JOURNAL OF CLINICAL AND DIAGNOSTIC RESEARCH

How to cite this article:
BARI A.U .CHILDHOOD CUTANEOUS LEISHMANIASIS.Journal of Clinical and Diagnostic Research [serial online] 2008 August [cited: 2008 August 14]; 2: 973-978.
Available from
http://www.jcdr.net/back_issues.asp?issn=0973-709x&year=2008&month= August &volume=2&issue=4&page=973-978 &id=302

ORIGINAL ARTICLE

Childhood Cutaneous Leishmaniasis

BARI A.U

ABSTRACT

Background: Cutaneous leishmaniasis (CL) is known for its wide variety of clinical presentations. Children are frequent victims of the disease but studies regarding clinical spectrum of the disease are sparse.

Objective: The aim of the study was to explore clinical spectrum of CL in children.

Study design: Case series

Place and Duration of Study: Combined Military Hospital Muzaffarabad from January 2006 to June 2008.

Patients and Methods: Children of all ages having lesions of cutaneous leishmaniasis anywhere on the body were included in the study. Various demographical features and clinical patterns of the lesions were recorded in all cases and subsequently categorized accordingly. Descriptive statistics were used for analysis.

Results: Ninety six cases of childhood cutaneous leishmaniasis were seen. Age range was 4 months to 15 years (mean age = 8.72 $\pm$ 4.47). Male to female ratio was 1.18:1. Majority of the patients (75%) had solitary lesions. Maximum lesion count was 4. All but one patient had lesions on face. Cheeks were most common sites followed by nose and lips. All children were treated with weekly intralesional injections of meglumine antomonate and duration of treatment ranged from 4 weeks to 16 weeks (mean = 10.58 $\pm$ 2.72). Predominant clinical pattern was psoriasiform plaque. All patients responded well to treatment with out any significant side effect.

Conclusion: Childhood cutaneous leishmaniasis makes a major portion of CL and its clinical spectrum is different from that of adult CL. Lesions are characteristically seen on face and respond favourably to weekly intralesional treatment with antimonial compound.

Key Words: Cutaneous leishmaniasis, Childhood cutaneous leishmaniasis, Paediatric leishmaniasis.

Corresponding Author:
Arfan ul Bari, Consultant Dermatologist,
Combined Military Hospital,
Peshawar Pakistan
Ph- 00.92.300.6004478, 00.92.301.6547007
e-mail: albariul@yahoo.com, albariul@gmail.com

Introduction

Leishmaniasis occurs widely throughout Africa, Asia, Europe, and North and South America and the incidence of leishmaniasis seems greater in the Old World than in the New World[1]. The disease is known to be endemic in various regions of Pakistan since many years, but recently it seems to have become an epidemic in the country[2]. CL affects various age groups depending on the infecting *Leishmania* species, geographic location, disease reservoir, and host immunocompetence. The extent and presentation of the disease depends on several factors, including the humoral and cell-mediated immune response of the host, the virulence of the infecting species, and the parasite burden[3],[4],[5]. Children are at greater risk than adults in endemic areas and malnutrition may also contribute to the development of the disease[5]. Old World disease primarily is caused by *Leishmania tropica* in urban areas and *Leishmania major* in rural areas. The incubation period is two to eight weeks, although longer periods have been noted. The disease begins as an erythematous papule at the site of the sandfly bite on exposed parts of the body. The papule

evolves into a nodule and eventually ulcerates and crusts over. There may be multiple lesions, especially when the patient has encountered a nest of sandflies. The ulcer is typically large but painless unless there is secondary bacterial or fungal infection. Old World leishmaniasis lesions tend to heal spontaneously in months. After healing, a depressed scar remains that is usually round but can be irregular. Satellite lesions with a nodular lymphangitis resembling sporotrichosis have been described.[3],[6][7]. CL can become disseminated (diffuse cutaneous leishmaniasis), especially in immunosuppressed persons. This illness can go on for years and does not heal spontaneously. Other unusual types of cutaneous disease include leishmaniasis recidivans, in which small nodules develop around a healed scar, and post_kala-azar dermal leishmaniasis, in which widespread cutaneous lesions arise after a visceral infection [3],[6],[7],[8],[9]. The mucosal form usually occurs after an initial cutaneous infection and may progress to destruction of mucosa and even cartilage [8],[10]. CL typically appears on exposed/uncovered body sites in children and face being the most unprotected and exposed is the commonest site for sandfly bite[11],[12]. The present study is aimed to encompass the whole clinical spectrum of the disease occurring in children of Northern region of Pakistan (Azad Jammu and Kashmir).

Patients and Methods

The study was conducted in Dermatology Outpatient Department, Combined Military Hospital, Muzaffarabad over a period of thirty months from January 2006 to June 2008. All patients between ages of 1 month to 15 years who reported during above-mentioned period from Muzaffarabad district and its surrounding areas irrespective of their age and sex and diagnosed as case of CL were registered. Information was gathered for each patient, including age, sex, geographic location, previous history of leishmaniasis, a stay in an endemic area, lesion location, and the number and size of lesions. Diagnosis was based on history of origin of the patient (endemic areas), and clinical characteristics of lesions (painless, non itchy, slowly evolving nodule, plaque or ulcer on exposed areas of the body, not responding to conventional antibiotics). Patients with a doubtful clinical lesion or who received some definitive treatment for CL were excluded from the trial. Patients who had lesions with apparent infections were first treated with short course of antibiotics and were again reassessed before inclusion. Clinical diagnosis was then confirmed by slit skin smear (SSS) and fine needle aspiration cytology smears (FNACS). Skin biopsy was done in selected cases. Clinically suggestive but smear/biopsy negative cases were subjected to therapeutic trial of weekly intralesional injections antimonial compound (meglumine antimonite) and those responding to two or more injections were also considered confirmed cases of CL. A separate proforma was filled for each patient. Various demographical features and clinical patterns of the lesions were recorded in all cases and subsequently categorized. Computer program SPSS version10 was used to manage and analyze data. Descriptive statistics (frequencies and percentages) were obtained for the variables where applicable. Mean and standard deviation were calculated for continuous variables.

Results

270 patients were registered as cases of CL during the study period. Out of these, 96 (35.5%) were children. Ages varied from 4 months to 15 years (mean age = 8.72 $\pm$ 4.47). Male to female ratio was 1.18:1. Almost all of them belonged to known endemic areas and predominantly (92%) from rural background. Majority of the patients (75%) had solitary lesions. Maximum lesion count was 4. All but one patient had lesions on face. Cheeks were most common sites followed by nose and lips. All children were treated with weekly intralesional injections of meglumine antomonate and the duration of treatment ranged from 4 weeks to 16 weeks (mean = 10.58 $\pm$ 2.72). Predominant clinical pattern was psoriasiform plaque. Other patterns were ulcers, nodules and papules. Some unusual morphologies seen were; cheilitis, perleche, furunculoid, verruciform and chancriform. 7 patients had post inflammatory hypopigmentation, 5 had hyperpigmentation, 4 had secondary infection and 1 child developed milia at the site of healed lesion. 2 developed

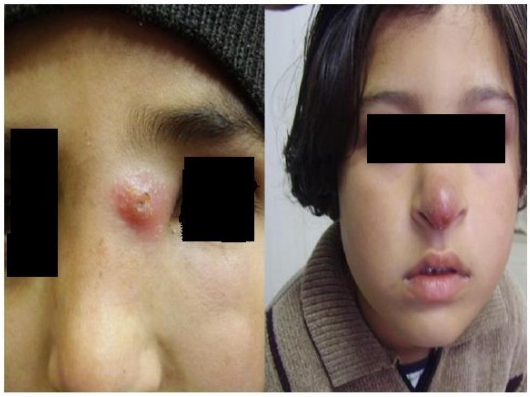
hypertrophic scars while 5 had atrophic scarring. Demographic features of the patients are given in [Table/Fig 1].Clinical features are tabulated in [Table/Fig 2] and different morphologies are shown as[Table/Fig3],[Table/Fig4],[Table/Fig5], [Table/Fig6],[Table/Fig7],[Table/Fig8],[Table/Fig 9],[Table/Fig10],[Table/Fig11],[Table/Fig 12].

(Table/Fig 1)Demographic features of patients of childhood cutaneous leishmaniasis (n=96)

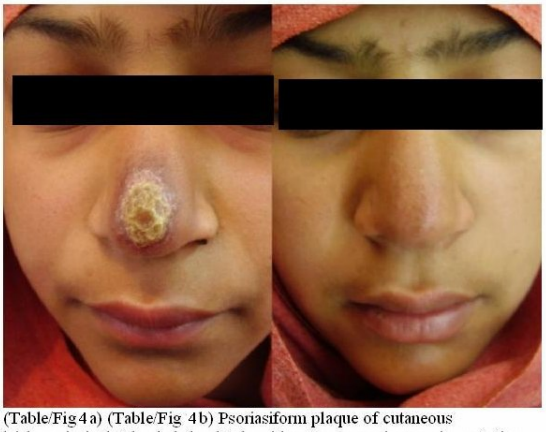
Parameters		No of Patients	Percentage
Age groups (years)	0-5	31	32.30%
	6-10	23	23.90%
	11-15	42	43.80%
Gender	Males	52	54.20%
	Females	44	45.80%
Geographical origin	Urban/Periurban	8	8.30%
	Rural	88	91.70%

(Table/Fig 2)Different clinical characteristics of childhood cutaneous leishmaniasis (n=96)

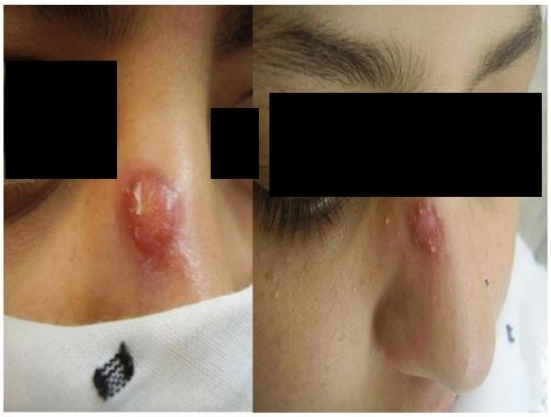
Parameters		No of Patients	Percentages
Number of lesions	1	72	75.00%
	2	16	16.66%
	>2	8	8.33%
Site of lesions	Cheeks	48	50.00%
	Nose	39	40.62%
	Lips	23	24%
	Chin	4	4.16%
	Forehead	3	3.12%
	Eyelids	2	2.08%
	Ear	1	1.04%
Duration of Treatment	4-8 weeks	20	20.80%
	>8 weeks	76	79.20%
Complications	Secondary Infection	4	4.16%
	Hyperpigmentation	5	5.20%
	Hypopigmentation	7	7.28%
	Hypertrophic Scars	2	2.08%
	Atrophic Scars	5	5.20%
	Milia	1	1.04%



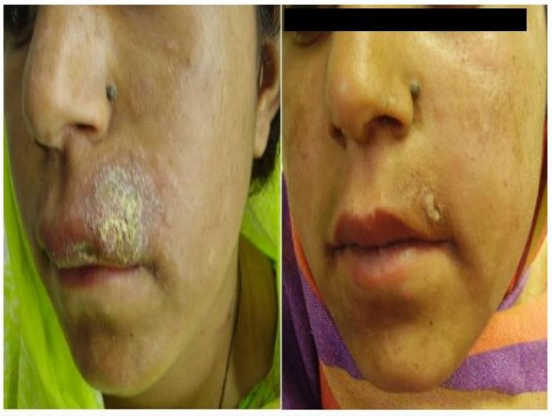
(Table/Fig 3 a) (Table/Fig3 b) Furuncle like lesions on bridge of the nose in one and on tip of the nose in another patient



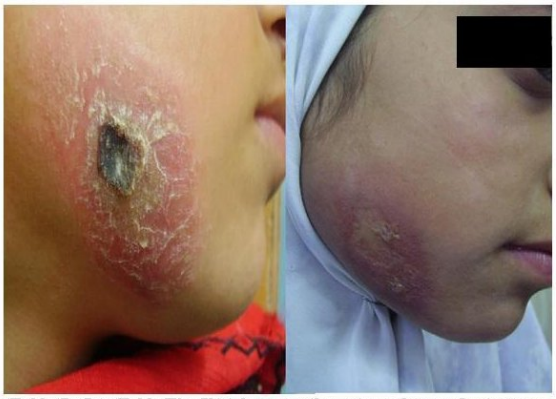
(Table/Fig 4 a) (Table/Fig 4 b) Psoriasiform plaque of cutaneous leishmaniasis that healed absolutely with out any scarring or pigmentation



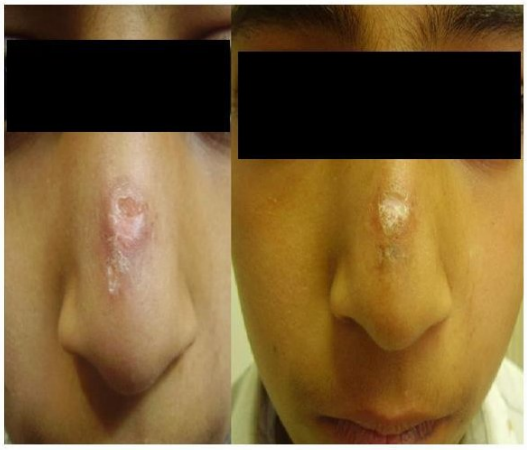
(Table/Fig 5 a) (Table/Fig 5 b) Small nodular nasal lesion of & post treatment milia formation in healed scar



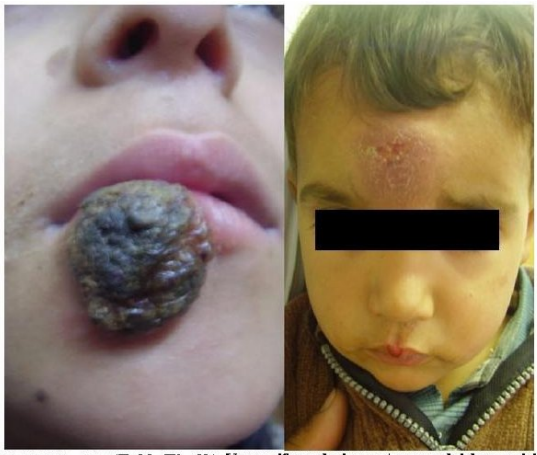
(Table/Fig 6 a) (Table/Fig 6 b) Psoriasiform plaque of cutaneous leishmaniasis on upper lip with perioral extension and healing with small hypertrophic scar.



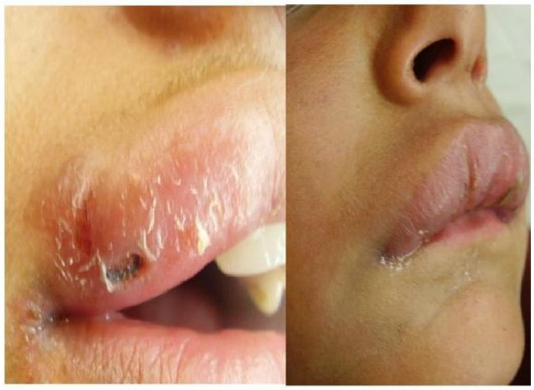
(Table/fig 7a) (Table/Fig 7b)A large erythematous plaque of cutaneous leishmaniasis with central crusting in a young girl that healed with atrophic scar.



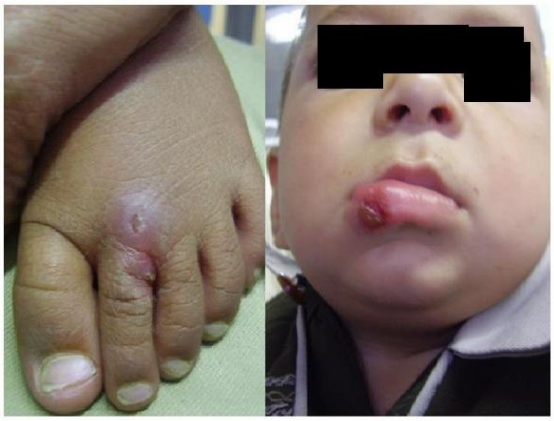
(Table/Fig 8a) (Table/Fig 8b) A small nodular plaque on nose healed with atrophic scar



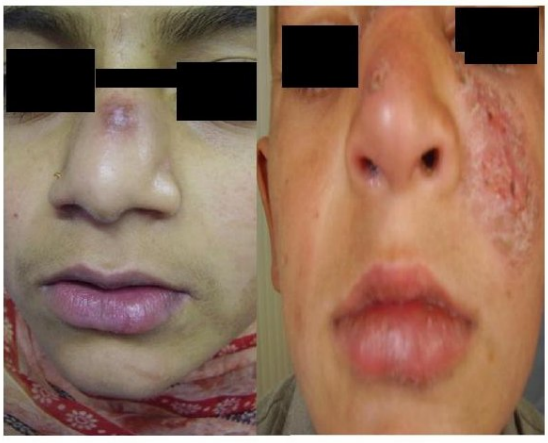
(Table/Fig 9a) (Table/Fig 9b) Verruciform lesion cutaneous leishmanaisis on lower lip in a young girl and a large psoriasiform plaque on forehead of a small child.



(Table/Fig 10 a) (Table/Fig 10 b) Lesions of lip leishmanaisis appearing as perleche in one child and chelitis upper lip in another.



(Table/Fig 11a) (Table/Fig 11b) Cutaneous leishmanaisis on dorsum of foot in and lip leishmaniasis in two infants.



(Table/Fig 12a) (Table/Fig 12b) Multiple lesions of cutaneous leishmanaisis on face of a young girl and a boy.

Discussion

The clinical picture in leishmaniasis depends not only on the infecting *Leishmania* species, but also on the host immune response, which is largely mediated through

cellular immunity. Other factors that affect the clinical picture include the number of parasites inoculated, site of inoculation and nutritional status of the host. Factors such as a non-indigenous individual, old age, co-infection with human immune deficiency virus (HIV), use of oral steroids, and even wound contamination with inorganic materials may alter the clinical picture of CL[3],[13],[14],[15],[16],[17]

Leishmaniasis has been described in all age groups. Although children are frequent victims of cutaneous disease most of the studies found in literature were of visceral leishmaniasis[3],[5],[13],[18]. We found that childhood CL is almost as common as adult disease in an endemic region but clinical pattern was different. Disease affected all age groups of children from infants to adolescents. It was most common in school going children and outdoor explorers. Surprisingly all patients had cutaneous lesions on their faces except one who had it on his foot. Cheeks and nose were the common locations as expected to be due to maximum exposure and least protected from sandfly bites. Frequent involvement of lip was a little unusual as lips are relatively under exposed and motile parts of face that can provide some protection against vector bites. Most of the lesions were typical solitary small lesions of acute CL and were amicably treated with weekly infiltration of lesions with meglumine antimonate. Although we encountered few atypical morphologies; like chancriform, verruciform, cheilitis and perleche mostly the pattern was typical of acute CL (posiasiform plaques, papules and nodules)[19]. No case of chronic cutaneous leishmaniasis, mucocutaneous, disseminated or post kala-azar dermal leishmaniasis was seen. Most of our observations are in agreement with other related studies but prevalence of the disease in childhood and ratio of facial involvement is higher in our study[11],[12],[20],[21],[22] Moreover we did not have any case of lupoid, mucocutaneous and recidevans. Furuncle like, nodular and psoriasiform morphologies on face can be expected as these do occur in certain other cutaneous disorders too. Cheilitis, chancriform and perleche may be explained on the basis of structural characteristic of lip. Verrucous lesion on face could be due to overt skin response to infectious agent. Exclusive facial involvement could possibly be due to the fact that children in these cold areas are almost always protected heavily with clothes and face while the hands remain the only exposed part of the body. Moreover the laxity of the facial tissue and the great vascularization may also be other facilitating factors for sandfly to bite. In some children CL lesions were initially misdiagnosed as impetigo, furuncle, echthyma or cellulites and they reported to us after having some courses of antibiotics. Therefore it is emphasized that a high clinical suspicion should always be observed while encountering such lesions in endemic areas of CL. The standard treatment in our study was intralesional meglumine antimoniate. The World Health Organization recommends one intralesional injection of 1-3 ml repeated at 2-day intervals until complete cure of affected tissues is achieved[23],[24] but due to logistic problems of the patients and cost effectiveness, we administered these injections weekly and the dose was adjusted according to the size of the lesions. Even with the once weekly injections, we did not face any treatment failure. Complication rate was also low and minor complications that occurred in some cases were largely expected of this treatment.

Conclusion

Childhood cutaneous leishmaniasis makes a major portion of CL in northern region of Pakistan and its clinical spectrum is different

from adult CL. Lesions are mostly typical and respond favourably to weekly intralesional treatment with antmonial compound.

References

[1] Klaus SN, Frankenburg S, Ingber A. Epidemiology of cutaneous leishmaniasis. Clin Dermatol 1999; **17**: 257-60.

[2] Bhutto AM, Soomro RA, Nonaka S, Hashiguchi Y. Detection of new endemic areas of cutaneous leishmaniasis in Pakistan: a 6-year study. Int J Dermatol 2003; 42: 543-8.

[3] Ghosn SH, Kurban AK. Leishmaniasis and Other Protozoan Infections. In: Wolff K, Goldsmith LA, Katz SI, Gilchrest BA. Paller AS, Leffell DJ. Eds. Fitzpatrick's Dermatology in General Medicine. Vol. 11, 7th ed. Mcgraw Hill Inc.2008: 2001-9.

[4] Bari AU, Rahman SB. Cutaneous leishmaniasis: an overview of parasitology and hosp-parasite-vector interrelationship. J Pak Assoc Dermatol 2008; **18**: 42-8.

[5] Kafetzis DA. An overview of paediatric leishmaniasis. J Postgrad Med 2003;49:31

[6] Hepburn NC. Cutaneous leishmaniasis: an overview. J Postgrad Med 2003; **49**:50-4.

[7] Murray HW, Berman JD, Davies CR, Saravia NG. Advances in leishmaniasis. Lancet 2005; **366**(9496): 1561-77.

[8] Motta AC, Arruda D, Souza CS, Foss NT. Disseminated mucocutaneous leishmaniasis resulting from chronic use of corticosteroid. Int J Dermatol 2003; **42**: 703-6.

[9] Landau M, Srebrnik A, Brenner S. Leishmaniasis recidivans mimicking lupus vulgaris. Int J Dermatol 1996; **35**: 572-3.

[10] Bari AU, Manzoor A. Mucocutaneous leishmaniasis. Does it really exist in Pakistan? J Pak Assoc Dermatol 2005; **15**: 200-3.

[11] Fenniche S, Souissi A, Benmously R, Ben Jannet S, Marrak H, Mokhtar I. [Childhood cutaneous leishmaniasis in Tunisia: retrospective study of 60 cases]. Med Trop (Mars) 2006; 66(5):456-60.

[12] Kharfi M, Benmously R, El Fekih N, Daoud M, Fitouri Z, Mokhtar I, et al. Childhood leishmaniasis: report of 106 cases. Dermatol Online J. 2004; 10(2):6.

[13] Lopez FV, Hay RJ. Parasitic Worms and Protozoa. In: Burns T, Breathnack S, Cox N, Griffiths C. Eds. Rook's Textbook of Dermatology. Vol ii, 7th ed. Blackwell Science Ltd. 2004: 32.35-32.47.

[14] Puig L, Pradinaud R. Leishmania and HIV co-infection: dermatological manifestations. Ann Trop Med Parasitol 2003; 97 Suppl 1: 107-114.

[15] Motta AC, Arruda D, Souza CS, Foss NT. Disseminated mucocutaneous leishmaniasis resulting from chronic use of corticosteroid. Int J Dermatol 2003; 42(9): 703-6.

[16] Convit J, Ulrich M, Perez M, Hung J, Castillo J, Rojas H, et al. Atypical cutaneous leishmaniasis in Central America: possible interaction between infectious and environmental elements. Trans R Soc Trop Med Hyg 2005; **99**: 13-17.

[17] Rahman SB, Bari AU. Cellular immune host response in acute cutaneous leishmaniasis. J Col Phys Surg Pak 2005; **15**: 463-6.

[18] Altaf C, Ahmed P, Ashraf T, Anwar M, Ahmed I. Clinicopathological features of childhood visceral leishmaniasis in Azad Jammu & Kashmir Pakistan. J Ayub Med Coll Abbottabad. 2005 Oct-Dec;17(4):48-50.

[19] Bari AU, Rahman SB. Many faces of Cutaneous Leishmaniasis. Indian J Dermatol Venereol Leprol 2008; **74**: 23-7.

[20] Sharifi I, Fekri AR, Aflatonian MR, Nadim A, Nikian Y, Kamesipour A. Cutaneous leishmaniasis in primary school children in the south-eastern Iranian city of Bam, 1994-95. Bull World Health Organ. 1998;76(3):289-93.

[21] Talari SA, Talaei R, Shajari G, Vakili Z, Taghaviardakani A. Childhood cutaneous leishmaniasis: report of 117 cases from Iran. Korean J Parasitol. 2006 Dec;44(4):355-60

[22] Jones J, J Bowling J, Watson J Vega-Lopez,F White J, Higgins E. Old world cutaneous leishmaniasis infection in children: a case series. *Archives of Disease in Childhood* 2005;90:530-31

[23] No authors listed. Control of the leishmaniases. Report of a WHO Expert Committee. World Health Organ Tech Rep Ser. 1990;793:1-158.

[24] Sharquie KE, Al-Talib KK, Chu AC. Intralesional therapy of cutaneous leishmaniasis with sodium stibiogluconate antimony.BrJDermatol.1988Jul;119(1):53-7