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The Effect of Inhaled Corticosteroid Therapy on Periodontal Status in Patients with Asthma

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Abstract: Asthma belongs to a broad group of allergic diseases and is the most common chronic disease found in children and adults up to four decades of age. Already published studies suggest that the use of inhaled anti-asthmatic medications affects both general health and oral health. The study included 120 adult patients aged 18 to 71 divided into three groups: I "PERIO-ASTHMA"—with asthma and periodontitis, II "ASTHMA"—with asthma without periodontitis, and control group III "CONTROL"—without asthma and periodontitis. The patients were monitored for oral health status for six months. Oral health was assessed by dental indices: DMF index, PI, BoP, PD, and CAL at interproximal sites. Significantly deeper pockets in the lateral segments of the dentition were observed in all study groups. In addition, the bronchial-only group had significantly deeper pockets in the lateral sextants than the control group at all time points. There was no significant worsening of periodontal status by CAL and PD in asthma patients treated with inhalation, irrespective of baseline periodontal status. Depending on the severity of asthma, an increase in BOP was observed in the central sextant in patients without periodontitis. The history of asthma and the use of ICS has an impact on the periodontal status of patients.

Keywords: asthma; periodontitis; periodontal status; inhaled corticosteroids; oral hygiene

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1. Introduction

Asthma belongs to a broad group of allergic diseases and is the most common chronic disease occurring in children and adults. In 2018, the prevalence of asthma was 3 per 100,000 patients [1]. The diagnosis of asthma is based on several tests, including spirometry and imaging studies [2]. It is now known that there is a bidirectional relationship between the condition of the oral cavity and the body's general health. The presence of pathological changes in the oral cavity negatively affects the course of some systemic diseases. Conversely, the existence of general pathologies affects disease processes in the oral cavity. More and more studies emphasize the relationship between gingivitis and periodontitis and cardiovascular diseases, increased risk of non-hemorrhagic stroke, diabetes, and respiratory diseases [3–8].

Inhaled corticosteroids (ICSs) are currently the most effective asthma-control medications. Many factors have been identified that influence the effective dose level that achieves maximum clinical effect, e.g., estimated efficacy index, duration of treatment, asthma severity, type of inhaler, patient age, and asthma duration [9]. Since the implementation of therapy with these drugs, the safety of their use for general health has been and still is

questioned. The problem is all the more critical because ICSs are currently the mainstay of treatment in children, adolescents, and adults. There are often indications for the use of medium or high doses. It is estimated that 20% of patients with asthma need to take high doses of ICS. Therapy is usually carried out over weeks, months, or even years. The primary function of these agents is anti-inflammatory. These drugs prevent the remodeling and fibrosis of the affected bronchial mucosa and prevent the development of permanent and irreversible changes in the respiratory system. The decision to start treatment with ICSs and determine its intensity and duration should be preceded by a risk–benefit balance [10].

When deciding whether to start treatment with inhaled corticosteroids, potential side effects should be considered. Inhaled corticosteroid therapy results in the deposition of the drug substance in the mouth and throat. Since there is a need to take these drugs for many years, the contact with and effect on the oral cavity is prolonged and repeated [11]. Local side effects of inhaled corticosteroid therapy occur in approximately 10–30% of patients [12]. The primary treatment contributes to the development of candidiasis accompanied by hoarseness and cough, dysphonia, thirst sensation and dry mouth, changes in the composition and rate of saliva secretion, disturbances in the hard tissues of the teeth, periodontium, and irritation of the oral mucosa and the area around the corners of the mouth [13].

Studies already published indicate that the use of inhaled anti-asthmatic drugs affects general health and oral health. Compared to healthy individuals, patients treated with ICS are characterized by: more severe caries, more frequent periodontal disease, significantly impaired oral hygiene, more frequent malocclusions and parafunction, which are attempts to extinguish stress and emotional disturbances resulting from the symptoms and course of the disease [14–16].

In conclusion, treatment with ICSs is not an indifferent therapy; however, the benefits of stabilizing the patient's condition and improving his quality of life, controlling respiratory symptoms, and preventing irreversible changes in the airways outweigh the negative effects of the therapy [17]. In the published literature, few studies address the relationship between asthma, chronic use of inhaled corticosteroids, and the presence or development of periodontal disease in adults. It seems that the determination of the influence of these drugs on the development of periodontopathic pathology may significantly contribute to the improvement of control, treatment, and prevention of adverse consequences of chronic therapy in everyday dental and periodontal practice.

The literature emphasizes the strong association of periodontal disease with normal salivary flow [18]. The influence of asthma on periodontal disease may be due to immunological and inflammatory processes in the patient's organism, taking anti-asthmatic medications, especially inhaled ones, or both [19]. The influence of saliva secretion on periodontal disease, highlighted earlier, is also essential when considering the impact of asthma on periodontal disease. The impairment of the physiology of periodontal tissues may result from a reduced protective effect of saliva, which is combined with increased dry mouth resulting from the process itself, which is asthma, which may cause mouth breathing in the patient, as well as changes in the composition of saliva and changes in the amount of saliva, but may also result from the effects of ICS [20]. This can affect changes in the amount of bacterial and immunological factors, significantly decreasing immunoglobulin A [19]. In addition, changes in the amount of immunoglobulin E and microelements, such as calcium and phosphorus, affect susceptibility to increased tartar accumulation [19]. The increased prevalence of periodontal disease in asthmatic patients, especially those taking ICS, may also be due to the effect of ICS on bone mineral density, including that in the maxilla and mandible [21].

The literature emphasizes that long-term use of ICS, especially in high doses, may increase bone fragility [22]. These facts may suggest an influence on the onset and progression of periodontitis in patients using ICS [23,24]. Because of the chronicity of therapy, it is worth evaluating the side effects of these medications on the oral cavity and, if possible, trying to reduce or eliminate them.

The literature emphasizes the occurrence of carious lesions frequently on the labial surfaces of the anterior teeth and the occlusal surfaces of the posterior teeth. Therefore, the authors decided to evaluate the oral cavity as a whole and divide it into frontal and lateral [25].

The purpose of this study was to evaluate the effect of ICS use on periodontal tissue status in different areas of the oral cavity, with particular emphasis on the central and lateral sextants. As a null hypothesis, it was assumed that ICS use by patients with asthma affects the deterioration of periodontal status. The following null hypothesis was postulated: H_0—There are no differences in periodontal status between groups of asthma and periodontal disease patients taking ICS, asthma patients without periodontal disease taking ICS, and generally healthy individuals. Alternative hypothesis: H_1—There are differences in periodontal status between groups of asthma and periodontal disease patients taking ICS, asthma patients without periodontal disease taking ICS, and generally healthy individuals.

2. Materials and Methods

The study was conducted with the approval of the Bioethics Committee at the Medical University (no. KB-0012/27/11). Patients of periodontal clinics who attended periodontal disease treatment, preventive check-ups, or hygiene visits were included in the study (Group I). Group II and III patients came in for follow-up visits, such as for professional teeth cleaning. Patients were introduced to the study and its purpose. The subjects were informed that participation in the study is entirely voluntary and refusal to participate would not affect their treatment in any way. They were also informed about the right to withdraw from the study at any time without giving any reason and bearing consequences. Having read the information about the research and having had any doubts clarified, the participants agreed in writing to participate in the study. The study was conducted from January to October 2014.

120 patients were recruited using convenience sampling, ranging from 18 to 71 years (arithmetic mean 41 years). Three 40-person study groups were formed:

- I group—periodontitis subjects with diagnosed asthma, treated with ICSs ("PERIOASTHMA" group);
- II group—persons with diagnosed asthma without periodontitis, treated with ICSs (group "ASTHMA");
- III group—healthy subjects without asthma and without periodontitis (group "CONTROL").

2.1. Eligibity Criteria

During the examination on the first visit, the patients were asked about any health complaints and medications they were taking. Among the patients who gave a history of asthma and took chronic ICS, those with periodontal disease were also distinguished. A pulmonologist made the diagnosis of asthma. In addition, a control group without periodontal disease or asthma was collected. Patients taking only other anti-asthma medications were excluded from the study. Patients with at least 12 teeth with at least two support zones preserved were included in the study.

Patients were monitored for oral health over six months. Initially, a baseline examination (BE) was performed, during which a history was taken, periodontal tissue status parameters were measured, and a dental prophylaxis and treatment plan was arranged depending on the individual needs of the subjects. Oral hygiene instruction with motivation and teaching of oral prophylaxis procedures was carried out. After the initial examination, non-surgical periodontal treatment (supragingival scaling, subgingival scaling, and root planning) was given to subjects diagnosed with gingivitis or periodontitis. An investigation after two weeks was conducted to standardize methods of hygienic behavior, including brushing, use of the interdental system, education including motivation, and renewed prophylactic recommendations related to the use of inhaled steroids (e.g., rinsing

the mouth after application). After non-surgical periodontal treatment, another periodontal examination was performed (examination one B1). Then, by assessing the clinical depth of periodontal pockets (PD—probing depth) and clinical attachment level (CAL—clinical attachment level) from interproximal sites, patients were assigned to different groups based on Page and Eke classification.

Subsequent examinations took place 3 and 6 months (second examination B2, third examination B3) after the baseline examination (BE). The occurrence of periodontal disease requiring intensive treatment or asthma requiring intravenous drug supply was treated as the study endpoint.

Physical examination—a questionnaire included patient data, information on previous treatment, general health status, and a simplified dental history. The physical examination included:

- Dental diagram;
- Examination of the DMF index: D—teeth with caries, M—extracted teeth, F—filled teeth [26];
- Plaque index (PI) according to Silness and Löe [27];
- Bleeding on probing (BOP) according to Ainamo and Bay [28];
- Clinical attachment level (CAL);
- Probing depth (PD).

Clinical examinations were performed under constant conditions of the dental office in natural and artificial light using a mirror and a dental and periodontal probe calibrated in 1 mm increments by Hu-Friedy (UNC 15).

Unfortunately, it could not be judged whether patients were compliant between study time points.

Examination after two weeks was conducted to standardize hygiene methods and behaviors, including brushing, interdental system use, education, motivation, and reiteration of prophylactic recommendations related to inhaled steroid use (e.g., rinsing the mouth after application).

Currently, asthma staging classification is based on a minimum level of treatment in-tensity that allows asthma to be well controlled. The first and second stages are mild asthma, the third and fourth are moderate, and the fifth is severe [29].

Step 1—Immediate low-dose ICS + formoterol
Step 2—Low-dose ICS or immediate low-dose ICS + formoterol
Step 3—Low-dose ICS + LABA (long-acting β2-mimetic)
Step 4—Intermediate dose of ICS + LABA
Step 5—High-dose ICS + LABA

Based on this classification, patients were classified into groups I to V, as noted in Table 1.

2.2. Methods of Examination and Follow-Up

Two physicians performed all clinical examinations. Two periodontal specialists performed each measurement to confirm the result obtained by the first physician. Analyzing the periodontal status at the second, follow-up visit (examination two weeks after the baseline examination), where clinical periodontal pocket depth (PD—probing depth) and clinical attachment level (CAL—clinical attachment level) were assessed, patients were assigned to different groups based on Page and Eke classification [30] as follows:

0—no periodontitis;
1—moderate periodontitis;
2—severe periodontitis.

PD and CAL were examined at 6 measurement points—on the proximal and distal surfaces at the points—tangential, lingual/sublingual, and buccal/labial sides.

Table 1. Baseline characteristics.

Tested Parameter	Group I PERIO-ASTHMA $n = 40$	Group II ASTHMA $n = 40$	Group III CONTROL $n = 40$	p-Value $\alpha = 0.05$	p-Value I/II	p-Value I/III $\alpha = 0.016$	p-Value II/III
Age—years (mean)	54	33	37	0.0001 *	0.000001 $	0.000003 $	0.566 $
Sex: M–W	32–8	24–16	25–15	0.115 ***			
Page–Eke classification Baseline							
0	0	40 (100%)	40 (100%)				
1	32 (80%)	0	0 (0%)	0.00001 ***			$p = 0.4739$ **
2	8 (20%)	0	0				
Page–Eke classification 2nd examination							
0	0	40 (100%)	38 (95%)				
1	32 (80%)	0	2 (5%)				
2	8 (20%)	0	0				

* Kruskal–Wallis ANOVA ** Pearson's Chi^2 with Yates' correction *** Pearson's Chi^2 for multivariate tables $ Mann–Whitney U test.

Clinical Attachment Level (CAL) Measurement Methodology

The distance from the enamel–cement junction to the bottom of the gingival pocket was measured in millimeters at six measurement points (on the vestibular side proximal-medial-distal and the oral side proper proximal-medial-distal). CAL was tested with a Hu-Friedy periodontal probe (UNC 15) with a scale every 1 mm with a force of 25 N.

2.3. Statistical Analysis of the Results

In the present study, 0.05 was used as the significance level α, and the calculated test statistics p were compared to this value. Due to the comparison of 3 groups, after revealing a significant difference between the groups in the overall tests, the Bonferroni correction of the significance level was applied in the post hoc analysis to avoid an error of the first kind (rejection of the true null hypothesis)—the new significance levels are marked in the tables. Nominal and ordinal variables were presented as frequencies (in percentages). For their analysis, Pearson's Chi-squared tests were used for multivariate tables and Pearson's Chi-squared test with Yates' correction. Because most variables did not have a normal distribution (confirmed by W, Shapiro–Wilk test), collected continuous variables were presented in tables as means with 95% confidence interval and medians with interquartile range 25–75%. Additionally, for this reason, nonparametric tests were used in the analysis.

Kruskal–Wallis ANOVA was used to compare variables between groups (independent variables), and, for post hoc analysis, Mann–Whitney U test with Bonferroni correction was used. In the within-group study of, e.g., outcomes at baseline and after six months (dependent variables), Friedman's ANOVA with post hoc analysis using Wilcoxon's paired-order test with Bonferroni correction was used. Data for analysis were collected and preprocessed using Microsoft Office licensed Excel (version 2007 for PC, 2011 for Mac). Statistical analysis was performed using Statistica version 10 licensed software from Statsoft.

3. Results

Group I "PERIO-ASTHMA"—Subjects diagnosed with asthma treated with ICS and periodontal disease. Eight patients (20%) had a duration of asthma of less than one year, thirteen patients (33%) had been ill for 1–3 years, and nineteen patients (48%) had asthma for more than three years. Among the subjects there were 16 patients (40%) with Gin level

2 of anti-asthma treatment intensity, 18 patients (45%) with level 3, 4 patients (10%) with level 4, and 2 patients (5%) with level 5. Twenty-seven patients reported the existence of comorbidities: 2 patients (5%) had diabetes mellitus, 17 patients (43%) were treated for hypertension, two patients had benign thyroid tumors and one was a mastectomy patient (8%), four patients (10%) had a history of hypothyroidism, and four patients (10%) had a diagnosis of autoimmune disease.

Group II "ASTHMA"—Asthma patients treated with inhaled corticosteroids without periodontitis. The duration of asthma was less than one year in 14 patients (35%), eight patients (20%) had been ill for 1–3 years, and 18 patients (45%) for more than three years. Among the subjects, there were 17 patients (43%) with the second degree of intensity of anti-asthmatic treatment, according to Gin, 17 patients (43%) with the third, five patients with the fourth (13%), and one patient (3%) with fifth. The existence of a history of comorbidities was reported by 14 patients (35%). Two patients (5%) were treated for hypertension, four patients (10%) had hypothyroidism, three patients (8%) were diagnosed with autoimmune diseases, and two patients (5%) had cancer.

Group III "CONTROL"—Subjects without asthma and periodontal disease. Five patients (12%) had a history of comorbidities: two patients (5%) were treated for hypertension and three patients (7.5%) had hypothyroidism. There were no statistically significant differences in asthma duration between group I (PERIO-ASTHMA) with a median of 36 months (IQR 24–96) and group II (ASTHMA) with a median of 33.5 months (IQR 7.5–120). Table 1 presents the detailed data.

Measurements of periodontal indices in central and lateral sextants were compared in each group. Detailed data are shown in Tables 2–4. Table 5 shows the intergroup comparison of periodontal indices.

Table 2. Comparison of clinical indicators between central and lateral sextants in group I (PERIO–ASTHMA).

	Central Sextants			Lateral Sextants			
	Median	**Quartile 25%**	**Quartile 75%**	**Median**	**Quartile 25%**	**Quartile 75%**	***** **(α = 0.05), p =**
Baseline							
CAL	0.438	0.087	1.403	1.408	0.637	2.042	0.000109
PD	2.500	2.315	2.771	2.656	2.374	2.953	0.000408
After 2 weeks							
CAL	0.438	0.099	1.403	1.406	0.637	2.061	0.000030
PD	2.336	2.208	2.669	2.386	2.236	2.849	0.008766
After 3 months							
CAL	0.467	0.073	1.410	1.440	0.628	2.134	0.000030
PD	2.469	2.198	2.642	2.546	2.299	2.866	0.003619
After 6 months							
CAL	0.462	0.073	1.432	1.482	0.623	2.157	0.000025
PD	2.271	2.094	2.514	2.573	2.290	2.703	0.000004

* Wilcoxon test.

Table 3. Comparison of clinical parameters between central and lateral sextants in group II (ASTHMA).

	Central Sextants			Lateral Sextants			
	Median	**Quartile 25%**	**Quartile 75%**	**Median**	**Quartile 25%**	**Quartile 75%**	* **(α = 0.05), p =**
Baseline CAL	0.000	0.000	0.000	0.000	0.000	0.138	0.036581
PD	2.094	1.990	2.218	2.278	2.166	2.420	0.000000
After 2 weeks CAL	0.000	0.000	0.000	0.000	0.000	0.138	0.036581
PD	2.073	2.010	2.146	2.242	2.086	2.405	0.000000
After 3 months CAL	0.000	0.000	0.000	0.000	0.000	0.148	0.038580
PD	2.052	2.000	2.188	2.280	2.121	2.371	0.000000
After 6 months CAL	0.000	0.000	0.000	0.000	0.000	0.148	0.031105
PD	2.000	1.927	2.063	2.207	2.121	2.297	0.000000

* Wilcoxon test.

Table 4. Comparison of clinical parameters between central and lateral sextants in the control group.

	Central Sextants			Lateral Sextants			
	Median	**Quartile 25%**	**Quartile 75%**	**Median**	**Quartile 25%**	**Quartile 75%**	* **(α = 0.05), p =**
Baseline CAL	0.000	0.000	0.000	0.000	0.000	0.000	0.999999
PD	1.823	1.573	2.000	1.981	1.680	2.085	0.000001
After 2 weeks CAL	0.000	0.000	0.000	0.000	0.000	0.161	0.001782
PD	1.740	1.583	1.956	1.886	1.781	2.049	0.000008
After 3 months CAL	0.000	0.000	0.000	0.000	0.000	0.179	0.001782
PD	1.729	1.573	2.000	1.909	1.685	2.063	0.000002
After 6 months CAL	0.000	0.000	0.000	0.000	0.000	0.161	0.001184
PD	1.677	1.563	1.792	1.839	1.730	2.025	0.000014

* Wilcoxon test.

Periodontal parameters were also compared between groups for four timepoints—on the study day, two weeks, three months, and after six months. Detailed results are presented in the Supplementary Materials (Tables S1–S11).

Table 3 shows comparison of clinical indicators between central and lateral sextants in group II. There were significant differences between central and lateral sextants for CAL, PD parameters in BE, after 2 weeks for CAL and PD, after 3 months for CAL and PD and for PI, and PD, CAL and PI after 6 months.

Table 4 shows comparison of clinical indicators between central and lateral sextants in group III. There were significant differences between central and lateral sextants for CAL, PD parameters in BE, after 2 weeks for CAL, BOP, and PD, after 3 months for CAL and PD and for PI, and PD, CAL and PI after 6 months.

Table 5 shows the intergroup comparison between periodontal indices for the whole mouth.

Table 5. Intergroup comparison of differences between studies of selected variables (CAL, PD, PI, BOP)—for the whole mouth.

	PERIO-ASTHMA n **= 40**	**ASTHMA** n **= 40**	**CONTROL** n **= 40**	*		**	
					I/II	**I/III**	**II/III**
	Median (IQR)	**Median (IQR)**	**Median (IQR)**	α **= 0.05**	α **= 0.017 (Bonferroni Correction)**		
CAL							
W-3	0 (−0.03–0.04)	0(0–0)	0(0–0)	0.9231			
W-1	0(0–0)	0(0–0)	0(0–0)	0.6774			
1–2	0 (−0.02–0.03)	0(0–0)	0(0–0)	0.5279			
2–3	0 (−0.01–0.03)	0(0–0)	0(0–0)	0.5991			
1–3	0 (−0.04–0.04)	0(0–0)	0(0–0)	0.9431			
PD							
W-3	−0.17 (−0.38–0.1)	−0.09 (−0.18–0.04)	−0.03 (−0.19–0.05)	0.3144			
W-1	−0.13 (−0.25–0.05)	−0.04 (−0.15–0.03)	−0.01 (−0.12–0.1)	0.0879			
1–2	0.06 (−0.1–0.21)	−0.01 (−0.13–0.16)	0(−0.1–0.13)	0.4782			
2–3	−0.14 (−0.27–0.03)	−0.05 (−0.19–0.04)	−0.06 (−0.11–0.04)	0.2363			
1–3	−0.05 (−0.26–0.14)	−0.07 (−0.18–0.09)	−0.05 (−0.13–0.04)	0.9462			
PI							
W-3	−0.08 (−0.24–0.01)	−0.05 (−0.12–0)	0(−0.05–0)	0.0141	0.9999	0.0151	0.1092
W-1	−0.09 (−0.17–0.01)	−0.04 (−0.12–0)	0(−0.03–0)	0.0052	0.8954	0.0041	0.0931
1–2	0.02 (−0.05–0.1)	−0.01 (−0.06–0.07)	0(0–0)	0.7973			
2–3	−0.04 (−0.15–0.05)	−0.02 (−0.08–0.03)	0(0–0)	0.2634			
1–3	−0.05 (−0.14–0.05)	−0.03 (−0.07–0.04)	0(−0.01–0)	0.5392			
BOP							
W-3	−0.06 (−0.48–0.39)	−0.05 (−0.27–0.07)	−0.08 (−0.2–0)	0.6411			
W-1	−0.25 (−0.53–−0.06)	−0.11 (−0.33–0.02)	−0.02 (−0.18–0)	0.0222	0.2091	0.0212	0.9999
1–2	0.17 (−0.13–0.35)	0(−0.07–0.09)	0(0–0.08)	0.0984			
2–3	0.14 (−0.08–0.46)	0.01 (−0.11–0.15)	0(−0.12–0)	0.0110	0.5992	0.0081	0.2559
1–3	0.13 (−0.1–0.49)	0(−0.11–0.18)	0(−0.05–0)	0.0120	0.3782	0.0082	0.4079
W-3	Difference between baseline and after 6 months						
W-1	Difference between baseline and after 2 weeks						
1–2	Difference between after 2 weeks and after 3 months						
2–3	Difference between after 3 months and after 6 months						
1–3	Difference between after 2 weeks and after 6 months						

* ANOVA Kruskal–Wallis, ** U Mann–Whitney.

4. Discussion

From the perspective of the continuous progress made in the therapy of asthma, which is an increasingly recognized condition in populations of developed countries, the adverse effects of corticosteroids used for this purpose remain a problem [31,32]. Even though they have a high therapeutic index and their systemic effects are limited, at the same time, about 80% of the fraction of inhaled ICS is deposited in the mouth and throat. Because of the very high local activity of modern inhaled drugs, they can act directly on oral

tissues, including periodontal tissues [31]. In asthma, periods of long-term remission may occur, but patients should be under constant medical care because its treatment lasts for life [33]. Many papers describe side effects after long-term treatment with ICSs on dental and periodontal status in children [14,34–40]. Although the disease occurs in patients of all age groups, only a little of the literature presents the effect of anti-asthmatic drug therapy on oral health in adults, hence the interest of our group in this topic and the attempt to collect the most interesting and detailed publications and compare them with the material of our group. The literature published to date has shown evidence of a relationship between asthma and periodontal disease, but the results are inconclusive. Some authors [41,42] describe an inverse relationship between periodontitis and the occurrence of asthma, supporting the so-called hygiene hypothesis [43]. Other authors [44,45] explain the lack of a significant association between asthma and periodontitis. Studies showing more frequent or more severe periodontitis symptoms significantly in asthmatic patients have also been published [46–48].

Periodontal status was assessed using basic periodontal parameters such as PD and CAL. The analysis of mean PD for the whole oral cavity in the study showed significant differences between all groups. The deepest pockets were noted in group I with a median of 2.58 mm, followed by group II with 2.19 mm, and the shallowest in the control group with 1.87 mm. This compares to the work of Komernik [49], who evaluated the effect of inhaled ICS therapy in the course of chronic obstructive pulmonary disease (COPD) in patients with periodontitis and showed a median PD greater than that in group of patients with periodontitis—3.41 mm. Considering the literature on periodontitis, the most similar results to ours were by Torressap [50], whose mean PD was 2.87 mm. In other papers, the mean PD ranged from a level of 3.1 mm to 3.66 mm [51]. When divided into shallow ($\leq$3 mm), medium (4–5 mm), and deep ($\geq$6 mm) pockets, the proportions (mean values) were: shallow: group I—86.02%, medium: group I—11.47%, deep: group I—1.51%.

The most similar proportions of pockets in a group of patients with periodontitis were published by Haffajje et al., respectively: shallow in group II—74%, medium in group II—24%, deep in group II—3% [52]. In contrast, in Sherman's work [53], the proportions were 43%, 48%, and 9%, respectively, significantly indicating more severe periodontal disease. In assessing the loss of connective tissue attachment, the only group we had that had a significantly greater loss for the entire mouth, with a median of 0.97 mm, was group I, which was due to the burden of periodontitis on this group. Additionally, it was found that the percentage of sites with CAL > 4 mm was 2.5%. From the literature collected by me, the most similar results to ours were obtained by Friedrich et al. [54], who studied 170 subjects with type 1 diabetes and assessed the relationship between periodontitis and respiratory allergies. For subjects with allergies, including asthma, the mean CAL was 1.56 mm, and the proportion of sites with CAL > 3 mm was 18.8%; in the group without allergies, the mean CAL was 2.31 mm, and the ratio of sites with CAL > 3 mm was 36%. According to the authors, these data are a strong argument supporting the inverse relationship between periodontal disease and respiratory allergies: the so-called hygiene hypothesis.

Friedrich et al. [41] conducted a study within the Study of Health in Pomerania (SHIP) project on a subpopulation of 2837 subjects, 438 of whom suffered from respiratory allergies (hay fever, house dust mite allergy, asthma). They examined the relationship to periodontal disease by assessing clinical loss of connective tissue attachment > 3 mm (4 surfaces on up to 14 teeth were concerned). The authors found it in 37% of the tested sites in control subjects, 24% in the hay fever group, 22% in the house dust mite allergy group, and 33% in the asthma group. Compared to the papers describing patients with periodontitis, the results from our group of patients with periodontitis and asthma were significantly lower. Haffajje et al. [52] evaluated the effect of non-surgical treatment in 57 adults with chronic periodontitis. The initial mean level of clinical assessed parameters was: percentage of sites with plaque present 66%; BOP 57%; PD 3.3; CAL 2.9; the proportion of shallow pockets 63%, medium 34%, and deep 3%.

Kim et al. [51], in 41 patients with periodontitis, conducted an analysis of the effectiveness of scaling and root planning and surgical treatment (OP), taking into account patient- or tooth-related risk factors. After initial periodontal management—supragingival scaling and OHI—PDs were measured at four sites at each tooth. For areas less than 3 mm, no additional periodontal measures were performed. Subgingival scaling was performed for pockets 3–6 mm. For pockets greater than 6 mm, surgical treatment was performed. Considering the division into anterior and premolar teeth, which corresponds to our division into central and lateral sextants, they showed significant differences in the initial mean PD for anterior teeth and premolars in clinical attachment and the plaque index. For the change in PD, there was a positive effect of baseline and a negative effect of cigarette smoking, interproximal site, premolar teeth, and baseline value of clinical attachment level. It is noteworthy that the final regression models did not include other patient-dependent factors such as age and gender. Shulman et al. [44], in a population-based cross-sectional study involving 238 asthmatic children aged 13–17 and 1250 healthy controls, found no statistically significant differences in the number of bleeding sites, the presence of supragingival and subgingival calculus, the number of sites with PDs greater than 3 mm, and the loss of clinical connective tissue at an attachment level greater than 2 mm.

Nowadays, the association between asthma and oral status is emphasized in the literature [55]. Ferreira et al. conducted a systematic literature review with meta-analysis. The authors stated that asthma may be a risk factor for periodontal disease in study populations. A meta-analysis showed that asthma patients had higher BoP and CAL than healthy controls [56].

Limitations of our study included the small study group. The study included only 120 participants, 40 in each study and control group. Unfortunately, the power of the study was also not calculated because all patients available at the time were included in the study. In a later stage of the study, we plan to increase the study group. The small size of the study groups may bias the results and inaccurately reflect the prevalence of the problem in the community. Unfortunately, the relationship between asthma treatment intensity and periodontitis was not determined, which we plan to do in future studies with a larger control group. Asthma treatment intensity showed what medications and in what doses patients were taking. The baseline characteristics also showed the patients' prevalence of comorbidities. Unfortunately, the differences between the groups were statistically significant. A limitation of our study was also the use of Page and Eke's classification. This is because the study was started before the new classification was introduced.

The results of our study are significant because of the increasing number of people with asthma. A dentist with knowledge of a patient's medical history can recommend a personalized prophylaxis and treatment plan.

5. Conclusions

Bronchial asthma patients affected by periodontitis have poorer oral hygiene than those with a healthy periodontium. Significantly deeper pockets in the lateral segments of the dentition were found in all study groups. In addition, significantly deeper gingival pockets were noted in the lateral sextants in the ASTHMA group than in the CONTROL group at all study periods. There was no significant worsening of periodontal status as assessed by CAL and PD in patients with bronchial asthma treated with inhaled GCS regardless of baseline periodontal status.

Supplementary Materials: The following are available online at https://www.mdpi.com/article/10.3390/app12010240/s1: Supplementary results—1, 2, 3. Tables S1–S10. Table S1 Comparison of examined parameters in groups—Baseline; Table S2 Comparison of examined parameters in groups—Examination after two week; Table S3 Comparison of the examined parameters in groups—after 3 months; Table S4 Comparison of the examined parameters in groups—after 6 months; Table S5 Intergroup comparison of differences between studies of selected variables (CAL, PD, PI, BOP)—for entire oral cavity; Table S6 Intergroup comparison of differences between studies of selected variables (CAL, PD, PI, BOP)—central segments; Table S7 Intergroup comparison of differences

between studies of selected variables (CAL, PD, PI, BOP)—lateral segments; Table S8 Comparison of differences between studies of levels of selected indicators between central and lateral sextants in group I (PERIO–ASTHMA); Table S9 Comparison of differences between studies of levels of selected indicators between central and lateral sextants in group II (ASTHMA); Table S10 Comparison of differences between studies of levels of selected indicators between central and lateral sextants in the control group; Table S11 Intergroup comparison of differences between clinical indices between lateral and medial segments.

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