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A REVIEW:- ON ORAL CANCER PATIENT SURVEY

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ABSTRACT:

This article focuses on saliva as a diagnostic medium of choice and the various biomarkers that are elevated in oral cancer. The outcome of Research proves that 90 % of the oral cancers are oral squamous cell carcinoma. The causative factors, risk factors, and screening for oral cancers are elaborated. Salivary biomarkers can be classified into genomic and protein biomarkers. Among Genomic biomarkers the role of micro RNAs are specified and the sensitivity of various protein biomarkers such as matrixmetalloproteinases, cytokines are studied.

I.INTRODUCTION

An ulcer can be defined as a loss of continuity in the skin, mucous membrane or oral mucosa. Head and neck cancers are cancers that start in the tissues and organs the head and neck.[1] They include cancers of the larynx (voice box), throat, lips, mouth, nose, and salivary glands. Most types of head and neck cancer begin in squamous cells that line the moist surfaces inside the head and neck (for example, the mouth, nose, and throat). Tobacco use, heavy alcohol use, and infection with the human papillomavirus (HPV)increase the risk of many types of head and neck cancer.[1] The risk factors for oral cancer depends on the gender, age, prolonged Sun exposure, tobacco use and alcohol, diet, HPV infection areca nut chewing ,and lodging the betel quid of the arecanut in the buccal vestibule leading to precancerous and cancerous lesions due to chronic irritation and significant changes in the oral mucosa adjacent or underneath it. This results in Extrinsic stains loss of esthetics requiring scaling and oral prophylactic measures. The most common symptom is a sore or an ulcer in the mouth that does not heal easily and which discharges or bleeds either on examination or spontaneously. Screening for oral cancer should include a thorough history and physical examination. The clinician should visually inspect and palpate the head, neck, oral, and pharyngeal regions. This procedure involves digital palpation of neck node regions, [1]

THE RISK FACTORS

Tobacco.

All forms of tobacco - cigarettes, pipes, cigars, and smokeless tobacco have been implicated in the development of OC . While tobacco confers the highest risk for OC of the floor of the mouth also, it is associated with an increased risk for all sites of OC. Tobacco use is responsible for 90% of OC deaths in males . [2]

Alcohol.

Alcohol use is a second independent major risk factor for the development of OC . For non-smokers it is the most important risk factor. Above 30 grams of alcohol per day, risk increases linearly with amount of alcohol consumed [2].

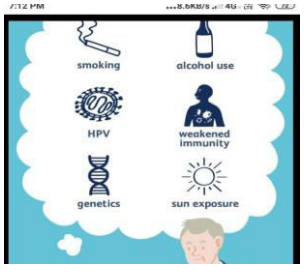


Fig.–Causes

Fig.3- Risk Factor

Chewing tobacco.

In addition to tobacco use, the use of chewing products such as betel nuts, paan, chaalia, gutka, naswar, and areca

increases the risk for OC; these products are socially acceptable in Southeast
Asia, the South Pacific Islands, and India [2]

Shammah.

Shammah, a traditional smokeless tobacco habit in the Arabian Peninsula, has a significant association between the prevalence of leukoplakia and the daily duration of shammah application in a dose-dependent manner [2].

Marijuana.

The carcinogenic properties of marijuana smoke are similar to those of tobacco. Marijuana use may interact with mutagen sensitivity and other risk factors to increase the risk of head and neck cancer[2].

Poor nutrition.

Dietary deficiencies, particularly of vitamin A, vitamin C, vitamin E, iron, selenium, folate and other trace elements have been linked to increased risk of OC . Intervention studies where diets have been supplemented have shown some beneficial effect on pre-malignant conditions and reducing the risk OC, but further work into diet and the role of chemoprevention in oral cancers is needed [2].

Ultraviolet light.

Solar irradiation is a major risk factor for cancer of the lip. The vast majority of lip cancer occur on the lower lip and many patients have outdoor occupations where sun exposure is increased. Lip cancer is three times more common in men than women which may be an effect of occupation, smoking and sun-exposure .

Irritation.

Although it has been suggested that chronic irritation to the lining of the mouth from poorly fitting or defective complete dentures may be a risk factor for OC, the majority of studies have shown no correlation [2].

Dental plaque.

Polymicrobial supragingival dental plaque is a possible independent risk factor, as it possesses a relevant mutagenic interaction with saliva, and thus oral health may be a co-factor in the development of OC .

Fig.4 – Causes of oral cancer

Mouth rinses.

Ethyl alcohol is added to mouth rinses as a solvent for other ingredients and as a preservative. Study shows that cancer was not statistically associated with mouthwash use in alcohol or tobacco users . Currently, there is insufficient clinical evidence to suggest a relationship between ethyl alcohol found in mouth rinses and OC ..

Etiology

The etiology of oral cancer in man is unknown. However, several pre-exist ing conditions have been found with such frequency in patients with oral cancer that they may be considered, at least in part. as contributory factors. For instance, chronic irritation seems to be related to the development of cancer, whereas a single episode of trauma does not. Other factors which may lead to the development of oral cancer include: El Ionizing radiation at therapeutic, not diagnostic, dosage levels. Fortunately, because of stricter rad iotherape utic guidelines, few patients are today ex posed to sufficient quantities of ionizing radiation to produce cancers of the oral cavity. El Chronic exposure to actinic radiation. [3,4]. Chronic exposure to the sun is a significant factor in the development of cancer of the lower lip, the most common form of oral cancer. The high incilip,• as well as epidermoid carcinoma of the lip in hot climates attests to the etiologic significance of long-term exposure to sunlight. Apparently, repeated exposure to ultraviolet solar rays over a period of 15-30 years results in

atrophic alterations of the exposed aspect of the lower lip, which

may develop into carcinoma. The carcinogenic action of solar rays varies according to intensity Dr. Osterkamp is in general dental practice in St. Louis, Missouri. Dr. Whitten is Associate Professor and Chairman. Department of Pathology, School of Dental Medicine, Southern Illinois University. and length of exposure and is probably **limited by pigmentation. In black people, for example, epidermoid carcinoma** of the lip is extremely rare. El The use of chewing tobacco and snuff. The prolonged use of these agents may lead to benign, premalignant and malignant lesions of the base of the tongue and the buccal mucosa. These habits, which involve holding unburned tobacco directly against the tissue in one area of the mouth, may produce a local concentration of the chemical constituents of tobacco, resulting in changes in the mucous membrane. In addition, other tobacco-related habits, such as chewing betel nut and pan, have been as sociated with dysplastic or neoplastic disease as a result of chemical irritation. A number of toxic agents, such as the lye used in the betel nut cud, have also been shown to have a contributory relationship to oral cancer. El The chronic ingestion of alcohol. This has been found to be a causative factor in the development of oral cancer, especially cancer of the floor of the mouth and tongue[3,4]. The decreased incidence of oral cancer in Great Britain in the last few years, in direct proportion to a decrease in alcohol consumption,supports this association. El The action of specific infectious agents. Atrophic leutic glossitis found in tertiary syphilis has, for instance, been associated with the development of cancer of the tongue. Epidemiological studies have found an increased incidence of oral cancer for cigarette smokers, as well as pipe and cigar smokers.2 Long-term exposure to the chemical carcinogens in tobacco and the trauma from heat and drying generated by cigarette smoking are of etiological significance in the development of cancer of the oral cavity and pharynx.

Symptoms:

- A sore, irritation, lump or thick patch in the mouth, lip, or throat
- A white or red patch in the mouth
- A feeling that something is caught in the throat
- Difficulty chewing or swallowing

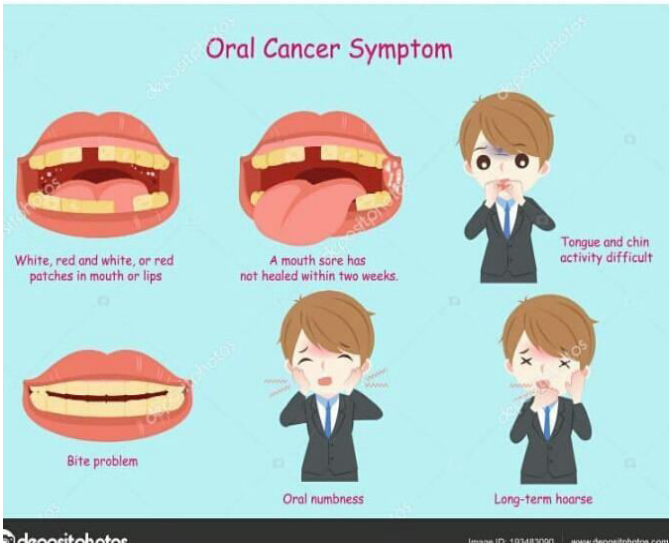
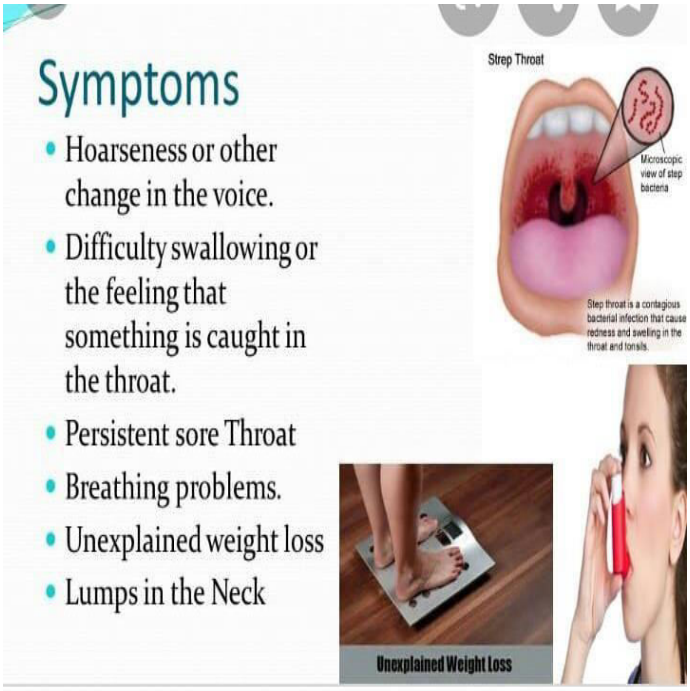


Fig. 5 – Symptos of oral cancer



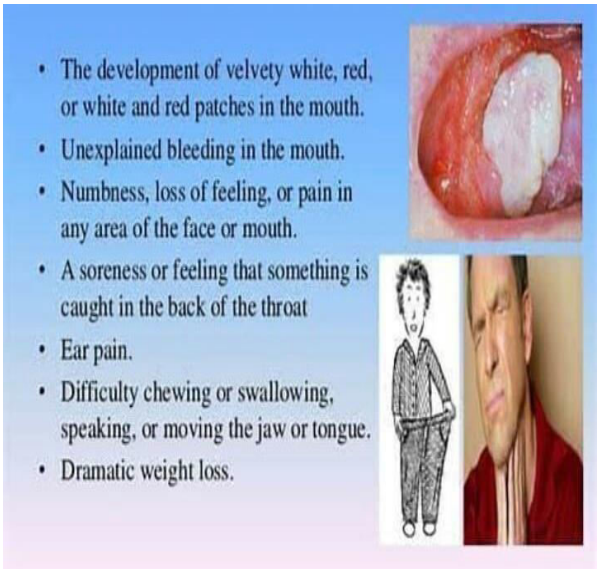


Fig 6- Symptoms

DIAGNOSIS

Most oral cancer screening programs include the simple visual inspection, whereas others attempt the use of toluidine blue, brush biopsy (exfoliative cytology) chemiluminiscence and fluorescence imaging. The last three screening methods in fact deal with the diagnosis of lesionsthat have already been detected by the patient, dentist or other clinician but a definitive diagnosis can only be made by a tissue biopsy. Diagnosis can be delayed by several months or more if the clinician treats the patient's complaints empirically with drugs instead of providing a thorough physical examination and workup.[1] Patients with complaints lasting longer than 2-4 weeks should be referred promptly to an appropriate specialist to obtain a definitive diagnosis. If the specialist detects a persistent oral lesion, a biopsy should be performed without delay. There are invasive and noninvasive methods of detecting oral cancer. Biopsy is defined as removal of tissue from a living individual for diagnostic examination. The health history, history of the lesion, clinical examination, radiographic examination, laboratory investigation, and Biopsy. Biopsies are of various types oral brush Biopsy, incisional wedge biopsy, excision biopsy, aspiration biopsy, tissue stabilization, specimen care, surgical closure. The diagnostic medium of choice is saliva because of the fact that the levels denote the systemic health and disease status. The first report of saliva as a diagnostic medium for oral cancer was published by Liao et al. who identified mutations in exon4, condon63 in 5 out of 8 patients with oral squamous cell carcinoma. Saliva can be utilized for early detection of oral cancer as this body fluid maintains continuous contact with these lesions. Diagnosis of OSCC is currently based on biopsy test, which is an invasive method. There is a need for developing a noninvasive screening tool[1] (

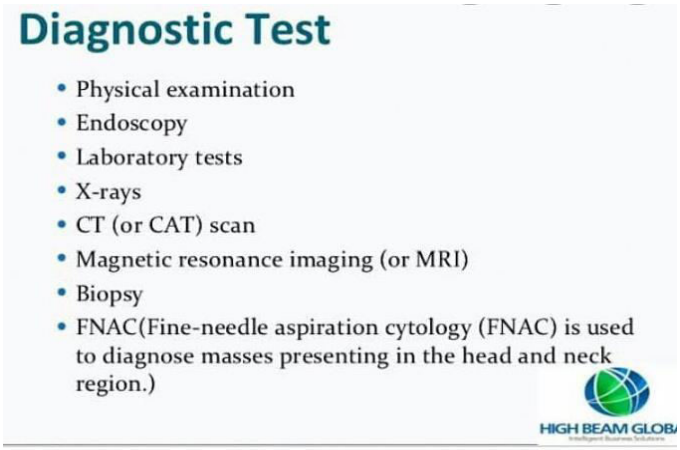


Fig – 7. Diagnostic Test Of oral Cancer

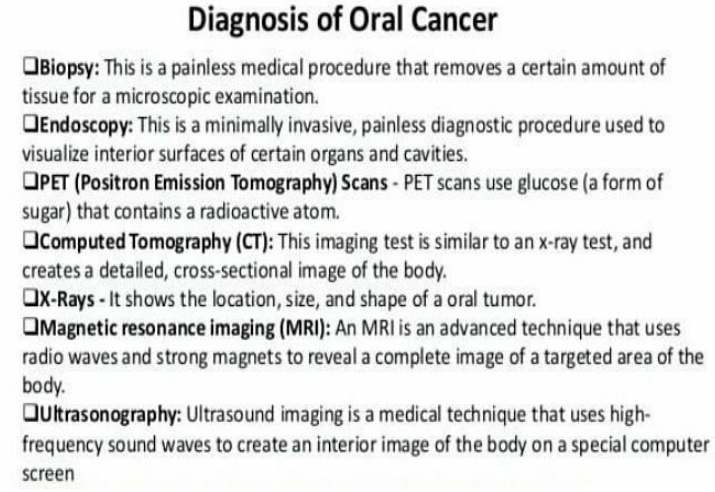
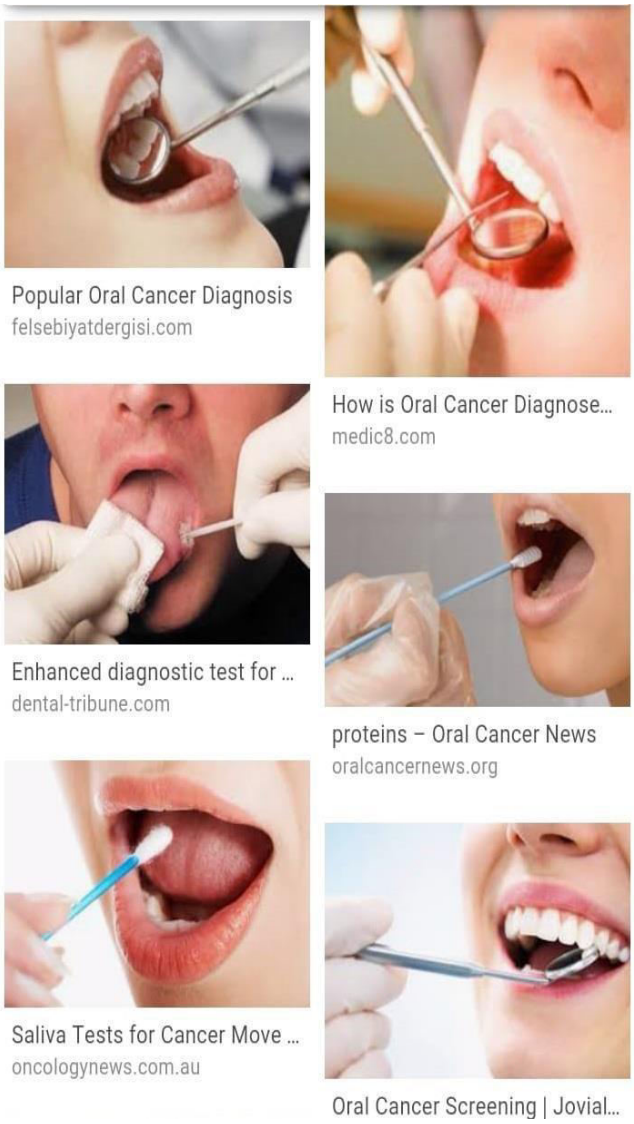


Fig 8- Diagnosis of oral cancer

Fig 9- Diagnosis



Treatment:

Fig 10- Drug use in oral cancer

Recent Innovations in Oral Cancer early Detection and Cure

The treatment modalities for cancer especially head and neck cancer depends on the TNM staging assessment, metastasis, Biopsy results, radiotherapy, chemotherapy. It is well known fact that Prompt detection of head and neck squamous cell carcinoma is vital to successful management. Microsatellite analysis of the DNA of exfoliated mucosal cells and pretreatment oral rinse helped to detect cancer.[11] The outlook for oral chemotherapy is positive. Having been for many years the poor relation, oral chemotherapy is poised to become a major force. Encouraging clinical trial results indicate that for the first time many of the oral anti-cancer drugs in development are actually better drugs rather than pale imitations of i.v. treatments. Many of these new agents, especially signal transduction inhibitors or angiogenesis inhibitors, will be available only as oral treatments with an i.v. dosage form either impractical or inappropriate. Oral treatment will reduce the number of inpatient and out-patient hospital visits with their associated medical and nursing administrative costs, avoid the expense of disposables (e.g. infusion equipment, pumps) and decrease the pharmacy workload. Chemotherapy costs account for only a small proportion of the direct cost of cancer care so it should be possible to set increased drug costs against the substantial

savings that will be made elsewhere. Drug budgets are, however, easily identified and this process will be easier in some countries than in others. This article reviews the diagnostic aids, medium of choice for investigation both invasive and noninvasive, role, validation and prevalidation of salivary biomarkers,[11] drug delivery systems, metabolomics recent developments in oral chemotherapy, both of traditional cytotoxics and novel, targeted agents, from the viewpoint of patients, physicians, and health-care providers.[11]

MANAGEMENT OF ORAL CANCER

Oral cancer is a serious disease that is on the increase. The most pressing need is early recognition and referral for specialist treatment. Too many cases present with advanced tumours. Radiotherapy and surgery remain the primary modalities of curative treatment, but understanding of tumour pathology and developments in surgical and radiotherapeutic technique have combined to produce a rational approach to management. In many instances 'radical' methods of

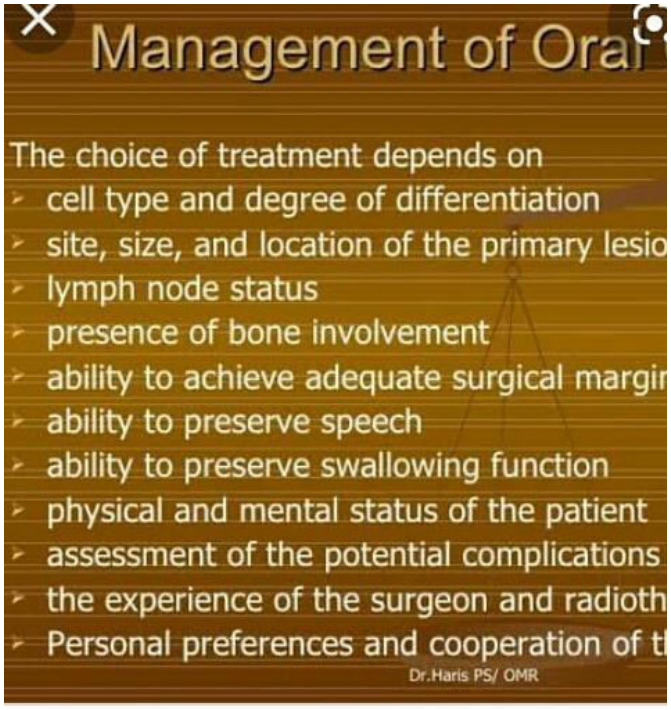


Fig 11- Management of oral Cancer

RADIOTHERAPY & CHEMOTHERAPY TREATMENT

Postoperative Radiotherapy ± Chemotherapy

-Risk profile and indications for adjuvant treatment Primary surgery is the traditional approach for resectable OSCC in most centers. For patients with unfavourable pathological features, postoperative radiotherapy (PORT) or postoperative concurrent chemo-radiotherapy (POCRT) have been shown to improve LRC and survival in several clinical trials . General indications for PORT include: T3 or T4 tumor; compromised surgical resection margins (<5 mm from the inked surface of the specimen); presence of lympho-vascular invasion (LVI) and/or peri-neural invasion (PNI); and positive lymph nodes with or without extracapsular invasion (ECE) (2,5). The presence of multiple risk factors is common in OSCC. To understand the loco-regional recurrence risk profile, Langendijk, et al. studied 801 head-and-neck cancer patients (73% of whom had OSCC) who underwent PORT in 1985-2000. They did not include their most favourable cases that did not require PORT in the report. A recursive partitioning analysis addressing loco-regional recurrence stratified patients into three risk groups: a) intermediate-risk: clear resection margins and no ECE, b) high-risk: T1, T2 and T4 tumor with close or positive margins or one pathological positive lymph node with ECE, c) very high-risk: T3 tumor with close or positive surgical margins or multiple pathological positive lymph nodes with ECE or an N3 neck (6). For the latter two groups, the 5-year LRC with PORT was unsatisfactory (78% and 58%, respectively). The authors concluded that more intensive approaches, such as POCRT with concurrent chemotherapy should be considered for these two sub-groups. Pathological stage I-II disease with sufficiently clear resection margins is generally considered low-risk and does not require PORT (7). Studies suggest that PNI alone appears to be nonpredictive for recurrence (8,9). However, the presence of LVI or microscopic tumor foci in muscle increased the risk of recurrence and PORT should be considered. Tumor thickness, or alternative synonyms such as “depth of invasion” or “tumor depth”, has been consistently identified as a predictor for cervical lymph node metastasis (10). Recent studies have shown that

pathological tumor thickness ≥ 4 mm combined with poorly differentiated pT1-2N0 OSCC tumors are associated with poor regional control and such patients may benefit from PORT (11). The effectiveness of PORT for T1 or T2 primary with completely resected N1 disease is yet to be confirmed. Currently, there is an on-going European clinical trial evaluating the effectiveness of PORT in pT1-T2pN1 oral cavity and oropharyngeal cancers with clear resection margins . The presence of microscopic positive margins and/or

ECE is considered high-risk for recurrence. The addition of chemotherapy to PORT for these patients resulted in a 13% absolute reduction in loco-regional relapse at 5-years in the EORTC trial 22931 reported by Bernier et al. and a 10% absolute reduction at 2-years in the

Radiation-related Toxicity ± Chemotherapy

Radiotherapy with or without chemotherapy to enhance disease control is associated with radiation-induced toxicity. The acute toxicity includes grade 2/3 oral mucositis and dysphagia. The incidence is generally high, especially with bilateral irradiation, though is usually selflimiting [12] . Common late toxicities include ORN, xerostomia, and dysphagia . The general impression of practitioners is that toxicity is enhanced by the use of concurrent chemotherapy with PORT. For example, the combined severe acute and late toxicity frequency in the RTOG trial 9501 reported by Cooper, amounted to 46% vs. 78% for PORT vs. POCRT respectively. For reasons that are not immediately apparent, the toxicity profile seems less clear in EORTC 22931; for example the severe muscular fibrosis rate was higher for POCRT (10% vs. 5%) but severe xerostomia was lower (14% vs. 22%) . These observations underline the need to prospectively include comprehensive toxicity data collection in future trials. Spontaneous ORN is dosedependent and related to the volume of mandible receiving radiotherapy beyond 50~60 Gy . The incidence is generally low (<15%) with conventional conformal radiotherapy (48) with the exception of one unexpected retrospective report in the recent literature [12] .

Intensity modulated radiotherapy (IMRT) is a newer method of radiotherapy that uses intensity-modulated beams that can provide multiple intensity levels allowing concave dose distributions and dose gradients with narrower margins than those possible using traditional radiotherapy . The incidence of ORN can be further reduced significantly by minimizing the percentage of mandibular volume exposed to >50~60 Gy using IMRT . In addition, careful oral dental hygiene and smoking cessation are also important for preventing ORN. Xerostomia rates are high with conventional radiotherapy when salivary gland and oral cavity mucosa are inevitably included in the radiation field. IMRT has demonstrated an ability to enhance salivary sparing to reduce the rate of permanent xerostomia without compromising disease control . It is also capable of reducing the rate of severe dysphagia compared to conventional radiotherapy[12] ..

ORAL CANCER PATIENT SURVEY

Nargis Dutta Memorial, Cancer Hospital, Barshi, Dist-Solapur

Patient Name:-

1) Bharat Baliram Netake (37) – Tongue Cancer

Address: At Post Barshi Dist-Solapur

2) Harun Babulal Mujawar (58) - Cheek Cancer

Address:Agalgoan , Dist-Solapur

3) Dashrath Ramchandra Mane (48) - Throat Cancer

Address: yevati, Dist –Osmanabad.

4) lilabai Pandurang Kamble (67)- Lip Cancer

Address: Mankeshwar , Dist –Solapur

5) Ramesh Balasaheb Thorat (42) – Neck Cancer

Address: Bori , Dist –Osamanabad

6) Amol Ramdas Awad (46) – Tongue Cancer

Address: Ranjan Gaon , Dist –Osamanabad

7) Ramdas Gopinath Shikre (46) – Tongue Cancer

Address: kari , Tq –Barshi, Dist Solapur

Fig 12 – Survey of Cancer Hospital , Barshi

CONCLUSION

Oral cancer refers to the malignancies that occur in the head and neck, the tongue, the lip, the oral mucosa and the other oro pharyngeal structures. Salivary biomarkers play a vital and noninvasive method of early detection of cancer. The changes in cellular level of DNA, the micro RNA profile, messenger RNA and the salivary biomarkers are discussed in detail. Salivary Metabolomics focuses on cellular functions. The recent innovations like micro satellite analysis of exfoliated cells during pretreatment rinse and electrochemical sensor have been beneficial in the treatment modalities in addition with the traditional modalities. Oral cancer has a low five year survival rate. Early detection of oral cancer could reduce the mortality and morbidity associated with this disease.

Saliva, which can be sampled non-invasively and is less complex than blood, is a good potential source of oral cancer biomarkers.[1]

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