Gingival Evaluation of the Pediatric Cardiac Patient

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Gingival Evaluation of the Pediatric Cardiac Patient

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Abstract: ***Purpose:*** *Children with congenital cardiovascular diseases (CCDs) who suffer from dental diseases have an increased risk of infective endocarditis. In the light of recent evidence, oral inflammatory diseases may also increase the severity of their cardiovascular condition. The purpose of this study was to evaluate the gingival status of children with congenital cardiovascular diseases in comparison to healthy children.* ***Methods:*** *Fifty 7- to 13-year-old children were included. The test group comprised 25 CCD children subdivided into three groups: (1) unrepaired ventricular septal defect; (2) aortic valve stenosis; and (3) coarctation of the aorta. The control group consisted of 25 healthy age- and gender-matched children. Gingivitis, plaque, calculus, and recession were measured on six sites per tooth on 12 teeth.* ***Results:*** *CCD children had significantly more gingivitis (P<.001), plaque (P<.001), recession (P>.02), and calculus (P<.001) than controls. Among the CCDs groups, no statistically significant differences were found for gingivitis, plaque, or recession.* ***Conclusions:*** *Children with congenital cardiovascular diseases had a higher prevalence of periodontal disease, evidenced by gingivitis, plaque, calculus, and recession. These children should be evaluated periodontally and their oral health monitored on a 3-month basis to prevent disease development, benefit cardiovascular condition, prevent endocarditis, and improve quality and longevity of life.* *(Pediatr Dent 2013;35:456-62) Received March 14, 2012 | Last Revision July 26, 2012 | Accepted July 27, 2012*

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The human organism is a single unit composed of a seemingly infinite number of biologic processes so intertwined that abnormalities of almost any of its parts or processes could have profound effects on other body areas.[1]

Today, a significant body of evidence supports the hypothesis that periodontal disease is associated with cardiovascular diseases,[2,3] and the results of many studies provide a number of biologically plausible mechanisms. The direct effect mechanism suggests that periodontal pathogens and their products result in damage to vascular endothelium. Monocytes and macrophages activated by periodontal inflammation enter vessel walls and produce cytokines that further increase inflammatory responses and propagate the atheromatous lesion. Growth factor production leads to smooth muscle proliferation in the vessel wall, and the damaged endothelium then activates platelet aggregation, potentiating a thromboembolic event. On the other hand, the indirect effect mechanism suggests that acute phase proteins, such as C-reactive protein (**CRP**) and fibrinogen, are produced in the liver in response to inflammatory or infectious stimuli. CRP induces monocytes and macrophages to produce tissue factors, which, in turn, stimulates the coagulation pathway and increases blood coagulability (with contribution of fibrinogen). This stimulates the complement cascade, further exacerbating inflammation.[4]

Recent clinical research has shown that moderate to severe periodontitis increases the level of systemic inflammation, a characteristic of all chronic inflammatory diseases, as measured by high sensitivity CRP, fibrinogen, and other biomarkers. At the same time, treatment of periodontal disease that is sufficient to reduce clinical signs of the disease decreases the level of systemic inflammatory mediators.[1,5,6]

Periodontal pathogens have been identified in atherosclerotic plaques, suggesting a role for these bacteria in early atherogenesis,[7] and periodontal disease has been suggested as a risk factor for coronary heart diseases that is independent of traditional risk factors, with relative risk estimates ranging from 1.24 to 1.34.[8] Therefore, poor oral hygiene, as the major cause of periodontal diseases, is associated with higher levels of risk of cardiovascular disease, low grade inflammation, and systemic inflammatory markers.[9]

In children, congenital cardiovascular diseases (**CCDs**) are among the most common developmental anomalies and are the leading cause of death from congenital malformations. Each year, at least nine out of every 1,000 infants born have CCDs. In the United States, approximately 36,000 children are born with a heart defect each year, and 1.3 million Americans alive today have some form of CCDs.[10]

The most common form of CCDs is ventricular septal defect (**VSD**), an opening in the ventricular septum, occurring in 50 percent of all children with congenital heart disease and in 20 percent as an isolated lesion. The incidence of VSD ranges from 1.56 to 53.2 per 1,000 live births.[11-16] Another form of CCDs is aortic valve stenosis (**AVS**), which results from fusion of the valve leaflets before birth. This stenosis causes mild to severe obstruction of the left ventricular outflow and accounts for seven percent of all congenital heart defects.[17] In addition, coarctation of the aorta (**COA**), which is typically in the region of the ligamentum arteriosum, is usually discrete but may be associated with diffuse hypoplasia of the aortic arch and isthmus. COA is more common in males, with a male-to-female ratio of 1.5:1, and is usually sporadic, but genetic influences can play a role.[11,18,19]

CCD children have less than optimal professional and home dental care and generally suffer poorer oral health than healthy children.[20,21] Almost half of the CCD children have never visited

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the dentist vs. only 35 percent of their healthy counterparts.[22] These children have also significantly more teeth with untreated dental caries [mean number of decayed, missing, and filled surfaces of teeth (dmfs) 5.2 vs. 2.2].[23] The greater bacterial load associated with high levels of dental plaque and gingivitis may put cardiac patients at unnecessary risk of developing bacterial endocarditis.[24] CCD children are also more likely to be colonized with specific HACEK (Hemophilus, Actinobacillus, Cardiobacter, Eikenella, Kingella) microbes than healthy children.[25]

CCD children require special care in dentistry because of their susceptibility to infective endocarditis from dental and periodontal infections. In addition, the heart condition may be worsened by the presence of periodontal disease, yet little information is available on the prevalence of gingival and periodontal disease in this population. To the best of our knowledge, no study has addressed the gingival evaluation of CCD children with specific cardiovascular conditions (VSD, AVS, and COA) and compared them with a matched control group.

The purposes of this study were to: (1) evaluate the gingival condition of children with congenital cardiovascular diseases within specific heart condition groups and compare them to a healthy matched control group; (2) determine if there is an association between CCDs and higher prevalence of gingivitis and periodontal disease; and (3) provide a basis for future research on the improvement of the oral health of CCD children.

Methods

The study protocol was approved by the Indiana University Institutional Review Board (approval no. 1007-84). The confidentiality of participants was strictly protected. In all cases, the procedures, possible discomforts or risks, and possible benefits were explained fully to the human subjects involved, and written parental informed consents and child assents were obtained.

This controlled cross-sectional analysis consisted of 50 7- to 13-year-olds; the test group children (N=25) were recruited from the Department of Pediatric Cardiology, Riley Hospital for Children, and the systemically healthy control group children (N=25) were recruited from the Department of Pediatric Dentistry, School of Dentistry, Indiana University, Indianapolis, Ind. This age range was selected to minimize the effect of permanent tooth eruption and before the onset of puberty.

The test group consisted of children with diagnosed acyanotic CCD and no other major systemic diseases. The group was divided into three (VSD, AVS, COA), and the following diagnoses and criteria were used for inclusion of patients: unrepaired ventricular septal defects of any size (VSD; N=SIX patients); unrepaired or residual aortic valve stenosis with a current peak gradient ≥30 mm of mercury, measured by echocardiography, with or without aortic insufficiency (AVS; N=14 patients); and unrepaired or residual coarctation of the aorta with a velocity by echocardiographic Doppler ≥2.5 m/second (COA; N=five patients).

Exclusion criteria for these patients included any genetic disorders or syndromes, significant neurological injury that may limit the ability of the patient to care for oneself, significant use of drugs, or any other significant disease beyond the cardiovascular system.

The control group consisted of 25 healthy children who had no systemic diseases, received treatment in a regular dental clinic, and matched the test group in age, race, gender, and socioeconomic status.

All oral health assessments were performed by one examiner. Before commencement of the study, however, the examiner was calibrated for repeatability and reproducibility in the use of the oral indices by a "gold standard" examiner, in line with previous studies.[26,27]

A 31-item validated questionnaire—which contained questions regarding the child's systemic health, child and parents' oral health, and parents' oral health education—was completed by the parents. The questions inquired specifically about the child's present health, past hospitalizations, diagnoses of chronic/severe medical conditions, last physical examination, and current medications. The parents were also asked about their and their child's brushing and flossing habits, last dental visit, last dental treatment, last dental cleaning, and whether the child received assistance with brushing. The parents also provided information about who taught them how to take care of the their child's teeth, who had the most influence on them to take their child to the dentist, who they think should teach parents about taking care of their children's teeth, the importance of following physician's instructions on the matter, and lastly, whether they believe there is a connection between their child's dental and general health. A complete medical history form was also completed by the parents.

The evaluations took place in a dental chair under standard lighting conditions. Since periodontal probing may cause bacteremia and, thus, carry a risk of infective endocarditis in CCD children, it was not performed. A mouth mirror, disclosing solution, and gauze were used for the examinations. Extraoral and intraoral soft tissue conditions were noted, and the following periodontal conditions were evaluated:

1. Gingivitis was measured by the modified gingival index (**MGI**).[28] This index was used because it avoids the periodontal probing of gingival pockets, makes it possible to detect early and more subtle visual changes in gingival inflammation, permits intra- and intercalibration of examiners, and is noninvasive upon repeated evaluations.
2. Dental plaque was assessed by the Turesky modified plaque index (**MPI**) using a disclosing solution.[29] Dental calculus was measured by the simplified calculus index (**CI-S**).[30]
3. Recession was evaluated by measuring the distance from the marginal gingiva to the cementoenamel junction in mm using a University of North Carolina (UNC) 15 periodontal probe (Hu-Friedy, Chicago, Ill., USA).

The gingival evaluation was performed on the following teeth on distobuccal, midbuccal, mesiobuccal, mesiolingual, midlingual, and distolingual sites: maxillary and mandibular permanent first molars or primary second molars; and maxillary and primary or permanent mandibular central and lateral incisors. A total of 12 teeth and six sites per tooth were evaluated in this study. These index teeth were chosen according to previously conducted studies in children[31-33] and using data recorded in patients attending the Department of Pediatric Dentistry, Indiana University, for routine care, which identified these teeth as having the highest prevalence of plaque and gingivitis.

Table 1. MEAN±(SD) FOR MGI, MPI, RECESSION, AND CI-S BY GROUP*

	Control (N=25)	CCDs-All (N=25)	CCDs-AVS (N=14)	CCDs-COA (N=5)	CCDs-VSD (N=6)
MGI	1.32±0.07	2.51±0.09	2.51±0.09	2.34±0.25	2.67±0.21
MPI	0.95±0.10	2.73±0.14	2.65±0.17	2.31±0.33	3.27±0.25
Recession	0.00±0.00	0.01±0.01	0.02±0.01	0.01±0.01	0.00±0.00
CI-S	0.01±0.01	0.15±0.03	0.14±0.03	0.03±0.02	0.26±0.05

* MGI=modified gingival index; MPI=modified plaque index; CI-S=calculus index; CCDs-All=total no. of children with congenital cardiovascular diseases; CCDs-AVS= aortic valve stenosis group; CCDs-COA=coarctation of the aorta group; CCDs-VSD= ventricular septal defect group.

Statistical methods

The MPI, MGI, CI-S, and mean recession were calculated for each subject by averaging across all assessed sites. Summary statistics were calculated by group. Comparisons between the CCD children and control group were performed using 2-sample *t* tests for MPI and MGI and using nonparametric Wilcoxon rank sum tests for CI-S and recession. Additional comparisons among the three CCD diagnosis groups were performed using analysis of variance (**ANOVA**) for MPI and MGI and using Kruskal-Wallis and Wilcoxon rank sum tests for CI-S and recession.

Comparisons between males and females for differences in MGI and MPI were made using 2-way ANOVA, with factors for group, sex, and sex-by-group interaction. Comparisons for recession and calculus were made using Wilcoxon rank sum tests. Questionnaire responses were summarized by group. Comparisons between the CCD children and the control group and among the three CCDs diagnoses were performed using Pearson's chi-square tests for nonordered categorical variables, Mantel-Haenszel chi-square tests for ordered categorical variables, and Wilcoxon rank sum tests for parent and child ages.

Results

This study enrolled 50 participants of a mean age of 10.5±0.4 years old, with 52 percent of participants being male and 48 percent being female. The test group consisted of 64 percent males and 36 percent females, and the control group consisted of 40 percent males and 60 percent females. CCD children had significantly higher MGI (*P*<.001), MPI (*P*<.001), recession (*P*>.02), and CI-S (*P*<.001) than the control group (Table 1, Figures 1-5). Among the three CCD diagnosis groups, VSD had significantly higher CI-S than COA (*P*>.01), with AVS not significantly different from COA (*P*=.08) or VSD (*P*=.06).

No statistically significant differences were found among the three CCDs groups for MGI (*P*=.45), MPI (*P*=.06), or recession (*P*=.36). All subjects had at least one site with MGI and MPI greater than zero; and 20 percent of CCD subjects and zero percent of control subjects had at least one site with recession greater than zero; 76 percent of CCDs subjects and 24 percent of control subjects had at least one site with CI-S greater than zero.

The correlation between plaque and gingivitis overall was 0.89. Within the control group and CCD group, the correlations were 0.70 and 0.59, respectively. The correlations for the two groups were not significantly different (*P*=.53). No gender differences were seen for any of the indices.

Regarding analysis of the questionnaire results, we observed that CCD children, compared to control group children, had fewer younger siblings (*P*>.01), a lower percentage of good health (*P*<.01), and a higher percentage of hospitalizations (*P*<.01), chro-nic disease (*P*<.01), and medications (*P*<.02). CCD children also had more recent dental cleanings (*P*>.04) vs healthy group children.

When comparing children in the CCD subgroups, those with CCDs-VSD received help brushing more frequently than patients with CCDs-AVS (*P*<.05). CCDs-COA had a lower percentage than CCDs-AVS (*P*<.04) that believed there is a dental/general health connection. CCD children had higher percentages of their parent's dentist (*P*>.04), pediatrician (*P*>.02), and cardiologist (*P*<.04) influencing them to take the child to the dentist vs the control group (Table 2). Parents of CCD children had received more recent dental checkups, dental cleaning, and dental treatment and had better oral hygiene habits than their counterpart parents. Those parents also had higher percentages believing the pediatrician (*P*<.01) and pediatric cardiologist (*P*<.02) should tell the parent about taking care of the child's teeth (Table 3).

Discussion

In the present study, it was found that CCD children have significantly higher levels of gingivitis, plaque, recession, and calculus than healthy control children. These findings are consistent with previously published studies that found the oral hygiene of CCD children to be poor.[20,21,24] In contrast to other studies, we focused on gingival health with three distinct cardiac groups and found that parents and their CCD children had more encouragement from the parent's dentist, pediatrician, and pediatric cardiologist to take the child to a dentist and also had a more recent dental cleaning and checkup vs the healthy group. Despite this, the inflammation and oral hygiene indices of these CCD children demonstrated an overall less healthy condition than the systemically healthy control group.

These results may be due to several factors.[22] First, CCD children have less-than-adequate oral hygiene by brushing and flossing less regularly than their healthy counterparts. In our

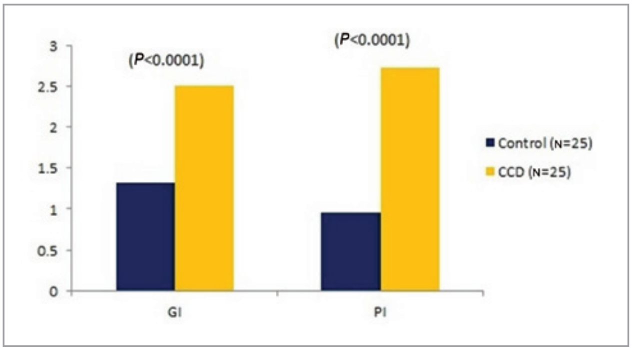

Figure 1. Mean for gingivitis and plaque by group. GI- Gingival index, PI-Plaque index, CCD- Total number of children with congenital cardiovascular diseases

Figure 2. Gingivitis measurement in a control patient. Figure 3. Gingivitis measurement in a test patient. Figure 4. Plaque measurement in a control patient using disclosure solution. Figure 5. Plaque measurement in a test patient using disclosure solution.

Table 2. QUESTIONNAIRE RESPONSES*

Who had the most influence on you to take your child to the dentist?		Control	CCDs	CCDs-AVS	CCDs-COA	CCDs-VSD	*P*-value	
							Control vs. CCDs	Among CCDs groups
Parent's dentist N (%)	No	24 (96)	19 (76)	11 (79)	4 (80)	4 (67)	>.04	.83
	Yes	1 (4)	6 (24)	3 (21)	1 (20)	2 (33)		
Child's dentist N (%)	No	22 (88)	17 (68)	9 (64)	5 (100)	3 (50)	.09	.19
	Yes	3 (12)	8 (32)	5 (36)	0 (0)	3 (50)		
Child's pediatrician N (%)	No	24 (96)	18 (72)	12 (86)	4 (80)	2 (33)	>.02	.05
	Yes	1 (4)	7 (28)	2 (14)	1 (20)	4 (67)		
Child's pediatric cardiologist N (%)	No	25 (100)	21 (84)	13 (93)	4 (80)	4 (67)	<.04	.33
	Yes	0 (0)	4 (16)	1 (7)	1 (20)	2 (33)		

*CCDs=total no. of children with congenital cardiovascular diseases; CCDs-AVS=aortic valve stenosis group; CCDs-COA=coarctation of the aorta group; CCDs-VSD= ventricular septal defect group.

study, only 48 percent of the CCD children brushed two times or more a day vs. 60 percent of the healthy group. de Oliveira et al.[9] found in adults that self-reported frequency of tooth-brushing is associated with cardiovascular disease risk, even after adjusting for age, sex, socioeconomic group, smoking, visits to the dentist, body mass index, family history of cardiovascular disease, hypertension, diagnosis of diabetes, C-reactive protein, and fibrinogen. In fully adjusted models, participants who brushed their teeth less regularly had a 70 percent increased risk of a cardiovascular disease event. This supports the relevance of our study data and the importance of oral hygiene in relation to the disease severity of CCD children.

Secondly, there is lack of oral hygiene following frequent ingestion of heavily sweetened medications and nutritional supplements.[34] This increases the number of carious lesions in patient's teeth, which may also serve as plaque reservoirs and cause gingival and, possibly, periodontal inflammation.

Thirdly, some CCD children take medications containing sucrose and diuretics, which can cause xerostomia and, therefore, predispose to gingivitis and periodontal diseases.[23,35,36] In addition, children with congenitally malformed hearts have a reduced oral health-related quality of life in some specific domains, including family stress and the psychological impact of the disease on the child that mitigates against establishing an adequate oral hygiene ritual that may directly and indirectly increase local and systemic inflammation.[22] It has been shown that CCD children have a different oral microflora than healthy children, independent of the degree of gingival inflammation, and that they have more severe gingival inflammatory conditions than age- and sex-matched healthy children.[25]

Finally the systemic condition of CCD children may have an impact on their periodontal status. In recent years, increasing evidence has supported the concept that the relationship between systemic and oral health is bidirectional,[37] and it has long been recognized that systemic conditions can contribute to the expression of periodontal disease. Additionally, metabolic disorders (diabetes), blood dyscrasias (leukemia), autoimmune disease (pemphigus), pregnancy, and puberty may all increase oral soft tissue inflammation and the incidence of periodontal disease.[4]

da Fonseca et al.[22] found that 20 percent of the parents of children with cardiac disease, vs. 10 percent of controls, were unsure of the relationship between oral and cardiac health. In our study, only 12 percent of the parents in the test and control groups were unsure of that relationship, with 84 percent of the parents in both groups believing that a strong association exists.

In this study, parental supervision of tooth-brushing was similar between the two groups, in contrast to other studies[24,38]; furthermore, nearly 90 percent of the patients in both groups were not helped with oral hygiene procedures, regardless of the differences in dental visits. This may be attributed to the relatively greater age of the patients who participated in our study (mean= 10.5±0.4 years old), since most of the children at this age who

Table 3. QUESTIONNAIRE RESPONSES

Who do you think should tell you about taking care of your child's teeth?		Control	CCDs	CCDs-AVS	CCDs-COA	CCDs-VSD	*P*-values	
							Control vs. CCDs	Among CCDs groups
Child's pediatrician N (%)	No	19 (76)	10 (40)	7 (50)	0 (0)	3 (50)	<.001	.12
	Yes	6 (24)	15 (60)	7 (50)	5 (100)	3 (50)		
Child's pediatric cardiologist N (%)	No	23 (92)	16 (64)	9 (64)	3 (60)	4 (67)	<.02	.97
	Yes	2 (8)	9 (36)	5 (36)	2 (40)	2 (33)		

* MGI=modified gingival index; MPI=modified plaque index; CI-S=calculus index; CCDs-All=total no. of children with congenital cardiovascular diseases; CCDs-AVS=aortic valve stenosis group; CCDs-COA=coarctation of the aorta group; CCDs-VSD=ventricular septal defect group.

are not mentally or physically handicapped are able to practice oral hygiene effectively without adult assistance or supervision.

A greater proportion of healthy children had received dental treatment in the previous six months than the CCD children, even though the CCD children had more recent dental checkups. There may be an unjustified reticence in the general dental community toward treating CCD children, and, in most cases, these patients are referred to a specialist. This, in turn, raises the cost of treatments and may prevent the parents of CCD children from seeking active treatment. Flossing of teeth was infrequent in both groups, and this important oral hygiene regimen could be improved, especially in the CCDs group.

Since the parents' oral health behavior can significantly influence that of the child, we decided to examine the child's home attitude to dental care. We found that parents of CCD children had received more recent dental checkups, dental cleaning, and dental treatment and had better oral hygiene habits than their counterpart parents. This may explain the more recent dental cleaning and dental checkups that the CCD children had experienced.

When looking at the cardiac subgroups, it should be noted that we chose the three most prevalent CCDs conditions in our hospital that would be acyanotic and nondebilitating (while reflecting the situation in the CCDs population) and yet be at a high level of severity with strict inclusion criteria. To reduce confounding factors, we excluded any genetic disorders or syndromes, significant neurological injury, significant use of drugs, and any other major disease beyond the cardiovascular system.

Among the three CCDs diagnosis groups, VSD patients had significantly higher calculus scores than COA patients and yet received help brushing more frequently than AVS patients. The COA group had a lower percentage of parents believing there is a dental/general health connection than the AVS group. Most of the differences (MPI, MGI, recession) between the three groups were nonsignificant, however, which emphasizes the overall inferior oral health status of the CCD children vs the healthy group.

CCD patients had significantly fewer younger siblings than their healthy counterparts. It may be that the parents of CCD children are preoccupied with their child, and his/her disease consumes their emotional and physical resources and affects the decision of having another child.

Regarding receiving professional advice about the importance of dental care, nearly 70 percent of both the control and test groups were encouraged by the child's dentist. Only 20 percent of the parents of CCD children, however, claimed that the pediatric cardiologist suggested they take care of their child's teeth, and only 16 percent claimed that the pediatric cardiologist was the one who influenced them to take their child to the dentist (Table 2). When the parents were asked who should inform them about taking care of their child's teeth, both groups believed that it should be the dentist. In the CCD group, only 36 percent believed that the pediatric cardiologist should be the one (Table 3).

Nevertheless, the data overall suggest that there may be a need for pediatric cardiologists to better understand the importance of oral health in their patients so that they can assume a major role in reinforcing this to parents and patients.

Gingivitis is an inflammatory response to the accumulation of dental plaque and may progress to periodontal disease. It has also been suggested that long-term, low-grade infectious challenges, like chronic gingival inflammation, may be of greater systemic importance than isolated, clinically obvious events, such as tooth extraction.[39] Franek et al.[40] found that left ventricular mass was higher in subjects with type 2 diabetes and concomitant gingivitis vs subjects with type 2 diabetes and periodontal health. In addition, experimental gingivitis was associated with endotoxemia and hyperactivity of circulating neutrophils.[41] Therefore, gingivitis and plaque can trigger a major systemic response.

There have been major breakthroughs in the understanding of inherited causes of CCDs, including the identification of specific genetic abnormalities for some types of malformations.[42] There remain, however, unexplored areas in understanding of these disorders and their influence on systemic and periodontal health.

Based on the emerging evidence of association between periodontal and systemic conditions,[43] a high level of oral care is recommended for CCD children, since neglect can worsen their already delicate cardiovascular condition and trigger infective endocarditis. The American Academy of Pediatric Dentistry recommends maintenance appointments every six months, or as indicated by the individual patient's risk status/susceptibility to disease.[44] Many patients presenting with recurrent gingivitis without additional attachment loss may be adequately maintained with maintenance performed semiannually. Numerous clinical studies suggest, however, that maintenance should be performed at intervals of fewer than six months at least four times per year, since that interval will result in a decreased likelihood of progressive disease vs. patients receiving maintenance on a less-frequent basis.[45]

Establishing a higher prevalence of gingival inflammation in CCD children can lead to increased awareness of the need for improved periodontal condition in these children, which can potentially benefit their cardiovascular condition, prevent endocarditis, and improve quality and longevity of life. An active collaboration should be formed between the pediatric dentist, periodontist, pediatrician, and pediatric cardiologist of CCD children.

Additionally, the American Heart Association has concluded that observational studies to date support an association between periodontal disease and cardiovascular disease, independent of shared risk factors. While current research does not yet provide evidence of a causal relationship between the two diseases, scientists have identified biologic factors, such as chronic inflammation, that independently link periodontal disease to the development or progression of cardiovascular disease in patients.[46] The lack of causal evidence should not diminish concern about the impact of periodontal status on cardiovascular health, since long-term interventional studies are needed to better understand the specific nature of the relationship between periodontal disease and cardiovascular disease.[47]

This study has some limitations. The examiner was not "blind" as to whether the subjects' were in the test or control group, since they were examined in either the Cardiac Department of Riley Children's Hospital or the pediatric clinic of the School of Dentistry, Indiana University; however, the examiner was "blind" to the subgroup of heart disease. No comprehensive periodontal examination was performed, as pocket probing can induce bleeding, however slight, which would have necessitated providing antibiotic premedication of the CCD children. Radiographic examination of alveolar bone levels would have been valuable in confirming the state of tooth support, but would have exposed the study participants to unwarranted radiation. Microbiological sampling of dental plaque may have confirmed differences in potentially significant infection reservoirs, and could be the subject of future study.

Conclusions

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

1. Children with congenital cardiovascular diseases had a significantly higher prevalence of periodontal disease, evidenced by gingivitis, plaque, calculus, and recession.
2. Among the CCD groups, no statistically significant differences were found for gingivitis, plaque, or recession.

3. CCD children should receive focused oral hygiene education, be evaluated periodontally, and their oral health monitored at least once every three months.

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References

1. Friedewald VE, Kornman KS, Beck JD, et al. The American Journal of Cardiology and Journal of Periodontology Editors' Consensus: Periodontitis and atherosclerotic cardiovascular disease. Am J Cardiol 2009;104:59-68.
2. Amabile N, Susini G, Pettenati-Soubayroux I, et al. Severity of periodontal disease correlates to inflammatory systemic status and independently predicts the presence and angiographic extent of stable coronary artery disease. J Intern Med 2008;263:644-52.
3. Mustapha IZ, Debrey S, Oladubu M, Ugarte R. Markers of systemic bacterial exposure in periodontal disease and cardiovascular disease risk: A systematic review and meta-analysis. J Periodontol 2007;78:2289-302.
4. Newman MTH, Klokkevold P, Carranza F. Carranza's Clinical Periodontology. 10th ed. Philadelphia, Pa: Saunders Elsevier; 1996:312-29.
5. Paraskevas S, Huizinga JD, Loos BG. A systematic review and meta-analyses on C-reactive protein in relation to periodontitis. J Clin Periodontol 2008;35:277-90.
6. Tonetti MS, D'Aiuto F, Nibali L, et al. Treatment of periodontitis and endothelial function. N Engl J Med 2007;356: 911-20.
7. Haraszthy VI, Zambon JJ, Trevisan M, Zeid M, Genco RJ. Identification of periodontal pathogens in atheromatous plaques. J Periodontol 2000;71:1554-60.
8. Humphrey LL, Fu R, Buckley DI, Freeman M, Helfand M. Periodontal disease and coronary heart disease incidence: A systematic review and meta-analysis. J Gen Intern Med 2008; 23:2079-86.
9. de Oliveira C, Watt R, Hamer M. Toothbrushing, inflammation, and risk of cardiovascular disease: Results from Scottish Health Survey. BMJ 2010;27:1-6.
10. American Heart Association. Introduction to congenital heart defects. Available at: "http://www.heart.org". Accessed August 11, 2013.
11. Fyler DCKJ, Lock J. Ventricular septal defect. In: Fyler DC, ed. Nadas' Pediatric Cardiology. 2nd ed: Philadelphia, Pa: Hanley and Delfus, Inc; 1992:435-57.
12. Moller JH, Moodie DS, Blees M, Norton JB, Nouri S. Symptomatic heart disease in infants: Comparison of three studies performed during 1969-1987. Pediatr Cardiol 1995;16: 216-22.
13. EB Clark. Etiology of congenital cardiovascular malformations In: Allen HD, Gutgesell HP, Clark EB, Driscoll DJ, ed. Moss and Adams' Heart Disease in Infants, Children, and Adolescents Including the Fetus and Young Adult. Philadelphia, Pa: Lippincott Williams and Wilkins; 2001:64-79.
14. Ooshima A, Fukushige J, Ueda K. Incidence of structural cardiac disorders in neonates: An evaluation by color Doppler echocardiography and the results of a 1-year follow-up. Cardiology 1995;86:402-6.
15. Roguin N, Du ZD, Barak M, Nasser N, Hershkowitz S, Milgram E. High prevalence of muscular ventricular septal defect in neonates. J Am Coll Cardiol 1995;26:1545-8.
16. Minette MS, Sahn DJ. Ventricular septal defects. Circulation 2006;114:2190-7.
17. Crawford MH. Current Diagnosis and Treatment in Cardiology. 2nd ed. New York, NY: The McGraw-Hill Companies, Inc; 2003:108.
18. Ferencz C, Rubin JD, McCarter RJ, et al. Congenital heart disease: Prevalence at live birth. The Baltimore-Washington Infant Study. Am J Epidemiol 1985;121:31-6.
19. Silversides CK, Kiess M, Beauchesne L, Bradley T, Connelly M, Niwa K, et al. Canadian Cardiovascular Society 2009 Consensus Conference on the management of adults with congenital heart disease: Outflow tract obstruction, coarctation of the aorta, tetralogy of Fallot, Ebstein anomaly, and Marfan's syndrome. Can J Cardiol 2010;26:e80-e97.
20. Rai K, Supriya S, Hegde AM. Oral health status of children with congenital heart disease and the awareness, attitude, and knowledge of their parents. J Clin Pediatr Dent 2009;33: 315-8.
21. Balmer R, Bu'Lock FA. The experiences with oral health and dental prevention of children with congenital heart disease. Cardiol Young 2003;13:439-43.
22. da Fonseca MA, Evans M, Teske D, Thikkurissy S, Amini H. The impact of oral health on the quality of life of young patients with congenital cardiac disease. Cardiol Young 2009; 19:252-6.
23. Stecksen-Blicks C, Rydberg A, Nyman L, Asplund S, Svanberg C. Dental caries experience in children with congenital heart disease: A case-control study. Int J Paediatr Dent 2004;14: 94-100.
24. Franco E, Saunders CP, Roberts GJ, Suwanprasit A. Dental disease, caries-related microflora, and salivary IgA of children with severe congenital cardiac disease: An epidemiological and oral microbial survey. Pediatr Dent 1996;18:228-35.
25. Steelman R, Einzig S, Balian A, et al. Increased susceptibility to gingival colonization by specific HACEK microbes in children with congenital heart disease. J Clin Pediatr Dent 2000;25:91-4.
26. Owens JD, Dowsett SA, Eckert GJ, Zero DT, Kowolik MJ. Partial-mouth assessment of periodontal disease in an adult population of the United States. J Periodontol 2003;74: 1206-13.
27. Wahaidi VY, Dowsett SA, Eckert GJ, Kowolik MJ. Neutrophil response to dental plaque by gender and race. J Dent Res 2009;88:709-14.
28. Lobene RR, Weatherford T, Ross NM, Lamm RA, Menaker L. A modified gingival index for use in clinical trials. Clin Prev Dent 1986;8:3-6.
29. Turesky S, Gilmore ND, Glickman I. Reduced plaque formation by the chloromethyl analogue of victamine C. J Periodontol 1970;41:41-3.
30. Greene JC, Vermillion JR. The simplified oral hygiene index. J Am Dent Assoc 1964;68:7-13.
31. Davidovich E, Schwarz Z, Davidovitch M, Eidelman E, Bimstein E. Oral findings and periodontal status in children, adolescents and young adults suffering from renal failure. J Clin Periodontol 2005;32:1076-82.
32. Hugoson A, Koch G, Rylander H. Prevalence and distribution of gingivitis-periodontitis in children and adolescents: Epidemiological data as a base for risk group selection. Swed Dent J 1981;5:91-103.

33. Guare Rde O, Ciampioni AL. Prevalence of periodontal disease in the primary dentition of children with cerebral palsy. J Dent Child 2004;71:27-32.
34. Bigeard L. The role of medication and sugars in pediatric dental patients. Dent Clin North Am 2000;44:443-56.
35. Farsi N, Al Amoudi N, Farsi J, Bokhary S, Sonbul H. Periodontal health and its relationship with salivary factors among different age groups in a Saudi population. Oral Health P rev Dent 2008;6:147-54.
36. Celenligil-Nazliel H, Palali A, Ayhan A, Ruacan S. Analysis of in situ proliferative activity in oral gingival epithelium in patients with xerostomia. J Periodontol 2003;74:247-54.
37. Williams RC, Offenbacher S. Periodontal medicine: The emergence of a new branch of periodontology. Periodontol 2000;23:9-12.
38. da Silva DB, Souza IP, Cunha MC. Knowledge, attitudes, and status of oral health in children at risk for infective endocarditis. Int J Paediatr Dent 2002;12:124-31.
39. Roberts GJ. Dentists are innocent! "Everyday" bacteremia is the real culprit: A review and assessment of the evidence that dental surgical procedures are a principal cause of bacterial endocarditis in children. Pediatr Cardiol 1999;20:317-25.
40. Franek E, Napora M, Blach A, et al. Blood pressure and left ventricular mass in subjects with type 2 diabetes and gingivitis or chronic periodontitis. J Clin Periodontol 2010;37:875-80.
41. Wahaidi VY, Kowolik MJ, Eckert GJ, Galli DM. Endotoxemia and the host systemic response during experimental gingivitis. J Clin Periodontol 2011;38:412-7.
42. Jenkins KJ, Correa A, Feinstein JA, et al. Noninherited risk factors and congenital cardiovascular defects. Current knowledge: A scientific statement from the American Heart Association Council on Cardiovascular Disease in the Young. Endorsed by the American Academy of Pediatrics. Circulation 2007;115:2995-3014.
43. Ridker PM, Silvertown JD. Inflammation, C-reactive protein, and atherothrombosis. J Periodontol 2008;79(8 suppl):1544-51.
44. American Academy of Pediatric Dentistry. Guideline on periodicity of examination, preventive dental services, anticipatory guidance/counseling, and oral treatment for infants, children, and adolescents. Reference Manual 2008-2009. Pediatr Dent 2008;30(suppl):112-8.
45. Cohen RE. Position paper: Periodontal maintenance. J Periodontol 2003;74:1395-401.
46. Lockhart PB, Bolger AF, Papapanou PN, et al. Periodontal disease and atherosclerotic vascular disease: Does the evidence support an independent association? A scientific statement from the American Heart Association. Circulation 2012;125:2520-44.
47. American Academy of Periodontology. Periodontal disease linked to cardiovascular disease. Available at: "www.perio.org". Accessed August 11, 2013.

Abstract of the Scientific Literature

Adolescent smoking and their use of smokeless tobacco

The purpose of the study was to determine the use of smokeless tobacco and smoking among adolescents participating in the 2011 national Youth Risk Behavior Surveillance System (YRBSS) survey. A total of 15,425 adolescents participated in the cross-sectional YRBSS survey. Data for the purpose of this study on youth smoking was restricted to those who provided responses on smoking and tobacco use (9,655). The sampling frame included children in grades nine through 12 from all states. Smokeless tobacco (chewing tobacco, snuff, or dip) use was defined as any use within the past 30 days. Likewise, smoking was defined as any cigarette use in the past 30 days. Other demographic, lifestyle, and risk-taking behavior variables were collected as part of the survey. Overall, 6.2 percent of respondents reported to be users of smokeless tobacco, while 16.8 percent smoked cigarettes. Children who used smokeless tobacco were at significantly greater odds of smoking cigarettes (unadjusted odds ratio=9.7). After adjusting for demographic, lifestyle, and risk-taking behaviors, the association was still significant between smokeless tobacco use and smoking on multiple regression analysis (odds ratio = 3.9). Those who used smokeless tobacco tended to be male, Caucasian, overweight, and engage in high-risk behaviors. This study provides strong evidence of an association between youth smoking and smokeless tobacco use.

Comments: *The dental profession is uniquely situated to provide smoking and tobacco cessation counselling to adolescents. Dental team members should ask youth in their practices about tobacco use. It is important to highlight the negative consequences that tobacco use can have on the oral cavity, including oral cancer, periodontal disease, and caries.* ***RJS***

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Wiener RC. Association of smokeless tobacco use and smoking in adolescents in the United States: An analysis of data from the Youth Risk Behavior Surveillance System survey, 2011. J Am Dent Assoc 2013 Aug;144(8):930-8.

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