

Cutaneous Leishmaniasis: About Two Cases

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Abstract

Case Report

Cutaneous leishmaniasis, also known as the “Oriental sore,” is an anthroponosis caused by a parasite of the *Leishmania* genus, transmitted by the bite of a sandfly (phlebotomine). It is endemic in our country. The polymorphic skin involvement warrants parasitological examination of a lesion smear and often histological analysis. Treatment typically involves local injections of Glucantime.

Keywords: Cutaneous leishmaniasis; face; ear.

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INTRODUCTION

Cutaneous leishmaniasis affects both sexes and all age groups. It is an anthroponosis in our country, capable of affecting the face or the ear. The ulcerative-vegetative form is frequently observed. We report two cases of cutaneous leishmaniasis along with a review of the literature.

CASE REPORT 1

A 4-year-old child with no significant medical history had traveled to the province of Sefrou. A few days after the trip, an ulcerated lesion appeared on the earlobe, prompting consultation. Despite multiple courses of topical and systemic antibiotics, no improvement was noted. A biopsy was subsequently performed, confirming the diagnosis (Figure 1).



Ear Cutaneous Leishmaniasis

CASE REPORT 2

A 73-year-old woman, followed for diabetes and hypertension, developed an ulcerated lesion on the

nasal dorsum after a stay in the Azilal region. The diagnosis was confirmed by biopsy (Figure 2).



Nasal Cutaneous Leishmaniasis

DISCUSSION

Cutaneous leishmaniasis, also known as the “Oriental sore,” is a parasitic infection still encountered in dermatology departments across the country. It can be confused with other dermatoses. Awareness and proper diagnosis are crucial for optimal patient management and for epidemiological study of this often underrecognized disease. Following a clinical overview of cutaneous leishmaniasis, we present our modest contribution to the study of this condition [1].

Cutaneous leishmaniasis requires a triad of factors for development:

The parasite (*Leishmania* genus): a flagellated unicellular organism presenting in two forms:

The promastigote form, found in the insect vector

The amastigote form, an ovoid shape measuring 2–5 microns in diameter, found in humans

Reservoir hosts: dogs, cats, wild rodents; humans can occasionally serve as reservoirs.

Vectors: small insects measuring 1–3 mm, resembling mosquitoes (sandflies). The hematophagous female inoculates the parasite into humans after ingesting it from a reservoir host.

Once this triad is established, cutaneous leishmaniasis presents as chronic, painless, polymorphic skin lesions with minimal or no pruritus and no systemic symptoms. Lesions appear in exposed areas (face, neck, hands, feet).[2]

The incubation period varies from 2 weeks to several weeks post-inoculation.

The initial phase manifests as a papular, infiltrated lesion with scales and crusts covering an ulcer on exposed skin. It is generally a solitary lesion.

Cutaneous leishmaniasis can present in many forms, such as [3]:

Chronic, painless ulcerations, sometimes secondarily infected. Facial lesions may mimic basal cell carcinoma.

Papulo-nodular or purely nodular forms

Papulo-squamous forms: may resemble chronic lupus or cutaneous sarcoidosis

Ulcerative-vegetative forms: can mimic mycobacterial or fungal infections

Mucosal involvement (e.g., nasal mucosa) may result from contiguous spread from adjacent cutaneous lesions (e.g., nasal ala).

Two distinct clinical forms deserve mention [4]:

Post-kala-azar dermal leishmaniasis: seen mostly in the Indian subcontinent, manifests as disseminated papulo-nodular lesions following visceral leishmaniasis.

Anergic leishmaniasis: associated with immune deficiency, characterized by widespread papulo-nodular infiltrated lesions, chronically evolving with predilection for nasal and auricular involvement.

However, the most frequently encountered form remains the ulcerated one.

The diagnosis should be considered in endemic areas and is based on histological examination and lesion smear microscopy.

Treatment involves local wound care for proper debridement and, when appropriate, systemic therapy, provided there are no formal contraindications. It is important to note that certain forms may heal spontaneously within a few months (on average, up to one year).

The main therapeutic agents are antimony derivatives, including [5]:

Glucantime®: at a dose of 0.05–0.10 g/kg/day for 2 weeks

Lomidine®: rarely used due to systemic side effects
Because systemic administration may lead to severe, sometimes fatal reactions, local injections are preferred and commonly used.

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