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Oral Hygiene and the Gastrointestinal System; A Literature Review

Mohamed Barakat[1]*, Ahmed Shady[2] and Ghulamullah Shahzad[3]

[1]*Department of Internal Medicine, Icahn School of Medicine at Mount Sinai-Queens Hospital Center, USA*

[2]*Department of Internal Medicine, New York Medical College-Metropolitan Hospital Center, USA*

[3]*Department of Internal Medicine, Division of Gastroenterology, Icahn School of Medicine at Mount Sinai-Queens Hospital Center, USA*

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***Corresponding author:** Mohamed Barakat, Department of Internal Medicine, Icahn School of Medicine at Mount Sinai-Queens Hospital Center, 82-68 164th street, Jamaica, NY 11432, USA, Tel: 718-883-4050; Fax: 718-883-6197; Email: Mo_barakat@hotmail.com

Abstract

Periodontal disease is a chronic inflammatory disease affecting the periodontium and is considered to be one of the most common conditions affecting oral hygiene. Poor oral hygiene has recently been associated with numerous systemic illnesses such as diabetes mellitus, rheumatoid arthritis, chronic kidney disease and coronary artery disease. There is limited data exploring the effects of poor oral hygiene on the gastrointestinal system. Literature search was performed using the Medline database, 2 authors performed the search independently. Search terms used were "oral hygiene" or "periodontal" or "periodontitis" or "gingivitis" and "gastrointestinal", "esophagus", "stomach", "liver", "intestine", "intestines", "inflammatory bowel disease", "bowel", "colon", "gall bladder", "gallstone", "pancreas". In this literature review, we explore the association between poor oral hygiene and the various organs of the gastrointestinal system including the esophagus, stomach, liver, bowel, pancreas and gallbladder.

Keywords: Oral hygiene; Periodontal; Periodontitis; Gingivitis; Gastrointestinal; Esophagus; Stomach; Liver; Inflammatory bowel disease; Pancreas

Introduction

Periodontal disease is a chronic inflammatory disease affecting the periodontium and is considered to be one of the most common conditions affecting oral hygiene [1]. There are over 800 species of bacteria identified in the oral cavity [2], many of which may contribute to poor oral hygiene with modifiable risk factors such as smoking [3]. Poor oral hygiene has recently been associated with numerous systemic diseases such as diabetes mellitus [4], obesity [5], rheumatoid arthritis [6], chronic obstructive lung disease [7], chronic kidney disease [8], coronary artery disease [9] and infections [10]. There is also an increased risk of malignancy associated with poor oral hygiene [11]. There is limited data exploring the effects of poor oral hygiene on the gastrointestinal system, our aim is to review the effects of poor oral hygiene on the gastrointestinal system. This review will include the effects of poor oral hygiene on the esophagus, stomach, small and large bowel, liver and pancreas.

Methods

A systemic review of the literature was performed using the Medline database. The search was performed using the terms "oral hygiene" or "periodontal" or "periodontitis" or "gingivitis" and "gastrointestinal", "esophagus", "stomach", "liver", "intestine", "intestines", "inflammatory bowel disease", "bowel", "colon", "gall bladder", "gallstone", "pancreas". A total of 708 articles were identified. After reviewing the titles and abstracts, 674 studies were excluded. A total of 34 articles were included in this literature review.

Esophagus

The esophagus as an area of interest in individuals with poor oral hygiene, in particular, the increased risk of cellular metaplasia, dysplasia and neoplasia. A review of two multicenter case control studies conducted in different countries by Conway [12] found that risk factors for head and neck cancer that were independent of tobacco use and alcohol consumption, were poor condition of the mouth [central Europe OR, 2.89 (95% CI, 1.74-4.81); Latin America OR, 1.89 (95% CI, 1.47-2.42)]; lack of toothbrush use [Latin America OR, 2.36 (95% CI, 1.28-4.36)], and daily mouthwash use [Latin America OR, 3.40 (95% CI,1.96-5.89]. Missing six to 15 teeth was found to be an independent risk factor for esophageal cancer [central Europe OR, 2.84 (95% CI, 1.26-6.41); Latin America OR, 2.18 (95% CI, 1.04-4.59)]. Similar results were found in a review Guha et al. [13].

Another case control study, by Macfarlane et al. [14], examining pathogenic risk factors in Barret's esophagus (BE) identified 46 bacterial species, with 10 species in common between the 2 groups. Both aspirate and biopsy samples from patients with BE had high levels of Campylobacter species (Campylobacter concisus and Campylobacter rectus), which have been linked to enteritis, periodontal infections, and tumor formation in animals, they were found in 4 (57%) of 7 patients with BE but in none of the control subjects. Microscopic examination revealed that bacteria on mucosal biofilms often occurred in micro colonies. The occurrence of nitrate-reducing Campylobacter species in patients with BE suggests that there may be a link in the initiation, maintenance, or exacerbation of disease processes leading to adenocarcinoma formation.

A prospective study was conducted in China following patients with teeth loss, found out that it was associated with a significantly elevated risk of developing cancer [15]. Tooth loss was associated with a relative risk (RR) (95% confidence interval, CI) of 1.3 (1.1-1.6) in the esophagus, 1.3 (1.0-1.6) in the gastric cardia, and 1.8 (1.1-3.0) in the gastric non-cardia. Further analysis revealed that this increased risk was most strongly associated with the loss of the first few teeth and primarily occurred in the younger members of the cohort. The hypothesis was that this may be related to alterations in oral bacterial flora and subsequent increases in the in-vivo production of carcinogens such as nitrosamines.

Inflammatory bowel disease

The incidence of inflammatory bowel disease (IBD) has recently increased [16]. There is the hygiene hypothesis that elucidates that the lack of microbial exposure because of improved hygienic conditions causes an immunologic imbalance that predisposes to autoimmune diseases, such as Crohn's disease (CD) and ulcerative colitis (UC) [17].

In an effort to identify the prevalence of periodontal disease in IBD, Koutsochristou et al. [18] studied 55 children in remission from a single outpatient IBD clinic, aged 4 to 18 years and 55 matched systemically healthy controls of a dental practice prospectively. In this case-control study, the evaluation included a dental questionnaire in both groups, assessment of the the decayed, missing, and filled tooth (dmf-t or DMF-T), simplified gingival, plaque control record and community periodontal treatment needs indices. Children with IBD compared with controls had a statistically significant ($P < 0.001$) higher dmf-t (2.95 versus 0.91) or DMF-T (5.81 versus 2.04) index and a higher gingival inflammation (simplified gingival, 40% versus 24%). Also, the community periodontal treatment needs was significantly higher compared with controls ($P < 0.001$); most of the patients with IBD needed treatment of gingivitis (47% versus 4%), and none of them had healthy periodontium (0% versus 69%). The results of this case-control study demonstrate a higher frequency of dental caries, more clinical signs of gingival inflammation, and increased periodontal treatment needs in children and adolescents with IBD despite similar oral hygiene status.

In a similar study by Brito et al. [19], 99 CD, 80 UC and 74 healthy controls were compared for DMF-T index and presence of periodontitis. Probing pocket depth (PPD), clinical attachment loss (CAL), bleeding on probing (BOP), plaque and DMF-T index were measured on all subjects. Significantly more patients with UC (90.0%; $p<0.001$) and CD (81.8%; $p=0.03$) had periodontitis than controls (67.6%). Among smokers, UC patients had significantly more periodontitis. CD had a greater mean DMF-T score (18.7 versus 13.9; $p=0.031$) compared with controls and UC had greater median PPD (2.2 versus 1.7 mm; $p<0.0001$) than controls. Among non-smokers, CD (2.4 mm; $p<0.0001$) and UC showed deeper pockets (2.3 mm; $p<0.0001$) compared with controls (1.5 mm). UC had a greater mean DMFT score (15.3 versus 12.1; $p=0.037$) compared with controls. CD and UC patients had higher DMFT and prevalence of periodontitis than controls, but smoking was an effect modifier.

Upon review, we found a questionnaire based case control study on IBD and non IBD patients [17]. Thirty one percent of IBD patients had ulcerative colitis (UC) and 69% had Crohn's disease (CD). For subjects with IBD, the frequency of brushing at disease onset was significantly higher than in controls ($P=0.005$). Also, the frequency of use of dental floss and breath freshener at disease onset was significantly higher in IBD patients ($P=0.005$ and<0.001, respectively). Patients with IBD more frequently visited their dentist at disease onset ($P<0.001$) and continued to visit their dentist more often ($P<0.001$). IBD cases had a higher frequency of dental complications such as tooth caries ($P=0.007$), oral ulcers ($P=0.04$) and dry mouth ($P=0.001$). These findings suggest that oral hygiene practices may cause alterations in the flora of the oral mucosa, which causes imbalance in the gut microbiome (dysbiosis), and thereby contributes to the pathogenesis of IBD. Conversely, the increased frequency of dental problems in IBD patients might be due, at least in part, to alterations in oral flora or to their disease. Another cohort study in Sweden conducted by Yin et al. [20], with 209 individuals who developed IBD (142 developed UC and 67 developed CD) revealed an inverse relationship between poor oral health and IBD, especially in individuals with severe oral problems. Loss of 5-6 teeth of the 6 teeth examined was associated with a lower risk of IBD (hazard ratio, 0.56; 95% confidence interval, 0.32-0.98). Having dental plaques that covered more than 33% of tooth surface was negatively associated with CD (hazard ratio, 0.32; 95% confidence interval, 0.10-0.97). These findings were heavily criticized in an editorial by Hujeol et al. [21] who claimed that the association of poor oral hygiene and its protective effect in UC is most likely related to smoking which is a confounding factor and has been shown to decrease the progression of the disease in a large meta-analysis [22].

A case-control study from Jordan by Habashneh et al. [23] included 260 Jordanian adults (101 with UC, 59 with CD and

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100 with no IBD). The prevalence of periodontitis was much higher among patients with CD and those with UC compared with subjects having no IBD in the age groups < 36 and 36-45 years old only. After adjusting for age and number of missing teeth, patients with CD (odds ratio 4.9, 95% confidence interval 1.8-13.2) and patients with UC (odds ratio 7.00, 95% confidence interval 2.8-17.5) had significantly higher odds of periodontitis than individuals without IBD. Further analysis revealed the severity of periodontitis to be significantly higher among patients with CD and patients with UC when compared with subjects having no IBD.

Historically, Campylobacter concisus was associated with the oral cavity and has been linked to gingivitis and periodontitis. Evidence to support the role of C. concisus in acute intestinal disease has come from studies that have detected or isolated C. concisus as sole pathogen in fecal samples from patients with diarrhea. The Campylobacter species' involvement in the onset of IBD was first investigated in the 1980's, however C. Jejuni failed to show an association with IBD. Aubenhus et al. [24] isolated species of Campylobacter concisus from fecal samples of patients with IBD in 2002. But it was not until 2009, when Zang et al. [25] compared the prevalence of Campylobacter concisus in fecal samples of pediatric patients with new onset CD and individuals with no CD. In this study, a significantly higher prevalence of C. concisus DNA ($P < 0.0001$) as well as C. concisus specific IgG antibody levels ($P < 0.001$) were detected in children with newly diagnosed CD (51%; 0.991 ± 0.447) as compared with controls. Additionally, C. concisus was successfully isolated from an intestinal biopsy of a child with CD. Man et al. [26] in their study also noted an increase in the prevalence of C. concisus DNA in children with newly diagnosed CD as compared with controls.

In another study by Brito et al. [27], multiple-comparison analysis showed that the groups (ulcerative colitis Vs Crohn's disesase) differed in bacterial counts for Bacteroides ureolyticus, Campylobacter gracilis, Parvimonas micra, Prevotella melaninogenica, Peptostreptococcus anaerobius, Staphylococcus aureus, Streptococcus anginosus, Streptococcus intermedius, Streptococcus mitis, Streptococcus mutans, and Treponema denticola ($P<0.001$). CD was associated with significantly higher levels of these bacteria than UC patients at either gingivitis or in periodontitis sites ($P<0.05$). CD patients had higher levels of P. melaninogenica, S. aureus, S. anginosus, and S. mutans compared with controls at gingivitis and at periodontitis sites ($P<0.05$). UC patients harbored higher levels of S. aureus ($P=0.01$) and P. anaerobius ($P=0.05$) than controls only in gingivitis sites. This study concluded that IBD patients harbor higher levels of bacteria that are related to opportunistic infections at inflamed oral sites that might be harmful for the crucial microbe-host interaction.

Gall bladder disease

Limited information is available regarding poor oral hygiene and gall bladder disease. A single study [28] recently evaluated the correlation between gall stone disease and poor oral hygiene in the US population using the Third National Health and Nutrition Examination Survey (NHANES III). It revealed that missing teeth and a reflection of bad oral hygiene, were found to be independent predictors of gall stones disease by multi-variate analysis.

Liver

The severity of periodontitis correlates with blood levels of interleukin-6, tumor necrosis factor-α, and endotoxins, such as lipopolysaccharides. These effects may be accentuated in the context of liver cirrhosis due to the reduced clearance of circulating endotoxins, bacteria and inflammatory mediators by the dysfunctional liver. In chronic liver disease, gut-derived endotoxemia and associated inflammatory responses can cause the progression of liver fibrogenesis and worsening liver function. A single center cross sectional study on liver transplant patients that included 110 patients by Kauffles at al. [29] found that improved dental care pre- and post-transplant, including screening for fungal infections, is recommended to avoid systemic infections.

Another interesting case control study by Masato et al. [30] found out that patients with NAFLD had higher infection rate with P.Gingivalis bacteria (46.7% Vs 21.7% Odds ratio 3.16), most of the detected bacteria in NAFLD patients was of the invasive genotype especially type II which was associated with accelerated progression of NAFLD on the mice models. Another study by Aberg et al. [31] found out that the need for multiple tooth extractions, a surrogate marker of dental infections, was associated with reduced time from diagnosis of liver disease to the need for liver transplantation ($P = 0.02$). This association was independent of age, sex, liver disease etiology and Model for End-Stage Liver Disease (MELD) score ($P = 0.04$). Among 38 patients with accurate laboratory follow-up data, the number of tooth extractions correlated with the change in MELD score during the year preceding dental examination ($r = 0.43$, $P = 0.03$). Spontaneous bacterial peritonitis caused by Streptococcus viridans occurred only among patients with multiple dental infections.

Pancreas

Numerous studies have demonstrated the effects of poor oral hygiene on the pancreas. Most notable is the effect of oral bacteria on pancreatic cancer. Based on a hypothesis that oral bacteria could be a risk factor in the pathogenesis of the pancreatic cancer pathway, the investigators of the European Prospective Investigation into Cancer cohort used an immunoblot array, to detect and quantify antibodies against 25 oral bacterial strains in sera from 405 pancreatic cancer cases and 416 controls [32]. They showed that individuals with high levels of antibodies against Porphyromonas gingivalis ATCC 53978, a pathogen that contributes to periodontal disease had a twofold higher risk of pancreatic cancer than individuals with lower levels of

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these antibodies. Interestingly, a different group with overall higher levels of antibodies had a lower risk (45%) than a group with overall lower levels of antibodies suggesting that possibly antibodies against certain commensal bacteria may reduce the risk of pancreatic cancer.

Mitsuhashi et al. [33] later showed an association between Fusobacterium species, another known pathogen contributing to periodontal disease and pancreatic cancer. In a cohort of 283 patient with pancreatic ductal adenocarcinoma (PDAC), Fusobacterium species was positive in 8.8% of cases and was observed to carry a higher risk of mortality. The presence of Fusobacterium species was an independent prognostic factor and could possibly be used as an indicator of prognosis. Both Fusobacterium species and Porphyromonas gingivalis have been proven to stimulate tumorigenesis via direct interaction with oral epithelial cells through Toll-like receptors [34].

An additional study by Fan et al. [35] examined the carriage of oral pathogens, Porphyromonas gingivalis and Aggregatibacter actinomycetemcomitans. They were associated with higher risk of pancreatic cancer (adjusted OR for presence vs absence=1.60 and 95% CI 1.15 to 2.22; OR=2.20 and 95% CI 1.16 to 4.18, respectively). Phylum Fusobacteria and its genus Leptotrichia were associated with decreased pancreatic cancer risk (OR per per cent increase of relative abundance=0.94 and 95% CI 0.89 to 0.99; OR=0.87 and 95% CI 0.79 to 0.95, respectively).

A recent meta-analysis showed a relative risk for periodontitis and pancreatic cancer to be 1.74 [95% confidence interval (CI) 1.41-2.15] and 1.54 for edentulism (95% CI 1.16-2.05) [36]. There was no evidence of heterogeneity for either variable, and no evidence of publication bias. The studies included reports from three continents, suggesting that the association is generalizable. Most of the included studies adjusted for variables thought to be associated with pancreatic cancer, such as gender, smoking, BMI, diabetes, and alcohol consumption.

Stomach

Helicobacter Pylori although most prevalent in the stomach, has also been described in the oral cavity of individuals with poor oral hygiene [37]. The association between poor oral hygiene and Helicobacter pylori (H. pylori) has been an area of study in the recent years. There have been several studies that reported on the prevalence, and relationship between the existence of H. pylori in oral cavity and in stomach of patients. Few studies have evaluated the relationship between gingival and periodontal disease and H. pylori infection. While some investigators have reported a positive association between the two conditions [38,39], others have reported that there was no association between H. pylori infection and periodontal diseases [40]. The International Agency for Research on Cancer of the World Health Organization (WHO) has designated H. pylorias a Group 1 carcinogen [41] which makes the correlation between oral H. Pylori and gastric H. Pylori a serious and important topic. One of the theories linking the prevalence of oral H. Pylori to recurrent gastric infections is the recolonization from dental plaque which was hypothesized to act as a reservoir for the pathogen [42].

Liu et al. [43] studied the prevalence of Helicobacter pylori in the oral cavity and the relationship between gastric H. pylori infection and the presence of the bacteria in the oral cavity of a Chinese population. A total of 443 dyspeptic patients participated in the study. Of the 443 study patients, oral H. pylori was found in the dental plaque of 263 (59.4%) and the stomach of 273 (61.6%). Additionally, in all age groups, the prevalence of gastric infection was significantly higher among the patients with positive tests for H. pylori in their dental plaque than in the patients with no H. pylori in their dental plaque ($P < 0.05$). This study demonstrated that oral cavity may be a potential reservoir for H. pylori, and the prevalence of oral H. pylori approximated that of gastric H. pylori in the studied population.

In a cross sectional study by Nasrolahei et al. [44], samples were taken over a period of one year from tooth surfaces of molars, premolars and incisors of 180 consecutive dyspeptic patients. During endoscopy, six biopsies were taken from the gastric antrum and corpus and histological examination for comparison. The study showed the presence of H. pylori in dental plaque of infected and uninfected patients. There was no significant association between H. pylori colonization in dental plaque and gastric infection. This study suggests that oral hygiene did not have a significant influence on the presence of H. pylori in dental plaque and that there was dental plaque as a reservoir of H.pylori, had no relationship to gastric infection.

Several studies have also assessed the relationship between treatment of gastric H.Pylori infections and it's effect on H.pylori prevalence in the oral cavity. In a study by Bago et al. [45] 56 patients with chronic periodontitis and gastric H. pylori, 23 (41.1%) harbored H. pylori in the oral cavity. Eradication rate in stomach was 78.3%, whereas in the oral cavity, H. pylori was not detected from any sample after the eradication therapy. Almost half of the patients with gastric H. pylori harbored the bacterium in the oral cavity. After the eradication therapy, H. pylori was not detected in the oral cavity, what suggests high effectiveness of the therapy protocol in the oral cavity. Contradicting findings were demonstrated in a study by. Miyabayashi et al. [46] In their study, 47 patients with gastric H. pylori received a therapeutic regimen consisted of 30 mg/day lansoprazole, 750 mg/day metronidazole, and 400 mg/day clarithromycin administered for 2 weeks. The eradication success rate was significantly lower in the oral H. pylori-positive cases (12/23, 52.1%) than in the negative cases (22/24, 91.6%) at 4 weeks after the therapy ($p = .0028$). Follow up after 2 years showed that only 16 of the 23 (69.5%) oral H. pylori-positive cases were disease-free, as compared to 23 of the 24 (95.8%) oral H. pylori-negative cases ($p = .018$). This study suggests that H. pylori in the oral cavity affected the outcome of eradication therapy and was associated with a recurrence of gastric infection. We recommend that oral H.

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pylori should be examined by nested PCR and, if positive, should be considered a causal factor in refractory or recurrent cases.

Conclusion

Periodontal disease and poor oral hygiene have a larger role than previously identified in systemic diseases including the gastrointestinal system. As previously stated, maintaining a healthy oral cavity can reduce the risk of morbidity and mortality from systemic and gastrointestinal disease and possibly reduce the risk of malignancy. Additional studies are needed to further consolidate these findings.

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