Treatment of coexistent psoriasis and lupus erythematosus

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Background: The coexistence of psoriasis and lupus erythematosus (LE) is rare. Anecdotal evidence suggests that anti–tumor necrosis factor alfa (TNF-α) agents may be efficacious in LE, although their use is commonly avoided in this disease because of concern for lupus flare.

Objective: We sought to describe the epidemiology, serologic findings, and therapeutic choices in patients with coexistent psoriasis/psoriatic arthritis and LE and to determine the risk of lupus flares with TNF-α inhibitors.

Methods: We performed a retrospective multicenter study of patients given the diagnoses of psoriasis (or psoriatic arthritis) and lupus erythematosus (systemic LE or cutaneous LE, including either subacute cutaneous LE or discoid LE) at 2 academic tertiary-care centers.

Results: A total of 96 patients with a mean age of 56 years was included. We report higher-than-expected rates of white race and psoriatic arthritis. One clinical lupus flare was observed in a patient receiving a TNF-α inhibitor, resulting in an incidence of 0.92% lupus flares per patient-year of TNF-α inhibitor use.

Limitations: Retrospective chart review, small sample size, and limited documentation.

Conclusion: Anti–TNF-α agents, ustekinumab, and abatacept may be valid treatment options for patients with concomitant LE and psoriasis. Clinical lupus flares in LE patients treated with TNF-α inhibitors were infrequent. (J Am Acad Dermatol 2015;72:253-60.)

Key words: psoriasis; lupus erythematosus; tumor necrosis factor alfa.

INTRODUCTION

Psoriasis (Ps) and lupus erythematosus (LE) are immune-mediated diseases. Whereas Ps is primarily characterized by upregulation of the helper T cell (Th) 17 and Th1 immune pathways, LE is associated with upregulation of the Th1, Th2, and Th17 pathways, with constitutive B cell activation and autoantibody production.[1-4]

Ps is common, affecting 1% to 3% of the US population,[5] whereas LE is much less common: Reported prevalence rates for SLE in the United States range from 20 to 150 cases per 100,000 for SLE.[6] Cutaneous LE (CLE; including discoid LE [DLE] and subacute cutaneous LE [SCLE]) has similar prevalence rates.[7] The coexistence of LE with Ps is rare.

Medications commonly used for LE, such as mycophenolate mofetil and azathioprine, are not particularly efficacious in cutaneous Ps therapy, and antimalarials are known to make Ps worse. Likewise, ultraviolet light phototherapy, used for many patients with cutaneous Ps, is generally avoided in SLE and CLE because of risk of photosensitivity or lupus exacerbation. Although TNF-α inhibitors are a well-established therapy for Ps, they often are avoided in LE because of the unclear role of TNF-α in the

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disease and reports of TNF-α inhibitor–induced LE and induction of antinuclear antibodies.[8-12]

The most recent case series of patients with concomitant Ps and LE was published in 1980.[13] We present a comprehensive case review of 96 patients from 2 academic medical centers with Ps or psoriatic arthritis (PsA) plus LE (systemic lupus erythematosus [SLE], DLE, or SCLE). We aimed to determine the risk of clinical LE flare with use of the TNF-α inhibitors and to evaluate clinical outcomes resulting from the use of biologics in patients with Ps and SLE/CLE.

CAPSULE SUMMARY

- The coexistence of psoriasis with lupus erythematosus is rare and presents a therapeutic challenge.
- We present a review of 96 patients with coexistent disease and analyze treatment regimens in this population.
- The TNF-α inhibitors, and the newer biologics, ustekinumab and abatacept, may represent valid treatment options with little risk for clinical lupus flare.

METHODS

After local Institutional Review Board approval, a retrospective chart review of patients with coexistent psoriatic and SLE/CLE was performed at 2 tertiary-care academic institutions: Tufts Medical Center and Brigham & Women's Hospital.

Electronic and paper chart reviews of patient medical history and laboratory and pathology reports were performed. Medical records from January 1990 to February 2013 were available from the Brigham & Women's Hospital, and records from 2007 to 2013 were available from Tufts Medical Center. Patients must have received a diagnosis of Ps or PsA and a diagnosis of at least one type of LE, SLE, DLE, or SCLE by International Statistical Classification of Diseases and Related Health Problems, Version 9 (ICD-9) diagnosis codes. Each diagnosis must have been confirmed in a clinical note written by a rheumatologist (SLE) or dermatologist (DLE, SCLE). If a diagnosis of SLE was found by ICD-9 usage, but clinical notes lacked written diagnosis of SLE by a rheumatologist, the chart and laboratory reports were reviewed for 1997 American College of Rheumatology (ACR) criteria for SLE. A patient with ≥ 4 ACR criteria for SLE by clinical notes and laboratory results and an ICD-9 diagnosis was then included. Patients with documentation in any clinical note of diagnosis of or concern for drug-induced LE (any subtype), drug-induced lupus syndrome, Ps, or PsA were excluded.

Medication data were collected on US Food and Drug Administration–approved systemic medications for Ps. Other systemic agents were included in one group, "non-biologic systemic agents," and length of use of these agents was not collected because much of these data were not documented in the charts reviewed. Analysis of the use of biologics was included only if the given patient had received it after all diagnoses of Ps, PsA, and SLE/CLE were established. A clinical lupus flare was defined as worsening of any one of the 1997 ACR criteria for SLE or a worsening of DLE or SCLE and must have been confirmed by a dermatologist's (SCLE, DLE) or rheumatologist's (SLE) clinical note.

All statistical analyses were performed using SPSS software version 9.0.

RESULTS

Ninety-six patients fulfilled inclusion and exclusion criteria (Table I). Eighty-four (87.5%) patients were women and 12 (12.5%) were men (Table II). Most (77.1%) patients (including those with SLE or CLE) were white and had chronic plaque Ps (81.3%). In patients with SLE/CLE, Ps was present in 87 (90.6%) and PsA in 50 (52.1%). Forty patients (42.7%) had both Ps and PsA in addition to LE. Nine patients (9.4%) had LE and PsA (per clinical documentation) without cutaneous Ps. There were 85 (89.6%) diagnoses of systemic LE, 21 (21.9%) of DLE, and 9 (9.4%) of SCLE. Of all patients seen at Brigham and Women's Hospital and Tufts Medical Center who received a diagnosis of either Ps/PsA or LE, 0.015% and 0.017% of patients per year, respectively, received the concurrent diagnoses of Ps/PsA and LE.

Cutaneous photosensitivity was present in 39.6% of patients. Fifty-three patients (55%) had Ps diagnosed before LE; in 43 (45%), the diagnosis of LE preceded diagnosis of Ps (Table II). There was a longer latency period when Ps/PsA was diagnosed before LE (mean, 18.4 years) compared with the converse (mean, 9.2 years).

A summary of treatments used, lengths of treatment, adverse effects, and compliance rates is given in Table I. Most patients (66.7%) had received methotrexate, cyclosporine, or another nonbiologic systemic agent. The percentage of patients who discontinued taking a drug because of poor efficacy was not statistically significantly different among treatment groups.

Rates of adverse effects with biologic agents were consistent with those seen in clinical trials of the medications used in patients with Ps/PsA alone.[14,15] Overall, discontinuations of medication because of adverse effects were most common with