DISSERTATION ON

"A PROSPECTIVE STUDY OF MANAGEMENT OF ORAL CAVITY CANCERS"

Dissertation submitted to

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

In the partial fulfillment of the regulations

for the award of the degree of

M.S. IN OTORHINOLARYNGOLOGY

BRANCH-IV

REGISTRATION NUMBER: 221914052

GOVERNMENT STANLEY MEDICAL COLLEGE

CHENNAI -600 001.

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CERTIFICATE

I certify that the dissertation titled "**A PROSPECTIVE STUDY OF MANAGEMENT OF ORAL CAVITY CANCERS**" submitted by **DR.GANGA. P**, for the degree of Master of Surgery (Otorhinolaryngology) in partial fulfillment of regulations of The Tamil Nadu Dr. M.G.R. Medical University, Chennai is the result of original research work undertaken by him during the academic year 2019-2022, in the Department of Otorhinolaryngology & Head and Neck Surgery, Government Stanley Medical College, Chennai.

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CERTIFICATE BY GUIDE

This is to certify that the Dissertation titled **"A PROSPECTIVE STUDY OF MANAGEMENT OF ORAL CAVITY CANCERS"** presented by **Dr.GANGA P** is an original work done in the Department of Otorhinolaryngology, Government Stanley Medical College and Hospital, Chennai in partial fulfillment of regulations of the Tamil Nadu Dr.M.G.R. Medical university for the award of degree of M.S. (Otorhinolaryngology) Branch IV, under my guidance, during the academic period of 2019- 2022.

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DECLARATION

I **GANGA P** solemnly declare that the dissertation, titled **"A PROSPECTIVE STUDY OF MANAGEMENT OF ORAL CAVITY CANCERS"** a prospective study submitted by me is a result of the original work carried out by myself under the guidance of

PROF. DR.M. GOWRISHANKAR, M.S.,DNB.,DLO, Professor and head, Department of Otorhinolaryngology and Head and Neck, Government Stanley medical college, Chennai.

I further declare that the result of research has not been submitted previously by myself or other persons in any conferences or journals.

This dissertation is submitted to The Tamil Nadu Dr. M.G.R Medical University in the partial fulfilment of the rules and regulations for the M.S. Degree examination in Otorhinolaryngology to be held on May 2022.

Date: DR. GANGA P

Place: Chennai – 01

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CONTENT

INTRODUCTION

Any uncontrolled growth of cells that cause the adjacent tissue impairment and can invade is known as cancer. Oral cancer ensues with a small, unfamiliar, unexplained growth or sore in the subsites of oral cavity that include lips, cheeks, tongue, hard and soft palate, the floor of the mouth. In India there is a rapid increase in oral malignancy can be seen. Oral malignancy is altogether higher in south compared with the west, and is about 70% of all cases reported in India. The five-year survival rate is only 20 %.[1]

Oral cavity cancers are the sixth most common in worldwide. It accounts up to 30% of cancers in India with the male female ratio is 2:1.[2]

Oral squamous cell carcinoma (OSCC) contributes for example 84- 97% of oral cancers. The premalignant lesions are leukoplakia, erythroplakia, fibrosis, candidal leukoplakia, lichen planus and congenital dyskeratosis are indicators of the preclinical phase of oral cavity cancers. Betel-quid chewing, spicy foods, pan masala products, sharp tooth excessive alcohol consumption, smokeless tobacco, nutrient-deficient diet, poor oral hygiene, and sustained viral infections, i.e., human papillomavirus (HPV) are some of the risk factors that causes oral cavity malignancies. Periodontal problems have a high-risk incidence of among the Indian population, where oral malignancy is mainly occurs due to the habit of pan chewing. Tobacco utilization (in any form) is a prime reason for malignant growth, noticeably in developing countries. Aside from tobacco, pan chewing, spicy food, sharp tooth, areca nut, lime, catechu, cinnamon, and so on, leading to oral cancer, particularly in the north-eastern parts of India.

The chronic use pan chewing causes continues exposure of oral mucosa along with abrasion of epithelium linings. Smokeless tobacco consumed both nasally and orally, showed an association between oral cavity cancers and potentially malignant oral disorders. Various techniques are used routinely to detect oral cancer such as biopsy, staining, physical and histopathological examination, spectroscopic and radiological techniques, etc. To check further physical, psychological, and financial losses to the patient diagnosis of cancer in the early stage is very important. Survival rate can be increased up to 90% when there is early diagnosis, timely and proper treatment. There are numerous novel techniques are developed that have merits as compared to the currently practiced conventional diagnostic methodologies.[1]

Status of oral cancer in India

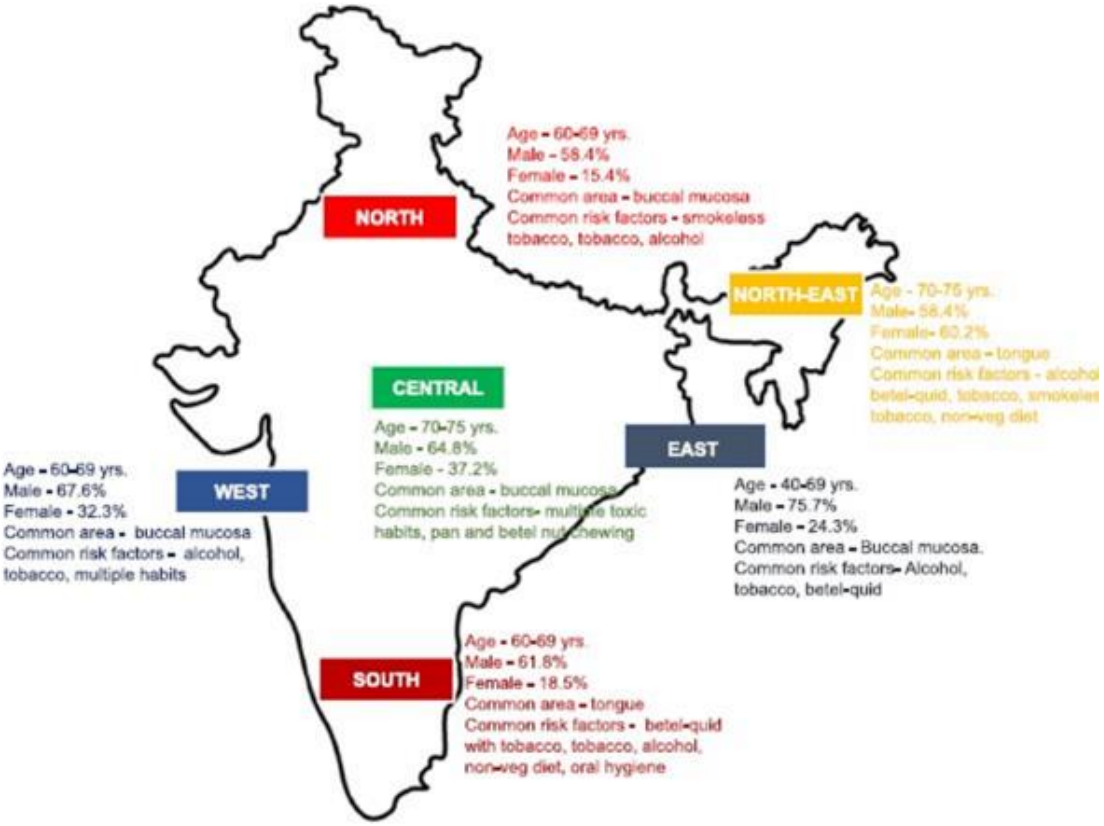


Figure 1

Among women in India, it remains the fourth most common cancer. India alone has more than one-third of the global oral cavity cancer burden (Cancer Today, 2018).[3]

Management of oral squamous cell malignancies remain essentially surgically driven. The major determining factor regarding management is the clinical extent or stage of the disease on presentation, with treatment generally consisting of broad local elimination to more "radical" elimination. Radiation therapy or a combination of both radiation therapy and surgery, depending upon clinical stage, are usually employed as management modalities. While chemotherapeutic options usually used in the case that such as large primary lesions, which are used adjunctively or palliatively in cases of very large or unresectable lesions. Such therapies, however, have not been able to extend prognosis or reduce mortality figures. Intra-arterial chemotherapeutic techniques may be useful in cases of advanced primary lesions where palliation is desired, with further clinical trials underway in order to determine efficacy.[4] Another important prognostic factor of oral cavity malignancy is the status of cervical lymph nodes. The cure rates will drop in to nearly half if the regional lymph nodes are involved. Thus, radical neck dissection places a main role in surgical management of cervical lymph node metastasis.[5]

The evolution of radical neck dissection first described by Crile in 1906 has continued as standard method of treating cervical node metastasis in patient with oral cavity malignancy. It is accomplished by a radical removal of all cervical lymphatic contents are at risk for metastasis and removal of sternocleidomastoid muscle, internal jugular vein, spinal-

accessory nerve and submandibular gland. The marked morbidity and functional disability demanded the modification of procedure especially preservation of spinal accessory nerve. Better understanding of lymph node drainage pattern of neck and indications for adjuvant postoperative radiation therapy changed the trend away from routine use of radical neck dissection.[6]

AIM AND OBJECTIVES

1. To study various management modalities of oral cancer in our hospital.
2. Comparison of different modalities of treatment in management of oral cancers.
3. Identify the risk factors associated with oral cancers.

REVIEW OF LITERATURE

Embryology of oral cavity

The head and neck are derived from lateral plate and paraxial mesoderm, ectodermal placodes and neural crest cells. The lateral cartilages and connective tissue of the lateral region of the head and neck formed by lateral plate mesoderm. The neural crest cells originate from neuroectoderm and migrate rostrally into the facial region forming the tissue structures and skeletal structures of face and ventrally into the branchial arches. The neurons of fifth, seventh, ninth, and tenth cranial sensory ganglia formed by ectodermal placodes and the neural crest cells. The head and neck development are marked by the formation of the branchial/oropharyngeal arches. These appear in the fourth and fifth weeks of embryologic development. The branchial arches are separated by branchial clefts. Pharyngeal pouches, form together with the development of branchial arches and branchial clefts. [7]

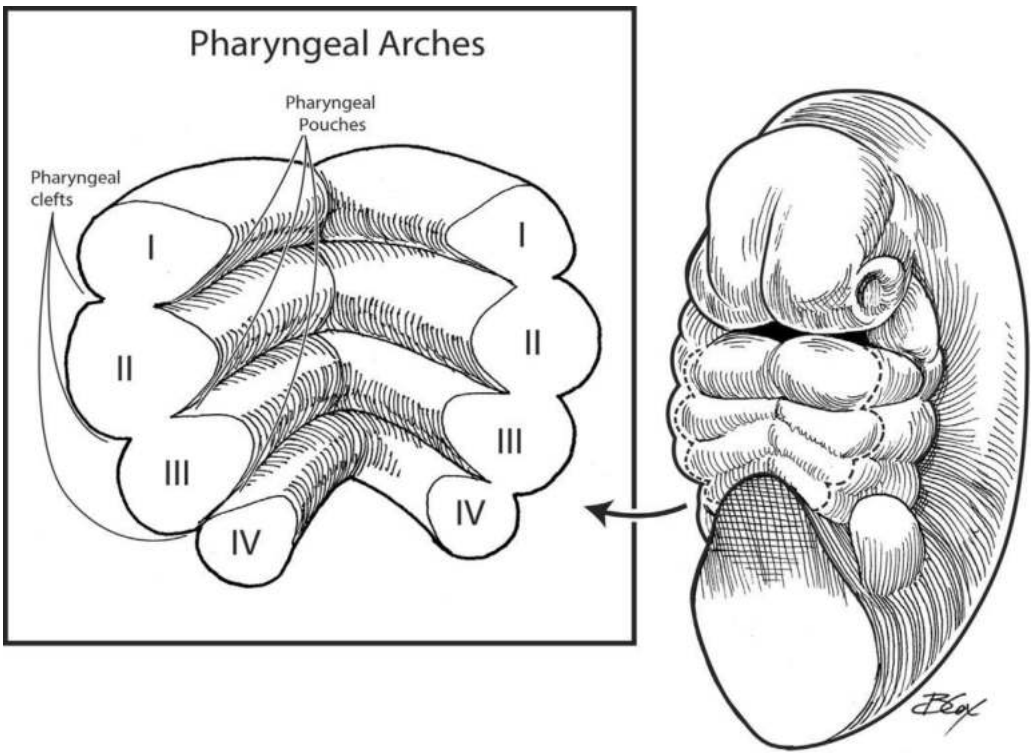


Figure 2

Development of tongue

The tongue develops in the form of two lateral lingual swellings and one medial swelling in 4th week of embryological age - the tuberculum impar. These swellings arise from the 2nd pharyngeal arch. The hypobranchial eminence is a second median swelling, formed from the mesoderm of the 2nd, 3rd, and fourth arches. From the posterior portion of the fourth branchial arch a third median swelling is formed. The arrival of the third median swelling marks the development of the epiglottis. The lateral lingual swellings enlarge until they outgrow the medial tuberculum impar and fuse to form the anterior two-thirds of the tongue. The V-shaped terminal sulcus separates the body of the tongue from the posterior third.

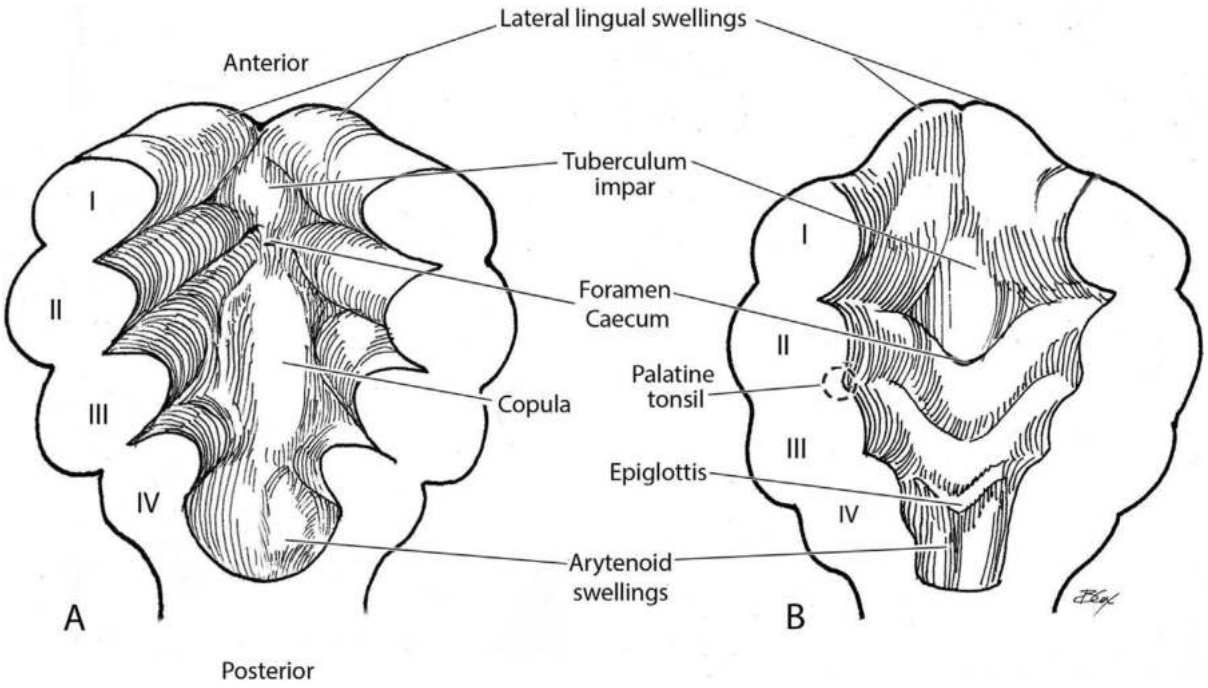


Figure 3

The muscles of the tongue are originating from occipital somite. Therefore, tongue musculature is supplied by the hypoglossal nerve. The mandibular branch of trigeminal nerve gives sensory innervation to the body of the tongue because the mucosa of the body is derived from 1st branchial arch.

Development of primary and secondary palate

The medial nasal prominences fuse with medial growth of the 2 maxillary prominences at a deeper level to form the intermaxillary segment. This segment is composed of the upper jaw component (carries 4 incisor teeth), the labial component (forms the philtrum of the upper lip), and the palatal component (form the triangular primary plate).

The palatine shelves form as outgrowths from the maxillary prominences at 6th week of embryological development and are directed downward on both side of the tongue. The palatine shelves attain a horizontal position at 7th week above the tongue and fuse to form the secondary palate. The midline of the fused palatine shelves appears the incisive foramen. Anteriorly both the secondary palate and the triangular primary palate fuse (figure 4). At the same time, the nasal septum grows down and joins with the upper aspect of the newly formed palate.[7]

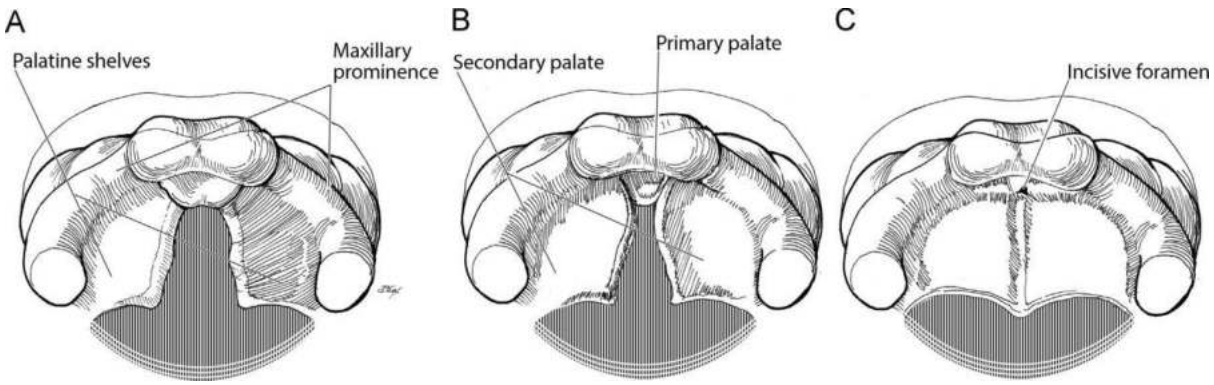


Figure 4

<u>The surgical anatomy of the oral cavity</u>

The oral cavity starts from the mucocutaneous junction called as the vermilion border of lip, posteriorly extends to the junction of soft and hard palate superiorly;

Laterally to the anterior tonsillar pillars: inferiorly to the junction of anterior two-thirds and posterior one-third of tongue where the circumvallate papillae are located. The oral cavity is

lined by keratinized stratified squamous epithelium.[8] The space between lips and cheek with teeth and gingivae is called vestibule of mouth. The oral cavity proper is the space contained by the teeth and gums. The roof is formed by the hard palate and floor by the mylohyoid muscle.

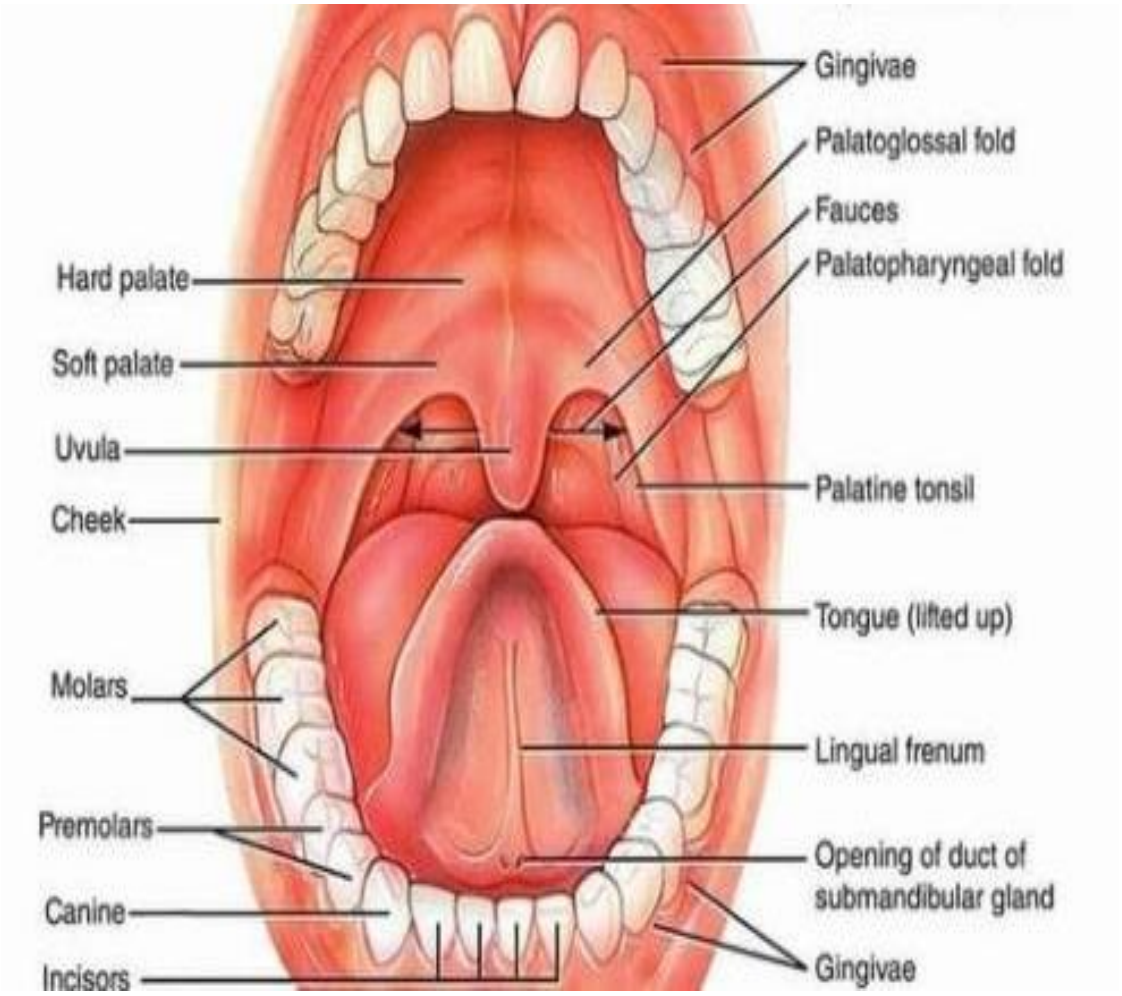


Figure 5

Oral cavity is divided in to 7 subsites

1.lips

2.alveolar ridges- upper and lower

3.oral tongue- anterior two-thirds

4.buccal mucosa- lines the inner aspect of lips and cheek including bucco alveolar gutters

5.hard palate

6.floor of mouth

7.retromolar trigone-behind the last molar tooth overlying ascending ramus of mandible

Lips and cheek

Lips and cheek are made up of fat and facial muscle. The oral cavity is surrounded by the lips.

Lips are fleshy folds externally lined by skin and internally by mucous membrane. Lips are consisting of

- skin
- muscle orbicularis oris
- submucosa with mucous labial glands and blood vessels
- mucous membrane.

Boundaries of lip;

Laterally: nasolabial fold

Superiorly: nose

Inferiorly: chin

The subunits of lip are:

1. Tubercle
2. Vermilion
3. Vermilion border
4. Cupid's bow
5. Philtrum.
6. Philtral columns.
7. Cutaneous upper lip.
8. Cutaneous lower lip.
9. Labiomental sulcus.
10. Chin.

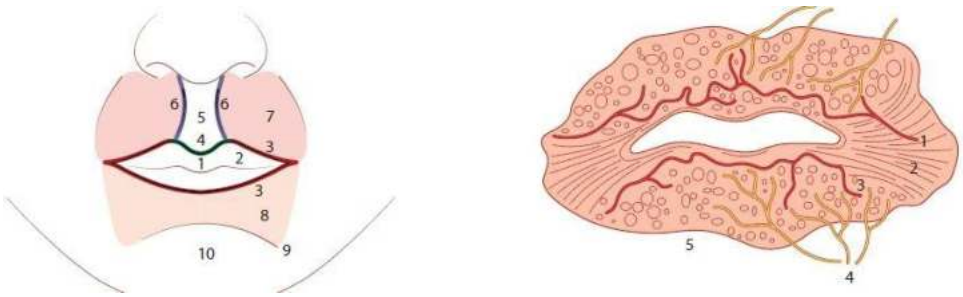


Figure 6

Cheeks are fleshy flaps, made up of

* Skin

*Superficial fascia containing some facial muscles, parotid ducts, mucous glands, nerves and vessels

*The buccinator covered with buccopharyngeal fascia which is pierced by parotid duct

*Submucosa containing mucous buccal glands and

*Mucosa

To the inner aspect of cheek, opposite to the crown of upper 2nd molar tooth there is opening of parotid duct. The entire vestibule is lined by mucous membrane.

The buccal mucosa is extending anteriorly from oral commissure to retromolar trigon posteriorly. An arbitrary line drawn from maxillary tuberosity to mandibular third molar's distobuccal aspect denotes the junction between buccal mucosa and retromolar trigon. mandibular and maxillary gingivo-buccal sulci forms the inferior and superior limits respectively.[9]

Boundaries and contents of buccal space

Medial wall: - buccinator muscle and overlying fascia. Medial wall extends from zygomatic arch superiorly and mandible inferiorly

Lateral wall: - skin, superficial fascia and deep portion by muscles of facial expression (zygomaticus major, risorius, quadratus labra superioris) and their fascia

Anterior boarder: - orbicularis oris muscle

Posterior boarder: - anterior boarder of masseter muscle

Contents of buccal space medial to lateral

1.Stensen's duct

2.Facial artery and vein

3.Lymphatic channel

4.Branches of the facial nerve.[10]

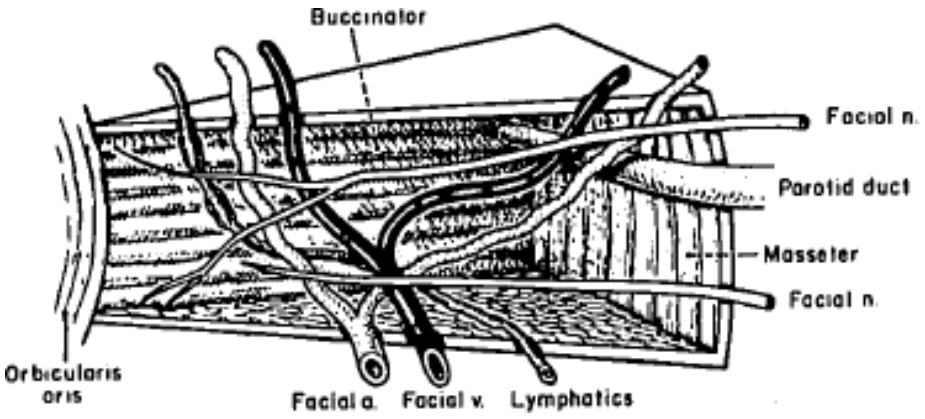


Figure 7

Gingivae

Gums are the soft tissue envelop of upper and lower alveolar processes which surround the neck of teeth. They consist of dense fibrous tissue under cover of stratified keratinized epithelium.

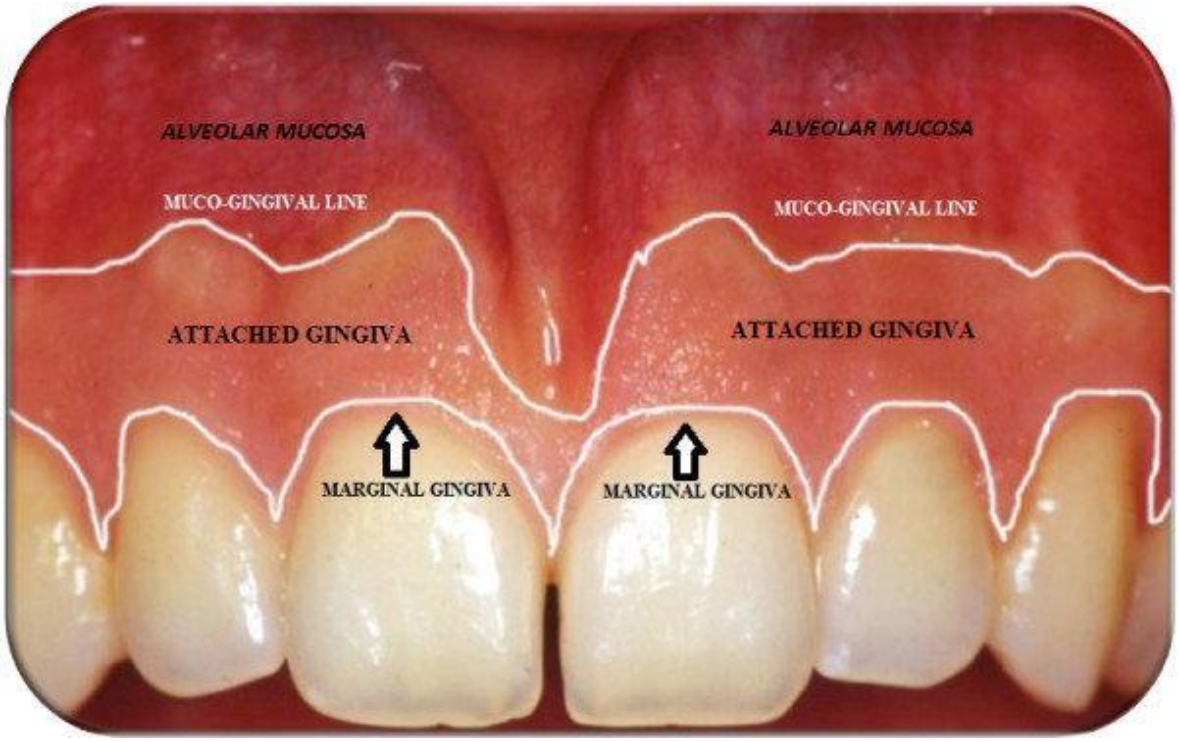


Figure 8

Retromolar trigon

It is a triangular area of mucosa overlying the ascending ramus of the mandible.

Base: In the region of mandibular third molar

Apex: adjacent to maxillary tuberosity

Laterally: buccal mucosa

Medially: anterior tonsillar pillar

Sensory supply: buccal branch of mandibular division of fifth nerve

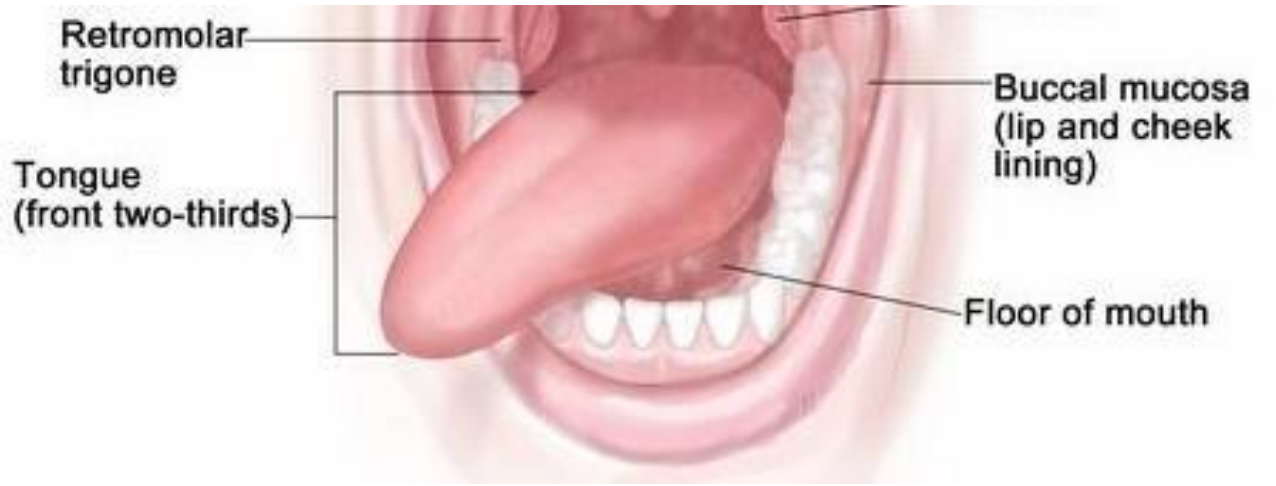


Figure 9

Hard palate and maxillary alveolus

Maxillary alveolus and hard palate belong to the maxilla. The bony alveolar process supports the maxillary teeth lined by mucoperiosteum and stratified squamous epithelium. Maxillary alveolus fuses medially with hard palate and laterally to buccal mucosa.

Palatine process of maxillae forms the anterior two-thirds of hard palate and posterior one third by horizontal plates of palatine bones. Minor salivary glands are located in the submucosa of

the hard palate. Thirty-three percentages of palatal tumors are derived from the epithelium of salivary glands. At the posterior most edge of palatine bone, hard palate fuses with soft palate.

Sensory supply of maxillary mucosa: branches from maxillary division of fifth nerve

Anterior hard palate nerve supply: nasopalatine nerve

Posterior palate: paired greater palatine nerves

Mandibular alveolus

Mandibular alveolus the intra oral part of mandible. The bony mandibular alveolar process is covered by mucoperiosteum and which supports the mandibular dentition. It medially joins with floor of mouth and laterally to buccal mucosa at gingival sulcus. It extends posteriorly and merges with retromolar trigon of same side.

Sensory supply: trigeminal nerve by its mandibular division

Mandibular alveolar carcinoma is triple times common compared with maxillary alveolar carcinoma

Tongue

The freely mobile anterior two-thirds of tongue is called the oral tongue. The circumvallate papillae demarcating it from posterior one third. The sub divisions are;

Tip

Dorsum

Lateral borders

Ventral surface

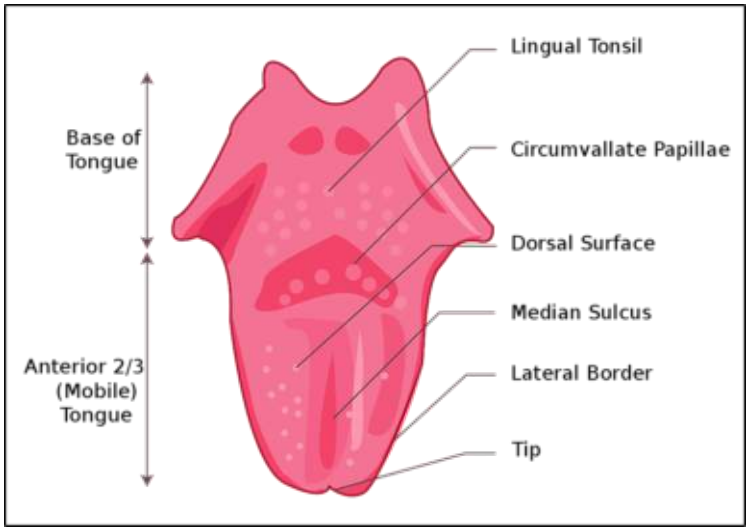


Figure 10

The floor of mouth communicates with the lateral and ventral borders, both the borders being supplied by non-keratinized stratified squamous epithelium.

The tip and dorsum are lined by a thick keratinized stratified squamous epithelium with specialized gustatory mucosa. Just below the mucosa the intrinsic muscles with the four paired extrinsic muscles;

Genioglossus

Hyoglossus

Styloglossus

Palatoglossus

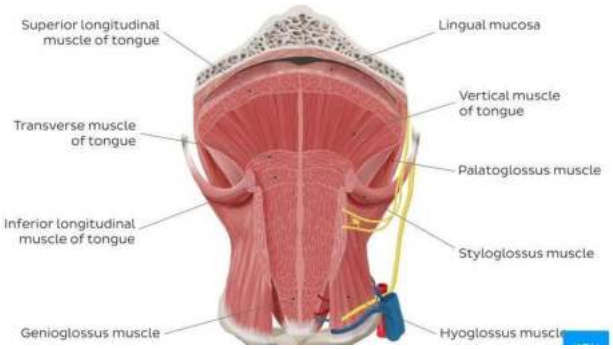


Figure 11

Motor supply: hypoglossal nerve except palatoglossus which is supplied by vagus nerve

Sensory supply: lingual nerve branch of mandibular division of fifth nerve

Taste sensation: facial nerve fibers which run with lingual nerve then passes to chorda tympani nerve

Tongue is mainly made up of genioglossus muscle along with some fibers of longitudinal and transverse intrinsic muscles. There are two sets of intrinsic and extrinsic muscles. The inferior surface of tongue attached to the mandible by a mucous membrane reflection over the floor of mouth connecting the lingual surface of gingivae. Tongue is having circumvallate, filiform and fungiform papillae.

Roles of tongue: -

Mastication

Deglutition

Speech

Taste

Floor of mouth

The floor of mouth is lined by mucous membrane.

Boundaries: -

Anteriorly: attached mucoperiosteum of the mandibular alveolus

Laterally: attached mucoperiosteum of the mandibular alveolus

Posteriorly: anterior tonsillar pillars

Medially: fuses with ventral and lateral aspects of tongue

It is lined by stratified squamous non keratinized epithelium with less dense submucosa. The structures underlying the mucosa are:

- Sublingual glands
- Submandibular ducts
- Minor salivary glands
- Lingual nerve
- Hypoglossal nerves
- Genioglossus muscle

Above structures are bounded laterally by mylohyoid muscle and medially by hyoglossus muscle.

Ducts of submandibular glands enter anteriorly both sides of lingual frenum

Sensory supply: lingual branch of mandibular division of fifth nerve.[9]

Lymphatics of oral cavity

Mainly oral cavity drains to submental, submandibular and also to upper deep cervical lymph nodes. Lip, alveolus (anterior part), floor of mouth and anterior third of tongue drains to the submental group. Middle third of the tongue and rest of the oral cavity drains mainly to upper deep cervical and submental groups. Except the tip of tongue, remaining parts of anterior two thirds of tongue drains through the floor of mouth to the same side submandibular nodes. But in the midline, there occurs some overlap. The lymphatics of tongue finally drains into the jugulo omohyoid nodes. The midline malignancies tense to metastasize in both sides.[11]

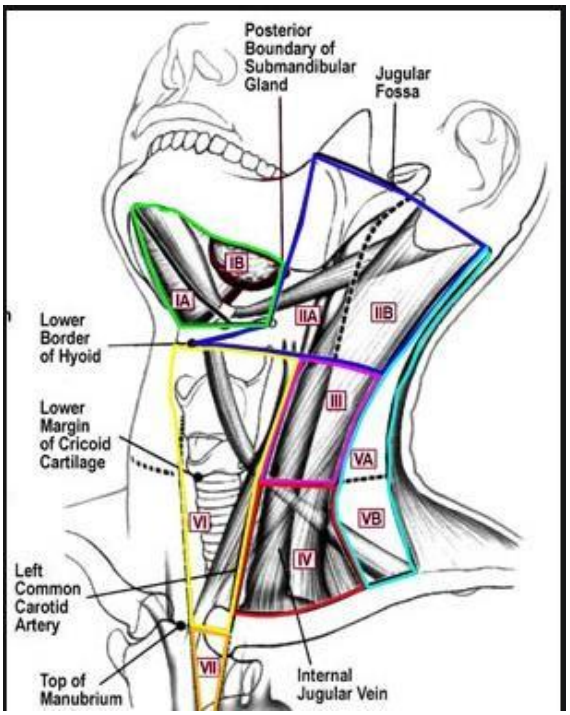


Figure 12

Level 1: Contains submental and submandibular triangles mounted by posterior belly of digastric muscles, the hyoid bone inferiorly and the body of mandible superiorly.

Level 2: Contains the upper jugular lymph nodes and extends from the level of hyoid bone inferiorly to the skull base superiorly.

Level 3: Contains the middle jugular lymph nodes from the hyoid bone superiorly to the crico-thyroid membrane inferiorly.

Level4: Contains the lower jugular lymph nodes from the cricothyroid membrane superiorly to the clavicle inferiorly.

Level5: Contains the posterior triangle lymph nodes mounted by the anterior border of trapezius posteriorly, the posterior border of the sternocleidomastoid muscle anteriorly and the clavicle inferiorly.

Level 6: Contains the anterior compartment lymph nodes from the hyoid bone superiorly to the suprasternal notch inferiorly. On each side the medial border of the carotid sheath forms the lateral border.

Level 7: Contains the lymph nodes inferior to the suprasternal notch in the upper mediastinum.[9]

Oral cavity - blood supply

Oral cavity gets it blood supply from the external carotid artery by its three branches. The cheek is supplied by facial artery. Tongue is supplied by the lingual artery. Palate is supplied by greater palatine artery which is a branch of maxillary artery through the greater palatine foramen.[8]

Oral cavity – nerve supply

Mucous membrane of the cheek gets its sensory supply above by maxillary division of the trigeminal nerve and mandibular division supplies below. The mucous membrane of the tongue on its anterior two third gets its common sensation via trigeminal component of lingual nerve. But its taste sensation is mediated by the chorda tympani component. All muscles of tongue except palatoglossus are innervated by hypoglossal nerve. Pharyngeal branch of vagus nerve innervates palatoglossus.[8]

Anatomy and Physiology of the Oral Mucosa

The oral mucosa is divided into three layers outer to inner

- Stratified squamous epithelium
- Basement membrane
- Connective tissue composed of the lamina propria
- Submucosa.

The buccal mucosa is permeable 4–4000 times greater than the skin epidermis and less than that of the intestinal mucosa. The order of permeability is sublingual > buccal > palatal. This is because the sublingual tissue nonkeratinized being relatively thin while palatal tissue is keratinized.

Epithelium

The buccal epithelium, made up of nearly 40–50 layers of stratified squamous epithelial cells. The mitotically active layer is the basal layer and produces epithelial cells, which migrate-

through intermediate layers. As the cells migration progresses to the surface, they increase in size and become flattened. Protein content is elevated and cytoplasmic organelles disappear. Cells are then shed. In 5-6 days, epithelial turnover occurs. Composition of the epithelium in the gingiva and hard palate (areas subject to stress) are keratinized, and rest are nonkeratinized. Hence, the thickness varies from 500 to 800 μm. Tonofilaments are large molecular weight (40–70 kDa) proteins are the important biochemical feature of the buccal epithelium. A matrix rich in carbohydrate–protein complexes surround the epithelial cells as a lubrication. The buccal epithelium is characterized by presence of gap junctions. [12]

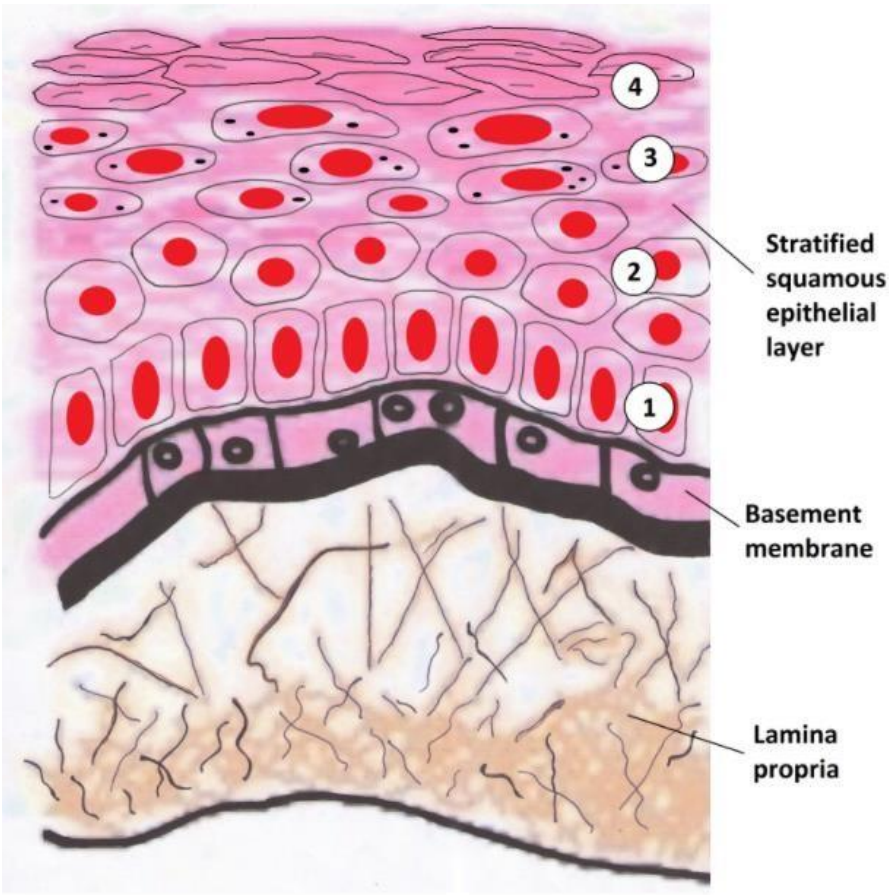


Figure 13: Histology of oral mucosa

Physiological functions of oral cavity

1.Mastication

Dentition serves to masticate the food and helps it for digestion. It is a combined activity and integration of the component part as a single unit.

2.Speech

Along with pharynx resonation of speech is carried out.

3.Taste

This function is accomplished by the presence of gustatory epithelium.

4. Absorption

Since the mucous membrane of the oral cavity is non-keratinized it is more permeable and absorption through mucous membrane is very fast compared to skin. Thus, it is used for topical administration of drugs for systemic action particularly floor of mouth.

5. Respiration

Oral cavity, is the secondary external opening for the respiratory tract. Most normal breathing takes place through the nasal cavity, but the oral cavity can be used to supplement or replace the nasal cavity's functions when needed.

6. Oral stage of deglutition

- Chewed food is softened by saliva and rolled into a bolus.
- Bolus is pressed against the hard palate as a result of contraction of the front part of the tongue.

- Swallowing commences by closure of the mouth and contraction of mylohyoid muscle.

7. Role of Saliva

Saliva is a dilute aqueous fluid that contains both inorganic and organic components. Three major salivary glands namely parotid, submandibular and sublingual salivary glands are the main source of secretion saliva. Apart from major glands, minor salivary glands also secrete saliva and are widely distributed in the oral cavity. Saliva pH ranges from 6.2 to 7.6.[13]

Etiology

- Betel nut chewing

The polyphenol content in the betel (areca nut) causes precipitation of the mucins totally which deteriorates the defense offered by the oral mucosal epithelium. Salivary mucins is significantly reduced for a regular betel chewer.[14,15]

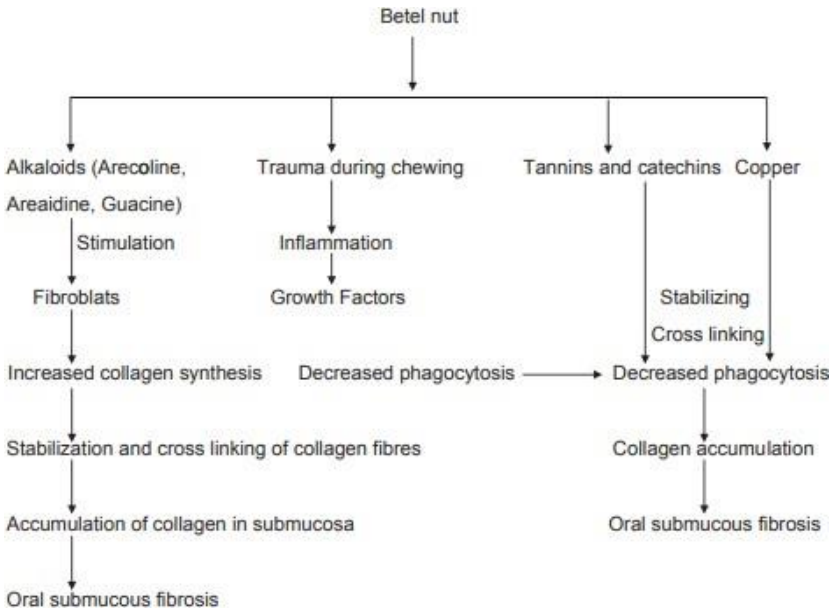


Figure 14

- Tobacco

There is clear-cut evidence of development of oral cavity cancer in proportion to duration and intensity of exposure to tobacco.[3,4]Smokeless tobacco use have been shown strong association with precancerous condition leading to oral cancers among urban Indian women [3]

- Smoking

There is a six times higher risk of developing malignancy in smokers compared to non-smokers which again depend upon the duration and pack year of smoking.[16]

- Nutrition and diet

Dietary intake of fruits, vegetables, carotenoids have shown protective effect for oral malignancy. There is an increased risk association with Plummer-Vinson syndrome.

- Genetical factors

Smoking and drinking has been correlated with p53 gene which is overexpressed in the cancer of oral cavity. Hence, p53 can be used as a potential tumor marker in the field of cancer treatment.[4]

- Infection

Human papilloma virus is found to be a possible causative factor especially HPV 16 and 18 are found to be in 15 to 20 percent of malignancies in oral cavity.[17]

- Dental status

The use of removable and fixed dentures, sharp edges have shown a decrease in the matrix metalloproteinases and it can be associated with squamous cell carcinoma of the oral mucosa.[18]

Premalignant lesions of oral cavity

1.**Leukoplakia**: WHO definition any white patch or plaque that cannot be characterized clinically or pathologically as any other disease. It is a definitive and most common risk of malignant changes in oral cavity.[3]

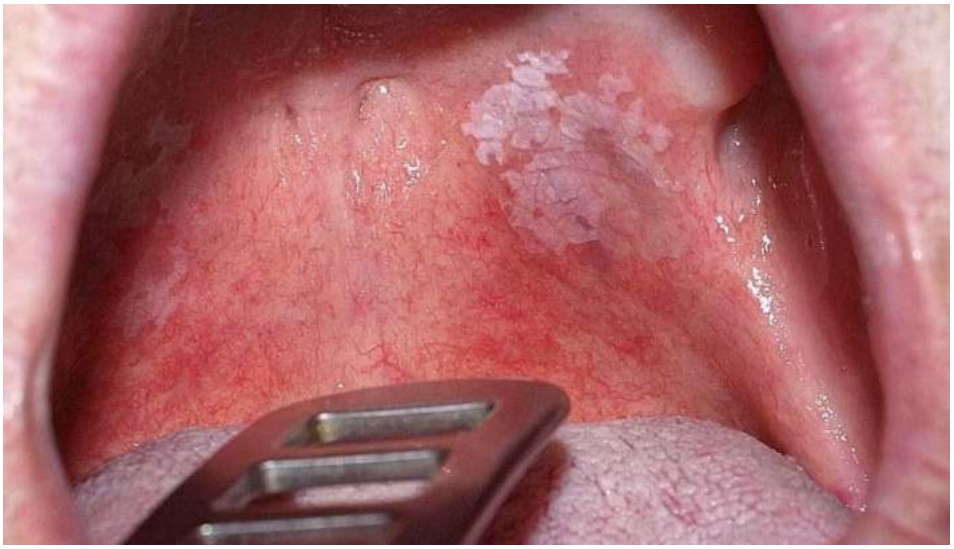


Figure 15

2.**Erthroplakia**: by definition any lesion of the oral mucosa which presents as bright red velvety plaques that cannot be characterized clinically or pathologically as any other recognizable condition. This also carries a definitive risk.

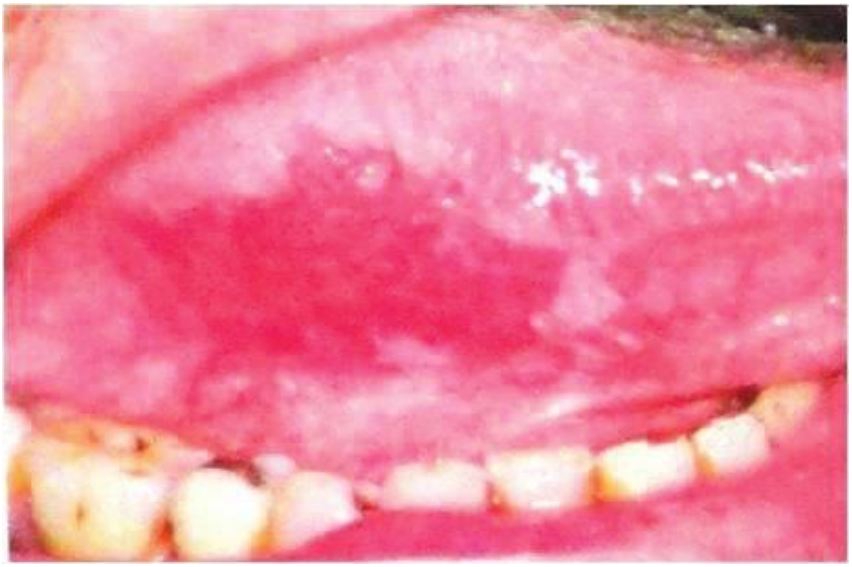


Figure 16

3. **Chronic hyperplastic candidiasis**: Dense plaques of keratin is seen in this condition.

various immunodeficiencies cause the growth of candida albicans which in turn invade the epithelium and have definitive risk of developing malignancy in oral cavity.

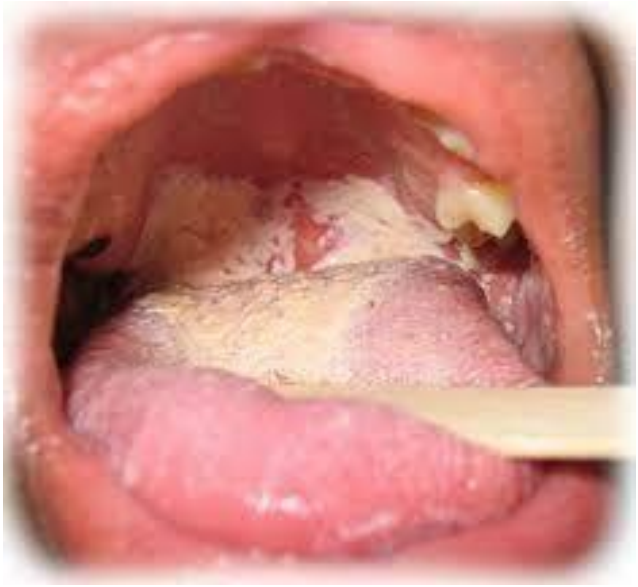


Figure 17

4. **Oral submucous fibrosis**: It is progressive in nature in which formation of fibrous bands under the oral mucosa.

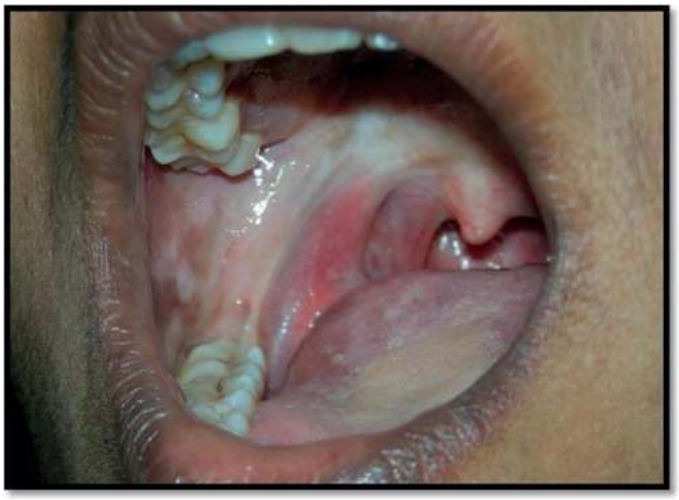


Figure 18

5.**Syphilitic glossitis**: It involves the interstitium causing end arteritis ending in sloughing of overlying mucosa.

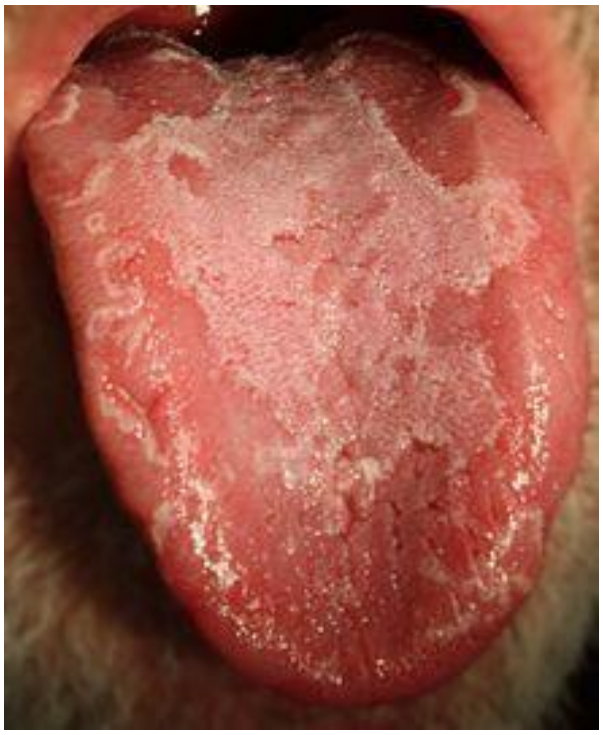


Figure 19

6.**Plummer-Vinson syndrome**: Epithelial atrophy seen in sideropenic dysphagia makes it vulnerable to carcinogens.[17]

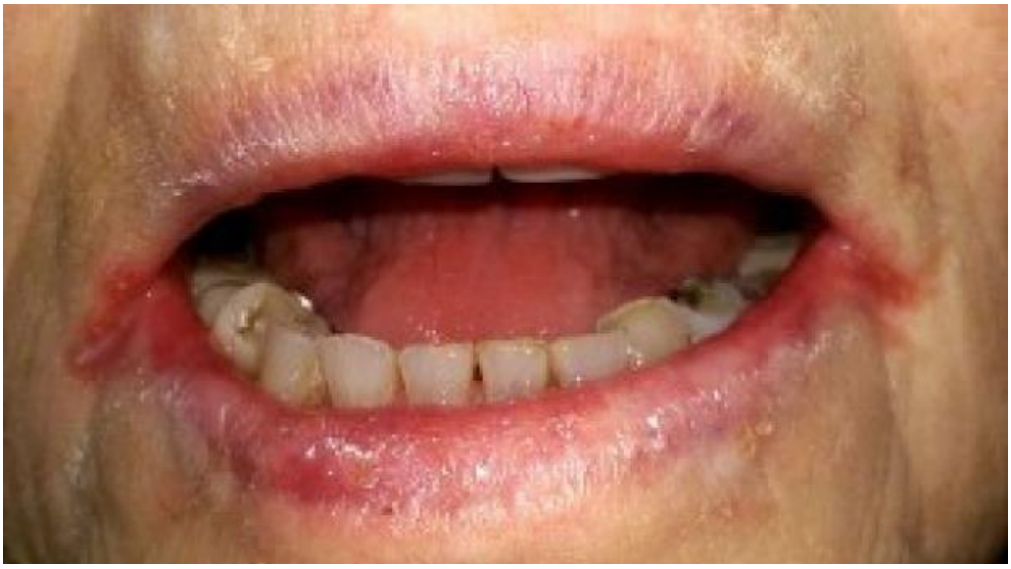


Figure 20

7. Oral lichen planus

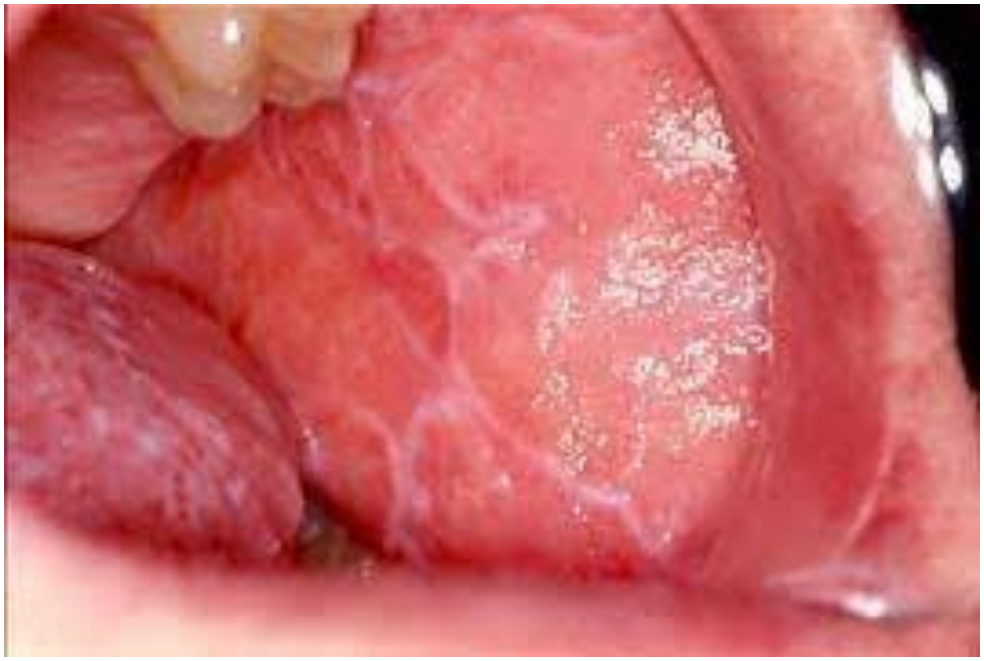


Figure 21

8. Discoid lupus erythematosus

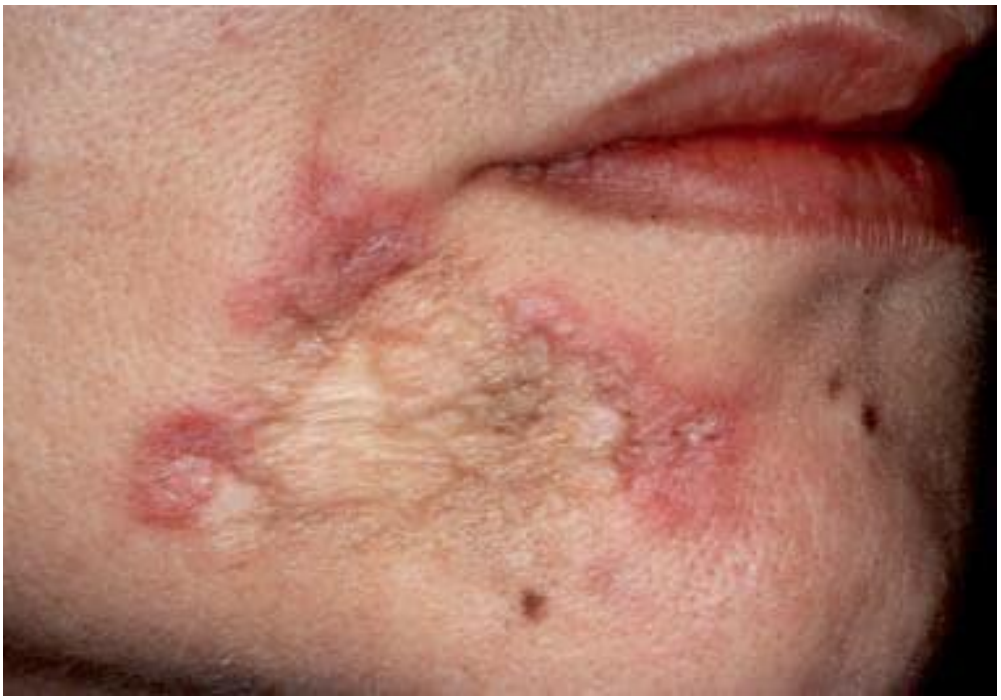


Figure 22

9.Dyskeratosis congenita

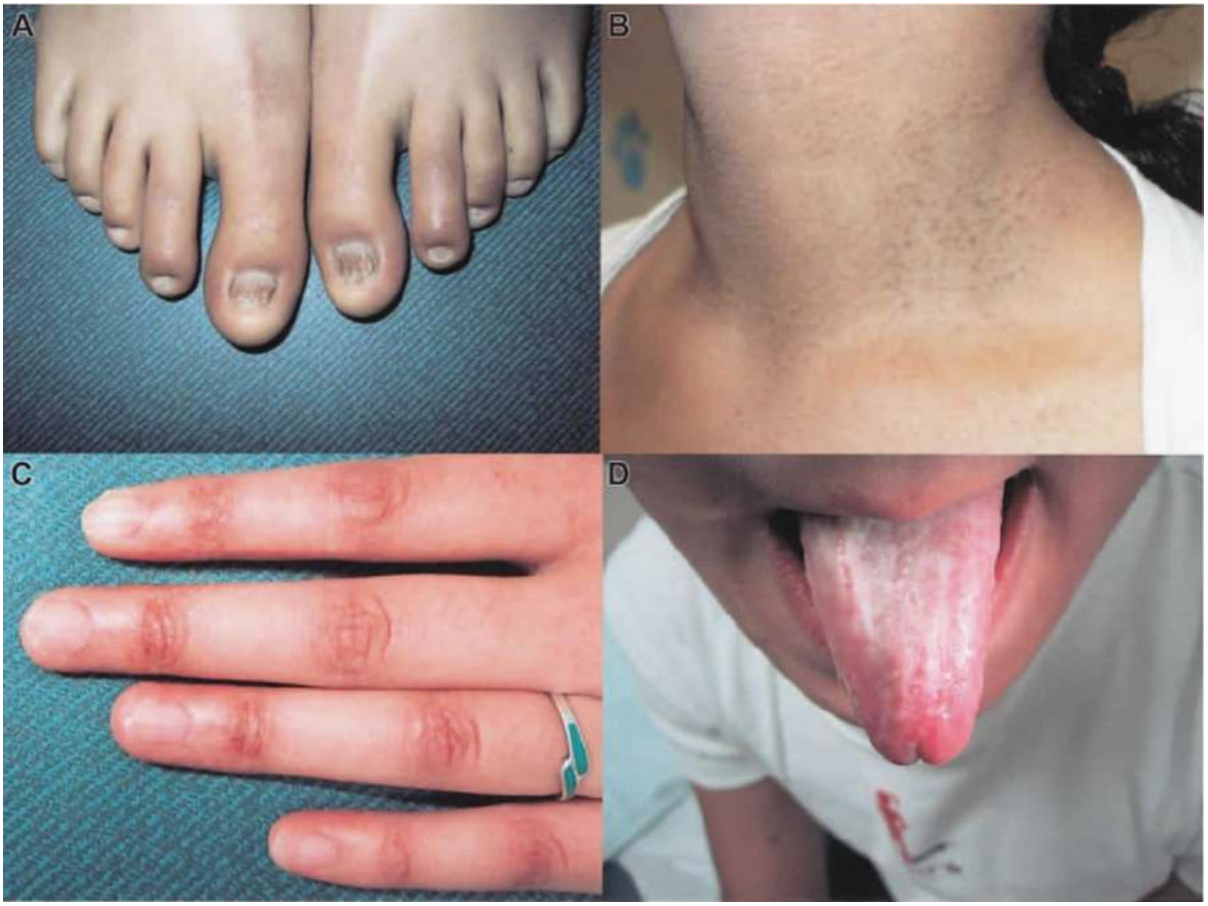


Figure 23

Macroscopic appearance of oral cavity malignancies

1.**Proliferative type**

Otherwise known as exophytic type. It is not a common type. Associated with good prognosis.in advanced stages there will be deep infiltration along with fleshy proliferative superficial involvement.

2 **Ulcerative types**

In this type edges are heaped up which bleeds on touch. The surrounding structures are easily involved by infiltration. Hard in nature with a high histological grade.

3.**Superficial plaque type**

Fine granulation and shallow in depth with less invasion. In patients with melanoplakia it is common.

Pathological types of CA oral cavity

1.<u>Squamous cell carcinoma</u>

Dermis is infiltrated by columns of epithelial cells but appears as if it is separate from the remaining of growth. There will be keratinization at the center of mass. The epithelial pearls/cell nests of keratin can be stained with eosin which appears similar as horny material over the skin. So, it is named as epidermoid carcinoma.[7]

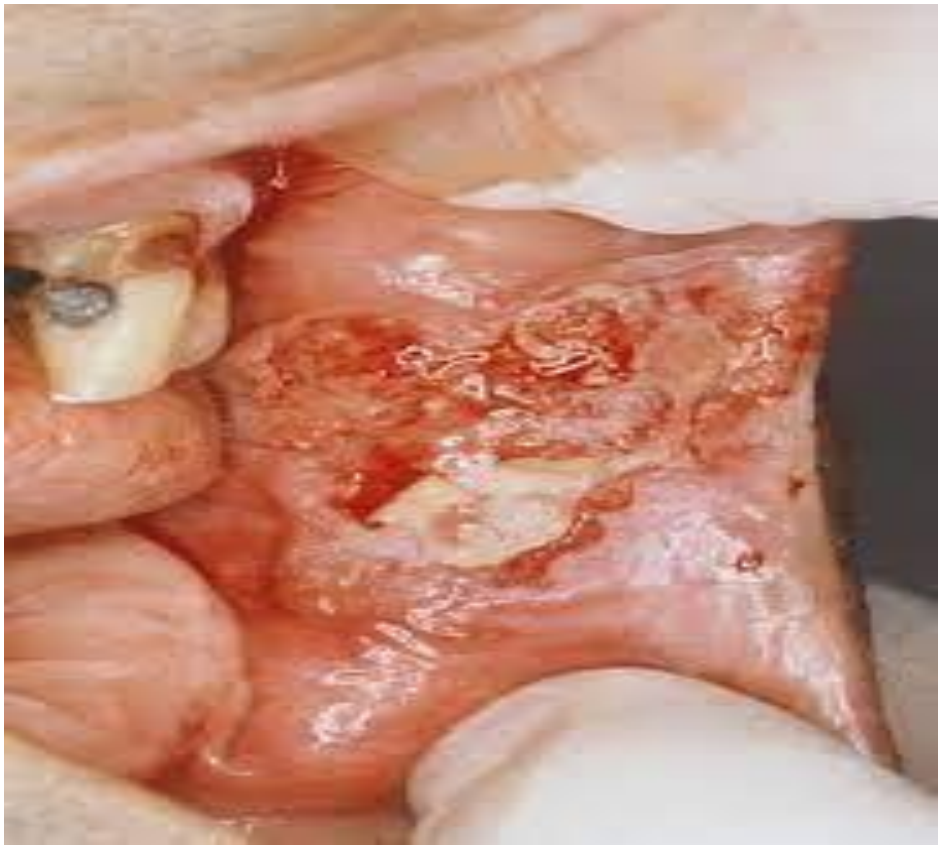


Figure 24

2.**Basaloid squamous cell carcinoma** It is the most aggressive variety of squamous cell carcinoma.

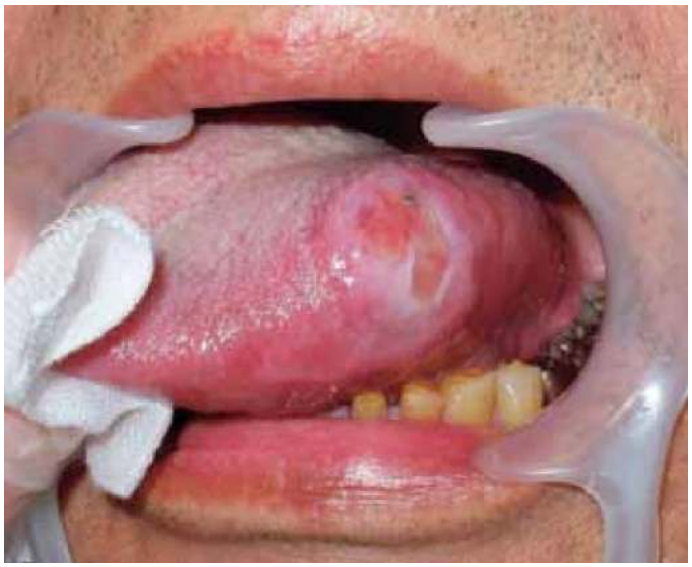


Figure 25

3.Verrucous carcinoma

It represents <5% of malignancies in oral cavity.it is also a variety of squamous cell carcinoma. This malignancy is low grade and metastasize uncommonly. Local excision is found to be sufficient for this variety.

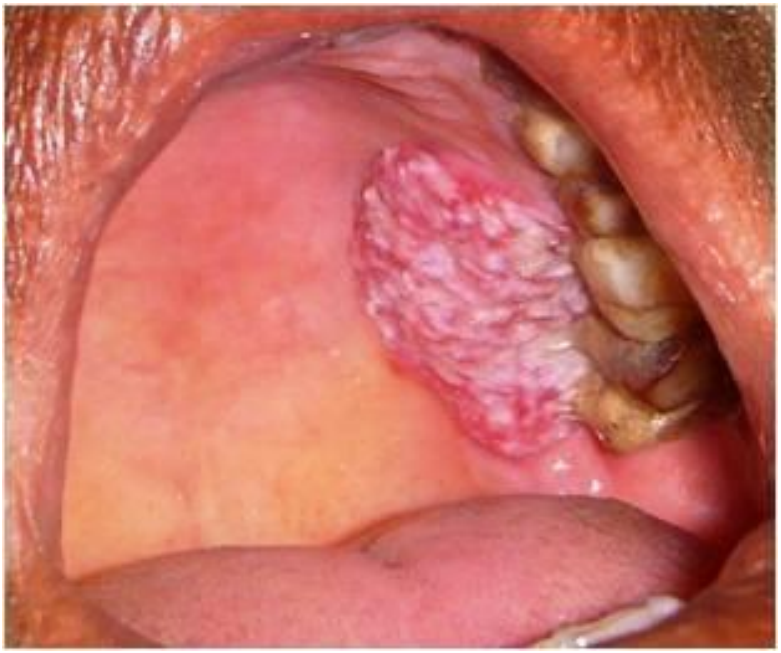


Figure 26

4. Mucoepidermoid carcinoma

It is a malignant epithelial tumor, first described by Stewart et al in 1945. The tumor is composed of both epidermoid type cells and mucous secreting cells in various proportions. It is the most common malignant neoplasm observed in major and minor salivary glands. This salivary gland carcinoma accounts for 5% of all salivary gland tumors. Intraorally mucoepidermoid carcinoma have strong predilection towards the palate.

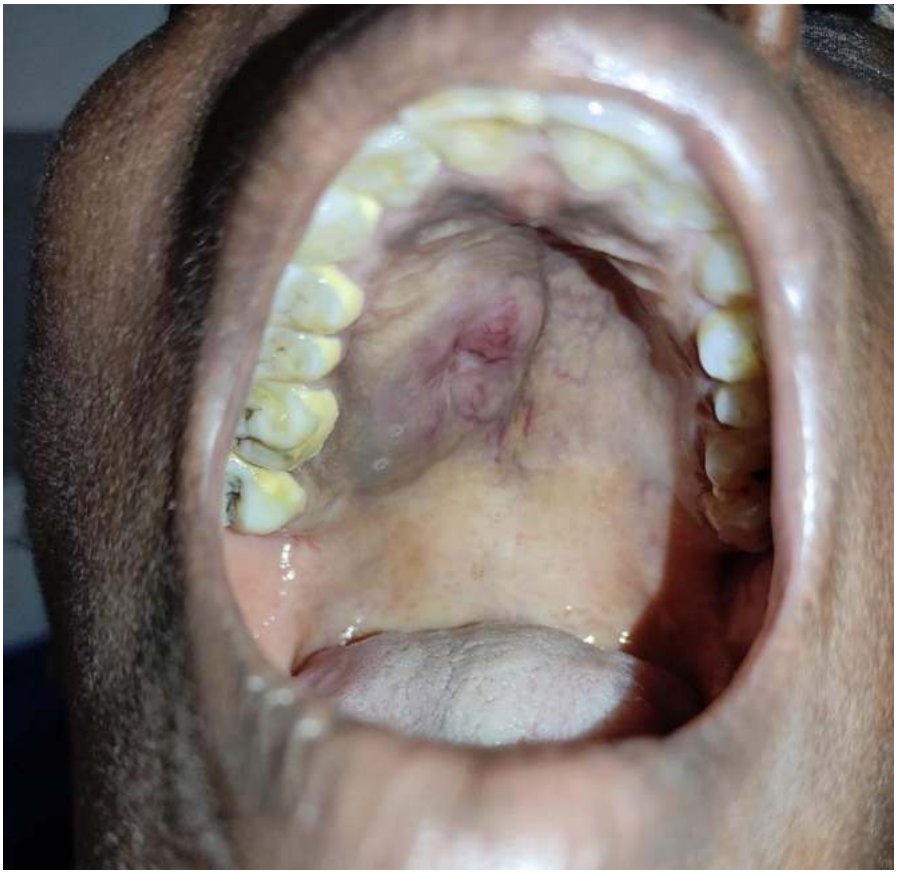


Figure 27

5. Adenoid Cystic carcinoma

Adenoid cystic carcinoma is a slow growing but aggressive neoplasm, previously known as cylindroma with a high tendency of recurrence. It is characterized by proliferation of ductal and myoepithelial cells. In solid, cystic, tubular, and cribriform patterns. It is the fifth most

common malignant epithelial tumor of salivary glands according to Armed forces institute of pathology.[19]

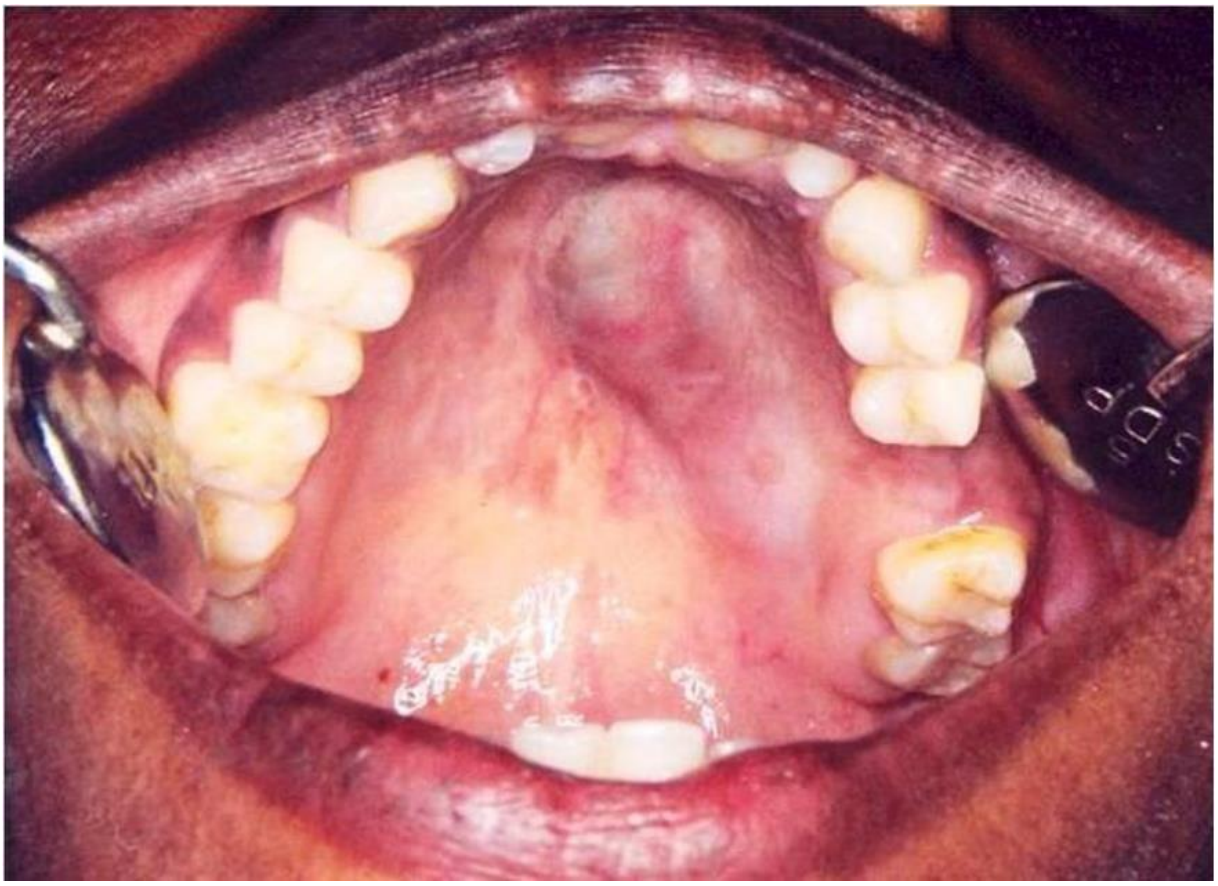


Figure 28

Clinical presentation of oral cavity cancers

1.<u>Lip</u>

Tumors are more common in the lower lip. It usually do not cross the midline. Presentation can be bleeding and foul-smelling discharge from the ulcer or nodular swelling or a neck swelling.

2.<u>Gums and alveolar ridge</u>

Presentation can be tumor on the gum, ulcers surrounding the teeth, bleeding from lesions, poorly fitting dentures, poor healing of alveolar ridge following dental extraction. In late stages, there will be trismus also.

3.Floor of mouth

The ulcerated lesions on the floor of mouth usually preceded with an induration. Tongue and inner side of mandible are involved in primary stages which cause speech disturbances, bleeding, pain and bad breath can be seen in late stages. Spread to level IIb and III which can be bilateral also.

4.Upper alveolar ridge and hard palate

Individuals practicing reverse smoking are more vulnerable to hard palate malignancies. Early symptoms are nodules which ulcerate and lately bleeding, foul smelling discharge, blockage of nasal cavity and tooth problems like pain and loosening. Sometimes bleeding to oroantral fistula.

5.Buccal mucosa

Usually present as a swelling or ulcer inside the cheek and the angle of mouth can have foul smelling discharge. If tumour infiltrates deep to the muscle buccinator, there will be trismus. In final stages or cutaneous fistula can occur due to involvement of the cheek. Presentation as a neck node is uncommon.

6.Retromolar trigone

It has a tendancy to erode the mandible early because of less submucosal tissue there. Presentation can be pain in front of the ear or as trismus.

7.Tongue

The common site for carcinoma tongue is the middle third of the lateral margins with extension

in to the ventral aspect and floor of mouth. The growth can be proliferative with areas of necrosis. The carcinoma tongue starts as an ulcer deep in a fissure or as an ulceration with infiltration of muscles underlying. The symptoms are fetor oris, excessive salivation and ankyloglossia. Pain comes very late due to the lingual nerve involvement. Neck lumps are common due to the presence of lymph node metastasis.[20]

METHODS OF SPREAD

LOCAL SPREAD

Direct invasion allows it to spread to nearby structures. It can infiltrate and ulcerate the skin, retromolar region and alveolus.

LYMPHATIC SPREAD

The submandibular node is the first group which affected from buccal mucosa carcinoma. When the disease advances, tumor spread from these nodes to the upper deep cervical nodes. Nodes in the posterior triangle affects very rarely and occurs only in very advanced stages. The oral cancer is confined to the organ of origin, before it disseminates to the lymphnodes.[9]

HAEMATOGENOUS SPREAD

Primary tumour or lymph nodal secondaries, to the distant organs cause invasion of blood vessels.

Lung is the most common site of distant metastasis followed by liver and bone.

Investigation

1.Radiology

Xray mandible and orthopantomogram is good method for ruling out the mandibular invasion and other bone invasion. CT scan is used mainly for antral tumours, pterygoid regions, vascular and perineural invasion, clinically impalpable lymph nodes can be easily detected by CT scan.

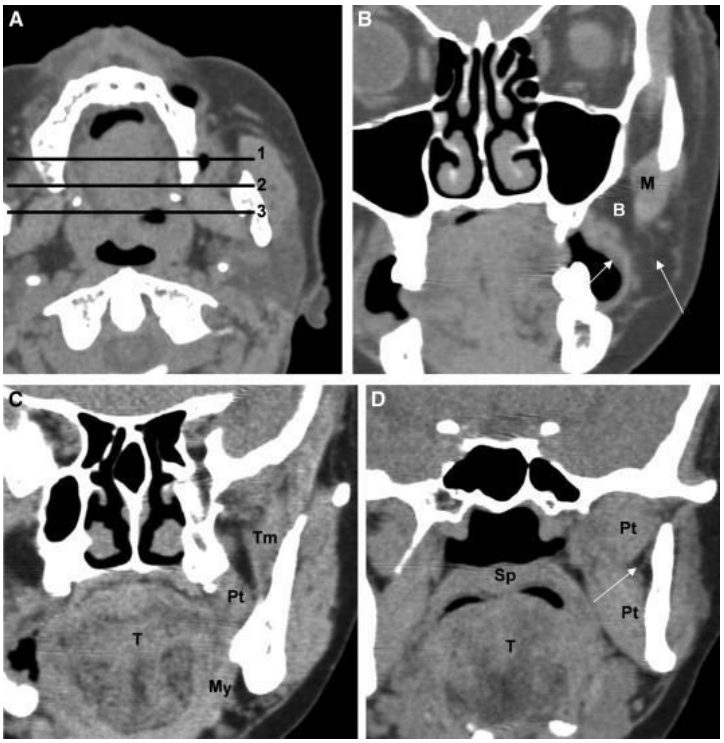


Figure 29

2.Fine needle aspiration cytology (FNAC)

FNAC is mainly used in diagnosis of malignancy in a lymph node or if the lesion present as a nodule or to find out recurrence of malignancy in the lymph node. It can also be worth in staging of the malignancy.

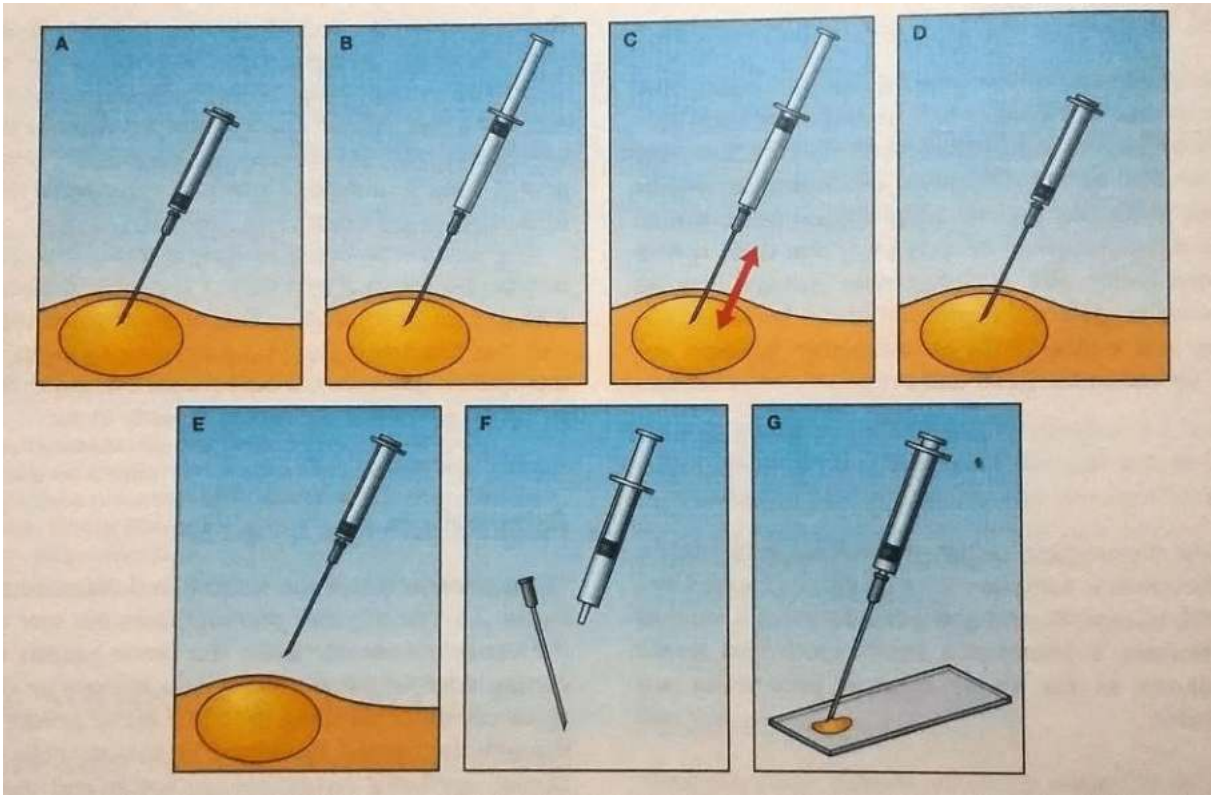


Figure 30

3.Biopsy

It is the most important investigation in establishing the diagnosis and staging oral cavity malignancies. It is a routinely done investigation for all suspected lesions in the oral cavity. Malignancy can be confirmed by positive biopsy report but cannot be excluded by a negative report.[21]

4.Esophagoscopy, Laryngoscopy, Bronchoscopy are used for more accuracy and to detect any other primary.

5.Complete blood count

6.Xray chest

7.USG abdomen

8.Dental opinion

9. VDRL

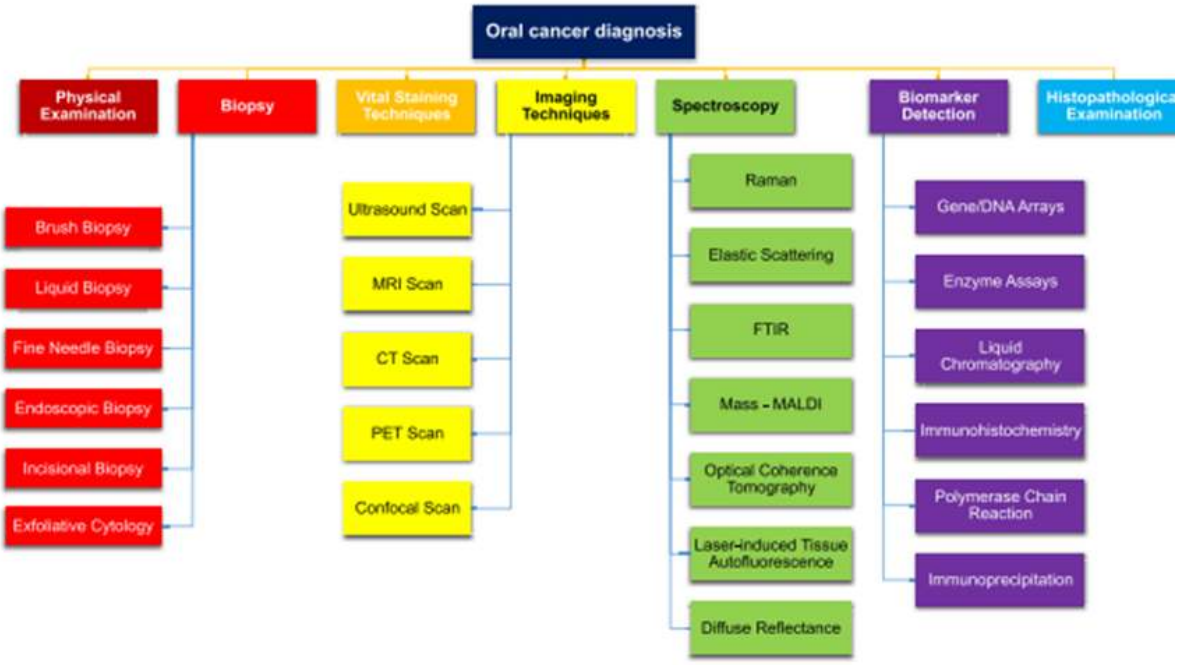


Fig 31. Techniques of oral cancer detection.[1]

Staging of oral cavity

AJCC TNM staging [9]

Primary tumor

Tx: Primary tumor cannot be assessed

T_0: No evidence of primary tumor

T_{is}: Carcinoma in situ

T_1: Tumor 2cm or less in greatest dimension and 5mm or less depth invasion.

T_2: Tumor larger than 2cm but 4cm or less in greatest dimension and depth of invasion not more than 10 mm.

T_3: Tumor larger than 4cm in greatest dimension and depth of invasion more than 10 mm

T4: Tumor (labial) invading cortical bone, inferior alveolar nerve, floor of the mouth, or facial skin

T4a: (Oral cavity) Tumor invades adjacent structures (Eg, through cortical bone, deep (extrinsic) muscles of tongue (genioglossus, hyoglossus, palatoglossus, and styloglossus), maxillary sinus and skin of face)

T4b: Tumor invades masticator space, pterygoid plates, or skull base or encases internal carotid artery (note; superficial erosion only the primary bone / gingival cavity is not sufficient to classify a tumor in T4.)

Regional lymph nodes (N)

Nx: Regional lymph nodes cannot be assessed.

N0: No regional lymph node metastasis.

N1: Metastasis in a single ipsilateral lymph node, 3 cm or less in maximum size

N2: Metastasis in a single ipsilateral lymph node, larger than 3 cm but with a maximum size of 6 cm; in multiples ipsilateral lymph nodes ,6cm or less in greatest dimension; in bilateral or contralateral lymph nodes ,6cm or less in greatest dimension

N2a: Metastasis in a single ipsilateral lymph node, larger than 3 cm but 6 cm or less in maximum size.

N2b: Metastasis in several ipsilateral lymph nodes, 6 cm or less in maximum size.

N2c: Metastasis to bilateral or contralateral lymph nodes 6cm or less in maximum size

N3: Metastasis in a lymph node larger than 6 cm.

Distant metastasis

Mx: Distant metastasis cannot be assessed

M0: No distant metastasis

M1: Distant metastasis

STAGES

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

Fig 32. Staging

Histological classification

GRADE I : Well differentiated

GRADE II : Moderately differentiated

GRADE III : Poorly differentiated[9]

Management of oral cavity malignancies

The management will vary individual to individual depending up on the site, staging, co morbidities, pathological grade of tumors and it includes single or a combination therapy of surgical excision and procedures, radiotherapy and chemotherapy.

In a study conducted with 105 patients having T1, T2, N0, N1 malignancy without metastasis, who were underwent only adequate radical surgery with the resected margins completely free of tumor; they followed up and concluded that treatment was adequate[22] {Markus et al.2000} .it is also avoiding the radiation induced side effects in oral cavity.

Early stage (I and II) oral cavity malignancy can be treated by surgery or radiation. Both modalities are having similar five-year survival rate.[23]

Advanced stages like T_3, T_4 managed by a multi-disciplinary team approach including medical and radiation oncology and head and neck surgery team. The survival rate is best with a combination of surgery and radiotherapy even though there is an increase in morbidity and side effects. [24]

Induction chemotherapy followed by surgery with or without postsurgical radiation therapy is optimum multidisciplinary therapy.[25]

According to national comprehensive cancer network guidelines.[26]

T_1 to T_2, N_0: primary tumour excision +/- unilateral or bilateral selective neck dissection or external beam RT+/- brachy therapy

T_3, N_0: primary tumour excision and reconstruction + unilateral or bilateral selective neck dissection followed by RT or CT/RT

T_1 - T_3, N_1 - N_3
T_4 any N } Primary tumour surgical excision + unilateral or bilateral selective neck dissection followed by RT or CT+RT

Unresectable: Chemo+ Radio therapy

Surgical management of oral cavity cancer

Methods of Surgery

Commonly practiced surgical methods for excision of the primary tumor are wide excision, complete oral resection, complete oral resection with hemi mandibulectomy, maxillectomy and hemiglossectomy.[27] Most of the cases the reconstruction is done by using the vascular flaps which allows healing and rehabilitation. Surgical excision preferred even in advanced stages of tumor and patients with tumor invasion of mandible.[28] Tumours approaching the mandible needs marginal mandibulectomy and invades the mandible needs hemi mandibulectomy. In case of close/doubtful margin after surgical resection should be avoided by usage of frozen section.[29,30]

Advantages: -

- Majority of the oral cancer are accessible per orally for surgical excision
- Patients with mandibular invasion and those with advanced tumor is preferred for surgical treatment [31]

  Ronald et al. conducted a retrospective study in 2003 of 105 patients who had previously untreated squamous cell carcinoma of floor of mouth or oral tongue who underwent surgical resection. 80% had simple per oral excision. In patients with T_1 lesion, 93% remined well after conservative surgery and that of T_2 lesion, 78% remined well after conservative surgery.
- Less time duration for treatment and few long-term sequelae.

Disadvantages: -

- Potential risk of anesthesia
- Functional disabilities

Mandibular involvement

Mandibular invasion of the tumor is non curable by radiotherapy and there are chances for perineural spread via inferior alveolar nerve. Direct mandibular invasion by tumor necessitates hemi mandibulectomy. Marginal mandibulectomy is considered for the tumors approaching but not invading the mandible.[32]

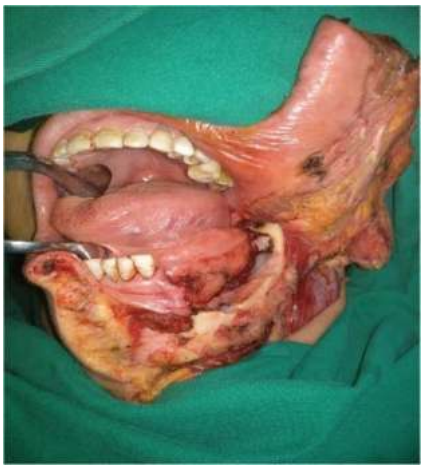


Fig 33 Marginal mandibulectomy

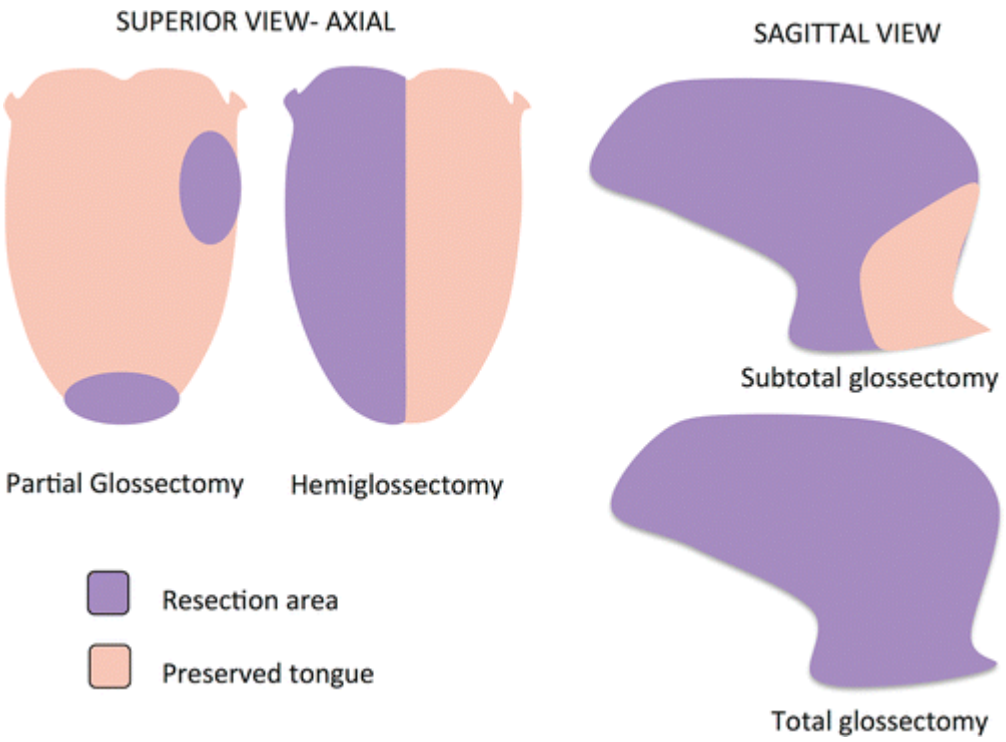


Figure34 Hemiglossectomy

Neck node management

As we discussed, neck nodes place a prognostic importance in oral cavity malignancies. There is predictable metastasis to neck nodes from oral cavity malignacies[5]. In a study it was found out that for N_0 neck dissections, staging selective neck dissection and radical neck dissection are equally effective. In case where there is an obvious pathology, modified radical neck dissection (where there is a sparing of spinal accessory nerve) and radical neck dissection are equally effective[6]. Contralateral lymph nodes should be managed in cases where the tumour is midline or approaches or crosses the midline, and for bilateral tumours. If the risk of occult neck node spread is more than 20% we should treat the clinically negative neck also.[33]

Since there are higher chances of nodal metastasis in T_3, T_4 tumours the neck treatment is mandatory for complete treatement[34]

The common technique in reconstruction are[2]

- Granulation and secondary intent
- Primary closure
- Split thickness skin grafts or allogenic dermal grafts
- Local advancement flaps
- Regional flaps
- Pedicled mucocutaneous flaps
- Free flaps
- Mandibular plating systems
- Osteo integrated implants for maxilla

Fore head flaps

For covering the defects inside the oral cavity, flaps from hairless areas of forehead are taken. The base of flap extends laterally from lateral edge of eyebrow to the anterior border of pinna. The vessel for flap is anterior branch of superficial temporal artery. The plane of dissection of the flap between the pericranium and aponeurosis where the loose areolar tissue is present.

Deltopectoral flap

It is an axial flap, blood supply based on first 3 to 4 perforating branches of internal mammary artery. It is commonly used in covering the defect outside the oral cavity than in side defects.

Pectoralis major flap

The blood vessel support is from pectoral branches of acromio thoracic artery.

Based from the area below and medial to nipple, muscle flap raised along with overlying skin and subcutaneous tissue. It is brought to cover the mucous membrane defect in oral cavity by tunnelling through a lower neck incision.

Techniques of radiotherapy in oral cancer management

Radiotherapy has an active role in early and advanced disease. The radiotherapy is meant to ensure a permanent and long term locoregional control of tumor. It is an alternative to mutilating operative procedures with a better quality of life than after a functional conservation surgery.

Principles of radiotherapy

- The volume of the primary tumour site that requires a high dose of radiation must contain a margin outside of all cancer cells that is equivalent to the volume of the primary tumour site that must be surgically removed**.**
- Regional lymphnodes and other structures which are typically included in continuity with the main tumour, are frequently gets irradiated along with primary tumor.
- Post-operative radiation at a dose of 4500–6000 cGy following excision of all grossly detected disease results in a very high frequency of tumour control with minimal detectable sequelae.

For head and neck cancer, the usual radiation treatment schedule consists of five daily treatment per week, fractions administered for five to seven continues weeks. 180 cGY to 225 cGY per fraction is the daily dose.

External beam radiotherapy (XRT)

- XRT: It is used as a curative treatment as well as in clinically positive neck nodes

  for tumour:70-75 gy

  for clinically positive node:65-70 gy

  elective neck node irradiation: 40-50 gy

- XRT with brachy therapy

  initially the tumour is given XRT at a dose of 45-50 gy followed by (2 weeks later) iridium wire implantation which deliver at a dose of 25-30 gy.

- XRT followed by surgery

  The radiation given at the tumour site and neck at a dose of 50 gy.

- XRT preceded by surgery

  in case of a sufficient local resection only T4 stage needs a post-operative irradiation at a dose 60 gy for avoiding local recurrence, for positive lymph nodes 50 gy dose is giving and 60 gy is given if there is suspected spillage of tumour, capsular rupture or perineural spread.[35]

Advantages:

- Tumour shrinkage in a centripetal manner.
- Lymphatics sterilization.
- Adequate clearance.
- It is very much useful in the sites where there is difficult surgical exposure.
- Less functional disability in terms of speech and deglutition.

Disadvantages:

- For late stages tumours, the response to XRT is minimal.
- There are side effects like mucositis, osteoradionecrosis, xerostomia.
- Salvage surgery after radiation failure will be having high morbidity and mortality.[36]
- Treatment duration is very low.

2.**Brachytherapy** Most commonly used for tumours in T_1 and T_2 stage which are accessible. It is a conservative method of treatment; the radiation volume is well defined.

Figure 35 Radiotherapy unit

Indications of radiotherapy

1. Control of small (T_1-T_2) tumours.

2. Presurgical: primary tumour along with regional node is radiated with 50gy dose which showed the reduction of tumour size and destruction of effect cell which made the surgery easier.[37]

3. Post-surgical: patients treated by surgical ablation followed by adjuvant RT have shown better survival and reduction in recurrence compared to combination therapy of RT and platinum based CRT.[38]

4. Elective management of clinically negative nodes. A radiation dose of 40 to 50 gy can be delivered to the neck where a higher rate of relapse is anticipating in more than 90 % of lymph node.

5. Post-surgical recurrence of tumour

6. Palliation

Adverse effects of radiotherapy

Early

- Mucositis
- Xerostomia
- Dysgeusia
- Difficulty in swallowing, difficulty in breathing
- Erythema and epidermolysis.

Late

- Necrosis of soft tissue
- Osteoradionecrosis of the mandible
- Hypothyroidism

Chemotherapy

In a study conducted among 26 patients who all were in a very late stage of head and neck squamous carcinoma are managed with Bleomycin, Oncovin, Mitomycin C and Methotrexate (BOMM) for a duration of 10 weeks. On responders and partial responders were treated with Adriamycin, Cisplatin and Cyclophosphamide (APC). There was 65% response rate to BOMM regimen whereas only 20% for APC regimen.[39]

Chemotherapeutic agents

Single agent

Single most effective agents include Cisplatin /5-FU /Methotrexate/Bleomycin.

Combination therapy

Cisplatin and 5-FU are the most effective combination regimen.

Other combinations are: -

- Cisplatin/Paclitaxel
- Carboplatin/ Infusional 5FU
- 5FU/ Hydroxyurea
- Cefuximab

Adjuvant chemotherapy

There is no effect proven in the disease-free survival but there is some reduction in the distant metastasis rate.[40]

Neoadjuvant chemotherapy

Results in 60 to 90 percent tumour regression and 20 to 25 percent incomplete responses.[41]

Various drugs used for chemotherapy

Cisplatin

Dose :50 -100 mg/m^2 bsa iv every 3 weeks

5 FU

Dose:800 – 1000 mg/m^2 bsa iv 3 – 5 days cycle every 28 days

Methotrexate

Dose: 2.5 – 5 mg daily, po; or 40 mg/m^2 iv weekly with leucovorin.

Vincristin

Dose: 2 – 10 mg/m^2 bsa, every 3 weeks

Cyclophosphamide

dose: 10-15 mg/m^2 iv every 2 – 3 weeks

Mitomycin

Dose:1.5 mg/m^2 iv weekly

Bleomycin

dose: 10 – 20 mg/m^2 iv once or twice weekly.

Summary of Role of Radiotherapy and Chemoradiotherapy in Oral Cavity Cancer.[42]

	External Beam Radiotherapy (EBRT)	Chemotherapy	Interstitial Brachytherapy
Primary setting	• Early disease when patient intolerant of surgery • Early disease when anticipated cosmetic consequence of surgery is a concern, especially for lip cancer involving commissure • Unresectable disease, usually combined with chemotherapy • Advanced disease for patients intolerant of surgery due to poor performance status or comobidities	• Advanced disease or unresectable disease, in combination with radiotherapy	• Early and superficial well-defined tumor located more than 5 mm from the mandible
Adjuvant setting	• Unfavorable pathological features • Combined with chemotherapy for positive resection margins and extracapsular nodal extension	• Combined with radiotherapy for positive resection margins or extracapsular nodal extension	• Brachytherapy alone for positive resection margins • In combination with external beam radiotherapy to augment radiotherapy dose to the high risk area
Salvage setting	• Adjuvant treatment after salvage surgery • Primary treatment modality, usually combined with chemotherapy if further surgery is not feasible	• Combined with radiotherapy	• Especially useful for re-irradiation: – for persistent or recurrent disease after previous radiation, – 2nd primary cancer occurrence within previous radiation field

Figure 36

The generalized postsurgical management can be simplified as below as it may vary from case to case.

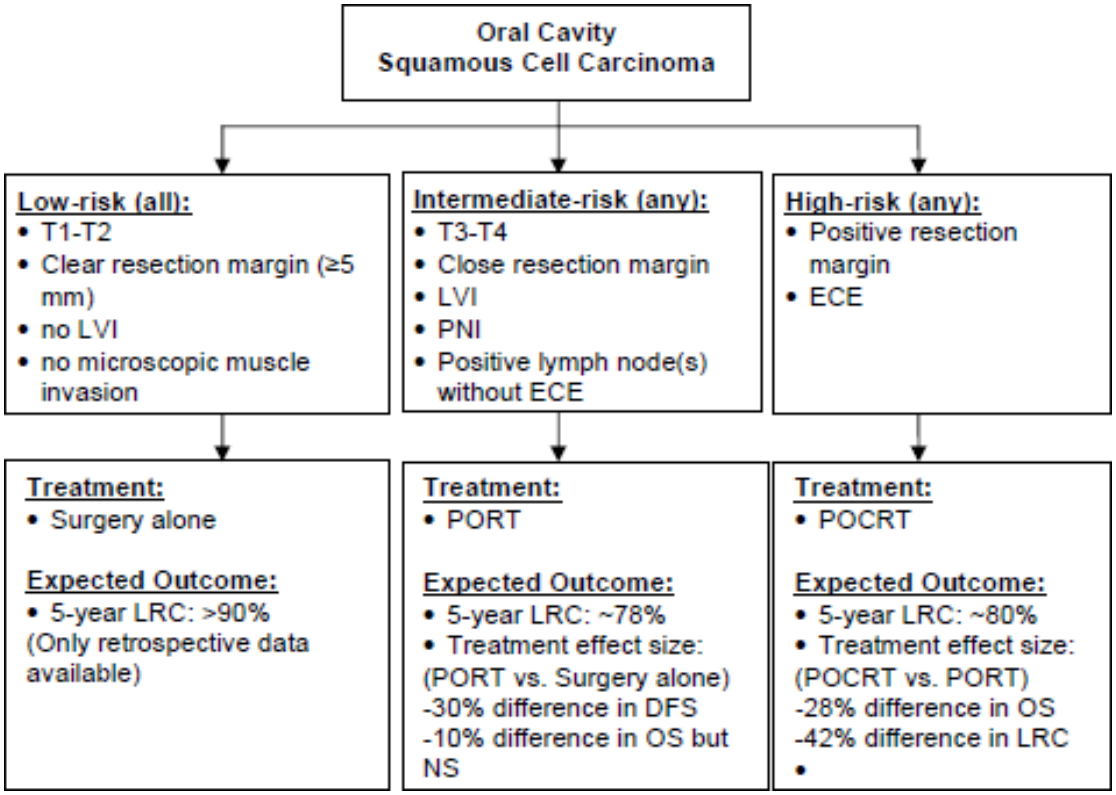


Fig.37 Summary of Risk Grouping and Role of Postoperative Radiotherapy +/- Chemotherapy. Abbreviations: LVI: lympho-vascular invasion; ECE: extra-capsular invasion; PORT: postoperative radiotherapy; POCRT: postoperative chemoradiotherapy; LRC: locoregional control; DFS: disease-free survival; OS: overall survival; NS: not statistical significant.

Epithelial growth factor receptor (EGFR) targeting biological agents are emerged as a potential modality in combination with radiotherapy or chemoradiotherapy and are currently under evaluation in clinical trials.[42]

MATERIALS AND METHODS

The purpose of the study is to evaluate various management modalities for oral cancer in our hospital, comparison of different modalities of treatment in management of oral cancers and identify the risk factors associated with oral cancers.

Type of study: Prospective

Study period: 1 year 6 months

Settings: Department of ENT and Head and Neck Surgery, Govt Stanley Medical College and Hospital, Chennai-1

Department of Surgical Oncology and Radiotherapy, Govt Stanley Medical College and Hospital, Chennai-1

Department of Dental Surgery, Govt Stanley Medical College and Hospital, Chennai-1

INCLUSION CRITERIA:

- Any patients having pathologically proven malignancy in any one or more sub site of oral cavity
- For all patients having oral cancers at any stage of presentation
- For patients having oral cancer at any age of presentation.

EXCLUSION CRITERIA:

a) Terminally ill patients

b) Patients not giving consent for study

c) Patients not willing for treatment.

Methodology

Patients with suspected lesion in the oral cavity (seven subsites) from ENT department, Surgical oncology and radiotherapy department and also from dental surgery department were identified and evaluated. Patients with biopsy proven lesion are directly taken in to the study. Patients with suspected lesions were biopsied and after getting the positive biopsy report for malignancies were entered in the study. All the patients underwent imaging studies in order to stage the disease correctly. After planning of the treatment by a multidisciplinary approach patient were evaluated with a questionnaire which includes details of the cancer like subsites, histology, grading, TNM staging, and grading of symptoms like pain, bleeding, swallowing and trismus. 6 weeks after completion of the treatment, patients were reevaluated for response of the malignancies and the difference in symptoms. The pretreatment evaluation findings and posttreatment evaluation findings were compared in terms of symptoms and response to the treatment. The evaluation of pain is carried out by Universal pain assessment scale which is given in below

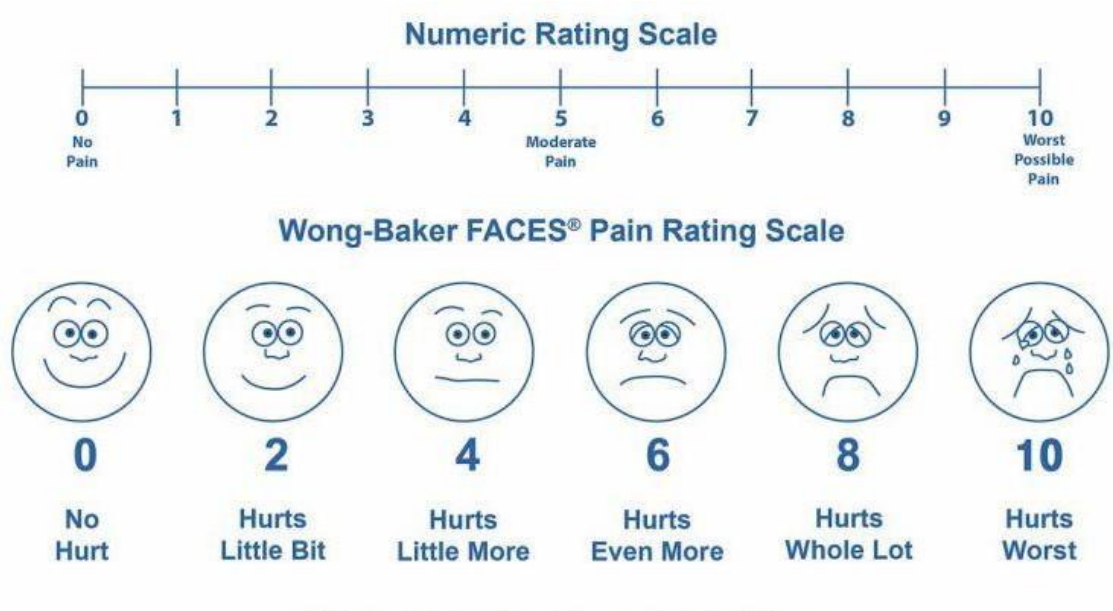


Figure 38

Symptoms like bleeding and swallowing carried out by clinical grading as

Zero – No difficulty in swallowing

1 - Mild difficulty in swallowing

2 – Moderate difficulty in swallowing

3 – Severe difficulty in swallowing

Trismus was assessed clinically as

1Finger(1F) – Severe trismus

2Finger(2F) – Moderate trismus

3Finger(3F) – Mild trismus

4Finger(4F) – Absent of trismus

The response to treatment after six weeks of completion of treatment was assessed using WHO evaluation response scale

	WHO
Method	Sum of products of two longest diameters in perpendicular dimensions (bidimensional; surface area)
No. of measured lesion	All lesions
Response	
CR	Disappearance of all known disease, confirmed at 4 wk[2]
PR	≥ 50% decrease from baseline, confirmed at 4 wk
SD	Neither PR nor PD criteria met
PD	≥ 25% increase, no CR, PR, or SD, new lesion (s), ≥ 25% increase in 1 lesion

Figure 39

The sample size was estimated by using nMaster software Version 2.0 by applying following details in the formula.

Formula

$$n = \frac{Z^2_{1-\alpha/2}\, p(1-p)}{d^2}$$

Where,

p : Expected proportion

d : Absolute precision

1- α/2 : Desired Confidence level

On substituting, in the formula

Single proportion - Absolute precision	
Expected proportion	0.96
Precision (%)	6
Desired confidence level (1- alpha) %	95
Required sample size	**41**

Based on the above parameter with an alpha of 0.05 (2 sided), the estimated sample size using the sample size formula for Single proportion. Where Z = 1.96 (statistically significant constant for 95% CI), p =96% (Proportion as a precipitating factor from previous study.), q = 4% (100-p), d = 6% absolute precision.

The above parameter and formula give us a sample size of 41 subjects, adding 20% non-response rate (20% of 41=9.5), the sample size of 50 was included in the study.

RESULTS AND OBSERVATIONS

The collected data were analysed with IBM SPSS Statistics for Windows, Version 23.0(Armonk, NY: IBM Corp). To describe about the data descriptive statistics frequency analysis, percentage analysis was used for categorical variables and the mean & S.D were used for continuous variables. To find the significant difference between the bivariate samples in Paired groups the Wilcoxon signed rank test was used. To find the significance in categorical data Chi-Square test was used. In the above statistical tools, the probability value 0.05 is considered as significant level.

1. ANALYSIS OF AGE DISTRIBUTION

Age distribution		
	Frequency	Percent
Upto 35 yrs	3	6.0
36 - 45 yrs	15	30.0
46 - 55 yrs	14	28.0
56 - 65 yrs	14	28.0
66 - 75 yrs	4	8.0
Total	50	100.0
Mean ± SD = 51 ± 11 yrs.		

Table 1

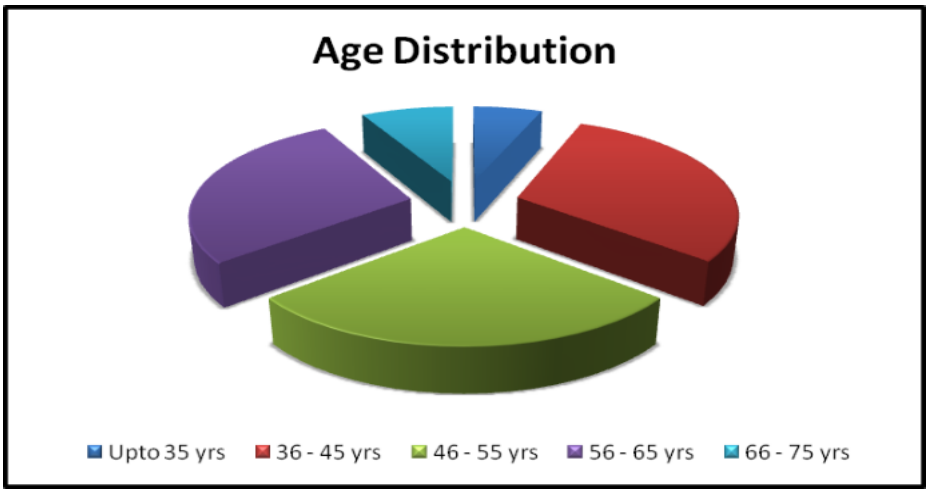


Figure 40

The above table shows the age distribution of the study population which consist of 6.0 % up to 35 years, 30% is 36 to 45 years, both 46 to 55 years and 56 to 65 years were 28% and 8% 66 to 75 years respectively, the mean ± standard deviation of the age were 51±11 years. The highest presentation was in the 36-45 years age group followed by equal distribution among the age group 46-55 and 56-65 followed by the age group 66-75 and only 6 % distribution among the age group less than 35, with mean distribution in the sixth decade

2.ANALYSIS OF GENDER DISTRIBUTION

Gender distribution		
	Frequency	Percent
Female	12	24.0
Male	38	76.0
Total	50	100.0

Table 2

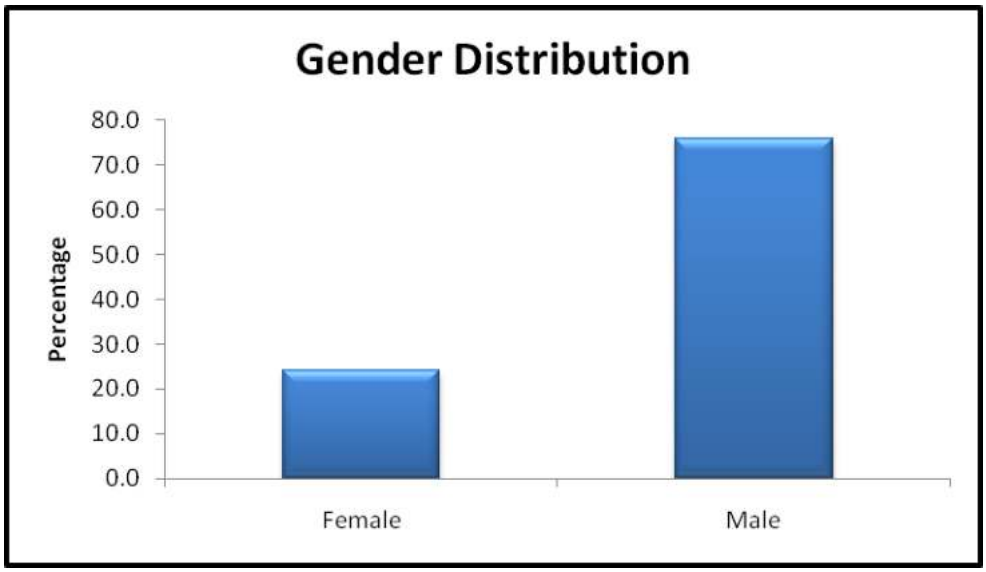


Figure 41

Out of 50 total population, 38 patients were male and 12 were females. The above table shows the gender distribution of the study population which consists of 24% female and 76% male. There is a male predominance with a male to female ratio of 3.1:1.

3a. ANALYSIS OF STAGE DISTRIBUTION

Stage	Number	Percentage (%)
Stage I	12	24
Stage II	16	32
Stage III	11	22
Stage IV	11	22

Table 3a

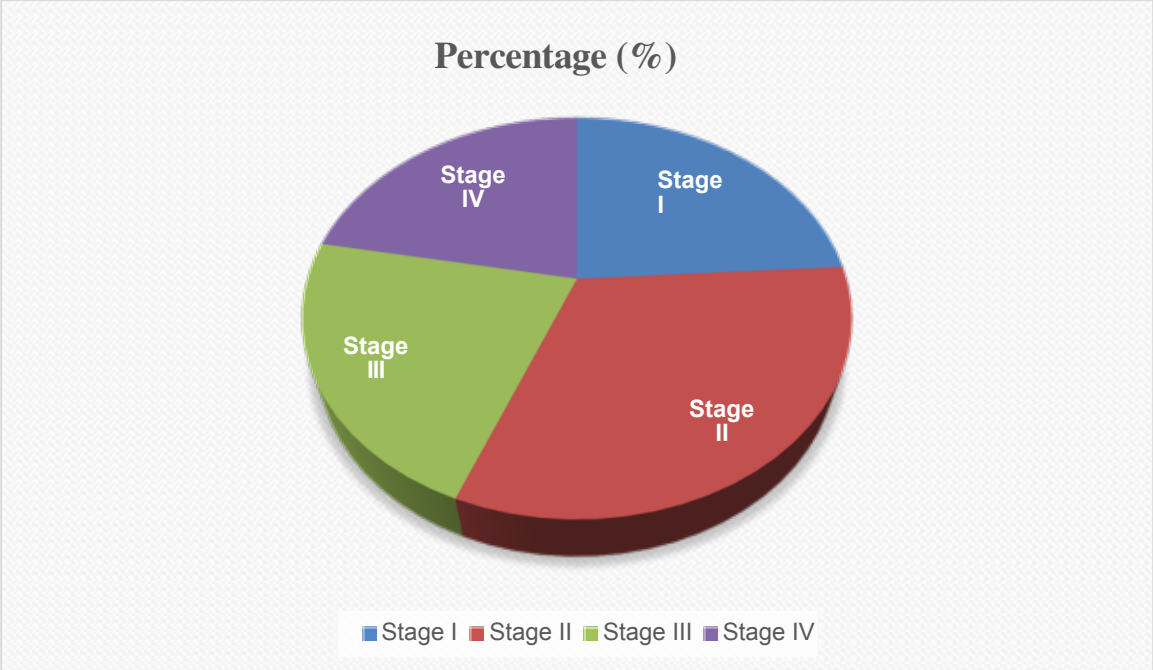


Figure 42

Out of 50 study populations, 24% presented at stage I, 32 % presented at stage II, and 22 % each for stage III and stage IV. The majority are presented in stage II followed by stage I, followed equally by stage III and stage IV.

3b. ANALYSIS OF STAGE DISTRIBUTION

Early (Stage I and Stage II)	56%
Late (Stage III and Stage IV)	44%

Table 3b

Stage I and stage II are considered as early-stage whether stage III and stage IV are considered as late-stage diseases. 56% of the study population presented in the early stage and 44 % were in the late stage. Majority of the study population presented in the early-stage disease.

4.ANALYSIS OF RISK FACTORS DISTRIBUTION

Risk factors distribution		
	Frequency	Percent
Alcohol	26	52.0
Betel nut	36	72.0
Smoking	22	44.0
Spicy food	12	24.0
Sharp tooth	7	14.0
Chronic oral sepsis	1	2.0
Pan masala	17	34.0
NIL	1	2.0

Table 4

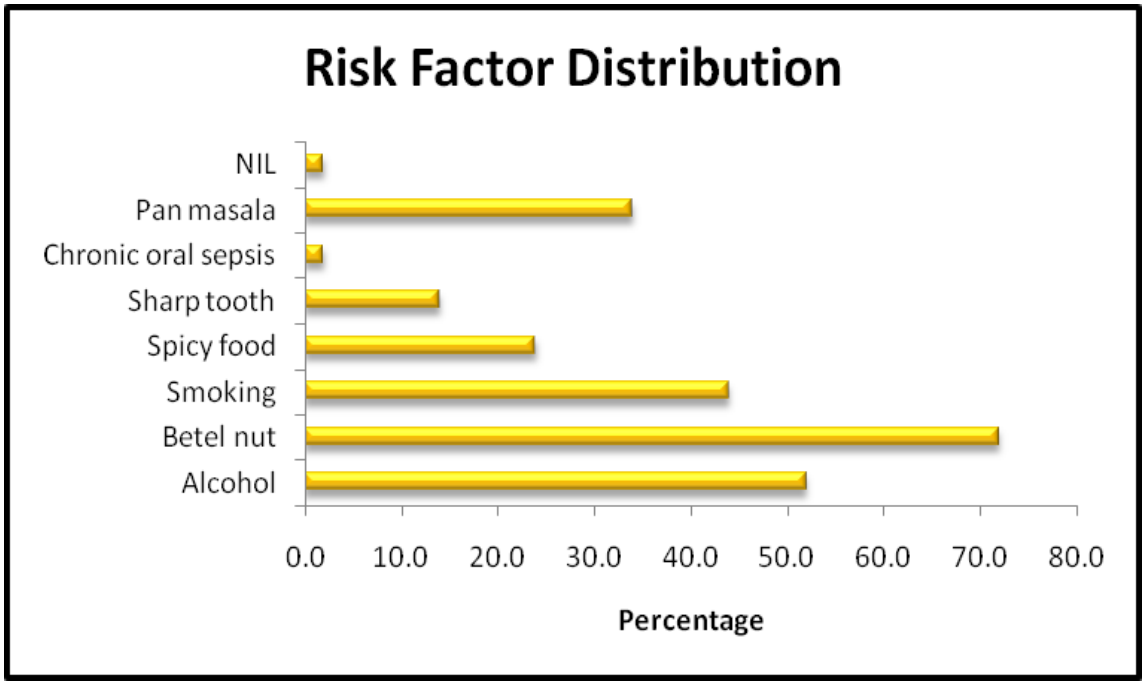


Figure 43

The above table shows the risk factors distribution of the study population which consists of 52% were alcoholic, 72% of them with betel nut habit, 44% of them were smokers, 24% of them were spicy food eaters, 14 % risk was due to sharp tooth,1% chronic oral sepsis, 34% of them were pan masala eaters and 2% were found with no risk factors. On analyzing the risk factors betel nut chewing was found to be the major risk factors in our study followed by alcohol, smoking, pan masala, spicy food, sharp tooth in the respective decreasing order with chronic oral sepsis was the least common risk factor. 2% of the study population was not having any risk factors.

5. ANALYSIS OF SUBSITE DISTRIBUTION

Subsite distribution		
	Frequency	Percent
Alveolus	1	2.0
Floor of mouth	1	2.0
Hard palate	7	14.0
Lip	1	2.0
Oral tongue	22	44.0
Buccal mucosa	18	36.0
Total	50	100.0

Table 5

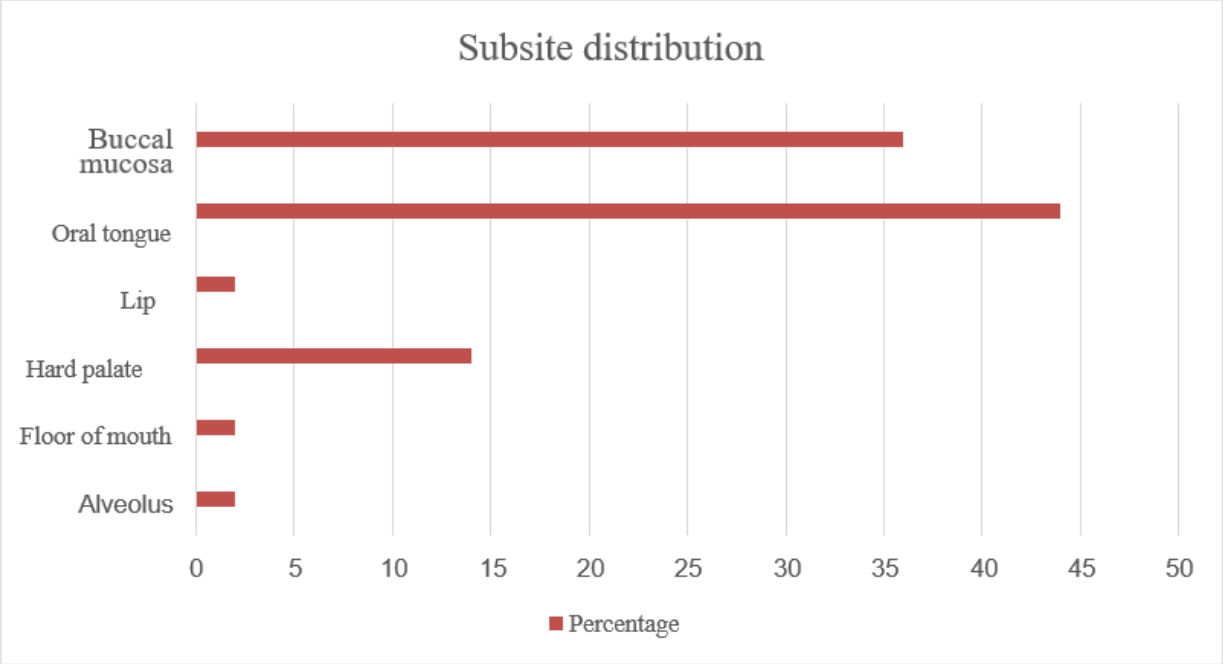


Figure 44

The above table shows the subsite distribution of the study population which consists of alveolus, floor of mouth & left commissure of lip 2%, hard palate 8%, oral tongue 44% and buccal mucosa 36%. In the majority of study population oral tongue was the commonest subsite followed by buccal mucosa, hard palate, respectively. The alveolus, floor of mouth and commissure of lip showed equal and low subsite presentation of oral cavity malignancy. In the buccal mucosa right and left side showed equal predominance. In case of hard palate, right side shows higher incidence than the left side.

6.ANALYSIS OF TYPE DISTRIBUTION

Type of CA distribution		
	Frequency	Percent
Adenoid cystic carcinoma	1	2.0
Mucoepidermoid carcinoma	1	2.0
Squamous cell carcinoma	46	92.0
Verrucous carcinoma	2	4.0
Total	50	100.0

Table 6

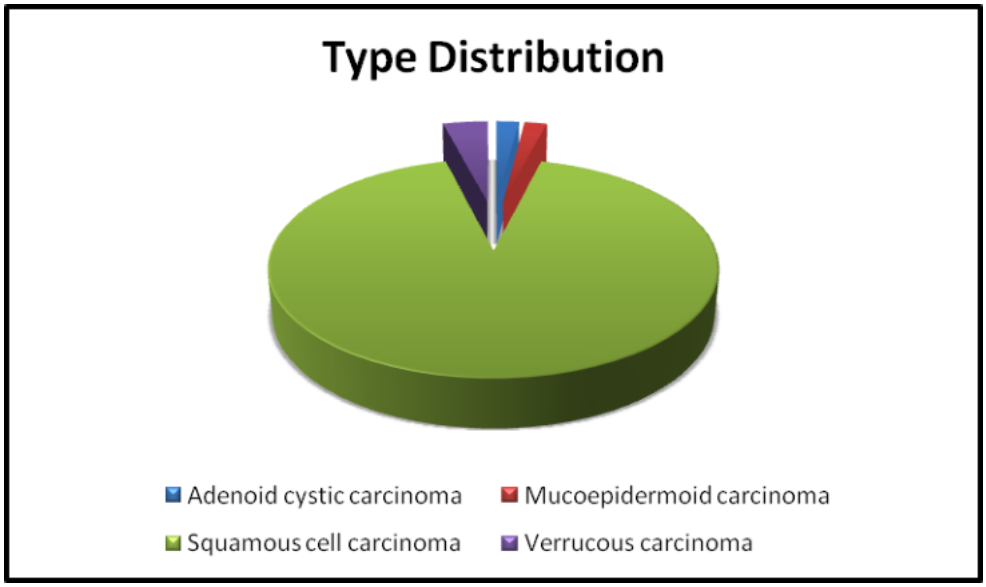


Figure 45

The above table shows the types of oral carcinoma distribution of the study population which consists of adenoid cystic carcinoma & mucoepidermoid carcinoma were 2%, verrucous carcinoma 4% and 92% of squamous cell carcinoma. Majority of the study population presented as squamous cell carcinoma followed by verrucous carcinoma. Adenoid cystic carcinoma and mucoepidermoid carcinoma showed equal and least predominance and both were located in the hard palate. Out of 50 study population 46 were diagnosed with squamous cell type and only single cases for adenoid cystic carcinoma and mucoepidermoid carcinoma type.

7. ANALYSIS OF GRADE DISTRIBUTION

Grade distribution		
	Frequency	Percent
Well differentiated	22	44.0
Moderately differentiated	28	56.0
Poorly differentiated	0	0.0
Total	50	100.0

Table 7

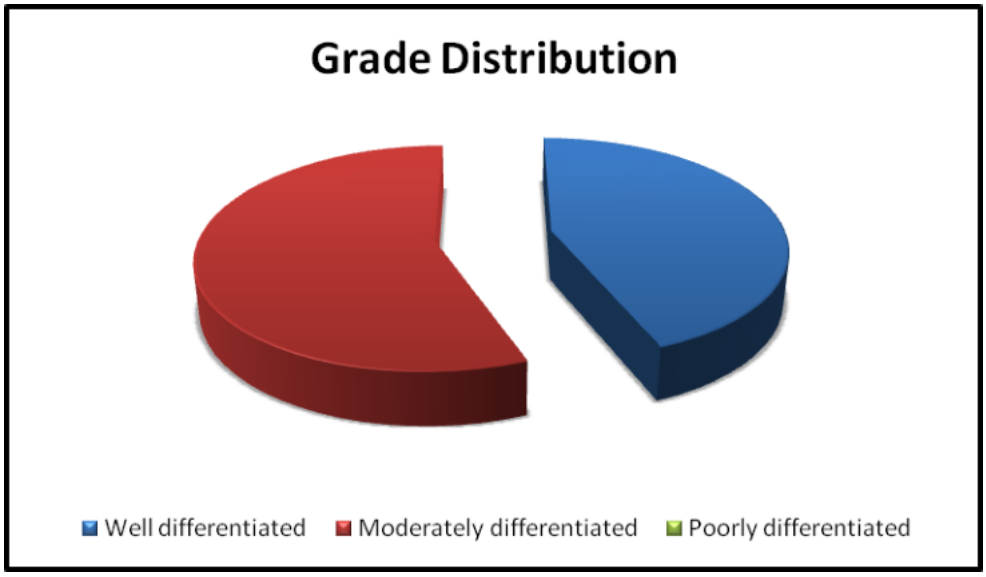


Figure 46

The above table shows the grades distribution of the study population which consists of 44% were well differentiated, 56% were moderately differentiated and no patients were poorly differentiated. Majority of the patients had moderately differentiated pathologic grade followed by well differentiated grade and none of the patients had poorly differentiated pathological grade.

8. ANALYSIS OF TREATMENT DISTRIBUTION

Treatment distribution		
	Frequency	Percent
Chemotherapy+Radiotherapy	36	72.0
Radiotherapy	3	6.0
Surgery	6	12.0
Surgery+Chemotherapy+Radiotherapy	3	6.0
Surgery+Radiotherapy	2	4.0
Total	50	100.0

Table 8

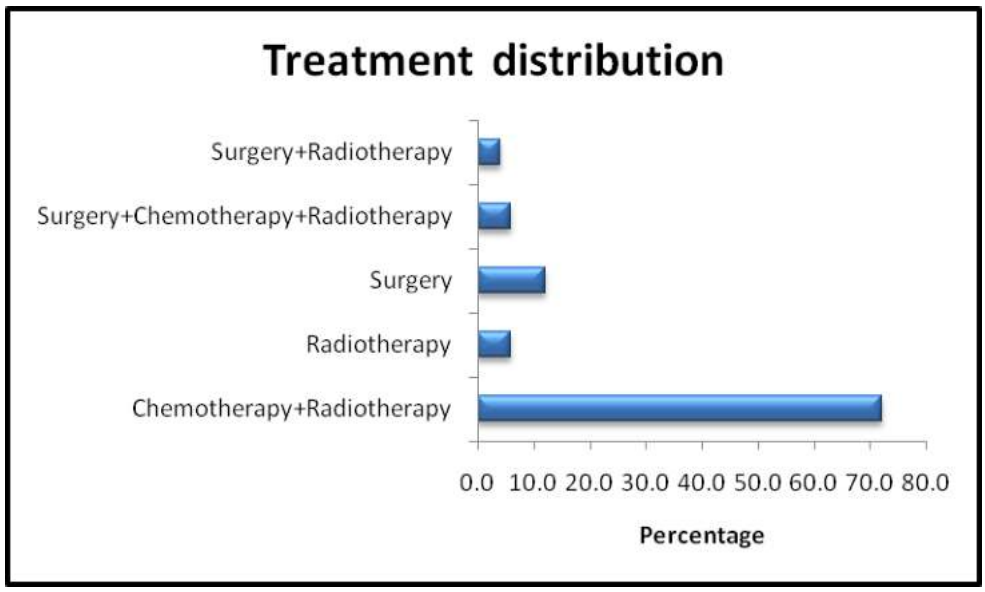


Figure 47

Out of 50 study population 36 patients underwent combination of chemotherapy and radiotherapy. Six patients underwent only surgical modality of treatment. Three patients underwent only radiotherapy and three patients underwent a combination of surgery, chemotherapy and radiotherapy. Only two patients underwent a combination of surgery followed by radiotherapy. The above table shows the treatment distribution of the study population which consists of 73% with chemotherapy+radiotherapy, 6% radiotherapy, 6% with surgery + chemotherapy radiotherapy, surgery+radiotherapy is 4% and 12% underwent the only surgery. Majority of the

people underwent chemotherapy + radiotherapy followed by people underwent only surgery. Least majority of people underwent surgery followed by radiotherapy.

9.ANALYSIS OF SURGERY DISTRIBUTION

Surgery distribution		
	Frequency	Percent
Composite resection + PMMC flap	1	2.0
Composite resection + PMMC flap+Modified radical neck dissection	1	2.0
Hemiglossectomy	1	2.0
Hemiglossectomy+ Modified radical neck dissection	1	2.0
Total maxillectomy	1	2.0
Inferior partial maxillectomy	1	2.0
Wedge resection + advancement flap	1	2.0
Wide local excision	3	6.0
Wide local excision +Marginal mandibulectomy	1	2.0

Table 9

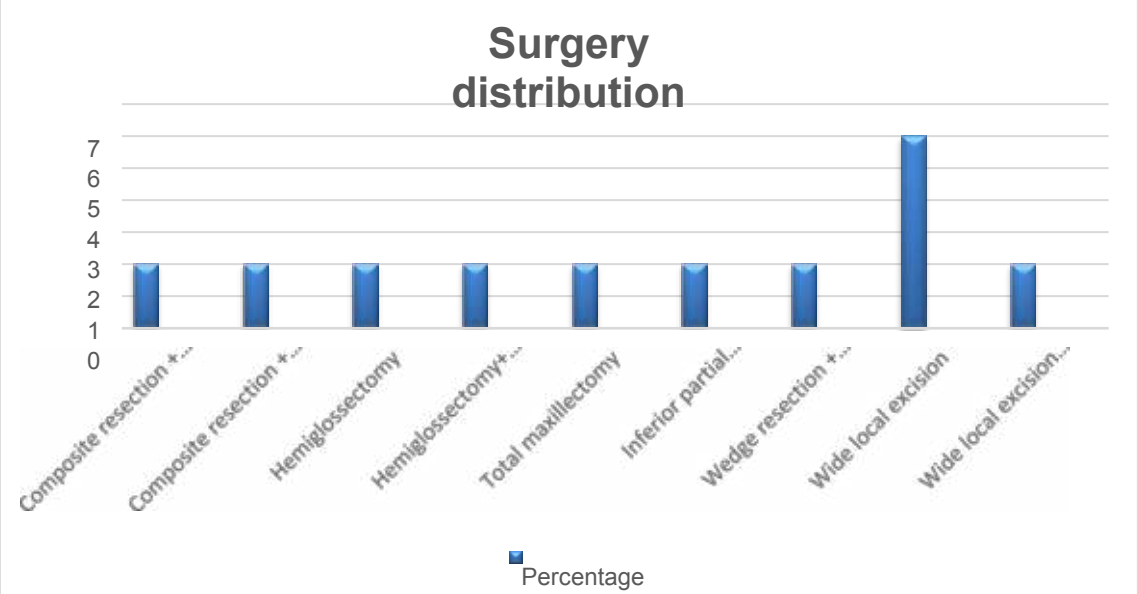


Figure 48

The above table shows the types of surgery distribution of the study population who underwent surgery. In our study only 11 patients underwent surgical management of which the majority underwent wide local excision. Only 3 patients out of 50 underwent wide local excision. One patient underwent wide local excision with marginal mandibulectomy. One patient underwent

wedge resection with advancement flap. One patient underwent inferior partial maxillectomy and one patient underwent total maxillectomy. One patient underwent hemiglossectomy and another one patient underwent hemiglossectomy with modified radical neck dissection. One patient underwent composite resection with pectoralis major myocutaneous flap (PMMC) and another patient underwent composite resection with PMMC flap with modified radical neck dissection.

10. ANALYSIS OF RESPONSE DISTRIBUTION

Response distribution		
	Frequency	Percent
Complete	21	42.0
Partial	23	46.0
Progressive	6	12.0
Total	50	100.0

Table 10

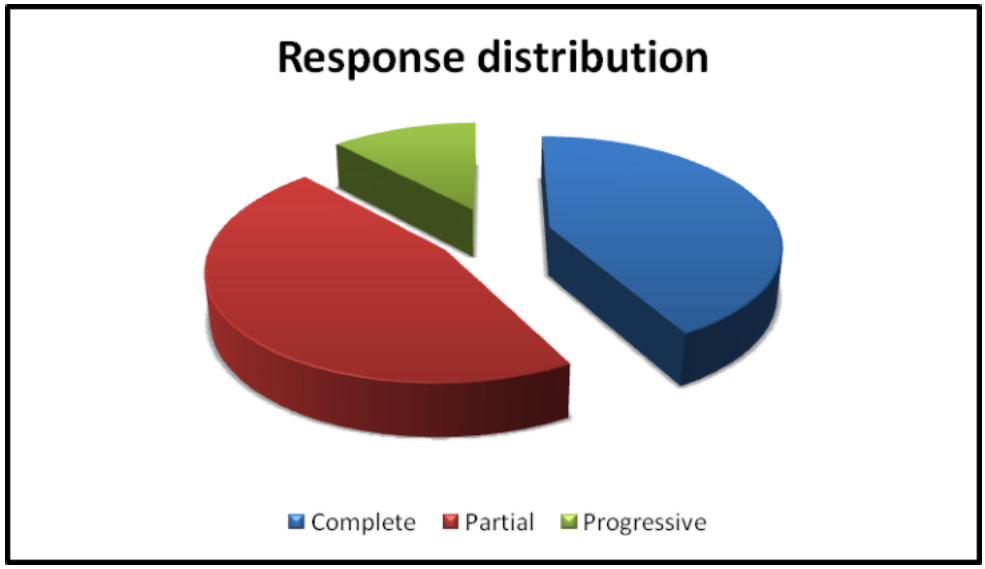


Figure 49

Response was analyzed after a duration of six week after completion of treatment using WHO response evaluation criteria in solid tumour. Out of 50 study population 21 patients showed complete response, 23 showed partial response and 6 patients showed progressive disease. The

above table shows the response distribution of the study population which consists of 42% complete response, partial response is 46% and 12% progressive disease. Majority of the patients showed only partial response to all treatment modalities and only 42 % showed complete response to various treatment modalities. 12% of study population showed progressive diseases despite of various treatment modalities.

11. ANALYSIS OF COMPARISON OF PAIN SCORE BETWEEN PRE AND POST TREATMENT BY WILCOXON SIGNED RANK

Pain score	Mean	N	SD	Z-value	p-value
Pre	6.5	50	2.1	5.623	0.0005 **
Post	3.7	50	2.5		
** Highly Statistical Significance at P < 0.01 level					

Table 11

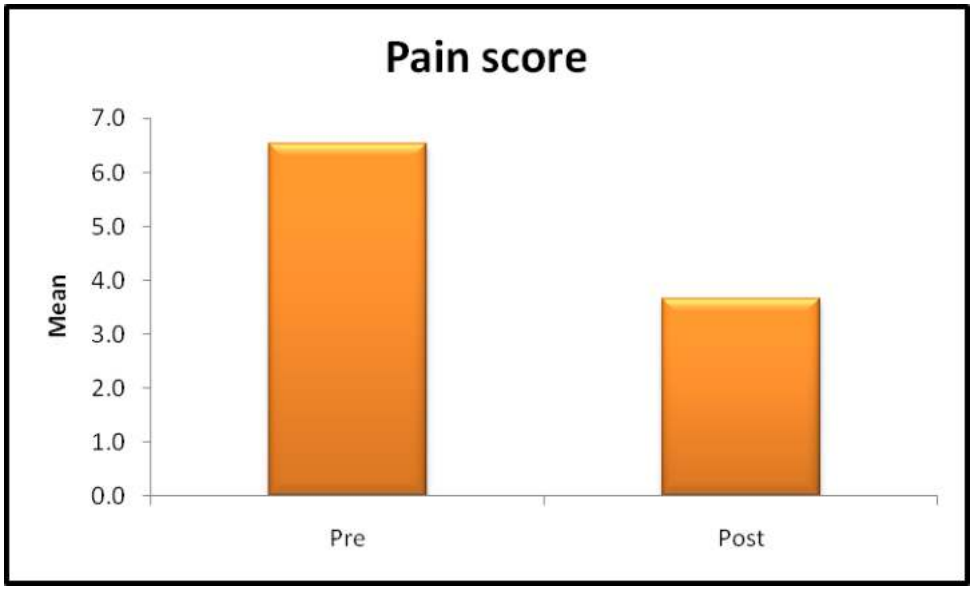


Figure 50

The above table shows the comparison of pain between pre and post treatment by using Wilcoxon signed rank test, which shows that there is a highly statistically significant difference between pre- and post-treatment in pain with mean ± standard deviation 6.5 ± 2.1 at pre after the treatment it

becomes 3.7± 2. Z value=5.623, p-value= 0.0005< 0.01. There is a significant reduction in terms of pain noted in the post treatment evaluation of all treatment modalities. Pretreatment and post treatment pain evaluation was carried out with universal pain assessment tool for patient self-assessment.

12. Comparison of bleeding between pre- and post-treatment by Pearson Chi square

Bleeding			Post treatment		Total
			No Symptoms	Mild	
Pre treatment	No symptoms	Count	22	0	22
		%	44.0%	0.0%	44.0%
	Mild	Count	19	4	23
		%	38.0%	8.0%	46.0%
	Moderate	Count	4	1	5
		%	8.0%	2.0%	10.0%
Total		Count	45	5	50
		%	90.0%	10.0%	100.0%

Table 12

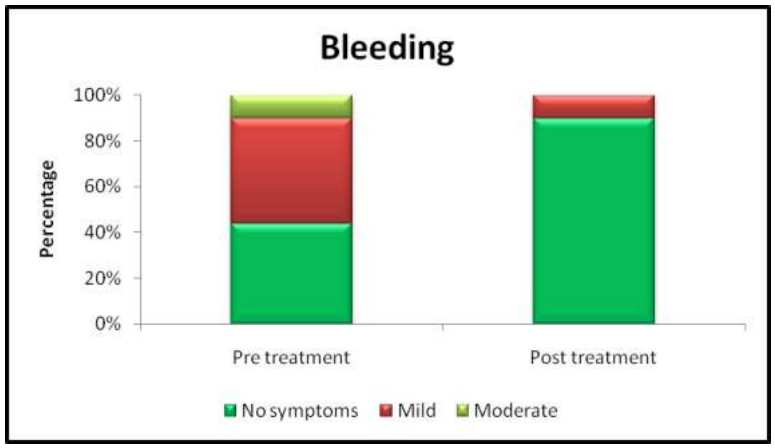


Figure 51

The above table shows the comparison of bleeding between pre and post treatment by using Pearson Chi-Square test, which shows that there is no statistically significant difference between pre- and post-treatment in bleeding, Chi-Square value = 4.396, p-value= 0.111> 0.05. Even though

there is no statistical significance the difference between pre and post treatment but the percentage of no symptoms were increased from 44% from pre to 90 % in the post treatment.

13. ANALYSIS OF COMPARISON OF TRISMUS BETWEEN PRE AND POST TREATMENT BY PEARSON CHI SQUARE

Trismus			Post treatment					Total
			1F	2F	3F	4F	Absent	
Pre treatment	1F	Count	2	2	4	0	0	8
		%	4.0%	4.0%	8.0%	0.0%	0.0%	16.0%
	2F	Count	0	8	10	1	0	19
		%	0.0%	16.0%	20.0%	2.0%	0.0%	38.0%
	3F	Count	1	1	8	0	1	11
		%	2.0%	2.0%	16.0%	0.0%	2.0%	22.0%
	Absent	Count	0	1	0	0	11	12
		%	0.0%	2.0%	0.0%	0.0%	22.0%	24.0%
Total		Count	3	12	22	1	12	50
		%	6.0%	24.0%	44.0%	2.0%	24.0%	100.0%

Table 13

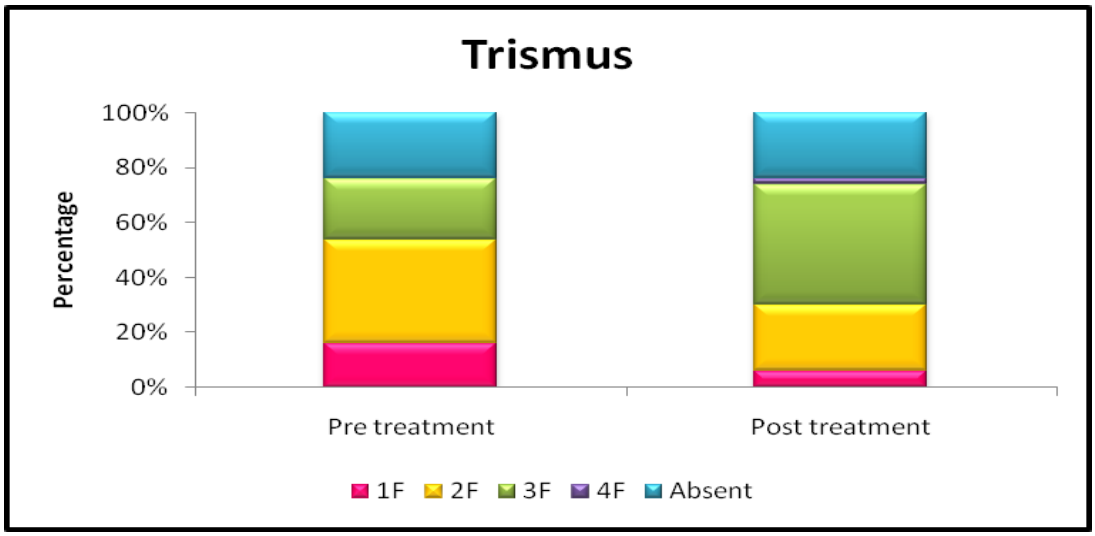


Figure 52

The above table shows the comparison of trismus between pre and post treatment by using Pearson Chi-Square test, which shows that there is highly statistically significant difference between pre-

and post-treatment in trismus, Chi-Square value = 51.447, p-value=0.0005 < 0.01. There is a significant reduction of trismus in the post treatment evaluation of all treatment modalities all together. The pretreatment and post treatment evaluation are carried out clinically by finger test.

14.ANALYSIS OF COMPARISON OF SWALLOWING BETWEEN PRE AND POST TREATMENT BY PEARSON CHI SQUARE

Swallowing			Post treatment			Total
			No Symptoms	Mild	Moderate	
Pre treatment	No symptoms	Count	34	0	1	35
		%	68.0%	0.0%	2.0%	70.0%
	Mild	Count	10	4	0	14
		%	20.0%	8.0%	0.0%	28.0%
	Moderate	Count	0	0	1	1
		%	0.0%	0.0%	2.0%	2.0%
Total		Count	44	4	2	50
		%	88.0%	8.0%	4.0%	100.0%

Table 14

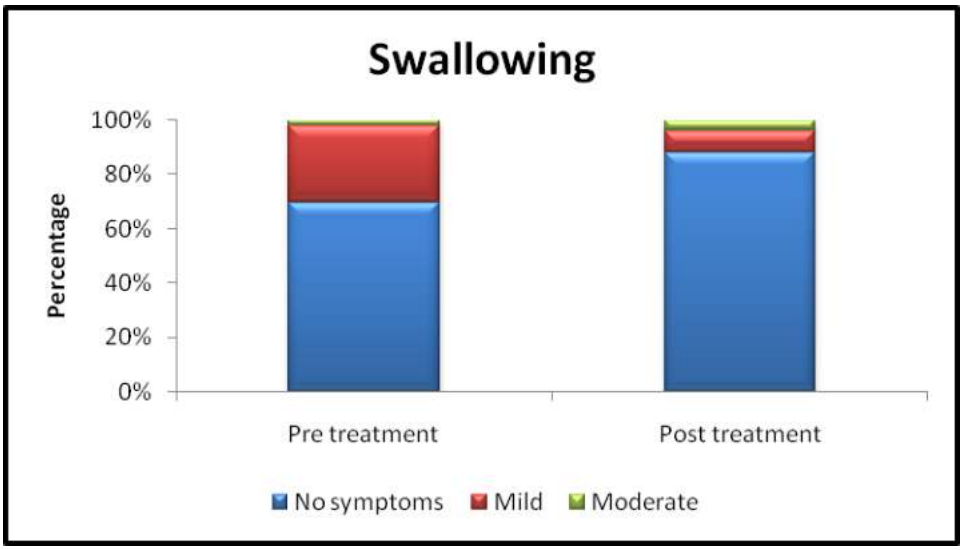


Figure 53

The above table shows the comparison of swallowing between pre and post treatment by using Pearson Chi-Square test, which shows that there is highly statistically significant difference

between pre- and post-treatment in swallowing, Chi-Square value =35.649, p-value=0.0005< 0.01. There is a significant reduction in terms of swallowing noted in the post treatment evaluation of all treatment modalities. Pretreatment and post treatment swallowing evaluation was carried out by clinical grading as: -

Zero – No difficulty in swallowing

1 - Mild difficulty in swallowing

2 – Moderate difficulty in swallowing

3 – Severe difficulty in swallowing

15.ANALYSIS OF COMPARISON OF TREATMENT AND RESPONSE BY PEARSON CHI SQUARE TEST

			Response			Total
			Complete	Partial	Progressive	
Treatment	Chemotheropy+Radiotherapy	Count	13	20	3	36
		%	61.9%	87.0%	50.0%	72.0%
	Radiotherapy	Count	3	0	0	3
		%	14.3%	0.0%	0.0%	6.0%
	Surgery	Count	3	2	1	6
		%	14.3%	8.7%	16.7%	12.0%
	Surgery+Chemotheropy+Radiotherapy	Count	1	1	1	3
		%	4.8%	4.3%	16.7%	6.0%
	Surgery+Radiotherapy	Count	1	0	1	2
		%	4.8%	0.0%	16.7%	4.0%
Total		Count	21	23	6	50
		%	100.0%	100.0%	100.0%	100.0%

Table 15

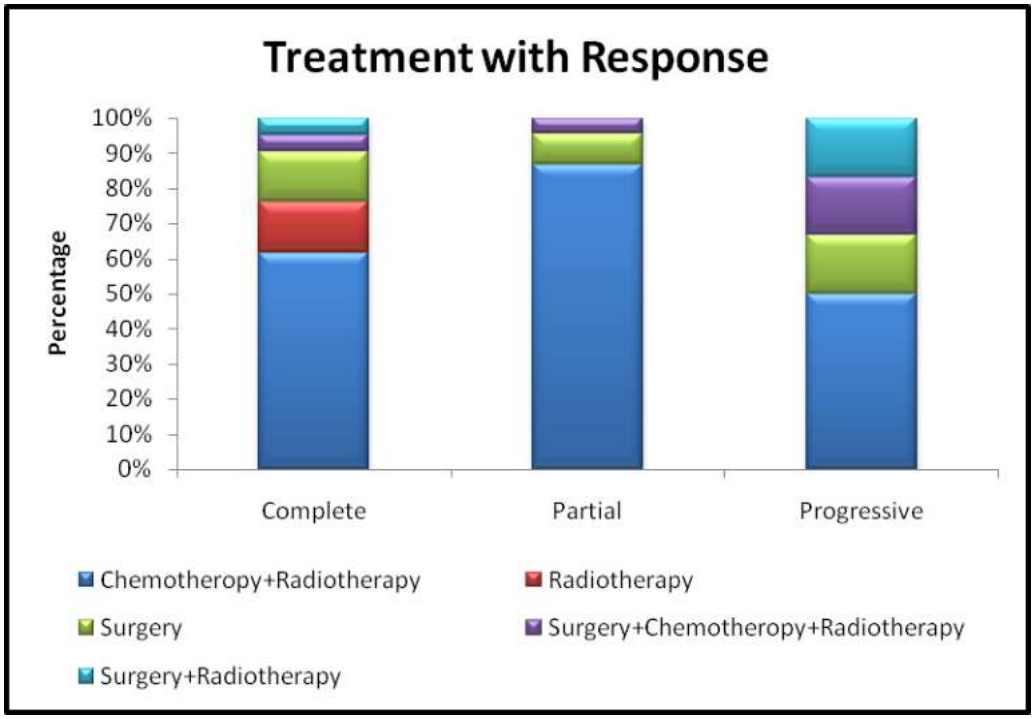


Figure 54

The above table shows the comparison between treatment and response by using Pearson Chi-Square test, which shows that there is no statistically significant association between treatment and response, Chi-Square value =10.621, p-value=0.224>0.05. Response to various modalities of treatment were assessed after six weeks of completion of treatment using WHO response evaluation criteria for solid tumors. There was no significant association founded out. But overall response to all treatment modalities improved. It shows that the response of each treatment will be vary in each case with the patient selection, stage of presentation, treatment of choice, compliance to treatment and post treatment care etc. Thus, the treatment modalities could not be compared in terms of response and could not be concluded that which one is best according to this study.

Summary

- The age distribution of the study population which consist of 6.0 % up to 35 years, 30% is 36 to 45 years, both 46 to 55 years and 56 to 65 years were 28% and 8% 66 to 75 years respectively, the mean ± standard deviation of the age were 51±11 years.
- The gender distribution of the study population which consists of 24% female and 76% male.
- The risk factors distribution of the study population which consists of 52% were alcoholic, 72% of them with betel nut habit, 44% of them were smokers, 24% of them were spicy food eaters,14 % risk was due to sharp tooth,1% chronic oral sepsis, 34% of them were pan masala chewers and 2% were found with no risk factors.
- The subsite distribution of the study population which consists of alveolus, floor of mouth & lip 2%, hard palate 8%, oral tongue 44% and buccal mucosa 36%.
- The types of carcinoma distribution of the study population which consists of adenoid cystic carcinoma & mucoepidermoid carcinoma were 2%, verrucous carcinoma 4% and 92% of squamous cell carcinoma.
- The grades distribution of the study population which consists of 44% were well differentiated, 56% were moderately differentiated and no patients were poorly differentiated.
- The treatment distribution of the study population which consists of 73% with chemotherapy+radiotherapy, 6% radiotherapy, 6% with surgery + chemotherapy radiotherapy, surgery+radiotherapy is 4% and 12% underwent the only surgery.

- The response distribution of the study population which consists of 42% complete response, partial response is 46% and 12% progressive disease.
- The comparison of pain between pre and post treatment by using Wilcoxon signed rank test, which shows that there is a highly statistically significant difference between pre- and post-treatment in pain with mean ± standard deviation 6.5 ± 2.1 at pre after the treatment it becomes 3.7± 2. Z value=5.623, p-value= 0.0005< 0.01.
- The comparison of bleeding between pre and post treatment by using Pearson Chi-Square test, which shows that there is no statistically significant difference between pre- and post-treatment in bleeding, Chi-Square value = 4.396, p-value= 0.111> 0.05. Even though there is no statistical significance the difference between pre and post treatment but the percentage of no symptoms were increased from 44% from pre to 90 % in the post treatment.
- The comparison of trismus between pre and post treatment by using Pearson Chi-Square test, which shows that there is highly statistically significant difference between pre- and post-treatment in trismus, Chi-Square value = 51.447, p-value=0.0005 < 0.01.
- The comparison of swallowing between pre and post treatment by using Pearson Chi-Square test, which shows that there is highly statistically significant difference between pre- and post-treatment in swallowing, Chi-Square value =35.649, p-value=0.0005< 0.01.
- The comparison between treatment and response by using Pearson Chi-Square test, which shows that there is no statistically significant association between treatment and response, Chi-Square value =10.621, p-value=0.224>0.05. Each treatment will vary in response depending up on various factors like patient selection, nature of treatment, compliance and post-operative care and so we could not conclude about the best choice of treatment in general according to our study.

DISCUSSION

The Indian subcontinent, especially India is regarded as the global epicenter of oral cancer because of its large population. Oral cancer is of significant public health importance to India. The reason being it is diagnosed at later stages which ends in low treatment outcomes and considerable expense to the patients who typically cannot afford this type of treatment. Also, rural areas in low- and middle-income countries also have limited access to trained health service providers. It results in treatment delay and advanced stages of oral cancer.

Earlier detection of oral cancer offers improved long-term survival and to improves treatment outcomes and makes healthcare affordable. Another reason is, oral cancer affects more those from the lower socioeconomic groups, due to higher exposure to risk factors such as the use of tobacco and pan masala. Lastly, the majority of cases present to a healthcare facility at later stages of cancer subtypes, thereby reducing chances of survival due to delays in diagnosis.[43]

In our study, we are discussing the various treatment modalities of oral cancer in our hospital and comparing the different modalities of treatment in the management of oral cancers, finally identifying the risk factors associated with oral cancers.

1.Age and sex distribution

Borse et al. [1] did a study in the western regions of Maharashtra and concluded that the highest occurrence of oral malignancy is reported in the age group of 60 years, followed by between 40-59 years with a male to female ratio of 2:1.

Varshitha et al. [44] did a study and concluded that men are more affected than women. In India, men are two to four times more affected than women due to the changes in behavioral and lifestyle patterns.

Singh et al.[45] conducted a study was to evaluate the epidemiological factors and clinical profile of oral cancer cases in northern India and concluded that the mean age of presentation of oral cancer was 47.84 years. The greatest number of cases in the study (n = 124; [25.8%]) were recorded in age group 51–60 years followed by age group 41–50 (n = 119; [24.8%]). The male to female ratio was 3.1:1.0.

Deepa et al.[46] concluded that the median age was 52 years and the male-to-female ratio was 3.4:1.

Thavarool et al.[47] conducted a retrospective study of a prospective database of oral cancer patients who underwent surgery from June 2009 to June 2013. Results showed that the majority of the respondents were males (136, 61.8%).

In our study, results showed that the mean age distribution of the study population was 51± 11 years (mean ± std deviation) and with a male to female ratio of 3.1:1. In our study highest presentation was in the 36-45 years age group with mean distribution in the sixth decade. The results of studies conducted by both Deepa et al. and Singh et al are similar to our study in both age and sex distribution. The male predominance of the disease is also given by Borse et al., Thavarool et al., and Varshitha et al similar to our study but in a different proportion.

2.Stage distribution

The distribution by stage of the patients in the study conducted by Singh et al. shows that 64.1% of patients had stage IV disease, followed by 28.2% with stage III disease. 55.3% had stage IVA disease and 8.1% of patients had stage IVB disease.

Three patients presented with stage IVC (0.7%) disease. Twenty-four (5.0%) of patients presented stage II and 13 (2.7%) patients with stage I disease.

In a study conducted by Krishnan Nair and R. Sankaranarayanan, the study population showed that 85% with stage I, 63% with stage II, 41% with stage III, and 15% with stage IV disease survived disease-free at 3 years.[48]

In our study, the present study population showed 24 % presented in stage I, 32 % presented in stage II, 22 % in stage III, and 22% in stage IV. Most of the cases were in stage II. Stage I and stage II are considered as early-stage while stage III and stage IV are considered as late-stage diseases. 56% of the study population presented in early-stage and 44 % were in late stage. The stage distribution in our study is different from the above-described studies.

3.Subsite distribution

Deepa et al.[46] concluded in their study that buccal mucosa (BM) was the most common subsite (64.94%). BM cancers (81.1%) were more likely to present in the advanced stage compared to tongue cancers (52%) (P = 0.000). The factors influencing survival were age (>50 years), advanced cT stage, nodal metastasis, overall stage, and presence of orocutaneous fistula.

Thavarool et al.[47] conducted a retrospective study in which the tongue was the most common site of cancer in more than half of the patients (113, 51.4%) followed by buccal mucosa (48, 21.8%) and lower alveolus (34, 15.5%).

Dhanuthai et al.[49] divided the subsites into lip, tongue, the floor of the mouth, gingiva, alveolar mucosa, palate, oral/labial mucosa, maxilla, and mandible.

The study concluded that the preferred sites for oral cancer were the tongue, labial / oral mucosa, gingiva, palate, and alveolar mucosa, respectively.

In our study population oral tongue was the commonest subsite followed by buccal mucosa, hard palate, respectively. The alveolus, floor of the mouth, and commissure of lip showed equal and low subsite presentation of oral cavity malignancy.

In the buccal mucosa, the right and left sides showed equal predominance. In the case of the hard palate, the right side shows a higher incidence than the left side.

As in our study, the tongue is the commonest subsite for oral cavity cancer in studies conducted by Thavarool et al. and Dhanuthai et al.

4. Risk factor distribution.

Thavarool et al.[47] conducted a retrospective study of a prospective database of oral cancer patients who underwent surgery from June 2009 to June 2013. Results showed that the majority of the respondents were tobacco users (188, 85.5%).

Ken Russell Coelho.[43] concluded that smoking and alcohol are known risk factors for oral cancer. They observed that 57% of all men and 11% of women aged 15 to 49 use some form of tobacco. Besides smoking, the use of smokeless tobacco is also widely prevalent, the use of betel quid also referred to as pan consists of pieces of areca nut, tobacco treated or not, aqueous calcium hydroxide (slaked lime), and some spices wrapped in a leaf of a betel vine leaf. This is very common and is socially and culturally accepted in many parts of India.

On analyzing the risk factors betel nut chewing was found to be the major risk factor in our study followed by alcohol, smoking, pan masala, spicy food, and sharp tooth in the respective decreasing order with chronic oral sepsis was the least common risk factor. Our results are

different from the above described two studies that could be due to demographic variation between the study populations.

5.Type distribution

Dhanuthai et al.[49] determined the prevalence and clinicopathologic features of oral cancer patients. Biopsy records from participating facilities were reviewed for oral cancer cases diagnosed from 2005 to 2014. Demographic data and the location of injuries were collected.

The lesion sites were divided into lip, tongue, the floor of the mouth, gingiva, alveolar mucosa, palate, oral/labial mucosa, maxilla, and mandible. Oral cancer has been divided into 7 categories: epithelial tumors, salivary gland tumors, hematologic tumors, bone tumors, mesenchymal tumors, odontogenic tumors, and others.

The three most common oral cancers in decreasing order of frequency were squamous cell carcinoma, non-Hodgkin lymphoma, and mucoepidermoid carcinoma.

Neville et al.[50] concluded that over 90 percent of these tumors are squamous cell carcinomas, which arise from the oral mucosal lining. Despite the easy accessibility of the oral cavity to direct examination, these malignant tumors often go undetected. Late-stage and survival rate for oral cancer has remained broadly unchanged over the past three decades.

In our study results shows the types of oral carcinoma distribution of the study population which consists of adenoid cystic carcinoma & mucoepidermoid carcinoma were 2%, verrucous carcinoma 4%, and 92% of squamous cell carcinoma. The majority of the study population presented as squamous cell carcinoma followed by verrucous carcinoma. Adenoid cystic carcinoma and mucoepidermoid carcinoma showed equal and least predominance and both were located in the hard palate.

The study results of Neville et al. and Dhanuthai et al. are similar to our study results that squamous cell carcinoma is the commonest type in oral cavity cancers.

6.Treatment distribution

In most of the studies, patients presented late, and hence the results of surgery could not be compared effectively. Radiotherapy was used as the first line of management of carcinoma of buccal mucosa in a series by M Krishnan Nair and R. Sankaranarayanan, the study population showed that 85% with stage I, 63% with stage II, 41% with stage III, and 15% with stage IV disease survived disease-free at 3 years.[48]

Fein et al.[51] concluded in their study that surgical treatment for T1 – T2 patients with the addition of postoperative twice a day radiotherapy is recommended in selected cases. For T3 – T4 patients twice a day preoperative radiotherapy is recommended as it reduces the extent of the surgical procedure. Surgery or radiotherapy as a single modality of treatment is generally recommended for early-stage diseases like stage I and stage II. Both have resulted in similar survival rates. But for patients with locally advanced disease, combined modality treatment is recommended.

Since the majority of cases present at a late stage, the therapy is more complex with the worst prognosis as per Bernier et al.[52]

In our study treatment distribution of the study, the population consists of 73% with chemotherapy+radiotherapy, 6% radiotherapy, 6% with surgery + chemotherapy radiotherapy, surgery+radiotherapy is 4% and 12% underwent the only surgery. The majority of the people underwent chemotherapy + radiotherapy followed by people who underwent only surgery. The least majority of people underwent surgery followed by radiotherapy.

The majority of the patients underwent chemotherapy and radiotherapy followed by surgery rather than combined modalities, even though the stage at presentation is early in 56 % the of sample size. We could not make out a significant relation between responses with a particular treatment modality. But overall, symptomatic improvement for the patients is seen in all treatment modalities after 4 weeks of completion of treatment.

7. Grade distribution

Pires et al.[53] conducted a study to report the demographic and clinicopathological features from OSCC diagnosed in an oral pathology service in south-eastern Brazil in an 8-year period. All OSCC diagnosed from 2005 to 2012 were reviewed, including histological analysis of all hematoxylin and eosin-stained slides and review of all demographic and clinical information from the laboratory records. Histological diagnosis and grade of the tumors rendered after analysis of the HE-stained slides revealed that, from 303 cases of conventional invasive OSCC, 93 cases (30.7%) were classified as well-differentiated OSCC, 138 cases (45.5%) as moderately differentiated tumors, and 72 (23.8%) as poorly differentiated tumors. Although there were some differences in the histological grade of the tumors when comparing each location, these results were not statistically significant (p=0.381).

From the remaining 43 OSCC samples, 26 cases (7.5%) were diagnosed as micro-invasive OSCC and the remaining 17 cases were diagnosed as OSCC variants, including 9 verrucous carcinomas (2.6%), 5 spindle cell carcinomas (1.4%), 2 basaloid (0.6%) and 1 papillary (0.3%).

The distribution of the histological grade of the conventional invasive OSCC (n=303) was also different when comparing the gender of the patients, as males were predominantly

affected by moderately differentiated and poorly differentiated tumors, while females presented mostly with moderately differentiated and well-differentiated tumors (p=0.004).

Hanemann et al.[54] did a study to histologically assess different types of oral squamous cell carcinoma and the silver-binding nucleolar organizer region (AgNOR) morphology in neoplastic cells, as well as to quantify the number of AgNORs in each type of carcinoma in order to relate AgNOR count and histologic grading. AgNORs were seen through a light microscope inside the cell nuclei as black to brownish dots since the yellow staining allowed easy visualization of individual NORs. The number and diameter of the NORs, usually round in shape, were variable and either diffusely distributed all over the nuclear area or grouped in a wide, round, and less intensely stained structure. The grouped AgNORs had smaller diameter and were more variable in amount than those diffusely distributed.

The number of AgNORs in every OSCC studied is showed that the mean number of AgNORs *per* nucleus was 3.20 (±0.61) for the well-differentiated group, 5.33 (±1.42) for the moderately differentiated one, 8.27 (±0.39) for the poorly differentiated one, and 10.08 (±0.27) for the undifferentiated one. Kruskal-Wallis test showed a statistically significant difference (p=0.01) among the mean numbers of AgNORs in each studied group. There were also statistically significant differences (p=0.01) for all two-by-two comparisons of groups.

Our study shows the grades distribution of the study population in which 44% were well-differentiated, 56% were moderately differentiated and no patients were poorly differentiated. Results of our study show that the majority of the patients had moderately differentiated pathologic grades followed by well-differentiated grades and none of the patients had poorly differentiated pathological grades.

Our study results are similar to the study conducted by Pires et al. that the majority of the patients had moderately differentiated pathologic grades.

8.Surgery distribution and Response to treatment

Dzioba et al.[55] showed in their study that one hundred and seventeen patients who had undergone partial glossectomy with reconstruction participated in this study. Results indicated no significant differences in swallowing function between baseline and 6 months post-surgery and no significant differences in speech function (SHI subscales) between baseline and 1-year post-surgery. Most quality-of-life domains returned to baseline levels by 1-year post-operation, while difficulties with dry mouth and sticky saliva persisted. They concluded that assessment time influenced patient-reported speech, swallowing, and quality of life outcomes, while the effects of treatment (for time) were only found for swallowing and quality of life.

Results of the study will help to guide clinical care and will be useful for patient counseling on short- and long-term functional and quality of life results from the surgical and adjuvant treatment of oral cancer.

Langdon et al.[56] did a study and showed that the overall 5-year survival figure, not corrected for age and sex was, 32.8%. The local recurrence rate for all sites was 44%, 90% of these recurred within 2 years of diagnosis. Analysis of the material has not provided a satisfactory explanation for this; lesions do not present at an earlier stage in females neither do they occur in younger patients. Survival rates analyzed by clinical staging at presentation confirm that the prognosis for early-stage lesions is very much better than for late-stage lesions, the 5-year survival for stage I is 50% whereas that for stage IV is only 20%.

Suarez-Cunqueiro et al.[57] led a study to evaluate the prevalence of the discourse and swallowing after radical surgery of oral cancer and oropharyngeal cancer the patient's viewpoint and to examine the association of these functional alterations with selected clinical characteristics regarding patients, tumors, and oncologic treatment. Speech problems were reported by 851 patients (63.8%), and swallowing problems were reported by 1006 patients (75.4%). The variables that presented a significant association with speech and swallowing impairment were sex, tumor location, pTNM stages, and stage of the tumor, treatment modality, and reconstruction type.

This survey, based on patient perception, suggests that those who undergo radiotherapy associated with the surgical removal of a tumor, have late-stage tumors (III-IV), or have tumors located on the floor of the mouth should be informed of the greater risk of persistent severe speech and swallowing problems.

Boysen et al.[58] concluded that clinical findings, treatment, and results have been recorded prospectively in 661 patients with carcinoma of the head and neck. In the study, with an average follow-up of 3 years, 7813 follow-up consultations revealed 220 recurrences. The overall "recurrence pick-up rate" and subsequent "cure rate" were 1:36 and 1:113 consultations, respectively.

In our study response was analyzed after a duration of six week after completion of treatment using WHO response evaluation criteria in solid tumour. In the study population only 11 patients underwent surgical management of which the majority underwent wide local excision. Only 3 patients out of 50 underwent wide local excision.

One patient underwent wide local excision with marginal mandibulectomy. One patient underwent wedge resection with an advancement flap. One patient underwent inferior partial maxillectomy and one patient underwent total maxillectomy. One patient underwent hemiglossectomy and another patient underwent hemiglossectomy with modified radical neck dissection. One patient underwent composite resection with pectoralis major myocutaneous flap (PMMC) and another patient underwent composite resection with PMMC flap with modified radical neck dissection. Out of 50 study populations, 21 patients showed complete response, 23 showed partial response and 6 patients showed progressive disease. The majority of the patients showed only partial response to all treatment modalities and only 42 % showed complete response to various treatment modalities. 12% of the study population showed progressive diseases despite various treatment modalities. Results of our study show that there is a significant improvement in terms of swallowing noted in the post-treatment evaluation of all treatment modalities which is similar to the study conducted by Suarez-Cunqueiro et al.

A multifaceted approach that integrates health education, tobacco and alcohol control, early detection, and early treatment is needed to reduce the burden of this eminently preventable cancer. How to accomplish this is known; astonishingly, it has not been applied in most countries, and not at all in the high-burden countries. Improving awareness among the general public and primary care practitioners, investing in health services to provide screening and early diagnosis services for tobacco and alcohol users, and providing adequate treatment for those diagnosed with invasive cancer are critically important oral cancer control measures.

SUMMARY AND CONCLUSION

- The various cancers in the oral cavity are treated by surgery or radiation or surgery followed by radiation or radiation along with chemotherapy or a combination of all three modalities.
- Multimodal treatment had a better cure rate compared to single-modality treatment.
- Radiotherapy is more effective in the early stages and in combination with other modalities in the late stages.
- In all the treatment modalities all together there is significant symptomatic improvement noted for pain, trismus, and swallowing.
- Significant relation could not be found out between particular treatment modality and response of tumour, six weeks after completion of treatment. Response to various modalities of treatment varies from person to person, stage of presentation, and general condition of the patient and could not be generalized and compared.
- Betel nut chewing is the most common risk factor associated with oral cancers followed by alcohol, smoking, pan masala, spicy food, sharp tooth and chronic oral sepsis in the decreasing order.

BIBLIOGRAPHY

1. Borse V, Konwar AN, Buragohain P. Oral cancer diagnosis and perspectives in India. Sensors International. 2020 Sep 24:100046.
2. Campana JP, Meyers AD. The surgical management of oral cancer. Otolaryngologic Clinics of North America. 2006 Apr 1;39(2):331-48.
3. Mishra GA, Pimple SA, Gupta SD. Smokeless tobacco use and oral neoplasia among urban Indian women. Oral diseases. 2019 Oct;25(7):1724-34.
4. Sciubba JJ. Oral cancer. American journal of clinical dermatology. 2001 Aug;2(4):239-51.
5. Shah JP. Patterns of cervical lymph node metastasis from squamous carcinomas of the upper aerodigestive tract. The American journal of surgery. 1990 Oct 1;160(4):405-9.
6. Andersen PE, Warren F, Spiro J, Burningham A, Wong R, Wax MK, Shah JP, Cohen JI. Results of selective neck dissection in management of the node-positive neck. Archives of Otolaryngology–Head & Neck Surgery. 2002 Oct 1;128(10):1180-4.
7. Magreni A, May JG. Embryology of the oral structures. Operative Techniques in Otolaryngology-Head and Neck Surgery. 2015 Sep 1;26(3):110-4.
8. Bailey and love's short practice of surgery 24th edition, hodder arnold.
9. Scott-brown's otorhinolaryngology head&neck surgery 8th edition.

10. Rodgers GK, Myers EN. Surgical management of the mass in the buccal space. The Laryngoscope. 1988 Jul;98(7):749-53.

11. Larson DL, Lewis SR, Rapperport AS, Coers III CR, Blocker Jr TG. Lymphatics of the mouth and neck. The American Journal of Surgery. 1965 Oct 1;110(4):625-30.

12. Johnston TP. Anatomy and physiology of the oral mucosa. Oral mucosal drug delivery and therapy. 2015:1-5.

13. Sachdeva A, Bhateja S, Arora G. Oral physiology. Indian Journal of Clinical Anatomy and Physiology 2019;6(3):256–259.

14. Menicagli R, Marotta O, Nunzia M, Casotti MT. Betel Chewing: A New Analysis, In Vitro and In Vivo, of the Risk Factors in Oral Cancer. The Gulf journal of oncology. 2019 May 1;1(30):13-21.

15. Anand R, Dhingra C, Prasad S, Menon I. Betel nut chewing and its deleterious effects on oral cavity. Journal of cancer research and therapeutics. 2014 Jul 1;10(3):499.

16. Principles of surgery, schwartz;8th edition, mcgraw hill.

17. Cawson RA, Langdom JD, Eveson JW. Surgical pathology of the mouth and jaws.1996.

18. Kochurova EV, Nikolenko VN, Mukhanov AA, Demenchuk PA. Dental status as a predictive factor in the development of squamous cell carcinoma of the oral mucosa. Stomatologiia. 2019 Jan 1;98(4):34-7.

19. Rajendran R. Shafer's textbook of oral pathology. Elsevier India; 2009.

20. Vincent T. Devita, Jr., Samuel Hellman, Stevan A. Rosenberg, Cancer Principles and Practices of Oncology, 7th Edition.

21. The American Society for Head and Neck Surgery and the Society of Head and Neck Surgeons. Clinical practice Guidelines for the Diagnosis and Management of cancer of the Head and Neck, 1996.

22. Wolfensberger M, Zbaeren P, Dulguerov P, Müller W, Arnoux A, Schmid S. Surgical treatment of early oral carcinoma—results of a prospective controlled multicenter study. Head & Neck: Journal for the Sciences and Specialties of the Head and Neck. 2001 Jul;23(7):525-30.

23. Lydiatt DD, Robbins KT, Byers RM, Wolf PF. Treatment of stage I and II oral tongue cancer. Head & neck. 1993 Jul;15(4):308-12.

24. Thawley SE, Panje WR. Comprehensive management of head and neck tumors, volume 1.

25. Watne AL. Treatment alterntives: Head and neck cancers. Cancer. 1984 Dec 1;54(S2):2673-81.

26. National Comprehensive Cancer Network. NCCN clinical practice guidelines in oncology: head and neck cancers. Version. 2016; 1:2012.

27. Ord RA. Local resection and local reconstruction of oral carcinomas. InOperative Maxillofacial Surgery 1998 (p. 278). Chapman & Hall Medical, London.

28. Spiro, R.H., Spiro, J.D., Strong, E.W. 2003. Surgical approachto squamous carcinoma confined to the tongue and the floor of the mouth.

29. Kovács AF, Schiemann M, Turowski B. Combined modality treatment of oral and oropharyngeal cancer including neoadjuvant intraarterial cisplatin and radical surgery followed by concurrent radiation and chemotherapy with weekly docebaxel–three-year results of a pilot study. Journal of Cranio-Maxillofacial Surgery. 2002 Apr 1;30(2):112-20.

30. Palme CE, Gullane PJ, Gilbert RW. Current treatment options in squamous cell carcinoma of the oral cavity. Surgical Oncology Clinics. 2004 Jan 1;13(1):47-70.

31. Current surgical treatment of squamous cell carcinoma of the head and neck oral oncol. 2007, March 43(3).

32. Andry G, Hamoir M, Leemans CR. The evolving role of surgery in the management of head and neck tumors. Current opinion in oncology. 2005 May 1;17(3):241-8.

33. Weiss MH, Harrison LB, Isaacs RS. Use of decision analysis in planning a management strategy for the stage NO neck. Archives of Otolaryngology–Head & Neck Surgery. 1994 Jul 1;120(7):699-702.

34. Tankéré F, Camproux A, Barry B, Guedon C, Depondt J, Gehanno P. Prognostic value of lymph node involvement in oral cancers: a study of 137 cases. The Laryngoscope. 2000 Dec;110(12):2061-5.

35. Feig BW, Berger DH, Fuhrman GM, editors. The MD Anderson surgical oncology handbook. Lippincott Williams & Wilkins; 2006.

36. Cohen EE, Lingen MW, Vokes EE. The expanding role of systemic therapy in head and neck cancer. Journal of clinical oncology. 2004 May 1;22(9):1743-52.

37. Blake cady, carcinoma of the oral cavity, evaluation and management.the surgical clinics of north america.

38. Gore SM, Crombie AK, Batstone MD, Clark JR. Concurrent chemoradiotherapy compared with surgery and adjuvant radiotherapy for oral cavity squamous cell carcinoma. Head & neck. 2015 Apr;37(4):518-23.

39. Wheeler RH, Baker SR, Liepman MK, Ensminger WD. Intensive sequential chemotherapy with bleomycin, oncovin, mitomycin C, and methotrexate followed by adriamycin, cisplatin, and cyclosphosphamide in squamous cell cancer of the head and neck. Medical and pediatric oncology. 1983;11(1):12-9.

40. National Cancer Institute Head and Neck Contracts writing Committee. Adjuvant chemotherapy for advanced head and neckcarcinoma. 1988. 60:301 – 311.

41. Clark, J. *et al.,* (1997), Induction chemotherapy with cisplatin, fluorouracil and high dose leucovorin for squamous cell cancer of head and neck – Journal of Clinical Oncology, 15:3100 –3110.

42. Huang SH. Oral cancer: Current role of radiotherapy and chemotherapy. Medicina oral, patologia oral y cirugia bucal. 2013 Mar;18(2): e233.

43. Coelho KR. Challenges of the oral cancer burden in India. Journal of cancer epidemiology. 2012 Oct 4;2012.
44. Varshitha A. Prevalence of oral cancer in India. Journal of Pharmaceutical Sciences and Research. 2015 Oct 1;7(10):845.
45. Singh MP, Misra S, Rathanaswamy SP, Gupta S, Tewari BN, Bhatt ML, Kumar V. Clinical profile and epidemiological factors of oral cancer patients from North India. National journal of maxillofacial surgery. 2015 Jan;6(1):21.
46. Nair D, Singhvi H, Mair M, Qayyumi B, Deshmukh A, Pantvaidya G, Nair S, Chaturvedi P, Laskar SG, Prabhash K, DCruz A. Outcomes of surgically treated oral cancer patients at a tertiary cancer center in India. Indian Journal of cancer. 2017 Oct 1;54(4):616.
47. Thavarool SB, Muttath G, Nayanar S, Duraisamy K, Bhat P, Shringarpure K, Nayak P, Tripathy JP, Thaddeus A, Philip S, Satheesan B. Improved survival among oral cancer patients: findings from a retrospective study at a tertiary care cancer centre in rural Kerala, India. World journal of surgical oncology. 2019 Dec;17(1):1-7.
48. Krishnan Nair M, Sankaranarayanan R, Padmanabhan TK. Evaluation of the role of radiotherapy in the management of carcinoma of the buccal mucosa. Cancer. 1988 Apr 1;61(7):1326-31.
49. Dhanuthai K, Rojanawatsirivej S, Thosaporn W, Kintarak S, Subarnbhesaj A, Darling M, Kryshtalskyj E, Chiang CP, Shin HI, Choi SY, Lee SS. Oral cancer: A multicenter study. Medicina oral, patologia oral y cirugia bucal. 2018 Jan;23(1): e23.

50. Neville BW, Day TA. Oral cancer and precancerous lesions. CA: a cancer journal for clinicians. 2002 Jul;52(4):195-215.

51. Fein DA, Mendenhall WM, Parsons JT, McCarty PJ, Stringer SP, Million RR, Cassisi NJ. Carcinoma of the oral tongue: a comparison of results and complications of treatment with radiotherapy and/or surgery. Head & neck. 1994 Jul;16(4):358-65.

52. Bernier J, Domenge C, Ozsahin M, Matuszewska K, Lefèbvre JL, Greiner RH, Giralt J, Maingon P, Rolland F, Bolla M, Cognetti F. Postoperative irradiation with or without concomitant chemotherapy for locally advanced head and neck cancer. New England Journal of Medicine. 2004 May 6;350(19):1945-52.

53. Pires FR, Ramos AB, Oliveira JB, Tavares AS, Luz PS, Santos TC. Oral squamous cell carcinoma: clinicopathological features from 346 cases from a single oral pathology service during an 8-year period. Journal of Applied Oral Science. 2013 Sep; 21:460-7.

54. Hanemann JA, Miyazawa M, Souza MS. Histologic grading and nucleolar organizer regions in oral squamous cell carcinomas. Journal of Applied Oral Science. 2011 Jun;19(3):280-5.

55. Dzioba A, Aalto D, Papadopoulos-Nydam G, Seikaly H, Rieger J, Wolfaardt J, Osswald M, Harris JR, O'Connell DA, Lazarus C, Urken M. Functional and quality of life outcomes after partial glossectomy: a multi-institutional longitudinal study of the head and neck research network. Journal of Otolaryngology-Head & Neck Surgery. 2017 Dec;46(1):1-1.

56. Langdon JD, Harvey PW, Rapidis AD, Patel MF, Johnson NW, Hopps R. Oral cancer: the behaviour and response to treatment of 194 cases. Journal of Maxillofacial Surgery. 1977 Jan 1; 5:221-37.

57. Suarez-Cunqueiro MM, Schramm A, Schoen R, Seoane-Lestón J, Otero-Cepeda XL, Bormann KH, Kokemueller H, Metzger M, Diz-Dios P, Gellrich NC. Speech and swallowing impairment after treatment for oral and oropharyngeal cancer. Archives of Otolaryngology–Head & Neck Surgery. 2008 Dec 15;134(12):1299-304.

58. Boysen M, Lövdal O, Winther F, Tausjö J. The value of follow-up in patients treated for squamous cell carcinoma of the head and neck. European journal of cancer. 1992 Feb 1;28(2-3):426-30.

CASE SHEET PROFORMA

Name: Age/Sex: IP/OP:

Occupation:

Address:

Phone no:

Pre-treatment evaluation date: Post treatment evaluation date:

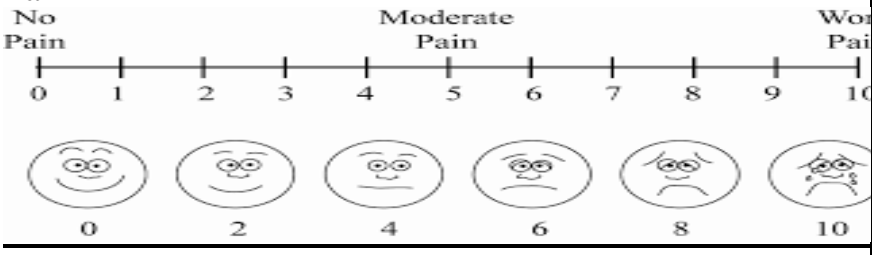
DIAGNOSIS

Sub site Type Grade

Stage TNM

Early/Late

SYMPTOMS

	SYMPTOMS	Pre treatment	Post treatment
1	Pain 		
2	Bleeding		
3	Trismus		

4	Speech alteration No/Yes If Yes		
5	Swallowing		
6	Perfomance status		

Treatment Major/Minor	Response to treatment RECIST

PERSONAL HISTORY

Smoking

Alcohol

Betel nut/tobacco chewing

Sharp teeth/dentures

Chronic oral sepsis

Spicy food intake

ABBREVIATIONS

Sl. No.	Abbreviation	Stands for
1	AJCC	American Joint Committee on Cancer
2	CA	Cancer
3	cGY	Centigray
4	CT	Computed tomography
5	CT	Chemotherapy
6	Eg	Example
7	et al.	And others
8	FNAC	Fine Needle Aspiration Cytology
9	gy	Gray
10	HE	Hematoxylin and eosin
11	HPV	Human papilloma virus
12	i.e.,	For example,
13	kDa	Kilodalton
14	Lt	Left
15	OSCC	Oral squamous cell carcinoma
16	PMMC	Pectoralis major myocutaneous flap
17	RT	Radiotherapy
18	µm	Micrometer
19	WHO	World Health Organization
20	XRT	External beam radiotherapy

PLAGIARISM CERTIFICATE

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W	URL: https://pubmed.ncbi.nlm.nih.gov/1591057/ Fetched: 2021-12-02T19:13:00.0000000	2

CERTIFICATE - II

This is to certify that this dissertation work titled "A PROSPECTIVE STUDY OF MANAGEMENT OF ORAL CAVITY CANCERS" of the candidate Dr. GANGA P with registration Number 221914052 for the award of M.S. in the branch of OTORHINOLARYNGOLOGY.

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Guide & Supervisor sign with Seal.

ETHICAL COMMITTEE APPROVAL LETTER

GOVERNMENT STANLEY MEDICAL COLLEGE & HOSPITAL,CHENNAI -01

INSTITUTIONAL ETHICS COMMITTEE

TITLE OF THE WORK	:"A PROSPECTIVE STUDY OF MANAGEMENT OF ORAL CAVITY CANCERS"
PRINCIPAL INVESTIGATOR	: DR. P.GANGA,
DESIGNATION	: PG IN MS ENT,
DEPARTMENT	: DEPARTMENT OF ENT, GOVT. STANLEY MEDICAL COLLEGE.

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 10.09.2020 at the Council Hall, Stanley Medical College, Chennai-1 at 10am.

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETAR
IEC, SMC, CHENNAI

MASTER CHART

												pre treatement symptoms				post treatment symptoms				
sno	name	age	sex	risk factor	subsite	type	grade	TNM	treatment	surgery	response	Pain(x/10)	Bleeding	Trismus	Swallowing	Pain(x/10)	Bleeding	Trismus	Swallowing	Stage
1	Mala	45	F	SF	Rt Hard palate	SCC	II	$T_2N_0M_0$	SX	Rt inf partial maxillectomy	CR	8	0	3F	0	4	0	3F		II
2	Elumalai	35	M	AL,BN,SF	Alveolus	SCC	I	$T_{4a}N_0M_0$	CT+ RT		PR	10	1	1F	0	8	0	3F	0	IV
3	Aruldas	62	M	Al,BN,SF,SM	Rt BM	SCC	I	$T_{4a}N_{2b}M_0$	CT+ RT		PR	10	2	2F	0	8	0	3F	0	IV
4	Sundaramurthy	55	M	BN,AL,SF	Lt BM	SCC	II	$T_3N_{2b}M_0$	Sx+ CT+RT	CR + PMMC flap+MRND	CR	8	1	2F	0	4	0	absent	0	IV
5	Govindan	45	M	AL,SM,SF	Lt Hard palate	SCC	I	$T_{4a}N_0M_0$	SX	Lt Total maxillectomy	PD	8	1	3F	1	8	1	3F	0	IV
6	Ranganathan	40	M	AL,BN,ST,SF	Oral tongue	SCC	II	$T_2N_0M_0$	CT+ RT		CR	8	1	2F	0	4	0	3F	2	II
7	Swaminathan	69	M	SM,BN,ST,SF	Oral tongue	SCC	II	$T_2N_0M_0$	SX+RT	Lt hemiglosectomy+ MRND	CR	2	2	absent	1	0	0	absent	0	II
8	Magendran	40	M	BN,SF,PM	Oral tongue	SCC	II	$T_2N_0M_0$	CT+RT		CR	8	1	3F	1	2	0	2F	0	II
9	Prakash	34	M	BN,PM	Lt BM	SCC	II	$T_{4B}N_{2b}M_0$	CT+RT		CR	8	1	1F	1	4	0	3F	1	IV
10	Saravanan	54	M	SM,AL,SF	Lt BM	SCC	II	$T_3N_0M_0$	RT		CR	8	1	2F	1	0	0	2F	0	III
11	Balan	60	M	SM,AL,BN	Lt BM	SCC	II	$T_2N_0M_0$	CT+RT		PR	4	0	1F	0	2	0	2F	0	II
12	Panchacharam	39	M	AL,BN,PM	Oral tongue	SCC	II	$T_2N_0M_0$	CT+RT		CR	4	1	absent	1	2	0	absent	0	II
13	Vatsala	55	F	ST,COS	Oral tongue	SCC	II	$T_2N_0M_0$	SX	Wide local excision	PR	4	1	absent	0	0	0	absent	0	II
14	Majutha begam	50	F	BN	Rt BM	SCC	II	$T_2N_0M_0$	SX	Wide local excision	CR	4	0	3F	0	2	0	3F	0	II
15	David	74	M	SM,AL	Hard palate	ACC		$T_3N_0M_0$	RT+CT		PD	2	0	absent	1	4	0	absent	0	III
16	Srinivasan	40	M	AL,BN	Lt Com of lip	VC		$T_1N_0M_0$	SX	Wide local excision	CR	6	0	2F	0	0	0	3F	0	I
17	Rajan babu	61	M	ST,BN	Oral tongue	SCC	I	$T_2N_0M_0$	CT+RT		PR	4	0	absent	1	2	0	2F	1	II
18	Indra	55	F	BN	Rt BM	SCC	I	$T_1N_0M_0$	CT+RT		CR	8	0	1F	0	4	0	2F	0	I
19	Saraswathi	38	F	NIL	Rt Hard palate	MEC	LOW	$T_3N_0M_0$	CT+RT		PR	4	0	absent	0	2	0	absent	0	III
20	Kumaresan	55	M	SM,AL,BN	Oral tongue	SCC	I	$T_3N_1M_0$	CT+RT		PR	6	2	3F	0	4	0	3F	0	III
21	Srinivasan	60	M	SM,AL,BN	Oral tongue	SCC	II	$T_1N_0M_0$	CT+RT		CR	8	2	2F	1	3	0	3F	0	I
22	Pushpa	40	F	ST	Lt BM	SCC	II	$T_1N_0M_0$	CT+RT		PR	6	0	2F	0	2	0	3F	0	I
23	Rajangam	54	M	SM,AL,BN,SF	Lt BM	SCC	II	$T_3N_1M_0$	CT+RT		PD	10	1	1F	2	10	1	1F	2	III
24	Jayakumari	63	F	SF,BN	Oral tongue	SCC	I	$T_2N_0M_0$	RT		CR	8	0	2F	1	6	0	3F	0	II
25	Arasu	45	M	BN,AL,SM	Rt BM	VC		$T_1N_0M_0$	RT		CR	6	0	2F	0	0	0	3F	0	I
26	Sundar	40	M	AL,PM	Oral tongue	SCC	I	$T_2N_0M_0$	CT+RT		PR	4	0	2F	0	0	0	3F	0	II
27	Sasikumar	28	M	PM	Oral tongue	SCC	II	$T_1N_0M_0$	CT+RT		CR	6	1	3F	0	2	0	absent	0	I
28	paramasivam	65	M	AL,SM,PM	Oral tongue	SCC	I	$T_2N_0M_0$	CT+RT		PR	8	1	2F	0	4	0	2F	0	II
29	Valayan	60	M	SM,BN,AL	Hard palate	SCC	II	$T_1N_0M_0$	CT+RT		CR	8	0	absent	0	2	0	absent	0	I
30	Jayakumar	48	M	PM,BN,AL	Lt BM	SCC	I	$T_2N_0M_0$	CT+RT		PR	10	1	2F	0	4	0	2F	0	II
31	Amulraj	48	M	PM,BN,AL	Rt BM	SCC	II	$T_1N_0M_0$	CT+RT		CR	8	1	1F	0	4	0	3F	0	I
32	Jyothi	60	F	BN,ST	Rt BM	SCC	I	$T_3N_1M_0$	CT+RT		PD	8	1	2F	0	8	1	2F	0	III
33	Chandrababu	39	M	PM,BN,AL	Oral tongue	SCC	II	$T_1N_0M_0$	SX	Lt hemiglosectomy	PR	6	1	3F	0	2	0	3F	0	I
34	Subhiach	61	M	AL,BN,SM	Oral tongue	SCC	II	$T_3N_0M_0$	CT+RT		PR	8	1	3F	0	6	0	3F	0	III
35	Jayaselan	48	M	PM,BN,AL	Oral tongue	SCC	II	$T_1N_0M_0$	CT+RT		CR	6	1	2F	0	4	0	3F	0	I
36	Karunakaran	45	M	PM,BN,AL	Oral tongue	SCC	II	$T_2N_0M_0$	CT+RT		CR	8	1	absent	1	2	0	absent	0	II
37	Sanjeevi	59	M	AL,BN,SM	Oral tongue	SCC	II	$T_2N_0M_0$	CT+RT		PR	8	0	absent	0	6	0	absent	0	II
38	Meena	50	F	BN,ST	Lt BM	SCC	II	$T_3N_1M_0$	CT+RT		PR	6	1	2F	0	4	0	2F	0	III
39	Nagarajan	57	M	PM,SM,AL	Floor of mouth	SCC	I	$T_4N_0M_0$	SX+CT+RT	WLE+Marg mandibulectomy	PD	8	2	1F	1	8	1	3F	1	IV
40	Raghavan	45	M	PM,BN,SM	Oral tongue	SCC	I	$T_2N_0M_0$	SX+RT	Wedge resection + adv flap	PD	6	0	2F	0	4	0	2F	0	II
41	Babu	36	M	PM,BN,SM,AL	Oral tongue	SCC	I	$T_{4A}N_{2B}M_0$	CT+RT		PR	6	0	absent	0	2	0	absent	0	IV
42	Ranganathan	58	M	SM,AL,BN	Rt BM	SCC	II	$T_4N_0M_0$	CT+RT		PR	8	0	3F	0	10	0	1F	0	IV
43	Palanivel	47	M	PM,SM,AL	Oral tongue	SCC	I	$T_{4A}N_{2B}M_0$	CT+RT		PR	6	1	2F	0	4	0	2F	0	IV
44	Saraswathi	55	F	SF	Hard palate	SCC	II	$T_1N_0M_0$	CT+RT		CR	4	0	absent	0	2	0	absent	0	I
45	Rukmani	70	F	BN	Hard palate	SCC	I	$T_3N_0M_0$	CT+RT		PR	2	0	absent	0	2	0	absent	0	III
46	Rahamathulla	39	M	PM	Rt BM	SCC	II	$T_1N_0M_0$	CT+RT		CR	4	0	3F	0	2	0	3F	0	I
47	Moorthy	58	M	PM,BN,SM	Oral tongue	SCC	II	$T_2N_1M_0$	CT+RT		PR	8	1	3F	0	4	0	3F	0	III
48	Ethiraj	65	M	BN,AL,SM	Rt BM	SCC	II	$T_{4a}N_0M_0$	CT+RT		PR	6	1	2F	0	4	1	2F	0	IV
49	Suruli	70	M	BN,SM,AL	Oral tongue	SCC	I	$T_4N_{2b}M_0$	CT+RT		PR	4	0	1F	1	4	0	1F	1	IV
50	Vijaya	48	F	BN,SF	Lt BM	SCC	I	$T_3N_0M_0$	SX+CT+RT	CR + PMMC flap	PR	8	0	2F	1	4	0	3F	0	III

PATIENT INFORMATION SHEET

TITLE: A PROSPECTIVE STUDY OF MANAGEMENT OF ORAL CAVITY CANCERS

I, Dr. Ganga P, Post graduate student, MS in Otorhinolaryngology, Government Stanley Medical College is going to undertake the study on the above-mentioned topic.

The purpose of this study is to study about the various management of oral cavity cancers and to study effectiveness of various management modalities

It also studies about the comparison of different modalities of treatment in management of oral cancers

It also helps to identify the risk factors associated with oral cancers

I assure that all the information provided by you will be kept highly confidential and privacy is assured. Your identity would not be revealed to anyone. The study may be published in scientific journal, but your identity will not be revealed.

Your participation in this study is voluntary and you can withdraw from this at any point of time

Signature/left thumb impression of the participant

INFORMED CONSENT

TITLE: A PROSPECTIVE STUDY OF MANAGEMENT OF ORAL CAVITY CANCERS

The content of the information sheet dated _____ that was provided have been read carefully by me/explained in detail to me, in a language that I comprehend and fully understood the contents.

I confirm that I have had the opportunity to ask questions.

The nature and purpose of the study and its potential risks/benefits and expected duration of the study and other relevant details of the study have been explained to me in detail.

I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal right being affected.

I agree to take part in the above study

_____

(Signature/Left thumb impression)

Name of the Participant: _____

Son/Daughter/Spouse of _____

Complete postal address: _____

This is to certify that the above consent has been obtained in my presence.

_____ Date:

Signature of the principal investigator Place:

1) Witness – 1

_____

Signature:

Name:
Address:

2) Witness – 2

_____

Signature:

Name:
Address:

INFORMED CONSENT

தகவல்தொடர்புஒப்புதல்படிவம்

TITLE A PROSPECTIVE STUDY OF MANAGEMENT OF ORAL CAVITY CANCERS

நான்தகவல்நகலில்கொடுக்கபட்டுள்ளமுழுவிவரங்களையும்கவனமாகப்படித்தேன். ஆய்வின்முழுவிவரங்களையும்தமிழில்எனக்குவிளக்கமாகஎடுத்துக்கூறப்பட்டது.

நான்இந்தஆய்வின்விவரங்களைமுழுமையாகபுரிந்துகொண்டேன்.

ஆய்வில்பங்குஎடுக்கும்போதுஏற்படும்சாத்தியமானஅபாயங்கள்மற்றும்பயன்களை நான்அறிந்துள்ளேன்.

மேலும், நான்எந்தஒருவேளையிலும்ஆய்வில்இருந்துதிரும்பமுடியும்என்றும், அதன்பின்னர்நான்வழக்கம்போல்மருத்துவச்சிகிச்சைப்பெறமுடியும்என்றும்புரிந்து க்கொள்கிறேன்.

நான்ஆய்வில்பங்குகொள்ளபணம்எதையும்பெறமுடியாதுஎன்றுஅறிந்துள்ளேன்.

நான்இந்தஆய்வில்பங்குஎடுப்பதன்மூலம்நான்என்னசெய்யபோகிறேன்என்றுதெரியு ம்.

நான்இந்தஆய்வில்என்முழுஒத்துழைப்பையும்கொடுப்பேன்என்றுஉறுதியளிக்கின்ற ேன்.

ஆய்வில்பங்கேற்பவர்பெயர்: *சாட்சி:*

பெயர்மற்றும்முகவரி: *பெயர்மற்றும்முகவரி:*

கையொப்பம்/விரல்ரேகை *கையொப்பம்/விரல்ரேகை*

ஆராய்ச்சியாளராககையொப்பம்மற்றும்தேதி

INFORMATION SHEET

தகவல்நகல்

TITLE A PROSPECTIVE STUDY OF MANAGEMENT OF ORAL CAVITY CANCERS

இந்தஆய்வில்உங்களிடம்கேட்கும்கேள்விகளுக்குமுழுமனதுடன்பதில்அளிக்க வேண்டும்.

இந்தஆய்வில்உங்கள்நாள்பட்டநோய்கள்,
உடல்நலம்தேடும்நடத்தைதொடர்பாகவிவரங்கள்பற்றியவிவரங்களபற்றிகே ட்கப்படும்

இந்தஆய்வில்உங்களுக்குஎந்தபின்விளைவும்ஏற்படாதுஎன்பதைநான்உறுதி யளிக்கிகேன்.

உங்களிடம்கேட்கும்கேள்விகளில்உங்களின்சுயவிபரம், குடும்பவிபரம்,
தொழில்விபரம், விபரங்கள்மற்று

ம்இதரவிபரம்அடங்கும்.

உங்களுக்குபணம்எதுவும்அளிக்கபடாதுஎன்பதைஇதன்மூலம்தெறிவிக்கிறே ன்.இந்தஆய்வில்பார்வைநரம்பியல்நோயாளிகளுக்குவண்ணபார்வைக்குமா றுபட்டஉணர்திறன்விளைவைமதிப்பீடுசெய்வதேஆய்வின்நோக்கம்இந்தஆ ய்வில்உங்களுக்குநேரடிபயன்எதுவும்இல்லைஎன்றாலும்நீங்கள்அளிக்கும்தக வல்மூலம்புதுயுக்திகள்வகுக்கப்படலாம்.
அதன்மூலம்வருங்காலத்தில்உங்களுக்கோஅல்லதுஉங்களைபோன்றமக்களு க்கோபயன்படலாம்.

உங்களின்விபரங்கள்எதுவும்மற்றவர்களுக்குதெரிவிக்கப்படாதுஎன்பதைஉ றுதியளிக்கிறேன்.

இந்தஆய்வின்முடிவுகள்பத்திரிக்கைளில்பிரசுரிக்கபடலாம்ஆனால்உங்களி ன்அடையாளம்எதுவும்காட்டப்படாது.

உங்களுக்குவிருப்பம்இல்லைஎன்றால்எப்போதுவேண்டுமானாலும்இந்தஆய் வில்இருந்துவிலகிக்கொள்ளலாம்.
அதனால்உங்களுக்குஎந்தபாதிப்பும்இல்லை.

கையொப்பம்/ இடதுபெருவிரல்ரேகை