AL-KUNOOZE SCIENTIFIC JOURNAL
Vol.11 No. 2 (2025)
ISSN 2706-6231 (ONLINE)
ISSN 2706-6223 (PRINT)

Human Leishmaniasis / a review

Shaimaa A. Alsamir[1*], Esraa AHAl Samir[2], Sundus W Alabdullah[3] , Mohammed A Mahdi[4] , Zainab MuntherAbdulwahhab[5].
Department of Biology[1234], Department of Geology[5],College of Science, University of Basrah, Basrah, Iraq
*Corresponding Author Email: shaimaa.hameed@uobasrah.edu.iq

ORCID ID: https://orcid.org/0000-0002-6528-3676

Received: 5-3-2025 Accepted: 18-3-2025.

Abstract

Leishmania is a flagellate protozoan that causes a chronic illness called leishmaniasis. Most cases of this worldwide illness are found in the Mediterranean region, South America, and some parts of Africa and Asia; There are three primary forms of leishmaniasis: cutaneous, mucocutaneous, and visceral. *Leishmania* diagnosis is made by determining the amastigote stage in clinical specimens by direct microscopic inspection. There are several therapies for cutaneous infections. The use of immune factors of leishmaniasis has helped in how the disease is has been treated in recent years.

Keywords: leishmaniasis, Flagellate protozoa.

Introduction

More than 20 species of *Leishmania* flagellate cause leishmaniasis, zoonotic chronic illness caused by obligate intracellular parasites that spread due to female sandfly bites from the genera *Lutzomyia* and *Phlebotomus*, carryings the infections in humans. *Leishmania donovani* and *Leishmania tropica* are two examples in which humans are the primary reservoirs; the predominant reservoirs are dogs and rodents [1].

The World Health Organization evaluated that there are (700,000 to 1 million) cases of leishmaniasis every year, of which (50,000 to 90,000) are thought to be visceral leishmaniasis [2].

Ninety-five percent of cases of cutaneous leishmaniasis are found in the Middle East, South America, and in the Central Asia or Mediterranean Basin.

In contrast, Visceral leishmaniasis is more common in Brazil, India, and eastern Africa. Over 95% of new Visceral leishmaniasis cases were from China, Brazil, India, Iraq, Kenya, Somalia Sudan, and Nepal in 2018, more than 85% of new cutaneous leishmaniasis cases were from Bolivia, Afghanistan, Brazil, Syria, Colombia, Algeria, Iran, Tunisia, Iraq, Pakistan, and Ethiopia [2].

Ultimately, reports of new cases of mucocutaneous leishmaniasis came from four countries: Ethiopia, Peru, Bolivia, and

Brazil, accounting for approximately 90% of occurrences [2].

Historically, based on its geographical location, leishmaniasis has been considered as either Old World or New World. *Leishmania infantum*, *aethiopica*, *major*, and *tropica* are the main causes of Old World leishmaniasis, which is found in Asia, Africa, and Europe[3].

"Leishmania amazonensis, *Leishmania panamensis, Leishmania Mexicana, Leishmania infantum chagasi or Leishmania braziliensis*" are subspecies *of L. infantum* in the New World that were originally known as *Leishmania chagasi* are the core causes of New World leishmaniasis, which in turn ensues throughout America [4].

L. infantum is the primary pathogenic species in cutaneous leishmaniasis and visceral leishmaniasis, and it is spread by *Phlebotomus arias* and *Phlebotomus perniciosus*, Although, additional reservoirs including hares and rats have been reported, dogs are the primary carrier of *L. infantum* [5].

In addition to its endemic form, leishmaniasis can also affect individuals with weakened immune systems, such as those receiving treatment for tumor necrosis factor (TNF) inhibitors or HIV infection [6].

Additionally, have occurred, including the Madrid pandemic that occurred between 2009 and 2013. Finally, leishmaniasis is becoming more common in wealthy nations because of factors such as globalization, migration, and international travel [3].

Pathogenesis

Infected female sandflies inject *leishmania* promastigotes into human skin; they become amastigotes by the phagocytosis of macrophages, proliferating within the cells and spreading the infection to more mononuclear phagocytized cells [1]. Sandflies pick up the infection by consuming contaminated cells when they are feeding on the blood of their hosts; After entering the sandfly's digestive tract, the amastigotes change into promastigotes [7].

Leishmaniasis has a different incubation periods depending on the clinical manifestation of the illness; however, in general, mucocutaneous leishmaniasis takes longer than two years, Visceral leishmaniasis takes three to nine months, and cutaneous leishmaniasis takes two weeks or less[8].

The host's immunological response and the species involved determine the clinical symptoms of leishmaniasis[1].

The immune response spectrum varies from a robust "T-cell" response that generates humoral response such as interferon (IFN, which raises the levels of antibodies. IFN-activated macrophages eradicate *Leishmania* species, but antibodies are unable to neutralize them. For this reason, humoral responses are incapable of managing impurity, whereas those with a robust immunity require lesions with few parasites. It is an uncontrolled infection that leads to diffuse CL. It should be mentioned that more severe types of illness, including MCL, are linked to an amplified "T helper type1 "response and higher expression of "CD8+ T cells" [1].

Leishmaniasis and body interactions

The bitten body site, which is usually found uncovered, like the face or extremities, cutaneous leishmaniasis begins with the creation of a papule. Usually, a papule grows into a nodule with an ulcerative propensity, For example, after contracting *L. Mexicana*, gum collectors in Central America and Mexico get the typical ulcerated lesion on their ear [9].

Sporotrichosis lesions, lymph node enlargement, and other conditions can result from an infection that spreads via the lymphatic system and causes single or many cutaneous leishmaniasis lesions [10].

In the New World, typical types such as verrucous, lupoid, annular, and eczematous scratches are more prevalent [11]. Within a few months, cutaneous leishmaniasis lesions can heal on their own, leaving a scar [11]. On the other hand, some instances worsen or spread [12].

The growth of papules along the edges of previously healed ulcers is a characteristic of the chronic, recurring types of *L. tropica* infections [13].

Numerous variables, including elevated arginase activity in polymorphonuclear leukocytes, have been associated with chronicity[14].Caused by *L.amazonensis*, *L.aethiopica*, or *L.Mexicana*, , diffuse cutaneous leishmaniasis manifests as many nodules or nonulcerated papules over the majority of the cutaneous infections; The lesions can cause major alterations to the face, giving the appearance of a leonine, similar to that of lepromatous leprosy, They also occur with parasites mucosal lesions[15].Mucosal involvement may develop concurrently with cutaneous contribution a year after cutaneous lesions have been vacant. The lymphatic or circulatory systems may both be used by the illness to spread.

More than 20% of patients may have mucosal involvement in endemic areas; although *L.braziliensis* is the primary cause of mucocutaneous leishmaniasis instances *L. guyanensis* ,*L. amazonensis* , ,and *L. panamensis* are also implicated [16].

The most often impacted sites are the oral and nasal mucosa [17]. Oral cavity lesions have the potential to extend to the larynx and oropharynx, impacting the cartilage and voice cords. The ulcerated cutaneous leishmaniasis lesions might result in deformity. Since the illness might be deadly, treatment is necessary to manage infection [18].

In Visceral Leishmaniasis, macrophages invade the spleen, liver, and bone marrow via the reticuloendothelial system. Weight loss, enlargement of the lymph nodes, hepatosplenomegaly, and fever are among the clinical symptoms [19].

Mostly, Visceral leishmaniasis is caused in immunocompromised people and children by *L. infantum*, and in adults by *L. donovani* and *L. chagasi* [20].

Additionally, skin symptoms may appear in patients. These may be general or specific, such as purpura and hyperpigmentation, nodules, ulcers, and papules. In the Hindu word, kala-azar means Hyperpigmentation, which translates to "black fever", Which is the darkening of the skin that could be seen in 9.88% of Visceral leishmaniasis patients 24, and it has recently been connected to elevated cortisol production [21].

Hyperpigmented regions may be a particular symptom of leishmaniasis because histology reveals amastigotes [5]. Developing post-kala-azar can manifest up to 20 years after therapy and is more prevalent in immunosuppressed patients[22].

However, in HIV-positive individuals, PKDL may occur along with visceral leishmaniasis or occasionally before it[23].

It is typified by,verrucous papules or flesh-coloured nodules that mostly affect the face but can also migrate to other parts of the body, It Occurs primarily in India and East Africa, with few occurrences reported in Spain[24].

Geographical location and host immunological response influence the clinical appearance of 90% cases in Asia, which manifest as macules, but the majority of cases in Africa are papules.

Immunosuppressed individuals may have unusual forms of infection, such as nodules that do not always include the face and a larger quantity of parasites in lesions [24].

Diagnosis

Finding the amastigotes stage of

Leishmania in patient samples by simple microscopic examination or molecular investigation based on kinetoplast or amplification of nuclear DNA, is the best way by which leishmaniasis is diagnosed. Rounded amastigotes range in diameter from 1-4 mm and feature a distinctive kinetoplast, a rod-shaped structure [25].

Due to the low sensitivity of some diagnostic methods, diagnosis requires a mix of procedures. For instance, biopsy specimens may be divided into sections for use in touch imprint cytology, histopathology, and culture [26].

The level of diagnostic sensitivity in vitro for Visceral leishmaniasis varies depending on the kind of tissue infected; for example, spleen tissue can reach up to 90%.It could be diagnosed by aspiration, yet there is a significant risk of intra-abdominal hemorrhage [27].

Bone marrow infections have been reported to range between 50% and 85%; even lower rates have been observed for peripheral blood and lymph nodes [28].

The kind of lesion studied determines the diagnostic sensitivity in infection; rates of up to 100% for nodular lesions contrast with relatively low rates for macular lesions, where more sensitive molecular approaches are required [29].

Histology can reveal particular abnormalities, including the presence of amastigotes stage in the macrophages of the skin, seen in 50-70%of cases, as well as nonspecific signs, like, pseudoepitheliomatous hyperplasia, ulceration, and a mixed inflammatory infiltrate [28].

Giant cells proliferate and the number of parasites decreases with the development of lesions; additional observations also have suggested tuberculoid granulomas, dermal fibrosis, and a high concentration of plasma cells in advanced stages [30].

Histologic patterns have been identified during leishmaniasis infection, usually including the occurrence of a large number of amastigote stages, a mixture of neutrophils, macrophages ,and plasma cells together with necrosis ; 3- early granulomas containing lymphocytes, epithelioid cells ,and plasma cells ; and 4- fully developed granulomas containing Langhans-type giant cells, also diffuse cutaneous leishmaniasis is characterized by a diffuse macrophage infiltration that contains a high number of amastigotes [30].

Another helpful diagnostic tool is dermoscopy[31]. Vascular structures Erythema, hairpin, comprising polymorphous, and arborizing vessels; crusts and erosion/ulceration are the most often reported dermoscopic features, Furthermore, teardrop shapes with white-yellow coloured and the pattern with white starburst were less prevalent but more distinctive features [32].

Other tests, such as serological and Montenegro skin test, are less helpful in cutaneous leishmaniasis diagnosis, in which Leishmanin is injected intradermally in the former, and the findings are evaluated and read similarly to the tuberculin test, It cannot be distinguished between an ongoing or former infection and is negative in diffuse cutaneous leishmaniasis, active Visceral leishmaniasis[33].The Western blot analysis, enzyme-linked immunoassay(ELISA), Immunofluorescence, and direct agglutination test are examples of effective serological assays used for Visceral leishmaniasis [34].

Antibody titers, however, do not distinguish between ongoing and prior infections and these titers decrease very slowly following treatment. Also , cross-reactivity with other antibodies (such as those that cause Chagas disease)can potentially impact on results[33].

Moreover, antibodies are frequently present in asymptomatic people in endemic

locations;therefore data must be carefully interpreted in light of the clinical setting.

ELISA has shown that cutaneous leishmaniasis brought on by *L. major* and *L.tropica* is associated with anti-galactosyl antibodies with high levels , These have the same restrictions as conventional serological tests, but they have a high sensitivity for visceral leishmaniasis(Boelaert *et al*., 2014) [35].

ELISA and the latex agglutination test using samples of urine in visceral leishmaniasis and an immunochromatographic strip that detects for peroxidoxin antigen in cutaneous leishmaniasis are examples of more modern antigen detection assays [36].

Treatment

Many leishmaniasis cases resolve on their own in less than two years; the species involved has a major influence on the variations in resolution times, with infections produced by *L. braziliensis* and *L. panamensis* having the highest propensity to persist. [37].

Cutaneous leishmaniasis can be categorized as simple or complicated based on this and other variables such anatomic location, infection intensity, and the host's immunological response, While complicated infections need systemic medications, simple infections can be treated conservatively or with local remedies [38].

Summarizes the characteristics of both basic and complicated cutaneous leishmaniasis has several therapies available, yet there is little evidence to support the alternatives for cutaneous leishmaniasis [39].

For patients who satisfy the requirements for uncomplicated cutaneous leishmaniasis, a watch-and-wait strategy can be used after balancing the risks and benefits, Lesions that do not heal on their own, when a quicker recovery is desired, or when there is a desire to minimize scarring, local therapy may be employed, Cryotherapy and intralesional pentavalent antimonial are the most often used local therapies[38], When both are used together, better results are obtained[39]. Additionally, topical paromomycin is utilized to treat cutaneous leishmaniasis, especially in the New World

There has been information of positive outcomes from several local therapies, including imiquimod; photodynamic therapy; and carbon dioxide laser therapy [40], which has demonstrated success rates of 93% and relatively minor side effects (hyperpigmentation, prolonged erythema, and hypertrophic scarring).

Patients who satisfy any of the requirements for cutaneous leishmaniasis are advised to get systemic treatments, Although liposomal amphotericin B is very effective, nephrotoxicity is a possibility [41].

Systemic pentavalent antibiotics have been used for treating complicated cutaneous leishmaniasis and mucocutaneous leishmaniasis for a long time, yet this use has limited the development of resistance in some locations [42].Azole compounds, miltefosine and pentamidine are more choices [43].

A three-month clinical investigation comparing once-daily doxycycline 200 mg with twice-daily chloroquine 250 mg has been showed effectiveness [44].

For individuals with cutaneous leishmaniasis caused by *L.braziliensis*, yearly examinations are advised in order to facilitate the early identification of mucocutaneous leishmaniasis progression[39].However, amphotericin B or miltefosine systemic therapy is typically required in India [2].

Prevention and Control

There are currently no human leishmaniasis preventive vaccinations available. The majority of leishmaniasis patients who recover do not re-infect

themselves, which makes vaccine research easier. Aside from vector control, early diagnosis and treatment are important control measures for cutaneous leishmaniasis and visceral leishmaniasis because infected humans serve as reservoirs [45].

Conclusions

The prevalence of cutaneous leishmaniasis has increased as a result of migration and foreign travel.

However, low indices of distrust between physicians, low sensitivity of some diagnostic tests, restricted access to molecular testing; Unfavourable treatment outcomes, and limited therapy alternatives might make care challenging. Atypical manifestations and the necessity for systemic therapy are linked to HIV coinfection and TNF inhibitor usage.

Conflicts of Interest: The authors declare that they have no conflicts of interest.

References

1. Scott P, Novais FO."Cutaneous leishmaniasis: immune responses in protection and pathogenesis".Nat Rev Immunol.2016;16:581-592.
2. World Health Organization ."media center factsheets". 2020; http://www.who.int.
3. Boggild AK, Caumes E, Grobusch MP, Schwartz E, Hynes NA, Libman M, *et al.* "Cutaneous and mucocutaneous leishmaniasis in travellers and migrants : a 20-year GeoSentinel Surveillance Network analysis".J Travel Med.2019;25:26-55
4. Lupi O, Bartlett BL, Haugen RN, Dy LC, Sethi A, Klaus SN, *et al.* "Tropical diseases caused by protozoa".J Am Acad Dermatol.2009;60:897-928.
5. Abadeas-Granado I, Navarro-Bielsa A, Ferrando-Lamana L."Hyperpigmentation as a guiding sign for the diagnosis of visceral leishmaniasis in a patient with human immunodeficiency virus (HIV)".Int J Dermatol. 2021;https://doi.org/10.1111/ijd.15429.
6. Bosch-Nicolau P, Ubals M, Salvador F, Sunchez-Montalv A, Aparicio G, Erra A, *et al.*"Leishmaniasis and tumor necrosis factor alpha antagonists in the Mediterranean basin switch in clinical expression".PLoS Negl Trop Dis.2019;13: e0007708
7. Reithinger R, Dujardin JC, Louzir H, Pirmez C, Alexander B, Brooker S."Cutaneous leishmaniasis".Lancet Infect Dis.2007;7:581-596.
8. Wirth DF, Rogers WO, Barker R Jr, Dourado H, Suesebang L, Albuquerque B."Leishmaniasis and malaria: new tools for epidemiologic analysis".Science 1989 ;234:975-987.
9. Quijano-Pitman F."Identification de la Leishmania tropic a Mexicana como agente etiologico de la Ulcera de los Chicleros".Gac Med Mex.2000;135:331.
10. Carvalho LMV, Pimentel MIF, Conceic¸o-Silva F, Vasconcellos CFE, Valete-Rosalino CM, Lyra MR, *et al.*"Sporotrichoid leishmaniasis: a cross-sectional clinical, epidemiological and laboratory study in Rio de Janeiro State, Brazil". Rev Inst Med Trop Sao Paulo. 2017;59:e33.
11. Meireles CB, Maia LC, Soares GC, Teodoro IPP, Gadelha MDSV, da Silva CGL, *et al.*" Atypical presentations of cutaneous leishmaniasis: A systematic

review".Acta Trop.2017;172:240-254.
12. Colmenares M, Kar S, Goldsmith-Pestana K, McMahon-Pratt D."Mechanisms of pathogenesis: differences amongst Leishmania species".Trans R Soc Trop Med Hyg. 2002;96:3-7.
13. Gitari JW, Nzou SM, Wamunyokoli F, Kinyeru E, Fujii Y, Kaneko S, *et al*."Leishmaniasis recidivans by *Leishmania tropica* in Central Rift Valley Region in Kenya".Int J Infect Dis.2018;74:109-116.
14. Mortazavi H, Sadeghipour P, Taslimi Y, Habibzadeh S, Zali F, Zahedifard F, *et al.* "Comparing acute and chronic human cutaneous leishmaniasis caused by *Leishmania major* and *Leishmania tropica* focusing on arginase activity".J Eur Acad Dermatol Venereol.2016;30:211-218.
15. Mariz BALA, Sinchez-Romero C, Alvarado NAP, Campos E MM, Almeida OP, Martinez-Pedraza R."Diffuse cutaneous leishmaniasis with oral involvement in a patient of Northern Mexico".Trop Doct.2019;49:303-306.
16. David C , Dimier-David L, Vargas F, Torrez M, Dedet JP."Fifteen years of cutaneous and mucocutaneous leishmaniasis in Bolivia: a retrospective study".Trans R Soc Trop Med Hyg.1993;87:7-9.
17. Salotra P, Sreenivas G, Beena KR, Mukherjee A, Ramesh V."Parasite detection in patients with post kala-azar dermal leishmaniasis in India: a comparison between molecular and immunological methods".J Clin Pathol.;56, 2003:840-853.
18. Marra F, Chiappetta MC, Vincenti V."Ear, nose and throat manifestations of mucocutaneous Leishmaniasis: a literature review".Acta Biomed. 2014;85:3-7.
19. Zijlstra EE, Musa AM, Khalil EA, el-Hassan IM, el-Hassan AM."Post-kala-azar dermal leishmaniasis".Lancet Infect Dis.2003;3:87-98
20. Burza S, Croft SL, Boelaert M."Leishmaniasis".Lancet.2018;392: 951-970.
21. ELkhair EB."Elevated cortisol level due to visceral leishmaniasis and skin hyper-pigmentation are causally related".Int J Sci Commer Humanit. 2014;2:86-92.
22. Stark D, Pett S, Marriott D, Harkness J."Post-kala-azar dermal leishmaniasis due to *Leishmania infantum* in a human immunodeficiency virus type 1-infected patient".J Clin Microbiol.2006;44:1178---1180.
23. Zijlstra EE."Biomarkers in Post-kala-azar Dermal Leishmaniasis".Front Cell Infect Microbiol.2019;9:228.
24. de Juan Martin F, Justa Roldin ML, Siez de Adana Pérez E, Navarro Serrano E, Bouthelier Moreno M, Gilaberte Calzada Y."Leishmaniasis dérmica postkala-azar “Postkala-azar dermal leishmaniasis".An Esp Pediatr.1997;46:63-64.
25. Alvar J, Aparicio P, Aseffa A, Den Boer M, Canavate C, Dedet JP, *et al*."The relationship between leishmaniasis and AIDS: the second 10 years".Clin Microbiol Rev.2008;21:334-359.
26. Mock DJ, Hollenbaugh JA, Daddacha W, Overstreet MG, Lazarski CA, Fowell DJ." Leishmania induces survival, proliferation and elevated cellular dNTP levels in human monocytes promoting acceleration of

HIV co-infection".PLoS Pathog.2012;8:e1002635.
27. Monge-Maillo B, Norman FF, Cruz I, Alvar J, Lopez-Vélez R."Visceral leishmaniasis and HIV coinfection in the Mediterranean region".PLoS Negl Trop Dis.2014;8:e3021.
28. Zijlstra EE."PKDL and other dermal lesions in HIV co-infected patients with leishmaniasis: review of clinical presentation in relation to immune responses".PLoS Negl Trop Dis.2014;8:e3258.
29. Aronson N, Herwaldt BL, Libman M, Pearson R, Lopez-Velez R, Weina P. "Diagnosis and Treatment of Leishmaniasis:Clinical Practice Guidelines by the Infectious Diseases Society of America (IDSA) and the American Society of Tropical Medicine and Hygiene (ASTMH)".Am J Trop Med Hyg.2017;96:24-45.
30. Mehregan DR, Mehregan DA , Mehregan AH."Histopathology of cutaneous leishmaniasis".Gulf J Dermatol Venereol.1997;4:1-9.
31. Llambrich A, Zaballos P, Terrasa F, Torne I, Puig S, Malvehy J."Dermoscopy of cutaneous leishmaniasis".Br J Dermatol.2009;160:756-761.
32. Ayhan E, Ucmak D, Baykara SN, Akkurt ZM, Arica M."Clinical and dermoscopic evaluation of cutaneous leishmaniasis".Int J Dermatol.2015;54:193-201.
33. Momeni Boroujeni A, Aminjavaheri M, Moshtaghian B, Momeni A, Momeni AZ."Reevaluating leishmanin skin test as a marker for immunity against cutaneous leishmaniasis".Int J Dermatol.2013;52:827-830.
34. Cota GF, de Sousa MR, Demarqui FN, Rabello A."The diagnostic accuracy of serologic and molecular methods for detecting visceral leishmaniasis in HIV infected patients: meta-analysis".PLoS Negl Trop Dis.2012;6:e1665.
35. Boelaert M, Verdonck K, Menten J, Sunyoto T, van Griens-ven J, Chappuis F."Rapid tests for the diagnosis of visceral leishmaniasis in patients with suspected disease". Cochrane Database Syst Rev.2014;6.CD009135.
36. Ghatei MA, Hatam GR, Hossini MH, Sarkari B."Performance of latex agglutination test in diagnosis of visceral leishmaniasis in Iran".Iran J Immunol.2009;6:202-207.
37. Cota GF, de Sousa MR, Fereguetti TO, Saleme PS, Alvarisa TK, Rabello A."The cure rate after placebo or no therapy in American cutaneous leishmaniasis: a systematic review and meta-analysis".PLoS One.2016;11:e0149697.
38. Aronson N ,Wortmann GW, Byrne WR, Howard RS, Bernstein WB, Marovich MA."A randomized controlled trial of local heat therapy versus intravenous sodium stibogluconate for the treatment of cutaneous *Leishmania major* infection".PLoS Negl Trop Dis.2010;4:e628.
39. Morton CA, Szeimies R-M, Basset-Séguin N, Calzavara-Pinton PG, Gilaberte Y, Haedersdal M."European Dermatology Forum guidelines on topical photodynamic therapy. 2019; Part 2:emerging indications field cancerization, photorejuvenation and inflammatory/infective dermatoses". J Eur Acad Dermatol Venereol.;2020;34:17---29.
40. Shamsi Meymandi S, Zandi S, Aghaie H, Heshmatkhah A."Efficacy

of CO(2) laser for treatment of anthroponotic cutaneous leishmaniasis, compared with combination of cryotherapy and intralesional meglumine antimoniate".J Eur Acad Dermatol Venereol .2011;25:587-591.

41. Solomon M, Baum S, Barzilai A, Scope A, Trau H, Schwartz E."Liposomal amphotericin B in comparison to sodium stibogluconate for cutaneous infection due to *Leishmania braziliensis".* J Am Acad Dermatol. 2007;56:612-615.
42. Arevalo J, Ramirez L, Adaui V, Zimic M, Tulliano G, Miranda-Veristegui C."Influence of *Leishmania (Viannia)* species on the response to antimonial treatment in patients with American tegumentary leishmaniasis".J Infect Dis. 2007;195:1846-51.
43. Neves LO, Talhari AC, Gadelha EPN, Silva Jonior RM, Guerra JA, Ferreira LC."A randomized clinical trial comparing meglumine antimoniate, pentamidine and amphotericin B for the treatment of cutaneous leishmaniasis by *Leishmania Guyanensis".*An Bras Dermatol.2011;86:1092-1097.
44. Malik F, Hanif MM, Mustafa G."Comparing the Efficacy of Oral Chloroquine versus Oral Tetracycline in the Treatment of Cutaneous Leishmaniasis".J Coll Physicians Surg Pak.2019;29:403-405.
45. Alvar J, Croft SL, Kaye P, Khamesipour A, Sundar S, Reed SG."Case study for a vaccine against leishmaniasis".Vaccine, 2013:244-29.

داء اللشمانيات البشري

شيماء عبد الرزاق السامر[1]، اسراء عبد الرزاق السامر[2]، سندس وليد العبد الله[3] ، محمد عباس مهدي[4] ، زينب منذر عبد الوهاب[5]
قسم علوم الحياة [1234] ، قسم علم الأرض [5] ، كلية العلوم ، جامعة البصرة ، البصرة ، العراق

الخلاصة:

اللشمانيا من الاوالي المسوطة التي تسبب مرضًا مزمنًا يسمى داء الليشمانيات. توجد معظم حالات هذا المرض العالمي في منطقة البحر الأبيض المتوسط ،أمريكا الجنوبية ، بعض أجزاء أفريقيا وآسيا ؛ هناك ثلاثة اشكال من داء الليشمانيات: الجلدي ، المخاطي ، والحشوي. تشخيص اللشمانيا يتم عن طريق تمييز طور المشيقة في العينات السريرية عن طريق الفحص المجهري المباشر. هناك العديد من العلاجات للاصابات الجلدية. استخدام العوامل المناعية اسهم في علاج المرض في السنوات الأخيرة.