ORIGINAL ARTICLE

Frequency and Clinical Association of Anti-CCP Positivity in Patients with Psoriatic Arthritis (PsA) and its Significance in Skeletal Involvement

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ABSTRACT

Objectives: To determine the prevalence of anti-CCP antibodies in patients with PsA and evaluate their association with clinical features.

Methodology: A cross-sectional study was conducted in the Department of Rheumatology, Liaquat National Hospital, Karachi. Sixty-one (61) PsA patients fulfilling CASPAR criteria were enrolled. Clinical assessments included joint distribution, axial/peripheral involvement, and skin manifestations. Anti CCP measured using ELISA Kits (cut-off value: <17 U/mL), X-rays, MRI, or CT scans were utilized where available, as part of routine clinical evaluation to characterize the pattern of skeletal involvement and were considered in conjunction with clinical findings to support the overall diagnosis. Associations between anti-CCP positivity and clinical features were analyzed using chi-square tests and logistic regression.

Results: Anti-CCP antibodies were detected in 31.1% of PsA patients. Anti-CCP positivity was significantly associated with the absence of skin involvement (p 0.008) and older age (p=0.025). No significant associations were observed between anti-CCP status and patterns of joint involvement, axial or peripheral disease. Logistic regression demonstrated significantly lower odds of anti-CCP positivity in patients with skin involvement (OR = 0.06, 95% CI: 0.006–0.582, p = 0.015).

Conclusion: A substantial proportion of PsA patients in this cohort tested positive for anti-CCP antibodies. Anti-CCP positivity may represent a distinct clinical subset in PsA and was associated with older age and an inverse relationship with skin involvement. It may also be associated with a distinct clinical pattern of musculoskeletal involvement. These findings highlight the potential role of anti-CCP antibodies in the clinical assessment of PsA and support their use in guiding diagnostic and therapeutic considerations.

Keywords: Anti-CCP antibodies, diagnostic biomarkers, joint involvement, psoriatic arthritis, rheumatoid arthritis, Pakistan

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INTRODUCTION

Psoriatic arthritis (PsA) is a chronic inflammatory disease in which arthritis is associated in most cases with psoriasis. The biological and clinical spectrum of PsA may present common elements with rheumatoid arthritis (RA; e.g., symmetrical arthritis of the hands, elevated acute phase proteins) or with the general class of spondyloarthropathies (e.g., dactylitis, enthesitis, sacroiliitis). Unfortunately, there is no specific serologic test for PsA. Rheumatoid factor (RF) contributed to the designation of PsA as an independent nosological entity, in the sense that patients with arthritis and psoriasis were usually seronegative for RF, differentiating them from RA patients, who are usually seropositive for RF, but its low specificity for RA motivated the search for a more reliable serologic test. Anti-cyclic citrullinated peptide antibodies (anti-CCP) met the demands: they proved a similar sensibility for RA (55-80%) but a higher specificity (96-98%)[1,2]. Anti-CCP antibodies have been detected in the early phase of RA and have been associated with severe radiological damage. A clinical prediction model, discriminating between self-limiting persistent non-

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erosive and persistent erosive arthritis, also includes the evaluation of anti-CCP antibodies[3]. Interestingly, citrullinated fibrin has been identified as one of the major citrullinated proteins in RA synovium, and anti-filaggrin, antikeratin antibodies, or anti-CCP antibodies have been detected in the synovial fluid (SF) of RA patients. Despite reports of the high specificity of the anti-CCP test, these antibodies have recently been detected in the serum of patients with psoriatic arthritis (PsA), suggesting relevant considerations about the correct diagnosis of this disease[4]. Anti-CCP antibodies are mainly produced in the synovium by the local plasma cells and are designed to bind to citrulline-containing antigenic determinants of synovial proteins. The enzyme peptidyl-arginine-deiminase generates citrulline residues by acting on the normal arginine residues[5].

In clinical practice, the titer of antiCCP antibodies is determined by an enzyme-linked immunosorbent assay (ELISA), using synthetic citrullinated peptides. The detection of anti-CCP antibodies may precede by several years the clinical onset of RA, for which they have a high positive predictive value and a strong association with female gender, disease activity, functional impairment, and erosive disease[6]. The studies that evaluated anti-CCP antibodies in PsA patients, reported a prevalence of 5.6-20%[7,8]. While PsA has been clinically studied in Pakistan, data on anti-CCP antibodies in this context remain scarce. A study at a tertiary care center reported 46% joint involvement in 100 psoriatic patients using Moll and Wright criteria[9]. Another study found a 46.4% PsA prevalence among 140 patients, all seronegative for RF, without assessing anti-CCP status[10].

Treatment-focused research has emphasized GRAPPA guidelines and individualized therapy, yet has overlooked anti-CCP antibodies. This gap highlights the need for evaluating anti-CCP antibodies to enhance diagnostic precision and aid differentiation of PsA from seronegative or atypical RA presentations, where clinical overlap can complicate diagnosis. While anti-CCP antibodies are highly specific for RA, their presence in PsA patients suggests potential diagnostic value.

In Pakistan, despite a growing body of clinical research on PsA, the prevalence and significance of anti-CCP antibodies in these patients remain largely uninvestigated. This study aims to address this gap by evaluating the presence of anti-CCP antibodies in PsA patients, thereby contributing to improved diagnostic accuracy and better-informed clinical decision-making in local rheumatology practice.

METHODOLOGY

IRB/ERC Approval: This cross-sectional study was conducted in the Department of Rheumatology, Liaquat National Hospital, Karachi, from December 2024 to June 2025. The study was approved by the Ethical Review Committee of Liaquat National Hospital (App#1122-2024-LNH-ERC), dated 4th December 2024.

The procedure was explained to each participant and informed consent was taken. Detailed clinical examinations were conducted to assess joint involvement, enthesitis, and psoriasis severity. Blood samples (1 to 5 milliliters (mL) per sample) were collected to determine aCCP positivity . Anti CCP was measured using ELISA Kits (cut-off value: <17 U/mL), X-rays, MRI, or CT scans where available, as part of routine clinical evaluation, , to characterize the pattern of skeletal involvement and were considered in conjunction with clinical findings to support the overall diagnosis. Radiological findings were not considered primary outcome measures, however, used only to support the overall clinical diagnosis. Anti-CCP testing was not available for all patients due to clinical practice variability within the institute, and only patients with available results were included in the anti-CCP analysis. Demographic and clinical characteristics of the patients were obtained from medical records. All the findings and examinations were recorded on a pre-designed pro forma. The inclusion criteria were:

1. Age > 18 years;
2. Diagnosis of PsA fulfilling CASPAR criteria;
3. Duration of disease greater than 6 months;
4. Availability of anti-CCP antibody testing (Either current or within the past 12 months);
5. Consent to participate.

The Exclusion Criteria were:

1. Patients with undifferentiated arthritis or overlap syndromes (e.g., PsA with lupus or systemic sclerosis);
2. Incomplete clinical or serological data (If core data points such as CCP status, RF, joint distribution, and psoriasis history are missing);
3. Pregnant or lactating individuals.

The data was entered and analyzed using Statistical Package for Social Sciences (SPSS-27). Chi-Square test and logistic regression were used to assess the association between aCCP positivity and the pattern of skeletal involvement, and to determine the odds ratio p-value <0.05 was set for statistical significance. Data was entered using SPSS (ver 27). Frequencies and percentages were computed. Chi-square test was used to find the association between the demographic and clinical outcomes with the frequency of aCCP. Logistic regression was utilized to find the possible effect of demographic/clinical outcomes to aCCP.

RESULTS

OpenEpi was used to determine the sample size with a prevalence of 4.5% of aCCP positive among PsA patients, and 95% confidence interval, ±5% maximum error in estimate, and 80% power of the test. This was a cross-sectional study. Convenient sampling was utilized to collect the information from the patients. Sixty-one (61) patients participated in the study. Twenty-six (26) were male, and the majority of them were between 18 and 40 years (49.2%) (Figure 1). Skin involvement was detected in 55 participants (90.2%). Regarding joint involvement, 44 participants (72.1%) had small joint involvement in terms of distal interphalangeal (DIP) joints. Seventeen participants (27.9%) were affected, compared to 44 (72.1%) who were not. Intermediate joints were involved in 19 participants (31.1%), and large joint involvement was observed in 44 participants (72.1%). (Figure 2) Inflammatory back pain was reported by 26 participants (42.6%). Peripheral involvement was present in 51 participants (83.6%), and axial involvement was reported by 26 participants (42.6%).

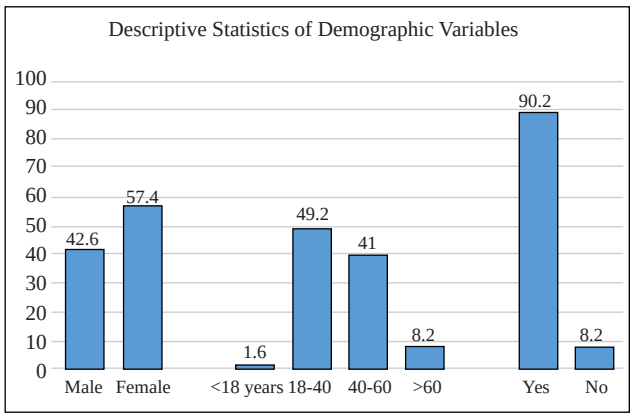

Figure 1: Descriptive Statistics for gender, age-groups, and skin involved

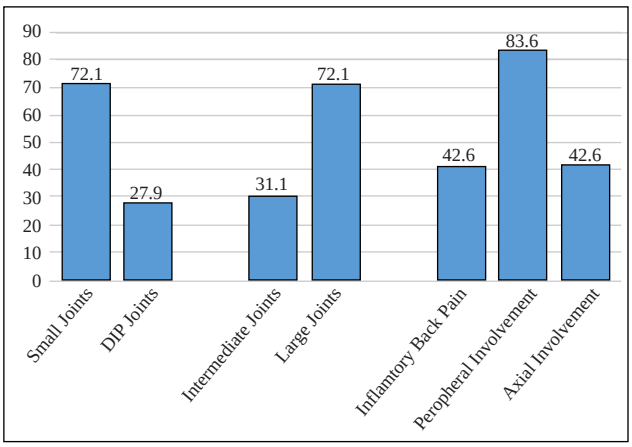

Figure 2: Descriptive Statistics of Joints Involved and Pain

Among the 45 participants tested for anti-CCP antibodies, 14 (31.1%) were positive for anti-CCP antibodies.

Table 1 shows the association of anti-CCP antibodies with various demographic and clinical factors. Anti-CCP antibody levels were assessed in a sample of 45 participants. Among males, 4 (25%) tested positive for anti-CCP, while 10 (34.5%) females tested positive. The difference was insignificant (p=0.738). The older patients (> 60 years) showed significantly higher positive anti-CCP (47.6%) than younger patients (16.7%) (p=0.025).

Table 1: Association of Anti-CCP with Clinical Features

Variable	Anti-CCP Positive n (%)	Anti-CCP Negative n (%)	Total (n)	p-value
Gender				
Male	4 (25.0)	12 (75.0)	16	0.738
Female	10 (34.5)	19 (65.5)	29	
Age Group				
18–40	4 (16.7)	20 (83.3)	24	0.025*
>60	10 (47.6)	11 (52.4)	21	
Skin Involvement				
Yes	9 (23.1)	30 (76.9)	39	0.008*
No	5 (83.3)	1 (16.7)	6	
Small Joint Involvement				
Yes	13 (38.2)	21 (61.8)	34	0.132
No	1 (9.1)	10 (90.9)	11	
DIP Joint Involvement				
Yes	6 (46.2)	7 (53.8)	13	0.286
No	8 (25.0)	24 (75.0)	32	
Intermediate Joint Involvement				
Yes	5 (45.5)	6 (54.5)	11	0.277
No	9 (26.5)	25 (73.5)	34	
Large Joint Involvement				
Yes	8 (28.6)	20 (71.4)	28	0.637
No	6 (35.3)	11 (64.7)	17	
Inflammatory Back Pain				
Yes	7 (33.3)	14 (66.7)	21	0.763
No	7 (29.2)	17 (70.8)	24	
Peripheral Involvement				
Yes	10 (28.6)	25 (71.4)	35	0.700
No	4 (40.0)	6 (60.0)	10	
Axial Involvement				
Yes	7 (33.3)	14 (66.7)	21	0.763
No	7 (29.2)	17 (70.8)	24	

Of those with skin involvement, 9 (64.3%) tested positive for anti-CCP, while 5 (83.3 %) without skin involvement tested positive. The association was statistically significant (p=0.008). None of the other factors (skin involved, small joints involved, DIP joints involved, intermediate involved, large joints involved, inflammatory back pain, peripheral involved, and axial involved) showed any statistically significant association with anti-CCP antibodies (p>0.05).

Binary logistic regression analysis was used to evaluate the odds of testing positive for anti-CCP in relation to various demographic and clinical factors (Table 2).

Table 2: Odds For Patients With Positive Anti Ccp (N=45)

	OR (95% CI)	p-value
Gender		
Male	0.633(0.162-2.483)	0.512
Female	Ref	
Age Group		
18-40 years	0.400(0.029-5.547)	0.495
40-60 years	2.000(0.153-26.187)	0.597
>60 years	Ref	
Skin		
Yes	0.060(0.006-0.582)	0.015*
No	Ref	
Small Joint involved		
Yes	6.190(0.708-54.157)	0.099
No	Ref	
DIP Joints Involved		
Yes	2.571(0.665-9.944)	0.171
No	Ref	
Intermediate Joints Involved		
Yes	2.135(0.565-9.484)	0.243
No	Ref	
Large Joint Involved		
Yes	0.733(0.202-2.662)	0.637
No	Ref	
Inflammatory Back Pain		
Yes	1.214(0.343-4.298)	0.763
No	Ref	
Peripheral Involvement		
yes	0.600(0.139-2.590)	0.494
No	Ref	
Axial Involvement		
Yes	1.214(0.343-4.298)	0.763
No	Ref	

None of the demographic or clinical factors showed any statistically significant odds ratio, except the skin involved. Skin involved (yes) showed a significantly low odd ratio of 0.06 (CI: 0.006 – 0.582) with p value of 0.015.

Since the univariate analysis showed no significant difference for any explanatory variable except the skin involved, a multivariate binary logistic regression was attempted.

Table 3 shows the bivariate correlation ; that only age, skin, and small joints involved had significant correlation with aCCP, keeping p-value <0.1, as the standard for entry of variable in the equation. The multivariate logistic analysis showed that the skin involved is the only variable with p<0.05.

Table 3: Model Developed by the Multivariate Logistic Regression

B		S.E.	Wald	df	p-value	Exp(B)	95% C.I. for EXP(B)	
							Lower	Upper
Skin	3.005	1.274	5.562	1	.018	20.184	1.661	245.233
F			4.502	2	.105			
Age(1)	-.105	1.722	.004	1	.952	.901	.031	26.322
Age(2)	1.577	1.723	.837	1	.360	4.839	.165	141.766
Constant	-.425	1.560	.074	1	.785	.654		

DISCUSSION

Prevalence and Clinical Significance of Anti-CCP Antibodies in PsA

Based on our study findings, 31.1 % of patients with psoriatic arthritis (PsA) tested positive for anti-cyclic citrullinated peptide (anti-CCP) antibodies. This percentage of anti-CCP positivity is notable higher than the previously reported range of 7-17 %. In comparison to rheumatoid arthritis (RA), where 70 - 85 % of patients show positive results for anti-CCP antibodies, the rate in PsA is considerably lower. Anti-CCP antibodies are commonly known as an indicator for RA, yet their significance in PsA remains unexplored[11,12]. A research study involving 77 individuals with PsA, found that 20% tested positive for anti-CPP antibodies. This percentage is higher than that seen in the general population but is similar to that of psoriasis patients without joint complications[4]. These results indicate that although anti-CPP antibodies are less frequent in PsA than RA, they still play a significant role within the spectrum of psoriatic diseases.

Various reasons contribute to the differences in the presence of anti-CCP antibodies among individuals with PsA. Variances in the criteria used to select participants for studies have an impact on research outcomes. Moreover, factors such as standards and testing techniques influence the reported prevalence rates significantly. Genetic elements also play a role,

with the presence of the HLA-DRB1 shared epitope being linked to anti-CCP positivity and potentially influencing how these antibodies develop in PsA patients[13]. The differences may stem from variations in the detection kits and positivity thresholds used. This further adds to the challenge of maintaining consistency in prevalence data[14,15]. By establishing updated detection methods and criteria for diagnosis, we can potentially address these variations, thereby enhancing the reliability of anti-CCP testing.

Though not as common in PsA, anti-CCP antibodies hold significant clinical importance due to their association with more severe disease manifestations in patients. Literature also indicates that individuals testing positive for anti-CCP antibodies are more likely to experience a polyarticular form of arthritis and be at risk of developing joint erosions and substantial deformities[11,13,16]. Analysis has revealed that PsA patients with anti-CCP antibodies typically have a more extensive joint involvement and experience heightened disease activity compared to individuals without these antibodies[17]. Therefore, this connection indicates that there might be a more aggressive spectrum of PsA when the level of serum anti-CCP antibodies is elevated.

It is also important to mention that individuals with PsA who test positive for anti-CCP antibodies are typically older, 62.43 years of age on average, compared to those who test negative and are usually 47.59 years old on average. This age contrast could signify a more chronic disease progression in individuals with anti-CCP antibody positivity. Furthermore, PsA patients with anti-CCP antibodies tend to experience broader and more severe manifestations of joint disease as they tend to show involvement of metacarpophalangeal, elbow, and shoulder joints. In our study, anti-CCP positivity demonstrated a significant inverse association with skin involvement, with patients lacking cutaneous manifestations more likely to be anti-CCP positive, a finding further supported by regression analysis showing significantly lower odds in patients with skin involvement. These findings suggest that anti-CCP antibodies could be a marker for more extensive disease in PsA.

Comparison with Rheumatoid Arthritis

The function of anti-CCP antibodies in PsA shows a notable contrast to their function in RA. In the case of RA, specifically, anti-CCP antibodies serve as an indicator of the disease, predicting the advancement and severity of the condition[13,17]. These antibodies are associated with increased disease activity, erosive effects, and more pronounced functional impairment compared to individuals who do not possess these antibodies[18]. Contrary to this, anti-CCP antibodies, despite being associated with severe disease characteristics like polyarthritis and erosive alterations, show a less pronounced effect in PsA when compared to RA[12,19]. For instance, a study revealed that PsA patients with anti-CCP positivity often experience severe clinical symptoms but do not consistently display significant radiographic changes[12,20]. The results indicate that anti-CCP antibodies can be associated with disease severity in PsA, although not to the same intensity or in the same way as in RA. In PsA, the connection between anti–CCP antibodies and radiological results presents a complex scenario, unlike RA. In RA, anti-CCP antibodies strongly correlate with bone erosion and joint deterioration, serving as a distinct indicator of disease advancement[21,22].

In PsA, however, the link between anti-CCP antibodies and radiologic consequences appears definitive. Patients with PsA who test positive for anti-CCP antibodies tend to experience more severe clinical manifestations, such as increased joint swelling and higher chances of developing erosive arthritis, compared to those who test negative for the antibody. It is important to understand that these symptoms may not always result in changes when viewed through imaging[10,19,20]. This discrepancy suggests differences in the underlying mechanisms of PsA and RA despite some overlapping characteristics between the two conditions.

In RA, specifically, the presence of anti-CCP antibodies is linked to bone damage through processes involving the activation of osteoclasts and the production of inflammatory cytokines[21]. In PsA, a similar mechanism takes place, but more moderate results are observed. Some research indicates that certain PsA patients possess anti-CCP antibodies in their system. However, they may not be pivotal in the disease process compared to rheumatoid arthritis RA[20].

PsA is distinguished by the joint's asymmetrical inflammation and heightened cytokine levels like IL-17 and IL-23. In contrast, RA manifests with symmetrical joint involvement and increased levels of IL-2, IL-33, and TNF á-levels[6]. The lower occurrence and inconsistent connection of anti-CCP antibodies with the consequences of PsA raises the need to explore other elements, such as genetic predisposition and environmental influences that could impact the advancement of the disease[11,18].

Diagnostic and Management Challenges

The presence of anti-CCP antibodies often impacts the response to treatment in PsA patients. Various studies show that PsA patients with anti-CCP antibodies are

less likely to exhibit favorable responses to TNF inhibitors than PsA without these antibodies. The activation of inflammatory pathways by anti-CCP antibodies interferes with TNF inhibitors' effectiveness in independently targeting these pathways. This resistance against TNF inhibitors can be attributed to the immune response triggered by the presence of anti-CCP antibodies[2]. The pressing issue is the importance of gaining an insight into the immune pathways triggered by anti-CCP antibodies to explore other treatment options or additional approaches for PsA patients who test positive for anti-CCP antibodies. Tailoring treatment plans based on the detection of anti-CCP antibodies can show the potential to enhance disease outcomes.

The presence of anti-CCP antibodies in PsA introduces a new complexity to the diagnosis and treatment process. It can be difficult to distinguish enthesopathy in patients with psoriasis from cases of overt PsA since as many as half of all psoriasis patients may have subclinical enthesopathy that could develop into PsA[5]. Identifying and addressing these issues early is crucial since delayed diagnosis can result in worse consequences, such as erosion of peripheral joints and reduced physical capabilities[8,9]. Therefore, tackling these diagnostic hurdles and ensuring early interventions for improved patient outcomes is essential. Research is still being conducted on the genetic basis of PsA. Although certain genetic loci, like PSORS1 on chromosome 6, are known, the full genetic makeup of PsA remains elusive[23]. It is pivotal to identify and comprehend how certain genetic variations impact disease vulnerability and response to treatment to progress further in this study area. Genome-wide association studies (GWAS) and further research examining HLA alleles could help connect the dots in understanding the intricate pathogenesis of PsA, thereby enhancing our capacity to provide customized and efficient therapies. It is recommended that forthcoming studies delve deeper into investigating the significance of anti-CCP antibodies and other biological markers in the pathogenesis of PsA to achieve improved treatment responses and disease outcomes.

CONCLUSION

In our study, anti-CCP antibodies were found in 31.1% of the PsA patients studied. Although this is far higher than previously reported figures, it still falls short of the percentage reported for RA. Anti-CCP positivity may represent a distinct clinical subset in PsA and was associated with older age and inverse relationship with skin involvement. Anti-CCP positivity is a determinant of disease outcome in PsA as it is associated with a distinct clinical pattern of musculoskeletal involvement in PsA. Patients with anti-CCP positivity tend to be insensitive to TNF inhibitors and should receive alternative or supplementary treatment. These results emphasize the potential of anti-CCP antibodies as a marker for more severe PsA and emphasize the requirement for tailored treatments for better patient outcomes.

Limitation

This study is limited by its single -center design and relatively small sample size, which may affect the generalizability of the findings.

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Authors' Contribution: **SRZ** conceptualized the Idea, conducted the literature search and study selection, performed data synthesis and interpretation, and wrote and revised the manuscript. **LN** contributed to the literature search and study selection, assisted in data synthesis and interpretation, and reviewed and revised the manuscript. **TP** assisted in conceptualization, contributed to data interpretation, and critically reviewed the manuscript for intellectual content. **ZA** participated in the literature review, data organization, and manuscript drafting and revision. **SAJ** supervised the study process, contributed to the interpretation of findings, and critically revised and approved the final manuscript.

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