The Prevalence of Periodontal-related Changes in Adolescents With Asthma: Results of the Third Annual National Health and Nutrition Examination Survey

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Abstract

Purpose: The purpose of this study was to explore the association between asthma and periodontal disease in adolescents using oral examination and health interview data from the Third National Health and Nutrition Examination Survey (NHANES III) 1988-1994.

Methods: The study population comprised 1,596 adolescents 13 to 17 years of age: 253 (16%) asthmatics and 1,358 (84%) nonasthmatic controls who were examined for bleeding on probing (BOP), subgingival calculus (SBC), supragingival calculus (SPC), probing depth greater than or equal to 3 mm (PD), and loss of periodontal attachment greater than or equal to 2 mm (LPA). The authors fitted separate multivariate GEE Poisson regression models adjusting for parents' income, gender, race, exposure to potentially xerogenic drugs (antihistamines, corticosteroids, and inhalers), tobacco exposure, and dental examination within the past year.

Results: None of the periodontal measures was associated with asthma severity or with the use of antiasthmatic drugs. However, several covariates had statistically significant odds ratios *(P*<.05).

Conclusions: There was no evidence to support the association between asthma and periodontal health in the adolescent population. Since the findings may be due to the inherent limitations of cross-sectional studies, the lack of knowledge about the daily dose of antiasthmatic medication, and the level of compliance with the therapeutic regimen, future studies should be longitudinal and monitor medication use. (*Pediatr Dent.* 2003;25:279-284)

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Asthma is a chronic inflammatory condition of the airways characterized by hyperresponsiveness and episodic, reversible symptoms of airflow obstruction.[1] The prevalence of asthma has been increasing since the 1980s across all age, gender, and racial groups and is higher among children than adults. The prevalence of asthma among US children less than 4 years of age increased by 260% from 1980 to 1994, and by 174% from 1980 to 1994 in children 5 to 14 years of age.[2]

Periodontal disease represents a reaction to bacterial plaque that causes chronic inflammation, gingival bleeding, increased pocket depths, and alveolar bone loss. Bacterial antigens trigger the immune response of the host, which eventually causes the effects of the disease.[3] As in asthma, the immune response is the mechanism involved in the pathogenesis and progression of the disease. Although most of the disease is associated with adults, a significant portion is seen in children and young adults.[3] Saliva exerts multiple actions related to the preservation of oral health.[4] These include antibacterial, antiviral, and antifungal actions. Saliva also buffers the fluid bathing the teeth and oral mucosa, and promotes mineralization of

enamel. Mucins in saliva protect the mucosa from damage. Immunoglobulin and growth factors are also found in saliva, although their exact functions are not clear at this time.[5-7]

Interaction between bacterial and immunological factors is thought to be a key factor in the destruction of periodontal tissue. Some of the above actions of saliva undoubtedly impact this interaction. At least 1 study suggests that salivary markers may provide a link to the severity of the disease.[8] Since many drugs modify salivary secretion in a significant percentage of the patients taking them, drugs used to treat asthma may negatively affect periodontal health.

Secretory IgA (sIgA) is actively secreted by the salivary glands and acts as the major specific immune defense mechanism in saliva. It is generally agreed that, by binding to soluble and particulate antigens, it acts as a first line defense of the mucosa.[9] Although the importance of IgA in periodontal disease is controversial, it exerts several biological actions that could impact this disease.[10] Stimulated whole and parotid saliva from patients with chronic periodontal disease was shown to contain elevated levels of sIgA, and the concentration of sIgA tended to be correlated with the severity of disease.[11,12] Östergaar[13] reported a reduction of sIgA levels in children with asthma. A direct correlation between asthma and sIgA levels was not established, and these children's asthma was precipitated by recurrent infection.[13] In contrast, Hyyppä found that the allergic immunoglobulin, IgE, which presumably diffuses passively into saliva, was elevated in patients with asthma as compared with healthy controls.[17]

Much of the drug-induced change in salivary function is predictable from the mechanism of action of the drug. The groups most often associated with dry mouth include drugs that interfere with autonomic control of the gland or with ion and water movement. More important, the xerogenic effect increases with the number of drugs taken. Except for antihistamines and anticholinergic agents, drugs used to treat asthma are, mechanistically, not associated with an inhibitory effect on salivary function.

Ryberg et al[14] found that asthmatic children exposed to daily inhalation of a $ß_2$-selective adrenergic bronchodilator (terbutaline or salbutarol) exhibited a decreased salivary flow rate (whole and parotid) and a diminished protein output when compared to healthy cohorts matched by age, sex, and socioeconomic status. Although salivary function of the asthmatic group was attenuated after at least 1 year on the drug, there were no significant differences in plaque or gingivitis between the 2 groups.

Six studies have examined the association between periodontal diseases and asthma. Laurikanen and Kuusisto,[15] McDerra,[16] and Hyyppä[17,18] reported that asthmatics had more gingivitis than controls, while Bjerkeborn et al[19] found no difference in gingivitis prevalence. McDerra[16] and Hyyppä[18] reported no difference in plaque. McDerra[16] and Wotman[20] reported that asthmatics had more calculus, while Hyyppä[17, 18] reported no difference in the amount of calculus. It is interesting to note that, when these studies are considered as a group, they fail to show consistent findings. This lack of consensus among studies may be attributed to differences in asthma severity, medication types,[21] lack of statistical power due to small samples, and the inherent limits of cross-sectional studies.[22] While evidence for the asthma-periodontal disease association is equivocal, it is nonetheless useful to consider possible mechanisms for such an association, if one does exist.

Interpretation of studies such as those discussed above is complicated by the fact that treatment of asthmatics involves the use of medications that affect inflammation and the immune response. An indirect factor could be the effects of some of the medications inducing dryness of the mouth. An association between asthma and periodontal disease might involve either pathological activation of the immune and inflammatory process,[23] antiasthmatic medications[24] or the interaction between them.

Other studies have demonstrated actions of antiasthma drugs that might potentially influence periodontal disease. Recently, inhaled corticosteroids have been associated with bone loss in asthmatic children.[25] Although this study examined overall bone mass, it raises the possibility that these drugs could impact bone loss in periodontal disease. Kargul et al measured the pH of saliva and interproximal plaque in asthmatic children using $ß_2$-adrenergic bronchodilator corticosteroid inhalers. Both saliva and plaque pH decreased over the 30-minute observation period.[26]

Given the large number of children and adolescents who suffer from asthma and the lack of consensus on the relationship between asthma and periodontal health reported, the authors approach the issue from a different direction than previous researchers using data from the Third National Health and Nutrition Survey (NHANES III) 1988-1994.[27] Thus, the purpose of this study was to determine if there is a significant difference between the periodontal health parameters in young asthmatics when compared to nonasthmatic controls. Further, the extent to which the use of selected antiasthmatic drugs is associated with periodontal health is examined.

Methods

The study population comprised 15% adolescents 13 to 17 years of age on whom all of the following components of a periodontal examination were performed:

1. bleeding on probing (BOP);
2. loss of periodontal attachment of 2 mm or more (LPA);
3. probing depth of 3 mm or more (PD);
4. the presence of subgingival calculus (SBC);
5. supragingival calculus (SPC).

The household youth questionnaire and examination files of NHANES III, which is a periodic survey conducted by National Center for Health Statistics, was used.[27] The data, gathered from 1988 through 1994, were based on a

complex, multistage sample plan and designed to provide national estimates of the health and nutritional status of the United States civilian, noninstitutionalized population aged 2 months and older. From 19,528 randomly selected households, 33,994 subjects were interviewed, 30,818 were examined in mobile examination centers, and 493 were examined at home. Examinations were performed by calibrated dentists and physicians, extensive health, social, and nutritional histories were obtained by interviewing the subjects or their parents, and blood specimens were drawn. A detailed discussion of the survey methods is presented by Brown et al.[28]

Asthmatic children were those whose parents reported physician-diagnosed asthma, asthma-related hospitalizations, acute outpatient visits, and episodes of wheezing in the previous 12 months. The authors used the approach of Halterman et al[29] to classify asthma presence and severity as:

1. severe=≥2 hospitalizations or ≥4 asthma-related acute visits;
2. moderate=≥1 hospitalization and ≥2 acute visits or ≥3 episodes of wheezing;
3. mild=no hospitalizations, ≥1 asthma-related acute visits, or ≥2 episodes of wheezing.

Subjects in the sample who met all of the criteria but did not have asthma served as nonasthmatic controls.

Periodontal measurements used were made at the mesiobuccal and midbuccal on a maximum of 14 permanent, fully erupted teeth (excluding third molars) in a randomly selected maxillary and mandibular quadrant. The distance from the cementoenamel junction to gingival crest was measured, as was the distance from the free gingival margin to the base of the sulcus. The difference between the 2 measurements represents loss of clinical periodontal attachment. Additionally, at each site, bleeding, subgingival calculus, and probing depths of 3 mm of more were categorized as being present or absent. These are standard indicators of periodontal health and have been used extensively.[30-32]

Prescribed asthma medications and the duration of use (of more than 30 days) were included for analysis. The authors reported on 3 types of drugs commonly used by asthmatics that have xerogenic or antinflammatory effects: (1) antiasthmatic inhalers, (2) antihistamines, and (3) corticosteroids. For subjects reporting the use of more than 1 drug type, the duration of use for each drug was added so that a subject who reported using an antihistamine for 30 months and an antiasthmatic inhaler for 24 months would have a total exposure time of 54 months.

Since dose and frequency were not reported, the authors explicitly assumed that the age-specific dose and frequency are equal for the 3 drug classes. The reported amount of time subjects used an antiasthmatic inhaler (a $ß_2$-adrenergic agonist), antihistamines, and corticosteroids was an independent variable. Other potential covariates were gender, age (to nearest year), race (white/nonwhite), and previous orthodontic treatment. To control for potentially differential access to care, the authors used family income (≥$20,000, <$20,000), and an indication by subjects of having a "dental visit in last year" (yes or no) obtained via interview. Exposure to tobacco was inferred if the subject reported smoking, living with a smoker, or had a serum cotinine level greater than 0.5 ng/mL, assayed by isotope dilution-liquid chromatography-tandem mass spectrometry.[33] This technique is highly specific and is capable of detecting levels as low as 0.030 ng/mL, allowing quantitative measurement of both low levels of tobacco-smoke exposure from environmental tobacco smoke and higher levels of exposure from active smoking.[33]

Since the NHANES III survey used multistage sampling, the authors used SAS-callable SUDAAN 8.0 to compute all descriptive statistics.[34] Spearman's correlation was used to explore the association between cumulative exposure to xerogenic drugs and gingival bleeding, calculus, probing depth, and loss of attachment. Kruskal-Wallis ANOVA was used to test differences in gingival bleeding, calculus, probing depth, and loss of attachment between moderate and severe asthmatic groups and healthy controls.

To adjust for clustering within the mouth, logistic generalized estimating equation (GEE) models (GENMOD, PC-SAS 8.1) with an exchangeable working correlation

Table 1. Distribution of Sample by Asthma Status, Race, Gender, Income, Annual Dental Visits, Tobacco Exposure, and History of Orthodontic Treatment

Asthma status	Race (N=1,596)		Gender (N=1,596)		Income* (N=1,580)		Age (N=1,596)				≥1 visit/year (N=1,578)*		Tobacco exposure* (N=1,595)		Orthodontic treatment* (N=1,595)	
	White	Non-white	Male	Female	<$20,000	≥$20,000	13-14	14-15	15-16	16-17	Yes	No	Yes	No	Yes	No
Controls	775	583	643	715	668	674	369	315	318	356	579	764	706	651	194	1,163
Mild	66	47	52	61	50	63	34	29	24	26	49	62	64	49	17	96
Moderate	78	35	45	68	38	75	35	24	27	27	51	61	54	59	20	93
Severe	7	5	7	5	6	6	1	4	3	4	4	8	7	5	3	9
Total	926	670	747	849	762	818	439	372	372	413	683	895	831	764	234	1,361

*Data missing for some subjects.

Table 2. Summary Statistics for Gingival Bleeding, Calculus, Probable Depth, and Loss of Attachment (LPA) by Disease Group

	N	Mean	SE	Design effect	Median	Min	Max	*P**
Bleeding sites								
Controls	1,250	2.9	0.2	5.1	2	0	21	
Mild asthma	100	2.6	0.3	1.2	2	0	15	
Moderate/severe asthma	118	3.6	0.8	4.0	2	0	15	.56
Supragingival calculus sites								
Controls	1,249	4.1	0.3	6.8	3	0	28	
Mild asthma	100	4.0	1.0	3.5	3	0	28	
Moderate/severe asthma	118	4.0	0.8	2.6	3	0	21	.96
Subgingival calculus sites								
Controls	1,249	0.1	0.2	5.9	0	0	24	
Mild asthma	100	0.6	0.2	1.4	0	0	14	
Moderate/severe asthma	118	1.4	3.4	2.7	0	0	18	.77
Sites with probing depth ≥3 mm								
Controls	1,249	2.1	0.2	6.9	1	0	18	
Mild asthma	100	2.1	0.5	3.1	1	0	17	
Moderate/severe asthma	117	2.0	3.1	1.2	1	0	15	.22
Sites with LPA ≥2 mm								
Controls	1,249	0.4	0.7	3.9	0	0	19	
Mild asthma	100	0.3	0.9	1.5	0	0	5	
Moderate/severe asthma	117	0.2	0.1	1.2	0	0	6	.10

**P* values are based on Kruskal-Wallis ANOVA.

Table 3. Studies of the Association between Asthma and Periodontal Disease

Author(s)	Year	Location	Age	Subjects	Design	Findings
Shulman et al	2003	USA	13-17	238 asthmatics	Population-based case control	No difference in gingivitis, probing depth, calculus, or loss of attachment
Laurikainen and Kuusisto	1998	Finland	25-40	33 asthmatics	Case control	Asthmatics had lower salivary pH and more gingival bleeding
McDerra	1998	USA	4-16	100 asthmatic patients at dental school clinic	Case control	Asthmatics had more gingivitis and calculus
Bjerkeborn et al	1987	Sweden	5-18	61 children with "extrinsic asthma" treated at university hospital	Case control	No difference in gingival bleeding
Hyyppä	1984	Finland	10-12	Patients with severe long-term asthma treated at pediatric clinic	Case control	Asthmatics had more gingivitis No difference in plaque or calculus
Hyyppä	1979	Finland	10-12	30 children with long-term asthma	Case control	No difference in amount of calculus More gingivitis
Wotman et al	1973	USA	4-15	25 asthmatic children	Case control	Asthmatics had more calculus

structure was used. The authors initially fitted separate fully saturated models for each dependent variable: bleeding on probing, loss of periodontal attachment, probing depth, and the presence of subgingival and supragingival as well as the previously described covariates. Using Wald chi-square tests, the authors sequentially eliminated the least significant covariates ($P>.10$). Interactions were tested and, if significant, were added to the model. In each model, the authors used odds ratios to measure the strength of association between the dependent variable, asthma severity, and the covariates (simultaneously adjusting for the presence of the other variables in the model), and the Wald chi-square was used to test the statistical significance ($P<.05$) of the odds ratios. An odds ratio (OR) of unity means that a covariate is not associated with the dependent (periodontal) variable.

Results

The examination, laboratory, and youth health interview data files were combined and the analysis was restricted to 1,596 adolescents 13 to 17 years of age who were categorized as having mild 113 (7%), moderate 113 (7%), and severe 12 (1%) asthma. Table 1 shows the distribution of asthma status, gender, income, race, last dental visit, and tobacco exposure. Since there were few severe asthmatics, the severe and moderate categories were combined for the multivariate analysis. None of the controls used antiasthmatic inhalers, steroids, or antihistamines for more than 30 days. Of the 238 asthmatics, 10 (4%) reported using antihistamines

more than 30 days, 31 (14%) reported using antiasthmatic inhalers more than 30 days, and only 1 (1%) reported using steroids more than 30 days. No significant positive association (Spearman's correlation) was found between cumulative exposure to these drugs and the prevalence of the previously mentioned variables *(P*>.10 for all correlations tested).

Table 2 shows summary statistics for gingival bleeding (BOP), SPC and SBC, PD, and LPA. Kruskal-Wallis ANOVA tests for differences between severe/moderate asthmatics and nonasthmatic controls showed no significant differences *(P*<.10 for all tests). Standard errors are adjusted for the design effect.

Separate GEE models were fitted for each of the 5 periodontal variables. Neither asthma nor cumulative exposure to antiasthmatic inhalers, corticosteroids, or antihistamines was significant in any of the models. No interactions were statistically significant. Females had a lower odds of BOP (OR=0.74; *P*<.0001) than males, subjects who had 1 or more dental visits in the past year had substantially lower odds of BOP (OR=0.30; *P*<.0001) than those with no dental visits, non-whites had lower odds of BOP (OR=0.80; *P*=.003) than whites, and subjects who were not exposed to tobacco had lower odds of BOP (OR=0.85; *P*=.030) than those with tobacco exposure.

Females had lower odds of having supragingival (OR=0.76; *P*<.0001) and subgingival (OR=0.67; *P*=.005) calculus than males, subjects without an annual dental visit had higher odds of having supragingival (OR=1.16; *P*<.0001) and subgingival calculus (OR=2.24; *P*<.0001) than subjects without an annual visit, and nonwhites had greater odds of having supragingival (OR=1.46; *P*<.0001) and subgingival calculus (OR=1.29; *P*=.030) than whites. Tobacco use was significant only for subgingival calculus with nonusers having significantly lower odds of having subgingival calculus (OR=0.63; *P*<.0001) than users.

Females had lower odds (OR=0.90; *P*=.0002) of having sites with a probing depth of ≥3 mm than males. Whites and subjects who reported no dental visit within the year had greater odds of sites with ≥3 mm probing depth (OR=1.14; *P*<.0001 and 1.15; *P*<.0001, respectively) than the reference groups. Subjects who reported no history of orthodontic treatment were less likely to have a probing depth ≥3 mm than those with past orthodontic treatment (OR=0.83; *P*<.0001). The only variable significantly associated with attachment loss ≥2 mm was not having regular dental visits (OR=1.09; *P*<.0001).

Discussion

Neither asthma nor the cumulative use of antiasthmatic drugs was significantly associated with periodontal health. Table 3 compares these results with those of previous studies. These results conflict with those of Hyyppä[17, 18] and McDerra[16] who found that asthmatic children (10-12 and 4-16 years of age, respectively) had more gingival inflammation than controls. Further, unlike the findings of this, McDerra[16] and Wotman et al[20] found that asthmatics 4 to 15 years of age had more calculus than controls. If there is, in fact, an association between asthma and periodontal disease that this study did not find, what factors might be responsible?

First, the subjects examined in NHANES III might have been taking lower doses of antiasthmatic drugs for shorter periods than those in other studies. For example, all the asthmatic children studied by McDerra[16] used an antiasthmatic inhaler. Halterman et al reviewed data from NHANES III and concluded, "Many children in the United States do not receive recommended maintenance medications."[29] Of the 238 asthmatics in this study sample, only 17% of severe, 4% of moderate, and 4% of mild asthmatics reported using antiasthmatic inhalers. This suggests the possibility that many of the subjects were undermedicated compared to contemporary clinical protocols or they were not taking the medications as instructed.[22]

Second, it is possible that the effects of asthma and antiasthmatic medication are different in adolescents than in children. Since periodontal measurements were not taken on children less than 13 years of age, the sample (13-17 years) might be beyond the age at which their periodontal health is affected by the medications or the asthma, per se. Finally, differences in periodontal health may be masked by hormonal changes in adolescence associated with puberty.

Conclusions

These results do not support an asthma-periodontal health association. If one exists, it is weak and of little clinical significance.

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