
CASE REPORT

An unusual rash in an elderly man

This disease is presumed to be transmitted between humans most commonly by droplet nuclei, as the nasal mucosa is frequently involved. It has also been documented in wild armadillos and might be transmitted from armadillo to human.

John Burns, PA-C

CASE

An 84-year-old white male was referred to a teaching hospital dermatology clinic in the southern United States for evaluation of a diffuse erythematous eruption that had been present for 2 years. The eruption was resistant to topical corticosteroids. The condition was asymptomatic despite its widespread nature, but it covered his arms, trunk, and lower extremities (see Figure 1). The eruption was largely confluent; however, there were multiple small nummular patches present. The patient denied any history suggestive of a contact dermatitis or drug eruption, though he was taking multiple medications. Family members reported no similar skin findings.

Histologic examination of a 4-mm punch biopsy specimen revealed a superficial and mid-perivascular infil-

trate dominated by lymphocytes without spongiosis (see Figure 2, page 44). Solar elastosis was found to be present in the dermis, and there was no evidence of vasculitis. Immunohistochemistry studies were performed, and results proved to be consistent with a non-specific reactive process.

At follow-up 1 month after the initial biopsy, the patient complained of numbness in both arms with no other associated neurologic complaints. The previous biopsy specimen was subjected to an acid-fast Fite stain (see Figure 3, page 45). The supplementary pathology

The author practices at Overton Brooks VA Medical Center, Shreveport, Louisiana. He has indicated no relationships to disclose relating to the content of this article.

FIGURE 1



Note the bilateral symmetry of the eruption and the numerous individual patches on the patient's trunk.

IN THIS ARTICLE

Key Points

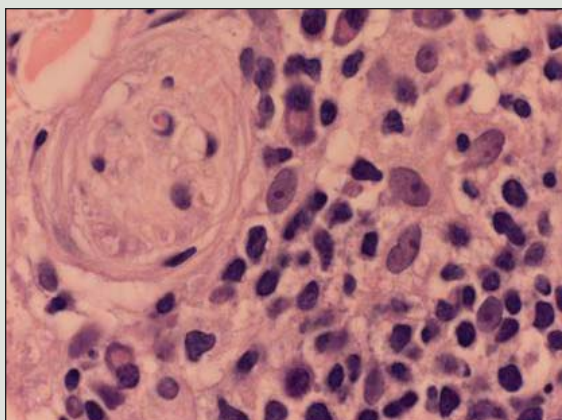
- Leprosy, caused by *Mycobacterium leprae*, may involve multiple organ systems: the skin, peripheral nerves, eyes, mucous membranes, and all visceral organs excluding the GI tract, lungs, and brain.
- Although mycobacteria may gain entry through abraded skin, leprosy is presumed to be more commonly transmitted between humans by droplet nuclei, as the nasal mucosa is frequently involved. Despite the public's fear of leprosy, most people will not develop the disease following exposure to the infection.
- Leprosy reactions are immunologic changes represented by clinical signs and symptoms that may occur during the course of leprosy. There are two major types of leprosy reactions.
- Leprosy treatment is quite effective; the World Health Organization has reported failure rates of approximately 1%.

Competencies

Medical knowledge	◆◆◆◆◆
Interpersonal & communication skills	◆◆◆
Patient care	◆◆
Professionalism	◆
Practice-based learning and improvement	◆
Systems-based practice	◆

For an explanation of competencies ratings, see the table of contents.

FIGURE 2



Hematoxylin-eosin stain of the biopsy specimen under oil immersion at 100x magnification reveals a superficial and mid-perivascular infiltrate dominated by lymphocytes without spongiosis.

report described acid-fast bacilli in foamy histiocytes, the perivascular infiltrate, and the nerves.

Does this patient have tinea corporis, a drug eruption, subacute cutaneous lupus erythematosus (SCLE), or leprosy?

DISCUSSION

Tinea corporis is a cutaneous fungal infection of the neck, trunk, arms, and/or legs. The typical clinical presentation is that of a scaling, well-circumscribed, slightly indurated, erythematous, pruritic plaque.¹ This patient's eruption was asymptomatic, diffuse, and predominantly confluent.

A drug reaction may manifest as a bilaterally symmetrical eruption involving most or all of the body's surface area. The history will include use of a medication started approximately 10 days before the eruption (unless there has been prior exposure), and the eruption is typically pruritic.^{2,3} Although this patient did have a diffuse, bilaterally symmetrical eruption, it was asymptomatic and there was no history of newly initiated medication.

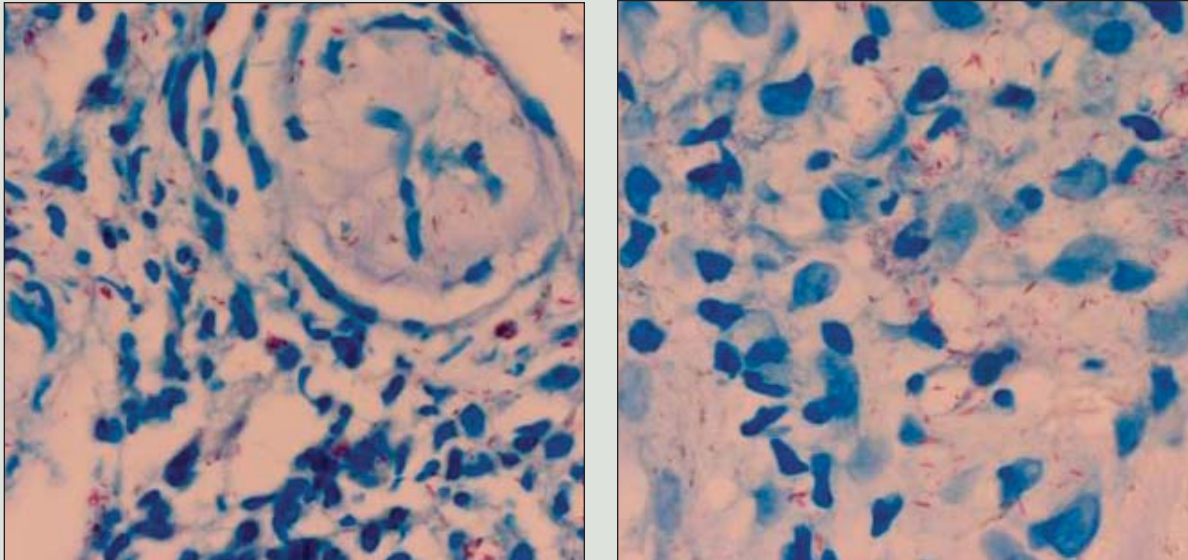
SCLE is more common in women. Typically there is a mildly symptomatic erythematous, scaling eruption commonly involving sun-exposed regions including the head, neck, upper trunk, arms, and dorsum of the hands. Histologic and laboratory testing help to confirm the diagnosis.⁴ This patient is male, the eruption involved non-light-exposed regions of the body, and the histologic evaluation was not consistent with SCLE.

The patient was diagnosed as having leprosy, or Hansen's disease, and was admitted to the hospital for evaluation and initiation of treatment.

Leprosy is found worldwide, including in the United States. In 2004, 23 states and Puerto Rico reported a total of 131 cases, 106 of which were in persons born abroad.⁵ Of the 25 endemic leprosy cases reported, 10 were in Texas and 4 were in Louisiana.⁵ Over the past 30 years, the number of newly documented cases of leprosy in this country has remained relatively unchanged.⁵ The average length of time to diagnosis in the United States is 2 years.⁶

Etiology Leprosy is caused by *Mycobacterium leprae*, an acid-fast bacillus.⁷ Although it may gain entry through abraded skin, it is presumed to be more commonly transmitted between humans by droplet nuclei, as the nasal mucosa is frequently involved. Despite the public's trepidation, most exposed persons will not develop the disease.^{6,8} Leprosy has been documented in wild armadillos and potentially could be transmitted from armadillo to human. On subsequent evaluation, this patient admitted to having handled multiple armadillos, most recently 6 to 7 years before the eruption. The incubation period for leprosy ranges from a few months to 40 years, with the average being 2 to 4 years.

FIGURE 3



Fite stain of the biopsy specimen under oil immersion at 100x magnification shows numerous acid-fast bacilli and that organisms have infiltrated a nerve. Note the foamy histiocyte in the image at right.

Multiple organ involvement Leprosy may involve the skin, peripheral nerves, eyes, mucous membranes, and all visceral organs excluding the GI tract, lungs, and brain. Leprosy is typically a progressive disease, and the extent of involvement is determined by the host's ability to mount an effective cellular immune response to *M leprae*. The degree of this response determines a patient's clinical presentation and classification.³

Classification Since 1998, the World Health Organization (WHO) has recommended that the initiation of multiple drug therapy may be based on the number of skin lesions present without a formal quantification of bacterial load. The classification is *paucibacillary* if the patient is not experiencing a leprosy reaction and has five or fewer skin lesions, implying a diminutive bacterial load. If the patient has six or more skin lesions, the classification is *multibacillary*, implying a high bacterial load. Table 1 presents recommended US treatment regimens for patients with paucibacillary and multibacillary disease. Although the WHO classification facilitates treatment, it is less effective in providing information regarding the potential for a leprosy reaction.^{9,10} The Ridley-Jopling system provides an insightful clinical classification based on the patient's immunologic response to the infection. It also permits a prediction of which patients could experience a leprosy reaction (see Table 2, page 46).

Tuberculoid leprosy denotes an adequate immune response to *M leprae*; *lepromatous leprosy* denotes an inadequate response. Interpolated between the tubercu-

loid and the lepromatous classifications are *borderline tuberculoid*, *borderline borderline*, and *borderline lepromatous*. Interpolated classifications are arranged according to declining immunologic response to the infection, beginning with borderline tuberculoid and ending with borderline lepromatous. Declining immunologic response manifests clinically with an increase in the number of lesions, nodularity of individual lesions, and bacterial load. A tendency for disease progression towards the lepromatous classification is observed in patients whose initial classifications represent a diminished immune response to the infection. Indeterminate leprosy is a transitory state that may terminate with resolution of infection or progress into one of the other categories of the Ridley-Jopling classification system, depending on immune response.^{3,11} *Continued on page 46*

TABLE 1
US treatment regimen for leprosy

Classification	Recommended treatment
Paucibacillary	Daily for 1 y: dapsone, 100 mg; plus rifampin, 600 mg
Multibacillary	Daily for 2 y: dapsone, 100 mg; plus rifampin, 600 mg; plus clofazimine, 50 mg

Data from US Department of Health and Human Services.¹⁰

TABLE 2

Clinical classification of leprosy based on immune response

Clinical manifestations	Indeterminate	Tuberculoid	Borderline tuberculoid	Borderline borderline	Borderline lepromatous	Lepromatous
Skin	One to few hypopigmented macules	Few hairless, well-defined, erythematous and/or hypopigmented macules or plaques, with scaling and elevated borders	Multiple defined, scaly, hairless, erythematous and/or hypopigmented macules or plaques	Multiple erythematous plaques and/or patches with annular configurations	Numerous shiny, erythematous macules, papules, and nodules; lesions are somewhat bilaterally symmetric	Initially lesions consist of numerous shiny, erythematous macules or papules, which later develop nodularity; lesions are bilaterally symmetric
Neurologic findings in conjunction with dermatologic findings	Skin lesions may have decreased sensation	Loss of sensation in skin lesions; may be enlarged nerves in the surrounding area of skin lesions	Loss of sensation in skin lesions; nerves in the tissue surrounding skin lesions may be enlarged	Skin lesions have moderately impaired sensation	Skin lesions may have slightly diminished sensation and there may be enlargement of nerves	Skin lesions have no sensory impairment, though later there is a peripheral neuropathy of the hands and feet
Mycobacteria present in skin lesions	None	None	Rare	Moderate	Many	Many

Data from Ridley DS and Jopling WH.¹¹

Leprosy reactions There are two major types of leprosy reaction, which is an immunologic change represented by clinical signs and symptoms that may occur during the course of leprosy.^{3,7} More than 25% of patients with borderline and lepromatous leprosy may experience a leprosy reaction during the course of their disease.⁸ Those with tuberculoid leprosy do not experience either type of reaction. Leprosy reactions may be triggered by multiple drug therapy for the infection, other infections, other medications, vaccinations, pregnancy, and physical and mental stress.⁷ Antimicrobial treatment should not be stopped in the presence of either reaction.³

Type 1 reactions result from the interaction of T lymphocytes with antigens derived from disintegrating *M leprae* and occur only in patients with borderline disease.⁷ Of course, the closer the disease is to the lepromatous classification, the greater the bacterial load and the

greater the potential antigen load. Type 1 reactions are marked by edema, increased erythema, and potential ulceration of existing skin lesions. Nerve damage, however, is the major concern. Clinically, nerves may increase in size and become tender. Neuritis can occur without symptoms but still damage the nerve. Type 1 reactions are typically managed with 40 to 60 mg of oral prednisone daily, which is slowly tapered once the reaction has been controlled. Mild reactions without neurologic changes may be treated with bed rest and aspirin or NSAIDs.^{3,9}

Type 2 reactions, also called *erythema nodosum leprosum*, occur in patients with borderline lepromatous and lepromatous disease. They may manifest with fever, arthralgias, myalgias, and anorexia—unlike type 1 reactions, which produce no systemic signs. Erythematous, tender nodules appear in large numbers, typically on the extensor surfaces of the extremities.

Conjunctivitis, neuritis, keratitis, iritis, synovitis, nephritis, hepatosplenomegaly, orchitis, and lymphadenopathy may also occur. The reaction may last from a few days to years.³ Type 2 reactions without neuritis may be managed with thalidomide; however, neuritis requires systemic corticosteroids.⁸ The frequency of this reaction type has been reduced to 5% or less since clofazimine was added to the treatment regimen.⁹

Histology Clinically involved tissue from a patient with tuberculoid leprosy will contain no acid-fast bacilli. Conversely, that from a patient with lepromatous leprosy will demonstrate an abundance of acid-fast bacilli. Foamy histiocytes are characteristic of lepromatous leprosy.¹¹

Treatment Leprosy treatment is quite effective; the WHO has reported failure rates of around 1%. If treatment failure occurs in a patient with multibacillary disease, the patient should be retreated with the same drug regimen because of the low prevalence of drug resistance. When paucibacillary disease does not respond to treatment, however, the retreatment should be using the regimen for multibacillary disease.⁸ Patients treated for paucibacillary disease should be seen every 6 months for 5 years; those treated for multibacillary disease should be reevaluated every 6 months for 10 years.⁹ Regarding the time frame for potential risk of transmitting infection following treatment initia-

tion, a patient may be considered noninfectious within 2 days of initiating treatment with rifampin.⁸

Outcome The patient in this case received a diagnosis of lepromatous leprosy, which was treated as multibacillary with multidrug therapy. He improved significantly and has yet to experience a leprosy reaction. □

Acknowledgements

The author thanks Michael Kent, MD, Susan Rowland, MD, Ronald Washburn, MD, Tushar Vachharajani, MD, and Naveen Atray, MD, for their assistance with this manuscript.

REFERENCES

1. Gupta AK, Chaudhry M, Elewski B. Tinea corporis, tinea cruris, tinea nigra, and piedra. *Dermatol Clin.* 2003;21(3):395-400.
 2. Crowson AN, Magro CM. Recent advances in the pathology of cutaneous drug eruptions. *Dermatol Clin.* 1999;17(3):537-560.
 3. Odom RB, James VWD, Berger TG. *Andrews' Diseases of the Skin: Clinical Dermatology.* 9th ed. Philadelphia, Pa: Saunders; 2000:430-444.
 4. Patel P, Werth V. Cutaneous lupus erythematosus: a review. *Dermatol Clin.* 2002; 20(3):373-385.
 5. Pfeifer LA. *A summary of Hansen's disease in the United States-2004.* Baton Rouge, La: National Hansen's Disease Programs; 2004.
 6. Hartzell JD, Zapor M, Peng S, Straight T. Leprosy: a case series and review. *South Med J.* 2004;97(12):1252-1256.
 7. Sehgal VN. Leprosy. *Dermatol Clin.* 1994;12(4):629-644.
 8. Jacobson RR, Krahenbuhl JL. Leprosy. *Lancet.* 1999;353:655-660.
 9. Moschella SL. An update on the diagnosis and treatment of leprosy. *J Am Acad Dermatol.* 2004;51(3):417-426.
 10. US Department of Health and Human Services. National Hansen's Disease programs; standard treatment regimens. Available at: http://bphc.hrsa.gov/nhdp/Standard_Treatment_Regimens.htm. Accessed February 14, 2007.
 11. Ridley DS, Jopling WH. Classification of leprosy according to immunity. A five-group system. *Int J Lepr Other Mycobact Dis.* 1966;34(34):255-273.
-