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Original Article

Evaluation of the Oral Health Status of Children with Asthma: A Cross-Sectional Study

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Abstract

Background: The asthma–caries relationship is inconsistent. We compared oral health in children with and without asthma.

Methods: This study included 33 children with asthma and 33 age-matched healthy controls (aged 5–11 years). A trained dental student collected data on oral hygiene, mouth breathing, parental education/occupation, and body mass index (BMI) from participants via a questionnaire. Asthma control was evaluated with the Childhood Asthma Control Test questionnaire (C-ACT; validated for ages 4–11), administered to the asthma group. The dental student then performed oral examinations using the World Health Organization (WHO) criteria (dmft for primary teeth $\leq$ 9 y; DMFT for permanent teeth > 9 y). A generalized linear model was used to assess the association between asthma and overall DMFT.

Results: The groups had identical mean age (7.82 ± 1.76 years) and similar sex distributions (asthma: 75.8% male; control: 81.8% male). Uncontrolled asthma (C-ACT < 19) affected 93.9% of patients. Mouth breathing was more prevalent among those with asthma (57.6%, $\varphi = 0.41$, $P = 0.001$). Unadjusted DMFT was higher in asthma (4.72 ± 3.28 vs.0.73 ± 1.01; Mann-Whitney test, $r = 0.64$, $P < 0.001$). After adjustment, asthma remained independently associated with a 3.62-point higher overall DMFT (95% $CI = 2.00$ to 5.24, $P < 0.001$); significant covariates were mother's education, materia alba and dental plaque, and BMI ($P < 0.05$). A control outlier (DMFT = 4) did not influence the findings. C-ACT score did not correlate with DMFT ($\rho = 0.20$, $P = 0.261$), and DMFT did not differ across medication categories ($P > 0.05$).

Conclusion: Asthma was independently associated with higher dental caries, possibly mediated by mouth breathing, but causality remains uncertain. Non-significant associations between severity and medication, as well as unmeasured salivary factors, warrant caution. Longitudinal studies are needed.

Keywords: Asthma, Child, Dental caries, DMFT, Oral hygiene

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Introduction

Asthma is a chronic inflammatory disorder that primarily affects small airways. It is characterized by reversible airway narrowing, mucus hypersecretion, and, in certain cases, irreversible airway remodeling due to fibrosis. The disease typically presents with episodic exacerbations interspersed with asymptomatic periods.[1] In 2021, over 260 million individuals worldwide were affected by asthma, with estimates predicting an increase to 275 million by 2050.[2] The prevalence of asthma is notably higher among preschool-aged children compared to adults, affecting approximately one in ten children.[3] Prior to puberty, the condition is nearly twice as prevalent in boys compared to girls, while after puberty, the prevalence between sexes tends to equalize.[4]

Dental caries represents another highly prevalent chronic condition in childhood.[5] Caries involving the primary dentition continues to impose a substantial global health burden among pediatric populations; the number of affected children increased from 49.3 million in 1990 to 52.5 million in 2021.[5] Epidemiological studies indicate that the risk of dental caries in both primary and permanent teeth is 1.5-2 times greater in asthmatic children compared to their healthy counterparts.[6] Nevertheless, the association between asthma and dental caries remains contentious. While some investigations have reported a significantly higher caries prevalence among children with asthma—suggesting asthma as a risk factor [7,8]—other studies have not found significant differences between asthmatic and non-asthmatic children.[9,10] This inconsistency is further reflected in recent longitudinal research. For example, a birth-cohort study observed no direct or indirect causal relationships between asthma symptoms and the development of primary tooth caries,[11] whereas a sibling-controlled design demonstrated accelerated caries progression in asthmatic children over three years.[12] In adults, an analysis of the Azar cohort found no association between asthma and overall DMFT (Decayed, Missing, and Filled Teeth index) or the missing (M) component; however, the decayed (D) component was significantly high in individuals with asthma.[13] These conflicting findings, together with the high prevalence of both conditions, highlight the necessity for further research—especially among pediatric populations.

Multiple mechanisms have been postulated to account for a possible association between asthma and dental caries. Mouth breathing is more common in asthmatic individuals than in healthy controls,[14] primarily because this population has a higher prevalence of chronic rhinitis.[15] A recent study reported a mouth-breathing prevalence of 65.9% in school-aged children with asthma.[16] Mouth breathing has been associated with adverse effects on periodontal health.[17] Pharmacological interventions for asthma, particularly inhaled medications, often cause xerostomia, thereby increasing susceptibility to dental caries.[3] Corticosteroid therapy can further diminish salivary flow and increase the risk of oral fungal infections, potentially impeding effective oral hygiene practices.[3] Repeated use of medications, increased severity of asthma, and polypharmacy are all factors capable of significantly altering the composition of saliva.[18] Recent reviews underscore that inhaler use can reduce salivary flow, lower oral pH, and promote plaque accumulation, collectively fostering an environment conducive to dental caries, dental erosion, and periodontal disease.[19] Moreover, improper inhaler technique can increase retention of drug particles in the oral cavity, thereby exacerbating the risk of dental caries,[18] whereas appropriate training in inhaler use may mitigate this risk.[20]

Although these biological alterations are well documented, prospective evidence directly linking them to subsequent changes in DMFT remains limited. Berce et al.[21] and Vitale et al. [22] identified reduced salivary flow, decreased oral pH, increased plaque accumulation, and shifts in the oral microbiota among asthmatic children; however, the prospective examination of these variables in relation to later DMFT changes is infrequent. Consequently, the extent to which these biological factors mediate the relationship between asthma and dental caries remains unclear.

Given the substantial and rising prevalence of both asthma and pediatric dental caries, the inconsistent findings in the literature, and ongoing uncertainty about underlying mechanisms, this study assessed the oral and dental health status of children with asthma to identify the factors influencing these outcomes.

Materials and Methods

Study design and participants

This cross-sectional investigation was performed between January 2023 and May 2023. The asthma group consisted of 5–11-year-old children who visited the dental clinic of a university-affiliated hospital, with parents self-reporting that their child was an asthmatic patient receiving treatment from a pediatric asthma and allergy specialist. The control group comprised healthy children aged 5–11 years, recruited from two schools in the same city, after numerous schools were requested and the two that consented to participate were selected. The controls were matched by age with the asthma group. The inclusion criteria for both groups were ages 5-11 years, normal mental and intellectual health, absence of other acute or chronic systemic disorders, and willingness to cooperate during an oral examination. Exclusion criteria included long-term antibiotic use (≥ 4 weeks), a history of other respiratory disorders requiring treatment, and unwillingness to cooperate. Eligible asthmatic children were recruited until the required sample size was reached; control children were recruited after data collection for the asthma group was complete. All evaluations for the asthma group were conducted at the dental clinic, whereas those for the control group took place at the schools.

Ethical considerations

The Ethics Committee of Kerman University of Medical Sciences approved the study (approval no. IR.KMU.REC.1401.426). All procedures complied with the Declaration of Helsinki. Principles of honesty and trustworthiness were followed during document review. The research aligned with community beliefs and customs and did not interfere with the regular patient treatment protocol. Informed written consent was obtained from parents, and verbal consent was obtained from all participating children.

Sample size

The necessary sample size was determined using Stata/MP version 17.0 (Stata Corp. LLC, College Station, TX, USA) according to the DMFT values documented by Ghasempour et al. (23). In that study, the mean (± SD) DMFT was 2.27 ± 2.65 for asthmatic children and 0.80 ± 1.41 for healthy children. With a two-sided $\alpha = 0.05$, power = 0.80, and a 1:1 allocation ratio, a minimum of 33 individuals per group was necessary.

Data collection

Data were collected using a standardized questionnaire and a clinical oral examination, both conducted by a single qualified dental student. Assessments for the asthma group were conducted at the university dentistry clinic, while those for the control group occurred at the schools. The identical questionnaire was administered to both groups and contained the following shared items, completed in the presence of both the parent and child:

Demographics and socioeconomic status: age, sex, body mass index (BMI, kg/m²), parental education (less than bachelor's, bachelor's, graduate degree), and occupation (employee vs. other; housekeeper vs. employed).

Oral hygiene behaviors: toothbrushing frequency (≤ 3 times/week vs. ≥ 1 time/day), dental floss use (never vs. ≥ 1 time/week), mouthwash use (never vs. ≥ 1 time/week), and pattern of dental visits (regular, symptomatic only, never).

Mouth breathing: This was evaluated using a binary yes/no question asked of the parent: "Does your child exhibit mouth breathing characterized by nocturnal snoring, sleeping with an open mouth, intermittent gagging, continuous runny nose, or drooling?"

Only for the asthma group, the questionnaire covered the additional medical history items:

Asthma-specific information: duration of asthma, utilization of anti-asthmatic medications (anticholinergics, beta-agonists, inhaled glucocorticoids, oral glucocorticoids), mouth rinsing after inhaler use, history of gastroesophageal reflux, adenoid hypertrophy, and allergic rhinitis (characterized by a history of seasonal sensitivity).

Asthma control assessment

The Childhood Asthma Control Test (C-ACT), a validated 7-item questionnaire for children aged 4–11 years, originally described by Liu et al.,[24] was used to assess asthma control. In our study, we used the Persian version of the C-ACT, previously validated by Kashaninia et al. [25] in Iranian asthmatic children, which demonstrated validity with a Cronbach's α of 0.63. During the dental appointment, both the parent and child completed the questionnaire. The child responded to items 1–4 on a 0–3 scale, while the parent responded to items 5–7 on a 0–5 scale, yielding a cumulative C-ACT score range of 0-27. C-ACT scores < 19 indicate uncontrolled asthma, whereas scores of ≥ 19 indicate controlled asthma.

Oral examination

During the dental examination, the dentist informed the children's parents of the examination's objective. The dentist conducted the oral examination with the child positioned in a dental chair under appropriate artificial lighting. No radiographs were obtained. The assessment was performed visually and by tactile examination with a dental probe and mirror, in accordance with the World Health Organization (WHO) 2013 guidelines.[26] Caries experience was documented as follows: for children ≤ 9 years, the dmft index (decayed, missing due to caries, and filled primary teeth) was used; for children aged > 9 years, the DMFT index (decayed, missing, and filled permanent teeth) was used. Both indices were combined into a single overall DMFT value for analysis. The examiner recorded the presence or absence of gingival inflammation (any redness or bleeding on gentle probing) and the presence of materia alba and dental plaque (detectable without disclosing agents) as binary (yes/no) clinical oral findings.

A single trained dental student conducted all oral examinations under the supervision of a pediatric dentist. The examiner was calibrated against the supervisor on the WHO criteria before data collection; however, formal intra-examiner reliability testing (e.g., kappa statistic) was not performed.

Statistical analysis

Data were analyzed using SPSS 27 (IBM Corp., Armonk, NY, USA). Categorical variables were summarized as n (%), and continuous variables as mean ± SD or median (IQR), depending on normality (Kolmogorov-Smirnov test). Groups were compared with independent t-tests, Mann-Whitney U-tests, or χ^2 tests as appropriate. Effect sizes (Cohen's d, $r = |z|/\sqrt{N}$, φ or Cramér's V) were reported alongside the test statistics. For Cohen's d, values of 0.2, 0.5, and 0.8 were considered small, medium, and large, respectively. For r, φ, and Cramér's V, benchmarks of 0.1, 0.3, and 0.5 were used for small, medium, and large effects. The independent asthma–DMFT association was evaluated with a generalized linear model (normal distribution, identity link, robust standard errors) that included group, sex, father's job, mother's job, father's education, mother's education, brushing frequency, floss use, mouthwash use, dental visit pattern, gingival inflammation, materia alba/dental plaque, age, and BMI. Partial η^2 (0.01/0.06/0.14 = small/medium/large effect) was computed from Wald χ^2. Model fit was assessed by the likelihood-ratio χ^2, deviance-based R^2, AIC, and BIC; adjusted means were obtained from estimated marginal means. Spearman's correlation was used to test the C-ACT–DMFT correlation in the asthma group. A pre-planned sensitivity analysis excluded any box-plot outliers and re-ran the primary model. Significance was set at $P < 0.05$ (two-tailed) for all tests.

Results

Study population

Sixty-six children (33 with asthma and 33 healthy controls) were recruited for the study. The groups were well matched for age (mean: 7.82 ± 1.76 years; minimum: 5 years; maximum: 11 years). The asthma group had 25 males (75.8%), and the control group had 27 males (81.8%), with no statistically significant difference in sex distribution ($P = 0.547$). BMI also did not differ significantly between groups ($P = 0.365$). A higher proportion of mothers in the asthma group were housewives compared to the control group (81.8% vs. 51.5%; $\chi^2 = 6.82$, $\varphi = 0.32$, $P = 0.009$), and parental education levels also differed. Children with asthma were less likely to brush their teeth at least once a day (39.4% vs. 63.6%; $\chi^2 = 3.88$, $\varphi = 0.24$, $P = 0.049$) but showed similar habits regarding flossing, mouthwash use, and dental visits. Gingival inflammation (21.2% vs. 3.0%; $\chi^2 = 5.12$, $\varphi = 0.28$, $P = 0.024$) as well as the presence of materia alba and dental plaque (66.7% vs. 42.4%; $\chi^2 = 3.91$, $\varphi = 0.24$, $P = 0.048$) were significantly more prevalent in the asthma group (Table 1).

Asthma characteristics

Among children with asthma, the mean C-ACT score was 14.42 ± 3.85, with 93.9% classified as having uncontrolled asthma (C-ACT score < 19) (Table 2). A minority required hospitalization (15.2%), and 57.6% reported rinsing their mouths after using a glucocorticoid inhaler. Adenoid hypertrophy and allergic rhinitis were present in 12.1%

Table 1. Comparison of demographic, socioeconomic, oral hygiene, and clinical oral characteristics between children with asthma and healthy controls

Characteristic	Category	Asthma (n = 33)	Control (n = 33)	Test statistic	Effect size	*P*-value
Age (years)	–	7.82 ± 1.76	7.82 ± 1.76	$t = 0.00$	$d = 0.00$	1.000‡
Sex	Male	25 (75.8%)	27 (81.8%)	$\chi^2 = 0.36$	$\varphi = 0.07$	0.547†
	Female	8 (24.2%)	6 (18.2%)			
BMI (kg/m²)	–	16.14 ± 3.78	17.01 ± 3.96	$t = 0.91$	$d = 0.23$	0.365‡
Father's job	Employee	13 (39.4%)	14 (42.4%)	$\chi^2 = 0.06$	$\varphi = 0.03$	0.802†
	Other jobs	20 (60.6%)	19 (57.6%)			
Mother's job	Housekeeper	27 (81.8%)	17 (51.5%)	$\chi^2 = 6.82$	$\varphi = 0.32$	0.009†*
	Occupied	6 (18.2%)	16 (48.5%)			
Father's education	Less than a bachelor's degree	20 (60.6%)	5 (15.2%)	$\chi^2 = 16.10$	$V = 0.35$	< 0.001†*
	Bachelor's degree	9 (27.3%)	13 (39.4%)			
	Graduate degree	4 (12.1%)	15 (45.5%)			
Mother's education	Less than a bachelor's degree	19 (57.6%)	2 (6.1%)	$\chi^2 = 22.48$	$V = 0.41$	< 0.001†*
	Bachelor's degree	13 (39.4%)	22 (66.7%)			
	Graduate degree	1 (3.0%)	9 (27.3%)			
Brushing frequency	≤ 3 times/week	20 (60.6%)	12 (36.4%)	$\chi^2 = 3.88$	$\varphi = 0.24$	0.049†*
	≥ 1 time/day	13 (39.4%)	21 (63.6%)			
Dental floss use	Never	27 (81.8%)	24 (72.7%)	$\chi^2 = 0.78$	$\varphi = 0.11$	0.378†
	≥ 1 time/week	6 (18.2%)	9 (27.3%)			
Mouthwash use	Never	31 (93.9%)	28 (84.8%)	$\chi^2 = 1.44$	$\varphi = 0.15$	0.230†
	≥ 1 time/week	2 (6.1%)	5 (15.2%)			
Dental visits	Never	8 (24.2%)	4 (12.1%)	$\chi^2 = 1.63$	$V = 0.11$	0.442†
	Only if they have a problem	18 (54.5%)	21 (63.6%)			
	Regular	7 (21.2%)	8 (24.2%)			
Gingival inflammation	No	26 (78.8%)	32 (97.0%)	$\chi^2 = 5.12$	$\varphi = 0.28$	0.024†*
	Yes	7 (21.2%)	1 (3.0%)			
Presence of materia alba and dental plaque	No	11 (33.3%)	19 (57.6%)	$\chi^2 = 3.91$	$\varphi = 0.24$	0.048†*
	Yes	22 (66.7%)	14 (42.4%)			

Note. † *P*-value from Pearson's chi-squared test. Effect size: φ (phi) for 2 × 2 tables, Cramér's *V* for larger tables.
‡ *P*-value from independent-samples *t*-test. Effect size: Cohen's *d* (pooled *SD*). * $P < 0.05$.
BMI, body mass index

and 54.5% of cases, respectively.

Oral health outcomes

Mouth breathing was significantly more prevalent in the asthma group than in controls (57.6% vs. 18.2%; $\chi^2 = 10.88$, $\varphi = 0.41$, $P = 0.001$; Table 3).

Overall DMFT—a combined index of primary-tooth dmft (for children ≤ 9 years) and permanent-tooth DMFT (> 9 years)—was substantially higher in the asthma group (mean = 4.72 ± 3.28, 95% *CI* = 3.56 to 5.89; median = 4.00, IQR = 2.00–7.00) than in controls (mean = 0.73 ± 1.01, 95% *CI* = 0.37 to 1.08; median = 0.00, IQR = 0.00–1.00; Mann-Whitney U test, $z = -6.49$, $r = 0.64$, $P < 0.001$). The same pattern was observed in the age-stratified analyses: primary tooth dmft (≤ 9 years: $z = -5.89$, $r = 0.66$, $P < 0.001$) and permanent tooth DMFT (> 9 years: $z = -2.89$, $r = 0.77$, $P = 0.001$) were significantly higher in asthma (Table 3). A box plot of the overall DMFT distribution revealed one control outlier (DMFT = 4), while the asthma group showed greater variability (Figure 1). Excluding this outlier did not materially affect any of the univariate results (see sensitivity analysis).

DMFT and asthma medications

Within the asthma group, DMFT scores were compared between users and non-users of the four major medication classes (Table 4). There were no significant differences for anticholinergics ($n = 4$ users, 29 non-users; $z = -1.01$, $r = 0.17$, $P = 0.317$), inhaled glucocorticoids ($n = 27$ users, 6 non-users; $z = -0.71$, $r = 0.12$, $P = 0.480$), or oral glucocorticoids (n = 13 users, 20 non-users; $z = 0.20$, $r = 0.04$, $P = 0.838$). All 33 asthmatic children used beta-agonists, so no comparison was possible.

Multivariable analysis

The generalized linear model, adjusted for demographic, socioeconomic, oral hygiene, and clinical oral findings (Table 5), demonstrated asthma as the strongest independent predictor of DMFT, with an adjusted increase of 3.62 points (95% *CI* = 2.00 to 5.24, Wald $\chi^2 = 19.18$, partial $\eta^2 = 0.29$, $P < 0.001$). Other significant covariates were mother's education (bachelor's vs. graduate degree: $B = 1.47$, 95% *CI* = 0.05 to 2.89, $P = 0.042$), presence of materia alba and dental plaque (no vs. yes: $B = -1.53$, 95%

CI = -2.85 to -0.21, P = 0.023), and BMI (per unit increase: B = -0.16, 95% CI = -0.30 to -0.01, P = 0.037). Brushing frequency, floss use, mouthwash use, dental visit pattern, gingival inflammation, father's job, father's education, sex, and age were not significant. The model explained approximately 49% of the variance in DMFT (R^2 = 0.487; likelihood ratio χ^2 (17) = 49.29, P < 0.001). The Akaike Information Criterion (AIC) and Bayesian Information Criterion (BIC) were 326.1 and 367.7, respectively. Model-adjusted means were 0.75 (SE = 0.68) for controls and 4.37 (SE = 0.85) for the asthma group.

Table 2. Evaluation of the severity and symptoms of the patients in the asthma group (n = 33)

Variable	Category	Value
History of hospitalization	No	28 (84.8%)
	Yes	5 (15.2%)
Rinsing mouth after using glucocorticoid inhaler	No	14 (42.4%)
	Yes	19 (57.6%)
Adenoid hypertrophy	No	29 (87.9%)
	Yes	4 (12.1%)
Allergic rhinitis	No	15 (45.5%)
	Yes	18 (54.5%)
Duration of asthma (weeks)	–	185.82 ± 134.59
Asthma control (C-ACT)	Controlled (≥ 19)	2 (6.1%)
	Uncontrolled (< 19)	31 (93.9%)
C-ACT Questionnaire score	–	14.42 ± 3.85

Note. Categorical variables are shown as n (%). Continuous variables are shown as mean ± SD
C-ACT, Childhood-Asthma Control Test

Asthma control and DMFT

No significant correlation was observed between C-ACT score and overall DMFT (Spearman's ρ = 0.20, P = 0.261; Figure 2). This non-significant finding likely reflected the strongly skewed asthma control distribution, with 93.9% of the sample classified as uncontrolled, resulting in limited variability in C-ACT scores.

Sensitivity analysis

Removal of the single control outlier (DMFT = 4) did not alter any univariate comparisons. The overall DMFT Mann-Whitney test remained significant (z = -5.34, P < 0.001), as did the tests for primary tooth dmft (z = -4.34, P < 0.001), permanent tooth DMFT (z = -3.09, P = 0.002), and mouth breathing (χ^2 = 10.35, P = 0.001). In the multivariable model, the asthma coefficient was essentially unchanged (B = 3.86, 95% CI = 2.26 to 5.46, P < 0.001), and the other significant covariates remained consistent. After excluding the outlier, mother's education was borderline (B = 1.34, 95% CI = -0.07 to 2.75, P = 0.063; partial η^2 remained 0.08). Thus, the outlier was retained in all primary analyses.

Discussion

This cross-sectional study showed that dental caries experience, measured by the combined dmft/DMFT index, was substantially higher in children with asthma compared to age-matched healthy controls, with a large effect size (r = 0.64). The difference persisted after adjustment for demographics, socioeconomic status, oral hygiene behaviors, and clinical oral findings (adjusted mean difference = 3.62 points, 95% CI = 2.00 to 5.24). Notably, asthma was the strongest independent predictor of caries in

Table 3. Comparison of dental examination findings between children with asthma and healthy controls (n = 66)

Variable	**Asthma group** (n = 33)	**Control group** (n = 33)	Test statistic	Effect size	P-value
Mouth breathing, n (%)					
No	14 (42.4)	27 (81.8)	χ^2 = 10.88	φ = 0.41	0.001†*
Yes	19 (57.6)	6 (18.2)			
Overall DMFT (n = 66)	4.72 ± 3.28 (4.00, 2.00–7.00)	0.73 ± 1.01 (0.00, 0.00–1.00)	z = -6.49	r = 0.64	< 0.001‡*
dmft (primary, ≤ 9 y, [n = 52])	4.85 ± 3.45 (4.50, 2.00–7.00)	0.81 ± 1.06 (0.00, 0.00–1.00)	z = -5.89	r = 0.66	< 0.001‡*
DMFT (permanent, > 9 y, [n = 14])	4.29 ± 2.75 (4.00, 2.00–6.50)	0.43 ± 0.79 (0.00, 0.00–1.00)	z = -2.89	r = 0.77	0.001‡*

Note. Values are presented as mean ± SD (median, IQR) for continuous variables. † chi-squared test; $\varphi = \sqrt{\chi^2 / N}$.
‡ Mann-Whitney U test; $r = |z| / \sqrt{N}$. y, year old. DMFT/dmft recorded per WHO criteria. Overall DMFT = combined dmft (primary) + DMFT (permanent). * P < 0.05

Table 4. Comparison of the overall DMFT score between children with asthma using and not using each category of asthma medication (n = 33)

Asthma medicine	Medication use	n	DMFT Mean ± SD#	DMFT Median (IQR)	z	r‡	P-value†
Anticholinergic	No	29	4.52 ± 3.08	4.00 (2.00–7.00)	-1.01	0.174	0.317
	Yes	4	6.25 ± 4.79	7.50 (1.25–10.00)			
Beta-agonist ¶	Yes	33	4.72 ± 3.28	4.00 (2.00–7.00)	–	–	–
Inhaled glucocorticoid	No	6	3.83 ± 3.49	4.00 (0.75–5.50)	-0.71	0.123	0.480
	Yes	27	4.93 ± 3.27	5.00 (2.00–7.00)			
Oral glucocorticoid	No	20	4.85 ± 3.28	4.00 (2.25–7.75)	+0.20	0.035	0.838
	Yes	13	4.54 ± 3.41	5.00 (1.00–7.00)			

Note. † Mann-Whitney U test. ‡ effect size, $r = |z| / \sqrt{N}$
¶ Beta-agonists were used by all 33 asthmatic children; no comparison was possible
# DMFT represents the dmft (primary teeth) and DMFT (permanent teeth) index

Table 5. Generalized linear model of DMFT with group, demographic, socioeconomic, and oral hygiene predictors (n=66)

Characteristic	Category	*B*	95% CI	Wald χ^2	Partial η^2	*P*-value
Group	Asthma vs. Control (ref)	3.62	2.00, 5.24	19.18	0.29	<0.001*
Sex	Female vs. Male (ref)	0.24	-1.33, 1.80	0.09	<0.01	0.765
Father's job	Other jobs vs. Employee (ref)	1.24	-0.27, 2.75	2.58	0.05	0.108
Mother's job	Housekeeper vs. Occupied (ref)	-0.51	-1.56, 0.55	0.88	0.02	0.348
Father's education	Less than bachelor's vs. Graduate (ref)	0.38	-1.98, 2.73	0.10	<0.01	0.754
	Bachelor's degree vs. Graduate (ref)	-0.05	-1.20, 1.11	0.01	<0.01	0.939
Mother's education	Less than bachelor's vs. Graduate (ref)	0.04	-2.92, 2.99	<0.01	<0.01	0.981
	Bachelor's degree vs. Graduate (ref)	1.47	0.05, 2.89	4.12	0.08	0.042*
Brushing frequency	≤3 times/week vs. ≥1 time/day (ref)	-0.04	-1.24, 1.15	<0.01	<0.01	0.943
Floss use	Never vs. ≥1 time/week (ref)	0.42	-1.09, 1.92	0.30	<0.01	0.586
Mouthwash use	Never vs. ≥1 time/week (ref)	-0.18	-1.67, 1.32	0.06	<0.01	0.815
Dental visits	Never vs. Regular (ref)	0.55	-1.28, 2.37	0.34	<0.01	0.558
	Only if problem vs. Regular (ref)	0.26	-0.98, 1.49	0.17	<0.01	0.685
Gingival inflammation	No vs. Yes (ref)	-0.87	-2.72, 0.98	0.85	0.02	0.356
Presence of materia alba and dental plaque	No vs. Yes (ref)	-1.53	-2.85, -0.21	5.17	0.10	0.023*
Age (years)	(per 1-year increase)	-0.15	-0.53, 0.23	0.59	0.01	0.441
BMI (kg/m²)	(per 1-unit increase)	-0.16	-0.30, -0.01	4.35	0.08	0.037*

Note. Model: Generalized linear model with normal distribution, identity link function, and robust standard errors. ref=reference category. Dependent variable: overall DMFT (dmft + DMFT). * $P<0.05$
Partial η^2=Wald χ^2 / (Wald χ^2 + dferror), where dferror=48
Overall model fit: Likelihood ratio $\chi^2(17)=49.29$, $P<0.001$; $R^2=0.487$ (based on deviance)
AIC=326.1; BIC=367.7
Adjusted means (*SE*): Control 0.75 (0.68), Asthma 4.37 (0.85)

the multivariable model, while brushing frequency, flossing, and mouthwash use were not significant, suggesting that the higher caries burden in asthma is not solely attributable to poorer oral hygiene. This finding aligns with recent data from a large national survey showing that individuals with asthma, including children, have a significantly higher rate of dental treatment visits compared with those without asthma,[27] consistent with an elevated caries burden. The prevalence of mouth breathing was also significantly higher in the asthma group, reinforcing its role as a potential mediator.

Our findings are consistent with a meta-analysis that reported significantly higher DMFT in asthmatic children [8] and with other studies showing elevated caries experience in pediatric asthma populations.[28] However, some investigations have found no significant difference between asthmatic and non-asthmatic children,[9,10,29] with one study even reporting lower caries in the asthma group.[30] One likely explanation for the discrepancy is the severity of asthma in the studied samples. In our study, 93.9% of asthmatic children had uncontrolled disease (C-ACT<19), whereas previous studies may have included milder cases. The heavily skewed distribution of asthma control also explains the non-significant correlation between C-ACT scores and DMFT; insufficient variability in control status prevented detection of a linear relationship. Thus, our results primarily apply to children with moderate-to-severe uncontrolled asthma.

The significantly higher prevalence of mouth breathing in the asthma group aligns with prior reports.[3,31] In a recent cross-sectional study, ALQassab et al.[16] reported a mouth breathing prevalence of 65.9% in asthmatic children, consistent with the 57.6% observed in our participants. Mouth breathing bypasses nasal filtration and humidification, leading to dryness of the oral mucosa, reduced salivary flow, and a lower pH, all of which favor caries development.[32–34] In our study, allergic rhinitis was present in more than half of the asthmatic children, a condition known to cause nasal obstruction and promote mouth breathing.[35,36] Adenoid hypertrophy, although less frequent, may also contribute.[37] These findings underscore the need to evaluate and manage upper airway comorbidities in asthmatic children as part of comprehensive oral health care.

Socioeconomic factors also played a role. The asthma group had a higher proportion of mothers who were housewives and lower parental education levels, and these differences were associated with less frequent brushing, consistent with previous reports.[38] In the multivariable model, mother's education remained a significant independent predictor of DMFT, while brushing frequency did not. This suggests that socioeconomic factors may influence caries risk through pathways beyond daily hygiene, such as dietary habits, access to preventive dental care, and health literacy. Mother's occupation as a housewife, which was more common in the asthma group, may also reflect time constraints or lower health awareness, highlighting the importance of targeted education for caregivers.

We found no significant differences in DMFT between users and non-users of anticholinergics, inhaled glucocorticoids, or oral glucocorticoids. Since all asthmatic children in this study used beta-agonists, no comparison with non-users was possible. The small number of non-users in several medication categories limited statistical power. Future studies should consider pooling medication classes or using larger samples. Although inhaled medications can reduce salivary flow and lower pH [19] and salivary characteristics have been reported to be significantly worse in asthmatic adolescents,[39] our findings suggest that the overall caries burden in asthma may be driven more by the disease itself and associated mouth breathing than by a single drug class.[7,40] Some studies have reported that bronchodilator use is associated with a higher prevalence of severe caries [41] and the form of medication (e.g., tablets vs. sprays) may influence caries risk.[42] Nonetheless, proper inhaler technique and mouth rinsing after use remain important preventive measures.[20]

Several mechanisms may explain the higher caries risk. Mouth breathing and reduced salivary flow, possibly exacerbated by beta-agonist use, can give rise to a dry, acidic oral environment that promotes enamel demineralization and the growth of cariogenic bacteria. Asthma has been associated with decreased salivary pH and increased *Streptococcus mutans* counts.[7] It has been hypothesized that elevated exhaled nitric oxide, a marker of airway inflammation, could form nitric acid in the moist oral cavity and contribute to enamel erosion; however, direct evidence for this pathway in asthmatic children is lacking, and it remains speculative.[43] Beyond salivary and microbial changes, altered expression of genes related to cellular immunity signaling pathways may also contribute to the higher caries risk in asthma.[44] Furthermore, higher serum levels of IgG against *S. mutans* and *Lactobacillus*, as well as elevated IgA, have been documented in asthmatic patients,[45] which may be partly driven by inhaled corticosteroid therapy.[46] Genetic variations, such as single nucleotide polymorphisms (SNPs) in enamel formation genes (e.g., AMBN rs4694075) and immune-related genes, have been associated with both asthma and dental caries susceptibility (47–51). Additional studies support the role of enamel formation genes in caries experience (52–54), suggesting a possible genetic basis for the asthma–caries relationship; however, the clinical relevance of these genetic associations remains unclear. In addition to these pathophysiological changes, oral alterations in asthma may also stem from the cariogenic potential of anti-asthmatic medications, including their effects on salivary flow, buffering capacity, and pH, as well as their unpleasant taste, which can promote snacking on sugary foods.[55–57] In the present study, we did not assess genetic or salivary biomarkers, which limits our ability to directly explore these mechanisms. Hypomineralized enamel lesions secondary to early-onset respiratory disease may also predispose asthmatic children to caries.[58,59]

This study used a comprehensive set of confounders, a multivariable generalized linear model, and a sensitivity analysis to ensure robust findings. However, some limitations of this study warrant consideration. The cross-sectional design precludes causal inference. The sample size was modest and, although powered to detect the expected DMFT difference, may be insufficient for subgroup analyses or for detecting small effects of certain covariates. The asthma group was identified through parental self-report among children attending a university dental clinic, and most had uncontrolled asthma; therefore, the findings may not apply to children with well-controlled disease or those managed in primary care settings. Controls were recruited from schools that agreed to participate, which may introduce selection bias. A single examiner performed all oral examinations without formal intra-examiner reliability testing, though the examiner was trained and supervised by a pediatric dentist. Additionally, we did not measure salivary flow, pH, or microbial profiles, which would have strengthened the mechanistic interpretation. Notably, oral health can be influenced by other systemic factors, such as blood group phenotypes [60] and recent viral respiratory infections,[61] which were not assessed in our study and warrant further exploration.

The large independent effect of asthma on dental caries, even after controlling for hygiene, underscores the need for proactive dental care in children with asthma, especially those with uncontrolled disease. We recommend that dentists and pulmonologists collaborate to integrate oral health assessments into routine asthma care. Practical

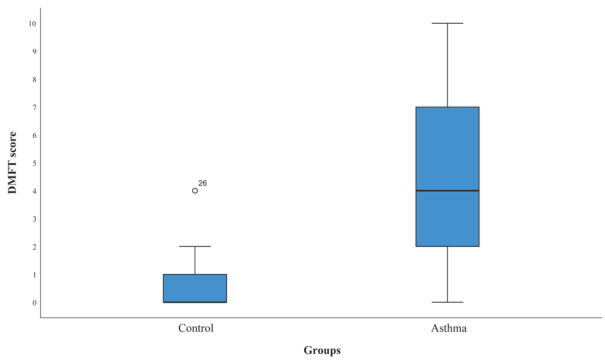

Figure 1. Distribution of overall DMFT/dmft in children with asthma and healthy controls. The bold horizontal line inside each box marks the median, the box covers the interquartile range (*IQR*), and the whiskers stretch to the farthest observation within 1.5 × *IQR*. The open circle labeled 26 indicates the single control outlier, with a DMFT of 4

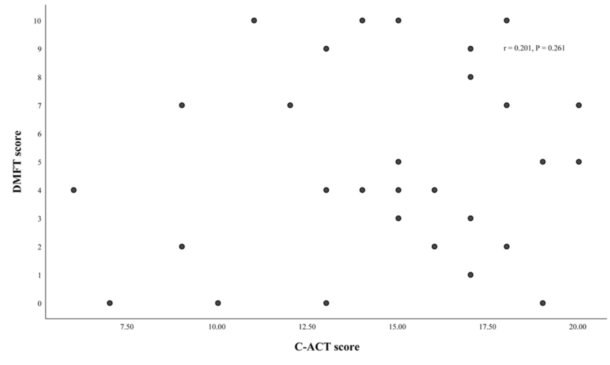

Figure 2. The correlation between C-ACT score and overall DMFT in asthma patients using Spearman's statistical test. C-ACT, Childhood-Asthma Control Test

measures include educating parents and children about the importance of twice-daily brushing with fluoride toothpaste and rinsing the mouth after inhaler use; using spacer devices to reduce oropharyngeal drug deposition; applying fluoride varnish and fissure sealants; and scheduling dental check-ups at least twice a year. Future research should employ longitudinal designs with larger, more diverse samples that include children with varying levels of asthma control. Studies should also collect salivary and microbiological data to test the mouth-breathing–caries pathway directly, and use advanced statistical methods to clarify the direct and indirect effects of asthma, medications, and oral hygiene behaviors.

Conclusion

In this study, children with asthma had a significantly higher overall DMFT score than healthy controls, even after adjusting for oral hygiene, socioeconomic factors, and clinical oral findings. Mouth breathing was substantially more prevalent in the asthma group and may be an important mediator. These findings indicate that asthma is independently associated with a higher burden of dental caries. Children with uncontrolled asthma, in particular, should receive enhanced preventive dental care, including regular check-ups, fluoride applications, and education on proper oral hygiene and correct inhaler use.

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Competing Interests

The authors declare no conflicts of interest.

Data Availability Statement

The data supporting this study's findings are available from the corresponding authors upon reasonable request.

Ethical Approval

The Ethics Committee of Kerman University of Medical Sciences reviewed and approved the study protocol (ethics code IR.KMU.REC.1401.426). All participants or their parents provided informed consent.

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