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Subgingival air-polishing treatment in patients with aggressive periodontitis

Tretman subgingivalnog peskarenja kod obolelih od agresivne parodontopatije

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Abstract

Background/Aim. Periodontal tissue maintenance therapy is an important phase of the overall periodontal disease therapy. This paper aims to determine subgingival air-polishing efficacy with glycine powder in putative paropathogens reduction, plaque index, gingival bleeding index and probing depth. **Methods.** The study included 44 patients with aggressive periodontitis of both sexes, aged between 21 and 50, divided into two groups. Subgingival air-polishing was applied in the first group and sonic scaling in the control (second) group. Biofilm samples were taken from 5 deepest periodontal pockets before the therapy and 3 and 15 months after it. Paropathogens *Aggreggatibacter actinomycetemcomitans*, *Porphyromonas gingivalis*, *Tannerella forsithya*, *Prevotella intermedia*, *Treponema denticola* were detected by PCR analysis. **Results.** Paropathogens values were decreased after applied treatments. There was a statistically significant reduction in the mean value of full-mouth plaque (FMPS), from 43.00 to 14.90 (first group) and from 44.71 to 15.54 (second group), full-mouth bleeding score (FMBS) from 42.55 to 13.85 (first group) and from 43.04 to 15.17 (second group), as well as in probing depth from 3.40 to 2.64 (first group) and from 3.85 to 2.91 (second group), three months after the therapy. **Conclusion.** Subgingival air-polishing successfully reduces putative paropathogens and clinical parameters three months after the treatment.

Key words:
periodontal diseases; periodontal pocket; glycine; therapeutics; bacteria; polymerase chain reaction.

Apstrakt

Uvod/Cilj. Terapija održavanja zdravlja potpornog tkiva zuba predstavlja važnu fazu terapije u celokupnom tretmanu obolelog parodoncijuma. Cilj rada bio je utvrditi efikasnost subgingivalnog peskarenja glicinskim prahom na smanjenje verovatnih paropatogena, kliničkih parametara plak indeksa, indeksa krvarenja gingive kao i dubine parodontalnog džepa. **Metode**. U istraživanju su učestvovala 44 bolesnika sa dijagnostikovanom agresivnom parodontopatijom, oba pola, uzrasta od 21 do 50 godina, podeljena u dve grupe. Prvoj grupi je rađeno subgingivalno peskarenje površine korena zuba glicinskim prahom, a kontrolnoj (druga grupa) je rađena klasična sonična obrada površine korena zuba. Uzorci biofilma uzeti su iz 5 najdubljih parodontalnih džepova pre terapije, tri i 15 meseci nakon terapije. Paropatogeni *Aggreggatibacter actinomycetemcomitans, Porphyromonas gingivalis, Tannerella forsithya, Prevotella intermedia, Treponema denticola* detektovani su PCR analizom. **Rezultati.** Vrednosti traženih paropatogena su opale posle primenjenih tretmana. Došlo je do statistički značajnog smanjenja srednjih vrednosti FMPS (*full-mouth plaque score*), sa 43.00 na 14.90 (prva grupa) i sa 44.71 na 15.54 (druga grupa), FMBS (*full-mouth bleeding score*) sa 42.55 na 13.85 (prva grupa) i sa 43.04 na 15.17 (druga grupa), kao i dubine sondiranja sa 3.40 na 2.64 (prva grupa) i sa 3.85 na 2.91 (druga grupa), tri meseca posle terapije. **Zaključak.** Subgingivalno peskarenja uspešno dovodi do smanjenja paropatogena i kliničkih parametara tri meseca nakon terapije.

Ključne reči:
periodontalne bolesti; periodontalni džep; glicin; lečenje; bakterije; polimeraza, reakcija stvaranja lanaca.

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Introduction

Periodontal diseases include chronic inflammatory-destructive changes in the periodontal tissue. The disease arises as a result of the response of host defense factors to the presence of bacteria in the dentogingival junction [1]. The periodontal disease, in the form of generalized aggressive periodontitis, is the most severe of all forms of the supporting tissue disease of the teeth. However, it does not happen often. Periodontitis is one of the leading factors of tooth loss in adult population [2]. Numerous factors are involved in the onset of this disease: microorganisms of dental biofilm, poor oral hygiene, bad habits, etc. Familial predisposition is prominent in this case, therefore, this form of periodontal disease is considered genetic [3, 4]. According to some authors, this form of periodontal disease is a consequence of a reduced immune response to dental biofilm antigen. Persons suffering from aggressive periodontitis have disorders of the function of neutrophil granulocytes and monocytes [5]. It is believed that the greatest potential in causing periodontal diseases is found in the following bacteria: *Aggregatibacter actinomycetemcomitans* (*Aa*), *Porphyromonas gingivalis* (*Pg*), *Prevotella intermedia* (*Pi*), *Tannerella forsythia* (*Tf*), *Treponema denticola* (*Td*), as confirmed by numerous studies [6–10]. These bacteria are designated as putative periodontal pathogens [11–13]. Since aggressive periodontitis is characterized by a disrupted function of defense of the organism from pathogenic microorganisms and other factors, this disease is very unpredictable, both by its clinical progression and its therapeutic response [14, 15]. Contemporary trends in dentistry require a quick, efficient and atraumatic approach to treating the disease. Minimally invasive procedures are used in the treatment of periodontal diseases as well. In this regard, in the treatment of periodontal pockets, researchers try to remove dental biofilm with its contents, which is the main cause of periodontal disease [16]. The removal of biofilm consequently leads to the reduction and withdrawal of inflammation from the soft wall of the periodontal pocket without damaging the surrounding periodontal tissue [17]. The minimal invasive procedure in processing periodontal pockets has replaced the previously used procedure of scaling and root planing (SRP) of the surface of the root, as Ower [18] wrote about it. Subgingival air-polishing using glycine powder on periodontal pockets (Full Mouth PerioFlow – FMPF) is one form of a minimally invasive procedure of treating periodontal pockets, as it is effective in the removal of biofilms from natural tooth structure [19], and at the same time, it does not damage both soft and hard periodontal tissue [20–22]. Flemmig et al. [23] also states there is no damage to soft tissue when subgingival air-polishing with glycine powder is used. This non-surgical, therapeutic approach includes treating the whole mouth as quickly as possible, which differs from the previous treatment that entailed working on periodontal pockets over a longer period of time [24]. FMPF involves the removal of biofilm, and other sediments from the root surface using compressed air, water and powder with varying types of abrasivity [19, 21]. Sodium bicarbonate has been used for air-polishing, which has effectively removed the biofilm. However, sodium bicarbonate particles were up to four times the particle size of glycine powder. Particles of sodium bicarbonate, formed in such a manner, left even larger abrasive traces on the root surface and caused greater damage to the soft tissue [25]. While glycine powder particles are 45–60 microns in size, sodium bicarbonate particles are up to 250 microns in size. In addition, the usage of the periodontal pockets polishing (PFLOW) method, including a mixture of glycine powder, causes considerably smaller damage to the soft tissue in comparison to the previous technologies [26, 27]. Furthermore, air-polishing, in comparison to other techniques of treatment of periodontal pockets, has the advantage of preserving the cement of the root of the tooth [20]. The new PFLOW technology uses a specially designed nozzle with three outlets, one for air, one for powder, and one for water. This improves the mixing of water and air with powder, the extraction of the powder from the depth of the periodontal pocket while preventing, at the same time, emphysema of the periodontal pocket soft tissue. Designed in such a manner, the nozzle allows access to the periodontal pocket with the depth of up to 5 mm. The new subgingival air-polishing method increases the efficiency of work and provides better comfort to the patient [28].

The goal of this paper was to determine the effect of subgingival glycine air-polishing on the subgingival periodontal pathogens during maintenance therapy. Moreover, it was intended to estimate the clinical parameters of periodontal tissue, like plaque index, gingival bleeding index and probing depth.

Methods

The maintenance therapy results were recorded 3 and 15 months after applying the first treatment. The study included 44 patients suffering from aggressive periodontitis, both genders of different ages, 27 were non-smokers, and 17 were smokers.

The patients were selected according to the criteria defined for diagnosing aggressive periodontitis [29].

Inclusion criteria were as follows: clinical and radiological features of generalized aggressive periodontitis; patients with good general health; at least 20 teeth present.

Exclusion criteria were as follows: clinical and radiological features of localized aggressive periodontitis; presence of some kind of systemic disease; pregnancy or lactation; scaling and root planing within three months of examination; periodontal surgery and/or antibiotic therapy within six months of examination.

The study was approved by the Research Board of the Medical Faculty, University of Banja Luka. The research has been conducted in full accordance with the ethical principle of the Declaration of Helsinki. All patients involved in the study had signed a letter of approval confirming they would take part in the study of their own will. Upon receiving their consents, thorough medical and dental histories were taken from the subjects. Dental exams and dental radiographs

analysis were done for each patient 15 months after the basic therapy. Two co-authors participated in the patient selection, as well as in the basic therapy, besides the main researcher, the author of the study (SM, TN). All clinical assessments, radiographic analysis, sampling for microbiological and laboratory tests were performed by one person for the uniformity of the results.

The samples of subgingival biofilm, required for microbiological analysis, were taken from the five deepest periodontal pockets with sterile paper points (#30, Absorbent paper points, Taper 0.2 VDW GmbH, Germany) for each patient separately. The selected area was well isolated from the saliva with cotton rolls and dried thoroughly. Samples of subgingival biofilm were taken by using five sterile paper points, with one paper point placed in each of the periodontal pockets. Ninety seconds later, the paper points were gently taken out from the pockets and placed in a sterile 1.5 mL Eppendorf tube and transported the same day to the laboratory that used the micro-IDent test (Hain Lifescience GmbH, Nehren, Germany) for the blind analysis. The necessary DNA extraction from periodontal pathogens was done between 24 and 48 hours from the moment of taking the sample. In case the analysis could not be done within this timeframe, the samples were frozen at -20° C and the analysis was carried out within seven days of the sampling time.

PCR analysis was applied for analyzing DNA from periodontal pockets, according to the manufacturer's instructions. The microbiological assessment of the samples consisted of isolating the DNA from the sample, followed by the PCR process, and concluded by the identification of PCR products (amplicons) by reverse hybridization.

Sampling and monitoring of the results was done at baseline and 3 and 15 months after the initial therapy. The initial division of the patients was followed, which means that during the maintenance therapy, one group underwent the subgingival air-polishing, while the second group had the classic sonic treatment of periodontal pockets. Samples of subgingival biofilm were taken from the same periodontal pockets. Taking samples from periodontal pockets was done before determining clinical parameters in order to avoid biofilm destruction by periodontal probing.

To assess the state of oral hygiene and gingivitis the following indexes were used: Full Mouth Plaque Score (FMPS) and Full Mouth Bleeding Score (FMBS) [30].

The FMPS is determined by examining four surfaces of the tooth (vestibular, oral, mesial and distal) and recording a positive or negative value for each surface of the tooth, depending on whether the dental biofilm was present or absent. All present teeth were examined, except for third molars. The presence (+) or the absence (-) of the dental biofilm is recorded in the dental record, and the total value of dental biofilm is expressed in percentages:

FMPS = Number of dental surfaces with biofilm ×100/number of examined dental surfaces.

Therefore, the FMPS is the percentage of teeth surface with plaque accumulation that was evaluated using a periodontal probe.

The FMBS is determined by probing the gingiva associated with four teeth surfaces (vestibular, oral, mesial and distal) and recording a positive or negative value for each surface of the tooth, depending on whether the gingiva bleeding was present 30 seconds after probing or not. The gingiva is probed with all teeth present, except for third molars. The presence (+) or the absence (-) of bleeding is recorded in the dental record, and the total value of this index is expressed in percentages:

FMBS = Number of dental surfaces where gingiva bleeding is present x100/ number of examined dental surfaces.

The FMBS is the percentage of teeth surface with bleeding upon.

To assess the condition of deeper structures of periodontal tissues, probing depth (PD) was measured with a periodontal probe. These measurements were made using the periodontal probe PCP-UNC 15®, Hu-Friedy, Chicago, IL, USA.

Clinical work in patients' mouths was performed in the following way:

During the first checkup and upon the completion of examination and sample taking, supragingival calculus was removed in all patients, supragingival air-polishing was done and subgingival concrements were removed as well. After this, the patients were divided into two groups.

Subgingival air polishing of root surfaces was performed on the first (test) group of patients, using a glycine amino acid in the form of 45–60 micron powder granules (EMS Air-Flow Master). Each root surface area was air-polished for 4 or 5 seconds. The nozzle for subgingival air-polishing is presented in Figure 1.

Fig.1 – Specially designed nozzle for subgingival air-polishing.

Sonic SRP of the root surfaces was performed on the second (control) group of patients. The procedure was done with a sonic device and periodontal nozzles (Figure 2).

Fig. 2 – Periodontal nozzles for sonic devices.

This systemic treatment of all periodontal pockets was performed in the following four appointments. The patients took care of their oral hygiene in the home environment according to our instructions. An ultra-soft manual toothbrush was used for oral hygiene.

The patients rinsed their oral cavities with 0.12% chlorhexidine digluconate (Curasept ADS 212, Curaden, Kriens, Switzerland) twice a day during the course of the therapy and continued to do so for another 10 days. Upon the completion of the fourth appointment and within 24 hours upon the finished treatment of periodontal pockets, the patients in both groups were prescribed antibiotic therapy, depending on the results of the microbiological analysis. In the tested group, there were no subjects allergic to penicillin or metronidazole. Therefore, the antimicrobial therapy consisted of amoxicillin 500 mg capsules and metronidazole 400 mg tablets or a combination of the two medicines. The subjects took the medication or medications three times a day for 7 days [31–33].

Follow-up checkups were performed 6 to 8 weeks after the first appointment and periodontal tissues were re-assessed (FMPS, FMBS, and PD were measured again). The second stage came three months after the start of the therapy. All the aforementioned measurements were repeated, as was the laboratory PCR analysis, which examined the possible presence of periodontal pathogens. On the basis of the obtained results, it was assessed whether or not a further therapeutic procedure is necessary and what kind.

A re-evaluation of the required parameters was performed 15 months after the first appointment to examine the effect of maintenance therapy. All parameters from the first visit were re-determined, and PCR analysis was also performed. Removal of any solid deposits was carried out using Gracy's curettes.

Statistical analysis

IBM SPSS Statistics 19.0: MS Office Word 2010 was the software used for the statistical analysis and tables.

The Wilcoxon sign test is a statistical comparison of the average of two dependent samples. The Wilcoxon sign test works with metric (interval or ratio) data that is not multivariate normal or with ranked/ordinal data. Generally, it is the non-parametric alternative to the dependent samples *t*-test. The Wilcoxon sign test tests the null hypothesis that the average signed-rank of two dependent samples is zero.

Mann-Whitney *U* test is used to compare two independent groups. This test is equivalent to a *t*-test of independent samples, however, this test does not assume a normal distribution of the analyzed data. This test is used when the analyzed data are not in normal distribution and it is inappropriate to use the *t*-test on independent samples.

Fisher's exact test is used when the sample size is too low to use chi-square (χ^2) test.

The χ^2 test serves to determine whether some of the obtained (observed) frequencies deviate from the frequencies expected under a particular hypothesis. This test also requires the correlation of two variables and it shows the probability of the variables' correlation.

Statistically significant values are assumed values of $p < 0.05$.

Results

Forty-four patients (24 females, 20 males; 27 non-smokers and 17 smokers) suffering from aggressive periodontitis were included in the study. Twenty and 24 of them were treated by the PFLOW and SRP, respectively.

PCR analysis demonstrates the reduction in the presence of the baseline bacteria observed through the monitoring period of 3 and 15 months, in relation to the number and percentage of patients examined (Table 1).

After the PFLOW therapeutic procedure, the percentage of patients with very elevated *Aa* decreased from 30% to 5% (3 months) and 5% (15 months). The percentage of patients in whom this bacteria was not detected increased from 45% to 80% and 80%, respectively.

After the SRP therapeutic procedure, the percentage of patients with very elevated *Aa* dropped from 29.2% to 8.3% and 8.3%, respectively. The percentage of patients in whom this bacteria was not detected increased from 50% to 87.5% and 87.5%, respectively.

The PFLOW treatment, applied at the beginning of the therapy, and 3 and 15 months after the maintenance therapy was performed, led to a reduction of the percentage of highly elevated *Pg* with the initial value of 65% to 5% and 5%, respectively. The percentage of patients who did not have *Pg* bacteria detected increased from the initial 10% to 50% and 45%, respectively. During the SRP therapeutic procedures, at the beginning of the treatment, 58.3% of patients had a very high *Pg* value of bacteria, and after 3 and 15 months of maintenance therapy, none of the patients had a very high value of this bacteria. The percentage of patients without detected bacterium *Pg* from the initial 16.7% increased to 54.2%, 3 months later and to 58.3%, 15 months later.

After the PFLOW therapeutic procedure, the percentage of patients with very high *Pi* bacteria decreased from the initial 15% to 0%, both 3 and 15 months after the performed maintenance therapy. In 45% of patients, *Pi* bacteria were

Table 1

Microbiological data expressed by the number and percentage of patients

Bacteria	Treatment		Checkup					
			T0		T3		T15	
			n	%	n	%	n	%
Aa	PFLOW	Not detected	9	45	16*	80	16*	80
		Slightly elevated	5	25	2	19	2	10
		Elevated	0	0	1	5	1	5
		Highly elevated	6	30	1*	5	1*	5
	SRP	Not detected	12	50	21*	87.5	21*	87.5
		Slightly elevated	5	20.8	1	4.2	1	4.2
		Elevated	0	0	0	0	0	0
		Highly elevated	7	29.2	2	8.3	2	8.3
Pg	PFLOW	Not detected	2	10	10*	50	9*	45
		Slightly elevated	2	10	9*	45	9*	45
		Elevated	3	15	0	0	1	5
		Highly elevated	13	65	1*	5	1*	5
	SRP	Not detected	4	16.7	13*	54.2	14*	58.3
		Slightly elevated	2	8.3	9*	37.5	9*	37.5
		Elevated	4	16.7	2	8.3	1	4.2
		Highly elevated	14	58.3	0*	0	0*	0
Pi	PFLOW	Not detected	9	45	18*	90	16*	80
		Slightly elevated	5	25	1	5	0*	0
		Elevated	3	15	1	5	0	0
		Highly elevated	3	15	0	0	0	0
	SRP	Not detected	10	41.7	20*	83.3	22*	91.7
		Slightly elevated	2	8.3	4	16.7	1	4.2
		Elevated	8	33.3	0*	0	0*	0
		Highly elevated	4	16.7	0*	0	0*	0
Tf	PFLOW	Not detected	0	0	10*	50	12*	60
		Slightly elevated	1	5	4	20	4	20
		Elevated	7	35	5	25	4	20
		Highly elevated	12	60	1*	5	0*	0
	SRP	Not detected	0	0	17*	70.8	15*	62.5
		Slightly elevated	3	12.5	5	20.8	5	20.8
		Elevated	5	20.8	2	8.3	3	12.5
		Highly elevated	16	66.7	0*	0	1*	4.2
Td	PFLOW	Not detected	2	10	10*	50	13*	65
		Slightly elevated	4	20	7	35	5	25
		Elevated	8	40	3	15	2*	10
		Highly elevated	6	30	0*	0	0*	0
	SRP	Not detected	1	4.2	16*	66.7	13*	54.2
		Slightly elevated	7	29.2	6	25	8	33.3
		Elevated	11	45.8	2*	8.3	3*	12.5
		Highly elevated	5	20.8	0*	0	0*	0

***Aa – Aggregaticacter actinomycetemcomitans*; *Pg – Porphyromonas gingivalis*; *Pi-Prevotella intermedia*; *Tf – Tannerella forsithya*; *Td – Treponema denticola*; PFLOW – Subgingival Air-polishing; SRP – Scaling and Root Planing; T0 – baseline (no treatment); T3 – 3 months after tretament; T15 – 15 months after treatment; n (%) – number (percentage) of patients.**

***statistical significance was present 3 and 15 months after PFLOW and SRP therapy compared with the baseline values ($p < 0.05$).**

not detected before therapy, while 3 months after therapy, the percentage of patients without these bacteria increased to 90%, and 15 months after initial therapy, the percentage was 80%.

Very elevated *Pi* bacteria were recorded in 16.7% of patients. After the SRP treatment (3 and 15 months duration), this value was 0%. The percentage of patients that did not have *Pi* bacteria was 41.7%, and after the maintenance therapy, this value was 83.3% (3 months) and 91.7% (15 months).

After the PFLOW treatment, the percentage of patients with highly elevated levels of *Tf* dropped from 60% to 5% after 3 months, and after 15 months, no patients with very high values of these bacteria were recorded. The percentage of patients without detected *Tf* bacteria increased from the initial 0% to 50% after 3 months and to 60% after 15 months.

After the SRP therapeutic procedure, the percentage of patients with very high *Tf* bacteria values from the initial 66.7% decreased to 0% after 3 months and to 4.2% after 15 months. The percentage of patients without detected *Tf* bacteria increased from the initial 0% to 70.8% after 3 months and to 62.5% after 15 months.

After the PFLOW treatment, the percentage of patients with a very elevated value of *Td* bacteria value from the initial 30% dropped to 0% after 3 months and to 0% after 15 months. The percentage of patients without detected *Td* bacteria from the initial 10% increased to 50% after 3 months and to 65% after 15 months.

The SRP treatment led to a reduction in the highly elevated *Td* bacteria from the initial 20.8% to 0% after 3 months and to 0% after 15 months. The percentage of patients without *Td* bacteria at the beginning of the treatment was 4.2%, 3 months after SRP treatment, this value was 66.7% and after 15 months 54.2%.

Full mouth plaque score

In the group of patients that underwent the PFLOW therapy, there was a statistically significant ($p < 0.05$) enhancement of the FMPS index value 3 months after the performed therapy.

However, 15 months after PFLOW therapy, FMPS index values were not statistically nor significantly different ($p > 0.05$) than the initial state (Table 2).

In the group of patients that underwent the SRP therapy, there was a statistically significant ($p < 0.05$) improvement in the FMPS index value 3 months after the therapy compared to the initial value. However, 15 months after the initial therapy, the FMPS index value was not statistically different ($p > 0.05$) compared to the initial value (Table 2).

Full mouth bleeding score

In the group of patients that underwent the PFLOW therapy, there was statistically significant ($p < 0.05$) improvement in the FMBS index value 3 months after the performed therapy. However, 15 months after PFLOW therapy, the values of the FMBS index were not significantly different ($p > 0.05$) compared to the initial state (Table 3).

In the group of patients that underwent the SRP therapeutic method, there was a statistically significant ($p < 0.05$) improvement in the FMBS index value 3 months

Table 2

Clinical data for FMPS

Parameters	PFLOW				SRP			
	n	mean ± SD	min.	max.	n	mean ± SD	min.	max.
FMPS T0	20	43.00 ± 24.66	12.00	81.00	24	44.71 ± 25.03	11.00	88.00
FMPS T3	20	14.90 ± 7.50	4.00	30.00	24	15.54 ± 7.33	2.00	30.00
FMPS T15	20	42.35 ± 24.68	12.00	88.00	24	45.25 ± 24.96	11.00	87.00

FMPS – Full Mouth Plaque Score; PFLOW – Subgingival Air-polishing; SRP – Scaling and Root Planing; T0 – baseline (no treatment); T3 – 3 months after treatment; T15 – 15 months after treatment; n – number of patients; min – minimum; max – maximum; SD – standard deviation.
***p* < 0.05, 3 months after PFLOW and SRP therapy.**

Table 3

Clinical data for FMBS

Parameters	PFLOW				SRP			
	n	mean ± SD	min.	max.	n	mean ± SD	min.	max.
FMBS T0	20	43.00 ± 24.66	12.00	81.00	24	44.71 ± 25.03	11.00	88.00
FMBS T3	20	13.85 ± 8.20	1.00	29.00	24	15.17 ± 9.28	2.00	37.00
FMBS T15	20	42.55 ± 22.14	9.00	84.00	24	43.04±19.36	9.00	75.00

FMBS – Full Mouth Bleeding Score; PFLOW - Subgingival Air-polishing; SRP – Scaling and Root Planing; T0 – baseline (no treatment); T3 – three months after treatment; T15 – 15 months after treatment; n – number of patients; min – minimum; max – maximum; SD – standard deviation.
***p* < 0.05, 3 months after PFLOW and SRP therapy.**

after the therapy compared to the initial value. However, 15 months after the initial therapy, the FMBS index value was not statistically different ($p > 0.05$) compared to the initial value (Table 3).

Probing depth

In the group of patients that underwent the PFLOW therapy, there was a statistically significant ($p < 0.05$) reduction in the probing depth 3 months after the initial value. However, 15 months after PFLOW therapy, the probing depth was not statistically significantly different ($p > 0.05$) compared to the initial state (Table 4).

In the group of patients that underwent the SRP therapeutic method, there was a statistically significant ($p < 0.05$) decrease in the depth of the pocket 3 months after the therapy compared to the initial value. However, 15 months after initial therapy, the probing depth value was not significantly different ($p > 0.05$) compared to the initial values (Table 4).

Correlation in the PFLOW therapy group was found between *Aa* bacteria and some clinical parameters. In the group of patients undergoing PFLOW therapy, there was a statistically significant ($p < 0.05$) medium-strong positive correlation, before the therapy itself (T0), between the bacteria *Aa* and the probing depth. A higher percentage of *Aa* bacteria was present in deeper periodontal pockets. Three months after PFLOW therapy (T3), there was a mean strong positive statistically significant correlation ($p < 0.05$) between the bacteria *Aa* and the PD. The correlation positivity shows that a higher percentage of *Aa* bacteria is present in deeper periodontal pockets. Statistically significant ($p < 0.05$) medium-strong positive correlation existed between *Aa* and FMBS parameter after 15 months (T15). The correlation positivity indicates that higher FMBS parameters increase the frequency of *Aa* bacteria (Table 5).

Discussion

When certain types of bacteria were tested, subgingival air-polishing of periodontal pockets accompanied by antimicrobial therapy showed equally efficient results as the sonic scaling of periodontal pockets. *Aggregatibacter actinomycetemcomitans* is frequently present in subgingival biofilm in patients suffering from generalized aggressive periodontitis, which is also confirmed by studies carried out globally [6, 10, 34]. *Aa* was found in slightly more than half (52.27%) of the patients in the tested group. The data from other studies state a similar prevalence of *Aa* in subjects suffering from aggressive periodontitis [7, 13, 35]. Taking into account the results received in this but also in some other studies, it can be noted that patients suffering from aggressive periodontitis have a lower percentage of *Aa*. A positive effect of the applied method is also proved by data that *Aa* was found in only 15.9% of the patients at the end of the study.

A high percentage of *Porphyromonas gingivalis* was detected during the study (86.36%). Among the tested periodontal pathogens in the studies of aggressive periodontitis, *Pg* was present in the very high percentage [13], which indicates a significant role of this periodontal pathogen in the pathogenesis of aggressive periodontitis. In some studies conducted in India, *Pg* was detected only in persons with advanced periodontitis, unlike the persons with healthy periodontal tissue, indicating a link between *Pg* and a disease of periodontal tissue [36]. High prevalence of this bacterium in subgingival biofilm in persons with generalized aggressive periodontitis indicates its connection with this

Table 4

Clinical data for PD

Parameters	PFLOW				SRP			
	n	mean ± SD	min.	max.	n	mean ± SD	min.	max.
PD T0	20	3.40±0.73	2.4	5.03	24	3.85±0.77	2.85	5.87
PD T3	20	2.64±0.41	2.09	3.44	24	2.91±0.61	2.19	4.79
PD T15	20	3.57±0.81	2.4	5.87	24	3.71±0.76	2.44	5.44

PD – periodontal depth; PFLOW – Subgingival Air-polishing; SRP – Scaling and Root Planing; T0 – baseline (no treatment); T3 – 3 months after treatment; T15 – 15 months after treatment; n – number of patients; min – minimum; max –maximum; SD – standard deviation.
***p* < 0.05, 3 months after PFLOW and SRP therapy.**

Table 5

Influence of PFLOW treatment on correlation between *Aggregatibacter actinomycetemcomitans* and clinical parameters in patients (n = 20) with aggressive periodontitis

Clinical parameters	T0		T3		T15	
	r	*p*	r	*p*	r	*p*
FMPS	0.170	0.475	0.127	0.593	0.180	0.448
FMBS	0.145	0.542	0.024	0.920	0.451	0.046
PD	0.471	0.036	0.474	0.035	0.085	0.721

PFLOW – Subgingival Air-polishing; T0 – baseline (no treatment); T3 – 3 months after treatment; T15 – 15 months after treatment; FMPS – Full Mouth Plaque Score; FMBS – Full Mouth Bleeding Score; PD – Periodontal Depth; r – coefficient of correlation; *p* – significance.

disease. The data from a study that used PCR methodology confirmed that *Pg* was present only in the regions with the disease of periodontal tissue, unlike the healthy regions where *Pg* was not identified [13, 37]. This bacterium was found in over 60% of subjects with aggressive periodontitis with pocket depth up to 5 mm and over 90% of subjects with pocket depth more than 5 mm [38]. This indicates that bacteria *Pg* is significantly correlated with aggressive periodontitis. The grater the destruction of periodontal tissue, the greater the percentage of bacteria *Pg*. Other studies, too, record a high prevalence of this putative periodontal pathogen among the subjects suffering from aggressive periodontitis, bringing it in connection with periodontal disease [7, 9]. A positive effect of applied therapies is also confirmed by the fact that this periodontal pathogen was found in 47.72% of the patients at the end of the therapy.

The prevalence of *Prevotella intermedia* was 56.81% in this study. In comparison with healthy regions of periodontal tissue, the microbiological profile of subgingival biofilm of the regions with aggressive periodontitis contained a significant amount of *Pi*, which confirms that *Pi* is a significant etiological factor in the occurrence of aggressive periodontitis [37]. Studies conducted in Japan point out that the prevalence of *Pi* was similar to the one registered in this study [39]. PCR analysis of subgingival biofilm in Chinese patients confirmed that *Pi* was a dominant periodontal pathogen in persons suffering from aggressive periodontitis [40]. A study conducted in Lebanon also confirmed that this was one of the possible causes of periodontal tissue disease. In the patients suffering from periodontitis, *Pi* was also present in subgingival biofilm samples, making it an integral part of the microbiological profile of patients suffering from aggressive periodontitis [11]. Studies conducted in Marocco indicated a higher prevalence of this periodontal pathogen in the subgingival biofilm of a diseased periodontium [7]. The data shows that the percentage of *Pi* detected at the end of therapy was 13.63%, which proves that the applied method had a positive effect.

The findings of this study confirm the role of *Tannerella forsythia* in the pathogenesis of periodontitis. Of all the five putative periodontal pathogens tested during this study, this was the only bacterium that was present in all patients of the study. Identical results were obtained in a study carried out among the Bulgarian patients suffering from aggressive periodontitis [13]. The periodontal pathogens were found to exist in high percentages in Maroccan patients as well, where the bacterium was dominant [7]. Persistent bleeding on probing, an indicator of an infection, encouraged numerous researchers to repeat their studies, which then confirmed the presence of *Tf* in those regions [41]. Studies conducted worldwide point out that *Tf*, as a resident of subgingival biofilm in persons suffering from aggressive periodontitis, was present in high percentages [6–8]. After therapy, this periodontal pathogen was present in only 38.63% of the patients, indicating a positive effect of the applied therapy.

Treponema denticola is also considered one of the possible causes of periodontal tissue destruction occurring in aggressive periodontitis, detected in 93.18% of the patients. Recent studies have shown the prevalence of *Td* in more than 80% of the patients and that is significantly associated with the severity of periodontal tissue destruction. High percentages of *Td* were found especially in the regions where periodontal pockets were deeper than 4 mm [36]. High percentages of *Td* in the subgingival biofilm were registered among the residents of different geographical areas, which was confirmed by numerous studies [6, 8, 9, 13]. The efficacy of the applied therapy is evident from the *Td* finding, the percentage being 40.90% at the end of therapy.

The efficacy of individual therapeutic procedures applied in this study was examined with the FMPS and FMBS indexes and monitoring of PD. Both therapeutic procedures, the PFLOW and the SRP, applied in the patients, produce an equally statistically significant reduction in the FMPS in both groups, from 43.00 to 14.90, (first group) and from 44.71 to 15.54 (second group) and FMBS, also in both groups, from 42.55 to 13.85 (first group) and from 43.04 to 15.17 (second group), three months after the treatment. These results match the results obtained in the studies carried out in China and some other parts of the world [9, 21, 31]. There are various mechanical control techniques of biofilm accumulation that consequently lead to a reduction in the signs of inflammation in periodontal soft tissue [41].

The results obtained in this study indicate that the new therapeutic procedure PFLOW successfully stopped further destruction of periodontal tissue by monitoring PD (probing depth), with the initial value of 3.40 dropping to 2.64 (first group) and from 3.85 to 2.91 in the second group, three months after therapy. Other studies also indicate that all of the applied techniques successfully reduce the levels of inflammation and stop further damage to the periodontal tissue [9, 42]. Monitoring the parameters of gingival inflammation and signs of periodontal destruction through PD, some other studies also find that PFLOW can be considered successful in removing the subgingival biofilm and therefore stop the further destruction of periodontal tissue [8, 27, 31]. Since both the SRP and PFLOW have similar clinical achievements, especially in maintaining healthy periodontal tissue, precedence can be given to the PFLOW as the patient accepts it more easily and the dentist spends less time working on it.

Fifteen months after therapy, the FMPS, FMBS and PD parameters showed statistical equality compared to the values recorded at the beginning of the study, indicating a drop in patient motivation and irregular oral hygiene maintenance. It is very important to emphasize to patients the role of biofilm in the occurrence and development of periodontal tissue destruction during every control checkup. Regular removal of biofilm reduces and eliminates the agglomeration of probable periodontal bacteria that have a large share in the occurrence of periodontitis. By constant motivation and education in

maintaining oral hygiene, patients must become aware of their own roles in maintaining the health of the periodontal tissue.

Conclusion

During the maintenance therapy of the health of the tooth-supporting tissue, there was no growth and proliferation of the required periodontal pathogens, after both 3 and 15 months. The new therapeutic procedure in the scope of minimally invasive treatment of periodontal tissue proved to be equally successful in the eradication of the studied putative periodontal pathogens, as well as in the reduction of the monitored clinical parameters such as plaque index, gingival bleeding index and PD, as the previous sonic SRP therapy.

The regular subgingival air-polishing and the regular re-motivation of patients for maintaining oral hygiene in home conditions in the first three months did not lead to an increase in the value of the clinical parameters monitored. However, a control examination after 15 months showed that there was no improvement in clinical parameters indicating that the motivation of patients undergoing the maintenance therapy of the health of the teeth supporting tissue had dropped. Based on this, it can be seen that the subgingival air-polishing of the surface of the root of the tooth has successfully led to the reduction of the tracked microbiological parameters, and it can be recommended to be used during the maintenance therapy. It is especially acceptable to be used in patients with a compromised periodontal status who have fixed prosthetics in order to prolong the duration of such prosthetic work. It is necessary to carry out more frequent controls with these patients so that the lack of motivation in maintaining oral hygiene is avoided.

The dentist is supposed to choose a mode of therapy concerning the feasibility of certain procedures, following the patients' needs.

R E F E R E N C E S

1. *Cekici A, Kantarci A, Hasturk H, Van Dyke TE.* Inflammatory and immune pathways in the pathogenesis of periodontal disease. Periodontol 2000 2014; 64(1): 57–80.
2. *Tonetti MS, Jepsen S, Jin L, Otomo-Corgel J.* Impact of the global burden of periodontal diseases on health, nutrition and wellbeing of mankind: A call forglobal action. J Clin Periodontol 2017; 44(5): 456–62.
3. *Vieira AR, Albandar JM.* Role of genetic factors in the pathogenesis of aggressive periodontitis. Periodontol 2000 2014; 65(1): 92–106.
4. *Rusyanti Y, Widyaputra S, Maskoen AA.* Periodontal tissue destruction in aggressive periodontitis: Determination of gene or environmental factors. Saudi Dent J 2019; 31(2): 290–9.
5. *Tapashetti RP, Sharma S, Patil SR, Guwa S.* Potential effect of neutrophil functional disorders on pathogenesis of aggressive periodontitis. J Contemp Dent Pract 2013; 14(3): 387–93.
6. *Könönen E, Müller HP.* Microbiology of aggressive periodontitis. Periodontol 2000 2014; 65(1): 46–78.
7. *Chahboun H, Arnau MM, Herrera D, Sanz M, Ennibi OK.* Bacterial profile of aggressive periodontitis in Morocco: a cross-sectional study. BMC Oral Health. 2015; 15: 25.
8. *Kargas K, Tsalikis L, Sakellari D, Menexes G, Konstantinidis A.* Pilot study on the clinical and microbiological effect of subgingival glycine powder air polishing using a cannula-like jet. Int J Dent Hyg 2015; 13(3): 161–9.
9. *Lu H, He L, Zhao Y, Meng H.* The effect of supragingival glycine air polishing on periodontitis during maintenece therapy: a randomized controlled trial. PeerJ 2018; 6: e4371.
10. *Lourenço TG1, Heller D, Silva-Boghossian CM, Cotton SL, Paster BJ, Colombo AP.* Microbial Signature Profiles of Periodontally Healthy and Diseased Patients. J Clin Periodontol 2014; 41(11): 1027–36.
11. *Al Yahfoufi Z.* Prevalence of Periodontal Destruction and Putative Periodontal Pathogens in the Same Lebanese Family. J Contemp Dent Pract 2017; 18(10): 970–6.
12. *Mombelli A, Almaghlouth A, Cionca N, Cancela J, Courvoisier DS, Giannopoulou C.* Microbiologic Response to Periodontal Therapy and Multivariable Prediction of Clinical Outcome. J Periodontol 2017; 88(12): 1253–62.
13. *Kotsilkov K, Popova C, Boyanova L, Setchanova L, Mitov I.* Comparison of culture method and real-time PCR for detection of putative periodontopathogenic bacteria in deep periodontal pockets. Biotechnol Biotechnol Equip 2015; 29(5): 996–1002.
14. *Teughels W, Dhondt R, Dekeyser C, Quirynen M.* Treatment of aggressive periodontitis. Periodontol 2000 2014; 65(1): 10733.
15. *Nibali V.* Aggressive Periodontitis: microbes and host response, who to blame? Virulence 2015; 6(3): 223–8.
16. *Sans M, Beighton D, Curtis MA, Cury JA, Dige I, Dommisch H,* et al. Role of microbial biofilms in the maintenance of oral health and in the development of dental caries and periodontal diseases. Consensus report of group 1 of the Joint EFP/ORCA workshop on the boundaries between caries and periodontal disease. J Clin Periodontol 2017; 44(Suppl18): S5–S11.
17. *Larsen T, Fiehn NE.* Dental biofilm infections – an update. APMIS 2017; 125(4): 376–84.
18. *Ower P.* Minimally-invasive non-surgical periodontal therapy. Dent Update 2013; 40(4): 289–90, 293–5.
19. *Lu H, He L, Zhao Y, Meng H.* The effect of supragingival glycine air polishing on periodontitis during maintenance therapy: a randomized controlled trial. PeerJ 2018; 6: e4371.
20. *Bozbay E, Dominici F, Gokbuget AY, Cintan S, Guida L, Aydin MS,* et.al. Preservation of root cementum: a comparative evaluation of power-driven versus hand instruments. Int J Dent Hyg 2018; 16(2): 202–9.
21. *Simon CJ, Munivenkatappa Lakshmaiah Venkatesh P, Chickanna R.* Efficacy of glycine powder air polishing in comparison with sodium bicarbonate air polishing and ultrasonic scaling - a double-blind clinico-histopathologic study. Int J Dent Hyg 2015; 13(3): 177–83.
22. *Bühler J, Amato M, Weiger R, Walter C.* A systematic review on the effects of air polishing devices on oral tissues. Int J Dent Hyg 2016; 14(1): 15–28.
23. *Cobb CM, Daubert DM, Davis K, Deming J, Flemmig TF, Pattison A,* et al. Consensus Conference Findings on Supragingival and Subgingival Air Polishing. Compend Contin Educ Dent 2017; 38(2): e1–e4.
24. *Flemmig TF, Arushanov D, Daubert D, Rothen M, Mueller G, Leroux BG.* Randomized controlled trial assessing efficacy and safety of glycine powder air polishing in moderate-to-deep periodontal pockets. J Periodontol 2012; 83(4): 444–52.

25. *Bühler J, Schmidli F, Weiger R, Walter C.* Analysis of the effects of air polishing powders containing sodium bicarbonate and glycine on human teeth. Clin Oral Investig 2015; 19(4): 877–85.
26. *Graumann SJ, Sensat ML, Stoltenberg JL.* Air Polishing: A Review of Current Literature. J Dent Hyg 2013; 87(4): 173–80.
27. *Caygur A, Albaba MR, Berberoglu A, Yilmaz HG.* Efficacy of glycine powder air-polishing combined with scaling and root planing in the treatment of periodontitis and halitosis: A randomised clinical study. J Int Med Res 2017; 45(3): 1168–74.
28. *Sultan DA, Hill RG, Gillam DG.* Air-Polishing in Subgingival Root Debridement: A Critical Literature Review. J Dent Oral Biol 2017; 2(10): 1065.
29. *Albandar JM.* Aggressive periodontitis: case definition and diagnostic criteria. Periodontol 2000 2014; 65(1): 13–26.
30. *The British Society of Periodontology.* Periodontology in General Dental Practice in the United Kingdom. A policy statement. Liverpool: The British Society of Periodontology; 2001.
31. *Usin MM, Tabares SM, Menso J, deAlbera ER, Sembaj A.* Generalized aggressive periodontitis: microbiological composition and clinical parameters in non-surgical therapy. Acta Odontol Latinoam 2016; 29(3): 255–61.
32. *Pretzl B, Sälzer S, Ehmke B, Schlagenhauf U, Dannewitz B, Dommisch H,* et al. Administration of systemic antibiotics during non-surgical periodontal therapy-a consensus report. Clin Oral Investig 2019; 23(7): 3073–85.
33. *Keestra JA, Grosjean I, Coucke W, Quirynen M, Teughels W.* Non-surgical periodontal therapy with systemic antibiotics in patients with untreated aggressive periodontitis: a systematic review and meta-analysis. J Periodontal Res 2015; 50(6): 689–706.
34. *Liu J, Zhao J, Li C, Yu N, Zhang D, Pan Y.* Clinical and microbiological effect of nonsurgical periodontal therapy on patients with chronic and aggressive periodontitis. Quintessence Int 2013; 44(8): 575–83.
35. *Belibasakis GN, Schmidlin PR, Sahrmann P.* Molecular microbiological evaluation of subgingival biofilm sampling by paper points and curette. Acta Pathol Microbiol Immunol Scand 2014; 122(4): 347–52.
36. *Kumawat RM, Ganvir SM, Hazarey VK, Qureshi A, Purohit HJ.* Detection of *Porphyromonas gingivalis* and *Treponema denticola* in chronic and aggressive periodontitis patients: A comparative polymerase chain reaction study. Contemp Clin Dent 2016; 7(4): 481–6.
37. *Stingu CS, Jentsch H, Eick S, Schaumann R, Knöfler G, Rodloff A.* Microbial profile of patients with periodontitis compared with healthy subjects. Quintessence Int 2012; 43: 23–31.
38. *Kulkarni PG, Gosavi S, Haricharan PB, Malgikar S, Mudrakola DP, Turagam N,* et al. Molecular Detection of Porphyromonas gingivalis in Chronic Periodontitis Patients. J Contemp Dent Pract 2018; 19(8): 992–6.
39. *Tomita S, Kasai S, Ihara Y, Imamura K, Kita D, Ota K,* et al. Effects of systemic administration of sitafloxacin on subgingival microflora and antimicrobial susceptibility profile in acute periodontal lesions. Microb Pathog 2014; 71–72: 1–7.
40. *Feng X, Zhang L, Xu L, Meng H, Lu R, Chen Z,* et al. Detection of eight periodontal microorganisms and distribution of Porphyromonas gingivalis fimA genotypes in Chinese patients with aggressive periodontitis. J Periodontol. 2014; 85(1): 150–9.
41. *Tomita S, Komiya-Ito A, Imamura K, Kita D, Ota K, Takayama S,* et al. Prevalence of Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis and Tannerella forsythia in Japanese patients with generalized chronic and aggressive periodontitis. Microb Pathog 2013; 61-62: 11–5.
42. *Jiao J, Zhang L, Meng HX, Shi D, Lu RF, Xu L,* et al. Clinical performance of non-surgical periodontal therapy in a large Chinese population with generalized aggressive periodontitis. J Clin Periodontol. 2018; 45(10): 1184–97.

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