Psoriatic arthritis (PsA) is a chronic inflammatory skeletal and muscular disease associated with psoriasis, which usually manifests itself in the peripheral arthritis, dactylitis, enthesitis and/or spondylitis. Early identification and diagnosis of PsA, as well as early start of therapy is important for improving the long-term outcomes of the disease. Clinical picture of PsA is heterogeneous, and doctors often face problems while determining treatment strategies. The purpose of our review was to discuss and interpret 2018 American College of Rheumatologists (ACR) and National Psoriasis Foundation (NPF) guidelines for active PSA treatment in adults using pharmacological and non-pharmacological methods. These recommendations may help both clinicians and patients to make their best decisions about disease management. The presence or absence of comorbid conditions such as inflammatory bowel disease, uveitis, diabetes or serious infections, as well as knowledge of medical history are factors enabling choice of an optimal therapy for a particular patient at a given time using a customized treatment approach. ACR/NPF guidelines for active PsA treatment recommend tumor necrosis factor inhibitors rather than small molecule oral drugs as a first-line therapy, precisely because these biological agents prevent progression of the disease and joint damage. Early and aggressive therapy is emphasized in patients with a newly diagnosed PsA.

Keywords: psoriatic arthritis; psoriasis; activity of the process; recommendations; treatment; management; tumor necrosis factor inhibitors; interleukin-17 inhibitors; interleukin-12/23 inhibitors; methotrexate; treat-to-target
distal phalangeal joints (DPJs) are a pathognomonic sign as sometimes they are the only ones bearing PsA marks. Axial spondyloarthritis usually occurs together with a peripheral arthritis. Some patients may have a quickly progressing and debilitating PsA; this PsA is also called mutilating.

Table 1 presents PsA clinical forms.

**Diagnostic criteria**
PsA is diagnosed according to CASPAR (Classification Criteria for Psoriatic Arthritis) established by W. Taylor et al. in 2006 [7]. These criteria have a high sensitivity and specificity both at an early and advanced PsA stage. These criteria enable diagnostics irrespective of a positive RA and absence of psoriasis, if typical PsA signs are present.

According to CASPAR, patients should have inflammatory joint conditions (arthritis, spondylitis or enthesitis) and ≥3 points out of those presented in Table 2.

PsA clinical picture is rather heterogeneous, which is why physicians rarely reach consensus as to the treatment options. We aim to review the American College of Rheumatologists (ACR) Guidance of 2018 on how to treat an active PsA in adults with pharmacological and non-pharmacological methods. Those recommendations may help both clinicians and patients to make optimum decisions about PsA management.

**Determination of PsA activity and severity; effectiveness of treatment**
Treatment guidance mainly concerns an active PsA form. While determining PsA activity, we should take into account at least one of the following symptoms: arthritis, dactyliitis, enthesitis, axial skeleton damage, active skin and/or nail lesions, extra-articular manifestations such as uveitis or inflammatory bowel disease (IBD). It is also essential to pay attention to the following inflammation markers: C-reactive protein (CRP) or erythrocyte sedimentation rate (ESR), and joint and bone imaging [8].

To evaluate the effectiveness of treatment, randomized controlled studies primarily use ACR criteria [9]:

**Table 1. PsA clinical forms**

| PsA clinical form                                      | Primary characteristics                                                                 |
|--------------------------------------------------------|-----------------------------------------------------------------------------------------|
| Distal form                                            | Primarily affects hand and foot DPJs. Classic isolated hand and/or foot DPJs is observed in 5 % of cases. Affected DPJs often occur in other clinical forms as well. |
| Asymmetric mono- and oligoarthritis                    | Typical of most PsA patients (up to 70 %). Primarily affects knee, radius, ankle and elbow joints; also hand and foot proximal phalangeal joints (PPJs), metacarpophalangeal and metatarsophalangeal joints. The total number of affected joints is ≤ 4. |
| Rheumatoid-reshsembling symmetric polyarthritis        | Occurs in 15-20% PsA patients, Characterized by pairs of joints being affected, as in RA. Often manifests itself as an asymmetric polyarthritis of ≥ 5 joints |
| Psoriatic spondylitis (isolated or in connection with a peripheral arthritis) | Characterized by a generalized spinal (spondylitis), sacroiliac inflammation (sacroilitis) similar to ankylosing [rheumatoid] spondylitis. However, most often sacroilitis is unilateral; 50 % of cases have it combined with a peripheral arthritis. Isolated spondylitis is rare (2-4%) |
| Mutilating arthritis                                   | A rare clinical form observed in 5 % of cases. Characterized by a wide-ranging joint surface resorption (osteolysis), shortening of fingers and/or toes, ‘telescopic’ deformation; multidirectional subluxations (windblown fingers). However localized (restricted) osteolysis may develop in all clinical forms |

**Notes:** DPJs - distal phalangeal joints; PPJs – proximal phalangeal joints; PsA - psoriatic arthritis; RA – rheumatoid arthritis.

**Table 2. CASPAR**

| Signs                                                                 | Points |
|-----------------------------------------------------------------------|--------|
| 1. Psoriasis: At the examination Patient’s history Family history     | 2      |
| 2. Psoriatic nail dystrophy (pinpoint marks, onycholysis, hyperkeratosis) | 1      |
| 3. Negative RF (but for latex test)                                   | 1      |
| 4. Dactyliitis: finger swollen at the examination Patient’s history   | 1      |
| 5. X-ray reveals abarticular bone proliferation similar to boundary growth (but for osteophytes) of hand and foot | 1      |
1. Number of painful joints (NPJ) (out of 68);  
2. Number of swollen joints (NSJ) (out of 66);  
3. General evaluation of disease activity by the physician using a visual analogue scale (VAS), mm;  
4. General evaluation of disease activity by the patient using a visual analogue scale (VAS), mm;  
5. Joint pain evaluation by the patient using a visual analogue scale (VAS), mm;  
6. Evaluation of functional ability loss (Health Assessment Questionnaire, HAQ);  
7. Acute stage indices (CRP, ESR).  

ACR20 criterion shows a 20 %-improvement of at least 5 out of 7 above-mentioned parameters (improvement of the first two is a must) compared to the initial level, i.e. a satisfactory therapeutic effect; ACR50 criterion shows a 50 %-improvement, i.e. a good therapeutic effect; ACR70 criterion shows a 70 %-improvement, i.e. an exemplary therapeutic effect. If the EULAR criteria of effectiveness are used in every day medical practice, then ACR criteria are primarily reserved for the research papers and clinical studies [10]. DAS index helps determine the degree of disease activity while its dynamics with treatment gives physicians an idea as to the treatment effectiveness rate. At the same time, ACR criteria could only prove or disprove treatment-based positive dynamics [11].

Patients and physicians should determine PsA and psoriasis activity in every special case, taking into account not only a degree of activity at a specific point in time but presence or absence of negative prognosticating factors in the long term. If the disease is found to be severe, it should have at least one of the negative prognosticating factors:  
- Erosive process;  
- Dactylitis;  
- Increased levels of PsA-related inflammatory markers (CRP, ESR);  
- Irrevocable joint damage;  
- Function-compromising joint deformations;  
- High disease activity provoking serious life quality issues (for instance, active inflammation at numerous sites, including dactylitis, enthesitis, restriction of several joint group functions);  
- Long-term impairment (for instance, vision loss);  
- Quick progress of the disease;  
- Active psoriasis infiltration at numerous sites.

As nowadays there are no agreed psoriasis severity markers, it should be determined by the physician and patient in every special case. In clinical studies, psoriasis severity and proliferation (area) index (PASI) ≥12 is combined with the damaged body surface area (BSA) ≥10 [12]. Examples of PsA and psoriasis severity are presented in Table 3. Finally, as the National Psoriasis Foundation (NPF) and the American Academy of Dermatology (AAD) are both elaborating the psoriasis treatment guidance, it’s not included on the recent ACR PsA guidance.

From guideline to practice  
The Guidelines of ACR/NFP’2018 were developed in order to help the healthcare staff to choose the best therapeutic option for PsA patients. Presence or absence of co-morbidities such as uveitis, inflammatory bowel disease (IBD), diabetes and severe infections, as well as awareness of medication history influence the PsA management strategy. In PsA case, there should be an objective examination of peripheral joints (for instance, to diagnose dactylitis), spine, skin and nails, evaluation of existing enthesiases. Physicians and patients should take into account existing co-morbidities and patient’s functional state at the time of decision-making as to the optimal therapy.

Early PsA identification and diagnostics as well as the early therapeutic intervention are of extreme importance for improving long-term disease outcomes [13]. Both pharmacological and non-pharmacological treatment (Fig. 1) may reduce PsA symptoms and signs and bring remission [14]. Nowadays clinicians and patients may choose out of a great range of pharmacological cures, among them symptomatic (non-steroid anti-inflammatory drugs (NSAIDs), systemic glucocorticoids (GCs), local (intra-articular) GC injections) and immune-modulating, among them oral small-molecule drugs (OSMDs), tumor necrosis inhibitor drugs (TNF inhibitors), interleukin inhibitors (IL inhibitors), immunoglobulin, janus kinase inhibitors (JK inhibitors).

### Table 3. Criteria of severe PsA and severe psoriasis

| Severe psoriatic arthritis | Severe psoriasis |
|---------------------------|------------------|
| • Erosive disease         | • PASI > 12      |
| • Increased levels of PsA-related inflammatory markers (CRP, ESR) | • BSA > 5-10% |
| • Irrevocable joint damage compromising their function | • Significant lesions of isolated sites (face, hands, feet, nails, folds, scalp) affecting function and life quality |
| • Highly active disease affecting life quality | • Deterioration of physical or mental state results in diagnosing moderate or severe psoriasis despite the small damage area |
| • Active inflammation at numerous sites, including dactylitis, enthesitis | |
| • Restricted function at several sites | |
| • Quickly progressing disease | |

Notes: PASI – Psoriasis Area and Severity Index; BSA – Body Surface Area Composite Tool for Assessing Psoriasis; CRP - C-reactive protein; PsA – psoriatic arthritis; ESR - erythrocyte sedimentation rate.
ACR/NFP’2018 Guidelines on PsA treatment

Active PsA in patients not receiving treatment (Fig. 2) [15-28].

For the treatment of PsA patients who didn’t receive any previous therapy (naïve), biological agents and TNF inhibitors in particular are considered the line of first defense. Basic drugs, such as Methotrexate (MTX), Sulfasalazine (SSZ), Cyclosporine, Leflunomide, might be used instead of TNF inhibitors in case of a non-severe PsA form and absence of severe psoriasis (as Table 3 puts it, the degree of severity is decided upon by the physician and patient), for those patients who prefer to take drugs per os rather than that by parenteral way, or have TNF inhibitor contraindications, such as congestive heart failure, previous severe or recurring infections, demyelinating diseases, casting doubt on the biological therapy benefit.

To treat active PsA in patients who didn’t receive any PsA-related therapy, it’s necessary to choose TNF inhibitors or basic therapy and small-molecule drugs over IL inhibitors – 17 or IL inhibitors – 12/23. The latter may replace TNF inhibitors in patients with a severe psoriasis and TNF inhibitor contraindications. Between IL inhibitors – 17 and IL inhibitors – 12/23, the former are more effective while the latter may serve as protectors against an inflammatory bowel disease (IBD) and/or severe psoriasis and/or severe PsA or in case a patient wishes the drug to be administered rarely.

If the choice is between NSAIDs and MTX, it’s suggested that MTX works better for active PsA or psoriasis patients. NSAIDs may be used instead MTX after a review of all possible contraindications and evaluation of side effects in patients without a severe PsA or psoriasis, and those with no risk of NSAID-associated cardio- and nephrotoxic effects.

Active PsA despite basic drugs administered per os (Fig. 3).

Some patients retain an active PsA despite taking basic drugs (Methotrexate (MTX), Sulfasalazine (SSZ), Cyclosporine, Leflunomide). In this case the healthcare providers should suggest TNF inhibitors, IL inhibitors – 17 or IL inhibitors – 12/23 rather than another basic drug. However, those patients who prefer to take drugs per os or have no signs of a severe PsA or psoriasis may still try another basic therapy option rather than TNF inhibitors, IL inhibitors – 17 or IL inhibitors – 12/23. It is worthy of note that another basic therapy option (Methotrexate (MTX), Sulfasalazine (SSZ), Cyclosporine, Leflunomide) may be prescribed in case of biological drug contraindications.

As it was previously mentioned, among the biological drugs TNF inhibitors have an edge over IL inhibitors – 17 or IL inhibitors – 12/23, Apremilast or Tofacitinibum. While choosing among those, keep in mind that IL inhibitors – 17 are more effective than IL inhibitors – 12/23, Abataceptum or Tofacitinibum while IL inhibitors – 12/23 – more effective than Abataceptum or Tofacitinibum [31]. Tofacitinibum may be substituted for TNF inhibitors in those patients who prefer to take drugs per os or have no signs of a severe psoriasis [32].

Switching to a new basic therapy option is a better strategy than adding a new one belonging to the same group (but for Apremilast). It is suggested only if a patient gave a partial response to the current per os basic therapy. In this case, adding Apremilast to the current basic therapy is more effective than administering Apremilast as a mono-therapy. Most studies of Apremilast’s effectiveness concern combined therapies with Methotrexate (MTX), Sulfasalazine (SSZ), Leflunomide [33]. Mono-therapy is preferred to a combined therapy only if a patient has significant side effects associated with the current basic medication (Methotrexate (MTX), Sulfasalazine (SSZ), Cyclosporine, Leflunomide).

It is interesting that a biological mono-therapy turns out more effective than a combined one, if MTX is used together with biological agents. While switching to a biological mono-therapy, one should stop taking basic medication if it’s a justified option chosen by both patient and healthcare provider [34]. Biological agent in combination with MTX replaces mono-therapy if a patient has a severe psoriasis, makes a partial response to a current MTX therapy or has a concomitant uveitis (this condition is MTX-treatable). Patients getting TNF inhibitors, especially Infliximab or Adalimumab, are recommended the combined therapy in order to delay or prevent antibody production.

### Table 3: Non-steroid anti-inflammatory drugs, glucocorticoids, local glucocorticoid injections

| Non-pharmacological treatment | Physiotherapy, occupational therapy, smoking cessation, weight loss, massage, physical exercise |
|------------------------------|-------------------------------------------------------------------------------------------------|
| Symptomatic therapy          |                                                                                                 |
| Basic therapy and small molecules | Methotrexate (MTX), Sulfasalazine (SSZ), Cyclosporine, Leflunomide, Apremilast                               |
| TNF inhibitors               | Etanercept, Infliximab, Adalimumab, Golimumab, Certolizumab                                     |
| IL inhibitors – 12/23        | Ustekinumab                                                                                      |
| IL inhibitors – 17           | Secukinumab, Ixekizumab, Brodalumab                                                           |
| CTLA4-Ig                     | Apremilast                                                                                      |
| Janus kinase inhibitors      | Tofacitinibum                                                                                    |

Fig. 1. PsA pharmacological and non-pharmacological treatment. Pharmacological methods are highlighted in blue and involve a basic therapy and small-molecule drugs, TNF inhibitors, interleukin-17 inhibitors (IL inhibitors – 17), interleukin-12/23 inhibitors (IL inhibitors – 12/23), CTLA4-immunoglobulin and Janus kinase inhibitor. Despite numerous non-pharmacological methods, only 6 are considered the most effective: physiotherapy, occupational therapy, smoking cessation, weight loss, massage, physical exercise.
Active PsA despite TNF inhibitors administered both as a mono- and combined therapy (Fig. 4).

Patients who retain PsA despite being treated by TNF inhibitor mono-therapy should be switched to a different TNF inhibitor rather than to IL inhibitors – 17 or IL inhibitors – 12/23, Abataceptum or Tofacitinibum. It is not advisable to add MTX to a current TNF inhibitor. In case of a complete absence of clinical effect from a previous TNF inhibitor or its significant side effects, IL inhibitors – 17 or IL inhibitors – 12/23, Abataceptum or Tofacitinibum should be prescribed instead of a different TNF inhibitor mono-therapy [35]. Abataceptum may be used if there is a serious or recurring infection but no severe psoriasis, according to the data on infection reduction when patients with RA were taking Abataceptum rather than TNF inhibitor [36]. If a patient prefers a per os administration, Tofacitinibum is a good alternative.

If PsA remains active despite TNF inhibitors, the second line of defense may involve IL inhibitors – 17 rather than IL inhibitors – 12/23, Abataceptum or Tofacitinibum. Among the latter group of drugs, IL inhibitors – 12/23 are more effective than Abataceptum or Tofacitinibum. IL inhibitors – 12/23 are used instead of IL inhibitors – 17 when a patient has an inflammatory bowel disease (IBD) or prefers a rare drug administration. Abataceptum is an alternative to IL inhibitors – 17 and IL inhibitors – 12/23 when there is a recurring serious infection. Tofacitinibum might replace IL inhibitors – 17 and IL inhibitors – 12/23 for patients who prefer per os administration or have a history of recurring serious Candida infection [36]. For every biological agent (TNF inhibitor, IL inhibitors – 17 and IL inhibitors – 12/23) mono-therapy seems a better option than a MTX-combined one. MTX combined with a biological agent is used instead of a biological mono-therapy when a patient has a severe psoriasis, partial response to the current MTX therapy, uveitis. Biological TNF inhibitor used should be either Infliximab or Adalimumab (to prevent immunogenic response) [38].

In case when even after a combined therapy with TNF inhibitor and MTX PsA still remains active, a switch should be for another TNF inhibitor and MTX, rather than for a TNF inhibitor mono-therapy. Continued administration of MTX together with TNF inhibitor seems efficacious because when combined with MTX biological agents demonstrate a consistent effect [39]. Mono-therapy with a different TNF inhibitor may be used is a patient suffers from MTX-associated side effects, wants to restrict the amount of medication or considers MTX therapy a burden. Combined therapy of IL inhibitors – 17 or IL inhibitors – 12/23 together with MTX is not advisable as a mono-therapy provides a better effect. However, if IL inhibitors – 17 or IL inhibitors – 12/23 elicited a partial response or a patient has a concomitant uveitis, these medications may be combined with MTX [40].

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**Notes:** TNF inhibitors - tumor necrosis inhibitor drugs; IL inhibitors – 17 – interleukin inhibitors; IL inhibitors – 12/23 - interleukin inhibitors – 12/23; MTX – Methotrexate; PsA – psoriatic arthritis. Review the alternative drugs: IL inhibitors – 17 or IL inhibitors – 12/23 might be used instead of TNF biological inhibitors in severe psoriatic patients or under TNF inhibitor contraindications; basic drugs per os, IL inhibitors – 17 or IL inhibitors – 12/23 under TNF inhibitor contraindications; basic therapy and small-molecule drugs in case of patient’s reluctance to start biological drugs and preference for per os administration, IL inhibitors – 12/23 in case a patient wishes the drug to be administered rarely. Review the alternative drugs: IL inhibitors – 17 or IL inhibitors – 12/23 might be used instead of basic therapy per os in severe psoriatic and PsA patients; IL inhibitors – 12/23 in case of a concomitant active inflammatory bowel disease (IBD) or in case a patient wishes the drug to be administered rarely. NSAIDs may replace MTX in patients with an inactive PsA following the cardiac and renal-associated risks. IL inhibitors – 12/23 may be used instead of MTX in a patient wishes the drug to be administered rarely.
Active PsA despite IL inhibitor – 17 mono-therapy (Fig. 5)
If PsA remains active despite IL inhibitor – 17 mono-therapy, TNF inhibitor is suggested; however, it should be unaccompanied by IL inhibitors – 12/23 or MTX. Prescription of a different IL inhibitor – 17 is also discouraged [40].

Switch to IL inhibitors – 12/23 is a more effective option than adding MTX to IL inhibitor – 17 or using a different IL inhibitor – 17. If a patient has a severe psoriasis and contraindications prevent TNF inhibitor use, IL inhibitor – 17 should be replaced by IL inhibitors – 12/23 [41].

Another biological IL inhibitor – 17 might be used instead of switching to TNF inhibitor or IL inhibitors – 12/23 if a patient has a secondary ineffectiveness due to a modern biological treatment, severe psoriasis or TNF inhibitor contraindications [42]. MTX may be added to the current IL inhibitor – 17 therapy instead of switching to TNF inhibitor or IL inhibitors – 12/23 if a patient had a partial response to IL inhibitor – 17 therapy in the past [42].

Active PsA despite IL inhibitor – 12/23 mono-therapy (Fig. 5).
If PsA remains active despite IL inhibitor – 12/23 mono-therapy, TNF inhibitor, MTX addition or switching to IL inhibitor – 17 are suggested. Switching to IL inhibitor – 17 is a better strategy than adding MTX to a current therapy [43]. Treatment may include IL inhibitor – 17 rather than TNF inhibitor if a patient has a severe psoriasis or TNF inhibitor contraindications [42]. MTX may be added to IL inhibitor – 12/23 instead of a switch to TNF inhibitor or IL inhibitor – 17 if a patient shows a partial response to this therapy; it may also be added instead of a switch to TNF inhibitor if there are TNF inhibitor contraindications [20].

T2T (Treat-to-target) therapy
If PsA is active, a well-known T2T (Treat-to-target) therapy is recommended. It presupposes the treatment until the cure is achieved. Dismissal of this strategy occurs when the healthcare providers have certain concerns as to the adverse or side effect intensification or onset, treatment costs or biological agents being incapable to control the current therapy.

Active PsA with spondylitis/axial damage despite NSAIDs
Basic drugs administered per os unfortunately are ineffective for axial PsA [44]. Patients, who retain active axial PsA despite NSAID treatment, should switch to TNF inhibitors, at the further stage – to IL inhibitor – 17, later – to IL inhibitor – 12/23. IL inhibitor – 17 is used instead of TNF inhibitors in case of a severe psoriasis or existing TNF inhibitor contraindications. IL inhibitor – 12/23 seems to be the least effective in this clinical situation, as the results of three randomized studies of Ustekinumab show lack of primary and secondary cut-off points achieved [45-47].

Active PsA with prevailing enthesitis in the untreated patients or patients who received basic drugs per os and did not show any response
The untreated patients with enthesitis may use TNF inhibitor biological agents as medications of the first...
line of defense [48]. Patients with active PsA who suffer from enthesitis despite taking basic drugs per os (the latter were prescribed for other PsA manifestations and other clinical situations) are recommended to switch to TNF inhibitors, IL inhibitor – 17 or IL inhibitor – 12/23 rather than to other medications of the same group. Usually TNF inhibitors are the line of first defense, IL inhibitor – 17 - the line of second defense, IL inhibitor – 12/23 – the line of third defense [48]. If a patient has a severe psoriasis and TNF inhibitor contraindications, he/she should start IL inhibitor – 17. If he/she prefers a rarer drug administration, has an inflammatory bowel disease (IBD), the best option is IL inhibitor – 12/23. Apremilast may replace TNF inhibitors in case a patient prefers per os drug administration or TNF inhibitor contraindications [33]. Per or NSAID’s efficacy in case of concomitant enthesites prevails over that of basic per os drugs. They may be prescribed is a patient has no cardiovascular conditions, ulcers, kidney diseases (or failures) or severe psoriasis, PsA. Tofacitinibum has an edge over Apremilast in case of PsA with prevailing enthesitis [37]. The latter, however, is advisable for patients with recurring infections.

**Active PsA with an active concomitant inflammatory bowel disease (IBD)**

To treat the previously untreated patients with an active PsA with an active concomitant inflammatory bowel disease (IBD), healthcare providers opt for TNF inhibitors but for Etanercept which is a biological molecule of soluble receptors and thus ineffective in this clinical situation). Basic per os drugs may be used by the patients with no severe PsA preferring per os drug administration or having TNF inhibitor contraindications [49].

If an active PsA is diagnosed along with an active inflammatory bowel disease (IBD) despite being treated with basic drugs, TNF inhibitor or IL inhibitor – 12/23 monoclonal antibodies are a medication of choice.

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**Fig 4. Active PsA despite TNF inhibitors as a mono- or combined therapy (adapted from [19]).**

Notes: TNF inhibitors - tumor necrosis inhibitor drugs; IL inhibitors – 17 - interleukin inhibitors- 17; IL inhibitors – 12/23 - interleukin inhibitors – 12/23; PsA – psoriatic arthritis; MTX – Methotrexate; oral small-molecule drugs (OSMDs). Biological drug mono-therapy is considered more effective than a combined one with MTX. If the patient did not show any response to TNF inhibitors, there might be alternative suggestions (IL inhibitors – 17 or IL inhibitors – 12/23, Abataceptum or Tofacitinibum; if there are serious TNF inhibitor– associated side effects (IL inhibitors – 17 or IL inhibitors – 12/23, Abataceptum or Tofacitinibum; if a patient showed a partial response to TNF inhibitors, especially immunogenic (add MTX); if a patient prefers per os administration (Tofacitinibum); if a patient has a severe psoriasis (IL inhibitors – 17); if a patient prefers a rare drug administration (IL inhibitors – 12/23). Alternative options should be considered in case of a concomitant inflammatory bowel disease (IBD) (IL inhibitors – 12/23, Tofacitinibum); preference for an intravenous administration (Abataceptum); serious or recurring infections (Abataceptum); preference for per os administration (Tofacitinibum); history of Candidosis (Tofacitinibum); preference for a rare drug administration (IL inhibitors – 12/23). Alternative options should be considered if a patient prefers an intravenous administration (Abataceptum); has serious or recurring infections (Abataceptum); prefers per os administration (Tofacitinibum). TNF inhibitor mono-therapy is to be suggested if a patient develops serious MTX-associated side effects or wants to restrict the amount of medication. IL inhibitors – 17+MTX combined therapy is an option if a patient has a partial response to therapy or uveitis. IL inhibitors – 12/23+MTX combined therapy is an option if a patient has a partial response to therapy or uveitis.
Moreover, TNF inhibitor-α monoclonal antibodies should be taken over soluble TNF inhibitor-α receptor (Etanercept). TNF inhibitor-α monoclonal antibodies are considered more effective than IL inhibitor – 12/23. The latter may be used in case of TNF inhibitor contraindications or a preference for a rarer drug administration [50].

Active PsA with concomitant diseases

Active PsA in diabetes patients who don’t receive basic per os therapy or biological agents. Patients with an active PsA and concomitant diabetes who don’t receive basic per os therapy (Methotrexate (MTX), Sulfasalazine (SSZ), Cyclosporine, Leflunomide) or biological agents are recommended to start with basic drugs (except for MTX) rather than with biological drugs. MTX exclusion is explained by the concerns about fatty hepatosis prevalence, MTX and biologics’ increased hepatotoxicity for this group of patients [51, 52]. TNF inhibitor may be substituted for basic per os therapy in case of a severe PsA or psoriasis, well-controlled diabetes with a potentially reduced infection risk.

Active PsA in patients with severe infections who don’t receive basic per os therapy or biological agents. Patients with an active PsA and severe infections who don’t receive basic per os therapy or biological agents should first address basic drugs rather than TNF inhibitors as severe infections are an absolute contraindication for the latter administration. It’s possible to consider such biological agents as IL inhibitor – 17 or IL inhibitor – 12/23.

Inoculation with live and inactivated vaccines for patients with an active PsA treated with bioagents

According to the Centers for Disease Control and Prevention (CDC), it is suggested to start treatment with biological agents without delay, and get patients inoculated with killed or inactivated vaccines based on the patient’s age, sex and inoculation history. If the live (unattenuated) vaccines are required, biological therapy of patients with an active PsA should be delayed. In case PsA manifestations are significant and the delay seems undesirable, the healthcare provider may consider starting the treatment and injecting live (unattenuated) vaccines at the same time.

Recommendations on non-pharmacological interventions in patients with an active PsA irrespective of pharmacological treatment

Patients with an active PsA may perform certain physical exercises or their combinations; undergo physiotherapy, occupational therapy, massage and
acupuncture. Physical exercises with a reduced load (for instance, tai chi, yoga, swimming) and load-bearing exercises (for instance, running) should be restricted. Load-bearing exercises may be used instead of reduced-load exercises if the patients prefer them and do not have contraindications. Clinicians should urge patients to quit smoking using various methods as smoking cessation was proved to be very effective by a number of randomized trials [53, 54]. PsA patients who have an excess body weight or obesity should reduce their body weight to improve the potential pharmacological reaction.

Conclusions

This review focuses on the American College of Rheumatologists (ACR) Guidance of 2018 on Psoriatic arthritis treatment. Its aim is to help the healthcare providers to achieve optimum PsA management. PsA is a heterogeneous and multifaceted inflammatory disease with a multitude of clinical manifestations (periarticular arthritis, psoriasis, nail lesions, enthesitis, dactylitis, axial impairments) requiring a certain differential and incremental approach. Despite an increase in PsA’s new therapies, their evidence-based efficacy is restricted. The Guidance will be edited with the new comparative study data being presented.

The ACR recognizes the patients’ preferences and attitudes being the pivotal element of the Guidance. Decisions made at the patient group’s meeting were submitted to vote at the peer committees to make sure that their viewpoint were included in the finalized version of the PsA Guidance. Among the feedback suggestions made by the patients they cite efficacy (for instance, to prevent further damage to the target organs, improve life quality, public involvement and functioning) and safety evaluation (for instance, to ensure low profile of side effects). Moreover, the patients discussed adverse treatment effects (for instance, fatigue, nausea, distemper) and their influence on life quality and public involvement drawing a conclusion that adverse event risks affected patients and healthcare providers’ decision making. Treatment-to-target (T2T) was found to be a complicated concept for patients’ understanding. Although they understood the value of improved results, they also thought that this strategy would increase their treatment and trip costs as they would have to travel more often to meet their doctors, also it would increase the negative treatment effects. Thus, making decision as to the treatment-to-target (T2T) option requires detailed discussions with patients, focusing your recommendations on their needs.

Potential aims of PsA treatment include determining minimal disease activity (MDA) and disease activity in psoriatic arthritis (DAPSA) [55, 56]. The Guidelines of ACR/NFP 2018 urge to treat an active PsA with TNF inhibitors (as a line of first defense) rather than with basic per os medication. The former are provisional, and the expert group drew several possible exceptions. There are certain terms under which basic per os medication is prescribed as a line of first defense. They emphasize upon the early and aggressive intervention in PsA patients as soon as the disease is diagnosed.

However, the above mentioned recommendations are provisional, and the expert group drew several possible exceptions. There are certain terms under which basic per os medication was prescribed as a line of first defense. They emphasize upon the early and aggressive intervention in PsA patients as soon as the disease is diagnosed.

At the same time, it was recognized that those recommendations do not take into account the whole PsA complexity, heterogeneity or the range of possible therapies (Glucocorticoids were not reviewed). Overall inconsistency in PsA’s clinical features and development is caused by co-existence of several domains in one and the same patient and could not be designed as an algorithm. Moreover, efficacy evaluation and severity reports, discrepancies in the inclusion/exclusion criteria of PsA clinical trials complicate the comparison of therapeutic methods. Alternative therapy and its influence on significant outcomes, such as joint damage, were not explored. Recommendations as to inoculation of the patients while undergoing Tofacitinib treatment were not included as they were not approved as PsA therapy.

As the National Psoriasis Foundation (NPF) and the American Academy of Dermatology (AAD) are both elaborating the isolated psoriasis treatment guidance, it’s not included in the present review.

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Сучасні рекомендації щодо лікування псоріатичного артриту згідно з рекомендаціями Американського коледжу ревматологів і Національної організації з псоріазу 2018 року

Резюме. Псоріатичний артрит (ПсА) — це хронічне за- пальне скелетно-м'язове захворювання, асоційоване з псоріазом, яке маніфестує зазвичай з периферичної ар- триту, а також тяжкого, рідко з'являється у вигляді гетерогенної хвороби. Клінічна картина ПсА є гетерогенною, і лікарі досить часто стикаються з проблемами при визначенні стратегій лікування. Наявність або відсутність попередніх методів лікування є факторами, що дозволяють зробити вибір оптимальної терапії для окремого па- цієнта в даний момент часу, застосовуючи індивідуаль- ний підхід. Рекомендації ACR/NFP щодо лікування активного ПсА рекомендують інгібітори фактора некрозу пухлини, а не пероральні низькомолекулярні препарати як препарати- рій першої лінії, саме ці біологічні агенти запобігають про- гресуванню ізумців, увеїт, діабет або серйозні інфекції, а також новину. Рання ідентифікація та діагностика ПсА, ранній початок терапії важ- 

Ключові слова: псоріатичний артрит; псоріаз; активність процесу; рекомендації; лікування; менеджмент; інгібіто- ри фактора некрозу пухлини; інгібітори інтерлейкін-17; інгібітори інтерлейкін-12/23; метотрексат; лікування до досягнення мети.
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Современные рекомендации по лечению псориатического артрита согласно рекомендациям Американского колледжа ревматологов и Национальной организации псориаза 2018 года

Резюме. Псориатический артрит (ПсА) — это хроническое воспалительное скелетно-мышечное заболевание, ассоциированное с псориазом, которое манифестируется обычно как периферический артрит, дактилит, энтезит и/или спондилит. Ранняя идентификация и диагностика ПсА, раннее начало терапии важны для улучшения долгосрочных исходов болезни. Клиническая картина ПсА является неоднородной, и врачи довольно часто сталкиваются с проблемами при определении стратегий лечения. Целью нашего обзора было представление и интерпретация рекомендаций Американского колледжа ревматологов (ACR) и Национальной организации псориаза (NFP) 2018 года по лечению активного ПсА у взрослых с использованием фармакологических и нефармакологических методов. Эти рекомендации по лечению ПсА могут помочь как клиницистам, так и пациентам достичь оптимальных решений по менеджменту болезни. В рекомендациях представлены стратегии выбора препаратов для лечения активного ПсА при различных клинических ситуациях и в зависимости от проводимой предыдущей терапии. Наличие или отсутствие коморбидных состояний, таких как воспалительное заболевание кишечника, увеит, диабет или сердечные инфекции, а также знания предыдущих методов лечения являются факторами, которые позволяют сделать выбор оптимальной терапии для отдельного пациента в данный момент времени, используя индивидуальный подход. Рекомендации ACR/NFP по лечению активного ПсА рекомендуют ингибиторы фактора некроза опухоли, а не пероральные низкомолекулярные препараты как препараты первой линии, именно эти биологические агенты предупреждают прогрессирование заболевания и повреждение суставов. Отмечается необходимость ранней и агрессивной терапии у пациентов с впервые выявленным ПсА. Ключевые слова: псориатический артрит; псориаз; активность процесса; рекомендации; лечение; менеджмент; ингибиторы фактора некроза опухоли; ингибиторы интерлейкина-17; ингибиторы интерлейкинов-12/23; метотрексат; лечение до достижения цели