



Postgraduate Surgery

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VOLUME 2

- Recent Guidelines
- Latest References
- Surgical Tips
- Important Facts
- Points to Remember
- Differential Diagnosis
- Clinical Exam Checklist
- AJCC Staging
- Cautions

Editors

Nilay Mandal
Supriyo Ghatak
Ankan Mondal
Bishan Basu
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Head and Neck Cancers

Shouptik Basu, Ramanuj Mukherjee

INTRODUCTION

Cancers of the head and neck comprise of multiple entities that have a complex management. In the recent years the trend is on the rise especially in the Indian subcontinent. Exposure to avoidable carcinogens are the prime risk factors of this disease. The management is primarily surgical but may require combined modality treatment based on the nature of the disease and its local extent. Surgical resections are to be followed up by complex reconstructions and radiation, chemotherapy or both. It is imperative that these patients be managed in a dedicated unit for the same. There has been a paradigm shift in the management.

SURGICAL ANATOMY OF THE LIPS AND ORAL CAVITY

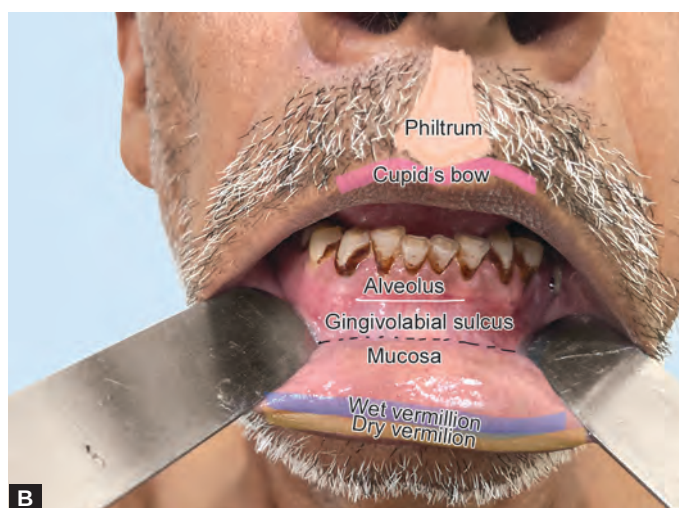
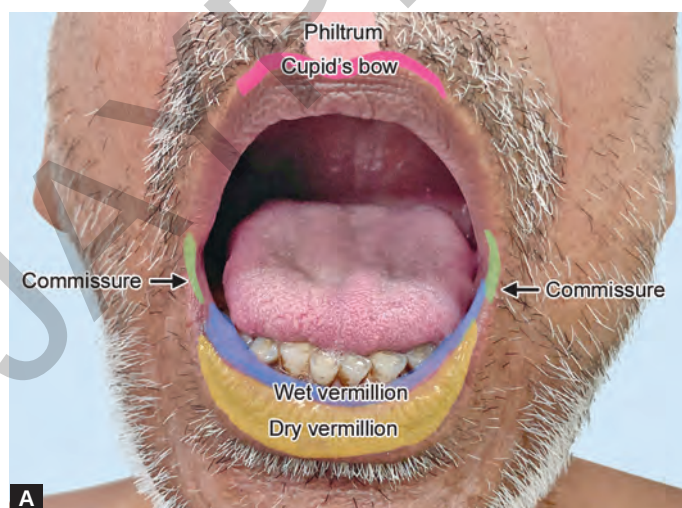
The surgical anatomy of the lip is very unique, the facial skin terminates at the dry vermilion (keratinized stratified squamous epithelium, and it lacks sebaceous glands).

The wet vermilion continues as the buccal mucosa inside the mouth. Both the lips meet on the either side in the commissures. The majority of the lip is formed by the orbicularis oris muscle. The lip develops fusion of the medial, lateral nasal processes and the maxillary processes. The lower lip is formed by the mandibular process. The labial arteries are derived from the facial artery and play a paramount importance during reconstruction (**Figs. 1A and B**). The nodal spread from the upper lip is the submandibular nodes, and from the lower lip spreads to the submandibular, submental and subdiaphragic nodes.

The oral cavity begins at the lips and ends at the anterior surface of the faucial arch. It is lined by squamous epithelium and contains minor salivary glands.

The anatomical subsites of the oral cavity are as follows (**Fig. 2**):

- Mucosal lip (upper and lower)
- Tongue (anterior two-thirds)
- Floor of mouth
- Gingiva (upper and lower)
- Buccal mucosa



Figs. 1A and B: Surgical anatomy of the lip.

- Retromolar trigone (portion of mucosa that lies behind the third molar tooth covering the anterior ramus of the mandible)
- Hard palate

Box 1 shows facts about lip and oral cavity malignancies.

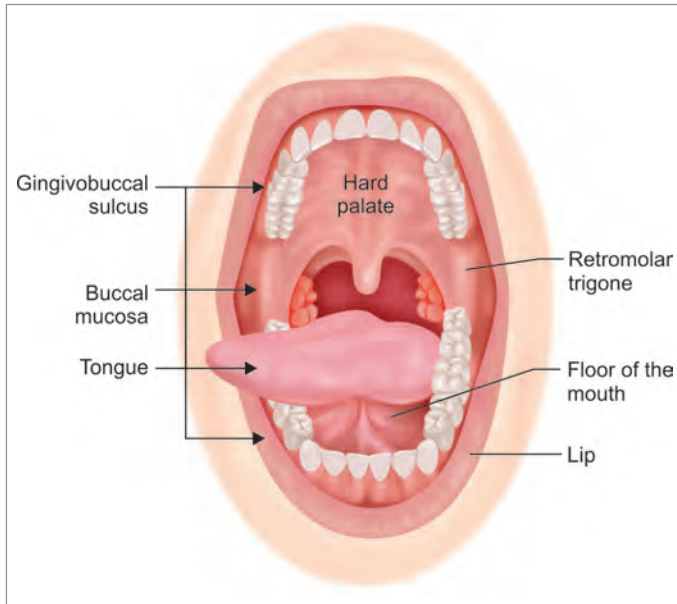


Fig. 2: Anatomic sites within the oral cavity.

BOX 1: Facts about lip and oral cavity malignancies.

- The most common sites of cancers are in the lower lip than the upper lip
- The soft palate is not a part of the oral cavity proper
- Most common site of oral cavity cancers in the West—lateral tongue
- Most common site of oral cavity cancers in India—gingivobuccal sulcus

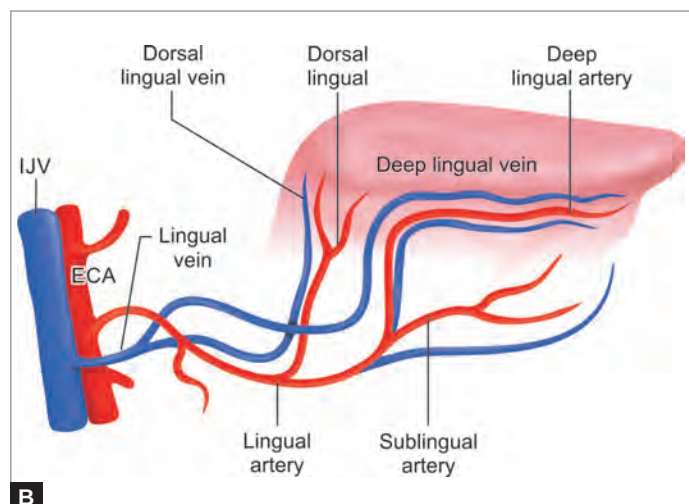
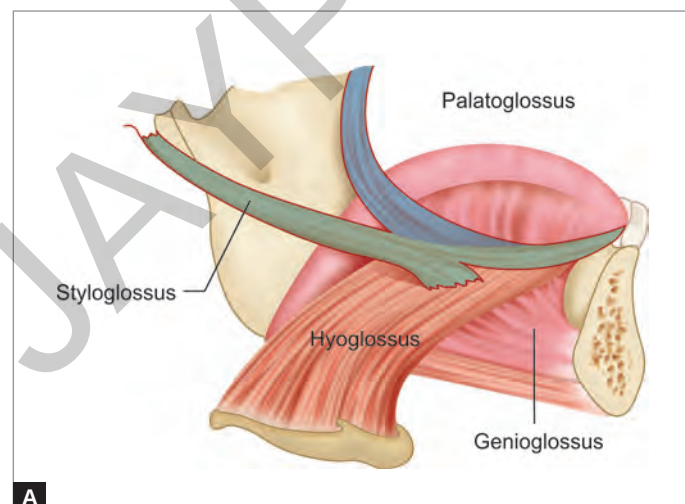
SURGICAL ANATOMY OF THE TONGUE

The freely mobile portion of the tongue is called the oral tongue and it lies anterior to the circumvallate papillae. It is divided into the tip, the lateral border(s), the dorsum, and undersurface (ventral surface).

The tongue aids in speech and mastication. The dorsum of the tongue is covered by thick epithelium, which has fungiform, filiform and circumvallate papillae, this overlying mucosa covers the intrinsic muscles of the tongue. The mucous membrane covers the ventral surface and it is attached with the floor of the mouth by the frenulum. The tongue has four paired extrinsic muscles of the tongue namely—(1) genioglossus, (2) hyoglossus, (3) styloglossus and (4) palatoglossus (**Fig. 3A**). The motor nerve supply to muscles of the tongue is via the hypoglossal nerve except the palatoglossus which is supplied by the pharyngeal plexus. The lingual nerve (branch of mandibular division of the trigeminal nerve) supplies the sensory innervation to the oral tongue, floor of the mouth and lower gingiva. The special (taste) sensation of the oral tongue is supplied by chorda tympani, branch of the facial nerve. The blood supply is by paired lingual arteries (**Fig. 3B**). Lymphatic drainage of the midline, lip and base of the tongue may occur bilaterally, mandates the surgeon to address the neck bilaterally during surgery.

SURGICAL ANATOMY OF THE PHARYNX

The pharynx is grossly divided into the nasopharynx, oropharynx and the laryngopharynx. The nasopharynx is situated behind the posterior nares and extends below to the soft palate. The Eustachian tubes form the lateral ends and the sphenoid bone forms the roof. The oropharynx extends from the hard palate to the hyoid. Anteriorly, it starts from the fauces. The hypopharynx is interspersed in between the oropharynx and the upper esophagus. The larynx lies anterior to it. The cricoid cartilage (corresponding



Figs. 3A and B: (A) Extrinsic muscles of the tongue; (B) Vascular supply of the tongue.

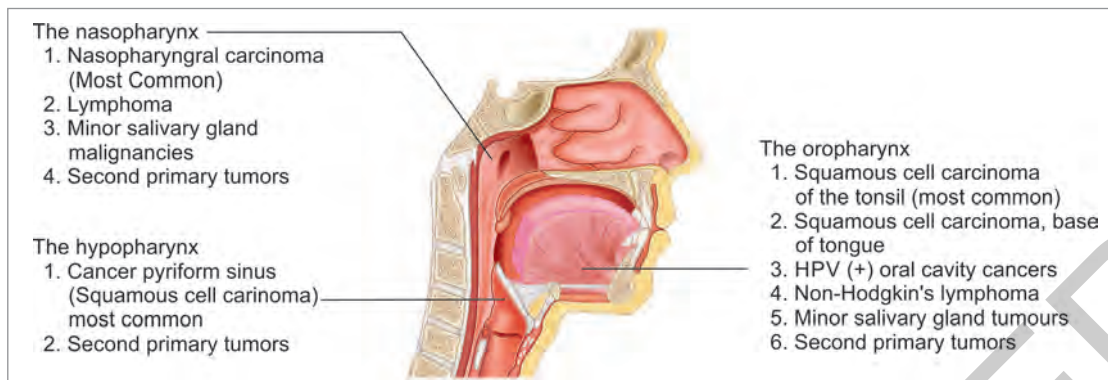


Fig. 4: Parts of the pharynx with various cancers in these areas.

BOX 2: Slaughter's hypothesis of "field cancerization" and the concept of second primary tumors.

- Oral cancer develops in multifocal areas of precancerous change
- Abnormal tissue surrounding the tumor, and their presence postsurgery
- Oral cancer often consists of multiple independent lesions that sometimes coalesce

Applicability: Oral cavity, oropharynx, and larynx; lung; esophagus; vulva; cervix; colon; breast; bladder and skin

to 6th cervical vertebra forms the lower limit of the pharynx). The common cancers arising from these areas are summarized in **Figure 4**.

Slaughter hypothesis of field cancerization (1953) is given in **Box 2**.

APPLIED ONCOLOGICAL ANATOMY OF THE NECK NODES

The lymphatics of the head and neck is two layered and separated by the deep cervical fascia. The superficial lymphatics drain into suboccipital, preauricular and postauricular, prevascular facial, and external jugular lymph nodes. These lymph nodes eventually drain into the deep jugular lymph nodes.

Primary-echelon lymph nodes that drain from a specific site in the head and neck region. Thus, the location of a palpable metastatic lymph node often may indicate the potential site of a primary tumor.

LEVELS OF THE NECK

The conventionally used description of the levels of the neck first was proposed from the Memorial Sloan Kettering Cancer Center in New York. The American Academy of Otolaryngology-Head and Neck Surgery has modified the above system by subdividing levels I, II,

and V into A and B categories for each of these levels. The **Figures 5A to D** and **Table 1** summarize the levels of the neck nodes—summarized below are the common primary lymphatic drainage sites, of various primaries.



Important Notes

- Primary carcinomas of the lateral border of the tongue have a somewhat higher risk of involvement of lymph nodes at level IV, particularly if there is already metastatic disease present at levels II or III.
- Sometimes there may also be "skip metastasis", in these cases.

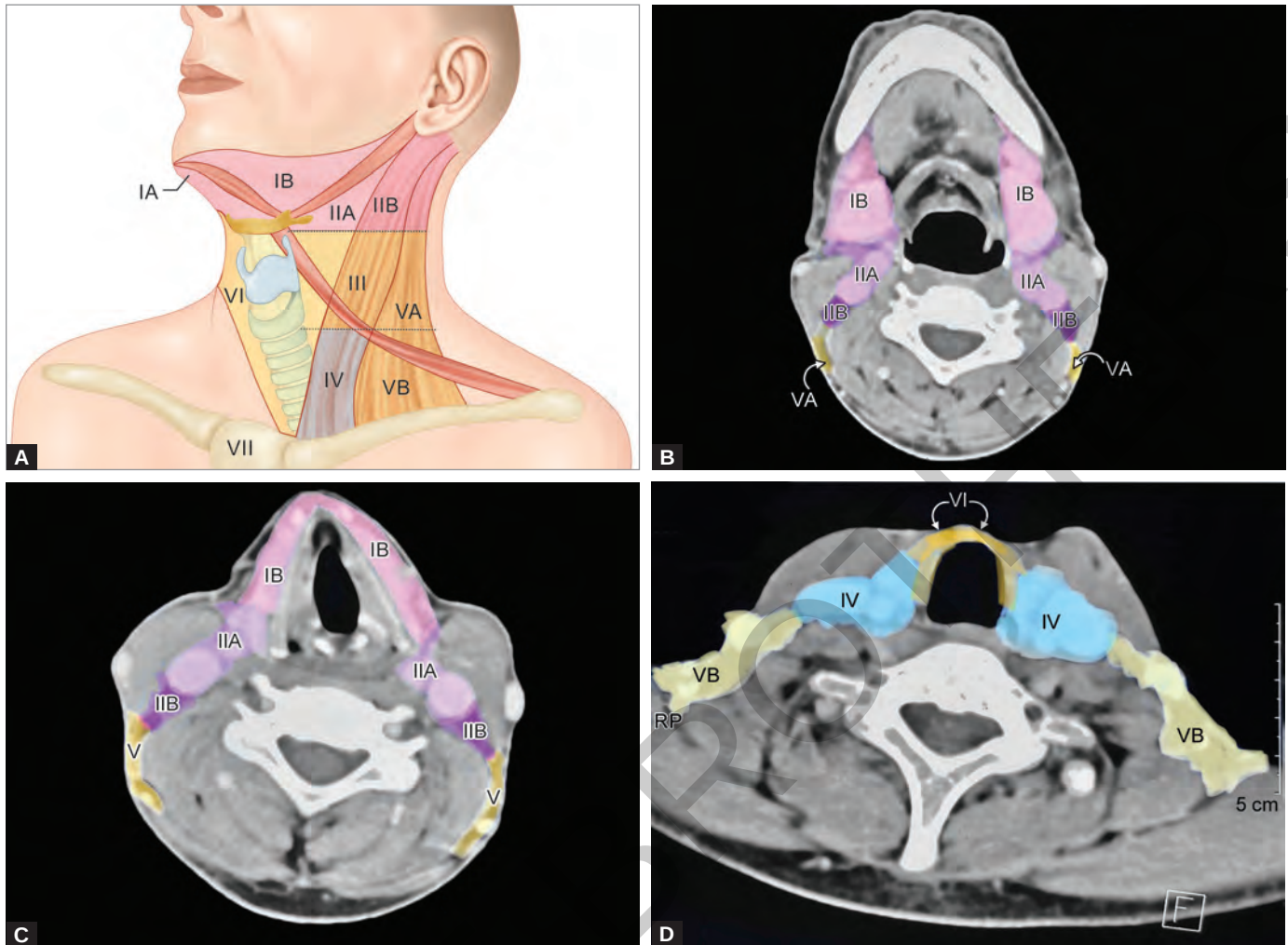
EPIDEMIOLOGY: WHAT IS THE CURRENT INDIAN SCENARIO?

Oral cavity and lip malignancies are the disease of the Indian subcontinent and have multifactorial etiology. The data pooled from GLOBOCAN reveal the incidence rate is 64.2%, with a 42% of new cases in the year 2018 and a mortality rate is 73.3%. Lip and oral cavity cancers account for 11.54% of all new cases in India and 10.16% of all deaths for both sexes. Lip and oral cavity cancer is the most prevalent cancer in men, accounting for 92,011 new cases (16.1%), while it ranks fourth in women. The incidence is maximum in the East Khasi Hills of Meghalaya due to the prevalence of Tobacco usage followed by Ahmedabad (Gujarat).^{1,2}

ETIOLOGY OF ORAL CAVITY CANCERS

The origin of oral cavity malignancies is multifactorial. They can be broadly divided into two headings, namely *proven carcinogens*, where there is definite evidence of the causation factor and the disease and *probably carcinogens* where the role is established but the evidence is not robust. The summary of the causation factor and the mechanism of carcinogenesis are summarized in **Table 2**.

There are some genetic disorders that act as predisposing factors for oral cavity cancers. They are summarized in **Box 3**.



Figs. 5A to D: (A) Levels of the neck; (B to D) Radiological levels of the neck.

TABLE 1: Levels of the neck, its boundaries, and the common structures present in each level.

Level of neck	Name	Superior	Inferior	Anterior	Posterior	Commonly involved in
IA	Submental	Symphysis of the mandible	Body of the hyoid	Anterior belly of the digastric	Anterior belly of the digastric	- Floor of mouth, anterior oral tongue, anterior mandibular - Alveolar ridge - Lower lip
IB	Submandibular	Body of the mandible	Posterior belly of the digastric	Anterior belly of the digastric	Stylohyoid muscles	- Oral cavity - Anterior nasal cavity, skin - Soft tissue structures of the midface - Submandibular gland
IIA	Upper jugular	Skull base	Horizontal plane at the level of the hyoid	Stylohyoid muscles	Vertical plane defined by the spinal accessory nerve	- Oral cavity - Nasal cavity - Nasopharynx - Oropharynx - Hypopharynx - Larynx - Parotid gland
IIB		Skull base	Horizontal plane at the level of the hyoid	Vertical plane defined by the spinal accessory nerve	Lateral border of the sternocleidomastoid muscle	

Continued

Continued

Level of neck	Name	Superior	Inferior	Anterior	Posterior	Commonly involved in
III	Middle jugular	Horizontal plane at the inferior border of the hyoid	Horizontal plane at the inferior border of the cricoid	Lateral border of the sternohyoid muscle	Lateral border of the sternocleidomastoid muscle	• Oral cavity • Nasopharynx • Oropharynx • Hypopharynx • Larynx
IV	Lower jugular	Horizontal plane at the inferior border of the cricoid	Clavicle	Lateral border of the sternohyoid muscle	Lateral border of the sternocleidomastoid muscle	• Hypopharynx • Thyroid • Cervical esophagus • Larynx
VA	Posterior triangle	Apex of the convergence of the sternomastoid and the trapezius	Horizontal plane at the inferior border of the cricoid	Posterior border of the sternomastoid	Anterior border of the trapezius	• Nasopharynx • Oropharynx • Cutaneous structures of the posterior scalp and neck
VB	Posterior triangle	Horizontal plane at the inferior border of the cricoid	Clavicle	Posterior border of the sternomastoid	Anterior border of the trapezius	
VI	Anterior compartment	Hyoid bone	Suprasternal notch	Common carotid artery	Common carotid artery	• Thyroid gland • Glottic and subglottic larynx • Apex of the piriform sinus • Cervical esophagus
VII	Superior mediastinal	Common carotid artery	Innominate artery	Sternum	Trachea, esophagus, and prevertebral fascia	• Thyroid cancer • Esophagus

TABLE 2: Various carcinogens implicated in head and neck cancers with their mechanism of carcinogenesis.

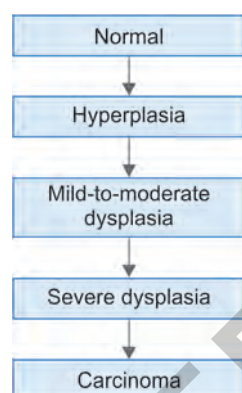
Proven carcinogens	
Agents	Mechanism of carcinogenesis
Tobacco: • Smoking • Smokeless tobacco • Vaping	• Polycyclic aromatic hydrocarbons and act as deoxyribonucleic acid (DNA) adducts • Promote activation of protein kinase
Alcohol	Acetaldehyde acts as noxious promoter of free radical, induces CYP2E1 and promotes change in mitochondrial DNA forming DNA adduct
Paan and Paan products: • Polyingredient oral dip products • Areca nut and quid	Causes local trauma and direct genetic mutation, due to slaked lime that dissolves in the mucosa: • Vasospasm leading to mucosal ischemia • Matrix metalloproteinases (MMP) activation leading to intense desmoplasia
Humanpapillomavirus (HPV)	The ability of HPV early proteins to degrade tumor suppressor gene/proteins in infected cells results in aberrant proliferation
Probable carcinogens	
Ill-fitting dentures	Chronic irritation of oral mucosa or trauma. Risk increases 4 times
Poor nutrition	Absence of vitamin C, flavonoids, polyphenols, and fibers that decrease the antioxidant effects are absent
Poor socioeconomic status	Inability to attain higher education is associated with higher mortality since it affects hygiene, nutrition, addiction habits

BOX 3: Genetic syndromes associated with head and neck cancers.**Genetic syndromes**

- Fanconi anemia
- Bloom syndrome
- Familial atypical multiple mole melanoma (FAMMM)
- Dyskeratosis congenita
- Ataxia telangiectasia
- Li–Fraumeni syndrome
- Xeroderma pigmentosa
- Rothmund–Thomson syndrome

NATURAL PROGRESSION OF HEAD AND NECK CANCERS

Head and neck cancers are predominantly mucosal diseases that arise out of a dysplastic epithelium that has originated from repeated insults to a normal mucosa. The pathway of spread to invasive malignancy has been summarized in the **Flowchart 1**.



Flowchart 1: Natural progression of head and neck cancers to invasive malignancy.

PRECANCEROUS LESIONS OF THE ORAL CAVITY**■ Definition(s) and Terminology**

The World Health Organization (WHO) in 1978 clubbed all the potential malignant lesions of oral cavity and broadly divided this into two categories—(1) precancerous conditions (clinical and histologic); and (2) predisposing conditions.

A precancerous lesion is a morphologically altered tissue in which oral cancer is more likely to occur than in its apparently normal counterpart.

A precancerous condition is a generalized state associated with a significantly increased risk of cancer.

In 2005, a consensus definition used a terminology called *oral potential malignant disorder (OPMD)* as a new terminology being explained as “a group of clinicopathologic diseases of the oral cavity that have arisen in the background of environmental and genetic changes including field changes in the aerodigestive tract mucosa and have a potential for development of invasive malignancy and beyond.” The change clubbed lesions and conditions as disorder with emphasis on field changes that occur.

“Potentially malignant” implies that only a group of patients with any of these mucosal changes will invasive carcinoma. But it does imply that invasive cancers can arise from the same region even with a normal mucosa.

Thus the present applicable definition to be used for OPMD is as “any oral mucosal abnormality that is associated with a statistically increased risk of developing oral cancer” (WHO 2005 consensus definition).

The following has been summarized in **Table 3**.

Most of these lesions will eventually progress to a frank invasive malignancy and the theory was proposed by Bouquet and Gnepp (1991), that is summarized in **Figure 6**.

TABLE 3: Premalignant lesions of the oral cavity.

Potentially malignant disorders (PMDs) (WHO 1978) Pre 2005 WHO consensus		Oral potentially malignant disorders (OPMD) WHO 2005 Bold signifies higher risk of transformation (%) signify transformation risk
Premalignant lesions:	Premalignant conditions:	
• Leukoplakia • Erythroplakia • Proliferative verrucous leukoplakia (PVL) • Vient leukoplakia • <i>Candida</i> leukoplakia • Reverse smoking's palate • Verrucous hyperplasia • Oral verrucous carcinoma • Dyskeratosis congenita	• Lichen planus • Discoid lupus erythematosus • Epidermolysis bullosa • Verruciform xanthoma • Graft-versus-host-disease • Cheilitis glandularis • Xeroderma pigmentosum • Syphilis (third stage) • Plummer–Vinson syndrome	• Leukoplakia (15.6–39.2%) • Erythroplakia (45%) • Palatal keratosis with reverse smoking • Sideropenic dysphagia • Lichen planus (0.4–3%) • Oral submucous fibrosis (7–26%) • Syphilis • Discoid lupus erythematosus • Xeroderma pigmentosum <i>Newly included (2020):</i> • Oral lichenoid lesion (OLL) • Oral graft-versus-host disease (OGVHD) <i>Removed in 2020:</i> • Oral epidermolysis bullosa (OEB)

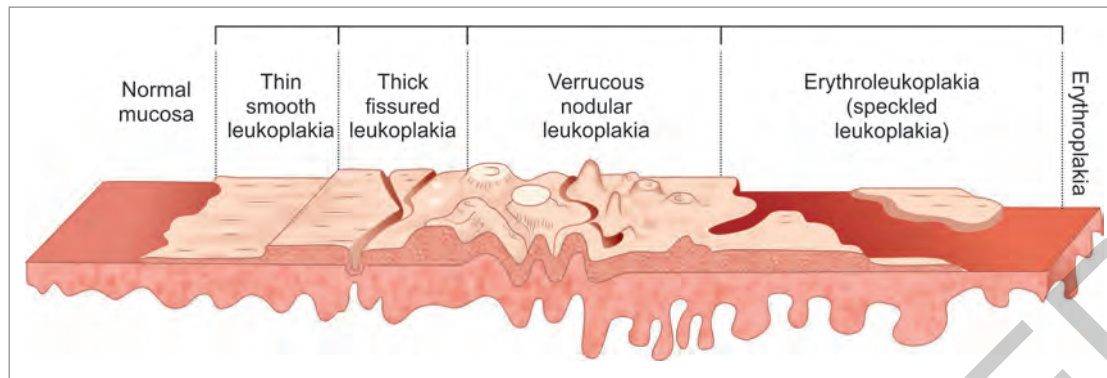


Fig. 6: Natural progression of head and neck cancers (Bouquet and Gnepp).

LEUKOPLAKIA

Definition: A predominantly white plaque of questionable risk having excluded (other) known diseases or disorders that carry no increased risk for cancer (WHO 2007). Thus leukoplakia is *always a clinical diagnosis of exclusion* which needs a histologic proof as necessary for management.

■ Diagnostic Points (WHO 2007)

1. A predominantly white patch/plaque that cannot be rubbed off (**Fig. 7**).
2. Circumscribed area and have well-demarcated borders. Irregular borders rarely with nonhomogenous leukoplakia.
3. No evidence of chronic traumatic irritation to the area.
4. It is persistent despite removal of the trauma/cause.
5. It does not disappear or fade away on stretching (retracting) the tissue.
6. Common differential causes ruled out.

■ Prevalence

- **Global prevalence:** 2.6%; India 4.8%
- **Gender:** Males >Female; (except proliferative verrucous leukoplakia)



Fig. 7: Leukoplakia (Black arrow).

- **Age > 50 years** at presentation
- **Site:** Smokers lateral border of tongue, floor of mouth and buccal mucosa, rest of mucosa
- **Nonsmokers:** More common in borders of tongue

The *clinical description* divides leukoplakia as (WHO 1980, 2007):

1. **Homogenous leukoplakia:** Uniformly white, flat and thin, cracked mud appearance.
2. **Nonhomogeneous leukoplakia:**
 - i. **Proliferative and verrucous:** Slow growing, raised surface (papillary proliferations, above the mucosal surface that may be heavily keratinized). Female:male ratio (4:1); maximum predisposition for malignant conversion. Difficult to eradicate with high recurrence rate. It is seen in lower Gingiva.
 - ii. **Ulcerated:** Lesion exhibits red area at the periphery of which white patches are present.
 - iii. **Nodular:** It is characterized by white specks or nodules on erythematous base.
 - iv. **Erythroleukoplakia:** Leukoplakia is present in association with erythroplakia. It is also called mixed lesion. It is associated with reverse smoking (Chutta smoking).

Staging of leukoplakia is done for oral leukoplakia (OL system) resembling TNM (WHO 2005) taking size (L) pathology (P) of the lesion. For example: Stage III is L3 (>4 cm), P1 (mild moderate epithelial dysplasia) or L3P2 (severe dysplasia)].

■ Management of Leukoplakia Principles

1. Confirmation of diagnosis if clinically "leukoplakia" otherwise reassessment after 2 weeks with removal of etiology and treatment of cause.
2. Biopsy (assisted by vital stains if necessary)
3. Grading leukoplakia as carcinoma (invasive/non-invasive), dysplasia.
4. Mild dysplasias are ordered deaddiction, dietary changes and regular follow-up
5. Moderate dysplasia needs follow-up versus LASER ablation
6. Severe dysplasia and CIS/invasive cancer will need surgical excision with at least a 3 mm pathologic margin.

BOX 4: Differential diagnosis of white patch in the oral cavity.

- **Trauma:** Thermal, mechanical, chemical
- **Infective:** Candida, oral hairy leukoplakia (OHL) [Epstein–Barr virus (EBV)], syphilis
- **Hereditary:** Genodermatosis
- **Dermatological:** Lichen planus, lupus erythem
- **Neoplasia:** Squamous cell carcinoma (SCC), in situ cancer
- **Idiopathic:** Leukoplakia

The role of chemoprophylaxis is debatable (see Screening and Prevention). **Box 4** shows the summary of differential diagnosis of “a white patch on the oral cavity”.

ERYTHROPLAKIA

Definition: Any lesion of the oral mucosa that presents as bright red velvety plaques which cannot be characterized clinically or pathologically as any other recognizable condition [WHO 2007 (**Fig. 8**)].

Etiology: Same as leukoplakia (See Etiopathogenesis).

Clinical subtypes:

- Homogeneous erythroplakia
- Erythroplakia interspersed with patches of leukoplakia
- Granular or speckled erythroplakia (speckled leukoplakia)

Differential diagnosis: Like leukoplakia, erythroplakia is a differential diagnosis for *red velvety lesion* in oral mucosa.

Diagnosis and treatment: Same as in leukoplakia with management in same lines.

Table 4 Describes the summary of differential diagnosis of “a red patch on the oral cavity”.

TABLE 4: Differential diagnosis of red patch in the oral cavity.

Clinical condition	Diagnosis
Infection	• Erythematous candidiasis • Histoplasmosis
Neoplasm	• Hemangioma • Kaposi's sarcoma
Inflammatory disorder	• Erythematous lichen planus • Discoid lupus erythematosus • Pemphigoid • Hypersensitivity reactions

ORAL SUBMUCOUS FIBROSIS (OSMF)

Definition: A chronic, insidious, scarring disease of the oral cavity, often with involvement of the pharynx and the upper esophagus.

Etiopathogenesis: Areca nut chewing in particular along with *Paan* (beetle leaf and beetle nut) with *gutkha*.

Clinical features: History of burning sensation in mouth, majority are silent. Clear white lesion, translucent, marbled white appearance of mucosa with restricted mouth opening and tongue movements (**Fig. 9**).

Grades of Oral Submucous Fibrosis with Clinical Highlights

- **Grade I:** Acute ulceration and recurrent stomatitis, mouth opening (MO) >cm
- **Grade II:** Mottled and marble-like sheets of epithelium palpable, MO: 2.5–3.5 cm
- **Grade III:** Pale firmly adherent mucosa with spread to oropharyngeal structures, MO: 1.5–2.5 cm



Fig. 8: Erythroplakia (Black arrow).



Fig. 9: Oral submucosal fibrosis (Black arrow).

- **Grade IVA:** Thickened mucosa, restricted tongue movement, MO <1.5 cm
- **Grade IVB:** Hyperkeratotic leukoplakia with SCC in situ changes

Management of Oral Submucous Fibrosis

- **Step 1:** Excision of fibrotic bands—scalpel, laser
- **Step 2:** Mucosa reconstruction—flaps, grafts, collagen membrane
- **Step 3:** Adjunctive procedures intraoperatively—coronoidectomy, masticatory muscle myotomy
- **Step 4:** Postoperative oral physiotherapy, dietary supplementation, and other medications

MANAGEMENT OF THE PRIMARY DISEASE IN ORAL CAVITY CANCERS (6 STEPS)

1. Clinical examination and imaging (clinicoradiological assessment)
2. Confirmation of diagnosis (biopsy)
3. Staging the disease
4. Multidisciplinary team (MDT)
5. Treatment
6. Rehabilitation

Clinical Presentation of Oral Cavity Malignancies

Primary tumors that originate from the mucosal surface of the oral cavity might vary in their clinical characteristics. A suspected oral malignancy can present as an ulcer, endophytic, exophytic lesion or a combination of both. The common presentation of the disease (stagewise) has been depicted in **Table 5** (**Figs. 10A and B**).

Important Notes

- Pain when present may be due to direct involvement of the underlying structures or due to infiltration of an underlying nerve.
- Referred pain to ear is also common which may be due to the nerves supplying the oral cavity, sharing a common pathway with the auriculotemporal nerve.
- Trismus, defined as a pathological restriction of the motion of the jaw, is a common presentation and may be due to the presence of OSMF or it may be due to disease involving pterygoid space or masseter space (**Figs. 11A to C**).
- Orocutaneous fistula is defined as an extraoral pathological communication between the oral cavity and mostly occurs due to tumor necrosis and infection (**Fig. 12**).
- Weight loss occurs primarily due to decreased oral intake and cancer cachexia due to the production of tumor necrosis factor alpha (TNF- α).

The common things to be looked into while examining the patient is summarized in **Box 5**. Depth of invasion (DOI) is the most important prognostic factor in cancer of the oral cavity. **Figure 13** shows assessment of the depth of the tumor on clinical examination by palpation.

TABLE 5: Stage-wise presentation of head and neck cancers.

Localized disease	Locally advanced disease	Metastatic disease
• Nodule • Ulcer • Combination of both nodule and ulcer • Halitosis	• Ulceroproliferative lesion • Skin infiltration • Otagia • Orocutaneous fistula • Trismus • Loss of teeth • Weight loss	• Breathlessness • Hemoptysis • Altered sensorium



Figs. 10A and B: (A) Early oral cavity carcinoma (Note the nodular lesion over the left buccal mucosa); (B) Locally advanced disease involving the skin.



Figs. 11A and B: (A) Evaluation of trismus with finger; (B) Evaluation of trismus in centimeters; (C) Management of trismus with MOPNER device.



Fig. 12: Orocutaneous fistula.

■ Imaging in Head and Neck Cancers

A thorough clinical examination must be done in conjunction with imaging of the primary tumor. The treating oncological

BOX 5: Clinical examination checklist in head and neck cancers.

Examination checklist:

- Size and extent of nodule
- Ulcer—margin, base, floor
- Bony invasion
- Trismus
- Coexisting premalignant lesions
- Status of tongue
- Neck node
- Evaluation of cranial nerves (V, VII, IX, X, XI, XII)

team is given a three-dimensional notion of the main by the image. The following are the objectives of imaging:

- Lesion stage (local extent, nodal spread, and metastatic detection)—the most typical sign, when the primary is clinically occult, identifying the lesion is necessary.
- Image-guided interventions; detection of new synchronous lesions or second primary tumors [e.g., fine needle aspiration cytology (FNAC) or biopsy]

- Evaluation of the patient's response to treatments (chemotherapy or radiation therapy)
- Follow-up imaging and surveillance



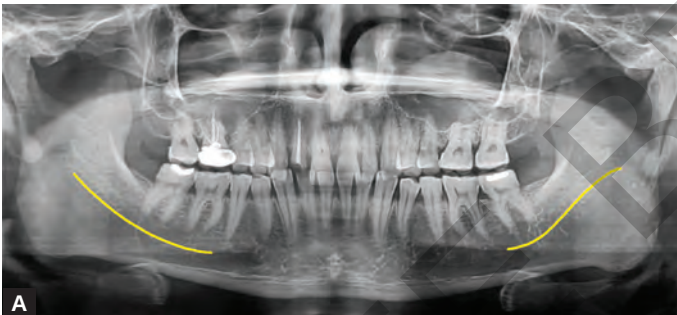
Fig. 13: Determination of tumor thickness on palpation (a surrogate clinical evaluation of depth of invasion).

Orthopantomogram

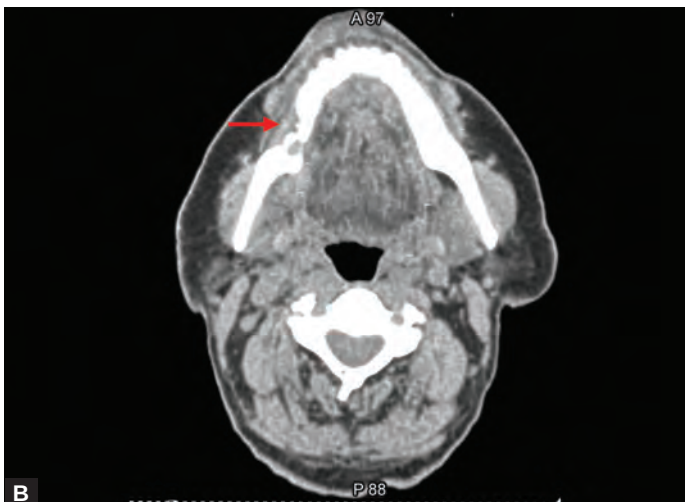
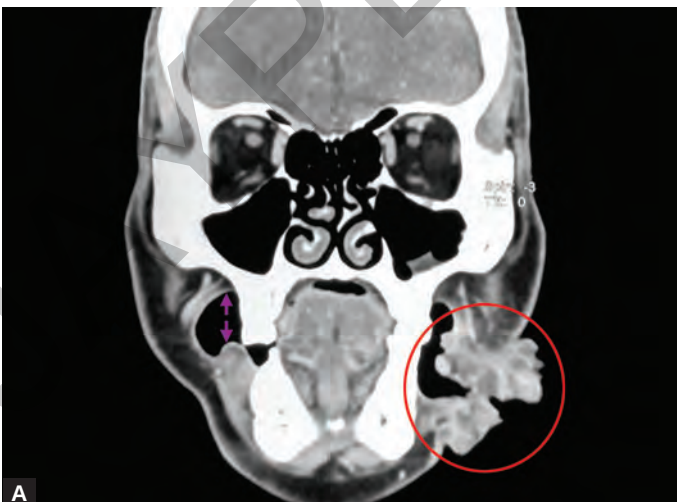
Orthopantomogram (OPG)/Panorex®—it is primarily used to assess the mandible prior to radiotherapy for dental prophylaxis. Preoperatively 30% of the bony cortex must be eroded for it to be picked up by orthopantomography (**Figs. 14A and B**).

Computed Tomography Scan

To achieve complete evaluation of the primary and any nodal disease, computed tomography (CT) should image the anatomy from the skull base to the thoracic inlet. Usually a multidetector CECT (with intravenous contrast agents, such as Iohexol is used) with sagittal reformatting of the images is preferred. Puffed cheek maneuver (**Fig. 15A**) helps to evaluate the lesions in the buccal cavity in which the patient is required to distend the oral cavity by blowing persistently and uniformly through closed lips. A soft tissue window generally caters for the tumor. CT has the highest specificity (87%) and magnetic resonance imaging (MRI) has the highest sensitivity for bony erosion. Bony erosion is an adverse prognostic factor and it requires a marginal or a segmental resection. Commonly used window is bone



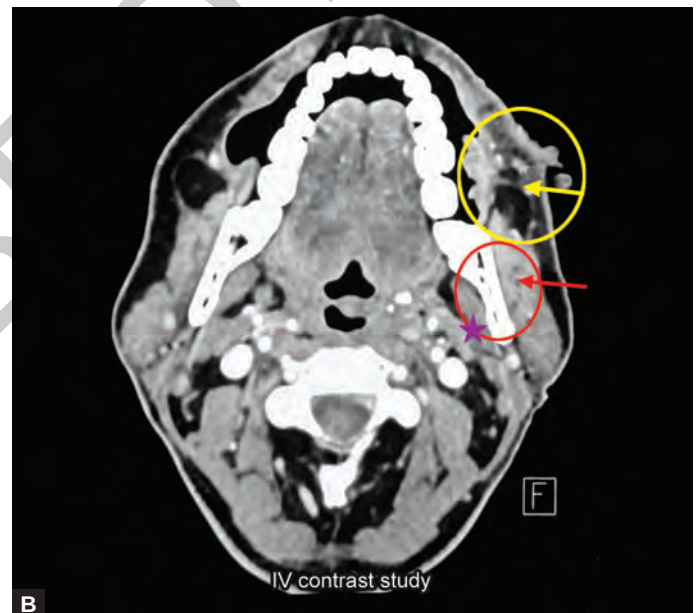
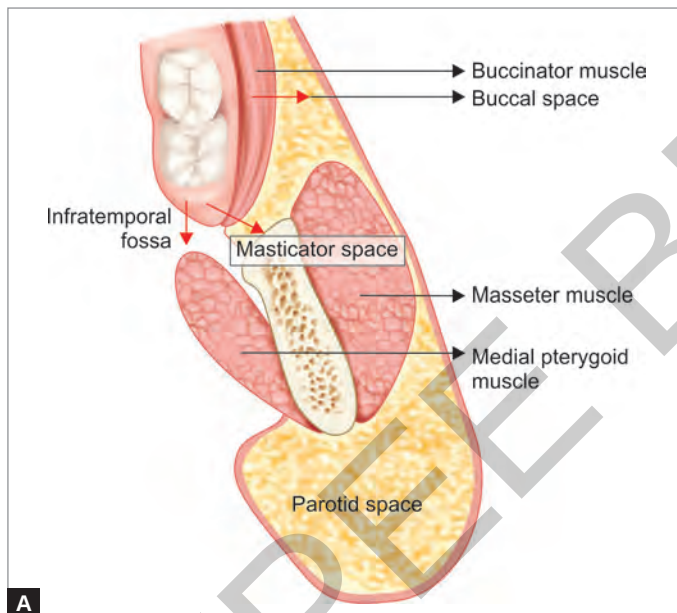
Figs. 14A and B: (A) Normal orthopantomogram (Yellow: Mandibular Canal); (B) Orthopantomogram showing mandibular erosion due to malignancy. Red arrow (Erosion of the Mandible due to tumor).



Figs. 15A and B: (A) Puffed cheek appearance (comparing the presence of a normal right side and tumor on the left side) (Purple: Upper and lower vestibule, Red circle: Tumor in the gingivobuccal sulcus); (B) CECT head and neck showing mandibular erosion (Red arrow).

TABLE 6: Spaces on head and neck imaging (related to resection of tumor only). (Refer Figs. 16A and B)

Space and its definition	Boundaries	Relevance
Masticator space enclosed within superficial layer of deep cervical fascia covering muscles of mastication (red arrow)	• <i>Inferiorly</i>: Lower border of mandible • <i>Superiorly</i>: Base of skull • <i>Medially</i>: Medial pterygoid muscle • <i>Lateral boundary</i>: Masseter and temporalis muscles	Contains the muscles of mastication and involvement of this area is considered T4 disease (locally advanced)
Infratemporal fossa/ITF (superior to purple star)	• <i>Medially</i>: Lateral pterygoid plate • <i>Laterally</i>: Mandible • <i>Superiorly</i>: Sphenoid and temporal bone • <i>Inferiorly</i>: Medial pterygoid muscle • <i>Anteriorly</i>: Posterior part of the maxilla • <i>Posteriorly</i>: Temporal bone	• Muscles of mastication, maxillary artery • Pterygoid plexus of veins • Facial nerve, otic ganglion, mandibular nerve Infratemporal Fossa involvement can be high or low. High ITF involvement is generally difficult to resect since the tumor may infiltrate the skull base and obtaining a R0 resection is difficult
Buccal space (yellow arrow)	• <i>Medially</i>: Buccinator • <i>Laterally</i>: Zygomaticus major	• It contains the buccal fat pad, retroantral fat pad, facial vessels and the parotid duct • Tumor may infiltrate via the retroantral fat pad into the infratemporal fossa



Figs. 16A and B: (A) Concept of spaces in head and neck cancers; (B) Radiological location of the spaces on imaging (Yellow: Buccal space; Red: Masticator space; Superior to purple star: Infratemporal fossa).

window or bone algorithm to evaluate the bony erosion (**Fig. 15B**).

Concept of spaces: CECT helps to determine a number of spaces in the head and neck region that helps the treating surgical team plan the resection. A summary of the spaces is highlighted in **Table 6** and the **Figures 16 A and B** depict the same.

CT Thorax

Multistation or lower neck nodal involvement or high-grade tumor histology, is an indication for CT thorax. Noncontrast CT of the chest can be screened for lung parenchymal

metastases but not the lymph nodes. A chest X-ray generally suffices for early cases.

Magnetic Resonance Imaging

This modality of imaging is equal to CT scan but has a better delineation of soft tissue. Superficial buccal lesions in the buccal space are easily assessed. Oral cavity cancer (**Fig. 17**), if there is a need to evaluate the extent of bone marrow invasion or in patients with extensive dental amalgam that may obscure the anatomy on CT. However, MRI overestimates the bony invasion. Any head and neck cancer with cranial nerve symptoms or if radiographic perineural

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