What is causing this man's worsening skin lesions?

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CASE

A 36-year-old man with HIV presented to the infectious-disease clinic for complaints of generalized, mildly pruritic, nontender skin lesions that worsened every time he took his antiretroviral medications.

History The patient said that the lesions had been present for 5 years but had worsened over the past 6 months. During that time, the lesions had been treated with topical corticosteroids, oral corticosteroids, and antifungal medications (topical and oral) without significant improvement. The lesions improved when he was nonadherent to his antiretroviral regimen. His most recent CD4 count was 35 cells/mm^3 and the plasma HIV-1 RNA viral load was greater than 100,000 copies/mL. This is consistent with a diagnosis of AIDS and nonadherence to antiretroviral medications.

A review of systems was significant for aching and pain in his hands, wrists, and feet. He denied fevers, chills, night sweats, weight loss, malaise, and cough. He immigrated from Southeast Asia 4 years ago and denied recent travel outside of the southeast United States. His primary source of income was as a sex worker, and he was homeless, living in hotels and on the street.

Physical examination The patient's vital signs were heart rate, 89 beats/minute; BP, 122/67 mm Hg; respirations, 16; and oral temperature, 98.7° F (37.1° C). He weighed 50 kg (110 lb) and was 5 ft 4 in (163 cm). The patient was cachectic but in no acute distress. The head, eyes, ears, nose, and throat examination was significant for oral mucocutaneous candidiasis and nontender anterior cervical adenopathy. Cardiovascular, respiratory, gastrointestinal, and genitourinary examinations were benign. The neurologic examination was significant for decreased two-point discrimination, decreased vibratory sensation, and inability to sense 10g monofilament test, consistent with peripheral neuropathy. Dermatologic examination revealed multiple, nontender, 2- to 4-cm round, erythematous, circumscribed lesions with raised borders across the patient's entire body, with a predominance on his arms, legs, and face.

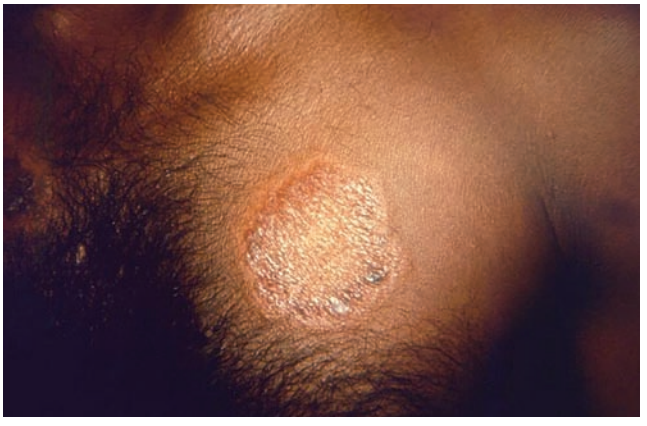

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FIGURE 1. Similar lesions on a different patient.

DIFFERENTIAL DIAGNOSIS

- systemic fungal infection
- systemic bacterial infection
- cutaneous mycobacterial infection (*M. tuberculosis*, *M. marinum*, or *M. leprae*)
- granuloma annulare

OUTCOME

Herpes simplex viral culture of the lesions, rapid plasma reagin, tuberculin skin test, and bacterial and fungal blood cultures were negative. Initial biopsy results were significant for lymphocytic infiltration, granulomatous lesions without necrosis, and were negative for aerobic and anaerobic bacteria, yeast, and other fungi. These results made systemic fungal or bacterial infection unlikely. Granuloma annulare was lower on the differential given the duration of the patient's symptoms, neurologic findings, and absence of necrosis on histopathology. The presence of granulomatous lesions with lymphocytes, along with the patient's history and physical examination findings, increased suspicion for mycobacterial disease.

A second fresh tissue culture biopsy was sent for acid-fast bacilli, with Ziehl-Neelsen staining, and revealed nontuberculous mycobacterium. The sample was forwarded to the National Hansen's Disease Program (NHDP) for further analysis with Fite acid-fast staining and histopathology,

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confirming the suspected diagnosis of cutaneous *M. leprae*. The patient was referred to the local county health department, which worked in conjunction with the NHDP for treatment planning and surveillance documentation. The patient was started on dapsone, rifampin, and clofazimine for multibacillary (lepromatous) leprosy, and his skin lesions slowly improved over the next 24 months. The patient was lost to follow-up shortly after he completed treatment.

DISCUSSION

Primary care providers treating HIV-positive patients should formulate a broad differential when evaluating atypical dermatologic complaints, especially in patients from outside of the United States.

Leprosy, also referred to as Hansen disease, is caused by infection with *M. leprae*, a slow-growing acid-fast bacillus difficult to culture on standard media. Typically transmitted through respiratory secretions, leprosy is not considered highly contagious, and has an incubation period of 3 to 7 years.[1]

Initial presentation can range from macular skin lesions to nodular, erythematous thickening of the peripheral nerves, which can progress to physical disfigurement, neuropathy, and motor dysfunction.[2] Cases of leprosy-related immune reconstitution syndrome in HIV-positive patients have been reported in the medical literature, and correlate well with the presentation and findings for this case.[3]

The incidence of leprosy is slowly climbing across the United States, with 178 new cases reported in 2015. Of the newly documented cases, 57% were in people born outside the United States, most of whom were from the South Pacific. Transmission also has been linked to zoonotic exposure to the nine-banded armadillo, predominately in Texas and Louisiana. A significant rise in leprosy has been reported in Florida, where this patient lived.[4]

NHDP treatment recommendations are based on WHO classification, although US recommendations call for a longer duration of therapy than that recommended by WHO.[4] Leprosy classification includes a continuum from tuberculoid forms (limited to local skin lesions) to lepromatous forms that manifest as systemic infection and debilitation. Treatment for adults with the tuberculoid (paucibacillary) form is dapsone 100 mg daily plus rifampin 600 mg daily for 12 months. For adults with the lepromatous (multibacillary) form, treatment is dapsone 100 mg daily, rifampin 600 mg daily, and clofazimine 50 mg daily for 24 months.

Although rare, leprosy should remain in the differential diagnosis for at-risk patients presenting with chronic, nonhealing granulomatous skin lesions of unknown cause.[2] Consult the NHDP for suspected cases. **JAAPA**

REFERENCES

1. Nolen L, Haberling D, Scollard D, et al. Incidence of Hansen's disease—United States, 1994-2011. *MMWR Morb Mortal Wkly Rep*. 2014;63(43):969-972.
2. Keystone JS. Imported leprosy: if you think about it, you can diagnose it. *Travel Med Infect Dis*. 2016;14(4):297-298.
3. Lawn SD, Bekker LG, Miller RF. Immune reconstitution disease associated with mycobacterial infections in HIV-infected individuals receiving antiretrovirals. *Lancet Infect Dis*. 2005;5(6):361-373.
4. US Department of Health and Human Services. Health Resources and Services Administration. National Hansen's Disease Program. A summary of Hansen's disease in the United States—2015. https://www.hrsa.gov/hansensdisease/pdfs/hansens2015report.pdf. Accessed June 2, 2017.

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