



Aphthous ulcer = Canker sores

- 1) superficial mucosal ulceration
- 2) extremely painful
- 3) in children lead to feeding difficulty
- 4) it happens for unknown reason
- 5) in most cases it spontaneously resolve after 7 to 10 days.

Morphologically: ① shallow ulcerations

② Covered by exudate

↓ (composition of neutrophils
inflammatory cells +
yellowish or white fibrin and cellular
cellular debris)

[as a membranous structure
above the ulcer]

③ surrounded by hyperemic

→ Maybe associated with Familial clusters
or associated with some immunological
diseases (celiac disease + inflammatory
bowel disease + Behçet disease)

Note: First presentation in Chron disease is Aphthous ulcer

good prognosis

Treatment: without
or some vitamins (A)
because they cause
reepithelization



Soft tissue lesions:

[1] Irritation Fibroma



- oral cavity condition lead to C.T. and fibroblast proliferation.

- Bening (بشيرة وجنس البزخ)

[2] Pyogenic granuloma



it's an
inflammatory
lesions

- Chronic irritation or inflammatory process or recurrent trauma and tissue injury

- it's a proliferation of newly formed blood vessels + edema + inflammatory cells



lead to form
a red to pink color
mass at the site of
lesion.

- it's alarming (rapidly proliferating)



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[3] ossifying fibroma

[4] peripheral giant cell granuloma

↓
• proliferation of blood vessels + stromal cells
Multinucleated ~~Macro~~ giant cell

• Reactive process against minor tissue injury

• peripheral? ! to differentiate it from giant cell granuloma inside bone (Mandible or Maxilla) which is called central Giant cell granuloma but peripheral giant cell granuloma it's in the soft tissue.

Herpes simplex virus infection:

① clinically is associated with stressful condition (as major surgery →) any ② condition that affect the immune system " أي حالة تؤثر على الجهاز المناعي " may cause HSV infection

③ excessive heat exposure



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types of HSV infections:-

• Primary infection:

- 1) in early age (children)
- 2) called primary gingivostomatitis
- 3) after resolution it's remain as latent infection
- 4) HSV appear in epithelial cells and most frequently in ganglion cells (spinal ganglia) in its neurons

• Secondary infection

- 1) at a stressful condition
- 2) The virus can be reactivated
- 3) by the nerve process (dendrites) in the mucocutaneous border

reactivation in adults called recurrent herpetic stomatitis.

Morphologically:- is a collection of vesicles (fluid filled spaces in the epidermis) this vesicles may ruptured or ulcerated

HSV infection * clinical diagnosis + self-limited or some antiviral used



Microscopically: viral DNA and its material (viral products) may accumulate inside the nucleus or cytoplasm.

Called in HSV infection Cowdry body. "homogeneous material in nucleus" → inclusion bodies. make

كَيْفَ تَكُونُ vesicles? epidermis made of keratinocytes. it has a intercellular connections between cells "desmosomes" that keep the connection of the epithelial layer. in infection these desmosomes ruptured and the fluid accumulate between cells to make the vesicles.

Tzanck test: Scraping of an ulcerating area → processing → staining → under the microscope

Oral Candidiasis (Thrush)

- most common in children
- agent mostly: *Candida albicans*
- predisposing condition related to (immunosuppression) + (stressful conditions)



Morphologically: it has a
pseudomembrane
↓
(fungal structures +
fibrin + cellular debris)
is a
loosely
attached
membrane [can be
scrapped easily]

after scraping it → under the
pseudomembrane there's an edematous
hyperemic mucosa. → may lead to
hyperplasia.

Deep fungal infection:-

- superficial and can be treated easily as in candidiasis
- mostly in immunocompromised patient. (HIV or cancer patients + organ transplanted patients)
- as: histoplasmosis
blastomycosis
- Mucor mycosis and Covid-19
↓
in oral and nasal area in covid
pandemic



Oral manifestations of systemic disease:

hairy leukoplakia

- Mucosal changing as a result for EPV infection.
- mostly in immunocompromised
- an important thing during HIV outbreak so it may precede AIDS.

Morphology: at the lateral border of the tongue + in its white structure there's an upward extensions "as hair"

Can't be scraped

- white patches: is Keratine produced by squamous cells and it's a spike like structure

Microscope: keratinized squamous in unusual sites inside the oral cavity +

balloon cells: (Keratinized cells) swollen and have a pale color "eosinophilic" when comparison with the surrounding keratinocytes.



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* pre-cancerous lesions

leukoplakia

erythroplakia

Changes still

In cell under

the basement membrane.



when it ruptures the basement membrane and penetrates it and reach the epithelial tissue → called invasion

membrane

الخلايا

displasia

Sample

CT scan

Thickening
raised
area

Leukoplakia: white patch or plaque that cannot be scraped

it has dysplasia

Full Thickness

Carcinoma in situ

rarely: it's a white

and red area

together as a

combined form between

leukoplakia and erythroplakia

called

leukoerythroplakia

or

Speckled leukoerythroplakia

1.5% of these lesions may progress to invasive carcinoma within 5 years



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Squamous Cell Carcinoma (SCC)

- The vast majority of head and neck cancer are SCC.
- head and neck SCC 6th most common neoplasm in the world.
 - ↓
- incidence 500,000 in USA and 6500,000 worldwide.

Pathogenesis: [1] infection with high risk HPV

disease at surface
Sexually transmitted

in oral cavity → HPV virus infection
mainly HPV-16 transmitted by sexual
is in extraordinary sexual activity (oral sex)

risk of infection ↑ associated with multiple partners.

[2] • tobacco and alcohol

in Asia

← [3] • chewing of Betel quid and paan

[4] • Actinic radiation

Note

Tongue SCC is in rise in young age individuals with no risk factor.



80% of oropharyngeal SCC
associated with HPV mainly 16

in the past 2 decades the
SCC increased 2 fold over

HPV has a vaccine

HPV associated SCC is
mainly in Post. Part of the tongue
tonsils

Post. wall of oropharynx
but is uncommon in the rest
of the oral cavity

- prognosis based on stage
of the cancer.
- HPV related SCC has
a good prognosis in comparison
with classic type.

Field Cancerization: in the cavity organs
all the surface exposed
to cancer + genetic
mutations.

- SCC genetic mutation are accumulated
(defect in repair genes the
accumulation of mutation is faster)



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Molecular pathology of SCC:-

- 1- **TP53** → Garden of the genome
→ Important in genetic mistakes (mutations)
function: detect the mutation

if repaired done
if not → apoptosis ← activate repair genes

- 2- **CDKN2A (p16)** → in cells the
↓
cyclin Depend.
Kinase inhibitor
cyclin + CDK make a complex for cell proliferation
↓
CDK-inhibitors to control the complex

mutation in oral cavity SCC
يقفل على الشفاه

- 3- **RB gene** bind to **E2F** → activation
when active → hypophosphorylation
when inactive → hyperphosphorylation

one of HPV products E7 bind to RB gene pocket and prevent it to bind to E2F → so E2F free and lead to cell division



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Types of oral cavity

Classic HPV
HPV associated SCC and Non HPV SCC

Classic: Keratinizing / old age
Pathogenesis at the rest of oral cavity
associated except (tonsils & post. part of the
with tongue & post. wall of oropharynx)
alcohol + tobacco

Presented by pre-cancerous lesions
as leukoplakia and erythroplakia
in early stage → distant metastasis

HPV:
SCC

non Keratinizing / young age
virus-related SCC
without preceding pathological
conditions as (leukoplakia and
erythroplakia)

associated with sexual activity
in early stage it made
a metastasis in regional lymph
node (cervical lymph node)

second primary
risk of good prognosis than classic
higher in classic than HPV SCC

Classic location
Low border of the lip
floor of the tongue
Lateral border of the tongue