

**Title of Lecture: Head AND Neck**

**Date of Lecture: 8/2/2015**

**Sheet no:3**

**Refer to slide no. : 1 ,Dr.Heyam**

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**🡪Slide #2 :**

* The first 3 lectures will be about head and neck :

1. diseases of the oral mucosa

2. diseases of the jaw (cysts)

3. diseases of the salivary glands

\*\* 80% of the exam questions will be from the lectures BUT you have to read the book ☺

\*\*reference: Robbins basic pathology. ( it's just 5 pages under the title of oral cavity )

**🡪Slide #3:**

**♥ Oral mucosa disease are 2 type :**

1. **inflammatory lesions:**
2. Aphthous ulcers,
3. herpes simplex, (viral infection)
4. candida.( fungal infection)

* Inflammation is not a disease ; it's body response to injury which might be infection ,trauma , or sun stroke …etc.
* Inflammation has consequences "collateral damage" and that’s why we treat it (Inflammation protects our bodies from injuries but it have systemic effect on our body , so we treat it).
* What is the difference between *inflammation* and *infection*?

# infection : microorganism cause inflammation and disease

# inflammation : it's the body response to many stimuli , one of them is the infection .

1. **proliferative and neoplastic lesions:**
2. fibrous proliferative lesions,
3. leukoplakia,
4. erythroplakia,
5. squamous cell carcinoma.( the most common malignancy in oral mucosa )

# proliferation 🡪 the cells are dividing forming a mass that is not necessary to be malignant , sometimes it could be benign or malignant and sometime it could not be neoplastic at all ; it's just a reaction .

**🡪 Slide #4 :**

**APHTHOUS ULCERS**

* it's a very superficial mucosal ulcerations.
* 40% of the population.
* more common in the first two decades of life.
* painful.
* recurrent.

**🡪Slide #5 :**

You can see white base of ulcer surrounding with a red hyperemic rim.

**🡪Slide #6 :**

\*cause: unknown

\*it have some association of certain disease :

1. Celiac disease : disease in GI system , and the patients have a sensitivity to gliadin protein in wheat ( wheat allergy ) , which is a disease of small intestine lead to malabsorption.

2. IBD ( inflammatory bowel disease ) : crohn's and ulcerative colitis , which a disease of large intestine .

3. Behcet disease : it's an autoimmune disease affect the oral mucosa .

## SO , if you see a patient with Aphthous ulcers , most likely he doesn't have underlying disease. But if complains from other things (for ex : diarrhea) we should advise him to see a doctor.

## Aphthous ulcers have some families predisposition.

**🡪Slide #7 :**

**Clinical features :**

* solitary or multiple ulcers.
* shallow, with hyperemic rim,
* covered by a thin white exudate.

**$$ exudates**: it's an edema fluid which is rich in protein , it's related to inflammation.

**$$ trasudate** : it's an edema fluid which is low in protein , it's related to different things , EX; heart disease or pulmonary edema.

* rimmed by a narrow zone of erythema.

**🡪Slide #8 :**

* resolve spontaneously in 7 to 10 days.
* recur.

**🡪Slide #9 :**

**HERPES SIMPLEX VIRAL INFECTION**

* two type : hsv1 and 2 that cause mucosal infection.
* primary infections in children 2-4 years , then become latent until the secondary infection present in the adult (it's recur )
* primary infections are usually asymptomatic, that’s why we don’t see it in children.
* 10- 20 % of cases it can reside as ulceration, this ulceration look like vesicles (sth with fluid inside) throughout the oral mucosa .

**🡪Slide #10 :**

It still superficial but it's deeper , and with irregular edges ( unlike Aphthous ulcer which have more regular edges )

**🡪Slide #11 :**

WHY it becomes reactivated ??

1. Immunity decrease
2. Pregnancy
3. Other infection

🖐 there is a list of things so you should refer to the book to read about this topic ☺

**🡪Slide #12 :**

**Herpes simplex site :**

* most common sites: lips, nasal orifices, buccal mucosa, gingiva and hard palate.
* reactivated ulcers occur at site of primary infection or adjacent mucosa innervated by the same nerve ganglion (walks around the nerve)

**🡪Slide #13 :**

**Herpes simplex clinical features :**

* appear as a group of small vesicles, each vesicles about (1 – 3) mm

**🡪Slide #14 :**

**Herpes simplex management :**

* Resolve in 7 – 10 days ( like Aphthous ulcer ) BUT in the immunocompromised people we give them antiviral drugs
* We give antiviral drugs **only** to the immunocompromised people ; because fear superimposed bacterial infection .
* Antiviral drugs either locally or orally

**🡪Slide #15 :**

In histopathology it's appear like nuclei fused together to form giant cells contain inclusions ( intranuclear inclusions )

**🡪Slide #16 :**

***ORAL CANDIDIASIS (THRUSH***)

* the most common ***fungal*** infection in the oral mucosa.
* candida albicans is a normal component of oral flora.
* candida albicans the most common strain of candida that cause this infection

**🡪Slide #17 :**

You can see a white membrane on the tongue , which you can scrape (so it’s called pseudomembrane) and you can see under it **"erythema"** because there is inflammation with that infection.

**🡪Slide #18 :**

**Causes of candida infection :**

1. certain strains on candida albicans
2. immunocompromised ( most common )
3. after antibacterial treatment which affect the flora.

**🡪Slide #19 :**

* **three clinical forms:**

1. pseudomembranous.( most common )
2. erythematous.(red without white thrush )
3. hyperplastic.( of squamous cell which recognized under the microscope by increase the number of layers )

**🡪Slide #20 :**

CANDIDA : PSEUDOMEMBRANOUS

* most common.
* thrush.( another name to pseudomembranous form)
* superficial , gray to white inflammatory membrane on tongue.
* composed of matted candida surrounded by fibrinosuppurative exudate. ( because it's infection )
* can be scraped off to reveal erythematous base.

**🡪Slide #21 :**

* mildly immunocompromised (after infection , diabetes , pregnancy): remains superficial.
* Severe immunosupression (anticancarous drugs , after transplantation , immuno suppression drugs ): can spread to deep sites.

**🡪Slide #22 :**

**Fibrous proliferative lesions :**

* 1. fibroma.
* 2. pyogenic granuloma**.**

**🡪Slide #23 :**

* submucosal nodular fibrous tissue masses.
* caused by chronic irritation resulting in reactive connective tissue hyperplasia.

**## Fibroma** : it's submucosal proliferative of fibrous tissue forming a small mass . it's not neoplastic it's just proliferative lesion caused by irritation

**🡪Slide #24 :**

* most common site: buccal mucosa , specially people who always biting their cheeks under stress conditions.
* this is an example about proliferative but **NON** neoplastic lesion.
* treatment: complete surgical excision, and removal of the source of irritation, or else it will come again as a new lesion (not recur)

**🡪Slide #25 :**

* **PYOGENIC GRANULOMA** : it's pedunculated masses in the gingival , they occur in children, young adults and pregnant women.

**🡪Slide #26 :**

* It's might be misdiagnosed as malignancy especially if it's rapidly growing
* It should be removed by surgical excision

**🡪Slide #27 :**

* Richly vascular 🡪 red in color
* It's haemangioma : proliferative of blood vessels
* Pyogenic granuloma is a miss naming (because “pyogenic” indicates pus formation & “granuloma” indicates a chronic inflammation.. but it’s not a chronic inflammation, nor it produces pus!!) , So " ***nodular proliferative haemangioma "*** is a better name.

**🡪Slide #28 :**

***PYOGENIC GRANULOMA HISTOPATHOLOGY***

* dense proliferation of immature vessels .

**🡪Slide #29 :**

**pyogenic granuloma /outcome and treatment :**

* can regress.
* or mature into deep fibrous masses.
* or develop into an ossifying fibroma.( fibroma contain bone )
* cause of neoplasm is usually **mutation**
* treatment: surgical excision

**🡪Slide #30:**

**LEUKOPLAKIA**

* leuko : WBC
* leukoplakia : it's a flat or slightly raised lesion which is white in colour
* W.H.O definition : white patch that cannot be scraped off and cannot be characterized clinically or pathologically as any other disease.
* it's definition by exclusion of the other causes ( no clear cause even under microscope ).

**🡪Slide #31 :**

White lesion cant be scraped off

**🡪slide #32 :**

* 3% of the population.
* 5 – 25% are premalignant
* may progress to scc.
* It's squamous hyperplasia
* Leukoplakia it's only diagnosid under microscope
* Leukoplakia under the microscope it could be

1. Squamous cell hyperplasia alone (not premalignant )
2. Squamous cell hyperplasia associated with dysplasia (premalignant)
3. Squamous cell hyperplasia associated with squamous cell carcinoma in situ (premalignant)

* As long as we don't have invasion it’s no considered SCC , it's leukoplakia
* Leukoplakia it's clinical term not histological term

**🡪slide #33 :**

* all lesions must be consideres premalignant until proven otherwise, by histology.

**🡪slide #34 :**

* spectrum of histological features.
* hyperkeratosis.
* or dysplasia.
* or carcinoma in situ.

**🡪Slide #35 :**

**ERYTHROPLAKIA**

* red , eroded area.
* flat or slightly depressed.
* less common than leukoplakia but has a higher risk of malignant transformation ( 50 % ).
* It's a patch red in color can't be scraped , can't be characterized Otherwise.
* It's more dangerous , 50% have potential to progress to malignancy

**🡪Slide #36 :**

Causes of leukoplakia and erythroplakia is mutation caused by irritation mainly due to Tobacco smoking .

**🡪Slide # 37 :**

**SQUAMOUS CELL CARCINOMA**

* 95% of oral cancers are scc.(most common tumor in the oral cacity)
* sixth most common neoplasm worldwide.
* long term survival less than 50%.
* diagnosed at late stage.
* It's invasive lesion
* The most common malignant tumor in oral cavity
* We try to discover them before malignant stage (when they are leukoplakia )

**🡪Slide # 38 :**

Deep ulcer with raised “rolled” edges.

**🡪Slide # 39 :**

* Occur and recur as multiple lesions can be present.
* patients surviving 5 years after dx have 35% chance of developing at least one new primary lesion within that interval.
* patients with small tumours have > 50% chance of 5 year survival but many die from second primary tumours
* They have many primary lesion and the lesion will recur, as long as the cause “chronic irritation due to alcohol and smoking” is present.

**🡪Slide # 40 :**

* field cancerization: multiple primary tumours develop independently due to chronic exposure of carcinogens.
* early detection of new premalignant lesions is critical for long term survival.
* Behaviour of Scc in mouth : they invade locally then go to the cervical lymph node then go distant mets (liver , lung , mediastinal lymph nodes )

**🡪Slide #41 :**

SCC PATHOGENESIS :

* two pathways:

1. oral cavity scc arising in chronic alcohol and tobacco users.

these have mutations related to carcinogens in tobacco.

1. scc arising in tonsillar crypts or base of the tongue.

these are related to hpv, mainly hpv16.

* hpv : human papilloma virus

How to lower the risk of HPV associated carcinoma ?

* By HPV vaccine even to 13-15 years old girls not for the oral mucosa but to lower the risk of cervical carcinoma.

**🡪 Slide #42 +#43 + #44 ( same as the slides)**

**🡪Slide #45 :**

It contain keratin

**🡪slide #46 (same as the slide )**

**عجــــبت لمن يقـــــــــيم بدار ذل .... وأرض الله واســـــــــعة فضــــــاها**

**فذاك من الرجــــــال قـــــــــــليل .... عقــــل بلـــيد ليس يعـــــلم من بناها**

**فنفســــك فــز بها إن خفت ضيما .... وخــــل الــــدار ومــــــــن بناهــــــا**

**فـــإنك واجــد أرضـــــــا بأرض .... ونفســــك لم تجــــد نفســــا ســـواها**

**ومن كـــــانت مــــنيته بــــأرض .... فـــليس يمــــوت في أرض ســــواها**

**" الإمام الشافي"**