DENTAL CARIES PREVALENCE IN CHILDREN AND YOUTHS WITH VERTICALLY-TRANSMITTED HIV/AIDS FROM THE PEDIATRIC HIV CLINIC (CALI, COLOMBIA) AND ITS RELATIONSHIP WITH BIOLOGICAL FACTORS, 2013[1]

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ABSTRACT. Introduction: *among the oral pathologies afflicting children with HIV/AIDS is dental caries, a preventable disease if detected and controlled in its early stages to avoid further complications. The aim of this study was to determine the prevalence of caries in children and youths with vertically-transmitted HIV/AIDS from the Pediatric HIV Clinic in Cali, Colombia, in 2013, and to explore relationships with biological factors.* ***Methods:*** *descriptive prevalence study in 101 clinical records of children and youths aged 1 to 17 years with vertically-transmitted HIV/AIDS from the Pediatric HIV Clinic in Cali. The ICDAS and DMF/def classification systems were used. Multivariate analysis and confounding variable adjustment were included to explore relationships with demographic, paraclinical, nutritional, and dental factors. The institutional ethics committees endorsed the study.* ***Results:*** *the prevalence of caries experience in children with HIV was 34.65% DMF/deft $_{(5-6)}$ 3.29 ± 3.06. This prevalence increases 83.17% with the ICDAS $_{2-6}$ system. The point prevalence of active caries was 74.26% DMFT 5.68 ± 5.48. No statistically significant association was found with any socioeconomic or immunologic variables. Patients with moderate to severe immunosuppression had OR 1.13 CI95% (0.33-3.81) p = 0.84. A probable association was found with plaque index OR 4.58 CI95% (1.44-14.55) p = 0.006 and caries experience OR 4.21 CI95% (1,09-16.13)* ***Conclusion:*** *HIV patients from the Pediatric Clinic show high caries prevalence when pre-cavitated lesions are assessed. No probable association was found between caries and immunological or clinical status, and therefore this aspect is not an additional risk factor.*

Key words*: dental caries, HIV/AIDS, epidemiology*

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INTRODUCTION

The human immunodeficiency virus (HIV) and acquired immunodeficiency syndrome (AIDS) are priorities in public health worldwide and their treatment is costly.[1, 2] In Colombia, 84 new HIV cases were identified in patients under 15 years of age by the year 2011.[3] According to some estimates, in 2011 there were 120 diagnosed cases of children under 14 (113 of them by vertical transmission), who were treated at the HIV program of the Pediatric Clinic of Cali, in southwestern Colombia.[4]

HIV patients' systemic compromise may be aggravated by opportunistic diseases, including oral lesions and tooth decay.[5] Therefore, these alterations should be diagnosed early and timely.

To diagnose tooth decay and make international comparisons, the WHO recommends using the def/DMF index,[6] which helps diagnose advanced caries in deciduous and permanent dentition respectively. On the other hand, classification systems are currently used to assess carious lesions from early stages,[7] including the International Caries Detection and Assessment System (ICDAS),[8-12] which was used in Colombia in the latest *Estudio Nacional de Salud Bucal ENSAB IV (2014)*, reporting caries experiences of 80 to 90% and caries prevalence of 75 to 85% in kids aged 3 to 18 years.[13]

Studies on caries in children with HIV worldwide are scarce; the reported caries values range from 50 to 80% and are generally disregarded,[14-17] making the population of children with HIV even more vulnerable because of lack of knowledge on the pathology and due to systemic conditions and local alterations related to the disease, causing a greater number of carious lesions.[18-19] The prevalence of caries reported in children with HIV depends on factors like type of caries classification system,[20] dentition type, and the studies' samples and objectives.[21-23]

In Colombia, there are few studies focusing on caries in HIV patients, and their objectives range from analyzing oral lesions[16] and describing oral health status,[17] to assessing the use of antiretroviral therapy,[24] but none has explored the risk factors associated with tooth decay.

HIV patients are at risk of caries because of their systemic compromise. This risk can arise for different reasons: 1) an alteration in the immune response to aggressor agents—including bacteria—, which promote the development of caries;[25] 2) the patient's immune status (CD4 count and viral load), the infiltration of the HIV virus and the proliferation of CD8 lymphocytes in salivary glands,[26] along with the use of HAART therapy,[27] decrease salivary flow (partially or totally), leading to alterations in saliva composition and modifying the normal condition of flora in the oral cavity, preventing saliva from performing its protective functions of clogging, clearing, mineral balancing, and sweeping of bacterial plaque, with the consequent appearance of dental caries; 3) retentive factors, like enamel structure defects, promote the production of mature plaque,[21] leading to the permanence of multiple bacteria;[28] 4) the nutritional status of patients acts as a predisposing factor to enamel defects in permanent dentition (systemic dietary factor);[29] 5) the use of HAART therapy with high concentrations of glucose acts as a substrate[21] for *Streptococcus mutans* and its ability to metabolize acids and produce cariogenic activity, mainly regulated by local dietary factors, its daily frequency in both foods and beverages, and the number of daily intake of carbohydrates;[30-34] 6) the social condition and vulnerability of these patients are closely linked to high levels of social marginalization and limited access to health programs and institutions.[35]

Globally, the studies exploring risk factors for caries in patients with pediatric HIV link the presence of caries to gingival inflammation, CD4/CD8 ratio < 0.5 ($p = 0.0026$),[36] and viral load,[25-37] but they have not found differences with not systemically-compromised children.[38]

Dental caries studies generally establish the prevalence of active caries (i.e., patients with active carious lesions or fillings at the time of the study), which corresponds to a point prevalence—or the "probability for an individual of a population to have the disease" at time *t*.[39-41] Dental caries experience is usually described as the number of patients with active caries, filled or missing teeth due to caries at the time of the study, which corresponds to a type of lifetime prevalence.

Considering the limited availability of studies with a strict methodology allowing to quantify the prevalence of caries in HIV patients in the pediatric population, the present study aims to determine caries prevalence and possible related factors in children and youths aged 1 to 17 years with HIV treated at the Pediatric HIV Clinic in Cali, Colombia, in 2013. Thus, the study estimated different types of prevalence according to the severity of dental caries.

MATERIALS AND METHODS

This research project was approved by the ethics committees of Universidad del Valle and the Pediatric HIV Clinic in Cali, Colombia (Clínica Pediátrica del VIH). According to Resolution 8430 of 1993, this is considered a "risk-free" study based on documental sources, like clinical records. The study complied with the principles of total confidentiality of the participants' identities. The institution granted access to the clinical records, safeguarding the confidentiality and legal reservation, in accordance with the Constitution, the laws, and the Habeas Data regulations in force in our country. This study was financed by an internal call for projects of Universidad del Valle.

A descriptive, transversal study on dental caries prevalence was conducted in 101 patients aged 1 to 17 years diagnosed with HIV/AIDS, registered in 2013 in the database of the Pediatric HIV Clinic in Cali, Colombia, with proper control and treatment. In this study, sampling frame and sample size were the same, as they correspond to the entire population enrolled in the Pediatric HIV Patient Care Program at Valle del Cauca in 2013, with vertically-transmitted HIV and full clinical, dental, and nutritional records.

The Pediatric HIV Clinic of Cali assesses and offers medical control to all children diagnosed with HIV/AIDS in the city. This control consists of valuation by each specialist; paraclinical tests every three months in the same laboratory; nutritional survey (24-hour count), offering data on food consumption and quantity of daily sugars; psychological assessment or monthly control of treatment adherence; assessment of growth, height, nutrition, and vaccination, as well as immunological control. In addition, it provides care by two pediatric dentists, who enter the information under the clinic's dental care protocols. The population treated at this clinic includes children living with their parents or guardians and those living in the Fundamor foundation, where they receive treatment and medical and dental assessment.

The inclusion criteria considered clinical records of children and youths aged 1 to 17 years who acquired HIV by vertical transmission and were diagnosed at birth by two positive viral loads after four months, and children over 18 months with at least two ELISA tests and Western Blot confirmatory test. As exclusion criteria, the clinical records of HIV patients who acquired the disease by reasons other than vertical transmission were discarded, as well as clinical records with incomplete dental data.

The socio-demographic variables and the initial clinical and immunological classification were obtained from the clinic's database. Variables related to current immune status (viral load and CD4 percentage) were taken directly from the latest lab tests, prior to dental diagnosis, and were entered on the clinical records by a physician. The time elapsed between these tests and the dental diagnosis was not greater than four months. The clinical and immunological status of pediatric HIV/AIDS patients was determined according to the reported CD4 values in relation to age, as established by the CDC classification,[2, 42, 43] according to current clinical symptoms, like this: Category N: asymptomatic; Category A: mild symptomatology; Category B: moderate symptomatology, and category C: severe symptoms. The immunological status was also evaluated as specified by the classification per amount and percentage of CD4+ lymphocytes, varying from category 1 to 3, in which age is an important factor in the interpretation of CD4 values.

Dental information was gathered from routine dental records of the Pediatric HIV Clinic in Cali, which are kept with two different protocols: one is the standardized protocol for caries diagnosis proposed by ICDAS (rounded instruments, good lighting, drying, and clean surface). This classification system describes the degree of severity of cavities like this: healthy tooth (0), first visual change in enamel (1), distinct visual change in enamel (2), localized enamel breakdown (3), underlying dark shadow from dentin (4), distinct cavity with visible dentin (5) extensive distinct cavity with visible dentin (6). The other is the protocol established by the WHO: DMF/def, the bacterial plaque diagnosis by Silness and Loe modified, where 0-30%: good and acceptable oral hygiene, and > 30%: poor oral hygiene, and the diagnosis of plaque retentive factors (enamel defects). Data quality for ICDAS diagnosis was validated, as it was recorded by two pediatric dentists standardized in these criteria, with inter- and intra-examiner reproducibility of kappa values of 0.80-0.85 and 0.85-0.89 respectively.

The dental record survey provided the following data: toothpaste use, number of daily brushings, brushing times, reason for consultation, place where dental care is provided, and time of last dental visit. Variables concerning the frequency of daily consumption of food and beverages and number of carbohydrates per day were obtained from the 24-hour nutritional survey, also registered in the nutrition clinical record.

For data control and quality, 10% of the clinical records were randomly selected to be manually re-entered, creating the database on the Epi Info 6® software. The newly typed data were verified for quality and exported to version 11 of the STATA® statistical package, licensed to Universidad del Valle. Data exploration was performed, as well as a descriptive analysis of categorical variables with frequencies and their differences, statistically evaluated with the Chi^2 test.

To estimate point prevalence (the percentage of children who at the time of diagnosis had at least one untreated carious lesion at any stage—early or cavitated—) and lifetime prevalence (caries experience—or percentage of children who have suffered the disease at some time in life—) in patients with HIV, "a patient with caries" was defined as anyone who had ICDAS 2-6 lesions.

To identify risk factors associated with caries in patients with pediatric HIV, unadjusted OR were determined with 95% confidence intervals, using the Chi^2 test and $p < 0.05$ significance level. A multivariate analysis was then carried out with an estimation logistic regression model, backwards method, using the presence or absence of caries as dependent variable; the variables with a $p < 0.20$ association were included. The likelihood rate test (LRT) was used to validate each representative variable contribution to the model. if p value was > 0.10, it was removed from the model; however, if some of these variables were important according to the literature, they were kept in the model.

Before conducting logistic regression, the assumption of independence was validated by means of correlation matrix (Pearson correlation coefficient) among independent variables, in order to evaluate collinearity. Those with high collinearity were eliminated or regrouped. The postestimation test was used for regression using the goodness of fit test (the Hosmer and Lemeshow test) to evaluate goodness of fit to the model. For all analyses, a value of $p < 0.05$ was considered statistically significant.

RESULTS

This study included 101 clinical records of HIV patients in June 2013. The average age of participating children was 10.38 ± 3.76 years, with a 1:1 male-female ratio. Forty children (39.6%) were institutionalized (living in Fundación Fundamor). Seventy-five children (74.26%) had their latest viral load and CD4 count tests less than three months before the day of dental diagnosis, and twenty-six (25.74%) had been tested no later than four months before dental diagnosis. 90% (n = 91) did not show oral lesions of any kind at the time of diagnosis; only 9.9% had lesions such as aphthae and acute necrotizing ulcerative gingivitis (ANUG). Twenty-five children (24.75%) reported some type of oral lesion in their medical records. 60.4% of children had hypomineralization and 3.96% had hypoplasia. Regarding oral health care, 97% of children (n = 98) did not use to brush immediately after taking the HAART therapy. As for use of fluoride toothpaste and amounts used, 12.12% (n = 12) of patients say they cover one third of the brush with toothpaste, 42.42% (n = 42) report covering half the brush with toothpaste, and 45.45% (n = 45) cover the entire brush with toothpaste when brushing.

The prevalence of caries experience in participating patients was DMF/def $_{(ICDAS\ 2\text{-}6)}$ 83.17% (n = 84), with a DMF/deft index ($_{ICDAS\ 2\text{-}6}$) 6.39 ± 4.66. The prevalence of active caries was DMF/def $_{(ICDAS\ 2\text{-}6)}$ 74.26% (n = 75); in permanent dentition it was DMF/def $_{(ICDAS\ 2\text{-}6)}$ 65.17% (n = 58) and in temporary dentition was 64.08% (n = 41). This prevalence decreases to a DMF/def index $_{(ICDAS\ 2\text{-}6)}$ of 34.65% (n = 35) and DMF/deft ($_{5\text{-}6}$) 3.29 ± 3.06 when only cavitated lesions are diagnosed. The values of the DMF/deft index corresponding to the different degrees and per dentition type are shown in table 1. In permanent dentition, the obturated teeth score is 1.24 and the lost teeth score is 0.034. In temporary dentition, the obturated teeth score is 1.01, while the lost teeth score is 0.53.

Table 1. *Decayed, missing, filled teeth index according to the ICDAS classification system and dentition type in children with HIV. Cali, Colombia, 2013*

Cut-off point ICDAS criteria	**General DMF/deft X ± SD**	**Deciduous dentition X ± SD**	**Permanent dentition X ± SD**
Non-cavitated $_{2\text{-}6}$	6.39 ± 4.66	4.34 ± 4.38	3.17 ± 3.04
Microcavitated $_{3\text{-}6}$	4.27 ± 3.81	2.88 ± 3.40	2.18 ± 2.41
ICDAS-t $_{4\text{-}6}$	3.50 ± 3.23	2.38 ± 2.96	1.69 ± 2.01
Cavitated $_{5\text{-}6}$	3.29 ± 3.06	2.28 ± 2.85	1.58 ± 1.85

The DMF ($_{5\text{-}6}$) healthy tooth index was 18.52 and the deft ($_{5\text{-}6}$) index was 10.58. This index drops to 14.25 when considering the precavitated lesions DMF ($_{2\text{-}6}$). The proportion of healthy patients was 18.81% (n = 19), the proportion of patients with precavitated lesions DMF/def $_{(ICDAS\ 2\text{-}4)}$ was 46.53% (n = 47) and the proportion of patients with only cavitated lesions was DMF/def $_{(ICDAS\ 5\text{-}6)}$ 34.65% (n = 35).

Table 2 shows the results of the bivariate analysis, showing similar behavior in all the socio-demographic, immunologic, and dental variables, except for bacterial plaque index (BPI) and caries experience, which showed statistically significant differences in terms of distribution between the two groups.

Table 2. *Description of socio-demographic, immunologic, and dental variables related to the presence of dental caries in children with HIV. Cali, Colombia, 2013*

	Total population	**Caries n = 82**	**No caries n = 19**	***p***
	N = 101 (100%)	n (%)	n (%)	
Socio-demographic variables				
Sex				
Female	51 (50.5)	42 (51.22)	9 (47.37)	
Male	50 (49.5)	40 (48.78)	10 (52.63)	0.76*
Institutionalized				
Yes	40 (39.6)	33 (40.24)	7 (36.84)	
No	61 (60.4)	49 (59.76)	12 (63.16)	0.78 *
Origin				
Cali = 0	68 (67.33)	55 (67.07)	13 (68.42)	
Out of Cali = 1	33 (32.67)	27 (32.93)	6 (31.58)	0.90 *
Immunologic variables				
Clinical status (n = 98)				
A	18 (18.37)	16 (20.25)	2 (10.53)	
B	26 (26.53)	21 (26.58)	5 (26.32)	0.47
C	54 (55.10)	42 (53.16)	12 (63.16)	0.31 *
Immune status (n = 98)				
1	30 (32.26)	28 (36.84)	2 (11.76)	
2	23 (24.73)	18 (23.68)	5 (29.41)	0.12
3	40 (43.01)	30 (39.47)	10 (58.82)	0.06
CD4 lymphocytes				
Cat. 1 with no immunosuppression	78 (77.23)	63 (76.83)	15 (78.95)	
Cat. 2 and 3 with moderate and severe immunosuppression	23 (22.77)	19 (23.17)	4 (21.05)	0.84 *
Viral load (copies/ml) (n, %)				
Undetectable	67 (66.34)	54 (65.85)	13 (68.42)	
Detectable	34 (33.66)	28 (34.15)	6 (31.58)	0.83 *
Diet				
Frequency of food consumption				
> 7 meals	50 (49.50)	43 (52.44)	7 (36.84)	0.22 *
< 7 times a day	51 (50.50)	39 (47.56)	12 (63.16)	
Daily carbohydrates				
> 2 carbohydrates	53 (52.48)	45 (54.88)	8 (42.11)	0.20 *
2 Carbohydrates	48 (47.52)	37 (45.12)	11 (57.89)	
% bacterial plaque - Silens and Loe				
16-20% - Poor oral hygiene	18 (17)	10 (12.20)	8 (42.11)	
> 30% - Bad oral hygiene	83 (83)	72 (87.18)	11 (58.89)	**0.002 ***
Bacterial plaque retentive factors				
Any retentive factor				
Yes	82 (81.19)	69 (84.15)	13 (68.42)	0.11 *
No	19 (18.81)	13 (15.85)	6 (31.58)	
Caries experience				
Yes	41 (40.59)	38 (46.34)	3 (15.79)	0.015 *
No	60 (59.41)	44 (53.66)	16 (84.21)	
Frequency of brushing	100			
More than two times	61 (61)	48 (58.54)	13 (72.22)	
Less than two times	39 (39)	34 (41.46)	5 (27.78)	0.28 *
Access to health services				
Time of last visit	100			
Less than 6 months	81 (81)	66 (81.48)	15 (78.95)	
More than 6 months	19 (19)	15 (18.52)	1 (21.05)	0.80 *

Reason for consultation	100			
Control and prevention	60 (60)	48 (59.26)	12 (63.16)	
Treatment and emergencies	40 (40)	33 (40.74)	7 (36.84)	0.75 *
*** Chi^2**				

Concerning the relationship between immunological variables and tooth decay, the highest percentage of patients with caries were classified in clinical stage C (42/82, representing 53.16%) and immune stage 3 (30/82, representing 39.47%), but the differences in caries proportions among clinical stages were not statistically significant.

Table 3 outlines the analysis of association between the variables of exposure and the presence or absence of caries, as well as OR, CI, and *p* value. There was statistically significant association with bacterial plaque only, and a greater association with caries in patients with poor oral hygiene, with OR = 5.23 (p = 0.002). In other words, patients with poor oral hygiene are 4.23 times more likely to have caries than patients with adequate oral hygiene.

Table 3. *Association between caries and variables of exposure in children with HIV. Cali, Colombia, 2013*

	Caries					**Chi^2**	**p**
	Yes (case)		No (control)				
Socio-demographic variables	n	%	n	%	OR (CI95%)		
Sex							
Female	42	51.22	9	47.37	1		
Male	40	48.78	10	52.63	0.85 (0,27-2,67)	0.09	0.76
Age (range)	10	(3-17)	11	(1-16)	1.05 (0.92-1.20)		0,40
Institutionalized							
Yes	33	40.24	7	36.84	1		
No	49	59.76	12	63.16	0.86 (0,26-2,69)	0.07	0.78
Immunologic variables							
CD4 lymphocytes							
No depression	63	76.83	15	78.95	1		
Moderate to severe depression	19	23.17	4	21.05	1.13 (0.33-3.81)	0.03	0.84
Viral load (copies/ml)							
Undetectable	54	65.85	13	68.42	1		
Detectable	28	34.15	6	31.58	1.12 (0.35-4.0)	0.04	0.83
Frequency foods/day							
> 7 meals	43	52.44	7	36.84	1.84 (0.67-5.28)	1.50	0.22
< 7 times day	39	47.56	12	63.16	1		
Carbohydrates/day							
> 2 carbohydrates	45	54.88	8	42.11	1.67 (0.60-4.55)	1.62	0.20
< 2 Carbohydrates	37	45.12	11	57.89	1		
% Silness and Loe							
Poor oral hygiene	10	12.20	8	42.11	1		

> 30% bad oral hygiene	72	87.18	11	57.89	5.23 (1.69-16.13)	9.42	0.002
Plaque retentive factors							
Yes	69	84.15	13	68.42	2.44 (0.78-10.45)	2.49	0.11
No	13	15.85	6	31.58	1		
Caries experience							
Yes	38	46.34	3	15.79	4.60 (1.24-17.02)		
No	44	53.66	16	84.21	1	5.97	0.015
Brushings per day							
More than two times	48	58.54	13	72.22	1		
Less than two times	34	41.46	5	27.78	1.84 (0.60-5.60)	1.16	0.28
Time of latest visit							
Less than 6 months	66	81.48	15	78.95	1		
More than 6 months	15	18.52	1	21.05	0.85 (0.22-4.03)	0.06	0.80
Reason for consultation							
Control and prevention	48	59.26	12	63.16	1		
Treatment and emergencies	33	40.74	7	36.84	1.17 (0.37-3.92)	0.10	0.75

OR = Odds Ratio, p = observed probability value < 0,05, Chi^2 test = Chi squared

The variables used in the logistic regression model for multivariate analysis and likelihood ratio test were those that yielded *p* values < 0.20 in the bivariate analysis: nutritional status $p = 0.04$, frequency of carbohydrates per day $p = 0.20$, BPI $p = 0.002$, plaque retentive factors $p = 0.11$, caries experience $p = 0.015$; and even though they were not statistically significant, the following were also included as suggested by theory: age, viral load and immunological status according to CD4 count, and institutionalization. The assumption of independence was verified by analysis of multicollinearity; values of Pearson's correlation coefficient among independent variables > 0.80 were used as a criterion for the presence of collinearity, which helped create a correlation matrix. Collinearity > 0.80 was not found among the variables; instead, there was a significant multicollinearity relationship between variables "response to caries" and "caries experience" (0.24), as well as "frequency of brushing" with BPI 0.19 and "caries experience" (0.34). Therefore, "frequency of brushing" was removed as a variable, due to multicollinearity with BPI and caries experience.

Finally, the model was forced with the following variables: CD4 count, institutionalization, age, bacterial plaque index, retentive factor, carbohydrates consumption, caries experience, and nutritional status, obtaining a multiple regression model by the stepwise method, with 0.10 backwards removal probability, finding out that the variables that best explain the presence of caries in children with HIV/AIDS, compared with caries-free kids, were BPI and caries experience—the factors that were independently associated with caries (table 4).

Table 4. *Adjusted multivariate analysis of factors associated with caries in kids aged 1 to 17 years of the Pediatric HIV/AIDS Clinic in Cali, Colombia, 2013*

Variable	Adjusted OR	*p*
BPI		
0 = low	1	
1 = poor oral hygiene	4.78 (1.48-15.36)	**0.009**
Caries experience		
0 = no experience	1	
1 = experience	4.21 (1.09-16.13)	**0.036**

The goodness-of-fit test was conducted, yielding $p > 0.83$, suggesting that the data fit to the model. The hypothesis that the model has a good fit at the end was not rejected.

DISCUSSION

The study of point prevalence and lifetime prevalence of dental caries among the population treated at the Pediatric HIV Clinic in Cali, Colombia, shows an increase in prevalence when non-cavitated lesions are included using the ICDAS classification system, which means that 47% of early lesions fail to be diagnosed because only the cavitated ones are detected, with the consequent underreporting.[44] Such underreporting can aggravate the systemic condition of the HIV population, as the life expectancy and quality of life of these patients are constantly increasing.

This study determined the prevalence of caries experience (including any traces of the disease, like decayed, missing, or filled teeth) and the point prevalence of caries (including non-treated caries only), considering that the "prevalence" of a disease should include all existing cases, both old and new.[39] This differs from what has been reported in studies at the national level, in which the term "caries experience" is used indicate the proportion of people with evidence of the disease at some point in life, either as active caries or as fillings or extractions due to caries[13]—which in reality is "lifetime prevalence". Similarly, what the literature calls caries prevalence is rather point prevalence of active caries, i.e. the proportion of people who have untreated or active caries at the time of diagnosis.[13]

The ICDAS classification system was used because some studies have shown its good validity;[10, 45] and while it has been pointed out that this system may require longer training sessions compared to other diagnostic systems, as well as decreased reliability if it is not standardized, and increased costs, the inclusion of early lesions can make the associations with some risk indicators more sensitive.[46]

In this study, validity is not affected by the use of diagnostic criteria since the diagnoses were performed by two previously standardized examiners. Quite the opposite, the differences in reported prevalence per lesion type (ICDAS > 2 = 74.26% vs ICDAS > 5 = 34.65%) validate the need to continue using these modified classification systems to avoid underreporting caries in early stages, preventing early lesions (white stains) from progressing into cavities, thus monitoring and treating them throughout life, improving the condition of these systemically-compromised patients. The fact that in this study prevalence triples depending on the index used is a warning for future studies, which should try to demonstrate more clearly the actual health status of the population, even in early stages of carious processes. A decision was made to take caries code 2 as an ICDAS cut-off point in order to make comparisons with other studies.[46]

The reports on caries prevalence in HIV/AIDS patients worldwide are scarce and their results are mixed because their primary objective is not to assess dental caries, variability in population ages, or differences in diagnostic criteria.

A few limitations arise in comparing the results of the present study with other studies conducted in this population worldwide, because such studies use other methods to diagnose caries,[14, 20] so one should be careful when comparing the reported prevalence in this study with that of the literature.

Caries prevalence in this study was similar to that reported by Ribeiro et al[20] using the Nyvad criteria in 57 children, finding out 73.2% caries prevalence with a modified DMFT score of 3.41 and a modified deft score of 7.01.

In terms of point prevalence of tooth decay in cavitated stages (ICDAS 5-6) among HIV patients, the findings of the present study are similar to those by Sahana et at,[21] who reported a value of 16.60% in the group aged 7 to 8 years and 21.42% in the group aged 10 to 12 years, but lower than those reported by three other authors: Beena,[47] in 104 children aged 2 to 14 years with HIV, reports a 76.47% prevalence of caries in permanent dentition, with a DMFT score of 3.00 ± 2.37; Flaitz et al,[48] in 173 children aged 6 to 12 years, reported a deft score of 3.7 and a DMFT score of 3.1, and Álvarez et al,[49] in 76 kids with HIV aged 1 to 16 years, report a DMF score of 1.28 ± 1.72 in the mixed dentition group and 2.8 ± 2.4 in the permanent dentition group, and a def score of 4.52 ± 6.2 in the temporary dentition group. In this study in permanent dentition, the results were higher than those reported by Sales-Perez et al,[23] who, in 90 children aged 1.7 to 16 years in Mozambique, found an average DMFT score of 0.6 ± 1.6 and lower levels for temporary dentition compared to those reported by Sahana et al[21] in children 2 to 6 years old (57.15%), with a deft score of 0.55 ± 1.08, while Beena et al[47] reported values of 58.62%, with deft 5.07 ± 5.29, and 86.20% with deft 3.81 ± 3.41 in mixed dentition.

The similar behavior of caries prevalence among patients with and without HIV has been explained by a significant increase in IgA among the HIV-positive population ($p < 0.05$) since these children have the ability to respond to cariogenic microorganisms, as reported by Castro et al.[50] Oral health care is an important component in people with HIV infection, since any alteration in these patients' dental structures and mucous membranes can affect quality of life and aggravate the disease; it is also a predictive factor to diagnose the systemic condition.[22, 51]

The constant appearance of HIV in children highlights the need for dentists and physicians to diagnose diseases such as tooth decay in early stages, focusing treatments on prevention and maintenance of a healthy oral cavity, thus avoiding complications;[47] the use of diagnostic criteria to identify carious processes in early stages is therefore relevant.

The most frequently reported risk factors for caries in adults and children with HIV are the immune status and the percentage of CD4 count.[52-53] This pattern is not so clearly present in this study, since the conditions of the infant population are very different from those of the adults, such as transmission type, the time elapsed since the disease was acquired, its evaluation, and the CD4 component (< 500 CD4). These results may be explained because an increased number of patients in the Pediatric HIV Clinic in Cali have been immunologically controlled over the years, and therefore very few patients (5) have severe immunosuppression, making it difficult to establish connections between immunological status and dental caries. Our findings are similar to those reported by Beena,[47] who found association between caries prevalence and decreased absolute CD4 count, although these findings are not statistically representative and differ from those reported by Castro et al,[54] who found out a high prevalence of caries in patients in advanced stages of the disease and with more severe degrees of immunosuppression.

No statistically significant associations were found between caries and detectable viral load (OR 0.17; $p = 0.86$), contrary to the report by Sahana et al,[21] who attribute the low prevalence of caries to knowledge on oral health and to the elimination of high sugar contents from children's diet.

The medicines prescribed for children infected with HIV are mostly syrups with a high sucrose content, which have been reported to produce low pH and are therefore highly cariogenic.[53] This study did not establish this cariogenic potential because, while it is true that the hosts are individuals, the exposure factor was the same for all patients as they have continuously received HAART therapy, usually take their medicines with water, and do not brush teeth afterwards.

A statistically significant association was found between the presence of dental caries and poor oral hygiene, assessed by the presence of bacterial plaque with OR = 5.23. This association was maintained when the model was adjusted to children with poor oral hygiene, showing independent association with caries, with an adjusted OR = 4.02. Aguiar Ribeiro et al[20] achieved similar results, finding out a correlation between the presence of carious activity and bacterial plaque thickness (Spearman correlation test, Rs = 0.49).

The present study explores only one biological model for the development of dental caries, which is a weakness of the study, considering the current approach to tooth decay, which transforms Keyes epidemiological triad into a multi-causal model composed by some determinant biological risk factors combined with concomitant social risk factors. Social factors could not be measured because no information was available in this regard.

The current knowledge gap on caries prevalence in HIV patients and its associated factors may indicate a lack of interest among researchers and health personnel towards oral health, thus neglecting this population and making it more vulnerable; this is then an invitation to include HIV patients in specific prevention programs.[55]

One of the strengths of this study is that, as a population census analysis, it controls external validity and differences between lifetime prevalence and point prevalence of dental caries. The information gathered from clinical records is reliable since patients were monitored monthly and data were collected by specialists in dentistry, medicine, nutrition, and laboratory. In addition, there was control of information bias in the dental variables. Immune status was correctly classified per age, and standardized personnel oversaw the ICDAS criteria, increasing sensitivity and specificity of the diagnosis.

The possible biases of the study are related to the patients' recollection to provide information about hygiene for the medical records.

The present study made it possible to know for the first time the oral health situation of pediatric patients with HIV in southwestern Colombia. Since these patients currently enjoy higher levels of life expectancy, they need more programs for the control oral diseases, including dental caries. The study also recognizes the importance of controlling the immune status of HIV patients as it helps control other risk factors.

Prospective studies are recommended to establish associations between caries and HIV in immunologically controlled and non-controlled patients, as well as further analysis of the correlation of immune status with average number of decayed teeth, lesions activity, and degree of severity.

CONCLUSIONS

The use of caries-recording systems including precavitated lesions is currently an important tool to characterize the stage of lesions. Punctual prevalence (active caries) and lifetime prevalence (caries experience) in patients from the Pediatric HIV Clinic in Cali are high when assessing cavitated and precavitated lesions (ICDAS 2-6), and thus the presence of bacterial plaque is a possible risk factor. The patients' immune status was not an important factor, considering that the study population was immunologically stable at the time of examination.

CONFLICT OF INTERESTS

The authors state that there was no conflict of interest.

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REFERENCES

1. WHO. UNAIDS. UNICEF. Global HIV/AIDS response: epidemic update and health sector progress towards universal access: progress report 2011. Geneva: World Health Organization, 2011.

2. Beltrán S, López P, Sierra A. Recomendaciones para el abordaje, diagnóstico y tratamiento de niños, niñas y adolescentes con VIH/SIDA. 2 ed. Cali : Catorse SCS; 2011

3. Colombia. Ministerio de Salud y Protección Social. Informe UNGASS – 2012: seguimiento de la declaración de compromiso sobre el VIH/Sida: informe mundial de avances en la lucha contra el Sida, Colombia 2012. Bogotá: Ministerio de Salud y Protección Social; 2012.

4. Niños con VIH en Cali tienen más opciones de vida [Internet]. El Pais. 2012 jul 02. Available in: http://www.elpais.com.co/cali/ninos-con-vih-en-tienen-mas-opciones-de-vida.html

5. Fejerskov O, Kidd E. Dental caries: the disease and its clinical management. 2 ed. Copenhagen, Denmark: Blackwell Munksgaard; 2008

6. Klein H, Palmer CE; Knutson JW. Studies on dental caries. I. Dental status and dental needs of elementary school. J Am Dent Assoc. 1938; 25(10): 1703-1705. DOI: https://doi.org/10.14219/jada.archive.1938.0274

7. Pitts NB, Deery C, Fyffe HE, Nugent ZJ. Caries prevalence surveys. A multi-country comparison of caries diagnostic criteria. Com Den Health. 2000; 17: 196-203.

8. Banting D, Eggertsson H, Ekstrand K, Ferreira A , Ismail A, Longbotto et al. Manual sobre los Criterios del Sistema Internacional de Detección y Valoración de Caries(ICDAS II), Taller realizado en Baltimore, Maryland el 12 al 14 de marzo del 2005.

9. Ekstrand KR, Martignon S, Ricketts DJN, Qvist V. The validity of a classification system for clinical activity assessment of primary coronal caries lesions. Caries Res. 2005; 39(4): 299-300. Abstract No. 38

10. Ismail AI, Sohn W, Tellez M, Amaya A, Sen A, Hasson H et al. The International Caries Detection and Assessment System (ICDAS): an integrated system for measuring dental caries. Community Dent Oral Epidemiol. 2007; 35(3): 170-178. DOI: https://doi.org/10.1111/j.1600-0528.2007.00347.x

11. Braga MM, Oliveira LB, Bonini GA, Bönecker M, Mendes FM. Feasibility of the International Caries Detection and Assessment System (ICDAS-II) in epidemiological surveys and comparability with standard World Health Organization criteria. Caries Res. 2009; 43(4): 245-249. DOI: https://doi.org/10.1159/000217855

12. Hernández-Ramírez JR, Gómez-Clavel JF. Determinación de la especificidad y sensibilidad del ICDAS y fluorescencia láser en la detección de caries in vitro. Rev ADM. 2012; 69(3): 120-124.

13. Colombia. Ministerio de Salud y Protección Social. IV estudio nacional de salud bucal (ENSAB IV). Bogotá: Ministerio de Salud y Protección Social; 2014

14. Eldridge K, Gallagher J. Dental caries prevalence and dental health behaviour in HIV infected children. Int J Paediatr Dent. 2000; 10(1): 19-26.

15. Pongsiriwet S, Lamaroon A, Kanjanavanit S, Pattanaporn K, Krisanaprakornkit S. Oral lesions and dental caries status in perinatally HIV-infected children in Northern Thailand. Int J Ped Dentistry. 2003; 13(3): 180–185.

16. Pinzón EM, Bravo SM, Méndez F, Clavijo GM, León ME. Prevalencia y factores relacionados con la presencia de manifestaciones orales en pacientes con VIH/SIDA, Cali Colombia. Colomb Méd. 2008; 39(4): 346-55

17. Marín M, Hurtado P, Santamaría L, Durango A, Patiño C, Toro M. Estado de salud bucal de niños con diagnóstico de VIH-sida en la fundación Eudes de Medellín. Presentación de casos y revisión de literatura. Rev Fac Odontol Univ Antioq. 2004; 15(2): 64-77

18. Ponnam SR, Srivastava G, Theruru K. Oral manifestations of human immunodeficiency virus in children: An institutional study at highly active antiretroviral therapy centre in India. J Oral Maxillofac Pathol. 2012; 16(2): 195-202. https://doi.org/10.4103/0973-029X.98499

19. Mosca N. With HAART success, managing dental caries is again important. HIV Clin. 2002; 14(1): 6-7

20. Ribeiro AdA , Portela MB, De-Souza IP. The oral health of HIV-infected Brazilian children. Int J Paediatr Dent. 2013; 23(5): 359–365. DOI: https://doi.org/10.1111/ipd.12008

21. Sahana S, Krishnappa SS, Krishnappa VS. Low prevalence of dental caries in children with perinatal HIV infection. J Oral Maxillofac Pathol. 2013; 17(2): 212-216. DOI: https://doi.org/10.4103/0973-029X.119742

22. Rwenyonyi CM, Kutesa A, Muwazi L, Okullo I, Kasangaki A, Kekitinwa A. Oral manifestations in HIV/AIDS infected children. Eur J Dentistry. 2011; 5: 291-298.

23. Sales-Peres SHdC, Mapengo MAA, Moura-Grec PGd, Marsicano JA, Sales-Peres AdC, Sales-Peres A. Oral manifestations in HIV+ children in Mozambique. Ciênc Saúde Coletiva. 2012; 17(1): 55-60 DOI: http://dx.doi.org/10.1590/S1413-81232012000100008

24. Trejos-Herrera AM, Mosquera-Vásquez M, Tuesca-Molina R. Niñez afectada con VIH/SIDA: calidad de vida, funcionalidad familiar y apoyo social en cinco ciudades colombianas. Salud UNINORTE. 2009; 25(1): 17-32

25. Buczynski AK, Castro GF, Souza IPR. O impacto da saúde bucal na qualidade de vida de crianças infectadas pelo HIV: revisão de literatura. Ciênc Saúde Coletiva. 2008; 13(6) : 1797-1805. DOI: 10.1590/S1413-81232008000600014

26. Cavasin Filho JC, Giovani EM. Xerostomy, dental caries and periodontal disease in HIV+ patients. Braz J Infect Dis. 2009; 13(1): 13-17

27. Nittayananta W, Talungchit S, Jaruratanasirikul S, Silpapojakul K, Chayakul P, Nilmanat A et al. Effects of long-term use of HAART on oral health status of HIV infected subjects. J Oral Pathol Med. 2010; 39(5): 397–406. DOI: https://doi.org/10.1111/j.1600-0714.2009.00875.x

28. Takahashi N, Nyvad B. The role of bacteria in the caries process: ecological perspectives. J Dent Res. 2011; 90(3): 294-303. DOI: https://doi.org/10.1177/0022034510379602

29. Nelson A, Albert JM, Lombardi G, Wishnek S, Asaad G, Kirchner HL et al. Dental caries and enamel defects in very low birth weight adolescents. Caries Res. 2010; 44(6): 509–518. DOI: https://doi.org/10.1159/000320160

30. Berkowitz RJ. Mutans streptococci: acquisition and transmission. Pediatr Dent. 2006; 28(2): 106-109.

31. Navazesh M, Mulligan R, Barrón Y, Redford M, Greenspan D, Alves M et al. A 4-year longitudinal evaluation of xerostomia and salivary gland hypofunction in the Women's Interagency HIV Study participants. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2003; 95(6): 693-698. DOI: https://doi.org/10.1067/moe.2003.230

32. American Academy of Pediatric Dentistry. Policy on early childhood caries (ECC): classifications, consequences, and preventive strategies. Pediatr Dent. 2008; 29(Suppl): 39-41

33. Caufield PW, Griffen AL. Dental caries: an infectious and transmissible disease. Pediatr Clin North Am. 2000; 47(5): 1001-1019.

34. Sharland M, Di-Zub GC, Ramos JT, Blanche S, Gibb DM; PENTA Steering Committee. PENTA guidelines for the use of antiretroviral therapy in paediatric HIV infection. Pediatric European Network for Treatment of AIDS. HIV Med. 2002; 3(3): 215- 226.

35. Gaitán-Cepeda LA, Sánchez-Vargas LO, Pavia-Ruz N, Muñoz-Hernández R, Villegas-Ham J, Caballos-Salobreña A. Candida bucal en niños mexicanos con VIH/sida, desnutrición o marginación social. Rev Panam Salud Pública. 2012; 31(1): 48–53.

36. Vieira AR, de-Souza IPR, Modesto A, Castro GF, Vianna R. Gingival status of HIV+ children and the correlation with caries incidence and immunologic profile. Pediatr Dent. 1998; 20(3): 169-172

37. Ribeiro AA, Portela M, Souza IP. Relation between biofilm, caries activity and gingivitis in HIV + children. Pesqui Odontol Bras. 2002; 16(2): 144-150

38. Fine DH, Tofsky N, Nelson EM, Schoen D, Barasch A. Clinical implications of the oral manifestations of HIV infection in children. Dent Clin North Am. 2003; 47(1): 159–174

39. Szklo M, Nieto J. Epidemiologia intermedia: conceptos y aplicaciones. Madrid: Díaz de Santos; 2003.

40. Moreno-Altamirano A, López-Moreno S, Corcho-Berdugo A. Principales medidas en epidemiología. Salud Pública Méx. 2000; 42(4): 337-348

41. Tapia-Granados JA. Medidas de prevalencia y relación incidencia-prevalencia. Organización Panamericana de la Salud. (OPS/OMS). Programa de Publicaciones. Washington. EE. UU. Med Clin (Barc). 1995; 105: 216-218.

42. Alejo A, Romero J, García-Gestoso ML. Infección por VIH en pediatría: En: Pachón J, Pujol E, Rivero A. Infección por el VIH: guía práctica. 2 ed. Sevilla: Sociedad Andaluza de Enfermedades Infecciosas; 2003. p. 441-468.

43. Dávila M, Gil M. Manifestaciones orales y caries dental en niños expuestos al virus de inmunodeficiencia humana. Rev Salud Pública. 2011; 13(5): 833-843.

44. Saldarriaga A, Arango CM, Cossio M, Arenas A, Mejía C, Mejía E et al. Prevalencia de caries dental en preescolares con dentición decidua área Metropolitana del Valle de Aburrá. Rev CES Odont. 2009; 22(2): 27-34

45. Ismail AI, Sohn W, Tellez M, Willem JM, Betz J, Lepkowski J. Risk indicators for dental caries using the International Caries Detection and Assessment System (ICDAS). Community Dent Oral Epidemiol. 2008; 36(1): 55–68. DOI: https://doi.org/10.1111/j.1600-0528.2006.00369.x

46. Mendes FM, Braga MM, Oliveira LB, Antunes JL, Ardenghi TM, Bönecker M. Discriminant validity of the International Caries Detection and Assessment System (ICDAS) and comparability with World Health Organization criteria in a cross-sectional study. Community Dent Oral Epidemiol. 2010; 38(5): 398-407. DOI: https://doi.org/10.1111/j.1600-0528.2010.00557.x

47. Beena JP. Prevalence of dental caries and its correlation with the immunologic profile in HIV-Infected children on antiretroviral therapy. Eur J Paediatr Dent. 2011; 12(2): 87-90.

48. Flaitz C, Wullbrandt B, Sexton J, Bourdon T, Hicks J. Prevalence of orodental findings in HIV infected Romanian children. Pediatr Dent. 2001; 23(1): 44-50.

49. Álvarez L, Hermida L, Cuitiño E. Situación de salud oral de los niños uruguayos portadores del virus de la inmunodeficiencia humana. Arch Pediatr Urug. 2007; 78(1): 23-28

50. Castro GF, Souza IP, Lopes S, Stashenko P, Teles RP. Salivary IgA to cariogenic bacteria in HIV-positive children and its correlation with caries prevalence and levels of cariogenic microorganisms.

Oral Microbiol Immunol. 2004; 19(5): 281-288. DOI: https://doi.org/10.1111/j.1399-302x.2004.00152.x

51. Damle SG, Jetpurwala AK, Saini S, Gupta P. Evaluation of oral health status as an indicator of disease progression in HIV positive children. Pesq Bras Odontoped Clin Integr João Pessoa. 2010; 10(2): 151-156. DOI: https://dx.doi.org/10.4034/1519.0501.2010.0102.0004
52. Davoodi P, Hamian M, Nourbaksh R, Ahmadi Motamayel F. Oral manifestations related to CD4 lymphocyte count in HIV-positive patients. J Dent Res Dent Clin Dent Prospects. 2010; 4(4): 115-119. DOI: https://dx.doi.org/10.5681%2Fjoddd.2010.029
53. Subramaniam P, Kumar K. Cariogenic potential of medications used in treatment of children with HIV infection. Spec Care Dentist. 2014; 34(3): 127-130. DOI: https://doi.org/10.1111/scd.12041
54. Castro GF, Souza IP, Chianca TK, Hugo R. Evaluation of caries prevention program in HIV+children. Braz Oral Res. 1997; 15: 91-97.
55. Rovaris NS, Martins TLG, Traebert J. Oral health condition of children living with HIV. Dent J. 2014; 2: 1-10.