

Psoriasis Guttata With Palmoplantar Involvement Clinically Mimicking Secondary Syphilis

Whitney A. High, MD; Mai P. Hoang, MD; Paul R. Bergstresser, MD

Papulosquamous eruptions involving the palms and soles are thought to be particularly suggestive of secondary syphilis. Alternative diagnoses exist, however, and include psoriasis guttata, atypical pityriasis rosea, and pityriasis lichenoides chronica (PLC). We describe the case of a patient with an abrupt onset of psoriasis guttata and extensive palmoplantar involvement. Results of serologic testing were negative for treponemical antibodies. Results of histopathologic examination demonstrated psoriasiform dermatitis with neutrophils in the epidermis; plasma cells were absent. Spirochetes were not demonstrated in a tissue sample using silver or immunohistochemical stains for Treponema pallidum. A broad differential diagnosis is required when evaluating papulosquamous eruptions with palmoplantar involvement. Although not well referenced in the medical literature, psoriasis guttata can indeed cause palmoplantar lesions that mimic those of secondary syphilis.

Cutis. 2005;76:358-360.

Papulosquamous eruptions involving the palms and soles are thought to be highly suggestive of secondary syphilis.¹ Nevertheless, alternative diagnoses include psoriasis guttata, atypical pityriasis rosea, and pityriasis lichenoides chronica (PLC), all of which occasionally exhibit palmoplantar involvement.²⁻⁴ We describe the case of a patient with an abrupt onset of psoriasis guttata. Palmoplantar

involvement and a remissive course strongly suggested secondary syphilis. Results of serologic testing, however, demonstrated no evidence of treponemical antibodies. Results of histopathologic examination included psoriasiform dermatitis with neutrophils in the epidermis. Plasma cells were conspicuously absent, and the results of silver and immunohistochemical staining for spirochetes did not demonstrate organisms in the tissue.

Case Report

A 27-year-old black woman presented to the dermatology clinic of Parkland Memorial Hospital in Dallas, Texas, with a rash of 4 weeks' duration. The diffuse eruption had had an abrupt onset. The patient denied prodromal symptoms such as fever, arthralgias, sore throat, or upper respiratory illness. Although distressing in appearance, the rash was asymptomatic. There was no personal or family history of skin disease. The patient had been in a monogamous sexual relationship for 2 years.

On physical examination, the patient exhibited an extensive papulosquamous eruption on the trunk and extremities that consisted of erythematous scaling papules and small plaques (Figure 1). Palmoplantar involvement also was noted (Figure 2). Her elbows, knees, scalp, and gluteal cleft were not extensively involved. Her fingernails showed no pitting, discoloration, or onycholysis. The oral mucosa appeared normal. Clinical suspicion of secondary syphilis was high, but additional studies were needed to confirm a diagnosis.

Despite serial dilution to prevent a confounding prozone phenomenon, results of a rapid plasmin reagin/VDRL test did not demonstrate nonspecific antibodies to *Treponema pallidum*, and the findings of the microhemagglutination-*T pallidum* test did not reveal specific antibodies to the organism. Test results were negative for human immunodeficiency virus. Results of a 6-mm punch biopsy from the left upper thigh revealed psoriasiform dermatitis with a sparse perivascular lymphocytic infiltrate; plasma

Accepted for publication September 9, 2004.

Dr. High is from the Department of Dermatology, University of Colorado Health Sciences Center, Denver. Dr. Hoang is from the Department of Pathology, and Dr. Bergstresser is from the Department of Dermatology, University of Texas Southwestern Medical Center at Dallas.

The authors report no conflict of interest.

Reprints: Whitney A. High, MD, Department of Dermatology, University of Colorado Health Sciences Center, PO Box 6510, Mail Stop F.703, Denver, CO 80045-0510 (e-mail: wahigh50@hotmail.com).



Figure 1. Diffuse papulosquamous eruption on the proximal extremities of a 27-year-old black woman one month after abrupt onset.



Figure 2. Limited papulosquamous small plaques on volar surface of the hands of the same patient. With treatment, complete resolution occurred within 3 months.

cells were not prominent. Focal parakeratosis was demonstrated, as was focal diminution of the granular layer. Neutrophils were noted within the epidermis and the stratum corneum epidermidis. Results of silver staining and immunohistochemical staining with monoclonal antibodies were negative for *T pallidum*.

The patient was treated with oral cephalexin 500 mg 4 times per day for 14 days, because the staff thought that subclinical streptococcal infection may have played a role in initiation of psoriasis guttata; triamcinolone 0.1% ointment was applied to the affected papules and plaques twice a day. After one month, marked clinical improvement was noted. By 3 months, the patient noted complete resolution of the eruption. On physical examination, only an asymptomatic keloid at the biopsy site and transverse onychomalacia (Beau lines) 6 to 8 mm from the proximal nail fold were noted.

Comment

Psoriasis guttata is a relatively rare form of psoriasis, accounting for just 2% to 10% of all cases.^{5,6} It may be substantially more common in children.⁷ The classic presentation consists of an abrupt onset of small, diffuse, salmon-colored, papulosquamous papules with silver scale.⁶ The appellation is derived from the Latin word *gutta*, which means *a drop*, and the clinical presentation often justifies this morphologic analogy. Primarily a disease of youth, psoriasis guttata typically affects persons younger than 30 years. The trunk and proximal extremities are commonly involved. Lesions on the palms and soles have been documented rarely in the literature.²

The pathophysiologic mechanism underlying psoriasis guttata is undetermined. Streptococcal infection in the weeks prior to eruption is a commonly suspected trigger.^{8,9} Autoimmune and molecular mimicry phenomena have been postulated, and

some streptococcal products and components have been found to cross-react with normal human epidermis.^{10,11} T cells and evolved cytokines, or superantigens, are believed to cause the characteristic inflammatory changes seen on histopathologic examination.^{12,13}

Microscopic findings in psoriasis guttata vary and include mild epidermal hyperplasia; focal spongiosis; disappearance of the granular layer; and exocytosis of neutrophils, which migrate into the epidermis to produce epidermal or subcorneal collections known as *spongiform pustules of Kogoj* or *Munro microabscesses*, respectively.¹⁴

Other conditions that should be considered in the differential diagnosis include secondary syphilis, PLC, and atypical pityriasis rosea. In this case, secondary syphilis was of particular concern given the substantial involvement of the palms and soles. Results of both rapid plasmin reagin test and a microhemagglutination-*T pallidum* test to screen for specific antibodies to *T pallidum* were negative. These serologic tests possess nearly 100% sensitivity in secondary syphilis.^{15,16} Because of the high rate of syphilis infection in patients seen at Parkland Memorial Hospital, serial dilutions to prevent prozone phenomenon are routinely performed.¹⁷

Microscopic examination of the biopsy specimen did not demonstrate the presence of plasma cells, which are characteristic of secondary syphilis. Results of silver staining were normal, as were those of immunohistochemical staining with antibodies to *T pallidum*, a technique known to increase sensitivity.^{18,19} PLC tends to follow a more protracted clinical course than psoriasis guttata, and the histopathologic findings in PLC often include focal interface dermatitis, dyskeratotic cells, and extravasated red blood cells—none of which were observed in this case.⁴

Atypical pityriasis rosea, more common in blacks than in whites, rarely involves the palms and soles.³ On clinical examination, the lesions of pityriasis rosea demonstrate a more delicate peripheral scale. A herald patch, not noted by our patient, occurs in more than 50% of cases.²⁰ Results of histopathologic examination of lesions in pityriasis rosea usually show mounds of parakeratosis and extravasated red blood cells, which were not seen in our case.²¹ Neutrophils in the stratum corneum epidermidis, present in this case, militate against pityriasis rosea and instead favor psoriasis guttata, though the difficulty in discriminating between these 2 diseases is well documented.^{21,22}

In conclusion, papulosquamous lesions of the palms and soles can occur in psoriasis guttata and may mimic closely the eruption of secondary syphilis, thereby necessitating appropriate serologic testing or skin biopsy in such situations. This case highlights the utility of a broad differential diagnosis when evaluating papulosquamous eruptions with palmoplantar involvement.

REFERENCES

1. Brown TJ, Yen-Moore A, Tyring SK. An overview of sexually transmitted diseases: part I. *J Am Acad Dermatol*. 1999;41:511-529.
2. Stankler L, Kerr NW. Prominent fungiform papillae in guttate psoriasis. *Br J Oral Maxillofac Surg*. 1984;22:123-128.
3. Imamura S, Ozaki M, Oguchi M, et al. Atypical pityriasis rosea. *Dermatologica*. 1985;171:474-477.
4. Chung HG, Kim SC. Pityriasis lichenoides chronica with acral distribution mimicking palmoplantar syphilid [letter]. *Acta Derm Venereol*. 1999;79:239.
5. Bell LM, Sedlack R, Beard CM, et al. Incidence of psoriasis in Rochester, Minn, 1980-1983. *Arch Dermatol*. 1991;127:1184-1187.
6. Farber EM, Nall L. Guttate psoriasis. *Cutis*. 1993;51:157-159, 163-164.
7. Nanda A, Kaur S, Kaur I, et al. Childhood psoriasis: an epidemiologic survey of 112 patients. *Pediatr Dermatol*. 1990;7:19-21.
8. Whyte HJ, Baughman RD. Acute psoriasis and streptococcal infections. *Arch Dermatol*. 1964;89:350-356.
9. Telfer NR, Chalmers RJ, Whale K, et al. The role of streptococcal infection in the initiation of guttate psoriasis. *Arch Dermatol*. 1992;128:39-42.
10. Perez-Lorenzo R, Zambrano-Zaragoza JF, Saul A, et al. Autoantibodies to autologous skin in guttate and plaque forms of psoriasis and cross-reaction of skin antigens with streptococcal antigens. *Int J Dermatol*. 1998;37:524-531.
11. Villeda-Gabriel G, Santamaria-Cogollos LC, Perez-Lorenzo R, et al. Recognition of *Streptococcus pyogenes* and skin autoantigens in guttate psoriasis. *Arch Med Res*. 1998;29:143-148.
12. Baker BS, Bokth S, Powles A, et al. Group A streptococcal antigen-specific T lymphocytes in guttate psoriatic lesions. *Br J Dermatol*. 1993;128:493-499.
13. Leung DY, Travers JB, Giorno R, et al. Evidence for a streptococcal superantigen-driven process in acute guttate psoriasis. *J Clin Invest*. 1995;96:2106-2112.
14. Brody I. Dermal and epidermal involvement in the evolution of acute eruptive guttate psoriasis vulgaris. *J Invest Dermatol*. 1984;82:465-470.
15. Hart G. Syphilis tests in diagnostic and therapeutic decision making. *Ann Intern Med*. 1986;104:368-376.
16. Castro R, Prieto ES, Santo I, et al. Evaluation of an enzyme immunoassay technique for detection of antibodies against *Treponema pallidum*. *J Clin Microbiol*. 2003;41:250-253.
17. Jurado RL, Campbell J, Martin PD. Prozone phenomenon in secondary syphilis: has its time arrived? *Arch Intern Med*. 1993;153:2496-2498.
18. Phelps RG, Knispel J, Tu ES, et al. Immunoperoxidase technique for detecting spirochetes in tissue sections: comparison with other methods. *Int J Dermatol*. 2000;39:609-613.
19. Hoang MP, High WA, Molberg KH. Secondary syphilis: a histologic and immunohistochemical evaluation. *J Cutan Pathol*. 2004;31:595-599.
20. Allen RA, Janniger CK, Schwartz RA. Pityriasis rosea. *Cutis*. 1995;56:198-202.
21. Ackerman AB. Histologic diagnosis of inflammatory skin disease: a method by pattern analysis. Philadelphia, Pa: Lea & Febiger; 1978.
22. Eslick GD. Atypical pityriasis rosea or psoriasis guttata? early examination is the key to a correct diagnosis. *Int J Dermatol*. 2002;41:788-791.