

Cutaneous Lupus Erythematosus/Lichen Planus Overlap Syndrome With Vitiligo-Like Depigmentation: A Case Report

Review began 09/21/2026

Review ended 10/10/2026

Published 10/18/2026

© Copyright 2026

Alkandari et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.118102

Danah Alkandari ¹, Hasan KH A H A Ashkanani ², Hawraa Mayouf ³, Ali Alajmi ⁴, Humoud Al-Sabah ⁴

1. Dermatology, Jaber Al-Ahmad Hospital, Kuwait City, KWT 2. Dermatology, Amiri Hospital, Kuwait City, KWT 3. Dermatology, Mubarak Al-Kabeer Hospital, Kuwait, KWT 4. Dermatology, As'ad K. Al-Hamad Dermatological Center, Sulaibikhat, KWT

Corresponding author: Danah Alkandari, danah-98@hotmail.com

Abstract

Cutaneous lupus erythematosus/lichen planus (CLE/LP) overlap syndrome is an uncommon interface dermatosis with features of both disorders. We describe a 60-year-old woman who initially developed pruritic, erythematous, scaly lesions on the dorsal hands, and facial erythema. The initial eruption resolved spontaneously within two months, without treatment, followed by persistent depigmented and erythematous patches at the previously affected sites. Wood's lamp examination accentuated the depigmentation. Histopathology of a depigmented dorsal-hand patch showed interface dermatitis with basal vacuolar alteration and a bandlike lymphocytic infiltrate. Melan-A staining on the same specimen demonstrated melanocyte loss. Direct immunofluorescence of perilesional skin showed immunoglobulin deposition at the dermoepidermal junction and shaggy fibrinogen staining. The findings supported CLE/LP overlap syndrome with subsequent vitiligo-like depigmentation. Hydroxychloroquine 200 mg twice daily and once-daily topical ruxolitinib were initiated; mild repigmentation at the oral commissure was documented clinically and with Wood's lamp examination at one month. Distinguishing coexisting vitiligo from post-inflammatory melanocyte loss remains challenging, and the temporal association does not establish causality.

Categories: Dermatology

Keywords: interface dermatitis, lichen planus, lupus erythematosus, overlap syndrome, vitiligo

Introduction

Cutaneous lupus erythematosus (CLE)/lichen planus (LP) overlap syndrome describes lesions exhibiting combined clinical, histopathological, and immunopathological features of CLE and LP, rather than separate lesions of each condition [1-3]. Earlier literature has also documented the coexistence of LE and LP, underscoring the importance of distinguishing coincidental disease from an overlap process [4]. Reported manifestations vary and may include erythematous, violaceous, scaly, or atrophic plaques, often in photoexposed areas. Histopathological interface change and immunoreactant deposition at the dermoepidermal junction may provide complementary evidence, although neither feature is independently specific [2,3]. The lichenoid reaction pattern should also be considered in the broader context of lichen planus and related lichenoid dermatoses [5]. The syndrome is rare; older publications reported only a limited number of cases, but an up-to-date cumulative case count cannot be inferred from those reports [3,6].

Depigmentation following interface dermatitis presents a diagnostic dilemma: it may reflect coexisting vitiligo or melanocyte destruction associated with the antecedent inflammatory process. We report CLE/LP overlap syndrome followed by melanocyte-depleted, vitiligo-like patches and discuss the limits of attributing this finding to a separate autoimmune pigmentary disorder.

Case Presentation

A 60-year-old Iranian woman with Fitzpatrick skin phototype IV, type 2 diabetes mellitus, and hypothyroidism presented with a two-year history of pigmentary changes affecting the face and dorsal hands. She recalled initially developing itchy, red, scaly lesions on the dorsal hands, accompanied by facial redness in the malar region (Figures 1a-1b). The malar erythema was reported retrospectively by the patient and was not independently documented by the treating clinicians during the initial inflammatory phase. The inflammatory lesions resolved spontaneously within approximately two months without medication. Subsequently, persistent depigmented to erythematous patches appeared at the previously affected sites. The patient did not seek medical consultation or receive any treatment during the initial inflammatory episode or the subsequent two-year period before presenting to our dermatology clinic. There was no history of photosensitivity, arthralgia or inflammatory arthritis, oral or nasal ulcers, sicca symptoms, Raynaud's phenomenon, constitutional symptoms, or renal, cardiopulmonary, or neurological symptoms. She reported no family history of autoimmune disease and no relevant medication exposures preceding the eruption. The autoimmune etiology of her hypothyroidism was not established.

How to cite this article

Alkandari D, Ashkanani H, Mayouf H, et al. (October 18, 2026) Cutaneous Lupus Erythematosus/Lichen Planus Overlap Syndrome With Vitiligo-Like Depigmentation: A Case Report. Cureus 18(10): e118102. DOI 10.7759/cureus.118102



FIGURE 1: Clinical progression of skin lesions in a patient with LP-LE overlap syndrome

LP: lichen planus; LE: lupus erythematosus

(a-b) Initial inflammatory phase, showing facial changes and erythematous, scaly plaques on the dorsal hand. The history of malar erythema was reported retrospectively by the patient and was not independently documented on clinical examination at that time. (c-d) Subsequent presentation showing well-demarcated depigmented to erythematous patches involving the face and dorsal hands

On examination, multiple well-defined depigmented to erythematous patches involved the forehead, nasal bridge, upper cutaneous lip, chin, and dorsal hands (Figures 1c-1d). Some residual erythema was present within the pigmentary changes. Scalp, nails, and mucous membranes were unremarkable. Wood's lamp examination showed marked accentuation of the depigmented areas, consistent with substantial pigment loss. Dermoscopy was not performed.

Laboratory investigations (Table 1) demonstrated leukocytosis ($15.79 \times 10^9/L$), thrombocytosis ($472 \times 10^9/L$), elevated erythrocyte sedimentation rate (ESR) (45 mm/h) and C-reactive protein (CRP) (9.71 mg/L), hyperglycemia (17.26 mmol/L), glucosuria, and mildly elevated liver enzymes. Antinuclear antibody (ANA) was positive at 1:80 (coarse speckled), anti-Ro/SSA weakly positive, and the reported lupus anticoagulant clotting time was prolonged (54.3 seconds; laboratory reference: 28.4-46.4). The specific lupus anticoagulant assay and confirmatory testing were not documented; this isolated result should not be interpreted as confirmed lupus anticoagulant positivity. C3 and C4 were normal; anti-dsDNA, anticardiolipin, and anti- β 2-glycoprotein antibodies were negative; and urine protein was negative. No systemic lupus features were elicited at this assessment; this does not definitively exclude future systemic disease.

Test	Result	Units	Reference range
WBC	15.79 (H)	$\times 10^9/L$	4.5-11.0
RBC	4.76	$\times 10^{12}/L$	3.8-4.8
Hemoglobin	128	g/L	120-150
Hematocrit	0.403	L/L	0.360-0.460
MCV	84.7	fL	83-101
MCH	26.09 (L)	pg	27.2-33.5
MCHC	318	g/L	315-345
RDW	13.2	%	11.6-14.6
Platelet	472 (H)	$\times 10^9/L$	150-450
ESR	45 (H)	mm/hr	0-20
Creatinine	86 (H)	$\mu\text{mol}/L$	44-80
Random blood glucose	17.26 (H)	mmol/L	4.1-6.1
eGFR	78.98	mL/min/1.73 m ²	≥ 60
ALT	38 (H)	U/L	1-33
GGT	63 (H)	U/L	5-36
ALP	122 (H)	U/L	46-116
C3	1.42	g/L	0.9-1.8
C4	0.22	g/L	0.1-0.4
C-reactive protein	9.71 (H)	mg/L	0-5
Urine glucose	4+ (H)	-	Negative
Urine protein	Negative	-	Negative
ANA	Positive, 1:80, coarse speckled pattern	-	Negative <1:80
Lupus anticoagulant	54.3 seconds (H)	Seconds	28.4-46.4 seconds
Anti-Ro/SS-A antibody	Weakly positive (H)	-	Negative
anti-dsDNA antibody	Negative	-	Not provided by laboratory
Anti- $\beta 2$ -glycoprotein IgG antibody	1	U/mL	<5
Anti- $\beta 2$ -glycoprotein IgM antibody	3	U/mL	<5
Anticardiolipin IgG antibody	5	GPL-U/mL	<10
Anticardiolipin IgM antibody	1	MPL-U/mL	<7

TABLE 1: Laboratory investigation results

WBC: white blood cell count; RBC: red blood cell count; MCV: mean corpuscular volume; MCH: mean corpuscular hemoglobin; MCHC: mean corpuscular hemoglobin concentration; RDW: red blood cell distribution width; ESR: erythrocyte sedimentation rate; eGFR: estimated glomerular filtration rate; ALT: alanine aminotransferase; GGT: gamma-glutamyl transferase; ALP: alkaline phosphatase; C3: complement component 3; C4: complement component 4; CRP: C-reactive protein; ANA: antinuclear antibody; SS-A: Sjögren syndrome-related antigen A; anti-dsDNA: anti-double-stranded DNA; IgG: immunoglobulin G; IgM: immunoglobulin M; H: high; L: low

Two punch biopsies were obtained from the dorsal hands. The first biopsy was taken from a depigmented patch for routine histopathological examination with hematoxylin and eosin (H&E) staining. Melan-A immunohistochemical staining was performed on the same biopsy specimen to assess melanocyte presence. The second biopsy was obtained from perilesional, sun-exposed skin on the dorsal hand for direct

immunofluorescence (DIF). Histopathology revealed compact hyperkeratosis, epidermal atrophy, hypergranulosis, and interface dermatitis with vacuolar degeneration of the basal cell layer. A band-like lymphocytic infiltrate predominantly involving the superficial dermis was observed. There was no significant dermal mucin deposition, and follicular involvement was not detected (Figures 2a-2b).

Melan-A immunohistochemical staining demonstrated an absence of melanocytes within the depigmented lesion. Direct immunofluorescence demonstrated linear granular deposition of IgG, IgA, and IgM along the dermo-epidermal junction, with colloid bodies, and linear shaggy fibrinogen deposition along the dermo-epidermal junction (Figures 3a-3d).

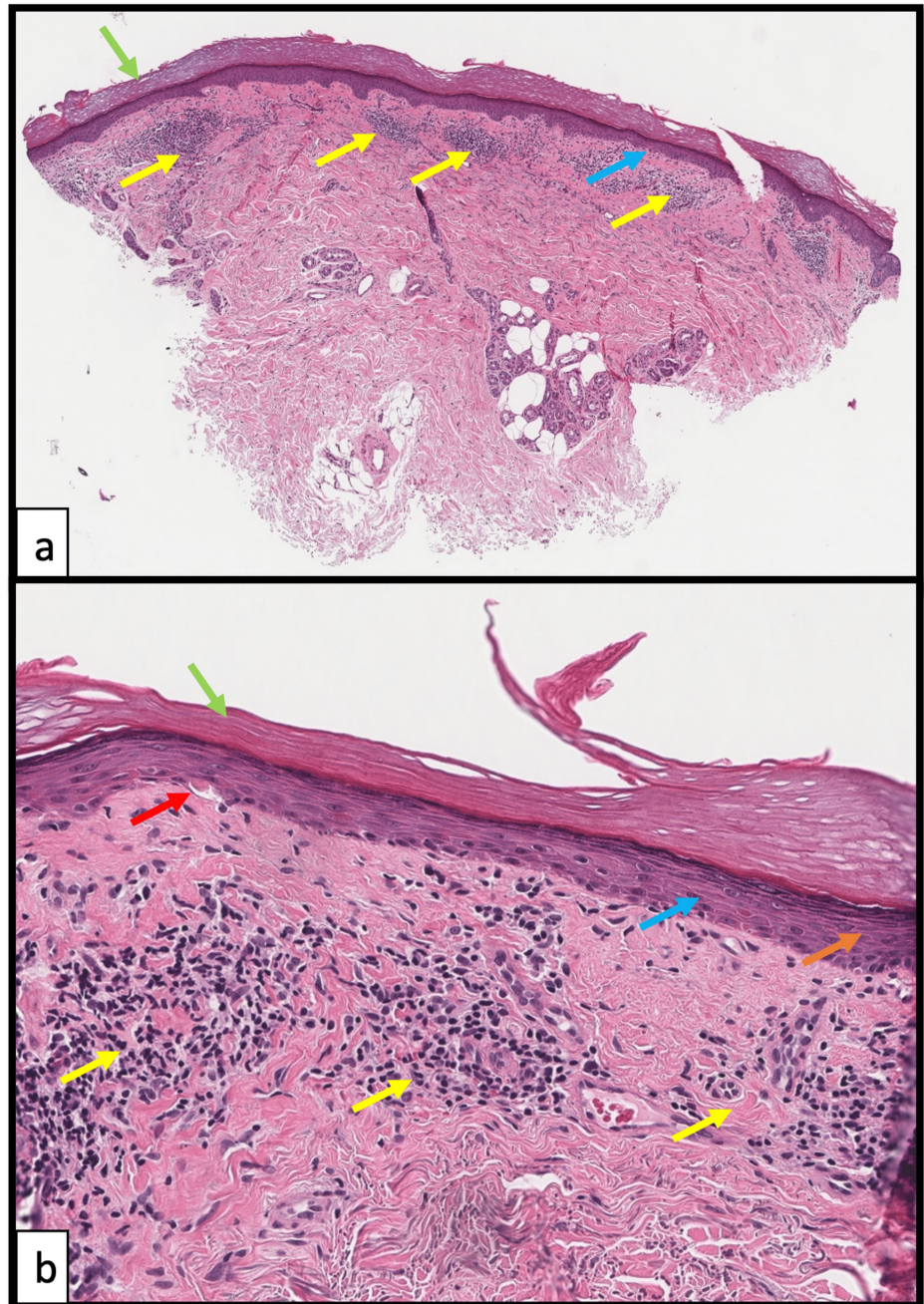


FIGURE 2: Histopathological features of a depigmented lesion from the dorsal hand

Low power demonstrating compact hyperkeratosis (green arrow), epidermal atrophy (blue arrow), and a bandlike lymphohistiocytic infiltrate at the dermo-epidermal junction (yellow arrow) (a, H&E stain x10). Higher power highlighting hypergranulosis (orange arrow), vacuolar degeneration of the basal cell layer, and colloid body (red arrow) (Civatte body) formation, consistent with interface dermatitis (b, H&E stain x40)

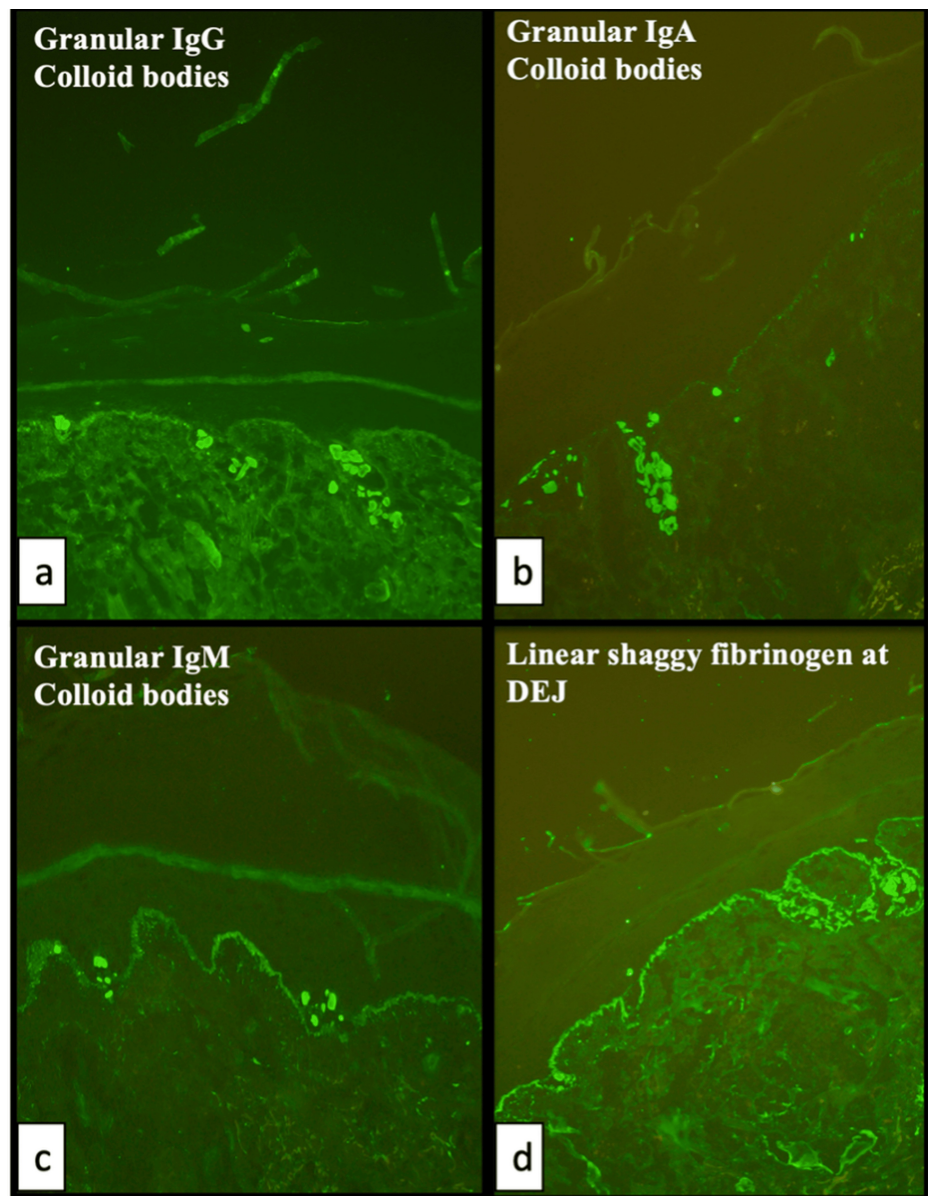


FIGURE 3: Direct immunofluorescence findings from perilesional, sun-exposed skin of the dorsal hand

(a, b, c x200) Linear granular deposition of IgG, IgA, and IgM along the dermo-epidermal junction with colloid bodies. (d x200) Positive staining for fibrinogen showing a linear, "shaggy" pattern along the dermo-epidermal junction, with associated colloid bodies

The combined clinical, histopathological, and immunopathological findings were considered compatible with CLE/LP overlap syndrome. The lupus-like component could not be classified confidently as a specific subtype of CLE. The depigmentation was described as vitiligo-like because post-inflammatory melanocyte loss could not be excluded.

Hydroxychloroquine 200 mg twice daily and topical ruxolitinib 1.5% applied once daily were initiated. At the one-month follow-up, mild repigmentation at the corner of the mouth was documented clinically and assessed with Wood's lamp examination, while depigmented patches persisted elsewhere on the face and dorsal hands (Figures 4-5). No active inflammatory lesions, new lesions, treatment-related adverse effects, or systemic symptoms were reported or observed during the follow-up period. Continued dermatology and rheumatology follow-up was arranged. Written informed consent was obtained from the patient for publication of this case report and accompanying clinical photographs.

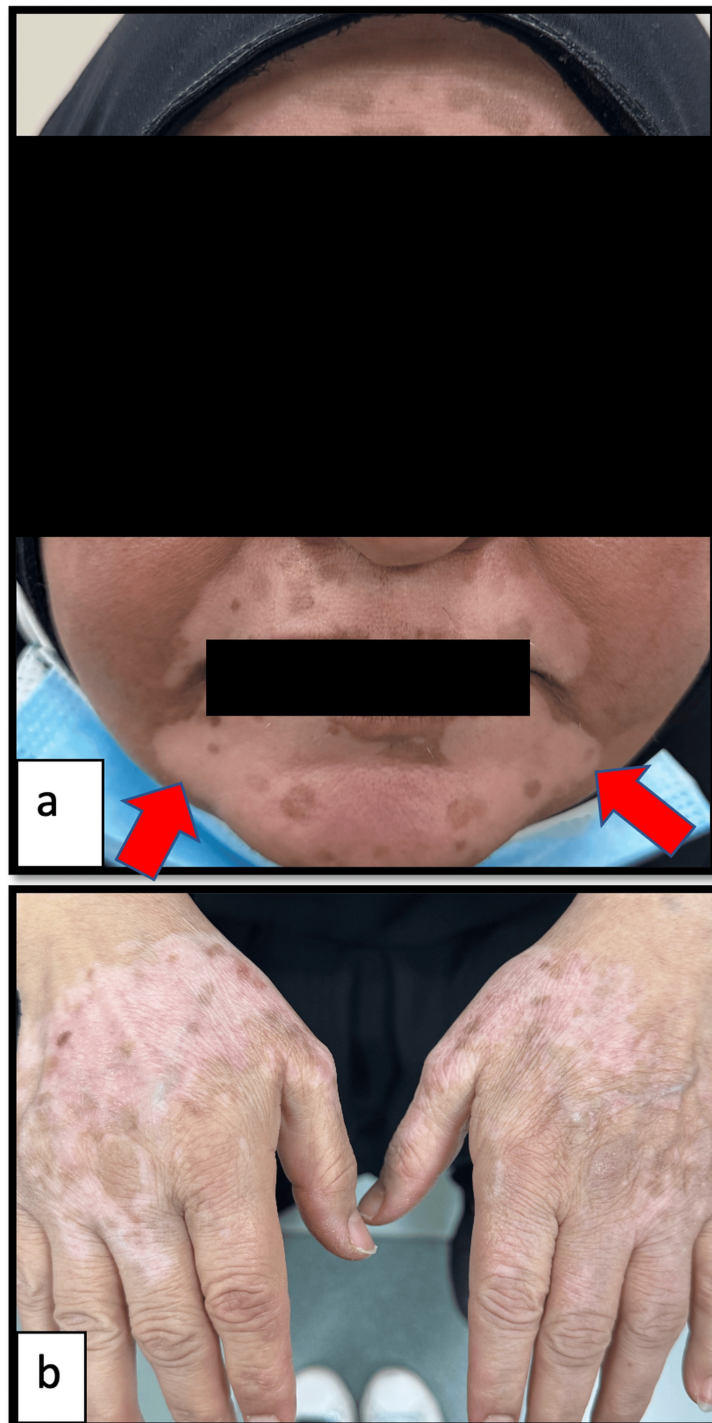


FIGURE 4: Clinical findings at one-month follow-up after treatment initiation

Clinical photographs of the face and bilateral dorsal hands obtained one month after initiating hydroxychloroquine 200 mg twice daily and topical ruxolitinib 1.5% once daily. Persistent well-demarcated depigmented patches are observed, with mild repigmentation at the oral commissure (a,b). No generalized repigmentation was documented

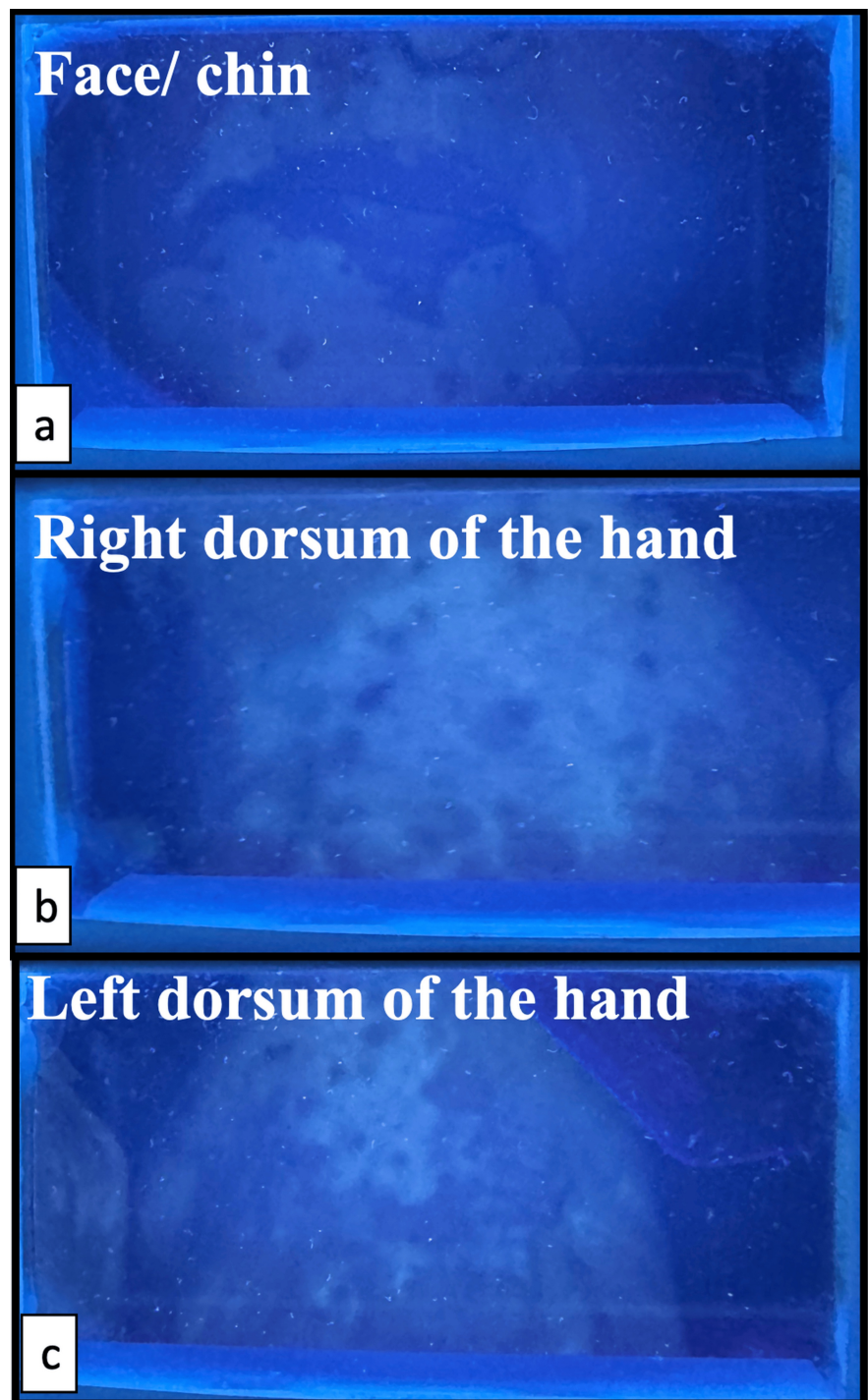


FIGURE 5: Wood's lamp examination demonstrating persistent depigmentation at one-month follow-up

Wood's lamp photographs of the face, perioral region, and bilateral dorsal hands obtained one month after treatment initiation. The depigmented patches demonstrate marked accentuation under Wood's lamp examination, with mild repigmentation noted at the oral commissure. Persistent depigmentation is evident at the remaining affected sites (a,b,c)

Discussion

CLE/LP overlap syndrome is distinguished from coincidental CLE and LP lesions by features of both conditions within the same clinicopathological process [2,3]. In this case, the LP-like component was supported by compact hyperkeratosis, hypergranulosis, basal vacuolar change, colloid bodies, and a band-like lymphocytic infiltrate, together with shaggy fibrinogen staining at the dermoepidermal junction. A CLE-like component was suggested by granular immunoglobulin deposition at the junction, positive ANA, weakly

positive anti-Ro/SSA, and a retrospectively reported history of malar erythema. None of these findings independently establishes CLE. The absence of significant dermal mucin and follicular involvement, together with the lack of characteristic features of a defined CLE subtype, precluded confident classification as discoid, subacute, or acute CLE. The lupus component therefore remained unclassifiable within the overlap presentation. The combined phenotype was compatible with the classic CLE/LP overlap pattern described by Jicha et al. [3], without independently establishing each component.

The DIF specimen was obtained from sun-exposed perilesional skin of the dorsal hand. Immunoglobulin deposition along the dermoepidermal junction may occur in sun-exposed skin and is not entirely specific for LE. Accordingly, the DIF findings were interpreted in conjunction with the clinical and histopathological features, including lichenoid interface dermatitis and immunoreactive colloid bodies, rather than as independent confirmation of CLE.

Distinguishing coexisting vitiligo from post-inflammatory depigmentation secondary to interface dermatitis remains challenging. Persistent, sharply demarcated depigmented patches, Wood's lamp accentuation, and absence of melanocytes on Melan-A staining support melanocyte loss but do not establish its mechanism. The distribution at sites of antecedent inflammation, residual erythema, and active histological interface injury make post-inflammatory melanocyte destruction plausible. Conversely, vitiligo may arise at sites of cutaneous inflammation, and a Koebner-like process is biologically possible [7,8]. Without dermoscopic documentation, lesions beyond previously inflamed areas, serial pigment mapping, or other discriminatory evidence, a separate diagnosis of vitiligo remains unconfirmed. We therefore use the descriptive term "vitiligo-like depigmentation" rather than attributing the findings to a proven Koebner phenomenon.

The initial inflammatory lesions resolved without therapy within approximately two months, whereas depigmentation persisted for approximately two years; interface inflammation on the later biopsy indicates that microscopic activity could persist despite clinical resolution of the original plaques. The case does not prove that the inflammatory process caused autoimmune vitiligo. Coexisting hypothyroidism is noteworthy but cannot be cited as evidence of autoimmune clustering without documentation of autoimmune thyroid disease [9,10].

Published cases illustrate the heterogeneity of CLE/LP overlap and the importance of distinguishing a true overlap phenotype from coincidental coexistence of LP and LE. Selected earlier reports and two recent cases are compared in Table 2 [2-4,6,10-12]. In particular, Martín-Zamora et al. reported resolution without residual hypopigmentation, contrasting with the persistent melanocyte-depleted depigmentation observed in our patient [12].

Study (reference)	Clinical context	Pigmentary findings	Interpretation/relevance
Romero et al., 1977 [2]	Early clinicopathological series of unusual LE/LP-like lesions	Variable atrophic and pigmentary changes; vitiligo not established	Foundational description of the overlap phenotype
Jicha et al., 2021 [3]	Case-based review and classification of CLE/LP overlap	Heterogeneous clinical morphologies; no single defining pigmentary pattern	Diagnostic framework distinguishing classic from possible overlap
Ahmed et al., 1982 [4]	Coexisting systemic LE and LP	Vitiligo reported in one patient	Coexistence does not necessarily establish a true cutaneous overlap lesion
Inalöz et al., 2001 [6]	CLE/LP overlap with scarring alopecia	Separate vitiligo diagnosis not established	Illustrates atypical appendageal involvement
Shahzadi et al., 2008 [10]	LP/LE overlap with hypothyroidism	No confirmed vitiligo mechanism documented	Thyroid comorbidity does not itself establish autoimmune pigmentary disease
Meghwar et al., 2025 [11]	30-year-old woman with LP/discoid LE overlap involving scalp, limbs, hands and oral mucosa	Pigmentary changes described; melanocyte depletion not demonstrated	Recent clinical comparator with a defined discoid LE component
Martin-Zamora et al., 2025 [12]	71-year-old woman with photoexposed erythematoviolaceous plaques and mixed LP/CLE features	No residual hypopigmentation after 3 months of hydroxychloroquine	Contrasts with persistent depigmentation in the present case

TABLE 2: Selected previously published reports of LE-LP overlap syndrome and related coexistence

CLE: cutaneous lupus erythematosus; LP: lichen planus

Summary of selected published cases illustrating the clinical heterogeneity of CLE-LP overlap syndrome, including clinical presentations, pigmentary findings, and diagnostic considerations. Reports of true CLE/LP overlap are distinguished from cases describing the coexistence of LE and LP. Although pigmentary alterations have been reported, their presence does not necessarily establish a separate diagnosis of vitiligo. The present case is notable for persistent vitiligo-like depigmentation with histologically documented melanocyte loss. This table represents a selective literature comparison rather than a systematic review of all published cases

Hydroxychloroquine 200 mg twice daily was prescribed for the CLE-like inflammatory component, and topical ruxolitinib 1.5% once daily was prescribed for the pigmentary component. JAK-STAT signaling is implicated in interferon-mediated melanocyte injury in vitiligo, although this rationale does not establish efficacy in CLE/LP overlap syndrome. At one month, mild repigmentation at the oral commissure was documented clinically and evaluated using Wood’s lamp examination, without evidence of generalized repigmentation. Longer follow-up is needed to assess treatment response and durability.

Limitations include the single-case design, retrospectively reported initial facial erythema, absence of dermoscopy, limited one-month follow-up, unconfirmed autoimmune etiology of hypothyroidism, unavailable details of the lupus anticoagulant assay and confirmatory testing, uncertainty regarding the mechanism of melanocyte loss, and the absence of long-term treatment outcomes. Clinical and immunofluorescence findings should not be interpreted as definitive evidence of a particular CLE subtype or separate vitiligo diagnosis.

Conclusions

CLE/LP overlap syndrome may be followed by persistent, vitiligo-like depigmentation with demonstrable melanocyte loss. This observation presents a diagnostic challenge because vitiligo and post-inflammatory melanocyte destruction may be indistinguishable on the currently available findings. Longitudinal examination, careful sampling, and correlation of pigmentary changes with inflammatory activity are important; a causal relationship between overlap-associated interface inflammation and vitiligo cannot be concluded from this case.

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: Danah Alkandari, Hasan KH A H A Ashkanani, Hawraa Mayouf, Ali Alajmi, Humoud Al-Sabah

Acquisition, analysis, or interpretation of data: Danah Alkandari, Hasan KH A H A Ashkanani, Hawraa Mayouf, Ali Alajmi, Humoud Al-Sabah

Drafting of the manuscript: Danah Alkandari, Hasan KH A H A Ashkanani, Hawraa Mayouf, Ali Alajmi, Humoud Al-Sabah

Critical review of the manuscript for important intellectual content: Danah Alkandari, Hasan KH A H A Ashkanani, Hawraa Mayouf, Ali Alajmi, Humoud Al-Sabah

Supervision: Danah Alkandari, Hasan KH A H A Ashkanani, Hawraa Mayouf, Ali Alajmi, Humoud Al-Sabah

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. Not applicable issued approval N/A. Institutional Review Board/ethics committee approval was not required for this single-patient case report in accordance with institutional policy. Written informed consent was obtained from the patient for publication of the clinical information and images included in this report. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Mahler V, Hornstein OP, Meyer S, Albrecht HP, Kiesewetter F: Lupus erythematosus/lichen ruber planus overlap syndrome. 5 cases in a patient sample of the Erlangen University Dermatology Clinic (1894-1995) (Article in German). *Hautarzt*. 1998, 49:295-302. [10.1007/s001050050744](https://doi.org/10.1007/s001050050744)
2. Romero RW, Nesbitt LT Jr, Reed RJ: Unusual variant of lupus erythematosus or lichen planus. Clinical, histopathologic, and immunofluorescent studies. *Arch Dermatol*. 1977, 113:741-8. [10.1001/archderm.1977.01640060037002](https://doi.org/10.1001/archderm.1977.01640060037002)
3. Jicha KI, Wang DM, Miedema JR, Diaz LA: Cutaneous lupus erythematosus/lichen planus overlap syndrome. *JAAD Case Rep*. 2021, 17:130-51. [10.1016/j.jdcr.2021.09.031](https://doi.org/10.1016/j.jdcr.2021.09.031)
4. Ahmed AR, Schreiber P, Abramovits W, Oestreicher M, Lowe NJ: Coexistence of lichen planus and systemic lupus erythematosus. *J Am Acad Dermatol*. 1982, 7:478-83.
5. Tziotziou C, Lee JY, Brier T, et al.: Lichen planus and lichenoid dermatoses: clinical overview and molecular basis. *J Am Acad Dermatol*. 2018, 79:789-804. [10.1016/j.jaad.2018.02.010](https://doi.org/10.1016/j.jaad.2018.02.010)
6. Inalöz HS, Chowdhury MM, Motley RJ: Lupus erythematosus/lichen planus overlap syndrome with scarring alopecia. *J Eur Acad Dermatol Venereol*. 2001, 15:171-4. [10.1046/j.1468-3083.2001.00223.x](https://doi.org/10.1046/j.1468-3083.2001.00223.x)
7. Patel P, Salimi M, Al-Kutubi H, Andrews K, Bhat J: PA32 Severe lichen planus with co-localization of vitiligo: a rare and unusual case. *Br J Dermatol*. 2023, 188:ljad113.336. [10.1093/bjd/ljad113.336](https://doi.org/10.1093/bjd/ljad113.336)
8. Zhang X, Lei L, Jiang L, et al.: Characteristics and pathogenesis of Koebner phenomenon. *Exp Dermatol*. 2023, 32:310-23.
9. Kassels A, Elsensohn AN, Kraus CN: Lichen planus is associated with other autoimmune conditions: a retrospective population-level study. *J Am Acad Dermatol*. 2024, 90:650-2. [10.1016/j.jaad.2023.11.016](https://doi.org/10.1016/j.jaad.2023.11.016)
10. Shahzadi N, Khurshid K, Pal SS: Lichen planus/lupus erythematosus overlap with hypothyroidism: a case report. *J Pak Assoc Dermatol*. 2008, 18:238-40. [10.66344/jpad.v18i4.603](https://doi.org/10.66344/jpad.v18i4.603)
11. Meghwar S, Batra M, Shabbir NZ, Devi P, Singh A, Ikram A, Verma A: A rare case of lichen planus/discoid lupus erythematosus overlap syndrome and its successful treatment. *Clin Case Rep*. 2025, 13:e70704. [10.1002/ccr3.70704](https://doi.org/10.1002/ccr3.70704)
12. Martín-Zamora AC, Gamboa-Flores A, Pozuelo-Díaz J, Campos-Hidalgo M: Erythematoviolaceous papules and plaques on sun-exposed areas. *JEADV Clin Pract*. 2025, 4:621-3. [10.1002/jvc.2.618](https://doi.org/10.1002/jvc.2.618)