Psoriasis and palmoplantar pustulosis associated with tumor necrosis factor-α inhibitors: The Mayo Clinic experience, 1998 to 2010

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Background: Tumor necrosis factor (TNF)-α antagonists have been associated with the induction of de novo or worsening psoriasis.

Objective: We sought to retrospectively examine the clinical characteristics and outcomes of patients with psoriasis associated with anti–TNF-α therapy.

Methods: We performed a retrospective review of patients with new-onset or worsening psoriasis during TNF-α inhibitor therapy between 1998 and 2010.

Results: Of the 56 patients (mean age at psoriasis onset, 48.1 years), 41 (73%) were female. In all, 22 patients (39%) had Crohn's disease and 14 (25%) had rheumatoid arthritis. Thirty patients (54%) were treated with infliximab, 19 (34%) with adalimumab, and 7 (12%) with etanercept. New-onset or worsening psoriasis occurred after a mean treatment duration of 17.1 months. Plaque psoriasis (n = 27), palmoplantar pustulosis (n = 25), scalp psoriasis (n = 12), generalized pustular psoriasis (n = 7), erythrodermic psoriasis (n = 2), and inverse psoriasis (n = 2) were the cutaneous presentations. Among the 39 patients for whom full treatment response data were available, 33 (85%) had a complete or partial response; combined response rates (complete and partial) were slightly higher among those who discontinued anti–TNF-α therapy (16 of 17 patients [94%]) than among those who continued anti–TNF-α therapy (17 of 22 patients [77%]).

Limitations: Retrospective nature, possible referral bias, and lack of complete follow-up for some patients are limitations.

Conclusion: Although some patients sufficiently controlled their psoriasis while *continuing* anti–TNF-α therapy, those who discontinued therapy achieved higher rates of complete response. Further studies should explore the efficacy and safety of switching to an alternative anti–TNF-α agent. (J Am Acad Dermatol 2012;67:e179-85.)

Key words: adalimumab; etanercept; infliximab; palmoplantar pustulosis; plaque psoriasis; psoriasis; tumor necrosis factor-α inhibitor.

Tumor necrosis factor (TNF)-α is a proinflammatory cytokine important in innate and acquired immunity. It is expressed predominantly by monocytes and tissue macrophages. TNF-α is involved in monocyte differentiation, chemokine expression, and T-cell regulation.[1] Inappropriate release of TNF-α causes inflammation and tissue destruction through the release of proinflammatory mediators such as interleukin (IL)-1.[1] Therefore, TNF-α has emerged as a target for treatment of inflammatory conditions.

TNF-α antagonists are used to treat a wide spectrum of moderate to severe inflammatory conditions,

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including psoriasis, rheumatoid arthritis, and inflammatory bowel disease.[1-4] Psoriasis is a T-cell immune-mediated disease and therefore often favorably responds to TNF-α antagonists.[4] Three TNF-α inhibitors are currently approved for the treatment of psoriasis: etanercept (Enbrel, Immunex Corporation, Thousand Oaks, CA), infliximab (Remicade, Centocor Ortho Biotech Inc, Horsham, PA), and adalimumab (Humira, Abbott Laboratories, Abbott Park, IL). The mechanism of response to etanercept has been reported to be dependent on the inactivation of myeloid dendritic cell genes and inactivation of the type 17 helper T-cell immune response.[5]

TNF-α inhibitors neutralize TNF-α and disrupt activation of inducible nitric oxide synthase–producing dendritic cells and other macrophage-like cells that function through the IL-12 and IL-23 pathway.[6] One common theory is that decreasing functional levels of TNF-α ultimately cause suppression of inflammation and keratinocyte hyperproliferation through the reduction of IL-6, IL-8, and colony-stimulating factors.[7]

Paradoxically, published cases have linked all 3 agents (etanercept, infliximab, and adalimumab) to de novo or worsening psoriasis, and to date there have been more than 200 cases of psoriatic eruptions associated with TNF antagonist therapy.[8-10] The aim of the current study was to retrospectively examine the clinical characteristics and outcomes of patients at Mayo Clinic who had new-onset or worsening psoriasis during anti–TNF-α therapy.

CAPSULE SUMMARY

- Tumor necrosis factor (TNF)-α inhibitor therapy has been associated with new-onset or worsening psoriasis and psoriasiform eruptions.
- Plaque psoriasis and palmoplantar pustulosis were the most common cutaneous adverse effects associated with TNF-α inhibitor therapy.
- Patients generally experienced some improvement in their psoriasis with adjuvant treatment regardless of whether anti–TNF-α therapy was discontinued; further studies should explore the efficacy and safety of switching to an alternative anti–TNF-α agent.

METHODS

Inclusion criteria

We used the institutional medical index and text retrieval system to identify patients who were receiving TNF-α inhibitor therapy and in whom psoriasis subsequently developed. All patients were treated at Mayo Clinic, Rochester, MN, between January 1, 1998, and July 31, 2010. The following inclusion criteria were used: (1) development of first-time psoriasis, (2) development of a new type of psoriasis, (3) dramatic worsening of old psoriasis, and (4) reactivation of psoriasis that was in long-term remission. These lesions must have occurred while the patient was receiving TNF-α inhibitor therapy. Patients who denied research authorization were excluded from the study. Our study was approved by the Mayo Clinic Institutional Review Board.

Data collection

In all, 56 cases of psoriasis during TNF-α inhibitor therapy were identified. The following data were abstracted from patient records: sex, age at onset, TNF-α inhibitor used at the time of skin lesions, concomitant drugs at the time of skin lesions, previous TNF-α inhibitors used, duration of TNF-α inhibitor therapy at the time of skin lesions, reason for TNF-α inhibitor use, family history of psoriasis, personal history of psoriasis, treatment of new skin lesions, whether TNF-α inhibitor therapy was continued, whether the TNF-α inhibitor was switched, whether the skin lesions were new-onset psoriasis or worsening or recurrence of established psoriasis, the type of psoriasis corresponding to the new skin lesions, and the clinical outcome after treatment. Skin lesions were classified into 7 types of psoriasis: palmoplantar pustulosis, generalized pustular, erythrodermic psoriasis, plaque psoriasis, guttate psoriasis, inverse psoriasis, and scalp psoriasis. These psoriasis types were not mutually exclusive; multiple types of psoriasis may have been diagnosed in one patient.

Response to treatment

Clinical outcomes of psoriasis treatment were classified into 5 categories: (1) complete response—total resolution of active lesions and lack of new lesions, or at least 95% improvement as qualitatively graded by the physician; (2) partial response—presence of active lesions and healing of some lesions with decreased extent and severity of lesions; (3) no response—persistence of old lesions or worsening of lesions; (4) relapse—recurrence of psoriatic lesions after a period of resolution or significant improvement; and (5) unknown response—clinical outcome was not documented or patient was lost to follow-up.

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