Crusted Demodicosis in an Immunocompetent Patient

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PRACTICE **POINTS**

- The *Demodex* mite, believed to be a commensal species in humans, has the ability to shift to a pathologic form in immunocompromised patients.
- Demodicosis can manifest in a variety of forms including pityriasis folliculorum, rosacealike demodicosis, and demodicosis gravis.

To the Editor:

Demodicosis is an infection of humans caused by species of the genus of saprophytic mites *Demodex* (most commonly *Demodex brevis* and *Demodex folliculorum*) that feed on the pilosebaceous unit.[1] *Demodex* mites are believed to be a commensal species in humans; an increase in mite concentration or mite penetration of the dermis, however, can cause a shift from a commensal to a pathologic form.[2] Demodicosis manifests in a variety of forms, including pityriasis folliculorum, rosacealike demodicosis, and demodicosis gravis. The likelihood of colonization increases with age; the mite rarely is observed in children but is found at a rate approaching 100% in the elderly population.[3] It is hypothesized that manifestation of disease might be due to a decrease in immune function or an inherited HLA antigen that causes local immunosuppression.[4]

A 51-year-old man who was otherwise healthy presented to our clinic with a crusting rash on the face of 9 weeks' duration. The rash began a few days after he demolished a rotting wooden shed in his backyard. Lesions began as pustules on the left cheek, which then developed notable crusting over the next 5 to 7 days and spread to involve the forehead, nose, and right cheek (Figure 1A).

The patient had no underlying immunosuppressive disease; a human immunodeficiency virus screen, complete blood cell count, and tests of hepatic function were all unremarkable. He denied a history of frequent or recurrent sinopulmonary infections, skin infections, or infectious diarrheal illnesses. He had been seen by his primary care physician who had treated him for herpes zoster without improvement.

At our initial evaluation, biopsy was performed; specimens were sent for histopathologic analysis and culture. Findings included a dermal neutrophilic inflammation, a dense perivascular and perifollicular lymphoplasmacytic infiltrate with foci of neutrophilic pustules within the follicles (Figure 2), numerous intrafollicular *Demodex* mites (Figure 3), perifollicular vague noncaseating granuloma, and mild sebaceous hyperplasia. Grocott methenamine-silver stain and acid-fast bacilli stain were negative.

Review of clinical and pathological data yielded a final diagnosis of crusted demodicosis with a background of rosacea. The patient was ultimately treated with a single dose of oral ivermectin 15 mg with a second dose 7 days later in addition to daily application of ivermectin cream 1% to affected areas of his rash. He had notable improvement with this regimen, with complete resolution within 6 weeks (Figure 1B). The patient noted mild recurrence 14 to 21 days after discontinuing topical ivermectin.

The 2 species of *Demodex* that cause disease in humans each behave distinctively: *D folliculorum*, with a cigar-shaped body, favors superficial hair follicles; *D brevis*, a smaller form, burrows deeper into skin where it feeds on the pilosebaceous unit.[1] Colonization occurs

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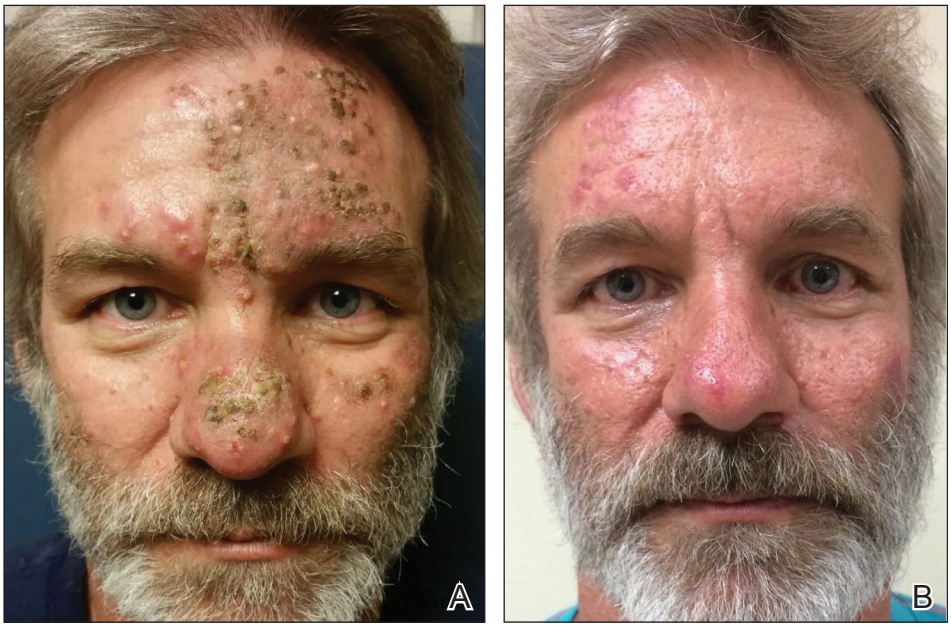

FIGURE 1. Crusted demodicosis. A, Pink to erythematous papules and pustules with crusting on the forehead, nose, and cheeks bilaterally, with greater involvement of the left side. B, Resolution of crusted papules and pustules after 6 weeks of therapy with oral ivermectin and ivermectin cream 1%. There was mild recurrence of pink papules on the forehead, as the patient had been without topical treatment.

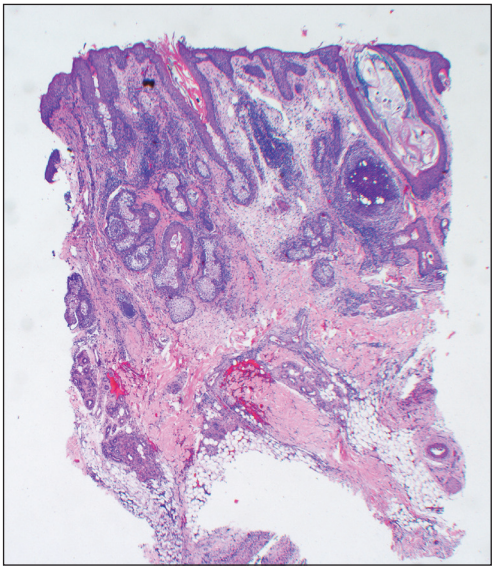

FIGURE 2. A dense dermal perivascular and perifollicular lymphoplasmacytic infiltrate with foci of neutrophilic pustules within the follicles (H&E, original magnification ×20).

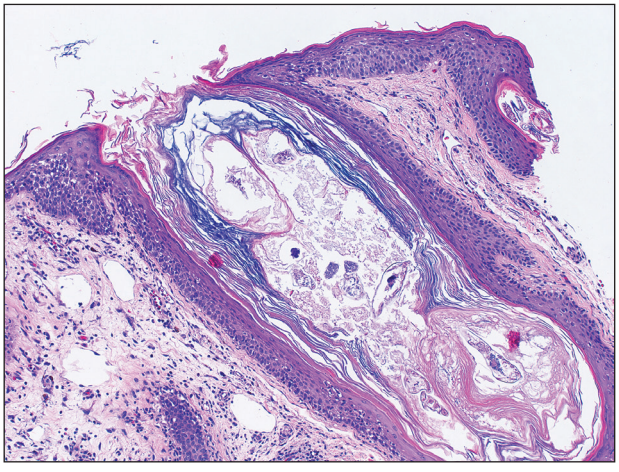

FIGURE 3. Numerous intrafollicular *Demodex* mites (H&E, original magnification ×100).

through direct skin-skin contact that begins as early as infancy and becomes more common with age due to development of sebaceous glands, the main source of nourishment for the mites.[2]

Demodicosis is classified as primary and secondary. In a prospective study of patients with clinical findings of demodicosis, Akilov et al[1] discovered that the 2 forms can be differentiated by skin distribution, seasonality, mite species, and preexisting dermatoses. Primary demodicosis is categorized by sudden onset of symptoms on healthy skin, usually the face. Secondary demodicosis develops progressively in patients with preexisting skin disease, such as rosacea, and can have a broader distribution, involving the face and trunk.[2] Clinical manifestations of demodicosis are broad and include pruritic papulopustular, nodulocystic, crusted, and abscesslike lesions.[5]

Most cases of demodicosis reported in the literature are associated with either local or systemic immunosuppression.[6-8] In a case report, an otherwise immunocompetent child developed facial demodicosis after local immunosuppression from chronic use of 2 topical steroid agents.[9]

Demodex infestation can be diagnosed using a variety of methods, including standardized skin surface biopsy, punch biopsy, and potassium hydroxide analysis. Standardized skin surface biopsy is the preferred method to diagnose demodicosis because it is noninvasive and samples the superficial follicle where *Demodex* mites typically reside. Diagnosis is made by identifying 5 or more *Demodex* mites in a low-power field or more than 5 mites per square centimeter in standardized skin surface biopsy.[2] Other potential diagnostic tools reported in the literature include dermoscopy and confocal laser scanning microscopy.[10,11]

There is no standard therapeutic regimen for demodicosis because evidence-based trials regarding the efficacy of treatments are lacking. Oral ivermectin 200 μg/kg in a single dose is considered the preferred treatment; it can be combined with oral erythromycin, topical permethrin, or topical metronidazole.[5-7,9]

Our case is unique, as crusted demodicosis developed in an immunocompetent adult. Demodicosis usually causes severe eruptions in immunocompromised persons, with only 1 case report detailing a papulopustular rash in an immunocompetent adult.[12,13]

The pathogenesis of demodicosis remains unclear. Many mechanisms have been hypothesized to play a role in its pathogenesis, including mechanical obstruction of hair follicles, hypersensitivity reaction to *Demodex* mites, immune dysregulation, and a foreign-body granulomatous reaction to the skeleton of the mite.[2,3] Our patient's particular infestation could have been caused by an exuberant reaction to *Demodex*; however, it is likely that many factors played a role in his disease process to cause an increase in mite density and subsequent manifestations of disease.

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