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**Original Research Article** 

# A Study to Determine the Effect of Kharra Chewing on Periodontal Tissues of Patients with Oral Submucous Fibrosis

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#### Abstract

Aim: The aim of the present study was to determine the effect of kharra chewing on periodontal tissues of patients with oral submucous fibrosis in Bihar region.

**Methods:** The study was conducted on 75 patients (aged 25–60 years) visiting the Department of Dentistry for one year. It abided by the Declaration of Helsinki of 1973 (revised 2000)

**Results:** Group III and Group II exhibited considerably greater levels of all clinical indices as compared to Group I, with statistical significance (P < 0.05). An examination of the differences between Group III, Group II, and Group I using Tukey's post hoc test found that there was a statistically significant difference between them. In Groups I, II, and III, the GI values were  $0.7 \pm 0.3$ ,  $1.8 \pm 0.2$ , and  $0.8 \pm 0.3$ , respectively. The PI values were  $0.9 \pm 0.5$ ,  $1.4 \pm 0.6$ , and  $2.2 \pm 0.5$ , to be more specific. PPD (overall) was measured to be  $3.3 \pm 0.7$  mm in Group III, whereas CAL (overall) was measured to be  $4.9 \pm 0.7$  mm. It was determined that there was no statistically significant difference in CAL between Group II and Group III. It was shown that Group I had a lower PPD and CAL. The frequency of chewing kharra was found to be  $6.12 \pm 2.58$  times per day in Group II and  $6.54 \pm 1.6$  times per day in Group III, respectively according to the results of the study. In Group II, the duration of kharra consumption was found to be  $3.97 \pm 2.52$  years, whereas in Group III, it was found to be  $7.06 \pm 2.72$  years.

**Conclusion:** Chewing kharra, according to the findings of the research, causes periodontal damage in individuals who have oral squamous cell carcinoma. Even in the presence of the premalignant state, it is possible to see a direct correlation between the poor periodontal health status and the behaviour that is detrimental to the oral health. **Keywords:** Arecoline, oral submucous fibrosis, periodontal destruction

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# Introduction

Periodontitis is an inflammatory condition of the tissues supporting the teeth, caused by certain microbes, leading to gradual deterioration of the periodontal ligament and alveolar bone, along with the creation of pockets, recession, or both. [1] Periodontitis is a prevalent inflammatory condition that may lead to excessive tooth movement and loss in adults. Tobacco usage is the primary cause of periodontitis.<sup>2</sup> Tobacco is offered in several forms, mostly for smoking and chewing. Other addictive forms include the act of sucking and smelling tobacco. Over 90% of lung cancer cases are caused by smoking tobacco, which is also a risk factor for stroke and other cardiovascular illnesses. [2.3] Oral submucous fibrosis is a chronic, complicated, premalignant disorder of the oral cavity. It is characterised by inflammation near the surface epithelium and the gradual development of fibrosis in the submucosal tissues, including the lamina propria and deeper connective tissues. There is a 1% chance of it transforming into cancer. As the condition advances, the jaws grow tight, causing the individual to be unable to open their mouth. Oral cancers are distantly connected to the habit of chewing areca nut or betel quid, which is prevalent in Southeast Asia and India and has been done for thousands of years, resembling tobacco chewing.

Both periodontitis and tobacco are primary causes of oral and oropharyngeal cancers. [4] Maternal tobacco usage may result in premature delivery and low birth weight in newborns. [5] As to a 1987 WHO research group report, individuals who use smokeless tobacco may reach blood nicotine levels equal to or surpassing those of tobacco smokers. [6] Smokeless tobacco comes in several forms. Gutkha is a prevalent kind of smokeless tobacco made from a mixture of powdered tobacco, areca nut, catechu, and slaked lime. The long-term spices, consequences of smokeless tobacco are uncertain when compared to those of smoking. [7] The illness is well known for its ability to become cancerous and is specifically linked to the practice of chewing areca

nut, the primary ingredient in betel quid. [8] Betel quid chewing is a long-standing tradition mostly observed in Southeast Asia and India for millennia. It resembles tobacco chewing in westernised cultures. This quid consists of areca nut, betel leaf, tobacco, slaked lime, and catechu. [9] Lime facilitates the active substance to remain in its freebase or alkaline state, allowing it to be absorbed into the circulation by sublingual absorption. Arecoline, an alkaloid present in the areca nut, induces salivation, discolours saliva to red, and acts as a stimulant.

The aim of the present study was to determine the effect of kharra chewing on periodontal tissues of patients with oral submucous fibrosis in Bihar region.

#### **Materials and Methods**

The study was conducted on 75 patients (aged 25– 60 years) visiting the Department of Dentistry, Netaji Subhas Medical College and Hospital, Bihta, Patna, Bihar, India for one year . The patients were sensitized about the purpose and design of the study and a written informed consent was obtained from them. A special pro forma recorded the detailed case history with periodontal indices and clinical examination.

The three groups with 25 patients each are classified as follows: Group I (n = 25) included patients with a healthy periodontium, Group II (n = 25) included patients with chronic periodontitis without OSMF, and Group III (n = 25) included patients with chronic periodontitis and OSMF.

The inclusion criteria for patients with chronic periodontitis were probing pocket depth (PPD)  $\ge 3$  mm, clinical attachment level (CAL)  $\ge 3$  mm, and habit of chewing kharra for at least 3 years >5 times per day. Patients with any systemic disease, allergies or drug usage, smokers, history of periodontal treatment in the past 6 months, or pregnant and lactating women were excluded from the study.

Periodontal indices such as gingival index (GI) [10] and plaque index (PI) [11], PPD, and CAL were measured with a graduated periodontal probe (PCP-UNC15®, Hu-Friedy, Chicago, IL, USA) for each tooth on all the six sites. On the basis of the region of quid placement, regional PPD and regional CAL scores were also calculated. OSMF was diagnosed on inspectory and palpatory findings adhering to the clinical classification by to the clinical classification by Ranganathan K et al. [12]

# Statistical Analysis

Statistical analysis was performed using the IBM SPSS Statistics Software version 20. The level of significance was set at P < 0.05.

#### Results

| radie 1: Comparison of chincal parameters among the groups |                      |                 |                  |          |
|--|----------------------|-----------------|------------------|----------|
| Parameters   | Group I<br>(control) | Group II (test) | Group III (test) | P- Value |
| GI   | 0.7±0.3              | 1.8±0.2         | 0.8±0.3          | 0.08     |
| PI   | 0.9±0.5              | 1.4±0.6         | 2.2±0.5          | 0.05     |
| PPDr (mm)  | 1.6±0.4              | 4.4±0.6         | 3.5±0.5          | 0.0064   |
| PPDo (mm)  | 1.3±0.4              | 4.0±0.6         | 3.3±0.7          | 0.07     |
| CALr (mm)  | 0                    | 4.6±0.4         | 5.2±0.7          | 0.04     |
| CALo (mm)  | 0                    | 4.3±0.5         | 4.9±0.7          | 0.05     |
| Frequency (times per day)                                  | 0                    | 6.12±2.58       | 6.54±1.6         | < 0.0001 |
| Duration (years)   | 0                    | 3.97±2.52       | 7.06±2.72        | 0.0009   |

 Table 1: Comparison of clinical parameters among the groups

All clinical parameters were significantly higher in Group III and Group II compared to Group I (P < 0.05). Pair-wise analysis using Tukey's post hoc test revealed statistically significant difference between Group III, Group II and Group I. GI for Groups I, II, and III was  $0.7 \pm 0.3$ ,  $1.8 \pm 0.2$ ,  $0.8 \pm 0.3$  and PI was  $0.9 \pm 0.5$ ,  $1.4 \pm 0.6$  and  $2.2 \pm 0.5$  respectively. In Group III, PPD (overall) was  $3.3 \pm 0.7$  mm and CAL (overall) was  $4.9 \pm 0.7$  mm. CAL between Group II and Group III was not found to be statistically significant. Group I showed a lower PPD and CAL. The frequency of kharra chewing in Group II and Group III was  $6.12 \pm 2.58$  times per day and  $6.54 \pm 1.6$  times per day, respectively. The duration of kharra consumption was  $3.97 \pm 2.52$ years and  $7.06 \pm 2.72$  years for Group II and Group III, respectively.

# Discussion

Kharra chewing (areca nut and tobacco) is the most prevalent addiction in central India. Arecoline, a natural alkanoid in areca nut, has genotoxic, carcinogenic, embryotoxic, and immunotoxic potential. [13] Several epidemiological and experimental investigations have established that quid chewing causes oral and oropharyngeal cancer, interferes with the microbial mechanism of neutrophils, and inhibits protein synthesis and attachment of fibroblasts. This in turn promotes bacterial colonization and periodontal infection. [14] As a result, the harmony between various periodontal structures is disrupted that leads to induced gingivitis and periodontitis. It also exerts excessive masticatory load on the tissues, leading to

wearing facets, attrition, sensitivity, food lodgment, and food impaction. [15]

The extracts also stain teeth and affects oral esthetics and social confidence. Arecoline also generates reactive oxygen species due to which lipid peroxidation is initiated and leads to a precancerous condition such as oral submucous fibrosis (OSMF). This chronic, insidious, disabling potentially malignant condition of the oral cavity is characterized by excessive production of collagen leading to inelasticity of the oral mucosa as well as atrophic changes of the epithelium reflected clinically as mucosal rigidity. [16] All clinical parameters were significantly higher in Group III and Group II compared to Group I (P < 0.05). Pairwise analysis using Tukey's post hoc test revealed statistically significant difference between Group III, Group II and Group I. GI for Groups I, II, and III was 0.7  $\pm$  0.3, 1.8  $\pm$  0.2, 0.8  $\pm$  0.3 and PI was 0.9  $\pm$ 0.5,  $1.4 \pm 0.6$  and  $2.2 \pm 0.5$  respectively. In Group III, PPD (overall) was  $3.3 \pm 0.7$  mm and CAL (overall) was  $4.9 \pm 0.7$  mm. CAL between Group II and Group III was not found to be statistically significant. Group I showed a lower PPD and CAL. The frequency of kharra chewing in Group II and Group III was  $6.12 \pm 2.58$  times per day and  $6.54 \pm$ 1.6 times per day, respectively. The duration of kharra consumption was  $3.97 \pm 2.52$  years and 7.06 $\pm$  2.72 years for Group II and Group III, respectively.

The areca nut in betel quid significantly contributes to the development of oral submucous fibrosis. [17-20] A 2004 research found a direct correlation between the frequency and length of chewing areca nut (without tobacco) and the development of oral submucous fibrosis. [21] Smoking and alcohol drinking, which are typical behaviours among areca nut chewers, do not individually contribute to the development of oral submucous fibrosis. [22] However, combining these with areca nut chewing might increase the chance of developing oral submucous fibrosis. Arecoline, an active alkaloid included in betel nuts, enhances fibroblasts to boost collagen synthesis by 150%. [23] Arecoline was shown to increase the mRNA and protein levels of cystatin C in individuals with oral submucous fibrosis in a dose-dependent manner. Cystatin C is a basic protein that is continuously up-regulated in several fibrotic disorders and is not glycosylated. [24]

We observed a significant difference in the periodontal parameters of particular region of kharra placement and the entire or overall teeth of same patients. The intragroup comparison in Group III between PPD region was significantly higher than PPD overall, indicating that the severity of destruction was dependent on the placement of kharra in patients with OSMF. The hardness of kharra as well as its interaction with the ingredient in turn depresses the hosts' resistance to local factors and causes greater calculus formation, accounting for the more periodontal destruction in its consumers.

# Conclusion

The research findings indicated that chewing kharra causes periodontal damage in people with OSMF. An evident link exists between the substandard condition of the gums and an unhealthy habit, even when a precancerous state is present. Additional multicentric studies with a broader population are needed to establish a correlation between the histopathologic impact of kharra on oral and periodontal tissues.

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