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Mucocutaneous Fungal Colonization in HIV-infected Children

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The prevalence of symptomatic mucocutaneous candidiasis in HIV-infected children is well documented. Information, however, of the carriage rate of potential fungal pathogens is lacking. In this study we determined the fungal colonization rate of multiple mucocutaneous sites from 13 HIV-infected and 12 control children.

The rate of yeast and mould colonization and the species of fungal isolates were essentially the same for both groups of patients. However, several HIV-infected children asymptomatic for thrush proved to be colonized by *Candida albicans*, and disseminated colonization with *Trichosporon beigelii* occurred in one HIV-infected child. All cultures for dermatophytes were negative. While the carriage rate with fungi other than *C. albicans* was not increased in the HIV-infected group, the isolates recovered are known pathogens in the immunocompromised host and the colonization of these organisms may be a potential source of infection. *Key words: acquired immunodeficiency syndrome; Candida; Trichosporon; yeasts.*

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Pediatric acquired immunodeficiency syndrome (AIDS) was first reported to the Centers for Disease Control in 1982. To date, approximately 4,000 children under the age of 13 have been diagnosed, and the number is rapidly increasing. Symptoms may appear as early as the neonatal period or as late as 12 years of age. More than 50% are symptomatic by 2 years.

Cutaneous infection is among the most common manifestations of AIDS in children (1, 2) and adults (3, 4) and is often the presenting sign of the disease. Opportunistic infections due to *Candida*, dermatophytes and various other fungi have been well documented in HIV-infected adults (4, 5). In the pediatric population, only the prevalence of mucocutaneous candidiasis has been examined (6, 7).

In this preliminary study, HIV-infected children and normal controls were compared to determine their carriage rate with *Candida* and other potential fungal pathogens.

MATERIALS AND METHODS

Thirteen HIV-infected children predominantly in an outpatient setting and 12 randomly selected, normal children in an outpatient setting without risk factors for acquiring HIV-infection were examined. The presence of HIV-antibodies was detected by Enzyme Linked Immunosorbent Assay (ELISA) and confirmed by a positive Western blot. All HIV-infected children tested positive at birth and were still positive at the time of this study. After the age of 15–18 months, children who still test positive for the HIV-antibody are considered infected with the virus (2, 8, 9). The children ranged in age from 1 to 8 years and were composed of 7 Hispanics, 5 Blacks and 1 Caucasian, with 5 males and 8 females. One was asymptomatic for HIV-infection (Category N), 6 had mild symptoms (Category A) and 6 had moderate – severe symptoms (Category B–C). The control group ranged in age from 1 to 3 years and was composed of 10 Hispanics and 2 Blacks, with 6 males and 6 females.

All patients were examined for cutaneous changes. Cultures were taken of the scalp, oral mucosa, right and left feet, toe and fingerwebs, as well as bilateral axilla and groin. Cultures were taken by one person using sterile cotton swabs premoistened with sterile water. The swabs were rubbed vigorously over each site and then streaked in a serpentine pattern (10) onto Sabouraud dextrose agar (SDA) and Mycosel agar (SDA with chloramphenicol and cycloheximide, Becton Dickinson Microbiology Systems, Cockeysville, MD, USA). Mycosel agar inhibits mould growth and therefore decreased the risk of underestimation of the number of more slow-growing fungi (i.e. dermatophytes). Laboratory contamination was avoided by having the cultures handled by a single, experienced technologist using scrupulous methods. Cultures were identified microscopically and by biochemical assays.

RESULTS

The number of positive fungal cultures from each specific mucocutaneous site varied from patient to patient. In some instances, in both HIV-infected and non-infected children, no fungi were recovered from a specific site, while in others there was growth of up to 3 different organisms. The mucocutaneous surface of the 13 HIV-infected children yielded a total of 83 positive fungal cultures (6.4/patient), whereas the 12 control children had 56 positive cultures (4.7/patient). HIV-infected children were colonized with a similar species population of yeasts and moulds as non-infected children. The results are summarized in Table I. Follow-up cultures were not feasible.

One HIV-infected child, a 1-year-old black male with a history of multiple hospital admissions, failure to thrive, oral thrush and scattered skin-colored papules had disseminated colonization with *T. beigelii*, as evident by the isolation of the organism

Table I. *Frequency of yeast isolated from HIV-infected and control children*

Site/Organism	Number of children	
	HIV-infected ($n = 13$)	Control ($n = 12$)
Oral mucosa		
Candida albicans	10 (77%)	2 (17%)
Candida parapsilosis	1 (8%)	2 (17%)
Trichosporon beigelli	1 (8%)	1 (8%)
Cutaneous		
Candida albicans	6 (46%)	2 (17%)
Candida parapsilosis	5 (38%)	6 (50%)
Rhodotorula rubra	4 (31%)	1 (8%)
Trichosporon beigelli	3 (23%)	3 (25%)

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from all mucocutaneous sites. *C. albicans* was also recovered from most sites. The child was severely ill at the time of the study and subsequently died from *Mycobacterium avium-intracellulare* (MAI) sepsis.

A variety of moulds were recovered from both patient populations. Those most frequently isolated include: *Aspergillus glaucus, A. niger, Penicillium species* and *Rhizopus species*. The rate of mould colonization was 46% in the HIV-infected children and 66% for the control group.

DISCUSSION

In our study, *C. albicans* was isolated from the oral mucosa of 10 HIV-infected children, of which 8 did not have evidence of oral thrush. Two of these 8 patients were being treated with ketoconazole. None of the other patients were receiving antifungal therapy. Two of the remaining 6 patients on subsequent follow-up visits were noted to have developed thrush, but 4 remained asymptomatic. None of the children were being treated with zidovudine, which can decrease the incidence of oral thrush (13). The 4 asymptomatic patients were of different HIV-clinical stages; 1 was Category N, 2 were A and 1 was C. Our findings in the pediatric population are similar to those seen in a recent study of HIV-positive adults by Torssander et al. (14), where the authors found a significantly higher rate of asymptomatic oral candidal colonization in HIV-positive patients than in the HIV-negative controls. It must, however, be kept in mind that due to the multiple parameters involved and the limited number of subjects studied, the statistical significance of our observation could not be evaluated, and further investigation is needed.

In this preliminary study there was a high carriage rate of moulds irrespective of the patient's HIV status. The fact that both our study populations are from indigent means may have some bearing on this finding. A study of the mycotic flora of the toewebs of 27 healthy male volunteers also recorded a similar high carriage rate of moulds and yeasts, far greater than previously realized (15). Nevertheless, some of the organisms isolated from our HIV-positive patients, even if transient skin contaminants, have the potential to cause serious infection.

T. beigelli, the only fungus other than *C. albicans* and the dermatophytes whose incidence has been studied in adult patients with AIDS (16), is the causative agent of white piedra, an innocuous superficial hair infection. Jones et al. (16) cultured scrapings from axillary and inguinal skin of HIV-infected adults and found the overall prevalence of *T. beigelii* to be 14%, a rate equal to the general population. To our knowledge, ours is the first report of the carriage rate of *T. beigelii* in a pediatric population. In this preliminary study, the controls and the HIV-infected children were colonized at a similar rate, in agreement with Jones et al. (16).

Although *T. beigelii* may be present on the skin without evidence of infection, it has recently proved itself to be an invasive pathogen (17). A correlation between the carriage rate of *T. beigelii* and invasive infection in immunosuppressed patients has been reported (18). We are the first to describe the widespread mucocutaneous colonization of *T. beigelii* in a pediatric patient with AIDS.

This preliminary study shows that asymptomatic colonization of *C. albicans* does occur in the HIV-positive pediatric population, and while the carriage rate with other fungi is not increased, the organisms isolated, known fulminant pathogens in the immunocompromised host (19–21), may be a source of infection and mortality.

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