ORAL PATHOLOGY

LECTURE #: 

DOCTOR: Falah Alsawairr
DONE BY: 

CORRECTED BY: Obaida Al-Jarrah

DAY & DATE: Sun, 18/9/2016

PRICE:

ABC Books
مكتبة تلاع العلي
شارع الجامعة الأردنية – جسر كلية الزراعة
عمارة العساف – 235 داخل المجمع

هاتف:
0797121818
06/5336475

CONTACT US: Ljneh Asnan Dental.c2013@gmail.com Dental Correctionn D.correction2013@gmail.com
Etiological classification of white lesions:

Hereditary
Traumatic
Idiopathic
Infective
Dermatological
Neoplastic

We talked about traumatic keratosis, causes:
1. **mechanical**: the most common cause of white lesions.
2. **chemicals** can lead to whitish lesion, common example is aspirin, some patients think that by swallowing the drug the effect of aspirin will take time, since it will go to the stomach then intestine, then to the liver then it will spread to reach the tooth, they think it won’t be effective. So they put the aspirin tablet directly near the tooth in the vestibule. They don’t know that the aspirin is an acid, which leads to necrosis of the tissue which gives the whitish lesion and if you remove this necrotic tissue you will end up with erosion.

* how to make sure it’s necrosis and not hyperkeratosis and hyperplasia?

When you wipe it with gauze, all the whitish lesions will be removed, and in their places there will be erosion and bleeding. So before wiping with gauze you should ask the patient, he could tell you yes I put it aspirin there yesterday for example.

- Management: remove the cause. The patient puts aspirin due to irreversible pulpitis, so you have to do root canal treatment or extraction if tooth not restorable, oral hygiene instructions, and mouth washes.

Other chemicals; some patients have allergy to specific components in toothpaste or mouthwash that leads to irritation then white lesions (Cinnamon). Chemicals in tobacco: tobacco in any form cigarettes, chewing, smokeless (put it in the vestibule) can lead to white lesions (hyperkeratosis and hyperplasia) because of toxic chemicals that lead to irritation of oral mucosa.

3. **Thermal**: acute trauma with heat lead to mucosal necrosis, the cause can be hot food or objects or heat from smoking, so smoking can be chemical and thermal cause. Pipe mainly at the dorsum tongue and palate, while in cigarette smoking affects almost all over the oral cavity. Sometimes you find whitish lesion on the lips if the patient smoke the whole cigarette.
Reverse smoking: they put the Glowing part inside their mouths, which lead to whitish lesions in the palate, and these lesions are high risk to become cancer.

**Nicotinic stomatitis:** heavy smokers or pipe smokers have nicotinic stomatitis. Whitish membrane at the junction between hard and soft palate with red spots and they can be elevated. The whitish lesion through hyperplasia and hyperkeratosis while the red areas are due to inflammation in the minor salivary glands.

Histologically (nicotinic stomatitis): hyperkeratosis, hyperplasia and squamous metaplasia in ducts of minor salivary glands, periductal inflammation, and increase in vascularity, which gives the red color.

* Nicotinic stomatitis as a lesion is not potentially malignant, not like reverse smoking.

But it’s dangerous because this is a sign that this patient is heavy smoker. Heavy smoking increase the risk of cancer, not necessary in the palate, can be in other places like tongue, floor of the mouth, etc.

Now we start with a new topic (start with lecture slides 2)

**Leukoplakia:** (Idiopathic white lesion)

**Defined as:**
- “A white plaque of questionable risk 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.

# White lesion without known cause.

So diagnosis of these lesions by excluding the other cause of white lesions.

**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 two types of leukoplakia:

-Homogeneous   -non-homogeneous

this classification is important as prognostic factor

<table>
<thead>
<tr>
<th>Homogeneous</th>
<th>Non-homogeneous</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-plaque-like, flat, uniform</td>
<td>If we see one of these things:</td>
</tr>
<tr>
<td>2-predominantly white plaque.</td>
<td>1-not flat surface, irregular nodular. (Thickening surface) some case may take a warty appearance.</td>
</tr>
<tr>
<td>3- may show shallow cracks/fissures on surface.</td>
<td>2-variation in color, show areas of redness producing a speckled appearance.</td>
</tr>
</tbody>
</table>

Only 10% of cases have dysplasia.

<table>
<thead>
<tr>
<th>Homogeneous</th>
<th>Non-homogeneous</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>If we see one of these things:</td>
</tr>
<tr>
<td></td>
<td>1-not flat surface, irregular nodular. (Thickening surface) some case may take a warty appearance.</td>
</tr>
</tbody>
</table>

50% of cases have dysplasia.

#Non-homogenous is more dangerous than homogenous.

red areas became red because of atrophy of that cells or thickness reduced by reduced keratinization. So it will become more prone to chemical irritation that affect mitosis cells in basal layer which may lead to uncontrolled proliferation (cancer) of epithelium if mutation occurred. So it has worst prognosis than white areas.

in white areas we expect hyperkeratosis hyperplasia.

case: (slide 8)

lesion at right side of the tongue. There is no trauma or infections, all teeth are okay. so its idiopathic red area (erythroplakia)
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. It hasn’t specific histological features

Etiology: is Unknown similar to leukoplakia
if smoker pt come with red lesion, how we know if the lesion because of smoking or not?
by ask him to stop smoking.

The Incriminated factors for leukoplakia are:
# Tobacco #Alcohol #candida #viruses: HPV16+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.
4)tertiary syphilis.

Histopathological features of leukoplakia:

No specific histological feature..there is a wide range in histological appearances:
* Ortho or para-keratosis 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 or not, because leukoplakia has a potential change to cancer we should take a biopsy to decide if there is a dysplasia or not.
(dysplasia means abnormal growth and proliferation of epithelial cells. It may be mild, moderate or severe depending on the affected thickness of epithelium. Mild has better prognosis)
Leukoplakias should be followed-up and managed because these lesions may change into cancer.

Pathologist classification of dysplasia according to its severity:
1- Basal cell hyperplasia.
2- Mild dysplasia in lower1/3 of thickness of epith.
3- Moderate dysplasia up to 1/2 of thickness epith.
4- Severe dysplasia more than 1/2
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.

Prognostic factors of leukoplakia: (risk factors)
*Family history: if the pt. has family history for oral cancer this will increase the susceptibility in changing the leukoplakia to cancer.
*Non-homogenous leukoplakia has higher chance to change to cancer.

If the pt. have homogenous lesion that changes with time to non-homogenous (speckle or modularity or ulcer or redness) he will have higher tendency to change into cancer.
*Size: increasing in size more than 2cm more susceptible to cancer.
*Duration: increase the duration increase the chance to change to cancer
*Site: leukoplakia in the floor of the mouth and ventral surface of the tongue (called sublingual keratosis) has more susceptibility to change to cancer.

BUT the most important prognostic factor is presence of DYSPLASIA (exam Q)

** Studies have revealed Ca or severe dysplasia in the excision specimens of approximately 5% of excised leukoplakias when the diagnostic biopsy specimens had revealed no dysplasia. SO the Risk of developing malignancy at lesion site is 5 times greater in pt. with leukoplakia than with normal person.
Dysplasia:
- Homogenous leukoplakia 10%
- Non-homogenous 50%
- Erythroplakia 80-90% either severe dysplasia or carcinoma in situ
or cancer (mild invasive)

But the diagnosis of dysplasia and estimating its degree is subjective with significant
intra- and inter-observer variability
we have to have criteria to know from where exactly should I take biopsy especially in
extensive cases. Or we have to take more than one biopsy.

Is there any consistently reliable biomarker used to further identify those dysplasias
that are more likely to progress to invasive cancer?

Classification and Staging:
This staging is used to predict the transformation risk.

1- Size: L1 (less than 2cm)
   L2 (2-4 cm)
   L3 (more than 4 cm)
We use the size to predict the prognosis.

2- Presence of dysplasia (P):
   P0: no dysplasia
   P1: mild to moderate dysplasia
   P2: severe dysplasia

Stages:
Stage1: L1P0
Stage2: L2P0
Stage3: L3P0 or L1L2P1
Stage4: L3P1, any L P2
When stage increases in #, the risk to progress to cancer increases.

corrected by: Obaida Al-Jarrah