

# Comparative Clinical Evaluation between Conventional Periodontal Treatment and Full Mouth Disinfection

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## Abstract

Periodontal disease is chronic and multifactorial, affecting protection and support tissues of the tooth. Its onset is due to the accumulation of bacterial plaque, in which are found microorganisms, mainly Gram-negative, which stimulate the host cells and the production of immune-inflammatory molecules. Thus, the objective of this research was to evaluate the effectiveness of two techniques of periodontal treatment through clinical parameters and laboratory tests. For this, 42 patients were randomly evaluated and divided into three groups of 14 patients each: Group 1 (control) - periodontally healthy patients; Group 2 - patients with moderate to severe chronic periodontitis treated with conventional periodontal treatment [quadrant scaling and root planing (Q-SRP)]; and Group 3 - patients with moderate to severe chronic periodontitis treated with full-mouth scaling and root planing (FM-SRP). All of these patients received periodontal treatment and were evaluated using the plaque and gingival indices, probing depth (PD), clinical attachment level (CAL), bleeding on probing (BOP), analysis of prostaglandin  $E_2$  ( $PGE_2$ ) isoform expression and analysis of gingival crevicular fluid (GCF), for a total period of 180 days. The results of the periodontal and laboratory parameters did not show significant differences statistically ( $p > 0.05$ ) when comparing the treatments at 180 days. Therefore, it can be affirmed that both periodontal treatments were effective, but without differences between them. Both treatments improved periodontal and laboratorial clinical parameters significantly. Thus, the professional should evaluate the case and choose the treatment that best suits the needs of the patient and availability.

**Key words:** Periodontal disease, conventional periodontal treatment, full mouth disinfection

## Introduction

Periodontal disease is an inflammatory process that occurs in response to bacterial plaque antigens that accumulate along the gingival margin. Its initial manifestation is gingivitis, characterized by hyperemia, edema, recession and gingival bleeding. If it is not treated early, it may progress to periodontitis. Periodontitis is a multifactorial chronic inflammation caused by microorganisms and characterized by the progressive destruction of tooth support tissues that causes loss of the periodontal

tissues, which can reduce the quality of life, the masticatory function and impair the aesthetics of the patient (Tonetti *et al.*, 2013).

Bacterial plaque is responsible for the onset and maintenance of periodontal disease, but host defense mechanisms are known to play an important role in its pathogenesis (Genco *et al.*, 2002). Several pro- and anti-inflammatory cytokines are produced by different cell types, playing an important role in the pathogenesis of periodontal disease, in order to limit this response (Garlet, 2010; Okada and Muramaki, 1998). It is believed that the inflammatory disease is an imbalance arising from a higher concentration of pro-inflammatory cytokines, and hence a lower concentration of anti-inflammatory cytokines, leading to the destruction of the tissues. Pro-inflammatory cells, such as interleukin-1 beta ( $IL-1\beta$ ) and tumor necrosis factor alpha ( $TNF-\alpha$ ), increase and

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induce the production of prostaglandin  $E_2$  ( $PGE_2$ ) and metalloproteinase matrices (MMPs), molecules that cause destruction of the extracellular matrix of the gingival tissue, periodontal ligament and alveolar bone resorption (Page, 1998).

High levels of  $PGE_2$  appear to cause different responses, suggesting that this mediator plays suppressive and stimulatory actions on the progression of periodontitis (Naito and Yoshikawa, 2005). The potential of  $PGE_2$  in suppressing release of interleukin 6 (IL-6) and  $TNF-\alpha$  occurs because these cytokines regulate the release of MMPs, which play a key role in the degradation of the extracellular matrix of connective tissue that occurs in periodontal disease (Harizi and Norbert, 2004).

In order to restore periodontal health to patients with periodontitis, Badersten *et al.* (1984) proposed and instituted periodontal treatment by means of conventional scaling and smoothing of contaminated root surfaces, performed by quadrants, in weekly or biweekly visits. This method has become the most commonly performed for periodontal disease. It is now known that the clinical success of this traditional model stems mainly from the reduction of periodontopathogens accompanied by an increase in the so-called beneficial bacteria (Cortelli *et al.*, 2010).

Quirynen *et al.* (1995) developed the full-mouth scaling and root planing (FM-SRP) treatment, that is, full mouth disinfection. The original protocol included the disinfection of the whole oral cavity in a period of 24 hours, besides the elimination of plaque and aggregated deposits to the dental surface and preventive measures of biofilm formation using chlorhexidine-based mouthwashes. In addition, disinfection of buccal microbial reservoirs such as the tongue and tonsils and subgingival irrigation of the periodontal pockets were treated three times in ten minutes, also with the use of chlorhexidine. The aim of this method was to eradicate, or at least suppress, periodontal pathogens in a short period of time in all pharyngeal niches (tongue, mucous membranes and saliva) in order to avoid the transmission of pathogens from untreated periodontal pockets to the recently instrumented ones, and also for the pockets in the stage of tissue repair.

This research aimed to evaluate the effectiveness of the two techniques of periodontal treatment through clinical parameters and laboratory tests.

## Methods

The study was a prospective clinical trial with a quantitative approach, carried out at the dental clinic of the State University of the West of Paraná. The data collection period of the project was 180 days and the full time of execution of the research occurred in a period of 14 months, beginning in September 2015 and ending in October 2016. The project was approved by the Research

and Ethics Committee in Human Beings at Unioeste, and the document was registered as n.1.219.516.

The inclusion criteria were: patients could be of both sexes, should have moderate to severe chronic localized or generalized periodontitis, have at least six sites with probing depths greater than 5 mm and clinical insertion level greater or equal to 4 mm, not in the same tooth, with bleeding on probing and gingival inflammation, and should be free of cavities at clinical examination. However, in the group of periodontally healthy patients, they all had sites with probing depth (PD) less than or equal to 3 mm, bleeding on probing less than or equal to 5% and no gingival inflammation, and were free of cavities at clinical examination. The teeth, for all groups, were in a normal position, with a minimum of 20 teeth in the arch, with clinical examination performed on the buccal, lingual/palatal, mesial and distal surfaces.

However, we did not select patients who, in the last six months, used antibiotic therapy, anti-inflammatory steroids or non-steroids, anticoagulants, immunosuppressants and cholesterol regulators, were pregnant or breastfeeding. Also, patients with any kind of systemic problem, using hormonal contraceptive or any other kind of hormone, smokers or who quit smoking in the last 5 years, and those who had periodontal treatment in the previous 6 months.

A total of 42 patients was selected, ranging from 25 to 65 years old, 14 healthy periodontal adults (control group) and 28 adults with moderate to severe periodontal disease, who were divided into two groups. This sample was based on calculation using the analysis of variance (ANOVA) test to calculate the sample size, as well as previous studies found in the literature (Bresolin *et al.*, 2013; Bresolin *et al.*, 2014; Toregeani *et al.*, 2016).

In relation to the groups with moderate or severe periodontitis, one group underwent conventional periodontal treatment, performing manual scraping per quadrant within a 7-day interval, and hygiene orientation. The second group was submitted to periodontal treatment by total mouth disinfection, performing complete scaling within 24 hours, with subgingival application of 0.12% chlorhexidine in the periodontal pockets and mouthwash for 15 days with the same solution. Patients were randomly separated.

## Clinical dental evaluation

The initial clinical examination was performed by a single previously trained examiner, who with a Williams no. 23 periodontal probe, measured:

1. Plaque index of O'Leary (1972): This index divides the tooth surface into four zones - buccal, distal, mesial, and lingual - and designates codes 0 for absence or 1 for presence of visible plaque (dichotomized for absence and presence of visible plaque).

2. Gingival index: The gingival inflammatory condition was evaluated by the Saxton and van der Ouderaa (1989) gingival index (GI), dichotomized for presence or absence of bleeding.
3. Probing depth: The distance from the bottom of the sulcus/pocket to the gingival margin, which was determined at six points: mesio-buccal, mid-buccal, disto-buccal, disto-lingual/palatal, mid-lingual/palatal and mesio-lingual/palatal for each tooth to be examined
4. Clinical attachment level: The distance from the cemento-enamel junction to the apical extent of the sulcus/pocket was determined at six points: mesio-buccal, mid-buccal, disto-buccal, disto-lingual/palatal, mid-lingual/palatal and mesio-lingual/palatal for each tooth to be examined.
5. Bleeding on probing: Presence of bleeding observed after 30 seconds following the probing depth measurement at the same six points.

The basic periodontal treatment consisted of appointments scheduled weekly in the Clinic of Dentistry of UNIOESTE, without restriction of duration. The whole treatment was performed by a single operator and consisted of instruction and motivation for oral hygiene, supragingival and subgingival scaling, root planing and coronary polishing, using manual and ultrasonic instrumentation under the effect of local anesthesia. For manual instrumentation, we used Gracey periodontal curettes 5/6, 7/8, 11/12 and 13/14 (Hu-Friedy, Chicago, IL, USA), and for ultrasonic instrumentation a piezoelectric device was used (Dabi Atlante, Ribeirão Preto, São Paulo, Brazil).

For all groups, the same mechanical plaque control instruction was given, as well as periodontal support therapy in the treated groups. The patients were evaluated for a total period of 6 months, with clinical examinations and analysis of the amount of gingival crevicular fluid (GCF) performed at 0, 3 and 6 month periods. Regarding the immunological analysis of PGE<sub>2</sub> expression was measured at 0 and 6 months.

### Laboratory evaluation

#### Expression analysis of PGE<sub>2</sub> isoform

For Group 1, 5 to 6 sites with probing depth less than or equal to 3 mm (shallow sites) were selected in different teeth. For Groups 2 and 3, 5 to 6 sites were selected with probing depth greater than or equal to 5 mm and bleeding on probing (deep sites).

First, the supragingival plaque was removed from the selected sites with a white conical Robson CA brush (Microdont, São Paulo, Brazil), and then the region was isolated with sterile cotton rolls and gently air-dried. Stagnant GCF was collected by introducing a cone of sterile absorbent paper held for 30 seconds at the selected sites: blood-contaminated specimens were

discarded. Cones containing the fluid from sites with the same characteristics of each patient were packed in a single Eppendorf tube containing 1 mL of phosphate buffered saline (PBS). After collection, the paper cones remained in the Eppendorf tubes for 40 minutes at room temperature. Soon after, centrifugation of the tubes was performed at 12000 rpm for 10 minutes at 4 degrees. The supernatant was pipetted and conditioned in a new sterile Eppendorf and frozen at -80° C. These samples were used to evaluate the amount of prostaglandin E<sub>2</sub> (PGE<sub>2</sub>) by enzyme-linked immunosorbent assay (ELISA; Toledo, 2012).

#### Gingival crevicular fluid analysis

With the use of the white conical Robson CA brush (Microdont, São Paulo, Brazil) a prophylaxis was performed and all plaque removed from the area. Three collections per patient were performed in the central portion of the buccal and lingual/palatal surfaces of random teeth, with filter paper strips (Whatman grade 1) of 2 x 15 mm inserted below the gingival margin for 30 seconds. The paper strips were immediately placed in 0.2% alcohol solution of ninhydrin for 1 minute. The strips were photographed and analyzed with a computer program (Image Pro Plus® Version 4.5.0.29, Media Cybernetics, Silver Spring, MD, USA) to determine the amount of fluid absorbed in mm<sup>2</sup> (Lagos *et al.*, 2011).

Statistical analysis was performed with the aid of the Bioestat 5.3 program (Instituto Mamirauá, Amazonas, Brazil). The Shapiro-Wilk test was used to evaluate the normality of the data. After checking the normality of the data in all periodontal and laboratorial parameters, the averages were compared within each group and presented in tables with the corresponding units and measures, with the average  $\pm$  standard deviation of the average using the ANOVA test and later the TUKEY test ( $p < 0.05$ ) for data analysis. For PGE<sub>2</sub> expression analysis, Student's *t*-test ( $p < 0.05$ ) was used to compare the expression at the initial and final analyses within the same group. For the calculations of the averages variations ( $\Delta$ ), data from the 1st exam (initial phase) and 3rd exam (6 months) were used, using the ANOVA test ( $p < 0.05$ ) for all parameters that presented with normal distribution, after the Shapiro-Wilk test.

### Results

From a total of 68 patients evaluated, 42 patients were selected, following the inclusion and exclusion criteria. There were 30.40% males in Group 1, 64.29% in Group 2, and 57.15% in Group 3. The mean age was  $40.30 \pm 7.89$  years (Group 1 -  $39.57 \pm 6.45$  years, Group 2 -  $41.27 \pm 8.29$  years, Group 3 -  $40.08 \pm 7.95$  years), and no statistical differences in sex and age among the groups were found ( $p > 0.05$ ).

## Clinical dental evaluation

Table 1 shows the averages of the plaque index, PD, insertion level, gingival index and bleeding on probing at 0, 3 and 6 months. The results of this clinical evaluation showed that, in relation to periodontal treatment, the Q-SRP and FM-SRP groups showed significant improvement ( $p < 0.05$ ) in the parameters studied during the 6-month period; however none of them was statistically different ( $p > 0.05$ ) when compared between the two techniques.

The control group had a statistically significant reduction in plaque index ( $p < 0.05$ ), but there were no statistical differences among the other parameters.

Table 2 shows only those sites with moderate to severe chronic periodontitis in clinical evaluations of PD and insertion level, and the treatments in Groups 2 and 3 showed significant improvements ( $p < 0.05$ ) at the periods evaluated, although neither of them presented with statistical superiority ( $p > 0.05$ ) when compared to each other.

## Gingival crevicular fluid analysis

Table 3 presents the means of analysis of GCF during treatment periods. The results demonstrated that there was a significant decrease of the fluid in the Q-SRP and FM-SRP groups ( $p < 0.05$ ) in the period evaluated. However, when comparing the averages of the variations between the treatment techniques, there was no statistically significant difference ( $p > 0.05$ ). The control group did not present a statistically significant reduction ( $p > 0.05$ ).

## Expression analysis of PGE<sub>2</sub> isoform

Table 4 presents the analysis of PGE<sub>2</sub> isoform expression over the 0, 3 and 6 month periods. The results demonstrated that there was a significant decrease of PGE<sub>2</sub> in the Q-SRP and FM-SRP groups ( $p < 0.05$ ) in the period evaluated. However, when comparing the averages of the variations between the treatment techniques, there was no statistically significant difference ( $p > 0.05$ ). The control group did not show statistically significant variation ( $p > 0.05$ ).

## Discussion

In 1995, Quirynen *et al.* initiated the full mouth disinfection treatment with the aim of reducing periodontal pathogens from all areas of the mouth in a single session, so that the disadvantage of reinfection during periodontal therapy could be minimized or avoided. Q-SRP and FM-SRP are effective methods for the treatment of periodontal disease, as demonstrated by a large number of studies (Bollen *et al.*, 1998; Fonseca *et al.*, 2015; Fang *et al.*, 2016). However, the clinical results of both treatments do not seem to show significant advantages when compared and analyzed by other studies (Sagar, 2014). Therefore, the objective of this study was to clinically compare the two techniques of periodontal treatment, followed over a period of 6 months.

**Table 1.** Clinical evaluation of healthy patients and patients with moderate chronic periodontitis.

|          | Groups             |                     |                     |                    |                     |                     |                    |                     |                     |                           |
|----------|--------------------|---------------------|---------------------|--------------------|---------------------|---------------------|--------------------|---------------------|---------------------|---------------------------|
|          | 1 (Control)        |                     |                     | 2 (Q-SRP)          |                     |                     | 3 (FM-SRP)         |                     |                     | Δ (0 - 6)                 |
|          | 1st test (0 month) | 2nd test (3 months) | 3rd test (6 months) | 1st test (0 month) | 2nd test (3 months) | 3rd test (6 months) | 1st test (0 month) | 2nd test (3 months) | 3rd test (6 months) |                           |
| PI (%)   | 15.81 ± 7.75       | 9.51 ± 4.45*        | 9.83 ± 4.26*        | 29.85 ± 10.78      | 12.31 ± 8.24*       | 7.50 ± 3.69*        | 31.10 ± 16.73      | 14.94 ± 7.13*       | 13.92 ± 5.77*       | 18.18 ± 9.12 <sup>b</sup> |
| GI (%)   | 2.78 ± 2.78        | 2.03 ± 1.83         | 1.87 ± 1.81         | 3.47 ± 1.65        | 1.90 ± 1.43*        | 1.49 ± 1.28*        | 6.99 ± 2.43        | 3.94 ± 3.08*        | 3.66 ± 2.69*        | 3.43 ± 2.56 <sup>b</sup>  |
| BOP (%)  | 2.52 ± 2.45        | 1.75 ± 0.69         | 1.34 ± 1.29         | 5.00 ± 3.68        | 2.38 ± 1.72*        | 1.99 ± 1.17*        | 10.20 ± 5.19       | 4.34 ± 2.90*        | 3.73 ± 2.93*        | 6.07 ± 4.96 <sup>b</sup>  |
| PD (mm)  | 1.71 ± 0.31        | 1.66 ± 0.17         | 1.69 ± 0.64         | 3.67 ± 0.93        | 3.03 ± 0.65*        | 2.88 ± 0.62*        | 3.50 ± 1.03        | 2.62 ± 0.76*        | 2.58 ± 0.74*        | 0.95 ± 0.88 <sup>b</sup>  |
| CAL (mm) | 1.83 ± 0.40        | 1.78 ± 0.29         | 1.73 ± 0.70         | 4.24 ± 0.97        | 3.36 ± 0.96*        | 3.21 ± 0.78*        | 4.25 ± 1.38        | 3.27 ± 0.72*        | 3.15 ± 0.72*        | 1.10 ± 0.90 <sup>b</sup>  |

Q-SRP, quadrant scaling and root planing; FM-SRP full-mouth scaling and root planing; PI, plaque index; GI, gingival index; BOP, bleeding on probing; PD, probing depth; CAL, clinical attachment level. The values represent average ± standard deviation and are expressed as percentages for PI, GI and BOP and in millimeters for PD and CAL. \*Statistically significant difference between averages within the same group and of the same parameter ( $p < 0.05$ ). <sup>a,b</sup>Statistically significant difference between Δ (average variations) between groups and in the same parameter ( $p < 0.05$ ).



**Table 2.** Clinical evaluation of probing depth and clinical attachment level of patients with moderate to severe chronic periodontitis.

|          | Groups                |                        |                        |                     |                       |                        |                        |                     |
|----------|-----------------------|------------------------|------------------------|---------------------|-----------------------|------------------------|------------------------|---------------------|
|          | 2 (Q-SRP)             |                        |                        |                     | 3 (FM-SRP)            |                        |                        |                     |
|          | 1st test<br>(0 month) | 2nd test<br>(3 months) | 3rd test<br>(6 months) | $\Delta$<br>(0 - 6) | 1st test<br>(0 month) | 2nd test<br>(3 months) | 3rd test<br>(6 months) | $\Delta$<br>(0 - 6) |
| PD (mm)  | 6.29 $\pm$ 0.63       | 5.48 $\pm$ 0.30*       | 5.19 $\pm$ 0.29*       | 1.10 $\pm$ 0.54     | 6.44 $\pm$ 0.86       | 5.44 $\pm$ 0.45*       | 5.02 $\pm$ 0.21*       | 1.42 $\pm$ 0.42     |
| CAL (mm) | 6.60 $\pm$ 0.65       | 5.87 $\pm$ 0.71*       | 5.27 $\pm$ 0.22*       | 1.33 $\pm$ 0.21     | 6.69 $\pm$ 0.98       | 5.95 $\pm$ 0.63*       | 5.49 $\pm$ 0.25*       | 1.20 $\pm$ 0.22     |

Q-SRP, quadrant scaling and root planing; FM-SRP full-mouth scaling and root planing; PD, probing depth; CAL, clinical attachment level. Values represent average  $\pm$  standard deviation. \*Statistically significant difference between means of the exams within the same group and of the same parameter ( $p < 0.05$ ). No statistically significant difference between  $\Delta$  (average variations) between groups and in the same parameter ( $p > 0.05$ ).

**Table 3.** Analysis of gingival crevicular fluid (GCF) volume.

|                           | Groups                     |                            |                            |                                   |                            |                             |                             |                                        |
|---------------------------|----------------------------|----------------------------|----------------------------|-----------------------------------|----------------------------|-----------------------------|-----------------------------|----------------------------------------|
|                           | 1 (Control)                |                            |                            |                                   | 2 (Q-SRP)                  |                             |                             |                                        |
|                           | 1st test<br>(0 month)      | 2nd test<br>(3 months)     | 3rd test<br>(6 months)     | $\Delta$<br>(0 - 6)               | 1st test<br>(0 month)      | 2nd test<br>(3 months)      | 3rd test<br>(6 months)      | $\Delta$<br>(0 - 6)                    |
| GCF<br>(mm <sup>2</sup> ) | 1034.56<br>$\pm$<br>529.77 | 1188.38<br>$\pm$<br>562.10 | 1014.50<br>$\pm$<br>301.41 | 20.06 $\pm$<br>22.83 <sup>a</sup> | 1917.06<br>$\pm$<br>717.67 | 1365.66<br>$\pm$<br>486.34* | 1256.94<br>$\pm$<br>456.82* | 660.12<br>$\pm$<br>260.84 <sup>b</sup> |
|                           |                            |                            |                            |                                   | 1954.08<br>$\pm$<br>974.25 | 1306.30<br>$\pm$<br>643.84* | 1271.36<br>$\pm$<br>477.35* | 682.71<br>$\pm$<br>496.89 <sup>b</sup> |

Q-SRP, quadrant scaling and root planing; FM-SRP full-mouth scaling and root planing. \*Statistically significant difference between means of the exams within the same group and of the same parameter ( $p < 0.05$ ). Different letters: Statistically significant difference between  $\Delta$  (average variations) between groups ( $p < 0.05$ ).

**Table 4.** Analysis of PGE<sub>2</sub> isoform expression.

|       | Groups                |                        |                        |                                   |                       |                        |                        |                                   |
|-------|-----------------------|------------------------|------------------------|-----------------------------------|-----------------------|------------------------|------------------------|-----------------------------------|
|       | 1 (Control)           |                        |                        |                                   | 2 (Q-SRP)             |                        |                        |                                   |
|       | 1st test<br>(0 month) | 2nd test<br>(6 months) | 3rd test<br>(6 months) | $\Delta$<br>(0 - 6)               | 1st test<br>(0 month) | 2nd test<br>(6 months) | 3rd test<br>(6 months) | $\Delta$<br>(0 - 6)               |
| ELISA | 0.208 $\pm$<br>0.005  | 0.205 $\pm$<br>0.009   | 0.205 $\pm$<br>0.009   | 0.003 $\pm$<br>0.004 <sup>a</sup> | 0.217 $\pm$<br>0.015  | 0.205 $\pm$<br>0.007*  | 0.206 $\pm$<br>0.007*  | 0.012 $\pm$<br>0.013 <sup>b</sup> |

Q-SRP, quadrant scaling and root planing; FM-SRP full-mouth scaling and root planing. \*Statistically significant difference between the averages of the first and second exams within the same group ( $p < 0.05$ ). <sup>a,b</sup>Statistically significant difference between  $\Delta$  (average variations) between groups ( $p < 0.05$ ).

The results presented in relation to the dental clinical evaluation showed that there was a statistically significant improvement of the periodontal disease with both types of treatment, analyzed as shown in *Tables 2* and *3*. These results are in agreement with the studies of Apatzidou and Kinane (2004) that compared the two therapies in a period of 6 months, where they evaluated the PD, the level of clinical insertion and bleeding on probing, and demonstrated that there was no statistically significant difference between the therapies proposed. The authors concluded that the clinician should select the therapy according to its practicality. The results of this study were corroborated by Koshy *et al.* (2005) and Santuchi *et al.*, (2015), who carried out studies to compare Q-SRP and FM-SRP, concluding that full mouth disinfection therapy had limited additional benefits compared to conventional therapy.

Farman and Joshi (2008) wrote a literature review aiming to compare the two techniques of periodontal treatment and concluded that the two forms are effective, and there is no statistical difference between them. In contrast, Quirynen *et al.* (2006) reported that there were benefits generated by FMD compared to the conventional technique and that these were partially due to the use of antiseptics.

Still, supporting our results, Swierkot *et al.* (2009) conducted a study based on clinical and microbiological analyses of conventional periodontal treatment and the whole mouth treatment for eight months. They demonstrated that the proposed treatment modalities were effective after 8 months, and that the use of chlorhexidine did not imply clinical and microbiological advantages.

The most common method of diagnosis of periodontal disease is based on clinical parameters such as PD, insertion loss and bleeding on probing, but they do not allow the identification of disease activity in individual regions (Page and Eke, 2007; Matarasso, 2013). In order to complement the diagnosis of periodontal disease, most of the studies are aimed at analyzing the host's inflammatory response using GCF, which is collected through a noninvasive measure of access to the pathophysiological state of the periodontium of a specific site, and in this way, immunological and biological methods can identify mediators released during periodontal infection (Uitto *et al.*, 2003; Castro *et al.*, 2003). The GCF is the result of the interaction between the bacterial plaque and the cells of the periodontal tissue, and their quantity varies greatly according to the degree of inflammation (Champagne *et al.*, 2003). The amount of GCF showed a decrease after the proposed periodontal treatments were performed, but without statistical superiority of either modality (*Table 2*). With this parameter, we could analyze that all the treatments were effective, causing a reduction of the fluid, and that FM-SRP did not bring any advantage. This result was similar to the conclusion obtained by Santana *et al.* (2014), showing that periodontal treatment through full mouth disinfection did not present sufficient clinical and microbiological results to justify its

use compared to conventional periodontal treatment, and could be used according to the wishes of the professional and the patient.

Patients with periodontitis exhibit high levels of proinflammatory cytokines such as IL-1 $\beta$ , TNF- $\alpha$ , and arachidonic acid metabolites such as PGE<sub>2</sub>. PGE<sub>2</sub> is the most potent mediator of alveolar bone loss in periodontitis and has been detected at higher levels in the gingival tissue and GCF proportional to the severity of periodontal disease (Kardesler *et al.*, 2008). Chibebe *et al.* (2008) evaluated GCF as a method of periodontal diagnosis and concluded that it has a predictive value, allowing the identification of the risk of future alteration at the site examined. This feature allows the identification of individuals who need more frequent follow-up and, therefore, greater possibility of prevention of the onset of periodontal disease, which will lead to new paradigms for the elaboration of effective treatment and prevention strategies for this disease.

In relation to the concentration of prostaglandin E<sub>2</sub> in the gingival fluid, both treatments promoted reduction of this cytokine, but without difference between the groups of moderate and severe periodontitis (*Table 4*). This is explained by the fact that PGE<sub>2</sub> has been understood as a key inflammatory mediator for the onset of periodontal disease, because it induces vasodilation and stimulates the synthesis of metalloproteinases (which degrade the tissue extracellular matrix) and leads to the destruction of connective tissue, besides acting on the bone tissue by inducing the synthesis of collagenase by osteoblasts, thus favoring the onset of bone resorption. Thus, the reduction of levels of this mediator would be expected after periodontal treatment (Alexander *et al.*, 1996; Paquette and Williams, 2000). However, in the studies of Del Peloso *et al.* (2008, 2009), no difference between groups was found in the levels of the mediators investigated in any of the periods studied. A possible explanation, according to the authors, would be the high variability that exists between individuals, which would prevent the verification of difference between the groups and even between the times of evaluation.

Killooy (2002) argued that clinical significance is a subjective assessment and should be based on statistical significance and clinical results. In addition, he listed some possible criteria that, according to him, should be included in the determination of periodontal therapy, such as statistics by percentage of sites that need treatment, morbidity, time to treat, cost, among others. Therefore, clinical significance needs to be better assessed on the basis of evidence (Fang *et al.*, 2016). In addition, there are obvious clinical references to post-treatment discomfort between treatments, and none of the patients had any serious adverse reactions during the studies of Fang *et al.* (2016) or our study, being, from a practical point of view, using FM-SRP as a way to complement Q-SRP.

## Conclusion

Therefore, we can observe that both periodontal treatments were effective in a short period of time, but without significant differences between them. However, both improved periodontal and laboratory clinical parameters significantly. Thus, the professional should evaluate the case of the patient and choose the treatment that best suits needs and availability to attend the consultations.

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