

# ***Candida Albicans* Colonization of Dental Plaque in Elderly Dysphagic Patients**

Efraim Aizen MD<sup>1</sup>, Paul A. Feldman MD MSc<sup>2</sup>, Ralph Madeb MD<sup>2</sup>, Jordan Steinberg MSc<sup>2</sup>, Steven Merlin MD MSc<sup>3</sup>, Edmond Sabo MD<sup>3</sup>, Valery Perlov MD<sup>1</sup> and Isaac Srugo MD<sup>2</sup>

<sup>1</sup>Fliman Geriatric Hospital, Haifa, Israel

Affiliated to Technion Faculty of Medicine, Haifa, Israel

<sup>2</sup>Department of Clinical Microbiology, Bnai Zion Medical Center, Haifa, Israel

<sup>3</sup>Department of Pathology, Carmel Medical Center, Haifa, Israel

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## **Abstract**

**Background:** Dysphagia is a common disorder among the elderly population. As many as 50% of nursing home residents suffer from dysphagia. It is important to identify patients at increased risk for colonization of dental and denture plaque by pathogenic organisms in order to prevent associated disease.

**Objectives:** To quantify the prevalence and evaluate the effect of dental and denture plaque colonization by *Candida albicans* in hospitalized elderly dysphagic patients as a complication of stroke, as well as the effect of systemic antimicrobial therapy on *C. albicans* colonization in these patients.

**Methods:** We evaluated dysphagia and antibiotic therapy as risk factors for dental and denture plaque colonization by *C. albicans* in elderly stroke rehabilitating patients with dysphagia, as compared to elderly non-dysphagic stroke and non-stroke rehabilitating patients on days 0, 7 and 14 following admission to the Fliman Geriatric Rehabilitation Hospital.

**Results:** The risk of *C. albicans* colonization of dental plaque was greater in dysphagic patients than in those without dysphagia on day 0 (50% vs. 21%,  $P = 0.076$ ), day 7 (58 vs. 15.2%,  $P = 0.008$ ) and day 14 (58 vs. 15.2%,  $P = 0.08$ ). Similarly, patients on antibiotic therapy were at greater risk for *C. albicans* colonization of dental plaque on day 0 (56 vs. 11%,  $P = 0.002$ ), day 7 (44 vs. 14.8%,  $P = 0.04$ ) and day 14 (39 vs. 19%,  $P = 0.18$ ). The risk of *C. albicans* colonization of denture plaque as opposed to dental plaques in non-dysphagic patients was significantly greater on day 0 (45.7 vs. 21.2%,  $P = 0.03$ ), day 7 (51.4 vs. 15.1%,  $P = 0.0016$ ) and day 14 (54.3 vs. 15.1%,  $P = 0.0007$ ). Dysphagia did not increase the risk of denture plaque colonization by *C. albicans*.

**Conclusions:** Both dysphagia and antibiotic therapy are risk factors for *C. albicans* colonization of dental plaque, and although dysphagia does not significantly increase colonization of denture plaque, denture wearers are at greater risk of such colonization.

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Dysphagia or swallowing dysfunction is a common disorder among the elderly. As many as 50% of nursing home residents suffer from dysphagia. Many of them have neurologic disease [1]. Dysphagia has been estimated to occur in 25–32% of patients as a complication of stroke [2]. The condition may result in severe complications, some of which are life-threatening. Patients suffering from acute or chronic dysphagia are at increased risk of aspiration pneumonia and death [3–5]. It has been estimated that

aspiration pneumonia occurs in 12–30% of stroke rehabilitation patients and is a significant cause of morbidity and mortality [5,6].

In elderly patients dental and denture plaque serve as a major reservoir for pathogenic microorganisms, many of which are common to the oral microflora. The continuous swallowing or aspiration of these microorganisms exposes patients to risk of infection, particularly in immunocompromised patients and the medicated elderly [7]. Denture plaque containing *C. albicans* causes not only oral candidiasis or denture-induced stomatitis but also root caries and periodontitis [7]. Colonization of dental and denture plaque by pathogenic microorganisms has been associated with several risk factors.

Patients with a low salivary secretion rate have an increased rate of plaque accumulation associated with an increase in the number of lactobacilli and *Streptococcus mutans* [8]. Poor oral hygiene and lack of mechanical elimination also contribute to proliferation and accumulation of dental and denture plaque and subsequent colonization and infection. High dental and denture yeast counts in elderly patients living in institutions are associated with poor oral hygiene [9]. Furthermore, old age is associated with changes in dental plaque and salivary microflora, resulting in higher proportions of lactobacilli, staphylococci and yeast [10,11]. It is important to identify patients at increased risk for colonization of dental and denture plaque by pathogenic organisms in order to prevent associated diseases. Elderly patients with dysphagia as a complication of stroke are at increased risk due to difficulties in oral hygiene, lack of mechanical elimination and poor salivary secretion.

Antibiotic therapy is commonly administered to hospitalized elderly patients for nosocomial infection. With their potential for altering the normal flora of the oral cavity, antibiotics may also influence plaque colonization. Our prospective study evaluated the prevalence of dental and denture plaque colonization by *C. albicans* in hospitalized elderly dysphagic patients as a complication of stroke and the effect of systemic antimicrobial therapy on *C. albicans* colonization in these patients.

## **Patients and Methods**

### **Study population**

Ninety-four patients were prospectively enrolled in our study, all of

whom were admitted to the Fliman Geriatric Rehabilitation Hospital between 1 April and 30 September 1998. They were divided into three groups: a) 26 stroke rehabilitation patients who suffered from dysphagia, b) 39 stroke rehabilitation patients without dysphagia, and c) 29 elderly rehabilitation patients without stroke (admitted for orthopedic rehabilitation). Each group was subsequently divided into two subgroups: presence or absence of antimicrobial treatment, and presence or absence of dentures (complete or partial). Patients with dysphagia received daily rinses with sodium bicarbonate dentifrice. Those receiving other medications that might have influenced saliva secretion (anticholinergics, antihistaminics, barbiturates) or other medication that might have affected their oral state (e.g., cytotoxic chemotherapy) were excluded. We also excluded patients suffering from conditions adversely affecting the oral state and salivary condition (xerostomia, Sjogren's syndrome, autoimmune exocrinopathies, dermatologic conditions affecting the oral mucosa, Parkinson's disease and cancer). There was no evidence of oral candidiasis (white plaques on the tongue or buccal mucosa, which can be scraped off easily) in any of the patients. Informed consent was obtained from all patients or their relatives.

#### Collection of samples

Samples of the dental or denture plaque were taken within 24 hours of admission (day 0) and then on days 7 and 14. Samples from supragingival molar plaque were collected above the gingiva on the same tooth by running a standard dental probe along the buccal surface. All plaque samples were processed within 2–4 hours after collection. Samples of denture plaque and dental plaque were not collected from the same patient.

#### Microbiologic procedures

Plaque samples were aseptically placed in an anaerobic chamber with Amis gel (Eurotube, Spain). Samples were then spiral-plated on PRAS tryptic soy agar plates containing Sabouraud agar and chromo agar (Hylabs, Israel). Each isolate was identified by means of colony morphology and biochemical reaction (Biomérieux, France). *C. albicans* was differentiated from other species of candida using the ID32C system (Biomérieux).

#### Antimicrobial treatment

Thirty-four (36.2%) patients received antimicrobial therapy for infection on day 0, which was continued for a minimum of 5 days. Twenty-five (26.6%) patients had urinary tract infection, 2 (2.1%) had cellulitis, 3 (3.2%) had upper respiratory tract infection and 2 (2.1%) had pneumonia. Of these patients 27.5% received cefuroxime, 20% amoxicillin + clavulanic acid, 12.5% ceftriaxone, 12.5% ciprofloxacin, 12.5% ofloxacin, 5% cloxacillin, 5% trimethoprim + sulfamethoxazole, and 2.5% tobramycin.

#### Statistical analysis

Association between categorical variables were tested using the chi-square test or the

Fischer's exact test when appropriate. Two-tailed *P* values of 0.05 or less were considered statistically significant.

## Results

Ninety-four patients (58% male) were included in the study. The main characteristics of the patients are summarized in Table 1. The mean length of time patients received antibiotic treatment did not differ among the groups.

Frequency rates of dental plaque colonization by *C. albicans* in dysphagic and non-dysphagic patients with or without antibiotic treatment on days 0, 7 and 14 are shown in Table 2. The frequency of colonization of dental plaque by *C. albicans* was significantly higher in the dysphagic stroke rehabilitating patients than in the non-dysphagic patients on day 7 (7/12, 58% vs. 5/33, 15.2%; *P* = 0.008) and day 14 (7/12, 58% vs. 5/33, 15.2%; *P* = 0.08). On day 0, there was a trend towards significance (6/12, 50% vs. 7/33, 21%; *P* = 0.076). Similarly, the frequency of dental plaque colonization by *C. albicans* was significantly higher in patients on antibiotic therapy than in those not on antibiotics on day 0 (10/18, 56% vs. 3/27, 11%; *P* = 0.002) and day 7 (8/18, 44% vs. 4/27, 14.8%; *P* = 0.04). On day 14, there was a trend towards significance (7/18, 39% vs. 5/27, 19%; *P* = 0.18). Frequency rates of denture and dental plaque colonization by *C. albicans* in non-

**Table 1.** Main characteristics of the rehabilitating patients

Characteristics	Dysphagia stroke rehabilitating	Non-dysphagic stroke rehabilitating	Non-dysphagic non-stroke rehabilitating
No.	26	39	29
Mean age (yrs)	71.35 ± 1,058–89	72.07 ± 1,358–96	75.8 ± 13
Range	58–89	58–96	65–91
Antibiotic therapy	13	12	9
Wearing dentures (with antibiotics)	14 (7)	18 (5)	17 (4)

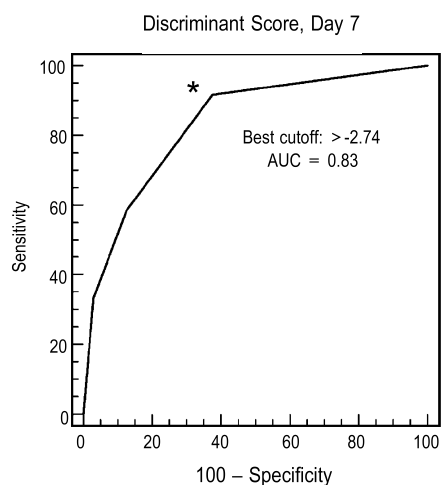
**Table 2.** Frequency rate of dental plaque colonization by *Candida albicans*

	Day 0	Day 7	Day 14
(% per group)			
<b>Dysphagia</b>			
With antibiotics (n=6)	4 (66.7)	4 (66.7)	4 (66.7)
Without antibiotics (n=6)	2 (33.3)	3 (50)	3 (50)
<b>Non-dysphagia</b>			
With antibiotics (n=12)	6 (50)	4 (33.3)	3 (25)
Without antibiotics (n=21)	1 (4.8)	1 (4.8)	2 (9.6)

**Table 3.** Frequency rate of denture versus dental plaque colonization by *Candida albicans* in non-dysphagic patients

	Denture (n=35)	Dental (n=33)	<i>P</i> value*	OR (95% confidence interval)
Day 0 (% per group)	16 (45.7)	7 (21.2)	0.03	3.13 (1.076–9.093)
Day 7 (% per group)	18 (51.4)	5 (15.1)	0.0016	5.93 (1.859–28.909)
Day 14 (% per group)	19 (54.3)	5 (15.1)	0.0007	6.65 (2.083–21.233)

\* *P* values computed by univariate chi square analysis



**Figure 1.** Receiver operating characteristic curve showing the best cutoff for depicting the discriminant score on day 7. An area under the curve (AUC) greater than 0.5 is considered to have predictive power.

dysphagic patients are given in Table 3. Colonization by *C. albicans* of denture plaque was significantly greater than of dental plaque in non-dysphagic patients on days 0, 7 and 14. The odds ratio ranged between 3 and 6. The receiver operating characteristic curve showed that the best cutoff for depicting the discriminant score was on day 7 [Figure 1].

The difference between denture plaque and dental plaque colonization in the dysphagic group was not statistically significant. Dysphagia and the use of antimicrobial therapy did not significantly affect the frequency of denture plaque colonization by *C. albicans*. The difference in frequency between the non-dysphagic stroke rehabilitating group and the non-dysphagic non-stroke rehabilitating group was not statistically significant.

## Discussion

Elderly patients suffering from dysphagia as a complication of stroke are at higher risk for aspiration of microorganisms from dental or denture plaque and for subsequent infection. These patients have difficulty maintaining oral hygiene and manifest reduced mastication ability and food bolus formation as well as reduced salivary secretion [13]. The result is a stagnant environment in the oral cavity, which fosters the formation of plaque and consequent colonization.

Dysphagic patients may also experience an alteration in the quantity and quality of saliva. Saliva is critical for maintaining oropharyngeal health [14,15]. It maintains a nearly neutral pH and contains the non-specific antimicrobial factors: lactoferrin, lysozyme and lactoperoxidase, immunoglobulin A and fungistatic histidine-rich polypeptides [16,17]. In addition, saliva helps maintain oral health through epidermal growth factor and lubrication of the tissues with a variety of glycoproteins [14]. Furthermore, the quality and quantity of saliva has been shown to play an important role in the host defense against *C. albicans* infection [16]. In our study, dysphagia was shown to be a risk factor for *C. albicans* colonization of dental plaque.

Antimicrobial therapy may result in suppression of the normal oral bacterial flora with an overgrowth of endogenous *C. albicans* [18]. It is believed that antimicrobials limit bacterial adherence to oral epithelium, thereby providing an opportunity for *C. albicans* colonization [19]. Furthermore, studies have shown that lactobacilli may interfere with the adhesion of *C. albicans* to fibrous material and epithelial cells [20]. However, in long-term hospital care patients, the level of mucosal and denture colonization by *C. albicans* bore a direct correlation with salivary counts of lactobacilli [9]. In our study, antibiotic treatment was shown to be a risk factor for *C. albicans* colonization of dental plaque. Although not all antimicrobials place patients at increased risk of colonization by fungi, the majority of those used in our study has been proven to produce such a result [18]. Dysphagic patients on antimicrobial therapy were found to be at even greater risk for *C. albicans* colonization of dental plaque than when either variable was evaluated separately (data not shown).

*C. albicans* is known to colonize the oral cavity in 30–45% of healthy asymptomatic patients [21]. Our study demonstrated a higher prevalence of *C. albicans* colonization of denture over dental plaque in the non-dysphagic patients. Denture wearers were at a three- to sixfold increased risk of colonization. These results are consistent with studies showing greater prevalence of yeast in denture wearers [22]. The presence of a denture, even a removable partial denture, impedes the flow from minor salivary glands and the free exchange of oxygen. The resulting low pH level and anaerobic environment facilitates the growth of *C. albicans*, especially on the intaglio (tissue-fitting) surface of dentures [22]. The prevalence of denture plaque colonization by *C. albicans* in dysphagic patients in our study did not differ significantly from that in non-dysphagic patients. Thus, while denture wearers are at greater risk of colonization, that risk is not increased with dysphagia. The inference to be drawn is that both dysphagia and the presence of dentures, given their similar limiting effects on the dynamic forces operating within the oral cavity, tend toward a similar result.

The frequent prevalence of oral candidiasis in elderly patients living in institutions has been associated with high dental and denture yeast counts [9]. Although *C. albicans* is responsible for many oral lesions, the patients in our study had no evidence of oral candidiasis or any other candida-related infections. Nonetheless, it is highly probable that *C. albicans* colonization of dental or denture plaque increases susceptibility to infection in stroke dysphagic patients.

Our findings show that despite standard oral care, the colonization by *C. albicans* increased in dysphagic patients following stroke, especially in patients on antimicrobial therapy and in denture wearers. Increased focus on methods of achieving higher levels of oral hygiene in elderly dysphagic patients can assist in reducing plaque colonization by *C. albicans* and reduce many of the risks associated with such colonization.

## References

1. Siebens H, Trupe E, Siebens A, et al. Correlates and consequences of eating dependency in institutionalized elderly. *J Am Geriatr Soc* 1986;34:192–8.

2. Groher ME, Bukatman R. The prevalence of swallowing disorders in two teaching hospitals. *Dysphagia* 1986;1:3–6.
  3. Niederman MS. Nosocomial pneumonia in the elderly patient: chronic care facility and hospital consideration. *Clin Chest Med* 1993;14:479–90.
  4. Walker AW, Robins M, Weinfeld FD. Clinical findings: the national survey of stroke. *Stroke* 1981;12:13–37.
  5. Horner J, Massey EW, Riski JE, Lathrop DL, Chase KN. Aspiration following stroke: clinical correlates and outcome. *Neurology* 1988;38:1359–62.
  6. Teasell RW, McRae M, Marchuk Y, Finestone HM. Pneumonia associated with aspiration following stroke. *Arch Phys Med Rehabil* 1996;77:707–9.
  7. Nikawa H, Hamada T, Yamamoto T. Denture plaque – past and recent concerns. *J Dent* 1998;26:299–304.
  8. Almstahl A, Wikstrom M. Oral microflora in subjects with reduced salivary secretion. *J Dent Res* 1999;78:1410–16.
  9. Budtz-Jørgensen E, Mojon P, Banon-Clement JM, Baehni P. Oral candidiasis in long-term hospital care: comparison of edentulous and dentate subjects. *Oral Dis* 1996;2:285–90.
  10. Terpenning M, Bretz W, Lopatin D, Langmore S, Dominguez B, Loesche W. Bacterial colonization of saliva and plaque in the elderly. *Clin Infect Dis* 1993;16:S314–16.
  11. Percival RS, Challacombe SJ, Marsh PD. Age related microbiological changes in the salivary and plaque microflora of healthy adults. *J Med Microbiol* 1991;35:5–11.
  12. Fisher LD, Van Belle G Biostatistics. A methodology for health sciences. *Wiley Interscience* 1993:630–55.
  13. Selley WG. Swallowing difficulties in stroke patients: a new treatment. *Age Ageing* 1985;14:361–5.
  14. Atkinson JC, Fox PC. Salivary gland dysfunction. *Clin Geriatr Med* 1992;8:499–511.
  15. Stuchell RN, Mandel ID. Salivary gland dysfunction and swallowing disorders. *Otolaryngol Clin North Am* 1988;21:649–61.
  16. Epstein JB, Truelove EL, Izutu KL. Oral candidiasis: pathogenesis and host defense. *Rev Infect Dis* 1984;6:96–106.
  17. Driscoll J, Duan C, Zuo Y, Xu T, Troxler R, Oppenheim FG. Candidacil activity of human salivary histatin recombinant variant produced by site-directed mutagenesis. *Gene* 1996;177:29–34.
  18. Budtz-Jørgensen E. Etiology, pathogenesis, therapy, and prophylaxis of oral yeast infection. *Acta Odont Scand* 1990;48:61–9.
  19. Jobbins J, Bagg J, Parsons K. Oral carriage of yeasts, coliforms and staphylococci in patients with malignant disease. *J Oral Pathol Med* 1992;21:305–8.
  20. Reid G, Tieszner C, Lam D. Influence of lactobacilli on the adhesion of *Staphylococcus aureus* and *Candida albicans* to fibers and epithelial cells. *J Int Microbiol* 1995;15:248–53.
  21. Arendorf TM, Walker DM. The prevalence and intra-oral distribution of *Candida albicans* in man. *Arch Oral Biol* 1980;57:37–40.
  22. Cardash HS, Helft M, Shani A, Marshak B. The prevalence of *Candida albicans* in denture wearers in an Israeli geriatric hospital. *Gerodontology* 1989;4:101–7.
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- Correspondence:** Dr. E. Aizen, Dept. of Geriatrics, Fliman Geriatric Hospital, P.O. Box 2263, Haifa 31021, Israel.  
Tel. (972-4) 830-7162  
Fax: (972-4) 822-6017  
email: eaizen\_il@yahoo.com