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. 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.[1] 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.

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 them 16 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

| 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       |

Table 1: Incidence and percentage of orofacial lesions

Figure 1: Hypopigmentation of oral mucosa

Figure 2: Depressed nasal bridge

Figure 3: Fissured tongue

Figure 4: Depapillated tongue
Orofacial lesions in treated leprosy patients

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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 lepra 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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