

# An Overview on Tumid Lupus Erythematosus

Sehnaz Sheikh\*

Department of Allergy and Rheumatology, National University, Gazipur, Bangladesh

## DESCRIPTION

Tumid Lupus Erythematosus (TLE) is a photosensitive variant of cutaneous Lupus Erythematosus (cutaneous LE) that often manifests as erythematous, edematous plaques. TLE is distinct from the other cutaneous lupus erythematosus subtypes in that it rarely develops into Systemic Lupus Erythematosus (SLE) [1]. Some people believe TLE is a different entity from lupus because of its poor link to SLE and the lack of serologic abnormalities in TLE patients. Tumid erythematosus lupus is characterized by smooth, non-scarring pink to violet-colored pimples on the skin with no other visible skin abnormalities like scarring.

It's a rare, photosensitive inflammatory skin condition characterized by erythematous urticarial plaques [2]. Lupus Erythematosus (LE) is a disease with a wide range of symptoms, from cutaneous signs to significant organ system involvement, as seen in SLE. Acute, sub-acute, and Chronic Cutaneous Lupus Erythematosus (CCLE) include Discoid Lupus Erythematosus (DLE), lupus panniculitis, hypertrophic LE, and TLE. Other systemic or cutaneous illness signs and symptoms are uncommon in TLE patients [3]. Correctly identifying TLE and excluding other cutaneous disorders, which it commonly mimics, is diagnostic difficulty. TLE is also linked to Systemic Lupus Erythematosus (SLE).

No distinct etiology for TLE has been identified. However, activating factors like Ultraviolet (UV) radiation have been linked to the progression of TLE lesions. Its link to autoimmune disease is debatable; if an autoimmune disease is suspected, an autoimmune workup may be ordered [4]. TLE is uncommon compared to Discoid Lupus Erythematosus (DLE). TLE's incidence and prevalence among various races and ethnicities are unknown. Immune changes, notably a decrease in T-regulatory cells and epidermal Langerhans cells and an increase in plasmacytoid dendritic cells, have been linked to TLE etiology. The presence of extensive dermal mucin deposition, as well as a superficial and deep perivascular and periadnexal lymphocytic infiltration, distinguishes TLE [3].

## Physical consequences of TLE

Although this observation is not specific to TLE, worsening of the eruption with sun exposure supports a diagnosis of TLE. When TLE is suspected, a full-body skin examination should be conducted [4]. Because TLE prefers the face, neck, chest, and back, these body parts should be given specific attention. Edematous, typically circular plaques with erythematous to violaceous colors appear as lesions. TLE lesions can last for days or weeks and recur frequently. Lesions can remit on their own; however, a person may experience recurrence during the summer months [5].

## Treatment

Affected patients should be reassured about the condition's benign nature and its uncommon link to SLE. TLE patients are encouraged to apply sunscreens with high UVA and UVB protection regularly and to participate in smoking cessation programs because their disease is linked to UV radiation and smoking [2]. The first-line topical anti-inflammatory therapy for TLE is topical corticosteroids. They are administered twice a day for one to two weeks and then decreased in the next two weeks, depending on their efficacy. Topical calcineurin inhibitors are approved for the treatment of atopic eczema, but because of their anti-inflammatory properties and favorable side effect profile, they are commonly utilized as steroid-sparing medicines for other purposes [1].

Antimalarial therapy is the gold standard in the treatment of CLE and SLE, as well as the first-line systemic therapy for TLE. Because of their well-known negative effects, systemic corticosteroids are rarely used in the treatment of TLE. For severe, aggravated illness, steroid pulse therapy could be tapered and stopped after 4-8 weeks [5].

## CONCLUSION

TLE is an uncommon clinical form of persistent cutaneous lupus erythematosus that manifests as photosensitive erythematosus and edema with small development to systemic lupus erythematosus. TLE lesions might be difficult to distinguish from other cutaneous conditions such as urticarial vasculitis and

**Correspondence to:** Sehnaz Sheikh, Department of Allergy and Rheumatology, National University, Gazipur, Bangladesh, E-mail: sheikh231987@gmail.bd

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lymphocytic infiltration. Because it only rarely connects with systemic autoimmune illness and does not cause skin damage, it is considered the most benign form of CLE.

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