

Case Letter

Atypical Presentation of Secondary Syphilis

To the Editor:

Syphilis is caused by *Treponema pallidum* and clinically presents with variable mucocutaneous features. The clinical features of secondary syphilis can be macular, maculopapular, follicular, papulosquamous, or pustular, as well as erythema multiforme (EM)-like targetoid lesions.¹⁻³ We describe a patient with secondary syphilis presenting as a diffuse, large, annular patch.

A 43-year-old man presented with a nonpruritic skin eruption on the left side of his chest and on both palms of 2 weeks' duration. He previously had undergone liver and kidney transplantations 5 years prior due to liver cirrhosis and end-stage renal disease. Since then, he had been taking prednisolone and mycophenolate mofetil. Physical examination revealed a diffuse, large, annular-shaped, erythematous patch on the left side of his chest and multiple erythematous macules on both palms (Figure 1). No other abnormalities were noted on physical examination. The patient admitted having sexual contact with a prostitute approximately 2 months prior to the onset of the lesion. In the serology tests, the VDRL test was reactive with a titer of 1:128, the *T pallidum* hemagglutination assay was reactive, and the fluorescent treponemal antibody absorption test was reactive (2+). A skin biopsy was performed on the chest and right palm with 4-mm and 3-mm punches, respectively. The biopsy of the right palm revealed psoriasiform epidermal hyperplasia with a basal vacuolar alteration. Lymphohistiocytic interface dermatitis with exocytosis and perivascular periadnexal lymphohistiocytic infiltration as well as a few plasma cell infiltrations also were evident. The specimen from the annular erythematous patch showed epidermal basal vacuolar change and a few necrotic keratinocytes without epidermal hyperplasia. Lymphohistiocytic interface dermatitis with exocytosis and superficial perivascular lymphohistiocytic

infiltration in the dermis also were observed (Figure 2A). Immunohistochemical analysis with a polyclonal antibody, anti-*T pallidum* antibody, revealed many spirochetes in both the palm and chest specimens (Figure 2B). The palm specimen was consistent with syphilis; however, the chest biopsy was consistent with EM. The patient was treated with penicillin G benzathine (2,400,000 U) by intramuscular injection once weekly for 3 consecutive weeks. Three weeks after initiation of therapy, the skin lesions resolved leaving postinflammatory hyperpigmentation. After 6 months there were no skin lesions and the VDRL test titer decreased to 1:4.

Secondary syphilis is known to present with a wide range of clinically diverse cutaneous lesions that can



Figure 1. Large, annular, erythematous patch on the left side of the chest (A). Multiple erythematous macules on both palms (B).

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The authors report no conflict of interest.

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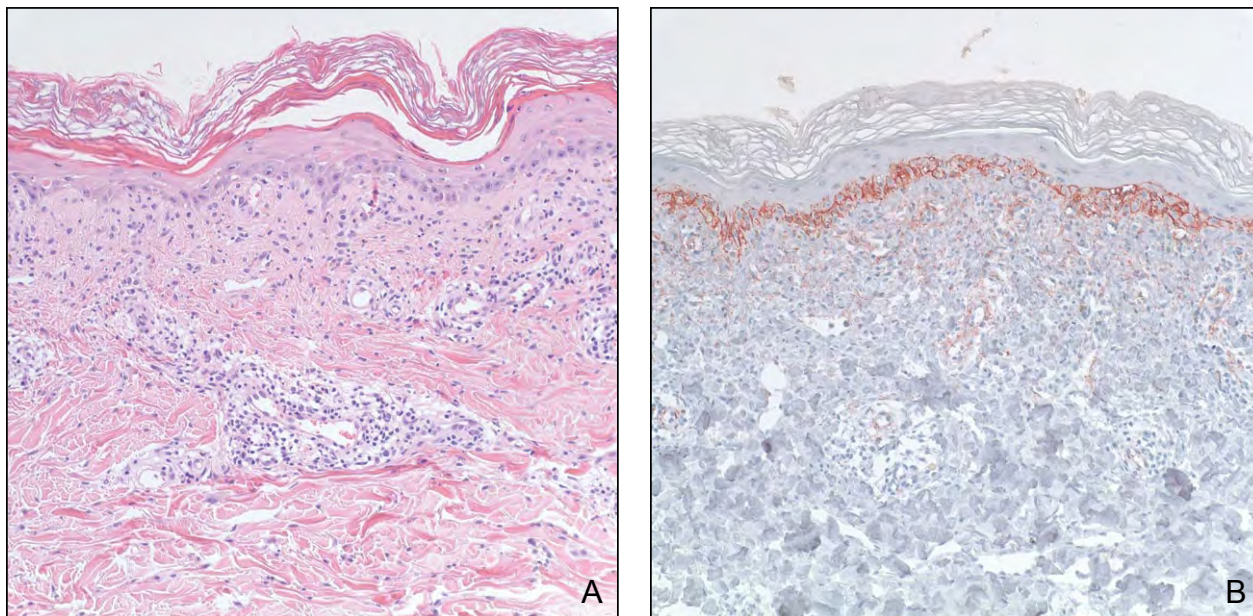


Figure 2. A skin biopsy from the left side of the chest revealed epidermal basal vacuolar change without epidermal hyperplasia; a few necrotic keratinocytes were observed. Lymphohistiocytic interface dermatitis with exocytosis and superficial perivascular lymphohistiocytic infiltration in the dermis also were seen (A)(H&E, original magnification $\times 200$). Immunohistochemical stain for the anti-*Treponema pallidum* antibody revealed spirochetes in the specimen from the chest (B)(original magnification $\times 200$).

mimic a number of dermatologic diseases.⁴ Typical features of secondary syphilis are generalized papulo-squamous eruptions, but annular eruptions, as in our case, also may be present.

We diagnosed the patient with secondary syphilis because of a high titer in the serologic tests for syphilis and the presence of *T pallidum* in the tissue, revealed by immunohistochemical stains. Erythema multiforme is a self-limiting inflammatory disease, which has been considered to be a hypersensitivity reaction to various etiologic factors.⁵ Syphilis is known to be a precipitating factor of EM, and there are a few reports of patients exhibiting targetoid patches from secondary syphilis. In these reports, the histopathologic features of EM-like targetoid patches resulting from syphilis were similar to those not from syphilis.² The histopathologic features of our case were consistent with EM; however, clinically he presented with a large annular erythematous patch, which is not a typical feature of EM. Abell et al⁶ reported that secondary syphilis can show considerable variation in histologic pattern and that clinical morphology of the eruptions does not necessarily correlate with histologic pattern, which could explain the discord between the clinical and histologic features evident in our case.

Syphilis is considered the great imitator and can rarely present as a large, annular, erythematous patch.

Furthermore, the histopathology of the annular patch in our case showed the features of EM. In conclusion, clinicians should consider the possibility of secondary syphilis for lesions that present with a large, annular, erythematous patch.

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