**Oral pathology
sheet2
done by : Lama abusharaf&Hadeel abu alruz**

**White lesions**

**White lesion: term used clinically to describe the appearance of lesions presenting as white area on oral mucosa.**

**Etiological classification of white lesions:**

**Hereditary**

**Traumatic(reactive white lesions)**

**Infective**

**Idiopathic**

**Dermatological**

**Neoplastic**

**Leukoplakia:**

**Defined as:**

* + **‘‘A white plaque of questionable risk (some of them will turn into SCC) having excluded (other) known diseases or disorders that carry no increased risk for cancer” WHO 2005**

**# a predominantly white lesion of the oral mucosa that can't be characterized as any other definable lesion.**

**# permanent White lesion without known cause.**

**So diagnosis of these lesions by excluding the other cause of white lesions.and we must take a biopsy to look for dysplasia( not to confirm diagnosis):to predict the prognosis
#dysplasia means that this lesion is going to develop into cancer but if we didn't found dysplasia that doesn't mean that this lesion is never going to be cancer ( dysplasia can occur at any time )
#clinical diagnosis**

**Prevalence:** **Less than 1% of people**

**Gender:**

**Male > Femal**

**Usually in elderly pt. or in middle age pt.**

**Site:**

**Affect any site on oral mucosa**

**\*leukoplakias involving the ventral tongue and\or floor of the mouth have high risk of malignant transformation than lesion at other site.**

**Size:**

**Vary from a quite small and circumscribed plaque to an extensive lesion involving a large area of oral mucosa.**

**Color: maybe: # white**

 **# whitish-yellow**

 **# gray**

**Characteristic of leukoplakia:**

**Potentially malignant.**

 **Clinical classification of leukoplakia: there are 2 types of leukoplakia:**

**Homogeneous 2\non-homogeneous 1\**

|  |  |
| --- | --- |
|  **Non-homogeneous**  |  **Homogeneous** |
| **If we see one of these things:****1-not flat surface, irregular nodular. (Thickening surface) some case may take a warty appearance.** **2-variation in color , show areas of redness producing a speckled appearance.(speckled leukoplakia)****3-ulcer.****Non-homogeneous lesion have a worse prognosis****50% of cases have dysplasia.** | **1-plaque-like,flat,uniform color****2-predominantly white plaque.****3- may show shallow cracks\fissures on surface.4- no ulcer****Only 10% of cases have dysplasia.** |

**#Non-homogenous is more dangerous than homogenous.**

**#How to differentiate between smoking keratosis and leukoplakia?**by stop smoking,if it disappeared it's smoking keratosis and if not it's leukoplakia

**Erythroplakia:**

**Is a bright-red patch on the oral mucosa which can't be categorized clinically or pathologically as being due to any other condition.**

**Erythroplakia lesion may be homogeneous with well-defined outline. Or . may be intermingled with patch of leukoplakia which called speckled leukoplakias or erythroleukoplakia.**

 **Histologically Erythroplakia may represent carcinoma in situ or even invasive carcinoma.**

**Etiology: is Unknown similar to leukoplakia**

 **The Incriminated factors for leukoplakia are:**

 **# Tobacco**

**# Alcohol**

**# candida**

**#viruses: HPV 16 + 18**

**# Epithelial atrophy**

**In atrophic area there are High chance to develop leukoplakia and oral cancer.**

**Causes of atrophy in oral mucosa:**

**1\Iron deficiency as : Sideropenic dysphagia, Patterson Kelly, plummer-vinson syndrome.**

**2\** **Vit. deficiency (vit. A&B).**

**3\submucous fibrosis.. especially in chewing habit area.(Asia)**

**4\tertiary syphilis.**

**Histopathological features of leukoplakia:**

**No specific histological feature..there is a wide range in histological appearances:**

**\* Ortho or parakeratosis or mixture in the same area. Hyperplasia of epithelium. \***

**In case of speckled there is an atrophy.\***

**\*Chronic cell infiltrate in lamina properia.**

**But the most imp. Feature for pathologist is presence of Dysplasia(no control on proliferation or differentiation of epithelium due to mutations) or not. because leukoplakia has a potential change to cancer we should take a biopsy to decide if there is a dysplasia or not.**

**Leukoplakias should be followed-up and managed because these lesions may change into cancer.
#cellular changes:
1-pleomorphism( variation of size of cells)
2- hyperchromatism
3-abnormal cellular polarity( basal cells aren't confind to the basal layer)
4- drop shape rete ridges
5- deep cell keratinization
6- increase in nuclear/cytoplasmic ratio
7- abnormal mitosis
8- abnormal cellualar maturation(no distinguished layers) and loss of cellular connectors(desmosomes)**

**Pathologist classification of dysplasia according to its severity:**

**1- Basal cell hyperplasia.**

**2- Mild🡪dysplasia in lower 1\3 of thickness of epith.**

**3- Moderate🡪dysplasia up to 1\2 of thickness epith.**

**4- Severe🡪dysplasia more than 2/3**

**5- Carcinoma in situ🡪full thickness of epith.**

**An** **increased in severity of dysplasia increase the risk of oral cancer.**

**Prognosis:**

**-unpredictable**

**- (0.3-18%) of cases could change to cancer.**

**Luekoplakia**

**-unpredictable**

**-(0.3-18%) of case could be change to cancer.**

**What determines if this lesion will transform into cancer is the presence of dysplasia ( main factor) , as dysplasia is sever there is a strong possibility of this lesion to become a cancer.**

**Other factors can help me to predict the like hood of transformation into cancer is the family history, : if the pt. have family history for oral cancer this will increase the susceptiblitiy in changing the leukoplakia to cancer.**

 **also if leukoplakia is not homogenous ,pickled, ulcerated , then the prognosis will be worse, because dysplasia here is higher**

**Enlargement or change in the character: means that one has white lesion its size is 1cm, after a while he comes back and it becomes larger, so we predict that the prognosis will be worse than other whose legion size didn’t change, same will be applied if he had white legion then its color becomes red or ulcerated.**

**Also the site help us to predict :if leukoplakia was in the floor of the mouth and ventral surface of the tongue, then the prognosis will be worse ,and its probability to transform to SCC is higher, because these areas are protected under the tongue and its unusual to have lesion there. whereas if I find leukoplakia on the lateral border of the tongue or buccal mucosa, its expected because these areas are exposed to irritation.**

**Also duration can help:**

**As the duration of having the lesion increase its probability to transform into SCC is higher.**

**Size : as the lesion size > 2cm2 , prognosis will be worse ( as size increase , it will be worse)**

**\*\*The Risk of developing malignancy at lesion site is 5 times greater in pt. with leukoplakia than with normal person.**

**Homogenous or not : if it is homogenous then its probability is 10%, if its not then dysplasia probability is 50% , if it was erythroplakia or atrophied ( at all areas of it) then its 98% will have sever dysplasia, or carcinoma , so u need biopsy here , and u should ask the patient about the history and etiology of this legion , if he didn’t give u a real reason( as candida infection ,trauma…) , u should think of dysplasia and SCC , u shouldn’t delay this.**

**There is one problem about biopsy ; from where to take it ? if there is ulcer take it from area next to the ulcer, or if there is a change in thickness u should take it from there.**

**Or some people use toluidine blue, that mark the most dysplastic area( highest in proliferation and mitosis) .**

**If there is 100 lesion of leukoplakia , take from them biopsy..then do excision to all of them and send them to the lab>> you will find that 50% of initial biopsy have dysplasia , but 55% of excisional biopsy have dysplasia.. so by biopsy only sometimes u will miss dysplastic areas by difference in comparison to excisional biopsy of 5%.**

**That’s why if it was large biopsy we take more than one biopsy.**

 **Dysplasia:- In \* Homogenous leukoplakia 10%**

**Non-homogenous 50%**

**Erythroplakia 80-90%**

**either sever or carcinoma in situ or cancer (mild invasive)**

**Other factors other than the histopathology can help me to predict the probability of transformation into cancer..( reliable biomarker, or stain by immunohistochemistry?0**

**Read about this ..**

**Leukoplakia staging :**

**Oral classification system , that depend on the size and dysplasia**

**Size :**

**: L1 (less than 2cm)**

 **L2 ( 2-4 cm)**

 **L3 ( more than 4 cm)**

**Dysplasia :**

 **D0: no dysplasia**

 **D1: mild to moderate dysplasia**

 **D2: severe dysplasia**

**Same as sizing system of SCC**

**Dysplasia :**

**D0 >> no dysplasia**

**D1 >> mild to moderate**

**D2 >> sever or carcinoma in situ**

**So mild and moderate , they gave them same prognosis**

**Stage 1 >> L1 D0**

**Stage 2 >> L2 D0**

**Stage 3 if the size: or small size and D1**

**Stage4: L3D1 or any size with D2**

**When stage increases in #, the risk of dysplasia to progress to cancer increases.**

**Other white legions that are more common than leukoplakia is**

 **:lichen planus**

**most frequent site is Buccal mucosa.**

**-can affect other site as tongue, gingiva, lip ,vestibule.**

**- least frequent (rarely seen) on the floor of the mouth & palate.**

**Distribution of L.P is bilaterally and symmetrical this will help in diagnosis.**

**Its easy to diagnose, common, chronic inflammatory disease of skin and mucous membranes, so it’s a skin disease that can exist in the oral cavity**

**the most common disease that we can see in pt. who visit our clinic.**

**-totally benign.**

**chronic inflammatory disorder affecting skin and mucous membranes🡪most diseas affecting mucosa.**

**affecting 1% of population.**

**Some have it only in skin or only in mucosa.**

**But skin lesions always help in diagnosis,**

**-most predisposes females 30-60%**

**affect middle age 30-50 years.**

**40% of the patients have skin and oral**

**35% only skin**

**25% oral lesions only**

**So % of oral lesions are 65%**

**25% of pt. have just oral manifestation without skin lesion..in this case we may have problem in diagnosis coz in lichen planus we depend on presence of skin lesion in diagnosis.**

**Skin legions : check the appeared areas of the hand >> purple pruritic papule**

**pruritic means : itchy**

 **\*Almost any area in sikn can affect but mostly in flexon surface of the wrist.**

**on the surface there is white lines, called wickhams striae**

**Duration of this lesions 1y -1.5y then disappear , duration is 18 months and not permanent**

**Diameter 2-3 mm .. , these people has nails problems as vertical ridges , atrophy, thinning in nails…**

**Don’t be confused ,, this lesion is different from psoriases.. lichen planus are papules , psoriases is white legions..**

**In oral cavity : the most common site is the buccal mucosa, cuz it is movable**

**\*Nail are involved in up to 10% of pt.they have atrophy and vertical ridges.**

**.**

**Also we can find it on lateral border of the tongue ,gingival also are common, least frequent**

**Is the floor of the mouth and the palate ..**

**Its distribution ; bilateral and symmetrical >> help in diagnosis**

**Appearance is characteristic : white lines criss-crossing , reticular ,roughness on buccal mucosa..**

**The most common type of lichen planus is the reticular type.**

**1-Reticular type:**

**-the most common type.**

**-Asymptomatic**

**-Site :appears on buccal mucosa bilateral🡪look like white spongy naevus.**

**-has wickham'sstriae🡪 has lace-like striae**

**-it seems like leukoedema, so we have to do stretch test for diagnosis.**

**-less frequent.**

**-Asymptomatic.**

**-white plaques resembling leukoplakia.**

**-Site: appear on buccal mucosa and on tongue or other sites.**

**3-Papular type:**

**-rare not common**

**-Asymptomatic.**

**-small white papules that may coalesce give plaque-lesion.**

**4-Atrophy type:**

**-more frequent than popularand plaque-like and the 2nd most type after reticular.**

**-atrophy🡪red in color look like Erythroplakia..so to differentiate between them we look for wickham'sstriae.**

**-Symptomatic… it cause pain because atrophy means loss of epi. That protects nerves, without epi. The tissue becomes so Sensitive.**

**-most common Siteis Gingiva..it look like gingivitis.**

**\*\*How to differentiate btn atrophy type & gingivitis??**

**Gingivitis: is the inflammation of marginal gingiva.**

**Atrophy type: redness is on the whole thickness of gingiva (marginal gingiva & attached gingiva) and this called desquamative gingivitis.**

**5-Bollous type:**

**-rare to see**

**-up to 2cm , mostly seen in skin not intraorally coz its easy to ruptures and produce an ulcers.**

**-Site: on posterior buccal mucosa.**

**6-Erosive type:**

**-Dangerouse.**

**-most painfull to the pt., difficulty in mastication,bleeding,suffering during swallowing.**

**-extensive areas of shallow irregular areas of epithelial loss (ulceration) 🡪**

**So its not red in color..its yellowish membrane duo to fibriline deposition.**

**-canoccure in any area,itsvery persistant (chronic duration of ulceration)**

**-Diagnosis of erosive type by recognize white area around the lesion**

**Which legion can confuse us with this is leukoedema >> white legion that has light lines but we said we do stretching and it will disappear, but lichen planus is keratosis that will not disappear ..**

**Skin lesion is not permenant they can disappear after one or two years and come back again in another place of the body… this is v.imp. when take history.**

**Other types : plaque :white spots like leukplakiao , asymptomatic, site is on lateral border of the tongue,**

**Both reticular and plaque don’t cause pain , only roughness**

**Third type also asymptomatic ; papular>> small whitish papules**

**Atrophic type ; mainly on gingiva, can be associated with other white lesions. And the gingival called here desquamative gingivitis. Don’t be confused , its not chronic gingivitis cuz the last is only on marginal gingiva,no atrophy , has inflammation, whereas the former is on whole attached gingival , has atrophy and no inflammation ,**

**This epithelium with desquamative gingivitis can cause pain an burning sensation ..**

**Other type is bullous type , which is rare in the oral cavity , common on skin>> vesicle or bullae that contain fluid then it will rupture**

**The most dangerous type is irrosive: not only atrophy but also superficial ulceration**

**Irrosion appears as yellowish membrane cuz of deposition of fibrin on any ulcer in the oral cavity . around it always there is white lines and that what can help in diagnosis( wickhams striae)**

**Its painful more than the atrophic type because it is ulcerations..so patient cant eat or drink**

**Histopathology can help in diagnosis of reticular type>> but usually we don’t take biopsy from it because its only a clinical diagnosis, but irrosive type we need to take a biopsy,**

**Histological feature of lichen planus:**

**1.Focalacanthosis—the epi. Is ortho or parakeratinized.**

**2. sawtooth rete pegs: this appearance results from acanthosis which make irregular elongation and widening of the rete processes.**

**3.Adense, well-defined band of T-lymphocyte in the superficial layer of epi.**

**4. Inflammation extendingto basal and parabasal cell layer.**

**5. liquefactive degeneration of the basal cells: which is the degeneration of the basal cells associated with oedema and lymphocytic infiltration.**

**6.Civatte bodies: the degenerating cells appear as hyaline condensed bodies and represent basal cells under going apoptosis.**

**The etiology of lichen planus:Unknown!!**

**Appears white lines because of acanthuses ( hyperplasia and huperkeratosis), but hyperkeratosis is not characteristic to lichen planus..**

**But it has dense , well defend band of T lymphocytes in the superficial lamina propria, extending to the basal cell layer,**

**Basal area is damaged and has lequifactive regeneration.**

**But May be associated with some condition like:**

**1\ infective agents such as bacteria.**

**2\ systemic diseases: DM, hypertension, ulcerative colitis, liver disease such as hepatitis C and graft-versus-host disease (GVHD).**

**3\ Psychiatric disorders: stress, depression.**

**4\Tobacco.**

**5\Decrease in vitamins.**

**6\ Lichenoid reaction: results from drugs antimalarial,gold,methyldopa,NSAID)and amalgam restoration.**

**The pathogenesis:**

**1-Type IV hyper sensitivity reaction will appear, caused by the well-defined band of T-lymphocyte that will excite the immune cells to come and fight the cells in the region.**

**2-lichenoid reaction is similar to lichen planus clinically.(in Certain patients who take certain drugs (antimalarial, methyldopa and NSAID) lesions will appear similar to lichenplanus.**

**And next to an old amalgam restoration a red lesion with whitish striated border will appear.**

**Histologically we can differentiate btw these lesions and lichen planus but not clinically.**

**3-A certain infective unknown agents come to the epithelium and its similar to the antigen of the keratinocytes, processed by langerhan's cells andpresented activate production of CD8.**

**So the treatment is by suppressing the immune reaction.**