

**A visually striking case of centropacial destructive ulceration and desquamative gingivitis from chronic cocaine use**



*To the Editor:* We appreciated the article from Magro et al,<sup>1</sup> which described pemphigus vegetans (PVeg)

limited to the nose and lips, allowing us to introduce another differential diagnosis: cocaine-induced midline destructive lesion (CIMDL). PVeg is a rare variant of the autoimmune vesiculobullous disease pemphigus vulgaris, characterized by vegetative plaques in intertriginous areas and oral mucosa.<sup>2</sup>



**Fig 1.** Clinical progression of cocaine-induced midline destructive lesion. **A**, Centropacial ulceration 20 months before presentation. **B**, Centropacial ulceration after incisional biopsy. **C**, Friable mucosal surfaces with erosions. **D**, Nikolsky-positive gingival epithelium indicating desquamative gingivitis.

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PVeg is caused by autoantibodies against desmogleins 1 and 3 and immunoglobulin G and immunoglobulin A antibodies against desmocollins.<sup>2</sup> Nasolabial PVeg is rare, with 6 reported cases.<sup>1</sup> PVeg should be considered in the differential diagnosis with CIMDL. CIMDL is a consequence of intranasal inhalation of cocaine and can lead to inflammation, ulceration, necrosis, and perforation of nasolabial structures.<sup>3,4</sup> We present a memorable CIMDL case with desquamative gingivitis and recommend adding CIMDL to PVeg mimickers.

A 55-year-old woman with a long history of chronic cocaine abuse, last used 2 weeks ago, presented with worsening centropacial ulcerations extending from the right naris to the upper cutaneous lip for the past 8 years; an intraoral examination revealed Nikolsky-positive gingival epithelium with friable oral erosions (Fig 1). The differential diagnoses included PVeg, pemphigus vulgaris, CIMDL, vegetative herpes simplex, other atypical infections, and malignancy. Incisional biopsy revealed ulcerated tissue with lymphoplasmacytic proliferation; the lymphoplasmacytic infiltrate comprised CD138<sup>+</sup> plasma cells and polytypic  $\kappa$  and  $\lambda$  immunoglobulin light chains. Additionally, CD20 and CD3 highlighted a mixture of small B and T cells negative for AE1/3. No eosinophilic microabscess was appreciable in the ulcerated tissue. A direct immunofluorescence was not performed. More importantly, no definite carcinoma was seen on histopathology, and peripheral flow cytometry result was negative. Infectious workup results for syphilis, HIV, herpes simplex virus, varicella-zoster, nontuberculous mycobacteria, and tissue cultures were negative. Desmogleins 1 and 3 antibodies were negative. Given the clinical, histological, and negative desmogleins findings, the patient was diagnosed with CIMDL.

Prolonged use of intranasal cocaine can destroy the nasal architecture and cause CIMDL, which may mimic several entities, including nasolabial and oral PVeg.<sup>3-5</sup> Therefore, a detailed physical examination, including the oral cavity, social history, and histopathology, is required for correct diagnosis.

Herein, we describe a case of CIMDL that mimicked PVeg due to concomitant desquamative gingivitis, thereby expanding the differential of PVeg to include CIMDL. Patients with nasolabial destructive ulcerations, desquamative gingivitis, and oral erosions should be evaluated for PVeg and CIMDL. We recommend additional testing for desmogleins 1 and 3 antibodies to exclude PVeg as part of the diagnostic workup of suspected CIMDL cases.

Lindsay M. Pattison, BA,<sup>a</sup> Daniel R. Antobi, BA,<sup>a</sup> Roya Nazarian, MD,<sup>a</sup> Michael Occidental, MD,<sup>a,b</sup> Bijal Amin, MD,<sup>a,b</sup> and Benedict Wu, DO, PhD<sup>a</sup>

From the Division of Dermatology, Montefiore Medical Center, Albert Einstein College of Medicine, Bronx, New York<sup>a</sup>; and Department of Pathology, Montefiore Medical Center, Albert Einstein College of Medicine, Bronx, New York.<sup>b</sup>

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*Correspondence to:* Lindsay M. Pattison, BA, Division of Dermatology, Montefiore Medical Center, Albert Einstein College of Medicine, 1300 Morris Park Avenue, Bronx, NY 10461

*E-mail:* [lindsay.pattison@einsteinmed.edu](mailto:lindsay.pattison@einsteinmed.edu)

#### Conflicts of interest

None disclosed.

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