Original Article

Dental Caries and Gingival Evaluation in Children with Congenital Heart Disease

Abstract

Background: Dental health is one of the most important health burdens of children health. The association between dental health and endocarditis has been already demonstrated, but there is controversy about different frequency of dental caries, periodontitis, and saliva microorganism in comparison to healthy population and children with congenital heart diseases (CHDs). In this study, we evaluated these differences. **Methods:** Seventy-six healthy children and 68 CHD patients were enrolled in the present case–control study. Dental decay, periodontitis, oral microorganisms, serum calcium, phosphorus, and frequency of carbohydrate and protein consumption of all participants were evaluated by standards method. **Results:** CHD patients experienced more periodontitis, but the difference was not significant (0.12 vs. 0.09, $P = 0.2$). In healthy children, the mean saliva colony counts of *Streptococcus mutans* were more significant (50639 ± 3324 vs. 35285 ± 27226, $P = 0.03$), which was diminished by adjusting the carbohydrate consumption. The mean colony count of Lactobacilli in children with CHD was nonsignificant higher than healthy children ($P = 0.3$). **Conclusions:** Pediatric patients with CHD experience insignificantly higher dental decay, periodontitis, and saliva Lactobacilli colony counts. The frequency of decayed tooth and gingival diseases in healthy children is high, and hence, more dental care attention in our health system is needed for healthy children.

Keywords: *Congenital heart disease, dental caries, periodontal disease*

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Introduction

One of the most important health problems in normal child population worldwide is dental caries and periodontal diseases.[1] These two global health problems are more considered in children with congenital heart disease (CHD).[2] The importance is because of CHD high prevalence of about 8–10/1000 births and the susceptibility of these children to endocarditis.[3] The infectious endocarditis is associated with induced bacteremia by dental procedures and dental brushing and flossing.[4]

Formation and progression of dental caries are related to oral microorganisms. *Streptococcus mutans* and *Lactobacillus* spp. are two main microorganisms.[5] *S. mutans* is colonized after first dental eruption and *Lactobacillus* is found in oral cavity before the first eruption.[6,7] Findings show a positive correlation between consumption of carbohydrates and colonization of these two microorganisms.[8] Studies have shown that *S. mutans* begins the initial formation of caries and the *Lactobacillus* causes further development of carious lesions.[9] Findings of some researches have shown that these organisms grow more in the oral cavity of patients with CHD. On the other hand, other groups of researchers suggested that initiation of dental caries in children with CHD may be due to low calcium in dental enamel of this population.[10] Furthermore, hypoxia in children with cyanotic CHD was another factor associated with dental caries in a number of studies.[11]

Periodontal disease is another risk of endocarditis in patients suffering from CHD.[12] Bad oral hygiene, taking different kinds of medications, insufficient food and minerals consumption are the risk factors for the initiation of periodontal disease in children with congenital cardiovascular disease.[13,14] Besides, research findings have suggested the positive correlation between periodontal disease and congenital cardiovascular problems.[15]

As far as we have searched, there are controversial data about the differences

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in oral health in normal children and patients with CHD.[16-18] This controversy is more prominent, especially in developing countries;[16] therefore, in the present study, we evaluate the saliva microorganism, dental caries, and periodontal disease in children with and without CHD. In addition, we seek to control for the effect of other causes of dental caries in CHD patients such as calcium and phosphorus and frequency of food ingestion.

Methods

In the present case–control study, 76 healthy and 69 children with CHD were included in the study. Ethical Committee of Isfahan University of Medical Sciences approved the protocol of this study (ethical number: 394244). The patients with CHD were enrolled as by convenience sampling from the patients referring pediatric heart clinics affiliated with the Isfahan University of Medical sciences. The healthy children were randomly enrolled from the children who accepted our calls for dental evaluation. Participants in two groups were matched by their age and sex. Informed consent was signed by parents of all children. Among included participants, those who took any kinds of antibiotics, probiotics, and synbiotics in the last month were excluded from the study.

The demographic data (age, gender, family income), medical history (kind of CHD, cyanotic or noncyanotic), and frequency of toothbrushing and flossing were collected. Parents filled out the food frequency questionnaire[19] for the frequency of carbohydrate and protein intake of their children in during the last month. Body weight and height of all children were measured by standards methods by pediatric resident, and their body mass index was calculated.[20]

Dental evaluation of study participants

One pediatric dentist examined all children and dental caries was reported according to the World Health Organization criteria as the number of decayed, missing, and filled teeth (DMFT).[21] Periodontitis was evaluated by gingival bleeding index. According to this method, gentle probing of orifice of the gingiva is done, and then, in 10 s, bleeding is observed. If bleeding happens, the test is positive and then the number of positive sites is recorded and expressed as a percentage of the number of the examined teeth site.[22] The simplified oral hygiene index[23] was used for detecting children oral hygiene. Before saliva sampling, the blood samples of the children were collected to measure the serum level of calcium and phosphorus among them. Two millimeters saliva of children was taken and brought to Alzahra Hospital Laboratory, Isfahan University of Medical Sciences, within 1 h after sampling.

Streptococcus mutans

For determination of *S. mutans* count, 20 µl of saliva sample was spread on Mitis Salivarius agar (Difco) supplemented with 0.2 U/ml bacitracin and sucrose (15% w/v). Serial dilution and spread plate technique were used for viable cell count. The duplicated agar plates of each dilution were incubated anaerobically (85% N_2, 5% CO_2, and 10% H_2) at 30°C for 72 h.[23] Observing the colonial morphology on Mitis Salivarius agar, hemolysis on blood agar medium and biochemical tests including Gram staining, Voges–Proskauer test, and sugar fermentation tests including mannitol, sucrose, raffinose, sorbitol, and salicin were used for the identification of *S. mutans*. Then, these colonies were counted by measuring the original concentration of *S. mutans* in the saliva in colony forming units (CFUs)/mL.[24]

In the present study, Rogosa agar (Unipath, Basingstoke, UK) was used for detecting total count of Lactobacilli in saliva samples. 20 µl of saliva samples was spread on Rogosa agar, and as per the above method, serial dilution and spread plate technique were used for viable cell count. Medium was also incubated anaerobically (85% N_2, 5% CO_2, and 10% H_2) at 30°C for 72 h. Lactobacilli were identified with colony characters by the Gram staining method. Lactobacilli appeared straight, rod-shaped and in pairs of varying length. Number of colonies was counted using a digital counter and its concentration in saliva was expressed in CFU/mL.[24,25] Two study groups were stratified into three categories according to DMFT (DMFT = 0, DMFT = 1–3, DMFT ≥3).[26] The colony count of *S. mutans* and Lactobacilli was categorized into low, medium, and high.[27]

Statistical analysis

The analyses were done by IBM SPSS Statistics 20.0 software (SPSS Inc., Chicago, Illinois). Student's *t*-test and Chi-square test were used to find the significant differences between the groups. Moreover, a $P \leq 0.05$ was considered statistically significant. This questionnaire is a validated semi-quantitative questionnaire[19,28] and is used for detecting the frequency consumption of carbohydrate and proteins. The data of this questionnaire were analyzed in Nutritionist III software (version 7.0; N-Squared Computing, Salem, OR, USA).

Results

One hundred forty-two children consisting of 74 healthy and 68 CHD children were assessed in the present study. Thirty-eight patients had cyanotic heart diseases and 44 children with congenital cardiovascular disease were reported to undergo surgery. The demographic data of all participants are shown in Table 1. There were not any significant differences in demographic data between the two groups.

Healthy child consumed significantly higher carbohydrate ($P = 0.01$), and also, the frequency of tooth brushing in this group was significantly higher than children with CHD ($P = 0.02$). According to Debris

Index, poor oral hygiene condition was not significantly higher in children with CHD in comparison with healthy children (17.2 ± 4.91 vs. 16.8 ± 4.19, $P = 0.09$). Although the DMFT score was higher in patients in comparison with healthy children, there was no significant difference (6.4 ± 2.46 vs. 5.5 ± 2.16; $P = 0.14$). Among study participants, 32.43% healthy children and 18.84% of the children with CHD experienced no or mild dental caries. According to gingival index, the children with congenital cardiovascular diseases experienced insignificantly higher periodontitis (2.34 ± 2.44 vs. 1.97 ± 2.33, $P = 0.2$). Only 30 (40.5%) healthy children and 21 (30.8%) children with CHD had no signs of periodontitis. In healthy children, the mean saliva colony counts of *S. mutans* were significantly higher (50639 ± 3324 vs. 35285 ± 27226, $P = 0.03$). Higher consumption of carbohydrate among children with CHD was responsible for this difference ($P = 0.07$). However, the mean colony count of Lactobacilli in children with CHD was insignificantly higher ($P = 0.3$). By adjusting the tooth brushing in the two groups, similar results were obtained. Tables 2 and 3 show that there is no significant correlation between the frequency of decay, missing, illed teeth and colony count of streptococcus mutans and lactobacilli.

Data in Tables 4 and 5 show no significant correlation between categorized saliva *S. mutans* and categorized saliva Lactobacilli and gingival bleeding index, respectively.

Table 1: Frequency of demographics variables among study participants

Data	Group	Mean±SD	*P*
Age (year)	Normal child	7.8±2.5	0.27
	CHD child	7.4±1.8	
Height (cm)	Normal child	126.3±15.8	0.26
	CHD child	123.6±12.6	
Weight (kg)	Normal child	25.1±11.6	0.08
	CHD child	22.3±7.8	
BMI (kg/m^2)	Normal child	15.08±3.4	0.056
	CHD child	14.08±2.6	
Ca (mg/dl)	Normal child	9.5±0.44	0.88
	CHD child	9.5±0.38	
pH (mg/dl)	Normal child	4.2±0.48	0.98
	CHD child	4.3±0.69	
Carbohydrate consumption	Normal child	121,928.65±51,724.70	0.01
	CHD child	102,977.15±33,960.36	
Protein consumption	Normal child	32,113.2±14,570.44	0.08
	CHD child	28,612.00±9241.73	

CHD=Congenital heart disease, BMI=Body mass index, Ca=Calcium, SD=Standard deviation

Discussion

Our data suggested no significantly higher saliva pathologic microorganism and also no significant correlation between saliva *S. mutans* and Lactobacilli and dental caries and periodontitis in the children with congenital cardiovascular diseases. Along with the present study findings, Balmar *et al*[29] and Hartzell[30] *et al* reported no more dental caries and bad oral hygiene in children with CHD. According

Table 2: Frequency of decayed, missing, and fi lled teeth among study participants according their streptococud mutans

Group	DMFT	Lactobacilli colony				Total	*P*
		<1000 (%)	1000-5000 (%)	5000-10,000 (%)	≥10,000 (%)		
Healthy child	<3	14 (50)	8 (28.6)	1 (28.6)	5 (17.9)	28 (100)	0.29
	≥3-7	8 (34.6)	11 (50)	2 (50)	1 (4.5)	22 (100)	
	>7	6 (24.0)	14 (56.0)	2 (56.0)	3 (12.0)	25 (100)	
CHD child	<3	6 (33.3)	10 (55.6)	-	2 (11.1)	18 (100)	
	≥3-7	9 (45)	9 (45)	-	2 (10)	20 (100)	
	>7	8 (32.0)	12 (48.0)	-	5 (20.0)	25 (100)	
Total		51 (36.96)	64 (46.38)	5 (3.62)	18 (13.04)	138 (100)	

DMFT=Decayed, missing, and filled teeth, CHD=Congenital heart disease

Table 3: Frequency of decayed, missing, and filled teeth among study participants according their Lactobacilli colony counts

Group	DMFT	Lactobacilli colony		Total (%)	*P*
		<10,000 (%)	10,000-100,000 (%)		
Healthy child	<3	15 (22.1)	53 (77.9)	68 (100)	0.19
	≥3-7	-	7 (100)	7 (100)	
	>7	-	-	-	
CHD child	<3	11 (20)	44 (80)	55 (100)	
	≥3-7	3 (50)	3 (50)	6 (100)	
	>7	1 (100)	-	1 (100)	
Total		30 (21.9%)	107 (78.1)	137 (100)	

DMFT=Decayed, missing, and filled teeth, CHD=Congenital heart disease

Table 4: Correlation of gingival bleeding and saliva *Streptococcus mutans* among healthy and congenital heart diseases child

Group	Gingival bleeding	Saliva *S. mutans*
Healthy child		
Gingival bleeding		
Correlation coefficient	1	0.03
P	-	0.83
Saliva *S. mutans*		
Correlation coefficient		1
P		-
CHD child		
Gingival bleeding		
Correlation coefficient	1	-0.07
P	-	0.58
Saliva *S. mutans*		
Correlation coefficient		1
P		-

S. mutans=*Streptococcus mutans*, CHD=Congenital heart disease

Table 5: Correlation of gingival bleeding and categorized saliva Lactobacilli among healthy and congenital heart diseases child

Group	Gingival bleeding	Categorized saliva Lactobacilli
Healthy child		
Gingival bleeding		
Correlation coefficient	1	0.18
P	-	0.12
Categorized saliva Lactobacilli		
Correlation coefficient		1
P		-
CHD child		
Gingival bleeding		
Correlation coefficient	1	−0.002
P	-	0.99
Categorized saliva Lactobacilli		
Correlation coefficient		1
P		-

CHD=Congenital heart disease

to these researchers, the dental care of children with CHD and knowledge of their parents about dental care and endocarditis influence children's oral hygiene. The patients of this study were enrolled from those referring heart clinics with special routine dental examination, which could partially explain our findings. On the other hand, the mean score of DMFT in healthy group was in moderate category of decayed tooth, showing that normal children may have poor dental hygiene and poor follow-up in our region in comparison with different parts of the world.[31,32]

Researchers demonstrated a correlation of saliva pathogenic microorganisms with dietary intake.[33,34] According to our results, healthy children use more carbohydrates, and also, *S. mutans* grows more significantly in their saliva. Investigations have shown the more growth rate of this organism is due to carbohydrate consumption in this group. This finding is in line with other studies.[35,36]

Our results could not show a significant correlation between higher Lactobacilli and *S. mutans* colony counts and DMFT in children with and without CHD. In studies of Gomar-Vercher *et al*.[37] and Hallett *et al*.,[17] saliva pathogenic microorganisms were not significantly correlated with dental caries and the researchers suggested other mechanisms other than saliva microorganisms which could be involved in tooth decay.[38]

We checked the calcium and phosphorus level of all participants, and all participants had normal ranges of these electrolytes. As a result, low calcium and phosphorous level could not correlate with moderate DMFT scores in both groups.

We used the saliva for microorganisms' colony counts. Umar *et al*.[24] found that in different age group, the saliva *S. mutans* colony counts had different reliability and correlation with decays, and it was lowest in adolescents and children. Therefore, our finding may be due to using saliva instead of dental caries microorganisms.

Periodontal diseases have been evaluated by different methods.[38,39] The gingival bleeding index was used in the present study as a tool for children periodontitis evaluation. Our findings show that the prevalence of periodontics is similar to that in other developing countries[40] but is higher than many countries in Latin America.[41] Cortelli *et al*. showed that the periodontitis was not associated with saliva microorganisms.[42] In line with our and Cortelli *et al*. results, Belstrom *et al*. confirmed that salivary microorganisms were not correlated with periodontitis.[43] Recently, immune-inflammatory host response has been introduced as the main cause of periodontal disease.[44] The factors which activate the pathway of arachidonic metabolism are currently known as the main cause of periodontitis other than pathogenic microorganisms;[45] therefore, as we evaluated the association of gingival index with *S. mutans* and Lactobacilli alone without considering other factors, our results could be justified.

Conclusions

Pediatric patients with CHD experience *in*significantly higher dental decay, periodontitis, and saliva Lactobacilli colony counts. The frequency of decayed tooth and gingival diseases in healthy children is high, and hence, more dental care attention in our health system is needed for healthy children.

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Conflicts of interest

There are no conflicts of interest.

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