The patient’s renal function remained normal throughout this period. Initial empirical treatment of the urethritis with erythromycin was unsuccessful, and treatment with albendazole (400 mg twice daily for 4 weeks) was instituted. His symptoms abated rapidly, and 10 days after albendazole therapy was discontinued, it was not possible to detect microsporidian spores in his feces or urine or smears of urethral scrapings, nasal discharge, or sputum. He remains asymptomatic 8 months after completing treatment with albendazole.

Patients with disseminated microsporidiosis may have mild symptoms of have chronic disease [2]. Our patient, who presented with urethritis and sinusitis months after the initial finding of spores in his feces, may represent the latter pattern. Microsporidian infection of the nasal tissues, sinuses, and renal tract has been documented [2, 3], but this is the first case in which microsporidiosis has been associated with urethritis. The detection of spores in this patient’s sputum may reflect pulmonary microsporidian colonization, but in light of the urethral smear findings and his normal renal function, the significance of the presence of spores in the urinary deposit is uncertain.

The initial electron microscopic finding of *Encephalitozoon*-like spores in fecal specimens, as well as their repeated detection, suggests that *Septata intestinalis* was the cause of the disseminated infection in this case. *S. intestinalis* is of uncertain taxonomic status because the spores are identical in appearance on electron microscopy to those of the genus *Encephalitozoon*. Reclassification of *S. intestinalis* as *E. intestinalis* has been suggested on genetic and immunologic grounds [7].

Other important features of this case are the patient’s dramatic clinical response to albendazole and the subsequent inability to detect microsporidian spores. Experience with albendazole in the treatment of microsporidiosis has been variable, perhaps because therapeutic response may be species dependent, dose dependent, or both [1, 8]. The findings in this case suggest that albendazole is useful for treating at least some cases of microsporidiosis.

**Failure to Detect Human Papillomavirus DNA in Oral Mucosa of Postmenopausal Women**

The rate of detection of DNA from human papillomavirus (HPV) was studied in cytological scrapings of clinically normal oral mucosa from 135 postmenopausal women, 53 (39.3%) of whom received cyclic estrogen replacement therapy. All the women, who were 55 years old at presentation, participated in the annual mass-screening program for the detection of cervical precancerous lesions. The results of cervical Pap smears were normal in all cases except one in which a woman had not received cyclic estrogen replacement therapy; this woman had cytological atypia consistent with HPV infection. No HPV-positive controls were included in this study.

Oral smears were collected from three sites of a clinically healthy oral mucosa (i.e., buccal surface, palate, and tongue) with use of a sterile interdental brush. The cytological samples were analyzed by means of PCR with use of the general primers (GP05 and GP06). DNA for PCR analysis was extracted from the scrapings by means of a modification of the procedure described by Miller et al. [1]. The adequateness of the DNA sample was confirmed by PCR amplification of β-globin in all specimens [2]. DNA extracted from CaSki cells containing HPV-16 DNA (~600 copies/cell) served as a positive control. In negative controls no DNA was added to the reaction mixture. The detection level of PCR analysis was tested by using DNA extracted from CaSki cells (from 10,000 cells to 1 cell). The PCR reaction with even a single CaSki cell resulted in a faintly detectable band in the gel under ultraviolet light (figure 1).

β-Globin was amplified in 131 of 135 cytological scrapings of the oral mucosa. On the agarose gel, amplified DNA (526–595 bp in size) was shown in 14 (10.7%) of 131 specimens. HPV positivity could not be confirmed by Southern blot hybridization, indicating that the general primers for HPV can amplify some other sequences that have not been identified as yet [2].

The presence of HPV DNA in clinically normal oral mucosa has been demonstrated previously [4–6]. Current data suggest that HPV DNA in oral mucosa is mostly latent [7]. Jalal et al. [6] detected HPV 16 (a genital type) DNA in cytological scrapings from oral mucosa of 13 (43%) of 30 young adults by means of PCR analysis. Because the fact that basal cells cannot usually be collected by scraping of oral mucosa, latent infections remain undetected while confined to the basal cells. Furthermore, the

**References**


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extremely high sensitivity of PCR analysis bears some inherent problems of contamination, which undoubtedly have contributed to false-positive results in some previous studies. To increase the reliability of the results of PCR analysis, considerable attention has been paid to the requirements of the PCR technique proper and to strict laboratory discipline, which in fact has lead to substantially lower rates of detection of HPV DNA in more recent studies.

To our knowledge, the present study includes the largest cohort of healthy postmenopausal women analyzed so far for HPV infection in clinically normal oral mucosa; PCR analysis was used to detect HPV. The present study also focused on whether estrogen replacement therapy might be involved in activation of HPV in oral mucosa. Regulation and activation of latent HPV infections are not well understood, although it is evident that viral functions are regulated by the state of differentiation of the squamous cell tract are at the highest risk for HPV-related cancers [9]. It has been found that the estrogen-sensitive cells in the genital tract are at the highest risk for HPV-related cancers [8]. Although the present study failed to confirm that estrogen treatment or estrogen deficiency affects the prevalence of HPV infection in postmenopausal women, it cannot be excluded that hormonal activity has an influence on the mucosal properties of the oral cavity.

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