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Effect of smoking on oral-mucosa - A Review

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Abstract

Introduction: Melanosis is a discolouration of the mucosa. Colour changes in the oral mucosa can occur due to many factors, one of which is the excessive consumption of cigarettes. In 2014, 22% of smokers had melanosis of the oral mucosa. In addition, white people more often experience melanosis in the oral cavity. This habit may broadly be classified as smoked tobacco and smokeless tobacco. May occur in up to one of five smokers, especially females taking birth control pills or hormone replacement than in men. Gingival pigmentation in children has been linked to passive smoking from parents and other adults who smoke. Clinic pathological study reported that the intensity of the pigmentation was more in the labial mucosa than in the buccal mucosa.

Objective: Aim of this study is to review oral melanosis in the oral cavity and its relationship with smoker.

Discussion: The gingiva is a part of the oral cavity that often experiences melanosis. The colour of the gingiva is determined by the thickness of the epithelium, the degree of keratinisation, the presence and level of melanin deposits, and the underlying connective tissue, including blood flow within the gingiva, in the presence of other pigments such as haemoglobin or oxyhemoglobin. Melanocytes are seen in the basal layer of the epithelium. The basal layer of the epithelium secretes melanin via dendrite projections to the interior of adjacent keratinocytes.

Conclusion: Melanosis of the gingiva often occurs, one of the causes that most often triggers melanosis of the gingiva, namely smoking. Therefore, it is necessary to pay more attention to cigarette consumption in the community.

Keywords: Melanosis, pigmented gingival, smoker.

Introduction

Oral melanosis is a benign focal pigmentary (brown or black) lesion of the oral cavity mucosa, though cases of melanosis have been reported on sinonasal, pharyngeal, conjunctival and laryngeal mucosa also. Oral pigmentation is caused by exogenous (foreign body implantation) and endogenous pigmentation. Primary

pigments responsible for endogenous pigmentation include melanin, melanoid, oxyhemoglobin, reduced haemoglobin and carotene. Out of these pigments, melanin is the most common endogenous pigment which is a non-haemoglobin derived brown pigment and is produced by melanocytes present in the basal layer of the epithelium [1, 2] .

Fig 1: Factors of Melanosis in Oral Cavity [2].

Melanocytes were first identified in the oral epithelium by Becker in 1927 and a few years later they were isolated from samples of gingival tissue by Laidlaw and Cahn. During early intrauterine life, melanoblasts (precursor of melanocyte) migrate from the neural crest to the epidermis and hair follicles. Later they differentiate into dendritic cells and appear in head and neck region after approximately 10 weeks of gestation. Melanocytes dendrites reach a number of keratinocytes in the close vicinity, and through these dendrites, melanin is transported and transmitted to these epithelial cells, process called melanogenesis [3] . Melanosis is a discolouration of the mucosa, colour changes in the oral mucosa can occur due to many factors, one of which is the excessive consumption of cigarettes. According to Tarakji et al in 2014, 22% of smokers had melanosis of the oral mucosa. In addition, white people experience melanosis more often in the oral cavity. Melanosis in the oral cavity is often a problem because it interferes with aesthetics and a smile. Discolouration of the mucosa is often found in the upper and lower labial gingiva. Gingiva is an important part of the oral cavity, gingiva has a big role in the chewing process. Often, discolouration of the gingiva occurs in the anterior part, so it is very disturbing to esthetics. Most iatrogenic pigmented lesions in the oral cavity are benign and their pigmentation is due to excessive production of melanin, which is produced by melanocytes. These cells are specialised dendritic cells present in the basal cell layer of the mucous membrane. The clinical visible pigmentation in the oral cavity depends on the number of melanocytes or the degree of melanin produced by these cells. The range of colour pigmentation varies from gray to brown to black to dark blue. The closer the pigmentation to the surface, the darker the colour (black); a melanin deposit before the basal cell layer will cause blue colour. Smoker's melanosis is due to long-term tobacco smoking. The pigmentation is usually distributed along the gingival layer in the upper and lower anterior teeth. It may also be seen in the soft palate, buccal mucosa, and floor of the mouth. Smoking cessation is the treatment of choice. The hyperpigmentation then disappears in a few months [4] . Tobacco habits are practiced in different forms, and many of these habits are specific to certain areas of India. This habit may broadly be classified as smoked tobacco and smokeless tobacco. May occur in up to one of five smokers, especially females taking birth control pills or hormone replacement than in men. Gingival pigmentation in children has been linked to passive smoking from parents and other adults who smoke. Clinic pathological study reported that the intensity of the pigmentation was more in the labial mucosa than in

the buccal mucosa. Also, they mentioned that the pigmentation was absent in smokeless tobacco users but mild pigmentation was observed away from the site of quid placement with the concurrent increase in number of melanocytes and melanocytes activity. Aim of this study is to review oral melanosis in the oral cavity and its relationship with smoker [5] .

Discussion

Ethiology Melanocytes nevi are a diverse group of benign tumours that arise as a consequence of melanocytes growth and proliferation. These lesions are uncommonly identified within the mucosa; the skin is much more commonly affected. The list of morphologically distinct nevi continues to expand. Among these, the intramucosal nevus is most frequently observed within the oral cavity; the blue nevus is the second most common. Less frequently observed are compound nevi and junctional nevi. Rare reports of oral melanocytes nevi exhibiting unique histologic patterns have also been described. Nonetheless, irrespective of the exact nevus subtype, they almost all present with similar clinical features [6, 7] . Oral nevi are usually identified in patients over the age of thirty. The lesion is typically asymptomatic and may present as a small, solitary, brown or blue, well-circumscribed nodule or macule. Some nevi may not exhibit any evidence of clinical pigmentation. Any mucosal site may be affected, but the hard palate, buccal and labial mucosal and gingival are most typical. Heavy smokers tend to experience melanosis of the gingiva. In cigarettes there are polycyclic amine compounds that can cause melanocyte activation to produce melanin. These pigmented manifestations are considered normal and generally no treatment is recommended except for aesthetic purposes. Quitting tobacco is sufficient in minimising the occurrence of melanosis in the gingival. Several treatments can be done to remove discolouration in the gingival such as ablation, namely by reducing the colour of the gingival which has changed colour to blackish brown or by performing cryosurgery. These treatments can be done if they feel disturbing or uncomfortable [8, 9, 10].

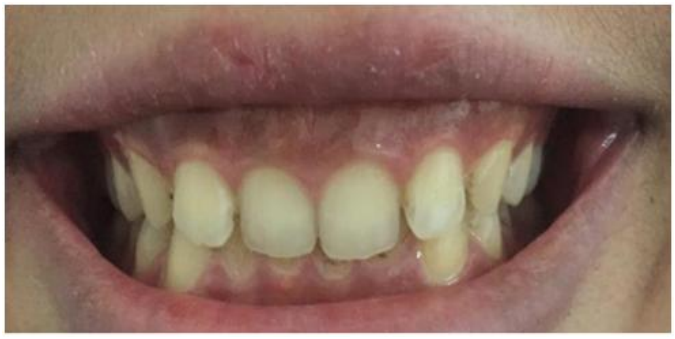

Figure 2: Melanocytes nevi

The gingiva is a part of the oral cavity that often experiences melanosis. The colour of the gingiva is determined by the thickness of the epithelium, the degree of keratinisation, the presence and level of melanin deposits, and the underlying connective tissue, including blood flow within the gingiva, in the presence of other pigments such as haemoglobin or oxyhemoglobin. Melanocytes are seen in the basal layer of the epithelium. The basal layer of the epithelium secretes melanin via dendrite projections to the interior of adjacent keratinocytes. Melanin is a granular endogenous non-haemoglobin pigment that gives a brown or black (eumelanin) colour to the skin, mucosa, hair, and eyes or sometimes a reddish colour (pheomelanin). In addition to tissue staining, the main function of this pigment is photoprotection, protecting DNA from UV rays [11, 12, 13] .

Theory says that at 25-31% smokers seen smoker's melanosis 19. However on This study also found several respondents in the absence of smoker's melanosis pigmentation, by 6.25%. No pigmentation this is not only affected by the length of smoking and the number

of cigarettes consumed in a day course, but also the environment around the smoker [14, 15, 16] .

Factors Affecting Oral Melanosis in Smoker

Active smokers can act as smoker passive. When someone becomes secondhand smoke, there is a mechanism for the formation of pigmentation through the second way according to Hajifattahi. On the respondent in the absence of pigmentation, they are likely do not become a passive smoker so the mechanism The formation of smoker's melanosis is not occur. Clinical picture of smoker's melanosis with smoking habits, namely the duration of smoking and the amount cigarettes consumed in one day are served most respondents a lot of pigmentation is widespread it turns out has a smoking habit of more than 20 years as much as 46.3%. The results of this study are in line with research conducted before, group the longest smoking duration is more than 14 years shows the highest percentage the presence of smoker's melanosis was 67.5%. This situation is also in accordance with the theory presented that the pigmentation intensity smoker's melanosis associated with the length of smoking someone [17, 18, 19, 20] .

Smoker's melanosis is influenced by the length of smoking each individual. The longer a person smokes the more likely he is to suffer. Smoker's melanosis. Pigmented lesions increase obvious during someone's first year of smoking and the location of the pigmentation increases if someone smokes the longer it takes. Smoker's melanosis is related to deposition of nicotine in the body.

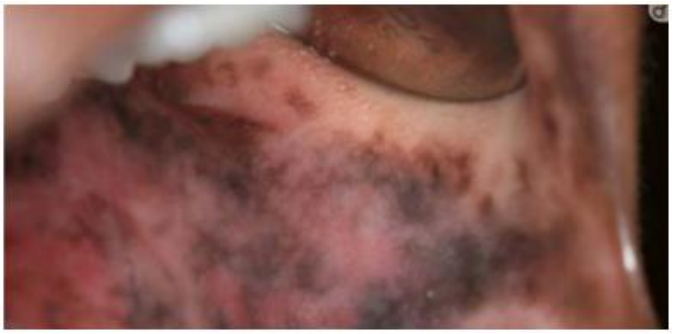

Figure 3: Melanin Pigmentation

Increased melanin pigment deposits in basal layer of the oral mucosal epithelium. Stimulation nicotine that's persistent and long lasting affect immediate onset of smoker's melanosis [21, 22, 23, 24] .

Malignancy in Oral Melanosis

In rare cases, oral nevi may be congenital or a manifestation of an unusual genetic disorder. More commonly, melanocytic nevi are acquired lesions, with genetic and environmental factors likely playing a role in their development. While cutaneous melanocytic nevi frequently exhibit somatic, activating mutations in the BRAF or NRAS oncogenes, it remains unclear if similar mutations are also implicated in the pathogenesis of oral mucosal nevi [25, 26, 27] .

Nevus cells are morphologically and biologically distinct from the melanocytes that are found within the basal layer of the oral epithelium. Nevus cells tend to be round, ovoid or spindled shaped while the basal layer melanocytes are dendritic in appearance. Intramucosal, compound and junctional nevi likely share a common pathogenesis. It is theorised that over time, the junctional nevus evolves into a compound nevus and eventually into an intramucosal nevus. The histologic difference between these three variants relates to the location of the nevus cells within the tissues [28, 29] . Since the differential diagnosis of focally pigmented lesions includes malignant melanoma, biopsy is required for accurate diagnosis of an oral melanocytic nevus. While

malignant transformation of cutaneous blue nevi has been reported, this phenomenon has not been reliably documented for oral blue nevi. Nonetheless, conservative surgical excision is the treatment of choice for these oral lesions [30] .

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