IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 14, Issue 6 Ver. VI (Jun. 2015), PP 77-79
www.iosrjournals.org

Orofacial Manifestations of Leprosy: A Review

Ahmed Hassan Kamil Mustafa
LECTURER IN Alfarabi medical colleges Riyadh, KSA
BDS, MFDRCSI, MSC.oral andmaxillofacial surgery

Abstract:
Background*: Leprosy is a chronic, non-fatal disease caused by Mycobacterium leprae. It can cause cutaneous lesions, peripheral nerve lesions and orofacial manifestations, including destruction of the alveolar premaxillary process associated with loss of the maxillary incisors.*
Objective:
The aims of this study were to highlight theorofacial manifestations of disease in published papers in pubmed.
Method*: Computerized search of published papers (2004 - 2015) in pubmed data base, using specific words such as orofacial features of leprosy, in English language.*
Result*: following computerized search in pubmed , about 38 articles about orofacial manifestations of leprosy were found in pubmed. Only 14 article were selected which conform to our criteria.*
Conclusion: *The orofacial manifestations of leprosy are well documented in literature and tend to occur in 20 – 60% of cases of leprosy and are more frequent in lepromatous leprosy.*
In conclusion, examination of leprosy patients should be extended to the oral mucosa because oral mucosa may be a secondary source of M.leprae transmission and infection.

I. Introduction

Leprosy is an infectious chronic disease with a wide range of clinical and serological manifestations(1).

Leprosy (Hansen's disease) is a chronic granulomatous disease caused by Mycobacterium leprae (Hansen's bacillus). Oral manifestations are reported to occur in 20-60% of cases, more common inlepromatous leprosy, and are well documented. They may involve both the oral hard and soft tissues(2).

Clinical presentations of leprosy are related to the immune response against M. Leprae. The first, tuberculoid leprosy(paucibacillary) that is characterized by high immune reaction to the organism, a few cutaneous lesions and little number of bacilli in skin biopsy specimens. . The second, lepromatous leprosy (multibacillary; LL), that usually develops in patients with reduced cell-mediated response and negative lepromin skin tests (3,4,5). Within this spectrum there are borderline and less common variations with intermediate lesions. The main clinical features of leprosy are listed as follows: erythematous or hypopigmented macules on the skin with reduced sensation, distinct sensory loss and peripheral nerve involvements, muscle weakness, nerve thickening, positive skin biopsy of acid – fast bacilli and positive skin smear. It is noted that a subject with one or two of these major findings should be considered as a leprosy case (6). It is transmitted from person to person and has a long incubation period (between two and six years).

Oral manifestations usually appear in lepromatous leprosy and occur in 20-60% of cases. They may take the form of multiple nodules (lepromas) that progress to necrosis and ulceration. The ulcers are slow to heal, and produce atrophic scarring or even tissue destruction. The lesions are usually located on the hard and soft palate, in the uvula, on the underside of the tongue, and on the lips and gums. There may also be destruction of the anterior maxilla and loss of teeth(7). The diagnosis, based on clinical suspicion, is confirmed through bacteriological and histopathological analyses, as well as by means of the lepromin test (intradermal reaction that is usually negative in lepromatous leprosy form and positive in the tuberculoid form). The differential diagnosis includes systemic lupus erythematosus, sarcoidosis, cutaneous leishmaniasis and other skin diseases, tertiary syphilis, lymphomas, systemic mycosis, traumatic lesions and malignant neoplasias, among other disorders. Treatment is difficult as it must be continued for long periods, requires several drugs with adverse effects and proves very expensive, particularly for less developed countries. The most commonly used drugs are dapsone, rifampicin and clofazimine. Quinolones, such as ofloxacin and pefloxacin, as well as some macrolides, such as clarithromycin and minocyclin, are also effective(7).

Objective

The aims of this study were to highlight the orofacial manifestations of disease in published papers in pubmed.

Method: Computerized search of published papers (2004 - 2015) in pubmed data base, using specific words such as orofacial features of leprosy, in English language.

Result: following computerized search in pubmed , about 38 articles about orofacial manifestations of leprosy were found in pubmed. Only 14 article were selected which conform to our criteria.

II. Discussion

In this present review study we tried to evaluate the orofacialfeatures of leprosy**.** The upper airways are the most important point of entry for bacillus and a main source for bacillary elimination in leprosy (4,8). In recent studies, oral mucosa seems to be the secondary (after nasal mucosa) site of M. Leprae infection and transmission (9,3). Also, involvement of oral mucosa may have a key role in leprosy transmission from adults to children (10). Indeed, it is established that these lesions tended to be more frequent over the first 5 years of the disease (11,12). For these reasons, evaluation of oral cavity should be routine for leprosy patients (3,4).

Prabhu et al.(14), in an excellent review of 700 leprosy patients showed that the prevalence of oral involvement in leprosy was 11.5% and also these lesions tended to occur in lepromatous leprosy. Some authors such as Motta et al.(4), Soni (14) and Palaskar (15) established that the oral lesions usually appear in the advanced stage of leprosy.In contrast to this hypothesis, de Abreu et al.(3), Brasil et al.(16) and Sharma et al.(17) demonstrated that oral lesions may occur in less advanced stages of the disease and also in these patients oral cavity involvement remains clinically hidden (absence of visible lesions) and may only detected histopatholosically. This can be one of the main explanations for different rates of oral involvement in leprosy seen in literature (3).It is suggested that oral involvement in leprosy occurs by hematogenous or lymphatic dissemination of M. Leprae (4,18). On the other hand, Bucci et al.(19) and Girdhar et al.(20) pointed out that nasal lesions may be precursors of oral lesions.De Abreu et al.(21), demonstrated that paucibacillary leprosy patients do not exhibit clinical or subclinical, involvement in the oral cavity. . The distribution of oral lesions in anotherstudy(1) are ranked as follows: soft palate, tongue (posterior one – third), tongue (anterior two –third), lips, uvula, tonsil, hard palate. According to WHO, the most affected sites of oral cavity in leprosy patients are: hard palate, soft palate, labial maxillary gingival and buccal mucosa (22).

Motta et al.(4), Brasil et al.(16) and Reichart (23) showed that the soft palate was affected most frequently in almost all cases of leprosy. Therate of facial involvemengt in leprosy was 28% in Prabhu's study (13).In another study the rate of facial involvement was reported to be 39% (1).Atrophy of nasal spine was not found in patients with tuberculoid leprosy (1).

III. Conclusion

The orofacial manifestations of leprosy are well documented in literature and tend to occur in 20 – 60% of cases of leprosy and are more frequent in lepromatous leprosy. In conclusion, examination of leprosy patients should be extended to the oral mucosa because oral mucosa may be a secondary source of M.leprae transmission and infection.

Auther	**Year**	**Type of study**
Horta et al	2015	Case report
Dhillon et al	2013	Case report
Ali et al	2011	Case report
Martinez et al	2011	Experimental study
Souza et al	2009	review
Motta et al	2008	Case report
De Abrue et al	2006 -2007	Experimental study
Chimenos et al	2006	Case report
Palaskar et al	2005	Case report
Boggild et al	2004	Prospective study
Meima et al	2004	
Aroujo et al	2003	review
Bhattacharya et al	2002	Systematic review
Scheepers et al	1998	Experimental study
WHO	1998	report
Sharma	1993	Case report
Soni	1992	Case report
Bucci	1987	review
Prabhu	1981	Case report
Girdhar	1979	Case series
Brasil	1974	Experimental study
Reichart	1974	Case series

Table: containing the names of the authers , the year , and the type of study

References

[1]. Horta-Baas G1, Hernández-Cabrera MF2, Barile-Fabris LA3, Del S Romero-Figueroa M4, Arenas-Guzmán R5.Multibacillary leprosy mimicking systemic lupus erythematosus: case report and literature review. Lupus. 2015 Mar 10. pii: 0961203315574557. [Epub ahead of print]

[2]. Dhillon M1, Mohan RS, Raju SM, Krishnamoorthy B, Lakhanpal M. Hansen's disease; Leprosy; mimicking; misdiagnosis; systemic lupus erythematosus. Lepr Rev. 2013 Jun;84(2):151-7.

[3]. de Abreu MA, Michalany NS, Weckx LL, Neto Pimentel DR, Hirata CH, de AvelarAlchorne MM. The oral mucosa in leprosy: a clinical and histopathological study. Braz J Otorhinolaryngol. 2006;72:312–6. [PubMed].

[4]. Motta AC, Komesu MC, Silva CH, Arruda D, Simão JC, Zenha EM. Leprosy-specific oral lesions: a report of three cases. Med Oral Patol Oral Cir Bucal. 2008;13:E479–82. [PubMed]

[5]. Ali K, Sittampalam G, Malik MA. Facial tuberculoid leprosy: case report. Br J Oral Maxillofac Surg. 2011;49:70–2. [PubMed]

[6]. Bhattacharya SN, Sehgal VN. Reappraisal of the drifting scenario of leprosy multi-drug therapy: new approach proposed for the new millennium. Int J Dermatol. 2002;41:321–6. [PubMed]

[7]. ChimenosKüstner E1, Pascual Cruz M, PinolDansis C, Vinals Iglesias H, Rodríguez de Rivera Campillo ME, LópezLópez J.Lepromatous leprosy: a review and case report. Med Oral Patol Oral Cir Bucal. 2006 Nov 1;11(6):E474-9.

[8]. Araújo MG. Leprosy in Brazil. Rev Soc Bras Med Trop. 2003;36:373–82. [PubMed].

[9]. Martinez TS, Figueira MM, Costa AV, Gonçalves MA, Goulart LR, Goulart IM. Oral mucosa as a source of Mycobacterium leprae infection and transmission, and implications of bacterial DNA detection and the immunological status. ClinMicrobiol Infect. 2011;17:1653–8. [PubMed]

[10]. Boggild AK, Correia JD, Keystone JS, Kain KC. Leprosy in Toronto: an analysis of 184 imported cases. CMAJ. 2004;170:55–9. [PMC free article] [PubMed].

[11]. Meima A, Richardus JH, Habbema JD. Trends in leprosy case detection worldwide since 1985. Lepr Rev. 2004;75:19–33. [PubMed].

[12]. Souza VA, Emmerich A, Coutinho EM, Freitas MG, Silva EH, Merçon FG. Dental and oral condition in leprosy patients from Serra, Brazil. Lepr Rev. 2009;80:156–63. [PubMed].

[13]. Prabhu SR, Daftary DK. Clinical evaluation of oro-facial lesions of leprosy. Odontostomatol Trop. 1981;4:83–95. [PubMed].

[14]. Soni NK. Leprosy of the tongue. Indian J Lepr. 1992;64:325–30. [PubMed].

[15]. Palaskar S. Histopathological study of apparently normal oral mucosa in lepromatous leprosy. Indian J Dent Res. 2005;16:12–4. [PubMed].

[16]. Brasil J, de AraújoOpromolla DV, de Souza-Freitas JA, Fleury RN. Pathological alterations in oral mucosa of lepromatous patients. Estomatol Cult. 1974;8:137–52. [PubMed].

[17]. Sharma VK, Kaur S, Radotra BD, Kaur I. Tongue involvement in lepromatous leprosy. Int J Dermatol. 1993;32:27–9. [PubMed].

[18]. Scheepers A. Correlation of oral surface temperatures and the lesions of leprosy. Int J Lepr Other Mycobact Dis. 1998;66:214–7. [PubMed]

[19]. Bucci F Jr, Mesa M, Schwartz RA, McNeil G, Lambert WC. Oral lesions in lepromatous leprosy. J Oral Med. 1987;42:4–6. [PubMed]

[20]. Girdhar BK, Desikan KV. A clinical study of the mouth in untreated lepromatous patients. Lepr Rev. 1979;50:25–35. [PubMed]

[21]. de Abreu MA, Alchorne MM, Michalany NS, Weckx LL, Pimentel DR, Hirata CH. The oral mucosa in paucibacillary leprosy: a clinical and histopathological study. Oral Surg Oral Med Oral Pathol Oral RadiolEndod. 2007;103:e48–52. [PubMed].

[22]. WHO Expert Committee on Leprosy. World Health Organ Tech Rep Ser. 1998;874:1–43. [PubMed].

[23]. Reichart P. Pathologic changes in the soft palate in lepromatous leprosy. An evaluation of ten patients. Oral Surg Oral Med Oral Pathol. 1974;38:898–904. [PubMed]