A 78-year-old man was referred to our clinic for evaluation of 2 painless round ulcers with an undermined edge and purulent discharge on the left posterior leg of 2 months’ duration. The ulcers had appeared following a presumed trauma. He had received repeated courses of oral antibiotics and antifungals without improvement. No regional lymphadenopathy could be detected. Biochemical analyses were within reference range. Human immunodeficiency viruses 1 and 2, hepatitis B and C antibodies, and a VDRL test were all negative.

**WHAT’S THE DIAGNOSIS?**

a. cutaneous tuberculosis  
b. necrobiotic xanthogranuloma  
c. pyoderma gangrenosum  
d. squamous cell carcinoma  
e. stasis ulcer
THE DIAGNOSIS:
Cutaneous Tuberculosis

The patient’s medical history was notable for bone tuberculosis (TB) treated in childhood. Skin biopsy revealed neutrophil infiltrates with necrosis without granulomas. A real-time polymerase chain reaction test detected Mycobacterium tuberculosis complex in the skin fragment, which was confirmed by culture of the biopsy specimen using a liquid growth medium that grew M tuberculosis. Tuberculous foci were not present on the lungs, gastrointestinal tract, kidneys, and bones by radiologic, microbiologic, and ultrasonographic investigations. The patient was started on 4 antituberculous drugs—isoniazid 300 mg, rifampicin 600 mg, ethambutol 1200 mg, pyrazinamide 1500 mg—once daily for 2 months followed by isoniazid 300 mg and rifampicin 600 mg once daily for another 4 months with resolution of the skin lesions.

Cutaneous TB is an infectious disease caused by M tuberculosis and accounts for only 1.5% of extrapulmonary TB cases.1,2 Similar to other forms of TB, a resurgence of cutaneous TB has been noted in parts of the world where human immunodeficiency virus infection is prevalent and remains to be one of the most elusive and more difficult diseases to diagnose.3 Thought to be a predominantly occupational disease, it is being encountered more frequently in healthy individuals where the source of infection remains unidentified in most cases.4 The clinical types depend on the method of infection, virulence of the bacillus, immune status of the host, and presence or absence of host sensitization to M tuberculosis.5 The route of infection is used to classify cutaneous mycobacteriosis.6 Inoculation from an exogenous source can produce TB verrucosa cutis in individuals who have previously been sensitized to M tuberculosis or tuberculous chancre in individuals without prior exposure to the bacterium.4 Cutaneous TB resulting from direct spread to the skin from an underlying contiguous structure in most cases spreads from lymph nodes and bone (sacrofudoderma). Immunosuppressed patients with advanced TB of the lung, gastrointestinal tract, or the genitourinary tract may present with periorificial TB.4 Dissemination to the skin caused by hematogenous spread can occur in the form of lupus vulgaris, miliary TB, or metastatic tuberculous abscesses (gumannas).5,6 A fourth category—cutaneous TB from paradoxical expansion—also was proposed. Paradoxical expansion is defined as the transient expansion of a preexisting lesion or the appearance of new lesions during appropriate anti-TB therapy.5

Although histopathology and protein chain reaction tests are useful, the gold standard for diagnosis is still the isolation of M tuberculosis on culture.5,6 Treatment regimens of cutaneous TB are similar to those of pulmonary TB, with a 4-agent regimen given for 2 months followed by a 2-drug regimen for the next 4 months.1,7 The differential diagnosis of leg ulcers includes stasis ulcer, necrobiosis xanthogranuloma, pyoderma gangrenosum, and squamous cell carcinoma, among others. Cutaneous biopsy, microbiological culture, and a high degree of suspicion are fundamental for the final diagnosis. Cutaneous TB should be suspected in immunocompetent as well as in immunosuppressed patients who present with ulcerated lesions that do not respond to antibacterial treatment.

REFERENCES