White lesions

Dr. Suhail Al-Amad

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White lesions

- Hereditary
  - Leukoedema
  - WSN
- Reactive
  - Trauma
  - Tobacco
- Infectious
  - Fungal
  - Viral
- Immune-mediated
  - OLP
  - Lupus
- Preneoplstic / Neoplastic
  - Leukoplakia
  - SCC

Leukoedema

- Common, not a disorder, but a normal variation.
- Asymptomatic —> incidental finding
- > in blacks.
- Diffuse white-gray opacification on the buccal mucosa; key:
  - Symmetrical
  - Disappears on stretching

Leukoedema

- Histopathology;
  - Hyperplastic epithelium (acanthosis) with parakeratinization
  - Intracellular oedema

- No treatment.

Source: Laskaris, Pocket Atlas of Oral Diseases 2006. Page 31

White Sponge Naevus

- Autosomal dominant.
- Mutations to genes responsible for producing keratin types 4 and/or 13.
- Asymptomatic —> incidental finding.
- Appears early in life
- Bilateral and symmetrical
- Thickened irregular white patch, affecting any mucosal tissue; buccal, GIT, genital mucosae.

White Sponge Naevus

- Histopathology;
  - Significantly thickened (hyperplastic) epithelium with parakeratinization
  - Hydropic changes
  - Parakeratin streaks extending into the epithelium.

- No treatment.

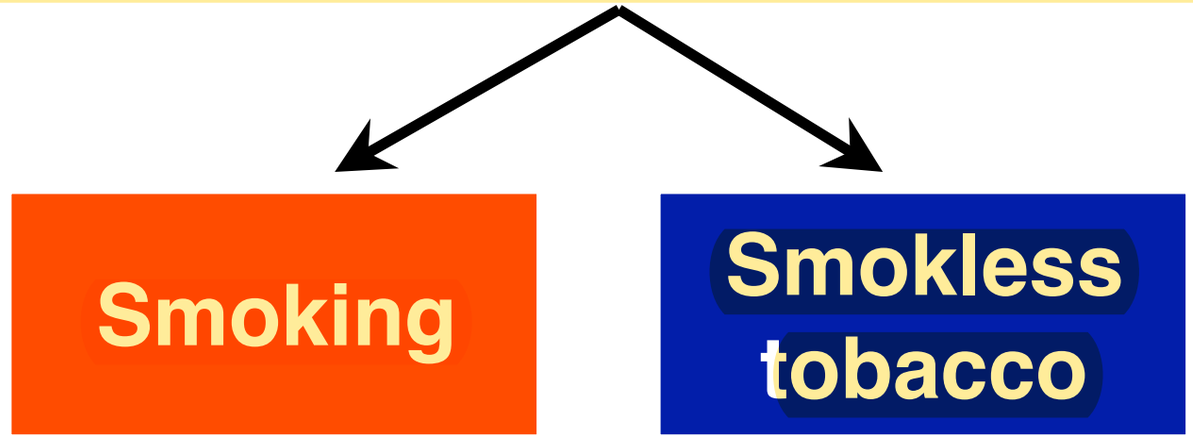
White lesions

Hereditary	Reactive	Infectious	Immune-mediated	Preneoplstic / Neoplastic
~~Leukoedema~~	Trauma	Fungal	OLP	Leukoplakia
~~WSN~~	Tobacco	Viral	Lupus	SCC

Trauma-induced white lesions

- Chronic low grade trauma.
- Trauma induces the epithelial cells to multiply (hyperplasia) and produce keratin (keratosis and hyperkeratosis).
- Cause-effect relationship is seen.
- Changes are reversible. Reduction or disappearance of the lesions should be observed.

Tobacco-induced white lesions



Tobacco-induced white lesions

Smoking

Smokless tobacco

•Heat and toxins in the smoke result in mucosal changes; (*mucosa becomes white*).

•Mainly seen in cigar and pipe.

•Affects all oral mucosae, but seen more prominently in the palate --> Nicotine Stomatitis

•Histologically: hyperplasia, hyperkeratosis of epithelium. Inflammation and squamous metaplasia in salivary gland ducts.

•Reversible, very low malignant potential.

•An indicator of heavy smoking.

Tobacco-induced white lesions

Smoking

Smokless tobacco

Sources: Newland JR. *et al* Oral Soft Tissue Diseases 2001

•Increasingly common in India, SE Asia (*cultural reasons*), and in North Europe and the US (*banning smoking in public areas*).

•Tobacco is either chewed or retaining in the mouth (mostly in the cheek pouch). The latter is called Snuff.

•Oral mucosal changes (**wrinkled white patch**) occur as a result of carcinogens, various additives and the high pH of smokeless tobacco.

•Asymptomatic, has a malignant potential for SCC or verrucous carcinoma.

White lesions

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Oral Candidosis

- The most commonly seen oral fungal infection is Candidosis, caused by *Candida albicans*.
- *Candida albicans* is a normal oral flora (>50% healthy carriers), it becomes pathogenic following local and/or systemic predisposing factors;

Local

- Xerostomia
- Reduced OVD
- Poor denture hygiene

Systemic

- Extremities of age
- Broad-spectrum antibiotics
- Corticosteriods
- Immune suppression; HIV, drugs
- Advancved malignancy/blood dyscrasias
- Hospitalization
- Malnutrition
- Endocrine disturbances
- Smoking

Classification

Oral candidosis can be classified as:

- Acute
  - atrophic
  - pseudomembranous
- Chronic
  - atrophic
  - hyperplastic
- Mucocutaneous
  - usually T cell deficiency (congenital or acquired)

Acute atrophic candidosis

- Sudden onset
- Generalised or focal areas of red/inflamed oral mucosa
  - associated with dentures (denture sore mouth)
  - antibiotic use (acute antibiotic stomatitis)
- No specific symptoms other than pain
- Difficult to diagnose
  - may get positive smears from red areas or associated dentures

Management

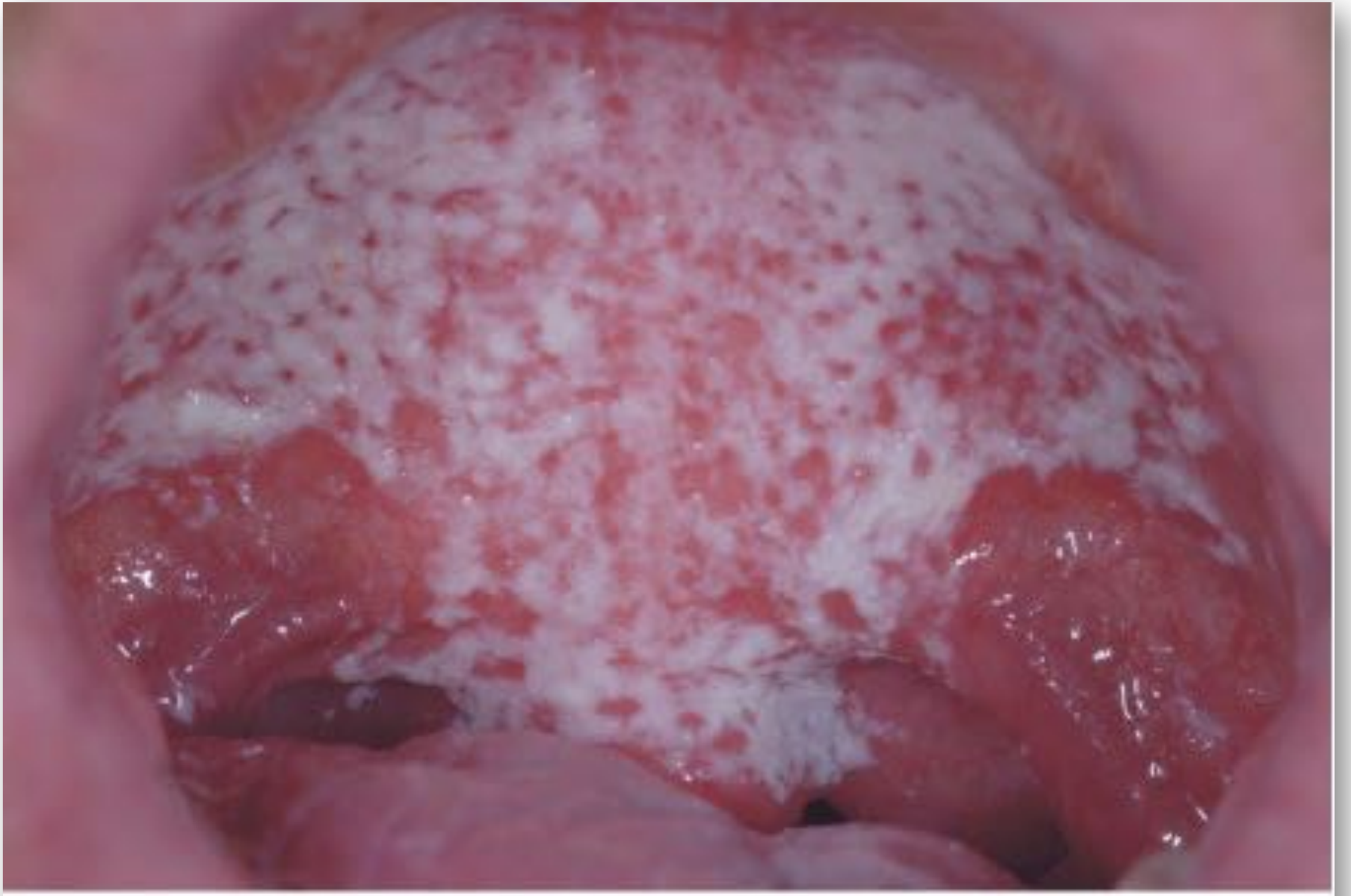
- Correct diagnosis
- Oral/denture hygiene
- Antifungal agents

Acute pseudomembranous candidosis

- Also known as *oral thrush*.
- Usually have history of predisposing factors
- Symptoms range from nothing to severe discomfort

Clinical features

- Areas of generalised or focal inflammation
- Presence of soft, white/yellow plaques that can be lifted off the mucosa
- These plaques represent inflammatory exudate, dead cells and fungal colonies



Source: Knoop KJ, Stack LB, Storrow AB, Thurman RJ: *The Atlas of Emergency Medicine, 3rd Edition:* http://www.accessmedicine.com

Image source: McGraw-Hill Access Medicine

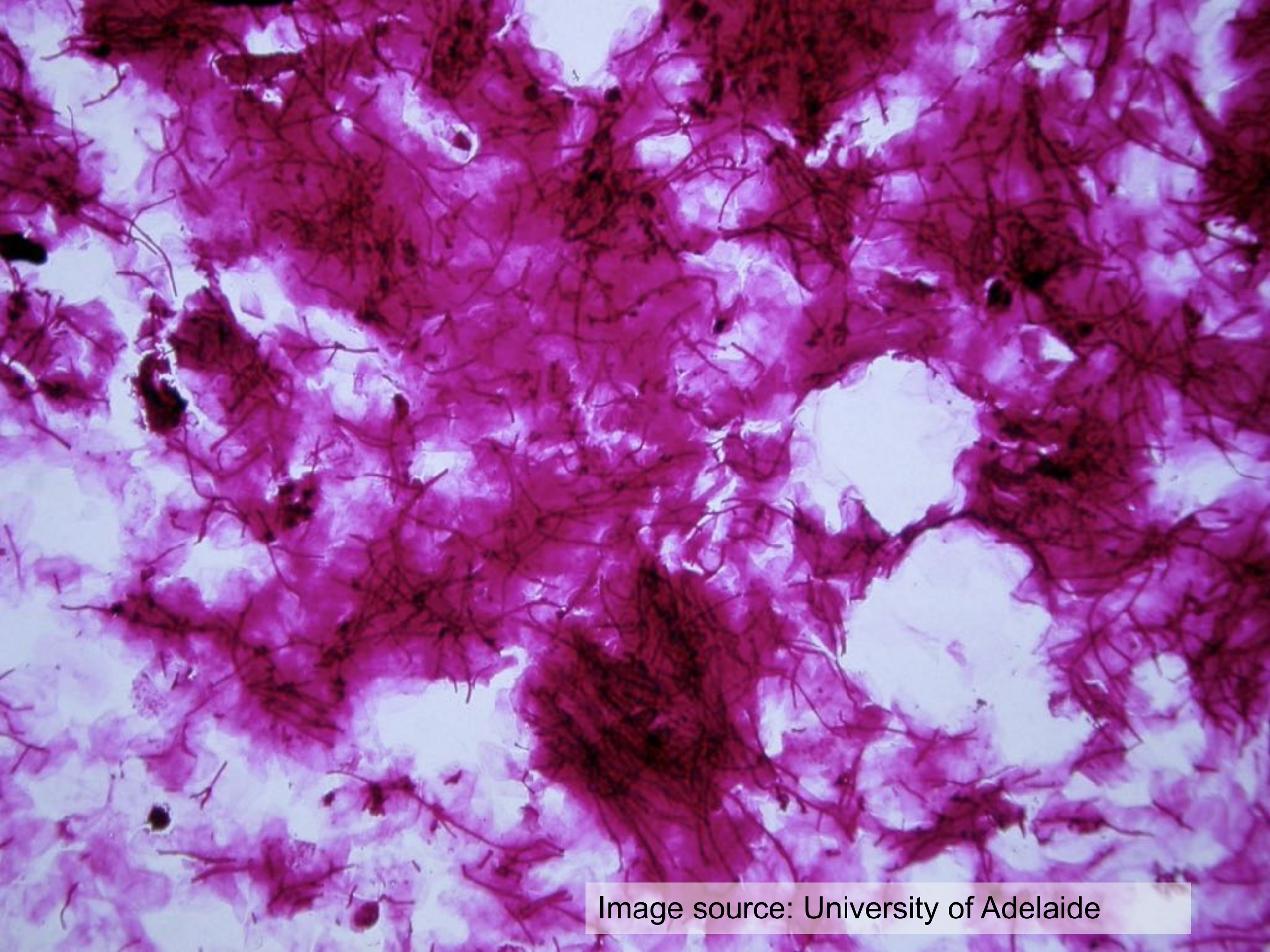


Image source: University of Adelaide

Acute pseudomembranous candidosis

- Diagnosis based on oral features and cytology smear
- Management depends on severity of symptoms and particular predisposing factors:
  - For a simple case, e.g. following antibiotic therapy:
    - topical antifungal agents and oral hygiene instruction
  - For a complex case, e.g. immuno-suppressed patient:
    - medical consultation and topical/systemic antifungal agents

Chronic atrophic candidosis

•Various forms with different clinical presentations including:

- median rhomboid glossitis
- papillary hyperplasia of the palate
- angular cheilitis
- non-specific red areas in mouth
- chronic denture stomatitis

Image source: University of Adelaide

Chronic Atrophic candidosis

- Generally symptomless but sometimes patients present with a mild burning sensation
- Diagnosis by recognition of clinical lesion(s) and cytology smear

Management

- Determine and treat predisposing factor(s)
- Local and/or systemic
  - topical antifungal agents

Chronic Hyperplastic Candidosis

- Lesions present as:
  - single or multiple area
  - fixed
  - white or mixed white/red patches on the mucosa
- May occur anywhere but common sites are the tongue and buccal mucosa
- Mainly seen in adults and usually symptomless
- Present for extended time period
- Do not always have easily determined predisposing factor(s)
  - e.g. mild xerostomia, subclinical anaemia, vitamin deficiency

Chronic Hyperplastic Candidosis

- Correct diagnosis important because lesion can resemble other pathology including lichen planus and early squamous cell carcinoma
- Requires biopsy as cytology smear not always reliable

Histopathology

- Hyperparakeratosis, acanthosis, Candidal organisms and inflammatory cells (‘microabscesses’) in superficial layers of epithelium. Generalised chronic inflammation in underlying CT.

Chronic Hyperplastic Candidosis

Management

- Confirm diagnosis
- Check and treat predisposing factors
- Topical antifungal agents

Epstein-Barr virus

Oral Hairy Leukoplakia

- Lateral border of the tongue
- Associated with HIV infection, clinical importance:
  - Marker for HIV disease progression
  - Marker of viral load
  - Indication of effectiveness of antiretroviral medication
- HIV negative patients
  - Organ transplants
  - Bone marrow/stem cell transplants

Oral hairy leukoplakia

- White patch
- Lateral border of tongue
- Often bilateral
- Irregular surface, prominent ridges or folds
- Extend onto ventral surface of tongue, flat appearance
- Involvement of buccal mucosa

Oral hairy leukoplakia

Histology

- Epithelial hyperplasia
- Acanthosis, parakeratosis
- Corrugated surface
- ‘Koilocyte-like’ cells in prickle cell layer
- Dysplasia
  - very rare
- Mild or absent inflammatory cell infiltrate

White lesions

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- Infectious
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  - OLP
  - Lupus
- Preneoplstic / Neoplastic
  - Leukoplakia
  - SCC

Oral Lichen Planus

- Chronic muco-cutanueous disease
- Most patients >40 years of age
- 65% patients female
- Common oral sites;
  - Buccal mucosa
  - Dorsal surface of tongue
  - Gingiva
- Often bilateral lesions
- Cutaneous lesions may be present

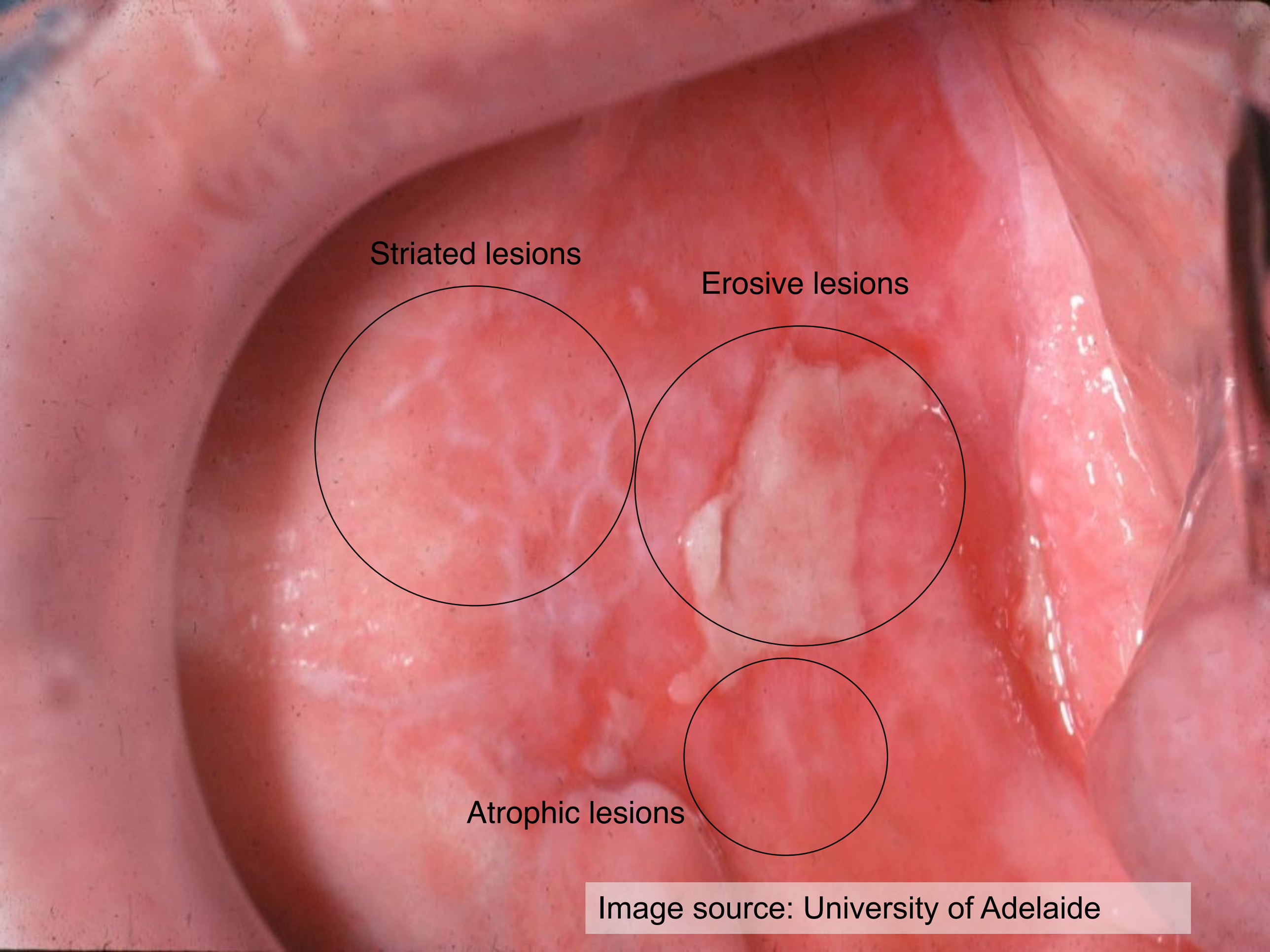
Reported prevalence

= 0.2-2.2% of the population

Oral Lichen Planus

Clinically;

- Lesions comprise
  - Striae
    - Most common
    - Sharply defined
    - Lacy, starry or annular patterns
  - Atrophic areas
    - Red areas, thin mucosa
  - Erosions
    - Shallow areas of ulceration
  - Plaques



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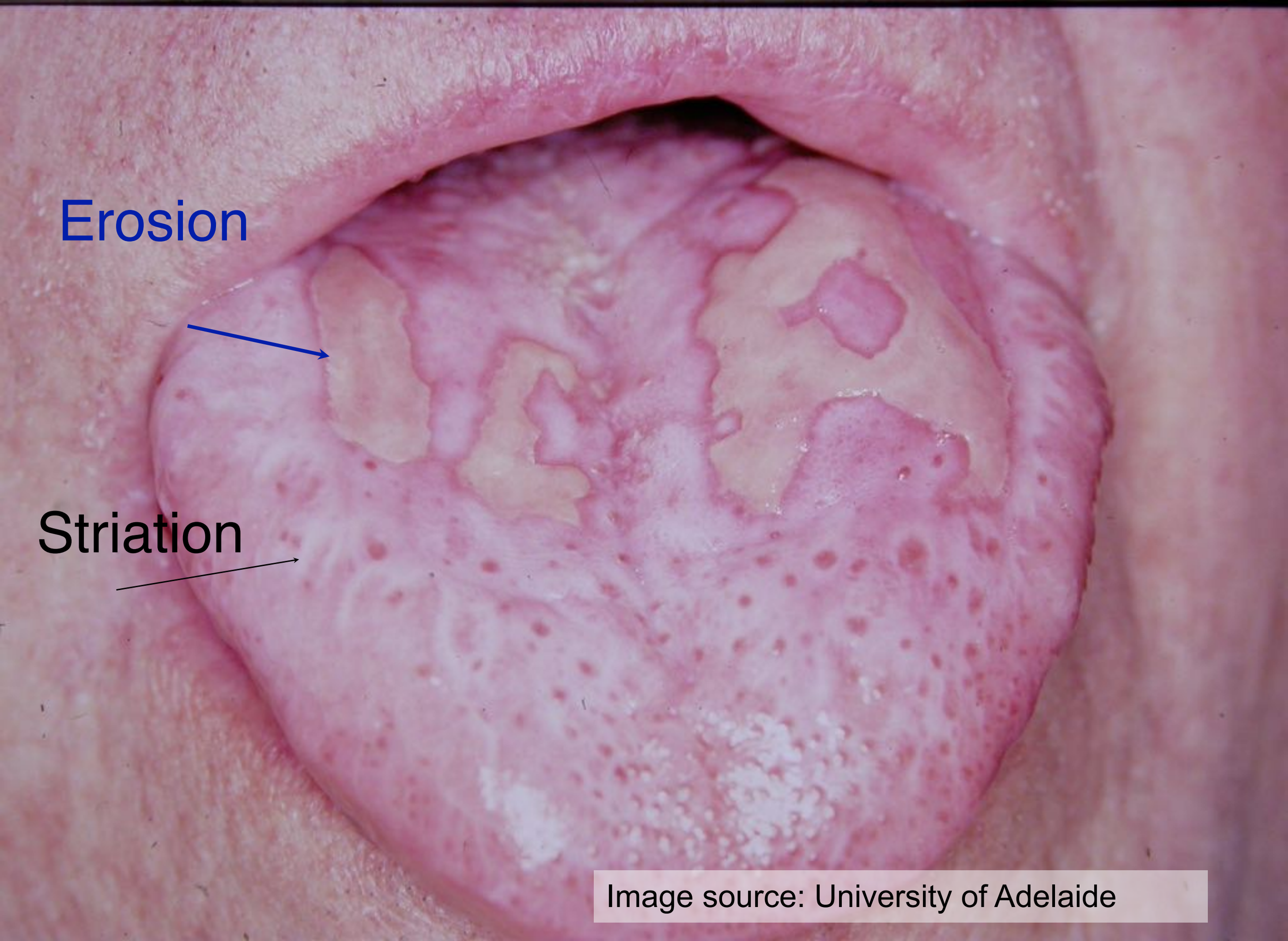


Image source: University of Adelaide

Oral Lichen Planus

Gingival lesions;

- Distinguish from other forms of gingival inflammation
- Often lesions atrophic
- Striae are uncommon, may be present in other areas of the mouth
- Oral hygiene difficult

Lichen Planus

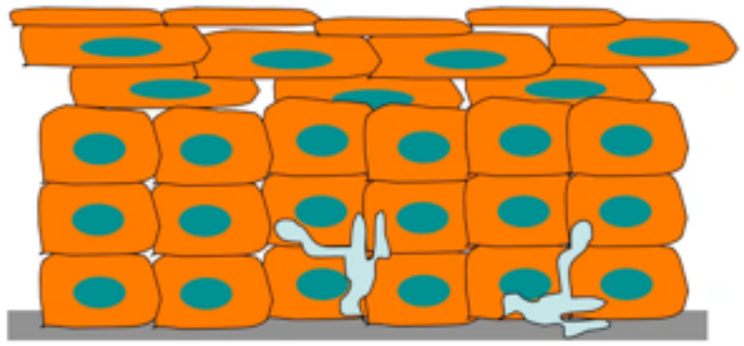
Skin lesions;

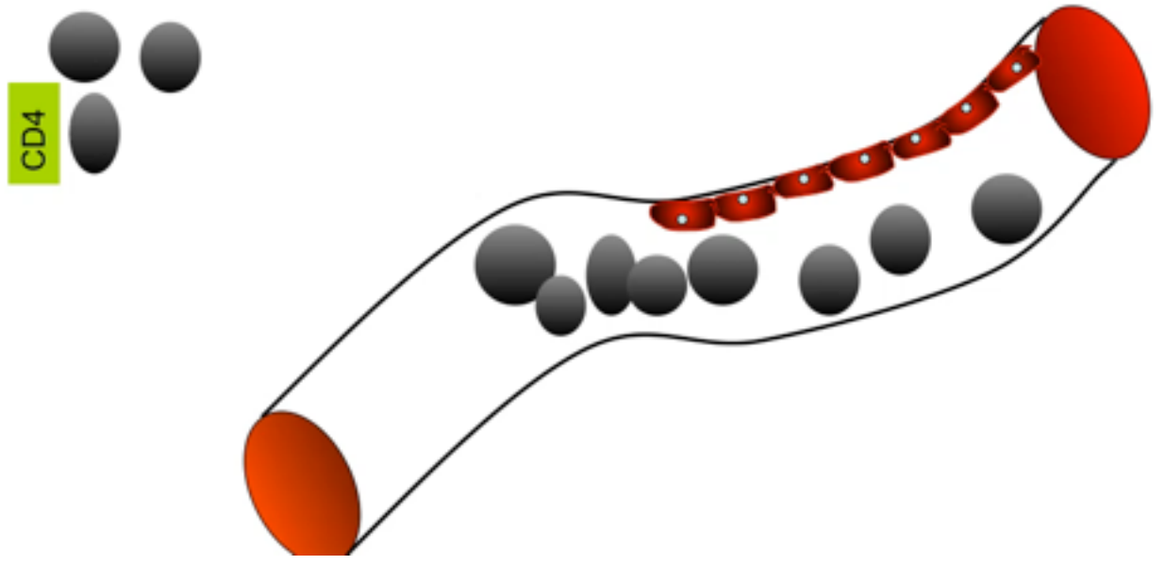
- Common
- Seen in 20-60% in patients with oral disease
- 4 **P**s --> *P*urple, *P*ruritic, *P*olygonal *P*apules
- Superficial fine white striae
  --> Wickham's striae
- Most common sites is flexor surface of wrists

Oral Lichen Planus

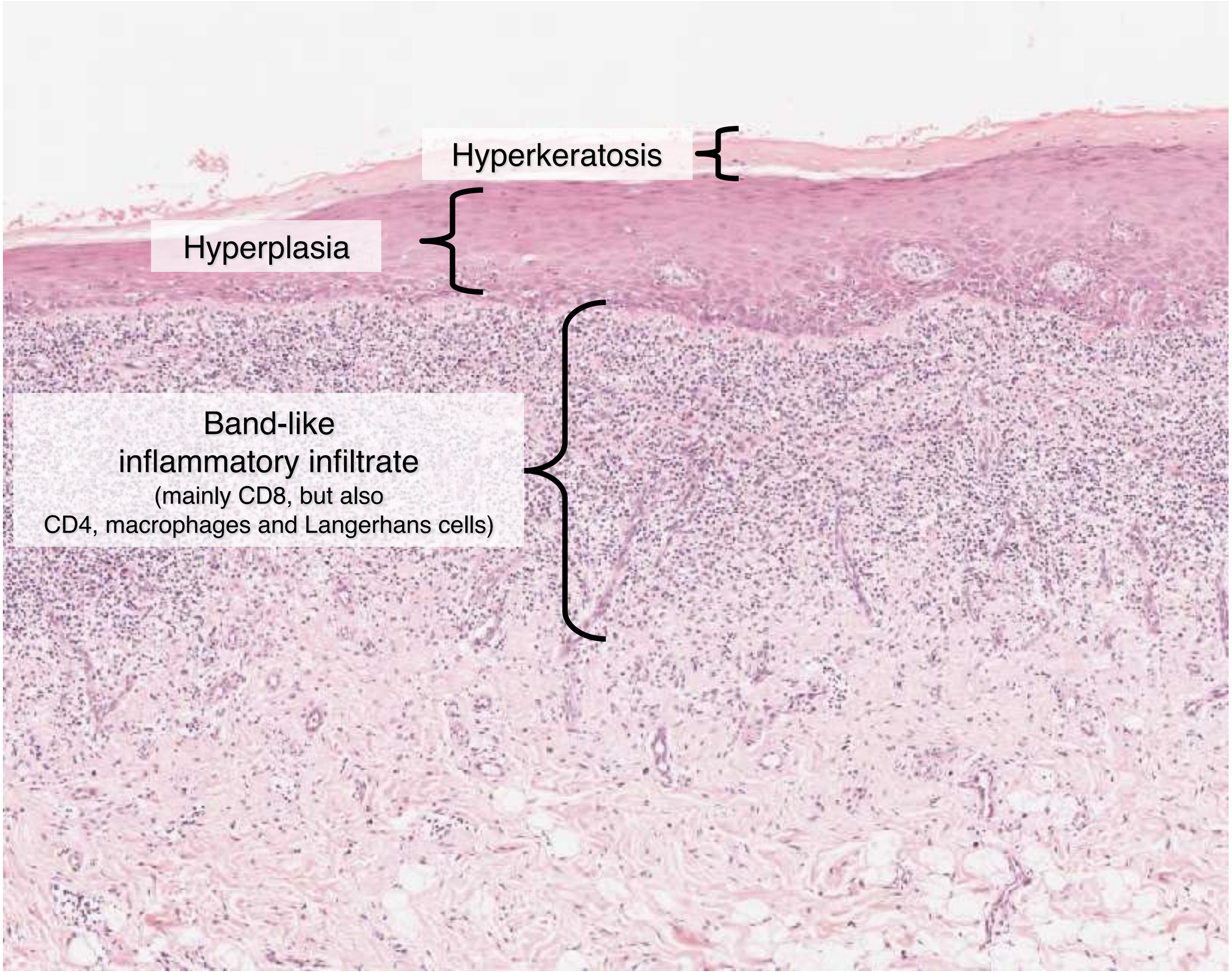
Histopatholoy;

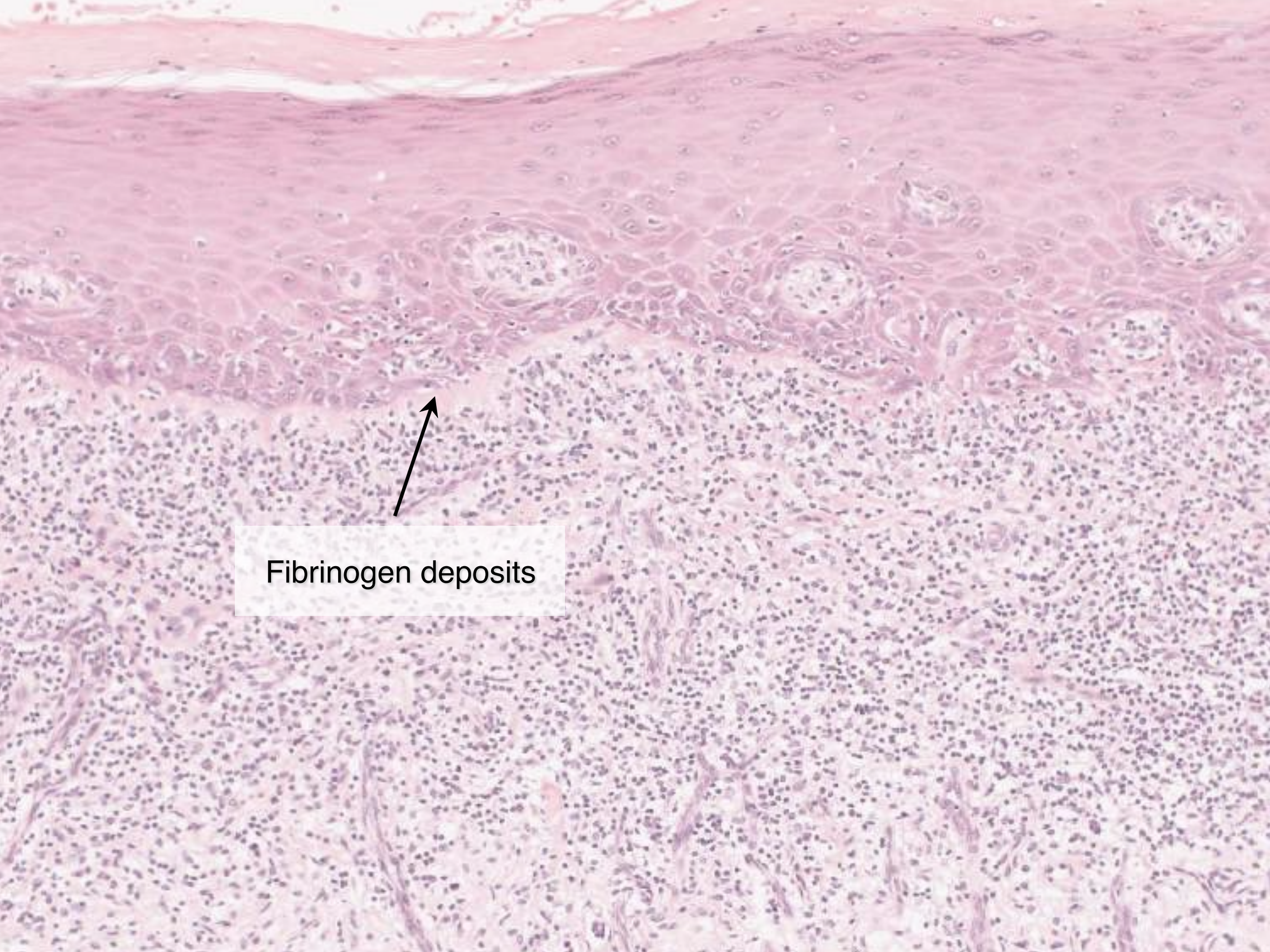
- Striated lesions
  - Hyperkeratosis, hyperparakeratosis
  - Saw-tooth appearance of rete pegs (mainly in skin)
  - Band-like lymphocytic infiltrate subjacent to basement membrane
  - Basal cell layer degeneration (vaculative necrosis)
    - Formation of **Civatte bodies**
  - Immunofluorescence studies demonstrate fibrinogen deposition in basement membrane

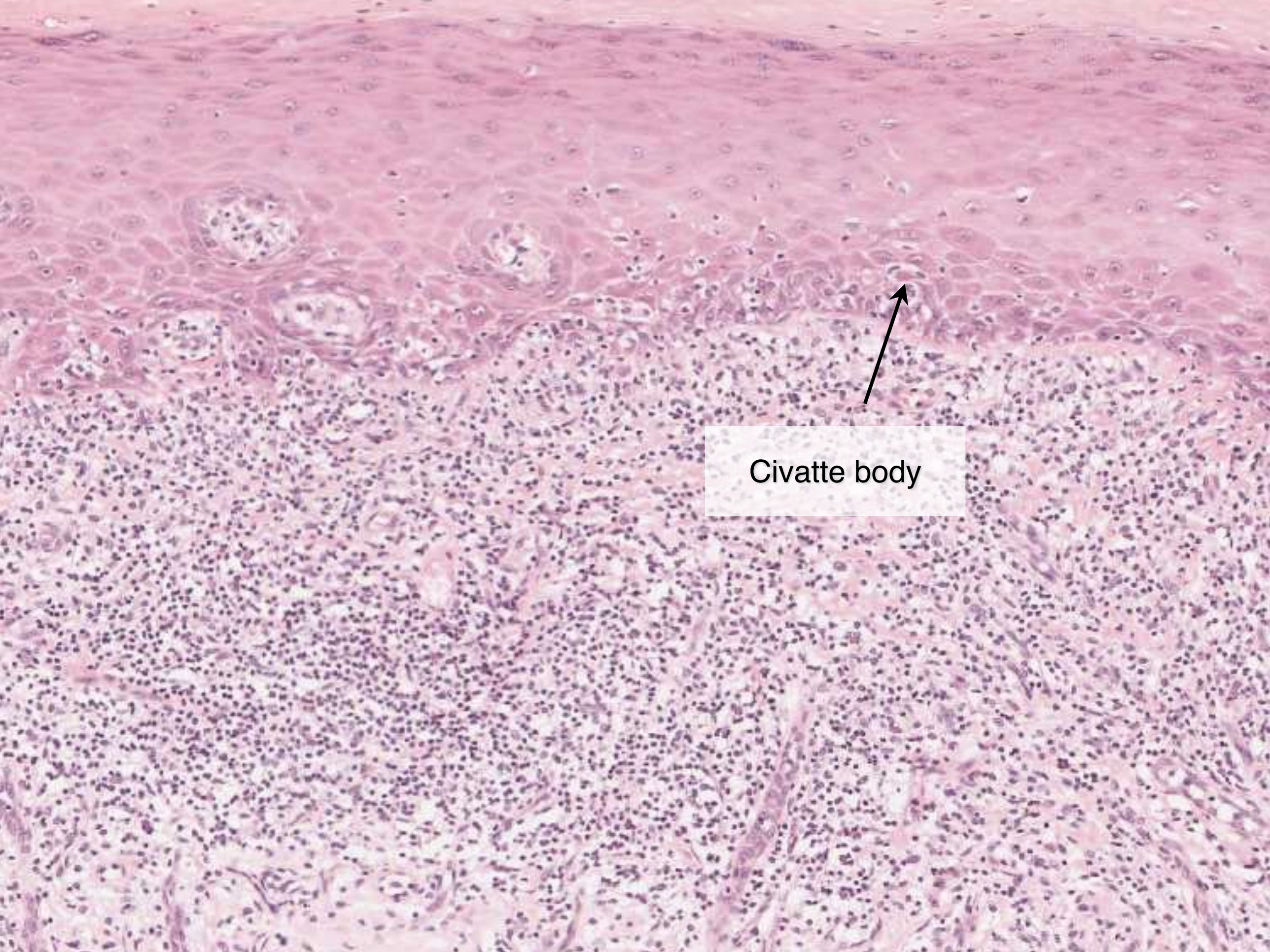




- Atrophic lesions
  - Severe thinning and flattening of epithelium


Hyperkeratosis
Hyperplasia
Band-like
inflammatory infiltrate
(mainly CD8, but also
CD4, macrophages and Langerhans cells)


Fibrinogen deposits


Civatte body

Oral Lichen Planus

Importance of OLP;

- Symptomatic
- Resembles more serious conditions
- Malignant potential
  - Lichenoid dysplasia
  - Reported transformation rate --> 0.2% - 3.7%
- ? Relation with Hep C

Lesions with “lichenoid” appearance

- Clinical and histological similarity to OLP;

1. Lichenoid contact lesions
2. Lichenoid drug reactions
3. Lichenoid lesions of graft versus host disease

Oral lichenoid contact lesions

- Allergic contact stomatitis
- Delayed immune related hypersensitivity reaction
- Restorative materials

Oral lichenoid drug reaction

- Allopurinol
- Angiotensin-converting enzyme II inhibitors
- Enalapril
- Frusemide
- Gold salts
- Hydroxychloroquine
- Mercury
- Methyldopa
- NSAIDS
- Phenothiazine
- Propranolol
- Quinidine
- Tetracyclines
- Thiazides

Oral Lichen Planus

- Control of symptoms
  - Chlorhexidine
- Corticosteroids
  - Modulate inflammation and immune response
  - Topical, local injection, systemic steroids
- Antifungal therapy
  - Treat secondary infection

Lupus erythematosus

- Two main forms of Lupus
  - Systemic
  - Discoid

Discoid lupus erythematosus

Clinical features;

- Female 9:1
- Middle age
- Skin lesions —> face and scalp, sun exposed areas
  - Rash
- Disc-shaped erythematous plaques
- Hair follicle involvement —> permanent hair loss
- Arthralgia
- 25% patients have mucous membrane lesions

Discoid lupus erythematosus

Oral lesions;

- Buccal mucosa, gingiva, vermilion
- Plaques or erosions
- White, keratotic striae
- Progression of DLE to SLE rare, although may potentially occur

Systemic lupus erythematosus

- Autoimmune disease
- Mild skin and mucous membrane involvement
- Aggravated by sunlight, UV light, fluorescent lights
- Autoantibodies directed against DNA
  - Immune complex reactions
- Multiple system involvement

Systemic lupus erythematosus

- Multiple system involvement

Skin	“Butterfly” rash, alopecia, photosensitivity
Joints	Non-erosive polyarthritis
Kidney	Glomerulonephritis
Mucous membranes	Oral ulceration
Nervous system	Polyneuritis, cerebritis
Vascular	Raynaud’s phenomenon
Heart	Pericarditis, endocarditis, myocarditis
Lungs	Pleuritis
Eyes	Anterior uveitis
Blood	Autoimmune haemolytic anaemia, thrombocytopaenia

Fever

Systemic lupus erythematosus

Clinically;

- Young woman
- Fever, malaise, anaemia
- Arthralgia
- Rash

Systemic lupus erythematosus

- Skin lesions
  - Erythematous rash
  - Malar process and bridge of nose
  - Characteristic butterfly distribution

- Oral lesions
  - Similar to that seen in DLE
  - Erosion, ulceration, keratosis
  - Vermilion, buccal mucosa, gingiva and **palatal involvement**

Systemic lupus erythematosus

Histopathology;

- Irregular pattern of acanthosis and atrophy of epithelium
- Basal cell layer degeneration
- Thickened basement membrane (PAS positive)
- Variable inflammatory cell infiltrate
  - Variable density
  - Not band-like, extends deep into connective tissue
  - Perivascular infiltrate

DIF;

- Anti-DNA histone
- Anti-single stranded DNA
- IgG anti-double stranded DNA

Systemic lupus erythematosus

Discoid
Lupus Erythematosus

Topical Steroids

Systemic
Lupus Erythematosus

Systemic Steroids
Immunosuppressive agents
Organ-specific treatments

White lesions

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