Dental Health in Asthmatic Children: A South Italy Study

Gianmaria Fabrizio Ferrazzano, DDS, PhD
Giancarla Sangianantoni, DDS, PhD *Tiziana Cantile, DDS, PhD*
Ivana Amato, DDS, PhD *Aniello Ingenito, MD*
Paolo Noschese, MD

ABSTRACT

Purpose: The purpose of this study was to examine the oral health status of children with mild intermittent or mild persistent asthma compared to healthy children.
Methods: A group of 124 asthmatic children was compared to 156 age-matched healthy children. Bitewing radiographs were taken and clinical examinations were carried out to record caries prevalence, caries experience in both dentitions, periodontal health and dental enamel defects.
Results: Caries prevalence for asthmatic patients was 39% in the permanent dentition and 32% in the primary dentition. Healthy subjects presented 36% caries prevalence in the permanent dentition and 26% in the primary dentition. There was no statistically significant difference between the 2 groups in caries experience. The differences between asthmatic subjects and healthy subjects for periodontal health and enamel defects were not statistically significant either.
Conclusions: Children affected by mild intermittent or mild persistent asthma do not seem to be more susceptible to oral diseases than healthy children. (J Dent Child 2012;79(3):170-5)
Received June 15, 2011; Last Revision September 6, 2011; Revision Accepted October 26, 2011.

KEYWORDS: DENTAL CARIES, ASTHMA, DENTAL ENAMEL HYPOPLASIA, PEDIATRIC DENTISTRY

Asthma is a chronic disease characterized by airway inflammation and bronchoconstriction. It is a serious global health problem affecting more than 100 million people worldwide and, in most countries, its prevalence has increased over the past 2 decades. Although it affects people of all ages, most cases of asthma occur in childhood, with a peak prevalence between 6 and 11 years old. [1]

This disease is one of the main causes of school absenteeism. In Italy, approximately 10% in children and adolescents are affected.[1]

In spite of the increasing prevalence of asthma, only a few studies have investigated oral health in asthmatic children over the last 20 years, and the results are controversial. Some authors have reported a correlation between childhood asthma and dental caries in children, suggesting that asthmatic children are at higher risk for oral diseases than non-asthmatic children due to either their disease and/or their pharmacotherapy.[2-8] Other authors, however, have found no such connection.[9-11]

Guidelines for the diagnosis and management of asthma developed by the National Institutes of Health (NIH) include a system of classification of asthma severity.[12] This system uses 3 variables (frequency of daytime symptoms, frequency of night-time symptoms, and lung function) to define 4 categories of asthma: mild intermittent asthma and 3 forms of persistent asthma (mild, moderate, and severe; Table 1). Fortunately, most asthmatic children have mild intermittent or mild persistent asthma.[12]

The purpose of this study was to investigate caries prevalence, caries experience, presence of periodontal

Drs. Ferrazzano, Sangianantoni, Cantile and Amato are staff dentists, and Dr. Ingenito is Professor, all in the Department of Pediatric Dentistry, School of Dentistry, University of Naples Federico II, Naples, Italy; and Dr. Noschese is staff medical doctor, Department of Ambulatory Pediatric Pulmonology, A. Cardarelli Hospital, Naples, Italy.
Correspond with Dr. Ferrazzano at gianmariafabrizio@yahoo.it

disease, and dental enamel defects in children with mild intermittent and mild persistent asthma.

METHODS

PARTICIPANTS

Asthmatic subjects were invited to participate in the Department of Pulmonology at the A. Cardarelli Hospital in Naples, Italy. Healthy children were randomly selected from 10 public schools within Italy's Regional Campanian district. The subjects' parents signed informed consent for participation after receiving verbal and written explanations about the study, which was approved by the ethics committee of the hospital (No. 1127/2009).

All subjects were examined by 3 pediatricians and classified according to the American Society of Anesthesiologists (**ASA**) and to guidelines for the diagnosis and management of asthma developed by the NIH and by the National Asthma Education and Prevention Program.[12] In order to participate, study children had to have a diagnosis of mild intermittent or mild persistent asthma, with or without a history of dental care. For the control group, subjects had to be from single, full-term births, in good health (ASA I), and without a history of dental care.

Exclusion criteria for both groups included a history of preterm birth, malnutrition or neonatal hypocalcemia, trauma to the primary dentition, fixed orthodontic appliances, and exposure to fluoridated water and fluoride supplements (use of fluoridated toothpaste was acceptable).

ORAL EXAMINATION

All subjects were examined in the hospital dental clinic by 3 calibrated dental professionals who used the same dental unit. A mouth mirror and the World Health Organization (**WHO**) community periodontal index (**CPI**) ballpoint probe, with air drying when necessary, were used.

The presence of dental caries was assessed by systematic evaluation of each child's caries experience using the decayed, missing, and filled teeth index for the permanent dentition (**DMFT**) and primary dentition (**dmft**). Two bitewing radiographs were taken according to European Academy of Paediatric Dentistry guidelines for use of radiographs in children.[13] A thyroid collar and a lead apron were used for children and for any

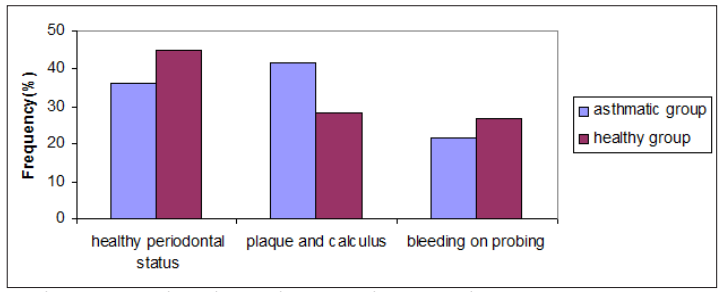

Figure 1. Community periodontal index values in asthma patients and healthy controls.

*Table 1. Classification of asthma**

Classification‡	Medication(s) used	Frequency	Clinical features before†
Mild intermittent	Inhaled ß2 agonist or ipratropium bromide, but not >3x/week (both used as need)	Symptoms ≤2 times per week Brief exacerbations (from a few hours to a few days) Night-time asthma symptoms <2x/month Asymptomatic and normal lung function between exacerbations	FEV_1, /PEV≥80% of predicted value PEF variability <20%
Mild persistent	Inhaled corcticosteroids or cromolym or inhaled ß2 agonist or ipratropium bromide or ß2 agonist tablets or syrup (all used daily)	Symptoms >2x/week but <1x/day Exacerbations that may affect activity Night-time asthma symptoms >2x/month	FEV_1/PEF≥80% of predicted values PEF variability 20-30%
Moderate persistent	Inhaled cortico steroids or inhaled ß2 agonist or ipratropium bromide or ß2 agonist tablets or syrup (all used daily)	Daily symptoms Exacerbations that affect activity Night-time asthma symptoms >1x/week Daily use of inhaled, short-acting ß2 agonist	FEV_1/PEF≥60% to <80% of predicted values PEF variability >30%
Severe persistent	Inhaled cortico steroids or inhaled ß2 agonist or ipratropium bromide or ß2 agonist tablets or syrup and/or oral corticosteroids (all used daily)	Continuous symptoms Frequent exacerbations Frequent night-time symptoms Limited physical activity	FEV_1/PEF≥60% of predicted values PEF variability >30%

**Sources: National Heart, Lung, and Blood Institute.[12]*

†*FEV= forced expiratory volume in 1 second; PEF=peak expiratory flow.*

‡ *The presence of one of the features of severity is sufficient to place a patient in that category. A person should be assigned to the most severe grade in which any feature occurs. A person's classification may change over time.*

assistant during X-ray exposures. A pointing cone and dental films (Kodak Ultraspeed, speed group D, Eastman Kodak Company, Rochester, N.Y.) were placed using a pediatric radiograph positioner. The CPI was recorded on the buccal and lingual/palatal surfaces of right maxillary central incisor, right maxillary first molar, left maxillary first molar, left mandibular central incisor, left mandibular first molar, right mandibular first molar, respectively, following WHO indications.[14] Enamel defects were assessed using the modified developmental defects of enamel (**DDE**) index in which the type (opacitity, hypoplasia, discoloration), number (single and multiple), demarcation (demarcated and diffuse), and location of defects on the buccal and lingual surfaces of teeth were recorded.[15] Additionally, all subjects received scaling and rubber-cup prophylaxis to remove extrinsic stains and debris prior to the oral examination.

INTEREXAMINER CALIBRATION

Before beginning the study, the examiners were calibrated for each index recorded. The kappa statistic was used to compare each of the 3 examiners to 1 examiner used as a gold standard in relation to the DMFT index measured

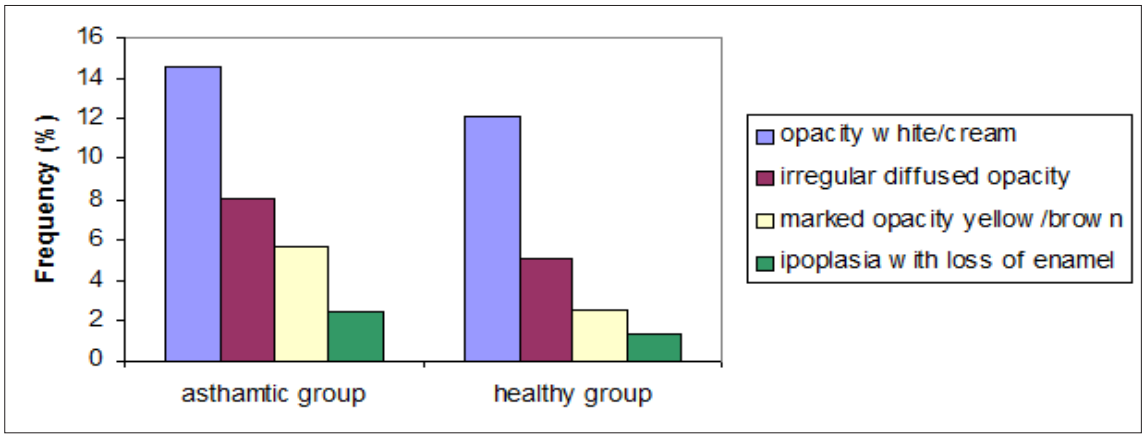

Figure 2. Distribution of enamel dental defects.

Table 2. DMFT Index and the Distribution of its Components for Healthy and Asthmatic Group*

		N	Mean	Std. Deviation	95% Confidence Interval for Mean		F	Sig.
					Lower Bound	Upper Bound		
DMFT	healthy group	156	1.03	1.60	.78	1.29	1.137	.287
	asthmatic	124	1.25	1.82	.93	1.57		
	group total	280	1.13	1.70	.93	1.33		
Decayed teeth	healthy group	156	.67	1.20	.48	.86	.066	.798
	asthmatic	124	.71	1.17	.50	.92		
	group total	280	.69	1.19	.55	.83		
Missing teeth	healthy group	156	.07	.43	.00	.14	.427	.514
	asthmatic	124	.71	.71	.02	.10		
	group total	280	.69	.6	.01	.10		
Filled teeth	healthy group	156	.32	.96	.17	.47	1.519	.219
	asthmatic	124	.48	1.15	.27	.68		
	group total	280	.39	.,05	.27	.51	0	5

**DMFT=decayed, missing, and filled teeth for permanent dentition.*

on 40 teeth for each examiner, CPI index measured on 30 molars and 30 incisors for each examiner, and DDE index measured on 40 teeth. Then, each examiner was compared against the other 2.

STATISTICAL ANALYSIS

Statistical analysis was performed using Statistical Package for the Social Sciences 10.0 software (SPSS Inc., Chicago, Ill., USA), with a confidence interval of 95% and a significance level of 5% ($P<.05$). The comparison of mean DMFT/dmft among groups was conducted using 1-way analysis of variance. The comparison of CPI treatment needs and DDE scores between the 2 groups was done using logistic regression analysis.

RESULTS

INTEREXAMINER CALIBRATION

The kappa-tests, comparing DMFT measured by each examiner to DMFT measured by an examiner used as a gold standard, yielded scores of 0.89, 0.78, and 0.82, respectively. For CPI, the kappa score was 0.96, 0.87, and 0.90 when comparing the gold standard examiner and examiners 1, 2, and 3, respectively; for the DDE index, the kappa value was 0.87, 0.77, and 0.83, respectively.

For the DMFT/dmft index, the kappa was 0.85 when comparing examiners 1 and 2, 0.78 when comparing examiners 2 and 3, and 0.81 when comparing examiners 1 and 3. For the CPI, there was perfect concordance between examiners, with a kappa of 1. The kappa score for the DDE index was 0.77 when comparing examiners 1 and 2, 0.74 when comparing examiners 2 and 3, and 0.83 when comparing examiners 1 and 3.

The analyzed sample consisted of 156 healthy subjects (87 males and 69 females), with a mean of age 8.78±1.43 years and 124 asthmatic subjects (68 males and 56 females) with mild intermittent form and mild persistent form (mean age=9.11±1.53 years). Caries prevalence in the permanent dentition was 39% in the asthmatic group and 36% in the control group, while it was 32% and 26% for the primary dentition in the asthmatic and control groups, respectively. In asthmatic children, the DMFT mean value was 1.25±1.82, with maximum and minimum values of 6 and 0, respectively. In healthy subjects, the DMFT mean value was 1.03±1.60, with maximum and minimum values of 6 and 0, respectively. The difference in the mean DMFT between the 2 groups was not statistically significant ($P<.29$). Table 2 shows the DMFT index and the distribution of its components. There was no statistically significant difference between asthmatic and healthy subjects for the decayed teeth ($P<.80$), missing teeth ($P>.51$), and filled teeth ($P<.22$).

In the asthmatic group, the dmft mean value was 1.10±1.89. In healthy subjects, the dmft mean value was 0.90±1.72. The difference in the mean dmft between the 2 groups was not statistically significant ($P<.36$). There was no statistically significant difference between the 2 groups in any dmft components (Table 3).

Table 3. dmft* Index and the Distribution of its Components for Healthy and Asthmatic Group

		N	Mean	Std. Deviation	95% Confidence Interval for Mean		F	Sig.
					Lower Bound	Upper Bound		
dmft	healthy group	156	.90	1.72	.62	1.17	.847	.358
	asthmatic	124	1.10	1.89	.76	1.43		
	group total	280	.99	1.80	.77	1.20		
decayed teeth	healthy group	155	.49	1.16	.31	.68	.170	.680
	asthmatic	124	.55	1.17	.34	.76		
	group total	279	.52	1.17	.38	.65		
missing teeth	healthy group	156	.24	.89	.10	.38	.790	.375
	asthmatic	124	.35	1.05	.16	.53		
	group total	280	.29	.96	.18	.40		
filled teeth	healthy group	156	.18	.69	.07	.29	.050	.824
	asthmatic	124	.16	.67	.04	.28		
	group total	280	.17	.68	.09	.25	0	5

** dmft=decayed, missing, and filled teeth for primary dentition.*

CPI data revealed that approximately 36% of the asthmatic subjects and 45% of control subjects had a healthy periodontal tissues. Approximately 42% of the asthmatic children and 28% of the control subjects had plaque and calculus, while approximately 22% of the asthmatic patients and 27% of the control subjects showed bleeding on probing (Figure 1). Logistic regression analysis showed that there was no statistically significant difference between asthmatic subjects and healthy subjects for CPI values.

Approximately 31% of asthmatic subjects had enamel dental defects vs 21% of healthy children. Fifteen percent of the asthmatic children had a marked opacity of white/cream color, 8% had irregular diffused opacity, 6% had marked opacity of yellow/brown color, and 2% had hypoplasia with loss of enamel (Figure 2). In the control group, marked opacity white/cream was recorded in approximately 12%, irregular diffused opacity in 5%, marked opacity yellow/brown in 3%, and hypoplasia with loss of enamel in 1% (Figure 2). Although the defects were more prevalent in the asthmatic patients, logistic regression analysis showed that there was no statistically significant difference between the 2 groups for DDE values.

DISCUSSION

The findings of this study showed that children suffering from mild intermittent or mild persistent asthma, despite their disease status and pharmacotherapy, did not appear to have a higher caries experience when compared with the healthy subjects. In fact, DMFT and dmft mean values were similar in both groups. This is in agreement with previous studies, which did not show a correlation between bronchial asthma and dental caries, supporting the hypothesis that the disease did not constitute a risk factor for tooth decay.[9,10] However, several studies suggested that people with asthma had a greater rate of caries development than their nonasthmatic counterparts.[2] This phenomenon was attributed to prolonged use of ß2 agonists, which was associated with diminished salivary production and secretion.[16-18]

It is possible that the discordant data found in the literature were due to the difficulty in examining the relationship between the disease condition and caries prevalence. In fact, asthma severity and medications used in its treatment often change over time. Furthermore, the onset of asthma may be difficult to assess. Many studies were unable to demonstrate any connection between asthma severity, period of exposure to medication, and caries prevalence. In fact, one of the limitations of these studies was an unequal distribution of patients according to the disease severity.[3,4] Therefore, to decrease this problem in our population, only patients with mild (66 patients) and moderate (58 patients) asthma were selected for this study.

In the present study, a higher prevalence of gingivitis was recorded in the asthmatic group; however, the difference between the 2 groups was not statistically significant. Gingivitis in asthmatic children could be due to several factors. Firstly, it could be the result of an altered immune response. In fact, the concentration of immunoglobulin E in gingival tissue was found to be elevated in asthmatic patients, which caused periodontal destruction.[19]Additionally, an enzyme group involved in inflammation—the arginine aminopeptidases—was found to be slightly elevated in the gingival fluid of asthmatic children, indicating that gingival inflammation was increased in asthma.[19,20] Moreover, the tendency to breathe through the mouth could cause the dehydration of alveolar mucosa, resulting in a worsening of the oral condition.[19] Finally, the use of inhaled steroids has been linked to increased levels of gingivitis.[21] Furthermore, a higher amount of calculus deposition and plaque wasn't recorded in asthmatic children compared to the control group, disagreeing with other authors who found a higher prevalence of calculus in asthmatic children due to increased levels of calcium and phosphorous in submaxillary saliva and parotid saliva.[22] It has been suggested that respiratory disorders were associated with DDE.[23] Few studies presented epidemiological data regarding DDE prevalence in children with asthma, and there is no evidence as to the potential role that pharmacological treatment plays in this association. The prevalence of enamel dental defects in this study was higher in the asthmatic group than the control group but not statistically significant. In both groups, the type of DDE recorded was the same (Figure 2), which is in agreement with other studies. [24,25] It has been hypothesized that, since ameloblasts are highly sensitive to oxygen supply, pediatric asthma patients with DDE have probably experienced previous episodes of oxygen deprivation.[23-27] Therefore, these defects might be related to the disease itself rather than to its treatment. However, our patients presented either mild intermittent or mild persistent asthma, which may have led to little oxygen deprivation, explaining why a positive correlation between asthma and DDE was not found in this study.

CONCLUSIONS

Based on this study, the following conclusions can be made:

1. Children affected by mild intermittent or mild persistent asthma do not seem to be more susceptible to caries than healthy children.
2. These subjects are similar to healthy subjects for periodontal health status and the presence of dental enamel defects.

REFERENCES

1. Tessari R, Migliore E, Balzi D, et al. Asthma prevalence estimated using a standard algorithm based on electronic health data in various areas of Italy. Epidemiol Prev 2008;32:56-65.
2. Arnrup K, Lundin SA, Dahllöf G. Analysis of paediatric dental services provided at a regional hospital

in Sweden: Dental treatment need in medically compromised children referred for dental consultation. Swed Dent J 1993;17:255-9.
3. Stensson M, Wendt LK, Koch G, Oldaeus G, Birkhed D. Oral health in preschool children with asthma. Int J Paediatr Dent 2008;18:243-50.
4. Ersin N, Gülen F, Eronat N, et al. Oral and dental manifestations of young asthmatics related to medication, severity and duration of condition. Int J Pediatr 2006;48:549-54.
5. McDerra EJ, Pollard MA, Curzon ME. The dental status of asthmatic British school children. Pediatr Dent 1998;20:281-7.
6. Milano M. Increased risk for dental caries in asthmatic children. Tex Dent J 1999;116:35-42.
7. Milano M, Lee JY, Donovan K, Chen JW. A cross-sectional study of medication-related factors and caries experience in asthmatic children. Pediatr Dent 2006;28:415-9.
8. Reddy DK, Hegde AM, Munshi AK. Dental caries status of children with bronchial asthma. J Clin Pediatr Dent 2003;27:293-5.
9. Meldrum AM, Thomson WM, Drummond BK, Sears MR. Is asthma a risk factor for dental caries? Findings from a cohort study. Caries Res 2001;35: 235-9.
10. Shulman JD, Taylor SE, Nunn ME. The association between asthma and dental caries in children and adolescents: A population-based case-control study. Caries Res 2001;35:240-6.
11. Bjerkeborn K, Dahllöf G, Hedlin G, Lindell M, Modéer T. Effect of disease severity and pharmacotherapy of asthma on oral health in asthmatic child-ren. Scand J Dent Res 1987;95:159-64.
12. Stoloff S. NIH News: update from the National Asthma Education and Prevention Program 2002. Manag Care 2003;12(1 Suppl):5-10.
13. Espelid I, Mejàre I, Weerheijm K. EAPD guidelines for use of radiographs in children. Eur J Paediatr Dent 2003;4:40-8.
14. Ainamo J, Barmes D, Beagrie G, Cutress T, Martin J, Sardo-Infirri J. Development of the World Health Organization community periodontal index of treatment needs. Int Dent J 1982;32:281-91.
15. Clarkson J, O'Mullane D. A modified index for use in epidemiological studies of enamel defects. J Dent Res 1989;68:445-50.
16. Shashikiran ND, Reddy V, Raju PK. Effect of antiasthmatic medication on dental disease: Dental caries and periodontal disease. J Indian Soc Pedod Prev Dent 2007;25:65-8.
17. Ryberg M, Moller C, Ericson T. Saliva composition and caries development in asthmatic patients treated with ß2-adrenoceptor agonists: A 4-year follow-up study. Scand J Dent Res 1991;99:212-8.
18. Maguire A, Rugg-Gunn AJ, Butler TJ. Dental health of children taking antimicrobial and non-antimicrobial liquid oral medication long term. Caries Res 1996;30:16-21.
19. Hyppa T. Gingival IgE and histamine concentrations in patients with asthma and in patients with periodontitis. J Clin Periodontol 1984;11:132-7.
20. Hyppa T. Studies of immunologic and inflammatory factors in saliva in patients with asthma and in patients with periodontitis. J Clin Periodontol 1981;8: 500-7.
21. McDerra EJ, Bjerkeborn K, Dahllof G, Hedlin G, Lindell M, Modeer T. Effect of disease severity and pharmacotherapy of asthma on oral health in asthmatic children. Scand J Dent Res 1988;22:297-301.
22. Mandel ID, Eriv A, Kutscher A, et al. Calcium and phosphorus levels in submaxillary saliva: Changes in cystic fibrosis and in asthma. Clin Pediatr (Phila) 1969;8:161-4.
23. Kellerhoff NM, Lussi A. Molar-incisor hypomineralization. Schweiz Monatsschr Zahnmed 2004;114: 243-53.
24. Guergolette RP, Dezan CC, Frossard WT, Ferreira FB, Cerci Neto A, Fernandes KB. Prevalence of developmental defects of enamel in children and adolescents with asthma. J Bras Pneumol 2009;35:295-300.
25. Van Amerongen WE, Kreulen CM. Cheese molars: A pilot study of the etiology of hypocalcifications in first permanent molars. J Dent Child 1995;62: 266-9.
26. Narang A, Maguire A, Nunn JH, Bush A. Oral health and related factors in cystic fibrosis and other chronic respiratory disorders. Arch Dis Child 2003;88:702-7.
27. Jälevik B. Enamel hypomineralization of permanent first molars: A clinical histomorphological and biochemical study. Swed Dent J 2001;149: 1-86.