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ORIGINAL ARTICLE

Microbial profile of the oral cavity of patients under mechanical ventilation is not influenced by the edentamento: an observational study

Perfil microbiano da cavidade oral em pacientes sob ventilação mecânica não é influenciado pelo edentameto: um estudo observacional

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Abstract

Objective: Understand whether the collection site (toothless or toothless) influences the frequency of bacteria in the oral cavity. It was performed as an observational, prospective, and cross-sectional study. **Methods:** Clinical samples of the oral surfaces of the teeth and/or cheek mucosa were collected in the oral cavity of 37 patients who underwent elective cardiac surgery in the preoperative period from May to July 2019. The clinical samples collected were subjected to identification of colonies and antimicrobial sensitivity tests. **Results**: It was observed that regardless of whether the collection site is toothless or toothless, the microbial profile, socio-demographic variables, comorbidities, and risk factors do not statistically influence the choice of the collection site. **Conclusions**: there wasn't statistical difference between the strains found at the collection sites. Practical Implications: the result found is relevant for other researchers that will work with oral cavity collections since the chosen collection site will not influence the frequency of strains found.

Keywords: Dental Plaque; Bacteria; Oral Health; Infection; Biofilms.

Resumo

Objetivo: Compreender se o local de coleta (com dentes ou desdentado) influencia na frequência de bactérias na cavidade oral. Foi realizado como um estudo observacional, prospectivo e transversal. **Métodos**: Amostras clínicas das superfícies orais dos dentes e/ou mucosa jugal foram coletadas na cavidade oral de 37 pacientes submetidos à cirurgia cardíaca eletiva no período pré-operatório de maio a julho de 2019. As amostras clínicas coletadas foram submetidas à identificação de colônias e testes antimicrobianos de sensibilidade. **Resultados**: Observou-se que independente do local de coleta ser dentado ou desdentado, o perfil microbiano, variáveis sociodemográficas, comorbidades e fatores de risco não influenciam estatisticamente na escolha do local de coleta. **Conclusões**: Não houve diferença estatística entre as cepas encontradas nos locais de coleta. O resultado encontrado é relevante para outros pesquisadores que trabalharão com coletas de cavidade oral, pois o local de coleta escolhido não influenciará na frequência de cepas encontradas.

Palavras-chave: Placa Dentária; Bactérias; Saúde Bucal; Infecção; Biofilmes.

INTRODUCTION

The oral cavity is a sterile human site until birth, when it changes over time[1,2,3]. The introduction of microorganisms may occur during birth, through contact with the microbiota of the amniotic cavity, aspiration of contaminated amniotic fluid, contact with microorganisms present in the birth canal, genital secretions, or maternal feces[1]. At birth or soon after birth, colonization begins[3]. Species of the genus *Streptococcus* are the first to colonize teeth and are initiators of acquired film development[4]. The metabolic activity of genera pioneers in adherence to the oral environment provides ideal conditions for colonization by other microbial species[5]. With aging, when all teeth are lost, the microbiota becomes similar to that of a child before the onset of tooth eruption[1].

The human oral cavity is an environment where many microorganisms inhabit, and contains almost half of all the microbiota in the human body[6]. Different sites within the oral cavity have a characteristic microbial composition, corresponding to the different biological and physicochemical characteristics mentioned above and specific to each sit[2,7]. In the mouth, there is a diverse amount of microorganisms, including viruses, fungi, archaeans, and even protozoa, with the bacterial group being the most predominant[2,8].

Microorganisms on tooth surfaces tend to form multi-species biofilms, which are incorporated into an extracellular matrix[7]. Biofilms are complex communities of microorganisms that

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produce a protective and adhesive matrix of glycocalyx[9]. Dental plaque consists of a complex and dynamic biofilm formed on the supra and subgingival surfaces of the teeth, oral mucosa, and dorsum of the tongue[10]. Teeth are the only uncoated surfaces in the body where bacterial levels can reach over[10,11] microorganisms per mg of dental plaque in healthy individuals[11]. Dental plaque is home to about 500 different bacterial species, varying according to local biological and physicochemical characteristics[2,6].

Oral microbiota grows and increases in diversity over time until its composition reaches a balance between the resident microflora and local environmental conditions. The resident microbiota acts as a barrier to transient / exogenous organisms[2,8,12]. However, changes in the environment, due to changes in diet, hormone levels, and oral hygiene, for example, can break this balance[2]. However, when this balance is disturbed, there is an inhibition of the growth of beneficial species and selection of acid-tolerant bacteria, increasing the risk of caries[8]. Compensated and transient bacteria will be responsible for several local infections (gingival, periodontal, endodontic, etc.), systemic, infections, and premature birth[12].

Thus, it is believed that dental plaque can be an important reservoir of infection-causing microorganisms[4,13,14,15]. This study aims to understand whether the collection site (toothless or toothless) influences the frequency of bacteria in the oral cavity.

METHODS

An observational, prospective, and cross-sectional study, approved by the Ethics and Research Committee of that hospital, was performed under protocol[3]. From May to July 2019, patients over 18 years of age were selected, submitted to elective heart surgery, and transferred to the Intensive Care Unit (ICU) in the postoperative period. Patients under 18 years of age or already under mechanical ventilation before surgery, or with VAP or tracheobronchitis, or patients who had their initial collection but had their surgeries cancelled, were excluded.

The sample calculation was based on the study of SOUZA e cols.[16], which observed an increase in the frequency of Streptococcus spp. in patients with aspiration pneumonia compared to patients who did not develop this condition; it is estimated that it is necessary to evaluate 35 patients to obtain a sample that represents, with 80% power and 95% confidence, the alternative hypothesis of this work (Kesley's method)[23].

After signing the Informed Consent Term, clinical oral samples were always collected by a single dentist. The methodology of material collection and sample characterization was based on the methodology used in the article by Hong and collaborators[17]. Biofilm samples were collected from the supra- and subgingival vestibular faces of 37 patients using calibrated and disposable plastic inoculation loops. The tooth to be collected was based on the absence criterion: 1) Right second lower molar; 2) Left second lower molar; 3) Right upper canine; 4) Left upper canine; 5) Right lower central incisor; 6) Left lower central incisor; 7) Right side jugal mucosa in toothless patients[10]. Bacterial growth inspection of the plates was performed after 48 hours of incubation in a 35°C incubator. The plaques were inspected to characterize the isolated colonies in terms of morphology, size, texture, edges, and pigments. Colonies with different morphological characteristics were identified by the MALDI-TOF methodology using VITEK MS® (bioMeriéux) equipment. Quality control was carried out according to the recommendations of the equipment manual. Isolated colonies were tested for antimicrobial sensitivity and the minimum inhibitory concentration was determined for each combination of microorganism/antimicrobial using Biomerieux VITEK equipment.

RESULTS

In our study, we collected clinical samples from 37 patients. The absolute frequencies of strains for each patient are expressed in parentheses: *Streptococcus gordoni* (14), *Streptococcus oralis* (15), Enterococcus faecalis (07), *Klebsiella pneumoniae* (08), S*erratia marcescens* (06), Staphylococcus *aureus* (03), Staphylococcus *epidermidis* (21), Staphylococcus *spp*. (03), *Pseudomonas aeruginosa* (07), *Streptococcus mutans* (18), Staphylococcus *capitis* (21), Staphylococcus *cohnii* (18), Streptococcus *anginosus* (18), *Streptococcus spp*. (06), *Leuconostoc* (06), *Coagulase-negative staphylococci* (SCN) (02), *Streptococcus mitis* (37) and *Enterobacter cloacae* (01). Of the 37 patients, 8 (21.62%) developed resistance to multiple drugs (table 1). The multidrug-resistant strains found were: *Serratia marcecens* (1), *Klebsiella pneumoniae* (2), *Enterobacter cloacae* (1), S*taphylococcus aureus* (1), *Staphylococcus cohnii* (2), *Staphylococcus capitis* (2) and *Staphylococcus epidermidis* (1). Of these eight patients, three presented more than one strain resistant to multiple drugs. The resistance mechanisms presented were *Klebsiella pneumoniae Carbapenemase* (KPC), Beta-Lactamase Extended Spectrum (ESBL), and MRSA production. The sample was characterized concerning gender, age, site of collection, the topography of collection, and jaw collection, performed by the chi-square test (table 2). In this table, you can see that these sociodemographic variables do not statistically influence the collection site. Table 3 refers to the relationship between comorbidities and risk factors and the collection screenings (toothless or toothled) performed by the chi-square test. According to this table, there are no significant differences between these conditions and the collection site. Table 1 lists the bacteria found and also the multiresistant bacteria at each collection site. According to this table, there is also no statistical difference between the frequencies of these strains (whether multi-R or not) and the collection site, which leads us to infer that regardless of whether the collection site is toothless or toothless, the microbial profile is the same.

Table 1. list of bacteria found and also the multiresistant bacteria at each collection site, performed by the chi-square test.

Microrganismos encontrados	Collection site			p-Value
	Total N (%)	Toohtless N (%)	Toothed N (%)	
Streptococcus mitis	37 (100.0)	13 (100.0)	24 (100.0)	1,000
Streptococcus oralis	15 (40.5)	5 (38.5)	10 (41.7)	0,850
Enterococcus faecalis	7 (18.9)	3 (23.1)	4 (16.7)	0,635
Klebsiella pneumoniae	8 (21.6)	2 (15.4)	6 (25.0)	0,498
Serratia marcecens	6 (16.2)	1 (7.7)	5 (20.8)	0,301
Staphylococcus aureus	3 (8.1)	1 (7.7)	2 (8.3)	0,946
Staphylococcus epidermidis	21 (56.8)	8 (61.5)	13 (54.2)	0,666
S. equorum	3 (8.1)	1 (7.7)	2 (8.3)	0,946
Pseudomonas aeruginosa	7 (18.9)	3 (23.1)	4 (16.7)	0,635
Streptococcus mutans	18 (48.6)	8 (61.5)	10 (41.7)	0,248
Staphylococcus capitis	21 (56.8)	8 (61.5)	13 (54.2)	0,666
Leuconostoc	7 (18.9)	2 (15.4)	5 (20.8)	0,686

Table 2. sample characterization with regarding to gender, age, site of collection, topography of collection and jaw collection, performed by the chi-square test.

Variables	Collection site			p-Value
	Total N (%)	Toothless N (%)	Toothed N (%)	
Sex				
Female	21 (56.8)	10 (76.9)	11 (45.8)	0,068
Male	16 (43.2)	3 (23.1)	13 (54.2)	
Age				
Up to 60 years	18 (48.6)	6 (46.2)	12 (50.0)	0,823
<60 years	19 (51.4)	7 (53.8)	12 (50.0)	
Origin				
Fortaleza/Zona Met.	30 (81.1)	12 (92.3)	18 (75.0)	0,199
Countryside	7 (18.9)	1 (7.7)	6 (25.0)	
Marital status				
Married	17 (45.9)	3 (23.1)	14 (58.3)	0,105
Unmarried	15 (40.5)	7 (53.8)	8 (33.3)	
Widower	5 (13.5)	3 (23.1)	2 (8.3)	
Profession				
Active	16 (76.2)	4 (66.7)	12 (80.0)	0,517
Retired	5 (23.8)	2 (33.3)	3 (20.0)	
Topography collection				
Jaw	4 (10.8)	0 (0.0)	4 (16.7)	0,000
Mandibule	20 (54.1)	0 (0.0)	20 (83.3)	
Mucosa Jugal	13 (35.1)	13 (100.0)	0 (0.0)	

Met.: metropolitan

Table 3. Relationship between comorbidities and risk factors and the collection screenings (toothless or toothless) performed by the chi-square test.

Comorbidities and Risk Factors	Collection site			p-Value
	Total N (%)	Toohtless N (%)	Total N (%)	
Comorbidities				
Valvulopathy	24 (64.9)	9 (69.2%)	15 (62.5)	0,682
Coronariopathy	16 (43.2)	5 (38.5%)	11 (45.8)	0,666
Congenital Heart Disease	1 (2.7)	0 (0.0%)	1 (4.2)	0,456
Heart Failure	4 (10.8)	1 (7.7%)	3 (12.5)	0,653
Other cardiopathies	2 (5.4)	0 (0.0%)	2 (8.3)	0,285
Respiratory infection	5 (13.5)	2 (15.4%)	3 (12.5)	0,806
Risk Factors				
Ex Smoker	10 (27.0)	4 (30.8)	6 (25.0)	0,706
Smiker	7 (18.9)	3 (23.1)	4 (16.7)	0,635
Ex Alcoholic	4 (10.8)	2 (15.4)	2 (8.3)	0,510
Alcoholic	7 (19.4)	3 (25.0)	4 (16.7)	0,551
HAS	24 (64.9)	9 (69.2)	15 (62.5)	0,682
Diabetes	10 (27.0)	5 (38.5)	5 (20.8)	0,249
Other	31 (83.8)	11 (84.6)	20 (83.3)	0,920

DISCUSSION

Healthcare-associated infection (HAIs) are mainly related to microbial transmission by professional hands, colonizing mainly the nasal cavities. However, colonization of other anatomical sites also contributes to the spread of pathogens. In this context, the mouth is a relevant site for study, since it gathers anatomical and physiological characteristics that make it a favorable site for microbial proliferation. Microorganisms can spread through the mouth by aspiration through oropharyngeal secretions or transmission by droplets of saliva when speaking, coughing, sneezing, or breathing[18].

Colonizers responsible for one of the most frequent hospital-acquired infections, ventilator-associated pneumonia (VAP), include *Staphylococcus aureus*, *Klebsiella pneumoniae*, *Acinetobacter baumanii*, *Haemophilus influenza*, and *Pseudomonas aeruginosa*[7]. VAP is one of the most common acquired infections in the intensive care unit (ICU) and has been associated with increased mortality[13,15,16,19]. It has an incidence ranging from 4 to 50 cases per 1,000 admissions[20] and occurs in 9% to 27% of intubated patients[19] for more than 48 hours[21]. The pathophysiology of VAP occurs due to the migration of pathogenic bacteria from the oral cavity to the lower respiratory tract[19,20]. These microorganisms accumulate in the subglottic secretions above the inflated balloon of the endotracheal tube and enter the lower airways below it, or due to the displacement of this balloon, or through microchannels that develop within the material of the same[13].

Thus, within 48 hours of hospital admission, the composition of oral microbiota in critical adults may change, characterizing a dysbiosis composed mainly of microorganisms of the hospital environment, gram-negative and/or gram-positive, which colonize the oral cavity and other sites of the upper respiratory tract[8]. According to Marino and collaborators[13], recent evidence has indicated that dysbiosis may occur in the dental plaque of patients with mechanical ventilation. According to Scannapieco and collaborators[14], the microorganisms in dental plaque of patients under mechanical ventilation are genetically identical to the strains of bronchoscopic cultures collected at the time of suspected pneumonia. Thus, it is believed that dental plaque can be an important reservoir of microorganisms causing infections[4,15,16]. Although the mechanisms of colonization by these microorganisms in immunosuppressed patients have not yet been fully clarified, studies suggest that the etiology of oral and oropharyngeal colonization is multifactorial[19, 22].

In our study, we collected clinical specimens from 37 patients and the bacteria found were: *Streptococcus gordoni*, *Streptococcus oralis*, Enterococcus faecalis, *Klebsiella pneumoniae*, *Serratia marcescens*, *Staphylococcus aureus*, *Staphylococcus epidermidis*, *Staphylococcus spp*., *Pseudomonas aeruginosa*, *Streptococcus mutans*, *Staphylococcus capitis*, *Staphylococcus cohnii*, *Streptococcus anginosus*, *Streptococcus spp*., *Leuconostoc*, Coagulase-negative Staph (SCN) and *Streptococcus mitis*. Among them, some are the cause of conditions such as VAP. The collection sites chosen were tooth-type (supra and subgingival region) and toothless type (in the cases of toothless patients), and the conclusion we reached

was that regardless of whether the collection site is toothless or toothless, the microbial profile is the same. Regarding the socio-demographic variables, comorbidities, risk factors, and the relationship with the collection site, none of these variables significantly influenced the choice of the collection site.

In our study, several possible strains causing VAP were collected. However, regardless of whether the collection site is toothless or toothless, the microbial profile is the same, a fact relevant for other studies that will work with collections in the oral cavity, since the collection site chosen will not influence the frequency of strains found.

The study had limitations: 1 some patients were admitted the day before surgery, and others were waiting for stabilization of their conditions; 2 patients who had valvulopathy received care prior to surgery in the dental sector of the hospital; the other patients did not; 3 due to financial limitations, we did not collect material from all patients on the day they were admitted to the hospital for comparison purposes and then 24 hours before surgery; 4 due to financial limitations, the study was conducted over a period of three months.

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