# Tumid Lupus Erythematosus

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*Tumid lupus erythematosus (TLE) is a variant of cutaneous lupus erythematosus. Most patients who present with these skin lesions are young women. The condition clinically resembles polymorphous light eruption, systemic lupus erythematosus (SLE), reticulated erythematous mucinosis, or gyrate erythema. Histopathologically, the lesions resemble classic lupus erythematosus because of their superficial and deep lymphohistiocytic inflammatory infiltrates and dermal mucin. However, unlike classic lupus erythematosus, there is little or no epidermal or dermo-epidermal involvement. Antinuclear antibody test results are usually negative. We describe 4 cases of TLE and discuss the differential diagnosis.*

There have been few reports of tumid lupus erythematosus (TLE) in the literature.[1-4] Most major textbooks of dermatology or dermatopathology mention this entity only briefly, if at all. Ackerman et al[5] consider TLE to be a manifestation of discoid lupus erythematosus (DLE). We describe 4 patients with TLE and review the list of controversial entities that overlap clinically and histologically with TLE.

## Case Reports

*Patient 1*—A 34-year-old white woman presented with an 8-year history of asymptomatic lesions on her face, neck, chest, and upper extremities. She reported that the lesions persisted indefinitely without treatment. Topical corticosteroids were not helpful. Oral prednisone cleared the lesions within a week; however, she often needed to stay on a low dose of prednisone (10–20 mg/d) to prevent recurrence of the lesions. The patient also complained of arthralgias and fatigue.

On physical examination, the patient had multiple nonscaly, annular, erythematous plaques on her chest, upper extremities, and face (Figure 1). Test results for antinuclear antibodies (ANA), Ro, La, and dsDNA were all negative. Histopathology results revealed superficial and deep perivascular and periadnexal lymphohistiocytic infiltrates with dermal mucin. There was no involvement of the epidermis, the dermo-epidermal junction, or the papillary dermis (Figures 2 and 3).

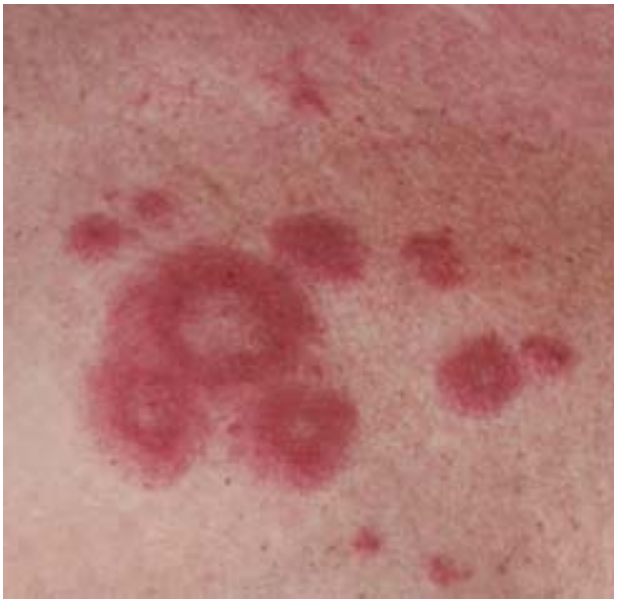

**Figure 1.** Erythematous papules and plaques on the chest of patient 1.

*Patient 2*—A 22-year-old white woman presented with asymptomatic lesions on her chest that had been present intermittently for 6 years. Her medical history was noncontributory.

Physical examination revealed multiple confluent, nonscaly, erythematous plaques on the patient's chest (Figure 4). Test results for ANA, Ro, La, and dsDNA were all negative. Histopathology results were identical to that of patient 1.

The eruptions on the patient persisted and worsened without treatment; however, they resolved after one week of treatment with oral hydroxychloroquine 200 mg twice daily. Repeated attempts to discontinue use of hydroxychloroquine resulted in a recurrence of the lesions within 6 months.

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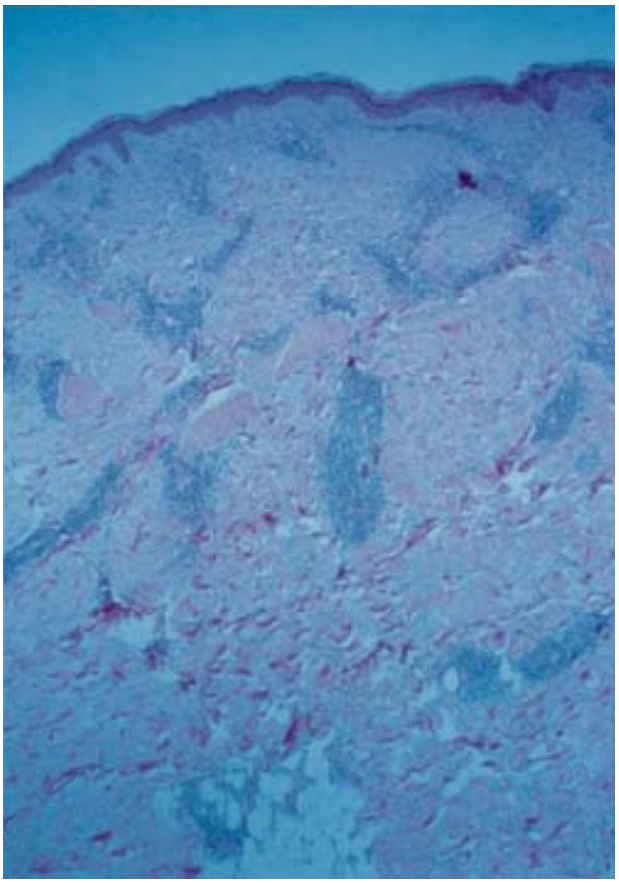

**Figure 2.** Superficial and deep perivascular and periadnexal lymphocytes with dermal mucin and no epidermal or dermo-epidermal involvement (H&E, original magnification ×40).

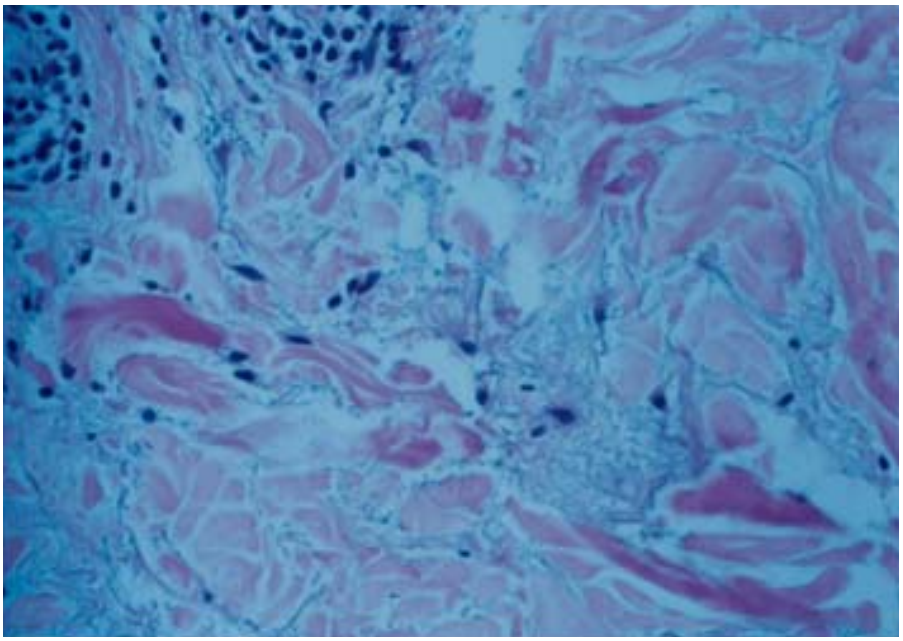

**Figure 3.** Dermal mucin (H&E, original magnification ×400).

*Patient 3*—A 28-year-old white woman presented with a 7-year history of intermittent facial eruptions. Her medical history included hypothyroidism, depression, spastic colon, gastritis, and back pain. Her medications included levothyroxine, venlafaxine for depression, and tramadol for back pain.

On physical examination, there were several erythematous, nonscaly plaques 1 to 2 cm in diameter on her forehead and cheeks. Test results for

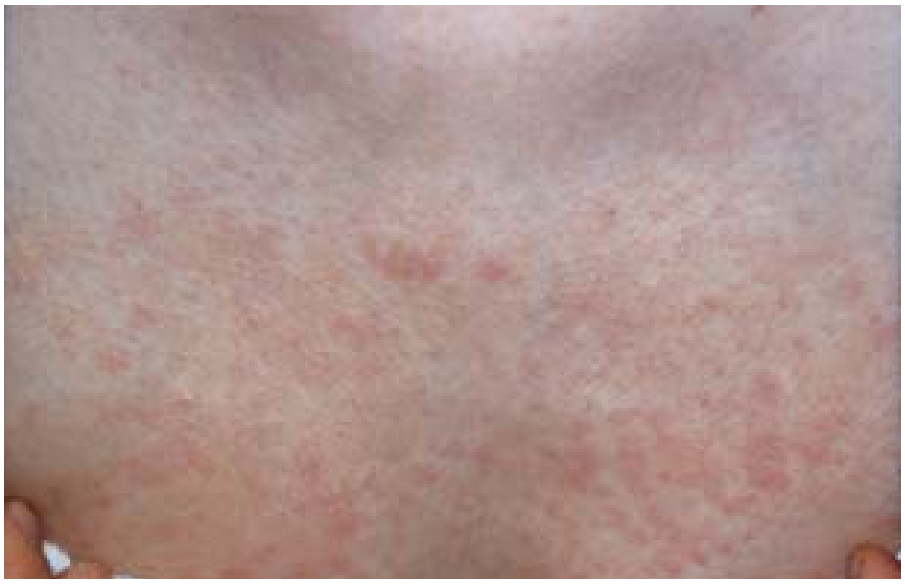

**Figure 4.** Confluent erythematous plaques on the chest of patient 2.

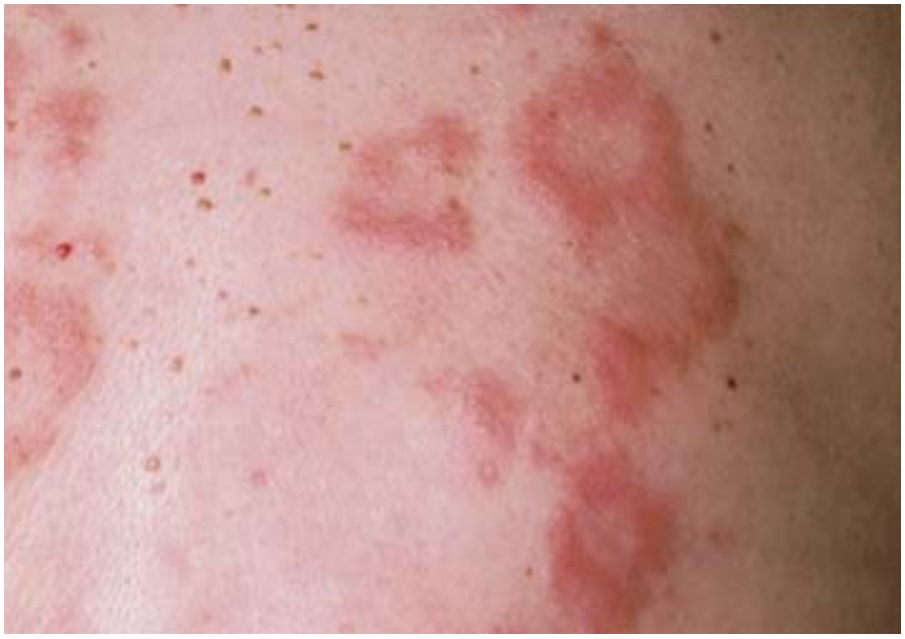

**Figure 5.** Annular erythematous plaques on the back of patient 4.

ANA, Ro, La, and dsDNA were all negative. Histopathology results were identical to that of the previous 2 patients.

*Patient 4*—A 48-year-old white woman presented with a 10-year history of recurrent asymptomatic lesions on her chest, back, neck, and arms. The lesions were persistent and resolved only after taking prednisone. The patient also complained of arthralgias whenever her skin eruptions flared. Her medications included sertraline (Zoloft®) and levothyroxine (Synthroid®).

On physical examination, there were several erythematous, nonscaly, annular plaques on her chest, back (Figure 5), and proximal upper extremities. Test results for ANA, Ro, La, and dsDNA were all negative. As in the previous 3 cases, histopathology results revealed superficial and deep perivascular and periadnexal lymphohistiocytic infiltrates with dermal mucin. However, there was a focal area of vacuolar involvement. Results of direct immunofluorescence of lesional skin from the patient's back were negative.

## Comment

TLE is a little-recognized entity that is probably more common than the few cases reported in the literature.[1-4] We believe TLE has been described as lymphocytic infiltration of Jessner-Kanof and as

reticulated erythematous mucinosis, among other terms. It is likely that many authors thought these were distinct entities. However, most current major dermatopathology textbooks now mention these entities as indistinguishable histopathologically from TLE. Clinically or histologically, there are no specific criteria that enable a specific diagnosis of either lymphocytic infiltration of Jessner-Kanof or reticulated erythematous mucinosis.[4] Ackerman et al[5] consider lymphocytic infiltration of Jessner-Kanof to be an expression of TLE, the plaque form of polymorphous light eruption (PLE), or gyrate erythema. Weyers et al[6] were unable to separate lymphocytic infiltration of the skin from the spectrum of lupus erythematosus using clinical, histopathologic, and immunofluorescence criteria. Dekle et al[7] suggest that Jessner-Kanof and TLE may be the same disease.

Most patients with TLE are young women who present with erythematous papules or plaques on the face, neck, chest, upper back, and upper extremities. The lesions often simulate lesions of systemic lupus erythematosus (SLE) or PLE. However, TLE is distinguished histologically from SLE by the absence or scarcity of epidermal or dermo-epidermal involvement and is distinguished from PLE by the absence of papillary dermal edema. Also, in contrast to SLE, patients with TLE often have negative test results for antinuclear antibodies.

In addition to lymphocytic infiltration of Jessner-Kanof, reticulated erythematous mucinosis, SLE, and PLE, the clinical and histopathological differential diagnosis of TLE includes subacute cutaneous lupus erythematosus, DLE, and deep gyrate erythema. The lesions of TLE may be indistinguishable from those of subacute cutaneous lupus erythematosus. However, patients with TLE usually have negative Ro and La antibodies. As previously mentioned, Ackerman et al[5] consider TLE to be a variant of DLE because TLE is a cutaneous variant of lupus erythematosus and most patients with TLE or DLE have negative antinuclear antibodies. However, the histology of both DLE and SLE has epidermal and prominent dermo-epidermal junction involvement with vacuolar degeneration and necrotic keratinocytes. Finally, deep gyrate erythema can be distinguished from TLE by the absence of dermal mucin.

In summary, we have presented 4 cases that clinically resemble cutaneous lupus erythematosus whose histopathology results showed features of lupus without the typical prominent interface changes. In addition, lupus serology results were all negative. Direct immunofluorescence was performed only on patient 4 because the test is now considered to be less reliable than previously thought. It has a high false-positive result in at least 20% of healthy adults.[8] There have been few reports of TLE. What's more, the classification of entities with histologic findings identical to those of TLE is controversial and not well defined. Thus, the percentage of patients with TLE who have a positive immunofluorescence is difficult to assess.

We present these cases as examples of TLE and have reviewed the differential diagnosis. Entities such as lymphocytic infiltrate of Jessner-Kanof and reticulated erythematous mucinosis, which are indistinguishable from TLE histopathologically and are not well defined clinically, should be categorized as manifestations of TLE.

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