

**TRAUMATICALLY DISEASES OF THE ORAL MUCOSA.  
CLINICAL PICTURE. POSITIV AND DIFFERENTIAL  
DIAGNOSIS. TREATMENT**



**Lecture № 2  
5 Year  
10 Semester  
V.Nicolaiciuc**

# TRAUMATIC DISEASES OF THE ORAL MUCOSA. CLINICAL PICTURE. DIFFERENTIAL DIAGNOSIS. TREATMENT

## Traumatic lesions of the oral mucosa

Oral mucosa has a high protective and regenerative abilities, so weak mechanical and physical influences to which it is exposed during a meal, do not have a significant impact on it.

Pathological changes in the mucosa are exposed to the same stimuli, but more power. Feature of mouth is that any traumatic mucosa immediately accompanied her infection.

The extent of damage and the clinical manifestations depend on the nature of the stimulus, the time and effort of its impact, location, individual characteristics, the general condition of the body, age.

### **Distinguish trauma:**

- mechanical (acute and chronic),
- physical (heat and radiation)
- chemical.



## Acute mechanical injury(trauma)

### **Etiology and pathogenesis:**

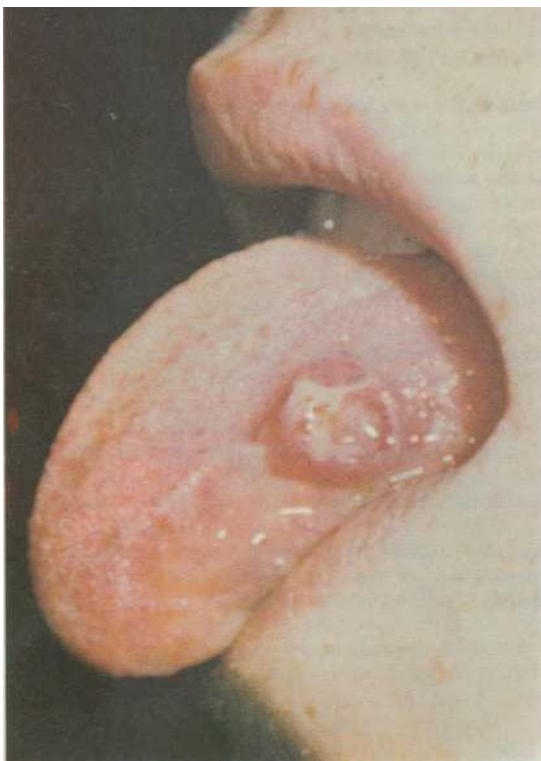
Is the result of stroke, bite, wounds or cutting with a sharp object. Injury may be accompanied by a violation of the integrity of the epithelium, which leads to ulceration or erosion, or interstitial hemorrhage occurs without compromising the integrity of the epithelium (hematoma). Around erosion develops an inflammatory reaction to the localized character of the actual infiltration of the mucosa.

### **Clinic picture:**

Showed a slight pains on the site of injury. When interstitial hemorrhage after 1-3 days revealed hematoma bluish-black in color. If the damage of the epithelium formed painful erosion, infiltrated the base, which is usually quickly epithelialized. In the case of secondary infection may progress to erosion nonhealing ulcer.

### **Diagnostics:**

Usually not difficult. The reason is easy to diagnose when collecting history.



Changes in the mucous membrane during acute mechanical injury

- a) An ulcer on the side of the tongue;
- b) Hematoma and erosion on the lateral surface of the tongue;
- c) Erosion and spot hemorrhage;
- d) An ulcer and a hematoma on the lateral surface of the tongue.



### **Treatment:**

- Inspect the wound
  - To stop the bleeding, using 0.5 - 1% solution of hydrogen peroxide or 5% solution of  $\epsilon$ -aminocaproic acid.
  - In severe pain hold applique, irrigation, bath or rinse 0.5-1% solution of novocaine, 0.5-1% lidocaine.
- Schedule irrigation solutions not irritants warm antiseptic (0.02% solution Frc, 0.5% solution etoniya, 0.02% solution ethacridine lactate, 1% solution Dimexidum), application of foam aerosols (panthenol , olazol, gipozol) or of 5% metiluratsilovoy ointment.
- When deep wounds sutured.

### **Chronic mechanical injury (trauma)**

#### **Etiology and pathogenesis**

The cause may be a long injury mucosa sharp-edged teeth, poorly made or outdated prostheses, located outside the arc teeth. Traumatic factors may be overhanging edge fills, wire bus or ligatures when splinting jaws, bad habits.

They have the character of long-term exposure, which starts and supports a mechanism catarrhal stage has hyperemia, exudation and **proliferation.**



The severity of each of them depends on the strength and duration of the stimulus.

Exudation is sufficiently severe. Exudates can be serous, sero-purulent and purulent. In purulent exudates mucosa exposed surface damage, which leads to erosion.

If untreated, develop chronic focal purulent inflammation. The result of this is the appearance of inflammation dekubitalnoy (traumatic) ulcers.

Inadequate stimulus of the oral mucosa can be any prosthetic or orthodontic appliances.

Denture passes chewing pressure on the mucous membrane, delay self-cleaning of the mouth, which leads to a change in the established microbial balance. Irritant has an intermediate portion of bridges, if it touches the mucous membrane of the alveolar region. Artificial crowns are also an irritant.

### **Clinic picture:**

- ❑ Complaints can't be a long time, but usually the patient indicates embarrassment, discomfort in the mouth, a small pain, swelling. Changes may take the form of catarrh inflammation, erosion or ulceration. The ulcers are painful, especially when eating and speaking.
- ❑ Dekubital ulcer is usually single, the mucous membrane around the swollen, hyperemic, moderately or severely painful. Ulcer has ragged edges and the bottom, covered with easy to shoot fibrinous or necrotic deposits.

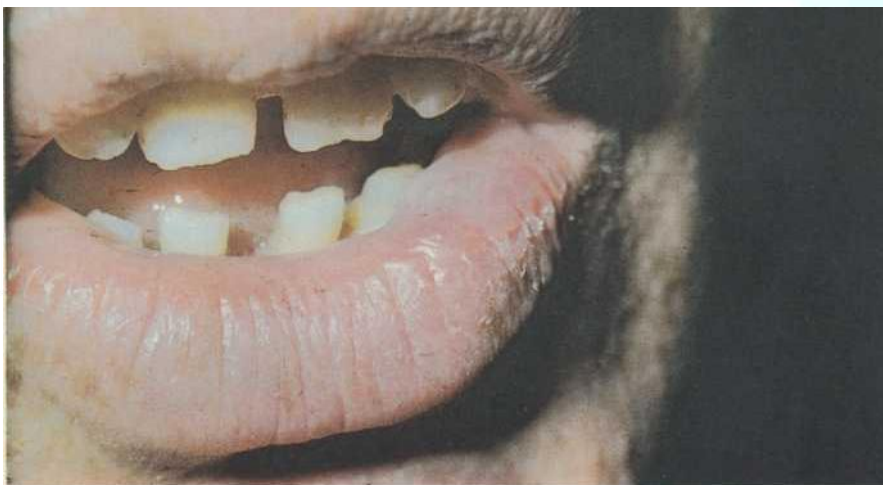


- ❑ In this case, the regional lymph nodes are enlarged and tender. Localized ulcers usually on the tongue and buccal mucosa through for bite line of tooth. With long-term existence of the edge and the base of ulcers compacted.
- ❑ Its depth varies, up to the muscular layer. Focus of inflammation usually coincide with zones of partial denture fit of prosthesis. Hyperemia of the mucous membrane without breaking the usually, observed when using removable dentures for 1-3 years.
- ❑ With longer use arise erosion, ulcers, hyper plastic papillomatoz growths. More likely to have chronic hypertrophic processes - hypertrophic gingivitis or papillitis, papillomatoz sprawl, prosthetic granuloma, traumatic abscess, hyperkeratosis. Long edge prosthesis irritation can lead to lobular fibroids, which has the form of several folds, parallel to the edge prosthesis.

### **Differential diagnosis:**

**Dekubital ulcers** must be differentiated with a cancerous ulcer, tuberculosis ulcers, chancre, tropic ulcer. Traumatic erosions and ulcers are differentiated pemphigus.

**Traumatic ulcers** painful on palpation, is an inflammatory infiltrate in the ground. It is characterized by the presence of irritating factor, after elimination of that ulcer heals in 3-5 days. Cytology no specific changes.

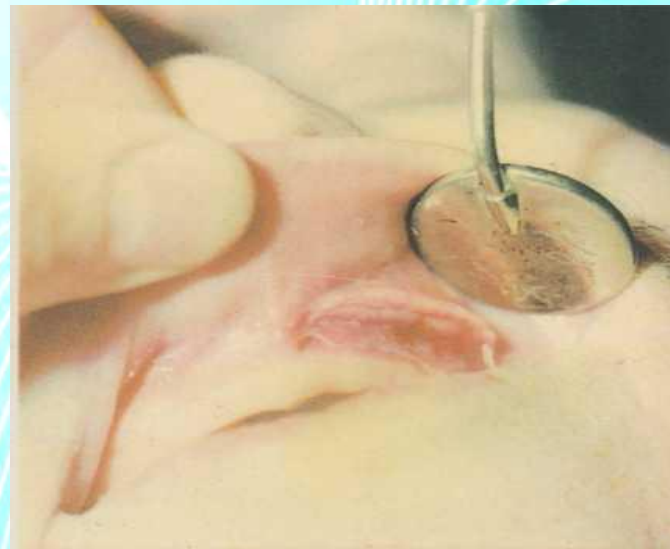
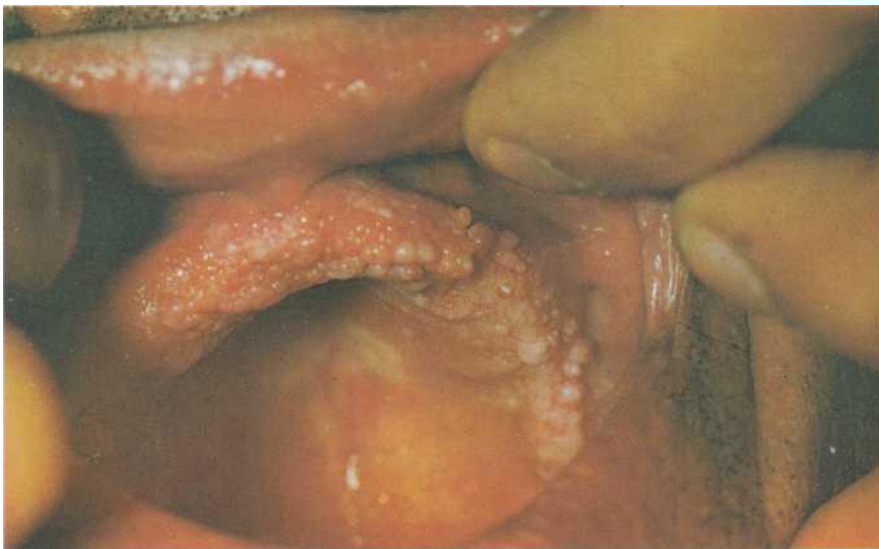


- a) An ulcer with infiltrated edges and keratinization;
- b) Ulcer with mild symptoms of infiltration



- a) An ulcer without inflammation;
- b) Deep ulcer with signs of malignancy.





- a) Papillomatous growth of the hard palate when using a removable prosthesis;
- b) Prosthetic granuloma (lobular fibroma) along the transitional fold. Papillomatous growth with a linear ulcer



**Cancerous ulcer** there a long time, has a thick edge and bottom, characterized by growths on the edges, resembling in appearance cauliflower, cornification edges. After the appearance of a cancerous ulcer pain subsides. Removal of the stimulus does not lead to healing. Cytology in scrapings found abnormal cells.

**Tuberculous ulcer** is characterized by sharp painful, soft jagged edges. The bottom of the ulcer with yellow granular inclusions (grains Trill). Cytology in scrapings from the surface of ulcers found Langhans giant cells.

**The chancre** is sealed at the bottom, the edges are smooth, dense, smooth bottom, painless, meet-red color. Regional lymph nodes are enlarged, firm consistency, mobile and painless. In scrapings from ulcers detected pale treponema (dark field microscopy). After 3 weeks after the chancre becomes positive Wassermann. Elimination of traumatic factors, if any, does not affect the chancre during which no treatment may be several weeks or even months.

**Trophic ulcer** is characterized by the existence of a considerable duration, sluggish stream, the presence of patients with chronic somatic diseases. Annoying irritants factor promotes healing of ulcers. In traumatic erosions, unlike pemphigus, Nikolsky sign negative, no smears akantolitic Ttsank cells. After eliminating the stimulus traumatic ulcers and erosions heal quickly.



## Treatment:

The first step is to eliminate or weaken the effect of irritating factor. Never carry a defective denture is recommended rational prosthesis. Need to replace poor quality non fixed prosthesis and fills.

Be sure to dental health (sanation) and multiple professional hygiene. With a sharp pain carried by any warm anesthetic anesthesia: 0.5-1% solution lidocaine 0.5-1% solution of novocaine, 2-4% solution piromekaine, in the form of applications, irrigation, baths or rinses.

If there is necrotic or fibrinous deposits on the surface of erosion or ulceration of recommended application of proteolytic enzymes for 8 - 10 minutes, after which the necrotic tissue or fibrinous deposits removed mechanically, and an ulcer or erosion treated with an antiseptic (0.02% solution Frc, 0, 5% solution of hydrogen peroxide rum, 0.5-1% solution etoniy, 0.5-1% solution Dimexidum).

Applications are imposed foam aerosols, metiluratsilovoy solkoserilovoy or ointments, and from the start of epithelialization - applications keratoplasticheskie funds for 15-20 minutes 3-4 times a day (sea buckthorn, rosehip Vinylinum, vitamin A or E in oil ).

Treatment of lobular fibroids is to correct prosthetic design, if possible, or just recommended rational prosthesis and applications keratoplastic funds for 15-20 minutes 3-4 times a day. If necessary, and the testimony of a lobular fibroids excised and made a new prosthesis.



## Thermal injury (burn)

### **Etiology and pathogenesis:**

The reason is the effect on the mucous membrane of the high temperature in case of accidents in the home and in the workplace. Thermal damage can occur by careless use electrocoagulation, electrophoresis or receiving too hot food.

The degree of damage depends on the temperature and duration of exposure, burns may be limited or diffuse, accompanied by the formation of bubbles or congestion area prior to the development of extensive deep tissue necrosis. Inflammatory changes may extend beyond affected site. Secondary infection complicates and slows down during epithelization.

### **Clinic picture:**

Pain at the time of impact of the stimulus, which passes quickly. Therefore crucial in the diagnosis of a medical history. Then there is the pain on action of any irritants, talking, eating and even at rest. Objectively determined by congestion, edema, painful blisters or surface erosion, which are formed at the opening of bubbles. In more severe cases defined area of necrosis, which can have color from off-white to dark brown.



## **Diagnostics:**

Diagnostics based on the history and objective data.

## **Treatment:**

Applications analgesics medication (0.5-1% solution of lidocaine, 0.5-1% solution of novocaine, 2-4% solution piromekaine, piromekaine ointment)  
Processing of weak warm antiseptics.

Widely used foam spray.

Since the beginning of epithelialization used applications keratoplasty.

## **Acute chemical damage**

### **Etiology and pathogenesis:**

Acute chemical damage to the mucous membranes of the mouth occur when exposed to a concentrated solution of alkali, acids Accidental injury in the home, at work, or a suicide attempt, as well as the effects of the substances used for the treatment of teeth (silver nitrate, alcohol, alcoholic tincture of iodine, resorcinol -formalin mixture or paste, arsenic paste, EDTA, phenol, etc.).



Chemical burns can develop from non fixed prostheses when inadequately polymerized plastic, it exposure monomer on the oral mucosa. Often in practice, there are burns of the mucous membranes of the mouth with the action of a aspirin, analgin, and various tinctures patients used independently when appear acute dental pain, if these drugs are applied to the painful tooth.

**Acids** cause damage to a lesser depth than the **alkali** as tissue proteins coagulate, forming a thick scab (deposits), but alkali make kollikvatsionny necrosis.

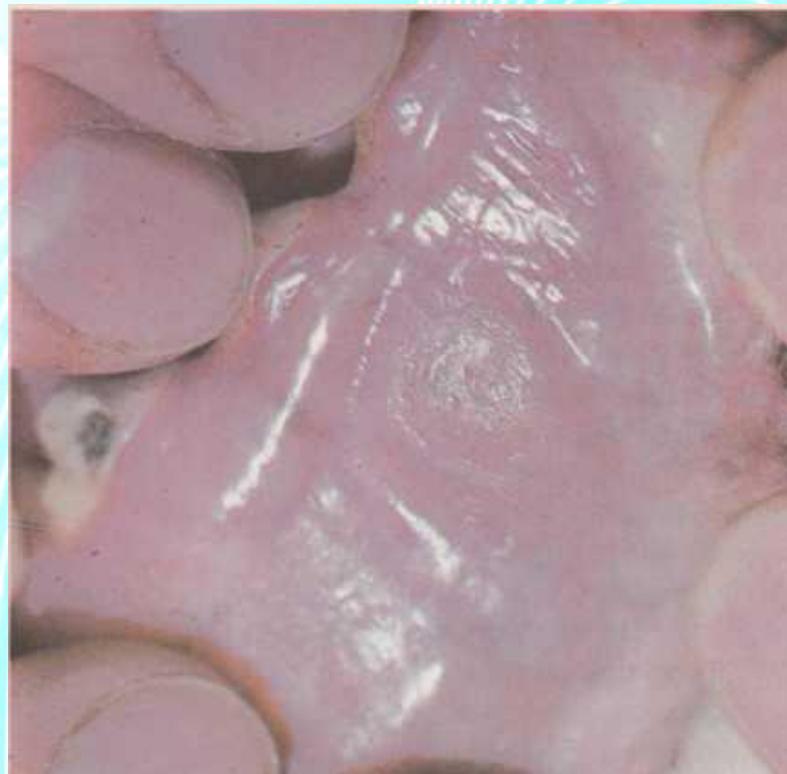
The toxic effect of arsenic blocks the tissue respiration and metabolism in cells, is compacted, protein denaturation and necrosis of tissue with a scab.

### **Clinic picture:**

If you get burned with acid in the mucosa of the mouth formed necrotic film, the color of which can be varied: brown (from sulfuric acid), yellow (nitric acid), white and gray (other acids).

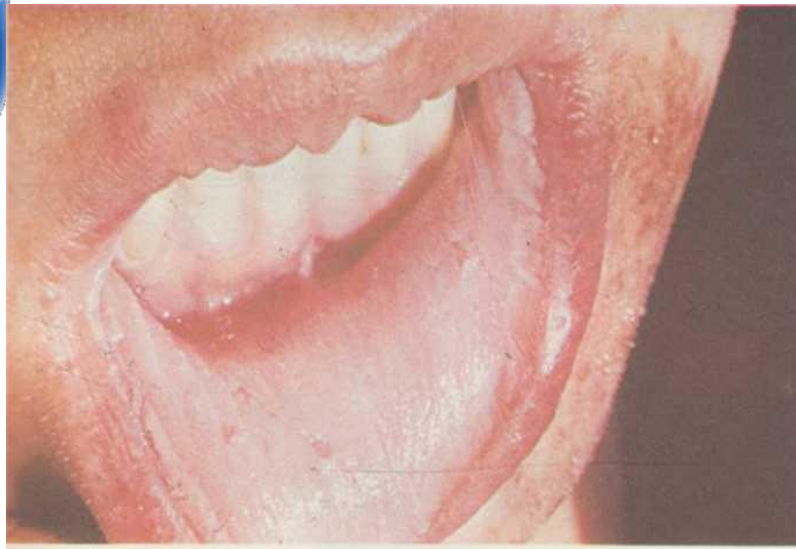
The films are the inflamed hyperemic, edematous, mucous lips, gums, cheeks, palate, and tightly connected with it.

Due to the impact of alkalis in the mucosa occur deeper damage, necrosis area extends to all layers of the mucosa. The area of impact is sharply, painful lesions, regardless of the depth of the lesion. Chemical burns cause severe suffering to the patient.



Necrotic changes in the mucous membrane from exposure to alkali and acid:

- a) Necrotic film on the ulcer - alkali burn;
- b) Focus of necrosis after a carbolic acid burn.



- a) Changes in the mucous membrane of the lips after exposure by analgine (hyperemia and point epithelial necrosis);
- b) Hyperemia and necrosis of the mucous membrane under the influence of resorcinol - formalin liquid.





## Diagnosis and differential diagnosis:

Must be differentiated chemical burn from an allergic reaction to the plastic, an amalgam or small concentrations of chemicals.

## Treatment:

Quickly remove the chemical agent. Copious irrigation, irrigation and rinsing with weak solution neutralizing agent or antidote. In their absence spend washing with water.

The **acid** burns can use soapy water, 1% solution of calcium carbonate, 0.1% solution of ammonia (15 drops of 10% solution in a glass of water), 1 - 2% solution of baking soda.

The **alkali** burns as neutralizing agents using 0.5% solution of citric or acetic acid (a quarter teaspoon of 70% acid in a glass of water), 0.1% solution of hydrochloric acid or 2% solution of sulfuric acid.

If you burn 30% solution **silver nitrate** mouth irrigate by hypertonic saline (3.5% solution of sodium chloride) or Lugol's iodine solution.

If you get burned **phenol** mucosa treated with 50% ethyl alcohol or castor oil.

If you get burned **arsenious acid** to treat the affected area with one of the antidotes to arsenic (Lugol solution, 1% solution iodinol, 5% solution unithiola, 30% solution of sodium thiosulfate) or powdered iodoform or magnesia.



Pain control substances in the form of applications 10% slurry anestezina in peach oil, 1% solution of novocaine with urotropine, 2-4% solution piromekaine, 1-2% solution of lidocaine or 2.1% solution three mekaine.

Necrotic areas excised with sharp excavator.

To remove the acute inflammation used corticosteroids applications to the affected areas (0.5% prednizolone ointment, 1% hydrocortisone ointment, 2.5% suspension of hydrocortisone), followed by irrigation of the mucosa weak antiseptic solutions in the form of heat or decoctions of herbs (chamomile, nettle, sage, etc.).

To accelerate epithelialization used keratoplastic as applications.

For a general treatment is indicate not irritate high-calorie diet, a multivitamin with trace elements, calcium preparations, antihistamines, vitamins A and E in the oil. In severe cases, detoxification therapy is administered intravenously

When scarring surgical treatment – cicatrectomy with followed plasticity.



**Simple Leukoplakia** clinically show a plain homogeneous surface that is mirror smooth to faintly dull. The impression of a white spot is produced by the keratinization and / or thickening of the mucosal epithelium, which loses the normal transparency. The color tone varies from milky-cloudy to opaque to resembling mother –of–pearl. Very discrete forms of simple Leukoplakia are not palpable. Marked lesions raised over the mucosal level become palpable.

### **Histologically**

- Simple Leukoplakias are characteristically epithelial condensation with hyperkeratotic surface layer as well as a thickening of the spinous cell layer (acanthoses).
- The keratinization of the mucosa can run a course similar to the physiologic occurrence on the skin of an ortokeratosis (cornification without nuclear remnants) or of a parakeratosis (cornification with nuclear remnants). The epithelial layer have a normal polarity.
- There is no epithelial atypia.
- Subepithelial lymphoplasmacellular infiltrate, if present at all.
- The simple Leukoplakias make up 50% of all oral Leukoplakias.
- When their origin is not known, the term used is idiopathic Leukoplakias. One should consider that idiopathic Leukoplakias may regress if mechanically irritative factor or smoking habits are eliminated, if the stimuli are maintained for a long time.



## Frictional keratosis

Under the classification frictional Leukoplakias fall keratinization of non keratinized mucosa or hyperkeratosis of normally cornified mucosal areas (irritative hyperkeratosis) which are related in a pathogenic manner to chronic trauma.

A long-acting mechanical stimulus on the oral mucosa can lead to keratinization in the same manner as production of a callus. This is also referred to as a pachydermia or as a white spot because a hyperkeratosis, particularly of the orthokeratotic type.

The squamous epithelium presents a normal stratification and orientation. This focal irritative hyperkeratosis are frequently found in the oral cavity usually close to sharp tooth edges, in suitable prostheses, defective support elements, and other problems (chronic cheek and lip chewing).

The surface may become secondarily hyperplastic or ulcerated as a result of mechanical irritation. Making the diagnosis more difficult. Unless the cause is eliminated, recurrence following surgical excision is inevitable.

**Denture hyperplasia** - are a specially likely to be found in patients who have worn a poorly fitting or otherwise faulty prosthesis for a long time. Mechanical stress resulting from anatomically incorrect or functionally inappropriate prosthesis (over extended or defective denture margins in the region of the movable mucosa. Lead to local tissue destruction and traumatic ulceration.

As the irritation continues on this inflamed mucosa, excess granulation tissue develops, that later becomes this fibroma – like tissue.



## Treatment:

Surgical removal of excess fibrous tissue.  
Appropriate prostodontic therapy is also provided.

## Smokers leukokeratosis

Another important example for dentist is the Leukoplakia caused by smoking irritation. Affected predominantly are have smokers, pipe smokers in particular. The cause is not so much thermal effect as the chemical stimulus of tobacco components. Leukokeratosis of smokers presents typical and almost unmistakable finding on the polate.

On the hard and sometimes soft polate, whitish hyperkeratotic epithelial changes of the oral cavity resemble a cobblestone pattern. The openings of the inflamed, swollen minor salivary glands do not show keratinization. They rise distinctly as small, red points from surrounding thickened, whitish mucosa.

Regions of the palate mucosa that are prosthetically covered are spared from the Leukokeratosis.



## Treatment:

Surgical removal of excess fibrous tissue.  
Appropriate prostodontic therapy is also provided.

## Smokers leukokeratosis

Another important example for dentist is the Leukoplakia caused by smoking irritation. Affected predominantly are have smokers, pipe smokers in particular. The cause is not so much thermal effect as the chemical stimulus of tobacco components. Leukokeratosis of smokers presents typical and almost unmistakable finding on the polate.

On the hard and sometimes soft polate, whitish hyperkeratotic epithelial changes of the oral cavity resemble a cobblestone pattern. The openings of the inflamed, swollen minor salivary glands do not show keratinization. They rise distinctly as small, red points from surrounding thickened, whitish mucosa.

Regions of the palate mucosa that are prosthetically covered are spared from the Leukokeratosis.



### **Treatment:**

1. Reduced tobacco consumption by the patient.
2. Total cessation of smoking.

After that the changes disappear. According to current knowledge the development of cancer from leukokeratotic lesion is unlikely. This finding is surprising, because smoking has been demonstrated to increase the cancer risk on the bronchial mucosa and on the lip mucosa.



# The End