

The Association Between Periodontitis and Oro digestive Cancer : A Comprehensive Review

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Abstract

Oro digestive (Gastrointestinal) cancer encompasses a group of malignancies affecting the oral cavity, esophagus, stomach, and other parts of the gastrointestinal tract. In recent years, there has been increasing interest in exploring the potential link between Oro digestive cancer and periodontitis, a chronic inflammatory disease of the oral cavity. This review aims to provide a comprehensive analysis of the current evidence regarding the association between periodontitis and Oro digestive cancer, highlighting potential mechanisms and clinical implications.

Keywords: Oro digestive, gastrointestinal tract, inflammatory.

Introduction

Oro digestive cancer is a significant global health concern, contributing to substantial morbidity and mortality. Periodontitis, characterized by chronic inflammation of the periodontal tissues, has been suggested as a possible risk factor for orodigestive cancer development and progression. The inflammatory reaction in the surrounding tissues is triggered by the periodontopathic organisms, which causes a steady flow

of bacterial and inflammatory markers into the saliva and blood. These then spread to distant locations and have a negative impact on overall health.¹ This section introduces the scope of the review, emphasizing the need to understand the association between these two conditions.

Potential Mechanisms

In this section, the review explores potential biological mechanisms linking periodontitis and orodigestive cancer. Michaud et al. Found a substantial link between orodigestive tract cancer and oral health status in a prospective cohort analysis.² Chronic inflammation, immunological alterations, and the role of specific bacterial pathogens are among the mechanisms considered.³ Additionally, the influence of shared risk factors such as tobacco use, alcohol consumption, and poor oral hygiene is discussed.

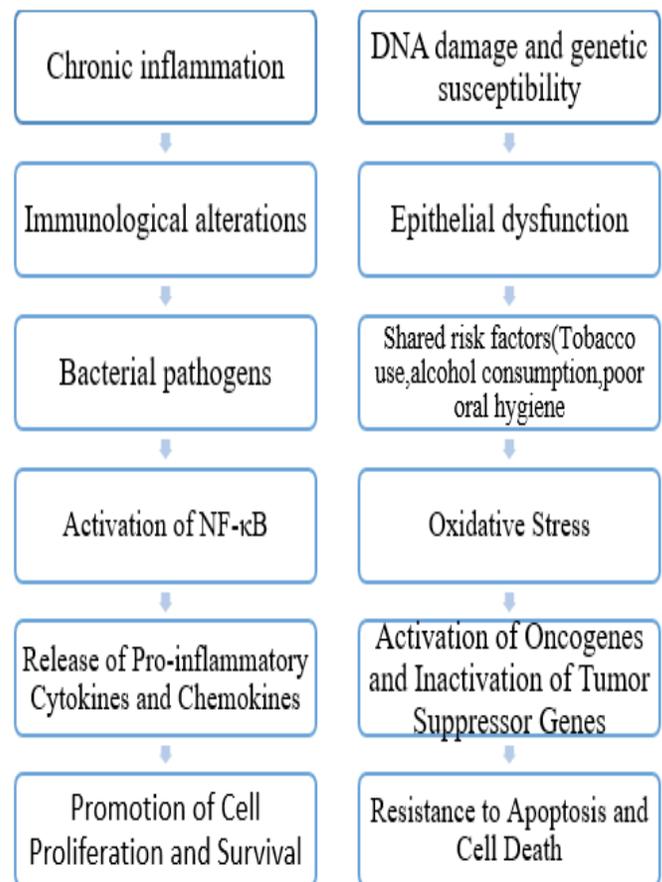
- **Chronic Inflammation:** Periodontitis leads to chronic inflammation in the oral cavity, contributing to the development of orodigestive cancer.
- **Immunological Alterations:** Chronic inflammation in periodontitis results in immunological alterations, including dysregulation of immune cells and

cytokine imbalance, creating a pro-inflammatory environment.

- **Bacterial Pathogens:** Specific bacteria associated with periodontitis contribute to chronic inflammation, promoting orodigestive cancer through their virulence factors and interactions with the immune system. According to Groeger et al.⁴ P. Gingivalis may interfere with anticancer immune response and impede ATP-dependent apoptosis of cancer cells by secreting nucleoside diphosphate kinase.
- **Activation of NF- κ B:** Inflammatory signals from periodontitis activate the NF- κ B pathway, which regulates the expression of genes involved in inflammation, cell survival, and tumor development.⁵
- **Oxidative Stress:** Chronic inflammation and bacterial pathogens generate oxidative stress, causing DNA damage and promoting genetic instability.
- **Release of Pro-inflammatory Cytokines and Chemokines:** Chronic inflammation in periodontitis leads to the release of pro-inflammatory cytokines and chemokines that can further propagate the inflammatory response and contribute to tumor development.
- **Activation of Oncogenes and Inactivation of Tumor Suppressor Genes:** Genetic alterations caused by chronic inflammation, DNA damage, and oxidative stress can activate oncogenes and inactivate tumor suppressor genes, promoting cellular transformation and tumor growth.
- **Promotion of Cell Proliferation and Survival:** The activated signalling pathways and altered gene expression patterns promote cell proliferation,

survival, and resistance to apoptosis, contributing to the expansion of cancerous cells.

- **Resistance to Apoptosis and Cell Death:** Altered cellular signalling and genetic alterations can disrupt the normal apoptotic pathways, leading to reduced cell death and increased cell survival, further fuelling tumor growth.
- **Orodigestive Cancer:** The cumulative effects of chronic inflammation, immunological alterations, bacterial pathogens, activation of NF- κ B, oxidative stress, release of pro-inflammatory mediators, activation of oncogenes, inactivation of tumor suppressor genes, promotion of cell proliferation, resistance to apoptosis, and cell survival contribute to the development and progression of orodigestive.⁵





Risk factors associated with both orodigestive cancer and periodontitis:

Poor Oral Hygiene: Inadequate oral hygiene practices, such as infrequent brushing and flossing, can lead to the buildup of plaque and tartar on the teeth and gum line resulting in gum inflammation, gingivitis, and eventually periodontitis. Poor oral hygiene is also a risk factor for oral infections and may contribute to the development of orodigestive cancer. In their case-control study, Abnet et al.⁶ found that a modest intervention of teaching basic oral hygiene practises reduced the incidence of esophageal squamous cell carcinoma in people living in high-risk districts of Iran who were matched for age, gender, and neighbourhood. When compared to controls, people with esophageal cancer had significantly higher decayed, missing, or filled teeth index (DMFT). Thus, there is a link between two markers of poor oral hygiene (a higher number of DMFT and a lack of bad oral hygiene) and the risk of esophageal cancer.

Genetic Factors: Certain genetic variations and susceptibility genes may influence an individual's risk of developing both orodigestive cancer and periodontitis.

Family history of these conditions can also indicate a potential genetic predisposition.

Immunocompromised Conditions: Individuals with weakened immune systems, such as those with HIV/AIDS, organ transplant recipients, or undergoing chemotherapy, have a higher susceptibility to both orodigestive cancer and periodontitis due to compromised immune function.

Chronic Inflammation: Chronic inflammatory conditions, such as chronic gastritis, gastroesophageal reflux disease (GERD), and inflammatory bowel disease (IBD), increase the risk of orodigestive cancer. Similarly, periodontitis is characterized by chronic inflammation in the gums, which can contribute to the development of orodigestive cancer.

Risk factors increase the likelihood of developing these conditions, but individual cases can vary, and other factors may also play a role. Additionally, addressing and managing these risk factors can help reduce the risk and improve overall health outcomes.

Clinical Implications

Screening and early detection

This section focuses on the clinical implications of the association between periodontitis and orodigestive cancer. It highlights the importance of oral health in the prevention and management of orodigestive cancer, emphasizing the need for interdisciplinary collaboration between dental and medical professionals. The potential for periodontal screening as an adjunctive tool for early detection and risk assessment is also explored.

Future Directions and Research Challenges:

The review concludes by addressing future directions for research in this field. It identifies key research gaps and challenges, such as the need for prospective studies, standardized diagnostic criteria, and the elucidation of underlying biological mechanisms. Recommendations

for promoting interdisciplinary collaboration and public health interventions are also provided.

Conclusion

This review consolidates the existing evidence on the association between periodontitis and orodigestive cancer. While numerous epidemiological studies suggest a potential link, further research is warranted to establish causality and elucidate underlying mechanisms. Nonetheless, the findings underscore the importance of oral health in the context of orodigestive cancer prevention and management.

By critically evaluating the current literature and highlighting research gaps, this comprehensive review aims to stimulate further investigation into the complex relationship between periodontitis and orodigestive cancer, potentially informing future preventive and therapeutic strategies.

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