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Short Report

The association of coronary artery disease by coronary CT angiography & cardiovascular outcomes in Psoriatic disease

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1. Introduction

Immune-mediated inflammatory diseases (IMID) are associated with an increased risk of atherosclerotic cardiovascular disease (ASCVD). Psoriasis (PsO), a prevalent chronic autoinflammatory skin condition, is particularly linked to a heightened ASCVD risk [1]. PsO patients face elevated risks of myocardial infarction (MI), stroke, and mortality [2]. Studies have highlighted inflammation as a pivotal driver of cardiovascular risk and severe psoriasis is associated with excess CV risk compared to mild psoriasis [1–4]. Additionally, psoriatic disease is associated with a high rate of cardiometabolic risk factors, and preventative therapies are often underutilized, including statin therapy. Patients with PsO have an increased propensity for all CV risk factors, with the strongest propensity to dyslipidemia [1]. Among an asymptomatic cohort, PsO patients have a greater burden of noncalcified plaque and increased high-risk plaque on coronary computed tomography angiography (CCTA) compared to the general population [3] Few studies have investigated the frequency and severity of CAD in patients with psoriasis, and the impact of plaque burden on CV outcomes among a clinically referred cohort. In addition, the prevalence of statin usage prior to CCTA testing in a clinically referred cohort and the increase in statin prescriptions following CCTA has not been examined.

2. Methods

The analysis involved PsO patients referred for CCTA at Brigham and Women's Hospital and Massachusetts General Hospital (Boston, MA) from 2004–2021. PsO was identified using two ICD-9/10 codes at least 30 days apart, excluding those with prior CAD, end-stage renal disease, or malignant cancer.

Demographic information and clinical data were obtained from the Mass General Brigham (MGB) Research Patient data Registry (RPDR) and Enterprise Data Warehouse (EDW). CCTA was performed using 64-slice or higher multidetector CT scanners. CAD severity was classified

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using the Coronary Artery Disease Reporting and Data System (CAD-RADS): no CAD (CAD-RADS 0), non-obstructive CAD (<50 % stenosis; CAD-RADS 1–2), and obstructive CAD (≥50 % stenosis; CAD-RADS 3–5) and ascertained using a validated natural language processing tool from clinical reports (REF 4) [5]. The ASCVD risk score was calculated according to the 2019 American College of Cardiology/American Heart Association (ACC/AHA) pooled cohort equations [6]. Variables included sex (male/female), race (Black vs. White/other non-Black Hispanic), and age at the time of CCTA. Clinical and laboratory data were obtained within two years before or one year after CCTA were incorporated, including total cholesterol, high-density lipoprotein cholesterol, systolic blood pressure, smoking status, and history of diabetes. Adequate information to calculate ASCVD risk scores was available for 71 % of the cohort (336/473).

The cause of death was determined from ICD-coded data, supplemented by electronic health records. CV risk factors were assessed through ICD codes and ICD/CPT procedural codes. Medication records for lipid-lowering therapies were extracted two years before and one year after CCTA. Psoriasis therapy was ascertained from clinical records based on prescriptions at the time of the CCTA. Full medical therapy is provided in **Supplemental 1.**

The primary outcome was major adverse cardiovascular events (MACE), defined as all-cause mortality, myocardial infarction (MI), or revascularization within 90 days post-CCTA. Cox proportional hazards modeling assessed the association between CAD severity and MACE, adjusting for age, sex, hypertension, and hyperlipidemia.

Numerical baseline characteristics were reported as medians (25th-75th percentiles) and analyzed with Kruskal-Wallis tests, while categorical baseline characteristics were presented as frequencies (percentages) and analyzed with Chi-square tests of association or Fisher's exact tests. When conducting survival analysis, patients were censored at date of querying vital statistics or date of death. In addition, patients not experiencing cardiovascular death were labeled as patients without cardiovascular death. Primary outcome of MACE events was analyzed with hazard rate ratios and 95 % confidence intervals and Kaplan-Meier curves were compared with log-rank tests.

In patients with PsO, univariable and multivariable Cox proportional hazard regressions were analyzed to assess the association of plaque with MACE events (all-cause mortality, myocardial infarction, and late revascularization). For multivariable Cox regression models, multicollinearity was checked with Spearman rank correlation and proportional hazard assumptions was checked with Schoenfeld residuals, as well as being adjusted for the following covariates: age, sex, hypertension, and hyperlipidemia. While primary composite outcome was reported as hazard ratios and 95 % confidence intervals for both unadjusted and adjusted models, CAD (plaque) categories were reported as frequencies and percentages. Two-tailed test with an alpha value of 0.05 were considered statistically significant and analyses were performed using Stata MP Version 18 (StataCorp, College Station, TX).

3. Results

The final cohort consisted of 473 patients (median age 59 [IQR 51–68]), with relatively equal distribution across sex (45.9 % female). CCTA revealed no plaque or stenosis in 128 (27.1 %), non-obstructive CAD in 204 (43.3 %), and obstructive CAD in 140 (29.6) **[Figure 1A]**. Patients with obstructive CAD were older (65 vs 59, $p < 0.001$) and disproportionately male (68.6 %, $p < 0.001$) relative to those with absent or nonobstructive CAD **[Figure 1B].** Cardiometabolic risk factors as shown **[Figure 1B]** were common and higher among PsO patients with obstructive CAD compared with non-obstructive, including hypertension (90.0 % versus 78.1 %), dyslipidemia (85.7 % versus 74.2 %), diabetes mellitus (37.1 % versus 24.4 %), and chronic kidney disease (9.3 % versus 6.3 %).The prevalence of current smoking was significantly higher among patients with obstructive CAD compared with those with absent or non-obstructive CAD (20 % vs 17.6 %, $p = 0.034$). For all patients in our study with an ASCVD risk score, the median (IQR) ASCVD risk score was 2.39 (0.65–8.27). The median scores demonstrated a graded increase across CAD categories: 0.59 (IQR 0.14–1.71) in patients with no plaque, 2.69 (0.90–7.84) in those with non-obstructive CAD, and 5.95 (1.67–14.81) in those with obstructive CAD ($p < 0.001$) **[Figure 1B].**

At baseline, the overall statin usage was 55.0 %, with 27.2 % among patients with absent plaque, 57.9 % in those with non-obstructive plaque, and 73.9 % in those with obstructive plaque **[Figure 1B].** Among the PsO group, 27.5 % of those with non-obstructive plaque and 46.4 % of those with obstructive plaque had an estimated ASCVD risk score above >7.5 %, a threshold often used for prevention-based therapies among the general population **[Figure 1B]**. A total of 8.4 % and 9.9 % of patients with non-obstructive and obstructive plaque, respectively, fell into the borderline (5–7.5 %) risk range. At one year following CCTA, statin prescriptions increased from 57.9 % to 68 % among patients with non-obstructive CAD and 73.9 % to 78.3 % with obstructive CAD ($p < 0.001$). At baseline, systemic immunosuppressive therapy was modest across the cohort, with 8.5 % of patients on biologics, 9.2 % on DMARDS, and 24.5 % receiving steroids. No difference was observed in biologic or steroid use across different plaque categories ($p = 0.178$). Full characteristics and medication details are provided in **Sup 1.**

Over a median follow-up of 5.6 years (IQR 3.6–8.7), those with obstructive CAD experienced the highest incidence of MACE (25 %), followed by those with non-obstructive CAD (8.8 %), and the lowest incidence in patients with absent CAD (5.5 %) **[Figure 1C]**. Patients with obstructive CAD have the highest unadjusted HR 5.73 (95 % CI 2.54–12.90 $p < 0.001$) compared with those with absent CAD. After adjustment for age, sex, hypertension, and hyperlipidemia, patients with obstructive CAD exhibited significantly elevated risk for MACE (aHR= 3.82, 95 % CI 1.52–9.64, $p = 0.004$). Non-obstructive CAD was not significantly associated with MACE in unadjusted (HR 1.99, 95 % CI 0.83–4.78, $p = 0.12$) or adjusted models (aHR 1.51, 95 % CI 0.60–3.81, $p = 0.39$).

4. Discussion

Among patients with PsO referred for CCTA, both non-obstructive and obstructive CAD were prevalent and associated with a stepwise increase in adverse outcomes, including all-cause mortality, myocardial infarction, and late revascularization. Unadjusted and adjusted hazards ratios (aHR) for the composite MACE outcome increased with CAD severity, demonstrating an increasing relationship of risk with plaque burden, although only obstructive plaque remained significantly associated with MACE when adjusting for covariates.

In addition to these primary findings, our study illustrates a critical gap between traditional risk assessment and observed disease burden. ASCVD risk estimations were calculable for 71 % with a median value of low-risk for the overall cohort 2.39 (0.65–8.27). Notably, participants with evidence of coronary plaque also demonstrated overall low estimated 10-year ASCVD risk, generally below guideline-based thresholds for statin initiation. This disconnect underscores a central limitation of

conventional risk calculators chiefly subclinical atherosclerosis in psoriasis may not be adequately captured, contributing to underrecognition of cardiovascular risk.

These observations carry important clinical implications. Psoriasis and other IMIDs are increasingly recognized as cardiovascular "risk enhancers" in the 2019 ACC/AHA prevention guidelines [6]. Yet, in practice, guideline uptake has been incomplete. Statin utilization in our cohort was determined from prescriptions recorded in the Electronic Health Record (EHR), which may be missing or not fully updated. Despite this limitation, only 68 % of patients with non-obstructive plaque were prescribed statins within one-year post-CCTA, highlighting suboptimal implementation of preventive therapy. Together, these findings suggest an imperative for interdisciplinary collaboration, including dermatology, rheumatology, and cardiology to ensure proactive CV screening and timely preventive treatment in this high-risk population.

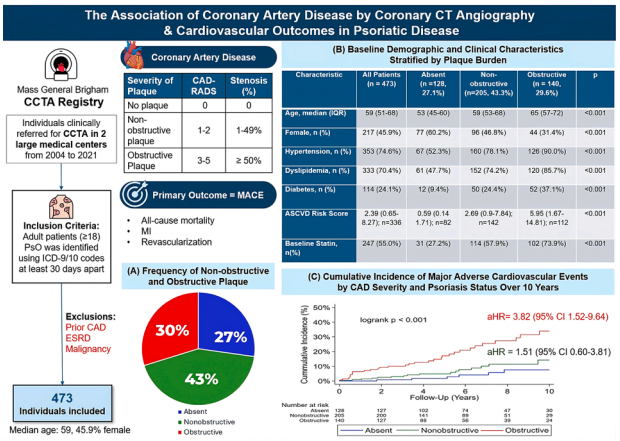
Our study also emphasizes important limitations. First, the modest cohort size and limited number of events constrained our ability to conduct comprehensive risk modeling. Multivariable adjustment was restricted to age, sex, hypertension, and hyperlipidemia to avoid overfitting; other important comorbidities such as diabetes and CKD could not be reliably included and warrant evaluation in larger studies.

Second, dermatologic data were incomplete at our tertiary care center, limiting assessment of PsO severity and disease duration on CV risk. These factors are known to influence CV outcomes [1]. However, we were able to ascertain DMARD, steroid, and biologic therapy, which can be considered a proxy, as immunosuppressive therapy is indicated for moderate or severe disease severity. In contrast, mild disease treatment consists of topical therapy [7]. Future studies should address how disease severity and biological treatments impact coronary plaque burden and CV health.

Finally, additional opportunities exist to apply advanced imaging tools to further elucidate inflammatory drivers of cardiovascular risk. Novel measures such as fat attenuation index (FAI) on CCTA have emerged as noninvasive biomarkers of coronary inflammation and may help clarify the interplay between systemic inflammation, plaque biology, and treatment effects in psoriasis [8]. Previous studies have introduced FAI as a novel imaging biomarker for detecting coronary inflammation via CCTA. Subsequent studies have investigated the potential of FAI as a noninvasive imaging biomarker for assessing coronary inflammation and monitoring therapeutic responses in patients with IMIDs like psoriasis [9,10].

The use of targeted biologic therapy for PsO was modest in this cohort. Investigating whether specific immune-modulating therapies (e.g., anti-IL-23, anti-IL-17, JAKi, or conventional DMARDs) for psoriasis reduce plaque progression, as well as exploring the impact of anti-inflammatory therapies such as colchicine on cardiovascular outcomes in psoriasis, represents a key direction for future research. Moreover, there is an expanding priority for interdisciplinary collaboration to ensure proactive screening and appropriate preventive therapy, highlighted by the rapidly evolving field of cardio-rheumatology [11].

In summary, this study underscores the prognostic importance of CAD severity in psoriasis and highlights the underutilization of preventive therapies, particularly statins, in a population at elevated risk. Our findings demonstrated that coronary atherosclerosis is prevalent among patients with psoriasis referred for coronary CT imaging and that reliance on traditional ASCVD risk calculators may underestimate CV risk in psoriasis, with implications for both risk stratification and preventive treatment. These observations support the growing need for interdisciplinary strategies and tailored preventive approaches to reduce CV morbidity and mortality in patients with IMIDs.



Central Figure: The Association of Coronary Artery Disease by Coronary CT Angiography and Cardiovascular Outcomes in Psoriatic Disease. This figure illustrates the distribution and prognostic impact of coronary artery disease (CAD) severity, as assessed by coronary CT angiography (CCTA) in patients with psoriatic disease (PsO). (A) Pie chart depicting the prevalence of coronary atherosclerosis among patients with PsO: 27 % had no plaque, 43 % had non-obstructive plaque (CAD-RADS 1–2), and 30 % had obstructive plaque (CAD-RADS 3–5). (B) Baseline characteristics stratified by plaque group. (C) Kaplan-Meier curves showing the cumulative incidence of major adverse cardiovascular events (MACE) encompassing all-cause mortality, myocardial infarction, or late revascularization over 10 years. Event rates increased with CAD severity, with obstructive CAD associated with the highest risk (HR = 5.73, 95 % CI: 2.54–12.90), followed by non-obstructive CAD (HR = 1.99, 95 % CI: 0.83–4.78), compared to patients without plaque (log-rank $p < 0.001$). Shown in the figure are the adjusted hazard ratio (age, sex, hypertension, and hyperlipidemia).

Ethical review statement

This work has not been published previously, and it is not under consideration for publication elsewhere. The articles publication has been approved by all authors. If accepted, the article will not be published elsewhere in the same form, in English or in any other language, including electronically, without the written consent of the copyright-holder.

CRediT authorship contribution statement

Jonathan A. Aun: Writing – review & editing, Writing – original draft. **Daniel M. Huck:** Writing – review & editing, Visualization, Supervision, Project administration, Methodology, Funding acquisition, Formal analysis, Conceptualization. **Stephanie A. Besser:** Writing – review & editing, Formal analysis, Data curation. **Arthur Shiyovich:** Writing – review & editing, Investigation, Data curation. **Milena Petranovic:** Investigation, Data curation. **Adam N. Berman:** Writing – review & editing, Investigation, Data curation. **Camila Veronica Blair:** Resources. **Christos P. Kotanidis:** Writing – review & editing, Resources. **Jon Hainer:** Software, Resources. **Dave W. Biery:** Writing – review & editing, Data curation. **Nayruti Trivedi:** Resources. **Khaled Abdelrahman:** Investigation, Data curation. **Rhanderson Cardoso:** Writing – review & editing. **Joseph Merola:** Writing – review & editing. **Michael Garshick:** Writing – review & editing. **Brian Ghoshhajra:** Writing – review & editing, Visualization, Conceptualization. **Sandeep Hegdire:** Writing – review & editing, Visualization, Conceptualization. **Marcelo Di Carli:** Writing – review & editing, Visualization, Conceptualization. **Ron Blankstein:** Writing – review & editing, Visualization, Supervision, Project administration, Investigation, Conceptualization.

Brittany N. Weber: Writing – review & editing, Writing – original draft, Visualization, Supervision, Methodology, Conceptualization.

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Brittany Weber reports a relationship with Novo Nordisk Inc that includes: consulting or advisory. Brittany Weber reports a relationship with Kiniksa Pharmaceuticals Corp that includes: consulting or advisory. Brittany Weber reports a relationship with Bristol Myers Squibb Co that includes: consulting or advisory. Brittany Weber reports a relationship with Horizon Therapeutics USA Inc that includes: consulting or advisory. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.ajpc.2025.101317.

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