ALCOHOLISM – HAND IN HAND WITH PREMALIGNANCY AND MALIGNANCY

1 Jaya Pradha 1 Associate professor

1 Thai Moogambigai Dental, College and hospital, Golden George Nagar, Mugappair, Chennai-600007

ABSTRACT:
Oral health - The health standard of the oral and related tissues that enable an individual to eat, speak or socialize without active disease, discomfort or embarrassment and that contributes to general well-being. Incidence and mortality rates of cancer of oral cavity in particular are rising in most areas of the world. Oral health is affected by wrong social habits or regular drinking practice of alcohol, leading to precancer and cancer. The type, quantity of alcohol and alcohol with smoking habits seems to have different roles in influencing the oral mucosa in transforming it to precancerous lesions. The aim of this article is to bring to limelight the pathogenesis, associated factors and inhibiting factors for developing precancerous and cancer lesions.

KEYWORDS: Alcohol, Precancer, Cancer.

INTRODUCTION
Epidemiological studies have often reported chronic alcohol consumption as well as smoking habits to be associated with occurrence of different kinds of oral carcinoma1 and have been quoted as one of the risk factors for developing oral cancer2. Recent studies indicate that alcohol increases the risk of oral premalignant lesions among tobacco users and never users of tobacco as well3. Therefore it is evident quote that alcohol is an independent risk factor for oral cancer. Virtually all oral squamous cell carcinomas arise from premalignant lesions, which are clinically diagnosed as leukoplakia or erythroplakia4. A better understanding of pathogenesis and etiology of the same is important for prevention of malignant lesions.

Pathogenesis:
A difficulty to establish the exact mechanism of alcohol carcinogenesis has been challenging all the way. Understanding the basic metabolism process of ethanol which is the main ingredient in alcoholic beverages is necessary to derive at the carcinogenesis process. Proposed mechanisms for alcohol induced carcinogenesis are:

1. Alcohol increases the penetration of carcinogens through the oral mucosa by increasing the solubility and permeability of oral mucosa5.
2. Chronic consumption causes mucosal atrophy and hyper-regeneration, thereby making the epithelium more susceptible to chemical carcinogens6.
3. The combination of increased influx of oxygen radicals and subsequent loss of cellular redox homeostasis can generate various DNA lesions (breaks, base modifications, degradation products of deoxyribose)7.
4. It potentiates the genotoxicity of carcinogenic agents.
5. Alcohol slows protein synthesis, so cell repair mechanism is inhibited and also inhibits DNA repair capacity.
6. Also has systemic effects such as malnutrition and immunosuppression.
7. Metabolism of ethanol to acetaldehyde which is a carcinogen.

Vol. - III  Issue 4  Oct – Dec 2011  76
The conversion to acetaldehyde is done in oral mucosa the further activities of aldehyde dehydrogenase is lacking in oral mucosa which could lead to the accumulation of acetaldehyde in oral tissues. Besides the ethanol in alcoholic beverages, it may contain other carcinogenic agents like congeners, asbestos, niacin, pyridoxine, N-nitroso compounds, urethane, ascorbic acid, and arsenic pesticide residues. The field cancerisation theory posses that several molecular alterations with distinct genetic changes occur frequently and multiple events or hits accumulate until malignant transformation occurs. In accordance with this theory alcohol plays the role of promoter.

**Aggrevating factors of pathogenesis:**

In many studies it has been quoted that man who use tobacco along with alcohol had ten times the risk of developing premalignant and malignant lesions than who never used tobacco and drink.

The possible reason for this additive effect could be because of the carcinogen acetaldehyde present in tobacco. This acetaldehyde when coupled with acetaldehyde resulting from alcohol metabolism increases the salivary tissue acetaldehyde levels. Heavy smokers who drink heavily are many times more likely to develop esophagopharyngeal cancers than non smokers who drink. This could be because the harmful cellular effects of chemicals and free radicals in cigarette smoke are potentiated if the cells have been already been damaged by chronic exposure to alcohol.

Studies have proved that alcoholic beverages leads to an increased risk in cancer with relatively increased risk associated with heavy drinking habits, at least for oral and upper aerodigestive tract carcinomas. The possible reasoning could be large quantity of alcohol come in direct contact with cavity and esophagus. Also a non topical systemic effect is likely since alcohol, alcohol come in direct contact with cavity and esophagus. The generation of 1-hydroxyethyl radicals following an acute dose of ethanol can be inhibited significantly by pretreatment with vitamin C or vitamin E (400 mg/kg i.p daily for 5 days). Specific dietary components like retinal, β carotene, δ tocopherol, vitamin A, riboflavin, zinc, selenium, turmeric oil, Chinese tea, resveratrol in wine have consistently been proved to confer protection from and even remission of carcinogenic processes.

**Cytologic alterations in oral mucosa**

Research has brought to the lime light the increase in nuclear areas, epithelial atrophy due to decrease in basal cellular size, dysplastic changes with keratosis and increased number of mitotic figures, increased micro nucleus frequency related to mutagenic effects of alcohol in buccal mucosa cells of chronic alcoholic smokers. A strong synergistic effect is cited when alcohol and tobacco intake in form of smoking or chewing, it has been reported a six fold increase in micronuclei frequency of alcoholic smokers when compared to individual who were strictly alcoholics or smokers confirming the synergistic effect.

**Inhibitors of pathogenesis in alcoholism**

The generation of 1-hydroxyethyl radicals following an acute dose of ethanol can be inhibited significantly by pretreatment with vitamin C or vitamin E (400 mg/kg i.p daily for 5 days). Specific dietary components like retinal, β carotene, δ tocopherol, vitamin A, riboflavin, zinc, selenium, turmeric oil, Chinese tea, resveratrol in wine have consistently been proved to confer protection from and even remission of carcinogenic processes.
CONCLUSION

Chronic exposure to ethanol may be associated with carcinogenic cytologic changes (oral premalignant and malignant lesions) in oral mucosa, even in the absence of tobacco smoking. The ability to control oral cancer depends on two cornerstones: prevention and early diagnosis. So high daily intake of alcohol needs to be curtailed by individual's determination. Healthy nutritive foods and encouragement to seek regular oral examination by dentist for early detection of premalignant and malignant lesions are the need of the hour to minimize the risks of the same.

References:


2. Seitz HK, Poschl G, Simannowski UA. Alcohol and cancer. Recent dev.Alcohol 1998;14:76-95


PMid:1834240

doi:10.1002/ijc.10723

doi:10.1126/science.275.5297.218
PMid:8950106


PMid:10203992

Corresponding Author

Dr. D. Jaya Pradha
Thai Moogambigai Dental College and hospital, Golden George Nagar, Mugappair, Chennai-600007
Mobile: 9884311130
Email: drjpkop@yahoo.co.in