Lichenoid granulomatous dermatitis revisited: A retrospective case series

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Background: Lichenoid granulomatous dermatitis (LGD) is an uncommon reaction pattern for which clinical correlates can be difficult to establish. LGD combines vacuolar degeneration with variable types of granulomas.

Objective: To determine clinical correlates of LGD.

Methods: The laboratory information systems at the University of Florida, the Medical College of Wisconsin, and Inform Diagnostics Research Institute were queried to identify 56 cases of LGD. Cases were reviewed for information regarding eosinophils, plasma cells, deep perivascular infiltrates, granuloma subtype, parakeratosis, epidermal atrophy, psoriasiform epidermal changes, pseudoepitheliomatous hyperplasia, periadnexal inflammation, vasculitis, and red blood cell extravasation.

Results: The most common clinical correlates were drug eruption (39.3%, n = 22) and lichenoid keratosis (19.6%, n = 11). Tattoo reaction, postherpetic dermatitis, and scabies or postscabietic dermatitis each accounted for 7.1% (n = 4) of cases. Pigmented purpuric dermatosis and lichen striatus each accounted for 5.4% (n = 3) of cases. Dermal eosinophils (P = .005) and psoriasiform epidermal changes (P = .055) were associated with drug hypersensitivity. Perineural (P = .049) and perifollicular (P = .003) inflammation were associated with tattoo reaction and postherpetic dermatitis. Red blood cell extravasation was helpful in cases of pigmented purpuric dermatosis (P = .049).

Limitations: This study is limited by its retrospective nature and statistical power.

Conclusion: Dermal eosinophilia, psoriasiform epidermal changes, periadnexal inflammation, and red blood cell extravasation might aid in the clinical diagnosis of patients with LGD. (J Am Acad Dermatol 2019;81:1157-64.)

Key words: drug eruption; giant cell lichenoid dermatitis; granulomatous dermatitis; lichenoid dermatitis; lichenoid granulomatous dermatitis; postherpetic dermatitis.

Lichenoid granulomatous dermatitis (LGD), also referred to as giant cell lichenoid dermatitis, is an uncommon tissue reaction pattern that manifests as an interface reaction with band-like lymphocytic infiltrates in close apposition to the epidermis and granulomatous inflammation in the dermis. LGD was first reported in 1986 by Gonzalez et al and later described in a series of 40 patients in 2000 by Magro and Crowson.[1,2] LGD has been associated with drug eruptions, cutaneous T-cell

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lymphoma (CTCL), hepatobiliary disorders, rheumatoid arthritis, id reactions to antecedent viral infections, and active infections (mycobacteria, fungi, syphilis).[1,2] Histologic features that suggest specific clinical diagnoses would be helpful to dermatopathologists who encounter LGD. In this study (the largest series of LGD to date), dermatopathologists correlated histologic features with demographics, morphology, distribution of lesions, and medication history to identify the most common clinical causes of LGD. We also reviewed the literature describing this unusual pattern.

CAPSULE SUMMARY

- Drug-induced hypersensitivity, lichenoid keratosis, and nondrug induced hypersensitivity (tattoo reaction, postherpetic dermatitis, scabies and postscabietic dermatitis) account for the majority of clinical correlates of lichenoid granulomatous dermatitis.
- Dermal eosinophils, psoriasiform changes, periadnexal inflammation, and red blood cell extravasation aid in the diagnosis of lichenoid granulomatous dermatitis. Nondrug-induced hypersensitivity and pigmented purpuric dermatosis were previously unrecognized causes.

MATERIALS AND METHODS

After institutional review board approval, we queried the electronic pathology databases of the University of Florida, Medical College of Wisconsin, and Inform Diagnostics Research Institute for descriptive diagnoses of LGD rendered during February 2013-February 2018. Queries were performed for key words "lichenoid" and "granulomatous" in diagnostic reports at the University of Florida and for "lichenoid granulomatous dermatitis" at Inform Diagnostics Research Institute. In total, 56 cases were identified that demonstrated the following 3 features: band-like inflammation of lymphocytic infiltrates in the papillary dermis, interface tissue reaction with basilar keratinocyte apoptosis, and groups of histiocytes with or without giant cells either admixed with the lichenoid inflammatory infiltrate or in the mid to reticular dermis. Cases meeting the inclusion criteria were evaluated (by Dr Motaparthi) by polarized microscopy; acid-fast, Fite-Faraco, Gömöri trichome, Grocott methenamine silver, and periodic acid–Schiff stains; and an immunohistochemical stain for *Treponema pallidum* antibody (APA135AA, Biocare Medical, Pacheco, CA). Cases lacking all 3 defining and requisite histologic features, available clinical data, or sufficient tissue blocks for evaluation by special and immunohistochemical stains were excluded.

Three dermatopathologists (Dr Motaparthi, Dr Walker, Dr Sokumbi) reviewed and recorded the following histologic parameters: frequency of eosinophils and plasma cells (absent, rare or scattered, numerous or in aggregates [>3 cells]), subtype of granuloma (sarcoidal, diffuse or interstitial, suppurative, necrotizing tuberculoid, nonnecrotizing tuberculoid, palisaded with or without necrobiosis), deep perivascular infiltrates, parakeratosis, epidermal atrophy, psoriasiform epidermal changes (confluent parakeratosis with hypogranulosis, intracorneal or intraepidermal neutrophilic microabscesses, regular epidermal hyperplasia), pseudoepitheliomatous hyperplasia, periadnexal (perifollicular, perineural, perieccrine) inflammation, extravasated red blood cells, and vasculitis. These histologic parameters were selected because they were previously reported as clinical correlates of LGD, including lichenoid drug eruptions, infections, hepatobiliary diseases, and CTCL.[2] At the time of histologic review, the dermatopathologists were blinded to the clinical data, including images, at the time of histologic review. Thereafter, the electronic medical records were reviewed (by Dr Motaparthi, Dr Walker, and Dr Sokumbi) for the available clinical data on age, sex, clinical morphology, anatomic distribution, symptoms, duration of clinical findings, and medication history. The clinical differential diagnoses of the treating dermatologists were considered during review of the electronic medical records. A diagnosis that was based on clinicopathologic correlation (CPC) was then rendered by each reviewer independently. After independent review, discordant interpretation was rendered by the reviewers in 4 (7.1%) of 56 cases. In this subset, group review of histologic features and clinical data lead to consensus, with at least 2 of 3 reviewers agreeing on the final CPC diagnosis. In the other 52 cases, there was no discordance among reviewers.

Fisher's exact test was used to examine the association between individual histologic variables and CPC-established diagnoses for the 56 patients with LGD. CPC-established diagnoses were categorized into 3 groups: drug-induced hypersensitivity (DIH), non-DIH, and solitary lichenoid lesions. Cases were designated as unspecified if categorization was unclear. CPC-established diagnoses were classified in this manner with the intent for dermatopathologists

Abbreviations used:

CPC: clinicopathologic correlation
CTCL: cutaneous T-cell lymphoma
DIH: drug-induced hypersensitivity
LGD: lichenoid granulomatous dermatitis
T_H1: T helper 1
T_H2: T helper 2

to provide more helpful information to clinicians who might find an isolated descriptive diagnosis of LGD challenging to correlate. Statistical significance between the histologic variables and the proportion of cases in each CPC category was determined. Two-tailed *P* values <.05 were deemed statistically significant. The open source statistical software package R (version 3.5.0) was used for all analyses performed. For the literature review, PubMed and Google Scholar were searched for the terms "lichenoid granulomatous dermatitis" and "giant cell lichenoid dermatitis." Publications were reviewed with an emphasis on the following: number of cases, demographic information, distribution and morphology of lesions, clinical differential diagnoses, histologic features, and ultimate clinical diagnosis.

RESULTS

In total, 56 cases of LGD met inclusion criteria. The CPC diagnoses were made by the investigators using information gathered from the electronic health records. The clinical differential diagnoses of the treating clinicians are summarized in Table I. The most common diagnoses were drug eruption (39.3%, 22/56) and lichenoid keratosis (19.6%, 11/56). Tattoo reaction, postherpetic dermatitis, and postscabietic dermatitis each accounted for 7.1% (4/56) of cases. Pigmented purpuric dermatosis (n = 3) and lichen striatus or acquired blaschkoid dermatitis (n = 3) each accounted for 5.4% of cases. Actinic granuloma (n = 2), contact dermatitis (n = 2), and Flegel disease (n = 1) were uncommon. Overall, male and female patients were equally represented, with a mean age of 57.7 (standard deviation 17.0, range 13-93) years.

Within the CPC diagnostic categories, the DIH group (n = 22) included cases of drug eruption; the non-DIH group (n = 14) included cases of lichenoid contact dermatitis, scabies and postscabietic dermatitis, postherpetic dermatitis, and tattoo reactions; the solitary lichenoid lesion group (n = 14) included cases of lichenoid keratosis, lichen striatus, and acquired blaschkoid dermatitis; and the unspecified group (n = 6) included cases of actinic granuloma, Flegel disease, and pigmented purpuric dermatosis. In this study, no cases of infection or lymphoma were identified upon query of the laboratory information system. Dermal eosinophils (numerous or in aggregates, $P = .005$) was significantly associated with DIH. Psoriasiform epidermal changes were associated with DIH, but this finding demonstrated only borderline statistical significance ($P = .055$). Perineural ($P = .049$) and perifollicular ($P = .003$) inflammation were significantly associated with non-DIH. Red blood cell extravasation ($P = .049$) was significantly associated with unspecified cases. Histologic variables and statistical analyses are summarized in Table II.

Table III (available at https://data.mendeley.com/datasets/ccpnbbm3rc/1) summarizes the literature review.[1-13] Most reports of LGD have been anecdotal, and before this publication, Magro and Crowson[2] had described the largest series (n = 40) of LGD cases. In that study, 35% of cases represented drug eruptions; id reactions (to herpes zoster, Epstein-Barr virus), active infections (*Mycobacterium tuberculosis, M. leprae,* fungi, syphilis), CTCL, hepatobiliary diseases, rheumatoid arthritis, and idiopathic lichenoid eruptions (lichen planus, lichen nitidus, lichen striatus, lichen planopilaris) accounted for the remainder.[2] Drug eruption,[1,4,6,7,9] postherpetic dermatitis,[5] mycobacterial infection,[8] and CTCL[12,13] were described in other series and reports of LGD. In contrast, fungal and bacterial infections and cutaneous lymphomas were not identified in this series.

DISCUSSION

Before this study, lichenoid keratosis (19.6%, n = 11), tattoo reaction (7.1%, n = 4), scabies or postscabietic dermatitis (7.1%, n = 4), pigmented purpuric dermatosis (5.4%, n = 3), actinic granuloma, lichenoid contact dermatitis (3.6%, n = 2), and Flegel disease (3.6%, n = 2) had not been described in association with LGD. In concordance with the prior literature, drug eruption (DIH) was the most common clinical diagnosis associated with LGD in this study. The most commonly implicated medications were antihypertensive agents: beta-blockers (atenolol, sotalol, metoprolol, n = 5), calcium-channel blockers (amlodipine, verapamil, diltiazem, n = 5), and angiotensin-converting—enzyme inhibitors, and angiotensin-receptor blockers (lisinopril, benazepril, losartan, n = 4). Proton-pump inhibitors (esomeprazole, omeprazole, pantoprazole), statins (atorvastatin, pravastatin), tumor necrosis factor—α inhibitors (infliximab, adalimumab), selective-serotonin reuptake inhibitors (escitalopram, fluoxetine, sertraline), nonsteroidal anti-inflammatory drugs (celecoxib, meloxicam), hydrochlorothiazide, and hypoglycemic drugs

Table I. Clinical diagnoses associated with lichenoid granulomatous dermatitis in this series

CPC diagnosis	Frequency, n (%)	Age, y, mean ± SD (range)	Sex, M:F	Clinical morphology	Clinical differential diagnoses
Drug eruption	22 (39.3)	65.5 ± 11.6 (53-90)	1:1.2	Hyperpigmented or violaceous macules or papules, salmon-colored to erythematous plaques with silvery scale, annular patches or plaques, erythroderma	Psoriasis, LP, MF, CLE, dermatophytosis, LDE, eczema/atopic dermatitis, vasculitis, pigmented purpuric dermatosis
Lichenoid keratosis	11 (19.6)	56.7 ± 10.8 (41-77)	1.2:1	Pink-red papules or plaques with or without scale	BCC, SCC, PN, DF, ISK, LK, arthropod bite reaction, AK
Tattoo reaction	4 (7.1)	47.8 ± 17.6 (25-72)	1:1	Erythematous or violaceous papules within tattoo	Allergic contact dermatitis, sarcoidosis, dermatophytosis, folliculitis
Postherpetic dermatitis	4 (7.1)	60.8 ± 26.1 (22-93)	1:3	Grouped papules on erythematous base, unilateral or dermatomal papules	Herpes simplex virus, varicella-zoster virus
Scabies and post-scabietic dermatitis	4 (7.1)	44 ± 9.2 (31-57)	3:1	Excoriated papules at acral sites	Scabies, granuloma annulare
Pigmented purpuric dermatosis	3 (5.4)	56.7 ± 6.3 (48-63)	1:2	Annular purpuric patch, nonpalpable purpura	Vasculitis, pigmented purpuric dermatosis
Lichen striatus and acquired blaschkoid dermatitis	3 (5.4)	33.3 ± 28.1 (13-73)	2:1	Linear papules or depigmented macules	Vitiligo, postinflammatory pigment alteration, linear psoriasis
Actinic granuloma	2 (3.6)	58.5 ± 8.5 (50-67)	1:1	Annular plaques with central atrophy	Granuloma annulare, necrobiosis lipoidica
Contact dermatitis	2 (3.6)	45.5 ± 9.5 (36-55)	1:1	Geometric plaques at site of patch testing	Allergic contact dermatitis
Flegel disease	1 (1.8)	80	1:0	Scaly papules	Porokeratosis

AK, Actinic keratosis; *BCC*, basal cell carcinoma; *CLE*, cutaneous lupus erythematosus; *CPC*, clinicopathologic correlation; *DF*, dermatofibroma; *ISK*, irritated seborrheic keratosis; *LDE*, lichenoid drug eruption; *LK*, lichenoid keratosis; *LP*, lichen planus; *MF*, mycosis fungoides; *PN*, prurigo nodule; *SCC*, squamous cell carcinoma; *SD*, standard deviation.

Table II. Histologic features and summary of statistical analysis (Fisher's exact test)

Histologic feature	DIH, n (%)	Non-DIH, n (%)	SLL, n (%)	Unspecified, n (%)	*P* value (significant association, if present)
Eosinophils*	9 (40.9)	1 (7.1)	0	0	**.005** (DIH)
Plasma cells*	10 (45.5)	4 (28.6)	5 (35.7)	2 (33.3)	.805
Deep perivascular infiltrates	10 (45.5)	6 (42.9)	4 (28.9)	2 (33.3)	.792
Interstitial or diffuse granulomas	17 (77.3)	12 (85.7)	10 (71.4)	4 (66.7)	.732
Parakeratosis	17 (77.3)	11 (78.6)	9 (64.3)	3 (50)	.504
Epidermal atrophy	6 (27.3)	4 (28.6)	0	1 (16.7)	.139
Psoriasiform epidermal changes	5 (22.7)	0	0	0	**.055** (DIH)†
Pseudoepitheliomatous hyperplasia	5 (22.7)	1 (7.1)	2 (14.3)	1 (16.7)	.693
Perineural inflammation	4 (18.2)	5 (35.7)	0	0	**.049** (non-DIH)
Perifollicular inflammation	0	6 (42.9)	3 (21.4)	0	**.003** (non-DIH)
Perieccrine inflammation	3 (13.6)	3 (21.4)	3 (21.4)	0	.719
Red blood cell extravasation	1 (4.5)	1 (7.1)	2 (14.3)	3 (50)	**.049** (unspecified)
Vasculitis	2 (9.1)	0	0	0	.600

Bolded values are statistically significant.
DIH, Drug-induced hypersensitivity; *SLL*, solitary lichenoid lesion.
*Numerous or aggregates (>3 cells).
†Borderline statistical significance.

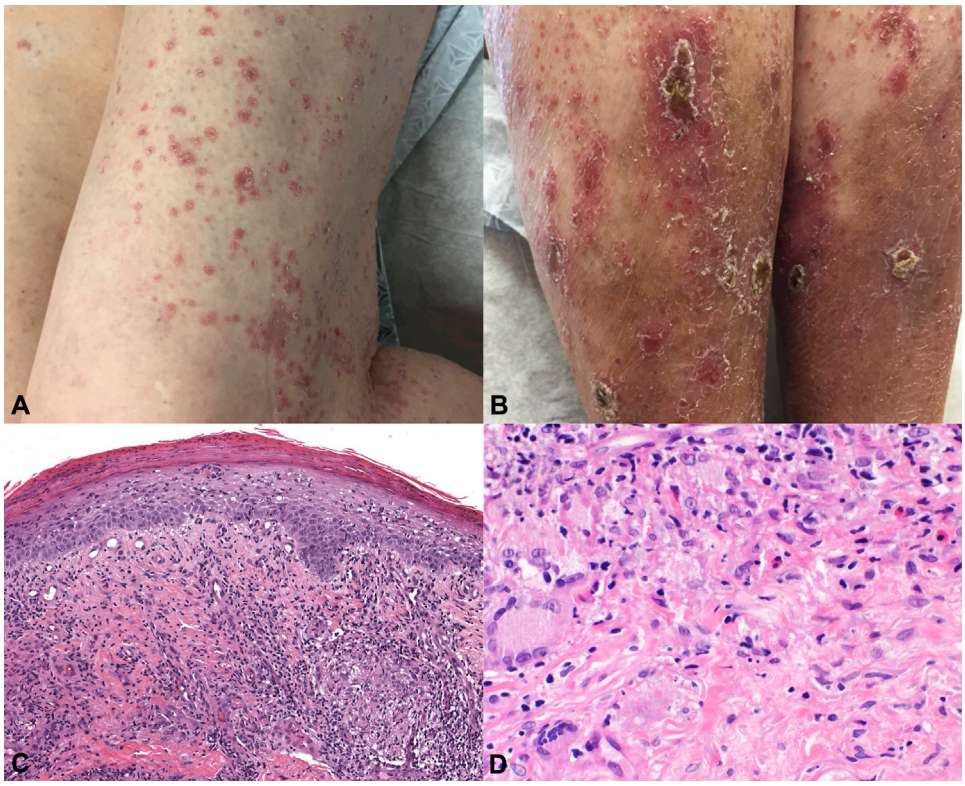

Fig 1. Lichenoid granulomatous drug eruption. **A** and **B**, Psoriasiform papules and plaques on the lower extremities of patients with recent history of tumor necrosis factor α inhibitor (adalimumab) administration. **C**, Confluent parakeratosis with neutrophils, vacuolar changes, and apoptotic keratinocytes overlying aggregates of tuberculoid granulomas. **D**, Scattered eosinophils adjacent to Langerhans giant cells. (**C** and **D**, Hematoxylin-eosin stain; original magnifications: **C**, ×125; **D**, ×400.)

(metformin, glipizide) were also implicated in the clinical histories. These medications overlap with those reported in the study by Magro and Crowson, which additionally identified antibiotics, antihistamines, and hydroxychloroquine.[2] Numerous or aggregated (>3) dermal eosinophils (P = .005)

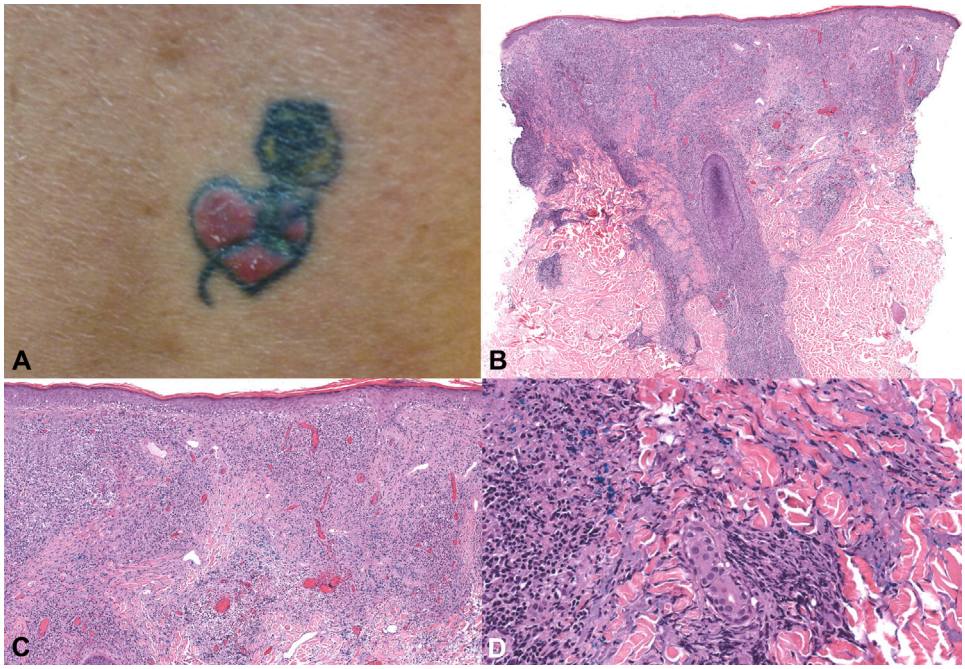

Fig 2. Lichenoid granulomatous tattoo reaction. **A**, Papules and plaques arising within a red tattoo. **B**, Diffuse (interstitial) granulomas, band-like inflammation, and prominent perifollicular inflammation in a patient with a blue tattoo. **C**, Vacuolar change overlying pigment incontinence, diffuse granulomas, and abundant tattoo ink. **D**, Perieccrine interstitial granulomas and tattoo ink. (**B**-**D**, Hematoxylin-eosin stain; original magnifications: **B**, ×40; **C**, ×100; **D**, ×400.)

were specific for drug eruption (DIH) in this study but have also been noted in other examples of drug-induced LGD.[2,9] Psoriasiform epidermal changes (P = .055) have not previously been described in the context of drug-induced LGD (Fig 1).

Perineural (P = .049) infiltrates were significantly associated with non-DIH cases, owing to perineural inflammation in postherpetic dermatitis (3 of 4) and tattoo reaction (2 of 4). Perifollicular (P = .003) inflammation was associated most closely with the non-DIH category and was identified in all cases of tattoo reaction (Fig 2). Although LGD is an established correlate of postherpetic dermatitis,[2,5] the description of this pattern underlying tattoo reaction is novel. Of note, perineural inflammation (Fig 3) is not wholly specific for non-DIH, given its presence in 4 of 22 cases of drug eruption (DIH). Before this study, follicular inflammation and perineural inflammation were described in postherpetic dermatitis.[2]

Red blood cell extravasation (P = .049) was associated with the unspecified category due to the inclusion of pigmented purpuric dermatosis (3 of 3 cases). Leukocytoclastic vasculitis was only identified in drug eruption (DIH, 2 cases), but this finding did not reach statistical significance (P = .600). In contrast, Magro and Crowson identified granulomatous vasculitis in LGD associated with drug hypersensitivity, infectious id reactions and active infections, hepatobiliary diseases, and rheumatoid arthritis.[2] Interstitial or diffuse was the most common subtype of granuloma (76.8%, n = 43), followed by palisaded with necrobiosis (8.9%, n = 5) and nonnecrotizing tuberculoid (8.9%, n = 5). Granuloma subtype did not correlate with a particular diagnostic category. Similarly, perieccrine inflammation did not specify a clinical diagnostic group; Magro and Crowson identified perieccrine inflammation in cases of drug eruption, infectious id and active infection, and hepatobiliary disease.[2] Categoric associations with plasmacellular aggregates, deep perivascular infiltrates, parakeratosis, epidermal atrophy, and pseudoepitheliomatous hyperplasia were also statistically insignificant.

The presence of concomitant lichenoid and granulomatous tissue reaction patterns might result from the recruitment of both cytotoxic CD8 T-cell lymphocytes and histiocytes initiated by self-derived or environmental antigens or superantigens.[14,15] Superantigens, including proteins derived from infectious agents, trigger host T helper 1 (T_H1) responses. This class of antigens is capable of simultaneously binding to the major

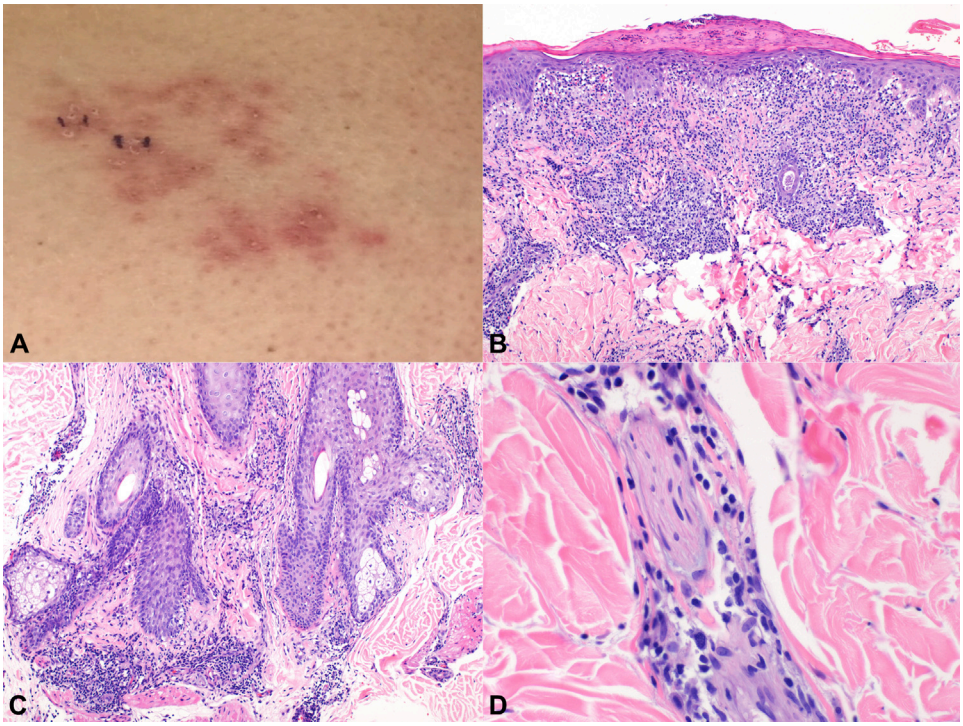

Fig 3. Lichenoid granulomatous postherpetic dermatitis. **A**, Grouped papules arising at the site of recurrent lumbosacral herpes simplex virus infection, with the most recent occurrence 2 weeks earlier. Viral culture was negative for herpes simplex virus growth. **B**, Lichenoid interface dermatitis overlying a diffuse granulomatous infiltrate. Immunostains for herpes simplex viruses 1 and 2 were negative for antigen. **C**, Perifollicular lymphohistiocytic infiltrate. **D**, Mixed perineural infiltrate. (**B**-**D**, Hematoxylin-eosin stain; original magnifications: **B** and **C**, ×100; **D**, ×400.)

histocompatibility complex class II receptor on antigen-presenting cells and T-cell receptor on CD4 T cells. The transduced signal results in the activation and clonal expansion of CD8 T cells and production of interferon γ, which results in the recruitment of macrophages to the skin.[16] Although T_H1 cells comprise the primary helper T-cell subset in the lichenoid tissue reaction, T helper 2 (T_H2) responses can also result in the activation of CD8 T cells.[17] In addition, T_H2 responses result in the production of interleukin 4 and 5, which results in the downstream recruitment of eosinophils to the skin. Thus, dual T_H1 and T_H2 responses to triggering antigens might drive concomitant recruitment of the macrophages and cytotoxic T cells responsible for the vacuolar interface reaction and eosinophils in LGD as typified by drug eruption (DIH).

This study is limited by its retrospective nature and by limited statistical power, given the smaller number of cases within each diagnostic category. As in prior series, data regarding treatment and outcomes is lacking for clinical correlates of LGD. Divergent from prior series, cutaneous lymphoma and active fungal and bacterial infections were not identified. This finding might be attributable to our study methods: cases were selected on the basis of a descriptive histologic diagnosis of LGD, which might have been rendered after exclusion of infectious or lymphoproliferative disorders. In conclusion, the most common clinical diagnoses underlying LGD are drug eruption and lichenoid keratosis. Tattoo reaction, postherpetic dermatitis, scabies or postscabietic dermatitis, and pigmented purpuric dermatosis are relatively common but previously unrecognized clinical correlates of LGD. Actinic granuloma, lichenoid contact dermatitis, and Flegel disease are uncommon causes. Clinical morphology and history, in tandem with attention to dermal eosinophilia, psoriasiform changes, and periadnexal inflammation, permit more specific diagnostic guidance. Numerous or aggregated dermal eosinophils and psoriasiform epidermal changes are relatively specific features indicative of drug eruption. Perineural and perifollicular inflammation are less specific but are associated with non-DIH; perineural inflammation is frequent in postherpetic dermatitis, and both features are common in tattoo reaction.

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REFERENCES

1. Gonzalez JG, Marcus MD, Cruz DJ. Giant cell lichenoid dermatitis. *J Am Acad Dermatol*. 1986;15:87-92.
2. Magro CM, Crowson AN. Lichenoid and granulomatous dermatitis. *Int J Dermatol*. 2000;39:126-133.
3. Viraben R, Dupré A. Lichenoid granulomatous papular dermatosis associated with human immunodeficiency virus infection: an immunohistochemical study. *J Am Acad Dermatol*. 1988;18:1140-1141.
4. Goldberg LJ, Goldberg N, Abrahams I, Silvers DN, Szaniawski W, Halperin AJ. Giant cell lichenoid dermatitis: a possible manifestation of sarcoidosis. *J Cutan Pathol*. 1994;21:47-51.
5. Córdoba S, Fraga J, Bartolomé B, García-Díez A, Fernández-Herrera J. Giant cell lichenoid dermatitis within herpes zoster scars in a bone marrow recipient. *J Cutan Pathol*. 2000;27:255-257.
6. Wolf IH, Smolle J, Cerroni L, Kerl H. Erythroderma with lichenoid granulomatous features induced by erythropoietin. *J Cutan Pathol*. 2005;32:371-374.
7. Ferran M, Gallardo F, Salar A, Iglesias M, Barranco C, Pujol RM. Granulomatous dermatitis with enlarged histiocytes: a characteristic pattern of granulocyte colony-stimulating factor. Report of two cases and review of the literature. *Dermatology*. 2006;212:188-193.
8. Breza TS, Magro CM. Lichenoid and granulomatous dermatitis associated with atypical mycobacterium infections. *J Cutan Pathol*. 2006;33:512-515.
9. Khelifa-Hamdani E, Touati-Serraj M, Perriard J, Chavaz P, Saurat JH, Kaya G. Giant cell lichenoid dermatitis in a patient with baboon syndrome. *J Cutan Pathol*. 2008;35S1:17-19.
10. Bulur I, Gokalp H, Erdem O, Gurer MA. A rare form of lichenoid tissue reaction: lichenoid granulomatous dermatitis. *J Med Cases*. 2015;6:95-97.
11. Ghasemibasir H, Khezrian L, Sobhan MR. Lichenoid and granulomatous dermatitis: report of two cases from Iran. *Iran J Dermatol*. 2015;18:136-139.
12. Garrido MC, Maroñas-Jimenez L, Ortiz PL, Rodriguez-Peralto JL. Lichenoid granulomatous mycosis fungoides. *Am J Dermatopathol*. 2017;39:614-617.
13. Belousova IE, Khairutdinov VR, Bessalova A, Kazakov DV. Mycosis fungoides manifesting as giant cell lichenoid dermatitis. *Am J Dermatopathol*. 2018;40:283-285.
14. Magro CM, Crowson AN. A distinctive cutaneous reaction pattern indicative of infection by reactive arthropathy-associated microbial pathogens: the superantigen ID reaction. *J Cutan Pathol*. 1998;25:538-544.
15. Sontheimer RD. Lichenoid tissue reaction/interface dermatitis: clinical and histological perspectives. *J Invest Dermatol*. 2009;129(5):1088-1099.
16. Mosmann TR, Sad S. The expanding universe of T-cell subsets: Th1, Th2 and more. *Immunol Today*. 1996;17:138-146.
17. Ekkens MJ, Shedlock DJ, Jung E, et al. Th1 and Th2 cells help CD8 T-cell responses. *Infect Immun*. 2007;75(5):2291-2296.