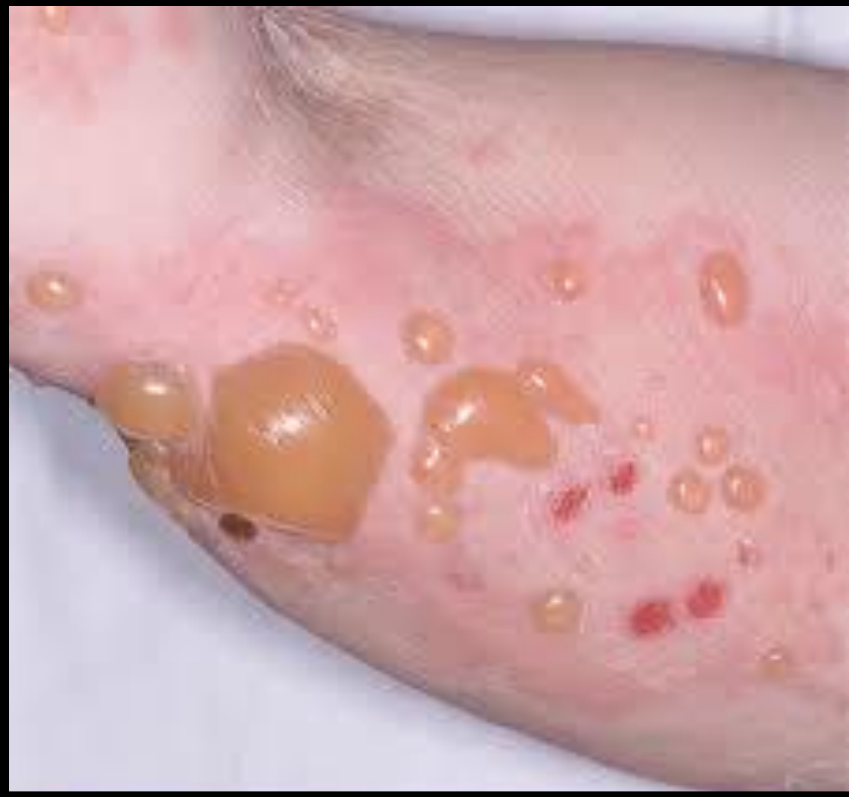
Immune- autoimmune diseases I.

Pemphigus group

Pemphigoid group

Erythaema multiforme group

Lichen group



Blistering diseases

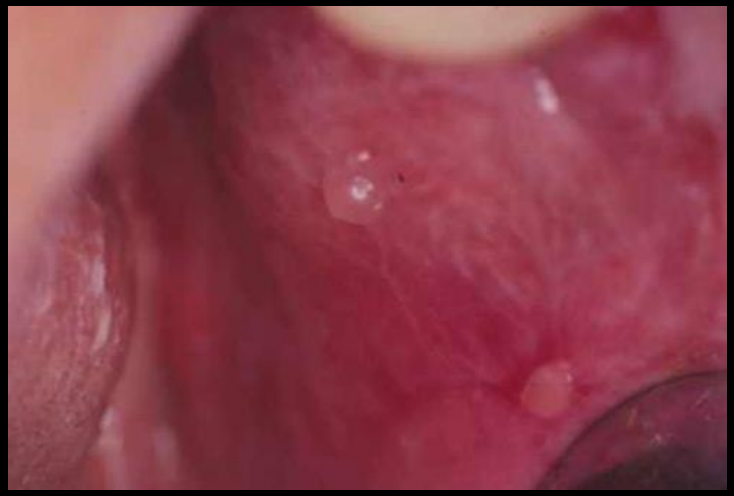
There are many oral diseases with blistering forms.

Blister can be:

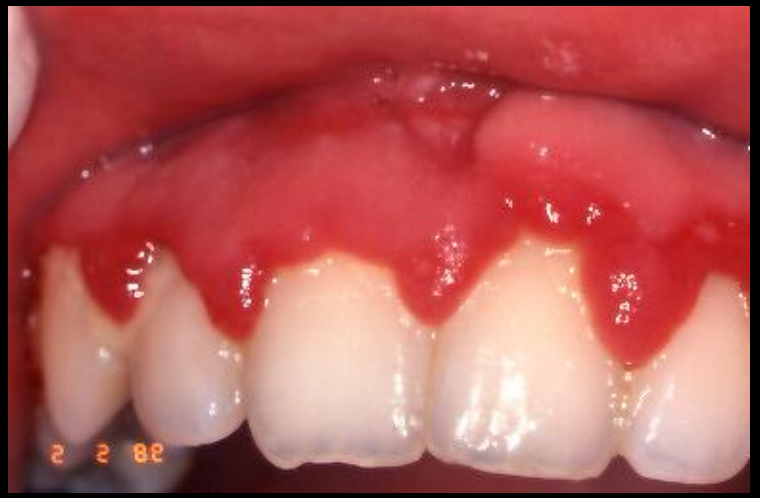
- **small and large**
  - **Vesicle**
  - **Bulla**

Can be:

- **intraepithelial**
- **and subepithelial**



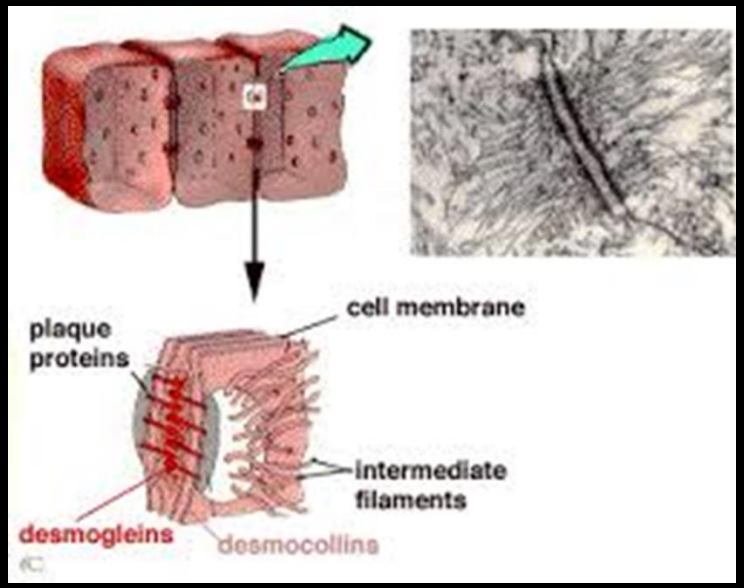
Lichen Oris

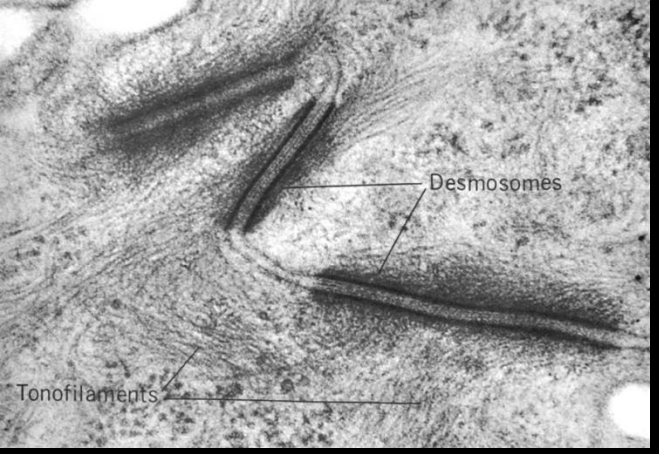


herpes

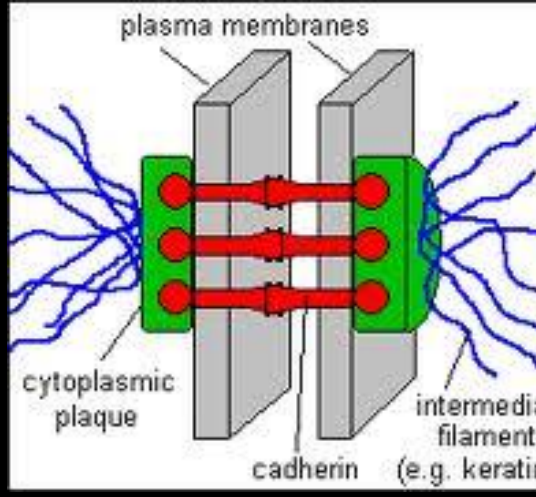
Epithelial Biology

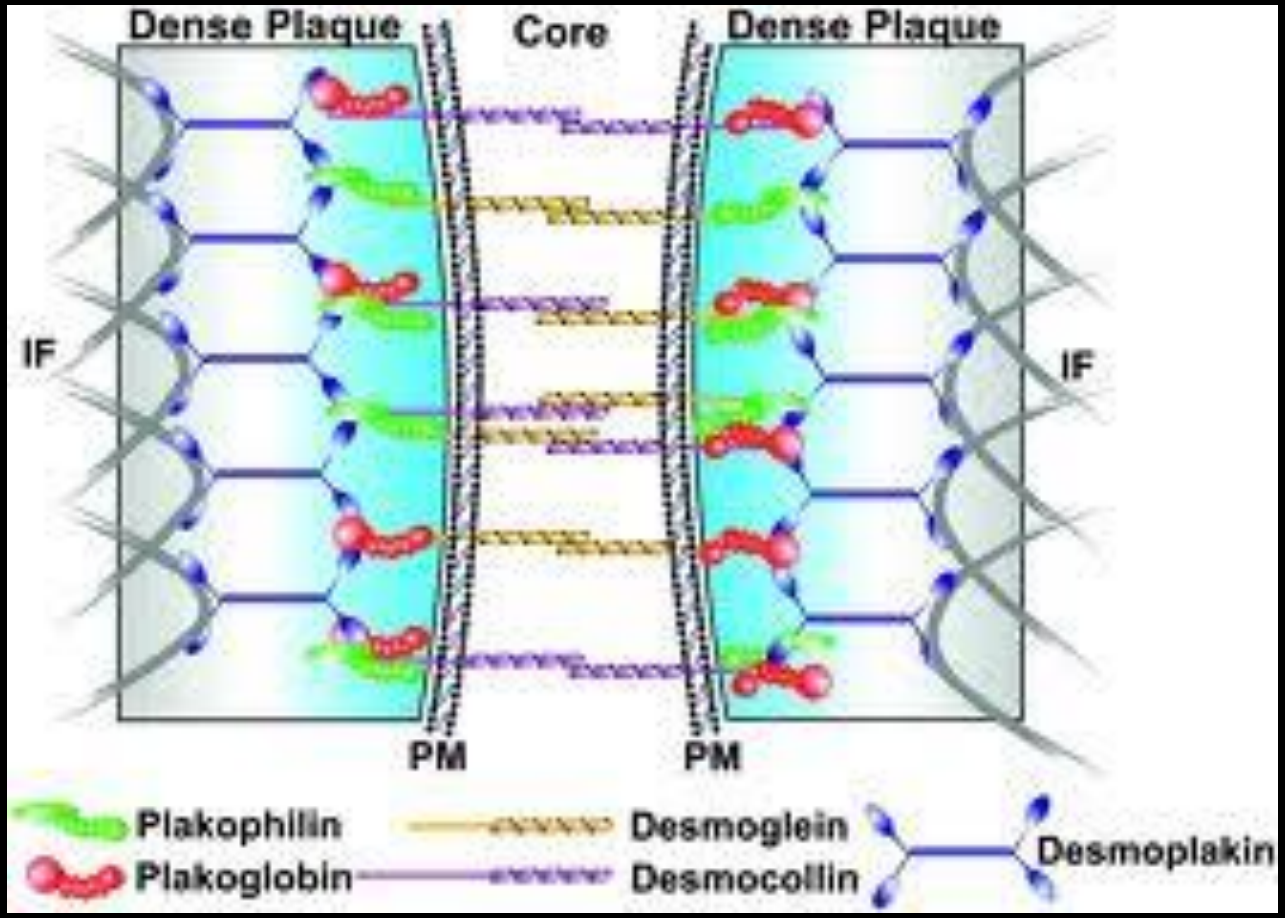
- Epithelium has a complex structure and many protein molecules required for epithelial integrity.
- The oral epithelium keratinocytes adherent to each other by desmosomes and via hemidesmosomes to the basal membrane and the underlying lamina propria and dermis.



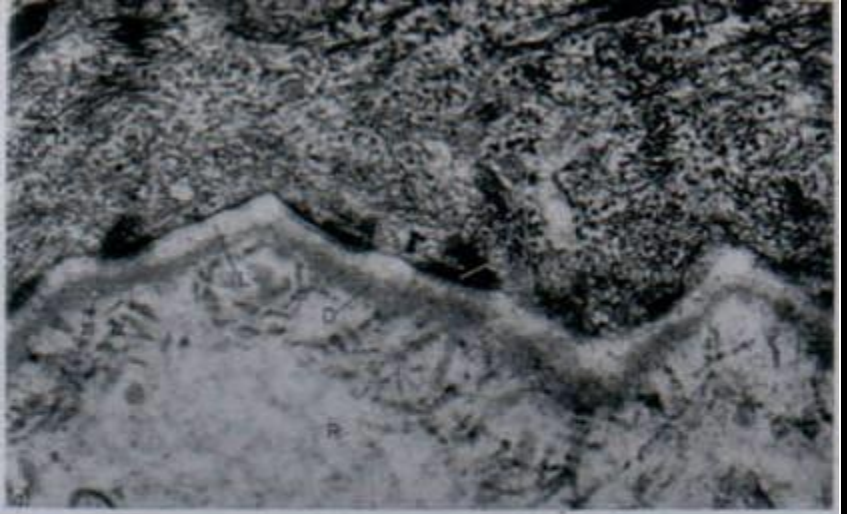

Desmosomes
Tonofilaments

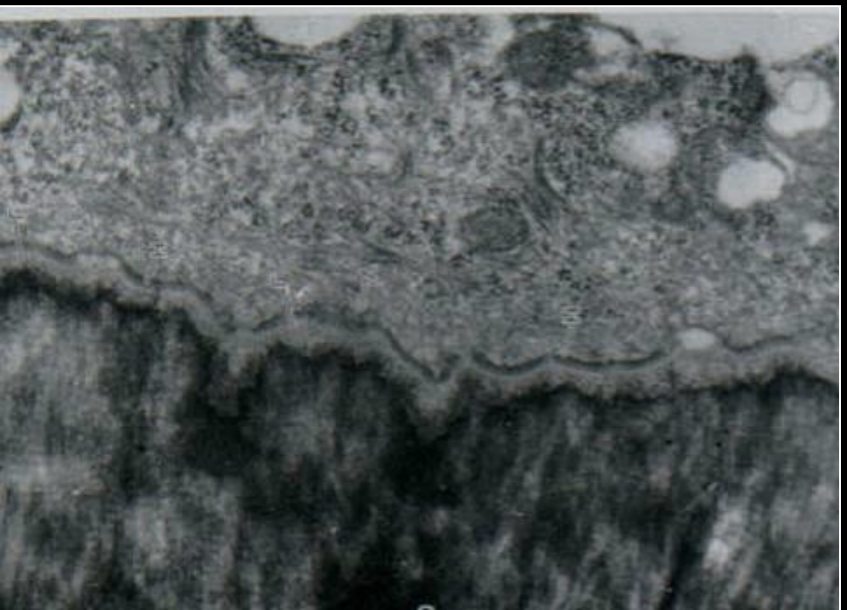
DESMOSOME

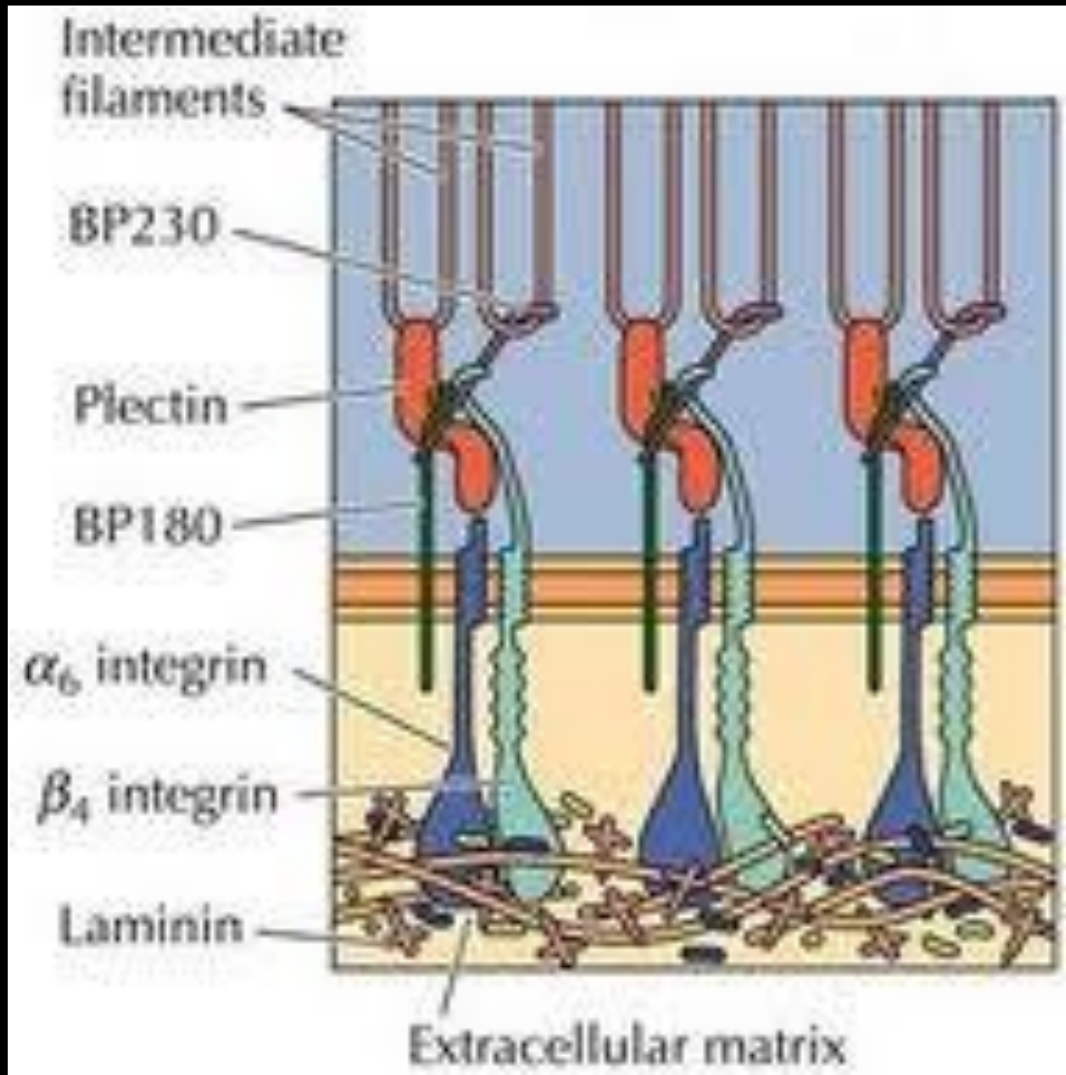

plasma membranes
cytoplasmic plaque
cadherin

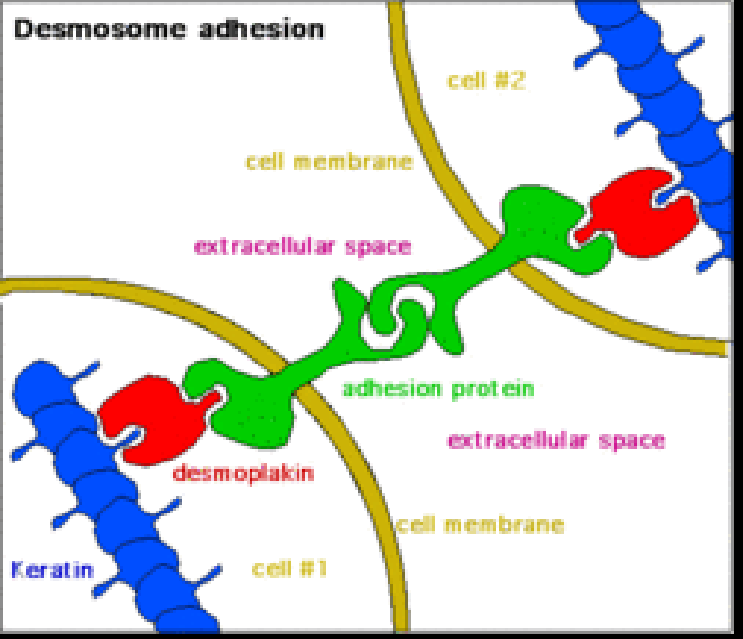

Dense Plaque
Core
Dense Plaque
IF
IF
PM
PM
Plakophilin
Plakoglobin
Desmoglein
Desmocollin
Desmoplakin

Hemidesmosomes

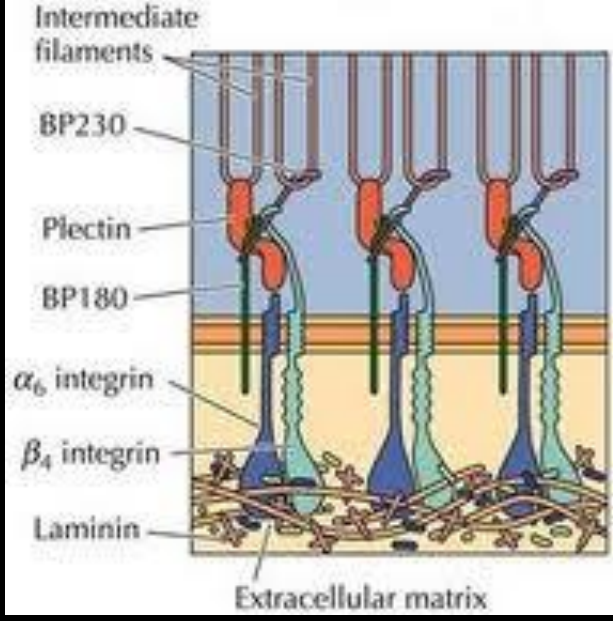


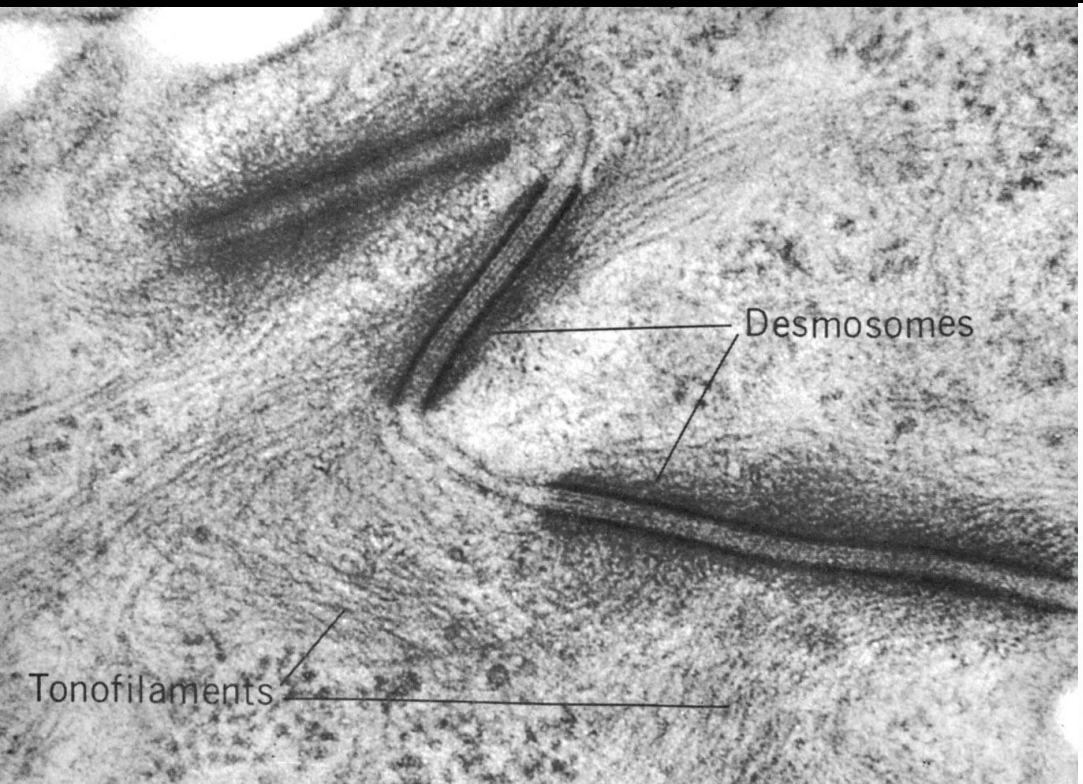


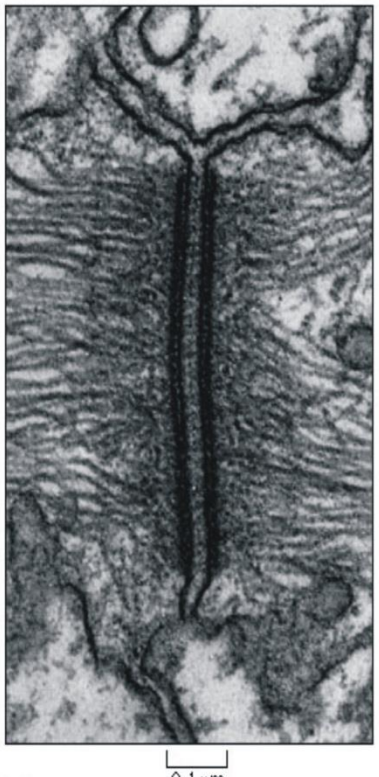


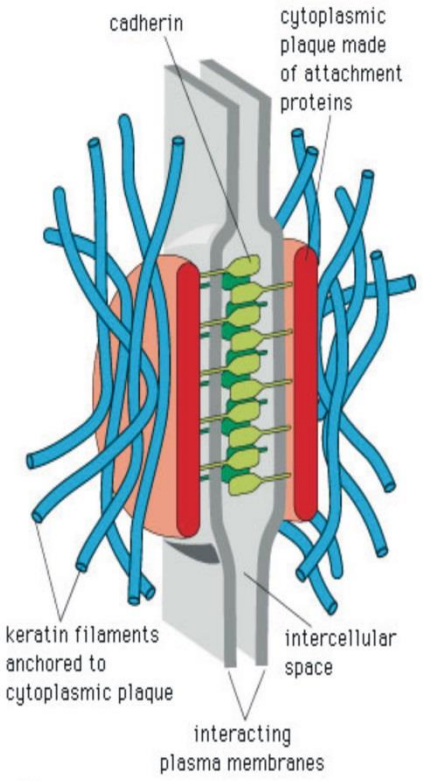


proteins that hold an individual's skin cells together can be attacked by the immune system.



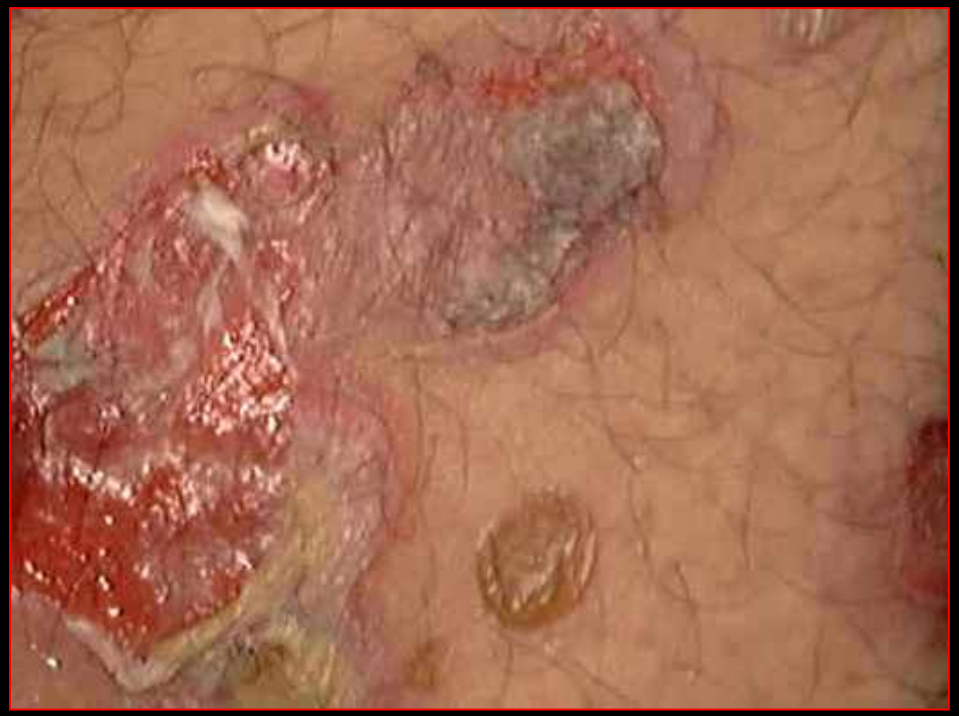






PEMPHIX –BLISTER

- It is a term derived from the Greek language.
- It means **blister or bubble**
- A group of potentially life threatening autoimmune, mucocutan diseases
- characterized by **intraepithelial or subepithelial blistering** affecting the skin and/or mucosal surfaces.
- 0.1-0.5/100 000 people become ill in this disease yearly.



Pemphigus group
Vulgaris (PV)

This is the most frequent form of PV on the skin and mucous membranes

causing blisters, ulcers or erosions on the skin and mucous surfaces.

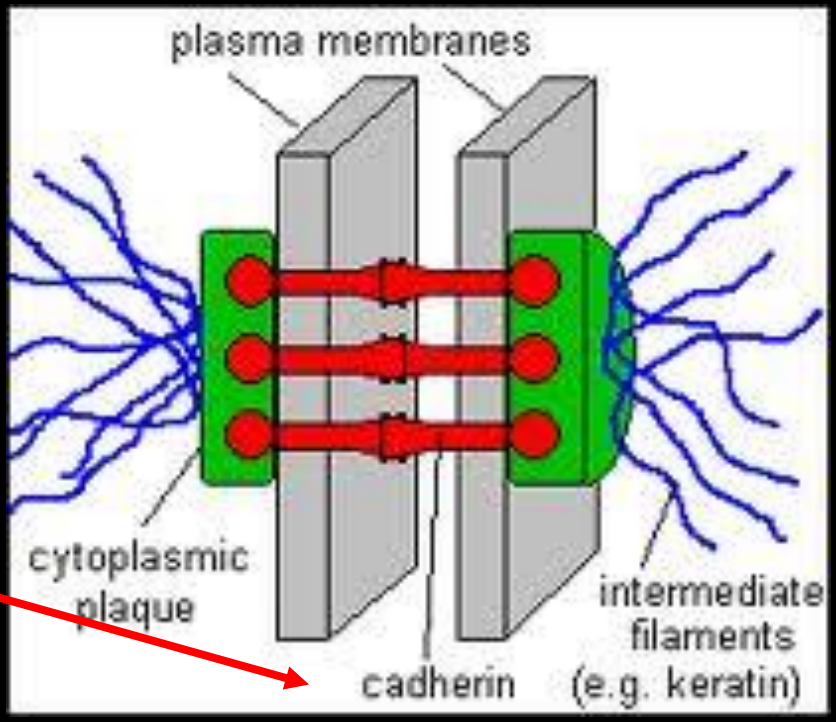
The IgG type autoantibody is produced against

CADHERIN

an adhesion molecule.

Desmoglein1 in the skin

Desmoglein3 (Dsg) in the mucous membrane.



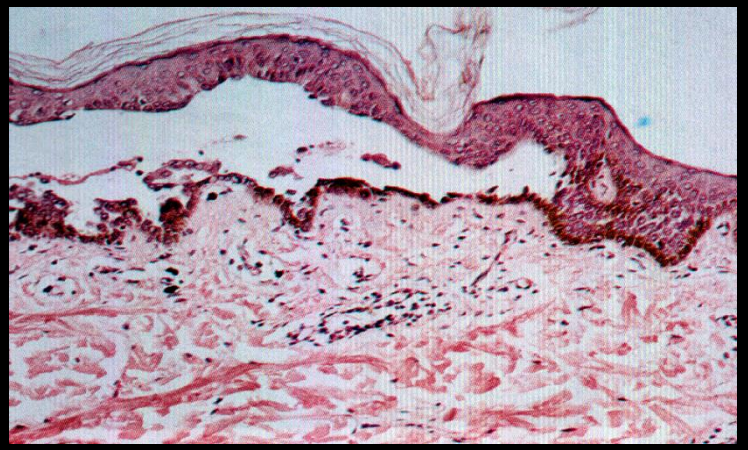
Oral mucosal symptoms

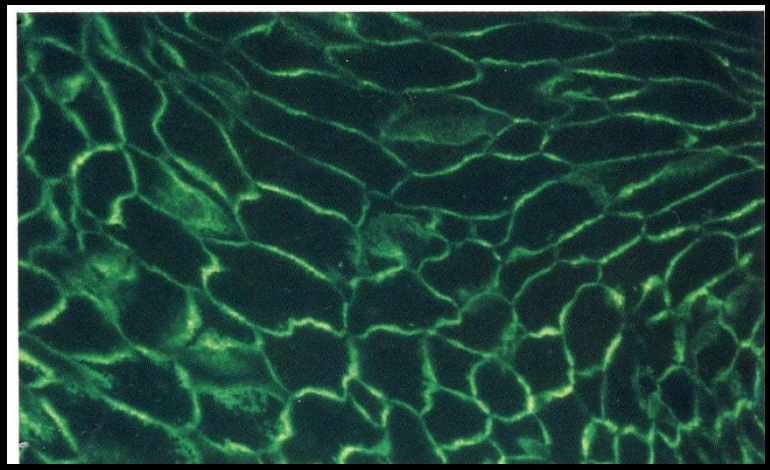
Dsg3

Damage of the intercellular area leads to the separation of keratinocytes, which is the so-called

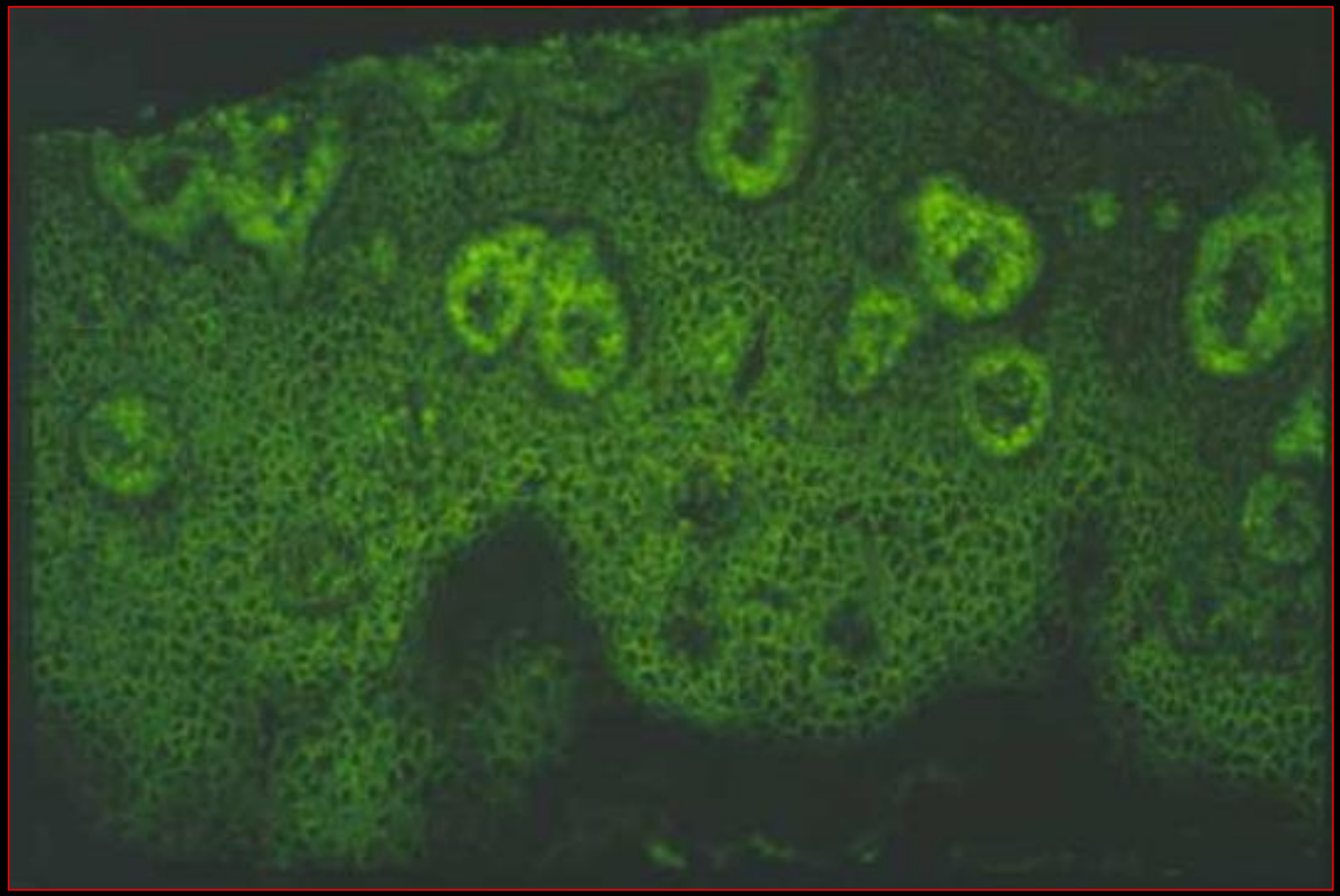
ACANTHOLYSIS

typical in Pemphigus.





Direct immonofluorescent techniques indicating the auto IgG



GENETIC background

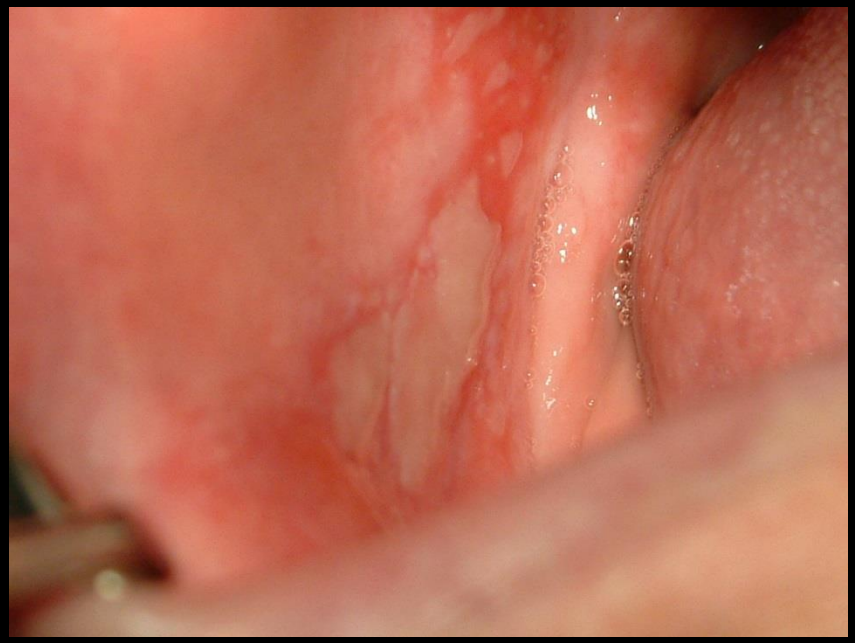
Strong genetic background of PV

certain ethnic groups

Ashkenazi Jews

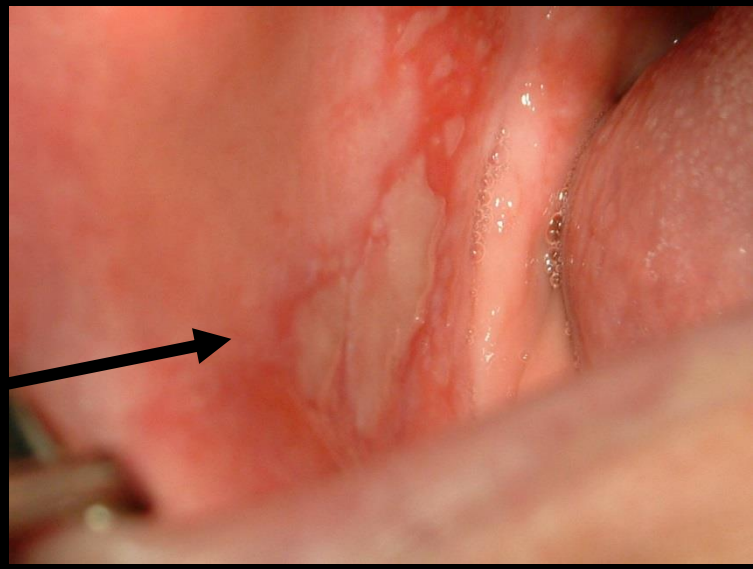
South Asian

Mediterranean origin.



ORAL MANIFESTATIONS OF AUTOIMMUNE BLISTERING DISEASES

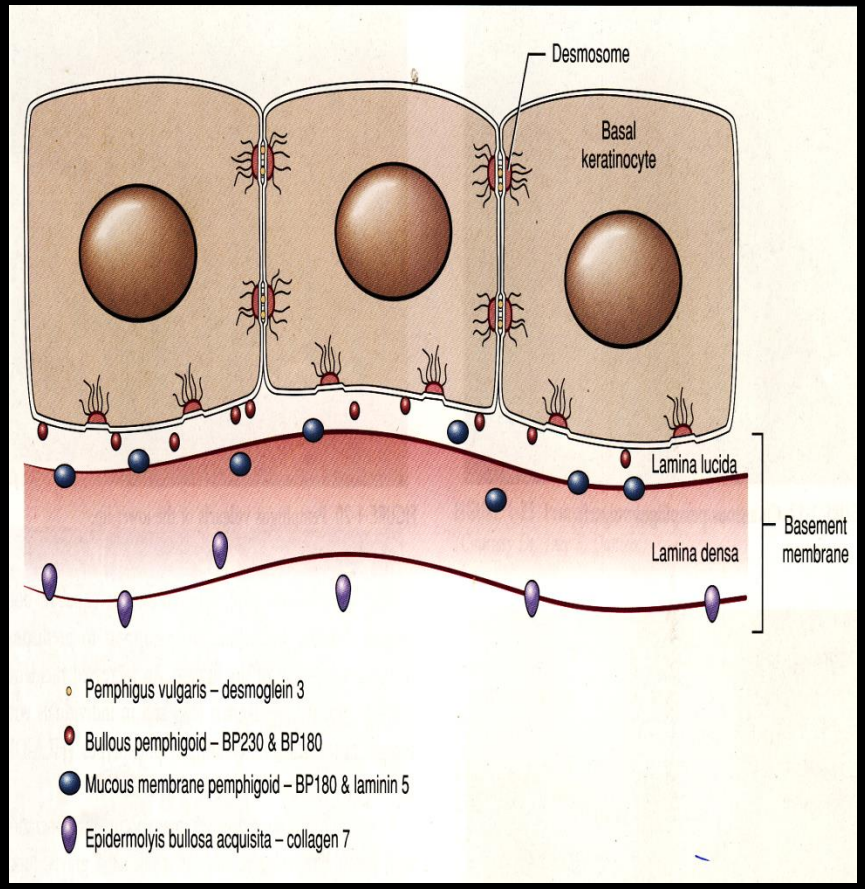
- It can affect any area of the oral cavity: gingiva, palate, buccal, tongue, floor of the mouth, and pharynx.
- Blisters are broken easily; they rarely are observed clinically.
- Instead, erosions and superficial ulcers more likely are observed.
-



Pathogenesis

antibodies are deposited intercellularly directed against the extracellular domains of **Dsg3,** by this way **oral lesions** appear at an early stage.

Development **of Dsg1 autoantibody** in Pemphigus Vulgaris correlates with the disease progression involvement of **skin** and mucosa other than oral.



Blisters in pemphigus vulgaris are associated with the binding of IgG autoantibodies to keratinocyte cell surface molecules.

- Antibodies bind to keratinocyte desmosomes and to desmosome-free areas of the keratinocyte cell membrane.
- The binding of autoantibodies results in a loss of cell-to-cell adhesion, acantholysis
- The antibody alone is capable of causing blistering without complement or inflammatory cells.

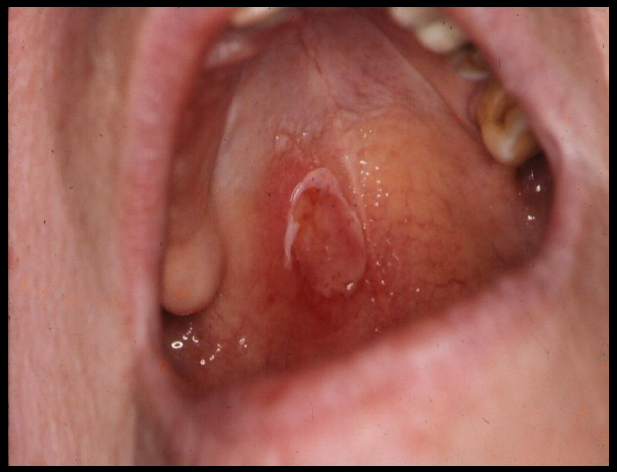
Pemphigus in the oral cavity Diagnosis and Treatment

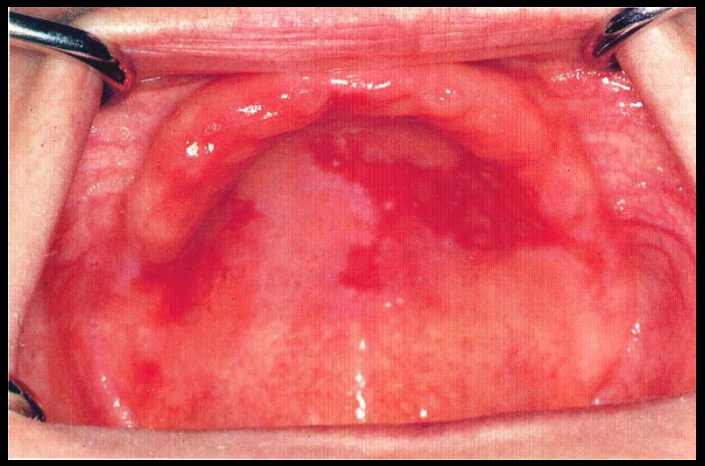
It is crucial to establish the diagnosis of P.V. as early as possible

adequate treatment should be commended.

Full history, biopsy, histopathological and immunological investigations are indicated.

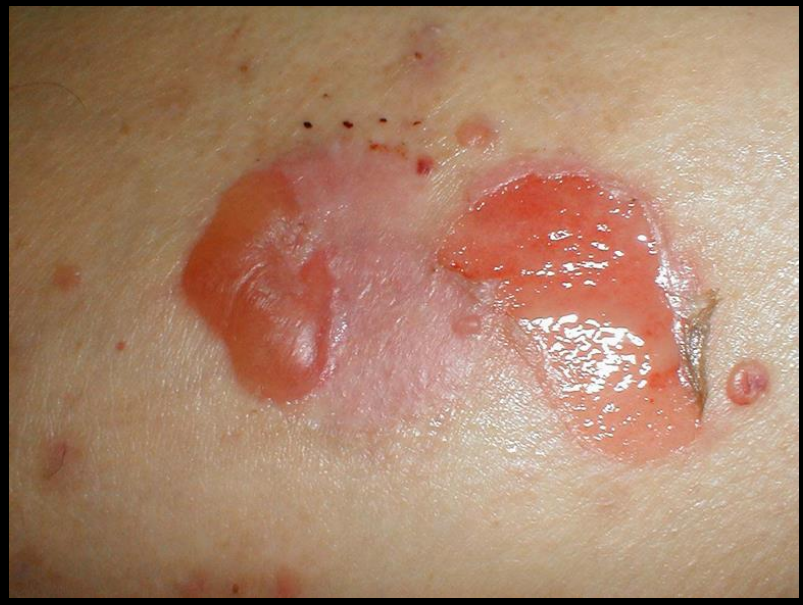
Biopsy of the perilesional tissue is essential to the diagnosis.

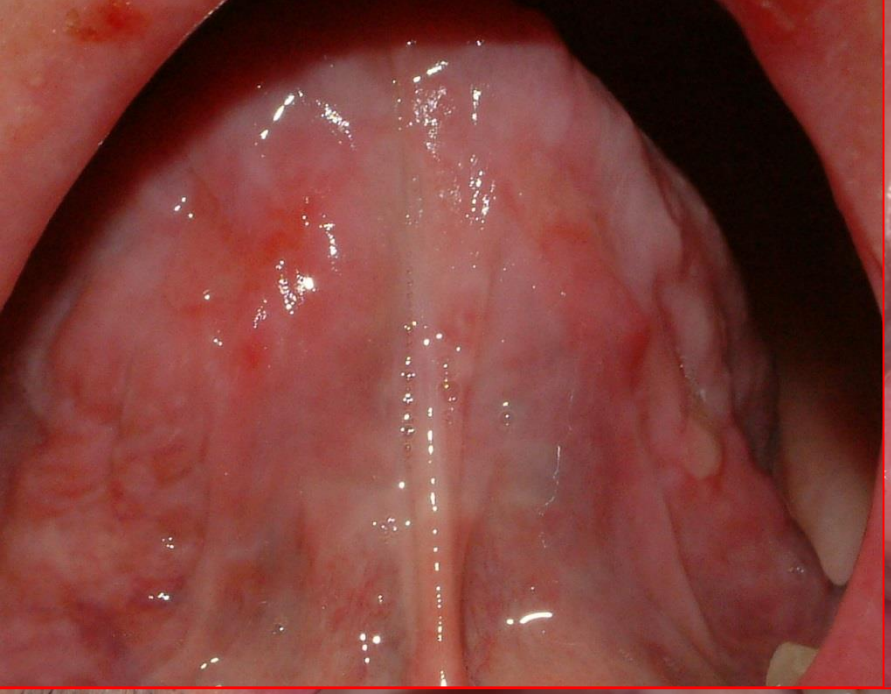


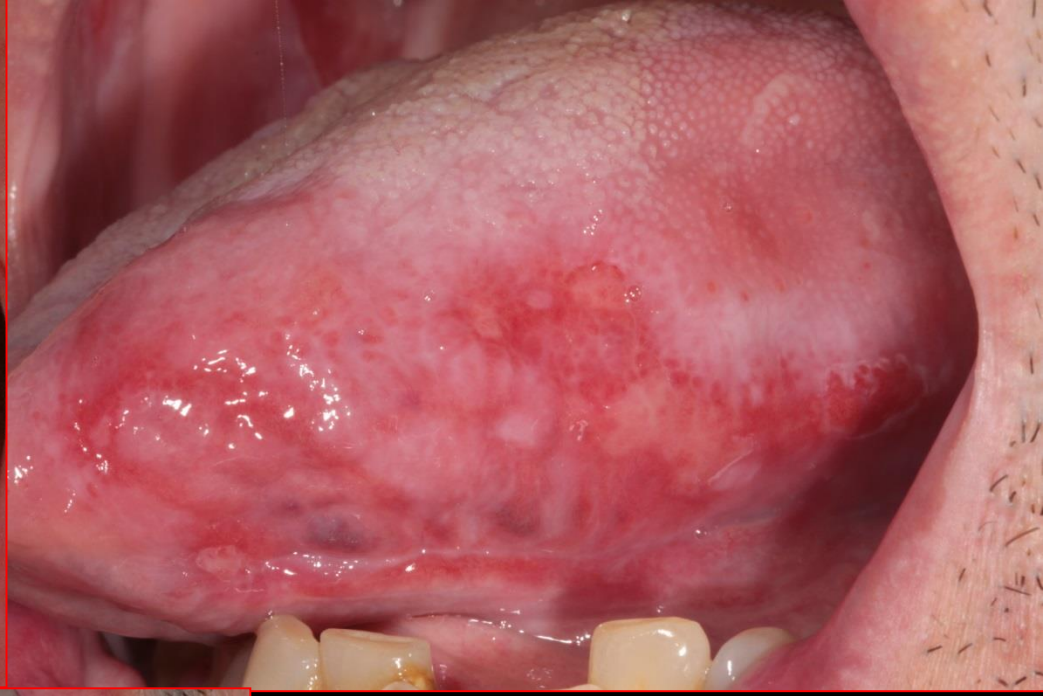


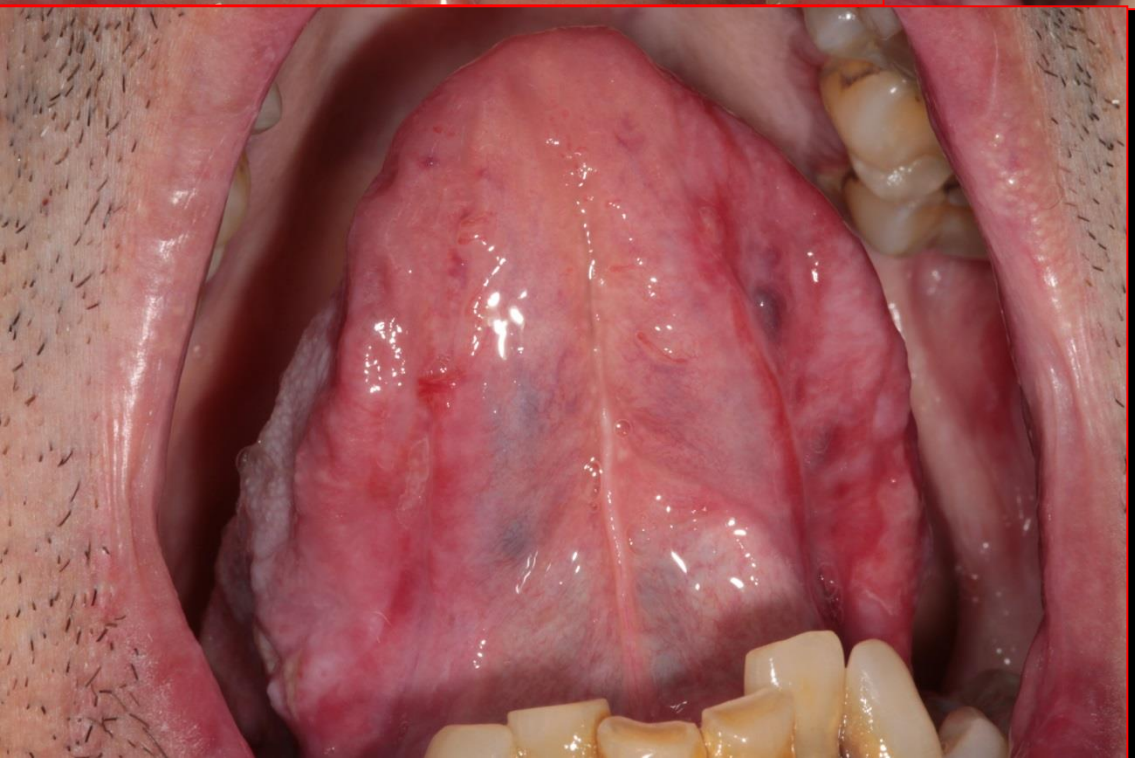
Clinical skin diagnosis

- **Nikolsky sign:** firm sliding pressure with a finger separates normal-appearing epidermis, producing an erosion. .
- **Asboe-Hansen sign:** Lateral pressure on the edge of a blister can spread the blister into clinically unaffected skin.

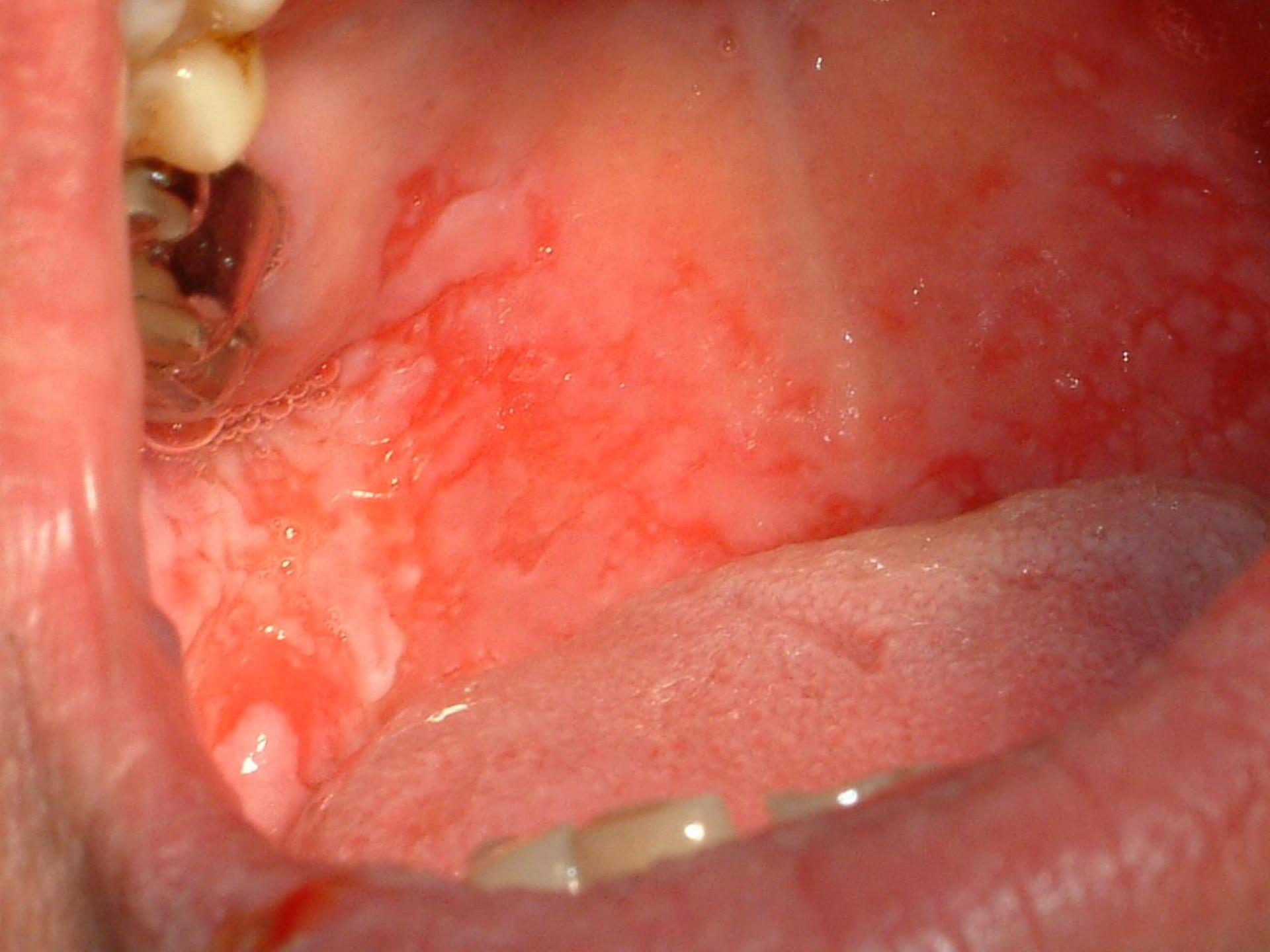


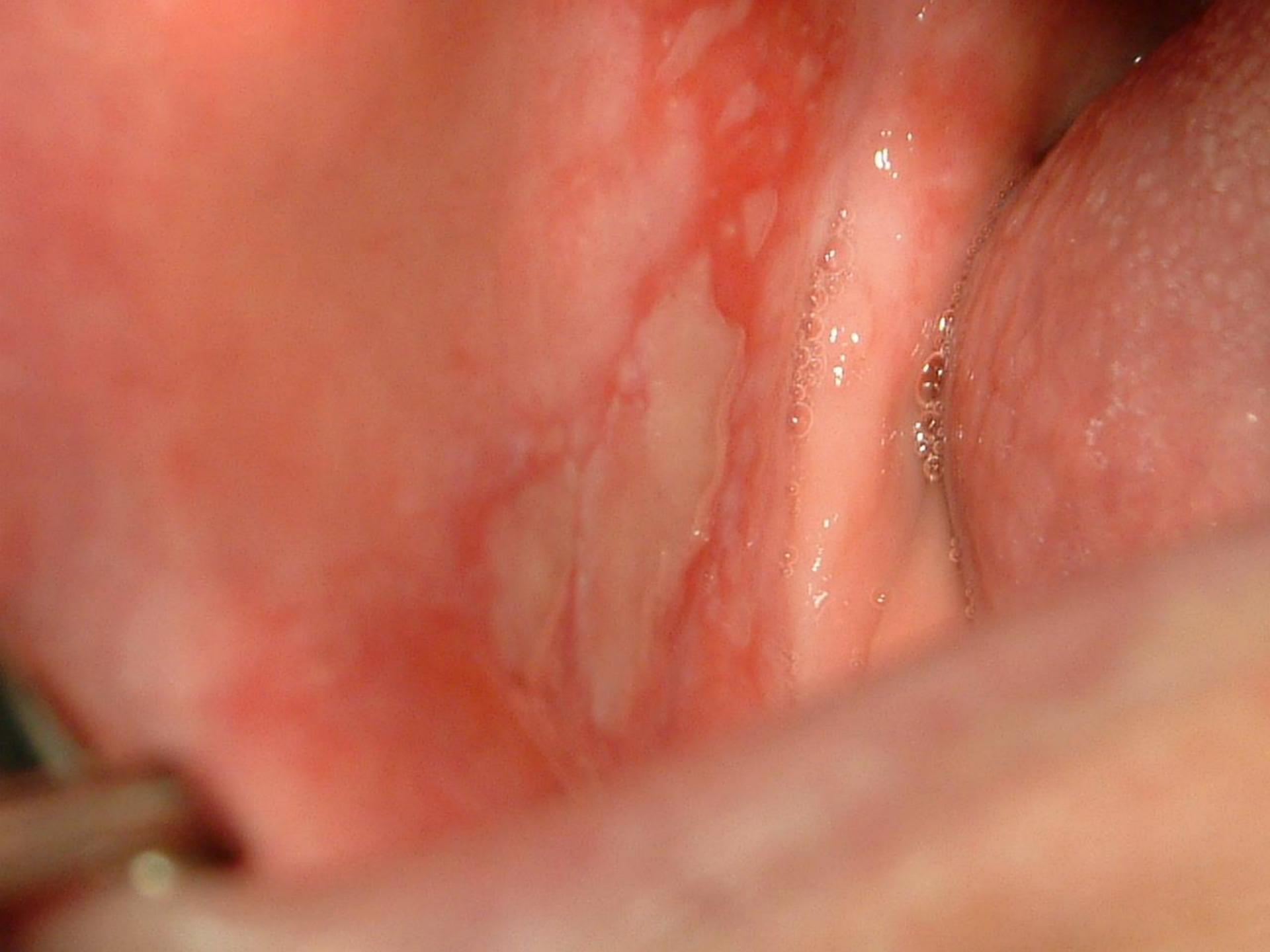


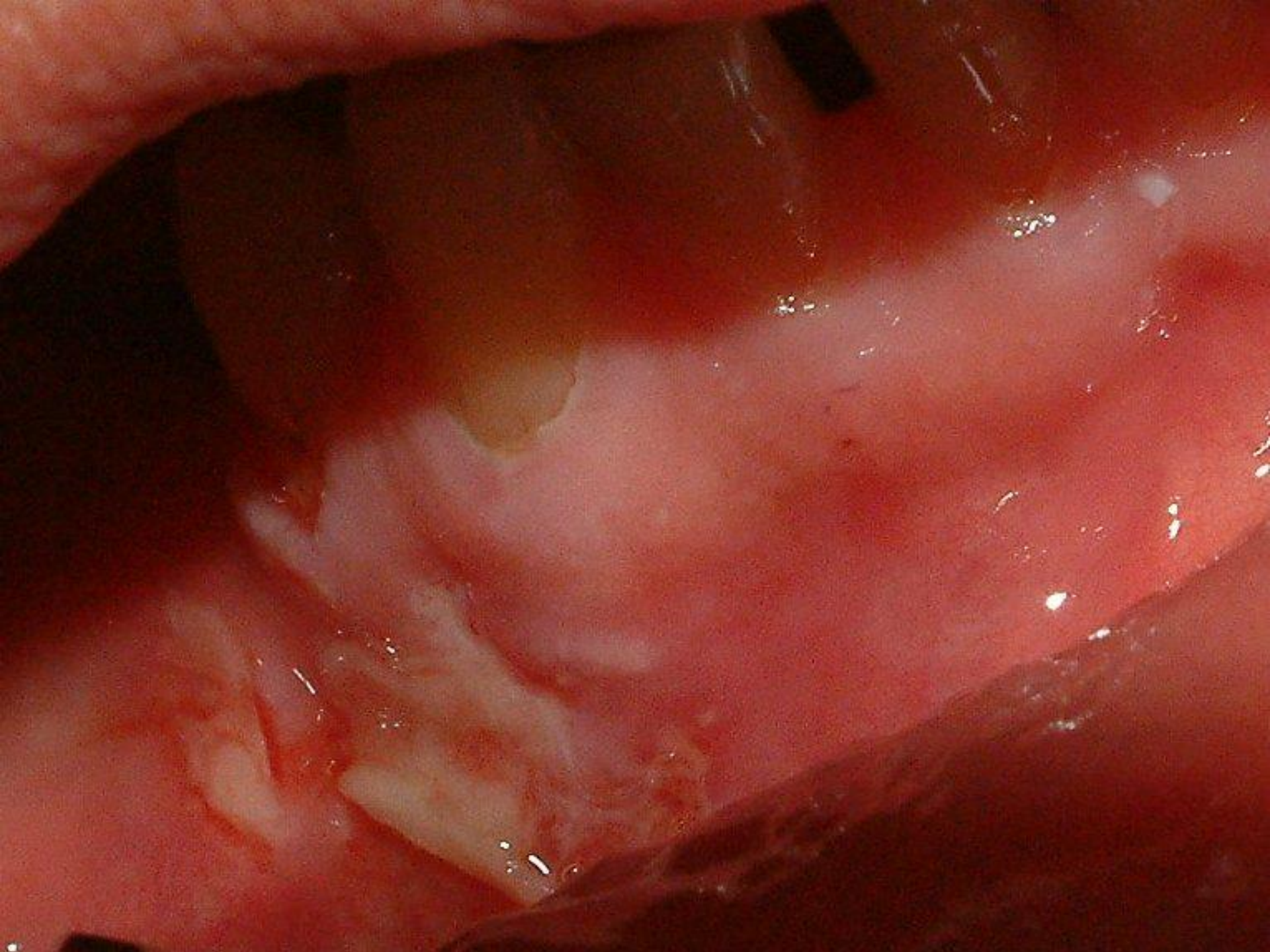


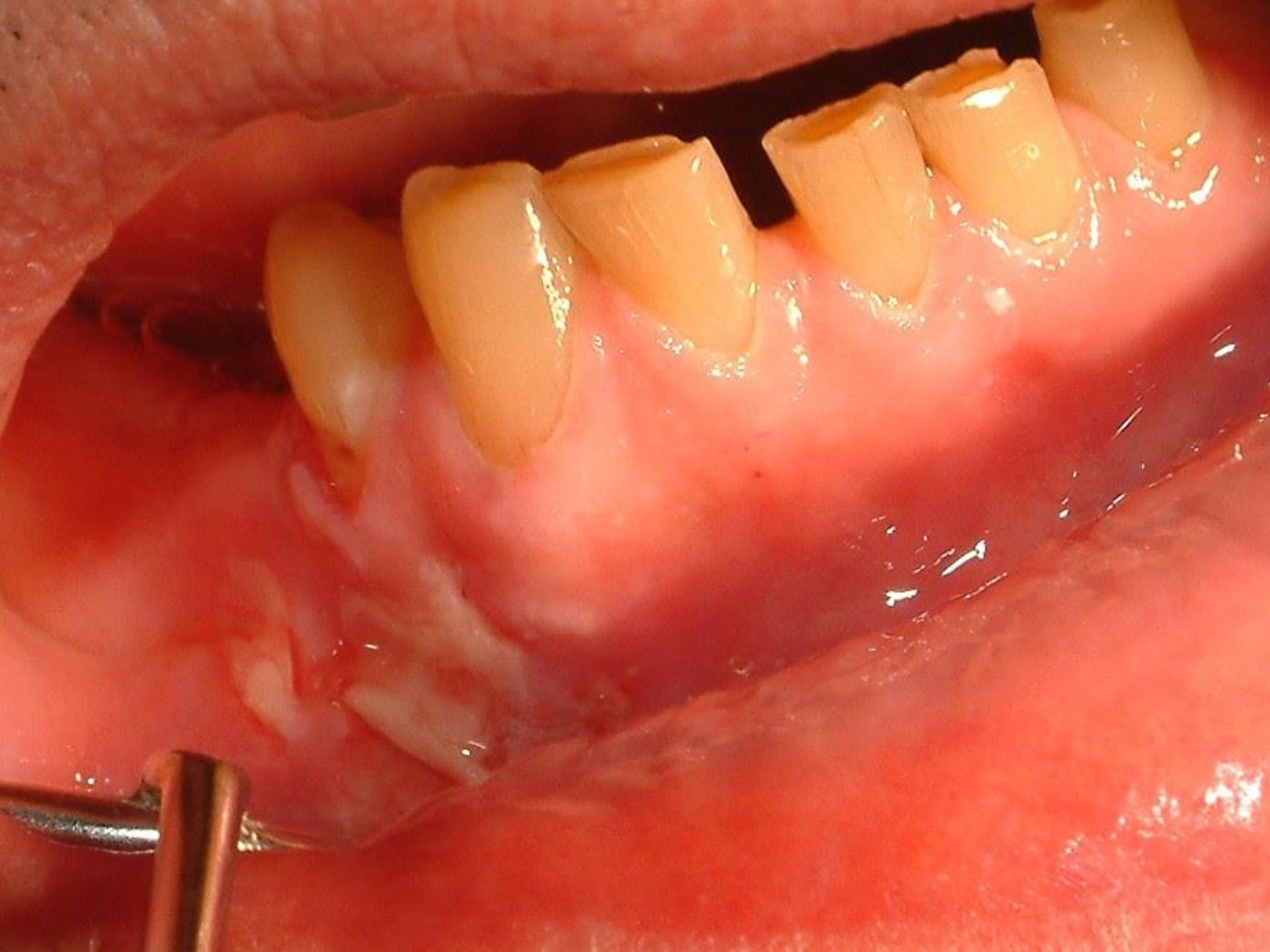


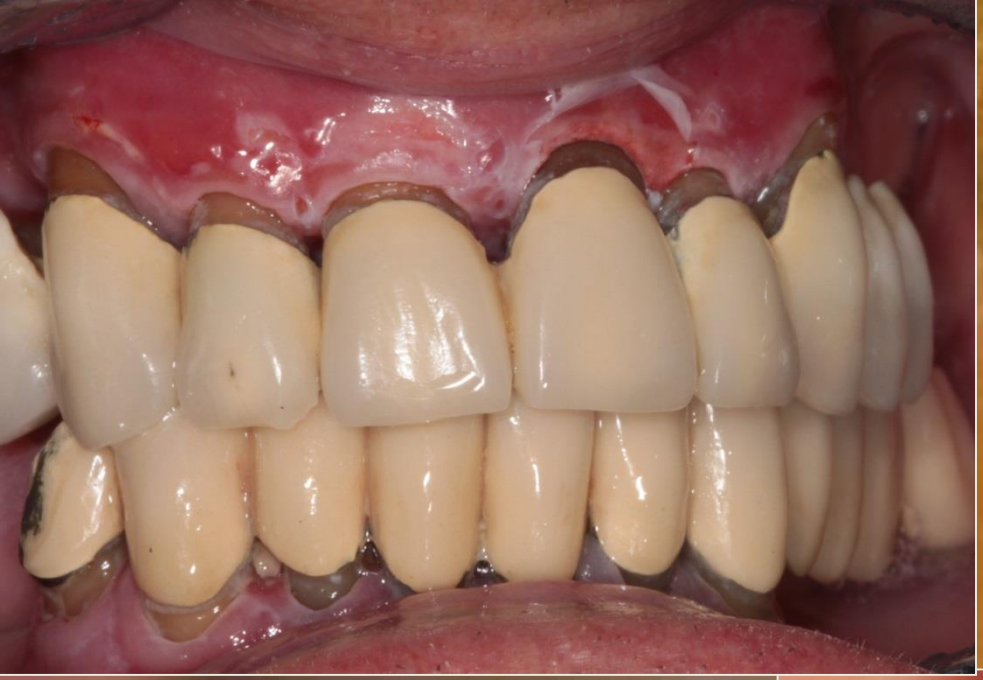
Pemphigus Vulgaris

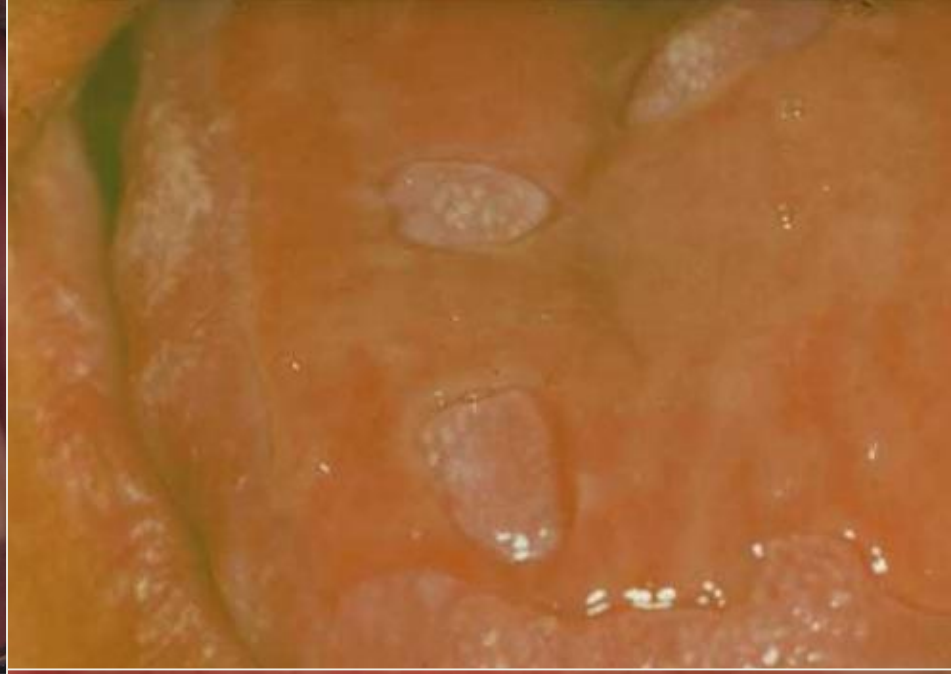


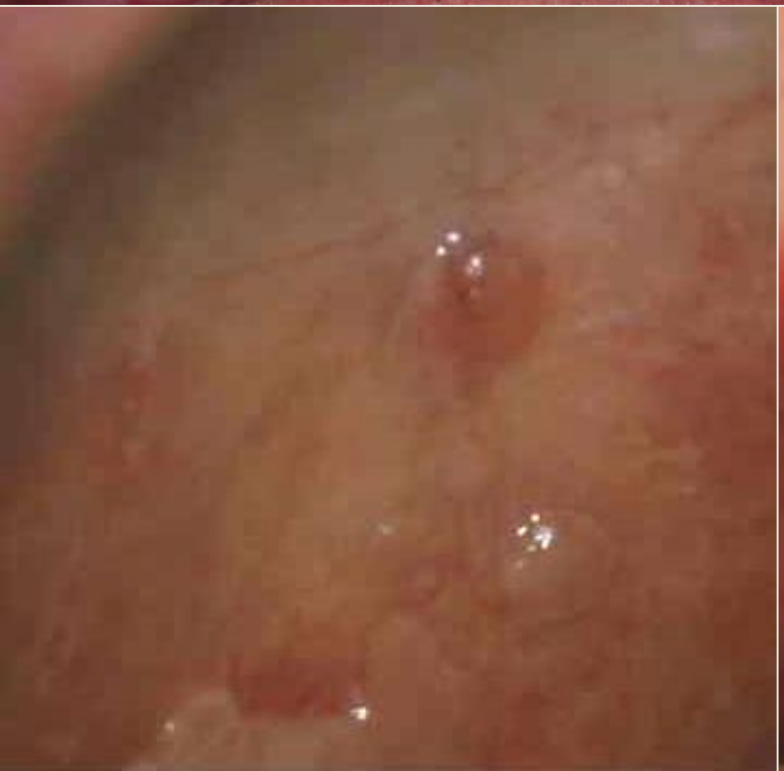


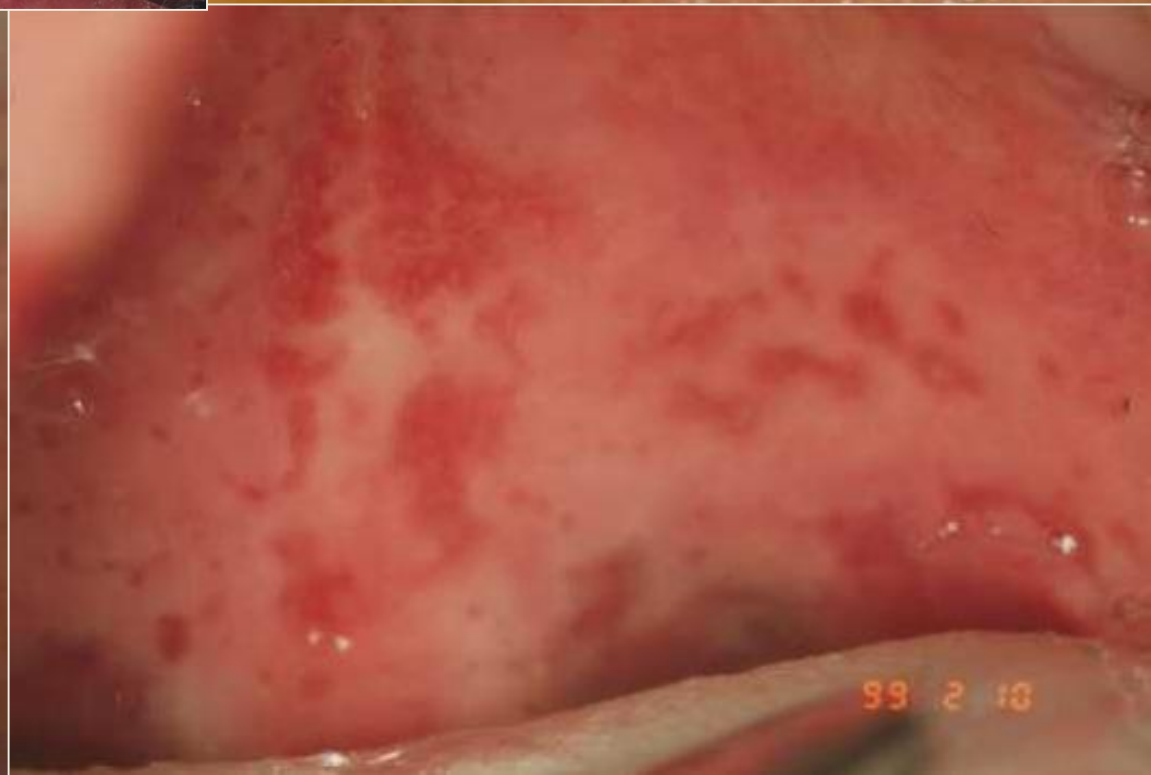








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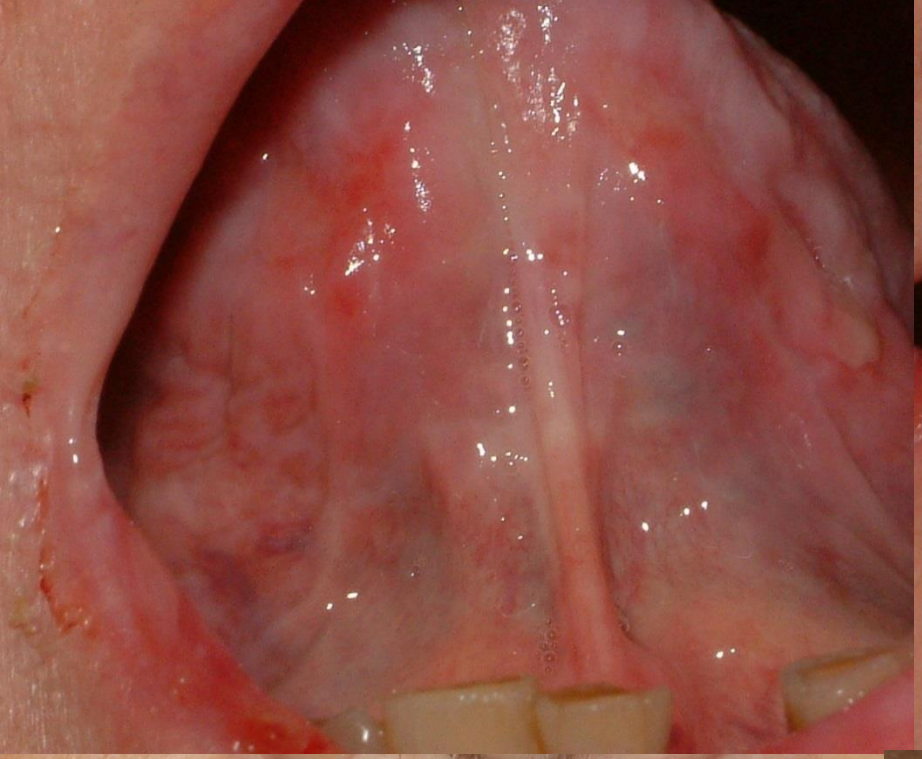
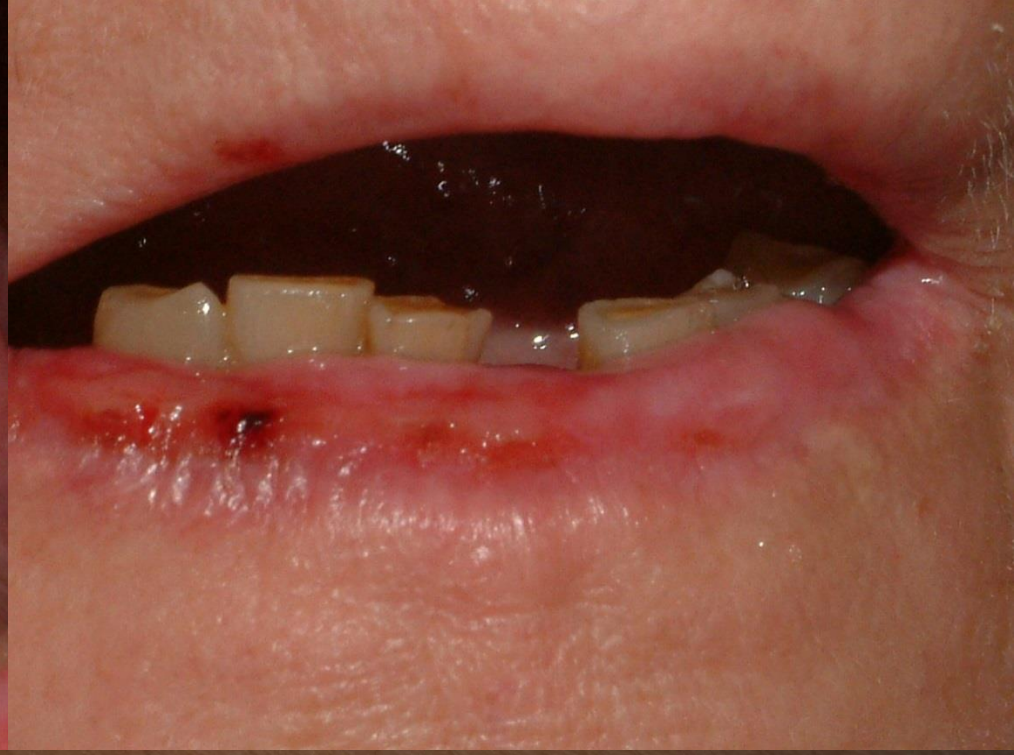
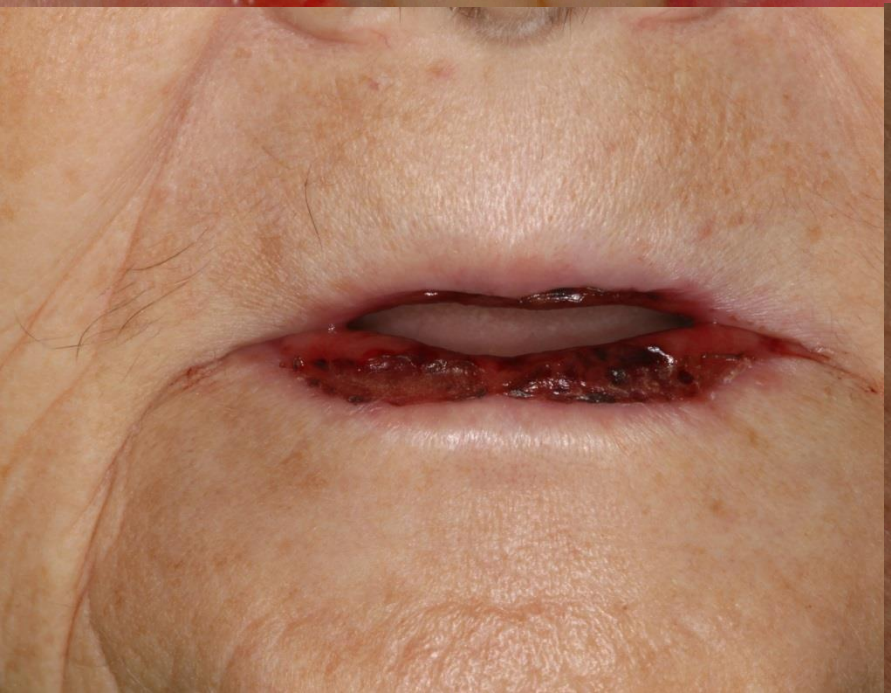
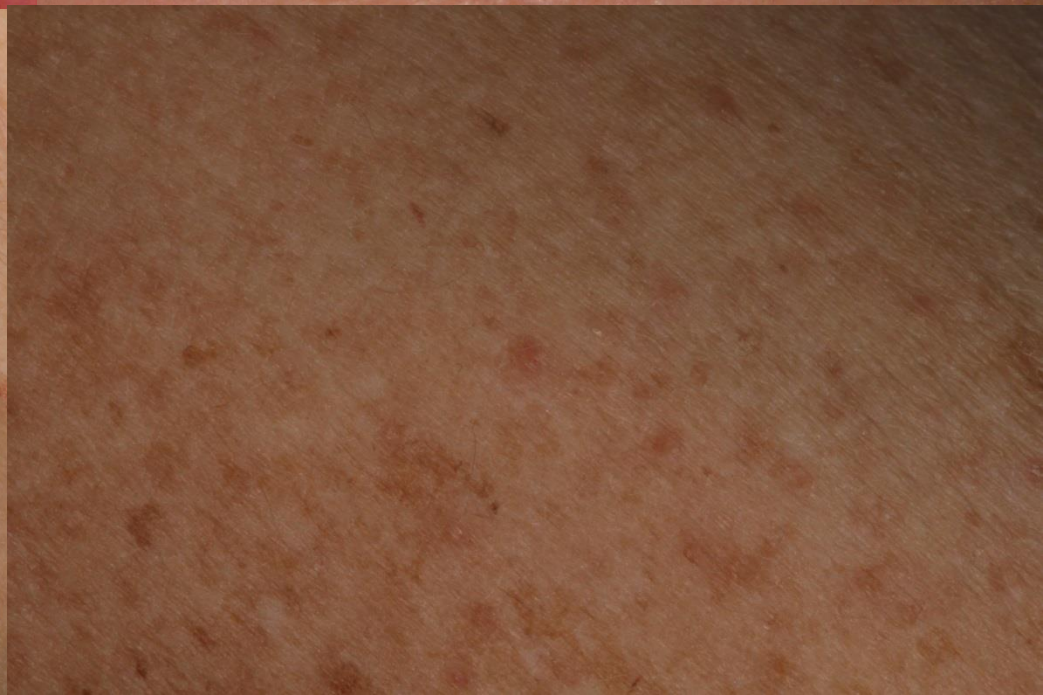
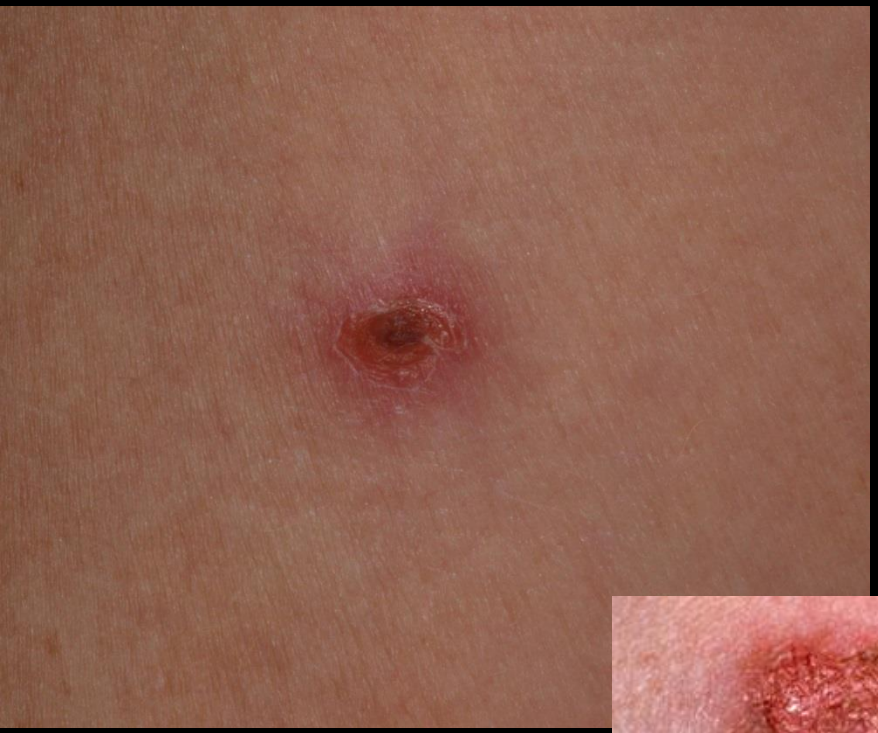
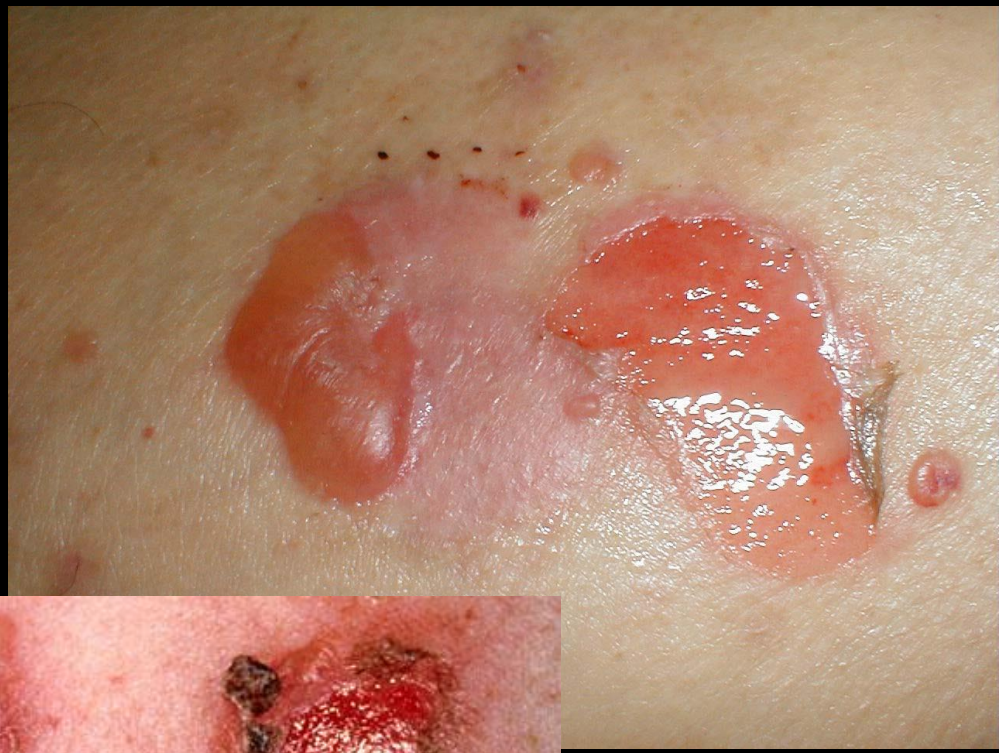
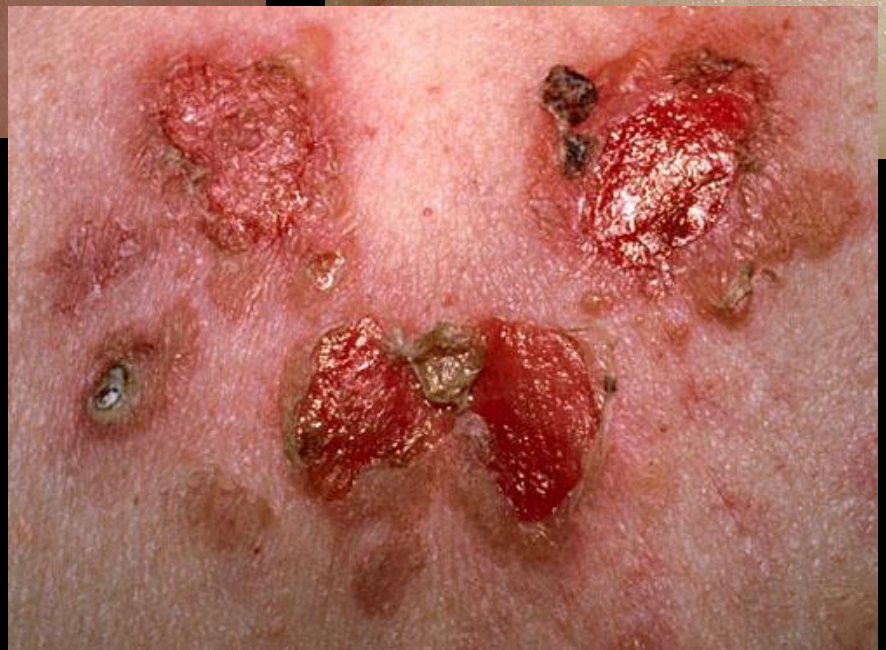





Pemphigus on the skin



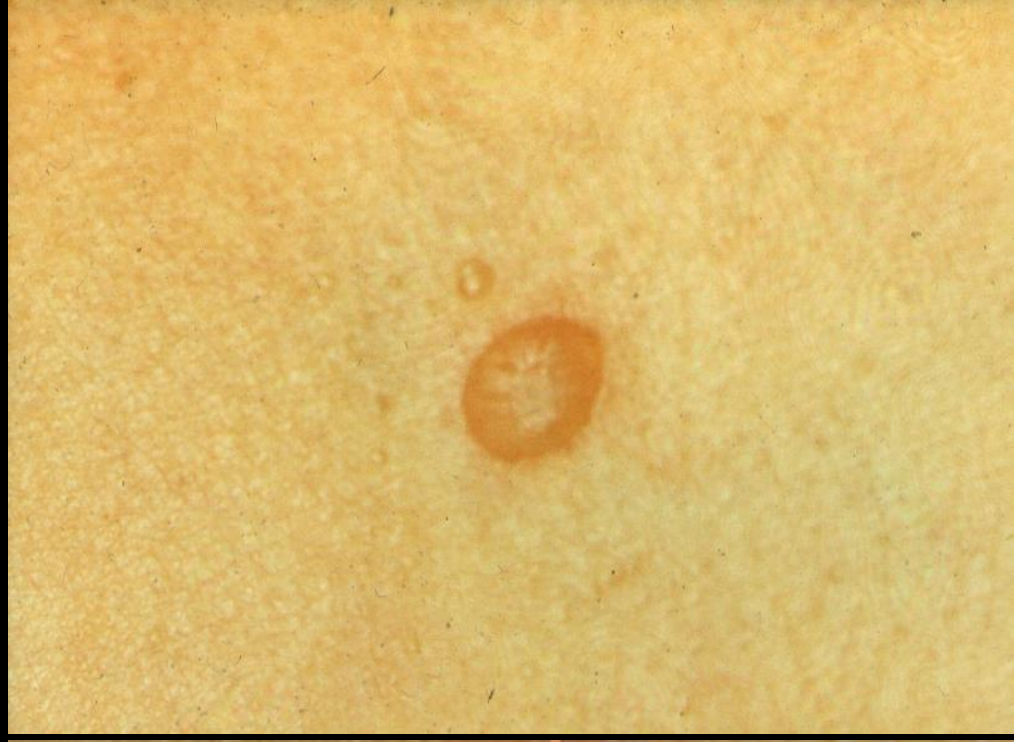


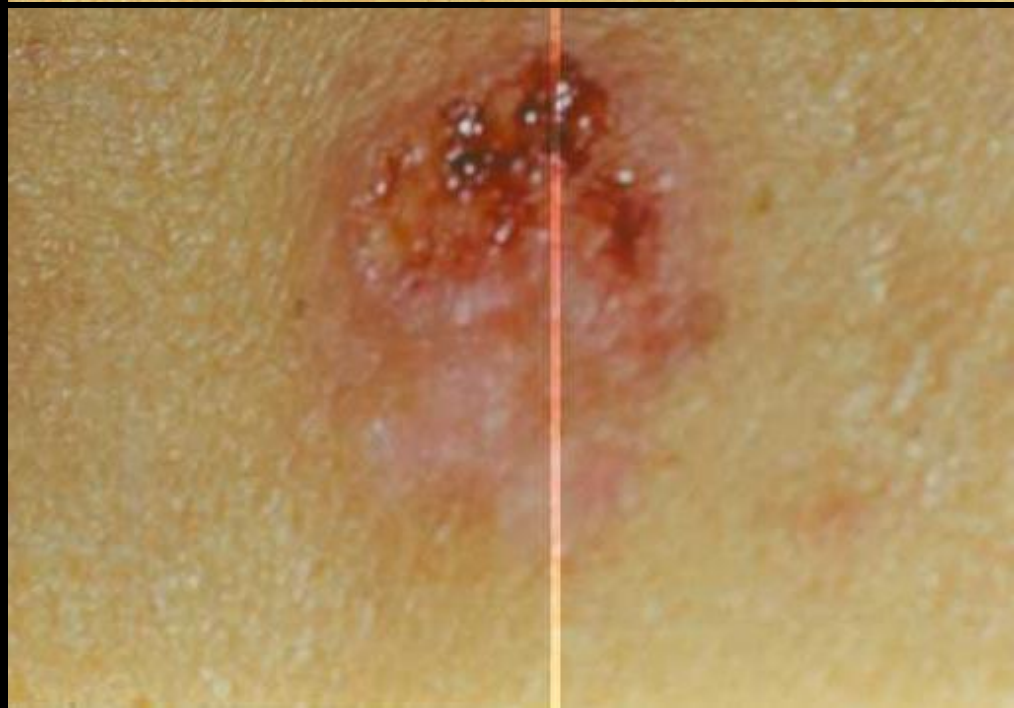


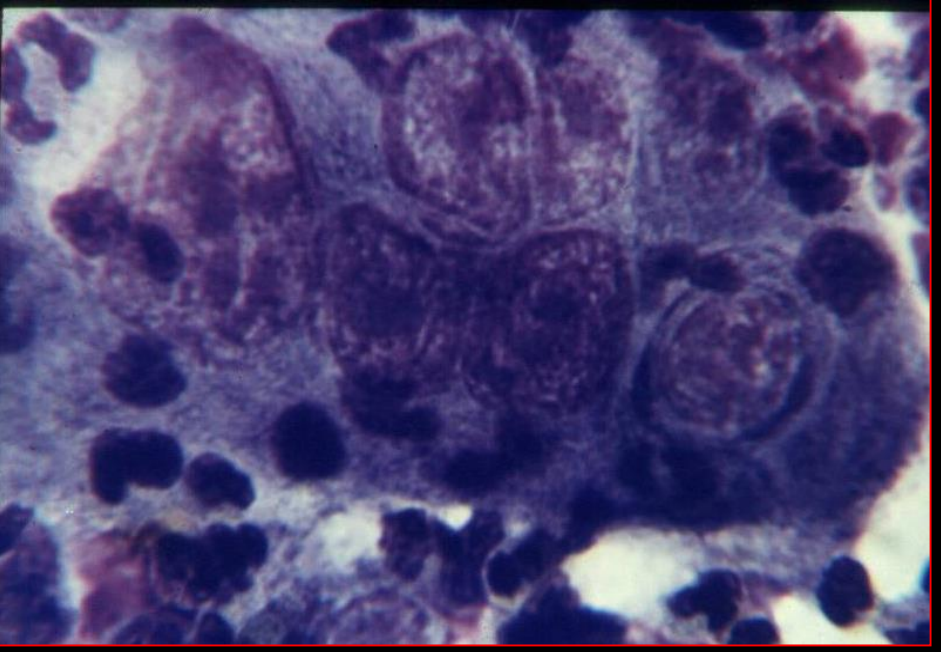
Skin lesions

Primary skin blister

Secondary erosion on the skin



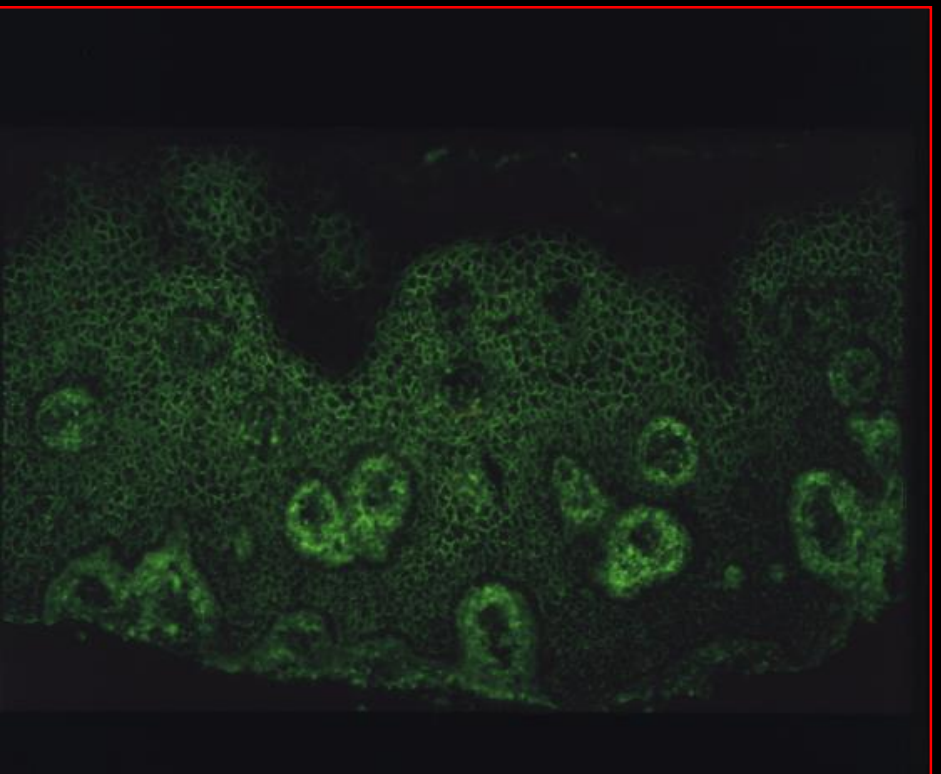




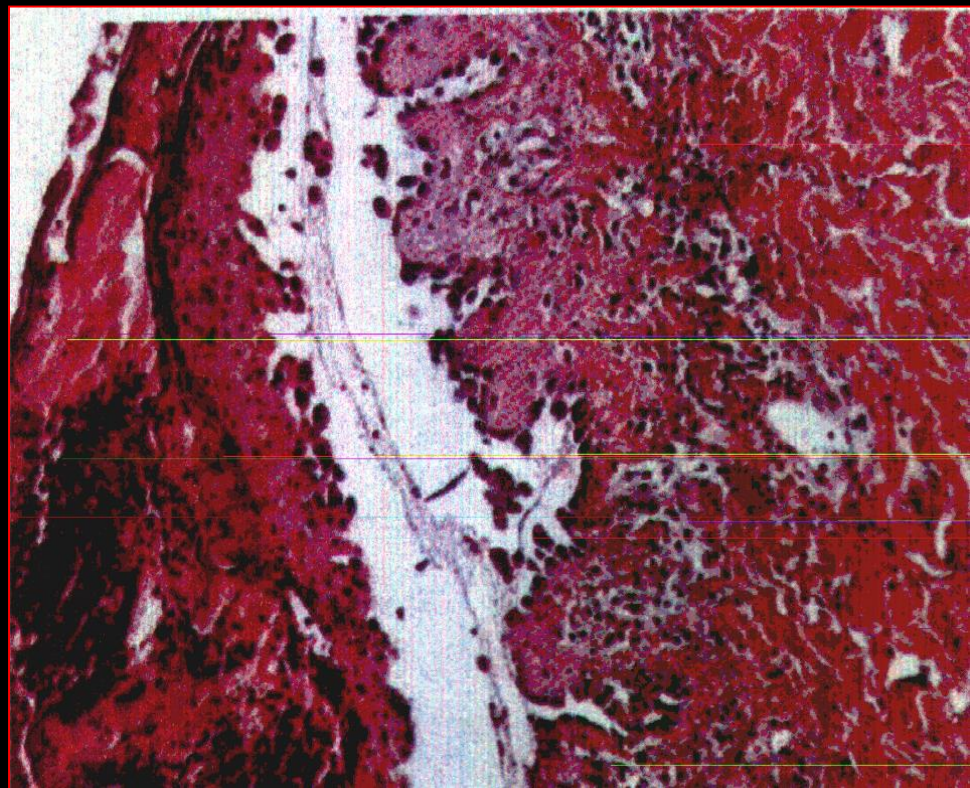
Tzanck cells

Prognostic sign to measure the level of anti Dsg1 and Dsg3 IgG

DIF

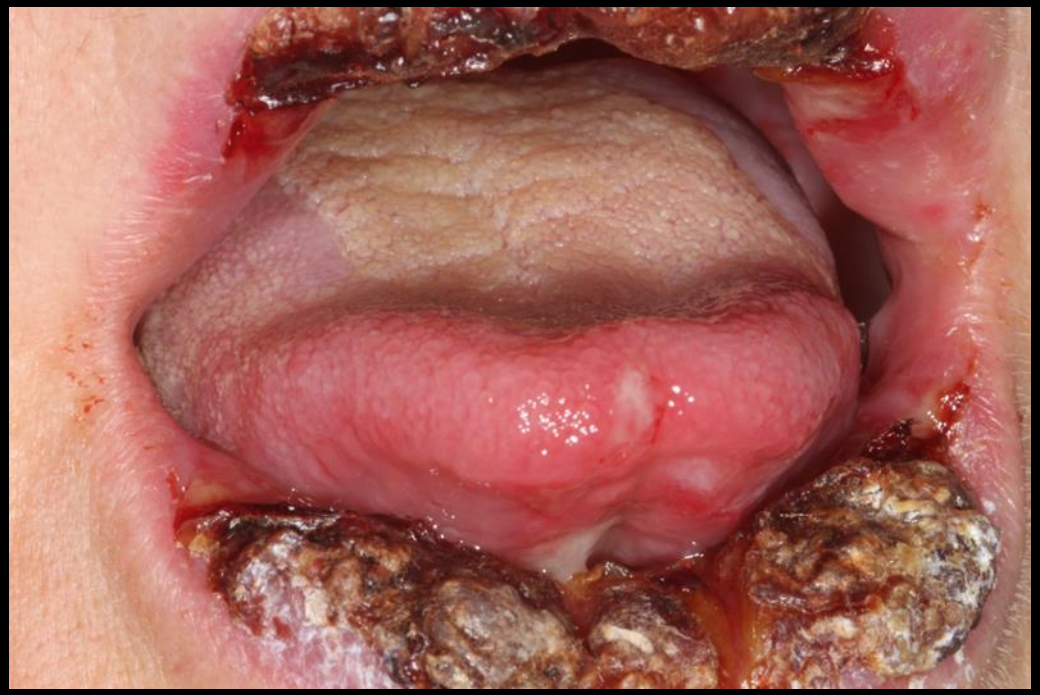


HE

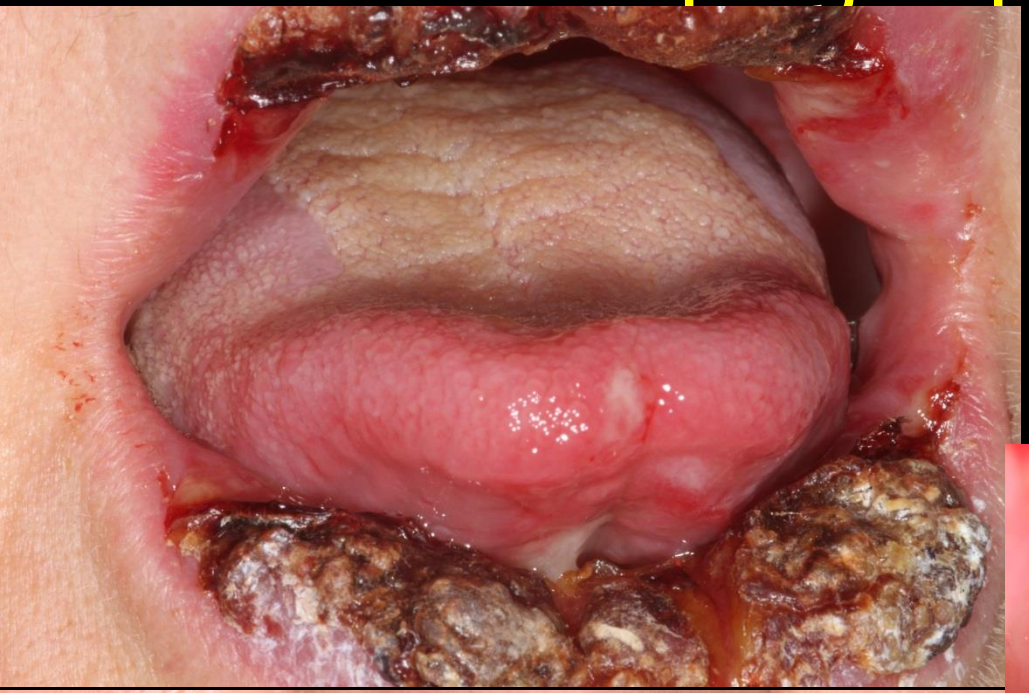


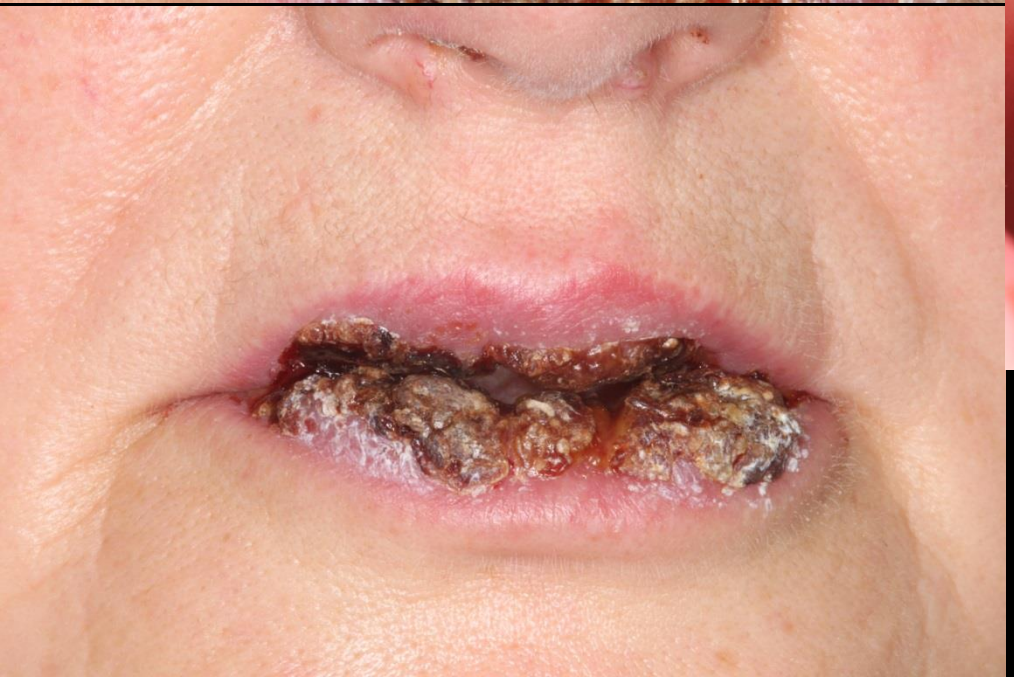
Pemphigus foliaceus is predominantly a skin disease.

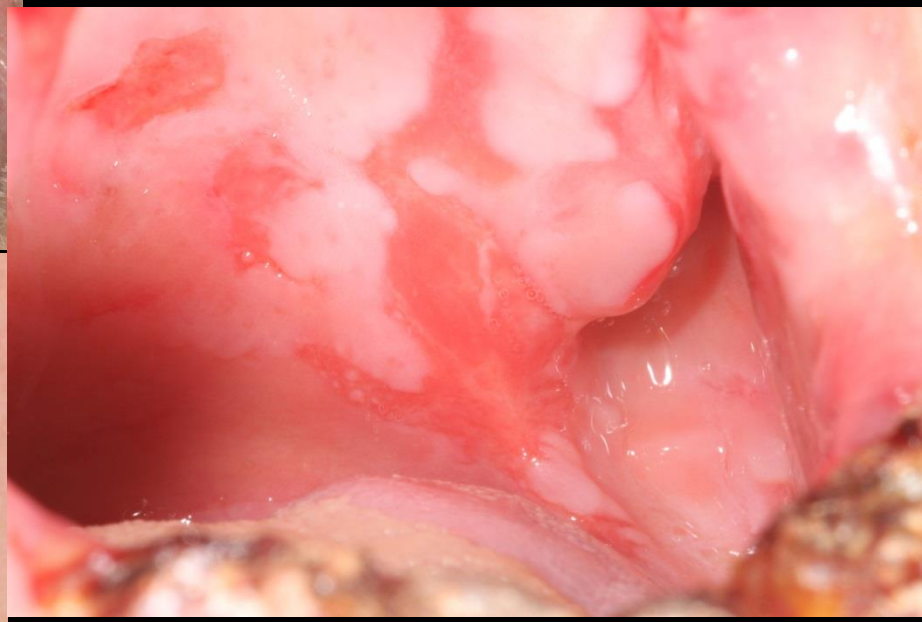
- Oral or other mucous membrane involvements are very rare.
- autoantibodies in pemphigus foliaceus, exclusively target desmoglein-1,

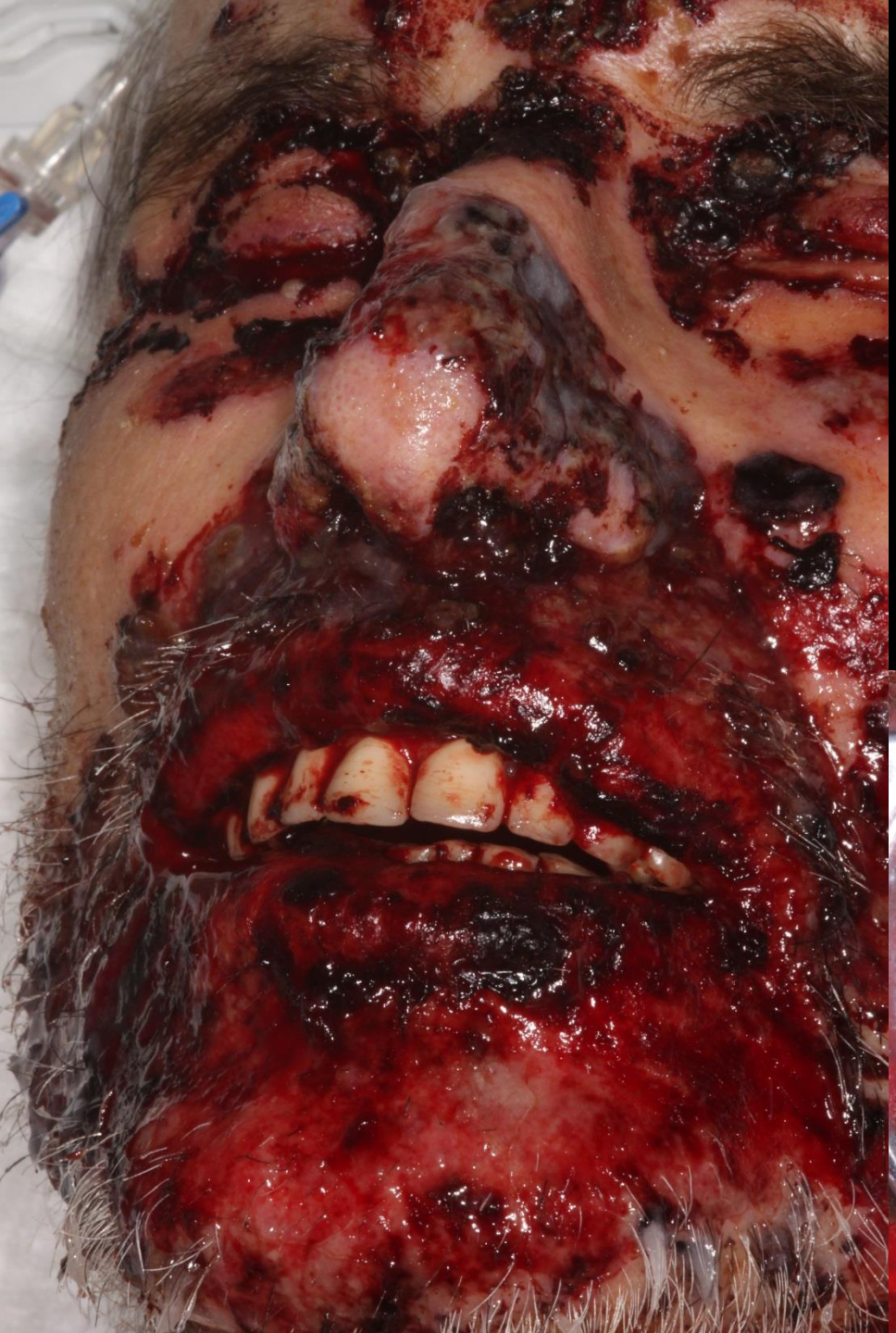


Pemphigus pholiaceus

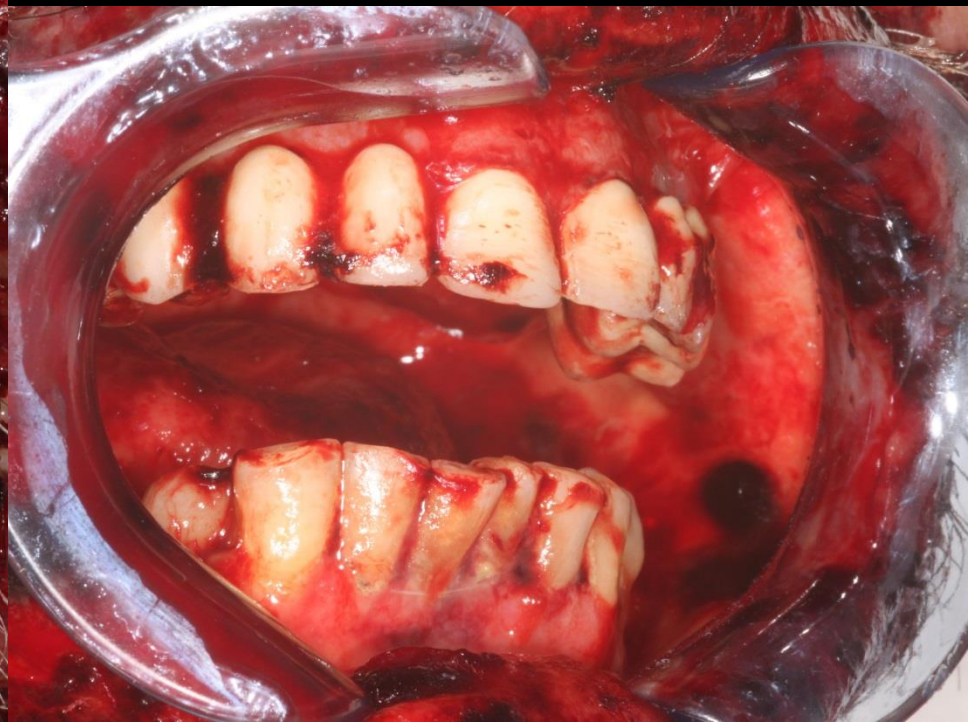








Paraneoplastic Pemphigus associated with lymphoproliferatíve diseases or cancer



Therapy

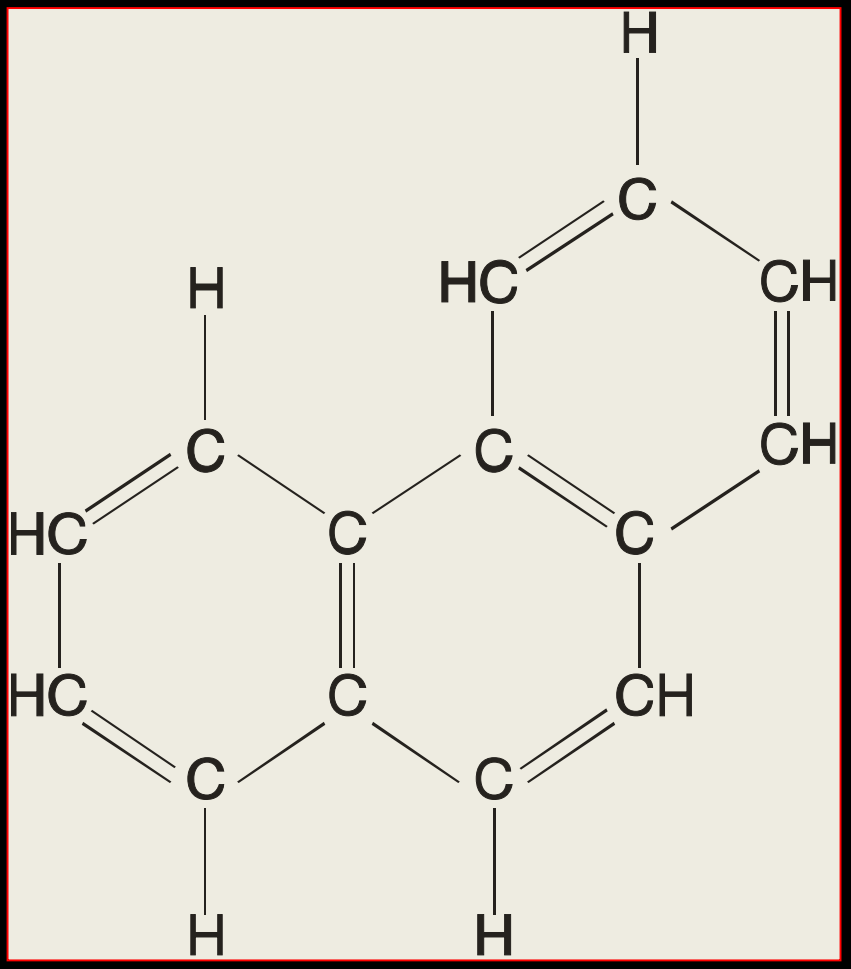
SYSTEMIC TREATMENT,

CORTICOSTEROID

is the mainstay therapy for patients with oral lesions,

transforming a fatal disease into one whose mortality is now below 10%.

The steroid level could be decreased until the patients are symptomless.

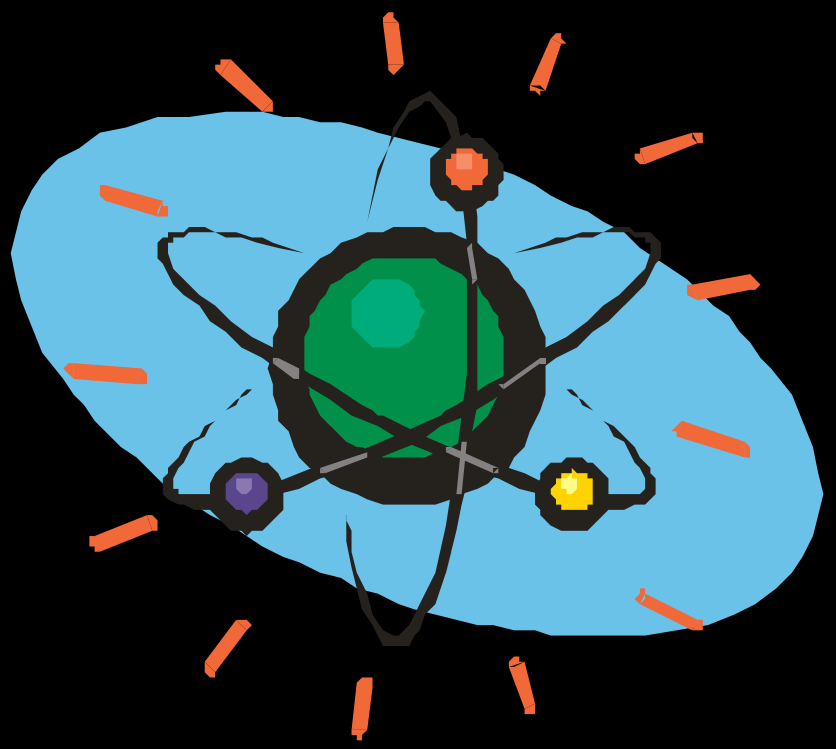


Steroid sparing materials

Azathioprin,
chlorambucil,
cyclophosphamide

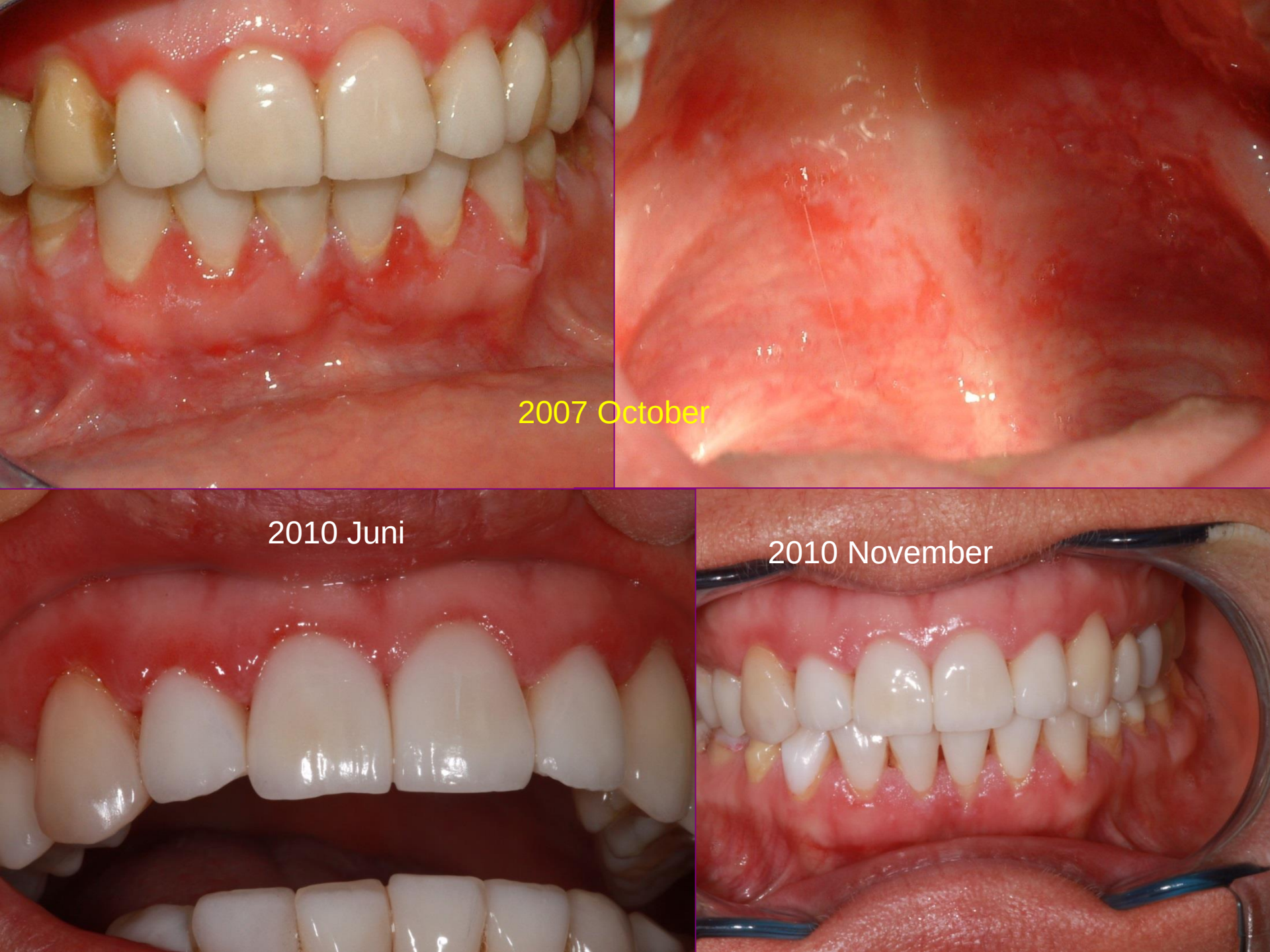
can be effective as adjuvant to steroid.

Methotrexate in low dose may be benefit.



Oral Care

- **Systemic** immunosuppressant are essential
- **Topical or** intralesional corticosteroid for localized oral lesions with low titer circulating antibodies
- The treatment also consists of **improving oral hygiene** minimizing irritations of the lesions.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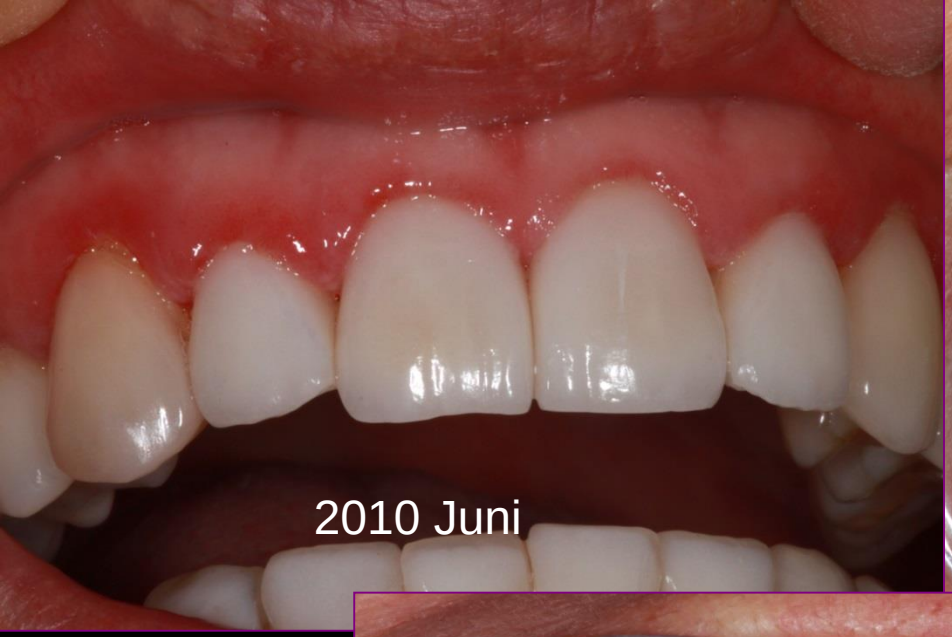
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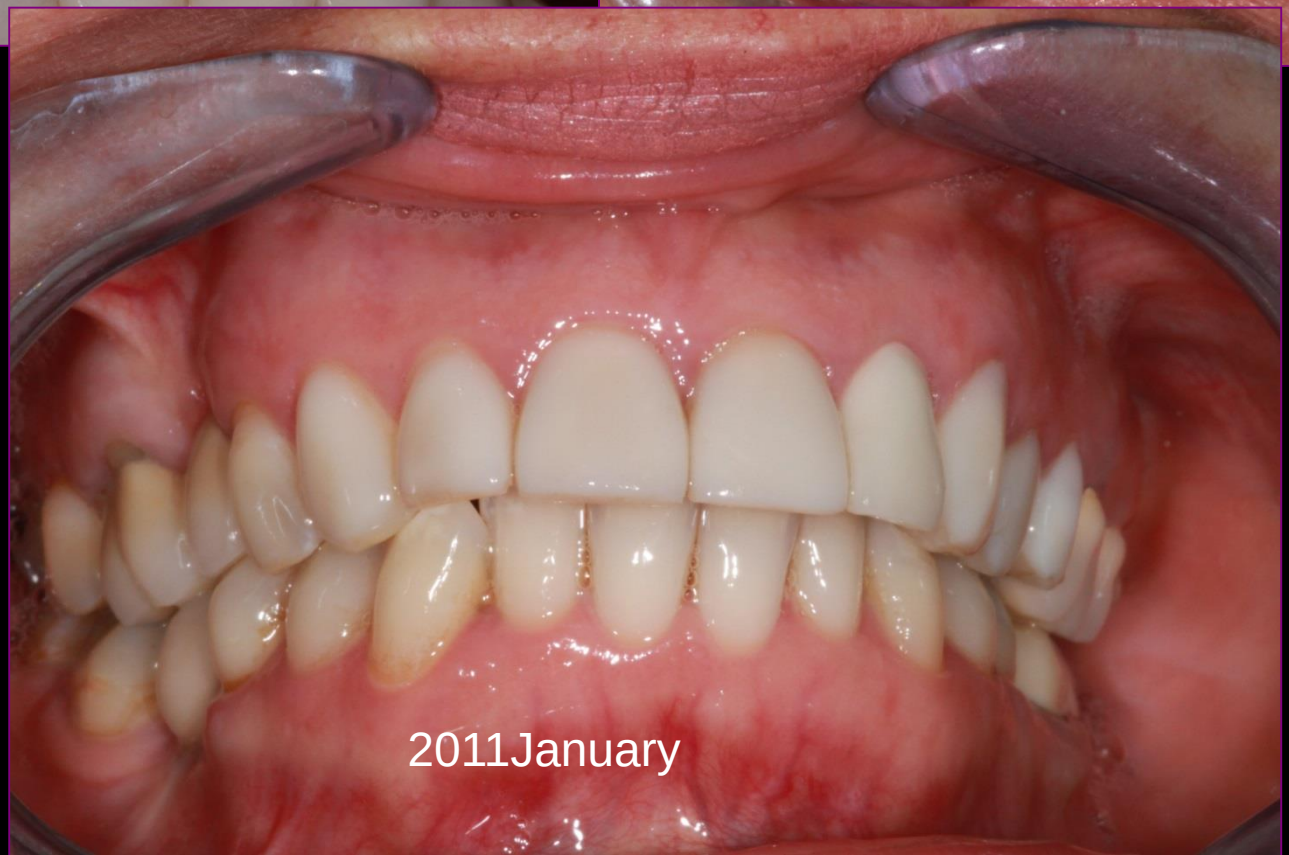
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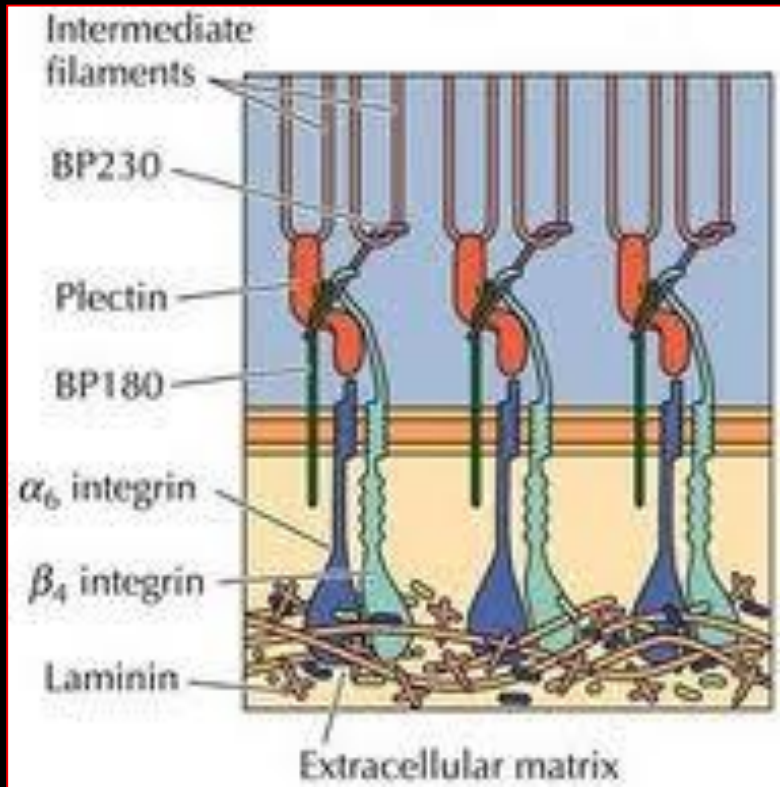
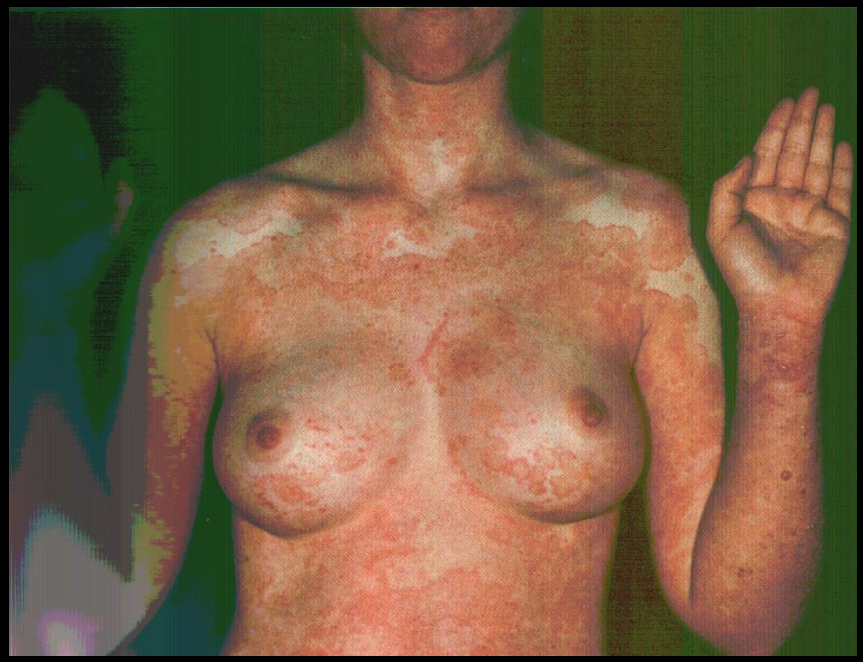
Pemphigoid Group

THE TARGET IS ONE OF THE PROTEINS OF HEMIDESMOSOMES



Diseases in this group

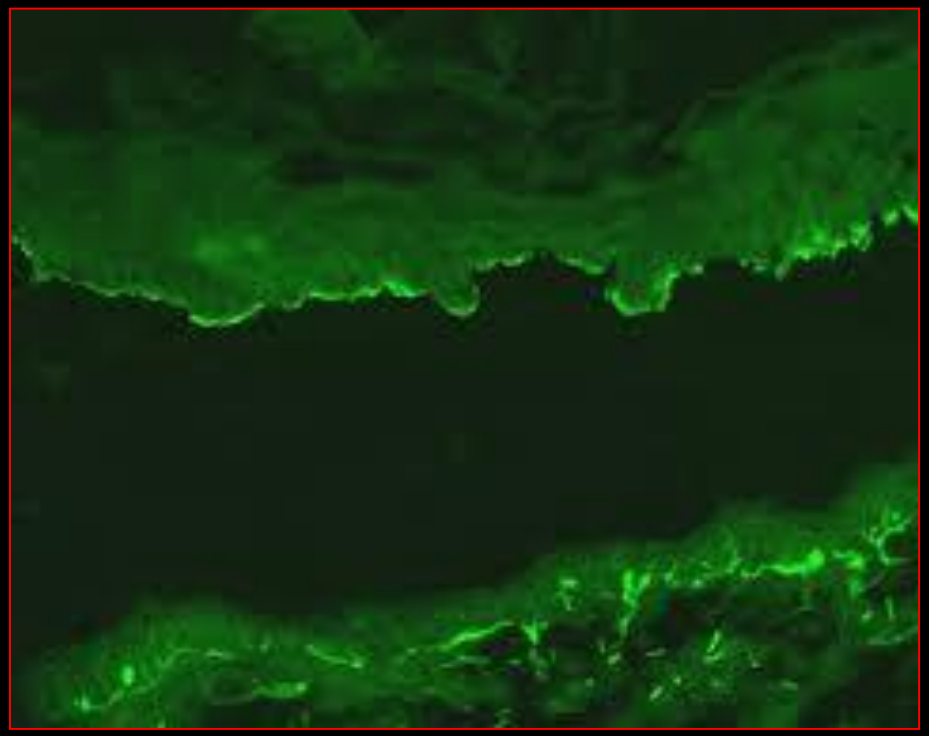
- **Bullous Pemphigoid**
- **Benigne Mucosal Pemphigoid**
- **Cicatrical Pemphigoid**
- **Herpes Gestationis**
- **Epidermolysis Bullosa Aquisita**



These diseases are autoimmune,

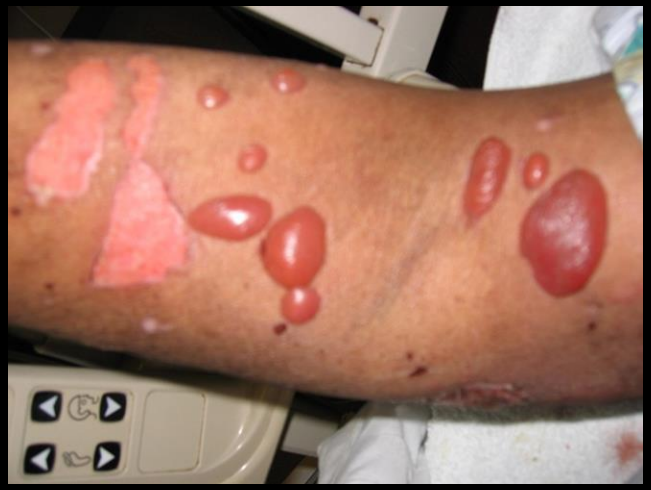
subepidermal,
blistering, producing blisters in the epidermal-subepidermal border.

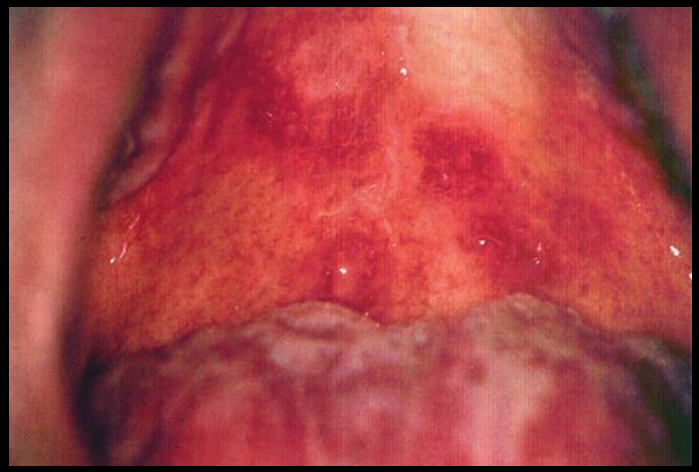
IgG and C3 can be seen by direct immunefluorescence in the basement membrane zone.



Bullous Pemphigoid (BP)

- It is a rare relatively benign autoimmune disease of the elderly.
- There is no racial or gender prevalence.
  - 24% of the cases have oral symptoms





Benigne Mucosal Pemphigoid (Cicatrical Pemphigoid)

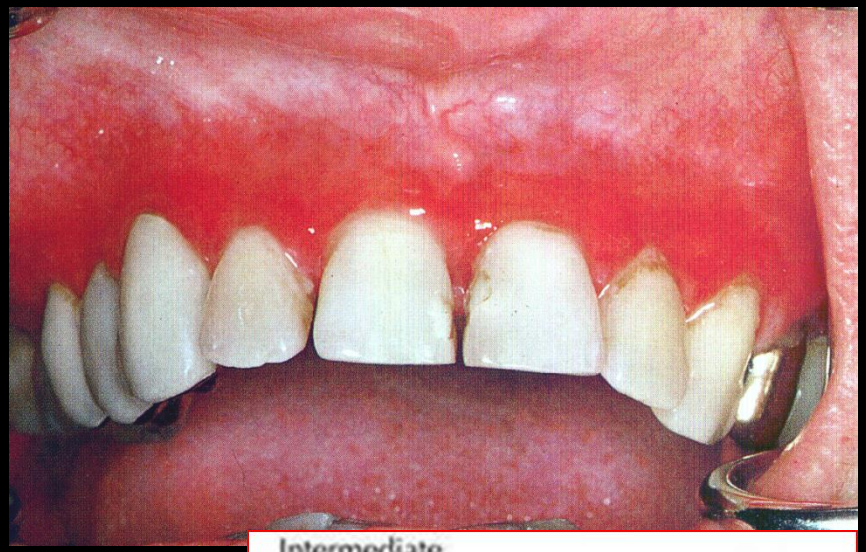
It is a rare subepidermal blistering and scaring disease

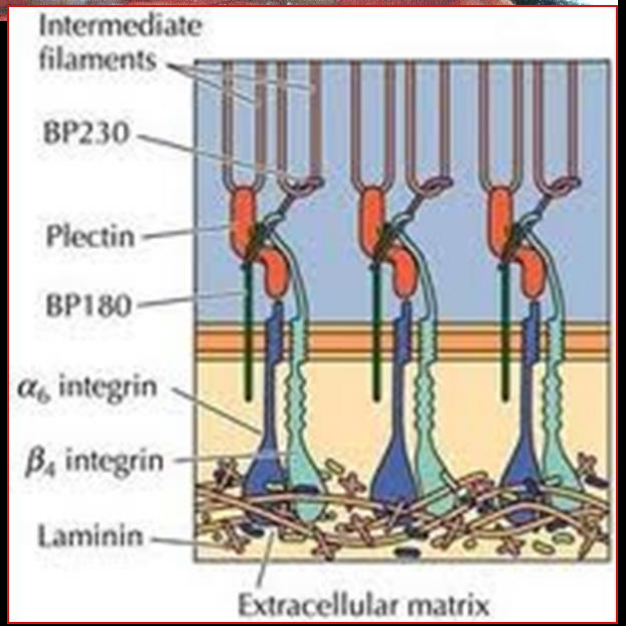
characterized by autoantibodies against BMZ antigens (lamina lucida proteins involved in human keratinocyte adhesion to extracellular matrix).

disease affects persons older than 40y

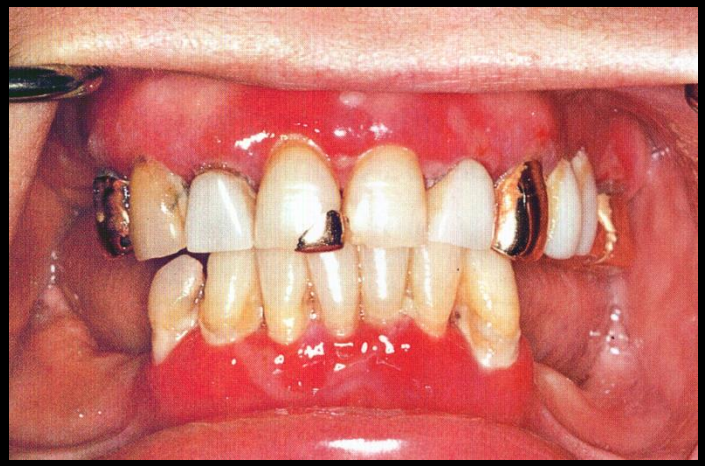
F/M ration a 2 : 1 .

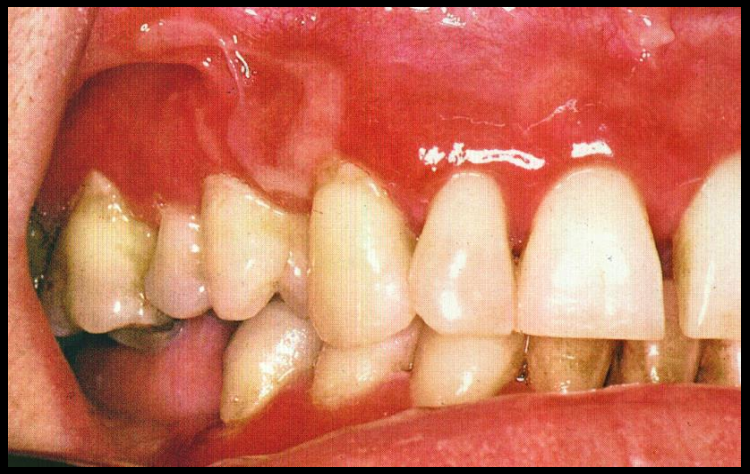
Frequent on the eyes.

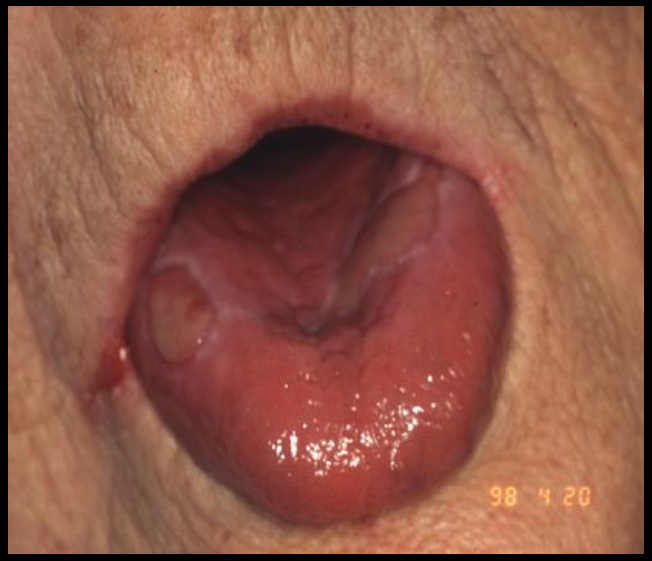


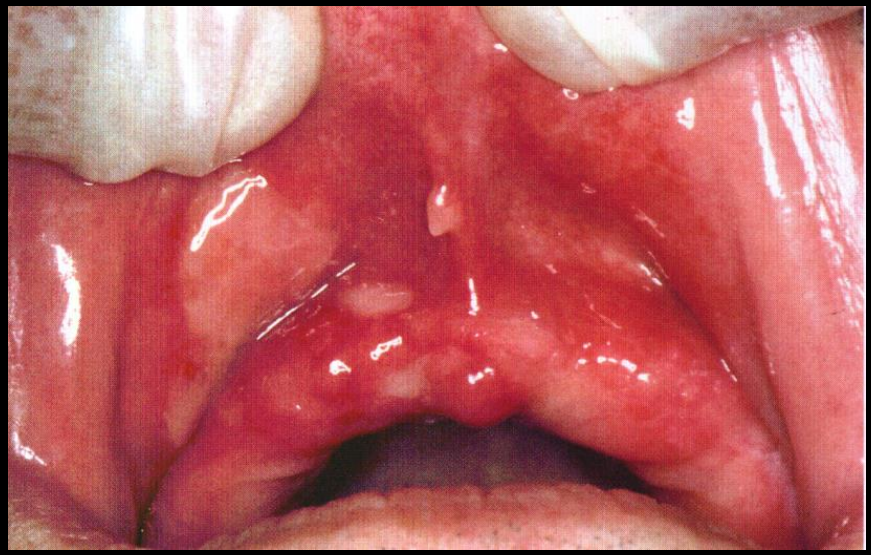


Oral mucosa are involved in 85% of the cases

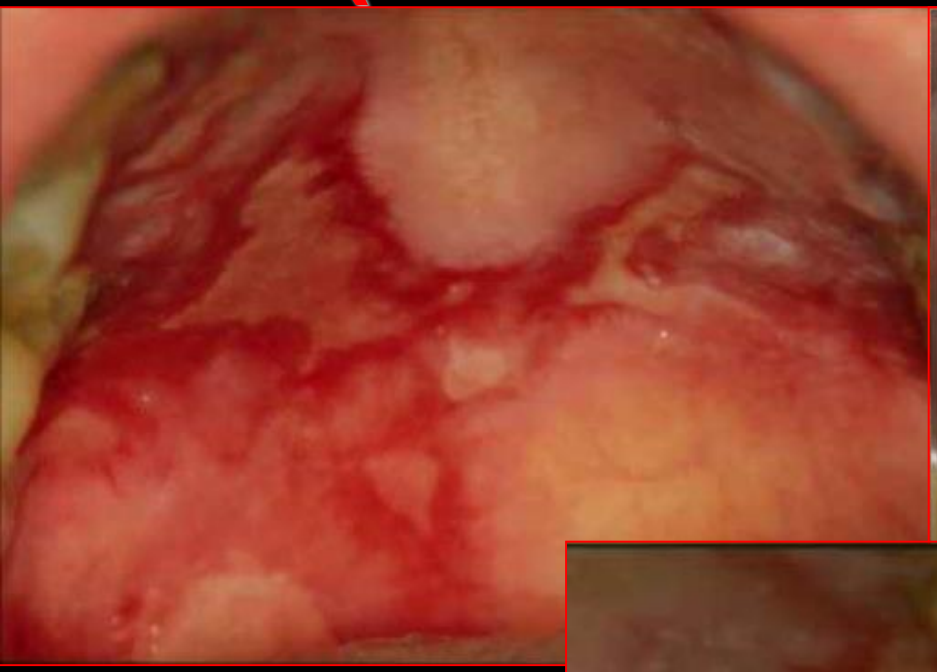


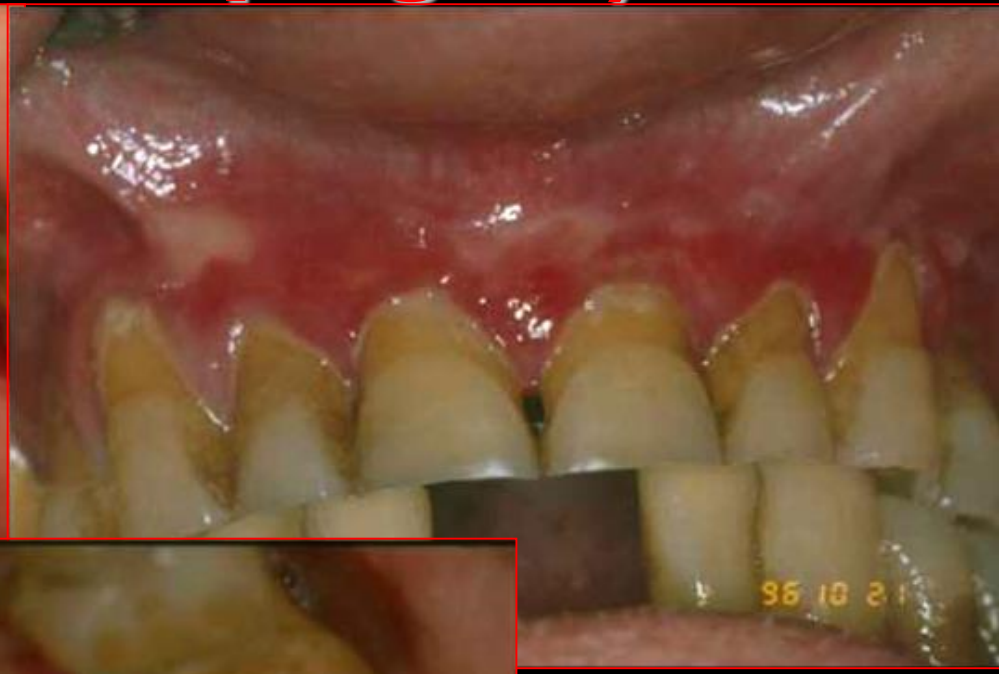


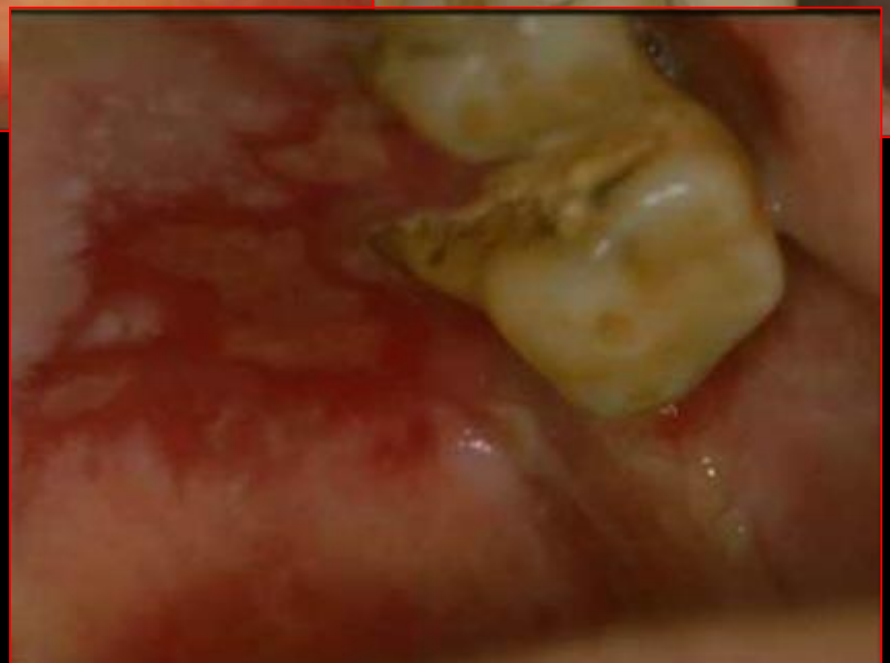




Benign Mucosal Pemphigoid (Cicatrical Pemphigoid)







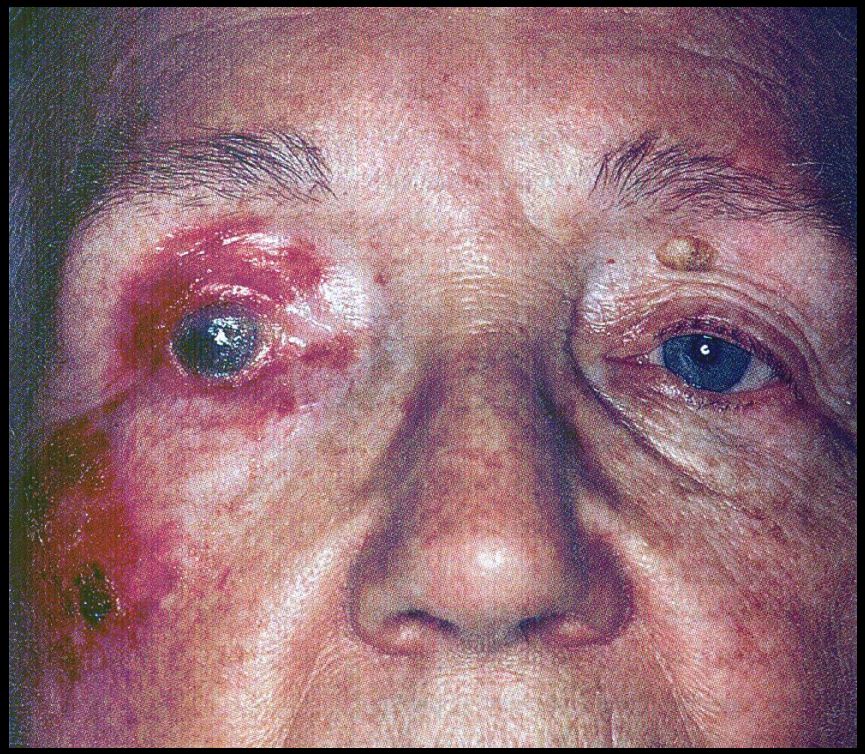
Occular and Skin lesions

Out of 65% all the cases. The conjunctiva fibrosis then erosions leads to opacificatio and blindness.

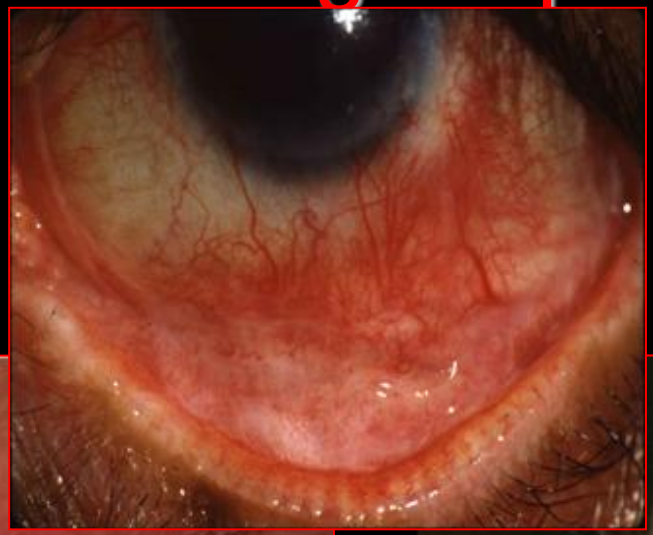
Skin lesions:

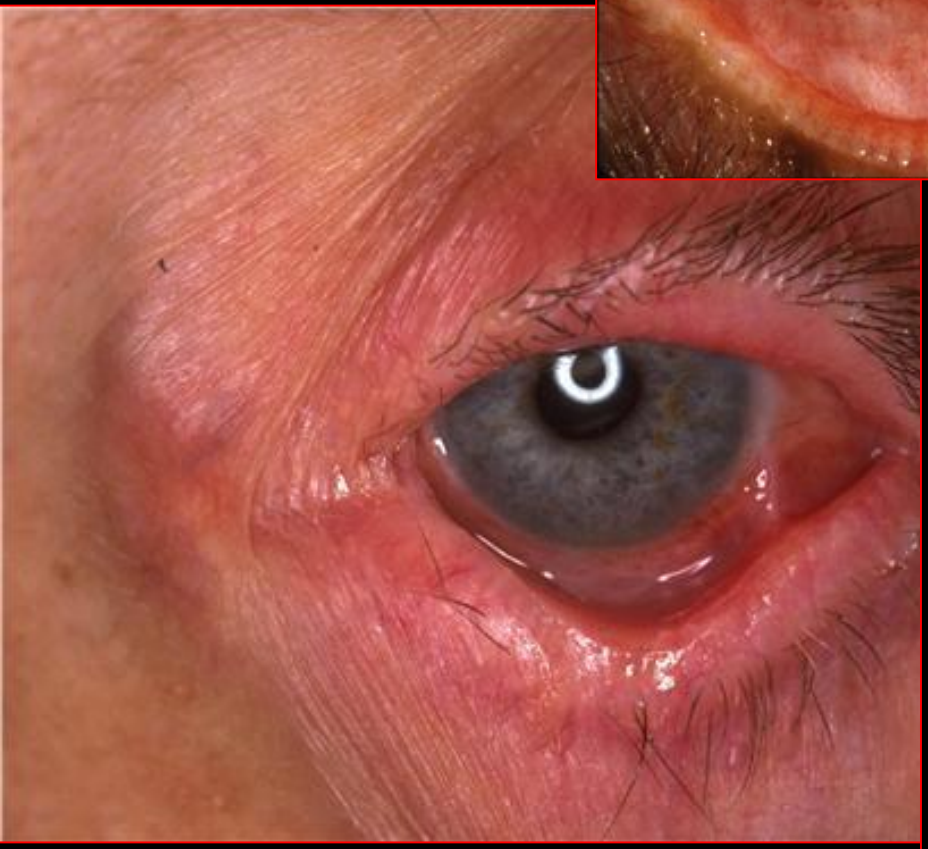
25% out of the cases on the face, neck and hairy scalp.

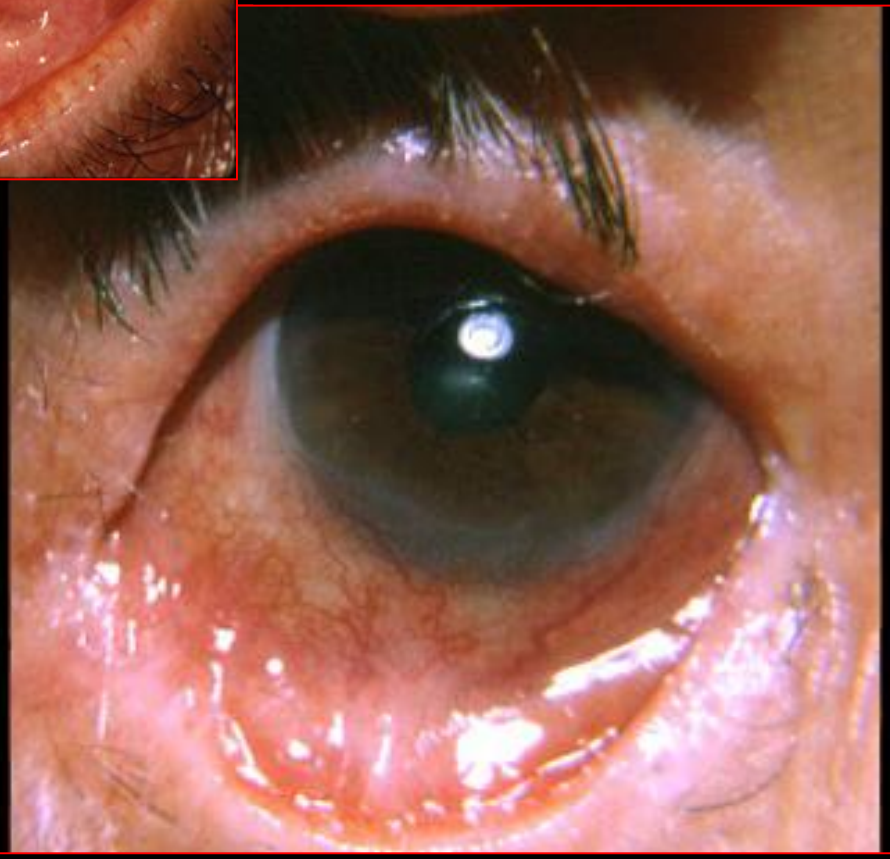
The bulla erupted and heal with atrophic scar.



Opthalmological problems



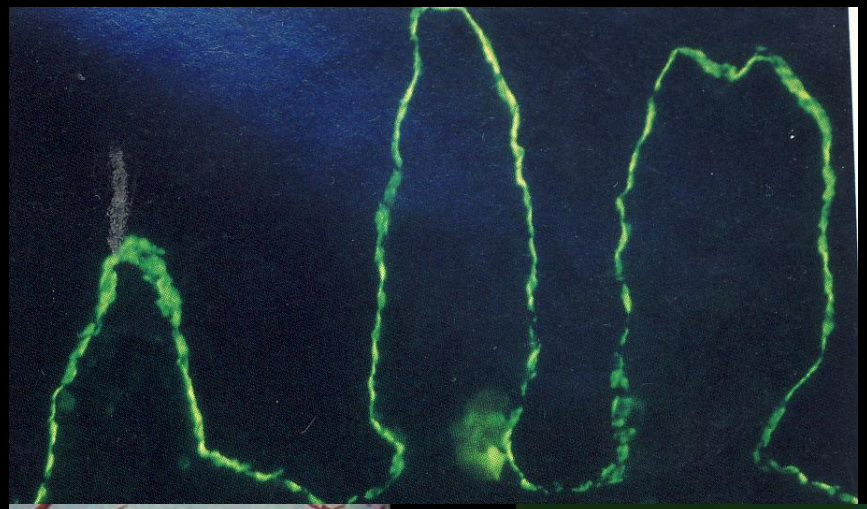


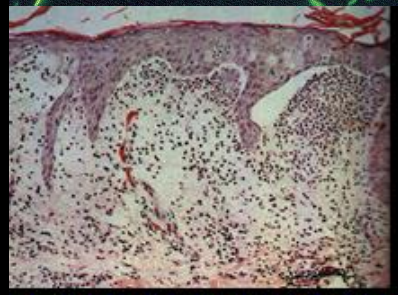


Diagnosis

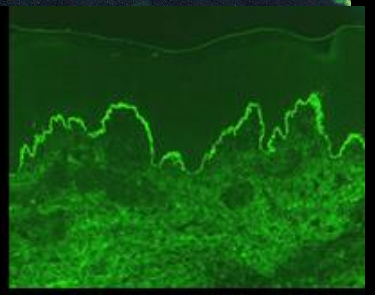
The biopsy shows a subepidermal cleft formation with little inflammation.

Direct immunfluorescence DIF of lesional, perilesional and normal mucous membrane shows linear deposition of complement and IgG less often IgA.

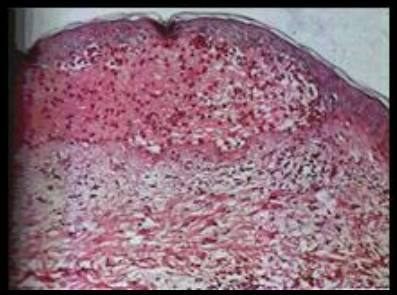


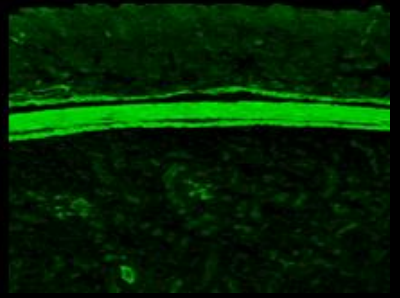


Eosinohil cells in the blister Cleft



DIF has a high diagnostic value . In the basal membtane zone IgG, C3, IgM and sometimes IgA can be shown.



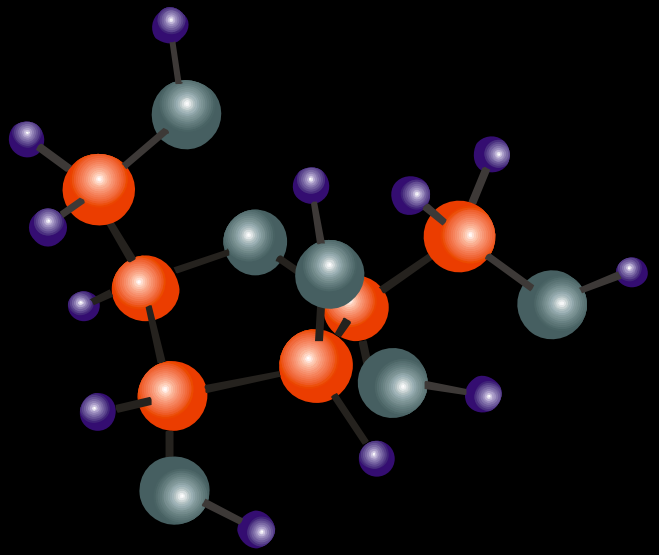


Therapy

Aggressively of the treatment depends on the severity of disease.

Oral, eye, genital, esophageal diseases needs aggressive treatment.

In the oral cavity intralesional corticosteroid,
lidocain,
Susp. Anaesth.

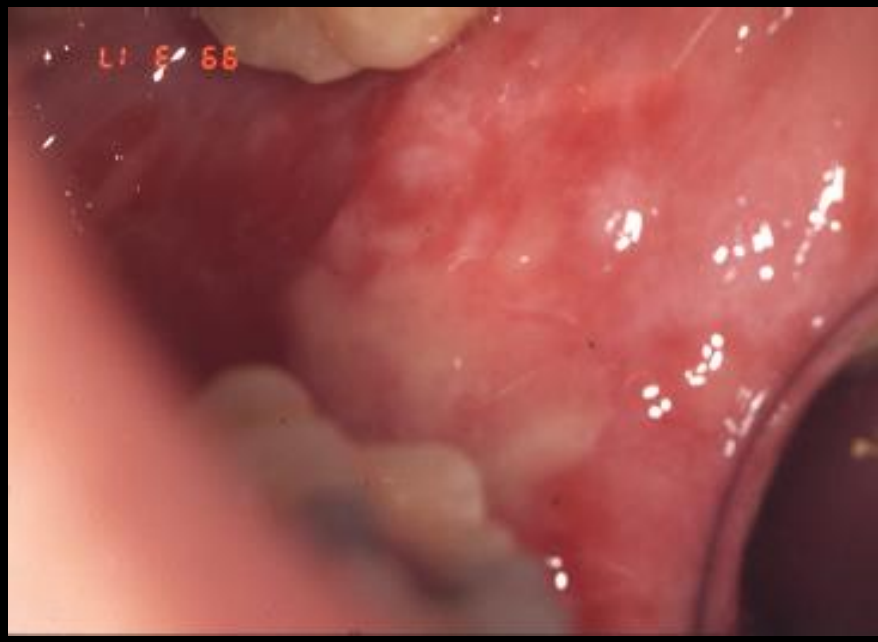


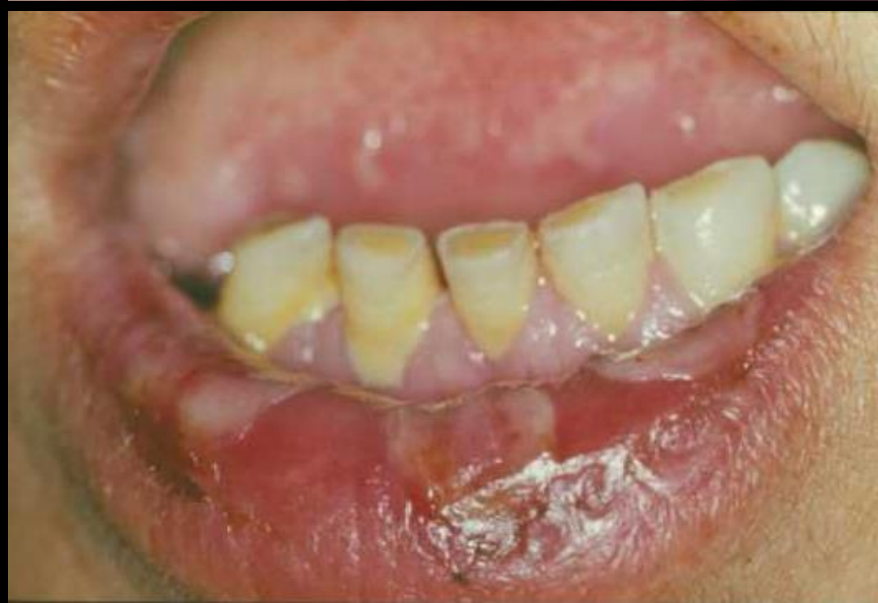
Systematically 125-150 mg corticosteroid daily.

Sometimes: Doxycyclin.

Erythema multiforme

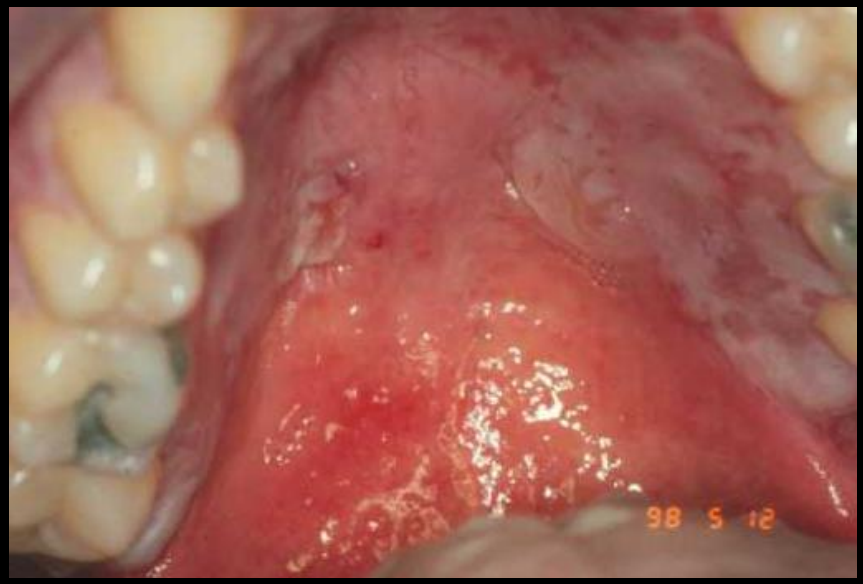
- It is one of the most common oral diseases
- Most of the dentists can meet them in his practice
- 24-48 hours after allergen exposition develop the clinical signs
- The autumn/ spring incidence is the most common but it can develop any time





Erythema multiforme (EM)

- is an acute, self-limited, and sometimes recurring skin condition that is considered to be a type III - IV hypersensitivity reaction
- associated with certain infections, medications, and other various triggers



Erythema multiforme (EM)

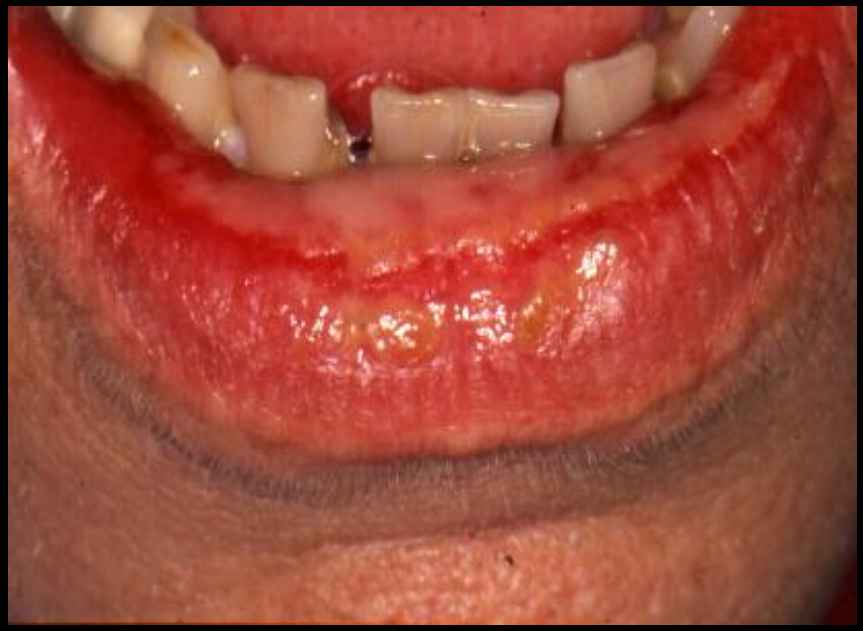
In early stage IgM later IgG containing immuncomplex (IC) produced. Complement system activated on alternative pathway.

The IC block the capillary vessel loops.

The C3a and C5a complement factors play role as

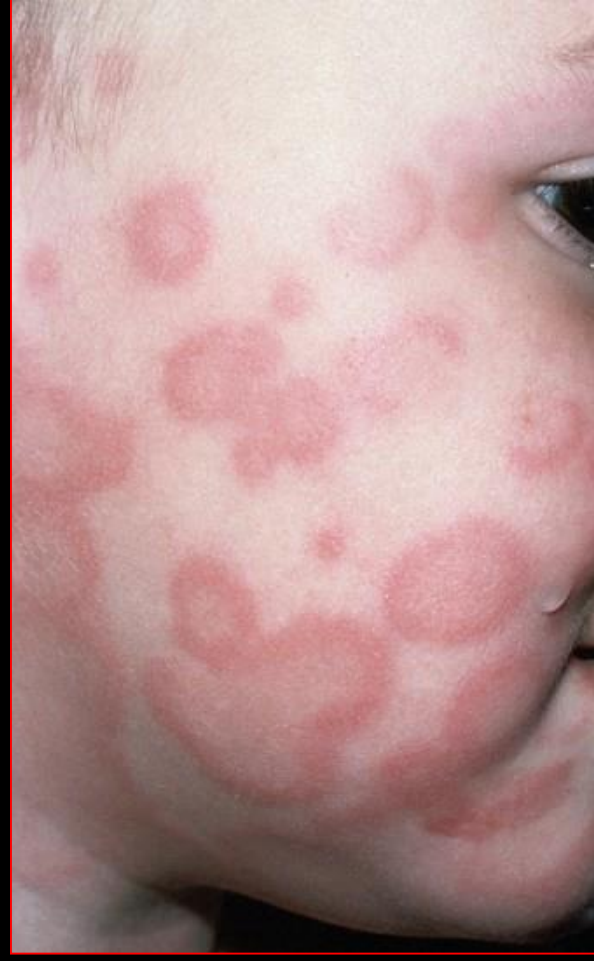
ANAPHILATOXIN

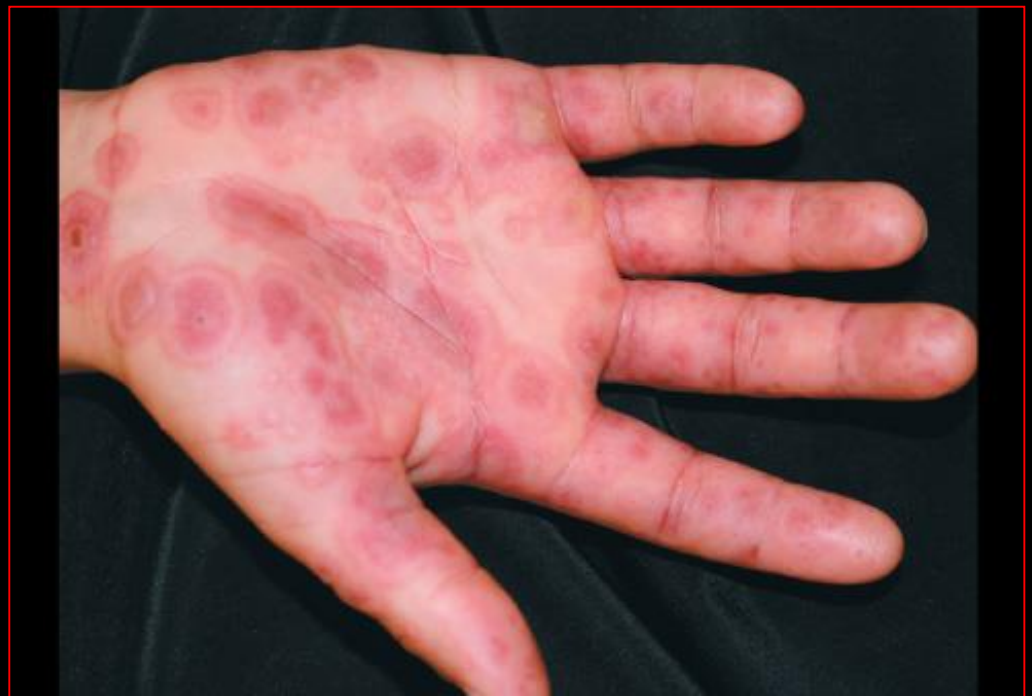
which can release histamine from mast cells and basophile granulocytes.

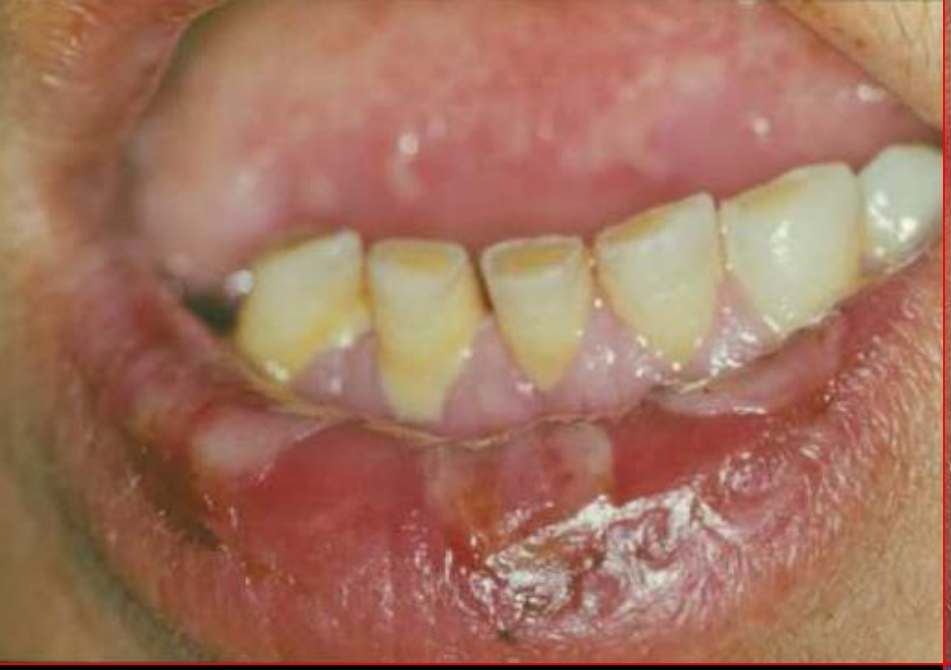


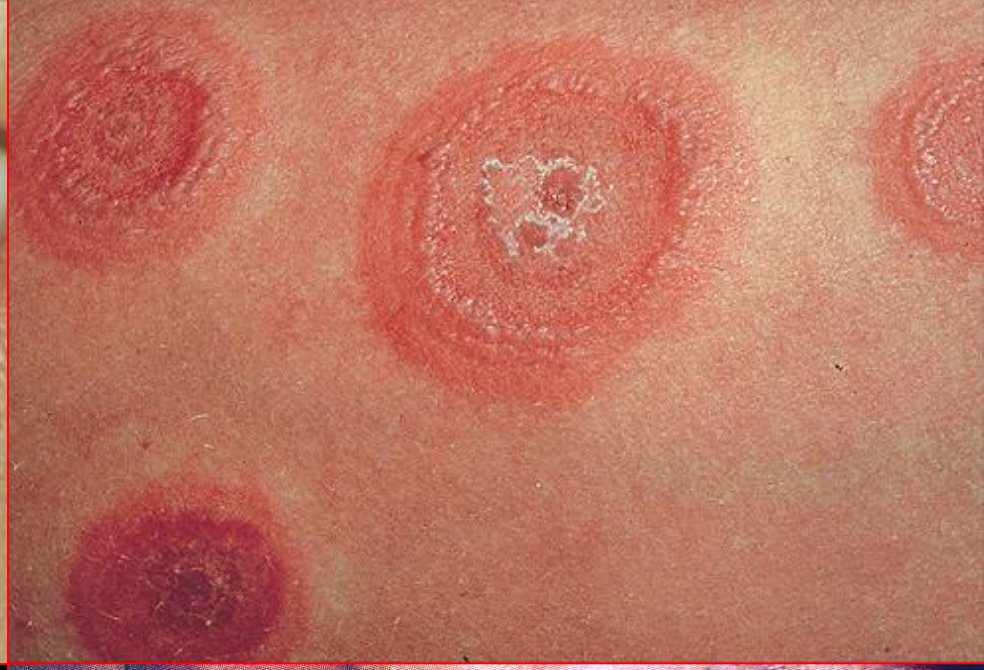
- **Erythema (redness) multiforme (EM) is usually a reaction of the skin and mucous membranes that occurs suddenly.**

- **It appears as a rash on skin and may include mucous membrane lesions**

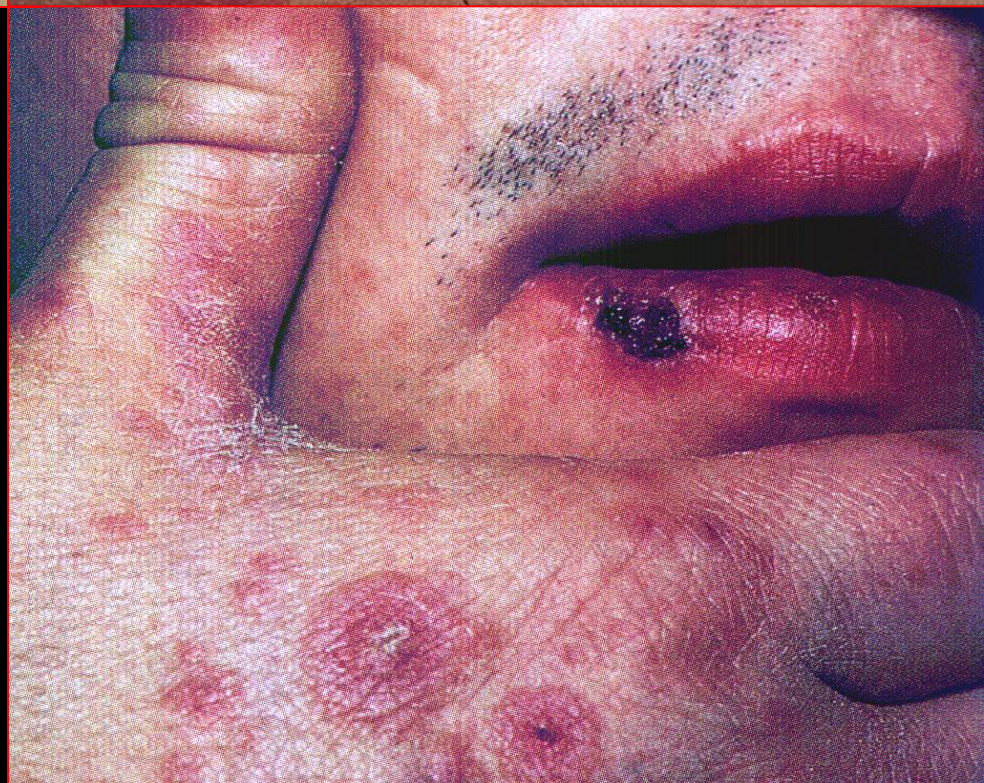








- The clinical symptoms develop 24-48 hours after antigen exposition.
- Frequency increase in spring and autumn but it can develop in every time.
- After 6-8 recurrences a longer interruption follow, and then begin again.



Recent classification

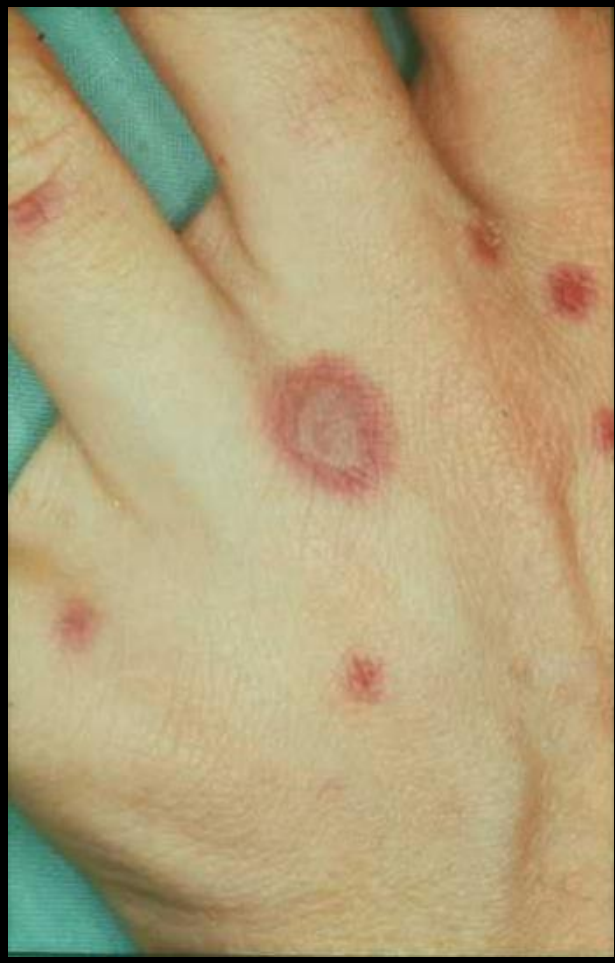
- Stevens-Johnson syndrome was separated from the erythema multiforme
-
- Stevens-Johnson syndrome and toxic epidermal necrolysis (TEN) are considered severity variants of a single entity.

- (1) erythema multiforme consisting of erythema minor and major

- (2) Stevens-Johnson syndrome / toxic epidermal necrolysis (SJS/TEN).

Clinical characteristics

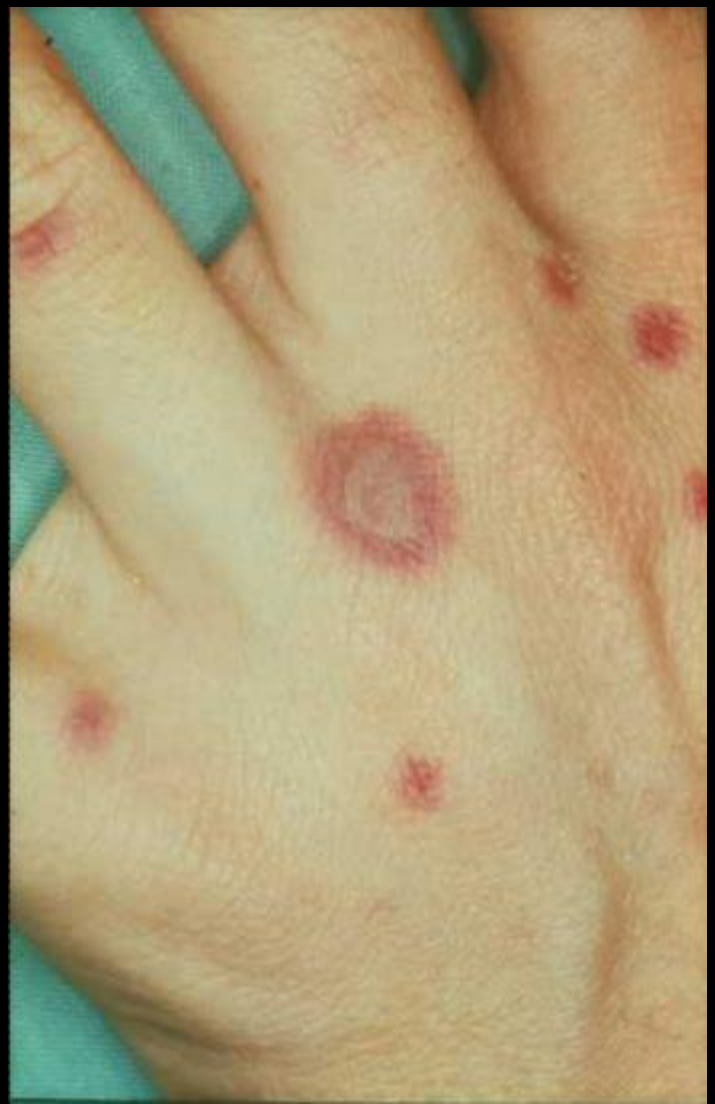
- Erythema multiforme minor - Typical targets papules distributed acrally
- Erythema multiforme major - Typical targets with leseions onmucous membranes;
- epidermal detachment involves less than 10% of total body surface area (TBSA).



- SJS/TEN - Widespread blisters on the trunk +mucous membrane erosions;
- Steven-Johnson syndrome epidermal detachment is less than 10%
- toxic epidermal necrolysis. 30% or more

common mild form is refer to as EM minor.

- skin rash that involves no more than one mucosal surface.
- classic iris or target lesions.
- They have bright red borders and small white bumps in the center.
- The causes:
- viral
- chemical products,
- antibiotics - specifically penicillin or cephalosporin.

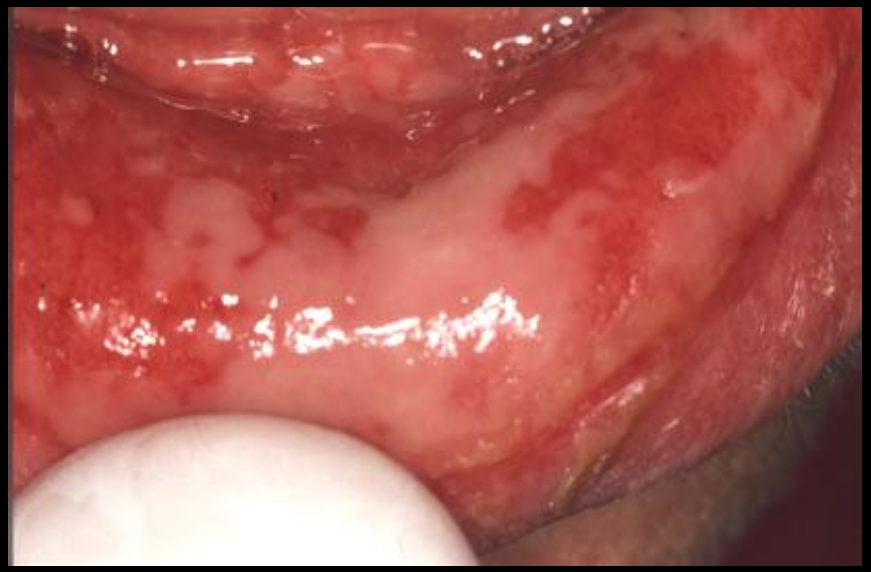


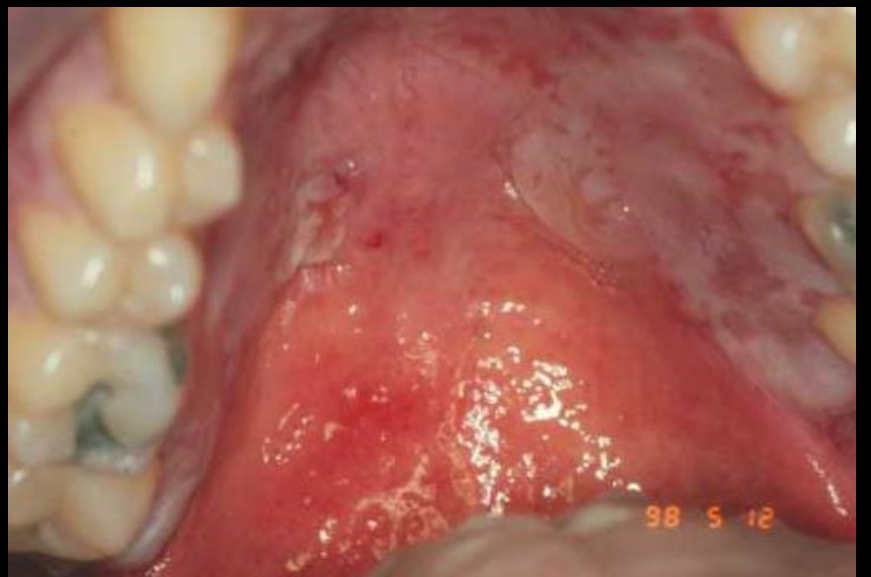
Postherpetic Erythema Exudativum Multiforme

Minor form:

.

In these cases the HSV play role not only infective agent, but as antigen too.

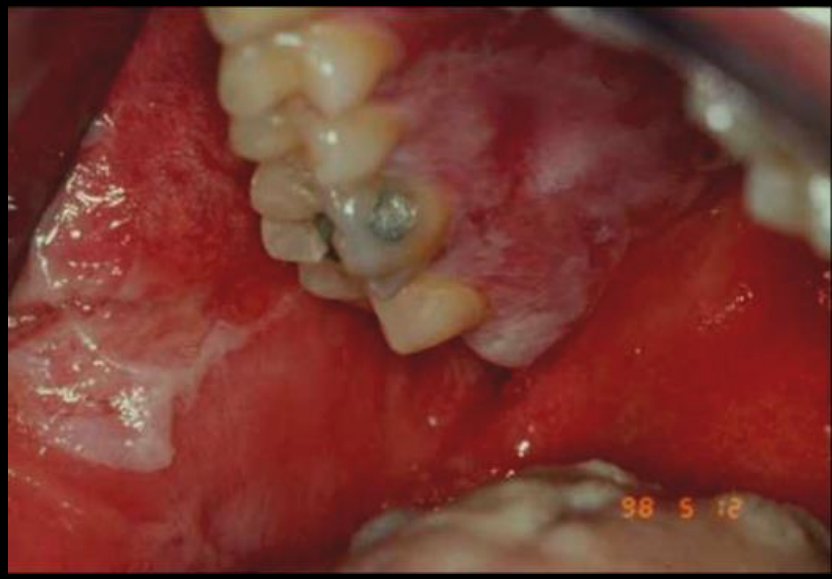




(HSV)–associated erythema multiforme

- this is now recognized as a variant of erythema multiforme, rather than Steven-Johnson syndrome.

- Erythema multiforme with mucosal involvement is now termed bullous erythema multiforme.



Erythema multiforme major

- More severe, potentially life-threatening disorder
-
- Most patients have extensive mucosal involvement.

- More than 50% of all cases are attributed to medications.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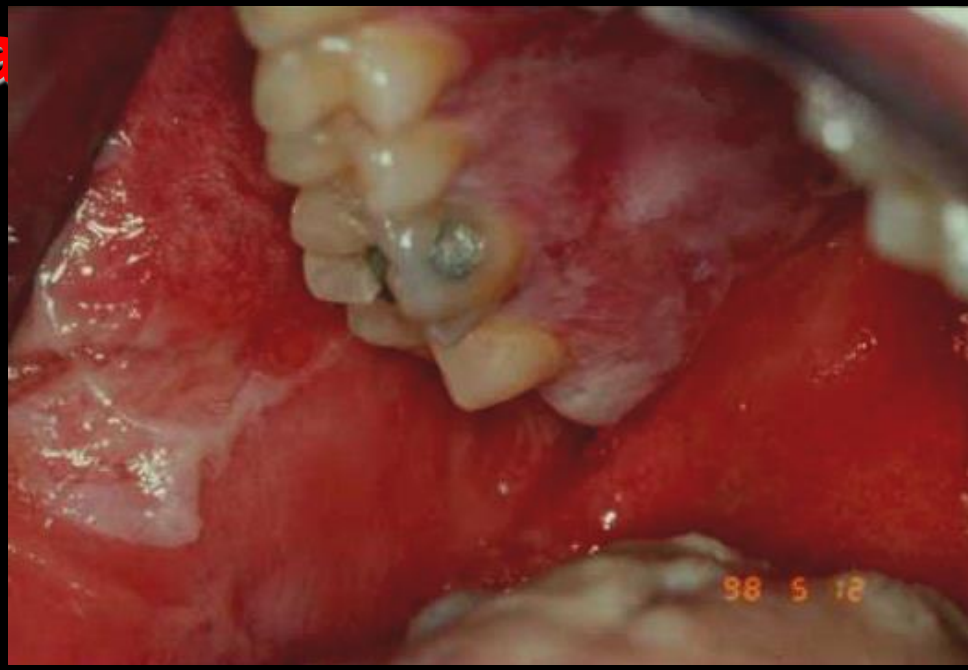
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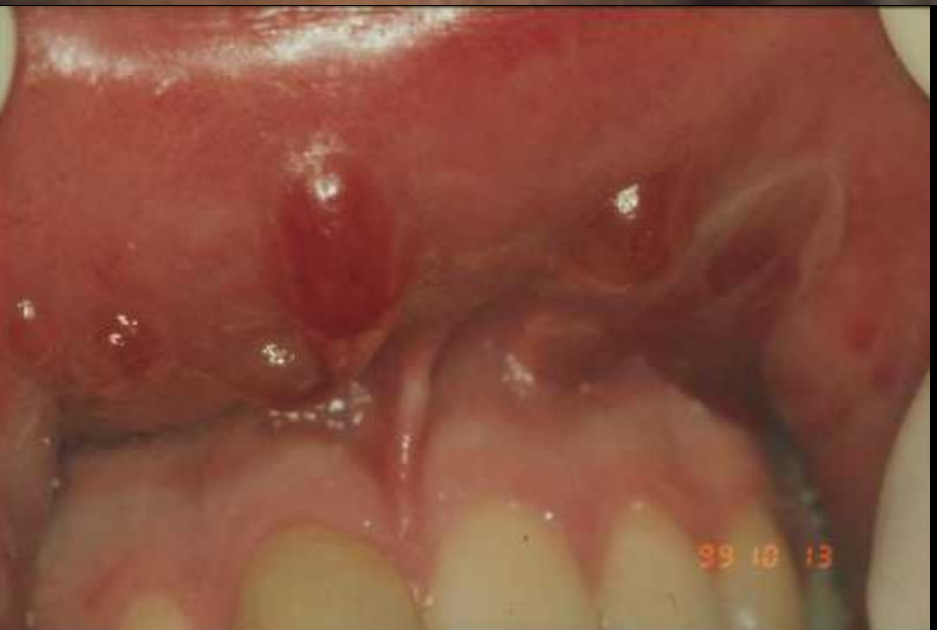
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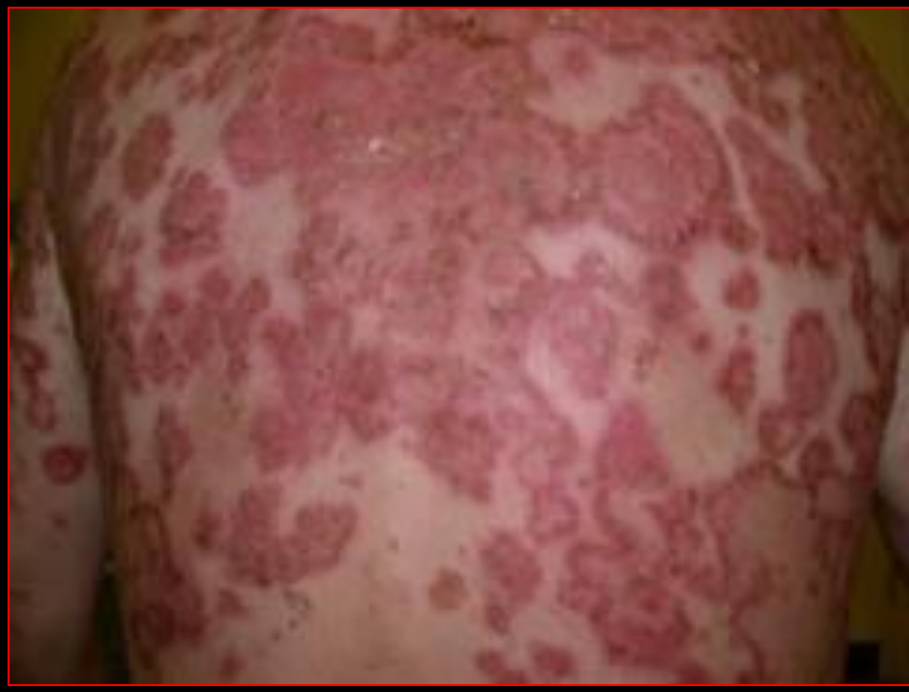
Stevens–Johnson Syndrome

- is named for
- Albert Mason Stevens
- Frank Chambliss Johnson,
- Two American pediatricians who jointly published a description of the disorder in the

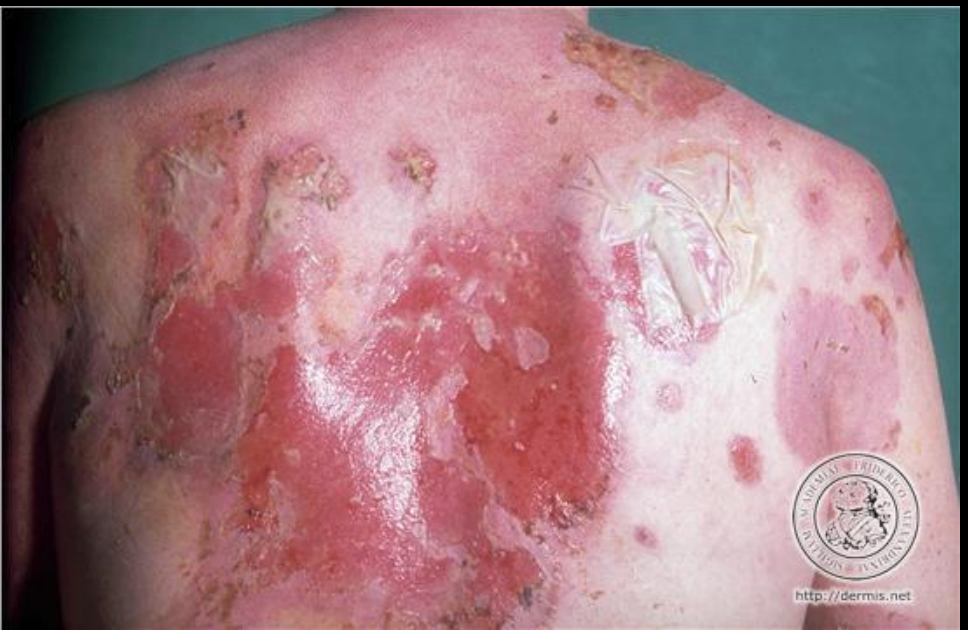


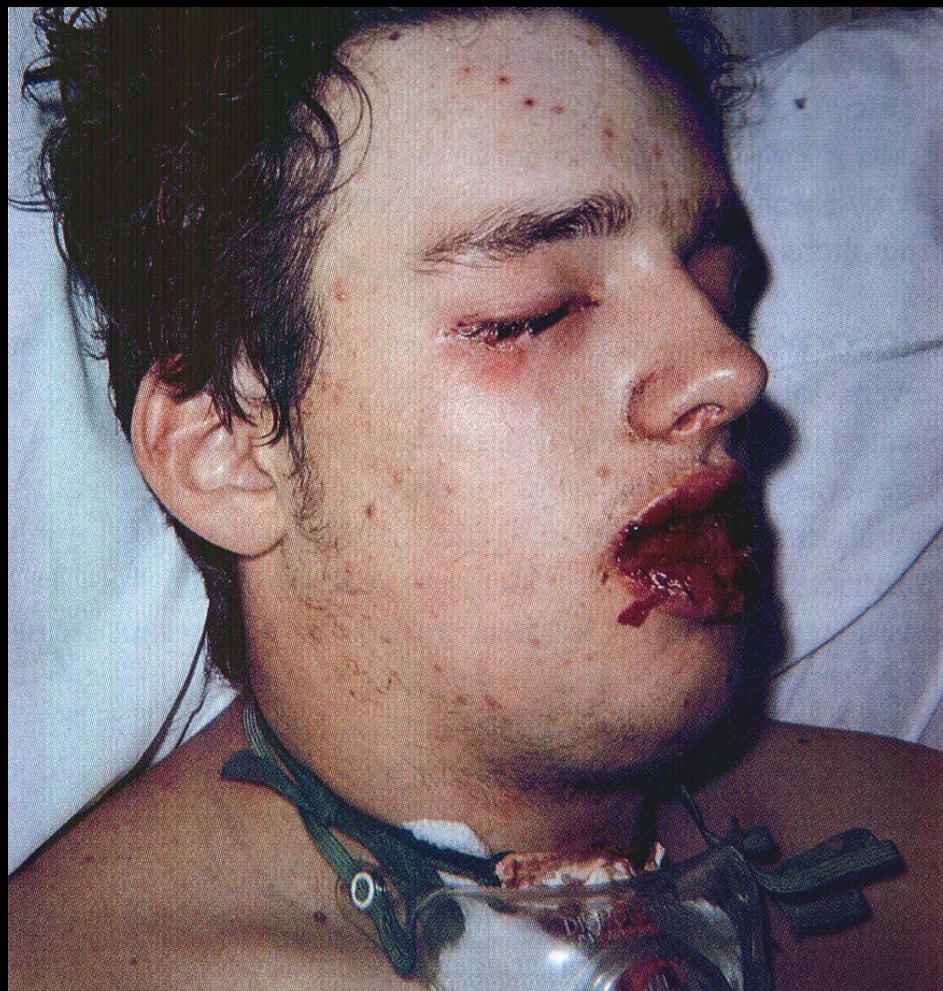
- American Journal of Diseases of Children in 1922

Steven-Johnson syndrome

.

Large blisters can be found not only in the oral cavity, but on the skin .





Stevens–Johnson syndrome (SJS)

- Usually begins with fever, sore throat, and fatigue,
- Bulla, erosions and ulcers appear in the mucous membranes, in the mouth, lips genital and anal regions.
- in mouth usually extremely painful and reduce the patient's ability to eat or drink.
- Conjunctivitis occurs in about 30% of children who develop SJS.
- A rash of round lesions about 3cm across arises on the face, trunk, arms and legs,

Stevens–Johnson syndrome (SJS)

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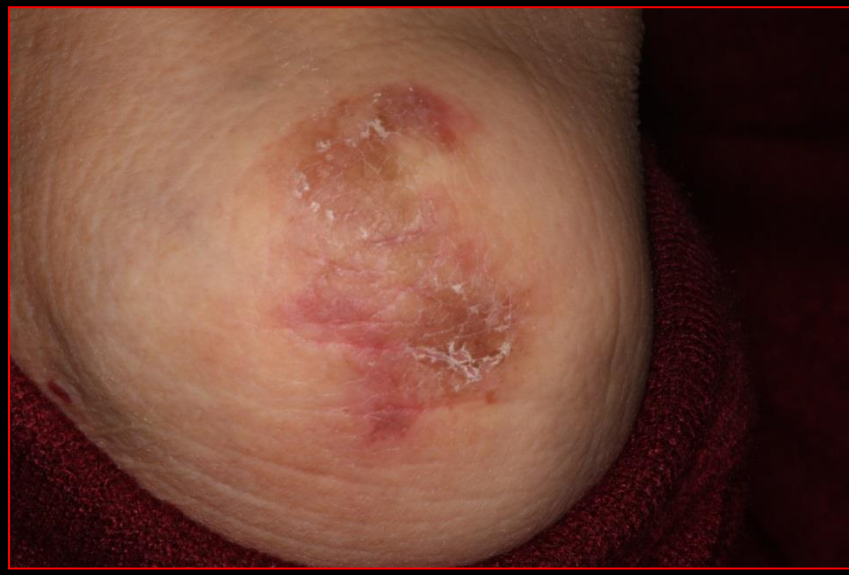
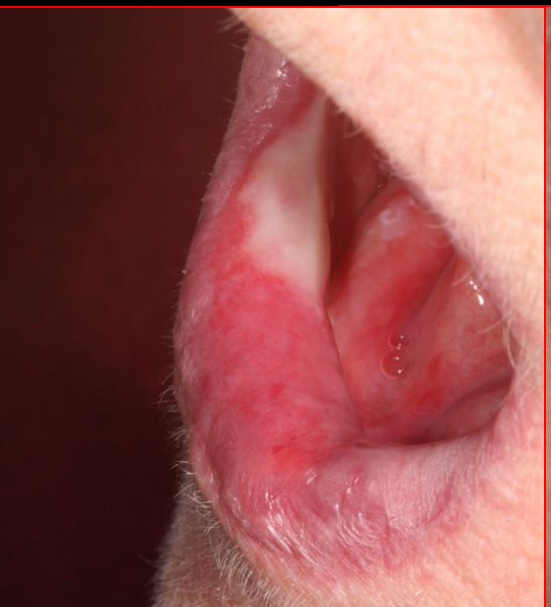
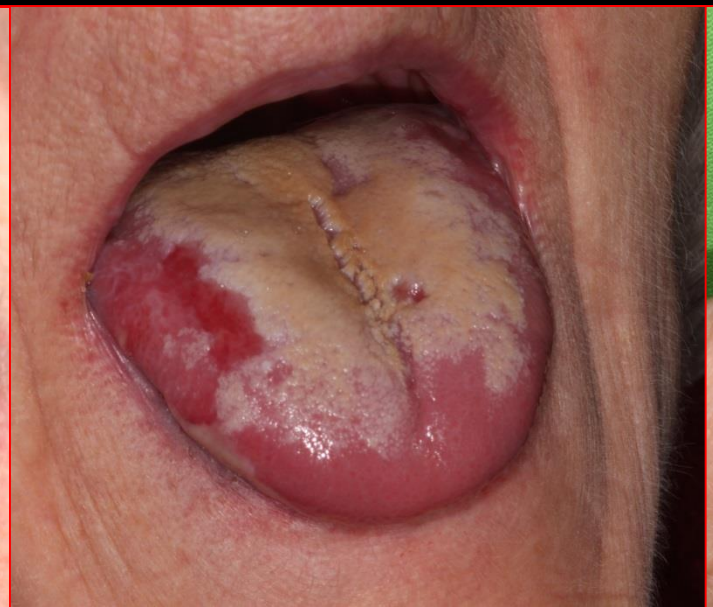
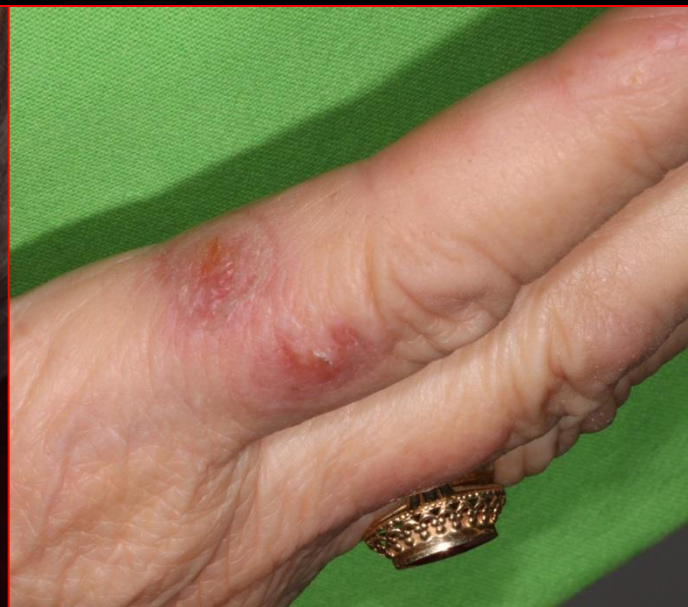
-





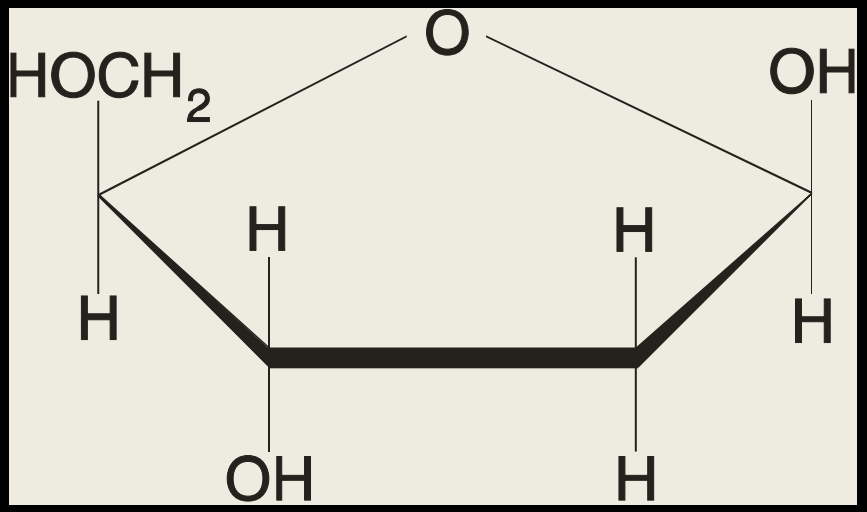




Therapy

- Anti-histamines systemically:
- Inj. Calcimusc i.v., Tab. Zyrtec, Caradonell,
- Caps. Fenistil
- Corticosteroid ??? (frequent recurrences)

- Anti-infective oral rinsing:
- Chlorhexidine
- Betadine
- Listerine



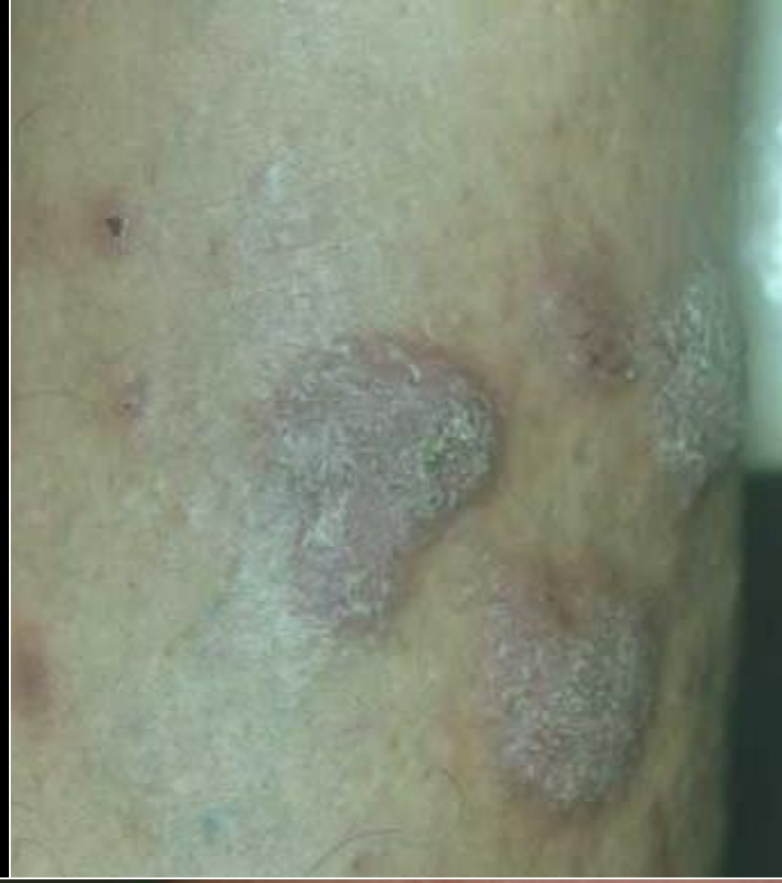
Oral Lichen Planus

The word Lichen originated from the Greek language

The world lichen means
MOSS

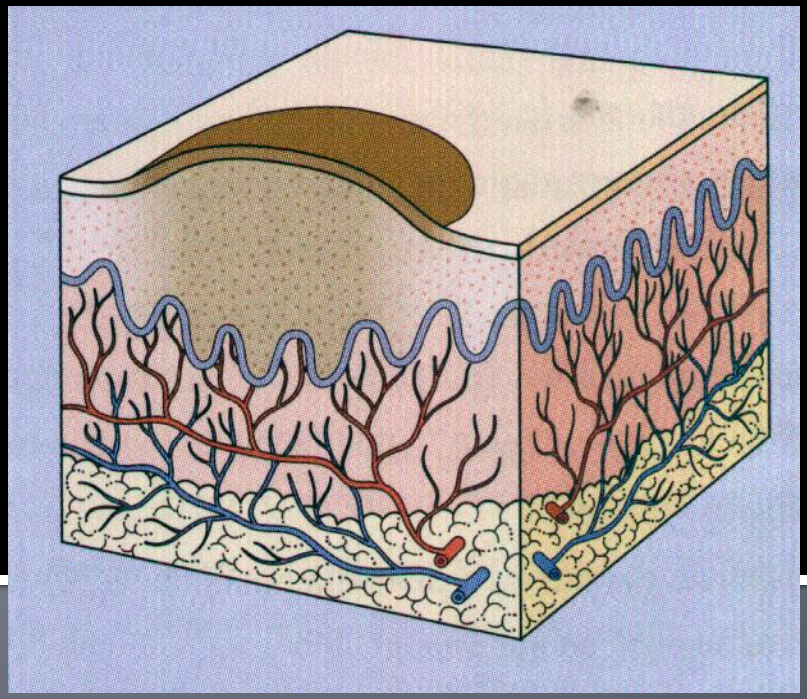
It can be found at the 142. scroll of Hypocrate
and mentioned by
Galenus and,
Omar Kajam.

The disease could have be the same as today.

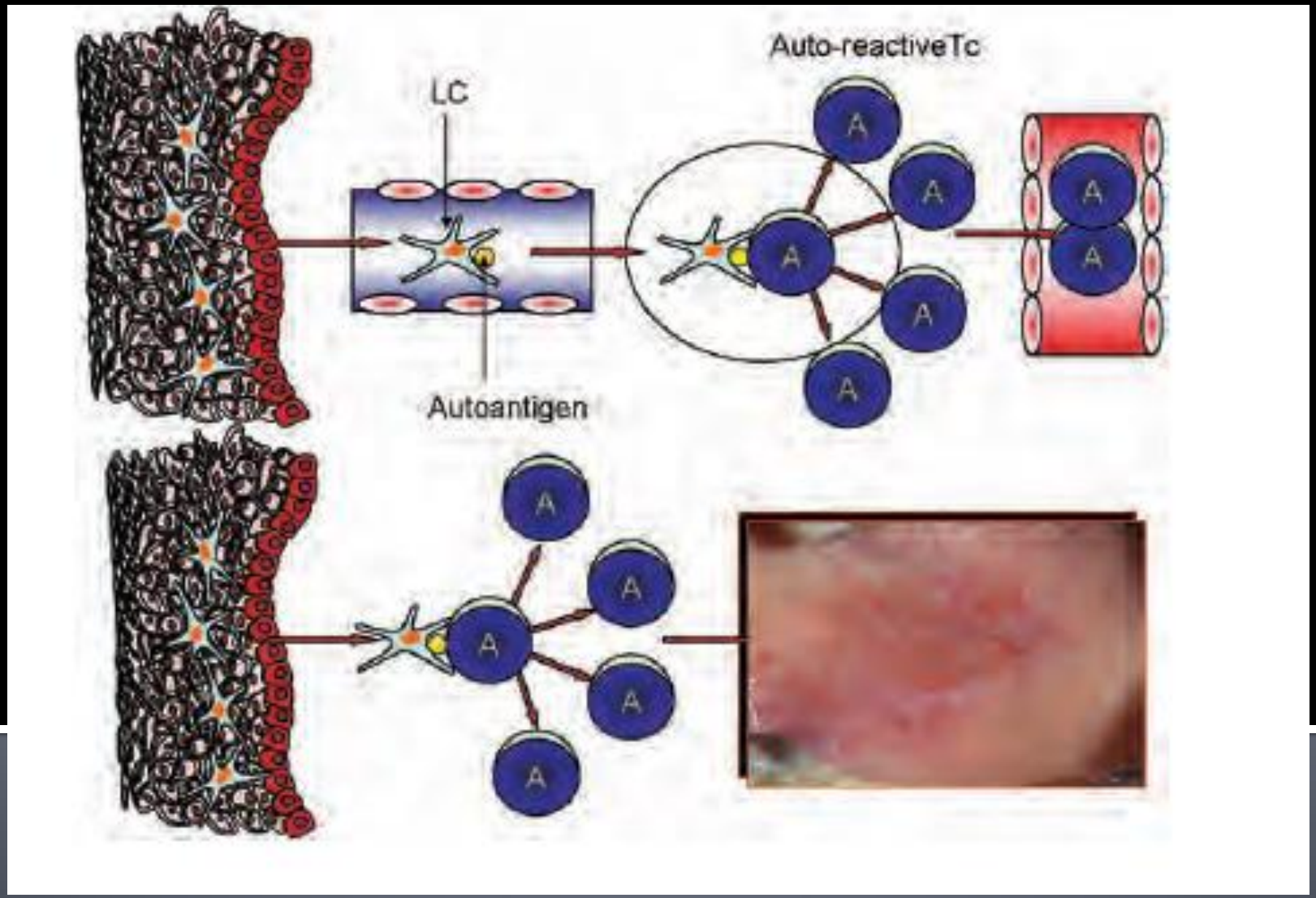


Oral Lichen Planus

The efflorescence is
PAPULA
The characteristic lichen-like drawing is the result of papular convergency
Wickham striae



Oral Lichen Planus

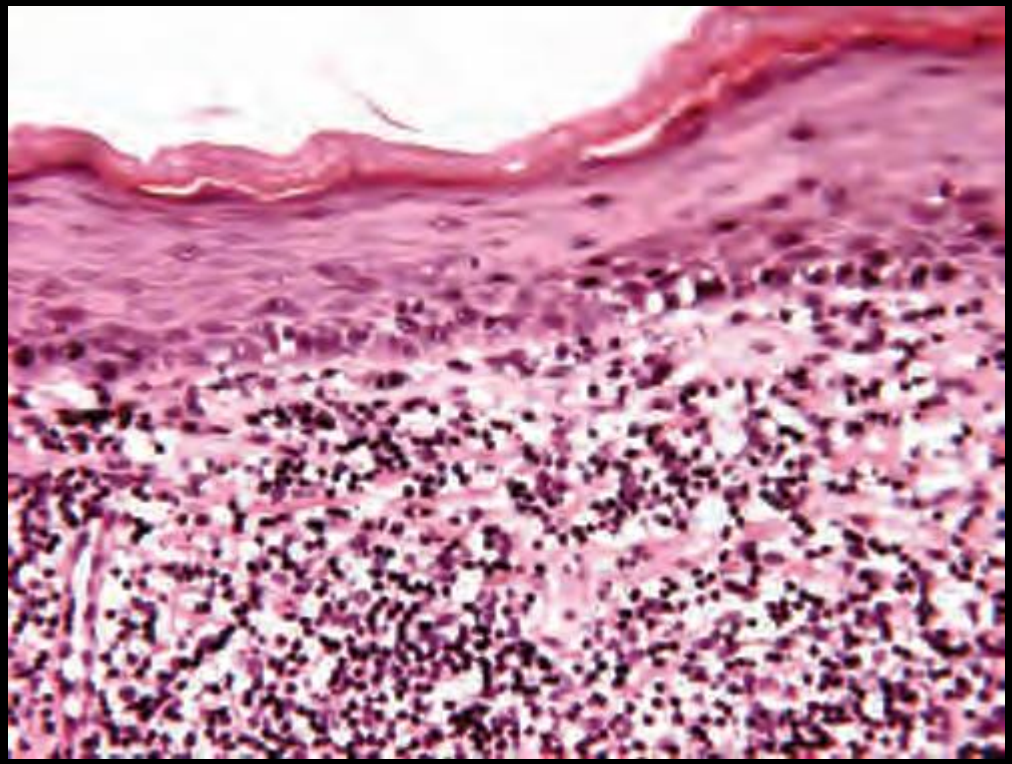


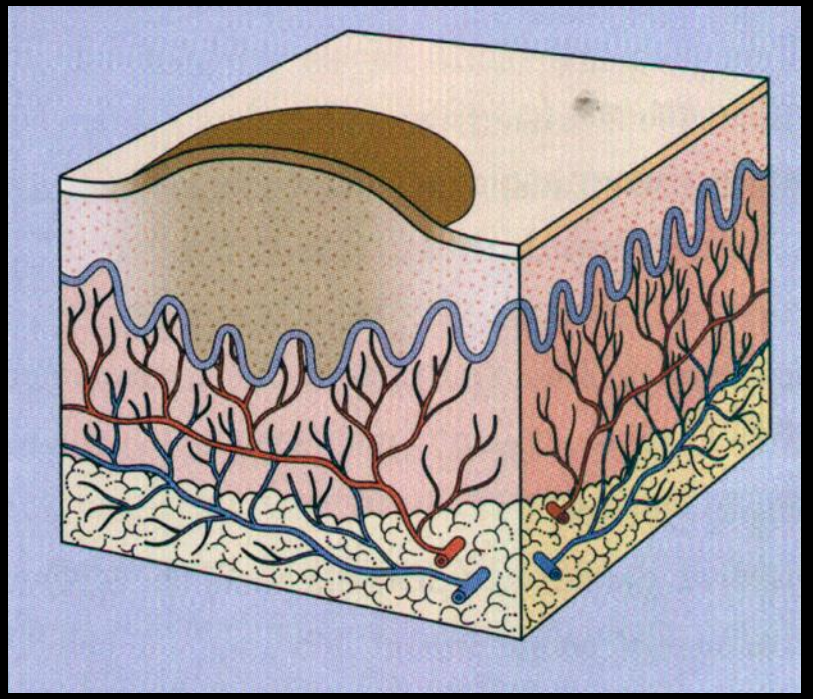
▼ RED AND WHITE TISSUE REACTIONS

- Autoreactive T lymphocytes cannot discriminate between inherent molecules of the body and foreign antigens.
- not one single peptide evokes the inflammatory response but several depending on the specificity of the autoreactive T lymphocyte.
- stress, may also be of importance
- negative social events some months before to the onset of the disease.

complicated :
to identify a single etiologic factor behind OLP

Oral Lichen Planus





Lichenoid reaction with a subepithelial infiltrate of inflammatory cells and liquefaction degeneration in the basal cell layer.

Oral Lichen Planus

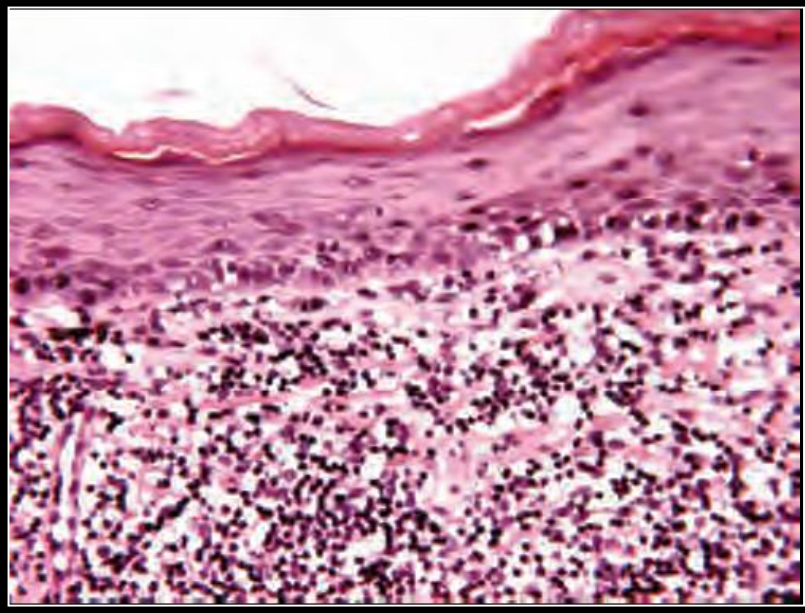
The histopathologic features of OLP

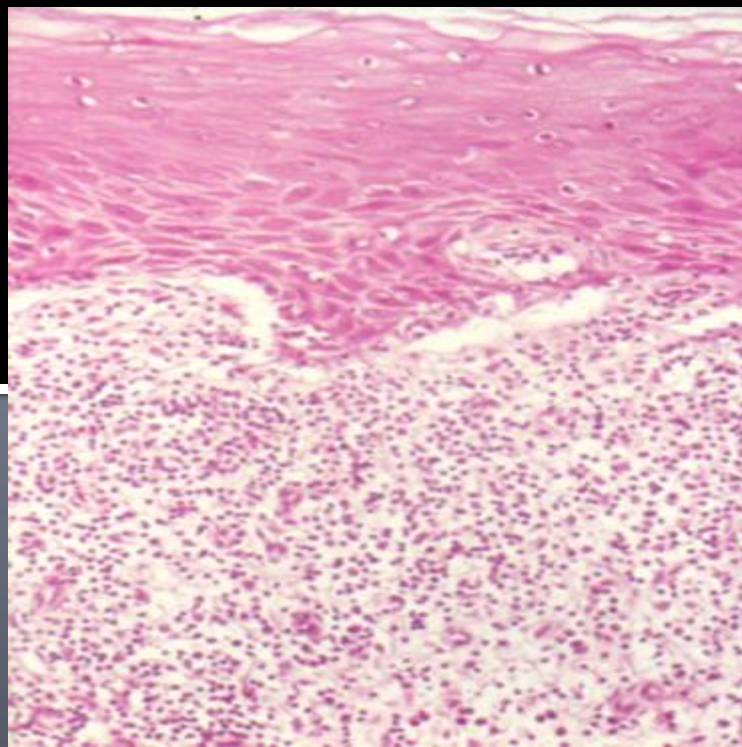
hyperparakeratosis
hyperorthokeratosis,
thickening of the granular cell layer

saw-toothed appearance to the rete pegs,
"liquefaction degeneration," or necrosis of the basal cell layer;

eosinophilic band beneath the basement membrane
fibrin covering the lamina propria.

dense subepithelial infiltrate of lymphocytes and macrophages is





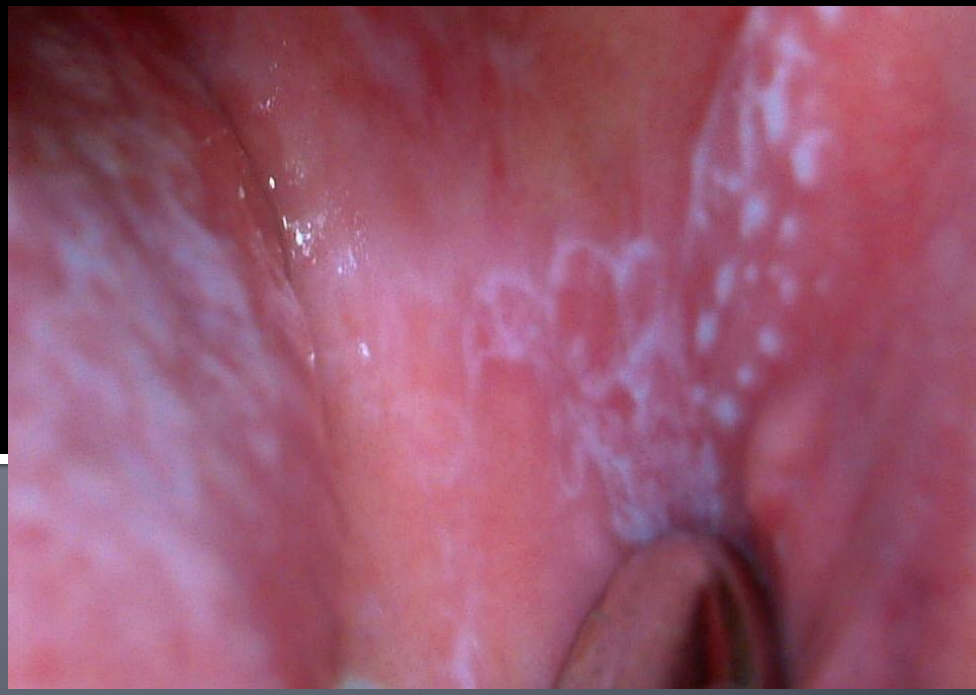
Oral Lichen Planus

Clinical Findings

OLP may contain both red and white elements

. The white and red components of the lesion :

- Reticulum
- Papules
- Plaque-like
- Bullous
- Erythematous
- Ulcerative

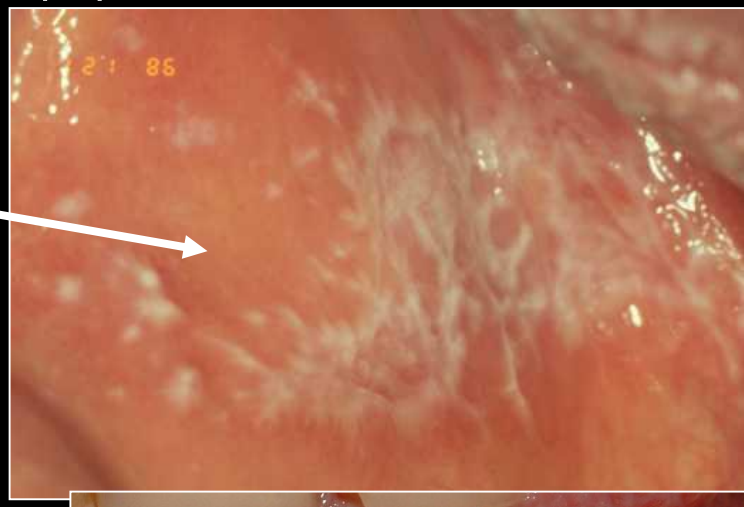


Oral Lichen Planus

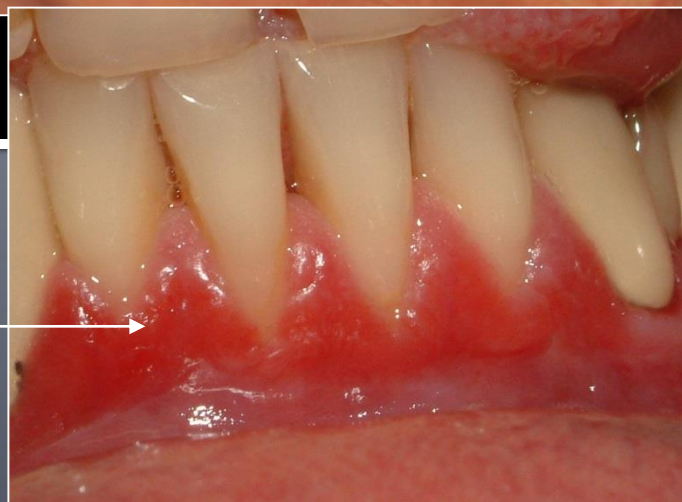
To establish a clinical diagnosis of OLP, reticular or papular textures have to be present.

in addition,

plaque-like,
bullous,
erythematous,
ulcerative areas are can be present,

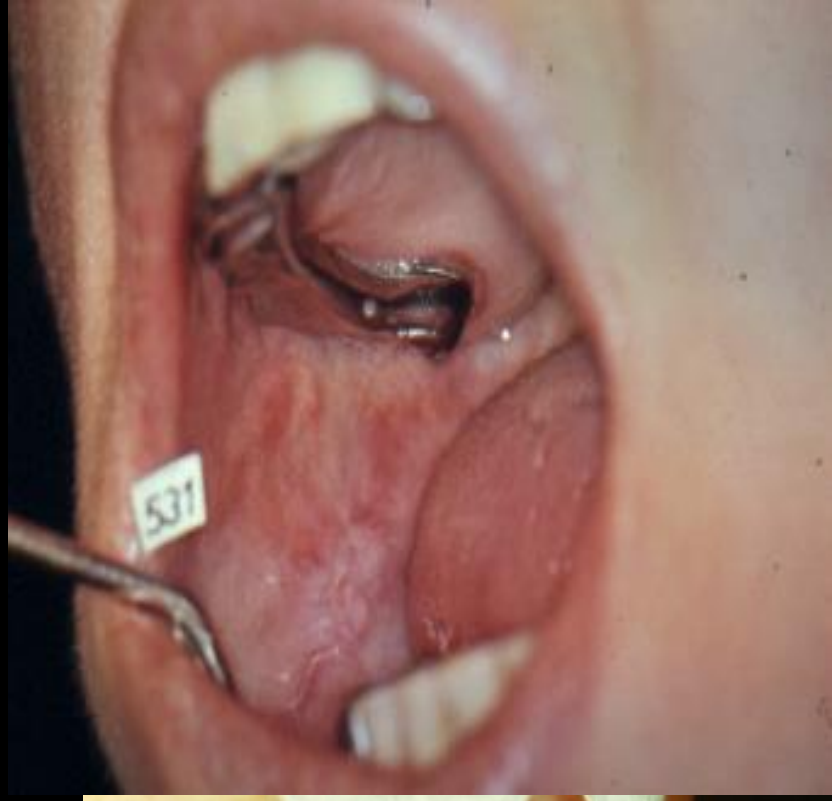


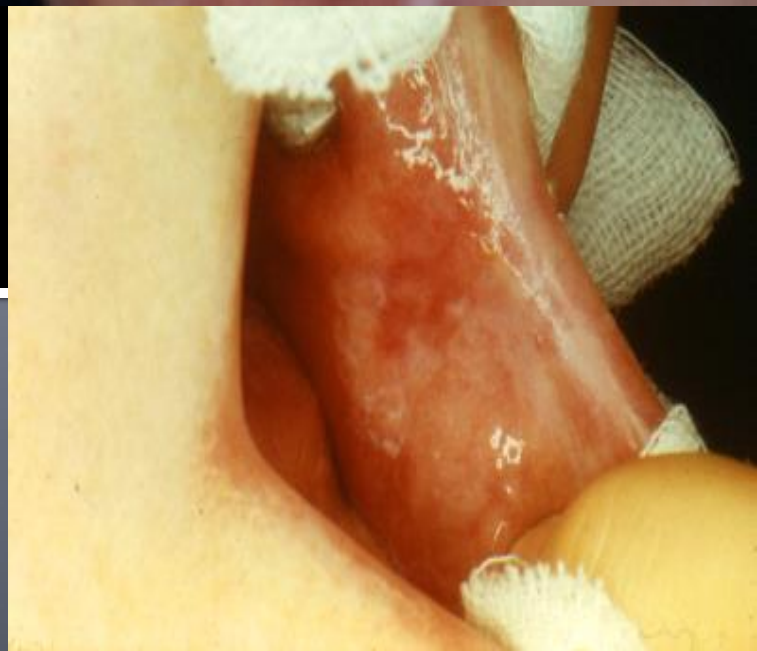
the OLP confined to the gingiva may be entirely erythematous, with no reticular or papular elements



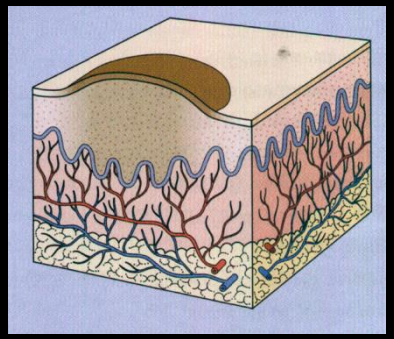
Oral Lichen Planus

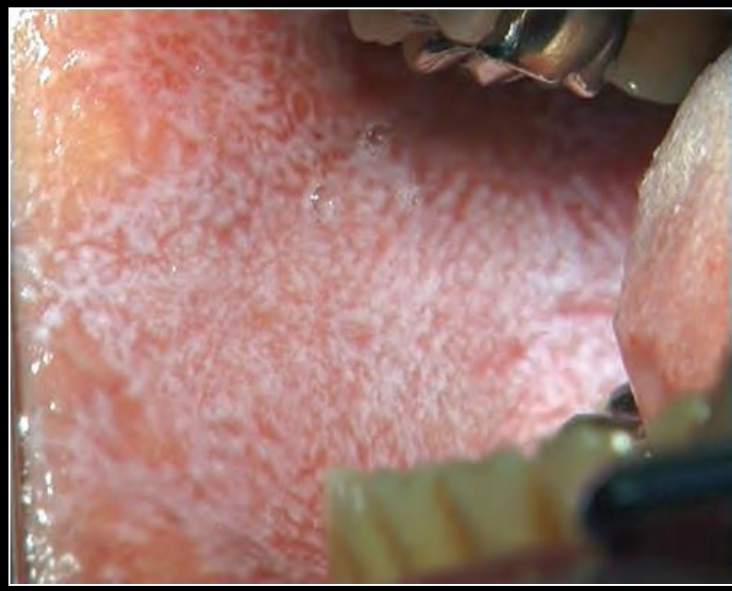
- Papular
- Reticular
- Plaque-like
- Bullous
- Atrophic
- Exulcerant





Oral Lichen Planus

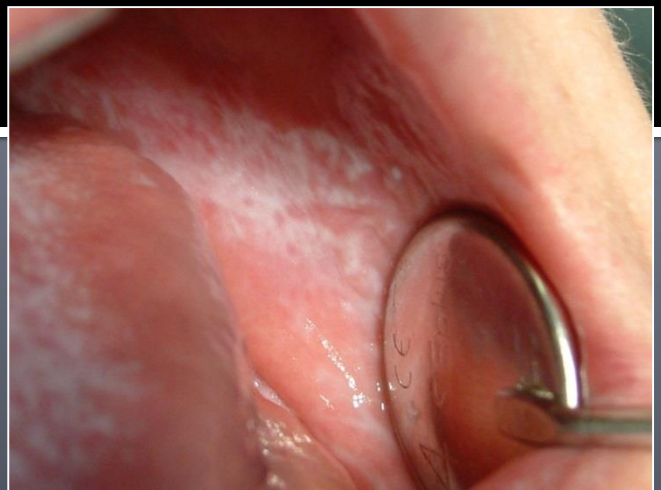


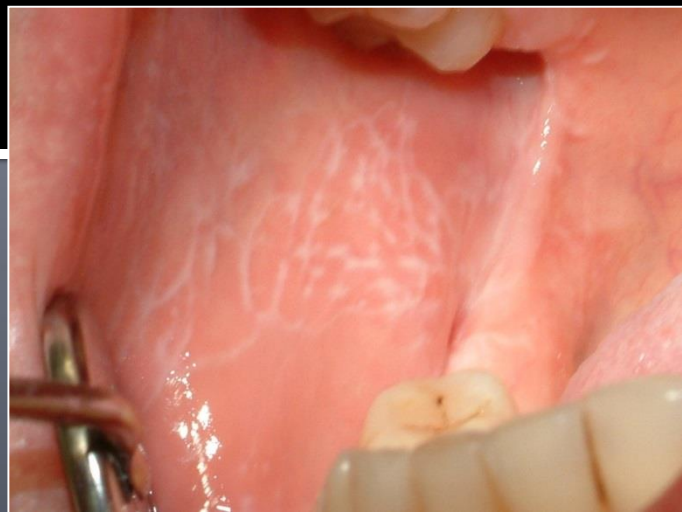


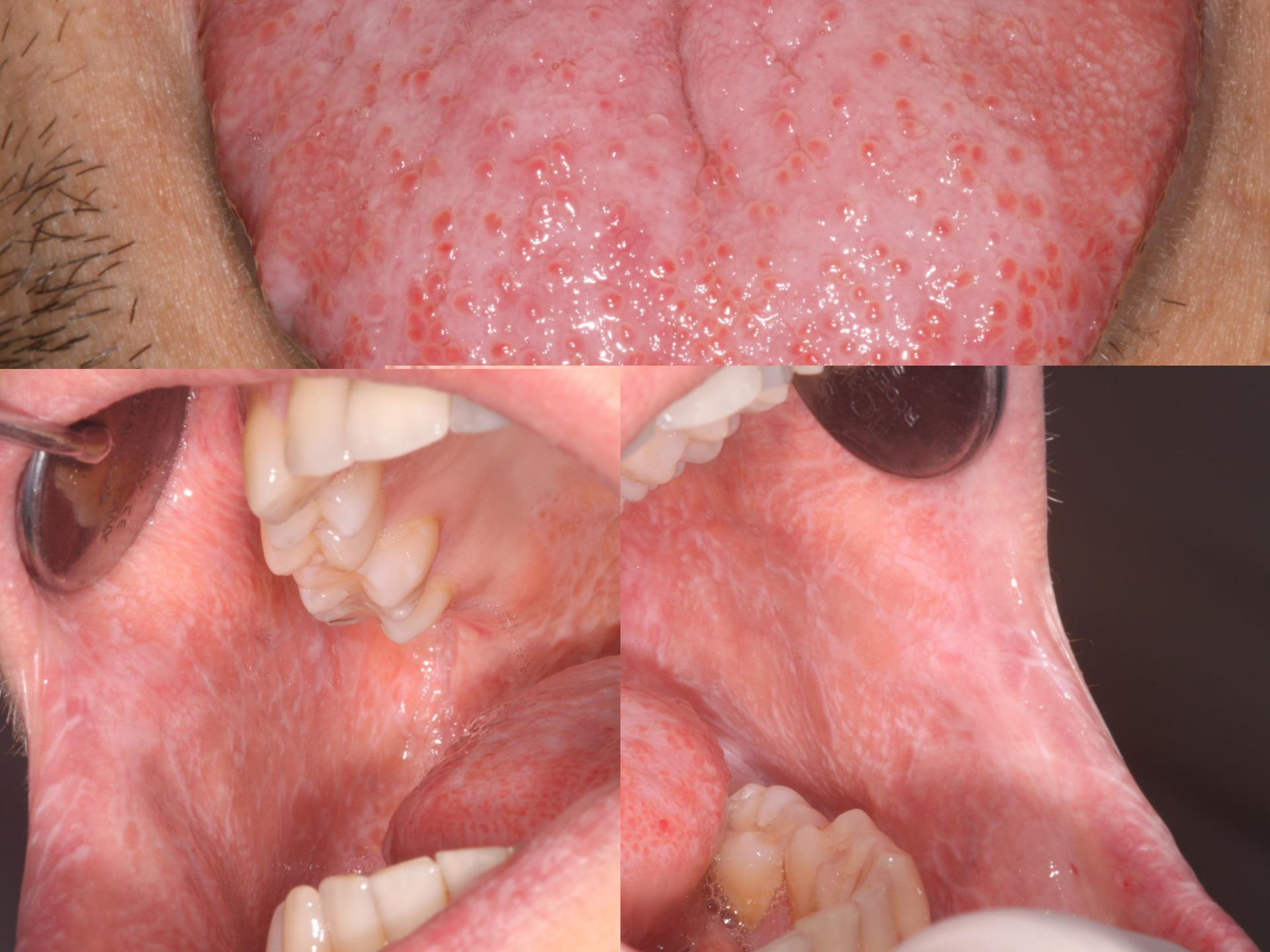
The papular type of OLP

is usually present in the initial phase of the disease characterized by small white dots,

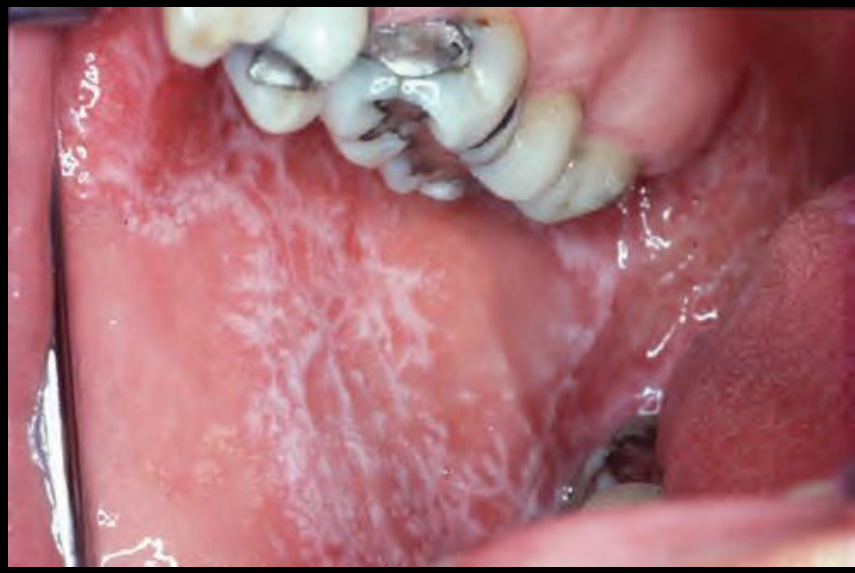
the papular elements merge with striae .







Oral Lichen Planus



The reticular form of OLP

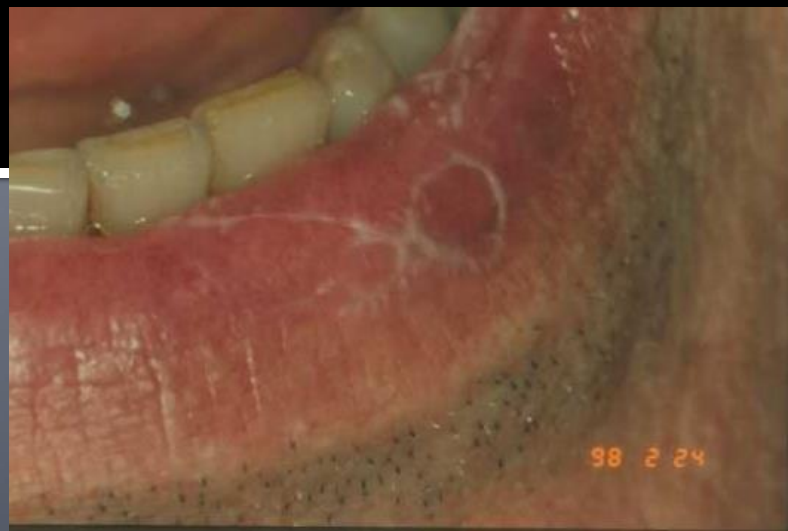
is characterized by fine white lines or striae

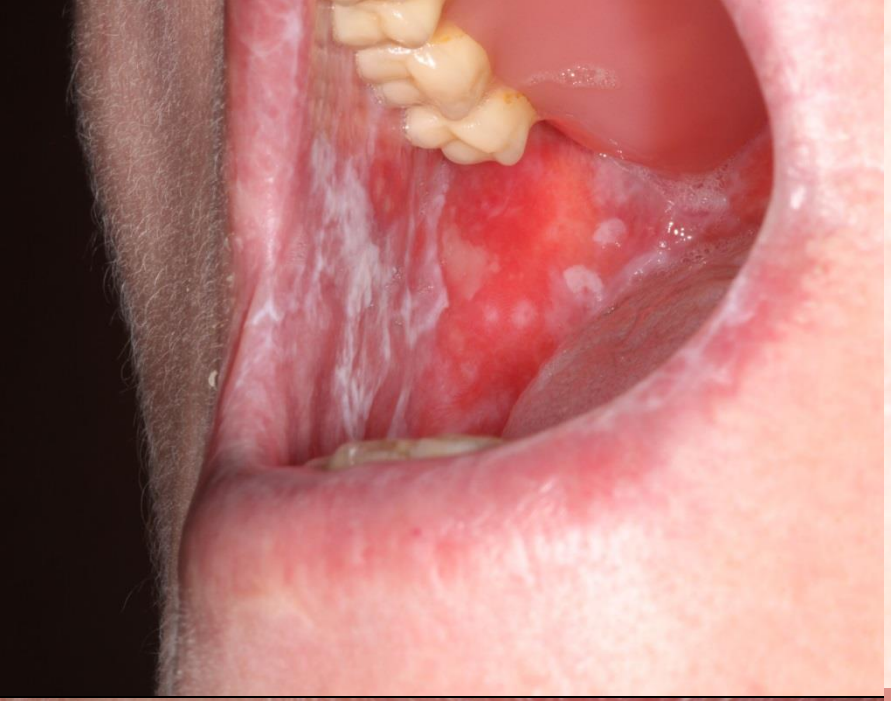
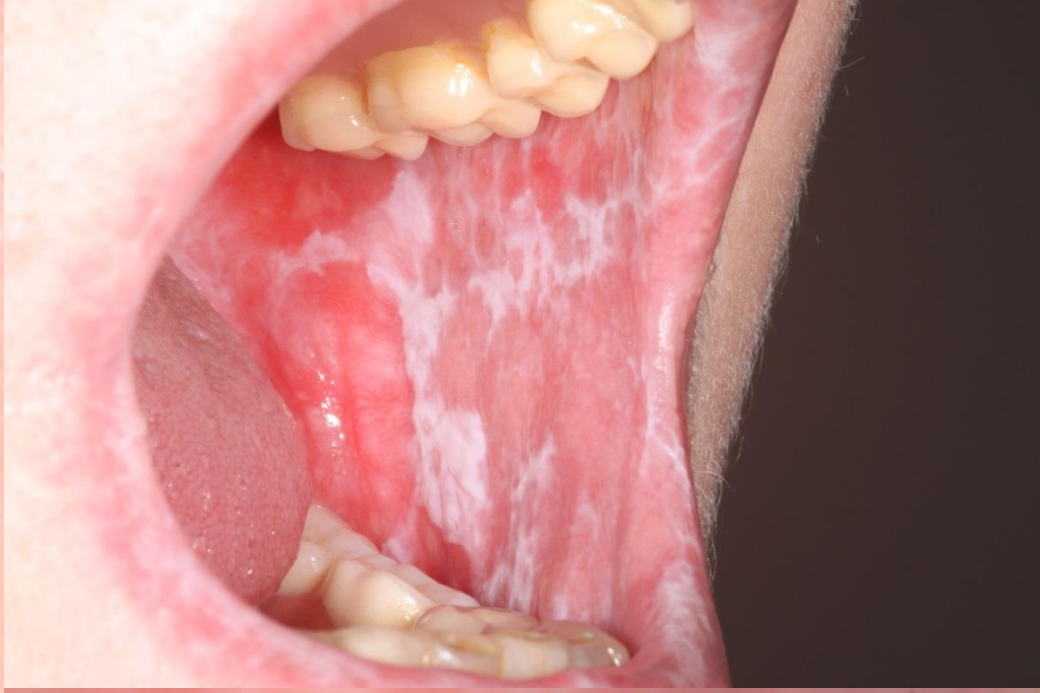
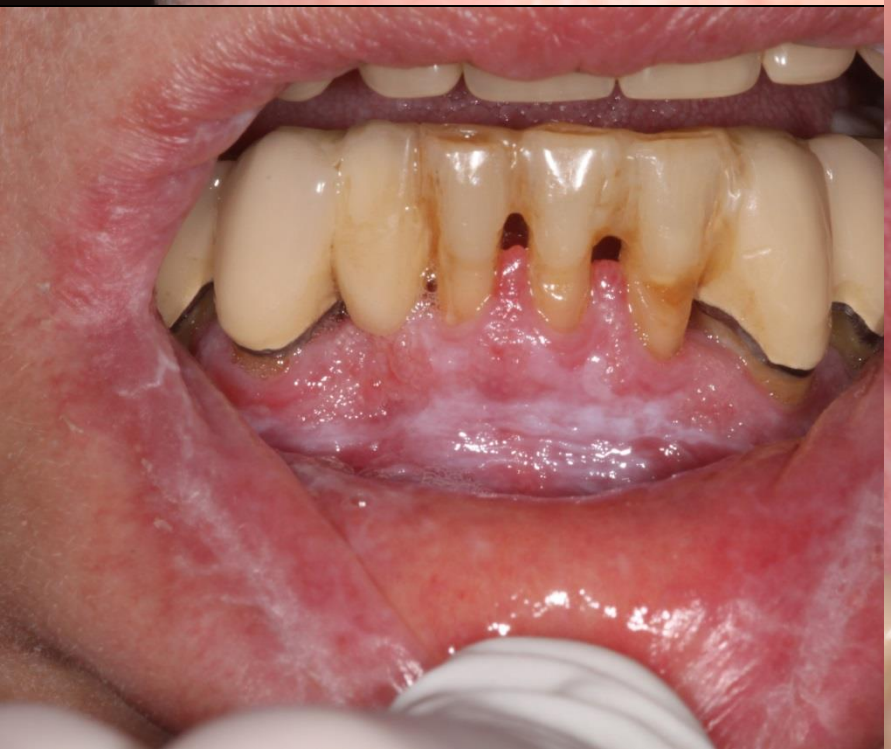
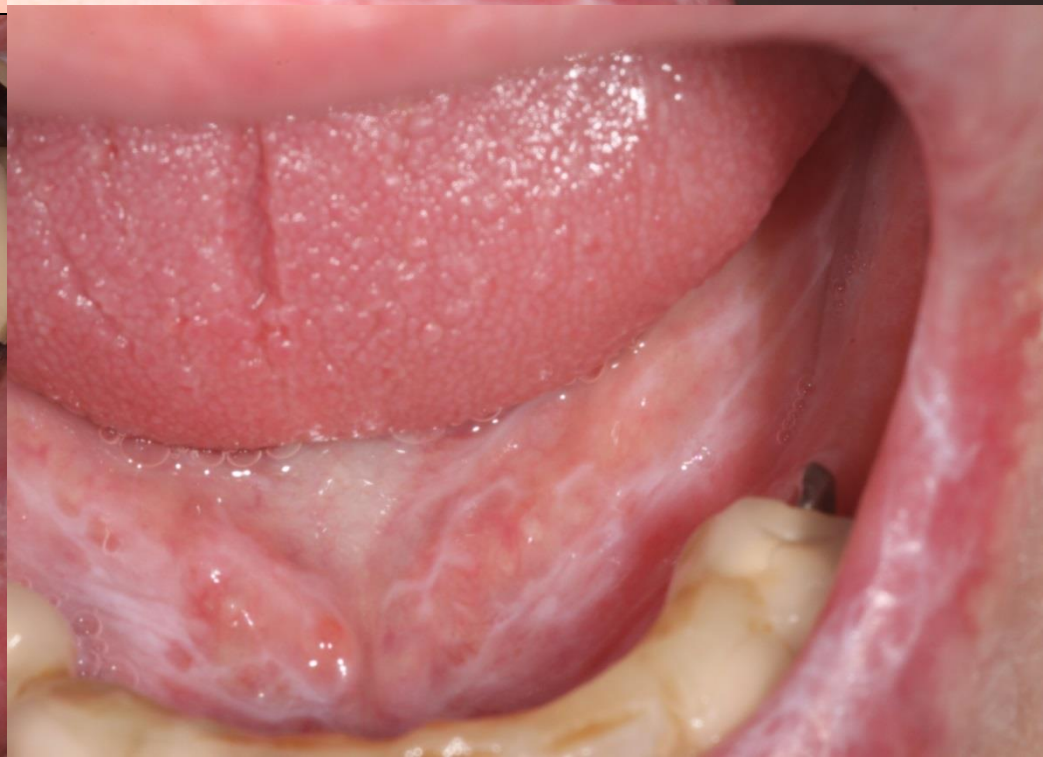
The striae may form a network

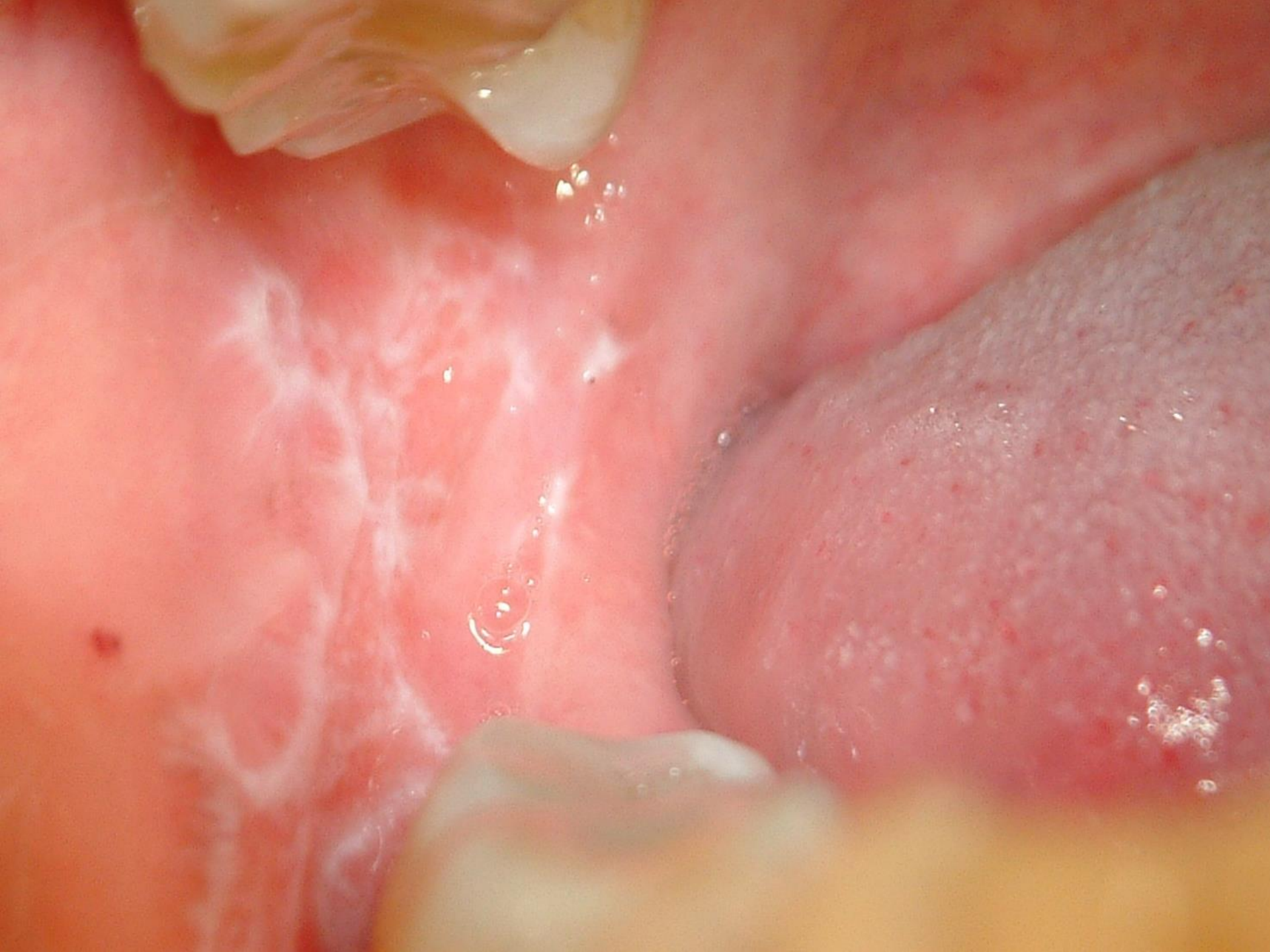
can also show annular (circular) patterns.

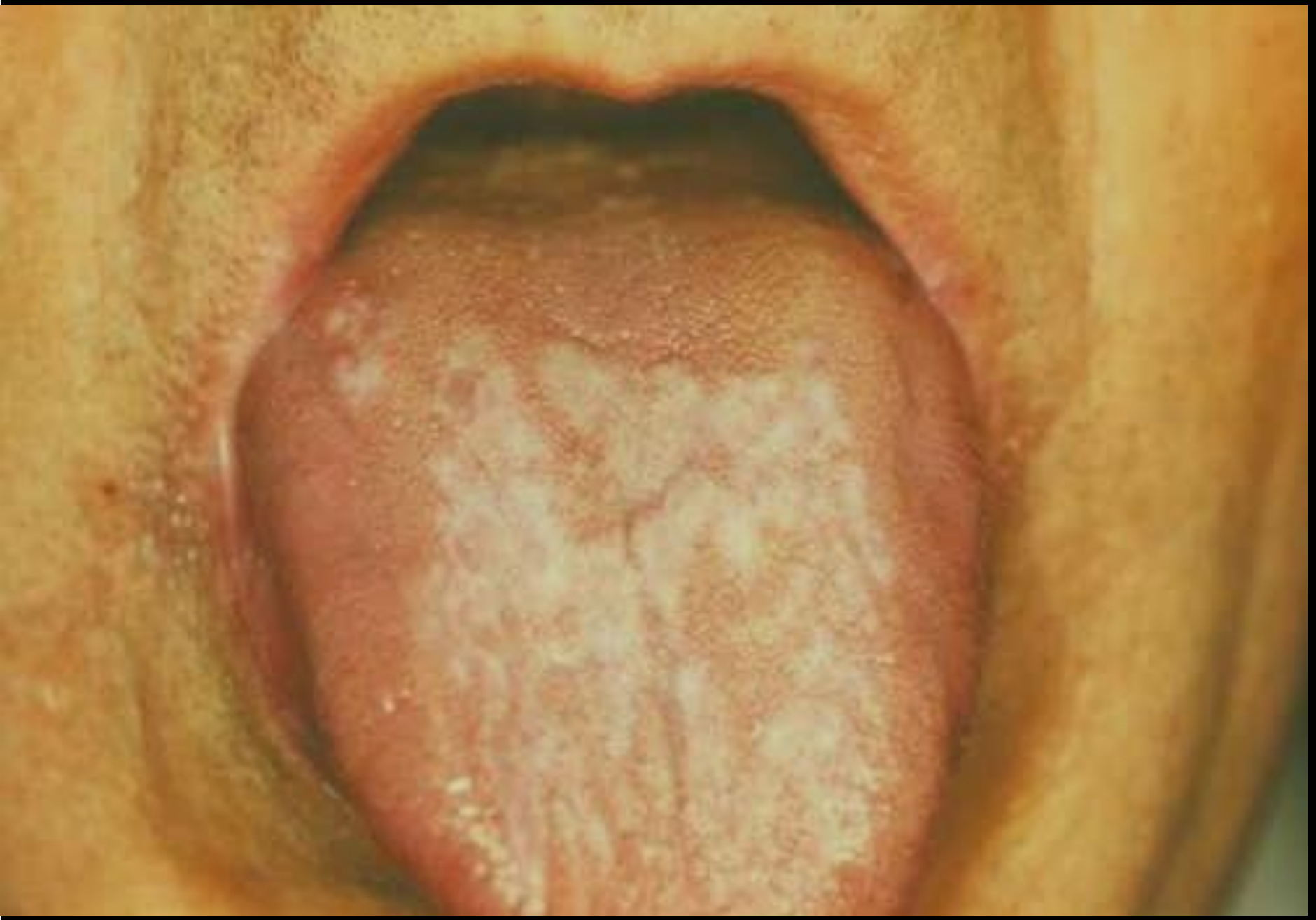
Most frequently in the buccal mucosa

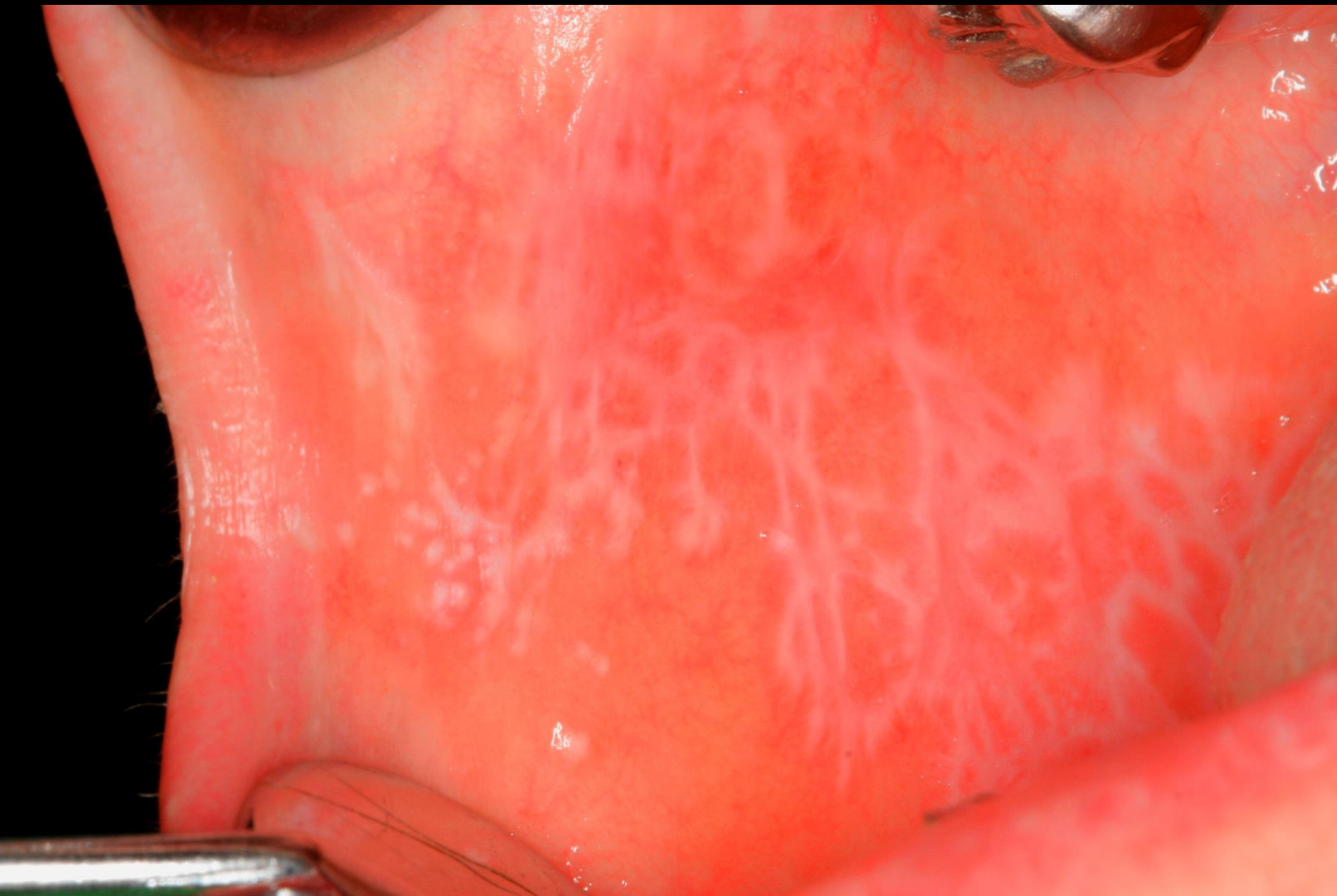
Reticular OLP can be observed at the vermilion border.

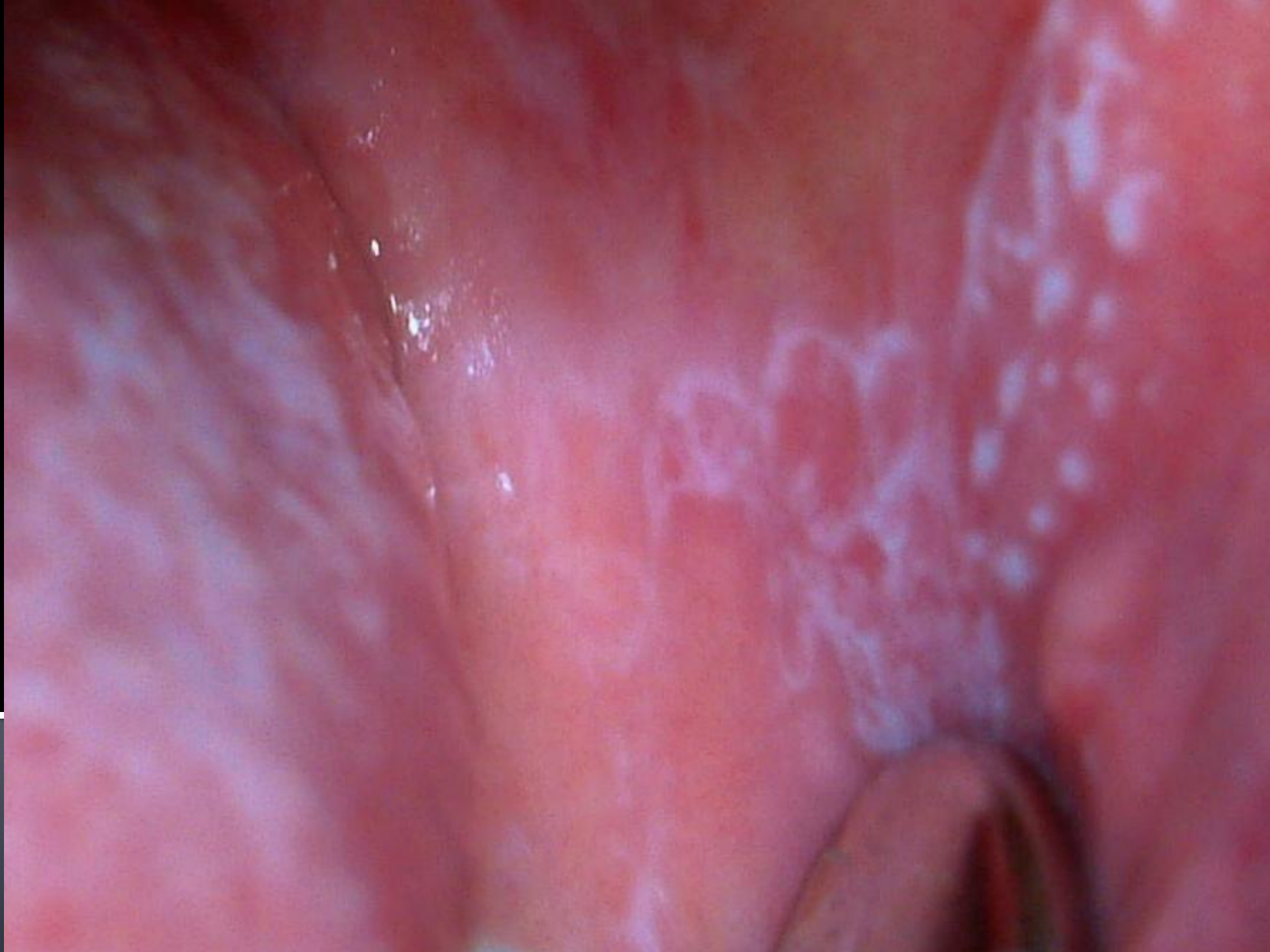


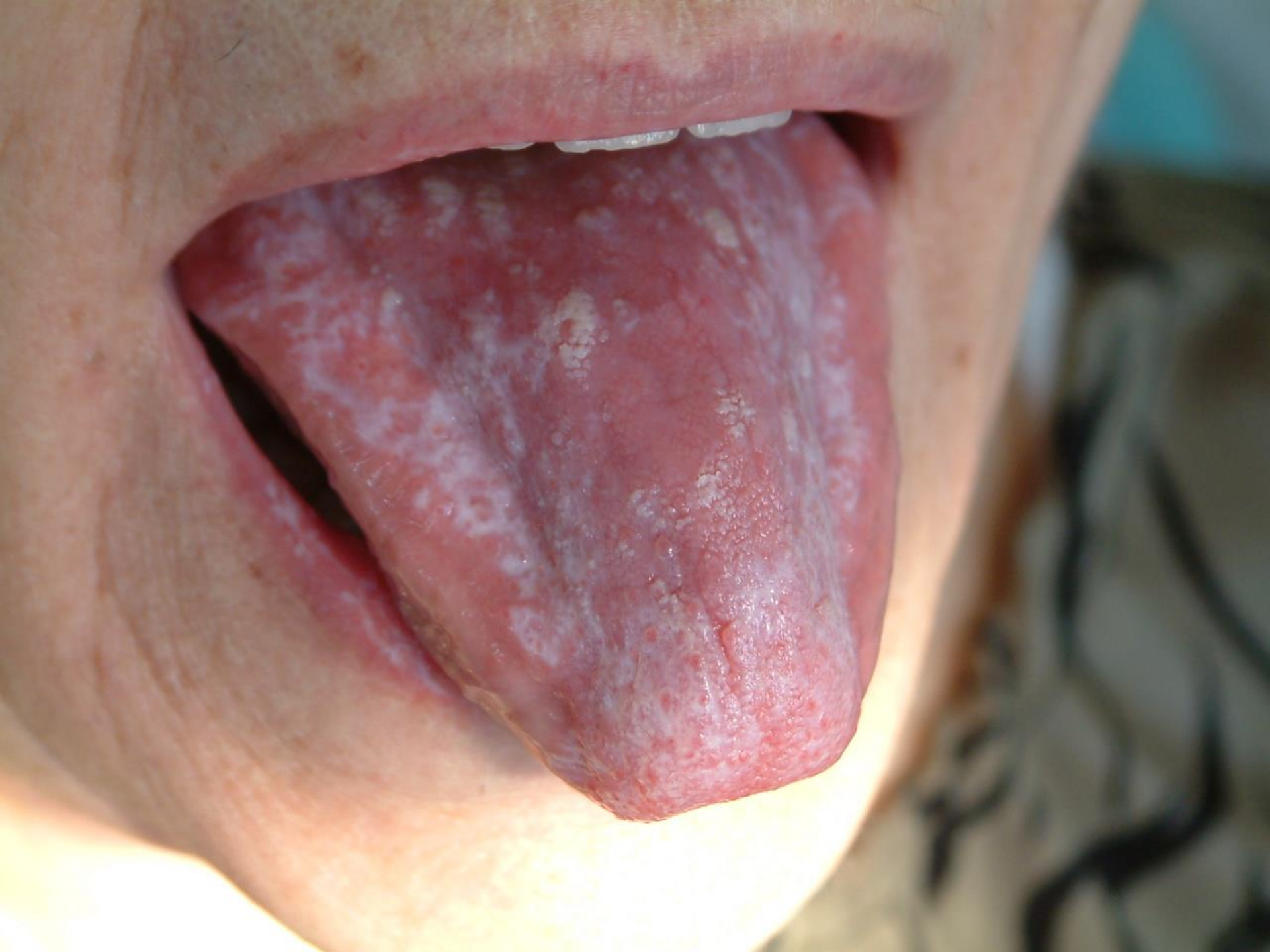





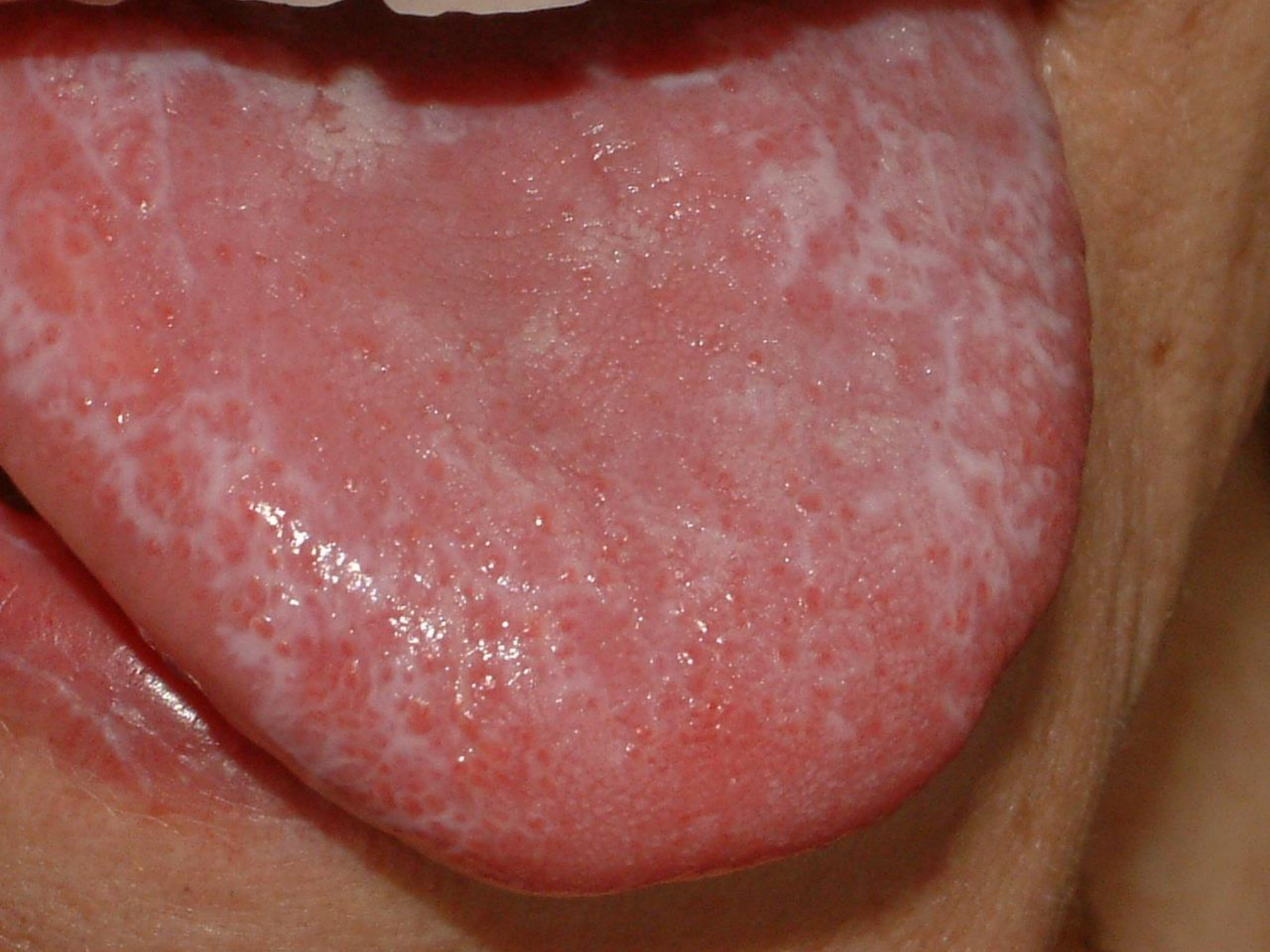


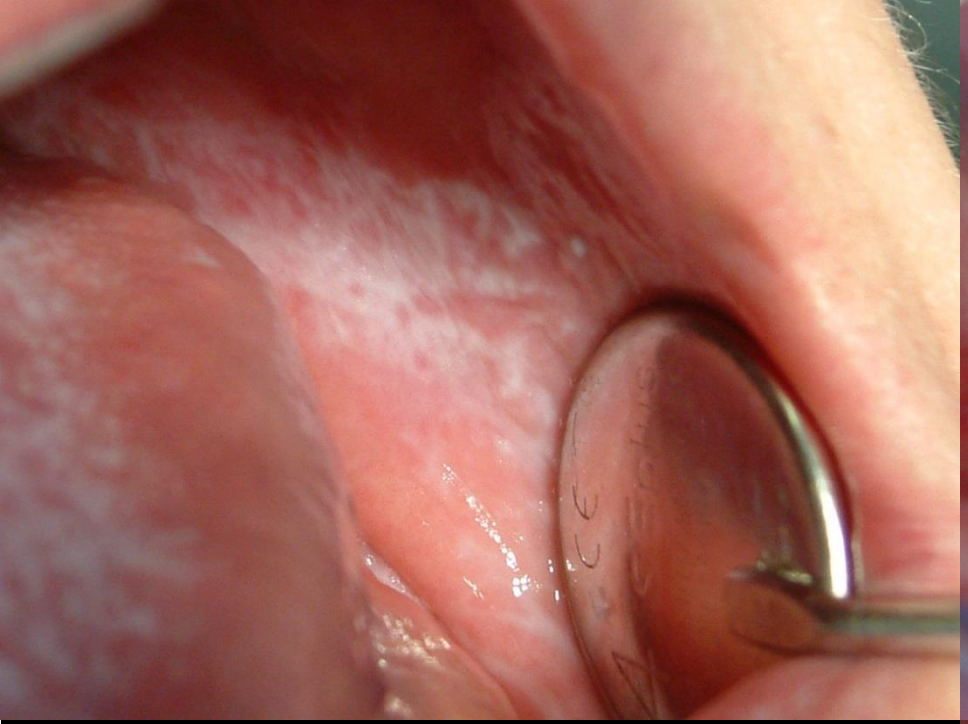
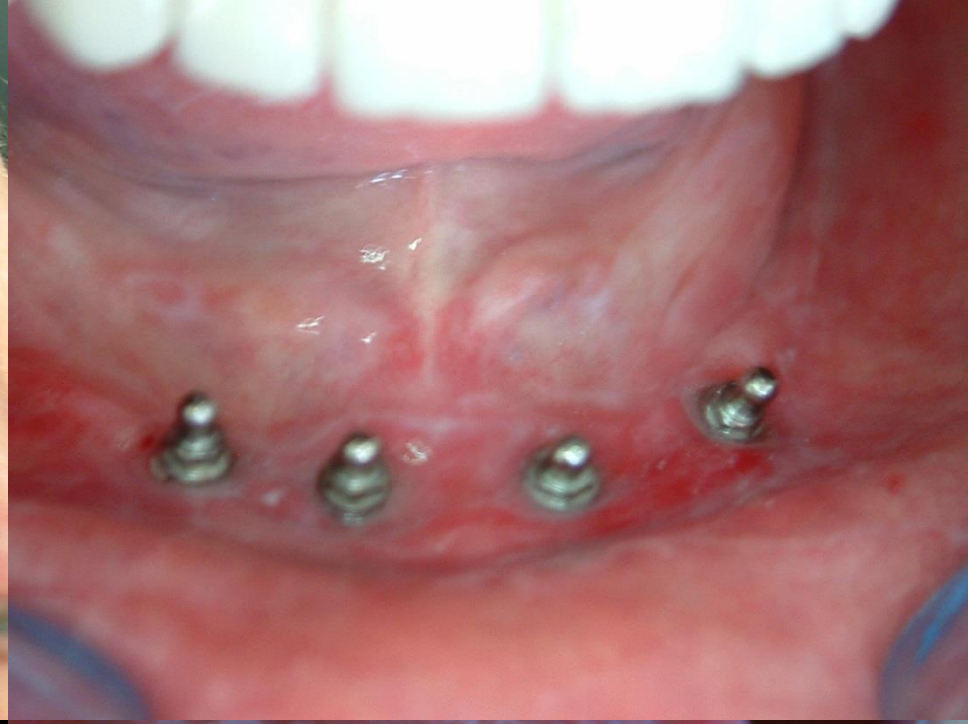
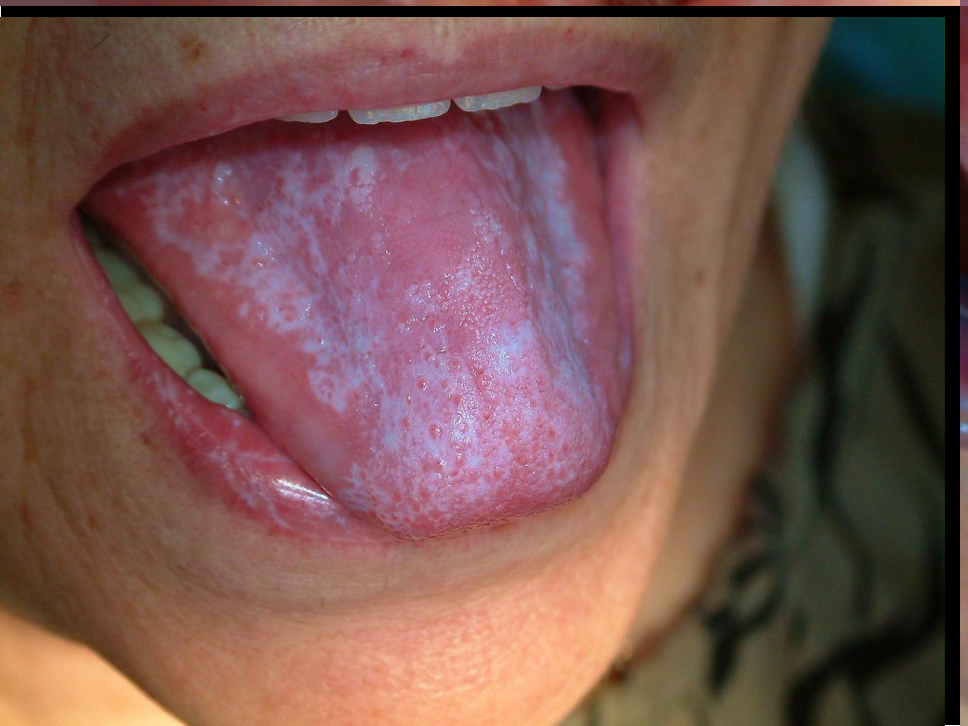
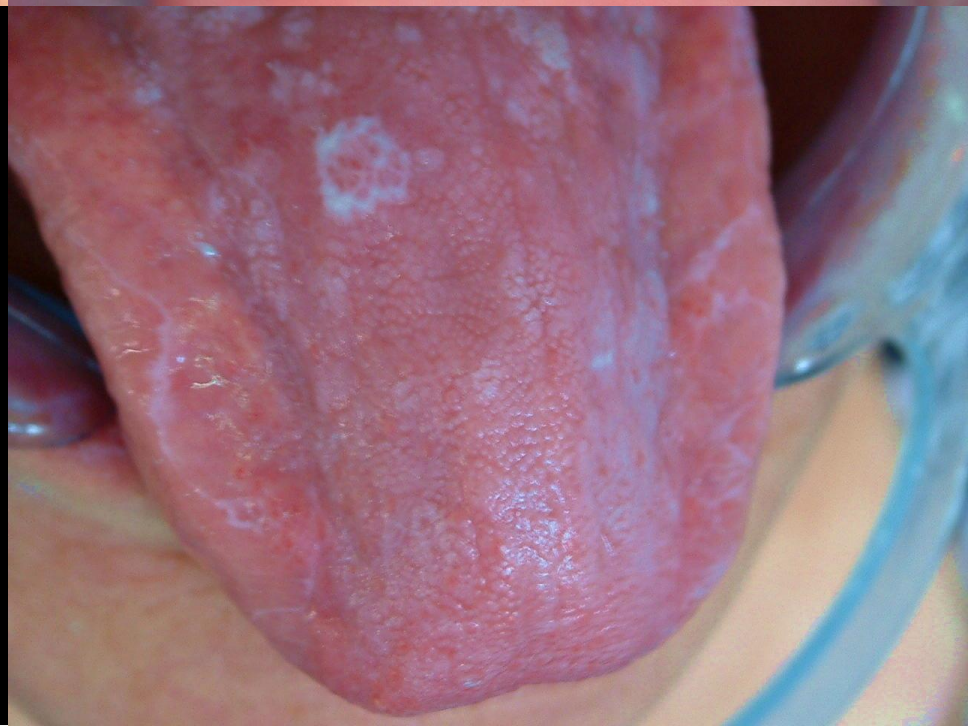


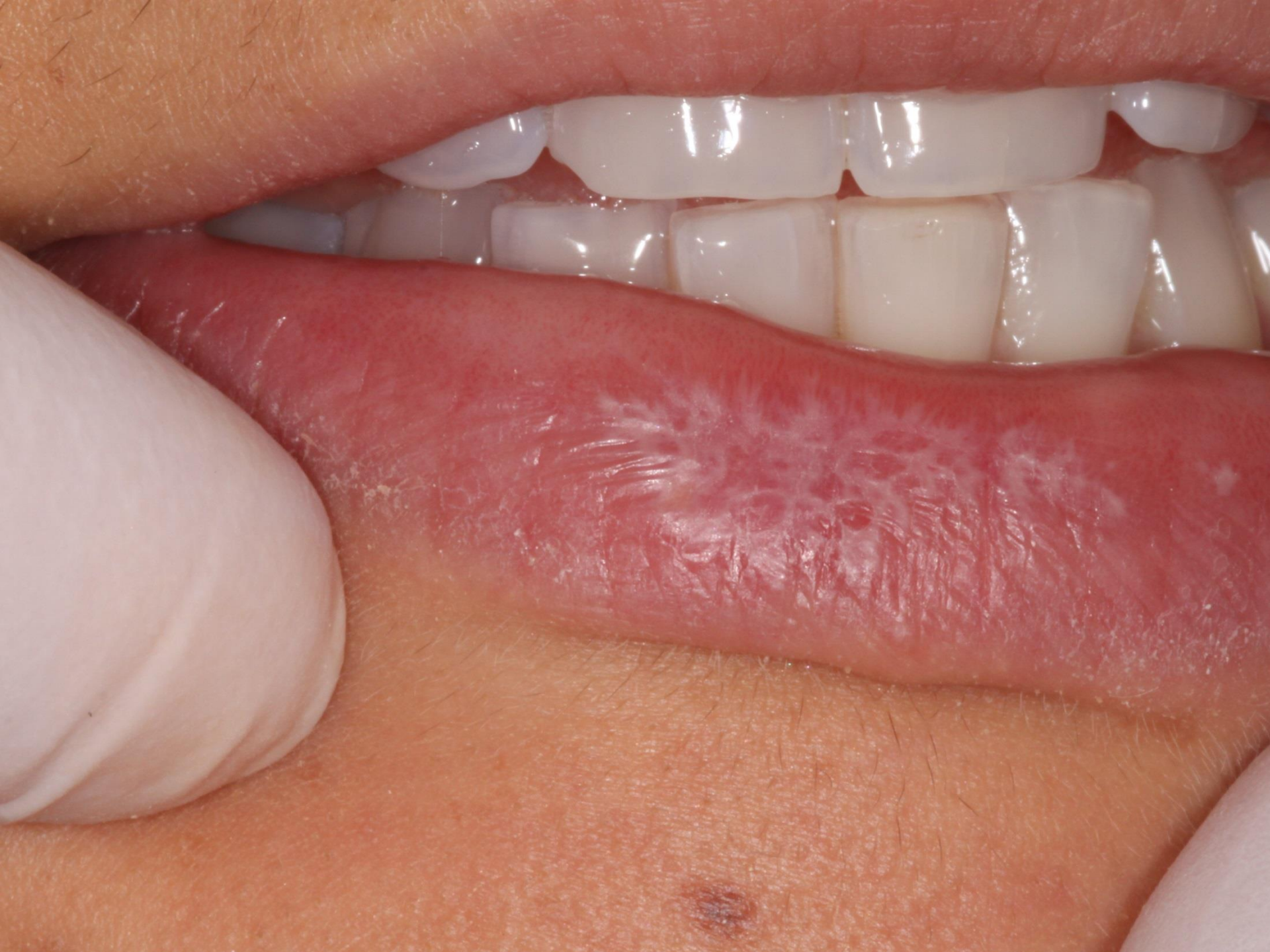




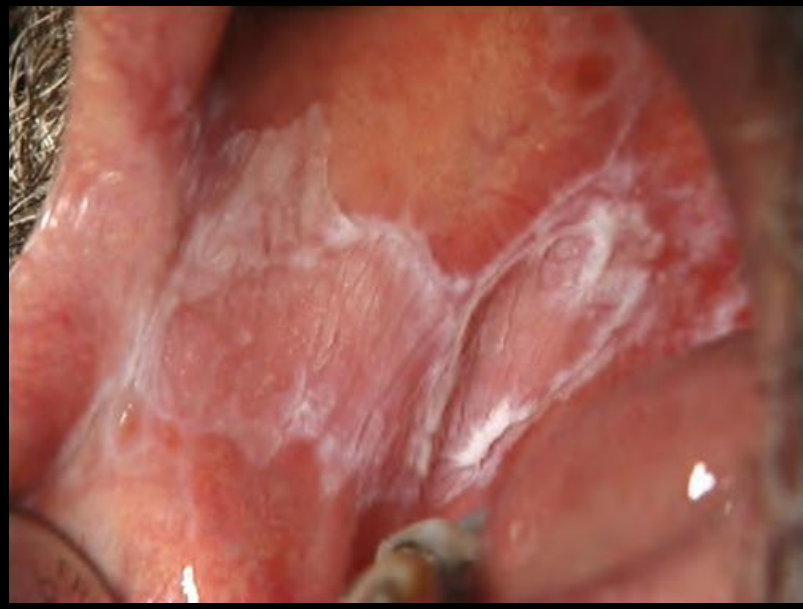




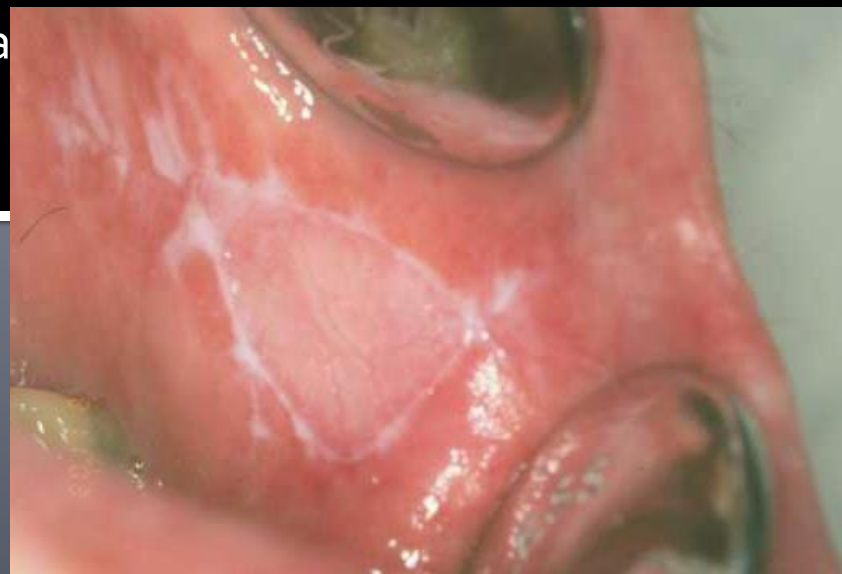


Oral Lichen Planus

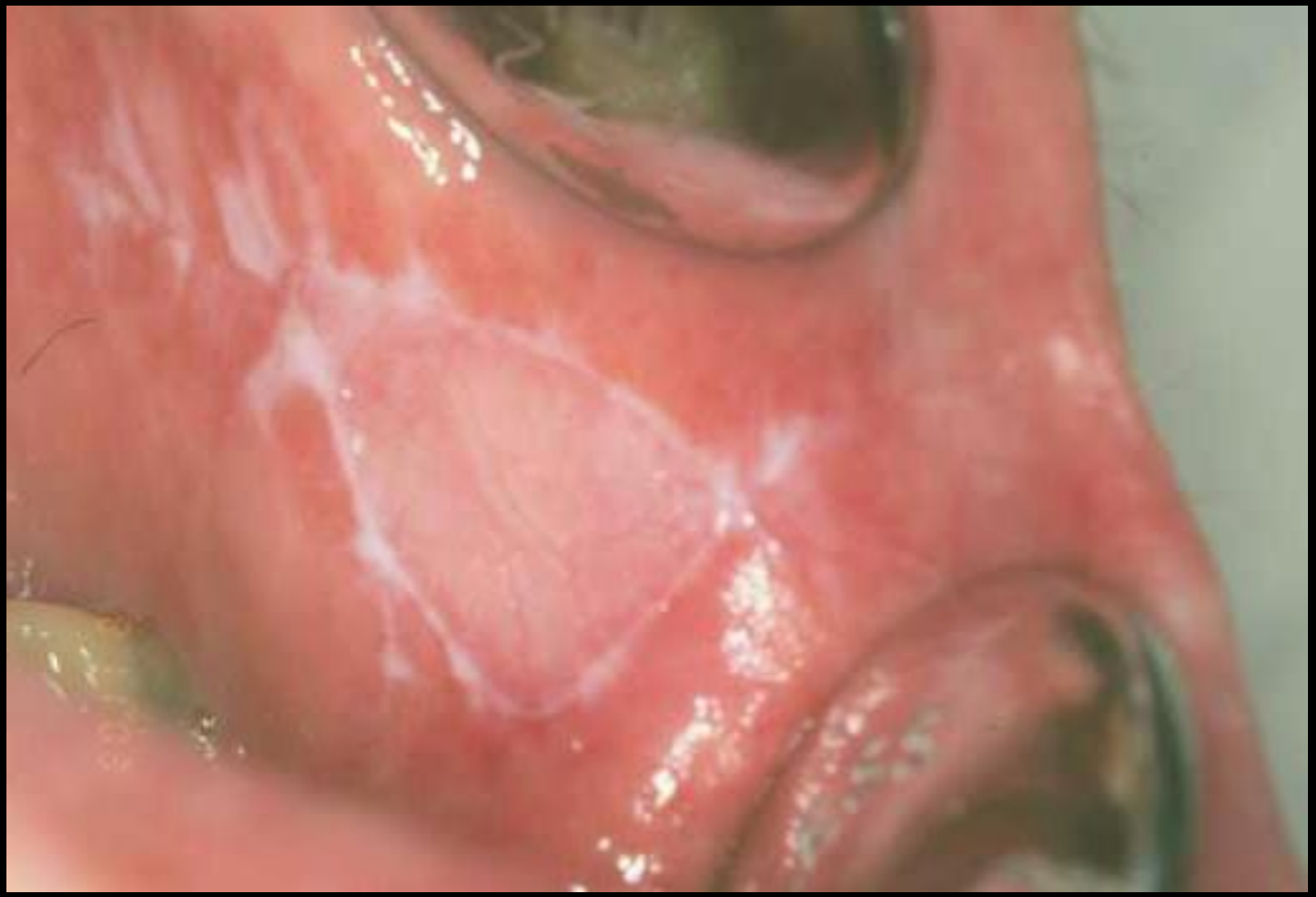


Plaque-type OLP

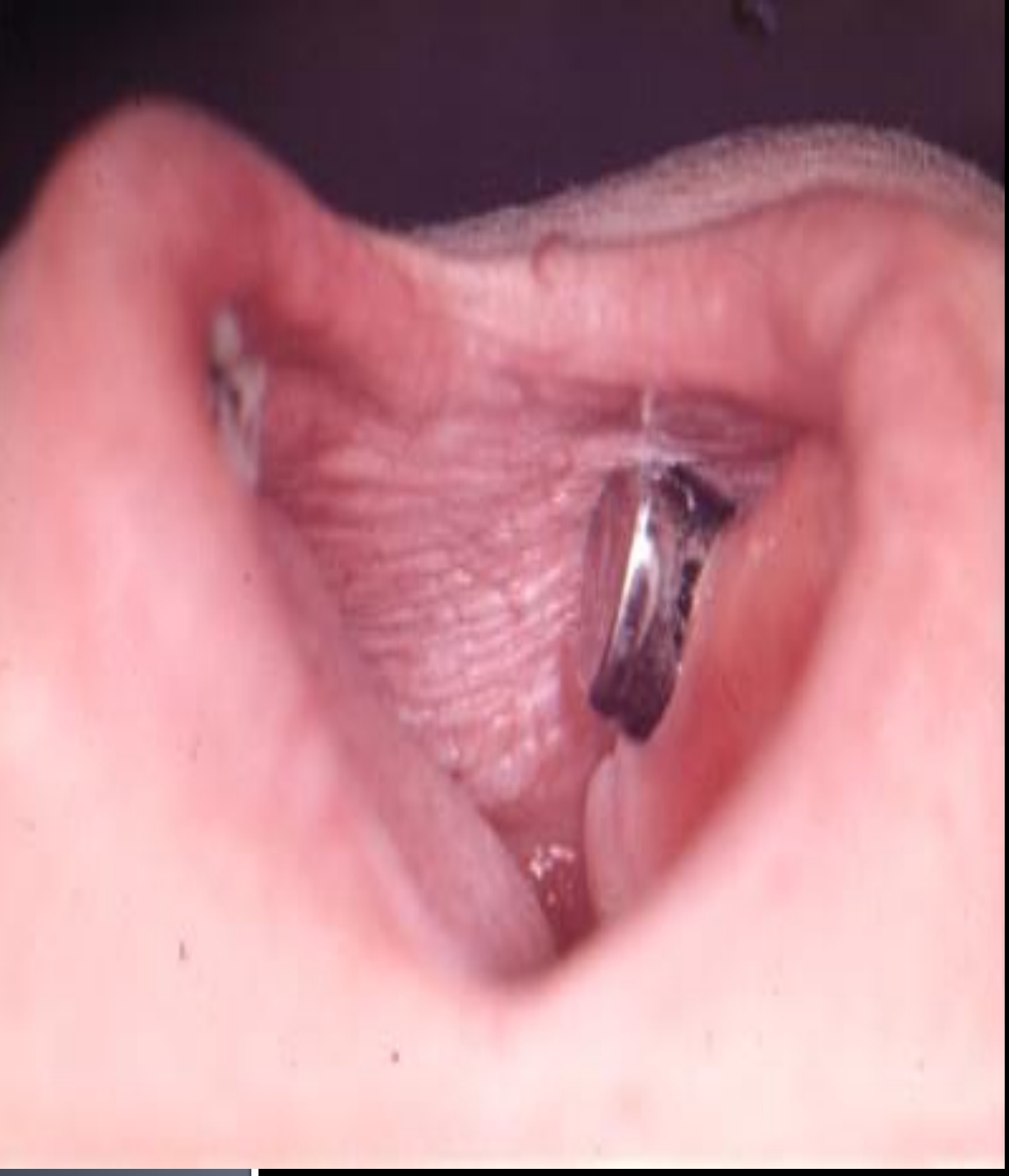
- Homogeneous well-demarcated white plaque often, surrounded by striae to homogeneous oral leukoplakias.
- Plaque-type lesions may clinically be similar
- The difference is the presence of reticular lesions
- In smokers following cessation, the plaque ma
- into the reticular type of OLP.



plaque-like OLP may transform into oral squamous cell carcinomas



plaque like lichen?? leukoplakia simplex + lichen ??

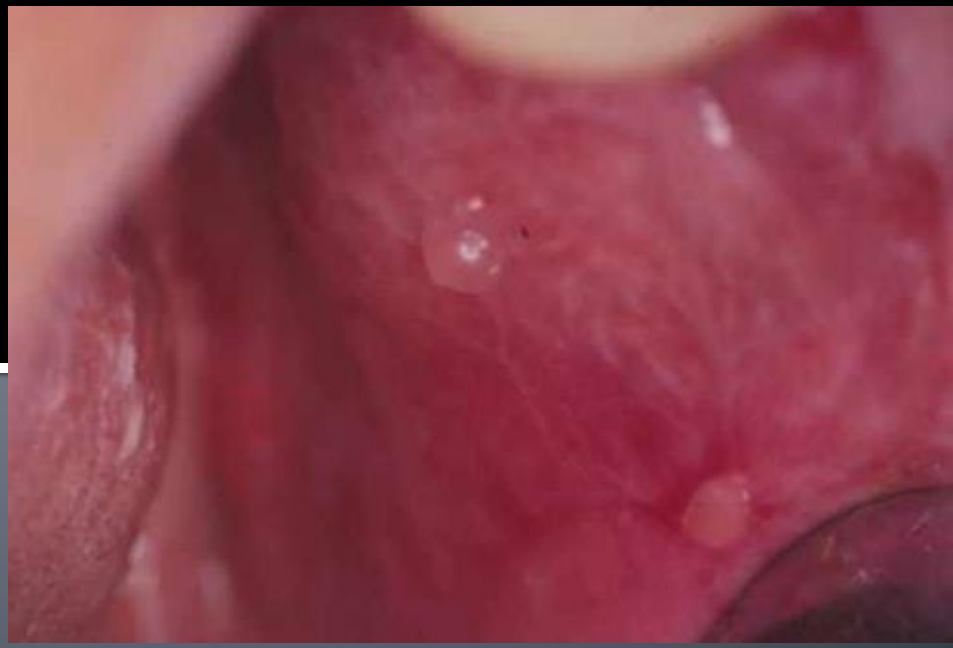


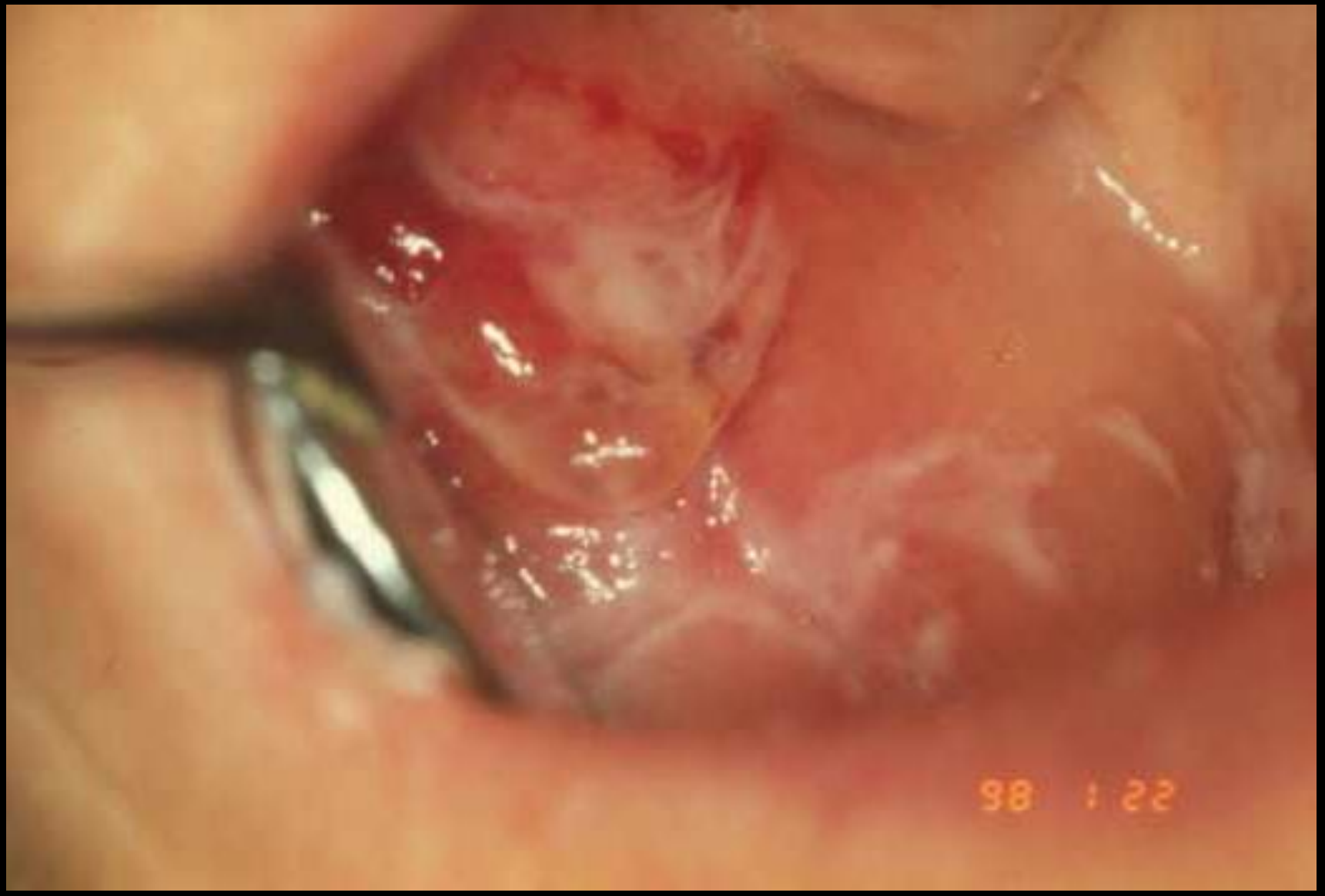
Oral Lichen Planus

bullous form

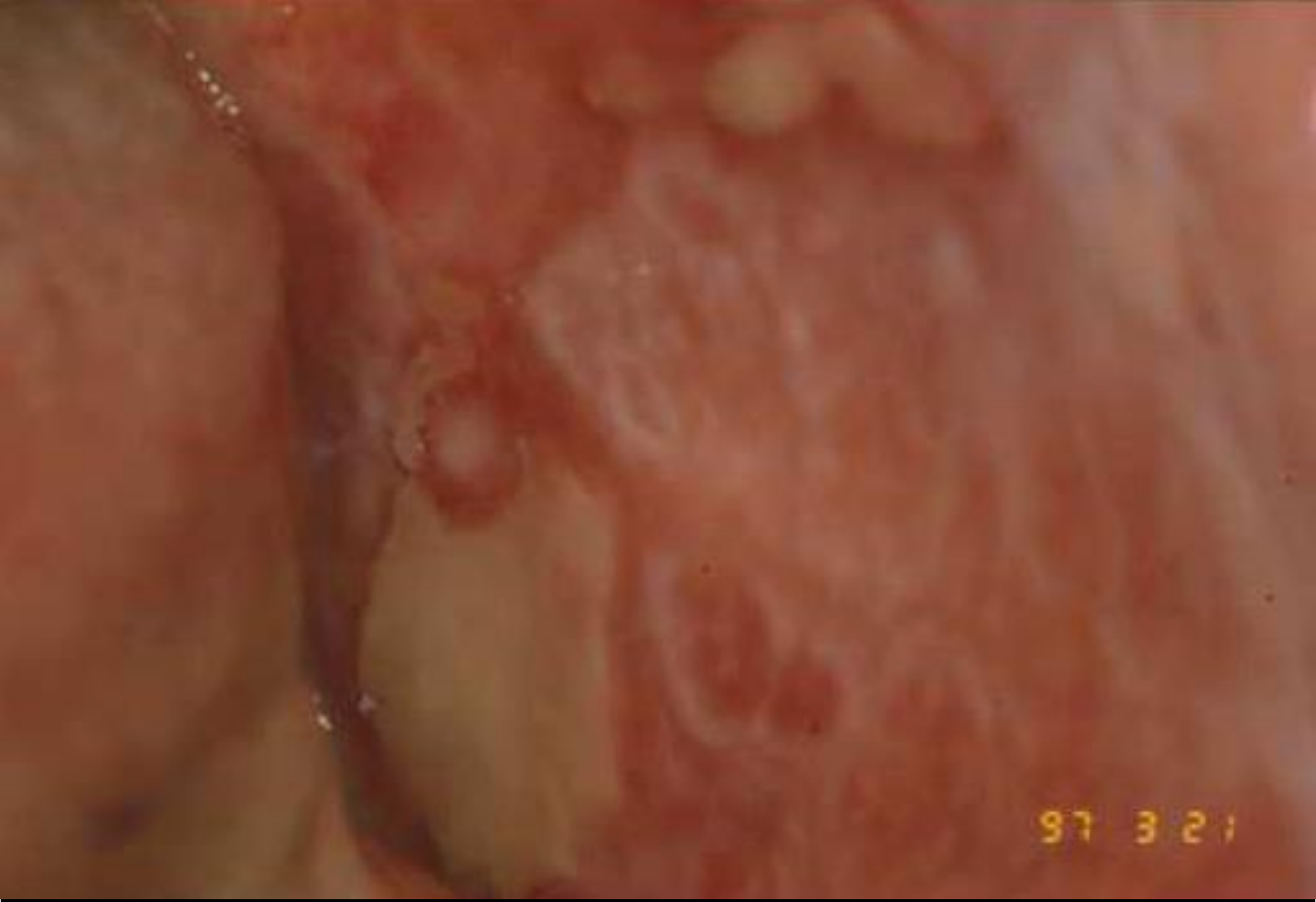
Typically, the reticular, papular, and plaque-like forms of OLP are asymptomatic, although the patient may experience a feeling of roughness.

The bullous form is very unusual but may appear as bullous strucures surrounded by a reticular network.

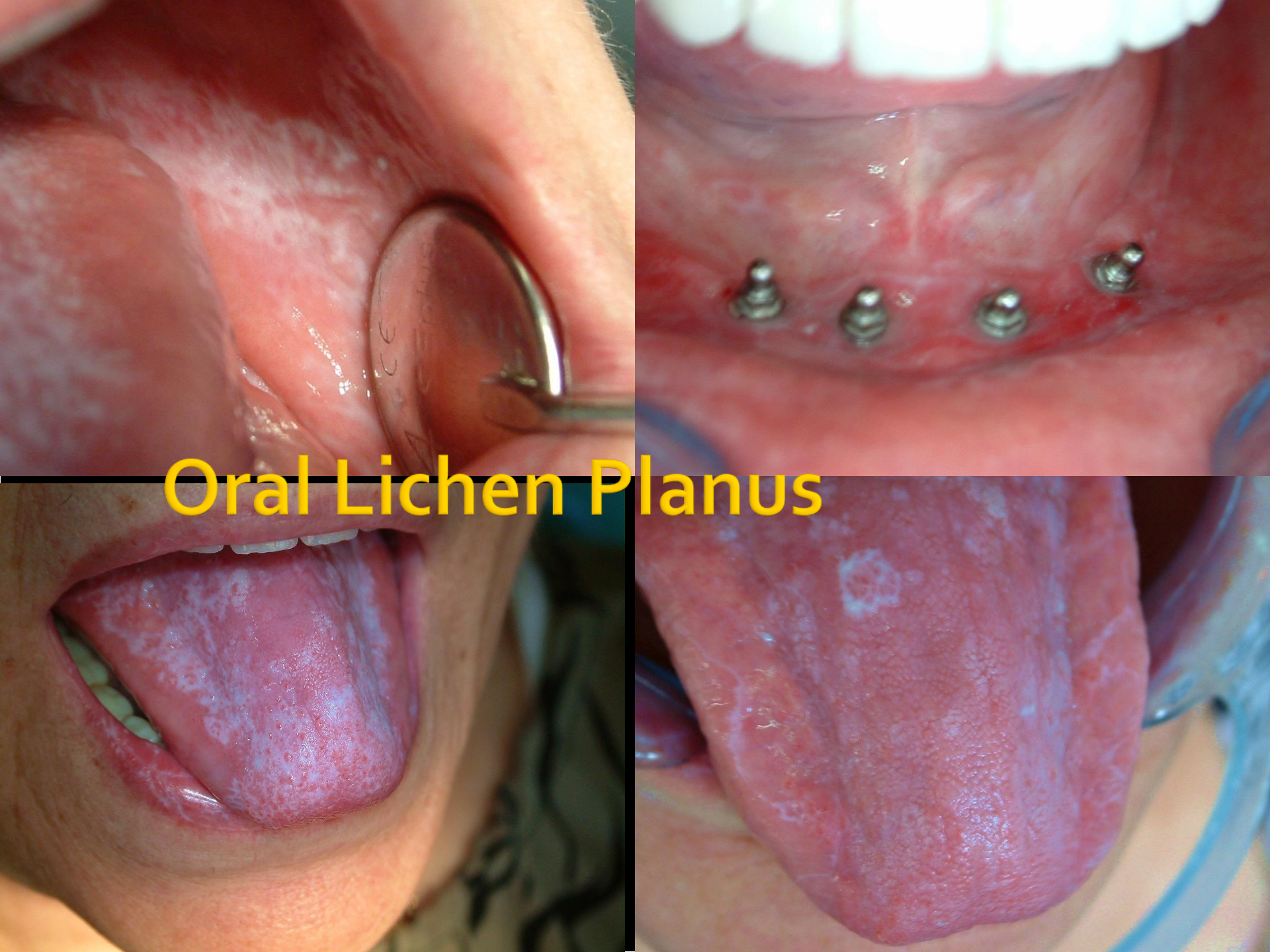




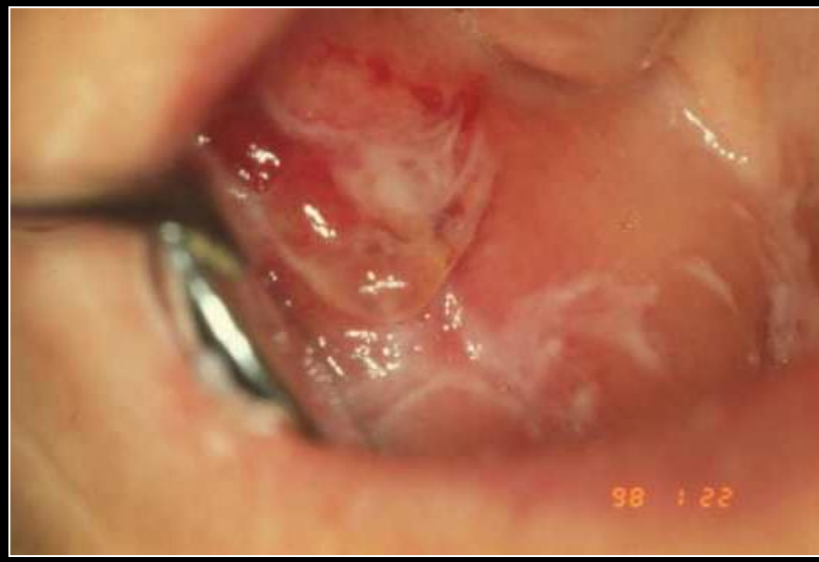
BULLOSUS LICHEN



COLLABALT BULLA A LICHEN RAJZOLAT FELETT


Oral Lichen Planus

Oral Lichen Planus

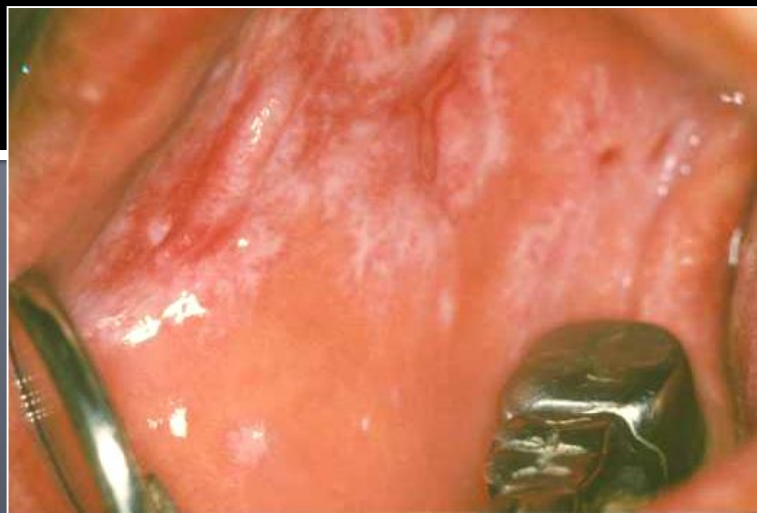


Ulcerative lesions

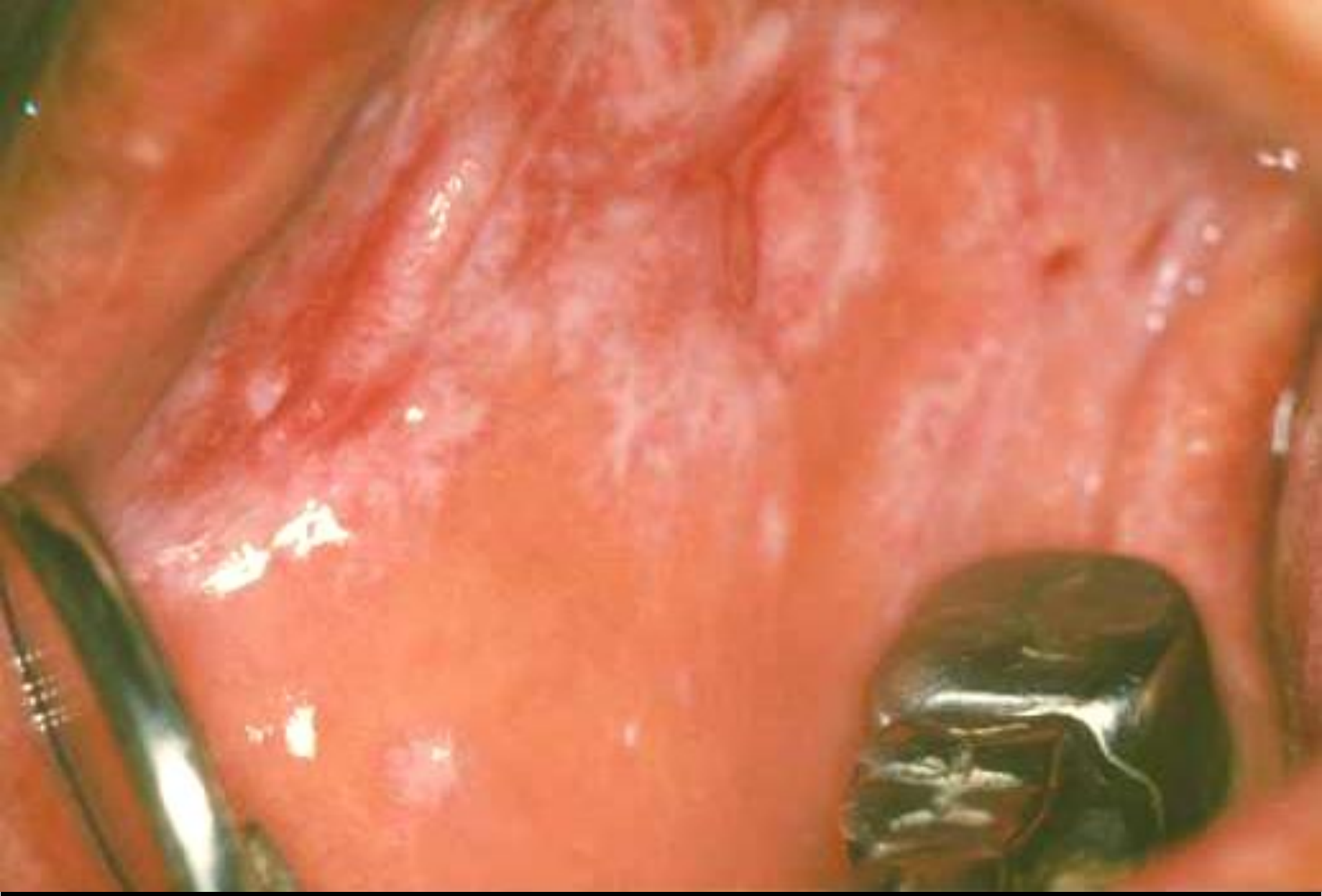
are the most disabling form of OLP

Clinically, the fibrin-coated ulcers are surrounded by an erythematous zone with white striae.

This appearance may reflect a gradient of the intensity of subepithelial inflammation



Patient complains of pain sensation in conjunction with food intake.



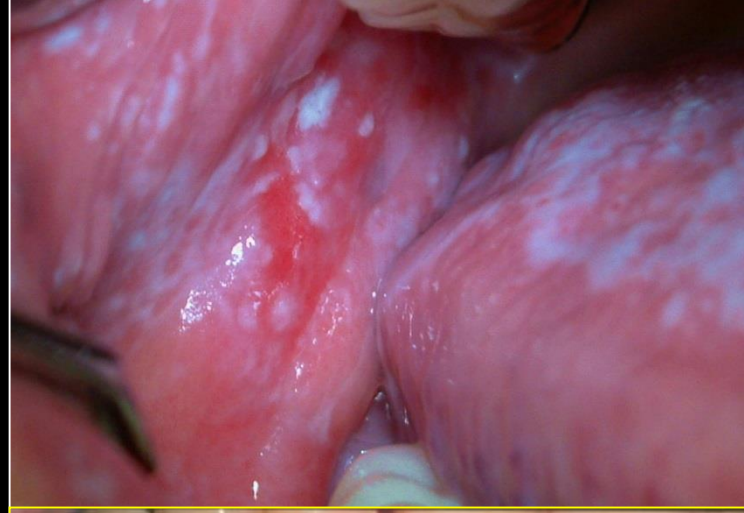
Oral Lichen Planus

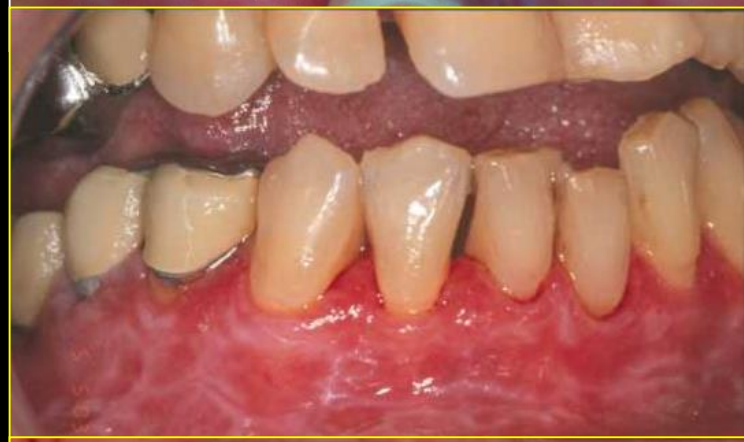
Erythematous (atrophic) OLP

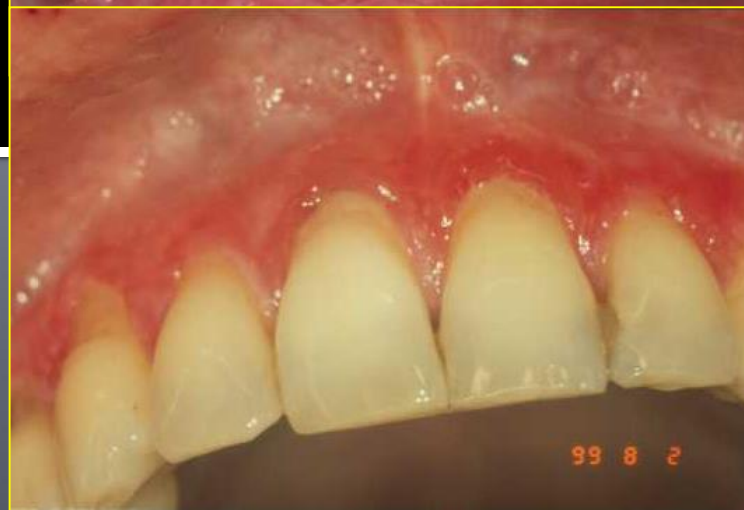
is characterized by a homogeneous red area. striae are frequently seen in the periphery.

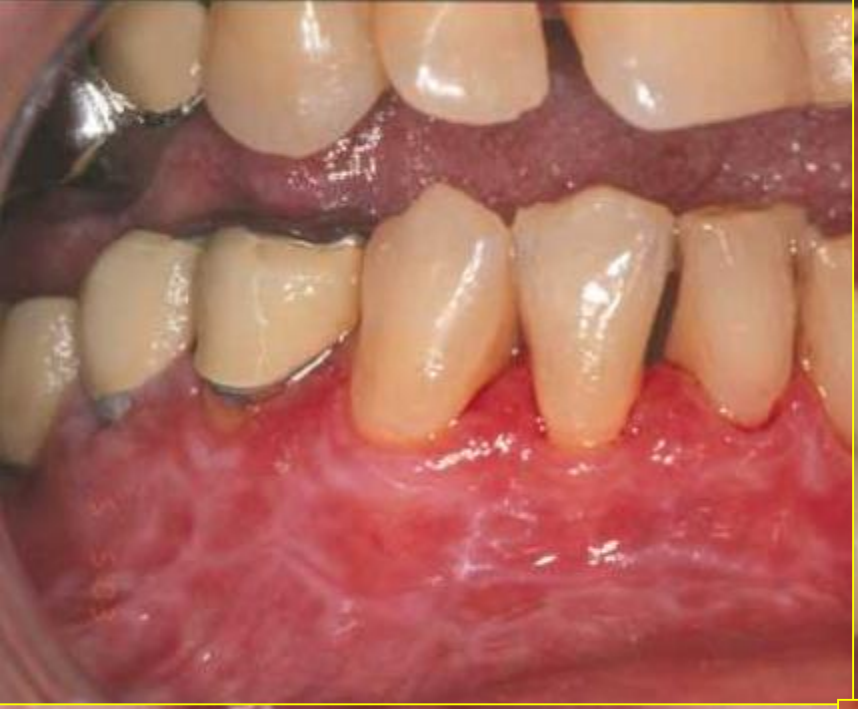
Some patients may display erythematous OLP exclusively affecting attached gingiva as desquamative gingivitis.

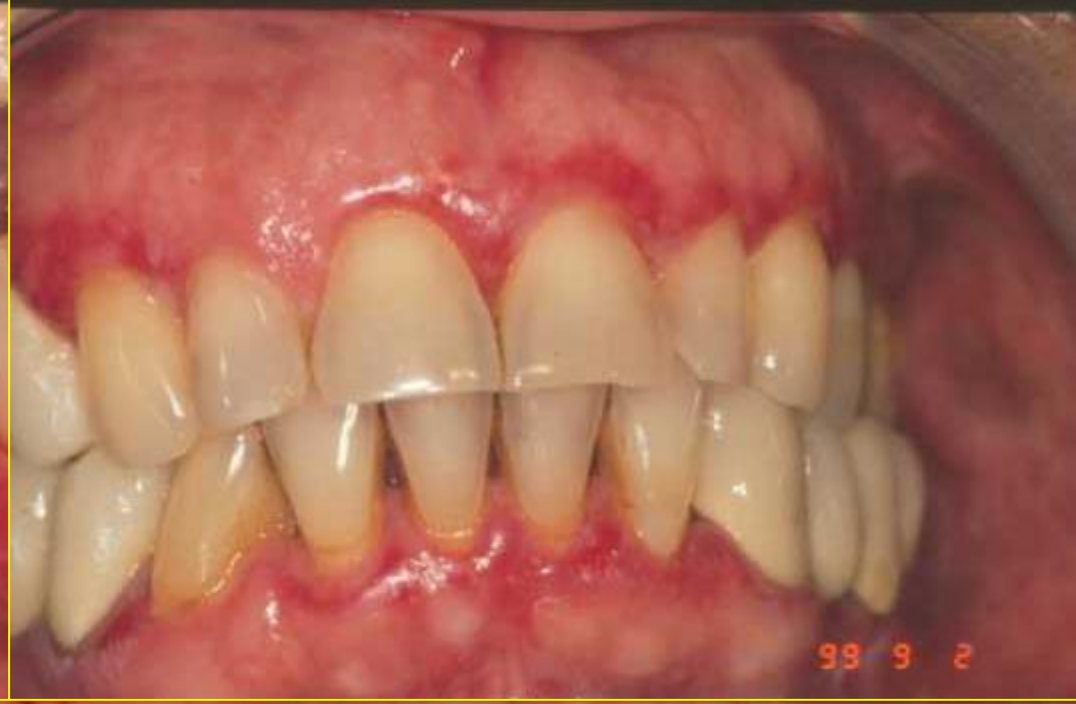
Erythematous OLP requires a histopathologic examination for a correct diagnosis







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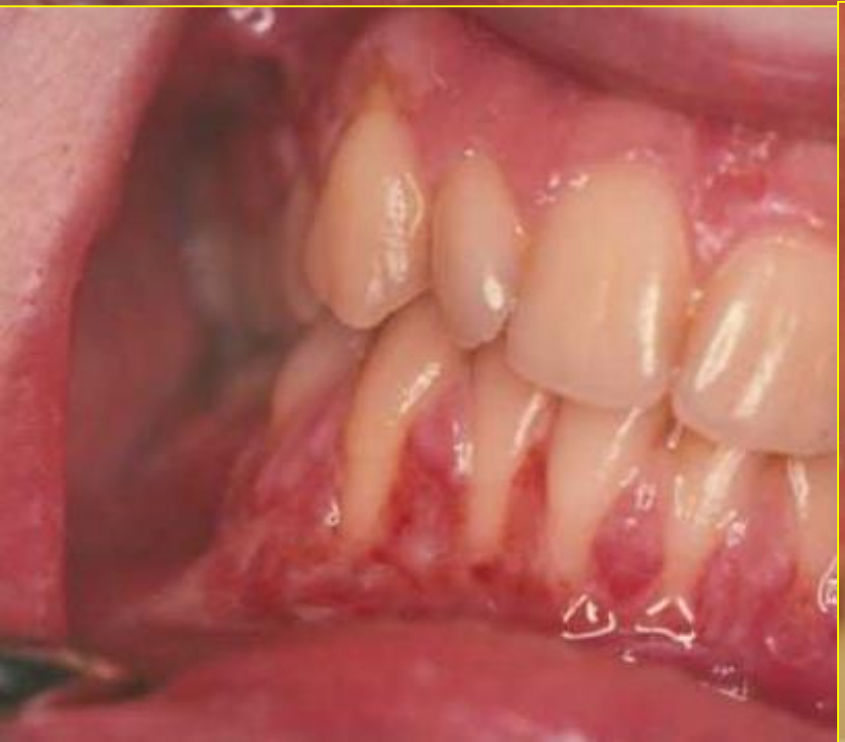

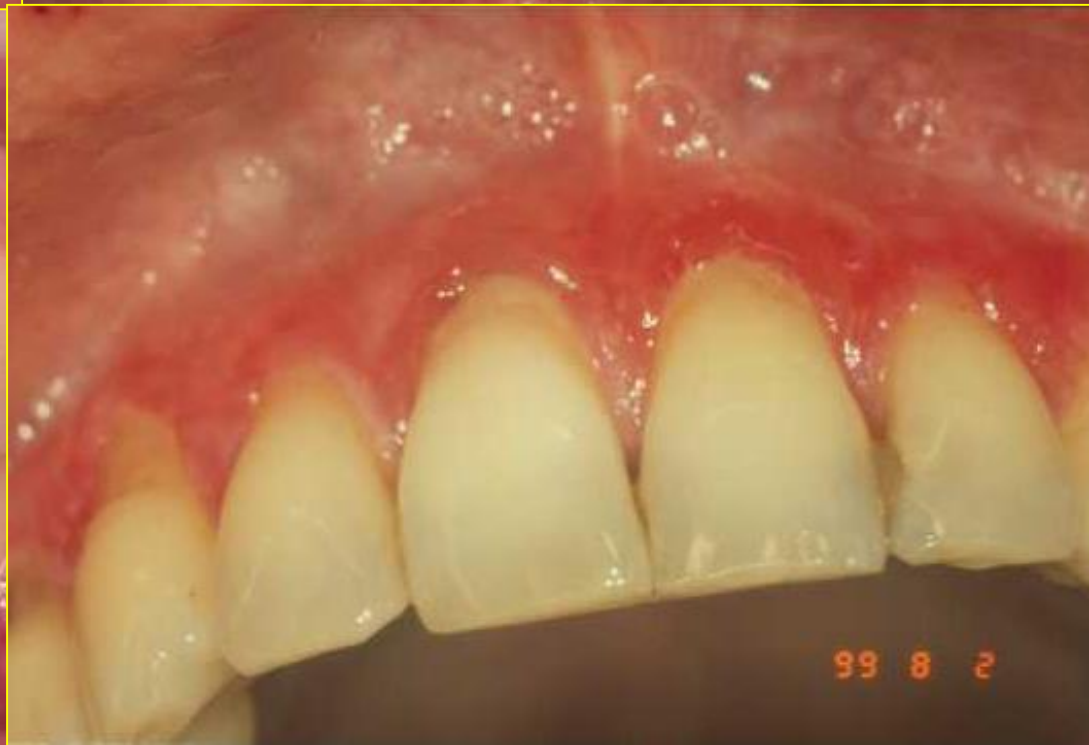
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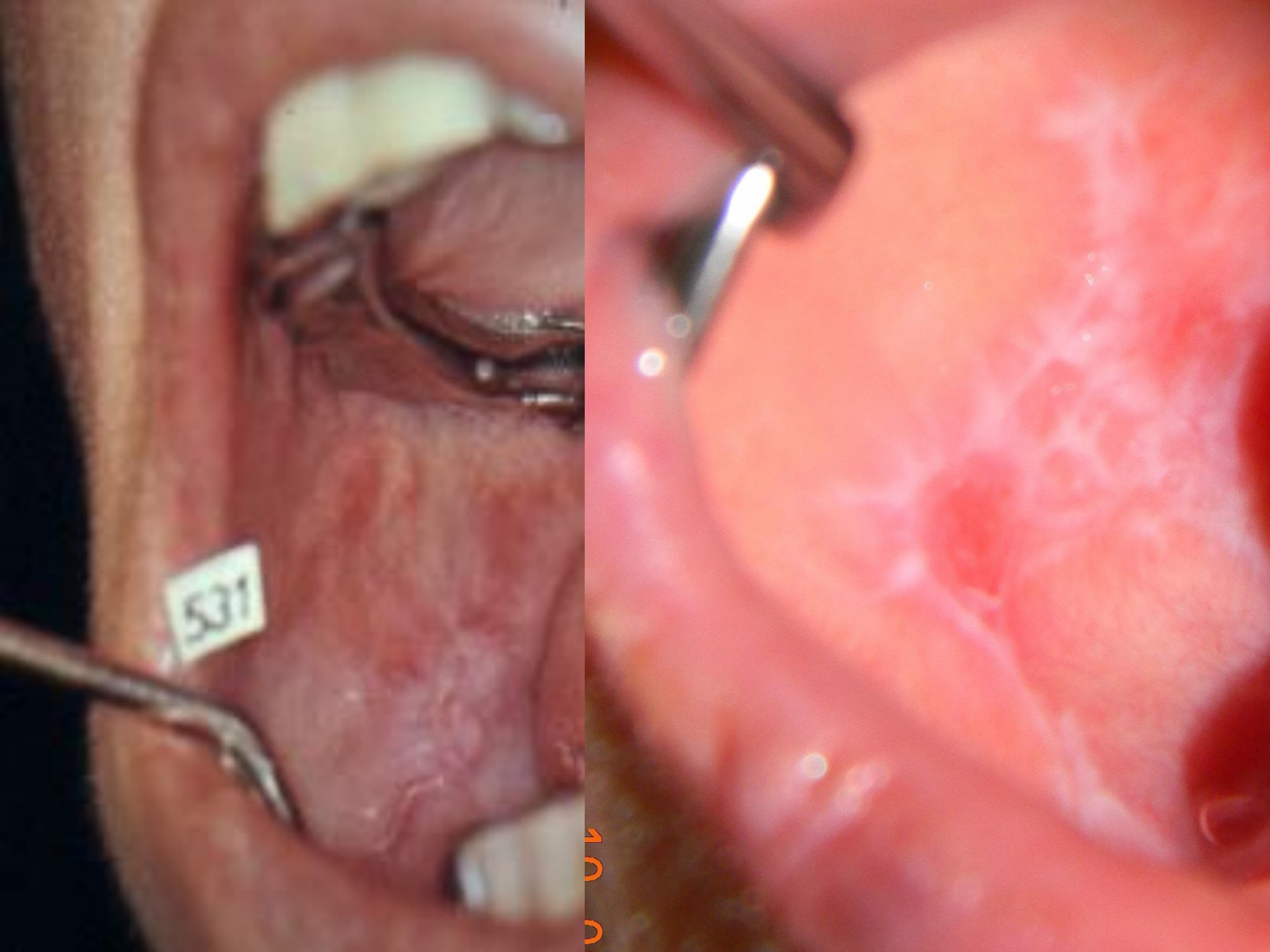
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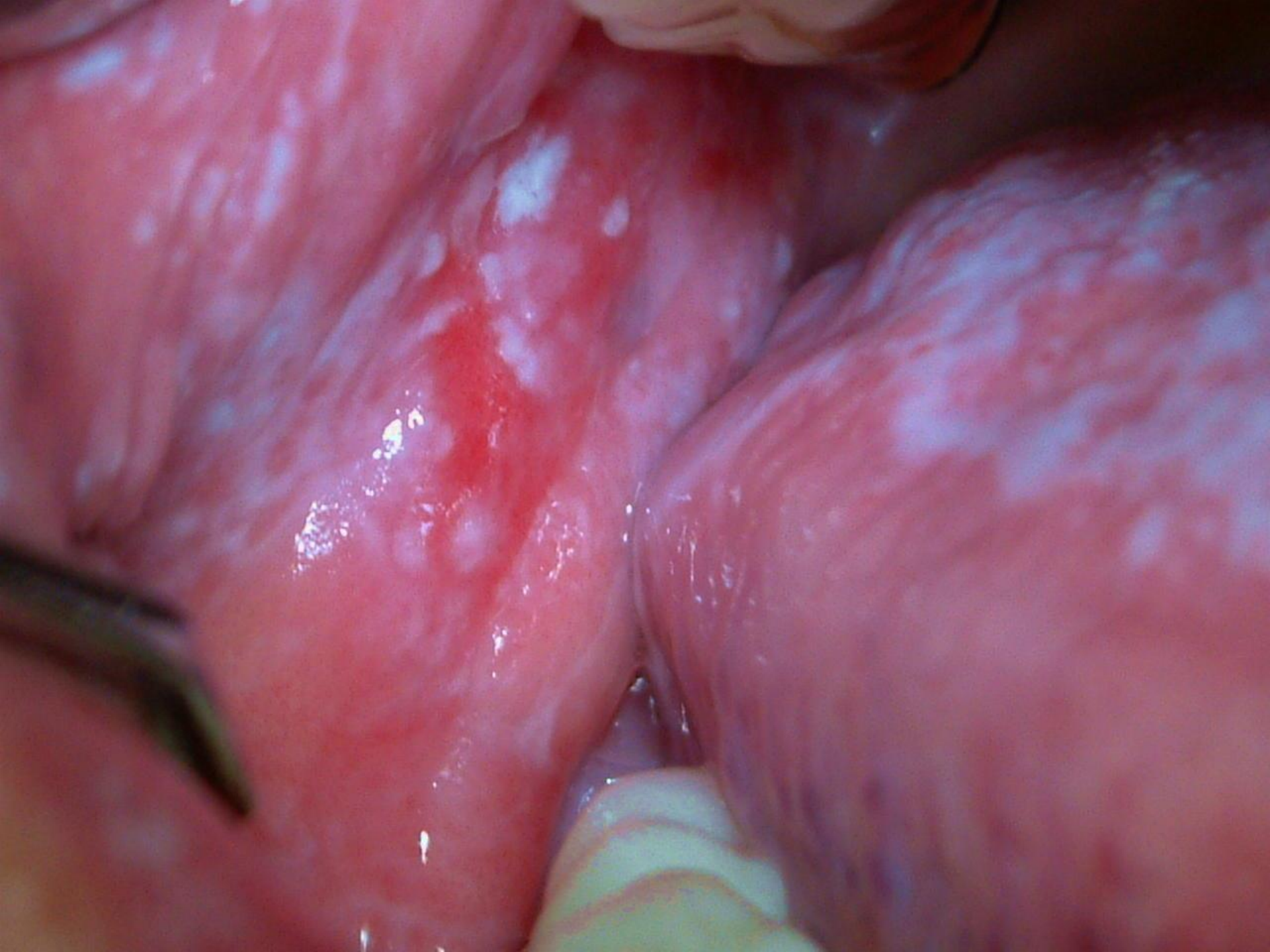
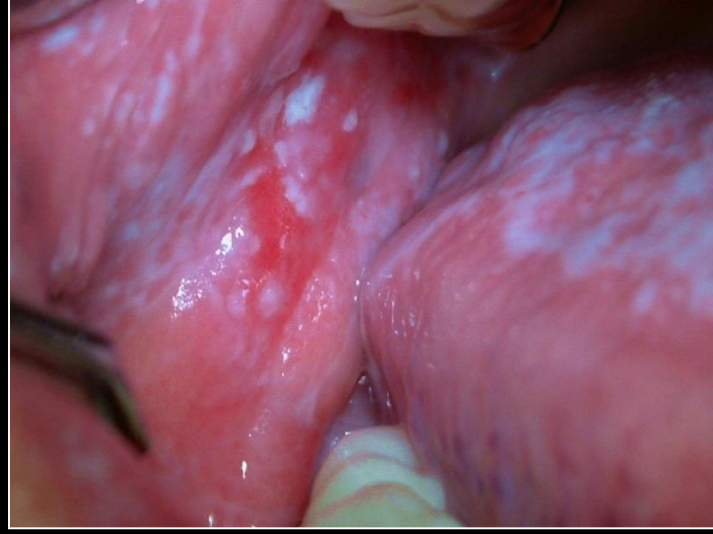


Oral Lichen Planus

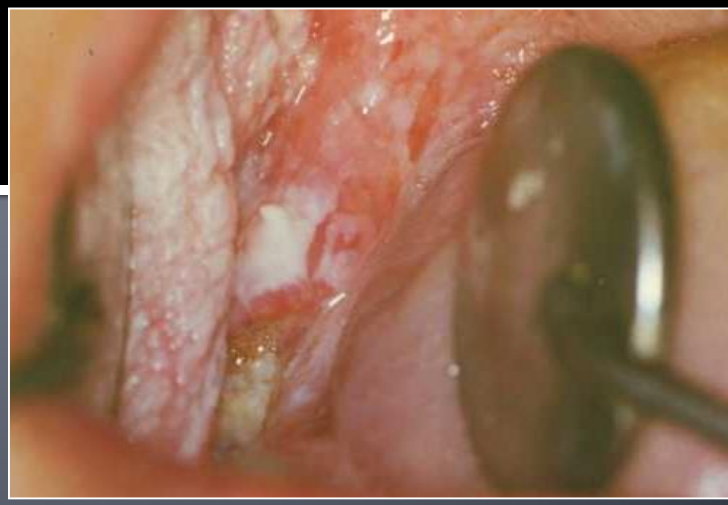


OLP is considered to be a premalignant condition

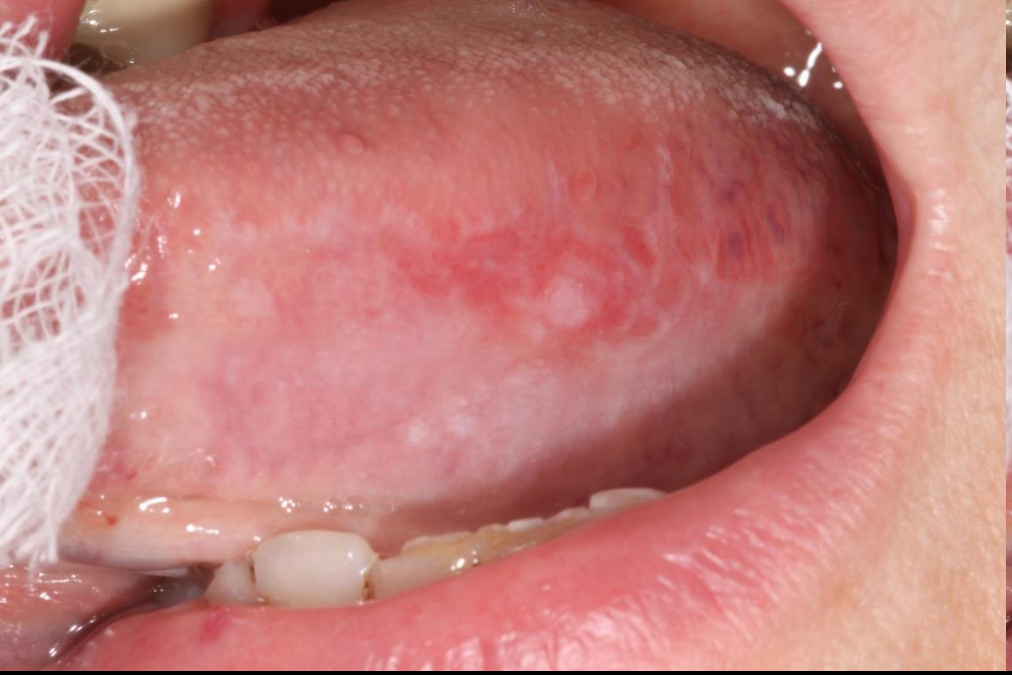
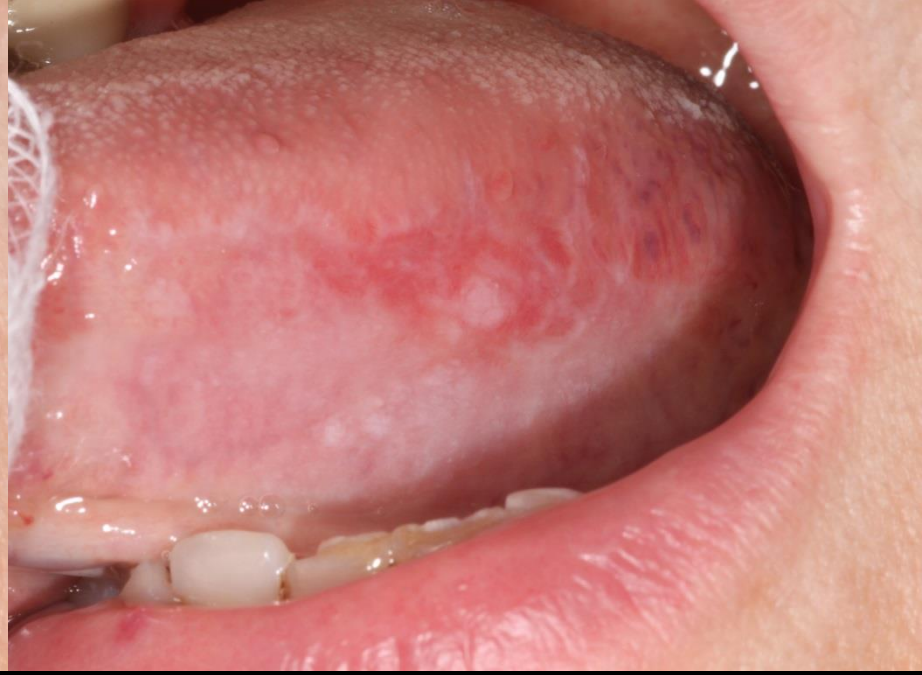
an increased risk of malignant transformation

predisposed to develop oral carcinomas,

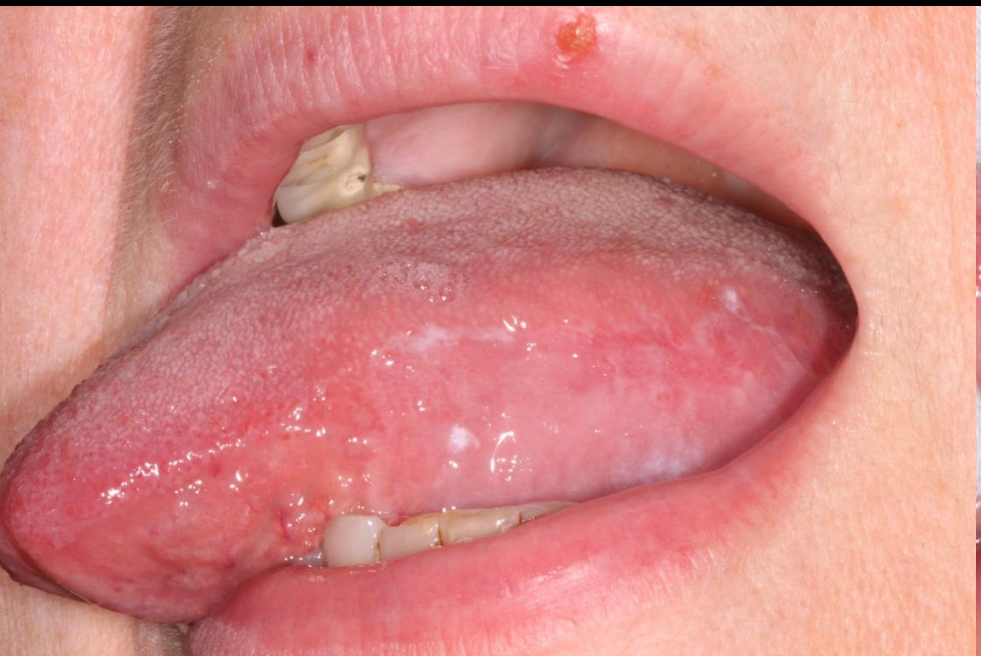
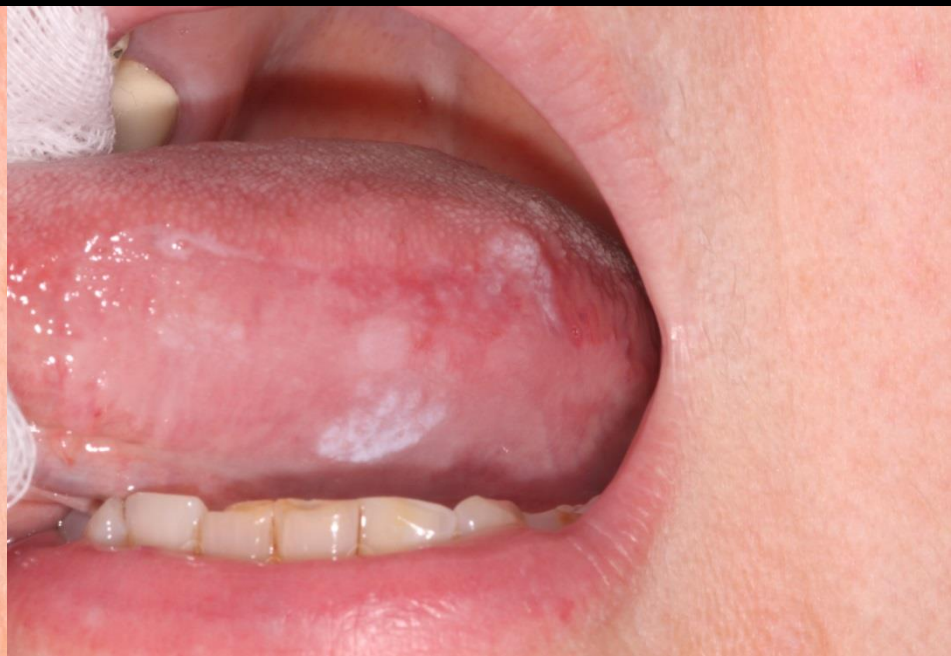
the risk is low and presumably with an incidence of 0.2% per year.

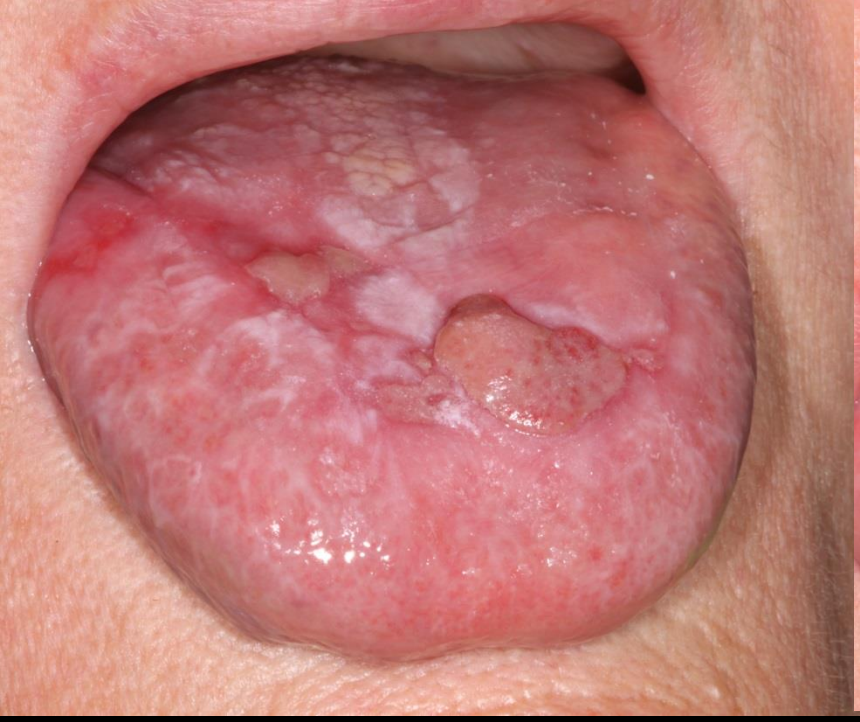


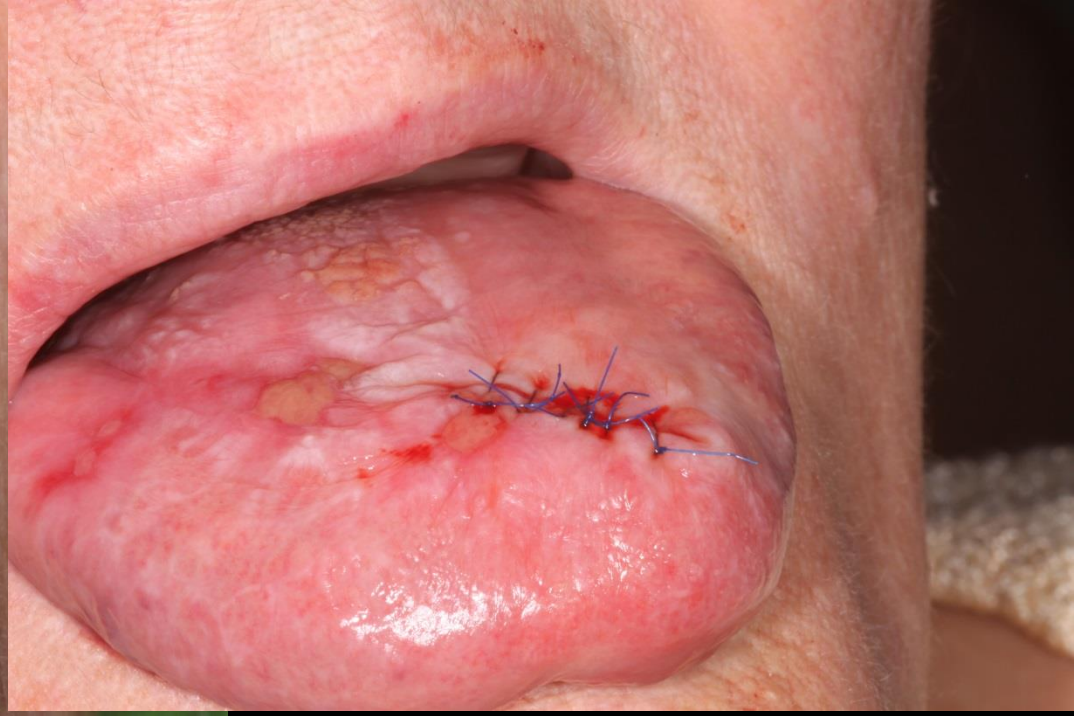
Oral leukoplakia and erythroplakia are premalignant lesions.

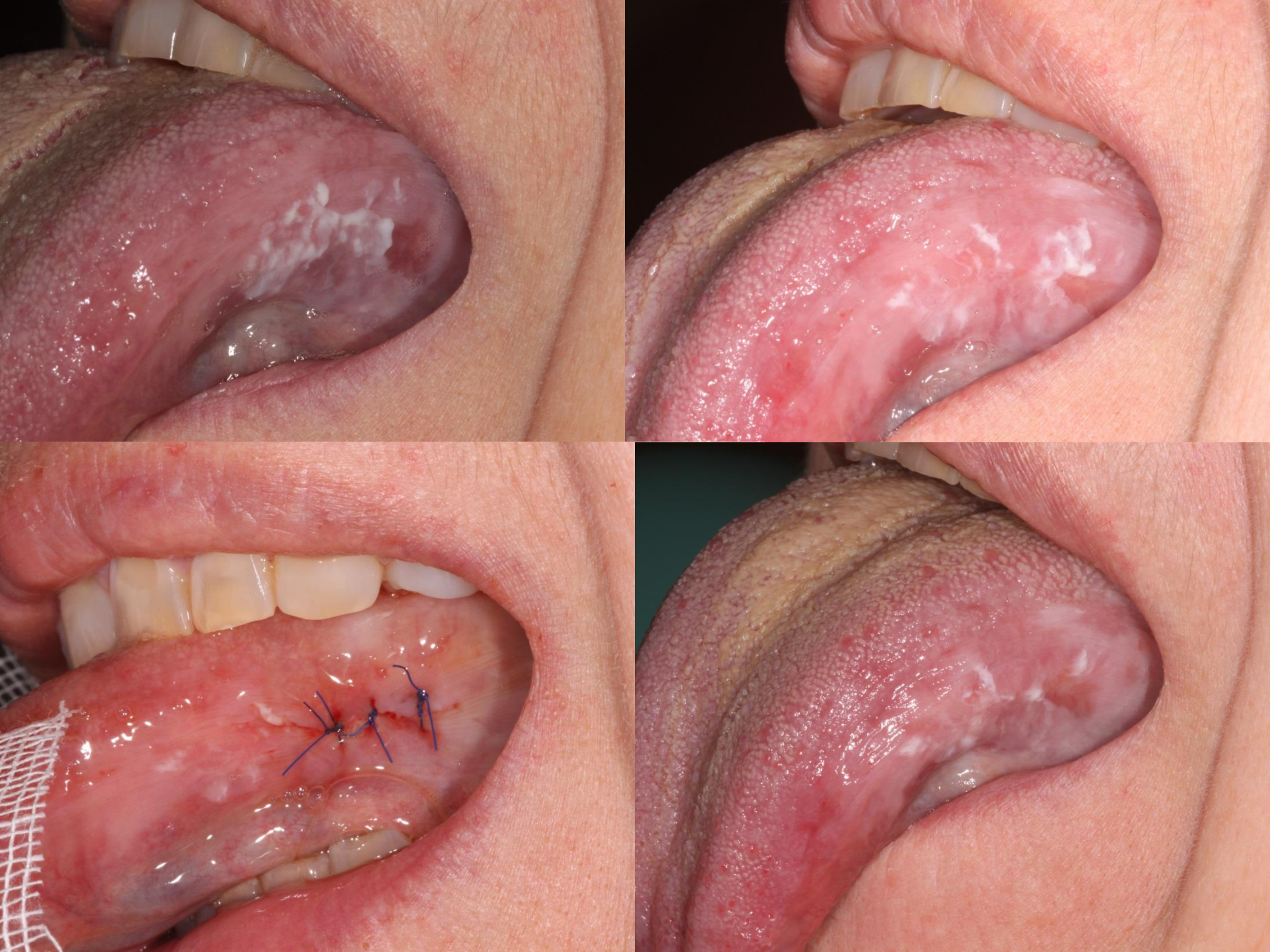
LICHEN followed-up



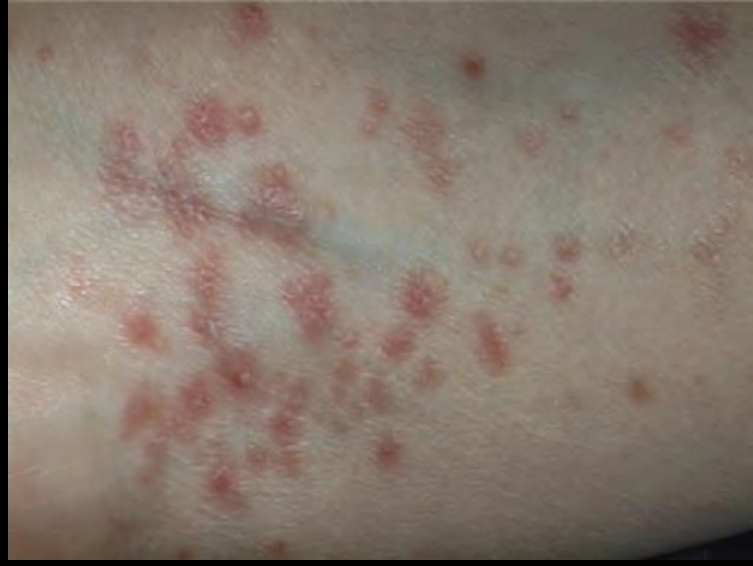


BIOPSIA!!!!



Oral Lichen Planus

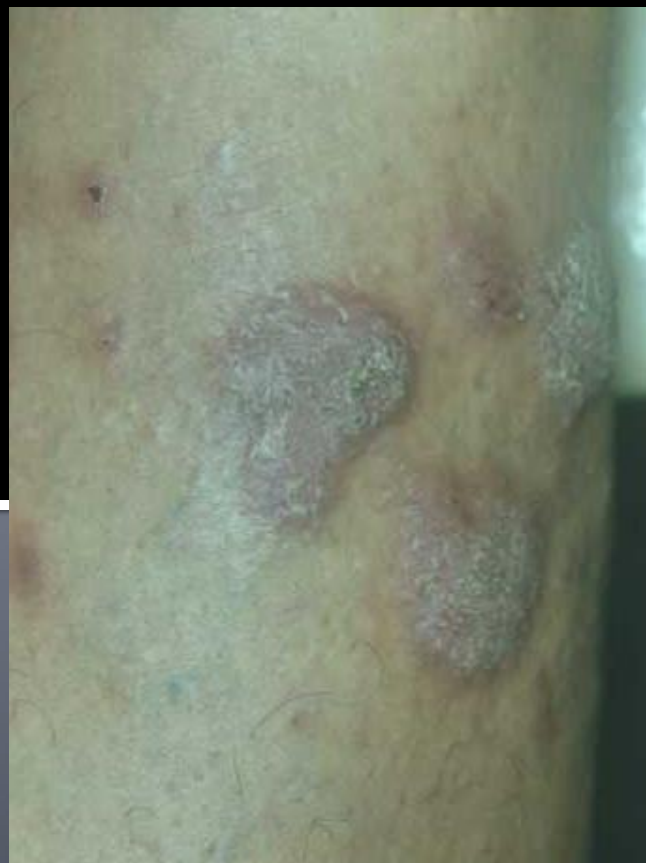
Lichen ruber planus



Clinical Manifestation

Cutaneous lesions may be encountered in approximately 15% of patients with OLP.

- pruritic erythematous
- flat papules

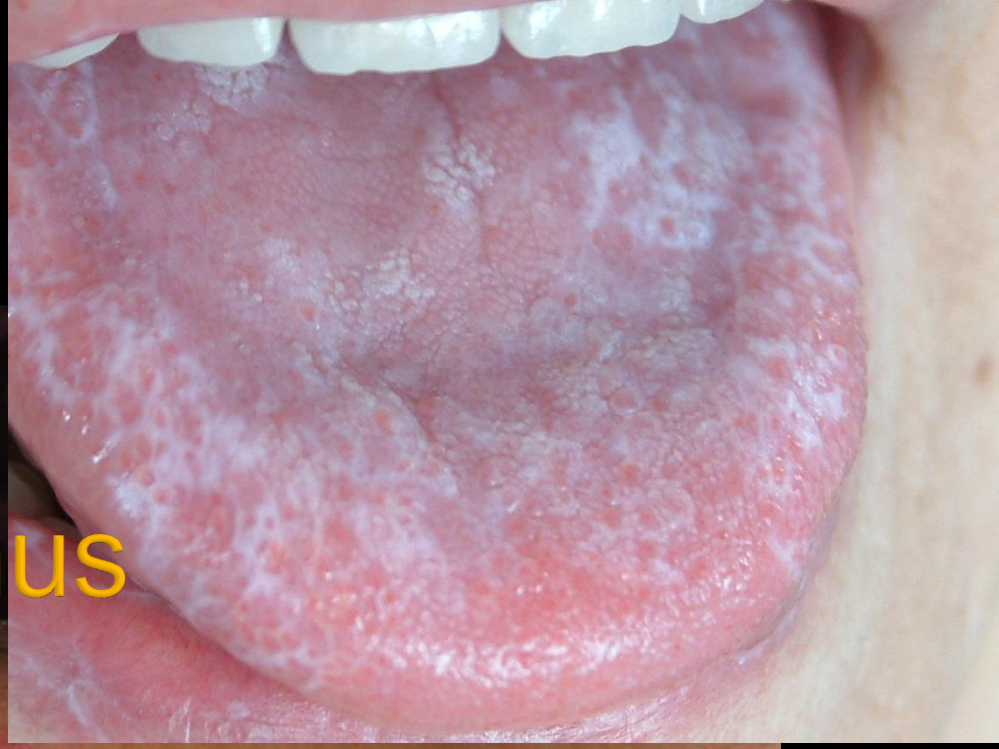


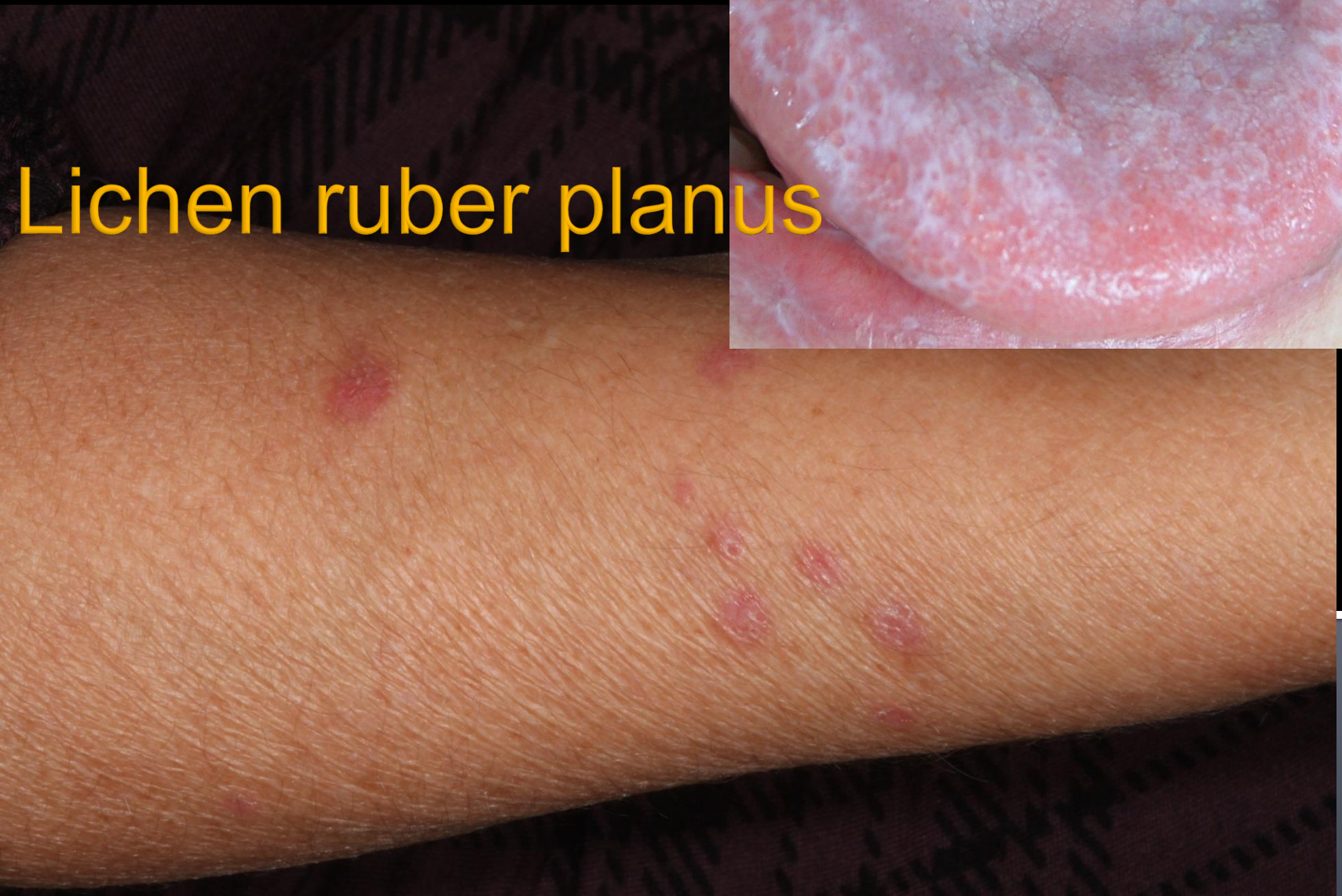
Predilection for the trunk and flexor surfaces of arms and legs

LICHEN RUBER PLANUS

PAPULO-SQUAMOSUS SKIN LESIONS

Lichen ruber planus





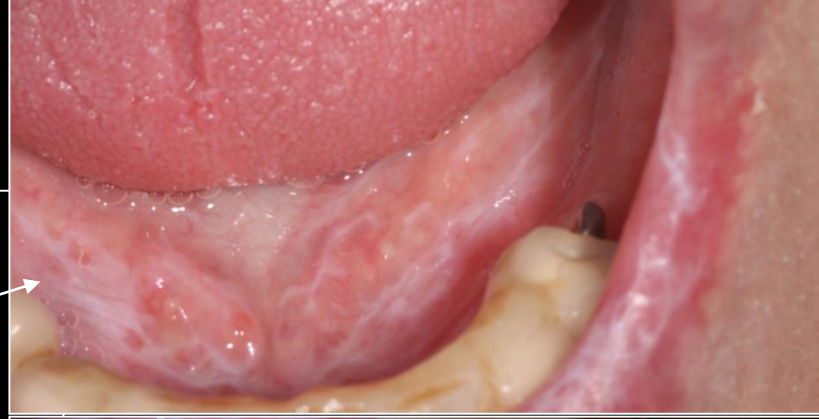
Oral Lichen Planus

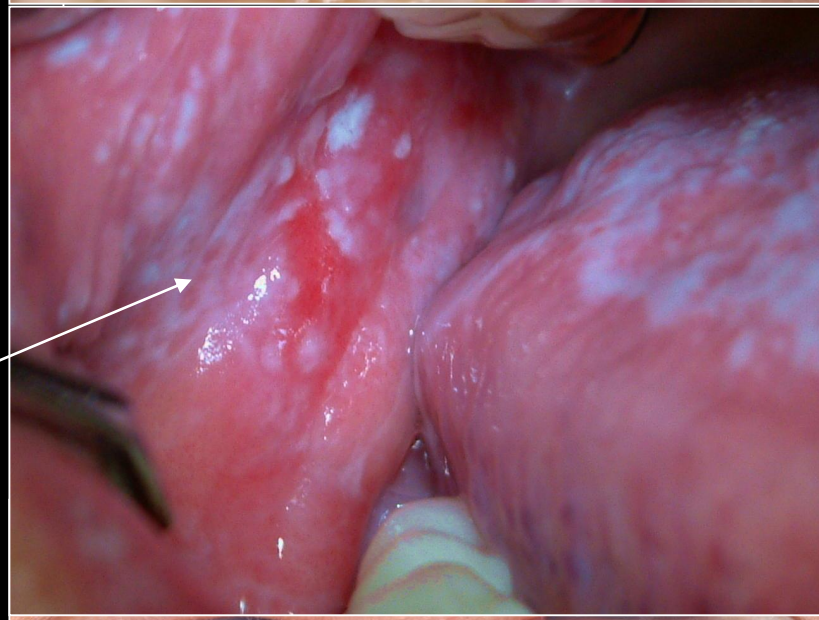
Diagnosis diagnosis

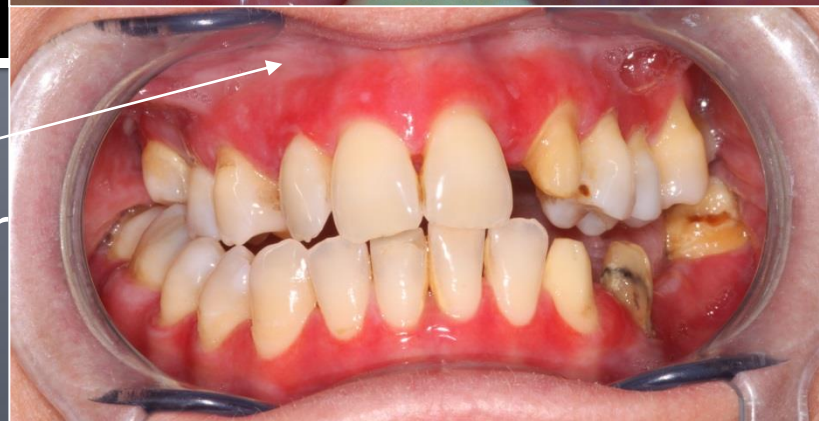
Papules or reticular components have to be present in order to establish a correct clinical diagnosis.

These pathognomonic components may exist
with plaque-like, erythematous, or ulcerative lesions.

In patients with gingival erythematous lesions, it may be difficult to find striae or papules.



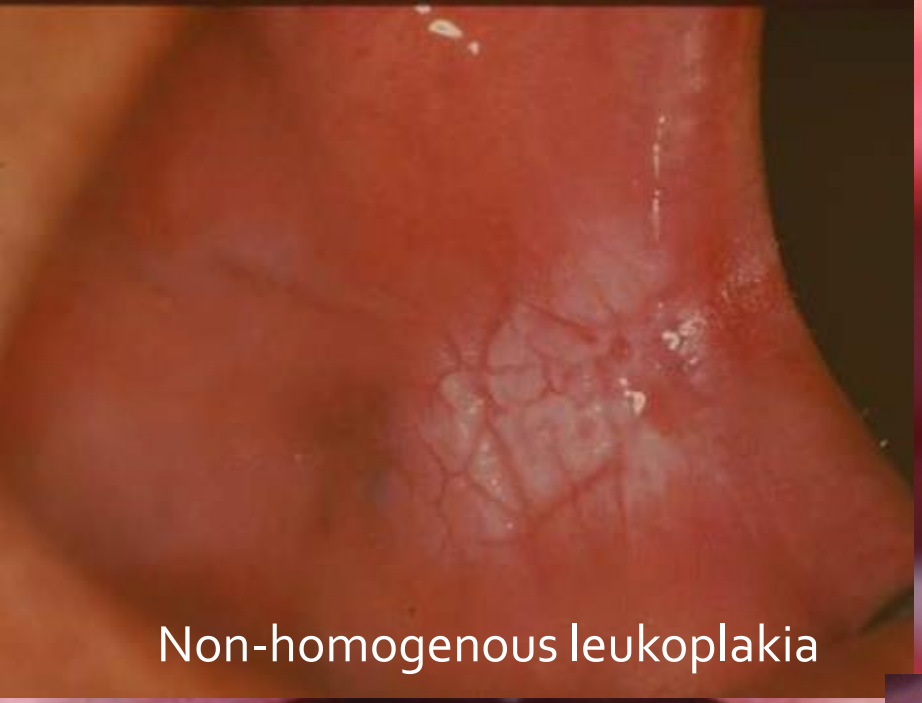




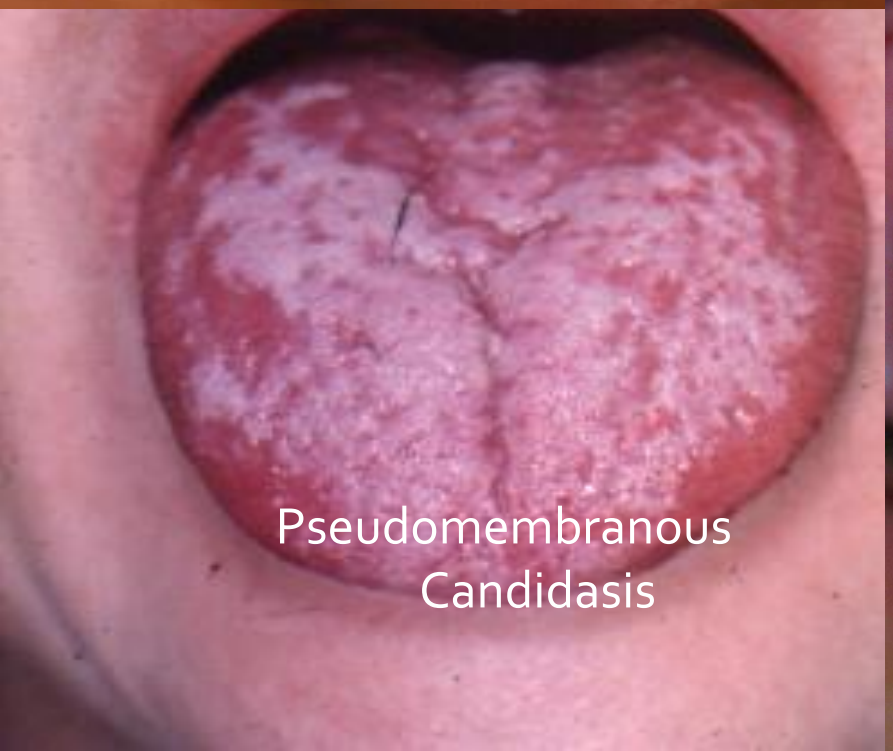
Oral Lichen Planus

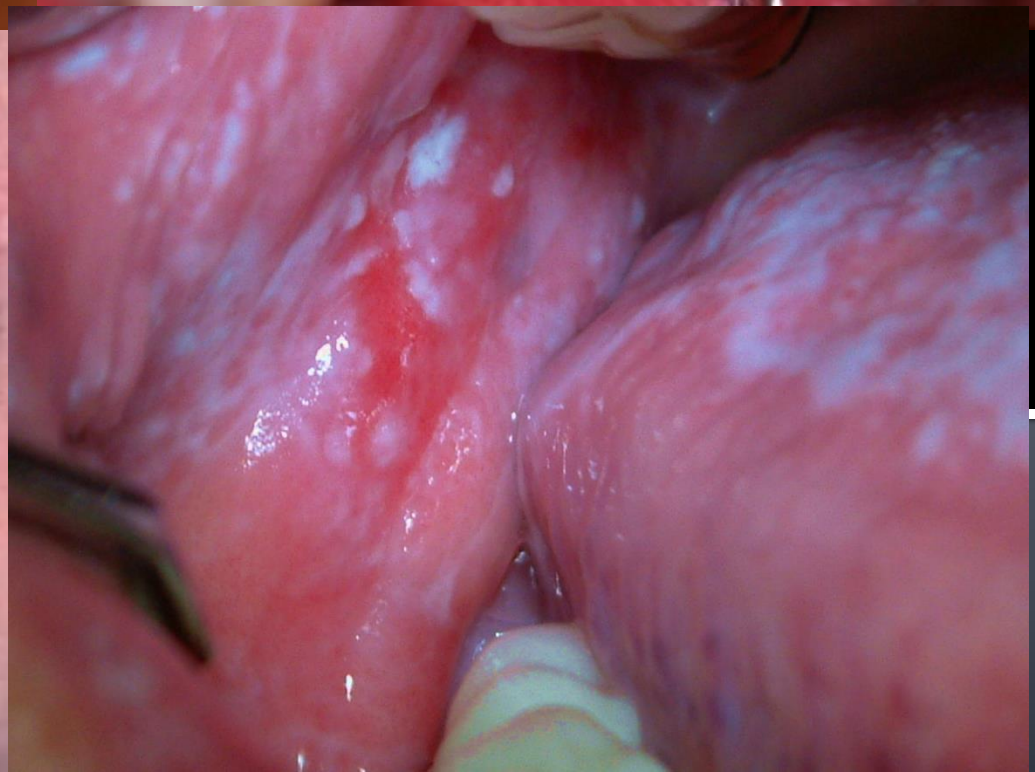
Differential diagnosis

- Leukoplakia
- Candidasis
- Pemphigus, Pemphigoid
- Morsicatio
- Glossitis mediana rhombica
- Fox-Fordyce foltok
- Leukoedema mucosae oris


Non-homogenous leukoplakia


Morsication
(cheek biting)


Pseudomembranous
Candidasis



Oral Lichen Planus

Management

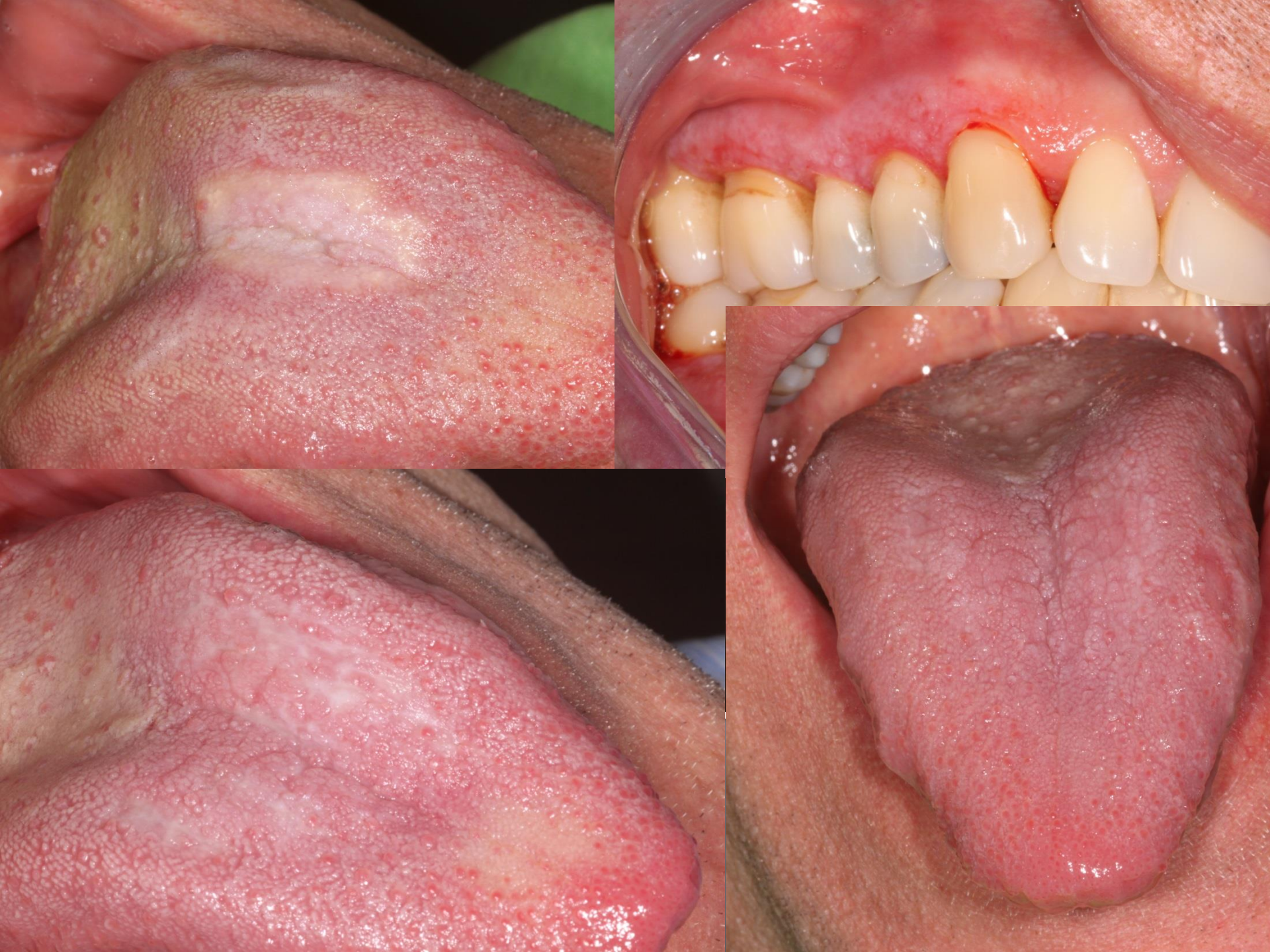
the etiology behind OLP is unknown!!!!!,

current treatment strategies are aiming at reducing or eliminating symptoms.

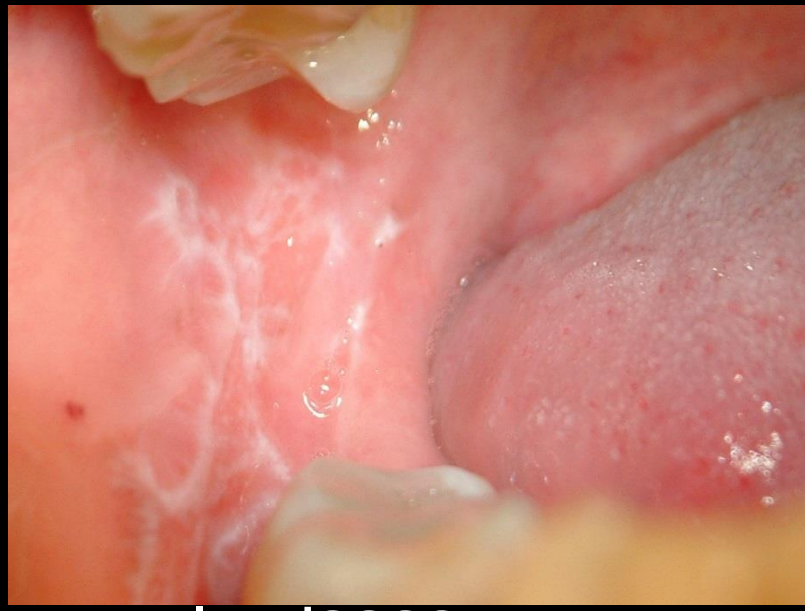
- topical drugs
- steroids,
- calcineurin inhibitors (cyclosporine and tacrolimus),
- retinoids,
- ultraviolet phototherapy.

Therapy I.

- Eliminate the Köbnering factors (change bad fillings, prosthetics, metals causing galvanism
- Antibacterial or antifungal treatment).
- Control HSV infection
- Change the immune complex producing drugs (if it is possible).
- Psychotherapy - cancerophoby

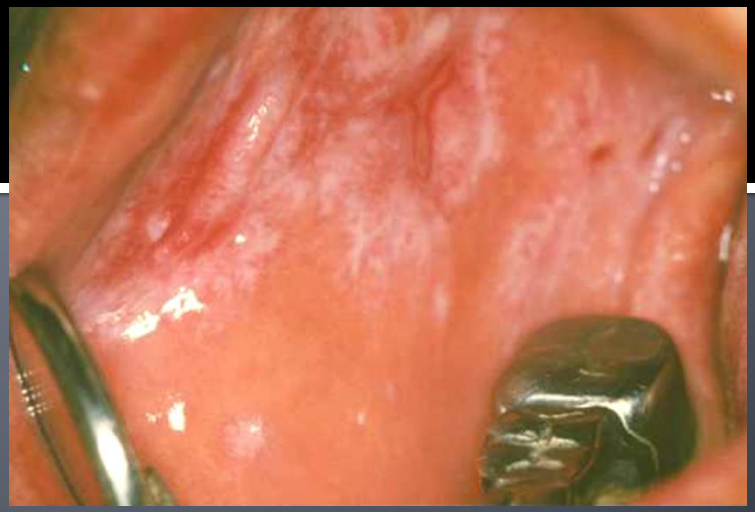


Therapy II.



- Topical Vitamin-A oil
- Systhemic Vitamin -A
- Chlorhexidin rinsing can be applied???? No

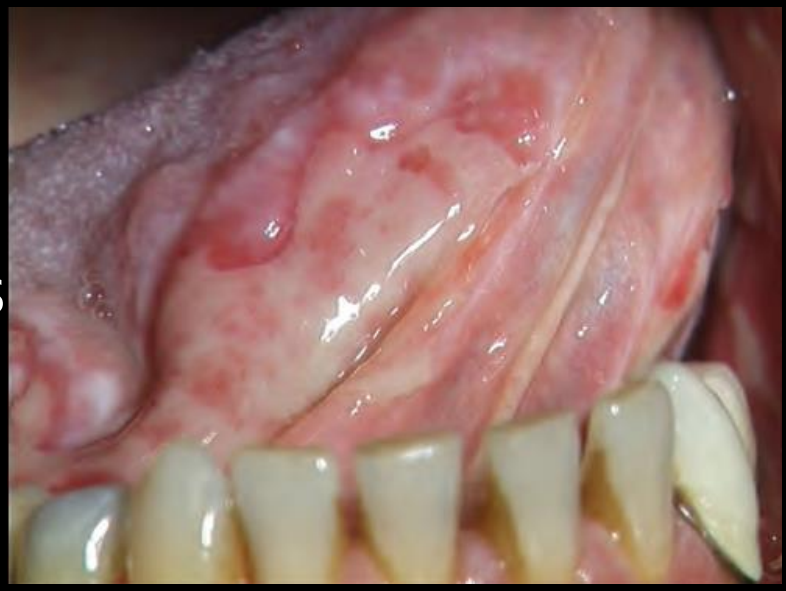
- Local corticosteroid treatment (Oxycort spraey, Dapsone,)



Therapy III.

- Immunomodulant treatment (suppressed immune system is activated, hyperimmune system is suppressed)
- β-levamisol
- Alpha-interferon
- GM-CSF

Oral Lichen Planus

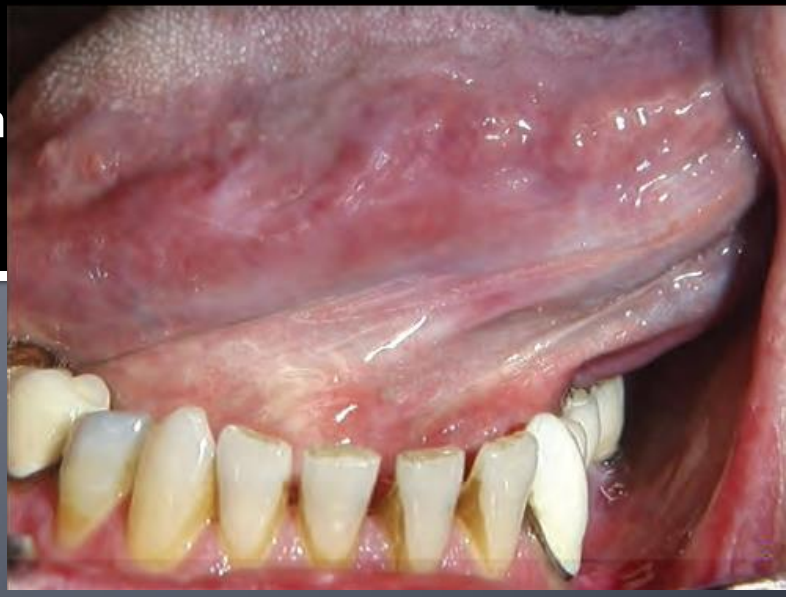


topical steroids are widely used as the primary treatment

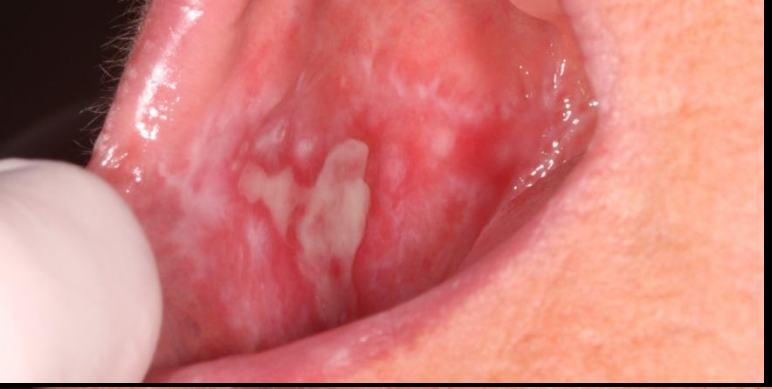
Systemic steroids

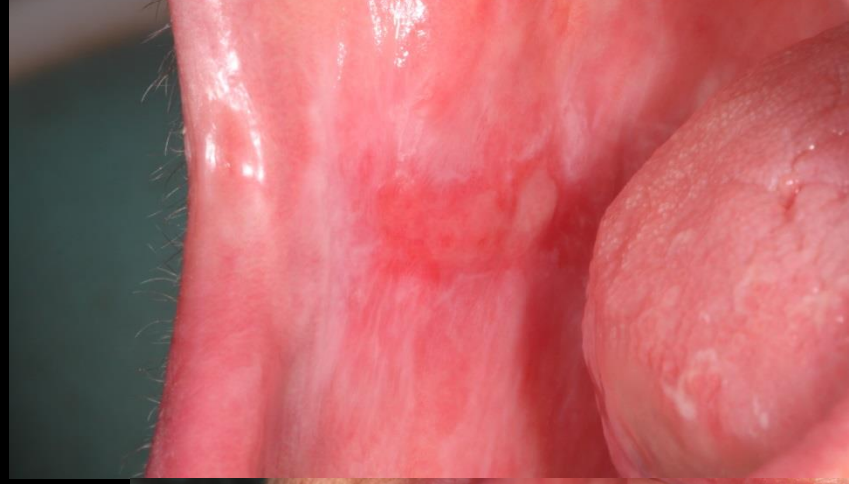
Ie. Medrol

One milligram per kilogram daily for 7 days followed by a reduction of 10 mg each subsequen day.



intermediate steroids: triamcinolone





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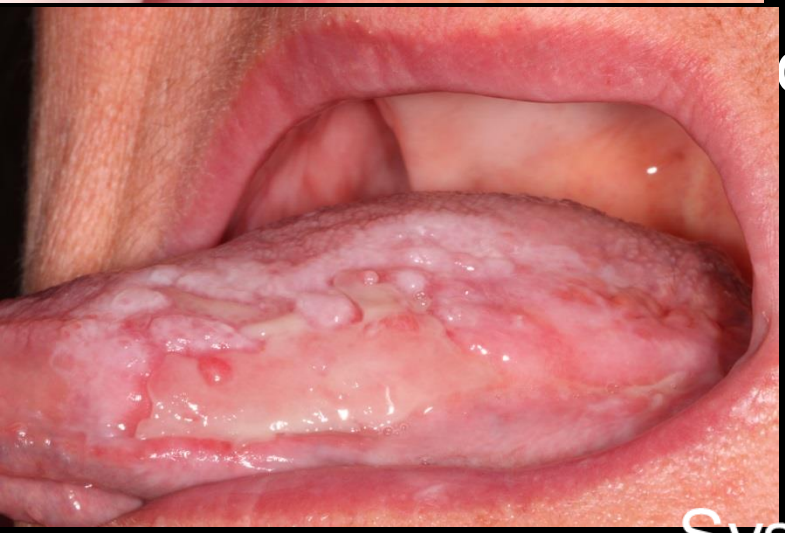
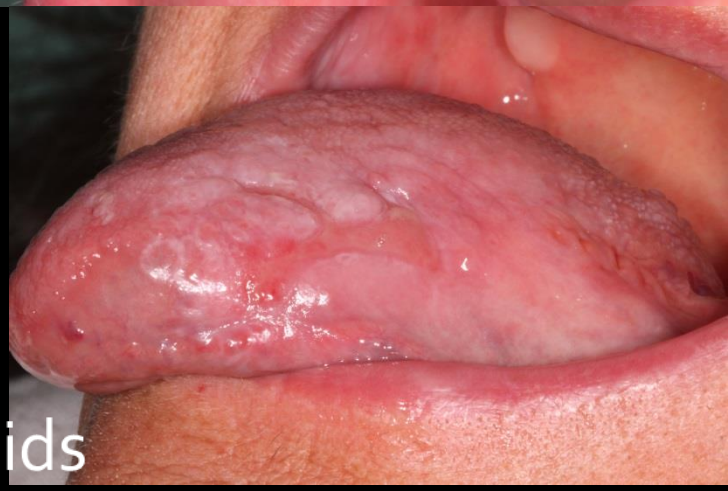
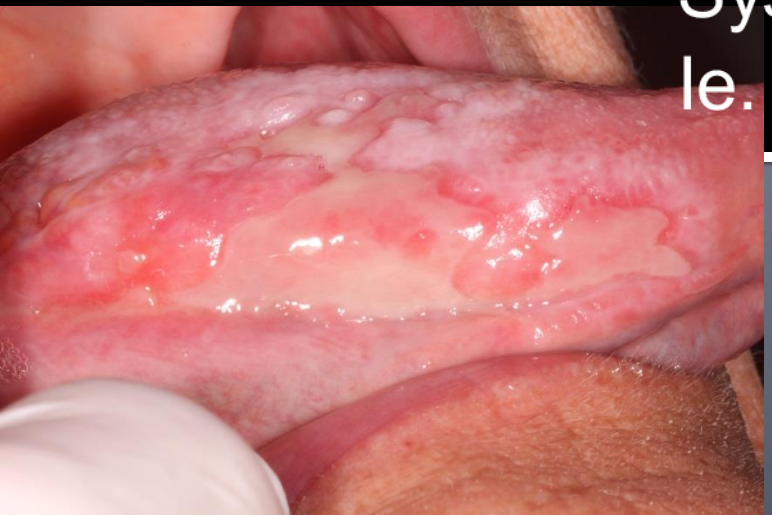
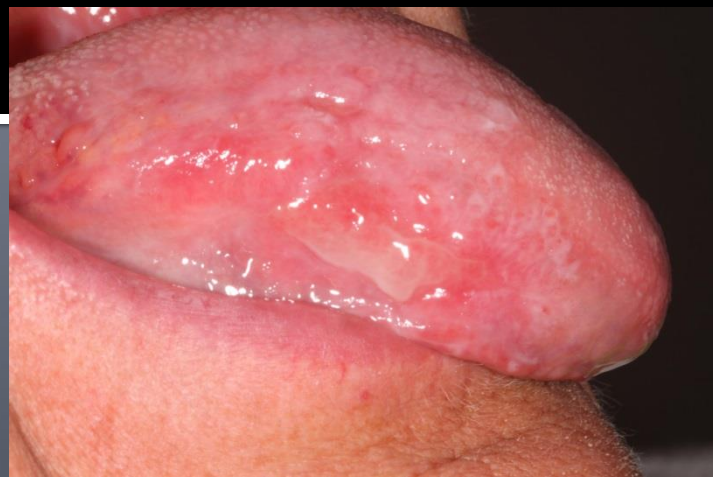




Systemic steroids

le. Medrol



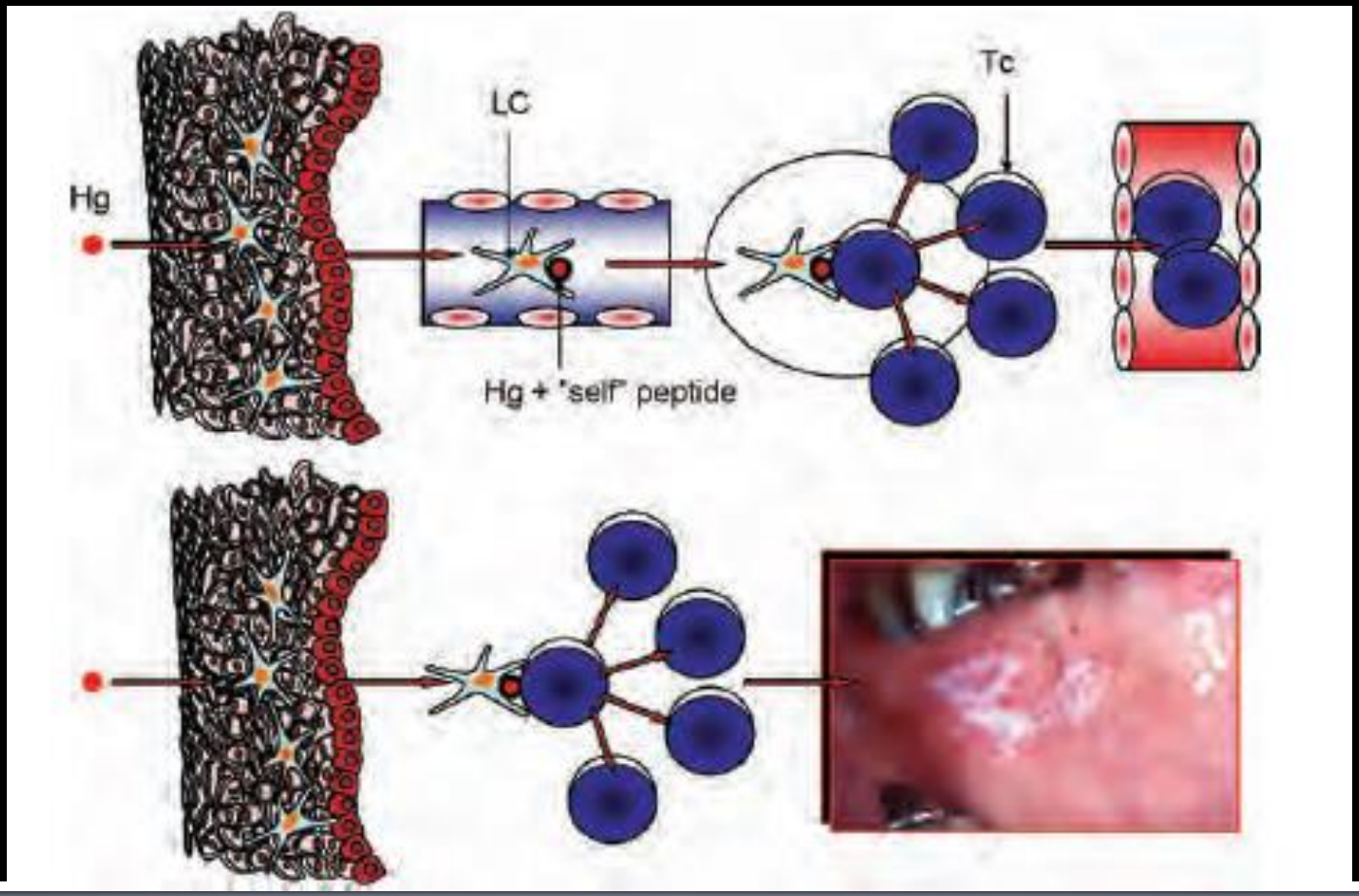


Oral Lichen Planus

Lichenoid reaction

- Lichenoid Contact Reactions
- Drug-Induced Lichenoid Reactions
- Lichenoid Reactions of Graft-versus-Host Disease
- Reactions to Dentifrice and Chlorhexidine

Lichenoid Contact Reactions

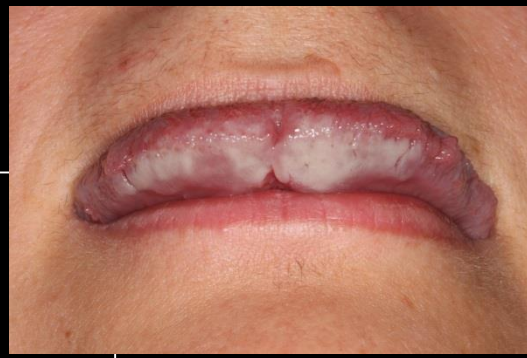


Etiology and Pathogenesis

Hg cannot be recognized by the T-cell receptor (TCR), Hg ions will bind to self-proteins of the oral epithelium, which will induce transformation changes of the protein.

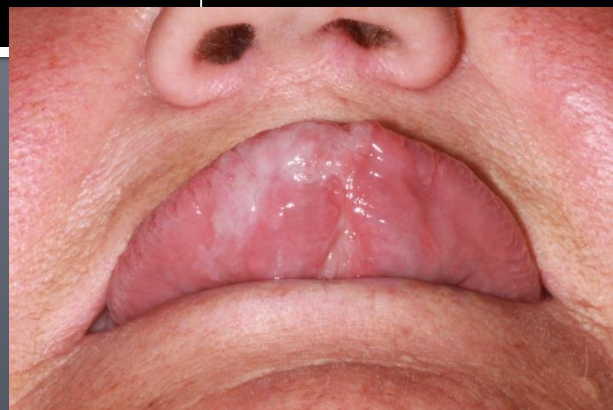
This assembly between Hg and protein will be perceived as nonself

Oral Lichen Planus

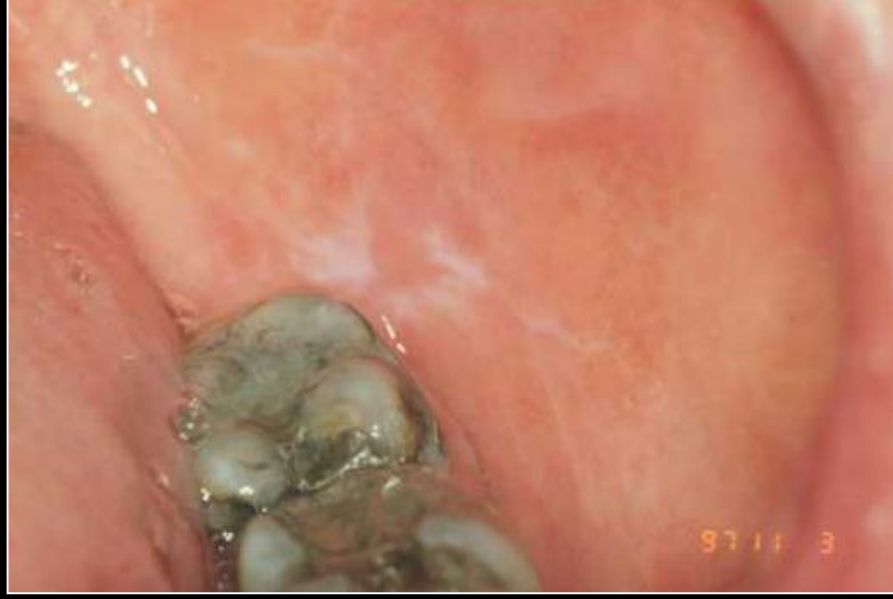


- Oral lichenoid contact reaction (LCRs) to dental materials,.

- Oral lichenoid drug eruptions have the same clinical and histopathologic characteristics as OLP.



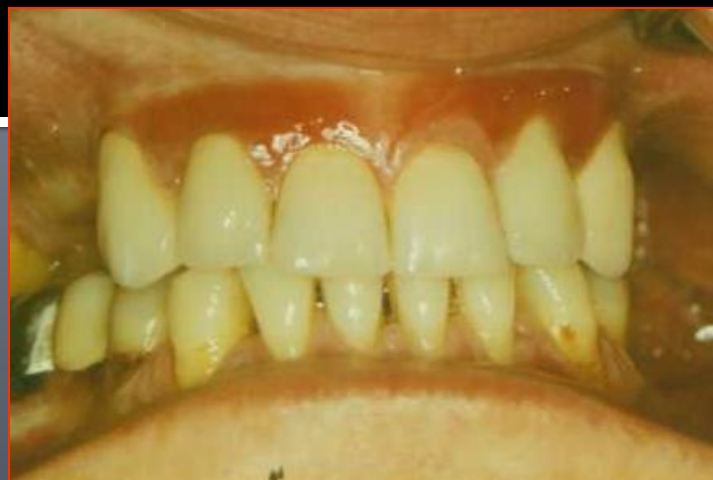
Lichenoid Contact Reactions

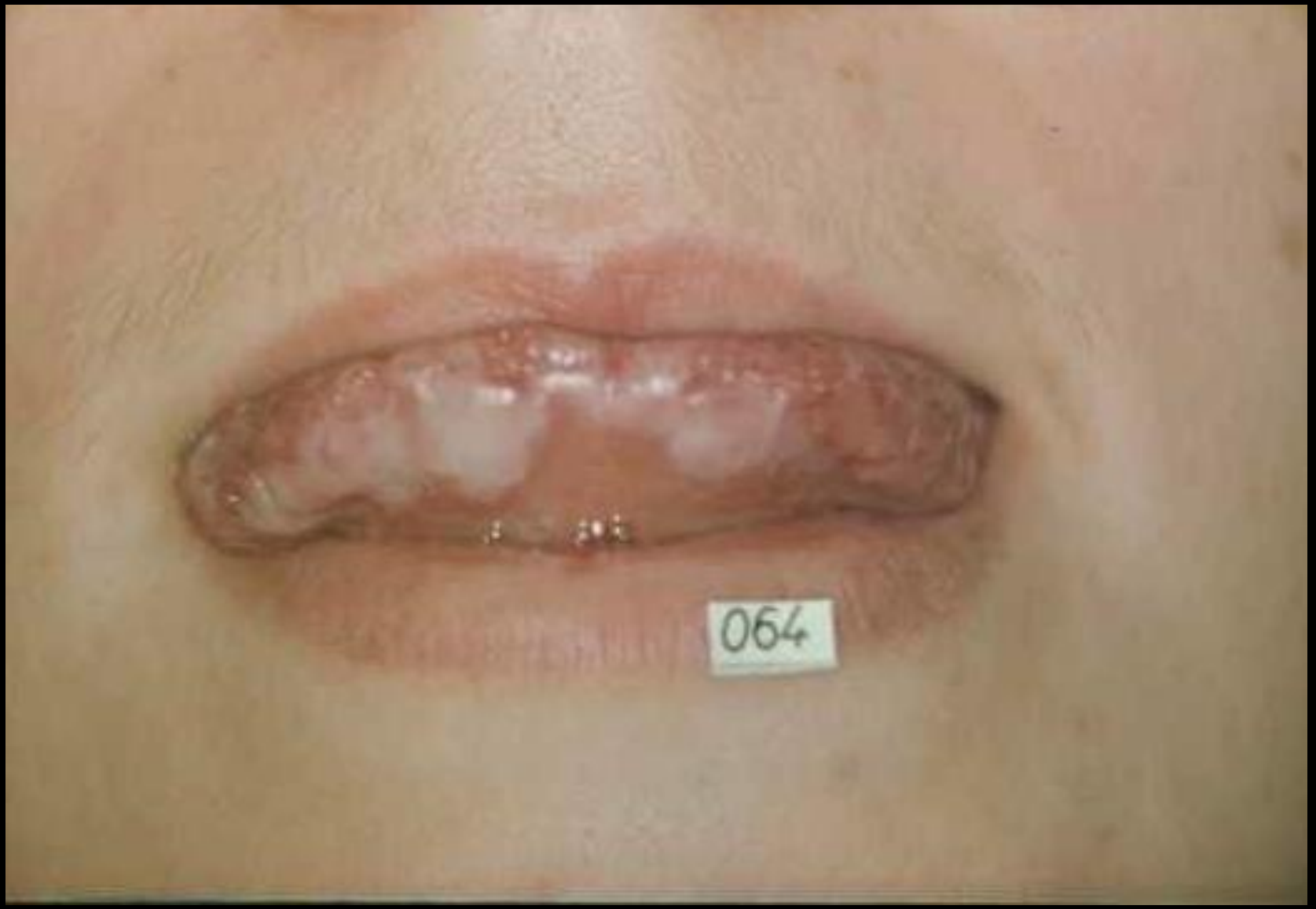


LCRs are a delayed hypersensitivity reaction against dental materials.

The majority of patients are patch test positive to mercury (Hg), or chromium

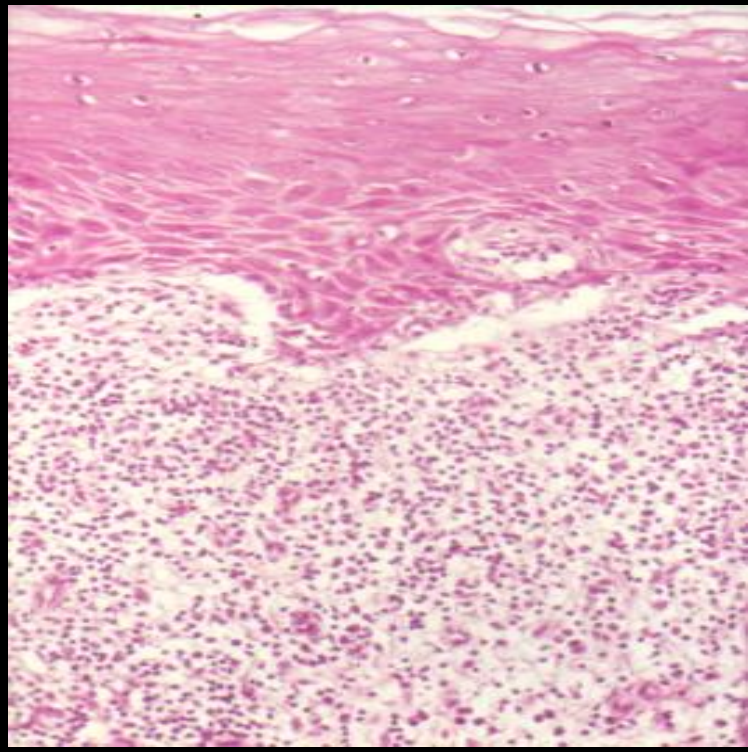
LCR is an allergic reaction..





Contact allergy to Cr/cobalt alloy

Oral Lichen Planus



Pathology- Histopathology

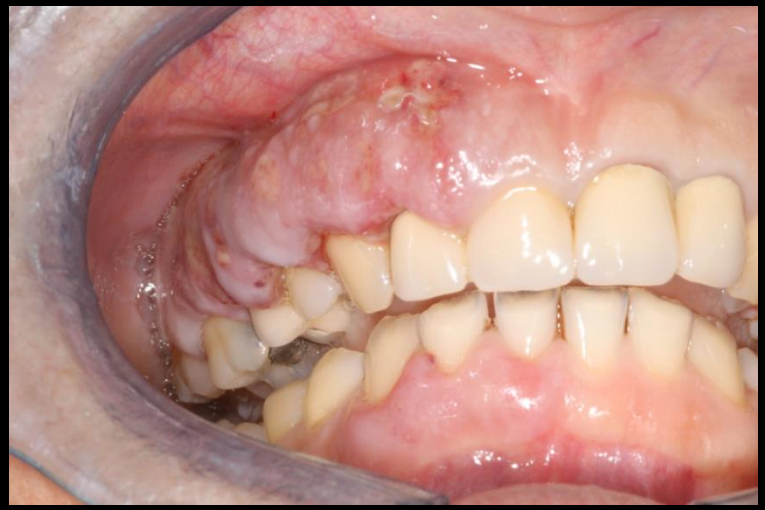
a histopathologic examination

Diffucult to differentiate between OLP, LCR,

Oral Lichen Planus

Oral graft versus host reaction I

GVHD has the same clinical appearance as OLP,



the lesion is more generalized.
The lichenoid reactions seen simultaneously with other symptoms –

- Xerostomia
- liver dysfunction
- localized skin involvement

Lichenoid Reactions of GVHD

Etiology and Pathogenesis

The major cause of GVHD is allogeneic hematopoietic cell transplantation, that attempts to reject the tissue

recognition of alloantigens by donor T lymphocytes.
an interaction between the recipient's APCs and the donor's T lymphocytes,

This interaction resembles the interaction between autoreactive T lymphocytes and APCs, hypothesized to play a role in the development of OLP.

In a third step, the inflammatory cascade resulting in clinical features compatible with a lichenoid reaction.

Oral Lichen Planus

Reactions to Dentifrice and Chlorhexidine
Delayed hypersensitivity reactions to toothpastes and mouthwashes

The clinical manifestations include fiery red edematous gingiva,

Similar lesions may involve other sites, such as the labial, buccal, and tongue mucosae.

the lesions heals after withdrawal of the allergen-containing agent.

Oral Lichen Pla

Desquamative gingigivitis

Erythematous OLP of the gingiva exhibits a similar clinical presentation as mucous membrane pemphigoid or pemphigus vulgaris.

In pemphigus the epithelium is easily detached from the connective tissue by a probe or a gentle searing force (Nikolsky's phenomenon).

No reticular or papular elements in the periphery of the ulcerations.

Oral Lichen Planus

Desquamative gingigivitis

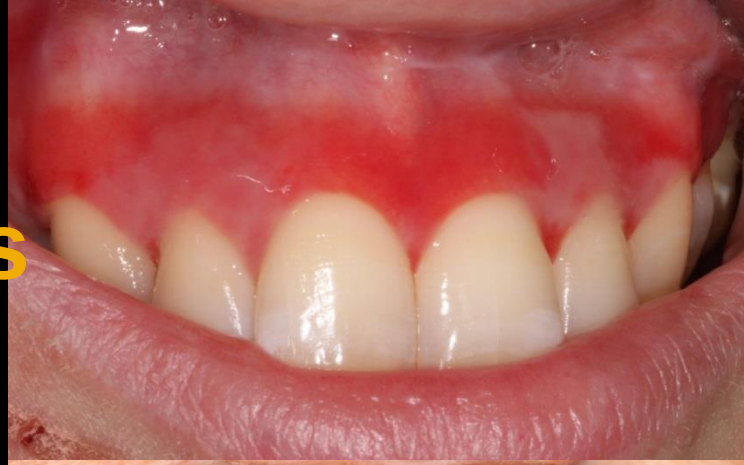
Erythematous OLP of the gingiva constitutes a therapeutic challenge.

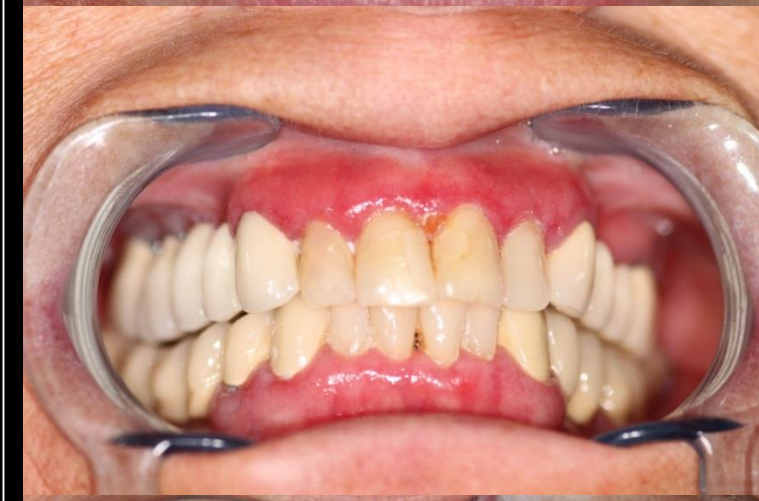
critical to remove sub- and supragingival plaque and calculus

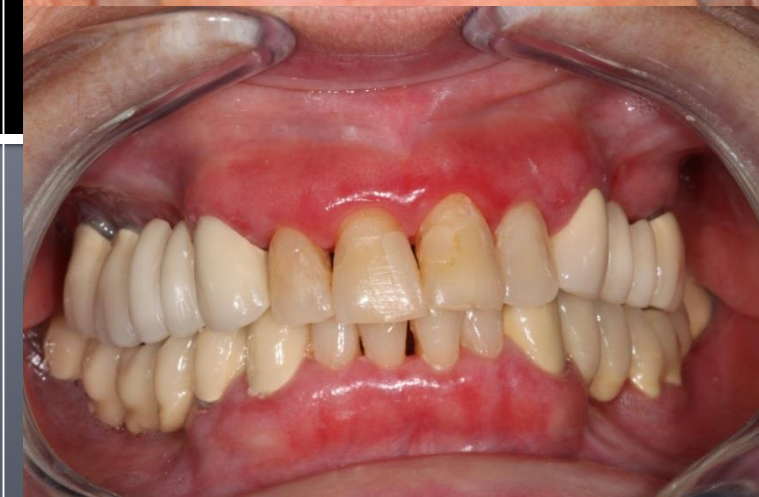
oral hygiene should be optimized prior to the beginning of steroid treatment.

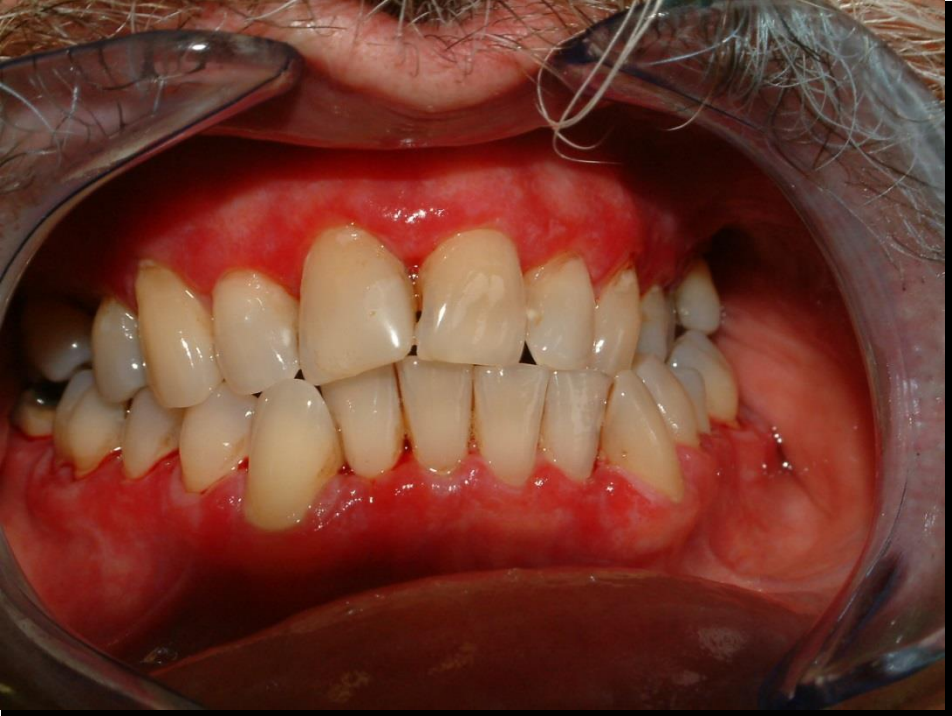
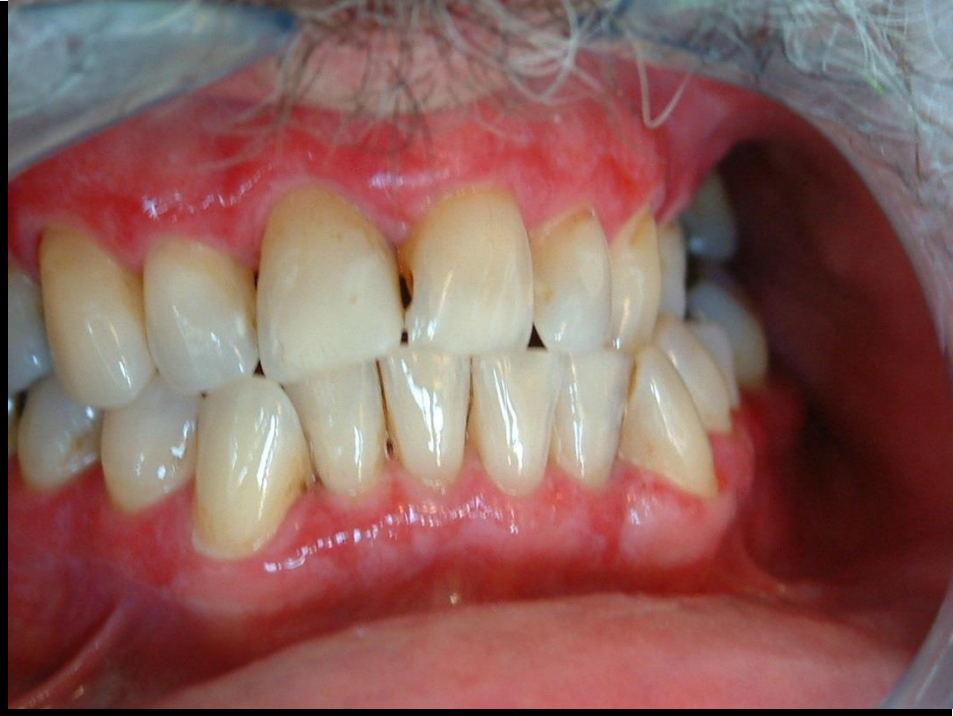
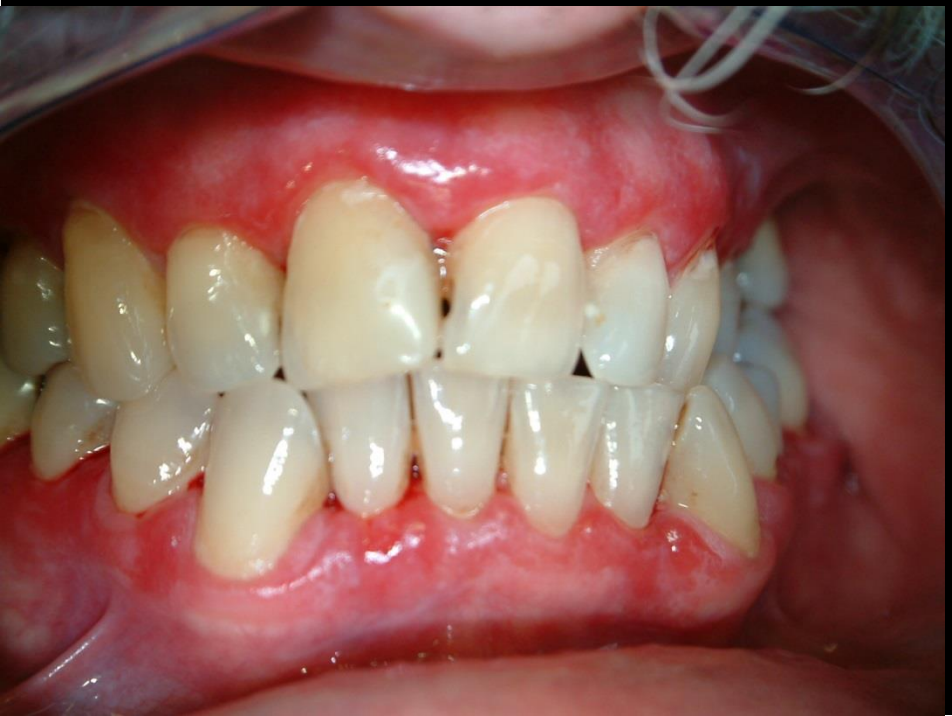
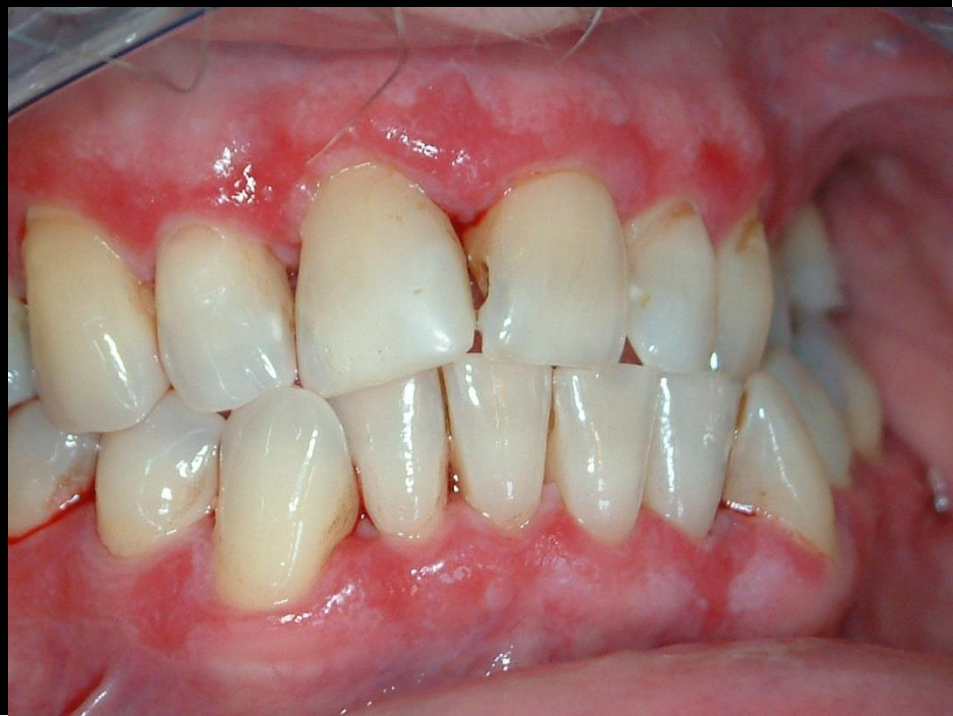
.

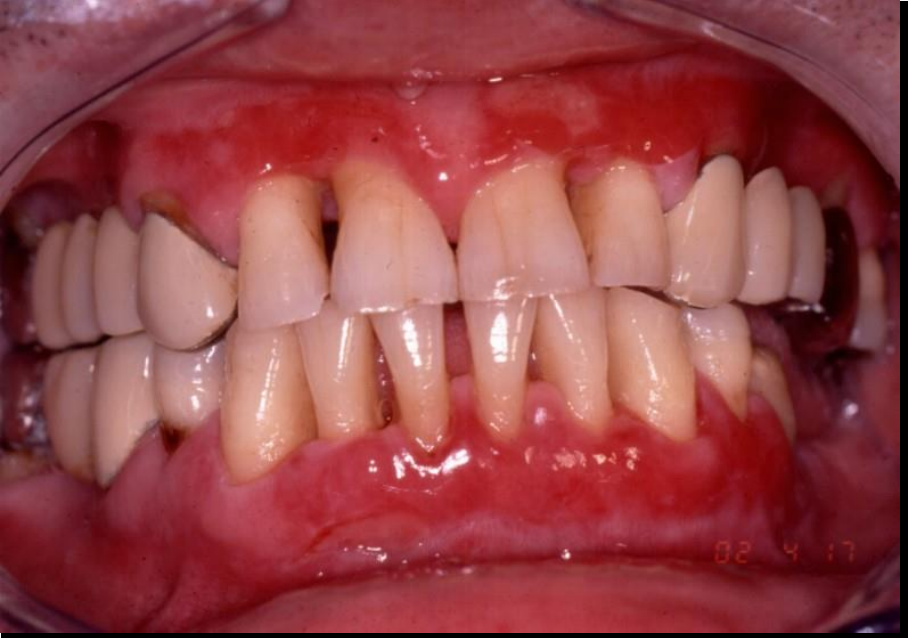
If symptoms persist, steroid gels





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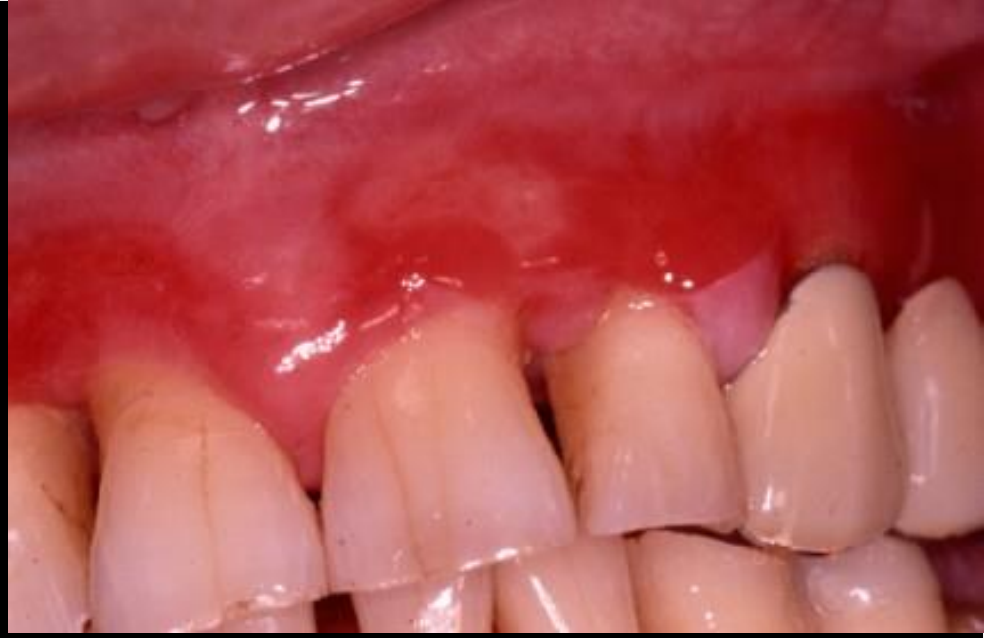

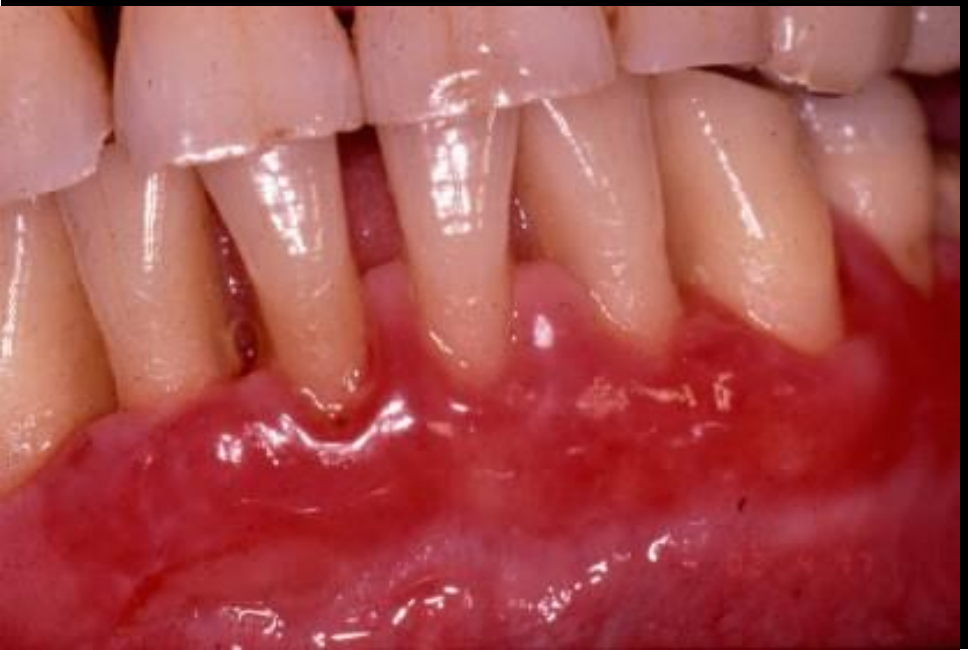

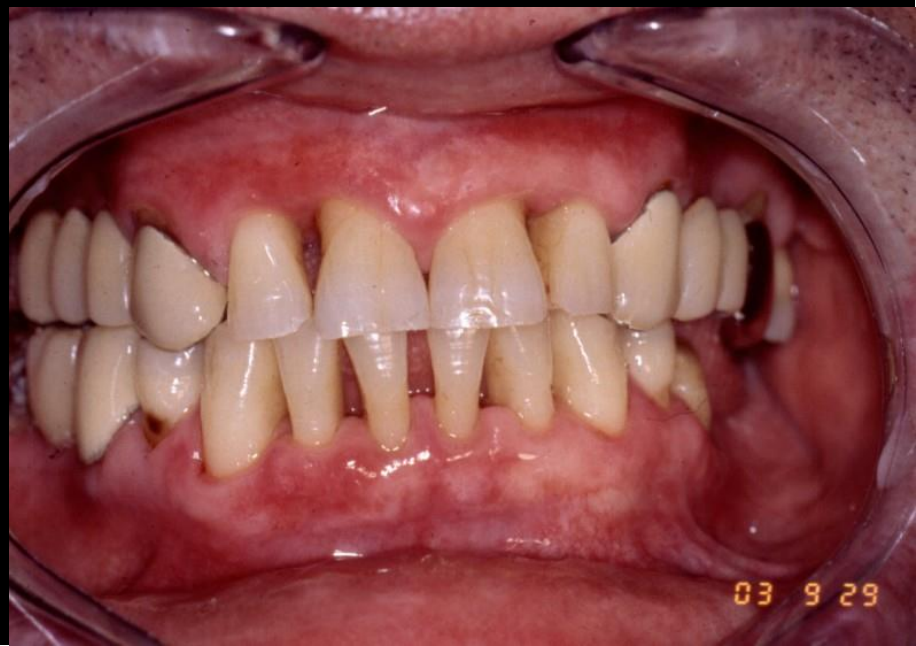
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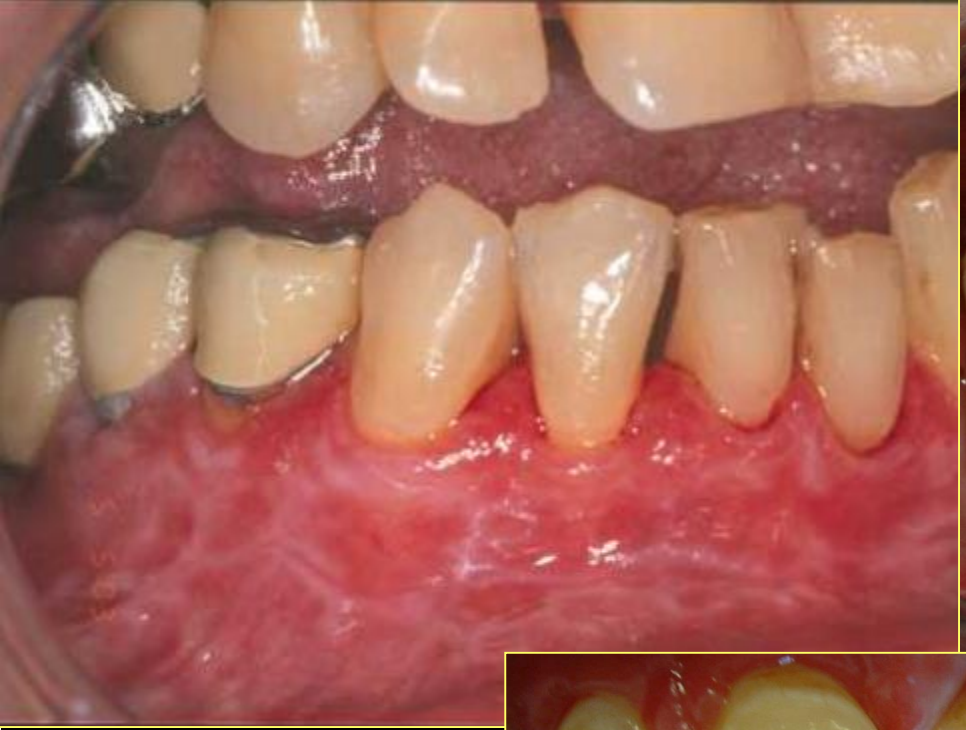
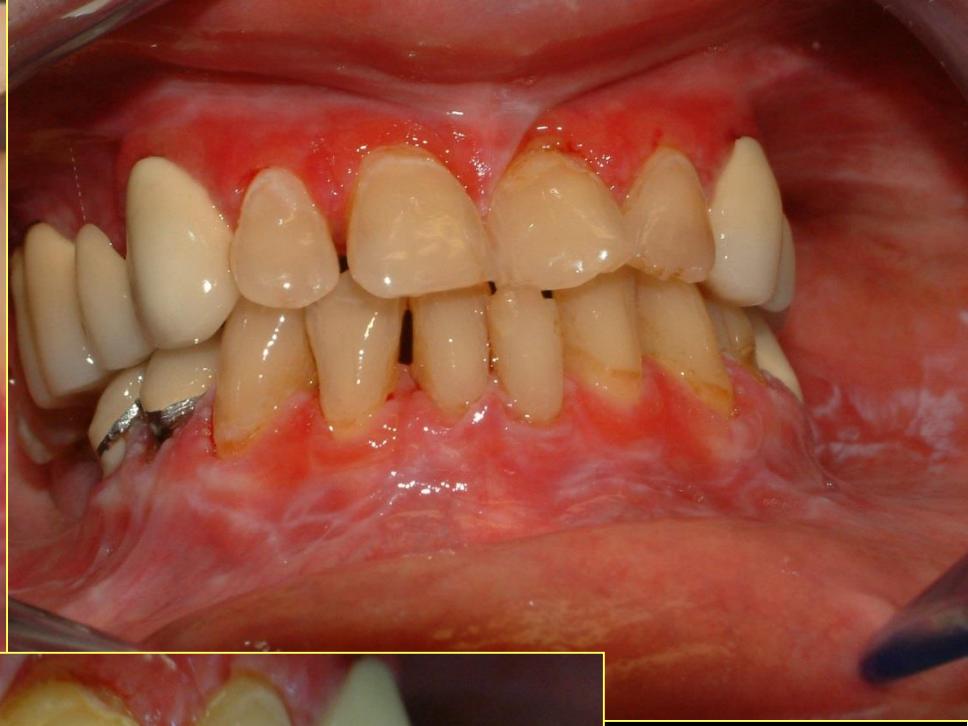
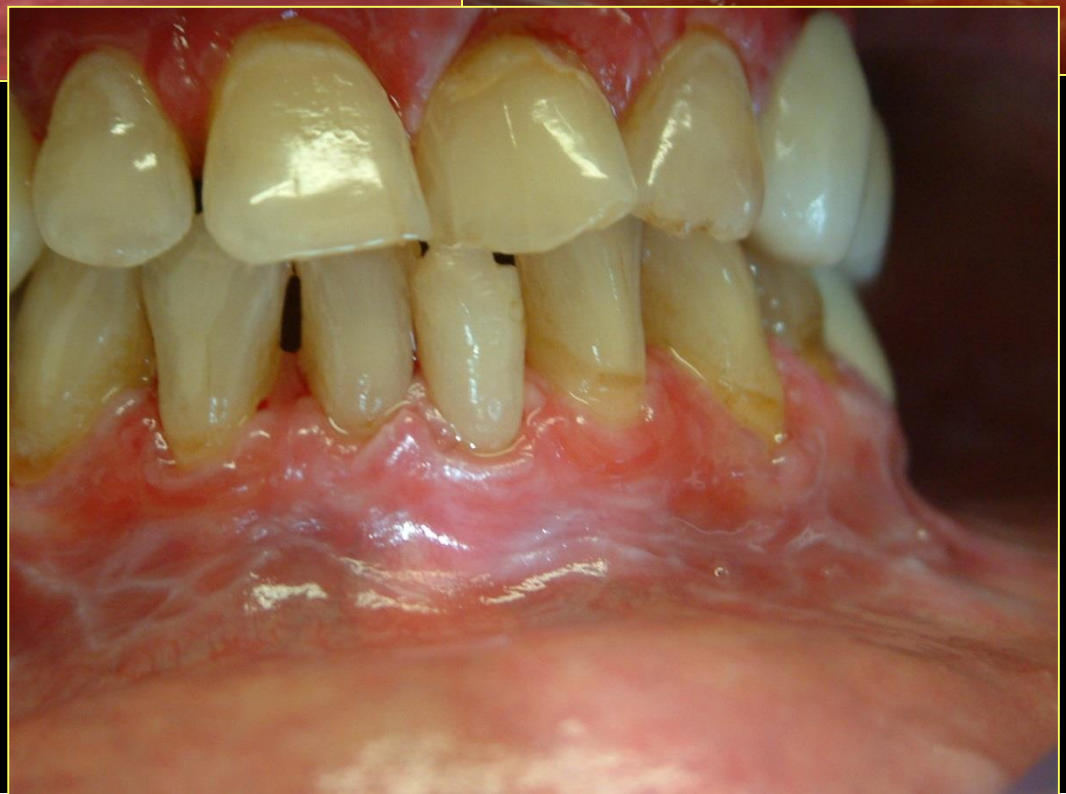

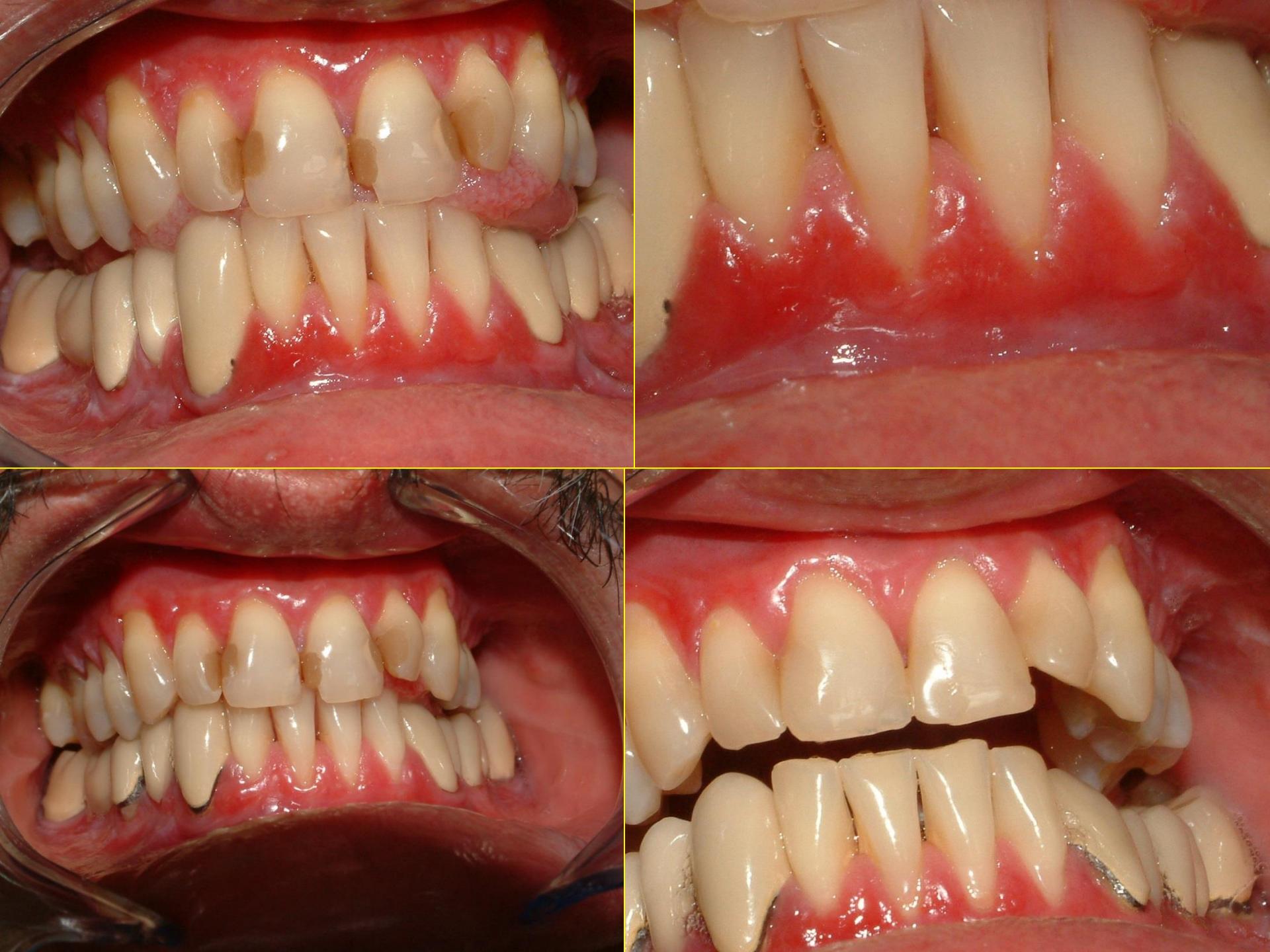

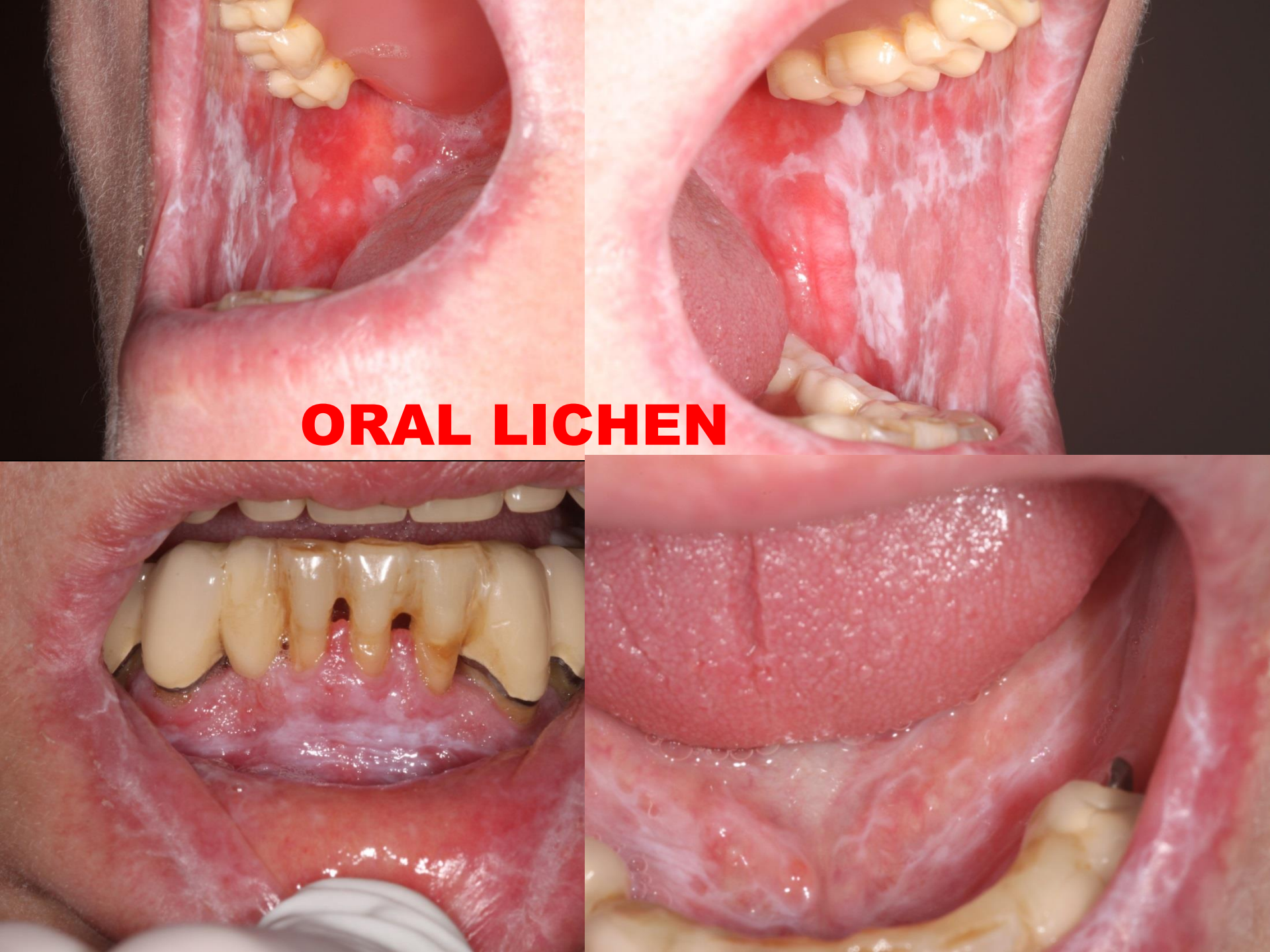
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