

Predictors of long-term outcomes in patients undergoing periodontal maintenance

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Abstract

Aim: This retrospective study aimed to characterize the baseline status of patients following periodontal maintenance, analysing the association between the long-term outcome of these patients, smoking, bruxism, and the main clinical and radiographic variables.

Material and methods: A sample of 174 patients with moderate to severe periodontitis was refined into homogeneous subsamples according to smoking and bruxism and the rate of tooth loss due to periodontal disease (TLPD): 0, 1–2, and >2 teeth. The association and the distribution (χ^2 test) of the variables within the subsamples were analysed.

Results: Smoking and bruxism were significantly associated with higher TLPD rates. Vertical and circumferential bone defects ($p < .0001$), and abfractions ($p < .0001$) were associated with bruxism and particularly with bruxism and TLPD >2.

Furcation defects ($p = .0002$), fewer radio-opaque subgingival calculus ($\chi^2 p < .0001$), a lower mean Gingival index ($\chi^2 p = .027$), and increased mean recessions >1.5 mm ($\chi^2 p = .0026$) were associated with smoking and higher TLPD rates. The mean baseline mobility, abfractions, and recessions characterized two basic types of TLPD.

Conclusions: Smoking, bruxism, and routine clinical and radiological parameters can be used to characterize the baseline status of patients with worse outcomes.

KEYWORDS

abfractions, bruxism, periodontal disease, periodontal prognosis, tooth loss

1 | INTRODUCTION

The identification of patients at risk of presenting a worse outcome and experiencing higher rates of tooth loss due to periodontal disease (TLPD) is a major research goal in periodontal prognosis. There are two distinct approaches to addressing the issue: characterizing the baseline status of patients with higher rates of TLPD during periodontal maintenance (PM), and assessing risk of disease progression and the resulting TLPD.

The study by Martínez-Canut (2015) analysed a subsample of 85 patients with higher TLPD rates and reported that these patients were

characterized by severe periodontitis (OR 3.8–7.1), smoking combined with bruxism (OR 3.8), fewer baseline teeth, and a younger age.

Several risk assessment tools in the second approach have been developed based on well-documented risk factors and have been assessed in longitudinal studies (Lang & Tonetti, 2003; Lindskog et al., 2010; Page et al., 2002; Page, Krall, Martin, Mancl, & Garcia, 2002) and their ability to identify patients with different levels of risk has been established (Lang, Suvan, & Tonetti, 2015).

However, due to incomplete knowledge on the subject, several risk factors have been analysed to predict the outcomes of patients

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following PM without considering possible differences between risk and prognostic factors (Martinez-Canut, 2015) and utilizing both terms interchangeably (Fardal, Grytten, Martin, Houlihan, & Heasman, 2016).

Baseline subgingival calculus and gingival inflammation are well-known risk factors whose role as prognostic factors has yet to be elucidated. The pioneer study characterizing patients under PM found that better outcomes were correlated with increased gingival inflammation while subgingival calculus did not correlate with a worse outcome (Wasserman & Hirschfeld, 1988).

The intra-bony component of a vertical defect has been associated with a reduced probability of tooth loss (Muzzi et al., 2006), whereas certain tools consider vertical defects to be a risk factor (Lindskog et al., 2010; Page et al., 2002). Data supporting the latter are limited to an animal study (Lindhe & Svamberg, 1974) and a study in a non-treated population in which the vertical defects were associated with further bone loss (Papapanou & Wennström, 1991).

Only two studies have addressed the association between bruxism and TLPD in patients following PM (Martinez-Canut, 2015; McGuire & Nunn, 1996). The results of these studies were consistent and showed that bruxism doubled the risk of TLPD, which is similar to smoking. However, bruxism is poorly understood and represents one of the most controversial issues in dentistry. Consequently, it is a matter that deserves serious scientific discussion (Manfredini, Ahlberg, Mura, & Lobbezoo, 2015; Manfredini, Mura, & Lobbezoo, 2016; Perlitsh, 2016).

A recent review (Manfredini, Winocur, Guarda-Nardini, Paesani, & Lobbezoo, 2013) found that the prevalence of bruxism in the general population is approximately 25%, whereas a twofold rate of bruxism (264 patients out of 500, 53%) has been reported in periodontal patients (Martinez-Canut, 2015). Therefore, the role of bruxism merits further research.

This retrospective study aimed to characterize the baseline status of patients following PM analysing the association between the long-term outcome of these patients, smoking, bruxism, and the main clinical and radiographic variables. These variables were gingival inflammation based on the GI, gingival recession, presence of radiopaque subgingival calculus (C+), tooth wear (incisal and occlusal attrition and abfractions), VCDs, FDs, and increased tooth mobility.

2 | MATERIAL AND METHODS

2.1 | Study population

The sample of this study consisted of 174 PM patients who were followed for a mean of 20.2 year (± 2.4). These patients were selected from the baseline sample of our previous study (Martinez-Canut, 2015) with the following criteria:

The inclusion criteria were the diagnosis of moderate and severe chronic periodontitis (Armitage, 1999), the absence of previous periodontal treatment and complete records on periapical radiographs at baseline, periapical radiographs of TLPD during the follow-up and intra-oral photographs at baseline and at the end of follow-up. The exclusion criteria were mild periodontitis, aggressive periodontitis

Clinical Relevance

Scientific rationale for the study: Certain patients experience higher tooth loss rates during periodontal maintenance. The purpose of this study was to characterize the baseline status of these patients.

Principal findings: Higher rates of tooth loss were associated with bruxism, smoking, and certain features associated with bruxism (vertical bone defects and abfractions) and smoking (FDs, less radiographically visible subgingival calculus and decreased gingival inflammation).

Practical implications: In combination with smoking and bruxism, several clinical and radiographical parameters were useful in characterizing the baseline status of patients in relation to their final long-term outcome, which enabled accurate predictions.

(Armitage, 1999), less than 36 and more than 70 years of age, the presence of serious disease with an influence on the periodontium, more than 6 non-replaced missing teeth and extensive restorations with natural teeth and implants.

2.2 | Treatment rendered and PM regimen

The active periodontal treatment and PM, which were previously described (Martinez-Canut, 2015) were similar for all of the patients and included oral hygiene instructions, scaling and root planing under local anaesthesia, and surgical treatment (modified Widman flap, osseous resective surgery, root resection, and periodontal regeneration) in 82% of the patients. Systemic antibiotic therapy with amoxicillin plus clavulanic acid, metronidazole or azitromycin was prescribed in 25% of the cases, corresponding to the most severe cases of periodontitis.

PM was scheduled every 4 months and soon after the intervals were shortened or lengthened by 1 or 2 months according to changes in probing pocket depth and or bleeding upon probing.

2.3 | Data collection

2.3.1 | Medical history

The patients completed a medical history questionnaire upon the baseline examination, and the health status was updated during the follow-up period.

2.3.2 | Clinical findings

Two clinical parameters were obtained from the patients' charts by one of the authors (M-C):

Baseline number of teeth, excluding teeth extracted during active periodontal treatment and third molars.

Tooth mobility (Lindhe & Nyman, 1977), for which a mean value was calculated for the whole dentition to identify patients with generalized increased mobility.

The following clinical parameters were recorded (M-C) at baseline and at the end of the follow-up period and they were on the basis of baseline records from the patients' charts and the intra-oral photographs taken at baseline and at the end of follow-up. Only the vestibular surface, from the second premolars to the central incisors, was evaluated.

Gingival recession: The distance from the cemento–enamel junction to the gingival margin. The maximum width of the clinical crown of an upper central incisor was the reference value for applying a rule of three to measure gingival recession on the photographs, which were scanned and magnified, using a computer.

Gingival inflammation: GI by Loe and Silness (1963) was routinely recorded at baseline in all patients on the vestibular surface of the teeth and confirmed with magnified photographs as described for gingival recession.

2.3.3 | Radiographic findings

A complete set of baseline periapical radiographs for each patient was examined by the authors (LI & M-C) in a darkened room, using a radiographic screen (67-0442, Dentsply Rinn, Elgin, IL, USA) and 2.5× magnification, to identify the presence and number of VCDs, the presence and degree of FDs (the most affected furcation entrance for each molar), and the presence of interproximal C+.

2.3.4 | Bruxism

The presence of bruxism was identified according to criteria previously described (Martinez-Canut, 2015), based on the self reported habits, confirmed during the follow-up, together with signs of tooth wear. A complementary reevaluation was performed under a multidisciplinary approach that included an expert in bruxism and orofacial pain (R), a prosthodontist (LI), and a periodontist (M-C). Consensus was required to identify probable bruxism (Lobbezoo et al., 2012). Baseline and final photographs of the whole sample were examined under magnification to register incisal, occlusal, and cervical tooth wear (Tooth Wear Index by Smith & Knight, 1984). Particular attention was paid to differentiating clenching from grinding.

2.3.5 | Smoking habits

Our previous research (Martinez-Canut, 2015) did not find significant differences between non-smokers and light smokers (less than 10 cigarettes per day); therefore, only heavy smokers of 10 or more cigarettes per day were considered smokers. The actual habit was confirmed during the follow-up period. Smokers who had quit for more than 5 years were considered non-smokers.

2.3.6 | Assessment of tooth loss and TLPD

For the extracted teeth, a clinical and/or a radiological evaluation was performed immediately prior to the extraction, to identify the reasons for tooth loss, which was classified as either TLPD or tooth loss because of other reasons.

The criteria to define TLPD were spontaneous exfoliation and bone loss >75% with grade III mobility, which caused pain under function or spontaneously. For molars, bone loss >50% associated with furcation lesion grade III and repeated abscesses. Teeth extracted for restorative purposes with bone loss >75 and grade III mobility, as well as endodontic complications with bone >75% without caries or root fracture were considered TLPD.

2.4 | Inter- and intra-examiner agreement

Intra-examiner agreement (clinical parameters) and inter-examiner agreement (radiological parameters) was verified (Kappa statistic).

2.5 | Statistical analysis

Data entry and descriptive and analytical statistical evaluations were performed by independent statisticians (ERATEMA, I.A & L.D.) utilizing the SSPS software program (IBM, SPSS Statistics, V.19, Armonk, NY, USA). The statistical analysis identified significant associations and differences in the distribution of the occurrence of the variables under study. Each subsample with an outcome of TLPD 0, 1–2 and >2 teeth was refined according to the smoking and bruxism status. Mann–Whitney *U* and Kruskal–Wallis tests were utilized for continuous variables with a non-normal distribution (abfractions, VCDs, FDs, and gingival recession) and ANOVAs and *t*-Student's tests were utilized for continuous variables with a normal distribution (GI).

The χ^2 test was used to analyse the distribution of the occurrence of the following categorical variables: presence or absence of C+; VCDs 0, 1–2 and more than 2; FDs grade 0–I, II and III, abfractions 0, 1–4 and more than 4; GI <1.7 and \geq 1.7 and gingival recession <1.5 and \geq 1.5 mm. The homogeneity of the subsamples was also evaluated. The significance level was set at $\alpha = .005$.

3 | RESULTS

3.1 | Patient's sample

The mean age of the patient's sample was 43.1 years (*SD* 6.95), and the age ranged from 36 to 70 year old. In addition, 102 patients were females (58.6%) and 72 males (41.4%). The subjects were mostly Caucasian and of European origin (98%) and had a high to middle socio-economic level. None of the patients had previously undergone periodontal treatment.

3.2 | Homogeneity of the samples

The TLPD groups 0, 1–2, and >2 teeth, as well as the groups characterized according to smoking and bruxism were homogeneous for age

and gender, but not for severe periodontitis, which was more prevalent as TLPD increased ($\chi^2 = .002$) and with smoking ($\chi^2 = .012$).

3.3 | Inter- and intra-examiner agreement

Intra- and inter-examiner agreement was well above the level of chance at 0.88–0.95 (kappa statistic $p < .001$ for individual variables).

3.4 | Distribution of the variables in the sample

Table 1 shows the main characteristics of the sample and differentiates the TLPD subsamples 0, 1–2, and more than 2 teeth, which were distributed according to smoking (in 63 patients, 36.2%) and bruxism (in 117 patients, 67.2%), either isolated or combined. The figures for the remaining variables under study are detailed, per patient as follows: mean GI, mean gingival recession, mean VCDs, mean FDs, presence of C+, and mean abfractions. These variables are distributed within the subsamples smoking positive/negative and bruxism positive/negative, depending on their statistical association with smoking and bruxism.

Seventy-four patients (51.3%) did not lose teeth, 45 (25.7%) lost 1–2 teeth and 55 (31.6%) lost more than 2 teeth. As the TLPD increased, the prevalence of patients with heavy smoking and bruxism increased, especially when combined. For TLPD >2, 3 patients (5.4%) did not present smoking and bruxism while 37 patients (67.2%) presented both factors.

3.5 | Analysis of the distribution of the occurrence of the variables under study

The following variables were significantly associated and could be used to determine the distribution of the occurrence of the variables.

Smoking ($\chi^2 = .001$) and bruxism ($\chi^2 = .0001$) were associated with increasing TLPD rate. The impact was much higher for smoking combined with bruxism ($\chi^2 < .0001$) resulting in a prevalence of TLPD >2 which was much higher than for each factor in isolation (Table 2).

The VCDs mean was three- to four-times higher with bruxism and higher TLPD rates, increasing in accordance with the TLPD rate ($p < .0001$, $\chi^2 = .028$ for TLPD 1–2 and = 0.004 for TLPD >2) and with bruxism ($p = .0001$; Table 1).

The mean FDs was three to four times higher with smoking and higher TLPD rates, increasing along with the TLPD rate ($p < .0001$, $\chi^2 = .032$ for TLPD 1–2 and = 0.005 for TLPD >2) and with smoking ($p = .0002$).

Abfractions mean increased with bruxism in the entire sample ($p < .0001$) and with bruxism for TLPD 0 ($p < .0001$, $\chi^2 < .0001$), and >2 ($p = .002$, $\chi^2 = .006$).

The prevalence of C+ decreased as TLPD increased and decreased with smoking. It was 75% for TLPD 0 and 1–2 teeth in non-smokers and 11.9% for TLPD >2 teeth in smokers. Here C+ increased in non-smokers in the entire sample ($p = .0001$) and in non-smokers with TLPD >2 ($\chi^2 = .003$, four times more prevalent).

Mean gingival recession increased as the TLPD rate increased ($p = .026$) and a mean recession >1.5 mm was significant for TLPD >2 ($\chi^2 = .031$).

The mean GI slightly decreased with smoking and as TLPD increased. Conversely, for TLPD 0 and 1–2 teeth, the prevalence of a mean GI ≥ 1.7 was four times higher in non-smokers. Mean GI <1.7 was more prevalent in smokers ($\chi^2 = .027$) and for TLPD 1–2 ($\chi^2 = .007$).

Table 2 depicts the associations between TLPD, FDs, and VCDs in the four subsamples depending on smoking and bruxism.

Figure 1A–L presents the 20–28 year follow-up of patients according to the TLPD rate, smoking, and bruxism.

3.6 | Characterization of patients losing more teeth

Among the 51 patients presenting the highest TLPD rate, the mean baseline tooth mobility ($p < .0001$ and $\chi^2 < .0001$ for mobility < and ≥ 1) enabled two distinct profiles of patients to be differentiated (both groups were homogeneous for age, gender and severe periodontitis), which were complementarily characterized by differences in abfractions ($p = .009$ and $\chi^2 = .003$) and in gingival recession ($p = .001$ and $\chi^2 = .039$ for mean recession < and ≥ 1). Therefore, as shown in Table 3, 26 patients (termed type 1), presented a baseline mean tooth mobility of 0.30, mean gingival recession of 1.21 and mean abfractions of 5.38, whereas the second group of 25 patients (termed type 2), presented a fivefold higher mean mobility of 1.31, close to a twofold higher gingival recession and an almost twofold lower mean abfractions. The mean TLPD rate of the type 2 patients was one tooth higher.

The clinical features of some of these patients is presented in Figures S1–S3. Figure S4 depicts the magnified image of several emerging abfractions. More detailed information on the role of bruxism and occlusal overload in these patients is presented in the supplementary material (Appendix S1).

3.7 | Distribution of centric and eccentric bruxism, tooth wear, tooth loss, and complications

Only 10 (8.5%) out of 117 patients with bruxism presented eccentric bruxism (incisal and occlusal wear 2 and 3) with flattening of the incisal and occlusal planes. The remaining 107 patients (91.4%) presented centric bruxism with occlusal wear 1 (wear facets). Group function and lack of canine guidance were common findings at baseline and almost the rule by the end of the follow-up period.

Abfractions were much more clearly associated with bruxism than occlusal wear. Table 4 details the mean abfractions and associated categories, in patients with and without bruxism, at baseline and at the end of the follow-up period. Abfractions were four times more prevalent and increased twice as much in degree in bruxists compared to non-bruxists. The baseline abfractions doubled in prevalence and degree at the end of the follow-up, and some of these final lesions emerged with a localized gingival recession, in the shape of Stillman's cleft (Stillman, 1921).

TABLE 1 Distribution of the baseline sample according to the number of teeth lost (TLPD 0, 1–2 and more than 2 teeth). Each one of these three samples is distributed according to the presence of heavy smoking and bruxism, either isolated or combined (subsamples S & B). It is detailed, per patient, the figures of the remaining variables under study: mean Gingival Index, mean gingival recession, mean furcation defects grade II and III, mean vertical defects, presence of radio-opaque calculus and mean abfractions. Changes in gingival recession and in abfractions during the follow-up are also presented

	Subsamples		n. pts. (%)	Subg. calculus	Mean Ging Index	n. pts (%) Ging Index <1.7	n. pts (%) Ging Index ≥ 1.7	Mean FDs II and III/pt	Subsamples S & B	VCDs/pt	n. (%) pts. >2 VDs	Mean (ED) abfractions 1	Mean (ED) abfractions 2	Mean Ging. rec
	S & B	S & B												
TLPD 0														
Total	74 (100%)				2.08 (0.4)			0.45 (1.1)		0.54 (1.1)		2.64 (2.44)	5 (4.10)	1.22 (0.98)
S– B–	28 (37.8%)	S– (n.65 pts)	48 (73.8%)		2.10 (0.4)	16 (24.6%)	49 (75.4%)	0.42 (1)	B– (n.34 pts)	0.56 (1.3)	3 (8.8%)	0.88 (1.49)	1.07 (1.73)	
S– B+	37 (50%)													
S+ B–	6 (8.1%)	S+ (n. 9 pts)	4 (44.4%)		1.91 (0.32)	3 (33.3%)	6 (66.7%)	0.53 (1.3)	B+ (n. 40 pts)	0.53 (0.93)	2 (5%)	3.80 (2.30)	7.43 (3.12)	
S+ B+	3 (4%)													
TLPD 1–2														
Total	45 (100%)				1.94 (0.47)			1.32 (1.4)		1.24 (1.55)		2.24 (2.22)	6.28 (3.37)	1.22 (0.77)
S– B–	10 (22.2%)	S– (n. 33 pts)	25 (75.7%)		2.02 (0.41)	8 (24.2%)	25 (75.8%)	0.7 (0.82)	B– (n. 15 pts)	0.4 (0.74)	0 (0%)	1.47 (1.77)	4.63 (2.26)	
S– B+	23 (51.1%)													
S+ B–	5 (11.1%)	S+ (n. 12 pts)	6 (50%)		1.74 (0.57)	6 (50%)	6 (50%)	1.42 (1.32)	B+ (n. 30 pts)	1.67 (1.69)	10 (33.3%)	2.63 (2.34)	6.90 (3.55)	
S+ B+	7 (15.5%)													
TLPD >2														
Total	55 (100%)				1.87 (0.47)			2.48 (2.32)		2.51 (2.13)		4 (3.75)	7.62 (3.80)	1.61 (1.06)
S– B–	3 (5.4%)	S– (n. 13 pts)	7 (53.8%)		2.02 (0.52)	6 (50%)	6 (46.2%)	1.1 (1.26)	B– (n. 8 pts)	0.62 (1.19)	1 (12.5%)	1 (1.51)	4 (4.25)	
S– B+	10 (18.1%)													
S+ B–	5 (9.1%)	S+ (n. 42 pts)	5 (11.9%)		1.83 (0.45)	19 (45%)	23 (54.8%)	2.63 (2.2)	B+ (n. 47 pts)	2.83 (2.1)	26 (55.3%)	4.51 (3.79)	8.41 (3.54)	
S+ B+	37 (67.2%)													

n. pts, number of patients; TLPD, Tooth loss due to periodontal disease; S, heavy smoking; B, bruxism; Sub, calculus, presence of radiopaque subgingival calculus; FDs II and III, furcation defects grade II and III; VDs, vertical and circumferential bone defects; abfractions at baseline (1) and at the end of the follow-up (2); mean Ging. rec, mean gingival recession. The variables under analysis are distributed within the subsamples smoking and bruxism depending on their statistical association with these factors: FDs are associated with smoking while VCDs are associated with bruxism.

TABLE 2 Distribution of TLPD, VCDs and FDs (mean values and categories of each variable) in the four subsamples according to bruxism and smoking

		n. pts	Total	S+/B+	B+	S+	S-/B-
Total		174	100%	47 (100%)	70 (100%)	16 (100%)	41 (100%)
TLPD	TLPD 0	74	(42.5%)	3 (6.4%)	37 (52.9%)	6 (37.5%)	28 (68.3%)
	TLPD 1-2	45	(25.9%)	7 (14.9%)	23 (32.9%)	5 (31.2%)	10 (24.4%)
	TLPD >2	55	(31.6%)	37 (78.7%)	10 (14.3%)	5 (31.2%)	3 (7.3%)
	Mean TLPD		1.77 (2.14)	3.94 (2.32)	1.07 (1.51)	1.63 (1.63)	0.54 (0.92)
VDs	VDs 0	92	(52.9%)	11 (23.4%)	38 (54.3%)	10 (62.5%)	33 (80.5%)
	VDs 1-2	40	(23.0%)	12 (25.5%)	18 (25.7%)	4 (25%)	6 (14.6%)
	VDs >2	42	(24.1%)	24 (51.1%)	14 (20%)	2 (12.5%)	2 (4.9%)
	Mean VDs		1.34 (1.81)	2.68 (2.15)	1.11 (1.50)	0.88 (1.45)	0.39 (1)
FDs	FDs 0-I	61	35%	3 (6.3%)	36 (51.4%)	1 (6.25%)	21 (51.2%)
	FDs 0-I and II	34	19.5%	8 (17%)	13 (18.5%)	4 (20%)	9 (21.9%)
	FDs II and III	79	45.4%	36 (76.5)	21 (30%)	11 (68.7%)	11 (26.8%)
	Mean FDs II		1.17 (1.4)	2 (1.7)	0.5 (1.2)	1.3 (1.5)	0.4 (1.1)
	Mean FDs III		1.32 (1.69)	2.5 (2.1)	0.42 (1)	1.1 (1.3)	0.36 (1)

B, bruxism; FDs, furcation defects; S, smoking; TLPD, Tooth loss due to periodontal disease; VCDs, vertical and circumferential bone defects.

The majority of teeth lost in bruxists were those lacking abfractions. Only 39 (12.6%) out of the 308 teeth extracted presented grade II and III abfractions. The teeth lost presented a widened periodontal space and a VCD.

Abfractions developed to a lesser extent in 8 out of 10 patients with eccentric bruxism (occlusal wear 2 and 3). Abfractions 2 and especially 3 developed on either the upper or the lower arches, but not on both. These lesions were extremely uncommon in mobile and pathologically migrated teeth.

Eighty-three patients (80%) presented acute symptoms of diffuse pain, 15 crown and root fractures (in 19 teeth, 12.8%), and 2 horizontal implant fractures.

3.8 | Development of an index to predict TLPD

Based on the analysed variables, a predictive index for TLPD that consisted of a simple addition of one score for each variable involved was developed. Thus, the final value ranged from the presence of 0-5 of the following variables: fewer C+ deposits, a GI below 1.7, VCDs and/or FDs grade II and III, mean gingival recession >1.5 mm and abfractions.

Table 5 shows the distribution of patients according to the index. For TLPD 0, 71 patients out of 74 (96%) presented an index of 0-2. For TLPD >2, 45 out of 51 (97.7%) presented an index of 3-5.

The mean TLPD corresponding to an index of 4 and 5 were, respectively, 2.7 and 4.6. For TLPD >2 teeth, index values of 3, 4, and 5 matched the number of teeth lost ± 1 in 43 out of 55 patients (78.1%). The higher the value of the Index, especially with bruxism and smoking, the higher the resulting TLPD rate, and the accuracy of the Index (Spearman correlation .680, $p = .0001$).

4 | DISCUSSION

The inclusion and exclusion criteria of this study were defined to overcome the inherent limitations and potential source of bias of a retrospective study design. This approach enabled gathering a patient's sample with reliable clinical and radiographic records. The final clinical photographs were actually useful to ensure the identification of bruxism in the long-term and under a multidisciplinary approach.

The present research has analysed the long-term outcome of patients following PM according to smoking, bruxism, and certain characteristic features associated with both factors. This approach provided a better understanding of bruxism, which lacked on longitudinal studies and defined criteria for identifying it, especially clenching.

Smoking has been found associated with higher TLPD rates in the present research, consistently with the results of many studies in patients undergoing PM for more than 5 years (McGuire & Nunn, 1996, 1999; König, Plagmann, Rühling, & Kocher, 2002; Fardal, Johannessen, & Linden, 2004; Chambrone & Chambrone, 2006; Eickholz, Kaltschmitt, Berbig, Reitmeir, & Pretzl, 2008; Jansson & Lagervall, 2008; Tsami, Pepelassi, Kodovazenitis, & Komboli, 2009; Leininger, Tenenbaum, & Davideau, 2010; Ravald & Starkhammar Johanson, 2012; Costa et al., 2014; Salvi et al. 2014, Martinez-Canut, 2015; Dannewitz et al., 2016), with few exceptions (Tonetti, Muller-Campanile, & Lang, 1998; Matthews, Smith, & Hanscom, 2001; Carnevale, Cairo, & Tonetti, 2007; Matuliene et al. 2010, Bäumer et al., 2011).

The increase on the risk of tooth loss reported in some of these studies (1.8, 2.1, 2.9, 3.3, 4, 5, and 8) was usually lower than the one reported in general population (2.5, 4, 7.2, and 14.1) (Bergstrom, 1989; Linden & Mullaly, 1994; Grossi et al., 1995; Tomar & Asma, 2000). When only heavy smoking was analysed, the increase on the



FIGURE 1 (a–d) are cases of TLPD 0 in non-bruxists, (a and b) present a wider periodontium in a 26 year follow-up in a non-smoker (a) and a 20 year follow-up in a smoker (b). (c–d) present a narrower periodontium with developing gingival recessions in non-smoking patients after 20 (c) and 23 years (d). (e and f) are TLPD 0 cases (after 20 and 22 years) of mild eccentric bruxism progressing to moderate bruxism (protrusive grinding) in a non-smoking patient (e) and in a smoking patient with abfractions grade (f). (g, h, and i) (after 20, 22 and 25 years, respectively) depict moderate eccentric bruxism progressing to severe bruxism in a non-smoking patients with TLPD 0 (g), TLPD 1–2 teeth (h) and TLPD >2 teeth (i). These three patients as well as the remaining cases of eccentric bruxism developed recession exclusively in the area of abfractions. Besides, these lesions developed to a lesser extent. (j, k, and l) (after 28, 20 and 26 years, respectively) are cases of mild to moderate centric bruxism with attrition mostly grade 0 and 1 progressing to severe centric bruxism with attrition grades 0–2 and abfractions grades 2–4. TLPD was (j), 1–2 teeth (k) and >2 teeth (l)

risk was around 2 in patients following PM (Martinez-Canut, 2015; McGuire & Nunn, 1999) as compared to 7 in the general population (Grossi et al., 1995).

These findings might reflect, to some extent, certain differences on the impact of a prognostic factor (in patients following PM) as compared to a risk factor in the general population (during the natural

TABLE 3 Characteristics of patients losing more teeth. Two basic types, 1 and 2, are distinguished according to several clinical and radiological parameters. Mean tooth mobility was the key parameter differentiating both types, followed by abfractions

	n. pts	Mean (ED) TLPD/pt	Mean (ED) mobility	% Mean Mobility	S & B groups	Mean (ED) VCD	Mean (ED) Abfrs/pt 1	Mean (ED) Abfrs/pt 2	Mean (ED) Ging recess	n. pts Ging recess > 1	Mean (ED) Ging Index
Total	51	4.48 (1.16)	0.77 (0.63)			2.61 (2.12)	4.35 (3.72)	8.25 (3.34)	1.64 (0.98)		
Type 1											
Total	26	4 (1.11)	0.30 (0.21)	100% <1	B+	2.50 (2.14)	5.38 (3.43)	8.67 (2.64)	1.21 (0.8)	14 (56%)	1.85 (0.48)
S+/B+	21										
B+	4										
S+	1				S+	0					
Type 2											
Total	25	4.96 (1.32)	1.31 (0.50)	100% ≥1	B+	2.72 (2.12)	3.28 (3.78)	7.63 (4.72)	2.08 (0.97)	19 (82%)	1.97 (0.5)
S+/B+	14										
B+	5										
S+	4				S+	1					
S-/B-	2				B-/S-	0					

Abfrs, abfractions; B, bruxism; Ging recess, gingival recession; n. pts, number of patients; S, heavy smoking; TLPD, tooth loss due to periodontal disease; VCD, vertical and circumferential bone defects.

course of the disease) and these differences may be partially attributed to the efficacy of PM. A recent systematic review concluded that the risk of TLPD was much lower in patients complying regularly with PM as compared to irregular compliers (Lee et al. 2015). Most of the patients analysed in the present research complied regularly with PM."

The impact of bruxism resulted in a threefold increase on the mean VDs and an almost sixfold increased when smoking participated. However, bruxism did not increase the mean FDs unless smoking participated, resulting in a sixfold increase on the mean FDs.

Data supporting the association of occlusal contacts and bruxism with periodontal disease is contradictory (Hanamura et al., 1987; Jin & Cao, 1992; Pihlstrom, Anderson, Aeppli, & Schaffer, 1986; Shefter & McFall, 1984; Yuodelis & Mann, 1965). However, two studies in patients following PM reported a twofold increase in the risk of TLPD associated with bruxism (Martinez-Canut, 2015; McGuire & Nunn, 1996) and an almost fourfold increase in the risk of losing more teeth when bruxism was associated with smoking (Martinez-Canut, 2015). The present research reinforced these findings and characterized these patients according to several clinical and radiological features. Our findings seem to indicate that VCDs and FDs are characteristic features of bruxism and smoking respectively. A particular type of bone defect seen in bruxists could not be categorized as either VCD or FD, since it was a localized extreme loss of supporting bone. This lesion was characteristic of posterior teeth with short and/or fused roots.

It has been shown that smoking increases bone loss (Bergström, Eliasson, & Preber, 1991; Rosa, Lucas, & Lucas, 2008) and is associated with increased prevalence of furcation involvement (Axelsson, Paulander, & Lindhe, 1988; Kerdvongnundit, 2000; Mullally & Linden, 1996). Our results confirm these findings and provide additional information on VCDs. Additional information on the link between bruxism and smoking is presented in supplementary material (Appendix S2).

Abfractions have been attributed to occlusal forces on the cervical area of the teeth and fall within the multi-factorial aetiology of non-carious cervical lesions (Grippio, Simring, & Coleman, 2012). However, these lesions have remained a theoretical process supported by engineering analysis using finite element models (Sarode & Sarode, 2013). Only one study reporting a 14-year follow-up of a patient with bruxism, abfractions, and occlusal wear was found in the literature at the time of writing (Pintado, DeLong, Ko, Sakaguchi, & Douglas, 2000). Consequently, the findings presented in this paper contribute to a better understanding of these lesions.

Abfractions have already been associated with bruxism (McCoy, 1982; Xhonga, 1977), wear facets (Badder, McClure, Scurria, Shugars, & Heymann, 1996; Mayhew, Jessee, & Martin, 1988; Schiller, Marquardt, & Albers, 1985; Telles, Pegoraro, & Pereira, 2000), and occlusal disturbances (Miller, Penaud, Ambrosini, Bisson-Boutelliez, & Briancon, 2003). The study by Miller et al. (2003) found that 10% of patients with abfractions presented bruxism (eccentric bruxism with increased occlusal attrition), while the remaining 90% presented occlusal disturbances (wear facets, lack of canine guidance, and group function).

Subsamples:	n. pts	Mean abfrs	Abfrs category	Mean abfrs	Abfrs category
		(SD)/pt T1	at T1 (n. pts and %)	(SD)/pt T 2	at T2 (n. pts and %)
S- B-/S+ B-	57	1.05 (1.56)	0 = 26, 45.6% 1 = 31, 54.3%	2.65 (2.71)	0 = 10, 17.5% 1 = 44, 77.2% 1-2 = 3, 5.2%
S- B+/S+ B+	117	3.79 (3.06)	0 = 13, 11% 1 = 85, 72.6% 1-2 = 16%	7.52 (3.38)	1 = 5, 4.2% 1-2 = 99, 84.6% 2-3 = 13, 11%

TABLE 4 Mean abfractions per patient and categories at T1 and T2 according to bruxism

Abfrs, abfractions and category (Tooth Wear Index by Smith & Knight, 1984); B, bruxism; n. pts, number of patients; pt, patient; S, heavy smoking; T1, at baseline; T2, at the end of the follow-up.

TABLE 5 Distribution of patients with the corresponding value of the index and the mean index value within each subsample of TLPD according to smoking and bruxism

	n. patients	n. teeth		n. patients with the corresponding value (0- 5) of the index						Mean index value/pt.
		lost	n. patients	0	1	2	3	4	5	
TLPD 0										
Total	74									1.51
S- B-	28	0		3	17	8				1.17
S- B+	37	0			12	22	2	1		1.78
S+ B-	6	0			5	1				1.16
S+ B+	3	0				3				2
TLPD 1-2										
Total	45									2.48
S- B-	10	1-2		1	4	3	2			1.68
S- B+	23	1-2			1	12	6	4		2.56
S+ B-	5	1-2				1	3	1		3
S+ B+	7	1-2				1	4	2		3
TLPD >2										
Total	55									
S- B-	3									2
		3	3			3				
S- B+	10									3.6
		3	3				2	1		
		4	4				3		1	
		5	2					1	1	
		6	1					1		
S+ B-	5									3
		3	2				1	1		
		4	1				1			
		>4	2			1	1			
S+ B+	37									4
		3	11				7	3	1	
		4	11				1	4	6	
		5	6				3	2	1	
		>5	9					5	4	

B, bruxism; n. pts, number of patients; n. teeth lost; number of teeth lost; S, heavy smoking; TLPD, tooth loss due to periodontal disease.

Furthermore, a similar percentage of patients with eccentric bruxism (8.5%) was found in this study. However, the 90% of occlusal disturbances reported by Miller et al. (2003) might to some extent correspond to the 92.4% of our patients with clenching, occlusal disturbances and abfractions. The lack of defined criteria to identify clenching would explain the different results and might indicate the possibility of under diagnosing a relevant and prevalent factor involved in TLPD. Only 10% of bruxists might present the conventional pattern of increased attrition.

The very low prevalence of abfractions found in mobile teeth has been previously reported (Miller et al., 2003).

Cemental tears have been attributed to occlusal trauma (Leknes, Lie, & Selvig, 1996), dental attrition (Lin et al., 2011), poor tissue repair capacity (Ishikawa, Oda, Hayashi, & Arakawa, 1996), and structural weakness of the cementum (Watanabe, Watanabe, Miyauchi, Minoru, & Watanabe, 2012). Several cemental tears progressing to abfractions or developing on existing abfractions were identified in this study.

The association of smoking, alcohol, and other substances with bruxism (Bertazzo-Silveira et al., 2016; Lavigne, Lobbezoo, Rompré, Nielsen, & Montplaisi, 1997; Ohayon, Li, & Guilleminault, 2001) might represent a possible additional pathway implicated in periodontal disease.

This study found a decreased GI associated with smoking and higher TLPD rates, which might be partially explained by the effect of smoking, decreasing bleeding on probing (Al-Bayaty, Baharuddin, Abdulla, Ali, & Al-Bayaty, 2013; Dietrich, Bernimoulin, & Glynn, 2004; Ramseier et al., 2015; Shimazaki et al., 2006) and the inflammatory reactions at the histopathological level (Naderi, Semyari, & Alahinia, 2015). Bleeding on probing and even spontaneous bleeding with other signs of inflammation were not associated with TLPD in patients under PM (Baümer et al., 2011; Faggion, Petersilka, Lange, Gerss, & Fleming, 2007; Tonetti et al., 1998). Irrespective of the level of evidence indicating that bleeding on probing predicts further attachment loss and tooth loss (for review, see Renvert & Persson, 2002), baseline gingival inflammation might represent a distinct condition from that after treatment.

According to our findings, C+ was more prevalent in non-smoking patients, as it has previously been reported (Martinez-Canut, Benlloch, & Izquierdo, 1999) and this might partially explain the complementary finding that fewer C+ deposits were associated with higher TLPD rates. A higher prevalence of subgingival calculus in smokers has also been reported (Bergström, 2005) which could be explained by differences in the patient's sample and the criteria utilized to assess calculus, without a clear distinction between supra and subgingival deposits.

Lastly, the extent to which differences in salivary composition between smokers and non-smokers (Zuabi et al., 1999), and systemic bone mineral (Brennan, Genco, Hovey, Trevisan, & Wactawski-Wende, 2007) influence the type of subgingival calculus might deserve further attention.

The most reliable predictors of an unfavourable outcome have been baseline VCDs and/or FDs associated with increased attrition and/or abfractions, especially in smoking patients. Therefore, the identification of emerging abfractions, VCDs, and FDs at early stages

of disease might help to make a more precise diagnosis and institute the most appropriate prophylactic and therapeutic measurements for a patient at risk of losing more teeth.

The usefulness and accuracy of the long-term outcome predictive index presented here could be validated retrospectively quite easily, using different samples of patients who followed PM over long-term periods.

A predictive index to anticipate the long-term outcome based on the presence of these features is proposed.

Two distinct types of patients at risk of losing more teeth were identified based on differences in the baseline mobility, abfractions and gingival recession.

5 | CONCLUSIONS

This study enabled the characterization of the baseline status of patients following PM according to the final outcome.

This characterization could be useful to identify patients at risk of losing more teeth. These patients were characterized by the presence of smoking and bruxism and several clinical and radiological features which were associated with smoking (FDs, a reduced GI and fewer C+) and bruxism (VCDs and abfractions).

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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SUPPORTING INFORMATION

Additional Supporting Information may be found online in the supporting information tab for this article.

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