Leprosy-specific oral lesions: A report of three cases

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Abstract

Leprosy is a chronic infection caused by *Mycobacterium leprae*, a bacillus that presents a peculiar tropism for the skin and peripheral nerves. The clinical spectrum of leprosy ranges from the tuberculoid form (TT) to the disseminative and progressive lepromatous form (LL). Oral lesions are rare but, when present, occur in the lepromatous form. This article describes the clinical and microscopic findings of three cases of LL with oral manifestations. All patients had the lepromatous form and their leprosy-specific oral lesions occurred in the palate. The diagnosis was based on clinical, serological and histopathological findings, and multidrug therapy for multibacillary leprosy was started and continued for 24 months. All patients completed treatment, but developed reaction episodes which were treated with prednisone and/or thalidomide. The authors emphasize the importance of oral mucosa evaluation by a dental health professional during patient care since oral lesions may act as a source of infection.

Key words: *Leprosy, lepromatous leprosy, Mycobacterium leprae, oral lesions.*

Introduction

Leprosy is a chronic infectious disease caused by *Mycobacterium leprae*, an acid-fast bacillus that presents a peculiar tropism for the skin and peripheral nerves. Leprosy is still a public health problem in many parts of the world. In 2006, the number of new cases detected fell globally when compared with 2005. However, Brazil still represents an important part of the global burden of the disease in the Americas (1).

Leprosy presents a clinical spectrum ranging from the tuberculoid form (TT), with lesions that are often self-healing, to the disseminate and progressive lepromatous form (LL). Within this spectrum there are borderline forms with intermediate lesions between the 2 polar forms (2). Oral lesions are uncommon in leprosy but when present occur in patients with the LL form (3-8). These lesions are generally asymptomatic ulcers or nodules sometimes rich in *M. leprae* (6,9,10) resembling nonspecific oral lesions. However they can maintain the focus of infection in endemic areas.

This article describes the oral lesions of leprosy of 3 LL patients, and alerts physicians to the importance of oral mucosa evaluation during patient care.

Case Reports

- *Patients.* The demographic data, clinical information, laboratory features, treatment and outcome of 3 patients with leprosy with oral manifestations were reviewed and are described here. All cases were retrieved from the files of the University Hospital of the Faculty of Medicine of Ribeirao Preto, Sao Paulo University (Brazil).

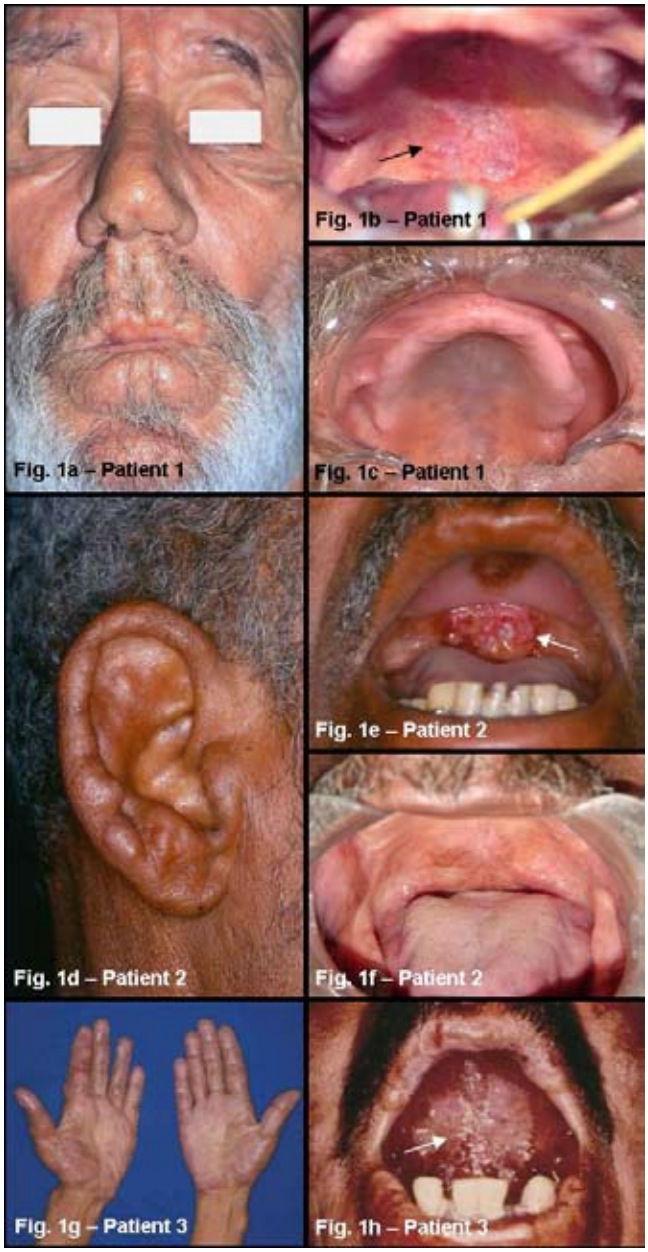

Fig. 1. Clinical presentation of patients: skin infiltration giving rise to the leonine facies (1a); oral ulcer in the hard and soft palate (1b and 1h); resolution of oral lesions after MDT (1c and 1f); nodules on the ear (1d); ulceration with granulomatous surface in the soft palate (1e); papules and nodules on the hands (1g).

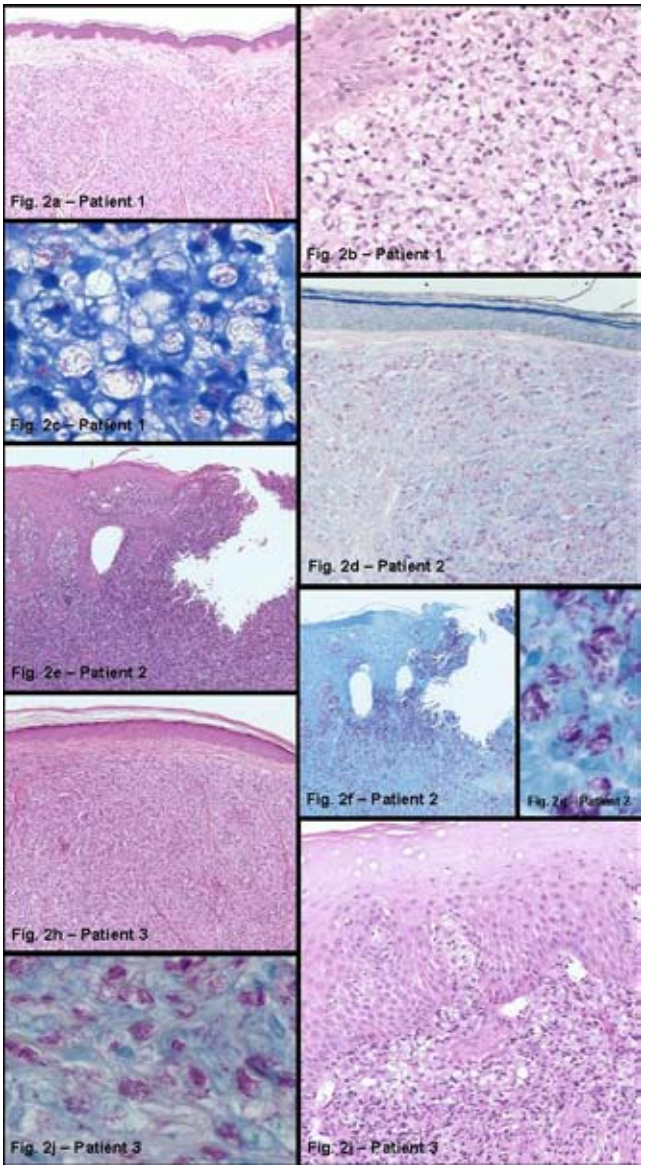

Fig. 2. Histopathological findings of the three patients reported: photomicrograph of skin lesions showing an atrophic epidermis with loss of the rete ridges. The papillary dermis appears as a clear band, and the deeper dermis is infiltrated with chronic inflammatory cells. Hematoxylin and eosin x100 (2a), and fite-faraco x200 (2d). Photomicrograph of oral lesions showing dense mononuclear inflammation with the presence of histiocytes with foamy cytoplasm vacuoles. Hematoxylin and eosin x200 (2b and 2i). Histiocytes with large amounts of acid-fast bacilli compatible with *M. leprae.* Fite-faraco x1000 (2c, 2e, 2h and 2j), and epithelial hyperplasia with areas of ulceration and a dense lympho-histiocytic inflammatory infiltrate. Hematoxylin and eosin x100 (2f), fite-faraco x100 (2g).

- *Clinical findings.* At the time of diagnosis, patient age ranged from 39 to 55. All patients were male rural workers, one of them white and two African-American. They presented skin and mucosal lesions as shown in Figure 1, and were classified as having LL (11). Mucosal disease was found in the palate and nasal mucosa in all cases. The soft palate was affected in all cases and the hard palate in two (Figures 1b, 1e and 1h) (Table 1).

- *Laboratory features.* Complementary tests for the diagnosis of leprosy were the Mitsuda reaction (MR), bacilloscopy, biopsy, and the determination of antibodies to phenolic glycolipid I (anti-PGL1) (Table 1). MR was negative in all patients, whereas bacilloscopy was positive in all of them. Histopathologic examination of the skin showed an atrophic epidermis with loss of the rete ridges in all patients; the papillary dermis appeared as a clear band, and the deeper dermis was infiltrated with chronic inflammatory cells (Figures 2a, 2d and 2h). Examination of the oral lesions showed epithelial hyperplasia with areas

Table 1. Demographic, clinical, and laboratory data of three patients with oral manifestations of leprosy

Patient Nº	Demographic data				Year*	Duration of disease§ (years)	Clinical form	Skin lesions	Mucosal lesions	Bacilloscopic exam	MR†	Anti-PGL1‡
	Age (years)	Sex	Race	Occupation								
1	55	M	W	Rural worker	2003	10	LL	+	Hard and soft palate	3+	-	1.3
2	54	M	A	Rural worker	2005	1	LL	+	Soft palate	5+	-	4.2
3	39	M	A	Rural worker	1996	1	LL	+	Hard and soft palate	3+	-	4.3

M: male; W: white; A: African-American; *year of first consultation; § time reported by patient; †MR: Mitsuda reaction; ‡cut-off = 0,03; LL: lepromatous leprosy: Antibodies to phenotic glycolipid 1.

of ulceration and a dense lymphohistiocytic inflammatory infiltrate with the presence of histiocytes showing foamy cytoplasm vacuoles with large amounts of acid-fast bacilli in all patients (Figures 2b, 2c, 2e, 2f, 2g, 2i and 2j). High levels of anti-PGL1 antibodies were detected in all patients (Table 1).

- *Treatolipid 1.ment and outcome*. Patients were treated with multidrug therapy for multibacillary leprosy consisting of 600 mg rifampicin daily, 300 mg clofazimine once a month/50 mg daily, and 100 mg dapsone daily, for a total of 24 doses. All patients completed treatment with resolution of their leprosy-specific skin and oral lesions (Figures 1c and 1f). However, they developed reaction episodes (erythema nodosum) during the treatment of leprosy, so they needed to be treated prednisone and/or thalidomide. All three patients are currently free of oral leprosy lesions.

Discussion

The upper airways are the main point of entry for the bacillus and a route for bacillary elimination in leprosy (9,12,13). For this reason, the control of mucosal lesions is very important. Mucosal involvement is particularly outstanding in the nose, probably due to the preference of *M. leprae* for cooler sites (10,12,13).

Oral lesions usually appear as ulcerations of the hard or soft palate (3,4,8), as observed in our 3 cases. However, they can affect any other site, including the tongue (5,9). In addition, these lesions may be nodular and ulcerated, as observed in patient 1. In general, oral involvement only appears in the advanced stages of LL, suggesting hematogenous or lymphatic dissemination of *M. leprae* (13). Two of our patients reported that they had noticed the disease one year before, a fact possibly indicating a delayed diagnosis since these patients presented the LL form.

Another possibility for the development of oral lesions is continuity, with nasal lesions possibly being precursors of oral lesions (3,9). Local treatment such as a mouthwash with antimicrobial solutions may be offered to reduce the risk of secondary infection. But it is unsatisfactory unless leprosy-specific treatment is instituted. All patients had nasal and oral lesions, with the latter being limited to soft tissues. Therefore, this suggests the occurrence of hematogenous dissemination rather than lesion by continuity.

The present findings suggest that the route of transmission may increase in endemic areas when the oral mucosa is affected, since viable bacilli have been detected at these sites (9). Thus, patient examination should be extended to the oral mucosa because in advanced leprosy the mouth can acquire the characteristics of a reservoir of bacilli, and thus may act as an important risk factor for transmission of the illness.

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