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ORAL CANCER- ITS RISK FACTOR

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ABSTRACT

The purpose of the present review is to evaluate the awareness of oral cancer, etiology, signs and symptoms, stages, risk factors, role of nutrition in this condition, and the risk of the oral cancer with use of different forms of tobacco consumption habits. Cancer is the eventual outcome of the interaction between genetic factors and environmental exposures. Nutrition and diet, as environmental factors and determinants of growth and body composition can contribute to the risk of some human cancers such as oral cancer. There is an interdependent relationship between nutrition and health of oral tissues. Oral health determines the type of food consumed and ultimately the nutritional level. To reduce the risk of oral and pharyngeal cancer, especially squamous cell carcinoma, the most common oral cancer, diet must be optimized, primarily to reduce calorie intake and the monounsaturated fatty acids. Consumption of fruits, vegetables, and cereals, which are the major source of vitamins and fiber, should be adequate in the daily diet. Optimal levels of daily allowance of micronutrients like vitamin C, E, antioxidants, zinc, β-carotene, and folate are effective in prevention of oral cancer. There is also evidence on the benefit of commencing central nutritional support preoperatively until patients can meet their nutritional requirements unaided postoperatively via the oral route.

INTRODUCTION

Oral cancer is a part of cancer group called head and neck cancer and is defined as an uncontrollable growth of cancerous cells that invades the mouth (called oral cavity) and the part of the throat behind the mouth called oropharynx.

Oral cancer starts with in the mouth or pharynx cells. Normally these cells grow and divide to form new cells, and when they get old they die allowing new cells to take their place. When this process is impaired the cells start to divide and grow abnormally the old cells do not die when they should and the result is an abnormal tissue mass or tumor (Alina morrow).

Cancer is a disease characterized by the development of a population of cells that have reduced normal regulation of growth, replication and differentiation, which invades surroundings or distant tissues. The maintenance of cellular and tissue integrity over a life time relies up on a tightly regulated series of process, some cell division and differentiated to programmed cell death(Apoptosis), (Jim Mann & Ruswell).

Oral cancer is the eleventh most common cancer in the world (Atessa *et al..,* 2010). Despite advances in the diagnosis and treatment of oral cancer, the proportion of oral cancer cases diagnosed at early and localized stage is still below 50% (Atessa *et al.,* 2010; Patton *et al.,* 2005). Oral cancer is one of the ten leading cancers in the world. In India, it is the most common cancer among males and third most common cancer in females (Mathew, 2007).

The predisposing factors for oral cancer are heavy of tobacco and awareness is the most significant factor in delaying diagnosis and treatment of oral cancer some oral cancers may be asymptomatic (or) some may experience symptoms differently, thus ignorance of early signs of oral cancer may be the most important delaying factor. (Lachlan and Graham2007).It accounts for approximately 2.4% of all cancers (Rhodus, 2005) with high incidence rate in developing countries (Peterson, 2003). Globally it represents an incidence of 3% and 2% of all cancers among men and women respectively (Greenlee, *et al* .,2001).

The primary cause of the very high incidence of oral cancer in South Asia is the widespread habit of chewing betel quid (or pan) and related areca but use (Beedi, 1996). There are reports that the risk profile of lead and neck cancer is also in expected to change epidemiological studies have shown that the

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incidence of lead and neck cancers is decreasing except in the young, for whom the etiology remains unresolved (Devi, 1987).

Because of the well-defined risk factors, long natural history with majority of cancers preceded by pre cancer lesions and the mouth is easily accessible for visual examination, oral cancers have the vast potential for prevention this is feasible only if there is increased awareness of the condition in the community, therefore these will be better compliance to preventive measures and risk reduction strategies.

Initially tobacco consumption in the form of chewing (or) smoking was proposed as the risk factor for oral cancer though it has shown to have additive and synergistic effects with tobacco (Notani, 1988, Sankaranarayanan, 1989, Franceschi, 1990). Lather diet has also been implicated in the etiology of oral cancer (Winn 1984, Notani, 1987; La vecchia 1997).

The association between Human papilloma virus (HPV) and lead and neck cancers was observed as early as 1960. During the past two decades, the data supporting HPV as a cousative agent in the development and progression of head and neck cancers, particularly that of oropharynx has accumulated.

A study from India showed that 70% of the oral cancers were as the result of either smoking or chewing (Jayant, 1977). This has demonstrated that the incidence of lead and neck cancer is declining in both developed nations and in India, except that of oral tongue cancer for which the incidence is on the rise. To investigate the risk factor profile, a retrospective study was carried out with data obtained from the people of slum area living in both the rural and urban slums of Vijayawada located in Krishna district. Further studies have provided more evidence of a link to use of tobacco and smoking has the increased risk of oral cancer.

Those who are in the habit of chewing tobacco daily run eight times more risk of developing mouth cancer than do the non-chewers. The risk of oral cancer is higher when chewing tobacco is started at an earlier age. A definite dose- effect relationship has also been observed how long the quid is kept in the mouth is also relevant. It has observed that some of the tobacco chewers keep tobacco in the mouth while they go to sleep; this increases greatly the risk of getting cancer.

Several studies have been done in the past regarding the factors behind the diagnostic delay or oral squamous cell carcinoma (OSCC) but early detection of it still remains disappointingly constant over recent decodes. OSCC can be a small problem in numerical terms, but it is considered as a highly lethal disease in world population (Binnie and Rankin, 1984) lack of awareness in the public of the various signs, symptoms and risk factors for oral cancer are all believed to be responsible for the diagnostic delay in a long venture they are often difficult to diagnose by routine clinical examination. Diagnosis of these diseases is mostly based on the microscopic study of cells and tissues (Richard *et al.,* 2002).

Most of the oral cancers are OSCC. In past three decades, the five- year survival rate has improved but still remains in the range of 53% to 60%. Most OSCC is not diagnosed until an advanced stage, which has been one of the major reasons for minimally improved survival rate over the years (Jemal*et al.,* 2009; Yi -Shing, 2011).

There has long been recognized that consumption of tobacco and smoking is the risk factor which associated with oral cancer, and the development of the tumor in the oral cavity is the major risk, in the smokers and tobacco chewers (W Johnson, 1980)

Diet plays an essential role in the prevention of oral cancer and a great way to get important nutrients is to eat a diet high in raw fruits and vegetables. In this diseased condition and increased caloric expenditure than intake of calorie, the metabolic rate is high, some recent studies shows that there arises micro nutrient deficiency and decreasing level of anti-oxidants in the oral cancer patients and the people who are consuming tobacco are having at more risk to oral cancer.

Physiology of oral cancer

Oral cancer is part of a cancer group called head and neck cancers, and is defined as an uncontrollable growth of cancerous cells that invades the mouth (i.e. oral cavity) and the part of the throat behind the mouth (i.e. pharynx)

Oral cancer status within the mouth (or) pharynx cells normally, these cells grow and divide to form new cells and when they get old, they die allowing new cells to take their place, when this process is impaired, the cells start to divide and grow abnormally, the old cells do not die when they should, and the result is an abnormal tissue mass (or) tumor.

Oral Cancer is a type of head and neck cancer that affects the mouth. It can form in the lining of the cheeks, guns, roof of the mouth, tongue and lips.

Types of oral cancer

Oral Cancer is classified according to two criteria: i.e.

1. The cancer location
2. The cells where the cancer starts

The cancer location

According to this criterion, there are two types of oral cancer i.e.

Oral cavity cancer

It is the cancer which starts in the mouth, includes the tongue, living of the cheeks, gums and teeth, upper or lower jaw, the hard palate (i.e. mouth roof), the mouths floor (i.e. the area beneath the tongue) and salivary glands.

Oropharyngeal cancer

It is the cancer that starts in the oropharynx which includes the soft palates, the base of the tongue, uvula and tonsils (one of two small masses of lymphoid tissue located on either side of the throat). Around 2/3 of the oral cancers are found in the mouth, while 1/3 are found in the pharynx.

The cells where the cancer starts

There are two types of oral cancer i.e.

Squamous cell carcinoma

This is a type of cancer that starts in the flat cells (squamous cells) that cover the surface of the oral cavity and squamous cells carcinoma represents more than 90% of all oral cancers. In its early stages, this cancer is confined to the lining layer of the cells and is called carcinoma in situ, but when it extends

beyond the lining, it is called invasive squamous cell carcinoma.

A variant of cell carcinoma is veracious carcinoma this is alow grade cancer that rarely metastasis and has a good prognosis, this type of oral cancer is common among patients who chew tobacco (or) use snuff (a fine- ground tobacco which is sniffed (or) shorted). It represents less than 5% of all diagnosed oral cancers.

Minor salivary gland cancer

This is a type of cancer which starts within the salivary glands located in the oral cavity and oropharynx lining tissue this is a rare type of Oral cancer (Jaggi, 1990)

Causesand Risk Factors of Oral Cancer

Approximately 90% of oral cancers in India can be attributed to tobacco chewing and smoking habit. These cancers almost always occur on the side of the mouth where the tobacco quid is kept and the risk of cancer rises drastically for these who keep the tobacco quid in the mouth overnight. A link also seems to exist between infection with HSV (Herpes Simplex Virus) and oral cancer (Jaggi, 1990)

Infection of Oral Cancer With Viruses

There are several viruses that seen to increase the risk for oral cancer. i.e.

Human papilloma viruses (HPV)

These are diverse group of DNA- based viruses that infect the skin and mucous membranes within the human body. Studies suggest that infection with HPV 16 and 18 (sexually transmitted viruses) increase the risk for oral cavity cancer and oropharynx cancer.

Epstein Barr virus

It is a virus from the herpes family that causes an asymptomatic infection called infectious mono nucleosis (i.e. a medical condition common among young adults and adolescents, characterized by fever, sore throat, muscle soreness, and fatigue, and sometimes, with a development of white patches on the tonsils (or) in the back of the throat). The connection between the Epstein-Barr virus and oral cancer is still being investigated.

Herpes simplex viruses

It is one of the causative factor herpes simplex viruses causes a viral infection. The connection between this virus and oral cancer is still being investigated (Werning).

Risk Factors of Oral Cancer

This disease has much in common with squamous cell carcinomas (SCC) arising elsewhere in the upper aero digestive tract, which share common risk factors.

Tobacco use

Tobacco has a linear do response carcinogenic effect in which duration is more important than the intensity of exposure. The major carcinogenic activity of cigarette smoke resides in the particulate (tar) fraction which contains a complex mixture of interacting cancer initiators, promoters, and co- carcinogens. In the late 1950s, a land mark case control study by Dr. Ernst Wynder established the link between tobacco use and occ.

This was followed a year later by a cohort study of more than 180,000 men that revealed a higher risk of death due to SCCHN in cigarette smokers than in men who never elevated risks for SCCHN death in cigar and pipe smokers.

Most importantly the strength and consistency of the association between smoking and SCCHN have been demonstrated in numerous case- control and odds ratios in the 3 to 12 fold range. Furthermore, these studies consistently showed a dose- response effect with an increased risk of SCCHN that was related to the duration and frequency of smoking and a decrease in risk that was related to the length of time since tobacco use had been terminated.

The strength of the association between tobacco use and SCCHN as well as the biologic possibility of well- established tobacco-induced carcinogenesis model have established tobacco as the chief etiological agent in SCCHN.

Although the risk of bronchogenic carcinoma appears to be less significant for cigar and pipe smokers than for cigarette smokers, these forms of tobacco use are also clearly associated with an increased risk of OCC (Bakar, *et al*.,).

The cooling of saliva containing carcinogens in low areas of the mouth may account for the frequent occurrence of oral carcinomas along the lateral and ventral surfaces of the tongue and floor of the mouth.

Smokeless tobacco use has also been associated with OCC. It may be related to risk of developing cancer. Smokeless tobacco users and pipe smokers who frequently position the quid or pipe stem in the same place. Often develop carcinomas at that site, which suggests that physical and thermal trauma may contribute to OCC.

Most tobacco consumed in India is in the form of unregulated tobacco products. Beedis (hand- rolled, filter-less tobacco cigarettes produced at home, mainly by millions of poor women and children) account for at least 40% of all tobacco consumed in India. Other common tobacco products in India include hookah (Traditional water pipe), Chutta (clumps of tobacco smoked with the lighted end inside of the mouth), Mishri a powdered tobacco rubbed on the guns and various other forms of smokeless tobacco products furthermore, Pano(a mixture of betel leaf, lime, catechu and areca nut) is commonly chewed in India and further more gutka (is one of the smokeless tobacco product contain Arsenic, Cadmium, lead, Mercury, Nicotine, Water menthol, time, Betel nuts, Catechu, Saffron and other permitted spices and flavors) are having more risk of OCC. These are either mixed with tobacco or alone and area strong risk factor independent of tobacco use for Caronia of the oral cavity.

Health effects of tobacco

The epidemiological studies in recent years have confirmed the harmful effects of tobacco. Tobacco use and exposure are associated with a wide range of debilitating diseases including various types of cancers, coronary heart disease, obstructive pulmonary diseases, peripheral vascular disease, stroke and acid peptic disease. In 2000, globally, the leading causes of death from smoking included cardiovascular diseases

(1.69million deaths), chronic obstructive pulmonary disease (0.97 million deaths) and lung cancer (0.85 million deaths) (Ezzati & Lopez). The latest US Surgeon General's Report (2004) indicates that smoking harms almost every organ of the body. By 2030, tobacco is projected to be the single biggest cause of death worldwide (World Bank*.)*.Worldwide, tobacco causes nearly five million deaths annually (one in ten adults) with 2.41 million deaths in developing and 2.43 million in developed countries. The death toll is projected to rise to 10million by 2030 with seven out of ten deaths in the developing world.

Alcohol use

Tobacco is not the only factor in the complex causality equation of these cancers. Alcohol, another etiologic agent for risk of SCCHN, is an important promoter of carcinogenesis and contributes to at least 75% of SCCHN. Furthermore alcohol appears to result in an increase in risk of developing SCCHN that is independent of tobacco use.

Excessive alcohol consumption of alcohol is another risk factor that directly causes oral cancer. Studies conducted in developed countries suggest that tobacco and alcohol. To gather increases the risk for oral cancer by almost 80% because they act synergistically.

Nevertheless, the major clinical significance of alcohol consumption appears to be that it potentiates the carcinogenic effect of tobacco at every level of tobacco use, although this effect is most striking at the highest levels of alcohol use. The magnitude of the effect is at least addictive but may be multiplicative for certain SCCHN subsides and at the highest levels of exposure.

Genetic Susceptibility

The predominant risk factor for SCCHN is a history of exposure to tobacco and alcohol. However because only a fraction of smokes and drinkers develop cancer, variations in genetic susceptibility may be equally important in the etiology of SCCHN. The SCCHN has a genetic component has also been supported by large family studies demonstrating a three- to eightfold increased risk of SCCHN in the first degree relatives of patients with SCCHN (WHO, 1997).
Furthermore, there is molecular epidemiologic evidence for genetic susceptibility in SCCHN patients.

According to this hypothesis, inherited differences in the efficiency of carcinogen metabolizing systems, DNA repair systems, or cell- cycle control/ apoptosis system influence an individual's risk for tobacco induced cancers. Identifying such at- risk individuals in the general population, by use of these biomarker assays, would substantially improve primary prevention, early detection and secondary prevention strategies.

Infectious Agents

Although it has been suggested that various infectious agents play a role in head and neck carcinogenesis only Epstein bar virus (EBV) and human papilloma virus (HPV) can be implicates as etiologic agents in head and neck carcinogenesis based on current scientific evidence. EBV infection appears to be associated with most nasopharyngeal carcinomas (Gillison *et al*.,). HPV -16 infection is associated with approximately 50% of Oropharyngeal carcinomas, but HPV does not appear to play major carcinomas, but HPV does not appear to play a major role in OCC.
HPV infection may also be involved in the etiology of SCC arising in the Sino nasal tract. Although herpes simplex virus infection has been suggested as a risk factor for OCC, this correlation ion has not been confirmed.

Marijuana

Marijuana smoke has 4 times more tar and 50% more benzopyrene and automatic hydrocarbons than does tobacco smoke. Although anecdotal evidence has long suggested that marijuana use is a risk factor for SCCHN, few studies have found direct evidence that marijuana is an etiologic factor for SCCHN because most marijuana users also use tobacco and alcohol (Paymaster *et al.,)*

A recent case- control study demonstrated a cigarette adjusted risk for SCCHN of 2.6 (95% confidence interval 1.1- 6.6) associated with marijuana use and evidence for a dose-response relationship (Mehta *et al.*,). However a large retrospective cohort of 64,855 members of a health maintenance organization found no association between marijuana use and tobacco related cancers (Reichart P).

Diet

The lack of vitamin- A,C and E, iron, selenium and foliate in the diet can increase the risk for oral cancer.

Epidemiologic evidence from traditional case- control studies has suggested that diets high in animal fats and low in fruits and vegetables may increase the risk for SCCHN (Thomas *et al.,*).

Several case–control studies have correlated that consumption of salted fish with increased risk of nasopharyngeal carcinoma, perhaps because of the high nitrosamine content of preserved foods such as salted fish (Orr & lancet 1933).

There is some evidence that vitamin-A and beta- carotene are responsible for the protective effect of diets high in fruits and vegetables and deficiencies in carotenoids appear to be a risk factor for SCCHN and lung cancers (Warnakulasuriya, 1995).

It is not known, however, which of the more than 500 carotenoids are protective, what chemical interaction occurs or what protective role of mother micronutrients in carotenoid-rich food may play. Others have found that high intake of vitamin–C and E is also protective(Thomas, 1993) .

Dietary intake is frequently difficult to assess and validate in particular it is often difficult to determine the constituent nutrients of specific foods.

Occupation

Although occupational exposures probably play a minor role in the development of SCCHN, they are major risk factor for malignancies of the sin nasal region. The most important exposures occur in the metal working and refining, wood working and leather/ textile industries (Maher *et al.,*)

Age Distribution

The incidence of oral cancer increases with age in all parts of the world. In the west 98% of cases are over 40 years of age. In

many cases patients are less than 35 years old, owing to heavy abuse of various forms of tobacco.
Furthermore, it is now clear that in many western countries there has been, during the post 2 (or) 3 decades; an alarming rise in the incidence of oral cancer (Particularly among younger men) a trend that appears to be continuing (Jordan J B, 1997).

Immuno Suppression and Lip and Oral cancer risk

Individuals with HIV/AIDS are at increased risk for a limited number of neoplasms, especially Kaposi's sarcoma, which is commonly present in the mouth and lymphoma. This occasionally presents as an oral mucosal (or) intra bone lesion in the jaws (Jordan, 1997).

SCCS of the mouth (or) pharynx are not, however, more common in these patients and the characteristic oral hairy leukoplakia (OHL) of HIV disease does not appear to undergo malignant transformation.

Immuno suppression organ transplant patients are at increased risk of lip cancer. However which is principally due to the effects of ultraviolet light, although the effects of smoking, if present continue to have their role (Winn, 2001).

Signs and Symptoms of Oral Cancer

Oral cancer is a type of head and neck cancer that affects the mouth. It can form in the lining of the cheeks, guns, roof of the mouth, tongue and lips.

Unfortunately, most oral cancers are asymptomatic in the early stages and the symptoms occur when the tumor has reached an advanced stage of development.

White and Red Patches in the Mouth or Lips

A white (or) red patch (or combination of both) inside the mouth or the lips are the most commonly experienced symptom of oral cancer. In the early stages, these white and red patches are warnings of abnormal cells, a pre- malignant condition. If left untreated, it can become cancerous.

A sore or bitter in mouth

A sore or bitter in the mouth (or) on the lip that won't heal is a symptom of oral cancer, Sores that lost longer than 2 weeks.

Difficulty in Swallowing

This symptom can include difficulty in chewing, moving the jaw, speaking or moving the tongue. This is a non- specific symptom of oral cancer and can be caused by many other conditions.

Earaches

Frequent or persistent earaches need to be evaluated by a physician. An earache is usually indicative of an infection however in some cases, it can be related to oral cancer.

Changes in the way teeth fit together

This can include dentures not fitting correctly oral cancer can also cause loose teeth

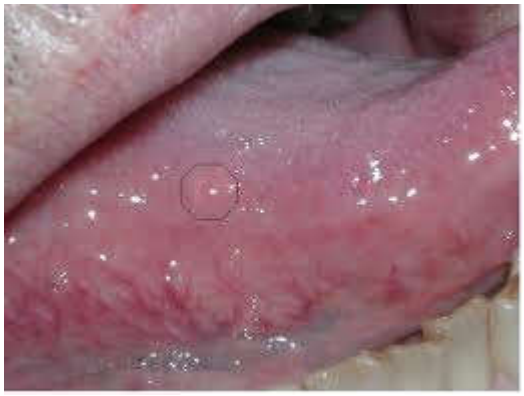
Bleeding from the mouth

Bleeding from the mouth can be caused by many things, but it is still symptom of oral cancer and needs to be checked out.

Some of the symptoms may include

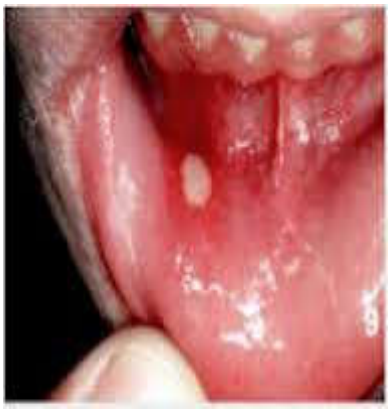
- A mouth sore or ulcer that does not heal (or) bleeds easily
- A white / red patches in the mouth
- An irritation lump or thick patch in the mouth, lip or throat
- Chewing (or) swallowing difficulties
- Unexplained numbers, loss of feeling, pain (or) tenderness in the face area, mouth or throat.
- Jaw swelling which causes the denture to not fit any more
- A lump (or) thickening on the lips, gums (or) in the mouth.

Sign and Symptoms of Oral Cancer

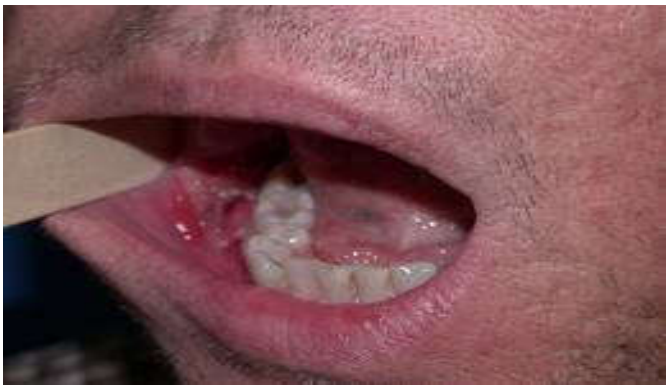


Early sign

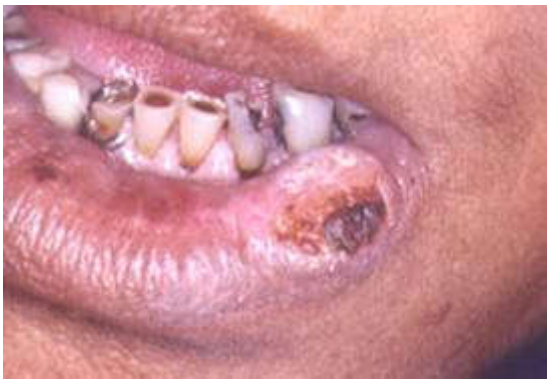
Mouth ulcers



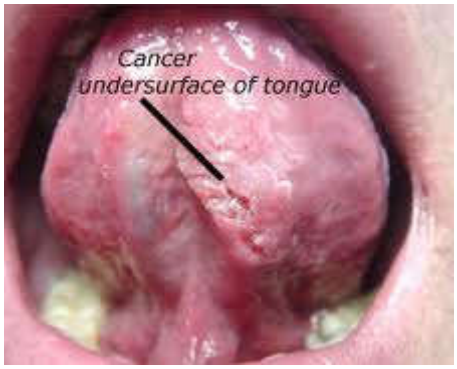
White patches on oral cavity



Red patches on oral cavity



Cancer sign on lips



Cancer on tongue

Stages of Oral Cancer

An important prognostic predictor for oral cancer is the clinicopatologic stage. A clinicopatologic stage describes the cancer developmental phase and is established according to several criteria i.e. 1.The tumor size 2.Cancer location 3.The cancer extent.

The most common staging system used for oral cancer is the American joint committee on oral cancer (AJCC). TNM, the TNM system refers to (Tumor features-size and invasion level), (N the lymph nodes involved-lymph nodes are part of the body immune system), (M-the cancer metastasis-Metastasis stage is the last development cancer stage when the cancer has spread to distal organs. (Organs situated far from the origin point).

Table 1

TNM staging of oral cancer :-
Primary Tumor (T)
Tx Primary tumor cannot be assessed
T0 No evidence of primary tumor
Tis Carcinoma in situ
T1 Tumor 2cm/ less in greatest dimension
T2 Tumor more than 2cm but not more than 4cm in greatest dimension
T3 Tumor more than 4cm in greatest dimension
T4 Tumor invades adjacent structures
(Ex:- Through cortical bone, into maxillary sinus, skin, Pterygoid muscle, deep muscle of tongue)
NODAL INVOLVEMENT (N)
NX Regional lymph nodes cannot be assessed
N0 No regional lymph node metastasis
N1 Metastasis in a single ipsi lateral lymph Node, 3cm or less in greatest dimension
N2 Metastasis in a single ipsilateral lymph node more than 3cm but not more than 6cm in greatest dimension; or in multiple ipsilateral lymph nodes; non more than 6cm in greatest dimension; or in bilateral (or) contralateral lymph nodes, none more than 6cm in greatest dimension
N2a Metastasis in a single ipsilateral lymph node more than 3cm but not More than 6cm in greatest dimension
N2b Metastasis in multiple ipsilateral lymph nodes none more than 6cm In greatest dimension
N2c Metastasis in bilateral or contralateral lymph nodes, none more Than 6cm in greatest dimension
N3 metastasis in a lymph node more than 6cm in greatest dimension
DISTANT METASTASIS (M)
Mx Distant metastasis cannot be assessed
M0 No distant metastasis
M1 Distant metastasis
STAGE GROUPING
Stage 0 Tis no mo
Stage I T1 no mo
Stage II T2 no mo
Stage III T3 no mo, T1 (or) T2 (or) T3 N1 Mo
Stage IV Any T4 lesion or
Any N2 (or) N3 lesions or
Any M1 lesion

Source:-Modified from AJCC manual for staging of cancer 1997, Ed: Fleming ID, *et al.*,

Nutritional support in oral cancer

Nutrition refers to the status of body cells in terms of necessary materials or nutrients required for physiologic growth and metabolism. Nutrition and health are closely connected and malnutrition can seriously endanger health. The consequences are a higher risk of developing disease. Of these, cancers are of special importance, because they are the second most important cause of premature adult death after cardiovascular disease, globally (Gallus, *et al.,*).

Cancer is the eventual outcome of the transformation of normal cells by DNA-reactive, genotoxic carcinogens and growth promotion of mutated cells by enhancing factors. Thus, cancer is the product of interaction of genetic factors and environmental exposures like ionizing radiation, smoking, specific infectious agents, and dietary factors, which develops over along time and goes through many stages.

According to World Health Organization (WHO) reports, 35-55% of human cancers and approximately 15% of oropharyngeal cancers can be attributed to dietary deficiencies or imbalances.

Energy balance and fat food diet

Energy balance results from the exact equilibrium between caloric intake and caloric expenditure. A caloric intake larger than caloric expenditure results in overweight or even obesity, which have been recognized as risk factors for the development of cancer(Gerber).

Investigations concerning the role of dietary lipids have demonstrated correlation between fat intake and carcinogenesis (Wiellett &, Stampfer). On the other hand, it was shown that

lipid consumption including its amount and composition is closely related to the development or prevention of tumors (Actis & Eynard).

Essential fatty acids such as fish oil and vegetables rich in n-3 polyunsaturated fatty acids must be incorporated into diet and have a protective effect against cancer. This effect would be related to an increasing production of antiproliferative metabolites. On the contrary, monounsaturated fatty acids like n-9 oleic acid, as the main source of fat, behave as a tumor promoter in breast, colon, oral, and salivary gland cancers(Clark SD, Larsson SC, Kumlin M,). Possible mechanisms through which fatty acids may influence carcinogenesis include effects on membrane integrity, increase in lipid peroxidase, and impairment of nutrient metabolism (Woutersen & Apple).

Antioxidants and fiber

Oxidative damage is recognized as playing a role in the pathogenesis of cancer, which could arise from incorrect nutritional habits and lifestyle practices. This process can cause DNA damage, which is a basic mechanism of cancer induction. Assessment of oxidative DNA damage in 24vegetarians, compared to 24 nonvegetarians, showed that DNA strand breaks oxidized purinewere significantly lower in vegetarians (Haegele). Sufficient antioxidative status (overthreshold values of natural essential antioxidants) is crucial in free radical defense.

Intakes of protective food commodities (e.g., fruits, vegetables, dark grain products, grain sprouts, and oil seeds) were significantly higher in vegetarians. On the other hand, their diet was significantly more rich as a source of antioxidants and fiber (Lesgards).

Micronutrients

Although a great deal of attention has been given to protein and malnutrition in patients with head and neck cancer, micronutrients like vitamin C, E, β-carotene, lycopene, folate, and zinc have important roles in carcinogenesis (Heimburger). It seems that apart from the antioxidant properties of these substances, polymorphism in the detoxifying enzyme GST activity and other metabolic genes can also modulate the risk of cancer. These processes are also modulated by the plasma level of micronutrients (Masala).

Studies have shown that DNA adducts, which are a reliable indicators of genotoxic damage, are significantly and inversely correlated with the intake of β-carotene and vegetables. (Masala). Stratification by GST genotype showed a strong inverse correlation of DNA adducts levels with increasing consumption of vegetables, fish, β-carotene, vitamin C, niacin, potassium, and folate(Pallic). Vitamin E and β-carotene can also cause regression of oral leukoplakia. (Nagao *et al.,*).

CONCLUSION

To conclude this review, regardless of all the type of oral cancer, it is still unknown that what really causes its development. Recent studies shows that usage of tobacco may be the leading cause of oral cavity and oropharyngeal cancer development and it is believed that smokers are more likely to develop oral cancer than the non smokers. The ability to control oral and oropharyngeal cancer will depend on two concerns such as prevention and early diagnosis. Screening and early detection of oral cancer using various diagnostic aids mentioned here will decrease the risk of oral cancer.

Malnutrition can cause poor oral health and poor oral health can indirectly cause of malnutrition. Diet plays a major role in prevention of the disease, hence taking balenced diet which contains sufficiant amount of micro nutrients and anti oxidents like vitamin-A, C, E, K, and folate are needed.

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