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Systemic Inflammation in Psoriasis: From Cutaneous Disease to Multiorgan Involvement

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Abstract

Psoriasis has evolved from being classified as a localized skin condition to being recognized as a chronic, immune-mediated systemic inflammatory disorder. This transition is primarily driven by the IL-23/Th17 axis, which facilitates "cytokine spillover" from the skin into the systemic circulation. This review synthesizes current evidence on the immunopathogenesis linking cutaneous inflammation to multiorgan involvement, a process often referred to as the "psoriatic march." We explore the roles of key mediators such as TNF-α, IL-17, and IL-6 in promoting endothelial dysfunction, insulin resistance, and subclinical systemic inflammation. Furthermore, we analyze the clinical utility of classical biomarkers (CRP, hs-CRP, ESR) and emerging hematologic indices (NLR, PLR) in assessing disease severity and cardiometabolic risk.

The article details the extensive multiorgan impact of psoriasis, including cardiovascular disease, metabolic syndrome, non-alcoholic fatty liver disease (NAFLD), psoriatic arthritis, and renal and neuropsychological morbidity. Finally, we evaluate the impact of conventional and biologic systemic therapies on reducing inflammatory markers and potentially attenuating cardiovascular risk. Given the complexity of the psoriatic march, this review emphasizes the critical need for a multidisciplinary approach involving dermatologists and internal medicine physicians. Early screening for comorbidities and effective systemic control of inflammation is essential to mitigate long-term complications and improve the quality of life for patients with moderate-to-severe psoriasis.

Keywords: Psoriasis; Systemic Inflammation; IL-23/Th17 Axis; Psoriatic March; Interleukin

Abbreviations: IL-23: Interleukin-23; Th17: T helper 17 cells; HLA-Cw6: Human Leukocyte Antigen Cw6; IL-17A: Interleukin-17a; IL-17F: Interleukin-17F; IL-22: Interleukin-22 ; NAFLD: Non-Alcoholic Fatty Liver Disease; CRP: C-Reactive Protein; IL-6: Interleukin-6; TNF-α: Tumor Necrosis Factor-Alpha; IFN-γ: Interferon-Gamma; PASI: Psoriasis Area And Severity Index; hs-CRP: High-Sensitivity C-Reactive Protein; ESR: Erythrocyte Sedimentation Rate; NF-κB: Nuclear Factor Kappa-Light-Chain-Enhancer Of Activated B Cells; NLR: Neutrophil-To-Lymphocyte Ratio; PLR: Platelet-To-Lymphocyte Ratio; MLR: Monocyte-To-Lymphocyte Ratio; BSA: Body Surface Area; TGF-β: Transforming Growth Factor-Beta; Bact DNA: Bacterial DNA; PGA: Physician's Global Assessment; HOMA-IR: Homeostatic Model Assessment For Insulin Resistance; BMI: Body Mass Index; NASH: Non-Alcoholic Steatohepatitis; PsA: Psoriatic Arthritis; RANKL: Receptor Activator Of Nuclear Factor Kappa-B Ligand; CKD: Chronic Kidney Disease; HR: Hazard Ratio; ESRD: End-Stage Renal Disease; EGFR: Estimated Glomerular Filtration Rate; FDG PET/CT: Fluorodeoxyglucose Positron Emission Tomography/Computed Tomography; MACE: Major Adverse Cardiovascular Events

Introduction

Psoriasis is no longer viewed as a disease confined to the skin; it is a chronic, immune-mediated systemic inflammatory condition driven primarily by the IL-23/Th17 axis [1,2]. Its development stems from a complex interplay between genetic susceptibility-most notably the HLA-Cw6 allele-and environmental triggers like infections, physical traumas, and certain medications [1,2]. These factors initiate immune dysregulation where aberrant dendritic cells secrete IL-23, promoting the expansion of Th17 cells. These cells release IL-17A, IL-17F, and IL-22, cytokines that act directly on keratinocytes to induce hyperproliferation and sustain inflammation [1]. This paradigm shift urges clinicians to adopt a comprehensive management approach that extends beyond the skin. The disease affects approximately 2–3% of the global population, with prevalence varying by geography, ethnicity, and environmental factors [4-6].

Rising obesity rates and aging populations are driving the global burden upward [3]. Beyond its physical manifestations, psoriasis carries a heavy psychosocial and economic weight; quality of life is often impaired to a degree comparable to that seen in diabetes or cardiovascular disease [5]. Furthermore, access to care remains inequitable, especially in resource-limited settings and among patients with skin of color, leading to significant under recognition [3]. Evidence from the past two decades confirms that psoriasis is a systemic disorder with multisystem involvement, including strong associations with psoriatic arthritis, metabolic syndrome, insulin resistance, and NAFLD [2,7]. Disease severity correlates directly with systemic inflammation; patients with moderate-to-severe psoriasis show elevated circulating levels of CRP, IL-6, and TNF-α, proving that cutaneous findings are but one manifestation of a broader process [1,3].

This progression is described as the "psoriatic march" [8]. Persistent immune activation causes inflammatory mediators to spill into the bloodstream, driving widespread endothelial dysfunction and vascular inflammation [1,8]. This environment facilitates the progression of atherosclerotic plaques, placing severe psoriasis patients at a cardiovascular risk comparable to those with type 2 diabetes [3,8]. Consequently, effective systemic treatment with biologic agents is now being studied as a way to attenuate this risk by reducing the overall inflammatory burden [7]. The purpose of this review article is to synthesize current evidence regarding the transition of psoriasis from a localized lesion to a multisystemic pathology, emphasizing the biological mechanisms involved. By highlighting these connections, we aim to provide a comprehensive framework that supports early intervention and multidisciplinary management to mitigate long-term inflammatory complications.

Immunopathogenesis of Systemic Inflammation in Psoriasis

Psoriasis is characterized by dysregulated interactions between innate and adaptive immune systems. Although it primarily manifests as a skin disorder, evidence demonstrates that psoriasis is a systemic inflammatory condition driven by complex immune pathways involving cytokines, immune cells, and chronic low-grade inflammation [9,10].

Key Immune Pathways

IL-23 / Th17 Axis

The IL-23/Th17 pathway plays a central role in pathogenesis. Dendritic cells produce IL-23, which promotes the differentiation and survival of T helper 17 (Th17) cells. These release pro-inflammatory cytokines, including IL-17A, IL-17F, and IL-22, stimulating keratinocyte proliferation and recruitment of immune cells. IL-17 also promotes neutrophil migration and amplifies inflammatory signaling, contributing to both local and systemic inflammation [1,3,11].

Role of TNF-α

TNF-α is a key cytokine that enhances immune cell activation, promotes endothelial dysfunction, and stimulates the production of additional inflammatory mediators. It increases vascular permeability and leukocyte migration, contributing to chronic inflammation within the skin and systemic circulation [4,12].

IL-6 and Systemic Inflammation

IL-6 is an important mediator of the acute-phase response, stimulating hepatic production of CRP and promoting immune cell activation. Elevated IL-6 levels are associated with insulin resistance and increased cardiovascular risk [5,13].

Cellular Components

Dendritic cells

Dendritic cells are essential initiators of the immune response in psoriasis. They detect environmental triggers such as infection, trauma, or stress and release cytokines including IL-23 and TNF-α. These cytokines activate T lymphocytes and initiate the inflammatory cascade that leads to psoriatic plaque formation [1].

T Lymphocytes (Th1, Th17)

T lymphocytes, particularly Th1 and Th17 cells, play a central role in the immune response in psoriasis. Th17 cells produce IL-17 and IL-22, which stimulate keratinocyte proliferation and inflammation. Th1 cells produce interferon-gamma (IFN-γ), which enhances immune activation and promotes inflammatory responses [3]. These immune cells circulate in the bloodstream and contribute to systemic inflammation, linking skin disease with multiorgan involvement, including joints, blood vessels, and metabolic tissues [3].

Neutrophils and Keratinocytes

Neutrophils are frequently found in psoriatic lesions and contribute to inflammation by releasing reactive oxygen species, enzymes, and cytokines. They form characteristic structures known as Munro microabscesses within the epidermis, a histological

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hallmark of psoriasis [2]. Keratinocytes are not only structural skin cells but also active participants in immune responses. They produce inflammatory mediators such as cytokines, chemokines, and antimicrobial peptides that amplify inflammation and recruit additional immune cells. This interaction between immune cells and keratinocytes creates a self-sustaining inflammatory cycle [1].

Cytokine Spillover into Systemic Circulation

Mechanism of Transition from Skin-Localized to Systemic Inflammation

In psoriasis, inflammatory cytokines produced in the skin enter the systemic circulation, leading to widespread immune activation. Cytokines such as TNF-α, IL-17, and IL-6 can circulate in the bloodstream and affect distant organs, including the cardiovascular system, liver, joints, and adipose tissue. This process is known as cytokine spillover and represents the key mechanism by which psoriasis evolves from a localized skin condition into a systemic inflammatory disease [2,6,14]. Endothelial cells exposed to circulating cytokines develop dysfunction, which promotes atherosclerosis and increases the risk of cardiovascular disease. Similarly, systemic inflammation contributes to insulin resistance and metabolic syndrome, common comorbidities in psoriasis patients [6,14].

Chronic Low-Grade Inflammation Model

Psoriasis is characterized by persistent low-grade inflammation that continues even in the absence of visible skin lesions. This chronic inflammatory state results from continuous activation of immune cells and sustained production of inflammatory cytokines. Over time, this systemic inflammation contributes to the development of multiple comorbidities, including cardiovascular disease, obesity, diabetes, and psoriatic arthritis [2]. The chronic low-grade inflammation model explains why psoriasis is associated with increased morbidity and mortality and highlights the importance of early diagnosis and systemic treatment to reduce long-term complications [10].

Circulating Inflammatory Biomarkers in Psoriasis

Classical Biomarkers

CRP

C-reactive protein (CRP) is a hepatic acute-phase reactant induced primarily by interleukin-6 and is widely used as a marker of systemic inflammation. In psoriasis, CRP levels are consistently elevated, supporting the recognition of psoriasis as a chronic systemic inflammatory disorder rather than a disease confined to the skin. Recent evidence from biomarker-focused reviews indicates that CRP correlates with disease severity indices such as the Psoriasis Area and Severity Index (PASI) and may decrease following effective systemic or biologic therapy, highlighting its utility as a dynamic marker of treatment response [15]. However, its sensitivity remains limited; a significant proportion of patients with active psoriatic disease exhibit normal CRP levels, particularly in psoriatic arthritis, where elevated CRP is observed in fewer than half of cases. This variability underscores the heterogeneity of systemic inflammation in psoriasis and limits the use of CRP as a standalone biomarker [16].

HS-CRP

High-sensitivity CRP (hs-CRP) enables detection of low-grade systemic inflammation and has gained importance in psoriasis due to its association with cardiometabolic comorbidities. Psoriasis is increasingly linked with a heightened risk of cardiovascular disease, driven in part by persistent subclinical inflammation. Elevated hs-CRP levels in psoriatic patients reflect this inflammatory burden and are associated with endothelial dysfunction, atherosclerosis, and metabolic syndrome. Contemporary mechanistic and translational reviews emphasize that systemic inflammation in psoriasis extends beyond the skin, involving cytokine-driven pathways that contribute to cardiometabolic risk, thereby supporting hs-CRP as a clinically relevant biomarker for risk stratification [17]. Furthermore, hs-CRP may provide greater sensitivity than standard CRP assays in detecting subtle inflammatory changes, particularly in patients with mild disease or those receiving treatment [17].

ESR

The erythrocyte sedimentation rate (ESR) is a nonspecific marker of inflammation that reflects increased plasma proteins, particularly fibrinogen, which promote erythrocyte aggregation. In psoriasis, ESR is often elevated in moderate-to-severe disease and may parallel systemic inflammatory burden. However, like CRP, ESR demonstrates considerable variability and limited specificity, as it is influenced by multiple non-inflammatory factors including age, anemia, and comorbid conditions. Recent reviews of inflammatory biomarkers in psoriasis emphasize that ESR lacks sufficient sensitivity and specificity to serve as a reliable standalone indicator of disease activity and is best interpreted alongside other biomarkers such as CRP or cytokine profiles [15]. Consequently, ESR is typically used as an adjunctive measure rather than a primary biomarker in both clinical practice and research settings [15].

Cytokines and Molecular Markers

IL-6

Interleukin-6 (IL-6) is a multifunctional cytokine involved in acute and chronic inflammation and plays a significant role in the systemic inflammatory milieu of psoriasis. IL-6 is elevated in psoriatic patients compared with healthy individuals and reflects both cutaneous and systemic inflammatory burden. It promotes differentiation of naïve T cells into Th17 cells, thereby linking upstream innate immune activation to the IL-23/IL-17 axis, which is central to psoriasis pathogenesis. Recent evidence highlights that IL-6 is not only a marker of disease activity but also contributes to cardiometabolic comorbidities such as atherosclerosis and metabolic syndrome in psoriasis patients. However, IL-6 levels are

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influenced by obesity and other comorbid conditions, limiting its specificity as a standalone biomarker [18,19].

IL-17

Interleukin-17A (IL-17A) is a key effector cytokine in psoriasis and a central driver of keratinocyte activation, neutrophil recruitment, and chronic inflammation. Circulating IL-17 levels are elevated in psoriasis and correlate with disease severity, reinforcing its role as both a pathogenic mediator and biomarker of inflammatory activity. IL-17 acts downstream of IL-23 and amplifies inflammatory cascades by inducing cytokines, chemokines, and antimicrobial peptides in keratinocytes. Clinical success of IL-17 inhibitors further confirms its central role in disease pathophysiology, with treatment-associated reductions in systemic inflammatory markers supporting its use as a dynamic biomarker of therapeutic response [20-22].

IL-23

Interleukin-23 (IL-23) is an upstream regulatory cytokine that sustains Th17 cell survival and promotes chronic inflammation in psoriasis. Elevated IL-23 levels have been observed in psoriatic patients and are associated with activation of the IL-23/IL-17 axis. IL-23 is primarily produced by dendritic cells and macrophages and plays a key role in maintaining inflammatory persistence. The therapeutic success of IL-23–targeting biologics further confirms its central role in psoriasis pathophysiology and validates the IL-23/IL-17 axis as a core disease-driving pathway in plaque psoriasis [23,24].

TNF-α

Tumor necrosis factor-alpha (TNF-α) is a central pro-inflammatory cytokine involved in both innate and adaptive immune responses and represents one of the earliest identified therapeutic targets in psoriasis. TNF-α is produced by activated dendritic cells, macrophages, T cells, and keratinocytes within psoriatic lesions and contributes to amplification of inflammatory signaling through activation of NF-κB and promotion of downstream cytokines, including IL-23 and IL-17. Elevated TNF-α expression has been demonstrated in lesional skin and, to a lesser extent, in the circulation of patients with moderate-to-severe psoriasis, supporting its role as a systemic inflammatory mediator rather than a purely cutaneous factor [25,26].

Hematologic Indices

Recent clinical research has focused on cost-effective, readily available hematologic markers derived from complete blood counts to assess the systemic inflammatory burden in psoriasis. Among these, the Neutrophil-to-lymphocyte ratio (NLR) has emerged as a significant indicator; it reflects the balance between the innate immune response (neutrophils) and the adaptive regulatory response (lymphocytes) [1,15]. Elevated NLR levels are consistently observed in patients with psoriasis compared to health controls, signaling a pro-inflammatory state that often mirrors the intensity of systemic involvement.

Similarly, the Platelet-to-lymphocyte ratio (PLR) serves as a marker of systemic inflammation and has been linked to increased pro-thrombotic risk and vascular damage in psoriatic patients [15,22]. Furthermore, monocyte-related indices, such as the monocyte-to-lymphocyte ratio (MLR), are increasingly recognized for their role in reflecting the activation of the monocytic lineage, which is crucial for the secretion of pro-inflammatory cytokines like TNF-α and IL-23. These indices provide a broader snapshot of the systemic "spillover" of inflammation from the skin into the hematopoietic system, offering a practical tool for monitoring patients in clinical settings where advanced cytokine profiling is not feasible [15-22].

Correlation with Disease Severity

The clinical utility of these inflammatory biomarkers is often measured by their correlation with the Psoriasis Area and Severity Index (PASI) score. Studies indicate that levels of CRP, IL-17, and hematologic indices like NLR and PLR demonstrate a positive correlation with PASI scores, meaning that as the cutaneous involvement and physical severity of the disease increase, so does the detectable systemic inflammatory burden [15,22-26]. This association reinforces the concept that skin is a primary source of systemic mediators; however, the correlation is not always linear across all patient phenotypes. Despite their promise, several limitations and sources of variability must be considered when interpreting these markers.

A significant challenge is the influence of comorbidities, particularly obesity and metabolic syndrome, which can independently elevate levels of CRP and IL-6, potentially confounding the assessment of psoriasis-specific inflammation [15]. Additionally, there is considerable inter-individual variability; a subset of patients with high PASI scores may present with relatively normal systemic biomarkers, while others with mild skin disease may show significant subclinical systemic activation [17]. This lack of standardized cut-off values and the inherent fluctuations in inflammatory markers limit their current use as standalone diagnostic tools, highlighting the need for a multi-marker approach in clinical practice [16,18].

Psoriasis as a Systemic Disease: Multiorgan Involvement

Cardiovascular System

Psoriasis is increasingly recognized as a chronic immune-mediated disease with systemic implications that extend beyond the skin, driven by persistent inflammation involving key cytokines such as IL-17, TNF-α, and IL-6 [27,28]. This sustained inflammatory state contributes to the involvement of multiple organ systems, particularly the cardiovascular and metabolic

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systems, establishing psoriasis as a multisystem disorder rather than a purely cutaneous condition [29]. Within the cardiovascular system, chronic inflammation promotes endothelial dysfunction, which represents an early and critical step in the development of atherosclerosis [29,30]. This altered vascular environment facilitates plaque formation through immune cell infiltration and lipid accumulation within the arterial wall, ultimately increasing the risk of major adverse cardiovascular events [30].

Epidemiological studies have consistently shown that patients with psoriasis have a higher risk of myocardial infarction and stroke compared to the general population, with reported increases in cardiovascular risk ranging from approximately 20% to 50%, particularly in moderate-to-severe disease [31]. This association is largely mediated by systemic inflammation, as evidenced by elevated levels of biomarkers such as C-reactive protein (CRP) and interleukin-6 (IL-6), which contribute directly to vascular damage and plaque instability [32]. Collectively, these findings highlight endothelial dysfunction and inflammation-driven atherogenesis as central mechanisms underlying cardiovascular comorbidity in psoriasis. However, although these associations are well established, the precise causal pathways linking psoriasis to cardiovascular disease remain incompletely understood and are likely influenced by a combination of genetic, environmental, and lifestyle factors.

Metabolic System

In addition to cardiovascular involvement, psoriasis is closely associated with metabolic dysfunction, particularly insulin resistance and the development of metabolic syndrome [33]. Chronic inflammation interferes with insulin signaling pathways, leading to impaired glucose metabolism and increased cardiometabolic risk [33,34]. Obesity further amplifies this inflammatory state, as adipose tissue functions as an active endocrine organ that secretes pro-inflammatory mediators, thereby exacerbating both psoriasis severity and systemic metabolic abnormalities [34]. Adipokines play a central role in linking psoriasis with metabolic disease.

Leptin, which is often elevated in patients with psoriasis, exerts pro-inflammatory effects by promoting cytokine production and endothelial dysfunction, contributing to a pro-atherogenic environment [35]. In contrast, adiponectin, an anti-inflammatory adipokine with insulin-sensitizing properties, is typically reduced in psoriasis, further enhancing metabolic dysregulation and cardiovascular risk [36]. Together, these alterations underscore the complex interplay between chronic inflammation, adipose tissue biology, and metabolic imbalance in psoriasis. Nevertheless, the directionality and causality of these associations remain subjects of ongoing investigation, and it is likely that bidirectional interactions between psoriasis and metabolic disease contribute to their coexistence.

Hepatic Involvement

Non-alcoholic fatty liver disease (NAFLD) affects 42.3% to 65% of psoriatic patients, and their risk is approximately 1.5–3 times higher than the general population [37-39]. The liver is a common extracutaneous target. Crucially, this correlation holds regardless of conventional metabolic risk factors like obesity and metabolic syndrome, indicating that hepatic damage is directly caused by chronic systemic inflammation in addition to its cardiometabolic comorbidities [37-39]. Mechanistically, the so-called hepato-dermal axis is driven by cutaneous-derived pro-inflammatory mediators - including TNF-α, IL-6, IL-17, and TGF-β - which promote insulin resistance, induce hepatic stellate cell activation, and stimulate collagen deposition and fibrogenesis [37,39].

This inflammatory milieu is further amplified by increased intestinal permeability and non-viable bacterial translocation, evidenced by the detection of bacterial DNA (bactDNA) in peripheral blood, which was significantly more prevalent in psoriatic patients with NAFLD than in those without (29.7% vs. 13.7%) [37]. Clinically, NAFLD severity correlates with disease burden as reflected by higher psoriasis severity index (PASI), body surface area (BSA), and physicians' global assessment scores (PGA) scores, while independent predictors of NAFLD include insulin resistance (HOMA-IR ≥2.15), male sex, BMI ≥30 kg/m^2, and elevated transaminases [37].

The prevalence of biopsy-confirmed NASH varies considerably - from approximately 1% in community cohorts under systemic treatment to 22% in tertiary referral centers - likely reflecting both patient selection and the modulatory effect of immunosuppressive therapy on hepatic inflammation [37,39]. Proactive hepatic screening is necessary, especially in individuals with early-onset or severe illness, because of the likelihood of development toward cirrhosis and hepatocellular cancer, even if extensive fibrosis is still uncommon [37-39]. The choice and monitoring of systemic psoriasis treatments, especially those with proven hepatotoxic potential, are directly impacted by these pathophysiological connections [40].

Musculoskeletal System

Peripheral arthritis, enthesitis, spondylitis, and dactylitis are among the diverse clinical domains that make up psoriatic arthritis (PsA), which is now understood as a systemic manifestation of "psoriatic illness" and necessitates interdisciplinary rheumatologic and dermatologic collaborative management [41-44]. Its pathogenesis is driven by a convergent cytokine network in which IL-23 initiates Th17 differentiation, leading to synovio-entheseal inflammation sustained by IL-17A and TNF-α [41,42].

This dual cytokine axis produces a hallmark feature of PsA: the concurrent occurrence of osteoclast-mediated bone erosion and

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osteoblast-driven new bone formation, where TNF-α promotes RANKL-dependent osteoclastogenesis and angiogenesis, while IL-17A recruits neutrophils and activates stromal cells [41,42,44]. The systemic burden is substantial-approximately 42% of patients carry three or more comorbidities, including cardiovascular disease, and obesity exhibits a bidirectional relationship with disease risk and activity [44]. The main purpose of early diagnosis and treatment is to preserve physical function and productivity at work [42,44].

Other Systems

Psoriasis is a multisystemic inflammatory disorder with a severity-dependent risk of renal and neuropsychological morbidity. Meta-analyses establish psoriasis as an independent risk factor for chronic kidney disease (CKD; pooled HR 1.53–1.65) and end-stage renal disease (ESRD; pooled HR 1.24–1.37), with severe disease dramatically elevating these risks (CKD HR 1.91; ESRD HR 2.72) [45,46]. Pathophysiology involves shared pleiotropic cytokines, where TNF-α induces NaCl retention and hypertension, while the Th17 axis mediates macrophage-driven tissue damage and eGFR decline [45,47,48].

This inflammatory milieu further bridges the renal system with neuropsychological distress. Elevated systemic cytokines are thought to influence brain function and mood regulation, where avoidance-oriented coping generates chronic daily stress that bidirectionally fuels the Th1/Th17 cascade, increasing rates of depression and anxiety [45,49,50]. Consequently, there is a clinical mandate for routine screening of blood urea nitrogen, serum creatinine, and microalbuminuria in moderate-to-severe cases to mitigate the substantial morbidity and premature mortality associated with these complex comorbid states [45,46,47].

The "Psoriatic March" Concept

The "psoriatic march" refers to a proposed stepwise pathogenic continuum where psoriasis acts as a systemic inflammatory disorder contributing to cardiometabolic and cardiovascular complications [51]. This model suggests that persistent immune activation in psoriatic skin, driven by TNF-α and the IL-23/IL-17 axis, extends into the systemic circulation to promote chronic low-grade inflammation. Within this environment, metabolic pathways become altered, favoring insulin resistance, which amplifies oxidative stress and reinforces a cycle of systemic dysfunction [52]. As this process advances, the sustained inflammatory burden leads to endothelial dysfunction, characterized by impaired nitric oxide availability and increased vascular permeability.

These changes create a prothrombotic vascular milieu that facilitates atherosclerosis, including lipid deposition and plaque formation [52,53]. Over time, these alterations may translate into clinically overt diseases, such as coronary artery disease or cerebrovascular events [53]. Despite its conceptual strength, the psoriatic march is a mechanistic model rather than a universally proven clinical sequence. While evidence links psoriasis to systemic inflammation, direct longitudinal data demonstrating a uniform progression to atherosclerosis remain limited [54-56]. Clinically, however, this framework justifies a proactive approach. Recognizing psoriasis as a systemic condition supports early screening for metabolic syndrome and cardiovascular risk factors, as timely control of inflammation through targeted biologic therapies may help interrupt this pathogenic cascade and reduce long-term morbidity [57].

Impact of Systemic Therapies on Inflammation

Conventional Systemic Treatments

Psoriasis management is determined by symptom severity, as effective treatment significantly reduces the risk of future comorbidities and joint complications [58]. Patients with mild disease (Body Surface Area [BSA] < 3%) are typically managed with intermittent topical agents, such as corticosteroids. While potent to high-potency corticosteroids demonstrate superior efficacy, their use is limited by adverse effects-including skin atrophy, folliculitis, and tachyphylaxis-when applied for more than 4 weeks [59]. To maintain remission and lower cumulative steroid doses, these are often combined with retinoids (tazarotene), vitamin D analogs (calcipotriene), or calcineurin inhibitors (tacrolimus) [59].

Topical therapy is insufficient when the disease involves joint manifestations or reaches a moderate-to-severe index (scores >10 in erythema, scaling, and induration). In such cases, systemic therapy and a multidisciplinary team including dermatologists and rheumatologists are required [58,59]. The first-line therapy for moderate-to-severe psoriasis is the non-biological agent Methotrexate. As a folate antagonist, it interferes with nucleic acid synthesis, showing symptomatic improvement in 60% of patients through immunosuppressive effects and decreased lymphoid cell proliferation [58,60].

While effective, it requires rigorous monitoring of liver, kidney, and blood counts, particularly because concomitant use of antibiotics, alcohol, or diuretics increases hepatotoxicity risk [58,60]. Additional conventional options include Cyclosporine, a calcineurin inhibitor primarily used as rescue treatment due to long-term risks of hypertension and renal failure, and narrowband UVB phototherapy (311-312 nm) [58]. For cases specifically involving psoriatic arthritis, oral phosphodiesterase-4 inhibitors like apremilast may be utilized [58].

Biologic Therapies

There are some new biological options with very good efficacy but very costly, those are strictly used in severe cases or failure of previous 2 trials of 6/week of systemic therapies, they have extra improvement of anxiety and depression. Tumor necrosis factor alpha inhibitors (infliximab, etarnecep) are useful in inpatient

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pustular psoriasis, but they lose efficacy over 2 or 3 years, also contraindicated in active tuberculosis or worsen heart failure.

Anti-interleukin therapies show a strong profile of efficacy and persistence over time, anti-IL-17 (bimekizumab), anti-IL-12 (ustekinumab), anti-IL-23 (guselkumab). Anti-IL-17 is better in psoriatic arthritis, but worsen inflammatory bowel disease, so high doses of anti-IL-12, anti-IL-23 are preferred for inflammatory bowel disease. Oral therapies available like tyrosine kinase 2 inhibitor (deucravacitinib) with a good safe profile (increase lipid, triglyceride levels, small increase of acne and herpes reactivation). Apremilast, they [63].

Effects on Systemic Inflammation

The clinical objective of modern psoriasis management has shifted from achieving clear skin to the comprehensive suppression of the systemic inflammatory milieu. Effective systemic intervention-particularly the use of biologic agents targeting the IL-17 and IL-23 pathways-has demonstrated a profound impact on circulating inflammatory markers.

Reduction in CRP and Cytokines

Successful treatment of moderate-to-severe psoriasis is consistently associated with a significant decrease in C-reactive protein (CRP) and high-sensitivity CRP (hs-CRP) levels [1,8]. Because CRP production in the liver is primarily driven by circulating IL-6, the reduction of these markers indicates a successful interruption of the "cytokine spillover" from psoriatic skin into the systemic circulation. Furthermore, targeted therapies have been shown to normalize the levels of various pro-inflammatory cytokines, including TNF-α, IL-17A, and IL-6, thereby dampening the chronic low-grade inflammatory state that characterizes the disease [22,26,58]. This biochemical improvement often precedes or parallels clinical skin clearance, serving as a dynamic indicator of therapeutic success and systemic stabilization [58].

Potential Cardiovascular Risk Reduction

Perhaps the most significant clinical implication of systemic therapy is the potential for cardiovascular risk reduction. By suppressing chronic inflammation, systemic treatments may slow or even partially reverse the "psoriatic march." Emerging evidence suggests that biologic therapies, specifically TNF-α and IL-17 inhibitors, can improve endothelial function and reduce vascular inflammation as measured by advanced imaging techniques like FDG PET/CT [27,58,62].

These treatments are thought to stabilize atherosclerotic plaques by reducing the recruitment of immune cells to the vascular wall and improving the bioavailability of nitric oxide [58]. While large-scale, long-term prospective trials are still ongoing to definitively quantify the reduction in major adverse cardiovascular events (MACE), retrospective cohort studies indicate that patients receiving consistent systemic treatment have a lower risk of myocardial infarction compared to those receiving only topical therapy or no treatment at all [27,58]. This underscores the critical importance of early and effective systemic control not just for dermatologic health, but for long-term cardiovascular survival.

Clinical Implications for Internal Medicine and Dermatology

Given the systemic inflammatory nature of psoriasis, management must extend beyond dermatological care to incorporate a multidisciplinary approach. Psoriasis is recognized as a chronic immune-mediated disease with multisystem involvement and an increased risk of cardiovascular, metabolic, and hepatic comorbidities, all of which contribute to elevated long-term complications and mortality [58,61]. Consequently, effective care requires close collaboration between dermatologists and internal medicine physicians for the comprehensive management of both cutaneous and systemic manifestations [58]. Screening for comorbid conditions is essential to optimize care and prevent major complications. Clinicians should actively assess cardiovascular, metabolic, and hepatic diseases.

Current guidelines emphasize early identification of cardiovascular risk factors, including hypertension, hyperlipidemia, diabetes, and obesity [60]. Because patients are at a heightened risk for atherosclerotic cardiovascular disease, regular cardiovascular risk assessment is a clinical necessity [59,62]. Psoriasis is also associated with insulin resistance and a significantly increased risk of developing type 2 diabetes mellitus; therefore, metabolic screening, including fasting glucose levels, should be routinely performed [63]. Furthermore, given the high prevalence of non-alcoholic fatty liver disease (NAFLD), clinicians should evaluate steatohepatitis and liver enzymes. Notably, liver disease may be present even with normal laboratory investigations, suggesting that imaging, such as liver ultrasound, should be considered in specific clinical cases [61].

Biomarkers are playing an increasing role in risk stratification and monitoring systemic inflammation. Traditional markers, such as C-reactive protein (CRP) and specific cytokines, correlate with disease severity and increased cardiovascular risk [64]. Additionally, hematologic and biochemical markers provide insight into the overall inflammatory burden and the risk of metabolic comorbidities [64]. While their routine use is not yet fully standardized, these markers guide individualized management and identify high-risk patients. Early identification of these comorbidities, paired with a multidisciplinary approach, significantly improves long-term clinical outcomes and the quality of life for patients with psoriasis.

Conclusion

Psoriasis is a profound systemic inflammatory disease that extends far beyond the basement membrane of the skin. The

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evidence presented in this review confirms that the "psoriatic march" is a biologically plausible sequence where persistent cutaneous immune activation leads to systemic vascular injury and metabolic dysregulation. The central role of the IL-23/Th17 axis and the subsequent cytokine spillover provide a mechanistic link to high-risk comorbidities such as myocardial infarction, stroke, and NAFLD. The integration of cost-effective hematologic indices alongside traditional biomarkers represents a significant advancement in monitoring the systemic inflammatory burden.

Furthermore, the success of targeted biologic therapies in normalizing these markers suggests that early, aggressive intervention can potentially modify the course of systemic disease and reduce cardiovascular mortality. Ultimately, the management of psoriasis requires a shift toward a multidisciplinary model where dermatologists and internists collaborate to screen for, and treat, the invisible systemic manifestations of this condition. Stopping the psoriatic march necessitates not only clear skin but the comprehensive suppression of systemic inflammation.

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