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Review

Current Treatment Strategies in Psoriatic Arthritis

Psöriatik Artritte Güncel Tedavi Stratejileri

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ABSTRACT

Psoriatic arthritis (PsA) is seen in 20% of psoriasis patients and sometimes manifests before dermatological signs of psoriasis. PsA usually has a favorable prognosis, but in some patients, it can cause a disabling form of arthropathy. Furthermore, patients with PsA may have multiple comorbidities like cardiovascular disease, gout, metabolic syndrome, anxiety, and depression. Hence, effective treatment is important. There are multiple newly developed biological and targeted agents aiming to improve the quality of life and reduce disability. Non-steroidal anti-inflammatory drugs, local/systemic steroids, conventional disease-modifying anti-rheumatic drugs, tumor necrosis factor inhibitors, interleukin (IL)-17 inhibitors, Janus kinase inhibitors, apremilast, and IL-12/23 inhibitors are drugs that can be used for different domains of PsA. There are still newly developping agents under investigation.

Keywords: Psoriatic arthritis, psoriasis, biological agents, treatment, management

ÖZ

Psöriatik artrit (PsA) psöriazisli hastaların %20'sinde görülebilen bir romatizmal hastalıktır ve bazen cilt bulgularından önce ortaya çıkar. Genellikle iyi prognozlu olmasına rağmen bazen engelliliğe neden olabilir. PsA'lı hastalar eş zamanlı kardiyovasküler hastalıklar, gut, metabolik sendrom, anksiyete ve depresyon gibi komorbiditelere sahip olabilirler. Bu nedenle etkili tedavi önem arz etmekte olup yeni geliştirilen biyolojik ve hedefe yönelik ajanlarla hayat kalitesi iyileştirilebilir ve engellilik azaltılabilir. Steroid olmayan anti-enflamatuvar ilaçlar, lokal ve sistemik steroidler, konvansiyonel hastalık modifiye edici ilaçlar, tümör nekroz faktör inhibitörleri, interlökin (IL)-17 inhibitörleri, Janus kinaz inhibitörleri, apremilast, IL-12/23 inhibitörleri farklı tutulum şekillerinde kullanılabilir. Bazı yeni ajanlar ise henüz araştırma aşamasındadır.

Anahtar Kelimeler: Psöriatik artrit, psöriazis, biyolojik ajanlar, tedavi, yönetim

INTRODUCTION

Psoriatic arthritis (PsA), a chronic inflammatory musculoskeletal disease, is seen in approximately 20% of psoriasis patients and it is one of the a member of the spondyloarthropathies. It causes peripheral arthritis, entesitis, sacroiliitis, dactylitis, spondylitis, nail and skin involvement, and has diverse clinical pictures which sometimes makes diagnosis tough even for experts (Figure 1) (1).

Psoriasis, which is a relatively common condition with a prevalence of approximately 2% in the adult population, is well-documented; however, data about PsA frequency is very limited, which may be due to the lack of widely accepted classification or diagnostic criteria. PsA is rare in childhood and the geriatric period (2). In 15% of PsA patients, musculoskeletal findings might emerge before dermatological signs, causing delay in diagnosis (1).

Underestimation of PsA symptoms may cause a delay in the diagnosis. PsA usually has a favourable prognosis but can also cause deforming destructive arthropathy (shortening or telescoping of digits, fusion of joints) like rheumatoid arthritis and reduced quality of life, social isolation and disability (3). In addition, PsA can cause some extra-articular manifestations like uveitis, inflammatory bowel disease, cardiovascular disease, metabolic syndrome, hypertension, diabetes mellitus, and hyperlipidemia. Hence, early diagnosis and treatment are crucial in improving clinical outcomes and reducing joint damage. Fortunately,

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there are multiple treatment options providing effective management (2). Goals of treatment include: alleviation of symptoms, improvement of joint functions, and prevention of comorbidities (1). Discovery of novel inflammatory pathways and development of new agents targeting these pathways has provided effective treatment of PsA (4).

Management requires a multidisciplinary approach including rheumatologists who care primarily for musculoskeletal involvement of the disease. Collaboration between the patient and the physician during treatment decision-making is important. Extra-articular findings and comorbidities should also be taken into account. Treatment should aim to reach remission or low disease activity (LDA) in long-standing disease, consisting of follow-ups at 3-month intervals, according to recent guidelines (treat-to-target strategy) (5). It would be rational to determine involved structures (peripheral arthritis, axial disease, dactylitis, enthesitis, skin or nail) and manage the medical treatment appropriately (6).

CLINICAL AND RESEARCH CONSEQUENCES

Therapeutical Agents Used in PsA

European Alliance of Associations for Rheumatology (EULAR, 2023), Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA, 2021), British Society for Rheumatology (BSR, 2022), and American College of Rheumatology/National Psoriasis Foundation (ACR/NPF, 2018) developed evidence-based recommendations for both pharmacological and non-pharmacologic treatment of PsA (5,7-9). Non-steroidal anti-inflammatory drugs (NSAIDs), systemic or local glucocorticoids, conventional synthetic disease-modifying anti-rheumatic drugs (csDMARD), tumor necrosis factor (TNF) inhibitors and newly developed biological agents are available choices in different conditions of the disease (Table 1) (7,10,11).

ACR/NPF 2018 recommended using TNF inhibitors over interleukin (IL)-17 inhibitors, IL-12/23 inhibitors, and oral small molecules like methotrexate, leflunomide, sulfasalazine, cyclosporine, and apremilast in the treatment of naive active PsA patients. In this guideline, 6% of recommendations were strong and 96% of them were conditional (9). EULAR 2023 guidelines recommended a csDMARD in patients with poor prognosis and polyarthritis. Glucocorticoids and NSAIDs were recommended as adjunctive therapy. In patients with an inadequate response to one csDMARD therapy, EULAR recommends the use of a biologic DMARD (bDMARD). In axial disease, EULAR recommended using NSAIDs, and for patients with inadequate response, the use of bDMARDs was recommended (5). BSR 2022 guideline proposed domain specific recommendations, which mainly consisted

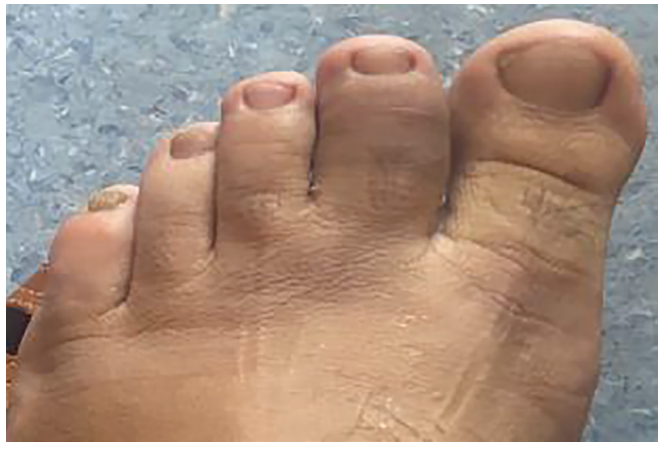

Figure 1. Dactylitis in the 2nd toe in a patient with PsA
PsA: Psoriatic arthritis

Table 1. Drugs available for PsA management

Type	Name
	NSAIDs, local and systemic steroids
Conventional synthetic DMARDs	• Methotrexate • Leflunomide • Sulfasalazine
Biological DMARDs	• Anti-TNF agents (adalimumab, certolizumab pegol, golimumab, etanercept, infliximab) • Anti-IL-12/23 (ustekinumab) • Anti-IL-17A (secukinumab, ixekizumab) • Anti-IL-17A/F (bimekizumab) • Anti-CTLA4 (abatacept) • Anti-IL-23-p19 (guselkumab, risankizumab)
Targeted synthetic DMARDs	• JAK inhibitors • Tofacitinib • Upadacitinib • PDE4 inhibitor: apremilast

NSAIDs: Non-steroidal anti-inflammatory drugs, TNF: Tumor necrosis factor, IL: Interleukin, JAK: Janus kinase, DMARDs: Disease modifying antirheumatic drugs, PDE4: Phosphodiesterase 4, CTLA4: Cytotoxic T-lymphocyte antigen 4, PsA: Psoriatic arthritis

of csDMARD as a first line in peripheral involvement, with step up to bDMARD if response was inadequate. In axial disease, it has been recommended that NSAIDs be used as the first choice, and TNF inhibitors, IL-17 inhibitors, or Janus kinase (JAK) inhibitors be used for intolerants/inadequate responders (8). The GRAPPA 2021 guidelines proposed strong recommendations, for different domains of disease. GRAPPA expert group recommended csDMARD, TNF inhibitors, IL-17, IL-23, JAK, phosphodiesterase 4 (PDE4) inhibitors for peripheral disease and NSAIDs, TNF, IL-17, and JAK inhibitors for active axial disease (7). Lifestyle recommendations were proposed in BSR 2022 and ACR/NPF 2018 guidelines (8,9). The treat to target strategy was recommended in ACR/NPF 2018, EULAR 2023, and BSR 2022. LDA or remission achievement was primarily aimed at achieving through the treat-to-target strategy (5,8,9).

Although various theories are proposed, the pathogenesis of PsA is not clearly recognized today. Multiple environmental factors (smoking, infections, stress, etc.,) in the presence of genetic susceptibility (genes like human leukocyte antigen B27, B08) are possible activators of innate and adaptive immune systems that take a role in the pathogenic processes of PsA. Emerging targeted drugs effective against these different pathological axes demonstrated promising results in the management of the disease (12).

Patients who had predominatly peripheral disease, NSAIDs, intra articular and systemic glucocorticoids can be used while csDMARDs like methotrexate, leflunomide and sulfasalazine are first line options with preference of methotrexate in those with significant skin involvement. Oral methotrexate 10 mg weekly can be initiated and gradually increased up to 20 mg weekly. It is important to keep in mind that methotrexate is associated with an increased risk of hepatic fibrosis in patients with obesity or diabetes mellitus. In patients with low skin burden, leflunomide 20 mg daily, can be used as an alternative to methotrexate. Sulfasalazine 2-3 grams daily is another option in patients with mild psoriatic skin involvement. Gastrointestinal adverse effects are the main reason for discontinuation of the drug. Systemic glucocorticoids are used for severe flare unresponsive to NSAIDs, but are suggested to be tapered slowly as rapid reductions are associated with erythrodermic or pustular flares. For axial disease, NSAIDs (at the higher end of the dose range, for example, indomethacin tablet up to 150 mg), physiotherapy and TNF inhibitors, as well as IL-17 and JAK inhibitors, are used for NSAID unresponders (5,7-9).

Pathogenesis of PsA and Targeted Therapies

The pathogenetic process starts with the activation of antigen-presenting cells (APC) by genetic predisposition and environmental factors. After the secretion of several cytokines, such as IL-1, IL-6, IL-17, IL-22, IL-23, and TNF-α, by APCs, T lymphocyte activation takes place, and the pathological process of PsA proceeds (12).

IL-17A, which is involved in the pathogenesis of PsA, causes synovial tissue proliferation, inflammation and bone resorption (13). T lymphocytes differentiate into T helper 17 cells and secrete IL-17 (predominantly IL-17A isoform), facilitated by the key cytokine IL-23 [p19 and p40 (shared with IL-12) subunits]. Also, it was shown that IL-23 levels are increased in synovial tissue and skin of PsA patients (14). PDE4 enzyme converts cyclic adenosine monophosphate (AMP) to AMP, causing an increase in inflammatory cytokine expression (15). JAKs affect some of the cytokines important in the pathogenesis of PsA, like interferon-γ, IL-12, IL-22, IL-23, and IL-6 (16). Tyrosine kinase 2 (TYK-2), a JAK, mediates signaling by IL-23 (17). Recently developed targeted therapies generally act on these pathways.

Secukinumab

Secukinumab is a selective and direct inhibitor of IL-17A (18). EULAR/GRAPPA recommends it for uncontrolled axial or peripheral disease, and this agent was also found to be effective in skin involvement (5,7). A randomised,-placebo controlled phase 3 study (ULTIMATE) showed significantly reduced clinical and sonographic enthesitis/synovitis scores PsA patients in the secukinumab group at 12 weeks, reaching a plateau at week 52 (19). In the CHOICE study (double blind, randomized), ACR 20% (ACR20) response rates of biologic-naive PsA patients at week 16 were found to be significantly higher in the secukinumab group compared to the placebo (20).

Bimekizumab

Bimekizumab, an IL-17F and IL-17A inhibitor, is approved for PsA in Europe. In a recent systematic review [66 studies, evaluating 22 biologic/targeted synthetic (b/ts) DMARDs] and network meta-analysis (41 studies), bimekizumab was found to be effective on skin involvement. According to network meta-analysis, for minimal disease activity (MDA), bimekizumab 160 mg/4 weeks was ranked first in b/ts-DMARD naive patients and second in TNF-inhibitor experienced patients. The safety profile was similar to other b/ts DMARDs (21). In a study including active PsA patients (n=347), at week 16, MDA was achieved in 44% of the bimekizumab group versus 6% in the placebo group.

After switching to bimekizumab in the placebo group, MDA was 47% in the bimekizumab group and 33% in the bimekizumab switched group. Bimekizumab was superior to placebo in skin, nails, joints, dactylitis and enthesitis domains in patients who were inadequent responder/intolerant to TNF inhibitors. The most common adverse events were oral candidiasis, nasopharyngitis, and urinary tract infection. Efficacy was sustained from week 16 to 52 in the bimekizumab group (22).

Ixekizumab

Ixekizumab is an IL-17A inhibitor, which was previously shown to be effective on multiple domains of PsA. The SPIRIT study, including 566 patients with PsA, showed significant joint and skin improvement in ixekizumab group than in adalimumab patients at week 52. Safety issues were similar to previous results (infections and injection site reactions) (23,24). In another study, the ixekizumab group achieved the treatment target more significantly than the placebo group with disease activity indices at week 24 (25). A phase-III study involving patients who were non-responders to or intolerant of TNF inhibitors showed that significantly more patients in the ixekizumab group than in the placebo group achieved ACR50, MDA, disease activity in PsA (DAPSA) ≤14, and Psoriasis Area And Severity Index (PASI) 100 at week 24, which persisted through week 52 (26).

Risankizumab

Risankizumab blocks the p19 subunit of IL-23, and is approved for PsA in many countries. In a phase III KEEPsAKE 1 study, 940 patients unresponsive/intolerant to csDMARDs, were randomized to risankizumab and placebo groups (subcutaneous 150 mg risankizumab vs placebo on weeks 0, 4, and 16). At week 24, more patients in the risankizumab group had improved significantly more than those in the placebo group regarding the ACR20 response. ACR50 and ACR70 response rates were also significantly higher in the risankizumab group. Joint, skin, and nail findings of PsA patients had benefited from continuous risankizumab treatment, and radiological progression was slower as well (27). Another phase III study, KEEPsAKE 2 trial, showed improvement of PsA signs and symptoms (joint, skin, fatigue) which were maintained for 52 weeks in patients inadequately responsive/intolerant to csDMARD/bDMARD. The long-term safety profile was consistent through week 52 (28). In a meta-analysis of 6 randomized controlled studies, risankizumab group had significantly more ACR20 response rates than placebo, PASI scores were lower at 24th weeks. Serious adverse events between two groups were not significantly different (29).

Guselkumab

Guselkumab targets the p19 unit of IL-23 and is approved for psoriasis and PsA. In the DISCOVER-2 trial, 652 patients were randomized to guselkumab and placebo groups. Stable improvements in ACR20, 50, 70 scores, and the enthesitis and dactylitis resolution scores were observed at the 100th week (30). Among the 285 participants of the COSMOS study, a significantly higher number of patients in the guselkumab group had an ACR20 response regarding skin, joint, and functional scores, than the placebo group (31). Data from DISCOVER-1 and 2 studies showed more patients in the guselkumab group had reached DAPSA LDA, DAPSA remission, and MDA at week 24 (32). Studies consisting of TNF inhibitor naive and experienced patient groups showed low rates of adverse events in both groups up to two years of follow-up (33).

Ustekinumab

Ustekinumab is a monoclonal antibody blocking both IL-12 and 23. In phase 3 PSUMMIT 1 and 2 studies, ustekinumab treated patients showed higher ACR20, 50, 70, DAS28-C-reactive protein responses and remission rates than placebo group at 24th weeks. Besides, complete resolution of enthesitis and dactylitis was higher in the ustekinumab group at week 24 (34). The pooled results from these two studies demonstrated greater changes in the modified Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) scores in the ustekinumab group than in the placebo group at week 24 (35). Concomitant methotrexate administration was not associated with the increased efficacy of ustekinumab (36).

Upadacitinib

Upadacitinib is a JAK inhibitor selectively effective on JAK-1. In a randomized controlled trial, Mease et al. (37) investigated its effectiveness in patients with PsA. Patients with upadacitinib had more ACR20 responses than placebo at week 12. MDA score was higher in the upadacitinib group at the 24th week. Rates of adverse events were similar between placebo and 15 mg upadacitinib groups (37). In a study comparing upadacitinib with adalimumab, ACR20, 50, 70 and MDA responses were higher in the upadacitinib group than in the adalimumab group at 24 weeks (38). In another study in patients with axial disease, the upadacitinib 15 mg group showed greater BASDAI and Ankylosing Spondylitis Disease Activity Score responses than placebo (39). Phase 3 studies of SELECT-PsA 1 and 2 demonstrated that upadacitinib was effective in all clinical manifestations of PsA. In addition, adverse effects like cardiovascular events, malignancy, venous thromboembolism did not increase with upadacinitib (40).

Filgotinib

Filgotinib is a newly developed oral agent under investigation for PsA. It selectively blocks JAK-1. In a phase 2 study, namely, the EQUATOR trial, filgotinib 200 mg was significantly superior to placebo in terms of improvements in active domains (peripheral arthritis, psoriasis, and enthesitis) of PsA . Furthermore, ACR20 response rates at week 16 were significantly higher in the filgotinib group than in the placebo. Filgotinib also significantly improved the quality of life of the patients more than the placebo group. Only one patient in the filgotinib group had herpes zoster infection (41).

Deucravacitinib

Deucravacitinib is an oral selective TYK-2 inhibitor. A phase 2 trial (203 patients) showed significantly higher ACR20 response rates and better quality of life scores with deucravacitinib 6 mg once daily than placebo at week 16. The most common adverse events were reported as upper respiratory tract infections, diarrhea, and headache (17).

Brepocitinib

Brepocitinib is a TYK-2/JAK-1 inhibitor that primarily affects the IL-12 and 23 signaling pathways. The phase-II study of the brepocitinib group, with 218 patients, showed higher ACR20, 50, 70, Psoriasis Severity Index scores, and MDA response rates at week 16 than the placebo. Response rates were improved or sustained through week 52. Mild/moderate infections were reported in the brepocitinib group (42).

Tofacitinib

Tofacitinib is an oral JAK inhibitor, and its efficacy and safety in active PsA were shown in phase 3 studies. Improvement in enthesitis generally increased over time (43). Its efficacy was greater than that of the placebo in PsA, regardless of concomitant methotrexate dose. Tofacitinib 5 mg was more efficacious with methotrexate >15 mg/week, (headache was more common), than <15 mg/week (44).

Apremilast

The oral PDE4 inhibitor apremilast (30 mg) was associated with significant ACR response rates in patients with PsA, and these rates were sustained at week 260. Swollen and tender joint counts, enthesitis, dactylitis, and psoriasis skin lesions were domains that benefited. The most common adverse effects reported were diarrhea, nausea, headache, and upper respiratory tract infections (45).

Abatacept

It is a selective T cell co-stimulation modulator and approved for PsA. It had efficient responses in PsA activity and reduced structural damage, psoriasis, enthesitis, and dactylitis. It has greater response rates in patients with poor prognostic factors (high disease activity and progressive disease) (46). Abatacept infusion also caused significant reductions in magnetic resonance imaging scores of synovitis and tenosynovitis of PsA patients more than placebo group (47).

CONCLUSION

PsA can frequently cause functional impairment or disability, eventually impairing the patients' quality of life. NSAIDs and glucocorticoids are used for musculoskeletal relief, but csDMARDs and bDMARDs are cornerstones of management. Besides lifestyle modification and non-pharmacologic recommendations, there are multiple current successful agents, and new drugs under investigation. Future research focusing on the pathophysiological mechanisms will help to better understand the disease and lead to the development of new therapeutic agents.

FOOTNOTES

Authorship Contributions

Consept: Ö.B., EK., Design: Ö.B., EK., Data Collection or Processing: Ö.B., EK., Analysis or Interpretation: Ö.B., EK., Literature Search: Ö.B., EK., Writing: Ö.B., EK.

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