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Assessment of Subclinical Coronary Atherosclerosis in Patients with Psoriasis and Psoriatic Arthritis; Do Biologics Play a Role?

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ABSTRACT

Background: Psoriatic arthritis (PsA) is a psoriasis-related inflammatory condition that significantly accelerates the onset of cardiovascular disease (CVD) via systemic inflammation and traditional risk factors. Pro-inflammatory cytokines like TNF and IL-17 are implicated in endothelial dysfunction and atherosclerosis, pointing to the utilization of targeted therapies.

Objective: This study investigates the effect of biologic agents on coronary atherosclerosis and left ventricular function in PsA patients using multislice CT coronary angiography (CCTA) and its correlation with disease activity scores.

Patients and methods: This cross-sectional study recruited 60 asymptomatic psoriatic patients with established PsA, without known cardiac, metabolic, or autoimmune comorbidities. Comprehensive clinical, laboratory, and cardiovascular evaluations, which included speckle tracking echocardiography and multislice CT coronary angiography, were conducted at baseline and after one-year follow-up. Disease severity was measured in terms of PASI, DAPSA, and CASPAR criteria to compare skin/joint involvement with cardiovascular risk.

Results: At baseline, treatment groups were comparable in demographics and disease severity. After one-year, anti-IL17 therapy significantly improved joint disease (DAPSA), skin disease (PASI) and left ventricular systolic function, and reduced coronary plaque burden more than anti-TNF or conventional treatments. Longer biologic treatment duration was independently associated with reduced cardiovascular risk, highlighting the cardio-protective effect of sustained biologic therapy.

Conclusion: Anti-IL17 therapy shows superior benefits in reducing joint disease activity, skin disease, and cardiovascular risk in PsA patients compared to anti-TNF and conventional treatments, supporting its role in comprehensive PsA care.

Keywords: Atherosclerosis, Biologic therapies, Cardiovascular disease (CVD), Psoriatic arthritis (PsA), Systemic inflammation.

INTRODUCTION

PsA is a chronic inflammatory autoimmune disorder strongly linked to psoriasis, a dermatological illness. PsA affects 6-41 % of individuals with psoriasis and is known for its diverse manifestations. A key concern for PsA is its strong link to various comorbidities, particularly CVD. The World Health Organization reports that CVD is responsible for 17.7 million deaths annually, representing 31% of global mortality. Although the precise mechanisms connecting PsA to CVD remain complex, research suggests that the chronic systemic inflammation characteristic of PsA, compounded by traditional cardiovascular risk factors like obesity, diabetes, hypertension, and smoking, significantly elevates the risk for CVD [(1)].

One of the key cardiovascular complications arising from atherosclerosis is increased arterial stiffness, which can impair left ventricular (LV) function. This heightened arterial stiffness, a direct consequence of atherosclerotic plaque buildup, contributes to the worsening of cardiac function and further elevates the risk of CVD in individuals with PsA. The interplay between systemic inflammation, atherosclerosis, and cardiovascular health underscores the importance of addressing inflammatory pathways in PsA to mitigate the long-term cardiovascular risks faced by these patients [(2,3)].

Given this context, understanding the effects of biologic therapies, particularly those targeting TNF and IL-17, becomes crucial not only for managing joint and skin symptoms of PsA but also for potentially reducing the cardiovascular burden associated with the disease [(4)].

Biologic treatments have transformed the management of mild to severe psoriasis, may offer not only improvements in skin health but also potential benefits for cardiovascular health. Specifically, anti-IL17 and anti-TNF therapies have been shown to significantly reduce systemic inflammation, a key driver of both psoriasis symptoms and cardiovascular risk [(5)]. Despite the promising effects of these therapies on psoriasis, their impact on subclinical atherosclerosis and cardiovascular outcomes remains an area of ongoing investigations. It is essential to understand whether these biologics can offer long-term cardiovascular protection, thereby reducing the burden of coronary artery disease in psoriasis patients [(6)].

In the present study, we assessed the impact of biologic therapies (Anti-IL17, Anti-TNF) and conventional treatments on coronary atherosclerosis in psoriatic patients. The study aimed to evaluate whether biologics could provide a stabilizing effect on coronary plaque and vascular health. This was accomplished using coronary computed tomography angiography (CCTA) to measure coronary calcium score (CCS),

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total plaque burden, and non-calcified plaque burden before and after one year of therapy. Furthermore, we examined how these imaging findings correlate with disease severity measures, such as PASI and DAPSA scores, to better understand the relationship between joint and skin disease activity and cardiovascular outcomes.

PATIENT AND METHODS

This cross-sectional study included 60 asymptomatic PsA patients who were recruited from the Rheumatology Department of Menoufia University Hospitals during the period (from March 2023 to April 2024). These participants had no prior history of cardiac issues.

To participate in the study, patients had to meet specific inclusion criteria. First, they were diagnosed with psoriasis by a dermatologist according to Psoriasis Area and Severity Index (PASI), and PsA diagnosis was made by a rheumatologist according to Psoriatic Arthritis (CASPAR) criteria. Participants were required to be over the age of 18 with normal liver and kidney functions.

Several exclusion criteria were applied to ensure the study's relevance and safety. Individuals with known hypertension or diabetes mellitus were excluded. Similarly, patients with a history of congenital heart disease, atrial fibrillation, or ischemic heart disease were not included in the study. Those with malignancies, chronic diseases, or other autoimmune conditions were also excluded. Finally, patients who were uncooperative or refused to sign the informed consent were not enrolled.

As part of the study, all participants underwent a series of procedures to gather comprehensive data about their health and condition.

First, a detailed medical history was collected for each participant, which included information such as the patient's name, age, gender, smoking habits, chronic conditions, and current medications. This was essential to establish a baseline for their overall health.

Next, a general examination was conducted, focusing primarily on the skin and nails. The goal was to identify any lesions and confirm the clinical diagnosis of psoriasis, ensuring that the condition was appropriately diagnosed and assessed.

In addition to the general examination, a local examination was carried out, which included two primary systems. The locomotor system was evaluated by assessing the tender joint count (TJC) and swollen joint count (SJC), which helped determine the level of joint involvement in PsA. A cardiovascular system examination was also performed to assess any potential cardiovascular issues.

To further evaluate the severity of the conditions, several scores were used. The diagnosis of PsA was made according to the Classification of Psoriatic Arthritis (CASPAR) criteria [(7)]. The severity of psoriasis was quantified using the Psoriasis Area and Severity Index (PASI) [(8)]. While, the disease activity of PsA was measured using the Disease Activity Index for PsA (DAPSA) [(8)].

Laboratory investigations were also performed to gain deeper insights into the participants' health. A Complete Blood Count (CBC) was conducted to assess their overall health status. Additionally, the Estimated Sedimentation Rate (ESR) was measured using the Westergren method, and C-Reactive Protein (CRP) levels were analyzed as part of the inflammatory assessment to better understand the degree of inflammation in each participant.

A conventional echocardiographic Doppler study, along with tissue Doppler imaging and 2D-speckle tracking imaging, was conducted using the Vivid 9 system from General Electric Healthcare (GE Vingmed, Norway), which was outfitted with a harmonic M5S variable frequency (1.7–4 MHz) phased-array transducer, in accordance with the guidelines of the American Society of Echocardiography [(9)], and linked to a single lead ECG. Tissue Doppler imaging was performed in the apical four-chamber view, where the average Em velocity was assessed and the E/Em ratio was computed. Two-dimensional strain analysis was conducted offline utilizing the Echopac program (General Electric version 1.8.1.X-Vingmed). At baseline and one year later, all strain pictures were acquired and digitally saved on a hard drive for offline analysis and global left ventricular longitudinal strain calculation.

Lastly, to assess coronary atherosclerotic burden, all participants underwent multislice CT coronary angiography using the same CT scanner (320-detector row Aquilion ONE ViSION, Toshiba, Japan). Prospective EKG gating, 100 or 120 kV tube potential, tube currents ranging from 100 to 850 mA, adjusted for the patient's body size, and a gantry rotation duration of 275 ms were all used throughout the scans. With a 0.25 mm slice increment, images were obtained at a slice thickness of 0.5 mm. Every scan was read while blindfolded. Using specialized software (QAngio CT, Medis; The Netherlands), the features of coronary plaque were examined throughout all major coronary arteries that were larger than 2 mm [(10)]. A plaque index was produced by dividing the plaque volume (in cubic millimeters) by the appropriate segment length (in millimeters) to account for varied coronary artery lengths. The sum of the calcified and non-calcified plaque burdens was used to determine the total plaque load.

Ethical approval:

Menoufia Faculty of Medicine Ethics Committee authorized this study. Prior to inclusion in the trial, each participating provided informed permission. The Helsinki Declaration was followed throughout the course of the study.

Statistical analysis

The statistical program SPSS version 24.0 was used for the statistical tests. Continuous variables were

subjected to a normality test; normally distributed variables were published as mean ± SD and compared using a one-way ANOVA (F-test); non-normally distributed variables were reported as median with IQR and compared using the Mann-Whitney U test when two groups were being compared or the Kruskal-Wallis test. Using the X^2-test, categorical variables were compared and displayed as percentages and frequencies. The clinical and imaging characteristics were evaluated using the Spearman correlation coefficient. Multivariate linear regression analysis was used to identify independent predictors of CAC and total plaque load during a one-year follow-up. A p-value of <0.05 indicated statistical significance.

RESULTS

Table 1 compares the clinical characteristics across three treatment groups: Anti-IL17, Anti-TNF, and Conventional. No significant differences were found in age, sex, disease duration, smoking, axial involvement, or skin involvement. However, peripheral involvement was significantly higher in the Anti-TNF group (100%) compared to the others (90% for anti-IL17 and 65% for Conventional). Mixed involvement, palm/sole affection, scalp affection, nail affection, and sacroiliitis showed no significant differences between the groups. Overall, peripheral involvement notably distinguished the Anti-TNF group, while other characteristics remain similar across the treatments.

Table (1): Demographic and baseline characteristics by treatment group

Variable	Anti-IL17 (n=20)	Anti-TNF (n=20)	Conventional (n=20)	Test	p-value
Age (yrs) Mean ±SD	38.05 ± 14.48	41.65 ± 13.79	49.25 ± 12.46	F-test 0.682	0.190
Sex				$\chi2$ 0.144	0.931
- Male	8 (40%)	7 (35%)	7 (35%)		
- Female	12 (60%)	13 (65%)	13 (65%)		
Disease duration (years) Median (IQR)	7(10.75-5-25)	7(11.5-3.25)	7(10-4.5)	H-test 0.330	0.848
Smoking	5 (25%)	2 (10%)	3 (15%)	$\chi2$ 1.680	0.432
Axial involvement	10(50%)	8(40%)	10(50%)	$\chi2$ 0.536	0.765
Peripheral involvement	17 (85%)	18 (90%)	16 (80%)	$\chi2$ 1.200	0.549
Mixed involvement	8(40%)	8(40%)	3(15%	$\chi2$ 3.851	0.146
Site of skin affected				$\chi2$ 0.141	0.932
- Extremity	12 (60%)	13 (65%)	12 (60%)		
- Axial extremities	8 (40%)	7 (35%)	8 (40%)		
Palm/sole affection	6 (30%)	4 (20%)	7 (35%)	$\chi2$ 1.149	0.563
Scalp affection	5 (25%)	7 (35%)	6 (30%)	$\chi2$ 0.476	0.788
Nail affection	6 (30%)	7 (35%)	8 (40%)	$\chi2$ 0.440	0.803
Sacroiliitis	10 (50%)	8 (40%)	7 (35%)	$\chi2$ 0.960	0.619

*: Statistically significant, SD: Standard deviation, $\chi2$: Chi-squared Test, F: One-way ANOVA test, H-test: Kruskal-Wallis test is a non-parametric statistical test.

Table 2: The laboratory markers showed no statistically significant differences across Anti-IL17, Anti-TNF, and Conventional treatment groups, with all p-values exceeding 0.05. Median values and IQRs were closely aligned, indicating comparable profiles in inflammation, hematologic status, and lipid levels. This suggests similar systemic effects among the therapies. The results may support the therapeutic equivalence in terms of lab safety parameters.

Table (2): Laboratory parameters at base line

Lab Marker	Anti-IL17 (Median/IQR)	Anti-TNF (Median/IQR)	Conventional (Median/IQR)	TEST	p-value
ESR (mm/hr)	25 (18, 40)	27 (19, 41)	26 (17, 39)	U	P1 = 0.662, P2 = 0.931, P3 = 0.743
CRP (mg/L)	10 (5, 14)	11 (6, 15)	9 (4, 13)	U	P1 = 0.219, P2 = 0.616, P3 = 0.148
Hemoglobin (g/dL)	12.4 (11.9, 13.15)	12.3 (11.8, 13.2)	12.55 (11.75, 13.4)	U	P1 = 0.645, P2 = 0.978, P3 = 0.674
WBC (/mcL)	7.8 (6.5, 9.8)	7.5 (6.4, 9.7)	7.3 (6.2, 9.5)	U	P1 = 0.519, P2 = 0.271, P3 = 0.437
Platelets (/mcL)	250 (230, 290)	248 (225, 285)	245 (220, 280)	U	P1 = 0.893, P2 = 0.207, P3 = 0.341
Total Cholesterol (mg/dL)	252 (240, 270)	255 (242, 275)	250 (235, 265)	U	P1 = 0.659, P2 = 0.423, P3 = 0.378
LDL (mg/dL)	145 (130, 160)	147 (132, 162)	144 (130, 158)	U	P1 = 0.751, P2 = 0.862, P3 = 0.609
HDL (mg/dL)	40 (34, 47)	39 (33, 46)	40 (33.5, 47)	U	P1 = 0.897, P2 = 0.872, P3 = 0.984
Triglycerides (mg/dL)	172 (159.5, 185)	170.5 (162, 185)	170 (161, 182)	U	P1 = 0.480, P2 = 0.924, P3 = 0.560

Median and range: non-parametric test. U=Mann-Whitney U test *=significant, P1=value between Anti-IL17 group and Anti-TNF group, P2= p-value between Anti-IL17 group and Conventional group, P3=p-value between Anti-TNF group and Conventional group.

Table 3: PASI scores show slightly higher medians in the biologic groups (Anti-IL17 and Anti-TNF) compared to the conventional group, but with no statistically significant differences, indicating comparable skin disease burden pre-treatment. DAPSA scores also appear uniformly elevated across groups, with medians ranging from 32.00 to 35.00 and all p-values > 0.05, suggesting no significant difference in psoriatic arthritis activity at baseline. The distribution of DAPSA activity levels (moderate vs. high) further supports this, showing a similar pattern across the three groups.

Table (3): Disease Severity Scores (PASI and DAPSA) – before treatment

Score	Time point	Anti-IL17 (n=20)	Anti-TNF (n=20)	Conventional (n=20)	Test	P value
PASI	**Before Treatment**	11.20 (IQR: 40.80 − 8.10)	11.25 (IQR: 19.85 − 7.40)	8.40 (IQR: 14.15 − 3.45)	U	P1 = 0.620 P2 = 0.096 P3 = 0.327
DAPSA	**Baseline**	32.00 (IQR: 28.00 − 37.00)	33.50 (IQR: 29.00 − 38.00)	35 (IQR: 27.00 – 36.00)	U	P1 = 0.642 P2 = 0.708 P3 = 0.561
DAPSA Activity Level	**Baseline**	12 Moderate / 8 High	14 Moderate / 6 High	15 Moderate / 5 High	$\chi 2$ 1.078	0.583

*=significant, P1=value between Anti-IL17 group and Anti-TNF group, P2= p-value between Anti-IL17 group and Conventional group, P3=p-value between Anti-TNF group and Conventional group.

Table 4: At 1-year follow-up, PASI and DAPSA scores improved most with anti-IL17, followed by anti-TNF, and worst with conventional therapy, and all between-group comparisons were statistically significant. The median PASI scores were best in the Anti-IL17 group (6.5) and worst in the Conventional group (13.8). DAPSA scores showed a similar positive trend with greater numbers of patients with remission or low disease activity in the Anti-IL17 group. These results highlight the high efficacy of anti-IL17 therapy in improving skin and joint outcomes.

Table (4): Disease Severity Scores (PASI and DAPSA) – 1-year follow-up

Score	Time point	Anti-IL17 (n=20)	Anti-TNF (n=20)	Conventional (n=20)	test	p-value
PASI	1-Year Follow-up	6.50 (IQR: 5.00 – 8.00)	9.20 (IQR: 7.50 – 11.00)	13.80 (IQR: 11.00 – 18.50)	U	P1 = 0.004* P2 = 0.018* P3 = 0.002*
DAPSA	1-Year Follow-up	9.5 (IQR: 11 − 8= 3)	11.5 (IQR: 12,5 − 9 = 3.5)	13.1 (IQR: 13.7 – 12.6 = 1.1)	U	P1 = 0.010* P2 = 0.000* P3 = 0.000*
DAPSA Activity Level	1-Year Follow-up	8 Remission/12 Low	5 Remission/15 Low	4 Remission/16 Low	$\chi 2$ 2.134	0.344

*=significant, P1=value between Anti-IL17 group and Anti-TNF group, P2= p-value between Anti-IL17 group and Conventional group, P3=p-value between Anti-TNF group and Conventional group.

Table 5 presents a summary of CCTA findings by treatment groups at baseline and 1-year follow-up. At baseline, the coronary features of the three groups were identical. When groups' plaque features were examined over a one-year period, the biologic-treated group's non-calcified plaque load decreased significantly more than that of the non-biologic-treated group. At 1 year, both Anti-IL17 and Anti-TNF groups had lower coronary calcium scores, total plaque burden, and non-calcified plaque burden compared to the conventional group. Anti-IL17 therapy considerably reduced the load of coronary plaque more than anti-TNF therapy did.

Table (5): Coronary CT angiography (CCTA) findings by group

CCTA Variable	Time point	Anti-IL17	Anti-TNF	Conventional	Test	p-value
Coronary Calcium Score	**Baseline**	314.0±69.04	322.0±60.09	324.4±57.55	F-test	P1 = 0.914 P2 = 0.859 P3 = 0.992
	1-Year Follow-up	25.5±13.6	93.00±9.23381	298.2±61.79	F-test	P1 = 0.009* P2 = 0.000* P3 =0.000*
Total Plaque Burden	**Baseline**	1.3800±0.35482	1.5280±0.3468	1.4500±0.36491	F-test	P1 = 0.392 P2 = 0.808 P3 =0.768
	1-Year Follow-up	0.8240±0.2924	1.1920±0.3257	1.7490±0.22107	F-test	P1 =0.005* P2 = 0.000* P3 =0.000*
Non-Calcified Plaque Burden	**Baseline**	1.2990±0.3508	1.2350±0.4484	1.2385±0.42759	F-test	P1 =0.695 P2 = 0.888 P3 =0.935
	1-Year Follow-up	0.730±0.44844	0.9890±0.3647	1.6275±0.28338	F-test	P1 = 0.004* P2 = 0.001* P3 =0.000*

*=significant, P1=value between Anti-IL17 group and Anti-TNF group, P2= p-value between Anti-IL17 group and Conventional group, P3=p-value between Anti-TNF group and Conventional group.

Table 6 shows that at one year, patients treated with biologic therapy had significant improvement of LV systolic (GLS) and diastolic functions (average E/E') than conventionally treated patients. Moreover, the Anti-IL17 group showed statistically significant difference in both parameters in comparison to the Anti-TNF treated group.

Table (6): Global longitudinal strain (GLS) and average E/E') by group

Parameter	Timepoint	Anti-IL17	Anti-TNF	Conventional	Test	p-value
GLS (- ve values)	Baseline	18.40± 1.95	18.60± 1.98	19.11± 1.843	F-test	P1 = 0.943 P2 = 0.498 P3 =0.696
	1-Year Follow-up	21.40±2.11262	19.90±1.74416	18.20±1.908	F-test	P1 = 0.004* P2 = 0.000* P3 =0.019*
Average E/E' by DTI	Baseline	12.3940±1.168	13.61± 1.26	12.33±1.057	F-test	P1 = 0.817 P2 = 0.985 P3 =0.732
	1-Year Follow-up	6.3155±0.993	9.1070±0.86102	15.8250±0.495	F-test	P1 = 0.000* P2 = 0.000* P3 =0.000*

*=significant, P1=value between Anti-IL17 group and Anti-TNF group, P2= p-value between Anti-IL17 group and Conventional group, P3=p-value between Anti-TNF group and Conventional group.

Table 7 highlights key correlations between clinical variables and cardiovascular imaging outcomes. Age shows a modest but significant link to increased CAC and plaque burden, while disease duration does not. Inflammatory markers (CRP, ESR) negatively correlate with CAC, suggesting a complex or inverse relationship. PASI and DAPSA scores are largely not significant, though higher DAPSA is positively associated with non-calcified plaque burden, pointing to a connection between joint activity and early atherosclerosis. Importantly, longer biologic therapy duration is strongly linked to reduced CAC and plaque burden, suggesting a cardio protective effect of sustained biologic treatment.

Table (7): Correlation between atherosclerosis and clinical/biological variables

Independent Variable	CAC Score (r, p)		Plaque Burden (r, p)		Non-Calcified Burden (r, p)	
Age	0.329	0.010*****	0.315	0.014*****	0.389	0.002*****
Disease Duration	-0.016	0.903	-0.071	0.590	-0.086	0.516
CRP	-0.288	0.026*****	-0.121	0.356	-0.069	0.602
ESR	-0.248	0.056	-0.249	0.055	-0.114	0.386
PASI Scores	-0.022	0.868	0.003	0.981	-0.047	0.722
DAPSA Scores	-0.058	0.658	0.036	0.783	0.378	0.003*****
Lipid Parameters	0.013	0.922	0.038	0.773	0.141	0.283
Biological Duration	-0.712	0.000*****	-0.523	0.000*****	-0.231	0.081

R: Correlation coefficient, *: Statistically significant.

The multivariate regression analysis identifies key factors influencing 1-year post-treatment coronary artery calcification (CAC) scores. Age and LDL levels are positively associated with higher CAC scores, while biological treatment duration was negatively associated, suggesting a protective effect. Longer biological treatment duration notably reduces CAC progression. Other factors like CRP, disease duration, PASI after treatment, DAPSA at 1 year, HDL, and triglycerides were not significant predictors in this model **(Table 8)**.

Table (8): Multivariate regression for predictors of 1-year post treatment of CAC Score

Predictor	Beta Coefficient	p-value	95% CI
Age	2.275	0.020*****	[0.374, 4.177]
Disease duration	1.691	0.519	[-3.569, 6.952]
CRP	0.062	0.940	[-1.572, 1.695]
PASI after treatment	-1.696	0.127	[-3.899, 0.506]
DAPSA post 1 year	0.117	0.070	[-0.010, 0.244]
LDL	8.052	0.006*****	[2.490, 13.614]
HDL	-0.681	0.838	[-7.373, 6.011]
Triglyceride	-0.828	0.677	[-4.817, 3.161]
Biological duration	-20.495	0.000*****	[-27.595, -13.396]

***: Statistically significant.**

In the multivariate regression analysis, three variables were found to be statistically significant predictors of total plaque burden at 1-year post-treatment: longer biologic therapy duration was associated with reduced plaque burden ($\beta = -0.052$), while higher LDL levels ($\beta = 0.037$) and higher DAPSA scores at 1 year ($\beta = 0.001$) were linked to increased plaque burden. Additionally, PASI after treatment ($\beta = -0.009$) and total cholesterol ($\beta = -0.020$) showed borderline significance, **Table 9**.

Table (9): Multivariate regression for predictors of 1-year post treatment of total plaque burden

Predictor	Beta Coefficient	p-value	95% CI
Age	0.005	0.254	[-0.004, 0.014]
Disease duration	-0.008	0.524	[-0.031, 0.016]
CRP	0.003	0.461	[-0.005, 0.010]
PASI after treatment	-0.009	0.086	[-0.019, 0.001]
DAPSA. post. 1 year	0.001	0.030	[0.000, 0.001]
Total cholesterol	-0.020	0.091	[-0.043, 0.003]
LDL	0.037	0.005	[0.012, 0.062]
HDL	0.024	0.117	[-0.006, 0.054]
Triglyceride	-0.009	0.303	[-0.027, 0.009]
Biologic. duration	-0.052	0.002	[-0.084, -0.020]

DISCUSSION

PsA, an inflammatory autoimmune disease linked to psoriasis, is a significant source of CVD risk due to chronic systemic inflammation induced by cytokines like TNF and IL-17. This inflammation accounts for atherosclerosis and arterial stiffness that worsen cardiac function. Biologic therapies targeting these cytokines can possibly reduce both PsA symptoms and cardiovascular risks, with studies now evaluating their impact on coronary plaque and vascular health via imaging studies like CCTA [(11)].

All three treatment groups in our study (Anti-IL17, Anti-TNF, Conventional) were demographically similar, except for significantly higher peripheral joint involvement in the Anti-TNF group. Inflammatory markers (CRP, ESR, WBC, platelets) were significantly more elevated in Anti-TNF users compared to others**.**

Baseline PASI and DAPSA scores were comparable among groups, with DAPSA higher in the Anti-TNF group. After one year, Anti-IL17 therapy showed the greatest improvement both in PASI score and in DAPSA (Remission in 40% of patients), significantly outperforming Anti-TNF and Conventional treatments.

The improvement in PASI score in the current study coincides with the study of **Kim and Krueger [(12)]** who demonstrated significant improvements in psoriatic skin manifestations in patients treated with interleukin 17A inhibitors or IL-23 inhibitor

In agreement with our finding **Ogdie *et al.* [(13)]** demonstrated that patients receiving secukinumab (an IL-17A inhibitor) achieved higher rates of DAPSA response compared to placebo. At week 12, higher proportions of patients receiving secukinumab 300 mg or 150 mg achieved DAPSA50 (65.2% and 44.4%, respectively) and DAPSA low disease activity or DAPSA-based remission (82.6% and 78.6%, respectively) compared to placebo (30.0% and 56.7%, respectively). These improvements were either sustained or enhanced further through week 52, with secukinumab 300 mg showing better results

Similarly, **Gao *et al.* [(14)]** in a systematic review and meta-analysis of randomized controlled trials reported that IL-17 inhibitors were more effective than TNF inhibitors in achieving higher rates of ACR70 responses. However, the study did not find significant advantages in ACR20 and ACR50 responses when comparing IL-17 inhibitors to adalimumab.

However, **Izumiyama *et al.* [(15)]** compared IL-17A inhibitors and TNF inhibitors in Japanese patients with PsA, found no significant difference in DAPSA improvement between both groups at weeks 12, 24, and 52. Besides, PASI scores did not show significant differences, indicating that both therapies had comparable effects on skin involvement.

In target trial emulation research, **Stisen *et al.* [(16)]** reported that in patients with PsA, TNF inhibitors, IL-17 inhibitors, and IL-23(p19) inhibitors were all likely considered to be successful treatments. The study found comparable response rates and clinical outcomes for the biologic treatments.

At baseline, coronary artery disease burden (CAC and plaque) was similar across our study groups. After one year, Anti-IL17 and Anti-TNF therapies significantly reduced CAC and plaque burden, while Conventional therapy showed worsening. Anti-IL17 also led to the most marked improvement in cardiac function (GLS and DTI E').

Shang *et al.* [(17)] found that even in those without cardiovascular risk factors, PsA patients frequently had subclinical poor myocardium deformation. The degree of PsA disease activity was correlated with apical rotation. Subclinical myocardial involvement in PsA can be identified using these novel speckle tracking echocardiography methods.

In agreement with our finding, **Tsiogka *et al.* [(18)]** evaluated coronary plaque morphologies using CCTA in biologic-naïve individuals treated with TNFα, IL-12/23, or IL-17 inhibitors in comparison to those receiving topical or light treatments. Patients receiving anti-IL-17 treatment saw a 12% decrease in non-calcified plaque load after a year ($p<0.001$), which was noticeably higher than the decreases seen in the anti-IL-12/23 and non-biologic groups.

In **Elnabawi *et al.* [(19)]** study involved 290 participants, biologic treatment (including anti-TNF) was related with a six percent reduction in non-calcified plaque burden ($p=0.005$) and a decrease in the necrotic core ($p=0.03$), compared to slow plaque development in non-biologically treated groups.

Consistent with our finding, **Frieder *et al.* [(20)]** study assessing cardiac function through various

parameters found that anti-IL-17 treatment produced the most significant results. Specifically, secukinumab, an anti-IL-17 agent, showed a 14% improvement in global longitudinal strain (GLS), a 41% improvement in GLS rate, and a 19% improvement in left ventricular twist compared to conventional therapies.

We found that Age and LDL were positively associated with CAC and plaque burden. Higher DAPSA scores predicted greater non-calcified plaque. Importantly, longer biologic therapy duration was strongly and independently associated with reduced CAC and plaque burden, confirming a potential cardio-protective role.

Similarly, **Garg *et al.*** [21] found that in PsA patients, CAC has been linked to greater LDL cholesterol levels and older age.

Higher disease activity, as measured by **Eder *et al.*** [22] study found the DAPSA, has been linked to more severe atherosclerosis in PsA patients. An observational study reported that increased inflammation over time, indicated by higher DAPSA scores, was associated with more extensive atherosclerotic plaques. However, this association was not significant after adjusting for traditional cardiovascular risk factors.

Longer duration of biologic therapy has been shown to reduce CAC and plaque burden in PsA patients. **Elnabawi *et al.*** [19] study demonstrated that regardless of conventional cardiovascular risk factors, biologic treatment was linked to a 6% drop in the burden of non-calcified plaque and a decrease in the necrotic core area. This suggests a potential cardioprotective role of biologic treatments in PsA.

CONCLUSION

Our study highlights the superior efficacy of Anti-IL17 therapy in improving skin, joint disease activity and reducing cardiovascular risk in PsA patients compared to Anti-TNF and conventional treatments. These findings support the use of targeted biologics, particularly IL-17 inhibitors, in comprehensive PsA management.

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