Psoriasis: Targets and Therapy

Dovepress
Taylor & Francis Group

Open Access Full Text Article

ORIGINAL RESEARCH

Are Systemic Drug Choices for Psoriasis by Dermatologists Compatible with Psoriatic Arthritis? Data from the German National Psoriasis Registry PsoBest

Christina Sorbe[1], Secilay Kargin[1], Ralph von Kiedrowski[2], Diamant Thaci[3], Ansgar Weyergraf[4], Christine Blome[1], Matthias Augustin[1], Brigitte Stephan[1]

[1]Institute for Health Services Research in Dermatology and Nursing (IVDP), University Medical Center Hamburg-Eppendorf (UKE), Hamburg, Germany; [2]Medical Study & Service Selters GmbH, Selters (Westerwald), Germany; [3]Institute and Comprehensive Center for Inflammation Medicine, University of Lübeck, Lübeck, Germany; [4]Outpatient and Studycenter on the Hase Gbr, Bramsche, Germany

Correspondence: Brigitte Stephan, Institute for Health Services Research in Dermatology and Nursing (IVDP), University Medical Center Hamburg-Eppendorf (UKE), Martinistraße 52, Hamburg, 20251, Germany, Tel +49 40 7410 55428, Email br.stephan@uke.de

Background: Plaque-type psoriasis (PSO) is a chronic inflammatory systemic skin disease. Psoriatic arthritis (PsA) is a frequent component requiring early treatment to prevent joint damage. Guidelines recommend differentiated drug decisions for both conditions.

Objective and Methods: Descriptive analysis of drug choices for patients with PSO with or without additional PsA of the German Psoriasis registry PsoBest from 2007 to 2022.

Results: The analysis comprises data of 17,310 patients with PSO: 18,6% with additional PsA (PSO+PsA), mean age 47.6 (± 14.8) years, 58.8% male, mean duration of PSO 16.4 years in patients without PsA (PSO-PsA; ± 14.3), 20.6 years in PSO+PsA (± 15.3, $p < 0.001$). PSO-PsA and PSO+PsA patients showed a marked burden of disease: PASI (15.7 (± 10.1) and 13.9 (± 10.6, $p < 0.001$)); DLQI (11.7 (± 7.2) and 12.3 (± 7.6; $p < 0.001$)). Before registry entry, 47.0% of patients received no systemic antipsoriatic treatment. Prior systemic medications were mainly non-biologics (40.4%), 12.6% were biologics, with a significantly higher rate in PSO+PsA patients (24.7% vs 9.8%). At registry baseline, the majority of the patients received non-biologic treatment (55.9%), with significantly higher rates for PSO-PsA patients (55.9% vs 34.8%). Biologics were used in 43.9% of all patients, with a significantly higher rate in PSO +PsA patients (65.9% vs 38.8%). Three hundred and three (9.4%) of PSO+PsA patients received treatments at baseline with approval for PSO, but not explicitly for PsA. Those patients had minor active joint involvement.

Conclusion: Early and effective treatment of PsA is crucial to prevent persistent damage of the joints. Although most patients received recommended systemic treatment for PSO+PsA, there is a small number of patients with prescriptions addressing mainly the inflammation of the skin and not explicitly PsA. To choose recommended medication for both entities we need to regard the entire systemic inflammation and interdisciplinary co-working should be implemented.

Keywords: biologics, systemic therapy, skin disease, dermatology

Introduction

Plaque-type psoriasis (PSO) is no longer perceived as a pure skin condition, but as a systemic inflammatory disease with affections on various organ systems.[1–6] The treatment requires inflammatory control adapted to the severity of the disease, which also has to regard the existing comorbidities.[7–11] A major aspect is the arthritic component that occurs in 6–42% of all persons with PSO disease.[12–15] It can be very heterogeneous usually classified into 5 subtypes relating to the joints involved.[16,17] In Germany, two large-scale national studies have shown that 20% of patients with PSO have concomitant PsA and with progress the inflammation can induce severely mutilating joint destruction.[10,12,18–22] Nail

Received: 20 February 2025
Accepted: 1 May 2025
Published: 30 May 2025

psoriasis has been shown to be the strongest predictor of PsA.[23] Early intervention with effective treatment of PSO is therefore particularly important in the presence of concomitant PsA.

Options for systemic treatment of psoriasis disease have changed dramatically during the last decades, and since the first approval of a biologic for PsA in 2003 and for PSO in 2004,[24] the number of disease-modifying anti-rheumatic drugs (DMARDs) increased with up to date more than 12 biologics (bDMARDs) and new targeted synthetic DMARDs like janus kinase inhibitors (tsDMARDs), which are increasingly replacing conventional therapies (csDMARDs) (Augustin et al 2023). The approvals of the medications are different for PSO with and without PsA, and there are also different international possibilities for treatment of PSO disease.[25]

The European Guideline EuroGuiDerm on the systemic treatment of moderate-to-severe PSO recommends appropriate systemic treatment and sufficient disease control with change to more effective biologics in case of treatment failure.[26,27] The recommendations by the European League Against Rheumatism (EULAR) for treatment of PsA also favor a rapid change from conventional anti-inflammatory drugs to biologics for patients with insufficient control of disease activity or poor prognostic factors like polyarthritis, dactylitis or joint damage.[28] Real-world data is needed to reflect the implementation of these guidelines in actual care for this patient group. It is therefore important to analyse registry data for this focus. Rheumatologists and dermatologists handle treatment of patients with PsA differently and the view on both entities has to regard the different point of view of the medical specialties.[21]

This study aimed to analyse real-world data for evidence of guideline-conform treatment of PsA and to focus on differences between systemic therapeutics for patients with PSO with or without PsA.

Materials and Methods

We used data of the German Psoriasis Registry PsoBest (PsoBest) which collects nationwide data of patients with psoriatic disease from over 1,100 dermatological offices and outpatient clinics. Patients with PSO and/or PsA are recruited at the start of a systemic drug, which has been naïve before and followed up to 10 years. The registry oversees up to date (December 2024) data from more than 24,000 patients. PsoBest is part of the ENCePP network and the network of European psoriasis registries (Psonet). It actively participates in the Europe-wide monitoring of the safety and effectiveness of psoriasis therapies. The data are documented using standardized questionnaires to be filled partly by the dermatologist, partly by the patient. Information on previous therapy is obtained from the dermatologist. The drug treatment started at inclusion must not have been used previously.

These descriptive analyses comprised all the quality-ensured data collected from 13th December 2007 (first-patient-in) until 31st December 2022 and therefore a long period including many changes in approval status. It focused on the systemic therapy chosen at baseline related to patient characteristics like sociodemographic data and clinical severity of the inflammatory disease, prevalence of comorbidities and presence of PsA at entry into the registry. It does neither contain follow-up periods nor changes in treating physicians. Patients were assigned to the PSO+PsA group using a pilot tested algorithm, which is also used in the GRAPPA (Group for Research and Assessment of Psoriasis and Psoriatic Arthritis) guideline:[29] Criteria were a) a physician's diagnosis of PsA or b) a probable diagnosis at the time of assessment accompanied by enthesitis or dactylitis. Patients without diagnosis and improbable rating of PsA were referred to as PSO-PsA. Visual analogue scales (VAS) from 0 to 10 were used in the patients and physician's questionnaires to assess the severity of PsA in terms of pain and disease activity.

Pretreatment was assessed directly in the physician's questionnaire.

The groups were described using standard statistical measures: mean and standard deviation for metric data, absolute and relative frequency for categorical data. The groups were compared using Fisher's exact test with a significance level of 0.05 and adjustment according to Bonferroni due to multiple testing. Missing data were not imputed. The analysis was carried out with IBM SPSS version 29.

Results

Patient Characteristics

The analysis includes data from 17,310 patients with a mean age of 47.6 (± 14.8) years and a majority being male (58.8%). Among all patients analysed, 81.4% (n = 14,093) were diagnosed with PSO only (PSO-PsA) and 18.6% (n = 3,217) with

additional PsA (PsO + PsA). Patients with additional PsA had an ensured diagnosis in 82.8%. Mean physician rating of disease activity was 4.7 on 0–10 VAS (± 2.7), patients rated 5.0 (± 2.8) on average. Pain of joints was rated 5.2 (± 2.7) by affected patients.

PSO+PsA patients were significantly less frequently male (50.6%%) than PSO-PsA patients (60.6%, $p < 0.001$). The mean age for PSO-PsA patients was 46.9 years (± 15.1), for PSO+PsA patients significantly higher (50.4 + 13.0, $p < 0.001$). The body mass index was significantly higher in PSO+PsA patients (29.4 + 0.2 vs 28.6 (± 6.0, $p < 0.001$)). About 34.1% (n = 4,729) of PSO-PsA patients showed a body mass index above 30, reflecting obesity. This rate was higher for PSO+PsA patients with 39.8% (n = 1,261). The mean duration of the PSO disease was 16.4 years in PSO-PsA patients (± 14.3) and 20.6 years in PSO +PsA patients (± 15.3, $p < 0.001$).

Mean PASI was 15.7 (± 10.1) in PSO-PsA patients, which was significantly higher than in PSO+PsA patients (13.9, ± 10.6, $p < 0.001$). A majority of patients in both groups showed a marked burden of disease with the severity scores PASI of 10 or more: 66.1% (n = 8,579) of PSO-PsA patients and 56.6% (n = 1,693, $p < 0.001$) of PSO-PsA patients. Mean DLQI was significantly higher in PSO+PsA patients: 12.3 (± 7.6) vs 11.7 (± 7.2, $p < 0.001$). A DLQI > 10, reflecting a markedly reduced health-related quality of life, was found in 53.2% (n = 7,237) of PSO-PsA patients and in 55.1% (n = 1,717) of PSO+PsA patients ($p < 0.049$).

Nail involvement was documented for 44.0% (n = 6,199) PSO-PsA patients and significantly more often in PSO+PsA patients with 59.2% (n = 1,903, $p < 0.001$, Table 1).

Pretreatment

Before registry entry, 47.0% of patients did not receive any systemic antipsoriatic treatment, 40.4% received only non-biologics and the remaining 12.6% received at least one biologic treatment prior to registry entry. Compared to PSO-PsA patients, the rate of non-treated patients was significantly lower in PSO+PsA patients: 28.6 vs 51.2%, $p < 0.001$. The rate of biologic experienced patients was significantly higher: 24.7% in PSO+PsA vs 9.8% in PSO-PsA patients ($p < 0.001$). In line, PSO-PsA patients were exposed to significantly less pretreatments: mean number 1.2 vs 1.9, $p < 0.001$. Patients exposed to nonbiologics prior to the registry entry, most commonly had 1, 2 or 3 pretreaments: 34.3%, 33.7% and 16.7% in PSO-PsA, 27.6%, 29.2% and 19.3% in PSO+PsA (Figure 1a). Biologic experienced PSO-PsA patients received most

Table 1 Patient Characteristics at Registry Baseline for Patients with Psoriasis without (PSO-PsA) and with Psoriatic Arthritis (PSO+PsA) from 13rd December 2007 Till 31st December 2022, n = 17,310

	Total	PSO-PsA	PSO+PsA	p
Patients, % (n)	100.0 (17,310)	81.4 (14,093)	18.6 (3,217)	
Age, mean (SD)	47.6 (14.8)	46.9 (15.1)	50.4 (13.0)	< 0.001
Male: Female*, % (n)	58.8: 41.2 (10,173: 7136)	60.6: 39.4 (8,546: 5,547)	50.6: 49.4 (1,627: 1,589)	< 0.001
BMI > 30, % (n)	35.2 (5,990)	34.1 (4,729)	39.8 (1,261)	< 0.001
BMI [kg/m^2], mean (SD)	28.7 (6.0)	28.6 (6.0)	29.4 (6.2)	< 0.001
PASI, % (n)				< 0.001
Mild (< 10)	32.9 (5,688)	33.9 (4,392)	43.4 (1,296)	
Moderate to severe (≥ 10)	59.3 (10,272)	66.1 (8,579)	56.6 (1,693)	
DLQI, % (n)				0.637
Mild (< 10)	44.9 (7,776)	46.8 (6,378)	44.9 (1,398)	
Moderate to severe (≥ 10)	51.7 (8,954)	53.2 (7,237)	55.1 (1,717)	
Duration of PSO [years], mean (SD)	17.2 (14.6)	16.4 (14.3)	20.6 (15.3)	< 0.001
Nail involvement, % (n)	53.2 (9,208)	44 (6,199)	59.2 (1,903)	< 0.001

https://doi.org/10.2147/PTT.S524083

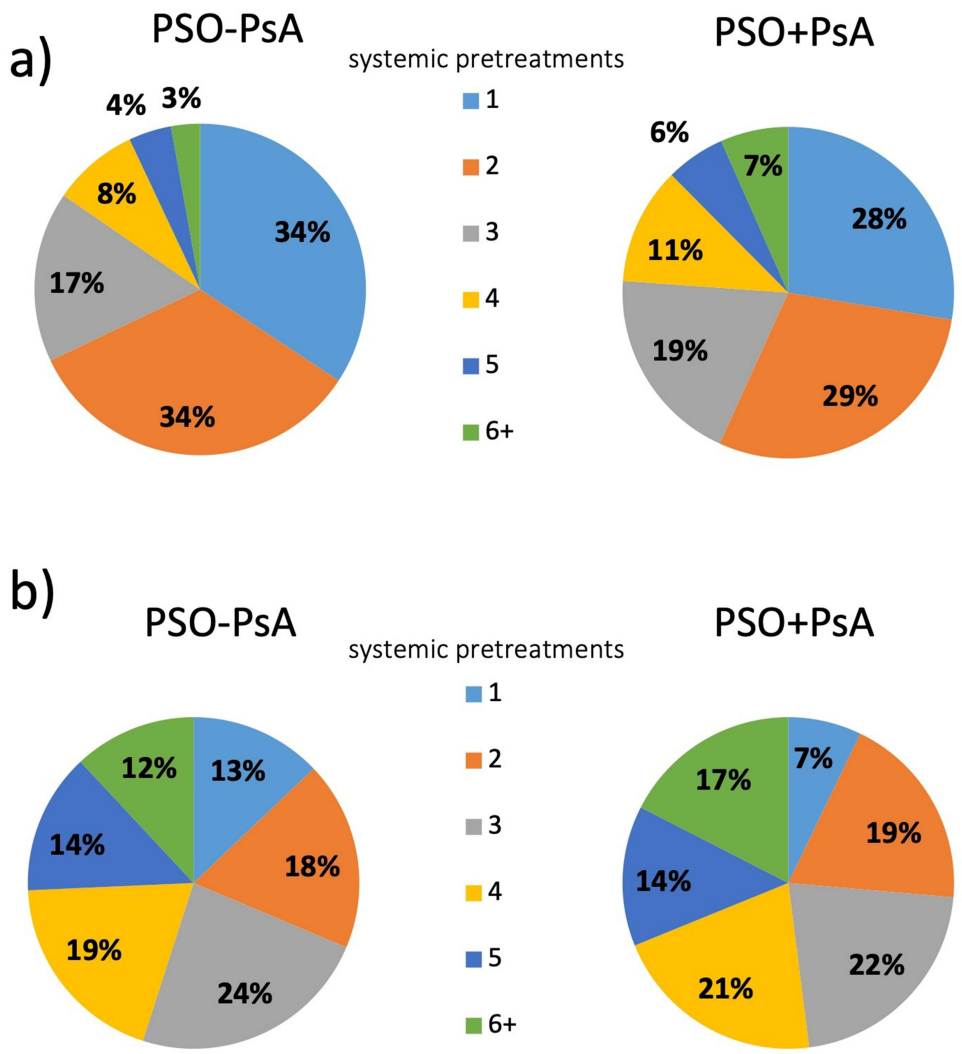

Figure 1 Proportion of patients by number of systemic antipsoriatic pre-treatment before registry inclusion and PsA status for patients exposed to (**a**) prior non-biologics and (**b**) prior biologics.
Note: The number of patients of (**a**) and (**b**) is not additive, as they may have received both biologics and non-biologics as prior therapy.

commonly 3 (23.6%), 4 (19.3%) or 2 (18.5%) pretreatments. In PSO+PsA patients rates were 21.6%, 20.9% and 19.2% (Figure 1b). Figure 2 shows more details regarding the type of biologic pretreatment. Naturally, due to the long data collection period with different drug approvals, TNF-alpha inhibitors are most frequently represented among the biologic therapies: 20.5 vs 6.1% of PSO-PsA and PSO+PsA patients.

Baseline Treatment

For 1,114 out of 17,310 patients analysed (6.4%), no information on the inclusion therapy was available at date of data cut-off. This concerns therapy data that was in the future at the time the visit was completed and has not yet been confirmed by follow-up information.

With 55.9%, the majority of the remaining patients received non-biologic treatment at baseline, mostly fumaric acid esters (28.5%) and methotrexate (20.7%). PSO+PsA patients received non-biologics significantly less frequent compared to PSO-PsA patients (34.8% vs 55.9%, $p < 0.001$). Biologics were used in 43.9% of all patients: 16.4% started an IL-12/23 or IL-23 inhibitor, 14.5% an IL-17 inhibitor and 12.9% TNF-alpha inhibitors (Table 2). When interpreting at the level of individual medicinal products, different authorisation periods and thus also different probabilities of observing the respective medicinal product must be taken into account.

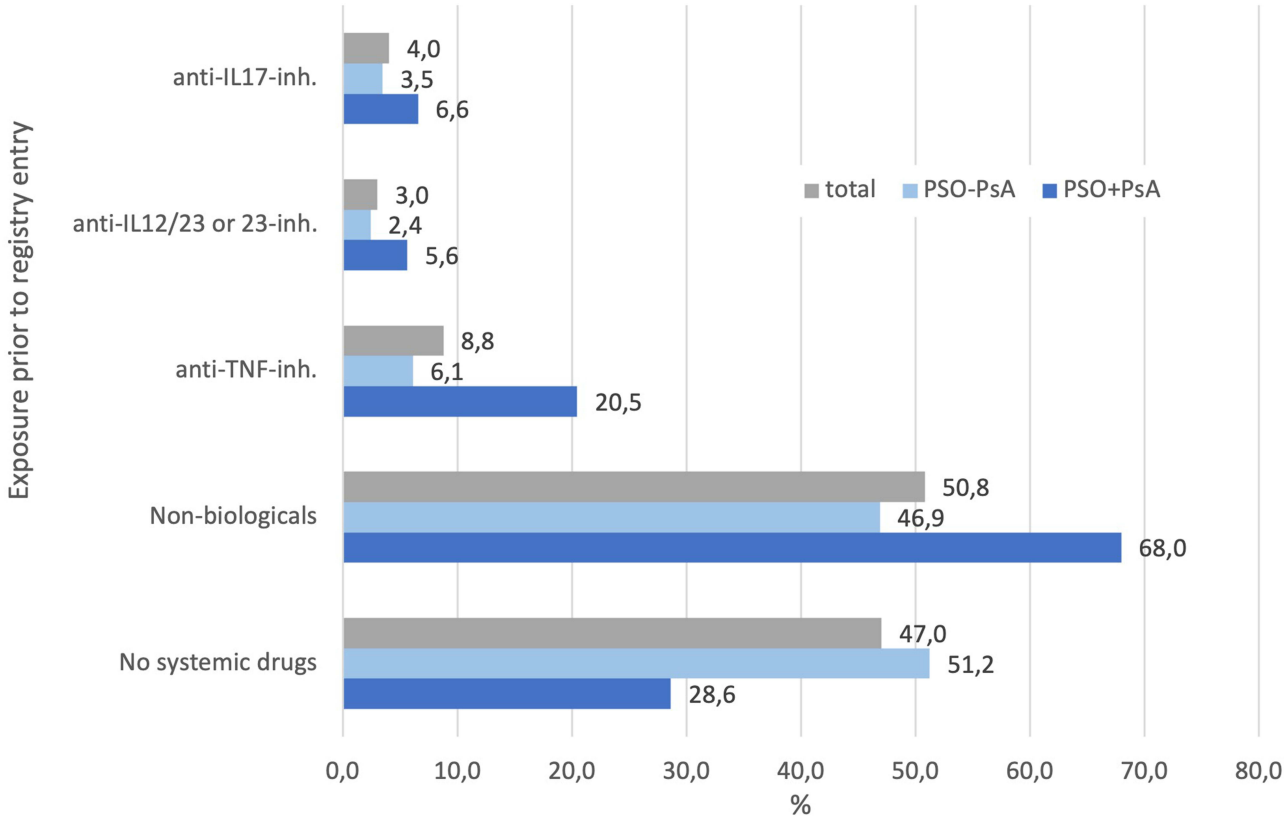

Figure 2 Proportion of patients with systemic antipsoriatic pre-treatment before registry inclusion (PSO-PsA n = 14,093; PSO+PsA n = 3,217; total n = 17,310).

When comparing PSO-PsA and PSO+PsA patients, the latter are significantly more likely to receive biologic therapy when joining the registry (65.9% vs 38.8%, $p < 0.001$). This can also be observed significantly in the subgroups of TNF-alpha inhibitors (24.8% vs 10.2%, $p < 0.001$) and IL-17 inhibitors (23.5% vs 12.4%, $p < 0.001$), with a corresponding but

Table 2 Baseline Treatment Choices for Systemic Therapy of PSO-PsA and PSO+PsA Patients at Entry Into the Registry

Systemic Treatment	Total (n = 16,196)		PSO-PsA (n = 13,163)		PSO+PsA (n = 3,033)		p
	n	%	n	%	n	%	
Biologics, n (%)	**7106**	**43.9**	**5106**	**38.8**	**2000**	**65.9**	**< 0.001**
TNFα- inhibitors, n (%)	2097	12.9	1344	10.2	753	24.8	< 0.001
Adalimumab, n (%)	1510	9.3	996	7.6	514	16.9	< 0.001
Certolizumab, n (%)	118	0.7	75	0.6	43	1.4	< 0.001
Etanercept, n (%)	352	2.2	218	1.7	134	4.4	< 0.001
Golimumab, n (%)	25	0.2	1	< 0.1	24	0.8	< 0.001
Infliximab, n (%)	92	0.6	54	0.4	38	1.3	< 0.001

(*Continued*)

https://doi.org/10.2147/PTT.S524083

Table 2 (Continued).

Systemic Treatment	Total (n = 16,196)		PSO-PsA (n = 13,163)		PSO+PsA (n = 3,033)		p
	n	%	n	%	n	%	
IL-17 Inhibitors, n (%)	2346	14.5	1633	12.4	713	23.5	< 0.001
Brodalumab, n (%)	367	2.3	310	2.4	57	1.9	> 0.999
Ixekizumab, n (%)	572	3.5	355	2.7	217	7.2	< 0.001
Secukinumab, n (%)	1407	8.7	968	7.4	439	14.5	< 0.001
IL-12/23 or IL-23 Inhibitors, n (%)	2664	16.4	2129	16.2	535	17.6	0.765
Guselkumab, n (%)	906	5.6	719	5.5	187	6.2	> 0.999
Risankizumab, n (%)	515	3.2	437	3.3	78	2.6	> 0.999
Tildrakizumab, n (%)	529	3.3	470	3.6	59	1.9	< 0.001
Ustekinumab, n (%)	714	4.4	503	3.8	211	7.0	< 0.001
Non-Biologics, n (%)	**9050**	**55.9**	**7993**	**60.7**	**1057**	**34.8**	**< 0.001**
Apremilast, n (%)	551	3.4	424	3.2	127	4.2	0.162
Ciclosporine, n (%)	372	2.3	332	2.5	40	1.3	0.002
Fumaric acid esters, n (%)	4621	28.5	4438	33.7	183	6.0	< 0.001
Leflunomide, n (%)	10	0.1	1	< 0.1	9	0.3	< 0.001
Methotrexate, n (%)	3360	20.7	2664	20.2	696	22.9	0.011
Retinoid, n (%)	152	0.9	147	1.1	5	0.2	< 0.001
Tofacitinib, n (%)	1	< 0.1	0	< 0.1	1	< 0.1	> 0.999

Notes: The number of patients is not additive, as they may have received more than one therapy, eg adalimumab plus methotrexate. For 1,114 patients, no information on the inclusion therapy was available at time of data cut-off.

not significant trend for IL12/23 or IL-23 inhibitors (17.6% vs 16.2%, $p < 0.765$). Accordingly, non-biologics were used significantly less often in PSO+PsA patients: 34.8% vs 60.7%, $p < 0.001$.

Among PSO+PsA patients, 303 (9.4%) of them received treatments at baseline with approval for PSO, but not explicitly for PsA. Those patients had an ensured diagnosis of PsA in 76.2% of cases. The disease activity was rated at median 5 or lower on 0–10 VAS in 68.2% of patients, which is a clear trend to minor active joint involvement. Accordingly, skin-related disease activity was somewhat higher than expected, with 52.0% of patients with a PASI of or above the median of 13.0.

Discussion

PSO disease is a complex systemic inflammation involving several organ systems and affects in a high proportion of patients the joints. In Germany, about 200,000 patients are estimated to suffer from PsA.[15,30,31] Articular involvement can progress to permanent joint destructing with marked reduction in quality of life.[18,32] The main therapeutic goals are a control of inflammatory activity and reduction of pain, prevention of long-lasting damage and maintaining quality of life.[33] Many patients are predominantly seen by dermatologists and need therefore adjusted systemic treatment for both the skin and the joints, which requires knowledge about approvals and recommendations for the choice of medication.

As promoted by the guidelines, systemic therapy aims to start early in order to prevent late damage. If treatment starts already with first detection of PsA, a favorable outcome is expected.[33–35] Usually, non-steroidal anti-inflammatory drugs (NSAIDs) were the first choice to treat acute inflammation and pain, followed by disease-modifying anti-rheumatic drugs

(DMARDs) with methotrexate as the conventional first choice. This changed during the last years with evidence for better control of inflammation by biologics. The national and international guidelines for treatment of PsA regularly update their recommendations based on study and real-world data for effectiveness of medications on the entire inflammation.

The German guidelines last updated from 2021[7,8] recommend methotrexate as first-line treatment only for peripheral active arthritis with consequent switch to bDMARDs with persistent inflammatory activity. Furthermore, the guideline does not give a recommendation for csDMARDs like ciclosporine or fumaric acid esters, and does not recommend acitretin as first-line treatment for PsA. A high proportion of patients of our cohort with PsA received non-biologic treatments not only before entry into the registry but also with inclusion, although patients showed a marked burden of the disease with the scorings of DLQI and PASI.

The EULAR recommendations from 2023[28] also favor bDMARDs before csDMARDs except for peripheral arthritis, which is only one of the 5 main PsA types previously described, and in cases with low treatment response it should be switched directly to biologics. In case of loss of efficacy of a biologic, the treatment should not be switched back to a csDMARDs but to another biologic. This is different from recommendations of countries[36] where the economical aspect plays a much higher role than in countries with high economical resources. PsA is a disease with a high socioeconomic impact and burden due to its long-lasting character.[37,38] This explains why ciclosporine is promoted as affordable alternative to biologics in cases of low treatment responses. Ciclosporine showed some effect on joint inflammation, but it is not clear if it can prevent long-term damage with treatment so far.[39–41]

The ACR guideline of 2018[42] for the treatment of PsA supports the choice of biologics before csDMARDs and only recommends their use in cases of less severe disease activity and emphasizes the low efficacy of this treatment group. Methotrexate should only be used in treatment-naïve patients and in less severe cases.

Besides the guidelines and recommendations, some drugs only have approval for PSO but not for PsA. This is applicable for fumaric acids esters, which was inclusion treatment for only 6.0% of PSO+PsA patients, and for retinoids (0.2%). Fumaric acid esters could only show little if any effect on PsA[43,44] and are also not very potent for the treatment of PSO compared to bDMARD,[45] but they can offer a good choice for oral systemic therapy in early stages or mild activity of the disease. Interestingly, also the IL17-inhibitor brodalumab (1.9%) and the IL23 inhibitor tildrakizumab (1.9%) have neither the FDA nor the EMA approval for PsA up-to-date (November 2024). Nevertheless, they show efficacy on inflammation of the joints.[46]

With development of new therapeutics, it is crucial to use evidence of comparison studies and real-world date of registries to optimize systemic therapy for the entire disease.[47] Studies found a marked underdiagnosis of PsA among patients with PSO,[18,48,49] perhaps due to the heterogeneity of the clinical appearance. A reliable differentiation of joint complaints with PsA from other origins can be challenging.[50] A screening tool like the GEPARD questionnaire can be of use but might detect PsA not in early stages.[51] Further efforts are made to detect early arthritic activity by screening tools,[52] ultrasound imaging,[53] fluorescence-optical imaging,[54] nailfold capillary assessment[55] or soluble biomarkers.[56] Especially enthesitis seems to help in indicating later PsA.[57] Combined efforts are needed to prevent delayed treatment. Interpretations must take into account that the PSA status is determined at baseline and also applies to this point in time, but that the previous therapies were administered in the past and thus may have been administered before PsA was present or probable. The collaboration of rheumatologists and dermatologists in treatment of PSO patients with joint symptoms is crucial to detect PsA early and to choose the appropriate medication.

Our data show a high proportion of patients with appropriate systemic prescriptions for their psoriasis disease. There is still a number of patients receiving medications with lower efficacy on the inflammation of skin and joints before and at entry into the registry who might need our attention in reaching optimized care. Although we expect comparable effects on the inflammation of the joint from biologics within a group like the IL-17 inhibitors or the IL-23 inhibitors, there exist up to date no approvals for one IL-17 inhibitor and one IL-23 inhibitor on the market for explicitly PsA. In initiating systemic therapies and with the wide range of options for the choice of medications physicians should regard approvals with confirmed study results and efficacy on both entities.

Limitations

The analysis was carried out within an ongoing observation. It can therefore not be guaranteed that all information on the patients' treatment is correct. However, the ongoing quality control in the registry ensures that the data is consistent.

Any comparison of the groups must be interpreted with caution. Due to the high discriminatory power of Fisher's exact test, the differences are often categorised as highly significant for large numbers of cases, even if the differences can be rather marginal. However, the majority of the differences shown are highly significant, are still considered significant even after a very conservative Bonferroni correction and are to be categorised as clinically relevant. PsA was stated by dermatologists at baseline from anamnestic data, and therefore a limitation might be the lacking re-assessment by rheumatologists at baseline.

The long period of 15 years covered by this analysis entails numerous changes to the respective approvals and recommendations of systemic therapeutics for PSO or PsA. Some medications such as adalimumab, had the entire period to be counted as an inclusion therapy. Others were no longer approved as an inclusion therapy during the course of the registry, such as fumaric acid esters, were only counted as inclusion criteria up to and including 2021. Most biologics were approved after 2015 and lower counts relate to less contribution time. Latest approved medications like janus kinase inhibitors also do not have high inclusion rates into the registry so far. However, the aim of this analysis was to look at the entire period and provide a first descriptive insight to this topic without any adjustment for disease severity and different approval periods. Further studies with consideration of the respective inclusion year are planned.

Conclusion

Guideline-conform medication needs to address the entire systemic inflammation. Early and effective treatment of PsA is crucial to prevent persistent damage of the joints. Although a majority of patients received recommended systemic treatment for PSO+PsA, there is a small number of patients with prescriptions addressing mainly the inflammation of the skin and not explicitly PsA. To choose recommended medication for both entities we need to regard the entire systemic inflammation and interdisciplinary co-working should be implemented, as early joint inflammation can rapidly progress to long-lasting damage. This applies for choice of medication by dermatologists as well as rheumatologists and needs a close interdisciplinary collaboration.

Data Sharing Statement

The data supporting this study are not publicly available due to restrictions established by the PsoBest Registry and European legislation.

Ethics Statement

The PsoBest registry received an ethics votum No. 2805 of the ethics committee of the Medical Association of Hamburg on 24 July 2007, last amendment 8 October 2024, and confirmed that there was no further ethical approval necessary for the retrospective analysis of the anonymised data in accordance with the ethical standards of the responsible committees (institutional or regional) and with the Helsinki Declaration of 1975, as revised in 1983.

Informed Consent Statement

Informed consent was obtained from all subjects involved in the PsoBest registry.

Acknowledgments

The authors would like to thank the participating registry sites and patients for their tireless efforts. The authors also thank the Scientific Communication Team of the IVDP, especially Julia Zechlin, Merle Twesten, and Paula Willer, for editing the article. We acknowledge financial support from the Open Access Publication Fund of UKE – Universitätsklinikum Hamburg-Eppendorf and DFG – German Research Foundation.

Funding

The PsoBest registry is/was supported by AbbVie, Almirall, Amgen, Biogen, BMS, Celgene, Hexal, Janssen-Cilag, LEO Pharma A/S, Eli Lilly, Medac, Novartis, Pfizer, UCB and Viatris. These companies do not have influence on the design of the registry, data collection, analyses, the publication decisions or development. This analysis received no further financial support and was conducted by the IVDP with own financial budget. It was part of the doctoral thesis of Secilay Kargin.

Disclosure

BS has received payments/honoraria for lectures, presentations and/or received grants and/or participated in clinical trials including the following companies: AbbVie, Almirall Hermal, Amgen, Beiersdorf, Boehringer Ingelheim, Bristol-Myers-Squibb, Celgene, Celltrion, Glaxo Smith Kline, Janssen-Cilag, LEO Pharma, Lilly, Medac, Novartis, Pierre Fabre, Sanofi Aventis, UCB. MA has received consulting fees and/or speaker honoraria and/or institutional research sup-port from the following pharmaceutical companies manufacturing drugs for psoriasis: AbbVie, Almirall, Amgen, Biogen, Boehringer, Bristol Myers Squibb, Celgene, Celltrion, Centocor, Eli Lilly, Fresenius, GSK, Hexal, Janssen, Klinge, LEO, MC2, Medac, Merck, MSD, Novartis, Pfizer, Sandoz, Sun, UCB and Viatris. CB reports grants and/or personal fees from Amgen, AstraZeneca, Pfizer, Bauerfeind, and Urgo, outside the submitted work. AW has served as a speaker, advisor and/or researcher for AbbVie, Almirall, Amgen, Arctic Bioscience, Biogen, Bristol-Myers-Squibb, Celgene, Hermal, Janssen, LEO, Lilly, Novartis, Pfizer, Sanofi, and UCB. DT is an adviser, speaker, and/or consultant for AbbVie, Almirall, Amgen, Asana Biosciences, Boehringer Ingelheim, Bristol Myers Squibb, Celltrion, Galderma, Janssen, Kyowa Kirin, LEO Pharma, Lilly, New Bridge, Novartis, Pfizer, Regeneron, Samsung, Sanofi/Genzyme, Tamro, Target RWE, UCB, Vichy, and Zuellig Pharma. He is also involved in research for AbbVie, LEO Pharma, and Novartis. RvK provides with his service company CMS3 GmbH consulting services, registry research, activities as an investigator in interventional and non-interventional studies, other services, and scientific lectures for the following companies: AbbVie, ALK Scherax, Almirall Hermal, Amgen. Beiersdorf Dermo Medical, Biofrontera, Biogen, BMS, Boehringer Ingelheim, Celgene, DermaPharm, Foamix, Gilead, Hexal, Janssen-Cilag, LEO Pharma, Lilly Pharma, Medac, Menlo, MSD, Mylan/Viatris, Novartis. CS and SK declare no conflicts of interest in this work.

References

1. Augustin M, Reich K, Glaeske G, Schaefer I, Radtke M. Co-morbidity and age-related prevalence of psoriasis: analysis of health insurance data in Germany. *Acta Derm Venereol*. 2010;90(2):147–151. doi:10.2340/00015555-0770
2. Augustin J, Wolf S, Stephan B, Augustin M, Andrees V. Psoriasis comorbidities in Germany: a population-based study on spatiotemporal variations. *PLoS One*. 2022;17(3):e0265741. doi:10.1371/journal.pone.0265741
3. Loganathan A, Kamalaraj N, El-Haddad C, Pile K. Systematic review and meta-analysis on prevalence of metabolic syndrome in psoriatic arthritis, rheumatoid arthritis and psoriasis. *Int J Rheum Dis*. 2021;24(9):1112–1120. doi:10.1111/1756-185X.14147
4. Maximilian R, Garbe C, Petersen J, et al. Epidemiology, comorbidity and risk factors for psoriatic arthritis: a health insurance claims database analysis. *Acta Derm Venereol*. 2021;101(10):adv00566. doi:10.2340/00015555-3879
5. Mistegård J, Gudbjornsson B, Lindqvist U, et al. Comorbidities in a cohort of 66 patients with psoriatic arthritis mutilans-results from the Nordic PAM Study. *Front Med*. 2021;8:629741. doi:10.3389/fmed.2021.629741
6. Payne K, Ciamponi F, Allen T, et al. Prevalence of multiple long-term conditions with psoriasis in England: a cohort study using the clinical practice research datalink. *JEADV Clin Pract*. 2024;3(1):117–127. doi:10.1002/jvc2.285
7. Nast A, Altenburg A, Augustin M, et al. Deutsche S3-Leitlinie zur Therapie der Psoriasis vulgaris, adaptiert von EuroGuiDerm - Teil 1: therapieziele und Therapieempfehlungen. *J Dtsch Dermatol Ges*. 2021;19(6):934–951. doi:10.1111/ddg.14508_g
8. Nast A, Altenburg A, Augustin M, et al. Deutsche S3-Leitlinie zur Therapie der Psoriasis vulgaris, adaptiert von EuroGuiDerm - Teil 2: therapiemonitoring, besondere klinische Situationen und Komorbidität. *J Dtsch Dermatol Ges*. 2021;19(7):1092–1117. doi:10.1111/ddg.14507_g
9. Elmets CA, Leonardi CL, Davis DMR, et al. Joint AAD-NPF guidelines of care for the management and treatment of psoriasis with awareness and attention to comorbidities. *J Am Acad Dermatol*. 2019;80(4):1073–1113. doi:10.1016/j.jaad.2018.11.058
10. Gisondi P, Bellinato F, Maurelli M, et al. Reducing the risk of developing psoriatic arthritis in patients with psoriasis. *Psoriasis*. 2022;12:213–220. doi:10.2147/PTT.S323300
11. Gisondi P, Altomare G, Ayala F, et al. Consensus on the management of patients with psoriatic arthritis in a dermatology setting. *J Eur Acad Dermatol Venereol*. 2018;32(4):515–528. doi:10.1111/jdv.14741
12. Gladman DD, Antoni C, Mease P, Clegg DO, Nash P. Psoriatic arthritis: epidemiology, clinical features, course, and outcome. *Ann Rheum Dis*. 2005;64(Suppl 2):ii14–7. doi:10.1136/ard.2004.032482
13. Ritchlin CT, Colbert RA, Gladman DD. Psoriatic Arthritis. *N Engl J Med*. 2017;376(10):957–970. doi:10.1056/NEJMra1505557
14. Pala E, Melikoğlu M, Karaşahin Ö, Alkan melikoğlu M. The frequency of association of nail involvement and psoriatic arthritis in psoriasis patients. *Eurasian J Med*. 2023;55(2):158–164. doi:10.5152/eurasianjmed.2023.53

15. Alinaghi F, Calov M, Kristensen LE, et al. Prevalence of psoriatic arthritis in patients with psoriasis: a systematic review and meta-analysis of observational and clinical studies. *J Am Acad Dermatol*. 2019;80(1):251–265.e19. doi:10.1016/j.jaad.2018.06.027
16. Moll JM, Wright V. Psoriatic arthritis. *Semin Arthritis Rheum*. 1973;3(1):55–78. doi:10.1016/0049-0172(73)90035-8
17. McHugh NJ, Balachrishnan C, Jones SM. Progression of peripheral joint disease in psoriatic arthritis: a 5-yr prospective study. *Rheumatology*. 2003;42(6):778–783. doi:10.1093/rheumatology/keg217
18. Radtke MA, Reich K, Blome C, Rustenbach S, Augustin M. Prevalence and clinical features of psoriatic arthritis and joint complaints in 2009 patients with psoriasis: results of a German national survey. *J Eur Acad Dermatol Venereol*. 2009;23(6):683–691. doi:10.1111/j.1468-3083.2009.03159.x
19. Reich K, Krüger K, Mössner R, Augustin M. Epidemiology and clinical pattern of psoriatic arthritis in Germany: a prospective interdisciplinary epidemiological study of 1511 patients with plaque-type psoriasis. *Br J Dermatol*. 2009;160(5):1040–1047. doi:10.1111/j.1365-2133.2008.09023.x
20. Ogdie A, Weiss P. The epidemiology of psoriatic arthritis. *Rheum Dis Clin North Am*. 2015;41(4):545–568. doi:10.1016/j.rdc.2015.07.001
21. Augustin M, Lindner L, Kühl L, et al. Charakterisierung von Patienten mit Psoriasisarthritis in der dermatologischen und rheumatologischen Versorgung: analyse von zwei Registern: characterization of patients with psoriatic arthritis in dermatologic and rheumatologic care: analysis of two registries. *J Dtsch Dermatol Ges*. 2023;21(10):1170–1178. doi:10.1111/ddg.15178_g
22. Lindqvist U, Gudbjornsson B, Iversen L, et al. Disease activity in and quality of life of patients with psoriatic arthritis mutilans: the Nordic PAM Study. *Scand J Rheumatol*. 2017;46(6):454–460. doi:10.1080/03009742.2017.1278787
23. Langenbruch A, Radtke MA, Krensel M, Jacobi A, Reich K, Augustin M. Nail involvement as a predictor of concomitant psoriatic arthritis in patients with psoriasis. *Br J Dermatol*. 2014;171(5):1123–1128. doi:10.1111/bjd.13272
24. Rich SJ, Bello-Quintero CE. Advancements in the treatment of psoriasis: role of biologic agents. *J Manag Care Pharm*. 2004;10(4):318–325. doi:10.18553/jmcp.2004.10.4.318
25. Papapetropoulos A, Topouzis S, Alexander SPH, et al. Novel drugs approved by the EMA, the FDA, and the MHRA in 2023: a year in review. *Br J Pharmacol*. 2024;181(11):1553–1575. doi:10.1111/bph.16337
26. Nast A, Smith C, Spuls PI, et al. EuroGuiDerm guideline on the systemic treatment of psoriasis vulgaris - part 1: treatment and monitoring recommendations. *J Eur Acad Dermatol Venereol*. 2020;34(11):2461–2498. doi:10.1111/jdv.16915
27. Mrowietz U, de Jong EMGJ, Kragballe K, et al. A consensus report on appropriate treatment optimization and transitioning in the management of moderate-to-severe plaque psoriasis. *J Eur Acad Dermatol Venereol*. 2014;28(4):438–453. doi:10.1111/jdv.12118
28. Gossec L, Kerschbaumer A, Ferreira RJO, et al. EULAR recommendations for the management of psoriatic arthritis with pharmacological therapies: 2023 update. *Ann Rheum Dis*. 2024;83(6):706–719. doi:10.1136/ard-2024-225531
29. Coates LC, Soriano ER, Corp N, et al. Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA): updated treatment recommendations for psoriatic arthritis 2021. *Nat Rev Rheumatol*. 2022;18(8):465–479. doi:10.1038/s41584-022-00798-0
30. Albrecht K, Binder S, Minden K, et al. Systematisches Review zur Schätzung der Prävalenz entzündlich rheumatischer Erkrankungen in Deutschland [Systematic review to estimate the prevalence of inflammatory rheumatic diseases in Germany. German version]. *Z Rheumatol*. 2023;82(9):727–738. doi:10.1007/s00393-022-01305-2
31. Hagenström K, Müller K, Garbe C, Augustin M. Prevalence of psoriasis and psoriatic arthritis in Germany - analysis of claims data. *J Dtsch Dermatol Ges*. 2024;22(1):45–54. doi:10.1111/ddg.15269
32. Kane D, Stafford L, Bresnihan B, FitzGerald O. A prospective, clinical and radiological study of early psoriatic arthritis: an early synovitis clinic experience. *Rheumatology*. 2003;42(12):1460–1468. doi:10.1093/rheumatology/keg384
33. Mease PJ, Armstrong AW. Managing patients with psoriatic disease: the diagnosis and pharmacologic treatment of psoriatic arthritis in patients with psoriasis. *Drugs*. 2014;74(4):423–441. doi:10.1007/s40265-014-0191-y
34. Gladman DD, Thavaneswaran A, Chandran V, Cook RJ. Do patients with psoriatic arthritis who present early fare better than those presenting later in the disease? *Ann Rheum Dis*. 2011;70(12):2152–2154. doi:10.1136/ard.2011.150938
35. Richard M-A, Barnetche T, Rouzaud M, et al. Evidence-based recommendations on the role of dermatologists in the diagnosis and management of psoriatic arthritis: systematic review and expert opinion. *J Eur Acad Dermatol Venereol*. 2014;28(Suppl 5):3–12. doi:10.1111/jdv.12560
36. Pal P, Giri PP, Sinha R. Cyclosporine in resistant systemic arthritis - a cheaper alternative to biologics. *Indian J Pediatr*. 2019;86(7):590–594. doi:10.1007/s12098-019-02912-9
37. Sondermann W, Ventzke J, Matusiewicz D, Körber A. Analyse der pharmazeutischen Versorgungssituation von Patienten mit Psoriasis-Arthritis auf Basis von Routinedaten der Gesetzlichen Krankenversicherung. *J Dtsch Dermatol Ges*. 2018;16(3):285–296. doi:10.1111/ddg.13464_g
38. Graier T, Salmhofer W, Jonak C, et al. Evolution of characteristics and biologic treatment effectiveness in patients of the Austrian psoriasis registry from 2004–2022. *J Dtsch Dermatol Ges*. 2023;21(12):1513–1523. doi:10.1111/ddg.15213
39. Colombo D, Chimenti S, Grossi PA, et al. Efficacy of cyclosporine A as monotherapy in patients with psoriatic arthritis: a subgroup analysis of the Synergy Study. *G Ital Dermatol Venereol*. 2017;152(3):297–301. doi:10.23736/S0392-0488.16.05301-3
40. Mrowietz U, Klein CE, Reich K, et al. Cyclosporine therapy in dermatology. *J Dtsch Dermatol Ges*. 2009;7(5):474–479. doi:10.1111/j.1610-0387.2009.07077.x
41. Soriano A, Pipitone N, Salvarani C. Cyclosporine in psoriatic arthropathy. *Clin Exp Rheumatol*. 2015;33(5 Suppl 93):S101–3.
42. Singh JA, Guyatt G, Ogdie A, et al. Special article: 2018 American College of Rheumatology/National Psoriasis Foundation guideline for the treatment of psoriatic arthritis. *Arthritis Rheumatol*. 2019;71(1):5–32. doi:10.1002/art.40726
43. Atwan A, Ingram JR, Abbott R, et al. Oral fumaric acid esters for psoriasis. *Cochrane Database Syst Rev*. 2015;2015(8):CD010497. doi:10.1002/14651858.CD010497.pub2
44. Balak DM. Fumaric acid esters in the management of psoriasis. *Psoriasis*. 2015;5:9–23. doi:10.2147/PTT.S51490
45. Sbidian E, Chaimani A, Garcia-Doval I, et al. Systemic pharmacological treatments for chronic plaque psoriasis: a network meta-analysis. *Cochrane Database Syst Rev*. 2021;4(4):CD011535. doi:10.1002/14651858.CD011535.pub4
46. Golbari NM, Basehore BM, Zito PM. Brodalumab. Available from: https://www.ncbi.nlm.nih.gov/books/NBK470324/. Accessed May 21, 2025.
47. Mease PJ, Menter MA. Quality-of-life issues in psoriasis and psoriatic arthritis: outcome measures and therapies from a dermatological perspective. *J Am Acad Dermatol*. 2006;54(4):685–704. doi:10.1016/j.jaad.2005.10.008
48. Mease PJ, Gladman DD, Papp KA, et al. Prevalence of rheumatologist-diagnosed psoriatic arthritis in patients with psoriasis in European/North American dermatology clinics. *J Am Acad Dermatol*. 2013;69(5):729–735. doi:10.1016/j.jaad.2013.07.023

49. Henes JC, Ziupa E, Eisfelder M, et al. High prevalence of psoriatic arthritis in dermatological patients with psoriasis: a cross-sectional study. *Rheumatol Int*. 2014;34(2):227–234. doi:10.1007/s00296-013-2876-z
50. Rech J, Sticherling M, Stoessel D, Biermann MHC, Häberle BM, Reinhardt M. Psoriatic arthritis epidemiology, comorbid disease profiles and risk factors: results from a claims database analysis. *Rheumatol Adv Pract*. 2020;4(2):rkaa033. doi:10.1093/rap/rkaa033
51. Härle P, Letschert K, Wittig B, Mrowietz U. Sensitivity of the GEPARD patient questionnaire to identify psoriatic arthritis in patients with psoriasis in daily practice: the GEPARD-life study. *Dermatology*. 2016;232(5):597–605. doi:10.1159/000448029
52. Mishra S, Kancharla H, Dogra S, Sharma A. Comparison of four validated psoriatic arthritis screening tools in diagnosing psoriatic arthritis in patients with psoriasis (COMPAQ Study). *Br J Dermatol*. 2017;176(3):765–770. doi:10.1111/bjd.14929
53. Crespo-Rodríguez AM, Sanz Sanz J, Freites D, Rosales Z, Abasolo L, Arrazola J. Role of diagnostic imaging in psoriatic arthritis: how, when, and why. *Insights Imaging*. 2021;12(1):121. doi:10.1186/s13244-021-01035-0
54. Koehm M, Ohrndorf S, Foldenauer AC, et al. Fluorescence-optical imaging as a promising easy-to-use imaging biomarker to increase early psoriatic arthritis detection in patients with psoriasis: a cross-sectional cohort study with follow-up. *RMD Open*. 2022;8(2):e002682. doi:10.1136/rmdopen-2022-002682
55. Fukasawa T, Toyama S, Enomoto A, et al. Utility of nailfold capillary assessment for predicting psoriatic arthritis based on a prospective observational cohort study. *Rheumatology*. 2023;62(7):2418–2425. doi:10.1093/rheumatology/keac664
56. Cretu D, Gao L, Liang K, Soosaipillai A, Diamandis EP, Chandran V. Differentiating psoriatic arthritis from psoriasis without psoriatic arthritis using novel serum biomarkers. *Arthritis Care Res*. 2018;70(3):454–461. doi:10.1002/acr.23298
57. Hum RM, Barton A, Ho P. Utility of musculoskeletal ultrasound in psoriatic arthritis. *Clin Ther*. 2023;45(9):816–821. doi:10.1016/j.clinthera.2023.07.017