http://dx.doi.org/10.1590/1981-86372022001220200104

| ORIGINAL

Full-mouth ultrasonic debridement in the treatment of patients with diabetes and severe chronic periodontitis: preliminary study

Debridamento ultrassônico de boca toda no tratamento da periodontite crônica severa em pacientes diabéticos: estudo preliminar

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ABSTRACT

Objective: The purpose of this study was to clinically evaluate the effect of one-stage full-mouth ultrasonic debridement in the treatment of diabetic subjects with severe chronic periodontitis. **Methods**: Sixteen patients with uncontrolled diabetes (glycated hemoglobin ≥ 7%) and severe chronic periodontitis (at least 8 teeth with a probing depth and bleeding on probing were selected and randomly assigned to 2 treatment groups: control group (n=7) - quadrant-wise scaling and root planning; test group (n=9) - full-mouth ultrasonic debridement in one session of 45 minutes. The following outcomes were assessed: plaque index, gingival index, bleeding on probing, probing depth, clinical attachment level and gingival margin position. All parameters were assessed at baseline, 1 and 3 months after treatment. Probing depth, clinical attachment level and gingival margin position were analyzed using ANOVA and Tukey test, and the other clinical parameters analysed with the Friedman and Mann-Whitney tests. **Results**: An improvement in probing depth, clinical attachment level was observed after treatment in both groups, but without statistically significant differences between them. After 3 months, in initially deep pockets, the PD decreased 1.6 mm and 1.7 mm in test and control group, respectively. Clinical attachment level gain ≥ 2 mm was statistically significant at sites in control (10.1%) and test (13,4%) groups in 3 months. **Conclusion**:

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How to cite this article

Meira ALT, Nobre C, Nascimento MC, Napimoga M, Casarin R, Bittencourt S, Ribeiro EDP. Full-mouth ultrasonic debridement in the treatment of patients with diabetes and severe chronic periodontitis: preliminary study. RGO, Rev Gaúch Odontol. 2022;70:e20220012. http://dx.doi.org/10.1590/1981-86372022001220200104

One-stage full-mouth ultrasonic debridement promoted similar clinical outcomes to those obtained with the scaling and root planning in the treatment of diabetic subjects with severe chronic periodontitis.

Indexing terms: Diabetes Mellitus. Chronic periodontitis. Dental scaling.

RESUMO

***Objetivo**: Avaliar clinicamente o efeito do debridamento ultrassônico de boca toda no tratamento da periodontite crônica severa em pacientes diabéticos. **Métodos**: Dezesseis pacientes diabéticos descompensados (Hemoglobina Glicada ≥ 7%) com periodontite crônica severa (mínimo de 8 dentes com profundidade de sondagem ≥ 5 mm e sangramento à sondagem) separados aleatoriamente em 2 grupos: Controle (7): raspagem e alisamento radicular por quadrante e Teste (9): debridamento ultrassônico sessão única de 45 minutos. Os parâmetros clínicos avaliados foram: Índice de placa, Índice gengival, Sangramento à Sondagem, Profundidade de Sondagem, Nível de inserção clínico e Posição da Margem Gengival. As variáveis foram avaliadas no início, 1 e 3 meses após o tratamento. Para análise das variáveis quantitativas (Profundidade de sondagem, Nível de inserção e Posição da margem gengival de bolsas moderadas) foram realizados ANOVA e teste de Tukey e para as demais foram utilizados os testes de Friedman e Mann-Whitney. **Resultados**: Observou-se melhora na profundidade de sondagem e nível de inserção clínica após o tratamento em ambos os grupos, sem diferenças estatisticamente significativas entre eles. Após 3 meses, nas bolsas inicialmente profundas, a profundidade de sondagem reduziu 1,6 e 1,7 mm nos grupos teste e controle, respectivamente. Houve um ganho estatisticamente significante no nível de inserção ≥ 2 mm nos grupos controle (10,1%) e teste (13,4%), aos 3 meses. **Conclusão**: O debridamento ultrassônico de boca toda promoveu resultados clínicos semelhantes aos obtidos com a raspagem e alisamento radicular no tratamento da periodontite crônica severa em indivíduos diabéticos.*

***Termos de indexação**: Diabetes Mellitus. Periodontite crônica. Raspagem dentária.*

INTRODUCTION

Periodontal disease (PD) is a chronic immunoinflammatory disorder caused by periodontopathogenic microorganisms that results in the destruction of periodontal support tissues and, eventually, tooth loss [1]. The role of biofilm as the primary etiological factor of periodontal disease has been clearly established in the literature [2]. However, biofilm alone does not explain the level of destruction often found in the periodontium [3]. Evidence shows that disease severity and progression may be associated with environmental and systemic factors, such as smoking and diabetes [4].

Diabetes as a metabolic disease has been associated with periodontal changes and described as a risk factor for increases in its prevalence and severity [5,6]. The possible explanations for this association point to the immunoinflammatory changes found in patients with uncontrolled diabetes, such as lower leukocyte chemotaxis, increase in the number of inflammatory mediators, reduction of matrix synthesis by fibroblast and changes in the functioning of the components of the extracellular matrix [7].

The biochemical bases that explain the association of hyperglycemia with greater severity of periodontal disease is a result of the increasing accumulation of advanced glycation end products (AGEs) in plasma and tissues. AGEs bind to advanced glycation end products (RAGEs) found in endothelial cells, monocytes and macrophages, nervous cells and muscle cells. The AGE-RAGE interaction in monocytes and macrophages increases cell oxidative stress, which results in a greater production and secretion of inflammatory cytokines, such as tumor necrosis factor alpha (TNF-α) and interleukin-1beta (IL-1β) [8]. These cytokines are directly associated with osteoclast differentiation and activity, as well as with the production of matrix metalloproteinase (MMP), responsible for collagen degradation. All these mediators also effectively participate in the pathogenesis of periodontal disease [9].

The presence of periodontal disease may also lead to an increase in the systemic level of inflammatory cytokines, such as TNF-α, interleukin-6 (IL-6), C-reactive protein (CRP) and fibrinogen. Patients with diabetes and high blood concentrations of these inflammatory markers may find it difficult to control glucose because these markers are associated with insulin resistance [10]. Therefore, similarly to what is found with other bacterial infections, diabetes and periodontal infection are associated bidirectionally, as the presence of one affects the other and, consequently, the careful control of one may help the treatment of the other [9].

Periodontal inflammation is commonly treated with scaling and root planning (SRP) [11]. However, in some cases this intervention seems not enough to restore or preserve periodontal health, which may be due to microorganism resistance or recolonization and even to the presence of risk factors associated with host defense [12]. The concern with reinfection of the periodontal pockets has led to the development of the concept of one-stage full-mouth disinfection [13].

In this context, Wennström et al. [14] described a new approach to periodontal treatment, called full-mouth ultrasonic debridement. This treatment is based on the concept of Smart et al. (1990) [15], who defined periodontal debridement as a more conservative instrumentation performed under light pressure during a limited period of time using an ultrasound unit. The main objective of ultrasonic debridement was to disrupt biofilm and remove bacterial endotoxins adhered to root cement to promote a biocompatible surface and, therefore, facilitate fibroblast attachment and tissue healing [16,17]. This approach was only possible because endotoxins were found to attach to cement weakly and superficially. Therefore, systematic root planing to remove cement does not seem to be justified or recommended [18].

Several studies confirmed the feasibility of full-mouth ultrasonic debridement (FMUD) to treat severe chronic periodontitis, as results were similar to those of SRP [18-20]. It seams relevant to investigate whether this therapeutic approach has advantages over conventional quadrant SRP in patients with diabetes, who are more susceptible to infections. Some of these advantages could be the reduction of repeated trauma and edema, which are responsible for the maintenance of high levels of proinflammatory cytokines, as well as the substantial reduction of bacteria that cause infection in a shorter period of time or with less visits to the dentist [8].

Thus, the aim of the present study was to evaluate the clinical effect of one-stage full-mouth ultrasonic debridement in the treatment of diabetic patients with severe chronic periodontitis.

METHODS

The present study was designed as a parallel, blinded, randomized and controlled clinical trial to compare periodontal results of patients with uncontrolled diabetes (HbA1c ≥ 7%) after SRP (control group) and after ultrasonic debridement (test group). This study was approved by the Research Ethics Committee of Bahiana School of Medicine and Public Health (EBMSP), Salvador, BA, Brazil, under protocol number 028/10. All patients were individually informed about the nature and objective of the study, as well as its risks and benefits, and signed an informed consent term to participate in the study.

Study population

Three hundred and fifty-seven patients referred from primary healthcare units or specialized dental care centers, or who spontaneously sought dental assistance for periodontal treatment, were examined at the Screening and Urgency Unit of EBMSP from July 2010 to January 2014. Of these, 16 patients were invited to participate in this trial meeting the following inclusion criteria: (1) diagnosis of severe chronic periodontitis by the presence of periodontal pockets with clinical attachment loss, bleeding on probing (BOP) and radiographic evidence of bone loss; (2) at least eight teeth with probing depth (PD) ≥ 5 mm and BOP (qualifying teeth): at least two of the eight qualifying teeth had PD ≥ 7 mm, and in two additional teeth the pockets had PD ≥ 6 mm; (3) uncontrolled diabetes (HbA1c ≥ 7%); and (4) ≥ 15 teeth in both jaws, wisdom teeth excluded.

Exclusion criteria were: periapical or pulp alterations on qualifying teeth, consumption of drugs known to affect periodontal status (antibiotic, anti-inflammatory, anticonvulsant, immunosuppressant, or calcium channel blocker) within the past 3 months, periodontal treatment, including subgingival scaling and root planning in the preceding 6 months, systemic and immunological disorders other than diabetes that require prophylactic antibiotic coverage or that could influence response to treatment, smoking, pregnancy and cardiac pace-makers users.

Treatment

Subjects initially received detailed information about the causes and consequences of periodontal disease, as well as instructions in proper self-performed plaque control measures, including interdental cleaning with dental floss and interdental toothbrushes. Standardized toothbrushes provided by Bitufo® (Itupeva, São Paulo, Brazil) were distributed. In the first visits, plaque retention factors (caries, excess of restorations, and supragingival calculus) were removed and condemned teeth extracted. The baseline measurements were done 30 days after this initial phase.

After this initial phase participants were randomly (by coin toss) assigned to one of the two groups by the researcher responsible for the treatments (MNC). The control group (7 patients) underwent conventional quadrant-wise SRP using Gracey curettes (Hu-Friedy®, Chicago, IL, USA) at one-week intervals between quadrants. The test group (9 patients) underwent one session of full-mouth ultrasonic debridement (FMUD) with a time limit of 45 min, using the ultrasonic scaler (Cavitron Select, Dentsply, New York, NY, USA) with specific tips (UI25SD10, Hu-Friedy, Chicago, IL, USA). Both treatments were conducted under local anesthesia.

After the active phase of treatment, patients were included in a supportive therapy program receiving weekly supragingival plaque control instructions during the first month. After the first 4 weeks, monthly reinforcement of oral hygiene instructions and prophylaxis with prophylactic paste and rubber cups were performed in all the patients. This maintenance program also included updates in the medical and dental history, intra and extra-oral exams and periodontal evaluations. At the third month recall visit, sites that exhibited PD ≥ 5 mm and bled on probing were reinstrumented with curettes and no time restriction.

Only one professional (MNC) was responsible for administering the treatment throughout the study. This professional was different from the calibrated examiner (ALTM) responsible for performing the clinical measurements. The examiner remained blinded throughout the study.

Clinical and glycemic monitoring

All clinical parameters were evaluated at baseline and at one and three months after periodontal treatment. Clinical parameters were evaluated at six sites in all teeth, except third molars: mesiobuccal, buccal, distobuccal, mesiolingual, lingual and distolingual. Supragingival biofilm was evaluated according to the presence of visible plaque index (VPI) and the gingival bleeding index (GBI), dichotomously 21 . BOP was also recorded dichotomously [22]. The other clinical parameters, such as PD, clinical attachment level (CAL) and gingival margin position (GMP), were measured using a manual standardized North Carolina periodontal probe (PCPUNC 15R Hu-Friedy, Chicago, IL, USA) by the same calibrated investigator (ALTM).

This investigator was calibrated for intraexaminer repeatability prior to the start of the trial. Three patients with chronic periodontitis were enrolled and duplicate measurements for PD and CAL were collected with an interval of 7 days. The intraclass correlation coefficient was 0.83 for PD and 0.87 for CAL.

Glycemic levels of all patients were controlled using the measurement of glycated hemoglobin (HbA1c) and fasting plasma glucose (FPG) at baseline and at one and three months after the treatment. HbA1c concentration percentage (%) was calculated using an immunoturbidimetric test, A1c fraction; FPG (mg/dL) was measured by glucose oxidase method. To standardize procedures for both groups, the tests were all performed at the same laboratory in the Teaching Outpatient Healthcare Department of Bahiana.

Statistical analysis

Results were analyzed using descriptive statistics, tables and graphs with absolute and relative frequencies, as well as means and standard deviations. Quantitative variables (PD, CAL, moderate-pocket GMP) were compared using

analysis of variance (ANOVA) and the Tukey test. Other clinical parameters, which were not normally distributed, were analyzed as nonparametric data using the Friedman and the Mann-Whitney tests. Qualitative variables were compared using a chi-square test. The level of significance was set at 5% for all tests.

RESULTS

Sixteen patients that completed the study and were followed up for three months after periodontal treatment were evaluated: 7 in the control group (SRP) and 9 in the test group (FMUD). After the completion of the study, considering the standard deviation (SD) of each group of the present clinical trial, the power value was 0.86 to detect a 1 mm difference in CAL between the two groups. Figure 1 shows the study flowchart.

Of the 16 participants, seven (45.5%) were women. Patient age ranged from 33 to 66 years, and mean age was 48.7 years. Data analysis at baseline indicated that groups were balanced for age, gender and type of diabetes (table 1).

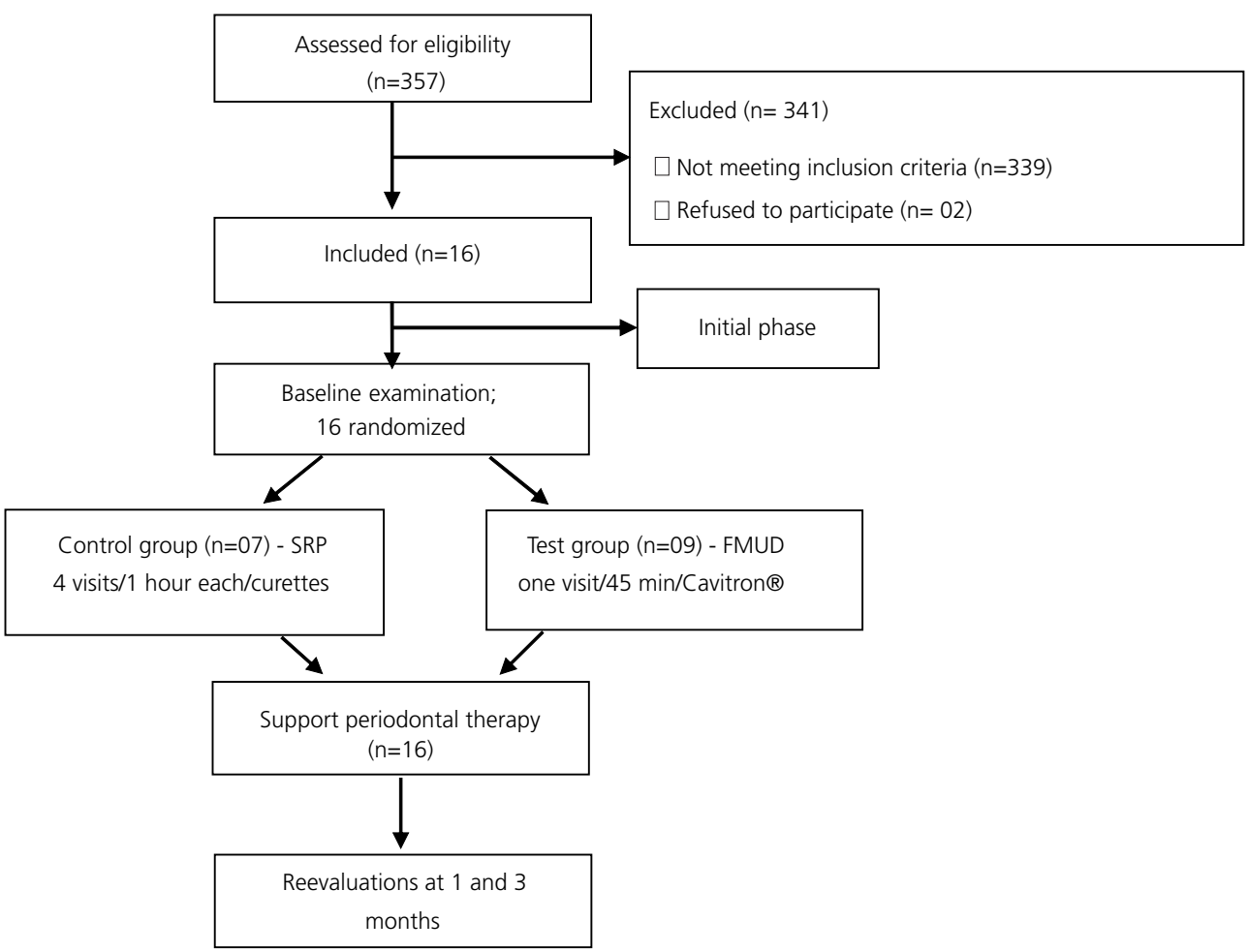

Figure 1. Study flowchart (Salvador, 2014).

Table 1. Demographic characteristics: age, sex and type of diabetes (Salvador, 2014).

	Age	Sex		Type of diabetes (%)	
Control group (n=7)	39 – 51 (mean: 45 years)	F 3	M 4	Type 1 2	Type 2 5
Study group (n=9)	33 – 63 (mean: 51 years)	F 4	M 5	Type 1 2	Type 2 7

Clinical results

Data analysis at baseline indicated that groups were balanced for clinical parameters. Regarding VPI, GBI and BOP, no significant difference between groups was observed at any of the examination intervals. Test group showed a statistically lower value of VPI only at 1 month when compared with baseline. The GBI was reduced in both groups at 3 months ($p<0.05$) (table 2).

Table 2. Mean and standard deviation (mm) values of VPI, GBI and BOP at baseline and at one and three months after periodontal treatment (Salvador, 2014).

		Baseline	1 month	3 months
VPI	Control group	30.8±14.5 Aa	20.5±10.4 Aa	24.2±11.8 Aa
	Test group	30.2±17.7 Aa	17.0±6.3 Ab	19.2±9.6 Aab
GBI	Control group	13.9±17.0 Aa	7.9±6.7 Ab	5.2±4.8 Ac
	Test group	8.9±8.7 Aa	5.5±4.1 Aab	3.9±3.2 Ab
BOP	Control group	52.3±19.0 Aa	45.6±16.0 Aa	33.3±16.6 Aa
	Test group	50.0±14.9 Aa	35.4±11.4 Aa	35.0±10.8 Aa

Note: Different letters (upper case: inter-group; lower case: intra-group) indicate statistically significant differences (Friedman and Mann-Whitney tests; $p<0.05$).

The results for PD, CAL and GMP refer strictly to the qualifying sites. They were classified according to PD values before treatment in moderate pockets (MP), the ones with PD of 5 and 6 mm; and deep pockets, when PD was ≥ 7 mm. In moderate pockets, both groups showed, at 1 and 3 month, a reduction in PD and a gain in CAL, without difference between groups (table 3).

Table 3. Mean and standard deviation (mm) values of PD, CAL and GMP in moderate pockets (MP) at baseline and after periodontal treatment (Salvador, 2014).

		Baseline	1 month	3 months
MP PD	Control group	5.4±0.1 Aa	4.6±0.5 Ab	4.6±0.4 Ab
	Test group	5.4±0.2 Aa	4.2±0.4 Ab	4.2±0.5 Ab
MP CAL	Control group	5.7±0.3 Aa	4.8±0.5 Ab	4.8±0.5 Ab
	Test group	5.8±0.5 Aa	4.8±0.6 Ab	4.7±0.7 Ab
MP GMP	Control group	0.3±0.3 Aa	0.2±0.2 Aa	0.2±0.2 Aa
	Test group	0.4±0.4 Aa	0.6±0.5 Aa	0.5±0.5 Aa

Note: Different letters (upper case: inter-group; lower case: intra-group) indicate statistically significant differences (ANOVA and Tukey test; $p<0.05$).

The same result pattern was found in the evaluation of PD and CAL in deep pockets (≥ 7 mm). These parameters improved in both the test and control groups after treatment, but there were no significant differences between groups (table 4).

Table 4. Mean and standard deviation (mm) values of PD, CAL and GMP in deep pockets (DP) at baseline and after periodontal treatment (Salvador, 2014).

		Baseline	1 month	3 months
DP PD	Control group	7.6±0.5 Aa	5.6±1.4 Ab	6.0±1.2 Ab
	Test group	7.6±0.6 Aa	5.8±0.8 Ab	5.9±1.3 Ab
DP CAL	Control group	8.1±1.2 Aa	5.8±1.5 Ab	6.4±1.5 Ab
	Test group	8.2±1.7 Aa	6.5±1.8 Ab	6.6±1.3 Ab
DP GMP	Control group	0.5±0.8 Aa	0.2±0.3 Aa	0.4±0.4 Aa
	Test group	0.6±1.3 Aa	0.7±1.3 Aa	0.7±1.2 Aa

Note: Different letters (upper case: inter-group; lower case: intra-group) indicate statistically significant differences (Friedman and Mann-Whitney tests; p<0.05).

The percentage of sites that needed re-treatment at 3 months, were similar between test (18.7%) and control (19.8%) groups (p=0.702). The analysis of the percentage of sites that had an increase of CAL ≥ 2 mm revealed that the test group had better results at one and three months of follow-up than the control group, and this difference was statistically significant (p=0.0001 at 1 month and p=0.028 at 3 month) (figure 2).

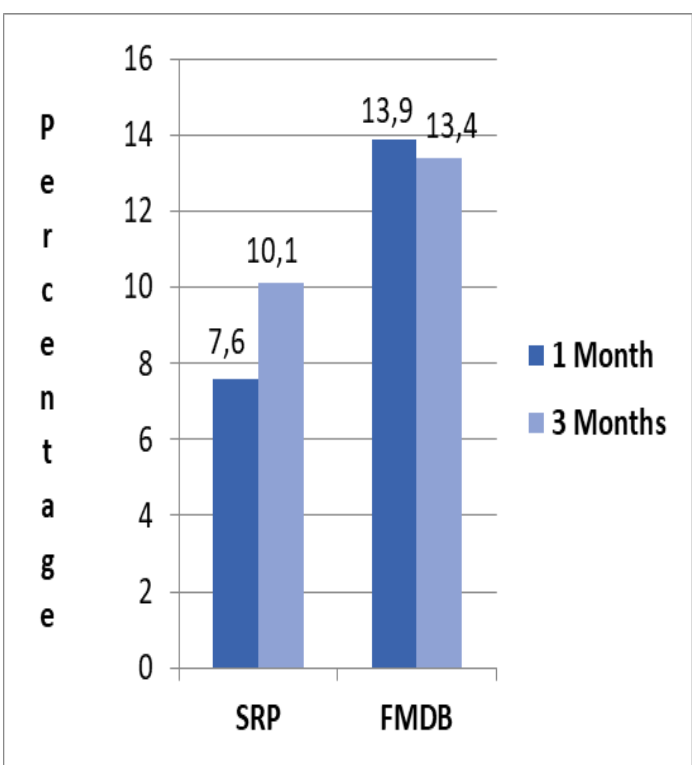

Figure 2. Percentage of sites that had an increase in CAL ≥ 2 mm at 1 and 3 months after periodontal treatment Chi-square test (p<0.05); SRP and FMDB groups.

The analysis of glycated hemoglobin and fasting plasma glucose results revealed that there were no statistically significant differences between groups or between time points (table 5). During the study, there were no reports of adverse effects, such as fever, discomfort or malaise during or after treatment, as well as no changes in diet, in use of glucose-lowering agents or in insulin dose.

Table 5. Metabolic data (mean ± standard deviation) in both groups and at different evaluation time points (Salvador, 2014).

		Baseline	1 month	3 months
HbA1c	Control group	9.48±1.53 Aa	8.63±1.68 Aa	8.54±1.51 Aa
	Study group	10.21±2.17 Aa	9.01±4.45 Aa	10.17±3.32 Aa
FG	Control group	173.4±47.3 Aa	214.7±70.2 Aa	167.6±63.4 Aa
	Study group	194.3±84.1 Aa	150.7±21.1 Aa	231.0±60.0 Aa

Different letters indicate statistically significant differences between time points within group (repeated measures ANOVA and Tukey test, $p<0.05$). There were no significant differences in any parameters between groups.

DISCUSSION

Several types of periodontal treatments have been tested in diabetic patients with chronic periodontitis. In patients with no systemic disease who have chronic periodontitis, full-mouth ultrasonic debridement has been shown to be a viable treatment alternative, with results that are similar to those of SRP in improving periodontal clinical parameters [20]. In this context, the present study evaluated the clinical effects of full-mouth ultrasonic debridement in the treatment of patients with diabetes and severe chronic periodontitis. In this novel approach to periodontal treatment the entire dentition is instrumented during a single session for a restricted period of time, which may be beneficial and advantageous for patients with diabetes.

Both type 1 and type 2 diabetes have the same biochemical mechanism in their association with periodontal disease, and, therefore, the response to periodontal treatment may be similar in patients with either type of diabetes [23]. In this study, participants had uncontrolled type 1 (4 patients) or type 2 (12 patients) diabetes (HbA1c ≥ 7%).

The present study demonstrated that in diabetic patients with severe chronic periodontitis, the FMUD resulted in clinical improvements similar to those achieved with the traditional approach of quadrant-wise SRP. The evaluation of CAL, which was the primary variable in this study, did not reveal any statistically significant difference between groups. This finding is valid for patients with adequate plaque control, as reported in the study. Since baseline measurements were done after the initial preparation, subjects had low baseline VPI (30.8% and 30.2% in control and test groups, respectively) and GBI (13.9% and 8.9% in control and test groups, respectively). At three months, VPI was 24.2% in the control group and 19.2% in the test group. Therefore, these study findings are similar to the ones reported by Santos et al. [24], who observed that VPI at three months was 33.9 % (FMUD) and 26.2 % (SRP).

The evaluation of PD of moderate and deep pockets also revealed that the results of SRP and FMUD were similar, favorable and significant in reducing PD. These findings are also in agreement with those reported by Santos et al. [24], who compared FMUD and SRP in the treatment of type-2 diabetic patients chronic periodontitis and found that both techniques were equally efficient after three months, with deep pockets presenting the best results, followed by moderate and shallow pockets.

In the present study, the clinical attachment gain in deep pockets at 3 months was 1.7 + 0.3 mm and 1.6 + 0.4 mm in the control group and test groups, respectively. These values are similar to those reported by Da Cruz et al. (2008) [5], that observed a CAL gain of 1.21 + 0.24 mm in the group of patients with diabetes that underwent SRP in a single 2-hour visit. Cirano et al. [25] found similar CAL gains (2.1 ± 0.1mm) after six months of ultrasonic debridement in patients with uncontrolled diabetes. This result may be assigned to the fact that, especially in deeper sites, the use of ultrasonic

instrument can have advantages because of the thinner and more delicate subgingival tips, which ensure a better access to the bottom of deep and narrow pockets [26].

The evaluating of sites that showed a gain of CAL ≥ 2 mm revealed that the results of the test group were better than those found for the control group. The difference between groups was statistically significant in the first (13.8% x 7.6%, p=0,001) and third months (10.1% x 13.4%, p=0.028). In the study conducted by Del Peloso Ribeiro et al. [20], there was a greater percentage of sites with attachment gains ≥ 2 mm at the 3 month evaluating period, 48.08% and 44.44% in the control group (SRP) and test groups (FMUD), respectively. This difference may be explained by the fact that their study included patients with severe chronic periodontitis, but without any systemic diseases, and it is known that patients with diabetes, particularly those without adequate glucose control as the ones included in the present study have healing difficulties that justify their poor response to periodontal treatment [25].

The results for the need of re-treatment should also be seen in light of the effects of diabetes in periodontal treatment. This is a clinical relevant parameter because is associated with clinical stability in the long run [27]. In the present study, 19.8% of the sites in the control group and 18.7% in the test group needed re-treatment at 3 months. These values are higher than the ones reported by Del Peloso Ribeiro et al. [20], who found that only 9.61% of the sites in the SRP group and 10.18% in the FMUD group needed retreatment. Again, Del Peloso Ribeiro et al. [20] included only systemic healthy subjects and for these reason, the studies should be compared cautiously. Moreover, the discussion of this parameter is complicated by the fact that it has not yet been commonly evaluated in patients with diabetes.

Some authors highlight the importance of using antimicrobial agents as an adjunct therapy to non-surgical periodontal treatment in individuals with diabetes [6,28,29]. However, as there is no consensus about this recommendation, and as there are no reliable results in the literature to justify its use as a protocol in the treatment of diabetic patients, the systemic or local use of antimicrobials was not adopted in the present study. Other authors, concerned about bacterial resistance, also have chosen not to include antimicrobial agents in their study protocol and found satisfactory clinical results [5,24]. Some of the studies that demonstrated the effectiveness of these agents did not include a comparison with periodontal treatment only, which makes it difficult to interpret their results impartially [28,29].

Most clinical trials that compared FMUD and SRP found similar clinical results after both treatments in healthy patients with severe chronic periodontitis [20,30]. Lang et al. [31] conducted a systematic review of studies using different treatments, which included full-mouth debridement with and without the use of antiseptics and conventional treatment (SRP), and found that, regardless of the treatment used, clinical results were positive, particularly when dental biofilm control was effective. This finding may also be interpreted favorably for the use of FMUD, because the reduction of instrumentation time and of dental structure loss seems to be an important clinical gain for the restoration of periodontal health and long-term maintenance in patients with uncontrolled diabetes. These patients need special attention during all visits, because their glucose levels may change while they are receiving dental care.

The present study did not find any improvement in glycemic status after periodontal treatment, which is in agreement with findings reported by Cirano et al. [25] that evaluated the effects of FMUD in patients with uncontrolled diabetes type 2. The effects of periodontal treatment on HbA1c and FPG levels of diabetic patients are controversial. The most recent review of the literature included 13 studies conducted from 2013 to 2015 [32]. Seven of those studies reported improvements in glucose levels after periodontal treatment. However, the authors suggested caution in the interpretation of results because of the study methods, which included differences in sample sizes, periodontal disease extension and severity, use of oral or systemic antimicrobials, follow-up time and type of periodontal treatment, among other factors [31-34].

The results of the present study are true for the sample analyzed. Because of the strict inclusion and exclusion criteria used to reduce the occurrence of confounding factors to a minimum, only 16 individuals were included. Also, studies with longer periods of follow-up are necessary to confirm the efficacy of FMUD and to analyze whether positive results are maintained in the long term.

CONCLUSION

Full-mouth ultrasonic debridement promoted similar significant clinical improvements when compared to scaling and root planning and thus may be considered as a viable approach to treat patients with uncontrolled diabetes and severe chronic periodontitis.

Collaborators

ALT Meira, researcher responsible for clinical evaluations, project follow-ups, data analysis and writing. C Nobre, researcher responsible for screening and initial preparation of patients. MC nascimento, researcher responsible for performing periodontal treatments in both groups. M NAPIMOGA, researcher responsible for immunological analysis of plaque and gingival fluid collections. R Casarin, researcher responsible for statistical analysis. S Bittencourt and E Del Peloso Ribeiro, researchers responsible for guidance, design and critical analysis of the research project.

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Received on: 8/7/2020
Final version resubmitted on: 5/10/2020
Approved on: 1/12/2020

Assistant editor: Fabiana Mantovani Gomes França