Review

Psoriatic dactylitis: from immunopathogenesis to anti-cytokine and targeted synthetic therapies

M. Fatica[1], P. Triggianese[1], P. Conigliaro[1], M. Tasso[2], N. Girolimetto[3], L. Costa[2], R. Scarpa[2], A. Bergamini[1], F. Caso[2], M.S. Chimenti[1]

[1]*Rheumatology, Allergology and Clinical Immunology, Department of Systems Medicine, University of Rome Tor Vergata, Rome, Italy;*
[2]*Rheumatology Research Unit, Department of Clinical Medicine and Surgery, School of Medicine and Surgery, University of Naples Federico II, Naples, Italy;*
[3]*Department of Rheumatology, Azienda USL-IRCCS di Reggio Emilia, Italy.*

*Mauro Fatica, MD**
*Paola Triggianese, MD, PhD**
Paola Conigliaro, MD, PhD
Marco Tasso, MD
Nicolò Girolimetto, MD
Luisa Costa, MD, PhD
Raffaele Scarpa, MD
Alberto Bergamini, MD
*Francesco Caso, MD, PhD***
*Maria Sole Chimenti, MD, PhD***

**These authors contributed equally.*
***Co-last authors.*

Please address correspondence to:
Raffaele Scarpa
Dipartimento di Medicina Clinica e Chirurgia,
Università di Napoli Federico II,
Via Sergio Pansini 5,
80131 Napoli, Italy.
E-mail: rscarpa@unina.it

Received on March 14, 2024; accepted in revised form on May 13, 2024.

Clin Exp Rheumatol 2024; 42: 2280-2287.

Key words: dactylitis, psoriatic arthritis, pathogenesis, clinical, DMARDs

Competing interests: none declared.

ABSTRACT

Objective. *Psoriatic arthritis (PsA) is an immune-inflammatory disease occurring in a subgroup of patients suffering from psoriasis. Dactylitis is recognised as a hallmark of PsA, being present in about 50% of patients. This article gives an overview of the complexity of psoriatic dactylitis, looking at clinical aspects as well as pathogenetic aspects and subsequent insights into treatment strategies.*

Methods. *The review focuses on the main evidence on pathogenesis, clinical features, and management of psoriatic dactylitis.*

Results. *In recent years, more studies have focused their attention on dactylitis in PsA patients, leading to a greater understanding of its pathogenesis and clinical presentation and to a growing expansion of the therapeutic armamentarium. Dactylitis is frequently associated with more severe PsA phenotype, often representing the initial feature of the disease. Its prompt recognition can be key for addressing early diagnosis and therapy of PsA, thus leading to better clinical and radiographic outcomes.*

Conclusion. *There has been considerable progress in understanding psoriatic dactylitis, but major challenges remain. Although there has been a recent expansion in the therapeutic armamentarium for psoriatic dactylitis, there is still a paucity of evidence on a precision approach to this manifestation.*

Introduction

Dactylitis is a condition in which the soft tissues between the metacarpophalangeal and proximal and/or distal inter-phalangeal joints are diffusely and uniformly swollen to the extent that the actual joint swelling could no longer be independently recognised (1). Therefore, the affected digit appears as a sausage-shaped structure that is easily distinguishable from adjacent ones. Dactylitis can verify in course of several infections such as tuberculosis ("Spina Ventosa"), blastomycosis, leishmaniasis, leprosy, Lyme disease, and soft tissues infections caused by group A β-haemolytic Streptococcus or Staphylococcus Aureus ("blistering distal dactylitis") (2-7). Dactylitis can be also the manifestation of a neoplastic (especially osteoid osteoma) or paraneoplastic process (8, 9), and other conditions such as traumatic events, sickle cell anaemia (caused by bone infarction due to an acute vaso-occlusion), sarcoidosis and gout (10-12). However, dactylitis is usually associated with spondylarthritis (SpA), and in particular it is recognised as the hallmark of psoriatic arthritis (PsA), being present in up to 50% of patients, without gender differences (13, 14). In fact, its prompt recognition can be crucial for reaching an early diagnosis of PsA and its presence can guide the therapeutic strategy (15). Hence, the aim of this review is to characterise the pathogenesis, clinical features, imaging findings and management of PsA dactylitis.

Pathogenesis of psoriatic dactylitis

Pathogenic mechanisms underlying PsA dactylitis are heterogeneous, but still unclarified (16-19). Regarding genetic background, different HLA alleles, such as B*27:05:02 and B*08:01:01-C*07:01:01 are related to a higher risk of dactylitis development; conversely, B*44 haplotypes have been associated to a reduced incidence of this condition (20, 21). Among environmental risk factors, biomechanical stress or repeated microtrauma seem to play a key role in the development of psoriatic dactylitis.

In fact, tendon pulleys, representing the interface between the sheath tendon and the digital soft tissues, are common sites for mechanical and traumatic stimuli. These regions endure significant frictional forces, potentially triggering the onset of flexor tenosynovitis (22, 23). Ultrasound (US) studies suggested that flexor tenosynovitis, extensor tendonitis and synovitis of the interphalangeal joints represent crucial factors sustaining dactylitis (24-27). Magnetic resonance imaging (MRI) studies have showed that tendon sheaths and small entheses associated with the pulleys seem to represent inflammatory sites relevant to the development of psoriatic tenosynovitis. Of note, microentesopathy along the entire length of digit flexor tendon has led to the concept of "digital polyenthesitis" (28-29). Intra-tendinous inflammatory changes occurring at the interface where the extensor tendon exerts pressure on the adjacent bony protuberance of the phalanx ("functional" enthesis) characterises extensor tendonitis (30). Other MRI studies have showed that the circumferential soft tissue oedema starts from the angle of the phalanx upon the insertion of the joint capsule, while the involvement of the nearby bone tissue occurs later (31). Therefore, the most plausible hypotheses suggests that dactylitis begins as a form of enthesitis that subsequently spreads to soft tissues and bone in response to trauma and represents a profound form of Koebner phenomenon. The Deep Koebner phenomenon triggers an inflammatory response by activating innate immune cells such as macrophages, neutrophils, and γδ T cells. This activation drives the production of pro-inflammatory cytokines, including tumour necrosis factor-α (TNF-α), interleukin (IL)-23 and IL-17 (32-39).

Clinical features of psoriatic dactylitis

In PsA patients, dactylitis can be either an isolated manifestation or associated with peripheral and axial involvement. Clinically, dactylitis can be an acute or chronic process, and in case of flares, it can involve a previous involved digit. Acute dactylitis presents as a painful, erythematous swelling of an entire finger or toe, while the chronic form shows as a non-tender, swollen digit, and it is also recognised as "cold" dactylitis. In both cases, dactylitis results from the inflammation of the tendon sheaths, synovium and soft tissues of a whole digit and it causes reduction of mobility of the involved digit (40-43). Dactylitis is usually asymmetrical, can affect multiple digits simultaneously and it involves more frequently feet, often at level of the fourth toe, and less usually hands, with a propensity for the second and third digit of the dominant hand (22). Dactylitis contributes to the reduction of joint function and quality of life of PsA patients (44, 45). Moreover, it is an index of disease severity, as evidenced by a more significant damage of dactylic digits than non-dactylic digits (46), and it can cause digital growth arrest in children (47). Several methods can be useful in clinical practice to assess dactylitis: for example, physicians can simply rely on the count of affected fingers during the clinical examination (48). The Leeds Dactylitis Index (LDI) is a score based on the use of the circumferentor, an instrument that evaluates the tenderness and circumference of a finger or a toe: by comparing the affected digit with the digit on the opposite hand or foot, it is possible to discriminate between a dactylitic digit and a normal digit if at least 10% difference in this ratio is present (49). The Ritchie index grades from 0 to 3 the digital tenderness, while a dichotomous score (0 for absence of tenderness, 1 for presence of tenderness) is used in the LDI-basic (LDI-b). Both Richie index and LDI-b can also be used to assess treatment response (49). LDI correlates with US inflammatory characteristics: in a recent Italian study involving ninety-one hands with dactylitis, the authors associated the ultrasound presence of soft tissue oedema and flexor tenosynovitis in PsA dactylitis with elevated LDI values. The LDI score appears to be mainly determined by the swelling of the digital hand and by the alteration of the periarticular tissues secondary to the proximal interphalangeal joint synovitis (50). A recent study has demonstrated significant differences in the measurement of digital circumference in healthy individuals when stratified by sex and Body Mass Index (BMI), with higher values in men and directly proportional to BMI (51). Therefore, the authors reccommend the inclusion of BMI in the LDI reference tables both in clinical practice and in randomised clinical trials (RCTs), since obesity is a frequent comorbidity in PsA (51, 52). Each digit can be assigned a score of 0 to 3 (no dactylitis, mild, moderate, severe) and the final score, obtained by summing the score for each digit, determines the composite index DSS (Dactylitis Severity Score) which ranges from 0 to 60 (53). Given its contribution in PsA disease activity, dactylitis is part of clinimetric indices such as Psoriatic Arthritis Disease Activity Score (PASDAS) and Composite Psoriatic Disease Activity Index (CPDAI) (54). Of note, Eder and colleagues associated the presence of each dactylitic digit with a 20% increased risk of developing future cardiovascular events, thus correlating the presence and severity of a digital inflammatory state with an atherosclerotic vascular inflammatory state (55).

Imaging findings in psoriatic dactylitis

The diagnosis of dactylitis is based on clinical examination and history supported by imaging studies, especially US and MRI. During dactylitis acute phase, flexor tenosynovitis both in grey scale and power Doppler is dominant (Fig. 1). Chronic phase is characterised by finger swelling and absence of inflammatory signs (cold dactylitis), while joint synovitis is prevalent. This suggests a key role of both peritendinous structures and the extracapsular soft tissues in inflammatory early phases, and a sequential involvement of joint structures (56-58). MRI can be useful in patients with uncertain diagnosis, and it should include T1-weighted saturated fat imaging sequences. At MRI evaluation, flexor tenosynovitis, joint synovitis and digital soft tissue oedema (described as circumferential, involving the flexor or extensor tendons and phalanges) of are the most common findings detectable in the early-onset dactylitis (59). Finger pulleys surround flexor tendons and are normally low-signal-intensity

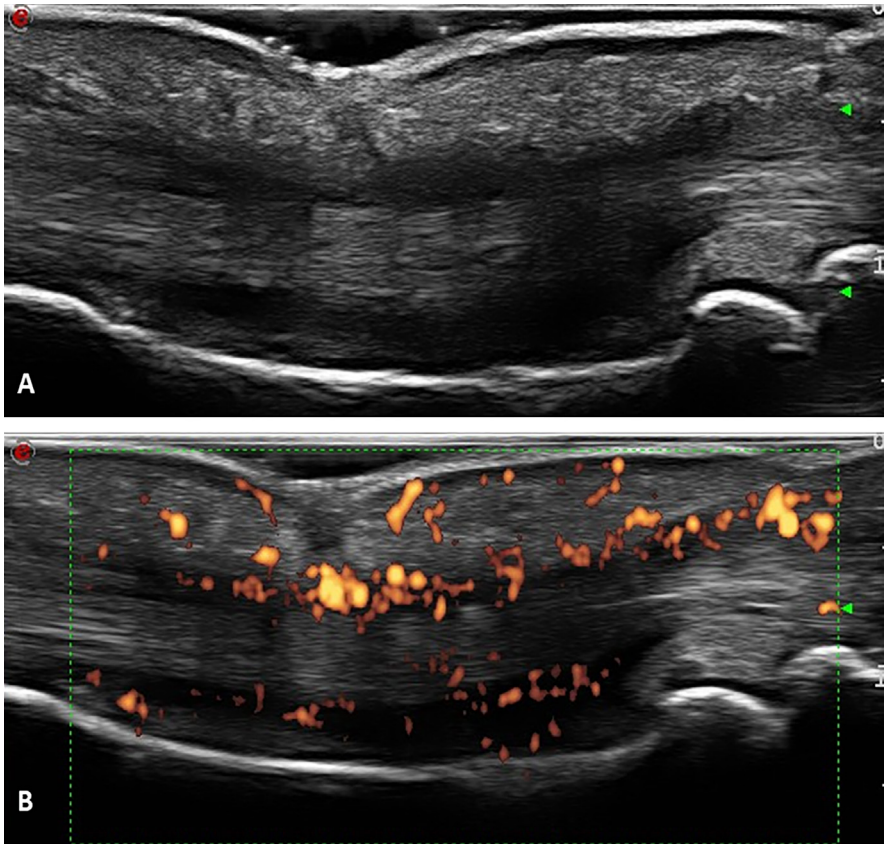

Fig. 1. A: Grey scale and **B**: power Doppler of volar ultrasonography of hand psoriatic dactylitic digit showing flexor tenosynovitis and soft tissue oedema.

structures, while up to 50% of dactylitic fingers show high signal intensity in T2-weighted sequences, especially at A2 pulley level (60). Other enthesis sites, such as the collateral ligament (75%) and extensor tendon (50%), can be involved (61). Extracapsular soft tissues oedema and synovitis of the interphalangeal joints are also frequent (62). Finally, MRI is useful to evidence bone tissue signal abnormalities, such as bone oedema (detected as focal or diffuse increase of signal intensity) and erosions at level of tendon insertions, both representing late findings (63).

Conventional radiography can detect soft tissue swelling and bone abnormalities such as bone cortex erosions, new bone formation and entheseal soft tissue calcifications. However, these aspects are evident only in late phases (56). X-ray imaging is also useful for addressing the diagnosis of acute calcific tendinitis of the fingers. In this case, dactylitis shows as periarticular calcification lesions, especially within the digital flexor tendon, vanishing within 2–3 weeks. 30% of these patients present a recent history of trauma (64). Regarding other causes of dactylitis, sarcoidal dactylitis is characterised by a specific pattern represented by non-caseating granulomatous inflammation of the phalanges, often involving the mid-distal part of the second and third digits, and adjacent soft tissues. In this case, MRI shows dermohypodermal and tenosynovial granulomatous inflammatory lesions associated with focal bone marrow replacement and soft tissue oedema (65). The presence of crystal deposition in the periarticular areas is typical of patients with tofaceous gout (12). Osteoid osteomas should be considered when a young patient presents with dactylitis of hard consistency on palpation due to reactive bone hyperostosis and perilesional tissue swelling which can be characterised especially by computed tomography (CT) (8).

Finally, imaging techniques, and especially US, are also useful for scoring dactylitis severity. The DACTylitis glObal Sonographic (DACTOS) score is obtained by summing the scores of subcutaneous soft tissue oedema, flexor tenosynovitis, peritendon extensor inflammation and synovitis, and it ranges from 0 to 25 (66). Currently, it serves as a valuable scoring system for diagnostic purposes, enabling the assessment of dactylitis complexity, and for monitoring treatment effectiveness (67, 68).

Therapeutical approach

Although dactylitis stands as a prominent domain of PsA, linked with diminished quality of life and radiographic damage progression, only a limited number of RCTs have thus far evaluated dactylitis resolution or improvements in dactylitis-related scores as primary endpoints. Therefore, the main therapeutic data are derived from real life experience and trials in which dactylitis is assessed as a secondary outcome (69). Non-steroidal anti-inflammatory drugs (NSAIDs) and corticosteroids (CS) are used as first-line therapy, according to the Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA) and European League Against Rheumatism (EULAR) recommendations (70-72). These drugs can temporally reduce inflammation and symptoms such as pain and swelling and, in patients with recurrent tenosynovitis episodes or isolated dactylitis, CS injections are a useful therapeutic option. (73, 74). For non-responders, the GRAPPA conditionally recommends the use of methotrexate (MTX), while other conventional synthetic disease-modifying anti-rheumatic drugs (csDMARDs) such as sulfasalazine (SSZ) and leflunomide (LEF) are not considered suitable for the treatment of dactylitis (70). On the other hand, both GRAPPA and EULAR made a strong recommendation in favour of different biologic synthetic disease-modifying anti-rheumatic drugs (bDMARDs) and targeted synthetic disease-modifying anti-rheumatic drugs (tsDMARDs) (70, 72).

Tumour necrosis factor-a inhibitors

Regarding tumour necrosis factor-a inhibitors (TNFi), just one RCT has evaluated dactylitis as primary endpoint. In the GO-DACT trial, MTX naive and bDMARDs naive PsA patients with

dactylitis received golimumab (GOL) plus MTX or placebo plus MTX. At week 24, the first group showed a significant higher proportion of patients achieving at least 20% improvement in LDI and at least 50% improvement in DSS (75). The superiority of GOL over placebo in measuring change from baseline in dactylitis score was also demonstrated in the GO-VIBRANT and CRESPA trials (76, 77). The efficacy of other TNFi on dactylitis has been evaluated as a secondary outcome in several RCTs. In the RAPID-PsA trial, certolizumab pegol (CZP) therapy showed a significantly greater improvement in LDI after 24 weeks when compared to placebo (78). Other RCTs assessed significant improvements in DSS in PsA patients receiving etanercept (ETN), infliximab (IFX) and adalimumab (ADA) therapy, respectively (79-81).

Interleukin-17 inhibitors

Secukinumab (SEC) demonstrated to be superior to placebo in different dactylitis-related outcomes in the FUTURE RCTs (82-84) In the *post-hoc* analysis pf the FUTURE 5 trial, the efficacy of SEC 150 mg and 300 mg on dactylitis was assessed as primary endpoint (85). In particular, more than 80% of patients receiving any dosage of SEC achieved complete resolution of dactylitis after 104 weeks, respect to only 34% of patients receiving placebo. Moreover, median time to resolution of dactylitis was faster with both dosage of SEC (57 days and 85 days, respectively) than placebo (168 days). The efficacy of SEC therapy on the resolution of dactylitis has also been documented to be similar to patients treated with ADA, as demonstrated by the head-to-head EXCEED trial (86). The pooled analysis of the SPIRIT P1 and P2 trials documented that ixekizumab (IXE) induced dactylitis resolution in a significantly higher proportion of patients compared to placebo after 24 weeks of therapy (87). A similar efficacy on dactylitis resolution between IXE and ADA treatment was shown in the SPIRIT head-to-head (H2H) trial (88). In the BE OPTIMAL RCT, PsA patients with baseline dactylitis receiving the dual interleukin-17 inhibitors (IL-17i), IL-17A and IL-17F inhibitor bimekizumab (BKZ) experienced a significantly greater proportion of complete dactylitis resolution than placebo, with similar rates to patients receiving ADA (89). In the analysis of the phase III AMVISION-1 and AMVISION-2 RCTs it emerged that patients receiving 140 mg or 210 mg of the IL-17A receptor subunit A inhibitor brodalumab (BRD) achieved complete resolution of dactylitis with significantly higher proportion compared to patients receiving placebo at weeks 12, 16 and 24, with higher rates with BRD 210 mg (90).

Interleukin-12/23 inhibitors

Both the actions of IL-12 and IL-23 can be inhibited by targeting the shared p40 subunit of these interleukins through the monoclonal antibody ustekinumab (UST) (69). Its efficacy on the dactylitic domain of PsA was mainly assessed as a secondary outocme in the PSUMMIT-1 and PSUMMIT-2 RCTs. In both trials, UST 45 mg and 90 mg proved to be significantly superior to placebo in inducing complete resolution of dactylitis after 24 weeks of therapy, especially in TNFi naive patients (91). Moreover, recent *post-hoc* analysis of the PsABio cohort confirmed complete resolution of dactylitis in 96.6% of patients up to 36 months of UST treatment in a real-world setting (92, 93).

Interleukin-23 inhibitors

The analyses of pooled data from the DISCOVER-1 and DISCOVER-2 RCTs demonstrated significantly higher rates of dactylitis resolution in PsA patients who received guselkumab (GSK) than placebo at week 24. Moreover, 75% of patients with dactylitis at baseline had complete resolution and 80% had at least 70% DSS improvement after 1 year of treatment (94). The KEEPsAKE 1 and KEEPSsAKE 2 RCT showed that risankizumab (RZB) induced a greater proportion of dactylitis resolution up to 52 weeks of therapy when compared to placebo (95, 96). On the other hand, tildrakizumab (TDK) did not improve LDI when compared to placebo at week 24 in a phase IIb study (97).

tsDMARDs

tsDMARDs, such as JAK inhibitors (JAKis) and the phosphodiesterase-4 inhibitor (PDE4i) apremilast (APR), have shown an acceptable safety profile and efficacy in RCTs, improving PsA symptoms, patients reported outcomes and quality of life (98). The OPAL Balance RCT demostrated significant improvements from baseline in DSS in PsA patients treated with tofacitinib (TOF) (99), while in a post-hoc analysis of two phase III studies TOF induced DSS improvements after just one month of therapy which were sustained up to 6 months, irrispective of dactylitis location and with minimal new dactylitis onset (100). Moreover, no gender differences regarding TOF effectiveness on dactylitic domain were observed (101). In the phase III SELECT-PsA 2 study, 50.9% and 58.0% of PsA patients who received upadacitinib (UPA) 15 mg or 30 mg achieved complete resolution of dactylitis after 56 weeks, respectively (102). In addition, a RCT compared both dosage of UPA with ADA and found similar improvements in the proportion of dactylitis complete resolution (103). Despite not still approved for PsA, promising results are emerging with the Tyrosine kinase 2 (TYK2) inhibitor deucravacitinib (DEU) (104), while filgotinib (FIL) was not superior to placebo in the phase II trial EQUATOR (105). In a pooled analysis of the PALACE RCTs, APR has been found effective for the treatment of active PsA, including sustained improvement in dactylitis up to 3 years. In particular, at week 24, patients receiving APR demonstrated a significantly greater mean change in dactylitis count when compared to placebo (106). Moreover, recent real-world Italian and Canadian multicentre studies showed the complete resolution of dactylitis in 44% and 100% of PsA patients after 12 months of APR treatment, respectively (107, 108).

CTLA4-Ig

Abatacept (ABT), a fusion protein formed of the cytotoxic T-cell lymphocyte antigen-4 (CTLA-4) combined with the Fc region of human IgG, binds to CD80 and CD86 on antigen-present-

ing cells (APC) inhibiting costimulatory signals necessary for T-cell activation (109). In a phase III RCT, ABT numerically improved the proportion of PsA patients achieving complete resolution of baseline dactylitis after 24 weeks of therapy compared to placebo (110). Currently ABT use is conditionally recommended by GRAPPA for the treatment of dactylitis (70).

Conclusions

Studies have demonstrated that various drugs exhibit similar efficacy in treating PsA dactylitis. A recent systematic review proved that IL-17i and IL-23i ranked the best for dactylitis resolution, followed by ADA and UST (111). Although there has been a recent expansion in the therapeutic armamentarium for psoriatic dactylitis, there is still little information regarding a precision approach to this manifestation. Translated into a real-life setting, clinicians currently have many effective drugs at their disposal, but queries can arise regarding the type and timing of the right drug to use. Making the matter more challenging is the fact that dactylitis can be a fleeting and self-limiting process. Furthermore, considering its close connection with biomechanical stress, its presence may not necessarily imply a lack of control of the psoriatic disease, for example in manual workers. Therefore, for the therapeutic choice, it is appropriate to consider dactylitis not as an entity *sensu stricto* but as a part of the whole complex psoriatic disease, considering which articular and extra-articular domains and comorbidities are present in each patient (112). However, early data from recent cellular and molecular studies shed lights to a new intriguing therapeutical perspective. Precision medicine allows to overcome the classic clinical approach based on signs and symptoms presented by the patient resulting in a potential better disease control. This approach was initially employed in oncology and subsequently translated to rheumatological diseases, including PsA (113). In this context, Miyagawa and colleagues demonstrated that PsA patient who received a personalised therapy based on their peripheral blood immunophenotype (the proportion of activated Th1 and Th17 allowed to choose therapy with TNFi, IL-17i or IL-12/23i) achieved a better disease control after 6 months than PsA patients who received the standard therapy (114). This immunophenotyping strategy, although it might not include either all Th1 and Th17 activation markers and other cells who play a key role in the pathogenesis of PsA, represents the dawn of a new personalised approach, highlighting the need of a cellular or tissue-based therapeutical strategy (115-117). In conclusion, the diagnosis of psoriatic dactylitis relies on clinical and imaging assessments. Its early recognition facilitates prompt therapy, mitigating functional impairment and improving patients' quality of life. Our review highlights how in recent years more studies than in the past have focused their attention on psoriatic dactylitis, enhancing our comprehension of its pathogenesis and expanding the therapeutic armamentarium. However, the absence of a specific molecular signature for dactylitis and the lack of predictive genetic, immunohistological, or serological biomarkers currently enable the choice of precision medicine-driven therapeutic strategies. Precision medicine for the treatment of PsA and other systemic autoimmune diseases is still a challenge, and further research is needed in this context.

References

1. ROTHSCHILD BM, PINGITORE C, EATON M. DACTYLITIS: implications for clinical practice. *Arthritis Rheum* 1998; 28(1): 41-47. https://doi.org/10.1016/s0049-0172(98)80027-9
2. JHA S, DHOORIA A, JAIN S: Tuberculous dactylitis: a rare form of skeletal tuberculosis. *J Clin Rheumatol* 2020; 26(5): e93. https://doi.org/10.1097/rhu.0000000000000925
3. HUSSEIN S, DAGENAIS P: Blastomycosis arthritis and dactylitis. *IDCases* 2022; 27: e01400. https://doi.org/10.1016/j.idcr.2022.e01400
4. SAKI N, GODARZI H, HEIRAN A: Leishmanial dactylitis: an unusual clinical presentation. *Dermatol Online J* 2017; 23(5): 13030/qt4d65s1m4
5. GUPTA S, LI C, THALLAPALLY VK, SHARMA P, NAHAS J: Chronic hand swelling and dactylitis in leprosy: a case report and review of the literature. *Cureus* 2021; 13(2): e13451. https://doi.org/10.7759/cureus.13451
6. STEPPAT A, SKAARUP ANDERSEN N, ANDREASEN CM: Rare case of Lyme borreliosis in a patient presenting with dactylitis and skin rash. *BMJ Case Rep* 2023; 16(2): e253182. https://doi.org/10.1136/bcr-2022-253182
7. ZHANG LW, WANG WJ, CHEN T: Blistering distal dactylitis. *CMAJ* 2022; 194(5): E167. https://doi.org/10.1503/cmaj.210685
8. BREDA L, SASSANO G, LA BELLA S, GENTILE C, SANSONE L, CHIARELLI F: Intra-articular osteoid osteoma mimicking juvenile psoriatic dactylitis. *Clin Exp Rheumatol* 2022; 40(5): 1058. https://doi.org/10.55563/clinexprheumatol/a55rk0
9. EVIATAR T, ELKAYAM O: Paraneoplastic dactylitis leading to the diagnosis of ovarian cancer. *Isr Med Assoc J* 2019; 21(5): 354-55.
10. DA SILVA JUNIOR GB, DAHER EDE F, DA ROCHA FA: Osteoarticular involvement in sickle cell disease. *Rev Bras Hematol Hemoter* 2012; 34(2): 156-64. https://doi.org/10.5581/1516-8484.20120036
11. ALAWNEH D, AL-SHYOUKH A, EDREES A: TNF inhibitor treating osseous sarcoidosis and dactylitis: case and literature review. *Clin Rheumatol* 2020; 39(7): 2219-22. https://doi.org/10.1007/s10067-020-04964-1
12. ANDRACCO R, ZAMPOGNA G, PARODI M, PAPARO F, CIMMINO MA: Dactylitis in gout. *Ann Rheum Dis* 2010; 69(1): 316. https://doi.org/10.1136/ard.2009.107755
13. OLIVIERI I, PADULA A, SCARANO E, SCARPA R: Dactylitis or "sausage-shaped" digit. *J Rheumatol* 2007; 34(6): 1217-22.
14. LUBRANO E, SCRIFFIGNANO S, FATICA M *et al*.: Psoriatic arthritis in males and females: differences and similarities. *Rheumatol Ther* 2023; 10(3): 589-99. https://doi.org/10.1007/s40744-023-00535-3
15. CASO F, COSTA L, ATTENO M *et al*.: Simple clinical indicators for early psoriatic arthritis detection. *Springerplus* 2014; 3: 759. https://doi.org/10.1186/2193-1801-3-759
16. CASO F, COSTA L, CHIMENTI MS, NAVARINI L, PUNZI L: Pathogenesis of psoriatic arthritis. *Crit Rev Immunol* 2019; 39(5): 361-77. https://doi.org/10.1615/critrevimmunol.2020033243
17. CHIMENTI MS, TRIGGIANESE P, DE MARTINO E *et al*.: An update on pathogenesis of psoriatic arthritis and potential therapeutic targets. *Expert Rev Clin Immunol* 2019; 15(8): 823-36. https://doi.org/10.1080/1744666x.2019.1627876
18. CHIMENTI MS, PERRICONE C, NOVELLI L *et al*.: Interaction between microbiome and host genetics in psoriatic arthritis. *Autoimmun Rev* 2018; 17(3): 276-83. https://doi.org/10.1016/j.autrev.2018.01.002
19. CHIMENTI MS, SUNZINI F, FIORUCCI L *et al*.: Potential role of cytochrome c and tryptase in psoriasis and psoriatic arthritis pathogenesis: focus on resistance to apoptosis and oxidative stress. *Front Immunol* 2018; 30(9): 2363. https://doi.org/10.3389/fimmu.2018.02363
20. WINCHESTER R, MINEVICH G, STESHENKO V *et al*.: HLA associations reveal genetic heterogeneity in psoriatic arthritis and in the psoriasis phenotype. *Arthritis Rheum* 2012; 64(4): 1134-44. https://doi.org/10.1002/art.33415
21. HAROON M, WINCHESTER R, GILES JT,

HEFFERNAN E, FITZGERALD O: Certain class I HLA alleles and haplotypes implicated in susceptibility play a role in determining specific features of the psoriatic arthritis phenotype. *Ann Rheum Dis* 2016; 75(1): 155-62. https://doi.org/10.1136/annrheumdis-2014-205461
22. MCGONAGLE D, TAN AL, WATAD A, HELLIWELL P *et al*.: Pathophysiology, assessment and treatment of psoriatic dactylitis. *Nat Rev Rheumatol* 2019; 15(2): 113-22. https://doi.org/10.1038/s41584-018-0147-9
23. JACQUES P, LAMBRECHT S, VERHEUGEN E *et al*.: Proof of concept: enthesitis and new bone formation in spondyloarthritis are driven by mechanical strain and stromal cells. *Ann Rheum Dis* 2014; 73(2): 437-45. https://doi.org/10.1136/annrheumdis-2013-203643
24. COATES LC, HODGSON R, CONAGHAN PG, FREESTON JE: MRI and ultrasonography for diagnosis and monitoring of psoriatic arthritis. *Best Pract Res Clin Rheumatol* 2012; 26(6): 805-22. https://doi.org/10.1016/j.berh.2012.09.004
25. GIROLIMETTO N, GIOVANNINI I, CREPALDI G *et al*.: Psoriatic dactylitis: current perspectives and new insights in ultrasonography and magnetic resonance imaging. *J Clin Med* 2021; 10(12): 2604. https://doi.org/10.3390/jcm10122604
26. FELBO SK, ØSTERGAARD M, SØRENSEN IJ, TERSLEV L: Which ultrasound lesions contribute to dactylitis in psoriatic arthritis and their reliability in a clinical setting. *Clin Rheumatol* 2021; 40(3): 1061-67. https://doi.org/10.1007/s10067-020-05483-9
27. NAREDO E, LARGO R, OLIVAS-VERGARA O *et al*.: What happens under the flexor tendons of the fingers in dactylitis? *Med Ultrason* 2023; 25(1): 42-47. https://doi.org/10.11152/mu-4026
28. MCGONAGLE D, CONAGHAN PG, EMERY P: Psoriatic arthritis: a unified concept twenty years on. *Arthritis Rheum* 1999; 42(6): 1080-86. https://doi.org/10.1002/1529-0131(199906)42:6<1080::AID-ANR2>3.0.CO;2-7
29. TAN AL, FUKUBA E, HALLIDAY NA, TANNER SF, EMERY P, MCGONAGLE D: High-resolution MRI assessment of dactylitis in psoriatic arthritis shows flexor tendon pulley and sheath-related enthesitis. *Ann Rheum Dis* 2015; 74(1):185-89. https://doi.org/10.1136/annrheumdis-2014-205839
30. BENJAMIN M, MCGONAGLE D: The anatomical basis for disease localisation in seronegative spondyloarthropathy at entheses and related sites. *J Anat* 2001; 199(5): 503-26. https://doi.org/10.1046/j.1469-7580.2001.19950503.x
31. HEALY PJ, GROVES C, CHANDRAMOHAN M, HELLIWELL P: MRI changes in psoriatic dactylitis--extent of pathology, relationship to tenderness and correlation with clinical indices. *Rheumatology* (Oxford) 2008; 47(1): 92-95. https://doi.org/10.1093/rheumatology/kem315
32. AZUAGA AB, RAMÍREZ J, CAÑETE JD: Psoriatic arthritis: pathogenesis and targeted therapies. *Int J Mol Sci* 2023; 24(5): 4901. https://doi.org/10.3390/ijms24054901
33. SCHETT G, RAHMAN P, RITCHLIN C, MCINNES IB, ELEWAUT D, SCHER JU: Psoriatic arthritis from a mechanistic perspective. *Nat Rev Rheumatol* 2022; 18(6): 311-25. https://doi.org/10.1038/s41584-022-00776-6
34. CHIMENTI MS, CASO F, ALIVERNINI S *et al*.: Amplifying the concept of psoriatic arthritis: The role of autoimmunity in systemic psoriatic disease. *Autoimmun Rev* 2019; 18(6): 565-75. https://doi.org/10.1016/j.autrev.2018.11.007
35. SHERLOCK JP, JOYCE-SHAIKH B, TURNER SP *et al*.: IL-23 induces spondyloarthropathy by acting on ROR-γt+ CD3+CD4-CD8- entheseal resident T cells. *Nat Med* 2012; 18(7): 1069-76. https://doi.org/10.1038/nm.2817
36. FIOCCO U, STRAMARE R, MARTINI V *et al*.: Quantitative imaging by pixel-based contrast-enhanced ultrasound reveals a linear relationship between synovial vascular perfusion and the recruitment of pathogenic IL-17A-F+IL-23+ CD161+ CD4+ T helper cells in psoriatic arthritis joints. *Clin Rheumatol* 2017; 36(2): 391-99. https://doi.org/10.1007/s10067-016-3500-x
37. BOUTET MA, NERVIANI A, GALLO AFFLITTO G, PITZALIS C: Role of the IL-23/IL-17 axis in psoriasis and psoriatic arthritis: the clinical importance of its divergence in skin and Joints. *Int J Mol Sci* 2018;19(2):530. https://doi.org/10.3390/ijms19020530
38. BELASCO J, LOUIE JS, GULATI N *et al*.: Comparative genomic profiling of synovium versus skin lesions in psoriatic arthritis. *Arthritis Rheumatol* 2015; 67(4): 934-44. https://doi.org/10.1002/art.38995
39. TINAZZI I, MCGONAGLE D, AYDIN SZ, CHESSA D, MARCHETTA A, MACCHIONI P: 'Deep Koebner' phenomenon of the flexor tendon-associated accessory pulleys as a novel factor in tenosynovitis and dactylitis in psoriatic arthritis. *Ann Rheum Dis* 2018; 77(6): 922-25. https://doi.org/10.1136/annrheumdis-2017-212681
40. RITCHLIN CT, COLBERT RA, GLADMAN DD: Psoriatic arthritis. *N Engl J Med* 2017; 376(10): 957-70. https://doi.org/10.1056/nejmra1505557
41. GLADMAN DD: Clinical features and diagnostic considerations in psoriatic arthritis. *Rheum Dis Clin North Am* 2015; 41(4): 569-79. https://doi.org/10.1016/j.rdc.2015.07.003
42. EDER L, GLADMAN DD: *Curr Rheumatol Rep* 2013; 15(3): 316. https://doi.org/10.1007/s11926-013-0316-4
43. NAPOLITANO M, CASO F, SCARPA R *et al*.: Psoriatic arthritis and psoriasis: differential diagnosis. *Clin Rheumatol* 2016; 35(8): 1893-901. https://doi.org/10.1007/s10067-016-3295-9
44. WALSH JA, OGDIE A, MICHAUD K *et al*.: Impact of key manifestations of psoriatic arthritis on patient quality of life, functional status, and work productivity: Findings from a real-world study in the United States and Europe. *Joint Bone Spine* 2023; 90(3): 105534. https://doi.org/10.1016/j.jbspin.2023.105534
45. LUBRANO E, SCRIFFIGNANO S, FATICA M *et al*.: Potential differences in clinical features, disease activity, function and impact of disease between oligo and polyarticular psoriatic arthritis. *J Psoriasis Psoriatic Arthritis* 2021; 6(1): 38-42. https://doi.org/10.1177/2475530320975379
46. DUBASH S, ALABAS OA, MICHELENA X *et al*.: Dactylitis is an indicator of a more severe phenotype independently associated with greater SJC, CRP, ultrasound synovitis and erosive damage in DMARD-naive early psoriatic arthritis. *Ann Rheum Dis* 2022; 81(4): 490-95. https://doi.org/10.1136/annrheumdis-2021-220964
47. OW NL, NOTTAGE M, BALE P, BUDDHDEV P: Digital physeal arrest following dactylitis in a child. *J Orthop Case Rep* 2023; 13(10):137-40. https://doi.org/10.13107/jocr.2023.v13.i10.3966
48. HEALY PJ, HELLIWELL PS: Measuring dactylitis in clinical trials: which is the best instrument to use? *J Rheumatol* 2007; 34(6): 1302-6.
49. HELLIWELL PS, FIRTH J, IBRAHIM GH, MELSOM RD, SHAH I, TURNER DE: Development of an assessment tool for dactylitis in patients with psoriatic arthritis. *J Rheumatol* 2005; 32(9): 1745-50.
50. GIROLIMETTO N, MACCHIONI P, TINAZZI I *et al*.: Association between Leeds Dactylitis Index and ultrasonographic features: a multicentre study on psoriatic hand dactylitis. *Clin Exp Rheumatol* 2020; 38(6): 1112-17.
51. SILVÉRIO-ANTÓNIO M, RODRIGUES AM, TEIXEIRA F *et al*.: Sex and body mass index impact on digit circumference for Leeds Dactylitis Index calculation. *Clin Exp Rheumatol* 2024; 42(1): 174-77. https://doi.org/10.55563/clinexprheumatol/v78pc5
52. KUMTHEKAR A, OGDIE A: Obesity and psoriatic arthritis: a narrative review. *Rheumatol Ther 2020*; 7(3): 447-56. https://doi.org/10.1007/s40744-020-00215-6
53. OGDIE A, COATES LC, MEASE P: Measuring outcomes in psoriatic arthritis. *Arthritis Care Res* (Hoboken) 2020; 72 (Suppl. 10): 82-109. https://doi.org/10.1002/acr.24242
54. HELLIWELL PS, FITZGERALD O, FRANSEN J *et al*.: The development of candidate composite disease activity and responder indices for psoriatic arthritis (GRACE project). *Ann Rheum Dis* 2013; 72(6): 986-91. https://doi.org/10.1136/annrheumdis-2012-201341
55. EDER L, WU Y, CHANDRAN V *et al*.: Incidence and predictors for cardiovascular events in patients with psoriatic arthritis. *Ann Rheum Dis* 2016; 75(9): 1680-86. https://doi.org/10.1136/annrheumdis-2015-207980
56. HAMARD A, BURNS R, MIQUEL A *et al*.: Dactylitis: a pictorial review of key symptoms. *Diagn Interv Imaging* 2020; 101(4): 193-207. https://doi.org/10.1016/j.diii.2020.01.005
57. D'AGOSTINO MA, TERSLEV L: Imaging evaluation of the entheses: ultrasonography, MRI, and scoring of evaluation. *Rheum Dis Clin North Am* 2016; 42(4): 679-93. https://doi.org/10.1016/j.rdc.2016.07.012
58. GIROLIMETTO N, MACCHIONI P, TINAZZI I *et al*.: Ultrasonographic evidence of predominance of acute extracapsular and chronic intrasynovial patterns in 100 cases of psoriatic hand dactylitis. *J Rheumatol* 2020; 47(2): 227-33.

https://doi.org/10.3899/jrheum.190046
59. SCARANO E, GILIO M, BELMONTE G *et al*.: Morphologic, dynamic and high-resolution microscopy MRI in early-onset spondyloarthritis finger dactylitis. *Skeletal Radiol* 2023; 52(6): 1211-19. https://doi.org/10.1007/s00256-022-04218-y
60. BAKEWELL CJ, OLIVIERI I, AYDIN SZ *et al*.: Ultrasound and magnetic resonance imaging in the evaluation of psoriatic dactylitis: status and perspectives. *J Rheumatol* 2013; 40(12): 1951-57. https://doi.org/10.3899/jrheum.130643
61. TAN AL, FUKUBA E, HALLIDAY NA *et al*.: High-resolution MRI assessment of dactylitis in psoriatic arthritis shows flexor tendon pulley and sheath-related enthesitis. *Ann Rheum Dis* 2015; 74(1): 185-89. https://doi.org/10.1136/annrheumdis-2014-205839
62. FASSIO A, MATZNELLER P, IDOLAZZI L: Recent advances in imaging for diagnosis, monitoring, and prognosis of psoriatic arthritis. *Front Med* (Lausanne) 2020; 7: 551684. https://doi.org/10.3389/fmed.2020.551684
63. KAELEY GS: Enthesitis in psoriatic arthritis (Part 2): imaging. *Rheumatology* (Oxford) 2020; 59(Suppl. 1): i15-i20. https://doi.org/10.1093/rheumatology/keaa040
64. SIEGAL DS, WU JS, NEWMAN JS *et al*.: Calcific tendinitis: a pictorial review. *Can Assoc Radiol J* 2009; 60(5): 263-72. https://doi.org/10.1016/j.carj.2009.06.008
65. RODRIGUEZ-GOMEZ M, FERNANDEZ-SUEIRO JL, WILLISCH A *et al*.: Multifocal dactylitis as the sole clinical expression of sarcoidosis. *J Rheumatol* 2000; 27(1): 245-47.
66. ZABOTTI A, SAKELLARIOU G, TINAZZI I *et al*.: Novel and reliable DACTylitis glObal Sonographic (DACTOS) score in psoriatic arthritis. *Ann Rheum Dis* 2020; 79(8): 1037-43. https://doi.org/10.1136/annrheumdis-2020-217191
67. GIROLIMETTO N, ZABOTTI A, TINAZZI I *et al*.: Sensitivity to change and clinical correlations of the novel DACtylitis glObal Sonographic (DACTOS) score in psoriatic arthritis. *Rheumatology* (Oxford) 2021; 60(9): 4103-11. https://doi.org/10.1093/rheumatology/keaa885
68. GIROLIMETTO N, MACCHIONI P, POSSEMATO N *et al*.: Musculoskeletal ultrasound in monitoring clinical response to treatment in acute symptomatic psoriatic dactylitis: results from a multicentre prospective observational study. *J Clin Med* 2020 27; 9(10): 3127. https://doi.org/10.3390/jcm9103127
69. SUNZINI F, D'ANTONIO A, FATICA M *et al*.: What's new and what's next for biological and targeted synthetic treatments in psoriatic arthritis? *Expert Opin Biol Ther* 2022; 22(12): 1545-59. https://doi.org/10.1080/14712598.2022.2152321
70. COATES LC, SORIANO ER, CORP N *et al*.: Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA): updated treatment recommendations for psoriatic arthritis 2021. *Nat Rev Rheumatol* 2022; 18(8): 465-79. https://doi.org/10.1038/s41584-022-00798-0
71. PALOMINOS PE, FERNÁNDEZ-ÁVILA DG, COATES LC *et al*.: Management of dactylitis in patients with psoriatic arthritis: an updated literature review informing the 2021 GRAPPA treatment recommendations. *J Rheumatol* 2023; 50(2): 265-78. https://doi.org/10.3899/jrheum.220311
72. GOSSEC L, KERSCHBAUMER A, FERREIRA RJO *et al*.: EULAR recommendations for the management of psoriatic arthritis with pharmacological therapies: 2023 update. *Ann Rheum Dis* 2024; 83(6): 706-19. https://doi.org/10.1136/ard-2024-225531
73. GIROLIMETTO N, MACCHIONI P, TINAZZI I *et al*.: Ultrasound effectiveness of steroid injection for hand psoriatic dactylitis: results from a longitudinal observational study. *Rheumatol Ther* 2021; 8(4): 1809-26. https://doi.org/10.1007/s40744-021-00383-z
74. CARRIERO A, LUBRANO E, PICERNO V, PADULA AA, D'ANGELO S: Corticosteroid injection treatment for dactylitis in psoriatic arthritis. Ther *Adv Musculoskelet Dis* 2021; 13: 1759720X211041864. https://doi.org/10.1177/1759720x211041864
75. VIEIRA-SOUSA E, ALVES P, RODRIGUES AM *et al*.: GO-DACT: a phase 3b randomised, double-blind, placebo-controlled trial of GOlimumab plus methotrexate (MTX) versus placebo plus MTX in improving DACTylitis in MTX-naive patients with psoriatic arthritis. *Ann Rheum Dis* 2020; 79(4): 490-98. https://doi.org/10.1136/annrheumdis-2019-216500
76. KAVANAUGH A, HUSNI ME, HARRISON DD *et al*.: Safety and efficacy of intravenous golimumab in patients with active psoriatic arthritis: results through week twenty-four of the GO-VIBRANT study. *Arthritis Rheumatol* 2017; 69(11): 2151-61. https://doi.org/10.1002/art.40226
77. CARRON P, VARKAS G, CYPERS H *et al*.: Anti-TNF-induced remission in very early peripheral spondyloarthritis: the CRESPA study. *Ann Rheum Dis* 2017; 76(8): 1389-95. https://doi.org/10.1136/annrheumdis-2016-210775
78. MEASE PJ, FLEISCHMANN R, DEODHAR AA *et al*.: Effect of certolizumab pegol on signs and symptoms in patients with psoriatic arthritis: 24-week results of a Phase 3 double-blind randomised placebo-controlled study (RAPID-PsA). *Ann Rheum Dis* 2014; 73(1): 48-55. https://doi.org/10.1136/annrheumdis-2013-203696
79. STERRY W, ORTONNE JP, KIRKHAM B *et al*.: Comparison of two etanercept regimens for treatment of psoriasis and psoriatic arthritis: PRESTA randomised double blind multicentre trial. *BMJ* 2010; 340: c147. https://doi.org/10.1136/bmj.c147
80. ANTONI CE, KAVANAUGH A, KIRKHAM B *et al*.: Sustained benefits of infliximab therapy for dermatologic and articular manifestations of psoriatic arthritis: results from the infliximab multinational psoriatic arthritis controlled trial (IMPACT). *Arthritis Rheum* 2005; 52(4): 1227-36. https://doi.org/10.1002/art.20967
81. MEASE PJ, GLADMAN DD, RITCHLIN CT *et al*.: Adalimumab for the treatment of patients with moderately to severely active psoriatic arthritis: results of a double-blind, randomized, placebo-controlled trial. *Arthritis Rheum* 2005; 52(10): 3279-89. https://doi.org/10.1002/art.21306
82. KAVANAUGH A, MCINNES IB, MEASE PJ *et al*.: Efficacy of subcutaneous secukinumab in patients with active psoriatic arthritis stratified by prior tumor necrosis factor inhibitor use: results from the randomized placebo-controlled FUTURE 2 study. *J Rheumatol* 2016; 43(9): 1713-17. https://doi.org/10.3899/jrheum.160275
83. NASH P, MEASE PJ, MCINNES IB *et al*.: Efficacy and safety of secukinumab administration by autoinjector in patients with psoriatic arthritis: results from a randomized, placebo-controlled trial (FUTURE 3). *Arthritis Res Ther* 2018; 20(1): 47. https://doi.org/10.1186/s13075-018-1551-x
84. MEASE P, VAN DER HEIJDE D, LANDEWÉ R *et al*.: Secukinumab improves active psoriatic arthritis symptoms and inhibits radiographic progression: primary results from the randomised, double-blind, phase III FUTURE 5 study. *Ann Rheum Dis* 2018; 77(6): 890-97. https://doi.org/10.1136/annrheumdis-2017-212687
85. KIRKHAM B, NASH P, REINA D *et al*.: Efficacy of secukinumab on dactylitis in patients with active psoriatic arthritis from the FUTURE 5 study. *Clin Exp Rheumatol* 2023; 41(3): 589-96. https://doi.org/10.55563/clinexprheumatol/vezf95
86. GOTTLIEB AB, MEROLA JF, REICH K *et al*.: Efficacy of secukinumab and adalimumab in patients with psoriatic arthritis and concomitant moderate-to-severe plaque psoriasis: results from EXCEED, a randomized, double-blind head-to-head monotherapy study. *Br J Dermatol* 2021; 185(6): 1124-34. https://doi.org/10.1111/bjd.20413
87. GLADMAN DD, ORBAI AM, KLITZ U *et al*.: Ixekizumab and complete resolution of enthesitis and dactylitis: integrated analysis of two phase 3 randomized trials in psoriatic arthritis. *Arthritis Res Ther* 2019; 21(1): 38. https://doi.org/10.1186/s13075-019-1831-0
88. SMOLEN JS, MEASE P, TAHIR H *et al*.: Multicentre, randomised, open-label, parallel-group study evaluating the efficacy and safety of ixekizumab versus adalimumab in patients with psoriatic arthritis naïve to biological disease-modifying antirheumatic drug: final results by week 52. *Ann Rheum Dis* 2020; 79(10): 1310-19. https://doi.org/10.1136/annrheumdis-2020-217372
89. MCINNES IB, ASAHINA A, COATES LC *et al*.: Bimekizumab in patients with psoriatic arthritis, naive to biologic treatment: a randomised, double-blind, placebo-controlled, phase 3 trial (BE OPTIMAL). *Lancet* 2023; 401(10370): 25-37. https://doi.org/10.1016/S0140-6736(22)02302-9
90. MEASE PJ, HELLIWELL PS, HJULER KF, RAYMOND K, MCINNES IB: Brodalumab in psoriatic arthritis: results from the randomised phase III AMVISION-1 and AMVISION-2 trials. *Ann Rheum Dis* 2021;80(2):185-193. https://doi.org/10.1136/annrheumdis-2019-216835
91. MCINNES IB, CHAKRAVARTY SD, APAOLAZA I *et al*.: Efficacy of ustekinumab in biologic-naïve patients with psoriatic arthritis by prior treatment exposure and disease duration: data from PSUMMIT 1 and PSUMMIT

2. *RMD Open* 2019; 5(2): e000990. https://doi.org/10.1136/rmdopen-2019-000990
92. GOSSEC L, THEANDER E, CHAKRAVARTY SD *et al*.: Response to treatment in psoriatic arthritis, the effect of age: analysis of patients receiving ustekinumab in the PsABio real-world study. *Arthritis Res Ther* 2023; 25(1): 100. https://doi.org/10.1186/s13075-023-03078-8
93. GOSSEC L, SIEBERT S, BERGMANS P *et al*.: Long-term effectiveness and persistence of ustekinumab and TNF inhibitors in patients with psoriatic arthritis: final 3-year results from the PsABio real-world study. *Ann Rheum Dis* 2023; 82(4): 496-506. https://doi.org/10.1136/ard-2022-222879
94. MCGONAGLE D, MCINNES IB, DEODHAR A *et al*.: Guselkumab, a selective interleukin-23 p19 subunit inhibitor, resolves dactylitis in patients with active psoriatic arthritis: pooled results through week 52 from two phase 3 studies. *ACR Open Rheumatol* 2023; 5(4): 227-40. https://doi.org/10.1002/acr2.11537
95. KRISTENSEN LE, KEISERMAN M, PAPP K *et al*.: Efficacy and safety of risankizumab for active psoriatic arthritis: 52-week results from the KEEPsAKE 1 study. *Rheumatology* (Oxford) 2023; 62(6): 2113-21. https://doi.org/10.1093/rheumatology/keac607
96. ÖSTÖR A, VAN DEN BOSCH F, PAPP K *et al*.: Efficacy and safety of risankizumab for active psoriatic arthritis: 52-week results from the KEEPsAKE 2 study. *Rheumatology* (Oxford) 2023; 62(6): 2122-29. https://doi.org/10.1093/rheumatology/keac605
97. MEASE PJ, CHOHAN S, FRUCTUOSO FJG *et al*.: Efficacy and safety of tildrakizumab in patients with active psoriatic arthritis: results of a randomised, double-blind, placebo-controlled, multiple-dose, 52-week phase IIb study. *Ann Rheum Dis* 2021; 80(9): 1147-57. https://doi.org/10.1136/annrheumdis-2020-219014
98. HARKINS P, BURKE E, SWALES C *et al*.: Are Janus kinase inhibitors safe and effective in treating the key clinical domains of psoriatic arthritis? A systematic review and meta-analysis. *Int J Rheum Dis* 2023; 26(1): 31-42. https://doi.org/10.1111/1756-185x.14447
99. NASH P, COATES LC, KIVITZ AJ *et al*.: Safety and efficacy of tofacitinib in patients with active psoriatic arthritis: interim analysis of OPAL Balance, an open-label, long-term extension Study. *Rheumatol Ther* 2020; 7(3): 553-80. https://doi.org/10.1007/s40744-020-00209-4
100. ORBAI AM, MEASE PJ, HELLIWELL PS *et al*.: Effect of tofacitinib on dactylitis and patient-reported outcomes in patients with active psoriatic arthritis: post-hoc analysis of phase III studies. *BMC Rheumatol* 2022; 6(1): 68. https://doi.org/10.1186/s41927-022-00298-4
101. EDER L, GLADMAN DD, MEASE P *et al*.: Sex differences in the efficacy, safety and persistence of patients with psoriatic arthritis treated with tofacitinib: a post-hoc analysis of phase 3 trials and long-term extension. *RMD Open* 2023; 9(1): e002718. https://doi.org/10.1136/rmdopen-2022-002718
102. MEASE PJ, LERTRATANAKUL A, PAPP KA *et al*.: Upadacitinib in patients with psoriatic arthritis and inadequate response to biologics: 56-week data from the randomized controlled phase 3 SELECT-PsA 2 study. *Rheumatol Ther* 2021; 8(2): 903-19. https://doi.org/10.1007/s40744-021-00305-z
103. MCINNES IB, ANDERSON JK, MAGREY M *et al*.: Trial of upadacitinib and adalimumab for psoriatic arthritis. *N Engl J Med* 2021; 384(13): 1227-39. https://doi.org/10.1056/nejmoa2022516
104. LÉ AM, PUIG L, TORRES T: Deucravacitinib for the treatment of psoriatic disease. *Am J Clin Dermatol* 2022; 23(6): 813-22. https://doi.org/10.1007/s40257-022-00720-0
105. MEASE P, COATES LC, HELLIWELL PS *et al*.: Efficacy and safety of filgotinib, a selective Janus kinase 1 inhibitor, in patients with active psoriatic arthritis (EQUATOR): results from a randomised, placebo-controlled, phase 2 trial. *Lancet* 2018; 392(10162): 2367-77. https://doi.org/10.1016/S0140-6736(18)32483-8
106. GLADMAN DD, KAVANAUGH A, GÓMEZ-REINO JJ *et al*.: Therapeutic benefit of apremilast on enthesitis and dactylitis in patients with psoriatic arthritis: a pooled analysis of the PALACE 1-3 studies. *RMD Open* 2018; 4(1): e000669. https://doi.org/10.1136/rmdopen-2018-000669
107. LO GULLO A, BECCIOLINI A, PARISI S *et al*.: Therapeutic effects of apremilast on enthesitis and dactylitis in real clinical setting: an Italian multicenter study. *J Clin Med* 2023; 12(12): 3892. https://doi.org/10.3390/jcm12123892
108. CHANDRAN V, BESSETTE L, THORNE C *et al*.: Use of apremilast to achieve psoriatic arthritis treatment goals and satisfaction at 1 year in the Canadian real-world APPRAISE study. *Rheumatol Ther* 2024; 11(2): 443-55. https://doi.org/10.1007/s40744-024-00641-w
109. HERRERO-BEAUMONT G, MARTÍNEZ CALATRAVA MJ, CASTAÑEDA S: Abatacept mechanism of action: concordance with its clinical profile. *Reumatol Clin* 2012; 8(2): 78-83. https://doi.org/10.1016/j.reuma.2011.08.002
110. MEASE PJ, GOTTLIEB AB, VAN DER HEIJDE D *et al*.: Efficacy and safety of abatacept, a T-cell modulator, in a randomised, double-blind, placebo-controlled, phase III study in psoriatic arthritis. *Ann Rheum Dis* 2017; 76(9): 1550-58. https://doi.org/10.1136/annrheumdis-2016-210724
111. MCINNES IB, SAWYER LM, MARKUS K *et al*.: Targeted systemic therapies for psoriatic arthritis: a systematic review and comparative synthesis of short-term articular, dermatological, enthesitis and dactylitis outcomes. *RMD Open* 2022; 8(1): e002074. https://doi.org/10.1136/rmdopen-2021-002074
112. AL-MOSSAWI H, TAAMS LS, GOODYEAR CS *et al*.: Precision medicine in psoriatic arthritis: how should we select targeted therapies? *Lancet Rheumatol* 2019; 1(1): e66-e73. https://doi.org/10.1016/S2665-9913(19)30008-6
113. RITCHLIN CT, PENNINGTON SR, REYNOLDS NJ *et al*.: Moving toward precision medicine in psoriasis and psoriatic arthritis. *J Rheumatol Suppl* 2020; 96: 19-24. https://doi.org/10.3899/jrheum.200122
114. MIYAGAWA I, NAKAYAMADA S, TANAKA Y: Optimal biologic selection for treatment of psoriatic arthritis: the approach to precision medicine. *Curr Rheumatol Rep* 2019; 21(5): 21. https://doi.org/10.1007/s11926-019-0817-x
115. NAJM A, GOODYEAR CS, MCINNES IB *et al*.: Phenotypic heterogeneity in psoriatic arthritis: towards tissue pathology-based therapy. *Nat Rev Rheumatol* 2023; 19(3): 153-65. https://doi.org/10.1038/s41584-022-00874-5
116. GIACOMELLI R, AFELTRA A, BARTOLONI E *et al*.: The growing role of precision medicine for the treatment of autoimmune diseases; results of a systematic review of literature and Experts' Consensus. *Autoimmun Rev* 2021; 20(2): 102738. https://doi.org/10.1016/j.autrev.2020.102738
117. OOMS A, AL-MOSSAWI H, BENNETT L *et al*.: Optimising psoriatic arthritis therapy with immunological methods to increase standard evaluation: the protocol of an open-label multicentre, parallel-group, two-arm randomised controlled study evaluation precision medicine approach in the treatment of psoriatic arthritis. *BMJ Open* 2023; 13(9): e078539. https://doi.org/10.1136/bmjopen-2023-078539