Internal medicine sheet #9
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Oral mucosa is a continuation of the skin, (any type of mucosa share many features with the skin), and so most of skin diseases have oral manifestations.

Oral mucosa is linked to the skin in embryological origin; both are originated from the ectoderm mainly, although some parts of the skin like the appendages originate from the mesoderm, and some parts of the oral mucosa originate from endoderm.

Oral mucosa sharing functions with skin, they both work as protective barrier, has immunological and secretory functions.

Oral manifestations can be the first manifestation of a skin disease, sometimes before months of systemic or skin manifestations, which exhibit the importance of us as dentists to refer the patient to other specialist as soon as possible, to prevent any complication.

The oral examination for a dermatologist is essential; it differs from the oral examination done by a dentist which is more comprehensive.

Oral mucosa and the skin differs in many aspects, such as the color which depends on the vascularity and \*(I can’t hear the word in the record at 6:33)\*, also the admix structures (like hair follicles, apocrine glands, eccrine glands and fordyces) are more abundant in the skin, but salivary glands founds only in the oral mucosa, they also differ in the texture and consistency, the skin is stronger to tolerate many external exposures

The skin has no saliva and always in contact with outer environment, so it must be keratinized as a protective feature.

It has many layers:

* Basal layer
* Spinous layer
* Granular layer
* Corneum layer

Oral mucosa usually isn’t keratinized due to the moist environment around it, and that compatible with its function, which is a little bit different from the skin.

The doctor showed us pictures for the whole layers in oral mucosa and the skin to differentiate between them, there’s a membrane between the dermis and epidermis called the basement membrane (dermal-epidermal junction) , it’s like a glue between them and contain lots of components, such as; laminin ,collagen (7,17), entactin and many others, any problem in these molecules will affect the adhesion between the dermis and epidermis, causes fragility then suppuration, ulceration, blistering and leads to many diseases we will discuss them later.

At the level of keratin, there’s a junction between the keratinocytes (Desmosomes), if it destructed, acanthylosis will happen, this will also cause fragility and leads to a disease called pemphigus.

If there’s more than one layer on top of the basement membrane, then we call it stratified.

There’s many ways to classify skin disorders:

* Pigmented lesions
* White lesions
* Red lesions
* Tumors
* Ulcers
* Blisters
* Affecting the teeth or not

Conditions that affect oral mucosa are divided to congenital and acquired conditions,

* Congenital conditions such as Marfan syndrome, Epidermolysis bullosa, Ectodermal dysplasia
* Acquired like lichen planus and Psoriasis, these disorders have genetic predispositions but they aren’t caused by genetic abnormalities as the congenital conditions, it means if someone got one of these disorders, then his relatives have a bigger chance to have the same disorder.

**LICHEN PLANUS**

It’s a common disease; it’s an immune-mediated disorder but not auto-immune disorder, mainly its affect both the skin and the oral mucosa, it’s very itchy, when you look to the rash, it’s not pink or red but something in between, it’s a little flat topped not elevated as a papule, polygonal not rounded, usually there’s papules and sometimes aggregates together to form one big \*(can’t hear the word at 11:41)\*, we can find Wickham’s striae (lacy pattern) on oral and skin lesions.

Lichen Planus could make Koebnerization.

Koebnerization (Koebner phenomenon): when the condition come out at areas of trauma, like when a person who has lichen planus cut himself accidently, lichen planus may appears at the area of the wound.

 Types of lichen planus:

Mainly **reticular or erosive** (there’s ulceration in this type so the patient can’t eat food and hard to clean his teeth as well), but there’s many types too:

* Bullous
* Atrophic
* Plaque-form
* Popular

Lichen Planus affects around 0.2% of the population, 25% of them have oral and skin manifestations while 30% with only oral manifestations, this type may be asymptomatic, because there’s no skin lesions which are very itchy.

There’s many things could increase the susceptibility to get lichen planus or make it more severe:

* Genetic tendency: between relatives
* Amalgam: leads to contact dermatitis changes in the oral mucosa like lichenoid changes
* Hepatitis C: causes severe lichen planus condition
* Graft-versus-host disease (GVHD)
* Medications: Beta-blockers, NSAIDs, ACE inhibitors and calcium-channels blockers (CCBs), these drugs may cause lichen planus or makes it worsening, all of these drugs are very common between patients so you can’t just tell the patient to stop using them, so we go with controlling the symptoms unless we can specify the drug that initiate lichen planus in the patient.

Lichen planus affects the buccal mucosa and the lateral border of the tongue usually; we see reticular or erosive lesions mainly, and they are symmetrical (found on both sides of the oral cavity) which helps in diagnosis.

Oral mucosa lesions presents earlier and lasts longer (around 10 years on average), while skin lesions lasts between 3-5 years.

We use many medications to treat and come over the symptoms of this disease:

* Chlorhexidine: it takes long time
* Betensol mouth rinses: a steroid oral wash, usually we dissolve 0.5mg in 10ml of water, sometimes we use 1 or 2mg tablets, then make the patient rinse his mouth with it for 3-5 minutes and not to swallow it.
* Calcineurin inhibitors
* Hydroxylchloroquine: it’s an anti-malarial drug, if the last 3 medications doesn’t work then we go to use this one
* Neuro-suppresants drugs: MMF, azathioprine, dapsone, methotrexate (Mtx)
* Rituximab: it’s a new biologics works on CD20 which found on the B-cells, it makes \*(can’t hear the word at 19:05)\* modulation, it used to treat pemphigus also.

Lichen planus may leads to scarring in the mouth, eye or the genital area, in the mouth scarring causes microstomia which affect maintenance of oral hygiene, any process includes scarring lesion means there’s a risk of malignancy .

There’s a definite risk of malignancy in lichen planus but is low.

Association of skin lesions on the skin around the genital area, the eye and the ear is important for a dentist to diagnose lichen plans before referring the patient to a dermatologist.

**Pemphigus Vulgaris**

It’s an auto-immune disease, there’s antibodies formed against desmoglyne (DSG) which is part of the desmosomes, this affect the junction between the keratinocytes causes a superficial blisters on the skin, we can’t see these blisters because they easily ruptured, unlike pemphigoid which has deep blisters.

In Pemphigus, the antibodies attacks different type of desmoglynes like DSG 1 and DSG 3 in the skin, causes Pemphigus foliaceus or Pemphigus vegetans or any type of Pemphigus, DSG 3 is more abundant in the oral mucosa so it’s mainly affected.

Oral manifestation for this disease comes earlier than skin manifestations by 5 months on average.

A long with genetic tendency there’s many drugs could worsen the disease like anti-TB medications, ACE inhibitors, CCBs and rifampicin.

There are a lot of sites where we can see manifestations of this disease, including the skin, mouth, eye, ear, genital area and other sites.

It’s unlikely to see the intact vesicles or bullae because they could rupture easily due to they are superficial, it’s mostly diluted and wide spread.

Ulcers formed in this disease are irregular and usually seen on the buccal mucosa, gingiva and the palate, they heals without scarring.

 Before treatment with steroids Pemphigus was a fatal disease, they die from the complications, infections, oral involvement and lack of nutrition, now it still significant, up to 15% of Pemphigus patients dies from the infection usually, Pemphigus patients around 50-60 years old.

Steroids treatment for Pemphigus patients lasts for a long time which is bad but we have to, we start our treatment with very high doses of steroids, 1mg per kg this is around 70-80mg dose sometimes we could use 40mg dose, then we lower the dose gradually until we reach 5mg and stay on this dose for almost a year, if we need to use high doses for a long time in some cases we use steroid sparing agents such as mycophenolate, azathioprine and, rituximab. In 75% cases we stop the treatment in 10 years, but some say remediation happens when all ulcers disappear.

Oral manifestations are harder to treat and take longer time than skin manifestation.

Paraneoplastic Pemphigus: in this type oral involvement is very severe much than the cutaneous involvement, so you have to look for malignancy signs like checking the lymph nodes, weight loss and loss of apetite

Ectodermal dysplasia
anything from the ectoderm can be affected “nails, teeth, hair, skin”, it can be “hidrotic or anhidrotic” it can be caused by the effect on different genes, like p63.
It can be x-linked, autosomal recessive.

Epidermolysis bullosa “eb”
erosion, scarring, ulcerations on the fingers and skin, it’s a rare genetic conditions, it has many types and the oral manifestations differ accourding to the type, for example dystrophic epidermolysis bullosa affects collagen 7, which is under the basement membrane, so the ulcerations that result will be deep, while junctional epidermolysis bullosa it affects the junction between the dermis and epidermis, it affects the laminins and the ulcers aren’t that deep, there is also eb simplex which affects keratin 5.
children with eb have skin fragility “in severe cases the skin can be removed from their body just by touching it” it can also affect the eyes, oral mucosa “blisters, microstomia, enamel”, dental hygiene will be bad, because the scarring is deep it may cause deformities “in the fingers”, the esophagus will be constricted “due to ulceration and scarring” and they can’t eat.
junctional epidermolysis bullosa can be autosomal recessive and dominant so you should ask about the family history in any case.
In junctional eb you can see enamel hypoplasia because the laminin is a component of the enamel, while in dystrophic eb the enamel isn’t affected due to the absence of collagen 7 from it.

Oral aphthous ulcers
can be caused by stress, iron deficiency, malnutrition, infections, celiac, paget, bullous, lupus and medications, all of those can cause aphthous ulcers
There are major and minor types

Paget’s disease
common in mediterranean communities, there are certain criteria that should be present to call the disease Paget’s disease; oral ulcers, genital ulcers, eye involvement “but remember that there is nothing that will happen in 100% of the cases, but these are the criteria”
the patients are young, they suffer from hypercoagulation state “thrombosis, aneuresims”, they suffer from pustules , erythema nodosum “can happen in streptococcal infections, contraceptive pills, during pregnancy, some medications” , pathergy “in the place of a trauma, ulceration or pustule will result”(it’s important) or arthralgia.
The ulcers are recurrent and happen more than 3 times/year, and they last longer than other ulcers.
Pustules and erythema nodosum aren’t specific for Paget’s disease.

orofacial granulomatosis
it’s a granulomatus disorder, it causes swellings in the lips and facial region, it may happen due to allergies to certain food “the swelling happen due to the effect on the lymphatics.

Non caseating granuloma relate to sarcoidosis and gross disease (swelling in lower or upper lip)

* We can use the patch test to help us ,but sometime not available or may some people

Suffer from allergies from sanimat or benzoate ,also we can depend on family history or

GI system to detect gastro tubercle

* Rule out :

1-TB

2-sarcoidosis

3-chrons disease

-erythema multiforme :

Important because its common in minor form

Have different type :

1-Erythema minor :

Common , have multi form in the mouth like erythema minor with extra mucosal

Involvement (mean repeatable involvement of skin less than 10%)usually from infection

Mostly from herpes simplex virus (HSV) and may include other infection

2-erythema major (sever erythema multiforme ) :

Relate to (stevens-johnson syndrome ) involvement skin more than 10%(10-30 %)

The most sever one is toxic epidermal necrosis ,usually drugs epidermolysis necrosis

Over 30%of skin involvement

* The erythema multi forme in typical description we say it (ring inside ring )may

 involvement the eye

In keratinocytes we have fas molecule and fas ligamentigly its a receptor to the drug

When drug linked to it lead to apoptosis and necrosis

And lead to loss of all epidermis , the skin very painful and dusky color ,also it affect on eyes , nails

\*note:

Not all skin affect just the keratinocyte

-mortality rate in sever cases is high it reach up to 90%

The problem in patient mouth in all mucosa the patient can’t eat

-we have scorten system its just use for prognosis , the elderly people have bad prognosis

If the score more than 5 the risk of dentist is 90 %

This patient must be in ICU unit for support because they don’t have any barrier

(loss of epidermis) the risk of infection is high

-minor is common ,and major (sever) is usually from toxic drug so must be stop this drug

Treatment :

Usually its supported therapy (ICU, give fluid , keep the patient hydrated , may give him immunoglobulin(antibody)we didn’t give steroids drug .

* Lupus erythromatosis :

Not common , other features :

1-Oral ulcer (deep oral ulcer and scaring )

2-hair loss

3- photo sensitive

4- renal involvement

5- hematological involvement

6- its affect on psychological aspect of the patient because its appearance.