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Characteristic of psoriatic arthritis patient with disease activity based on DAPSA score: A literature review

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Abstract

Psoriatic arthritis (PsA) is a chronic inflammation that affects peripheral and axial joints. The prevalence of PsA tends to increase in recent years. As many as 0.1 - 0.2% of the general population suffer from PsA. A study stated that out of 1,442,357 people with PsA, 21,825 deaths occurred. This mortality rate may continue to increase over time. PsA is most common in individuals aged 40 to 50 years with the incidence of PsA in men and women being almost equal. PsA cannot be underestimated and needs proper diagnosis and assessment of disease activity for good management. Early diagnosis is very important in PsA. The diagnosis of PsA can be made based on the Classification Criteria for Psoriatic Arthritis (CASPAR). Assessment of disease activity is essential in the management of PsA. Based on several studies, the most widely and easily used PsA disease activity assessment instrument is Disease Activity in Psoriatic Arthritis (DAPSA) score. Literature reviews on psoriatic arthritis patient with disease activity are still limited. Therefore, researchers are interested in conducting research on the characteristic of psoriatic arthritis patient with disease activity based on DAPSA score. However, further research is needed with more comprehensive variables to explore the characteristic and disease activity of psoriatic arthritis.

Keywords: Psoriatic arthritis; Disease activity; DAPSA Score; Chronic; Joint

1. Introduction

Psoriatic arthritis (PsA) is a chronic inflammation that affects peripheral and axial joints [17]. The prevalence of PsA tends to increase in recent years [42]. As many as 0.1 - 0.2% of the general population suffer from PsA. Of these PsA patients, 10 - 40% of them have been diagnosed with psoriasis before [33]. A study stated that out of 1,442,357 people with PsA, 21,825 deaths occurred [28]. This mortality rate may continue to increase over time. PsA is most common in individuals aged 40 to 50 years with the incidence of PsA in men and women being almost equal [27].

Psoriatic arthritis (PsA) can cause both intraarticular and extraarticular clinical manifestations [35]. The clinical manifestations of PsA related to synovial tissue, enthesitis, arthritis, skin and nail lesions that cause stiffness, pain, swelling around the joints or back, and extraarticular [20]. These clinical manifestations become a problem in the patient's daily life, and can even reduce the patient's quality of life and productivity. PsA cannot be underestimated and needs proper diagnosis and assessment of disease activity for good management [33]. Early diagnosis is very important in PsA. The diagnosis of PsA can be made based on the Classification Criteria for Psoriatic Arthritis (CASPAR). Early diagnosis of PsA should be intensified by assessing clinical signs and symptoms at clinical visits to minimize irreversible damage to joints, skin, and other manifestations [35].

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Assessment of disease activity is essential in the management of PsA. Based on several studies, the most widely and easily used PsA disease activity assessment instrument is Disease Activity in Psoriatic Arthritis (DAPSA) score. This index is a combination of the number of swollen joints, the patient's general assessment and pain, and acute phase reactants [2, 4]. The DAPSA score calculation formula is performed by summing tender joint counts (TJC68), swollen joint counts (SJC66), patient global activity (PtGA), patient pain (PP), and C-reactive protein (CRP) [5, 13]. DAPSA is widely used for observational studies and clinical trials because it is easy to use and has good validity to provide an overview of PsA disease activity [38].

Literature reviews on psoriatic arthritis patient with disease activity are still limited. Therefore, researchers are interested in conducting research on the characteristic of psoriatic arthritis patient with disease activity based on DAPSA score.

2. Review Content

2.1. Psoriatic Arthritis

2.1.1. Definition of Psoriatic Arthritis

Psoriasis arthritis (PsA) is a multisystemic and chronic inflammation of the musculoskeletal system associated with cutaneous psoriasis and commonly affects the axial skeleton, such as the peripheral joints of the hands and feet [29]. PsA is a seronegative inflammatory spondyloarthropathy that is most commonly comorbid with psoriasis [6, 7]. The main manifestations of PsA are skin manifestations (skin and nail psoriasis) and musculoskeletal manifestations (arthritis, enthesitis, and dactylitis) [43]. The prevalence of PsA tends to increase in recent years [42]. As many as 0.1 - 0.2% of the general population suffer from PsA [33]. PsA is most common in individuals aged 40 to 50 years with the incidence of PsA in men and women being almost equal [27].

2.1.2. Pathophysiology of Psoriatic Arthritis

Psoriasis arthritis (PsA) is characterized by inflammation that results in uncontrolled proliferation and dysfunctional differentiation of keratinocytes and low-grade inflammation. This chronic inflammation occurs due to disturbances in the body's immune system, triggering manifestations in the form of psoriasis plaques and joint inflammation. Psoriasis plaques that occur show acanthosis (epidermal hyperplasia) on histology examination which overlays inflammatory infiltrates. This inflammatory infiltrate consists of dermal dendritic cells, macrophages, T cells, and neutrophils [34]. The pathogenesis of PsA involves several components, such as genetic factors, environment, and immune system [13].

Genetics

Psoriasis arthritis (PsA) has a genetic component supported by patterns of familial aggregation. Offspring of individuals with PsA have a high risk of developing PsA [8]. Individuals who are monozygotic twins are at two to three time’s higher risk of developing PsA than normal individuals or dizygotic twins [18].

Environment

Environmental factors are related to the pathogenesis of PsA, such as trauma and stress. Some studies say that trauma is more likely to trigger PsA compared to rheumatoid arthritis (RA) or ankylosing spondylitis (AS) [29, 32]. Mechanical trauma can cause *koebner phenomenon* with suspected end nerve activation at the trauma site [40]. Physical trauma can trigger the nervous system with the release of substance P in the skin and joints. Substance P activates endothelial cells to produce vascular endothelial angiogenic factors (VEGF), transforming growth factor (TGF) and angiopoetin 2 (ANG2). The production of these compounds will trigger abnormal angiogenesis and recruitment of TDC8+ cells that will cause chronic inflammation in the skin and joints [9, 12].

Immunity

Immune factors that play a role in PsA are proinflammatory cytokines, especially interleukin (IL)-17. IL-17 cytokines are the main effectors in the pathogenesis of PsA, especially IL-17A. T cells also have a role in the pathogenesis of PsA and are associated with the secretion of proinflammatory cytokines, including tumor necrosis factor alpha (TNF-α), IL17A, IL-22, interferon gamma (IFN-γ), and other cytokines. A study states that the main source of IL-17A in PsA is T helper 17 (Th17) cells. Th17 cells can also be differentiated and stimulated to produce the cytokine IL-23. Cytokine IL-23 promotes the survival of Th17 cells [13, 18]. Increased numbers of Th17 cells are found in the blood and skin of psoriasis patients and in the synovial fluid of PsA patients [34].

2.1.3. Clinical Manifestation of Psoriatic Arthritis

Clinical manifestation of psoriatic arthritis can be divided into intraarticular and extraarticular.

Intraarticular Manifestation

Arthritis

The arthritis manifestations of PsA, according to Wright and Moll's criteria in 1973, are described into 5 types; asymmetric oligoarthritis, symmetric polyarthritis, predominant distal interphalangeal (DIP), predominant spondyloarthritis, and destructive arthritis (mutilans) [13, 21]. Some studies state that approximately 63% of PsA patients experience polyarthritis and 13% of PsA patients experience oligoarthritis. Distal interphalangeal joint (DIP) predominance is experienced by less than 5% of patients, but DIP predominance can develop in all types of arthritis manifestations of PsA patients. The rarer type is predominantly spondyloarthritis although spinal manifestations are quite common in PsA patients (approximately 40 - 70%). The destructive type of arthritis, mutilating arthritis, is the least common type and is associated with flail joints. Mutilated arthritis can occur in patients who have uncontrolled PsA [12, 13, 34].

Enthesitis

Enthesitis is an inflammation of the tendon and ligament insertions in the bone. Enthesitis is a feature of all spondyloarthropathies and is a hallmark of PsA. Enthesitis is found in 38% of patients with PsA. The most common body parts affected by enthesitis are the achilles tendon and plantar fascia insertions. Patients complain of pain in these parts of the body and sometimes look swollen on physical examination. Patients with enthesitis may present with no symptoms. Diagnosis of enthesitis can be made using ultrasonography (US) which is more sensitive than clinical palpation [13, 14].

Dactylitis

Dactylitis is a diffuse swelling of the fingers (hands or feet) that has *sausage-shaped characteristics*. Dactylitis can be found in 29% to 33.5% of PsA patients in the early stages and 48% of patients experience dactylitis in the follow-up stage [13]. Dactylitis in PsA patients can occur acutely with manifestations of swelling, redness of the skin, and pain or chronically with manifestations of swelling without inflammation. Dactylitis is also closely related to polyarthritis, bone formation, and bone erosion [28, 35].

Spinal manifestation

Spinal manifestations in PsA involve axial joints (spondylitis, sacroilitis, and arthritis of the hip and shoulder joints). Manifestations in the sacroiliac joints and spine in PsA are rare when compared to peripheral joints. The cervical is the spinal part that is commonly involved in PsA. More than 50% of PsA cases have cervical symptoms whose progressivity is comparable to the severity of peripheral joint arthritis. Inflammation in the sacroiliac joint can be seen unilaterally and back pain and pain in the buttock area are obtained and can be asymptomatic [13, 15].

Extraarticular Manifestation

Psoriatic skin

The skin manifestation in PsA patients is psoriatic skin lesions characterized by the presence of erythema plaques and thickened and silver skin. These plaques can form due to a response to infection or trauma known as the *koebner phenomenon* [1, 3]. After 7-14 days of injury, about 25% of PsA patients experience this *koebner phenomenon*. In this situation, the skin lesions that occur are the patient's traumatized skin (movement, scalp, redness, etc.) [10, 22].

Psoriatic nail

Psoriatic nail is a manifestation that is often found in PsA. Clinical manifestations of psoristic nail depend on the structures that experience the inflammatory process [34]. Psoriatic nail found in 83% of PsA patients and often result in functional impairment and pain. The severity of psoriatic nail is closely related to enthesitis, progressive arthritis, and polyarticular disorders [16, 23].

Others

Extraarticular manifestations in PsA are not as common when compared to rheumatoid arthritis (RA) patients. These extraarticular manifestations include iritis and uveitis which are found in 7-8% of PsA who have spinal manifestations. Some studies state that inflammatory bowel disease (IBD) is one of the extraarticular manifestations detected in biopsies of PsA patients and is usually asymptomatic [10, 18]. Another extraarticular manifestation can be chronic kidney disease (CKD) which was found in 42.85% of cases. PsA is also associated with obesity, type 2 diabetes mellitus, hypertension, metabolic syndrome, fatty liver, and increased risk of cardiovascular disease [35]. Extraarticular

manifestations that occur in PsA patients are closely related to the psoriatic area severity index (PASI), severity of joint pain, and joint damage [13].

2.1.4. Diagnosis of Psoriatic Arthritis

The diagnosis of PsA is based on the Classification Criteria for Psoriatic Arthritis (CASPAR). PsA can occur in patients with musculoskeletal inflammatory diseases that include joint, spinal, or enthesitis that score at least three of the five CASPAR points with at least 1-2 points of evidence of psoriasis. This criterion has a specificity of 98.7% and a sensitivity of 91.4%. The classification of PsA is based on the following CASPAR table. Early diagnosis of PsA and early initiation of therapy are important to improve long-term outcomes [33, 39]. Screening PsA patients based on their clinical signs and symptoms at each clinical visit can minimize the delay in PsA diagnosis [35].

Table 1 Classification Criteria for Psoriatic Arthritis (CASPAR)

Criteria	**Description**	**Point**
Evidence of psoriasis (one of a, b, c)		
Current psoriasis	Psoriatic skin or scalp disease present today as judged by a rheumatologist or dermatologist	2
Personal history of psoriasis	A history of psoriasis that may be obtained from patient, family doctor, dermatologist, rheumatologist, or other qualified health-care provider	1
Family history	A history of psoriasis in a first or second degree relative according to the patient's reporting	1
Psoriatic nail dystrophy	Typical psoriatic nail dystrophy including onycholysis, pitting and hyperkeratosis observed on current physical examination	1
A negative rheumatoid factor	By any method except latex but preferably by ELISA or nephelometry, according to the local laboratory reference range	1
Dactylitis (a or b)		
Current dactylitis	Current swelling of entire digit	1
History of dactylitis	A history of dactylitis recorded by a rheumatologist	1
Radiological evidence of juxta-articular new bone formation	III-defined ossification near joint margins (but excluding osteophyte formation) on plain X-rays of hands or feet	1

2.2. Disease Activity

Psoriatic arthritis activity has varied clinical manifestations that are not easy to assess. Assessment of the disease activity should include assessment of the manifestations of joint inflammation, psoriatic skin, global by the patient, pain assessment (Visual Analog Scale or VAS), physical function, and patient quality of life (QoL) [13]. Currently, PsA disease activity assessment includes several domains, including the Composite Psoriatic Disease Activity Index (CPDAI), Psoriatic Arthritis Disease Activity Score (PASDAS), and Disease Activity in Psoriatic Arthritis (DAPSA) [15, 26].

The CPDAI instrument assesses 5 components, namely peripheral joint manifestations, skin, enthesitis, dactylitis, and spinal. The CPDAI is a sensitive instrument in detecting changes in the enthesitis, dactylitis, and skin domains. However, the CPDAI does not involve laboratory signs of inflammation and has complex mathematical calculations [19]. The PASDAS instrument is an indicator that combines patient and physician assessment. The components assessed in PASDAS are global disease, tender joint counts, swollen joint counts, dactylitis, enthesitis, physical components, and C-reactive protein (CRP). The weakness of this PASDAS instrument is that the calculations are complicated and long [36].

Furthermore, the Disease Activity in Psoriatic Arthritis (DAPSA) instrument is an instrument that combines two indicators of joint disorders. DAPSA was developed through cohorts and validated by clinical trials. Assessment of PsA disease activity using the DAPSA score includes components of swollen joint counts (SJC), tender joint counts (TJC), CRP levels, pain scale with VAS, and a scale to assess the patient's global activity [20, 37].

2.3. Disease Activity in Psoriatic Arthritis (DAPSA) Score

DAPSA score is a continuous index that measures PsA disease activity consisting of functional and structural impairments [30, 43]. DAPSA score includes several parameters in PsA disease activity. DAPSA score parameters are joint involvement assessed by 68 tender joint counts (TJC68), 66 swollen joint counts (SJC66), patient global assessment (PtGA), patient pain (PP), and acute phase serum response represented by C-reactive protein (CRP) [38]. The DAPSA score calculation formula is performed by summing DAPSA score parameters [13].

The use of DAPSA score to assess PsA disease activity has been validated because it shows correlation, discrimination and criterion validity, and its sensitivity to changes in observational data and clinical trials [37]. DAPSA has a sensitivity of 77.17% and specificity of 88.75% in describing minimal disease activity (MDA). The measurement of the DAPSA score can be considered easy and uncomplicated. The advantages of using the DAPSA score to assess PsA disease activity make DAPSA widely used in observational studies and clinical trials [27, 36].

DAPSA score >28 indicates severe disease activity, a DAPSA score of 15-28 indicates moderate disease activity, a DAPSA score of 5-14 represents low disease activity (DAPSA-Low Disease Activity or DAPSA-LDA), a DAPSA score of 0-4 represents remission (DAPSA-Remission or DAPSA-REM) [43]. The DAPSA also has limitations, namely the lack of definition of disease activity and response criteria in its use in clinical trials and practice to date. These limitations have been minimized by lowering the criteria for PsA disease activity and treatment response [38].

2.3.1. Tender Joint Counts (TJC68)

Tender joint counts (TJC68) are performed by palpating the joint to determine the presence or absence of tenderness in the joint, which indicates that there is synovitis and inflammation in the joint. TJC68 has a score of 0–68. The joints involved in the TJC68 examination are 2 temporomandibular joints, 2 sternoclavicular joints, 2 acromioclavicular joints, 2 shoulder joints, 2 elbow joints, 2 wrist joints, 8 distal interphalangeal (DIP) joints, 20 proximal interphalangeal (PIP) joints, 10 metacarpophalangeal (MCP) joints, 10 metatarsophalangeal (MTP) joints, 2 knee joints, 2 tarsal joints, 2 ankle joints, and 2 hip joints [11, 41].

2.3.2. Swollen Joint Counts (SJC66)

Swollen joint counts (SJC66) are performed by palpating the joint to determine the presence or absence of swelling which indicates there is active synovitis and inflammation in the joint [23, 25]. The swelling that occurs is also related to soft tissue swelling in the joint. The SJC66 has a score of 0–66 and the calculation does not involve the hip joint [9].

2.3.3. Patient Global Assessment (PtGA)

Patient global assessment (PtGA) is an examination to determine the patient's assessment of disease activity using the Visual Analog Scale (VAS). The VAS is used to assess the patient's general health and the impact of PsA on the patient. PtGA has a score of 0–10 and includes patient consideration in the involvement of musculoskeletal, skin, and nail manifestation in PsA disease using VAS. This examination is subjective [22, 25].

2.3.4. Patient Pain (PP)

Patient pain (PP) is an assessment of pain by patient assessment using the Visual Analog Scale (VAS). It is used to determine the patient's pain intensity at present, in the last 24 hours, or in the last 7 days. The PP examination has a score of 0–10 and is subjective. Pain examination with VAS can measure and assess the presence of pain related to PsA disease or not [24, 31].

2.3.5. C-Reactive Protein (CRP)

C-reactive protein (CRP) or acute phase protein is a serum biomarker in the diagnosis and prognosis of PsA. CRP is one of the recommended laboratory tests in determining PsA disease activity. Elevated CRP is found in some PsA patients. High PsA levels have predictive value for a worse prognosis [21]. CRP examination on the DAPSA score can be measured in mg/dL. CRP examination can see signs of inflammation in PsA patients whose levels increase by 2-3 times the normal level [13]

3. Conclusion

In conclusion, paper highlights and emphasizes the characteristic of psoriatic arthritis patient with disease activity based on DAPSA score to evaluate the disease activity with the aim of assessing clinical manifestation and minimizing irreversible damage to joints, skin, and other manifestations. Therefore, further research is needed related to the

application of DAPSA score to assess disease activity at clinical practice by collecting more complete data on psoriatic arthritis patients.

Compliance with ethical standards

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Disclosure of conflict of interest

No conflict of interest to be disclosed.

References

[1] Aletaha D, Smolen JS. Diagnosis and Management of Rheumatoid Arthritis. JAMA. 2018 Oct 2;320(13):1360–72.

[2] Aletaha D, Alasti F, Smolen J. Disease activity states of the DAPSA, a psoriatic arthritis specific instrument, are valid against functional status and structural progression. Annals of the Rheumatic Diseases. 2016 Jul 25;76(2):418–21.

[3] Barnas JL, Ritchlin CT. Etiology and Pathogenesis of Psoriatic Arthritis. Rheumatic Disease Clinics of North America [Internet]. 2015 Nov;41(4):643–63. Available from: https://www.rheumatic.theclinics.com/article/S0889-857X(15)00055-1/abstract

[4] Coates LC, Nash P, Kvien TK, Gossec L, Mease PJ, Rasouliyan L, et al. Comparison of remission and low disease activity states with DAPSA, MDA and VLDA in a clinical trial setting in psoriatic arthritis patients: 2-year results from the FUTURE 2 study. Seminars in Arthritis and Rheumatism. 2020 Aug;50(4):709–18.

[5] Cruz LV, Farani JB, Costa JR, Victor, Ruschel B, de F, et al. Patients with longstanding psoriatic arthritis can achieve DAPSA remission or low disease activity and it correlates to better functional outcomes: results from a Latin-American real-life cohort. Advances in rheumatology. 2024 Jan 2;64(1).

[6] Cruz-Correa OF, Pollock RA, Machhar R, Gladman DD. Prediction of Psoriatic Arthritis in Patients With Psoriasis Using DNA Methylation Profiles. Arthritis & Rheumatology. 2023 Nov 10;75(12):2178–84.

[7] Datta D, Podder I, De A, Das S. Psoriatic Arthritis: A Comprehensive Update for Dermatologists with Review of Literature. Indian Journal of Dermatology [Internet]. 2022 [cited 2023 Oct 2];67(4):381–6. Available from: https://pubmed.ncbi.nlm.nih.gov/36578730/

[8] Davidson A, Diamond B. Autoimmune Diseases. Mackay IR, Rosen FS, editors. New England Journal of Medicine. 2001 Aug 2;345(5):340–50.

[9] Duarte-García A, Leung YY, Coates LC, Beaton D, Christensen R, Craig ET, et al. Endorsement of the 66/68 Joint Count for the Measurement of Musculoskeletal Disease Activity: OMERACT 2018 Psoriatic Arthritis Workshop Report. The Journal of Rheumatology [Internet]. 2019 Aug 1;46(8):996–1005. Available from: https://www.jrheum.org/content/46/8/996

[10] Ehrenfeld M. Spondyloarthropathies. Best Practice & Research Clinical Rheumatology [Internet]. 2012 Feb;26(1):135–45. Available from: https://www.sciencedirect.com/science/article/pii/S1521694212000034

[11] Englbrecht M, Wang Y, Ronneberger M, Manger B, Vastesaeger N, Veale DJ, et al. Measuring joint involvement in polyarticular psoriatic arthritis: An introduction of alternatives. Arthritis Care & Research. 2010 Feb 26;62(7):977–83.

[12] Fearon U, Veale DJ. Pathogenesis of psoriatic arthritis. Clinical and Experimental Dermatology. 2001 Jun;26(4):333–7.

[13] FitzGerald O, Elmamoun M. Psoriatic arthritis. Kelley & Firestein's Textbook of Rheumatology. 2017;11th ed:1285–308.

[14] FitzGerald O, Ogdie A, Chandran V, Coates LC, Kavanaugh A, Tillett W, et al. Psoriatic arthritis. Nature Reviews Disease Primers [Internet]. 2021 Aug 12;7(1):59. Available from: https://pubmed.ncbi.nlm.nih.gov/34385474/

[15] Gossec L, McGonagle D, Korotaeva T, Lubrano E, de Miguel E, Østergaard M, et al. Minimal Disease Activity as a Treatment Target in Psoriatic Arthritis: A Review of the Literature. The Journal of Rheumatology. 2017 Nov 15;45(1):6–13.

[16] Harden JL, Krueger JG, Bowcock AM. The immunogenetics of Psoriasis: A comprehensive review. Journal of Autoimmunity. 2015 Nov; 64:66–73.

[17] Haroon M, FitzGerald O. Psoriatic arthritis: complexities, comorbidities and implications for the clinic. Expert Review of Clinical Immunology. 2016 Jan 28;12(4):405–16.

[18] Hayter SM, Cook MC. Updated assessment of the prevalence, spectrum and case definition of autoimmune disease. Autoimmunity Reviews [Internet]. 2012 Aug 1;11(10):754–65. Available from: https://www.sciencedirect.com/science/article/abs/pii/S1568997212000225?via%3Dihub

[19] Helliwell PS, Kavanaugh A. Comparison of Composite Measures of Disease Activity in Psoriatic Arthritis Using Data From an Interventional Study With Golimumab. Arthritis Care & Research. 2014 Apr 22;66(5):749–56.

[20] Helliwell PS, FitzGerald O, Mease PJ. Development of Composite Measures for Psoriatic Arthritis: A Report from the GRAPPA 2010 Annual Meeting. The Journal of Rheumatology. 2012 Feb;39(2):398–403.

[21] Houttekiet C, de Vlam K, Neerinckx B, Lories R. Systematic review of the use of CRP in clinical trials for psoriatic arthritis: a concern for clinical practice? RMD Open. 2022 Feb;8(1):e001756.

[22] Kuchekar AB, Pujari RR, Kuchekar SB, Dhole SN, Mule PM. Psoriasis: A comprehensive review. International Journal of Pharmacy & Life Sciences. 2011 Jun;2(6):857–77.

[23] Lee S, Mendelsohn A, Sarnes E. The burden of psoriatic arthritis: a literature review from a global health systems perspective. P & T: A Peer-Reviewed Journal for Formulary Management [Internet]. 2010 Dec 1;35(12):680–9. Available from: https://pubmed.ncbi.nlm.nih.gov/21197266/

[24] Littlewood S, MacPhie E. Evaluation of a Psoriatic Arthritis Response Criteria Standardization Training Session for Clinicans. Rheumatology. 2014;53(1):447–553.

[25] Mease PJ. Measures of psoriatic arthritis: Tender and Swollen Joint Assessment, Psoriasis Area and Severity Index (PASI), Nail Psoriasis Severity Index (NAPSI), Modified Nail Psoriasis Severity Index (mNAPSI), Mander/Newcastle Enthesitis Index (MEI), Leeds Enthesit. Arthritis Care & Research. 2011 Nov;63(S11):S64–85.

[26] Mease PJ, Gottlieb AB, Ogdie A, McInnes IB, Chakravarty SD, Emmanouil Rampakakis, et al. Earlier clinical response predicts low rates of radiographic progression in biologic-naïve patients with active psoriatic arthritis receiving guselkumab treatment. Clinical rheumatology. 2023 Oct 3;

[27] Ocampo D V, Gladman D. Psoriatic arthritis. F1000Research. 2019 Sep 20; 8:1665.

[28] Ogdie A, Haynes K, Troxel AB, Love TJ, Hennessy S, Choi H, et al. Risk of mortality in patients with psoriatic arthritis, rheumatoid arthritis and psoriasis: a longitudinal cohort study. Annals of the Rheumatic Diseases [Internet]. 2012 Dec 21 [cited 2019 Aug 4];73(1):149–53. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3883139/

[29] Pfeil A, Heinz M, Hoffmann T, Weise T, Renz DM, Franz M, et al. The relationship between structural analysis of the hand and clinical characteristics in psoriatic arthritis. Scientific Reports. 2022 Nov 7;12(1).

[30] Proft F, Käding H. Same, same or different? Commonalities and differences between spondyloarthritis and its subsets of axial and peripheral spondyloarthritis with psoriatic arthritis and its diverse phenotypes. RMD Open. 2023 Apr 1;9(2):e002872–2.

[31] Pukšić S, Bolton-King P, Sexton J, Michelsen B, Kvien TK, Berner Hammer H. DAPSA and ultrasound show different perspectives of psoriatic arthritis disease activity: results from a 12-month longitudinal observational study in patients starting treatment with biological disease-modifying antirheumatic drugs. RMD Open [Internet]. 2018 Nov [cited 2019 Dec 3];4(2):e000765. Available from: https://rmdopen.bmj.com/content/4/2/e000765

[32] Punzi L, Pianon M, Bertazzolo N, Fagiolo U, Rizzi E, Rossini P, et al. Clinical, laboratory and immunogenetic aspects of post-traumatic psoriatic arthritis: a study of 25 patients. Clinical and experimental rheumatology [Internet]. 1998;16(3):277–81. Available from: https://pubmed.ncbi.nlm.nih.gov/9631749/

[33] Rasyid MFA, Anggraini DI, Wardhana MF, Hamzah MS. Psoriasis Arthritis. Jurnal Agromedicine [Internet]. 2021 Dec 2;8(1). Available from: https://juke.kedokteran.unila.ac.id/index.php/agro/article/view/2982

[34] Rendon A, Schäkel K. Psoriasis Pathogenesis and Treatment. International Journal of Molecular Sciences. 2019 Mar 23;20(6):1475.

[35] Ritchlin CT, Colbert RA, Gladman DD. Psoriatic Arthritis. The New England journal of medicine [Internet]. 2017;376(10):957–70. Available from: https://www.ncbi.nlm.nih.gov/pubmed/28273019

[36] Salaffi F, Ciapetti A, Carotti M, Gasparini S, Gutierrez M. Disease Activity in Psoriatic Arthritis: Comparison of the Discriminative Capacity and Construct Validity of Six Composite Indices in a Real World. BioMed Research International. 2014; 2014:1–12.

[37] Schoels M, Aletaha D, Funovits J, Kavanaugh A, Baker D, Smolen JS. Application of the DAREA/DAPSA score for assessment of disease activity in psoriatic arthritis. Annals of the Rheumatic Diseases. 2010 Jun 4;69(8):1441–7.

[38] Schoels MM, Aletaha D, Alasti F, Smolen JS. Disease activity in psoriatic arthritis (PsA): defining remission and treatment success using the DAPSA score. Annals of the Rheumatic Diseases. 2015 Aug 12;75(5):811–8.

[39] Smolen JS, Schoels M, Aletaha D. Disease activity and response assessment in psoriatic arthritis using the Disease Activity index for PSoriatic Arthritis (DAPSA). A brief review. Clinical and experimental rheumatology [Internet]. 2015;33(5 Suppl 93):S48-50. Available from: https://pubmed.ncbi.nlm.nih.gov/26471734/

[40] Veale D, FitzGerald O. Psoriatic arthritis. Best practice & research Clinical rheumatology [Internet]. 2002 Sep;16(4):523–35. Available from: https://pubmed.ncbi.nlm.nih.gov/12406425

[41] Wang Y, Xiao Y, Li F, Gu Y, Yang M, Zhang L, et al. The Clinical Characteristics of Psoriatic Arthritis: A Cross-Sectional Study Based on the Psoriatic Arthritis Cohort of West China Hospital. Rheumatology and therapy [Internet]. 2023 Feb 16;10(3):775–84. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC10140229/

[42] Wei JCC, Shi LH, Huang JY, Wu XF, Wu R, Chiou JY. Epidemiology and Medication Pattern Change of Psoriatic Diseases in Taiwan from 2000 to 2013: A Nationwide, Population-based Cohort Study. The Journal of Rheumatology. 2018 Jan 15;45(3):385–92.

[43] Wervers K, Vis M, Tchetveriko I, Gerards AH, Kok MR, Appels CWY, et al. Burden of Psoriatic Arthritis According to Different Definitions of Disease Activity: Comparing Minimal Disease Activity and the Disease Activity Index for Psoriatic Arthritis. Arthritis Care & Research. 2018 Nov 28;70(12):1764–70.