Candida albicans: Colonization, role and effects of this opportunistic pathogen on orthodontic appliances

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Candida albicans: Colonization, role and effects of this opportunistic pathogen on orthodontic appliances

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Abstract

Candida albicans is a commensal yeast that is normally present in the oral cavity of the population. This opportunistic pathogen is frequently isolated from the human mouth but its presence in the oral cavity is not indicative of disease. Colonization of the oral cavity by Candida albicans involves the acquisition and maintenance of a stable yeast population. Micro-organisms are continually being removed from the oral cavity by host clearance mechanisms, and so, in order to survive and inhabit this eco-system, Candida albicans cells have to adhere and replicate. Orthodontic and other oral appliances seem to favor candidal presence.

The purpose of this paper is to review the literature, with specific attention to colonization, intra-oral density of the candidal organisms and Candida carriage status in orthodontic patients during treatment.

Introduction

Candida albicans is a commensal yeast and its presence in the oral cavity is not indicative of disease. In fact, many individuals has Candida albicans as a minor component of their oral flora, and they have no clinical symptoms. Only a proportion of the population is colonized by Candida albicans, and only a subset of these individuals develops candidiasis.

Discussion

This yeast is normally present in small numbers in the oral flora: it is an opportunistic pathogen present in about 50-60% of the healthy human population, and becomes pathogenic when the host immune defence is undermined (for example in HIV infection). Therefore, oral candidiasis results from yeast overgrowth and penetration of the oral tissues when the host's physical and immunological defenses have been undermined. Tissue invasion may be assisted by secreted hydrolytic enzymes, hyphal formation, and contact sensing. While these and other phenotypic characteristics may endow certain Candida species or strains with a competitive advantage in the oral cavity, it is the host's immune competence that ultimately determines whether clearance, colonization, or candidiasis occurs.

Oral candidiasis presents clinically in many forms and this reflects the ability of the yeast to colonize different oral surfaces and the variety of factors which predispose the host to Candida colonization and subsequent infection. Oral presentations of candidiasis vary from the large white plaques of pseudomembranous candidiasis on the tongue and buccal mucosa to the palatal erythematous lesions of chronic atrophic candidiasis, and to angular cheilitis on the labial commissures (Samaranayake, 1990; Scully et al., 1994; Shay et al., 1997).

Several studies have reported that predisposing factors (such as mouth breathing and HIV-infection) may increase the variability of Candida species in the oral cavity: therefore, the health of an individual is a predisposing factor for its colonization. The oral cavity presents many niches for Candida albicans colonization, and the yeast is able to adhere to a plethora of ligands. A large number of sites in the oral cavity can be colonized; in healthy individuals, Candida albicans is most commonly isolated from the midline of the middle and posterior thirds of the tongue, the cheek, or the palatal mucosa (Arendorf and Walker, 1979, 1980; Borromeo et al., 1992).

Colonization of the oral cavity by this yeast can be defined as the acquisition and maintenance of a stable population of Candida albicans cells which does not give rise to clinical disease. It depends on the rate of yeast cells enter the oral cavity, their growth, and removal of cells from the mouth by swallowing and oral hygiene.

Micro-organisms are continually being removed from the oral cavity by host clearance mechanisms, and so, in order to survive and inhabit this eco-system, Candida albicans cells have to adhere and replicate.

The following is an example scheme:

- Rate of removal > rate of acquisition and growth → clearance.
• Rate of removal = rate of acquisition and growth → colonization.
• Rate of removal < rate of acquisition and growth (and there is tissue damage) → candidiasis.

In the table I are listed several factors that influence candidiasis and colonization of the oral cavity by Candida albicans.

1. Acquisition: in humans, Candida albicans preferentially colonizes mucosal surfaces, and the intestinal tract is believed to be a major reservoir for infection (Odds, 1988; Cole et al., 1996). Candida albicans can colonize practically any site in the gastrointestinal tract, from the oral cavity to the rectum and peri-anal tissues, allowing anal-oral inoculation to occur (Soll et al., 1991). This yeast survives better on moist surfaces than dry inanimate objects, but if the degree of contamination is high enough, viable cells will remain on dry surfaces for at least 24 hours. Candida species can enter the oral cavity by manual inoculation, saliva transfer, or contaminated food and drink.

2. Maintaining an oral Candida population: the entry of Candida cells into the oral cavity is not sufficient for colonization, but they must be stably maintained. Since the oral cavity is a continuous-flow environment, yeast cells will be washed out by saliva and swallowed unless they adhere and replicate. Adhesion is therefore of critical importance in colonization and it is mediated between moieties of the Candida cell wall and host surfaces.

3. Adherence to oral surfaces: Candida albicans can adhere in a number of surfaces in the oral cavity (buccal epithelial cells, inert polymers of dental prostheses, teeth, and other oral micro-organisms). Colonization may contribute to the deterioration of the oral devices. This yeast showed a great adherence to acrylic and adherence is increased on rough acrylic and silicone rubber surfaces compared with smooth surfaces (Verran and Maryan, 1997). The acrylic base for dentures supported less adherence of C. albicans than tissue conditioners and a soft liner (Okita et al., 1991).

Many factors may influence the process of adherence to oral epithelial cells (Table II).

The effects of these components on the adherence of Candida albicans differ: some of them increase the adhesion capacity, whereas others show inhibitory activity. Biasoli et al. observed a correlation between the capacity for yeast to adhere and its ability to colonize mucosal surfaces. Candida presents the highest values of adherence to oral epithelial cells relative to other Candida species. Boshet al. verified that moderate stress may affect the process of microbial colonization and the adherence of this yeast to epithelial cells by altering the secretory activity of salivary glands. Because adherence is an important virulence factor in Candida, the inhibition of this process is an important strategy in the prevention of oral candidosis. Its antigens, host proteins, anti fungal agents and antibodies have been used to inhibit Candida albicans adherence to host cells. IgA seems to play an important role by causing fungal aggregation and preventing the adherence to mucosa or oral surfaces.

4. Growth: In order to maintain Candida populations in the oral cavity, cells must grow and multiply at a rate at least equal to that of clearance. Anymetabolic activity that helps this yeast acquire carbon or nitrogen will aid its growth and survival in the oral cavity. Competition with other oral micro-organisms for nutrients, such as glucose, affects the growth rate of Candida cells. It is recognized that antibiotic treatment, which reduces the number of oral bacteria, is a predisposing factor for oral candidiasis (Samaranayake, 1990). Oral bacteria are present in most oral sites at concentrations much higher than Candida albicans, and so the Candida cells must compete with them for adhesion sites and nutrients, and be exposed to bacterial toxins and byproducts.

5. Evading host clearance mechanisms: immune system defects are a major risk factor for candidiasis. Innate defenses include the epithelial barrier and anti-candidal compounds in saliva (such as lysozyme, histatins, lactoferrin, and calprotectin). Acquired immunity includes the production of immunoglobulins and, if tissues are penetrated, the involvement of macrophages and polymorphonuclear leukocytes. The major immunoglobulin in saliva is secretory IgA.

As already said, the primary etiological agent of oral candidiasis is the yeast Candida albicans; however, other species that cause disease less commonly include Candida tropicalis, Candida glabrata, Candida krusei, Candida parapsilosis, Candida guilliermondii and Candida dubliniensis.

Among the many factors that contribute to the higher prevalence of Candida albicansin the oral cavity are its excellent ability to adhere and the presence of many cell receptors, which confer versatility and resistance to removal by the fluids that bathe these surfaces. Adhesion and colonization of the oral cavity by Candida albicans is an initial step in candidosis. The pathogenicity of Candidaspp. is due to enzyme production, tissue invasion, and their capacity to adhere to oral mucosa.
Orthodontic and other oral appliances seem to favor candidal presence, so we will analyze the relationship between this yeast and orthodontic patients. The presence of orthodontic and other oral appliances seems to alter the oral ecological environment. Hence, these appliances may tip the balance to favor the existence of Candida species. Biofilms on removable orthodontic appliances act as reservoir of microorganisms, capable of modifying the environmental condition of oral cavity and are difficult to be removed with routine hygiene measures.

Topaloglu-Ak et al. (2011) showed that mutans and Lactobacillus sp. counts increased significantly 6 months after the insertion of fixed/removable orthodontic appliances in the oral cavity. A significant increase for Candida albicans presence was noted after 3 months compared with baseline for fixed appliances. Long-term utilization of orthodontic appliances may have a negative effect on microbial flora and increase the risk of new carious lesions and periodontal problems.

Silva et al. (2013) compared the presence of Candida species in saliva and the levels of anti-Candida albicans IgA in children with or without orthodontic appliances. The results showed that Candida albicans is the species most frequently isolated from the oral cavities of patients in both groups, followed by Candida tropicalis. No correlation was observed between the level of anti-Candida albicans IgA in saliva and the presence of this yeast in its adherence to epithelial cells. In this study, the quantity of IgA was low in children of both the experimental group (who were users of removable orthodontic appliances for at least 6 months) and the control group (who were not users of any orthodontic appliances). Salivary IgA generally increases with age because the secretory immunological mechanism develops simultaneously with the humoral immune system. In patients with dental prosthesis or removable orthodontic devices, salivary IgA reduces the adherence of Candida albicans to polystyrene. Their results indicate that anti-Candida albicans IgA is not the most important factor to determine Candida carrier status.

Hence, Candida colonization is a consequence of orthodontic treatment and can lead to oral candidosis as a complication of removable appliance treatment. The installation of metal devices leads to an increase in the salivary concentration of metal ions and in the growth of salivary Candida spp. Ronsani et al. (2011) examined the relationship between released metal ions (Ni++, Fe++, Cr++, Co++ or a mixture of these metal ions) and Candida virulence, in order to evaluate whether metal ions affect fungal virulence. The results revealed that all ions, except Co++, caused increases in biofilm biomass. Their results indicate that metal ions released during the degradation of orthodontic appliances can modulate virulence factors in C. albicans biofilms.

Therefore, during orthodontic treatment, it is important to minimize colonization to prevent active infection that could consequently interfere with treatment. Hygiene is the most important factor in managing colonization. In their study, Decelis et al. (2012) tested the efficacy of NitrAdine to reduce Candida and they concluded that it may reduce the Candida burden in maxillary removable appliances. Carvalhinho et al. (2012) analyzed the susceptibilities to antifungal agents (fluconazole, econazole, miconazole and ketoconazole, amphotericin B and nystatin), mouth rinses and essential oils of patient's mouth with fixed orthodontic appliances. The results showed that all isolates tested were susceptible to amphotericin B, nystatin and fluconazole; one isolate was resistant to econazole (2.5%) and the other to ketoconazole (2.5%). Econazole and ketoconazole had the highest percentages of susceptible dose dependent (SDD), 55 and 95%, respectively. The study of mouth rinses showed a high variability of efficacy against C. albicans. The results showed that the isolates susceptibility to essential oils differed. The profile activity was: cinnamon > laurel > mint > eucalyptus > rosemary > lemon > myrrh > tangerine. The susceptibility of econazole-SDD isolates to cinnamon and lemon was higher than those of the econazole-S yeasts. In contrast, econazole-SDD isolates were less affected by laurel than econazole-S counterparts.

Results and Conclusions

The limited amount of literature demonstrated that the density of Candida increases; the most common Candida species isolated in the orthodontic patients is Candida albicans and that there seems to be a direct relationship between the presence of a removable appliance, Candida and low salivary pH levels. It is important to emphasize that no healthy patients developed Candida infection from the orthodontic appliances. However, there seems to be a trend that some non-Candida carriers converted to Candida carriers following the insertion of the appliances by unknown mechanism. This may indicate a more cautious approach when providing orthodontic treatments to immunocompromised children concerning the possible increased risk of candidal
infection.

More opportunistic bacteria and fungi are detected in orthodontic patients than in non-orthodontic patients. Opportunistic bacteria adhere to saliva-coated metallic brackets to the same degree as oral streptococci. The isolation frequencies of opportunistic bacteria and fungi increase during orthodontic treatment, suggesting the importance of paying special attention to oral hygiene in orthodontic patients to prevent periodontal disease and the aggravation of systemic disease in immunocompromised conditions.

Moreover, the use of dental devices significantly increased the prevalence of yeasts in periodontal pockets in patients presenting gingivitis.

In conclusion, orthodontic appliances may favor the adherence of Candida to epithelial cells but do not influence the presence of these yeasts in saliva, and the levels of anti-Candida albicans IgA do not correlate with yeast adherence or presence of this yeast in the oral cavity.

Patients should be recalled within short time intervals to be motivated for oral hygiene during their orthodontic therapy.

References


Illustrations
Illustration 1

Table I: Factors that influence candidiasis and colonization of the oral cavity.

<table>
<thead>
<tr>
<th>Candidiasis</th>
<th>Colonization</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tissue colonized</td>
<td>Acquisition (entry of cells into the oral cavity)</td>
</tr>
<tr>
<td>Virulence factors expressed by the Candida cells</td>
<td>Attachment and growth of cells</td>
</tr>
<tr>
<td>Host response</td>
<td>Penetration of tissues</td>
</tr>
<tr>
<td></td>
<td>Removal of cells from the oral cavity</td>
</tr>
</tbody>
</table>
Illustration 2

Table II: Factors that influenced the adherence of Candida to oral epithelial cells

<table>
<thead>
<tr>
<th>Yeast-related factors</th>
<th>Host-related factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>expression of adhesion proteins</td>
<td>sexual hormones</td>
</tr>
<tr>
<td>presence of germinative tubes</td>
<td>presence of fibrin and fibrinogen</td>
</tr>
<tr>
<td>production of extracellular polymers and enzymes</td>
<td>presence of salivary compounds (including mucine, salivary proteins, and secretory IgA)</td>
</tr>
</tbody>
</table>