Vegetative and verrucous plaques in an immunosuppressed patient: Blastomycosis-like pyoderma

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INTRODUCTION

Blastomycosis-like pyoderma (BLP) is a rare skin condition aptly named because of its clinical and histologic resemblance to cutaneous blastomycosis. It classically presents as verrucous and vegetative plaques with pustules and raised borders in immunosuppressed patients.[1] Lesions tend to occur on sun-exposed sites, and multifocal areas of involvement are common. It has been referred to as *coral reef granuloma* due to its distinctive clinical appearance.[2] BLP is considered to be an exaggerated inflammatory reaction to bacteria, most commonly secondary to staphylococcal infection.[2] We report a case of BLP to remind the clinician of this rare and unique presentation of bacterial infection.

CASE PRESENTATION

A 64-year-old man with ulcerative colitis receiving infliximab for the past several years presented for dermatologic evaluation. One year prior, the patient had cut his left leg on a barnacle while docking his fishing boat. The wound healed, but 4 months later, he then noticed a new "bump" with serous drainage on his leg at the same site as the prior injury. On presentation, the patient had a 9 × 4-cm, friable, verrucous to vegetative crusted plaque and hyperkeratotic satellite nodule on the left leg (Fig 1). There was scant purulent drainage. On the left side of the nose and left cheek, there was an ill-defined, 4 × 8-cm hyperkeratotic to vegetative erythematous plaque with scattered pustules (Fig 2).

Abbreviation used:

BLP: blastomycosis-like pyoderma

Biopsy specimens from both the nose and leg were taken for histologic evaluation and tissue culture. Both specimens showed irregular to pseudoepitheliomatous epidermal hyperplasia with granulomatous and plasmacytic inflammation and variable reparative dermal fibrosis (Fig 3). Aerobic bacterial culture results were positive for *Pseudomonas aeruginosa* and *Morganella morganii*, both sensitive to ciprofloxacin. Tissue culture results for fungi and acid-fast bacilli were negative. Infliximab was discontinued, and the patient was treated with a 6-week course of oral ciprofloxacin 500 mg twice daily. Two months after initial presentation, all lesions had completely resolved, leaving only postinflammatory changes (Fig 4). After 6 months of follow-up, there was no evidence of recurrence.

DISCUSSION

Although the pathogenesis of BLP is not clearly defined, it is thought to be an exaggerated tissue response to bacterial infection in immunocompromised patients.[3] *Staphylococcus aureus* is the most common causative bacteria, but other

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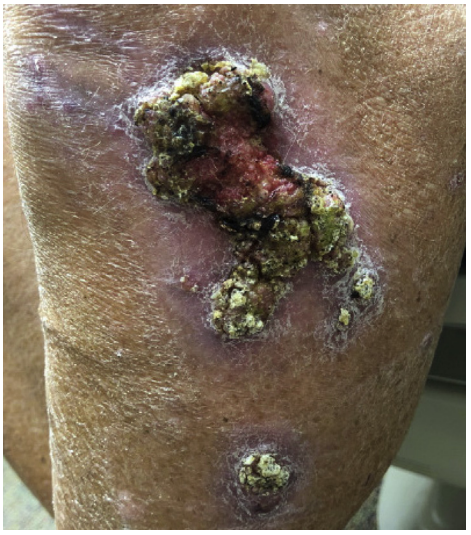

Fig 1. Blastomycosis-like pyoderma: friable, verrucous to vegetative crusted plaque on the left leg.

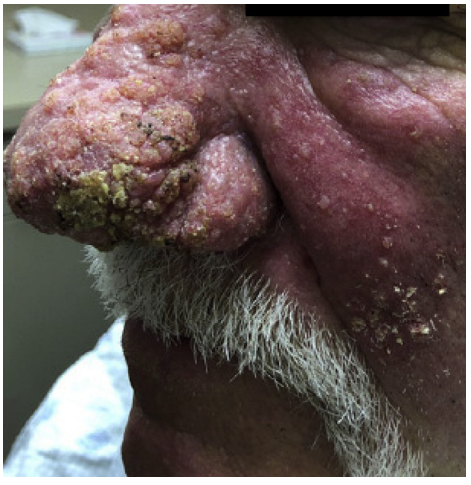

Fig 2. Large, ill-defined hyperkeratotic plaque with pustules on the nose and cheek.

Gram-positive (β-hemolytic streptococci), Gram-negative (*Pseudomonas species, Escherichia coli Proteus* species) and anaerobic (*Prevotella* species) bacteria have been isolated as well.[4] BLP has been reported in patients with HIV, hematologic malignancies, and poor nutrition and even after tattooing.[3] Our patient was immunosuppressed because of his chronic infliximab therapy, predisposing him to BLP. *P aeruginosa* and *M morganii* were isolated from our patient's lesions, and both were sensitive to ciprofloxacin. *Pseudomonas* is a recognized pathogen in BLP. Although *Morganella* species have never been implicated in BLP, *M. morganii* is an increasingly prevalent opportunistic pathogen with a broad

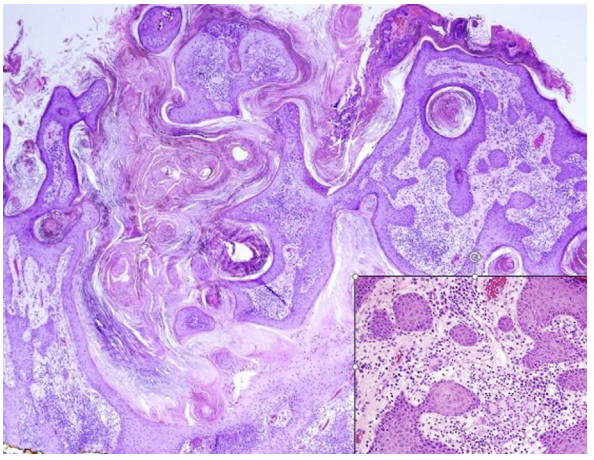

Fig 3. Skin biopsy shows serous scale crust and irregular to pseudoepitheliomatous hyperplasia (hematoxylin-eosin stain, original magnification ×100). Inset: The inflammatory infiltrate is composed of histiocytes forming small granulomas and numerous plasma cells (hematoxylin-eosin stain, original magnification ×200).

disease spectrum.[5] It seems reasonable that both bacteria played a causative role in this case.

The most common histopathologic findings of BLP include pseudoepitheliomatous hyperplasia, granulomatous inflammation, suppurative inflammation, and the formation of neutrophilic abscesses.[2] The differential diagnosis for this entity is broad and includes deep fungal infection, atypical mycobacterial infection, leishmaniasis, treponematoses, halogenoderma, pyoderma gangrenosum, pemphigus vegetans, and squamous cell carcinoma. Squamous neoplasia with massive neutrophilic inflammation has been reported, causing clinical and histologic confusion with BLP.[6] Culture of at least 1 pathogenic bacteria, characteristic histopathologic findings, and exclusion of the aforementioned diagnoses support a diagnosis of BLP.

Directed antibiotic therapy is the treatment of choice, although recurrence is fairly common.[7] Other reported treatment modalities used in conjunction with or after directed antibiotic therapy include acitretin, topical antibiotics, steroids, surgery, curettage and electrodesiccation, and laser therapy.[7]

This case nicely demonstrates a classic presentation of a rare disease. BLP should be a diagnostic consideration in immunosuppressed patients who present with verrucous and vegetative plaques.

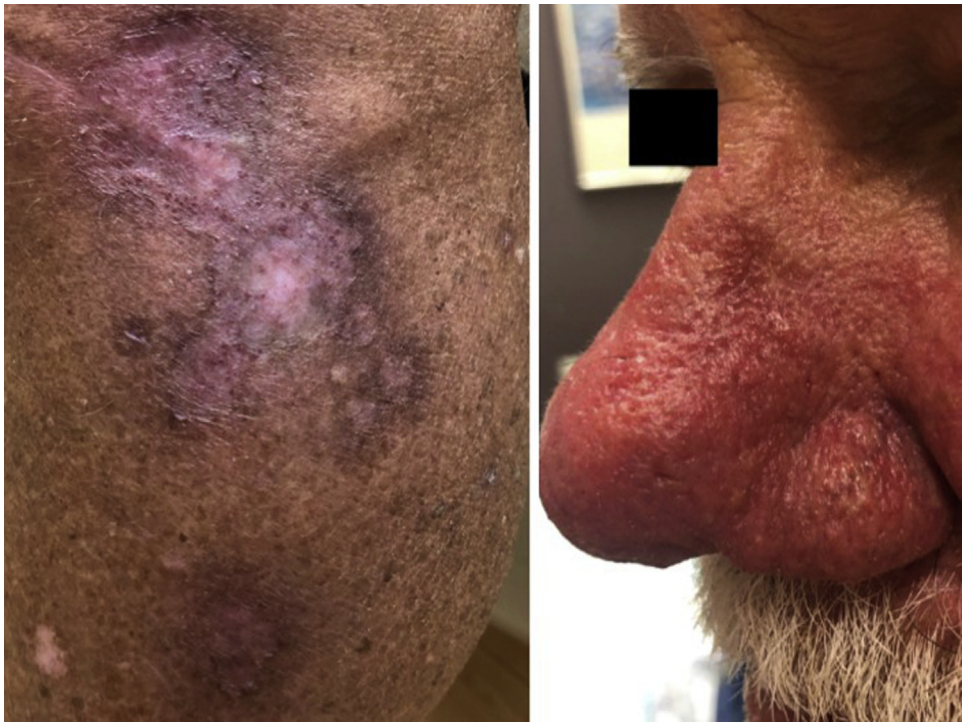

Fig 4. Complete resolution after treatment with appropriate antibiotics.

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