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The Impact of Primary Hyperparathyroidism on the Oral Cavity

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Context: Primary hyperparathyroidism (HPT) is a systemic disease causing bone loss. Periodontal disease is a local inflammatory disease characterized by alveolar bone loss. The older literature records that HPT is associated with loss of radicular lamina dura and brown tumors of the bone, but contemporary studies are lacking.

Objective: The objective of the study was to determine the effects of HPT on oral bony structures and periodontal disease in a contemporary population.

Design: This was a cross-sectional, case-controlled study.

Setting: The study was conducted at the clinics of endocrine surgery and hospital dentistry.

Patients and Other Participants: Fifty-nine patients, 39 with HPT and 20 thyroid controls, were included in the study.

Main Outcome Measures: Periodontal clinical measures and dental radiographic analyses were used in this study.

Results: HPT patients were more likely to have tori and reductions in radicular lamina dura on dental radiographs. Widening of the periodontal ligament space surrounding teeth correlated with serum PTH levels. Panoramic radiographs demonstrated reduced cortical bone thickness at the angle of the mandible in HPT patients but no evidence of brown tumors or other overt pathologies.

Conclusions: Changes in the oral cavity observed in patients with HPT suggested both decreased cortical density and increased likelihood of oral tori. The contemporary oral manifestations of primary HPT are different from those previously reported, and health care providers should be aware of newer, more subtle findings that may be present when treating patients with HPT. **(*J Clin Endocrinol Metab* 91: 3439–3445, 2006)**

PRIMARY HYPERPARATHYROIDISM (HPT) is a systemic disease that causes hypercalcemia and affects bone remodeling. The incidence of HPT over the age of 40 yr in the United States is 1 in 500 for women and 1 in 1000 for men (1). Diagnosis of HPT is made most often by the finding of elevated serum calcium and confirmed by concomitant elevation of PTH (2). Parathyroidectomy is one treatment for HPT and is indicated in symptomatic patients and those patients with criteria such as hypercalcemia, hypercalciuria, and reduced bone density as outlined in recent guidelines (2). Metabolic bone diseases such as HPT may affect the entire skeleton and may cause abnormalities in the oral cavity as well, possibly by inducing alterations in oral hard tissues or by exacerbating common pathological processes clinicians encounter daily such as periodontal disease.

There is a paucity of available literature pertaining to oral changes due to HPT. Whereas osseous changes have been classically reported as oral manifestations of HPT, these have been described mainly in patients in the late stages of HPT and before the advent of improved diagnostic techniques (3). There are no cross-sectional or prospective reports of the impact of primary HPT on periodontal health. The specific objectives of this study were to determine the systemic effects of primary HPT on differences in bone density and architecture and commonly sought parameters of periodontal disease by means of a comprehensive investigation of oral health.

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Abbreviations: ADA, American Dental Association; AI, antegonial index; AL, attachment loss; BOP, bleeding upon clinical probing; GI, gonial index; HPT, hyperparathyroidism; MI, mental foramen index; PDL, periodontal ligament.

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Patients and Methods

This study was carried out by faculty and graduate students at the University of Michigan Medical and Dental Schools. The study design and protocol were reviewed and approved by the University of Michigan Medical School Institutional Review Board. Patients were drawn from the pool of all patients referred to the Division of Endocrine Surgery, Department of Surgery, University of Michigan, for treatment of primary HPT and were similar to those described by Burney *et al.* (4). Control patients had thyroid abnormalities, most commonly the presence of goiter, and did not demonstrate overt signs of altered skeletal metabolism. Such patients are commonly used as controls in studies of HPT (5). Patient consent to the study was obtained via a written document, including the purpose of the study, duration, risks involved, benefits, and confidentiality information. Children, women unsure of their pregnancy status, and patients requiring antibiotic prophylaxis for subacute bacterial endocarditis were excluded. Dental examinations were performed when patients were evaluated for preoperative history and physical exam, typically 2–4 wk before their endocrine surgery.

All patients underwent a comprehensive clinical examination. Clinical parameters obtained by a calibrated examiner (A.D.P.) included number of teeth, clinical attachment levels assessed with a periodontal

probe, and bleeding upon clinical probing (BOP) of the gingival sulcus, which is indicative of the inflammatory status of the periodontium. Calibration was performed to facilitate the collection of accurate clinical measurements as is recommended for clinical studies on periodontal disease patients (6). The examiner performed repeated measurements on subjects who had similar oral conditions as those patients being studied until their readings demonstrated reproducibility and agreement (>90%) with a standardized clinician. Furcation involvement is a clinical measure of destruction of the soft tissue and bone in between the roots of multirooted teeth (most typically molar teeth) and was evaluated for all upper and lower molar (multirooted) teeth by routine examination with a periodontal probe. Mandibular width was determined with a caliper placed at the widest area of the posterior mandible, both right and left sides. An average of these two values was then determined per patient. Fremitus is a measure of tooth displacement created by the patient's own occlusal force and reflects alterations in the underlying osseous support. Fremitus was evaluated on all teeth by digital palpation while having the patient simulate chewing/tapping movements. The incidence of furcation and fremitus was recorded as a percentage of patients, with a positive finding (not percentage of teeth). Standardized bitewing and periapical films of the mandibular posterior teeth along with nonstandardized panoramic radiographs were obtained and processed with the same equipment for each patient. Described methods were used to standardize intraoral films (7). Horizontal levels of bone support surrounding teeth were evaluated, contributing to the classification of the periodontal condition according to the American Dental Association (ADA). The ADA classification system rates the disease category based on the severity of bone loss, with I being the mildest (no bone loss) and IV the most severe (8).

Radiographic measurements of radicular lamina dura and interdental alveolar bone density were determined at mandibular first molars, a region reported to be sensitive to alterations due to metabolic bone disease (9, 10). Radicular lamina dura and alveolar density were measured by a blinded examiner (M.T.) using computer-assisted image analysis and a pinpoint density measure tool (ImageJ v1.34; NIH software, Bethesda, MD) along the mesial and distal of the molars (see Fig. 2A). Such measures are highly reproducible with errors in the 3–5% range, and in a previous study, such radiographic findings were corroborated with histological analysis in the bone surrounding dental implants (11).

The average density of replicated pinpoint measures was obtained at two different radicular lamina dura areas (see Fig. 2A): cortical bone layer immediately adjacent to the root surface at the coronal third and at the apical third of the root. The interdental alveolar bone density was measured using the average density of two 1.0-mm^2 regions of interest measured at two areas: at the center of the trabecular crestal bone area 2 mm below the bone crest and at the interradicular area at the middle third of the root length (see Fig. 2A). The adjusted mean values for radicular lamina dura and trabecular density were calculated by normalizing the raw density measurements according to the equation ($y = a + bx$) generated by measuring three gray levels of the aluminum step-wedge radiographic standard density of each individual image. In this equation, x is the raw density value, a is the intercept constant, b is the slope coefficient, and y is the normalized value.

Periodontal ligament (PDL) (a fibrous attachment spanning the tooth root cementum to the alveolar bone) width was also evaluated on radiographs. The PDL was detected as the radiolucent area between the root and the more radiopaque cortical lamina dura. An examiner (A.D.P.) determined PDL width after reaching 90% concurrence in repeated measures of patient radiographs using the Image ProPlus computer software program (Media Cybernetics, Silver Spring, MD). Mean PDL width values of five to six regions of interest were calculated for each patient and compared among and between groups. In addition, a qualitative evaluation of the trabecular pattern was evaluated by two periodontists (W.V.G. and L.K.M.) blinded to the group designations. Radiographs were assigned to one of three categories (loose, medium, or dense trabeculation) and assigned the numerical value of 1, 2, or 3, respectively, and ratings for the two investigators were averaged together.

Panoramic x-rays were evaluated for the presence of brown tumors and/or any other overt pathology (see Fig. 4). Furthermore, because panoramic bone density has been correlated to systemic bone mineral density (12, 13), the following radiomorphometric indices were measured by one blinded examiner (T.F.T.) on each panorex: 1) cortical thickness below the mental foramen index (MI); 2) gonial index (GI); and 3) antegonial index (AI). AI was determined at a point identified by extending a best-fit straight line along the anterior border of the ascending ramus extending down across the lower border of the mandible. A tangent to the lower border was drawn and then a perpendicular to the tangent drawn along which the AI cortical thickness was determined (14). GI is a measurement of cortical thickness at the angle of the mandible and was measured as previously described (15). Briefly, at the lower cortical border of the angle of the mandible, a point was identified at which the inferior border of the mandible and the posterior ascending ramus intercepted, and the GI cortical thickness was measured along this perpendicular. MI was determined as the mean of the cortical thickness of the lower border of the mandible below the two mental foramina (16). All measurements were performed on panoramic films that were digitized and analyzed via computer-assisted projection with a millimeter incremental tool.

Statistical analysis

Statistical analysis was performed by a statistician (B.T.W.) using the SPSS 13.0 statistical software program (SPSS Inc., Chicago, IL). The collected data were initially examined for unusual values and outliers. The HPT and control groups were compared in terms of their means on several measures using independent-sample *t* tests. If the assumption of a normal distribution for the variable of interest was violated in one or both of the two groups, the Mann-Whitney *U* nonparametric test was used to compare the groups. Bivariate correlations were also assessed to determine which variables were highly correlated with each other.

Because a difference in the incidence of tori was seen between groups, logistic regression analysis was used to determine significant predictors of having a torus present. Multiple regression analysis using the variable selection technique of backward selection (17) was used to determine predictive models of attachment loss (AL) and number of teeth. All underlying assumptions of regression modeling were verified in each case, and necessary variable transformations were conducted to satisfy these assumptions. In particular, the AL response was log transformed to obtain constant variance and normality for the random errors in the regression model for AL.

Results

The study population included 54 Caucasians, three African-Americans, one Asian, and one person from another ethnic category. There were 42 females and 17 males (Table 1). Thirty-nine HPT patients and 20 thyroid control patients were evaluated. There were no significant differences between the groups with regard to age and gender. The national average for females having HPT, compared with their male counterparts, is approximately 72% of the total HPT population, and this study closely matched this (2). There was no difference in smoking status between groups, recorded as patients either never having smoked (nonsmokers) *vs.* current or past history of smoking (smokers). Diabetics were not in the exclusion criteria for the study; however, there were no uncontrolled diabetics in either population.

As expected, HPT patients exhibited higher serum calcium levels and elevated PTH levels. Serum calcium levels were routinely obtained on all patients and were normal in the thyroid control group. The normal value for calcium is 8.6–10.2 mg/dl and PTH is 10–65 pg/dl. The duration of hypercalcemia was known for 17 of the HPT patients and ranged from 1 to 25 yr, with most falling between 1 and 5 yr. Five of the HPT patients were on estrogen hormone replacement therapy. Of the thyroid control patients, three had a positive estrogen history and one was taking tamoxifen. Of the thyroid control patients, 14 were in the normal range,

TABLE 1. Patient data

	HPT	Thyroid controls	*P* value
No. of patients	39	20	
Age (yr)	54.7 (26–79)	50.8 (27–79)	NS
Gender	72% female, 28% male	70% female, 30% male	NS
Smoking history (% positive)	50	31.6	NS
Serum calcium (mg/dl)	10.7 (8.9–12.8)	9.3 (8.6–10.2)	$P < 0.05$
Serum intact PTH (pg/dl)	192.8 (53–669)	NA (norm 10–65)	NA
Teeth	25.2 (13–31)	26.3 (13–31)	NS
BOP (%)	23.4 (3–47)	21.2 (3–66)	NS
CAL (mm)	2.76 (1.76–4.04)	3.01 (1.96–6.83)	NS
Furcation (%)	48.4	43.8	NS
Fremitus (%)	36.4	44.4	NS
ADA classification	I (2), II (24), III (9), IV (1)	I (0), II (13), III (5), IV (2)	NS

Data are shown as average (range of values) or mean percentage. Serum PTH was not available for thyroid controls; normal range of PTH values is indicated in *parentheses*. Furcation involvement is a clinical measure of destruction of the soft tissue and bone between the roots of multirooted teeth (most typically molar teeth). Fremitus is a measure of tooth displacement created by the patient's own occlusal force. Furcation and fremitus involvement were both expressed as a percent of the patients with positive signs of involvement. ADA classification is severity of periodontal disease according to the ADA, in which I is least severe and IV is most severe. Number of patients in each category is indicated; an ADA classification was unable to be determined for three patients in the HPT group (lacking radiographs of entire dentition). Teeth, Number of teeth in the oral cavity; CAL, mean clinical attachment loss; NS, not significant; NA, not available.

four had low, and one had a slightly elevated TSH level. Forty-five percent of the HPT patients were symptomatic, with the most typical symptoms being kidney stones, fatigue, and joint pain. Information regarding bone density was available for 11 of the HPT patients, of which seven demonstrated evidence of bone loss based on T-scores of less than −1.0 in one or more of the following sites: hip, femur, and/or lumbar spine. Four patients had T-scores ranging from −2.08 to −4.38. Three patients had scores between −1.0 and −2.0. Four patients had no evidence of bone loss on scan or had T-scores between 0 and −1.0.

No significant differences were seen between groups in regard to number of teeth, percentage of BOP, furcation occurrence, fremitus, or the classification of the periodontal disease status via the ADA system. A regression model fitted using backward selection demonstrated that age and BOP were significant positive predictors of AL. A predictive model for number of teeth resulted in age being the only significant predictor (having a negative relationship).

Intraoral findings demonstrated a significantly greater incidence of torus/tori in the HPT group with the incidence of tori nearly 3-fold greater in HPT patients (Fig. 1). A torus is a bony protuberance in the oral cavity, usually found in the midline of the hard palate or along the lingual border of the mandible in the premolar region (unilateral or bilateral). Intraoral findings also demonstrated a trend for greater mean mandibular width in HPT patients ($P = 0.18$).

Computer-assisted radiographic analysis of periapical films revealed significant reductions in the OD of the radicular lamina dura in patients with HPT (Fig. 2B). The interdental alveolar density was also significantly reduced, and there was a trend ($P = 0.085$) toward reduced interradicular alveolar density in the HPT group (Fig. 2C). A significant correlation was observed between PDL width and serum PTH levels (Fig. 3). That is, as the severity of the disease increased (increased serum PTH levels), PDL width also increased ($r = 0.47$, $P < 0.05$). There was also a significant relationship between the presence of a torus and increased PDL width within the HPT group because patients in the HPT group with a torus had significantly greater PDL width ($P < 0.05$). Because this finding could be due to other differences between people that have a tori and those that do not (*e.g.* more older people have a tori, for example, and older people are more likely to have a higher average PDL), a regression model of PDL was fit, controlling for gender and age and including the tori variable, and patients with a torus present were still found to have a significantly higher mean PDL ($P < 0.05$). The pattern of trabecular bone as qualitatively determined averaged 2.04 ± 0.14 for the control group and 2.00 ± 0.14 for the HPT group (trabecular pattern was scored 1–3) with no significant differences. There was a trend toward a correlation between the quantitative measurements of alveolar density and the qualitative categorization of trabecular pattern in which the average computer-assisted interdental OD for radiographs placed into the loose category (score = 1) by either periodontist was 2.63 ± 0.33 and the average for the dense category (score = 3) was 3.32 ± 0.19 ($P = 0.076$).

Panoramic evaluation revealed reduced cortical bone thickness ($P < 0.05$) at the mandibular ramus, GI of HPT

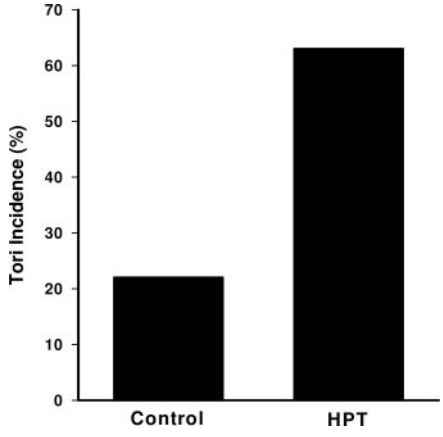

FIG. 1. Incidence of tori. Percentage of patients in each group presenting with maxillary and/or mandibular tori is presented. Patients with HPT were more likely to have a torus present (odds ratio = 4.9; 95% confidence interval, 1.311–19.074; $P < 0.01$).

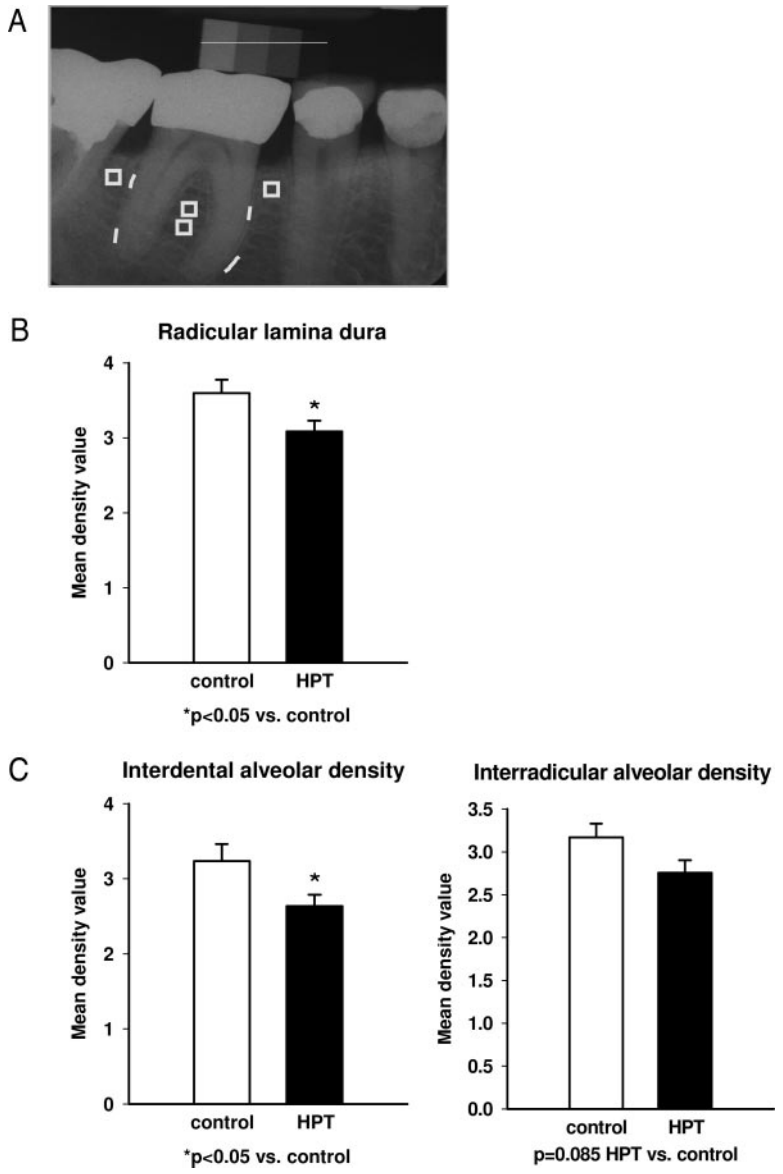

FIG. 2. Quantitative periapical radiographic analysis. Quantitative values for standardized dental radiographs were determined using a pinpoint density measure tool and NIH image software. A, Radiograph demonstrates the sites of trabecular (*squares*) and radicular lamina dura measured (*lines* correspond to measured sites of cortical bone immediately adjacent to the root surface at the coronal third, and at the apical third of the root length). The values are relative to the step wedge (*top center*) that has a linear scoring of 1–4. B, Mean density score for radicular lamina dura on mesial and distal surfaces is presented as mean ± SEM. C, Mean density score for interdental (measured at center of trabecular crestal area 2 mm below the bone crest) and interradicular (measured at the area between the roots at the middle third of the root length) alveolar density presented as mean ± SEM.

patients (Fig. 4). Also, increased GI demonstrated a negative relationship with tori, and logistic regression analysis demonstrated that increases in GI tended to be associated with a lower probability of having a torus present ($P < 0.10$). There was no difference in AI or MI indices in HPT *vs.* thyroid control patients (data not shown). There was no evidence of gross loss of lamina dura or brown tumors in any patient radiographs.

Discussion

Our findings show that available descriptions of the oral manifestations of HPT are outdated. The clinical and radiographic effects of HPT on oral health were described in 1945 and 1962, and these changes are still associated with HPT today (18–20). In the present cross-sectional study, a more current and accurate description was sought. There was no difference found between patients with HPT and thyroid controls with regard to the number of teeth present or the amount of clinical attachment loss, and no differences were seen in other periodontitis parameters such as bleeding on probing and furcation involvement. Patients with HPT, however, had a higher likelihood of having an oral torus, and HPT was associated with reduced radicular lamina dura, reduced interdental alveolar bone density, and reduced cortical bone at the GI.

HPT and periodontal disease both share a predilection toward bone resorption but differ in their etiology and extent. End points in periodontal disease are connective tissue AL and tooth loss, both variables evaluated in this study. Number of teeth is also of interest due to a previous link between tooth loss and whole-body bone mass (21). However, this relationship is still somewhat controversial because some studies do not report a link among systemic bone mineral density, tooth loss, and periodontal disease (22–24). In the present study, there was no significant difference between the two groups regarding number of teeth or AL,

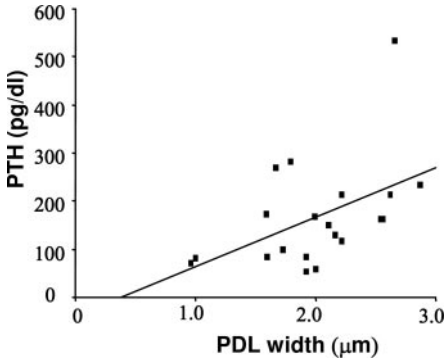

FIG. 3. Regression analysis of PDL width measured from periapical radiographs relative to serum PTH levels in HPT patients. There was a strong positive correlation between serum PTH levels and PDL width ($r = 0.474$; $P = 0.035$).

whereas signs reflecting bone loss such as reduced cortical bone and alveolar density were greater in the HPT group. The findings of age and BOP as positive predictors for AL are consistent with the natural course of periodontal disease (25, 26). That there were increased signs of bone loss in the HPT group *vs.* thyroid controls but no alterations in clinical periodontal parameters suggests that the systemic disease impacts the oral cavity but that reduced bone density to the extent found in this study does not predispose to alterations in the soft tissue attachment apparatus consistent with the clinical presentation of periodontal disease. It is possible with a predisposing inflammatory condition that the addition of the systemic insult of HPT could worsen an already existing condition, but in itself HPT was not capable of resulting in attachment loss.

Although changes such as the interdental alveolar bone were found in not only the radicular lamina dura but also areas of trabecular bone, the two-dimensional nature of this radiographic examination precludes the ability to separate out cortical bone of the lingual and facial plates from trabecular bone. Hence, the reduction in bone density could be attributed to alterations in the cortical bone of the mandible and unrelated to trabecular bone. The older literature reported a ground glass appearance in jaws of HPT patients due to replacement of mineralized skeletal tissue with fibrous connective tissue. That no qualitative differences in trabecular pattern were observed may be due to the earlier diagnosis of HPT today rather than to the lack of possible dramatic effects of HPT on this bone.

The high frequency of tori in HPT patients is the most intriguing and novel finding of this study. Tori typically appear in early adulthood and grow slowly over time with the incidence in normal populations reported to be between

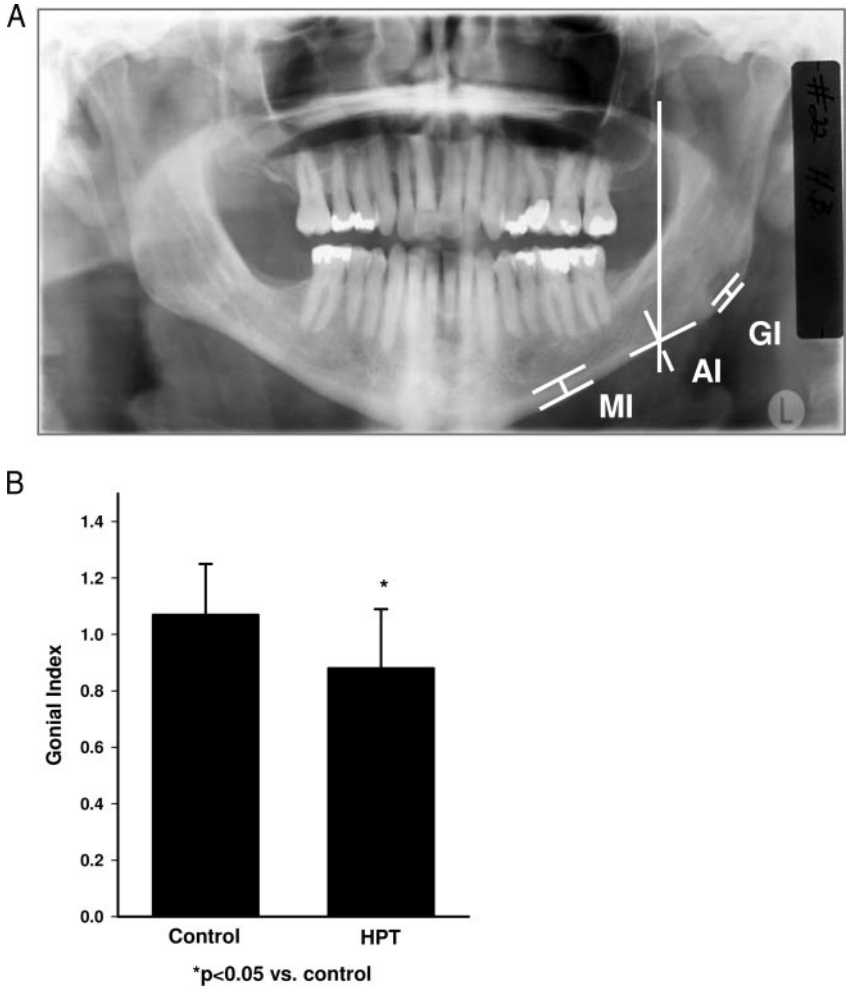

FIG. 4. Panoramic radiographic indices. Panoramic radiographs were analyzed for cortical bone width at three areas: AI, MI, and GI, as shown in A. B, Graph of GI in study patients shows that patients with hyperparathyroidism had lower GI values. There was no difference in values for AI or MI between HPT or thyroid control patients (data not shown).

17 and 27% (27–30). The pathogenesis of tori has long been debated and is generally thought to be multifactorial with contributions of genetic and environmental factors, including occlusal (biting) forces. What are the possible mechanisms involved with the increased incidence of tori present in the HPT group? Recent findings in the noncraniofacial skeletons of patients with HPT suggest that HPT leads to a preferential loss of cortical bone and preservation or increase in trabecular bone (31). It has been proposed that the presence of hypercalcemia in HPT patients is preceded by longer periods of elevated PTH levels (2). It could be that the tori represent an expansion of trabecular bone at the expense of cortical bone in response to elevated PTH levels with possible contribution from the mechanical forces present in the oral cavity (Fig. 5). Anabolic actions of PTH in animals are higher in sites of greater loading (32). Furthermore, PTH mediated endosteal resorption has been shown to be compensated by PTH-mediated periosteal apposition with an increase in overall bone size (2). Recent studies (33, 34) correlated bone mineral density in postmenopausal Caucasian women to the occurrence of palatal tori and oral exostoses. It is also possible that there are etiological factors and mechanisms involved in both HPT and oral tori and that a cause-and-effect relationship is not present.

Another intriguing finding is the correlation between PDL width and serum PTH levels, with the greatest PDL width found in patients with the highest PTH levels. It is reasonable to extrapolate that if increasing serum PTH levels were left unchecked or untreated, the PDL width could eventually develop into radiographically detectable radicular lamina dura loss. PDL width in patients with a torus was higher than those without a torus, and this intriguing relationship remained when issues of gender and age were taken into account. This is consistent with the HPT patient population having reduced radicular lamina dura density and may reflect the localized impact of PTH in the PDL space to evoke cortical bone loss in the area adjacent to the tooth, whereas compensatory mechanisms are taking place for increased bone growth in the form of the tori more laterally.

Indices on dental panoramic radiographs have been evaluated for their relationship to systemic bone mass and proposed as screening tools for osteopenia (12, 35). The present study detected reduced GI values in the HPT group and hence reduced cortical thickness at the angle of the mandible. A decrease in cortical bone in the HPT patients would be consistent with the preferential catabolic effects of PTH on cortical bone.

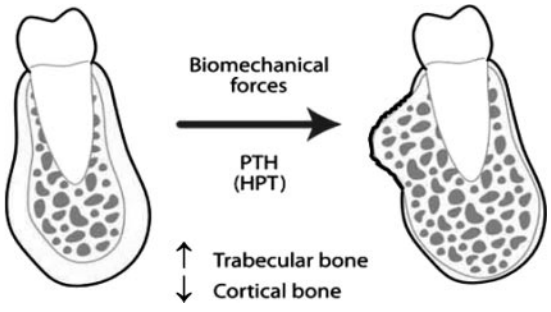

FIG. 5. Model of osseous structure in HPT patients. The data in the present study suggest that in the oral cavity, the effect of elevated PTH in HPT patients reduces cortical bone and protects trabecular bone. In combination with the biomechanical forces particular to the oral cavity, cortical bone loss and trabecular expansion result in an increased incidence of tori.

Whereas it would have been interesting to compare the radiographic and other findings of this study with information obtained from dual-energy x-ray absorptiometry scans, we were unable to do this. There are numerous studies that have already correlated oral and systemic bone levels (36), and although it may have provided more information, this was not the major focus of this work. Dual-energy x-ray absorptiometry scan information was available in only a minority of patients and was not standardized. Other possible limitations of this study were that a complete data set on the menopausal status of female patients was not available, and serum PTH levels were not obtained on thyroid control patients. Serum PTH levels would not routinely be obtained for normocalcemic patients not suspected to have HPT; hence, there is a possibility that there could have been individuals with normocalcemic HPT or subtle secondary HPT in the thyroid control population. Furthermore, some of the thyroid control patients were on thyroid hormone replacement, which could alter bone metabolism if subclinical hyperthyroidism was present. Although thyroid hormones alter bone metabolism, such alterations are typically associated with very high doses for long duration (37, 38). Finally, the study population was imbalanced because there were not equal numbers of patients in the HPT and thyroid control groups. This can result in an issue of power because imbalance could lead to a lack of power to detect significant effects. Randomization of the patients into the two groups was not possible because this was essentially a convenience sample. Future studies might benefit from a balance in the sizes of the groups. We think it quite unlikely that any of these limitations affect the overall conclusions of this study.

This study indicates that the contemporary profile of HPT manifestations in the oral cavity is different from described half a century ago. Whereas previously, oral manifestations were severe enough that they might be the first sign of disease, this is no longer the case in a contemporary population of patients undergoing surgery for HPT. The changes found are much more subtle and include anticipated reductions in cortical bone but do not appear to have a significant impact on periodontal health. Nevertheless, new findings such as increased incidence of oral tori may reflect a form of anabolic action of PTH in bone.

In conclusion, patients with primary HPT demonstrated: 1) increased incidence of tori; 2) reduction in indices of cortical bone (lamina dura and gonial index); 3) a correlation between serum PTH levels and PDL width; but 4) no striking differences in measures of periodontal health, compared with the thyroid control group. Furthermore, no evidence of previously reported oral findings associated with HPT, specifically gross loss of radicular lamina dura, brown tumors, or striking ground glass radiographic bone appearance, was observed. These findings suggest that whereas there are osseous alterations in the oral cavity associated with HPT, these changes are not nearly as dramatic as previously reported, likely a function of different disease severity and presentation profiles in the current era.

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