Cutaneous Disseminated Emmonsiosis Due to Emmonsia pasteuriana in a Patient With Cytomegalovirus Enteritis

*Emmonsia pasteuriana* is a newly emerging dimorphic fungal pathogen first isolated in 1998 from a cutaneous disseminated infection in an Italian patient with human immunodeficiency virus (HIV) infection.\(^1\) In 2013, attention was drawn to 13 cases of disseminated *E pasteuriana* infection in South Africa.\(^2\) We present herein a case of cutaneous disseminated *E pasteuriana* infection in a patient with cytomegalovirus (CMV) enteritis in China.

**Report of a Case** | A woman in her 30s presented a 3-month history of scattered asymptomatic cutaneous nodules. Medical history was significant for urticaria of 8 years’ duration and diarrhea of 14-months’ duration. She had received prednisone for 2 weeks to treat urticaria before the onset of skin nodules. At the time of presentation, she was emaciated and had no other known chronic medical conditions. She had not traveled recently but had had contact with a domestic hamster 1 month before skin lesion onset. Physical examination revealed four 1-to-2-cm diameter nodules with central ulceration or thick adherent crust on the left side of her face (Figure 1), left axilla, and bilateral forearms without lymphadenopathy. Findings of the general physical examination were normal.

Both skin lesion exudate smear and skin biopsy revealed numerous yeast cells within histiocytes; the yeast cells were rarely extracellular (Figure 2). Skin biopsy specimens yielded a thermally dimorphic fungus; morphologic characteristics and analysis by internal transcribed spacer sequence 5 DNA sequencing (GenBank accession number: syu2014.sqn syu2014 KP260922) identified it as *E pasteuriana*. Disk diffusion antifungal susceptibility testing revealed that the isolated fungus was sensitive to voriconazole, fluconazole, itraconazole, and amphotericin B. Fungal cultures of blood, urine, and stool clinical specimens were negative. Capsule endoscopy revealed ileal mucosa multiple hyperplasia and ulceration. Intestinal biopsy and immunostaining results were consistent with CMV enteritis; CD4\(^+\) and CD8\(^+\) lymphocyte counts were 570.96/mL and 287.92/mL, respectively.

Findings of chest radiography and HIV antibody and serum immunoglobulin and complement C3 and C4 evaluation were normal.

A 2-month regimen of oral voriconazole resolved the cutaneous nodules, but the diarrhea persisted. After treatment was begun with intravenous ganciclovir, the diarrhea improved significantly. There was no recurrence of skin eruption or diarrhea during the 3-month follow-up.

**Discussion** | The *Emmonsia* genus contains 3 species: *E parva*, *E crescens*, and *E pasteuriana*. The first 2 are adiaspiromycosis pathogens in rodents and rarely in humans. In infected tissues, they usually present with distinctive adiaspores.\(^3\) To our knowledge, except for 1 case each from Italy\(^1\) and Spain,\(^4,5\) *E pasteuriana* infection has been limited to HIV-infected patients in South Africa.\(^2\) In the present case, negative HIV antibody results and normal lymphocyte counts and humoral immunity were found, while HIV viral load and T-cell function were not evaluated, which is a limitation of our report. The patient’s cachexia and malnutrition, presumed to be from CMV colitis and recent prednisone treatment, likely contributed to her immunosuppression.

*Emmonsia pasteuriana* is present in infected tissue as intrahistiocytic yeasts. It can be easily mistaken for other fungal infections characterized by the presence of histiocytes with phagocytosed yeast cells, eg, histoplasmosis. The infection transmission route remains elusive. Our patient developed cutaneous nodules 1 month after close contact with her hamster, and a successful experimental animal model of *E pasteuriana* infection was constructed only when hamsters were infected via intratracheal inoculation,\(^6\) suggesting that contact with hamsters could be an *E pasteuriana* infection risk factor.

The *E pasteuriana* infection transmission route remains elusive. Our patient developed cutaneous nodules 1 month after close contact with her hamster, and a successful experimental animal model of *E pasteuriana* infection was constructed only when hamsters were infected via intratracheal inoculation,\(^6\) suggesting that contact with hamsters could be an *E pasteuriana* infection risk factor.

The skin is the most commonly involved organ in *E pasteuriana* infection.\(^2\) Our patient presented only 4 discrete cutaneous nodules, and *E pasteuriana* was only isolated from skin biopsy specimens. This implies that direct inoculation of *E pasteuriana* could have caused the cutaneous nodules.

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**Corresponding Author:** Setsuya Aiba, MD, PhD, Department of Dermatology, Tohoku University Graduate School of Medicine, Seriryomachi 1-1, Aoba-ku, Sendai, Miyagi, 980-8574, Japan (saiba@med.tohoku.ac.jp).

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**Letters**

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**Figure 1. Cutaneous Manifestation of Emmonsia pasteuriana Infection**

Facial emmonsiosis lesion. Crusted nodule at the left corner of mouth.
In conclusion, *E pasteuriana* can infect immunocompromised patients. There is potential risk that more Asian cases may emerge. Further investigation of *E pasteuriana* geographic distribution and reservoirs would reveal more information.

Xu Hua Tang, MD, PhD  
Hui Zhou, MD, PhD  
Xing Qi Zhang, MD, PhD  
Jian De Han, MD  
Qian Gao, MD

**Author Affiliations:** Department of Dermatology, The First Affiliated Hospital, Sun Yat-sen University, Guangzhou, China.

**Corresponding Author:** Qian Gao, MD, Department of Dermatology, The First Affiliated Hospital, Sun Yat-sen University, No. 58, Zhongshan 2nd Road, Guangzhou, China, 510080 (qgaol63@126.com).

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**A Case of Dominant Dystrophic Epidermolysis Bullosa Responding Well to an Old Medication**

Epidermolysis bullosa (EB) is an inherited disease characterized by fragile skin and bullae or erosion formation, either spontaneously or on minor skin trauma. Molecular defects within the epidermis or the skin’s basement membrane indicate which subtype of EB a patient has. Collagen VII is affected in dystrophic EB.1,2 The mainstay of disease management involves wound care, symptom palliation, and prevention or treatment of complications. However, there is still no consistently effective treatment that minimizes or prevents formation of bullae on minor skin trauma.

**Report of a Case** | A 58-year-old man with a history of biopsy-diagnosed dominant dystrophic EB since childhood presented to the dermatology clinic with a 6- to 8-month history of increasing spontaneous bullae formation. Clinical manifestations were primarily cutaneous, acral in distribution, and affecting mainly his hands, legs, feet, and nails. His eyes, oral cavity, gastrointestinal tract, and genitourinary tract were not involved. Family history revealed that his father and older brother were also affected, and there was no history of consanguinity. Patient was treated with oral minocycline, 100 mg, twice daily.

At a 6-week follow-up appointment, the erosions were healing, and he had not had any new bullae formation. At a subsequent 6-week follow-up appointment, the patient reported...