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Smoking as a Risk Factor for Dental Implant: A Literature Review

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ABSTRACT

Implants are now widely used as a method for replacing missing teeth. Along with its high success rate implant shows many failures too. Various factors are responsible for causing implant failure. Smoking is one of the commonest causes. Smoking being one of the deleterious habits causes harmful effects on our body. Similarly, it shows its harmful effects on implant. The aim of this review article is to highlight these effects. Implant failure rates are seen more in smokers when compared. Osseo integration is affected by components of smoke leading to failure. Maxillary site is more prone for marginal bone loss and has shown higher rates of failure.

Key words: Smoking, Implant, Osseo integration, Implant failure

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INTRODUCTION

The ability of replacing missing teeth with implants placed directly into jaw is one of the most important advancements in modern dentistry. Implant dentistry can solve a variety of restorative issues, ranging from one tooth to a whole arch. As a result, dental implant therapy has become the most important aspect of clinical treatment. Along with its increasing popularity implant failures, on the other hand, are still common and are concern for patient and dentist [1].

The following two groups of risk factors for implant failure can be found:

- Patient related aspects include patient's overall health, smoking habits, bone amount and quality, oral health and so on [2].
- Implant characteristics like dimensions, operative technique, type of implant and its location, coating, time between extraction of tooth and insertion of implant, and between placement of implant and loading [3].

Out of above mentioned factors smoking remains one of major factor responsible for causing failure of implant [4,5]. Smoking is a common practice among people all around the world. In the year 2000, 4.83 million individuals died from nicotine addiction over the world. This figure alone is adequate to demonstrate the harm that tobacco does [6]. Nicotine is the major component of smoke which raises the respiration rate, blood pressure and heart rate, as well as making them more active. Unfortunately, these effects fade after about 20 minutes, leaving the tobacco user needing another take [7].

LITERATURE REVIEW

Effects of smoking

Cigarette smoking releases almost 4000 different gases and compounds, like nitrogen, carbon dioxide, carbon monoxide, hydrogen cyanide, benzene, nicotine, anatabine, anabasine, ammonia and nornicotine, out of which affinity of carbon monoxide for haemoglobin is greater than that of oxygen, causing oxygen to be displaced from the haemoglobin and causing a decrease in tissue oxygen tension [8]. Nicotine, a highly addictive component of cigarette smoke, has been linked to a number of illnesses [9,10].

Smoking has an impact on a person's overall health as well as their oral health [11]. It causes periodontal disease, root caries, and peri-implantitis, as well as increases the risk of premalignant and malignant lesions in the mouth. It also stains teeth and restorations, affects taste, and delays healing of wound of various oral surgeries. By increasing expression of pro-inflammatory cytokines like IL-1, smoking leads to alveolar bone resorption and also causes tissue damage [12]. Dry socket is frequent among smokers due to fibrinolytic activity produced by smoking [13].

High amounts of nicotine exposure may affect bone growth following distraction osteogenesis [14].

Effects of smoking on periodontal health

The fact that smoking is linked to a higher severity and prevalence of periodontal disease shows that the host-bacterial interactions cause more extensive periodontal damage. Changes in the sub gingival plaque composition leads to changes in host response to bacterial challenge and creates imbalance between bacterial challenge and host response [15].

Microbiological effects

Members of the red and orange complexes were found to be significantly more prevalent in smokers than nonsmokers which included Peptostreptococcus micros, Tannerella forsythia, Treponema denticola, Prevotella intermedia and others [15]. Increased incidence of periodontal infections was due to increased colonisation of these microbes at shallow sites with pocket depth up to 4 mm. No differences found between smokers and non-smokers with pockets greater than 4 mm. It was seen that mandible is less prone for these organisms than maxilla, therefore resulting in more damage in maxilla.

Immune inflammatory responses

Smoking has a significant impact on the immune inflammatory response, which leads to a rise in the spread and severity of periodontal disease. Smoking's negative effects appear to be due to suppression of the immunological response by bacterial invasion. Neutrophils are a crucial part of host response to bacterial infection, and changes in their quantity or function can lead to systemic and localised illnesses. Phagocytosis, killing and chemo-taxis are the important actions performed by neutrophils. Neutrophils from the oral cavity, saliva and peripheral blood of smokers have shown functional changes in all these actions [15].

Furthermore, levels of antibodies to periodontal pathogens, which are required for phagocytosis and bacterial death mainly immunoglobulin G_2, have been found to be lower in smokers suggesting that smokers may have less protection against periodontal bacteria. Increased amounts of tumour necrosis factor-a, as well as prostaglandin E_2, neutrophil elastase, and matrix metalloproteinase-8, have been observed in gingival crevicular fluid of smokers [15].

Physiological effects

When comparing smokers to non-smokers, the microcirculation appears to respond differently to plaque accumulation. As the inflammation develops in smokers, there is reduction in gingival crevicular fluid, gingival blood vessels and bleeding on probing. Smokers have lower sub gingival temperatures than non-smokers, and it takes them longer to recover from the vasoconstriction induced by local anaesthetic application. These findings imply that smokers' gingival microvasculature differs significantly from that of non-smokers and that these differences result in lower blood flow and clinical symptoms of inflammation [15].

Effect of smoking on mucosal immunity

The initial line of defence in oral and respiratory mucosa is made up of a variety of cell types, all of which play important role in guarding against infections and toxicants. Epithelial cells present in mucosa of respiratory tract provide physical and mechanical barrier against harmful smoke particles by mucociliary action [16]. They also release cytokines and chemokine which help in regulating host defence response. Neutrophils, macrophages, lymphocytes and monocytes monitor airway mucosa and help in phagocytizing inhaled particles. This mucosal cell types work together to generate an integrated defence response that protects us from any disruption induced by inhaled toxins that could otherwise harm us. Tobacco smoke initiates multiprolonged attack on the mucosal cells, resulting in detrimental effects. It has negative effect on mucociliary cascade leading to increased mucus formation and influencing cytokine and chemokine production. It also leads to inflammation and reduces phagocytic activity of phagocytes [16].

Wound healing

The effects of smoking on healing of wounds have been suspected for a long time now. Smoking causes arteriolar vasoconstriction and reduces blood flow [17,18]. Catecholamine release causes vasoconstriction and decreased tissue perfusion, increases platelet adhesiveness and blood viscosity, which increases likelihood of increased fibrinogen, micro vascular occlusion, carboxyhaemoglobin and altered polymorphonuclear leukocyte function [19,20].

Smoking slows the healing process following a variety of mucogingival procedures [21-24]. A variety of harmful by products released after smoking are considered as risk factors for impaired healing after the procedures [25].

Relation of smoking with dental implant

A variety of elements must come together for an implant to be successful, from successful surgery to high quality prosthesis and routine maintenance.

As previously noted, smoking is substantial risk factor for failure of implant. Bain and Moy were first one to recognize smoking as an important factor in causing failure, reporting higher failure rates in smokers *i.e.* 11.28% than non-smokers *i.e.* 4.76% with 44 out of 390 implants failing in smokers and only 86 out of 1804 implants failing in non-smokers [26].

Increased tobacco usage leads to higher rate of implant failure, according to certain research. In study on mandibular implant over dentures, Fartash concluded that smokers who smoked 30-40 cigarettes per day have a greater rate of implant failure [27]. Lindquist also discovered that smokers who smoked more than fourteen cigarettes per day had more marginal bone loss

around implants than individuals who smoked less number [28].

Smoking has little effect on the osseointegration process; rather, its harmful consequences appear after the 2nd stage surgery. In a study which involved about 2000 patients, Gorman, et al. [29] discovered that smokers had considerably greater failures after second-stage surgery. Lambert, et al. [30] discovered a trend of higher failures in smokers between the time of uncovering and the time of insertion. Because implant wounds were closed, the intimate adaptation of implant to bone tissue did not allow the same magnitude of interruption in healing by vasoconstrictive action of nicotine, the author postulated that the effect of tobacco on healing after implant placement was different from that occurs after extraction of tooth. However, once the implants were exposed, tobacco had a deleterious effect on the soft tissues surrounding them, similar to how periodontal tissues were harmed. According to him smokers had 1.5 times more chances of implant failures than non-smokers after three year period of placement.

Smokers show higher rate of marginal bone loss which is seen more in maxilla than in mandible [17,30]. According to Lambert, et al. upper implants failed 1.6 times more frequently than lower ones in smokers. de Bruyn, Collaert and Esposito, et al. also proved the predilection for maxilla from their studies [18,31]. The reason behind maxillary bone being most commonly affected is most likely to be the inferior quality of bone, making it more prone to the harmful effects of smoking.

When anterior and posterior mandibles are compared in smokers, posterior mandible shows less failure compared to anterior. This may be because the posterior region is protected by the tongue against the harmful influence of tobacco smoke [12]. Queiroz, et al. [32] conducted a study in which he collected salivary samples from 41 people and used l-ornithine measurement to assess levels of salivary arginase activity. He found increased activity in smokers with dental implant. He hypothesised that higher arginase activity would result in reduced nitric oxide production, putting smokers at risk of bacterial infection and failure of implant.

Tobacco has also been found to have a deleterious impact on the healing of dental implants in several animal studies [33].

Correa, et al. investigated effect of smoking on Osseo integration in rabbit tibias, exposed to intermittent cigarette smoke inhalation. After sixty days of implant insertion, smoking had harmful effect on density and amount of bone around the implant. According to the study, presence of cigarette particles, heat and high concentration of hazardous components may cause these effects to be more prominent in oral cavities of smokers [26,34-37].

Stefani, et al. [38] studied effects of nicotine in rabbits on peri-implant bone healing, finding that regular treatment of 0.93 mg/kg of nicotine dramatically reduced proportion of bone implant contact after 42 days of placement of implant.

Cesar-Neto et al. [39] investigated effects of nicotine administration and inhalation on osseointegration of implant. Over the course of sixty days, animals were exposed to smoke inhalation and nicotine was administered subcutaneously. Inhaling cigarette smoke had a deleterious impact on bone implant contact and the amount of bone in implant screws. Nicotine administration, on the other hand, had no effect on either of them.

DISCUSSION

Smoking leads to harmful effects on the body right from oral cavity to the general health of an individual. It affects all therapeutic procedures which are done in that individual.

Nicotine remains the major component of smoke leading to such effects. Nicotine, at concentrations greater than 0.2 mg/mL, can cause vacuolation of cells on the surface of dental implants. It also gets accumulated in lysosomes as a result of cell membrane permeabilization. Water penetrates the cell *via* osmotic response when the gradient concentration into the lysosomes rises, causing these structures to enlarge and form vacuoles. The Osseo integration process could be harmed by the mechanism.

Talking about the effects of tobacco smoke on dental implant, various studies which were carried out showed that it has deleterious effects on dental implant. The major impact which smoking has is the failure of implant mainly after the second stage surgery.

Implant location plays a major role in determining the failure. Maxillary implants tend to result in more failures as compared to mandibular. This has been hypothesised on the basis of considering maxillary bone as a weak bone than mandibular.

Based on the suspected effects of smoking on implant Osseo integration, quitting smoking would be the most crucial goal to achieve in order to avoid such detrimental consequences. According to certain research, smoking cessation (both temporary and permanent) can counteract the harmful effects of smoking on bone repair surrounding dental implants [34,39,40].

According to Bain and Moy [41] patient should stop smoking one week before surgery to allow the reversal of enhanced platelet adhesion and blood viscosity also the effects of nicotine. The patient should refrain from smoking for minimum two months. After placing the implant by which time bone healing will have advanced to the osteoblastic phase and early Osseo integration will have occurred.

CONCLUSION

Before planning for implant surgery in the patient his/her smoking history should be adequately obtained. If positive history is obtained they should be motivated to stop the habit and they should be educated about all the

possible effects on their overall health as well as on their oral health and should be told about the doubtful success of the implant which is going to be inserted.

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