ORIGINAL ARTICLE

Evaluation of the orofacial lesions in treated leprosy patients

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ABSTRACT

Background: Leprosy is primarily a disease of developmental countries. About 4 million people have or are disabled by leprosy. Eighty-six percent of leprosy patients reside in Southeast Asia and Brazil. India accounts for up to 70% of total cases. **Aim:** To evaluate the incidence of orofacial lesions in treated leprosy patients. **Materials and Methods:** Thirty treated leprosy patients were examined clinically and the percentage of orofacial lesions were evaluated. **Results:** On evaluating the orofacial lesions, incidence of hypopigmentation on face and oral mucosa were highest (63%) followed by depressed nasal bridge and fissured tongue (33%). The incidence of crenated tongue was seen to be the lowest (3.3%). **Conclusion:** Orofacial lesions in leprosy patients develop insidiously, generally are asymptomatic and are secondary to nasal changes. Oral lesions may contribute to the diagnosis of the disease and be attributed to involvement of *Mycobacterium leprae*.

Key words: Leprosy, multibacillary, orofacial lesions, paucibacillary

INTRODUCTION

Leprosy also known as Hansen's disease is a chronic multi-system disease caused by *Mycobacterium leprae* (Hansen's bacillus). It is a transmissible infectious disease mainly affecting the skin, peripheral nerves and mucous membrane.

The World Health Organization (WHO) classification (1997) is based on the number of skin lesions and identifies two broad categories: Paucibacillary (PB) disease (one to five lesions) and multibacillary (MB) disease (six or more lesions). PB leprosy is characterized by the less number of skin lesions which includes tuberculoid leprosy and borderline tuberculoid leprosy. PB is further divided to single lesion PB (SLPB) and PB (with two to five lesions). MB includes borderline leprosy, borderline lepromatous leprosy and lepromatous leprosy. MB exhibits ill-defined, hypopigmented macules or papules on the skin and mucous membrane.[1]

Approximately 600,000 new cases of leprosy are detected every year. Male are affected more than female with 2:1 ratio. Usually occurs at young age. Median age of onset is 2–5 years for tuberculoid form and 8–12 years for lepromatous form. Up to 19–60% of patients have been reported with lesions involving the oral cavity.[2] As described by Pinkerton in 1932, the early oral manifestations include congestion, infiltration and formation of nodules; ulceration, atrophies and fibrosis. Facial manifestations include macules, hypopigmentation, lepromatous formation, distorted facial appearances or skeletal deformities with loss of eyebrows and eye lashes which is known as facies leonine. Sensory loss due to involvement of the nerves is also seen. In advance stages; oral mucosa presents ulceration, congestion, infiltration, atrophy and scarring. Lesions of lips include microchelia followed by microstomia. Intense fibrosis may be seen in uvula.[3] Very few studies are there in the literature regarding the incidence of orofacial lesions. Thus, the present study was carried out to evaluate the incidence of orofacial lesions in treated leprosy patients.

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MATERIALS AND METHODS

The study group included 30 treated leprosy patients from the leprosy colony at Raichur. Patients who were not under treatment were excluded from the study. The patients were examined extra and intraorally for lesions; observations included depressed nasal bridge, hypopigmented macules, abnormalities of tongue, shrunken uvula, eruptions of the buccal mucosa, lesions in lips and microstomia. After recording the finding, percentage of orofacial lesions were calculated.

RESULT

All the 30 leprosy patients examined presented as a MB form of leprosy. The average ages of the whole sample ranged from 35 to 50 years and among them16 were males and 14 were females. All the patients were under multidrug regime therapy (MDT) as per the World Health Organization (WHO) guidelines.

Among 30 leprosy patients examined, 19 patients (63.3%) presented with hypopigmentation on face and oral mucosa [Figure 1], 10 patients (33.3%) presented with depressed nasal bridge [Figure 2] and fissured tongue [Figure 3], four patients (13.3%) presented with depapillated tongue [Figure 4], coated tongue [Figure 5], shrunken uvula [Figure 6], lesions in lips [Figure 7], and microstomia. Other findings observed were ulcerations on buccal mucosa (10%) and crenated tongue (3.3%) [Table 1].

DISCUSSION

Leprosy was first described in the ancient Indian texts from the 6th century BC, as a nonfatal, chronic infectious disease caused by *M. leprae*. It primarily affects the skin, peripheral nerves, respiratory system, eyes and patient's immune response. Cell-mediated immunity is considered to be crucial defense against the disease and the magnitude of this immunity defines the extent of the disease.[3] Orofacial lesions in leprosy patients

Table 1: Incidence and percentage of orofacial lesions

Orofacial lesions	No. of patients	Percentage
Depressed nasal bridge	10	33.3
Hypopigmentation on face and oral mucosa	19	63.3
Tongue		
Fissured	10	33.3
Depapillated	4	13.3
Coated	4	13.3
Crenated	1	3.3
Shrunken uvula	4	13.3
Eruptions on the anterior pillars, uvula, palate, and buccal mucosa	5	16.7
Ulceration of buccal mucosa	3	10.0
Lesions on lips	4	13.3
Microstomia	4	13.3

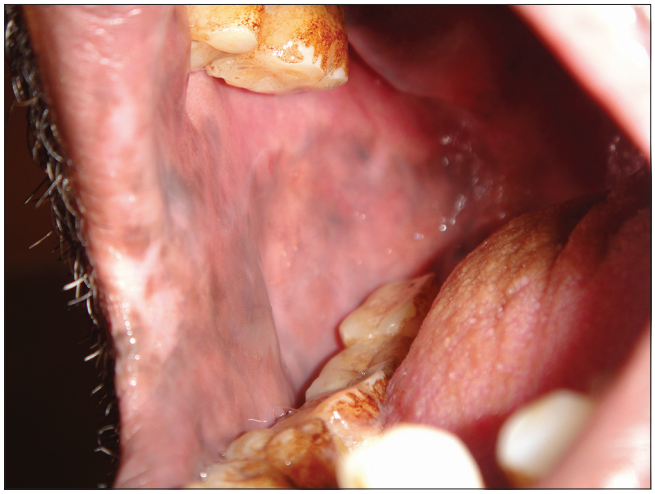

Figure 1: Hypopigmentation of oral mucosa

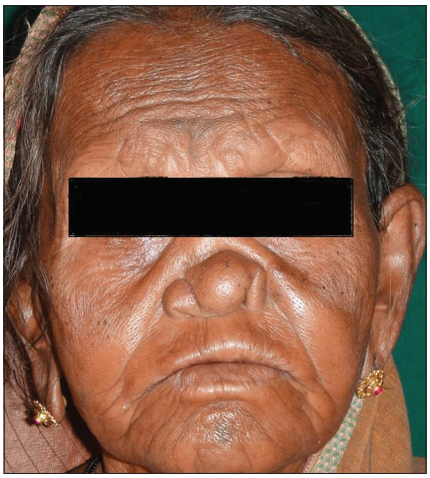

Figure 2: Depressed nasal bridge

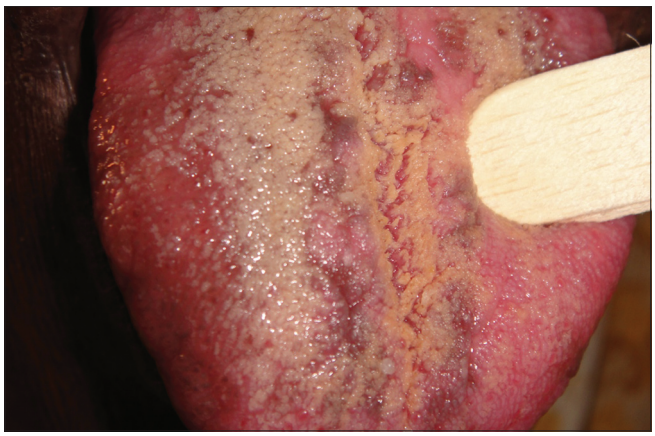

Figure 3: Fissured tongue

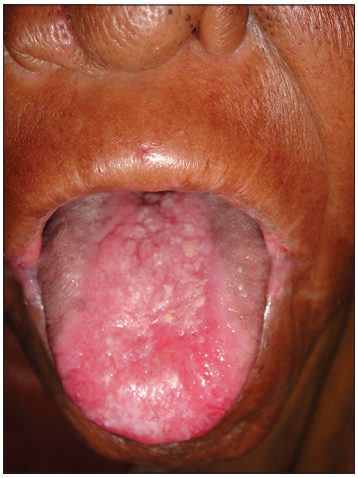

Figure 4: Depapillated tongue

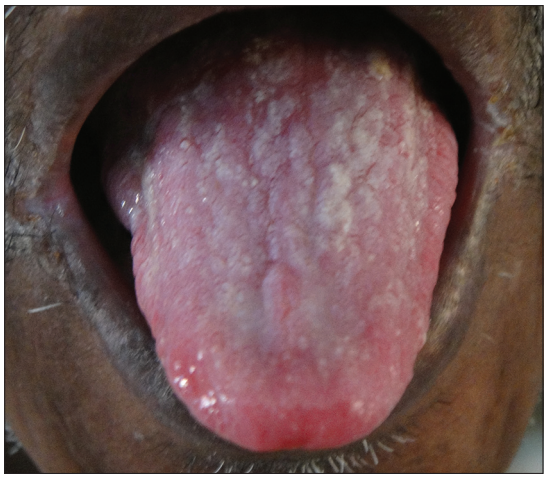

Figure 5: Coated tongue

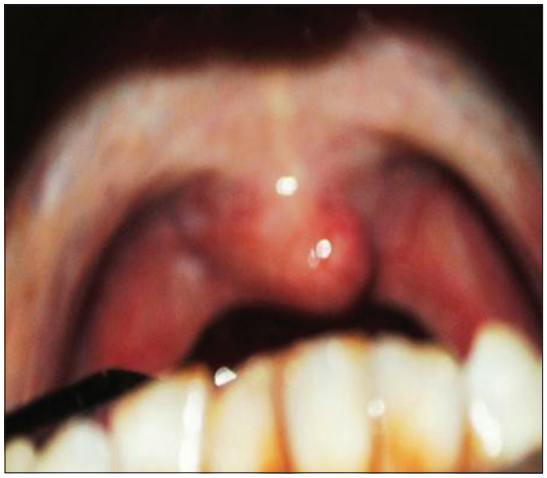

Figure 6: Shrunken uvula

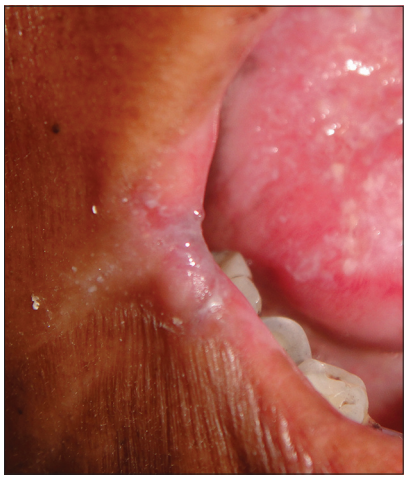

Figure 7: Lesions in lip

are rare and develop insidiously, generally are asymptomatic and are secondary to nasal changes.

In our study, the commonly observed feature was hypopigmentation of skin and mucosa, depressed nasal bridge and fissured tongue. However, the least observed was crenated tongue. Other findings were ulceration on mucosa, lesions on lips, microstomia, eruptions on the buccal mucosa depapillated and coated tongue. The incidence of these orofacial lesions in our study was in accordance with Rawalani *et al*., (2008).[2]

The previous studies conducted by Reichart *et al*.,(1976)[4] Hubscher *et al*., (1979)[5] Bucci *et al*., (1987)[6] and Kumar *et al*., (1988)[7] showed that the hard and soft palate were most frequently affected. Recent studies conducted by Santos *et al*., (2000)[8] and Martins *et al*., (2007)[9] showed no characteristic oral lesions in leprosy patients undergoing treatment. The gradual reduction of oral lesions in the recent studies and present study may be attributed to the efficacy of the MDT along with early diagnosis of the disease.

The changes are attributed to involvement of *M. leprae*. Studies have shown the presence of leprae bacilli in oral smears as a viable source of infection in leprosy.[10] *M. leprae* prefers temperature less than body temperature for its living. Considering this fact, a patho-physiologic mechanism is postulated for oral involvement: A nasal lesion with obstruction leads to mouth breathing; this causes decrease in the intra oral temperature harboring bacilli for multiplication.[10,11] This may in turn lead to ulceration and necrosis of the soft tissue which heals by secondary intention. In advanced cases of leprosy, mouth can acquire the characteristics of being a reservoir for the bacilli. This may act as an important risk factor for transmission of the disease.[12] Thus, the patient's oral cavity has to be examined thoroughly.

CONCLUSION

The molecular and immunological studies have shown that oral mucosa may be a secondary source of infection of leprae bacilli, nasal cavity being the primary. In the present study we observed decreased number of orofacial lesions which may be due to early diagnosis and usage of MDT. Though multi-drug therapy is able to clear *M. leprae* effectively, a small number of persisting organism may still remain. The persisting organisms could act as a causative factor for the oro-facial lesions in leprosy patients. Hence, research in this aspect is further required to know the exact causative factor for mucosal and skeletal alterations.

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