Oral Pathology

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Learning outcomes, Recorded lectures, lecture notes, lecture PowerPoint slides, past papers, forums, Recommended readings:

http://elearning.ju.edu.jo

Moodle

USERNAME

PASSWORD
White Lesions
Oral epithelium

Basement membrane

Lamina propria

'Sub-mucosa' contains blood vessels and nerves

Muscle or bone
Differential diagnosis:

<table>
<thead>
<tr>
<th>Cause</th>
<th>Lesion</th>
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</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Leukoedema</td>
</tr>
<tr>
<td>Hereditary</td>
<td>White sponge naevus</td>
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<tr>
<td></td>
<td>Oral manifestation of genodermatoses</td>
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<tr>
<td>Traumatic</td>
<td>Mechanical (e.g. frictional keratosis)</td>
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<td></td>
<td>Chemical (e.g. aspirin burn)</td>
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<tr>
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<td>Thermal (e.g. smoker’s keratosis)</td>
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<tr>
<td>Infective</td>
<td>Candidosis</td>
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<td></td>
<td>Hairy leukoplakia</td>
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<td></td>
<td>Syphilitic leukoplakia</td>
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<tr>
<td>Dermatological</td>
<td>Lichen planus</td>
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<tr>
<td></td>
<td>Lupus erythematosus</td>
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<tr>
<td>Idiopathic</td>
<td>Leukoplakia</td>
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<tr>
<td>Neoplastic</td>
<td>Squamous cell carcinoma</td>
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</tbody>
</table>
Leukoedema:
- Variation of normal
- ↔ Racial pigmentation
- **Site:** BM bilaterally, ↓ LB of tongue

**Clin:**

- Asymptomatic
- Diffuse, translucent, grayish-white, folded lesion
“Stretch test."
Hist:

- Mild parak & acanthosis
- Intracellular edema
- Normal LP
Relationship with smoking
Hereditary Lesions
**White Sponge Nevus: (Oral Epithelial Nevus)**

- **AD**

- **Mutation in genes of keratin 4 & 13.**

- **Clinically:**
  - Asymptomatic
  - Whitish, soft
  - Irregularly thickened
  - Usually bilateral (all OM)
- Border
- Other sites
**Hist:**

- Acanthosis
- Moderate-marked hyperparakeratosis
- Marked intracellular oedema (prickle & parakeratin)
  - × Dysplasia
  - × Inflammation in LP
Pachyonychia Congenita:

- AD
- Extreme thickening of nails (≈ birth)
- Palmoplantar hyperkeratosis & hyperhidrosis
- White patches on D or LBT or BM
- Hist: ≈ WSN
- **Dyskeratosis Congenita**: عسر التقرن الخلقي
  - ? Mode of inheritance
  - Pigmentation of skin
  - Dystrophic nails
  - Destructive periodontitis
  - Hyperkeratosis of oral & other MMS
  - Premalignant
Tylosis: ثفن

- AD
- Palmoplantar hyperkeratosis
- Esophageal Ca in later life
- ± Oral hyperkeratosis
Hereditary Benign Intraepithelial Dyskeratosis:

- AD (North Carolina)
- Conjunctival plaques \(\rightarrow\) blindness
- Oral white folds and plaques

**Hist:**

- Acanthosis &
- premature keratinization
Follicular Keratosis (Darier’s disease):

- AD & sporadic
- Face, trunk, ears & scalp: ↑ k papules (coalesce & infected)
- Orally: Small whitish papules on K mucosa

Hist:

- Hyperk
- Suprabasal clefts
- Dyskeratotic cells
Traumatic Keratosis
A) Mechanical:

- Frictional Keratosis
- Prolonged mild abrasion
- Tooth, restoration, denture
- Biting

- Clin:
  - Dense white patch
  - Rough surface
  - Cheek biting
How to differentiate cheek biting keratosis from white sponge nevus

Traumatic Keratosis and Gender
Dx:
- Cause
- Size & shape
- Resolves when cause removed

Hist:
- Hyperplasia & Hyperk
- × Dysplasia
- Scattered CICI in LP
B) Chemical:

- Aspirin burn
- Cinnamon
**Tobacco:** either form

Hyperkeratosis & hyperplasia
C) Thermal:

- Smoking ⇒ white (BM, Tong & palate)
- Pipe: tongue/palate
Cigarette: lip

Reverse smoking
Nicotinic Stomatitis:

- Long-term pipe smokers
- **Clin: Palate**
  - White w multiple, small, round papules w red centers
Hist:

- Dilated ducts with squamous metaplasia & periductal CICI
  - Hyperkeratosis & hyperplasia
Relation with oral cancer
Dermatological white lesions
Lichen Planus: الحزاز المنبسط

- CID of skin & mms affecting ≈ 1%
- 30-60 years of age, 60% F
- 40% skin & oral; 35% skin; 25% oral
Skin:

- **Purplish, pruritic papules**

- Wickham’s Striae

- Nails

- **2-3 m in Φ**

- Duration
Oral lesions:
- Most frequent site
- Other sites
- Least frequent
- Distribution
Types:

- **Reticular:**
  - Lacework, Wickham's Striae
  - Asymptomatic
  - Site
**Plaque-like**

- Approximated Leukoplakia
- Asymptomatic
- Site

**Papular**

- Small white papules
- May coalesce, asymptomatic
Atrophic

- ≈ Erythroplakia often with striae
- Gingiva
- Desquamative gingivitis
- Symptomatic

Bullous

- Up to 2 cm
- Brief
- Posterior BM
Erosive

- Shallow *irregular* areas of epithelial loss
- Smooth, raised *yellowish membrane*

- Can be very persistent
- Striae
- Symptomatic
**Hist:**

- Focal acanthosis w hyperparak/orthokeratosis
- A dense, w-d band of T-lymph in superficial LP
- Involvement of the basal and parabasal cell layers by inflammation
- Sawtooth rete pegs
Civatte bodies

Normal epithelium

Liquefactive degeneration

Civatte bodies
Does Lichen Planus have any malignant potential?
**Etiology:**

- Infective agents
- **Systemic disease:** DM, Hypertn, U colitis, liver disease & GVHD
- Psychiatric disorders

**Pathogenesis:** ≈ type IV hypersensitivity

- ?Ag ≈ Ags on kerationocytes
- Processed by Langerhan’s cells & presented
- Activate production of CD8
- **Lichenoid reaction:**

<table>
<thead>
<tr>
<th>Factor</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dental restorative materials</td>
<td>Amalgam</td>
</tr>
<tr>
<td></td>
<td>Gold</td>
</tr>
<tr>
<td></td>
<td>Polymerized plastics</td>
</tr>
<tr>
<td>Medications</td>
<td>Anti-malarials</td>
</tr>
<tr>
<td></td>
<td>Oral hypoglycaemics</td>
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<td></td>
<td>NSAIDs</td>
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<tr>
<td>Food and food additives</td>
<td>Flavourings: cinnamon and derivatives</td>
</tr>
</tbody>
</table>
Are there any clinical or histological differences between Lichen planus and Lichenoid reactions?

Does Patch testing useful in identifying patients who will have Lichenoid reactions?
Lupus Erythromatosus: الذئبة الحمراء

- C.T disease with two main forms:

1. Chronic discoid LE:
   - Face, scalp & ears
   - Scaly red patches
   - ± Butterfly pattern
- **Oral lesions**
  - In $\approx 50\%$
  - BM, vermilion border
  - Discoid erythema w white keratotic border $\pm$ radiating striae
- **Hist:**
  - Ortho/parak epith
  - Hyperplasia/atrophy
  - ± Liquefactive degeneration
  - Subepith & deep perivascular lymphocytes
  - **DIF:** linear deposits (IGg, C3 & fibrinogen) in BM.
2. Systemic LE:

- More common
- Fatigue, malaise, fever, psychosis, lymphadenopathy
- Kidney
- Arthritis, heart & lung, anemia, vasculitis, rash
- Oral lesions in \( \approx 20\% \), more severe erythematous patches/BM
Idiopathic white lesions
Leukoplakia:

- **Deftn:** “A white plaque of questionable risk having excluded (other) known diseases or disorders that carry no increased risk for cancer”
  WHO 2005

- **Epidemiology:**
  - < 1%
  - M>F
  - Older
  - Site
- Clinically:
  - Size
  - Colour
  - Homogenous: plaque-like ± surface variations
Non-homogenous

Ulcerated

Speckled
Verrucous

Nodular
Erythroplakia:

A bright-red patch on the OM which cannot be characterized clinically or pathologically as being due to any other condition.
- **Etiology**: Unknown, incriminated factors
  - Tobacco
  - Alcohol
  - Candida
  - Viruses: HPV 16
Epithelial atrophy:

- Iron ↓ (Patterson-Kelly, Plummer-Vinson Syndrome)
- Vit ↓: A & B
- **Hist:** no specific histological features
  - Hyper-orthk or hyper-parak or both
  - Hyperplasia or atrophy
  - CICI in LP
Dysplasia:

- Nuclear & cellular pleomorphism
- Nuclear hyperchromatism
- Disturbed polarity of basal cells
- Drop-shaped rete pegs
- Deep cell keratinization
- ↑ N/C ratio
- ↑ & abnormal mitosis
- Basal cell hyperplasia
- Disturbed maturation
- Loss of intercellular adherence
- **Dysplasia:**
  - Basal cell hyperplasia
  - Mild
  - Moderate
  - Severe dysplasia or
  - Low-grade
  - High grade
- **Prognosis:**
  - Unpredictable (0.3-18%) over prolonged periods.

- **Risk factors:**
  - Family history
  - Female, particularly non-smoker
  - Non-homogenous
  - Enlargement or Δ in character
  - Site: FOM, Vtongue, Retromolar
  - Extensive or spreading lesions

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The risk of developing malignancies at lesion sites is 5 times greater in patients with leukoplakia than in patients without leukoplakia.
Dysplasia

Homogenous Leukoplakia: 10%  Non-homogenous leukoplakia: 50%

Erythroplakia: 80-90% are either severe dysplasia or Ca
Ca or severe dysplasia in **excision** specimens of 5% of excised leukoplakias when the **diagnostic biopsy specimens had no dysplasia.**
Is there any consistently reliable biomarker used to further identify those dysplasias that are more likely to progress to invasive cancer?
L (size of the leukoplakia)
- L1: <2 cm
- L2: 2–4 cm
- L3: >4 cm

P (pathology: dysplasia)
- P0: No
- P1: Mild or moderate
- P2: Severe

OL-staging system
- Stage I: L1P0
- Stage II: L2P0
- Stage III: L3P0 or L1L2P1
- Stage IV: L3P1, any L P2