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# Hypertrophic Discoid Lupus Preceding the Diagnosis of Systemic Lupus Erythematosus With Active Lupus Nephritis: A Case Report

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## Abstract

Systemic lupus erythematosus (SLE) is a systemic autoimmune disease with heterogeneous cutaneous and visceral manifestations. We report the case of a 26-year-old Japanese woman with SLE who initially presented with a diffuse eruption resembling Kaposi varicelliform eruption and was subsequently found to have severe organ involvement, including active lupus nephritis. The patient had a history of atopic dermatitis, and the eruption was initially treated as Kaposi varicelliform eruption; however, the lesions continued to worsen. The subsequent development of arthralgia, alopecia, and fatigue prompted further investigation, which revealed elevated anti-DNA antibody levels, hypocomplementemia, and newly detected proteinuria. Renal biopsy confirmed active lupus nephritis. Re-evaluation of the cutaneous lesions and the skin biopsy revealed marked hyperkeratosis, epidermal thickening, elongated rete ridges, basal vacuolar degeneration, and lymphocytic infiltration, while direct immunofluorescence demonstrated granular IgA and IgM deposition along dermal vessels. These clinicopathological findings, together with the newly established diagnosis of SLE, supported the diagnosis of hypertrophic lupus, the hypertrophic variant of discoid lupus. Treatment with glucocorticoids, mycophenolate mofetil, voclosporin, and hydroxychloroquine led to remission of the systemic manifestations, including lupus nephritis, with parallel improvement of the skin lesions. This case illustrates that the hypertrophic variant of discoid lupus can mimic other dermatological disorders and delay recognition of systemic disease. Careful assessment of extracutaneous findings is therefore important when evaluating atypical or treatment-refractory skin lesions.

**Categories:** Rheumatology, Internal Medicine, Allergy/Immunology
**Keywords:** combination therapy, cutaneous lupus erythematosus, differential diagnosis, lupus nepritis, skin eruption, systemic lupus erythematosus

## Introduction

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease characterized by heterogeneous clinical manifestations and multiorgan involvement, ranging from cutaneous and musculoskeletal symptoms to renal, hematologic, and central nervous system disorders. Among these diverse features, cutaneous manifestations represent one of the most frequent and clinically informative components of the disease. Skin lesions not only contribute substantially to patient morbidity but also serve as important diagnostic clues, often preceding or accompanying systemic involvement.

The spectrum of cutaneous findings in SLE is remarkably broad. Well-recognized lesions such as malar rash, chilblain lupus, and Raynaud's phenomenon are commonly encountered and readily prompt consideration of SLE in clinical practice. However, SLE may also present with less typical or subtle cutaneous abnormalities [1]. For example, annular erythema is frequently observed in patients with anti-SS-A antibodies, while livedo reticularis has been associated with antiphospholipid antibodies. In addition, hypertrophic lupus, a rare hyperkeratotic variant of discoid lupus erythematosus (DLE), can mimic other hyperkeratotic dermatoses and may therefore be overlooked in the early stages. These atypical manifestations may resemble other dermatologic or autoimmune conditions, complicating the diagnostic process and potentially delaying referral to a rheumatologist.

The diagnostic challenge posed by such non-classical skin findings is clinically significant. Because SLE can lead to organ-threatening or life-threatening complications, including lupus nephritis, neuropsychiatric lupus, and severe hematologic abnormalities, timely recognition and accurate classification are essential for initiating appropriate immunosuppressive therapy and preventing irreversible organ damage. Therefore, careful evaluation of cutaneous lesions, including those outside the typical spectrum, is crucial for early diagnosis and optimal disease management.

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## Case Presentation

A 26-year-old woman with a history of atopic dermatitis noticed worsening of a skin eruption. A month later, Kaposi varicelliform eruption (KVE) associated with atopic dermatitis was clinically diagnosed at an outside hospital. Herpes simplex virus (HSV) polymerase chain reaction (PCR) or viral culture from skin lesions was not performed, and only HSV IgM serologic testing was obtained. Valacyclovir was administered empirically; however, the eruption failed to improve. Because lesion-based virological confirmation was unavailable, KVE could not be definitively excluded at that stage.

Approximately two weeks after the initial diagnosis, she was referred to the Department of Dermatology at our hospital. A skin biopsy was performed, but no definitive diagnosis was established. Histopathological examination showed marked hyperkeratosis and dyskeratosis, epidermal thickening with spongiosis, elongated rete ridges, and vacuolar degeneration of the basal layer, accompanied by lymphocytic infiltration around small dermal vessels. These findings were atypical and not diagnostic at the time.

Four months after the referral to the Department of Dermatology, the development of arthralgia, alopecia, and fatigue prompted blood testing, which revealed an elevated anti-DNA antibody level (radioimmunoassay (RIA) 130 IU/mL). Until that time, the skin lesions had been managed with topical glucocorticoids alone. She was therefore admitted to the Department of Rheumatology for further evaluation.

On admission, her temperature, blood pressure, and pulse rate were within normal ranges. Numerous well-demarcated, elevated, scaly papules, approximately the size of a fingernail, were distributed over almost the entire body, including the face, ears, and limbs, with a predominance in sun-exposed areas (Figure *1A*, *1B*). Multiple erosions and hemorrhagic crusts were present on the lips and oral mucosa (Figure *1C*). Her atopic dermatitis was stable, with no pruritus or excoriations. Bilateral symmetrical lower-leg edema and predominantly lower-extremity arthralgia were also noted.

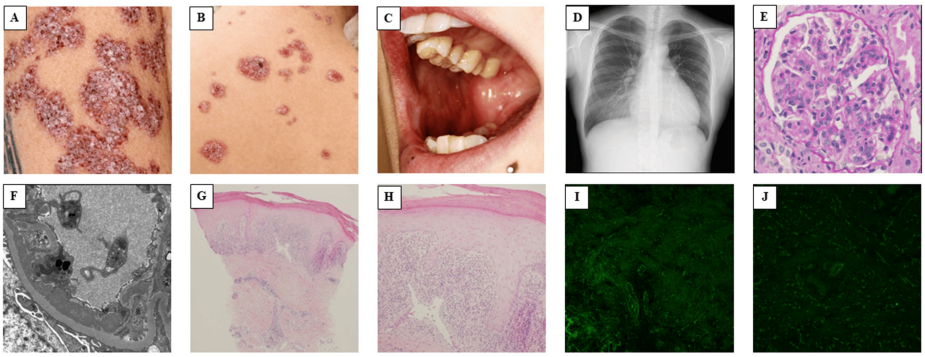



**FIGURE 1: Clinical findings**

(A, B) Clinical appearance showing “oyster-shell-like” (rupioid) hyperkeratotic eruptions on the forearms (A) and back (B).

(C) Oral mucosal involvement showing multiple erosions and hemorrhagic crusts on the lips and oral cavity.

(D) Chest radiograph demonstrating cardiomegaly.

(E, F) Renal biopsy showing marked mesangial proliferation and lobulation on periodic acid–Schiff (PAS) staining (periodic acid–Schiff stain, original magnification x60 (E), and subendothelial as well as mesangial electron-dense deposits on electron microscopy (electron microscopy, original magnification x3000 (F).

(G, H) Skin biopsy showing marked hyperkeratosis and dyskeratosis of the stratum corneum, epidermal thickening, spongiosis, elongated rete ridges, vacuolar degeneration of the basal layer, and damage to the dermoepidermal junction (hematoxylin and eosin stain, original magnification x4 (G) and x10 (H)).

(I, J) Direct immunofluorescence (DIF) of the skin biopsy showing granular immunoglobulin deposits along the walls of small dermal vessels, consistent with immune complex–mediated vascular involvement (direct immunofluorescence, original magnification x100; IgA immunostaining (I), IgM immunostaining (J)).

Laboratory testing showed lymphopenia (698 /μL) and anemia (hemoglobin, 7.9 g/dL). Immunoserological testing demonstrated that the anti-DNA antibody level (RIA: 200IU/mL) had further increased compared with the value obtained at the prior outpatient visit, along with elevated anti-ribonucleoprotein (RNP) (1970U/mL) and anti-Smith (Sm) (44.3U/mL) antibodies and hypocomplementemia (CH50, 7/mL; C3, 16mg/dL; C4, 3mg/dL). Anticardiolipin IgG antibodies were positive, and repeat testing 14 weeks later also remained positive. There were no Raynaud’s symptoms or puffy fingers suggestive of mixed connective tissue disease, and no evidence of thrombosis suggestive of antiphospholipid syndrome was detected on

lower-limb venous ultrasonography or brain magnetic resonance angiography. No history of pregnancy complications was reported. Although the estimated glomerular filtration rate was preserved, urinalysis showed microscopic hematuria and overt proteinuria, with a urine protein-to-creatinine ratio (UPCR) of 3.88 g/g creatinine (Table *1*).

| Parameter | | Patient Value | Unit | Reference Range |
|---|---|---|---|---|
| Hematology | WBC | 4500 | /μL | 3,300–8,600 |
| | Neutrophils | 3510 | /μL | 1,800–7,000 |
| | Lymphocytes | 697 | /μL | 1,000–4,000 |
| | Eosinophils | 117 | /μL | 50–500 |
| | RBC | 270 × $10^4$ | /μL | 380–500 × $10^4$ |
| | MCV | 89.6 | fL | 83–101 |
| | MCH | 28.4 | pg | 28–34 |
| | Hb | 7.9 | g/dL | 11.6–14.8 |
| | Hct | 24 | % | 35.1–44.4 |
| | Platelets | 16.7 × $10^4$ | /μL | 15.8–34.8 × $10^4$ |
| Coagulation / Fibrinolysis | PT | 100 | % | 70–130 |
| | PT-INR | 0.98 | | 0.85–1.15 |
| | APTT | 28.4 | sec | 24.0–39.0 |
| | D-dimer | 3.57 | μg/mL | < 1.0 |
| Biochemistry & Cardiac Markers | Total bilirubin | 0.4 | mg/dL | 0.4–1.2 |
| | AST | 18 | IU/L | 13–30 |
| | ALT | 6 | IU/L | 7–23 |
| | γ-GTP | 15 | IU/L | 9–32 |
| | BUN | 16 | mg/dL | 8.0–20.0 |
| | Serum creatinine | 0.66 | mg/dL | 0.46–0.79 |
| | eGFR | 89 | mL/min/1.73 $m^2$ | ≥ 60 |
| | CK | 69 | IU/L | 41–153 |
| | Serum iron | 38 | μg/dL | 40–188 |
| | Ferritin | 211 | ng/mL | 5.0–157.0 |
| | TIBC | 232 | μg/dL | 250–450 |
| | Sodium | 138 | mEq/L | 138–145 |
| | Potassium | 4 | mEq/L | 3.6–4.8 |
| | Chloride | 105 | mEq/L | 101–108 |
| | Total protein | 8 | g/dL | 6.6–8.1 |
| | Albumin | 3.4 | g/dL | 4.1–5.1 |
| | NT-proBNP | 2772 | pg/mL | < 125 |
| | CRP | 1 | mg/dL | < 0.14 |
| | RF | < 10 | IU/mL | < 15 |
| | ANA (Speckled pattern) | 1:640 | titer | < 1:40 |
| | Anti-DNA antibody | 200 | IU/mL | < 6.0 |

| | | | | |
|---|---|---|---|---|
| Immunology & Serology | Anti-RNP antibody | 1970 | U/mL | < 22.0 |
| | Anti-Sm antibody | 44.3 | U/mL | < 7.0 |
| | Anti-SS-A antibody | 1 | U/mL | < 10.0 |
| | Anti-cardiolipin antibody-IgG | 41.9 | U/mL | < 10.0 |
| | Lupus anticoagulant (dRVVT) | 0.9 | | < 1.2 |
| | MPO-ANCA | < 1.0 | U/mL | < 3.5 |
| | PR3-ANCA | < 1.0 | U/mL | < 3.5 |
| | CH50 | 7 | /mL | 30.0–45.0 |
| | C3 | 16 | mg/dL | 73–138 |
| | C4 | 3 | mg/dL | 11–31 |
| | IgG | 2567 | mg/dL | 870–1700 |
| | IgA | 295 | mg/dL | 110–410 |
| | IgM | 121 | mg/dL | 33–190 |
| | Haptoglobin | 185 | mg/dL | 19–170 |
| | Direct Coombs test | Positive (+) | | Negative |
| | Indirect Coombs test | Positive (+) | | Negative |
| Urinalysis & Kidney Markers | Urinary pH | 6.5 | | 5.0–7.5 |
| | Urinary RBC | > 100 | /HPF | < 1 |
| | Urinary WBC | 5–9 | /HPF | < 1 |
| | UPCR | 3.88 | g/gCr | < 0.15 |
| | Urinary protein | 3+ | | Negative |
| | Urinary ketone bodies | Negative (-) | | Negative |
| | Urinary occult blood | 3+ | | Negative |
| | Hyaline cast | 1+ | | Negative |
| | Epithelial cast | 1+ | | Negative |
| | Granular cast | 1+ | | Negative |

**TABLE 1: Laboratory findings on the first day of hospitalization**

WBC, white blood cell; RBC, red blood cell; MCV, mean corpuscular volume; MCH, mean corpuscular hemoglobin; Hb, hemoglobin; Hct, hematocrit; Plt, platelet; PT, prothrombin time; APTT, activated partial thromboplastin time; Bil, bilirubin; AST, aspartate aminotransferase; ALT, alanine aminotransferase; γ-GTP, gamma-glutamyl transpeptidase; BUN, blood urea nitrogen; Cre, creatinine; eGFR, estimated glomerular filtration rate; CK, creatine kinase; Fe, iron; TIBC, total iron-binding capacity; Na, sodium; K, potassium; Cl, chloride; TP, total protein; Alb, albumin; NT-proBNP, N-terminal pro-B-type natriuretic peptide; CRP, C-reactive protein; ANA, antinuclear antibody; DNA, deoxyribonucleic acid; RNP, ribonucleoprotein; Sm, Smith; SS, Sjögren syndrome; MPO, myeloperoxidase; ANCA, antineutrophil cytoplasmic antibody; PR3, proteinase 3; CH, hemolytic complement; Ig, immunoglobulin; RF, rheumatoid factor; pH, potential of hydrogen; UPCR, urine protein-to-creatinine ratio.

Chest radiography showed cardiomegaly, with a cardiothoracic ratio of 56.3% (FIGURE *1D*). Echocardiography demonstrated a reduced left ventricular ejection fraction of 48%, diffuse wall thickening, and pericardial effusion. Although coronary angiography was not performed, no ST-T abnormalities were observed on electrocardiography, and serum levels of creatine kinase (CK), CK-MB, and troponin T remained within the normal range; therefore, the likelihood of coronary ischemia was considered low. Serological testing for acute viral infection was negative. Renal biopsy showed marked mesangial proliferation and lobulation by light microscopy, with endocapillary hypercellularity with leukocyte infiltration and substantial luminal reduction indicating active inflammation. Electron-dense deposits were seen in the subendothelial and mesangial regions by electron microscopy, and a full-house immunofluorescence pattern (IgG, IgA, IgM, C3, and C1q) was present (Figure *1E*, *1F*). Chronic lesions were limited, with focal global glomerulosclerosis observed in a small subset of glomeruli. No crescents or necrotic lesions were identified.

These findings were consistent with diffuse lupus nephritis (International Society of Nephrology (ISN)/Renal Pathology Society (RPS) class IV(A)). The patient fulfilled the 2019 European Alliance of Associations for Rheumatology (EULAR)/ American College of Rheumatology (ACR) classification criteria for SLE [2].

The cutaneous lesions consisted of well-demarcated, elevated, “oyster-shell-like” plaques with scaling that were distributed widely over the body. On re-examination of the skin biopsy specimen, the stratum corneum showed marked hyperkeratosis and dyskeratosis, with epidermal thickening and spongiosis. The rete ridges were elongated, with vacuolar degeneration of the basal layer, damage to the dermoepidermal junction, and lymphocytic infiltration around small vessels and within the superficial dermis (Figure *1G*, *1H*). Direct immunofluorescence showed mild IgA and IgM deposition along vascular walls without lupus band formation (Figure *1I*, *1J*). These vascular immune deposits were interpreted as evidence of systemic immune-complex activity associated with SLE rather than as findings specific to cutaneous lupus. Because the atopic dermatitis was clinically stable, pathological changes related to prurigo or scratching were considered unlikely to account for these findings. Moreover, the prominent liquefactive degeneration observed in the basal layer further supported that the lesions were inconsistent with those typically seen in atopic dermatitis. No characteristic features of psoriasis, such as elongation of regular epidermal projections or loss of the granular layer, were observed. In contrast, several findings suggestive of lichen planus-including irregular, serrated epidermal thickening, partial granular layer thickening, liquefactive degeneration of the basal layer, and band-like lymphocytic infiltration at the epidermal-dermal junction-were prominent, and therefore lichen planus could not be excluded. In the context of the newly established diagnosis of SLE, re-evaluation of the clinical and histopathological findings supported a diagnosis of hypertrophic lupus, the hypertrophic variant of DLE, which was considered the most compatible explanation for the patient’s cutaneous lesions.

A three-day course of intravenous methylprednisolone (mPSL) pulse therapy at 500 mg/day was initiated on hospital day 1, followed by oral prednisolone (PSL) at 25 mg/day (0.5 mg/kg) from hospital day 4. Hydroxychloroquine was added as standard therapy on hospital day 14. Given the presence of active lupus nephritis together with hematological and other organ manifestations, we selected triple therapy with glucocorticoids, mycophenolate mofetil, and voclosporin, primarily to treat the nephritis. After completion of mPSL pulse therapy, the UPCR decreased from 3.88 to 0.76 g/g creatinine. Anti-DNA antibody levels also declined; however, C3 and C4 remained low. Because persistent hypocomplementemia was considered to suggest ongoing serological activity despite improvement in other disease parameters, PSL was converted to betamethasone on hospital day 31. By hospital day 38, complement levels had normalized. Thereafter, betamethasone was converted back to PSL to facilitate standard glucocorticoid tapering and long-term dose adjustment. The UPCR subsequently became undetectable. The patient was discharged on hospital day 46 (Figure *2*).

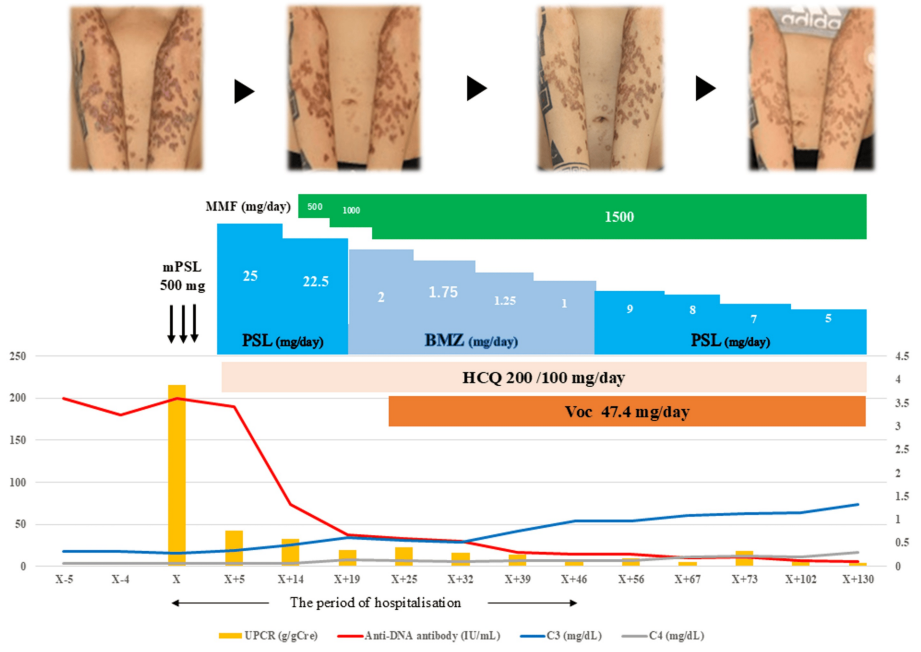



**FIGURE 2: Clinical course**

MMF, mycophenolate mofetil; mPSL, methylprednisolone; PSL, prednisolone; BMZ, betamethasone; HCQ, hydroxychloroquine; Voc, voclosporin; UPCR, urine protein-to-creatinine ratio.

After discharge, no recurrence of organ involvement, including lupus nephritis, was noted, and glucocorticoids were tapered without difficulty. Five months after discharge (seven months after treatment initiation), repeat echocardiography showed improvement in the left ventricular ejection fraction to 63%. As systemic disease activity came under control, the skin lesions also improved, leaving only residual hyperpigmentation. Glucocorticoids were discontinued eight months after discharge (approximately 10 months after treatment initiation), and the hypertrophic cutaneous lupus lesions continued to resolve.

## Discussion

The atypical cutaneous presentation in this patient delayed the diagnosis of SLE by approximately six months. The cutaneous presentation in this patient initially obscured the diagnosis of SLE. Importantly, hypertrophic lupus represents a variant of chronic cutaneous lupus erythematosus (CLE) and is pathologically characterized by marked hyperkeratosis, epidermal hyperplasia, and interface dermatitis. These histopathological features are distinct from the cutaneous manifestations of SLE, which are primarily clinical phenomena such as malar rash, photosensitivity, or vasculitic lesions and do not necessarily correspond to the histological pattern of CLE [1]. In this case, the skin biopsy demonstrated both CLE-consistent interface changes and granular immunoglobulin deposition along small dermal vessels. We interpreted the latter as evidence of systemic immune-complex-mediated vascular involvement rather than as a defining feature of cutaneous lupus. Recognizing this distinction was essential for integrating the cutaneous findings with the patient's evolving systemic symptoms.

In this patient, the hypertrophic discoid lupus lesions occurred in the setting of SLE and therefore represent cutaneous involvement associated with systemic disease rather than primary CLE. Although a skin biopsy had been performed early in the clinical course, the initial pathological assessment did not establish a diagnosis of CLE. Recognition of extracutaneous symptoms, particularly arthralgia and fatigue, prompted investigation for systemic autoimmune disease and ultimately led to the diagnosis of SLE. Subsequent re-evaluation of the skin biopsy showed vascular immunoglobulin deposition without lupus band formation, a finding compatible with immune complex-mediated vascular involvement in SLE [3]. Because the atopic dermatitis was stable, changes related to prurigo or scratching were considered unlikely to explain the biopsy findings. Taken together with the clinical morphology, these findings supported the diagnosis of hypertrophic lupus.

Hypertrophic lupus was first described by Bechet as a variant of DLE [4]. DLE is associated with ultraviolet exposure and smoking, and it is primarily observed in African American populations [5]. The hypertrophic form is uncommon, accounting for approximately 2% of chronic CLE, and appears to be particularly rare in Asian populations [6]. Histopathologically, hypertrophic lupus is characterized by prominent hyperkeratosis, epidermal hyperplasia, interface damage, and inflammatory cell infiltration [7,8]. Clinically and histopathologically, however, hypertrophic lupus can resemble other disorders, including KVE, lichen planus, and keratoacanthoma [7,8]. In this case, re-evaluation of the cutaneous lesions in the context of the newly established diagnosis of SLE provided essential diagnostic context for recognizing hypertrophic lupus. This diagnostic challenge highlights the importance of recognizing extracutaneous manifestations and considering autoimmune evaluation when atypical cutaneous lesions are accompanied by abnormal laboratory findings, including hematological abnormalities or urinary abnormalities.

Evidence regarding the optimal treatment of hypertrophic lupus remains limited. Ocon et al. reported marked improvement of refractory hypertrophic DLE following treatment with anifrolumab [9]. Although this observation suggests that anifrolumab may represent a therapeutic option for hypertrophic lupus, our patient had active class IV lupus nephritis; therefore, treatment selection was primarily guided by current lupus nephritis recommendations. Because active class IV lupus nephritis was considered the manifestation with the greatest immediate prognostic importance, guideline-supported triple therapy including voclosporin was selected [10-12].

Following treatment, lupus nephritis and other organ manifestations improved markedly, and the hypertrophic cutaneous lesions gradually resolved in parallel with overall disease control. Although evidence supporting conversion from PSL to betamethasone in SLE is limited, persistent hypocomplementemia despite improvement in other disease activity markers was considered to suggest residual immunological activity. PSL was therefore temporarily replaced with betamethasone. Betamethasone, a long-acting glucocorticoid with a longer biological half-life than PSL, was selected with the expectation that it might provide more sustained immunosuppressive activity [13-15]. Once complement levels normalized, PSL was resumed to allow gradual dose reduction according to standard tapering practice, and sustained remission was subsequently achieved.

## Conclusions

This case highlights hypertrophic lupus as a rare and diagnostically challenging cutaneous manifestation occurring in association with SLE. The presence of atypical, treatment-refractory skin lesions together with emerging extracutaneous manifestations prompted further evaluation, leading to the diagnosis of active lupus nephritis and systemic disease. Careful integration of dermatologic, serological, and systemic findings is therefore essential to avoid diagnostic delay and to enable timely management of organ-threatening

complications.

# Additional Information

## Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

**Concept and design:** Satoshi Suzuki, Hinaho Arai

**Acquisition, analysis, or interpretation of data:** Satoshi Suzuki, Hinaho Arai, Tomoya Otani, Ayuka Orimo, Marina Shinoura, Takuya Nishi, Akane Hashizume, Keigo Ikeda, Shinji Morimoto

**Drafting of the manuscript:** Satoshi Suzuki, Hinaho Arai

**Critical review of the manuscript for important intellectual content:** Satoshi Suzuki, Hinaho Arai, Tomoya Otani, Ayuka Orimo, Marina Shinoura, Takuya Nishi, Akane Hashizume, Keigo Ikeda, Shinji Morimoto

**Supervision:** Shinji Morimoto

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