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1578-2190/


# Coexistence of Tumid Lupus Erythematosus and Discoid Lupus Erythematosus☆

## Coexistencia de lupus eritematoso túmido y lupus eritematoso discoide

*To the Editor:*

In 1909 the term lupus erythematosus tumidus was coined by Hoffmann,[1] and in 1930 Gougerot and Burnier[2] described the cases of 5 patients with similar clinical pictures consisting of nonscarring, erythematous, indurated facial lesions without surface changes. This condition, also known as tumid lupus erythematosus (TLE), has been largely overlooked in the literature, but has been recently characterized as a subtype of cutaneous lupus erythematosus (CLE) with peculiar clinical, photobiological, histological, and prognostic features.

A 70-year-old woman with a history of hypertension and Hashimoto thyroiditis was seen for asymptomatic skin lesions on the face that had appeared during the summer 5 months earlier and were not associated with any systemic clinical signs. The appearance of the facial lesions coincided with worsening of pre-existing lesions on the scalp and associated hair loss.

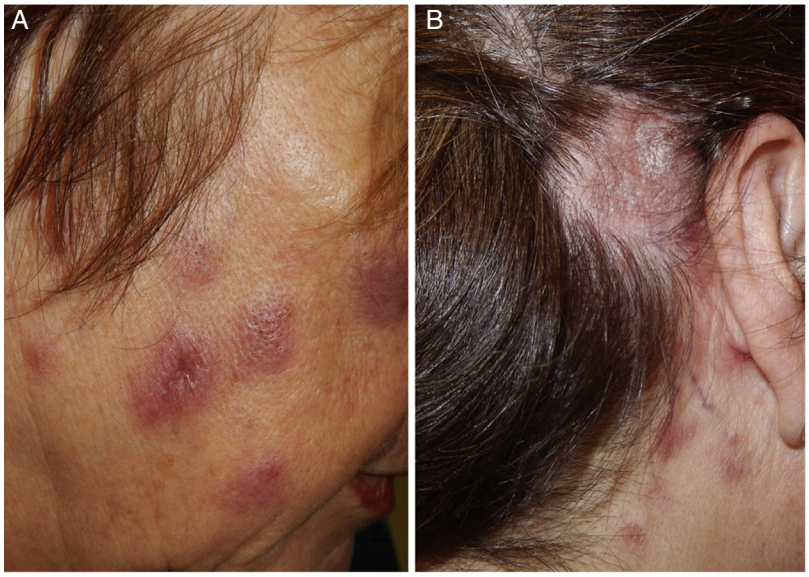


**Figure 1** A, Succulent, erythematous-violaceous facial plaques, without surface changes. B, Brownish, desquamative, alopecic erythematous plaque located in the retroauricular area of the scalp.

☆ Please cite this article as: Abadías-Granado I, Sánchez-Bernal J, Felipo-Berlanga F, Ara-Martín M. Coexistencia de lupus eritematoso túmido y lupus eritematoso discoide. Actas Dermosifiliogr. 2019;110:253–255.

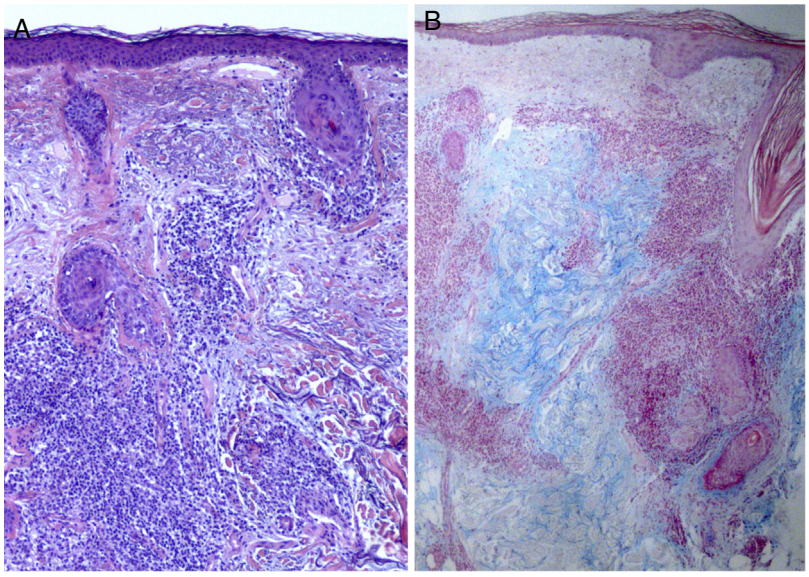


**Figure 2** Facial skin biopsy showing discrete epidermal atrophy with moderate perivascular and periadnexal lymphocytic infiltrate and abundant deposition of mucin in the dermis. A, Hematoxylin-eosin, original magnification ×10. B, Alcian blue, original magnification× 4.

Physical examination revealed the presence of erythematous, edematous, infiltrated, nondesquamative plaques on the face, cervical region, and upper chest (Fig. 1A), as well as erythematous, desquamative, alopecic plaques on the parietal and right retroauricular areas of the scalp (Fig. 1B).

Results of previous laboratory tests performed in another center revealed that the patient was positive for antinuclear antibodies (ANA) (1:320) and negative for anti-Ro and anti-La antibodies. All other parameters were within the normal range. The results of a biopsy were consistent with lymphocytoma cutis. Given the suspicion of a lymphoproliferative process induced in response to CLE, new biopsies and laboratory tests were performed. The results revealed a decrease in ANA levels to 1:80. The pathological report of the facial lesion described discrete epidermal atrophy and a perivascular and periadnexal lymphocytic infiltrate, with no signs of interface dermatitis and abundant mucin deposition in the dermis (Fig. 2). The results of the scalp biopsy were compatible with discoid lupus erythematosus (DLE) (Fig. 3).

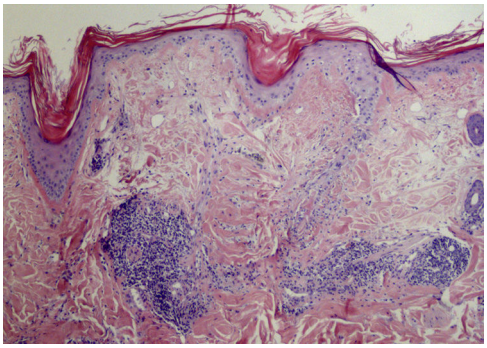

**Figure 3** Scalp biopsy showing perivascular and periadnexal lymphocytic dermatitis with epidermal atrophy, parakeratosis, and the presence of horny plugs in the follicular ostia (hematoxylin-eosin, original magnification × 20).

Based on these results the patient was diagnosed with CLE with concomitant DLE and TLE. The patient began treatment with photoprotection, 0.05% clobetasol propionate, and hydroxychloroquine at an initial dose of 400 mg/24 h followed by subsequent maintenance therapy at 200 mg/24 h, resulting in progressive clinical improvement.

The mean age of TLE patients is 36.4 to 38.5 years, and women and men appear to be affected equally.[3,4] Clinically, TLE is characterized by the appearance in sun-exposed areas of erythematous, succulent, urticariform, nondesquamative plaques that heal without scarring or hypopigmentation. Other characteristic features are a higher frequency of photosensitivity, as determined by phototesting, and a lower percentage positivity for anti-double-stranded DNA (dsDNA), anti-Ro (Sjögren's-syndrome-related antigen A), and anti-La (Sjögren's-syndrome-related antigen B) antibodies than described for other CLE subtypes.[3,4] Associated systemic disease in these patients appears to be very rare, albeit possible.[4,5] Histology reveals perivascular and periadnexal lymphocytic infiltrate and abundant deposition of interstitial mucin in the dermis. Compared with other CLE subtypes, the epidermis shows only mild alterations (or is intact), and basal vacuolization, hyperkeratosis, epidermal atrophy, and follicular plugging are less marked.[6] Treatment with systemic antimalarials is effective in approximately 90% of patients, as compared with 50% of DLE patients.[3] In addition to other CLE subtypes, the differential diagnosis should include polymorphic light eruption, Jessner lymphocytic infiltrate, reticular erythematous mucinosis, and pseudolymphoma,[3] all of which have clinical and microscopic characteristics that resemble those of TLE. Consensus is lacking regarding several aspects of TLE, including its differential diagnosis, classification, and microscopic characteristics. Because several of its features are distinct from those of other forms of lupus, some authors question the

origin of TLE, and consider it a photodermatosis outside the CLE spectrum.[7,8] However, we believe that classification of TLE as a true lupus subtype is justified based on the evidence published to date, in particular the coexistence of TLE and DLE lesions in certain patients,[3–5,9,10] as in the present case. Our description of a case of coexisting TLE and DLE adds to the small number of such cases reported in the literature, and should help resolve some of the controversy surrounding TLE, facilitating earlier diagnosis of this entity and better management of affected patients.

## Conflicts of Interest

The authors declare that they have no conflicts of interest.

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1578-2190/


# Paradoxical Arthritis Due to Ixekizumab in a Patient With Plaque Psoriasis☆

## Artritis paradójica por ixekizumab en un paciente con psoriasis en placas

*To the Editor:*

Our patient was a 46-year-old man who had been diagnosed with plaque psoriasis 27 years earlier and reported no previous axial or peripheral joint signs. Over the preceding years he had responded poorly to narrowband ultraviolet-B phototherapy, methotrexate, cyclosporine, etanercept, adalimumab, ustekinumab, and infliximab. At the time of the consultation he had numerous generalized plaques (psoriasis area and severity index [PASI], 10). Treatment was started with subcutaneous ixekizumab at the usual dose. After the first dose of 160 mg, the patient reported generalized migratory arthralgias that intensified and became disabling after the second dose of 80 mg, prompting an emergency visit to the rheumatology service. Physical examination revealed pain and marked limitation of cervical spinal cord mobility with no involvement of the lumbar or sacroiliac spinal cord (normal response in the Schober test and negative response to sacroiliac manipulation), pain and limited mobility of the scapular and pelvic girdles, pain and mild swelling of the carpal joints, and pain without swelling in the ankles, knees, and the small joints of the fingers. The painful joint count (PJC28) was 10 (carpal joints, knees, shoulders, and second and third bilateral metacarpophalangeal joints), and the swollen joint count (SJC28) was 2 (carpal joints). Based on these findings, the patient was diagnosed with paradoxical arthritis. With the patient's consent ixekizumab treatment was discontinued, and he was treated with a tapering dose of oral prednisone (20 mg/d) for 10 days, resulting in almost complete resolution of the joint problems. Upon reaching the end of the corticosteroid regimen, the patient was treated with secukinumab at the usual dose (300 mg). After 10 months of coordinated monitoring by the dermatology and rheumatology services, the patient's psoriasis had markedly improved (PASI, 1), with no adverse effects or joint symptoms.

Ixekizumab is a humanized IgG4 monoclonal antibody that acts to neutralize IL-17A, and in clinical trials has shown high efficacy in patients with plaque psoriasis[1] and psoriatic arthritis,[2] with an acceptable safety profile[3] and no

☆ Please cite this article as: Vidal D, Ros S, Reina D. Artritis paradójica por ixekizumab en un paciente con psoriasis en placas. Actas Dermosifiliogr. 2019;110:255–256.