

## A Clinical and Epidemiologic Evaluation of Oral Cancer in India

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### ABSTRACT:-

In India, where the disease ranks first among all cancers in male patients and third among malignancies in female patients, this article discusses the epidemiology and clinical aspects of oral cancer. It has been thoroughly researched if chewing betel quida, which contains tobacco leaves or stem, and other tobacco habits, causes oral cancer. The impact of alcohol, food, and dental hygiene practices in India, however, needs further study. Also mentioned is the great prospect for early identification and intervention programs that the well-established oral precancerous lesions afford. The peak age frequency of occurrence occurs at least ten years earlier than what is suggested by literature from the West. A 2: 1 prevalence of male patients is revealed by the sex ratio. Only ten to fifteen percent of cases show localized phases. The high percentage of advanced cases, as demonstrated by available studies, is mostly to blame for the dismal survival rate. This review emphasizes the fantastic possibility for additional research and efforts in prevention and control of oral cancer in India.

**Keywords:** Oral Surgery, Oral Pathology, Oral Med

### INTRODUCTION: -

One of the top 10 most prevalent malignancies worldwide, oral cancer has significant regional variations in incidence. Where there is a high prevalence of betel quid chewing, bidi smoking, and alcohol and cigarette use, oral cancer is also prevalent. In Southeast Asia, where more than 100,000 new cases are reported each year, it is therefore a prevalent malignancy. In publications published in scientific journals, textbooks, and reports of various

scientific bodies, there is a great deal of diversity in the description and understanding of the subsite distribution for the phrases oral cancer, mouth cancer, or cancer of the oral cavity. Most other cancer locations, such as the lung, stomach, or liver, which have widely accepted definitions, are exempt from this ambiguity. In several surveys, oropharyngeal cancer is frequently mistaken for mouth cancer. Many writers neglect to list the sites that they include in categories like mouth cancer or oral cancer. The main goals of descriptive epidemiology and clinical investigations are believed to be eliminated by this diversity, which also prohibits meaningful comparisons of incidence rates, death rates, and related epidemiologic and clinical data.

### **How big the problem is:**

Studies on the significant morbidity rate that has been reported have provided the majority of the epidemiologic data on oral cancer in India. The shortcomings of the death registration system are the source of the paucity of significant mortality-based studies. In India, oral cancer is the most common malignancy in men and the third most common in women. [4] Oral cancer was estimated to account for 12% of all malignancies and 8% of all cancers in women, according to estimates based on weighted averages of crude incidence rates for three major metropolitan areas included in the National Cancer Registry Project. [4] Surveys conducted in several cancer hospitals in India show that among all cancers, mouth cancer occurs between 15% and 20% of the time. [4-6]

### **Etiologic research**

Betel quid chewing with tobacco has long been associated with the high frequency of oral cancer and precancerous lesions in India. [7] The betel quid (pan quid) is made by wrapping slices of dried or fresh areca nut (*Areca catechu*) and natively cured, dried tobacco leaves or tobacco stems in fresh green betel leaf (*Piper bet/e*) that has been covered with a paste made of slaked or shell lime (calcium hydroxide). To this basic chew, several other condiments, sweeteners, flavourings, and spices may occasionally be added in some parts of India, depending on the person's preferences. Some people might choose not to add tobacco to the standard quid. Between 5 and 60 minutes are spent chewing the quid (pan) while keeping it compressed on the buccal mucosa. Some people keep the quid while they sleep; a person may eat as many as 5 to 15 pans in a single day. In our hospital, the mean age at which this habit began was 22.7 years for male oral cancer patients, 25.1 years for female patients, and 29 and 35 years, respectively, for age-, religion-, and sex-matched control individuals.

Hirayama [9] came to the conclusion that chewing tobacco and lime mixture plays a significant part in the etiology of oral cancer by generating cancer at the place where the quid is usually maintained in his or her thorough study of oral and pharyngeal cancer in Southeast Asia. Chewers are four to fifteen times more likely to acquire oral cancer than nonchewers are, depending on the type and quantity of betel quid used. The likelihood of developing cancer is directly correlated with the frequency and intensity of chewing, according to data

from numerous studies conducted in regions where betel quid chewing is common. Virtually every case of oral cancer is linked to patients who chew tobacco in the form of betel quid. Chewing tobacco was strongly linked to oral cancer and precancer in large-scale house-to-house surveys in rural India. [8] However, Jayant [13] came to the intriguing statistical conclusion that epidemiologic data on betel chewing without cigarettes are insufficient for drawing meaningful conclusions.

Lower socio-economic groups most frequently consume toddy and arrack, two locally produced alcoholic beverages served in pots. These are prepared using a wide range of regional creativity. The former has significant levels of alcohol (between 50% and 70%), as well as charcoal, ammonium hydroxide, and several types of spices. Higher socioeconomic classes consume "foreign liquors," which are domestically produced alcoholic beverages including beer, wine, whiskey, rum, and gin. In order to find any real associations, it is believed that the role of alcohol needs to be extensively explored further in the Indian population by stratifying the different intraoral sites. Alcohol may play a role in malignancies of the floor of the mouth and the anterior two thirds of the tongue, according to correlation studies. [7]

The investigation into dietary issues, for example, has practically been eclipsed by the overwhelming evidence that tobacco use is the primary cause of oral cancer in India. Intriguingly, ingestion of vegetables was shown to be negatively correlated with the frequency of oral leukoplakia in a recent dietary survey of Christian fisherman in Trivandrum. According to Hirayama [10], malnutrition was the reason why non-tobacco chewing vegetarians had a relative risk of oral cancer that was more than three times higher than that of non-vegetarians.

### **Research in natural history**

Leukoplakia, submucous fibrosis, and erythroplasia are three clearly defined oral precancerous lesions that can develop during the protracted incubation period between the start of carcinogenic tobacco use and the emergence of invasive oral cancers; leukoplakia is the most prevalent and well-researched of these lesions. More than 75% of oral malignancies are said to develop in leukoplakias that have already developed. It has been demonstrated in follow-up studies that individuals in whom oral leukoplakias are reported to have turned malignant over observation periods ranging from a few months to several years had the majority of oral malignancies. [3]

Submucous fibrosis is a disorder that only affects people from Pakistan and India. In India, the reported prevalence rate ranges from 0.2% to 1.2%. A preliminary estimate indicates that India is home to no fewer than 250,000 instances. [7] Oral mucosa stiffness, symptoms related to limited mobility, trismus, stricturing at angles of the mouth, difficulty to expand the tongue, impaired pronunciation, and intolerance to spicy-hot food are the main characteristics of the condition. The infected mucosa may feel like it has firm submucosal bands. There are

many theories as to the actual cause of the condition, including hyponutrition, an allergic reaction of the oral mucosa to chili peppers, areca nuts, etc. According to reports, the disease starts in the connective tissue and causes subsequent changes in the epithelium.

The presence of these precancerous lesions assists in identifying a segment of the population among high-risk cigarette users who is most likely to acquire oral cancer. The average age of those with preleukoplakia, leukoplakia, and oral cancer was reported to be 43.5 years, 45.6 years, and 57.6 years, respectively, in a study of age distribution.

Preleukoplakia affected 22% of patients, leukoplakia affected 34% of patients, and oral cancer affected 75% of patients. These individuals represent the three clinical phases of oral carcinogenesis, and the observed disparities in their ages suggest the time course of mouth cancer biology. Leukoplakia's malignant transformation has reportedly not been seen in individuals under the age of 35, and it is three and a half times more common in those 55 and older than in those between the ages of 35 and 54. [7] The development of screening programs for high-risk populations can benefit greatly from these observations.

### **Medical profile**

15% to 20% of all cancer cases in India's various cancer hospitals are oral cancer cases. We described the clinical profile of 2007 patients with mouth cancer to another publication in an effort to characterize the demographic characteristics of patients with oral cancer. We discovered that the male to female ratio was 1.81:2. 68 percent of the patients identified as Hindu, 20 percent as Christian, and 12 percent as Muslim; this distribution matched that of these religious groupings in the overall population. For male patients, the mean age at diagnosis was 57.1 years, compared to 58.6 years for female patients. For both men and women, the age-frequency distribution peaked in the sixth and seventh decades, respectively.

### **Diagnosis and survival**

In India, there are less research on oral cancer survival compared to epidemiologic studies, in part because follow-up rates are low and because many patients are unable to complete their prescribed treatments due to socioeconomic issues.

It would seem that poor survival is caused by the advanced stages at which patients seek treatment and the dearth of options for salvage surgeries based on the reported studies, which are primarily case series or tiny randomized trials.

### **Chances for research in the prevention and treatment of cancer**

For research aiming at preventing and controlling overt disease, India's high prevalence of oral precancerous lesions and high morbidity from oral cancer present significant prospects. Health education initiatives, chewing and smoking cessation clinics, the role of surgery in localized precancerous lesions, the role of chemopreventive medications like beta carotene and retinoids in more extensive lesions, oral cancer screening programs, and the role of diet

in oral carcinogenesis are just a few of the programs that need to be further evaluated along these lines. In partnership with the environmental carcinogenesis unit of the British Columbia Cancer Research Centre, Vancouver, Canada, studies examining the role of beta carotene and vitamin A in the management of oral precancerous lesions are now being conducted at our center. An intervention study for the primary prevention of oral cancer among 15,000 Indian smokers in Kerala, India, appears to show that, generally speaking, improvements in the population's tobacco habits can be attained by educational initiatives. This study showed that 37% of the intervention cohort and 12% of the control cohort ceased or cut back on their cigarette use, and that after 5 years, the intervention cohort had a significant (75%) reduction in leukoplakia. An earlier study found that stopping or significantly reducing tobacco usage caused more precancerous lesions to regress. [8]

## **CONCLUSION :-**

The conclusion is that among the few human cancers for which our understanding of the etiology has reached a position that permits design of strategies aimed at reduction and ultimately elimination of morbidity from these malignancies, mouth cancers caused by beta-I quid and cigarettes.

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