Incidence and Factors Associated with Systemic Lupus Erythematosus in Patients Initially Presenting with Cutaneous Lupus Erythematosus at the Institute of Dermatology: A Retrospective 8-Year Study

Tarakorn Chankrachang[1], Sasima Thammarucha[*2]

[1]College of Medicine, Rangsit University, Pathum Thani, Thailand
[2]Institute of Dermatology, Bangkok, Thailand
[*]Corresponding author, E-mail: tsasima@hotmail.com

Abstract

Cutaneous lupus erythematosus (CLE) primarily affects the skin but can progress to systemic lupus erythematosus (SLE), a severe autoimmune disease. Progression rates vary widely across populations and may be influenced by genetic, clinical, and environmental factors, However, data on this transition in the Thai population remain scarce. Most previous studies have focused on Western populations, while research in Asian cohorts has been limited by small sample sizes, short follow-up durations, and inconsistent diagnostic criteria. This has led to uncertainty regarding risk factors for SLE development in CLE patients. To address these gaps, this study investigates the incidence of SLE among CLE patients in Thailand and identifies key clinical and laboratory predictors of disease progression. We conducted a retrospective study of 240 CLE patients diagnosed between January 2016 and December 2023 at the Institute of Dermatology, Thailand. Demographic, clinical, and laboratory data were analyzed using descriptive statistics and logistic regression to assess factors associated with SLE progression. Odds ratios (OR) and 95% confidence intervals (CI) were calculated to determine the strength of associations. Among 240 CLE patients, 12 (5%) developed SLE, with most being female (75%) and over 50 years old (50%). Chronic CLE (91.67%), particularly discoid lupus erythematosus (83.33%), was the predominant subtype. The median time to SLE diagnosis was 3.04 years. Significant clinical associations included arthritis ($p = 0.050$) and arthralgia ($p < 0.005$). Key laboratory markers were leukopenia (OR = 113.12, 95% CI: 8.35–1531.69, $p < 0.001$), and proteinuria (OR = 25.83, 95% CI: 1.94–343.94, $p = 0.014$) This study provides novel insights into the progression of CLE to SLE in Thai patients, addressing this population's lack of epidemiological data. The findings highlight leukopenia and proteinuria as strong predictors of SLE development, emphasizing the importance of routine laboratory monitoring in CLE patients. Identifying high-risk individuals early may enable targeted surveillance and timely intervention, ultimately improving patient outcomes. Future prospective studies are needed to validate these findings and refine risk stratification strategies in CLE patients.

Keywords: *Cutaneous lupus erythematosus (CLE), Systemic lupus erythematosus (SLE), Incidence, Risk factors*

1. Introduction

Cutaneous lupus erythematosus (CLE) is an autoimmune disorder characterized by a range of clinical presentations, including acute CLE (ACLE), subacute CLE (SCLE), and chronic CLE (CCLE). These subtypes are typically distinguished based on clinical features, alongside histopathological and laboratory findings, as well as the duration of symptoms. It is found more in females than males and more in adults than children, with an average age of 48.5 years. The cause of the disease is still unknown. It is believed that there are several factors that may trigger the disease, including genetics, environmental factors, hormones, and ethnicity (Black et al., 2021; Ameer et al., 2022; Filotico, & Mastrandrea, 2018). While CLE is often confined to cutaneous involvement, a subset of patients eventually develops systemic lupus erythematosus (SLE), a severe autoimmune disease affecting multiple organs. Skin lesions occur in approximately 70-80% of SLE patients at some stage of the disease (Elmgren, & Nyberg, 2023), and in up to 25% of cases, cutaneous symptoms appear as the initial presentation (Vale, & Garcia, 2023; Arkin et al., 2015).

The reported incidence of CLE progression to SLE varies widely, with rates ranging from approximately 5% to 25% depending on study populations and follow-up duration (Vale, & Garcia, 2023;

Zhou et al., 2020; Insawang et al., 2010). Higher progression rates have been observed in Western cohorts, (Kaul et al., 2016; Stojan, & Petri, 2018). Whereas studies in Asian populations, remain limited and inconclusive. South Korea reported a higher rate of 20.8% (Baek et al., 2020). A Thai study of 101 CLE patients found that about 10% developed SLE, though with mild systemic symptoms (Chanprapaph et al., 2021). However, small sample sizes limit the identification of clear risk factors. Epidemiological data on CLE and SLE in Thailand are scarce, and most research is based on Western populations, which may not reflect Thai-specific genetic and environmental factors. Given these differences, local studies are needed to improve understanding of disease progression and develop better screening and monitoring strategies for CLE patients at risk of SLE.

The progression from CLE to SLE presents diagnostic challenges, especially in Thai patients, due to the diversity of CLE subtypes and overlapping features with other conditions. Additionally, genetic factors, environmental influences, and healthcare access may affect disease progression differently than in Western populations. This study aims to assess the incidence of SLE in patients initially diagnosed with CLE at the Institute of Dermatology in Thailand and to identify key clinical and laboratory factors associated with disease progression. Our findings will help improve risk assessment, early intervention, and monitoring of CLE patients at risk of developing SLE.

2. Objectives

The objective is to determine the incidence of systemic lupus erythematosus (SLE) and identify the factors associated with its development in patients initially presenting with cutaneous lupus erythematosus (CLE) at the Institute of Dermatology, Thailand, over an 8-year retrospective period.

3. Materials and Methods

3.1 Study design

This retrospective cohort study was conducted at the Institute of Dermatology, Thailand, over an 8-year period. Medical records of patients diagnosed with cutaneous lupus erythematosus (CLE) were reviewed to assess the incidence and factors associated with the development of systemic lupus erythematosus (SLE), which was diagnosed according to the SLICC criteria, EULAR/ACR criteria or ICD-10.

3.2 Study population

Inclusion criteria

1. All Thai patients aged 18 years or older
2. Patients diagnosed with cutaneous lupus erythematosus (CLE) based on clinical evidence or imaging findings, with LE-specific features, including:
   - subacute cutaneous LE (SCLE) (ICD-10 L93.1): annular or papulosquamous form
   - chronic cutaneous LE (CCLE) (ICD-10 L93.0, L93.22, L93.23, L93.2.2): classis DLE, hypertrophic DLE, mucosal DLE, lupus profundus/lupus panniculitis, chilblain LE, and LE tumidus
3. Patients who received follow-up treatment and had a duration of CLE before the diagnosis of SLE for at least 6 months at the Institute of Dermatology, starting from the start of treatment

Exclusion criteria

1. Patients diagnosed with overlap syndrome including systemic lupus erythematosus (SLE), progressive systemic sclerosis (PSS), polymyositis (PM), dermatomyositis (DM), Sjögren's syndrome (SS), rheumatoid arthritis (RA), and mixed connective tissue disease (MCTD)
2. Patients diagnosed with drug-induced DLE and drug-induced SCLE
3. Patients without biopsy results or direct immunofluorescence (DIF) testing for the diagnosis of CLE
4. Patients whose treatment history, according to the case record form, is incomplete (e.g., the diagnosis does not specify the type of CLE or the location of the lesion)

3.3 Research procedures

1) Electronic medical records of patients with cutaneous lupus erythematosus at the Institute of Dermatology were searched from January 1, 2016, to December 31, 2023, covering a total period of 8 years.
2) Excel was used to search for specific disease names according to ICD-10, including cutaneous lupus erythematosus, subacute lupus erythematosus, discoid lupus erythematosus, lupus profundus/panniculitis, tumid lupus erythematosus, and other lupus erythematosus variants, and then this information was further reviewed for each case.
3) The hospital uses a soft con system to access patient information to collect data according to the case record form, including age, gender, ethnicity, body mass index (BMI), family history of SLE, smoking history, treatment follow-up, CLE lesion characteristics, CLE lesion distribution, other symptoms, and recorded laboratory test results.
4) The obtained data were used for data analysis according to the objectives by assessing the incidence of SLE and identifying factors related to the occurrence of SLE in CLE patients at the Institute of Dermatology.

3.4 Research instrument

This study examined data from electronic medical records of patients with cutaneous lupus erythematosus using the hospital's soft con system to access patient data and collect data according to the case record form. The data used in the study were as follows: 1) General data, including age, gender, underlying diseases, race, body mass index (BMI), family history of SLE, smoking history, and follow-up treatment; and 2) Clinical data, including CLE lesion characteristics, CLE lesion distribution, other symptoms, and laboratory test results.

3.5 Data analysis

Statistical analyses were performed using SPSS version 25.0. Descriptive statistics were employed to summarize demographic, clinical, and laboratory data. The incidence of SLE was calculated as the proportion of CLE patients who developed SLE during the study period. Logistic regression analysis was performed to identify factors associated with the progression to SLE. All statistical tests were two-tailed, with significance defined as p-value < 0.05. The analysis was adjusted for potential confounding variables, including underlying diseases, subtypes of CLE, generalized area of involvement, oral ulcers, non-scarring alopecia, leukopenia, proteinuria, and baseline ANA titer.

4. Results and Discussion

4.1 Results

A total of 240 patients diagnosed with cutaneous lupus erythematosus (CLE) were included in this study, of whom 12 (5%) developed systemic lupus erythematosus (SLE). Among these patients, the majority were female (75%) and aged over 50 years (50%). The median follow-up duration was 3.04 years (range, 0.06-7.10 years). Additionally, the median time from CLE diagnosis to SLE diagnosis was 2.09 years (range, 0.06-5.04 years). Most patients (91.67%) had no reported family history of SLE, and only a few had a history of smoking or comorbidities. No significant differences were found in demographic factors (sex, age, BMI, family history, smoking) between non-SLE and SLE patients. However, diabetes mellitus showed a trend toward higher prevalence in SLE patients (16.67% vs. 3.07%, $p = 0.068$), though it did not reach statistical significance. Detailed demographic characteristics are presented in Table 1.

Among the patients who progressed to SLE, chronic cutaneous lupus erythematosus (CCLE) was the most common CLE subtype (91.67%), with discoid lupus erythematosus (DLE) accounting for 83.33%. Lesions were generalized (50.00%) and presented with scaling (58.33%), scarring (41.67%), and atrophy (50.00%). Photosensitivity was observed in 8.33% of patients. Musculoskeletal symptoms were also significantly associated with SLE progression, particularly arthralgia ($p < 0.005$) and arthritis ($p = 0.050$).

While arthritis showed borderline statistical significance, its clinical relevance should not be overlooked, as joint involvement is a well-recognized early sign of systemic disease in lupus patients.

Table 1 Descriptive statistics of demographic variables

Characteristic	**Non-SLE (n=228)**	**SLE (n=12)**	***p*-value**
Gender			
Male	76 (33.33)	3 (25.00)	0.775
Female	152 (66.67)	9 (75.00)	
Age			
18 - 29 years	48 (21.05)	3 (25.00)	0.519
30 - 49 years	92 (40.35)	3 (25.00)	
≥ 50 years	88 (38.60)	6 (50.00)	
BMI (kg/m^2)			
Underweight (BMI <18.5 kg/m^2)	19 (8.33)	2 (16.67)	0.423
Normal (BMI 18.5-22.9 kg/m^2)	91 (39.91)	6 (50.00)	
Overweight (BMI 23-24.9 kg/m^2)	42 (18.42)	2 (16.67)	
Obesity (BMI ≥ 25 kg/m^2)	76 (33.33)	2 (16.67)	
Family history of SLE			
No	25 (10.96)	1 (8.33)	1.000
Not specified	203 (89.04)	11 (91.67)	
Smoking			
No	15 (6.58)	1 (8.33)	0.335
Yes	21 (9.21)	2 (16.67)	
Not specified	192 (84.21)	9 (75.00)	
Underlying diseases			
Hypertension	35 (15.35)	1 (8.33)	1.000
Diabetes mellitus	7 (3.07)	2 (16.67)	0.068
Chronic kidney	4 (1.75)	0	1.000
Dyslipidemia	20 (8.77)	2 (16.67)	0.303
Atopic dermatitis	1 (0.44)	0	1.000
Others	39 (17.11)	2 (16.67)	1.000
Follow-up data (years)			
Follow-up duration, median (range)	1.11 (0.06-7.10)	3.04 (0.06-7.10)	0.175[a]
Time to develop SLE, median (range)	-	2.09 (0.06-5.04)	

Data are presented as number (%), Fisher's exact test, a = Mann-Whitney U test, *p*-value < 0.05

Several laboratory parameters were significantly associated with SLE development. Leukopenia (WBC < 4,000/mm³) was the strongest predictor ($p < 0.001$), with 91.67% of SLE patients exhibiting this abnormality. Proteinuria was also significantly associated with SLE ($p = 0.001$), suggesting early renal involvement. Additional laboratory markers included low complement levels ($p < 0.001$), and the presence of SLE-specific antibodies ($p = 0.001$) were observed more frequently in SLE patients. These findings are summarized in Table 2.

Table 2 Clinical and laboratory characteristics

Clinical dermatologic	**Non-SLE (n=228)**	**SLE (n=12)**	***p*-value**
Subtypes of CLE			
SCLE	11 (4.82)	1 (8.33)	0.468
CCLE	217 (95.18)	11 (91.67)	
Subacute CLE			
Annular form	11 (4.82)	1 (8.33)	0.468
Papulosquamous form	1 (0.44)	0	1.000

Clinical dermatologic	Non-SLE (n=228)	SLE (n=12)	*p*-value
Chronic CLE			
DLE	179 (78.51)	11 (91.67)	0.468
Classic DLE	164 (71.93)	10 (83.33)	0.520
Hypertrophic DLE	7 (3.07)	0	1.000
Mucosal DLE	8 (3.51)	1 (8.33)	0.375
Lupus profundus	28 (12.28)	0	0.370
LE tumidus	9 (3.95)	0	1.000
Area of involvement			
Generalized	100 (43.86)	6 (50.00)	0.676[a]
Localized	128 (56.14)	6 (50.00)	0.676
Scalp	54 (23.68)	4 (33.33)	0.491
Face	65 (28.51)	3 (25.00)	1.000
Nose	13 (5.70)	0	1.000
Ear	21 (9.21)	0	0.607
Lip	14 (6.14)	2 (16.67)	0.186
Characteristics			
Scaling	93 (40.79)	7 (58.33)	0.230[a]
Scarring	157 (68.86)	5 (41.67)	0.062
Atrophy	103 (45.18)	6 (50.00)	0.744[a]
Photosensitivity	2 (0.89)	1 (8.33)	0.143
Other clinical manifestations			
Present	23 (10.09)	2 (16.67)	0.362
Non-scarring alopecia	10 (4.39)	1 (8.33)	0.438
Oral ulcers	13 (5.70)	1 (8.33)	0.522
Musculoskeletal			
Arthritis	0	1 (8.33)	0.050
Arthralgia	5 (2.19)	3 (25.00)	0.005
Hematologic			
Autoimmune hemolysis	3 (1.32)	0	1.000
Leukopenia (WBC < 4,000/mm^3)	30 (13.16)	11 (91.67)	<0.001
Thrombocytopenia (Plt. < 100,000/mm^3)	2 (0.88)	1 (8.33)	0.143
Renal			
Kidney function (urea, creatinine)	3 (1.32)	0	0.460
Proteinuria	5 (2.19)	4 (33.33)	0.001
Antinuclear antibody (ANA)			
Negative baseline	85 (37.28)	1 (8.33)	0.060
Positive baseline	143 (62.72)	11 (91.67)	0.060
ANA titer; 1:80-1:160	68 (29.82)	2 (16.67)	0.517
ANA titer; 1:320-1:1280	55 (24.12)	6 (50.00)	0.081
ANA titer; 1:2560	19 (8.33)	3 (25.00)	0.085
Pattern of positive ANA			
Speckled	96 (41.67)	8 (66.67)	0.088[a]
Homogenous	43 (18.86)	3 (25.00)	0.705
Nucleolar	16 (7.02)	0	1.000
Centromere	1 (0.44)	0	1.000
Cytoplasmic	6 (2.63)	1 (8.33)	0.305
Immunologic			
Antiphospholipid antibody			
Negative	16 (7.02)	1 (8.33)	0.595
Not specified	212 (92.98)	11 (91.67)	
Complement			
Abnormal	1 (0.44)	4 (33.33)	<0.001

Clinical dermatologic	Non-SLE (n=228)	SLE (n=12)	*p*-value
Normal	17 (7.46)	0	
Not specified	210 (92.11)	8 (66.67)	
SLE-specific antibodies			
Negative	97 (42.54)	7 (58.33)	0.001
Positive	4 (1.75)	3 (25.00)	
Not specified	127 (55.70)	2 (16.67)	

Data are presented as number (%), SCLE = Subacute CLE, CCLE = Chronic CLE, Fisher's exact test, a = Pearson Chi-Square test, *p*-value < 0.05

Table 3 Clinical and laboratory factors for SLE determined using a binary logistic regression model

Variable	Non-SLE (n=228)	SLE (n=12)	OR (95% CI)	*p*-value
Underlying disease				
Yes	70 (92.11)	6 (7.89)	2.68 (0.54 to 13.35)	0.228
No	158 (96.34)	6 (3.66)	ref.	
Subtypes of CLE				
SCLE	11 (91.67)	1 (8.33)	1.23 (0.10 to 14.60)	0.871
CCLE	217 (95.18)	11 (4.82)	ref.	
Generalized area of involvement				
Yes	100 (94.34)	6 (5.66)	0.54 (0.10 to 3.04)	0.488
No	128 (95.52)	6 (4.48)	ref.	
Oral ulcer				
Yes	13 (92.86)	1 (7.14)	1.44 (0.08 to 24.94)	0.803
No	215 (95.13)	11 (4.87)	ref.	
Non-scarring alopecia				
Yes	10 (90.91)	1 (9.09)	0.71 (0.02 to 25.09)	0.849
No	218 (95.20)	11 (4.80)	ref.	
Variable	**Non-SLE (n=228)**	**SLE (n=12)**	**OR (95% CI)**	***p*-value**
Leukopenia (WBC < 4,000/mm^3)				
Yes	30 (73.17)	11 (26.83)	113.12 (8.35 to 1531.69)	<0.001
No	198 (99.50)	1 (0.50)	ref.	
Proteinuria				
Yes	5 (55.56)	4 (44.44)	25.83 (1.94 to 343.94)	0.014
No	223 (96.54)	8 (3.46)	ref.	
Baseline ANA titer				
Positive	143 (92.86)	11 (7.14)	4.11 (0.39 to 43.21)	0.239
Negative	85 (98.84)	1 (1.16)	ref.	

Data are presented as number (%), Binary Logistic Regression, *p*-value < 0.05

Binary logistic regression analysis identified leukopenia (OR = 113.12, 95% CI: 8.35–1531.69, $p < 0.001$) and proteinuria (OR = 25.83, 95% CI: 1.94–343.94, $p = 0.014$) as key predictors of SLE progression, underscoring the importance of hematologic and renal abnormalities in early disease transition. Other laboratory parameters, such as low complement levels and the presence of SLE-specific antibodies, were also more prevalent in the SLE group but did not reach statistical significance in logistic regression analysis. as presented in Table 3.

Potential confounding factors may have influenced these findings, including baseline disease severity, follow-up duration, and treatment variations. The small number of SLE cases also limits statistical

power. However, leukopenia and proteinuria are associated with SLE progression, highlighting the importance of regular monitoring in CLE patients with systemic symptoms. Identifying high-risk patients early could allow for closer follow-up and timely intervention to slow disease progression.

4.2 Discussion

This retrospective study aimed to investigate the incidence and factors associated with the progression from cutaneous lupus erythematosus (CLE) to systemic lupus erythematosus (SLE) in patients presenting at the Institute of Dermatology in Thailand over an 8-year period. The findings provide insights into the clinical and laboratory characteristics that may predict the development of SLE in patients initially diagnosed with CLE, particularly focusing on subacute cutaneous lupus erythematosus (SCLE) and chronic cutaneous lupus erythematosus (CCLE).

The study revealed that out of 240 patients with CLE, 12 (5%) developed SLE during the follow-up period. Most of these patients were female (75%) and over 50 years old (50%) (Black et al., 2021; Murphy et al., 2019; Grönhagen et al., 2011). Key findings included a significant association between SLE development and clinical manifestations such as arthralgia, as well as laboratory findings including leukopenia (WBC < 4,000/mm^3) and proteinuria. This study supports previous research showing that women have a higher incidence of SLE, particularly during their reproductive years, with risk increasing with age (Kaul et al., 2016; Petersen et al., 2018). The average time from CLE diagnosis to the development of SLE was 2.09 years, suggesting that the transition from CLE to SLE can occur relatively rapidly. This shows the importance of closely monitoring CLE patients in the early and ongoing stages. The 5% progression rate observed in our findings align with some Asian studies but shows regional variation. South Korea reported a higher rate of 20.8% (Baek et al., 2020), which may reflect genetic differences or variations in healthcare systems. A Thai study found 10% progression rate with milder symptoms (Chanprapaph et al., 2021). Western rates range from 5% to 25%, but differences in genetics, healthcare, and follow-up must be considered (Zhou et al., 2020; Vera-Recabarren et al., 2010).

This study found that patients who developed SLE had generalized and localized lesions equally, inconsistent with previous research indicating that generalized DLE has been previously associated with a higher risk of progressing to SLE than localized DLE (Lee, & Werth, 2018; Clayton, & Sontheimer, 2019), Meanwhile, Vera-Recabarren et al. (2010) reported a 25% progression rate from CLE to SLE, with higher rates in patients with acute CLE (ACLE) compared to chronic CLE (CCLE), and another study found that SCLE had a higher incidence of SLE than DLE (Grönhagen et al., 2011). Some CLE subtypes in our study, like mucosal DLE and lupus profundus, showed no significant link to SLE progression. This could indicate a lower risk of systemic transition, though the small sample size may have limited the detection of meaningful associations.

Genetic, immune, and environmental factors may influence CLE-to-SLE progression. Triggers like UV exposure, infections, and stress have been linked to lupus and could explain geographic differences in progression rates (Black et al., 2021; Grönhagen et al., 2011; Chong et al., 2012). The lower progression rate in our study compared to other Asian cohorts may be due to genetic background, lifestyle, or healthcare access. Further research is needed to identify high-risk patients better and improve early intervention.

Musculoskeletal symptoms, particularly arthralgia ($p < 0.005$) and arthritis ($p = 0.050$), were significantly associated with SLE development. Although arthritis had borderline significance, its clinical relevance should not be dismissed, as it is a common precursor to systemic involvement in SLE. The presence of joint symptoms in CLE patients should, therefore, prompt closer clinical monitoring, which is consistent with the literature indicating that joint pain and arthritis are common early symptoms of SLE (Ameer et al., 2022; Cojocaru et al., 2011; Vera-Recabarren et al., 2010). In addition to musculoskeletal symptoms, laboratory abnormalities were also predictive of SLE progression. Leukopenia was the strongest predictor in our logistic regression analysis (OR = 113.12, 95% CI: 8.35–1531.69, $p < 0.001$), reinforcing its role as an early hematologic indicator of systemic autoimmunity. However, the wide confidence interval suggests a small sample size effect, underscoring the need for larger studies. This finding is supported by previous studies that have identified leukopenia as a common hematologic abnormality in SLE patients (Ameer et al., 2022; Cojocaru et al., 2011). Proteinuria (OR = 25.83, 95% CI: 1.94–343.94, p =

0.014) also proved a significant risk factor. Proteinuria may indicate early renal involvement, which is a hallmark of systemic disease in SLE. The results of the study suggest that routine laboratory tests in CLE patients should be performed to detect abnormalities in the disease at an early stage. The study also found that a positive baseline antinuclear antibody (ANA) test was present in 91.67% of patients who developed SLE, although this was not statistically significant in the logistic regression model. This is consistent with the known high prevalence of ANA positivity in SLE patients (Ameer et al., 2022; Cojocaru et al., 2011). However, the study did not find a significant association between specific ANA patterns or titers and SLE development, which may be due to the small sample size of SLE patients in this study.

This study underscores the need for better screening and monitoring of CLE patients at risk for SLE. We propose a risk-based approach:

1) Routine blood and urine tests – Regular CBC and urinalysis to detect leukopenia and proteinuria, key predictors of SLE.
2) Closer follow-up for musculoskeletal symptoms – Monitor patients with arthralgia or arthritis for early systemic involvement.
3) Risk stratification – A scoring system combining clinical (arthralgia, arthritis) and lab markers (leukopenia, proteinuria, complement levels) to identify high-risk patients.

This study has limitations. The small number of SLE cases may have led to wide confidence intervals and missed risk factors. Retrospective design limited data on medication use and disease severity. Varying follow-up durations and loss to follow-up could underestimate progression because this study was conducted at a tertiary care center. Larger prospective studies with standardized follow-ups are needed to confirm these findings. A risk prediction model should be developed using clinical and laboratory markers to stratify CLE patients by SLE risk, while genetic and environmental studies may clarify CLE-to-SLE progression in Asian populations.

5. Conclusion

This study examined the incidence and factors associated with the progression from CLE to SLE over an 8-year period at the Institute of Dermatology in Thailand. The results showed that 5% of CLE patients developed SLE, with significant associations found between SLE progression and factors such as leukopenia and proteinuria. Chronic CLE, especially discoid lupus erythematosus (DLE), was the most common subtype among those who progressed to SLE, highlighting its potential as a predictor of disease progression. These findings highlight the need for routine laboratory monitoring and risk-based patient stratification to improve early detection and management. Future research should focus on developing a risk prediction tool to assist clinicians in identifying high-risk CLE patients and preventing disease progression. This would ultimately improve patient outcomes and reduce the morbidity associated with SLE.

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