

CUTANEOUS LEISHMANIASIS IN TRIPOLI

(A study of 36 cases)

M L Khatri, M Shafi and M Mosadiq

The present report is an analysis of thirty six cases of cutaneous leishmaniasis seen in Tripoli over a period of two years, with special emphasis on clinical features and epidemiology. Factors likely to be responsible for the increased incidence of the disease are discussed.

Key words : Cutaneous leishmaniasis, Tripoli.

Cutaneous leishmaniasis caused by a protozoan, *Leishmania tropica*, occurs in a variety of clinical types. The commonest type is acute cutaneous leishmaniasis, in which a nodular lesion appears on a site exposed to the bite of the sandfly. It generally enlarges to 1-3 centimeters in size. In the dry type, the nodule remains unchanged until healing takes place in 6 to 18 months with scar formation. In the wet type, the lesion soon ulcerates before it ultimately heals with a scar. In chronic leishmaniasis, nodular lesions appear at the edge of the healed areas and the process continues for many years, while in disseminated anergic leishmaniasis, there are generalised 1-2 centimeter nodular lesions which appear in the patients having poor immunological resistance to the parasites. The late acute leishmaniasis is characterised by development of lesions due to reinfection, many years after healing of the initial disease.

Two different sub-species of *Leishmania tropica*¹ have been postulated—*Leishmania tropica* var major, causing cutaneous leishmaniasis wet type and *Leishmania tropica* var minor causing cutaneous leishmaniasis dry type. The parasite is transmitted to the human and mammalian hosts by the sandfly, mostly *Phlebotomus papatasi*. The reservoir host varies according to the geographical regions.² In the Mediterranean areas it is the dog, in the Middle East, it is man in urban areas and rodents in the rural

areas. In Africa and Asia it is a wild rodent especially gerbil. The appearance of the parasite in the human host is in the amastigote form (*Leishman Donovan body*). In the sandfly, or when it is in culture, it is seen in the flagellated or promastigote (leptomonad) form. The type of the disease caused by *Leishmania* depends upon the type or strain of *Leishmania*³, the immunologic response, the general state of nutrition and health of the host.^{4,5}

Cutaneous leishmaniasis is endemic all around the Mediterranean coast, particularly North Africa, in Asia Minor, Asia including China and Southern Russia and Central and South America.⁶ Formal epidemiological study has not yet been done in Libya. Bhaktaviziam⁷ has seen 23 cases of this disease during 1978-79 at the Central Hospital, Tripoli (Libya). In the past 2 years, there has been a constant increase in the incidence and nearly 60 cases were seen during this period. The present study is an analysis of 36 out of these cases.

Materials and Methods

Thirty six cases of cutaneous leishmaniasis were studied in detail from September, 1981 to December, 1983. As far as possible, the cases were admitted in the hospital for an initial assessment and treatment. Details of the history, physical findings, laboratory data and follow-up were recorded in a special proforma for this purpose. The criteria for the diagnosis of cutaneous leishmaniasis were based on geographical history, clinical findings, tissue smear and histopathological findings.

From the Department of Dermatology, Central Hospital and Faculty of Medicine, Al Fateh University, Tripoli, Libya (SPLAJ).

Address correspondence to : Dr. M. L. Khatri.

Results

Majority of the patients were in the age group of 11-50 years. The youngest was one year old while the eldest was 72 years. Of the 36 cases studied, 25 (69%) were males and 11 (31%) were females (Table I).

Table I. Age and sex incidence of the patients.

Age group in years	Number of patients			Percentage
	Male	Female	Total	
0-5	1	1	2	5.5%
6-10	1	1	2	5.5%
11-20	6	-	6	16.7%
21-30	4	1	5	13.9%
31-40	7	1	8	22.2%
41-50	5	3	8	22.2%
51-60	1	3	4	11.1%
61 onwards	-	1	1	2.8%
Total	25	11	36	

Twenty five patients were Libyan nationals, 3 were Tunicians, 2 Algerians, 4 Turks and 2 Philipinos.

Eleven patients were resident of Tripoli, five of them had visited Yefren, one to one and a half month before the time of appearance of the lesions. The other six patients had visited Garyan, 4 of them frequently while 2 patients had visited one month before the appearance of the lesions. From this, we can infer that the incubation period in these cases was approximately one month. Rest of the 25 patients had not visited any other place away from their place of residence within 3 to 4 months before appearance of the lesions. Out of them, the maximum number (6) of patients were from Garyan. Three patients were from Zavia, 2 each from Sabratha, Jadu, Yefren and Rahebat and 1 each from Ziletan, Alzilal, Tarhuna, Surman, Zanjur, Suk Al-Khamis and Beniwalid. These areas are situated between 100 to 150 km south west of Tripoli. From the above information, it seems that these areas might be having an endemic focus of the disease.

History of similar disease in the family members was found in two patients from Rahebat who were brother and sister. Four students who went in an army training camp together to Yefren also developed lesions almost simultaneously. Four other patients who were working in the same agriculture farm also came together.

The minimum duration recorded was one month and the maximum was 7 months. Most of the patients came 2-3 months after appearance of the lesions. Progress of the lesions was gradual and continuous in all the patients. In most of the patients, the lesions started as a furuncle-like nodule but in some as a papule. In 31 patients, the lesions ulcerated after varying intervals, 2 weeks in 14 patients, 4 weeks in 11 patients, 6 weeks in 4 patients, 4 months in one patient and 5 months in another patient. The lesions in 5 patients did not ulcerate.

Before coming to us, 26 patients had not taken any treatment. Three patients had used local antibiotic ointments and 7 had taken local and systemic antibiotic without any significant changes in the lesions.

Sites of involvement were dorsum of hands (15), outer aspects of forearms (14), face (15)



Fig. 1. Multiple scaly plaques on face with lymphoedema on both eyelids.

(Fig. 1), lower part of legs (10) and dorsum of feet (8). The other sites affected were neck (1), trunk (1), and thigh (1).

Majority of the patients had a single lesion (Fig. 2), while the number varied from two to



Fig. 2. A single nodular lesion on face.

six in others (Fig. 3). One patient had twenty lesions. The smallest lesion was 0.5 cm and the biggest was 5 cm in diameter. Most of the lesions were 2 to 3 cm.



Fig. 3. Multiple nodular lesions on hands and forearms.

The type of lesions are shown in table II.

Table II. The clinical types of lesions.

	Ulcers		Plaques		Nodules
	Oozing	Dry	Non-scaly	Scaly	
Number patients	25	2	1	5	3

Secondary infection was seen in 20 patients, regional lymphadenopathy was noticed in 5 patients, lymphangitis in 3 and chronic lymphoedema in one case.

All the patients were in good general health, one patient had rheumatoid arthritis.

Tissue smears stained with Giemsa stain showed LT bodies in 27 cases (Figs. 4 and 5).

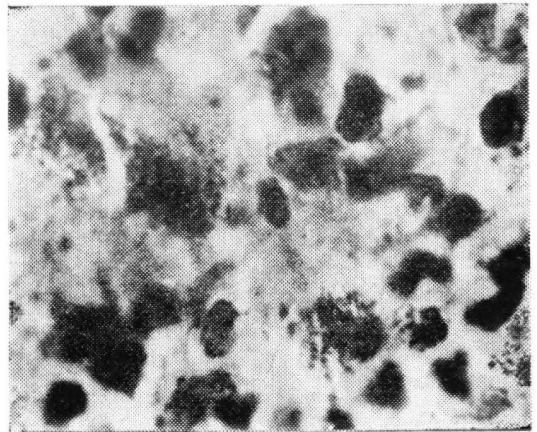


Fig. 4. LT bodies inside a macrophage in a histopathologic section (Giemsa stain).

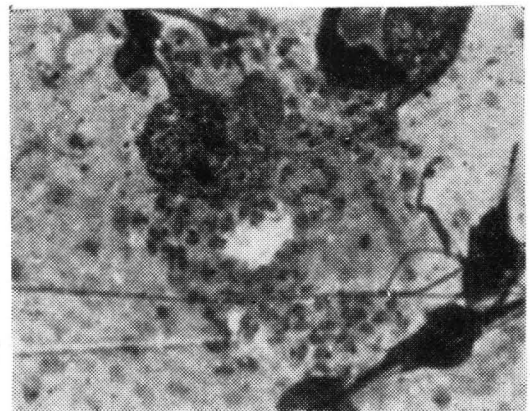


Fig. 5. LT bodies inside a macrophage in a tissue smear.

The parasite was not seen in the smear from 9 cases. Histopathological examination was done in 21 patients. Giemsa stained histopathological section of 18 patients showed LT bodies, while in 3 cases LT bodies were not seen but tubercloid granulomas with some giant cells were observed. Culture of parasite was not done. Bacterial culture of the discharge from the oozing lesions done in 20 cases, showed coagulase positive staphylococci in 12 patients, both staphylococci and beta haemolytic streptococci in 4, *Pseudomonas aeruginosa* and *E. coli* in 2, and *Streptococcus faecalis* and *E. coli* in 1. ESR was raised in 3 patients (30, 68 and 70 mm respectively), one out of them showed leucocytosis (14000). Two more patients showed leucocytosis (11000 and 14000 respectively). Liver function tests were normal in all the patients. One patient was found to be diabetic.

As there is no effective drug treatment available for cutaneous leishmaniasis, different forms of treatment were tried in these cases. Twenty six patients received systemic antibiotics like tetracycline, co-trimoxazole or cephaloridine for a period of two weeks, without any significant improvement in the clinical status. Injectable antimony treatment was administered to 14 patients; out of these trivalent antimony (Foudin) at a dose of 5 ml (8.5 mg of trivalent antimony per ml), daily, intramuscularly for 10 days was given to 10 patients, while pentavalent antimony (sodium stibo-gluconate) at a dose of 6 ml (600 mg) daily intramuscularly for 10 days was given to 4 patients. Out of these 14 cases treated with antimony preparations, 4 showed very good results, 6 showed slight improvement and 3 patients did not show any change in the lesions. In one case the treatment was stopped because of side effects (nausea and vomiting). Out of the patients not responding to antimony therapy, two were given rifampicin 600 mg daily for 4 weeks without any improvement. These patients were further continued on rifampicin along with INH 300 mg daily for 4 weeks leading

to regression of the lesions to almost two thirds of the previous size.

Five patients were followed-up for 1 year, 7 for 9 months, and 10 for 6 months. Twelve patients could be followed upto 3 months only while 2 patients did not come for follow up after the initial treatment in the hospital for 2 weeks. Healing of the lesions with mild to moderate scar formation took place after 1 year in 4 patients.

Comments

Cutaneous leishmaniasis is distributed extensively along the Mediterranean coast mainly North Africa.⁶ Present study reveals that most of the cases come from an area 100 to 150 km south-west of Tripoli, with the maximum incidence from Garyan and nearby areas. Even the 11 patients who were residents of Tripoli, gave history of visits to these places. We could not find a single case from Tripoli who did not visit these areas. It shows that Garyan and nearby areas are endemic for this disease.

The age and sex predilection and the clinical features are by and large similar to those reported in other studies.^{6,8}

Antibiotics like tetracycline, cephaloridine or co-trimoxazole are generally useless, while both types of antimony preparations have been tried by many workers with good results.^{1,6} Results with rifampicin have been variable.⁹ Two of our patients treated with 600 mg of rifampicin daily for 4 weeks did not show any improvement. Further treatment with a combination of rifampicin and INH for one month, showed significant improvement. These patients are still continuing this treatment. Pace¹⁰ has also observed very good results with similar combination therapy. Recently, cryotherapy with CO₂ snow has been used successfully in the treatment of cutaneous leishmaniasis.¹¹ Lately ketoconazole has also been tried with beneficial effect.¹²

References

1. Griffiths WAD and Croft SL : Cutaneous leishmaniasis, Post-graduate Doctor, 1980; 3 : 388-394.
2. Domonkos NA : Andrew's Diseases of the Skin, sixth edition, WB Saunders Company, Philadelphia, 1971, p 476.
3. Kozovniko PV : Two nosological forms of cutaneous leishmaniasis, Amer J Trop Med Hyg, 1963; 12 : 719-724.
4. Kiblavu I and Kurban AK : Outbreak of cutaneous leishmaniasis in Nafa, Northern S A, Leb Med J, 1979 : 30 : 39-40.
5. Kurban AK and Farah FS : Cutaneous leishmaniasis, Medical Digest, 1966; 34 : 113-117.
6. Harman RRM : Parasitic worms and protozoa (Cutaneous leishmaniasis), in : Textbook of Dermatology, 3rd Ed; Editors Rook A, Wilkinson DS and Ebling FJG : Oxford, Blackwell Scientific Publications, 1979; p 902.
7. Bhaktaviziam C : Leishmaniasis, a comparative study (Editorial), Ind J Dermatol Venereol Leprol, 1979; 45 : 315-317.
8. Agarwal SK, Chadda VS, Agarwal SK et al : A study of epidemiology of human cutaneous leishmaniasis in Bikaner (Rajasthan), Ind J Dermatol Venereol Leprol, 1981; 47 : 303-306.
9. Even Paz Z, Weinrauch L, Livshin R et al : Rifampicin treatment of cutaneous leishmaniasis, Intern J Dermatol, 1982; 21 : 110-112.
10. Pace JL : Cutaneous leishmaniasis (letter to the Editor), Arch Dermatol, 1982; 118 : 880.
11. Bassiouny A, El Meshad M, Talaat M et al : Cryosurgery in cutaneous leishmaniasis, Brit J Dermatol, 1982; 107 : 467-474.
12. Urcuyo FG and Zaias N : Oral ketoconazole in the treatment of leishmaniasis, Intern J Dermatol, 1982; 21 : 414-416.