Fungi are important agents of human disease. The genus Candida gathers the most important fungal pathogens. They can cause a wide range of human diseases from superficial mucosal infections to life-threatening invasive infections. Normally, fungi are saprophytic residents of oral mucosa and the 40-60% of healthy adults harbour commensal Candida in their mouth without signs and symptoms of candidiasis [1]. The most common cause of oropharyngeal candidiasis is the polymorphic species Candida albicans. Candida dubliensis was identified in the Irish HIV infected and AIDS population in the early 1990s [2]. There are many phenotypic similarities between Candida albicans and Candida dubliensis that pose the problems in their identification and previously led to misidentification of these two species. Epidemiological studies have shown that Candida dubliensis is prevalent throughout the world and it is associated with oropharyngeal infections in patients with human immunodeficiency virus (HIV) virus [3].

Data acquired from its isolation in healthy and immunocompromised patients are variable and there is no still consensus on the epidemiological relevance of this species. It has been reported that non controlled glycemia predisposes to oral candidiasis in diabetic patients and the density of Candida growth is increased in patients with diabetes mellitus. The mechanism by which the diabetes predisposes to high oral concentration of Candida has not yet been established [1]. The Bharathi et al.’s [4] study is an interesting investigation which underlines the higher percentage of asymptomatic oral carriage of Candida in HIV-positive patients (54%) versus diabetic patients (44%) and healthy individuals (24%). These results confirm those ones from previous investigations [5-6], but interestingly the percentage of HIV-positive patients seems to be higher only in those individuals with a very low blood cell CD4+ count [7-8]. However, there’s an increasing need of further long-term longitudinal epidemiological studies worldwide in order to better establish the real incidence and prevalence of asymptomatic oral carriage of Candida in these three categories of patients, and ascertain whether or not race, sex, and age, other than immunocompetent status, might have any influence on carrier state. With these epidemiological data, we may try to prove conclusively whether or not there is any tight relationship between diabetes, HIV-positive status and oral carriage of Candida, who really runs a higher risk of developing such infections, how many of them develop candidiasis and why, and, last but not less important, whether or not any preventive treatment might be of some benefit.

REFERENCES


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