

Lichenoid granulomatous dermatitis: A case with dramatic desquamation and multiple potential causes

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Oluwatosin Odeshi¹, Bret Kenny² , Siddharth Kogilwaimath³, Irina Oroz¹ and Allison Osmond⁴

Abstract

Lichenoid granulomatous dermatitis, also known as giant cell lichenoid dermatitis, represents an uncommon mixed-pattern dermatitis with histopathologic features of interface dermatitis with band-like lymphocytic inflammation of the dermal-epidermal junction, with thinning of the epidermis or lichen planus-like changes, and granulomatous inflammation involving the dermis. Lichenoid granulomatous dermatitis can manifest with a variety of primary lesions and has various clinical associations. We present a challenging clinicopathologic case of lichenoid granulomatous dermatitis in a 58-year-old woman with dramatic cutaneous desquamation in the context of a complex medical history and multiple potential triggers.

Keywords

Lichenoid granulomatous dermatitis, peripheral eosinophilia

Introduction

Lichenoid granulomatous dermatitis (LGD), also known as giant cell lichenoid dermatitis, was first described by Gonzalez and colleagues in 1986.¹ LGD represents an uncommonly encountered mixed-pattern dermatitis with histopathologic features of band-like lymphocytic inflammation of the dermal-epidermal junction, with or without lichen planus-like changes, and granulomatous inflammation involving the dermis.^{1–4} Notwithstanding these mixed histologic findings, LGD is a distinct histologic entity microscopically. However, the same cannot be said about the clinical morphology of LGD, which presents with a wide range of primary lesions including erythematous-violaceous macules-plaques with varying amount of scale.³ LGD also has diverse clinical associations including but not limited to drug eruption, infectious processes, endocrinopathies, cutaneous T-cell lymphoma (CTCL), hepatobiliary disorders, and rheumatoid arthritis.^{1–5} We present a challenging clinicopathologic case of LGD in a 58-year-old woman with a purpuric, exfoliative rash and a substantially complex medical history with multiple potential causes.

Case presentation

A 58-year-old woman living in rural Saskatchewan, Canada presented with a 2-month history of a pruritic, desquamating,

purpuric, exfoliating skin rash affecting the extremities and trunk and sparing the face and mucosal membranes (Figure 1). The eruption began after the introduction of multiple antibiotic regimens for the management of septic arthritis associated with a unilateral hip arthroplasty 6 months prior. The patient initially was seen by Internal Medicine and had a complex medical history including hypothyroidism, alcoholic liver cirrhosis, long-standing seronegative rheumatoid arthritis, vitiligo, gout, peripheral eosinophilia with pulmonary infiltrates (presumed eosinophilic pneumonia), and bilateral knee arthroplasty. Family history was positive for sarcoidosis affecting the patient's sister. Investigations at the time of presentation were notable for microcytic anemia, peripheral eosinophilia, and elevated ALP, AST, GGT, IgE, IgG1, and IgG3 levels. ANA, dsDNA, ANCA panel, and Anti-Jo1 antibody were negative, along with HIV, hepatitis,

¹Department of Dermatology, Saskatchewan Health Authority, Saskatoon, SK, Canada

²Faculty of Medicine, Memorial University, St. John's, NL, Canada

³Department of Infectious Diseases, Saskatchewan Health Authority, Saskatoon, SK, Canada

⁴Department of Pathology and Laboratory Medicine, Saskatchewan Health Authority, Saskatoon, SK, Canada

Corresponding Author:

Bret Kenny, Faculty of Medicine, Memorial University, 300 Prince Philip Drive, St. John's, NL A1B 3V6, Canada.
Email: bret.kenny@mun.ca





Figure 1. Poorly demarcated, exfoliative, erythematous eruption involving the neck and upper chest (a), and arms (b). Multiple erosive papules with overlying crust were notable on the arms (c). Violaceous-to-dark brown macules and patches were scattered on the arms, neck, and chest.

and syphilis serologies. Computed tomography (CT) of the chest showed nonspecific areas of ground-glass opacity, bilaterally.

Punch biopsies in areas of scaling over the right thigh were performed and showed a mixed dermatitis (Figure 2). Histopathologically, there was an interface dermatitis with numerous Civatte and colloid bodies at the dermoepidermal junction. The epidermis was significant for lichen planus-like changes with irregular acanthosis and prominent “saw tothing” and variable compact orthohyperkeratotic scale. The papillary dermis showed perivascular and interstitial lymphohistiocytic inflammation with eosinophils, as well as rare, small, non-necrotizing granulomas. Granulomas were associated with lymphocytes and eosinophils. There was no lymphoid atypia, nor was there any evidence of an acute vasculitis after multiple deeper levels through the tissue block. The deep reticular dermis was unremarkable. Other pertinent negatives included the absence of parasites and other organisms, “flame figures,” and polarizable or refractile material. PAS/Alcian blue histochemical stains performed at the time were negative for fungal organisms and dermal mucin (not shown). GMS, ZN, and Fite histochemical stains performed at the time were negative for fungal and acid-fast bacteria respectively (not shown). A differential diagnosis based on the appearance of the skin lesions at the time of the biopsy included a vesiculobullous disorder, so a separate punch biopsy was submitted in Michel’s media for direct immunofluorescence (DIF). Cytoid bodies were positive for IgA, IgM, IgG, C3, and fibrinogen (not shown), consistent with an interface dermatitis (not shown).

A highly favored clinical diagnosis was cutaneous manifestation of eosinophilic granulomatosis with polyangiitis (EGPA) given the presence of bilateral pulmonary infiltrates on CT scan and bloodwork with peripheral eosinophilia. EGPA could not be excluded entirely given the granulomas present histologically, despite the lack of other histologic findings typically seen in EGPA. Therefore, the case was sent for external review by five expert dermatopathologists who

agreed the case was most compatible with “lichenoid and granulomatous dermatitis” due to the lack of typical triad of vasculitis, granulomata, and eosinophils seen in EGPA. Correlation with ANCA status as well as further investigation of the patient’s lung findings was recommended to definitively rule out a cutaneous manifestation of EGPA.

The patient’s antibiotic regimen was switched to amoxicillin-clavulanate and skin lesions were managed with topical corticosteroids and topical tacrolimus. The skin lesions eventually improved in line with a downward trending peripheral eosinophilia. Unfortunately, the patient has since been lost to follow-up.

Discussion

The histologic patterns of lichenoid dermatitis and granulomatous inflammation are commonly encountered as separate entities. However, it is extremely uncommon for both of these histologic patterns to coexist simultaneously in a single biopsy. The patient in this case presented with a dramatic desquamating rash in the context of several contributing and/or confounding comorbidities and medications which posed a significant diagnostic challenge requiring clinicopathologic correlation.

There has been a wide range of reported clinical morphologies of LGD including violaceous macules and papules, erythematous papules and plaques with or without scale, unilateral or dermatomal papules, annular plaques with central atrophy, and miliary-like lesions with lichenoid papules appearing to be the most common clinical presentation of LGD in the literature.³ A review of the literature revealed no reported cases of LGD presenting with a generalized purpuric, exfoliative eruption as seen in this case.

The causes of LGD are diverse and include a range of inflammatory, autoimmune, and infectious conditions, as well as several known triggering medications.^{1–11} Notably, our patient has multiple comorbidities and a complex medical history, including diseases which have been implicated with

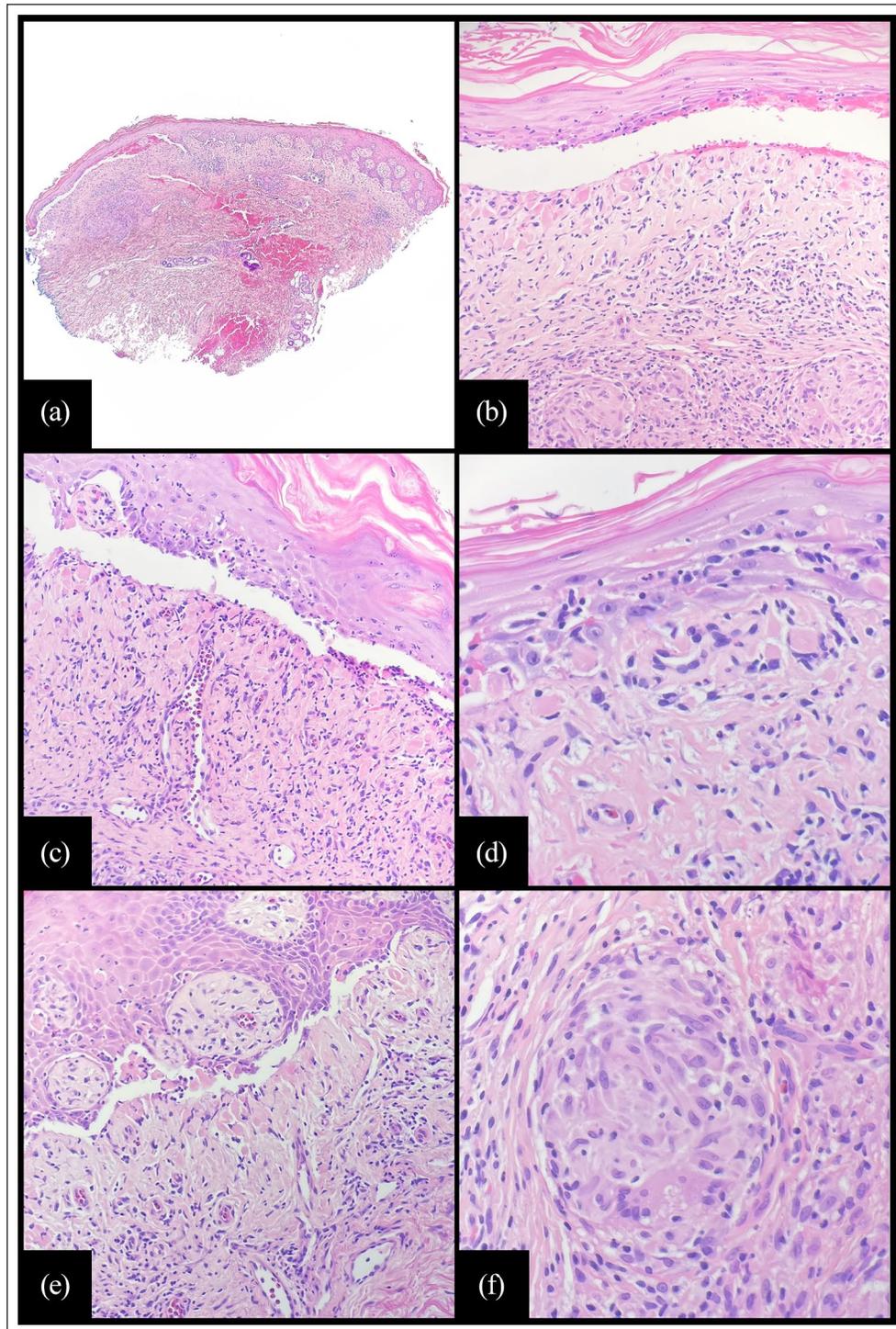


Figure 2. Photomicrograph, Hematoxylin and eosin (H&E) stains. Punch biopsy of skin notable for both thinning of the epidermis (a and b) and hyperplastic changes (c). Brisk vacuolar-type interface dermatitis was evident (c and d), with Civatte and colloid bodies readily identified (d). In other areas, classic lichen planus-like changes were appreciated with irregular epidermal acanthosis and prominent “saw tothing” of the epidermis and overlying compact orthohyperkeratotic scale. The papillary dermis was significant for perivascular lymphohistiocytic inflammation with eosinophils as well as small, non-necrotizing granulomas (e and f).

LGD, such as hepatobiliary disease, endocrinopathy, rheumatoid arthritis, and infection. Our patient initially presented with bilateral, nonspecific areas of ground-glass opacities on chest CT, which raised EGPA and sarcoidosis as potential

causes of the cutaneous eruption. However, no convincing serologic evidence for EGPA was revealed as the patient’s ANCA work-up was negative. EGPA has not been reported in association with LGD-like manifestations to date.

Our patient was also being treated with multiple potentially triggering medications including allopurinol for gout, sulfasalazine for rheumatoid arthritis, and ceftriaxone, ertapenem, and penicillin V for a septic joint. Implicated drug classes in LGD include antimicrobials, sulfur-containing drugs, nonsteroidal anti-inflammatory drugs, and various other drug classes.^{1,4,6,9} There has been no clearly established timeline between introduction of a triggering drug and the development of symptoms in the literature. Braswell et al.² correlated histologic features with common clinical causes of LGD. The study suggested that numerous or aggregated dermal eosinophils and psoriasiform epidermal changes, consistent with our case, are relatively specific features suggestive of drug eruption.¹

The clinical findings of peripheral eosinophilia and a working radiologic diagnosis of eosinophilic pneumonia on CT scan at the time of presentation confounded this case. There have been no reported associations between peripheral eosinophilia, eosinophilic pneumonia, and LGD. However, there is a strong association between peripheral blood eosinophilia and sarcoidosis as well as peripheral blood eosinophilia and rheumatoid arthritis activity.^{2,12} Complicating matters, eosinophilic pneumonia can accompany and mask sarcoidosis.^{11,13,14} Serum angiotensin-converting enzyme (ACE) level was within normal limits decreasing the likelihood of sarcoidosis however and our patient had no historical clinical manifestations or histologic findings in keeping with her medical history. Thus, on balance, the entire clinicopathologic picture favored to be most consistent with LGD manifesting as extreme desquamation due to medication/drug effect or secondary to rheumatoid arthritis.^{2,12,15}

This case represented a diagnostic challenge from clinical, radiologic, dermatologic, and histologic perspectives. The clinical morphology of LGD is generally not specific and LGD presenting as seen in our case has not been reported in the literature. In addition, our patient's multifactorial disease state introduced several confounding factors (and red herrings); yet ultimately directed the clinical team to including rheumatoid arthritis as one of the most likely potential causes, in addition to medication/drug effect. Ultimately, a combination of all of the clinical pieces helped solve this diagnostic dilemma and highlights the inherent need to weave all clinical and histologic data in assessing complex dermatology patients.

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Informed consent

The patient gave consent for use of her case for presentation in this manuscript.

ORCID iD

Bret Kenny  <https://orcid.org/0000-0003-1768-0295>

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