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Review Article | **Oral and Maxillofacial Surgery**

Oral Cancer: Updated Review of Literature

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Abstract

Oral cancer considered one of the most 10 cancer among world population [1]. In the period between 1995- 2015 Saudi cancers registries detect 172,424 cancer cases, 3184 were oral cancer cases. 1.5 Per 100000 for female population and 1.4 per 100000 among male population, the majority of cases are from jazan region [2, 3]. The aim of this review is to explain the basic and essential aspects of oral cancer focusing on squamous cell carcinoma , starting from its definition to epidemiology in Saudi Arabia as well world wild as well addressing carcinogenesis , potential malignant diseases , and premalignant lesions. As oral cancer is preventable disease, prevention will be addressed as well.

Keywords: oral cancer, squamous cell carcinoma, oral cancer staging, Oral premalignant lesions.

INTRODUCTION

Oral cancer, particularly for oral and maxillofacial surgeons is very important to understand. It is among the top 10 cancer despite the improvement of research in term of prevention and therapeutic ways. Due to delayed in clinical detection and early diagnosis, oral cancer cases presented with regional lymph node involvement and distance metastatic (43% and 10%) of all oral cancer cases respectively. The goal of this paper is explain and deeply understanding the basic clinical, histological, and therapeutic aspects of oral cancer [1].

Definition and Classification

Oral cancer is a malignant lesion recognized as oral squamous cell carcinoma. 90% of cancers that arising in the oral area are histologically from squamous cell. Oral cavity composed fromthe buccal mucosa, lips, upper and lower alveolar ridges, gingival tissues, retromolar area, hard and soft palate, and floor of the mouth [1, 4].

Anatomy of the Oral Cavity

The Superior border of the oral cavity starting from the vermillion border to the junction of the hard and soft palates. Where is the inferior border starting from the vermillion border to the circumvallate papillae of the tongue? The lateral border starting from the buccal mucosa to the tonsillar pillars [1].

The tongue is a mass of muscle that is almost completely covered by a thick mucous membrane. The functions of the tongue are taste sensation, mastication, deglutition, and articulation. Embryonically the tongue appear at 5 weeks of gestation from the first branchial arch. The first developmental structures are two lateral lingual swellings and one median swelling known as tuberculum impar. Two weeks later those structure will merge to form the anterior two third of the tongue.

The tongue has three surfaces: tip, body which composed of (ventral, and dorsal surfaces), and the base. The body of the tongue is separated into right and left part by the median sulcus, and the body of the tongue and the base is separated by the terminal sulcus which is a V-shaped sulcus with a foramen on its tip called foramen cecum. At the base of the tongue lingual tonsils located, they form the inferior part of the Waldeyer's ring [4].

Over the tongue surfaces, Four different types of lingual papillae: circumvallate (vallate), foliate, filiform, and fungiform.The circumvallate papillae are flat, prominent papillae that are surrounded by troughs, approximately eight to 12 located directly anterior to the terminal sulcus. Circumvallate papillae contain 250 taste buds. foliate papillae located on the lateral surfaces of the tongue and contain 1000 taste buds, where as thefiliform papillae are located over the dorsal

surface of the tongue and they are the most abundant papillae in the tongue, however they are not participate in taste sensation. Fungiform papillae are mushroom shaped papillae located at the tongue tip and the lateral surfaces as well, they are around 200-300 papillae and contain 1600 taste buds. The ducts of the lingual glands of von Ebner secrete lingual lipase and participate into the process of lipolysis.

The taste buds can recognize all the different taste: bitter, acid, sweet, umami and salt. They contain taste receptors, basal cells, and edge cells. When food molecule binds to this receptors, they start to depolarized leading to calcium influx and releasing of neurotransmitter followed by stimulation of the suppling nerve, In the anterior two thirds of the tongue, the chorda tympani branch of the facial nerve (cranial nerve VII) is stimulated, and in the posterior third The lingual-tonsillar branch of the glossopharyngeal nerve (cranial nerve IX) is responsible for special sensation.

Muscles of the tongue can be divided to four intrinsic and four extrinsic muscles. Intrinsic muscles alter the shape of the tongue; whereas extrinsic muscles alter its position. The extrinsic muscles of the tongue are the genioglossus, hyoglossus, styloglossus, and palatoglossus. The hypoglossal nerve provides the motor innervation to all muscles of the tongue except the palatoglossus, which is supplied by the pharyngeal plexus [4].

The blood supply of the tongue and floor of the mouth derived from braches of the lingual artery (dorsal, sublingual, and deep lingual). The venous drainage of the tongue via lingual vein which drain into retromandibular vein which join at the end with common facial vein [1, 4].

Epidemiology

Cancer considers one of the most leading causes of death all over the world. In the developing world it is considering as the second – leading cause of death. The International Agency for Research on Cancer (IARC) stated that in 2018, 354, 864 new cases of oral cancer were reported, representing 2% of all cancer cases [2].

Around 66% of new oral cancer cases are diagnosed in developing countries, 25% of them are detected in Sri Lanka, India, Pakistan and Bangladesh [3]. In Europe only 15500 oral cancer cases were diagnosed, representing 5.5% of all cancer cases reported in 2004. In 2019, American cancer society reported 53000 new oral cancer cases, 10860 died due to this cancer. In Arab countries, the prevalence of oral cancer is concentrated between western and southeast Asia. While this type of cancer is relatively uncommon across Arab gulf countries [2, 3, 5].

A study analysis conducted by M. Alshehri, used a Data from the Saudi Cancer Registry from its establish in 1994 to 2015, stated that 172,424 cancer cases were detected at this period. 3184 were oral cancer. The prevalence of oral cancer among population is 2.9 per 100000 people. For females, it was 1.5, and for males, it was 1.4. The highest number of cases was detected in Jazan region was the lowest were in Hail region. 75% of cases diagnosed after the age of 50 years [3].

Etiology and Risk Factors

Smoking considered one of the causative factors that lead to oral cancer. Chewing Tobacco significantly has been linked to development of Squamous cell carcinoma of the buccal mucosa (site of chewing). Around 85% of patients with oral cancers are smokers [5]. Studies by Ibrahim *et al.*, found that consuming shamma increased the risk fordeveloping oral cancer 29-fold [3], another study have shown that the oral cancer increase by 12 folds among smokers when they compared it to non-smokers [6, 7].The risk for oral cavity cancer development in nonsmokers is thought to be between 5% and 30%.

Alcohol as well is another risk factor for oral cancer, People who use tobacco and alcohol, these risk factors appear to be synergistic and result in a multiplicative increase in risk, 30 to 36 times higher for people who smoke and drink heavily [5]. On the other hand, Epidemiologic studies suggest that the intake of vitamin A, β-carotene, and α - tocopherol may reduce the risk of developing oral cavity cancers [8].

Mechanisms of Carcinogenesis

Developing of oral cancer is processed in multiple steps, starting from exposure to risk factor until clinically detection lesion. Smoking contains agents that may act as mutagens. Also, tobacco smoke extract has been shown to activate the epidermal growth factor receptor (EGFR) in vitro and EGFR activation has been shown, in turn, to increase the production of prostaglandins, including PGE2 which may act in a positive feedback fashion by increasing EGFR signal transduction. Cyclin-D1 is frequently overexpressed in head and neck cancer and increased cyclin-D1 activity is a downstream event triggered by EGFR activation [8].

Genetic alterations that are present early in the course of carcinogenesis are mutations or deletions of chromosome 3p and 9p. Telomerase activation also occurs early in carcinogenesis. Mutations or deletions at chromosome 17p (involving the p53 tumor suppressor gene), and chromosome 13q and chromosome 18q generally are seen later in the process. Patients whose tumors contain HPV mRNA have a significantly lower rate of deletions of chromosomes 3p, 9p, and 17p, suggesting an alternate molecular mechanism in these patients. The viral proteins E6 and E7 have been shown

to cause deregulation of the cell cycle by inactivating p53 and retinoblastoma protein, which may be the mechanism of HPV-mediated carcinogenesis [8].

Tumor Biology

Studies have shown that oral cancers arise from a premalignant progenitor cell line, starting from this cell line a process called clonal expansion takes place to develop. Clonal expansion continues with the cumulative genetic alterations and changes until frank malignancy occurs [9].

Genetic alteration play a significant role for development of oral cancer ,The two major genetic alterations include inactivation of tumor suppressor genes (TSGs) and the activation of proto-oncogenes. Alterations occur in early mutagenesis, within the change from normal mucosa to premalignant mucosa. Loss of chromosome region 9p21 is found in 70% to 80% of all oral carcinoma [10].

Viral infection has been linked to development of oral and oropharyngeal carcinoma. The most common virus associated with the development of oral and oropharyngeal carcinoma is the human papillomavirus (HPV). HPV 16 and HPV 18 are most associated with oropharyngeal SCCs. The mechanism of HPV mutagenesis appears to differ from those of tobacco and alcohol abusers. HPV viral oncoproteins may degrade and inactivate TSGs rather than mutating them.HPV 16 and 18 encode two early gene proteins, E6 and E7, which are thought to induce the genetic and cellular changes. E6 is thought to inactivate or degrade p53, thereby arresting cellular division. E7 is believed to be involved with the degradation of Rb, which also disrupts cell cycle regulation. There is evidence supporting that HPV-mediated oral and oropharyngeal carcinomas differ from “traditional” carcinomas. Proliferative verrucous hyperplasia and verrucous carcinoma may also be associated with HPV-related cellular changes. Patients with HPV-positive carcinomas are likely to be younger, nonsmokers, have early lymph node metastasis, and have a social history of more frequent heterosexual and orogenital sexual contacts. Oropharyngeal, in particular tonsillar, carcinomas have the highest rates of HPV DNA detection ranging from 45% to 67% of those studied compared with oral cavity lesion whereas HPV DNA has been found in 12% to 18% [9-11].

Diagnosis

Diagnosis and detection of oral cancer start from systemic and well organized approach , from history taking, reporting significant risk factors, and focused head and neck examination. Followed by requesting proper radiological investigations, and obtained the final diagnosis after biopsy results. In order to be systematic and well organized during clinical evaluation of cancer patients, we need to start from primary lesion assessment, regional assessment, full radiological workup, and biopsy taking for final diagnosis and staging.

Primary Tumor Assessment

Physical examination should be performed on every patient with specific emphasis on the head and neck exam (inspection, palpation, otoscopic exam, indirect laryngoscopy, and when indicated nasopharyngolaryngoscopy) and a neurological exam with emphasis on cranial nerves V, through XII. The most common presenting complaint of patients with tongue tumors is a sore or lump. Cancer of the tongue mucosa may present as an indurated ulcer with raised edges or as an exophytic growth. Bleeding from the surface of the lesion is a characteristic of malignancy and immediately raises suspicion for a neoplastic process.

Patients may present with myriad complaints such as a non-healing sore in the mouth (>2 wk), loosening of teeth, ill-fitting dental prosthesis, trismus, otalgia, or weight loss. Examination of the oral cavity should include removal of all dental appliances and use of a dental mirror for indirect evaluation of the nasopharynx and hypopharynx.Bimanual palpation is critical to assess any involvement of structures such as the deep musculature of the tongue, floor of the mouth, buccal mucosa, salivary structures, or bony mandibular structures.Assessment of the lateral tongue and posterior pharynx is assisted by anterior and lateral traction on the tongue with cotton gauze [12].

A synchronous tumor is described as a second histologically confirmed malignancy. This malignancy must be a distinct and geographically separated by normal non-neoplastic mucosa, and not of metastatic origin from the index lesion. It must also be discovered at the time of initial tumor evaluation. If the second primary tumor is discovered at a later time, it is considered a metachronous tumor (warren and gate discerption).

Assessment of Regional Metastases

The head and neck lymph nodes need to be carefully palpated. Standing behind the patient and starting by palpate the lower cervical lymph nodes moving up to the submental and submandibular group to the posterior occipital group.The thyroid gland should also be palpated for masses, enlargement, or tenderness. The trachea should be inspected for any deviation or fixation.

The presence of a single lymph node with metastatic disease reduces the patient’s 5-year survival by 50%. In turn, the presence of extracapsular spread decreases this survival by another 50%. Analysis showed that lymph nodes greater than 3 cm had a 73.7% chance of extracapsular spread; 2 to 3 cm, a 53.3% chance; 1 to 2 cm, a 44.3%; and less than 1 cm, a 28.8% chance [12].

Neck is divided into six "surgical levels" based on anatomic structures over 300 lymph nodes in the neck. Level I include the submental and submandibular nodal groups.

1. Level IA, the submental group, is bounded by the hyoid bone inferiorly, the mandibular symphysis superiorly, and the anterior bellies of the digastrics muscles laterally.
2. Level IB, the submandibular group, is bounded by the posterior belly of the digastric inferiorly, the mandibular body superiorly, the anterior belly of the digastrics muscle anteriorly, and the stylohyoid muscle posteriorly.

Level II includes upper jugular lymph nodes surrounding the internal jugular vein and adjacent spinal accessory nerve.

1. Level IIA is bounded inferiorly by a horizontal plane made by the inferior body of the hyoid bone, superiorly by the skull base, anteriorly by the stylohyoid muscle, and posteriorly by a vertical plane defined by the spinal accessory nerve.
2. Level IIB is bounded inferiorly by a horizontal plane made by the inferior body of the hyoid bone, superiorly by the skull base, anteriorly by a vertical plane defined by the spinal accessory nerve, and posteriorly by the lateral border of the sternocleidomastoid muscle (SCM).

Level III includes middle jugular lymph nodes surrounding the internal jugular vein. It is bounded inferiorly by a horizontal plane defined by the inferior border of the cricoid cartilage, superiorly by the horizontal plane defined by the inferior body of the hyoid bone, anteriorly by the lateral border of the sternohyoid musculature, and posteriorly by the lateral border of the SCM or sensory branches of the cervical plexus.

Level IV includes the lower jugular lymph nodes surrounding the internal jugular vein. It is bounded inferiorly b the clavicle, superiorly by the horizontal plane created by the inferior border of the cricoid cartilage, anteriorly by the lateral border of the sternohyoid musculature, and posteriorly by the lateral border of the SCM or sensory branches of the cervical plexus.

Level V includes all the nodes in the posterior triangle, the spinal accessory and transverse cervical nodes, and all of the upper, middle, and lowers jugular lymph nodes on the posterior aspect of the SCM.

1. Level VA is bounded inferiorly by the horizontal plane created by the inferior border of the cricoid cartilage, superiorly at the apex found at the convergence of the SCM and trapezius muscles, anteriorly by the posterior belly of the SCM or sensory branches of the cervical plexus, and posteriorly by the anterior belly of the trapezius muscle.
2. Level VB is bounded inferiorly by the clavicles, superiorly by the horizontal plane created by the lower border of the hyoid bone, anteriorly by the posterior belly of the SCM or sensory branches of the cervical plexus, and posteriorly by the anterior border of the trapezius muscle.

Level VI includes the pretracheal, paratracheal, and prelaryngeal or so-called delphian lymph nodes. It is bounded inferiorly by the suprasternal notch, superiorly by the hyoid bone, and laterally by the common carotid arteries. This level is also known as the anterior compartment [12, 13].

Radiological Investigations

Panoramic radiograph considered the first needed head and neck radiograph as it's the most available screening tool in the dental clinic. Chest x-ray considered the minimum radiological investigations to rule out the distant metastasis, however recently pan CT have been used to detect metastasis.

Head and neck CT id one of the most significant and useful tool in the head and neck oncology field.A lymph node is considered abnormal when it is greater than 1.5 cm in the jugulodigastric region or greater than 1 cm in other regions of the neck.Shape has been suggested as a criterion to help distinguish pathologic nodes. The shape of a normal or hyperplastic lymph node resembles a bean; as opposed to round or sphere like metastatic nodes frequently present [14].

MRI is superior to CT in term of detection of soft tissues lesion, however due to its cost and time consuming many clinicians prefer using CT over MRI [15].

Sensitivity of sonography in the detection of cervical lymph node metastasis is 89% to 95% and specificity is 80% to 95%. This specificity can be increased with the use of US-guided fine-needle aspiration (FNA). US-guided FNA is also a useful tool for evaluating a neck mass suspicious for carcinoma when no other primary site can be found (i.e., "unknown primary").

The use of 2-18F-fluoro-2-deoxy-D-glucose (FDG) PET relies on the enhanced metabolic activity of tumoral tissue in the body, of which increased glycolysis is usually a biochemical hallmark. FDG, a radiolabeled glucose analog, is preferentially taken up within tumor cells that exhibit increased glycolysis; they can be detected from the increased signaling in that tissue. A prospective study by Adams and coworkers98 showed a higher sensitivity and specificity FDG-PET (90%, 94%) compared with CT (82%, 85%) and MRI (80%, 79%). Distant metastases increase as the disease progresses and more frequently include the lungs, and to a lesser extent, bone and liver. This is a reason to use

PET/CT for assessing the distant spread of the cancer in patients with disease recurrence of progression. In 10-30% of patients distant metastases are detected at the time of death [14].

Biopsy

Biopsy can be performed in the clinic under local anesthesia, many clinicians prefer to take biopsies from multiple sites (avoid the center because it's usually showed necrotic tissues).

Over 90% of head and neck cancers (including the oral cavity tumors) are SCC. The World Health Organization classifies squamous tumors of the head and neck in different histologic subtypes [16]:

1. Conventional.
2. Verrucous.
3. Basaloid.
4. Papillary.
5. Spindle Cell (Sarcomatoid).
6. Acantholytic.
7. Adenosquamous.
8. Cuniculatum

The most common variants include verrucous, exophytic or papillary, spindle cell (sarcomatoid), basaloid and adenosquamous carcinoma.The characteristics that predict aggressive behavior include perineural infiltration, lymphatic invasion, and tumor extension beyond the capsule of the lymph nodes. Broder's grading system was the first of the systems, which initiated quantitative grading of cancer. This classification system was based on the estimated ratio of differentiated to undifferentiated elements in the tumor. There are four histologic grades based on the amount of keratinization:-

1. Well-differentiated tumor-> 75% keratinization.
2. Moderately differentiated tumor-50-75% keratinization.
3. Poorly differentiated tumor-25-50% keratinization.
4. Anaplastic or undifferentiated tumor-< 25% keratinization [16].

TNM Classification of Tumors of the Head and Neck

Table 1: TNM classification (T for primary tumor) [7, 13]

Tx	**Primary tumor cannot be assessed**
T0	No evidence of primary tumor
Tis	Carcinoma in situ
T1	Tumor ≤ 2 cm in greatest dimension, ≤ 5mm depth of invasion (DOI)
T2	Tumor ≤ 2 cm, DOI > 5mm and ≤ 10 mm or tumor > 2 cm but ≤ 4 and ≤ 10 mm DOI
T3	Tumor > 4 cm in greatest dimension, or any tumor > 10 mm DOI
T4a	Local disease moderately advanced: (Lips) - Tumor invades cortical bone cortical, inferior alveolar nerve, floor of the mouth or skin of the face (chin) (Oral cavity) - Tumor invades only adjacent structures (for example, cortical bone cortical, [mandible or maxilla], extrinsic muscle of the tongue, maxillary sinus or skin of the face)
T4b	Local disease very advanced: Tumor invades masticator space, pterygoid plates, base or the skull and / or encases internal carotid artery

Table 2: TNM classification (N for involved lymph nodes) [7, 13]

Nx	**Region lymph nodes cannot be assessed**
N0	No region lymph nodes metastasis
N1	Metastasis in single ipsilateral lymph, ≤ 3 cm in greatest dimension ENE (-)
N2	Metastasis in single ipsilateral lymph, > 3 cm, but ≤ 6 cm in greatest dimension ENE (-) Metastasis in multiple ipsilateral lymph nodes, none > 6 cm in greatest dimension ENE (-) Metastasis in bilateral o contralateral lymph nodes, none > 6 cm in greatest dimension ENE (-)
N2a	Metastasis in single ipsilateral lymph, > 3 cm, but ≤ 6 cm in greatest dimension ENE (-)
N2b	Metastasis in multiple ipsilateral lymph nodes, none > 6 cm in greatest dimension ENE (-)
N2c	Metastasis in bilateral o contralateral lymph nodes, none > 6 cm in greatest dimension ENE (-)
N3	Metastasis in a lymph node > 6 cm in greatest dimension ENE (-) or metastasis in any node (s) and clinically overt ENE (+)
N3a	Metastasis in a lymph node > 6 cm in greatest dimension ENE (-)
N3b	Metastasis in any node (s) and clinically overt ENE (+)

Table 3: TNM classification (M for metastases) [7, 13]

M0	No distant metastasis
M1	Distant metastasis

Table 4: staging system of oral cancer [7, 13]

staging	T	N	M
0	Tis	N0	M0
I	T1	N0	M0
II	T2	N0	M0
III	T3 T1 T2 T3	N0 N1 N1 N1	M0
IVA	T4a T4a T1 T2 T3 T4a	N0 N1 N2 N2 N2 N1	M0
IVB	Any T T4b	N3 Any N	M0
IVC	Any T	Any N	M1

Treatment Options

The first step in management of oral cancer is tumor board based management. Proper management requires a multidisciplinary team with the following members: head and neck surgeon, dentist, prosthodontist, reconstructive surgeons, medical oncologist, radiation oncologist, speech therapist, rehabilitation therapist, social worker, and psychologist.

The corner stone of oral cancer treatment is surgical intervention, which is surgical resection with safety margin 1-1.5 cm, with or without neck dissection according to lymph nodes involvement.

If regional nodes are positive, cervical lymph node dissection is usually done in the same procedure. Neck dissection must be standardized (i.e. complete anatomical dissections, instead of random biopsies) in these situations to prevent incomplete surgery. Elective neck dissection is recommended for patients who have a oral cavity tumors with a minimum thickness of 4 mm [3], although some researchers believe that tumor thickness of 2-3 mm would be a more appropriate cut off. Supraomohyoid neck dissection is recommended in patients with a clinical stage N0 who are treated surgically. There is evidence of skip metastases through the levels of the neck [7] and in some cases just involving level IV without involving the first levels. Therefore some authors recommend extended supraomohyoid neck dissection [7, 20].

Sentinel lymph node (SLN) biopsy is another new option to standard elective neck dissection for identifying an occult cervical metastasis in patients with an early (T1 or T2) oral tongue cancer in centers where expertise for this procedure exists [18, 19]. Patients who are found to have metastatic disease in their SLN's must undergo a completion neck dissection while those without a positive SLN can be observed with close follow up [19].

Radiation therapy for cancer of the oral cavity may be administered as external beam radiotherapy (EBRT) or interstitial implantation alone. Larger lesions are frequently managed using external beam radiotherapy, which includes the primary site and regional lymph nodes (even if not clinically affected). Radiation therapy with curative intent usually involves daily treatment for 6 to 7 weeks (total dose: 60-70 Gy). Potential Complications of radiotherapy include dry mouth, tissue fibrosis, trismus, bone necrosis, hypothyroidism, and dysphagia [20].

The definitive indications for postoperative radiotherapy are positive margins, multiple positive nodes with metastatic disease, and extra capsular nodal extension. Less certain indications include lymphovascular space invasion, perineural spread, single encapsulated positive lymph node, and thick tumors. Tumors with a thickness between 3 to 9 mm have 44% subclinical node positivity and a 7% local recurrence rate and tumors with a thickness greater than 9 mm thickness have 53% subclinical node positivity and a 24% local recurrence rate [7, 20].

Recently the results of the RTOG-0234 examining concurrent chemoradiotherapy and cetuximab in the postoperative treatment of patients with head and neck squamous cell carcinoma (HNSCC) with high-risk pathologic features was published [21]. The study recruited 238 patients were with stage III to IV HNSCC with gross total resection showing positive margins and/or extracapsular nodal extension and/ or two or more nodal metastases. Patients were randomly assigned to 60 Gy radiation with cetuximab once per week plus either cisplatin 30 mg/m2 or docetaxel 15 mg/m2 once per week. With a median followup of 4.4 years, 2-year overall survival (OS) was 69% for the cisplatin arm and 79% for the docetaxel arm; 2-year disease-free survival (DFS) was 57% and 66%, respectively. DFS in this study was compared with that in the chemoradiotherapy arm of the RTOG-9501 trial, which had a hazard ratio of 0.76 for the cisplatin arm versus control (P=0.05) and 0.69 for the docetaxel arm versus control (P=0.01), reflecting absolute improvement in 2-year DFS of 2.5% and 11.1%, respectively. The delivery of postoperative chemoradiotherapy and cetuximab to patients with HNSCC is possible and tolerated with predictable toxicity. The docetaxel regimen shows favorable outcome with improved DFS and OS relative to historical controls and has commenced formal testing in a phase II/III trial [17, 21].

Follow up Protocol

Follow-up protocols are based on the risk of relapse, second primaries, treatment sequelae, and toxicities includes a history and physical (including a complete head and neck exam; mirror and fiberoptic examinations as clinically indicated every 1 to 3 months for the first year, every 2-6 months for the second year, every 4 to 8 months years 3 to 5, and every 12 months after 5 years. If the neck was radiated, the NCCN guidelines recommend thyroid stimulating hormone (TSH) testing every 6 to 12 months. Smoking and alcohol counseling as clinically indicated [22].

CONCLUSION

Oral cavity cancer requires a multidisciplinary team approach for proper management. Early referral to a specialized center may help for treatment of this cancer in its early stage which will improve outcomes.

DISCLOSURE OF CONFLICT OF INTEREST

None.

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