CLINICAL ORAL IMPLANTS RESEARCH

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Tobacco consumption induces alveolar crest height loss independently of mandibular bone mass and bone density

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Key words: alveolar bone loss, bone mineral density, cone-beam computed tomography, periodontitis, radiography, tobacco

Abstract

Aim: To investigate the relationship of tobacco consumption with alveolar crest height (ACH) loss and mandibular bone mass estimated by digital panoramic radiography and cone-beam computed tomography (CBCT).

Material and methods: We studied 315 patients (43.2% [*n* = 136] men and 56.8% [*n* = 179] women) with mean age of 36.6 ± 5.3 (range 21–30 years (16.2%), range 31–46 years (83.8%); 9% (*n* = 71) were smokers (>10 cig./day). A mean of 13.0 ± 2.0 mandibular teeth were present: 37.5% of patients had 6–12 teeth and 62.5% 13–16 teeth. We analyzed 315 digital panoramic radiographs (2D) and calculated the ACH, mandibular cortical width, and basal and alveolar bone gray level values. ACH and bone density were also measured on CBCT (3D) in the 110 patients scheduled for implantation.

Results: In the univariate analysis, ACH loss was greater in older patients ($P = 0.012$) and in those with fewer mandibular teeth ($P < 0.001$) and showed a relationship with tobacco consumption that was close to significant ($P = 0.079$). In the multivariate analysis, the number of mandibular teeth ($P < 0.001$) and tobacco consumption ($P = 0.048$) were significantly associated with ACH. Alveolar and basal bone densities were associated, respectively, with number of mandibular teeth ($P = 0.012$) and cortical width ($P = 0.030$).

Conclusion: In a Caucasian population aged 21–46 years, tobacco consumption was significantly associated with ACH loss. However, ACH loss showed no significant relationship with mandibular bone mass estimated either as mandibular cortical width index or bone density on digital panoramic radiographs or as bone density on CBCT. ACH loss was a local event independent of mandibular bone mass status.

Cigarette smoking has been associated with alveolar crest height (ACH) loss in different conditions, including chronic periodontitis (Bergstrom 2004; Levin & Levine 2010; Bahrami et al. 2011); post-menopause (Hildebolt et al. 2000; Payne et al. 2000; Rosa et al. 2008); and peri-implant disease (Levin et al. 2008; Maximo et al. 2008), and it has been shown to have a negative impact on bone regeneration after periodontal treatment (Maximo et al. 2008).

Clinicians have relied on clinical examinations and radiographs to determine the amount of periodontal destruction. Only a few studies have compared the effectiveness of panoramic radiography and intraoral single-tooth radiography to detect alveolar bone loss. One study on the accuracy of panoramic and intraoral radiography, using open surgery measurement as the gold standard, reported underestimations of bone loss ranging from 13% to 32% for panoramic films and from 9% to 20% for intraoral single-tooth films (Akesson et al. 1992). Another group found a close agreement in bony height assessment between panoramic and intraoral radiographs and also evidenced a symmetry between left and right sides of maxilla and mandible (Persson et al. 2003). Kim et al. (2008) investigated patients with aggressive periodontitis, characterized by rapid bone level changes, and affirmed that a preorienta-

Date:
Accepted 11 May 2013

To cite this article:
Mesa F, Souki N, Galindo-Moreno P, Velasco-Torres M, O'Valle F, Bravo M. Tobacco consumption induces alveolar crest height loss independently of mandibular bone mass and bone density.
Clin. Oral Impl. Res. **25**, 2014, 1034–1040
doi: 10.1111/clr.12207

tion with respect to the expected bone loss is possible using panoramic radiographs. Panoramic radiographs have also proven useful to determine the prevalence of infrabony defects in both maxilla and mandible (Muller & Ulbrich 2005).

The mandible is highly sensitive to alterations in body bone mass, and numerous studies have demonstrated a correlation between mandibular and skeletal (vertebral) bone densities (Kribbs et al. 1990; Law et al. 1996; Takaishi et al. 2005; Kavitha et al. 2012). Bone quality differs among individuals (Nelson & Megyesi 2004) and can be assessed on different types of radiographs. Various indexes are available for mandibular bone quality assessment on panoramic X-rays based on the manual measurement of anatomical structures (Bras et al. 1982; Yang et al. 1987; Benson et al. 1991; Taguchi et al. 1995). Taguchi et al. (Taguchi et al. 1996) measured the mandibular cortical width (MCW) on a vertical line to the center of the foramen, passing through both cortical bones. Index values were correlated with bone mass density values obtained by quantitative computerized tomography (CT), considered the gold standard test.

The utilization of 2D (digital panoramic radiography) or 3D (cone-beam CT) (CBCT) image analysis is a rapid and simple method to determine bone mineral density expressed in gray values; results have shown a high correlation with density values obtained by dual-energy X-ray absorptiometry (Nackaerts et al. 2007) and by conventional CT (Nomura et al. 2010), gold standard techniques in 2D and 3D, respectively.

The objective of this study was to investigate the relationship of tobacco consumption with ACH loss and mandibular bone mass estimated by two different radiographic methods: 2D analysis to measure the crest height, MCW index, and alveolar and basal bone density; and 3D analysis (CBCT) to measure the crest height and alveolar bone density.

Material and methods

Sample

This retrospective study initially considered all 1609 panoramic radiographs taken across all dental disciplines during 2008 at the University of Granada Dental School Clinic. We first excluded radiographs from patients under 21 years old to avoid the effects of bone development and from those more than 50 years old to avoid the effects of menopause on bone mass in the women. Radiographs were then excluded if they corresponded to patients with a history of bone metabolism disease, active osteoporosis treatment (estrogen hormone, calcium, calcitonin, vitamin D, fluorides, or bisphosphonates), history of radiotherapy, or the presence of <6 teeth in mandible. The following exclusion criteria were applied to the remaining panoramic radiographs: observation of mesial drift in a tooth of interest; poor visibility of mental foramen, mandibular cortical bones, or cement–enamel junction (due to caries, fillings, or crown); and the presence of an artifact or development defect in the radiograph, leaving a final study sample of 315 panoramic radiographs corresponding to 315 patients. Of these 315 patients, 110 were candidates for implant surgery and were also studied with CBCT.

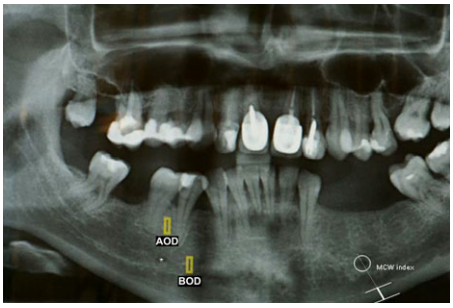

Fig. 1. Digital panoramic radiograph with the three measurement variables: AOD: alveolar optical density; BOD: basal optical density; MCW index. Asterisk: mental foramen.

Radiological assessment

2D analysis

All panoramic radiographs were taken by a single operator using the same analog orthopantomograph (Xmind TOME®; Satelec, Orion Corporation Soredex, Helsinki, Finland) at 3× magnification and exposure values of 70 Kv (male) or 65 Kv (female), 10 mA, and 19 s. Radiographs were then digitalized using an HP Scanjet G2710® scanner (Hewlett Packard) at a resolution of 600 dpi. Dent-a-View® version 1.0 (Digident CR, Wehmer Co, NJ, USA) image analysis software program was applied by a single calibrated researcher to calculate the alveolar crest height, MCW index, and radiographic optical bone density, avoiding interobserver variability. We used the absorption level option of the image analysis software program, which offers greater sensitivity for data collection (range 0–4095) in comparison with the gray level option (range 0–255). The internal validity of the study was maximized by our application of rigorous inclusion and exclusion criteria to the patients and radiographs.

The ACH was expressed as the mean percentage and calculated as A/B x 100, where "A" is the distance from the cement–enamel junction to the alveolar crest (at the most coronal location of the bone margin adjacent to the ligament space) and "B" is the distance from the cement–enamel junction to the apex (Jacobs, et al. 1998). Mesial and distal

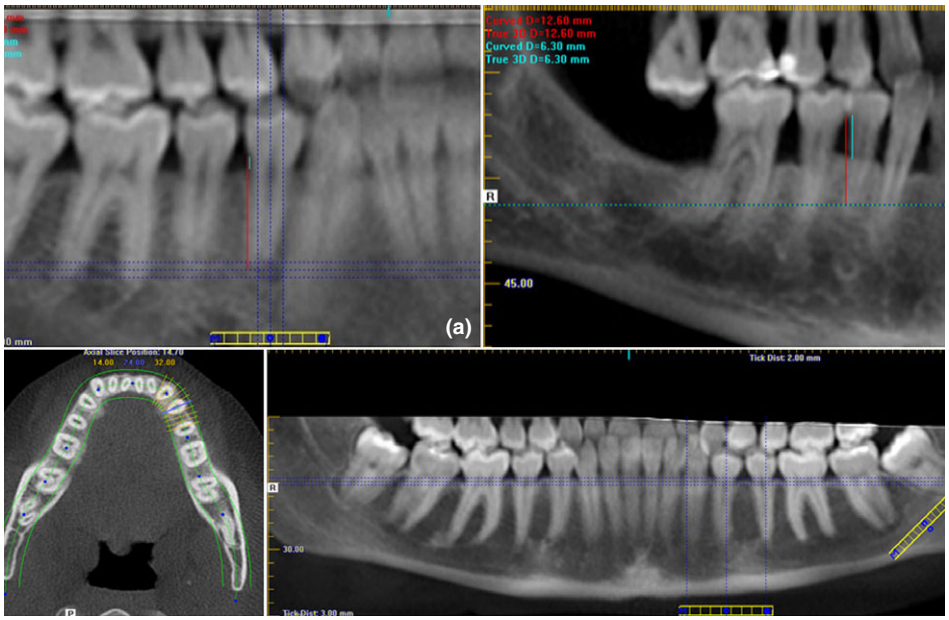

Fig. 2. Representatives CBCT from patients with adult periodontitis. Red line: distance between enamel-cement junction and radicular apex. Blue line: distance between enamel-cement junction and crestal bone. (a) Non-smoker patient. Red line: 12.9 mm; blue line: 1.5 mm. Alveolar crest height loss: 11.6%. (b) Smoker patient. Red line: 12.60 mm. Blue line: 6.30 mm. Alveolar crest height loss: 50% (c) Illustration of the digital imaging procedure used to obtain a plane from the CBCT image equivalent to the plane in a panoramic radiograph.

measurements were made on teeth 37, 41, and 44 (Ramfjord's mandibular teeth, dental nomenclature FDI) (Ramfjord, SP. 1959) or on the adjacent teeth when absent. Given the normal anatomical variance, bone loss was only considered when the distance from cement–enamel junction to alveolar crest was >1 mm, and a loss ≤10% was not included as bone loss in the subsequent analyses.

The MCW index was calculated by measuring a perpendicular line from the mental foramen through both mandibular cortical bones (Fig. 1); a single determination on one side is adequate, as reported by Taguchi, due to the close correlation between values on either side of the mandible (Taguchi et al. 1995).

Radiographic bone density was determined by calculating the optical density of a rectangular area (3 mm^2) in two regions of interest: alveolar bone in the premolar area vertical to the mental foramen without touching the cortex of the two premolars (AOD, Fig. 1) and basal bone mesial to the foramen and apical to the dental roots (BOD, Fig. 1). The image analysis software program calculated the bone density from mean gray levels, and this value was used as an estimation of the mandibular alveolar bone mass (Jonasson 2009). The coefficient of variation for placement of the rectangular device, measured after a 4-week interval, was 1.7%.

3D analysis

Of the 315 patients in the study, a preoperative planning 3D CBCT study was performed in the 110 who were candidates for dental implantation, using an i-Cat® tomograph (Imaging Sciences International, Inc, Hatfield, PA, USA) at 120 kV, 3–8 mA, and scan time of 20 s. Alveolar crest height and bone density (in gray levels) were determined by means of the equipment software (Imaging Sciences International, Inc). To obtain an image equivalent to a conventional panoramic radiograph, we reconstructed a 10-mm-thick curve along the alveolar crest and then made vertical measurements from this medial plane for each mandibular tooth of interest to determine the loss of alveolar crest height (Fig. 2a–c). The mean density of a 3-mm^2 region of interest in the premolar teeth perpendicularly above the mental foramen was recorded in gray levels (CBCT values). Twenty randomly selected 3D radiographs were examined (M.V-T) twice, 2 weeks apart, to determine the intra-examiner reproducibility; the intraclass correlation coefficient was 0.881, indicating good intra-examiner agreement.

Clinical assessment

Data on the age, sex, and tobacco consumption (cigs./day) of the patients were gathered from their clinical records. All patients with ACH loss had been diagnosed with periodontitis according to attachment loss, probing depth, and radiography findings.

Ethical considerations

The study was performed in accordance with the principles of the Declaration of Helsinki and was approved by the ethical committee of the School of Dentistry, University of Granada.

Statistical analysis

Sample Power 2.0 (SPSS Inc., Chicago, IL, USA) was used for the statistical power calculation, yielding estimated sample sizes of non-smokers ($n = 242$) and smokers ($n = 71$) to allow the detection of a standardized difference of 0.4 (between small – 0.2 – and moderate – 0.5) (Cohen 1988) in with a power of 84% ($\beta = 0.16$) and α error of 0.05.

SPSS Windows version 17.0 (SPSS Inc.) was used for the statistical analysis of data in Tables 1, 2, and 3 (M.B.); the tests used are reported in table footnotes. Multiple regression analysis (Table 4) was performed with ACH (by 2D and 3D analysis) as dependent variable and sex; age; tobacco use; nº teeth; MCW index; and AOD, BOD, and CBCT values as predictors, after converting them into dummy variables. Because the main purpose in this study was to analyze the adjusted effect of tobacco on ACH, we forced all variables in the model, after excluding CBCT due to the lack of data. This model was built for ease of use with SUDAAN 7.0 (RTI, RTP, Piedmont, NC,

Table 1. **Patient description and morphometric characteristics of the studied mandibles from 2D panoramic radiographs and 3D tomograms ($n = 315$)**

Variable	*n* (%)	Median	Mean ± SD
Sex			
Male	136 (43.2)		
Female	179 (56.8)		
Age (years)			
21–30	51 (16.2)	37.0	36.6 ± 5.3*
31–46	264 (83.8)		
Tobacco			
Non-smoker	242 (77.3)		
<10 cig./day	43 (13.7)		
10–20 ($n = 19$) and >20 ($n = 9$) cig./day	28 (8.9)		
Not known (*n*)	(2)		
Number of mandibular teeth present			
6–12	118 (37.5)	13.0	13.0 ± 2.0*
13–16	197 (62.5)		
Mandibular cortical width index (MCW) (mm)			
2.2–4.2	140 (44.4)	4.30	4.37 ± 0.95*
4.3–7.5	175 (55.6)		
Alveolar optical density (AOD) (%)			
794–1500	49 (15.6)	2038	2059 ± 517*
1501–2000	95 (30.2)		
2001–2500	105 (33.3)		
2501–3302	66 (21.0)		
Basal optical density (BOD) (%)			
758–1500	77 (24.4)	1859	1916 ± 546*
1501–2000	110 (34.9)		
2001–2500	80 (25.4)		
2501–3441	48 (15.2)		
Cone-beam computed tomography gray levels (CBCT)			
314–599	38 (34.5)	662	661 ± 155*
600–799	52 (47.3)		
800–1070	20 (18.2)		
Not collected (*n*)	(205)		
Alveolar crest height loss (ACH) (%)			
0–10	98 (31.1)	15.0	16.2 ± 11.2†
10.1–20	144 (45.7)		
20.1–40	61 (19.4)		
40.1–60	11 (3.5)		
60.1–80	1 (0.3)		

*Mean ± standard deviation. Mean is calculated from the original values (i.e., with no collapsing of categories).
†The mean was calculated by assigning the average value for each category (5 for 0–10, 15 for 10.1–20, 30 for 20.1–40, 50 for 40.1–60, and 70 for 60.1–80).

Table 2. **Univariate associations of studied variables with alveolar crest height loss (ACH) (*n* = 315)***

Variable	*n*	Alveolar bone loss (%) 0–10 (*n* = 98)	10.1–20 (*n* = 144)	20.1–40 (*n* = 61)	≥40.1 (*n* = 12)	Mean ± SD‡	*P*-value†
Sex							
Male	136	50.0	39.6	36.1	66.7	16.0 ± 12.6	0.830§
Female	179	49.0	60.4	63.9	33.3	16.3 ± 10.1	
Age (years)							
21–30	51	19.4	18.8	6.6	8.3	13.1 ± 8.8	0.012§
31–46	264	80.6	81.2	93.4	91.7	16.8 ± 11.6	
Mean ± SD¶		35.6 ± 5.3	36.3 ± 5.5	38.5 ± 4.6	38.0 ± 5.7		
Tobacco							
Non-smoker	242	81.6	79.6	67.2	66.7	15.5 ± 10.9	0.079**
<10 cig./day	43	11.2	12.7	21.3	8.3	17.8 ± 10.8	
10–20 and >20 cig./day	28	7.1	7.7	11.5	25.0	20.0 ± 13.9	
Not known (*n*)	(2)	–	(2)	–	–		
Number of mandibular teeth							
6–12	118	23.5	31.3	68.9	66.7	20.9 ± 12.8	<0.001§
13–16	197	76.5	68.7	31.1	33.3	13.3 ± 9.1	
Mean ± SD¶		13.5 ± 1.9	13.2 ± 1.9	11.8 ± 2.0	12.3 ± 1.6		
MCW index (mm)							
2.2–4.2	140	32.7	50.7	50.8	33.3	17.0 ± 10.2	0.225§
4.3–8.7	175	67.3	49.3	49.2	66.7	15.5 ± 12.0	
Mean ± SD¶		4.52 ± 0.89	4.28 ± 1.07	4.31 ± 0.72	4.59 ± 0.84		
AOD							
794–1500	49	17.3	12.5	19.7	16.7	17.0 ± 13.3	0.481**
1501–2000	95	31.6	33.3	23.0	16.7	14.7 ± 9.6	
2001–2500	105	28.6	36.8	31.1	41.7	16.7 ± 11.1	
2501–3302	66	22.4	17.4	26.2	25.0	16.9 ± 11.9	
Mean ± SD¶		2068 ± 558	2022 ± 470	2116 ± 561	2143 ± 508		
BOD							
758–1500	77	28.6	23.6	24.6	0.0	14.3 ± 9.0	0.245**
1501–2000	110	33.7	36.8	31.1	41.7	16.2 ± 11.1	
2001–2500	80	23.5	27.1	23.0	33.3	16.7 ± 12.3	
2501–3441	48	14.3	12.5	21.3	25.0	18.3 ± 12.6	
Mean ± SD¶		1896 ± 567	1877 ± 511	1982 ± 594	2211 ± 455		
CBCT							
314–599	38	40.0	36.7	22.7	25.0	14.2 ± 10.0	0.427**
600–799	52	45.7	38.8	72.7	25.0	17.2 ± 11.0	
800–1070	20	14.3	24.5	4.5	50.0	16.7 ± 12.8	
Mean ± SD¶		643 ± 151	669 ± 178	653 ± 95	781 ± 146		
Not collected (*n*)	205	63	95	39	8		

MCW, mandibular cortical width; AOD, alveolar optical density; BOD, basal optical density; CBCT, gray levels CBCT.
*The table shows the percentage distribution of different variables for each category of bone loss, together with quantitative descriptions when applicable.
§Calculated considering the alveolar crest height loss as quantitative variable.
‡The alveolar crest height loss (%) results are presented according to their distribution among the categories (0–10, 10.1–20, etc.) and also as mean ± SD, which was quantitatively calculated using the mean value of each category (5 for 0–10, 15 for 10.1–20, 30 for 20.1–40, etc.), without collapsing categories.
¶Student's *t*-test for independent groups.
†Mean ± standard deviation, with the original data (i.e., with no collapsing of categories).
**ANOVA.

USA) with the specification of SRS (simple random sampling).

Results

Imaging techniques were used to study 315 patients. Table 1 exhibits data on the patients and the morphometric description of the mandibles. In the univariate analysis (Table 2), ACH loss was greater in older patients ($P = 0.012$) and in those with fewer mandibular teeth ($P < 0.001$) and showed a relationship with tobacco consumption that was close to significant ($P = 0.079$). The multivariate analysis (Table 4) found the number of mandibular teeth ($P < 0.001$) and the tobacco consumption ($P = 0.048$) to be significantly associated with ACH loss. Alveolar and basal bone densities were associated with the number of mandibular teeth ($P = 0.012$) and the cortical width ($P = 0.030$), respectively (Table 3).

Discussion

The results of our radiographic observations in this large sample of cases indicate that cigarette smoking induces ACH loss independent of the mandibular bone mass or bone density, as confirmed by multiple regression statistical analysis. The percentage of smokers in our patient population (22.7%), which yielded an adequate sample size for this study (power of 90% [$\beta = 0.10$], $\alpha = 0.05$), was consistent with the proportion of smokers in the general adult population of Spain (26.2%) according to a Europe-wide survey in 2009 (INE 2009 www.msps.es/estadEstudios/estadisticas/EncuestaEuropea/Nota_tecnica_EES09.pdf), supporting the validity and representativeness of our study. Bergstrom (2004) studied the long-term influence of chronic smoking and concluded that it accelerates the periodontal bone height reduction rate and that cessation of the habit produces a return toward the rate observed in non-smokers. In a study of young people, Levin & Levine (2010) found that smoking had adverse effects on clinical periodontal variables and on alveolar bone height and density, acting as a potential risk factor for

Table 3. **Association of different variables with AOD and BOD* (*n* = 315)**

	AOD		DOB	
Variable	Mean ± SD	*P*-value	Mean ± SD	*P*-value
Sex				
Male	2067 ± 475	0.807*	1891 ± 525	0.476*
Female	2053 ± 548		1935 ± 562	
Age (years)				
21–30	2074 ± 524	0.820*	1904 ± 588	0.875*
31–46	2056 ± 517		1918 ± 538	
Tobacco				
Non-smoker	2062 ± 525	0.933†	1940 ± 544	0.361†
<10 cig./day	2033 ± 400		1823 ± 520	
10–20 and >20 cig./day	2074 ± 633		1857 ± 615	
Number of mandibular teeth				
6–12	2106 ± 538	0.225*	2016 ± 554	0.012*
13–16	2031 ± 504		1856 ± 533	
MCW index (mm)				
2.2–4.2	1988 ± 518	0.030*	1858 ± 570	0.097*
4.3–8.7	2116 ± 511		1962 ± 522	

AOD, alveolar optical density; BOD, basal optical density; MCW, mandibular cortical width.
*Student's *t*-test for independent groups, with original values (without collapsing categories) of AOD and BOD.
†ANOVA.

Table 4. **Multivariate linear regression model* with alveolar crest height loss (ACH) as dependent variable† (*n* = 313‡)**

Variable	β ± SE§	*P*-value
Male (female as reference)	1.2 ± 1.3	0.350
Age 21–30 years (31–46 as reference)	−1.7 ± 1.4	0.247
Tobacco (non-smoker as reference)		
10–20 and >20 cig./day	4.5 ± 2.4	0.048
<10 cig./day	3.0 ± 1.5	
Mandibular teeth 6–12 (13–16 as reference)	7.2 ± 1.4	<0.001
MCW 2.2–4.2 (4.3–8.7 as reference)	0.8 ± 1.2	0.479
AOD (2501–3302 as reference)		
794–1500	3.5 ± 3.2	0.180
1501–2000	−0.5 ± 2.1	
2001–2500	1.4 ± 1.8	
BOD (2501–3441 as reference)		
758–1500	−4.1 ± 2.6	0.483
1501–2000	−2.0 ± 2.2	
2001–2500	−1.4 ± 2.0	
Constant	13.2 ± 2.0	

MCW, mandibular cortical width; AOD, alveolar optical density; BOD, basal optical density.
*Built forcing all variables, after excluding CBCT due to lack of information for this variable (205 missing values).
†Considered as quantitative, with assignation of the average value for each category (5 for 0–10, 15 for 10.1–20, 30 for 20.1–40, etc.).
‡After excluding two patients with missing tobacco information.
§Coefficient ± standard error.

alveolar bone loss, even with low tobacco consumption. In populations of post-menopausal women, Rosa et al. (2008) and Payne et al. (2000) showed that a cigarette smoker was more likely to lose alveolar bone height and density in comparison with a non-smoker with a similar degree of periodontitis. In contrast, the present study of a large non-menopausal population shows that smoking was only significantly related to ACH loss and that this loss was not related to bone mandibular density.

No data were available on the radiographic assessments of the patients, but all patients with alveolar bone loss had been diagnosed with periodontitis, and the reduction in alveolar height was due to inflammation and not due to physiological remodeling from tooth loss or mesial drift (exclusion criteria). Chronic periodontitis is a slowly progressive disease, and the ACH loss represents the accumulated effect of periodontitis from its onset (Rutger Persson et al. 2003). In the present study, we also examined whether the ACH loss digitally evaluated on radiographs was associated with a reduction in mandibular bone mass or in alveolar or basal bone optical density that might exacerbate alveolar bone destruction. The use of absolute linear measures is not the method of choice in panoramic radiographs, unless the radiographs are standardized (Muller et al. 2005). Pepelassi et al. (2000) reported that periapical radiography was better than panoramic radiography for the detection of osseous defects. However, neither the mental foramen nor the mandibular cortical bones can be visualized using periapical radiography, and both are necessary to calculate the indexes used in this study. We excluded radiographs with poor visibility of the alveolar bone level adjacent to the teeth. Furthermore, the loss of ACH was expressed relative to the root length, thereby obviating the need to localize other reference points. Finally, although periapical radiographs are preferable for the detection of osseous defects in individual teeth, we consider that superior standardization is achieved with panoramic radiographs in population studies of Ramfjord's mandibular teeth.

It is well known that considerable optical density information is lost during the scanning process (Schulze et al. 2002). However, we scanned at a high resolution (600 dpi), and the loss of information would have affected all panoramic radiographs equally and would not affect the validity of our comparative data.

The MCW index, selected as radiomorphometric index, has been significantly associated with the bone mass density of the skeleton in general (e.g., spine and femur) and with biochemical markers of bone turnover (Taguchi et al. 1996; Karayianni et al. 2007; Morita et al. 2009; Leite et al. 2010; Taguchi 2010). Measurements in this anterior area of the mandible have proven more accurate to predict bone mass alterations in comparison with those at the angle or ramus (Morita et al. 2009; Horner et al. 1996), and they are more easily performed due to the lack of superposition of anatomical structures.

Our findings revealed no significant difference in mean MCW index between the patients with and without ACH loss according to the radiographic findings. The group with greatest bone loss showed the highest mean MCW index, but only 12 patients were in this group.

Over the past 10 years, only three clinical studies and one radiological study of periodontitis have compared the use of different methods (as in the present study) to examine the relationship between clinical attachment/alveolar bone level and bone mass density at various sites. von Wowern et al.

(2001) determined the density at mandible and forearm in 24 young adults with severe periodontitis and reported periodontitis to be a local disorder not associated with systemic bone mineral alterations, although no control group was included and there was no consideration of the influence of smoking, gender, or other potential confounders. Inagaki et al. (2001) studied metacarpal bone density in 190 Japanese women and found a relationship between periodontitis and reduced bone mass in both pre- and post-menopausal subjects. Hattatoglu-Sonmez et al. (2008) used dual-energy X-ray absorptiometry to study lumbar vertebrae and left hip joint and found no association between periodontal clinical variables and bone mass density in 85 premenopausal women, although they did not radiographically verify the alveolar bone levels. Nackaerts et al. (2008) explored the relationship between the radiographic bone quality on digital panoramic X-rays and the relative alveolar bone level in 94 women and found a weak but significant negative correlation between the mandibular bone density and percentage loss of alveolar bone height; however, they did not stratify the results by age or menopausal status.

Various studies have confirmed a greater destruction of the alveolar bone, increased clinical attachment loss, and higher percentage of tooth mobility and furcation among heavy smokers (Bergstrom 2004; Levin & Levine 2010; Rudziniski 2010; Rudzinski & Banach 2011). We found no significant correlation between ACH and bone density (gray levels) measured by applying specific imaging software to 2D panoramic radiographs or 3D tomograms. These results suggest that the inflammation responsible for the alveolar bone destruction in cigarette smokers with periodontitis is not influenced by the density of underlying bone structures. According to the present findings, alveolar bone destruction in periodontal patients who smoke cannot be predicted from radiographic mandibular bone density findings.

Conclusions

In this Caucasian population of men and women between 21 and 46 years, tobacco consumption showed a significant relationship with ACH loss. However, ACH loss showed no significant relationship with mandibular bone mass estimated either as the mandibular cortical width index or bone density on digital panoramic radiographs or as the bone density on cone-beam computed tomograms. In our series, ACH loss was a local event independent of mandibular bone mass status. These findings support the proposition that periodontal disease is a localized disorder and not associated with systemic bone density status.

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