Lecture # : 3

Doctor : Faleh Sawair

Done by : Name

Corrected by : Dania tafesh

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Price :
Dermatological white lesion

Its skin disease & this disease has oral manifestation as white lesion.

1) Lichen planus: (easier in Dx.)
   - A common disease that we can see in patients who visit our clinics.
   - Chronic inflammatory disorder affecting skin and mucous membranes.
   - Affecting 1% of population.
   - Mostly females (60%), 30-60 y
   - Affect middle age 30-50 years.
   - 40% of pt. have skin + oral lesion both areas are affected.
   - 35% of pt. have just skin lesion without oral manifestation.
   - 25% of pt. have just oral manifestation without a skin lesion. In this case we may have problem in diagnosis because the presence of skin lesion helps in diagnosis.

So........

How many patients have oral lesions? 65%
how many patients have skin lesions? 75%

→ Feature of skin lesion:

* Purplish, pruritic (itchy), papules (2-3mm) in diameter, has white striation on its surface called Wickham's striae. These striae help in Dx.

* Almost any area in skin can be affected but mostly in flexor surfaces of the wrist.

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

* Skin lesion is not permanent they can disappear after one or two years and come back again in another place of the body... this is very important in history taking.
**Note:** we have to distinguish between lichen planus and Psoriasis (الصدفية) which is a red or white lesion on skin and it doesn’t have a Wickham's striae also it is more profused.

**Oral lichen planus:**

- most frequent site is buccal mucosa and the patient feels roughness in the surface.
- can affect other sites as tongue, gingiva, lip, and vestibule.
- Least frequent (rarely seen) on the floor of the mouth & palate.
- Distribution of lichen planus is bilateral and symmetrical so this will help in the diagnosis.

**Types of lichen planus:**

1- **Reticular type**
   - The most common type.
   - Asymptomatic.
   - Site: appears on the buccal mucosa bilaterally → look like white spongy nevus.
   - has wickham's striae → has lace-like striae.
   - It seems like leukoedema, so we have to do stretch test for diagnosis.

2- **Plaque-like:**
   - Less frequent.
   - Asymptomatic.
   - White plaques resembling leukoplakia.
   - Site: appear on the buccal mucosa and on tongue or other sites.

3- **Papular type:**
   - rare and not common.
   - Asymptomatic.
   - Small white papules that may coalesce giving plaque-lesion.
4-Atrophic type:

- More frequent than papular and plaque-like and the 2nd most common type after reticular.
- Atrophy → red in color look like Erythroplakia.. So to differentiate between them we look for Wickham’s striae.
- Symptomatic... it causes pain because atrophy means loss of epithelium that protects nerves, without epithelium the tissue become more sensitive.
- Most common Site is the Gingiva.. It looks like gingivitis.

**How to differentiate between atrophic type & gingivitis??

- **Gingivitis:** is the inflammation of marginal gingiva.
- **Atrophy type:** redness is on the whole thickness of the gingiva (marginal gingiva & attached gingiva) and this called desquamative gingivitis.
- On of the causes of desquamous gingivitis is lichen planus.

5-Bollous type:

- rare to see
- Up to 2cm, mostly seen in the skin and not intraorally because its easy to ruptures and produce an ulcers.
- Site: on posterior buccal mucosa.

6-Erosive type:

- Most Dangerous.
- Most painful to the pt., difficulty in mastication, bleeding, suffering during swallowing.
- extensive areas of shallow irregular areas of epithelial loss (ulceration) → So it’s not red in color. It’s a yellowish membrane due to fibrin deposition.
- can occur in any area, its very persistent (chronic duration of ulceration)
- Diagnosis of erosive type happen by recognizing whitish areas around the lesion
• **Histological feature of lichen planus:**

1. **Focal acanthosis**—the epithelium is ortho or parakeratinized.
2. **Saw-tooth rete pegs** أنسان المنشار: this appearance results from acanthosis which make irregular elongation and widening of the rete processes.
3. **A dense, well-defined band of T-lymphocyte** in superficial lamina propria and it is attacking the epithelium.
4. **Inflammation** extending to the basal and parabasal cell layer.
5. **Liquefactive degeneration of the basal cells:** which is the degeneration of the basal cells associated with edema and lymphocytic infiltration.
6. **Civatte bodies:** the degenerating epithelial cells appear as hyaline condensed bodies and represent basal cells undergoing apoptosis.

???

Does Lichen Planus have any malignant potential????

OLP is regarded by several authors as a pre-malignant condition, and patients with OLP have been recommended to have their lesions monitored two to four times annually especially the erosive type.

• **Etiology:**

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

• **The pathogenesis:**

1- Type IV hyper sensitivity reaction, changes of the antigens on keratinocytes will attract T-lymphocyte that will come and damage the basal epithelial cells.
2- A certain infective unknown agents come to the epithelium and the are similar to the antigens of the keratinocytes, processed by Langerhans cells and presented to activate production of CD8.

So the treatment is by suppressing the immune reaction.
2) **Lichenoid reaction:**

- Lesions resembling lichen planus and results from drugs: antimalarial, gold, methyldopa, NSAID) or old amalgam restorations.

Are there any clinical or histological differences between Lichen planus and Lichenoid reactions?

The authors conclude that involvement of the lips was the only clinically significant difference between these two diseases and that people with OLR often have involvement of the lips. Infiltration, atrophic epithelium, “saw-tooth” rete pegs and Max Joseph area were histopathologically reliable criteria for distinguishing OLP, while deep infiltration of the connective tissue and hyperparakeratosis were the criteria for the diagnosis of OLR. OLR consists of more eosinophils, plasma cells and granulocytes in comparison to OLP lesions.

Patients with OLR who have amalgam fillings near lesions should have them replaced, i.e. when possible they should be referred to patch test, as well as when drug-induced OLR are suspected.

It seems that OLR are more prone to malignant alteration in comparison to OLP. Furthermore, it seems that histopathology reveals clear differences between OLP and OLR.

Does Patch testing useful in identifying patients who will have Lichenoid reactions?

Skin patch testing is a valuable tool to confirm clinically suspected oral lichenoid reactions. Pathology diagnoses of oral lichenoid reactions did not correlate with patch test results.

3) **Lupus erythromatosus:**

Two main forms of this disease are recognized:

1. Chronic discoid lupus erythematosus.
2. Systemic lupus erythematosus.

1. **Chronic discoid lupus erythematosus:**

- This is (Localized) disease.
- It's restricted to the skin of the face, scalp & ears
- Present as Scaly red patches and butterfly pattern (lesion has symmetrical distribution over the nose & cheeks).
- Oral lesions are present in 50% of the cases.
• Can affect any part of the oral mucosa (cheeks, vermilion zone), there will be a discoid area of erythema surrounded by a white keratotic border with radiating striae like sun.

**The histological appearance:**

1) Ortho- or parakeratinized.

2) Hyperplasia or atrophy.

3) Keratin plugging.

4) subepithelium and *deep* perivascular lymphocytes (this is the difference between chronic discoid and lichen planus).

5) liquefactive degeneration in the basal cell layer.

6) Direct immunofluorescence test will be +ve for IgG, C3 and fibrinogen (coz they will be deposit as a granular linear in the basement membrane).

**2. Systemic lupus erythematosus:**

*which is disseminated disease involving almost every organ of the body.

*the lesions in sys.LE. Include skin rashes that occur on cheeks.

*Oral lesion occurs in 20% of the cases described as superficial erosions & erythematous patches on the buccal mucosa.

* There is systemic symptoms: fatigue, malaise, fever psychosis and lymphadenopathy.
Heart Oral Infections

Viral infection

- a case present with multiple ulcers and the gingiva is inflamed. So we think about viral infection.

A. HERPES SIMPLEX VIRUS

- Herpes simplex virus type 1 and type 2.
  - Type 1 that mostly affects the oral cavity (oral herpes sores around the mouth and lips) above the waist. Type 2 that cause genital herpes mostly "below the waist".
  - HSV is lytic to epithelial cell, once it is attached to epithelial cells it causes lyses and becomes latent in neural tissue which is the most important characteristic of HSV's (neuroinvasive virus).
  - So, as we said that HSV-1 and HSV-2 persist in the body by becoming latent and hiding from the immune system in the cell bodies of neurons. Once they infect primarily, they don't go but they become latent in neural tissue and what’s important to us is the trigeminal ganglion.

- More than 90% of adults have Ab's against HSV's, so it's very common infection, every one of us have been exposed to HSV. However, it doesn't mean that everyone had the clinical manifestation.
- Once you get infection with the saliva containing the virus, the virus will enter into breaks in the skin or can penetrate intact mucus membrane like the oral cavity.
  So, if there are cuts in the skin, virus can enter. But in oral cavity it can easily penetrate the intact mucus membrane.
• HSV might cause damage in the epithelial cell leading to primary infection and later on the virus will be transported through the peripheral nerves to the ganglion. In the ganglion it becomes latent away from the Ab's and body immunity.

• Once the chance of another infection comes again and re-attacks the body causing 2ry infection, and it will travel again through the peripheral nerves to the epithelium, oral cavity, and causing 2ry infection.

• The 2ry infection usually appears in 1/3 of the patient that are affected by 1ry infection (2ry recurrent infection).

• The 1ry infection is mostly subclinical which means that the patient has the virus but he doesn't know that he has been infected; no signs or symptoms develop or sometimes non-specific signs and symptoms, like: pharyngitis, but he doesn't know that he has primary infection of HSV.

?????How to know that the pt had a sub-clinical infection in the past????

• About 13% of children have had symptomatic 1ry infection by age 9.

• So, primary infection is usually subclinical but sometime it can have clinical manifestation.

• The age to be affected by the virus is the first 5 years of age. The 1ry infection, as we said, mostly in young children that might be exposed to infected saliva. Followed by incubational period of 5 days (5 days or 1 week without symptoms). Then there are prodromal symptoms – non-specific to herpes- fever, malaise. Followed by appearance of numerous small (2-3mm in diameter) vesicles on the keratinized and non- keratinized areas of oral mucosa. So, it can affect any part or area in the oral cavity.

• These small vesicles (2-3mm) contain clear fluid, but usually we don't see much of these clinical vesicles because they rapidly rupture and become multiple irregular ulcers.

• Early stage => vesicles
• Late stage => ulcers (rupture of vesicles).

• The ulcers can appear anywhere in the oral cavity but mainly on the dorsum of the tongue. In addition, one of the most important presentations of the primary HSV infection is widespread gingivitis. So once you see these two manifestations you have to suspect primary Herpetic gingivostomatitis mainly in infancy or childhood.
• In children, this infection can spread extra-orally because they can't control saliva. By drooling leading to ulcerations on chin or nose or can spread to the eyes by child’s fingers.
• Other manifestations are inability to swallow, tender cervical lymphadenopathy, dysphagia and drooling in children (saliva coming out the oral cavity). This may persist around 7-10 days then it will heal without any complications.
• However, the viral shedding into saliva (viral presence in saliva) stays at place after 3 weeks of healing and can spread to other children.
• Other important manifestation of primary herpetic gingivostomatitis is presence of *Circumoral crusting* because of the ulcerations on the vermilion zone that bleed and coagulate forming crusts.
• In adults, the primary infection usually present as *pharyngeotonsillitis* (inflammation in the posterior part of the cavity). In addition to the non-specific features; malaise, fever and dysphagia.

Soooooooollllll…..

- In children ☐ try herpetic gingivostomatitis with inflammation of the gingiva.
- In adults ☐ only inflammation of the posterior part of oral cavity; pharyngeotonsillitis.

**How to confirm the diagnosis of herpes?**

Usually **clinical** manifestations but you might take:

1) Tissue culture; scraping of skin vesicles or oral ulcers then sending them through viral transport media to the virology lab and put it on tissue and check for cytopathic changes in the cells and tissue –if they rupture or not- within 24-48 hrs.

2) Serology test to check Ab's against herpes simplex virus.

3) You might take a smear to check changes that happen in the cells. Usually epithelial cells are multi-nucleated with inflammatory cells around. In addition to the epithelial cells with margination of chromatin.

4) Biopsy; to see the histopathological changes. Herpes and other viruses usually cause intra-epithelial vesicles, these vesicles are intra-epithelial that have cleavage within the epithelium, in addition to ballooning degeneration.

Once the cells get infected with the virus, the virus will multiply and at the end there will be rupture of epithelial cells leading to space formation that become a space for accumulation of fluid and vesicles formation. You will notice also inflammatory infiltration in the lamina propria.
**Herpetic whitlow** is a lesion on a finger or thumb caused by the HSV.

It is often contracted by health care workers that come in contrast with the virus; it's most commonly contracted by dental workers and medical workers exposed to oral secretions without wearing gloves.

It's characterized by multiple vesicles or ulcerations on fingers associated with severe pain, fever and lymphadenopathy (systemic symptoms). So we have to take our precautions and wear gloves before any examination even if there were no symptoms or signs of infection.

Questions: **What about the duration of herpetic whitlow? Is it recurrent?**

**Is it possible to have herpetic whitlow in a seropositive Dentist (e.g., one with a history of HSV infection)?**

If he is already exposed to the virus before and he examines pt with HSV in the saliva without using gloves, the virus penetrates and enters the body. **Is it able to cause HWL or not?**

**✓ Recurrent herpetic infection:**

As we said, 1/3 of us will develop recurrent herpetic infection and the virus will travel from the ganglia through the peripheral nerves to the nerve endings causing local symptoms—it doesn’t develop systemic symptoms; no fever, no malaise neither lymphadenopathy. It causes only **local symptoms** such as formation of vesicles and followed by rupture of these vesicles. The patient has the virus in the latent form in the ganglia; the immunity becomes not that strong to be able to prevent the transportation of the virus from ganglia to the lips.

Usually starts as tingling sensation (burning sensation at the site of future infection) followed by vesicles after few hours. Then vesicle will rupture rapidly and left with ulcers. Then these ulcers will heal after around 7-10 days. So the whole process takes approximately 8-10 days or 2 weeks if it gets complicated because of 2ry infection.

**✓** The most common site of herpetic infection in the oral cavity is on the junction between the vermillion zone and skin or close to the nose. In general, it is easy to diagnose—clusters of vesicles—**Herpes Labialis – حمو**. They are called "حمو" because they follow other febrile illnesses or called **حمو– بطأر** because they may follow common cold.

**✓ One of the causes of reactivation of the virus is stress (common in students during exams), trauma, sunlight, febrile illness, menstruation, gastric upset, immunosuppressant or some kind of food (ex. chocolate, nuts).**
Recurrent lesions may develop intra orally not only on the lips, that may follow dental treatment like anesthesia in the palate which may participate in causing herpes recurrent infection. Most common site is the palate. Usually it appears unilaterally at the area of the greater palatine nerve. And It is hard to diagnose the intra oral infection of HSV, not as easy as the herpes labialis. But as a rule, always think about viral infection if you notice multiple irregular ulcers.

The differential diagnosis is herpes zoster, when we notice recurrent intra oral unilateral multiple irregular ulcers. So How to confirm that it is HSV not zoster????

Q: Is it possible to have shedding of HSV particles within the saliva in the absence of manifestation of 1ry or 2ry infection?

What is the Correlation between patient’s age and recurrence rate???

“A GOAL WITHOUT A PLAN IS JUST A WISH”

GOOD LUCK