Lepr Rev (1983) **54,** 119–127

The rate of loss of maxillary anterior alveolar bone height in patients with leprosy*†

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Received for publication 18 October 1982

Summary Alveolar bone loss and periodontal status were measured radiographically and clinically in 22 patients with leprosy after a 4-year interval. The average reduction in alveolar bone height in the anterior maxilla ranged from 0·09 to 0·13 mm per year, being lowest in patients with lepromatous disease. These results are similar to previous measurements of attachment loss, a comparable parameter, in Norwegian patients without leprosy who exhibit good oral hygiene and much better than Sri Lankan patients with poor oral hygiene similar to that found in these patients with leprosy. These data suggest that previous observations of increased alveolar bone loss in patients with lepromatous disease are the result of bone lost before treatment and that reduced bone loss in the presence of abundant dental plaque and poor oral hygiene may be related to immune dysfunctions in patients with leprosy.

Introduction

Skeletal manifestations of leprosy in and around the oral cavity were first described as *facies leprosa*,[1, 2] a tripartite resorption of the maxillary bone involving the nasal surface of the hard palate, its anterior extension, the anterior nasal spine, and its oral projection, the alveolar bone supporting the maxillary incisor teeth. These discoveries made from examinations of the skeletal remains of a medieval population of Danes with leprosy have recently been documented in

* Supported in part by a grant from the Heiser Program for Research in Leprosy and Grant No. 135/77 from the University of Malaya.

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0305-7518/83/054119+09 $01.00

a monograph.[3] Clinical studies of contemporary populations of patients with leprosy have confirmed and extended these observations.[4–9] These studies have established that resorption of alveolar bone in the anterior maxilla is a characteristic manifestation of leprosy, being most pronounced in the lepromatous form of the disease.

The purpose of the present report is to extend observations made earlier[8] on a population of treated patients in Malaysia. Specifically, our aim was to perform radiographic and clinical examinations on as many former patients as possible in order to determine the rate of loss of maxillary alveolar bone over 4 years and to determine the incidence of local etiologic factors for the pathogenesis of periodontal disease in this population. We have found that the rate of alveolar bone loss over 4 years in these patients is extremely low, comparing favourably with measurements of attachment loss recorded in larger clinical studies of patients without leprosy, and that bone loss is less than that expected by the usual indicators of periodontal inflammation. These results suggest that the previously recorded differences between patients with lepromatous and other types of the disease could be due to accelerated bone loss in untreated lepromatous patients, and that treatment greatly reduces the risk of alveolar bone loss.

Materials and methods

All available patients (22) from a previous study[8] at the National Leprosy Control Centre, Sungei Buloh, Selangor, Malaysia were re-examined clinically and radiographically 4 years later. Twenty-five patients from our previous study had been sent back to their residence during this 4-year interval, in accordance with the national policy of decentralization of treatment, and were not available for follow-up. All patients were under continuous treatment for leprosy during this period and none exhibited any evidence of relapse. All procedures were explained in detail to each patient who gave written informed consent in advance.

Radiographic examination of alveolar bone in the anterior maxilla was performed in each patient using the paralleling long-cone technique as described previously.[8] Alveolar bone height was measured on these radiographs using a modification (Figure 1) of the Schei method.[10] Measurements of the distances between the apical foramen, the crest of alveolar bone and the cemento-enamel junction (Figure 1) were made on both sides of the maxillary central incisors using adjustable, fine-toothed calipers and a micrometer. Readings were made to the nearest 0·05 mm, and duplicate measurements differed less than 3%. Alveolar bone height on the four sides of the two maxillary central incisors was calculated (Figure 1) as: $AB/AC \times 100$ and then expressed as the mean of these four measurements. Percentage alveolar bone loss for each patient was determined as: 100 – alveolar bone height. This measurement taken in 1982 was then subtracted

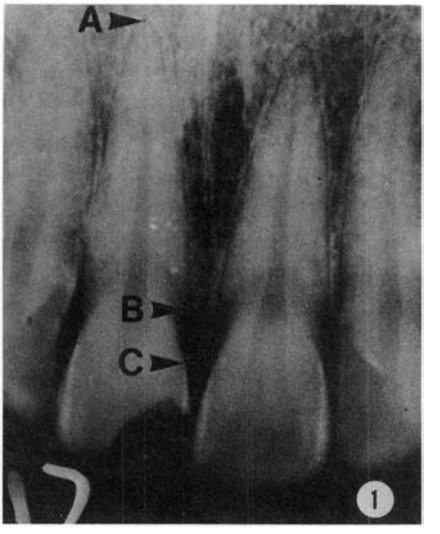

Figure 1. Periapical radiograph of the maxillary central incisors illustrating the radiographic landmarks used to measure alveolar bone loss. From above downward, these points (arrowheads) are the apical foramen (A), crest of the interproximal alveolar bone (B) and the cemento-enamel junction (C). Alveolar bone loss was expressed as the percentage reduction in alveolar bone height and calculated as follows: $100-(AB/AC \times 100)$. Alveolar bone loss in this patient was 14·8%. The patient identification number is in the lower left corner of the radiograph ($\times 2{\cdot}08$).

from the 1978 calculation performed under identical conditions to determine the change in alveolar bone height over 4 years.

Clinical examination of these patients in 1982 was more extensive than in 1978 and included complete charting of each patient's mouth. For each tooth we recorded the mean periodontal probing depth for 6 points and tooth mobility on a scale of 0–3.[11] The periodontal status of each patient was evaluated by the Gingival Index[12] and the Plaque Index.[13] These data were used to assess the general periodontal condition of each patient which was then correlated with the change in alveolar bone height.

Statistical evaluation of the results was performed using the Student's *t* test.[14]

Results

The reduction in alveolar bone height in the anterior maxilla for the 22 patients in this follow-up study is shown by disease type in Figure 2.

The rate of loss of alveolar bone height over 4 years in these three groups of patients is shown in Table 1. The data presented were obtained by subtracting the 1978 measurements of alveolar bone loss from those obtained for the same patient in 1982. The rate of alveolar bone loss in the patients with lepromatous

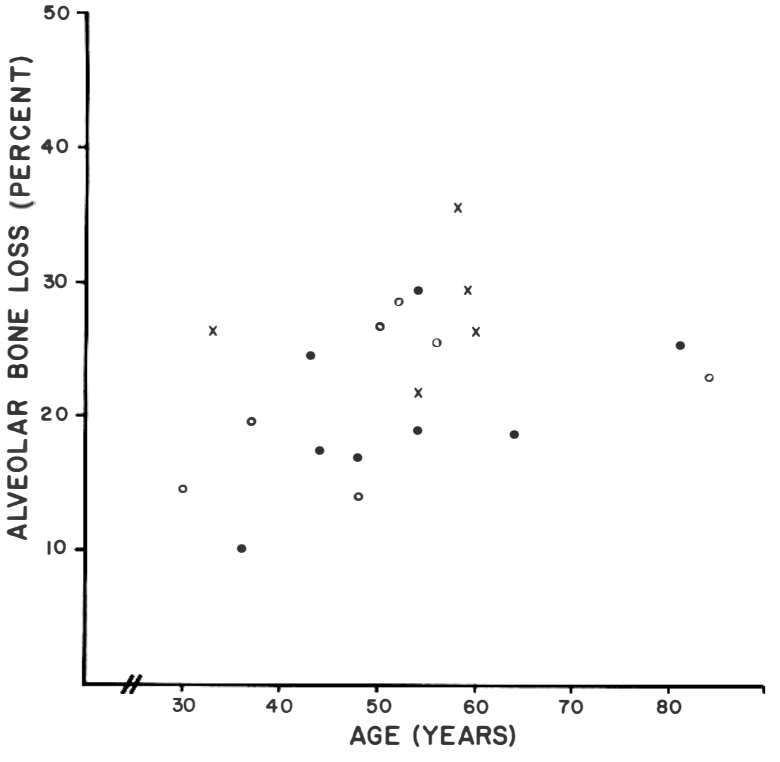

Figure 2. Graph depicting the reduction in alveolar bone height (percentage alveolar bone loss) by patient age and disease type in 1982. x, lepromatous; ●, borderline; ○, tuberculoid.

Table 1. The rate of loss of alveolar bone in the anterior maxilla by disease type

Type of leprosy	Number of patients	Range of ages	Mean loss of alveolar bone height in 4 years	
			(%)	(mm)
Lepromatous	6	33–60	2·1	0·34
Borderline	9	36–81	3·2	0·51
Tuberculoid	7	30–84	2·2	0·35

disease is not significantly different from the other patients. The original height of alveolar bone around the maxillary central incisor is closely approximated by the distance AC in Figure 1. This distance is actually the mean root length of this tooth (13·0 mm) plus 3·0 mm, the average curvature of the cervical line on the mesial and distal, a total of 16·0 mm.[15] Using this measurement, the actual loss of

alveolar bone height can be calculated (Table 1) to be between 0·34 and 0·54 mm over 4 years depending on disease type.

Notations of changes in bone height, patient age and various clinical parameters used to indicate periodontal status for each patient are shown in Table 2. The first two columns restate data from Figure 2. The mean age for lepromatous, borderline and tuberculoid patients is 52, 53 and 51 years respectively (Figure 2 and Table 2). The average alveolar bone loss for lepromatous patients (29·5%) is significantly greater ($P \leq 0{\cdot}05$) than that for tuberculoid patients (21·8%) but not significantly different from that for borderline patients (22·2%) (Table 2, Figure 2). The third column shows the change in alveolar bone height from 1978. The extremes ranged from a net gain of

Table 2. Rate of maxillary anterior alveolar bone loss and periodontal status of patients by age and disease type

Type of leprosy	Age of patient	Alveolar bone height		Plaque index (0–100%)	Gingival index (0–3·0)	Tooth mobility (>1)	Periodontal pocket		Deepest pocket in max. anterior (mm)
		1982 (% loss)	Change from 1978				(>3 mm)	(>4 mm)	
Lepromatous	33	26·5	−3·3	20	1·5	No	Yes	No	4
	49	36·7	0	100	2·0	Yes	Yes	Yes	2
	54	21·8	+2·2	10	1·0	No	No	—	1
	58	35·7	−3·6	65	1·3	No	Yes	No	3
	59	29·6	−4·2	100	2·0	Yes	Yes	Yes	5
	60	26·6	−3·8	15	0·4	No	Yes	Yes	2
Borderline	36	10·0	0	40	1·2	No	No	—	2
	43	24·9	−4·9	70	1·3	No	No	—	3
	44	17·3	−4·3	40	1·5	No	Yes	No	2
	48	17·0	0	75	1·3	No	Yes	Yes	3
	49	37·2	−4·3	70	1·0	Yes	Yes	Yes	3
	54	18·9	−2·0	35	0·6	No	No	—	2
	54	29·6	−5·5	100	1·8	No	Yes	Yes	5
	64	18·6	−3·8	80	1·0	No	No	—	2
	81	25·9	−3·8	100	1·1	Yes	Yes	Yes	3
Tuberculoid	30	14·8	−2·8	25	1·0	No	No	—	2
	37	19·5	−8·3	100	2·0	No	Yes	Yes	3
	48	13·7	+1·3	50	1·2	No	No	—	2
	50	27·0	−5·0	30	1·3	No	Yes	No	2
	52	28·9	0	50	1·0	No	Yes	No	1
	56	25·4	−2·5	60	1·2	Yes	Yes	Yes	4
	84	23·1	−1·9	40	1·2	Yes	Yes	Yes	4

2·2% (+2·2) for the third patient listed to a loss of 8·3% (−8·3) for the 37-year-old tuberculoid patient. Seven of the 22 patients had either a net gain in bone height or no loss during this 4-year period. Changes in alveolar bone height between lepromatous, borderline and tuberculoid patients were not statistically significant.

Several indices of periodontal status are shown in columns 4–9. The plaque index (column 4) is a calculation of the incidence of supragingival plaque on the surfaces of teeth. In our patients this ranged from 10 to 100%. The gingival index is a clinical estimate of inflammation in the gingiva and is scored from 0 (absence of inflammation) to 3 (colour changes and spontaneous bleeding). Only 2 patients had scores of less than 1·0 (mild inflammation but no bleeding on probing). Tooth mobility is scored on a scale of 0–3 (moderate movement in 2 directions and depressable). A reading of greater than 1·0 (column 6) indicates tooth mobility significantly greater than that present normally. The plaque index shows the extent of bacterial colonization (plaque) of the tooth surface, the gingival index the patient's local inflammatory response and ulceration (bleeding) of the pocket lining, and pocket depth (columns 7, 8) the progression of gingival enlargement and/or destruction of alveolar bone and periodontal ligament fibres holding the tooth to alveolar bone. Tooth mobility (column 6) then results from reduced tooth support and tends to be a later manifestation of periodontal disease. Data in columns 4–8 refer to the entire mouth, those in column 9 to the maxillary anterior region. Review of these data shows that plaque deposits are found around most teeth in most patients, all patients exhibit inflammatory changes in the gingiva, but that pocket depths vary. More than half the patients had no pockets greater than 4 mm (column 8), a measurement generally considered to be the limit of normal or indicative of minimal pathology. There is good correlation between these parameters and bone changes in the 2 patients at the extremes (+2·2 and −8·3). The most notable exception is the second patient listed where no bone loss is accompanied with a plaque index of 100%, a gingival index of 2·0, progressive mobility and pocketing (except in the maxillary anteriors).

Discussion

These data show that alveolar bone loss in the anterior maxilla is greater in patients with lepromatous leprosy compared to those with tuberculoid disease but that the rate of bone loss over 4 years is not different in patients with lepromatous, borderline or tuberculoid leprosy who have been under continuous treatment. Thus, treated patients with lepromatous disease are at no greater risk of alveolar bone loss than patients with other types of leprosy. These data suggest that the increased alveolar bone loss seen in patients with lepromatous disease may result from an unusual susceptibility of untreated lepromatous patients to maxillary alveolar bone loss. A study of the correlation of this bone loss and the

known duration of untreated disease in a large group of lepromatous patients could test this hypothesis.

The rate of alveolar bone loss (Table 1) in these patients compares favourably with that of larger populations of patients without leprosy. Over 1,000 patients were studied[16] in Norway and Sri Lanka for 6 years, during which time periodic measurements of plaque accumulation, gingival index and loss of attachment were made. Loss of attachment is a clinical measurement determined by probing and requires a prior loss of alveolar bone and periodontal ligament attaching tooth to bone. Thus, longitudinal measurements such as in their study correlate well with alveolar bone loss. They found the rate of attachment loss in Norwegians to be 0·08 mm/year and in Sri Lankans to be 0·29 mm/year. The main clinical difference in these two populations was that the Norwegians exhibited significantly less plaque and gingival inflammation and much better oral hygiene than the Sri Lankans.[17] Data from Table 1 converted to annual loss of alveolar bone show that our treated patients with leprosy lost 0·09–0·13 mm bone height per year. Thus, the patients in the present study lost alveolar bone (attachment) at a rate similar to the Norwegians. However, their oral hygiene was much worse than the Norwegians', being similar (Table 2) to that recorded for the tea plantation workers in Sri Lanka.[16, 17] This association of low bone loss and poor oral hygiene in patients with leprosy is puzzling, because numerous studies have confirmed a direct correlation between accumulation of plaque, alveolar bone loss and the severity of periodontal disease.[18, 19] The answer may lie in the complex host immune response in the periodontium where lymphocytes, macrophages and other components of the immune system responding to the microflora are believed to play key roles in the pathogenesis of periodontal disease.[20, 21] Recently evidence[22] has been provided for a selected suppression of lymphoproliferation by macrophages and T-lymphocytes in patients with leprosy. This inability of patients with leprosy to respond to certain antigens may also protect them from alveolar bone loss in the presence of numerous local factors that ordinarily increase bone loss. This clinical dilemma should be solved by a better understanding of the specific immune defects in leprosy and knowledge of the specific oral microflora[23] in patients with leprosy. The initial maxillary alveolar bone loss in patients with leprosy might be directly attributable to some local effect of *Mycobacterium leprae* on bone cells, the greater susceptibility of lepromatous patients being attributable to the bacillary concentrations in the nasal mucosa. Once treated, patients with leprosy may be protected from this influence on alveolar bone. In addition, the specific immune derangements that make patients susceptible to leprosy in the first place may also protect them from alveolar bone loss associated with periodontal disease. The fate of *M. leprae* and the patient's susceptibility to periodontal disease may well be decided by the immune response.

The observation that several patients exhibited either a gain or no loss of bone height over 4 years deserves comment. Some of these patients (Table 2) exhibited

good oral hygiene but this was not universal. The loss of alveolar bone is believed to be asynchronous. [24] Radiographic observations of increased bone height are not rare,[25] but are seen primarily after optimal oral hygiene and reduction of inflammation.[26–28] It is conceivable that the compromised immune response in patients with leprosy may not only protect them from bone loss proportional to the intensity of dental plaque but may also permit restoration of bone height. Answers to these questions will have to await more information. Clearly, the initial susceptibility of lepromatous patients to localized alveolar bone loss and the lack of correlation of bone loss and oral hygiene status in treated patients with leprosy deserve further study. What appears to be clear at this point is that early treatment of patients with lepromatous leprosy can reduce alveolar bone loss. This may be a fortunate clinical development because the well-known hand deformities of leprosy limit manual dexterity in patients with advanced disease and good oral hygiene in these patients may be impossible without assistance.

Acknowledgements

We thank the Director and staff of the National Leprosy Control Centre, Sungei Buloh, Selangor, Malaysia for their enthusiastic support. We particularly thank Mr Chiew Hock Koon our superb, invaluable assistant and the patients who agreed to this follow-up study. We are also indebted to Colgate-Palmolive (Malaysia) for their donation of home care kits. We have profited from reviews of the manuscript by Drs M F R Waters, V Moller-Christensen and H Loe. We thank Dr L Lipworth for assistance with statistical analyses and M Manzello for typing the manuscript.

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