure are central safety concerns. Acitretin may be useful in selected cases, particularly hyperkeratotic or pustular forms, but teratogenicity makes strict pregnancy prevention essential. Apremilast and newer oral targeted options provide additional choices, although response level, cost and availability vary. No systemic drug should be selected



casually; the correct question is not “Which drug is strongest?” but “Which therapy gives the best benefit-risk balance for this patient?”

Biologic therapy represents the most pathogenesis-based treatment strategy. Agents targeting TNF-alpha, IL-17 or IL-23 pathways can produce major improvement in appropriately selected patients. Their value is especially high in moderate-to-severe plaque psoriasis, difficult special-site disease and some cases with psoriatic arthritis. However, biologics require screening for infections, assessment of contraindications, adherence to injection schedules and long-term pharmacovigilance. They also create an economic issue: the best biological option scientifically may not be available or affordable in every health system. A realistic article must mention this because treatment recommendations that ignore access are incomplete.

Differential diagnosis is another essential area. Seborrheic dermatitis, tinea corporis, pityriasis rosea, lichen planus, eczema, secondary syphilis, cutaneous lupus and drug eruptions may mimic psoriasis. In typical plaque psoriasis, diagnosis is usually clinical. Biopsy may be helpful when morphology is unusual, treatment response is poor or serious mimics must be excluded. Fungal testing may be needed for inverse or annular lesions before topical steroids are intensified, because steroid use can mask and worsen dermatophyte infection.

Long-term success depends on a patient-centered strategy. The patient should be asked about itching, pain, sleep, embarrassment, clothing limitations, occupational difficulties and treatment acceptability. Advice should include avoidance of smoking, moderation of alcohol, weight management where relevant, stress reduction and careful skin care. These measures do not replace medical treatment, but they reduce triggers and improve overall outcomes. The physician must also avoid unrealistic promises. Psoriasis often cannot be permanently cured, but it can be controlled well enough for many patients to live normally. This distinction is honest, medically accurate and psychologically safer than promising a final cure.

Table 4. Risk-based follow-up questions for patients with psoriasis

Clinical domain	Question to ask	Reason
Joint involvement	Do you have morning stiffness, swollen fingers, heel pain or back pain?	Early detection of psoriatic arthritis prevents disability.
Quality of life	Does psoriasis affect sleep, study, work, clothing or relationships?	Small visible lesions may carry major burden.
Triggers	Did a flare follow infection, stress, trauma, smoking, alcohol or a medicine change?	Trigger control can reduce relapse frequency.
Treatment use	How exactly do you apply your creams or medicines?	Incorrect use is a common reason for failure.
Comorbidity	Have blood pressure, weight, glucose and lipids been checked?	Psoriasis is associated with cardiometabolic risk.

Conclusion

Psoriasis is a chronic immune-mediated inflammatory disease with diverse clinical forms and systemic implications. Its etiology cannot be reduced to a single cause. Genetic predisposition, immune dysregulation, infection, mechanical trauma, lifestyle factors, stress and medication-related



triggers interact to shape onset and relapse. The most common presentation is plaque psoriasis, but scalp, nail, inverse, guttate, pustular and erythrodermic variants require specific diagnostic attention.

A scientifically sound management plan begins with accurate staging. Body surface area is useful, but it is insufficient by itself. Special-site involvement, nail disease, pruritus, pain, psychological burden, functional limitation and symptoms of psoriatic arthritis must be included. Treatment should be stepwise and individualized: topical therapy for limited disease, phototherapy for appropriate moderate cases, and systemic or biologic treatment for moderate-to-severe, refractory or disabling disease. Safety monitoring, infection screening, pregnancy considerations and comorbidity assessment are compulsory parts of responsible care.

The practical conclusion is clear: psoriasis must be managed as a long-term inflammatory condition, not as a temporary rash. The strongest treatment strategy combines evidence-based medicine, realistic patient education, follow-up and attention to quality of life. Such an approach improves not only skin clearance but also social functioning, emotional stability and prevention of complications.

References

1. World Health Organization. Global Report on Psoriasis. Geneva: World Health Organization, 2016.
2. National Institute for Health and Care Excellence. Psoriasis: Assessment and Management. Clinical guideline CG153. London: NICE, last reviewed 2025.
3. American Academy of Dermatology. Psoriasis Clinical Guideline: Topical Therapy, Severity Assessment and Management Recommendations. Rosemont: AAD, current online guideline.
4. European Dermatology Forum and EuroGuiDerm. Guideline for the Systemic Treatment of Psoriasis Vulgaris. Updated edition, 2025.
5. Vaisov A.Sh. Skin and Venereal Diseases. Translated title from Uzbek: Teri va tanosil kasalliklari. Tashkent: Yangi Asr Avlodi, 2009.
6. Arifov S.S., Eshboyev E. Skin and Venereal Diseases. Translated title from Uzbek: Teri va tanosil kasalliklari. Tashkent, 1997.
7. Arifov S.S. Skin and Venereal Diseases. Translated title from Uzbek: Teri va tanosil kasalliklari. Tashkent, 2010.
8. Arifov S.S. Psoriasis. Translated title from Uzbek: Psoriaz. Tashkent, 2019.
9. Djumayeva M.K., Haydarova H.A., Naimova Sh.N. Causes and Treatment Methods of Psoriasis. Translated title from Uzbek: Psoriaz kasalligining kelib chiqish sabablari va davolash usullari. Education, Science and Innovative Ideas in the World, 2024.
10. Boehncke W.H., Schon M.P. Psoriasis. The Lancet, 2015.
11. Armstrong A.W., Read C. Pathophysiology, Clinical Presentation, and Treatment of Psoriasis: A Review. JAMA, 2020.
12. Rendon A., Schakel K. Psoriasis Pathogenesis and Treatment. International Journal of Molecular Sciences, 2019.
13. Menter A., Strober B.E., Kaplan D.H. et al. Joint AAD-NPF Guidelines of Care for the Management and Treatment of Psoriasis with Biologics. Journal of the American Academy of Dermatology, 2019.
14. Menter A., Gelfand J.M., Connor C. et al. Joint AAD-NPF Guidelines of Care for the Management of Psoriasis with Phototherapy and Systemic Nonbiologic Agents. Journal of the American Academy of Dermatology, 2020.



15. Elmets C.A., Korman N.J., Prater E.F. et al. Joint AAD-NPF Guidelines of Care for the Management and Treatment of Psoriasis with Topical Therapy. Journal of the American Academy of Dermatology, 2021.
16. Nestle F.O., Kaplan D.H., Barker J. Psoriasis. New England Journal of Medicine, 2009.