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Original Research Paper

Dental Science

DENTAL AND MUCOSAL EFFECTS OF ARECA NUT CHEWING: CASE REVIEW

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ABSTRACT

South Asia has a long history of chewing Areca nut combined with betel quid. These habits are over hundreds of years old and Archaeological evidence from Thailand, Indonesia, and the Philippines suggests they have been used in tandem for at least 4000 years [1]. India is one of the largest consumers of Areca nut, and consumption of areca nut with betel leaf is very common among the below poverty line population. Dental and mucosal effects of these substances vary from mild to severe, where mild being gingival keratinization and sever being oral malignant squamous cell carcinoma. Keeping the areca nut in mouth for longer period of time is a common way of consumption, this most of the times leads to sever gingival recession and mucosal alterations.

KEYWORDS : Areca Nut, Betel Quid, Leukoplakia, Gingival keratinization, Gingival recession, oral health

INTRODUCTION

After caffeine, alcohol, and nicotine, Betel nut is the fourth most widely used psychoactive drug in the globe.In a number of chewing products such as mawa, paan, gutkha, khaini, and paan masala, Betel nut is present.Reasons for using betel nut include euphoria, tiredness, increased salivation, satiation, and even toothache relief.[2][3].

The International Cancer Research Agency regards paan and gutkha as substances that contain known human carcinogens, the primary culprit being the betel nut[5].Betel nut was categorized as a human carcinogens by the World Health Organization and the International Agency for Research on Cancer.[4]

These impacts can be split into two wide categories: those influencing the tough dental tissues, including teeth, their supporting periodontium, and the temporomandibular joint (TMJ) and the soft tissues that make up the mucosa lining the oral cavity[6].This paper seeks to examine the impacts on oral health of chewing Areca nut and betel quid.

Case Report

A 48 years old patient presented with severe gingival recession, presence of black deposits on the roots of lower anterior teeth, loss of sensations in lower ant teeth, bleeding gums, whitish discoloration of lower anterior mucosa.

Chief complaint of patient was bleeding near the deposits and generalized soreness of lower anterior region. Patient had a history of chewing areca nut combined with betel quid for over 20 years. Patient also smokes 1 pack cigarettes per week.

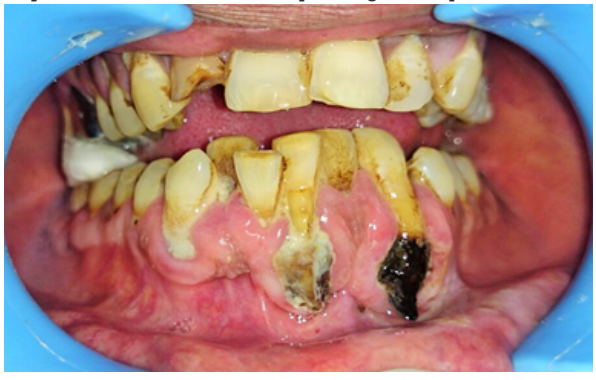

Figure 1: Patients Initial Clinical Presentation

After the initial check up it was not clear what was the black deposit on the roots of lower canine. So, investigation was done, complete blood work, x-rays and detailed history. History revealed that the initial color of the deposit was yellow and it changed to brown and then black. Also, it was found that the patient had a habit of keeping the areca nut in front of this specific tooth. Radiograph presented with bone loss in the mandibular anterior region and showed presence of calculus near both canines and central incisor (Figure 1)

After careful consideration it was found that the black deposit in front of mandibular canine root was discolored calculus due to areca nut, as the discoloration was only seen near the pouch where the patient kept the areca nut in his mouth (Figure 2)

It was also observed that there was white discoloration of the mucosa near the root of the canine, also in and around the pouch where areca nut was usually kept (figure 3)

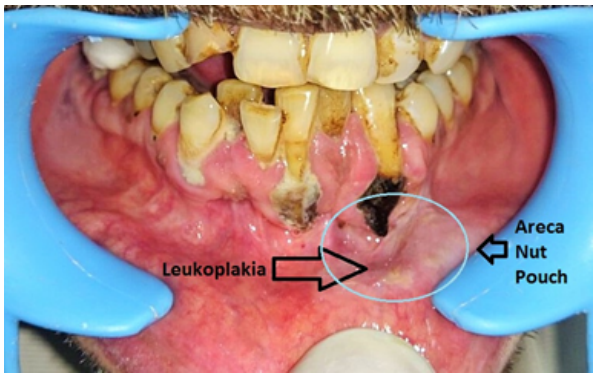

Figure 2: Areca Nut Pouch And Leukoplakia

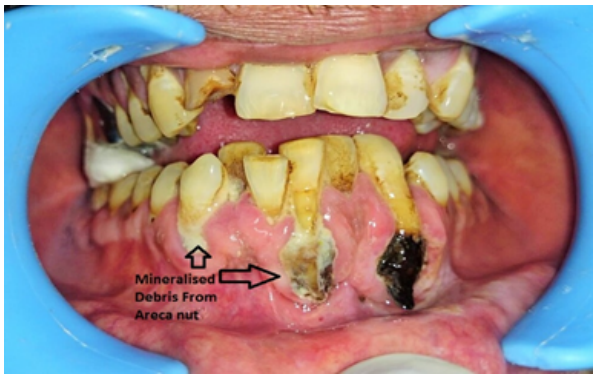

Figure 3: Calculus Deposits And Discoloration Of Calculus

Initial treatment plan was to remove the calculus and black deposits to see what is beneath them and the limit of the disease. Also, a biopsy was planned along with scaling to check the changes in the mucosa which was turned white.

After the scaling and biopsy, it was found that the roots of lateral incisor and canine was completely separated from the buccal gingival mucosa and bone but was attached from other sides to the periodontium. Chronic generalized periodontitis was the final diagnosis. (Figure 4)

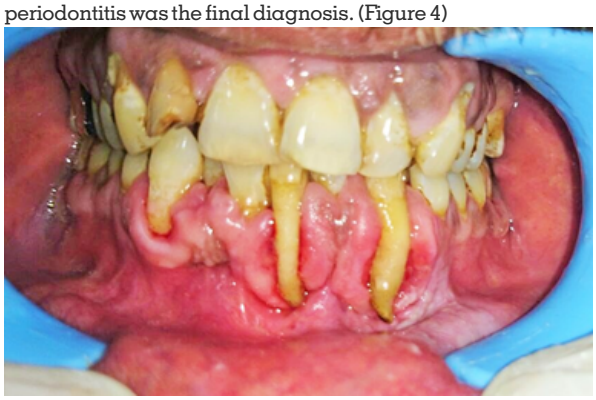

Figure 4: Post Scaling And Deposit Removal

Results of the biopsy showed Leukoplakia. Along with other treatments, the etiological factor which was areca nut in this case was prohibited and Nicotine gums were prescribed for the addiction.

After the initial phase of treatment, the next phase of treatment plan was to remove the canine and lateral incisors, gingivoplasty and prosthetic treatment of the missing space.

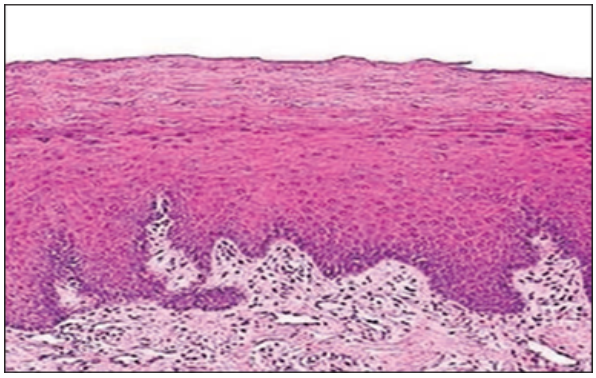

Figure 5. Histological Image Of Oral Leukoplakia [7]

DISCUSSION

Carbohydrates, fats, proteins, polyphenols, alkaloids and mineral matter are the main chemical components of the areca nut [8]. Areca nut's primary alkaloids include iscoline, iscaidine, guvaine, and guvacoline that cause fibroblast proliferation. The flavinoids are responsible for decreasing the enzyme collagenase secretion that is indirectly accountable for increasing the amount of collagen fibers.
Copper-dependent lysyl oxidase is accountable for cross connecting and stabilizing the collagen fibers that form tight fibrous bands.[9][10][11]

Typically, the oral impacts of smokeless tobacco are seen both on the mucosal surfaces where the item is positioned and on the neighboring periodontium. Clinically, the lesion from ordinary tissues is generally obviously demarcated. The site or lesion impacted may be white or yellow-brown and may develop a thickened and wrinkled appearance with enhanced tobacco product use. Tobacco-associated leukoplakia is the best clinical diagnosis to be allocated to these white lesions.[12] (Table 1)

By definition, leukoplakia is a white patch or plaque that cannot be clinically or pathologically defined as any other disease [13] Histologically, the modifications range from epithelial dysplasia to hyperparakeratosis. Studies discovered a correlation between low consumption of vitamin C and oral lesion. The incidence and severity of lesions show a dose-response relationship, best predicted by the quantity, frequency and length of smokeless tobacco use.[14]

Studies have shown that the presence of dysplastic changes cannot be predicted by habit or clinical gradation of the lesion. The most definitive technique of determining whether a lesion has developed into cancer is to biopsy the most suspect site. There may be no immediate concern for dentists about the systemic impacts of smokeless tobacco use. However, understanding of these impacts can help in counseling patients. Systemic impacts include reliance on nicotine or addiction, transient blood pressure rises, and cardiac illness.[15][16][17] Withdrawal symptoms such as drowsiness, nervousness, headache, irritability, and cravings have been reported.37 Smokeless tobacco can contain high sodium concentrations that can lead to increased blood pressure.[16]

Table 1: Distribution Of Oral Mucosal Lesions Based On Habits [18]

Oral Mucosal Lesions	Habit						Total
	Smoker		Chewer		Mixed		
	n	%	n	%	n	%	
Leukoedema	4	66.7%	-	-	2	33.3%	6
Leukoplakia	5	26.3%	5	26.3%	9	47.4%	19
Median Rhomboid Glossitis	4	66.7%	1	16.7%	1	16.7%	6
Oral Lichen planus like lesion (OLP)	-	-	5	83.3%	1	16.7%	6
Oral Squamous cell Carcinoma (OSMF)	1	7.1%	8	57.1%	5	35.7%	14
Oral Submucous Fibrosis	-	-	15	75.0%	5	25.0%	20
Pan-Chewer's lesion	-	-	12	100.0%	-	-	12
Palatal Erythema	3	42.9%	-	-	4	57.1%	7
Smoker's Melanosis	32	72.7%	-	-	12	27.3%	44
Smoker's Palate	6	100.0%	-	-	-	-	6
Tobacco Pouch Keratosis	-	-	9	90.0%	1	10.0%	10
Total	55	100%	55	100%	40	100%	150

CONCLUSION

Smokeless smoking predisposes a person to an enhanced danger of developing oral cancer. It also has a number of negative impacts on the teeth and adjacent buildings. All healthcare suppliers should evaluate the tobacco use habits of patients and actively employ programs for tobacco prevention, cessation and therapy.

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