

## Oral manifestations of HIV positive children

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*Oral manifestations in HIV positive children were observed in thirty-eight HIV infected children that have received care at the Special Care Dentistry Center (SCDC) of the School of Dentistry, University of Sao Paulo. Results have shown that 52.63% of the children presented at least one oral manifestation related with HIV/AIDS. Angular cheilitis occurred in 28.94%, parotid gland bilateral enlargement, pseudomembranous candidiasis and erythematous candidiasis in 18.42%, conventional gingivitis in 13.15%, herpes simplex in 5.26%, hairy leukoplakia, recurrent aphthous ulcer and condyloma acuminatum in 2.63%. Although enamel hypoplasia occurred in 23.68%, this could not be attributed specifically to HIV infection.*

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### INTRODUCTION

In the city of Sao Paulo, epidemiological data indicate that HIV infection has been spreading and is now an important cause of death in the pediatric group. In Brazil, the first pediatric case of aids occurred in 1983. In 1990 there were 313 notified cases, and nowadays, 10 years later, the number of children living with aids is 762. Among them, 84% were infected through the vertical route.

The second transmission route occurs through contaminated needles, which are utilized by drug users, followed by sexual transmission. Because in most countries the vertical route is the most important one, several studies have evaluated protocols that reduce this transmission risk.

Connor<sup>1</sup> demonstrated that an early treatment of seropositive pregnant mothers with Zidovudine (AZT) reduces chances of transmission to babies to 8% from 25%. Other studies have shown that Vitamin A supplementing prevents perinatal transmission and reduces

morbidity from many complications associated with the HIV infection.<sup>2,3</sup>

HIV infection progression is faster and more severe in children, due to the immaturity of the immune system. Its clinical course can be described with a bimodal curve, where approximately 25% of infected children develop AIDS in their first year of life, with a rapid progression of the disease. The remaining 75% develop the disease later and slowly.<sup>4,5</sup>

Orofacial manifestations are among the earliest and most common clinical signs of pediatric HIV disease. They are considered prognosis indicators for the disease. Thus, several authors demonstrated that the presence of oral candidiasis in children indicates a poor prognosis. Parotid gland enlargement is indicative of a better prognosis or long term survival.<sup>5-7</sup>

The purpose of this study was to determine the prevalence of oral manifestations associated with pediatric HIV infection in Brazilian children, seen at Special Care Dentistry Center (SCDC), School of Dentistry of the University of Sao Paulo.

### PATIENTS AND METHODS

Thirty-eight HIV positive children aged 2 to 13 years old, receiving routine dental care at the SCDC, were examined. Data obtained from medical past history, clinical examination and complementary exams were transcribed to an application form, especially designed for this study.

Data such as age, racial group, route of HIV transmission, signs and symptoms associated with HIV/Aids, opportunistic diseases, oral diseases, and antiretroviral therapy were collected.

Physical examination included also palpating sub-mandibular and retroauricular cervical areas, palpating

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**Table 1.** Gender, age, CD4+ level, category of immunosuppression, oral lesions related with HIV/Aids and systemic therapy applied

| Patient | Gender/age (years) | CD4   | Category of immunosuppression* | Oral lesions related with HIV/Aids** | Therapy            |
|---------|--------------------|-------|--------------------------------|--------------------------------------|--------------------|
| 01      | F/08yrs            | 278   | 2                              |                                      | AZT+DDI            |
| 02      | F/09yrs            |       |                                | EC+PC                                | AZT+DDI            |
| 03      | F/08yrs            |       |                                | ACh+PE+EC                            | AZT+3TC            |
| 04      | M/03yrs            |       |                                | ACh+EC                               | AZT+DDI+ NElf      |
| 05      | M/07yrs            | 111   | 3                              | PE+PC                                | 3TC +D4T           |
| 06      | M/08yrs            | 781   | 1                              |                                      | AZT+DDI            |
| 07      | M/02yrs            |       |                                |                                      | AZT+DDI+ NElf      |
| 08      | F/07yrs            | 959   | 1                              | PE                                   | Gamaglobulin       |
| 09      | M/08yrs            | 307   | 2                              |                                      | AZT+DDI            |
| 10      | M/04yrs            | 1.478 | 1                              | EC+ACh                               | AZT+ 3TC           |
| 11      | F/07yrs            | 862   | 1                              | ACh+PC+RAU+HS                        | AZT+DDI            |
| 12      | F/13yrs            |       |                                | ACh                                  | 3TC+D4T+Ritonavir  |
| 13      | M/07yrs            | 718   | 1                              | ACh+EC                               | AZT+DDI            |
| 14      | F/10yrs            | 211   | 2                              |                                      | 3TC +Ritonavir+D4T |
| 15      | F/05yrs            | 845   | 2                              |                                      | AZT+ DDI+ NElf     |
| 16      | F/08yrs            | 682   | 1                              |                                      | D4T+3TC+ Ritonavir |
| 17      | M/09yrs            |       |                                |                                      | Gamaglobulin       |
| 18      | F/06yrs            |       |                                |                                      | No Treatment       |
| 19      | M/04yrs            |       |                                | ACh+PE                               | No Treatment       |
| 20      | F/09yrs            | 688   | 1                              | HS                                   | AZT+DDI            |
| 21      | F/10yrs            | 310   | 2                              | EC+PE                                | AZT+DDI            |
| 22      | F/08yrs            |       |                                |                                      | AZT+DDI            |
| 23      | F/09yrs            | 75    | 3                              | PC+Cac                               | AZT+ 3TC           |
| 24      | F/06yrs            | 513   | 1                              |                                      | AZT+DDI            |
| 25      | M/04yrs            |       |                                | PE                                   | AZT+DDI            |
| 26      | F/06yrs            |       |                                |                                      | AZT+DDI            |
| 27      | M/06yrs            | 580   | 1                              | PC                                   | AZT+3TC            |
| 28      | M/08yrs            | 142   | 3                              |                                      | 3TC+ Ritonavir+D4T |
| 29      | M/08yrs            | 1.429 | 1                              | PE+ACh                               | AZT+DDI            |
| 30      | F/07yrs            | 845   | 1                              |                                      | Gamaglobulin       |
| 31      | M/11yrs            |       |                                |                                      | AZT+ 3TC           |
| 32      | M/04yrs            | 391   | 3                              |                                      | AZT+ DDI+ NElf     |
| 33      | M/06yrs            |       |                                |                                      | AZT+DDI            |
| 34      | M/07yrs            |       |                                | ACh+PC+EC+PE+HL                      | No Treatment       |
| 35      | F/4                | 1670  | 1                              |                                      | No Treatment       |
| 36      | M/3                | 1157  | 1                              |                                      | AZT + DDI          |
| 37      | M/6                | 778   | 1                              | ACh                                  | 3TC + D4T + NElf   |
| 38      | F/4                | 1192  | 1                              | ACh+ EC                              | AZT + 3TC + NElf   |

\*1.Absence of Immunossuppression; 2.Moderate Immunossuppression; 3.Severe Immunossuppression

\*\*Angular Cheilitis (ACh); Pseudomembranous Candidiasis (PC); Erythematous Candidiasis (EC); Herpes Simplex (HS); Hairy Leukoplakia(HL); Parotid Enlargement(PE); Condyloma Acuminatum (CAC); Recurrent Aphthous Ulcer (RAU).

parotid glands, and observing dermatological alterations. We considered as oral manifestations those lesions diagnosed during the appointments. Previous manifestations reported by the patient were not considered. Diagnoses of these manifestations were performed by direct clinical examination, and by complementary exams, such as biopsies, cytological examinations, culture, and radiography, according to each case.

**RESULTS**

Of the 38 HIV infected children who were examined, 19 were boys, and 19 were girls. The average age was

seven years old, ranging from 2 to 13 years. As to racial groups, 23 (60.52%) were melanoderm, and 15 (39.47%) were leucoderm. All children were infected through the vertical route. The CD4+ count, category of immunossuppression, oral lesions related with HIV/Aids presented, and therapy modality are in Table 1.

All patients presented with enlarged cervical lymph nodes. At least one oral lesion related with HIV/Aids was found in 20 (52.6%) patients. Table 2 summarizes the frequency of each oral lesion. The most prevalent was candidiasis (36,8%), followed by angular cheilitis (28,9%) and parotid enlargement (18.4%). Unspecific

gingivitis and enamel hypoplasia were available, but they weren't considered as HIV related lesions.

Thirty-one children were using antiretroviral therapy medication associations, and three were taking gamaglobulin.

Table 2. Prevalence of oral manifestations

| Oral lesions             | N° patients | % patients |
|--------------------------|-------------|------------|
| Candidiasis              | 14          | 36.8       |
| Angular Cheilitis        | 11          | 29.0       |
| Enamel Hypoplasia        | 09          | 23.7       |
| Parotid Enlargement      | 07          | 18.4       |
| Conventional Gingivitis  | 05          | 13.1       |
| Herpes Simplex           | 02          | 5.2        |
| Recurrent Aphthous Ulcer | 01          | 2.6        |
| Hairy Leukoplakia        | 01          | 2.6        |
| Condyloma Acuminatum     | 01          | 2.6        |

**DISCUSSION**

Candidiasis was the most frequent buccal mucosa opportunistic infection found in 65.78% of children examined. Lesions comprised eleven cases of angular cheilitis, seven cases of pseudomembranous candidiasis, and seven cases of erythematous candidiasis. The high prevalence of candidiasis has been reported in other studies on HIV infection in children. All cases of angular cheilitis were considered as candidiasis, because they were resolved with antifungal therapy. Moreover, previous studies support the idea of angular cheilitis being caused by *Candida*.<sup>8</sup>

Parotid enlargement was found in 18.42% of the children examined, and this is in accordance with findings from others, who reported a 20 to 47% frequency.<sup>7</sup> All cases observed were bilateral, asymptotic, and disfiguring. Although no patients related it, we observed clinically a low salivary flow in five of the seven children affected. These data suggest a relation between parotid enlargement and decreasing salivary flow. However more studies are necessary with more patients.

Hairy leukoplakia is extremely rare in children. We found this lesion in a boy, patient number 34 (Table 1), who presented with erythematous candidiasis, parotid bilateral enlargement, low salivary flow, and many caries. The lesion was white, with a corrugated surface along the lateral borders of the tongue. A non-invasive diagnostic method, scraping the lesion and using *in situ* hybridization, is particularly useful in children.<sup>9,10</sup>

Periodontal alterations found in the patients examined included four cases of gingivitis, which we considered as conventional or non-specific for HIV/Aids. They were clinically characterized by an enlargement in volume, smooth and shiny surface and bleeding

when touched. In all cases, they were associated with the presence of bacterial plaque, reacting favorably to dental prophylactic treatment. Linear gingival erythema, necrotizing gingivitis, and necrotizing periodontitis were not found in our study.

Condyloma acuminatum was seen in one patient, number 23, a girl whose seropositive mother and seronegative brother also had this infection on the buccal mucosa. According to the history of the case, they often shared the same toothbrush.

Enamel hypoplasia, which was found in nine patients, has been included in this study not only for its quantitative significance, but rather to warn future studies about the importance of verifying a possible relation between its occurrence and HIV infection, or therapies adopted by the mother and the child during odontogenesis.

We obtained CD4+ counts of the 24 patients. at the moment of examination, Then, with the age of the patients, we established the category of immunosuppression.<sup>11</sup> We observed that even in patients classified with absence of immunosuppression (category 1) the oral lesions were present. Nevertheless, these oral alterations were generally angular cheilitis and parotid enlargement. Only in two patients we observed discreet erythematous candidiasis, and one case of herpes simplex.

Over the last five years, the implementation of new drug protocols has allowed HIV infected children to be maintained alive and asymptomatic. Even so, orofacial manifestations should be considered a good indicator of disease progression in children. Since the mouth is easily examined, certain oral signs may be used to change and improve the treatment of HIV infection.

Finally, dentists can play a role both in the early detection of HIV related oral lesions and in training primary care physicians and pediatricians to recognize these oral lesions.

**CONCLUSION**

Oral manifestations frequently occur in HIV seropositive children. They are important to clinical staging of the infection, diagnosis and treatment, which allows for a longer and better life for patients. A careful oral examination of HIV infected children at regular intervals is important to preserve the quality of life of these patients.

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