

LETTERS TO THE EDITOR

When SLE patients have rosacea – key distinguishing features

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INTRODUCTION

Facial erythema with ANA positivity often leads to Rheumatology referral for acute cutaneous lupus suspicion. Simultaneously, patients with SLE (systemic lupus erythematosus) might present to the clinic with malar rash. In both cases, differential diagnosis with rosacea should be considered given its prevalence in general population and major differences in therapeutic options and follow-up.

We will focus on the distinguishing features between these two entities to aid clinical decision and management.

CLINICAL DIFFERENCES

Rosacea is a chronic skin condition which affects approximately 5% of the world population, being most common in adult women of lighter phototypes (I, II and III Fitzpatrick phototype). It involves mainly the central face (including the nasolabial fold), forehead and chin and presents with flushing, erythema, papules, pustules, telangiectasia and phymatous changes (Figure 1). Ophthalmologic symptoms can also occur, such as redness and tearing. Several triggers have been identified: sun exposure, hot beverages, spicy foods, alcohol, exercise, anxiety and skin barrier disruption. Disease phenotypes can be divided in four different subtypes: erythematotelangiectatic, papulopustular, phymatous (most common in the nose, manifesting with thickened irregular skin) and ocular rosacea¹.

Dermoscopy in rosacea is especially helpful for erythematotelangiectatic disease. In these cases, a characteristic vascular pattern of linear or linear-branching vessels arranged in polygons on a diffuse background

may be observed. In papulopustular rosacea, dermoscopy may show follicular dilatation, follicular plugs, white scales and small papules or pustules². In rosacea patients, dermoscopic examination allows the visualization of Demodex mites in approximately 10% of patients³. However, demodex-related findings should be interpreted cautiously as dermoscopy does not reliably visualize mites directly, but may show indirect signs such as Demodex tails, Demodex follicular openings or whitish amorphous follicular material, which are more suggestive of Demodex proliferation².

SLE is less prevalent than rosacea, affecting 0,3% of people worldwide but also being more frequent in young females. However, a considerable amount of patients with SLE develop acute cutaneous lupus erythematosus (ACLE), therefore, in this specific population, differential diagnosis with rosacea is of particular importance. ACLE presents with skin rash in sun exposed areas, typically a facial rash described as a 'butterfly rash' in the malar area and nasal bridge, sparing nasolabial folds (Figure 2). Sun exposure is a common trigger to ACLE and rosacea however, other factors such as spicy food and alcoholic drinks do not trigger ACLE⁴.

In ACLE, dermoscopy findings are less specific, being described as a multicomponent pattern including an erythematous/pinkish background, polymorphous vessels (dotted, linear or tortuous patterns) white or yellowish scales, follicular plugging, and pigmentary changes. The relative prominence of these features may vary according to lesion activity, disease stage and skin phototype^{5,6}.

Other forms of cutaneous lupus such as subacute cutaneous lupus and chronic cutaneous lupus present with clinical features very distinct from rosacea and they are not taken into consideration for these differential diagnosis purposes^{4,7}.

HISTOPATHOLOGY DIFFERENCES

The histopathological features of rosacea vary according to clinical subtype and lesion stage. Common findings include superficial dermal vascular ectasia or telangiectasia, dermal edema, solar elastosis (accumulation of thickened, basophilic, elastic fibers in the upper dermis, that result from chronic UV damage) and a peri-

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Submitted: 07/04/2026

Accepted: 14/07/2026

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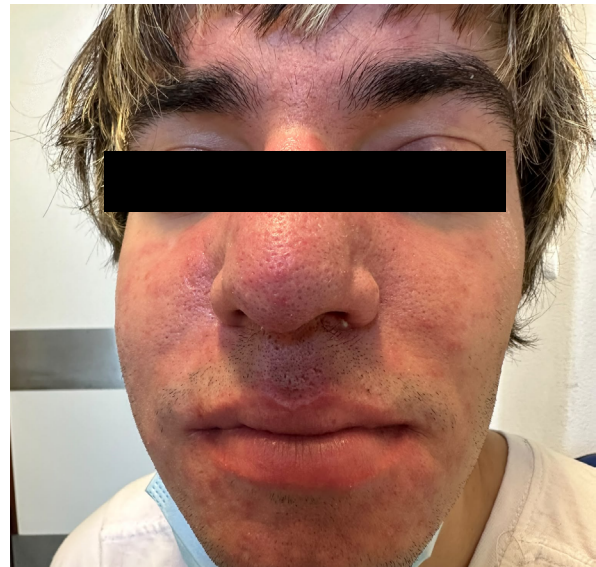


Figure 1. Right - SLE patient with erythematous rash with telangiectasias, typical of rosacea. Left – Patient with papulopustular rash consistent with rosacea.



Figure 2. SLE. Patient with acute cutaneous LE flare sparing the nasolabial fold. Image courtesy of Dr. Nikita Khmelinskii)

follicular and perivascular lymphohistiocytic infiltrate. Papulopustular rosacea more often shows follicular involvement, including follicular spongiosis (intercellular edema in the epidermis), inflammatory cell exocytosis into follicles, neutrophilic folliculitis or follicular pustules, occasionally associated with *Demodex* mites. Epidermal spongiosis, acanthosis (thickening of the skin's epidermis) and parakeratosis (retention of nuclei in the

stratum corneum) may be present but are nonspecific and should not be considered defining features^{8,9}.

ACLE is characterized by vacuolar interface dermatitis, basal vacuolar alteration, scattered apoptotic or dyskeratotic keratinocytes and a superficial and sometimes deep perivascular and periadnexal lymphocytic infiltrate. Dermal edema and increased dermal mucin are important supportive features, while basement membrane thickening may be subtle or absent in acute lesions and is more prominent in chronic forms of cutaneous lupus erythematosus. Although dilated vessels and erythrocyte extravasation may occasionally be encountered, these are not the most diagnostically helpful features^{7,10}.

CONCLUSION

Distinguishing rosacea from ACLE is critical, as these conditions differ fundamentally in pathophysiology and management. Rosacea presents with malar rash involving the nasolabial folds, papules, pustules and telangiectasia associated with several environmental triggers whereas ACLE affects sun exposed areas of the skin, sparing the nasolabial folds and is commonly associated with other systemic symptoms. Histopathology can further support the diagnosis when clinical assessment is inconclusive, particularly when *Demodex* mites are identified in rosacea patients. This distinction has direct therapeutic consequences. Rosacea is managed with topicals and oral antibiotics and trigger avoidance whereas ACLE, while also requires trigger avoidance (mainly photoprotection) requires immunosuppressive treatment (topic and/or systemic) and systemic disease monitoring (Table 1).

TABLE I. Comparison of rosacea and acute cutaneous lupus erythematosus across key diagnostic domains

	Rosacea	ACLE
Epidemiology	~5% world population; adult women; Fitzpatrick phototypes I–III	~0.3% world population; female predominance; any phototype
Distribution	Central face: malar eminences, nose, forehead, chin	Sun-exposed areas, typically malar eminences, nasal bridge (butterfly pattern)
Nasolabial fold involvement	Involved	Spared (key distinguishing feature)
Ocular findings	Ocular rosacea: redness, tearing, blepharitis, keratitis	Periorbital oedema; photophobia; sicca symptoms scleritis
Morphology	Flushing, erythema, papules, pustules, telangiectasia, phymatous changes	Erythematous macular/papular rash; no pustules
Triggers	Sun exposure, hot beverages, spicy food, alcohol, exercise, anxiety, skin barrier disruption	Sun exposure (main trigger); hormonal changes, infections, stress; NOT food/alcohol
Systemic symptoms	Absent	SLE related symptoms such as arthralgia, arthritis, serositis, nephritis, fatigue, fever
Serologic findings	Negative ANA (or low-titre, non-specific); no lupus-specific antibodies	ANA positive (often high-titre); anti-dsDNA, anti-Sm, anti-Ro/SSA, anti-La/SSB may be positive
Dermoscopy	Polygonal linear/linear-branching vessels on erythematous background; follicular plugs; white scales; papules/pustules; <i>Demodex</i> signs (tails, follicular openings)	Multicomponent pattern: pink-red background, polymorphous vessels; white/yellowish scale, keratotic follicular plugging, pigmentary changes
Histology	Vascular ectasia, dermal edema, solar elastosis, perifollicular/perivascular lymphohistiocytic infiltrate; neutrophilic folliculitis or follicular pustules. Epidermal spongiosis and acanthosis are nonspecific.	Vacuolar interface dermatitis, apoptotic keratinocytes, superficial/deep perivascular and periadnexal lymphocytic infiltrate, dermal mucin deposition, basement membrane thickening (more prominent in chronic forms)
Treatment	Topicals (such as metronidazole); oral antibiotics (doxycycline); trigger avoidance; laser for vascular lesions	Photoprotection; topical corticosteroids/calcineurin inhibitors; hydroxychloroquine; systemic immunosuppression for systemic disease (corticosteroids, cDMARD or bDMARD according to organ involvement and disease severity)

Failure to differentiate between these entities may lead to inappropriate treatment either unnecessary immunosuppression or undertreatment of a systemic autoimmune disease.

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