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CASE REPORT

Impact of Smoking on Oral Mucosa

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Abstract

Introduction: The discoloration of the oral mucosa is influenced by the presence and degree of dilation of the subepithelial corium blood vessels and the amount of melanin pigment. Changes in the oral mucosa of smokers encompass various significant pathological conditions, including smoker's melanosis and hyposalivation. **Case Report:** A 27-year-old male presented to the dental hospital with complaints of black discoloration on the lower jaw gingiva, persisting for the past two years. He exhibited widespread black spots on the upper and lower jaws without pain. The patient also reported dry lips. He had a smoking history of seven years, with a consumption of 10 cigarettes per day. Examination revealed pigmented lesions, and the patient's stimulated salivary flow rate was 0.7 ml/min. **Discussion:** The oral cavity is a primary gateway for toxic substances from cigarettes, while saliva is the main biological fluid exposed to cigarette smoke, which contains various toxic compounds responsible for structural and functional changes in saliva. Exposure to cigarette smoke can lead to reduced salivary flow rate (hyposalivation), changes in salivary components that may result in malignancies, and changes in the color characteristics of the oral mucosa, such as the appearance of pigmented lesions known as smoker's melanosis. Management of hyposalivation in patients includes providing education to improve hydration and encouraging smoking cessation. **Conclusion:** Smoking affects the condition of an individual's oral cavity, impacting both saliva and the oral mucosa. It is crucial for dentists to educate patients about the use of tobacco and its effects on oral health.

Keywords: Hyposalivation, oral mucosa, smoking, smoker's melanosis

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INTRODUCTION - PENDAHULUAN

The oral mucosa acts as a protective barrier by secreting essential substances and facilitating sensory perception. Its functions include protecting the underlying tissues from various stimuli and activating sensory responses such as temperature, touch, pain, and taste perception.[1] Oral pigmentation, characterized by changes in the color of the mucosa and gingiva, can arise from various sources, such as exogenous factors like medications and smoking, endogenous conditions like diseases, or genetic predispositions.[2] The prevalence of oral pigmentation varies among populations, with higher rates observed in individuals with darker skin. Anterior gingival pigmentation often affects aesthetics when smiling. Additionally, gingival pigmentation can serve as a clinical indicator of systemic diseases or medication side effects. In smoker's, oral melanin pigmentation frequently occurs due to melanin's protective action against oxidative stress caused by cigarette smoke.[3] Changes in the oral mucosa of smoker's encompass significant pathological conditions, including smoker's melanosis and hyposalivation. A smoker's melanosis is a hyperpigmentation of the mucosa often found in smokers, particularly in the gingival area, caused by melanin accumulation in response to chronic irritation from cigarette smoke. Additionally, hyposalivation, or reduced saliva production, is commonly observed in smokers and can lead to complications such as xerostomia, increased infection risk, and dental caries.[1,3]

Mechanical, chemical, and thermal stimulation of the salivary glands by smoking can temporarily increase saliva production. In the short term, some studies show an increase in saliva flow due to stimulation; however, the long-term effects of tobacco use on salivary flow rate remain unclear. Long-term smoking is an external factor that reduces saliva production, leading to hyposalivation. Hyposalivation is caused by various factors, such as salivary gland diseases, systemic diseases, chemotherapy, radiation, or side effects of various medications. Conventional smokers often experience subjective feelings of xerostomia and halitosis.[3,4]

This case report outlines how to identify and understand oral mucosa changes, such as smoker's melanosis and hyposalivation, in smokers.

CASE REPORT - STUDI KASUS

A 27-year-old male presented to the dental hospital with complaints of black discoloration on the lower jaw gingiva, which had been present for the past two years. He also had black spots on both the upper and lower jaws without any pain. The patient reported dry lips. He had a smoking history of seven years, consuming ten cigarettes per day. There was no family history of similar complaints. The patient had no history of systemic diseases and was not taking any medications. He rarely consumed vegetables but frequently ate fruits. His daily water intake was less than 2 liters, and he brushed his teeth twice daily.

Extraoral examination revealed dry lips with desquamation and pigmentation on both the upper and lower lips (Figure 1.A). Intraoral examination showed diffuse brownish-black macular lesions on the gingiva of both jaws (Figure 1.B). Fissured lesions were observed on the dorsum of the tongue, and yellowish-white plaques that could be wiped off were noted on the posterior two-thirds of the tongue (Figure 1.C). A stimulated salivary flow rate test was performed in response to the dry mouth complaint, yielding a result of 0.7 ml/5 minutes, indicating hyposalivation.

Based on anamnesis, clinical examination, and supplementary tests, a diagnosis of oral manifestations of long-term tobacco use was established, which included smoker's melanosis on the lips and gingival mucosa, coated tongue, and mild hyposalivation. The differential diagnosis for smoker's melanosis was physiological pigmentation. The patient was informed about the findings, specifically the black discoloration on his gingiva and lips, which was attributed to

smoking. The pigmentation was not malignant. The patient was educated on maintaining oral hygiene by brushing his teeth and tongue twice daily. He was also advised to consume 2 liters of water daily, get adequate rest, and quit smoking.

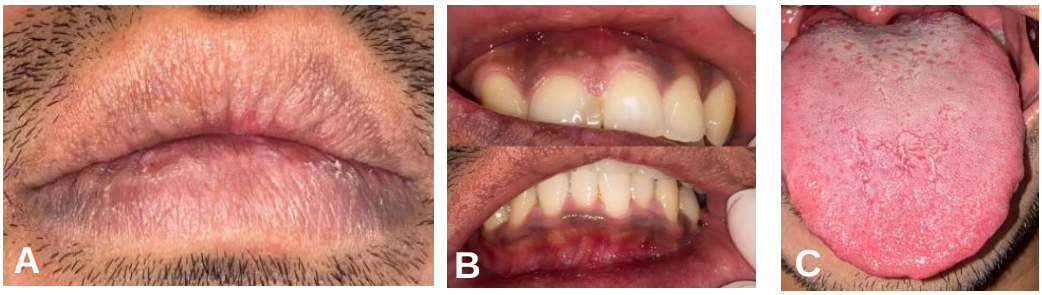

Figure 1A. Dry lips with desquamation, B. Generalized pigmentation of the gingiva, C. There is a yellowish-white plaque on the tongue.

DISCUSSION

The discussion section delves deeper into the specific mechanisms and clinical implications of smoking on oral health. Smoking introduces various chemicals into the body, with significant detrimental effects on oral health. A single cigarette can release up to 4,000 chemicals, including nicotine and tar, which impact the function of salivary glands and the health of the oral mucosa.[5] These substances are divided into gaseous components (92%) and solid particles (8%). The main gaseous components include carbon monoxide, carbon dioxide, hydrogen cyanide, ammonia, nitrogen oxides, and hydrocarbons. Solid particles contain tar, nicotine, benzanthracene, benzopyrene, phenol, cadmium, indole, carbazole, and cresol, all of which are toxic, irritative, and carcinogenic.[5] Nicotine induces vasoconstriction, reducing blood flow to the salivary glands, which decreases saliva production.[6] Tar and other chemicals in cigarette smoke stimulate melanin production in mucosal cells, leading to smoker's melanosis. Several studies indicate that cigarette smoke can trigger pigmentation of the labial and anterior gingival regions. Other studies have shown that gingival pigmentation can also occur in passive smokers.

The long-term effects of smoking include a reduction in the salivary flow rate and pH, which increases the risk of dry mouth, dental caries, gingivitis, tooth mobility, calculus, and halitosis. Additional oral cavity disorders induced by smoking include periodontal disease, smoker's melanosis, leukoplakia, nicotine stomatitis, smokeless tobacco keratosis, submucosal fibrosis, hairy tongue, and oral cancer.[6,7] Nicotine, when absorbed into the bloodstream through the lungs and oral mucosa, has widespread effects on the endocrine, musculoskeletal, and nervous systems. It increases blood flow, heart rate, nausea, and xerostomia. Nicotine that is absorbed is distributed to glands such as the adrenal glands and salivary glands. Prolonged exposure to nicotine in the salivary glands leads to pathological processes, especially in acinar cells and myoepithelial cells.[6] Nicotine exposure causes swelling in acinar cells and an increase in the number of intra-acinar secretory granules. Most of these granules are immature and contain low concentrations of glycoproteins, while mature dark granules containing high concentrations of glycoproteins drastically decrease. Chronic nicotine exposure leads to exhaustion in the acinar cells of the salivary glands, resulting in reduced saliva production.[6] Additionally, chronic smoking causes morphological changes in myoepithelial cells, disrupting the function of the salivary glands. Myoepithelial cells are responsible for saliva secretion, stabilizing gland structure, and producing proteins that suppress tumors.[6]

Smoking also affects the levels of cortisol and Immunoglobulin A (IgA) in saliva. It has been proven that smoking can lower total protein, calcium (Ca), and lead (Pb) levels in saliva. Furthermore, smoking significantly reduces salivary flow rate and increases dry mouth-related

oral and dental disorders. Tobacco smoking is considered a risk factor for oral cancer. Alcohol synergizes with tobacco as a risk factor for all upper aerodigestive tract squamous cell carcinomas.[3] Saliva serves as the first biological medium to encounter external substances, protecting the mucosa of the upper digestive tract. Various agents exert carcinogenic effects through changes in saliva's chemical composition. Saliva possesses a higher total antioxidant capacity compared to plasma. It also contains polypeptides, including immunoglobulins and enzymes such as lactoferrin, lysozyme, and histamine. These polypeptides play a critical role in defense mechanisms against free radicals (oxidative stress), thereby preventing oral cancer.[3,4]

The patient's tongue showed white plaques identified as coated tongue. The etiology of coated tongue remains unclear, but predisposing factors include oral lesions, poor oral hygiene, dehydration, medication use, and a soft diet. Poor habits such as smoking and alcohol consumption, gastrointestinal disturbances, systemic illness, fever, and severe illness (total bed rest) can lead to hyposalivation, which in turn causes xerostomia (dry mouth), reducing tongue movement and facilitating coated tongue formation.[8,9] Under normal conditions, the tongue is covered by a mucus layer, desquamated epithelial cells, and food debris. A healthy tongue is in constant motion, and saliva flows normally, resulting in a thin white layer. When tongue movement is reduced, saliva flow is low, and fever is present, a thick white layer can form on the tongue. Keratin formed on the tongue is desquamated and ingested during food consumption. Typically, the amount of keratin produced matches the amount desquamated, maintaining a normal appearance. Imbalance in this process leads to a coated tongue, possibly due to slow desquamation or consumption of softer, less abrasive foods.[9] Coated tongue can also form when keratin production exceeds desquamation and ingestion rates. Increased keratin production is often due to excessive tongue irritation from hot beverages or tobacco smoking. Keratin accumulation on the filiform papillae (taste buds) of the tongue gives it a whitish appearance.[9] Clinically, coated tongue appears as a layer covering the tongue, which can be white, brown, or black, depending on the pigments involved. Intrinsic factors like chromogenic organisms combined with extrinsic factors such as food, drink, tobacco, and candy colors contribute to coated tongue coloration. In severe cases, coated tongue may be accompanied by tongue pigmentation, appearing as black or brown discoloration.[9] The color of the oral mucosa is determined by the thickness of the epithelium, the degree of keratinization, melanin deposition, and the connective tissue, including blood vessels and other pigments like hemoglobin or hemosiderin. Lysis of red blood cells can cause red, blue, or brown hues. Melanin synthesized by melanocytes appears brown, blue, or black, depending on the amount and depth of melanin. Physiological melanin pigmentation of the oral mucosa typically occurs in black individuals, genetically determined by the amount of melanin produced.[10]

Smoker’s melanosis is a characteristic change in the color of oral mucosa exposed to cigarette smoke, resulting from melanin deposition in the basal cell layer. Smoker’s melanosis is a benign oral mucosal disorder, but it can affect aesthetics if left untreated. It appears in 25-31% of smokers and significantly increases during the first year of smoking. Pigmentation expands with prolonged smoking, indicating a higher risk of developing smoker’s melanosis with longer smoking durations. [10] Smoker’s melanosis has a prevalence of about 31%, primarily affecting the labial mandibular gingiva. According to Hedin (1977), smoker’s melanosis is most commonly seen on the attached gingiva of the lower jaw (88%), followed by the upper jaw (85%), buccal mucosa (30%), and palate (23%) in a study of 467 participants. The pigmentation is reversible, typically disappearing years after smoking cessation.[10] The cause of smoker’s melanosis is the physical effect of tobacco on oral tissues from heat and direct nicotine stimulation, which induces melanocytes in the basal epithelial cells to produce excessive melanin, leading to increased melanin deposition. Microscopically, clusters of brown granules

(melanin) are seen in the basal layer and lamina propria of the mucosal epithelium.[10] Pigmentation occurs due to stimulant substances in cigarette smoke reaching gingival melanocytes through mucosa and saliva or systemic circulation. In the first route, nicotine and benzopyrene reach gingival melanocytes via mucosa and saliva. Thermal of cigarette smoke, containing nicotine and benzopyrene, stimulates melanocytes to produce more melanosomes, increasing melanin pigment in the lamina propria and depositing in the basal epithelial cells. In the systemic route, most smoke enters the bloodstream as smokers breathe through their noses allowing nicotine and benzopyrene to circulate and indirectly affect melanocytes. Smoker's melanosis does not pose a health risk, with no signs of malignancy or cell degeneration microscopically, thus classified as benign mucosal melanosis. The condition resolves upon smoking cessation.[10]

The differential diagnosis of smoker's melanosis includes physiologic pigmentation. The table below compares the differential diagnoses in this case:

Table 1. The differential diagnosis of smoker's melanosis

	Smoker's Melanosis	Physiologic pigmentation
Etiology	Use of tobacco products stimulating melanocyte production in tissues.	Occurs during pregnancy due to increased hormone levels.
Location:	Attached gingiva, lips, or palate.	Occurs on all oral mucosa, including the tips of fungiform papillae on the dorsum of the tongue.
Management:	No treatment except for aesthetic concerns; gingival depigmentation may be performed.	No treatment except for aesthetic concerns; gingival depigmentation may be performed.

Management of smoker's melanosis involves educating patients about the condition and encouraging smoking cessation. Patients should be informed that the pigmentation is benign and reversible upon smoking cessation. Aesthetic concerns can be addressed through gingival depigmentation if necessary. Maintaining proper oral hygiene and adequate hydration is crucial in managing the condition. Dentists should provide patients with comprehensive education on the effects of smoking on oral health and emphasize the importance of quitting smoking to prevent further oral health issues.

CONCLUSION

Smoking significantly affects the oral cavity, impacting both saliva and oral mucosa. Long-term smoking reduces salivary flow rate and alters saliva physiology, potentially leading to malignancies. This case highlighted oral mucosal changes, such as gingival and lip pigmentation (smoker's melanosis) and coated tongue. It is crucial for dentists to educate patients about smoking and its impact on oral health. Effective management includes patient education on smoking cessation, maintaining proper oral hygiene, and addressing aesthetic concerns if necessary.

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