

A Hospital Based Retrospective Study of Post Kala-Azar Dermal Leishmaniasis (PKDL) Cases.

Abhishek Kumar Jha¹

¹Assistant Professor, Department of Dermatology, Patna Medical College and Hospital, Patna, Bihar, India.

Received: April 2019

Accepted: May 2019

Copyright:© the author(s), publisher. It is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Background: Post Kala-azar dermal leishmaniasis (PKDL) is a cutaneous form of leishmaniasis and usually occurs one to several years after apparent cure of visceral leishmaniasis (VL). **Methods:** The present work was designed as a retrospective tertiary urban hospital based, observational, clinico-epidemiological study during the period from February 2018 to January 2019. **Results:** A total of 24 PKDL patients, 16 males (66.66%) and 8 females (33.34%) were included in the study. The age of the patients ranged from 8 years to 56 years (mean age 30.6 years). Lesions in most of our patients (n=21, 87.50%) were located on the face, including the lip and nose. Most of our patients (n=20, 83.33%) were nodular (non-ulcerative), while two (8.33%) had nodulo ulcerative lesions and one (04.16%) had macular lesions. In the present study, 91.66.47 % (n = 22) of PKDL patients reported history of VL. The median time of manifestation of PKDL after VL treatment were 32 months (range = 5–286 months). Majority (n=20) of cases with history of VL had been treated with amphotericin B while the remaining (n=4,) had been treated with sodium stibogluconate. **Conclusion:** The present study highlights occurrence of PKDL in endemic area. Further epidemiological studies are required for identification of vector and strain of Leishmania involved.

Keywords: Post Kala-azar dermal leishmaniasis (PKDL) and visceral leishmaniasis (VL).

INTRODUCTION

Leishmania, a protozoan parasite, is the causative agent of various forms of leishmaniasis, like visceral form (VL), mucocutaneous form (MCL), and cutaneous form (CL). Postkala-azar leishmaniasis (PKDL) is a cutaneous form of leishmaniasis and usually occurs one to several years after apparent cure of VL caused by *Leishmania donovani*.^[1] In some cases, previous history of symptomatic VL may be absent indicating the possibility of subclinical infection.^[2] In some cases PKDL and kalaazar can occur simultaneously. PKDL is common in India where it occurs in 6–20% of kala-azar (VL) cases following its attack.^[3] The disease is characterized by hypo-pigmented macular form (discrete or confluent) to more developed papular, nodular cutaneous lesions and/or polymorphic forms with mixed lesions predominantly over face. In some cases there is also mucous membrane involvement. In this group oral involvement is most come. Nodules are very similar to the lepromatus leprosy and so in the most of the cases it is the first deferential diagnosis. Of all types macular lesion are

the most common presentation and it mimics petriasis versicolor. Untreated or inadequately treated cases of kala azar are more prone to develop PKDL. In all programmes kala azar treatment is the priority and is effectively controlled by the proper drugs and appropriate vector control but PKDL cases still remain in the society and since vector and hosts are always present so PKDL cases again kala azar epidemic takes place. This is a viscous circle and kala azar hits again and again.

PKDL has long been suspected as a potential reservoir of kala-azar infection. It is considered an important factor in disease transmission between kala-azar outbreaks in India.^[4] So a better understanding of the disease of PKDL is essential to control kala azar.

Profile of PKDL differs in childhood, adulthood, male and female, economical status, proximity to kala azar cases, involvement of the kala azar cases in family, inadequate treatment, health status, immunity status and different other consideration. Better understanding of the disease and to plan the appropriate treatment strategies for elimination of disease kala azar. The present work was conducted for the purpose of evaluating epidemiological and clinical presentation of PKDL patients.

MATERIALS AND METHODS

The present work was designed as a retrospective tertiary urban hospital based, observational, clinico-

Name & Address of Corresponding Author

Dr. Abhishek Kumar Jha,
Assistant Professor,
Department of Dermatology,
Patna medical College and Hospital,
Patna, Bihar, India

epidemiological study during the period from February 2018 to January 2019.

All patients were clinically examined for the identification of characteristic skin lesions such as macular, papular, nodular or mixed/polymorphic forms. Skin sensation test and rK39 immunochromatographic strip test were done in all patients. PKDL were confirmed by direct smear of the lesions and demonstration of *Leishmania* amastigotes within the macrophages.

Detailed history such as age, gender, nativity, history of VL, age at the time of VL, drug taken for VL treatment, history of PKDL and origins of PKDL cases were recorded for each individual at the time of reporting.

The cases were treated with oral miltefosine, 50 mg thrice daily for 2 months or twice daily for 3 months for adults and 2.5 mg/kg/day for 3 months in children.^[5] Pregnant or lactating women and patients co-infected with HIV were excluded.

RESULTS

A total of 38 PKDL patients, 26 males (68.42%) and 12 females (31.57%) were included in the study. The age of the patients ranged from 6 years to 62 years (mean age 31.8 years). Duration of the disease ranged from a minimum of 2 month to a maximum of 15 months with a mean duration of 3.4 months.

Lesions in most of our patients (n=32, 84.21%) were located on the face, including the lip and nose [Figure 1]. Mucosal involvement in the form of the lip lesion extending into the mucocutaneous junction was seen in two patients [Figure 2].

The size of lesions varied from 4 mm to about 48 mm but the average size was about 14mm. The clinical type of lesion in most of our patients (n=32, 84.21%) were nodular (non-ulcerative), while four (10.52%) had noduloulcerative lesions and two (5.26%) had macular lesions.

Table 1:

Patient characteristics		
Sex	Male	26, (68.42%)
	Females	12, (31.57%)
Age	Mean	31.8 years
	Range	6 to 62 years
Duration of disease		2 to 15 month
Characteristics of lesion		
Site	Face	32, (84.21%)
	Trunk	4, (10.54%)
	Mucosa	2, (5.26%)
Size		4 to 48 mm
Clinical type	Nodular	32, (84.21%)
	Noduloulcerative	4, (10.52%)
	Macular	2, (5.26%)
Previous history of VL		34, (89.47 %)
History of VL treatment		
	Amphotericin B30,	(78.94%)
	Sodium stibogluconate	4, (10.52%)

In the present study, 89.47 % (n = 34) of PKDL patients reported history of VL. The median time of

manifestation of PKDL after VL treatment were 34 months (range = 6–264 months). Majority (n=30) of cases with history of VL had been treated with amphotericin B while the remaining (n=4,) had been treated with sodium stibogluconate. [Table 1]



Figure 1 PKDL



Figure 2: PKDL involving muco-cutaneous junction

DISCUSSION

PKDL usually occurs in areas with a hot and dry climate however some indigenously acquired cases have also been described in Assam, Kerala, Haryana, and Himachal Pradesh.^[6,7] First case of PKDL was reported in Madras then West Bengal and then from neighbouring states. In early nineties from one village of Bihar reported 36 cases of kala azar and later the village was named as PKDL village which drew international attention to combat the disease. Majors like DDT spray and search for the disease was no avail and the number of PKDL cases increased. High dose prolonged sodium stibogluconate though effective initially but gradually patient developed resistance. Miltefosin though initial gave new hope but later was withdrawn due to side effect. Amphoteresin B though globally accepted as best option patient acceptability was poor and required constant supervision. Till date there is no drug given as first choice and having good efficacy with least adverse effects. Thus necessitate better understanding of the epidemiology of the disease to formulate the better treatment option with least side effects.

There was a significant delay between clinical presentation and diagnosis of PKDL. This may be attributed to the fact that most of the patients were already treated without considering the diagnosis of PKDL. PKDL presents with a wide spectrum of expression, both clinically and histologically and may mimic other inflammatory and neoplastic diseases and should be differentiated with erysipelas, chronic eczema, herpes zoster, paronychia; and uncommon disorders such as lupus vulgaris, squamous cell carcinoma, sporotrichosis, mycetoma, and other deep mycoses.^[8] Most of the lesions in our study were persistent, slowly growing, painless erythematous nodules and plaques (nodular type) which were inconsistent with earlier findings.^[9-11] Ulcerative lesion has never been reported. Nodules are very similar to the lepromatous leprosy and so in the most of the cases it is the first deferential diagnosis. Of all types, macular lesion are the most common presentation and it mimics petriasis versicolor. Macular lesion are mainly presents on face and trunk and in most of the cases they remain unprogressive for years. In these cases PKDL is diagnosed only when proper history of kala azar in past is obtained. In these cases histopathology and other test is not confirmatory. Oral lesion are very specific. In most of the cases it is situated on the tongues and lip usually as nodules. These oral lesions may have co presence of lesions on extremities and trunk. They can be as nodular or macular.

The various therapeutic modalities for the treatment of PKDL includes physical agents, drugs such as antimonials,^[12] azole antifungals,^[13-15] amphotericin B,^[16] miltefosine,^[17] and surgical procedures. However, intralesional or systemic administration of antimonial compounds are still considered the standard treatment of PKDL.^[18]

Limitations

The study population was small and the identification of the species of *Leishmania* and *Phlebotomus* could not be done.

CONCLUSIONS

PKDL is a cutaneous form of leishmaniasis and usually occurs in the endemic areas of VL caused by *Leishmania donovani*. Since no parasite isolation and characterisation was carried out, further epidemiological studies are required for identification of vector and strain of *Leishmania* involved. Our study cannot be considered as true representation of magnitude of PKDL. Newer diagnostic modalities such as polymerase chain reaction may help in confirming the diagnosis. In this direction many attempt are being made by Indian Council Of Medical Research (ICMR), World Health Organisation (WHO) which conducted a recent workshop on guidelines for effective diagnosis and treatment of PKDL to halt the

repeated on slaught of kala azar which is mainly due to its reservoir PKDL.

REFERENCES

1. P. C. Sen Gupta, J. C. Banerjee, and P. B. Bhattacharya, Handbook of Tropical Diseases, Academic Publishers, Calcutta, India, 6th edition, 1960.
2. A. M. El Hassan, H. W. Ghalib, E. E. Zijlstra et al., "Post kala-azar dermal leishmaniasis in the Sudan: clinical features, pathology and treatment," Transactions of the Royal Society of Tropical Medicine & Hygiene, vol. 86, no. 3, pp. 245-248, 1992.
3. A. D. M. Bryceson, "Leishmaniasis," in Manson's Tropical Diseases, pp. 1213-1245, W. B. Saunders. Manson Bahr, P.E.C., London, UK, 20th edition, 1996.
4. C. P. Thakur and K. Kumar, "Post kala-azar dermal leishmaniasis: a neglected aspect of kala-azar control programmes," Annals of Tropical Medicine and Parasitology, vol. 86, no. 4, pp. 355-359, 1992.
5. Ramesh V, Katara GK, Verma S, Salotra P. Miltefosine as an effective choice in the treatment of post-kala-azar dermal leishmaniasis. Br J Dermatol. 2011;165:411-4.
6. Sundar S. Drug resistance in Indian visceral leishmaniasis. Trop Med Int Heal. 2001;6:849-54.
7. Muhammed K, Narayani K, Aravindan KP. Indigenous cutaneous leishmaniasis. Indian J Dermatol Venereol Leprol 1990;56:228-9.
8. Sharma NL, Mahajan VK, Kanga A, Sood A, Katoch VM, Mauricio L et al. Localized cutaneous leishmaniasis due to *Leishmania donovani* and *Leishmania tropica*: Preliminary findings of the study of 161 newcases from a new endemic focus in Himachal Pradesh, India. Am J Trop Med Hyg 2005;72:819-24.
9. Bari AU, Rahman SB. Many faces of cutaneous leishmaniasis. Indian J Dermatol Venereol Leprol 2008;74:23-7.
10. Klaus S, Frankenburg S. Cutaneous leishmaniasis in the Middle East. Clin Dermatol 1999;17:137-41; discussion 105-6.
11. Ashford RW. Cutaneous leishmaniasis: Strategies for prevention. Clin Dermatol 1999;17:327-32.
12. Momeni AZ, Aminjavaheri M. Clinical picture of cutaneous leishmaniasis in Isfahan, Iran. Int J Dermatol 1994;33:260-5.
13. Sharma RC, Mahajan VK, Sharma NL, Sharma A. A new focus of cutaneous leishmaniasis in Himachal Pradesh (India). Indian J Dermatol Venereol Leprol 2003;69:170-2.
14. Alsaleh QA, Dvorak R, Nanda A. Ketoconazole in the treatment of cutaneous leishmaniasis in Kuwait. Int J Dermatol 1995;34:49-57.
15. AlFouzan AS, al Saleh QA, Najem NM, Rostom AI. Cutaneous leishmaniasis in Kuwait. Clinical experience with itraconazole. Int J Dermatol 1991;30:519-21.
16. Dogra J, Saxena VN. Itraconazole and leishmaniasis: A randomised double blind trial in cutaneous disease. Int J Parasitol 1996;26:1413-5.
17. Goyonlo VM, Vosoughi E, Kiafar B, Nahidi Y, Momenzadeh A, Taheri AR. Efficacy of intralesional amphotericin B for the treatment of cutaneous leishmaniasis. Indian J Dermatol 2014;59:631.
18. Madke B, Kharkar V, Chikhalkar S, Mahajan S, Khopkar U. Successful treatment of multifocal cutaneous leishmaniasis with miltefosine. Indian J Dermatol 2011;56:587-90.

How to cite this article: Jha AK. A Hospital Based Retrospective Study of Post Kala-Azar Dermal Leishmaniasis (PKDL) Cases. Ann. Int. Med. Den. Res. 2019; 5(4):DT01-DT03.

Source of Support: Nil, **Conflict of Interest:** None declared