SMOKING: COSTING YOU MORE THAN MONEY

STUDENT NAME

 Going into a gas station or market, according to Laxman and Annaji (2008), an estimated 1.1 billion people in this world pay a ridiculously expensive price for their smoking habits. What they do not know is that smoking is costing them more than just the money they provide. There has been ongoing research and studies linking smoking and periodontal disease since mid-last century. During my time researching I have come across a few conflicting conclusions, but none that deviate from the ultimate fact that smoking is an independent risk factor that directly affects the initiation and severity of periodontal disease. Coming from a family of smokers, I felt convicted to learn more about its negativeeffects in the oral cavity in hopes to shed some light on the topic with my family members. As a hygienist, I was intrigued to know the effects of smoking on the oral tissues, if pathogenicity of biofilm differs from that of a non-smoker and how smoking interferes with healing and the success of periodontal therapy. These three components will help me recognize the different characteristics of oral tissues affected by smoking, aid me in educating a patient, as well as allowing them to understand how smoking could hinder the success of their therapy.

 Within the oral cavity we have what is known as the periodontium. The periodontium is made up of essential parts that allow for tooth stability and function; the alveolar bone is your tooth socket that houses your tooth, the periodontal ligament anchors the tooth from the bone to the cementum by bundles of fibers, the cementum protects the dentin, and the gingiva is your protective tissue layer for all those previously mentioned. These structures are the target of destruction for periodontal pathogens. In a normal healthy mouth, the onset of disease would start with reddening of the gums, inflammation and bleeding. As we have learned through the course of this hygiene program, when an individual is a smoker their gingival tissues are less likely to exhibit these symptoms (even while they’re diseased) due to the vasoconstriction of the tissues caused by the ingestion of Nicotine. During this time, the gingival tissues are starving for blood and nutrients needed for function and health. I think that this is one of the main factors as to why this disease in smokers tends to readily progress into periodontitis or even worse periodontal disease. Smoking suppresses vascular reaction, ultimately restricting the inflammation and bleeding(Perry, Beemsterboer, & Essex, 2014, p. 66). Regardless of the type of tobacco used, *Periodontology for the Dental Hygienist*explain that the toxic effects go further affecting the oral cavity at a cellular level; Smokers show increased keratinization in the epithelial cells and Polymorphonuclear Leukocytes have a reduced capability to phagocytize substances. I believe this cellular activity is essential in creating a state of homeostasis within the tissues. They also mentioned that individuals that smoke tend to harbor more nicotine metabolites in their crevicular fluid and saliva(Perry et al., 2014, pg.66), ultimately making it more difficult to restore oral conditions. Wilkins(2013) also discussed cellular impairment;he stated once the periodontal diseasehas developed, immune response is compromised; neutrophils are impaired therefore decreasing adherence, chemotaxis, and phagocytosis.Our bodies’ building blocks are cells, and if they are impaired it hinders the essence of our wellbeing- not just in our oral cavity but also in our entire body.

 The destruction of the oral tissues while you have periodontal disease is related to the biofilm accumulation within your oral cavity. Prior to researching this topic, I assumed that smokers had greater pathogenicity within their biofilm due to the increase in toxins from the nicotine. However, analyzing the impairment of cell function and lack of vascularization in the oral tissues it haslead me to wonder about which microbesare more suitable to survive in those conditions. In the articles I read, they presented a few different ideas regarding this concept. For example, in the journal article “Smoking and Periodontal Disease”the author reflects on the finding of a study conducted by Bagaitkar and Associates regarding the exposure of P. gingivalis (2012). Their research concluded that most likely, tobacco smoke represents an environmental stress to which P. gingivalis adapts and can alterthe expression of several virulence factors(Borojevic, 2012). For example, it may contribute to a micro-flora with less potential for inflammation. Laxman and Annaji(2008) also touched upon the pathology of smokers versus non-smokers; they referenced a study conducted in Erie County that resulted in smokers having higher proportions of A. actinomycetemcommitans, P. gingivalis, and T. forsythesis and they reported higher prevalence of other exogenous organisms in smokers such as Escheria coli and Candida albicans. Over all, the literature I read all acknowledged further research needs to be done; at this moment there are a lot of unknown underlying factors that may be affecting the results when it comes to tobacco use/smoking and periodontal disease.

 When treating periodontal disease the main objective is to reduce the pocket depth and maintain bone levels by removing all supra-gingival and sub-gingival irritants. Pocket reduction will allow an individual to gain more attachment from tooth to bone, for more stability and function. Ultimately, my goal as a hygienist (when possible)and a huge benefitof pocket reduction is having a pocket depth that is possible for the patient to keep clean and maintain. This is more difficult for smokers, studies show that after scaling and root planing therapy they have less probing depth reductions and less attachment gain than nonsmokers(Perry, Beemsterboer, & Essex, 2014, p. 106). A lot of factors contribute to the disruption and impairment ofa smoker’shealing response. A large factor is the lack of blood flow and vascularity of the gum tissues to stimulate cellular activity. In addition, levels of toxins from the Nicotineactively alter antibody production of IgA and IgG found in the oral cavity(Laxman & Sridhar, 2008, p. 5). More specifically, it affects IgG, weakening a smoker’s immunity against the pathogen A. actinomycetemcommitans(Laxman & Sridhar, 2008, p. 5). This not only delays healing but may leave a patient more susceptible to bacteria and infections in the area treated, ultimately jeopardize the success of the procedure.

 As a clinician, recognizing signs that may indicate disease is imperative. During my research, I was able to gain knowledge on, not only what to look for, but also understand why the tissues react as they do and what may be the causative factors. Given the amount of people who habitually smoke, when treating these patients I can be confident in explaining to them what is occurring at a more microscopic level. I believe that the more information I can provide as a clinician, the better I can overcome objections and treatment resistance. I feel this is necessary because the average person normally does not take periodontal disease seriouslyin comparison to many other conditions since they cannot see or feel it. In regards to pathogens found in periodontal patients, I am really looking forward to learning more about the presence of nicotine in the oral cavity and its effects on microbial growths and resistance. This is the one area that I don’t think I will be discussing with my patients, but I would love to learn more about for my own personal gain given that my articles only touched on the subject very broadly. In all, smoking affects both the person and their prognosis when seeking treatment and I believe the literature I analyzed truly supported this idea. As a practicing hygienist, with the goal of seeking optimal health and recovery- if a patient continues to smoke they place a limit on the healing response to their therapy. I will definitely be placing a new emphasis not only on the treatment needing to be rendered, but also on the fact that it does not stop once they leave the dental office. In order to ensure results, the patient needs to do their part and think of what is best for them; ideally, the best thing would be for them to be proactive with their hygiene along with quitting smoking.

 Ever since I can remember I have always been anti-cigarettes and anti-smoking, this paper simply helped me reinforce the reasons why it is a terrible habit to have from a clinical standpoint. When I start actively practicing as a dental hygienist, I feel convicted to push for the cessation of smoking. I plan to find local resources, smoking cessation programs, alternatives that may help individuals who truly want to make the effort. I am not saying smoking is the only problem contributing to periodontal disease; however, it is a crucial component in delaying diagnosis since it masks many of the signs and it also impairs healing and the immune response of oral tissues after therapy. The treatment of periodontal disease goes far beyond just the two-hour appointment at the dental office, it requires a life style change; prioritizing oral hygiene and maintenance as a main component, possibly changing their diet and quitting unhealthy habits like smoking. The effects of smoking go far beyond what I have discussed previously, it creates more grave health issues like contributing to oral cancer, lung cancer, cardiovascular issues etc. These are life-threatening complications, far beyond periodontal disease. As a hygienist I will be asking mypatients’ one question: Is it worth it?

References

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