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THE HISTORY AND EVOLUTION OF PSORIATIC ARTHRITIS

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Abstract

Psoriatic arthritis (PsA) is a chronic inflammatory disorder characterised by a heterogeneous clinical presentation involving both the skin and joints. Although the coexistence of psoriasis and arthritis has been recognised since antiquity, PsA remained largely unacknowledged as a distinct disease entity until the mid-20th century. Early descriptions in historical literary sources and archaeological findings provided indirect evidence of PsA, but the conceptualisation of the disease evolved significantly in the 19th and 20th centuries. The introduction of classification criteria by Moll and Wright in 1973 and subsequent developments such as the CASPAR criteria in 2006 established a framework for diagnosis and research. Advances in the understanding of PsA pathogenesis, particularly the discovery of the tumor necrosis factor-α and interleukin (IL)-23/IL-17 pathways, paved the way for biologic therapies and treat-to-target strategies, which have dramatically improved clinical outcomes. This narrative review summarises the key historical milestones and therapeutic breakthroughs that have shaped modern PsA management.

Keywords: Psoriatic arthritis, historical review, evolution of classification criteria, history of therapeutic approaches

INTRODUCTION

Psoriatic arthritis (PsA) is an inflammatory disease characterised by heterogeneous clinical features involving both the skin and joints. Historically, despite early descriptions of psoriatic lesions—including Galen's introduction of the term "psoriasis (PsO)" in the second century *anno Domini*—PsO was frequently misclassified as leprosy until the 19th century, and its association with arthritis remained insufficiently understood. Although the coexistence of PsO and arthritis has been recognised since antiquity, PsA was not formally acknowledged as a distinct clinical entity until the mid-20th century. With advances in the understanding of disease pathogenesis, numerous biological agents have been introduced for the treatment of PsO and PsA since the early 2000s. These therapeutic innovations, together with improved clinical outcomes, have substantially enhanced the awareness and recognition of both conditions (1,2).

The Historical Journey of Psoriasis

In ancient Egypt, as early as 2000 before common era (BCE), therapeutic preparations for skin conditions resembling PsO reportedly included mixtures of goose fat, cat and dog excrement, sea salt, and urine. Goose fat, much like olive oil, was thought to moisturise the skin and alleviate symptoms such as pruritus. Hippocrates (460-377 BCE) described dry, scaly skin lesions under the term "lopoi". Due to similarities in clinical appearance, PsO was frequently misdiagnosed as leprosy in antiquity. The term "psora" or "PsO" was first introduced in the 1st century BCE in

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the works of Pedanius Dioscorides, derived from the Greek verb "ψάω" (to scratch), referring to pruritic skin lesions. Later, Galen of Pergamon (129-216 common era) employed the term "psora" to denote scaly dermatoses. During the Middle Ages, PsO patients were often misclassified as lepers and subjected to harsh measures, including social isolation, execution, and even burning (1-3).

By the 1800s, Robert Willan had classified cutaneous lesions into eight categories, with those in the second category corresponding to PsO. The Austrian dermatologist Ferdinand Hebra, in the 1840s, developed an atlas of skin diseases that remained influential for many years. Hebra was the first to clearly distinguish leprosy from the PsO and is widely regarded as the father of dermatology (1,2).

For many years, PsO was treated primarily with coal tar preparations. In the 1950s, topical corticosteroids and methotrexate were introduced as therapeutic options, followed by the advent of phototherapy in the 1960s. The 1970s witnessed the introduction of cyclosporine for PsO treatment, which represented another important milestone in therapeutic approaches (1,2).

In the 1990s, advances in the understanding of the molecular and cellular pathogenesis of autoimmune inflammatory diseases laid the groundwork for the era of biologic therapies. The first biologic agents, alefacept and efalizumab, targeted co-stimulatory signals involved in T-lymphocyte activation. However, their efficacy was limited, and their use was discontinued due to an increased risk of serious infections. Subsequently, the introduction of tumor necrosis factor inhibitors (TNFi), particularly etanercept and infliximab, revolutionised the treatment of PsA. TNFi agents became the gold standard owing to their ability to prevent joint damage. Later, the development of interleukin (IL)-12/23 inhibitors (ustekinumab), IL-17 inhibitors (secukinumab, ixekizumab), and IL-23 inhibitors (guselkumab), marked a significant turning point in PsA management (4).

Archaeological Evidence of Psoriatic Arthritis

In 1981, Rogers et al. (5) reported radiological findings consistent with PsA in Saxon skeletons dated to the 13th century, excavated in England. The presence of a "pencil-in-cup" appearance and spinal syndesmophytes led the authors to conclude that these individuals likely had PsA These cases may represent the earliest known patients with PsA. Retrospectively identifying observations of PsA prior to the 19th century is highly challenging and, in most cases, impossible. However, there are a few notable exceptions. In 1992, Ronald A. Bloom and Patricia Smith described a skeleton dated approximately 2,000 years ago from the Ein Gedi necropolis in Israel and suggested PsA (or arthritis secondary to inflammatory bowel disease) as a possible diagnosis. Moreover, in 1996, skeletal remains dated to the 5th-6th centuries, discovered near the Martyrius Monastery in Jerusalem, were consistent with arthritis mutilans, the most destructive form of PsA (3). In recent years, advanced methodologies such as paleogenetics and computed tomography (CT)/micro-CT re-evaluation of skeletal remains have been increasingly utilized to differentiate PsA from other forms of arthritis in antiquity, thereby strengthening the validity of paleopathological interpretations (6,7).

Psoriatic Arthritis in Historical Literary Sources

In 1674, Fray Felipe Colombo documented the life of Fray Pedro de Urraca, a Mercedarian monk who had served in Peru (8). The report noted a diagnosis of gouty arthritis at the age of 29, accompanied by cutaneous lesions resembling leprosy and progressive deformities affecting the small joints of the hands, as well as, the knees and shoulders. This description is regarded as one of the earliest literary accounts suggestive of PsA.

The Recognition of the Psoriasis-Arthritis Association

In their 1813 book A Practical Synopsis of Cutaneous Diseases, Robert Willan and Thomas Bateman noted the presence of arthritis in patients with PsO. Subsequently, in 1818, Jean Louis Marc Alibert described a case of PsA in his writings, representing one of the earliest documented accounts of the condition (9).

The Origin of the Term Psoriatic Arthritis

In 1860, Pierre Bazin introduced the term "PsO arthritique" (10). Thus, Bazin is considered the originator of the concept and nomenclature of PsA. Later, in 1888, Charles Bourdillon used the term "PsO et arthropathies" in his writings.

In the past, arthritis associated with PsO was known by various terms, such as rheumatisme psoriasique; PsO arthropathique; PsO arthritique; polyarthrite psoriasique; arthropatia psoriatica; arthropathia psoriatica; PsO arthropathica; PsO arthritica; arthritis psoriatica; polyarthritis psoriatica; artrite psoriasica; psoriatischen arthropathie; PsO-arthritis; and artropatia łuszczycowa (11). Pierre Bazin classified all dermatological diseases pragmatically into two groups—"arthritic" and "herpetic"—rather than adopting a conceptual approach. In this context, his definition of PsO arthritica does not fully correspond to the current concept of PsA but instead refers to a specific subset of PsO (3). Today, the most widely used terms are PsA and psoriatic arthropathy.

In their 1937 publication, Jeghers and Robinson (12) used the term PsA for the first time. Later, in 1951, Vilanova and Piñol

(13) emphasised that PsA represented a distinct entity from other inflammatory arthropathies. The discovery of rheumatoid factor in 1940 (14) undeniably contributed to this conclusion.

In 1952, the American orthopaedic surgeon Mary Stults Sherman published one of the earliest comprehensive studies on PsA in the English-language literature. Sherman highlighted distinctive features such as distal interphalangeal (DIP) joint involvement, arthritis mutilans, and axial disease, emphasizing that PsA is a unique clinical entity. She also evaluated the effects of ACTH and cortisone on both cutaneous and articular manifestations but noted that these therapies provided only transient benefits (15).

With the publication of Wright's (16) 1956 paper, PsA became more widely recognised and was accepted as a distinct disease entity. Wright subsequently published two additional articles in 1959 in which he compared clinical data from patients with rheumatoid arthritis, PsO, and PsA (17,18). Following these developments, the American Rheumatism Association [now the American College of Rheumatology (ACR)] officially recognized PsA as a separate entity in 1964 (19).

During the 1960s, the overlapping clinical features among various seronegative arthritides, including PsA, and their strong association with spondylitis were recognised. Moll and colleagues subsequently proposed the term 'seronegative spondyloarthritides' to describe this group of disorders (11). The identification of the association between *HLA-B27* and ankylosing spondylitis in 1973, followed by its extension to other spondylitis-associated diseases, such as ulcerative colitis and PsO, provided further evidence supporting the concept of these conditions as a related family of disorders (11).

By the late 20th century, the misconception of PsA as a "benign disease" had been refuted, and the existence of severe forms capable of causing deformity and disability was recognised. Longitudinal studies conducted at the Toronto PsA Clinic underscored the importance of early and aggressive treatment, laying the foundation for modern therapeutic paradigms (20).

Early studies delineated the subtypes of PsA, including asymmetric oligoarthritis, polyarticular arthritis, and spondyloarthritis. DIP joint involvement and periostitis were also highlighted as distinguishing features of PsA. In the 1980s, *HLA-B27* was identified as being associated with axial disease, while *HLA-B38* and *HLA-B39* were linked to peripheral polyarthritis, contributing significantly to the understanding of the phenotypic heterogeneity of the disease (20).

The Development of PsA Classification Criteria

In their landmark 1973 publication, Wright—regarded as the father of spondyloarthritis—and Moll described the clinical subtypes of PsA and proposed the first set of classification criteria (21). According to the Moll and Wright (21) criteria (Table 1), patients meeting three specified criteria can be classified as having PsA. Later, in 1984, Vasey and Espinoza (22), who authored the PsA section in a textbook on spondyloarthritis, introduced an alternative classification system. Although similar to the Moll and Wright (21) criteria, this system provided a more refined definition of peripheral and axial patterns (Table 2).

In 1991, the European Spondyloarthropathy Study Group proposed a set of criteria (23) that included the presence of synovitis or inflammatory back pain in combination with PsO or a history of PsO. The most recent classification system, developed by the Classification Criteria for Psoriatic Arthritis (CASPAR) study group, was published in 2006 (24). Notably, the CASPAR criteria allow for the classification of patients as having PsA even in the absence of PsO (Table 3). Recent revisions have further improved the sensitivity of PsA classification criteria.

Group for Research and Assessment of Psoriasis and Psoriatic Arthritis

The first large-scale collaborative group established to address PsA was the CASPAR group. The contributions of CASPAR members, who are predominantly comprised of European rheumatologists, to the development of the classification criteria published in 2006 cannot be overstated. During the group's 2001 meeting, it was decided to include PsA experts from outside Europe, as well as specialists from non-rheumatology disciplines, in recognition of the multisystemic nature of the disease. Consequently, at a meeting held in 2003, the expanded group adopted the name Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA). Today, GRAPPA is a highly influential organisation that collaborates with institutions such as the ACR, Assessment of SpondyloArthritis International Society, European Alliance of Associations for Rheumatology (EULAR), and Outcome Measures in Rheumatology to guide decisions on the diagnosis, monitoring, and treatment of PsA (25).

The discovery of the TNF-α and IL-23/IL-17 axes has deepened the understanding of PsA pathogenesis and paved the way for the development of biologic agents. TNF inhibitors such as etanercept, infliximab, and adalimumab, along with next-generation biologics such as ustekinumab, secukinumab, and ixekizumab, have established the foundation for a targeted treatment paradigm in PsA. These advances have markedly altered the natural course of PsA and facilitated the widespread adoption of treat-to-target strategies (26). In the past decade, targeted oral therapies have further expanded the therapeutic armamentarium. Janus kinase (JAK) inhibitors such as tofacitinib

Table 1. Moll and Wright (21) criteria for PsA classification

1. Inflammatory joint disease (arthritis, spondylitis, or sacroiliitis), and
2. Psoriasis, and
3. Absence of rheumatoid factor

PsA: Psoriatic arthritis

Table 2. Vasey and Espinoza (22) criteria for PsA classification (1984)

1. Psoriasis or psoriatic nail lesions, and
2. Peripheral pattern* or axial pattern**

*More than 4 weeks of arthritis of DIPJ; or asymmetrical peripheral arthritis (included sausage digit); absent RF or rheumatoid nodule; or radiographic changes (pencil-in-cup deformity, whittling of terminal phalanges, fluffy periostitis and bony ankylosis).
**More than 4 weeks of spinal pain and stiffness with the restriction of motion, or grade 2 symmetric sacroiliitis or grade 3 or 4 unilateral sacroiliitis according to the New York criteria.
PsA: Psoriatic arthritis, DIPJ: Distal interphalangeal joint, RF: Rheumatoid factor

Table 3. CASPAR criteria for the classification of PsA (24)

A patient with arthritis, spondylitis, sacroiliitis, or enthesitis is classified as diagnosed with PsA if the patient score based on the following parameters

Parameters	Points
Current psoriasis	**2**
Personal or family history of psoriasis	**1**
Nail dystrophy on physical examination	**1**
Negative rheumatoid factor	**1**
Dactylitis, current or history of dactylitis (confirmed by a rheumatologist)	**1**
Radiographic evidence of juxta-articular new bone formation	**1**

CASPAR: Classification Criteria for Psoriatic Arthritis, PsA: Psoriatic arthritis

and upadacitinib are now approved for PsA, and incorporated into treatment recommendations, while the selective tyrosine kinase 2 inhibitor deucravacitinib, currently approved for plaque PsO, is under evaluation for PsA. The most recent EULAR 2024 recommendations and the GRAPPA 2022 update endorse a domain-based, treat-to-target approach, emphasizing early initiation of conventional synthetic disease-modifying anti-rheumatic drugs (DMARDs) (preferably methotrexate) for peripheral arthritis, avoidance of chronic oral glucocorticoids, and escalation to biologic DMARDs or targeted synthetic DMARDs—including JAK inhibitors—according to clinical domains and comorbidities (27,28).

CONCLUSION

The historical trajectory of PsA spans from early misclassifications and literary descriptions to its recognition as a distinct clinical entity with validated classification criteria. Advances in immunopathogenesis have shifted treatment from traditional approaches to biologics and targeted synthetic agents, shaping today's treat-to-target paradigm. Continued progress in molecular phenotyping and precision medicine is expected to further refine patient stratification and optimise future management strategies.

Footnotes

Authorship Contributions

Concept: B.Ö., İ.G., Design: B.Ö., İ.G., Data Collection or Processing: İ.G., Literature Search: B.Ö., İ.G., Writing: B.Ö.

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REFERENCES

1. Burg G, Geiges M. Lepra vulgaris. History of psoriasis. J Turk Acad Dermatol. 2014;8:1483r1.
2. Reid C, Griffiths CE. Psoriasis and treatment: past, present and future aspects. Acta Derm Venereol. 2020;100:69-79.

3. Pasero G, Marson P. The antiquity of psoriatic arthritis. Clin Exp Rheumatol. 2006;24:351.

4. Mease P. A short history of biological therapy for psoriatic arthritis. Clin Exp Rheumatol. 2015;33(Suppl 93):S104-8.

5. Rogers J, Watt I, Dieppe P. Medical history: arthritis in Saxon and mediaeval skeletons. Br Med J (Clin Res Ed). 1981;283:1668-70.

6. Zias J, Mitchinson MJ, Xipell JM, Epstein L, Tamarin A. Psoriatic arthritis in a fifth-century Judean Desert monastery. Clin Rheumatol. 1996;15:616-9.

7. Poma A, Lombardi G, Fornaciari A, et al. Paleopathology and immunogenetics of Cardinal Carlo de' Medici (1595-1666): a multidisciplinary study. Rheumatology (Oxford). 2013;52:691-5.

8. Colombo F. El Job de la ley de gracia retratado en la admirable vida del siervo de Dios venerable padre Fray Pedro Urraca. Imprenta de DP Marín; 1790.

9. Alibert JL. Précis théorique et pratique sur les maladies de la peau. Caille et Ravier; 1822.

10. Bazin E. Leçons sur les affections cutanées de nature arthritique et dartreuses. Paris: Delahaye. 1860:311.

11. O'Neill T, Silman AJ. Historical background and epidemiology. Baillieres Clin Rheumatol. 1994;8:245-61.

12. Jeghers H, Robinson LJ. Arthropathia psoriatica: report of case and discussion of the pathogenesis, diagnosis and treatment. JAMA. 1937;108:949-52.

13. Vilanova X, Pinol J. Psoriasis arthropathica. Rheumatism. 1951;7:197-208.

14. Waaler E. On the occurrence of a factor in human serum activating the specific agglutination of sheep blood corpuscles. Acta Pathol Microbiol Scand. 1940;17:172-88.

15. Espinoza LR, Helliwell P. Psoriatic arthritis: Mary Stults Sherman, a forgotten figure in its history. Clin Rheumatol. 2014;33:1335-6.

16. Wright V. Psoriasis and arthritis. Ann Rheum Dis. 1956;15:348.

17. Wright V. Rheumatism and psoriasis: a re-evaluation. Am J Med. 1959;27:454-62.

18. Wright V. Psoriatic arthritis: a comparative study of rheumatoid arthritis, psoriasis, and arthritis associated with psoriasis. AMA Arch Dermatol. 1959;80:27-35.

19. Blumberg BS, Bunim JJ, Calmns E, Pirani CL, Zvaifler NJ. ARA nomenclature and classification of arthritis and rheumatism (tentative). Arthritis Rheum. 1964;7:93-7.

20. Gladman DD. Natural history of psoriatic arthritis. Baillieres Clin Rheumatol. 1994;8:379-94.

21. Moll JM, Wright V. Psoriatic arthritis. Semin Arthritis Rheum. 1973;3:55-78.

22. Vasey FB, Espinoza LR. Psoriatic arthritis and infection. In: Infections and the rheumatic diseases. Cham: Springer International Publishing; 2019. p. 393-7.

23. Dougados M, van der Linden S, Juhlin R, et al. The European Spondylarthropathy Study Group preliminary criteria for the classification of spondylarthropathy. Arthritis Rheum. 1991;34:1218-27.

24. Taylor W, Gladman D, Helliwell P, Marchesoni A, Mease P, Mielants H. Classification criteria for psoriatic arthritis: development of new criteria from a large international study. Arthritis Rheum. 2006;54:2665-73.

25. Gladman DD, Mease PJ, Helliwell PH. GRAPPA historical perspective. In: Psoriatic arthritis and psoriasis: pathology and clinical aspects. New York: Springer; 2016. p. 7-14.

26. Espinoza LR. The history of psoriatic arthritis (PsA): from Moll and Wright to pathway-specific therapy. Curr Rheumatol Rep. 2018;20:58.

27. 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;83:706-19.

28. 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;18:465-79.