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The path to the medical standard of treatment for patients with psoriatic arthritis — a practical guide

ABSTRACT

Rapid advances in pharmacotherapy have resulted in significant changes in recommendations for the treatment of psoriatic arthritis (PsA) in recent years. The most recent guidelines for the treatment of PsA are the 2019 recommendations of the European League Against Rheumatism (EULAR) and the 2021 Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA). The wide range of currently available modern drugs provides ample opportunity to select an effective therapy individually for each patient. Psoriatic arthritis (PsA) is a very heterogeneous disease, so the choice of a particular drug should be tailored to the needs of the individual patient. When making therapeutic decisions, it is crucial to identify the predominant musculoskeletal symptoms; however, the coexistence of extra-articular symptoms, such as skin lesions and nail involvement, and other concomitant conditions must also be taken into account. This paper presents current recommendations for the treatment of PsA in Poland, with a particular focus on biologic treatment.

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KEY WORDS: psoriatic arthritis; treatment; biologics

INTRODUCTION

Psoriatic arthritis (PsA) is an autoimmune rheumatic disease characterised by the coexistence of inflammatory joint lesions with psoriasis (PsO) [1]. Due to its clinical picture and the absence of a rheumatoid factor, according to the most frequently used classification of rheumatic diseases that was proposed in 1983 by the American Rheumatism Association (ARA — now the American College of Rheumatology — ACR), PsA is classified as a type II rheumatic disease, i.e. arthritis with spondylitis (so-called seronegative spondyloarthropathies) [2]. This is a group of diseases with a complex clinical picture, involving inflammation of the peripheral joints but also changes in the axial skeleton (sacroiliitis and spondylarthritis). In the course of spondyloarthritides, arthritis is often also accompanied by changes in the skin, eyes or intestines, which is also evident in the case of PsA [3].

EPIDEMIOLOGY OF PSA

In the European population, the prevalence of PsA is estimated to be approximately 2–3% of the general population. However, according to various literature sources, the prevalence of coexisting arthritis in PsO patients ranges from a few to more than 40%, indicating a prevalence of PsA in approximately 0.3–1% of the general population. However, it is possible that these data are still underestimated [3]. To date, no indicator has been identified that definitively predicts the onset of arthritis in PsO patients. There has also been no clear correlation between the extent of skin lesions and the development of PsA. In addition, it is also possible to have PsA without PsO itself — that is, cases of arthritis without obvious skin involvement [4]. The disease usually begins between 20 and 50 years of age and occurs with similar frequency in both sexes (except for the axial form of PsA, which is more common in men) [3].

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PATHOGENESIS OF PSA

The aetiology of PsA has not been clearly defined. Genetic and environmental factors have been cited among the causes responsible for the development of the disease [5, 6]. The more frequent familial occurrence of the disease suggests the influence of heredity. Also, studies involving twins indicate a genetic predisposition in the development of both PsO itself and PsA. It is likely that the inheritance of PsO and PsA is polygenic. Most data have been obtained from studies of the human leukocyte antigen (HLA) system genes. The strongest association with PsO was found for the *HLA-Cw6* gene (carriers of the HLA-Cw-0602 allele were found to have a significantly higher risk of developing psoriasis compared to the general population, and additionally the presence of this allele was associated with an earlier onset and more severe course of the disease). However, the role of the Cw-06 allele in PsA patients is not well understood. An association between PsA and the presence of the HLA-B27 antigen has also been suggested. However, candidate genes for the diagnosis of PsA are still not clearly identified — therefore, genetic testing is not recommended in routine clinical practice [5]. Environmental factors associated with the development of psoriatic lesions include smoking, poor diet, use of certain medications (e.g., β-blockers, interferon), mechanical trauma or stress [6]. Bacterial infections are also likely to play a role in the pathogenesis of PsA [7]. The involvement of viral infections has also been considered [8]. Stimulation of the development of PsO and PsA by infections is likely to be mediated by so-called superantigens, which may be bacterial or viral antigens [9].

It is likely that the influence of adverse environmental factors, combined with a susceptible genetic background, leads to immune activation involving, inter alia, T lymphocytes and pro-inflammatory cytokines, which act through osteoclastogenesis and stimulation of mature osteoclasts [10, 11]. Under physiological conditions, bone — as an active tissue — undergoes constant remodelling. Bone-synthesising cells, the osteoblasts, and bone-resorbing cells, the osteoclasts, are involved in this process. In PsA, this balance is disrupted. A more detailed understanding of bone changes in the course of PsA has become possible through the identification of factors responsible for bone remodelling: receptor activator of nuclear factor-κB (κB-RANK), its ligand (RANKL, receptor activator of nuclear factor-κB ligand) and osteoprotegrin (OPG). The RANK/RANKL/OPG system plays an important role in osteoclast activation and osteolysis. RANKL, by binding to RANK, induces osteoclastogenesis and inhibits apoptosis of mature osteoclasts. Osteoprotegrin is a naturally occurring antagonist of RANKL. The main role of osteoprotegrin is to bind and subsequently neutralise RANKL, thus preventing osteolysis [10, 12]. Inflammation in PsA probably leads to a disruption of this balance. Pro-inflammatory cytokines such as interleukins (IL): IL-1β, IL-6, IL-8, IL-11, IL-17, interferon gamma (INF-γ) or tumour necrosis factor alpha (TNFα), increase RANKL expression while inhibiting osteoprotegerin production. As a result, a higher RANKL/OPG ratio is observed in PsA, leading to excessive osteolytic activity and the bone changes characteristic of PsA [11, 13].

CLINICAL PICTURE OF PSA

Typically, the symptoms of PsA develop gradually and discreetly. The disease usually progresses with periods of exacerbation and remission [14]. Arthritis usually appears some time after the skin lesions, although sometimes (in approximately 15–20% of cases) it may be the first symptom. In contrast to rheumatoid arthritis (RA), asymmetry of joint lesions is typical. The involved joints are usually swollen, there is pain on pressure, sometimes also morning stiffness and restricted joint mobility. However, rheumatoid nodules are not observed [14].

The clinical picture of PsA varies from discrete inflammatory lesions of a single joint to massive destructive multiple joint lesions. Lesions may involve axial or peripheral joints. There are several forms of PsA according to the predominant clinical picture (Tab. 1) [15].

In addition, the clinical picture of PsA may also include lesions in other organs, such as iritis (less commonly choroiditis) or inflammatory bowel disease (IBD) [16]. Importantly, the components of the metabolic syndrome (obesity, diabetes mellitus, hypertension, and lipid disorders) are found more frequently in PsA patients compared to the general population. This results in a significant increase in cardiovascular risk in this patient group [17].

Table 1. Clinical forms of psoriatic arthritis (PsA)

Disease form	Notes
Asymmetric arthritis	Most common, typical form of PsA
Symmetric arthritis	Form which is clinically similar to rheumatoid arthritis
Axial form	With involvement of sacroiliac joints and spine
Form with predominant involvement of the distal interphalangeal joints	Form which is restricted to distal interphalangeal joints
Destructive arthritis	So-called *arthritis mutilans*, involving distal and proximal interphalangeal joints

Table 2. Diagnostic criteria for psoriatic arthritis (PsA) according to 2006 CASPAR criteria

2006 CASPAR criteria
1. Psoriatic skin lesions present (2 points) or nail psoriasis (1 pt) or family history of psoriasis (1 pt)
2. *Dactylitis* (1 pt)
3. Periarticular proliferative lesions on radiographic imaging (1 pt)
4. Negative serum rheumatoid factor (1 pt)
A minimum score of 3 points is necessary for diagnosis

DIAGNOSIS OF PSA

The diagnosis of PsA is often made with some delay, due to the initially discrete joint symptoms that are dominated by skin manifestations. Great diagnostic difficulties are also presented by "PsA without PsO", i.e., cases of arthritis without obvious skin involvement [1]. Currently, CASPAR criteria (classification criteria for psoriasis arthritis) of 2006 are used to diagnose PsA. According to these criteria, the diagnosis of PsA requires the presence of active arthritis and a score of at least three points for the following criteria (Tab. 2) [18].

The diagnostic criteria according to CASPAR are relatively simple to apply and have a sensitivity and specificity of 91% and 98%. As part of these criteria, it has also become possible to diagnose atypical-onset (without active skin lesions) and early-onset (without radiographic changes) PsA [18].

ASSESSMENT OF PSA ACTIVITY

Due to the heterogeneous clinical presentation of PsA, there is no single, universal method that can be used to assess disease activity. The choice of individual parameters to be assessed depends on the form of PsA to cover as many aspects of the disease as possible. Adequate assessment of disease activity is very important to select appropriate therapy and monitor treatment effects [15]. According to the GRAPPA recommendations, the scales shown in Table 3 are applicable for assessing the activity of the peripheral form of PsA [19].

In clinical practice, however, the most commonly used methods are those originally developed to assess the activity of RA (DAS-28) — for the peripheral form, and ankylosing spondylitis (BASDAI) — for the axial form (Tab. 4) [1].

It is most often the DAS-28 and BASDAI scales that are used in GP practices. Also, an overall assessment of disease activity by physician and patient on a five-point Likert scale is used to facilitate assessment. However, it is important to remember that accurate assessment of disease activity is critical for therapeutic decision-making and necessary for monitoring treatment effects [1].

TREATMENT OF PSA

In the treatment of PsA, as in all rheumatic diseases, the key is to make the diagnosis as soon as possible and initiate effective treatment that results in the resolution of clinical symptoms and prevents the progression of structural changes in the musculoskeletal system and the development of organ complications. The aim of therapy is to achieve remission or at least low disease activity within 3–6 months, according to the treat-to-target principle, and then maintain this state. This often requires combined therapy and the involvement of multiple specialists. Interdisciplinary collaboration is important, such as with a gastroentrologist (if IBD coexists), an ophthalmologist (for uveitis), a dermatologist (to confirm and treat PsO). Optimal management of PsA patients also requires non-pharmacological strategies, such as patient education and regular physical exercise. However, pharmacotherapy remains the cornerstone [20, 21].

Table 3. Summary of parameters included in individual scales for assessing disease activity in peripheral psoriatic arthritis (PsA)

Tested parameter	DAS-28	PsA-DAS	CPDAI	DAPSA	GRACE	MDA
Laboratory markers of inflammation (CRP or ESR)	Yes	Yes	No	Yes	No	No
Number of swollen joints	Yes	Yes	Yes	Yes	Yes	Yes
Number of painful joints	Yes	Yes	Yes	Yes	Yes	Yes
Patient's assessment of disease activity (patient's VAS)	Yes	Yes	No	Yes	Yes	Yes
Physician's assessment of disease activity (physician's VAS)	No	Yes	No	No	No	No
Enthesitis	No	Yes	Yes	No	No	Yes
Dactylitis	No	Yes	Yes	No	No	No
Extent and severity of skin lesions	No	No	Yes	No	Yes	Yes
Functional changes and quality of life	No	Yes	Yes	No	Yes	Yes

DAS-28 — 28-joint count Disease Activity Score; PsA-DAS — Psoriatic Arthritis Disease Activity Score DAS); CPDAI — Composite Psoriatic Disease Activity Index; DAPSA — Disease Activity Index for Psoriatic Arthritis; GRACE — GRAPPA Composite Exercise Index; MDA (Minimal Disease Activity); CRP — C-reactive protein; ESR — erythrocyte sedimentation rate; VAS — visual analogue scale

Table 4. Psoriatic arthritis (PsA) activity scores according to the Disease Activity Score-28 (DAS-28) and the Bath Ankylosing Spondylitis Disease Activity Index (BASDAI)

DAS-28 scale for peripheral forms of PsA	BASDAI scale for axial forms of PsA
< 2.6 disease remission 2.6–3.2 — low disease activity > 3.2–5.1 — moderate disease activity — **with coexisting unfavourable prognostic factors, biological treatment to be considered** > 5.1 — high disease activity — **biological treatment to be considered**	> 4 — suggests high disease activity, **biological treatment to be considered**

Table 5. Drug classes that are used in clinical practice in the treatment of psoriatic arthritis (PsA) in Poland

Drug class	Notes
Non-steroidal anti-inflammatory drugs (NSAIDs)	Selective and non-selective cyclooxygenase inhibitors
Glucocorticosteroids (GCs)	Topically applied
Conventional synthetic disease-modifying antirheumatic drugs (csDMARDs)	Most commonly: methotrexate, or leflunomide, sulfasalazine Alternatively: cyclosporine A, azathioprine or chloroquine/hydroxychloroquine
Biologic disease-modifying antirheumatic drugs (bDMARDs)*	Tumour necrosis factor alpha (TNFα) inhibitors: adalimumab, certolizumab pegol, etanercept, golimumab, infliximab Interleukin-17 (IL-17) inhibitors: ixekizumab, secukinumab Interleukin-23 (IL-23) inhibitors: guselkumab, risankizumab
Janus kinase inhibitors (JAKi)*	Tofacitinib, upadacitinib

*refunded under the B35 drug programme

Psoriatic arthritis (PsA) is a heterogeneous disease, so the choice of treatment should be tailored individually for each patient. When making therapeutic decisions, it is crucial to identify the predominant musculoskeletal symptoms, as the treatment of axial symptoms is different from that of peripheral symptoms (arthritis, enthesitis, dactylitis). The coexistence of extra-articular symptoms, such as skin lesions and nail involvement, and other concomitant conditions, such as cardiovascular diseases, must also be considered. The wide range of currently available drugs provides ample opportunity to select the appropriate therapy for a given clinical situation [20, 21]. The drug classes used in clinical prac-

Table 6. European League Against Rheumatism (EULAR) recommendations for the treatment of psoriatic arthritis (PsA)

2019 EULAR recommendations	
1	Treatment should aim to achieve remission or low disease activity by regular monitoring of disease activity and appropriate adjustment of therapy
2	NSAIDs may be used in the relief of subjective and objective musculoskeletal symptoms
3	Topical injectable GCs should be considered as adjunctive treatment in PsA; systemic GCs can be used with caution, at the lowest effective dose
4	In patients with polyarthritis, treatment with csDMARDs should be initiated promptly, with methotrexate being the preferred drug in patients with significant skin lesions
5	In patients with monoarthritis or oligoarthritis, especially with adverse prognostic factors such as structural damage, high ESR/CRP levels, dactylitis or nail involvement, treatment with csDMARDs should be considered
6	In patients with peripheral arthritis and an inadequate response to at least one csDMARD, treatment with bDMARDs should be initiated, with IL-17 inhibitor or IL-12/23 being the preferred drugs in those with significant skin lesions
7	In patients with peripheral arthritis and an inadequate response to at least one csDMARD and at least one bDMARD, and where bDMARD treatment is inappropriate, a JAKi should be considered
8	In patients with mild disease and an inadequate response to at least one csDMARD, where both bDMARD treatment and JAKi treatment are inappropriate, a PDE4 inhibitor may be considered
9	In patients with unequivocal enthesitis and an inadequate response to NSAIDs or topical injectable GCs, bDMARD treatment should be considered
10	In patients with active dominant axial form of the disease and an inadequate response to NSAIDs, a bDMARD — which, according to current practice, is a TNF inhibitor — should be considered; in those with significant skin lesions, an IL-17 inhibitor may be the preferred drug
11	In patients without an adequate response to a bDMARD or with intolerance to a bDMARD, a change to another bDMARD or csDMARD* should be considered, including one change to a drug from the same group†
12	In patients in sustained remission, careful gradual de-escalation of DMARD therapy may be considered

NSAIDs — non-steroidal anti-inflammatory drugs; GCs — glucocorticosteroids; csDMARDs — conventional synthetic disease-modifying antirheumatic drugs; ESR — erythrocyte sedimentation rate; CRP — C-reactive protein; bDMARDs — biologic disease-modifying antirheumatic drugs; csDMARDs — conventional synthetic disease-modifying antirheumatic drugs; TNF — tumour necrosis factor; IL — interleukin; JAK — janus kinase; PDGE4 — phosphodiesterase 4

tice for the treatment of PsA in Poland are shown in Table 5.

The latest recommendations for the treatment of PsA are the 2019 EULAR recommendations (Tab. 6) [20] and the 2021 GRAPPA (Fig. 1) [21].

Both EULAR and GRAPPA recommendations recognise the use of NSAIDs as first-choice drugs, especially if inflammation affects one or more joints in the peripheral or axial form [20, 21]. However, the mechanism of action of this group of drugs can control or reduce the complaints arising from PsA only in patients with less active disease [22].

Topical injectable GCs are still sometimes used as adjunctive therapy in limited forms of the disease, for example, *dactylitis* [20]; however, they are unlikely to be recommended in more recent GRAPPA guidelines [21]. Therefore, the use of GCs should be limited to specific cases only, in the form of topical injections. When used systemically, GCs raise concerns about the exacerbation of skin lesions, although data on this are inconclusive. However, the cautious use of systemic GCs is recommended and GCs are unlikely to be recommended for the treatment of PsA [20, 21].

In patients with polyarticular forms of PsA and monoarthritis or oligoarthritis, but with the presence of unfavourable prognostic factors (such as the presence of laboratory markers of inflammation, visible structural damage on imaging studies, dactylitis or nail involvement), prompt initiation of treatment with conventional synthetic disease-modifying antirheumatic drugs (csDMARDs) such as methotrexate, sulfasalazine and leflunomide is recommended. All these drugs are widely used in Poland. In patients with severe skin lesions, methotrexate is usually preferred [20]. Less commonly, cyclosporine A, azathioprine or chloroquine/hydroxychloroquine are used in specific cases, as their efficacy has been fully documented [23].

BIOLOGICAL AND INNOVATIVE TREATMENT

Unfortunately, in some PsA patients — despite the use of pharmacotherapy with csDAMRDs, satisfactory treatment outcomes

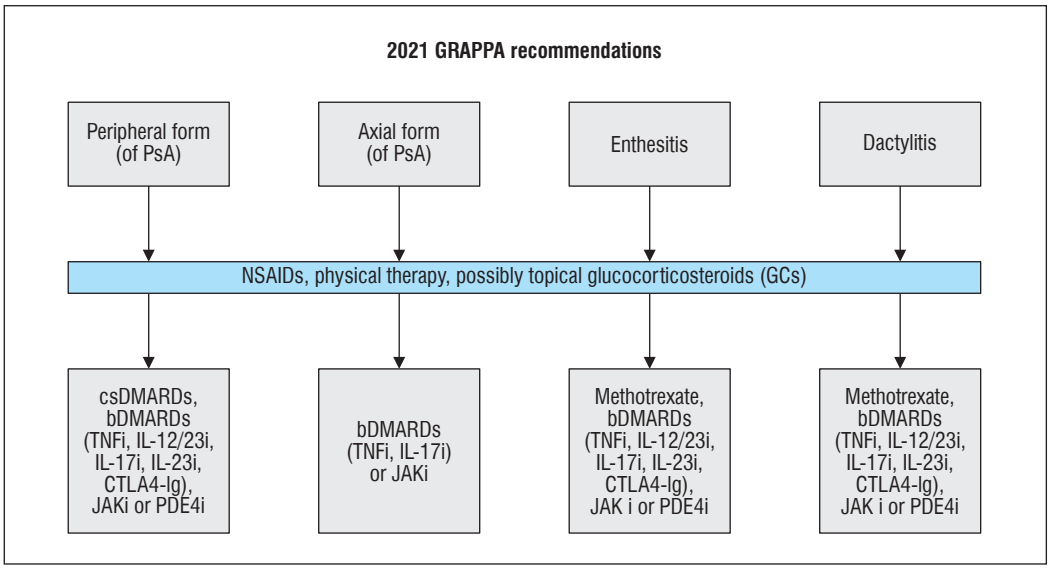

Figure 1. Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA) recommendations for the treatment of psoriatic arthritis (PsA) NSAIDs — non-steroidal anti-inflammatory drugs; csDMARDs — conventional synthetic disease-modifying antirheumatic drugs; bDMARDs — biologic disease-modifying antirheumatic drugs; i — inhibitor; TNF — tumour necrosis factor; IL — interleukin; **CTLA4-Ig — ???;** JAK — Janus kinase; PDE4 — phosphodiesterase 4

cannot be achieved. Although the duration of treatment necessary for the therapy to be considered ineffective has not been clearly defined in the case of PsA (as in the case of RA), this should be a period of time appropriate to the profile of the drug in question. This period is usually about 3 months. The most advanced therapy lines are dedicated to this group of patients: biologics and targeted synthetic DMARDs (tsDMARDs) [20, 21]. In the Polish setting, however, it is standard practice for a patient to be eligible for a drug programme only after failure of at least two csDMARDs used for 3 months each (in peripheral form) or two NSAIDs used for 4 weeks each (in axial form). It should be noted that, unlike in RA, in the case of PsA there are no uniform methods that can be used to assess disease activity and define treatment failure. Consequently, PsA can be considered active according to different assessment methods — therefore, it is important to select the appropriate method to assess disease activity depending on the predominant clinical presentation, as mentioned previously [19]. In addition, some special situations are allowed in the choice of treatment for PsA. When the main symptom of PsA is *enthesitis* or *dactylitis*, biological or targeted synthetic treatment should be considered, even if csDMARDs have not been used previously and NSAID therapy has not been successful. These recommendations are based on literature data showing that csDMARDs do not achieve satisfactory treatment outcomes in cases of *enthesitis* or *dactylitis* [20, 21]. For the axial form of PsA, it is also not advisable to take csDMARDs before choosing the next line of therapy (biologics or tsDMARDs) used immediately after NSAIDs. This is a recommendation carried over from recommendations for ankylosing spondylitis [22]. Current recommendations also recommend considering biologics or tsDMARDs when csDAMRDs are poorly tolerated [20, 21].

The choice of drug (biologics and tsDMARDs) in the next line of therapy is up to the clinician — there are no clear guidelines as to which drug to prefer. This prompts clinicians to analyse each case in detail and make individual choices [20, 21]. The choice of drugs is wide, with original biologics, bioequivalent drugs (corresponding to reference drugs in terms of quality, efficacy, safety profile and immunogenicity) and tsDMARDs [currently represented by JAKis] that are currently available for the treatment of PsA. The following drugs are currently available in drug programmes in Poland:

— TNFα inhibitors: adalimumab, certolizumab pegol, etanercept, golimumab, infliximab;
— interleukin-17 (IL-17) inhibitors: ixekizumab, secukinumab;
— interleukin 23 (IL-23) inhibitors: guselkumab, risankizumab;
— JAKis: tofacitinib, upadacitinib.

The choice of the appropriate drug depends not only on the clinical presentation of the disease and comorbidities, but also on patient preference and economic considerations.

In patients with significant skin lesions, IL-17 or IL-12/23 inhibitors may be the preferred drugs. In contrast, in patients with active dominant axial form of the disease, according to current practice, a TNFα inhibitor is the most commonly chosen first-choice drug. Similarly, TNFα inhibitors are preferred when IBD and/or uveitis coexist. When treatment with a biologic is inappropriate, a JAKi is most often chosen as first-line therapy. In patients without an adequate response to one drug, a change to another drug should be considered, including — most often — one change to a drug from the same group, followed by a choice of subsequent drug mechanisms of action [20, 21]. Unlike in RA, in the case of PsA — based on the currently available literature — it is not possible to conclude unequivocally that the combination of a csDMARD with biological treatment is more effective than monotherapy, although this combination is most often recommended and used [1]. In patients who have long-term remission, a reduction in the dose of the drug or a prolongation of the interval between doses may be considered [20, 21].

DRUG PROGRAMMES IN POLAND

In Poland, all biologics and tsDMARDs are available to patients free of charge (reimbursement by the National Health Fund — NFZ) only under drug programmes. The B35 programme is available for patients with active forms of PsA [in the International Classification of Diseases and Related Health Problems (ICD-10, 10th Revision): L40.5, M07.1, M07.2, M07.3]). The current drug programme provisions are published on the website of the Ministry of Health (https://www.gov.pl/web/zdrowie/choroby-nieonkologiczne) [24]. It is always advisable to make sure of the current provisions, which strictly define the eligibility criteria for treatment. However, in special situations, by decision of the Coordination Team for Biological Treatment in Rheumatic Diseases, a patient may be eligible for treatment in the event that some of the criteria described in the programme are not met if the treatment is in line with current recommendations and medical knowledge. Therefore, it is worth considering such a route and submitting a non-standard application directly to the Coordination Team for Biological Treatment in Rheumatic Diseases to allow more patients to receive therapy. Follow-up and monitoring of the patient during therapy is also strictly defined by the provisions of the drug programmes, so it is necessary to be aware of their current contents during the course of therapy. The entire eligibility and treatment process must be documented on the Therapeutic Programme Monitoring System (System Monitorowania Programów Terapeutycznych, SMPT) platform (https://csm-swd.nfz.gov.pl/) [25].

Unfortunately, the restriction of biologics and tsDMARDs to use only within drug programmes means that the availability of innovative therapies in Poland is still at a very low level. It is estimated that only 1.8% of Poles with PsA use biological treatment [26].

The authors of this study hope that drug programmes will be further modified to ensure greater accessibility to modern therapies in Poland for patients with rheumatic diseases. Due to the loss of patent protection of key TNFα inhibitors in rheumatology, such as adalimumab, etanercept and infliximab, bioequivalent drugs are the fastest growing group of therapies and offer the greatest hope for lower therapy costs [27]. Based on the current provisions of the Reimbursement Act, which qualify the inclusion of a bioequivalent drug in reimbursement and oblige the responsible entity to reduce the price of the bioequivalent drug by at least 25% compared to the price of the reference drug, it can be expected that the cumulative savings for the public payer from the introduction of bioequivalent drugs for monoclonal antibodies and fusion/soluble receptor proteins will be more than EUR 100 million per year in Poland. This gives some hope for the future but will require some systemic changes that are related to the limitations of drug programmes used in Poland [28]. The medical community hopes, among other things, for a further relaxation of the criteria for including patients in drug programmes (possibility of therapy after the failure of 1 csDMARD for patients with poor prognostic factors), which will allow therapy to be aligned with global guidelines [20]. It may also be interesting to include biologics and bioequivalent drugs in outpatient treatment, beyond drug programmes, as is the case in many European countries. An increase in the number of centres providing biological treatment is an opportunity for greater access to this therapy for patients. It seems that further new drugs will also be available in Poland in the near future, which have found their place in the latest recommendations for the treatment of PsA devel-

oped by GRAPPA in 2021. More therapeutic options have been included in the GRAPPA recommendations, covering further groups of drugs, including IL-23 and cytotoxic T-lymphocyte antigen 4 (CTLA-4) inhibitors [21]. All of this gives hope for even more effective treatment of PsA in the future.

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D.S., W.S. — manuscript preparation.

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CONFLICT OF INTEREST

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References

1. Samborski W, Brzosko N. Łuszczycowe zapalenie stawów — diagnostyka i leczenie. Uniwersytet Medyczny im. K. Marcinkowskiego w Poznaniu, Poznań 2014.
2. Decker JL. American Rheumatism Association nomenclature and classification of arthritis and rheumatism (1983). Arthritis Rheum. 1983; 26(8): 1029–1032, doi: 10.1002/art.1780260813, indexed in Pubmed: 6603849.
3. Gladman DD, Antoni C, Mease P, et al. Psoriatic arthritis: epidemiology, clinical features, course, and outcome. Ann Rheum Dis. 2005; 64 Suppl 2(Suppl 2): ii14–ii17, doi: 10.1136/ard.2004.032482, indexed in Pubmed: 15708927.
4. Elkayam O, Ophir J, Yaron M, et al. Psoriatic arthritis: interrelationships between skin and joint manifestations related to onset, course and distribution. Clin Rheumatol. 2000; 19(4): 301–305, doi: 10.1007/pl00011173, indexed in Pubmed: 10941813.
5. Winchester R, FitzGerald O. The many faces of psoriatic arthritis: their genetic determinism. Rheumatology (Oxford). 2020; 59(Suppl 1): i4–i9, doi: 10.1093/rheumatology/kez325, indexed in Pubmed: 32159794.
6. Caso F, Costa L, Chimenti MS, et al. Pathogenesis of Psoriatic Arthritis. Crit Rev Immunol. 2019; 39(5): 361–377, doi: 10.1615/CritRevImmunol.2020033243, indexed in Pubmed: 32422017.
7. Weisenseel P, Laumbacher B, Besgen P, et al. Streptococcal infection distinguishes different types of psoriasis. J Med Genet. 2002; 39(10): 767–768, doi: 10.1136/jmg.39.10.767, indexed in Pubmed: 12362037.
8. Taglione E, Vatteroni ML, Martini P, et al. Hepatitis C virus infection: prevalence in psoriasis and psoriatic arthritis. J Rheumatol. 1999; 26(2): 370–372, indexed in Pubmed: 9972971.
9. Skov L, Baadsgaard O, Olsen JV, et al. Superantigens. Do they have a role in skin diseases? Arch Dermatol. 1995; 131(7): 829–832, doi: 10.1001/archderm.131.7.829, indexed in Pubmed: 7611801.
10. Hofbauer LC, Khosla S, Dunstan CR, et al. The roles of osteoprotegerin and osteoprotegerin ligand in the paracrine regulation of bone resorption. J Bone Miner Res. 2000; 15(1): 2–12, doi: 10.1359/jbmr.2000.15.1.2, indexed in Pubmed: 10646108.
11. Ritchlin CT, Haas-Smith SA, Li P, et al. Mechanisms of TNF-alpha- and RANKL-mediated osteoclastogenesis and bone resorption in psoriatic arthritis. J Clin Invest. 2003; 111(6): 821–831, doi: 10.1172/JCI16069, indexed in Pubmed: 12639988.
12. Hsu H, Lacey DL, Dunstan CR, et al. Tumor necrosis factor receptor family member RANK mediates osteoclast differentiation and activation induced by osteoprotegerin ligand. Proc Natl Acad Sci U S A. 1999; 96(7): 3540–3545, doi: 10.1073/pnas.96.7.3540, indexed in Pubmed: 10097072.
13. Ritchlin C, Haas-Smith SA, Hicks D, et al. Patterns of cytokine production in psoriatic synovium. J Rheumatol. 1998; 25(8): 1544–1552, indexed in Pubmed: 9712099.
14. Gladman DD. Psoriatic arthritis. Rheum Dis Clin North Am. 1998; 24(4): 829–844, x, doi: 10.1016/s0889-857x(05)70044-2, indexed in Pubmed: 9891713.
15. Szechiński J, Garwolińska H, Bernacka K. Spondyloartropatie. Reumatologia. 2000; 38(Suppl): 68–73.
16. Makredes M, Robinson D, Bala M, et al. The burden of autoimmune disease: a comparison of prevalence ratios in patients with psoriatic arthritis and psoriasis. J Am Acad Dermatol. 2009; 61(3): 405–410, doi: 10.1016/j.jaad.2009.02.015, indexed in Pubmed: 19700012.
17. Gladman DD, Ang M, Su L, et al. Cardiovascular morbidity in psoriatic arthritis. Ann Rheum Dis. 2009; 68(7): 1131–1135, doi: 10.1136/ard.2008.094839, indexed in Pubmed: 18697777.
18. Taylor W, Gladman D, Helliwell P, et al. CASPAR Study Group. Classification criteria for psoriatic arthritis: development of new criteria from a large international study. Arthritis Rheum. 2006; 54(8): 2665–2673, doi: 10.1002/art.21972, indexed in Pubmed: 16871531.
19. Tillett W, McHugh N, Orbai AM, et al. Outcomes of the 2019 GRAPPA Workshop on Continuous Composite Indices for the Assessment of Psoriatic Arthritis and Membership-recommended Next Steps. J Rheumatol Suppl. 2020; 96: 11–18, doi: 10.3899/jrheum.200121, indexed in Pubmed: 32482762.
20. Gossec L, Baraliakos X, Kerschbaumer A, et al. EULAR recommendations for the management of psoriatic arthritis with pharmacological therapies: 2019 update. Ann Rheum Dis. 2020; 79(6): 700–712, doi: 10.1136/annrheum-dis-2020-217159, indexed in Pubmed: 32434812.
21. Coates LC, Soriano ER, Corp N, et al. GRAPPA Treatment Recommendations domain subcommittees. 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, indexed in Pubmed: 35761070.
22. van der Heijde D, Ramiro S, Landewé R, et al. 2016 update of the ASAS-EULAR management recommendations for axial spondyloarthritis. Ann Rheum Dis. 2017; 76(6): 978–

991, doi: 10.1136/annrheumdis-2016-210770, indexed in Pubmed: 28087505.
23. Ash Z, Gaujoux-Viala C, Gossec L, et al. A systematic literature review of drug therapies for the treatment of psoriatic arthritis: current evidence and meta-analysis informing the EULAR recommendations for the management of psoriatic arthritis. Ann Rheum Dis. 2012; 71(3): 319–326, doi: 10.1136/ard.2011.150995, indexed in Pubmed: 21803753.
24. Obwieszczenie Ministra Zdrowia z dnia 20 lutego 2023 r. w sprawie wykazu leków, środków spożywczych specjalnego przeznaczenia żywieniowego oraz wyrobów medycznych na 1 marca 2023 r.. https://www.gov.pl/web/zdrowie/obwieszczenie-ministra-zdrowia-z-dnia-20-lutego-2023-r-w-sprawie-wykazu-lekow-srodkow-spozywczych-specjalnego-przeznaczenia-zywieniowego-na-1-marca-2023-r..
25. https://csm-swd.nfz.gov.pl/.
26. Yin L, Zhao H, Zhang H, et al. Remdesivir Alleviates Acute Kidney Injury by Inhibiting the Activation of NLRP3 Inflammasome. Front Immunol. 2021; 12: 652446, doi: 10.3389/fimmu.2021.652446, indexed in Pubmed: 34093539.
27. Du Y, Zong M, Guan Q, et al. Comparison of mesenchymal stromal cells from peritoneal dialysis effluent with those from umbilical cords: characteristics and therapeutic effects on chronic peritoneal dialysis in uremic rats. Stem Cell Res Ther. 2021; 12(1): 398, doi: 10.1186/s13287-021-02473-9, indexed in Pubmed: 34256856.
28. Yang X, Bao M, Fang Yi, et al. STAT3/HIF-1α signaling activation mediates peritoneal fibrosis induced by high glucose. J Transl Med. 2021; 19(1): 283, doi: 10.1186/s12967-021-02946-8, indexed in Pubmed: 34193173.