Submitted: 18 September, 2024 Accepted: 11 November, 2024 Published: 03 July, 2025 DOI:10.22514/jocpd.2025.088

ORIGINAL RESEARCH

Journal of Clinical Pediatric Dentistry

Analysis of the association between tooth number anomalies and precocious puberty in children

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Abstract

Background: Children with precocious puberty are often at an elevated risk for conditions like hypertension, obesity, diabetes and infertility. Some studies suggest a correlation between precocious puberty and dental development, including dental maturity, dental age and malocclusion. However, the relationship between precocious puberty and tooth number anomalies remains largely unexplored. Here, we investigate the link by analyzing two distinct populations. **Methods**: A total of 253 children were recruited using convenience sampling and categorized into two primary groups based on predefined inclusion and exclusion criteria: Group A (69 with tooth number anomalies, 56 without) and Group B (65 with precocious puberty, 63 without). **Results**: The results show that children with precocious puberty had significantly higher prevalence rates of tooth number anomalies and increased height, weight and body mass index (BMI) compared to those without ($p < 0.05$). Among children with precocious puberty, those with tooth number anomalies exhibited significantly higher levels of luteinizing hormone (LH) and follicle-stimulating hormone (FSH) compared to those without anomalies ($p < 0.05$). However, there are no significant differences in height, weight, BMI, LH or FSH levels between children with and without tooth number anomalies ($p > 0.05$). Further analysis showed that the independent risk factors for precocious puberty included sleeping with lights on, absence of a napping habit, low paternal education level, lack of intimacy with the father, frequent consumption of out-of-season fruits and fried foods, and maternal menarche before the age of 13 years. (Odds Ratio (OR) = 0.285, 2.971, 1.741, 2.085, 0.228, 0.266, 0.277; $p < 0.05$). **Conclusions**: Overall, this study indicates that children with precocious puberty have a higher prevalence of tooth number anomalies. Environmental, dietary, familial and genetic factors may influence the onset and progression of precocious puberty. Early assessment and timely intervention for tooth number anomalies in these children are crucial for their development.

Keywords

Precocious puberty; Tooth number anomalies; Sex hormones; Children; Risk factors

1. Introduction

Precocious puberty is characterized by the early onset of physical and psychological signs of puberty, including the development of secondary sexual features, such as breasts, start of menstruation and accelerated bone age beyond the expected maturity for the child's chronological age. In clinical terms, girls are considered to experience precocious puberty if breast development occurs before the age of 8 years or menarche begins before 9 years, while boys are diagnosed with precocious puberty when testicular and penile enlargement occur before the age of 9 years [1–3]. Multiple factors, including genetics, environment, diet, family history and lifestyle, may influence the onset of precocious puberty. The prevalence of precocious puberty has shown a rising trend globally, although incidence rates differ across countries and regions [4]. Pediatric endocrinology centers have reported a 2- to 3-fold increase in the number of children diagnosed with precocious puberty [5, 6]. Worldwide, the prevalence is estimated at 3.5 cases per 100,000 children annually, with a disproportionately higher incidence in females at 29 cases per 100,000 women, reflecting a female-to-male ratio of 20:1 [7]. In the United States, an observational study found that 10% of white girls and 23% of black girls had initiated puberty by the age of 7 years [8]. In Europe, approximately 5% of girls develop breast tissue by 8 years of age [7], while a Danish study reported a prevalence of 0.2% in girls and less than 0.05% in boys [4]. In China, the prevalence is estimated at 0.43%, with a higher incidence in girls compared to boys [9]. Precocious puberty not only limits a child's final adult height but also leads to emotional disturbances, resulting in psychological challenges such as anxiety, low self-esteem and mood instability. These

children face a heightened risk of developing conditions such as hypertension, obesity, diabetes, and infertility in adulthood, emphasizing the need for early diagnosis and intervention. Therefore, understanding the risk factors associated with precocious puberty is essential for designing preventive strategies and timely interventions.

Tooth number anomalies, which include congenital tooth loss and supernumerary teeth, represent the most common dental developmental abnormalities [10]. These anomalies can result from genetic predispositions, developmental disorders or systemic conditions [11–13]. Besides impairing dental function, such as chewing, aesthetics and speech, these anomalies can adversely affect children's self-confidence. The concept of Dental Anomaly Patterns (DAP) describes clusters of multiple dental anomalies, which often occur together [14]. DAP usually involves two or more concurrent dental anomalies, which may include missing teeth, microdontia, reduced tooth size, delayed tooth development and eruption and underbite, particularly in deciduous teeth and other manifestations could include palatal cusp displacement, maxillary first premolar transposition, mandibular lateral incisor-to-canine cusp transposition and abnormal angulation of the distal segment of an unerupted mandibular second premolar [15]. Genetic factors are recognized as potential causes of DAP or associated dental abnormalities (ADA), suggesting a complex relationship between genetics and dental anomalies [16]. A study conducted in Spain estimated the prevalence of dental agenesis within a primary healthcare region, revealing that the observed associations between certain systemic conditions and developmental dental anomalies may point to a shared genetic etiology [17]. In children, mixed dentition begins around the age of six to seven years, with primary teeth being replaced by permanent teeth. Several studies have explored the relationship between precocious puberty and dental development, reporting associations with dental maturity, dental age, malocclusion and mandibular growth patterns [18–20]. For instance, Kjellberg *et al*. [21] identified delayed tooth eruption in individuals with growth hormone (GH) deficiency. Additionally, assessments of the hypothalamic-pituitary axis, including measurements of luteinizing hormone (LH) and follicle-stimulating hormone (FSH), have been employed to explore endocrine function related to puberty and growth [22].

Currently, the incidence of tooth number anomalies and precocious puberty is on the rise among children in China. While numerous studies have investigated these conditions independently, limited research has explored the potential association between them. This study aims to examine the relationship between tooth number anomalies and precocious puberty by analyzing two distinct populations: children with tooth number anomalies and children diagnosed with precocious puberty, and to also identify risk factors associated with precocious puberty to support efforts aimed at preventing both tooth number anomalies and precocious puberty in children.

2. Materials and methods

2.1 Research subjects

This study included 253 children, with the sample size determined based on previous research conducted by Kim [23] and Tabakcilar [24]. A convenient sampling method was employed (Fig. 1), and the participants were recruited from the Stomatological Hospital affiliated with Zunyi Medical University between October 2022 and December 2023. Among the participants, 54.2% (N = 129) were girls, and 45.8% (N = 124) were boys. The participants were divided into two major groups, which were further subdivided into two subgroups. Group A consisted of 69 children with tooth number anomalies (Group A1: Tooth number anomalies precursor group) and 56 children without such anomalies (Group A2: Non-anomalies group). Group B comprised 65 children with precocious puberty (Group B1: Precocious puberty precursor group) and 63 children without precocious puberty (Group B2: Non-precocious puberty group). Informed consent was obtained from both the children and their parents prior to sample collection and questionnaire administration.

2.2 Inclusion criteria

Group A: girls aged 5–8 years old and boys aged 5–9 years old; diagnosed with supernumerary teeth or congenitally missing teeth; clear and complete cone beam computed tomogram (CBCT) images obtained at our hospital; and no history of cleft lip and palate, cranioclavicular dysplasia, Gardner syndrome, chondroepiphyseal dysplasia, or ectodermal dysplasia syndrome.

Group B: girls aged 5–8 years old and boys aged 5–9 years old; met the diagnostic criteria for precocious puberty [25]; clear and complete CBCT images obtained at our hospital; and voluntary cooperation of children's parents in completing the study questionnaire.

2.3 Exclusion criteria

Group A: history of extraction of supernumerary teeth or treatment for congenitally missing teeth; presence of severe artifacts or lack of clarity in CBCT images; parental refusal to allow sex hormone testing for the child; and presence of cleft lip and palate, cranioclavicular dysplasia, Gardner syndrome, chondroepiphyseal dysplasia, or ectodermal dysplasia syndrome.

Group B: precocious puberty due to organic lesions or a clearly identified etiology; history of extraction of supernumerary teeth or treatment for congenitally missing teeth; presence of severe artifacts or lack of clarity in CBCT images.

2.4 Research methods

2.4.1 Imaging data acquisition

Oral CBCT imaging for children in each group was performed by the same oral radiologist under consistent conditions. Two investigators independently collected and analyzed the imaging data to identify tooth number anomalies, including congenitally missing teeth and supernumerary teeth. CBCT [26] is increasingly utilized across medical disciplines for its ability to enhance diagnostic precision and guide treatment strategies. This imaging modality offers high-resolution, three-

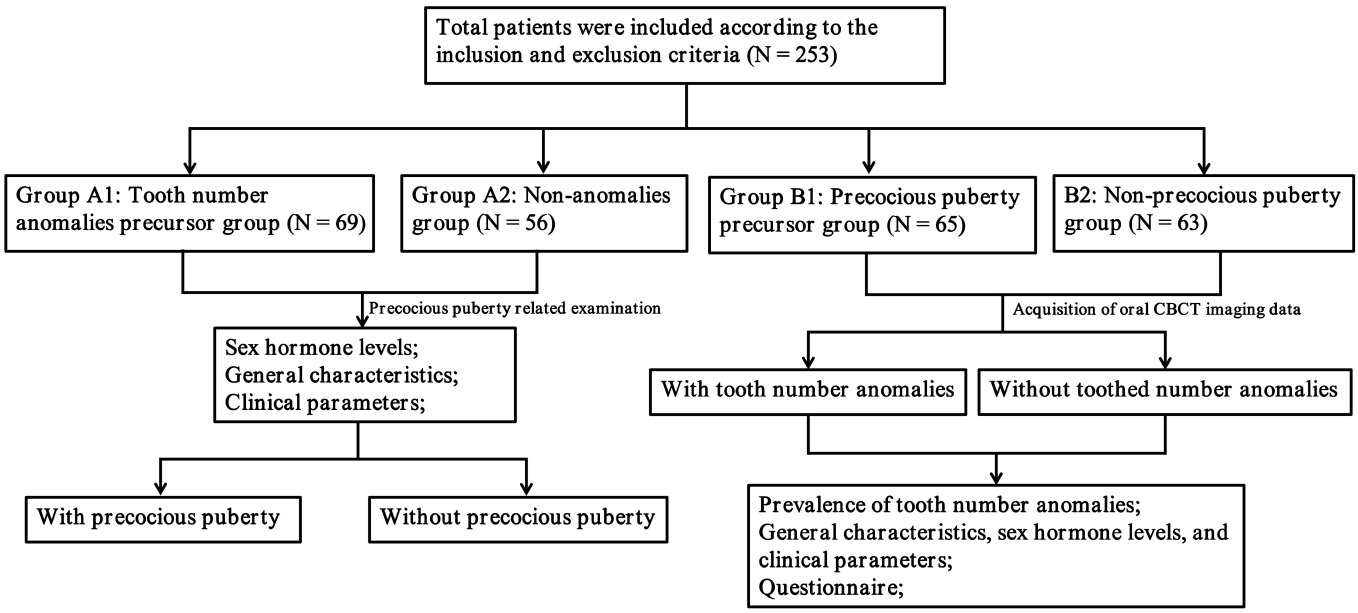

FIGURE 1. Flow diagram of patient classification. CBCT: Cone beam computed tomogram.

dimensional images, enabling accurate assessment of the oral cavity, dentition and maxillofacial structures, particularly in cases involving abnormal tooth development or jaw deformities. The diagnostic criteria for tooth anomalies were based on the 5th edition of Pediatric Dentistry [27].

2.4.2 Sex hormone test

Blood samples were collected in the early morning while the participants were in a fasting state, and the serum levels of LH and FSH were measured using chemiluminescence immunoassay with appropriate reagents.

2.4.3 Physical examination

Height and weight measurements were obtained using standard mechanical height measuring tapes and electronic weighing scales, respectively. The participants were instructed to remove hats, shoes and socks, wear light clothing, and undergo measurements on an empty stomach. Professional technicians, trained according to standardized protocols, conducted all physical assessments. Body mass index (BMI) was calculated using the formula BMI = Weight/Height^2 (kg/m^2).

2.4.4 Questionnaire survey

The questionnaires were developed following a thorough review of relevant literature and consultations with experts to ensure appropriateness and reliability, with a final reliability coefficient exceeding 0.85. They were distributed as paper forms to the parents of participating children and collected upon completion. In conjunction with clinical records, the questionnaires were used to obtain key data required for the study, covering a range of variables, which included general demographic information such as gender, age, habitual residence and whether the child was an only child. The questionnaires also gathered information on living habits, including daily sleep duration, napping patterns, use of lights while sleeping, physical activity and screen time. Dietary habits were assessed by inquiring about picky eating behaviors and the regular consumption of nutritional supplements, sweets, out-of-season fruits and vegetables, fried foods, legumes and milk. The family environment was evaluated based on the parents' marital status, educational levels, family atmosphere and the degree of parental closeness. Additionally, hereditary factors were recorded, such as the mother's menarche age and any family history of precocious puberty. Environmental exposures were assessed by determining whether the child lived near a chemical factory, had a preference for romance novels or television (TV) dramas, frequently used adult personal care products, or regularly interacted with plastic products. All questionnaires were completed voluntarily by the parents after receiving standardized instructions from the researchers to ensure consistency in responses. Two researchers independently recorded the data on-site, followed by a verification process to ensure the accuracy and consistency of the collected information.

2.5 Statistical methods

Data were analyzed using SPSS 18.0 software (IBM Corporation, Chicago, IL, USA). Normally distributed data are presented as mean ± standard deviation (SD), and non-normally distributed data are reported as medians with quartiles. Categorical variables were expressed as N (%) and analyzed using the chi-square test. Group differences were assessed using *t*-tests, chi-square tests and Kruskal-Wallis tests as appropriate. Binary logistic regression analysis was conducted to identify factors influencing the occurrence of precocious puberty. Statistical significance was defined as $p < 0.05$.

3. Results

3.1 Analysis of the detection rate of tooth number anomalies and its correlation with precocious puberty in each group

3.1.1 Comparison of physical examination findings and sex hormone levels in Group A

Although the comparison analysis between children with and without tooth number anomalies showed that the height, weight, BMI and serum levels of LH and FSH were lower in the tooth number anomalies precursor group compared to the non-anomalies group, these differences were not statistically significant ($p > 0.05$, Table 1).

3.1.2 Comparison of the detection of tooth number anomalies in Group B

The precocious puberty precursor group (B1) showed a significantly higher incidence of tooth number anomalies compared to the non-precocious puberty group (B2). Specifically, 24.6% (16/65) of the children in B1 were found to have tooth number anomalies, whereas only 7.9% (5/63) of children in B2 exhibited these anomalies, and this difference was statistically significant ($p < 0.05$, Table 2). Further analysis was conducted to determine whether supernumerary teeth or congenital tooth loss were associated with precocious puberty. The results showed that the detection rate of supernumerary teeth was 7.7% in the precocious puberty precursor group (B1) and 1.6% in the non-precocious puberty group (B2). Congenital tooth loss was detected in 16.9% of children in B1 and 6.3% of children in B2. Although the incidence of both types of anomalies was higher in the precocious puberty precursor group, the differences were not statistically significant ($p > 0.05$, Table 3).

3.1.3 Comparison of sex hormone levels in children from the precocious puberty precursor group

The analysis of sex hormone levels revealed significant differences between children with and without tooth number anomalies in the precocious puberty precursor group (B1), with the LH and FSH levels being higher in children with tooth number anomalies compared to those without ($p < 0.05$, Table 4).

3.1.4 Comparison of general clinical information and physical examination results between the precocious puberty group and the non-precocious puberty group

The comparison of general clinical data between the precocious puberty precursor group (B1) and the non-precocious puberty group (B2) showed significant differences in height, weight and BMI ($p < 0.05$). Children in the precocious puberty precursor group had higher values for these parameters compared to those in the non-precocious puberty group. However, no statistically significant differences were found for gender, age, habitual residence or whether the child was an only child ($p > 0.05$, Table 5).

3.2 Analysis of factors influencing precocious puberty

3.2.1 Living habits

The analysis of living habits revealed significant differences between the precocious puberty precursor group (B1) and the non-precocious puberty group (B2). Children in the precocious puberty group were more likely to have no napping habits, sleep with lights on frequently and engage in less daily exercise ($p < 0.05$, Table 6).

TABLE 1. Comparison of physical examination and sex hormone test results in Group A.

Variables	Group A1 (N = 69)	Group A2 (N = 56)	*t*	*p*
Age (yr)	7.41 ± 1.524	7.55 ± 1.138	0.587	0.558
Height (cm)	123.40 ± 9.076	124.32 ± 8.081	0.592	0.555
Weight (kg)	24.73 ± 4.437	25.24 ± 5.168	0.593	0.554
BMI (kg/m^2)	15.94 ± 1.716	16.31 ± 2.457	0.954	0.343
LH (U/L)	0.35 ± 0.244	0.42 ± 0.250	1.798	0.075
FSH (U/L)	1.82 ± 1.110	1.89 ± 0.646	0.467	0.641

Group A1: Tooth number anomalies precursor group; Group A2: Non-anomalies group. BMI: Body mass index; LH: Luteinizing hormone; FSH: Follicle-stimulating hormone.

TABLE 2. Comparison of the detection of tooth number anomalies in Group B.

Variables	Group B1 (N = 65)	Group B2 (N = 63)	χ^2	*p*
Tooth number anomalies				
Yes	16 (24.6)	5 (7.9)	6.489	0.011
No	49 (75.4)	58 (92.1)		

Group B1: Precocious puberty precursor group; Group B2: Non-precocious puberty group.

TABLE 3. Comparison of supernumerary teeth and congenital tooth loss among children with tooth number anomalies in Group B.

	Tooth number anomalies in Group B (N) (%)		*p*
	Group B1 (N = 11)	Group B2 (N = 5)	
Supernumerary teeth	5 (7.7)	1 (1.6)	0.365
Congenital tooth loss	11 (16.9)	4 (6.3)	0.097

Group B1: Precocious puberty precursor group; Group B2: Non-precocious puberty group.

TABLE 4. Comparison of LH and FSH levels in the precocious puberty precursor group ($\bar{x} \pm s$).

	Group B1		*t*	*p*
	With tooth number anomalies (N = 16)	Without tooth number anomalies (N = 49)		
LH (U/L)	3.46 ± 1.750	2.12 ± 2.313	2.266	0.027
FSH (U/L)	4.61 ± 1.917	3.32 ± 1.622	2.756	0.008

Group B1: Precocious puberty precursor group. LH: Luteinizing hormone; FSH: Follicle-stimulating hormone.

TABLE 5. Comparison of general clinical data and physical examination results between the two groups.

Variables	Group B1 (N = 65)	Group B2 (N = 63)	χ^2/t	*p*
Gender (N) (%)				
Male	6 (9.2)	11 (17.5)	2.837	0.092
Female	59 (90.8)	52 (82.5)		
Age ($\bar{x} \pm s$, yr)	7.23 ± 0.994	7.05 ± 1.138	3.562	0.053
Habitual residence (N) (%)				
Urban	54 (83.1)	51 (81.0)	0.098	0.754
Suburban	11 (16.9)	12 (19.0)		
An only child (N) (%)				
Yes	23 (35.4)	20 (31.7)	0.190	0.663
No	42 (64.6)	43 (68.3)		
Height ($\bar{x} \pm s$, cm)	137.64 ± 6.613	124.32 ± 8.077	10.221	<0.001
Weight ($\bar{x} \pm s$, kg)	36.25 ± 7.539	25.29 ± 5.039	9.694	<0.001
BMI ($\bar{x} \pm s$)	18.96 ± 2.672	16.29 ± 2.334	6.013	0.001

Group B1: Precocious puberty precursor group; Group B2: Non-precocious puberty group. BMI: body mass index.

3.2.2 Dietary habits

In terms of dietary habits, children in the precocious puberty precursor group (B1) exhibited significantly higher consumption of out-of-season fruits and vegetables as well as fried foods compared to those in the non-precocious puberty group (B2) ($p < 0.05$). However, no statistically significant differences were observed for other dietary variables (Table 7).

3.2.3 Family environment

The analysis of family environment revealed significant differences between the precocious puberty precursor group (B1) and the non-precocious puberty group (B2) regarding the father's education level and the child's relationship with the father ($p < 0.05$). However, there were no statistically significant differences between the two groups in terms of parents' marital status, family atmosphere, mother's education level, or the child's relationship with the mother ($p > 0.05$, Table 8).

3.2.4 Genetic factors

The analysis of genetic factors associated with precocious puberty revealed that a significantly higher proportion of mothers with early menarche (before the age of 13) was found in the precocious puberty precursor group (B1) compared to the non-precocious puberty group (B2) ($p < 0.05$, Table 9).

TABLE 6. Comparison of children's living habits between the precocious puberty precursor group and the non-precocious puberty group (N, %).

Variables	Group B1 (N = 65)	Group B2 (N = 63)	χ^2	p
Napping habits				
Yes	21 (32.3)	38 (60.3)	10.101	0.001
No	44 (67.7)	25 (39.7)		
Sleep with lights on frequently				
Yes	23 (35.4)	9 (14.3)	7.569	0.049
No	42 (64.6)	54 (85.7)		
Daily sleep time				
<8 h	12 (18.5)	11 (17.5)		
8–10 h	48 (73.8)	45 (71.4)	0.442	0.802
>10 h	5 (7.7)	7 (11.1)		
Daily exercise time				
<1 h	28 (43.7)	12 (19.4)		
1–2 h	25 (38.5)	31 (49.2)	9.014	0.011
>2 h	12 (17.8)	20 (31.4)		
Daily use time of electronic products				
<1 h	11 (16.9)	19 (30.2)		
1–2 h	37 (56.9)	36 (55.4)	5.357	0.069
>2 h	17 (26.2)	8 (14.4)		

Group B1: Precocious puberty precursor group; Group B2: Non-precocious puberty group.

TABLE 7. Comparison of children's dietary habits between the precocious puberty precursor group and the non-precocious puberty group (N, %).

Variables	Group B1 (N = 65)	Group B2 (N = 63)	χ^2	p
Preferences (certain foods, unhealthy foods)				
Yes	41 (63.1)	38 (60.3)	0.103	0.748
No	24 (36.9)	25 (39.7)		
Consumption of nutritional supplements (often)				
Yes	6 (9.2)	1 (1.5)	2.288	0.130
No	59 (90.8)	62 (98.5)		
Consumption of dessert/ sugary drinks (often)				
Yes	27 (41.5)	22 (34.9)	0.593	0.441
No	38 (58.5)	41 (65.1)		
Consumption of out-of-season fruits and vegetables (often)				
Yes	21 (32.3)	7 (9.5)	8.411	0.004
No	44 (67.7)	56 (90.5)		
Consumption of fried food (often)				
Yes	27 (41.5)	11 (17.5)	8.886	0.003
No	38 (58.5)	52 (82.5)		
Consumption of beans/soy products (often)				
Yes	39 (60.0)	33 (52.4)	0.755	0.385
No	26 (40.0)	30 (47.6)		
Consumption of milk (often)				
Yes	37 (56.9)	36 (57.1)	0.001	0.980
No	28 (43.1)	27 (42.9)		
Consumption of food packaged in plastic (often)				
Yes	32 (49.2)	28 (44.4)	0.294	0.587
No	33 (50.8)	35 (55.6)		
Consumption of carbonated beverages (often)				
Yes	9 (13.8)	5 (7.9)	1.147	0.284
No	56 (86.2)	58 (92.1)		

Group B1: Precocious puberty precursor group; Group B2: Non-precocious puberty group.

TABLE 8. Comparison of the Family Environment of Children in the Precocious Puberty Precursor Group and the Non-Precocious Puberty Group (N, %).

Variables	Group B1 (N = 65)	Group B2 (N = 63)	χ^2	p
Parents' marital status				
First marriage	57 (87.7)	58 (92.1)		
Divorced	6 (9.2)	4 (6.3)	2.069	0.355
Remarried	2 (3.1)	1 (1.6)		
Family atmosphere				
Harmonious	21 (32.3)	28 (44.4)		
General	39 (60.0)	33 (52.4)	2.677	0.268
Not harmonious	5 (7.7)	2 (3.2)		
Father's education level				
Junior high school education	12 (18.5)	9 (14.3)		
High school education	37 (56.9)	25 (39.7)	6.610	0.037
College education or above	16 (24.6)	29 (46.0)		
Mother's education level				
Junior high school education	21 (32.3)	16 (25.4)		
High school education	25 (38.5)	22 (34.9)	1.655	0.437
College education or above	19 (29.2)	25 (39.7)		
Relationship with father				
Intimacy	12 (18.7)	23 (36.5)		
General	46 (70.8)	37 (58.7)	2.588	0.010
Lack of intimacy	7 (10.5)	3 (4.8)		
Relationship with mother				
Intimacy	29 (44.6)	34 (54.0)		
General	33 (50.8)	28 (44.4)	1.345	0.179
Lack of intimacy	3 (4.6)	1 (1.6)		

Group B1: Precocious puberty precursor group; Group B2: Non-precocious puberty group.

TABLE 9. Comparison of genetic correlates between the precocious puberty precursor group and the non-precocious puberty group (N, %).

Variables	Group B1 (N = 65)	Group B2 (N = 63)	χ^2	p
Mother's menarche time				
Before the age of 13	32 (49.2)	14 (22.2)	10.137	0.001
After the age of 13	33 (50.8)	49 (77.8)		
Family history of precocious puberty				
Yes	7 (10.8)	1 (1.6)	5.245	0.022
No	58 (89.2)	62 (98.4)		

Group B1: Precocious puberty precursor group; Group B2: Non-precocious puberty group.

3.2.5 Environmental exposure

The analysis of environmental exposure factors showed no statistically significant differences between the two groups regarding whether the children lived near a chemical industrial zone, whether they had a preference for romance novels or TV dramas, or whether they frequently used adult skin care products and cosmetics (*e.g.*, lotions, lipsticks, nail polish). Similarly, no statistical difference was observed in the frequency of plastic product use, such as plastic packaging bags and disposable plastic tableware ($p > 0.05$, Table 10).

TABLE 10. Comparison of environmental exposure between the precocious puberty precursor group and the non-precocious puberty group (N, %).

Variables	Group B1 (N = 65)	Group B2 (N = 63)	χ^2	p
Living environment (chemical industry zone)				
Yes	9 (13.8)	5 (7.9)	0.147	0.397
No	56 (86.2)	58 (92.1)		
Love romance novels or TV series				
Yes	17 (26.2)	9 (15.9)	2.784	0.095
No	48 (73.8)	54 (84.1)		
Use of adult skincare/cosmetics (often)				
Yes	3 (4.6)	1 (1.6)	0.227	0.634
No	62 (95.4)	62 (98.4)		
Use of plastic products (often)				
Yes	32 (49.2)	28 (44.4)	0.294	0.587
No	33 (50.8)	35 (55.6)		

Group B1: Precocious puberty precursor group; Group B2: Non-precocious puberty group.

3.3 Binary logistic regression analysis of factors influencing the occurrence of precocious puberty in children

To control for the influence of multiple variables, binary logistic regression analysis was conducted. Independent variables were selected based on indicators that demonstrated significant differences in the previous univariate analysis, with corresponding values assigned for analysis. The occurrence of precocious puberty was defined as the dependent variable (Table 11). The binary logistic regression analysis identified several independent risk factors associated with the development of precocious puberty, such as frequent sleeping with lights on, absence of a napping habit, low paternal education level, lack of a close relationship with the father, frequent consumption of out-of-season fruits and vegetables, regular consumption of fried food, and early maternal menarche (before the age of 13) (Table 12).

4. Discussion

This present study explored the relationship between tooth number anomalies and precocious puberty in children. Early identification of precocious puberty is essential because delayed diagnosis and intervention can result in reduced growth potential and psychosocial complications [28, 29]. Several studies have identified screening or predictive markers for precocious puberty, with dental development reported as a potential indicator. In addition, it has been previously highlighted that there might be a link between dental development and precocious puberty. For example, Roberts *et al.* [30] reported significantly delayed dental development in patients with idiopathic precocious puberty. Baik *et al.* [31] proposed the use of dental maturity as a screening tool for precocious puberty by evaluating the calcification stages of the mandibular second premolars and molars. These findings indicate the

TABLE 11. Variable assignments for binary logistic regression analysis.

Variables	Variable assignments
Precocious puberty	Yes = 1, No = 2
Consumption of out-of-season fruits and vegetables (often)	Yes = 1, No = 2
Consumption of fried food (often)	Yes = 1, No = 2
Sleep with lights on (often)	Yes = 1, No = 2
Napping habits	Yes = 1, No = 2
Daily sleep time	<8 h = 1, 8–10 h = 2, >10 h = 3
Daily exercise time	<1 h = 1, 1–2 h = 2, >2 h = 3
Father's education level	Junior high school education = 1, High school education = 2, College education or above = 3
Relationship with father	Intimacy = 1, General = 2, Lack of intimacy = 3
Mother's menarche time	Before the age of 13 = 1, After the age of 13 = 2
Family history of precocious puberty	Yes = 1, No = 2

TABLE 12. Binary logistic regression analysis of factors influencing precocious puberty in children.

Variables	B	SD	Wald	*p*	OR	95% CI
Consumption of out-of-season fruits and vegetables (often)	−1.480	0.461	10.289	0.001	0.228	0.092–0.562
Consumption of fried food (often)	−1.323	0.400	10.965	0.001	0.266	0.122–0.583
Napping habits	1.089	0.367	8.781	0.003	2.971	1.446–6.105
Sleep with lights on (often)	−1.256	0.442	8.065	0.005	0.285	0.120–0.678
Father's education level	0.554	0.265	4.372	0.037	1.741	1.035–2.926
Relationship with father	0.735	0.332	4.911	0.027	2.085	1.089–3.995
Mother's menarche time	−1.284	0.392	10.740	0.001	0.277	0.129–0.597

B: Regression coefficients; SD: Standard deviation; OR: Odds ratio; CI: Confidence interval.

potential of dental assessments for identifying central precocious puberty. Similarly, Lee *et al*. [18] observed that children with precocious puberty had more advanced dental development compared to their peers, and Tabakcilar *et al*. [24] compared dental age with skeletal age, using panoramic and wrist radiographs and found that children with precocious puberty had dental ages significantly higher than their chronological ages. These findings suggest the relevance of understanding the association between dental development and the onset of puberty, as teeth may offer valuable insights into the timing and progression of puberty, particularly in the context of precocious puberty, facilitating early diagnosis and management.

In clinical practice, gonadotropin-releasing hormone (GnRH) stimulation tests are used to differentiate between central and peripheral precocious puberty based on patients' clinical presentations. However, these tests involve repeated sampling and are time-consuming and costly. Therefore, our present study did not differentiate between subtypes of precocious puberty but instead grouped all cases under the precocious puberty precursor group. The imaging data from 65 children in the precocious puberty precursor group and 63 children in the non-precocious puberty group were analyzed, and the findings revealed a higher prevalence of tooth number anomalies in children with precocious puberty compared to those without (Table 2), suggesting a potential association between precocious puberty and tooth number anomalies. Additionally, the children in the tooth number anomalies precursor group exhibited lower values for height, weight, BMI, LH and FSH compared to children without such anomalies. Although these results suggest that children with tooth number anomalies do not exhibit a predisposition toward precocious puberty, differences in the clinical presentation of puberty may exist. The sequence of pubertal changes and the rate of progression may vary, presenting either as a sudden onset or a gradual, intermittent process, often influenced by fluctuations in estrogen, androgens or both. Given these variations, it is essential to monitor the growth and development of children with tooth number anomalies closely to detect early signs of precocious puberty. Early detection and timely intervention are crucial to prevent the potential adverse effects of early maturation on growth, psychological well-being, and long-term health outcomes.

In this study, tooth number anomalies in the precocious puberty precursor and non-precocious puberty groups were further categorized into supernumerary teeth and congenital tooth loss. Kim *et al*. [23] reported that children with precocious puberty exhibited a higher incidence of abnormal maxillary dental development compared to the control group. Their case-control study also found an increased prevalence of maxillary median supernumerary teeth in children with precocious puberty, suggesting a possible association between precocious puberty and abnormal maxillary dental development. Herein, our findings similarly indicated a significant difference in the incidence of tooth number anomalies between the two groups. However, no statistically significant variation was observed in the occurrence of supernumerary teeth (Table 3). This discrepancy could be attributed to the limited sample size in the present study, highlighting the need for larger sample sizes in future research.

The onset of puberty involves increased secretion of kisspeptin and neurokinin B, which stimulate GnRH neurons to secrete gonadotropins in a pulsatile pattern. The subsequent release of GnRH promotes the secretion of LH and FSH [32], which stimulate the production of gonadotropic sex hormones to facilitate the development of secondary sexual characteristics. Through a negative feedback mechanism, sex hormones also regulate hypothalamic-pituitary function. From a histoembryological perspective, the pituitary gland plays a critical role in the migration of neural crest cells involved in oral cavity formation [33]. The anterior pituitary shares a developmental origin with the neural crest cells of the oral cavity, while the posterior pituitary shares an origin with the mesenchyme of the maxillofacial region. The pituitary gland resides in the sella turcica (pyriform saddle), a bony structure associated with dental development. Previous studies have reported that malformations of the sella turcica are linked to dental developmental disorders [34]. Additionally, hormones secreted by the pituitary gland have been implicated in both dental development and jaw growth. Abnormal pituitary development has been shown to result in morphological abnormalities of the sella turcica, potentially leading to dental anomalies [35]. In the present study, children in the precocious puberty precursor group who exhibited tooth number anomalies had elevated levels of LH and FSH compared to those without such anomalies (Table 4), thereby suggesting that differences in sex hormone levels between children with and without tooth number anomalies

may be associated with underlying pituitary developmental abnormalities. Further research is needed to elucidate the exact mechanisms underlying these associations.

Recently, much emphasis has been placed on precocious puberty, making it a growing global health concern [36]. Although its pathophysiology remains unclear, numerous studies have tried to identify factors contributing to its onset. In this study, children in the precocious puberty precursor group exhibited higher height, weight, and BMI compared to those in the non-precocious puberty group, which aligns with findings from previous reports [37–39]. Liu *et al*. [9] found that overweight and obesity were linked to precocious puberty, although this association was gender-specific and influenced by environmental factors. Similarly, Rosenfield *et al*. [40] observed a significantly higher prevalence of precocious puberty in girls with elevated BMI between the ages of 8.0 and 9.6 years. Additionally, Bigambo *et al*. [41] highlighted the importance of childhood BMI as a risk factor for precocious puberty, particularly among girls. The improved quality of life among the general population has prompted lifestyle changes in children. Increased use of electronic devices such as mobile phones, televisions, and computers has reduced time spent on physical activity and disrupted sleep patterns, adversely affecting children's growth and development. As a result, environmental and genetic factors are regarded as key contributors to the onset of puberty [42]. Research into the genetic basis of central precocious puberty continues to offer insights into its underlying mechanisms [43, 44]. Some studies suggest that the timing of the mother's menarche is closely related to the onset of puberty in her child, with the child's menarche often occurring around the same age as the mother's [45]. Other studies have proposed that precocious puberty may be influenced by various factors, including sex, bone age, exercise habits, E2, FSH, LH, leptin levels, maternal menarche timing, living environment, dietary patterns and sleep duration [46]. For example, Jessen *et al*. [47] identified an association between the onset of puberty and changes in sleep duration through a cross-sectional case-control study. In the present study, binary logistic regression analysis identified several independent risk factors for the onset of precocious puberty, which included frequent sleeping with lights on, absence of napping habits, low paternal education level, lack of closeness with the father, frequent consumption of out-of-season fruits and vegetables, regular consumption of fried foods, and maternal menarche before the age of 13. Previous research has shown that excessive light exposure from sleeping with lights on at night can disrupt the pineal gland's melatonin secretion, which in turn promotes the onset of precocious puberty [48]. Additionally, the consumption of out-of-season fruits and vegetables may expose children to "growth promoters" used in their production, potentially accelerating maturation. This phenomenon differs from the hormonal effects associated with soybean products [49]. Given these findings, children with a family history of precocious puberty or whose mothers experienced early menarche should undergo regular follow-up with specialists to enable early detection, intervention, and treatment, minimizing the adverse effects of precocious puberty. A related study [50] found that traditional dietary patterns were negatively associated with children's BMI and positively linked to parental education. In particular, higher maternal education was associated with healthier protein dietary patterns, possibly due to the parents' greater awareness of promoting their children's well-being. Parents with richer educational backgrounds are more likely to possess the knowledge needed to reduce their children's exposure to factors that increase the risk of precocious puberty, underscoring the importance of parental education in preventing early maturation.

With the rise of the Internet and rapid media dissemination, children now have easy access to electronic devices, exposing them to largely unscreened content. This unrestricted access increases the likelihood of encountering age-inappropriate material and subjects them to subtle environmental influences. Studies have identified regular exposure to romantic media as a risk factor for precocious puberty [51]. Although no statistically significant associations were observed in this study regarding children's environmental exposures, the percentage of children in the precocious puberty precursor group who lived near chemical factories, enjoyed romance novels or TV dramas, regularly used adult daily products, and frequently utilized plastics was higher than in the non-precocious puberty group.

This study provides new insights into the relationship between tooth number anomalies and the onset of early puberty in children. The diagnosis of precocious puberty was confirmed by specialist doctors, ensuring the reliability and accuracy of the results. However, several limitations need to be addressed. Despite efforts to maintain data quality, potential information bias could not be fully avoided, as the questionnaire relied on parental self-reporting, which may have introduced reporting bias. Additionally, the study was limited to a single hospital in Zunyi, Guizhou, China, resulting in a selective cohort. The use of convenience sampling within one hospital may have influenced the findings and restricted the generalizability of the results. Furthermore, the relatively small sample size limits the strength of the conclusions, highlighting the need for future research with larger and more diverse populations. Another limitation is that the questionnaire identified cases of precocious puberty but did not differentiate between subtypes, such as central precocious puberty. Future studies should aim to explore the specific characteristics that predispose individuals to central precocious puberty. Increasing the sample size and distinguishing between subtypes could improve the understanding of these associations, facilitating early detection and more targeted interventions.

5. Conclusions

This study revealed a higher prevalence of tooth number anomalies in children with precocious puberty compared to those without. Precocious puberty was associated with elevated sex hormone levels, frequent sleeping with lights on, lack of napping habits, low paternal education, poor father-child intimacy, consumption of out-of-season fruits and vegetables, frequent intake of fried foods and maternal menarche before the age of 13. Clinicians should consider a child's lifestyle, diet, family environment, and genetic background when working to prevent precocious puberty.

Upon confirming precocious puberty, it is important to assess the child's tooth count and consult the stomatology department if necessary to address any noticeable dental signs. For children with abnormal tooth counts, regular monitoring of their development and follow-up with a pediatric endocrinologist are recommended.

AVAILABILITY OF DATA AND MATERIALS

The data presented in this study are available on reasonable request from the corresponding author.

AUTHOR CONTRIBUTIONS

XS and MML—designed the study, conduction the experiment, data analysis, and wrote the paper. SHL and JY—contributed to analysis and interpretation of findings and critically revised the manuscript. QGS—designed the study, and revised the manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This study was approved by the Ethics Committee of the Affiliated Stomatological Hospital of Zunyi Medical University, No. YJSKTLS-2020-2023-006H. Parents or legal guardians of participants provided informed consent and agreed to publication details of this study.

ACKNOWLEDGMENT

We thank all the children and their parents for their participating in this study.

FUNDING

This study was financially supported by the Zunyi Science and Technology Support Project [ZSKHHZZ(2020)294].

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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How to cite this article: Xin Shu, Mingmin Lv, Shenhu Liang, Jing Yang, Qinggao Song. Analysis of the association between tooth number anomalies and precocious puberty in children. Journal of Clinical Pediatric Dentistry. 2025; 49(4): 172-183. doi: 10.22514/jocpd.2025.088.