Scientific Research Publishing

Case Reports in Clinical Medicine, 2026, 15(11), 477-483
https://www.scirp.org/journal/crcm
ISSN Online: 2325-7083
ISSN Print: 2325-7075

A Case of Occult Psoriatic Arthritis: Diagnosis and Management

Kamel El-Reshaid[1*], Shaima Al-Bader[2], Abdulla Al-Refaee[3]

[1]Department of Medicine, Faculty of Medicine, Kuwait University, Kuwait City, Kuwait
[2]Department of Dermatology, Asad Al-Hamad Centre, Kuwait City, Kuwait
[3]Department of Dermatology, Amiri Hospital, Kuwait City, Kuwait
Email: *Kamel@hsc.edu.kw

How to cite this paper: El-Reshaid, K., Al-Bader, S. and Al-Refaee, A. (2026) A Case of Occult Psoriatic Arthritis: Diagnosis and Management. *Case Reports in Clinical Medicine*, **15**, 477-483.
https://doi.org/10.4236/crcm.2026.1511060

Received: September 28, 2026
Accepted: November 6, 2026
Published: November 9, 2026

Copyright © 2026 by author(s) and Scientific Research Publishing Inc.
This work is licensed under the Creative Commons Attribution International License (CC BY 4.0).
http://creativecommons.org/licenses/by/4.0/

Open Access

Abstract

Background: Acute arthritis (<6 weeks): trauma, infections (viral and bacterial), crystals (gout), reactive arthritis, and as the early phase of chronic ones, viz. osteoarthritis, psoriasis (P), chronic infections, and autoimmune diseases (rheumatic, rheumatoid, lupus, and vasculitis). **Case:** A 32-year-old white male presented with severe arthritis in the 2nd to 5th toes in both feet for 2 weeks. He had a negative history and physical examination, as well as laboratory and immunological tests for autoimmune diseases, gout, and infections. On examination, he had enthesitis and dactylitis. Diagnosis was suspected by typical nail findings of P and confirmed by detecting occult scalp lesions that was confirmed by dermatoscopy. Other etiologies of arthritis were excluded by laboratory, serological, bacteriological, and radiological tests. His arthritis and skin lesions improved within 2 weeks after treatment with Cyclosporine A (CsA) 100 mg twice daily. After 3 months, the CsA dose was reduced to 50 mg twice daily and was maintained for 2 years. Subsequent to this therapy, new skin lesions did not evolve, and arthritis did not recur after 2 more years of follow-up. **Conclusion:** Occult P can manifest as arthropathy. Nail changes and small skin/scalp lesions may aid in diagnosis, and the disease is amenable to CsA therapy.

Keywords

Arthritis, Cyclosporine A, Dermatoscopy, Psoriasis, Therapy

1. Introduction

Arthritis is defined as an acute or chronic inflammation in the joints that includes pain, swelling, limitation of movement, and joint deformities. Nearly 100 types of arthritis exist, with a prevalence of 21%, and it is the leading cause of disability in

the USA [1]. If misdiagnosed or poorly managed, it can result in severe pain, anxiety, activity limitations, disability, and medical costs [2]. Hence, definitive diagnosis is essential to avoid complications and adverse effects of its drug therapy, viz. non-steroidal anti-inflammatory drugs, corticosteroids, disease-modifying agents, biological agents, and JAK inhibitors [3]. Acute arthritis (<6 weeks): trauma, infections (viral and bacterial), crystals (gout), reactive arthritis, and as the early phase of chronic ones viz. osteoarthritis, psoriasis (P), chronic infections and autoimmune diseases (rheumatic, rheumatoid, lupus and vasculitis) [4]. P is a common chronic skin disease characterized by itchy red patches with large silvery-white scales on top [5]. Diagnosis is established clinically, dermatoscopically, and can be confirmed by skin biopsy [6]. Nail changes are common in P and have a lifetime incidence of 80% - 90% [7]. On the other hand, arthritis was detected in 30% of patients with P skin disease [8]. In this case report, we present our means of diagnosis and management of a patient with acute arthritis due to P, yet without overt skin disease.

2. The Case



Figure 1. Arthritis of 2nd to 5th toes in left (A) and right (B) feet at presentation and after treatment in (C) and (D).

A 32-year-old man presented with severe arthritis in the 2nd to 5th toes in both feet for 2 weeks. He denied previous medical illness, surgery, allergy, chronic intake of medications, and recent trauma to toes. On his initial examination, he was conscious, oriented x3, and without distress of shortness of breath, yet was in distress of pain in his toes. He was normotensive, afebrile, and had a body weight of 77 kg. He had severe bilateral sausage-like swelling of the whole toes (dactylitis) and inflammation of tendons and ligaments at bone attachments (enthesitis), as seen in **Figure 1**. Systemic examination did not show abnormality. Since he had nail changes

suggestive of psoriasis (Figure 2), a detailed search disclosed a few psoriatic scalp lesions that were confirmed by dermatoscopy (Figure 3). Laboratory investigations showed normal peripheral leucocyte and platelet counts. Hemoglobin was normal. Serum glucose, urea, creatinine, electrolytes, uric acid, amylase, liver function tests, cholesterol, and TSH were normal. ESR, CRP, procalcitonin, and angiotensin-converting enzyme levels were normal. Urine routine and microscopy were normal. Serum complements (C3 & C4) were normal. Serum protein electrophoresis, HIV, anti-CCP, ANA, ANCA, and hepatitis B & C serology were negative. Chest X-ray and high-resolution chest CT did not show infiltrate or lymphadenopathy. Fungal nail infection was excluded by microscopic and dermatoscopic examination after potassium hydroxide. Vascular insufficiency was excluded by Doppler ultrasound study. He was treated with 1 g of solumedrol IV for 3 consecutive days to relieve the pain, as well as cyclosporine A 100 mg twice daily. After 3 months, the CsA dose was reduced to 50 mg twice daily and was maintained for 2 years. Subsequent to this therapy, new skin lesions did not evolve, and arthritis did not recur after 2 more years of follow-up.



Figure 2. Nail changes (discoloration, subungual hyperkeratosis and pitting) in the patient's fingers.

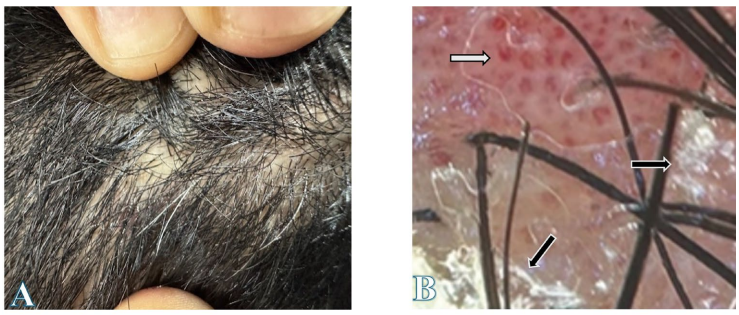


Figure 3. Occult scalp psoriasis (A) and after magnification (dermatoscopy) in (B) showing linear convoluted vessels (white arrow), white areas with scales (black arrows).

3. Discussion

In our patient, diagnosis of P arthritis was established by excluding local trauma, focal infection, vascular insufficiency, as well as systemic diseases, viz. autoimmune

disease (rheumatoid, systemic lupus erythematosus, sarcoidosis and reactive arthritis) by serological and radiological tests. Interestingly, his ESR and CRP were within normal limits, indicating a lack of systemic inflammatory disorder despite its acute flare. The typical nail changes were a useful clinical indicator of P. Hence, a detailed search disclosed occult small scalp lesions characteristic of P. The differential diagnosis of P includes dermatological conditions similar in appearance such as discoid eczema, lichen planus, fungal infections, seborrheic eczema, pityriasis rosa, cutaneous T-cell lymphoma, and secondary syphilis [9]. However, for the scalp, seborrheic dermatitis is the main one. Unlike P, 1) its erythematous borders are sharper and accompanied by perifolliculitis, 2) its scales are fine and greasy, which are different from the silver-white large scales of scalp psoriasis, and dermoscopy is useful [10]. In general, nail changes can disclose many systemic medical diseases, yet pitting is characteristic of P [11]. In advanced P, nail changes; fungal infection should be excluded by microscopic and dermatoscopic examination after potassium hydroxide [12]. P arthritis typically involves painful inflammation of the joints and surrounding connective tissue and can occur in any joint, but most commonly affects the joints of the fingers and toes. This can result in a sausage-shaped swelling of the fingers and toes known as dactylitis and rarely the hips, knees, spine (spondylitis), and sacroiliac joint (sacroiliitis) [13]. Skin manifestations of psoriasis tend to occur before arthritic manifestations in about 75% of cases [8]. With regard to management of psoriasis, it stems from our knowledge of its genetic predisposition with autoimmune triggering factors. Globally, it affects 4.4% of the population worldwide, yet it is 5 times more common in the Western world compared to those of Asian descent [14]. Moreover, its rate and prevalence vary according to age, region, and ethnicity; a combination of environmental and genetic factors is thought to be responsible for these differences [5]. It is a genetic disease that is triggered by environmental factors [15]. Nearly 1/3 of patients have a family history and a 70% chance of developing it in identical twins [16]. Gene mutations in proteins involved in the skin's ability to function as a barrier have been identified as markers of susceptibility for developing psoriasis [17]. In P, skin cells are replaced every 3 - 5 days rather than the usual 28 - 30 days. These changes are believed to stem from the premature maturation of keratinocytes induced by an inflammatory cascade in the dermis involving dendritic cells, macrophages, and T lymphocytes (three subtypes of immune cells) [18]. These immune cells move from the dermis to the epidermis and secrete inflammatory chemical signals (cytokines) such as interleukin-36γ, tumour necrosis factor-α, interleukin-1β, interleukin-6, and interleukin-22. These secreted inflammatory signals are believed to stimulate keratinocytes to proliferate [19]. The inflammatory cytokines found in psoriatic nails and joints (in the case of psoriatic arthritis) are similar to those of psoriatic skin lesions, suggesting a common inflammatory mechanism [20].

Previously, there was no known cure for psoriasis, but various treatments can help control the symptoms. These treatments include corticosteroids, methotrex-

ate, and specific biological agents (adalimumab (Humira), certolizumab (Cimzia), etanercept (Enbrel), and golimumab (Simponi). They also include infliximab (Remicade), ustekinumab (Stelara), secukinumab (Cosentyx), ixekizumab (Taltz), guselkumab (Tremfya), and abatacept (Orencia). However, they can increase the risk of both common and rare infections, such as tuberculosis. Moreover, oral ones, viz. 1) Tofacitinib (Xeljanz) increases the risk of blood clots in the lungs, serious heart issues, and cancer, and 2) Apremilast (Otezla) is associated with diarrhea, nausea, and headaches [21]. Previously, we selected CsA for treatment of severe pustular P and amyloidosis secondary to it [22] [23]. Our management was based on the pathogenesis of P and CsA action via blocking IL-2, which is essential in the cascade of T-cell recruitment, based on [24]. Hence, we used the drug for our patient with P arthritis. It proved to be safe and effective in short- and long-term follow-up. Moreover, we were able to decrease its dose after 3 months and limit its duration to 2 years to avoid nephrotoxicity [25].

4. Conclusion

Occult P can manifest as arthropathy. Nail changes and small skin/scalp lesions may aid in diagnosis, and the disease is amenable to CsA therapy.

Data Sharing Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Informed consent for publication of the case was obtained and is available on request.

Author Contributions

1) Kamel El-Reshaid: Conceptualization, analysis, and writing the final article.

2) Shaima Al-Bader: Case detection, data collection, and initial draft preparation.

3) Abdulla Al-Refaee: Data collection (initial and follow-up) and initial draft preparation.

All authors reviewed the manuscript and approved the final version.

Conflicts of Interest

The authors declare that they have no potential conflict of interest related to the contents of this article.

References

[1] (2023) Arthritis and Rheumatic Diseases. National Institute of Arthritis and Musculoskeletal and Skin Diseases, US National Institutes of Health.

[2] Theis, K.A., Murphy, L.B., Guglielmo, D., Boring, M.A., Okoro, C.A., Duca, L.M., *et al.* (2021) Prevalence of Arthritis and Arthritis-Attributable Activity Limitation—United States, 2016-2018. *MMWR. Morbidity and Mortality Weekly Report*, **70**, 1401-1407. https://doi.org/10.15585/mmwr.mm7040a2

[3] Fraenkel, L., Bathon, J.M., England, B.R., St Clair, E.W., Arayssi, T., *et al.* (2021) American College of Rheumatology Guideline for the Treatment of Rheumatoid Arthritis. *Arthritis Care & Research* (*Hoboken*), **73**, 924-939.

[4] Senthelal, S., Li, J., Ardeshirzadeh, S., *et al.* (2023) Arthritis. StatPearls.

[5] Parisi, R., Symmons, D.P.M., Griffiths, C.E.M. and Ashcroft, D.M. (2013) Global Epidemiology of Psoriasis: A Systematic Review of Incidence and Prevalence. *Journal of Investigative Dermatology*, **133**, 377-385. https://doi.org/10.1038/jid.2012.339

[6] Raychaudhuri, S.K., Maverakis, E. and Raychaudhuri, S.P. (2014) Diagnosis and Classification of Psoriasis. *Autoimmunity Reviews*, **13**, 490-495. https://doi.org/10.1016/j.autrev.2014.01.008

[7] Tan, E.S.T., Chong, W. and Tey, H.L. (2012) Nail Psoriasis. *American Journal of Clinical Dermatology*, **13**, 375-388. https://doi.org/10.2165/11597000-000000000-00000

[8] Goldenstein-Schainberg, C., Favarato, M.H.S. and Ranza, R. (2012) Conceitos atuais e relevantes sobre artrite psoriásica. *Revista Brasileira de Reumatologia*, **52**, 98-106. https://doi.org/10.1590/s0482-50042012000100010

[9] Weller, R.P.J.B., Hunter, J.A.A., Savin, J.A. and Dahl, M.V. (2008) Clinical Dermatology. 4th Edition, Wiley. https://doi.org/10.1002/9781444300086

[10] Kim, G.W., Jung, H.J., Ko, H.C., Kim, M.B., Lee, W.J., Lee, S., *et al.* (2011) Dermoscopy Can Be Useful in Differentiating Scalp Psoriasis from Seborrhoeic Dermatitis. *British Journal of Dermatology*, **164**, 652-656. https://doi.org/10.1111/j.1365-2133.2010.10180.x

[11] Gollins, C.E. and de Berker, D. (2021) Nails in Systemic Disease. *Clinical Medicine*, **21**, 166-169. https://doi.org/10.7861/clinmed.2021-0243

[12] Leung, A.K.C., Lam, J.M., Leong, K.F., Hon, K.L., Barankin, B., Leung, A.A.M., *et al.* (2020) Onychomycosis: An Updated Review. *Recent Patents on Inflammation & Allergy Drug Discovery*, **14**, 32-45. https://doi.org/10.2174/1872213x13666191026090713

[13] Krawczyk-Wasielewska, A., Skorupska, E. and Samborski, W. (2013) Sacroiliac Joint Pain as an Important Element of Psoriatic Arthritis Diagnosis. *Advances in Dermatology and Allergology*, **2**, 108-112. https://doi.org/10.5114/pdia.2013.34161

[14] Skayem, C., Taieb, C., Halioua, B., Baissac, C. and Saint Aroman, M. (2025) Epidemiology of Psoriasis: A Worldwide Global Study. *Acta Dermato-Venereologica*, **105**, adv42945. https://doi.org/10.2340/actadv.v105.42945

[15] Menter, A., Gottlieb, A., Feldman, S.R., Van Voorhees, A.S., Leonardi, C.L., Gordon, K.B., *et al.* (2008) Guidelines of Care for the Management of Psoriasis and Psoriatic Arthritis. *Journal of the American Academy of Dermatology*, **58**, 826-850. https://doi.org/10.1016/j.jaad.2008.02.039

[16] Krueger, G. and Ellis, C.N. (2005) Psoriasis—Recent Advances in Understanding Its Pathogenesis and Treatment. *Journal of the American Academy of Dermatology*, **53**, S94-S100. https://doi.org/10.1016/j.jaad.2005.04.035

[17] Roberson, E.D.O. and Bowcock, A.M. (2010) Psoriasis Genetics: Breaking the Barrier. *Trends in Genetics*, **26**, 415-423. https://doi.org/10.1016/j.tig.2010.06.006

[18] Palfreeman, A.C., McNamee, K.E. and McCann, F.E. (2013) New Developments in the Management of Psoriasis and Psoriatic Arthritis: A Focus on Apremilast. *Drug Design, Development and Therapy*, **7**, 201-210. https://doi.org/10.2147/dddt.s32713

[19] Nestle, F.O., Kaplan, D.H. and Barker, J. (2009) Psoriasis. *New England Journal of Medicine*, **361**, 496-509. https://doi.org/10.1056/nejmra0804595

[20] Rendon, A. and Schäkel, K. (2019) Psoriasis Pathogenesis and Treatment. *Interna-*

tional Journal of Molecular Sciences, **20**, Article 1475. https://doi.org/10.3390/ijms20061475

[21] Chan, J.J. (2025) Psoriasis: An Update on Topical and Systemic Therapies. *Australian Prescriber*, **48**, 87-92. https://doi.org/10.18773/austprescr.2025.026

[22] El-Reshaid, K. and Shaima, A. (2019) Long-Term Maintenance Therapy with Cyclosporine a in Adults with Generalized Pustular Psoriasis. *Journal of Drug Delivery and Therapeutics*, **9**, 19-21. https://doi.org/10.22270/jddt.v9i5.3535

[23] El-Reshaid, K., Al-Bader, S. and Madda, J.P. (2024) Systemic Amyloidosis Secondary to Psoriasis: A Rare, Autoimmune and Genetically-Determined Disorder That Is Amenable to Treatment with Cyclosporin A—Cyclosporin a for Psoriasis-Induced Amyloidosis. *Open Journal of Nephrology*, **14**, 226-232. https://doi.org/10.4236/ojneph.2024.142021

[24] Matsuda, S. and Koyasu, S. (2000) Mechanisms of Action of Cyclosporine. *Immunopharmacology*, **47**, 119-125. https://doi.org/10.1016/s0162-3109(00)00192-2

[25] Shihab, F.S. (1996) Cyclosporine Nephropathy: Pathophysiology and Clinical Impact. *Seminars in Nephrology*, **16**, 476-536.