Psoriasis—A Chronic Immune-Mediated Disease: Comprehensive Review of Mechanisms, Diagnosis and Treatment

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Abstract

Psoriasis is a persistent inflammatory skin condition driven by immune system dysregulation, leading to excessive growth of keratinocytes and the development of thick, scaly, and inflamed plaques. It affects around 2–3% of people worldwide and poses a major burden on patients' physical health, emotional well-being, and social functioning. This review aims to deliver an in-depth understanding of psoriasis by discussing its epidemiology, underlying mechanisms, clinical characteristics, and current therapeutic strategies. Despite major progress in topical formulations, light-based therapies, and systemic treatments, issues such as side effects, resistance to therapy, and the need for lifelong disease control remain unresolved. The paper highlights the importance of a collaborative, multidisciplinary approach to achieve effective disease management and better patient outcomes. In addition, it explores the latest advancements in psoriasis therapy, focusing on biological agents that act on novel molecular targets, combined treatment approaches, and the move toward personalized care using biomarkers and multi-omics-based technologies. Overall, this review seeks to contribute to continued efforts aimed at improving both clinical outcomes and quality of life for individuals living with psoriasis.

Keywords: Psoriasis, inflammatory skin disorder, immune dysregulation, keratinocyte hyperproliferation, epidemiology, pathophysiology, clinical presentation, topical therapy, phototherapy, systemic therapy, adverse reactions, resistance, chronic management, multidisciplinary care, biologic therapy, combination strategy, personalized medicine, biomarkers, multi-omics approaches.

Introduction

Psoriasis is a persistent, immune-mediated disorder that primarily involves the skin and joints. It is defined by an abnormally rapid turnover of keratinocytes, leading to the development of thickened, scaly, and erythematous plaques that are often associated with itching and irritation. Affecting approximately 2–3% of the global population, psoriasis imposes a substantial physical, psychological, and socioeconomic burden on affected individuals. The disease exhibits multiple clinical variants, each with distinctive morphological and pathological characteristics. Among these, chronic plaque psoriasis is the most prevalent, accounting for nearly 80–90% of all cases. It typically presents as well demarcated, silvery-scaled plaques localized on common sites such as the elbows, knees, and scalp [1]. Other subtypes, including guttate, inverse, pustular, and erythrodermic psoriasis, present unique diagnostic complexities and therapeutic challenges [2].

Psoriasis is increasingly recognized as a systemic inflammatory disorder rather than a purely cutaneous condition. It is associated with several comorbidities, notably cardiovascular diseases, metabolic syndrome, and psychiatric conditions such as depression and anxiety [3]. Up to 30% of patients develop psoriatic arthritis, characterized by inflammation of the joints resulting in pain, stiffness, and swelling. Over time, significant advances have been made in disease management, including the use of topical agents, phototherapy, systemic medications, and biologic therapies. However, therapeutic decision making remains challenging due to potential adverse effects, drug resistance, and the requirement for lifelong management [4].

This review aims to present a detailed overview of psoriasis—its epidemiology, pathophysiology, clinical presentation, and available therapeutic modalities—while emphasizing the importance of a multidisciplinary approach to enhance patient care and overall quality of life.

Types of Psoriasis

Psoriasis is a chronic inflammatory skin disorder characterized by an accelerated cycle of epidermal cell turnover, leading to the accumulation of thick, scaly lesions on the skin surface. It affects roughly 2–3% of the global population and can significantly impair quality of life due to its chronic nature, visibility, and associated discomfort.

1. **Plaque Psoriasis**:

This is the most common type of psoriasis, presenting as raised, inflamed, and scaly patches that may occur on any part of the body, particularly the elbows, knees, scalp, and lower back. These lesions are typically well-defined and covered with silvery-white scales [5].

2. **Guttate Psoriasis:**

Characterized by numerous small, drop-shaped lesions, guttate psoriasis often develops suddenly following an upper respiratory tract infection, especially streptococcal pharyngitis. It primarily affects children and young adults and may resolve spontaneously or progress to chronic plaque psoriasis.[5]

3. **Inverse Psoriasis**:

Inverse psoriasis manifests as smooth, shiny, and red lesions that develop within skin folds such as the groin, under the breasts, and buttocks. The affected areas are prone to irritation from friction and sweating, often complicating diagnosis and management [5].

4. **Pustular Psoriasis**:

This form is defined by the presence of white pustules filled with sterile pus, surrounded by erythematous skin. It may be localized (commonly on the palms and soles) or generalized, potentially covering large body areas and causing systemic symptoms [6].

5. **Erythrodermic Psoriasis:**

A rare but severe type of psoriasis characterized by diffuse redness, intense itching, and pain affecting most of the body surface. It can disrupt normal thermoregulation and lead to severe complications, making it a dermatological emergency that requires immediate medical intervention [7].

6. **Nail Psoriasis:**

An autoimmune inflammatory condition that alters the appearance and texture of the nails, more frequently affecting fingernails than toenails. It may cause pitting, discoloration, thickening, and separation from the nail bed. Nail psoriasis can also serve as an early indicator of psoriatic arthritis [5][6].

7. **Psoriatic Arthritis:**

A chronic, inflammatory arthropathy associated with psoriasis, characterized by joint pain, stiffness, and swelling. If left untreated, it can cause irreversible joint damage and functional disability, highlighting the need for early diagnosis and intervention [6]

Epidemiology

According to the World Psoriasis Day Consortium, psoriasis affects nearly 125 million individuals worldwide, representing approximately 2–3% of the global population [8]. In Western countries, the prevalence is around 2%, whereas the lowest prevalence has been documented in East Asia, estimated at 0.12%. Within Europe, the United Kingdom ranks 21st globally for psoriasis prevalence, with nearly 1.1 million individuals living with the disease [9]. Research from The University of Manchester and the University Medical Center Hamburg-Eppendorf reports that approximately 1.7% of the UK population is affected by this chronic inflammatory skin condition.

Although psoriasis occurs worldwide, its prevalence varies substantially across different geographic and ethnic populations [4]. In the United States, approximately 2% of the population is affected. Exceptionally high prevalence rates have been observed among individuals in the Faroe Islands, where one investigation recorded a 2.8% prevalence. Conversely, psoriasis is relatively rare among Japanese populations and is virtually absent among certain Aboriginal Australian and South American Indian communities [Convit J The incidence of psoriasis among Latin-American Indians was investigated]

Prevalence in Children

A total of twelve studies encompassing ten populations of children under 18 years of age were analyzed to determine pediatric psoriasis prevalence [11]. The highest prevalence was reported in Germany, where a lifetime prevalence study involving 16,500 participants revealed a rate of 1.37%. In contrast, the lowest prevalence was documented in Taiwan, showing 0% prevalence in two separate point prevalence studies conducted among 4,067 and 3,273 schoolchildren, respectively. Overall, European pediatric populations demonstrated a higher prevalence compared to African and Asian populations [10].

Prevalence in Adults

Among adults, twenty-five studies assessed the prevalence of psoriasis, with populations generally defined as individuals aged 15 years and above. Reported prevalence values ranged between 0.51% and 11.43%, depending on geographic region and diagnostic criteria. The highest adult prevalence was found in Norway (11.43%), followed by the United Kingdom (1.31%). Studies conducted in Australia indicated rates varying from 2.30% to 6.60%, while research from Brazil reported a prevalence of
1.30%, which represents the only available epidemiological data from South America [11].

Etiology

Psoriasis is a common chronic condition that can develop in individuals of any age, though it most frequently appears between the ages of 15 and 35, and may also arise later in life. Importantly, psoriasis is not contagious and cannot be transmitted through physical contact. Evidence suggests a familial pattern, indicating a strong genetic predisposition. In normal skin physiology, keratinocytes form in the deeper layers of the epidermis and migrate to the surface approximately every 28–30 days. However, in individuals with psoriasis, this cell turnover occurs much more rapidly—within about 14 days— leading to the accumulation of dead skin cells that form the characteristic scaly plaques [12].

The precise cause of psoriasis remains uncertain, but it is widely accepted to result from an interaction between genetic susceptibility and environmental triggers. Although the condition is most prevalent among Caucasian and light-skinned individuals, it can affect people of any ethnicity and impacts both genders equally. The immune system plays a pivotal role in disease development, where abnormal immune activation drives chronic inflammation [13]. Psoriasis is a heterogeneous disorder comprising several clinical subtypes, with plaque psoriasis (psoriasis vulgaris) being the most common form. It manifests as dry, raised, and scaly lesions typically found on the elbows, knees, scalp, and lower back [14].

Major Factors Contributing to Psoriasis Etiology [15]

1. **Genetic Factors:**

Psoriasis often shows familial clustering, suggesting a hereditary basis. Genetic variations such as HLA-Cw6 have been strongly linked to increased disease risk and earlier onset.

2. **Immune System Dysfunction:**

An overactive immune response, primarily mediated by T lymphocytes, targets healthy skin cells, resulting in inflammation and accelerated keratinocyte turnover.

: **3. Environmental Triggers**

- Infections: Streptococcal throat infections are recognized triggers for guttate psoriasis.
- Skin Trauma: Injuries such as cuts, burns, or sunburns may induce psoriatic lesions, known as the Koebner phenomenon.

4. **Lifestyle Factors:**

- Stress: Psychological or emotional stress may initiate or worsen psoriatic flares.
- Smoking and Alcohol: Both are associated with disease exacerbation and poor treatment outcomes.

5. **Medications:**

Certain drugs-including lithium, beta-blockers, antimalarials, and NSAIDs—have been implicated in triggering or aggravating psoriasis.

6. **Hormonal Changes:**

Fluctuations in hormone levels, particularly during puberty or menopause, can influence disease activity.

7. **Diet and Obesity**:

Obesity is a recognized aggravating factor, especially in intertriginous areas, and dietary patterns may affect disease severity, though the exact mechanisms are not yet fully understood.

Understanding these etiological contributors is crucial for effective management and prevention of flare-ups in patients with psoriasis.

Symptoms

Psoriasis is a chronic autoimmune disorder that primarily affects the skin, but can also involve joints and other systems. Although symptoms vary by subtype, most patients share certain hallmark characteristics. The predominant form, chronic plaque psoriasis, accounts for 80–90% of cases and typically presents as symmetrical, erythematous plaques with overlying silvery-white scales.

Common Symptoms Across Psoriasis Variants

1. **Plaque Psoriasis**:

The most frequent type, it presents as well-defined, round or oval plaques with loosely adherent silvery scales. Lesions typically occur on the elbows, knees, scalp, and lower back, but may appear anywhere. In severe cases, skin fissures near joints may bleed or become painful [1].

2. **Guttate Psoriasis**:

Characterized by numerous small, drop-like papules that emerge suddenly, often following a streptococcal infection. The lesions, typically 2–6 mm in diameter, are distributed across the trunk and limbs [16].

3. **Inverse Psoriasis:**

Presents as smooth, shiny, and erythematous lesions in intertriginous regions such as the armpits, groin, and under the breasts. This variant lacks the typical scaling of plaque psoriasis and is often worsened by moisture and friction [17].

4. **Erythrodermic Psoriasis:**

A rare and severe form that can involve extensive body surface areas, producing widespread redness, peeling, and intense itching or burning. It may cause systemic complications such as protein and fluid imbalance, making it a potentially life-threatening condition requiring urgent care [18].

5. **Nail Psoriasis:**

Affects fingernails and toenails, causing pitting, thickening, discoloration, and separation from the nail bed (onycholysis). Severe cases may lead to nail crumbling or complete detachment [19].

6. **Psoriatic Arthritis (PsA):**

A chronic inflammatory joint disease seen in some psoriasis patients. It causes joint pain, stiffness, swelling, and can affect tendons and ligaments, resulting in enthesitis and dactylitis (commonly known as “sausage digits”). PsA may also contribute to fatigue and limited mobility, significantly impairing quality of life [8].

Pathophysiology

Psoriasis is a multifactorial autoimmune condition with a complex and still-evolving pathophysiological understanding. It is recognized as an immune-mediated inflammatory skin disease occurring in genetically predisposed individuals who are exposed to specific environmental triggers [21].

The disease involves multiple cellular and molecular components, including T cells, antigen-presenting cells (APCs), keratinocytes, macrophages, natural killer (NK) cells, and Langerhans cells. These immune cells interact through a cascade of cytokines and growth factors, such as vascular endothelial growth factor (VEGF) and keratinocyte growth factor (KGF), promoting inflammation and epidermal hyperplasia [24].

Unlike many autoimmune disorders, psoriasis lacks a standardized animal model, making research heavily reliant on human clinical and translational studies. The immune dysregulation in psoriasis leads to chronic skin inflammation, driven by aberrant activation of T lymphocytes, particularly Th1, Th17, and Th22 cells. These cells secrete pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF-α), interleukin-17 (IL-17), interleukin-23 (IL-23), and interleukin-22 (IL-22).

These cytokines orchestrate keratinocyte hyperproliferation, abnormal differentiation, and recruitment of inflammatory cells into the skin, leading to the formation of psoriatic plaques. Additionally, genetic factors such as the PSORS1 locus are strongly associated with familial psoriasis.

Overall, psoriasis results from a complex interplay of genetic susceptibility, immune dysregulation, and environmental stimuli. Understanding these mechanisms is essential for the development of targeted therapies, such as biologic agents that block specific cytokine pathways, thereby improving both disease control and patient outcomes [6].

Genetic and Molecular Factors

Genetic predisposition plays a crucial role in psoriasis susceptibility and disease expression. Several gene loci have been implicated, including HLA-Cw*0602, SLC9A3R1, NAT9, and RAPTOR. Among these, HLA-Cw*0602, located within the major histocompatibility complex (MHC), is the most well-established genetic marker, present in approximately 50% of psoriasis patients. This allele is thought to present self-antigens that provoke an inappropriate immune response.

Genes such as SLC9A3R1 and NAT9 (both located on chromosome 17q) regulate signal transduction and cell growth, with certain variants contributing to sustained inflammation and increased keratinocyte reactivity. Similarly, RAPTOR, a component of the mTOR signaling pathway, influences T-cell growth and activation, and genetic variations may lead to altered immune regulation [26].

Molecular studies have revealed that over 1,300 genes exhibit differential expression between lesional and non-lesional skin. Upregulation of cytokines and chemokines such as CCL20 and IL-23 contributes to immune cell recruitment and maintenance of chronic inflammation. Furthermore, elevated numbers of plasmacytoid dendritic cells (pDCs) producing interferon-alpha (IFN-α) highlight the role of innate immunity in disease initiation. Myeloid dendritic cells (mDCs) secrete IL-12 and IL-23, promoting Th1 and Th17 differentiation, thereby reinforcing the inflammatory cycle [27].

Angiogenesis also contributes to psoriatic pathology. Elevated levels of vascular endothelial growth factor (VEGF) and interleukin-8 (IL-8) produced by keratinocytes promote new blood vessel formation, enhancing immune cell infiltration into the skin. These vascular changes support persistent inflammation. Treatments targeting cytokine pathways, such as TNF-α inhibitors and IL-17 blockers, have demonstrated efficacy by disrupting these inflammatory loops and reducing keratinocyte hyperproliferation [28].

In summary, psoriasis arises from a dynamic interplay of genetic, immunologic, and environmental factors. The reciprocal activation of T cells, dendritic cells, and keratinocytes, mediated through a network of cytokines, chemokines, and growth factors, culminates in the chronic inflammatory phenotype of psoriasis [29].

Co-occurring Diseases

Psoriasis extends beyond a cutaneous condition and is associated with multiple systemic comorbidities. Common coexisting disorders include cardiovascular disease, metabolic syndrome, obesity, hypertension, dyslipidemia, and non-alcoholic fatty liver disease (NAFLD) [30]. The systemic inflammatory burden characteristic of psoriasis contributes to endothelial dysfunction, promoting atherogenesis and increasing the risk of cardiovascular events [4][33].

Additionally, individuals with psoriasis show higher rates of psychological disorders, particularly depression and anxiety, which severely impact quality of life. Thus, a comprehensive, multidisciplinary approach that integrates both dermatological and systemic management is essential for optimal care [33].

Psoriasis and Type 2 Diabetes Mellitus

A strong association exists between psoriasis and Type 2 Diabetes Mellitus (T2DM), with prevalence rates of approximately 11.6% among psoriatic patients [4]. This connection is attributed to shared genetic factors and inflammatory mechanisms, where persistent systemic inflammation induces insulin resistance, a key contributor to T2DM [33].

Elevated pro-inflammatory cytokines—including TNF-α, IL-17, and IL-23—play central roles in impairing insulin signaling [32]. Furthermore, adipocytokine imbalances, such as reduced adiponectin and increased resistin and vaspin, exacerbate metabolic dysfunction. Psoriatic patients frequently exhibit elevated HOMA-IR levels, indicating insulin resistance.

Markers of endothelial dysfunction, such as soluble intercellular adhesion molecule-1 (sICAM-1) and matrix metalloproteinase-9 (MMP-9), are elevated in both psoriasis and T2DM, emphasizing systemic inflammation as a shared mechanism [31]. Notably, therapies that reduce psoriatic inflammation often correlate with improved insulin sensitivity. Therefore, effective anti-inflammatory management can help reduce T2DM risk, highlighting the necessity for integrated metabolic and dermatologic care [34].

Pharmacotherapy

The primary goal of psoriasis pharmacotherapy is to inhibit rapid keratinocyte turnover, reduce inflammation, and alleviate symptoms. Treatment strategies are selected based on disease severity, extent of skin involvement, and response to prior therapies.

Psoriasis management encompasses three major categories of treatment:

1. Topical Therapies:
   These include corticosteroids, vitamin D analogs, calcineurin inhibitors, and coal tar preparations, primarily used for mild to moderate psoriasis.

2. Phototherapy (Light Therapy):
   Exposure to ultraviolet (UV) light, particularly UVB and PUVA (psoralen + UVA), helps slow keratinocyte proliferation and modulate immune activity.

3. Systemic Therapies:
   Used for moderate to severe psoriasis, systemic options include methotrexate, cyclosporine, acitretin, and biologic agents such as TNF-α inhibitors (adalimumab, etanercept) and IL-17 or IL-23 inhibitors (secukinumab, ustekinumab). These therapies target specific inflammatory pathways, offering long-term disease control and improved patient quality of life.

Topical Therapy

Topical corticosteroids are the most frequently prescribed agents for the management of mild to moderate psoriasis. These medications, available in forms such as oils, creams, and ointments, act by reducing inflammation and slowing keratinocyte proliferation [12]. Mild corticosteroids such as hydrocortisone are recommended for sensitive areas like the face and skin folds, whereas potent corticosteroids such as clobetasol are preferred for thicker plaques and stubborn lesions [13]. However, prolonged use may result in skin thinning, tolerance, and diminished effectiveness over time [37].

Vitamin D analogues such as calcipotriene and calcitriol serve as another cornerstone of topical therapy. These synthetic vitamin D derivatives inhibit keratinocyte proliferation and are often combined with corticosteroids for enhanced efficacy [36]. Topical retinoids, including tazarotene, promote keratinocyte differentiation and normalize epidermal growth, although they may cause irritation, erythema, and photosensitivity; thus, they are contraindicated in pregnant or breastfeeding women [8].

Calcineurin inhibitors like tacrolimus and pimecrolimus are beneficial in reducing inflammation, especially in delicate regions such as periocular or intertriginous areas. Their use, however, is restricted due to the potential risk of lymphoma and skin malignancies with long-term therapy [13]. Additional topical agents include salicylic acid, which assists in scale removal, and coal tar, known for its anti-inflammatory and antipruritic properties but limited by its odor and messy application.

Light Therapy (Phototherapy)

Phototherapy remains a standard treatment for moderate to severe psoriasis, involving controlled exposure to natural or artificial ultraviolet (UV) light. Available modalities include broadband UVB, narrowband UVB, and PUVA (psoralen plus UVA). Among these, narrowband UVB has become the preferred approach due to greater efficacy and reduced adverse effects [38].

Excimer laser therapy, which utilizes targeted UVB light, delivers focused treatment to affected areas, minimizing exposure to surrounding healthy skin and requiring fewer sessions [39]. Despite its benefits, phototherapy can occasionally lead to erythema, dryness, itching, and photosensitivity reactions [13].

Systemic Treatments

For moderate to severe psoriasis, or cases unresponsive to topical and phototherapy approaches, systemic medications are indicated. These include steroids, retinoids, biologics, methotrexate, and cyclosporine. Intralesional corticosteroids such as triamcinolone can be administered directly into resistant plaques for localized effect [12].

Retinoids like acitretin reduce epidermal turnover but are contraindicated during pregnancy and lactation due to risks of teratogenicity and systemic side effects like xerosis and myalgia [36]. Biologic agents—including etanercept, infliximab, and adalimumab—have revolutionized treatment by targeting specific immune pathways, although they are costly and associated with increased infection risk, necessitating tuberculosis screening before initiation.

Methotrexate, administered weekly, inhibits DNA synthesis, reducing both cell proliferation and inflammation, but requires regular liver function monitoring to prevent hepatotoxicity. Cyclosporine provides rapid improvement in severe cases but is restricted to short-term use (≤1 year) due to risks of nephrotoxicity and hypertension [13].

Emerging Therapies

Despite the progress achieved with current treatments, the search for safer and more effective options continues. New pharmacological agents are under investigation, such as ISA247 (voclosporin), which demonstrated promising results in Phase III clinical trials for moderate to severe plaque psoriasis [37]. Similarly, Bz-423, a 1,4-benzodiazepine derivative, has shown preclinical efficacy by targeting keratinocyte proliferation and promoting apoptosis of hyperactive cells [8].

In summary, although multiple therapies exist—from topical treatments to systemic biologics—the development of novel agents remains essential to overcome limitations such as side effects, tolerance, and cost, ultimately improving patient outcomes and quality of life.

Adverse Drug Reactions (ADRs)

Management of psoriasis often requires long-term pharmacotherapy, and each therapeutic class carries the potential for adverse drug reactions (ADRs). Understanding and mitigating these effects is critical to ensure safe and effective treatment.

Topical Treatments

- Anthralin: Slows epidermal growth but commonly causes skin irritation and staining of skin, clothing, and surfaces [30].
- Coal Tar: Provides anti-inflammatory benefits but may lead to dryness, photosensitivity, and irritation [32].
- Corticosteroids: Effective anti-inflammatory agents but prolonged use can result in skin atrophy, osteoporosis, ulceration, and cataract formation.
- Salicylic Acid: Helps in desquamation but may induce irritation or hair fragility when applied on the scalp [40].
- Vitamin D Analogues: Agents such as calcipotriene and calcitriol may cause local irritation and burning sensation, limiting patient adherence [44].

Light Therapy (Phototherapy)

Phototherapy effectively reduces psoriatic inflammation but carries potential risks such as mild burns, skin aging, and a heightened risk of skin cancer with prolonged exposure. Photochemotherapy (PUVA), combining psoralen with UVA light, may cause nausea, erythema, and pruritus [39].

Systemic Treatments

- Retinoids: May cause teratogenicity, hepatotoxicity, and hyperlipidemia, along with dryness and arthralgia [33].
- Methotrexate: Associated with fatigue, gastrointestinal discomfort, bone marrow suppression, and irreversible liver injury on chronic use.
- Cyclosporine: Carries risks of nephrotoxicity, hypertension, and increased infection susceptibility, especially with prolonged therapy.
- Biologics: While effective, biologics can predispose patients to serious infections, tuberculosis reactivation, and potential malignancies [34].

Monitoring and Management

Due to these risks, continuous patient monitoring is essential. Regular liver function tests, renal assessments, and hematological evaluations help in early detection of toxicity. Patient education about ADR recognition and timely reporting significantly improves treatment safety and efficacy.

Clinical Diagnosis

The diagnosis of psoriasis is primarily clinical, based on visual inspection and characteristic lesion morphology. The most common form, chronic plaque psoriasis, accounts for 80–90% of cases and presents with well-demarcated, erythematous plaques covered by silvery-white scales [1]. Less common variants include inverse, pustular, guttate, erythrodermic, and annular psoriasis, each distinguishable by distinct morphological features [2].

<u>Morphological Differentiation</u>

1.Plaque Psoriasis
Lesions present as dry, sharply defined plaques with loosely adherent silvery scales, typically distributed symmetrically over the elbows and knees, though they can occur anywhere on the body [41].

2.Guttate Psoriasis

Characterized by numerous small, drop-like papules and plaques measuring 2–6 mm in diameter, primarily scattered over the trunk and limbs [16].

3. Inverse Psoriasis

Presents as smooth, shiny, and discolored (red, brown, or purple) patches, often moist to touch, localized to skin folds such as the axillae, groin, and under the breasts [17].

Inverse psoriasis presents as smooth, shiny, discolored plaques within intertriginous areas (e.g., axillae, groin, inframammary folds). Lesions may feel moist, and irritation is commonly worsened by friction and sweating.

4.Erythrodermic Psoriasis

Erythrodermic psoriasis is characterized by diffuse erythema and inflammation resembling a severe burn or sunburn, typically involving >90% of body surface area. Patients often experience intense pruritus and a burning sensation [18]. This presentation constitutes a dermatologic emergency due to risks of thermoregulatory dysfunction, fluid/protein loss, and systemic complications.

5. Nail Psoriasis

Nail psoriasis alters the appearance and structure of fingernails and toenails. Findings include thickening, pitting (pinpoint depressions), shape and color changes, and tenderness or pain. Severe cases may progress to onycholysis (nail plate detachment) and crumbling [19][43].

Differential Diagnosis

Key conditions to distinguish from psoriasis include atopic dermatitis, contact dermatitis, lichen planus, secondary syphilis, mycosis fungoides, tinea corporis, and pityriasis rosea. Careful morphologic assessment often suffices for diagnosis. In atypical or equivocal cases, a skin biopsy can be helpful. Tinea is differentiated by fungal culture and KOH preparation of skin scrapings.

Discussion

Psoriasis is a chronic, immune-mediated disorder characterized by keratinocyte hyperproliferation and cutaneous inflammation, often causing significant quality-of-life impairment due to persistent lesions, pruritus, and pain. Optimal care typically requires multimodal therapy tailored to disease severity, treatment response, and patient preferences. This case underscores the value of a multidisciplinary approach—combining topical agents, phototherapy, and lifestyle interventions—while acknowledging that systemic therapies (including biologics) may be necessary in more severe or refractory disease [23][44].

Conclusion

Psoriasis is a multifaceted autoimmune disease with a substantial global burden. Advances in pathophysiologic understanding have enabled targeted therapies spanning topicals, phototherapy, systemics, and biologics. Nonetheless, challenges persist in achieving durable remission and managing comorbidities (e.g., cardiometabolic and mental health conditions). Future progress hinges on personalized treatment, novel therapeutics, and holistic care frameworks that address both physical and psychosocial needs. With sustained innovation and comprehensive patient support, the overall burden of psoriasis can be meaningfully reduced.

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