

Clinicopathological Characteristics of Lichenoid Drug Eruption: A 17-Year Retrospective Study

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Purpose: This study aims to characterize the etiology, clinicopathological features, laboratory findings, management strategies, and clinical outcomes of patients diagnosed with lichenoid drug eruption (LDE).

Patients and Methods: A retrospective review was conducted on all biopsy-confirmed LDE cases from January 2008 to October 2024. Data collection included clinical characteristics, causative medications, histopathological as well as laboratory findings, and therapeutic responses.

Results: Of the 34 patients, half were male, with a mean age of 65.1 years. LDE most commonly presented as erythematous to violaceous lesions, appearing as patches and plaques, predominantly on the extremities. Histopathology showed lichenoid infiltrates in all cases, with focal infiltration in 58.8%. Parakeratosis was present in 17.7%. Necrotic keratinocytes were found in the basal layer in 82.4% and in the spinous layer in 38.2% of cases. Eosinophilic infiltration was observed in approximately one-third of patients. The most common offending agents were calcium channel blockers (CCBs) (42.5%), followed by statins (25.8%). The median latency period from drug initiation to symptom onset was 10.1 months (interquartile range [IQR] 3.0–45.5). Discontinuation of the causative agent combined with topical corticosteroid therapy resulted in clinical improvement in 73.5% of patients, with a median resolution time of 56 days (IQR 28.0–77.0).

Conclusion: Antihypertensive drugs, especially CCBs, were the most frequently implicated triggers of LDE. In addition to lichenoid dermatitis, eosinophilic infiltration served as a key histopathological hallmark of this condition. Timely identification and withdrawal of the causative medication, combined with appropriate treatment, are essential for achieving favorable clinical outcomes.

Keywords: adverse drug reactions, calcium channel blockers, drug eruption, histopathology, lichenoid dermatitis, lichen planus

Introduction

Lichenoid drug eruption (LDE) is an uncommon cutaneous adverse drug reaction, accounting for approximately 2–3% of all drug eruptions.¹ It is believed to arise from a T-helper 1 (Th1) cell-/type 1 innate lymphoid cell-driven immune response involving interferon- γ (IFN- γ), which activates cytotoxic CD8⁺ T cells and natural killer cells, ultimately resulting in basal keratinocyte apoptosis.²

Clinically and histologically, LDE can closely mimic idiopathic lichen planus (LP). However, LDE is more likely to manifest with more polymorphic and larger lesions, typically lacking Wickham striae, and displays more prominent desquamation with psoriasiform or eczematous features.³ Additionally, a photodistributed pattern is observed in many patients. Histopathological examination reveals that LDE lesions share certain features with idiopathic LP, complicating differentiation. Nevertheless, the detection of eosinophils, plasma cells, and focal parakeratosis can support a diagnosis of LDE.³ Although LDE is not classified as a severe cutaneous adverse drug reaction, it can significantly impair quality of life and may result in chronic pigmentary changes. Pruritus, one of its most prominent symptoms, can further exacerbate this impact.⁴ Diagnosis remains challenging due to similarities in clinical and histological presentations with classic LP, diverse underlying causes, and difficulties in identifying the offending medication, particularly given the often prolonged latency period and delayed resolution even after drug withdrawal.⁵ To date, there are no standardized clinical or histological criteria for diagnosing LDE, and studies on this condition remains limited.⁶

In the present study, we aimed to evaluate the etiology, clinical and histopathologic features, treatment outcomes, and prognosis of LDE through a 17-year retrospective analysis of histologically verified cases.

Materials and Methods

Study Design

A retrospective cohort study was conducted at the outpatient dermatology clinic of a university-affiliated hospital (Ramathibodi Hospital, Mahidol University, Bangkok, Thailand). Medical records of all patients with histopathologically established LDE, diagnosed between January 2008 and October 2024, were thoroughly reviewed and analyzed. This study was approved by the Institutional Review Board of Mahidol University, which waived the requirement for informed consent for the review of medical records (Protocol number MURA2023/700). All patient data were anonymized prior to analysis to ensure confidentiality. The study was conducted in accordance with the principles outlined in the Declaration of Helsinki and complied with all relevant guidelines and regulations regarding the ethical use of human data.

Study Population

Patients aged 18 years old or older with both clinical and histological diagnosis indicative of LDE were included. Exclusion criteria comprised incomplete medical or histological records, unavailable biopsy specimens for re-evaluation, coexisting hepatitis B, hepatitis C, or HIV infection, and the presence of hair or nail findings suggestive of idiopathic LP.

Data Collection

Demographic data were systematically collected from electronic medical records using a case record form, and encompassed age, sex, underlying diseases, and current medications. Clinical characteristics (eg rash onset, lesion color, anatomical locations, distribution pattern) were documented, as well as suspected culprit medications, history of drug allergies (including type and severity of reactions), latency period, time to improvement, and treatment administered.

Histopathological features were recorded and included epidermal changes (ie normal, atrophy, hyperplasia, parakeratosis, compact hyperkeratosis, hypergranulosis, and hypogranulosis) and dermal characteristics including type of interface changes, location of necrotic keratinocytes, distribution and pattern of inflammatory infiltrates, cytologic composition, and degree of pigmentary incontinence. The degree of pigmentary incontinence was classified as mild (<10 melanophages/high power field [HPF]), moderate (10–20 melanophages/HPF), and severe (>20 melanophages/HPF).⁷ Basic investigations such as complete blood count, renal and hepatic function tests, hepatitis B and C profiles) were performed. Antinuclear antibody (ANA) testing was carried out in cases suspected of photoaggravated autoimmune disease.

To identify the potential causative agent(s), Naranjo algorithm⁸ was applied. Medications with scores that falling into the “probable” or “definite” categories were considered likely culprits. Patch testing, phototesting, and photopatch testing were also conducted when clinically indicated.

Statistical Analysis

All statistical analyses were executed using STATA version 18.0 (StataCorp LLC, College Station, TX, USA). Descriptive statistics were used to summarize demographic and clinical data. Continuous variables were presented as means with standard deviations or medians with ranges, depending on data distribution. Categorical variables were reported as frequencies and percentages.

Results

Patient Demographics

Of the 37 patients diagnosed with LDE, 3 patients were excluded due to HIV infection. A total of 34 patients were included in the study (Table 1). The mean age at onset was 65.1 ($\pm$ 14.3) years old. Of these, half (17/34) of the patients were male. The most common comorbidities were hypertension (64.7%) and dyslipidemia (61.8%), followed by diabetes

mellitus (35.3%). The median onset of the cutaneous lesions was 2 months (interquartile range [IQR] 1.0–6.0; range 0.2–133.8 months). The latency period (defined as the duration between drug exposure and the onset of symptoms) ranged from 0.4 to 120 months with a median of 10.1 months (IQR 3.0–45.5).

Clinical Features and Distribution Patterns

Regarding clinical presentation, the most common lesion morphology was patches (67.7%), followed by plaques (47.1%), macules (32.4%), and papules (23.5%), respectively (Table 1). Erythematous to purplish discoloration was the most frequently observed, present in 61.8% of patients, while greyish and brownish hues were noted in 58.8% and 44.1%, respectively. All patients experienced pruritus. In terms of lesion distribution, lesions were predominantly

Table 1 Demographic and Clinical Characteristics of Patients Diagnosed with Lichenoid Drug Eruption

Variables	n = 34
Age at onset, mean $\pm$ SD, years	65.1 $\pm$ 14.3
Sex, n (%)	
• Male	17 (50.0)
• Female	17 (50.0)
Comorbidities, n (%)	
• Hypertension	22 (64.7)
• Dyslipidemia	21 (61.8)
• Diabetes mellitus	12 (35.3)
Duration of rash, median (IQR), months	2 (1.0–6.0)
Latency period of culprit drug(s), median, (IQR), months	10.1 (3.0–45.5)
Morphology of rash, n (%)	
• Macules	11 (32.4)
• Papules	8 (23.5)
• Patches	23 (67.7)
• Plaques	16 (47.1)
Color of rash, n (%)	
• Erythematous to purplish	21 (61.8)
• Greyish	20 (58.8)
• Brownish	15 (44.1)
Distribution of rash, n (%)	
• Localized	20 (58.8)
• Generalized	12 (35.3)
• Photodistributed	2 (5.9)
Location of rash, n (%)	
• Face	6 (17.6)
• Neck	19 (55.9)
• Trunk	18 (52.9)
• Axillae	2 (5.9)
• Extremities	25 (73.5)
• Hands and feet	3 (8.8)
• Hair and nails	1 (2.9)
• Mucosa	2 (5.9)

Abbreviations: IQR, interquartile range; n, number; SD, standard deviation.



Figure 1 Clinical feature of lichenoid drug eruption: multiple greyish patches localized on the lower extremities.

localized (58.8%), with generalized and photodistributed patterns seen in 35.3% and 5.9% of cases, respectively. The extremities were the most commonly affected sites, accounting for 73.5%, followed by the neck (55.9%) and trunk (52.9%). Mucosal involvement was detected in two patients (5.9%), affecting both the oral and genital mucosa. [Figure 1](#) illustrates a typical clinical presentation of lichenoid drug eruption, showing multiple greyish patches localized on the lower extremities.

Histopathological Findings

The histopathologic findings of LDE are summarized in [Table 2](#). Regarding epidermal changes, epidermal hyperplasia, hypergranulosis, and parakeratosis were revealed in 50.0%, 44.1%, and 17.7% of cases, respectively. All biopsy specimens demonstrated lichenoid infiltrates, with the majority displaying a focal pattern (58.8%), while the remainder showed band-like lichenoid infiltrates. Lymphocytic infiltrates were present in all cases (100.0%), and eosinophilic

Table 2 Histological Features of Lichenoid Drug Eruption

Variables	n = 34
Epidermis, n (%)	9 (26.5)
• Normal	
• Atrophy	7 (20.6)
• Hyperplasia	17 (50.0)
• Parakeratosis	6 (17.7)
• Compact hyperkeratosis	19 (55.9)
• Focal hypergranulosis	15 (44.1)
• Focal hypogranulosis	1 (2.9)
Dermal infiltrate, n (%)	
• Lichenoid infiltration	
– Focal lichenoid infiltration	20 (58.8)
– Band-like lichenoid infiltration	14 (41.2)
Inflammatory cells, n (%)	
• Lymphocytes	34 (100.0)
• Eosinophils	11 (32.6)
• Plasma cells	3 (8.8)
• Red blood cells	9 (26.5)

(Continued)

Table 2 (Continued).

Variables	n = 34
Dyskeratosis, n (%)	
• Stratum basale	28 (82.4)
• Stratum spinulosum	13 (38.2)
• Stratum granulosum	1 (2.9)
• Stratum corneum	1 (2.9)
Pigmentary incontinence, n (%)	
• Mild (<10 cells/HPF) ^a	7 (20.6)
• Moderate (10–20 cells/HPF) ^a	15 (44.1)
• Severe (>20 cells/HPF) ^a	12 (35.3)

Note: ^a number of melanophages.

Abbreviations: HPF, high power field; n, number.

infiltration (Figure 2) was noted in approximately one-third of patients (32.6%). Necrotic keratinocytes were predominantly found in the basal layer (82.4%) and, to a lesser extent, in the spinous layer (38.2%). Pigmentary incontinence was identified in varying degrees: mild in 20.6% of cases, moderate in 44.1%, and severe in 35.3%.

Laboratory Investigations

Basic investigations, including complete blood count, renal and hepatic function tests, and hepatitis B and C serologies, revealed no significant abnormalities. Antinuclear antibody (ANA) testing was evaluated in four patients who were suspected of having photoaggravated autoimmune disease. Three of these cases yielded positive ANA results. Among them, two patients had known underlying autoimmune conditions; one with undifferentiated spondyloarthropathy and another with systemic sclerosis coexisting with Sjögren's syndrome. The other ANA-positive patient had a history of hypertension, however, further investigations for autoimmune diseases were negative.

Phototesting was evaluated in one of two patients who manifested a photodistributed pattern, showing normal responses to visible light, ultraviolet A, and ultraviolet B. Two patients underwent patch testing. One case demonstrated a positive reaction to benzocaine, which was considered not relevant. The other reacted to thimerosal (non-relevant) and nickel, which was deemed past relevant. Photoprovocation and photopatch testing were not conducted, as there was no clinical suspicion of photoallergic or phototoxic reactions in these cases.

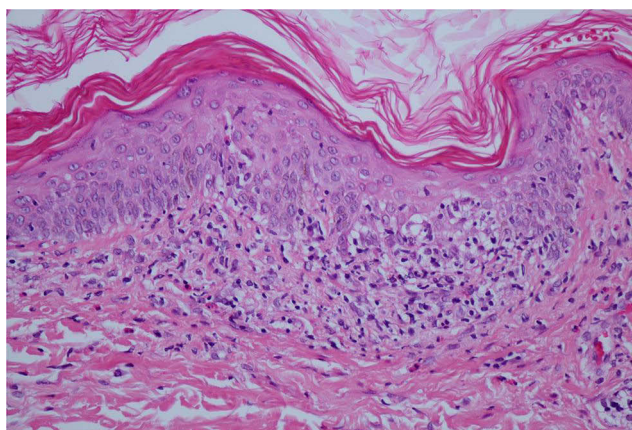


Figure 2 Histopathology of lichenoid drug eruption: focal lichenoid infiltrate of mainly lymphocytes admixed with eosinophils in associated with scattered basal necrotic keratinocytes and focal parakeratosis (Hematoxylin and Eosin stain, x400).

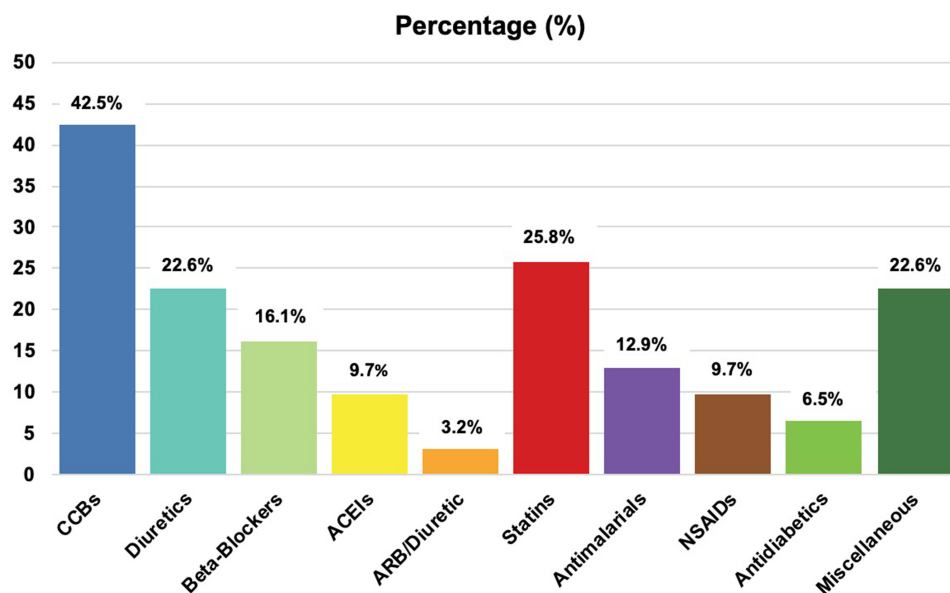


Figure 3 Frequencies of causative drugs associated with lichenoid drug eruption.

Causative Medications

Figure 3 illustrates the frequencies of causative agents of LDE in this study. Antihypertensive drugs were the most frequently implicated, with calcium channel blockers (CCBs) being the most common within this group (42.5%), followed by diuretics (22.6%), beta-blockers (16.1%), angiotensin-converting enzyme inhibitors (ACEIs) (9.7%), and angiotensin receptor blocker (ARB)/diuretic combination (3.2%). Statins ranked second among the associated drug classes, accounting for 25.8% of cases, followed by antimalarial agents (12.9%), nonsteroidal anti-inflammatory drugs (NSAIDs) (9.7%), and antidiabetic drugs (6.5%). Miscellaneous agents were documented in 22.6% of cases and included carboplatin/paclitaxel, allopurinol, sulfasalazine, vitamin C, Thai herbal remedies, Chinese herbal medications, and traditional herbal pills. Drug causality was assessed using the Naranjo algorithm with all cases classified as “probable” adverse drug reactions.

Treatment and Outcomes

All patients received topical corticosteroids as the mainstay of treatment. In addition, two patients (5.9%) were treated with a short course of systemic corticosteroids. Suspected causative drugs were withdrawn in 55.9% of patients. Clinical improvement was established in 73.5% of patients, with a median time to improvement of 56 days (IQR 28.0–77.0).

Discussion

LDE is a relatively rare, immune-mediated cutaneous adverse reaction. It was first described in 1929 as a response to arsenic, which was used for the treatment of syphilis.⁹ Since then, an increasing number of cases have been reported, helping to establish LDE as a distinct clinical entity. The pathogenesis is hypothesized to involve cross-reactivity between the drug and keratinocytes both acting as antigens,⁵ leading to T-cell mediated autoimmune damage of basal keratinocytes. In addition, CD8+ T cells, plasmacytoid dendritic cells, and granzyme B-containing cells are believed to contribute to the inflammatory process.^{5,10,11} In spite of these insights, the exact mechanisms remain incompletely understood.

This 17-year retrospective study details the clinical and histopathologic characteristics of one of the largest series of biopsy-confirmed LDE cases to date. The findings highlight CCBs as the most frequent causative agents and identified eosinophilic infiltration in addition to focal lichenoid dermatitis as a key histologic feature that aids in differentiating LDE from idiopathic LP. The prolonged latency period in our cohort underscores the diagnostic challenges and the

importance of obtaining a comprehensive and detailed medication history in affected patients. These insights may guide clinical practice, as well as aid in diagnosis and in the identification of causative agents.

A total of 34 patients with LDE were evaluated with a mean age at onset was 65.1 years. The results were comparable to the findings of Halevy et al³ who also noted that the mean age of affected patients in their study was 66 years. While some studies have disclosed a male predominance,⁵ and others a female predominance,¹² our study found an equal distribution between males and females.

The present study revealed a latency time with a median of 10.1 months which was longer than previously published data.^{1,5} A study by Maul et al, in which immune checkpoint inhibitors (ICIs) were the most common culprit drugs, reported a latency of 15.7 weeks (range 0.1–208 weeks). Conversely, Bhanja et al, whose study primarily involved antihypertensive agents, found a shorter latency period of 2.2 months (range 15 days–6 months). These discrepancies may be attributed to differences in drug types, dosage, concurrent medications, and individual host predisposition.⁵ Despite the longer latency period in our cohort, the median time to clinical improvement in our study aligned with findings in prior studies.

Regarding clinical features, most of the patients in our study presented with erythematous to purplish patches. The majority of patients (58.8%) exhibited a localized distribution predominantly affecting the extremities (73.5%), followed by the neck (55.9%), and trunk (52.9%). These findings were in line with previous studies that also reported a localized involvement, particularly on the extremities.^{5,12} However, Bhanja et al described a generalized and photodistributed pattern involving face, trunk and extremities in their study.¹ The literature suggests that LDE tends to have more polymorphic, psoriasiform or eczematous morphology, with more pronounced desquamation, typically lacks of Wickham striae,^{1,3,9,13,14} and usually spares mucosal sites as well as hairs and nails,¹⁵ when compared to idiopathic LP.¹² Our results were consistent with these previous observations.^{1,3,9,13,14}

We observed photodistributed involvement in two patients, associated with manidipine and ibuprofen. Similar lesion patterns have been reported in the literature,^{3,16} with common triggers including hydrochlorothiazide, enalapril, quinapril, naproxen, ketorolac, piroxicam, and carbamazepine.¹⁷ Persistent photosensitivity may result from prolonged drug retention in the skin or the activation of endogenous photosensitizers, which can generate photoantigens following prior photoallergic reactions.^{17,18} Although a phototest was performed in one patient with NSAID-induced LDE and yield negative result, photoprotection was recommended for all affected patients.

Regarding histopathology, although histopathologic features may vary depending on the biopsy stage, eosinophilic infiltration has been suggested as a characteristic finding in LDE.^{10,19} In our study, eosinophilic infiltrates were observed in a subset of cases, supporting its potential role as a diagnostic clue. In our study, other key histopathological findings included epidermal hyperplasia, focal hypergranulosis, focal lichenoid infiltration, necrotic keratinocytes in the stratum basale and moderate pigmentary incontinence. Notably, the presence of focal parakeratosis seen in our study was consistent with the findings by Van den Haute et al.¹⁹ In contrast to previous studies, we identified only one case of focal disruption in the granular layer, and did not find infiltration around deep vessels, increased number of clusters of necrotic keratinocytes, or the presence of plasma cells, as indicated in earlier studies.^{10,19,20}

A 2022 literature review, analyzing 323 LDE cases from 163 published reports, identified ICIs as the most frequent culprit drugs (42.1%), followed by tyrosine kinase inhibitors (TKIs) (12.0%), and anti-TNF alpha agents (4.0%).⁵ Before the introduction of biologics, LDE was consistently associated with the intake of gold salts.²¹ Other common culprits included ACEIs, beta-blockers, methyldopa, antimalarials, and diuretics.⁵ In our study, antihypertensive drugs were the most frequent culprits, particularly CCBs, followed by statins. These findings were similar to the previous reports.^{1,22} The absence of ICI-associated LDE cases in the present study may reflect lower prescription rates, underdiagnosis, or underreporting. Likewise, genetic and immunologic variations among populations may contribute to variations in adverse event profiles.²³

Although no specific therapy exists for LDE, various treatment options have been described, including topical urea, topical corticosteroids, topical antipruritics (eg menthol, camphor, polidocanol), oral retinoids, and methotrexate.^{24–26} In our study, all 34 patients were treated with topical corticosteroids, while 2 patients (5.9%) also received systemic corticosteroids. The culprit drugs were ceased in 55.9% of patients, however, continued in 44.1% of patients due to

treatment necessity. Overall, the majority of patients (73.5%) showed clinical improvement, including 8 cases (23.5%) who improved despite continued exposure to the suspected drugs. This improvement even with ongoing use of the culprit drug may be attributed to several factors. One possibility is the development of drug tolerance, in which hypersensitivity responses diminish over time.^{27,28} Moreover, corticosteroid therapy may effectively suppress the localized immune reaction, even in the continued presence of the offending agents.²⁹

Our study has some limitations, including a retrospective design, a relatively limited sample size due to the rarity of this condition, and a single-center study. Additionally, we lacked of data on *in-vivo* and *in-vitro* testing for culprit drug identification. Future prospective multi-center study with larger cohorts and comprehensive drug testing would shed more light on this rare cutaneous adverse drug reaction.

Conclusions

LDE commonly presented as erythematous to purplish patches on the extremities. Although rare and benign, it could significantly impact the quality of life. In our study, the most common triggers were calcium channel blockers and statins. Accurate diagnosis relies on clinical and histopathological correlation, with eosinophilic infiltration serving as a key histologic feature. Early recognition, prompt discontinuation of the offending drug along with appropriate therapy are crucial for optimal outcomes.

Abbreviations

ACEI, angiotensin-converting enzyme inhibitor; ANA, antinuclear antibody; ARB, angiotensin receptor blocker; CCB, calcium channel blocker; HIV, human immunodeficiency virus; HPF, high power field; ICI, immune checkpoint inhibitor; IFN- γ , interferon- γ ; IQR, interquartile range; LDE, lichenoid drug eruption; LP, lichen planus; NSAID, nonsteroidal anti-inflammatory drug; Th1, T-helper 1 cell; TKI, tyrosine kinase inhibitor.

Data Sharing Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Ethics Approval and Informed Consent

This study was approved by the Institutional Review Board of Mahidol University, which waived the requirement for informed consent for the review of medical records (Protocol number MURA2023/700). All patient data were anonymized prior to analysis to ensure confidentiality. The study was conducted in accordance with the principles outlined in the Declaration of Helsinki and complied with all relevant guidelines and regulations regarding the ethical use of human data.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Funding

The authors received financial support from Faculty of Medicine Ramathibodi Hospital, Mahidol University.

Disclosure

The authors report no conflicts of interest in this work.

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