

## Medical Mystery: Painless Ulcers — The Answer

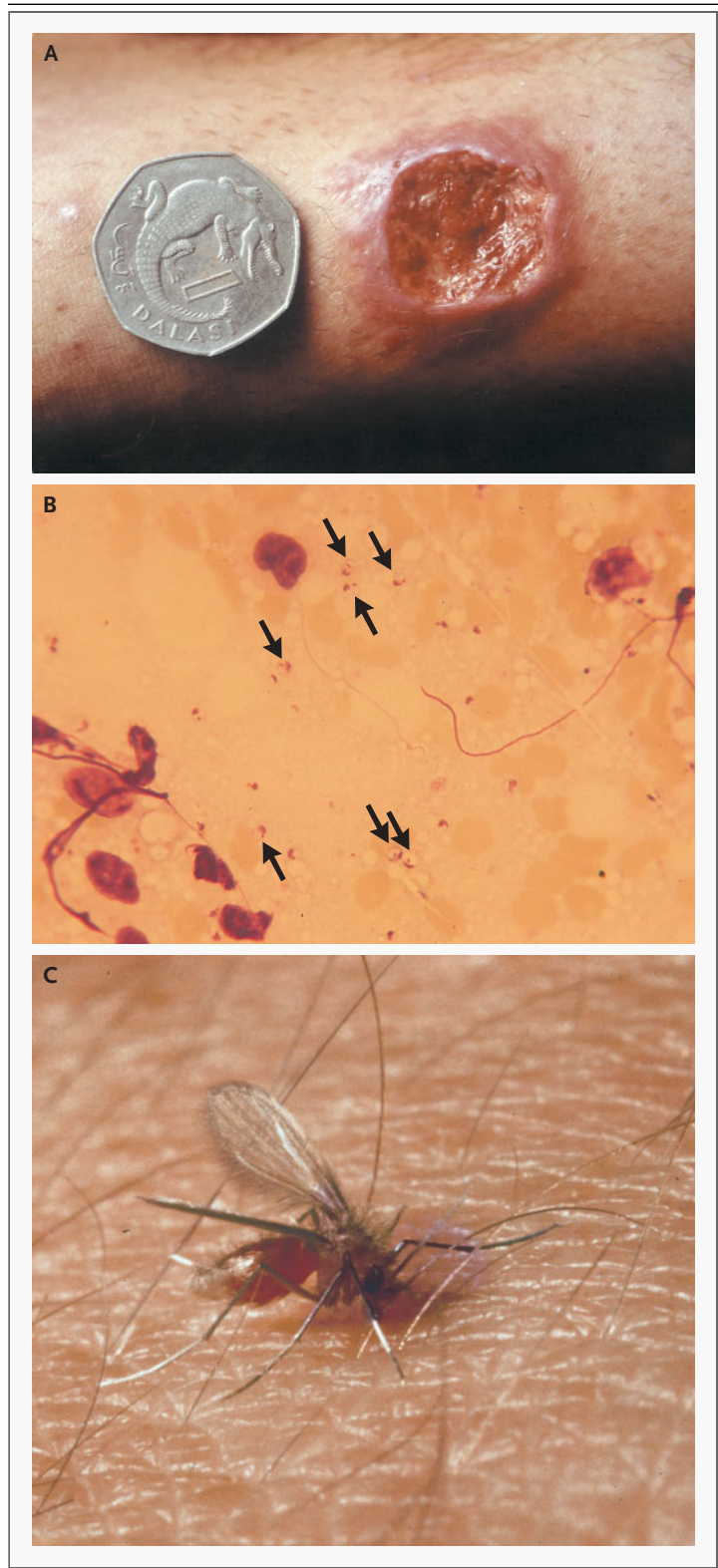
**TO THE EDITOR:** The medical mystery in the April 1 issue<sup>1</sup> involved a 23-year-old Peace Corps volunteer in Gambia. Painless ulcers developed on his lower legs (Fig. 1A) and did not respond to local or systemic antimicrobial therapy.

A slit-skin smear from the margin of the ulcer showed leishmania amastigotes (Fig. 1B). Given the patient's geographic exposure, these parasites were *Leishmania major*. The main animal reservoirs for the protozoan in this geographic area are burrowing rodents, and the vector is the phlebotomus sandfly (Fig. 1C). The lesions remain localized and usually heal spontaneously within one year, with scarring, although pentavalent antimonial compounds may accelerate recovery. The differential diagnosis includes tropical ulcer, usually painful and rapidly responsive to  $\beta$ -lactam therapy, and Buruli ulcer, caused by *Mycobacterium ulcerans*. Buruli ulcers initially spread subcutaneously and therefore characteristically have undermined borders.

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*Editor's note:* We received 861 responses to this medical mystery — 60 percent from physicians in practice, 20 percent from physicians in training, 16 percent from medical students, and 4 percent from other readers. Responses were received from 59 countries. Sixty percent correctly identified this condition as due to leishmania. Cutaneous leishmaniasis results from the bite of an infected sandfly, which produces a slow-healing skin ulcer. The most common alternative diagnosis was Buruli ulcer, caused by *M. ulcerans*, suggested by 20 percent. Treponemal infection was suggested by 6 percent, and pyoderma gangrenosum by 3 percent. Other suggested diagnoses include coining, spider bite, botryomycosis, nocardiosis, African histoplasmosis, leprosy, and tuberculosis. Many insightful comments and terms appeared in the responses, in-



**Figure 1. Leishmania in a Peace Corps Volunteer.**

Panel A shows a painless ulcer on the man's leg. Panel B shows leishmania amastigotes (arrows; Giemsa,  $\times 100$ ). Panel C shows the phlebotomus sandfly, the vector of the parasite.

cluding “Avoid sandflies,” “Baghdad boil,” and “We have seen a lot of this in troops returning from Iraq.” One respondent wrote, “Unlike the crocodile portrayed on the coin, the predator in this case gave up its tail several weeks before the

photo was taken. The patient has Old World cutaneous leishmaniasis — the Rose of Jericho.”

1. Morris-Jones S, Weber M. A medical mystery — painless ulcers. *N Engl J Med* 2004;350:1442.

## Case 6-2004: Severe Burns from a Nightclub Fire

**TO THE EDITOR:** Sheridan et al. (Feb. 19 issue)<sup>1</sup> fail to consider that carbonaceous sputum, confusion, agitation, dyspnea, and lactic acidosis in their burned patient are all signs of potential cyanide poisoning from smoke inhalation. This oversight is troubling and, unfortunately, common. Since one of us and our colleagues described the role of cyanide in death due to smoke inhalation and its strong association with increased plasma lactate concentrations,<sup>2</sup> numerous investigators have confirmed the importance of cyanide in this setting. The patient under discussion had multiple potential sources of lactate, including possible crush injury, extensive burns, carbon monoxide intoxication, and hypovolemia. The source of lactic acid in patients with burns is of more than academic interest. Jeng et al.<sup>3</sup> confirmed that lactate values are inversely related to survival in patients with burn injuries. They suggested many potential sources of hyperlactatemia but never considered cyanide. We have recently shown that lactate is a valuable early marker in pure cyanide poisoning<sup>4</sup> but not in pure carbon monoxide poisoning.<sup>5</sup> Cyanide poisoning is a deadly but treatable complication of smoke inhalation that should be considered in every patient who presents with altered mental status, hypotension, a low respiratory rate, or lactic acidemia.

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1. Case Records of the Massachusetts General Hospital (Case 6-2004). *N Engl J Med* 2004;350:810-21.

2. Baud FJ, Barriot P, Toffis V, et al. Elevated blood cyanide concentrations in victims of smoke inhalation. *N Engl J Med* 1991;325:1761-6.

3. Jeng JC, Jablonski K, Bridgman A, Jordan MH. Serum lactate, not base deficit, rapidly predicts survival after major burns. *Burns* 2002;28:161-6.

4. Baud FJ, Borron SW, Megarbane B, et al. Value of lactic acidosis in the assessment of the severity of acute cyanide poisoning. *Crit Care Med* 2002;30:2044-50.

5. Benaissa ML, Megarbane B, Borron SW, Baud FJ. Is elevated plasma lactate a useful marker in the evaluation of pure carbon monoxide poisoning? *Intensive Care Med* 2003;29:1372-5.

**THE DISCUSSANTS REPLY:** Dr. Borron and colleagues bring up the issue of cyanide toxicity in inhalation injury. This consideration did not apply to this patient, since she presented to our institution approximately 11 hours after the injury and her measured cyanide concentration (0.4 mg per deciliter) was well within the normal range. Her lactic acidosis was due to hypovolemia. Specific treatment for cyanide exposure in patients with inhalation injury is an area of ongoing controversy. In what is perhaps the most comprehensive study of cyanide toxicity in inhalation injury, 364 cases of death due to fire were evaluated.<sup>1</sup> Cyanide poisoning was found infrequently and was not itself an important cause of death. The authors concluded that specific treatment of cyanide poisoning is rarely necessary and that even if cyanide poisoning is present, it can be well managed with standard supportive care.

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1. Barrillo DJ, Goode R, Esch V. Cyanide poisoning in victims of fire: analysis of 364 cases and review of the literature. *J Burn Care Rehabil* 1994;15:46-57.