> we refer to last year's sheet and we add extra information

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*White lesions*

The normal color of the mucosa is pink, and as we know the epithelium is avascular ; not pink in color ; so the pinkish color comes from the red color of oxygenated blood present in the underling connective tissue modified by the overlying thickness of epithelium , by presence or absence of keratin , and by the activity of the melanocytes in the basal layer.(and by the amount of vascularity in the C.T)

so we expect that if there is an increase in the keratin layer at the surface (hyperkeratinization ); either orthokeratosis or parakertosis, or increase in the thickness of epithelium ; acanthosis or hyperplasia , or if it was nonkeratinized and became keratinized(keratosis) or edema within the epithelium or if there is a dense inflammatory cell infiltrate , these will prevent us from seeing the color of blood and will give the lesions the whitish appearance , so when you see a white lesion you expect one of these structural changes

But be sure first that this color is not a deposit on the surface, because sometimes deposit of something gives whitish color. You check it bygauze;you scrape the lesion, if it is external deposit like material Alba or plaque on the gingival (in patient with very poor oral hygiene) which gives the color of a whitish lesion when you scrape it with gauze the whitish color will disappear. The same applies to coated tongue; which is whitish dorsum of the tongue due to accumulation of desquamated cells and debris in patients with febrile illness or due to fasting.

Be careful when you see whitish/yellowish lesions that it can be caused by loss of these layers (ulcer), acute ulcers: bleeding with time we’ll have granulation tissue and Fibrinous exudates and bacteria and debris which give the yellowish color. So erosion or ulceration in the oral cavity will appear whitish or yellowish.

* You have to differentiate between coated and hairy tongue, both look white layer on the dorsum of the tongue . if this layer is **constant** this is called **hairy tongue** that happens due to elongation in the filiform papillae ( increase in the keratin ) it might be brown or black in heavy smoking patients (this is a protection mechanism ). but if it’s a deposit of desquamated cells , food debris , bacteria ,or others on the surface , in temporary conditions , like febrile illness , fasting , or respiratory tract infection or other illness , this is called **coated tongue** , it’s **temporary ;** related to the illness , where there is no mastication or swallowing , etc , so we have deposit of things over dorsum of the tongue which give the dorsum whitish color that disappears when tongue function retains.
* classification of white lesions is according to the etiology of the white lesion :

1. Hereditary causes, oral epithelial naevus or white sponge naevus , leukoedema and other rare lesions.

2. Traumatic due to *chronic mild irritation* ( acute conditions would result in ulcers not keratin deposition), mechanical (frictional keratosis) or chemicals that cause irritation of the oral cavity, or thermal.

3. Infection; fungal, candidal. We have three types of candidal infection that give a white lesion in the oral cavity; other candidal infections appear red lesions. Syphilitic leukoplakia( in tertiary lesions of syphilis) although it’s rare now, it gives a white lesion in the oral cavity. Viral infection like hairy leukoplekia in immunocomrmized patients Like HIV patients

1. Dermatological: skin and oral lesion that appear as white like lichen planus, and lupus erythematosus.
2. Sometimes you see white lesion that can be a sign or manifestations for oral caner or potentially malignant lesion.
3. Idiopathic: without any one from the causes above, called idiopathic white lesion or leukoplakia.

***Hereditary:***

*White sponge naevus*:

from the name it’s white , sponge like , autosomal dominant ; there is a family history ; so there is early presentation of this white lesion , ( usually other types of white lesions appear in adult , so when you have a patient with chronic white lesion in the oral cavity , you have to think about hereditary cause and one of them white sponge naevus) - genetic cause : mutation of genes coding for keratin 4 or 13 , which make a disturbance in the epithelium , that gives the whitish appearance .

- clinically : asymptomatic , painless whitish lesion , soft like a sponge , irregular with no defined border ; it’s a gradual transition to the normal mucosa , most commonly it’s bilateral at the buccal mucosa , and as we said the patient is a child with family history .

Can affect also soft palate and the tongue, and it’s rare to be seen at the gingival margin. the lesion surface is usually shaggy (not uniformed).

* one of its specific features is that it can affect other sites other than the oral cavity, other mucus membranes like nose , esophagus , genital regions , so this can help you in the diagnosis.(rarely affected areas are dorsum of the tongue and gingival margins)

- histologically : ( the structural changes ) acanthosis ( hyperplasia ) , moderate\_marked hyperparakeratosis , and marked intracellular edema which is called basket-weave appearance , this edema happen mainly in prickle cell layer (this edma is the cause of the spongy appearance and softening). alsoyou can see condensation of keratin around nucleus , and this we can see it in smear , under the microscope we see condensation of eosinophilic material around the nucleus , which is the keratin , this is a cellular change in white sponge naevus . No dysplasia in the epithelium so it’s ***not potentially malignant***. No inflammation in the lamina propria so the problem was not initiated in the connective tissue, white sponge naevus started in the epithelium and as we said it’s a genetic mutation in the keratin.

* lamina propria : connective tissue under the epithelium . lamina propria with the epithelium is called oral mucosa .
* this lesion has a good prognosis >> wont undergo dysplasia

*Leukoedema*:

*Leuko* :white

*Edema in the epithelium*

White lesions at the buccal mucosa , usually bilateral , translucent , look like lichen planus . present in high percentage of people mainly people with dark skin , the darker the skin the higher the percentage of leukoedema . as we said most commonly at buccal mucosa , but we can see them at the lateral border of the tongue . asymptomatic , diffuse translucent , grayish white folded lesions .

How to confirm diagnosis?

**stretch test** : when you stretch the buccal mucosa using the mirror or your hand and the mirror,the lesions disappear . this confirm the diagnosis so no need for biopsy or other tests , it’s very easy to diagnose . the lesions disappear because they mainly made of edema in the epithelium so when you stretch the mucosa the thickness of epithelium become normal again so the lesions disappear .

this means that the lesion is not made of keratin ,if the lesion is made of keratin , stretching won’t change anything in it .

histologically: and specially in buccal mucosa you can see mild parakeratosis and intracellular edema like white sponge naevus, with normal lamina propria(no inflammation,no dysplasia).acanthosis because of the intracellular edema.

Prognosis :it’s a variation of normal,but you have to know how to diagnose it because we will take later a lesion that appears as white lines on the buccal mucosa which is lichen planus,when I do stretching for this lesion it does not disappear because it is made of keratin.

How to differentiate between leukodema and White *sponge naevus?*

* the coming lesions we are not going to talk about them in details , but you have to read about them from the reference , they are rare but in all of them , we see lesions that look like white sponge naevus, hereditary ( in young patients ) and have other manifestations :

1. pachyonychia congentia :

characterized by thick nails and whitish patches in the oral cavity (white lesion in the oral mucosa)that histologically looks like white sponge naevus .

1. dyskeratosis congentia :

there is a problem in the keratin in the epithelium, you have to remember that they have two things in the oral cavity:

A-Whitish lesion in the oral cavity, , which is ***potentially malignant*** .

\*Remember: White sponge naevus it’s a whitish lesion but it’s ***not potentially malignant.***

B-the patients have destructive perodontitis (severe perodontial disease around the teeth)

1. tylosis :

leads to esophageal cancer in the patient . thick keratin at the feet & soles and in palms . and they may have Oral hyperkeratosis.

1. hereditary benign intraepithelial dyskeratosis :

in the eye presented by dense conjunctiva that can lead to blindness , whitish lesions in the oral cavity .

1. follicular keratosis :

there is a suprabasal cleft in the epithelium like pemphigus it makes intraepithelial clefting , and like vesiculobolus disorders ( lesions that make bolus or vesicles as a result from the separation ) that we will talk about it later on .

\*note :refer to book to see the main features of the above 5 lesions.

***traumatic keratosis :***

oral mucosa is more common to be affected by trauma,and you as adentist one of the factors that cause trauma to oral cavity. But dentists usually cause ulcers.

*mechanical* is the most common cause of white lesions . trauma by mild chronic irritation or friction , not acute trauma \_ acute trauma lead to ulcers and hematoma \_ . as a result from the chronic trauma there will be hyperplasia and hyperkeratosis , and these two give the whitish lesion . hyperplasia and hyperkeratosis are made to protect the mucosa .

- this is called frictional keratosis , it’s mechanical , prolonged mild abrasion or irritation ; can be from : a) the teeth , b) habits like lip or cheek biting or friction between lip and teeth to relieve stresses) , c) rough tooth surface , d) rough lingual restoration that can irritate the lateral border of the tongue , e) dentures make white lesions in the vestibule , f) Vertical tooth brushing ( some patient brush the gingival ) this type is associated with recession with whitish membrane on the gingival , and usually these patients show good oral hygiene and they are usually educated g) alveolar ridge in edentulous spaces with mastication , they become traumatized and whitish.h)habits of sucking the lip,so there will be irritation of the lip on the lower teeth.

- clinically : dense whitish patches , with rough surface , usually lesion from the trauma have rough surfaces because when the patient bites , he removes some parts from the epithelium , so some regions become keratotic and thick and others become thin due to erosion and removal of epithelium.

* How to differentiate cheek biting from white sponge naevus?

From history if there is cheek biting ,trauma. From the clinical image both are seen most bilaterally on buccal mucosa. traumatic keratosis has definite bonders not gradual like white sponge naevus , but more important you have to pay attention to the extension of the lesions , in traumatic keratosis the lesion is only parallel to the occlusal line in the area that the patient can reach in cheek biting , it can never reach the vestibule . plus the presence of family history in white sponge naevus . in white spongy naevus there are other sites affected while in the traumatic keratosis the mouth is only affected .

- diagnosis of traumatic keratosis : first you have to find the cause , that fits the shape and the size and the location of the lesion , if there is no cause there is no diagnosis, then when you find the cause ,you have to remove the cause , and expect that the lesion will heal with time , it takes more time than ulcers , but within three to four weeks the whitish color should be less clear .if it doesn’t heal we should take a biopsy and search for another cause.

Examples:

1. We have roughness on the buccal surface of a tooth, and we have a lesion covering all the buccal mucosa >here the cause does not fits the lesion
2. We have roughness on restoration on the lingual surface of a lower tooth, and we have a lesion on the lateral border of the tongue >here the cause fits the lesion because the tongue is moving and the roughness may cause a friction on all the lateral border of the tongue.

Histologically : hyperplasia and hyperkeratosis , No dysplasia . the irritation reaches the lamina propria so we will find in it scattered chronic inflammatory cell infiltrate .

**Linea alba**: sometimes the patients have a white line parallel to the occlusal line , and they don’t have cheek biting habit , this happen due to spontaneous irritation to buccal mucosa during occlusion , other patients suck their buccal mucosa , which lead to elevation in the buccal mucosa parallel to the occlusal line . It happens involuntarily, white lines are not very extended.

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*chemicals* can lead to whitish lesion , common example is aspirin , some patient think that by swallowing the drug the effect of aspirin will takes time , since it will go to the stomach then intestine , then to the liver then it will spread to reaches 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 .

\* 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 pt 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 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 hyperkeratosis and hyperplasia . and they are potentially malignant.

*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 pip 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: 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 in the palate, but in other places of the oral cavity: like tongue, floor of the mouth, etc.

\*remember: cancer of the palate is a rare condition.