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CASE REPORT

Eosinophilia Induced by Biosimilar Adalimumab (CinnoRA) in a Patient with Psoriasis and Psoriatic Arthritis: A Case Report

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Background: Psoriasis is a chronic inflammatory skin disorder that can significantly impact quality of life. Biologic therapies, such as TNF-alpha inhibitors, have improved clinical outcomes but may rarely cause hematologic abnormalities, including eosinophilia. Eosinophilia is uncommon but can be associated with allergic reactions or organ involvement.

Case Presentation: We report a 33-year-old male with psoriasis and psoriatic arthritis who developed asymptomatic marked eosinophilia after eight doses of biosimilar adalimumab (CinnoRA). Baseline peripheral blood eosinophil percentage was 3.2%, which increased to 19.9% during therapy. Alternative causes, including parasitic infection, allergy, and hematologic disease, were excluded. CinnoRA was discontinued, and eosinophil counts normalized during follow-up.

Conclusion: This case illustrates that unexplained eosinophilia can occur during TNF-alpha inhibitor therapy. While routine monitoring is not universally recommended based on a single case, clinicians should consider eosinophilia as a possible adverse reaction, especially in symptomatic patients or those with persistently elevated counts.

Keywords: eosinophilia, adalimumab, TNF-alpha inhibitors, psoriasis

Introduction

Psoriasis is driven by Th1 and Th17-polarized CD4 T cells that produce pro-inflammatory cytokines such as TNF-alpha, leading to acanthosis and papillomatosis.[1] Blocking TNF-alpha improves clinical outcomes in psoriasis[2,3] and results in fewer side effects than those seen with classic immunosuppressive medications such as cyclosporine or methotrexate. In the past decade, TNF-alpha inhibitors have been used as first-line treatment for autoimmune diseases, including rheumatoid arthritis, ankylosing spondylitis, Crohn's disease, uveitis, and psoriasis.[4,5] Although generally safe and well tolerated, side effects occasionally occur. Eosinophilia is reported in the product sheet of adalimumab (Humira, CinnoRA), occurring in approximately 1% of patients.[6] In the eHealthMe database, eosinophilia occurred during adalimumab therapy in 0.07% of cases.[7] Only a few reports have described eosinophilia in psoriasis patients receiving these biologics, and most cases occurred within the first months of therapy.[8–12] Besides blood eosinophilia, eosinophilic pathologies were also associated with TNF-alpha inhibitors.[13,14] Eosinophilic infiltrates in solid organs, although uncommon, represent a significant complication of TNF-alpha inhibitor therapy.[15,16]

The mechanism by which TNF-alpha inhibition leads to blood eosinophilia remains unclear. The generation of IgE-class-switched antibodies may contribute to IgE-mediated drug hypersensitivity and subsequent eosinophilia; furthermore, anti-TNF-alpha agents, especially adalimumab, might also be able to influence eosinophil apoptosis.[17]

Here, we briefly report the case of a man with concurrent psoriasis and psoriatic arthritis who developed blood eosinophilia during treatment with adalimumab (cinnoRA).

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Case Presentation

A 33-year-old male with a history of psoriasis, initially diagnosed at age 11, presented with classic plaque-type psoriasis. His condition had been well-controlled with topical corticosteroids during adolescence. At age 22, he experienced a significant flare that required escalation of treatment. In 2020, due to ongoing disease activity, therapy with acitretin (Neotigazone) was initiated and continued until 2023, providing partial control. By 2023, the patient developed psoriatic dactylitis, metabolic syndrome, and extensive plaque psoriasis covering 30% of body surface area (PASI score: 19.2), prompting initiation of adalimumab (CinnoRA) at a dose of 40 mg subcutaneously every two weeks.

The baseline white blood cell count (WBCc) prior to starting adalimumab on August 20, 2023 was 5980, and the peripheral blood eosinophil percentage (PBEp) was 3.2%. After eight doses (December 23, 2023), WBCc increased to 11,220, with PBEp at 19.9%. The patient remained completely asymptomatic, without rash, urticaria, respiratory symptoms, or systemic complaints. Laboratory follow-up showed a progressive rise in absolute eosinophil count, peaking at 2250 cells/μL (Figure 1), accompanied by a mild increase in total WBC count (Figure 2).

A comprehensive work-up was performed to exclude secondary causes of eosinophilia. Hematologic malignancy was ruled out by complete blood count with differential, peripheral smear, and absence of abnormal cells. Parasitic infection was excluded by stool ova and parasite examination and serology. Allergic disease was excluded by negative skin prick testing and allergen-specific IgE assays. Serum IgE was mildly elevated on two occasions, but there was no history of atopy. Inflammatory markers (ESR, CRP) and interleukin-6 were normal.

Management involved a multidisciplinary team including dermatology, rheumatology, and hematology specialists. Pulmonary involvement was excluded by a pulmonologist through clinical assessment and imaging. Given the persistent and marked eosinophilia, CinnoRA was discontinued. No alternative biologic was started immediately, and the patient was monitored closely. Eosinophil counts gradually decreased, returning to normal within two months of drug cessation. Follow-up continued for six months, during which the patient remained free of eosinophilia and without signs of organ involvement.

Because our patient was experiencing disease relapse (Figure 3), with enthesitis, sacroiliac pain, and scattered psoriatic lesions, and given the limited therapeutic options available in Iran, we were obliged to restart the drug. The patient's asymptomatic status, both clinically and in laboratory evaluations during the first episode, also supported the decision to re-challenge. The eosinophil count increased again, and at present the patient has an asymptomatic eosinophilia of approximately 960 cells/μL and is under regular monitoring.

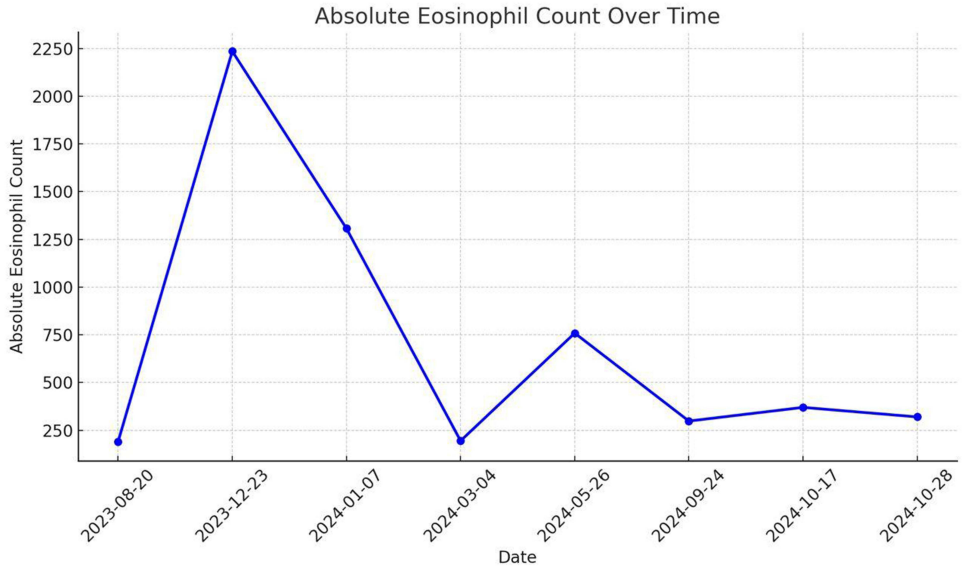

Figure 1 Trend of absolute eosinophil count over time during treatment with biosimilar Adalimumab (CinnoRA). The absolute eosinophil count peaked at approximately 2250 cells/μL after the eighth dose, followed by gradual normalization after discontinuation of the drug.

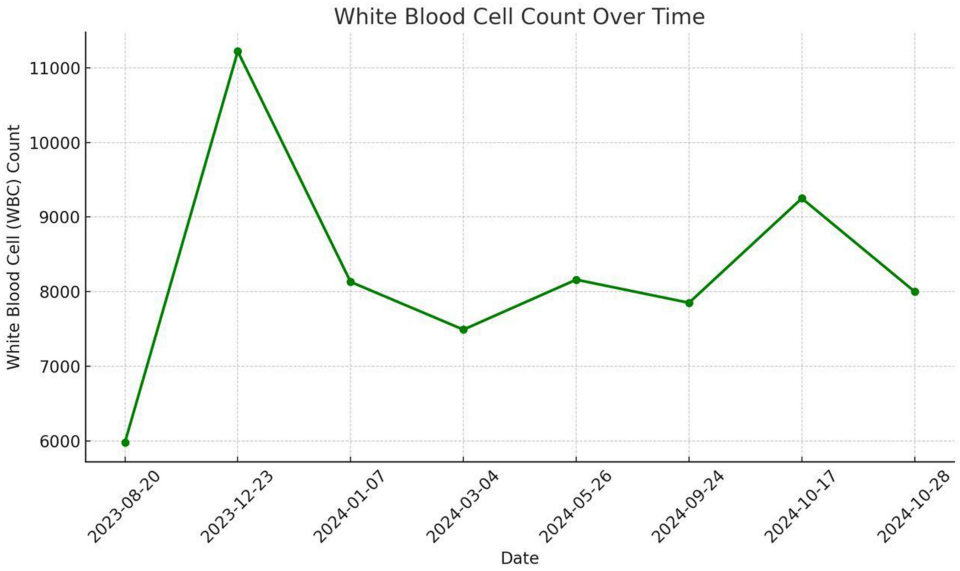

Figure 2 Trend of white blood cell (WBC) count over time during treatment with biosimilar Adalimumab (CinnoRA). WBC count showed a transient increase during therapy and returned to baseline after cessation of treatment.

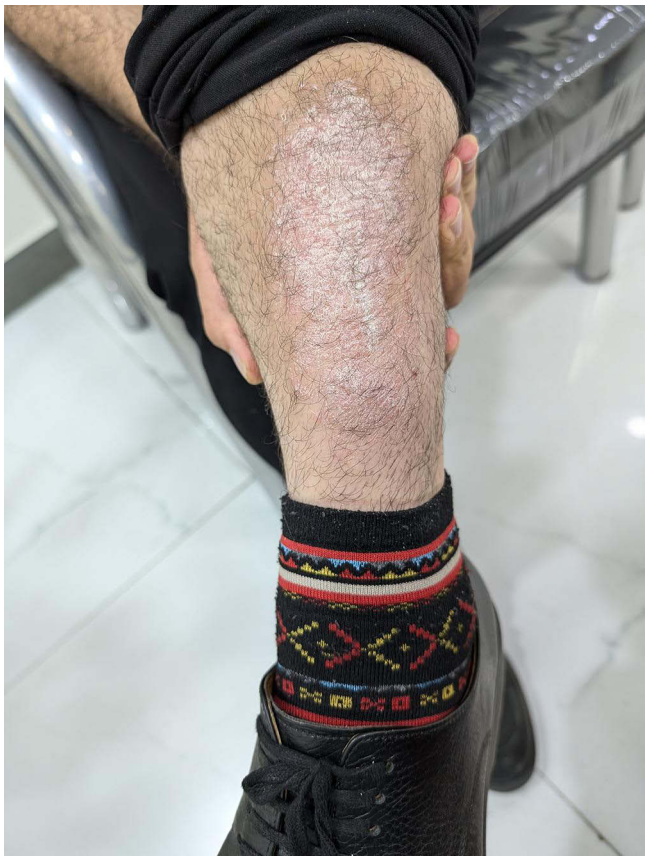

Figure 3 Clinical image showing a treatment-resistant psoriatic plaque.

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Discussion

Drug-induced eosinophilia is relatively rare but clinically relevant, particularly in patients receiving TNF-alpha inhibitors such as adalimumab, which are commonly used in the management of chronic inflammatory diseases including psoriasis.

TNF-alpha blockade with agents such as adalimumab may disinhibit Th2 responses, resulting in enhanced eosinophil survival, activation, and proliferation. Additionally, TNF-alpha inhibition may impair eosinophil apoptosis and facilitate IgE-mediated hypersensitivity, further promoting eosinophilic responses.[9,17,18]

In our patient, eosinophilia appeared after several months of CinnoRA therapy, was asymptomatic, and resolved after drug withdrawal. This temporal relationship, along with exclusion of alternative causes, supports a causal association. The brochures describing adverse effects are written for the originator drug, whereas biosimilars are not exactly the same as the original. This is the first case showing eosinophilia with CinnoRA. The occurrence after drug initiation, resolution after withdrawal, and recurrence upon rechallenge all support a drug-related event.

Comparison with previous reports shows variability in presentation. Malisiewicz et al[8] reported both de novo and pre-existing eosinophilia during TNF-alpha inhibitor therapy. Guidelli and Fioravanti[10] described severe eosinophilia in patient with psoriatic arthritis resolving after adalimumab withdrawal. Vester et al[11] reported intermittent eosinophilia without clinical sequelae. Our case is similar in being asymptomatic, but differs in the gradual onset over several months. Importantly, among the reported cases, only our case demonstrated reproducible eosinophilia upon re-challenge, providing the strongest evidence of causality. Table 1 summarizes selected reports of eosinophilia during adalimumab therapy in psoriasis and related conditions.

Clinicians should interpret asymptomatic eosinophilia cautiously. In some cases, eosinophil counts may normalize without intervention, while in others, drug withdrawal is warranted to prevent possible organ involvement. Persistent eosinophilia can be a harbinger of eosinophil-mediated damage to the heart, lungs, gastrointestinal tract, or nervous system.[15,16]

Limitations of this report include the single-patient design, limited duration of follow-up, and absence of tissue biopsy to assess for subclinical eosinophilic infiltration.

Given the rarity of this event, we do not recommend universal routine CBC monitoring solely on the basis of this case. However, eosinophilia should be considered a possible adverse effect during TNF-α inhibitor therapy, especially in symptomatic patients or those with persistently elevated counts. In addition to reactive causes, persistent hypereosinophilia raises the possibility of primary clonal eosinophilic syndromes, such as chronic eosinophilic leukemia driven by

Table 1 Summary of Published Cases of Adalimumab-Associated Eosinophilia

Author, Year	Age/ Sex	Underlying Disease	Onset After Starting Drug	Peak Eosinophil	Symptoms	Outcome
Malisiewicz et al, 2011[8]	24/M	Psoriasis	3 months	1270/μL	Asymptomatic	Resolved after withdrawal
Vester et al, 2012[11]	58/F	Acrodermatitis continua of Hallopeau	1 months	3600 / μL	Asymptomatic	Resolved after withdrawal
Guidelli & Fioravanti, 2014[10]	59/M	Psoriatic Arthritis	5 months	2393/μL	Asymptomatic	Resolved after withdrawal
Chiriac et al, 2016[9]	42/F	Psoriasis	2 months	Not reported	Symptomatic (Fatigue, rash)	Resolved after withdrawal
Sargin et al, 2018[12]	39/M	Ankylosing spondylitis	Variable	Mild to Moderate	No major complications	Not specified
Tallon & Cockcroft, 2018[16]	55/F	Psoriasis	6 months	Not reported	Pulmonary infiltrates	Cough improved after withdrawal
Present case, 2024	33/M	Psoriasis/Psoriatic Arthritis	4 months	2233/μL	Asymptomatic	Resolved after withdrawal Recurrence on re-challenge

activating mutations like the FIP1L1-PDGFRA fusion gene. These primary hematologic disorders often require targeted therapy rather than simple withdrawal of the inciting agent.[18,19]

Conclusion

This case suggests eosinophilia as a potential adverse effect of the biosimilar adalimumab (CinnoRA). The reproducible recurrence upon re-challenge supports drug-related effect. Although asymptomatic in this case, persistent eosinophilia carries a risk of progression to organ involvement and should not be overlooked. While universal routine CBC monitoring is not currently warranted, clinicians should be alert to unexplained eosinophilia and report such events. For biosimilars in particular, where long-term post-marketing safety data are still limited compared with reference biologics, case-based evidence is crucial to strengthen pharmacovigilance systems and guide future recommendations.

Ethics Approval and Consent to Participate

Institutional approval was not required for publication of the details of this case. This case report was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Disclosure

The authors declare no conflicts of interest related to this work.

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