Multiple Papules and Pustules on the Face and Neck

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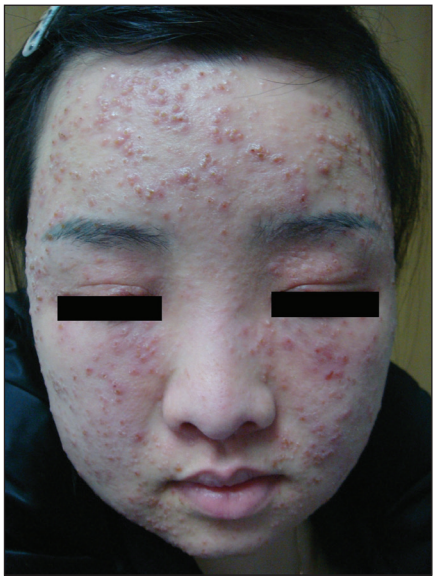

A 26-year-old woman presented to our clinic with multiple papules and pustules on the face and neck. One year prior, the patient had developed a pruritic rash on the face after using a new over-the-counter skin care product. An outside physician had diagnosed the rash as contact dermatitis and prescribed tacrolimus cream 0.1%. Initially, the patient noted improvement, but the rash recurred intermittently over the next year. She continued using the cream, but 2 months prior to the current presentation, the patient developed more papules and pustules on the face, prompting further evaluation.

Physical examination at the current presentation revealed widespread papules and pustules on the face and neck. Due to the patient's aesthetic concerns, a more invasive biopsy was avoided, and purulent fluid from the lesions was collected for microscopic examination.

WHAT IS YOUR **DIAGNOSIS?**

a. acne vulgaris
b. bacterial folliculitis
c. demodicosis
d. rosacea
e. tinea corporis

PLEASE TURN TO PAGE E8 FOR THE DIAGNOSIS

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THE **DIAGNOSIS:** Demodicosis

Direct microscopic examination of the purulent fluid revealed a considerable number of actively motile *Demodex* mites (Figure). Based on the microscopy results and the patient's history of prolonged topical immunosuppressive therapy, a known risk factor for *Demodex* overgrowth, a diagnosis of demodicosis was made. The patient was prescribed a single dose of oral metronidazole 2 g as well as metronidazole solution 0.5% to be applied 3 times daily. The folliculitis gradually improved and eventually resolved completely.

Demodex is a parasitic mite inhabiting the pilosebaceous units of human skin. Evidence suggests the vast majority of adults carry these mites. *Demodex* mites maintain a balance with the human immune system in appropriate microenvironments, with the immune system controlling their numbers without eliciting an inflammatory response; however, immunosuppression, as induced by topical corticosteroids and other immunomodulators, can lead to an increase in *Demodex* mite populations on facial skin. Clinical manifestations and severity of demodicosis are highly variable, ranging from nonspecific dry, sensitive skin and papules to nodules or granulomas, depending on mite density, the cutaneous microenvironment, and the host immune response.[1] Consequently, demodicosis often is mistaken for other dermatologic conditions with similar skin lesions.

High *Demodex* mite density is considered a pathogenic factor in demodicosis; therefore, determining *Demodex* mite density is essential to the diagnosis of demodicosis. Standard skin surface biopsy and direct microscopic examination commonly are used methods for measuring *Demodex* mite density; however, the accuracy of these methods is subject to the technical proficiency of the investigator. Noninvasive examination tools like dermoscopy and confocal laser scanning also offer advantages in diagnosing demodicosis. Dermoscopy, by direct contact with skin lesions, typically reveals gelatinous filaments extending from the follicular openings.

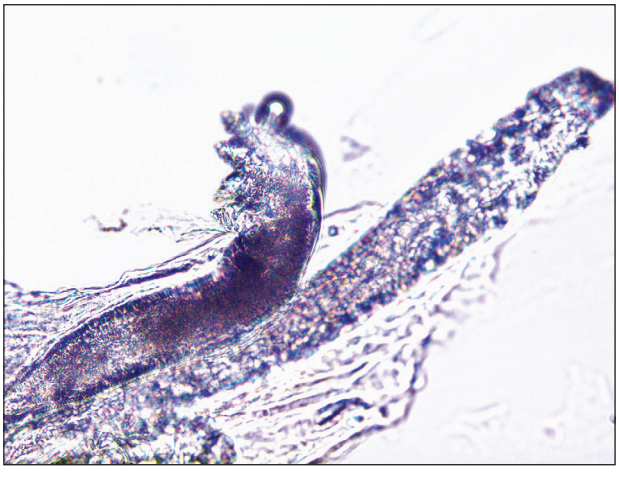

FIGURE. Live *Demodex* mites obtained from purulent fluid prepared with mineral oil and viewed under direct microscopy (original magnification ×100).

Importantly, *Demodex* mite density alone does not determine the severity of clinical symptoms. In addition, mites may migrate to the skin surface or reside deep within follicles, rendering them difficult to detect with standard examination methods.[1] Therefore, diagnostic criteria should extend beyond mite proliferation to include characteristic clinical lesions, response to acaricidal therapy, and normalization of mite density.

Rosacea was included in the differential diagnosis for our patient, but it typically manifests in the central facial area (eg, forehead, nose, chin). Patients may have a history of facial flushing associated with alcohol consumption, heat exposure, or emotional stress.[2] Additionally, rosacea typically has an insidious onset and does not erupt suddenly within a short period of time; however, our patient presentd with a sudden onset of widespread papules and pustules on the face without facial flushing, and there was no exacerbation upon exposure to heat or emotional stress. Furthermore, rosacea tends to be recurrent and challenging to cure, whereas our patient responded rapidly to treatment without recurrence. Therefore, the likelihood of rosacea was minimal. Histopathologic examination also can differentiate between rosacea and demodicosis. Histologically, the features of rosacea include dilated blood and lymphatic vessels and infiltration of T lymphocytes, macrophages, and mast cells around blood vessels, often with increased solar elastosis and dermal edema.[3] Demodicosis can reveal *Demodex* mites within the infundibulum of hair follicles, with dense neutrophil and monocyte infiltration around and between the infundibula.[4]

Bacterial folliculitis is primarily characterized by perifollicular erythema, papules, and pustules, often accompanied by pain. Positive bacterial culture of purulent fluid is indicative.[5] Our patient's lesions shared certain similarities with bacterial folliculitis but lacked the characteristic pain, instead exhibiting pronounced pruritus. Remarkable therapeutic efficacy was observed following topical acaricidal treatment, thus rendering the diagnosis of bacterial folliculitis less probable.

Acne vulgaris is a noninfectious folliculitis caused by follicular occlusion. Abnormal keratinization leads to the obstruction of follicles by keratin, hindering the outflow of sebum from the follicles. Sebum

accumulation within the follicles provides a rich substrate for *Propionibacterium acnes*, which metabolizes sebum into proinflammatory free fatty acids, resulting in the formation of comedones, papules, and pustules.[5] Our patient did not exhibit comedonal lesions on the face and lacked a seborrheic complexion, hence diminishing the likelihood of acne vulgaris.

Tinea corporis is another intensely pruritic condition, especially in areas subjected to prolonged use of topical immunosuppressants. It is caused by dermatophyte fungi and typically manifests as erythematous pruritic patches, often presenting as ring-shaped lesions with active margins and sometimes accompanied by scaling.[6] While long-term use of immunosuppressants may be a risk factor for fungal infections and increase the probability of tinea corporis, our patient's presentation of papules and pustules without a ring-shaped configuration or scaling diminished the likelihood of tinea corporis.

Our patient represents an intriguing case of an eruptive form of demodicosis induced by long-term intermittent and inconsistent application of topical immunosuppressive agents. Demodicosis encompasses a spectrum of clinical presentations, including pityriasis folliculorum, rosacealike, folliculitislike, and perioral dermatitis–like forms.[1] It is prone to misdiagnosis, as it is clinically similar to other conditions, such as acne, rosacea, or bacterial folliculitis, and it also is susceptible to missed diagnosis. Demodicosis tends to erupt in immunocompromised individuals, and the use of topical immunosuppressive and corticosteroid medications can exacerbate *Demodex* activity. Dermatologists should be aware that demodicosis is not a rare skin disorder, and timely identification and diagnosis can reduce the incidence of disease and improve quality of life for affected patients. Conversely, the consequences of misdiagnosis can be severe, with inappropriate treatment potentially exacerbating the condition.

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