

Case Report

Open Access, Volume 5

Adalimumab triumphs: Psoriatic arthritis case report**Debashis Priyadarshan Sahoo^{1*}; Gwenette Andrea War²**¹Senior Resident, Department of General Medicine, All India Institute of Medical Sciences (AIIMS), Guwahati, India.²Assistant Professor, Department of General Medicine, All India Institute of Medical Sciences (AIIMS), Guwahati, India.***Corresponding Author:****Debashis Priyadarshan Sahoo**Senior Resident, Department of General Medicine,
All India Institute of Medical Sciences (AIIMS),
Guwahati, India.

Email: hpydps@gmail.com

Received: Sep 23, 2024

Accepted: Oct 17, 2024

Published: Oct 24, 2024

Archived: www.jcimcr.org

Copyright: © Sahoo DP (2024).

DOI: www.doi.org/10.52768/2766-7820/3309

Abstract

This case report highlights the successful treatment of a patient with psoriatic Arthritis (PsA) using Adalimumab, a monoclonal antibody targeting tumour necrosis factor-alpha. The patient, diagnosed with PsA concurrent with skin psoriasis, exhibited significant joint pain, swelling, and disability. There was an incomplete response to conventional treatment with Disease Modifying Anti-Rheumatic Drugs. Following the initiation of Adalimumab therapy, a rapid improvement was observed in both joint symptoms and skin lesions. The patient reported a marked reduction in pain, increased mobility, and enhanced quality of life. Adalimumab's effectiveness was further supported by its favourable safety profile, with no serious adverse events documented during the treatment period. This case underscores the therapeutic potential of Adalimumab in managing PsA and provides evidence for its dual efficacy in addressing both joint and skin manifestations. Further research is recommended to establish long-term treatment outcomes and refine management strategies for patients with PsA.

Keywords: Psoriatic arthritis; Psoriasis; PsA; Adalimumab; TNF- α .**Introduction**

Psoriatic Arthritis (PsA) is a chronic inflammatory autoimmune disorder that affects both the joints and skin, occurring in approximately 30% of individuals with psoriasis [1]. The condition leads to significant morbidity and reduced quality of life. Treatment options for PsA include Non-Steroidal Anti-Inflammatory Drugs (NSAIDs), conventional Disease-Modifying Anti-Rheumatic Drugs (DMARDs), and biologic therapies [2]. Adalimumab (ADA), a fully human monoclonal antibody targeting Tumour Necrosis Factor-Alpha (TNF- α), has shown promise in alleviating symptoms and improving functional ability in PsA patients [3]. This case report highlights the effective management of PsA with Adalimumab in a patient with previously inadequate responses to other treatments.

Case presentation**Patient information:** The patient, a female in her 50s, had a

5-year history of psoriasis and presented with persistent asymmetric joint pain, swelling, redness, and early morning stiffness affecting multiple small joints, particularly the distal interphalangeal joints of the upper limbs, for the past 8 months. She reported significant fatigue and difficulty performing daily activities due to the pain.

Clinical examination: Upon examination, the patient was anxious with a pulse rate of 90 beats per minute, blood pressure of 118/74 mmHg, and a respiratory rate of 18 cycles per minute. Physical examination revealed tenderness and swelling in the distal interphalangeal joints, metacarpophalangeal joints, and knees. The Tender Joint Count (TJC) and Swollen Joint Count (SJC) were 22/28 and 17/28, respectively, with a Visual Analog Scale (VAS) score of 8/10. Dermatological examination showed erythematous plaques with silvery scales on the proximal interphalangeal joints, elbows, and knees, consistent with psoriasis (Figure 1). Other systemic examinations were unremarkable.



Figure 1: Bilateral hand deformities with notable swelling and distortion of the Proximal Interphalangeal (PIP) and Distal Interphalangeal (DIP) joints, indicative of synovitis and chronic joint inflammation. Nail involvement is prominent, with dystrophic changes such as onycholysis, thickening, and discoloration, characteristic of psoriatic onychodystrophy. Scaly erythematous plaques are visible over several joints, suggesting concurrent cutaneous psoriasis).



Figure 2: The radiograph of both hands demonstrates advanced joint pathology consistent with psoriatic arthritis. Notably, there is significant narrowing of the joint spaces, particularly at the proximal and distal interphalangeal joints (PIP and DIP), indicative of severe cartilage loss. The affected joints show erosive changes and pencil-in-cup deformities, typical of destructive arthropathy seen in psoriatic arthritis. Periarticular osteopenia and subchondral sclerosis are also present. The Metacarpophalangeal (MCP) joints appear relatively spared, though there is evidence of some degenerative changes. Overall, the findings reflect progressive joint destruction and deformity, indicative of untreated or inadequately controlled psoriatic arthritis).

Diagnostic assessment: Initial laboratory tests revealed elevated C-Reactive Protein (CRP) at 152 mg/L and an Erythrocyte Sedimentation Rate (ESR) of 90 mm/hour. Haemoglobin was 10.2 g/dL, total white blood cell count was 6800/ μ L with 67% neutrophils, platelet count was 190,000/ μ L, urea was 34 mg/dL, and creatinine was 1.1 mg/dL. Urinalysis was normal. Rheumatoid factor and anti-CCP antibodies were negative. X-rays of the hands showed joint space narrowing, bony erosions, and pencil-in-cup deformities, with evidence of soft tissue swelling (Figure 2). HLA-B27 was positive. The patient was diagnosed with PsA and was considered for an escalation of treatment.

Treatment course: Prior to presentation at our clinic, the patient had been on Methotrexate 15 mg weekly, Sulfasalazine 1 gram daily, and a short course of Prednisolone 20 mg, with minimal symptom relief. Given the severity of her arthritis and inadequate response to conventional therapies, Adalimumab

was introduced after discontinuing Methotrexate. The patient received an initial dose of Adalimumab 80 mg subcutaneously, followed by a maintenance dose of 40 mg every other week. She was trained on self-administration and monitored for adverse effects.

Outcome and follow-up: At the 12-week follow-up, the patient reported significant improvement in joint pain, with a reduction in the VAS score from 8 to 2. Objective assessment showed decreased joint swelling and tenderness, with TJC and SJC dropping to 2/28 and 1/28, respectively. The psoriasis lesions improved substantially, with over 75% reduction. Laboratory tests indicated a marked decrease in inflammatory markers, with CRP levels reduced to 3 mg/L and ESR to 10 mm/hour. The patient also reported improved quality of life, with fewer limitations in daily activities as measured by the Health Assessment Questionnaire. No significant adverse reactions were observed.

Discussion

PsA is a multifaceted condition that shares both genetic and clinical characteristics with other forms of spondylarthritis. Although PsA is less prevalent compared to skin psoriasis, it can affect up to 30% of individuals with psoriasis [4]. Notably, in about 84% of PsA cases, skin symptoms precede the onset of joint symptoms, highlighting a significant connection between the two manifestations of the disease [5]. Understanding the underlying mechanisms of PsA is crucial for developing effective treatment strategies. The pathophysiology of PsA involves several key factors, particularly cytokines such as Tumour Necrosis factor-Alpha (TNF- α) and interleukins IL-17 and IL-23 [6]. These molecules play a central role in mediating inflammation and the immune response. Immature dendritic cells in PsA patients exhibit increased expression of Toll-like receptor 2, which triggers a T helper 1 (Th1) response [7]. This response leads to elevated production of TNF- α , IL-12, and Interferon-gamma (IFN- γ) [4-8]. The excessive production of these pro-inflammatory cytokines contributes to the development and progression of both skin and joint symptoms, underscoring the importance of targeting these pathways in treatment. One of the most successful treatments for PsA is Adalimumab, a monoclonal antibody that specifically targets TNF- α . By inhibiting this key cytokine, Adalimumab provides significant relief from joint pain, swelling, and stiffness that are hallmark symptoms of the condition [3-9]. Moreover, it also improves skin lesions associated with psoriasis, addressing the dual challenges of PsA. This dual effect is particularly beneficial for patients who experience both skin and joint symptoms, providing a comprehensive approach to treatment.

Adalimumab is administered via subcutaneous injection, which enhances patient adherence to the treatment regimen [3]. This method allows patients the convenience of self-administration at home, removing the need for frequent visits to healthcare facilities. This convenience is essential for long-term management, as consistent therapy is crucial for controlling symptoms and preventing disease progression. The efficacy and safety of Adalimumab have been supported by robust clinical trial data, establishing it as an effective option for managing PsA [10-12]. The favourable safety profile of Adalimumab makes it a preferable choice, as many patients tolerate it well over extended periods. However, like all medications, it is essential for healthcare providers to monitor patients for any potential side effects and to tailor treatment plans to individual patient needs.

Conclusion

This case report highlights the successful management of Psoriatic arthritis with Adalimumab in a patient who had an unsuccessful conventional treatment. The significant improvement in joint and skin symptoms underscores the importance of biologic agents in PsA management. Future research should focus on long-term outcomes across diverse patient populations and optimal treatment strategies.

Declarations

Acknowledgments: None.

Contributions from the writers: The final text was actively written, edited, and approved by both authors.

Availability of data and material: This article does not fall under the category of data sharing because no data sets were created or examined for this investigation.

Conflicts of interest: No potential conflicts of interest were disclosed by the authors with regard to the publication of this paper.

Funding: The publication of this paper was all done without any financial assistance from the author.

Informed consent: The patient himself gave their written approval for the publication of his information in this article.

References

1. Tiwari V, Brent LH. Psoriatic Arthritis. In Treasure Island (FL): StatPearls Publishing. 2024. <http://www.ncbi.nlm.nih.gov/books/NBK547710>.
2. Gossec L, Kerschbaumer A, Ferreira RJO, Aletaha D, Baraliakos X, et al. EULAR recommendations for the management of psoriatic arthritis with pharmacological therapies: 2023 update. *Ann Rheum Dis*. 2024; 83(6): 706-19. <https://ard.bmj.com/content/83/6/706>.
3. Weinblatt ME, Keystone EC, Furst DE, Moreland LW, Weisman MH, et al. Adalimumab, a fully human anti-tumor necrosis factor alpha monoclonal antibody, for the treatment of rheumatoid arthritis in patients taking concomitant methotrexate: The AR-MADA trial. *Arthritis Rheum*. 2003; 48(1): 35-45.
4. Campanati A, Marani A, Martina E, Diotallevi F, Radi G, et al. Psoriasis as an Immune-Mediated and Inflammatory Systemic Disease: From Pathophysiology to Novel Therapeutic Approaches. *Biomedicines*. 2021; 9(11): 1511. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8615182>.
5. Management of psoriatic arthritis from the view of the dermatologist | *Nature Reviews Rheumatology*. 2024. <https://www.nature.com/articles/nrrheum.2011.125>.
6. Jang D In, Lee AH, Shin HY, Song HR, Park JH, et al. The Role of Tumor Necrosis Factor Alpha (TNF- α) in Autoimmune Disease and Current TNF- α Inhibitors in Therapeutics. *Int J Mol Sci*. 2021; 22(5): 2719. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7962638>.
7. Candia L, Marquez J, Hernandez C, Zea AH, Espinoza LR. Toll-like receptor-2 expression is upregulated in antigen-presenting cells from patients with psoriatic arthritis: A pathogenic role for innate immunity? *J Rheumatol*. 2007; 34(2): 374-9.
8. Gauthier T, Chen W. IFN- γ and TGF- β , Crucial Players in Immune Responses: A Tribute to Howard Young. *J Interferon Cytokine Res*. 2022; 42(12): 643-54. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9917322>.
9. Mease PJ. Adalimumab: An anti-TNF agent for the treatment of psoriatic arthritis. *Expert Opin Biol Ther*. 2005; 5(11): 1491-504.
10. Abitbol V, Benkhalifa S, Habauzit C, Marotte H. Navigating adalimumab biosimilars: an expert opinion. *J Comp Eff Res*. 2024; 12(11): 230117. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC10690439>.
11. Lu X, Hu R, Peng L, Liu M, Sun Z. Efficacy and Safety of Adalimumab Biosimilars: Current Critical Clinical Data in Rheumatoid Arthritis. *Front Immunol*. 2021; 12: 638444. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8055954>.
12. Burmester GR, Gordon KB, Rosenbaum JT, Arikian D, Lau WL, et al. Long-Term Safety of Adalimumab in 29, 967 Adult Patients from Global Clinical Trials Across Multiple Indications: An Updated Analysis. *Adv Ther*. 2020; 37(1): 364-80. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6979455>.