

Trends Leading to Oral Cancer

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ABSTRACT

Oral health disparities have exacerbated since the popularity of chewable tobacco among the general population in Pakistan. Karachi South ranks second in the incidence of oral cancer and this frequency is steadily rising. Several molecular studies have shown a link between the use of chewable tobacco products and oral submucosal fibrosis. Oropharyngeal cancer, is being driven by HPV is now well known around the world. Studies in Pakistan on squamous cell carcinoma and precancerous oral lesions, found a high prevalence of Human Papillomavirus (HPV) in patients with male sex showing significant correlation with HPV infection. This paper highlights the causes and risks of the oral epidemic that is threatening our society.

KEY WORDS: Oral Cancer, Human Papilloma, Smokeless Tobacco.

The silent and looming epidemic of oral cancer unfortunately affects disadvantaged sects of our society; the lower socioeconomic group. The main target is the young and the children irrespective of racial ethnicity. Oral health disparities have exacerbated since the popularity of chewable tobacco among the general population. According to statistics published in 2005 Karachi South ranks second in the incidence of oral cancer, in both genders and worldwide this incidence was rated as highest ever reported.¹ A strong evidence suggest a steady rise of this frequency. Experts around the world agree that this startling increase in the incidence of oropharyngeal cancer, is being driven by HPV. In 2008, a study² on squamous cell carcinoma found a high prevalence of Human Papillomavirus (HPV) in Pakistani patients with male sex showing significant correlation with HPV infection. A similar study recently published in JCPSP in March 2012, showed alarming frequency of Human Papillomavirus in gutka eaters having precancerous oral lesions in a set population of Karachi³. This review highlights the causes and risks of the oral epidemic that is threatening our society.

This chewable tobacco habit interweaved into the cultural setup of South Asians especially with the strong inclination of children toward it, warrants urgent action. Gutka (betel, areca nut, lime and tobacco concoction), marketed locally since 1975, one of several precarious smokeless tobacco formulations, is the cause of worst observable oral lesions. The link between the use of Gutka and other head and neck malignancies is now

well established⁴. The other products most popular among our masses include Naswar⁵, Paan⁶ and Chaliya (Areca nut)⁷. All these preparations contain tobacco with other hazardous ingredients added for taste and flavor⁸. Users of these live in the misconception that its nicotine is harmless, whereas, on the contrary smokeless tobacco plunk more nicotine into bloodstream than cigarettes which makes giving-up even harder than quitting cigarette smoking, changing the overall performance of a person making him use more and more to get the kicking effect he wants. The use of any tobacco product has both immediate and long-term effects on health and overall well-being. Smokeless tobacco stains, cause gums to recede and damage the oral mucosa producing mouth ulcers, Bad breath, precancerous lesions and finally oral cancer. Nicotine from smokeless tobacco also raises blood pressure and cholesterol levels, and can increase the risk of heart attack.

According to worldwide statistics there are more than 481,000 new cases of oral cancer every year. In the United States alone someone dies from oral cancer every hour of every day. In the west oral cancer is mostly attributed to oral sex whereas, in Asia often it is connected with precancerous changes as a result of chewable habit⁸ That probably, is the reason that cancer development in Asians is mostly in the buccal mucosa (cheeks) compared to the Western countries where tongue and the floor of the mouth are the primary sites and less common sites are gingiva, labial mucosa, and hard palate⁹. The habitual eaters place the chewable tobacco in the mandibular or labial groove and suck on it slowly for 10-15 minutes and then either throw it away or simply let it sit in the mouth till it slowly dissolves⁴. The early symptoms start with a condition known as bete chewer's mucosa, a discoloured areca nut-encrusted change, is often found where the quid particles are retained. Areca nut chewing is implicated in ora

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leukoplakia and submucous fibrosis, both of which are potentially malignant in the oral cavity.

The association of HPV with oral and oropharyngeal carcinogenesis is well documented. The exposure of oral mucosa to chewable tobacco causes abrasions making the mucosal surface susceptible to HPV thus making it easier for the virus to gain entry into the basal layer of squamous epithelium. Once inside the cells of the epithelial basal layer, replication of the virus occurs in the nuclei of the infected cells, and the production of mature virions occurs in the suprabasal epithelial cell layers². Infection with HPV high risk types 16, 18, 31, 32 and 45 lead to precancerous lesions and subsequently to oral cancer. Clinical, Pathological, and Molecular progression of Oral Cancer is often masked. Benign squamous hyperplasia can often appear normal mucosa though it already harbors early genetic changes. Cellular repopulation in geographically distinct areas gives rise to multiple clinical lesions with different histopathological patterns although they arise from the same cell.

HPV infection is highly transmissible, has a variable incubation period that can culminate in latent infection with low HPV DNA copy-number in basal cells insufficient to support transmissibility in subclinical infection that is active but without clinical signs; or in clinical infection leading to benign, potentially malignant or malignant epithelial lesions. The squamous epithelium of the gingiva, hard palate and the dorsum of the tongue are completely keratinized with a superficial horny layer. The histology of oral mucosa resembles that of the uterine cervix and other lower genital tract or skin. On the basis of these morphological similarities, the presence of both the mucosal and cutaneous HPV types in different squamous cell lesions of the oral mucosa can be elucidated.¹⁰

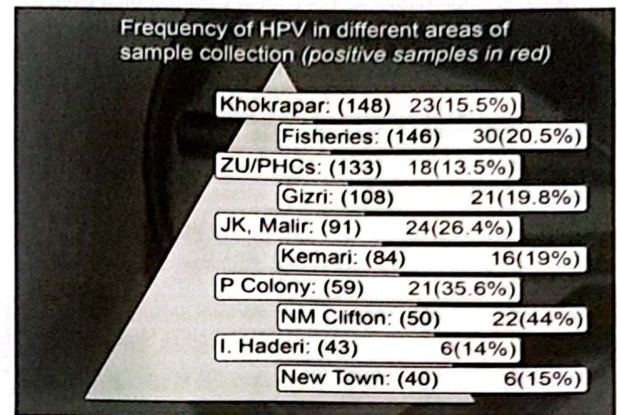
During the oral cancer awareness campaign, many camps were set up in Karachi to counsel the chewers and to find out the frequency of HPV in their oral rinse (Figure 1). Some chewers plainly refused to quit when counseled. They said they simply cannot work without chewer in their mouth. This shows its connection with conditioning as studied by Pavlov¹¹, who experimented by ringing bell while he fed his dogs. After a few repetitions, dogs would salivate by just ringing of the bell. These chewers build up associations which over time slowly weave their way through daily activity in their lives until they cannot work without a tobacco chewer in their mouth. Around 2000 people were counseled, in camps at 10 different locations, who belonged to 25 various technical blue collar professions and had conditioned response linkage. This habit is insidious and stubborn, and millions of lives are lost every year worldwide to it.

The most alarming finding was their awareness about the consequences of this habit. When showed the

frightening picture of the face of a mouth cancer victim, they had their own story of someone close.

Trends leading to Oral cancer are preventable. It only requires identification of risk factors, awareness of

Figure 1: Frequency of HPV in Different Areas of Sample Selection



consequences, preventive education and intervention. Intervention can be the greatest tool if the target population is identified along with its tool. Marketing candies with similar flavor or taste can be an alternative attraction for habitual teenagers. Awareness campaigns through media and educational institutes regarding information on oral hygiene, awareness of risk factors and symptoms and the importance of seeking early professional help around the target populations should be the aim for healthy Pakistan. Population screening would reduce the incidence of oral cancer, but requires careful planning, new research projects and monitory support. Participation of family physicians and medical students for early detection of oral cancer is the need of time.

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