Papular Acneform Eruption With Mucositis

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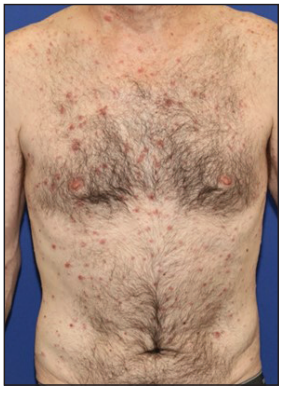

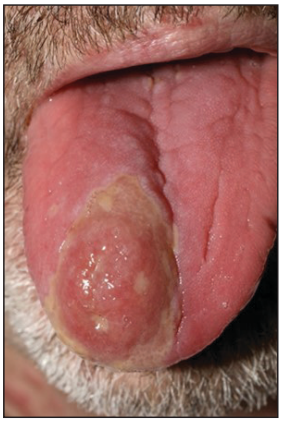

A 48-year-old man with a history of ulcerative colitis that was well-controlled with adalimumab presented with a generalized acneiform eruption involving the face, chest (top) and back, as well as a well-defined ovoid ulcer on the anterior aspect of the tongue (bottom) of 2 months' duration. Prior treatment with prednisone 60 mg daily for 14 days resulted in no improvement. He denied unintentional weight loss, cyclic fever, or arthritis. A complete blood cell count with differential showed mild anemia (hemoglobin, 11.6 g/dL [reference range, 13.2–16.6 g/dL]) with a differential cell count that was within reference range for each cell type. The erythrocyte sedimentation rate was elevated at 44 mm/h (reference range, 0–22 mm/h). A 4-mm punch biopsy specimen of an indurated cystic papule on the torso was obtained.

WHAT'S YOUR **DIAGNOSIS?**

a. Behçet disease
b. pustular psoriasis
c. SAPHO (synovitis, acne, pustulosis, hyperostosis, osteitis) syndrome
d. steroid-induced acne
e. syphilis

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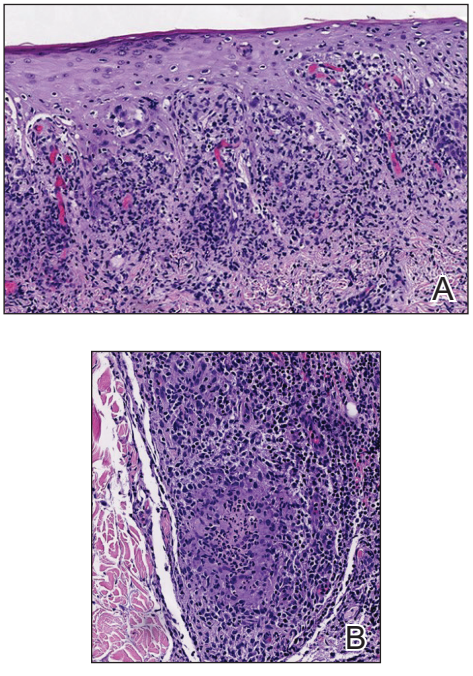
THE **DIAGNOSIS:** Syphilis

Histopathology revealed psoriasiform hyperplasia, endothelial cell swelling, and a brisk lichenoid inflammation with plasma cells (Figure, A). There also was pustular folliculitis in association with well-formed granulomatous inflammation and a prominent number of plasma cells (Figure, B). *Treponema pallidum* immunostaining showed numerous organisms in the epidermal and follicular epithelium. Rapid plasma reagin was found to be positive with a titer of 1:128. Evaluation for neurosyphilis through lumbar puncture was negative; the patient also was HIV negative. All of our patient's skin lesions cleared after a 3-week course of weekly intramuscular benzathine G injections. Due to his substantial clinical improvement, the patient was subsequently lost to follow-up.

Syphilis, an infectious disease caused by the spirochete bacterium *T pallidum*, has a well-known natural history defined by various stages classically categorized as primary, secondary, latent, or late (tertiary).[1] The classic lesion in primary syphilis is the chancre, a painless ulcer with raised borders that develops within approximately 3 weeks following the initial inoculation.[2] Secondary syphilis manifests with mucocutaneous findings in up to 97% of patients, and untreated patients develop secondary syphilis at a rate of approximately 25%.[3] Although mucocutaneous findings in secondary syphilis can vary widely, patients most commonly develop a diffuse maculopapular exanthem, and 40% develop mucosal findings including genital ulcers, mucous patches, and condylomata lata.[1] In latent syphilis, there is seroreactivity, but otherwise there are no clinical symptoms. A clear symptomatic history of prior primary or secondary syphilis may be known or unknown. Latent syphilis is divided into early and late phases, and the World Health Organization designates 2 years after the first suspected exposure as the cutoff point for early and late latency.[4] During the first 4 years of latent syphilis, patients may exhibit mucocutaneous relapses. Our patient denied any sexual activity for more than 3 years prior to presentation. Because of the start of iatrogenic immunosuppression during this period, this case was classified as late latent syphilis with mucocutaneous reactivation.



Histopathology of an indurated cystic papule on the torso. A, Psoriasiform hyperplasia, lichenoid inflammation with plasma cells, and endothelial cell swelling were present (H&E, original magnification ×12). B, Pustular folliculitis and granulomatous inflammation with plasma cells also were noted (H&E, original magnification ×12).

Behçet disease was included within the differential diagnosis but is characterized by multiorgan systemic vasculitis that causes various mucocutaneous findings including aphthous ulcers, papulopustular lesions, and genital ulcers.[5] Histopathologic features are nonspecific, and the clinical finding of recurrent genital and oral ulceration should be present for diagnosis. This disease predominantly occurs in East Asian or Mediterranean populations and is otherwise rare in White individuals.

SAPHO (synovitis, acne, pustulosis, hyperostosis, osteitis) syndrome is a rare disorder consisting of skin, joint, and bone manifestations.[6] Severe acne generally is accompanied by palmoplantar pustulosis along with pain and joint tenderness involving the anterior chest and axial skeleton, both of which were absent in our patient.

Pustular psoriasis can be localized or generalized. Localized presentations frequently are acral and may be associated with a variable degree of nail dystrophy and arthritis. Generalized presentations are characterized by hyperemic, well-defined patches with variable numbers of pustules.[7] The pustules are the consequence of exuberate neutrophilic exocytosis into the epidermis and are nonfollicular.

Steroid-induced acne may be considered in the proper clinical setting of an acneform eruption with a prior history of systemic steroid treatment. However, additional findings of mucositis would not be expected, and although our patient was prescribed prednisone from his primary care physician prior to presentation to our clinic, this medication was given after the onset of the cutaneous eruption.

Syphilis commonly is referred to as the great mimicker due to its potential diverse morphologic presentations, which can involve acneform eruptions, though rare.[8] In

the setting of mucositis, generalized acneform eruptions should raise suspicion for the possibility of syphilis, even in the absence of other more classic cutaneous features.

REFERENCES

1. Forrestel AK, Kovarik CL, Katz KA. Sexually acquired syphilis: historical aspects, microbiology, epidemiology, and clinical manifestations. *J Am Acad Dermatol*. 2020;82:1-14.
2. Sparling PF. Natural history of syphilis. In: Holmes KK, Mardh PA, Sparling PF, et al, eds. *Sexually Transmitted Diseases*. McGraw Hill; 1990:213.
3. Clark EG, Danbolt N. The Oslo study of the natural course of untreated syphilis: an epidemiologic investigation based on a re-study of the Boeck-Bruusgaard material. *Med Clin North Am*. 1964;48:613.
4. Sule RR, Deshpande SG, Dharmadhikari NJ, et al. Late cutaneous syphilis. *Cutis*. 1997;59:135-137.
5. Wilder EG, Frieder J, Sulhan S, et al. Spectrum of orocutaneous disease associations: genodermatoses and inflammatory conditions. *J Am Acad Dermatol*. 2017;77:809-830.
6. Carneiro S, Sampaio-Barros PD. SAPHO syndrome. *Rheum Dis Clin North Am*. 2013;39:401-418.
7. Bachelez H. Pustular psoriasis and related pustular skin diseases. *Br J Dermatol*. 2018;178:614-618.
8. Domantay-Apostol GP, Handog EB, Gabriel MT. Syphilis: the international challenge of the great imitator. *Dermatol Clin*. 2008; 26:191-202, v. doi:10.1016/j.det.2007.12.001