Autoimmune and vesiculobullous diseases of the oral cavity.

Autoimmune and vesiculobullous diseases of the oral cavity.

Microspecimens:

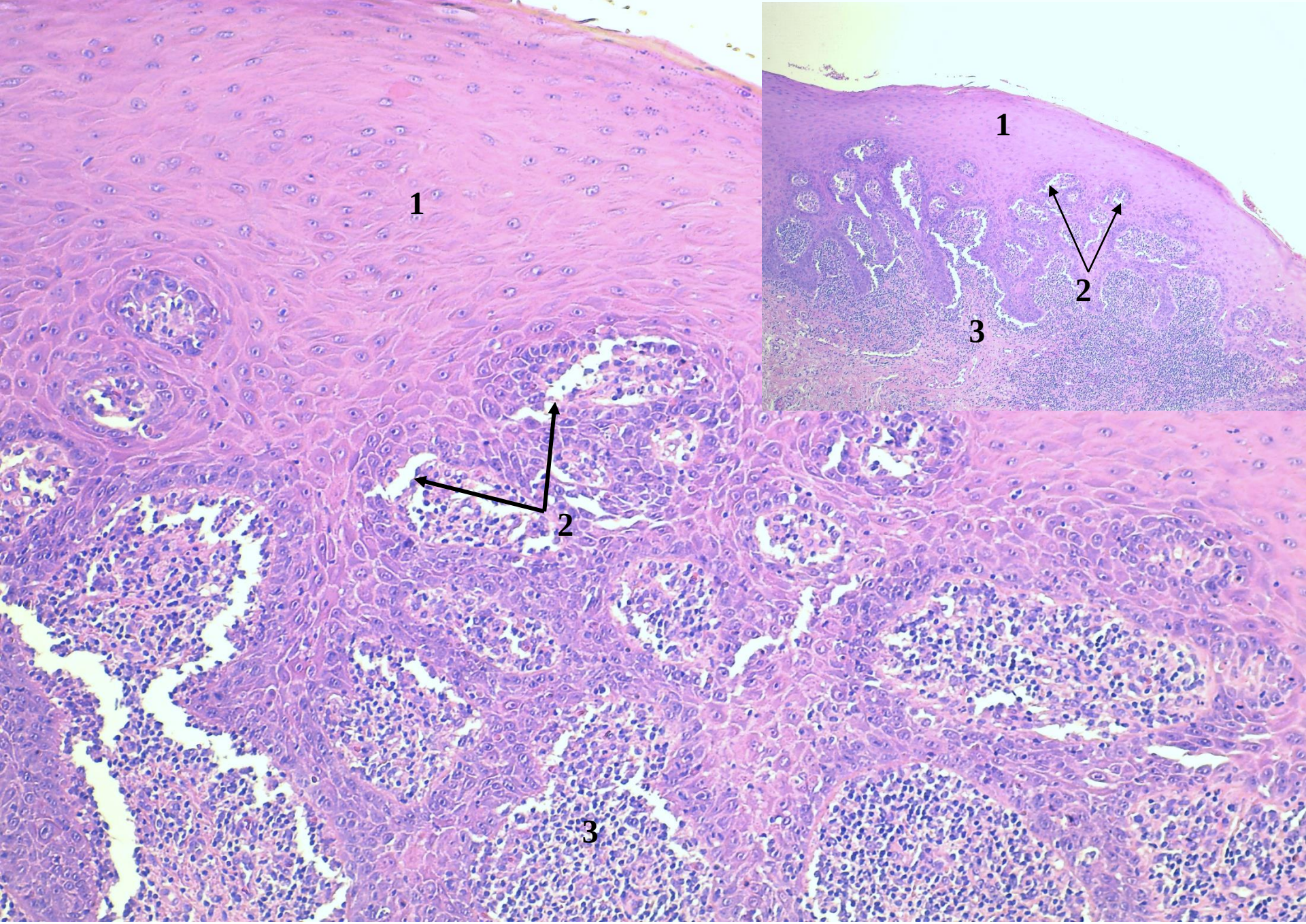
№ OP 27. Oral lichen planus. *(H-E. stain).*

Indications:

1. Hyperplasia of the surface epithelium.
2. Basal layer vacuolization.
3. Lymphocytic infiltrate at the epithelium–connective tissue interface

The microscopic criteria for lichen planus include hyperkeratosis, basal layer vacuolization with apoptotic keratinocytes, and a lymphophagocytic infiltrate at the epithelium–connective tissue interface. Over time, the epithelium undergoes gradual remodeling, resulting in reduced thickness and occasionally a sawtooth rete ridge pattern. Within the epithelium are increased numbers of Langerhans cells (as demonstrated by immunohistochemistry), presumably processing and presenting antigens to subjacent T cells. Discrete eosinophilic ovoid bodies representing the apoptotic keratinocytes are noted at the basal zone. These colloid, or Civatte, bodies are seen in other conditions such as drug reactions, contact hypersensitivity, LE, and some nonspecific inflammatory reactions. Direct immunofluorescence demonstrates the presence of fibrinogen in the basement membrane zone in 90% to 100% of cases. Although immunoglobulins and complement factors may be found as well, they are far less common than fibrinogen deposits.

Clinical overdiagnosis of lichen planus, coincidental occurrence of lichen planus and oral cancer, and microscopic confusion with dysplasias that have lichenoid features have contributed to the controversy over malignant potential of this disease. Nonetheless, it appears that there is a small but bona fide risk that oral squamous cell carcinoma will develop in oral lichen planus, but this risk is very low (approximately 1% at 5 years) and probably is lower than reported rates. If malignant transformation occurs, it is more likely to be associated with erosive and atrophic forms of the disease and particularly in those who smoke. Because lichen planus is a chronic condition, patients should be observed periodically and should be offered education about the clinical course, rationale of therapy, and possible risk of malignant transformation.



№ OP 27. Oral lichen planus. *(H-E. stain).*

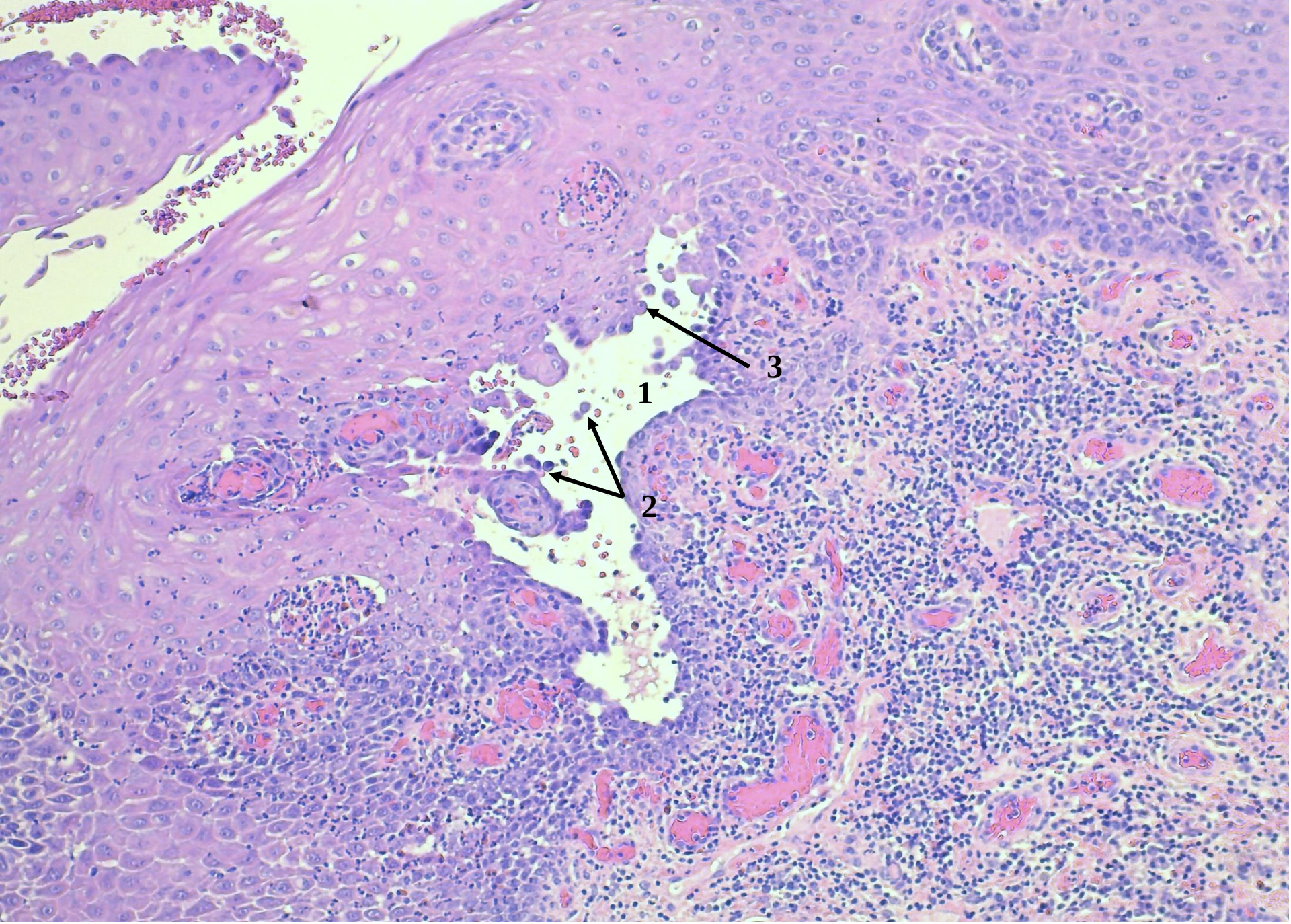
Microspecimens:

№ OP 29. Oral Pemphigus. *(H-E. stain).*

Indications:

1. Intraepithelial separation (vesicle)
2. Free-floating keratinocytes (Tzanck cells).
3. Adherent basal cells over the lamina propria.

Pemphigus vulgaris appears as intraepithelial clefting with keratinocyte acantholysis. Loss of desmosomal attachments and retraction of tonofilaments result in free-floating, or acantholytic, Tzanck cells. Bullae are suprabasal, and the basal layer remains attached to the basement membrane. In addition to standard biopsy, confirmation of pemphigus can be made with the use of direct immunofluorescence (DIF) testing. DIF testing uses a biopsy specimen in an attempt to demonstrate autoantibodies already attached to the tissue. This is preferable to less sensitive indirect immunofluorescence, which uses patient serum to identify circulating antibodies. In pemphigus vulgaris, DIF testing of perilesional tissue almost always demonstrates intercellular autoantibodies of the IgG type. C3 and, less commonly, IgA can be detected in the same intercellular fluorescent pattern. Paraneoplastic pemphigus demonstrates an antigen-antibody interaction and complement activation producing intraepithelial suprabasal acantholysis, as well as immunoglobulin deposition along the basement membrane zone, leading to severe and intractable stomatitis. Of note, this form of immunopathology extends to other tissues, including the lungs, heart, bladder, and liver, with autoantibodies attacking or denaturing components of the cytoplasmic portion of the desmosome (desmoplakins I and II).



№ OP 29. Oral Pemphigus. *(H-E. stain).*

Microspecimens:

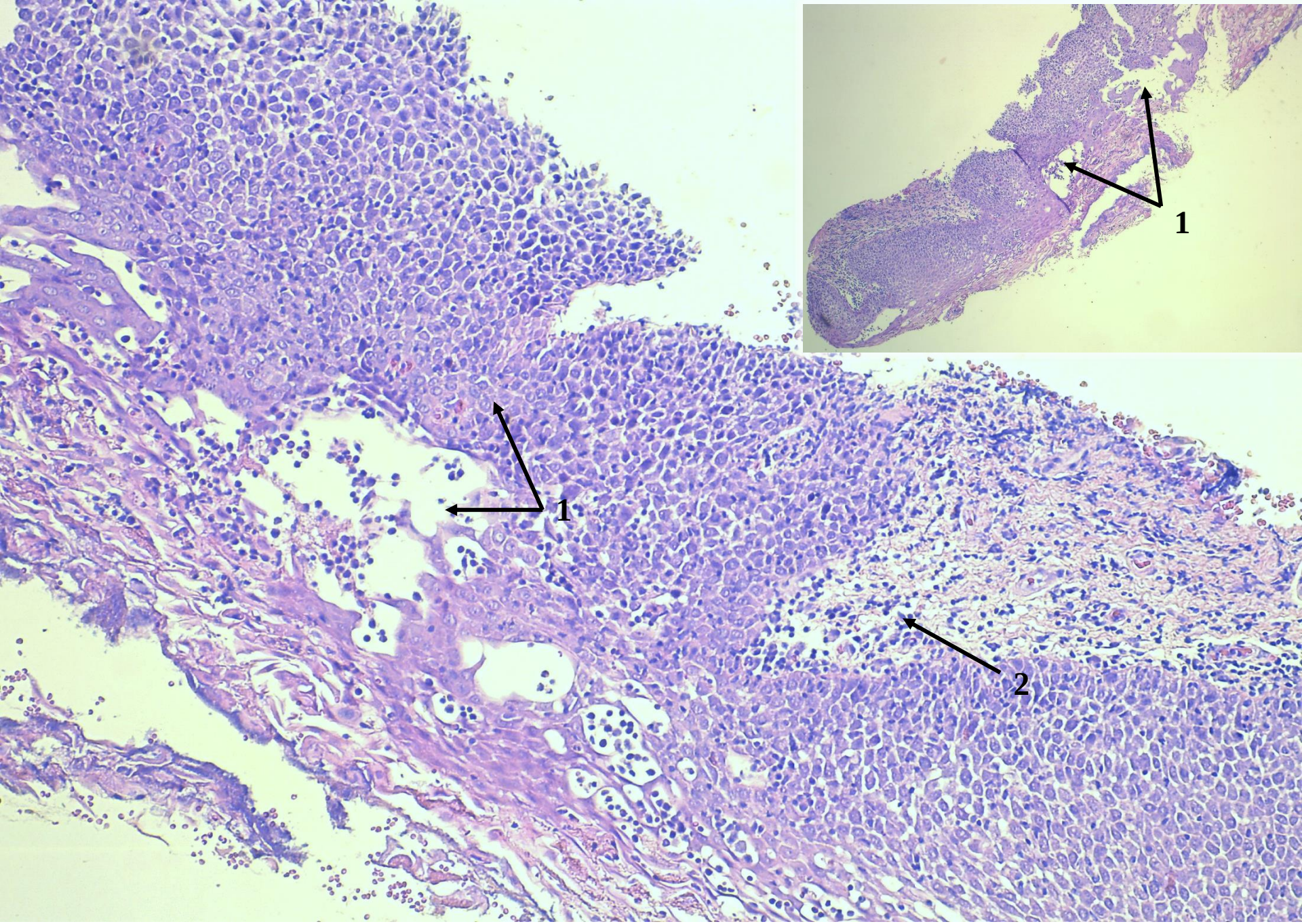
№ OP 30. Oral erythema multiforme. *(H-E. stain).*

Indications:

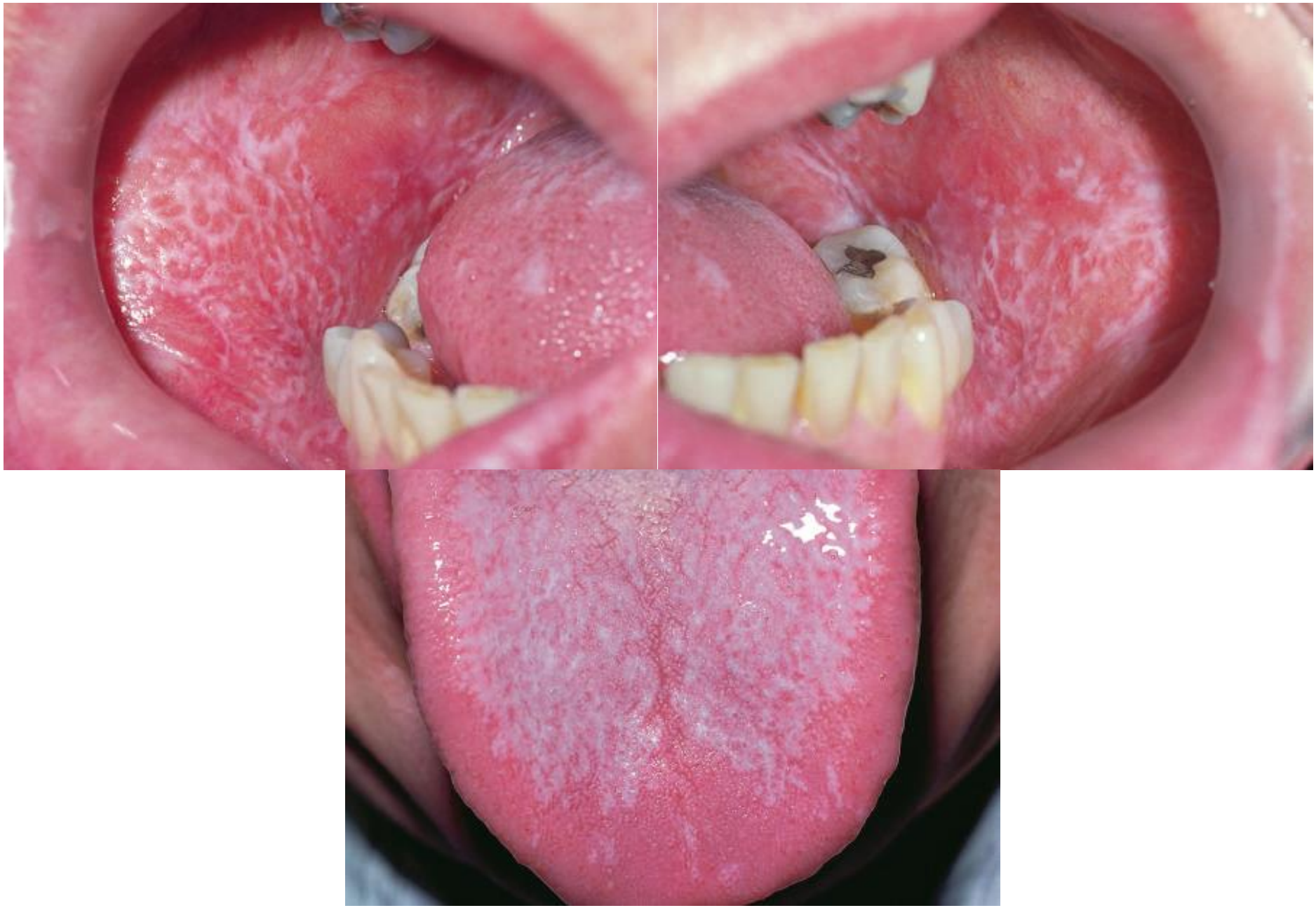
1. Epithelial hyperplasia and spongiosis.
2. Lymphocytic infiltrate.

The microscopic pattern of EM varies but consists of epithelial hyperplasia and spongiosis. Basal and parabasal apoptotic keratinocytes are usually seen. Vesicles occur at the epithelium–connective tissue interface, although intraepithelial vesiculation may be seen. Epithelial necrosis is a frequent finding. Connective tissue changes usually appear as infiltrates of lymphocytes and macrophages in perivascular spaces and in connective tissue papillae. Immunopathologic studies are nonspecific for EM. The epithelium shows negative staining for immunoglobulins. Vessels have been shown, however, to have IgM, complement, and fibrin deposits in their walls. This latter finding has been used to support an immune complex vasculitis cause for EM. Autoantibodies to desmoplakins 1 and 2 have been identified in a subset of EM major–affected patients, suggesting that both cell-mediated and humoral immune systems may contribute to the pathogenesis of EM.

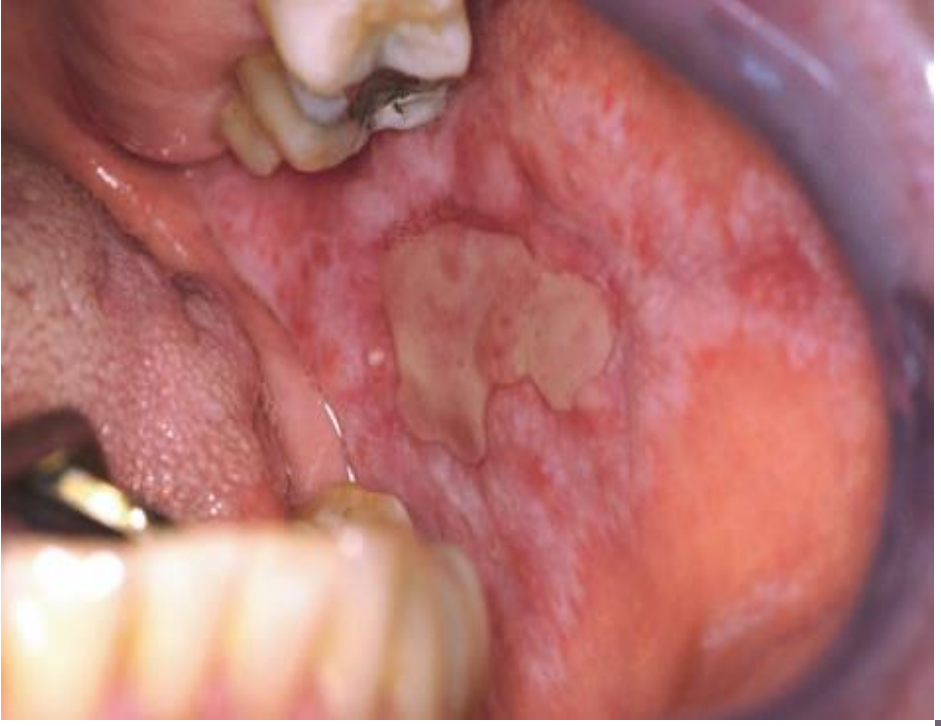
The basic cause of EM is unknown, although a hypersensitivity reaction is suspected. Some evidence suggests that the disease mechanism may be related to antigen-antibody complexes that are targeted for small vessels in the skin or mucosa. In about half of cases, precipitating or triggering factors can be identified. These generally fall into the two large categories of infections and drugs. Other factors, such as malignancy, vaccination, autoimmune disease, and radiotherapy, are occasionally cited as possible triggers.



№ OP 30. Oral erythema multiforme. *(H-E. stain)*

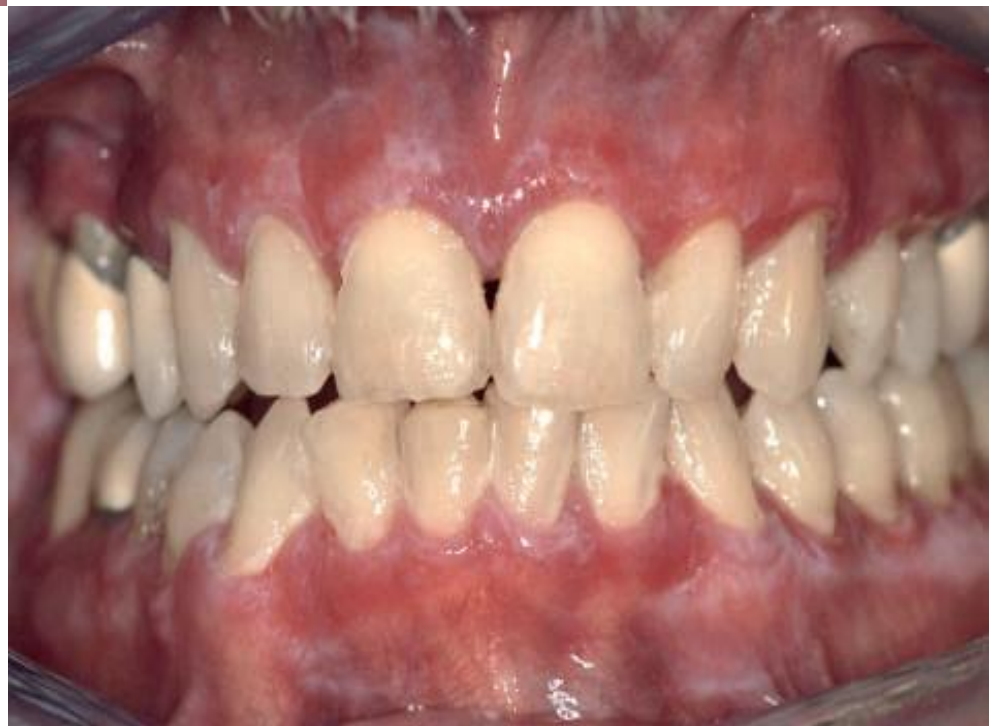


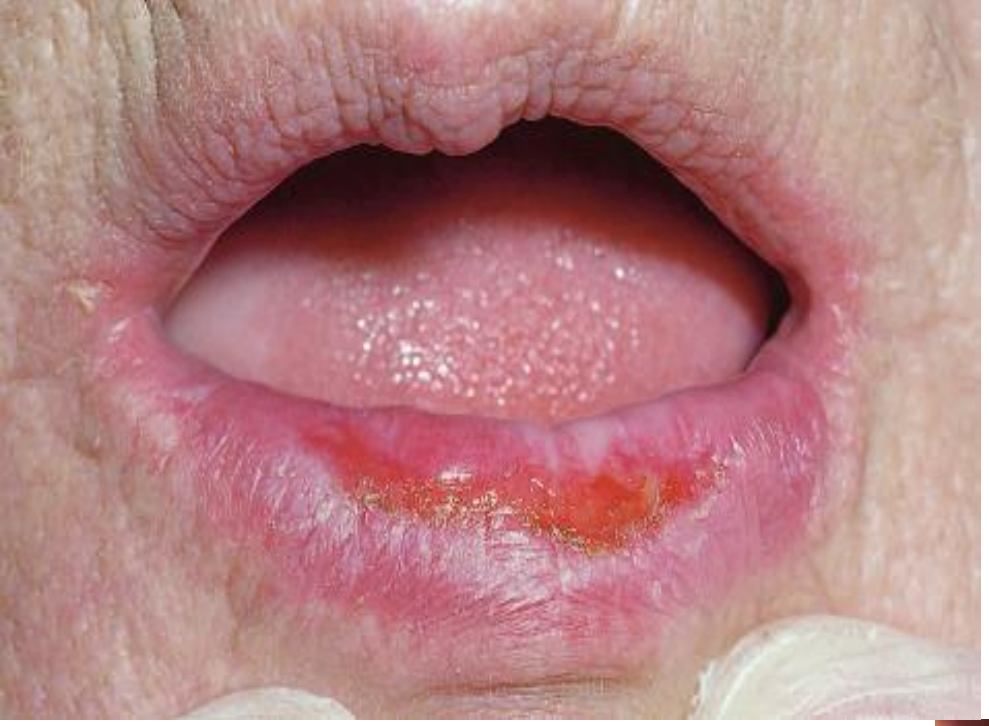
Oral lichen planus, reticular form.



Erythematous lichen planus of the gingiva.

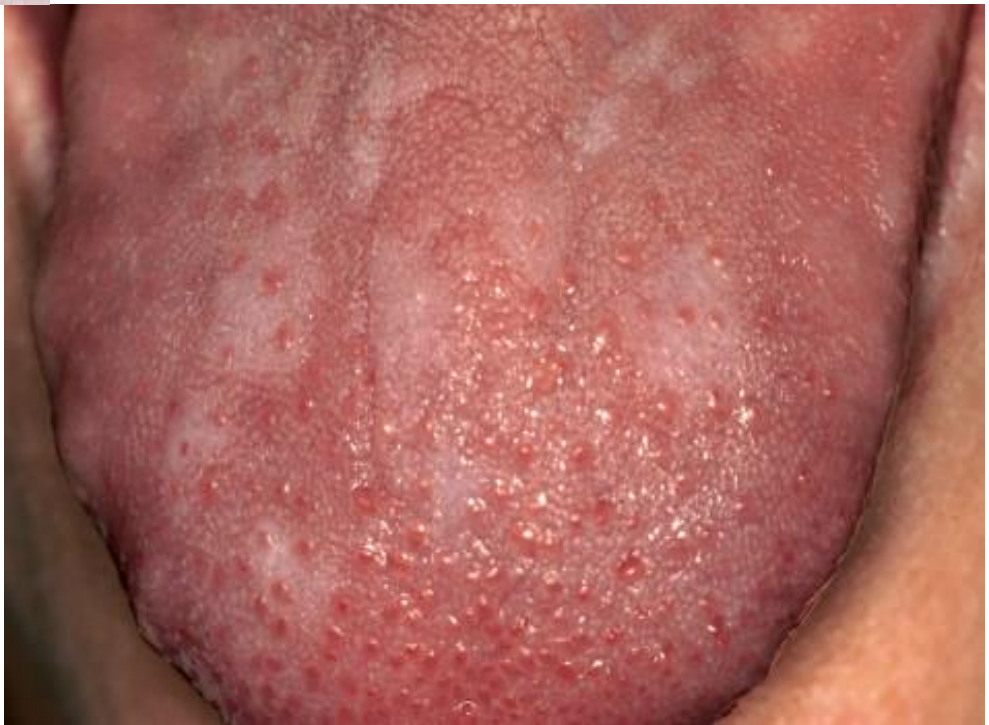
Oral lichen planus, erosive form.

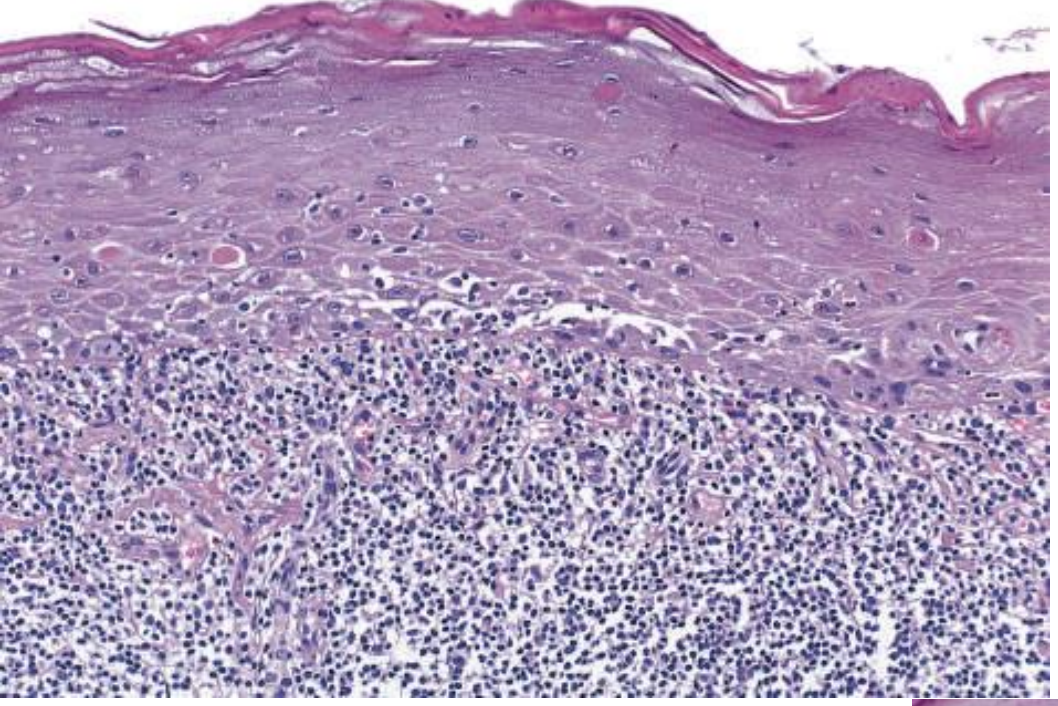


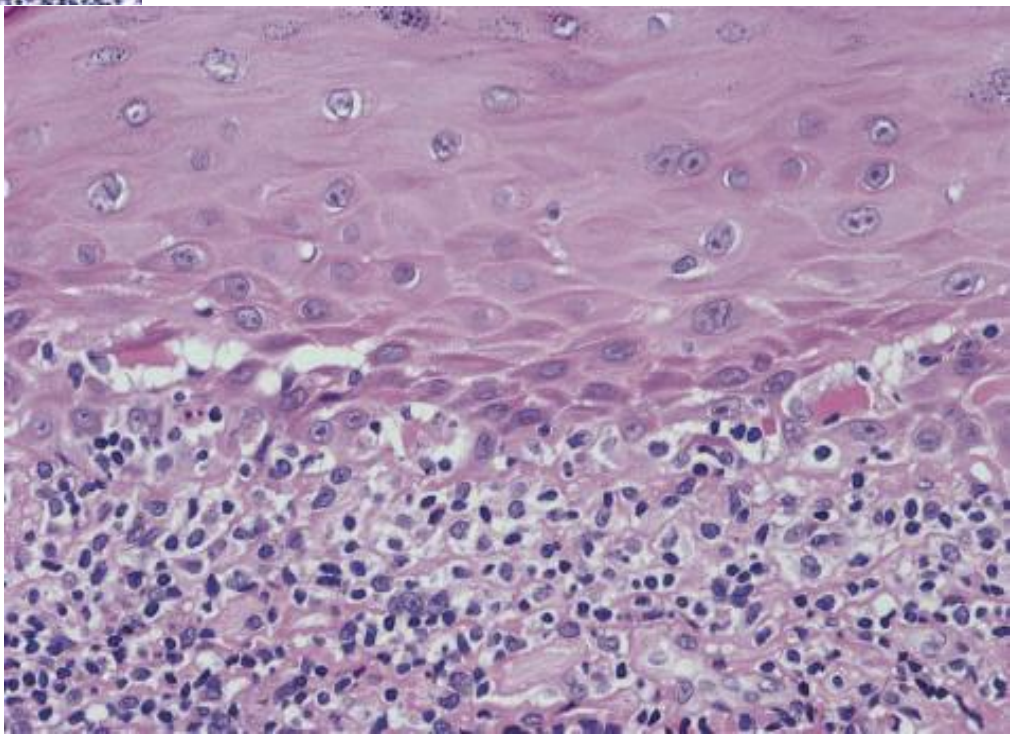


Oral lichen planus, plaque form.

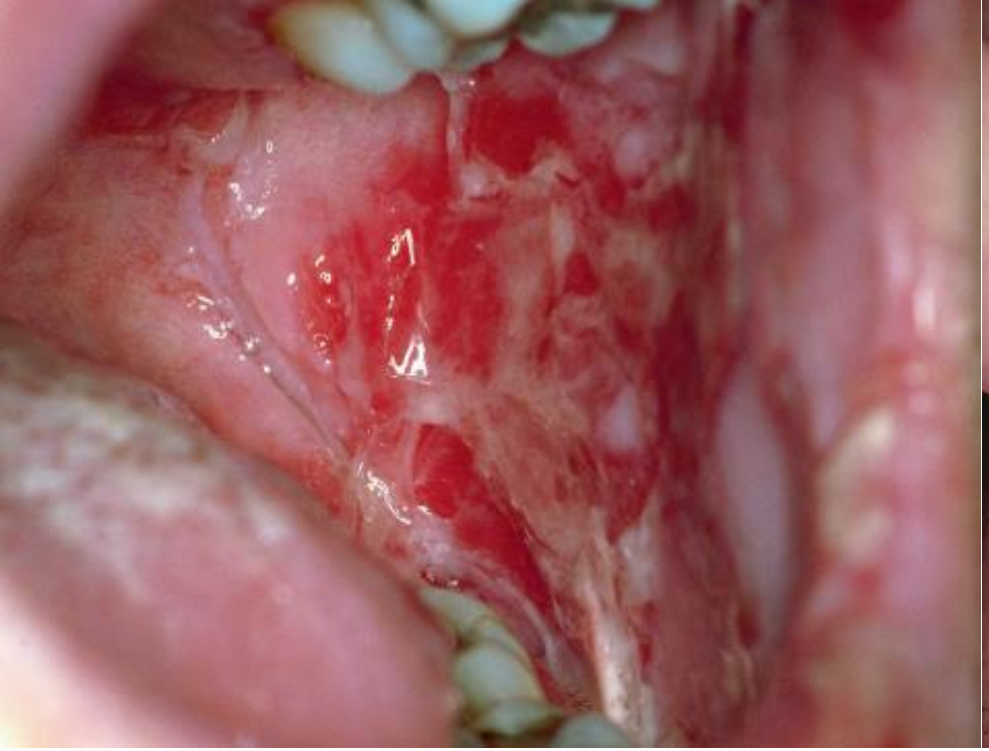
Erosive lichen planus of the lip.

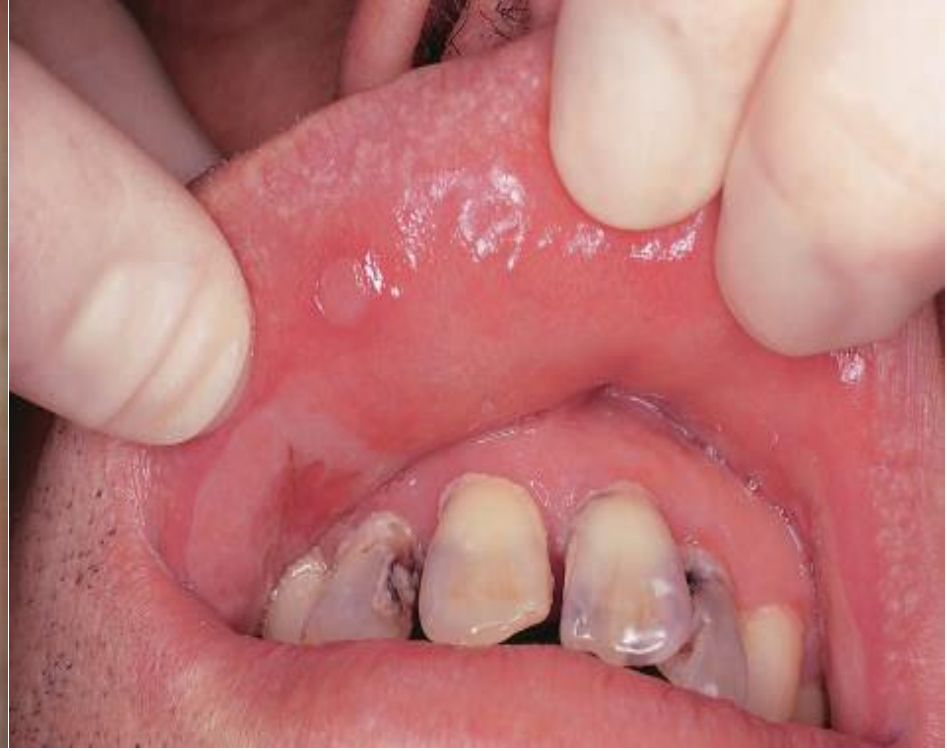






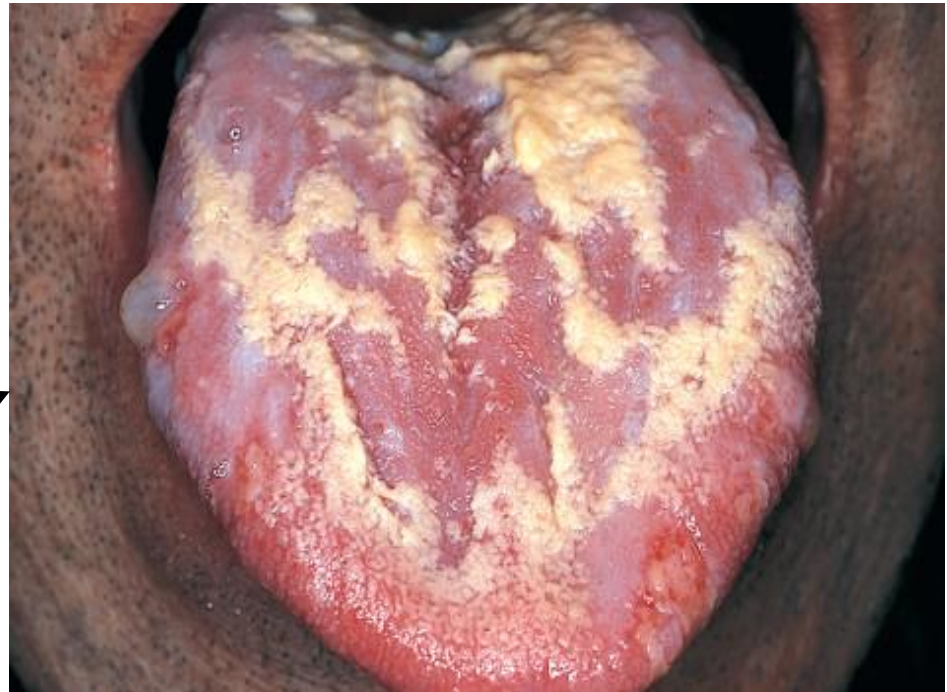
Lichen planus biopsy specimen showing hyperkeratosis, interface lymphocytic infiltrate, and basilar vacuolization with apoptosis.

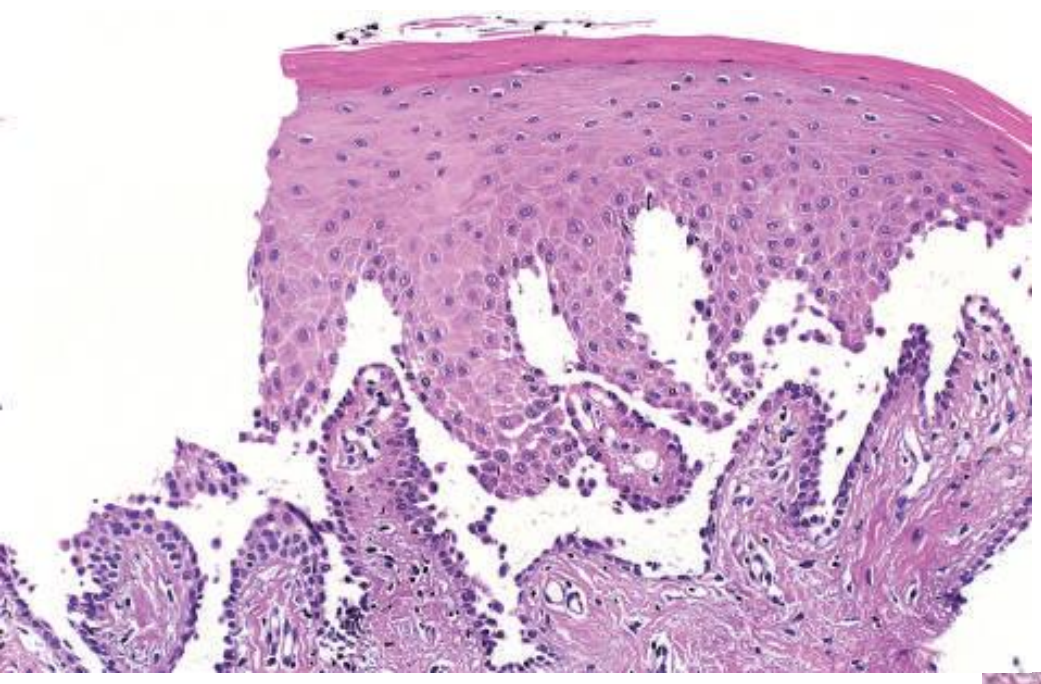


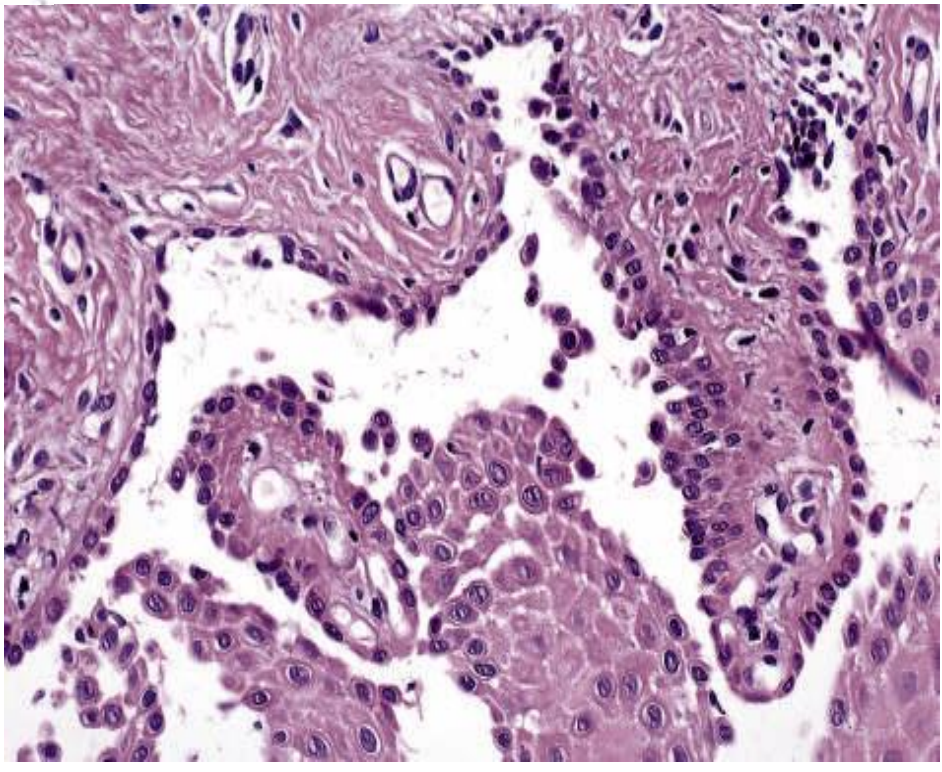


Oral pemphigus vulgaris, buccal mucosa.
Surface slough with ulceration and bleeding.

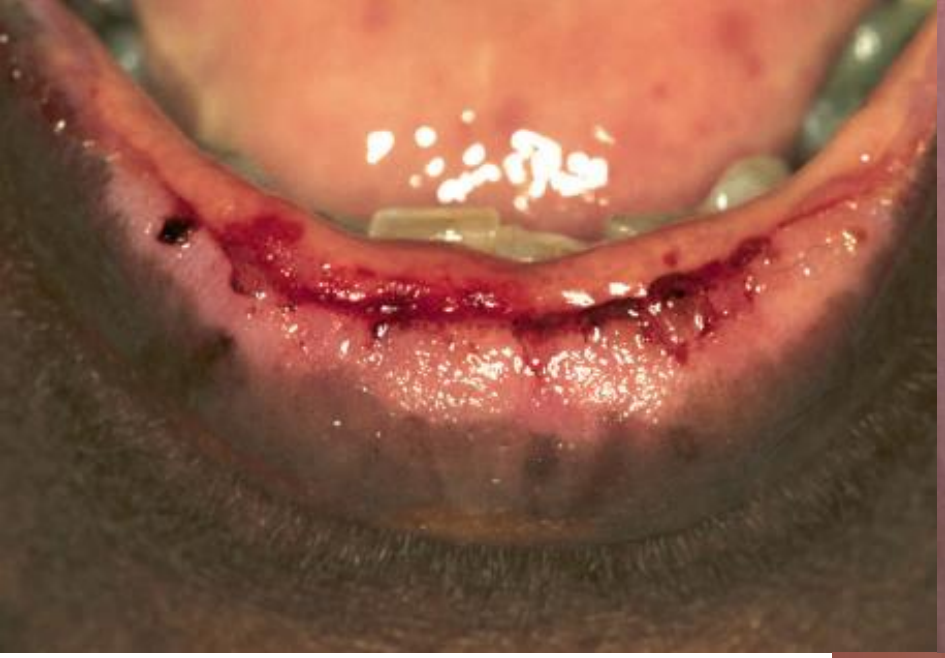
Pemphigus bulla and ulcers

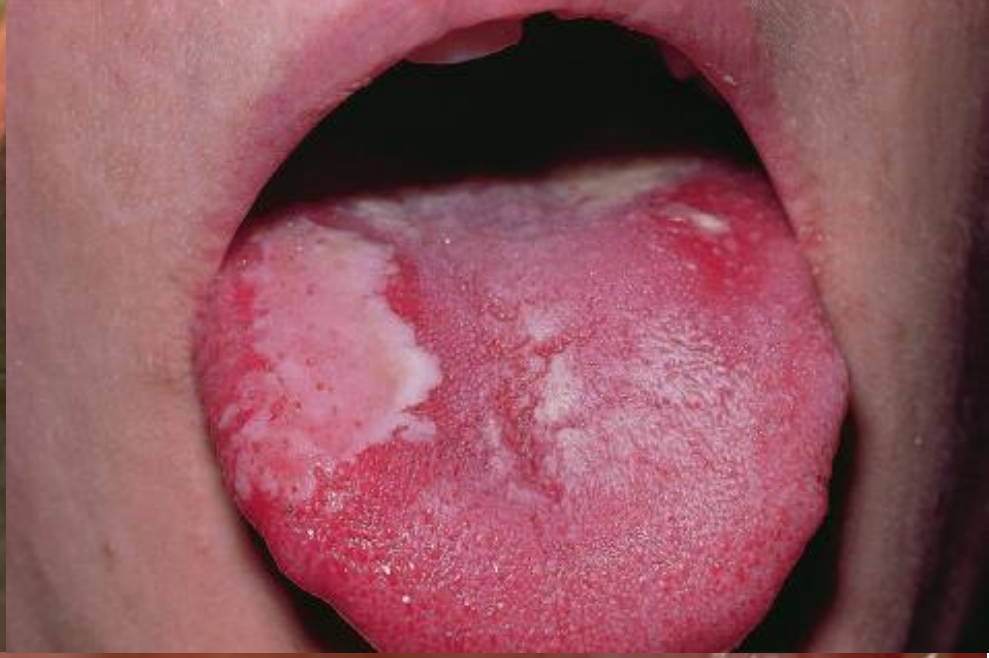






Oral pemphigus vulgaris showing intraepithelial separation and Tzanck cells.





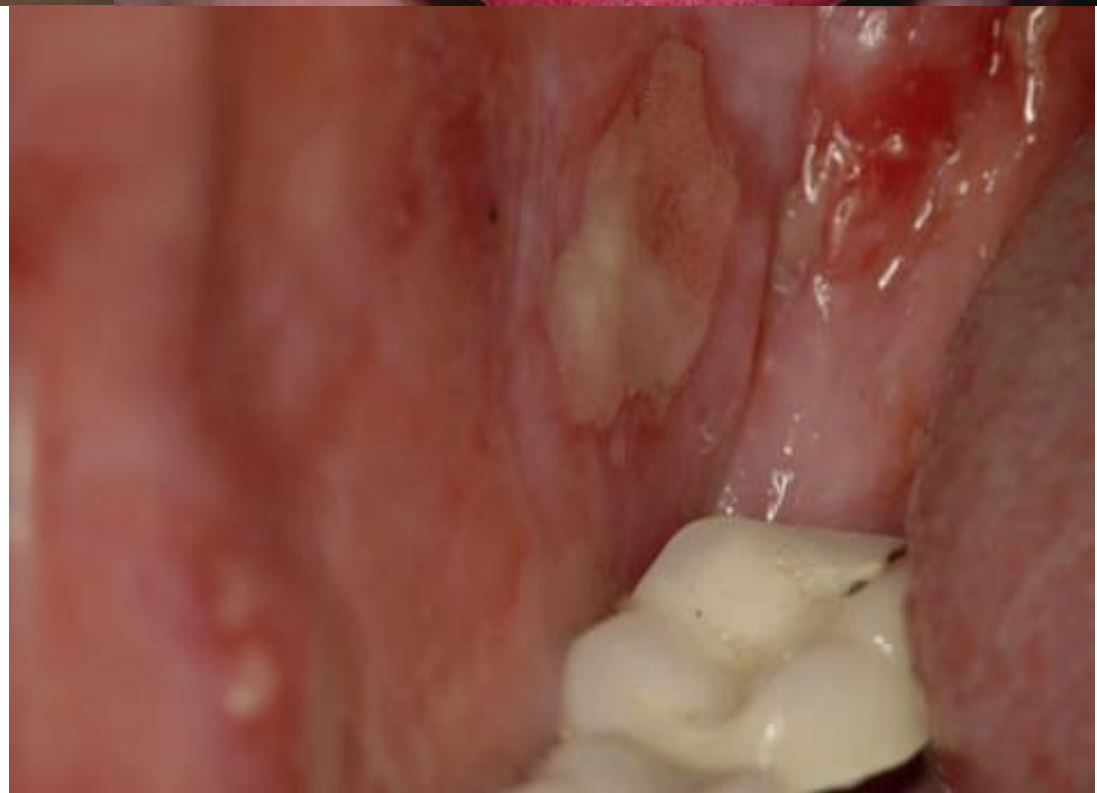
Erythema multiforme ulcers.

Etiology

Minor (less severe) form usually triggered by herpes simplex virus

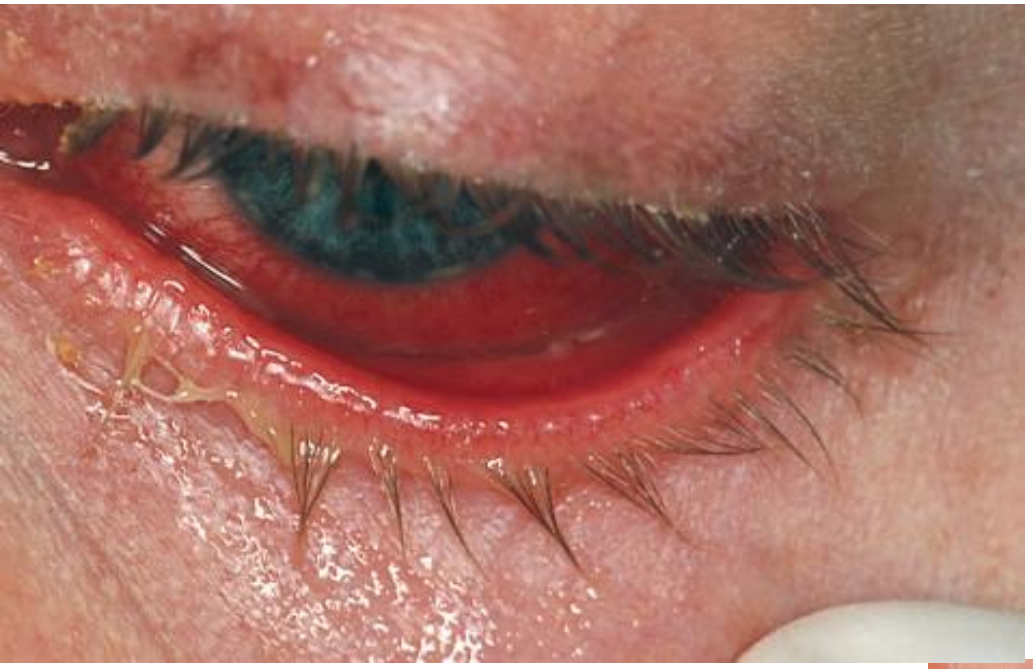
Major form (Stevens-Johnson syndrome) often triggered by drugs

Hypersensitivity reaction to infectious agents, drugs, or idiopathic



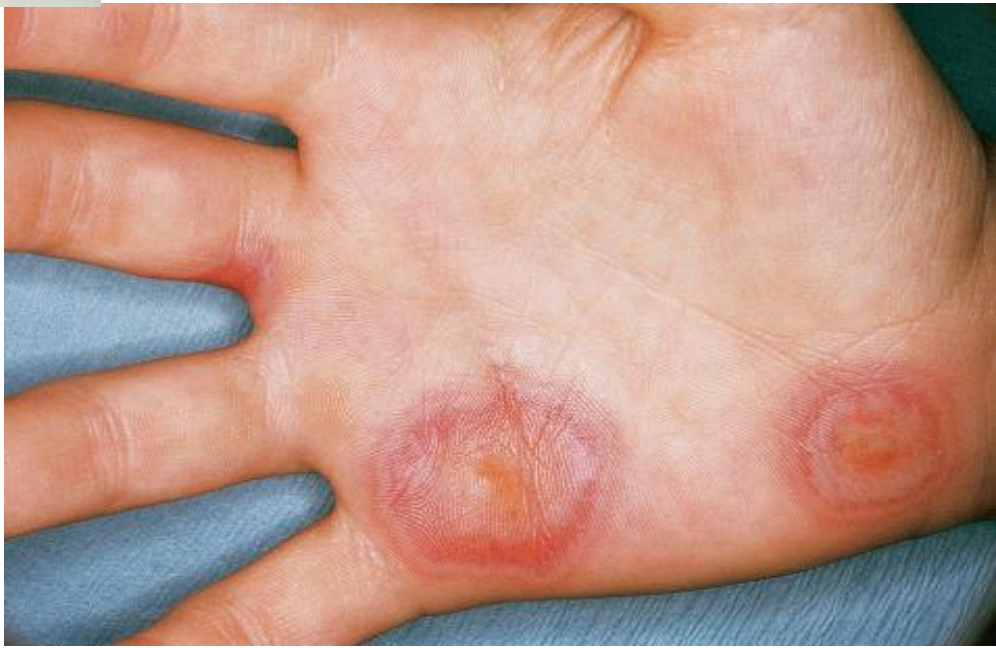
Clinical Features

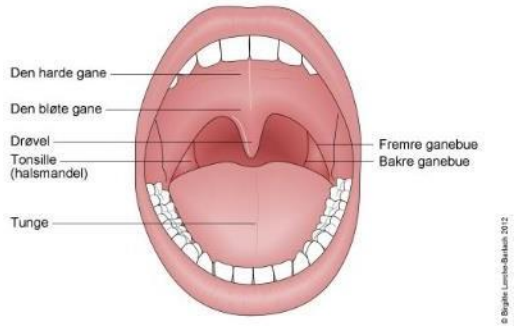
Multiple oral ulcers and/or target skin lesions. Self-limiting, but may recur



Ocular lesions in patient with erythema multiforme

Erythema multiforme cutaneous target lesions.

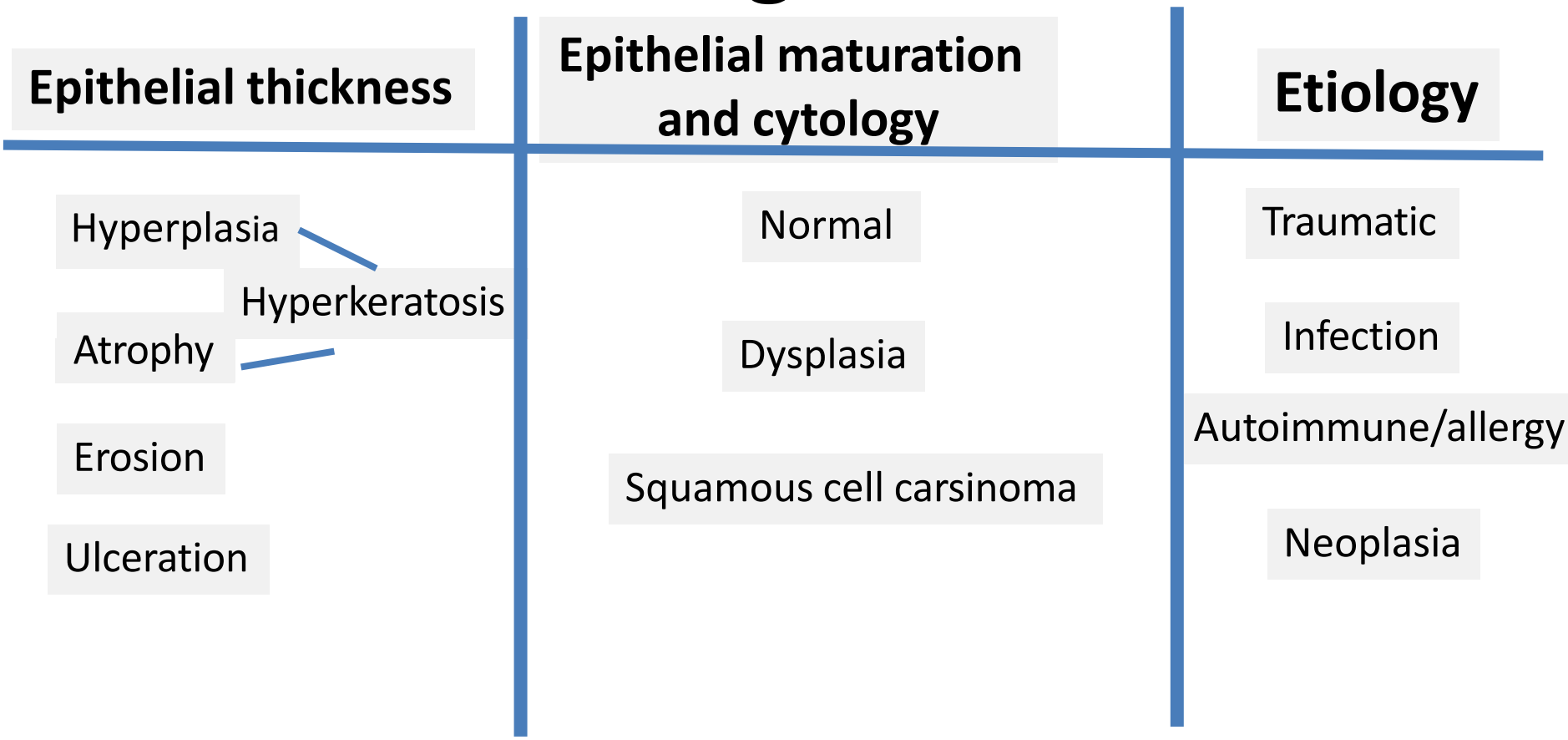




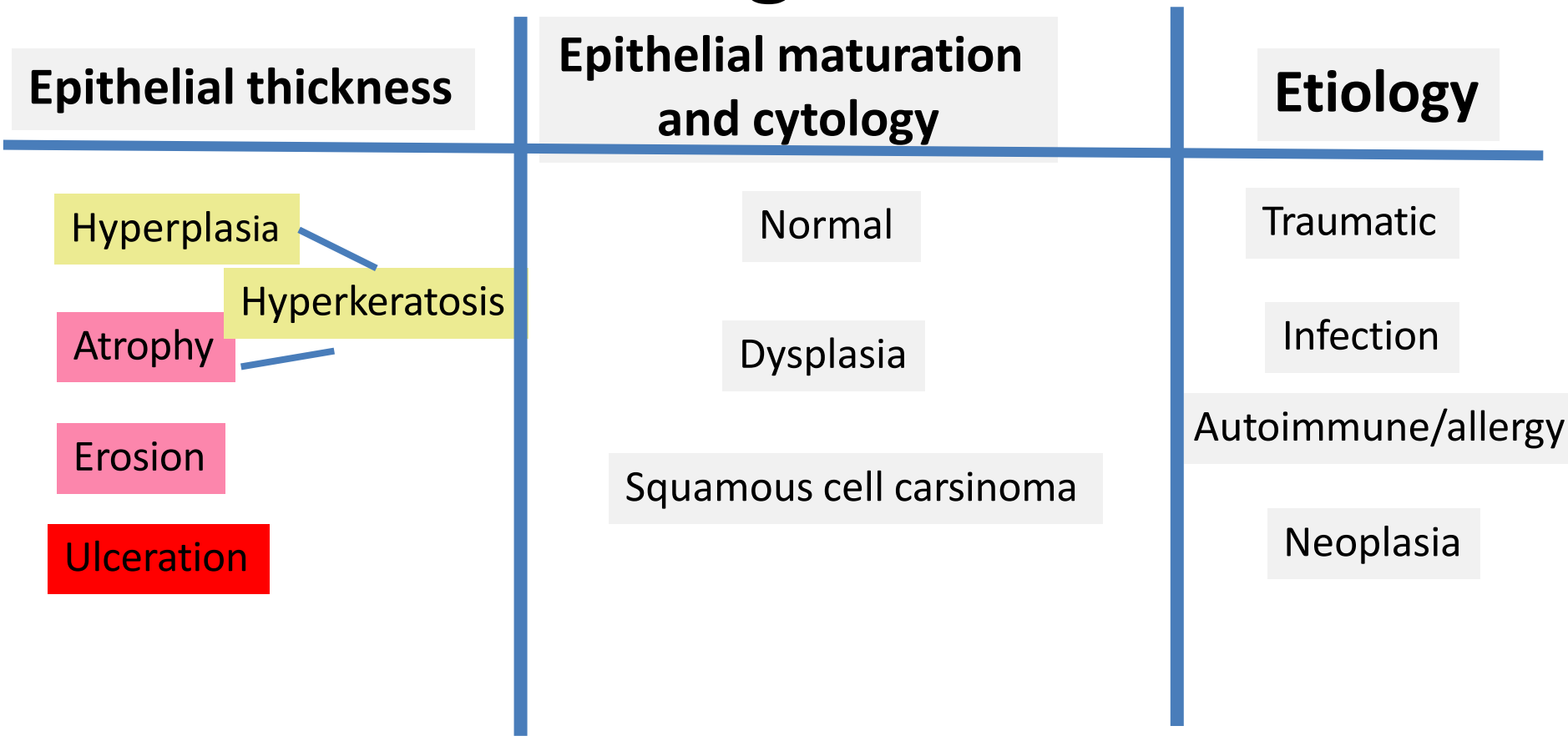
Lesions in the oral mucosa

Epithelial ⟷ Connective tissue

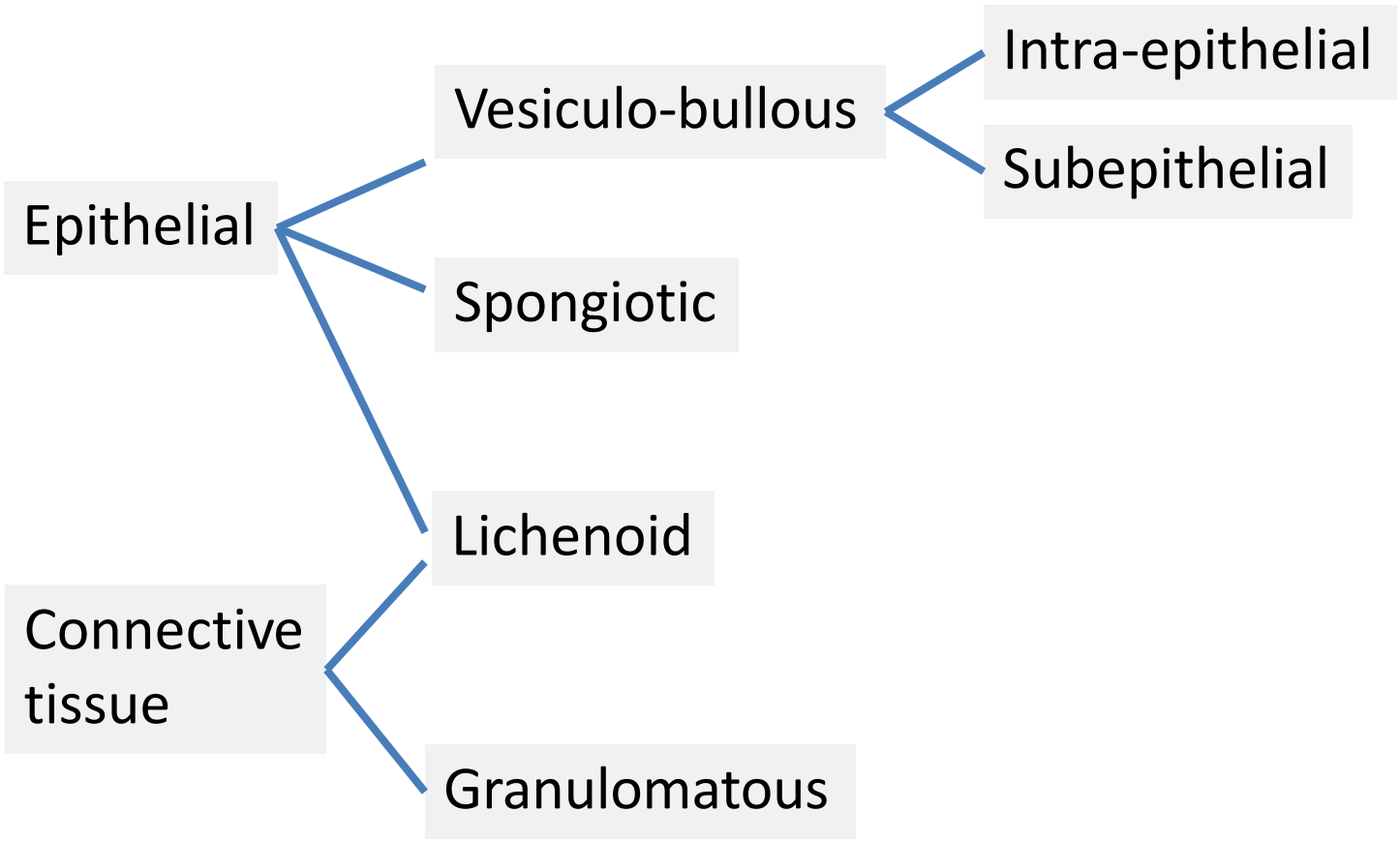
Epithelial changes



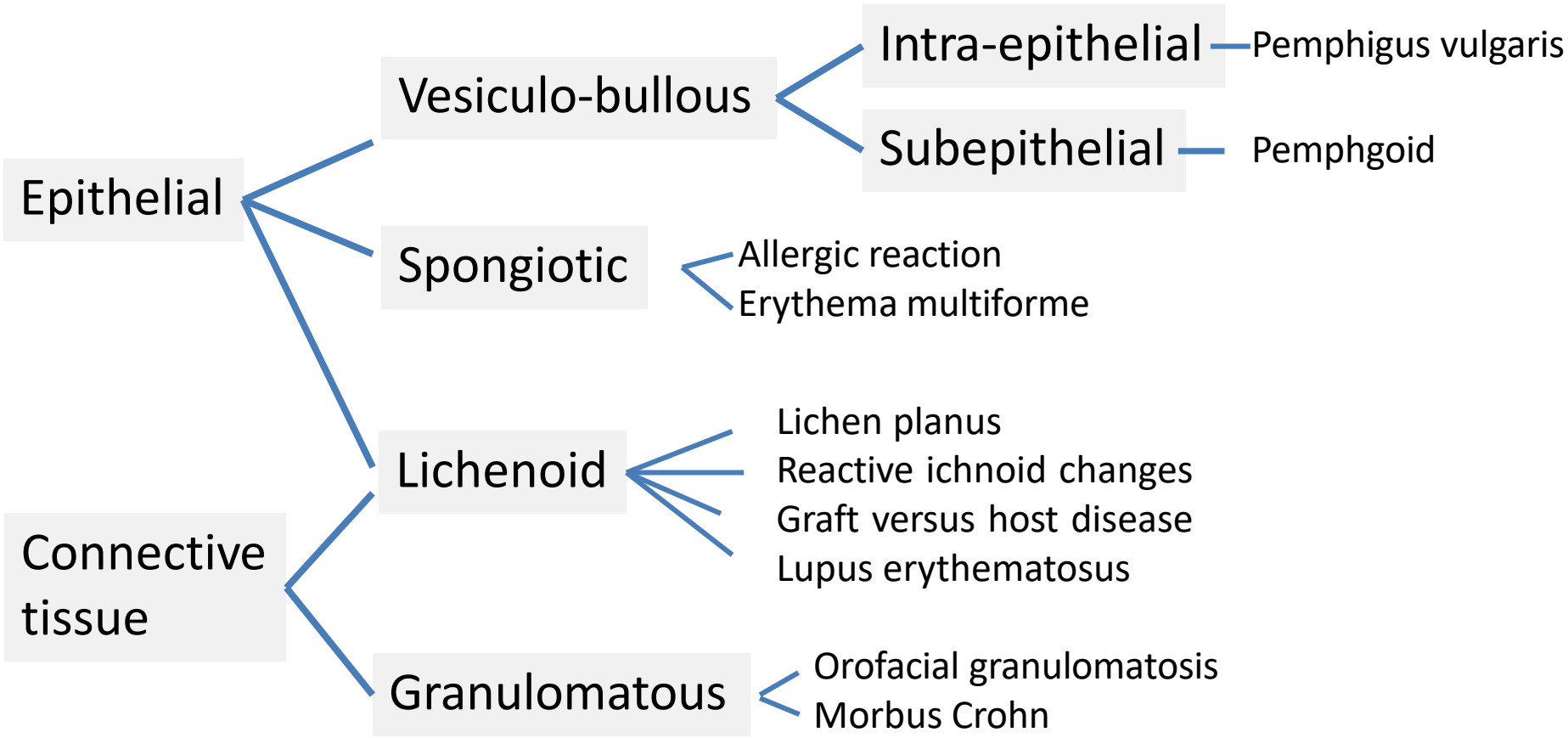
Epithelial changes



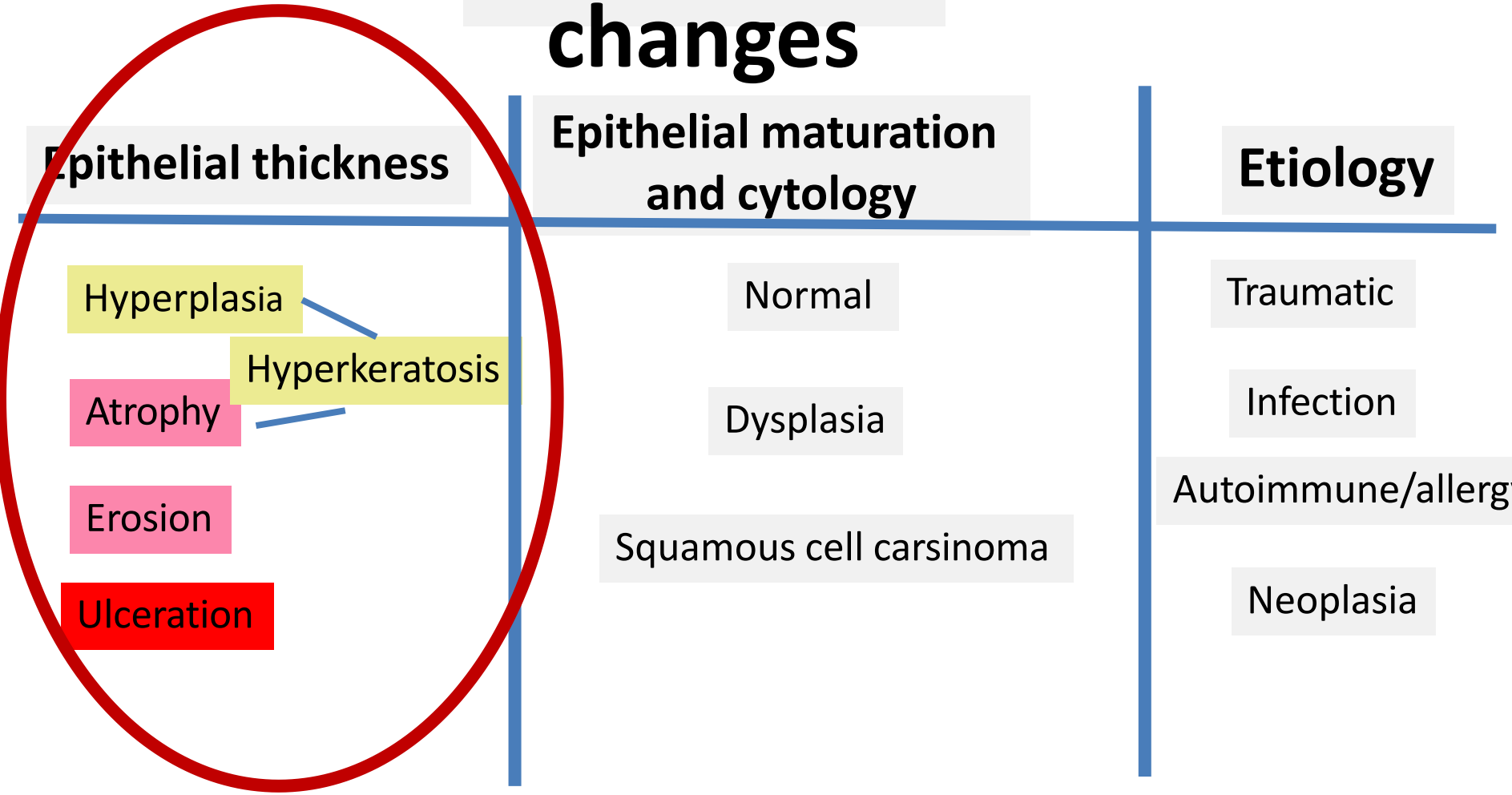
Inflammatory diseases in oral mucosa



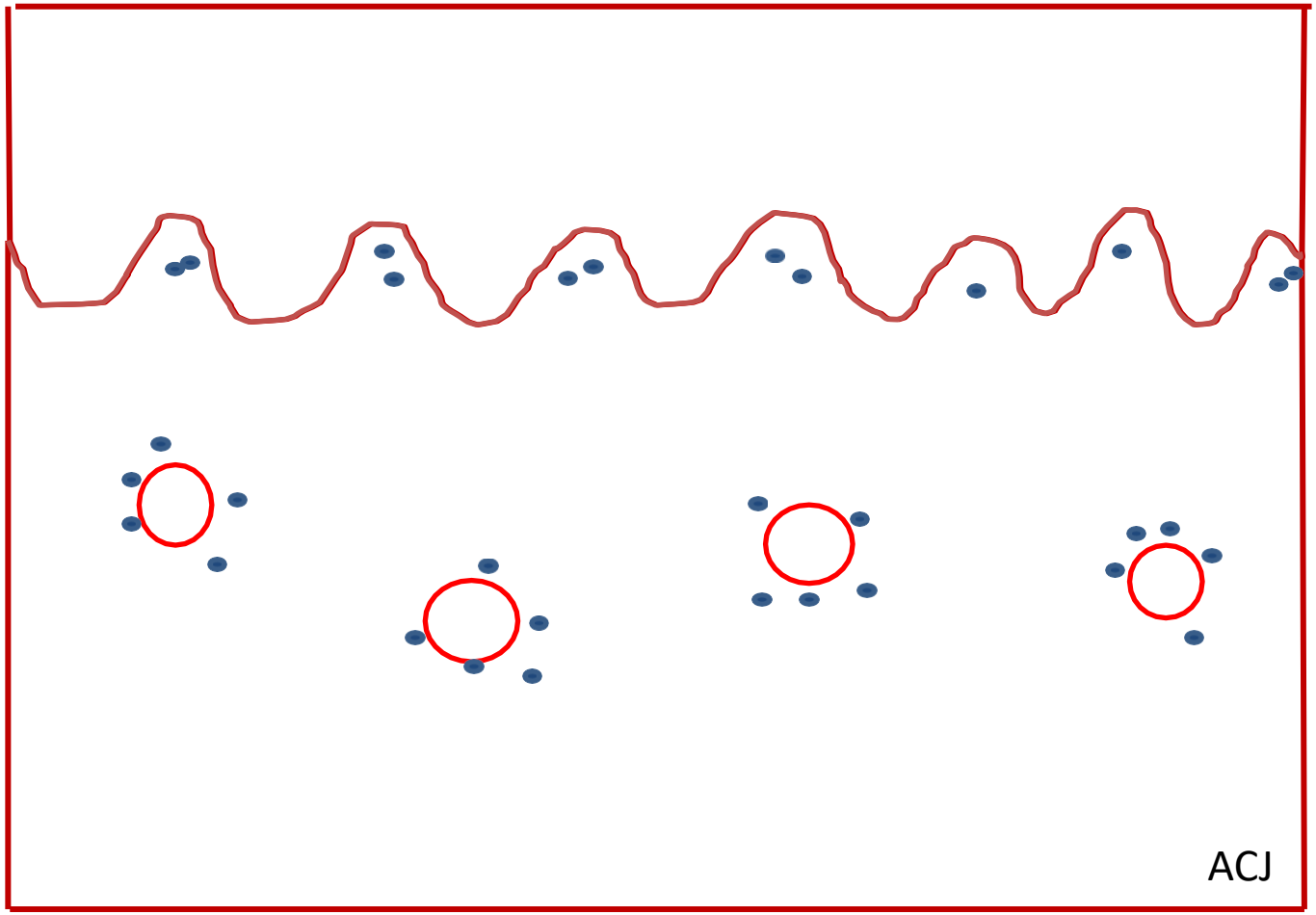
Inflammatory diseases in oral mucosa

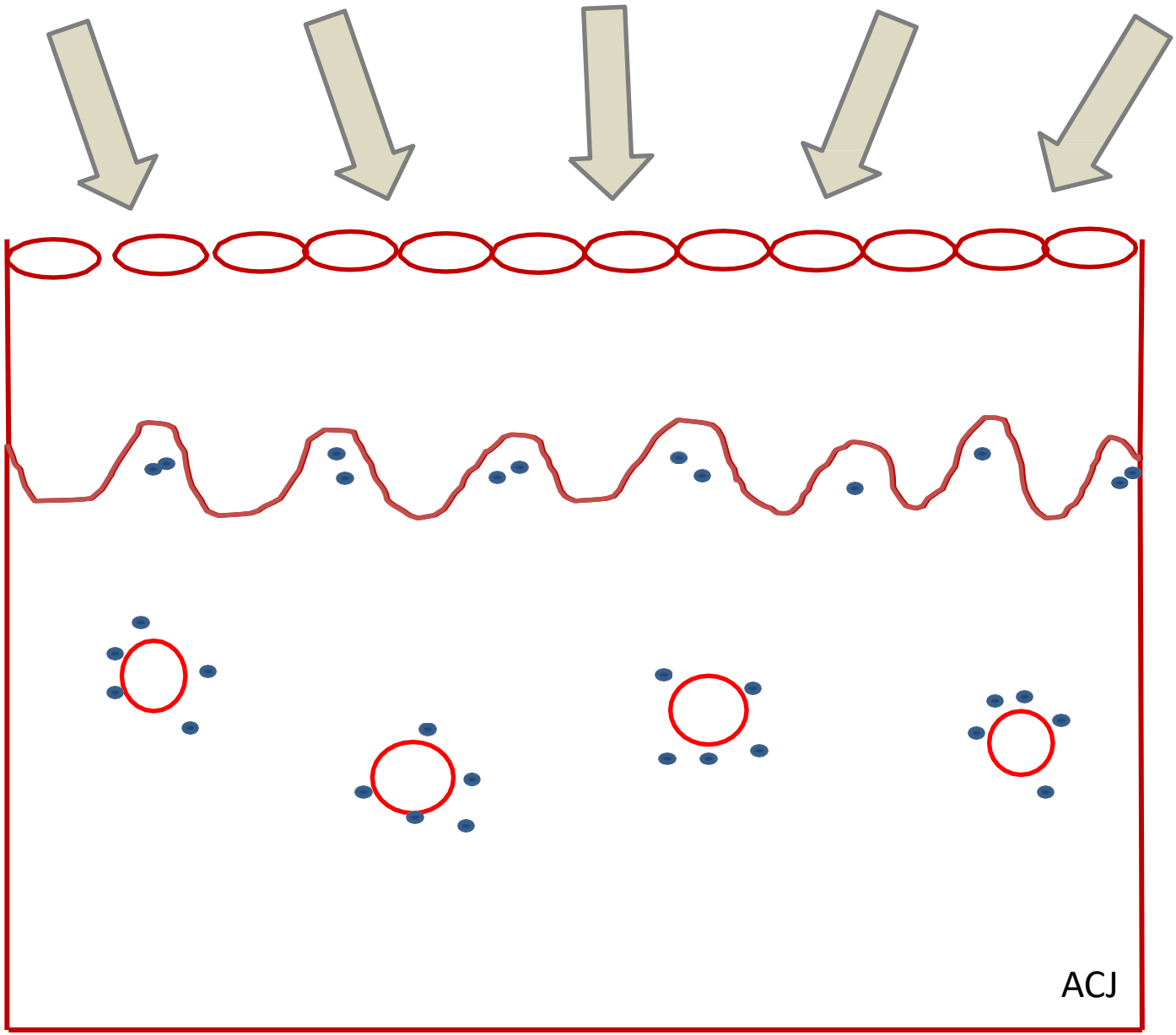


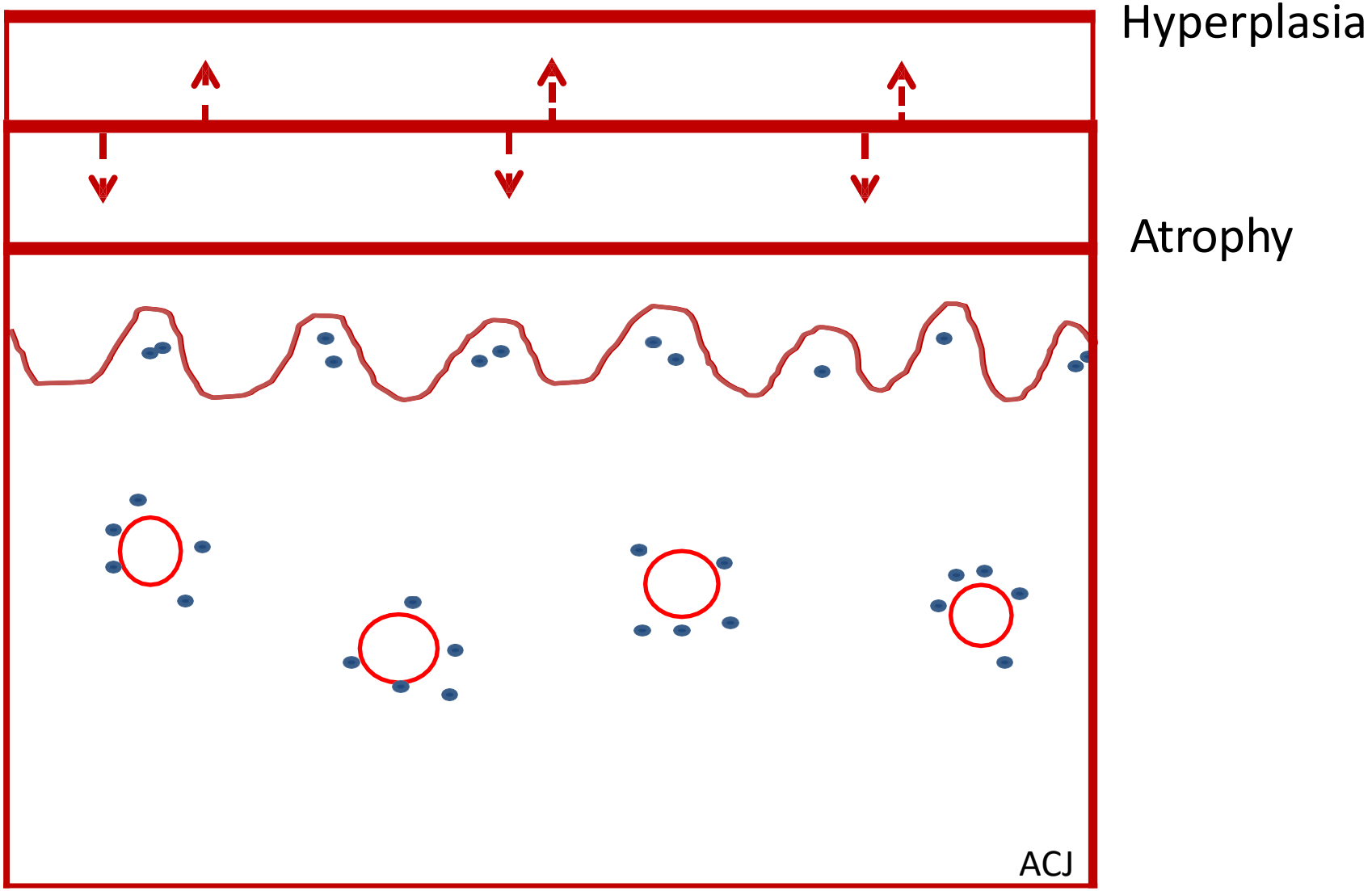
Epithelial changes



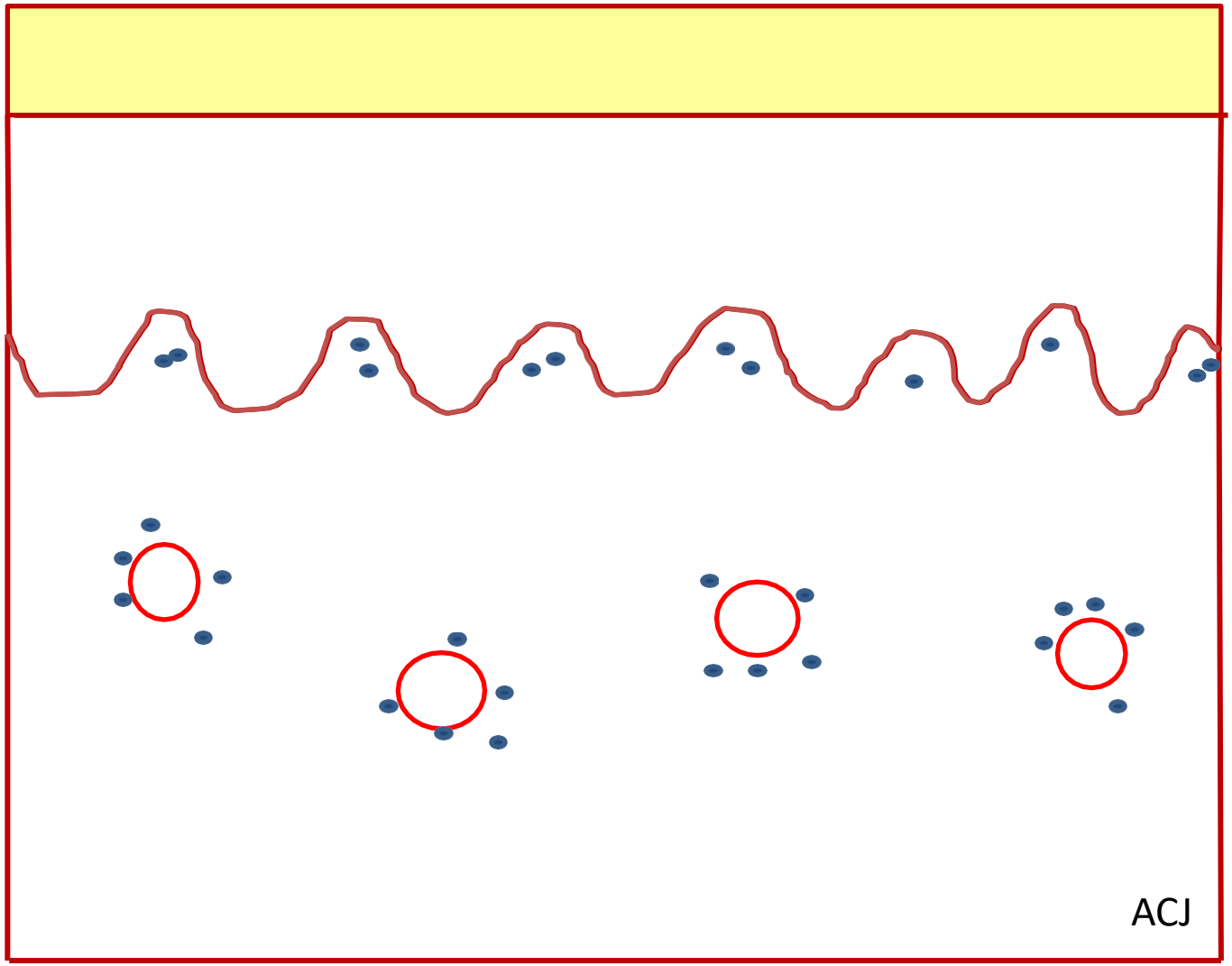
Normal oral mucosa

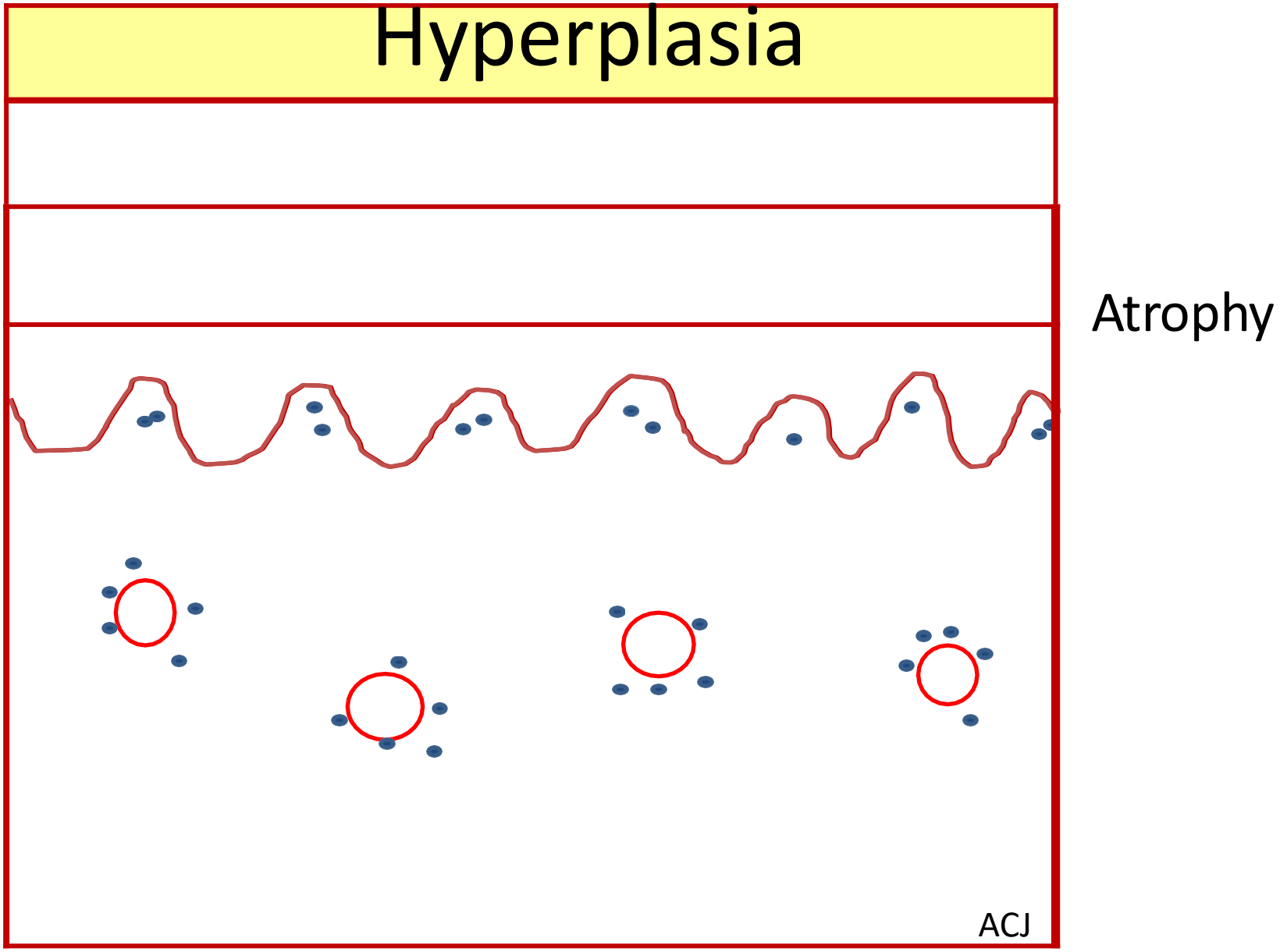


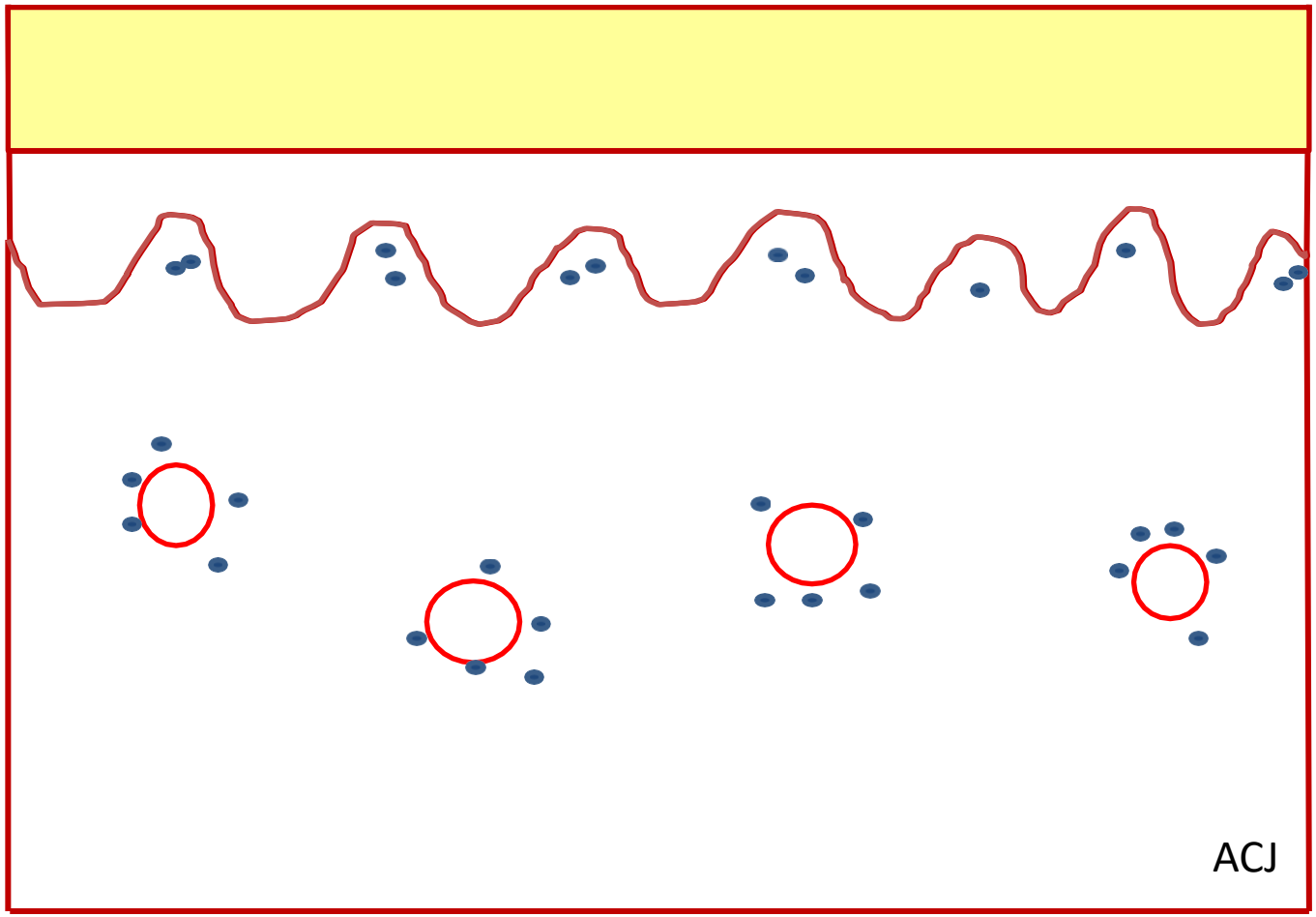

ACJ


Hyperplasia
Atrophy
ACJ

Hyperkeratosis




Hyperplasia
Atrophy
ACJ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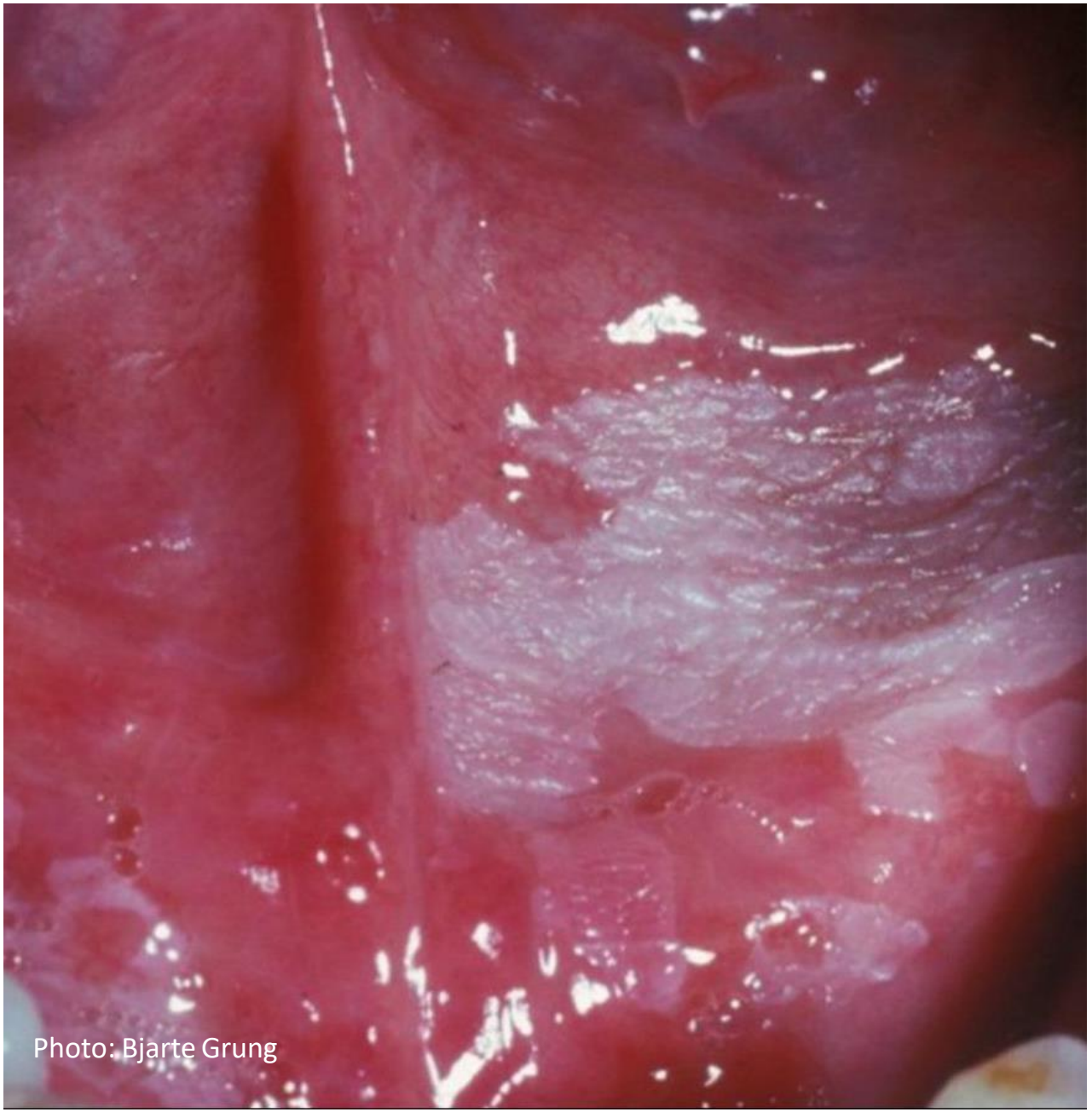


Photo: Bjarte Grung

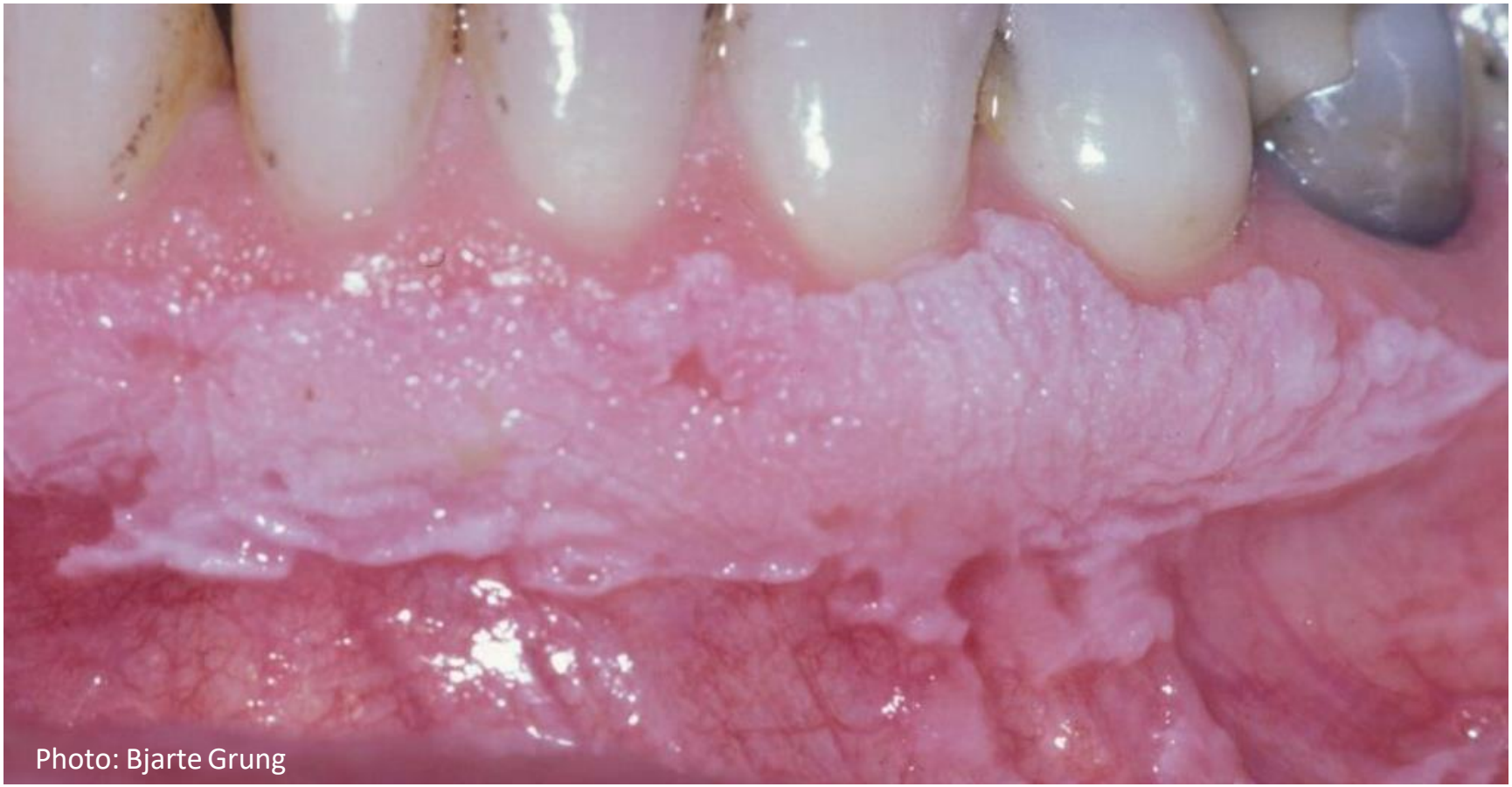
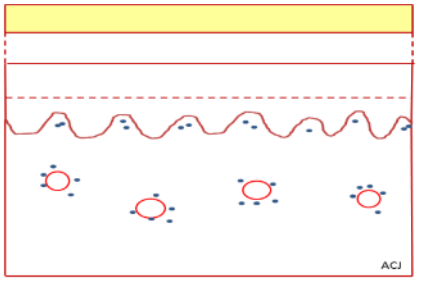
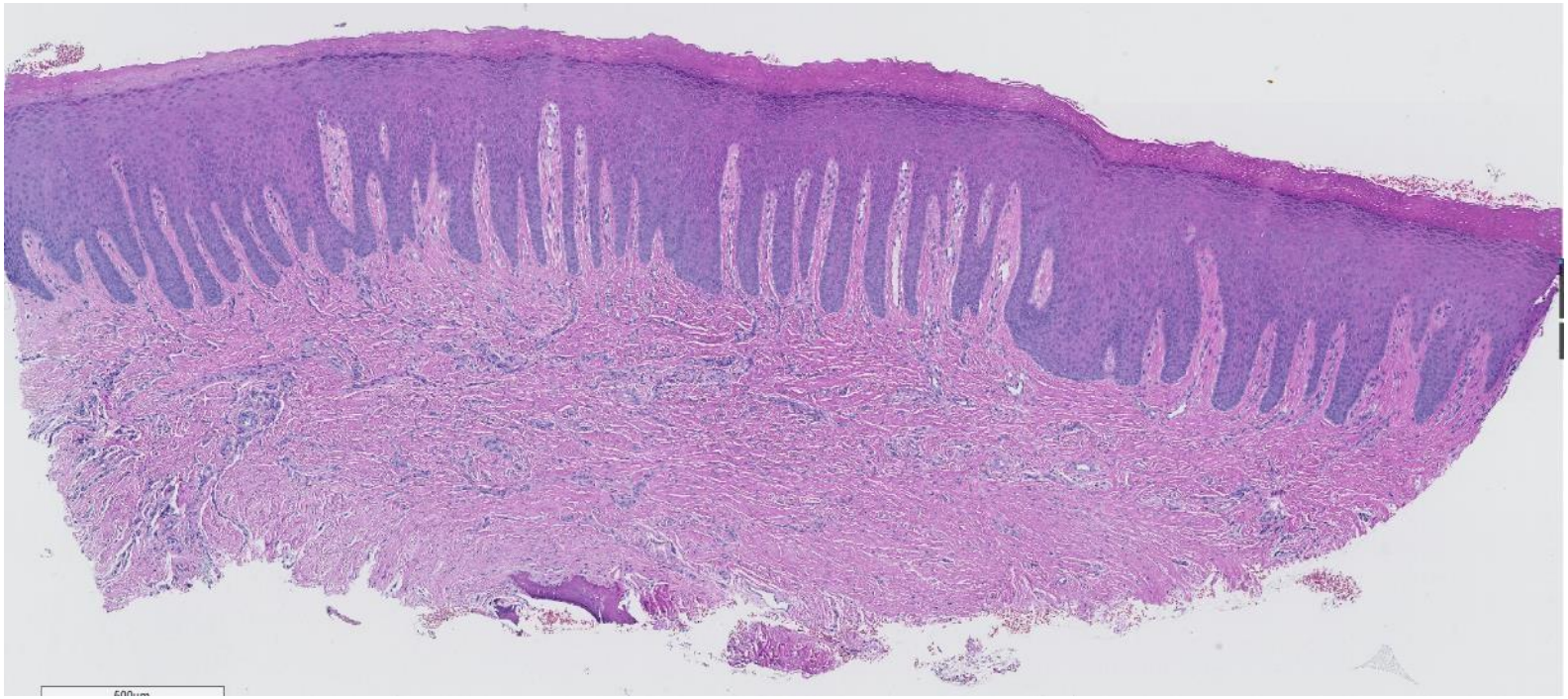


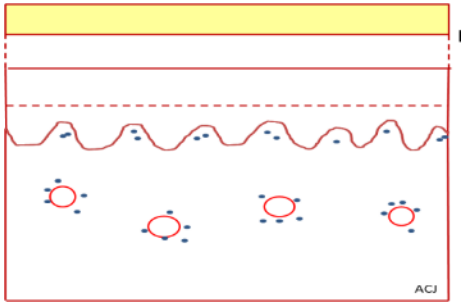
Photo: Bjarte Grung

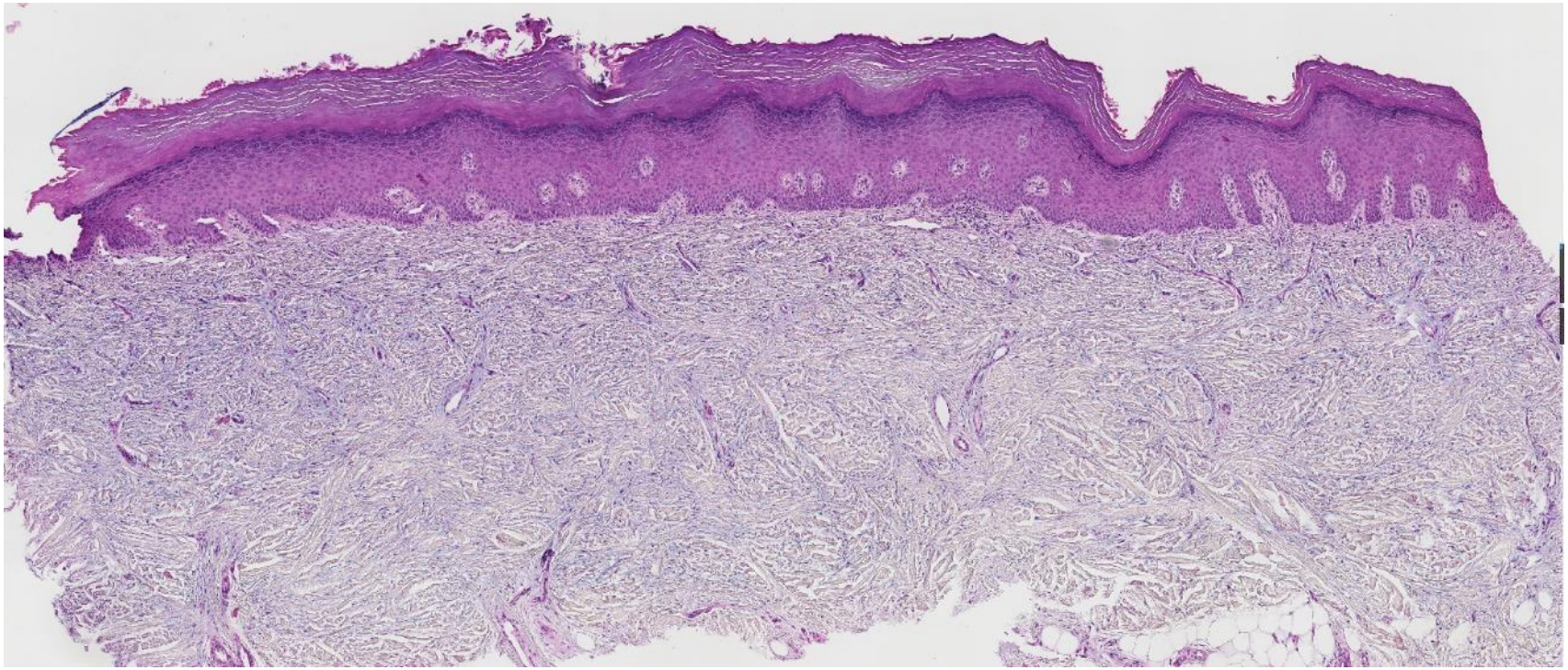
Male, 72 years old. Whitish lesions on edentulous upper jaw mucosa - alveolar ridge. No symptoms. Smokes 15 cigarets daily





Female 49 years old. Biopsy from hard palate, right side.
No symptoms.





Female, 69 years old. Whitish, radiating lesion with scattered redish areas, from buccal mucosa, left side. No ulceration. TD: Lichen planus.

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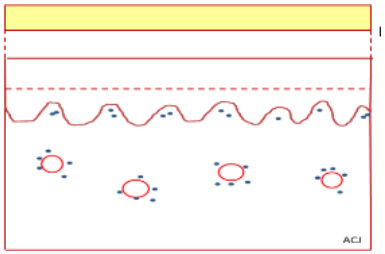
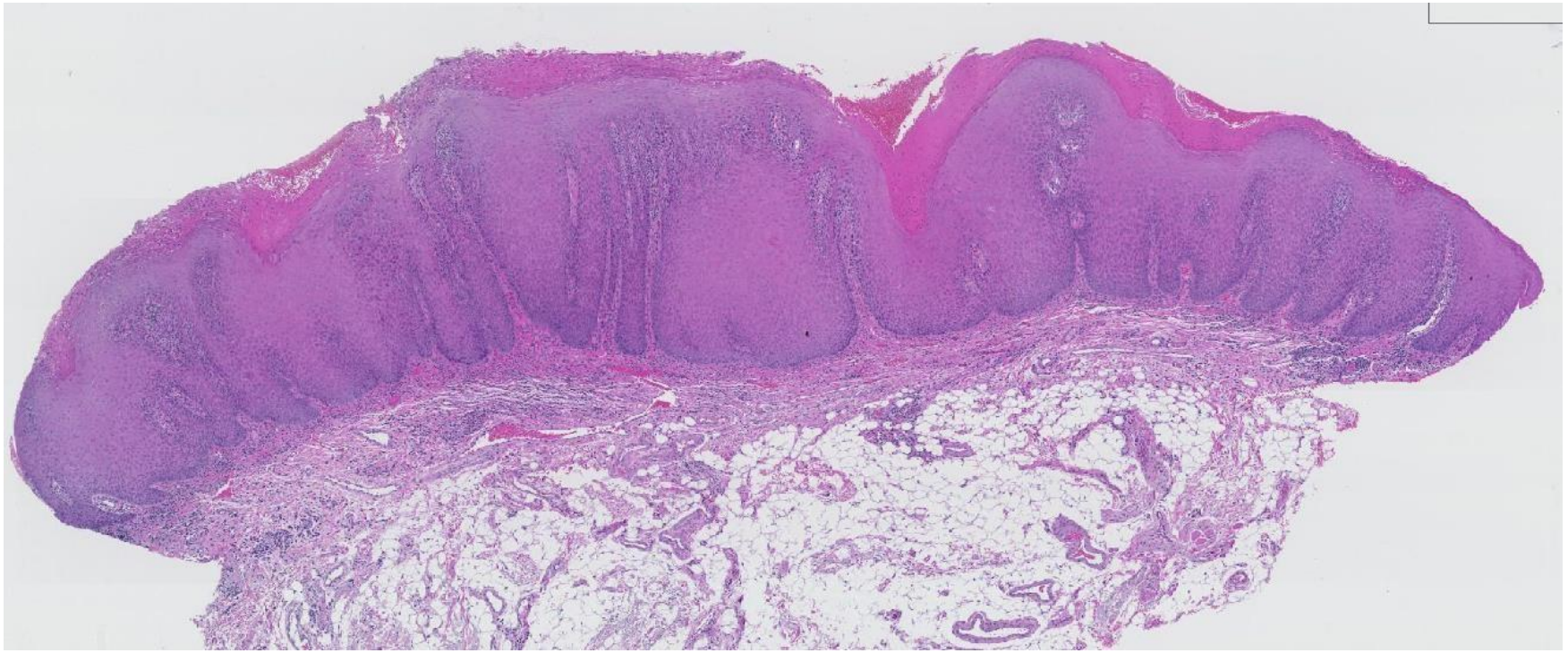
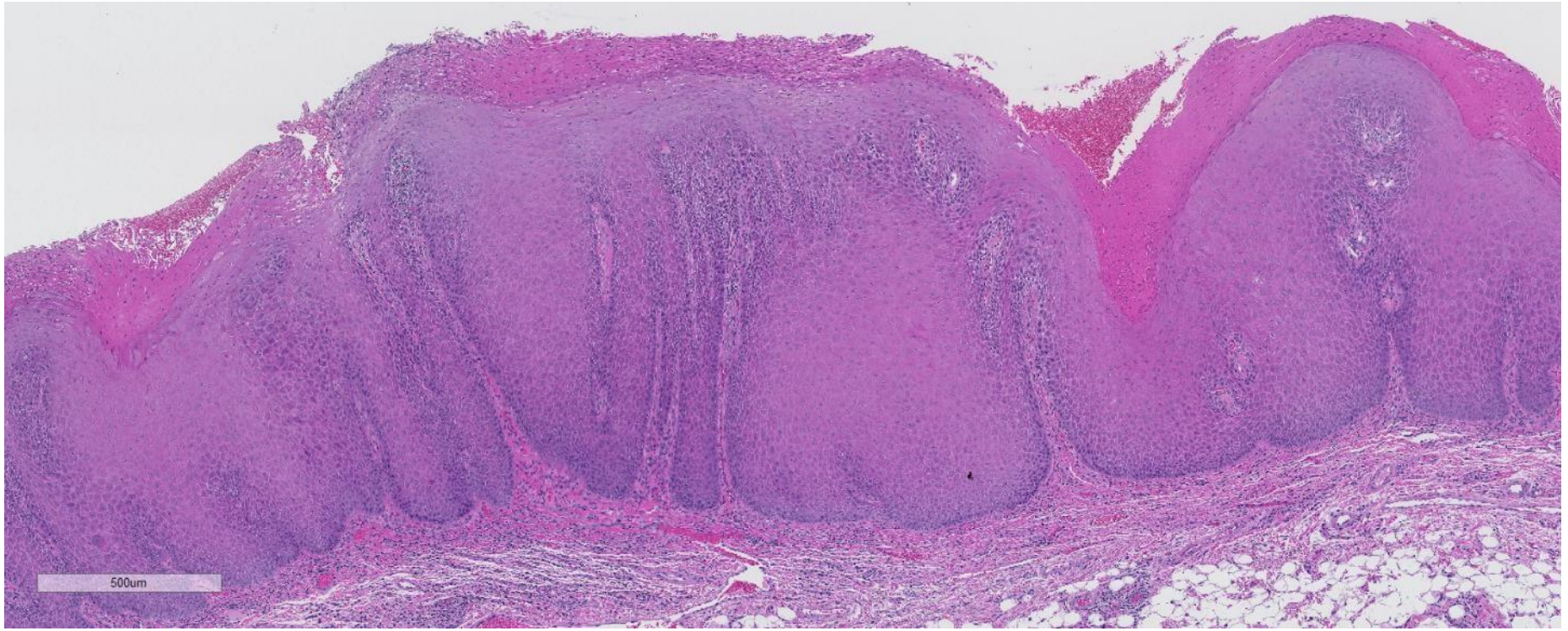
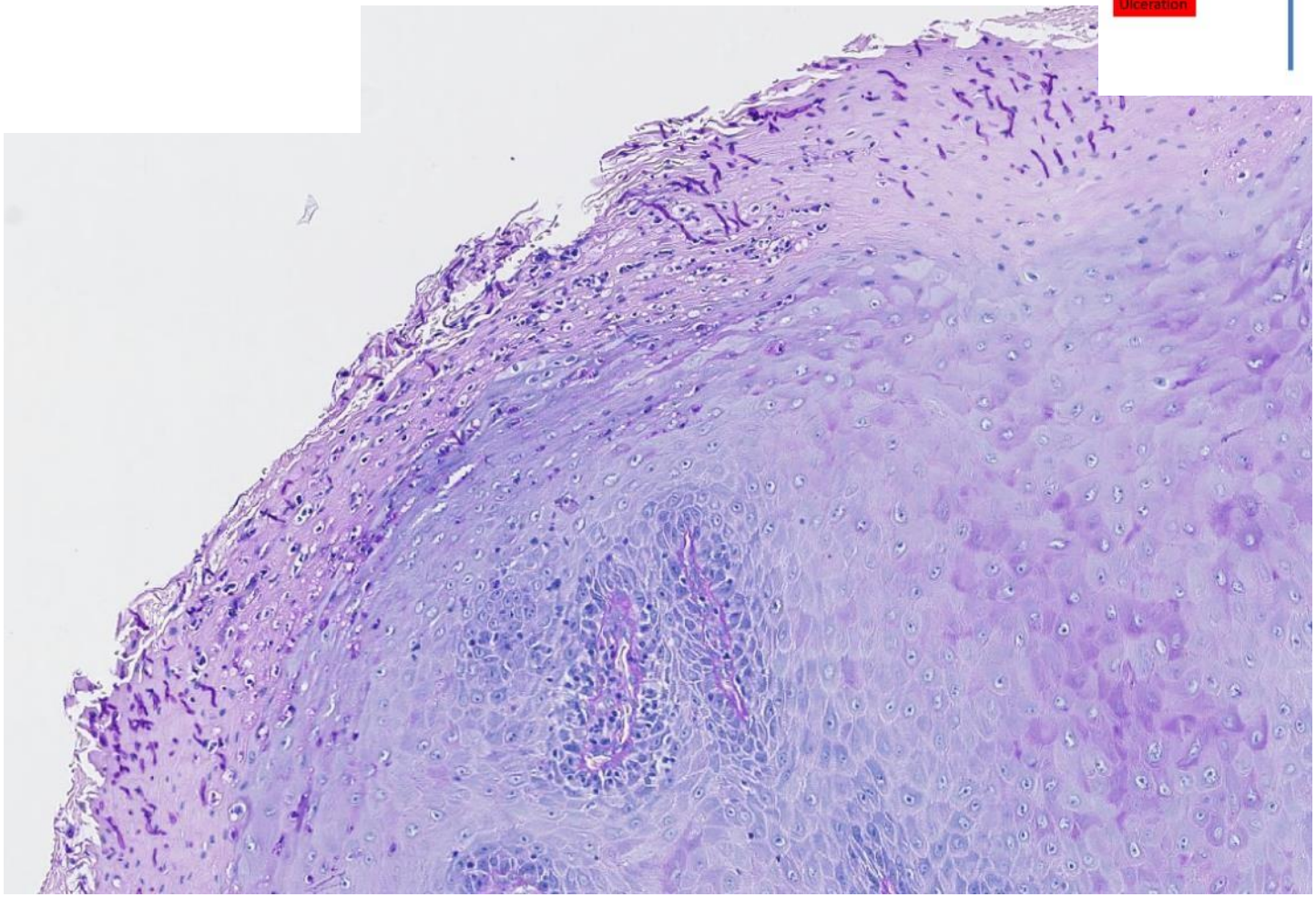




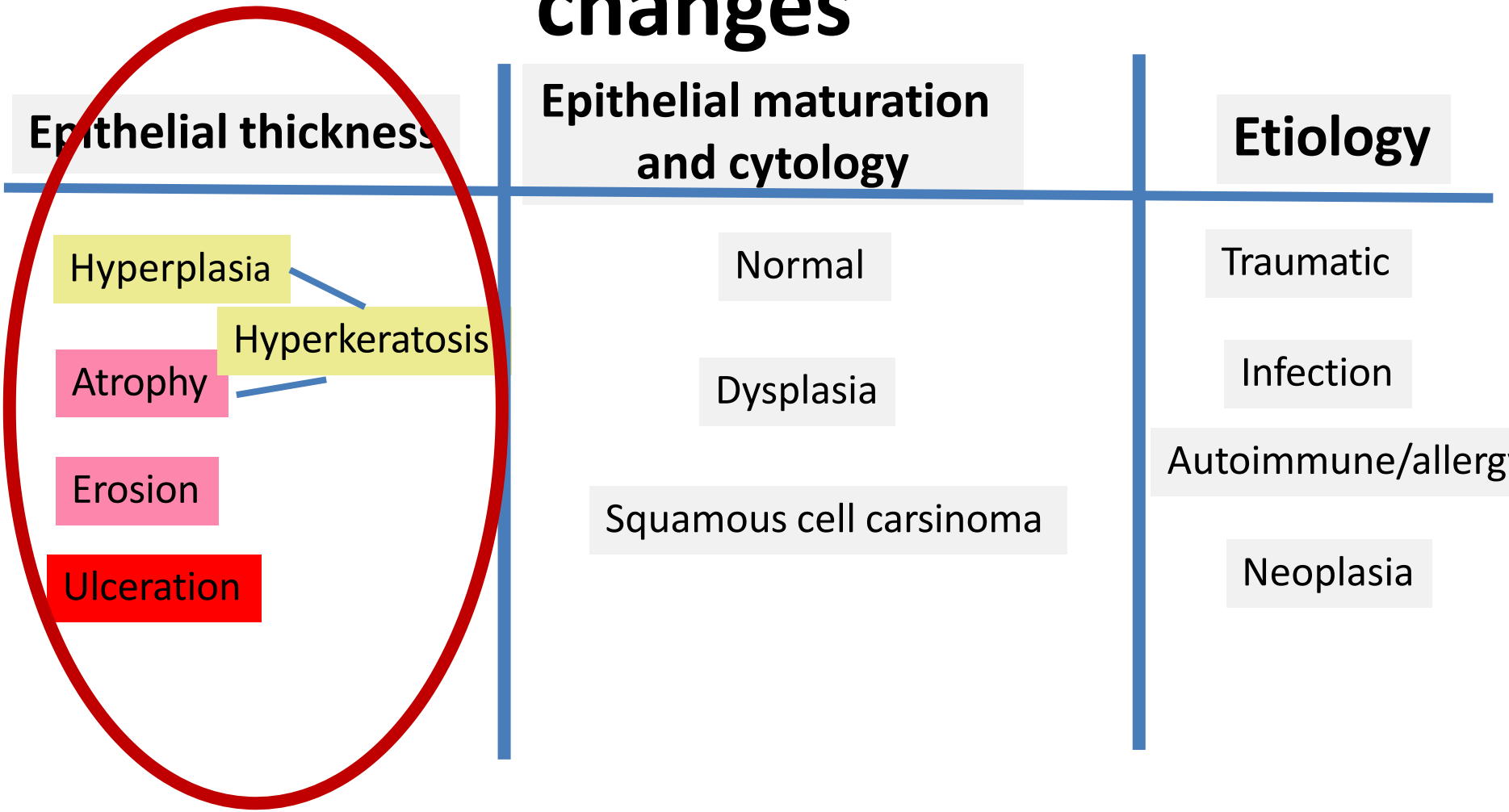

500um

Epithelial changes

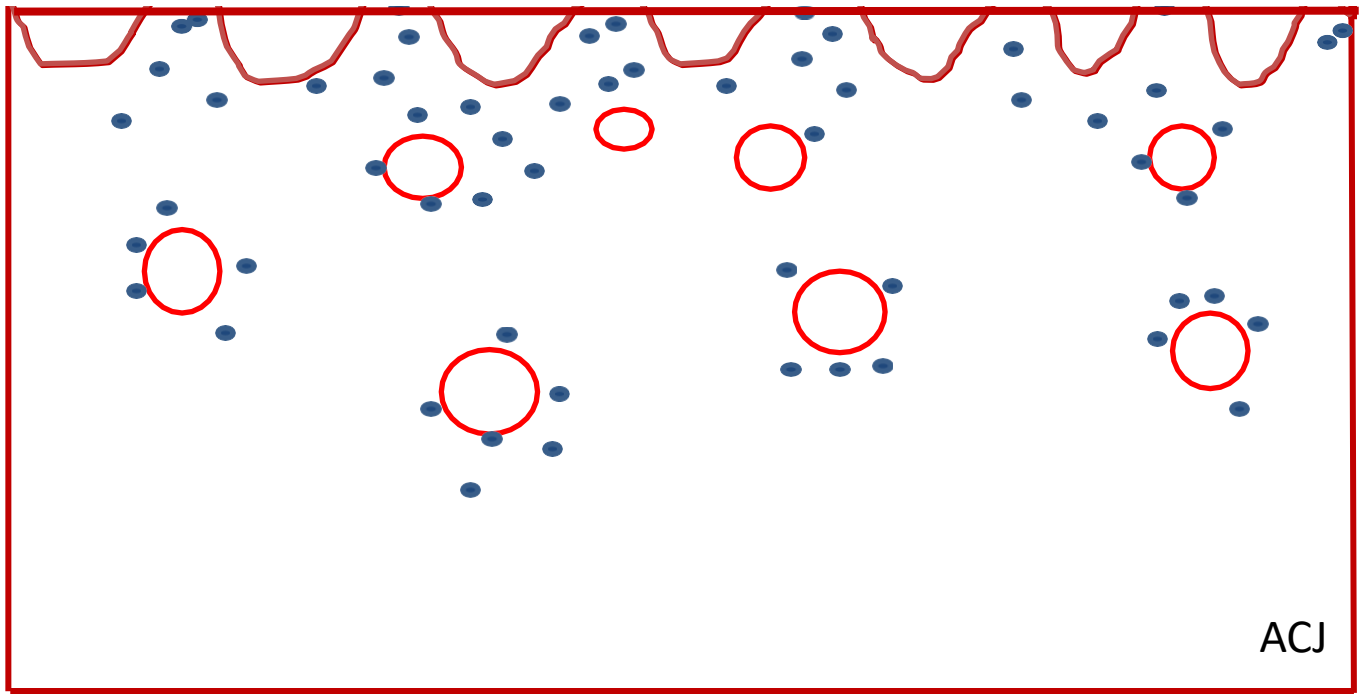
Epithelium thickness	Epithelium maturation and cytology	Etiology
Hyperplasia	Normal	Traumatic
Hyperkeratosis	Dysplasia	Infection
Atrophy	Squamous cell carsinoma	Autoimmune/allergy
Erosion		Neoplasia
Ulceration		



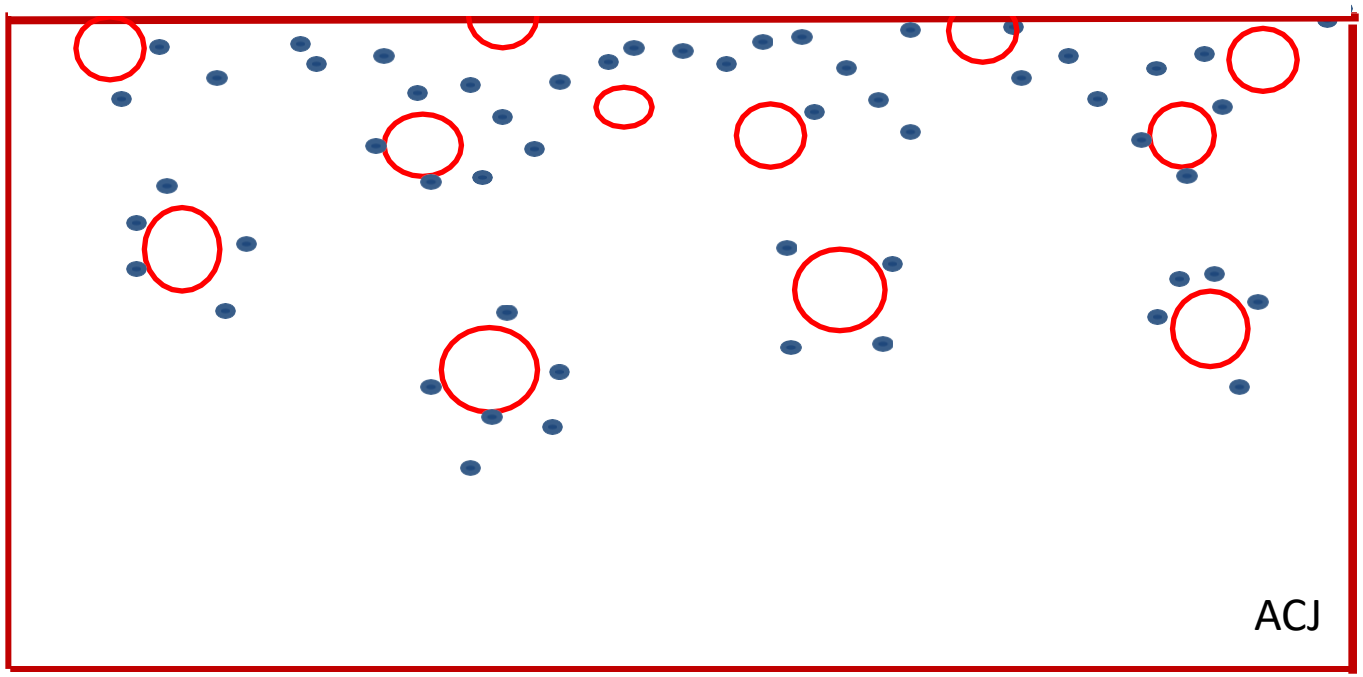
Epithelial changes



Erosion



Ulceration



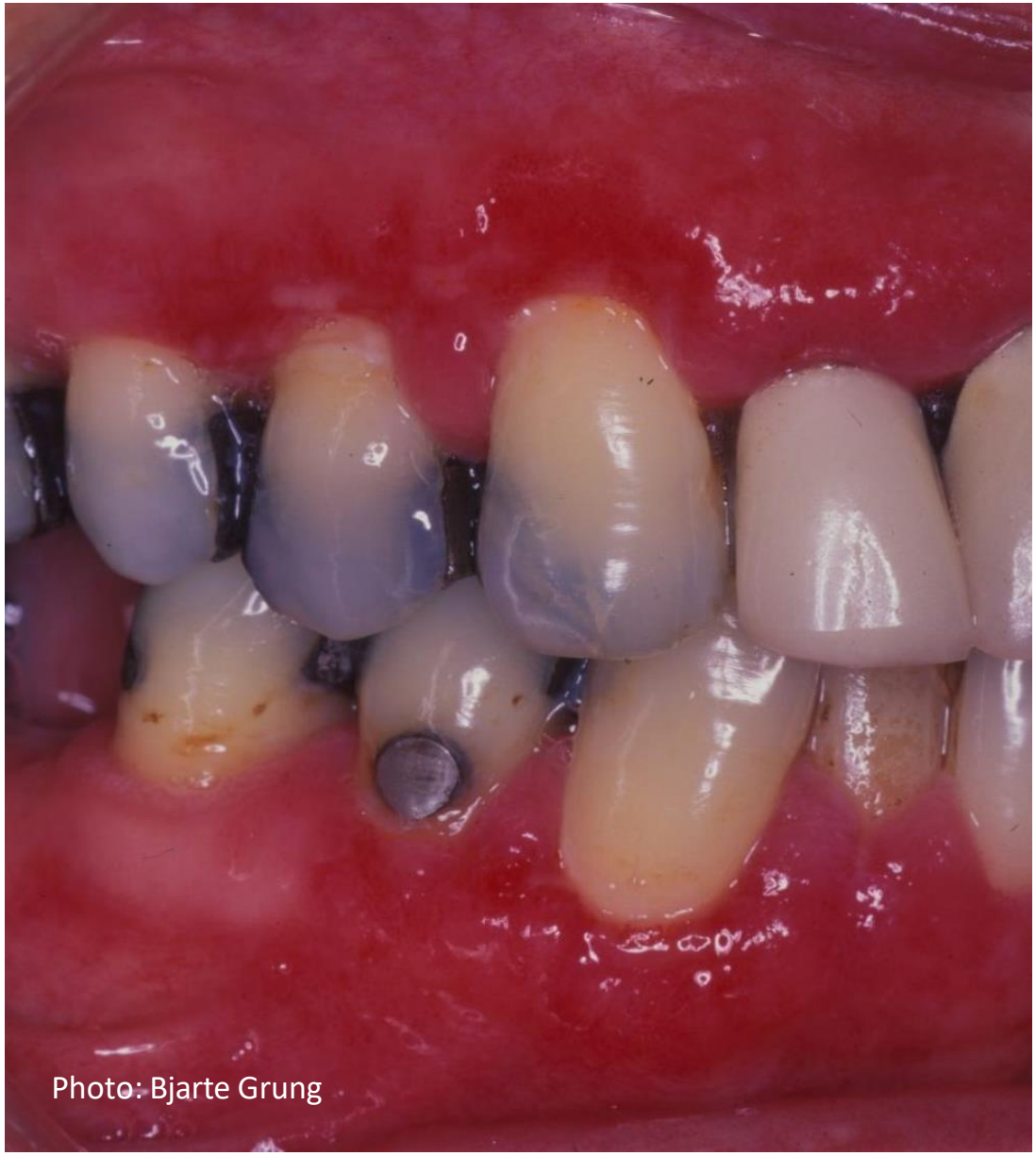


Photo: Bjarte Grung

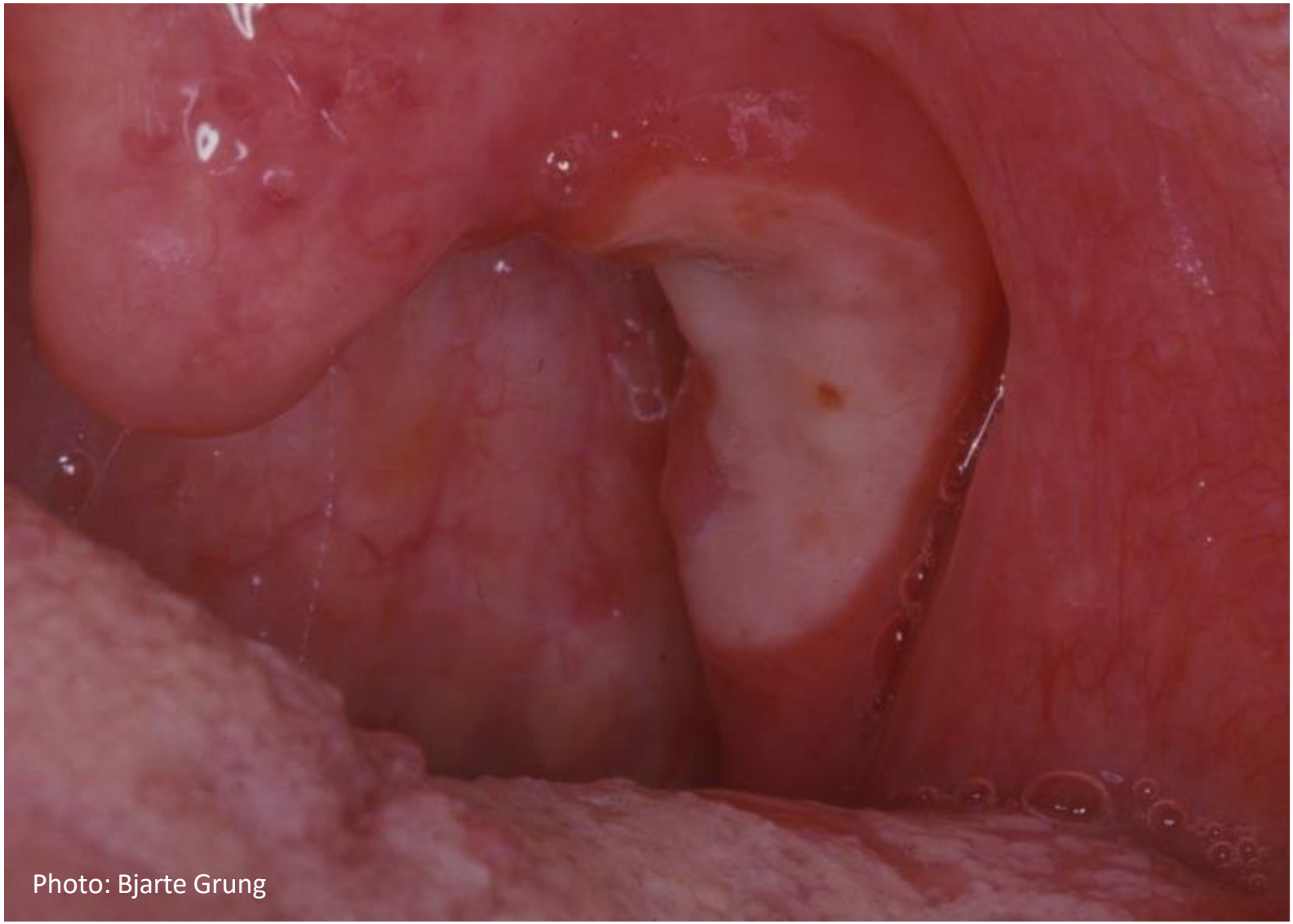
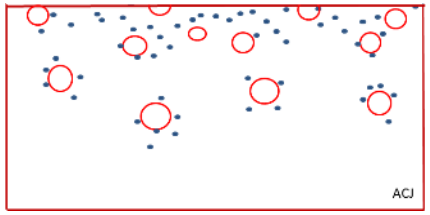
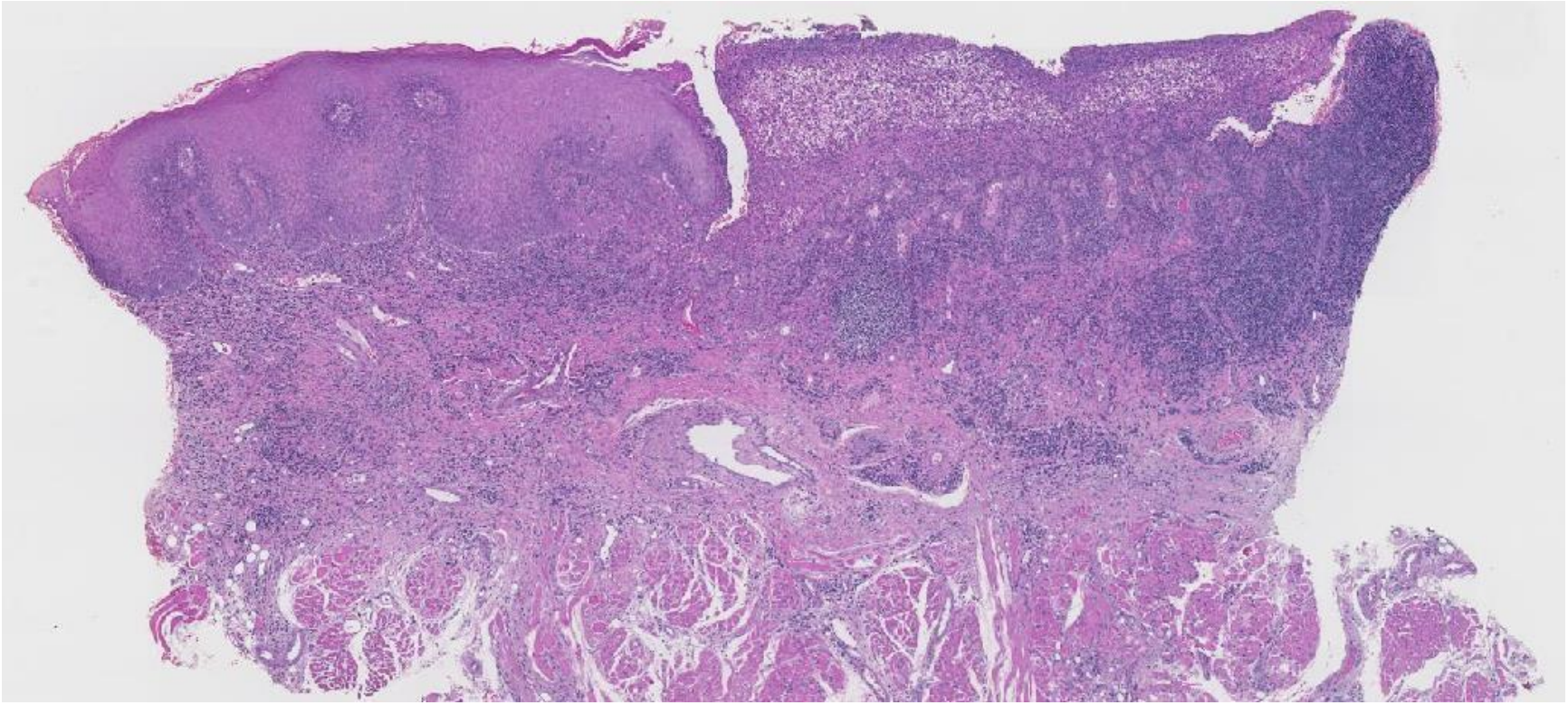
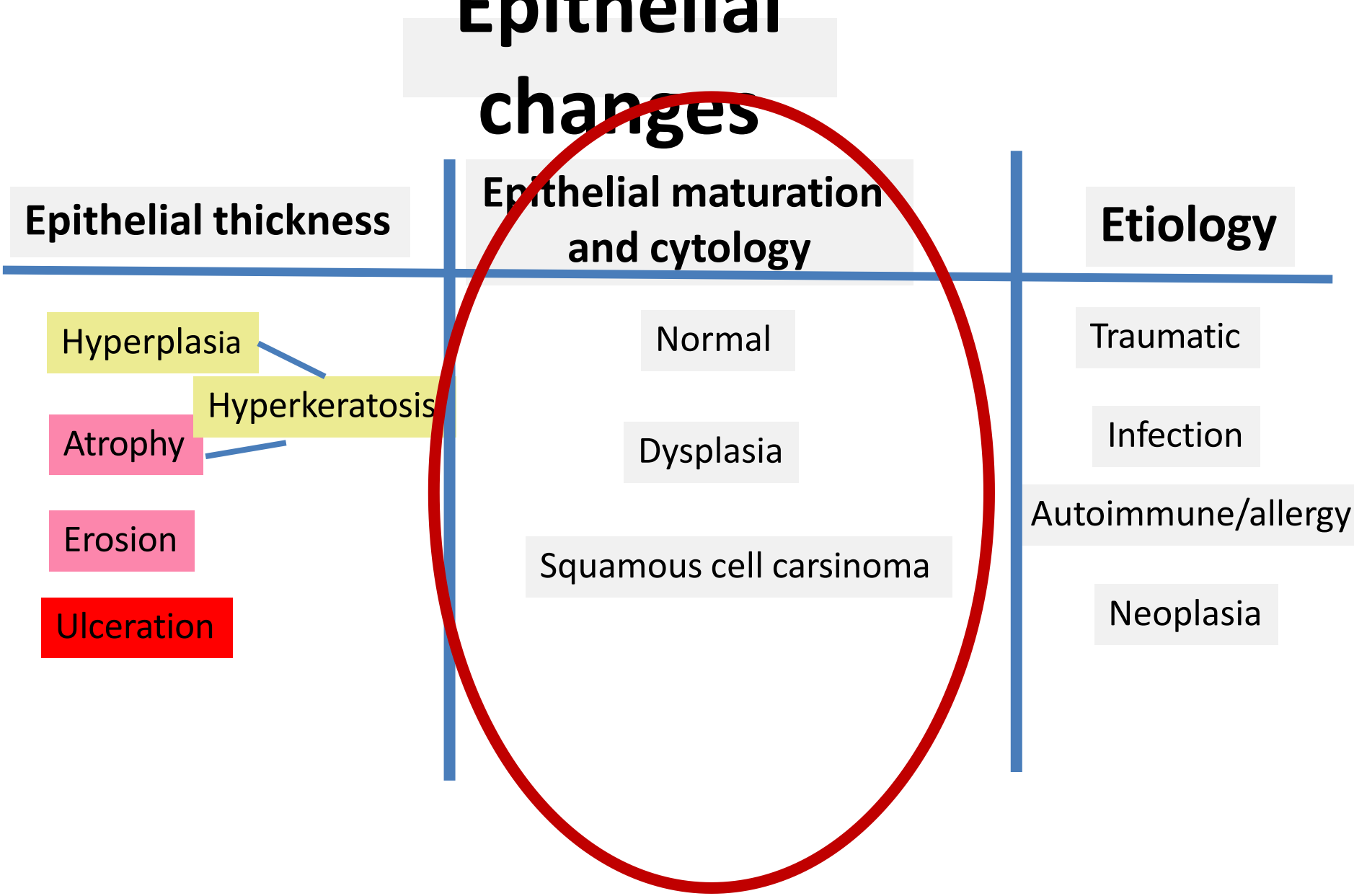


Photo: Bjarte Grung

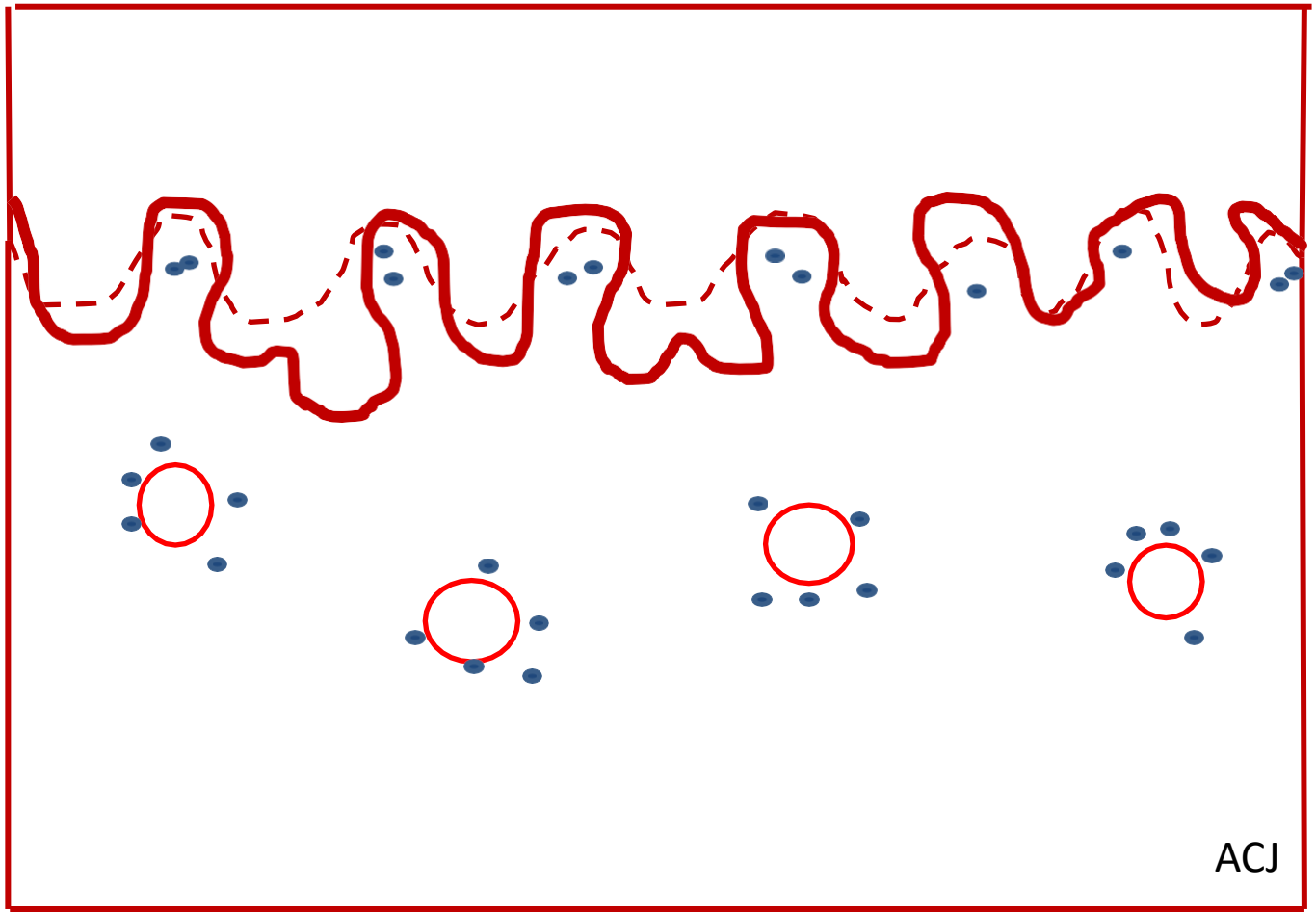
Female, 77 years old. Lesions lasted for 30 years. Whitish lesions and ulcerations on tongue and gingiva. Biopsy from rim of the tongue, right side.

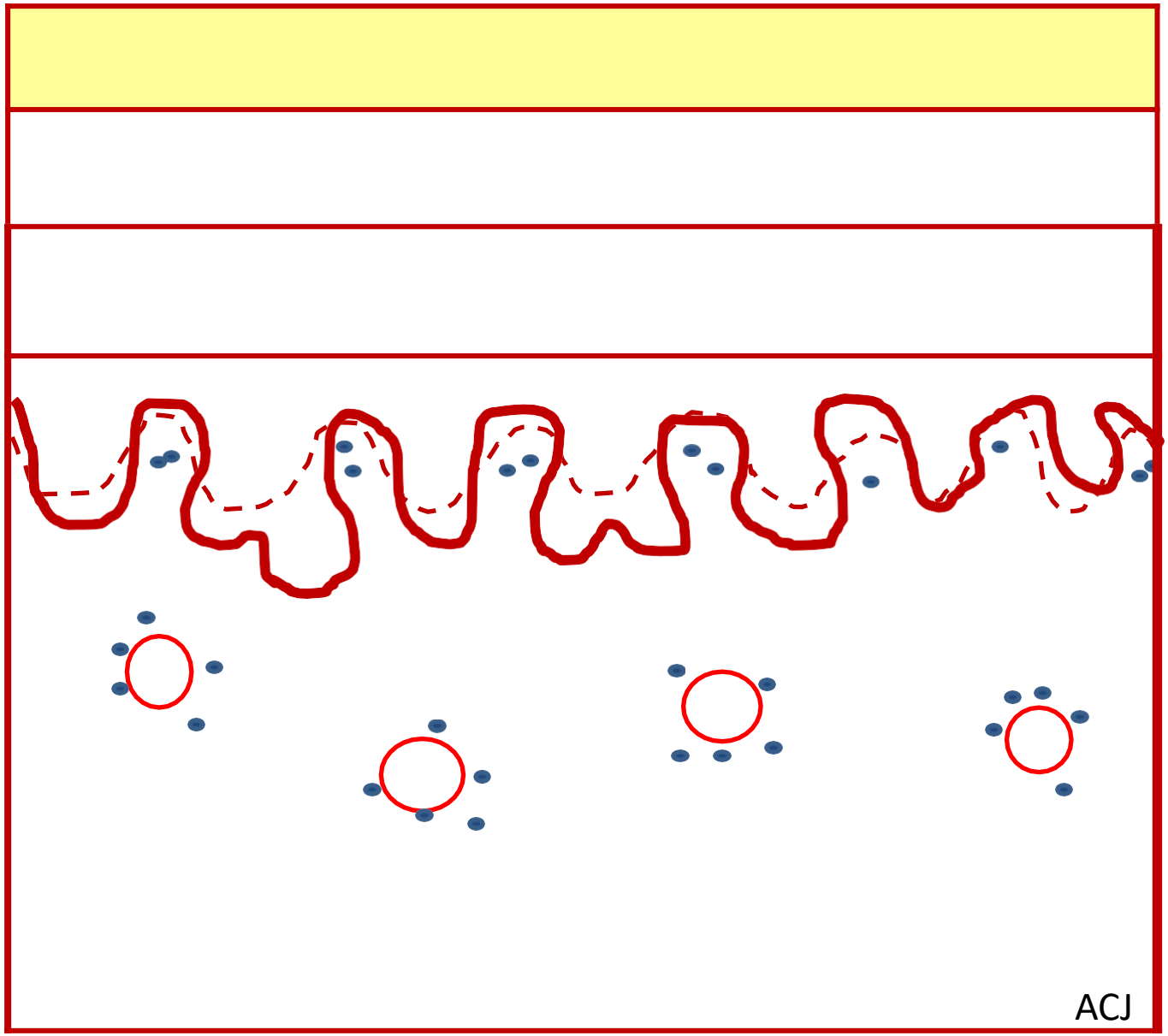





Epithelial changes
Epithelial thickness
Epithelial maturation and cytology
Etiology
Hyperplasia
Hyperkeratosis
Atrophy
Erosion
Ulceration
Normal
Dysplasia
Squamous cell carsinoma
Traumatic
Infection
Autoimmune/allergy
Neoplasia

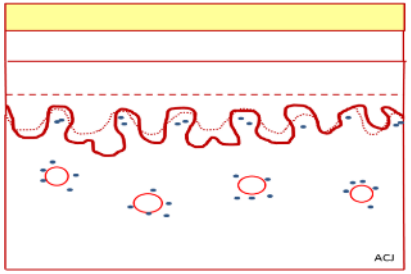
Dysplasia



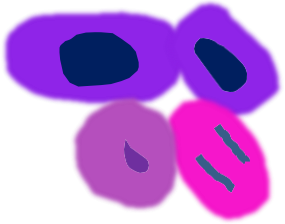

ACJ

Criteria for dysplasia

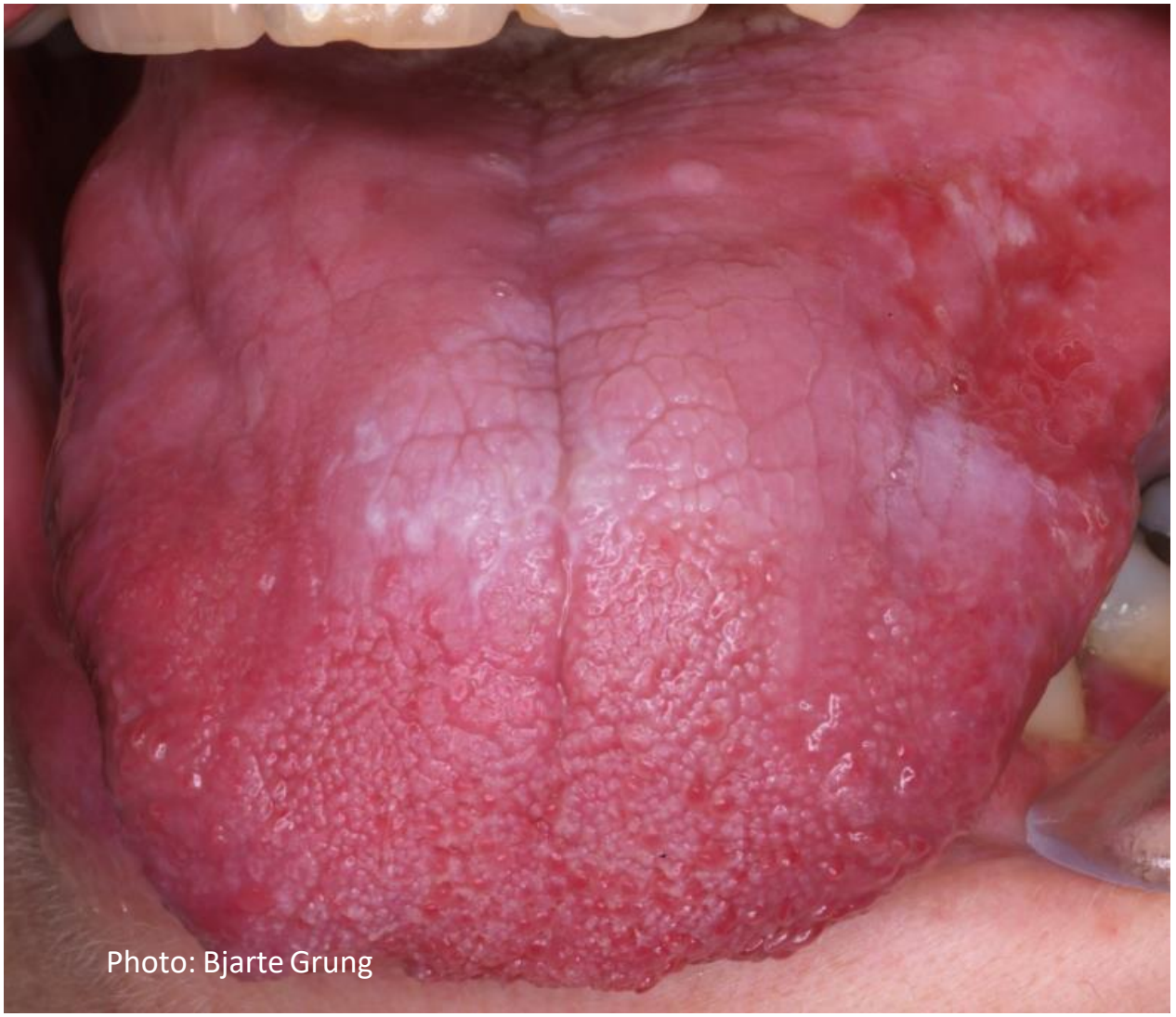
Architecture of epithelium



Cytology



Female, 61 years old. Mixture of red and white, looks like atrophic lichen planus, alternatively erythro-leukoplakia. I will not be surprised if there is dysplasia



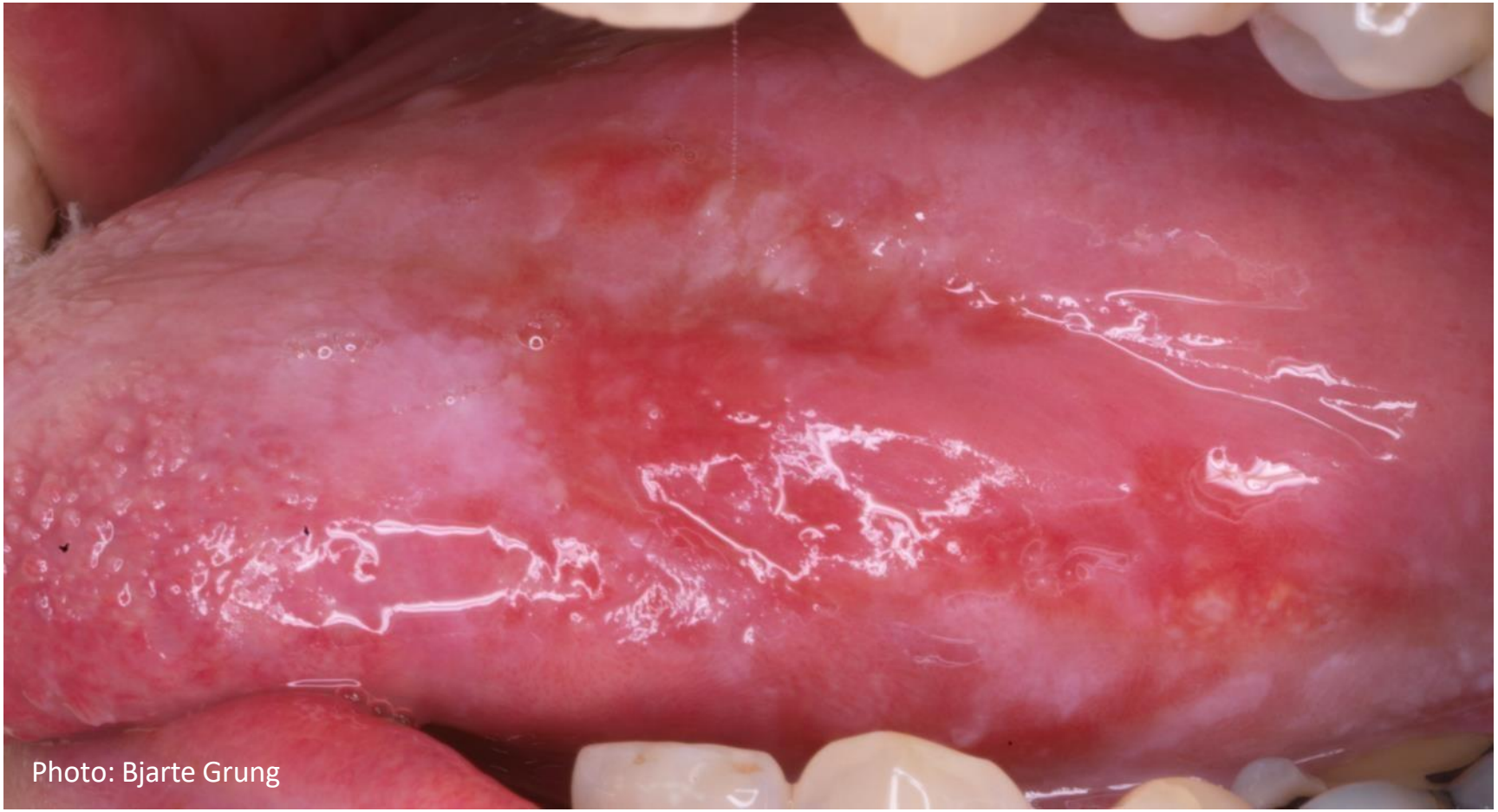
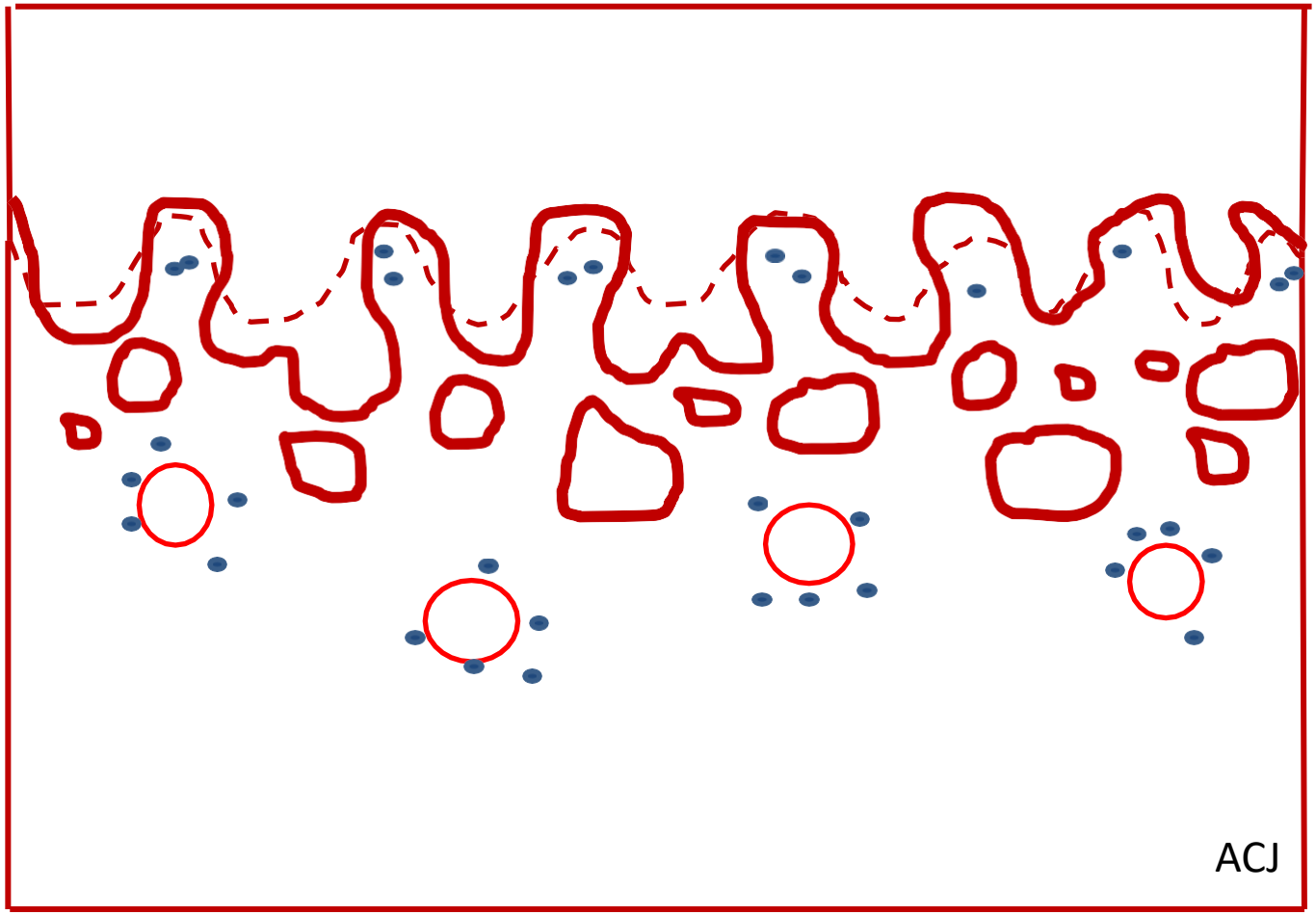
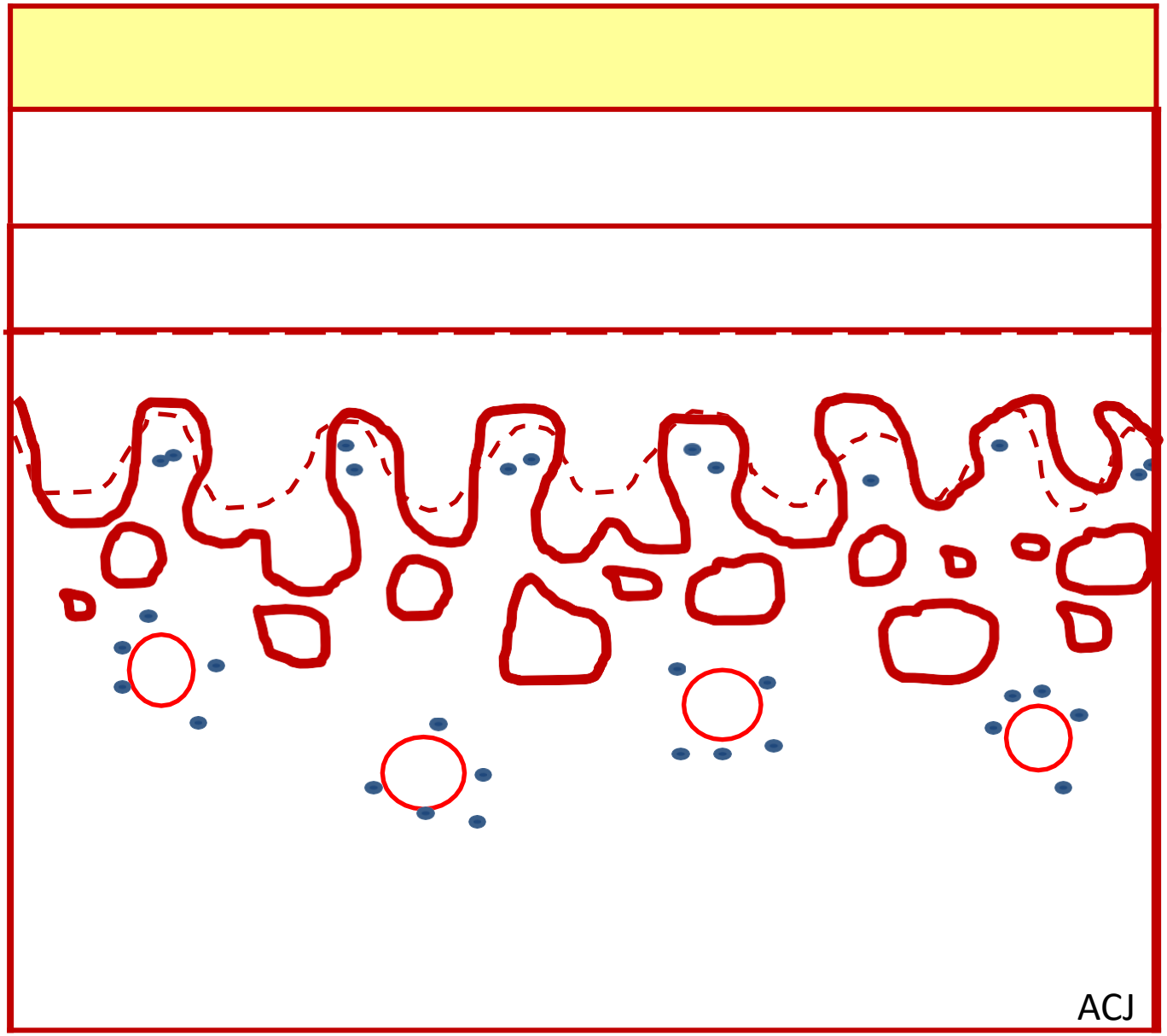


Photo: Bjarte Grung

Squamous cell carcinoma




ACJ

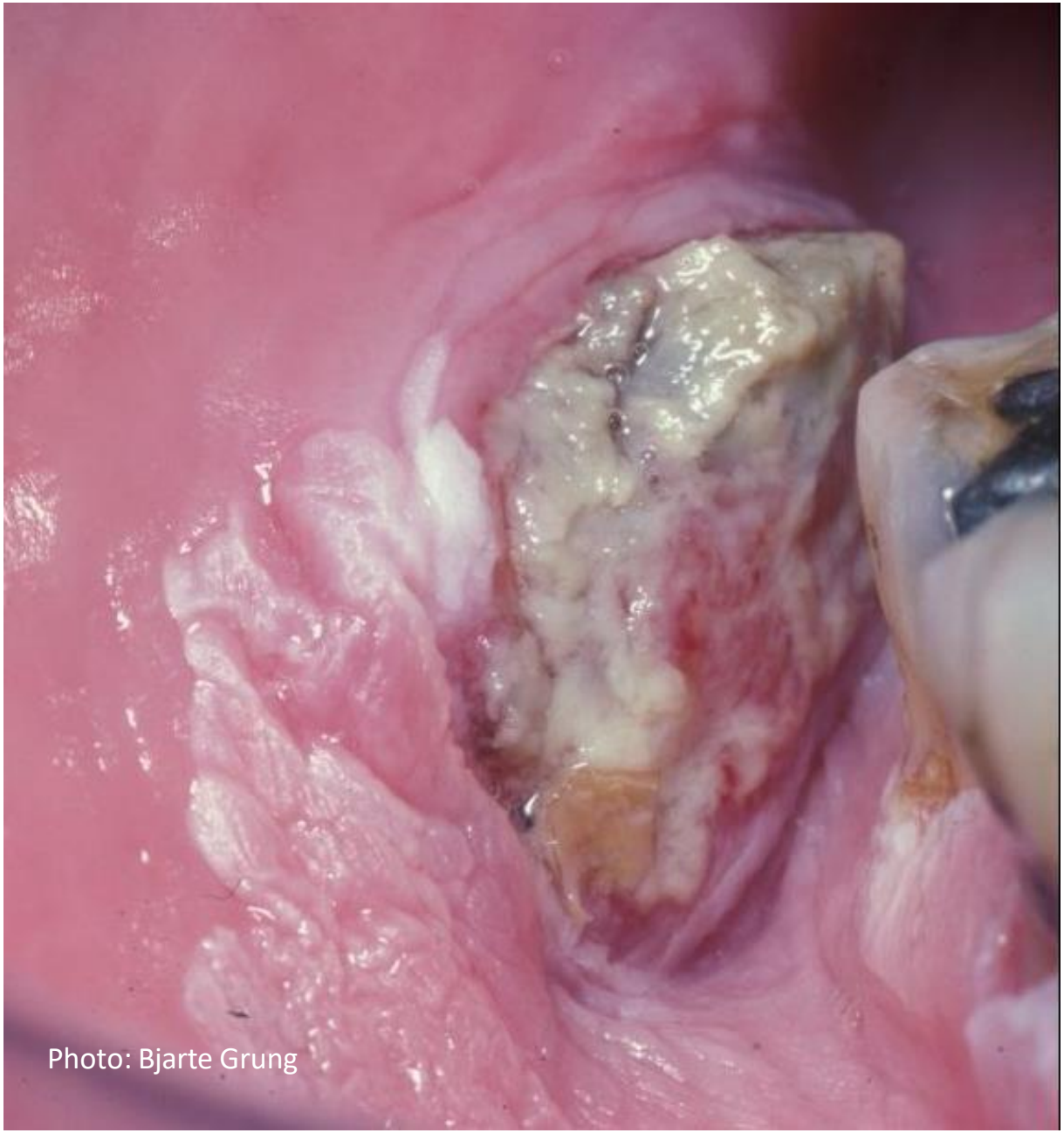


Photo: Bjarte Grung

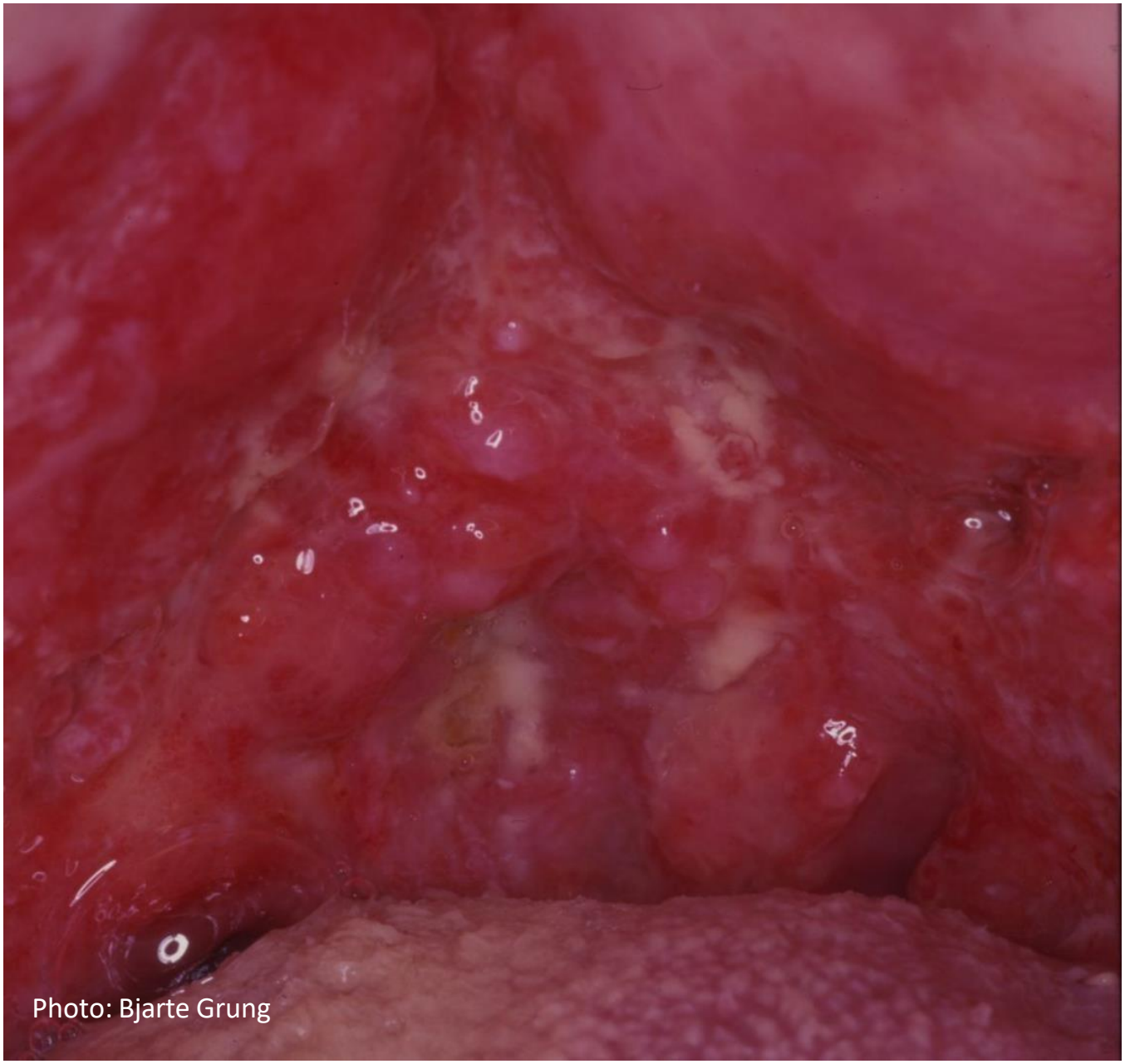


Photo: Bjarte Grung

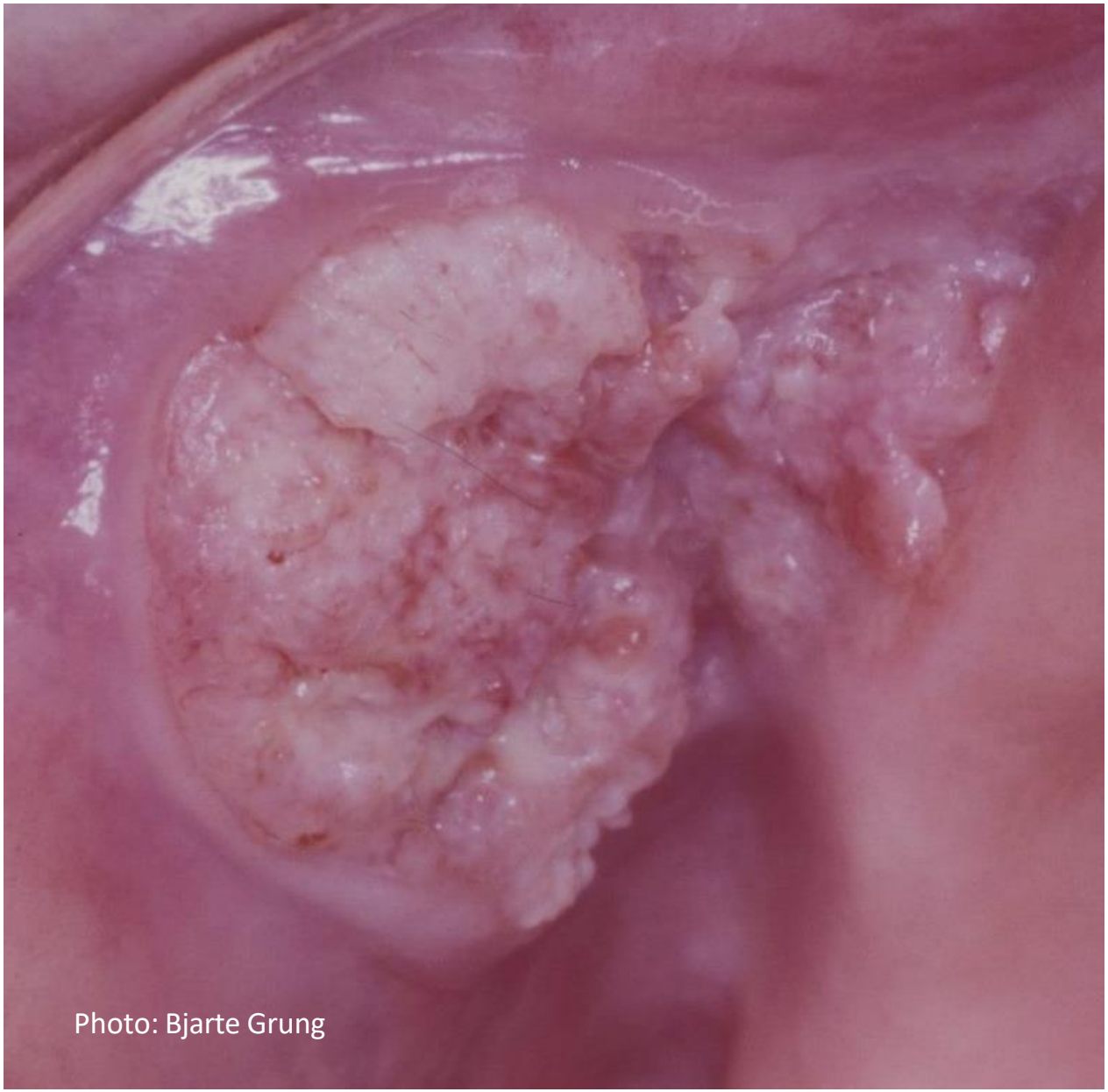
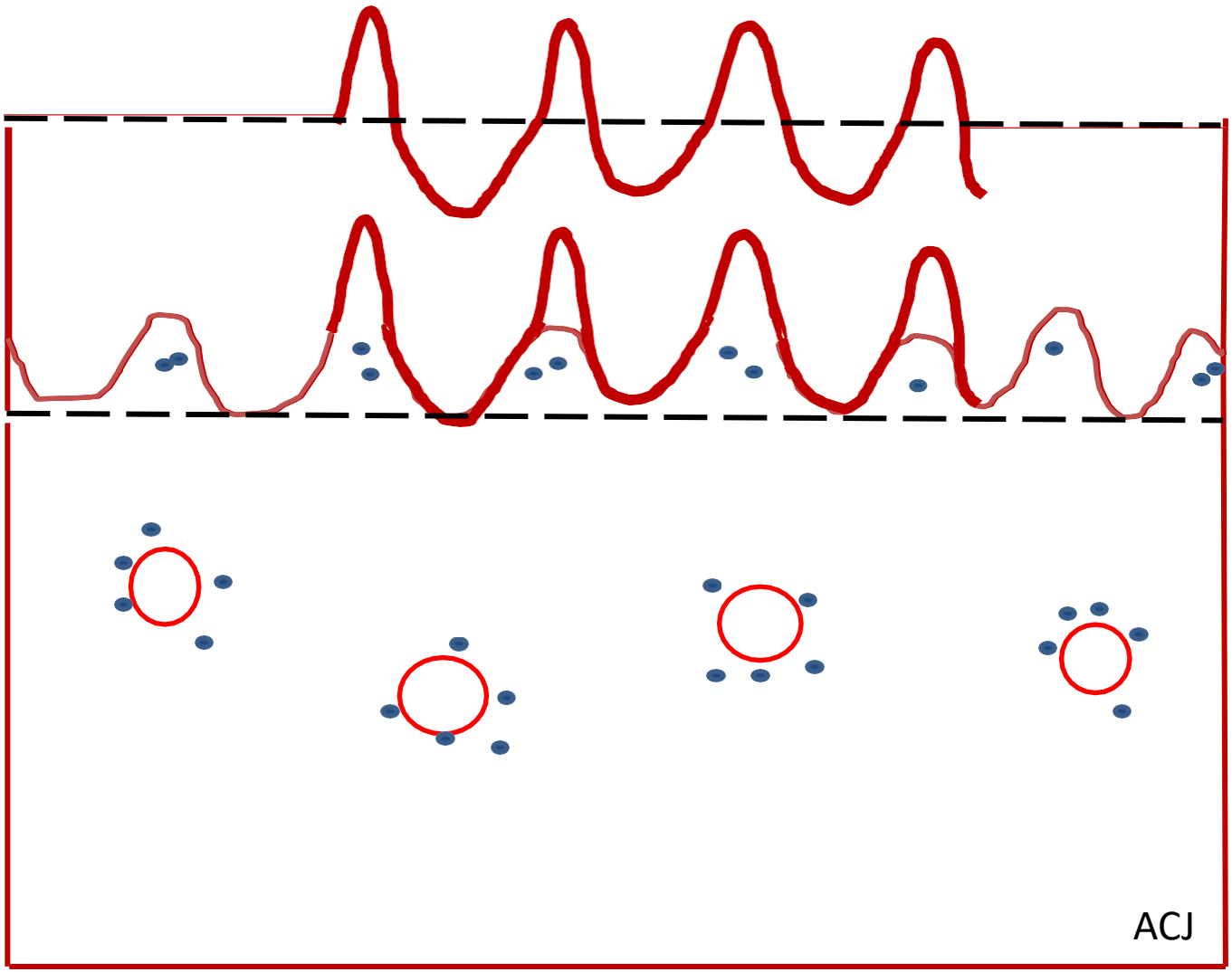
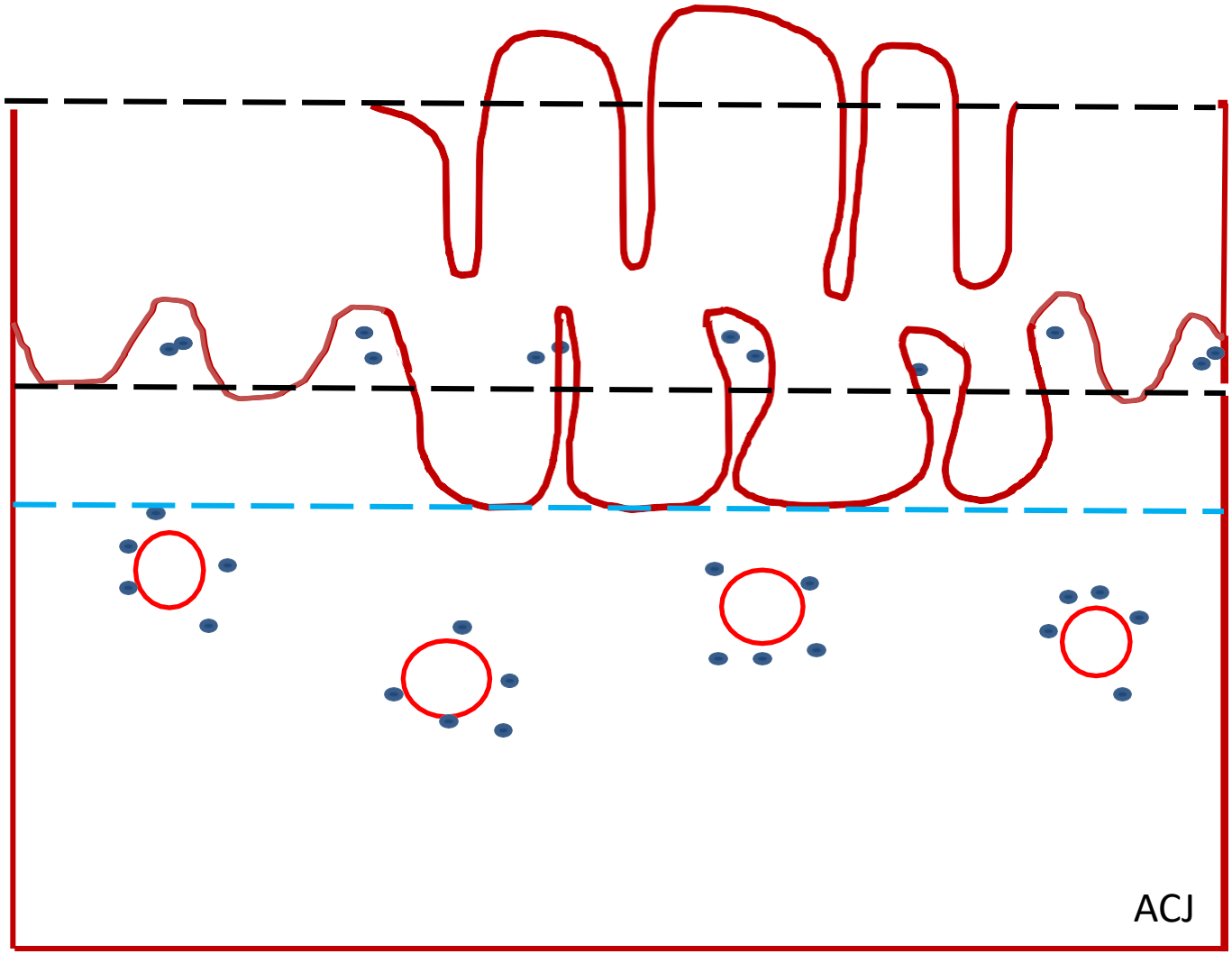


Photo: Bjarte Grung

Verrucous hyperplasia

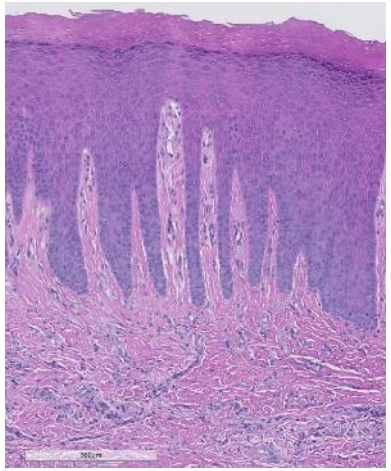


Verrucous carcinoma

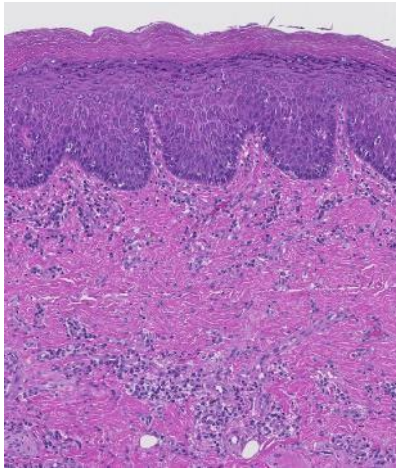


Grading of epithelial atypia

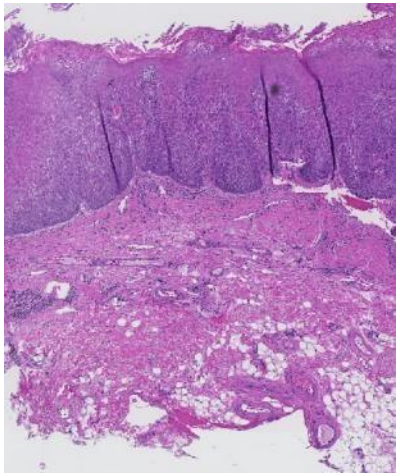
Hyperplasi and hyperkeratosis



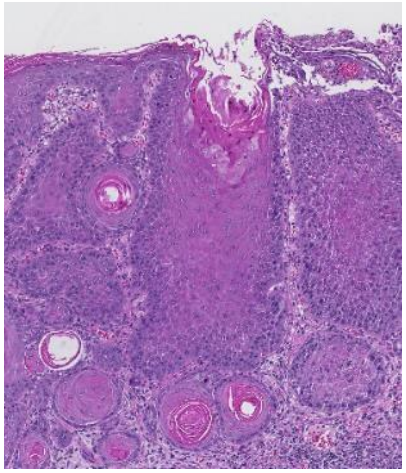
Mild/moderate dysplasia



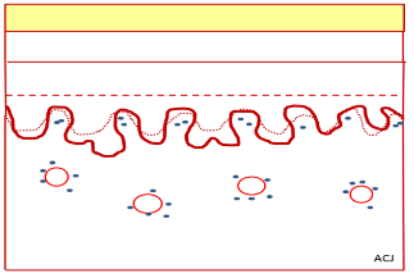
Severe dysplasia

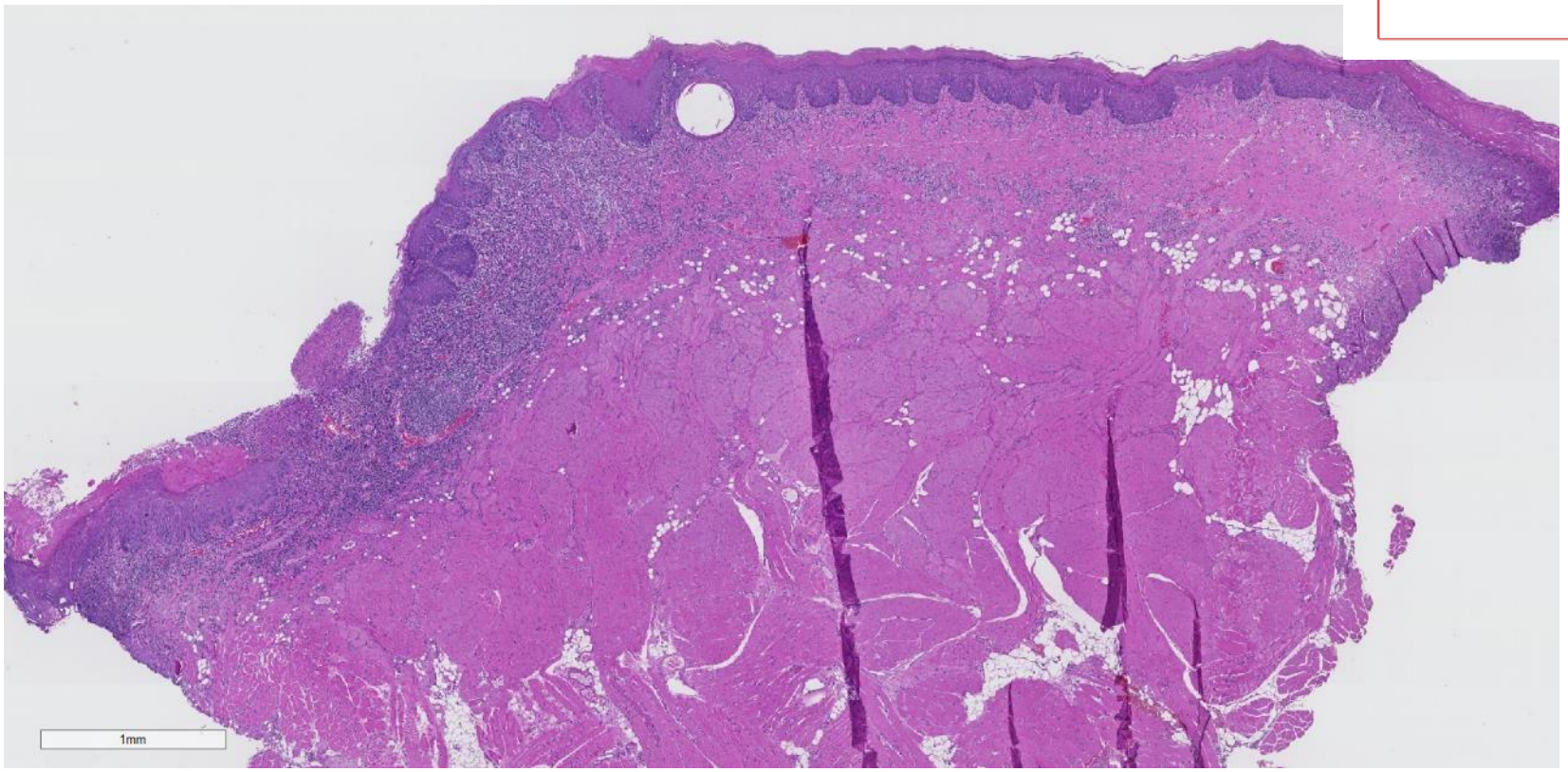


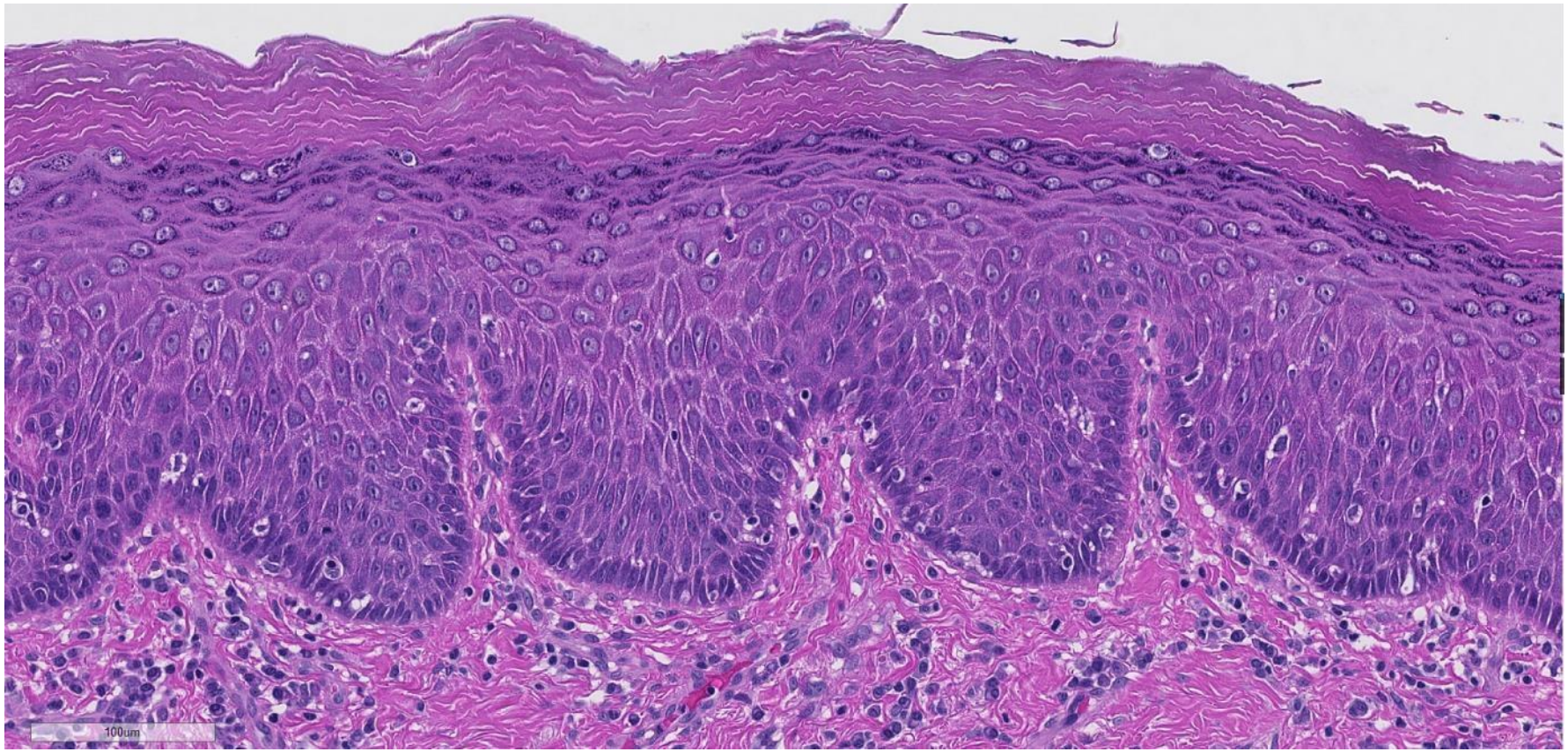
Squamous cell carcinoma



Male, 47 years old. Leukoplakia/erythroplakia from the rim of the tongue, right side. Looks suspicious. Carsinoma in situ?



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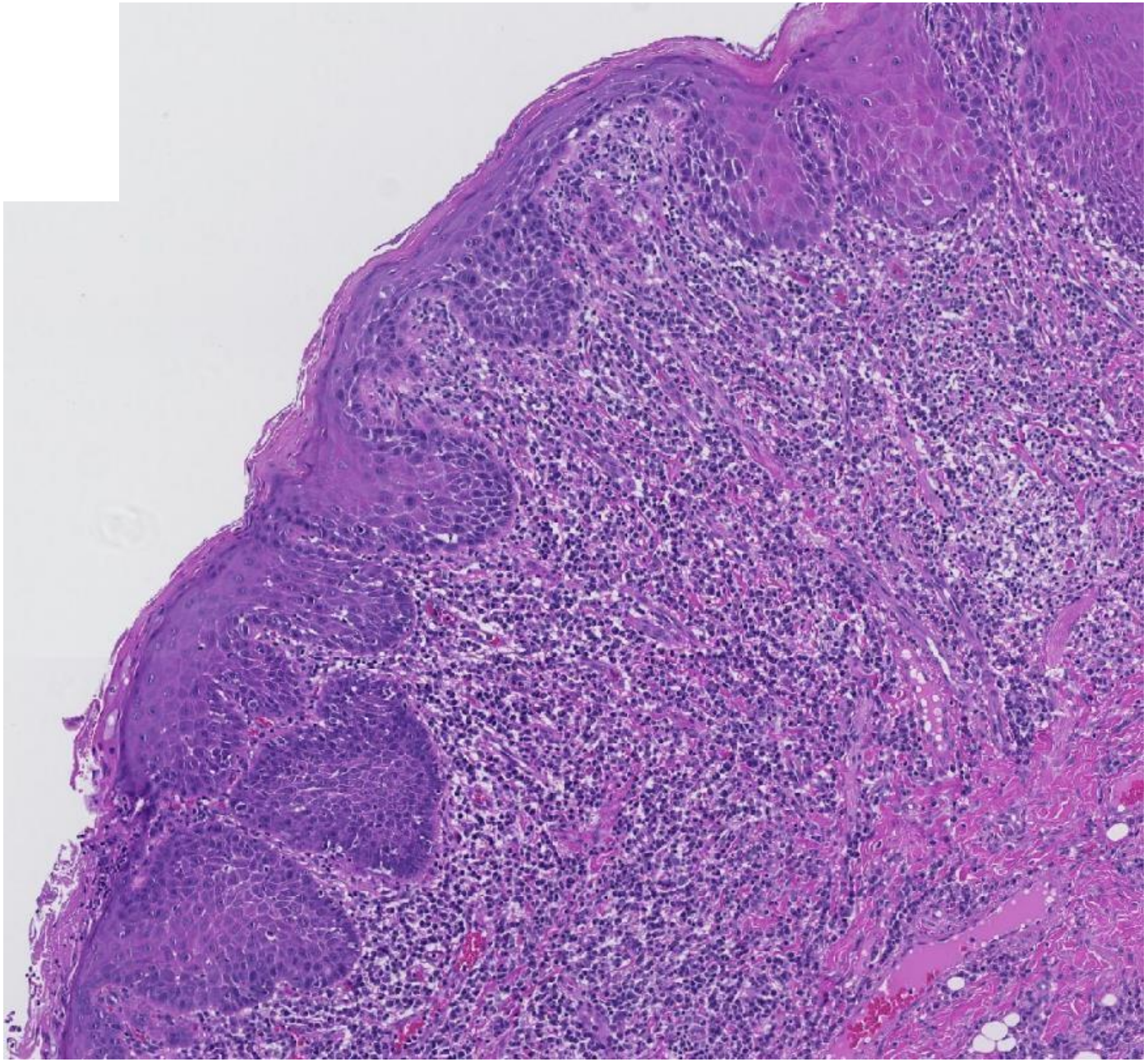

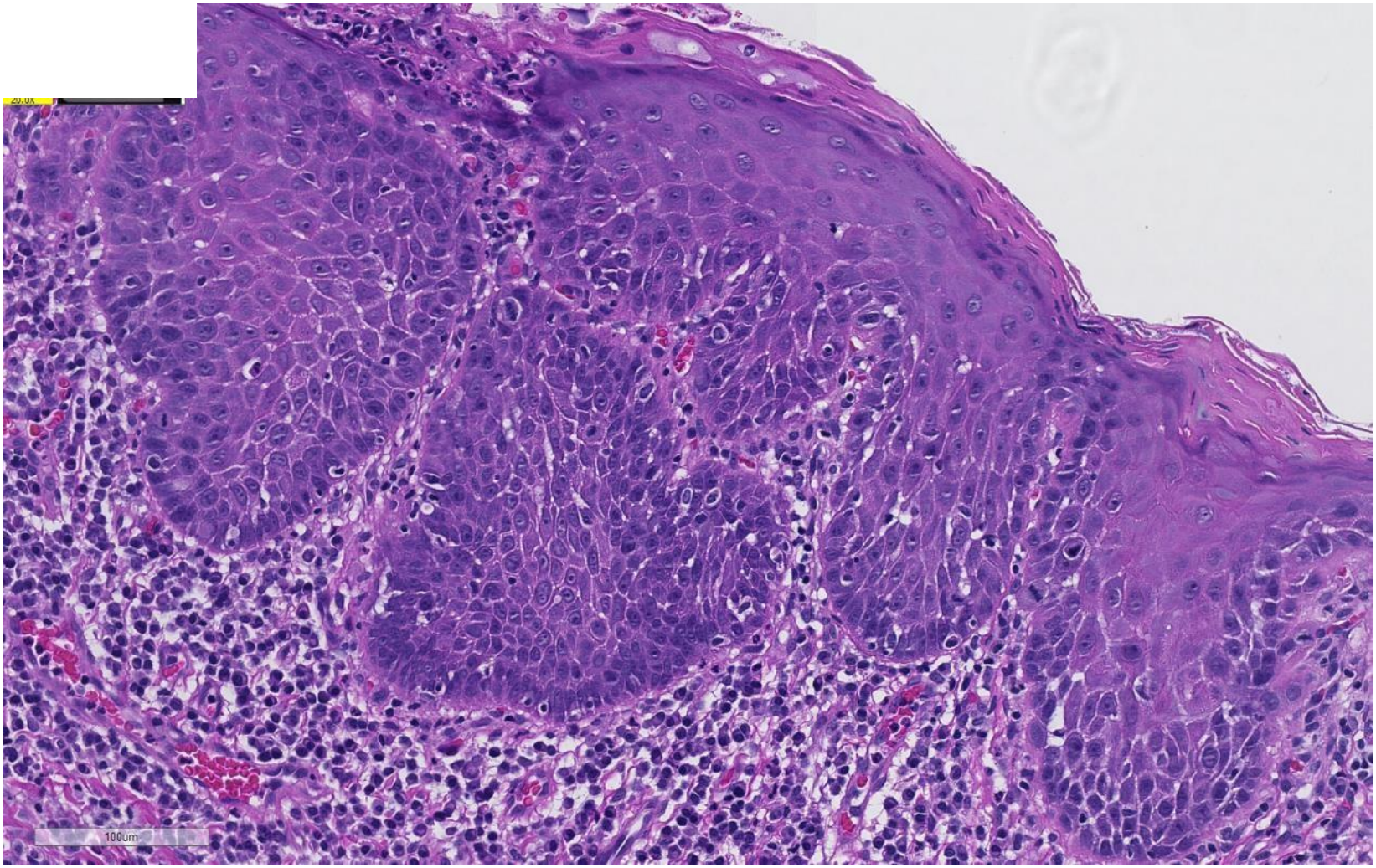
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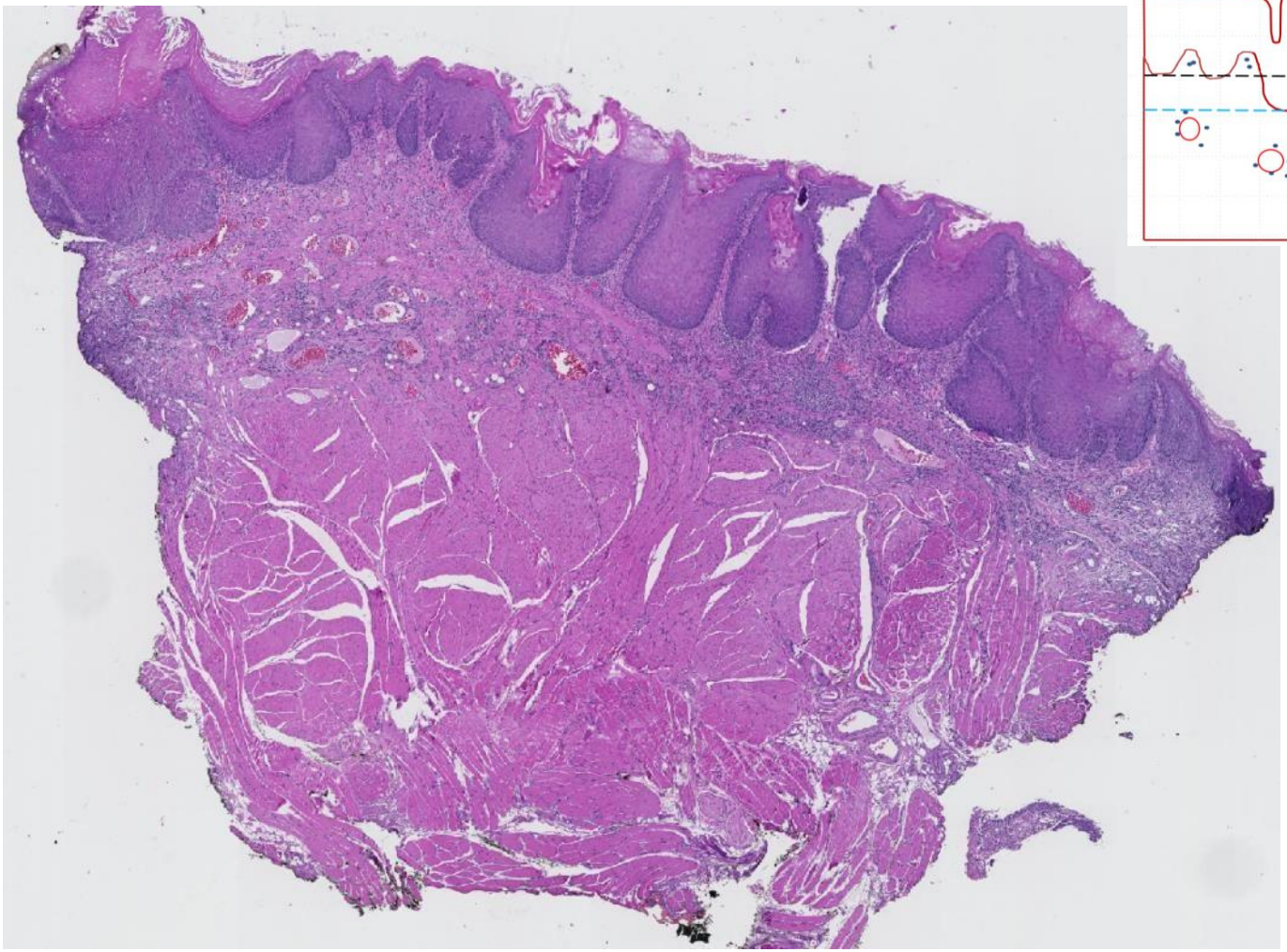

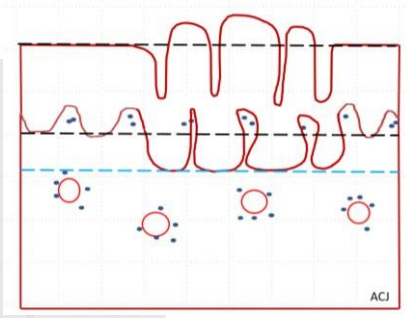
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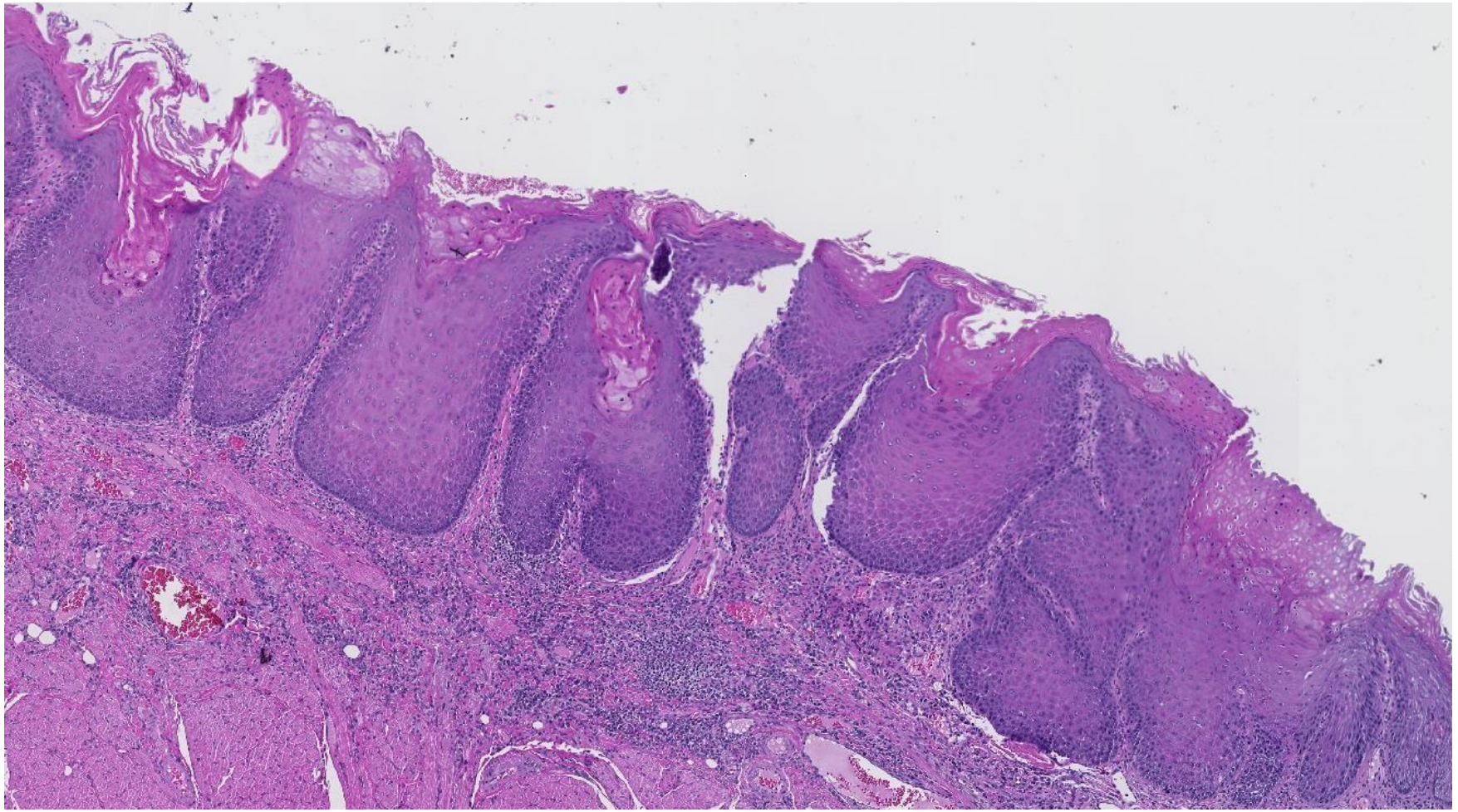

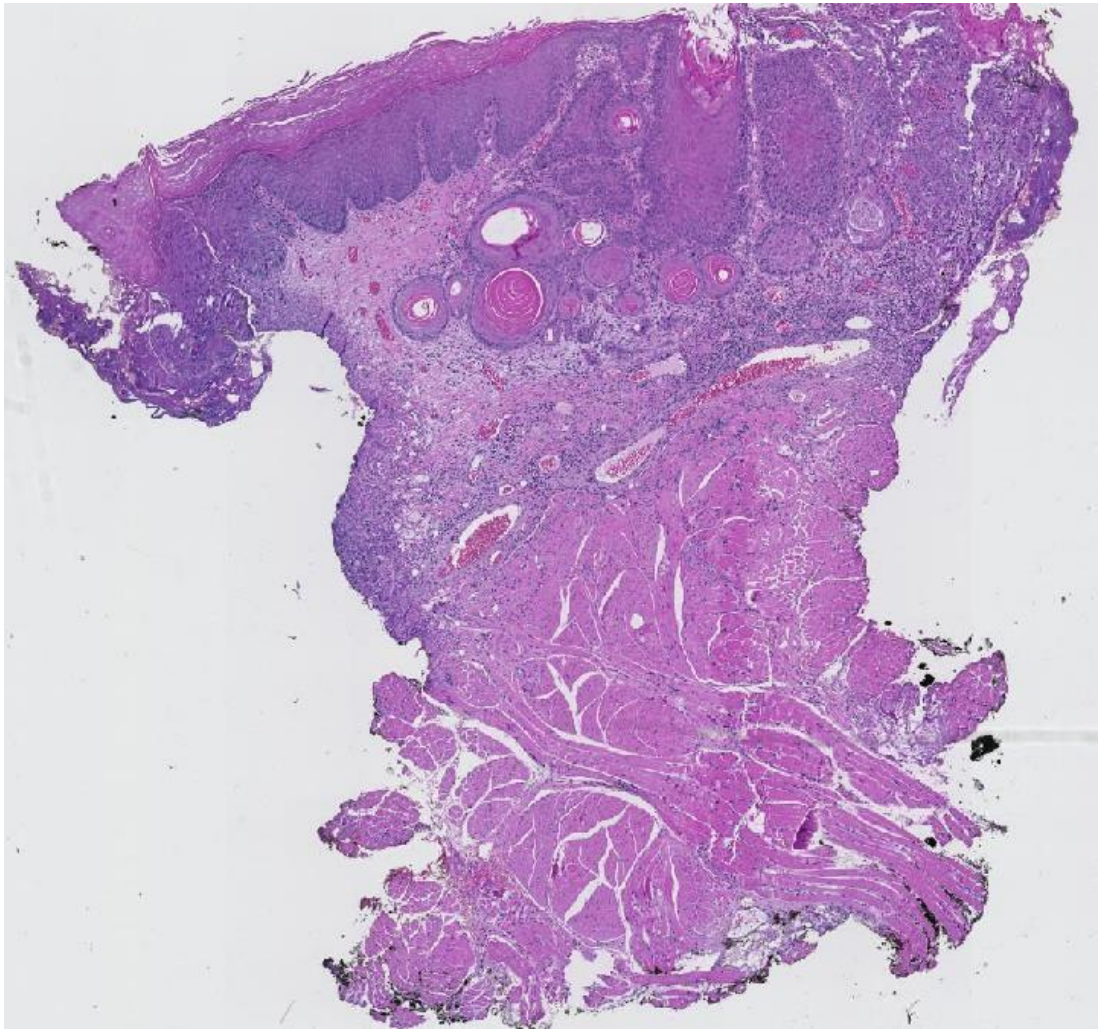

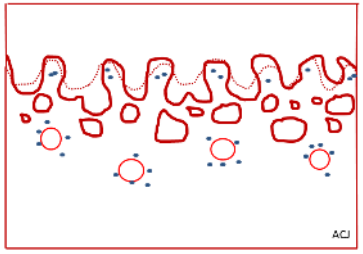
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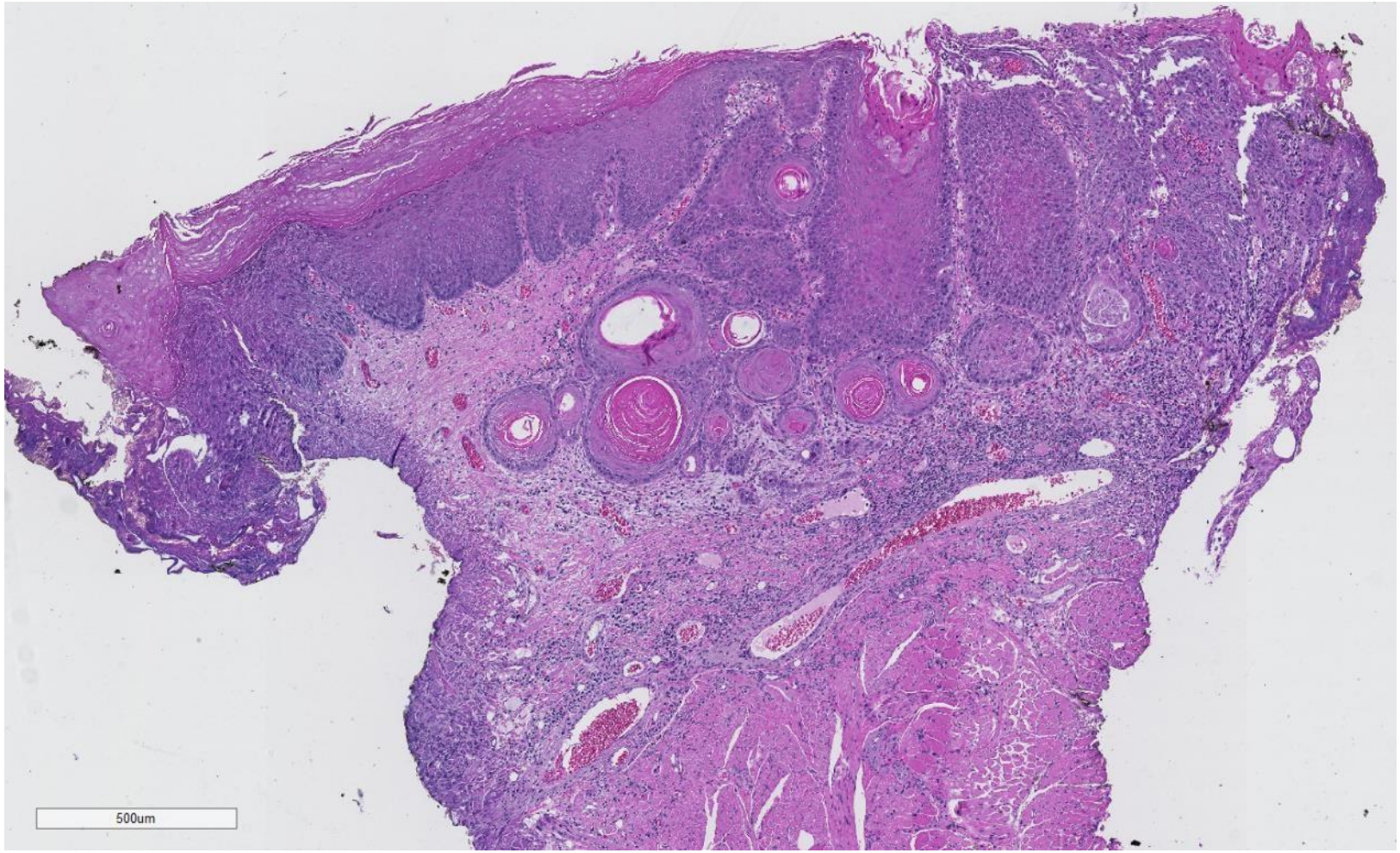
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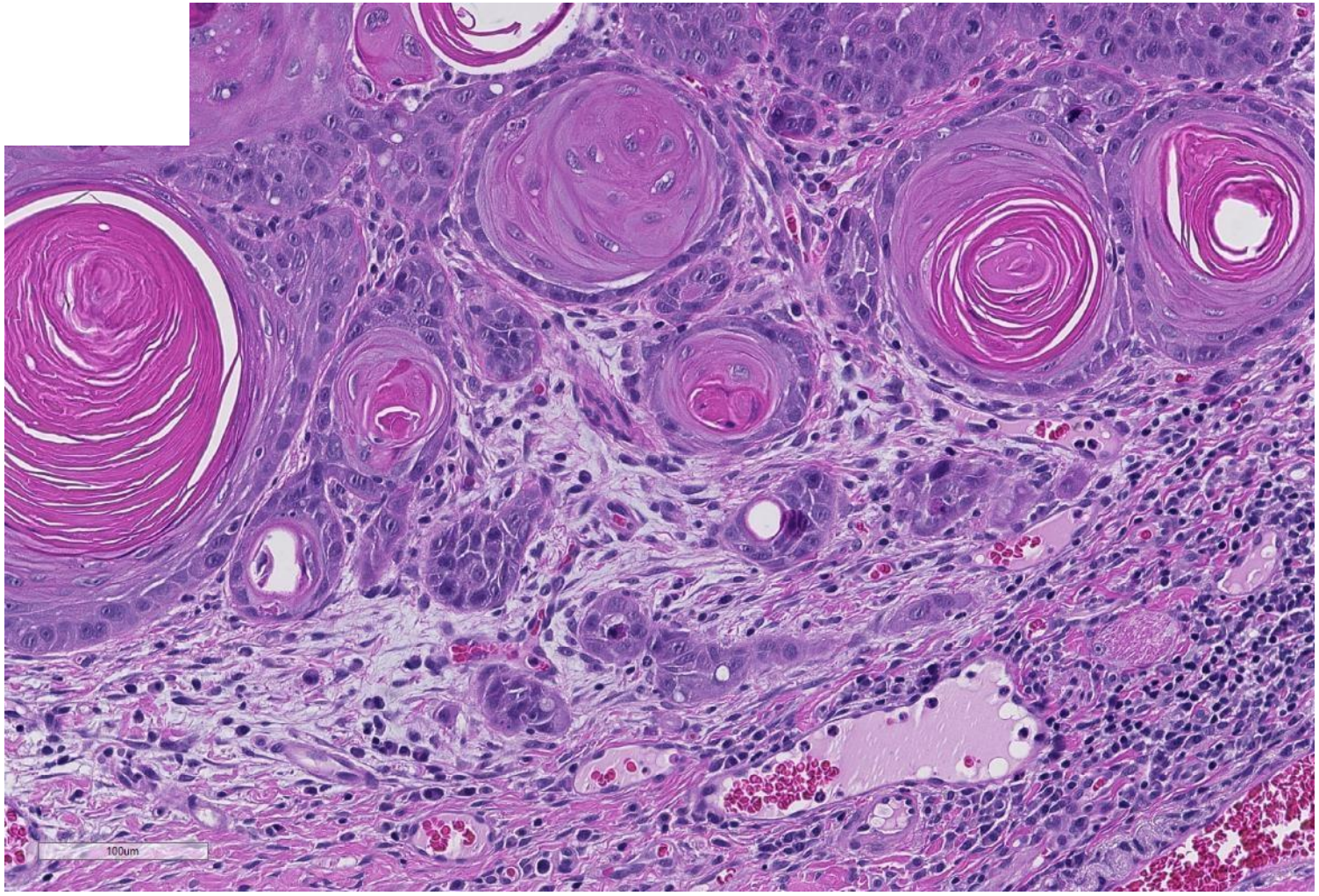
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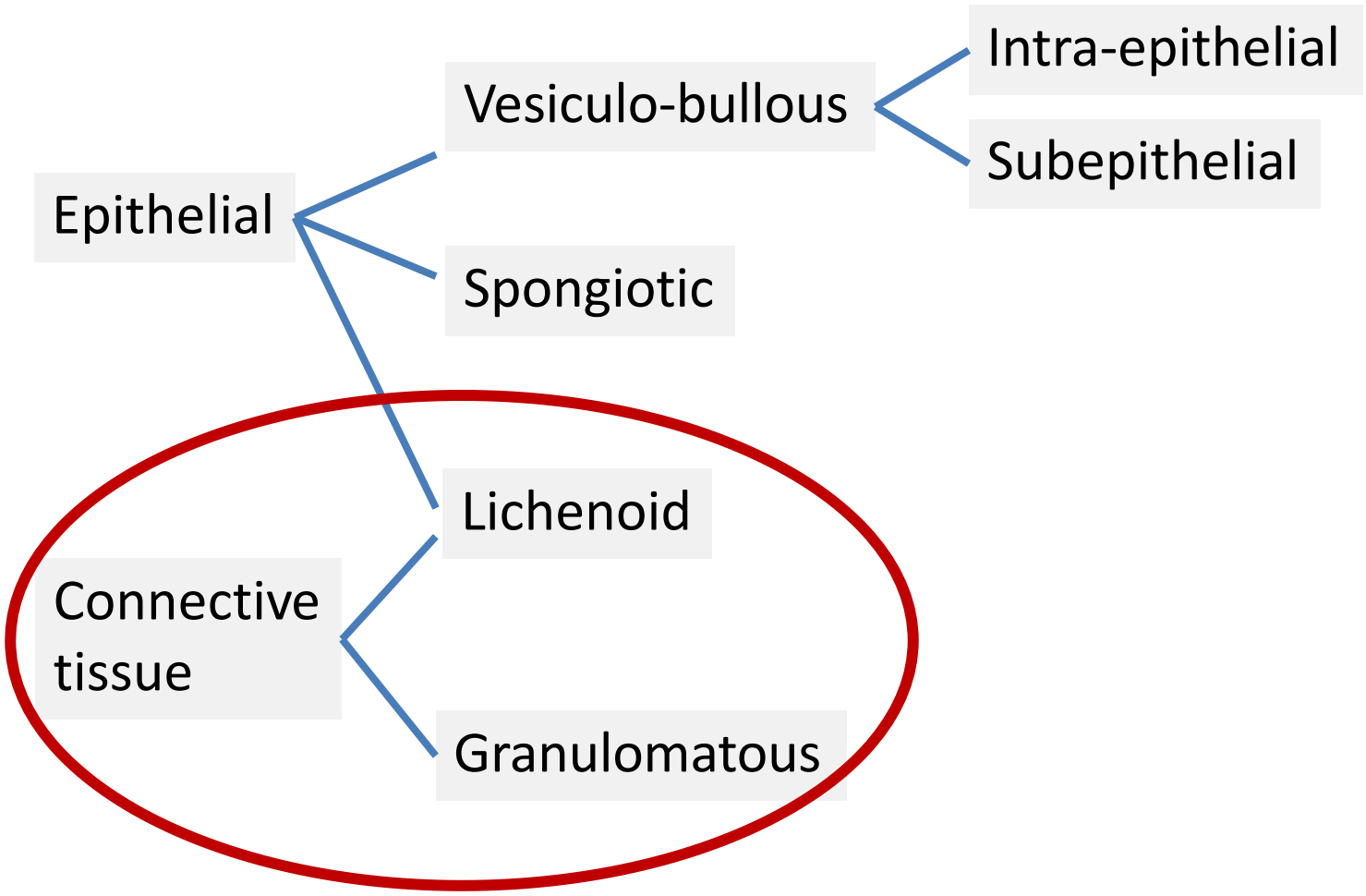
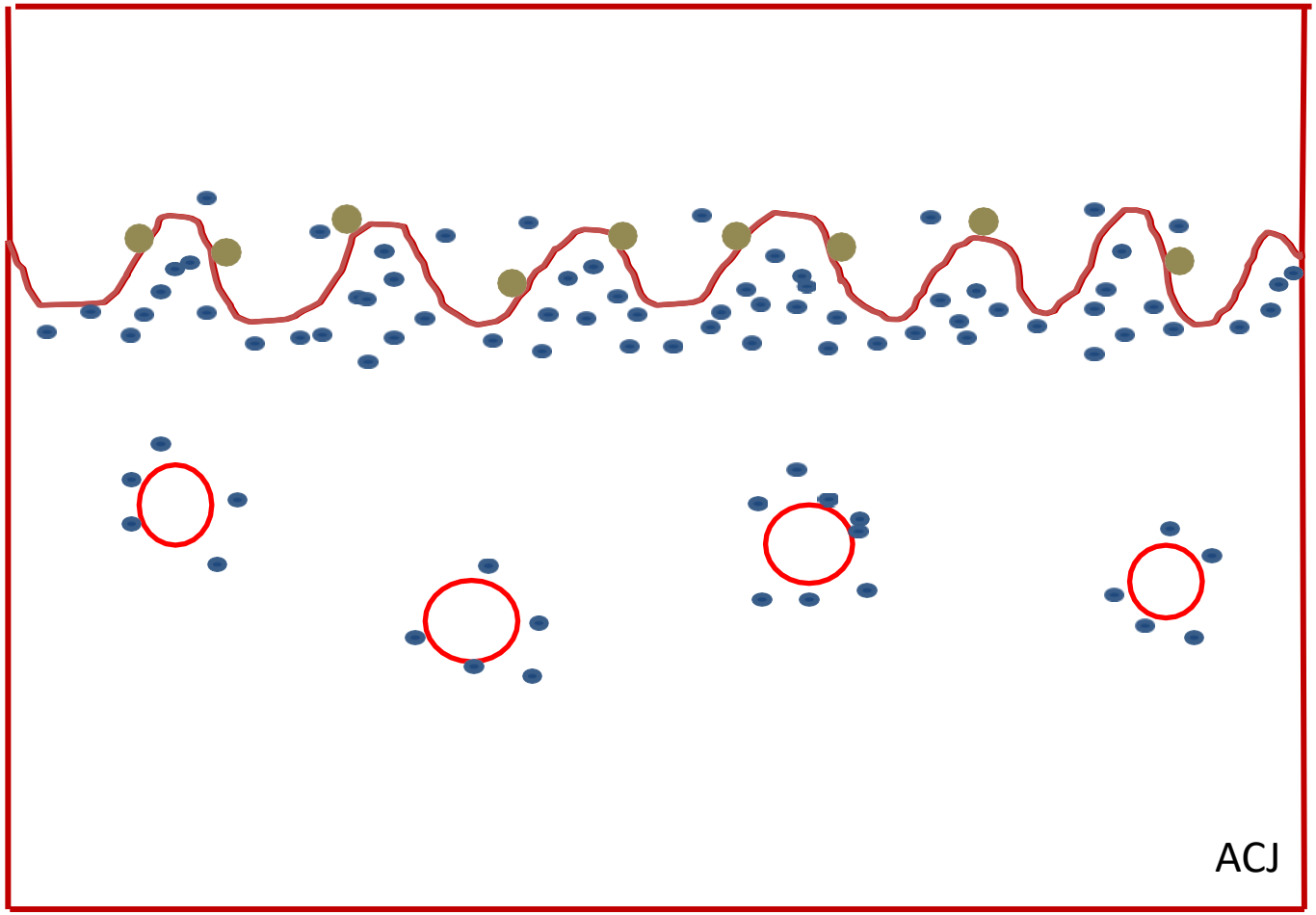
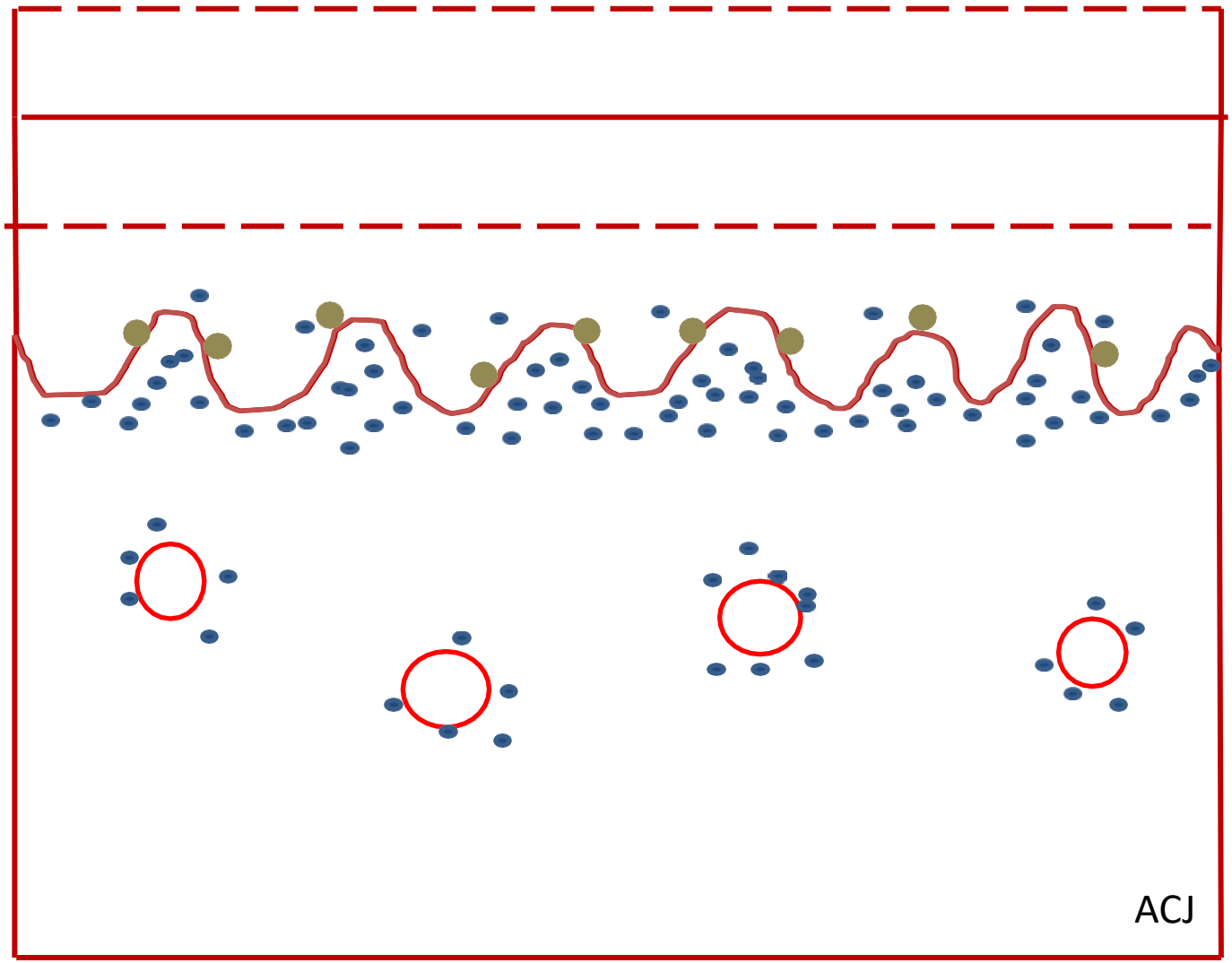
Inflammatory diseases in oral mucosa



Lichenoid reaction pattern

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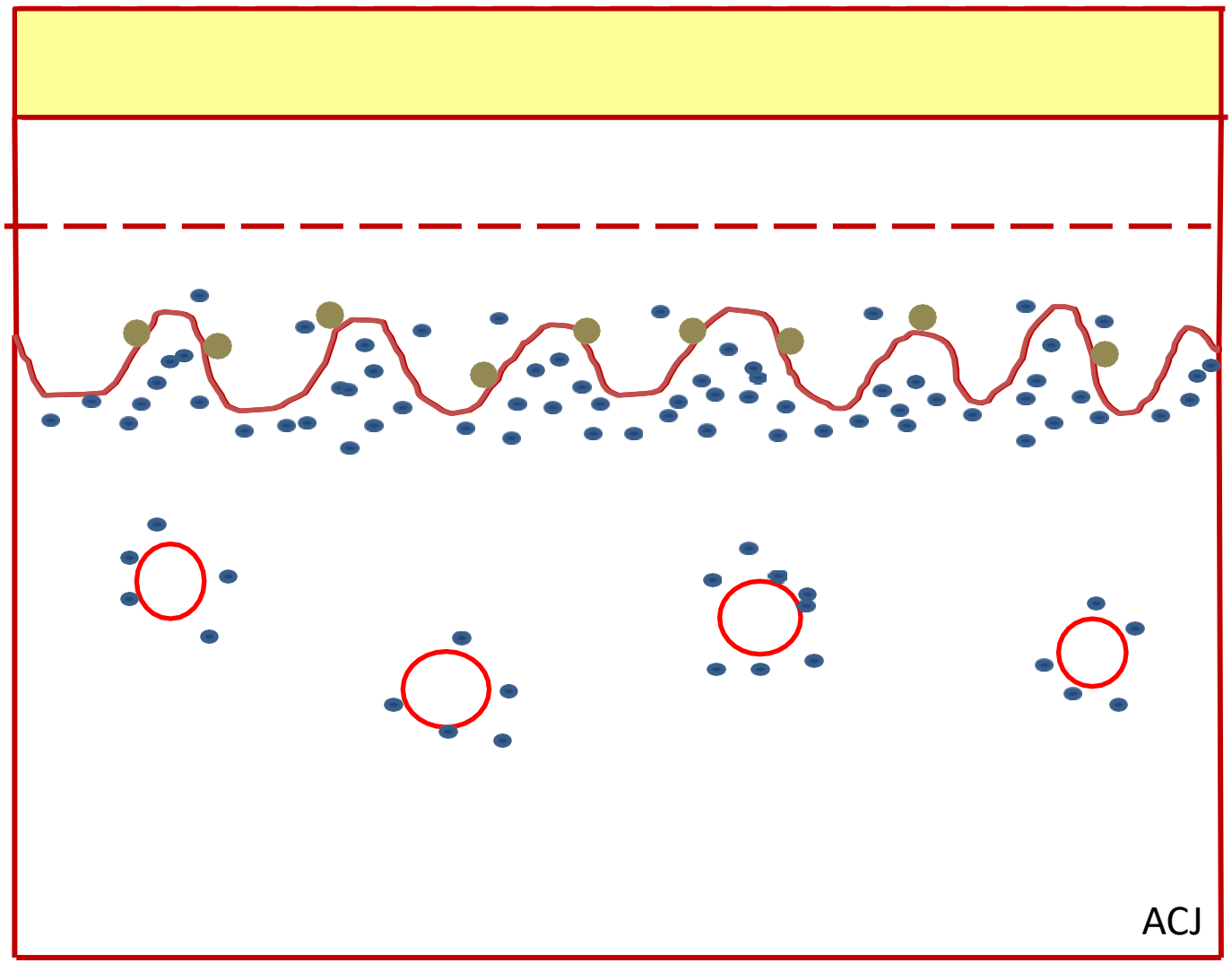
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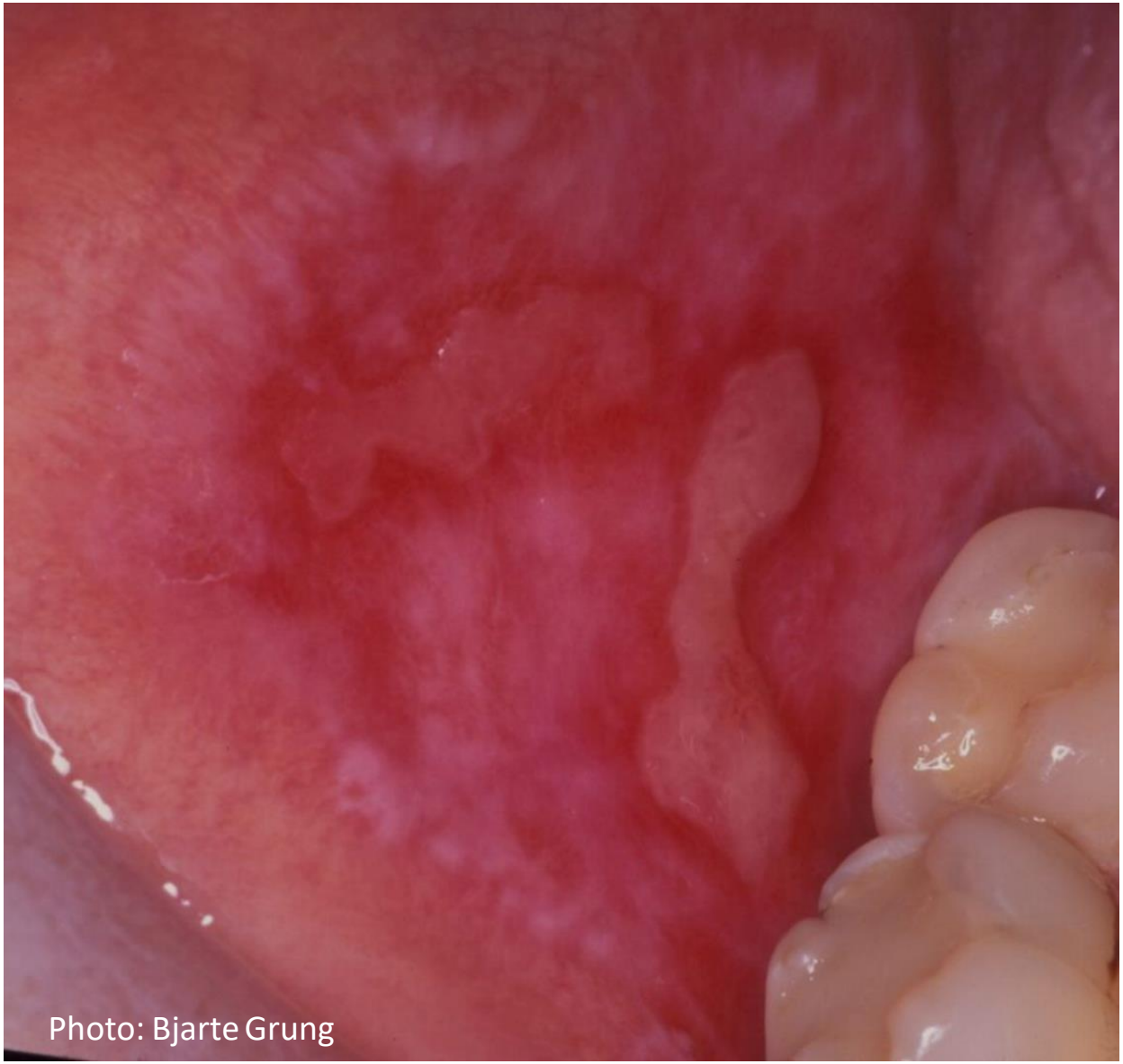


Photo: Bjarte Grung

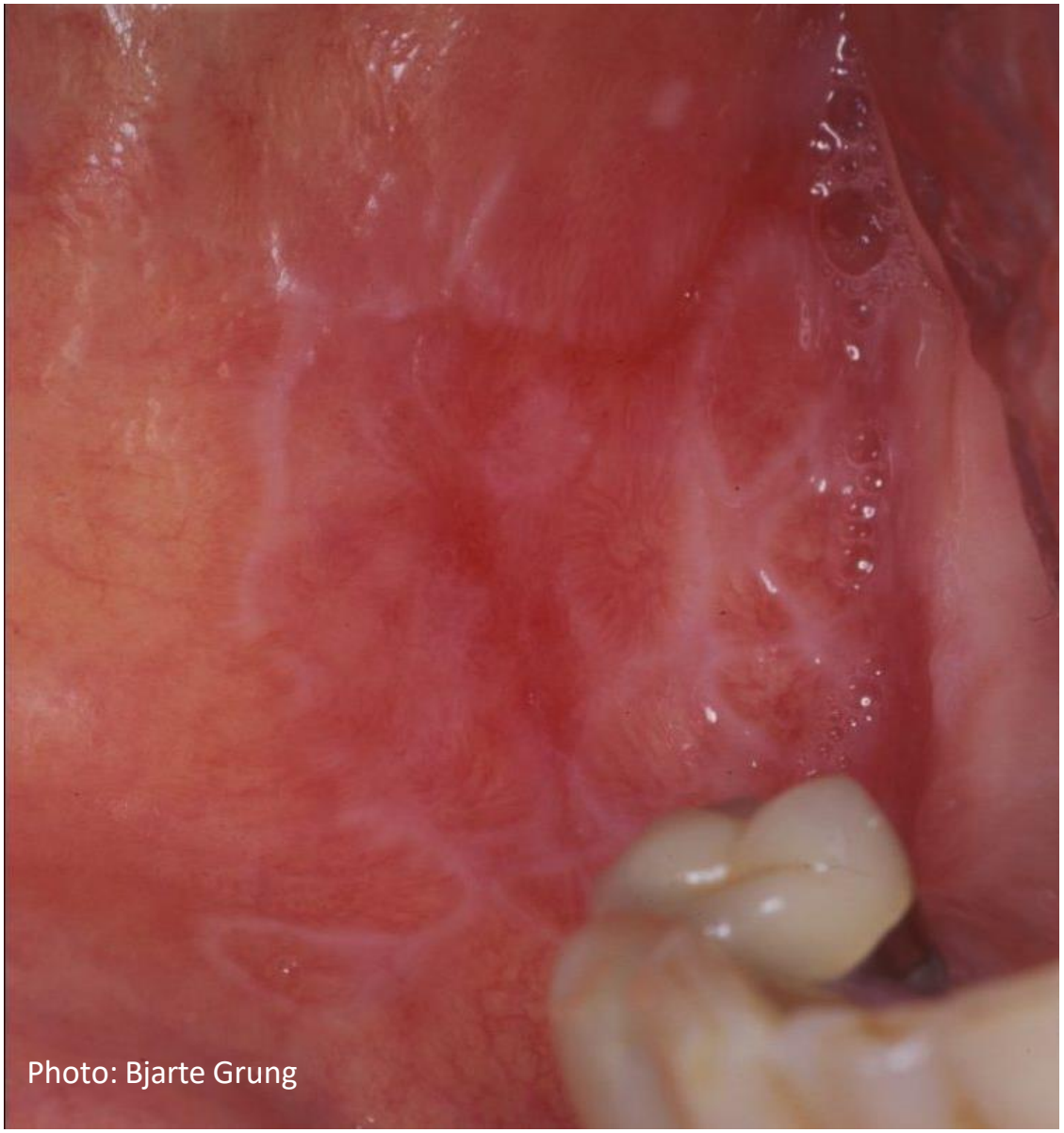


Photo: Bjarte Grung

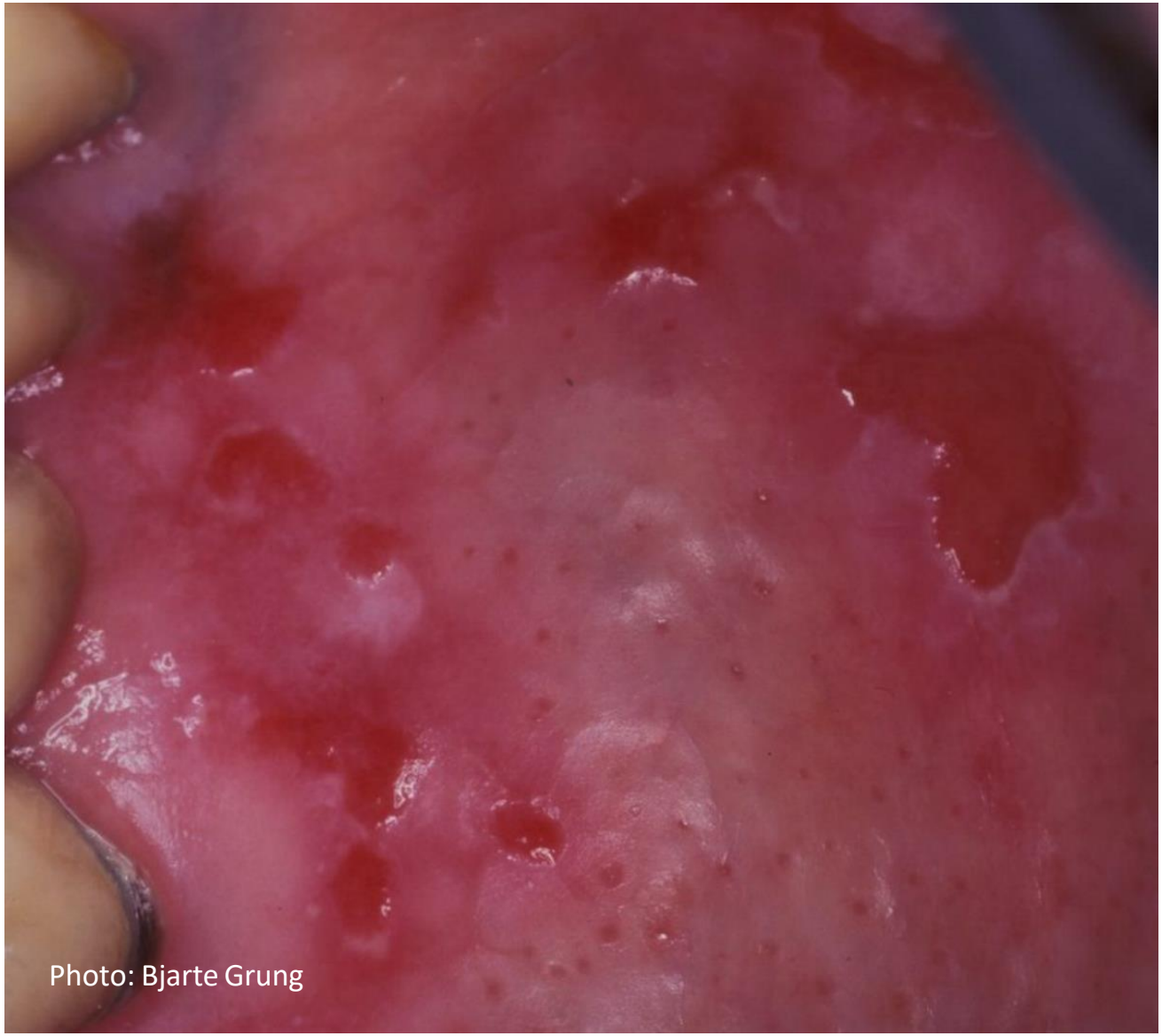
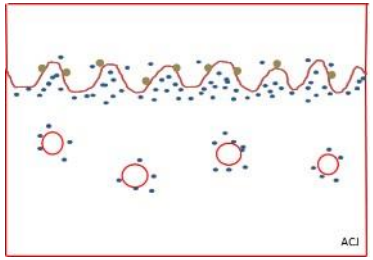
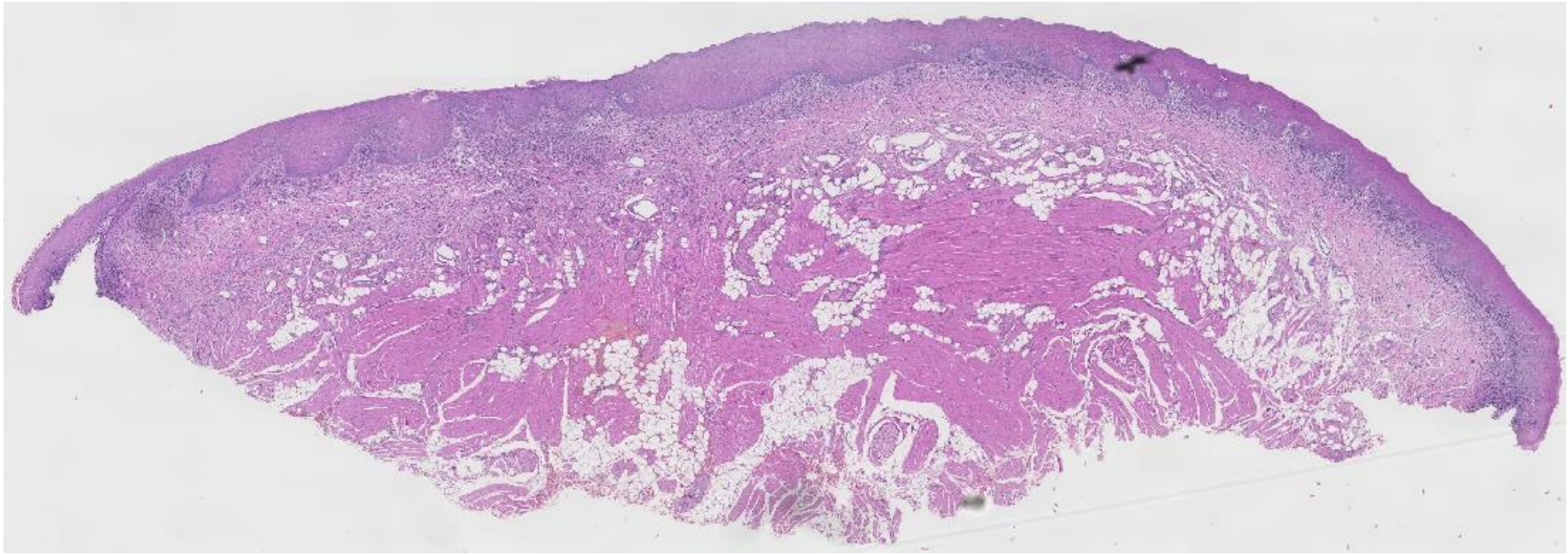
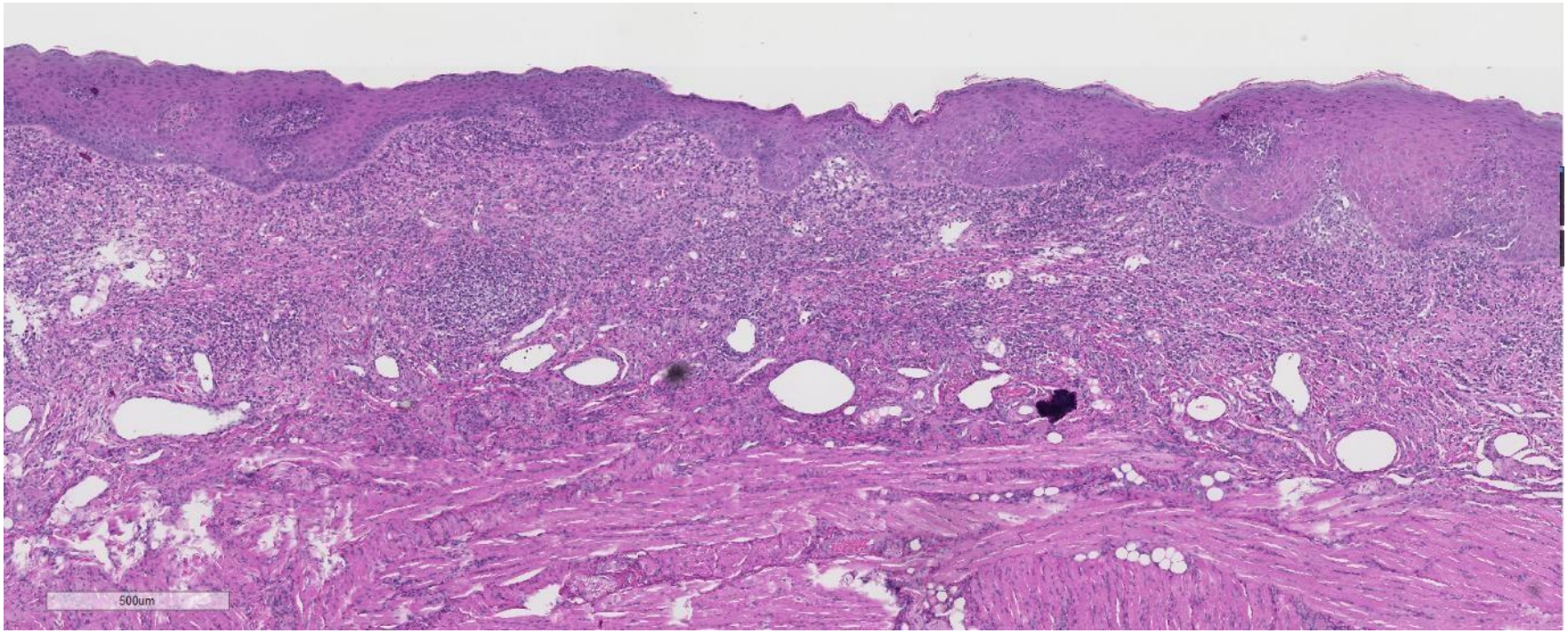


Photo: Bjarte Grung

Female, 55 years old. Bilaterally on tongue and buccal mucosa, atrophic/erosive areas with thin whitish striae.
TD: Lichen planus



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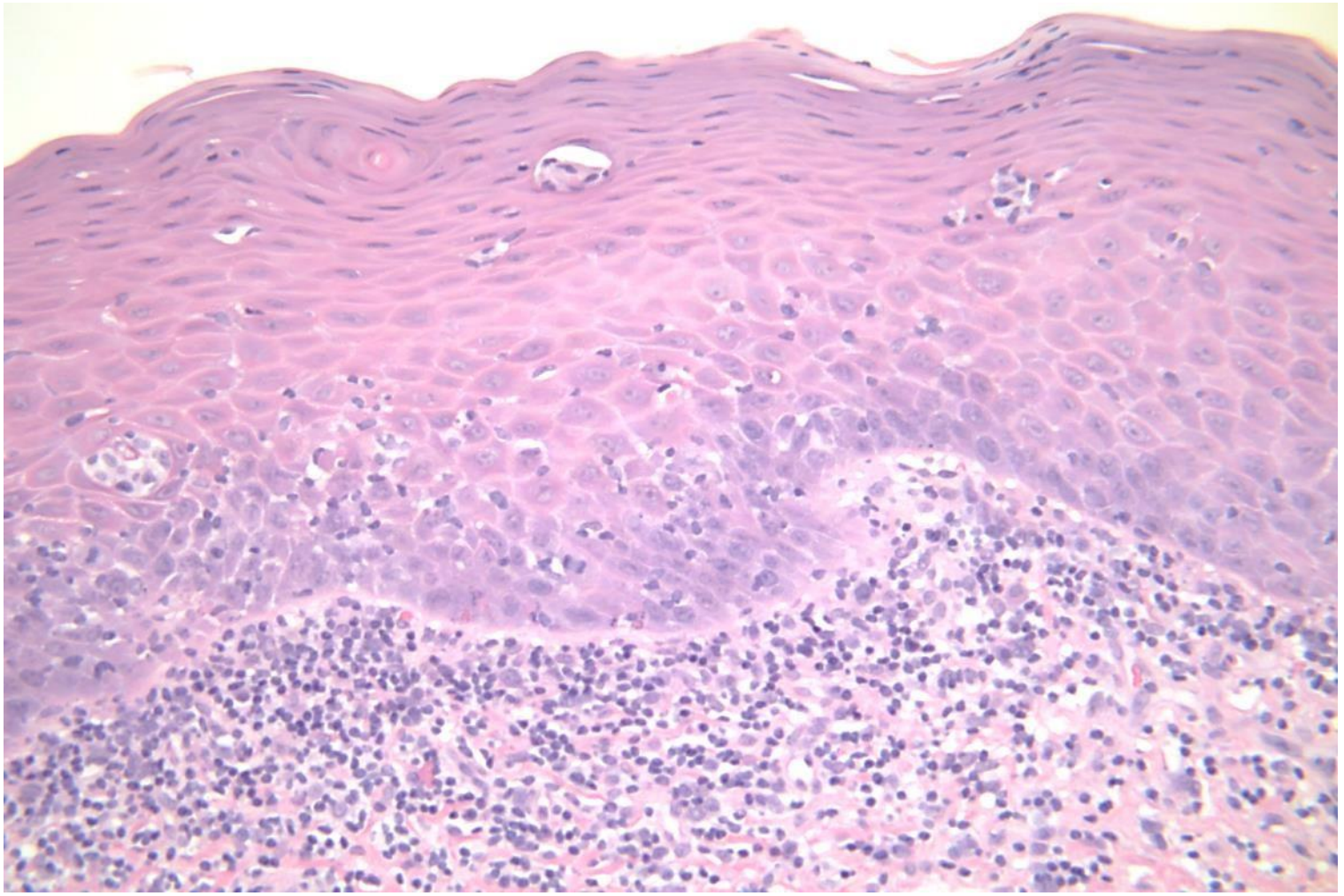


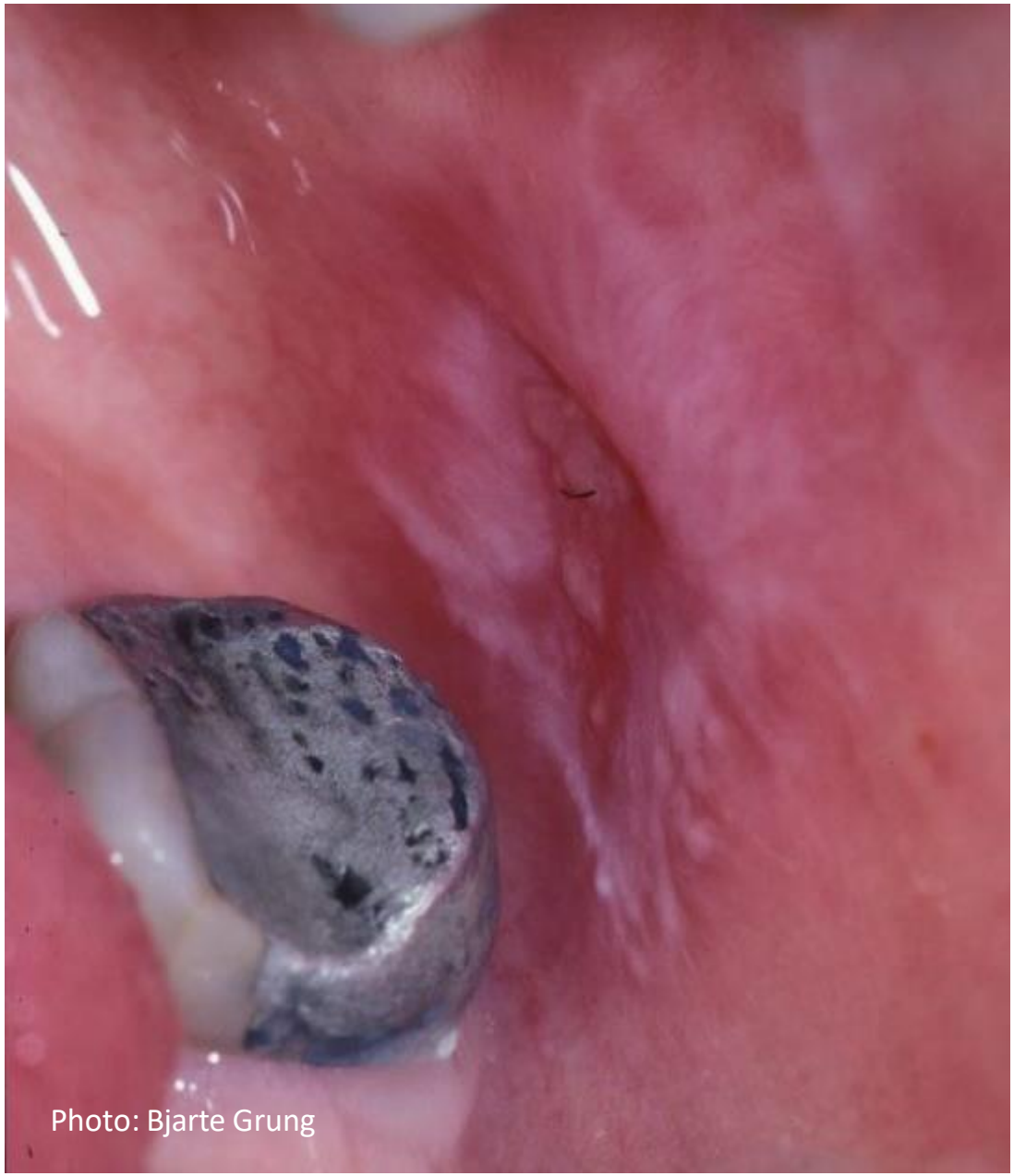
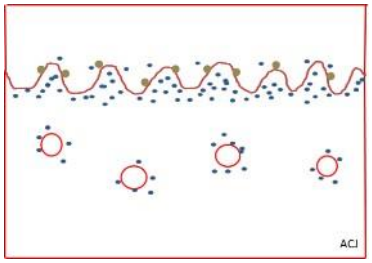
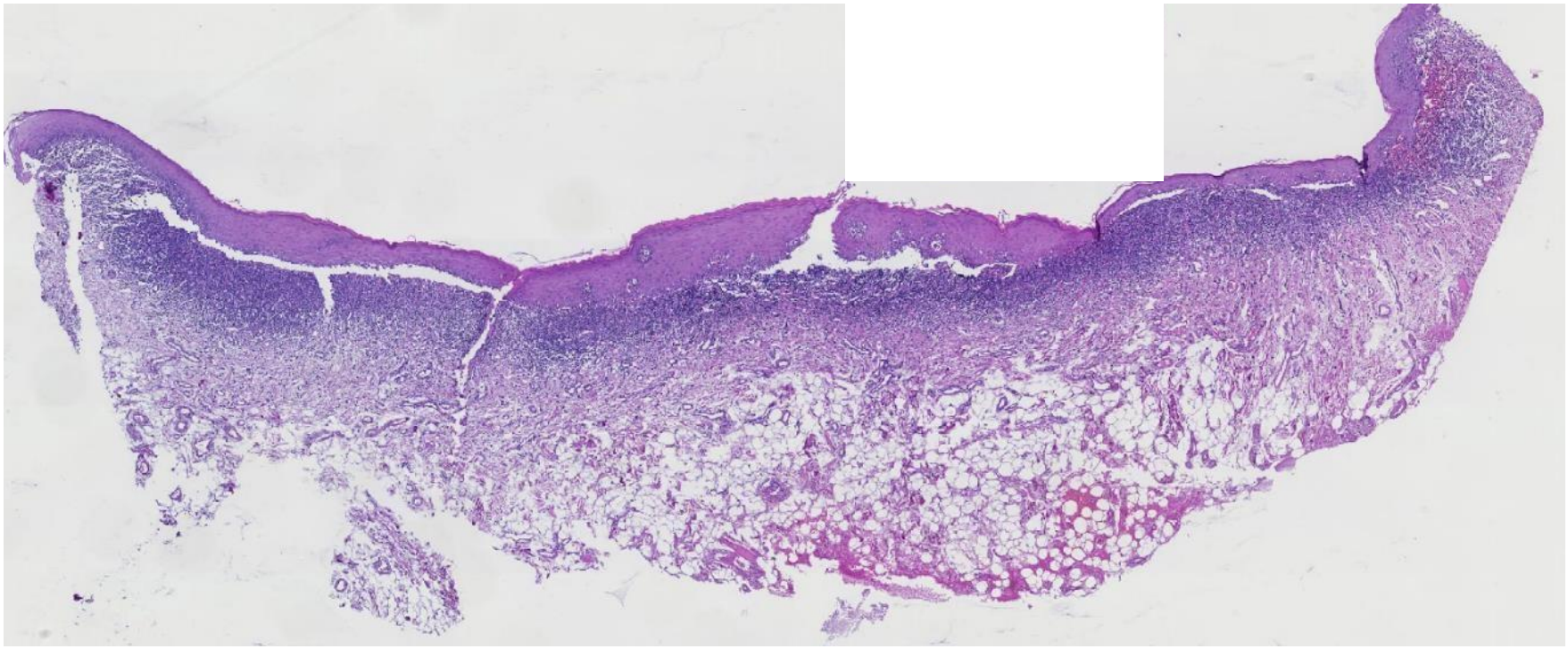
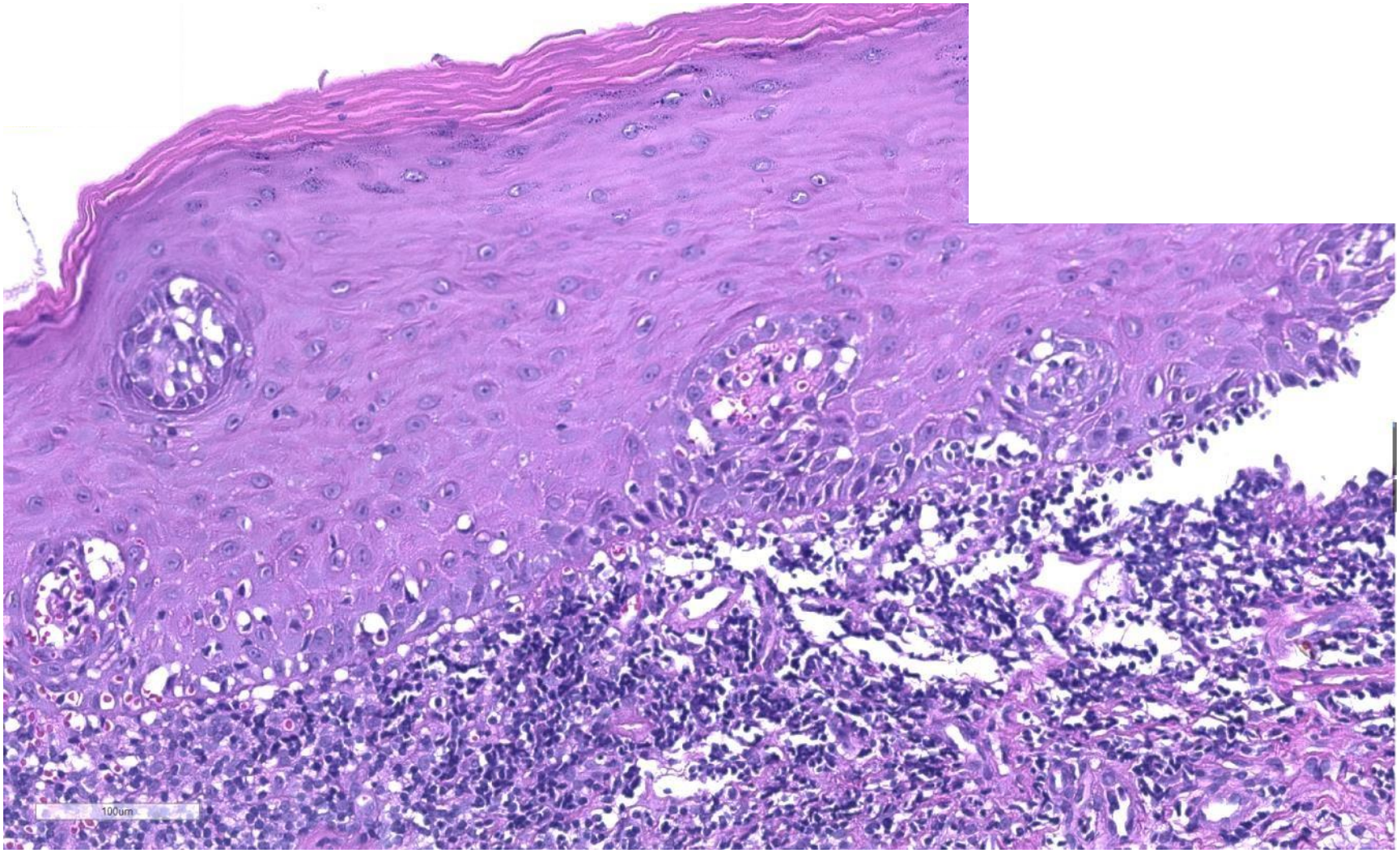


Photo: Bjarte Grung

Female, 83 years old. Whitish lesions in gingiva and buccal mucosa, buccal to molars right side. No ulceration.
TD: Lichenoid lesion



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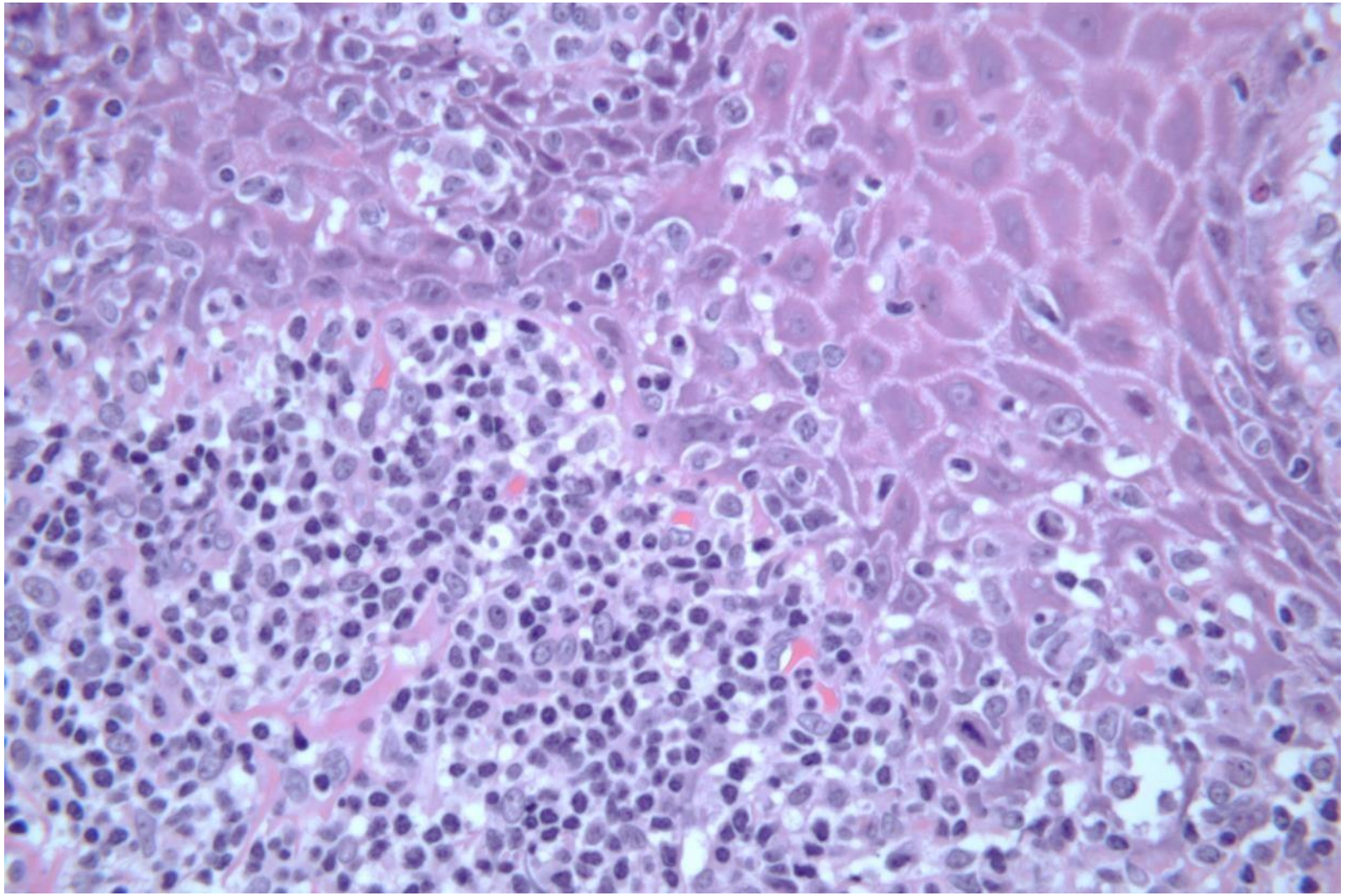
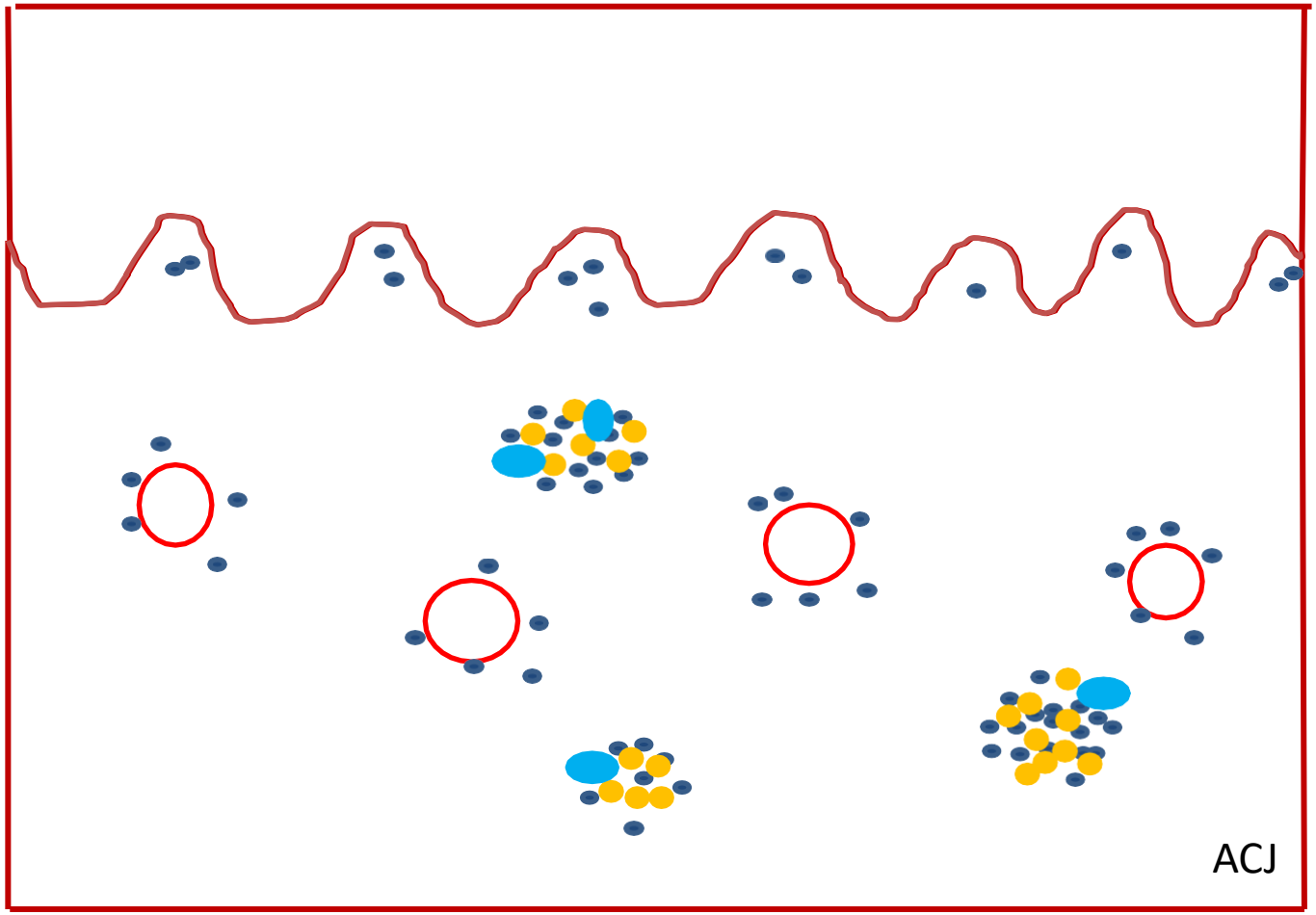


Granulomatous inflammation



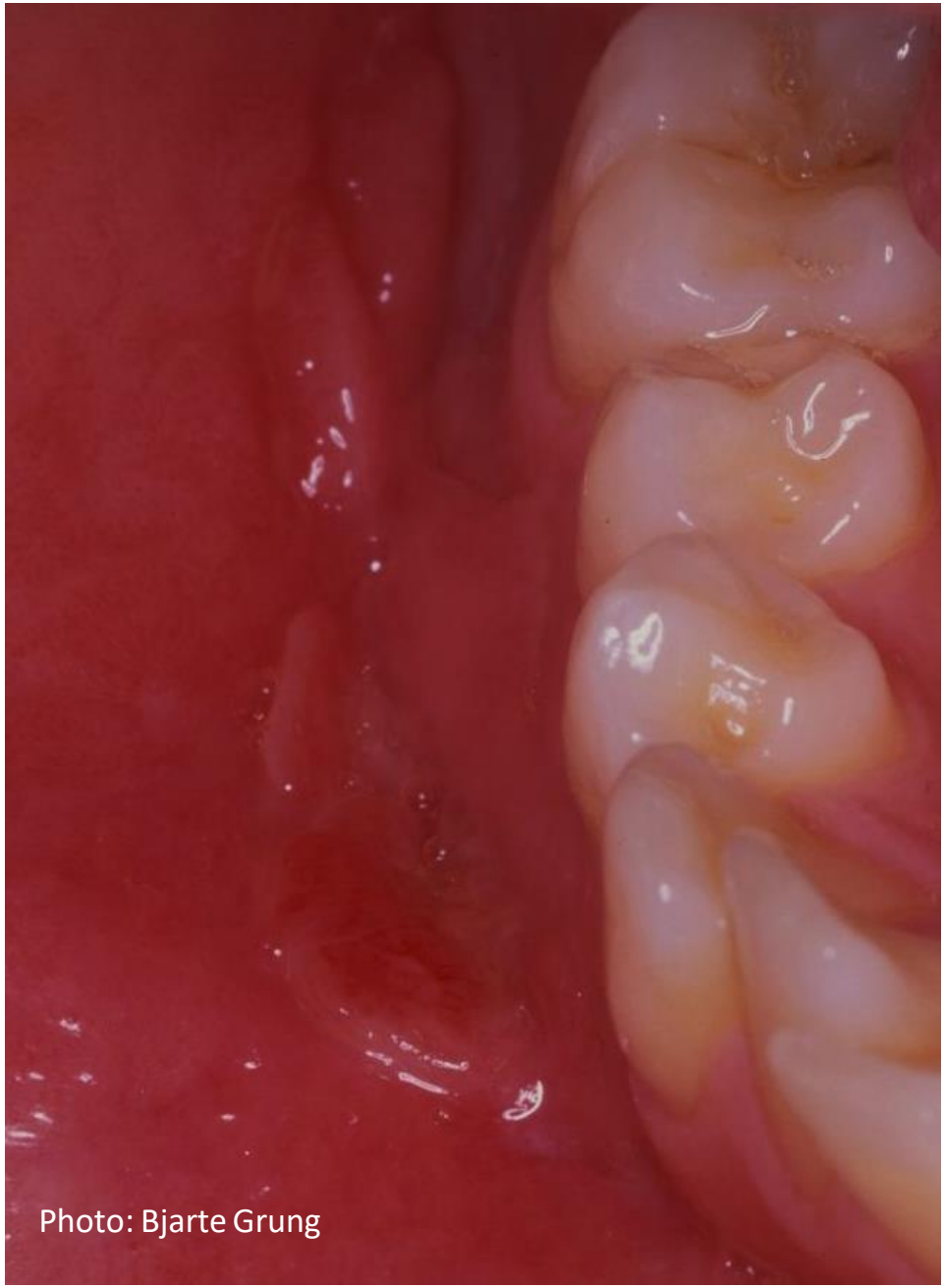
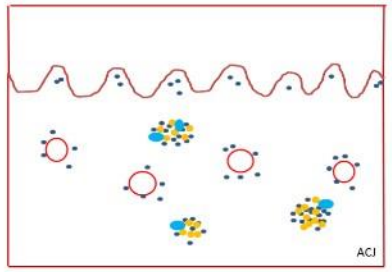
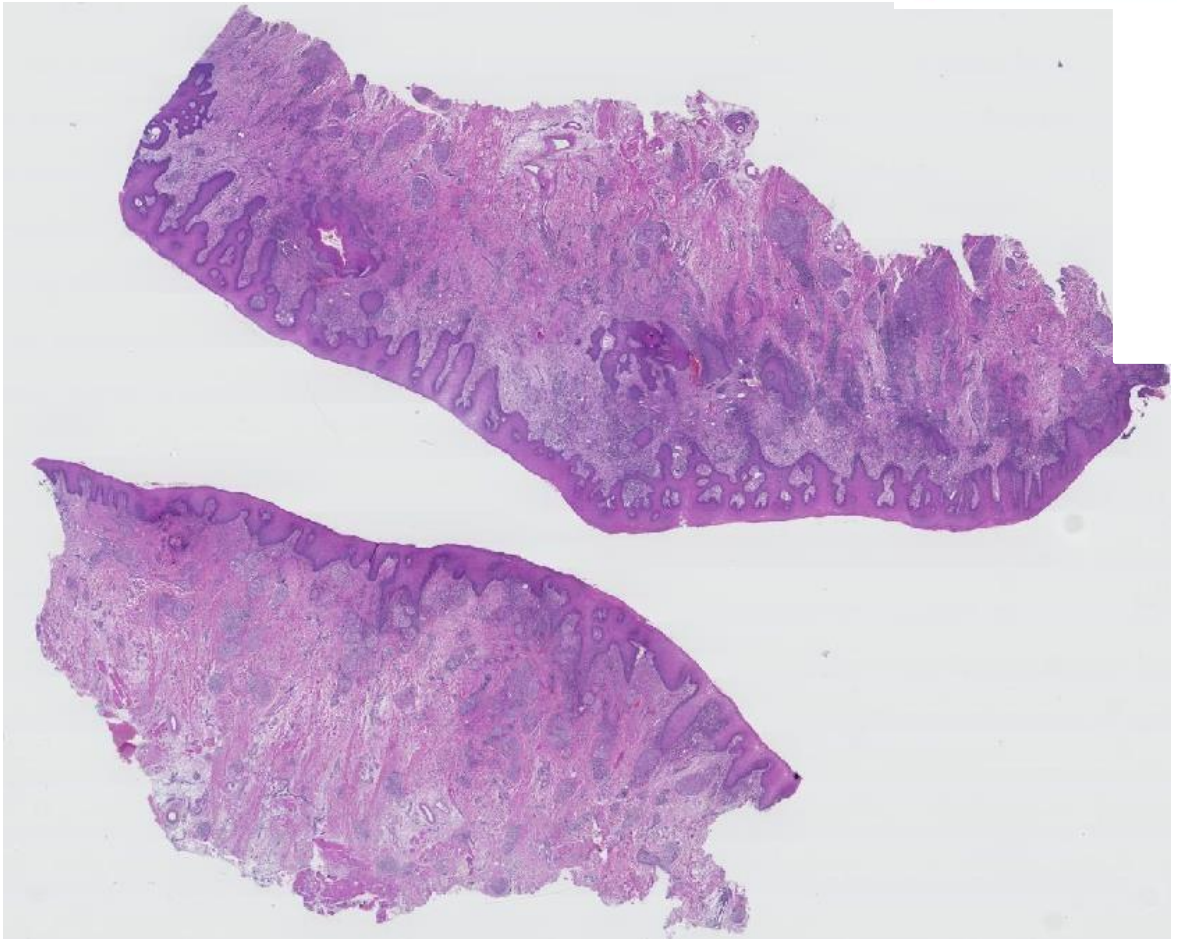
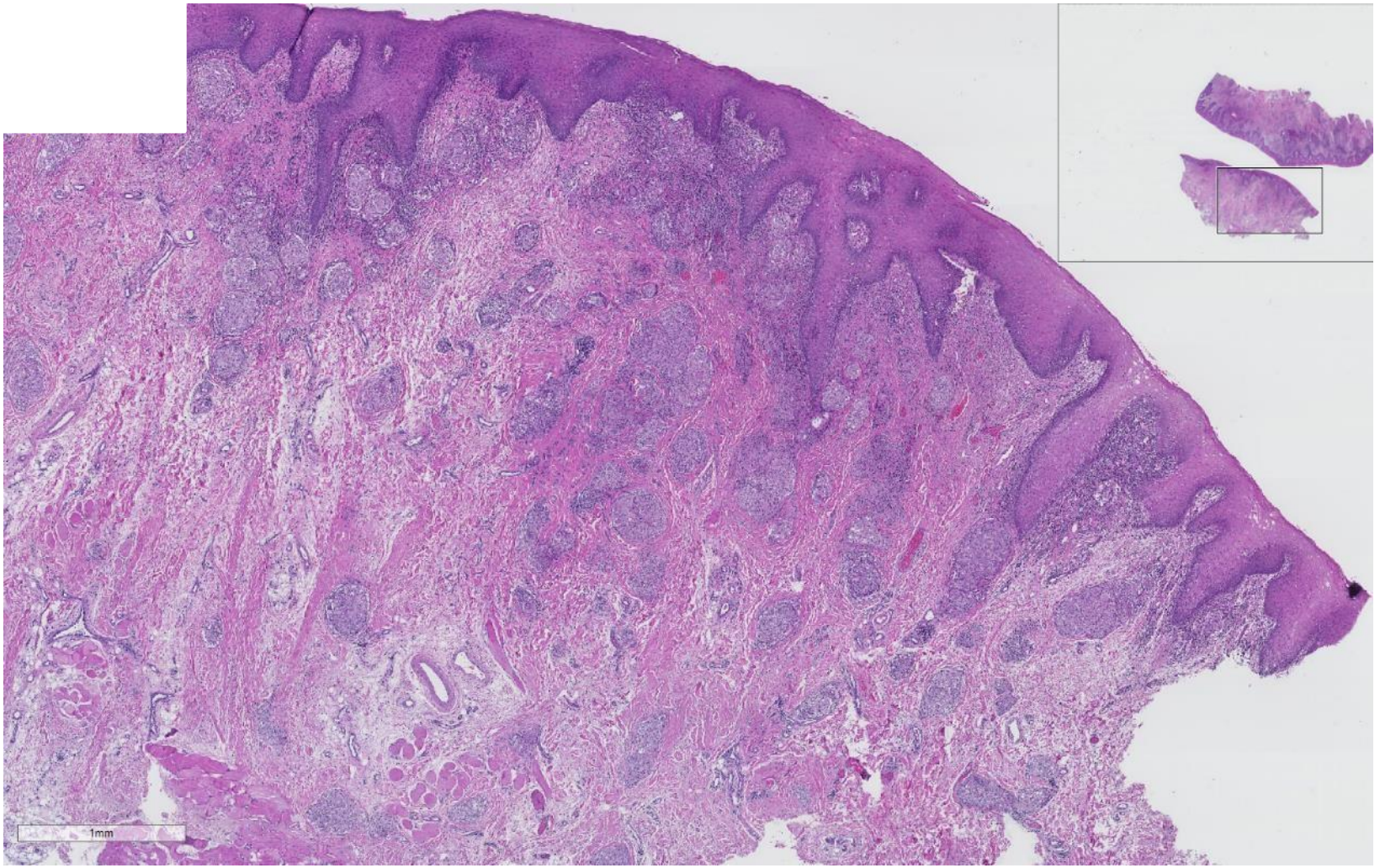


Photo: Bjarte Grung

Boy, 14 years old. Diagnosed with Morbus Crohn. Sore exophytic ulcers bilaterally in the oral cavity.
Clinically compatible with Mb Crohn.



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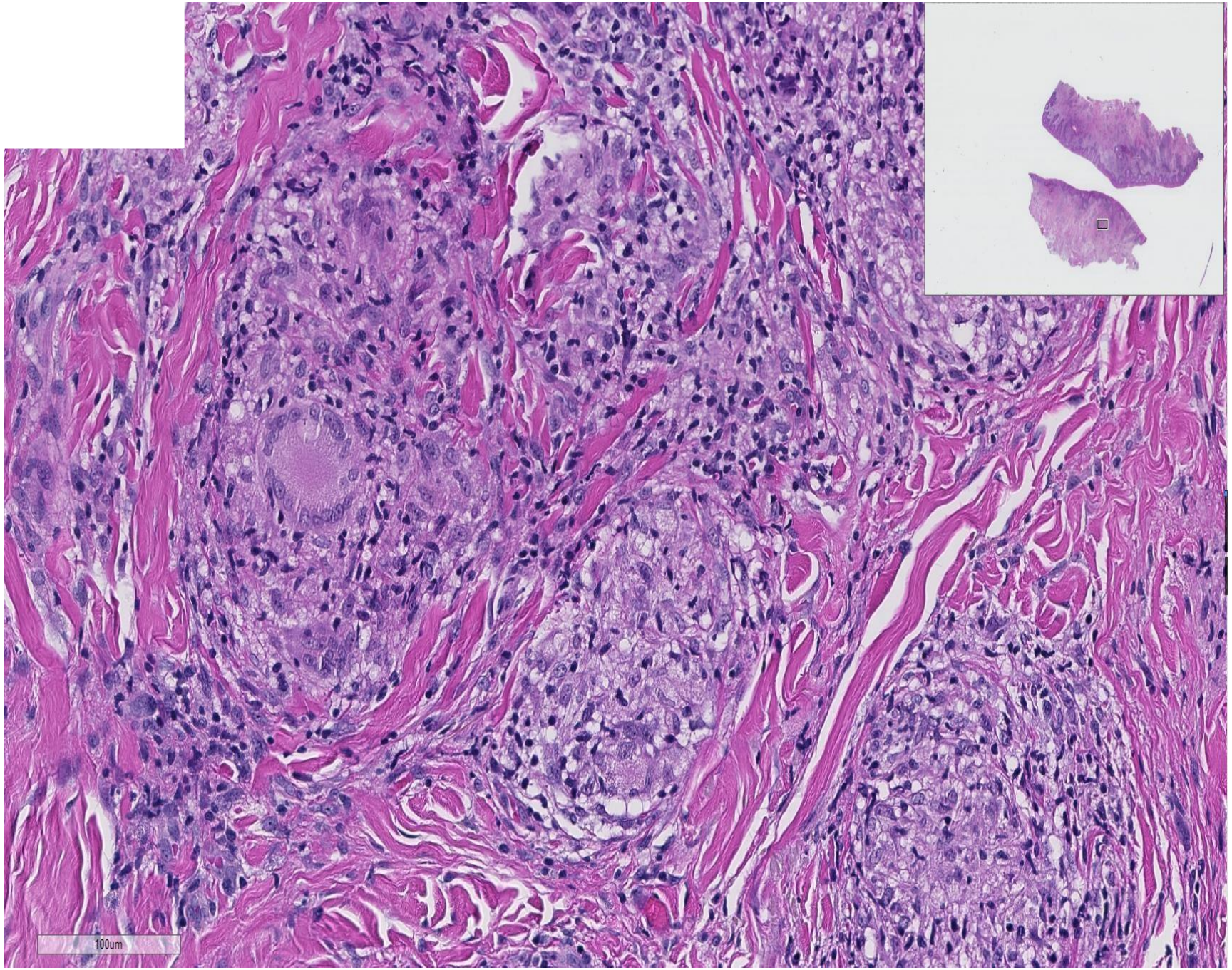
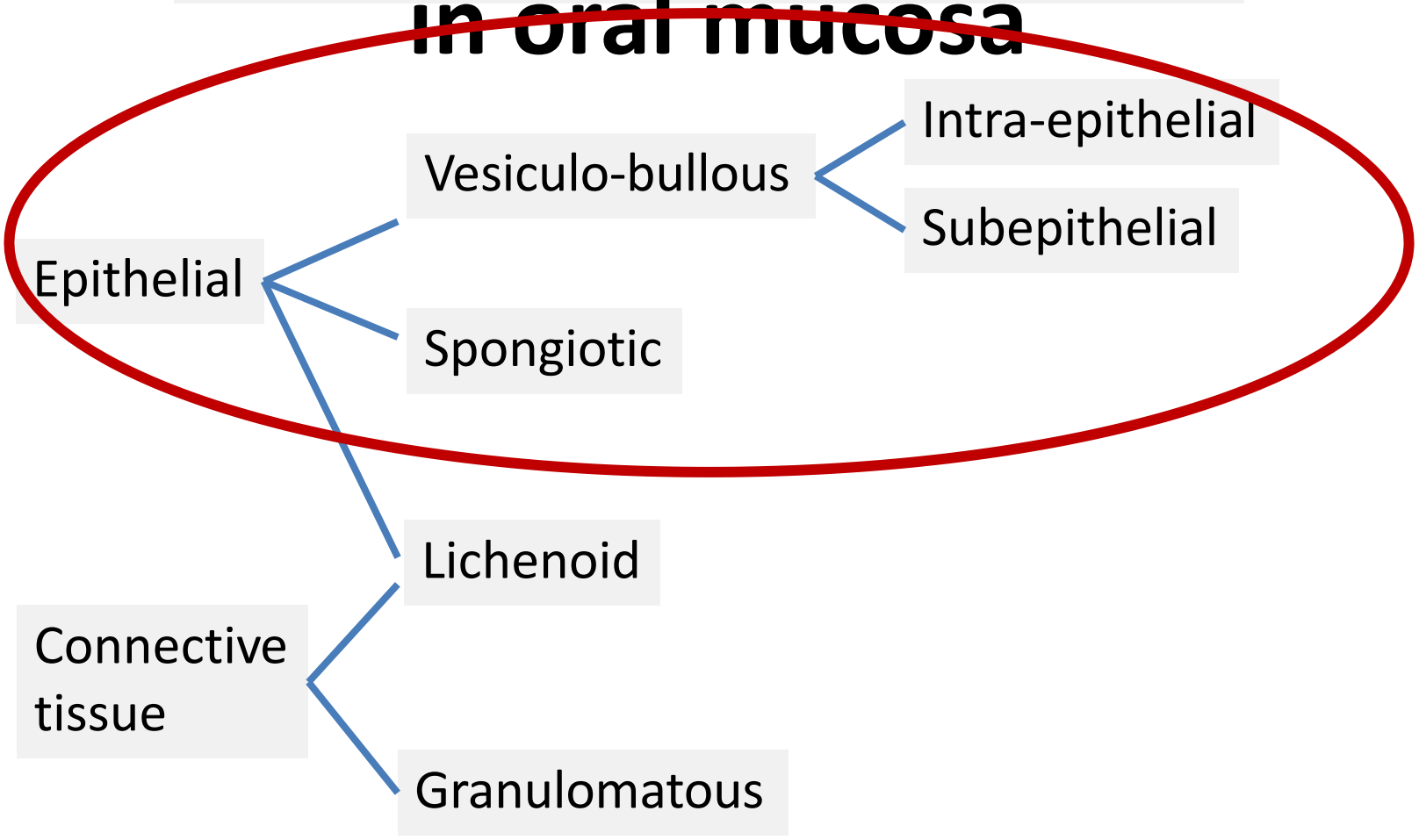

100um

Inflammatory diseases in oral mucosa



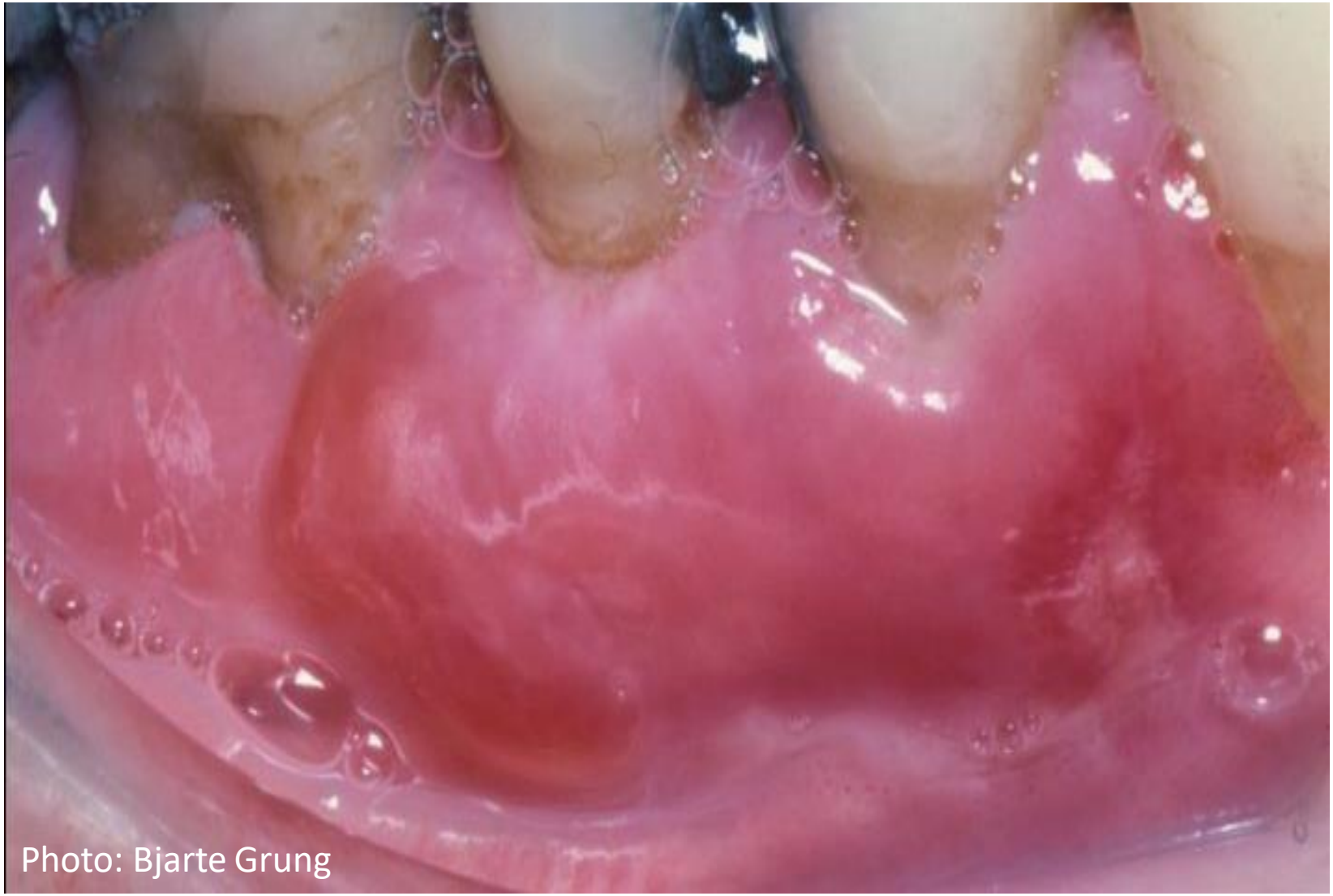


Photo: Bjarte Grung

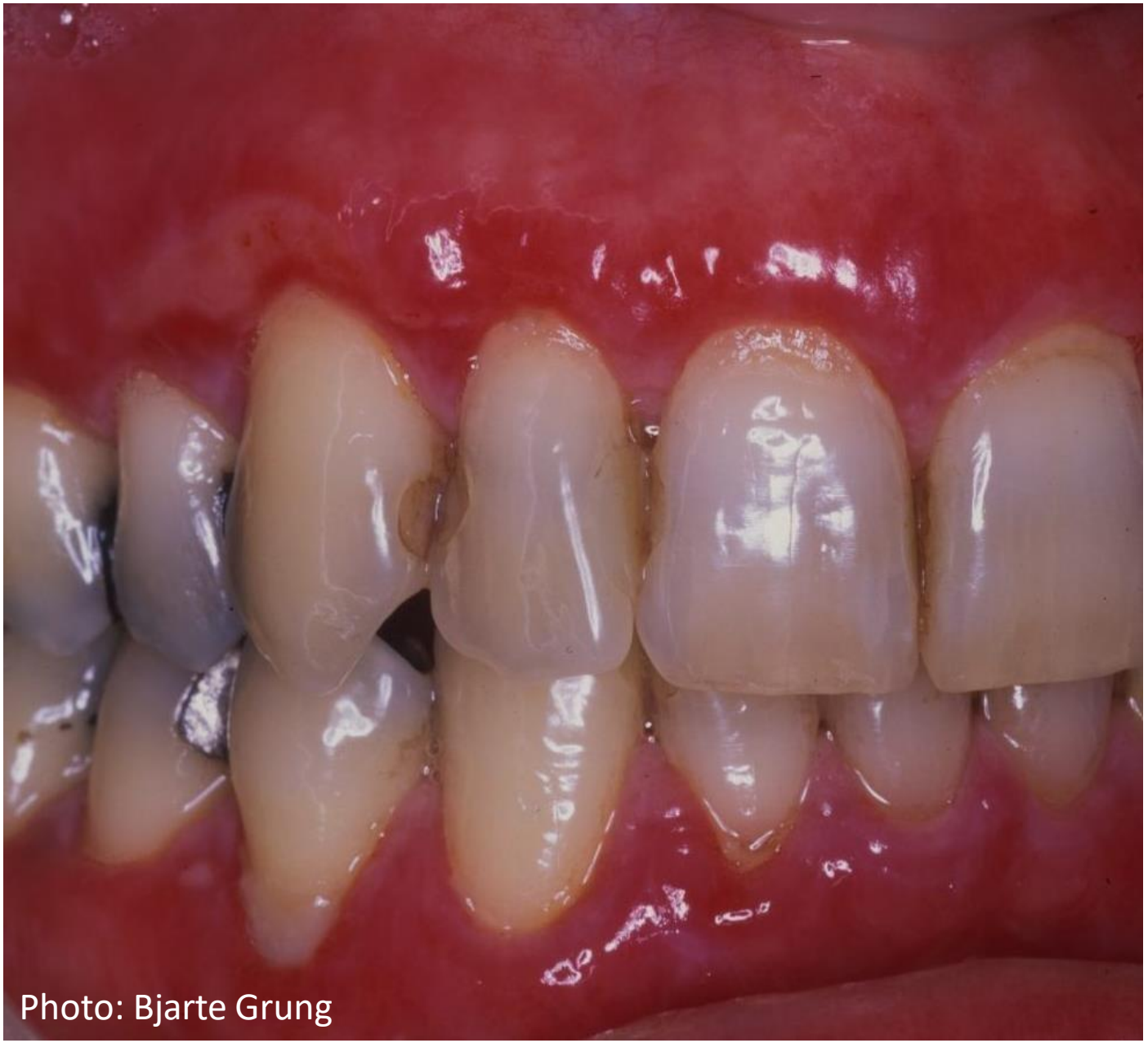
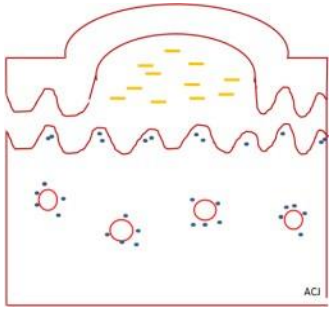
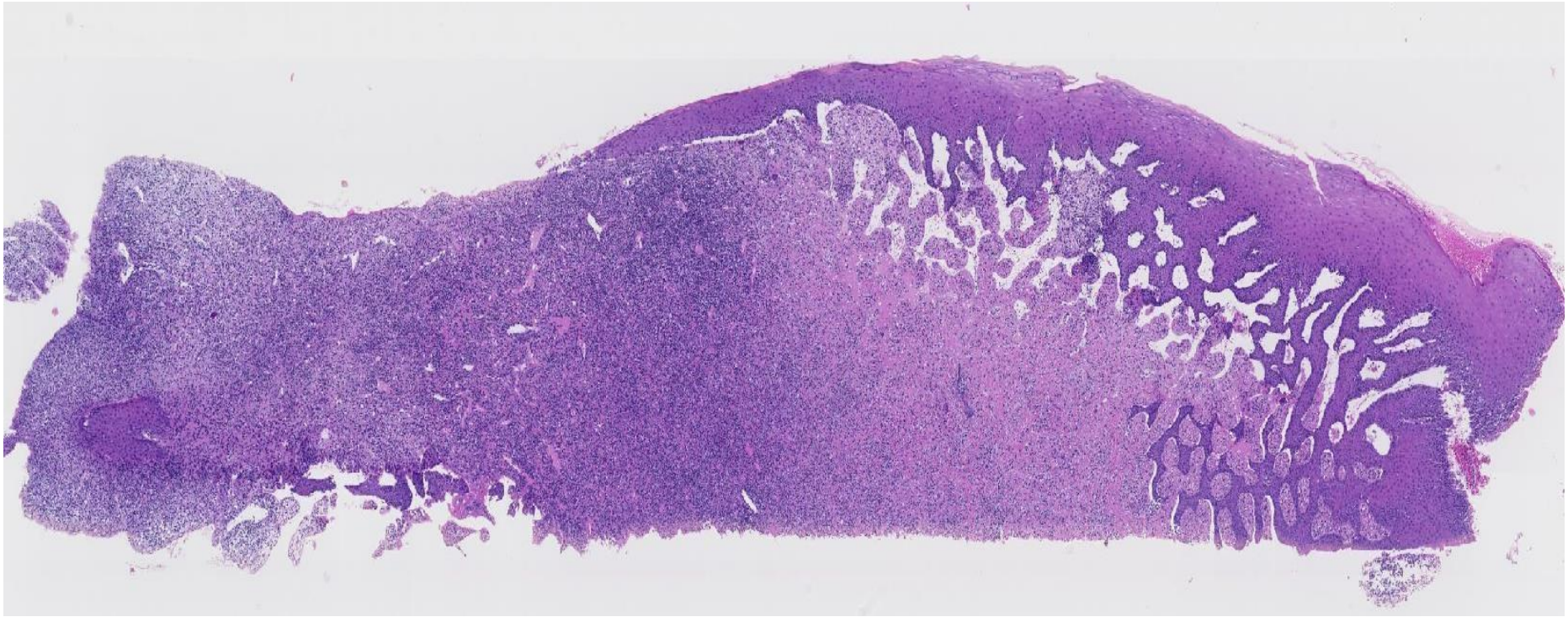
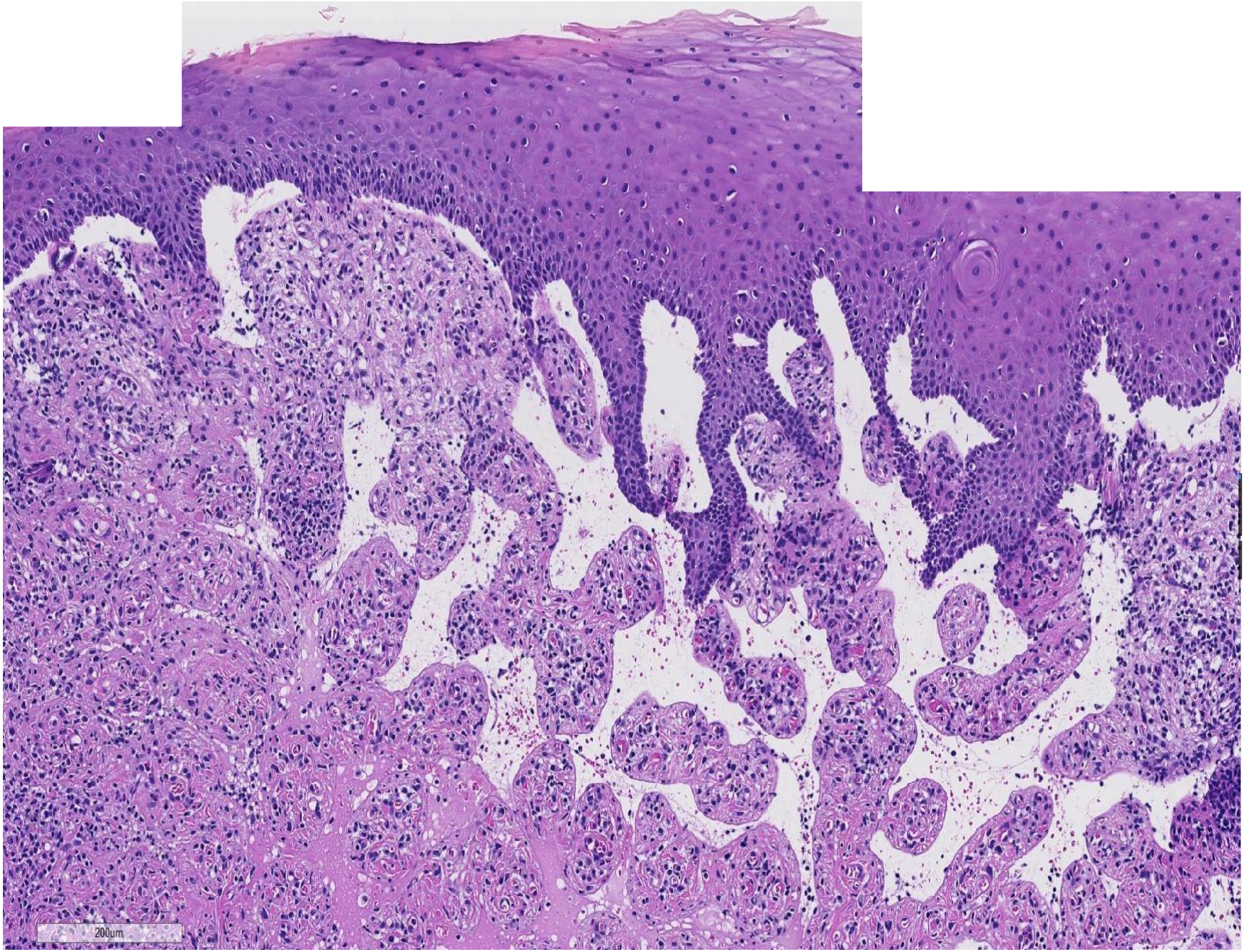


Photo: Bjarte Grung

Female, 67 years. Bullous rash, located to flexors and face/neck. Painful. Heals with crusts.
Ora mucosal enanthema, mainly related to attached gingiva. Looks like oral mucosa pemphigoid. Positive Nicolsky's sign. (Pemphigus?)



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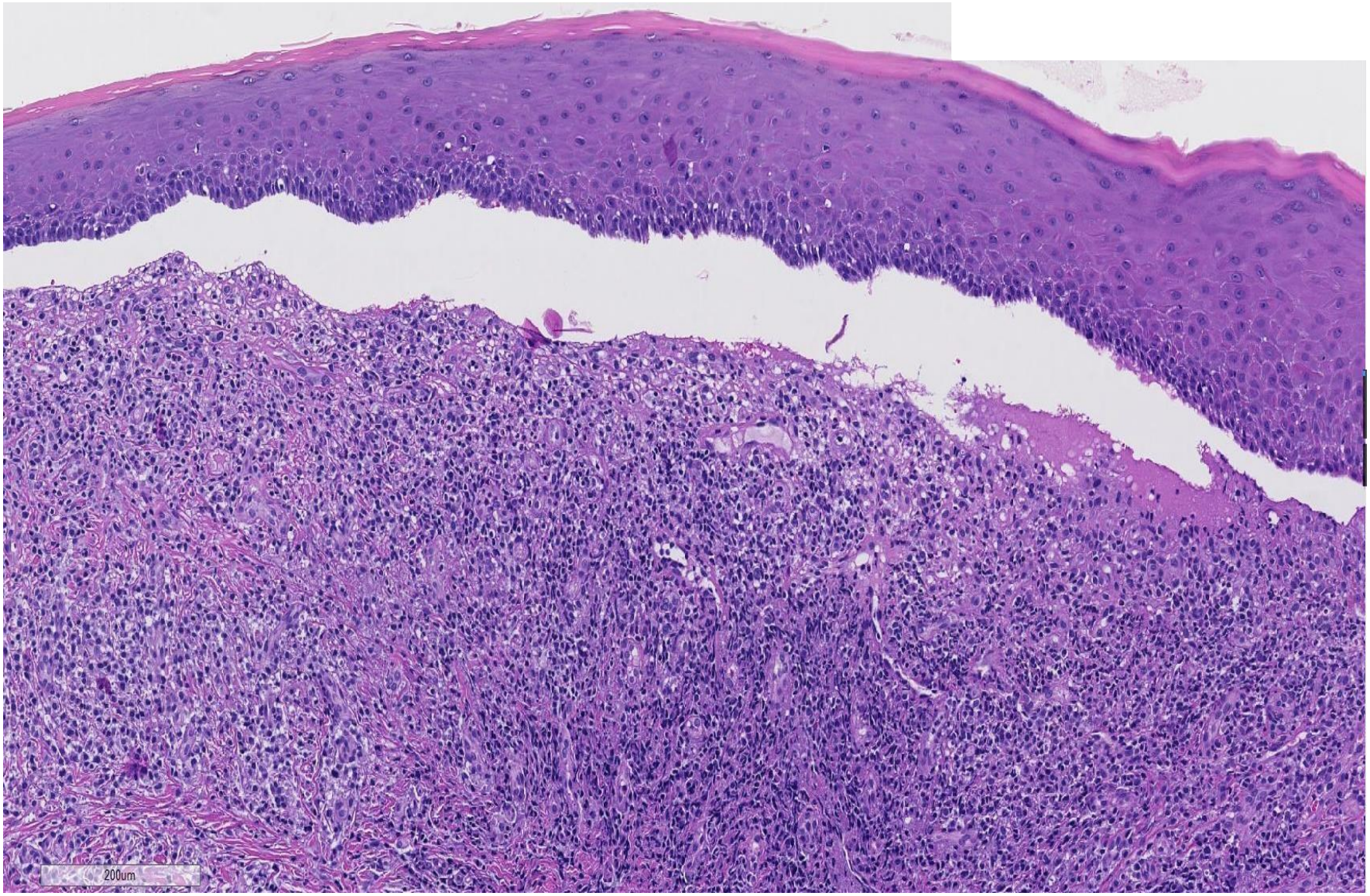
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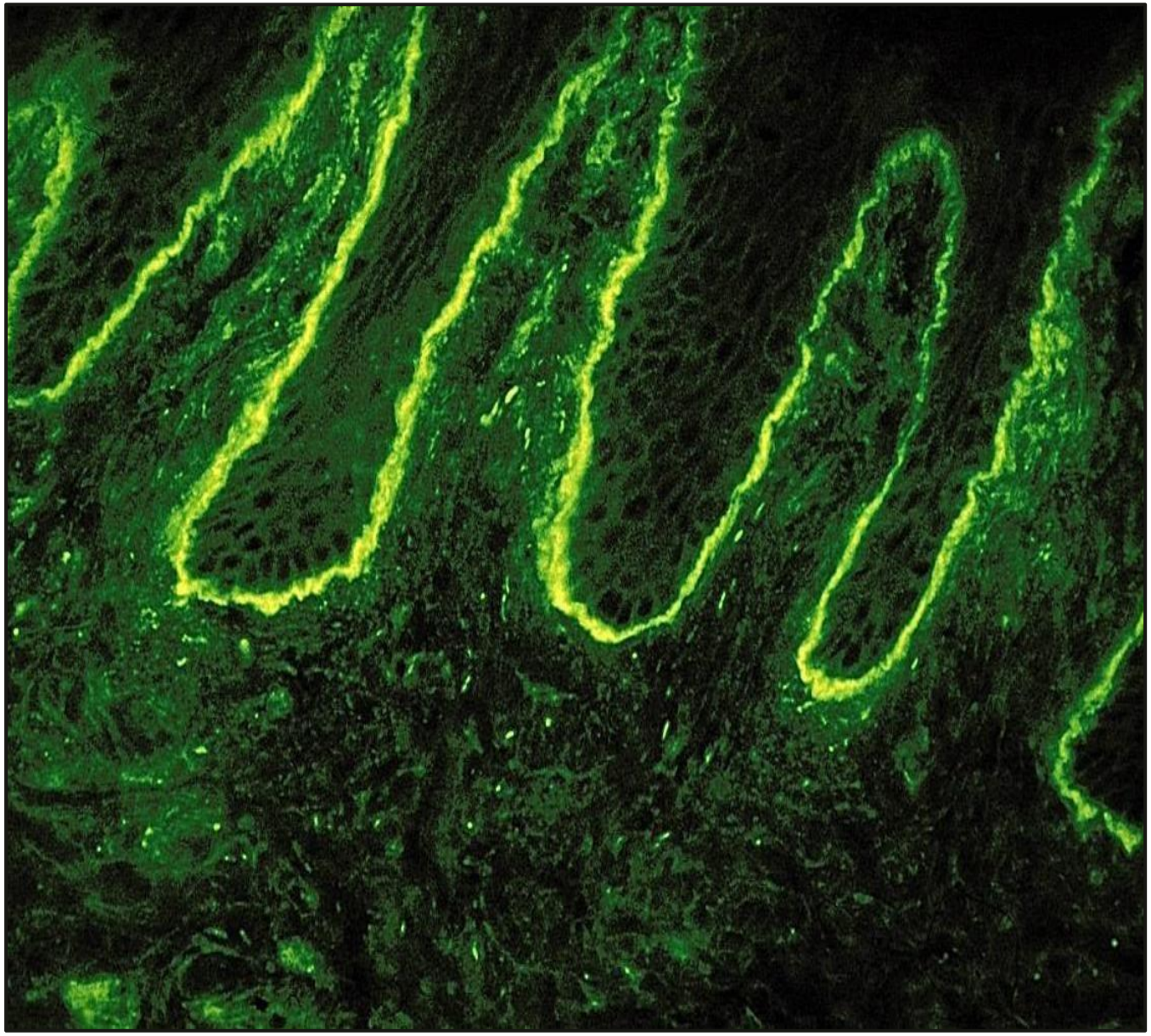


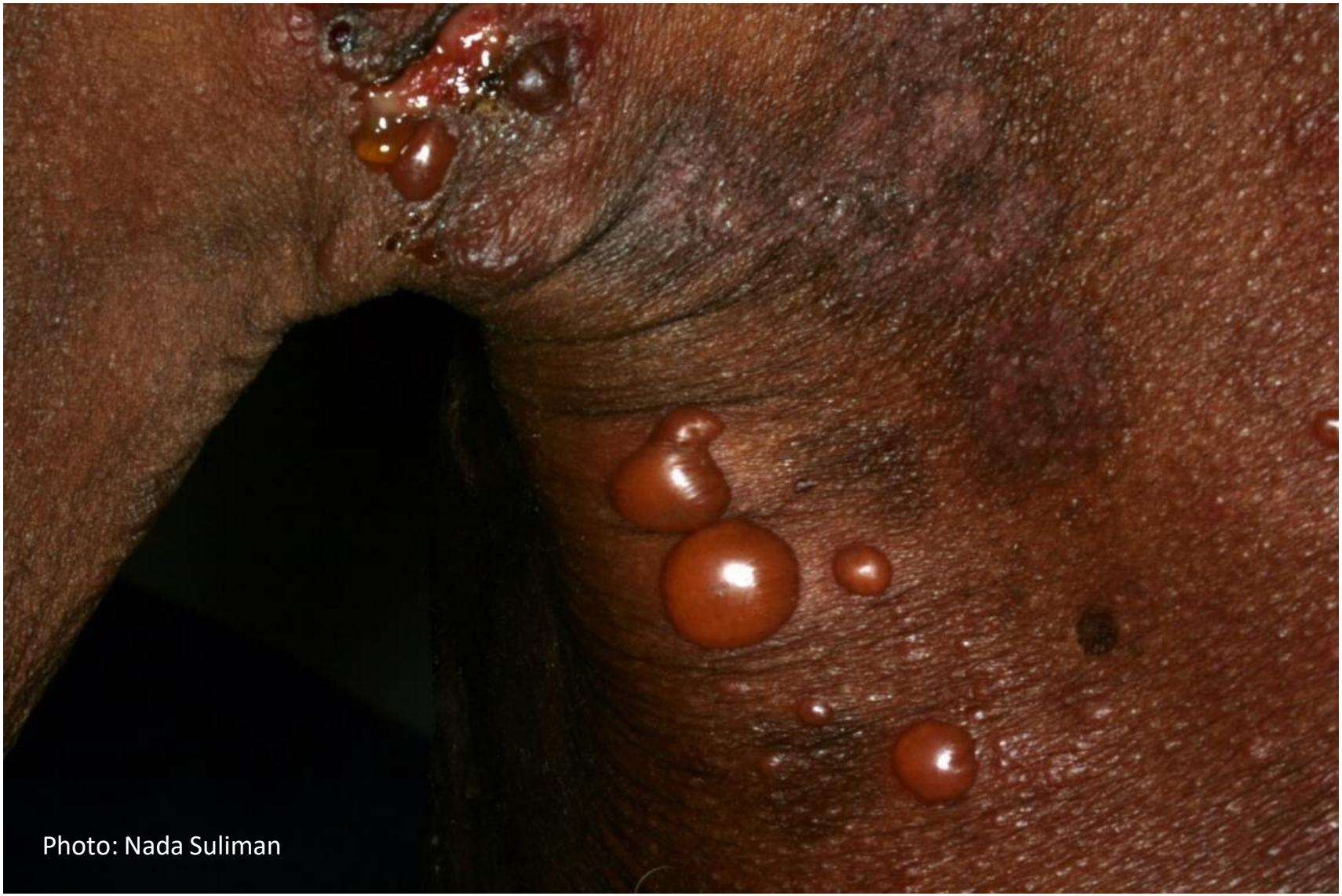
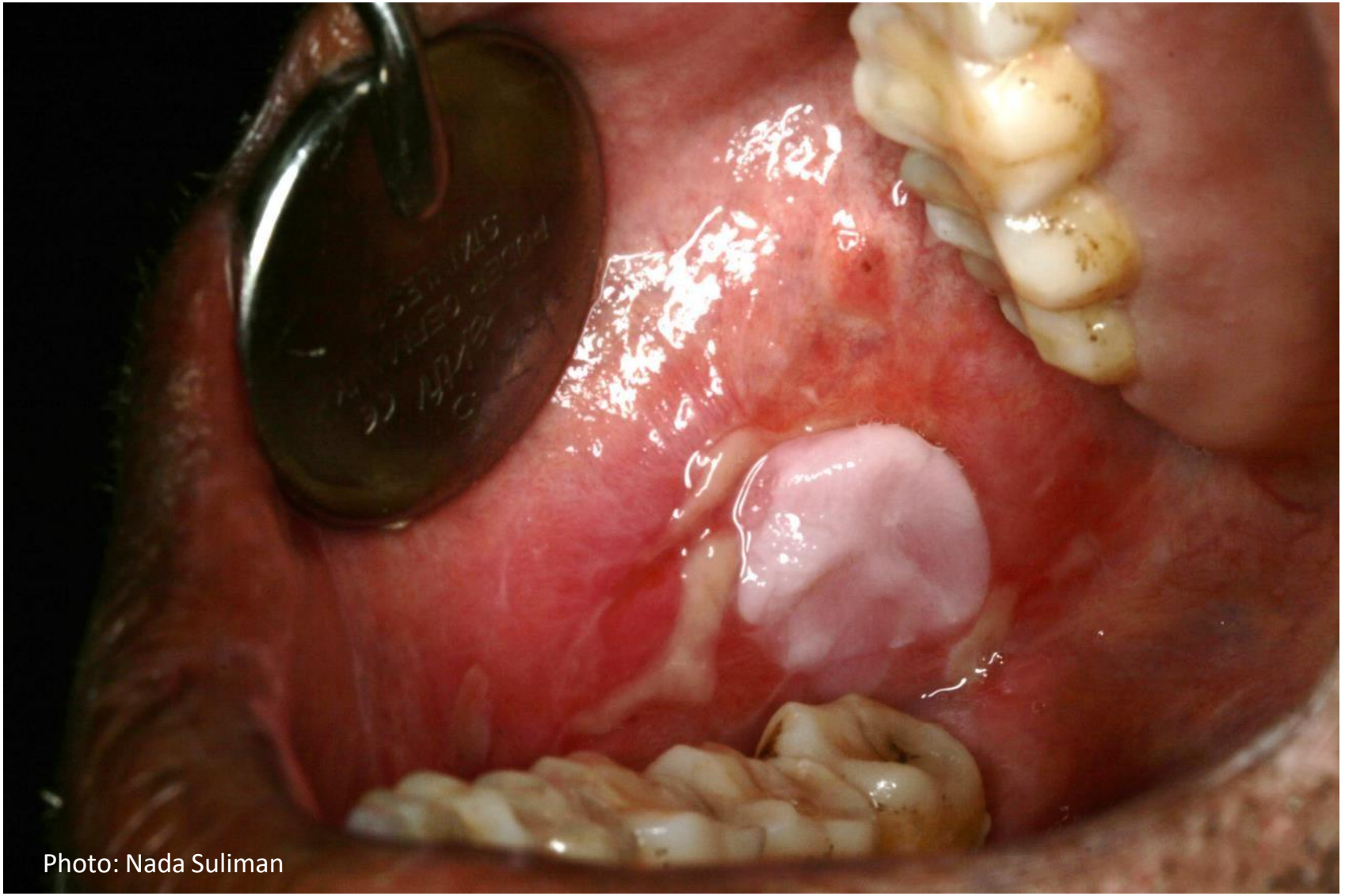


Photo: Nada Suliman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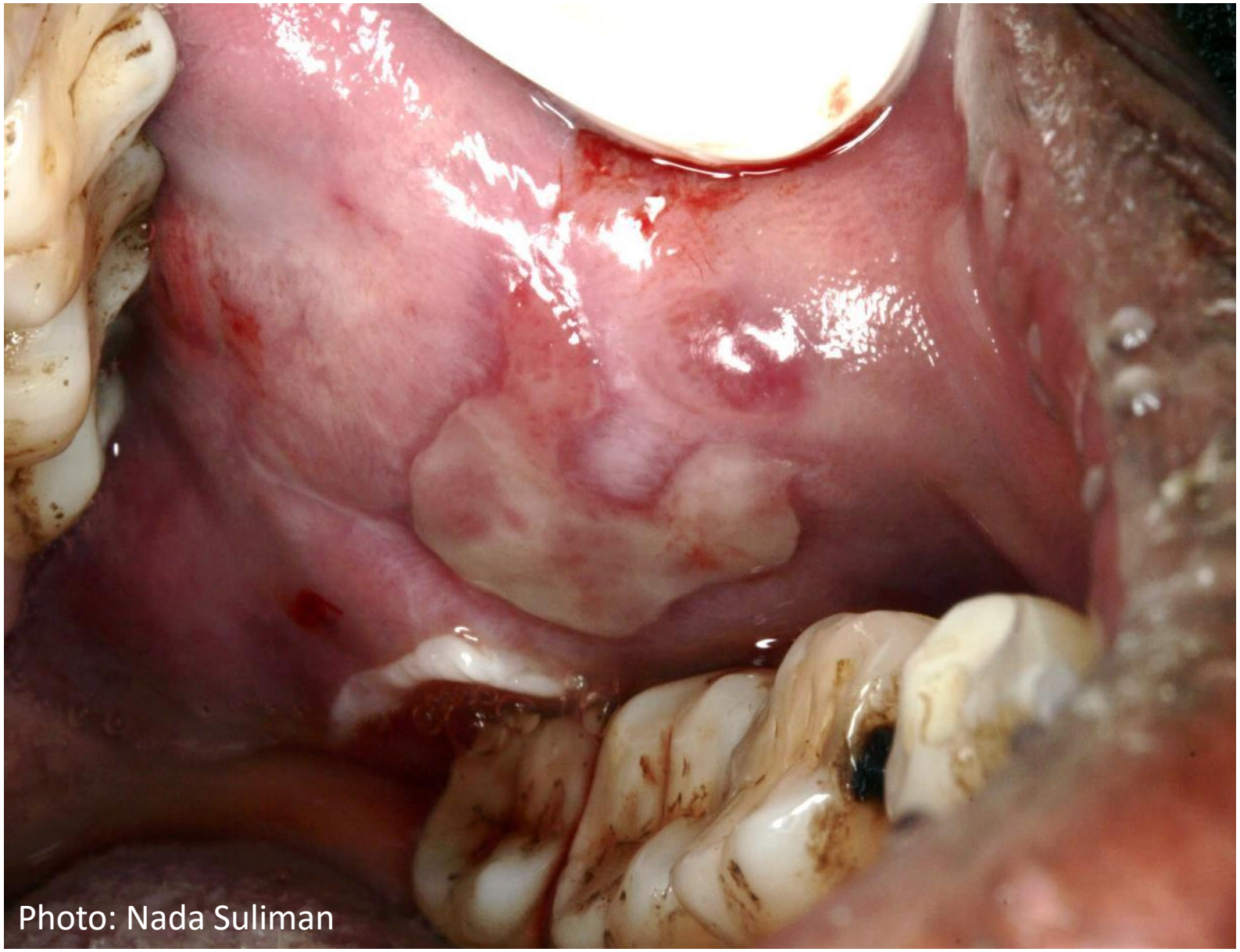
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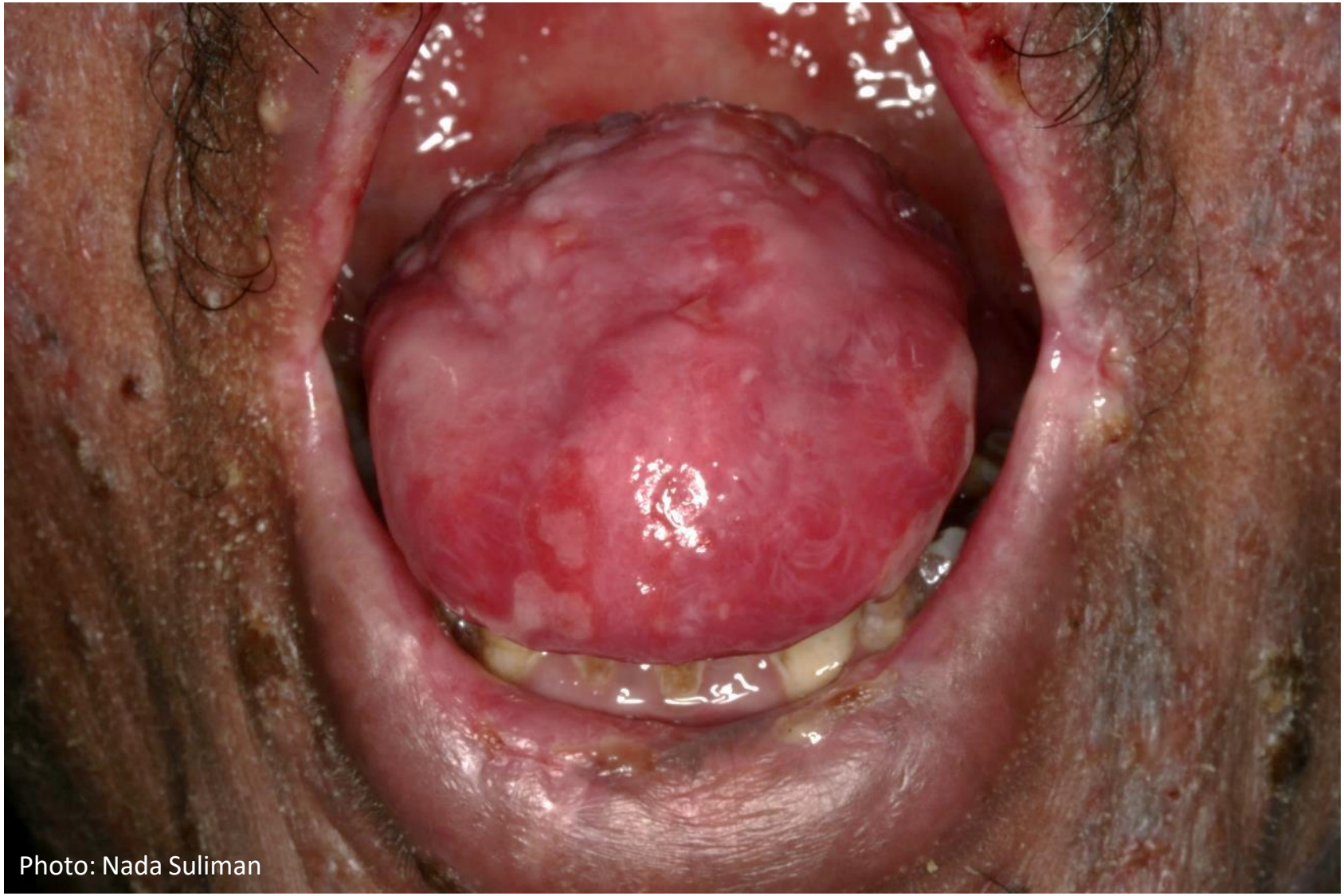
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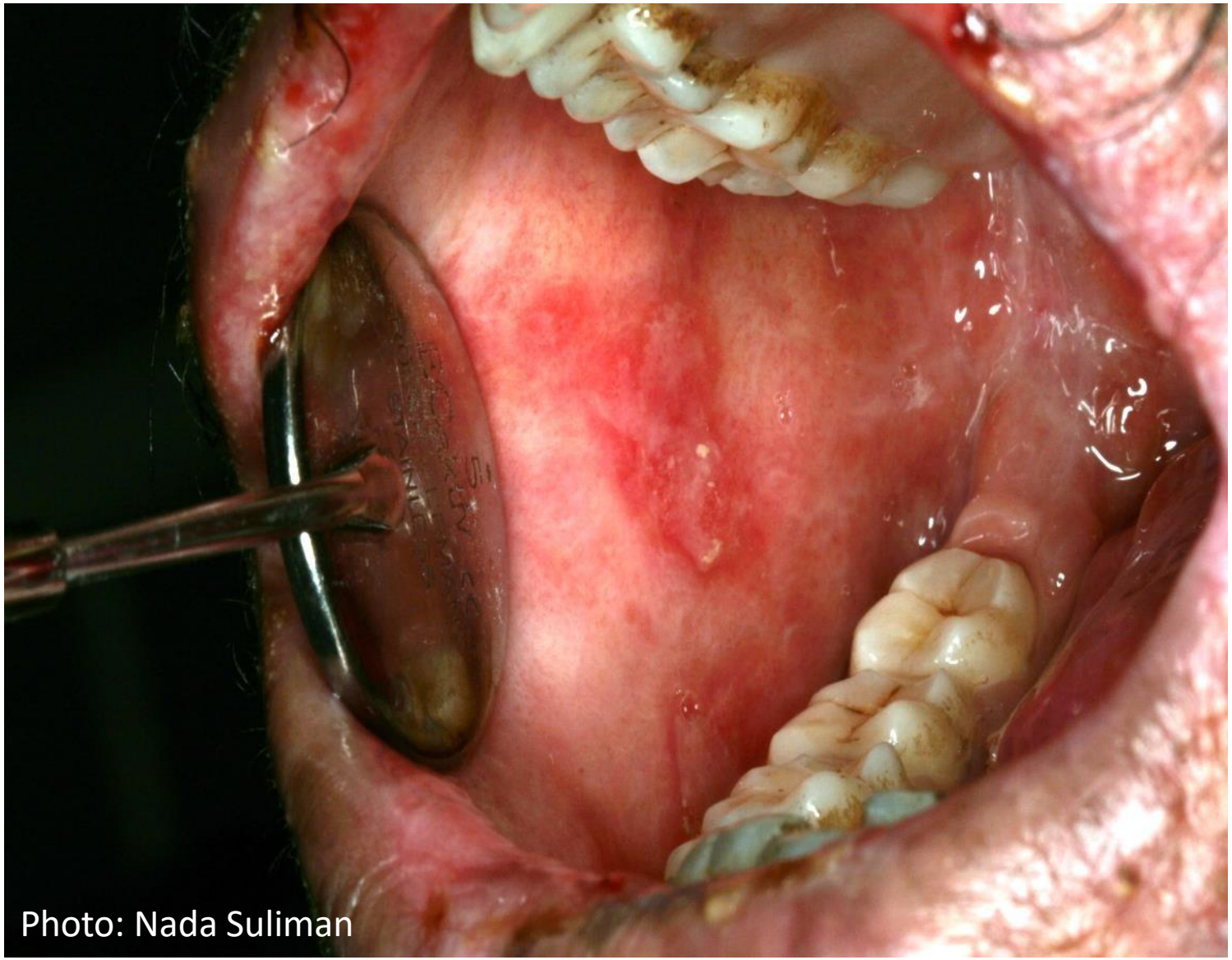
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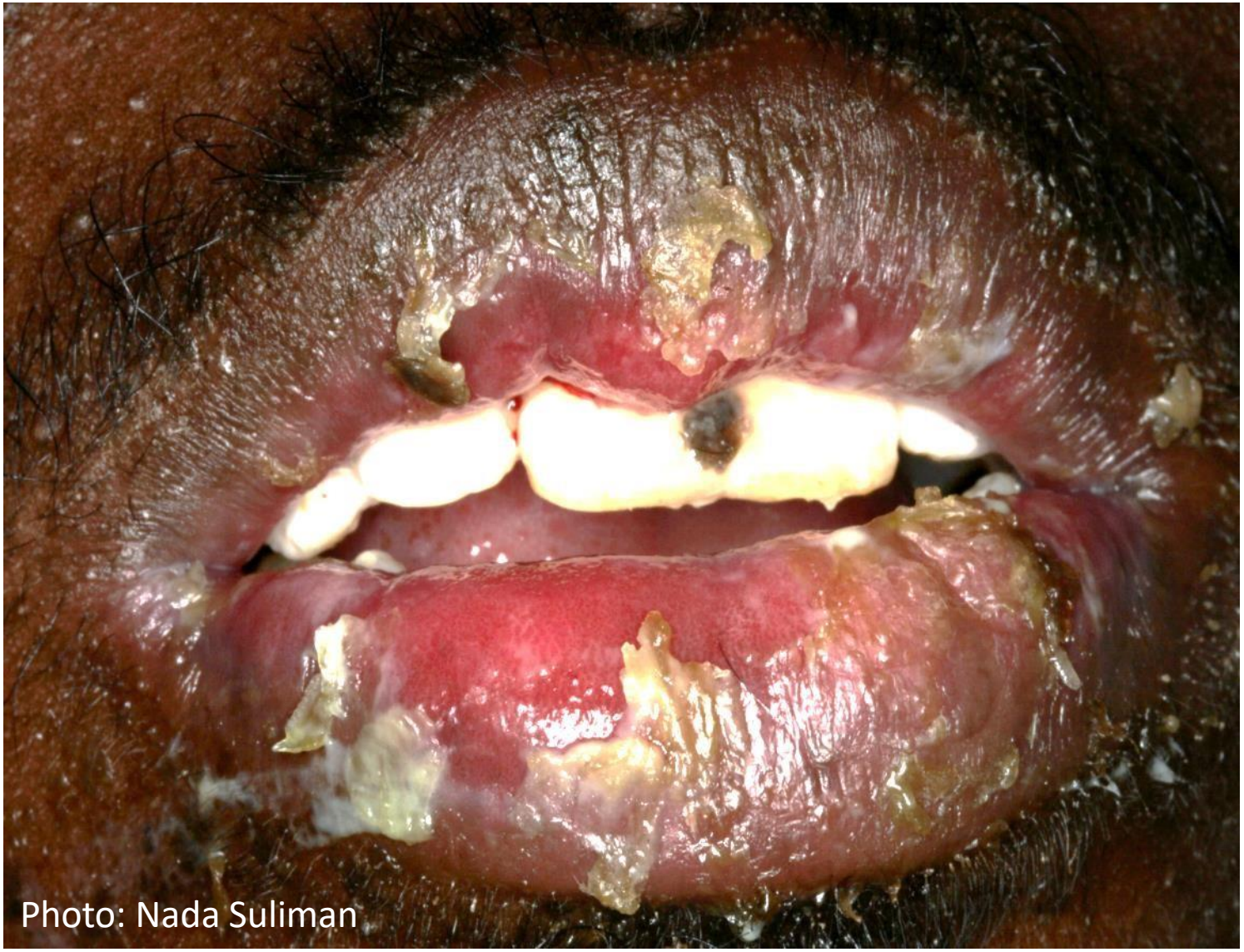
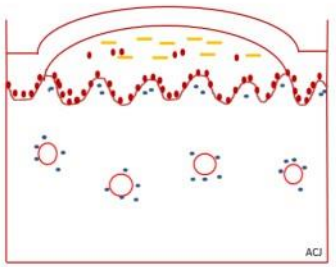
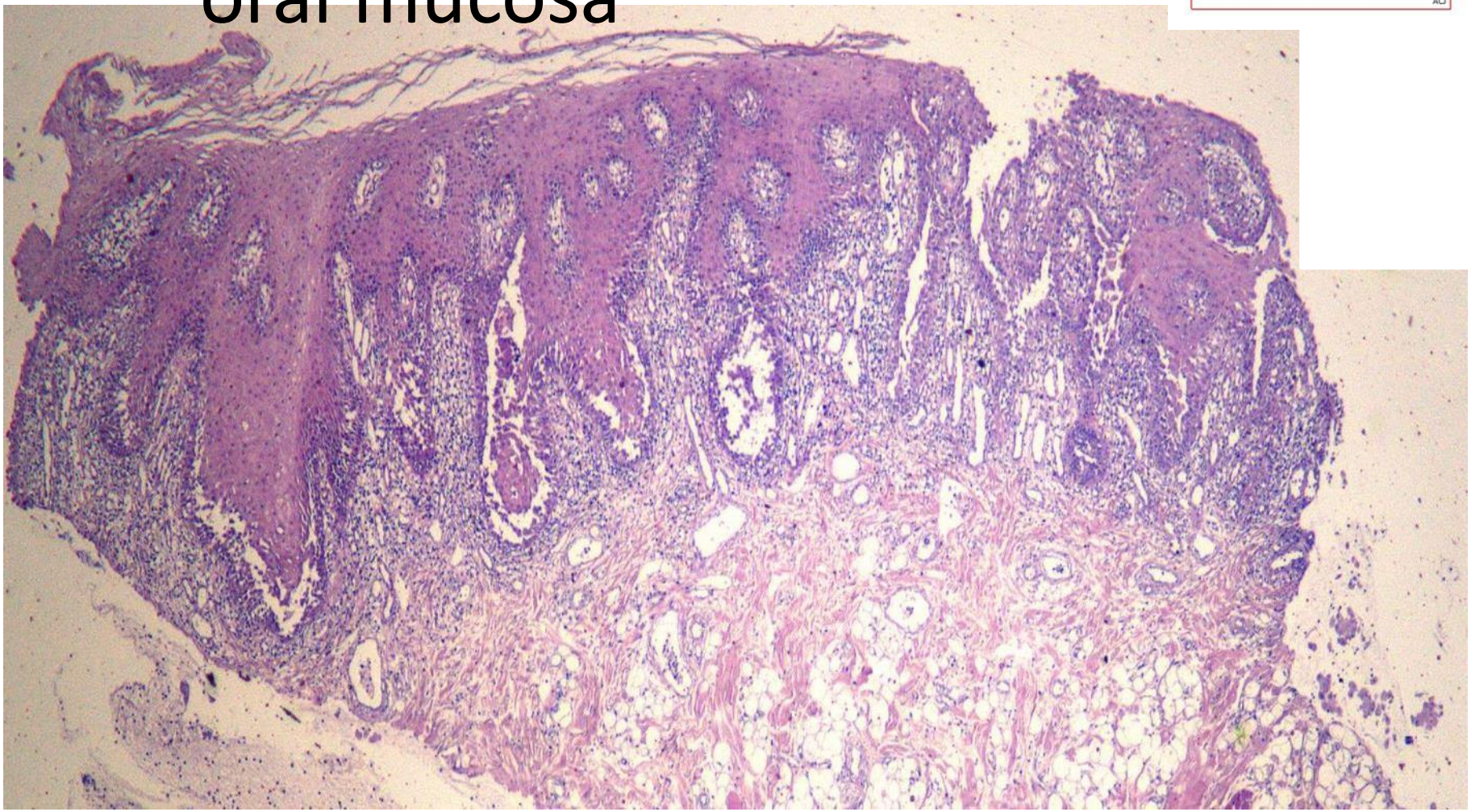
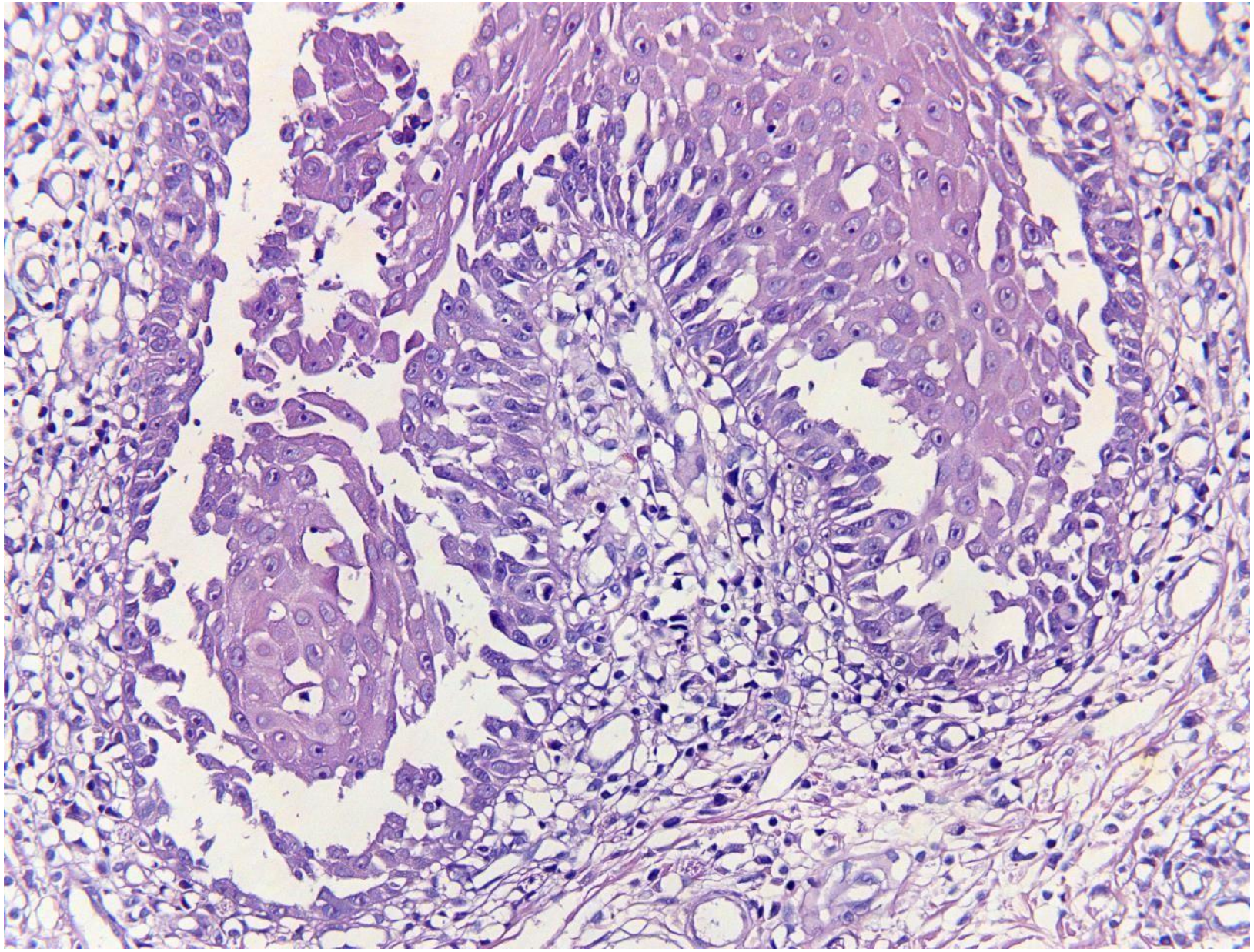


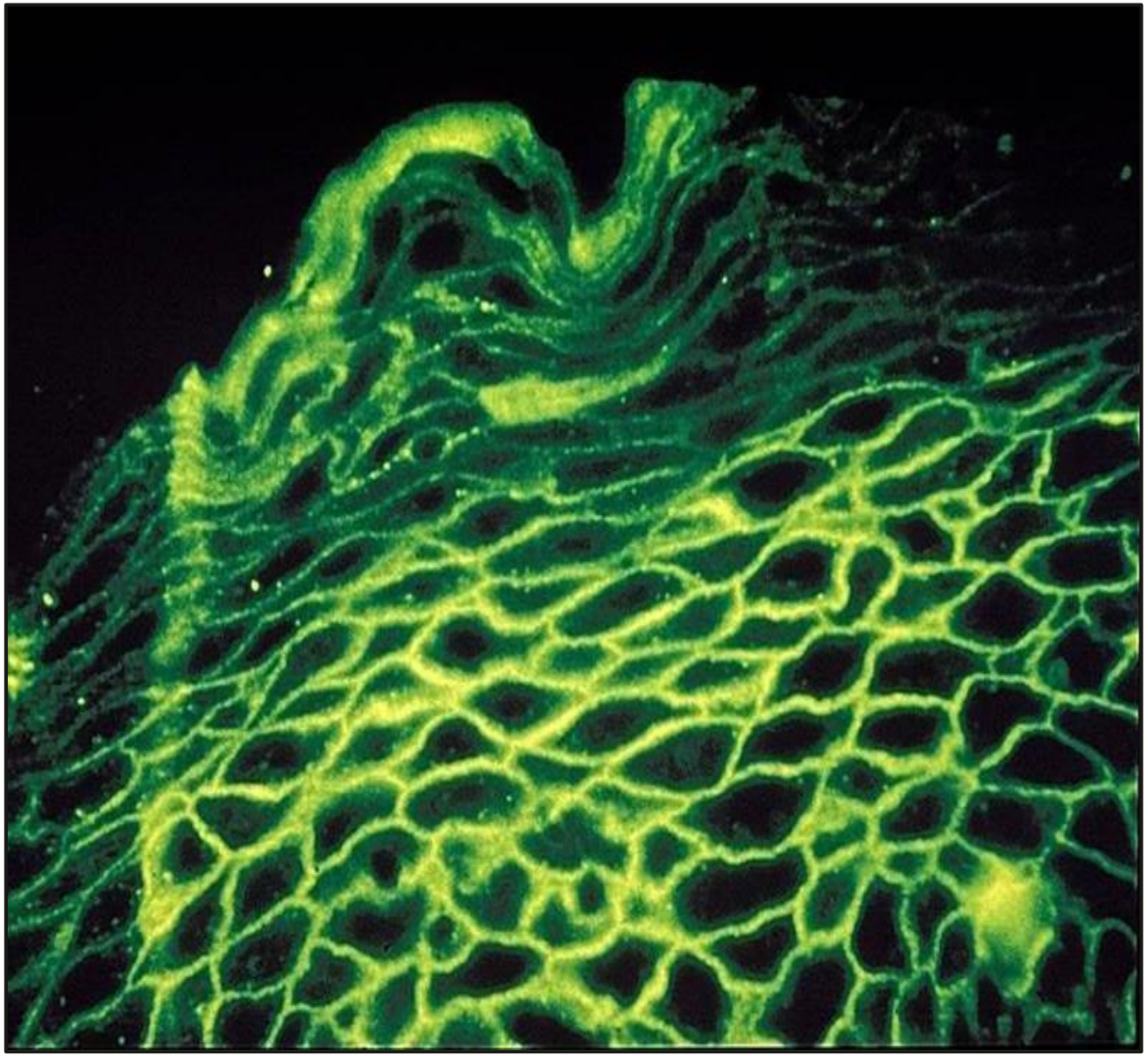
Photo: Nada Suliman

Female, 60 years.
Blisters on skin and oral mucosa

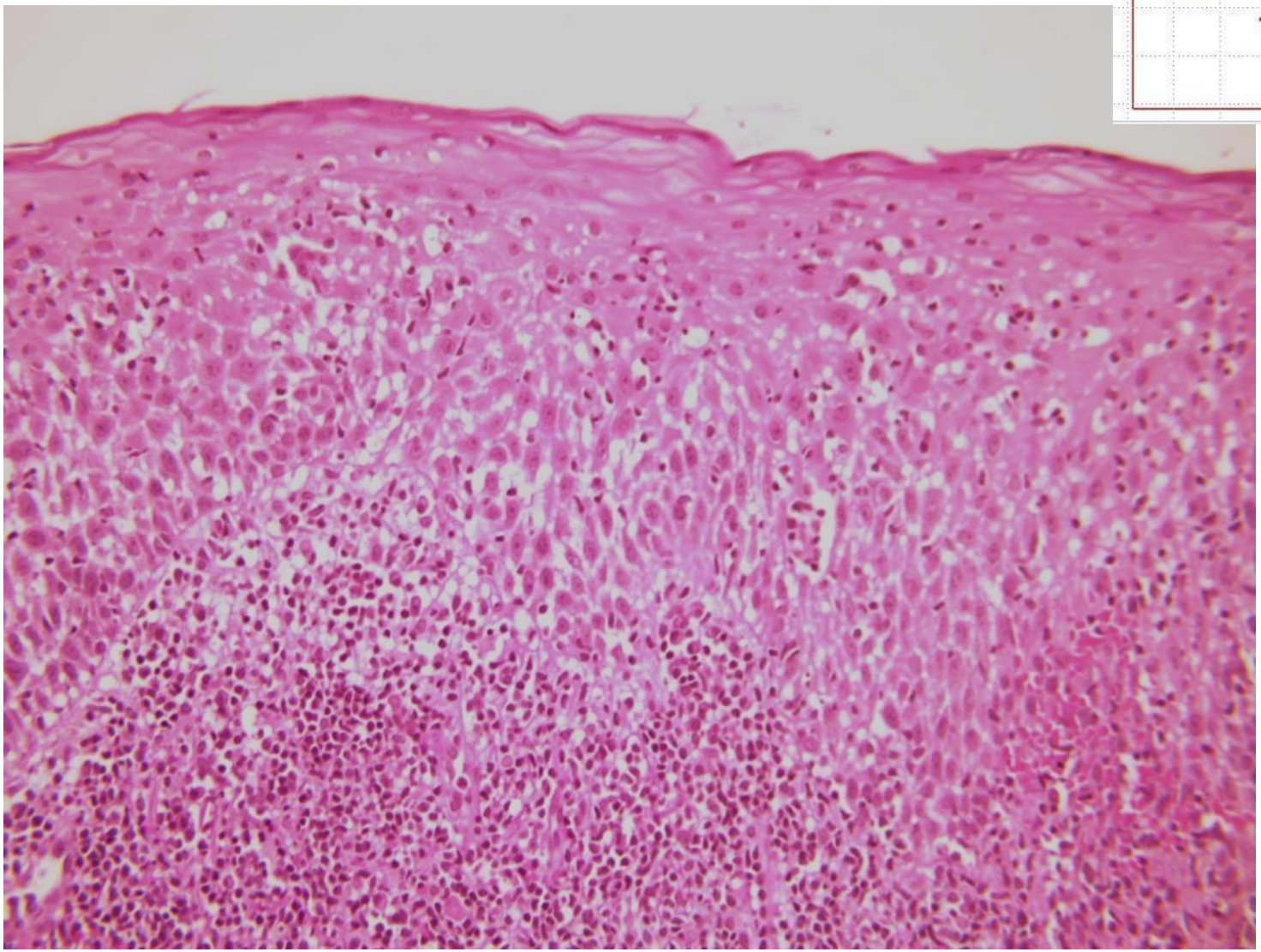


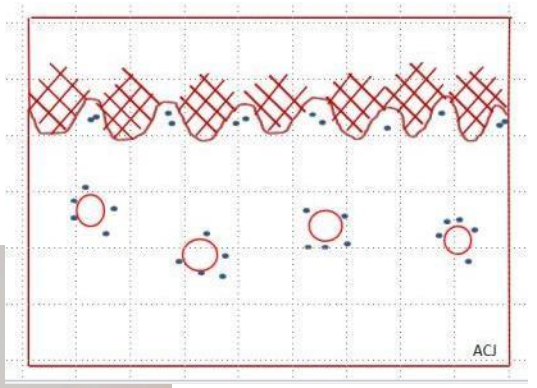


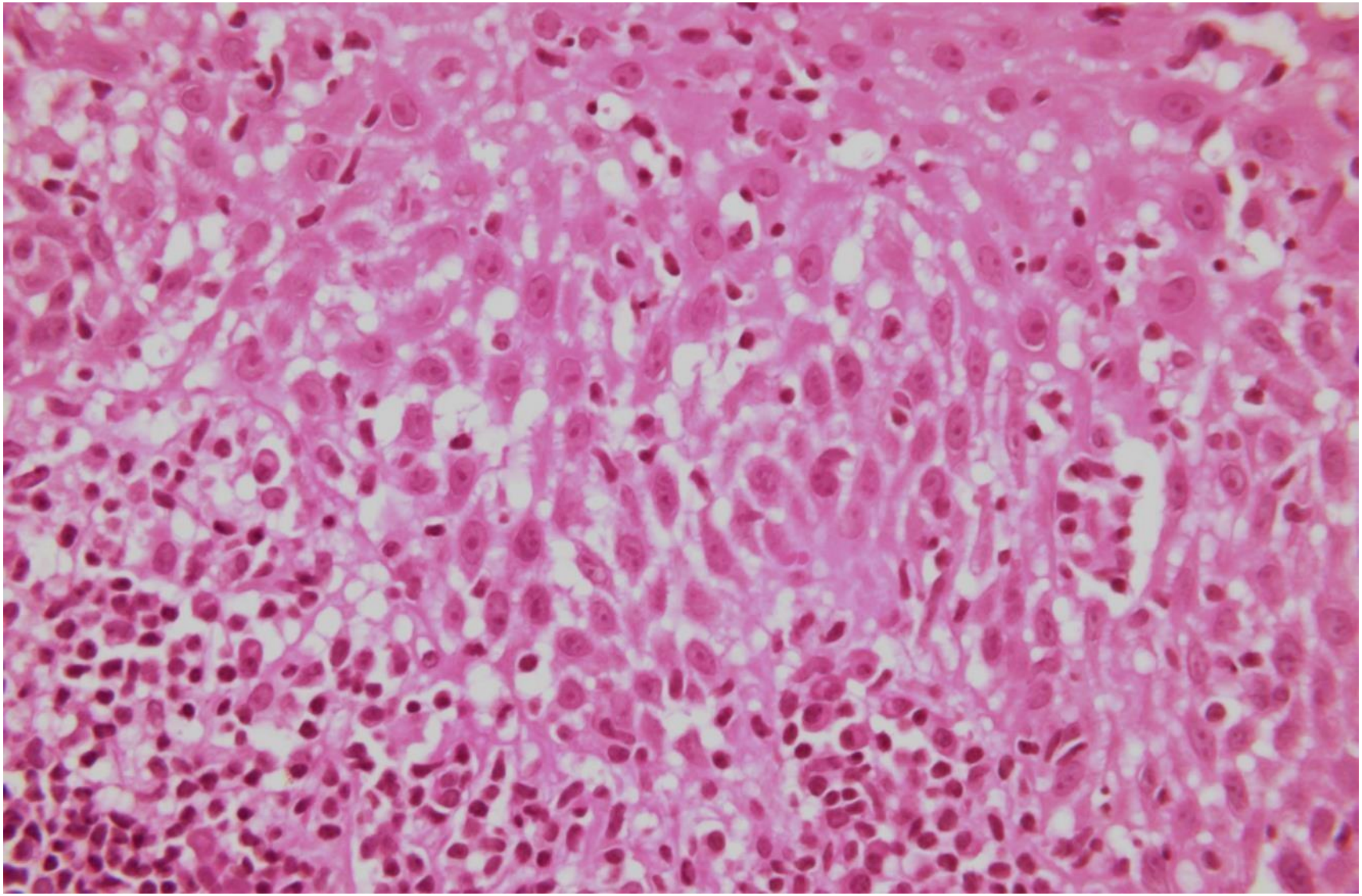




Female, 77 years. Redness and soreness in oral mucosa. Uses several medicaments (blod pressure, anti-inflammatory)







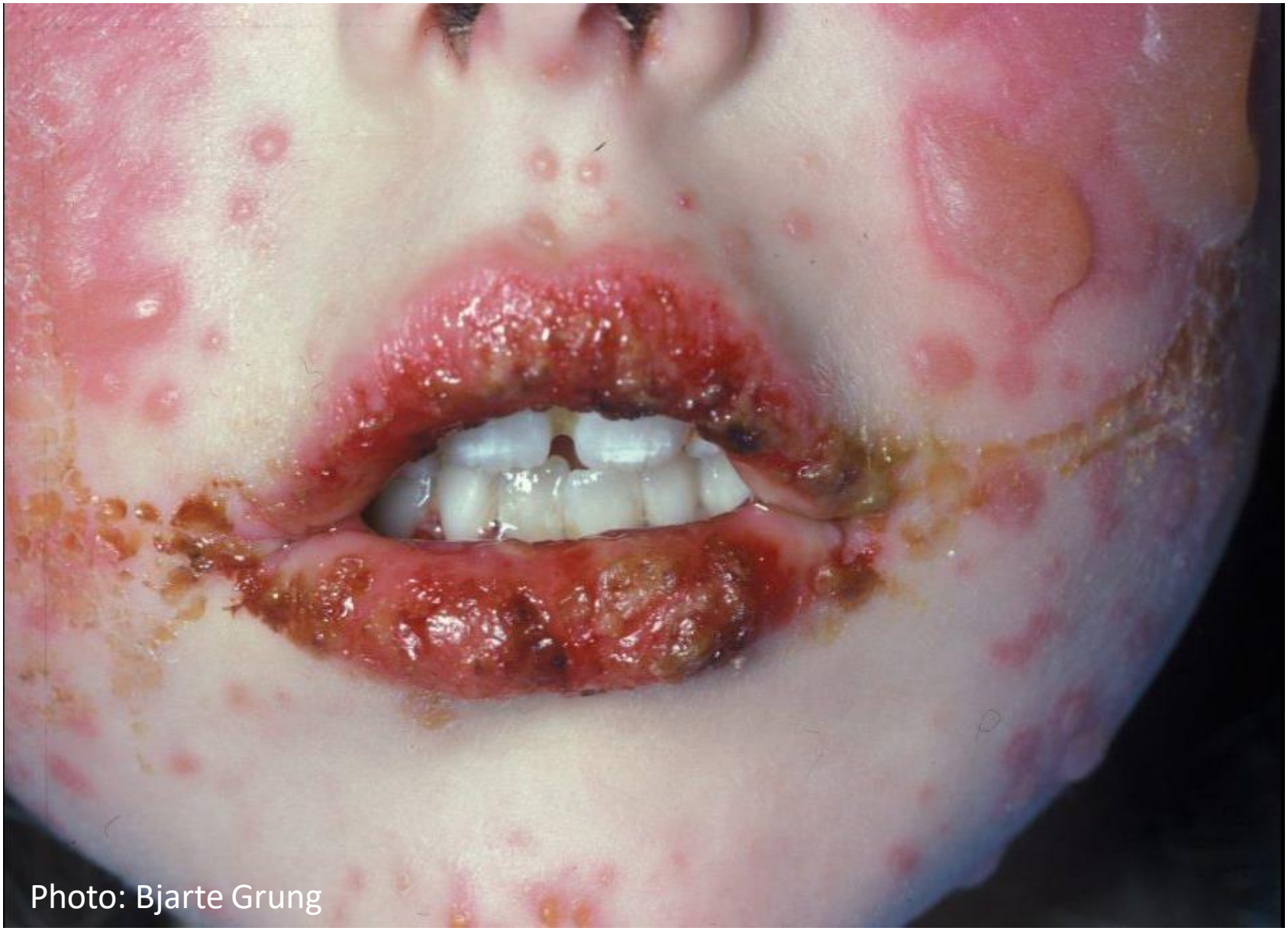
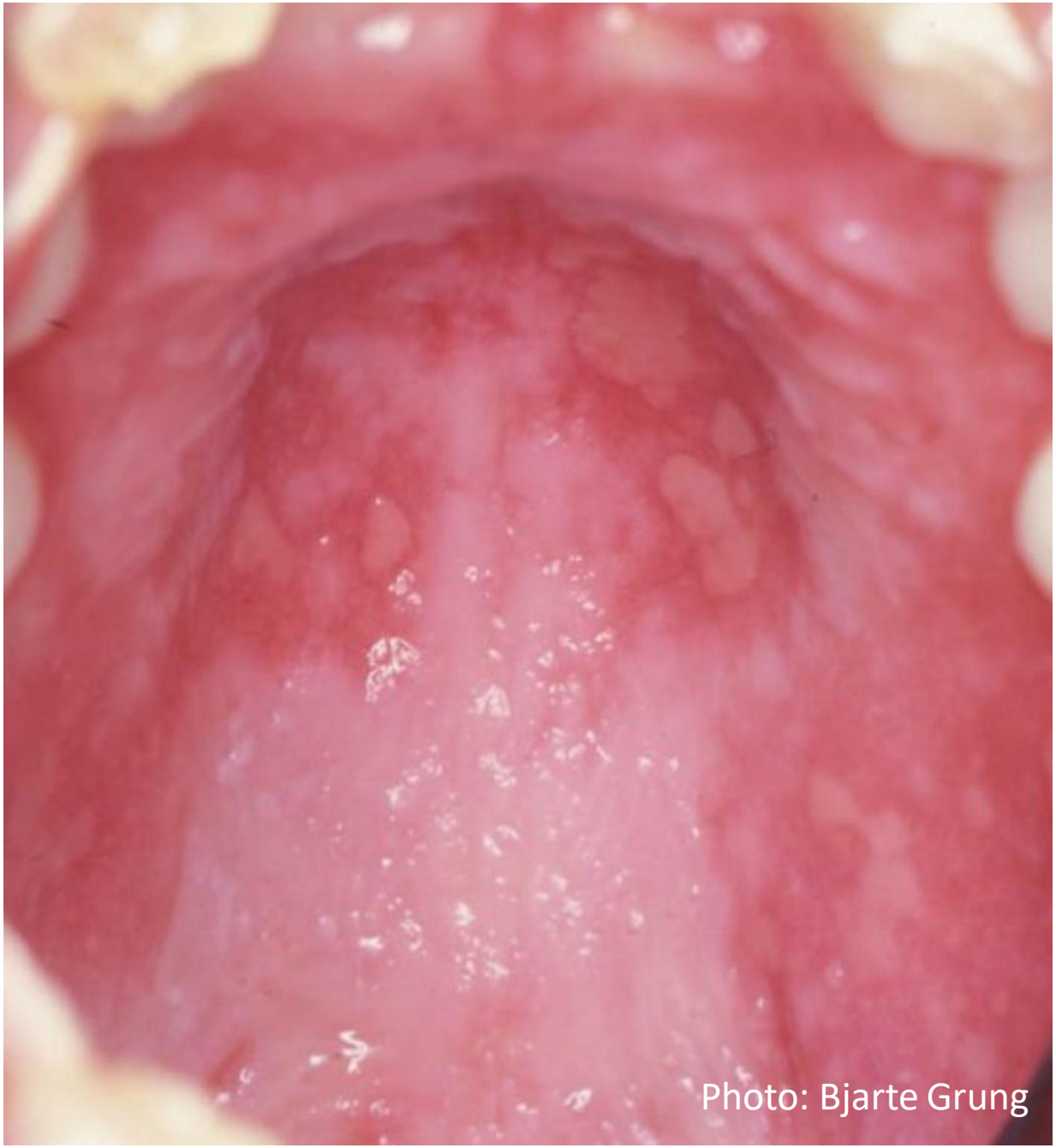


Photo: Bjarte Grung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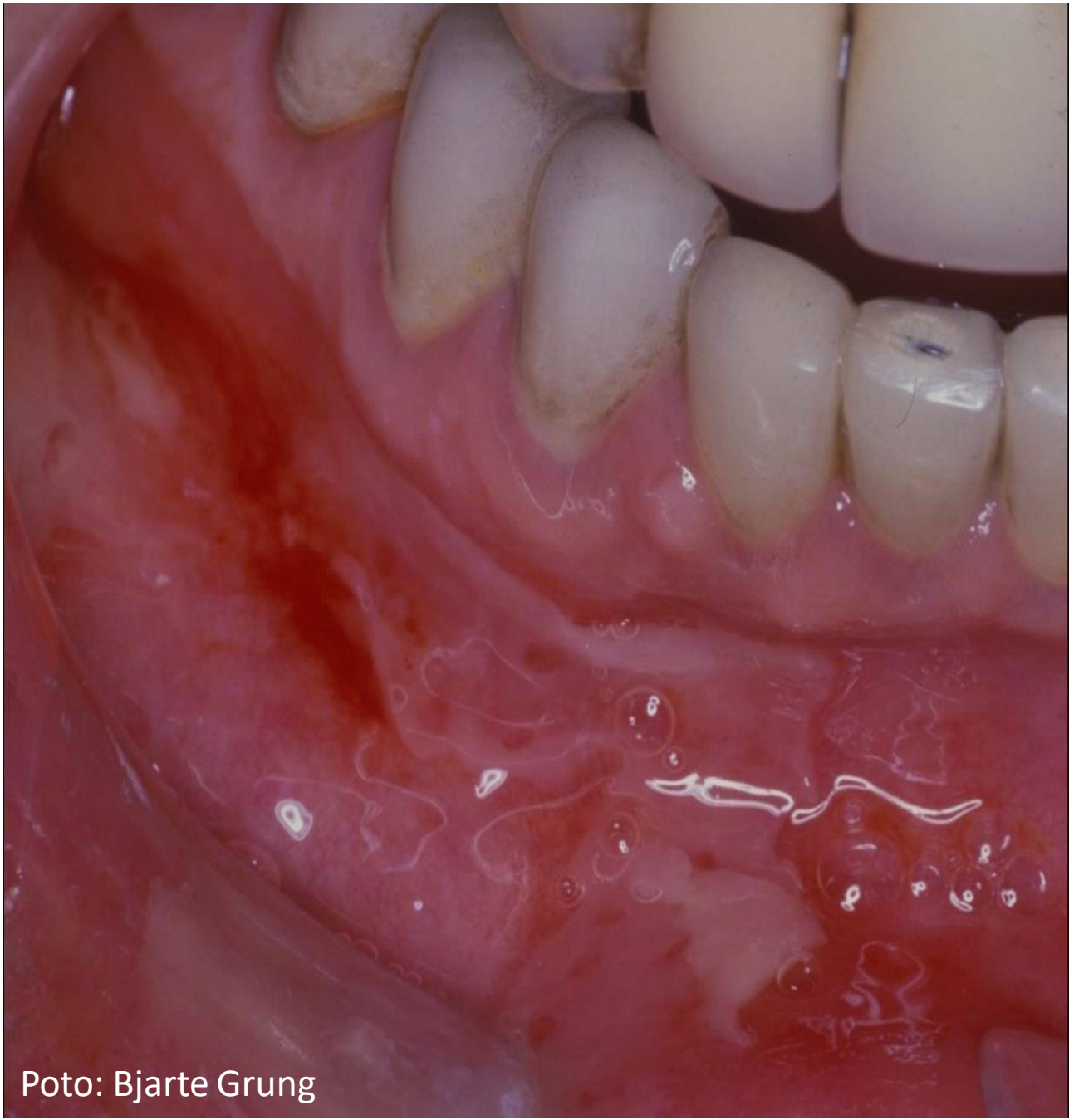
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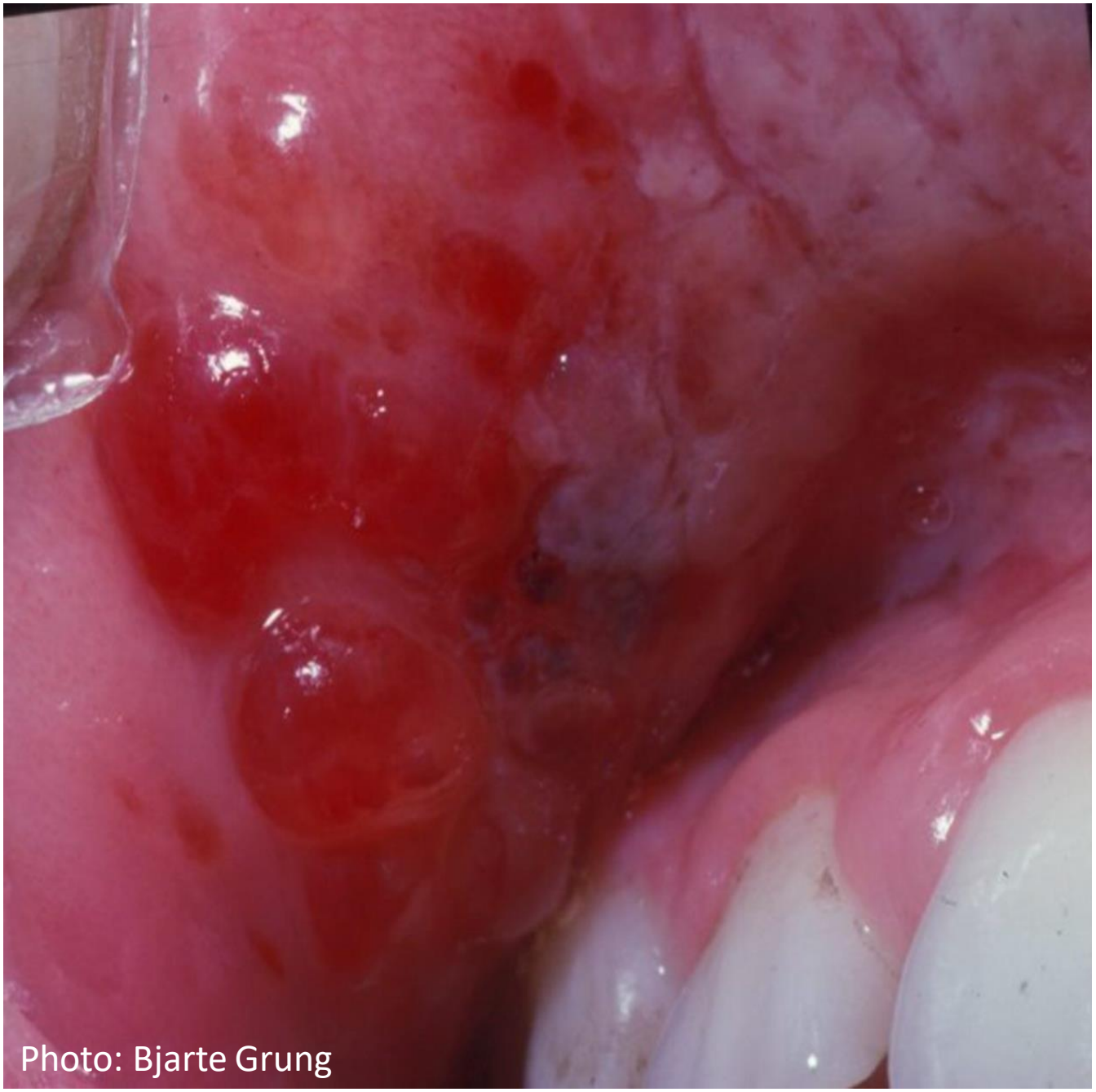
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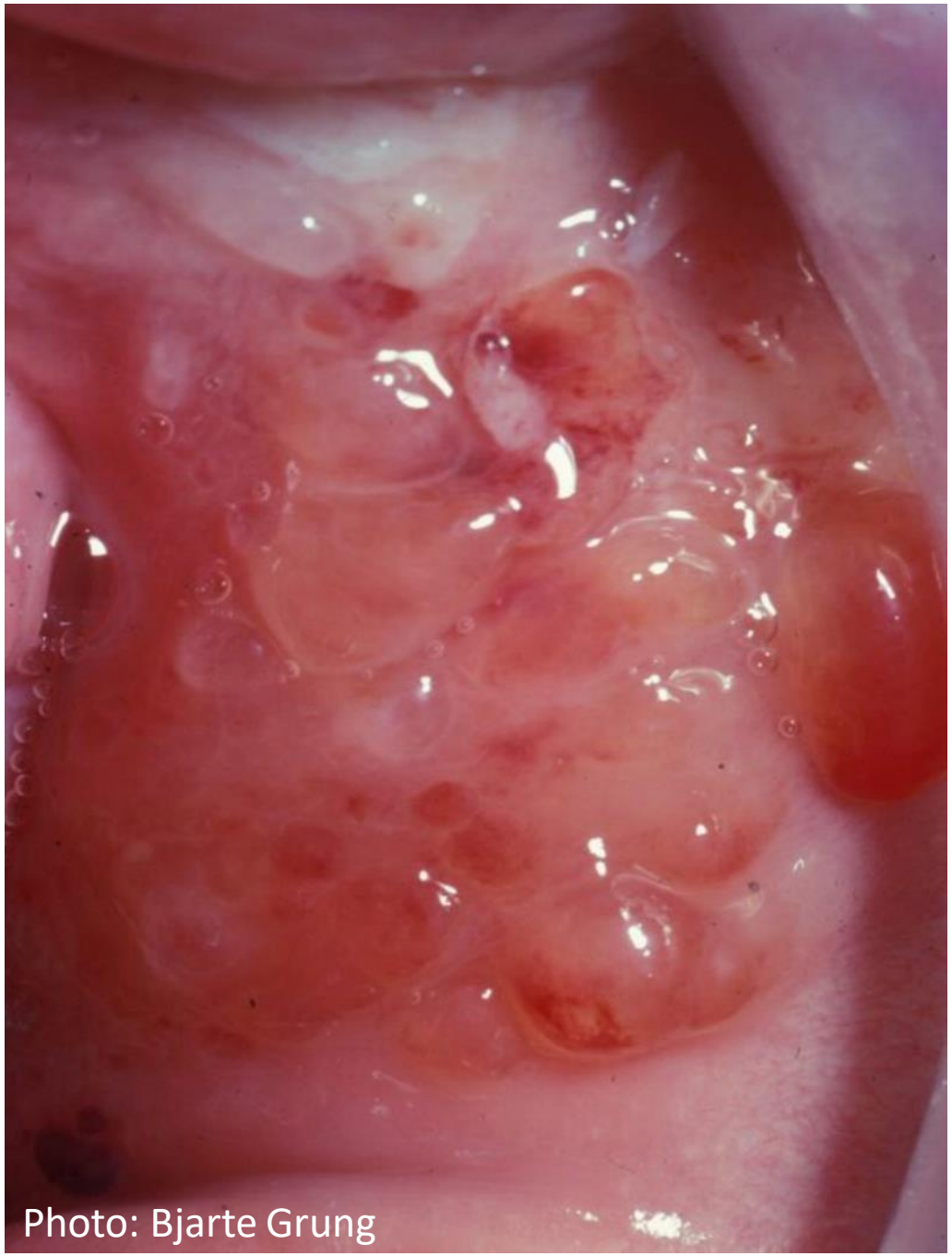
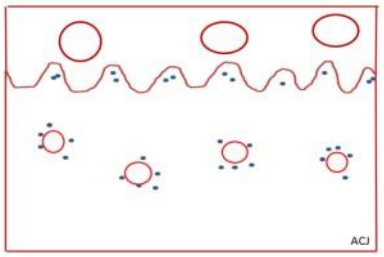
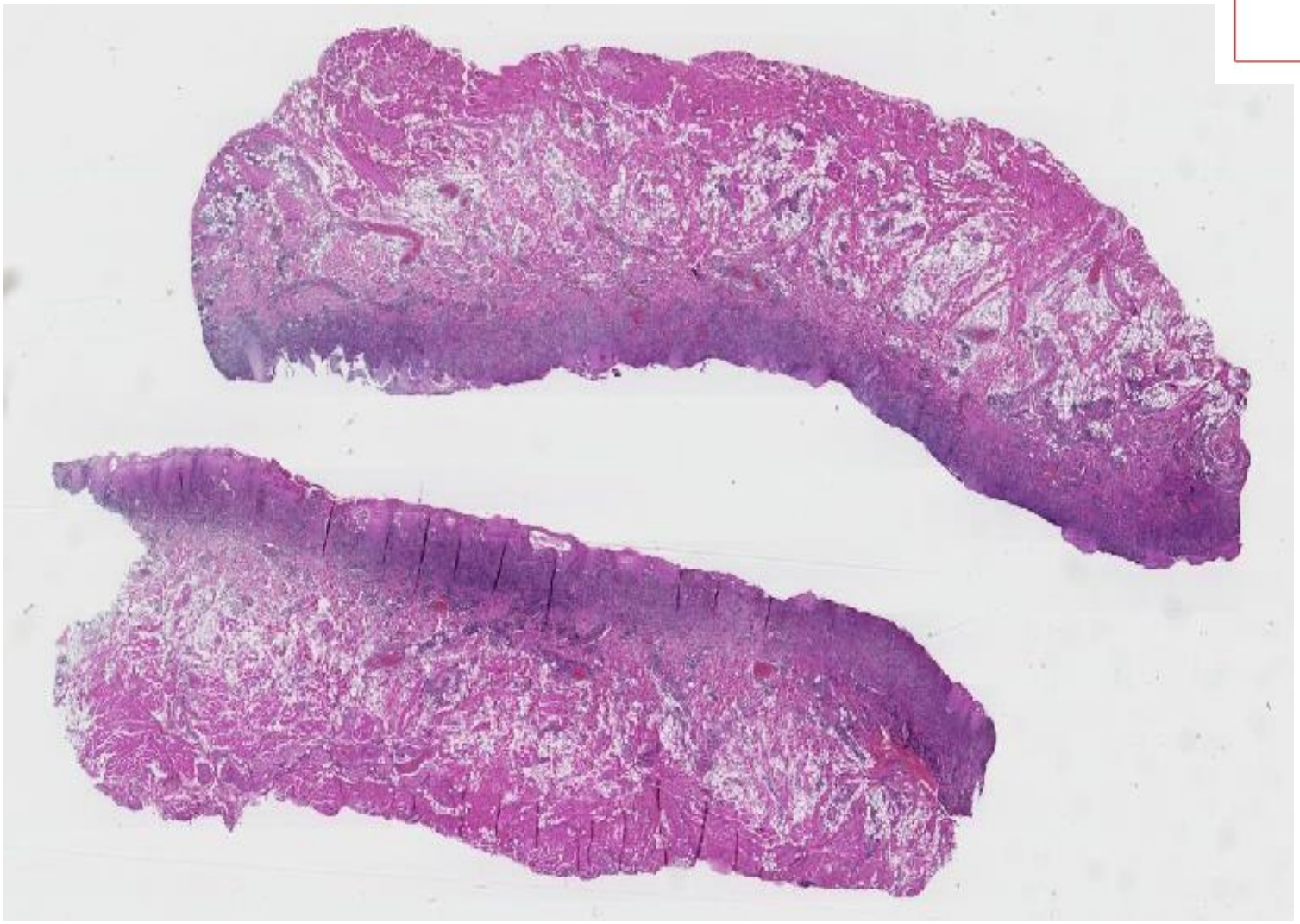


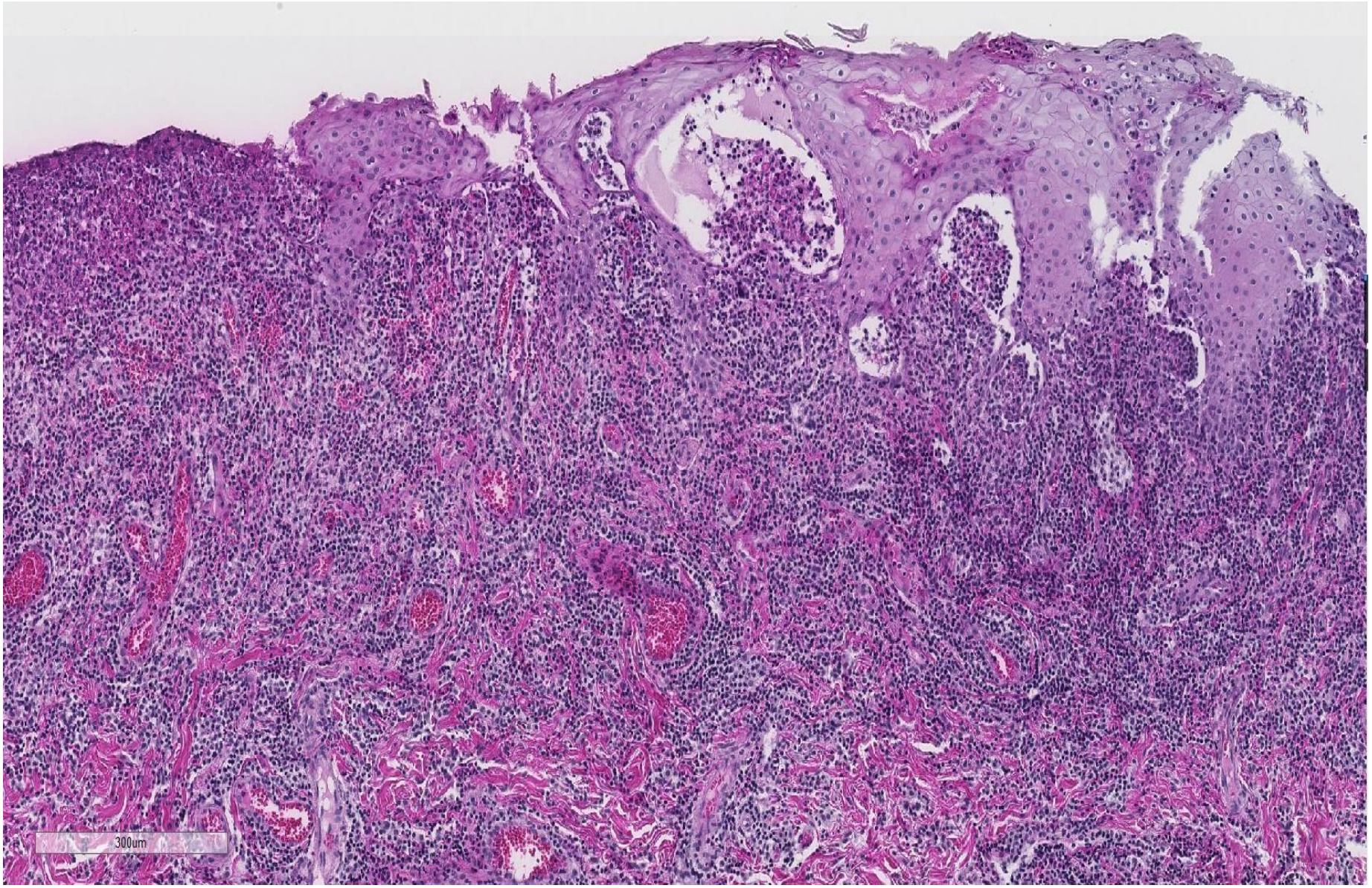
Photo: Bjarte Grung

Female , 46 years. Recurrent ulcerations in oral mucosa, often also on skin.

TD from dermatologist: erythema multiforme.






300um