Original Article

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Pattern of Leprosy: A Histomorphological Study with Clinical Correlation in Ajmer District

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ABSTRACT

Background: Leprosy (Hansen's disease) still continues to be an important public health problem. The present study was carried to correlate histological diagnosis of skin biopsies of untreated leprosy cases with clinical diagnosis using Ridley- Jopling classification.

Material and Methods: 124 skin biopsies of untreated leprosy cases over a period of 6 years were included. Paraffin sections of biopsies were stained with Haematoxylin & Eosin, Ziehl- Neelsen's & Fite stains examined and classified histopathologically according to Ridley-Jopling scale and then correlated with clinical diagnosis.

Results: Prevalence rate of leprosy in Ajmer district is 0.16 per 10,000 population per annum. Among the 124 biopsies, most cases were of indeterminate type (39.51%), followed by LL type (17.74%), TT type (16.93%), BT type (10.48%), BL type (07.25%), BB type (05.64%), histioid type (2.41%). Most commonly the patients were affected in 4th decade of life. Male and female ratio was 2.26:1. Most common clinical presentation was anesthesia.

Conclusion: Correlation of clinical and histopathological features along with bacteriological index appears more useful for accurate typing of leprosy than considering any of the single parameters alone. This helps the clinician for better care and management of the patients.

Keywords: *Leprosy, Ridley Jopling Classification, Histopathology*

Introduction

Leprosy is one of the leading causes of physical disabilities, which contribute to intense social stigma resulting in discrimination of patients and their families. Leprosy is known since ancient times as "Kushtaroga". The causative agent of leprosy, Mycobacterium Leprae, was discovered in 1873 by Armauer Hansen.[1]

Worldwide, two to three million people are estimated to be permanently disabled because of leprosy. World health organization (WHO) launched a 5 year "Global leprosy strategy 2016-2020" in April 2016 titled 'accelerating towards leprosy free world'. This was built on the earlier 5-year strategy 2011- 2015 that focused on early leprosy detection to reduce disabilities. In India, the National leprosy eradication Programme (NLEP) is centrally sponsored health scheme of the Ministry of Health and family welfare, Government of India which strategize and makes plans which are implemented by states and union territories. Due to their efforts, from a prevalence rate of 57.8/10,000 in 1983, India has succeeded in bringing down the prevalence rate to 0.66/10,000 in 2016. Despite the above successes, the fact remains that India continues to account for 60% of new cases reportedly globally each year and is among the 22 "global priority countries" that contribute 95% of world numbers of leprosy warranting a sustained effort to bring the numbers down.[2] It is an important public health problem in most of the developing countries. Hence, control of communicable disease is based on identifying and destroying or attacking the causative organism.[3]

Leprosy or Hansen's disease, is a chronic granulomatous infectious disease. It is a slowly progressive chronic disease which mainly affects peripheral nerves and skin which can express itself in different forms, depending on the immune status of the host.

Depending on degree of immunity, clinical and histopathological features, various types of leprosy gradually may develop.[4] Histopathological examination of skin or nerve biopsies and demonstration of acid-fast bacilli in histopathological section and in slit skin smear, aid in the diagnosis of leprosy.[5]

The clinical manifestations of leprosy are varied and diverse and can mimic a variety of unrelated diseases.

Presentation may vary from an insignificant skin lesion to extensive disease-causing profound disability/ deformities.[6]

Since exact typing of leprosy is sometimes clinically not possible, added to this, the poor results obtained by slit skin smear leads to false negative diagnosis. To prevent this, histopathological examination should be done in all suspected cases.

Biopsy specimen for histopathology can be valuable aid to reach confirmatory diagnosis, its subtypes, differential diagnosis, prognosis of the disease and assessment or regression of the disease in patient under treatment and also for research. [7,8]

The spectrum of leprosy is a continuum, and patients may move in either direction according to host response and treatment. The standard delineation follows that of "Ridley and Jopling", with categories defined, along the spectrum by combination of clinical, microbiologic, histopathologic, and immunologic indices: -

- TT (Tuberculoid)
- BT (Borderline Tuberculoid)
- BB (Mid Borderline)
- BL (Borderline Lepromatous)
- LL (Lepromatous)

The term borderline is used to denote patterns that share some features of both tuberculoid and lepromatous leprosy. [9,10]

Material and Methods

The study was carried out on the skin punch biopsies of thickness 4mm from untreated cases of leprosy taken in the Department of Dermatology and reported in the histopathology section of the Department of Pathology, J.L.N Medical College, Ajmer, Rajasthan (referral hospital of Ajmer district) between June 2014 to Dec 2019. After adequate fixation for about 8-12 hours in 10% formalin, the biopsies are submitted for routine processing, followed which the paraffin embedded sections of 3-5µ thickness are stained with haematoxylin and eosin stain and all cases of leprosy were examined for: - a) Epidermal atrophy, epithelioid granulomas, number and distribution of lymphocytes, histiocytes and foam cells. b) Infiltration of nerves, blood vessels and adnexa. c) Grenz zone.

Sections stained with Ziehl Neelsen's stain and Modified Fite's stain were examined for lepra bacilli in all cases. Histopathological findings were graded into TT, BT, BB, BL and LL according to Ridley and Jopling scale. Sections showing scattered non- specific lymphohistiocytic infiltration with cellular reaction within dermal nerve or presence of bacilli in subepidermal zone/ arrectores pilorum muscle/ dermal nerve were classified as indeterminate leprosy and also included for purpose of analysis. Biopsies which did not include full depth of dermis together with a portion of subcutaneous fat were considered as inadequate and not classified histologically.

Clinical diagnosis of leprosy cases (as provided by department of Dermatology) using Ridley and Jopling scale was correlated with the results of histopathologic examination of their respective biopsies which did not reveal histology of the leprosy (non-specific) or showing features of reactional leprosy were excluded from clinico-histopathological correlation.

Result

The present study included 124 skin biopsies from the patient who were clinically diagnosed as leprosy from June 2014 to Dec 2019. Population of Ajmer district as per census of 2011 is 2,583,052. Prevalence rate of leprosy in Ajmer district is 0.16 per 10,000 population per annum.

Among the 124 biopsies most cases were of indeterminate type 49(39.51%) followed by LL type 22(17.74%) cases, 21(16.94%) cases were of TT, 13(10.48%) cases were of BT, BL were 9(7.25%), 7(5.64%) cases were of BB and 3(2.41%) cases were of histoid.

In present study patients ranged from 3 years to 84 years. Among them 38(30.64%) of the patients were in 4th decade, 29(23.33%) were in 3rd decade, 18(14.51%) were in 6th decade, 14(11.29%) were in 5th decade, 12(9.67%) were in 7th decade, 8(6.45%) were in 2nd decade and 3(2.41%) were in 8th decade.

There were 86 (69.35%) male patients and 38(30.64%) female patients with male to female ratio (M: F) of 2.26:1.

Among 14 clinically diagnosed BT cases 9(64.28%) were of BT type, 2(14.28%) were of TT type, 1(7.14%) were LL, 2(14.28%) were IL. Among 5 clinically diagnosed BL cases, 3(60%) were of BL type and 2(40%) were of BT type. Among 15 clinically diagnosed LL cases, 14(93.33%) were LL type, 1(6.66%) were of IL type. Among 13 clinically diagnosed ENL cases, 8(61.53%) were of TT type, 1(7.69%) were of BL type.

Maximum concordance was seen in mid borderline and histoid type leprosy (100%) followed by LL (93.33%). Concordance was more towards lepromatous pole than tuberculoid pole.

On histopathological correlation with clinical presentation, it is seen that in indeterminate leprosy the most common presentation was anaesthesia, in borderline leprosy it

was nodule or anaesthesia, in borderline tuberculoid it was anaesthesia, in tuberculoid it was anaesthesia, in borderline lepromatous it was papule, in lepromatous it was anaesthesia, in histoid it was nodule or nerve thickness. Overall, for most of the cases the most common presentation for any type of leprosy is anaesthesia.

Discussion

Leprosy or Hansen's disease is a slowly progressive infection caused by Mycobacterium leprae affecting the skin and peripheral nerves. It is exclusively a disease of humans and only source of infection is a leprosy patient. Leprosy still continues to be an important public health problem.

Accurate diagnosis is of fundamental importance to all aspects of leprosy epidemiology, management and prevention of disability. Under diagnosis will lead to continued transmission of disease and much needless sufferings. Histopathological examination of skin lesion is an important tool in accurate diagnosis and classification of leprosy and still remains the gold standard.

The prevalence rate of leprosy in Ajmer district is 0.16 per 10,000 population. Most of the cases are due to migrant population especially from West Bengal in Ajmer, particularly in Dargah region.

Disease occurrence in leprosy is often related to age at detection rather than age at onset of disease. It is known to occur at all ages ranging from early infancy to very old age.[11]

Of the 124 patients in the present study, the patients with age group of 30-39 years (4th decade) were affected most and patients below 9 years were affected least.

Similar observations were made by Kaur et al. [12] in other studies. Although exact reason cannot be given for this age distribution, variable and long incubation period may be considered.[13]

Generally, leprosy is believed to be more common in males than in females.[14] Majority of the patients who underwent the biopsy were males (69.35%) with the male to female ratio of 2.26:1, which is similar to the findings of other authors. [15,16]

Male predominance may be because of many factors like industrialization, urbanization and more opportunities for contact in males. Social customs and taboos may account for the smaller number of females reporting for treatment to the hospital.[15]

There are several factors that influence the sex predominance in endemic areas. The main factors causing

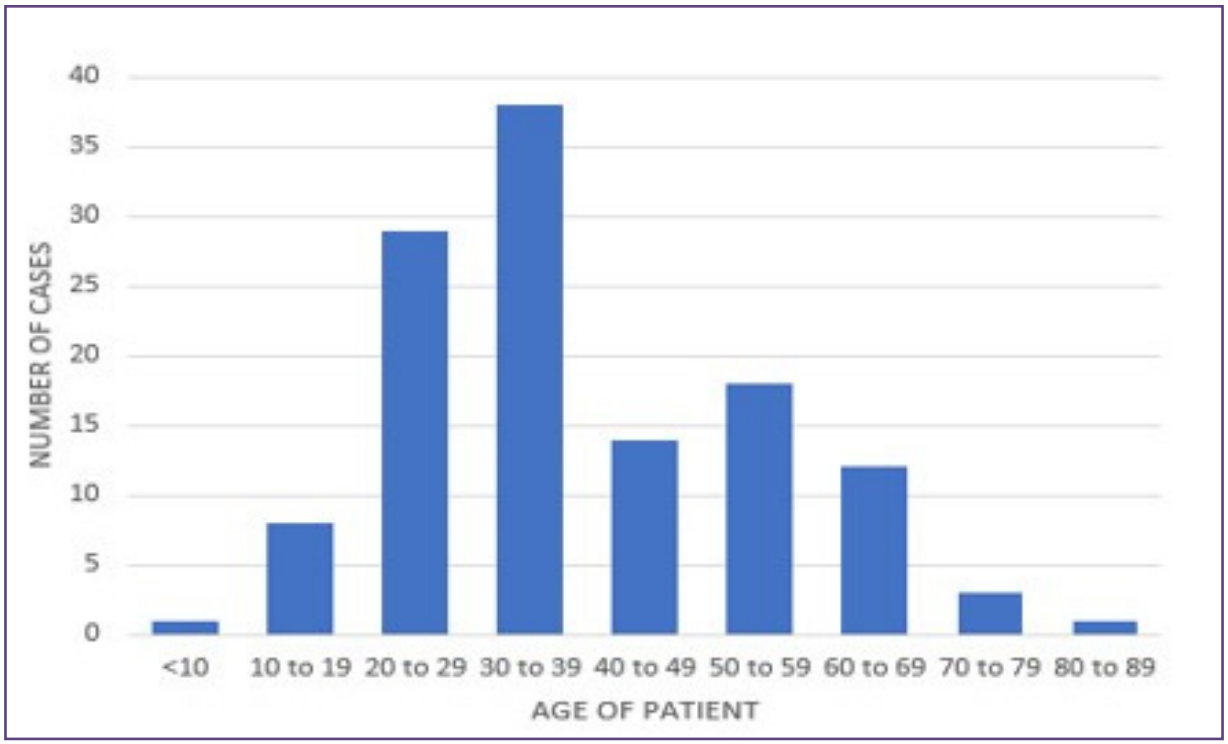

Fig. 1: Age wise distribution

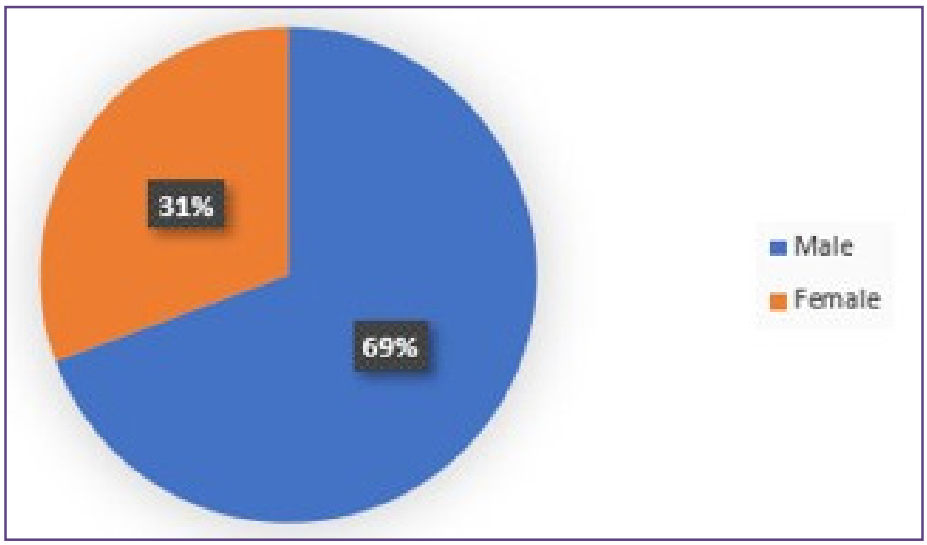

Fig. 2: Sex wise distribution.

Fig. 3: Erythematous patch on forearm.

Fig. 4: Hypopigmented patch

Fig. 5: Facial infiltration of nodules and madarosis

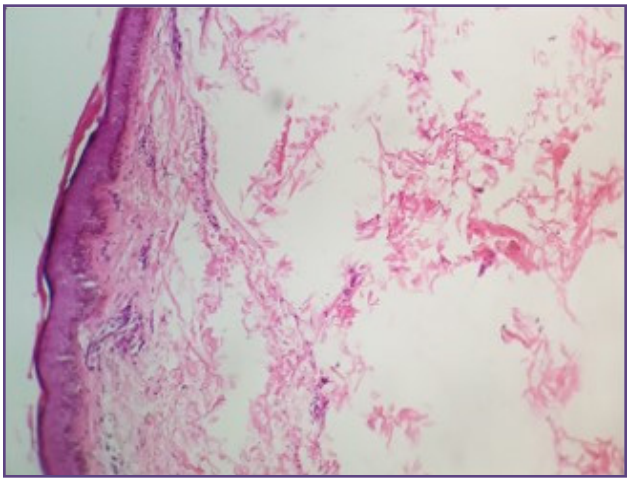

Fig. 6: Indeterminate leprosy (10X)

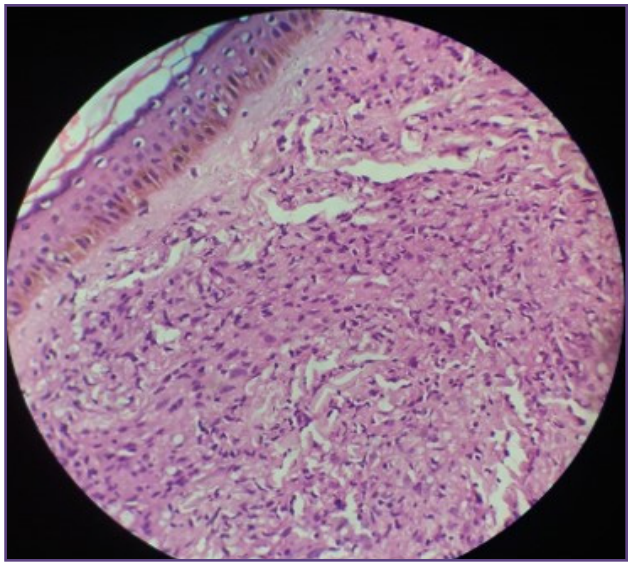

Fig. 7: Histoid leprosy

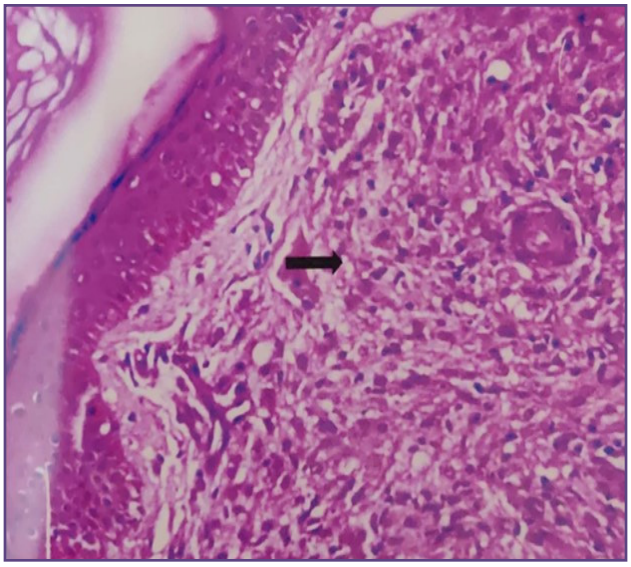

Fig. 8: Tuberculoid leprosy (20X) showing granuloma formation (Arrow)

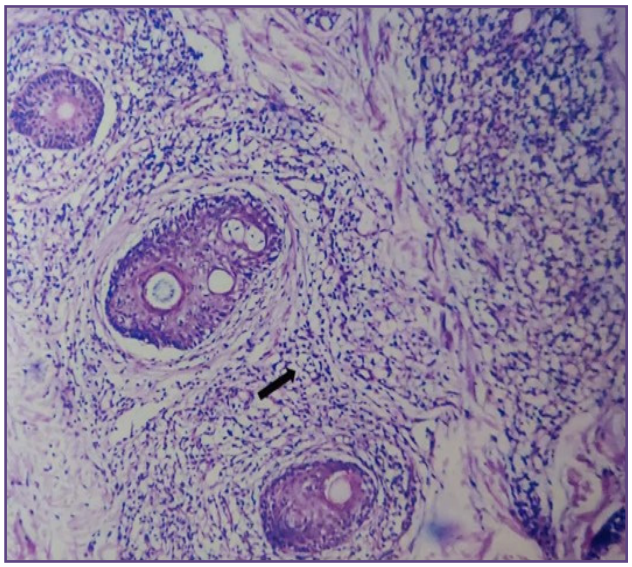

Fig. 9: Lepromatous leprosy (10X) showing infiltration of macrophages (arrow) around hair follicles in dermis.

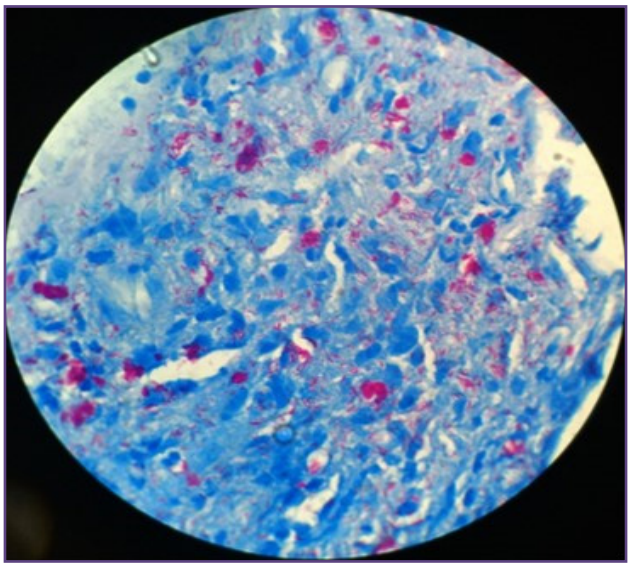

Fig. 10: Histoid leprosy. Fite stain (400X)

the sex difference is the opportunity for contact and practically no difference is noted when the opportunity for contact remains the same.[17]

The most common encountered type of leprosy was indeterminate leprosy 49 biopsies (39.51%), second common type was LL 22 biopsies (17.74%), BB 7 biopsies (5.64%) was the least encountered type. In our study, indeterminate was more common, which may be due to increased awareness caused by national programmes leading to early diagnosis.

Overall, the most common presentation in all types of leprosy is anaesthesia. Same observations were observed by Verma OP[18] and Gurubasavaraj H et al[19] in their study where most common lesion was anaesthesia.

No other similar study has been performed on leprosy in Ajmer district. Limitation of the following study is that it considers data received only in the J.L.N hospital and the biopsy done in the private sector is not considered in the following study.

Conclusion

Considering the data of the present study, indeterminate leprosy is the most common subtype, which shows the impact of national programme for leprosy, resulting in awareness and early diagnosis.

According to our study indeterminate leprosy cannot be identified in view of clinical symptoms only as other types of leprosy can present as same clinical presentation of indeterminate leprosy.

Biopsy specimen for histopathology is a valuable aid to reach confirmatory diagnosis, prognosis of the disease and progression or regression of the disease in patient under treatment and also for research.

As there can be some degree of overlapping among different types of leprosy both clinically and histopathologically. Correlation of clinical and histopathological features along with bacteriological index appears more useful for accurate typing of leprosy than considering any of the single parameters alone. This helps the clinician for better care and management of patients. [20,21,22]

Abbreviations and symbols

IL- Indeterminate leprosy

BB- Borderline leprosy

TT- Tuberculoid leprosy

BL- Borderline lepromatous leprosy

LL- Lepromatous leprosy

BT- Borderline tuberculoid

A- Anesthesia (Loss of sensation)

HP- Hypopigmentation

N- Nodule

P- Papule

TU – Trophic Ulcer

C- Contracture

E- Erythematous patch

W- Weakness

NT- Nerve thickness

GSA- Glove and stocking anesthesia

Conflict of interest

None

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Reference

1. Rees RJW, Yound DB. The Microbiology of leprosy. In: Hastings RC, Opromolla DVA. Editors, leprosy, 2nd ed, New York, Churchill Livingstone;1994. p.49-83.
2. P. Narasimha Rao, Sujai Suneetha. Current situations of leprosy in India and its future implications. Indian Dermatol Online J.2018; 9(2): 83-89.
3. Ganapathy R, Revankar CR. Leprosy, Controle. In: Valia RG, Valia AR, Editors, Textbook and Atlas of Dermatology, Bombay Bhalani Publishing House;1994. p.1427-1437.
4. Polycarpou A, Walker SL, Lockwood DN. The clinical and immunological features of leprosy. BrMed Bull 2006; 78:103-21.
5. Tiwari M, Ranabhat S, Maharjan S. Clinico-histopathological correlation of leprosy: A retrospective study of skin biopsy specimens in Chitwan Medical College. Inter J Medical Sci Res Prac. 2015; 2(1):8-11.
6. Nayak SV, Shivarudrappa AS, Nagarajapa AH, Sacchidanand S, Ahmed SM. Role of modified rapid AFB method in histopathological sections of hansen's disease. Ind J Dermatol Venerol Leprol 2003; 69:173-4.
7. Shantaram B, Yawalkar SJ. Leprosy Differential disgnosis. In: Valia RG, Valia AR, editors. Textbook and atlas of Dermatology. Bombay Bhalani Publishing house;1994. p.1385-91.
8. Ridley DS, Jopling WH. Classification of leprosy according to immunity: a five-group system. Int J Lepr 1966; 34:255.
9. Fite GL, Mansfield RE. The role of histopathology in the study of leprosy. Arch Dermatol 1969; 100:178-83.
10. Britton WJ, Lockwood DNJ. Leprosy. Lancet 2004; 363:1209-1219.
11. Mitra K, Biswas S, Saha B, Dasgupta A. Correlation between clinical and histopathological criteria for the classification of leprosy. Ind J Dermatol Venerol Leprol 2001; 16:135-7.
12. Noordeen SK. The epidemiology of leprosy. In: Hastings RC, editor, leprosy.New York: Churchill Livingstone, 1985.p.15-29.
13. Suneetha S, Arundhati S, Chandi S, Kurian N, Chacco CJG. Histological studies in primary neuritic leprosy: Changes in the apparent normal skin. Lepr Rev 1998; 69:351-357.
14. Sharma Anuja, Sharma RK, Goswami KC, Bhardwaj Subhash. Clinicohistopathological correlation in leprosy. JK SCIENCE, July- September 2008; 10(9):120-23.
15. Gupte MD. Leprosy: Epidemiology. In: Valia RG, Valia AR, editors, Textbook and atlas of Dermatology. 2nd ed. Mumbai: Bhalani Publishing House; 2001.p.1543-1552.
16. Sehgal VN, Gorpade A, Saha K. Urban leprosy an appraisal from Northern India. Lepr Rev 1984; 55:159-166.
17. Vargas-Ocampo F. Analysis of 6000 skin biopsies of the National Leprosy Control Programme in Mexico. Int J Lepr 2004; 72(4):427-430.

18. Verma OP. Some epidemiological features of leprosy in a rural area in Hoogly district. Lepr India 1976; 48(4): 371-81.
19. Gurubasavaraj H, Kumar P, Shashikala PS, Shivamurthy V. Histomorphological study of leprosy. Afr J Med Health Sci 2013; 12:68-73.
20. Chaturvedi RM. Epidemiological study of leprosy in Mewani Suburb of Bombay. Lepr Rev 1984; 55:159-166.
21. Bhatia AS, Katoch K, Narayanan RB, Ramu G, Mukherjee A, Lavania RK. Clinical and histopathological correlation in the classification of leprosy. Int J Lepr 1993;61(3):433-38.
22. Gillis TP, Williams DL. Polymerase chain reaction and leprosy. Int J Lepr 1991;59(2):311-16.

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