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REVIEW ARTICLE

Cardiovascular Imaging in Psoriasis: A Critical Review of Current Evidence, Techniques, and Therapeutic Implications

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Abstract

Psoriasis is a systemic inflammatory disease increasingly recognized for its association with elevated cardiovascular risk, driven by immune-mediated endothelial dysfunction and accelerated atherosclerosis. Non-invasive cardiovascular imaging has consistently demonstrated a spectrum of subclinical abnormalities in patients with moderate-to-severe psoriasis, including coronary microvascular dysfunction, high-risk plaque burden, vascular inflammation, myocardial remodeling, and increased arterial stiffness. This critical review summarizes the role of different imaging modalities in detecting psoriasis-associated cardiovascular alterations and evaluates emerging data on the impact of systemic therapies. Imaging abnormalities in psoriasis often precede clinical events and can support early risk reclassification and referral. Biologic therapy is associated with improved coronary microvascular function and with reductions in noncalcified plaque burden and high-risk plaque features on coronary computed tomography angiography (CCTA), and statins reduce vascular ^{18}F-fluorodeoxyglucose (FDG) uptake on positron emission tomography (PET) in psoriasis cohorts. Outcome data that link these imaging changes to reduced events remain limited. Imaging should be used selectively, with coronary calcium scoring or carotid ultrasound for risk refinement in asymptomatic adults and targeted CCTA or transthoracic echocardiography (TTE) when symptoms, examination, or screening suggest cardiovascular involvement.

Key Points

Patients with moderate-to-severe psoriasis may have hidden cardiovascular abnormalities even in the absence of symptoms.

Advanced imaging techniques can reveal early signs of coronary microvascular dysfunction and atherosclerosis.

Recognizing the impact of systemic inflammation on the cardiovascular system can guide more effective treatment strategies and improve long-term outcomes.

1 Introduction

Psoriasis is a chronic immune-mediated inflammatory disease that affects approximately 2–3% of the global population [1]. Psoriasis is increasingly recognized as a systemic disorder, associated with multiple comorbidities, including an increased risk of cardiovascular disease (CVD) [2]. Patients with moderate-to-severe psoriasis face a higher incidence of major adverse cardiovascular events (MACE), such as myocardial infarction, stroke, and cardiovascular death [3]. Chronic systemic inflammation appears to be the main driver linking psoriasis and cardiovascular disease. Activation of the Th1/Th17 immune axis has been implicated in systemic inflammation and may contribute to endothelial dysfunction, oxidative stress, and atherosclerosis, although definitive mechanistic links remain incompletely established [4–6]. This axis is thought to trigger systemic inflammation, metabolic dysfunction, and ultimately cardiovascular disease, a process commonly referred to as the "psoriatic march" [7].

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Subclinical cardiovascular alterations are frequently observed in patients with psoriasis: imaging studies have reported increased coronary plaque burden, vascular inflammation, coronary microvascular dysfunction (CMD), and early myocardial remodeling [8–11]. However, results remain inconsistent across studies. While some studies demonstrate significant vascular and myocardial changes, others report only modest alterations, leading to debate over their clinical and prognostic relevance [12]. Differences in imaging techniques, study design, and patient characteristics likely contribute to the heterogeneity of findings. Whether systemic treatments, particularly biologic agents targeting tumor necrosis factor (TNF), interleukin (IL)-12/23, or IL-17, can reduce cardiovascular alterations remains under investigation. Preliminary evidence suggests potential benefits, such as reductions in vascular inflammation, CMD, and plaque burden [8, 13, 14]. In addition, statins have been investigated as potential modulators of vascular inflammation in psoriasis, and psoriasis-specific ^{18}F-fluorodeoxyglucose positron emission tomography (FDG-PET) studies provide preliminary evidence in this setting [15].

Dermatologists are often the first and most consistent point of contact for patients with psoriasis. Knowing the timing and options for noninvasive cardiovascular imaging can refine risk assessment and guide co-management and is therefore clinically relevant. Current guidelines provide limited and sometimes inconsistent recommendations for cardiovascular evaluation in patients with psoriasis, irrespective of the presence of established CVD. Conversely, a wide range of imaging techniques is available, ranging from invasive to noninvasive and from costly to relatively accessible. A clear understanding of these modalities and their clinical implications is essential for dermatologists to navigate patient care and collaborate effectively with cardiologists.

This review aims to critically evaluate the role of cardiovascular imaging in psoriasis, summarizing the current evidence, assessing the strengths and limitations of different imaging techniques, and exploring the impact of systemic therapies on cardiovascular outcomes.

2 Literature Search Methodology

We performed a focused, narrative review of cardiovascular imaging in adult patients with psoriasis. Our sources included PubMed/MEDLINE and Embase, from inception to May 2025. We combined terms for psoriasis (e.g., "psoriasis" and "psoriatic arthritis") with imaging modalities ("transthoracic echocardiography," "coronary computed tomography angiography," "coronary artery calcium scoring," "epicardial adipose tissue," "pericoronary adipose tissue," "FDG-PET," "cardiovascular magnetic resonance," "carotid ultrasound," "arterial stiffness," "pulse wave velocity," and "tonometry"). References of key articles were also screened.

We included peer-reviewed original research, meta-analyses, and clinical guidelines focusing on adult populations. We excluded non-English publications, pediatric cohorts, and case reports/series, unless they provided significant methodological insights.

3 Transthoracic Echocardiography

3.1 Coronary Flow Reserve

Coronary flow reserve (CFR) reflects the ability of the coronary circulation to dilate and increase blood flow in response to elevated myocardial metabolic demand. In healthy individuals, CFR typically ranges from 3 to 6, indicating that coronary blood flow can increase at least threefold above baseline when needed [16]. A CFR $\leq$ 2.5 may suggest the presence of significant coronary artery stenosis (> 70%), CMD in the absence of obstructive coronary artery disease, or a combination of both. Importantly, when epicardial coronary arteries appear normal, a reduced CFR points to an underlying dysfunction of the coronary microcirculation (CMD) [17]. CMD, identified by a reduced CFR, is a condition that can precede overt coronary artery disease (CAD) and is therefore a powerful predictor of adverse cardiovascular outcomes [18]. While positron emission tomography (PET) remains the gold standard for its measurement [16], CFR can also be assessed noninvasively via transthoracic Doppler echocardiography [19].

Growing evidence indicates a significant impairment in CFR in patients with psoriasis, as assessed by transthoracic Doppler echocardiography, reinforcing the association between systemic inflammation and CMD [10, 13, 14, 20–24]. Osto et al. first reported that CFR was significantly lower in patients with psoriasis than in controls (3.2 versus 3.7; $p = 0.02$) [13] in a cohort of 56 young patients (mean age 37 years) without clinical CVD. A meta-analysis reported a pooled standardized mean difference (SMD) of − 0.71 (95% confidence interval [CI]: − 0.97 to − 0.45), quantifying the magnitude of CFR impairment in psoriasis across studies [25]. Although the prognostic impact of CFR differences has not been prospectively validated in psoriasis, comparable impairments in other clinical populations have been linked to increased risk of MACEs; indeed, a 1-unit decrease in CFR has been shown to translate into an approximately twofold higher risk of cardiovascular death or heart failure (hazard ratio [HR] ≈ 2.0), suggesting that even modest CFR reductions in psoriasis may carry meaningful implications [26]. One study

found that patients with psoriasis with a CFR ≤ 2.5 had an almost tenfold increased risk of cardiovascular events (odds ratio: 9.9; 95% CI 6.1–15.8; $p < 0.0001$), suggesting that CFR may serve as a valuable marker for cardiovascular risk stratification in this population [10].

In addition to confirming the association between psoriasis and CMD, some studies have also investigated the relationship between disease severity and CMD, reporting an inverse correlation between CFR and Psoriasis Area and Severity Index (PASI) scores. This further supports the link between coronary microvascular impairment and systemic inflammatory burden in patients with psoriasis. Piaserico et al. demonstrated that each one-point increase in PASI is associated with a 5.8% higher risk of developing CMD [24].

Disease duration and the presence of psoriatic arthritis (PsA) also contribute to CFR reduction in psoriasis. Osto et al. identified both longer disease duration and PsA as independent predictors of CMD [13]. Piaserico et al., in a cohort of 503 patients, confirmed both factors as independent predictors [24]. Gullu et al. observed an inverse correlation between CFR and disease duration, while Ikonomidis et al. showed more severe CFR impairment in patients with PsA than in those with only cutaneous involvement [21, 27]. These findings suggest that chronic inflammation, amplified by PsA and prolonged disease, may worsen microvascular dysfunction over time.

3.2 Impact of Psoriasis Pharmacotherapy on CFR

Given the key role of inflammation in CMD, recent studies have examined the effects of immunomodulatory treatments on CFR. In a prospective psoriasis cohort, TNF inhibitor therapy was associated with a significant increase in Doppler-derived CFR, with larger absolute gains in patients with baseline CFR ≤ 2.5, suggesting that targeted biologic therapy may partially reverse CMD [23].

Many Doppler studies quantified CFR as coronary flow velocity reserve (CFVR), a velocity-derived surrogate measured by transthoracic echocardiography and interpreted equivalently as coronary vasodilatory capacity. Across these reports, CFVR was reduced in psoriasis versus controls and improved with systemic anti-inflammatory therapy; for example, Tona et al. observed a significant improvement in CFVR following systemic anti-inflammatory treatment with drugs including adalimumab, etanercept, infliximab, ustekinumab, or cyclosporine, reinforcing the hypothesis that immunomodulation positively impacts coronary microvascular function [14]. The mechanisms underlying these improvements likely involve suppression of inflammatory cytokines, leading to enhanced endothelial nitric oxide bioavailability, reduced oxidative stress, and attenuation of microvascular remodeling [23]. These findings support the concept that CMD in psoriasis is not only a marker of systemic inflammation but may also be a modifiable target through appropriate therapy.

Despite these promising results, several critical gaps remain. The long-term impact of biologic therapies on MACEs is yet to be elucidated, and it remains uncertain whether improvements in CFR and CFVR translate into reduced cardiovascular morbidity and mortality. In addition, head-to-head studies comparing the efficacy of different biologic classes, such as TNF inhibitors versus IL-17 or IL-23 inhibitors, on CMD are lacking, leaving an unresolved question of which therapeutic strategies are optimal for cardiovascular risk mitigation in this population. Moreover, the clinical utility of CFR and CFVR for routine cardiovascular risk stratification in psoriasis has not been firmly established, underscoring the need for larger, prospective trials with extended follow-up periods.

3.3 Cardiac Structure, Function and Epicardial Adipose Tissue

Transthoracic echocardiography (TTE) has been employed to detect a range of subclinical cardiac abnormalities in patients with psoriasis, extending beyond microvascular alterations. Notably, psoriasis has been linked to a higher prevalence of subclinical left ventricular diastolic dysfunction, occurring in 8–36% of patients with psoriasis vulgaris and in 28–63% of those with psoriatic arthritis [28]. In a case–control study involving young adults without traditional cardiovascular risk factors, mild diastolic impairment was identified in approximately 36% of individuals with psoriasis, whereas no such abnormalities were detected in the control group [29]. Another study confirmed this evidence and showed that treatment of psoriasis with TNF inhibitors could restore the diastolic dysfunction [23]. Diastolic dysfunction reflects early myocardial stiffness or impaired relaxation, changes that can occur before any detectable impairment in systolic contractility. This phenotype may resemble heart failure with preserved ejection fraction (HFpEF), a condition in which patients experience heart failure symptoms due to diastolic dysfunction despite having normal systolic function. HFpEF is particularly common in individuals with inflammatory and metabolic conditions [30, 31]. In this context, a key pathogenic paradigm involves a sequence of events in which coronary microvascular inflammation and dysfunction play a central role [32].

Even though systolic function by conventional ejection fraction usually remains preserved, speckle-tracking studies consistently demonstrate reduced myocardial strain in psoriasis. A recent meta-analysis confirmed significant decreases in both global longitudinal and circumferential strain in patients with psoriasis, indicating early subclinical systolic dysfunction despite normal ejection fraction [33]. Chronic inflammation in psoriasis may also promote subtle

structural remodeling of the heart. Some echocardiographic studies report an increased incidence of left ventricular hypertrophy or wall thickening in psoriasis cohorts [34], and elevated inflammatory biomarker levels (e.g., serum amyloid A) have been linked to higher left ventricular mass indices in these patients, suggesting that disease activity contributes to ventricular remodeling [35]. In addition, echocardiographic studies have consistently demonstrated increased epicardial fat thickness in psoriasis compared with healthy controls [36, 37]. This increase appears to be independent of concomitant metabolic syndrome, suggesting a disease-specific effect [38].

Finally, two large cohorts have examined incident aortic stenosis in psoriasis [39, 40]. A Danish nationwide study (~5.1 million adults) reported a higher incidence with a severity gradient after multivariable adjustment [39]. In a contemporary UK Biobank cohort, psoriasis was likewise associated with new-onset aortic stenosis, with no association observed for other degenerative valve diseases and a stronger effect in women [40].

To date, no echocardiographic studies in psoriasis have prospectively evaluated statin therapy on CFR/CFVR, diastolic indices, or myocardial strain.

4 Coronary Computed Tomography Angiography and Coronary Calcium

Coronary computed tomography angiography (CCTA) is a non-invasive imaging modality that assesses CAD by detecting both calcified and noncalcified plaques, as well as vascular inflammation. Unlike invasive coronary angiography, CCTA offers a detailed evaluation of coronary artery morphology with minimal procedural risks. Advances in technology now allow low-dose protocols, equivalent to less than 1 year of natural background radiation [41]. This enhanced safety profile has expanded its clinical application, making CCTA an essential tool for evaluating subclinical atherosclerosis, particularly in populations with increased cardiovascular risk.

4.1 Measures of Atherosclerotic Burden

Growing evidence suggests that psoriasis is associated with a higher prevalence of coronary atherosclerosis, an increased burden of noncalcified plaques, and a greater prevalence of plaques with high-risk characteristics, which contribute to elevated cardiovascular risk [42]. Beyond assessing luminal stenosis, CCTA quantifies coronary artery calcification through the coronary artery calcium score (CAC), a marker of atherosclerosis progression that typically reflects more advanced stages of the disease [43]. It also allows for the assessment of epicardial adipose tissue (EAT) and pericoronary adipose tissue (PCAT), both emerging markers of vascular inflammation [44].

Multiple studies have demonstrated that psoriasis is associated with increased coronary plaque burden, particularly noncalcified plaques. In a prospective study on 105 patients, Lerman et al. reported significantly higher total arterial plaque burden and noncalcified burden in patients with psoriasis compared with controls [45], even though they were younger and had fewer traditional cardiovascular risk factors. Patients with psoriasis also had a similar prevalence of high-risk plaques compared with statin-eligible hyperlipidemic controls [45]. Further research by Joshi et al. and Elnabawi et al. demonstrated that total arterial plaque burden was mainly driven by noncalcified components and that noncalcified plaque burden correlated with disease severity (PASI score), independent of traditional risk factors [8, 46].

The association of psoriasis not only with overall plaque burden but also with a higher prevalence of high-risk plaques—which are more prone to rupture and to cause severe stenosis, thereby contributing to increased cardiovascular event risk—has been confirmed by several reports. A meta-analysis of 14 studies (1427 patients) confirmed a higher prevalence of CAD and high-risk plaques detected by CCTA [47]. Collectively, these findings underscore the role of systemic inflammation in both increased atherosclerotic burden and plaque vulnerability. The observed correlation between PASI score and high-risk plaque features suggests that heightened inflammatory activity may influence coronary plaque behavior. While more research is needed to clarify this relationship, these data support early cardiovascular risk assessment in individuals with psoriasis.

CAC scoring has offered insight into atherosclerosis in psoriasis, although findings are mixed. Some studies report elevated CAC in patients with psoriasis, while others do not, thereby highlighting noncalcified plaque burden as a potentially more relevant risk marker [48]. A meta-analysis involving 3039 patients confirmed increased CAC levels, but found no correlation with PASI scores or disease duration [49]. These results suggest that CAC may underestimate cardiovascular risk in psoriasis, as many patients have substantial noncalcified plaque burden despite low CAC scores.

4.2 Pericoronary and Epicardial Adipose Tissue on CCTA

Beyond coronary plaques and stenosis, EAT has gained attention as a marker of cardiovascular inflammation and metabolic dysfunction in psoriasis. EAT, the fat deposit surrounding the myocardium, has been implicated in the regulation of local inflammatory processes and has been shown to play a role in the progression of CAD [50]. In a longitudinal study of 301 patients, O'Hagan et al. reported

that greater EAT volume correlated with higher fibro-fatty plaque burden and left ventricular mass, an early indicator of cardiac remodeling [51]. A recent systematic review and meta-analysis of 10 studies ($n = 1287$) demonstrated that patients with psoriasis exhibit significantly increased EAT compared with healthy controls [52].

The role of EAT in psoriasis is complex, with imaging studies yielding conflicting results. While echocardiography shows increased EAT thickness, comprehensive volumetric computed tomography (CT) assessments have failed to consistently detect a significant increase in total EAT volume. This raises the possibility that the pro-inflammatory effects of EAT are independent of its absolute volume or that psoriatic inflammation selectively targets certain cardiac regions.

Pericoronary adipose tissue (PCAT), assessed via CCTA using the perivascular fat attenuation index (PFAI), has been linked to coronary inflammation and cardiovascular events [53]. PCAT may contribute to disease through dual mechanisms: acting as a paracrine source of inflammatory mediators affecting the coronary wall ("outside-in") and serving as a sensor that reflects underlying vascular inflammation through compositional changes ("inside-out") [54]. Data concerning psoriasis remain conflicting. While a case report has described increased PCAT attenuation in patients with psoriasis and metabolic comorbidities [55], a prospective study by Bao et al. found lower PFAI values in patients with psoriasis versus controls [9]. This unexpected finding challenges the hypothesis that chronic low-grade inflammation in psoriasis directly translates into increased coronary inflammation. In contrast, Elnabawi et al. observed a reduction in PFAI after 1 year of biologic therapy, independent of plaque burden [56]. These contrasting results highlight the complexity of interpreting PFAI in psoriasis and underscore the need for studies integrating metabolic, inflammatory, and imaging parameters.

4.3 Impact of Psoriasis Pharmacotherapy on CCTA Metrics

Emerging evidence suggests that systemic biologic therapy for psoriasis may lead to measurable improvements in coronary atherosclerosis as assessed by CCTA. Three complementary analyses from a single longitudinal US cohort have consistently demonstrated atheroprotective changes following biologic treatment [8, 45, 51]. In the first analysis, conducted in 215 patients, biologic therapy was associated with a 6% reduction in noncalcified plaque burden following 1 year of biologic therapy, accompanied by a significant decrease in necrotic core volume. Notably, plaque regression was significantly greater in biologic-treated patients compared with those not receiving biologics and remained independently associated with biologic therapy after adjusting for traditional cardiovascular risk factors [8]. A subsequent study examined changes in plaque characteristics according to clinical response, showing that patients achieving significant PASI improvement under systemic/biologic therapy (including methotrexate, TNF and IL-12/23 and IL-17 inhibitors) experienced marked reductions in total and noncalcified plaque burden, whereas those with worsening PASI exhibited plaque progression [45].

Finally, longitudinal follow-up of biologic-naïve patients confirmed significant reductions in total arterial plaque, noncalcified plaque, and fibrous burden after initiation of biologic therapy, together with a trend toward reduced EAT volume [51]. Globally, these findings support the hypothesis that targeted inhibition of inflammatory cytokines may slow or even partially reverse atherosclerotic progression in patients with psoriasis.

However, as with other imaging-based findings, the optimal therapeutic strategy remains to be defined. The differential effects of TNF inhibitors, IL-17 inhibitors, and IL-23 inhibitors on vascular inflammation and plaque composition require further investigation to determine which agents offer the greatest cardiovascular benefit. We found no psoriasis-specific CCTA studies testing statins with plaque burden/composition or PCAT indices as endpoints.

5 Positron Emission Tomography

FDG-PET is a noninvasive imaging technique that uses ^{18}F-fluorodeoxyglucose (FDG) to quantify tissue metabolic activity. As FDG uptake reflects cellular metabolism, this method is widely used to detect inflammation, malignancies, and cardiovascular disease [57].

In cardiovascular imaging, FDG-PET detects vascular inflammation by measuring glucose uptake in arterial walls. Elevated FDG uptake in the aorta and carotid arteries has been associated with increased risk of MACEs [58, 59].

5.1 Vascular and Systemic Inflammation

FDG-PET/CT has been employed to assess vascular inflammation in patients with psoriasis. Increased FDG uptake in the aorta and carotid arteries of patients with moderate-to-severe psoriasis indicates the presence of subclinical atherosclerosis and an elevated cardiovascular risk [60].

Multiple studies have confirmed increased vascular inflammation in psoriasis using FDG-PET/CT [61–64]. Beyond the skin and arterial wall, FDG-PET can detect immune–metabolic activity in adipose tissues, spleen, and bone marrow. In psoriasis cohorts, uptake in these compartments correlates with aortic vascular inflammation [61, 63].

In a clinical trial involving 33 patients with psoriasis and 84 PET/CT scans, Kothekar et al. introduced the PET–PASI

score, which quantifies metabolic activity in psoriatic skin lesions [60]. PET–PASI strongly correlated with clinical PASI, supporting the ability of FDG-PET to capture skin disease severity through metabolic imaging. Together, these findings support the utility of FDG-PET/CT as a sensitive tool for detecting both cutaneous and vascular inflammation in psoriasis.

Elevated FDG uptake in aortic segments (measured with aortic target-to-background ratio) appears to correlate with PASI score, even after adjustment for age, sex, and Framingham risk score, thereby confirming the association between cutaneous and vascular inflammation [62, 65].

Vascular inflammation associated with psoriasis has been shown to be comparable with that observed in other chronic inflammatory diseases, such as rheumatoid arthritis (RA). Rose et al. used FDG-PET/CT to compare vascular inflammation among patients with psoriasis, RA, and healthy controls [64]. Despite similar cardiovascular risk profiles across groups, the study demonstrated significantly higher vascular inflammation in both patients with psoriasis and RA compared with healthy individuals, even after adjusting for cardiovascular risk factors. The study reported that regional aortic vascular inflammation, measured as mean metabolic volumetric product, was significantly increased in both psoriasis and RA. These findings indicate that patients with psoriasis, similar to those with RA, exhibit heightened vascular inflammation that may contribute to an increased cardiovascular risk. This aligns with epidemiological evidence suggesting a higher prevalence of premature atherosclerosis in both conditions [66].

5.2 Impact of Biologic Therapy and Statins on Vascular FDG-PET Findings

Vascular inflammation detected by FDG-PET/CT has been shown to change with statin therapy. In 83 patients with psoriasis, Kaiser et al. showed that statin therapy reduced FDG uptake in the aorta and its branches, indicating lower vascular inflammation [15]. This benefit was observed even in patients with established cardiovascular disease, suggesting that the anti-inflammatory effects of statins persist regardless of existing atherosclerotic burden. Beyond lipid lowering, statins exert pleiotropic anti-inflammatory effects, including reductions in vascular inflammation and C-reactive protein levels (CRP) [67]. From a pathophysiological perspective, early use of lipid-lowering agents may help interrupt the "psoriatic march," the progression from skin inflammation to systemic atherosclerosis. The observed reduction in vascular inflammation with statins supports this approach.

Moreover, data from a small cohort suggested a potential benefit from the use of various biologic agents in reducing vascular inflammation in the thoracic aorta over a 6-month period, as measured by FDG-PET/CT [68]. This effect was not observed in patients with psoriasis treated with nonsystemic therapies or in control subjects with noninflammatory joint and skin diseases.

Conversely, a more rigorous randomized, double-blind study evaluating the effects of the TNF-α antagonist adalimumab on vascular inflammation in the ascending aorta did not demonstrate a significant reduction in FDG uptake [69]. These findings raise questions about the generalizability of treatment effects across different vascular regions.

In this context, other authors have argued that selecting the ascending aorta as the representative segment for evaluating vascular inflammation may not be optimal [70]. Indeed, Hjuler et al. reported that increased aortic wall inflammation in patients with psoriasis, compared with noninflammatory controls, was present in all segments of the aorta except the ascending portion [71].

Consistent with this complexity, prospective studies assessing whole aortic vascular inflammation with other agents, including apremilast, ustekinumab, and secukinumab, have shown conflicting results, primarily reporting no significant effect [72–75].

FDG-PET/CT has emerged as a valuable tool not only for assessing cutaneous inflammatory activity but also for detecting subclinical vascular inflammation. A consistent association between PASI score and arterial FDG uptake supports the link between systemic inflammatory burden and vascular disease in psoriasis. Interestingly, reductions in vascular inflammation have been observed following early intervention with lipid-lowering agents, particularly statins, suggesting a potential preventive role in halting the so-called psoriatic march. Nevertheless, studies evaluating the impact of different biologic agents on vascular inflammation have produced surprisingly inconsistent findings, underscoring the need for further standardized and adequately powered investigations.

6 Cardiovascular Magnetic Resonance and Cardiac Remodeling

Cardiovascular magnetic resonance (CMR) has emerged as a tool for assessing subclinical cardiac dysfunction in inflammatory diseases, offering tissue characterization beyond conventional echocardiography and computed tomography. Advanced parametric techniques, including native and post-contrast T1 mapping for detecting diffuse myocardial fibrosis and T2 mapping for identifying myocardial edema, enable a noninvasive, quantitative assessment of myocardial pathology. In addition, late gadolinium enhancement (LGE) imaging allows for the detection of focal myocardial fibrosis, providing data on chronic structural remodeling [76, 77].

6.1 Tissue Characterization: Fibrosis, Edema, and Remodeling

Despite the well-documented association between psoriasis and CVD, studies investigating myocardial alterations via CMR in patients with psoriasis remain scarce. However, the available evidence suggests that subclinical myocardial involvement is a feature of psoriasis-related cardiovascular risk. In a prospective CMR study of 60 patients with psoriasis, Gröschel et al. reported reduced myocardial strain despite preserved ejection fraction, alongside elevated native T1 and extracellular volume (ECV) consistent with diffuse interstitial fibrosis and chronic remodeling, potentially predisposing to heart failure with preserved ejection fraction (HFpEF) [11]. LGE revealed nonischemic fibrosis in ~ 30% of patients [78], whereas T2 mapping did not reveal active inflammation. This pattern may reflect chronic remodeling in the setting of systemic inflammation and concurrent immunomodulatory therapy. Similar findings have been described in other inflammatory diseases, including RA and systemic lupus erythematosus (SLE) [79, 80].

In contrast, a retrospective study by Goldenberg et al. involving 47 patients with psoriasis found significantly increased septal T2 times compared with controls, suggesting myocardial edema and ongoing inflammation [81]. This difference remained significant after multivariable analysis, consistent with inflammatory myocardial involvement as seen in other cardiomyopathies [82].

However, referral bias and incomplete information on systemic immunomodulatory therapy limit causal interpretation.

6.2 Clinical Implications and Limitations

Despite the limited number of studies and their inherent methodological constraints, the available data suggest that CMR is a promising tool for detecting cardiac remodeling in patients with psoriasis. Findings from both prospective and retrospective studies indicate that myocardial fibrosis and subclinical dysfunction can be identified even in the absence of overt cardiovascular disease, reinforcing the potential role of CMR in risk stratification and disease monitoring.

While these preliminary findings support the potential role of CMR in detecting subclinical myocardial involvement in psoriasis, prospective studies with standardized imaging protocols and well-defined patient cohorts are needed to determine its clinical utility for routine cardiovascular risk stratification and therapy monitoring.

7 Carotid and Femoral Ultrasound

High-frequency carotid ultrasound is an effective noninvasive technique to visualize artery structure and detect early signs of atherosclerosis. When combined with Doppler ultrasound, it allows for the measurement of blood flow velocity to assess arterial function. Cardiovascular risk may be evaluated on the basis of carotid intima-media thickness (CIMT), a marker of early atherosclerosis; degree of stenosis, which predicts stroke risk; and the presence, size, and composition of atherosclerotic plaques. Carotid ultrasound has been widely used in patients with systemic inflammatory diseases, including moderate-to-severe psoriasis, to evaluate cardiovascular risk [83–91].

Patients with moderate-to-severe psoriasis consistently exhibit a significantly increased CIMT compared with healthy controls, with CIMT positively correlating with both disease duration and PASI [86, 87]. Notably, CIMT has been shown to decrease after achieving minimal disease activity with monoclonal antibody treatment [88]. Treatment with IL-17 inhibitors resulted in a significant improvement in CIMT after 6 months, across the carotid, brachial, and femoral arteries [89].

In addition, ultrasound can be employed in clinical research to assess macrovascular endothelial dysfunction via the flow-mediated dilation (FMD) of the brachial artery. Since atherosclerosis is a systemic process, endothelial function may be investigated in various vascular beds.

A study involving 50 patients with psoriatic arthritis who lacked classical cardiovascular risk factors reported significantly impaired flow-mediated dilation on brachial ultrasound [83]. FMD was negatively correlated with CRP and erythrocyte sedimentation rate (ESR), suggesting that chronic low-grade inflammation contributes to atherogenesis [84].

Recently, femoral artery ultrasound has gained attention as a complementary tool to carotid ultrasound for detecting subclinical atherosclerosis [91]. It may enable earlier detection of vascular changes, particularly in young patients with moderate-to-severe psoriasis. Combining carotid and femoral ultrasound could improve cardiovascular risk stratification and enhance detection of coronary artery disease (CAD) compared with carotid ultrasound alone.

However, data on femoral ultrasound in psoriasis remain limited. A study of 70 patients with psoriasis reported a significantly higher prevalence of femoral plaques, even in the absence of an increase in carotid plaques [90]. These findings suggest that femoral ultrasound may be more sensitive for detecting early atherosclerosis in this population [91].

8 Arterial Stiffness and Arterial Tonometry

Arterial stiffness is a validated surrogate marker of subclinical cardiovascular damage and an independent predictor of adverse cardiovascular events [92]. Pulse wave velocity (PWV), particularly carotid-femoral PWV (cf-PWV), is considered the gold standard for assessing aortic stiffness, reflecting the speed at which the pressure wave travels through the arterial tree [93]. In psoriasis, increased PWV has been interpreted as a surrogate of early vascular damage and chronic inflammatory vascular remodeling [94–96].

In a controlled study by Ikonomidis et al., cf-PWV was significantly elevated in patients with psoriasis compared with healthy controls, with comparable values to patients with angiographically proven CAD [20]. These findings were accompanied by elevated circulating levels of IL-6 and malondialdehyde (MDA)—a biomarker of lipid peroxidation and oxidative stress—as well as by impaired CFR and reduced left ventricular longitudinal strain in patients with psoriasis. Notably, the study did not report any significant correlations between cf-PWV and either PASI scores or disease duration.

Gisondi et al. similarly reported significantly higher cf-PWV in 39 patients with moderate-to-severe plaque psoriasis compared with controls, and this difference persisted after adjustment for major cardiovascular risk factors [94]. Notably, psoriasis duration positively correlated with arterial stiffness, while PASI scores showed no significant relationship.

A recent prospective study by Makavos et al. investigated cf-PWV in patients with moderate-to-severe psoriasis undergoing biologic therapy for at least 6 months [95], with parallel assessment of global longitudinal strain (GLS) and 4-year follow-up for MACEs. PWV improved after 6 months of treatment; importantly, this reduction was significant only in patients who remained free from MACE, while those who experienced events did not show a statistically meaningful improvement. Higher PWV was associated with MACE on univariate analysis, but this association lost significance in the multivariable model, likely owing to confounding factors such as hypertension and diabetes, which were both prevalent in the cohort. PWV did not retain additive prognostic value over SCORE2 after adjustment, but its partial improvement post treatment in MACE-free individuals suggests a potential role for biologic therapy in modulating arterial stiffness. Another controlled-study reported improved aortic stiffness in patients with inflammatory arthropathies (rheumatoid arthritis, ankylosing spondylitis, or psoriatic arthritis) with anti-TNF-α therapy [97]. However, it should be noted that these absolute changes are small, and their prognostic significance remains uncertain, as thresholds for clinically meaningful reductions in PWV have not been validated in psoriasis.

Agoglia et al. measured cf-PWV in a cross-sectional cohort and found that approximately one-fifth of patients with psoriasis met a stiffness threshold [96]. Multivariate analysis demonstrated a significant independent association between older age and higher arterial stiffness. Interestingly, greater cumulative methotrexate exposure was associated with significantly lower odds of increased arterial stiffness, suggesting a potential protective cardiovascular effect. No significant associations emerged between elevated cf-PWV and ongoing biological therapy.

Overall, current evidence supports arterial stiffness as an early cardiovascular alteration in psoriasis, although certain aspects remain controversial. The relationship between arterial stiffness and clinical disease severity is conflicting. Furthermore, the effects of treatment for psoriasis on arterial stiffness vary considerably, with biologic therapies showing promise in specific contexts but lacking definitive long-term data [95, 97]. Longitudinal research is required to clarify the precise impact of systemic treatments on vascular remodeling and cardiovascular risk in psoriasis.

Non-invasive techniques such as flow-mediated dilation (FMD) and reactive hyperemia peripheral arterial tonometry (RH-PAT) have been employed to assess endothelial function in psoriasis. However, evidence suggests a limited sensitivity of these modalities in detecting treatment-induced vascular changes [98–101].

In a prospective study of 26 adults with moderate-to-severe psoriasis treated for 8–12 weeks with phototherapy, conventional systemic agents, or biologics, Cohen-Barak et al. observed no significant RH-PAT change despite clinical improvement, with a trend toward worsening in participants with normal baseline indices [98]. Similarly, Nakao et al. found no significant reactive hyperemia index (RHI) variation after two infliximab infusions, although a decline in RHI predicted nonresponse [99]. In a 5-year study on patients with PsA, Ortolan et al. observed stable FMD values despite reductions in CRP and disease activity [100], and Von Stebut et al. noted only modest FMD improvement after 1 year of anti-IL-17A therapy [101]. Overall, while FMD and RH-PAT may detect baseline endothelial dysfunction, their role in monitoring treatment response remains limited owing to low reproducibility and modest responsiveness.

9 Practical Recommendations for Dermatologists

9.1 Risk Models and Psoriasis-Specific Factors

Traditional cardiovascular risk models, such as the Framingham risk score, often underestimate the true burden of CVD in patients with psoriasis. This underestimation is largely due to the systemic inflammation and

Fig. 1 Comparative overview of cardiovascular imaging modalities used to assess subclinical atherosclerosis and inflammation in patients with psoriasis. Each technique presents specific advantages and limitations in terms of accessibility, resolution, cost, radiation exposure, and sensitivity to structural or inflammatory changes. *CCTA* coronary computed tomography angiography, *CMR* cardiac magnetic resonance, *LGE* late gadolinium enhancement, *PET* positron emission tomography, *US* ultrasound

immune dysregulation inherent to the disease, which are not fully accounted for in standard risk calculators. As a result, incorporating imaging to detect subclinical CVD offers a critical opportunity to improve risk stratification and guide more effective medical therapy and disease management in this population. An overview of the strengths and limitations of each imaging modality is shown in Fig. 1 and presented in Table 1.

According to the 2023 ACC/AHA/ASE/ASNC/ASPC/HFSA/HRS/SCAI/SCCT/SCMR/STS Multimodality Appropriate Use Criteria for the detection and risk assessment of chronic coronary disease, CAC scoring is recommended for asymptomatic individuals with an estimated 10-year cardiovascular risk exceeding 7.5% [102]. In such cases, CAC scoring is generally beneficial, with the advantages of imaging outweighing the risks of no evaluation. While not mandatory for all, CAC scoring is considered an appropriate and valuable option for tailoring cardiovascular care, on the basis of clinical judgment and patient preferences.

Current primary prevention guidelines remain non-specific to psoriasis. The 2021 European Society of Cardiology (ESC) guideline formally recommends risk up-adjustment for rheumatoid arthritis but provides no validated numeric modifier for psoriasis [103]; the 2019 ACC/AHA guideline lists chronic inflammatory diseases (including psoriasis) as risk enhancers without quantitative effect sizes [104]. In line with these gaps, imaging data show that both standard and "psoriasis-modified" scores underestimate subclinical atherosclerosis when benchmarked against vascular ultrasound and CCTA in psoriasis cohorts [105].

For individuals at higher risk—specifically those with a 10-year cardiovascular risk greater than 20%—other imaging modalities such as CCTA, stress echocardiography, or stress CMR may be more suitable for comprehensive evaluation.

Table 1 Cardiovascular imaging modalities in moderate-to-severe psoriasis

Imaging modality	Key parameters assessed	Findings in psoriasis	Clinical relevance	Response to psoriasis treatment
Transthoracic Doppler Echocardiography (TTE)	CFR, CFVR, CMD	Reduced CFR/CFVR; linked with PASI, disease duration, PsA [10, 13, 26]	Early CMD detection; prognostic for cardiovascular risk	Significant CFR improvement post anti-TNF/biologic therapy [14, 23]
Coronary Computed Tomography Angiography (CCTA)	Non-calcified plaque burden, CAC, high-risk plaque features	Elevated non-calcified plaques, some CAC elevation; plaque characteristics linked to psoriasis severity [8, 45, 47, 49]	Detects subclinical coronary atherosclerosis; plaque instability risk assessment	Biologic therapy reduces non-calcified plaque and inflammation markers [8, 45, 56]
Positron Emission Tomography (FDG-PET/CT)	Vascular inflammation, PET-PASI score, CMD	Increased FDG uptake in aortic/carotid walls; PASI correlates with vascular inflammation [60–62]	Detects and quantifies vascular inflammation; monitors systemic inflammation	Inflammation reduced post-statin therapy in psoriasis patients [15]; biologic therapy has not shown consistent reductions in vascular FDG uptake
Cardiovascular Magnetic Resonance (CMR)	Myocardial strain, T1/T2 mapping, ECV, LGE	Reduced strain; increased fibrosis (T1/ECV); inconsistent inflammation (T2) findings [11, 81]	Identifies early myocardial changes; useful for detecting cardiac remodeling	Limited data; fibrosis may persist despite therapy [11]
Carotid & Femoral Ultrasound	CIMT, plaque index, FMD%	Increased CIMT and plaque prevalence; linked to PASI and disease duration [86, 87]	Early atherosclerosis detection; potential routine screening tool	CIMT decreases with IL-17 inhibitor therapy [88, 89]

CAC coronary artery calcification, *CCTA* coronary computed tomography angiography, *CFR* coronary flow reserve, *CFVR* coronary flow velocity reserve, *CIMT* carotid intima-media thickness, *CMD* coronary microvascular dysfunction, *CMR* cardiovascular magnetic resonance, *ECV* extracellular volume, *LGE* late gadolinium enhancement, *FDG-PET/CT* fluorodeoxyglucose positron emission tomography/computed tomography, *FMD%* flow-mediated dilation percentage, *IL-17* interleukin-17, *PASI* psoriasis area and severity index, *PsA* psoriatic arthritis, *TNF* tumor necrosis factor, *TTE* transthoracic doppler echocardiography

For example, a 50-year-old white man with treated hypertension (140/80 mmHg), total cholesterol of 240 mg/dL, HDL of 50 mg/dL, and a smoking habit would have an Atherosclerotic Cardiovascular Disease (ASCVD) risk score of approximately 13.3%. Using a psoriasis-adapted approach (mSCORE = SCORE × 1.5, as recommended in AAD–NPF 2019) [106], his adjusted risk could approach or exceed 20%, thereby qualifying for advanced imaging to assess for subclinical or obstructive coronary disease.

Beyond traditional metrics, disease-specific factors such as psoriasis severity and duration may also significantly impact cardiovascular risk. A study assessing the relationship between PASI and CVD outcomes found that each one-point increase in PASI was associated with a 4% increased risk of cardiovascular (CV) events [107]. Stratified risk levels showed: PASI ≤ 5: HR 1.41 (95% CI 0.79–2.53), PASI 5–10: HR 2.23 (95% CI 1.16–4.26), PASI > 10: HR 2.32 (95% CI 1.10–4.89). In addition, another study focusing on CMD in patients with severe psoriasis reported similar findings: each one-point increase in PASI was linked to a 5.8% rise in CMD risk, and each year of disease duration was associated with a 4.6% increased CMD risk [24]. These findings suggest that a PASI ≥ 10 or a disease duration ≥ 10 years corresponds to a 40–60% increased risk of cardiovascular complications. In addition, the presence of psoriatic arthritis appears to further worsen CV risk in patients with psoriasis [108, 109].

Finally, epidemiologic evidence in psoriatic disease (including psoriatic arthritis) indicates that incident heart failure most commonly presents with a preserved-ejection-fraction phenotype [110]. This pattern is consistent with the echocardiographic evidence of diastolic impairment and the CMR evidence of remodeling, while prospective psoriasis-focused studies are needed to quantify HFpEF prevalence.

9.2 Clinical Integration and Future Perspectives

Given this evidence, we recommend that patients with a PASI score greater than 10, disease duration over 10 years, and/or diagnosed psoriatic arthritis may benefit from a comprehensive cardiovascular evaluation. This could include assessments capable of detecting both macrovascular and microvascular dysfunction, such as transthoracic Doppler ultrasound or CCTA, as part of their overall cardiovascular evaluation.

The routine use of imaging assessments as part of primary prevention in asymptomatic patients with severe psoriasis would represent a significant paradigm shift in clinical practice. While the potential cost implications are considerable, they must be carefully weighed against the substantial burden of CVD in this high-risk population and the broader impact on healthcare systems.

Given the rapidly expanding evidence base supporting the ability of imaging, particularly CCTA, to detect key prognostic markers, its integration into the primary prevention pathway could be pivotal in optimizing individualized cardiovascular risk assessment. Upcoming clinical trials and further advancements in technology may help establish its broader role in asymptomatic populations, which would, in turn, bolster its application in high-risk subgroups such as individuals with severe psoriasis.

Furthermore, whether the treatment of psoriasis is definitively associated with a reduction in cardiovascular events requires further investigation. If such treatments are indeed proven to reduce cardiovascular events, a key question arises: can imaging effectively stratify risk to guide appropriate therapeutic interventions for primary prevention in these patients? A growing body of research suggests that employing multimodal imaging to assess subclinical cardiovascular disease in individuals with chronic inflammatory conditions has the potential to offer valuable insights into the progression of inflammatory atherosclerosis [111]. This, in turn, could guide clinical care aimed at mitigating atherosclerotic advancement and ultimately reducing the risk of future cardiovascular events.

Another unresolved issue is whether improvements in flow-based and plaque-level metrics are primarily a consequence of psoriasis clearance or reflect a direct cardioprotective effect of specific biologic agents. If the former is true, therapies that achieve superior skin lesion resolution (typically newer biologics) may also confer greater cardiovascular benefits. Conversely, if a direct cardiotropic mechanism is operative, further investigation into the most effective molecular targets for mitigating cardiovascular inflammation is warranted.

As detailed in section 9.1, guideline positions differ: the 2019 ACC/AHA framework supports selective use of CAC scoring to refine risk in adults at borderline or intermediate estimated risk when treatment decisions are uncertain, whereas the 2021 ESC prevention guideline does not endorse routine imaging-based risk stratification and limits consideration to CAC (or carotid plaque) for risk reclassification in selected individuals.

10 Conclusion

Noninvasive cardiovascular imaging consistently reveals subclinical microvascular dysfunction, high-risk noncalcified coronary plaque, vascular inflammation, myocardial remodeling, and increased arterial stiffness in moderate-to-severe psoriasis. These signals, together with epidemiologic observations in psoriatic disease, support heightened cardiovascular vigilance in dermatology settings. Guideline positions differ, as the 2019 ACC/AHA framework supports

selective use of CAC for risk reclassification, whereas the 2021 ESC prevention guideline does not endorse routine imaging-based risk stratification, so imaging should be applied judiciously and in collaboration with cardiology. A pragmatic approach for dermatologists is a two-step pathway: provide standard cardiovascular risk assessment to all adults with psoriasis; then, for patients flagged by severity (for example PASI ≥ 10), long disease duration (≥ 10 years), psoriatic arthritis, or abnormal screening results, arrange early cardiology referral to jointly determine the next steps. Selective CAC or carotid ultrasound can assist risk reclassification in asymptomatic adults, and targeted CCTA and TTE are appropriate when symptoms are present or when screening identifies a combination of high-risk abnormalities (for example, sustained hypertension plus markedly elevated LDL cholesterol, or glycemic values in the diabetes range). Well-designed prospective studies are needed to determine whether imaging guided care and psoriasis treatments can reduce MACEs and to define actionable thresholds for test selection and follow up.

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Declarations

Conflicts of Interest J.T., M.P., R.M., A.S., C.C., F.T., and S.P. have no conflicts of interest to declare.

Ethics Approval Ethical approval was not required for this study, as it is a review of previously published data.

Consent to Participate Not applicable.

Consent for Publication Not applicable.

Availability of Data and Material No new data were created or analyzed in this study. Data sharing is not applicable to this article.

Code Availability (Software Application or Custom Code) Not applicable.

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