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Photo Vignette

An unusual erysipelas-like presentation

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Abstract

A 61-year-old man presented with erysipelas-like cutaneous leishmaniasis.

Case synopsis

A 61-year-old man presented with a three-month history of a slowly enlarging lesion on the right temple that had become progressively painful and spread to involve the right eyelid. He had been on holiday in Ibiza five weeks before the onset of symptoms.

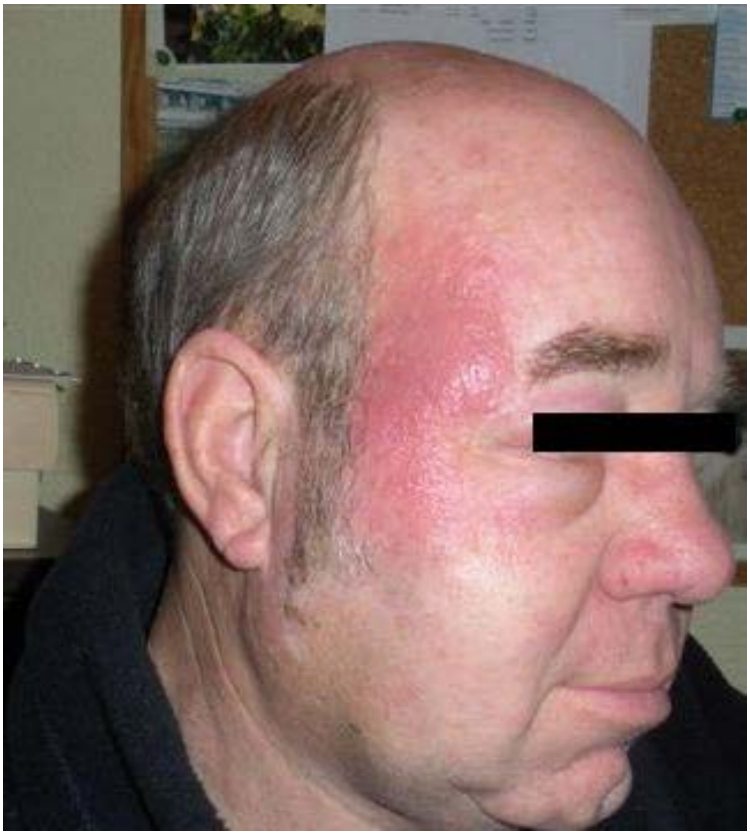

Figure 1. An erythematous, edematous plaque with central crust and superficial ulceration on the right temple and prominent peri-orbital edema

Clinical examination revealed an erythematous, edematous plaque with central crust and superficial ulceration on the right temple and prominent periorbital edema resembling erysipelas. He did not respond to treatment with flucloxacillin and topical steroid (Figure 1).

Histological findings: Incisional biopsy (Figure. 2) from the lesion demonstrated a dense dermal mixed inflammatory cell infiltrate. In addition, there were numerous small round to oval basophilic organisms in histiocytes that exhibited weak Giemsa staining.

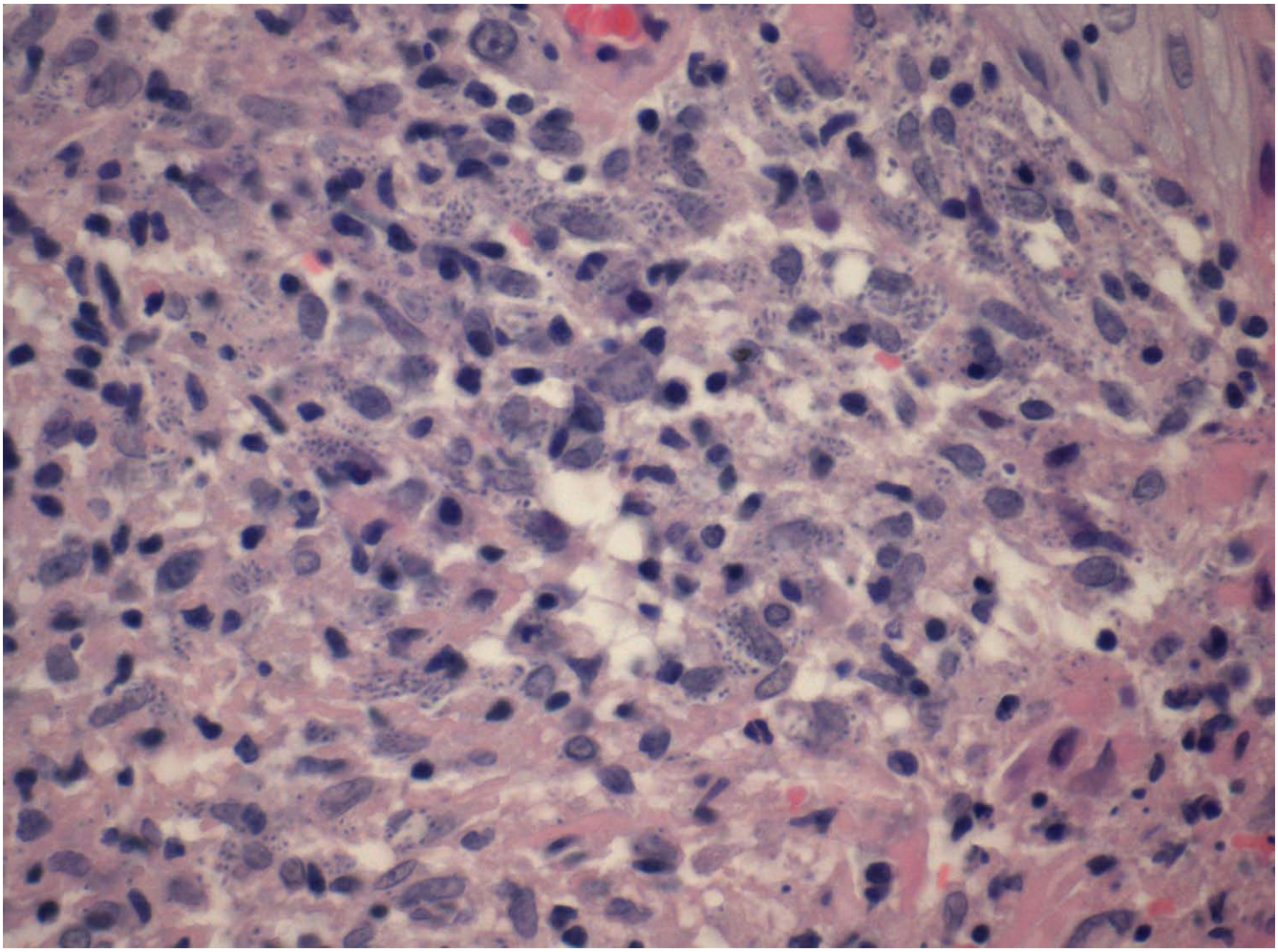

Figure 2. Numerous small round to oval basophilic organisms weakly stained with Giemsa stain found in histiocytes.

Laboratory findings: *Leishmania donovani* complex DNA was detected on skin biopsy PCR. Full blood count demonstrated pancytopenia, although bone marrow was normocellular with no histological or PCR evidence of leishmaniasis; abdominal ultrasound was normal. Blood leishmania IFAT and PCR were negative. HIV serology was negative. Bacterial swab grew no pathogens.

Treatment : In view of the severity of the infection and the proximity to the patient's eye, he was commenced on intravenous sodium stibogluconate (20mg/kg) for 20 days. Treatment was complicated by cardiac complications associated with prolonged QT interval related to hypokalemia (2.3mmol/L) attributed to sodium stibogluconate. He has since completed treatment and has residual mild periorbital edema.

Discussion

Cutaneous leishmaniasis (CL) is caused by the intracellular protozoa of the genus Leishmania, usually transmitted to human beings and animals by infected female sandflies. However, it can be transmitted via contaminated needles, blood transfusions and vertically. The disease is endemic in 82 countries, and WHO estimates 10 million people suffer cutaneous leishmaniasis today [1]. CL in the Old World is caused by L *major*, L. *tropica*, L *infantum,* and L. *donovani*, which are present in southern Europe, North Africa, the Mediterranean, the Middle East, the Indian subcontinent, and Central Asia [1]. Although several *Leishmania spp* can cause CL in human beings, most infections probably remain symptomless [2]. The classical course of this disease begins with the appearance of small papules, which progress to ulcerated plaques or nodules on exposed sites. We report a patient who demonstrated a rare and unusual presentation of CL mimicking periorbital cellulitis.

The erysipelas-like presentation of CL is rarely reported. The reason for clinical cutaneous leishmaniasis pleomorphism is unclear, but variations in parasite virulence and host factors, including abnormal host immune response, malnutrition, and immunosuppression have been postulated to affect the presentation [3,4]. Suspicious lesions can be sampled by aspiration, scraping, and biopsy. Parasitological diagnosis remains the gold standard, which includes microscopic examination of Giemsa-stained biopsy smears or aspirates and histological examination of skin biopsies. Molecular confirmation of CL is by PCR. However, its use is often limited by the availability of laboratory infrastructure and cost. WHO recommends treating patients with CL who present with large, multiple or disseminated lesions with pentavalent antimonial drugs (i.e. sodium stibogluconate) at 20mg/kg per day (given IV or IM) for 20 or 28 days for cutaneous or mucosal involvement, respectively [5]. In the US, the drug may be obtained from the Centers for Disease Control (CDC).

Although pentavalent antimonial drugs accelerate cure and reduce scarring, they can potentially cause serious side effects such as cardiotoxicity. The incidence of secondary bacterial infections in the lesions of CL ranged from 21.8% to 54.2% [6].

This case highlights an unusual presentation of CL in an apparently low-risk immunocompetent individual, the presence of leishmaniasis in southern-Europe, and the potential complications of treatment. The associated pancytopenia with no evidence of bone marrow or visceral involvement is suggestive of an immune-mediated epiphenomenon.

References

1. 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)
2. Goto H, Lindoso JA. Current diagnosis and treatment of cutaneous and mucocutaneous leishmaniasis. Expert Rev Anti Infect Ther. 2010;8:419–433. (PMID: 20377337)
3. Masmoudi A, Hariz W, Marrekchi et al. Old World cutaneous leishmaniasis: diagnosis and treatment. J Dermatol Case Rep. 2013 June 30;7(2): 31-41. (PMID: 23858338)
4. Reithinger R, Dujardin JC, Louzir H. Cutaneous leishmaniasis. Lancet Infect Dis. 2007. Sep;7(9):581-96 (PMID:17714672)
5. http://www.cdc.gov/parasites/leishmaniasis/health_professionals/index.html
6. Ziaie H, Sadeghian G. Isolation of bacteria causing secondary bacterial infection in the lesions of Cutaneous Leishmaniasis. Indian J Dermatol. 2008;53(3):129-31. (PMID: 19882011)