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Periodontitis and Leukoplakia: Is There an Interconnection?

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Abstract

Background: Leukoplakia is an asymptomatic, potentially malignant lesion in the oral mucosa which often develops into oral cancer. Various follow-up studies done show that between <1 and 18% of oral pre-malignant lesions will develop into oral cancer. Periodontitis is related to different pathological states in the oral cavity including pre-malignant and malignant lesions. Frequently, periodontal sites are involved in proliferative types of leukoplakia. Therefore, Periodontitis increases the risk of oral leukoplakia and, therefore, the risk of mucosal lesions predisposing to oral cancers.

Methods: A 39 year old male patient reported to the Department Of Periodontology with chief complaint of bleeding of gums and whitish patch on his right and left buccal mucosa since 6 months. A Complete clinical examination and detailed case history was recorded for the same. Proper treatment plan was planned and implemented. This cased showed a strong corelation between periodontitis and oral leukoplakia.

Conclusion: Poor oral hygiene and the ensuing of plaque accumulation results in a chronic inflammatory process, creating an environment that promotes the development premalignant lesions and oral carcinoma. Our study supports the hypothesis that periodontitis is an individual risk factor for Oral premalignant lesions. Periodontal disease is a chronic inflammatory condition that may be prevented with regular dental visits and with the maintenance of the Oral hygiene. Dentists play an important role in preventing oral cancer by evaluating the socioeconomic status of each patient and appraising their lifestyle and habits. Therefore this case report mainly focuses on interrelationship between maintenance of good periodontal health and development of oral premalignant lesions.

Keywords: Premalignant lesion, Risk factors, Leukoplakia, Periodontitis.

Introduction

One of the most common oral potentially malignant disorders (PMDs) affecting the oral cavity is oral leukoplakia (OL). In the first international conference on OL (1984) in Malmo, Sweden, OL was defined as "a white patch or plaque that cannot be characterized clinically or pathologically as any other disease and is not associated with any physical or

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chemical causative agent except use of tobacco." In the year 1997, the WHO defined leukoplakia as "a predominantly white lesion of the oral mucosa that cannot be characterized as any other definable lesion. van der Waal in 2007[1] suggested a new definition that includes histological confirmation, but this has not been yet assessed by the WHO, "A predominantly white lesion or plaque of questionable behaviour having excluded, clinically and histopathologically, any other definable white disease or disorder.

Periodontitis is related to different pathological states in the oral cavity including pre-malignant and malignant lesions. This is also true for smoking, the most important risk factor for oral leukoplakia and for periodontitis.[2] Frequently, periodontal sites are involved in proliferative type. Various reports suggested that an association may exist between periodontitis and premalignant lesions.[3] Data from the NHANES study suggest associations between periodontal disease and the risk of precancerous lesions.[4]

The association between periodontal disease and oral neoplasm is biologically plausible and may be explained by:

1. Broken mucosal barrier in the presence of periodontal disease and consequent enhanced penetration of carcinogens such as tobacco and alcohol.
2. Increased cellularity in blood vessels and connective tissue in chronic inflammation. Association between chronic inflammation and cancer is coupled with the development of chronic diffuse epithelial hyperplasia which is regarded as a common precursor to intraepithelial neoplasia.
3. Immunosupression as a common mechanism leading both to periodontal disease and oral cancer. For example, major concentrations of defensins (which have antibacterial, antiviral, and antitumor activities and are likely to play an important role in killing periodontal pathogens) found in neutrophils and epithelia suggest potential implications for critical immune surveillance within periodontal attachment.
4. Viruses such as human papilloma virus (HPV) and herpes simplex virus 1 (HSV 1) or Candida albicans that are found both in oral cancer and periodontal disease.
5. Bacterial overgrowth in patients with poor oral hygiene may lead to an increased rate of metabolites with possible carcinogenic potential. For example, higher microbial production of carcinogenic acetaldehyde from ethanol has been shown in patients with poor oral Hygiene.

Periodontitis is an inflammatory response to oral microbiotome and is associated with bone loss and tooth loss.[5] Exposure to inflammatory conditions promotes the infiltration of immune cells to the oral mucosa. Inflammation and immune related mediators have been accepted as the hallmarks of malignant transformation

Case Report

A 39 year old male patient reported to the Department Of Periodontology with chief complaint of bleeding of gums and whitish patch on his right and left buccal mucosa since 6 months. A Complete clinical examination and detailed case history was recorded for the same . On eliciting personal history, the patient has a habit of smoking since the last 12 years, 3 times a day. Patient had no significant medical history and was not under any medication.

On clinical examination, no abnormalities were detected extraorally. Inspection of the lesion intraorally revealed a uniform whitish patch on the right and left buccal mucosa at the line of occlusion, measuring approximately 3 cm × 2 cm at its greatest diameter [Figure 1&2].

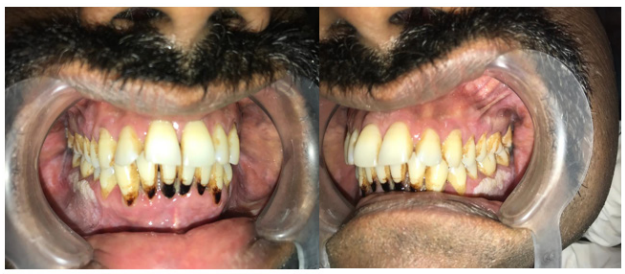

Figure 1 & 2: White Patch Seen on Right and Left Buccal Mucosa

Cracked mud appearance was seen with the overlying mucosa whitish in colour. Colour was non scrapable, non -stretchable and non -palpable.

VAS score for burning was 0. The examination of oral mucosa for lesions – oral leukoplakia status was done and covered all areas of the oral cavity including the tongue and lips.

Periodontal status was assessed. The examination was conducted under sterile clinical conditions keeping all safety protocols in mind. Assessment included pocket probing depth, clinical attachment loss (CAL), Plaque index, bleeding on probing (BOP).

All fully erupted teeth were assessed excluding third molars. Attachment loss and probing depth were assessed with a periodontal probe.

Colour of the gingiva was pale pink everwhere and bleeding was present even on slightest provocation. The lesions were nonscrapable and nontender. It was raised 0.5 mm over the surface. No bleeding from the site was noticed.

Based on the history and clinical examination, a provisional diagnosis of bilateral homogeneous leukoplakia was considered.

Excisional biopsy of the lesions was performed and the specimen was submitted for histopathological examination which revealed Oral Leukoplakia.

A final diagnosis of Oral Leukoplakia was confirmed based on the history, clinical examination, and histopathological report. Elevation of salivary interleukin-6 (IL-6) concentrations in leukoplakia cases with coexisting periodontitis and the additional effect of smoking supports the role of periodontal inflammation.

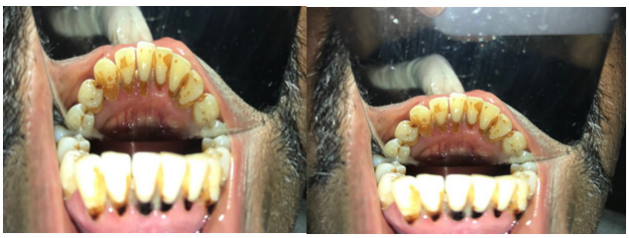

Figure 3 & 4: Showing Labio-Lingual Recession Seeen In Mandibular Anteriors

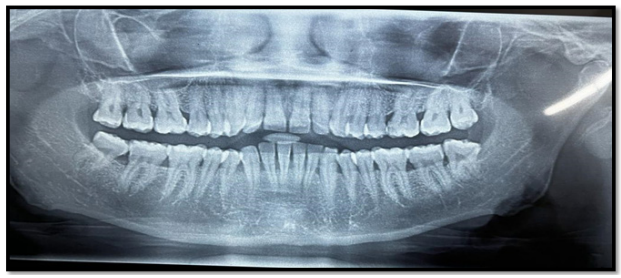

Figure 5: Orthopantomogram of the Patient

Periodontal Status: Orthopantomogram was advised for the patient which revealed the following–

1. Recession was present w.r.t 31,32.33,34, 41,42,43,44,45 both on labial and lingual aspect.
2. Periodontal pocket were present with vertical or angular bone loss seen with maxillary and mandibular posterior teeth with average pocket probing depth of 5-7mm.
3. Miller's grade 1 mobility was seen with 31,32,41,42
4. Average clinical attachment level was 3-4mm.
5. Plaque Index(Silness and Loe)was calculated which came out to be poor .

Treatment Plan

Patient was advised habit cessation and counselling of the patient was done making him aware of his current oral health and its possible consequences if he didn't stop his habit of tobacoo chewing .Velscan examination was done.Vital staining of the lesion was also performed. Cap Aquasol A twice a day for 15 days was prescribed.Tab A to Z OD was given was advised.After treatment of the lesion periodontal treatment was given followed by scaling and root planning. Patient was recalled after 15 days to check for all the clinical parameters which include pocket probing depth, clinical attachment loss ,bleeding on probing ,mobility.Regenerative procedures will be done after taking patient informed consent followed by treatment of recession and mobility .Patient will be recalled after ever 7 days and will be kept on the maintenance phase.

Results and Discussion

In the present case report, we found evidence that gingivitis and periodontitis (as characterized by Bleeding on probing, pocket depth ,bone loss ,clinical attachment loss) is associated with the occurrence of leukoplakia in a dose-dependent manner. This relationship was independent of smoking despite the fact that smoking contributes significantly to the odds of having leukoplakia .

Possible pathogenetic mechanisms for the associations shown are completely unknown and therefore speculative. A possible explanation could be the accumulation of carcinogenic metabolites produced by periodontopathogenic bacteria. From the results it may be concluded that there is a continuously increasing risk of leukoplakia with increasing severity of periodontitis or gingivitis. Increased concentrations of inflammatory markers suggest that tissues irritated by defence processes such as periodontitis are vulnerable to premalignant transformations. Thus, studies reported associations between chronic infections or inflammation and cancer. Such associations were also reported with periodontitis. Elevation of salivary interleukin-6 (IL-6) concentrations in leukoplakia cases with coexisting periodontitis and the additional effect of smoking supports the role of periodontal inflammation and relationship to tobacco use.

Ever since ***Seymour*** stated in 2010 that poor oral hygiene may affect the risk of OC, a link between PD and the development of OC has been suspected[6].

Periodontitis is an inflammatory process affecting the supporting structure of the teeth, including the gingiva, periodontal ligaments and the bone [7].

Elevated plasma levels of several proinflammatory cytokines, acute phase proteins and proteinases can be observed in periodontal disease [8]. The development of a malignant lesion can be associated with inflammation itself, peculiarly as it causes oxidative damage on the cell's DNA[9].

The recent studies of ***Gopinath*** **et. al.** ***and Geng*** **et. al**. on the development of Oral cancer suggests that there is a direct link between periodontal pathogens and the carcinogenesis.[10] In Clinical Attachment level and Pocket probing depth were used to evaluate the existence/lack of periodontitis and its severity (periodontal stage), ensuring a more reliable result. There is a clear positive link between the severity of periodontitis and the incidence of Oral lesions.

Moreover, according to **Colotta et.** al., periodontal lesions include some inflammatory mediators (for example, IL1-ß and TNF-α) associated with carcinogenesis [11].

Alcohol consumption, ageing, smoking and poor oral hygiene are important risk factors for periodontitis [12, 13,14,]. It should be noted that the same factors can also be associated with carcinogenesis in the oral cavity.

According to the observation of **Hashim et.** al., good oral hygiene may reduce the risk of Oral premalignant lesions and oral carcinomas[15]

Conclusion

Poor oral hygiene and the ensuing accumulation of plaque accumulation results in a chronic inflammatory process, creating an environment that promotes the development of Oral premalignant lesions. Our study supports the hypothesis that periodontitis is an individual risk factor for Oral premalignant lesions and oral cancer . The risk of the Oral Squamous Cell Carcinoma increases at more severe stages of periodontitis. Periodontal disease is a chronic inflammatory condition that may be prevented with regular dental visits and with the maintenance of the Oral hygiene. Dentists play an important role in preventing oral cancer by evaluating the socioeconomic status of each patient and appraising their lifestyle and habits. Motivating patients at risk of Oral carcinoma to maintain sufficient oral hygiene is crucial and easily achievable. Preserving the periodontal health and monitoring the individuals with lifestyle risk factors who are periodontally compromised may minimize the risk of Oral Cancer .

In conclusion, our findings give new hints into the complex inter-relationship between systemic and local diseases. Periodontal inflammation may be considered as an additional risk acting synergistically with smoking and/or metabolic.

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