**18/10/2015 Oral Pathology (2) lec#3**

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**Lupus Erythromatosus:**

 C.T disease

**Two main forms of this disease are recognized:**

**1\Chronic discoid lupus erythematosus.**

**2\ Systemic lupus erythematosus.**

**1\** **Chronic discoid lupus erythematosus:**

 **\*** which is (Localized) disease.

 \* its 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(seen also in lichen planus)

\*can affect any part of the oral mucosa (cheeks, vermillion zone).
\* there will be a discoid area of erythema surrounded by a white keratotic border with radiating striae like sun ( this sun appearance is not seen in all ptns ).

 **The histological appearance:**

1\ Ortho- or parakeratinized.

2\ Hyperplasia or atrophy.

3\ Keratin plugging.

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

5\ liquefactive degeneration in the basal cell layer ( as lichen planus).

6\ Direct immunofluorescence test will be +ve for IgG, C3 and fibrinogen (cuz they will be deposit as a granular linear “well defined bands of igG,c3 which are autoantibodies” in the basement membrane&

-ve test for lichen planus).

**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.

 **Oral infections “ slide2 “**

 **Viral infection**

* **Herpes simplex virus
\***it’s a common recurrent virus.
\*8-types

**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", can affect the oral cavity 10%.

HSV is lytic to epithelium cell, once is attached to epithelial cell it causes lyses and becomes latent to 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 .

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, what we mostly care about is the trigimnial 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, 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 pt that are affected by 1ry infection (2ry recurrent infection).

The 1ry infection is mostly subclinical which means that the pt has the virus but doesn't know that he has been affected; 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 ptn had been exposed to the infection in the past?**we can do a serology and antibodies tests for HSV

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

So, primary infection is usually subclinical but sometime 13% it can have clinical manifestation. This is called :
 **Primary herpetic gingivostomatitis**

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 contain clear fluid, but usually we don't see much of these clinical vesicles on the oral cavity because they are rapidly rupture and become *multiple irregular ulcers*.( u can see vesicles on the skin or the vermillion but not on the oral cavity since they are rapidly ruptured )

Early stage => vesicles

Late stage => ulcers (ruptured 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 of saliva to the chin , nose or eyes leading to ulcerations almost all over the face. This is misdiagnosed as a chickenpox, but keep in mind that chickenpox spreads all over the body not only the face .

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 can appear –inflammation in the posterior part of the cavity. In addition to the non-specific features; malaise, fever and dysphagia.

In children 🡪 1ry herpetic gingivostomatitis with inflammation of gingiva.
In adults 🡪only inflammation of the posterior part of oral cavity; pharyngeotonsillitis.

**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?

How to perform diagnosis of herpes?
Usually clinical manifestations but you might take:

1. Tissue culture; scraping of skin vesicles or oral ulcers then send 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 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 you see 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.

**Recurrent herpetic infection**:

\*Primary infection happens only once in life.

As we said, 1/3 (up to 30%) 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 pt has virus in 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(clear or yellow fluid due to 2ry bacterial infection)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 common site of herpetic infection in the oral cavity is on the junction between the vermilion 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 نفط برد because they may follow common cold.
( you can use a topical antiviral in prodromal phase to reduce it’s severity )

One of the causes of reactivation of the virus is stress (common in students during exams), trauma, dental 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 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 or buccal maxillary gingiva . 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.*

Correlation btw ptn age and recurrence rate ??

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? In other words ; is it possible to have recurrent herpes without vesicles appearance or any clinical manifestation ?