

Cutaneous leishmaniasis: A case report

M. Marovt, R. Kokol, A. Stanimirović, and J. Miljković

A B S T R A C T

Cutaneous leishmaniasis is the most common form of leishmaniasis caused by flagellate protozoa of the genus *Leishmania* transmitted by sand fly bites. Old World leishmaniasis is endemic in the Mediterranean Sea and the neighbouring countries. We present a case of a 10-year-old boy with a cutaneous leishmaniasis in form of papules on the nose, right and left lower leg. Histopathological examination showed diffuse dermal infiltrate predominantly of macrophages with admixture of few lymphocytes, eosinophils and plasma cells. In most of macrophages amastigotes were seen. Because of higher rate of travel and work abroad increased number of sporadic cases of cutaneous leishmaniasis in non-endemic areas should be taken into account.

Case report

A 10-year-old boy presented with approximately 3 month history of papules on the nose, right and left lower leg (Figures 1–3). The boy was staying in southern Croatia a few months before. Physical examination showed solitary papules on erythematous surface with central umbilication. Some of papules were already in regression. Local antibiotic ointments were unsuccessful. On first control examination lesions on the nose and left lower leg became erosive, lesions of the right lower leg were unchanged. Because of unclear clinical appearance biopsy was performed. Histopathological examination showed diffuse dermal infiltrate predominantly of macrophages with admixture of few lymphocytes, eosinophils and plasma cells. In most of

macrophages amastigotes were seen. On their surface and occasionally extracellularly rod-shaped kinetoplasts were noticeable (Fig.4). With negative indirect hemagglutination assay (IHA) systemic involvement could be ruled out. The boy was put on 5% chloramphenicol 10% sulfur in miconazole ointment. After 2 months only residual hyperpigmentation was noticed. No new lesions appeared on further examinations.

Discussion

The most common form of leishmaniasis is cutaneous leishmaniasis. Cutaneous leishmaniasis is mainly caused by *Leishmania (L.) major*, *L. infantum*, and *L. tropica* (1). Leishmaniasis is endemic in 88 countries, 67 of them in the Old World and 21 in the New World

K E Y W O R D S

cutaneous
leishmaniasis,
non-endemic
areas



Figures 1–3. Solitary papules on erythematous surface with central umbilication on the nose, right leg, and lower left leg.

(2). Old World leishmaniasis is endemic in the Mediterranean Sea and the neighbouring countries. The annual incidence is worldwide about 400,000 cases with a prevalence of approximately 350 million people infected (3). More than 90% of cutaneous leishmaniasis worldwide can be found in Afghanistan, Iran, Saudi Arabia, Syria, Brazil and Peru. Majority of cases of cu-

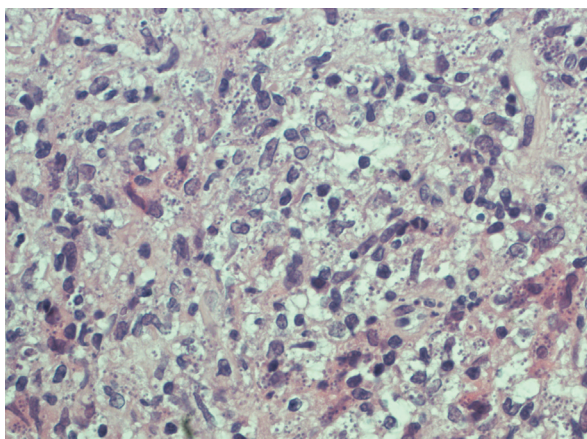


Figure 4. Diffuse dermal infiltrate predominantly of macrophages in most of them are amastigotes and occasionally extracellular rod-shaped kinetoplasts.

taneous leishmaniasis is found in adult men between 20 and 40 years (3). Tourists and workers from the endemic areas have an increased risk of cutaneous leishmaniasis.

The most widely used disease categorization is based on clinical and geographic appearance. The three clinical forms of leishmaniasis are divided in cutaneous, mucocutaneous, and visceral disease. Cutaneous leishmaniasis is divided based on its clinical appearance into localised, diffuse, recurrent, and post-kala azar dermal leishmaniasis. Old World leishmaniasis is caused by *Leishmania* species found in Africa, Asia, the Middle East, the Mediterranean, and India, clinically presenting in cutaneous or visceral disease. New World leishmaniasis is caused by *Leishmania* species found in Central and South America clinically presenting in cutaneous, mucocutaneous, and visceral disease.

Following the bite, localized cutaneous leishmaniasis (LCL) typically develops from an inflammatory papule to an ulcer. The incubation period is highly variable. The lesions are usually in non covered regions of the body, mainly the face, neck or arms.

The diagnosis of leishmaniasis is confirmed by detection of parasites in the smear-specimen of cutaneous lesions, the lymph node, bone marrow, spleen or of the spinal-fluid. Histopathological findings are granulomas consisting of lymphocytes, histiocytes and plasma cells with epitheloid cells (4). Frequently *Leishmania* can be found within the histiocytes and cultivated in Nicolle-Novy-Mac Neal- (NNM-) medium for up to 40% of cases (3, 5). *Leishmania* can also be detected by serological tests (indirect immunofluorescence and enzyme-linked immunosorbant assay). The highest sensitivity and specificity can be reached by polymerase chain reaction using the isolated DNA from

the lesion and validated genus-specific PCR primers (6). No vaccine is available yet for the prevention of leishmaniasis.

At one extreme, LCL shows a vigorous immune response, with most cases resolving without intervention. This form of disease exhibits a helper T-cell subtype 1 immune response, with antibody-mediated uptake of *L. major* by dendritic cells and interleukin 2, interferon-gamma, and interleukin 12 as the prominent cytokines that induce disease resolution (7). At the other extreme, with visceral or diffuse cutaneous disease, patients exhibit relative anergy to the *Leishmania* organism and have a prominent helper T-cell subtype 2 cytokine profile.

The differential diagnosis of LCL is extensive and includes impetigo, pyoderma gangrenosum, deep fungal infection, mycobacterial infection, sarcoidosis, and squamous cell carcinoma (8, 9).

Previously treatment was recommended for every case of CL. This is no longer the conventional practice. Many *Leishmania* species or subspecies of the *Leishmania* protozoa have different virulence and clinical predilections, so the treatment must be tai-

lored to every individual. Old World disease tends to be self-limiting. Leishmaniasis caused by this species does not necessarily need to be treated unless the lesion is in a cosmetically or functionally sensitive site. In New World leishmaniasis treatment is very often the standard of care because of higher recurrence range of chronic ulcers, recidivant lesions, or mucocutaneous involvement. Multiple treatment options are used throughout the world for cutaneous disease. Beside to oral and parenteral medications (pentavalent antimony, liposomal amphotericin B and some other), local therapy options for cutaneous leishmaniasis include (1) cryotherapy, (2) infiltration of sodium stibogluconate, (3) local heat therapy, and (4) various topical paromomycin preparations.

Conclusion

Cutaneous leishmaniasis is a rare disease in Slovenia, last cases were reported in 1996 (10). Because of higher rate of travel and work abroad increased number of sporadic cases of CL in non-endemic areas should be taken into account.

REFERENCES

1. Stanimirovic A, Stipic T, Skerlev M, et al. Treatment of cutaneous leishmaniasis with 20% paromomycin ointment. *J Eur Acad Dermatol Venereol.* 1999;13:214-7.
2. Blum J, Desjeux P, Schwartz E, et al. Treatment of cutaneous leishmaniasis among travellers. *J Antimicrob Chemother.* 2004;53:158-66.
3. Hengge UR, Marini A. Cutaneous leishmaniasis. *Hautarzt.* 2008;59:627-32.
4. Saleem K, Ayaz B, Shaikh A. Histological grading patterns in patients of cutaneous leishmaniasis. *J Coll Physicians Surg Pak.* 2007;17:650-3.
5. Mashhood AA, Khan IM, Nasir S, et al. Fine needle aspiration cytology versus histopathology in the diagnosis of cutaneous leishmaniasis in Pakistan. *J Coll Physicians Surg Pak.* 2005;15:71-3.
6. Bhutto AM, Soomro FR, Baloch JH, et al. Cutaneous leishmaniasis caused by *Leishmania (L.) major* infection in Sindh province, Pakistan. *Acta Trop.* 2009;111(3):295-8.
7. Von Stebut E. Immunology of cutaneous leishmaniasis: the role of mast cells, phagocytes and dendritic cells for protective immunity. *Eur J Dermatol.* 2007;17:115-22.
8. Schwarz KJ. Diagnosis and differential diagnosis of cutaneous leishmaniasis. Report on seven cases observed in Zurich. *Schweiz Med Wochenschr.* 1970;100:2073-8.
9. Herrmann A, Wohlrab J, Sudeck H, et al. Chronic lupoid leishmaniasis. A rare differential diagnosis in Germany for erythematous infiltrative facial plaques. *Hautarzt.* 2007;58:256-60.
10. Marolt-Gomiscek M, Radsel-Medvescek A. Infekcijske bolezni. Ljubljana: Tangram, 2002:497-504.

AUTHORS' ADDRESSES

Maruška Marovt, MD, Department of Dermatology, University Medical Centre, Maribor, Ljubljanska 5, Maribor, Slovenia, corresponding author
Rok Kokol, MD, same address
Andrija Stanimirović, PhD, School of Health Studies, University of Zagreb, Croatia
Jovan Miljković, PhD, same address