Tobacco Use and Oral Health

Tobacco use in smoked and smokeless forms has a potentially significant and negative impact on oral health and prosthodontic therapy. Local and systemic outcome of tobacco use on oral health depends upon method, frequency, and duration of use and is dose dependent. Smoking and smokeless tobacco have been associated with tooth loss, caries, periodontal disease, oral soft tissue changes, dental implant failure, peri-implant disease, and oropharyngeal cancer. For a tobacco user, decreasing or eliminating the tobacco habit can reduce the impact of tobacco use on oral health. Cessation may reduce but not completely eliminate the patient’s lifetime risk for disease and complications. The best means of eliminating the impact of tobacco on oral health is by never beginning its use.

Smoking, Edentulism, and Systemic Disease

Studies have reported correlations with smoking and tooth loss. Some have associated smoking with lower socioeconomic status and less education. Adults who smoke have greater risk of tooth loss. Children with ongoing exposure to secondhand smoke also have greater risk. Edentulism is further associated with an increased risk of a number of systemic diseases, including diabetes, hypertension, coronary artery disease, asthma, and cancer. Although no study with direct correlation exists, associations exist among smoking, tooth loss, edentulism, and greater risk of systemic disease. It has not been established if these relationships are causal or casual in nature.

Caries Risk

Smoking has been correlated with increased caries incidence. In addition, other factors related to those who smoke may contribute to caries. For example, smoking in a household has been associated with increased incidence of caries for children in that household. In vitro evidence suggests nicotine may increase biofilm formation. Caries risk may be greater with smokeless tobacco, possibly related to the prolonged exposure to sugars. Those who do not smoke or use smokeless tobacco may have reduced caries risk.

Smoking and Periodontitis

In general, smokers have greater periodontal morbidity than nonsmokers. The vasculature, humoral immune response, cellular immune response, and the inflammatory response may be affected. Numerous cross-sectional, case-control, and cohort studies demonstrate a statistically significant association between smoking and compromised periodontal health. Limited evidence also suggests smoking interferes with non-surgical and surgically related periodontal therapies. These studies reported outcomes related to commonly evaluated periodontal endpoints such as pocket depth, clinical attachment loss, periodontal bone level, and number of teeth lost.
Patients may be periodontitis susceptible as a result of numerous host, environmental, and genetic factors, and smoking can be a cofactor. Evidence suggests smoking is an additional risk factor for tooth loss during periodontal maintenance. Therefore, although periodic periodontal therapy may assist in managing periodontitis for patients who smoke or have a history of smoking, this therapy may not reduce the larger systemic burden that smoking has on patient susceptibility, resistance, and healing. The best method to reduce the risk of smoking on periodontal disease is to not smoke.

**Smoking, Dental Implant Failure, and Peri-implant Disease**

Smoking is associated with increased risk of implant failure (RR = 2.23), increased risk of postoperative infections (RR = 2.01), and greater marginal bone loss. Smoking is also reportedly correlated with a greater peri-implant disease incidence, including peri-implant mucositis and peri-implantitis. Periodontitis and a history of periodontitis may also be associated with peri-implant biological complications. Periodic periodontal therapy may assist in reducing the incidence of peri-implantitis. When surgical intervention is considered to improve peri-implant supportive tissues, smoking or smoking history may compromise healing and the treatment outcome. The best method to reduce the effect of smoking on implant failure and peri-implant disease is to not smoke.

**Smokeless Tobacco and Gingival Recession**

Smokeless tobacco has also been associated with localized gingival recession and could lead to mucogingival defects and caries. Recession can be corrected surgically, and numerous studies describe methods to address the loss in gingival tissue height that arises from frequent and repeated tobacco use in the same intraoral location. Cessation of tobacco use will not reverse gingival recession. Soft tissue pathosis, dentin hypersensitivity, surgery, and caries originating from tobacco use can be avoided by not using smokeless tobacco.

**Oral Mucosal Changes with Smoking and Smokeless Tobacco**

Smoking is associated with a number of surface epithelial changes that affect tissue appearance. For example, smoker’s melanosis associated with cigarette or pipe smoking can occur on maxillary and mandibular alveolar mucosa or buccal mucosa and commissures, respectively. Nicotinic stomatitis from smoking is associated with hard palate mucosa, including hyperkeratosis and acanthosis of palatal epithelium, inflammation of salivary glands and connective tissues, and salivary gland duct squamous metaplasia. Cessation of tobacco use leads to disappearance of pigmentation or stomatitis over time.
Oral mucosa leukoplakia is potentially premalignant\(^2^4\) with a reported prevalence among heterogeneous studies of 2.60\%.\(^2^5\) Incidence may be 6 to 10 times greater in smokers compared to non-smokers.\(^2^6\) A smoking dose-response relationship also exists with developing oral leukoplakia, and the lesion may regress upon smoking cessation.\(^2^7\) When a leukoplakia is identified during oral cancer screening, smoking cessation should be recommended, and the patient should be referred for appropriate medical follow-up and monitoring.

Smokeless tobacco use is associated with buccal or labial mucosal changes at the site where the individual holds the tobacco. This keratosis is dependent upon the type of tobacco and how it is used. The surface epithelium exhibits hyperkeratosis and acanthosis. Continuous smokeless tobacco use might place the individual at greater risk of carcinoma due to the higher concentration of nitrosamines in the processed tobacco. Conversely, mucosal tissue can resolve to normal within weeks after cessation.\(^2^3\)

**Oropharyngeal Cancer**

Patients with oropharyngeal cancers generally have a 5-year survival of not more than 50 percent.\(^2^8\) Oral cancer is usually found in the floor of the mouth, the ventrolateral surface of the tongue, and the soft palate. Among cigarette smokers, nearly all cancers were found in these locations.\(^2^9\) Heavy tobacco users have a 5- to 25-time greater risk of oral cavity and oropharynx cancer.\(^3^0\) Furthermore, those who smoke and drink heavily further increase risk compared to each factor individually.\(^3^1\)-\(^3^4\) Those who heavily drink and smoke may have a 35-time greater risk than those who neither smoke nor drink.\(^3^5\) Asymptomatic, early squamous cell carcinomas are generally reddish in appearance, may have a granular or smooth surface, and are not elevated or elevated < 1 mm. They also typically have intact surface epithelium, are not ulcerated, have no bleeding, and no induration.\(^3^6\)

For those with increased risk based on smoking and alcohol drinking habits, the need for periodic oral cancer screenings is emphasized to ensure early recognition and treatment of a potentially life-threatening oropharyngeal lesion.

**Impact of Tobacco Use on Prosthodontic Therapy**

The prosthodontist’s goal is to provide patient-centered care that meets patient expectations by means that are esthetic, functional, affordable, and predictable. Evidence suggests smoking can compromise a patient’s long-term prosthetic outcome whether the prosthesis is natural tooth or dental implant supported. These compromises could relate to tooth loss, ongoing caries management, periodontitis, the need for dental prostheses, the need for ongoing prosthetic maintenance, dental implant failure, peri-implantitis, and possibly early prosthetic replacement. In addition, the use of smoked or smokeless tobacco is associated with mucosal changes and an increase risk of oropharyngeal carcinoma.
For all the above reasons, patients who smoke should be counseled to cease smoking for their individual oral and systemic health. For those who are partially or completely edentulous, smoking cessation may still reduce the incidence of complications, thereby improving long-term prognosis for prosthodontic care. The best means of eliminating the impact of tobacco on oral health is by never beginning its use.

References


References cont.


References cont.


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