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THE RELATIONSHIP BETWEEN ORAL MICROFLORA AND COLONIZATION RESISTANCE IN INFLAMMATORY PERIODONTAL DISEASES IN PATIENTS WITH GASTROINTESTINAL DISORDERS

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Abstract
The study examines the interconnection between oral microflora composition and colonization resistance in patients with inflammatory periodontal diseases associated with gastrointestinal disorders. Quantitative and qualitative microbiological analyses were performed on samples collected from various oral biotopes, including the gums, tongue, cheeks, and palate. The findings reveal a significant decrease in resident microorganisms, such as Lactobacillus and Streptococcus, and an increase in opportunistic pathogens, including Candida spp., Klebsiella, and E. coli. The alterations in the oral microbial landscape are linked to systemic changes driven by gastrointestinal pathologies, contributing to the severity of periodontal inflammation. These data provide evidence for the role of systemic factors in modulating oral microbial homeostasis and highlight the need for integrated diagnostic and therapeutic strategies in clinical practice.

Keywords: Oral microbiome, colonization resistance, inflammatory periodontal diseases, gastrointestinal disorders, Candida spp., Klebsiella, Streptococcus, microbial dysbiosis, oral biotopes, systemic-pathological interaction.

Introduction

Oral microflora is a highly specialized microbial community, integral to maintaining the stability of the oral environment and ensuring colonization resistance against pathogenic microorganisms. Inflammatory periodontal diseases disrupt this balance, and systemic conditions, such as gastrointestinal disorders, further exacerbate these changes by altering the microbial composition. Recent studies have identified a significant reduction in resident microorganisms, such as Lactobacillus and Streptococcus, in patients with gastrointestinal pathologies, accompanied by an increased presence of opportunistic species, including Candida spp., Klebsiella, and E. coli. These microbial shifts not only reflect systemic influences on the oral cavity but also contribute to the progression of inflammatory processes in periodontal tissues, leading to impaired local immunity and increased susceptibility to secondary infections.

This research focuses on a detailed microbiological analysis of oral biotopes in patients with inflammatory periodontal diseases associated with gastrointestinal disorders. The primary objective is to evaluate the relationship between systemic gastrointestinal pathologies and changes in oral microbial composition, emphasizing the role of microbial dysbiosis in periodontal

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inflammation. The findings aim to advance understanding of systemic-local interactions in microbial ecology and provide a foundation for integrated therapeutic strategies.

Materials and Methods

The study was conducted at the Department of Dentistry of the Bukhara State Medical Institute between 2020 and 2023. A total of 138 patients participated in the research, stratified by age and gender into categories ranging from 18 to 60 years. The study cohort consisted of 108 patients diagnosed with inflammatory periodontal diseases in combination with gastrointestinal disorders, as well as a control group of 30 healthy individuals. Patients with systemic immune deficiencies, a history of antibiotic or probiotic use within three months prior to the study, or smoking habits were excluded to ensure the reliability of the results.

Microbiological sampling was carried out in compliance with strict aseptic conditions. Samples were collected from four oral biotopes: the gingival epithelium, the dorsal surface of the tongue, the buccal mucosa, and the hard palate. Sterile cotton swabs pre-moistened with isotonic saline were used for the collection, and the samples were immediately transferred into transport media for subsequent analysis in the microbiology laboratory.

The analysis involved quantitative and qualitative assessments of the microbial flora. Standard culture techniques were employed on selective and differential media to determine colony-forming units (CFU/cm²) across different biotopes. Biochemical assays and MALDI-TOF mass spectrometry were utilized for species identification, including both aerobic and anaerobic microorganisms. Fungal identification focused on the Candida genus using chromogenic media to differentiate species.

To examine microbial density and composition, the collected data were statistically analyzed. Descriptive statistics provided mean and standard deviation values for microbial densities, while inferential methods, such as the Mann-Whitney U test, were used to compare groups. Correlation coefficients were calculated to evaluate the relationship between microbial characteristics and clinical parameters of periodontal and gastrointestinal conditions. A p-value of less than 0.05 was considered statistically significant.

The study was conducted following ethical guidelines as outlined in the Declaration of Helsinki. Informed consent was obtained from all participants, and ethical approval was granted by the institutional review board of the Bukhara State Medical Institute.

Literature Review

Oral microflora plays a fundamental role in maintaining colonization resistance, which prevents the overgrowth of pathogenic microorganisms and supports periodontal health. Inflammatory periodontal diseases, particularly in the presence of gastrointestinal disorders, disrupt this delicate balance, leading to alterations in the density and diversity of microbial communities [1, pp. 1–8]. Research has demonstrated that patients with gastrointestinal pathologies exhibit a marked increase in opportunistic pathogens, such as Candida spp., Klebsiella, and E. coli, accompanied by a reduction in beneficial species, including Lactobacillus and Streptococcus [2, pp. 481–490; 3, pp. 5721–5732].

The link between gastrointestinal disorders and periodontal inflammation is mediated by microbial translocation and systemic immune dysfunction. Dysbiosis of the gut microbiota contributes to the upregulation of inflammatory cytokines and an overall reduction in immune homeostasis, which

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exacerbates the inflammatory processes in periodontal tissues [4, pp. 3–11; 5, pp. S5–S11]. Studies have identified an increase in periodontal pathogens, such as Porphyromonas gingivalis and Fusobacterium nucleatum, in patients with systemic conditions, highlighting their role in disrupting host immunity and promoting tissue destruction [6, pp. 108–122; 7, pp. 745–759].

Evidence further suggests that the weakening of colonization resistance in the oral cavity results from systemic inflammatory conditions. This weakening is characterized by reduced populations of beneficial microorganisms and a concurrent increase in opportunistic fungal species, notably Candida albicans, which thrive in the altered oral environment [8, pp. 485–491; 9, pp. 45–60]. Such microbial shifts are closely associated with the severity of periodontal inflammation, emphasizing the bidirectional relationship between systemic and oral health [10, pp. S22–S36; 11, pp. 72–80].

Alterations in salivary composition, pH levels, and immune mediators due to gastrointestinal disorders have also been identified as factors influencing oral microbial homeostasis. These changes facilitate the overgrowth of periodontal pathogens and exacerbate tissue damage, further underlining the systemic-local interplay in microbial ecology [12, pp. 463–470; 13, pp. 409–419]. Understanding these interactions is crucial for developing integrated diagnostic and therapeutic strategies aimed at restoring microbial balance and improving clinical outcomes in patients with inflammatory periodontal diseases and gastrointestinal disorders [14, pp. 7–23].

Results and Discussion

The study involved a comprehensive microbiological analysis of oral biotopes in 138 participants, including 108 patients diagnosed with inflammatory periodontal diseases and gastrointestinal disorders, and 30 healthy individuals in the control group. The assessment was focused on microbial density and diversity across four oral sites: the gingiva, tongue, buccal mucosa, and palate. Quantitative and qualitative shifts in the oral microbiome were identified, reflecting significant alterations in colonization resistance and microbial homeostasis in the patient group compared to the control group.

A notable reduction in the density of resident microorganisms, such as Lactobacillus and Streptococcus salivarius, was observed in patients with inflammatory periodontal diseases. In the gingiva, the density of Lactobacillus decreased to 1.10±0.12 CFU/cm², compared to 1.80±0.10 CFU/cm² in the control group, representing a statistically significant decline ($p<0.05$). Similarly, Streptococcus salivarius density in the same biotope decreased from 4.21±0.15 CFU/cm² in healthy individuals to 2.35±0.20 CFU/cm² in patients.

Conversely, an increase in opportunistic pathogens was detected, particularly in Candida albicans and Klebsiella pneumoniae. The density of Candida albicans in the gingiva rose from 2.07±0.15 CFU/cm² in the control group to 4.45±0.25 CFU/cm² in patients, while Klebsiella pneumoniae density increased from 0.08±0.02 CFU/cm² to 2.45±0.12 CFU/cm². These results underline the disruption of microbial equilibrium in patients with concurrent systemic and oral conditions.

The composition and density of microbial populations varied across the four studied biotopes. The tongue consistently exhibited the highest density of Streptococcus mutans in the patient group (2.59±0.15 CFU/cm²), whereas the palate demonstrated the lowest microbial density overall. Table 1 summarizes the microbial density in each biotope for both groups.

Table 1. Microbial Density Across Oral Biotopes (CFU/cm², M±m)

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Microorganism	Gingiva (Control)	Gingiva (Patients)	Tongue (Control)	Tongue (Patients)	Buccal Mucosa (Control)	Buccal Mucosa (Patients)	Palate (Control)	Palate (Patients)
Lactobacillus	1.80±0.10	1.10±0.12	1.60±0.08	0.95±0.10	1.08±0.02	0.88±0.05	1.00±0.05	0.80±0.10
Streptococcus salivarius	4.21±0.15	2.35±0.20	2.83±0.10	2.00±0.12	1.53±0.05	1.02±0.08	1.22±0.06	0.95±0.05
Candida albicans	2.07±0.15	4.45±0.25	3.11±0.12	4.00±0.20	0.80±0.05	3.42±0.18	0.60±0.02	2.35±0.15
Klebsiella pneumoniae	0.08±0.02	2.45±0.12	0.12±0.01	2.35±0.15	0.10±0.02	1.80±0.10	0.05±0.01	1.50±0.12

The observed reduction in Lactobacillus and Streptococcus densities reflects a decline in colonization resistance, a critical barrier against opportunistic pathogens. These microorganisms are known to maintain oral homeostasis through their metabolic activity and ability to inhibit pathogenic colonization. The sharp increase in Candida albicans and Klebsiella pneumoniae densities highlights the role of opportunistic pathogens in disrupting local immunity, contributing to exacerbated inflammation in periodontal tissues.

The significant microbial shifts in patients with gastrointestinal disorders suggest systemic influences on the oral microbiota. Gastrointestinal dysbiosis, characterized by an altered gut microbial composition, may influence salivary properties and immune responses, creating favorable conditions for pathogenic overgrowth in the oral cavity. These findings align with previous studies that demonstrate the bidirectional impact of systemic inflammation and local microbial changes on periodontal health.

The results underscore the importance of addressing both systemic and local factors in managing periodontal diseases, particularly in patients with gastrointestinal disorders. Therapeutic approaches should focus on restoring microbial balance through targeted interventions, including the use of probiotics to enhance colonization resistance and antimicrobial strategies to reduce opportunistic pathogens. Further research is necessary to elucidate the mechanistic pathways linking systemic and oral microbial ecosystems and to optimize integrated treatment protocols.

Conclusion

This study highlights the significant alterations in the oral microbiome and colonization resistance in patients with inflammatory periodontal diseases and gastrointestinal disorders. The findings demonstrate a marked reduction in resident microorganisms, such as Lactobacillus and Streptococcus, and a concurrent increase in opportunistic pathogens, including Candida albicans and Klebsiella pneumoniae. These microbial changes reflect systemic influences originating from gastrointestinal dysbiosis, which exacerbate local inflammatory processes in periodontal tissues.

The results underline the importance of an integrated therapeutic approach addressing both systemic and local factors to restore microbial balance and improve clinical outcomes in such patients. Future research should focus on exploring the mechanistic interactions between systemic conditions and oral health, as well as developing targeted strategies to mitigate microbial dysbiosis and enhance colonization resistance.

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