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The Impact of Tobacco Smoking and Electronic Cigarette on Periodontal Health: Narrative Review

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Abstract: *Periodontal diseases are defined as an inflammatory process associated with bacterial activity and mediated by the host immune response. This aggression can result in loss of connective tissue attachment and consequently bone loss. It is well established that cigarette smoking is considered a risk factor in the development of periodontitis. The progression of the disease is directly related to the frequency of smoking, with heavy smokers showing more severe forms of the disease compared to light smokers.*

In recent years, e-cigarette smoking has become a popular recreational activity among adolescents and young adults in America. Some previous studies show that direct exposure to e-liquids has also been shown to produce harmful effects on ligament cells. Numerous case reports demonstrate the presence of oral mucosal lesions, tongue or ulcerative lesions, dental caries, toothache, periodontal disease in patients using e-cigarettes, as recently reviewed, as well as dental fractures and avulsions due to e-cigarette explosions.

The objective of this literature review was to describe the impact of tobacco smoking and electronic cigarette on periodontal health. A narrative literature review was carried out with the search for the crossing of words "electronic cigarette", "Tobacco", "Smoking", "Periodontitis", "Nicotine", both in Spanish and English in the databases of PubMed, Google Scholar including original articles, literature reviews, meta-analysis and clinical cases. ***Conclusion:*** *Based on the recorded literature, it is concluded that smoking influences cellular, molecular, microbiological, periodontal surgical procedures, periodontal maintenance and post-therapy periodontal tissue repair. In addition, the use of electronic cigarettes may equally affect people's periodontal health when compared to conventional cigarette smoking.*

Keywords: *Periodontal disease; electronic cigarette; tobacco smoking; cigarette.*

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I. Introduction

The World Health Organization (WHO) cites tobacco-related death and disease as drivers of poverty, as they force people to bear high medical expenses. It is well established that cigarette smoking is considered a risk factor in the development of periodontitis (1-3).

Electronic cigarettes may influence periodontal health. Previous studies show that direct exposure to e-liquids has also been shown to produce harmful effects on periodontal ligament cells (4).

Electronic cigarettes (ECIG) first appeared on the market in 2007 as an aid to smoking cessation. (5) In recent years, e-cigarette smoking has become a popular recreational activity among adolescents and young adults in America. There are currently more than 40 million e-cigarette users worldwide, in 2014, there were an estimated 2.4 million ECIG smokers in the United States (6).

New nicotine products, particularly e-cigarettes, have become increasingly popular in the last decade. E-cigarettes are sometimes considered a less harmful alternative to tobacco use and there is some evidence of their potential role as an aid to smoking cessation. However, there are concerns about their health consequences, especially in users who are not tobacco smokers, and also when used long-term (7).

Vaping involves using an electronic device with a specifically formulated liquid that usually, but not always, contains nicotine. (8) The nicotine-free cigarette still poses risks of exposure to chemicals released in the process of heating the device, such as aluminum, copper and lead. (9) The liquid is heated in the device and forms a viscous aerosol that is inhaled by the smoker. Some of the aerosol is absorbed into the bloodstream, some remains

attached to structures in the oral cavity, and the rest is expelled into the atmosphere. The device consists of four components: a mouthpiece, a tank or reservoir to contain the liquid, a heating element and a lithium battery (10).

Although there are countless variations in product design, e-cigarettes typically include the following basic components: airflow sensor; microprocessor; battery; aerosol generator; and solution tank (11).

There are currently more than 10,000 e-liquid formulations available on the market. These formulations basically have three components: a base, nicotine and flavors (12).

E-liquid usually consists of nicotine, propylene glycol and/or glycerol carriers, flavoring agents and other possible additives (e.g., ethyl alcohol, stabilizers, pharmacologically active compounds without nicotine).
They are available in a wide variety of flavors. These flavors provide tastes or fragrances similar to candy, fruit, baked goods, beverages, menthol and tobacco. The chemical components of these flavors include saccharides, esters, acids, and aldehydes (12).

In vitro studies have shown that some of these flavors significantly promote biofilm formation and enamel demineralization compared to a base/nicotine control when used in electronic cigarette devices (13).

The aim of the present study is to conduct a scientific literature review on the relationship of smoking and e-cigarette in the oral cavity and periodontitis.

II. Prevalence of e-cigarettes and periodontitis

According to the WHO Global Report on Trends in Tobacco Use Prevalence 2000-2025 (third edition) the total number of tobacco users worldwide has decreased from 1397 billion in 2000 to 1337 billion in 2018.

Global Burden of Disease study (2017) suggests that periodontitis is the sixth most prevalent condition worldwide. Its frequency has increased slightly since 1990 and ranges from 10.5% to 12% of the population, depending on the region.

Periodontal diseases are defined as an inflammatory process associated with bacterial activity and mediated by the host immune response. This aggression can result in loss of connective tissue attachment and consequently bone loss (8).

At a time when various measures are being taken to reduce smoking and limit smoking in public places, electronic cigarettes have emerged as an alternative to cigarettes and as a possible smoking cessation tool (14).

E-cigarette smoking has become a popular recreational activity among adolescents and young adults. It is well established that cigarette smoking is considered a risk factor in the development of periodontitis. Patients who smoke have been shown to suffer more severe forms of periodontitis (8).

Public opinion considers the use of electronic cigarettes to be a healthier alternative to combustion tobacco and to reduce the risks of morbidity and mortality, mainly cancer (lung, mouth) and cardiovascular diseases (15,16).

The term "smoking" has given way to the term "vaping", which consists of inhaling a "liquid" in the form of an aerosol, produced by a small electronic vaporization device that does not require combustion.

Disease progression is directly related to smoking frequency, where heavy smokers show more severe forms of the disease compared to light smokers. Different studies classify smoking frequency differently, but according to one review, smoking less than 9 cigarettes per day is considered light smoking and more than 31 is considered heavy smoking (17).

III. Periodontal disease

Periodontal disease is referred to as a chronic inflammatory disease that leads to the destruction of periodontal tissues where components and supporting structures of the teeth (gingiva, bone, and periodontal ligament) are affected, which could result in tooth loss and contribute to systemic inflammation (18). The pathophysiological situation of the disease persists through episodes of activity and inactivity, evolving until the affected tooth is extracted or the microbial biofilm is therapeutically removed and the inflammation subsides (19). The severity of the disease depends on environmental and host risk factors, both modifiable (smoking) and non-modifiable (genetic susceptibility) (19).

IV. Risk factors

Risk factors, such as diabetes, genetics and smoking, have been associated with susceptibility, prevalence and severity of the disease (3,2). It is well established that cigarette smoking is considered a risk factor in the development of periodontitis. A recent systematic review showed that smoking increases the risk of developing periodontitis by 85% (20).

It has been shown that patients who smoke suffer more severe forms of periodontitis. (4) The progression of the disease is directly related to the frequency of smoking, where heavy smokers show more severe forms of periodontitis compared to light smokers (9).

Different studies categorize the frequency of smoking differently, but according to one review, smoking less than 9 cigarettes per day is considered light, and more than 31 is considered heavy smoking (6).
The safety of these electronic devices is also questionable because of the potential risk of burns, lip lacerations and hematomas, neck fractures and dental injuries, such as fractures and avulsions, are some of the complications described in the literature (21).

V. Smoking

Smoking is the most important environmental factor associated with non-inflammatory diseases of the oral cavity, in addition to being a major cause of periodontitis, which is a limiting factor in dental health (22). Not only smoking, vaping could influence periodontal health. Some previous studies show that direct exposure to e-liquids has also been shown to produce harmful effects on periodontal ligament cells (4). Reactive aldehydes derived from e-cigarette aerosol can cause carbonylation of proteins, which can lead to matrix destruction and bone loss during periodontitis.

Vaping without nicotine still poses risks of exposure to chemicals released in the heating process of the device, such as aluminum, copper, and lead (2). Tobacco contains more than 7,000 toxic chemicals, including several known human carcinogens (23). E-liquid usually consists of nicotine, propylene glycol and/or glycerol carriers, flavoring agents, and other possible additives (e.g., ethyl alcohol, stabilizers, non-nicotinic pharmacologically active compounds) (24).

Each flavor has a unique chemical profile and, therefore, probably a unique toxic release profile. Importantly, many of the flavorings added to liquid ECIG are generally recognized as safe when ingested orally, but have an unknown safety profile when heated, aerosolized, and inhaled (25).

VI. Relationship between periodontitis and smoking

Smoking can dysregulate immune responses, leading to increased or suppressed inflammation at mucosal surfaces, impaired immunity to pathogens, and modulation of micro vascularization affecting healing and resolution of inflammation (25).

In vitro studies have shown that e-cigarette exposure has a relatively modest effect on oral streptococcal growth and survival compared with cigarette smoke (26). Evidence suggests that the risk of periodontal disease associated with e-cigarette use is lower than that associated with smoking, but higher than that observed in nonsmokers (22).

VII. Oral manifestations of e-cigarettes and smoking

Associations with a wide range of conditions have been reported, including throat irritation, gingival bleeding, and oral trauma from e-cigarette device bursting.

Numerous case reports demonstrate the presence of oral mucosal lesions, tongue or ulcerative lesions, dental caries, toothache, periodontal disease in patients using e-cigarettes, as recently reviewed, as well as dental fractures and avulsions due to e-cigarette blasting (7, 27).

Oral signs of smoking, such as halitosis and dental pigmentation, are easily detectable by oral health professionals, which puts them in a unique and advantageous position to offer smoking cessation, even when no pathology is present (28, 29).

Hyperplastic candidiasis constitutes a relatively frequent lesion of the of the oral mucosa in e-cigarette users. It was found in a high percentage in the retro commissural area of the group of electronic cigarette consumers potentially favored by a pH change induced by the chemical compounds present in the liquids (30).

Nicotine affects the exocrine glands, increasing salivary secretion, so the increase in the amount of tartar in smokers may be due to an effect of tobacco smoke on the properties of saliva. Nicotine is also associated with an increase in the prevalence of C. albicans, promoting its growth and proliferation (31).

With certain flavors, researchers were able to show a fourfold increase in microbial adhesion to enamel, a twofold increase in biofilm formation, and a 27% decrease in enamel hardness. By-products of propylene glycol heating are acetic acid and lactic acid, which could contribute to enamel demineralization. The "dry mouth" sensation created by some vaporizing liquids could also lead to excessive consumption of soft drinks and sports drinks (13). COVID-19 is associated with the use of e-cigarettes by young people and with dual use of e-cigarettes and cigarettes (32).

VIII. Consequences of smoking on periodontal treatment

Smoking also has an impact on the response to periodontal treatment; smokers show only a 50% to 75% improvement in their clinical parameters after scaling and root planning compared to non-smokers (9).

Studies have shown that the results of non-surgical periodontal therapy are compromised in cigarette smokers compared to never smokers (33-35).

Long-term exposure to cigarette smoke has been reported to compromise the growth of human gingival fibroblasts, increasing collagen degradation (36).

Another study analyzed the effects of smoking on periodontal parameters and found significant increases in plaque index, pocket depth and clinical attachment loss levels in cigarette smokers compared to non-smokers (37).

Tobacco smoking has been shown to produce a proinflammatory effect by stimulating the secretion of specific cytokines and radical oxygen species that play a role in the destruction of periodontal tissues (38). In addition, nornicotine (a metabolite of nicotine) stimulates the formation of reactive oxygen species, which endanger periodontal tissues (39).

These factors may compromise periodontal wound healing and the regenerative capacity of periodontal tissues of smokers. Other proposed mechanisms for compromised postoperative periodontal healing in smokers include: (a) altered neutrophil function, (b) amplified prevalence of periodontopathogen microbes, (c) decreased vascular outflow and lymphocyte proliferation, (d) reduced production of immunoglobulin G, (e) impaired fibroblast adhesion and function (28,40).

The pathways by which smoking affects the incidence and progression of periodontitis are unclear. However, some hypotheses about possible mechanisms have been put forward. Among them is the effect of smoking on the composition of the microbiota, on the immune response and on the healing capacity of the periodontium (41). Smoking may lead to a change in the composition of the subgingival biofilm with an increase in the prevalence of periodontal pathogens. In addition, smoking has been implicated in delaying the recruitment of neutrophils into periodontal tissues, which would increase the acute immune response. This fact would increase the threshold of aggression necessary for the periodontal tissue to initiate the inflammatory cascade. In addition, it has been suggested that humocould shift the balance of neutrophil activities towards a destructive one (42,43).

IX. Conclusion

Based on the recorded literature, it is concluded that smoking influences cellular, molecular, microbiological, periodontal surgical procedures, periodontal maintenance and post-therapy periodontal tissue repair. In addition, the use of electronic cigarettes may equally affect people's periodontal health when compared to conventional cigarette smoking.

From the clinical point of view, the dentist should have more influence on the patient to quit smoking, since the relationship that develops in the office has a direct impact on the results of the treatment, so multidisciplinary teams should be formed to work to ensure that as many patients as possible quit smoking.

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