Free literature review on oral cancer

Science, Genetics

Factors such as race, sex and age may be significant benchmarks in the analysis or understanding of epidemiological details such as incidence and mortality rates of oral cancer. 1 For instance, the incidence and mortality rates in men are twice more than in women. 1 Based on race, African American males have a higher incidence rate than White males. 1 This also applies between a comparison between African American females and White females respectively.

Despite the occurrence of new cases of oral cancer, the overall incidence rate trend over the last 30 years has been declining. 2 The incidence rate of oral cancer among has experienced the greatest decline between the years of 2000 and 2004 especially among the black males. In certain cases this rate has been declining at a rate of 1% between 2002 and 2012. 3 On the contrary, the incidence rate of oral cancer among black females has increased over a 30-year period of between 1975 and 2004. 2

Mortality rates have also been declining. This decline in mortality has been greatest between 1995 and 2004. 2The age adjusted mortality rates was higher in males than in females based on a period between 1975 and 2002. 4Latinos recorded the lowest mortality rates. 4In the United States, the death rate attributed to oral cancer is 2. 4 per 100, 000 men and 0. 7 per 100, 000 women as recorded between 2005 and 2009. 4 In a country such as the UK, smoking is responsible for 85% of the deaths related to oral cancer.

A number of risk factors are linked with the causation of oral cancer. These include tobacco and alcohol use that provide or introduce chemical and physical irritants that alter hormonal imbalance leading to the development of cancer. Majority of oral cancer patients are tobacco smokers. The risk

factors associated with tobacco and alcohol use are 75% preventable. 5 However, the oral cancers whose cause is unknown account for the remaining 25% and are usually not is preventable. 5 Tobacco is responsible for the development of intra-oral carcinogens. Tobacco smoke contains more than 300 carcinogens and some these are also present in non-smoked tobaccos. 8 Tobacco smoke has carbon monoxide, thiocyanate, nicotine and their metabolites. Alcohol is associated as being the cause of oral cancer. Chewing of tobacco, which is common in Asian countries, provides a source of carcinogens. 5Tobbaco contains betel quid, which is responsible for increasing exposure to carcinogenic tobacco-specific nitrosamines. 5 Alcohol acts as a solvent and enhances the penetration of carcinogens into the target tissues. 5Forms of alcohol include wine liquor and beer. Beer and wine have a higher chance of causing oral cancer than hard liquor. Acetaldehyde, present in alcohol is associated as being the tumor promoter in humans. 5 HPV (human papillomavirus) has also been associated as a risk factor for oral cancer. 5The virus itself can be detected in condylomas, squamous cell papilloma and malignant oral lesions. 5 In the oral cavities, HPV likelihood is 59%, while, in the pharynx and larynx, it is 43% and 33% respectively. 5 The proportion of HPV infected lesions that develop to malignant tumors is small. Thus, other risk factors contribute in the malignant transformation.

Poor diets also contribute to the development of oral cancer. Fruits and vegetables containing vitamin A can be used to reduce or suppresses oral neoplasia. 5most of the vegetables and fruits that are used in protecting or preventing oral cancer are rich in b-carotene, vitamin E and vitamin C and have anti-oxidant properties. 5However; the micronutrient responsible have

not yet been identified. Iron deficiency associated with Plummer-Vinson syndrome, which causes oral epithelial atrophy is associated with cancer of the upper air and food passages. Therefore, dietary iron is significant in preventing this condition.

Family history of cases of oral cancer may be a risk factor. Patients may lack the ability to repair DNA damaged by the tobacco carcinogens for instance the benzo-alpha-pyrene diol epoxide. 5 This may be inherited in cases where patients' parents have a history of a defective benzo-alpha-pyrene diol epoxide. An immune system affected by a disease such as HIV increases an individual chance of developing cancer. In HIV infected patients, the most apparent oral malignancy is referred to as Kaposi's sarcoma. 5 The aetiological agent in this case is the Human Herpes virus type 8. 5 Oral cancers of the lip occurs in transplant patients receiving immunosuppressive therapy. 5Candida induces epithelial proliferation and produces carcinogens. Poor oral hygiene has also been linked to contribute to the development of oral cancer. 6 Poor oral hygiene fosters the carcinogenic action of tobacco. 6 The molecular changes of oral cancer indicate a genetic change that accumulates over period of years. The genetic changes occur because of oncogene activation and tumor suppressor gene inactivation. This then causes de-regulation of cell production and death. 5The genetic changes involve gene amplification and overexpression of oncogenes, which include Epidermal Growth Factor, erbB-2, myc, mutations deletions, cyclin D1 and hypermethylation leading to p16 and p53 tumor suppressor gene inactivation. Used as an indicator for negative growth signals, the tumor suppressor genes, p16 and p53 can be inactivated through point mutations

and deletions. The loss of p16 is present in advanced pre-malignant lesions. 6 These genes perform the function of ensuring there is cell cycle regulation, which involves cell cycle arrest and apoptosis. 5Gene deletions that are common in oral cancer cause the of chromosome 9p21. 6Addiotionally, in this region there are the homozygous deletions, which are common in oral cancer. 6 The loss of chromosome 17p is present in almost 60% of the invasive lesions. Presence of genomic instability occurs because of the loss of heterozygosity and microsatellite instability. 6 Increased loss of tumor suppression gene p53 causes an increase in uncontrolled cell division and progressive genomic instability.

Additionally, the deregulation of apoptosis-related genes has also been associated with promoting carcinogenesis. 5the over-expression of apoptosis regulators such as p53 and bcl-2 is common in oral cancers and oral lesions. Thus, the abnormal expression of BCl-2 and p53 contribute to the development of oral cancer. Epigenetic alterations such as methylation also affect the tumorigenesis process. 6The epigenetic modifications cause a loss in genetic expression and play a role in the multiple processes involved in carcinogenesis. These alterations inactivate the DNA repairing genes enhancing malignant progression in the process. 6

The clinical symptoms of oral cancer vary. It may change from a small erythematous patch to a large swelling area of ulceration. On the lip, oral cancer can be identified as a painless ulcer. Additionally, some oral cancers may be presented in the form of raised nodular surface, which are characterized by various color variations such as red or white. Areas commonly affected include the floor of the mouth and the tongue. In certain

cases, the oral cancer may invade the oral cavity, which then affects the underlying issues. The lesions that develop are normally painless and not easily detected in early stages. Histopathology features include heavy pigmentation, large epithelioid melanocytes. The lesional cells are hyperchromatic and have a granular pattern of chromatin and high nucleochromatic ratios. 7

The treatment of oral cancer, like other cancers, requires a multidimensional approach with the input from several medical practitioners and family support. This is especially so since the treatment may call for surgery, radiotherapy, dental intervention, chemotherapy and even nutritionists. The treatment procedure implemented is dependent on the stage of the disease. Early stages are treatable through surgery without the need of reconstructive surgery. The effects of such treatment procedures include the effect they have on an individual’s chewing habits, speech and even dental and facial appearance. Family ought to provide adequate care to such patients at such times and even encourage them to aid in psychological treatment. The major treatment approach for oral cancer is surgery. Surgery is applied in the advanced stage of the disease. 8Surgery targets to cut out the tumor. This is normally applied by ensuring that a good section of the underlying tissue to ensure that the cancer does not spread. 8 In the case of lip cancer, Mohs surgery can be used. This method allows the removal of tissues piece by price and at the same time checking margins from each cut out of the tissue. 8 Skin grafts and flaps can also be used in situations where the cut out tissue is deep. The skin grafts and flaps are used where a substantial portion of the tongue or floor of the mouth has been removed.

Where the cancer has affected the lymphatic nodes, dissection of the neck nodes is done. This is done by creating a cut or incision down the side of the neck. However, this procedure may resulting in damaging of accessory nerve and the patient may develop a stiff shoulder during the procedure. 8

Radio therapy is also a common form of treatment. Radiotherapy is applied in the primary treatment of small tumors instead of surgery. 8 Additionally, after surgery, radiotherapy can be applied to ensure that any affected tissue that remains is completely removed. Chemotherapy is applied in conjunction with surgery and radiotherapy. 8 drugs used in chemotherapy include cisplatin, carboplatin, 5-fluorouracil and bleomycin. 8Cetuximab, an antibody, inhibits cell growth by blocking the action of epidermal growth factor receptor. This increases the survival in patients with head and neck cancer compared to when chemotherapy is applied alone. 8

The recovery process and the probability of recovery are determined by certain factors. The level of advancement of the condition is very important determinant of whether the chances of recovery are high or low. The chances of survival are higher when the detection of the cancer is early. Similarly, if the disease has advanced too much then the chances are lower. Furthermore, prognosis is determined by whether the cancer has advanced to the blood vessels. The size of the tumor and the age of the patient also affect the treatment process and the probability of recovery. Quitting smoking also advances the chances of recovery for some patients, so long as they have not yet undergone radiotherapy.

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