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Research Article

Autoantibody profile in patients with psoriatic arthritis before and after bDMARD therapy

Psoriatik artritli hastalarda bDMARD tedavisi öncesi ve sonrası otoantikor profili

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Abstract

Aim: This study aimed to evaluate the real-life prevalence and changes of commonly tested autoantibodies, including antinuclear antibody (ANA), rheumatoid factor (RF), and anti-cyclic citrullinated peptide (anti-CCP), among patients with psoriatic arthritis (PsA) before and after biologic disease-modifying antirheumatic drug (bDMARD) therapy.

Material and Methods: Patients with PsA from the Hacettepe University Rheumatology Biologic Database (HUR-BIO) were retrospectively evaluated. Demographic characteristics, seropositivity rates, antibody titers, and ANA pattern subtypes were recorded both before and after the initiation of bDMARD therapy.

Results: Among 520 patients (69.4% female, mean age 39.2 ± 5.2 years), 69% demonstrated positivity for at least one autoantibody prior to bDMARD therapy. ANA exhibited the highest frequency of seropositivity, increasing from 40.0% before treatment to 55.3% after treatment. Concurrent RF and anti-CCP positivity were observed in 2.8% and 6.3% of patients before and after treatment, respectively. The most frequent ANA patterns were AC-4,5 prior to, and AC-1,4,5 following therapy. Of 31 patients tested at both time points, 6 (19.4%) converted from ANA-negative to ANA-positive following bDMARD initiation.

Conclusions: Real-world data indicate that only 20–30% of patients with PsA were seronegative for all three routinely assessed autoantibodies. Consistent with previous reports, ANA positivity rates significantly increased following bDMARD therapy.

Keywords: psoriatic arthritis, biologic disease-modifying antirheumatic drugs, autoantibody, serology

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Öz

Amaç: Bu çalışmanın amacı, biyolojik ajanlarla tedavi edilen psoriatik artrit (PsA) hastalarında sık kullanılan otoantikorların gerçek yaşam verilerindeki prevalansını değerlendirmektir.

Gereç ve Yöntemler: Hacettepe Üniversitesi Romatoloji Biyolojik Veritabanı'na (HUR-BIO) kayıtlı psoriatik artritli hastalar, biyolojik tedavi başlanmadan önce ve başlandıktan sonra elde edilen antinükleer antikor (ANA), romatoid faktör (RF) ve anti-siklik sitrüline peptid (anti-CCP) profilleri açısından değerlendirildi. Laboratuvar testleri ile biyolojik tedavi başlangıcı arasındaki süre, seropozitiflik oranları, antikor titreleri ve ANA desen alt tipleri kaydedilip analiz edildi.

Bulgular: Toplam 520 PsA hastasının %69,4'ü kadındı; tanı yaşı ortalama 39,2 ± 5,2 yıl, hastalık süresi ortalama 3,3 ± 5,2 yıl idi. Biyolojik tedavi öncesinde test yapılan hastaların %69'unda en az bir otoantikor pozitifliği saptandı. ANA, en sık gözlenen otoantikor olup tedavi öncesinde %40,0, tedavi sonrasında ise %55,3 oranında pozitifti. Eş zamanlı RF ve anti-CCP pozitifliği, sırasıyla tedavi öncesinde %2,8 ve tedavi sonrasında %6,3 oranında görüldü. En sık ANA paternleri tedavi öncesinde AC4–5, tedavi sonrasında ise AC1–4–5 olarak belirlendi. Hem tedavi öncesi hem sonrası ANA testi bulunan 31 hastanın 6'sında (%19,4) biyolojik tedavi sonrasında negatiften pozitife dönüş gözlendi.

Sonuç: bDMARD tedavisi alan PsA hastalarına ait gerçek yaşam verileri, hastaların yalnızca %20–30'unun üç temel otoantikor açısından seronegatif olduğunu göstermektedir. Önceki çalışmalarla uyumlu olarak, biyolojik tedavi sonrası ANA pozitifliği oranlarında artış saptanmıştır.

Anahtar Kelimeler: psoriatik artrit, biyolojik hastalık modifiye edici antiromatizmal ilaçlar, otoantikor, seroloji

Introduction

Psoriatic arthritis (PsA) is a chronic inflammatory disease classified within the spectrum of spondyloarthritis. Its diagnosis can be challenging due to overlapping clinical and serological features with other inflammatory arthritis, particularly rheumatoid arthritis (RA) [1,2]. Although no specific biomarker or autoantibody currently exists to facilitate the differential diagnosis, PsA is generally characterized as a seronegative disease, by definition negative for rheumatoid factor (RF) [3,4].

Nevertheless, several studies have reported varying frequencies of RF and anti-cyclic citrullinated peptide (anti-CCP) antibody positivity, as well as antinuclear antibody (ANA) positivity, among PsA populations [5]. Despite these observations, data remain limited regarding the prevalence and evolution of these autoantibodies in patients requiring advanced treatment modalities, particularly biological disease-modifying antirheumatic drugs (bDMARDs).

It is known that ANA positivity may develop during bDMARD therapy; however, evidence specific to PsA cohorts remains scarce [6-8]. Understanding autoantibody profiles may have implications for both diagnosis and treatment monitoring in clinical practice.

Therefore, the aim of this study was to assess the prevalence and titers of RF, anti-CCP, and ANA, along with ANA subtypes, before and after bDMARD therapy in patients with PsA

Material and Methods

Established in 2005, the Hacettepe University Rheumatology Biologic Database (HUR-BIO) is a single-center registry that prospectively collects data on patients with inflammatory arthritis treated with biologic disease-modifying antirheumatic drugs (bDMARDs). Data for the present study were extracted from this registry.

Patients with PsA were evaluated for RF, anti-CCP, and ANA status before and after the initiation of bDMARD therapy. Serum IgG RF was measured by nephelometry (IMMAGE System, Beckman Coulter, USA) and anti-CCP antibodies were measured using commercial enzyme-linked immunosorbent assay (ELISA) (Euroimmun Diagnostics, Germany), with positivity defined as ≥20 IU/mL for IgG RF and ≥5 IU/mL for IgG anti-CCP (second-generation assay). ANA was detected by indirect immunofluorescence assay (IFA) on HEp-2 cells (Euroimmun Diagnostics, Germany), with titers ≥1:100 considered positive.

When multiple test results were available, the measurement obtained closest to the bDMARD initiation date was selected for the "before bDMARD" group, and the result closest to the most recent follow-up was used for the "after bDMARD" group. The analyses included ANA, RF, and anti-CCP positivity rates, antibody titers, and patient characteristics such as sex and age at testing. ANA staining patterns were classified according to the International Consensus on ANA Patterns (ICAP) [9].

The diagnosis of PsA was established by the treating rheumatologist. Additionally, positivity of the Classification for Psoriatic arthritis (CASPAR) was retrieved.Demographic characteristics (age, sex, smoking status, body mass index (BMI), and family history of psoriasis) and disease features (dactylitis, enthesitis, and nail involvement) were collected for all patients, with particular focus on those who were positive for at least one autoantibody and those who were triple-negative.

Ethical approval was obtained from the Hacettepe University Faculty of Medicine Ethics Committee (No. KA-22005), and the study was carried out in accordance with the Declaration of Helsinki. All participants provided written informed consent.

Statistical Analysis

Statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS) software, version 25.0 (IBM Corp., Armonk, NY, USA). Demographic and descriptive data were expressed as median [interquartile range (IQR)] or mean (standard deviation, SD), depending on the data distribution. Normality was assessed both visually (via histograms and probability plots) and analytically (using the Kolmogorov–Smirnov test, skewness, and kurtosis). Results for count data were reported as valid percentages.

Results

Among the 520 PsA patients (93.8% CASPAR positive) included in the study, 69.4% were female, with a mean (SD) age at diagnosis of 39.2 (5.2) years and a mean (SD) disease duration of 2.8 (5.2) years. When comparing patients with a triple-negative autoantibody profile to those with at least one autoantibody positivity before bDMARD initiation, some clinical and demographic differences were observed (Table 1). Patients with autoantibody positivity were diagnosed with PsA at an older age than triple-negative individuals (p = 0.02). Dactylitis was significantly more frequent among triple-negative patients compared with those with autoantibody positivity (p = 0.02). Although other differences did not reach statistical significance, several numerical trends were noted. A family history of psoriasis was more frequent among triple-negative patients (25% vs 15.7%), whereas obesity and smoking history were more common in autoantibody-positive patients (43.7% vs 34.4% and 51.4% vs 40.6%, respectively). Enthesitis and nail involvement were also slightly more prevalent in those with autoantibody positivity (48.9% vs 43.5% and 45.3% vs 26.9%, respectively).

When comparing ANA-positive and ANA-negative patients before bDMARD initiation, some trends were observed, although differences did not reach statistical significance in any of the parameters. Patients with ANA positivity tended to be diagnosed at an older age, and the proportion of females was numerically higher in this group. Obesity was also more frequent among ANA-positive patients. Enthesitis was more common in ANA-positive individuals compared with ANA-negative patients (50.0% vs 31.6%, p = 0.12), whereas rates of dactylitis and smoking history were similar between groups.

Before biologic treatment RF, anti-CCP and ANA tests were available for 310, 144 and 104 patients, respectively. After biologic treatment initiation, the number of tested patients increased across all assays. RF positivity showed a modest increase from 9.6% to 11.5%, with comparable titers before and after therapy (median 28.7 vs 28.9 IU/mL). Anti-CCP positivity similarly rose slightly (8.3% to 11.3%), while median titers did not increase (139.1 to 67.5 IU/mL). Concomitant RF and anti-CCP positivity increased from 2.8% to 6.3% of tested patients. ANA positivity rose from 40.4% (42/104) before treatment to 55.3% (73/132) after treatment, accompanied by a shift toward higher titers (1/320 or greater in 27.3% vs 16.7% of cases). When all antibodies were considered together, the proportion of patients with at least one autoantibody positivity increased from 69.2% before biologic initiation to 78.7% afterward, whereas triple-negative cases declined from 30.8% to 21.3%.

Among patients with serial ANA assessments, 6 who were ANA-negative before biologic initiation developed ANA positivity after treatment. The biologic agents used in these cases included infliximab (n = 2; 33.3%), adalimumab (n = 2; 33.3%), certolizumab pegol (n = 1; 16.7%), etanercept (n = 1; 16.7%), and guselkumab (n = 1; 16.7%), with some patients receiving more than one biologic sequentially. The remaining 13 patients (68.4%) were persistently ANA-negative during follow-up, despite exposure to a range of biologics, most commonly adalimumab (n = 7; 53.8%), certolizumab pegol (n = 4; 30.8%), etanercept (n = 2; 15.4%), golimumab (n = 2; 15.4%), secukinumab (n = 1; 7.7%), and ustekinumab (n = 1; 7.7%), with several patients receiving multiple agents.

Among ANA-positive patients, diverse immunofluorescence patterns were observed both before and after bDMARD treatment. The most frequently detected patterns were those containing AC4 (n = 23, 22.1%) and AC5 (n = 20, 19.2%), which often co-occurred in combinations such as AC4-AC5 or AC1-AC4-AC5. The AC2 pattern was observed in 10 patients (9.6%), sometimes in association with AC4 or AC8. Less frequent patterns included AC8 (n = 5, 4.8%) and AC1 (n = 3, 2.9%). A cytoplasmic pattern accompanied nuclear patterns in 6 patients (5.8%). while other patterns were not identified before bDMARD (Table 3).

Table 1. Patient and disease characteristics at bDMARD initiation.

	All patients n=520	Patient with triple negative autoantibody profile before bDMARD (n=32)	Patients with at least one autoantibody positivity before bDMARD (n=72)	ANA negative before bDMARD (n=62)	ANA positive before bDMARD (n=42)	P1 *	P2**
Female gender, n (%)	361 (69.4)	25 (78.1)	56 (77.8)	48 (77.4)	36 (85.7)	0.96	0.29
PsA diagnosis age, mean (SD), years	39.2 (12)	36.9 (10.9)	42.6 (11.6)	39 (12.1)	42.8 (10.8)	0.02	0.10
PsA disease duration, mean (SD), years	2.8 (5.2)	2.3 (3.5)	2.7 (5.3)	2.8 (4.3)	2.6 (5.6)	0.75	0.22
PsO family history, n (%)	147 (30.1)	8 (25)	11 (15.7)	16 (25.8)	6 (14.6)	0.26	0.18
BMI > 30, n (%)	195 (39.2)	11 (34.4)	31 (43.7)	25 (40.3)	22 (52.4)	0.37	0.22
Smoking (ever), n (%)	299 (59.2)	13 (40.6)	37 (51.4)	30 (48.4)	21 (50)	0.31	0.87
Dactylitis (ever), n (%)	74 (23.8)	9 (37.5)	7 (14)	11 (23.9)	6 (20)	0.02	0.69
Entheisitis (ever), n (%)	104 (39.4)	10 (43.5)	22 (48.9)	12 (31.6)	15 (50)	0.67	0.12
Nail involvement, n (%)	122 (40.9)	7 (26.9)	24 (45.3)	14 (29.8)	10 (32.3)	0.11	0.82

Abbrev.; PsA: Psoriatic arthritis, PsO: Psoriasis, BMI: Body mass index, ANA: Anti-nuclear antibody, bDMARD: biologic disease modifying anti-rheumatic drugs.
*Comparison between patients with triple negative autoantibody profile before bDMARD vs patients with at least one autoantibody positivity before bDMARD.
**Comparison between ANA negative before bDMARD vs ANA positive before bDMARD.

Table 2. Demographics and ANA, RF, Anti-CCP test results of patients before and after biologic treatment.

			Before biologic initiation	After biologic initiation
ANA	Age, mean (SD), years		43.5 (12.7)	46.7 (11.6)
	Gender, F:M		84:20	97:35
	Time interval between test and biologic start, months, median (IQR)		7.4 (0.84-17.83)	32.6 (14.93-72.33)
	Positivity, n (%)		42/104 (40.4)	73/132 (55.3)
	Titer n (%)*	1/100	28 (66.6)	38 (52)
		1/160	7 (16.7)	14 (19.1)
		1/320	7 (16.7)	17 (23.2)
		1/1000	0	3 (4.1)
RF	Age, mean (SD), years		43.3 (12.5)	47.9 (11.9)
	Gender, F:M		225/85	211/67
	Time interval between test and biologic start, months median (IQR)		4.1 (0.35-16.75)	31.63 (13.10-64.08)
	Positivity, n (%)		30/310 (9.6)	32/278 (11.5)
	Titer, IU/ml		28.7 (22.35-98.5)	28.9 (21.9-110)
Anti-CCP	Age, mean (SD), years		44.3 (12)	48.6 (12.1)
	Gender, F:M		110/34	75/22
	Time interval between test and biologic start, months, median (IQR)		3.23 (0.30-11.5)	35.13 (12.40-75.43)
	Positivity, n (%)		12/144 (8.3)	11/97 (11.3)
	Titer, IU/ml		139.1 (20.38-250)	67.5 (16.77-139)
RF+Anti-CCP concomitant positivity, n (%)			4/138 (2.8)	6/97 (6.3)
At least one antibody positivity, n (%) **			72/104 (69.2)	100/127 (78.7)
Triple antibody negativity, n (%)			32/104 (30.8)	27/127 (21.3)

Abbrev.; ANA: Anti-nuclear antibody, RF: Rheumatoid factor, Anti-CCP: Anti- Cyclic citrullinated peptide, F:Female, M:Male, IQR: Interquartile range, IU/ml: International units per milliliter.
*titer is not given for one patient in patients with positive ANA after biologic treatment.
**total number of patients include patients with at least one positive test where triple negative patients, patients with missing data on any autoantibody were excluded.

Table 3. ANA subtypes.

	Before biologic initiation, n=42			After biologic initiation, n=73*			
Subtype \ Titer	1/100	1/160	1/320	1/100	1/160	1/320	1/1000
AC2	6	1	0	9	2	0	0
AC3	0	0	0	0	0	1	0
AC8	2	0	0	1	0	0	0
AC1-AC8	0	0	1	0	0	0	0
AC2-AC8	0	1	1	0	0	0	0
AC1-AC15	0	0	0	1	0	0	0
AC4-AC5	11	2	1	11	2	1	0
AC1-AC4-AC5	1	1	2	6	9	13	3
AC2-AC4-AC5	0	0	0	1	1	0	0
AC4-AC5-AC8	2	0	0	1	0	0	0
AC4-AC5- cytoplasmic	3	1	1	1	0	0	0
AC1-AC4-AC5- cytoplasmic	1	1	0	6	0	2	0
AC4-AC5-AC8- cytoplasmic	2	0	1	0	0	0	0
AC1-AC4-AC5-AC11	0	0	0	1	0	0	0

*subtype is not given for one patient
Numbers are indicating number of patients

Discussion

This study demonstrated that nearly 70% of PsA patients had at least one autoantibody positivity prior to bDMARD treatment, challenging the long-held perception of PsA as a predominantly "seronegative" disease. In line with previous literature, RF and anti-CCP antibodies remained uncommon; however, the observed rise in concomitant RF and anti-CCP positivity after biologic exposure warrants further investigation. ANA positivity was notably frequent at baseline and showed a further 15% increase following bDMARD initiation, suggesting a potential treatment-related induction of autoantibodies.

When comparing PsA patients with and without baseline autoantibody positivity, several demographic and clinical distinctions became apparent. Patients exhibiting at least one autoantibody were diagnosed with PsA at an older age, suggesting that cumulative immune dysregulation over time may predispose to humoral autoimmunity [10]. Obesity and history of smoking were also more frequent in this group, both recognized contributors to systemic inflammation and loss of immune tolerance [10,11]. In contrast, triple-negative patients more often displayed a positive family history of psoriasis and higher rates of dactylitis, features that align closely with the classical psoriatic disease spectrum and the concept of seronegativity. Collectively, these findings imply that PsA patients with serological reactivity may represent a somewhat distinct phenotype, characterized by later onset, greater inflammatory burden, and metabolic comorbidities, whereas purely seronegative individuals tend to show more traditional psoriatic features and stronger familial clustering.

In the literature, the prevalence of RF and anti-CCP antibodies in PsA varies but consistently remains low compared with rheumatoid arthritis. Alenius et al. reported RF positivity in 11% and anti-CCP in 7% of PsA patients, noting that anti-CCP positive individuals often had a polyarticular pattern [12]. Similarly, Popescu et al. found anti-CCP positivity in 12.2% of PsA cases, with higher rates of polyarticular involvement and more frequent biologic use, suggesting that anti-CCP positivity may identify a subset with more aggressive disease [13]. Silvy et al. observed RF positivity in 15% and anti-CCP in only 1.7% of PsA patients [14]. In our cohort, RF and anti-CCP positivity rates (approximately 10% and 8–11%, respectively) were within the reported ranges with anti-CCP positivity near the upper limit reported in the literature. However, this was an expected result since the patients in our cohort were patients who required advance treatment modalities. The observed modest increase after biologic treatment initiation and the rise in concomitant RF and anti-CCP positivity (from 2.8% to 6.3%) were in line with prior observations, potentially reflecting immune modulation [15].

The prevalence of ANA positivity in PsA has been reported between 14% and 52%, which is consistent with our findings and reflects differences in methodology and disease spectrum across studies [5]. Similar to previous work, ANA titers in our

cohort were predominantly low to moderate, aligning with the observation that most psoriatic patients exhibit ANA reactivity of limited clinical consequence [6]. Importantly, our data demonstrated both an increase in ANA positivity and in titers after biologic treatment initiation, in concordance with the previous reports describing biologic-induced seroconversion, particularly with TNF-α inhibitors. In line with Bardazzi et al., who reported ANA emergence in nearly half of infliximab-treated patients, and Chimenti et al., who observed new ANA and anti-dsDNA antibodies in 16% and 8% of cases, respectively, infliximab again emerged as the agent most frequently associated with seroconversion [7,8]. Overall, our results corroborated that ANA induction was relatively common during biologic therapy in PsA and infliximab was the most frequently associated one observed in newly positive group and not seen in consistently negative group,

In our assessment, most ANA-positive patients exhibited AC4 and AC5 patterns, representing fine and coarse speckled nuclear staining. The AC4 pattern is among the most frequent findings in routine HEp-2 IFA testing and is commonly observed in asymptomatic individuals or patients without systemic autoimmune disease [9]. In a previous study most of the patients were showing anti-DFS70 reactivity, where ANAs against specific nuclear antigens were evaluated using a line immunoassay and the predominant IIF pattern was fine speckled which mainly corresponds to the AC4 pattern [6]. Following bDMARD exposure, we observed a shift in ANA subtypes, with the AC1 pattern appearing in combination with AC4-AC5 (e.g., AC1–AC4–AC5), accompanied by higher titers. The emergence of AC1, typically associated with homogeneous nuclear staining, may reflect drug-induced lupus–like changes or anti-dsDNA–related activity, consistent with prior reports of new dsDNA antibody development in patients receiving TNF inhibitors [7,8]. However, in our study, extractable nuclear antigen testing was not performed, and interpretations were based solely on the immunofluorescence pattern profile, limiting antigen-specific correlations.

Limitations of the study

This study has several limitations. First, its retrospective design precludes establishing temporal causality between biologic exposure and autoantibody development. Second, not all patients had complete testing for all three autoantibodies both before and after treatment, which may have introduced selection bias and limited comparative analyses. Additionally, ENA testing was not routinely performed in the cohort, resulting in insufficient longitudinal ENA data; this limited our ability to evaluate whether specific ENA specificities changed with biologic therapy. Third, the absence of data correlating antibody changes with lupus-like features, disease activity measures, or treatment response or long-term outcomes restricts interpretation of their functional or clinical significance. Additionally, many patients received sequential biologic therapies, making it difficult to attribute seroconversion to a specific drug class.

In conclusion, up to 70% of PsA patients requiring advanced treatment modalities demonstrated positivity for at least one autoantibody before biologic initiation, increasing to nearly 80% after bDMARD therapy. Autoantibody-positive patients were more likely to be older, obese, and have a history of smoking, suggesting that these factors may contribute to, or serve as markers of heightened immune reactivity in this patient group.

Declaration of conflicting interests

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Ethics approval

This study was approved by the Hacettepe University Faculty of Medicine Ethics Committee with protocol number KA-22005.

Authors' contribution

GA: Concept, Design, Methodology, Data Collection and/or Processing, Analysis and/or Interpretation, Literature Review, Writing of the Article. NEG: Data Collection and/or Processing, Analysis and/or Interpretation. BS: Methodology, Materials, Data Collection and/or Processing, Critical Review. ZS: Methodology, Materials, Data Collection and/or Processing, Critical Review LK: Design, Data Collection and/or Processing, Supervision. UK: Concept, Design, Methodology, Supervision, Data Collection and/or Processing, Analysis and/or Interpretation, Critical Review.

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