

Ecthyma associated with *Moraxella* and *Staphylococcus epidermidis*



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INTRODUCTION

Ecthyma is an invasive form of impetigo that penetrates deep into the dermis. This cutaneous infection is characterized by a crusted papule or plaque beneath which a deep ulcer forms. Although typically associated with group A β -hemolytic streptococci, other pathogens have the ability to produce an ecthyma.¹ Here we present a case of ecthyma from which *Moraxella* and *Staphylococcus epidermidis* were isolated. *Moraxella* has never been reported to cause ecthyma, and there is 1 reported case of ecthyma caused by *S epidermidis*.²

CASE REPORT

The patient is an 81-year-old man with hypertension and obesity who was admitted for management of acute pancreatitis. In the setting of narcotic-induced hypoventilation and obesity, the patient had respiratory failure requiring noninvasive positive pressure ventilation (NIPPV). On the seventh day of his hospital stay, he had a new 1.5-cm x 2.0-cm mildly tender necrotic ulcer on the bridge of his nose. The ulcer had an erythematous base and slightly indurated, erythematous border (Fig 1).

The patient denied headache, visual changes, epistaxis, cough, hematemesis, fevers, chills, night sweats, ocular pain, rhinorrhea, nasal congestion, dysphagia, odynophagia, and stomatodynia. He denied recent foreign travel, insect bites, and exposure to animals. He had no history of HIV or tuberculosis infection.

Several infections were included in the differential diagnosis, including bacterial infections and mycobacterial infections. Because of the rapid progression

Abbreviation used:

NIPPV: noninvasive positive pressure ventilation

of the lesion, the differential diagnosis included aggressive angioinvasive fungal infections such as zygomycosis. Vasculitic and vasculopathic causes were also considered in the setting of the patient's acute illness.

Two 3-mm punch biopsy sections were obtained from the indurated, inflamed edge of the eschar, including both affected and unaffected skin. Routine histology findings showed deep epidermal erosion, fibrin deposition, necrosis of the hair follicles and sebaceous glands with neutrophils, and evidence of bacteria within the erosion and papillary dermis. Tissue culture found heavy growth of β -lactamase-negative *Moraxella* species and heavy growth of *S epidermidis*. The patient did not have fever, bacteremia, or lymphadenopathy. Blood cultures were normal. The patient was started on a 2-week course of sulfamethoxazole-trimethoprim, 800 mg - 160 mg twice daily, and bacitracin-polymyxin B ointment topically twice daily. The ecthyma quickly resolved. He was seen in follow-up 2 months later, with a well-healed, 2-cm cicatrix without surrounding erythema (Fig 2).

DISCUSSION

To the best of our knowledge, this is the first report of ecthyma produced by *Moraxella* and *S epidermidis*. *Moraxella* is a gram-negative bacterium. The most common clinically significant species of *Moraxella* is *Moraxella catarrhalis*, which is part

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Fig 1. Necrotic eschar covering ulcer with an erythematous border on the nasal bridge.

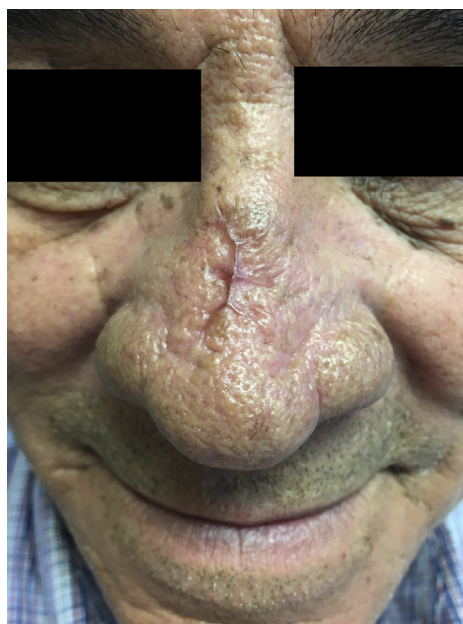


Fig 2. Nasal bridge after 2-week course of sulfamethoxazole-trimethoprim.

of the normal human flora of the upper respiratory tract. *M catarrhalis* can be transmitted via droplet spread or direct contact with contaminated secretions. It is a common cause of sinusitis and otitis media, forms a biofilm within the nasopharynx, and can cause bacteremia in patients with trans-nasal devices.³⁻⁴ Several case reports document preseptal cellulitis caused by *Moraxella* in patients who are colonized by *Moraxella* within their nasopharynx or sinus cavities.⁵⁻⁷ This organism is also capable of initiating more morbid conditions, such as necrotizing fasciitis.⁸

Bacterial skin infections by *Moraxella* typically follow a 3-step process.⁹ First, *Moraxella* adheres to the extracellular matrix of epithelial cells via integral outer membrane proteins, the ubiquitous surface protein A, and the type IV pili.¹⁰ The second step is invasion of the host epithelium by *Moraxella*. This invasion may be facilitated by a microfilament-dependent trigger uptake mechanism, as has been shown in a tissue invasion culture assay of pulmonary epithelial cells.¹¹ The multifunctional type IV pili also enhances *Moraxella's* ability to invade the host epithelium and establish a biofilm along mucous membrane surfaces.¹² This active method of invasion is in contrast to the classical model of microtubule-dependent host cell-mediated endocytosis. Finally, the third step is immune system evasion and the production of inflammatory toxins. *Moraxella* has the unique ability to inhibit the toll-like receptor 2-mediated proinflammatory response

via ubiquitous surface protein A interaction with a host cell receptor.¹⁰ Thus, *Moraxella* can effectively evade the immune system by means of T-cell response inhibition.

Similar to *Moraxella* in the respiratory tract, *S epidermidis* is a part of the normal human skin flora. Also like *Moraxella*, *S epidermidis* is capable of epithelial cell adhesion, invasion, and biofilm formation. These functions are facilitated by various exopolysaccharides and proteases.¹³ *S epidermidis* expresses the polysaccharide intercellular adhesin, which enables biofilm formation. *S epidermidis* also secretes several exotoxins, which injure host tissue and proteins. For example, the serine protease Esp degrades fibrinogen and complement factor C5, and the protease SepA degrades human antimicrobial peptides.¹³ In this way, *S epidermidis* exhibits virulent behavior.

Although uncommon, *S epidermidis* is reported to cause deep skin infections alone and in combination with other bacterial species. *S epidermidis* alone is reported to cause an ecthyma gangrenosum infection in an allogenic bone marrow transplant patient.² Concomitant infection with *S epidermidis* and *Pseudomonas aeruginosa* was documented in a unique case of ecthyma gangrenosum in a patient with chronic renal failure.¹⁴

Thus, we present a rare case of ecthyma produced by *Moraxella* and *S epidermidis*. The patient may have been colonized by *Moraxella* and *S epidermidis* in the respiratory tract and on the skin, respectively.

Just as excoriations or arthropod bites can provide a portal of entry for superinfection,¹⁵ an initial abrasion caused by the pressure and friction of the NIPPV mask likely facilitated invasion by *Moraxella* and *S. epidermis*. Moreover, the rapid formation of the ulcer, the presence of a thick hemorrhagic crust, continued progression after discontinuation of the NIPPV mask, presence of bacteria within the deep epidermal erosion, heavy growth of both organisms from the ulcer border on culture, and resolution with a thick scar support the clinical diagnosis of ecthyma.^{15,16} This case suggests the ability of *Moraxella* and *S epidermidis* to produce an ecthyma and emphasizes the importance of considering both pathogens as potential causes of deeper soft tissue infections, particularly in ill patients receiving assisted ventilation or other instrumentation or manipulation of the upper respiratory tract.

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