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ORIGINAL ARTICLE

Assessment of cases of leprosy- A clinical study

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ABSTRACT:
Background: Leprosy is a chronic infectious disease caused by Mycobacterium (M.) leprae. The present study assessed cases of leprosy. **Materials & Methods:** 48 clinically confirmed cases of leprosy of both genders were included. Slit skin smear was performed in each case at the time of diagnosis and 6 monthly thereafter. **Results:** 36 patients were of primary cutaneous and 12 were of primary neuritic, among primary cutaneous most common variety was TT seen in 12 and in primary neuritic was PN seen in 5. Grade 1 deformity was seen in 9 and grade 2 in 4. Lepra reaction type 1 was seen in 3 and type 2 in 8 patients. The difference was significant (P< 0.05). **Conclusion:** Histopathological examination is must for confirmation of diagnosis in doubtful cases of leprosy. Maximum case were of primary cutaneous type.
Key words: Leprosy, Slit skin, Mycobacterium leprae.

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INTRODUCTION

Leprosy is a chronic infectious disease caused by Mycobacterium (M.) leprae. Worldwide, 210,758 new cases were diagnosed in 2015. The highest incidence is found in India, Brazil, and Indonesia.[1] While the exact route of transmission remains unknown, nasal droplet infection is thought to be most likely. The pathogen primarily affects the skin and peripheral nervous system. The disease course is determined by individual host immunity. Clinically, multibacillary lepromatous variants are distinguished from paucibacillary tuberculoid forms. Apart from the various characteristic skin lesions, the condition is marked by damage to the peripheral nervous system.[2]
Leprosy still continues to be one of the major public health problems in many countries including India. Though in 2010-2011, Annual New Case Detection Rate (ANCDR) has been reduced to 4.12% from 10.93 during 2009-2010, there are still 48.6% of MB cases detected in 2010-2011.[3] The principle of reducing the load of infection is the cornerstone of leprosy control. Early diagnosis and early adequate drug treatment is very important aspect to reduce the load. For this, most of the times, clinical judgment and skin smear examination is adequate. But in some cases, to label only on clinical bases is difficult.[4]
Mycobacterium leprae is a non-motile, acid-fast rod, 4–7 µ m long. Microscopically, M. leprae appears red on Ziehl-Neelsen stain as the dye carbolfuchsin cannot be washed out by hydrochloric or sulfuric acid.[5] Mycobacterium leprae cannot be cultured on any known medium but only in animal cultures. Using animal cultures, the pathogen only grows in mouse paws and the nine-banded armadillo. Low temperatures facilitate the growth of M. leprae, which grows slowly and divides only about every twelve days. The M. leprae genome was decoded in 2001. It contains about 3.3 million base pairs; the number of functional genes is 1,600 and is thus much lower than in M. tuberculosis.[6] The present study assessed cases of leprosy.

MATERIALS & METHODS

The present study comprised of 48 clinically confirmed cases of leprosy of both genders. All were included after they agreed to participate and gave their written consent.
Demographic profile such as name, age, gender etc. was recorded. A careful examination was performed. Family/contact history was also noted. Slit skin smear was performed in each case at the time of diagnosis and 6 monthly thereafter. Results thus obtained were subjected to statistical analysis. P value less than 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Total- 48		
Gender	**Males**	**Females**
Number	30	18

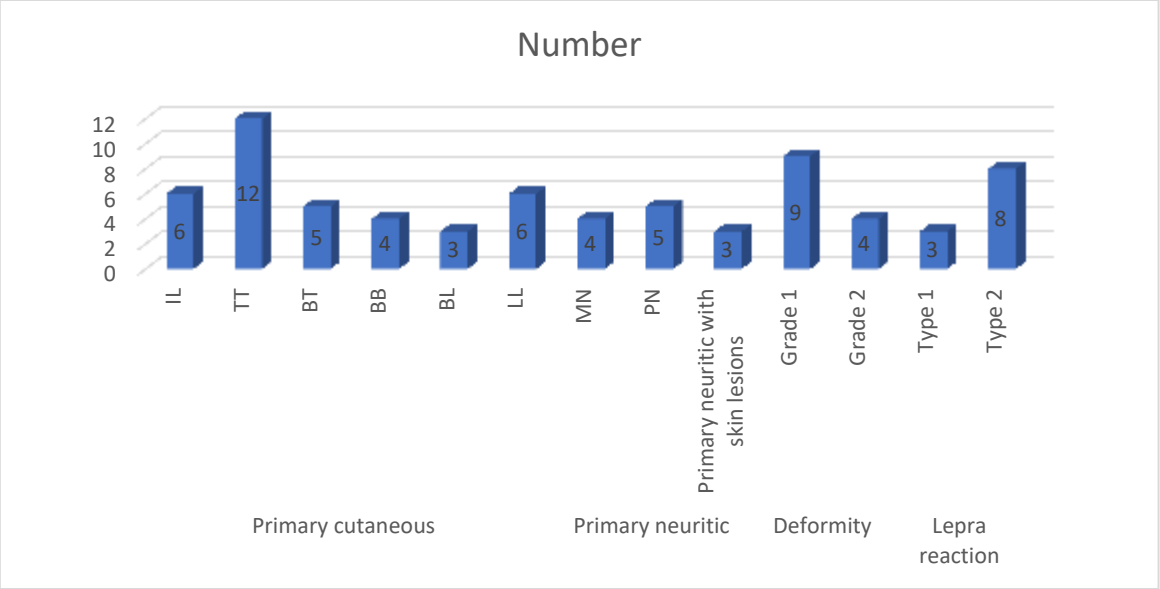
Table I shows that out of 48 patients, males were 30 and females were 18.

Table II Clinical profile of patients

Parameters	Variables	Number	P value
Primary cutaneous	IL	6	0.01
	TT	12	
	BT	5	
	BB	4	
	BL	3	
	LL	6	
Primary neuritic	MN	4	0.12
	PN	5	
	Primary neuritic with skin lesions	3	
Deformity	Grade 1	9	0.04
	Grade 2	4	
Lepra reaction	Type 1	3	0.05
	Type 2	8	

Table II, graph I shows that 36 patients were of primary cutaneous and 12 were of primary neuritic, among primary cutaneous most common variety was TT seen in 12 and in primary neuritic was PN seen in 5. Grade 1 deformity was seen in 9 and grade 2 in 4. Lepra reaction type 1 was seen in 3 and type 2 in 8 patients. The difference was significant (P< 0.05).

Graph I Clinical profile of patients



DISCUSSION

The clinical variability of leprosy is essentially determined by the microorganism's tropism for the skin and peripheral nerve tissue and by the patient's genetically determined and individually variable susceptibility to M. leprae.[7] Depending on the clinical variant, the clinical morphology in the skin varies considerably. There are marked differences both in the number of lesions as well as their distribution pattern.[8] Occult subclinical infections are common in endemic regions. Manifest disease occurs in only about 5–10 % of infected individuals; the subsequent disease course is determined by the patient's genetically determined immune status in relation to the pathogen. The lesions are macular, hypochromic and sometimes poorly demarcated. Initially, there is neither erythema nor infiltration.

These hypopigmented macules can occur anywhere on the body.[9] Even in endemic regions, the predominantly young patients are frequently misdiagnosed as having tinea versicolor, pityriasis alba associated with atopic diathesis, vitiligo, or post-inflammatory hypopigmentation associated with eczema. Indeterminate leprosy can last for up to five years. Towards the end of this disease stage, there may be initial signs of subtle neurological deficits such as decreased sweating and/or a loss of thermosensitivity, whereas pain sensitivity is still intact. The onset of these symptoms indicates the transition to a more advanced stage.[10] The present study assessed cases of leprosy.

In present study, out of 48 patients, males were 30 and females were 18. Thakkar et al[11] assessed the

therapeutic efficacy of antileprosy therapy. Two hundred and fifty clinically diagnosed leprosy patients attending skin outdoor patient department (OPD) were included in the study. A total of 250 patients attended the clinic with male to female ratio of 1.7:1. The highest incidence was noted in 17-40 years of age group. In the clinical disease spectrum, 40% patients were in the borderline spectrum followed by tuberculoid leprosy (TT) (29.2%), lepromatous leprosy (LL) (26.8%), and 3.9% of indeterminate leprosy (IL). A total of 18% of patients were of primary neuritic leprosy. A total of 8.3% patients had definite history of contact in the family or neighborhood. Clinicopathological correlation was noted in 60% of patients with maximum disparity (52.9%) in the borderline group of patients. A total of 52.8% were MB (Multibacillary) and 47.2% were PB (Paucibacillary) cases. Morphological index became negative after 6 months in all patients. Mean fall of bacteriological index after 6 months was 0.19, while after 1 year, it was 1.05.

We found that 36 patients were of primary cutaneous and 12 were of primary neuritic, among primary cutaneous most common variety was TT seen in 12 and in primary neuritic was PN seen in 5. Grade 1 deformity was seen in 9 and grade 2 in 4. Lepra reaction type 1 was seen in 3 and type 2 in 8 patients. Jacob and Arunthathi[12], 67% of primary neuritic leprosy patients developed skin lesions on long-term follow-up. Follow-up of the patients with pure neuritic leprosy shows the development of skin lesions in 35% of cases over 3-5 years with or without treatment. This suggests that neuritic symptoms probably are the earliest symptoms of leprosy before development of skin lesions and that is why, patients of pure neuritic leprosy must be followed up for the long term. Deformities were observed in 42.8% of the cases (grade 1-29.2%, grade 2-13.6%). Leprosy, a social stigma, would really mean deformities and disabilities by an affected person. One of the important measures to prevent such impairments is to diagnose the disease early and institute proper treatment promptly. So recent strategy has focused on new case detection with intense screening of deformities.

CONCLUSION

Authors found that histopathological examination is must for confirmation of diagnosis in doubtful cases of leprosy. Maximum case were of primary cutaneous type.

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