Dental caries in HIV-infected children

R. Bruce Howell, DDS, MS John Jandinski, DDS, MS Paul Palumbo, MD Zia Shey, MS, DMD Milton Houpt, DDS, PhD

Introduction

Although AIDS was described as a new and distinct disease¹ in 1981, there have been few reports of the oral manifestations of the disease in children.²⁻⁴ Early reports described soft tissue lesions but provided little information in regard to dental caries. In recent years, as greater numbers of children with AIDS have been examined, the problem of extensive dental caries has been identified. This investigation, which is part of a larger study,⁵ reports the caries prevalence in a group of 55 HIV-infected pediatric patients, located in Newark, New Jersey, a nonfluoridated community.

Materials and Methods

Patients

From September 1989, to December 1990, 55 patients who were enrolled in the Children's Hospital of New Jersey AIDS Program participated in an oral screening examination. These patients represented approximately 40% of available patients with teeth for whom permission was obtained for screening examination participation. All of the patients were seropositive for HIV-1, diagnosed by a repeated positive ELISA (HIV) and confirmed by Western blot (DuPont Co., Jonesboro, AZ). Children who were too young to be diagnosed by

serology (< 15-24 months) had other evidence of HIV infection (e.g., a positive culture for HIV). Thirty-two females and 23 males with a median age of 5 years and a mean age of 5.5 years (range 1 to 11 years) participated in the study. Thirty-five were African American, 10 Hispanic, eight Caucasian, one Haitian, and one Oriental.

Table. Description of sample

little or no caries

Gender Mean Age N deft defs Q Years റ് Total sample 55 32 23 5.5 5.3 ± 2.8 14.7 ± 10.6 Patients with 25 15 10 5.4 10.4 4.5 29.9 19.8 extensive caries Patients with 30 17 13 5.6 1.0 ± 1.4 2.0 ± 2.9

Fifty of the 55 patients had contracted the disease via perinatal transmission of the virus, three were infected via blood transfusion, and two had been abused sexually.

Procedure

Informed consent was obtained from the patient's parents or guardians and the oral examination was performed at the Children's Hospital AIDS Program outpatient specialty clinic. This examination was part of

the monthly follow-up examination to monitor disease progression. The oral examination was conducted by one examiner (BH) with pen light illumination, mouth mirror and explorer, and gauze to dry the teeth. A lesion was recorded if there was an obvious break in the enamel, or if there was evidence of undermining of the enamel. If the clinical crown of the tooth was lost completely due to dental caries, or if the tooth had been extracted, a score of five surfaces decayed for posterior teeth and four surfaces decayed for anterior teeth was recorded in the defs index.

Four patients were re-examined subsequent to their original examination to test within-rater reliability; there was 100% agreement between the findings of both examinations.

Results

Of the 55 patients, almost half had extensive or rampant caries (Table). Twenty-five patients presented with extensive caries (at least 10 surfaces decayed), and 13 of those patients had rampant caries (at least 30 surfaces or 10 teeth decayed). All but seven of those patients had a primary dentition and were age 5 or younger. Patients with extensive caries presented with means of 29.9 ±

19.8 defs, and 10.4 ± 4.5 deft. This compares with $2.0 \pm$ 2.9 defs and 1.0 ± 1.4 deft for the remaining 30 patients who had little or no caries. Twelve patients demonstrated a caries pattern similar to nursing caries, that is, one involving maxillary incisor teeth or incisor and first primary molar teeth, and 10 of those patients had rampant or extensive caries.

Twenty of the 55 patients had permanent molars erupted, yet only two presented with any caries in the permanent teeth.

SCIENTIFIC ARTICLES

Discussion

The findings of this study demonstrate a rather high prevalence of dental caries in HIV-infected children, particularly those with a primary dentition who were age 5 or younger. Those children had far more dental caries in their primary teeth than a sample of normal children studied by Brunelle.⁶ Since the teeth did not appear to have any clinically evident enamel defects, it is postulated that the high prevalence of caries was due to local or systemic environmental factors rather than defective tooth formation. Yeh et al.⁷ suggested that there might be modifications to the salivary flow as found in adults. However, this was not clinically evident and was not investigated as part of our study. The children grew up in a nonfluoridated community and came from a low socioeconomic level in which parents or guardians had little awareness of proper oral health. These factors also might have influenced their dental decay.

Many of the children had received zidovudine for treatment of their disease and nystatin for treatment of *Candida* infection. Since both of those drugs contain relatively high concentrations of sucrose, the drug ingestion might have contributed to the dental caries. In addition, during infancy many of the patients had received a nutritional supplement that had a relatively high concentration of sucrose. The use of this supplement during infancy might explain the high prevalence of decay in the primary dentition with relatively little decay in the permanent teeth of the older patients. Since dietary histories for the patients were not available, the relationship between tooth decay and the use of sucrose-containing drugs and the dietary supplement could not be investigated properly. This relationship remains an important area for future study of dental caries in the HIV-infected child.

This study was approved by an internal review board, and all benefits and risks were explained fully to the parents or guardians of the patients involved.

Dr. Howell was a postdoctoral student, Department of Pediatric Dentistry, UMDNJ — New Jersey Dental School (currently in private practice at Provo, Utah). Dr. Jandinski is associate professor, Department of Oral Biology, Pathology and Diagnostic Sciences, UMDNJ — New Jersey Dental School. Dr. Palumbo is assistant professor, Division of Allergy, Immunology & Infectious Diseases Department of Pediatrics, UMDNJ — New Jersey Medical School and Children's Hospital of New Jersey. Dr. Shey is professor and Dr. Houpt is professor and chairman, Department of Pediatric Dentistry, UMDNJ — New Jersey Dental School.

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