Clinical, Cosmetic and Investigational Dermatology

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CASE REPORT

A Presumptive Case of Mucocutaneous and Visceral Leishmaniasis in Nonendemic Country

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Abstract: Leishmaniasis is a complex disease caused by *Leishmania* parasites and transmitted through the bite of infected sandflies, that is classified into cutaneous leishmaniasis (CL), mucocutaneous leishmaniasis (MCL), and visceral leishmaniasis (VL). The increase in international travel has resulted in cases of leishmaniasis emerging in nonendemic regions, often creating challenges in diagnosis. We report a case of presumptive MCL and VL in a 20-year-old female who complained of multiple skin rashes on the face and body, accompanied by erosions on the lips and eyes. A physical examination showed erosions on the eyes, lips, vulva, and "volcanic" noduloulcerative skin lesions covered with adherent crusts on the face, neck, upper and lower extremities. A biopsy of the skin lesion revealed structures resembling *Leishman-Donovan bodies*, then the diagnosis of MCL was established. The patient was given 200 mg/day of itraconazole. On the 12th day of itraconazole treatment, the patient experienced shortness of breath and was hospitalized in the Internal Medicine Department. The laboratory examination showed anemia, thrombocytosis, elevated C-reactive protein, and elevated erythrocyte sedimentation rate levels. Computed tomography of the thorax revealed ground-glass opacity, suggestive of lung inflammation. Bone marrow aspiration showed features of hemophagocytic lymphohistiocytosis, and abdominal ultrasound revealed hepatomegaly. The patient was subsequently diagnosed with VL and was treated with amphotericin B. Unfortunately, the patient died due to respiratory failure. Diagnosing leishmaniasis in nonendemic countries is challenging due to the lack of diagnostic tools to identify the protozoa. Dermatologists must recognize clinical signs, be familiar with supportive examinations, and proficiently treat suspected leishmaniasis.

Keywords: mucocutaneous leishmaniasis, non-endemic country, visceral leishmaniasis

Introduction

Leishmaniasis is a complex disease caused by the parasite *Leishmania*, which spreads through the bite of infected sandflies. This disease is classified by the World Health Organization (WHO) as one of the neglected tropical diseases and typically affects regions with subtropical and tropical climates.[1,2] People in poverty are disproportionately affected by the illness, which is frequently made worse by conditions including malnutrition, population displacement, inadequate housing, limited financial resources, and compromised immune systems.[1,3]

According to data from the WHO in 2022, among the 200 countries, 99 of them reported being endemic for leishmaniasis.[3] Approximately 700,000 to 1 million new cases are reported each year.[1,3] Leishmaniasis remains a significant public health concern in many endemic regions worldwide. In 2023, an estimated 83% of global visceral leishmaniasis (VL) cases were concentrated in seven countries—Brazil, Ethiopia, India, Kenya, Somalia, South Sudan, and Sudan. Likewise, six countries, namely Afghanistan, Algeria, Brazil, Pakistan, Peru, and the Syrian Arab Republic, reported more than 5000 cases of cutaneous leishmaniasis (CL) each, together accounting for 83% of globally reported CL incidence.[3,4] Until 2005,

Received: 19 August 2025
Accepted: 25 October 2025
Published: 3 November 2025

Southeast Asian countries such as Malaysia, the Philippines, Singapore, Thailand, Vietnam, and Indonesia were traditionally recognized as non-endemic for leishmaniasis.[5,6] However, according to WHO data up to November 2024, Thailand is the only Southeast Asian country with endemic leishmaniasis.[3] This may be caused by increased human mobility and globalization, which have expanded the at-risk population for leishmaniasis and simultaneously pose a risk of geographic expansion of *Leishmania* species.[4,7] Presently, leishmaniasis is regarded as an emerging infection in Southeast Asian countries.[5]

The presence of *Leishmania spp*. in Indonesia, both in animals and humans, has never been reported, despite reports of human cases of CL, a differential diagnosis with histoplasmosis.[8] The rise in cases of leishmaniasis occurring in non-endemic regions often poses challenges to diagnosis. A thorough medical history, including travel to endemic areas or exposure to sandfly bites, should be obtained. Although sandfly bites can be painless, leading patients to potentially overlook the inoculation.[4,7,9]

Depending on the species and host immune status, leishmaniasis can be broadly categorized as CL, mucocutaneous leishmaniasis (MCL), and VL.[1,3,9] Clinical manifestations of CL can be present as papules, plaques, ulcers, or nodules.[1,9] The diagnosis represents a challenge because CL can mimic many other diseases, like lupus vulgaris, lepromatous leprosy, syphilis, lymphoma, Kaposi sarcoma, basal cell carcinoma, and squamous cell carcinoma, resulting in incorrect diagnoses and undesirable outcomes.[9,10] The term MCL refers to the involvement of mucosal tissues by *Leishmania spp*., in addition to cutaneous involvement. Generally, MCL appears from days to years following CL.[2,11] VL is a systemic infection, indicating the spread of parasites through the reticuloendothelial system and the visceralization of a primary cutaneous infection. Typical symptoms include fever, pancytopenia, and organomegaly.[1,2,12]

Leishmaniasis can be diagnosed through molecular, microscopic, histopathological, and serological methods, including polymerase chain reaction (PCR), tissue smear or biopsy examination, parasite culture, the Montenegro test, and detection of anti-K39 antibodies.[1,7] Histopathology remains a valuable tool, as the presence of numerous intra- and extracellular amastigotes (*Leishman–Donovan bodies*) within histiocytes is considered a hallmark of the disease; however, PCR generally provides higher sensitivity. This case report presents the first presumptive case of MCL and VL diagnosed at a tertiary referral hospital in West Java, Indonesia, offering important insights into the recognition of this rare infection in a non-endemic region.

Case Presentation

A 20-year-old female was consulted at a tropical dermatology clinic in our hospital with a complaint of multiple skin rashes on the face and body. One year before seeking treatment, the patient initially complained of erosions on the lips and eyes, accompanied by a sore throat. Papules and nodules then appeared on the neck, hands, and feet, which turned into ulcers that felt painful five months before consultation. Following a dermatologist's diagnosis of cicatricial pemphigoid at a nearby hospital, the patient was referred to an allergo-immunology clinic in our hospital. A biopsy was performed from the skin lesion on the right cheek of the patient, and histopathological results showed connective tissue stroma infiltrated by lymphocytic inflammatory cells, histiocytes, polymorphonuclear cells (PMN), plasma cells, and eosinophils. Granuloma formation consisting of multinucleated giant cells was observed. Structures resembling *Leishman-Donovan bodies* were seen among histiocytes and inside multinucleated giant cells (Figure 1). The patient was diagnosed with leishmaniasis and was then referred to the tropical dermatology clinic for further management. There were no symptoms of a stuffy nose, epistaxis, coryza, or hyperemia. Similar complaints in the family, friends, and neighbors were denied. Previously, the patient worked as a textile factory worker in a nonendemic area of leishmaniasis, and the history of contact or insect bites before skin lesions appeared was denied. The patient had never traveled to another country and reported only commuting between home and the workplace in daily life.

The physical examination showed underweight, a normal body temperature, and no signs of nasal perforation. Plaques, nodules, and ulcers with a "volcanic" noduloulcerative morphology covered with adherent crusts were found on the face (Figure 2), neck (Figure 2), upper (Figure 3) and lower extremities (Figure 4). There were also erosions on the eyes, lips (Figure 2), and vulva. Nasal septum crusting, ulceration, and lymph node enlargement were not found. A direct microscopic examination with 10% potassium hydroxide showed no fungal elements. Anti-human immunodeficiency virus (HIV) and syphilis serology tests were nonreactive. Special staining using periodic acid-Schiff (PAS) on histopathological examination

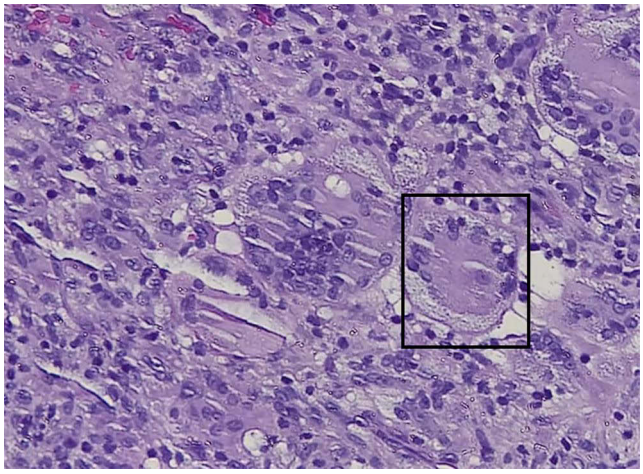

Figure 1 Histopathological findings of ulcerated lesion on the cheek showed structures resembling *Leishman-Donovan* bodies (black rectangle) (H&E, x200).

Figure 2 Noduloulcerative lesions and erosions on the face (**A**) and noduloulcerative lesions on the neck (**B**).

and culture showed no fungal elements. Taken together from clinical manifestations and laboratory results, the patient was then diagnosed with presumptive MCL and given itraconazole 200 mg/day for 14 days.

On day 12 of itraconazole treatment, the patient visited the emergency room due to shortness of breath. Laboratory tests showed anemia, thrombocytosis, elevated C-reactive protein (CRP), and elevated erythrocyte sedimentation rate (ESR) levels. The patient was then hospitalized by the Internal Medicine Department. Computed tomography (CT) of the thorax revealed ground-glass opacities, suggestive of lung inflammation. Bone marrow aspiration showed features of hemophagocytic lymphohistiocytosis (HLH), and abdominal ultrasonography (USG) revealed hepatomegaly. A repeat biopsy was performed from the skin lesion on the neck for microscopic examination, and the result revealed structures resembling amastigotes (Figure 5). Based on the results of laboratory, radiology, and microscopic examinations, the patient was diagnosed with presumptive MCL and VL and was treated with 50 mg per day of intravenous amphotericin B (AmB). However, on day-6 of AmB treatment, the condition of the patient was worsening, and she was transferred to the High Care Unit (HCU). The patient passed away on the second day in HCU due to respiratory failure.

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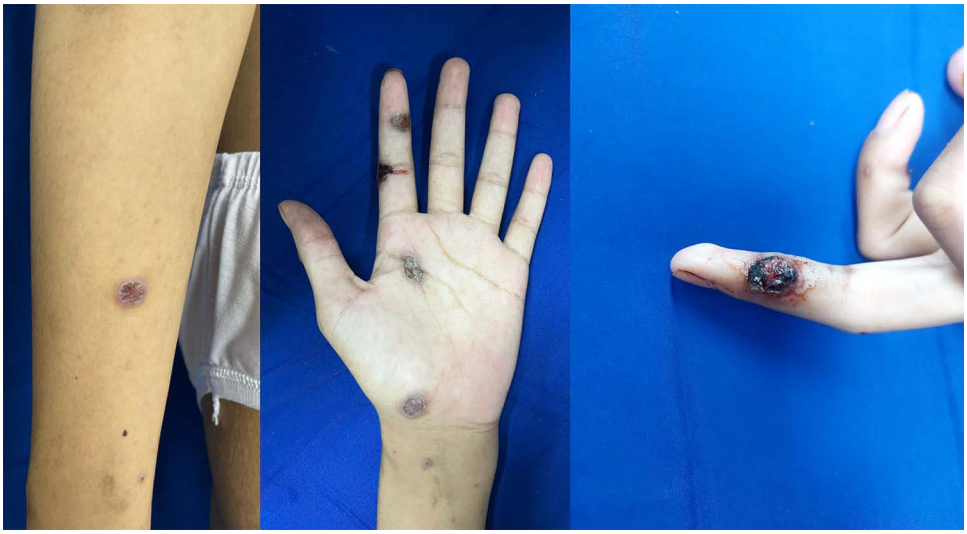

Figure 3 Noduloulcerative lesions on the upper extremities.

Discussion and Conclusion

Over 90% of MCL cases are concentrated in Bolivia, Brazil, Ethiopia, and Peru.[3] The most commonly involved species are *L. braziliensis, L. panamensis, L. amazonensis*, and *L. guyanensis*.[1,9,13] MCL involves the mucous membrane in addition to the skin and may spread via direct extension or hematogenous/lymphatic routes, affecting areas such as the throat, ocular, and genital regions.[1,11,13] MCL can either occur alongside CL or VL, or it can precede them. Typically, MCL manifests from days to years after CL. Skin lesions begin as erythematous papules, which grow into nodules or plaques over the course of a few weeks. They frequently ulcerate and become crusted. The characteristic skin lesions of CL form a "volcanic" noduloulcerative morphology.[1] However, mucosal involvement might also be the initial and detectable pathological condition. Typically, these clinical manifestations are attributed to species within the *L.donovani* complex, most commonly *L. infantum*. Isa et al[14] in 2019 reported 15 MCL cases with localized lesions on the lip, while five patients had skin involvement concurrent with lip involvement. The patient in this case report presents with a "volcanic" noduloulcerative pattern of skin lesions. In addition, mucosal involvement included crusting and erosion on the lips and eyes, as well as erosion in the genital area, which appeared before skin lesions, supporting the diagnosis of MCL.

Visceral leishmaniasis, also known as kala-azar, is the most severe form of the disease and can be life-threatening. Clinically, it presents with fever, splenomegaly, hepatomegaly, lymphadenopathy, pancytopenia, hypergammaglobulinemia, and weight loss.[1,2,12] The primary species involved are *L. donovani* and *L. infantum*. Ninety percent of cases occur in Bangladesh, Ethiopia, India, Nepal, Brazil, and Sudan.[1,3] Thailand is one of the 99 countries that report endemic VL.[3] Hematogenous dissemination of the parasite to the reticuloendothelial system follows skin inoculation. Cough is a possible symptom of VL, and together with the presence of pulmonary rales and dyspnea, it indicates lung involvement in VL. Hepatosplenomegaly is also one of the most important clues for diagnosing VL.[15] In this case report, the patient had coughing and shortness of breath, with a ground glass opacity feature from the thoracic CT scan, and hepatomegaly from the abdominal ultrasound supporting the diagnosis of VL.

Diagnostic methods for leishmaniasis include PCR, smear tissue examination under a microscope, histologic examination with Giemsa or hematoxylin-eosin stain, culture in Novy-McNeal-Nicolle (N-N-N) medium, the Montenegro test, and detection of anti-K39 antibodies and anti-Leishmania immunoglobulin G (IgG) in serum.[7,16,17] The detection of *Leishmania* parasites in bone marrow, lesion aspirate, or other biological samples remains the gold standard for the diagnosis of leishmaniasis.[17]

To identify the amastigotes, there are various smear strategies that have a 50% to 80% effectiveness rate. These samples can be obtained through tissue scrapings or fine-needle aspirates, then air-dried, fixed with methyl alcohol,

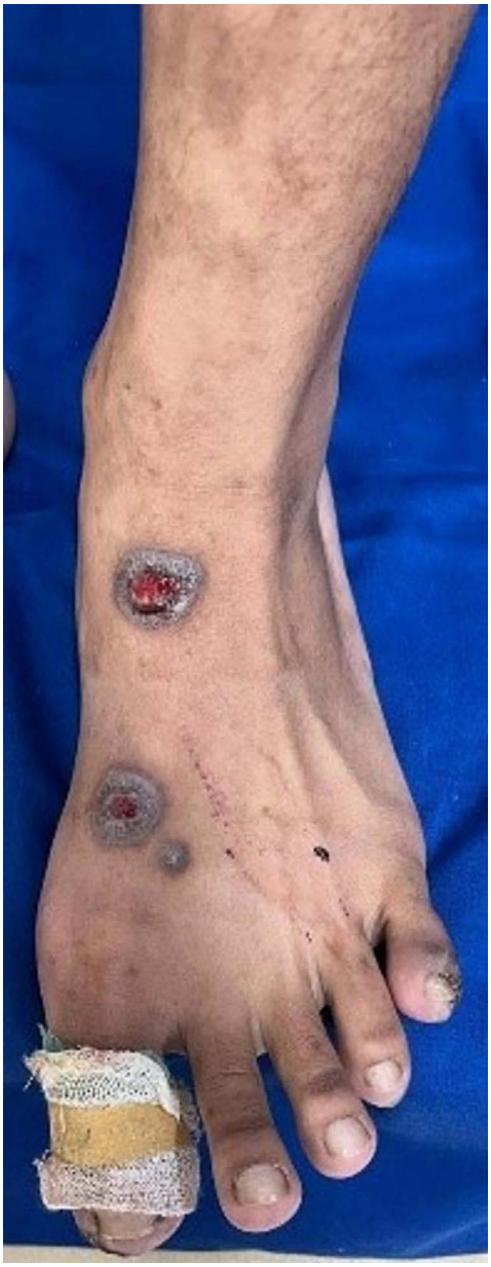

Figure 4 Noduloulcerative lesions on the lower extremities.

stained with Giemsa, and examined under oil-immersion microscopy.[1] A study conducted in Sri Lanka found that *Leishmania* amastigote forms were observed in 22% of samples for Giemsa staining.[18] The skin tissue sample from the patient in this case report was examined under a microscope using Giemsa staining, and the results were suggestive of *Leishmania spp*. amastigotes, supporting the diagnosis of MCL.

Histopathologic analysis is a crucial diagnostic technique. The presence of many extracellular and intracellular amastigotes (also known as *Leishman-Donovan bodies*) among histiocytes is the hallmark of the disease, which has a sensitivity of 50% to 70%.[1,9] A study by Al Hucheimi et al[19] showed that the sensitivity of PCR (92.5%) was significantly higher than that of direct smears (66.7%) and histopathological examination (59.6%). PCR and culture were not performed in this case report due to facility limitations, making it difficult to establish a definitive diagnosis of leishmaniasis in non-endemic countries, such as Indonesia. Structures resembling *Leishman-Donovan bodies* were

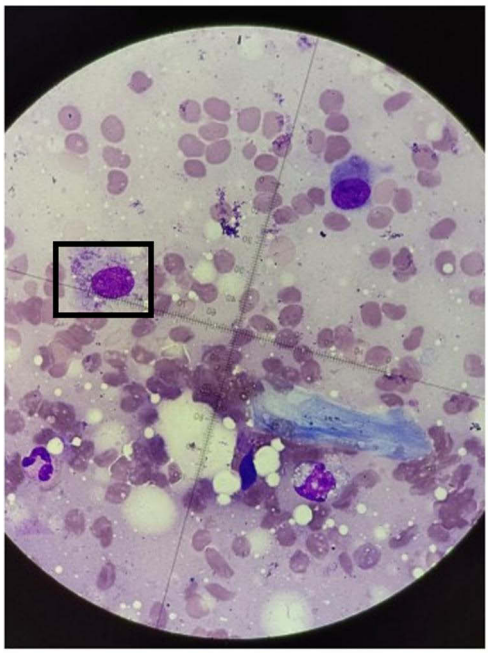

Figure 5 Direct microscopic examination of ulcerated lesion on the neck showed structures resembling amastigotes (black rectangle) (Giemsa, 100x).

revealed in the histopathological examination of this patient, leading to a diagnosis of presumptive MCL. Given that Indonesia is a non-endemic country, the possibility of transmission through local sandfly species cannot be ruled out.

For diagnosing VL, a biopsy from the spleen, bone marrow, or lymph node is required to observe the protozoans under a microscope.[7,13] This technique has high sensitivity, up to 86%, but it is an invasive procedure and does not permit species identification.[12] A tissue biopsy from internal organs was not performed on the patient in this case report.

Hemophagocytic lymphohistiocytosis (HLH) is a rare and potentially fatal complication of VL. It is characterized by excessive inflammation and tissue damage resulting from abnormal immune activation and an overactive inflammatory response. A morphological examination of a bone marrow aspiration can help establish the diagnosis of HLH.[20,21] In this case report, bone marrow aspiration showed features of HLH, which is possibly secondary to VL.

Several treatment options have been suggested for leishmaniasis, and for many years, pentavalent antimonials like sodium stibogluconate and meglumine antimoniate have been regarded as first-line drugs. However, the only Food and Drug Administration (FDA)-approved drugs are intravenous liposomal AmB and oral miltefosine.[9,13] AmB is a medication that fights *Leishmania* species with notable effectiveness. Ergosterol, a substance found in the cellular membranes of fungi and protozoa, binds to AmB to form a transmembrane channel that ultimately results in monovalent ion leakage and cell death.[11] After the emergence of antimonial resistance in India, AmB became the first preferred drug for the cure of VL.[16] WHO recommends a dosage of 2–3 mg/kg/day for 20 days, but successful shorter treatments with a higher dosage (3–5 mg/kg/day) have also been reported.[3] Even though the treatment is effective, many of the patients experience side effects such as fever, hypokalemia, myocarditis, and, most notably, nephrotoxicity, which necessitates hospitalization.[16,17] Since only AmB was available, the patient in this case report was then administered AmB at 50 mg/day. Unfortunately, the patient died due to respiratory failure.

There is a growing interest in anti-leishmanial agents that can be administered orally.[22,23] Azole therapy, which includes fluconazole, ketoconazole, and itraconazole, presents a potential alternative that meets this requirement. Azole antifungal agents exhibit anti-leishmanial properties by inhibiting the role of cytochrome P-450 in the 14α-demethylation of lanosterol in fungi. This inhibition prevents the synthesis of ergosterol, leading to the buildup of 14α-methyl sterols. As a result, the suppression of sterol biosynthesis hampers the growth of *Leishmania*.[22] Based on a study conducted by Amato et al in Brazil, 6 out of 10 MCL patients treated with itraconazole for 3 months showed improvement in skin lesions. In this case report, the patient was given 200 mg/day itraconazole while attending the outpatient clinic. However,

clinical improvement could not be assessed because the patient experienced shortness of breath as a result of lung involvement with VL and was changed to AmB treatment.

In conclusion, establishing a diagnosis of leishmaniasis in non-endemic countries presents its own set of challenges. The unavailability of diagnostic tools such as PCR and culture makes it difficult to identify the protozoa. The lack of experience leads to delayed diagnosis, which in turn results in delayed treatment. Therefore, it is important for dermatologists to recognize the clinical signs, be familiar with supportive examinations, and be proficient in providing therapy for patients suspected of having leishmaniasis.

Abbreviations

CL, Cutaneous leishmaniasis; CT, Computed tomography; FDA, Food and Drug Administration; HLH, Hemophagocytic lymphohistiocytosis; IgG, Immunoglobulin G; MCL, Mucocutaneous leishmaniasis; PAS, Periodic acid-Schiff; PMN, Polymorphonuclear cells; USG, Ultrasonography; VL, Visceral leishmaniasis; WHO, World Health Organization.

Ethics Statement

The ethical approval for this case report was obtained from the Research Ethics Committee of Dr. Hasan Sadikin General Hospital Bandung with the registry number DP.04.03/D.XIV.6.5/418/2024. The family members provided informed consent to participate in the study, in accordance with the Declaration of Helsinki.

Consent for Publication

Since the patient has passed away, written informed consent for the publication of this case report and the accompanying images was obtained from the family members. Approval for the publication of the case details has been obtained from Dr. Hasan Sadikin General Hospital.

Acknowledgments

The authors would like to thank the staff of Dermatology and Venereology Department, Faculty of Medicine, Universitas Padjadjaran – Dr. Hasan Sadikin General Hospital.

Funding

This case report did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Disclosure

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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