

REVIEW ARTICLE

Effect of Smoking on Wound Healing

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ABSTRACT

Smoking has long been suspected to adversely affect wound healing. It causes various ill-effects including premalignant lesions and cancers. Tobacco affects postoperative wound healing following surgical and nonsurgical tooth extractions, routine maxillofacial surgeries, implants, and periodontal therapies. Smoking tobacco is also associated with catecholamines release, resulting in vasoconstriction and decreased tissue perfusion. Smoking is believed to suppress the innate and host immune responses, affecting the function of neutrophils, the prime line of defense against infection. Thus, the association between smoking and delayed healing of oral tissues following surgeries is evident.

Keywords: Dental extractions, Dry socket, Healing, Smoking, Tobacco.

How to cite this article: Mufti N, Mufti S. Effect of Smoking on Wound Healing. *Int J Prev Clin Dent Res* 2016;3(4):288-290.

Source of support: Nil

Conflict of interest: None

INTRODUCTION

Tobacco appears to be as old as human civilization and was introduced into India by Portuguese traders during AD 1600. It spread like fire to such a great extent that today India is the second largest producer and consumer of tobacco in the world.¹ Smoking has its influence on general as well as oral health of an individual. A primary relationship between smoking and coronary heart disease, stroke, subclinical atherosclerosis, chronic obstructive pulmonary disease, pneumonia, low birth weight, and various cancers has been established without doubt. Pregnant women who smoke tobacco have increased risk of stillbirth.² As far as oral health is concerned, it increases the risk of periodontal disease, oral

precancerous and cancerous lesions, root caries, and peri-implantitis. It also causes taste derangement, staining of teeth and restorations, as well as delayed wound healing after extractions, periodontal procedures, and orthognathic surgeries. Smoking upregulates the expression of proinflammatory cytokines, such as interleukin-1, which contributes to increased tissue damage and alveolar bone resorption.³ Nicotine may have an effect on cellular protein synthesis and impairs the gingival fibroblast's ability to adhere, thus interfering with wound healing and/or exacerbating periodontal disease.⁴ The exact mechanism by which smoking compromises wound healing is unknown. Various mechanisms hypothesized include cytotoxicity of nicotine, carbon monoxide, and hydrogen cyanide to the cells involved in wound healing, vasoconstriction, and decreased tissue perfusion due to catecholamine release, increased platelet adhesiveness, and blood viscosity, leading to the augmented risk of microvascular occlusion, increased levels of fibrinogen, carboxyhemoglobin, and compromised polymorphonuclear leukocyte function.^{5,6}

SMOKING AND ITS EFFECTS ON WOUND HEALING

Wound healing is a dynamic process that involves number of phases, i.e., inflammatory phase, proliferation phase, and maturation phase. Inflammatory phase is the body's natural response to injury. After injury, clot is formed. Once the hemostasis is achieved, blood vessels dilate to allow the essential cells, antibodies, white blood corpuscles, growth factors, enzymes, and nutrients to reach the injured area. Neutrophils and macrophages come into their action. During proliferative phase, fibroblast lay down the collagen to form the granulation tissue along with angiogenesis and later epithelialization occurs. Maturation phase involves remodeling of collagen. Cellular activity reduces and the number of blood cells decreases in number. When cigarette is inhaled, the toxins can directly poison the cilia or pass the cilia barrier, undergo tissue absorption, enter into bloodstream, and gain access to other parts of body. Cigarette smoke constitutes number of toxic contents with each cigarette smoked, approximately 20 to 30 mL of carbon monoxide and 2 to 3 mg of nicotine are inhaled.⁷ Carbon monoxide produced during combustion of tobacco also reduces capillary blood flow.

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A clinical study has shown that a single cigarette can reduce the peripheral blood velocity by 40% in 1 hour.⁸ Nicotine exerts several effects that influence wound healing. First, proliferation of red blood corpuscles (RBCs), fibroblasts, and macrophages is diminished.⁷ Second, it increases platelet adhesiveness that causes microclot formation, raising the risk of microvascular occlusion and tissue ischemia.⁹ Third, nicotine produces cutaneous vasoconstriction. In addition, when carboxy-hemoglobin levels rise in blood, the oxygen dissociation curve shifts to the left, which means oxygen is less able to dissociate from RBCs and diffuse into the tissues, so the decrease in the levels of oxygen available for tissue perfusion results into cellular hypoxia and diminished wound healing.^{10,11} Wound healing also requires enzymes. Hydrogen cyanide inhibits enzyme system, i.e., required for oxidative metabolism and oxygen transport at cellular level.¹² Altogether, the ill-effects of toxic substances of smoking have potential to diminish the conditions that are required for proper wound repair and healthy scar formation.

DRY SOCKET

Information on number of cigarettes smoked per day and the postoperative smoking habit of each patient was recorded by Rees TD et al.¹² According to them, heat released from burning tobacco and tobacco along with its byproducts may act as contaminant in the surgical site together with the suction applied to the cigarette that might dislodge the clot from the alveolus interfering healing of the socket. According to Meechan et al,¹³ fibrinolytic activity caused by smoking decreases the blood supply to the surgical site after extraction, and dry socket was found common amongst these smoker patients. Extractions were performed in 2,417 patients and incidence of painful socket was analyzed. It was observed that postoperative poor filling of blood in socket was found more likely to develop painful socket. Postoperative socket filling with blood was greatly reduced in smokers compared with nonsmokers. Incidence of painful socket in heavy smokers (who smokes 20 or more cigarettes per day) was higher compared with nonsmokers. According to López-Carriches et al,¹⁴ after extraction of third molar, smokers were more prone to develop trismus than nonsmokers. However, they did not observe significant difference in relation to pain. According to Sweet and Butler,¹⁵ the suction associated with cigarette might dislodge the blood clot from alveolus socket and interrupt the healing. Though the exact mechanism by which smoking predisposes the socket become painful remains unclear, smoking seems to be strictly associated with the occurrence of dry socket.

INTERFERENCE OF SMOKING ON PERIODONTAL THERAPY

Cigarette smoking is a significant risk factor for periodontal diseases and impairs healing after periodontal surgeries.¹⁶ A number of clinical studies have been performed to compare the response of smokers and nonsmokers to different types of surgical and nonsurgical periodontal therapies,¹⁷⁻²¹ and it was found that smoking has a strong negative impact on regenerative therapy, including osseous grafting,¹⁹ guided tissue regeneration,²²⁻²⁵ or combination of these treatments,²⁶ and 80% failure rate in the treatment of furcation involvement defects. The majority of studies found that gingival grafting for root coverage procedure is less successful in smokers than nonsmokers.²⁷⁻²⁹

SMOKING AND HEALING POSTORAL SURGERY

Smoking is reported to be an important factor responsible for postoperative infections, leading to hindrance in bone healing. A meta-analysis by Rosen PS et al²³ reported the magnitude of the association between cigarette smoking and bone mass and showed that smokers presented reduce bone mass, compared with nonsmoker.¹⁴

DISCUSSION

Cigarette smoking affects the oral cavity in various ways, ranging from staining of the teeth to more serious diseases, such as oral cancer. Smoking has various risk factors including impaired wound healing. Treatment complications and failures in smokers inspired them for investigation of relative risks associated with smoking and the mechanism for compromised wound healing. Harmful effects of nicotine were believed to damage erythrocyte precursors as well as vasoconstriction and inhibition of epithelization. Prevalence of ulcerative gingivitis, acute necrotizing ulcerative gingivitis, and periodontitis is also increased in smokers.³⁰ Pindborg³¹ observed tissue ischemia as a etiology for these diseases based on gingival vascular anatomy and the lack of collateral circulation to the papillary gingival.

CONCLUSION

The components of cigarette smoke clearly have an inhibitory effect on wound healing. Combined effects of nicotine, carbon monoxide, and hydrogen cyanide are tissue ischemia, cellular hypoxia, inhibition of proliferation of epithelial cells, vasoconstriction, poisoning of enzymes, and a decrease in oxygen carrying capacity of blood cells required for wound healing. Smokers undergoing any oral surgery should be encouraged by their dentists to cease smoking, emphasizing that smoking can increase

complications and reduce the success rate of these procedures. Clinician has to decide whether or not to commence any complicated treatment in high-risk situations, but once it is decided to go ahead, the patient's informed consent is essential before starting treatment.

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