

Toxic Epidermal Necrolysis-Like Cutaneous Lupus in Pediatric Patients: A Case Series and Review

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Bullous eruptions in patients with underlying systemic lupus erythematosus (LE) can mimic toxic-epidermal necrolysis (TEN), a rapidly progressive mucocutaneous reaction usually associated with medication use. Differentiating between classic drug-induced TEN and TEN-like cutaneous LE is important but difficult. We report a series of 3 patients with pediatric systemic LE who were admitted with severe worsening of skin disease resembling TEN. However, the initial photo-distribution of the eruption, subacute progression, limited mucosal involvement, mild systemic symptoms, supportive biopsy and laboratory results, and lack of culprit drugs was more suggestive of a TEN-like cutaneous LE. These patients recovered with various systemic immunosuppressive medications including methylprednisolone, intravenous immunoglobulin, and plasmapheresis. Our cases are rare and demonstrate key clinical and histologic features of TEN-like cutaneous LE in young patients and the importance of differentiating this entity from drug-induced TEN.

Diagnosing bullous eruptions in pediatric patients with systemic lupus erythematosus (SLE) is difficult as the differential diagnosis is broad and includes drug reactions such as toxic-epidermal necrolysis (TEN)/Stevens-Johnson syndrome (SJS), bullous SLE, Rowell syndrome, and TEN-like cutaneous lupus erythematosus (LE).

TEN/SJS are severe idiosyncratic reactions characterized by extensive mucocutaneous necrosis with blistering and systemic symptoms. TEN-like cutaneous LE has been reported in the setting of systemic lupus flares though very few cases have been reported in children.¹

We report 3 cases of TEN-like cutaneous LE in patients with pediatric SLE. Though they were initially thought to have drug-induced TEN/SJS, they were ultimately diagnosed with TEN-like cutaneous

LE, highlighting the importance of recognizing this rare entity.

CASE 1

A 15-year-old white girl was admitted for a 1-month history of progressive rash and fever. She was diagnosed 9 months earlier with SLE, manifested by malar rash, positive antinuclear antibody (ANA), positive anti-double stranded DNA (dsDNA) antibody, and mild leukopenia and anemia, for which she had been treated with hydroxychloroquine and prednisone. She improved with this treatment and had been weaned to 5 mg of prednisone. One month before admission, she developed a photodistributed rash on the face, upper trunk, and extremities, and hydroxychloroquine was discontinued because of concerns for a drug

abstract

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reaction. Hydroxychloroquine was restarted 4 days before admission as her rash was thought to be most consistent with a cutaneous LE flare. On the day of admission, she had acute worsening of cutaneous lesions, bullae, and fever.

Physical examination revealed pink papules and annular plaques with overlying erosions and vesiculation distributed on photoexposed skin (Fig 1 A and B). Conjunctival hyperemia and fibrinous exudates on the lips and hard palate were noted. External genital examination was normal. Laboratory tests were significant for leukocytosis with neutrophil predominance, transaminitis, increasing anti-dsDNA antibody, and decreasing complement proteins 3 (C3) and 4. Due to concerns for drug reaction, medications including hydroxychloroquine were discontinued. Skin biopsy revealed interface dermatitis involving the dermal appendages and individually necrotic keratinocytes (Fig 2A). Direct immunofluorescence (DIF) was positive for lupus band, demonstrating granular staining of the basement membrane with immunoglobulin (Ig) M, IgG, C3, and IgA (Fig 2B). Because this can also be seen in bullous LE, enzyme-linked immunosorbent assay was performed for circulating collagen VII antibodies, and was negative.

Despite treatment with methylprednisolone and dapsone, the patient continued to have extension of the rash to the buttocks, thighs, and back (Fig 1C). Repeat biopsy revealed extensive epidermal necrosis that can be seen with TEN/SJS or TEN-like cutaneous LE (Fig 2C). The patient was given intravenous immunoglobulin G (IVIg) 3 g/kg over 3 days and started on mycophenolate mofetil. Dapsone was discontinued. Our patient slowly improved and was discharged on prednisone and mycophenolate mofetil. One year after



FIGURE 1

A, Patient in case 1 with annular and confluent, erythematous patches with superficial erosions on the photo-exposed areas of the upper chest. B, Flaccid bullae on erythematous scaly patches and plaques on the upper arm in the same patient. C, Extension of the erythematous eruption to the entire back despite treatment with systemic corticosteroids and dapsone in the same patient.

hospitalization, hydroxychloroquine was resumed without incident.

CASE 2

An 11-year-old Asian girl was admitted with worsening skin eruption over the preceding month. She had a 4-month history of SLE with positive ANA, positive anti-dsDNA antibody, positive

anti-Smith antibody, positive antiribonucleoprotein antibody, hypocomplementemia, acute cutaneous lupus, oral ulcers, and class III lupus nephritis. Prednisone and hydroxychloroquine were started 4 months before admission and mycophenolate mofetil was started 1 month prior.

On admission, she had confluent, bright pink, scaly, annular plaques

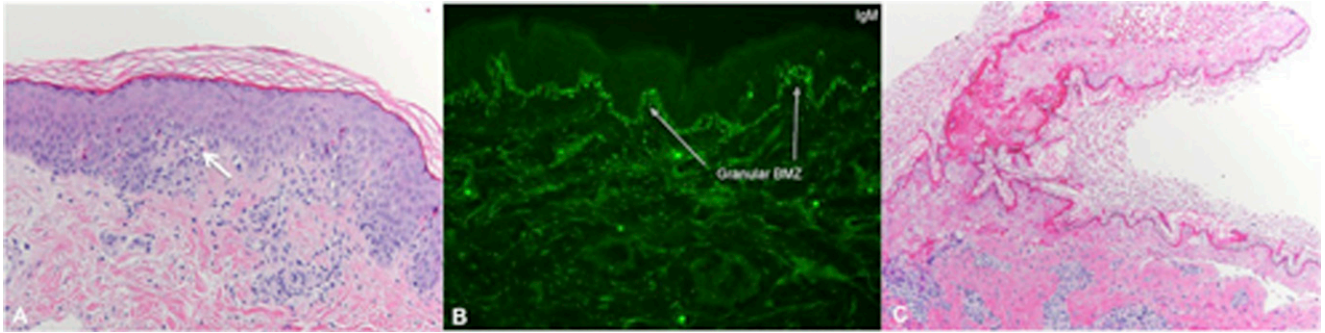


FIGURE 2

A, Skin biopsy of patient in case 1 demonstrating vacuolar interface dermatitis along the basement membrane (white arrow) and scattered necrotic keratinocytes consistent with cutaneous lupus (hematoxylin and eosin, 20×). B, Granular staining of the basement membrane zone with IgM demonstrating a positive lupus band on DIF. C, Full thickness and diffuse epidermal necrosis with epidermal blistering (hematoxylin and eosin, 20×).

on sun-exposed areas of the head, neck, upper trunk, and arms. Erosions were present on the neck, back, shoulders, lips, and vulva. Her laboratory studies revealed

transaminitis, increasing anti-dsDNA antibody, and decreasing C3 and complement protein 4. Due to concerns for possible drug reaction, hydroxychloroquine

and mycophenolate mofetil were discontinued.

Despite high-dose methylprednisolone, she continued

TABLE 1 Summary of Cases of SJS/TEN-like LE in Pediatric Patients

Author	Age, y/ Gender	Photo- distribution	Mucous Membrane Involvement	Previous LE Diagnosis	Pertinent Laboratory Abnormalities	Histology	Therapy
Yu	15/girl	Yes	Lips	Yes	ANA 1:1280, increasing anti-dsDNA, decreasing complement levels, anemia	Extensive epidermal necrosis and subepidermal clefting. Interface dermatitis around pilosebaceous unit.	Systemic steroids, intravenous IgG, mycophenolate mofetil, dapsone
Yu	11/girl	Yes	Lips, ocular, vaginal	Yes	ANA 1:1280, anti-RNP positive, anti-Smith positive, increasing anti-dsDNA, decreasing complement levels, anemia, transaminitis	Not available	Systemic steroids, intravenous IgG, plasmapheresis
Yu	18/boy	Yes	Lips	Yes	Increasing anti-dsDNA, decreasing complement levels, anemia, thrombocytopenia, transaminitis, elevated amylase and lipase	Extensive epidermal necrosis with sparse perivascular and interface lymphocytic dermatitis	Systemic steroids, mycophenolate mofetil
Lee et al ²	12/girl	Yes	Oral, lips, ocular	No	ANA >1:800, anti-dsDNA positive (trend unknown), anti-Smith positive, anti-Ro/SSA positive, anti-La/SSB positive, anti-RNP positive, hypocomplementemia (trend unknown), leukopenia, thrombocytopenia	Basal vacuolization with necrotic keratinocytes with perivascular lymphocytic infiltrate	Systemic steroids and hydroxychloroquine
Jang et al ³	16/girl	Yes	Lips	No	ANA 1:640, anti-dsDNA positive (trend unknown), anti-Ro/SSA positive, anti-Smith positive, hypocomplementemia (trend unknown), leukopenia, anemia	Subepidermal clefting with interface dermatitis and marked necrosis of suprabasal keratinocytes	Systemic steroids, topical steroids and hydroxychloroquine

RNP, ribonucleoprotein.

TABLE 2 Diagnostic Features of the Differential for Epidermal Necrosis in SLE

	TEN	SJS/TEN-like Cutaneous LE	Bullous LE	Rowell Syndrome
Clinical	Flu-like prodrome; Dusky macules that coalesce; Bullae and sloughing of epidermis	May have preceding diagnosis of lupus; Bullae and sloughing of epidermis	Tense vesicles and bullae on sun-exposed areas	Features of DLE; Targetoid macules with centripetal spreading
Nikolsky sign	Positive	Positive or negative	Negative	Negative
Mucous membrane involvement	Severe	Less severe and predominantly oral mucosa	Sometimes	Sometimes
Serology (ANA, anti-dsDNA, anti-Ro/La, RF)	Negative	ANA positive; Anti-dsDNA positive (often); Anti-Ro/SSA or La/SSB positive (often); RF negative	ANA positive; RF negative	ANA positive (speckled); Anti-Ro/SSA or La/SSB positive; RF positive
Histopathology	Full thickness epidermal necrosis with sparse superficial lymphocytic inflammatory infiltrate	Full thickness epidermal necrosis with sparse superficial lymphocytic inflammatory infiltrate; Features of interface dermatitis	Vacuolar degeneration of basal layer resulting in subepidermal blister with predominantly neutrophilic infiltrate	Necrotic keratinocytes with subepidermal blistering and a sparse lymphocytic infiltrate consistent with erythema multiforme
IF	Negative	Direct: May show granular IgM, IgG, and/or C3 binding at the BMZ (lupus band); Indirect: Negative	Direct: Linear or granular IgG, C3, IgM, and IgA binding at BMZ; Indirect: Collagen VII antibodies binding on the dermal side of the BMZ	Negative
Drug etiology	Most cases	No	Some cases	No
Course	Evolves over 3–5 d and heals over 3–4 wk. Scarring may occur. Mortality up to 30%–40%.	Subacute course with rapid improvement within 1–2 wk after treatment	Rapid response to dapsone treatment with cessation of blister formation within 1–2 d. May have intermittent exacerbations	May recur over many years

BMZ, basement membrane zone; IF, immunofluorescence; RF, rheumatoid factor. Adapted from Ryan et al⁴ and Mandelcorn et al.¹ Altered with permission from Ryan E, Marshman G, Astill D. Toxic epidermal necrolysis-like subacute cutaneous lupus erythematosus. *Australas J Dermatol.* 2012;53(4):305.

having fevers, and she developed bilateral eye drainage, photophobia, and increased skin involvement and tenderness. IVIG 3 g/kg (total) was given but she continued to worsen. She developed *Staphylococcus aureus* infection of the eyes and skin, for which she was treated with appropriate antimicrobial agents.

Given the TEN-like features, a diagnosis of TEN-like cutaneous LE was rendered. Plasmapheresis was started, and she steadily improved with reepithelialization of denuded epidermis. She was restarted on hydroxychloroquine 1 year later without recurrence of the TEN-like cutaneous LE.

CASE 3

An 18-year-old Asian man with a 2-year history of SLE manifested by discoid lupus erythematosus (DLE), lupus nephritis, lupus cerebritis,

Coombs-positive hemolytic anemia, and myocarditis was admitted for fever, malaise, myalgias, and worsening diffuse rash. This occurred 2 weeks after receiving clindamycin for wisdom teeth extraction. The rash started on the face and progressed to the trunk and extremities. Physical examination revealed hemorrhagic erosions and crusting on the lips, soft palate, scalp, and face, with trace injection of conjunctiva. Violaceous papules and plaques with flaccid vesicles were seen on the trunk and extremities without involvement of the genital mucosa. Laboratory testing was significant for decreasing C3, increasing anti-dsDNA antibody, mild leukopenia, anemia, transaminitis, elevated creatine kinase, and elevated amylase and lipase.

Skin biopsies demonstrated subepidermal blister with full thickness epidermal necrosis

and interface dermatitis most consistent with TEN-like cutaneous LE. Methylprednisolone and mycophenolate mofetil were administered with substantial systemic and cutaneous improvement and normalization of laboratory values. He was discharged on oral clindamycin for *S aureus* skin infection without subsequent flare in the skin eruption.

Four months after discharge, he was admitted again with diffuse erythematous plaques with central erosions and hemorrhagic crusting, milder but similar to previous flare. There were no recent changes in his medications to suggest a drug reaction. Interestingly, before the initial SLE diagnosis, he reportedly had a similar mucocutaneous eruption that was initially diagnosed as SJS. As his course evolved, he was ultimately diagnosed with SLE.

TABLE 3 Revised Diagnostic Criteria for Rowell Syndrome

Major Criteria (Must Fulfill All)	
1. Presence of CCLE (DLE or chilblain lupus)	
2. Presence of EM-like lesions (typical or atypical)	
3. At least 1 positivity among speckled ANA, anti-Ro/SSA, and anti-La/SSB antibodies	
4. Negative DIF on EM-like lesions	
Minor Criteria (Must Fulfill at Least 1)	
1. Absence of infectious or pharmacologic triggers	
2. Absence of typical EM locations (acral and mucosal)	
3. Presence of at least 1 American Rheumatism Association criterion for diagnosis of SLE besides discoid rash and ANA and excluding photosensitivity, malar rash, and oral ulcers	

CCLE, chronic cutaneous lupus erythematosus; EM, erythema multiforme. Reprinted with permission from Torchia D, Romanelli P, Kerdel FA. Erythema multiforme and Steven-Johnson syndrome/ toxic epidermal necrolysis associated with lupus erythematosus. *J AM Acad Dermatol*. 2012;67(3):421.

TABLE 4 Histopathological Features of LE and TEN/SJS

	LE	TEN/SJS
Epidermal	Ortho-/hyperkeratosis, atrophic epidermis, few necrotic keratinocytes at the dermo-epidermal junction, vacuolar degeneration of the basal keratinocytes, thickened basement membrane zone	Necrotic keratinocytes throughout, minimal vacuolar degeneration of basal keratinocytes
Dermal	Moderate to dense, superficial and deep lymphocytic infiltrate, plasma cells, interstitial mucin	Sparse, superficial lymphocytic infiltrate
Adnexal involvement	Periadnexal lymphocytic infiltrate, vacuolar degeneration of hair follicle infundibulum with few necrotic keratinocytes	Few to many necrotic keratinocytes without significant inflammatory infiltrate

Reprinted with permission from Ziemer M, Kardaun SH, Liss Y, Mockenhaupt M. Stevens-Johnson syndrome and toxic epidermal necrolysis in patients with lupus erythematosus: a descriptive study of 17 cases from a national registry and review of the literature. *Br J Dermatol*. 2012;166(3):577.

DISCUSSION AND REVIEW OF THE LITERATURE

TEN-like cutaneous LE in pediatric SLE is rare. We have described 3 additional cases similar to others previously reported (Table 1). This form of cutaneous LE can be difficult to differentiate from the other blistering dermatoses occurring in the setting of SLE including TEN/SJS, bullous SLE, and Rowell syndrome (Table 2).

TEN/SJS is an acute vesiculobullous reaction characterized by rapidly progressive, painful mucocutaneous erosions, widespread flaccid bullae, epidermal necrosis, and prominent systemic symptoms. It is triggered by a medication in 80% to 95% of cases.^{1,5} Latency of drug exposure to onset of TEN/SJS is usually within 3 weeks, and sooner with previous exposure. TEN/SJS occurs at a higher

frequency in patients with immune-altering diseases such as pediatric SLE (1%–2%).^{6,7}

Bullous SLE⁸ and Rowell syndrome^{9,10} are rare manifestations of lupus, particularly in children. In previous reports, the terms bullous SLE and Rowell syndrome may have been used less specifically to describe any blistering dermatoses in SLE.^{9,11,12} More recently, Rowell syndrome diagnostic criteria have been revised to clarify Rowell syndrome from other variants of LE and erythema multiforme (Table 3).¹³

Similar to the 4 cases of TEN-like LE in adults presented by Ziemer et al,¹⁴ our cases demonstrated clinical features of LE that led us to this diagnosis. All of our patients had an existing diagnosis of SLE and all initially developed a photodistributed annular eruption resembling

cutaneous LE, which progressed to diffuse epidermal necrosis subacutely over the course of weeks to months. Mild mucous membrane involvement was observed with mild lip erosions predominating, though case 2 also demonstrated conjunctivitis and genital erosions. The onset of worsening cutaneous eruption also coincided with decreasing complement levels and increasing anti-dsDNA antibodies, consistent with worsening SLE. All patients had mild fevers, myalgias, and malaise. Cases 1 and 2 had mild transaminitis; case 3 had significant transaminitis. Overall, the degree of systemic involvement is milder than in drug-induced TEN.

Our cases also demonstrated histopathologic features of LE, which can be helpful in differentiating from TEN/SJS (Table 4). Interface dermatitis present on an early biopsy in case 1 and in case 3 helped with the diagnosis of cutaneous LE. Later biopsies in cases 1 and 3 were less helpful, demonstrating full thickness epidermal necrosis characteristic of TEN/SJS.

Though a drug reaction was considered in all cases and nonessential drugs were discontinued, rechallenge with the potential etiologic drugs did not elicit a repeat reaction, as would be expected in TEN/SJS. There are reports of hydroxychloroquine-induced TEN,^{15,16} which was a concern in cases 1 and 2 leading to its initial discontinuation, although both patients have had subsequent reintroduction of hydroxychloroquine without incident. Differentiating drug-induced TEN/SJS in patients with LE from TEN-like cutaneous LE may be difficult, and it is possible that some previously reported cases may have been misclassified.^{14,17–19}

Of the cases we included here and reviewed from the literature, no patients died as a result of his or her

illness compared with a mortality rate of 7.5% reported in pediatric TEN.²⁰ In a series of 4 adult patients with TEN-like cutaneous lupus, 2 died of their disease,¹⁴ whereas neither of the 2 previously reported pediatric cases were fatal.^{2,3} Although the small number of reported patients limits conclusions, it may be that pediatric patients with TEN-like cutaneous LE have a lower mortality compared with children with drug-induced TEN/SJS. Therapy for TEN-like cutaneous LE eruption has not been well studied. In the cases reviewed, parenteral corticosteroids, IVIG, and plasmapheresis were effective.

We describe 3 pediatric cases of TEN-like cutaneous LE associated with SLE. The paucity of significant systemic symptoms, lack of recurrence with drug rechallenge, and underlying flaring SLE distinguishes these cases of TEN-like cutaneous LE from drug-induced TEN/SJS. Prompt recognition with early biopsy of lesions in TEN-like cutaneous LE is important to prevent inappropriate diagnosis of drug allergy and allow early institution of appropriate treatment.

ABBREVIATIONS

ANA: antinuclear antibody
 C3: complement protein 3
 DIF: direct immunofluorescence
 DLE: discoid lupus erythematosus
 dsDNA: double stranded DNA
 Ig: immunoglobulin
 IVIG: intravenous immunoglobulin G
 LE: lupus erythematosus
 SJS: Stevens-Johnson syndrome
 SLE: systemic lupus erythematosus
 TEN: toxic-epidermal necrolysis

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