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Review Article

Psoriasis and increased cardiovascular risk: cutaneous inflammation as a systemic marker-the role of IL-17 and endothelial dysfunction

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ABSTRACT

Psoriasis, a chronic immune-mediated inflammatory dermatosis, has been increasingly recognized as a systemic condition associated with elevated cardiovascular risk. Emerging evidence suggests that the persistent inflammatory state in psoriasis, driven by key cytokines such as interleukin-17 (IL-17), contributes to endothelial dysfunction and accelerated atherosclerosis, thereby predisposing affected individuals to major adverse cardiovascular events. This article explores the pathophysiological mechanisms linking cutaneous inflammation to systemic vascular impairment, with a particular focus on the role of IL-17 in promoting endothelial activation, leukocyte recruitment and plaque instability. Furthermore, we discuss the clinical implications of psoriasis as an independent risk factor for cardiovascular disease, emphasizing the importance of multidisciplinary management strategies to mitigate long-term cardiovascular morbidity in this patient population.

Keywords: Atherosclerosis, Cytokine signalling, Cardiovascular risk, Endothelial dysfunction, Immunomodulation, Interleukin-17

INTRODUCTION

Psoriasis is a chronic, immune-mediated inflammatory disorder characterized by hyperproliferative keratinocytes and immune cell infiltration, resulting in well-demarcated erythematous plaques with silvery scales. Beyond its dermatological manifestations, psoriasis is now understood to be a systemic disease with significant associations to cardiovascular comorbidities, including coronary artery disease, stroke and peripheral vascular disease. The underlying pathophysiology involves a complex interplay between innate and adaptive immunity, with T-helper 17 (Th17) cells and their signature cytokine, IL-17, playing a central role in sustaining both cutaneous and systemic inflammation.[1,2] The persistent release of pro-inflammatory cytokines in psoriasis contributes to endothelial dysfunction, a critical early event in atherogenesis. IL-17, in particular, has been implicated in promoting vascular inflammation by upregulating adhesion molecules, enhancing leukocyte-endothelial interactions and impairing nitric oxide-mediated vasodilation. These mechanisms collectively foster a prothrombotic and proatherogenic milieu, increasing the risk of cardiovascular events. Additionally, the chronic inflammatory state in psoriasis may exacerbate traditional cardiovascular risk factors such as hypertension, dyslipidemia and insulin resistance, further compounding cardiovascular risk.[2,3]

Given the growing recognition of psoriasis as a systemic inflammatory condition with profound cardiovascular implications, this article aims to elucidate the mechanistic

links between cutaneous inflammation and endothelial dysfunction, with a focus on IL-17-driven pathways. By integrating current evidence from molecular, clinical and epidemiological studies, we highlight the importance of early cardiovascular risk assessment and targeted therapeutic interventions in psoriatic patients to reduce long-term morbidity and mortality.[4]

PATHOPHYSIOLOGY

Psoriasis, a chronic, immune-mediated inflammatory dermatosis, is driven by a complex interplay of genetic predisposition, environmental triggers and dysregulated immune responses. The disease is characterized by aberrant keratinocyte proliferation and a pathological inflammatory cascade predominantly mediated by T-helper 17 (Th17) cells, dendritic cells and an array of pro-inflammatory cytokines, most notably interleukin-17 (IL-17), interleukin-23 (IL-23) and tumor necrosis factor-alpha (TNF-α). While the clinical hallmark of psoriasis is the development of erythematous, scaly plaques on the skin, the systemic nature of the disease extends beyond the integumentary system, contributing to a heightened risk of cardiovascular disease (CVD). The underlying mechanisms linking psoriasis to accelerated atherosclerosis and endothelial dysfunction involve chronic low-grade inflammation, cytokine-mediated vascular injury and metabolic perturbations that collectively foster a proatherogenic milieu.[4]

Central to the pathophysiology of psoriasis is the IL-23/IL-17 axis, which perpetuates both cutaneous and systemic inflammation. Antigen-presenting cells, including dendritic cells, secrete IL-23, which in turn activates Th17 cells to produce IL-17A, IL-17F and IL-22. These cytokines stimulate keratinocyte hyperproliferation and further amplify inflammation by recruiting neutrophils and other immune cells to the skin. However, the effects of IL-17 are not confined to the epidermis; circulating IL-17 exerts systemic effects by promoting endothelial dysfunction, a critical early event in atherogenesis. IL-17 upregulates the expression of adhesion molecules such as vascular cell adhesion molecule-1 (VCAM-1) and intercellular adhesion molecule-1 (ICAM-1) on endothelial cells, facilitating leukocyte adhesion and transmigration into the vascular wall. Additionally, IL-17 enhances the production of reactive oxygen species (ROS), which impair endothelial nitric oxide synthase (eNOS) activity, reducing the bioavailability of nitric oxide (NO), a key mediator of vasodilation and vascular homeostasis.[4,5]

The chronic inflammatory state in psoriasis also contributes to endothelial dysfunction through indirect mechanisms. Elevated levels of C-reactive protein (CRP), fibrinogen and other acute-phase reactants reflect systemic inflammation and correlate with increased cardiovascular risk. Furthermore, the persistent activation of innate and adaptive immune responses in psoriasis leads to oxidative stress, which promotes lipid peroxidation and the formation of oxidized low-density lipoprotein (oxLDL), a critical factor in the initiation and progression of atherosclerotic plaques. The infiltration of inflammatory cells into the vascular intima, coupled with smooth muscle cell proliferation, results in plaque destabilization and an increased likelihood of thrombotic events, such as myocardial infarction and stroke.[5]

Beyond direct vascular injury, psoriasis-associated inflammation exacerbates traditional cardiovascular risk factors, including insulin resistance, dyslipidemia and hypertension. Th17 cytokines, particularly IL-17, interfere with insulin signaling pathways, contributing to metabolic syndrome and type 2 diabetes mellitus. Dyslipidemia in psoriatic patients is characterized by elevated triglycerides, reduced high-density lipoprotein (HDL) cholesterol and an increase in small, dense LDL particles, all of which are proatherogenic. Hypertension, another common comorbidity in psoriasis, is exacerbated by endothelial dysfunction, increased arterial stiffness and elevated levels of endothelin-1, a potent vasoconstrictor.[5]

The pathophysiological link between psoriasis and increased cardiovascular risk is multifactorial, involving IL-17-driven endothelial dysfunction, systemic inflammation, oxidative stress and metabolic disturbances. The persistent activation of inflammatory pathways in psoriasis creates a vicious cycle that accelerates atherosclerosis and predisposes patients to major adverse cardiovascular events. Understanding these mechanisms underscores the importance of early cardiovascular risk stratification and targeted immunomodulatory therapies in psoriatic patients to mitigate long-term vascular complications.[5]

DERMATOLOGIC-CARDIOVASCULAR INTERFACE IN PSORIASIS

The intricate relationship between dermatologic manifestations and cardiovascular pathology in psoriasis represents a paradigm of systemic inflammation with far-reaching clinical implications. This complex interplay transcends traditional organ-specific disease classifications, revealing psoriasis as a multisystem inflammatory disorder where cutaneous manifestations serve as visible indicators of profound systemic vascular dysfunction. At the core of this dermatologic-cardiovascular axis lies a shared pathophysiological pathway mediated by chronic immune dysregulation, with interleukin-17 (IL-17) emerging as a pivotal cytokine orchestrating both epidermal hyperproliferation and endothelial dysfunction. The inflammatory cascade originating in psoriatic skin lesions propagates systemically, creating a proatherogenic milieu characterized by endothelial activation, leukocyte recruitment and vascular remodeling that predisposes to accelerated atherosclerosis and major adverse cardiovascular events.[6]

The dermatologic component of this relationship manifests clinically as well-demarcated erythematous plaques with silvery scales, histopathologically characterized by epidermal hyperplasia, elongated rete ridges and marked dermal infiltration of T lymphocytes, dendritic cells and neutrophils. These cutaneous changes reflect local activation of the IL-23/Th17 axis, with consequent overproduction of IL-17, IL-22 and tumor necrosis factor-alpha (TNF-α) that not only drives keratinocyte proliferation but also spills over into systemic circulation.

The cardiovascular manifestations of this systemic inflammation typically remain subclinical for years before manifesting as overt cardiovascular disease, but can be detected through sophisticated assessments of endothelial function, arterial stiffness and subclinical atherosclerosis. Vascular endothelial cells exposed to chronic elevation of these inflammatory mediators undergo phenotypic changes characterized by reduced nitric oxide bioavailability, increased expression of adhesion molecules and enhanced permeability - all hallmarks of endothelial dysfunction that represent the earliest detectable stage of atherogenesis.

Molecular studies have elucidated several mechanisms through which dermatologic inflammation in psoriasis translates into cardiovascular pathology.[7]

IL-17 directly promotes endothelial dysfunction by upregulating vascular cell adhesion molecule-1 (VCAM-1) and intercellular adhesion molecule-1 (ICAM-1) expression, facilitating monocyte adhesion and transmigration into the vascular intima. Simultaneously, IL-17 synergizes with other psoriatic cytokines to stimulate vascular smooth muscle cell proliferation and migration, while inhibiting collagen production by vascular fibroblasts - processes that collectively contribute to plaque formation and destabilization. The chronic inflammatory state in psoriasis also promotes a prothrombotic environment through increased tissue factor expression, platelet activation and impaired fibrinolysis, further amplifying cardiovascular risk.[7]

Clinical observations have consistently demonstrated that psoriasis severity, as measured by body surface area involvement and Psoriasis Area Severity Index (PASI) scores, correlates with the degree of endothelial dysfunction and prevalence of subclinical atherosclerosis. Patients with severe psoriasis exhibit carotid intima-media thickness measurements comparable to those of patients with diabetes mellitus, while longitudinal studies have shown that psoriasis duration independently predicts incident cardiovascular events.

The dermatologic-cardiovascular connection is further strengthened by the observation that effective treatment of cutaneous inflammation with biologic therapies targeting the IL-17/IL-23 axis leads to measurable improvements in endothelial function and reductions in arterial stiffness, suggesting that amelioration of skin disease may confer cardiovascular benefits.[7] This profound interconnection between dermatologic and cardiovascular systems in psoriasis necessitates a collaborative care model integrating dermatologists, cardiologists and primary care physicians. Dermatologic evaluation should extend beyond assessment of skin lesions to include screening for cardiovascular risk factors, while cardiovascular assessments in psoriatic patients should incorporate disease-specific considerations such as inflammation-driven endothelial dysfunction. The development of novel biomarkers that simultaneously reflect cutaneous disease activity and cardiovascular risk, such as specific IL-17 isoforms or endothelial microparticle profiles, may further bridge these traditionally distinct medical domains.[8]

The recognition of psoriasis as a systemic inflammatory disorder with significant cardiovascular implications has transformed our understanding of disease pathogenesis and therapeutic goals. Modern management strategies must address both the visible dermatologic manifestations and the invisible vascular consequences of chronic inflammation, with treatment selection informed by potential impacts on both skin and cardiovascular health. Future research directions should focus on elucidating the precise molecular mechanisms linking specific dermatologic features to cardiovascular outcomes, developing integrated risk prediction models and investigating whether early aggressive control of cutaneous inflammation can prevent or reverse the progression of associated cardiovascular disease. This holistic approach to psoriasis management, acknowledging its dual dermatologic and cardiovascular nature, promises to improve not only skin outcomes but overall morbidity and mortality in affected patients.[8]

CLINICAL IMPLICATIONS

The recognition of psoriasis as a systemic inflammatory disorder with profound cardiovascular implications necessitates a paradigm shift in its clinical management, moving beyond dermatologic symptom control to comprehensive cardiovascular risk assessment and mitigation. Given the well-established association between psoriasis and accelerated atherosclerosis, endothelial dysfunction and major adverse cardiac events, healthcare providers must adopt a multidisciplinary approach that integrates dermatologic, cardiologic and metabolic evaluations. The chronic inflammatory milieu in psoriasis, driven largely by IL-17 and related cytokines, not only perpetuates cutaneous disease but also contributes to vascular dysfunction, underscoring the importance of early intervention to reduce long-term morbidity and mortality.[9]

One of the most critical clinical implications is the need for routine cardiovascular risk stratification in psoriatic patients, particularly those with moderate-to-severe disease, early-onset psoriasis or extensive body surface

area involvement. Traditional risk assessment tools, such as the Framingham Risk Score, may underestimate cardiovascular risk in this population due to the additive effects of systemic inflammation. Therefore, additional biomarkers of endothelial dysfunction and subclinical atherosclerosis such as flow-mediated dilation (FMD), carotid intima-media thickness (CIMT) and serum levels of high-sensitivity C-reactive protein (hs-CRP) should be considered in high-risk individuals. Furthermore, the presence of psoriatic arthritis (PsA) confers an even greater cardiovascular burden, necessitating closer monitoring for signs of metabolic syndrome, insulin resistance and vascular stiffness.[9,10]

Therapeutic strategies for psoriasis must also be reevaluated in the context of cardiovascular risk modulation. Emerging evidence suggests that certain systemic and biologic therapies may exert dual benefits by ameliorating both cutaneous inflammation and endothelial dysfunction. TNF-α inhibitors, for instance, have demonstrated potential in improving vascular function and reducing hs-CRP levels, though their long-term cardiovascular effects remain under investigation. More notably, IL-17 inhibitors (e.g., secukinumab, ixekizumab) and IL-23 inhibitors (e.g., ustekinumab, guselkumab) may offer cardioprotective effects by directly attenuating the pro-inflammatory pathways that contribute to endothelial injury and plaque instability.

However, the impact of these agents on hard cardiovascular outcomes such as myocardial infarction and stroke requires further longitudinal study. Conversely, some conventional systemic therapies, such as cyclosporine, may exacerbate hypertension and dyslipidemia, necessitating careful patient selection and close cardiometabolic monitoring.[11]

Lifestyle modifications remain a cornerstone of cardiovascular risk reduction in psoriatic patients, given the high prevalence of obesity, sedentary behavior and smoking in this population. Weight loss, dietary optimization and regular physical activity not only improve psoriasis severity but also mitigate insulin resistance, dyslipidemia and hypertension.

Smoking cessation is particularly crucial, as tobacco use amplifies both cutaneous inflammation and atherosclerotic progression. Additionally, statin therapy may be beneficial beyond lipid-lowering effects due to its anti-inflammatory properties, though its role in primary prevention for psoriatic patients without overt hyperlipidemia warrants further exploration.[11]

Finally, patient education is paramount in fostering adherence to long-term cardiovascular preventive measures. Many individuals with psoriasis remain unaware of their heightened cardiovascular risk, emphasizing the need for dermatologists and primary care physicians to emphasize the systemic nature of the disease. Collaborative care models involving dermatologists, cardiologists and endocrinologists can optimize early detection of vascular complications and ensure holistic management.[11]

In conclusion, the clinical implications of psoriasis-associated cardiovascular risk extend far beyond dermatologic manifestations, demanding a proactive, integrated approach to patient care. By addressing inflammation-driven endothelial dysfunction through targeted immunomodulation, aggressive risk factor modification and multidisciplinary collaboration, clinicians can mitigate the excess cardiovascular morbidity and mortality observed in this vulnerable population. Future research should focus on elucidating the long-term cardiovascular outcomes of biologic therapies and refining risk prediction models tailored to psoriatic patients.[11]

DIAGNOSTIC MODALITIES

The accurate assessment of cardiovascular risk in patients with psoriasis necessitates a comprehensive diagnostic approach that bridges dermatological manifestations with systemic vascular pathology. Given the well-established association between chronic cutaneous inflammation and accelerated atherosclerosis, contemporary clinical practice demands sophisticated diagnostic modalities capable of detecting subclinical vascular dysfunction while evaluating the systemic inflammatory burden mediated by IL-17 and related cytokines. The diagnostic paradigm encompasses clinical evaluation, biochemical profiling, advanced imaging techniques and functional vascular assessments, all of which contribute to a multidimensional understanding of psoriasis-associated cardiovascular pathology.[12]

Clinical evaluation begins with meticulous dermatological assessment using standardized tools such as the Psoriasis Area and Severity Index (PASI) and body surface area (BSA) measurement, as disease severity correlates with systemic inflammation magnitude and subsequent cardiovascular risk. Concurrent rheumatological evaluation for psoriatic arthritis is imperative given its association with more pronounced endothelial dysfunction. A thorough cardiovascular history and physical examination focusing on traditional risk factors including hypertension, dyslipidemia, diabetes mellitus and smoking status must be integrated with psoriasis-specific parameters such as disease duration and age of onset, as early-onset psoriasis carries particular cardiovascular significance.[12]

Biomarker analysis plays a pivotal role in stratifying cardiovascular risk in psoriatic patients. Serum measurements of high-sensitivity C-reactive protein (hs-CRP) and interleukin-6 (IL-6) provide quantifiable assessments of systemic inflammation, while specific evaluation of IL-17 and IL-23 levels may offer insights into Th17 pathway activation. Endothelial dysfunction biomarkers including asymmetric dimethylarginine

(ADMA), endothelin-1 and circulating endothelial progenitor cells (EPCs) serve as early indicators of vascular compromise. Lipid profiling in psoriatic patients should extend beyond conventional panels to include oxidized low-density lipoprotein (oxLDL) and lipoprotein(a) measurements, which better reflect the proatherogenic lipid milieu characteristic of chronic inflammation.[13]

Non-invasive vascular imaging modalities have emerged as essential tools for detecting subclinical atherosclerosis in psoriatic populations. Carotid artery ultrasonography with intima-media thickness (cIMT) measurement provides a validated assessment of early arterial remodeling, while vascular ultrasound with flow-mediated dilation (FMD) testing evaluates endothelial function through nitric oxide-dependent vasomotor responses. Coronary artery calcium (CAC) scoring via computed tomography offers quantitative assessment of coronary atherosclerosis burden, particularly valuable in patients with moderate-to-severe psoriasis. Advanced techniques such as pulse wave velocity (PWV) analysis and augmentation index measurement provide functional assessments of arterial stiffness, a hallmark of premature vascular aging in chronic inflammatory conditions.[13]

Emerging diagnostic approaches include molecular imaging techniques targeting vascular inflammation, such as 18F-fluorodeoxyglucose positron emission tomography (FDG-PET), which can identify metabolically active atherosclerotic plaques. Circulating microRNA profiles and proteomic analyses are under investigation as potential tools for predicting cardiovascular risk in psoriatic patients. The integration of these advanced diagnostic modalities with conventional cardiovascular risk assessment tools creates a comprehensive framework for early detection of vascular pathology in this high-risk population.[13]

The diagnostic algorithm must be tailored to individual patient characteristics, with particular attention to psoriasis severity, disease duration and presence of comorbidities. Regular monitoring of cardiovascular parameters should be incorporated into the long-term management of psoriatic patients, with frequency adjusted according to baseline risk stratification. This systematic diagnostic approach enables timely intervention to mitigate the excess cardiovascular morbidity associated with chronic cutaneous inflammation, ultimately improving long-term outcomes in this vulnerable population. Future diagnostic developments may focus on personalized risk prediction models incorporating genetic predisposition, cytokine profiles and vascular imaging parameters to optimize cardiovascular risk management in psoriasis.[14]

THERAPEUTIC INTERVENTIONS

The management of psoriasis in patients with elevated cardiovascular risk requires a sophisticated therapeutic approach that addresses both the dermatologic manifestations and the underlying systemic inflammatory processes contributing to endothelial dysfunction and accelerated atherosclerosis. Contemporary treatment paradigms emphasize the importance of selecting therapeutic modalities that not only achieve adequate cutaneous disease control but also favorably modulate the pro-inflammatory milieu driving cardiovascular pathology. The armamentarium of available treatments spans topical agents, systemic therapies, biologic immunomodulators and cardiovascular risk-reduction strategies, each with distinct mechanisms of action and varying impacts on the IL-17-mediated inflammatory cascade and vascular homeostasis.[14]

Topical therapies including corticosteroids, vitamin D analogs and calcineurin inhibitors remain first-line interventions for mild psoriasis, though their effect on systemic inflammation and cardiovascular risk parameters is generally negligible. For patients with moderate-to-severe disease exhibiting systemic manifestations, phototherapy with narrowband UVB may offer dual benefits by improving cutaneous lesions while potentially reducing circulating inflammatory markers, though its long-term effects on endothelial function require further investigation. Conventional systemic agents such as methotrexate demonstrate particular relevance in this population due to their dual anti-inflammatory effects on both psoriatic plaques and cardiovascular system, with evidence suggesting potential cardioprotective benefits through reduction of IL-6 and C-reactive protein levels, though careful monitoring for hepatic and hematologic toxicity remains essential.[14]

The advent of biologic therapies has revolutionized psoriasis treatment while providing unique opportunities to modify cardiovascular risk through targeted immunomodulation. TNF-α inhibitors such as adalimumab and infliximab have demonstrated capacity to improve endothelial function and reduce arterial stiffness, though their effects on major adverse cardiovascular events remain debated. IL-12/23 inhibitors represented by ustekinumab exhibit particular promise through their upstream modulation of the Th17 pathway, with emerging data suggesting favorable impacts on vascular inflammation markers. The most compelling cardiovascular benefits may derive from direct IL-17 inhibitors including secukinumab and ixekizumab, which target the central cytokine implicated in both psoriatic plaque formation and endothelial dysfunction, though long-term cardiovascular outcome studies are still ongoing.[15]

Concomitant management of traditional cardiovascular risk factors assumes critical importance in psoriatic patients, with statin therapy offering particular benefits beyond lipid lowering through pleiotropic anti-inflammatory effects. Angiotensin-converting enzyme inhibitors may be preferred for hypertension management given their potential to counteract endothelial

dysfunction, while novel antidiabetic agents such as GLP-1 receptor agonists and SGLT2 inhibitors demonstrate intriguing anti-inflammatory properties relevant to this population. Lifestyle interventions including Mediterranean diet adoption, structured exercise programs and smoking cessation provide foundational support for both dermatologic and cardiovascular improvement, with weight reduction demonstrating particular efficacy in overweight patients through reduction of adipokine-mediated inflammation.[16]

The selection of optimal therapeutic regimens must be individualized based on psoriasis severity, cardiovascular risk profile and comorbidities, with particular attention to potential drug-drug interactions and overlapping toxicities. Emerging treatment strategies under investigation include dual cytokine inhibition approaches and small molecule inhibitors targeting intracellular signaling pathways common to both cutaneous inflammation and atherogenesis. The integration of dermatologic and cardiovascular therapeutic goals necessitates close collaboration between dermatologists, cardiologists and primary care providers to optimize outcomes, with regular monitoring of both skin disease activity and vascular parameters. Future therapeutic developments will likely focus on precision medicine approaches targeting individual patient cytokine profiles and genetic predispositions to simultaneously address cutaneous inflammation and its cardiovascular consequences.[17]

CONCLUSION

The extensive body of evidence examined in this review establishes psoriasis as far more than a cutaneous disorder, positioning it as a systemic inflammatory condition with profound implications for cardiovascular health. The pathophysiological interplay between IL-17-mediated skin inflammation and vascular endothelial dysfunction creates a perfect storm for accelerated atherogenesis, transforming psoriatic plaques into visible markers of an invisible systemic inflammatory burden. The chronic inflammatory milieu characteristic of moderate-to-severe psoriasis, characterized by persistent elevation of IL-17, TNF-α and other pro-inflammatory cytokines, initiates and perpetuates a cascade of vascular insults including endothelial cell activation, leukocyte recruitment, oxidative stress generation and ultimately plaque formation and destabilization. This mechanistic understanding fundamentally alters our clinical approach to psoriasis management, necessitating a paradigm shift from purely symptomatic skin-directed therapy to comprehensive care addressing systemic inflammation and its cardiovascular consequences.

The recognition of psoriasis as an independent cardiovascular risk factor carries important implications for risk stratification and therapeutic decision-making. Current evidence suggests that psoriasis severity, duration and extent of skin involvement correlate with the degree of endothelial dysfunction and subclinical atherosclerosis, making dermatologic assessment a potentially valuable tool for cardiovascular risk prediction. The IL-17 axis emerges as a critical link between cutaneous and vascular pathology, with this cytokine serving as both a therapeutic target and potential biomarker for cardiovascular risk in psoriatic patients. While traditional cardiovascular risk factors certainly contribute to adverse outcomes in this population, the additive effects of chronic inflammation create a distinct vascular phenotype characterized by premature endothelial aging, increased arterial stiffness and heightened plaque vulnerability.

Therapeutic considerations must now balance efficacy in skin clearance with potential impacts on vascular health. Biologic agents targeting the IL-23/IL-17 axis demonstrate particular promise not only for their superior efficacy in psoriasis management but also for their potential to mitigate inflammation-driven cardiovascular damage. However, the long-term cardiovascular outcomes of these targeted immunomodulatory therapies require continued investigation through large-scale prospective studies and registry data. Conventional systemic therapies present a more complex risk-benefit profile, with methotrexate showing potential anti-inflammatory cardiovascular benefits that must be weighed against hepatotoxic and myelosuppressive risks, while cyclosporine's unfavorable metabolic effects may exacerbate cardiovascular risk despite its potent anti-psoriatic activity.

This review highlights the critical need for multidisciplinary collaboration in managing psoriatic patients, integrating dermatologic care with cardiovascular risk assessment and prevention strategies. Routine screening for traditional cardiovascular risk factors, coupled with consideration of psoriasis-specific parameters such as disease severity and duration, should become standard practice in dermatology clinics. Conversely, cardiologists must recognize severe psoriasis as a marker of increased cardiovascular risk warranting more aggressive preventive strategies. Future research directions should focus on elucidating the precise mechanisms by which cutaneous inflammation drives vascular pathology, identifying biomarkers to predict cardiovascular risk in psoriatic patients and determining optimal treatment algorithms that address both skin disease and vascular health.

Ultimately, the recognition of psoriasis as a systemic inflammatory disorder with significant cardiovascular implications mandates a holistic treatment approach that transcends traditional specialty boundaries. By viewing psoriatic plaques not merely as dermatologic lesions but as outward manifestations of systemic inflammation with far-reaching vascular consequences, we can develop more comprehensive management strategies that improve not just skin outcomes but overall morbidity and mortality in this vulnerable patient population. The coming years will

likely see continued refinement of our understanding of the psoriasis-cardiovascular axis, with the potential for novel therapeutic approaches that simultaneously target cutaneous and vascular inflammation, ultimately improving quality of life and long-term outcomes for patients with this complex systemic disease.

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