Indian J Lepr 2017, 89 : 197-202

http://www.ijl.org.in

Original Article

Leprosy and its Periodontal Manifestations in the Anterior Maxilla

VR Kaipa[1], SP Reddy[2], N Kiran Kumar[3], C Srikanth[4], T Kaveti[5], SR Kosuri[6], M Lohit Reddy[7], S Lingamaneni[8]

Received : 22.03.2017 Accepted : 30.09.2017

Oral health is important for all sections of society, including special groups like leprosy afflicted people. Oral manifestations in leprosy are known, however, most of these afflictions have been observed in pre-chemotherapy / pre-MDT periods. This study has been carried out in 50 leprosy patients, admitted to Government General Hospital Leprosy Wing, Khammam, Telangana and compared with age matched healthy people. Patients within the age range of 30-60 years and diagnosed with leprosy and otherwise systemically healthy are included. The periodontal status of the maxillary anterior sextant (canine to canine) region is studied in both the groups. Clinical parameters measured were - dental plaque index of Silness and Loe and periodontal disease by Ramfjord's index. PPD and CAL was measured using UNC-15 probe and all six sites per tooth were assessed. The mean plaque scores measured in leprosy is 2.62, which is statistically significant (P<0.01) compared to that measured in control groups which is 1.73. Mean probing pocket depth in leprosy patients is much higher than that in controls and most of indoor patients included in this study belong to multi-bacillary forms, these may not reflect the epidemiology of these manifestations. This pilot study just highlights the problem which needs to be studied and managed at the community level.

Key words : Oral Manifestations, Dental Plaque, Leprosy

Introduction

Periodontitis is a host-microbial interaction driven inflammatory disease affecting the supporting tissues of the teeth, ultimately resulting in increased probing pocket depth development, gingival recession or both. Various

[1] VR Kaipa , MDS
[2] SP Reddy, MDS
[3] N Kiran Kumar, MDS
[4] C Srikanth, MDS
[5] T Kaveti, MDS
[6] SR Kosuri, MDS
[7] M Lohit Reddy, MDS
[8] S Lingamaneni, MDS

Department of Peridontics, Mamata Dental College and Government General Hospital, Khammam, Telengana, 507002, India
Corresponding author : Dr. Vinay Ram Kaipa, E-906, Rajapushpa Retreat, Gandipet Road, Near Narsingi Jn., Kokapet, Hyderabad, Telangana-500075, India, **Email:** ramskaipa@gmail.com

systemic effects of periodontal disease and the influence of systemic diseases on the periodontium have been proved. For nearly two thousand years, leprosy is feared by humanity (Suzuki et al 2012). Leprosy is a chronic granulomatous disease caused by the bacteria *Mycobacterium leprae* (Han et al 2008) or *Mycobacterium lepromatosis* (Han et al 2014). The name leprosy is derived from the Latin word lepra meaning scaly. G.A. Hansen identified *Mycobacterium leprae* as the causative agent for leprosy. Hence it is also known as Hansen's disease. Usually the disease has low infectivity and prolonged incubation periods (Ridley and Jopling 1966). It commonly affects the skin, peripheral nervous system, eyes, respiratory tract, etc. If left untreated, may lead to permanent damage including disfigured skin sores, loss of sensation in arms and legs, muscle weakness etc.

According to WHO, 58.85% of new leprosy cases in the world are in India, 1.27 lakhs new cases of leprosy were reported in India during 2013-14. Though India is leading in terms of leprosy cases, several international and national initiatives led to decrease in the number of cases over the past few decades.

WHO classified leprosy into paucibacillary and multibacillary based on bacterial load (Suzuki et al 2012). Paucibacillary has five or fewer and multibacillary has more than five poorly pigmented numb skin patches. Ridley and Jopling have classified leprosy into tuberculoid, lepromatous and borderline (Ridley and Jopling 1966).

Oral manifestations in leprosy are known, albeit rare, have been reported. Leprosy frequently affects the anterior maxilla and often undergoes oppressive destruction in some patients (Waaler 1952). However, most of these afflictions were observed in pre-chemotherapy / pre-MDT periods. Fewer studies reported the periodontal status of patients with leprosy in the recent years. Therefore, we conducted a study in Khammam, Telangana, India to evaluate the periodontal status of patients suffering with leprosy compared to corresponding controls.

Materials and Methods

Location: The study was conducted over a period of three months, from August to October of 2014 and included 50 leprosy patients, under institutionalized treatment at Government General Hospital, Leprosy wing, Khammam, Telangana.

Permissions: The study protocol was approved by the Human Ethics Committee of Mamata Dental College and Hospital, and required permissions were obtained from the Government General Hospital, Khammam. Patients were briefed regarding the said study and informed consent was obtained from all the subjects during examination.

Design and Subjects: The subjects included in the study had an age range of 30-65 years (mean, 61 ± 2.04). All the test group subjects were under standard chemotherapy recommended by our national programme - Rifampicin (600mg/month), Dapsone (100mg/daily) and Clofazimine (300mg/month, 50mg daily) for multibacillary forms and Rifampicin (600mg/month) and Dapsone (100mg/daily) for the paucibacillary forms of the disease. Of the 50 subjects in the study group, about 84% patients (42 patients) had lepromatous leprosy, 12% (6 patients) had the borderline form, and 4% (2 patients) had tuberculoid leprosy. 50 healthy age matched controls in accord with test group, who are not under any medication constituted the control group.

Of the institutionalized leprosy patients, majorities were males and only 3 female patients were available. So, we included only male patients in

the study who were matched to control group, which also consisted males.

Inclusion criteria: Patients within the age range of 30-60 years and diagnosed with leprosy, any of the three forms - Lepromatous, Borderline (Borderline tuberculoid-BT / Borderline borderline - BB/ Borderline lepromatous - BL) and Tuberculoid, these cases were otherwise systemically healthy.

Exclusion criteria: Patients with a history of oral prophylaxis or any other dental treatment in the past 6 months, usage of antibiotics in the past 6 months (except antileprotic drugs), smokers and history of any other systematic disease.

Following an initial oral examination, the periodontal status of the maxillary anterior sextant (canine to canine) region was done, in both the groups. Clinical parameters measured were, dental plaque index by Silness and Loe; and periodontal disease by Ramfjord index. PPD and CAL was measured using UNC-15 probe and all six sites per tooth were assessed (Costa et al 2009).

Statistical analysis: To determine the differences between groups, Student's t-test and ANOVA test were used. SPSS version 20 software was used to analyze the results. P-values ≤ 0.01 is considered as significant.

Results

Statistically significant differences (P< 0.01) were seen in all the assessed parameters between the study and control groups.

The plaque scores: The mean plaque scores measured using Silness & Loe plaque index in Leprosy was 2.62, which is statistically significant (P< 0.01) compared to that measured in control groups which was 1.73. (Table 1)

Probing pocket depth & Clinical attachment loss scores: There were statistically significant differences in the measured probing pocket depths and the clinical attachment loss than in controls. A mean probing depth of 4.14 and an average clinical attachment loss of 4.82 are seen in leprotic group which is significantly higher than

Table 1 : Comparison of and Plaque index in leprotic and control groups (Loe & Silness Plaque index)

Group	N	Mean	Std. Deviation	Std. Error Mean
Leprosy	50	2.62	0.52	0.073
Control	50	1.73	0.52	0.073

T= 8.55, P< 0.01

Table 2 : Comparison of Probing pocket depth (PPD) in leprotic and control groups

Group	N	Mean	Std. Deviation	Std. Error Mean
Leprosy	50	4.14	1.19	0.16
Control	50	3.31	0.002	0.14

T= 3.37, P< 0.01

Table 3 : Comparison of Clinical attachment loss (CAL) in leprotic and control groups

Group	N	Mean	Std. Deviation	Std. Error Mean
Leprosy	50	4.82	1.16	0.164
Control	50	4.03	1.31	0.1853

T= 3.19, P< 0.01

Table 4 : Intra group comparison within the leprotic group (ANOVA)

Group	N	Mean	Std. Deviation	Std. Error Mean
Loe & Silness plaque index	50	2.62	0.52	0.07
Probing pocket depth (PPD)	50	4.14	1.19	0.16
Clinical attachment loss	50	4.82	1.16	0.16

F= 62.77, P< 0.01

in the control group (P< 0.01) which had a mean probing depth of 3.31 and an average clinical attachment loss of 4.03. (Tables 2 , 3)

The intergroup analysis for the measured values showed statistically significant (P< 0.01) results. (Table 4)

Since the number of individuals with the different forms of leprosy was small; the correlation was not analyzed and the entire group was compared with the controls.

Discussion

Periodontal disease is known to be caused by a myriad of host and microbial interactions. Host microbial interaction leads to the instigation and development of periodontal disease (Keith et al 2000). Thus, the resultant mounted host response varies which results in varied tissue destruction. Leprosy in a similar manner has a varied clinical response due to differences in host immune response (Pinheiro et al 2011). Other common features like chronicity and involvement of cytokines in the pathogenesis of the diseases are seen both in leprosy and chronic periodontitis (Aravindhan et al 2014).

Relationship has been reported to exist between the chronic diseases such as leprosy and periodontitis (Reichart et al 1976). Leprosy can manifest as chronic periodontitis in the oral cavity (Sheskin 1973). Maxillary central incisors seem to be affected more in leprotic patients with gingiva, uvula, hard and soft palate being the most commonly effected sites (Girdhar and Desikan 1979, Bombach and Reichart 1987). *M. leprae* favors temperatures a little below the body temperature for multiplication (Shereef 1992). Based on this lower temperature affinity of *M. lepra* (Scheepers et al 1998) postulated that the mean surface temperature changes are responsible for the differential involvement of the various site in the oral cavity. Frequency of involvement is inversely related to the surface temperature; lower the temperature, higher the frequency of involvement (Barton 1974). In patients with lepromatous leprosy, nasal obstruction and stuffiness are common which leads to mouth breathing causing surface temperature to be low over the dorsum of the tongue, hard palate and the soft palate (Shereef 1992, Barton 1974 and Sharma et al 1985). This leads to an increased amount of bone destruction in the anterior maxilla. So, in our study we evaluated the periodontal status of anterior maxillary teeth in leprotic patients.

Moller - Christensen, a Danish medical historian, paleopathogist & osteoarcheologist and his colleagues (Moller - Christensen et al 1952), termed the facial changes resulting from typical bone alterations as *facies leprosy* (Brasil et al 1973). A triad of lesions described to be associated with facial leprosy, which together constitutes a syndrome (Barton 1974 and Brasil et al 1973). Facies leprosy includes atrophy of anterior nasal spine, atrophy and recession of the alveolar processes of the premaxilla, and endonasal inflammatory changes.

Also the gingiva that is usually affected is the area behind the upper central incisors, and it synapses with the lesions of the hard palate (Reichart 1976). Hard palate shows the most varied type of

lesions. The disease may present as erythematous or reddish papules which gradually increase in size and number and coalesce to form a generalized nodular submucosal infiltrate. As the disease progresses, the mucosa loses its shininess and gives a matt like appearance (Mukherjee et al 1979). Occasionally the nodules of the palate ulcerate leading to palatal perforation (Prabhu and Daftary 1981). Even though serious changes in the mid-forward portions of the hard palate are usually seen, some have found the soft palate to the be more commonly affected area (Handa et al 2003).

Fibrosis with partial loss or at times complete destruction of uvula might be seen in extreme cases of leprosy (Moller-christensen et al 1952). Tongue displays *cobble stone appearance* with the presence of nodules. Leprotic patients show fissuring of tongue which is a unique feature (Scheepers 1998).

Leprous involvement of the lips can occur, which may present as macrocheilia, presence of flat topped nodules and microstomia (Souza et al 2009). Handa et al (2003) in his study on chronic macrocheilia, observed leprous macrochelia in 10.7% of the 28 patients screened (Aravindhan et al 2014).

Our study comprised of systemically healthy individuals as controls and cases who are positive for *M. leprae* as the study group. Since the number of individuals in the study group with different types of leprosy was small and as mentioned earlier, the number of female patients screened was also very less, only male patients with any of the forms of leprosy were taken; correlation was not made with different types of leprosy and the entire group (cases) consisted of male patients was compared against controls. As detailed in the results, the mean plaque scores found in the leprosy patients were 2.62, which is higher and statistically significant than 1.73 that is found in the controls. Even the mean probing pocket depth in leprosy patients was much higher than that in controls and is in accordance with the study done by Souza et al (2009). Clinical attachment level found in leprosy cases was also greater and statistically significant than controls. These results are in accordance with a study conducted by Aravindhan et al (2014). Our study results showed a positive correlation between leprosy and periodontitis which is in accordance with the study done by Núñez-Martí et al (2004). It will be important to emphasize that most of indoor patients included in this study belong to multi-bacillary forms; these may not reflect the epidemiology of these manifestations. Further, the delay in starting the treatment and other complications like reactions may also be important compounding factors. All these issues need to be investigated in population based studies.

For a periodontist, awareness about the orofacial manifestations of leprosy is imperative and the required precautions to be taken during the administration of treatment to such patients. Early diagnosis and treatment of the oral lesions is better, as the oral manifestations are usually an expression of advanced involvement of the disease.

Test group patients showed greater probing pocket depths and plaque indices in comparison with the control group. Leprotic effects such as altered tongue and masticatory muscles, finger mutilation, masticatory defects, gingival sensitivity problems lead to poor oral hygiene maintenance ultimately resulting in periodontitis.

Acknowledgements

We express our gratitude to Dr. Aditya, Prof., Dept of Community Medicine, Mamata Medical College, for helping us with the statistics.

References

1. Aravindhan TR, Waleed K, Ponnandai KS et al (2014). Periodontal findings in patients with Hansen's disease. *Asian Pac J Trop Med.* **7**: S108-S110.

2. Barton RP (1974). Lesions of mouth, pharynx and larynx in lepromatous leprosy. *Lepr Rev*. **46**: 130.
3. Bombach B, Reichart P (1987). Periodontal findings in patients with leprosy. *Leprosy Review.* **58**: 279-89.
4. Brasil J, Opromolla DV, Freitas JA, Rossi JE (1973). Histologic and bacteriologic study of lepromatous lesions of the oral mucosa. *Estomatol Cult.* **7**: 113-9.
5. Costa FO, Guimarães AN, Cota LOM, Pataro AL, Segundo K, Cortelli SC (2009). Impact of different periodontitis case definitions on periodontal research. *J Oral Sci*. **51**: 199-206.
6. Girdhar BK, Desikan KV (1979). A clinical study of the mouth in untreated lepromatous patients. *Lepr Rev.* **50**: 25-35.
7. Han XY, Fleur MA, Siew EC, Betina W (2014). Analysis of the Leprosy Agents *Mycobacterium leprae* and *Mycobacterium lepromatosis* in Four Countries. *Am J Clin Pathol.* **142**: 524-32.
8. Han XY, Seo Y-H, Sizer KC (2008). A new *Mycobacterium species* causing diffuse lepromatous leprosy. *Am J Clin Pathol.* **130**: 856-64.
9. Handa S, Saraswat A, Radotra BD, Kumar B (2003). Chronic macrocheilia: A clinicopathological study of 28 patients. *Clin Exp Dermatol*. **28**: 245-50.
10. Keith LK, Joni AC, Jill ER, William VG (2007). Novel host response therapeutic approaches to treat periodontal diseases. *Periodontology 2000.* **43**: 294-315.
11. Moller-Christensen V, Bakke SN, Melsom RS, Waaler E (1952). Changes in the anterior nasal spine of the alveolar process of the maxillary bone in leprosy. *Int J Lepr*. **20**: 335-40.
12. Mukherjee A, Giridhar BK, Desikan KV (1979). Histopathology of tongue lesions in leprosy. *Lepr Rev.* **50**: 37-43.
13. Núñez-Martí JM, Bagán JV, Scully C, Peñarrocha M (2004). Leprosy: dental and periodontal status of the anterior maxilla in 76 patients. *Oral Dis*. **10**: 19-21.
14. Pinheiro RO, de Souza Salles J, Sarno EN, Sampaio EP (2011). *Mycobacterium leprae* - host-cell interactions and genetic determinents in leprosy: an overview. *Future Microbiol.* **6**: 217-30.
15. Prabhu SR, Daftary DK (1981). Clinical evaluation of orofacial lesions of leprosy. *Odontostomatol Trop.* **4**: 83-95.
16. Reichart P, Ananatasan T, Reznik G (1976). Gingiva and periodontium in lepromatous leprosy: a clinical, radiological, and microscopic study. *J Periodontol*. **47**: 455-60.
17. Reichart P (1976). Facial and oral manifestations in leprosy. An evaluation of seventy cases. *Oral Surg Oral Med Oral Pathol.* **41**: 385-9.
18. Ridley DS, Jopling WH (1966). Classification of leprosy according to immunity: a five-group system. *Int J Lepr.* **34**: 255-73.
19. Scheepers A (1998). Correlation of Oral Surface Temperatures and The Lesion In Leprosy. *Int J Lepr.* **66**: 215-17.
20. Sharma VK, Kumar N, Kaur S (1985). Involvement of tongue in leprosy. *Indian J Lep.* **57**: 841- 44.
21. Shereef PH (1992). Hypopigmented macules in leprosy – A histopathological and histochemical study of melanocytes. *Indian J Lepr*. **64**: 189-91.
22. Sheskin J, Hermal J, Evan TJ (1973). Taste sensation in leprosy. *Int J Lepr Other Mycobact Dis.* **41**: 580.
23. Souza VA, Emmerich A, Coutinho EM et al (2009). Dental and oral condition in leprosy patients from Serra, Brazil. *Lepr Rev.* **80**: 156-63.
24. Suzuki K, Akama T, Kawashima A et al (2012). Current status of leprosy: epidemiology, basic science and clinical perspectives. *J Dermatol.* **39:** 121-9.
25. Waaler E (1952). Changes in the maxillary bone in leprosy. *Int J Lepr*. **20**: 335-40.

How to cite this article : Kaipa VR, Reddy SP, Kumar NK et al (2017). Leprosy and its Periodontal Manifestations in the Anterior Maxilla. *Indian J Lepr.* **89** : 197-202.