Nasal septal and mucosal disease associated with pyoderma gangrenosum in a cocaine user

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Key words: cocaine use/abuse; cutaneous vasculitis; levamisole; malignant pyoderma; pyoderma gangrenosum; vasculopathic skin necrosis.

INTRODUCTION

The manifestations of habitual cocaine use can mimic tumors, infections, and immunologic diseases and can affect multiple organs, causing nasal septal perforation, cutaneous vasculitis, vasculopathic skin necrosis, pyoderma gangrenosum (PG),[1] and lung disease.[2] Levamisole is a veterinary antiparasitic drug often added to cocaine in the United States to cheaply enhance or extend cocaine's euphoric effects. It is present in about 70% of seized cocaine in the United States, and it is believed to play a direct role in induction of vasculopathy, vasculitis, and PG.[1] Early distinction of these disorders can be challenging. Here we present a case of cocaine abuse disorder manifesting as cutaneous and nasal mucosal PG, perforated nasal septum, and pneumonitis.

CASE REPORT

A 53-year-old white woman with history of type 1 diabetes mellitus and recreational drug abuse presented to the dermatology department with a 6-month history of a painful, nonhealing ulcerated lesion on the back. The lesion was initially diagnosed as an abscess, and repeat incision and drainage, oral antibiotics, and supportive wound care failed (Fig 1). Tissue cultures for bacteria and fungus were negative. The patient underwent excision and repair. Histopathologic examination found ulceration with undermining of adjacent skin and acute suppurative and plasma cell–rich inflammatory infiltrates (Fig 2). The repair began to dehisce centrally, resulting in an 8.5- × 1.0-cm linear, focally ulcerating, and violaceous plaque with several areas of focal suppurative discharge (Fig 3).

Abbreviations used:

GPA: granulomatosis with polyangiitis
MP: malignant pyoderma
PG: pyoderma gangrenosum

The patient was also found to have beefy red friable and focally pustular soft tissue swelling of the nasal mucosa (Fig 4). Results of a nasal swab culture were consistent with secondary colonization. Punch biopsy found an acute suppurative folliculitis, with follicular rupture and destruction (Fig 5). Nasoendoscopy found mucosal edema, ulceration of bilateral nasal passages, and a large nasal septum perforation. The changes of skin on the back were diagnosed as PG and those of the nasal mucosa as early lesions of PG.[3]

Our patient's home medications included insulin and lisinopril. Test results for rheumatoid factor, antinuclear antibody, antistreptococcal antibody, DNase B antibody, and cryoglobulin were normal. Complete blood count, serum and urine immunofixation, and colonoscopy were normal. Nasal septal perforation workup was negative for antineutrophil cytoplasmic antibodies measured by indirect immunofluorescence and enzyme linked immunosorbent assay. Anticardiolipin, $\beta 2$ glycoprotein, and lupus anticoagulant test results were normal. Evaluations for hepatitis A, B, and C infection were negative.

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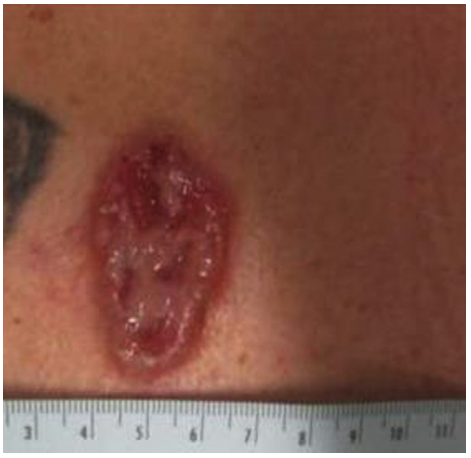

Fig 1. Original 4-cm (L) × 2.2-cm (W) ulcerated plaque, pre-excision (left upper back).

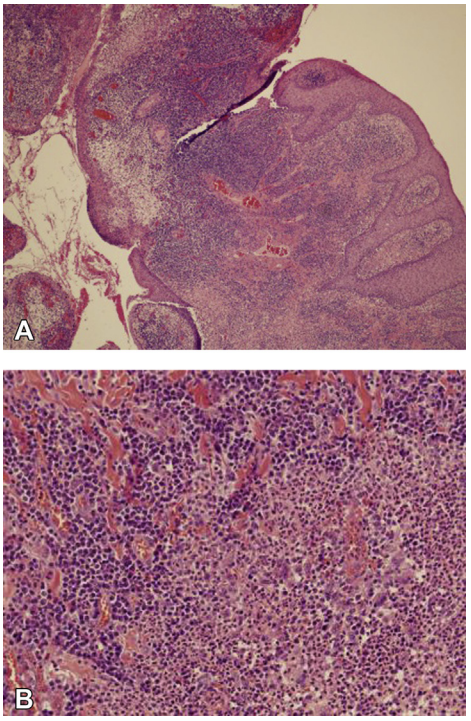

Fig 2. Skin of back. **A**, The periphery of the ulcer undermines adjacent skin and is partially re-epithelialized. **B**, The ulcer bed harbors acute suppurative and plasma cell–rich inflammatory components. The histologic features are consistent with pyoderma gangrenosum. (Hematoxylin-eosin stain.)

Chest radiography followed by computed tomography found right-sided multifocal pneumonitis and mild reactive lymphadenopathy. Bronchoalveolar lavage was negative for fungal, bacterial, *Pneumocystis jiroveci*, and mycobacterial organisms; no malignant cells were identified.

Urinalysis was negative for both protein and red blood cells, and renal function remained normal throughout follow-up. A urine toxicology screen was positive for cocaine, whereas results of serum levamisole testing performed 2 weeks later were negative.

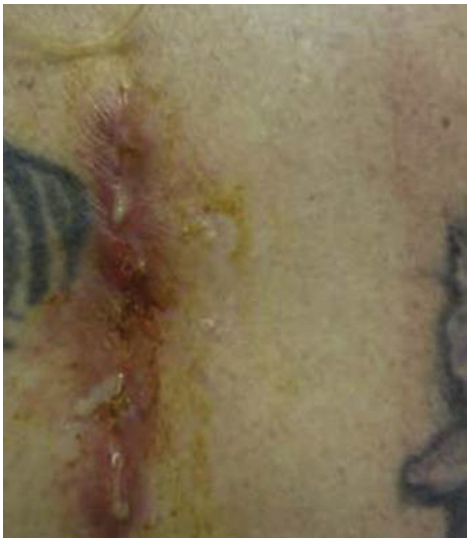

Fig 3. Ulcerating postexcision wound.

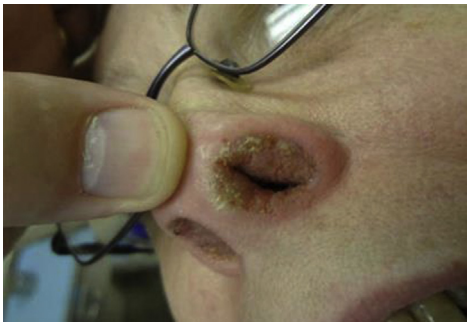

Fig 4. Pretreatment. Nasal mucosal tissue with beefy, friable, slightly erythematous and edematous tissue.

Treatment with intralesional triamcinolone injection, local wound care, oral prednisone, dapsone, and topical tacrolimus was unsuccessful (Fig 6, *A*). A repeat urine toxicology screen confirmed continued use of cocaine at this time. Treatment of refractory PG was instituted with oral cyclosporine combined with oral prednisone and topical tacrolimus and cocaine discontinuation, resulting in gradual improvement (Fig 6, *B*).The nasal mucosal overgrowth rapidly responded as well (Fig 7). She successfully discontinued cyclosporine treatment without recurrence of skin or nasal PG lesions.

DISCUSSION

Our patient had unusual sites of PG—nose and upper back—and poor response to standard treatment. Head and neck involvement in PG occurs in 5% of all PG cases.[4] Nasal involvement with PG is rarely reported and can involve the nasal septum and bridge of the nose leading to septal perforation

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