BULLETIN OF STOMATOLOGY AND MAXILLOFACIAL SURGERY
Volume 21, Issue 3

DOI: 10.58240/1829006X-2025.3-313

CASE REPORT

HISTOPATHOLOGYC AND DERMOSCOPIC FINDINGS OF SUBACUTE CUTANEOUS LUPUS ERYTHEMATOSUS

Khachik Khachikyan, MD, PhD, DMSc,[1]* Hovhannes Hovhannisyan[2,3], Armine Hakobyan[2], Anj Giragos[2], Anahit Topchyan[2]

[1.]Professor, Head of Department of Dermatology and STIs of Yerevan State Medical University after M. Heratsi, Yerevan, Armenia
[2.]Assistant of Department of Dermatology and STIs of Yerevan State Medical University after M. Heratsi, Yerevan, Armenia
[3]National center for burns and dermatology of MO, Yerevan, Armenia
* Corresponding author:Professor Khachik Khachikyan, Head of Department of Dermatology and STIs of Yerevan State Medical University after M. Heratsi, National center for burns and dermatology, 25a Davtashen, Yerevan, Armenia, 0054; e-mail: xxm666699@mail.ru
Received: *Feb 10, 2025;* ***Accepted:*** *Mar.28. 2025;* ***Published:*** *Apr. 10, 2025*

ABSTRACT

The relevance, pathogenesis, classification, and clinical characteristics of a rare disease-subacute cutaneous lupus erythematosus-are presented.
A clinical case in a 60-year-old woman without signs of organ pathology is also described.
A dermatoscopic examination was performed. In the affected areas on the facial skin, twisted, short linear, occasionally branching, unfocused vessels, predominantly bright red in color, forming incomplete polygonal structures, were identified. In the affected areas on the skin of the extremities, irregularly arranged dotted-granular and twisted short linear vessels of dull red color were visualized, along with structureless zones of varying degrees of discoloration, exhibiting whitish-orange shades.
Pathomorphologically, the process is characterized by mild focal hyperkeratosis, vacuolar degeneration of the basal layer of the epidermis, a mildly expressed perivascular lymphohistiocytic inflammatory infiltrate, as well as a moderately expressed periadnexal (perieccrine) lymphohistiocytic inflammatory infiltrate.
Based on the medical history, clinical presentation, and the results of dermoscopic and histological examinations, the diagnosis of psoriasiform subacute cutaneous lupus erythematosus was established.

Keywords: Psoriasiform Subacute Cutaneous Lupus Erythematosus dermoscopyc features, pathomorphological features.

INTRODUCTION

Cutaneous lupus erythematosus (CLE) is a chronic autoimmune inflammatory disease affecting the connective tissue of the skin. It is characterized by a relapsing course, immune dysregulation, and a heterogeneous clinical presentations.
CLE can manifest as an isolated skin condition or as part of systemic lupus erythematosus (SLE). The annual incidence of CLE and SLE in the United States and European countries is approximately 4 and 3 cases per 100,000 people, respectively. Notably, without therapeutic intervention, 5–18% of CLE patients develop SLE within 3–5 years.[1–4]
The diagnosis of CLE relies on a comprehensive evaluation of patient history, clinical manifestations, biochemical and immunoserological findings. Dermatoscopic and histopathological examinations also play a crucial role in differential diagnosis.[1,5]

PATHOGENESIS

CLE is a multifactorial disease in which genetic, environmental and immunological factors play a significant role in pathogenesis.
The key genetic mechanisms contributing to the disease include:

- initiation and enhancement of antibody formation, associated with HLA gene variants (HLA-DR3, HLA-DR2, HLA-DRw52, HLA-DRw6, HLA-DQ1, HLA-DQ2, etc.);
- increased interferon-α (IFN-α) activity, which has a proinflammatory effect due to genetic variations in the *IRF5* and *STAT4* genes;
- accumulation of cellular debris, triggering an autoimmune response due to mutations in genes responsible for apoptotic cell clearance (*C1q, TREX1, DNASE1*);
- polymorphisms in the *PTPN22* (T-cell signal regulator) and *TNFAIP3* (inflammatory inhibitor) genes, contributing to immune dysregulation. [6, 7]

The key environmental triggers include ultraviolet UV-radiation (UVR), smoking, and certain medications.[3, 5, 11-14]

UV exposure can initiate and exacerbate skin lesions by:

- inducing keratinocyte apoptosis and increasing the expression of nuclear Ro/SSA and La/SSB autoantigens;
- triggering IFN-α hyperproduction.[11, 16]

Smoking negatively impacts SCLE through several mechanisms:

- oxidative stress, which damages cell membranes, promotes Ro/SSA and La/SSB autoantigen release, and amplifies autoimmune inflammatory responses;
- immune dysregulation, activating TNF-α, IL-6, IFN-γ, and other proinflammatory cytokines;
- increased photosensitivity. [4, 8]

Several medications have been linked to drug-induced SCLE, including:

- terbinafine;
- TNF-α inhibitors;
- anticonvulsants;
- calcium channel blockers;
- β-blockers; among others.[1, 2, 15]

CLINICS

CLE is classified into three clinical and histological subtypes:

- acute cutaneous lupus erythematosus (ACLE);
- subacute cutaneous lupus erythematosus (SCLE);
- chronic cutaneous lupus erythematosus (CCLE).[1–4, 8]

The following forms of ACLE are recognized:

- localized: presents with the characteristic "butterfly rash" over the nose and cheeks;
- widespread: manifests as maculopapular rashes;
- vesicular: characterized by the formation of subepidermal blisters .[2, 5]

SCLE typically presents as lesions on sun exposed skin areas, predominantly on the upper third of the trunk and upper limbs. [4]:

- annular or polycyclic form: non-scarring annular or polycyclic rashes;
- papulosquamous form: erythematous-squamous or psoriasiform plaques with tightly adherent scales and mild hyperkeratosis;
- target lesions (Rowell's syndrome): resembling erythema multiforme, necrotizing vasculitis, and other conditions.

CCLE manifests in various clinical forms:

- chronic discoid form: cicatricial localized or disseminated lesions;
- hypertrophic or warty form: characterized by extensive hyperkeratosis;
- deep form (lupus panniculitis): presents with subcutaneous nodules;
- acral form: resembles frostbite, affecting distal skin areas;
- mucocutaneous form: includes ulcers and plaques in the oral cavity. [2, 5]

Non-specific clinical signs of cutaneous lupus erythematosus include vascular manifestations (vasculopathy, leukocytoclastic vasculitis, urticaria, Raynaud's phenomenon, thrombophlebitis, fingertip necrosis) and follicular abnormalities (alopecia areata, telogen effluvium).[5, 9, 10]

HISTOPATHOLOGY

The histopathological pattern of CLE (ACLE, SCLE, CCLE) is primarily characterized by interface dermatitis — manifested mainly by a lymphocytic inflammatory infiltrate (cytotoxic CXCR3+ lymphocytes) localized predominantly in the perivascular and periadnexal areas. Notably, apoptotic or necroptotic keratinocytes are present. This pattern also includes the expression of

Khachik Khachikyan, Hovhannes Hovhannisyan, Armine Hakobyan, Anj Giragos, Anahit Topchyan. Histopathologic and deramatoscopic findings of subacute cutaneous lupus erythematosus.Bulletin of Stomatology and Maxillofacial Surgery.2025;21(3).313-318.doi:10.58240/1829006X-2025.3-313

interferon-regulated cytokines and chemokines, epidermal and follicular hyperkeratosis, the presence of mucin in the dermis, and other associated features. [2, 3, 12, 17]

CLINICAL CASE

Patient M., a 60-year-old female of Yerevan, Armenia, sought consultation in November 2024 at the National Center for Burns and Dermatology of the Ministry of Health of the Republic of Armenia. She presented with complaints of rashes on the skin of her face, upper, and lower extremities.

The patient reports having had the condition for 20 years. Previously, she underwent multiple treatments for presumed diagnoses, including psoriasis and Devergie's disease, among others. She has periodically received Diprospan injections, with varying degrees of effectiveness. There is no known family history of skin diseases. No concomitant conditions have been identified.

The patient is primarily concerned about the rash on her face and extremities, accompanied by mild itching.

Objective findings: slightly infiltrated erythematous-squamous lesions are observed on the cheeks, periorbital region, eyebrows, and nasal root. Similar morphological elements are present on the upper and lower extremities, with additional follicular lesions (Figures 1–3)

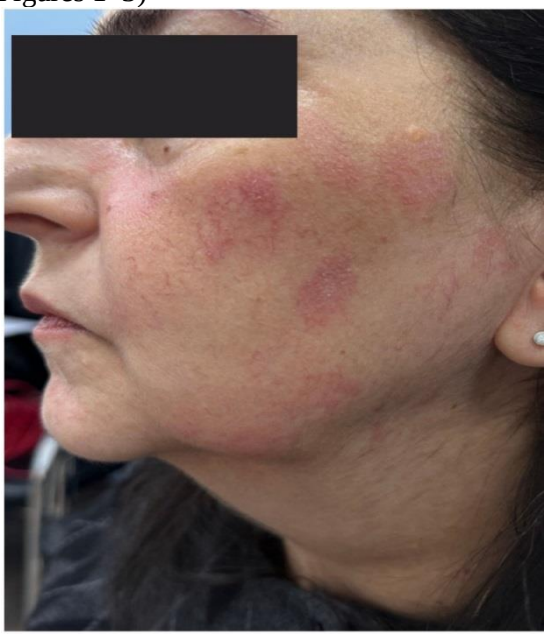


Figure 1. Clinical images of skin lesions on the face

Figure 2. Clinical images of skin lesions on the face and extremities

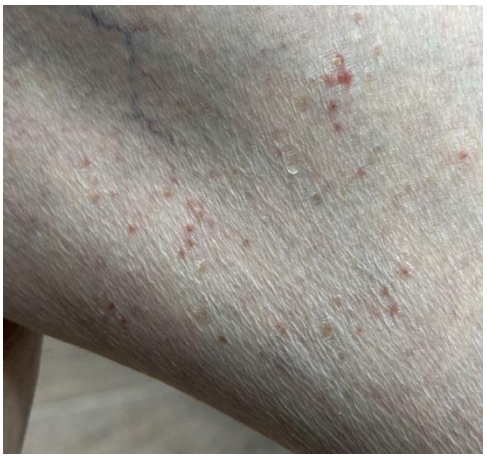


Figure 3. Clinical images of skin lesions on the face and extremities

Laboratory findings:

- general clinical and biochemical blood tests, as well as general urine analysis, show no abnormalities;
- blood cortisol level (9 a.m.): 135.7 nmol/l;
- antibodies to phospholipids (IgM/IgG): negative;
- antibodies to cardiolipin (IgG, IgM, IgA): negative;
- antinuclear IgG antibodies: Positive.

DERMOSCOPIC FINDINGS

These dermoscopic findings describe different structural characteristics of skin lesions on the face and extremities.

Facial Lesions:

- background: erythematous;
- vascular pattern: varying diameters, predominantly bright red, forming incomplete polygonal structures tortuous, short-linear, and branching vessels;

- additional features: small, irregularly placed white superficial scales, whitish-yellowish structureless areas.

Extremity Lesions:

- background: yellowish-pinkish;
- vascular pattern: irregularly placed dull red in color punctate-granular and short-linear tortuous vessels;
- additional features: whitish-orange discolored structureless zones, indistinct scaly structures, focal hyperpigmentation(figure,4,5).

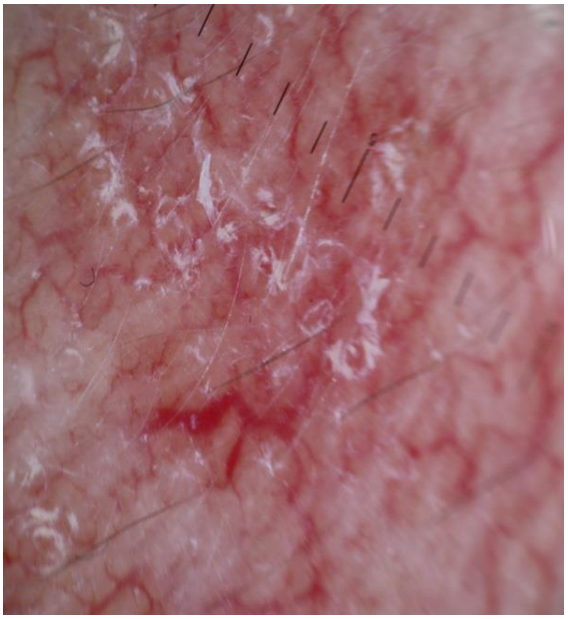


Figure 4.

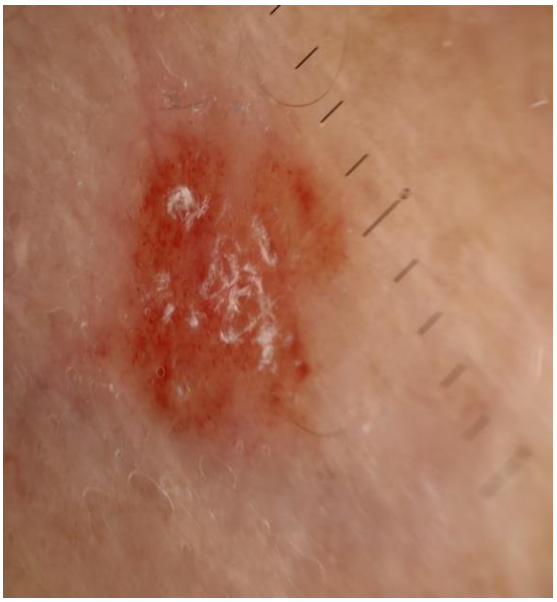


Figure 5. Dermatoscopic images of facial skin lesions: bright red polymorphic vessels, small irregularly distributed white scaly structures, and whitish-yellow structureless zones are visualized against an erythematous background.

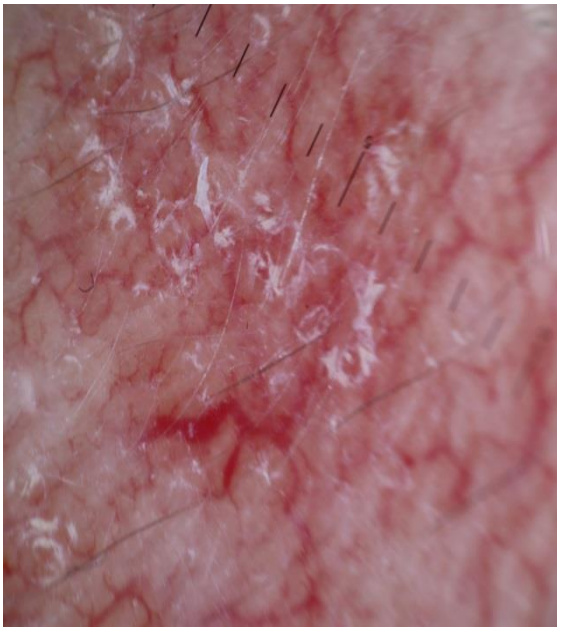


Figure 6

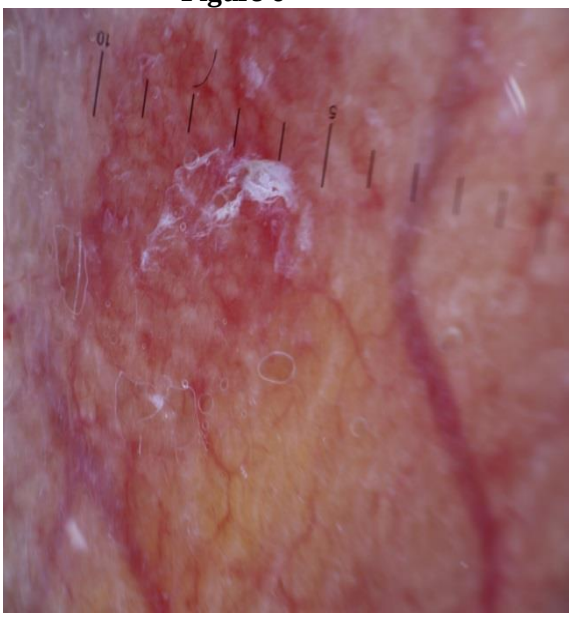


Figure 6,7.Dermatoscopic images of skin lesions on the extremities: irregularly distributed punctate-granular and tortuous short-linear vessels of a dull red color, along with whitish-orange structureless zones, are visualized against a yellowish-pink background.

PATHOMORPHOLOGICAL RESULTS

The main morphological changes are represented by: mild focal hyperkeratosis and vacuolar degeneration of the basal layer of the epidermis (Figure 8 and 9); perivascular lymphohistiocytic inflammatory infiltrate (Figure 10);periadnexal (perieccrine) lymphohistiocytic inflammatory infiltrate(Figure 11).

Conclusion: based on the above data, the diagnosis was: psoriasiform (papular-squamous) subacute cutaneous lupus erythematosus.

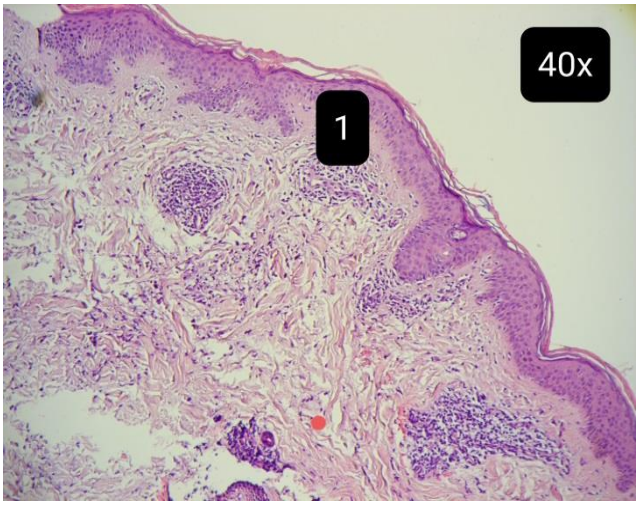


Figure 8.

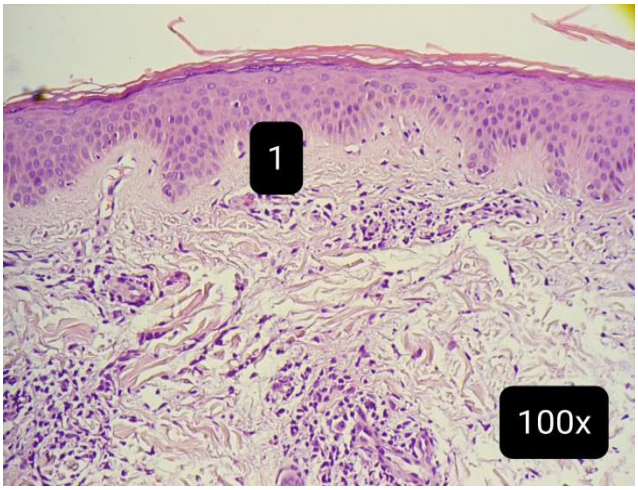
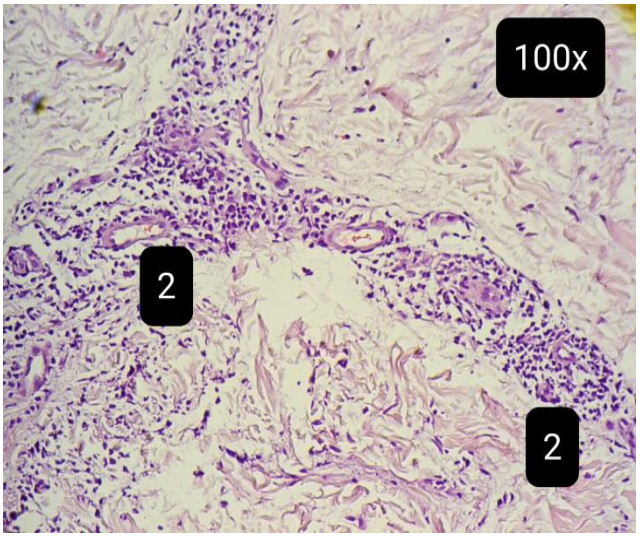


Figure 9.



Fugure 10.

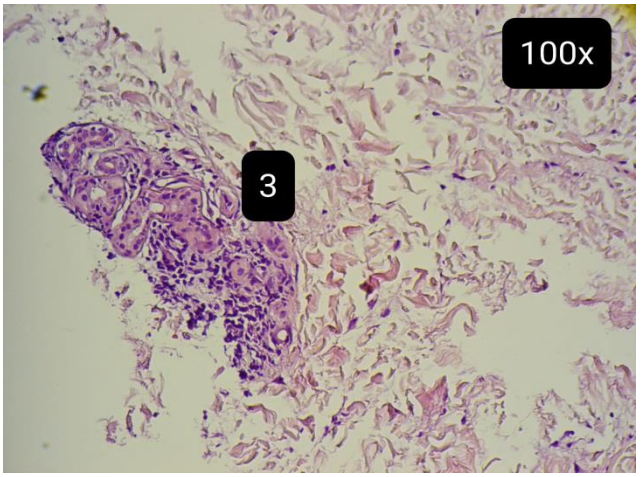


Figure 11.

Figure 8-11. Histological images of skin biopsy specimens from lesions (hematoxylin and eosin staining, ×40, x100): Fig. 8-9 show focal hyperkeratosis and vacuolar degeneration of the basal layer of the epidermis; Fig. 10 shows a perivascular lymphohistiocytic inflammatory infiltrate; Fig. 11 shows a periadnexal (perieccrine) lymphohistiocytic inflammatory infiltrate

CONCLUSIONS

The presented clinical case demonstrates the importance of dermatological and histological examinations in the diagnosis of subacute psoriasiform cutaneous lupus erythematosus.

DECLARATIONS

Conflicts of Interest

The authors declare no conflicts of interest.

Funding

Self Funded

Ethical Approval

Not Applicable

Informed Consent

Not Applicable

Acknowledgments

Not Applicable

REFERENCES

1. Petersen M.P., Moller S., A Bygum A. et al. Epidemiology of cutaneous lupus erythematosus and the associated risk of systemic lupus erythematosus: a nationwide cohort study in Denmark. Lupus 2018; 27(9): 1424-1430.doi:10.1177/0961203318777103.
2. Wenzel J. Cutaneous lupus erythematosus: new insights into pathogenesis and therapeutic strategies. Nat Rev Rheumatol 2019;15:519–532. https://doi.org/10.1038/s41584-019-0272-0
3. Filotico R., Mastrandea V. Cutaneous lupus erythematosus:clinico-pathologic correlation. Giornale Italiano di Dermatologia e Venereologia 2018;153(2):216-29.DOI:10.23736/S0392-0488.18.05929-1
4. Everton C. S., Garcia L.C. Cutaneous lupus erythematosus: a review of etiopathogenic, clinical, diagnostic and therapeutic aspects. Anais Brasileiros de Dermatologia 2023; 98; 3: 355-372. https://doi.org/10.1016/j.abd.2022.09.005.
5. Vale C.S., Garcia L.C. Cutaneous lupus erythematosus: a review of etiopathogenic, clinical, diagnostic and therapeutic aspects. Anais Brasileiros de Dermatologia 2023; 98; 3:355-372. DOI: 10.1016/j.abd.2022.09.005
6. Ruiz-Larrañaga O., Migliorini P., Uribarri M. et al. Genetic association study of systemic lupus erythematosus and disease subphenotypes in European populations. Clin Rheumatol 2016, 35 (5): 1161–1168.DOI: 10.1007/s10067-016-3235-8
7. Chen H.W, Grant Barber G., Benjamin F Chong B.F. The Genetic Landscape of Cutaneous Lupus Erythematosus. Front Med (Lausanne) 2022; 2; 9: 916011.DOI: 10.3389/fmed.2022.916011
8. Cooper E.E., Pisano C.E., Shapiro S.C. Cutaneous Manifestations of “Lupus”: Systemic Lupus Erythematosus and Beyond. International Journal of Rheumatology 2021; 19 May; Article number 6610509.doi.org/10.1155/2021/6610509
9. Sunderkotter C.H., Zelger B., Cheen K-R. et al. Nomenclature of cutaneous vasculitis: dermatologic addendum to the 2012 revised International Chapel Hill Consensus Conference nomenclature of vasculitides.ArthritisRheumatol.2018;70(2):171–184. doi: 10.1002/art.40375.
10. Udompanich S., Chanprapaph K., Suchonwanit, P. Hair and scalp changes in cutaneous and systemic lupus erythematosus. Am J Clin Dermatol 2018, 19 (5): 679–694. 29. DOI: 10.1007/s40257-018-0363-8
11. Kuhn A., Bijl M. Pathogenesis of cutaneous lupus erythematosus.Lupus.2008;17;5:389-93. 10.1177/0961203308090019
12. Grönhagen C.M., Nyberg F. Cutaneous lupus erythematosus: An update. Indian Dermatol Online J 2014; 5: 7-13.DOI: 10.4103/2229-5178.126020
13. Kuhn A., Wenzel J., Weid H. Photosensitivity, apoptosis, and cytokines in the pathogenesis of lupus erythematosus: a critical review. Clin Rev Allergy Immunol 2014;47(2):148-62.DOI: 10.1007/s12016-013-8403-x.
14. Kim A., Chong B.F. Photosensitivity in cutaneous lupus erythematosus. Photoderm Photoimm Photomed 2013;29(1):4-11.doi: 10.1111/phpp.12018.
15. Marzano A.V., Vezzoli P., Crosti C. Drug-induced lupus: an update on its dermatologic aspects. Lupus 2009; 18: 935-40.DOI:10.1177/0961203309106176
16. Wenzel J., Zahn S., Mikus S. et al. The expression pattern of interferon-inducible proteins reflects the characteristic histological distribution of infiltrating immune cells in different cutaneous lupus erythematosus subsets. Br J Dermatol 2007, 157 (4): 752–757.DOI: 10.1111/j.1365-2133.2007.08137.x
17. Lauffer F., Jargosch M., Krause L. et al. Type I immune response induces keratinocyte necroptosis and is associated with interface dermatitis. J Invest Dermatol.2018,38(8):1785–1794. DOI: 10.1016/j.jid.2018.02.034