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Authors

Fathi, Ramin
Fathi, Ahmad

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Case presentation

A case of cutaneous leishmaniasis found in Indiana

Ramin Fathi, BA, Ahmad Fathi, MD

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Rush University Medical Center, Chicago, Illinois

Correspondence

Ramin Fathi, BA

Rush University Medical Center

Chicago, Illinois, USA

rfathi1@gmail.com

Abstract

Cutaneous leishmaniasis was diagnosed in an Indiana resident, an 80-year-old man who had visited Afghanistan 7 months earlier. Although cultures were negative, skin biopsy demonstrated round to oval bodies that stained strongly positive with Giemsa staining. His ulcerated plaques cleared readily with oral ketoconazole.

Case Synopsis

An 80-year-old man presented with growing indolent lesions on both hands for 4 months. The plaques appeared raised, ulcerated, and relatively non-tender. They measured 1-5 cm in diameter and were located on the dorsum of the left hand and the dorsum of the right fourth finger. Pink-bluish induration of all borders was noted (Figure 1) with several 1 cm pink indurated nodules extending to the middle of the dorsal aspect of the right forearm (not shown). Lymph nodes were not palpable and the rest of the physical exam was unremarkable. Past history was significant only for coronary artery disease. The patient had visited Jalalabad, Afghanistan 7 months prior. The patient previously tried topical corticosteroids and systemic and topical antibiotics with no response. Positive lab tests include low RBC (3.47 M/uL; normal 4.20-5.70), low Hgb (11.0 g/dL; normal 12.0-17.5), low Hct (31.9%; normal 35.0-52.5), and neutropenia (50.6%; normal 55.0-72.0). A bacterial culture of the lesions grew only normal flora. A punch biopsy was taken for histologic and immunohistochemical analysis (Figure 3, Figure 4).

Lesional biopsies of both specimens demonstrate a similar spectrum of changes. The skin surface demonstrates extensive ulceration, necrosis, acute inflammation, and bacterial overgrowth. The underlying dermis displays a dense, predominantly chronic inflammatory infiltrate with numerous granulomas. Present diffusely throughout the specimen are small bluish round to oval bodies. The organisms additionally stain strongly with Giemsa. GMS and Ziehl-Neelsen fail to demonstrate fungal or acid-fast organisms. Immunohistochemical staining demonstrates a mixed T and B cell population with T cells predominating (CD3, L-26). CD5, CD10, CD23, BCL-2 and BCL-6 do not demonstrate aberrant staining patterns. Evidence for involvement by non-Hodgkin's lymphoma is not detected. Biopsy specimens were sent to the Center for Disease Control laboratories, which confirmed the diagnosis of Cutaneous Leishmaniasis. Culture and PCR confirmed the diagnosis of CL and speciation proved to be *Leishmania Tropica*. The patient was effectively treated with 200 mg of oral ketoconazole daily for 1 month, which produced resolution of all lesions and satellite nodules; residual scarring remained (Figure 2).



Figure 1 Ulcerated plaques with violaceous borders

Figure 2 Scarred plaques after treatment with ketoconazole

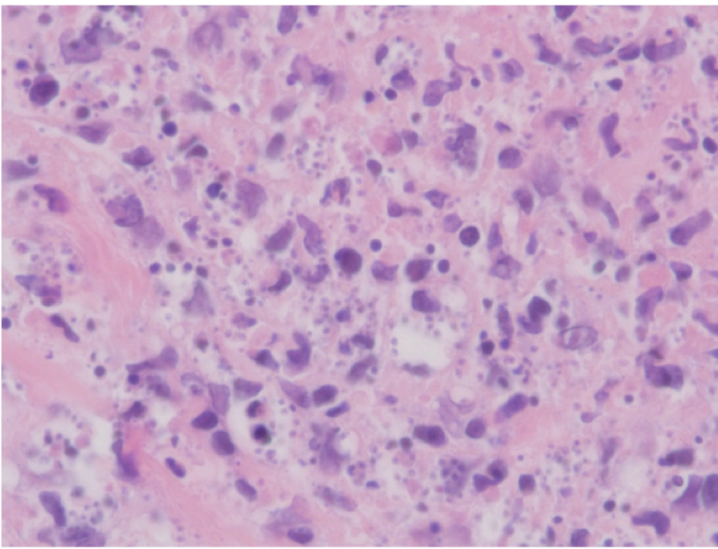


Figure 3 Small bluish round to oval bodies

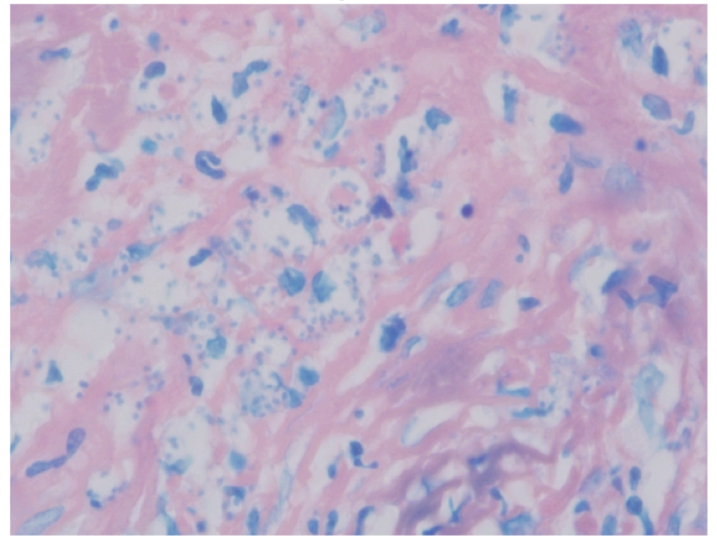


Figure 4 Organisms stain strongly with Giemsa

DISCUSSION

Cutaneous leishmaniasis (CL) is a vector-borne disease transmitted by the bite of the sand fly caused by several species of the obligate intramacrophage protozoa of the genus *Leishmania*. The annual incidence of CL, caused mainly by *L. major* and *L. tropica*, is 1 to 1.5 million cases with 90% confined to Afghanistan, Algeria, Brazil, Iran, Peru, Saudi Arabia, and Syria [1]. Patients at risk in the United States include overseas travelers to endemic areas. CL infections can occur even after brief stays in endemic areas. However, infections can remain subclinical or become clinically apparent after a variable incubation period that averages several weeks. Typically in the localized form, the lesions evolve from papules, to nodules, to ulcerative plaques varying in size from 0.5 to 3 cm with a central depression and raised, indurated border. After months to years, an atrophic scar forms [2].

The diagnosis ideally would be confirmed by demonstration of the parasite, but this can be difficult. Examination of Giemsa-stained slides under oil immersion for amastigotes, the tissue form of the parasite, is the most commonly used technique. Amastigotes are distinguished by their characteristic size (2-4 μm in diameter), shape (round to oval), and internal organelles (the nucleus and kinetoplast). The cytoplasm typically stains pale blue and the nucleus and kinetoplast stain pinkish-red or violet-blue. Stains to rule out mycobacterial, fungal, and other infectious causes as well as lymphoproliferative disorders should be used [3]. If the parasite cannot be detected by smear, Nicolle-Novy-McNeal medium culture, histopathologic examination, or leishmanin skin tests can be used [4]. The Montenegro skin test uses leishmanial antigen to induce a cell-mediated response. Polymerase Chain reaction is the most sensitive diagnostic test [5].

The gold standard for treatment of CL is stibogluconate sodium, a pentavalent antimonial solution (96% cure rate), but it must be given parenterally [3]. Other options including *-azole* derivatives can be used depending on the risk of mucosal leishmaniasis and the degree to which the skin lesions are bothersome because CL can be a self-limited disease. Systemic treatments are generally given to CL patients with big, multiple, or disseminated lesions, those who have simple lesions involving cosmetically sensitive areas or joints, or those who present with nodular lymphangitis [6]. Mild disease is often managed with local care alone and may not require other specific therapies [7].

REFERENCES

1. Khatami A, Firooz A, Gorouhi F, Dowlati Y. Treatment of acute Old World cutaneous leishmaniasis: a systematic review of the randomized controlled trials. *J Am Acad Dermatol*. 2007 Aug;57(2):335.e1-29. Epub 2007 Mar 6. PMID: 17337090
2. González U, Pinart M, Reveiz L, Alvar J. Interventions for Old World cutaneous leishmaniasis. *Cochrane Database Syst Rev*. 2008 Oct 8;(4):CD005067. PMID: 18843677
3. Herwaldt BL. Leishmaniasis. *Lancet*. 1999 Oct 2;354(9185):1191-1199. PMID: 10513726
4. Ceyhan AM, Yildirim M, Basak PY, Akkaya VB, Erturan I. A case of erysipeloid cutaneous leishmaniasis: atypical and unusual clinical variant. *Am J Trop Med Hyg*. 2008 Mar;78(3):406-8. PMID: 18337335
5. Meymandi SS, Bahmanyar M, Dabiri S, Aflatonian MR, Bahmanyar S, Meymandi MS. Comparison of cytologic giemsa and real-time polymerase chain reaction technique for the diagnosis of cutaneous leishmaniasis on scraping smears. *Acta Cytol*. 2010 Jul-Aug;54(4):539-45. PMID: 20715653
6. Reithinger R, Mohsen M, Wahid M, Bismullah M, Quinnell RJ, Davies CR, Kolaczinski J, David JR. Efficacy of Thermoherapy to Treat Cutaneous Leishmaniasis by *Leishmania tropica* in Kabul, Afghanistan: A Randomized, Controlled Trial. *Clin Infect Dis*. 2005 Apr 15;40(8):1156-1158. PMID: 15791515
7. Ramanathan R, Talaat KR, Fedorko DP, Mahanty S, Nash TE. A Species-Specific Approach to the Use of Non-Antimony Treatments for Cutaneous Leishmaniasis. *Am J Trop Med Hyg*. 2011 Jan;84(1):109-117. PMID: 21212212