

Oral Squamous Cell Carcinoma

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Cancer

- Is an uncontrolled Proliferation of Cells.
 - Cells never stop dividing.
 - some fast, others slow, this what distinguish malignant from benign. Benign eventually will stop dividing.
 - Cancers are clones, originating from single cell.
- Therefore cancer is a Genetic Disease, arising from mutated genes.

CARCINOMA

SARCOMA

Oral Squamous Cell Carcinoma

Definition and Cell of Origin

Causes

Process of Carcinogenesis (Cancer development)

Local Spread and Metastasis

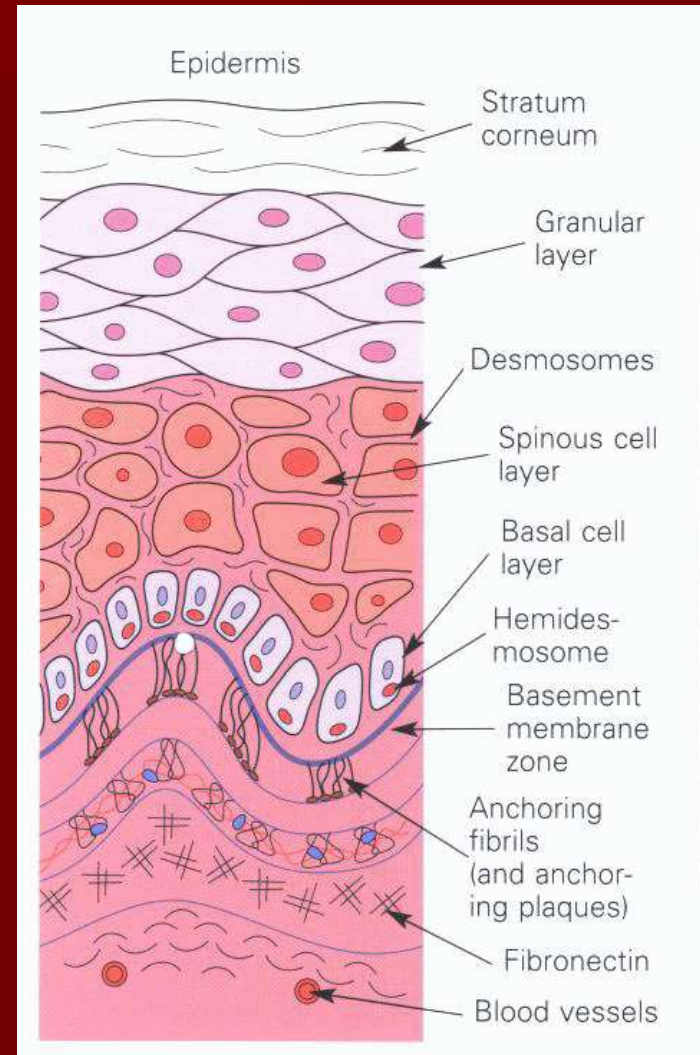
Progression

Clinical Presentation and Staging

Treatments

Origin

- The cell of origin of Oral Squamous Cell Carcinoma is from oral Keratinocyte.



Causes

- DNA mutation. Either
 - Spontaneous Mutation.
 - Induced Mutation by exposure to a range of mutagens,
 - Chemical,(smoking)
 - physical, (sharp tooth)
 - Viral , (Human papilloma virus)
- Deficiency in the enzymes that repair DNA damage
Xeroderma Pigmentosum.
- Genetic Difference in the Immune Response,
also influence development of OSCC.



DNA mutations

- DNA mutations affects a number of **GENES**, disturbing the growth control
 - Oncogenes genes whose over activity, driving cell proliferation
 - Tumor Suppressor Genes, P16, P53. Reduction in activity.

Risk Factors for Cell Mutations

- Tobacco, Alcohol, Betel,
- Radiation Exposure
- Infection,
- Immunincompetence,
- Environmental التلوث البيئي
- Genetic Factors
- Low fruit and Vegetable intake
- High Sugar and Fat intake

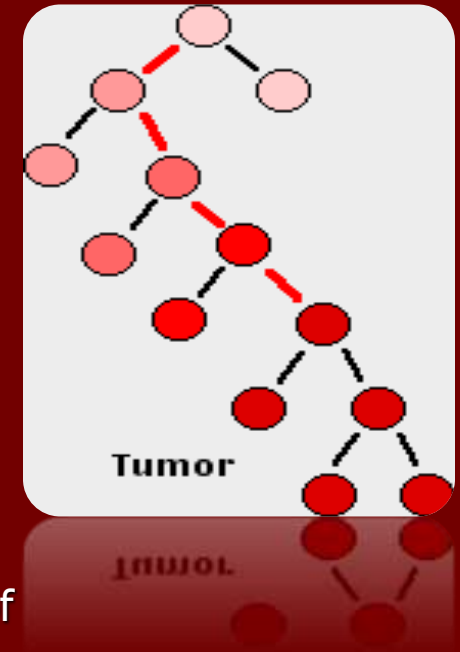
Other Risk Factors for Cell Mutations

- Relationship with Human Pappilloma Virus (HPV), especially Oropharyngeal Carcinomas.
- Relationship with Candida and Syphilis Infection.
- Relationship with poor oral hygiene, sharp tooth, edge of a denture or periodontal disease
- Defect in immunity, HIV, AIDS.
- Diseases such as Xeroderma Pigmentosum
Fanconi Anemia, LiFraumeni Syndrome,
Discoïd Lupus Erythamatosi, Diabetes
- Potentially Malignant Disorders, (Leukoplakia)

Development of Cancer

Process of Carcinogenesis

- A single cell in a tissue suffers a mutation (red line) in a gene involved in the growth cell cycle, oncogen or tumor suppressor gene
 - This results in giving that cell a growth advantage over other dividing cells in the tissue. میزه نمو
 - As this cell develops into a clone, some of its descendants suffers another mutation (red line) in another cell cycle gene This further deregulate the cell cycle of that cell and its descendance
 - Eventually, so many mutations have occurred that the growth of that clone becomes completely unregulated
- The result : full-grown Cancer.



Process of Carcinogenesis

The various changes in the DNA can progress from **normal Keratinocyt** to a **pre-malignant cell** that is characterized by the ability to proliferate in a less controlled fashion than normal.

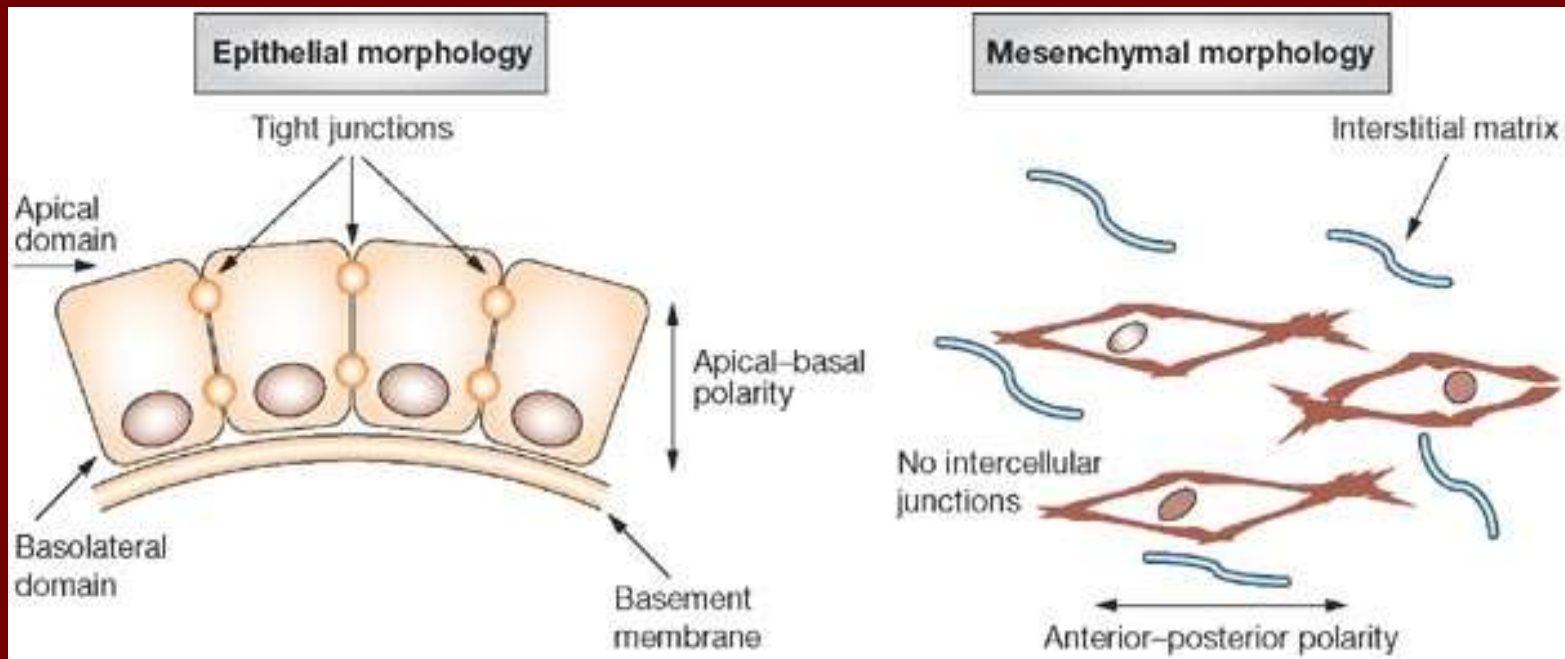
The cells then become autonomous) الأعتماذ الذاتى and **true cancer** cell, which is characterized by Invasion across the epithelial basement membrane.

Crispian Scully, Jose Bagan, 2009, Oral Oncology.

This is the early stage of tumor development and what follows is the progression of tumor to a more Advance stage.

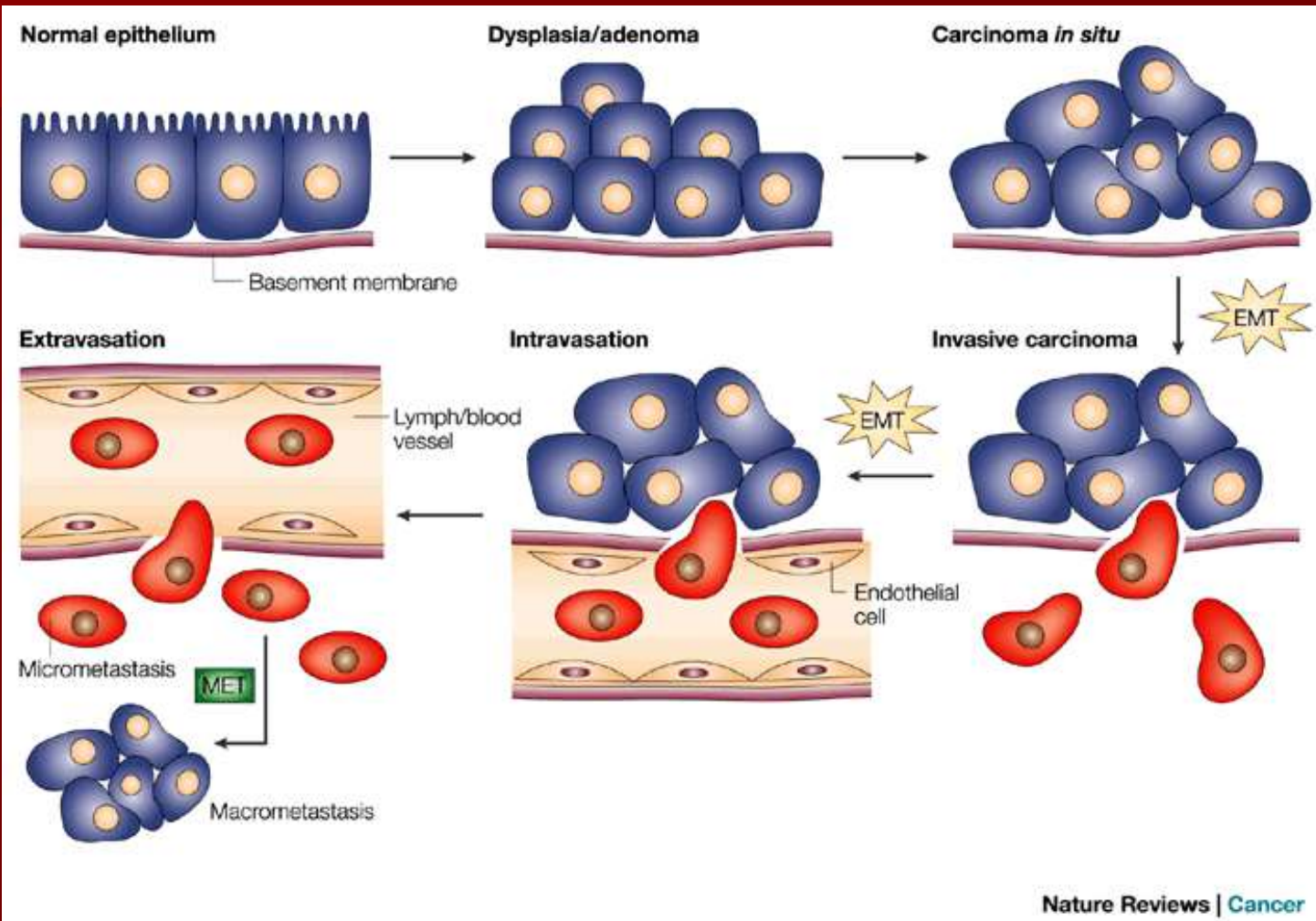
Local Spread and Metastasis

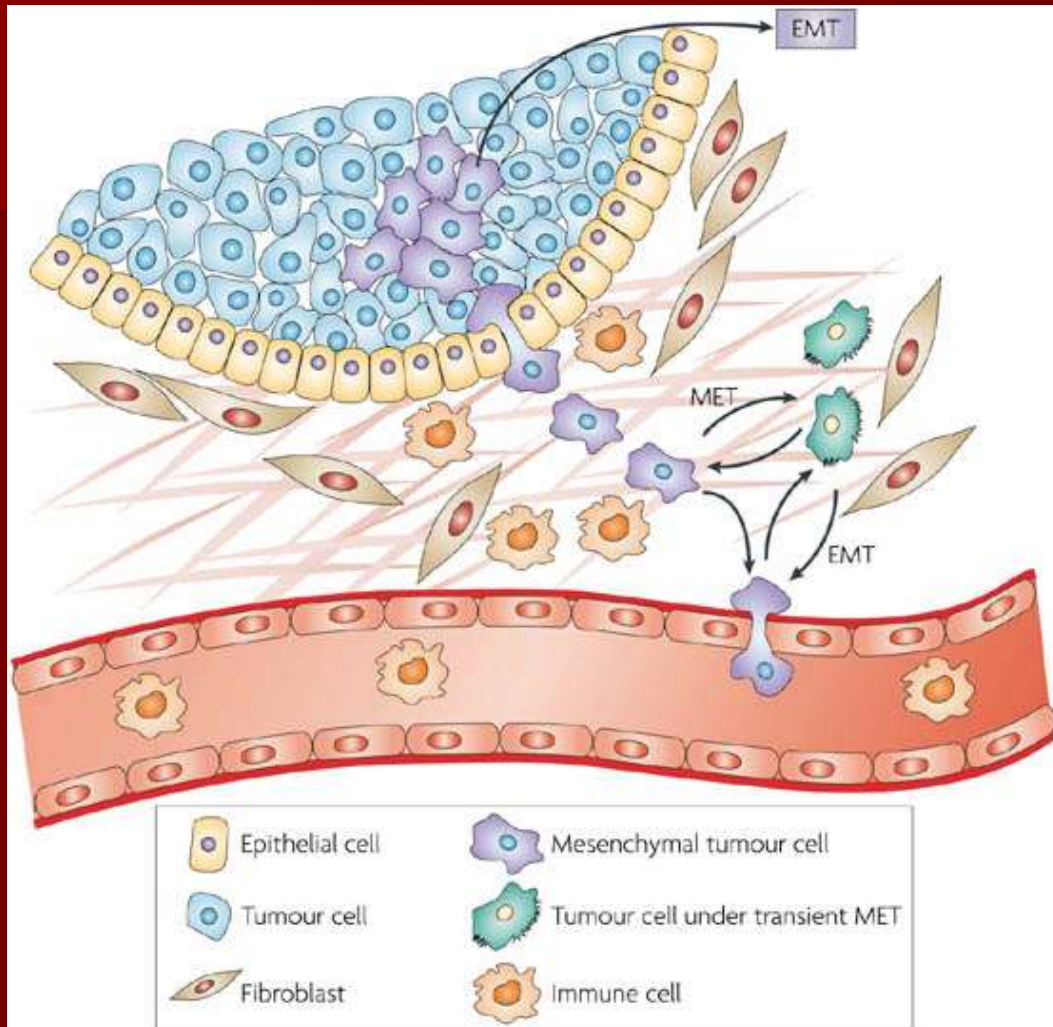
Epithelial Mesenchymal Transition.



Local Spread and Metastasis of Cancer

Epithelial Mesenchymal Transition (EMT) This is the process by which Epithelial cell lose their cell polarity and cell-cell adhesion, and gain migratory and invasive properties to become mesenchymal stem cells; which are multipotent stromal cell that can differentiate into a variety of cell type.





Tumor Progression

(In The Early stage of tumor development)

Once the Tumor has formed

Further growth depends on T. H. interaction

The HOST provide, Nutrition (+)

Growth Factors (+)

Hormones (+)

Immunity (-)

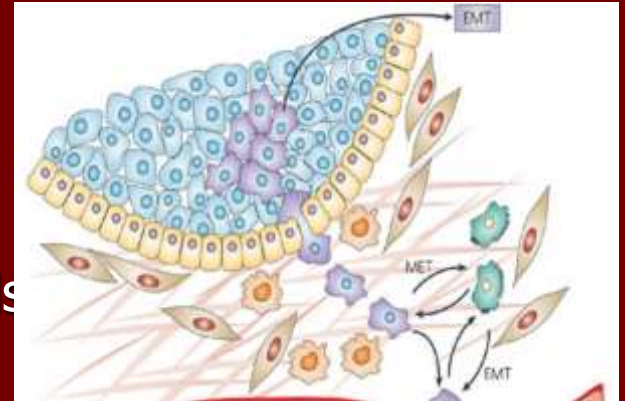
Immunity present a real THREAT to tumor cells
in their early stages of development.

The tumor takes a **slow** spontaneous growth

Delay in autonomy

تأخير في التحكم الذاتي

للخلايه السرطانيه



LOW IMMUNITY Can occur in

■ Old Age Patients

- 1- There is Reduction in T-Cell function.
- 2- Decrease in primary immune response of B-Cell, especially for those responses requiring T-Cell interaction .

- Makinodon T. Immunodeficiency and aging. The immune System function and therapy of dysfunction. Edited by G. Gloria and A.Eshkel. p. 56.

■ Patients with Head and Neck Cancer.

- 1 - There is depression in most aspects of cell mediated immune response .

Scully C . The immunology of cancer of the head and neck wit Particular reference to oral cancer. Oral surg Oral Med Oral Pathol 1982;53(2):157-169

- 2- There is functional depression of NK activity.

Vinzenz K, Micksche M. Systemic and regional natural

Cytotoxicity in patients with head and neck cancer .
J Maxillofac surg 1986;14(5):270-275

■ In old age patients

- 1- There is an increase incidence of OSCC.
- 2- The growth rate of primary tumor is very slow.
- 3- Metastasis to cervical L.N. is very late.
unless the primary tumor has been violated with a biopsy, or incomplete excision of primary tumor.

Ali Alshawi, Age and biopsy as predictive factors in the management of oral squamous cell carcinoma

basrah journal of surgery 2010, volume 16, 80-

Clinical Presentation

Is usually late because,

- Lesion is asymptomatic.
- No regular attendance and negligence of patients
- Failure of General Practitioner to make a diagnosis.

Clinical Manifestation

Site

OSCC, usually invade multiple sites by direct extension.

Tongue..floor..alveolus..tonsillar region

Floor of mouth..post.alveolus..lingual sulcus,
Cheek, gingiva, maxilla, maxillary sinus

Trigon of, (retromolar region+ floor of mouth+tongue + pterygomandibular raphe)

Clinical Presentation

Early Lesion,

- Exophytic mass with area of ulceration.
- Ulcer in a depth of fissure of tongue.
- Superficial Ulceration
- Leukoplakia
- Erythroplakia with peripheral streaks of leukoplakia.

Clinical Presentation

Typical Lesion of OSCC

ULCER.

- Hard,
- Raised everted edge.
- Indurated.
- Granular bed, bleeds easily.



Verrocous Carcinoma



Exophytic Carcinoma



What you look for Ulcer

Site

Size

Shape

Surface

Base

Number

Lymph Node Involvement

Signs and Symptoms

Pain.

Dysphagia. involvement of Masticatory muscles

Restricted Mouth Opening.

Swelling.

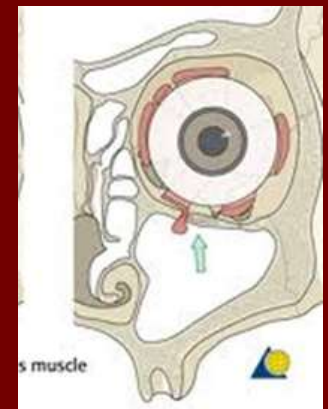
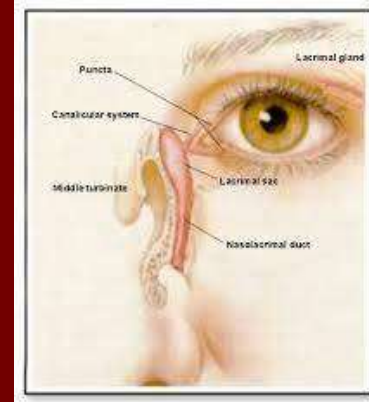
Fungating Mass

Eye symptoms

Nose Symptoms

Mobility of Teeth.

Unhealed Extracted tooth socket.



Management.

- The patient
General Medical Condition of the Patient.
- The Tumor
Local Assessment of the Tumor
 - Diagnosis
 - Prognosis
 - = Prognostic Indicators
 - Clinical Staging
- The Surgeon
Treatment plan

1- The Patient

Any Medical Condition that can compromise
treatment

2-The Tumor

Local Assessment

■ Diagnosis

Is primarily Clinical.

Careful History, Date of Onset of signs, symptom.

Clinically, look for Site, size, Texture, Tethering, Indurations.

L.N. Metastasis , Distance Metastasis.

Investigation

Biopsy

CT,MRI

Tumor

Prognosis توقعات سير المرض Prognostic Indicators

- Size.
- Site. The more posterior, the poorer is the prog.
- Depth of Invasion.
- Local Spread, to muscles, perineural, bone.
- Involvement of Lymph Nodes.
- Distance Metastasis.
- Histopathology.
- Multiple Primaries, (Field of Cancerization).
- Tumors who have been treated before.

Prognosis

Prognostic Indicator

- Depth of Invasion.

Tumor Depth of greater than 1.5 cm. is considered to be of poor prognosis

Prognosis

Prognostic Indicators

➤ Local Spread.

Behavior of oral carcinoma in Young, Old patients

Invasion of local soft tissues,

Superficial infiltration, just beneath m.m. or

Deep spread to muscles.

Perineural spread.

Invasion of Bone

Through anatomical structures

Through Node metastasis

Through the crest of alveolus of edentulous jaw.

Prognosis

Prognostic Indicators

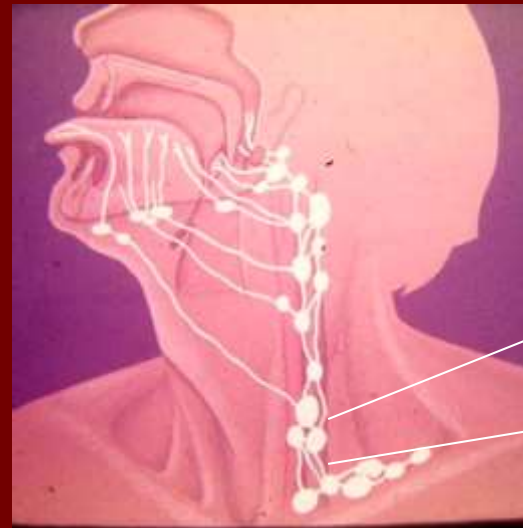
➤ Lymph Node Involvement

- Regional L.N. involvement, result in 50% reduction in 5 year survival Rate.
- Transcapsular spread of the tumor result in a poor prognosis.
- L.N. metastasis in the lower cervical group of nodes is an adverse prognostic factor.



Submental, Submandibular
Jugulo-digastric

Supra-omohyoid



Deep jugular

Posterior triangle

Prognosis

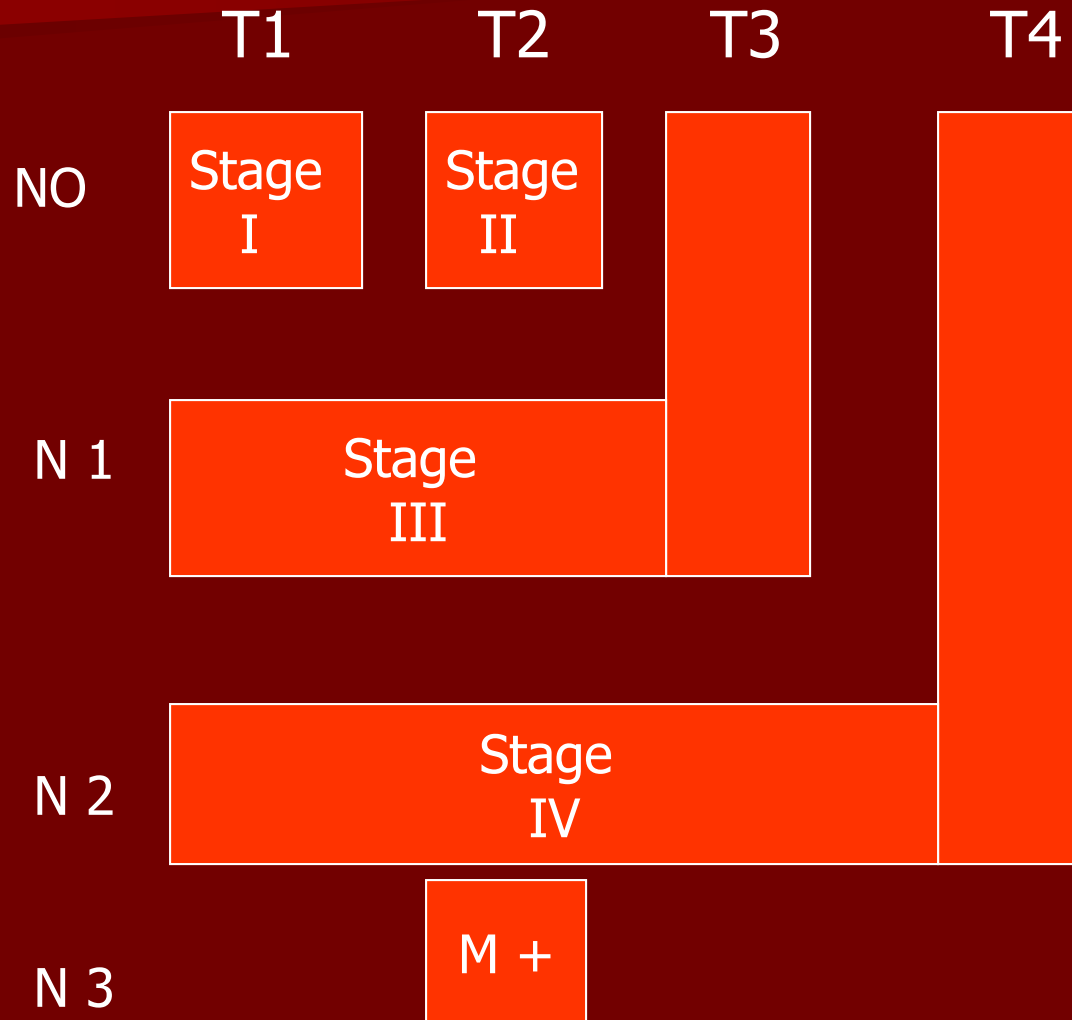
Prognostic Indicators

- Histopathology.
 - The loss of Epithelial Stratifications.
 - Presence of tumor cells in the Stroma.
 - Poorly developed Immune Inflammatory Response within and around tumor.

Clinical Staging of Oral Cancer

➤ TNM

تصنيف مراحل المرض



T1 = < 2 cm Tis = tumor in situ

T2 = 2—4 cm

T3 = 4—6 cm

T4 = > 6 cm. invading adjacent st.

N0 = no regional L.N. met

N1 = ipsilateral adenopat
single, < 3 cm.

N2 = single node 3—6 cm.
or multiple nodes.

N3 = large ipsilateral or
contralateral.

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■ Treatments

Principle of Treatment.

- Objective of Treatment ?
- What are the Limitations of Treatment ?
- Quality of Life ?

Treatment must prolong the patient life as a reasonable human being.

Cure of the disease must never be worse than the disease itself.

Objectives of Treatment

- Cure
- Cure with sever disability
- Palliative Treatment تخفيف من معاناه المريض
- Salvage Treatment

Treatment

Treatment must prolong the patient life with reasonable function and esthetics.

Cancer patients require accurate

- Diagnosis
- Evaluation of present condition
- Treatment Planning
- Careful reconstruction
- Rehabilitation.

Early Diagnosis is the most single important factor that improves the end result of H&N cancer

Treatment

Surgery

Radiotherapy

Chemotherapy

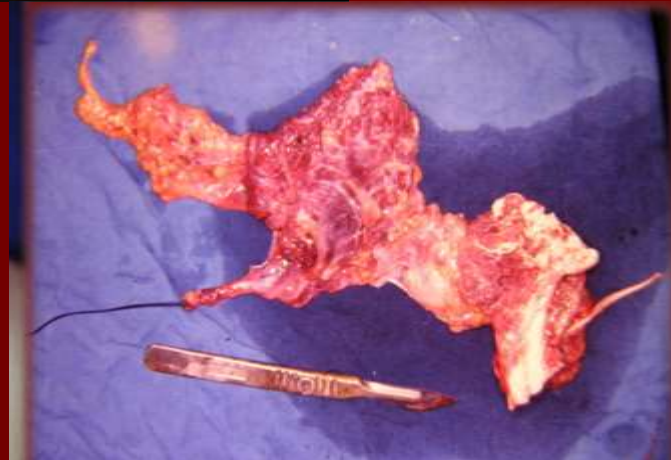
Surgery

Radical Excision of the Tumor.

- 1- Preoperative assessment of extent of tumor, C.T., MRI.
- 2- Normal anatomy and function should be retained whenever possible, but never be allowed to compromise the excision.
- 3- Cosmetically acceptable,
Consider Surgical Reconstruction with Flaps

Radical Neck Dissection

Enblock Resection of the Primary Tumor and
L.N.of Neck in one peice



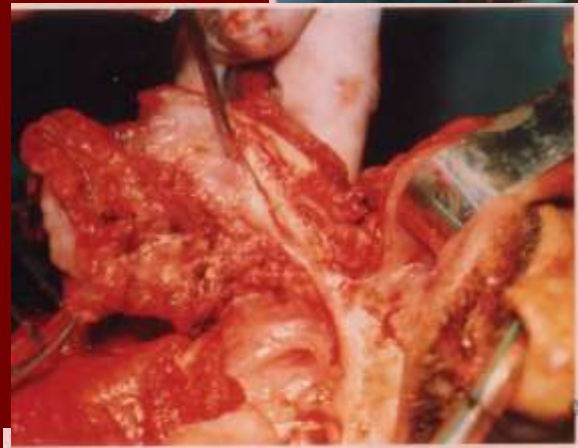
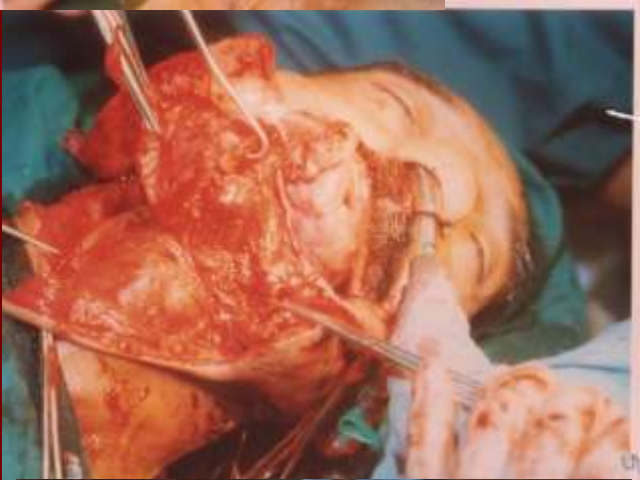
Supra-omohyoid Neck Dissection

Selective Cervical Lymph Nodes
Dissection, that removes the content of
Submental, Submandibular (L I)

Jugulo-digstric L.N. (L II)

Jugulo Omohyoid L.N (L III) and their
lymph Nodes Bearing Tissues located
anterior to the Cutaneous Branches of the
Cervical Plexus.





Modified Neck Dissection

Radical neck dissection

With preservation of

Internal Jugular Vein

Spinal Accessory Nerve

Sternomastoid muscle

Radiotherapy

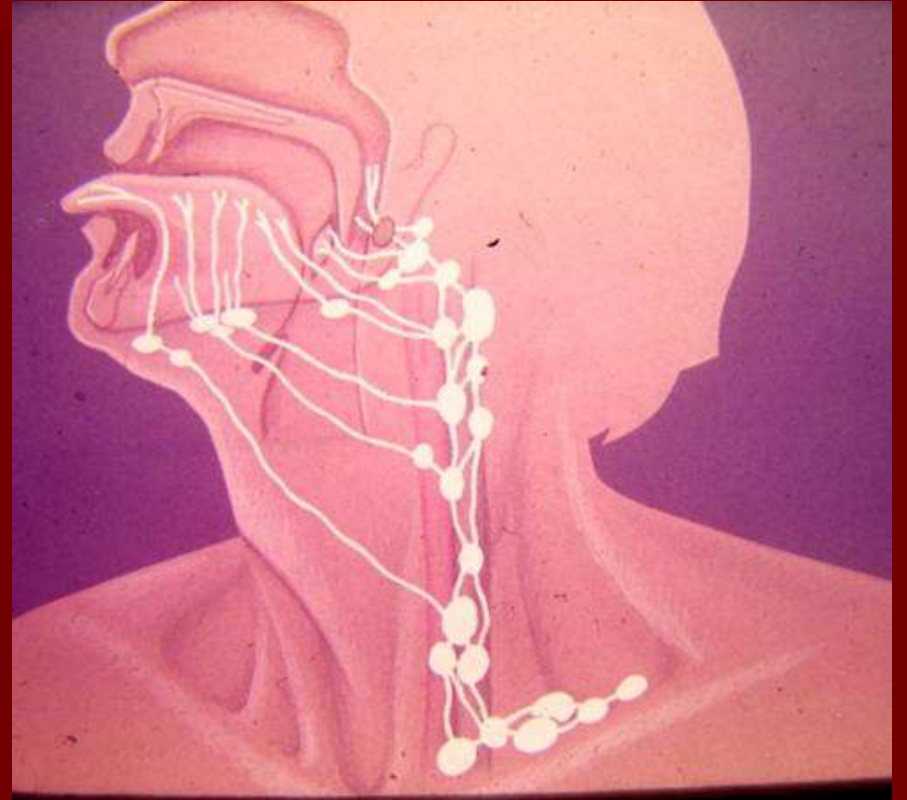
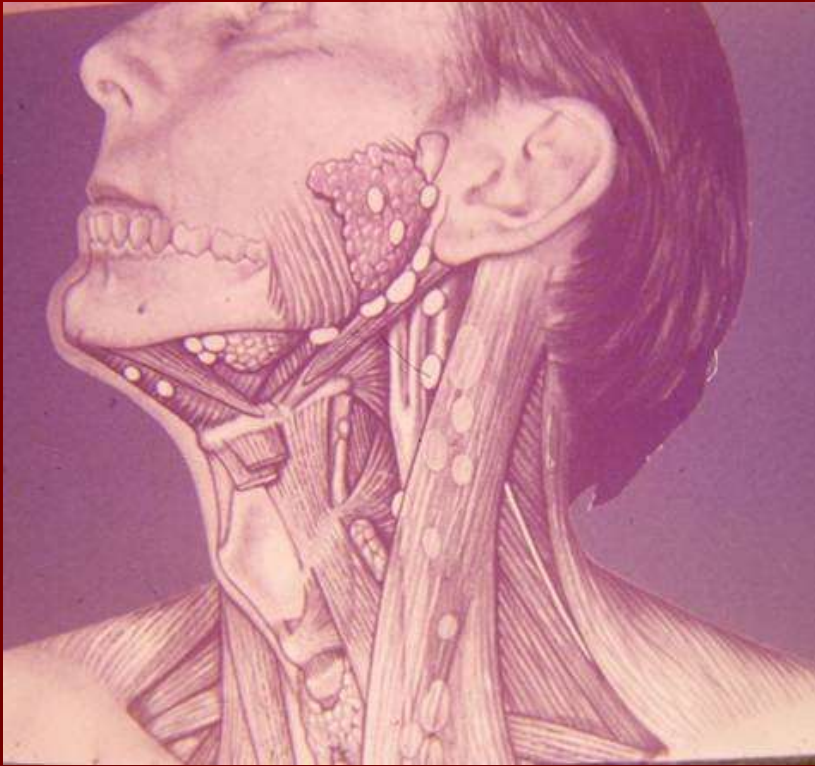
Radio sensitivity depends on

1-Type of Oral Cancer.

2-Size of the tumor,

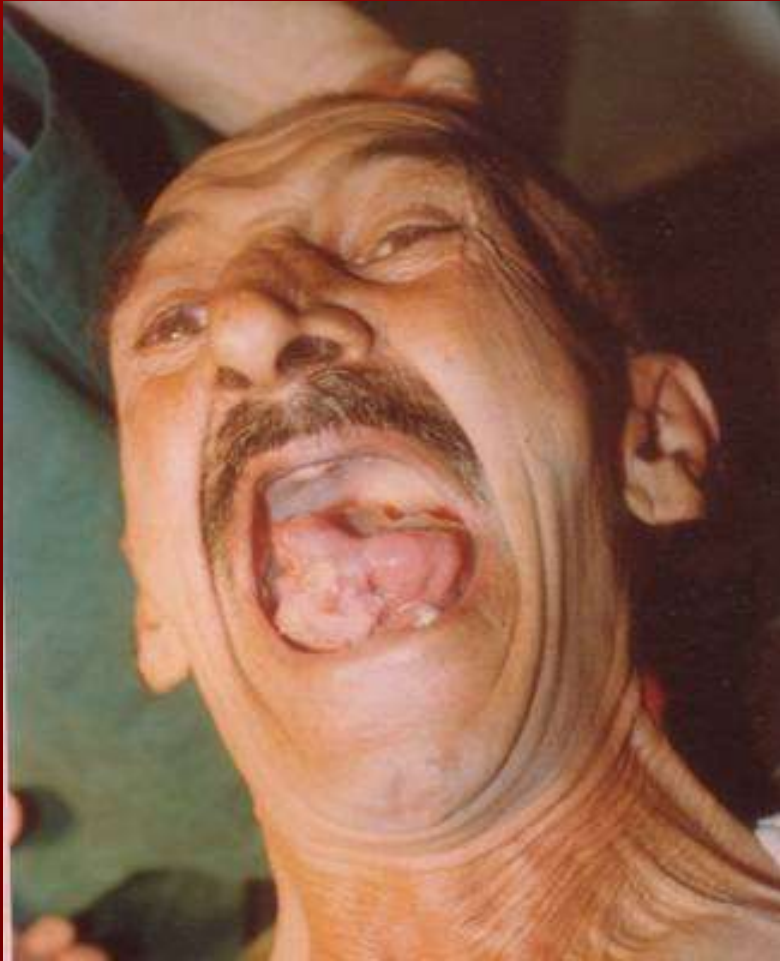
The more cells are present the more likely are some to survive irradiation and regenerate the tumor.

As tumor grows, it tends to outstrip its blood supply so that more cells become hypoxic and therefore more radioresistance.

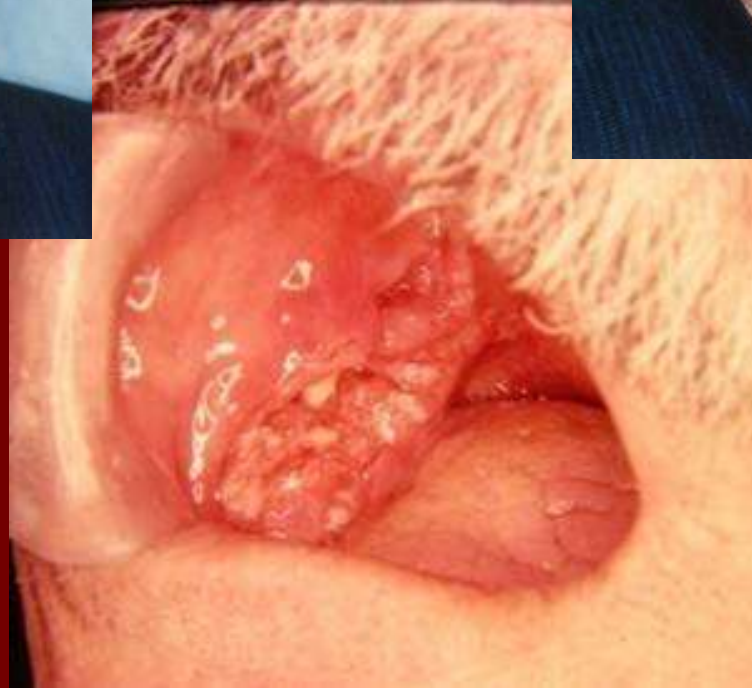


CA Tongue





CA Cheek



CA Mandible







Case No 4

