Cutaneous Leishmaniasis: A Clinical Reset

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Introduction

Cutaneous leishmaniasis (CL) is a parasitic disease caused by protozoa of the genus Leishmania transmitted by infected female sandflies.[1] The World Health Organization (WHO) classifies CL among the neglected tropical diseases, currently estimates 600,000 to 1 million new cases occur worldwide annually. The WHO identifies high endemicity in the Middle East, Central Asia, North Africa, and Latin America. Environmental change, population displacement, and urbanization have been associated with rising incidence in several regions.[2] Because it thrives predominantly in resource-limited settings, war zones, and displaced population camps, the World Health Organization (WHO) classifies CL as a high-priority neglected tropical disease. Although CL carries a low mortality rate, its morbidity is profound. The lesions target exposed areas of the body like the face and arms, leaving behind dense, atrophic scars upon healing.[4] In endemic communities, these permanent physical markings carry severe social consequences, frequently subjecting affected individuals particularly women and children to intense social isolation, psychological trauma, and diminished economic opportunities[1-4]

Symptoms and Clinical Presentation

The incubation period of cutaneous leishmaniasis is highly variable, commonly ranging from two weeks to several months following the initial bite of an infected phlebotomine sandfly. The clinical presentation begins as a localized, small, erythematous papule or nodule at the site of inoculation, typically on exposed body regions such as the face, neck, arms, and legs. Over several weeks, this initial lesion undergoes progressive central necrosis, developing into a classic, painless ulcer characterized by a raised, indurated, and well-demarcated border with a sunken center. Depending on the specific Leishmania species and the environmental setting, the ulcer bed can manifest as a "moist" lesion exuding purulent, serous debris or as a "dry" lesion covered by a thick, hyperkeratotic, crusty scab.[5] As the infection develops within the dermal layers, patients frequently exhibit localized lymphadenopathy or sporotrichoid spread, where secondary satellite nodules erupt linearly along regional lymphatic drainage pathways. While uncomplicated lesions often undergo spontaneous involution over many months or even years, the healing process invariably leaves behind permanent, hypopigmented, and atrophic scars that cause significant physical disfigurement. Furthermore, the clinical course can be complicated by secondary bacterial or fungal superinfections of the open wound, which can introduce severe localized pain, accelerate tissue damage, and mask the classic diagnostic features of the primary parasitic infection.[2,6]

The primary symptoms of Cutaneous Leishmaniasis (CL) include:

Skin Bumps: Small red papules or nodules that appear weeks to months after a sandfly bite.

Volcano Ulcers: Bumps that break open into circular sores with raised edges and a sunken center.

Scabs or Crusts: Liquid or dead tissue that dries over the open sore.

Painless Sores: Ulcers that do not hurt or itch unless infected by bacteria.

Swollen Glands: Enlarged lymph nodes near the site of the skin sore.

Atrophic Scars: Permanent, sunken marks.[5]

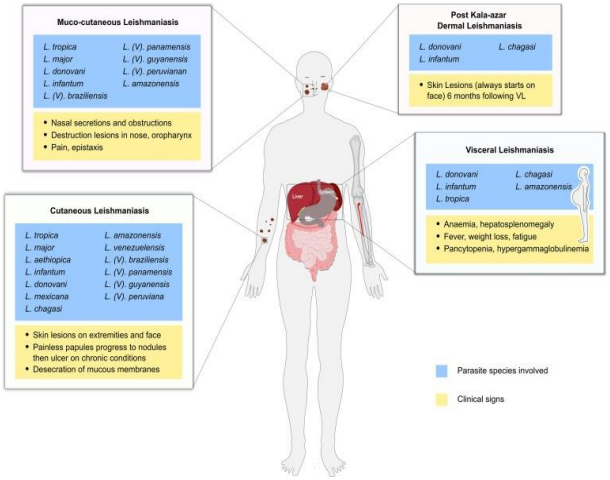


Figure 2: *Symptoms of Leishmania.*[7]

Pathology and Pathogenesis

The pathology of cutaneous leishmaniasis (CL) involves a complex interplay between the protozoan parasite, the sandfly vector, and the host's cell-mediated immune system. The process begins during a blood meal when an infected female phlebotomine sandfly inoculates flagellated, motile metacyclic promastigotes directly into the mammalian host's dermis. Once inside the tissue, these promastigotes release salivary proteins that modulate the local microenvironment, delaying host coagulation and blunting early inflammatory signals to facilitate survival. Local phagocytic cells, primarily dermal macrophages and dendritic cells, quickly engulf the parasites via receptor-mediated endocytosis.[8] Once internalized within the host cell's phagolysosome, the promastigotes adapt to the hostile, acidic environment by transforming into non-motile, oval amastigotes. These amastigotes replicate systematically via binary fission, eventually multiplying to a volume that ruptures the host macrophage and permits the infection of neighboring mononuclear cells. The severity and clinical trajectory of the resulting tissue damage depend heavily on the specific invading Leishmania species interacting with the genetically determined immune response of the host. A protective host response relies on a robust T-helper 1 (Th1) cell-mediated pathway, characterized by the secretion of interleukin-12 (IL-12) and interferon-gamma (IFN-γ). These cytokines activate macrophages to produce nitric oxide and reactive oxygen species, which successfully destroy the intracellular amastigotes and lead to localized, self-healing lesions. Conversely, an unregulated T-helper 2 (Th2) response promotes the release of interleukins like IL-4 and IL-10, which suppress macrophage activation and cause uncontrolled parasite replication, chronic granulomatous inflammation, severe epidermal hyperplasia, and focal dermal necrosis.[4-8]

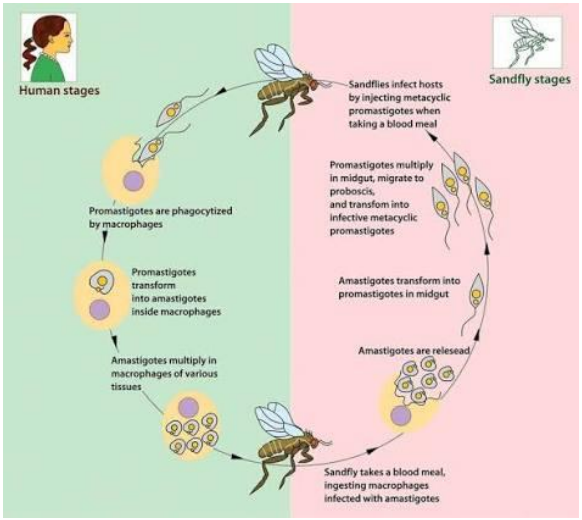


Figure 2: *Lifecycle of Leishmania.*[9]

Treatment

The treatment strategy for cutaneous leishmaniasis (CL) is highly individualized, depending on the number, size, and anatomical location of the lesions, the causative Leishmania species, and the host's clinical status (1). Therapeutic options are broadly categorized into localized treatments for uncomplicated cases and systemic therapies for complex, disseminated, or mucosal infections (1,2). The primary clinical goals are to accelerate lesion healing, minimize disfiguring scar formation, and eradicate intracellular parasites to prevent clinical recurrence or mucosal migration). For localized, uncomplicated CL typically defined as fewer than four small lesions excluding the face, joints, or fingers topical and physical interventions are the first-line standard to minimize systemic toxicity. Local infiltration of pentavalent antimonial, such as sodium stibogluconate or meglumine antimoniate, directly into the ulcer borders is frequently combined with cryotherapy using liquid nitrogen to achieve high cure rates. Additionally, topical formulations containing paromomycin have demonstrated significant efficacy in destroying superficial dermal amastigotes without systemic side effects. Thermotherapy, which delivers controlled localized heat directly to the lesion, is also utilized as a non-invasive physical therapy to disrupt parasite replication cycles.[10]

Prevention and control

- Early diagnosis and effective treatment
- Vector control
- Effective disease surveillance
- Control of animal reservoir hosts
- Mobilization and awareness of community

Conclusion

Cutaneous leishmaniasis remains a highly significant yet neglected public health challenge, causing profound socioeconomic and psychological morbidity across endemic regions globally. While rarely fatal, the chronic, disfiguring cutaneous ulcerations and resulting permanent cicatrization perpetuate cycles of social stigma and economic isolation for vulnerable populations. Controlling this complex vector-borne disease requires moving beyond mono therapeutic strategies toward integrated public health frameworks that harmonize active clinical surveillance, vector suppression, and animal reservoir management. Progress in managing cutaneous leishmaniasis depends on accelerating the development of shorter, less toxic topical and oral therapies to replace traditional antimonial regimens. Furthermore, mitigating the long-term global burden relies heavily on strengthening community health infrastructures, expanding access to early diagnostic platforms like PCR, and sustaining funding for preventative vaccine research. Ultimately, a unified multi-sectoral commitment is essential to alleviate the disease burden and protect populations in resource-limited landscapes.[10,11]

References

1. World Health Organization. Leishmaniasis [Internet]. Geneva: WHO; [cited 2026 Jul 21]. Available from: https://www.who.int/news-room/fact-sheets/detail/leishmaniasis
2. de Vries HJC, Schallig HDFH. Cutaneous leishmaniasis: a 2022 update. Clin Microbiol Rev. 2022;35(4):e0000822. doi:10.1128/cmr.00008-22.
3. Centers for Disease Control and Prevention. Leishmaniasis: Signs and symptoms [Internet]. Atlanta (GA): CDC; [cited 2026 Jul 21]. Available from: https://www.cdc.gov/leishmaniasis/signs-symptoms/index.html
4. DermNet NZ. Leishmaniasis [Internet]. Auckland: DermNet New Zealand Trust; [cited 2026 Jul 21]. Available https://dermnetnz.org/topics/leishmaniasis
5. Cutaneous Leishmaniasis. In: IntechOpen [Internet]. London: IntechOpen; 2023. Chapter 5. Available from: intechopen.com
6. Boudier L, Robert-Gangneux F. Cutaneous leishmaniasis: 2026 guidelines for diagnosis and treatment algorithms. France. PubMed [Internet]. 2026 June 15 [cited 2026 Aug 8]. Available from: nih.gov
7. System biology and engineered therapeutics in cutaneous leishmaniasis ecosystems. Int J Pharm [Internet]. 2026 Aug 1 [cited 2026 Aug 8];127250:

Journal Pre-proof. Available from: sciencedirect.com
8. Karim S, Rahman M. Cutaneous Leishmaniasis: Etiopathogenesis, epidemiology, clinical features, and management strategies. PubMed [Internet]. 2025 Dec 13 [cited 2026 Aug 8];Article ID 41389001. Available from: nih.gov
9. Cutaneous leishmaniasis [Internet]. Geneva: Drugs for Neglected Diseases initiative (DNDi); 2025 Dec 15 [cited 2026 Aug 8]. Available from: dndi.org
10. Leishmaniasis. In: DermNet [Internet]. New Zealand: DermNet New Zealand Trust; 2024 [cited 2026 Aug 8]. Available from: dermnetnz.org
11. Ruiz-Postigo JA, Jain S, Secundino N, Valenzuela JG. Global surveillance and programmatic tracking of cutaneous leishmaniasis burdens: 2024 updates. WHO Weekly Epidemiol Rec [Internet]. 2025 Jan 17 [cited 2026 Aug 8];100(3):13-28. Available from: who.int