Predicting probing depth reduction after periodontal non-surgical treatment in smokers according to the nicotine dependence and the number of cigarette consumed

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ABSTRACT

Introduction: Smoking is considered as a risk factor for the poor outcomes after periodontitis non-surgical treatment (PNST). The aim of this short communication is to predict probing depth reduction after periodontal non-surgical treatment in smokers according to the nicotine dependence (FTND) and the number of cigarette consumed (NCC).

Methods: This work is a post-hoc study of a prospective controlled study on the effect of oral hygiene instructions and PNST on periodontal outcomes. This short communication focused only on the current conventional smokers (N = 34), based on specific smoking indicators, and on probing depth (PD) parameter that were recorded at baseline (time 0), after oral hygiene instruction (time 1) and 3 months after PNST (time 2).

Results: The 34 smokers had a mean age 46.5 ± 11.5 years. The NCC- and FTND-based predictions allowed to show in a specific nomogram the PD values 3 months after PNST for each NCC and FTND category.

Conclusion: Two nomograms are proposed for prognostic purposes and allow patients to understand the impact of smoking on periodontitis according to the number of cigarette consumed and the level of nicotine dependence. These nomograms might be also used for supporting smoking cessation.

Clinical significance: In smoker patients with periodontitis, there is a need to predict, for both patient and clinicians, the impact of the number of cigarettes consumed and the level of nicotine dependence on probing depth after oral hygiene instructions and debridement. Two nomograms are proposed for prognostic purposes.

1. Introduction

Periodontitis is a chronic and inflammatory disease characterized by the presence of gram negative bacteria that leading to the progressive destruction of the tooth supporting tissues [1]. Moreover, the evolution of the disease, as well as the outcomes after periodontitis non-surgical treatment, are influenced by several risk factors including smoking [2, 3, 4, 5, 6]. As we demonstrated in a previous study [7], on 91 patients (32 non-smokers, 25 former smokers and 34 current smokers), the improvements of periodontal parameters in current smokers were only visible after PNST and were in general significantly lower compared to the two other groups.

For smokers with periodontitis, however, the understanding of what could be the periodontal tooth prognosis after PNST remains unclear.

This short communication proposes nomograms for both clinicians and patients to better understand probing depth reduction after PNST according to the degree of smoking dependence and the number of cigarettes consumed.

2. Material and methods

2.1. Population

34 current conventional smokers of the 91 patients of the original study admitted and treated for periodontitis at the Department of Periodontology, Buccal Surgery and Implantology of the University Hospital, Liege, Belgium. The original study design, data collection, sample size calculation, and results have been published elsewhere [7].

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2.2. Smoking assessment

The smoking status was recorded in two ways: (1) the number of cigarettes consumed per day (NCC), and (2) the Fagerström test for nicotine dependence (FTND). NCC was categorized into classes of 3 cigarettes (1–3, 4–6, 7–9 and so on). The FTND consists of six questions described in 1991 by Heatherton and co-authors [8, 9]. Thus, by summing all six responses, the FTND score ranges between 0 and 10, the higher the score the stronger the smoking dependence.

2.3. Probing depth

Among clinical periodontal parameters collected, the present article focused on maximal probing depth (PD). A graduated manual periodontal probe1 was used to take measurements at the six sites of each tooth (mesio-buccal, mid-buccal, disto-buccal, mesio-lingual, mid-lingual and disto-lingual). PD was recorded at baseline (time T0, diagnosis of periodontitis), after OHI (time T2) and 3 months after PNST (time T2).

2.4. Statistics

PD longitudinal data were analysed by linear mixed effects models and described by two separate equations, one including time, NCC and an interaction term (time × NCC), and another including time, FTND and an interaction term (time × FTND). Then, for each class of NCC and each score of FTND, a prediction of PD reduction from baseline was calculated and displayed on a graph as a nomogram. Results were considered significant at the 5% critical level (P < 0.05). All calculations and graphs were performed with SAS version 9.4 for Windows and R version 3.5.

2.5. Ethical committee and informed consent

The present work is a post hoc study that has been approved by the ethical committee of the University Hospital of Liege, Belgium (B707201421977) and was registered on clinicaltrial.gov (NCT04061460). The goals of the study were carefully explained, and all patients signed an informed consent form.

3. Results

The 34 smokers had a mean age 46.5 ± 11.5 years. Nineteen (55.9%) presented with severe (stage IV) periodontitis, 97.1% for a rapid rate of progression (grade C) and 52% for a generalized form of periodontitis. The mean NCC was 16.7 ± 5.5 (range: 5–30) and the mean FTND was 5.2 ± 1.8 (range: 1–9). At baseline, PD max averaged 8.0 ± 1.2 mm. It stayed at 8.0 ± 1.2 mm after OHI, then dropped to 6.3 ± 1.9 mm three months after PNST. No correlation was found at baseline between PD values and NCC (r = -0.15, P = 0.40) and between PD values and FTND (r = 0.014, P = 0.94).

3.1. NCC-based predictions

When fitting a linear mixed model to PD time-related data, a significant decrease was found after PNST (coefficient ±SE: -4.0 ± 0.78, P < 0.0001), no NCC effect was observed (0.032 ± 0.042, P = 0.44), however a significant time interaction was noted between the results after PNST and NCC (0.14 ± 0.050, P = 0.0044). The equation allowed predicting PD values 3 months after PNST for each NCC category. For example, for NCC (1–3) the predicted value was 4.9 mm and for NCC (28–30) it was 7.8 mm. The predictions for each NCC category are displayed in the nomogram of Figure 1.

3.2. FTND-based predictions

When fitting a linear mixed model to PD time-related data, a significant decrease was found after PNST (coefficient ±SE: -3.7 ± 0.80, P < 0.0001), no FTND effect was observed (0.009 ± 0.13, P = 0.94), however a significant time interaction was noted between the results after PNST and FTND (0.39 ± 0.15, P = 0.016). The equation allowed predicting PD values 3 months after PNST for each FTND score. For example, for FTND = 1 the predicted value was 4.7 mm and for FTND = 10 it was 8.2 mm. The predictions for each FTND score are displayed in the nomogram of Figure 2.

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4. Discussion

Although the impact of smoking on the outcomes after PNST is well-known among dental practitioners, it is less understandable for patients. Therefore, there is a need to have a comprehensive tool for patient aiming to realize, at the time of the periodontitis diagnosis, what can be the impact of smoking after their PNST. This short communication proposes visual tools for patients and dental practitioners that can contribute to give a baseline prognosis of the probing depth reduction 3 months after PNST, according to NCC and FTND. The nomograms clearly show that the expected reduction can be substantially different according to smoking intensity and nicotine dependence.

The negative effect of smoking on periodontal outcomes after PNST have been widely described in the literature [7, 10, 11, 12, 13, 14, 15, 16, 17]. In a systematic review on the effect of smoking on periodontal non-surgical therapy, the authors concluded that smokers will experience less reduction in probing depth than nonsmokers [18], with a mean difference in probing depth reduction of 0.133 mm (95%CI [0.038–0.227], P = 0.006). In another clinical study, a multilevel modelling analysis on the effects of smoking on healing response to PNST, also demonstrated that smokers had less favourable probing depth reduction at deep sites [17]. Therefore, the fact that smoking impairs periodontal recovery is evidenced-based. However, in the available literature, no distinction between the number of cigarettes consumed per day or the level of nicotine dependence on the probing depth reduction after PNST was made. The NCC commonly used is a quantitative marker of smoking, related to the pack-year indicator corresponding to the frequency of exposition of specific molecules as hydrocarbons and nitrosamines which can induce over time carcinogenesis [19]. By contrast, the FTND represents a qualitative marker of smoking, with a questionnaire linked to the smoking behavior and the nicotine dependence [20, 21]. Therefore, the instant of the day or the general condition can modulate the way of patient smokes, indeed the impact the level of dependence [8], perceptible thanks to the FTND [22, 23]. Based on this difference between quantitative and qualitative markers of smoking, a recent cross-sectional study on the severity of periodontitis [24] was done, and the authors concluded that the combination of FTND and NCC in discerning disease severity was slightly superior to each indicator separately (ROC curve analysis: AUC = 0.746, P = 0.027). The nomograms show that even in smokers with periodontitis the improvement of probing depth can substantially differ according to the actual number of cigarettes consumed or the level of nicotine dependence. Assuming a baseline PD max level of 8 mm, the reduction 3 months after PNST according to NCC ranged from 2.5% (NCC 28–30) to 38.8% (NCC 1–3). For FTND, no reduction was found for FTND = 10 (~2.5%) and the reduction reached 41.3% for FTND = 1.

5. Conclusion

The expected probing depth reduction 3 months after PNST can be substantially different according to smoking intensity and nicotine dependence. Therefore, dental practitioners and their patients can use the two nomograms based on qualitative and quantitative markers of smoking to predict at baseline the probing depth reduction 3 months after PNST. These specific tools can also be used for supporting smoking cessation.

Declarations

Author contribution statement

Leila Salhi: Conceived and designed the experiments; Performed the experiments; Analyzed and interpreted the data; Contributed reagents, materials, analysis tools or data; Wrote the paper.
Adelin Albert: Conceived and designed the experiments; Analyzed and interpreted the data; Contributed reagents, materials, analysis tools or data; Wrote the paper.
Laurence Seidel: Analyzed and interpreted the data.
France Lambert: Contributed reagents.

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Data availability statement

Data included in article/supp. material/referenced in article.
Declaration of interest's statement

The authors declare no conflict of interest.

Additional information

No additional information is available for this paper.

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