SHAFER HINE LEVY

A Textbook of Oral Pathology

FOURTH EDITION

THOMA'S

Volume two

Oral pathology

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ORAL PATHOLOGY

Clinical-Pathologic Correlations

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Oral & Maxillofacial Pathology

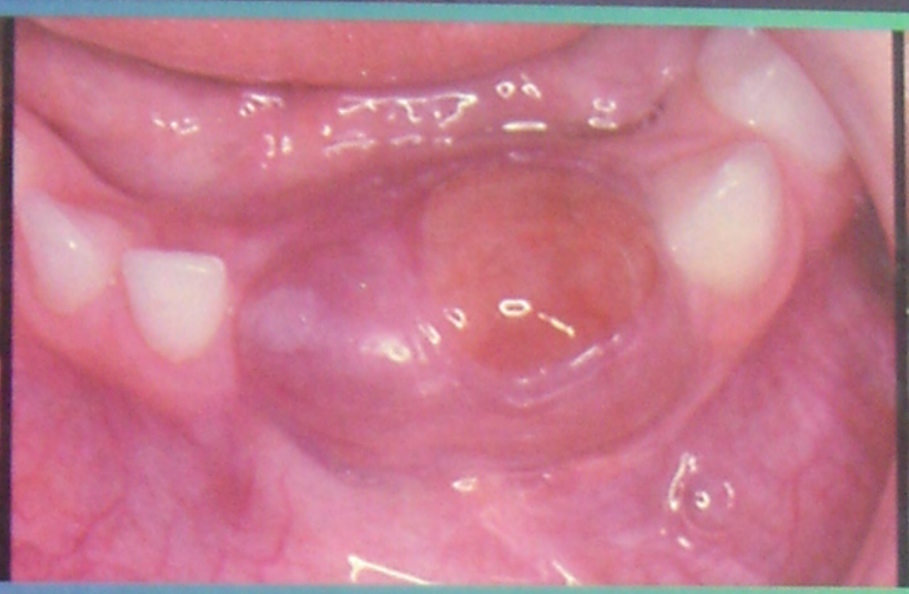
Chapter One: Introduction to Oral Pathology

A. Oral Pathology Defined
B. Important Factors in Pathology (Oral)
    1. Age
    2. Sex
    3. Location
    4. Symptoms-chief complaint
    a. Bonus
    5. Etiology--definition
    6. Radiographic Findings
    7. Pathogenesis--defined
    8. Treatment
C. Branches of Oral Pathology
    1. Basic (fundamental)
    2. Diagnostic
    3. Experimental
    4. Evaluative

月/日/星期	時間/時數	課　程　主　題	專/兼	授課老師
2/19/Wed.	9:10-12:00AM	Odontogenic tumors (I)	專	林正伸AP.
2/26/Wed.	9:10-12:00AM	Introduction	專	林立民 P.
2/27/Thr.	9:10-12:00AM	Odontogenic tumors (II)	專	林正伸AP
3/5/Wed.	9:10-12:00AM	Benign & malignant tumors of oral cavity	專	林立民 P.
3/12/Wed.	9:10-12:00AM	Developmental disturbance of oral & paraoral structure (I)	兼	醫學院 P.
3/19/Wed.	9:10-12:00AM	Developmental disturbance of oral & paraoral structure (II)	專	林立民 P.
3/26/Wed.	9:10-12:00AM	Regressive alteration of the teeth, caries. Pulpitis & periapical lesion	專	陳玉民VS. 印憲梅VS.
4/2/Wed.		Spring vacation		
4/9/Wed.	9:10-12:00AM	Bacterial, viral & mycotic infections	專	黃湧澧VS.
4/16/Wed.		Midterm examination		
4/23/Wed.	9:10-12:00AM	Periodontal diseases	專	蔡吉政 P.
4/30/Wed.	9:10-12:00AM	Cysts of head & neck	專	林正伸AP.
5/7/Wed.	9:10-12:00AM	Diseases of jaw bone & TMJ	專	印憲梅VS.
5/14/Wed.	9:10-12:00AM	Salivary gland diseases	專	林正伸AP.
5/21/Wed.	9:10-12:00AM	Injury & repair (I) physical & chemical injuries of oral cavity (II) wound healing	兼	陳玉民VS.
5/28/Wed.	9:10-12:00AM	Skin & bullous diseases of oral cavity Diseases of nerve & muscle	兼	陳玉民VS.
6/4/Wed.	9:10-12:00AM	Oral aspects of metabolic diseases	兼	醫學院 P.
6/11/Wed.	9:10-12:00AM	Diseases of blood & blood-forming organs	兼	林正伸AP.
6/18/Wed.		Final examination		

Oral & Maxillofacial PATHOLOGY







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CHAPTER 10

Epithelial Pathology

CHAPTER OUTLINE

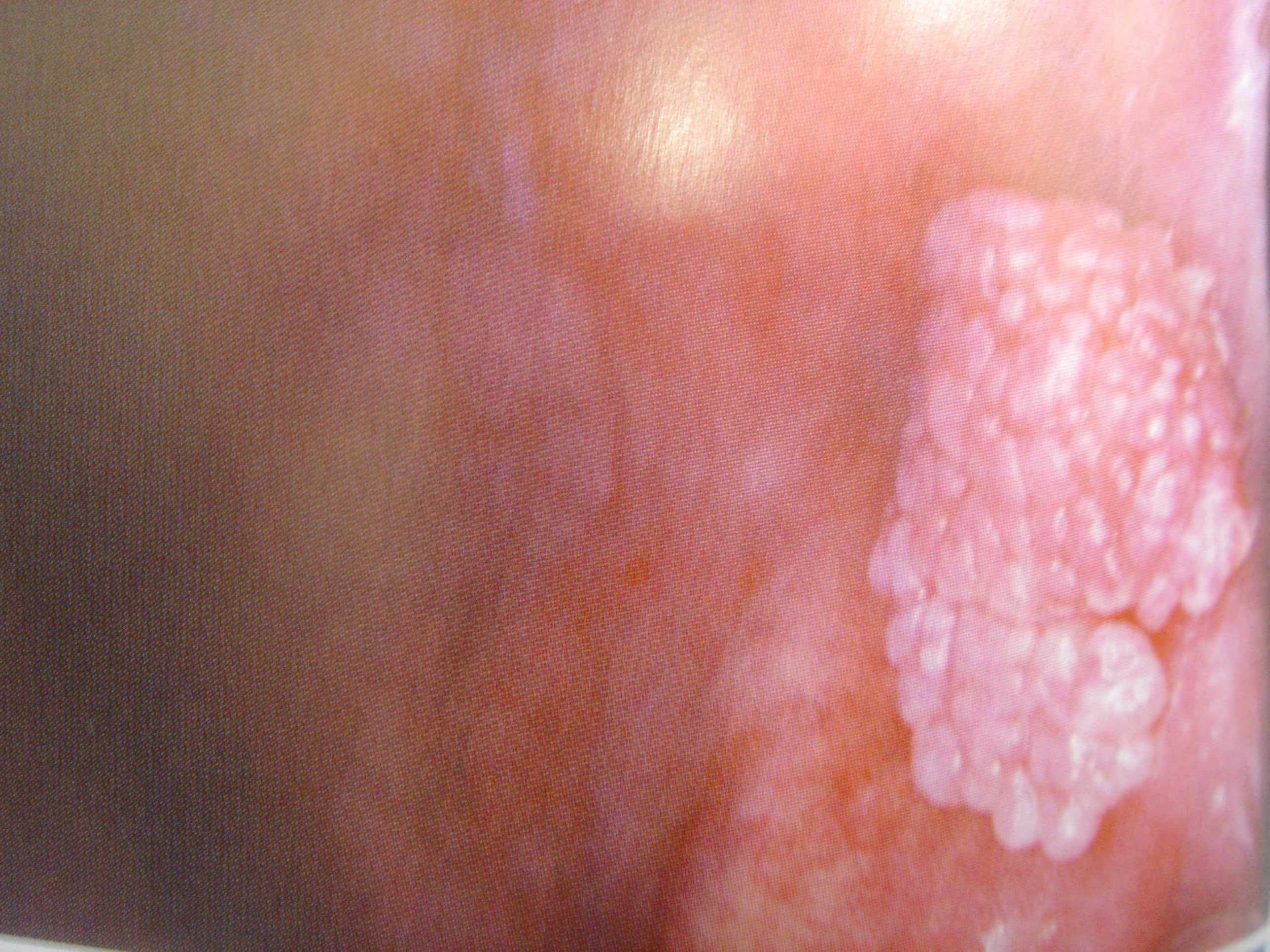
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  Halo Nevus
  Spitz Nevus
  Blue Nevus
Leukoplakia
Erythroplakia
Smokeless Tobacco Use and Smokeless Tobacco Keratosis
Oral Submucous Fibrosis
Nicotine Stomatitis
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Melanoma
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  Nodular Melanoma
  Lentigo Maligna Melanoma
  Acral Lentiginous Melanoma

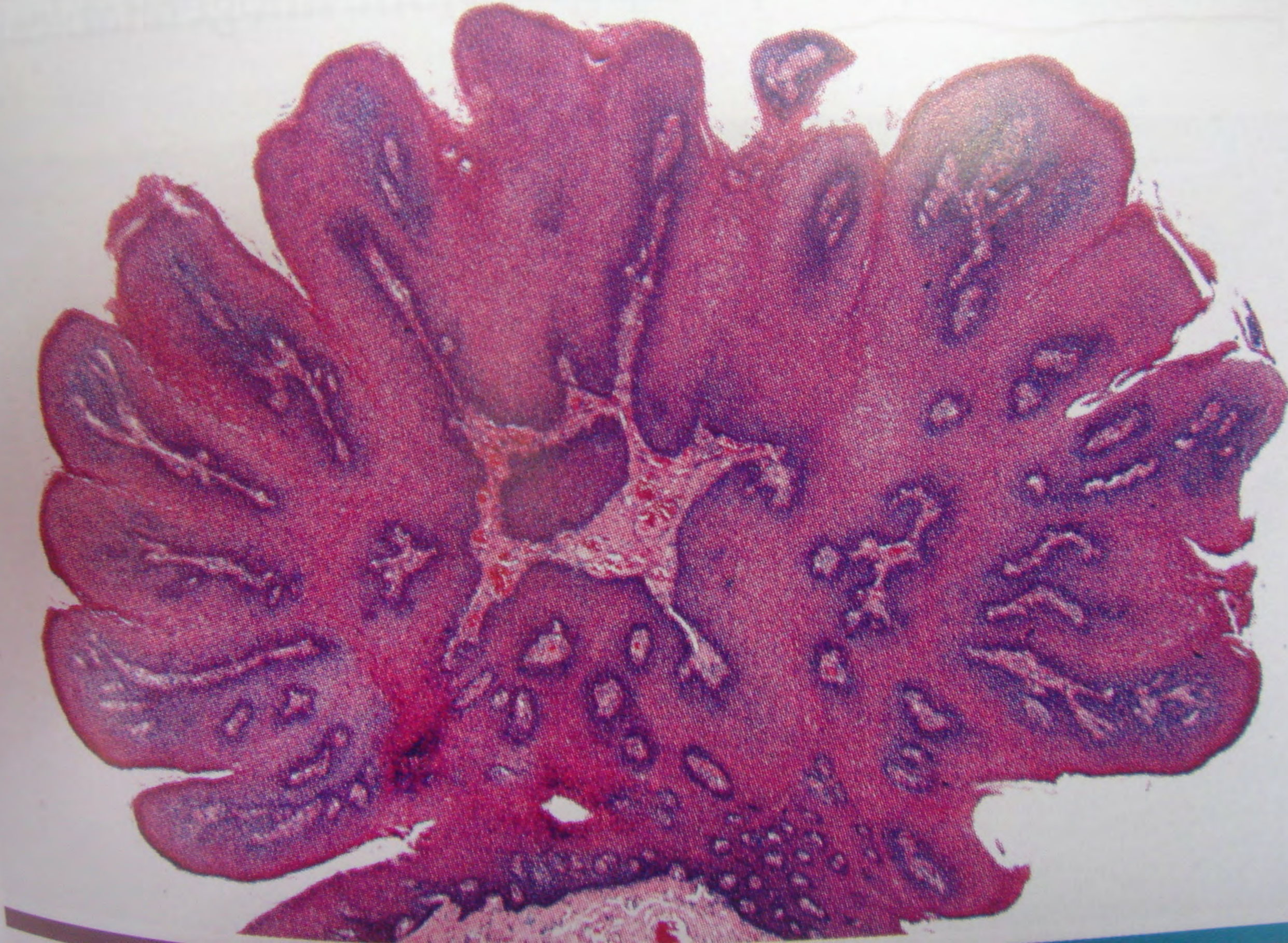
SQUAMOUS PAPILLOMA

The **squamous papilloma** is a benign proliferation of stratified squamous epithelium, resulting in a papillary or verruciform mass. Presumably, this lesion is induced by the human papillomavirus (HPV). HPV comprises a large family (more than 100 types) of double-stranded DNA viruses of the papovavirus subgroup A. Recent research has shown that 81% of normal adults have buccal epithelial cells that contain at least one type of HPV, although case control studies using more rigorous criteria have usually shown distinct differences, with high levels of HPV in oral lesions and low levels in normal controls.









occurs in one of every 250 adults and makes up approximately 3% of all oral lesions submitted for biopsy.

Clinical Features

The squamous papilloma occurs with equal frequency in both men and women. Some authors have asserted that it develops predominantly in children, but epidemiologic studies indicate that it can arise at any age and, in fact, is diagnosed most often in persons 30 to 50 years of age. Sites of predilection include the tongue, lips, and soft palate, but any oral surface may be affected. This lesion is the most common of the soft tissue masses arising from the soft palate. most common site.

The squamous papilloma is a soft, painless, usually

juvenile-onset type may obstruct the airway.

Histopathologic Features

The papilloma is characterized by a proliferation of keratinized stratified squamous epithelium arrayed in finger-like projections with fibrovascular connective tissue cores (Figure 10-4). The connective tissue cores may show inflammatory changes, depending on the amount of trauma sustained by the lesion. The keratin layer is thickened in lesions with a whiter clinical appearance, and the epithelium typically shows a normal maturation pattern (Figure 10-5). Occasional papillomas demonstrate basilar hyperplasia and mitotic activity, which can be mistaken for mild epithelial dysplasia. **Koilocytes**, virus-altered epithelial clear cells with small dark (pyknotic) nuclei, are sometimes seen high in the prickle cell layer.

epithelial clear cells with small dark (pyknotic)
sometimes seen high in the prickle cell layer.

Treatment and Prognosis

Conservative surgical excision, including the
lesion, is adequate treatment for the oral
papilloma, and recurrence is unlikely. Freque
have been left untreated for years with no rep
formation into malignancy, continuous enla
dissemination to other parts of the oral cav

carcinoma will develop in long-standing laryngeal papillomatosis, sometimes in a smoker or a patient with a history of irradiation to the larynx.

VERRUCA VULGARIS (COMMON WART)

Verruca vulgaris is a benign, virus-induced, focal hyperplasia of stratified squamous epithelium. One or more of the associated human papillomavirus (HPV) types 2, 4, 6, and 40 are found in virtually all examples. Verruca vulgaris is contagious and can spread to other parts of a person's skin or mucous membranes by way of autoinoculation. It infrequently develops on oral mucosa but is extremely common on the skin.

Clinical Features

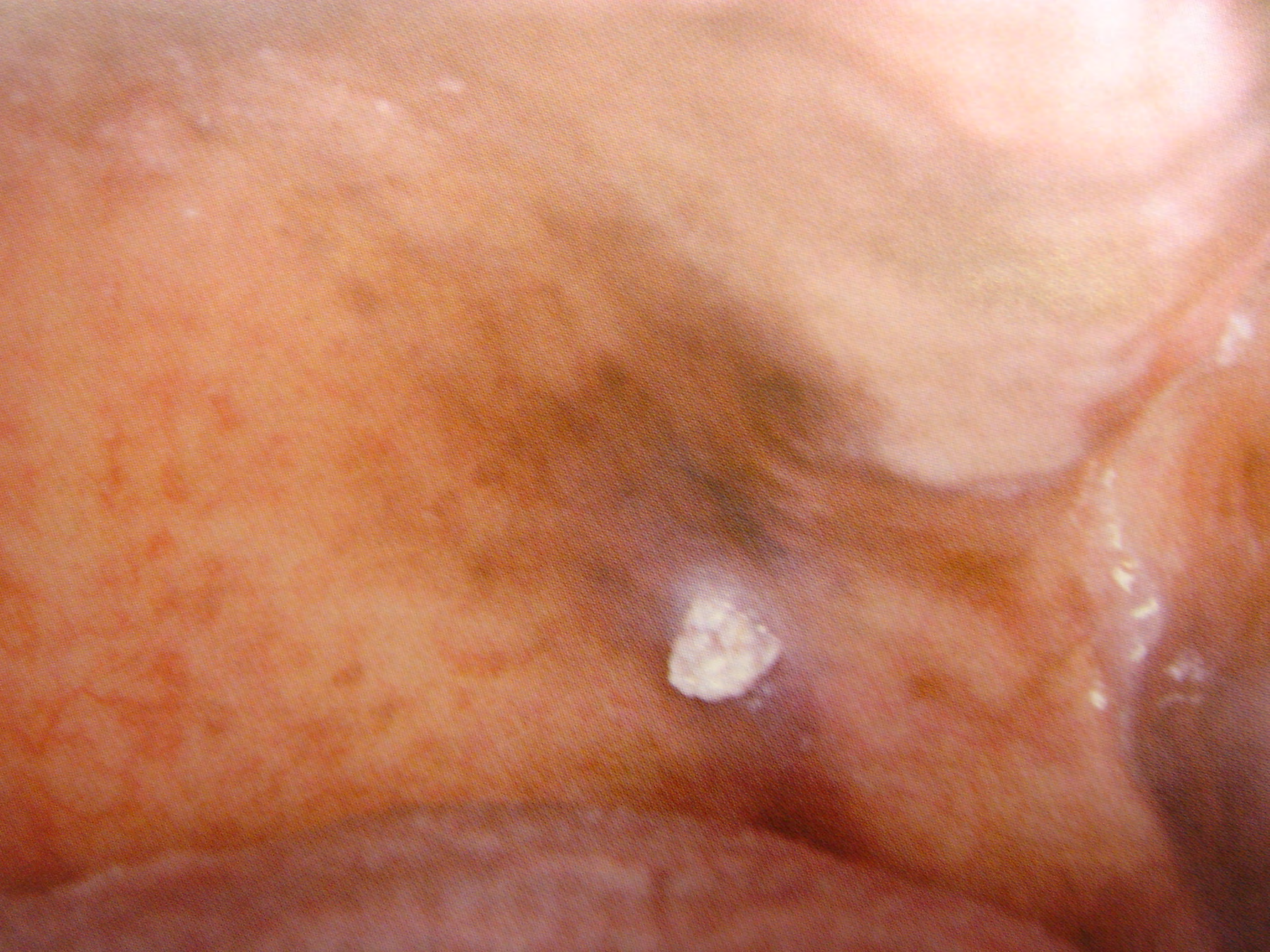
garis is contagious and can spread to other parts of a person's skin or mucous membranes by way of auto-inoculation. It infrequently develops on oral mucosa but is extremely common on the skin.

Clinical Features

Verruca vulgaris is frequently discovered in children, but occasional lesions may arise even into middle age. The skin of the hands is usually the site of infection (Figure 10-6). When the oral mucosa is involved, the lesions are usually found on the vermilion border, labial mucosa, or anterior tongue.







genital lesi

Treatment and Prognosis

Skin verrucae are treated effectively by liquid nitrogen cryotherapy, conservative surgical excision or curettage, or topical application of keratinolytic agents (usually containing salicylic acid and lactic acid). Oral lesions are usually surgically excised, or they may be destroyed by laser, cryotherapy, or electrosurgery. Cryotherapy induces a subepithelial blister that lifts the infected epithelium from the underlying connective tissue, allowing it to slough away. All destructive or surgical treatments should extend to include the base of the lesion.

Recurrence is seen in a small proportion of treated

Tumor-like lesions

1. Solitary bone cysts
2. Aneurysmal bone cysts
3. Juxtaarticular bone cyst
4. Metaphyseal fibrous defect

5. Eosinophilic granuloma

6. Fibrous dysplasia

7. Myositis ossificans

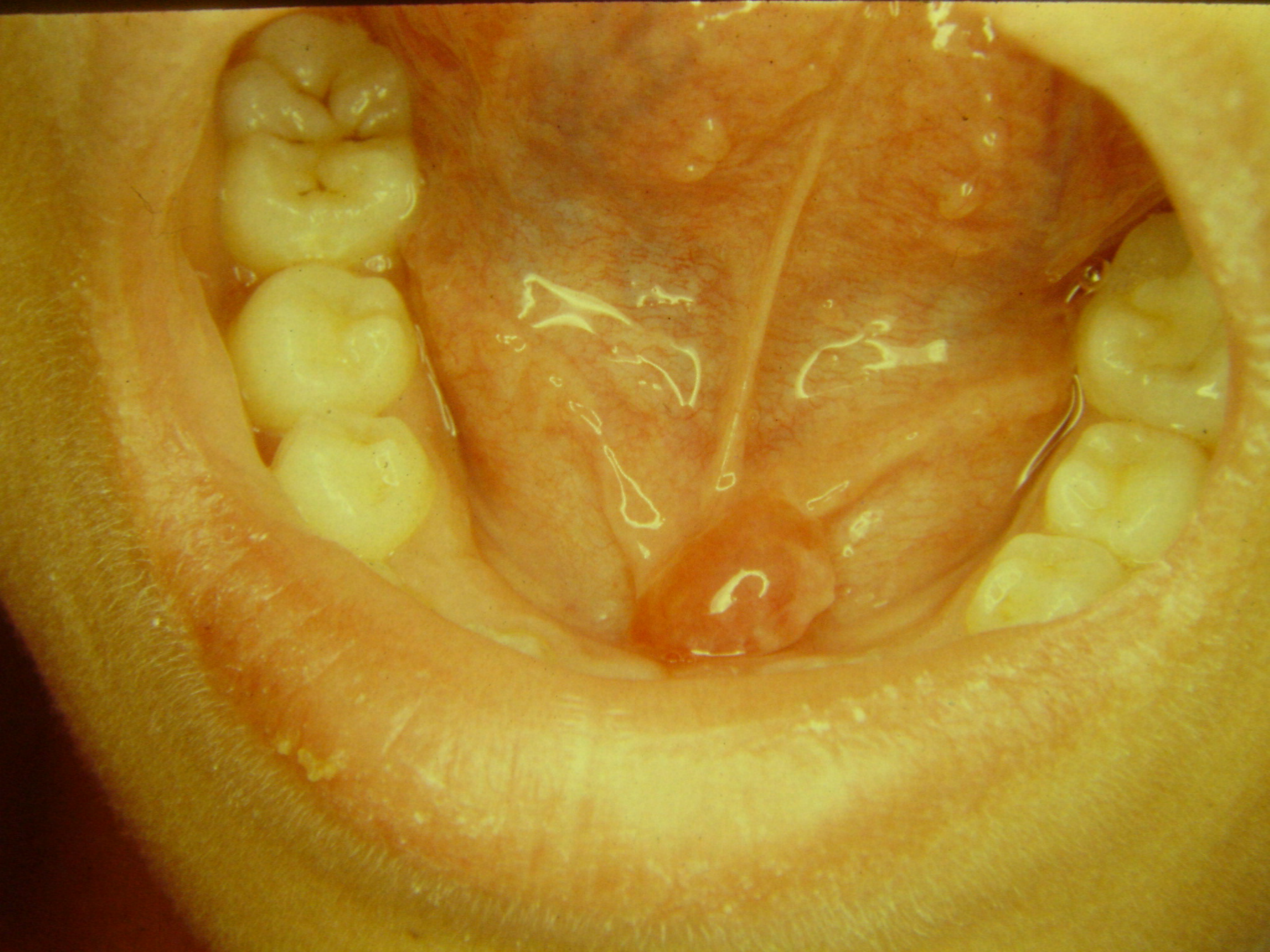
8. Brown tumor













BENIGN AND MALIGNANT TUMORS OF ORAL CAVITY

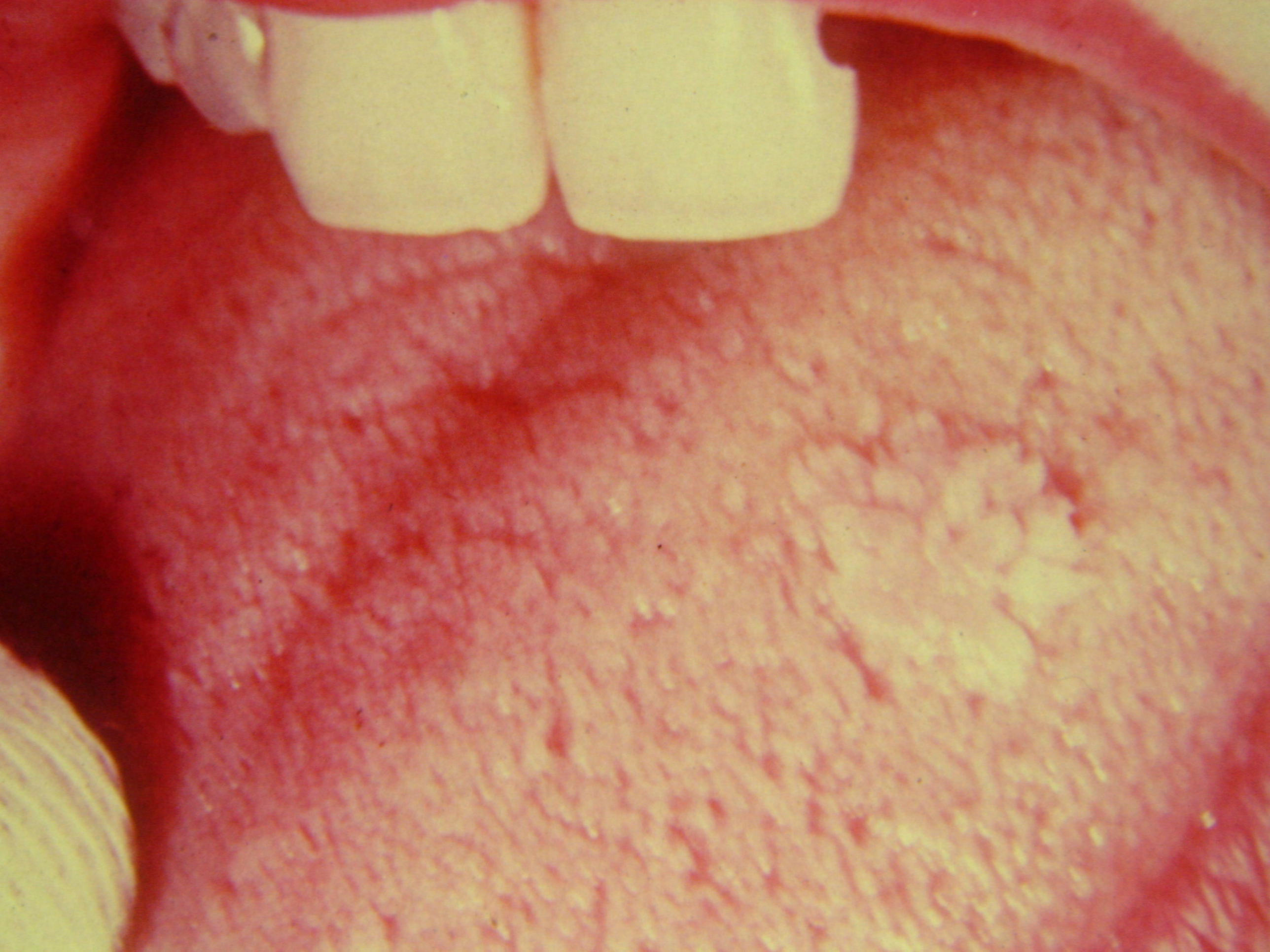
BENIGN TUMORS OF EPITHELIAL TISSUE ORIGIN

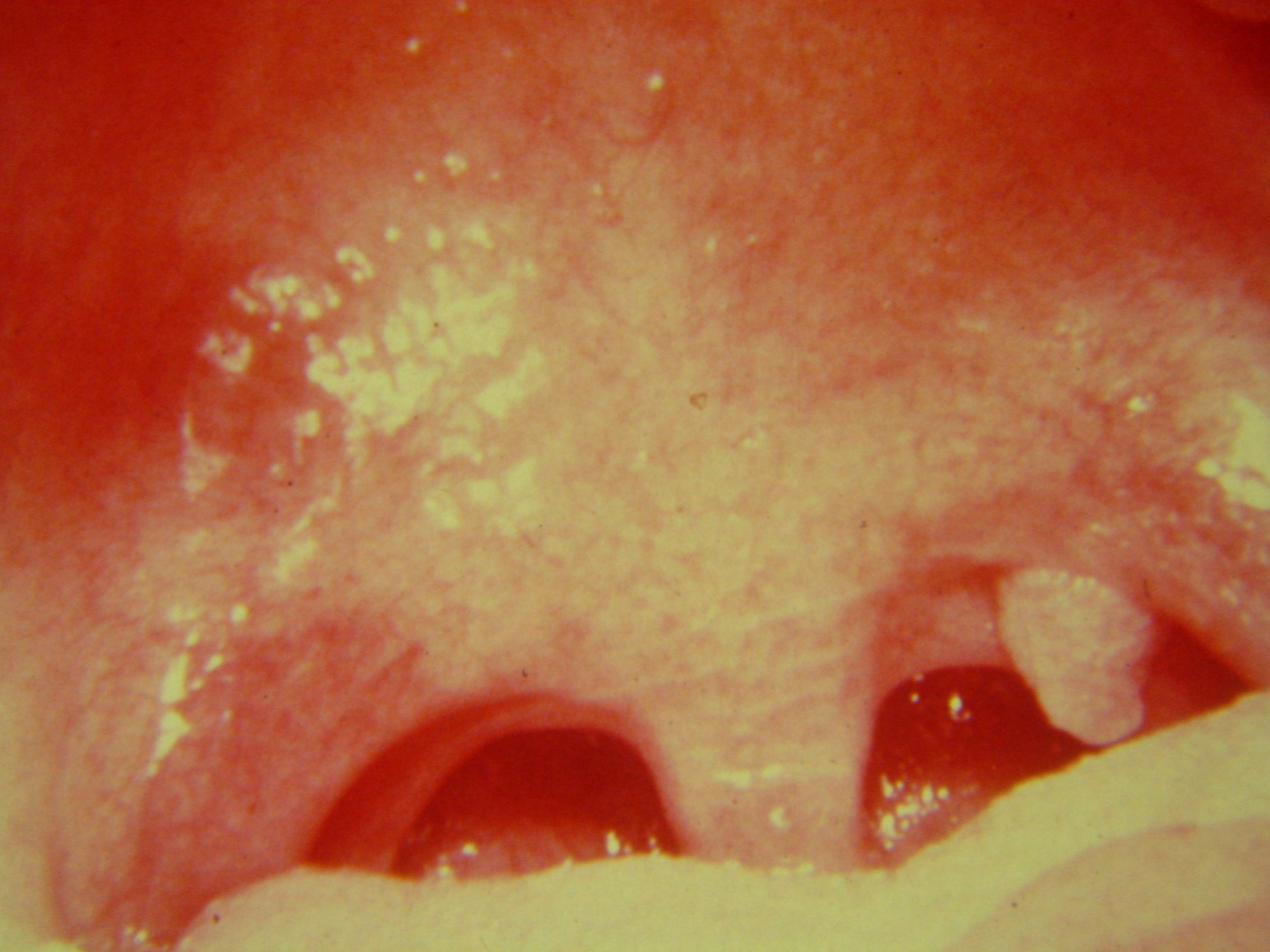
*Papilloma

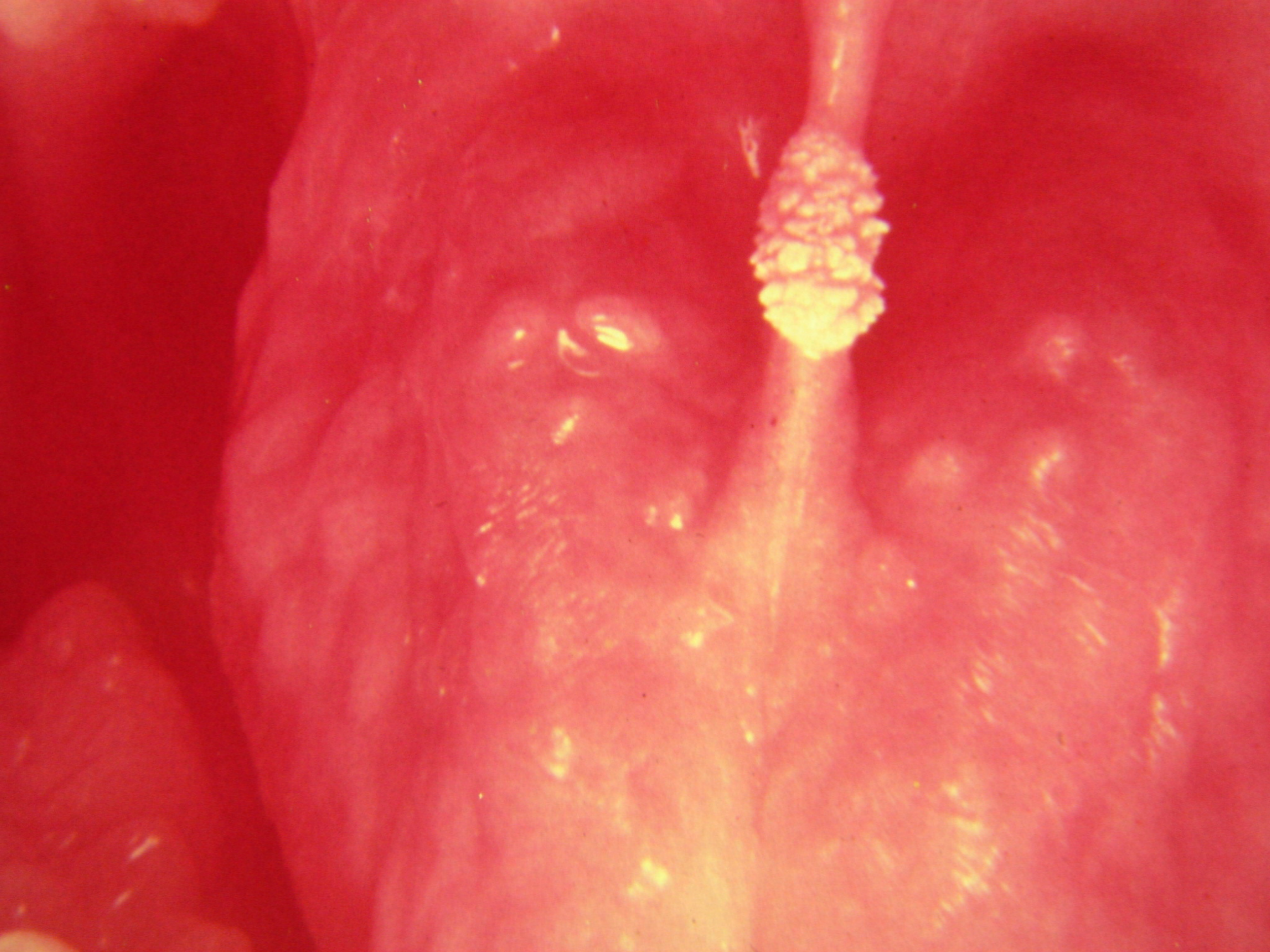
*Squamous acanthoma

*Keratoacanthoma

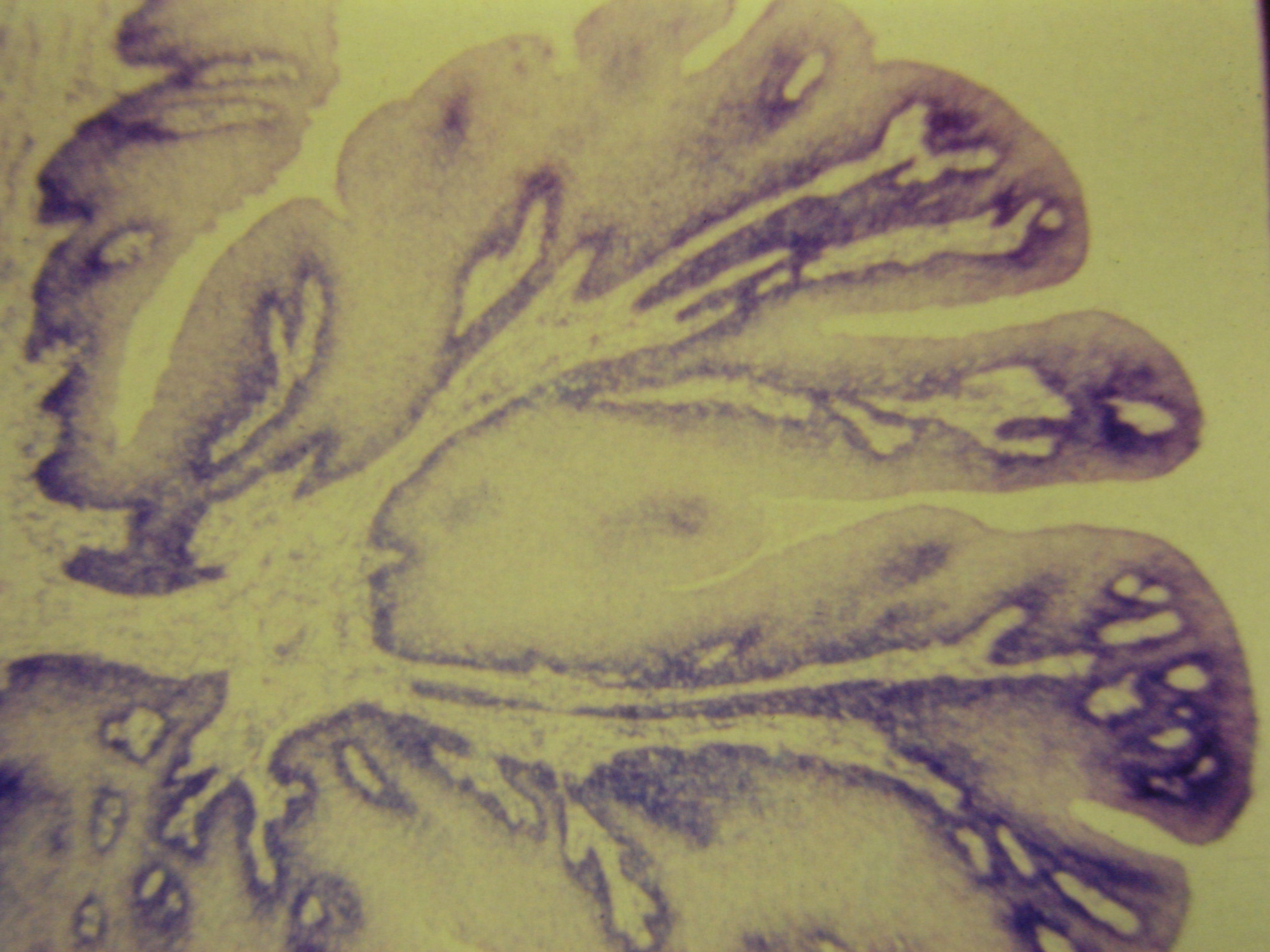
*Pigmented cellular nevus

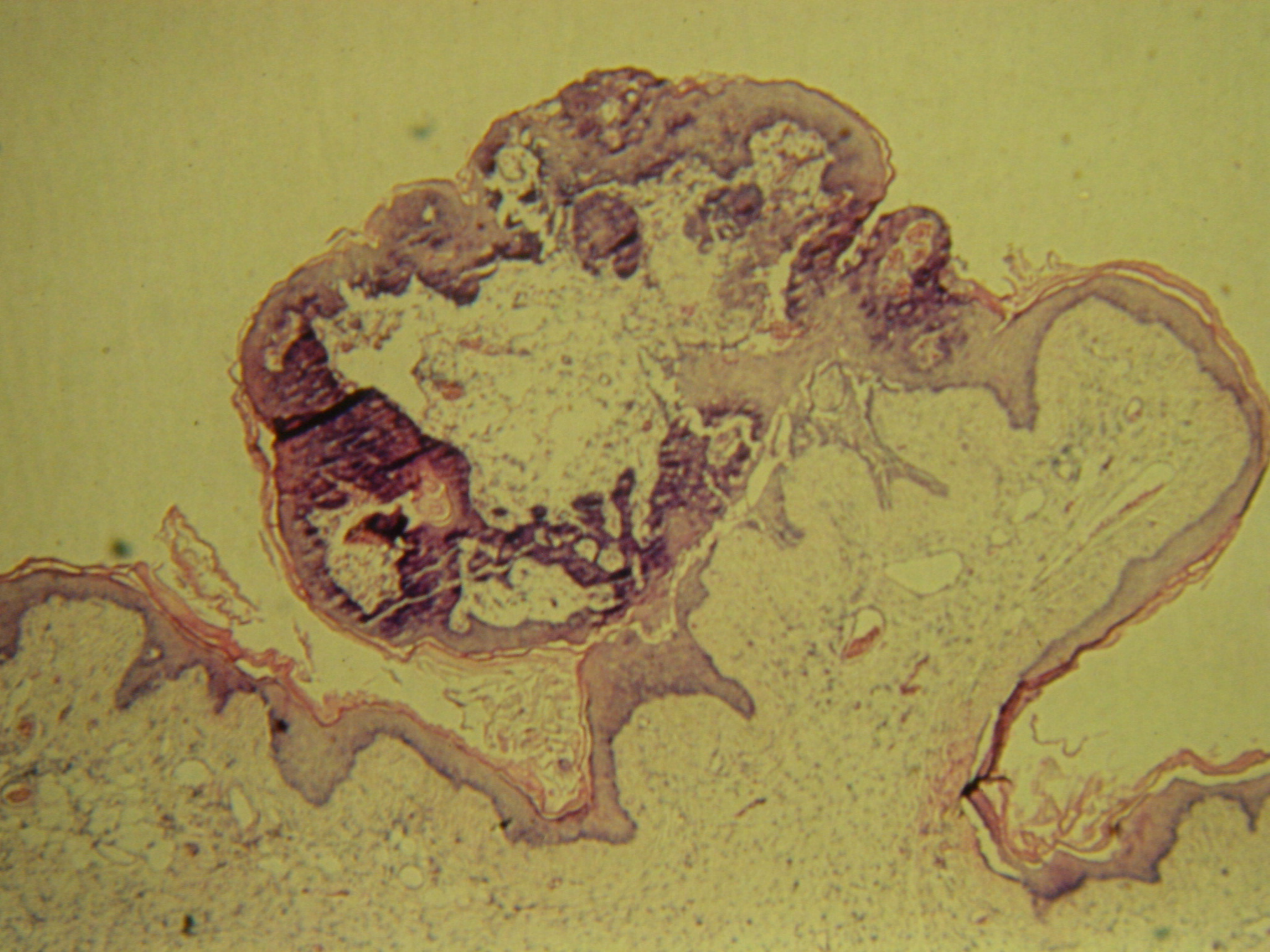


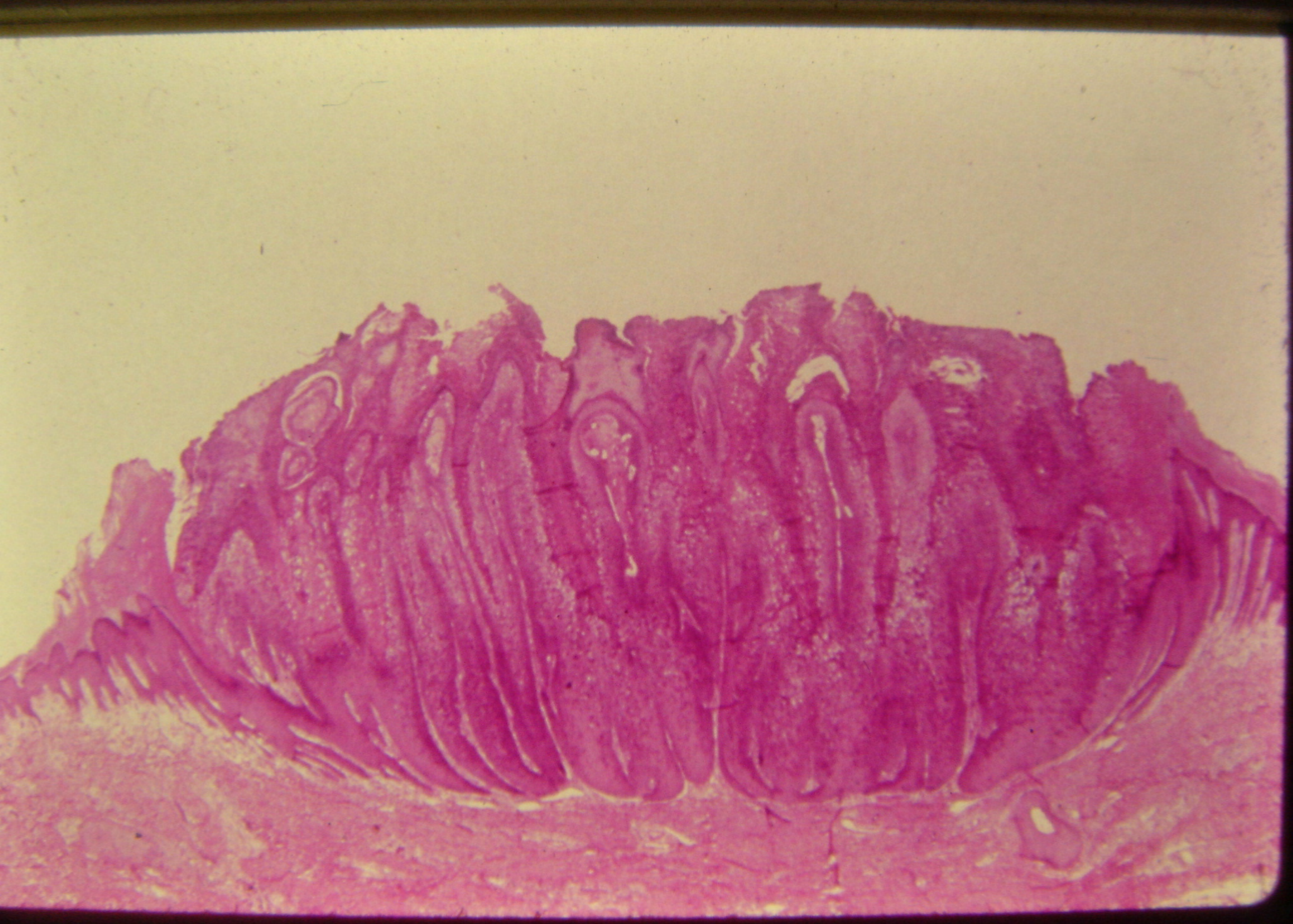


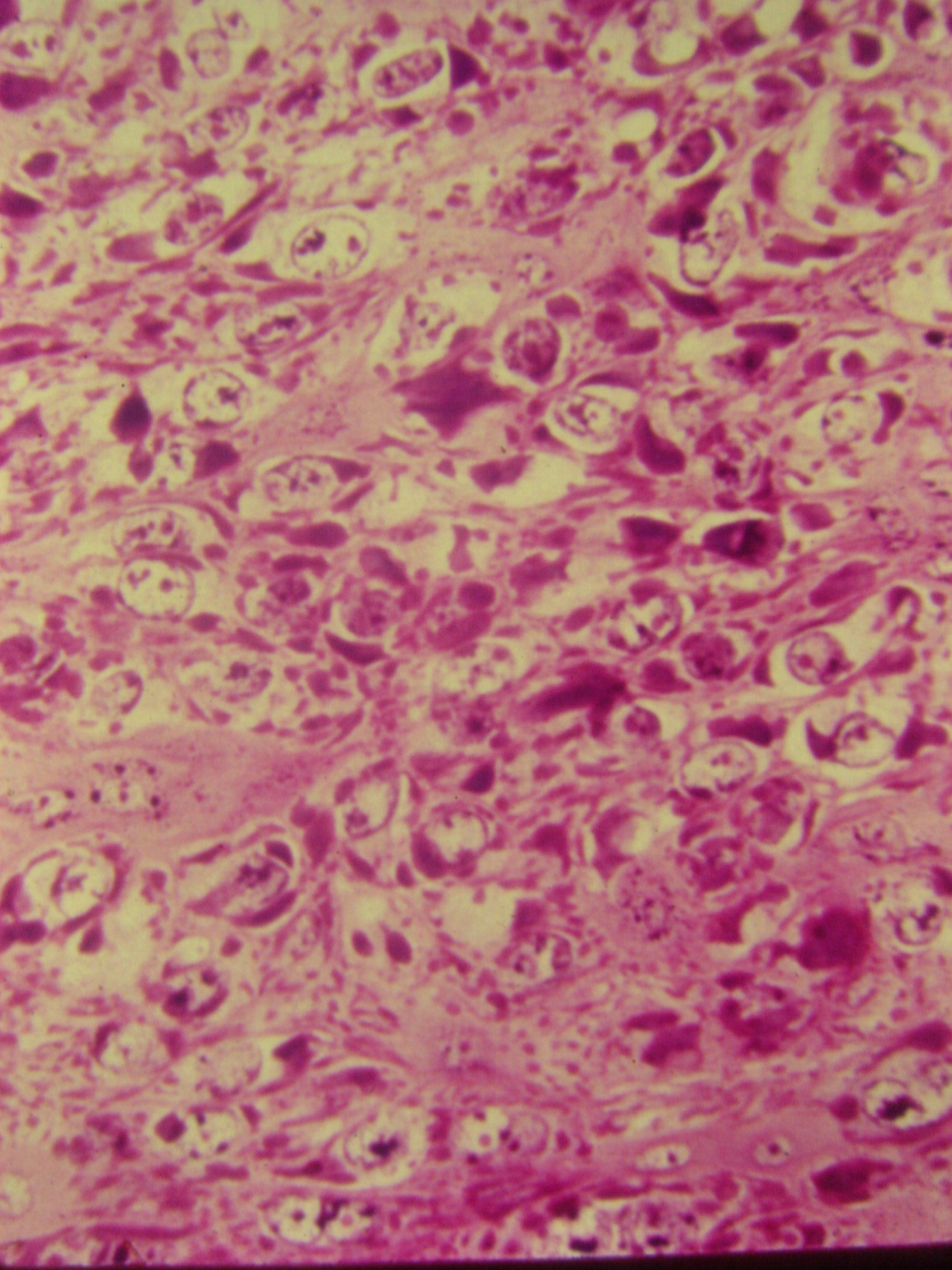












zoster or shingles. It is significant that the first three of these viruses have also been implicated in certain malignant diseases in the human as well: (1) EBV—the African jaw (Burkitt's) lymphoma and nasopharyngeal carcinoma, (2) CMV—Kaposi's sarcoma, and (3) HSV type II—carcinoma of the uterine cervix. To date, VZV has not been linked to any human malignant neoplasms.

The type II herpes simplex virus, which at one time was most commonly associated with herpes genitalis, is very similar in many biologic characteristics to type I herpes simplex virus, which at one time was most commonly associ-

Keratoacanthoma

*Resembles epidermoid ca.

*Genetic & viral factors

*Man-woman(50-70 yr)

*90% on skin, cheek, nose, hand

*elevated umbilicated or crateriform
c̄ a depressed central core or plug

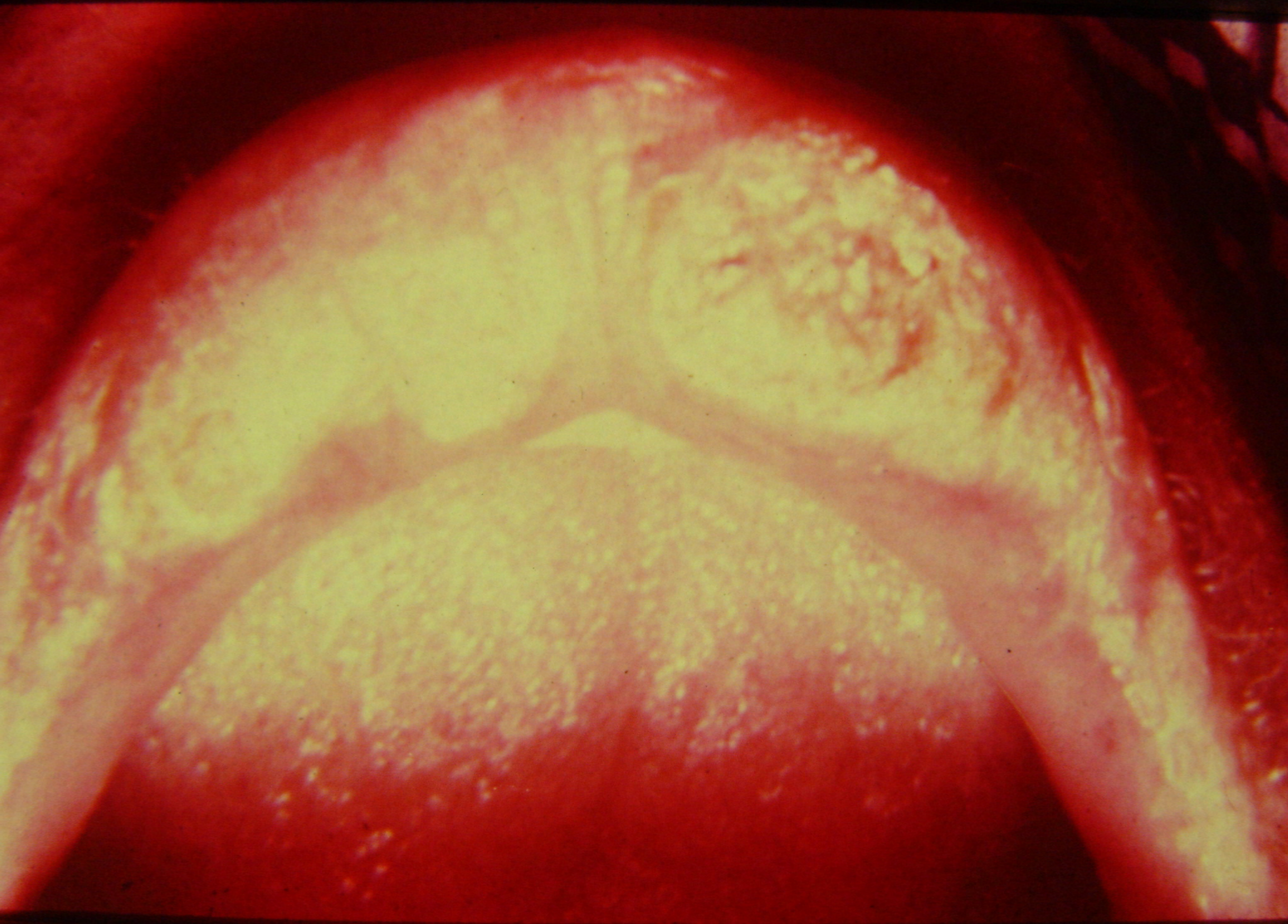
*1.0-1.5cm in diam.,painful,lymphaden.

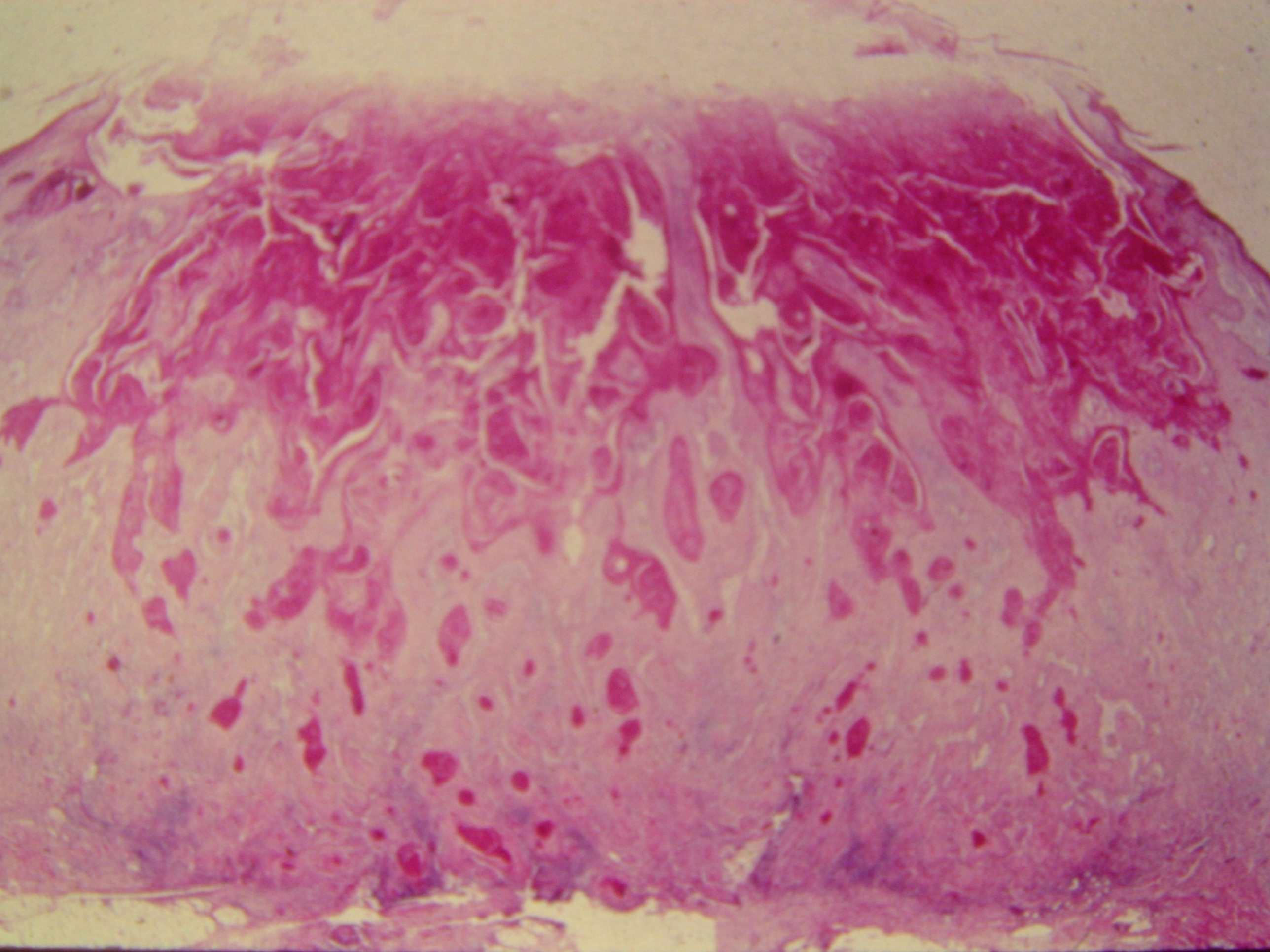
*Histo- hyperplastic sq. epi. growing into underlying connective tissue

*Para or orthokeratin c̄ central plugging

*Normal adjacent epi. elevated, abrupt change

*Tx:surgical excision, spontaneous regression (not 100%)





Acquired nevi(appear 8 month of life)

1. Intradermal nevus(common mole) 55%
2. Junctional nevus 3%
3. Compound nevus 6%
4. Spindle cell and/or epithelioid cell nevus /%
5. Blue(Jadassohn-Tieche) nevus common & cellular blue nevus 36%





Congenital nevi

*"small" nevi 3-5cm to 1cm in diam.

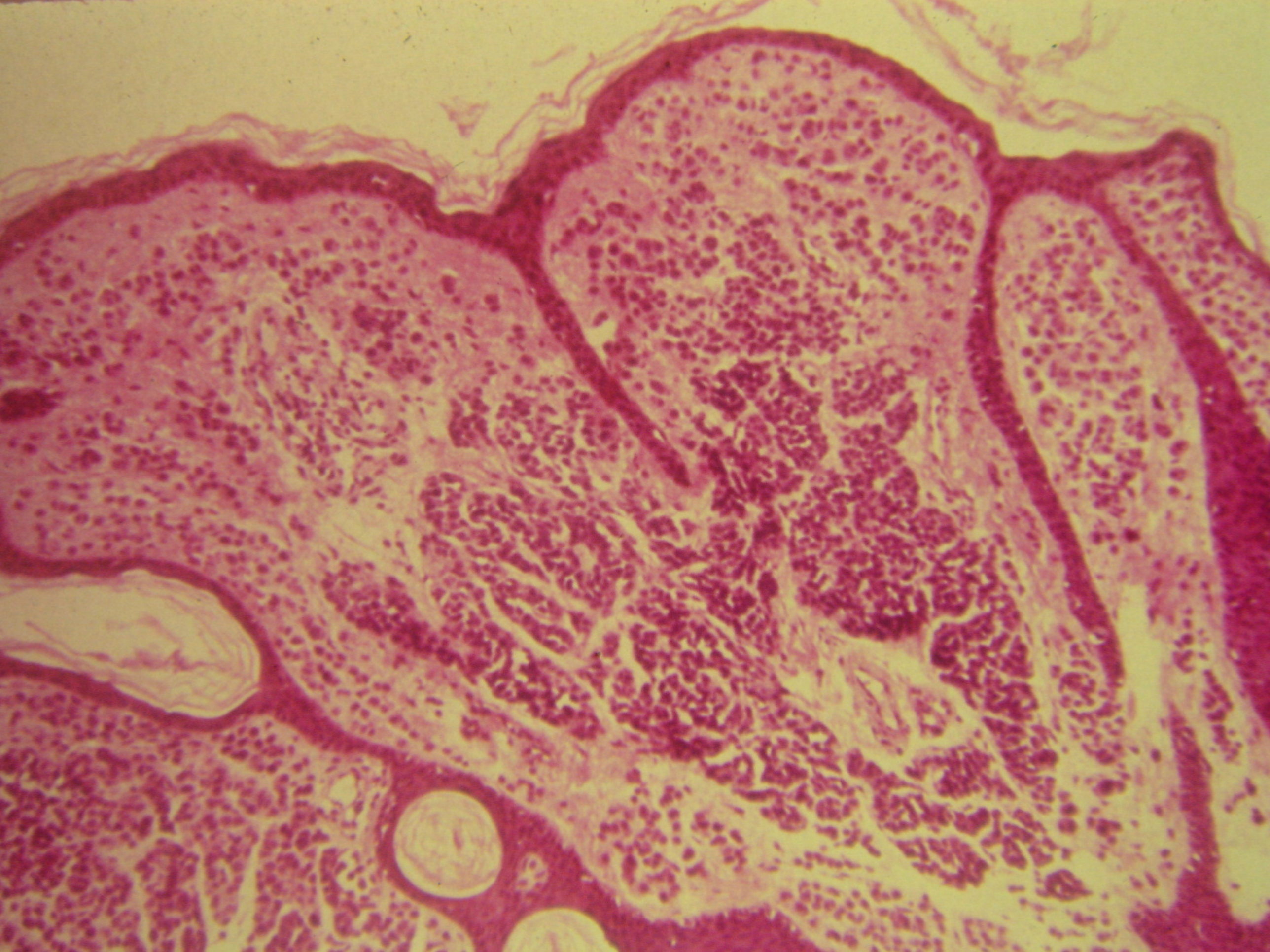
*"garment" nevi ≥10cm(14% malig. trans)

*occur in 1 to 2.5% of neonate

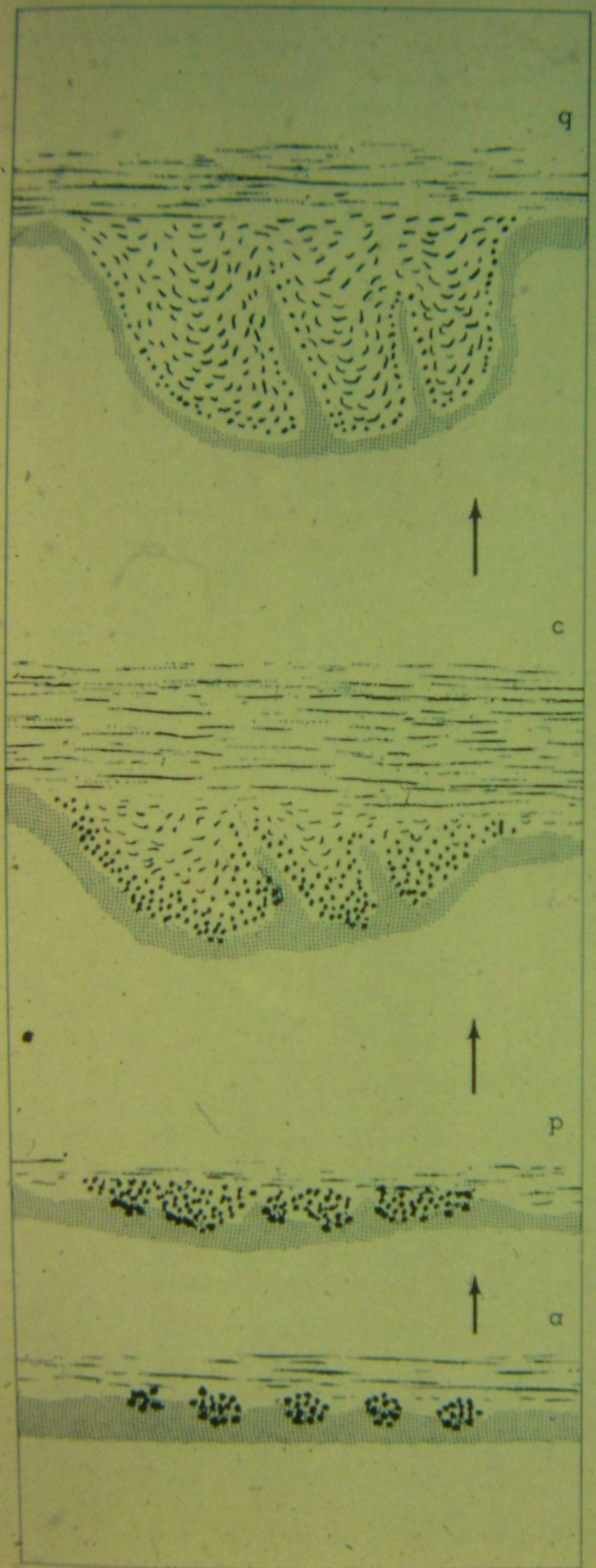
*D.D. melanotic macule, premalignant melanosis, amalgam tattoo

Histology

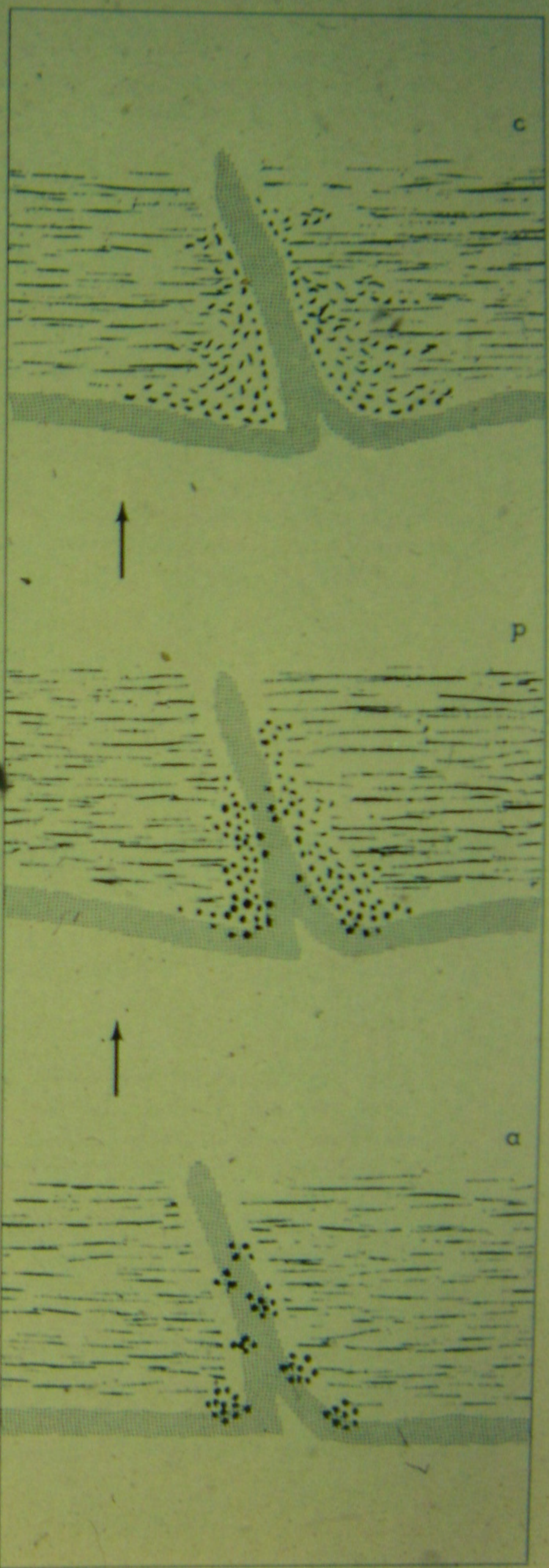
* cells growing in solid sheets, cords & nests.
* large, round or polyhedral, mitotic activity
* syncytial pattern (multinuclei mass) stroma infiltrated by lymphocytes
* large & polyhedral with indistinct outlines.
* cytoplasm stains lightly eosinophilic.

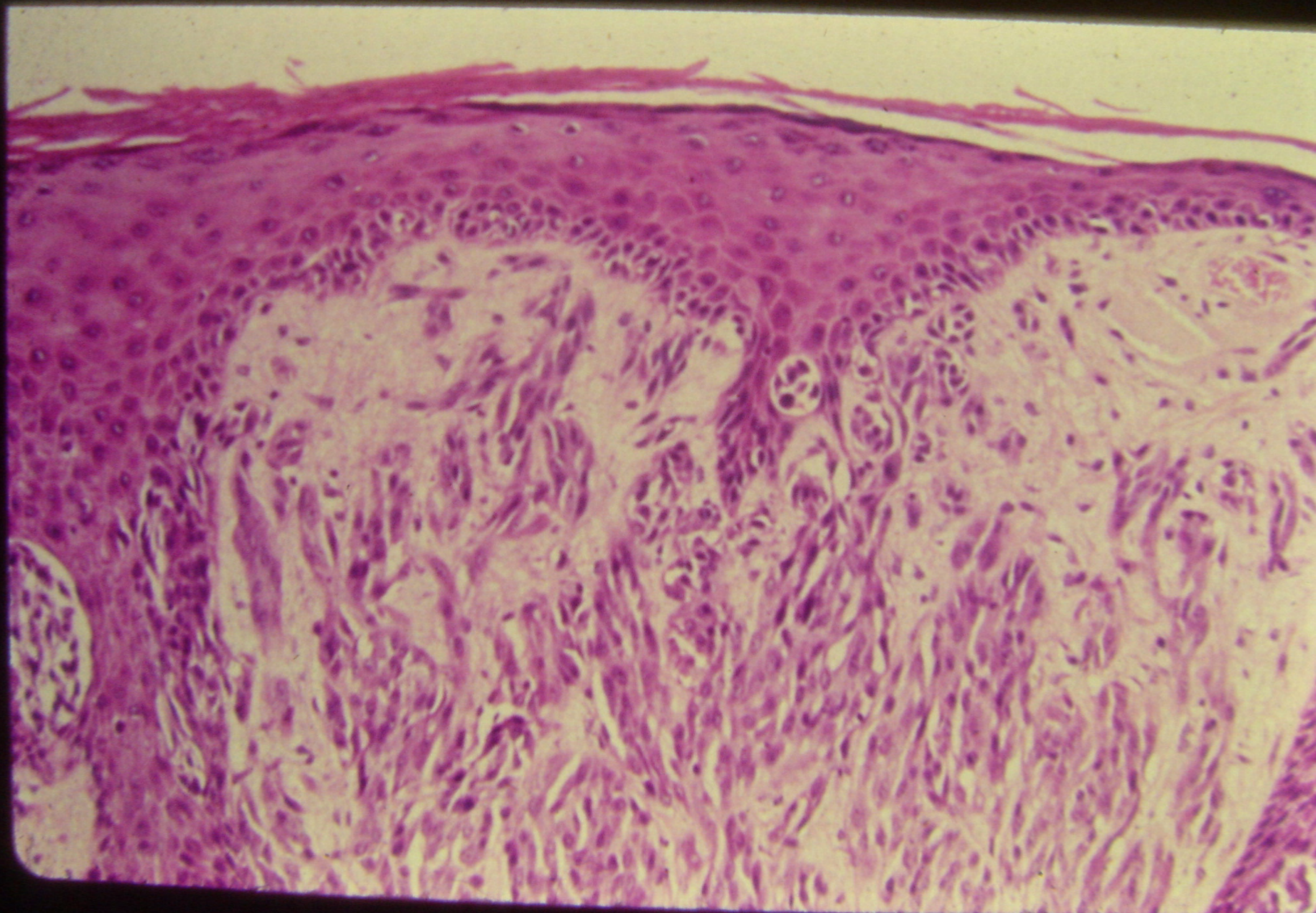


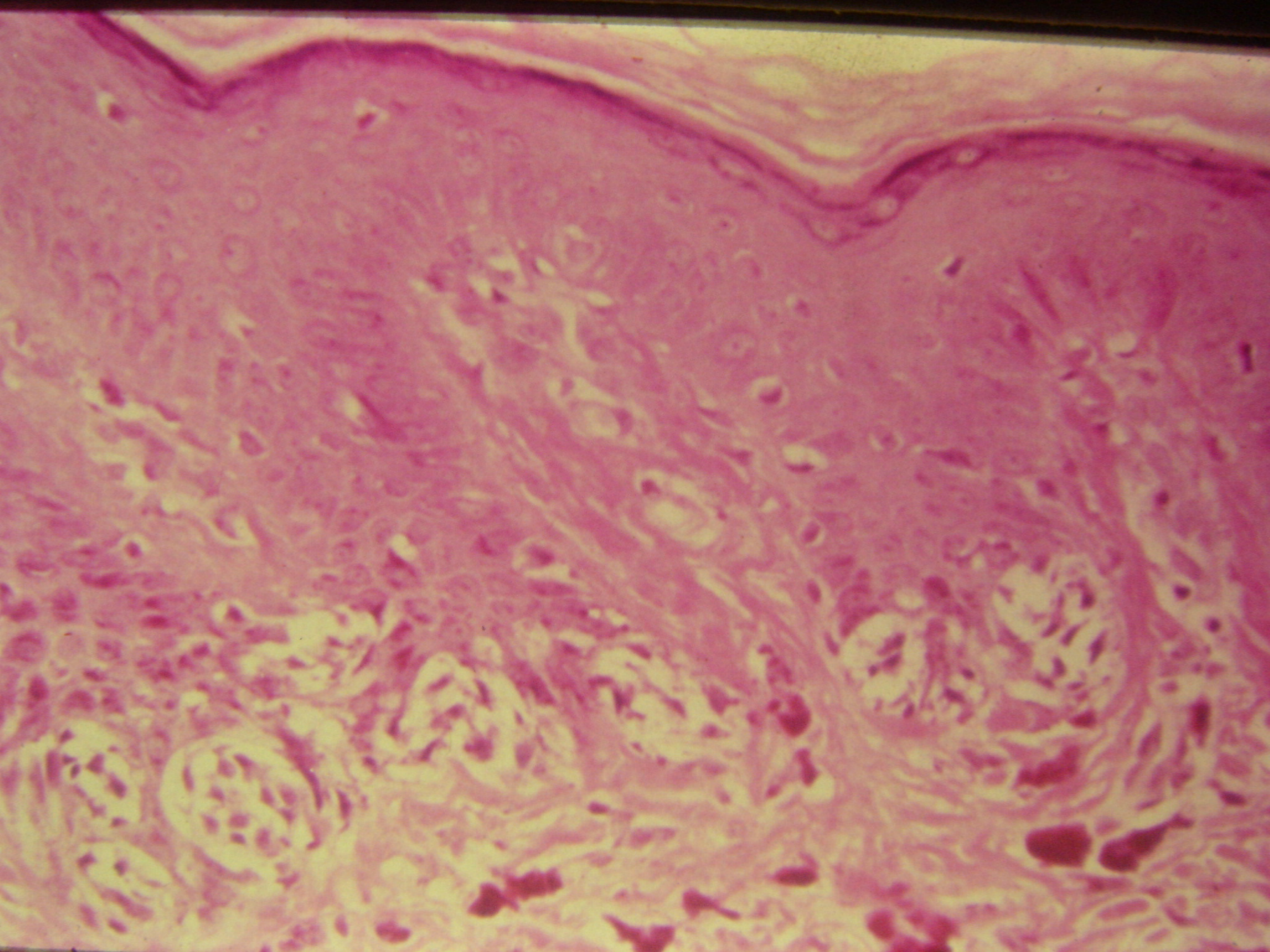
Lid. 32

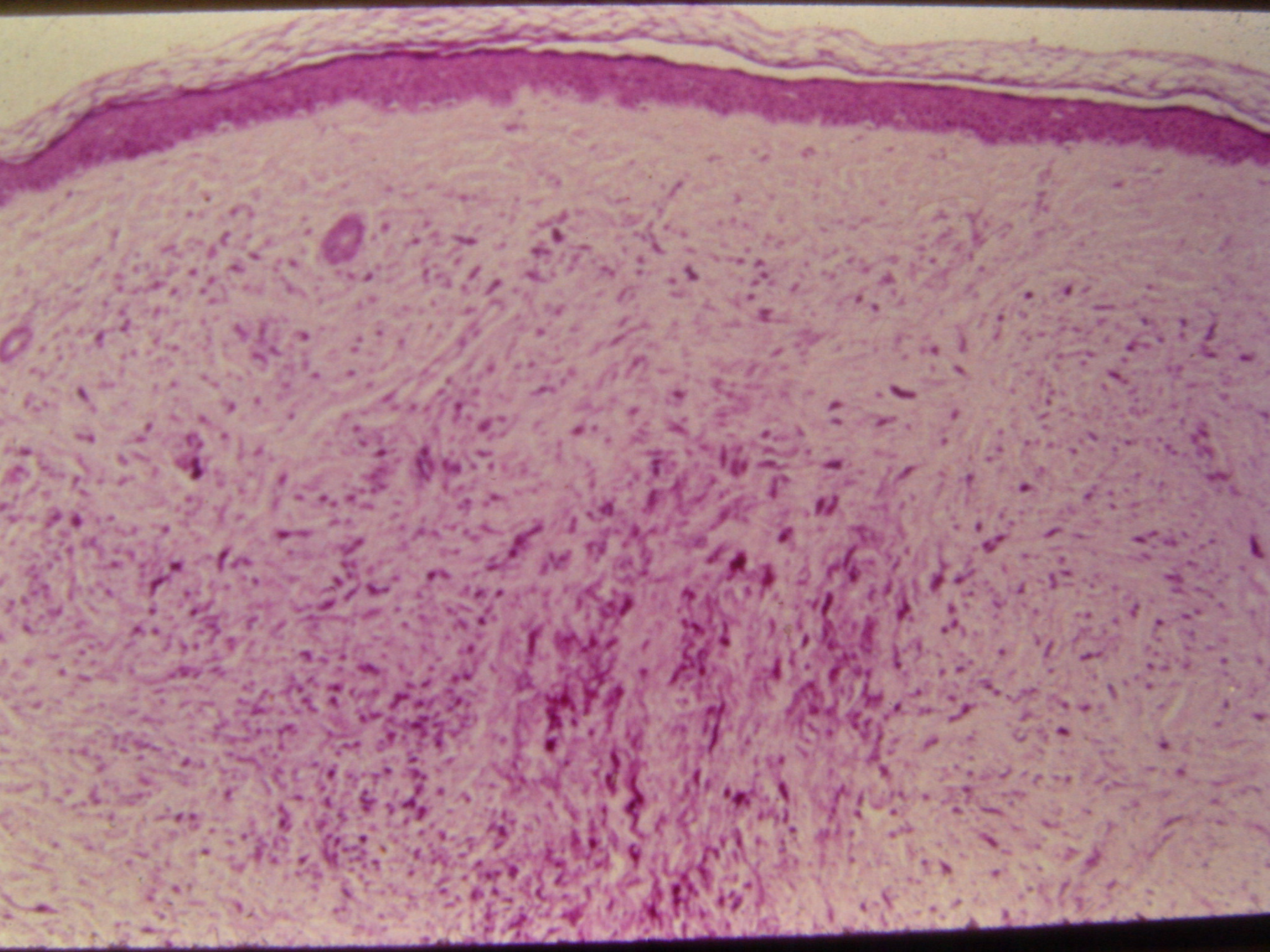


Lid. 33









"PREMALIGNANT" LESIONS OF EPITHELIAL TISSUE ORIGIN

*Leukoplakia

*Leukoedema

*Intraepithelial carcinoma
(Carcinoma in situ)

*Erythroplakia

*Oral submucous fibrosis

Leukoplakia

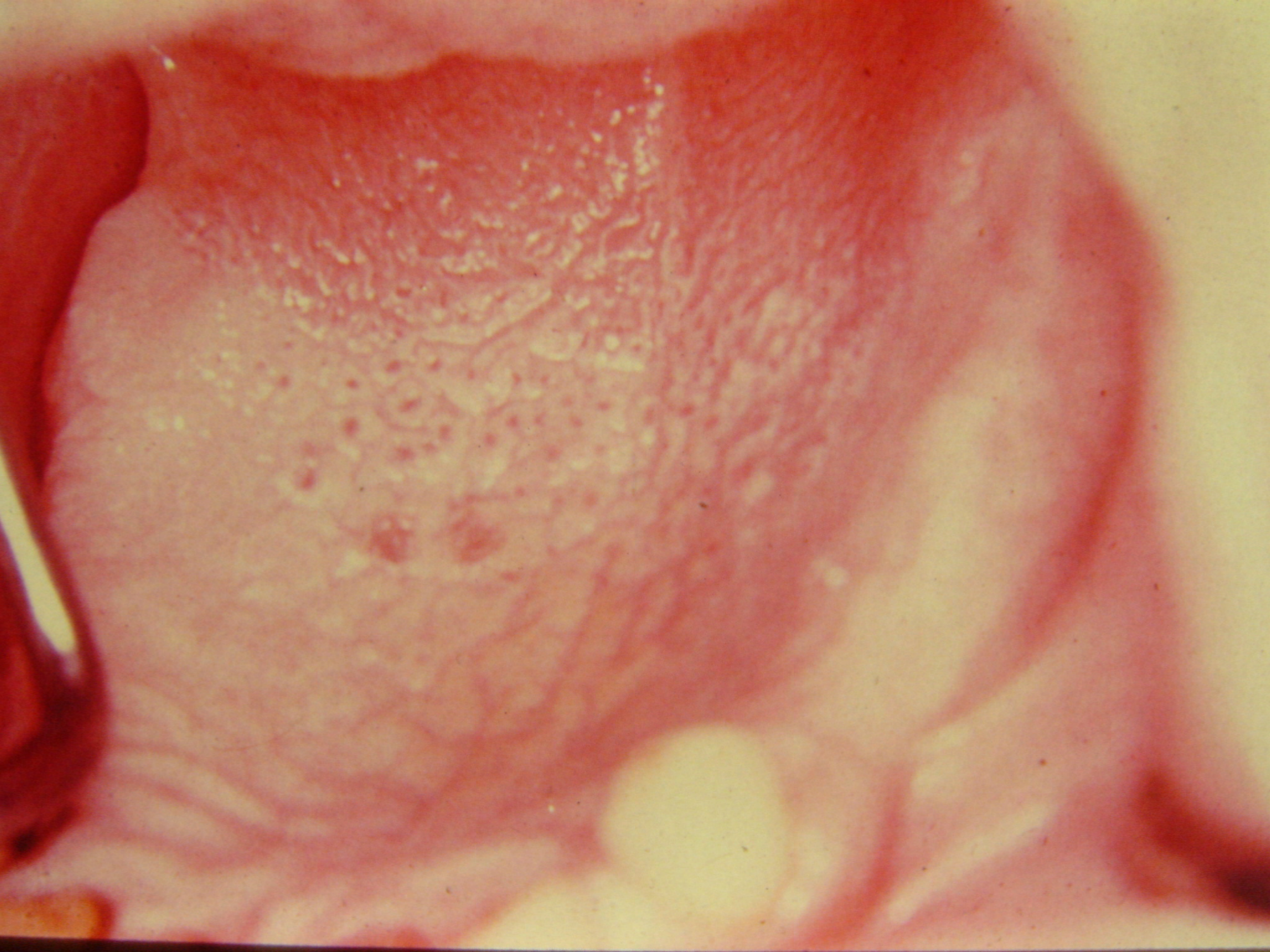
*White patch or plaque

*Oral m.m.,vulva, uterine cervix

*Urinary bladder, renal pelves,URT

*D.D.:L.P.,syphilis,white spongy nevus,
moniliasis,L.E.,chemical burn

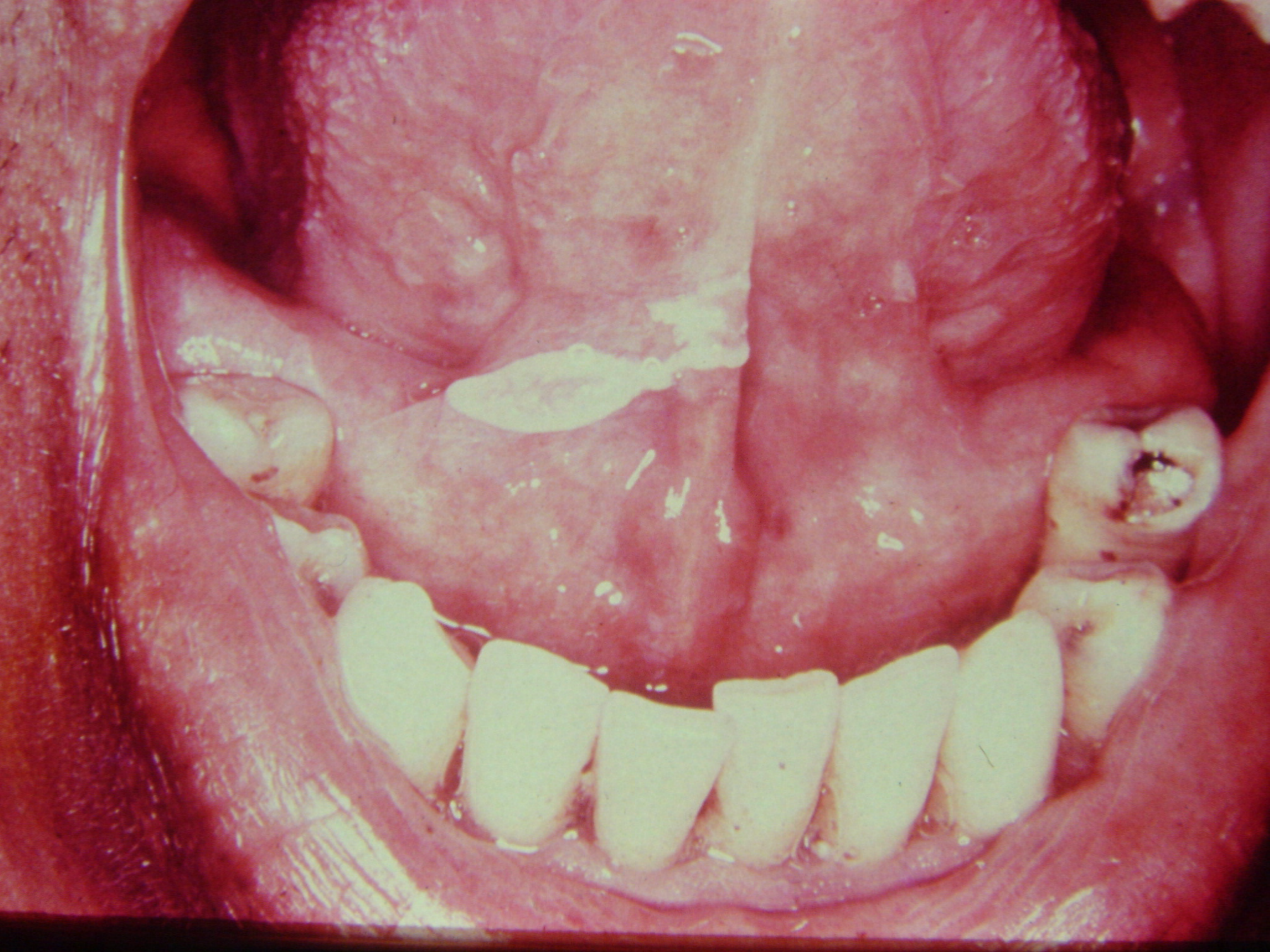
*Oral premalignancy

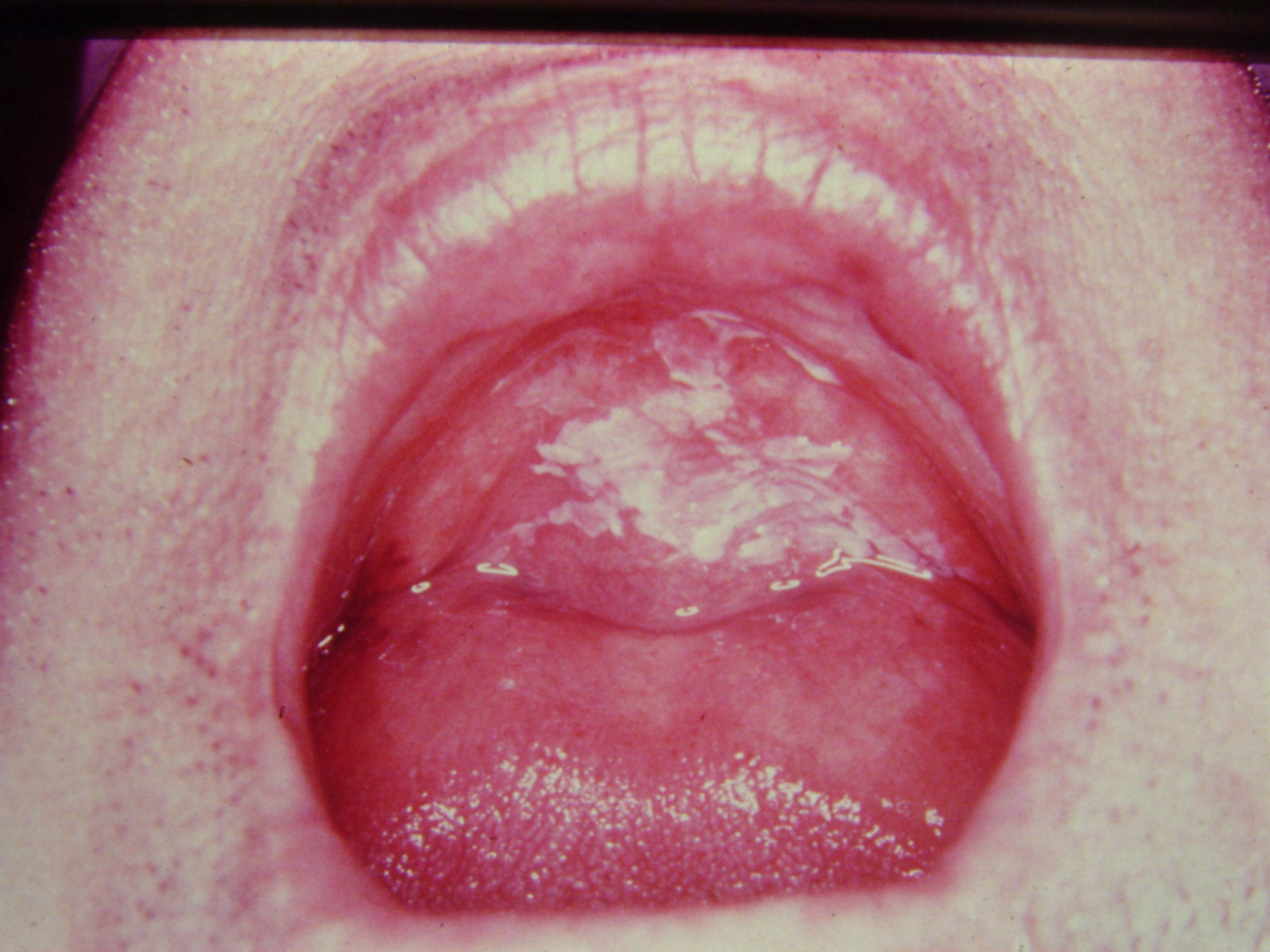






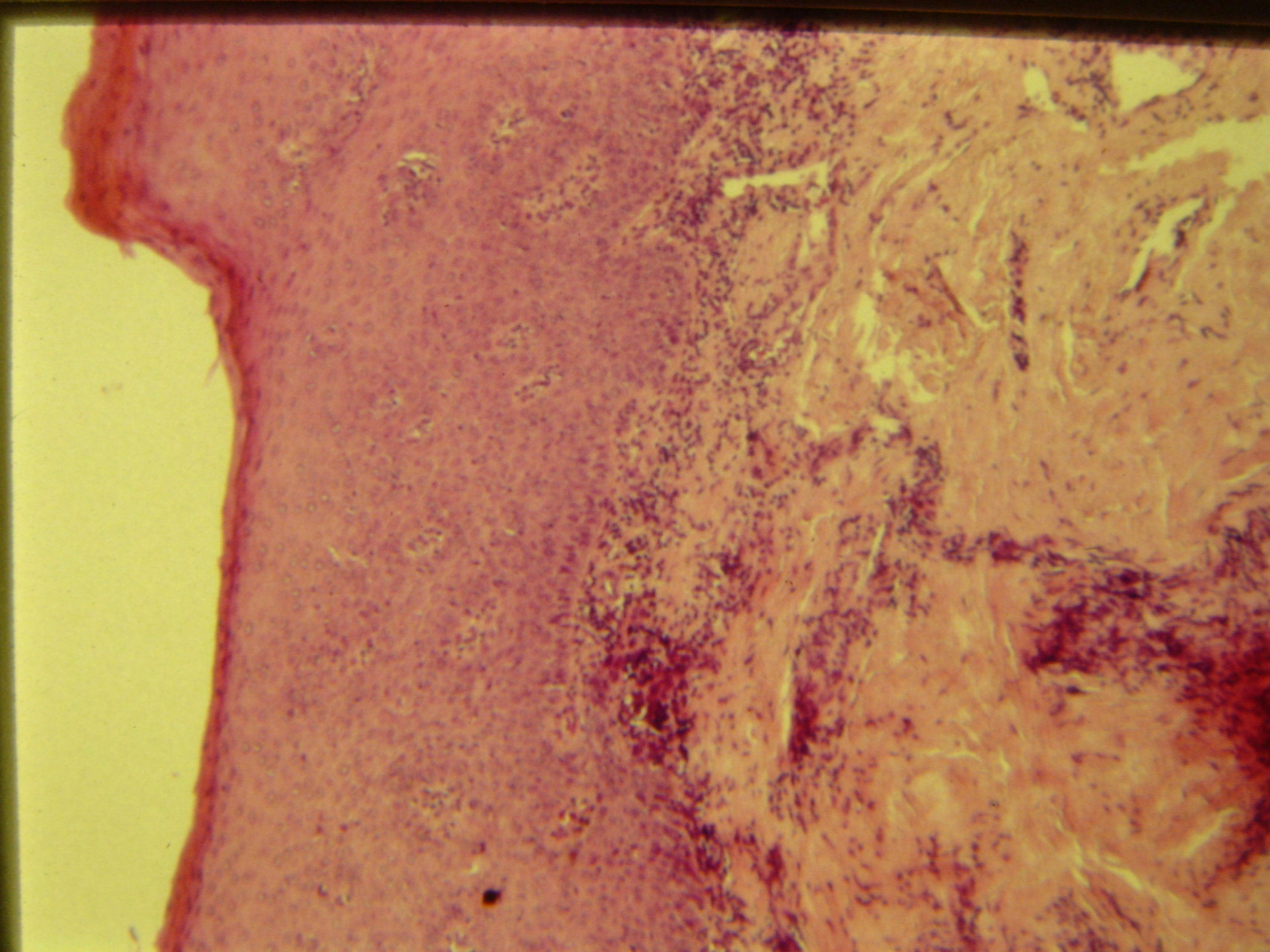


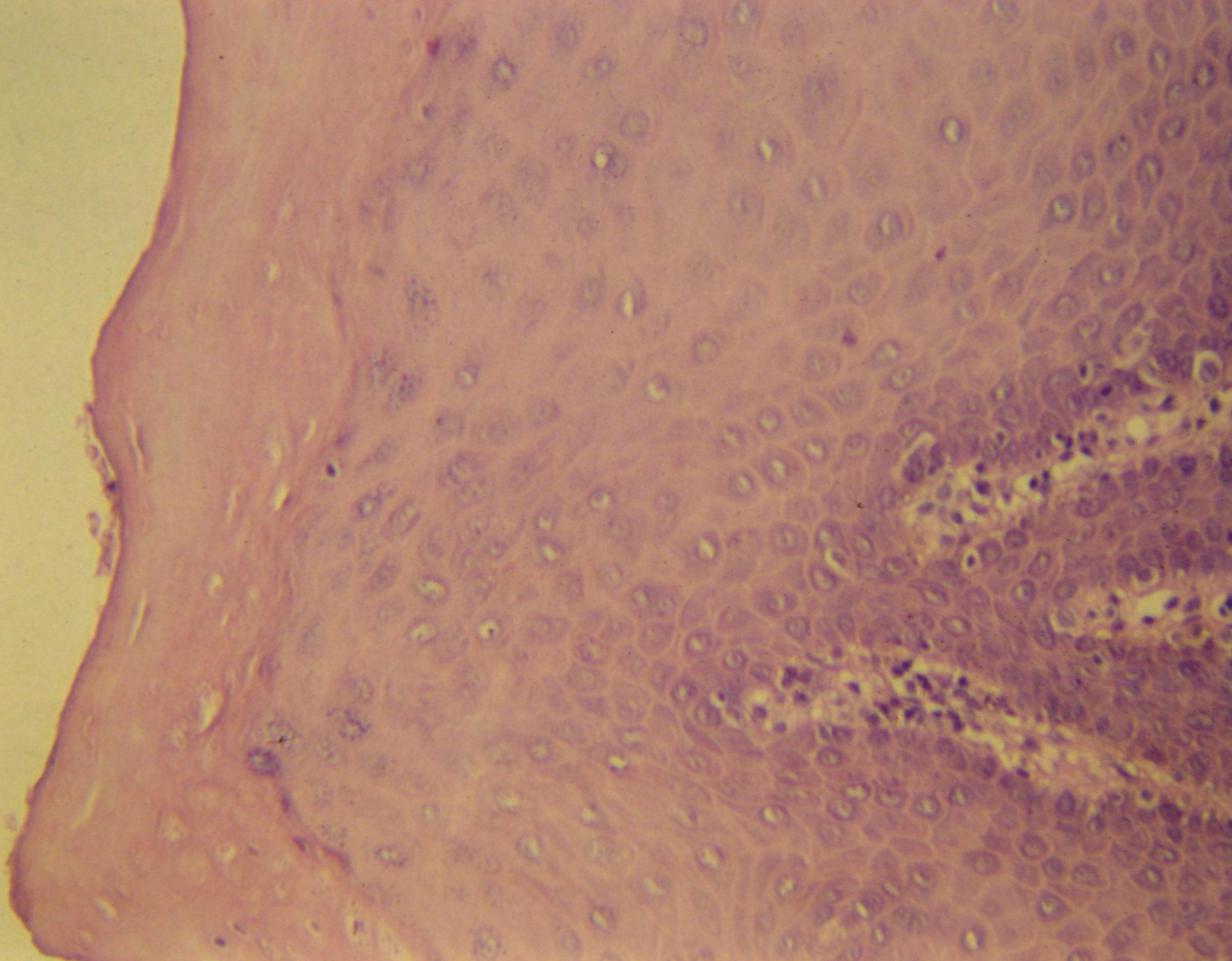


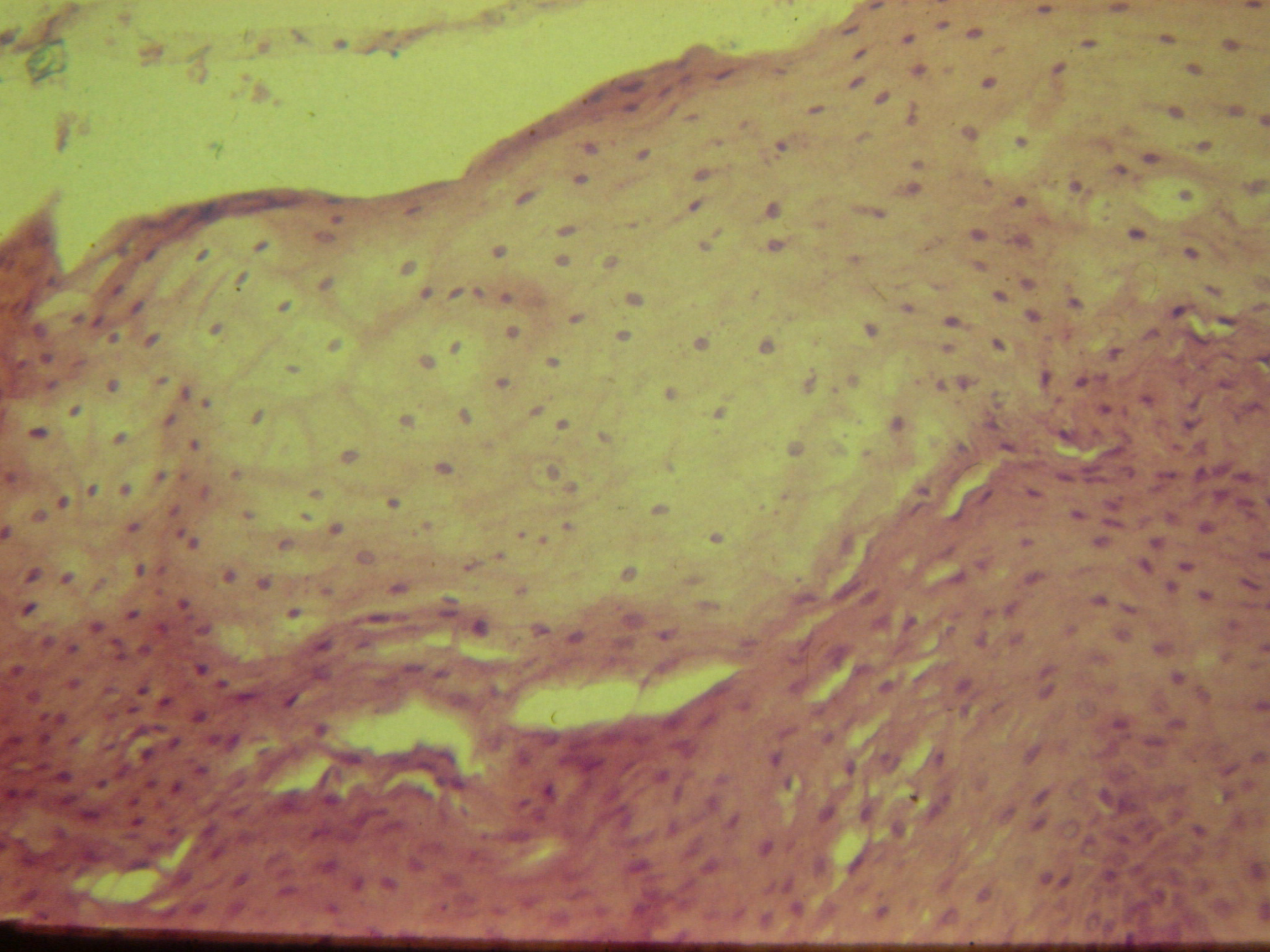


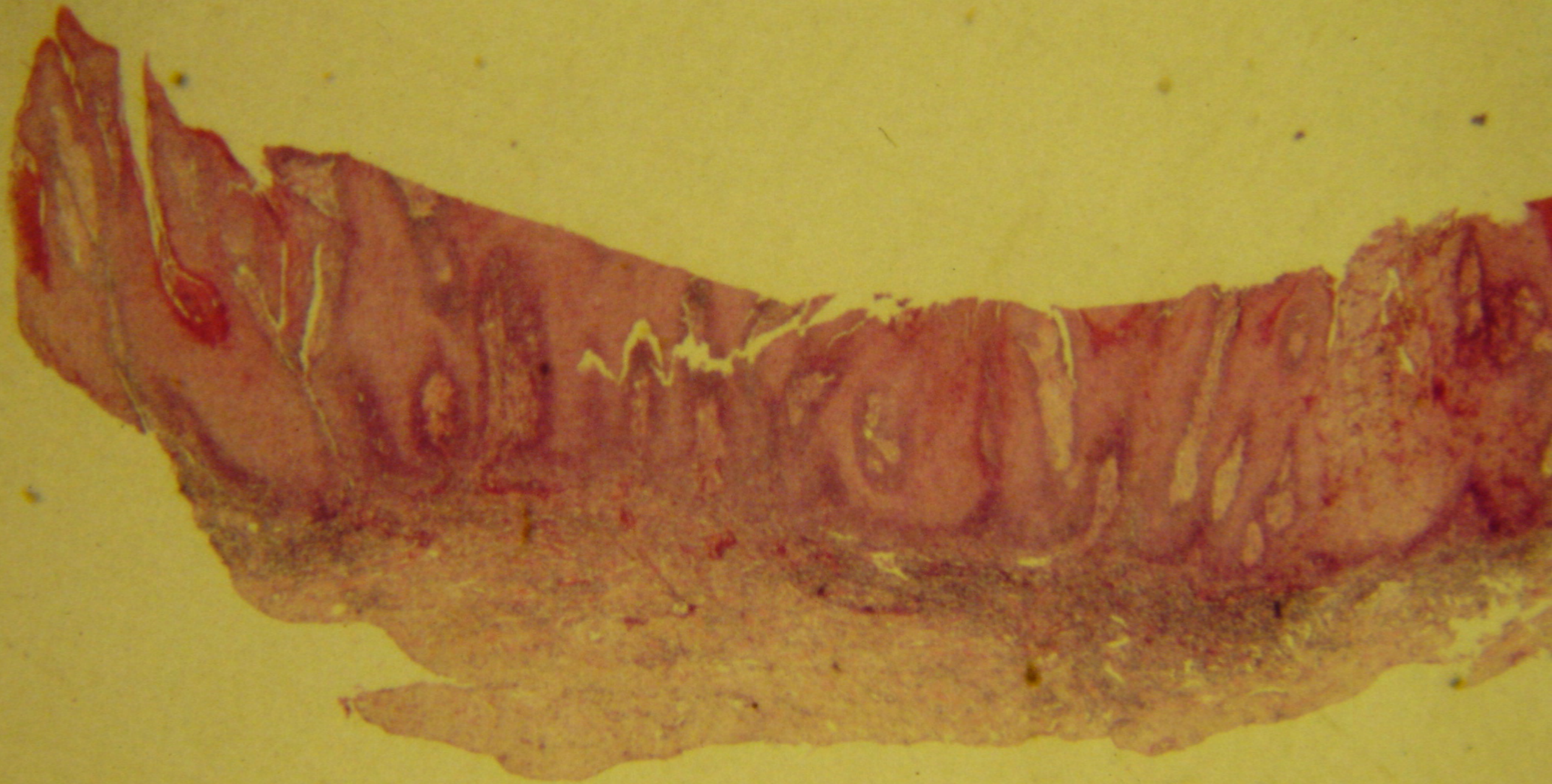
Histology Features

1. Hyperorthokeratosis
2. Hyperparakeratosis
3. Acanthosis
4. Dysplasia
5. Carcinoma in situ
   (intraepithelial ca.)









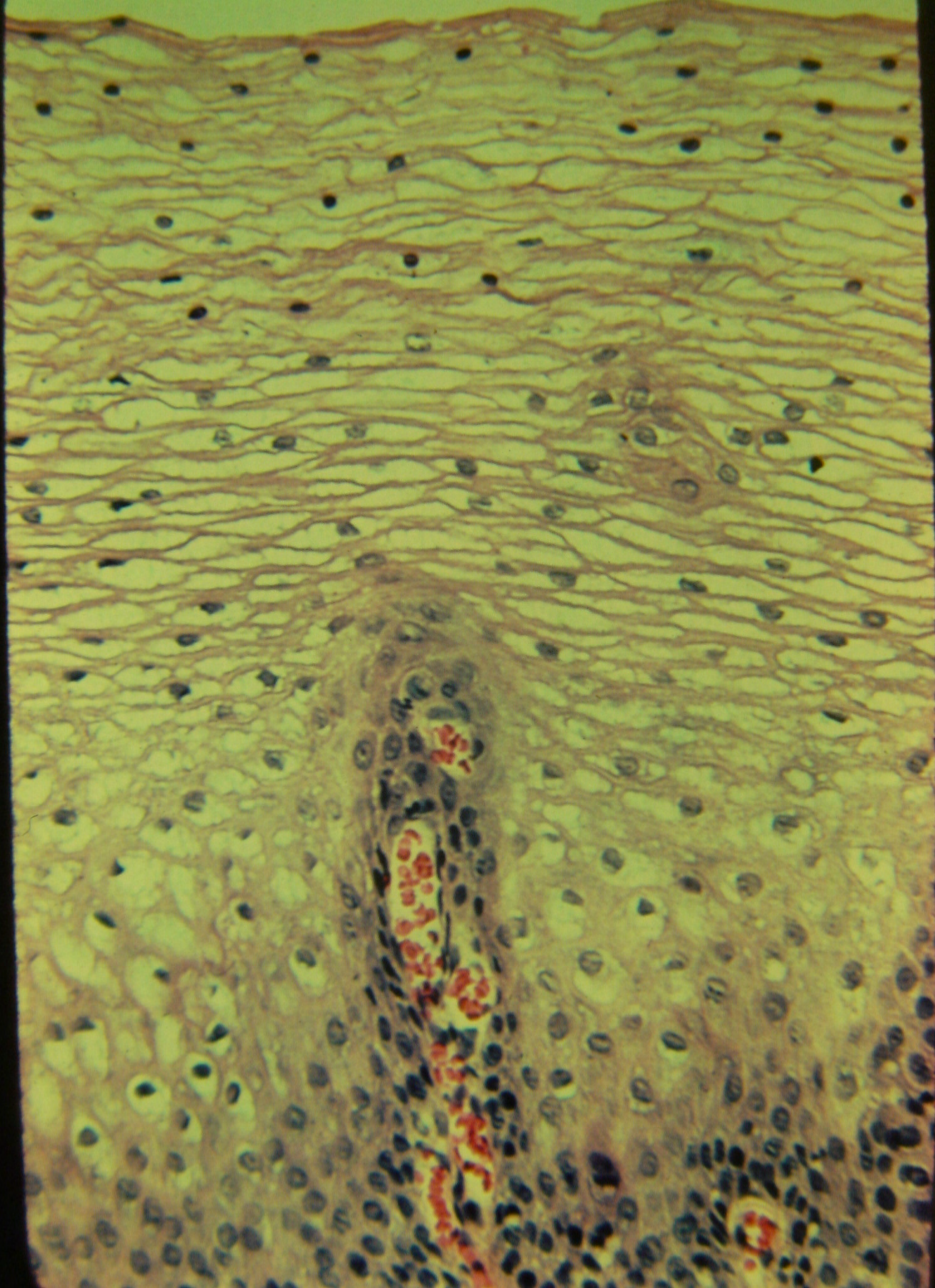
Treatment(leukoplakia)

1. elimination of any recognizable irritating factor
2. discontinuance of use of tobacco or alcohol
3. correction of any malocclusion

4. replacement of ill-fitting denture

5. administration of vit A,B complex, estrogens,x-ray therapy,fulguration. surgical excision,topical chemotherapy





Carcinoma in situ

1.frequently on the skin

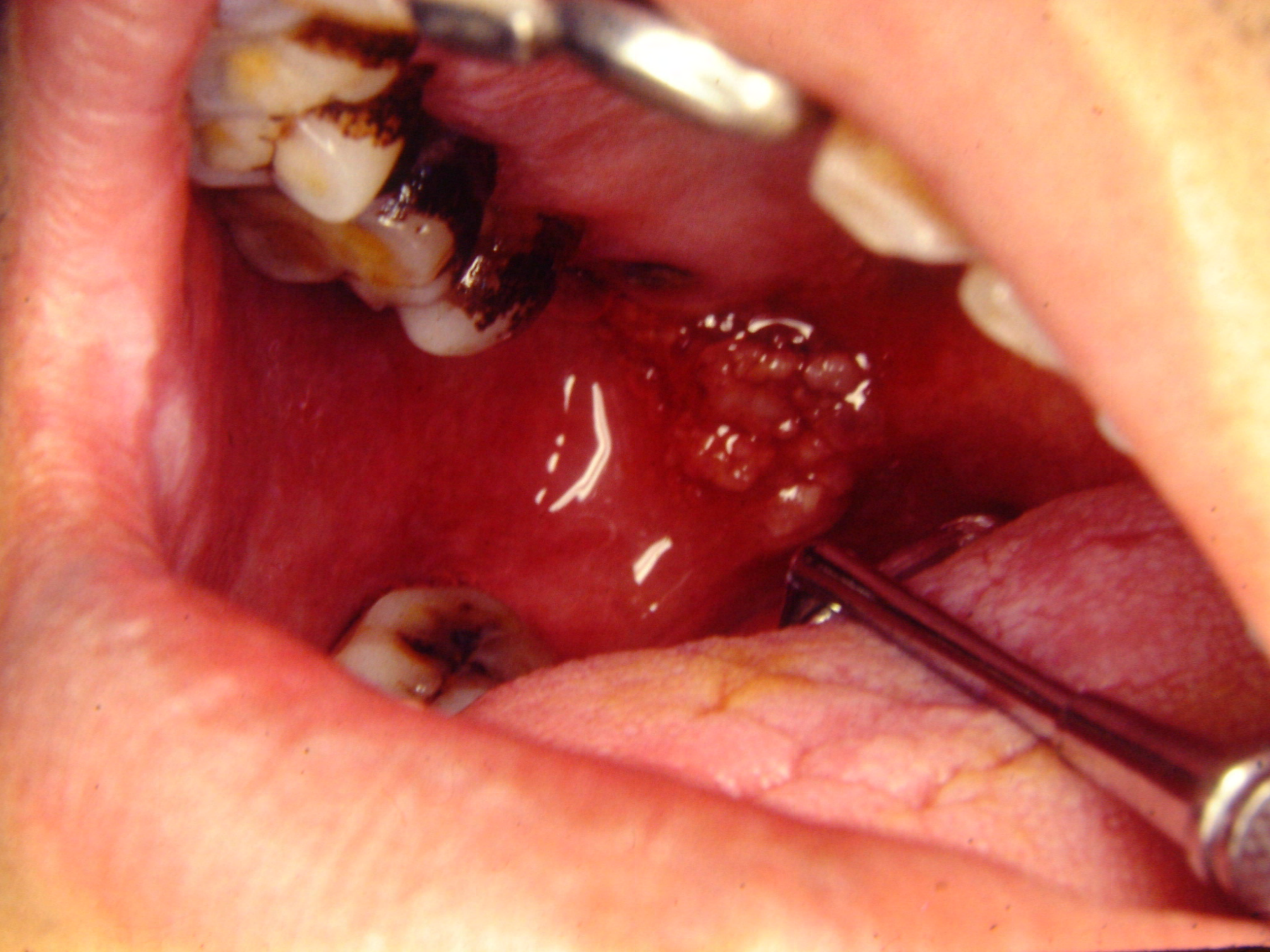
2.precancerous dyskeratosis process
or laterally spreading intraepi ca

3.Bowen's disease-arsenical therapy

4.floor of mouth(23%),tongue(22%),lip(20%)

5.OBGyn-spontaneous regression
(not in oral cavity)(??)





Carcinoma in situ

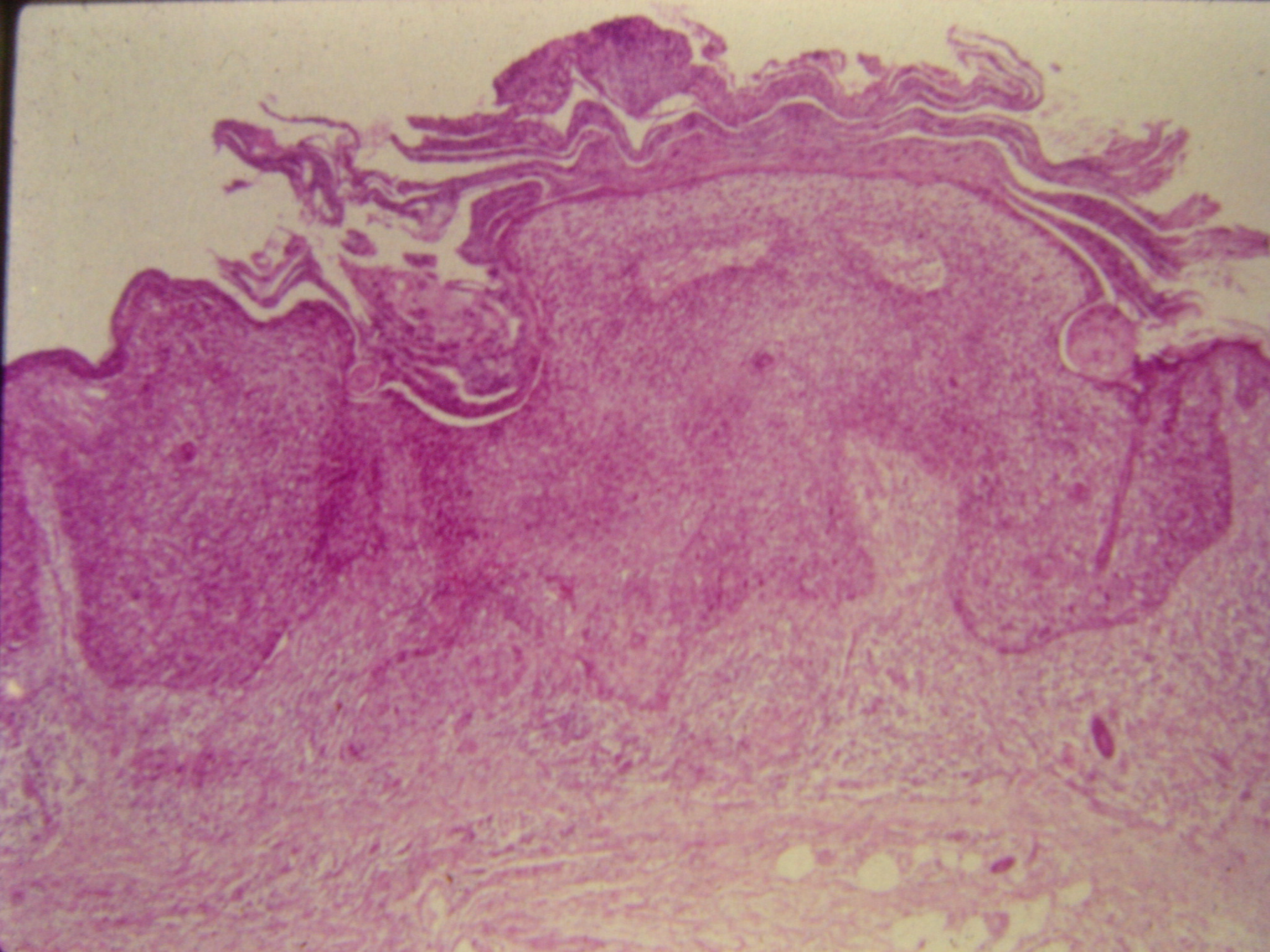
*"top to bottom" basilar hyperplasia

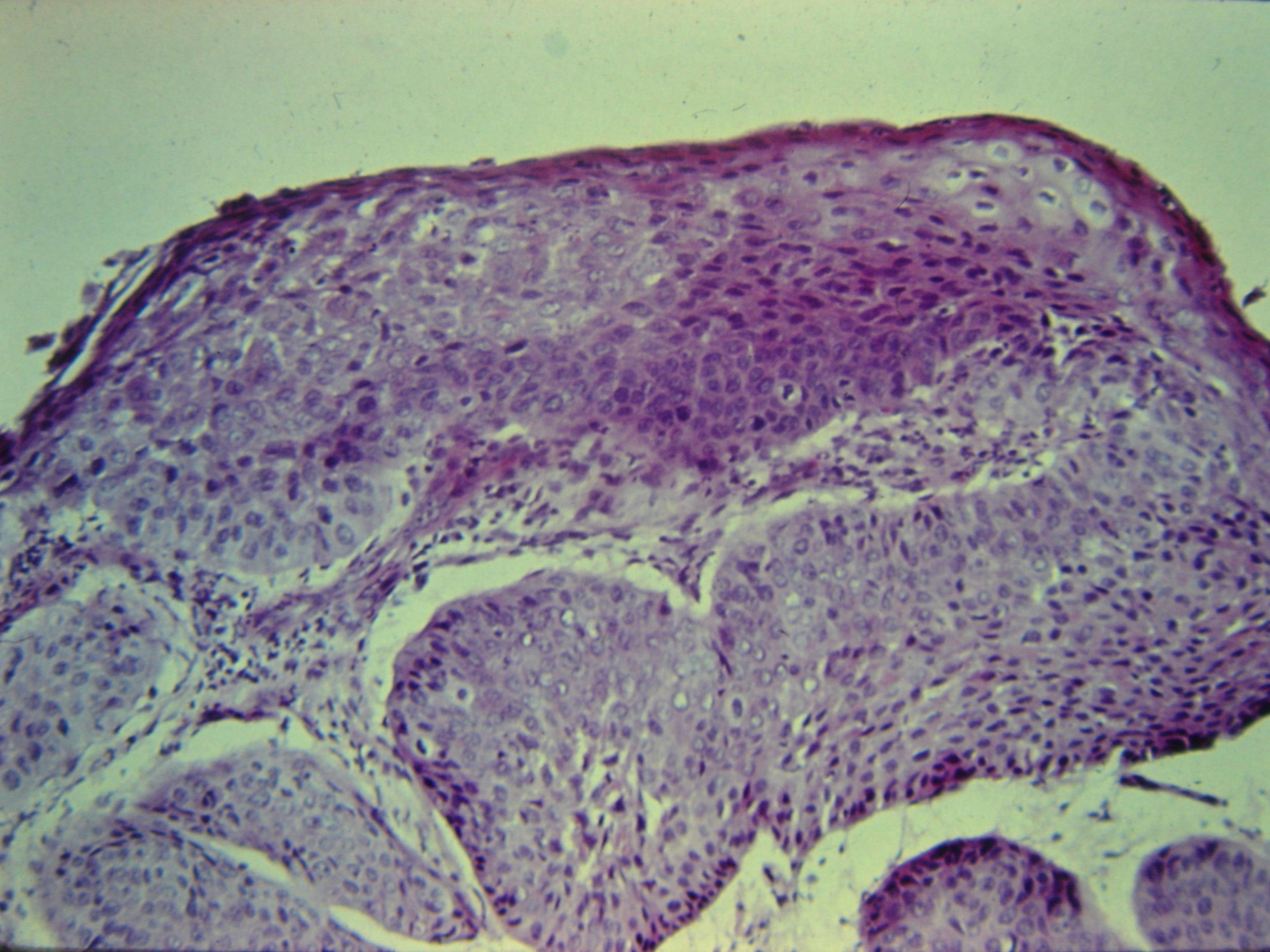
*loss of polarity,hyperchromatism,
pleomorphism(少)

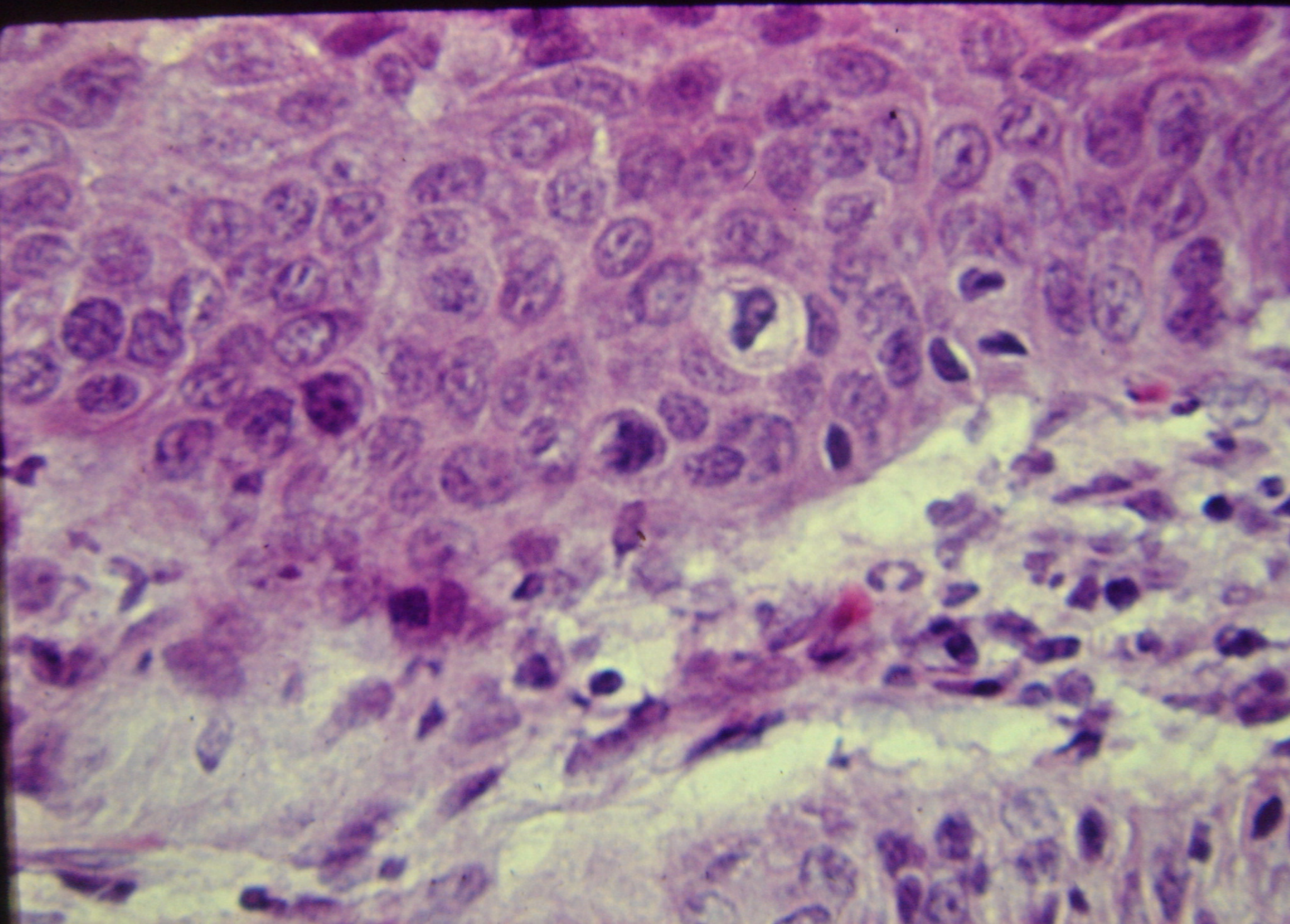
*increase mitosis, dyskaryosis,N/C chang

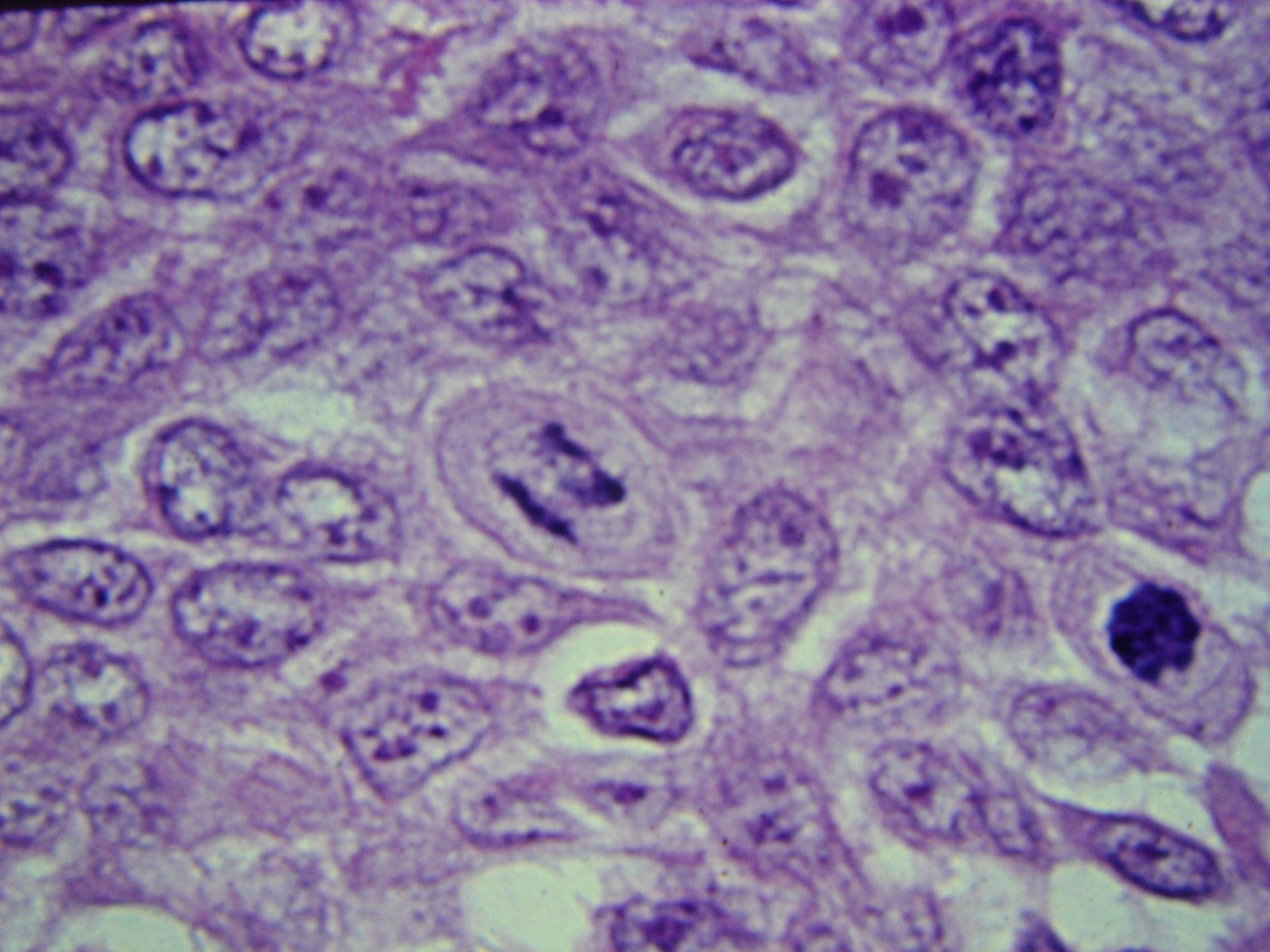
*speckled leukoplakia,erythroplakia

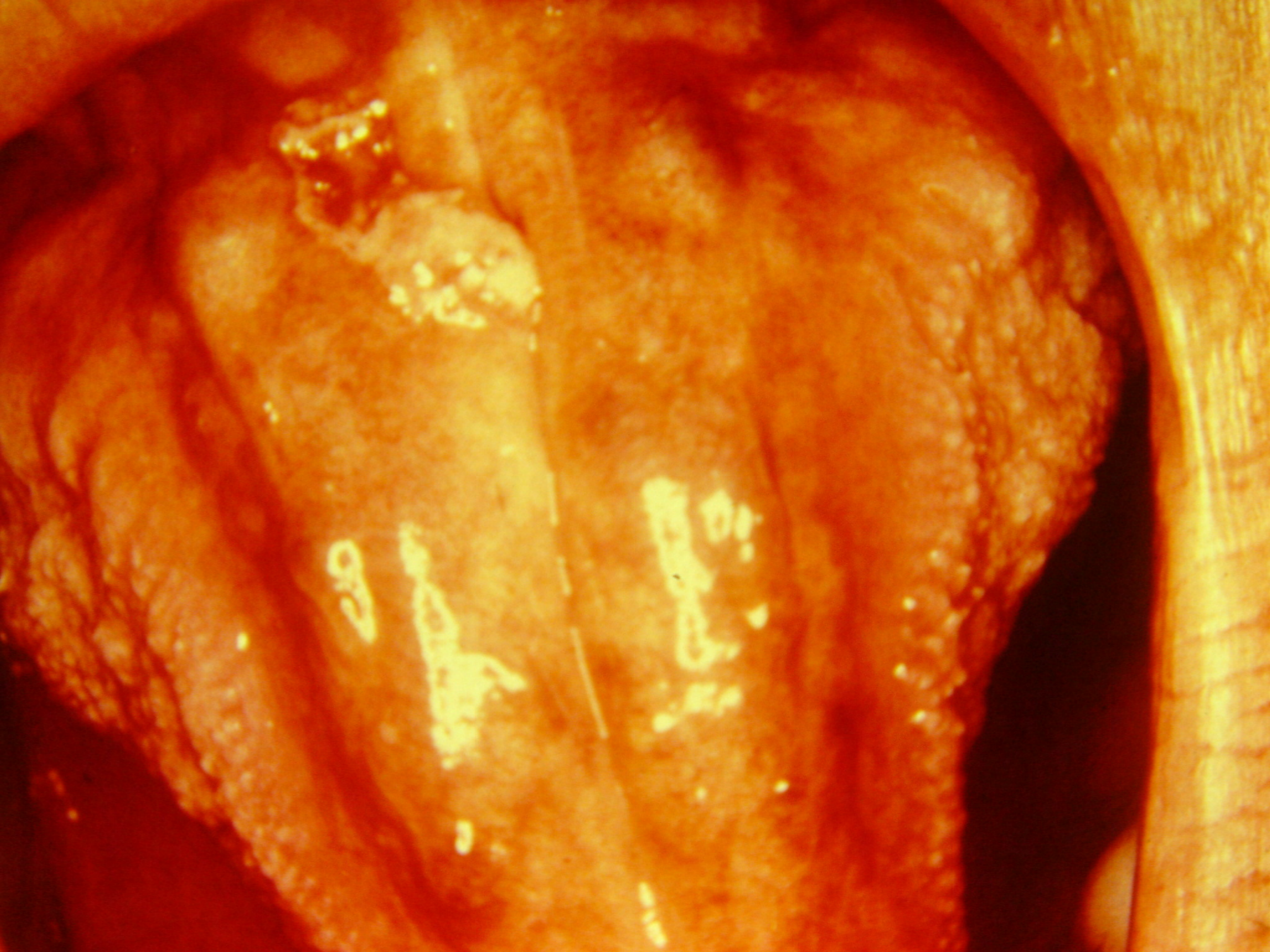
*skin↯,Bowen's disease











Erythroplakia

*Erythroplakia-by Queyrat in 1911

*glands penis of an elderly syphilitic person

*mild dysplasia to cacinoma in situ to invasive ca

*similar to Bowen's disease

*D.D.:candidosis,T.B.,Histoplasmosis, denture irritation

Forms

1. Homogenous(bright red,soft velvety)
2. Interspersed c̄ patches of leukoplakia
3. Elevated red lesions c̄ granular or nodular surface(speckled leukoplakia)

Erythroplakia

*most cases invasive ca(50%) or in situ(40%)
or epi. dysplasia(9%)

*multicentric origin

*very thin conn. tiss. papilla

*Tx:penis-5Fu,same as ca

Criteria for Dx of epithelial dysplasia

1. Increased abnormal mitosis
2. Individual cell keratinization
3. Epithelial pearl c̄ in spinous layer
4. Alterations in N/C ratio
5. Loss of polarity & disorientation of cells

6. Hyperchromatism of cells
7. Large prominent nucleoli
8. Dyskaryosis or nuclear atypism(★)
9. Poikilocarynosis or division of nuclei without division of cytoplasm
10. Basilar hyperplasia







Oral submucous fibrosis

*precancerous condition

*Southeast Asia

*inflam→fibroelastic change→epi atropy

→stiffness→trismus→can't eat

*chillies,Vit B↓,protein↓,betal nut chewing

Clinical

*burning sensation(spicy foods)

*vesicles,ulceration,recurrent stomatitis

*excessive salivation or xerostomia

*similar to systemic sclerosis or scleroderma

*mucosa-blanched opaque,fibrotic bands



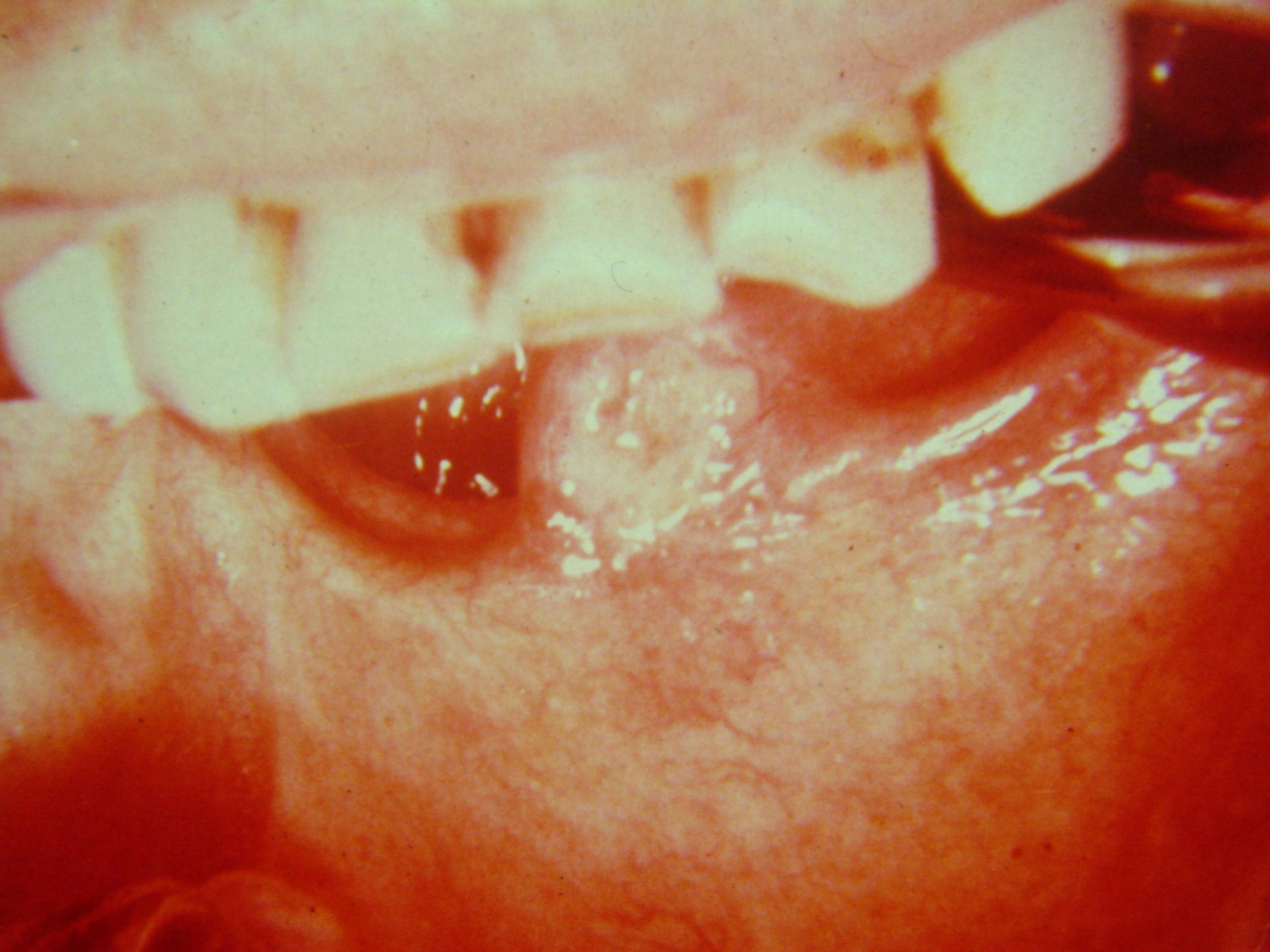












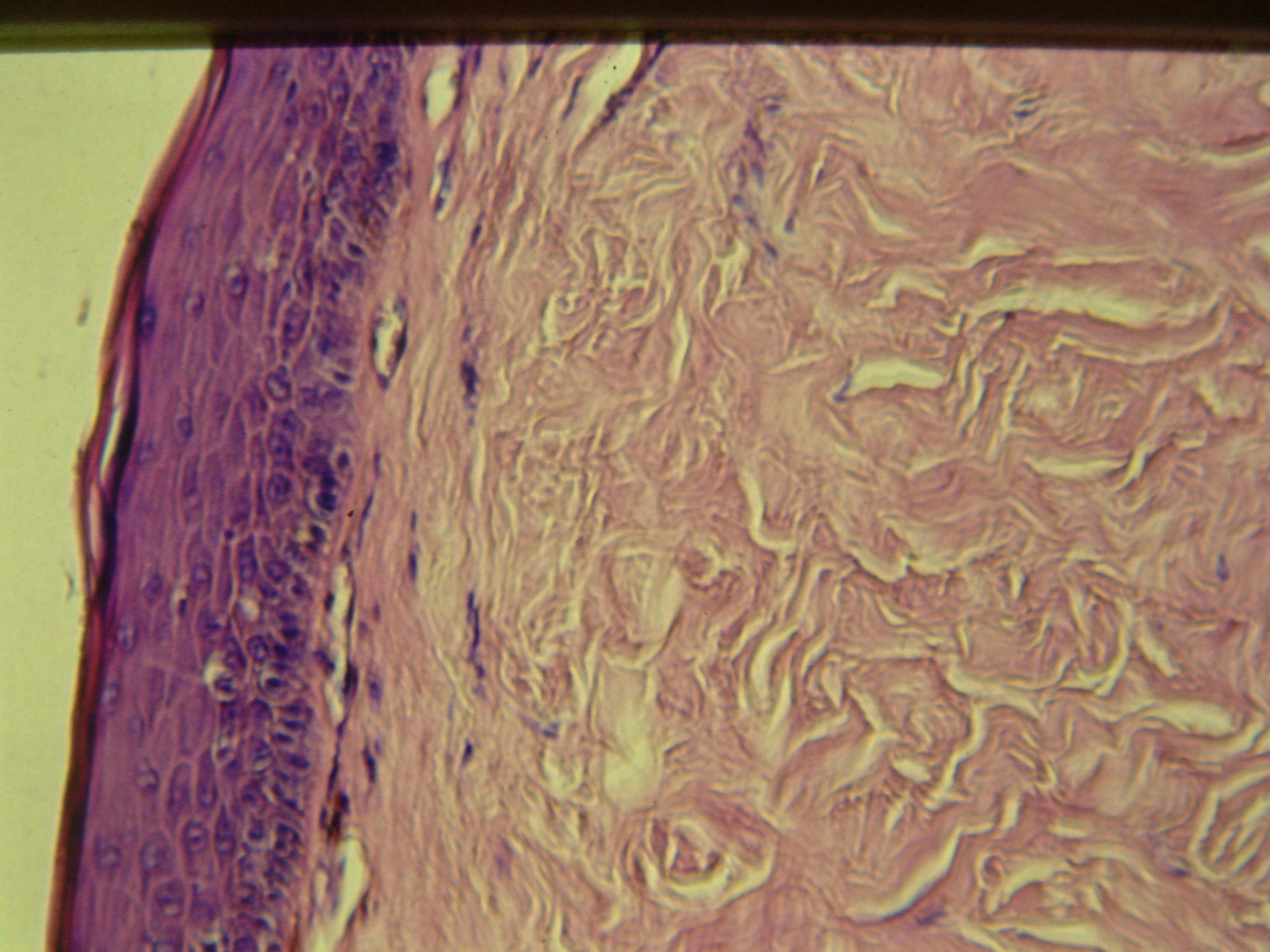
Histology

*atrophic epi

*severe hyalinization c homogenization of collagen bundles, b.v.↓, fibroblast↓

*chr. inflammatory cell

*atypia changes(13-15%)



Treatment

1. systemic or local corticosteroids
2. 40% relate to oral ca
   (surgical tx)





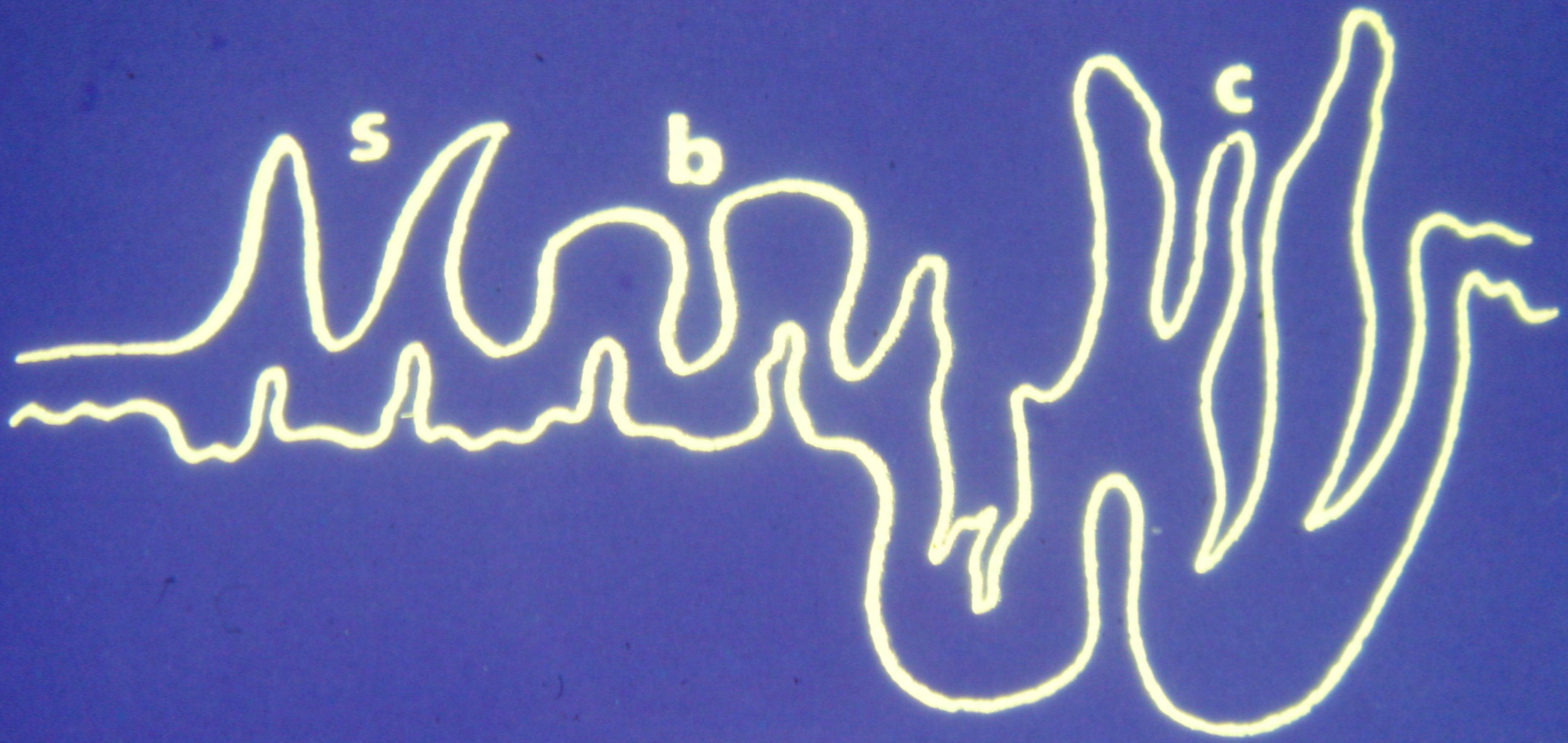


FIG. 13. Diagram illustrating the main distinguishing histologic
feature between verrucous hyperplasia and verrucous carcinoma.
Verrucous hyperplasia. "sharp" variety. b: Verrucous hyperplasi
'blunt' variety. c: Verrucous carcinoma.







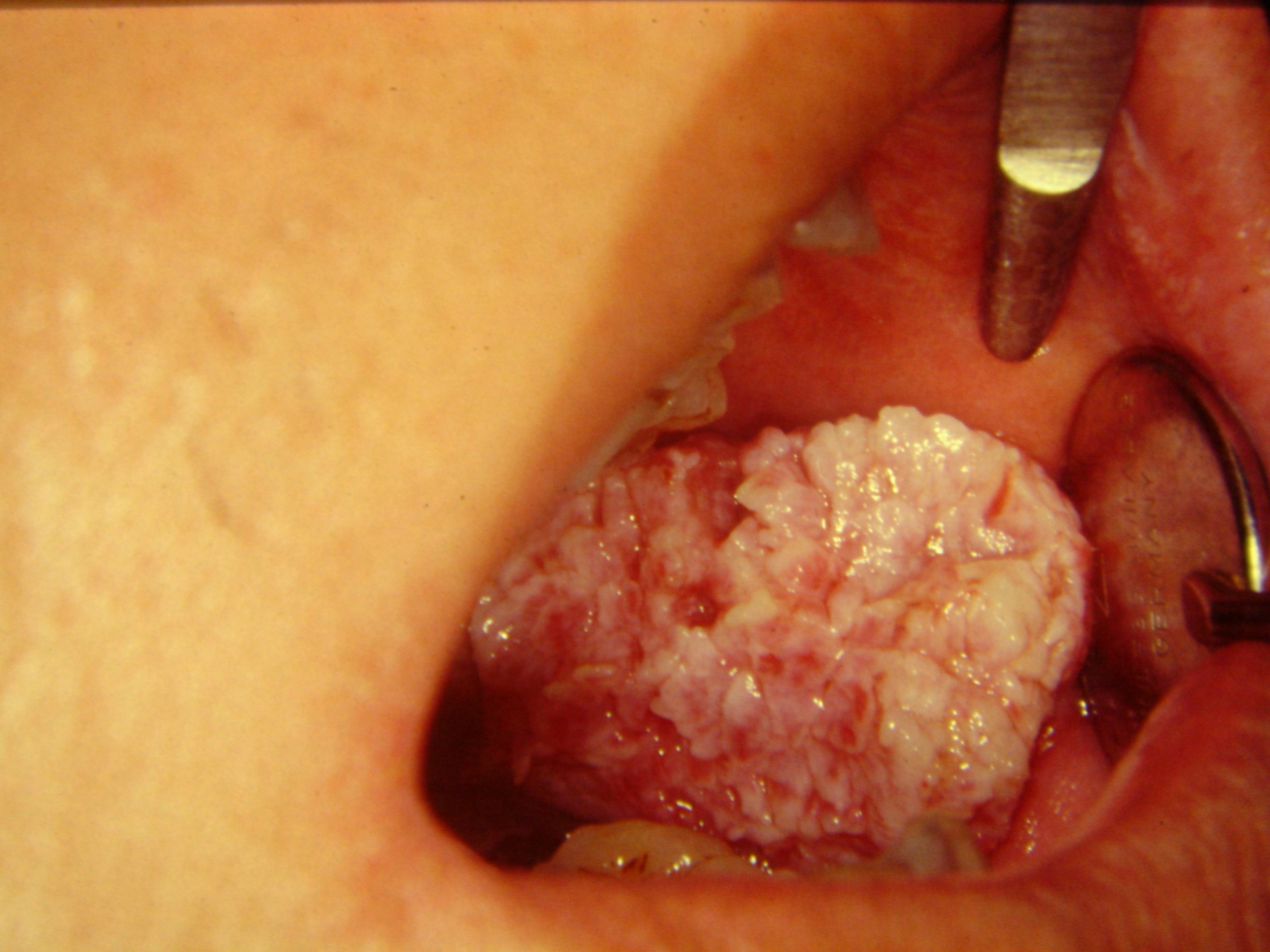


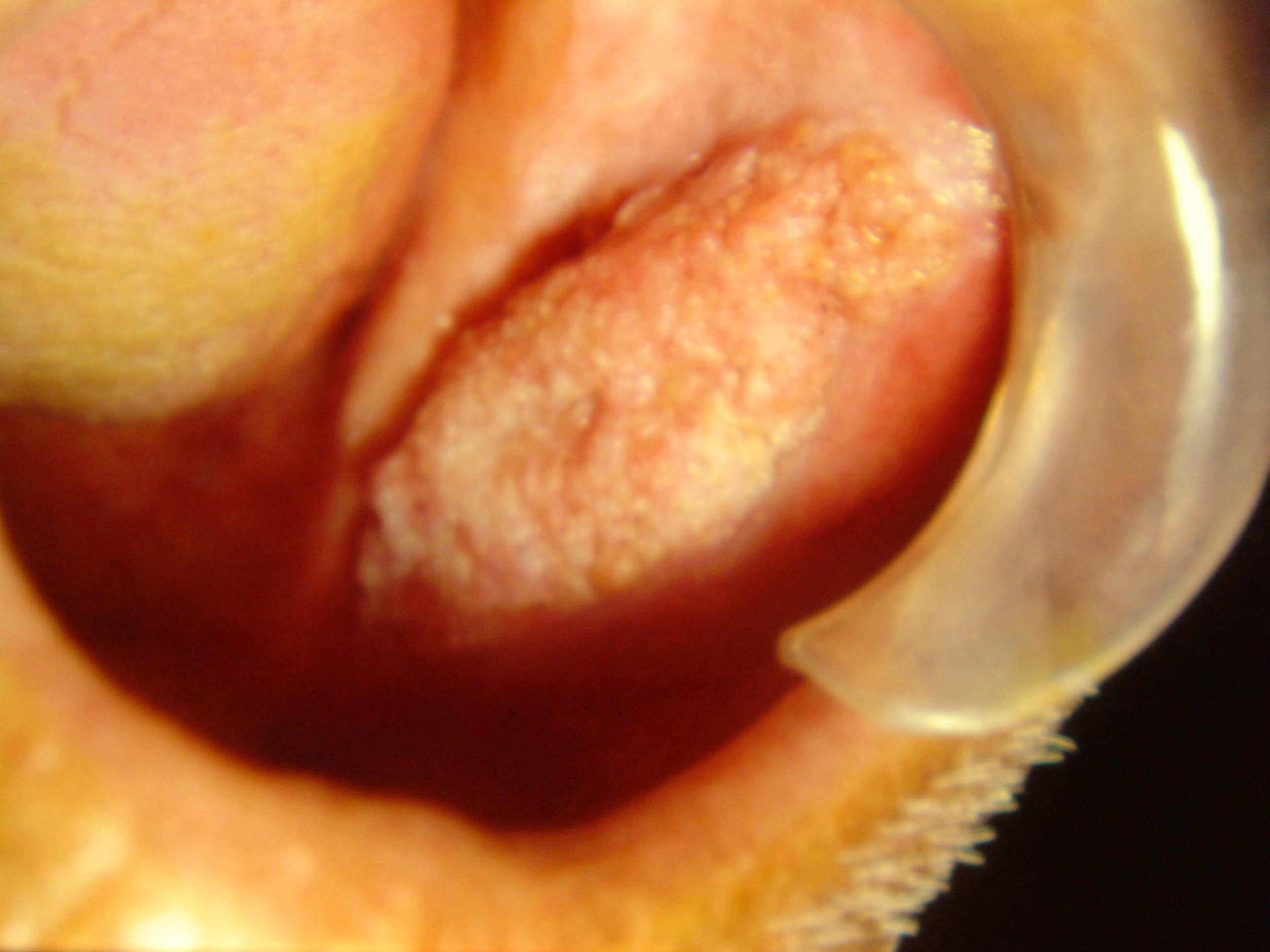




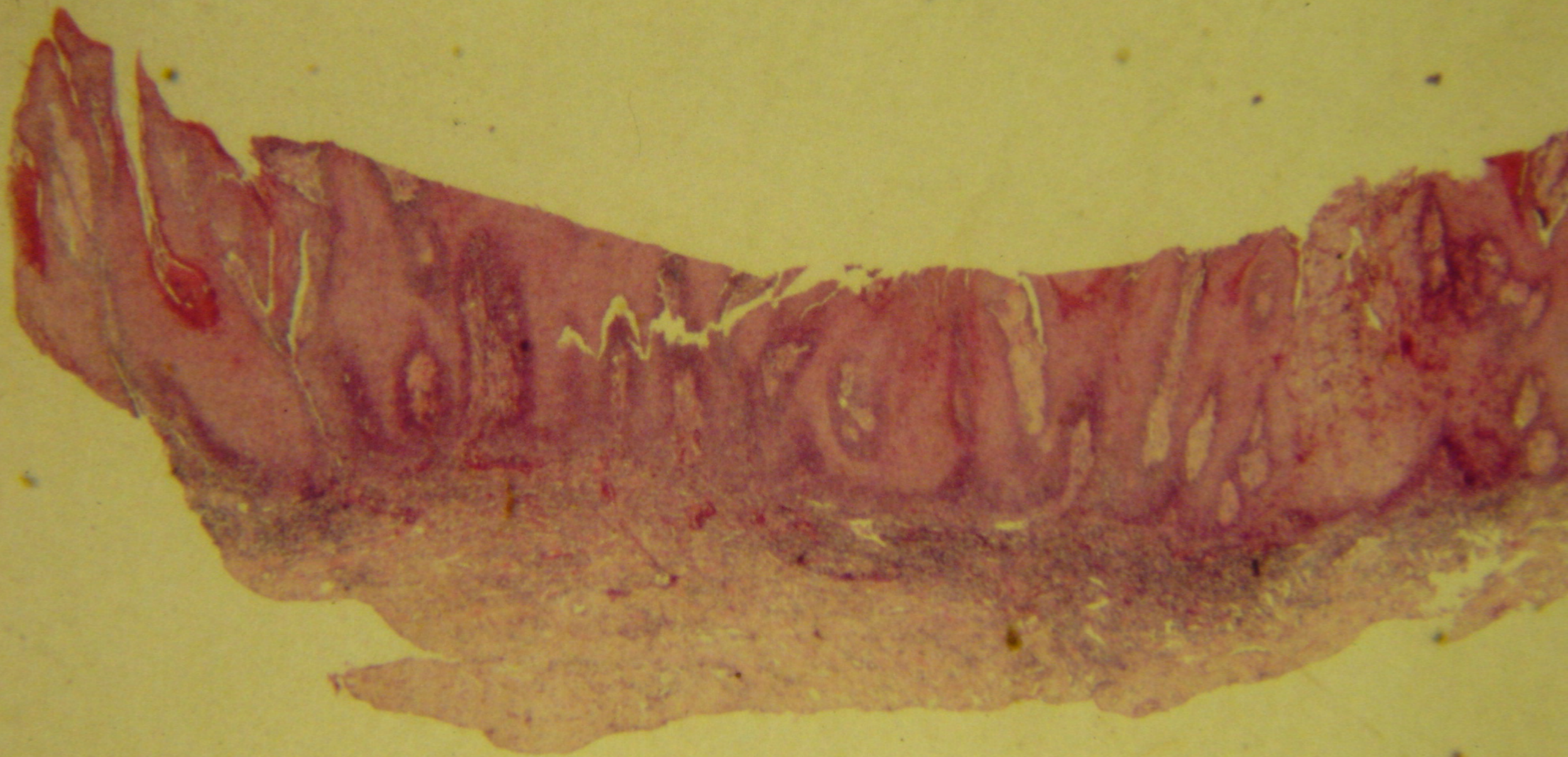








80-834



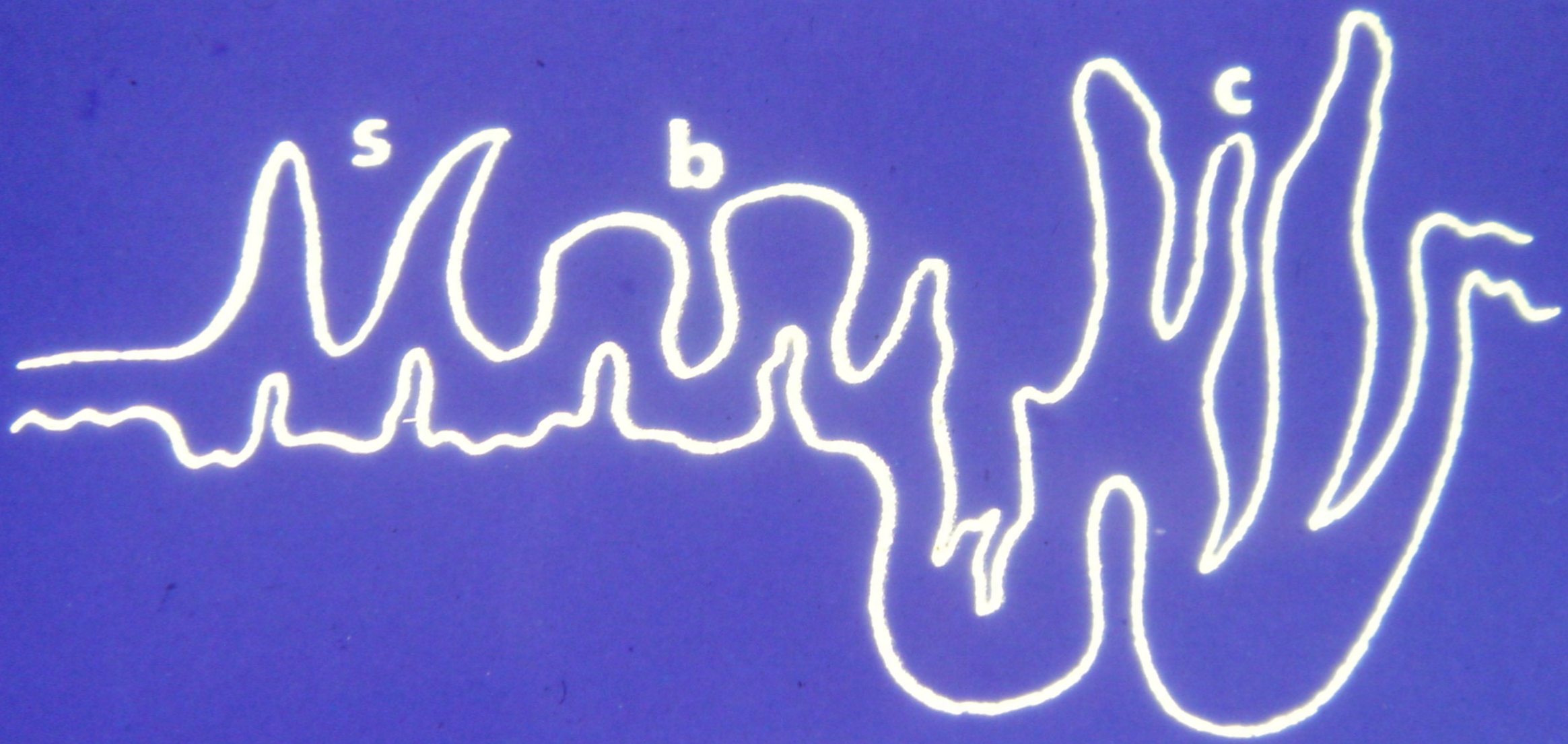


Fig. 13. Diagram illustrating the main distinguishing histological feature between verrucous hyperplasia and verrucous carcinoma. s: Verrucous hyperplasia. "sharp" variety. b: Verrucous hyperplasia. 'blunt' variety. c: Verrucous carcinoma.

Neoplasm

Neoplasia

*Persistent,abnormal autonomous proliferation of cells. It is the result of a permanent cellular defect-independent growth

*Very similar to normal in appearance

*Autonomy of tumors has been overemphasized

*Spontaneous regression occur(very few)

MALIGNANT TUMORS OF EPITHELIAL TISSUE ORIGI

*Basal cell carcinoma

*Epidermoid carcinoma

(Squamous cell carcinoma)

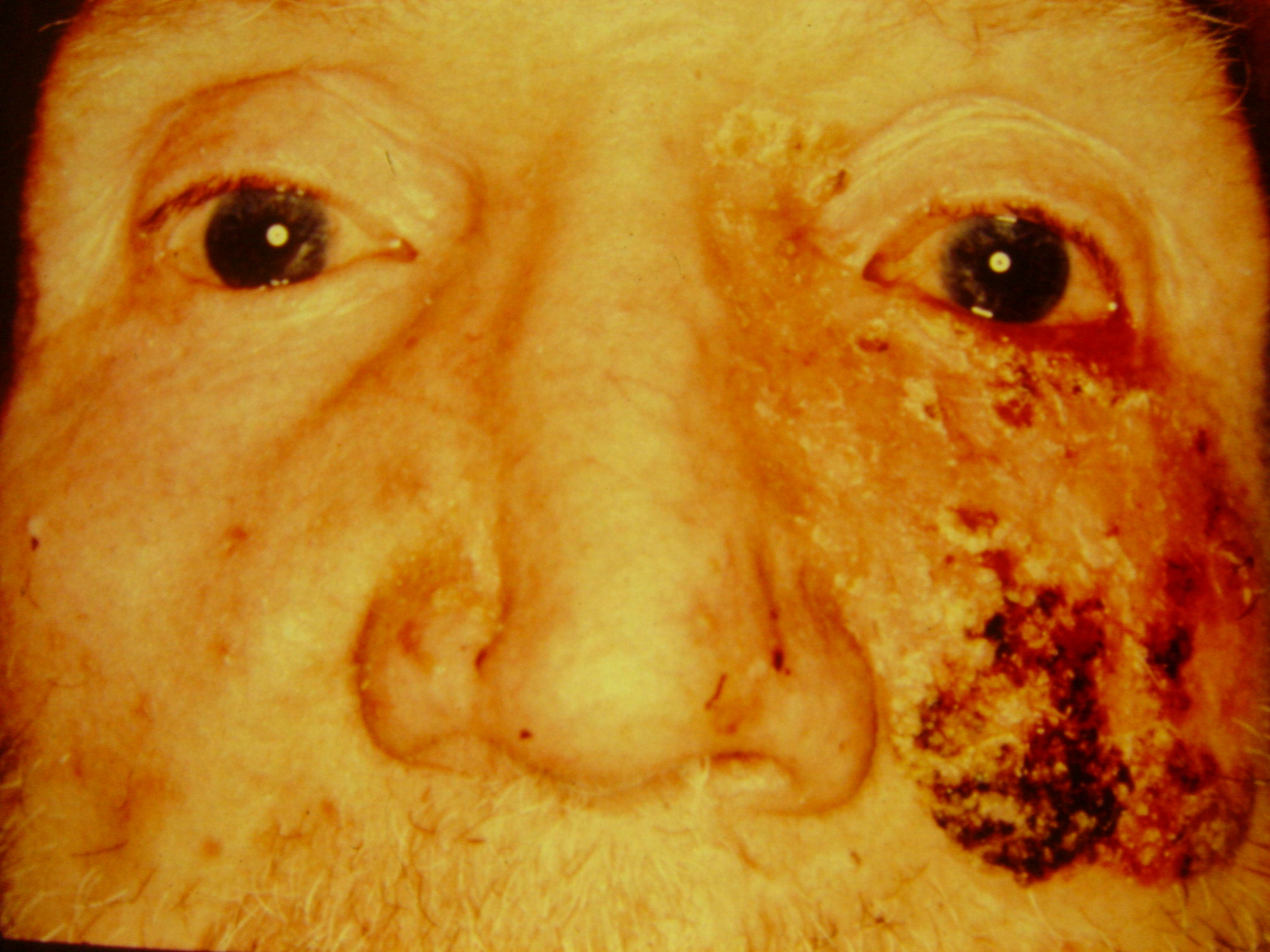
*Verrucous carcinoma

*Spindle cell carcinoma

Basal cell carcinoma

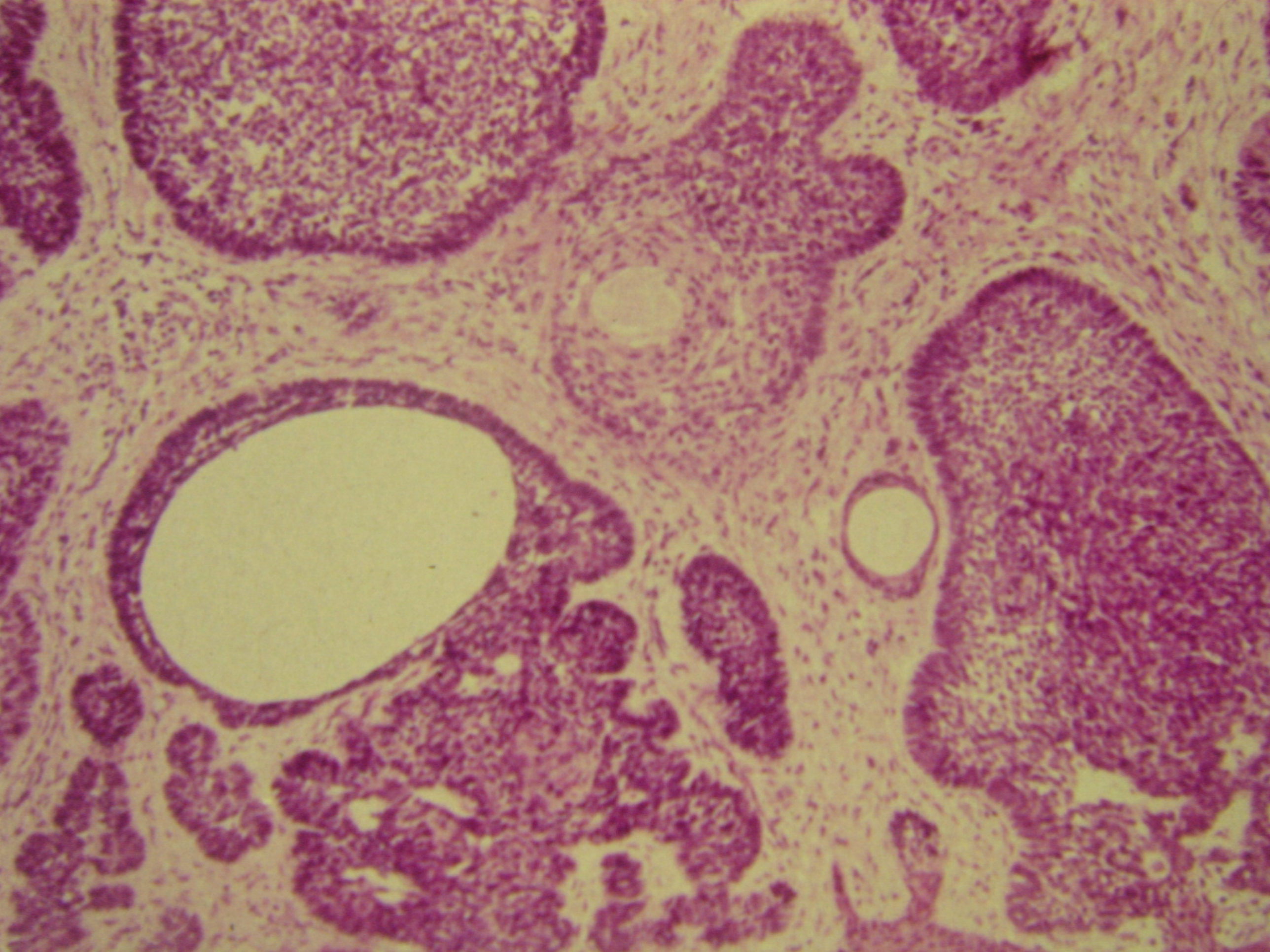
* most common type of ca. in men.
* primary tumor site- head & neck 85%
* metastasis --- 0.1% (cotran)
* Etiology ---ultraviolet radiation.
* Never seen in the oral cavity.
* adenoid cystic, keratotic. B.C.C.
* "Jaw cyst- bifid rib-basal cell nevus" syn



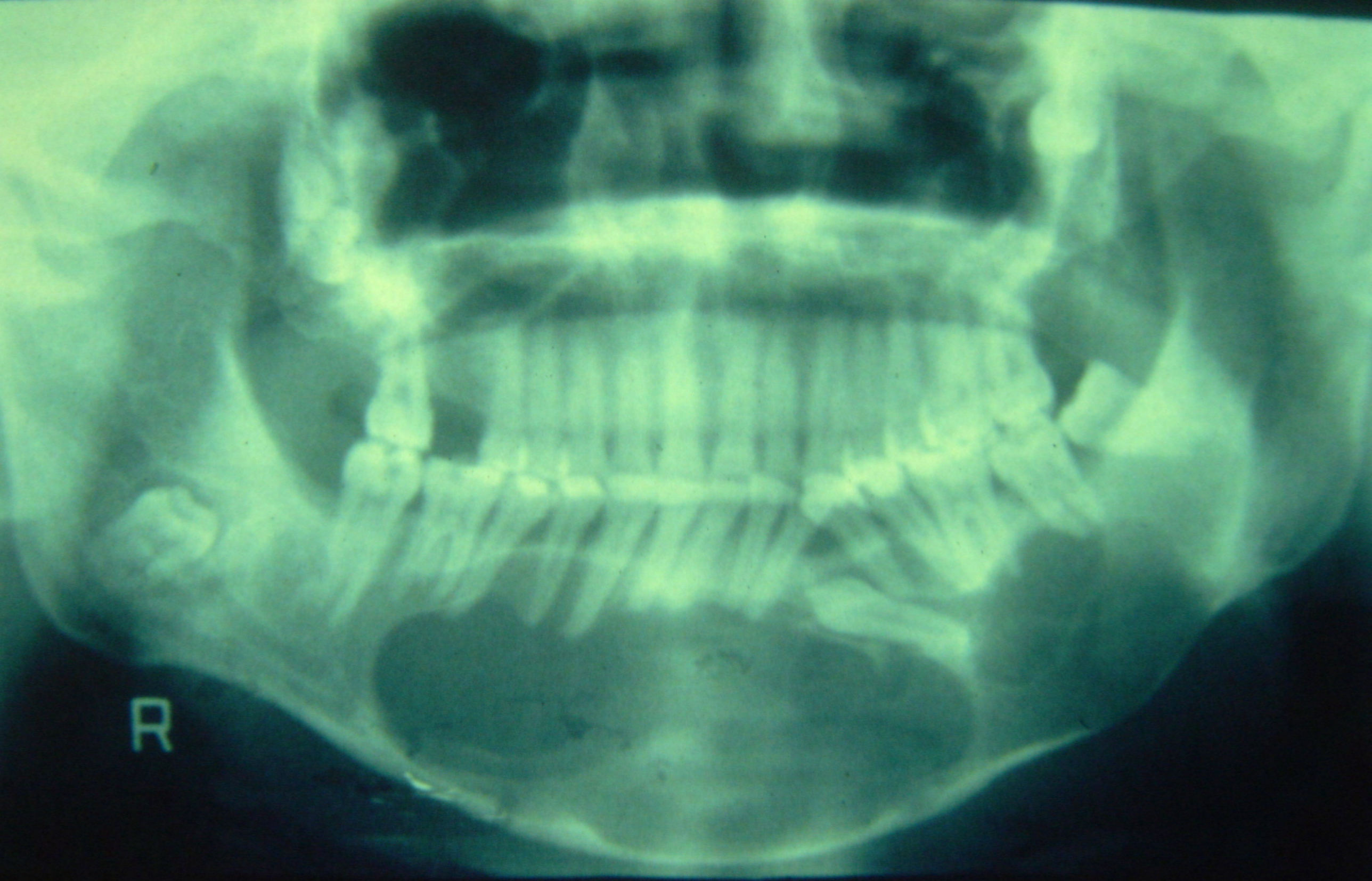


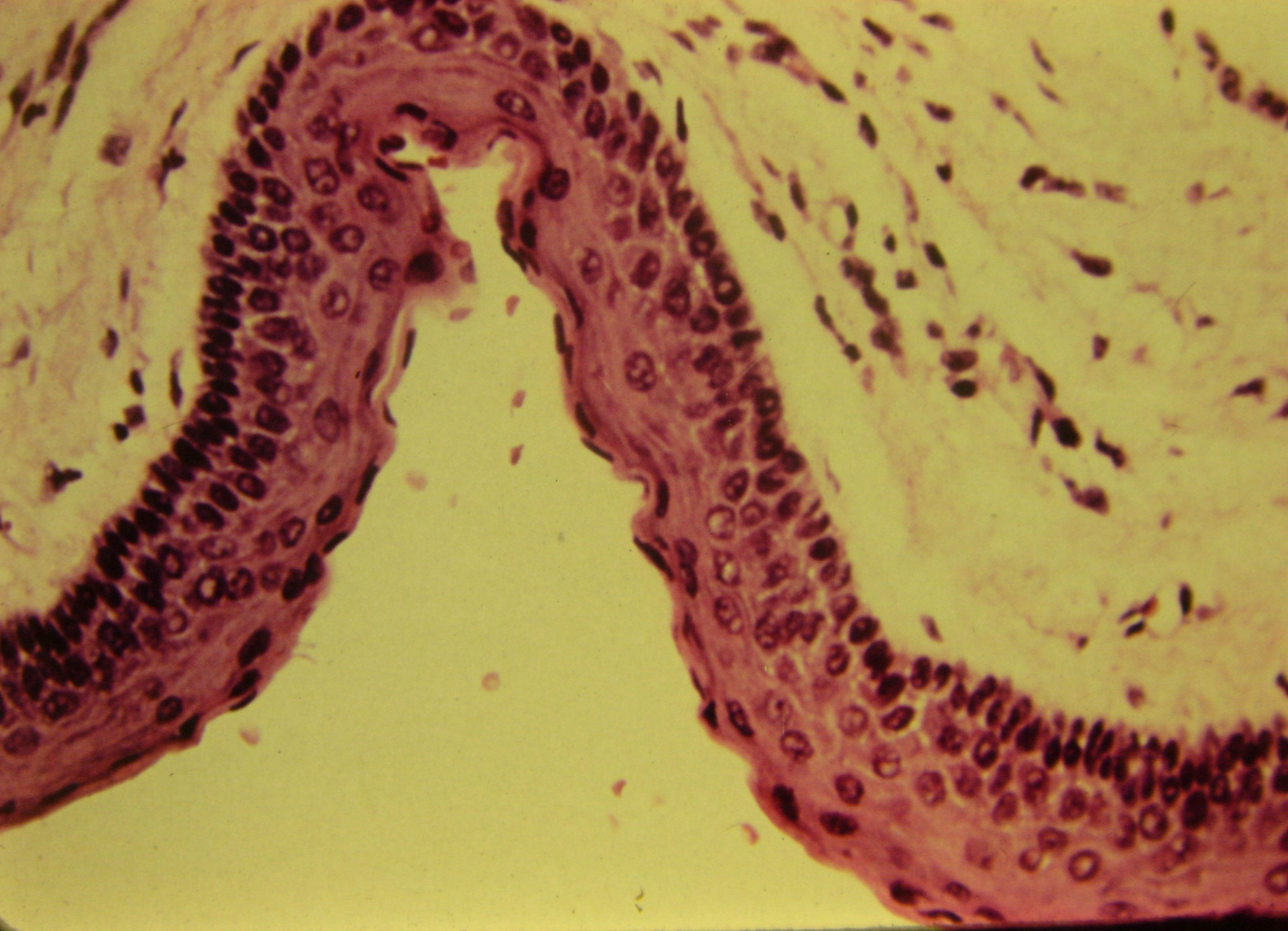


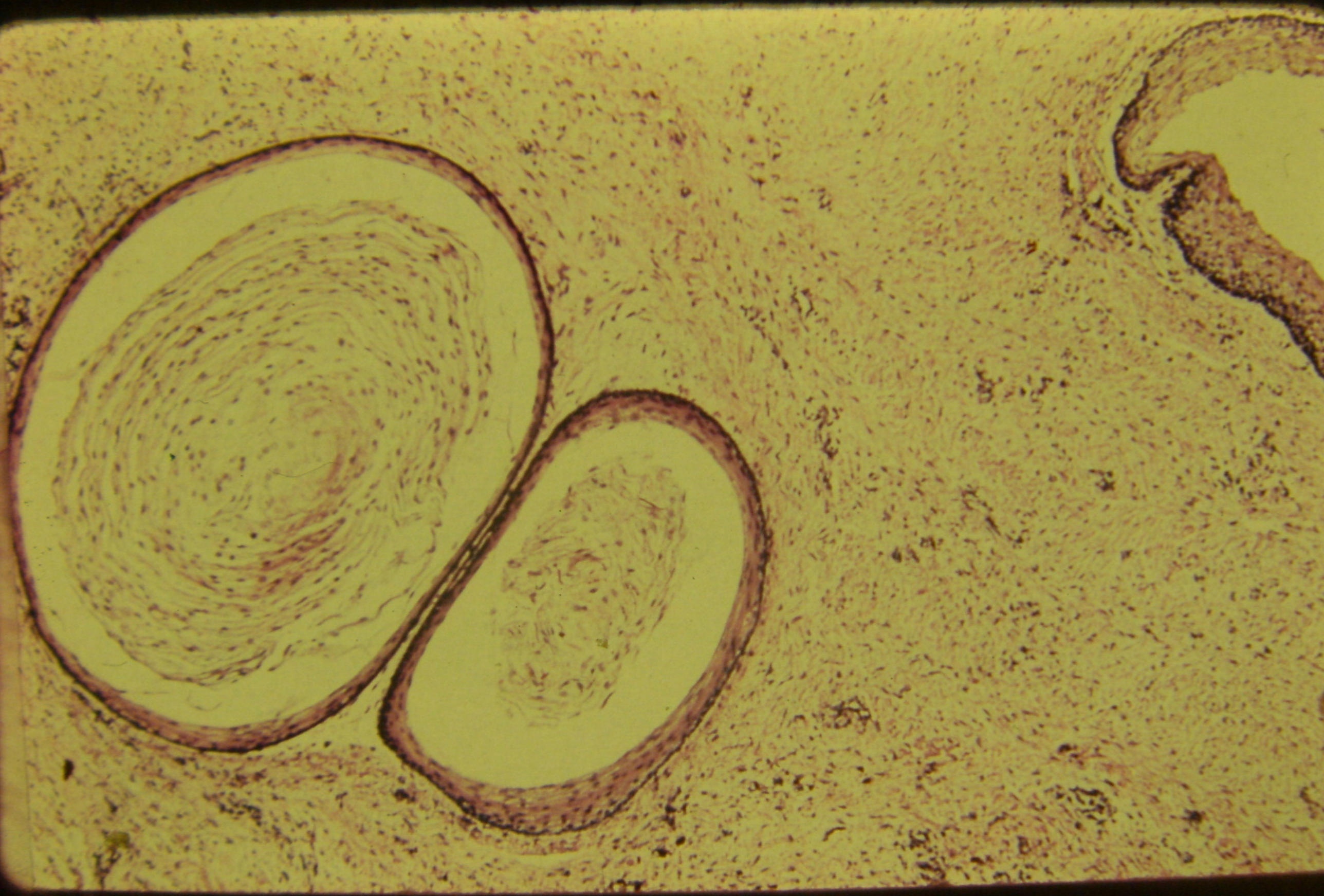




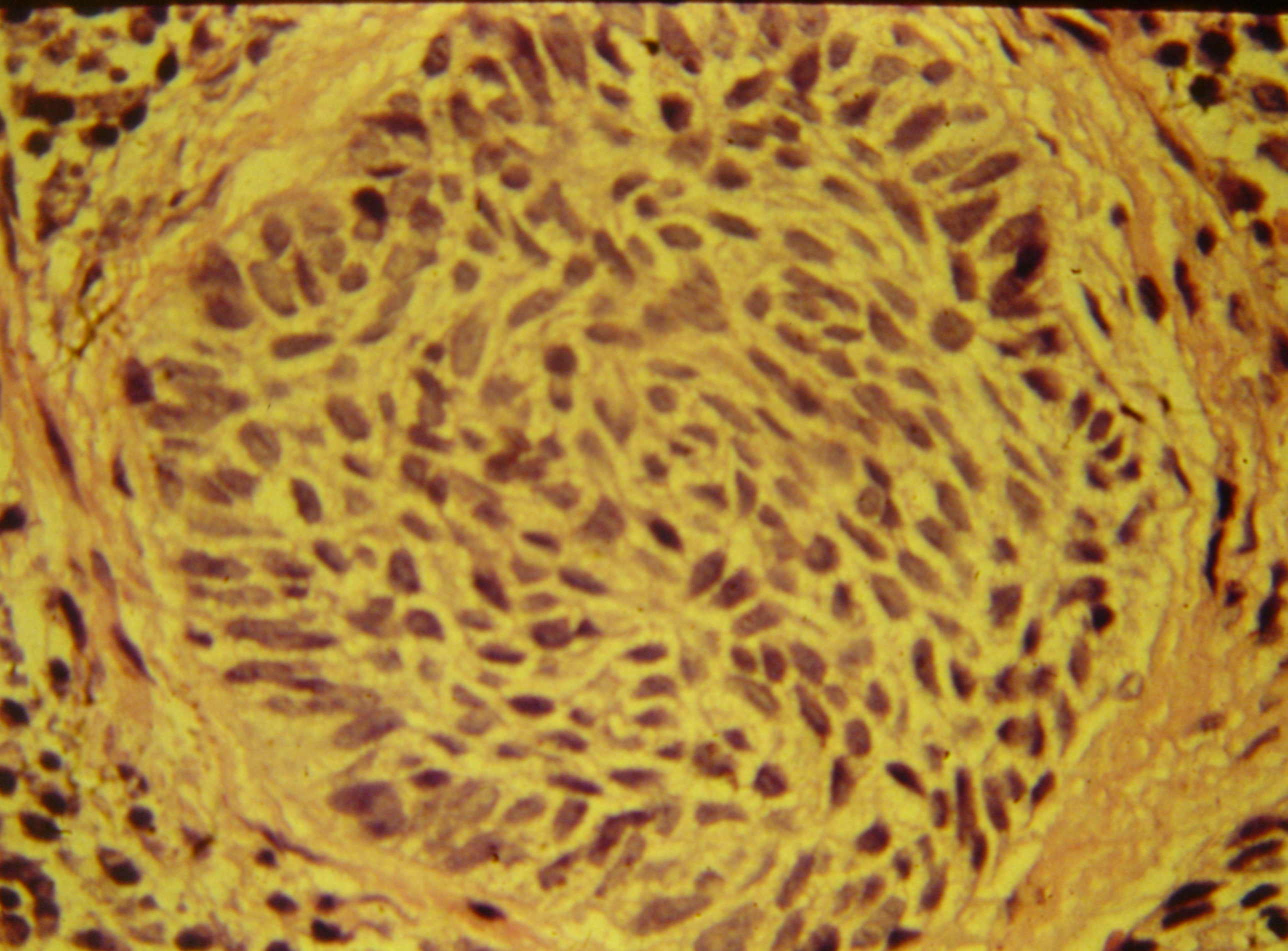


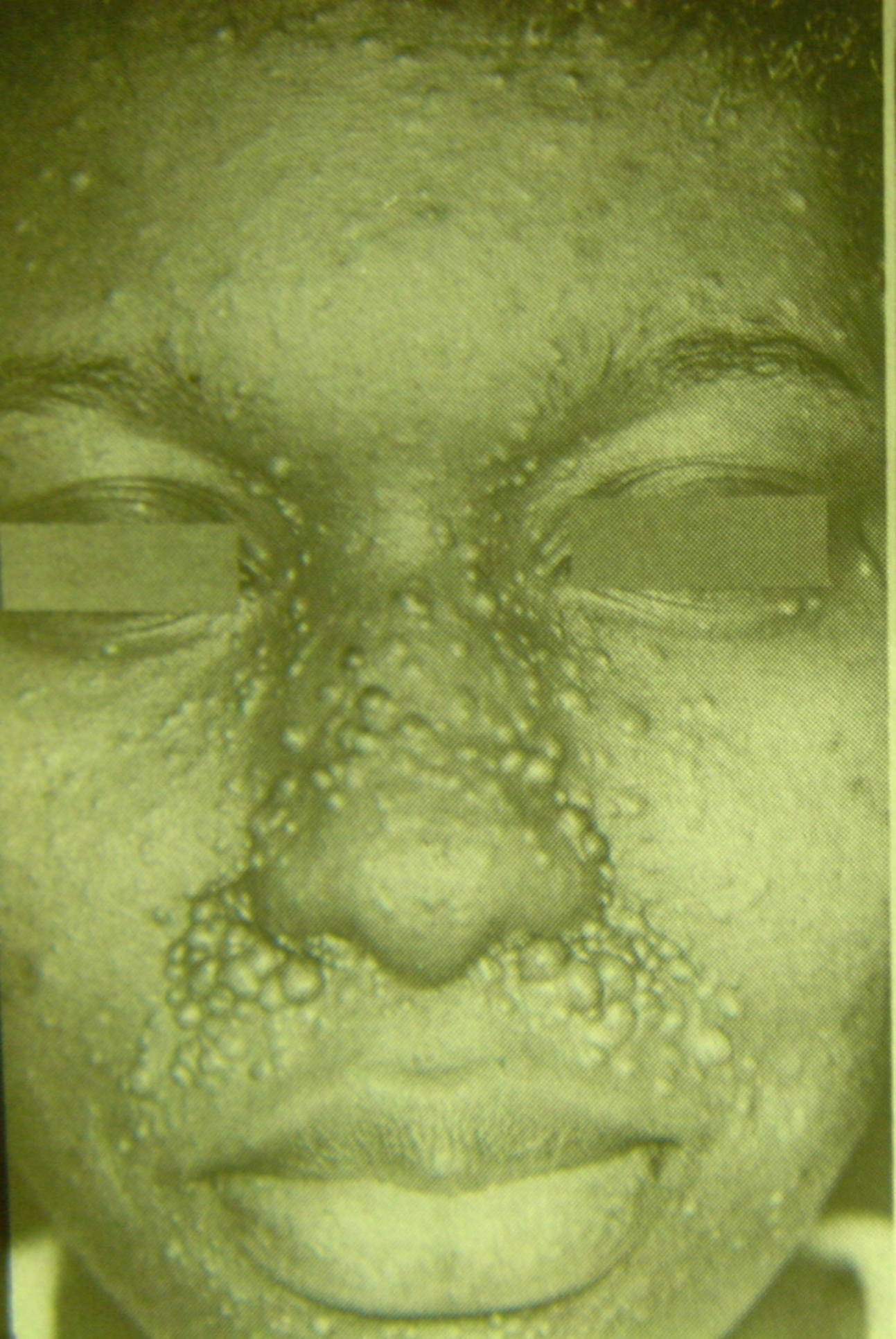

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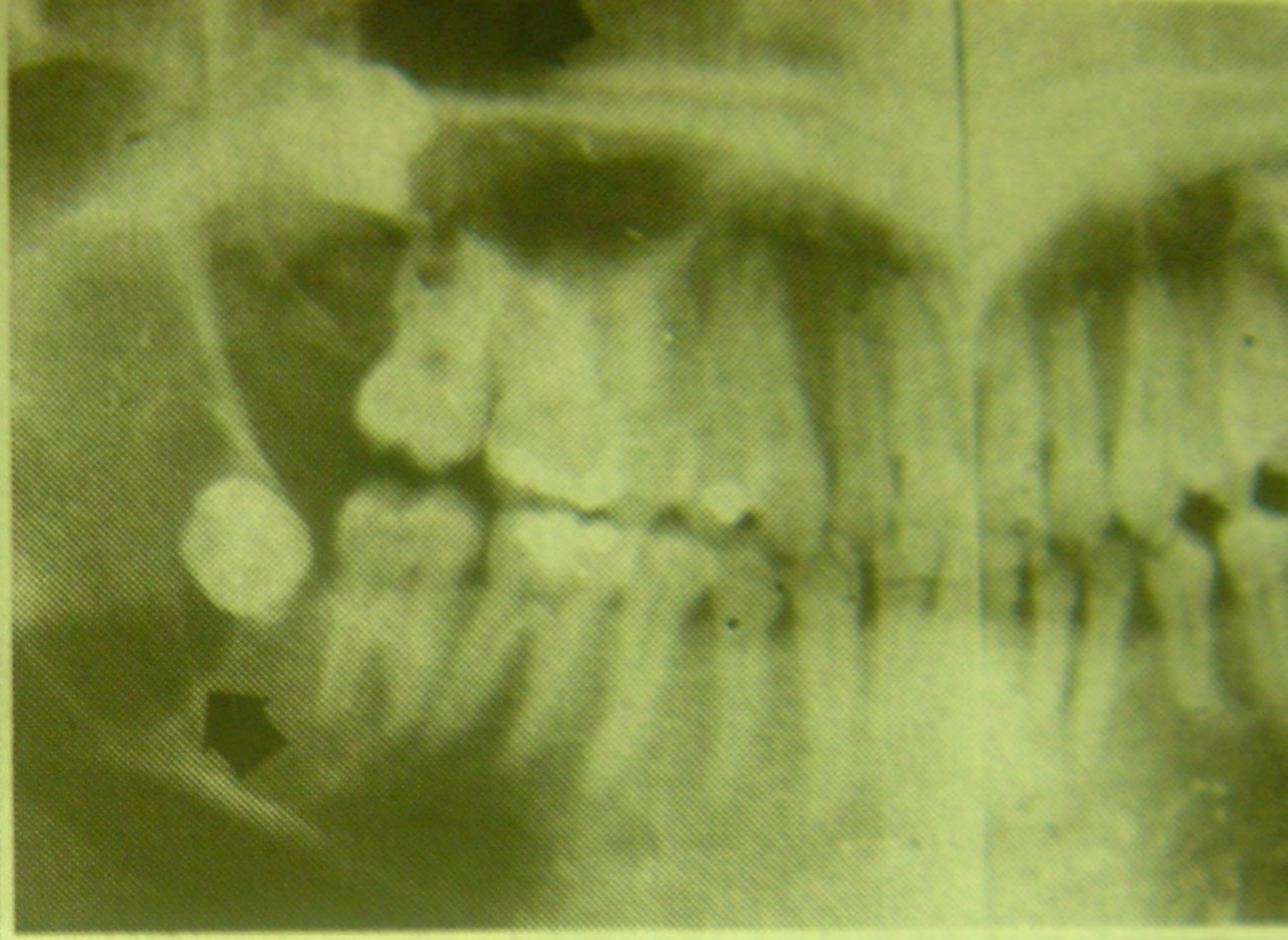


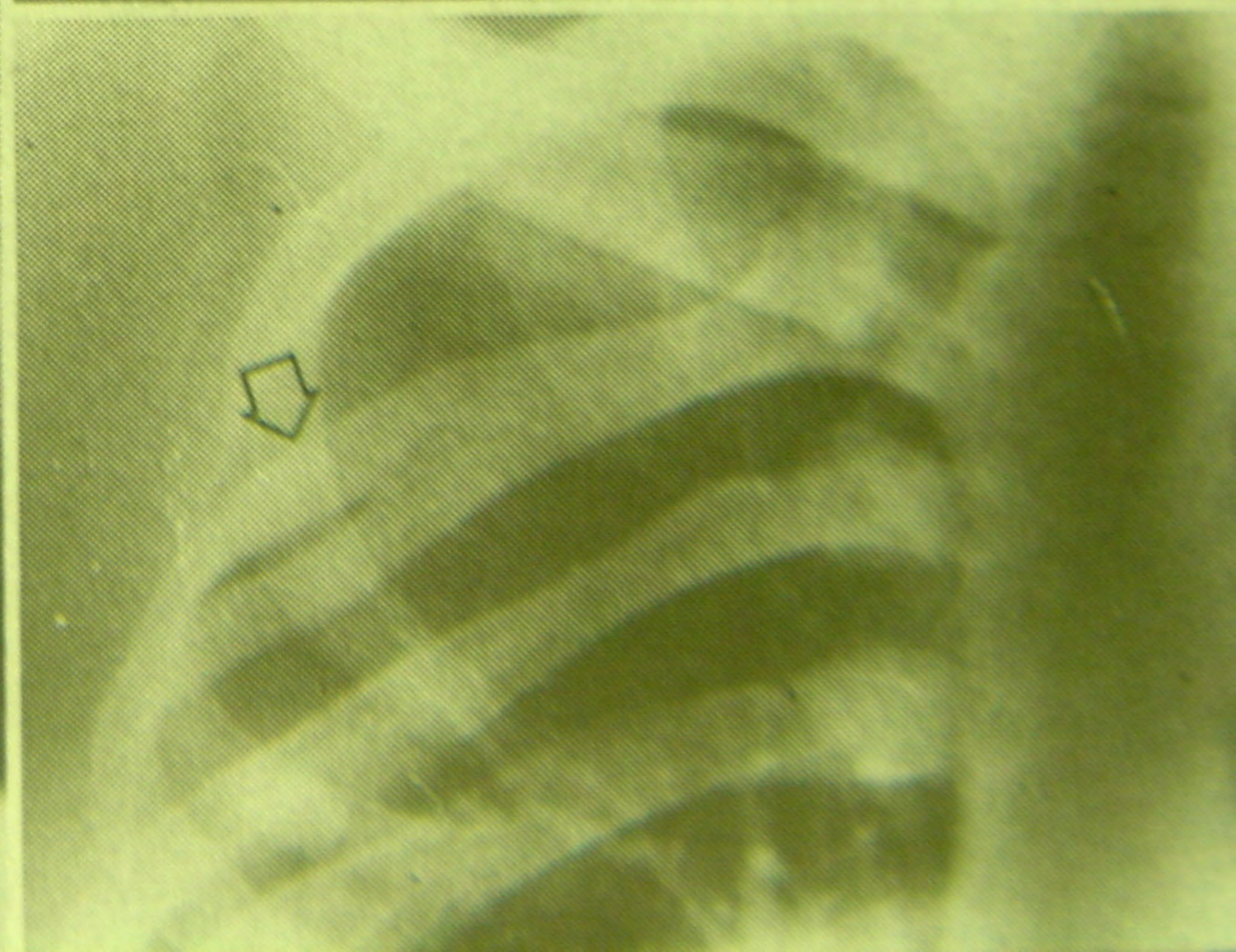


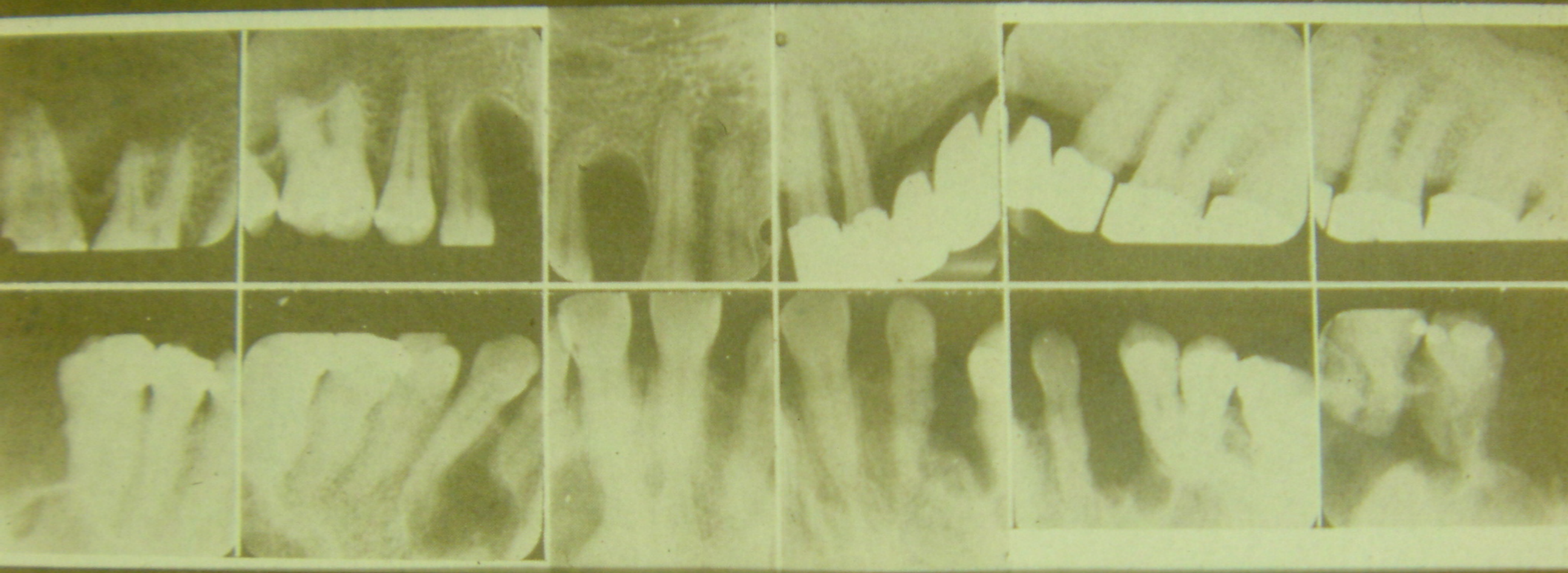












Neoplasia

*Persistent,abnormal autonomous proliferation of cells. It is the result of a permanent cellular defect-independent growth

*Very similar to normal in appearance

*Autonomy of tumors has been overemphasized

*Spontaneous regression occur(very few)

Top ten cancers in Taiwan

1. Liver & gall bladder
2. Rectum and colon
3. Cervix
4. Lung
5. Breast
6. Stomach
7. Oral cavity
8. Prostate
9. Skin
10. Urinary baldder

FUNDAMENTAL UNDERSTAINDING

How Cancer Arises

An explosion of researh is uncovering the long-hidden molecular underpinnings of cancer-and suggesting new therapies

By Robert A. Weinberg

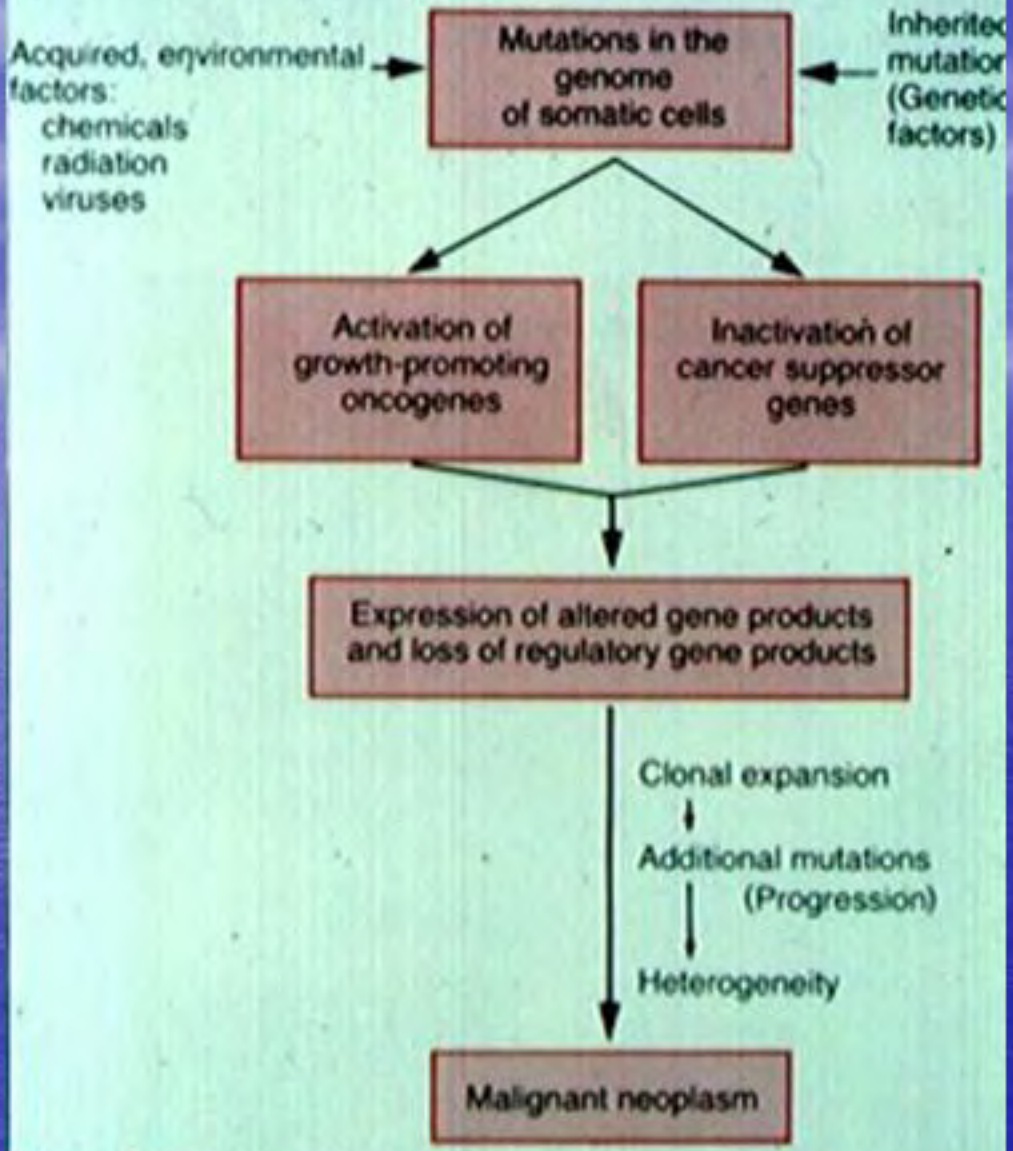
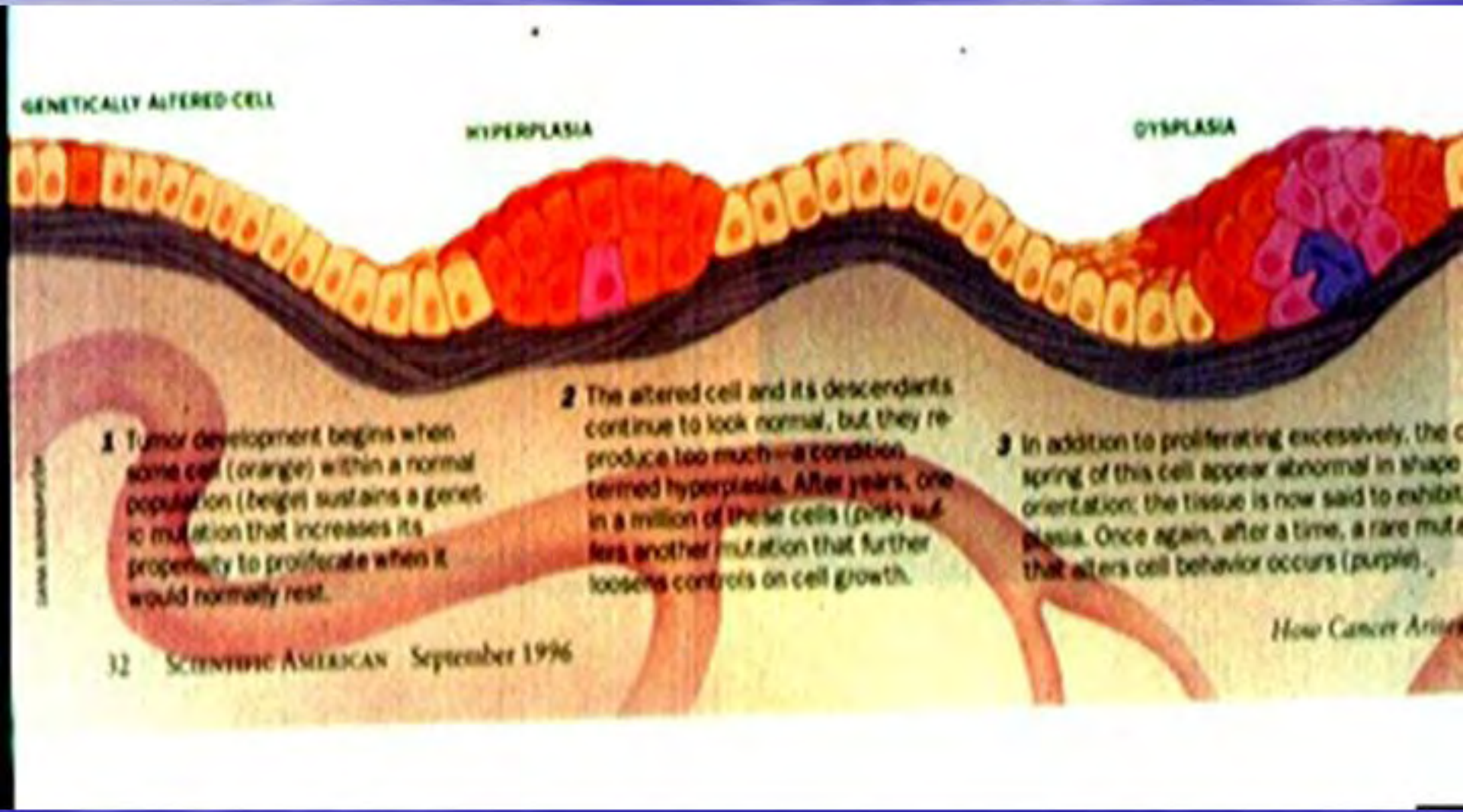


Figure 7-16. Flow chart depicts a simplified scheme of cancer pathogenesis.

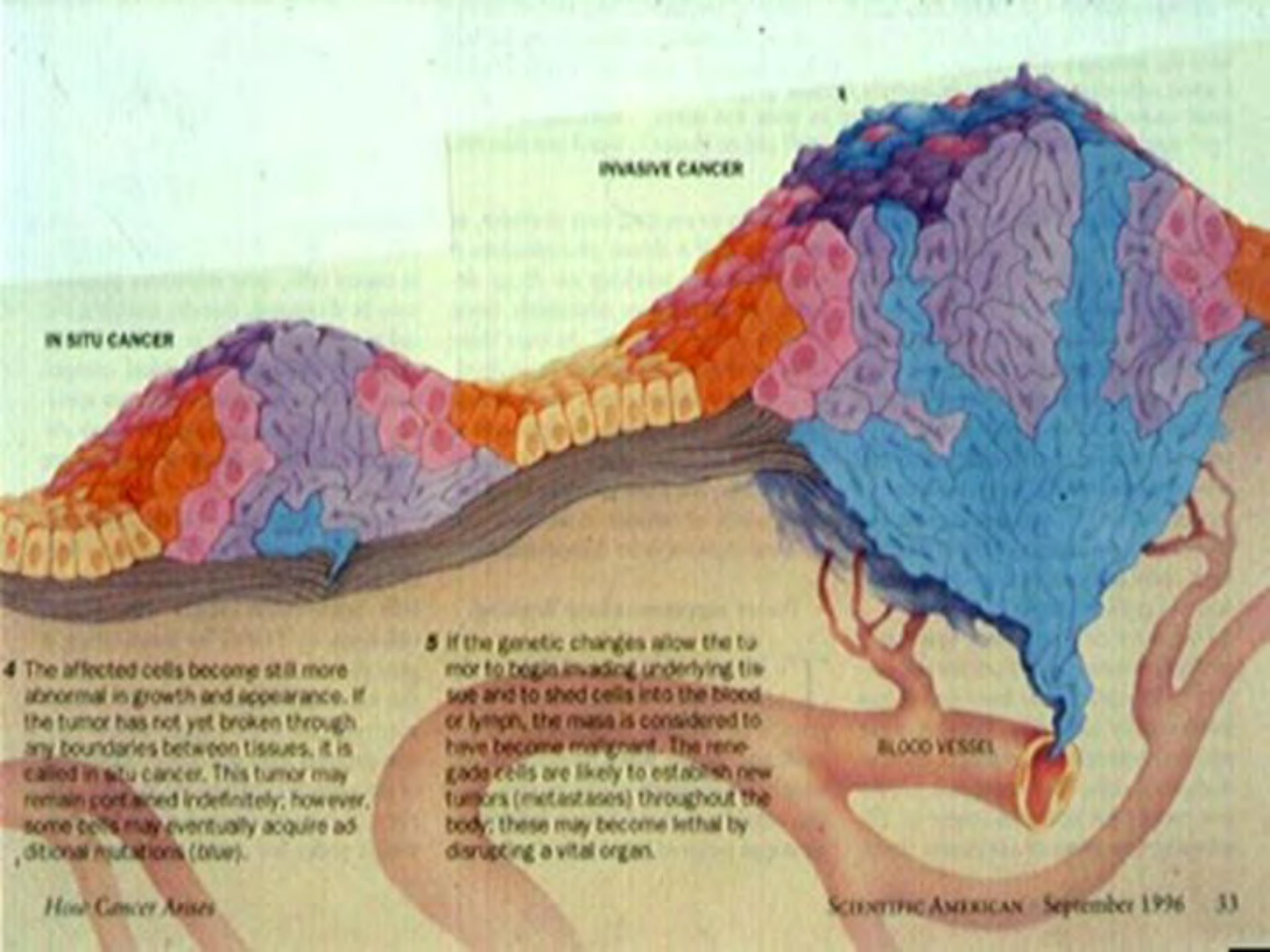
TABLE 4. *Critical genetic alterations in multistage carcinogenesis*

Activation of cellular protooncogenes; i.e., ras, myc, erb B-2, bcl-2
Inactivation of tumor suppressor genes; i.e., Rb, p53
Inactivation of putative metastasis suppressor genes; i.e., nm23
Increasing genomic instability; i.e., aneuploidy, DNA hypomethylation, reactive oxygen species mutagenesis


INITIATION
PROMOTION
STAGES 1,2....
PROGRESSION
INCREASING MALIGNANCY



How Cancer Arises



4 The affected cells become still more abnormal in growth and appearance. If the tumor has not yet broken through any boundaries between tissues, it is called in situ cancer. This tumor may remain contained indefinitely; however, some cells may eventually acquire additional mutations (blue).

5 If the genetic changes allow the tumor to begin invading underlying tissue and to shed cells into the blood or lymph, the mass is considered to have become malignant. The renegade cells are likely to establish new tumors (metastases) throughout the body; these may become lethal by disrupting a vital organ.

天下文化

基因聖戰

擺脫遺傳的宿命

GENOME

The Story of the Most Astonishing Scientific Adventure of Our Time
—The Attempt to Map All the Genes in the Human Body



by Jerry E. Bishop & Michael Waldholz



一連串的基因變異, 一步步導致癌症發生

Etiology factors

- Tobacco
- Alcohol
- Syphilis
- Sunlight
- Nutritional def.
- Heat, trauma, sepsis
- Virus

Etiology

*tobacco, alcohol, oral sepsis, irritation
syphilis, vit. def., endocrine dis. gal-
vanism, actinic radiation

Virus

Epstein Barr (EBV) - infectious monocleosis

cytomegalovinus (CMV) - cytomegalic inclusion dis

Herpes simplex (HSV) - Herpes

Varicella-zoster (VZV) - Chicken pox-herpes zoster

Table 15-1. Some characteristics of typical benign and malignant tumors*

	Benign	Malignant
Growth rate	Slow	Rapid
Mitoses	Few	Many
Nuclear chromatin	Normal	Increased
Differentiation	Good	Poor
Local growth	Expansive	Invasive
Encapsulation	Present	Absent
Destruction of tissue	Little	Much
Vessel invasion	None	Frequent
Metastases	None	Frequent
Effect on host	Often insignificant	Significant

*Many tumors show exception to one or more of these characteristics.

Table 2–6. Definition of the TNM Categories of Malignant Tumors about the Oral Cavity

T—Primary Tumor
- TIS–Carcinoma in situ.
- T1—Tumor 2 cm. or less in greatest diameter.
- T2—Tumor greater than 2 cm. but not greater than 4 cm. in greatest diameter.
- T3—Tumor greater than 4 cm. in greatest diameter.

N–Regional Lymph Nodes
- N0—No clinically palpable cervical lymph node(s); or palpable node(s) but metastasis not suspected.
- N1—Clinically palpable homolateral cervical lymph node(s) that are not fixed; metastasis suspected.
- N2—Clinically palpable contralateral or bilateral cervical lymph node(s) that are not fixed; metastasis suspected.
- N3—Clinically palpable lymph node(s) that are fixed; metastasis suspected.

M–Distant Metastasis
- M0—No distant metastasis
- M1—Clinical and/or radiographic evidence of metastasis other than to cervical lymph nodes.

TNM system

T—size

X -- can't be assessed

T1-- ≤ 2 cm

T2-- 2-4 cm

T3-- ≥ 4 cm

or invades adjacent structure , cortical bone, deep m., tongue,maxillary sinus, skin

N- N1—single ,同側 < 3cm

N2—(1) single > 3cm , < 6 cm

(2) 同側, multiple , < 6 cm

N2 兩側或對側 < 6 cm

a- single ≥ 3cm , ≤ 6cm

b- multiple < 6cm

c- bilateral or contralateral

N3 meta LN > 6cm

M1- Distant metastasis

Table 2–7. Clinical Stage-Grouping of Carcinoma of the Oral Cavity

Stage I:	T1 N0 M0	
Stage II:	T2 N0 M0	
Stage III:	T3 N0 M0	
	T1 N1 M0	
	T2 N1 M0	
	T3 N1 M0	
Stage IV:	T1 N2 M0	T1 N3 M0
	T2 N2 M0	T2 N3 M0
	T3 N2 M0	T3 N3 M0
	Or any T or N category with M1.	

Verrucous carcinoma

* Ackerman 1948
* larynx, esophagus, nasal fossa, sinuses
  skin, scrotum, vagina, cervix.
* slow-growing, chiefly-exophytic
* superficial invasive, low meta..
* majority - buccal mucosa, gingiva, ridge
* rugae-like folds with deep clefts.

Oral florid papillomatosis

represent one & the same disease (V.C)
tobacco chewer, snuff, betel nut.
The epi. well diff. little mito.
cleft space, parakeratin plugging
basement membrane will app. intact.
Tx- surgery. X-ray radiation.

Table 2. Clinicopathologic characteristics of verrucous carcinoma.

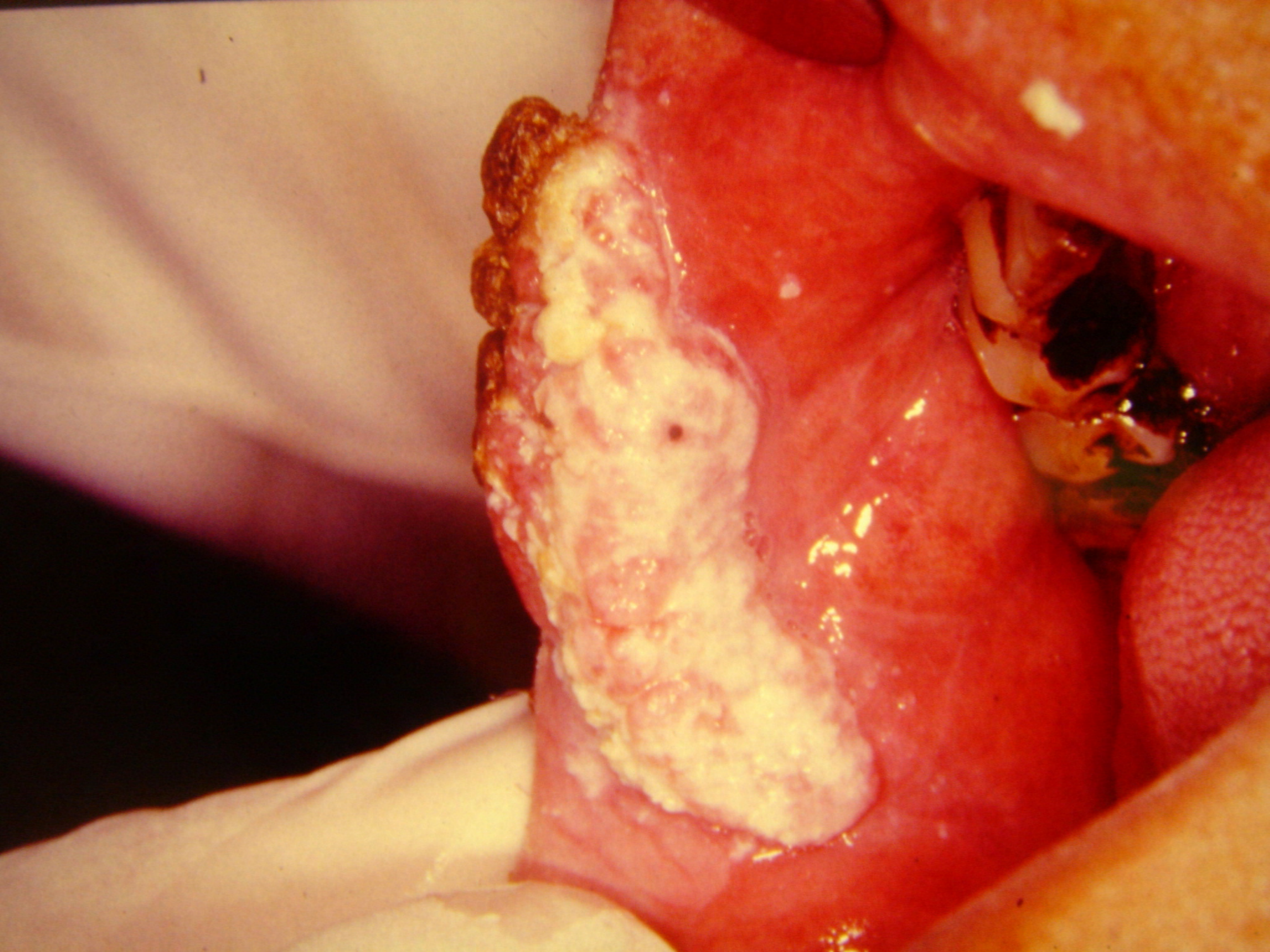
Sites of predilection	Oral cavity, larynx
Age/sex	Men over 50 years of age
Habits	Tobacco users, poor hygiene
Grade of malignancy	Low-grade, of local significance only
Metastatic ability	None in bona fide cases
Gross appearance	Exophytic or fungating; usually keratinizing
Associated mucosal changes	Leukoplakia, metachronous or synchronous squamous cell neoplasms
Differentiation of cells	High degree, uniform. Cleft-like spaces or cysts with keratin
Cytologic features of malignancy	Rare to absent
Depth of lesion	Pushing or blunt invasion
Cellular (host) response	Usually prominent

TABLE 2. Comparison of Pathologic and Clinical Findings Between Squamous Carcinoma and Verrucous Squamous Carcinoma

Features	Squamous cell carcinoma	Verrucous Squamous cell carcinoma
Macroscopic aspect	Various appearances	Fungating, cauliflower-like tumor
Cellular differentiation	Various degrees	High
Cytologic features of malignancy	Present	Absent or occasional
Margins	Infiltrating	Pushing
Cellular response	Variable	Prominent
Cleft-like spaces and small cysts	Usually absent	Present
Grade of malignancy	Moderate or high	Low
Local metastases	Usually present	Absent
Distant metastases	May be present	Absent
Prognosis	Depends on various factors. but is never as favorable as in verrucous squamous carcinoma	Excellent







Carcinoma of the lip

* Elderly men. lower lip (88.3%)
* tobacco (pipe smoking), syphilis
  sunlight. poor oral hygiene , leukoplakia
* ipsilateral - submental or submax.
* contralateral
* early Tx - good prognosis









Carcinoma of the buccal mucosa

* chewing tobacco, betel nut.
* betel nut quid contains tobacco slaked lime
* leukoplakia
* along or inferior to a line opposite the plane of occlusion



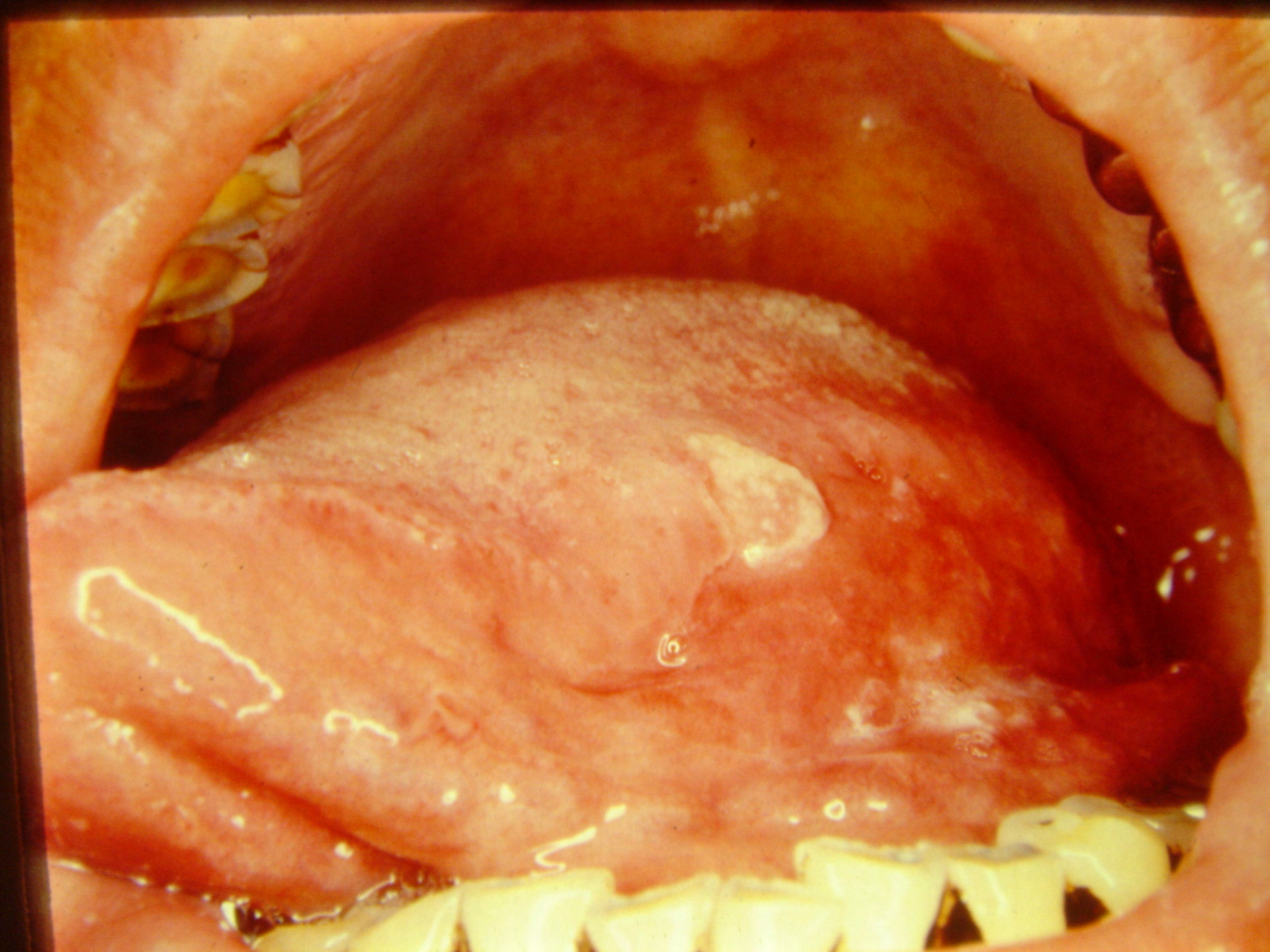




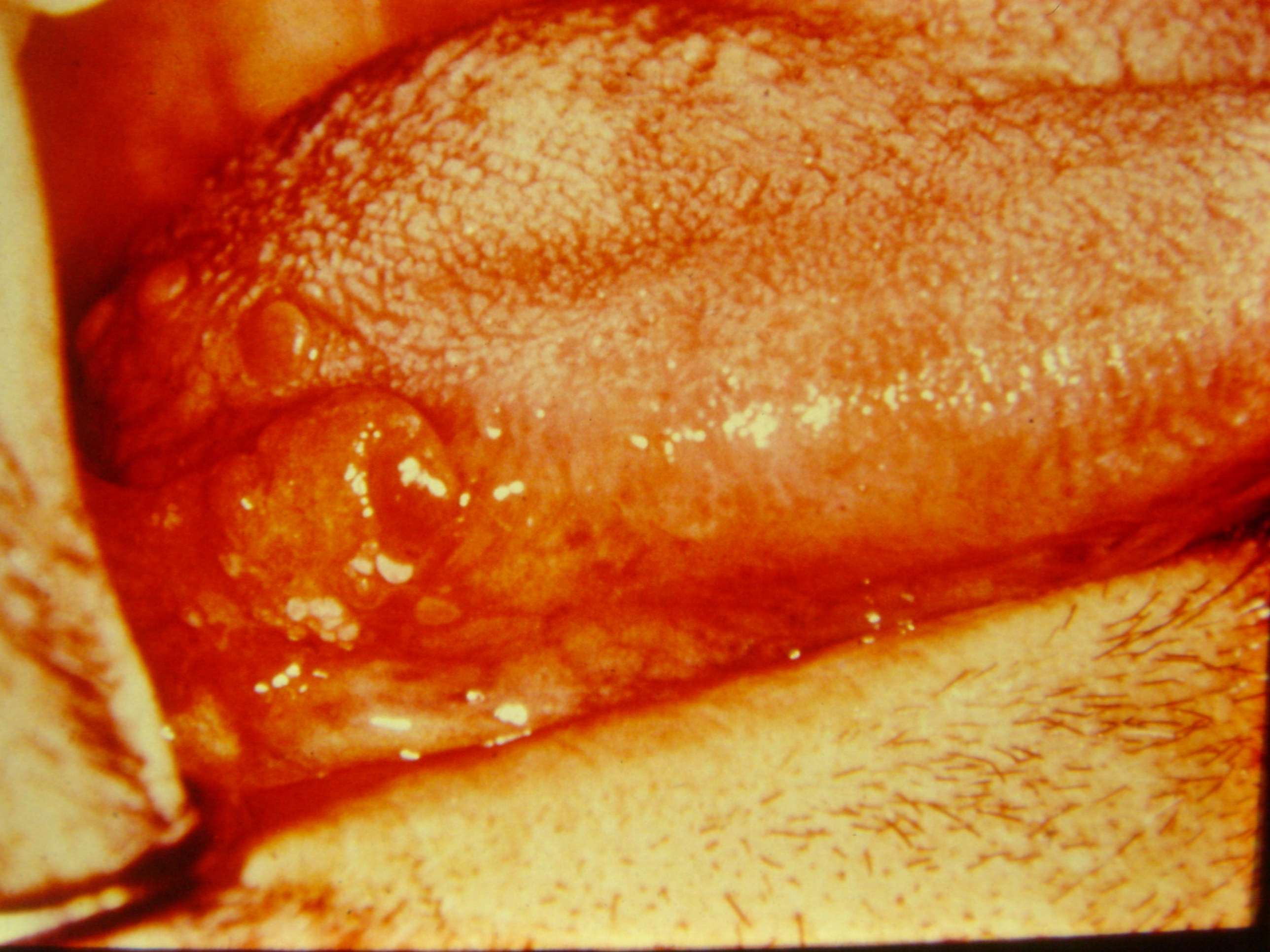
Carcinoma of the tongue

* 25 -50% of oral ca..
* women > men
* syphilis (arsenic Tx)
* leukoplakia, poor oral hygiene.
* alcohol tobacco
* 69% cervical metastases
* Tx - radiation. surgery
* 5 year cure rate - 25%









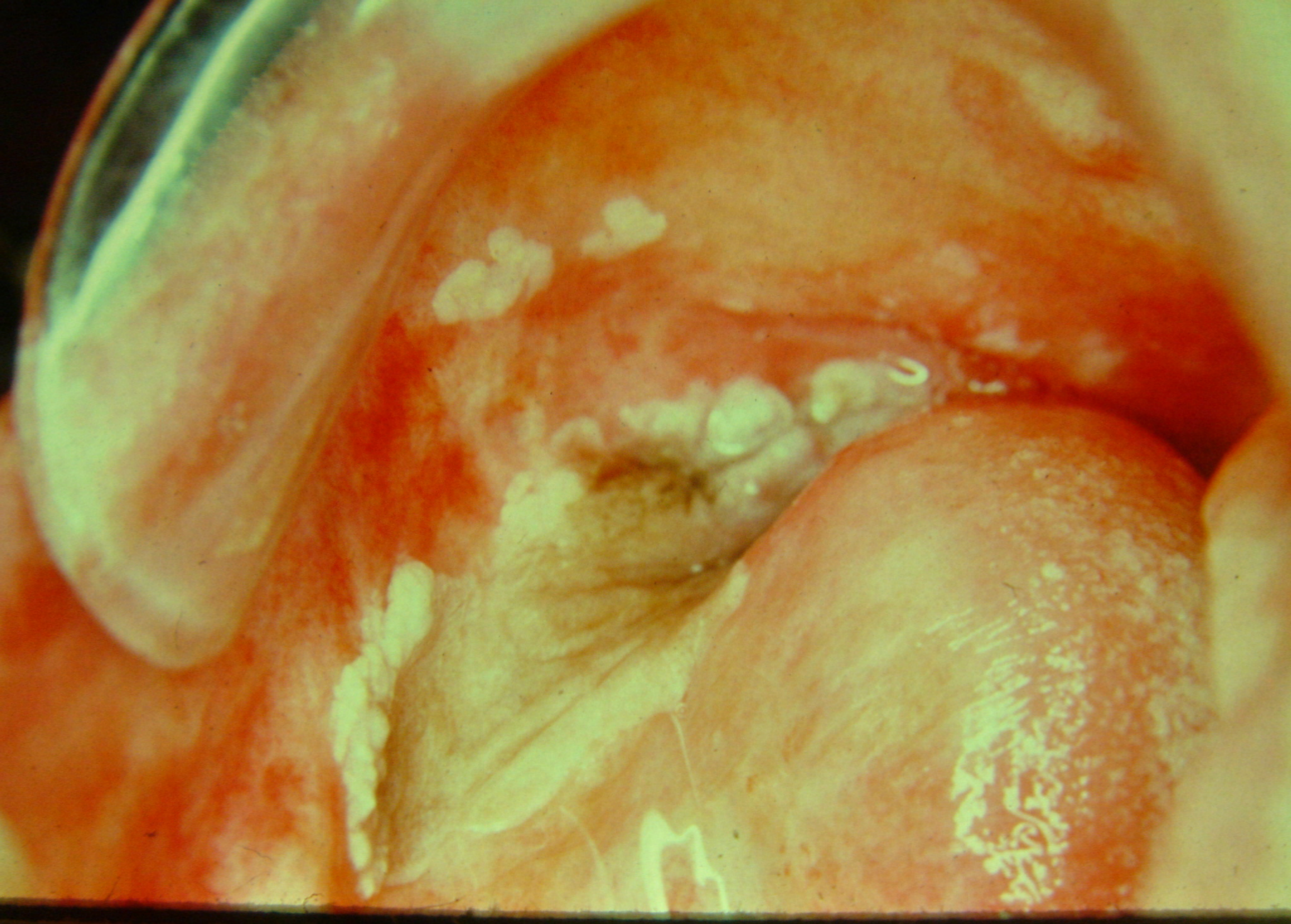





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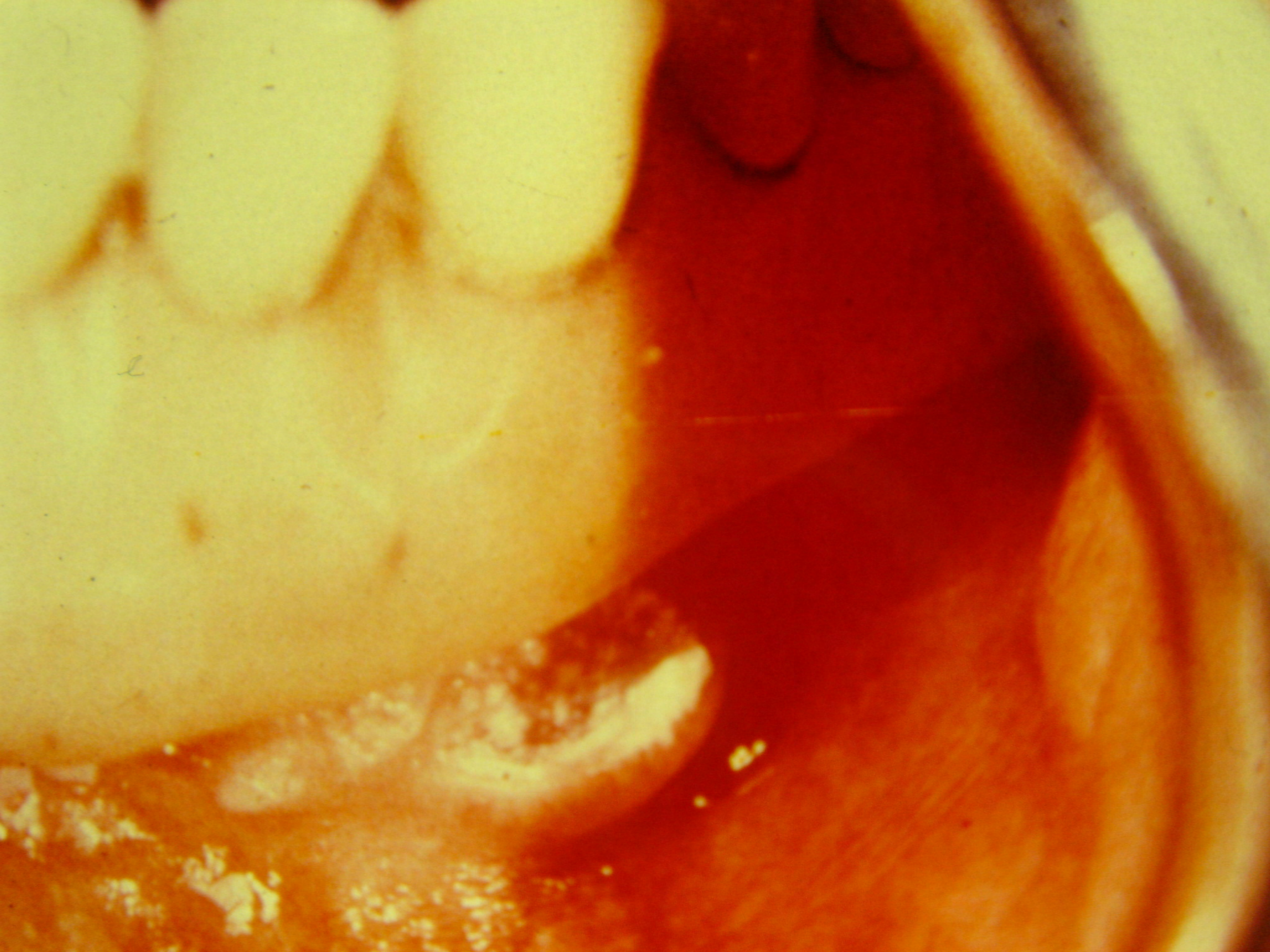


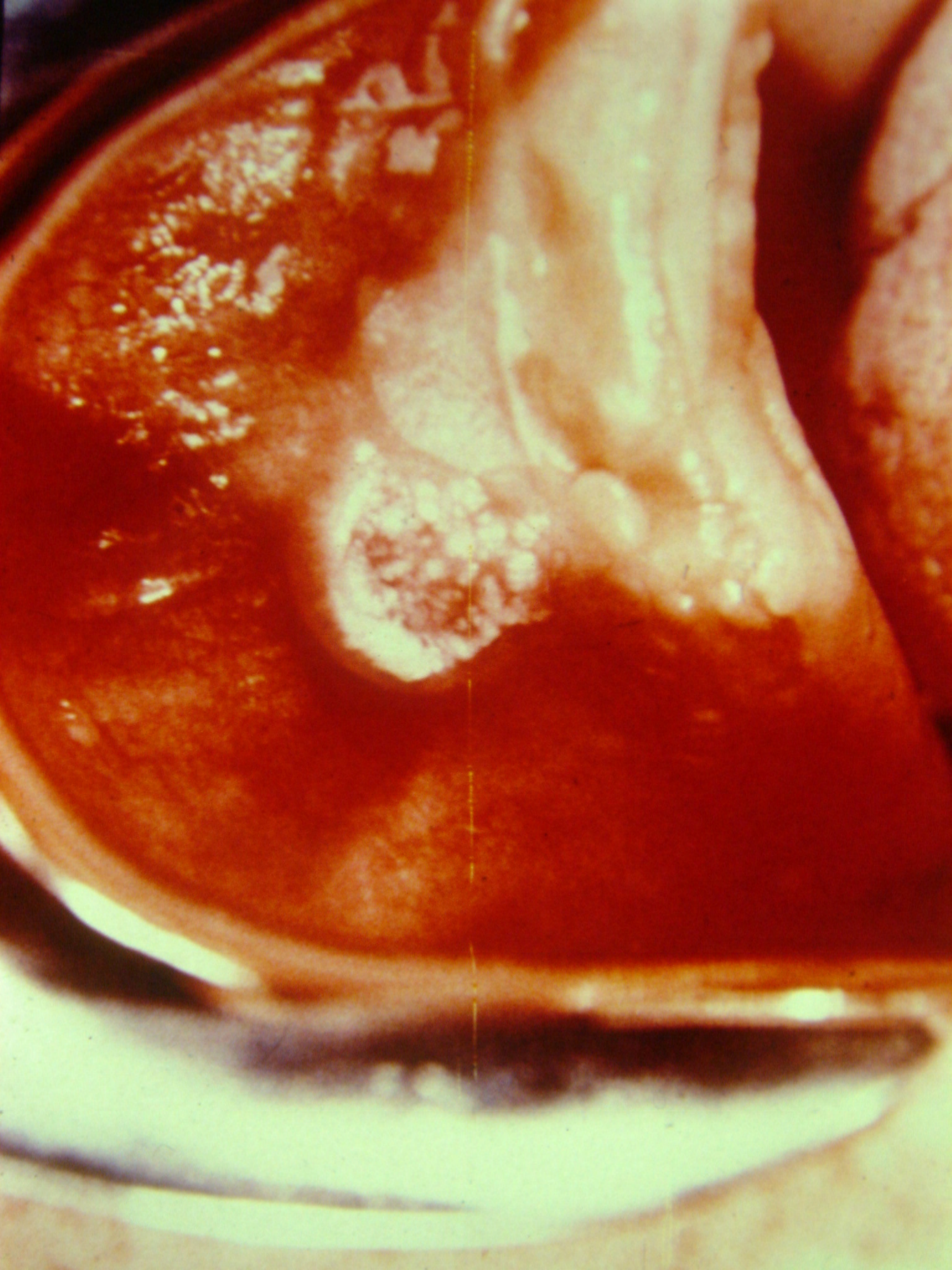
Carcinoma of gingiva

* similar to dental infection
* delay in Dx - poor prognosis
* 10 - 22% mand. > max.
* extraction w'd.
* ulceration, exophytic, granular, verrucous.
* meta. - common.









Carcinoma of palate

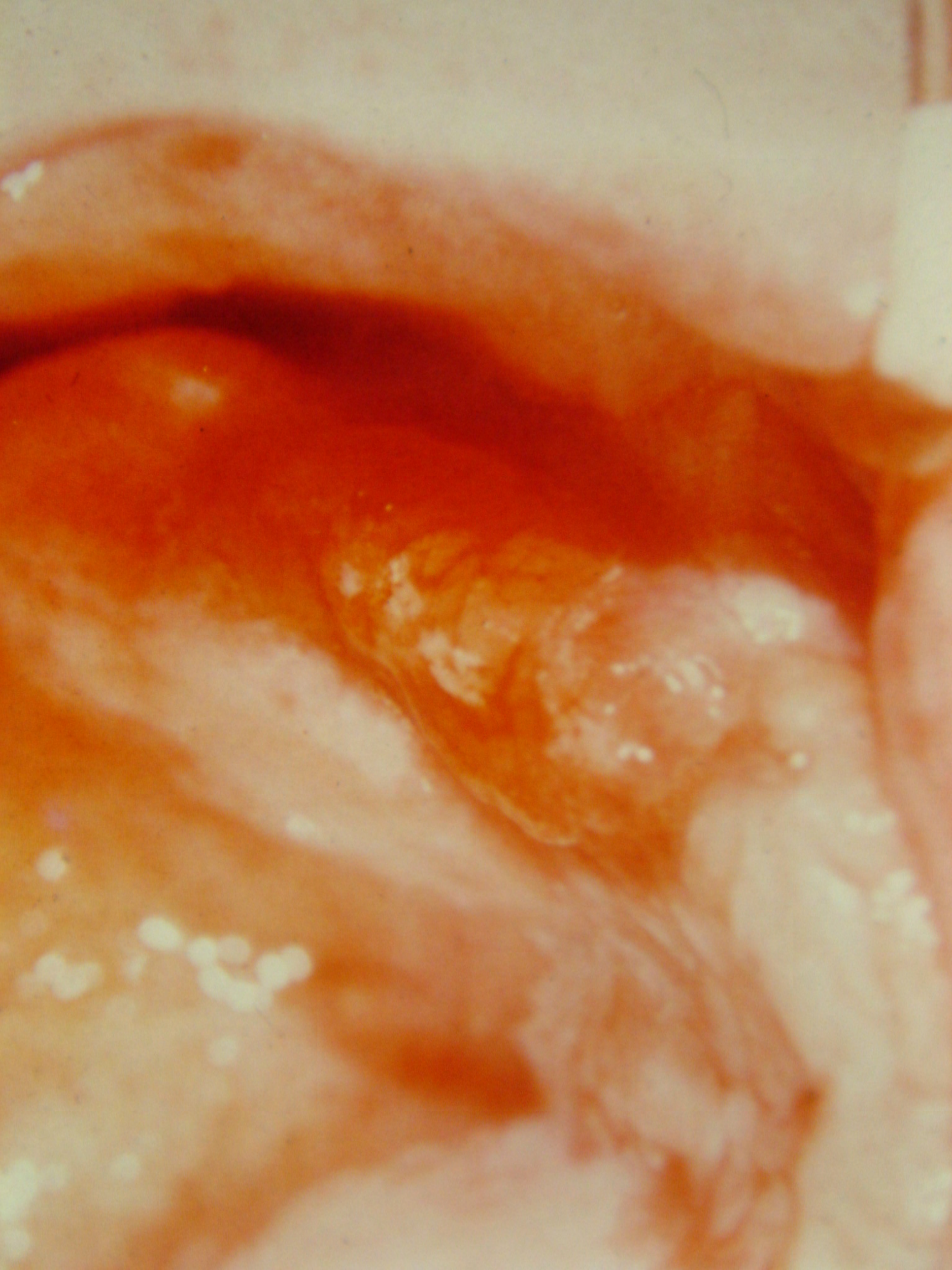
* 9% of oral epi. ca..
* soft palate (53%) hard (34%) both (13%)
* poor defined, ulcerated painful lesion
* frequently crosses the midline
* may invade into the bone, nasal cavity.

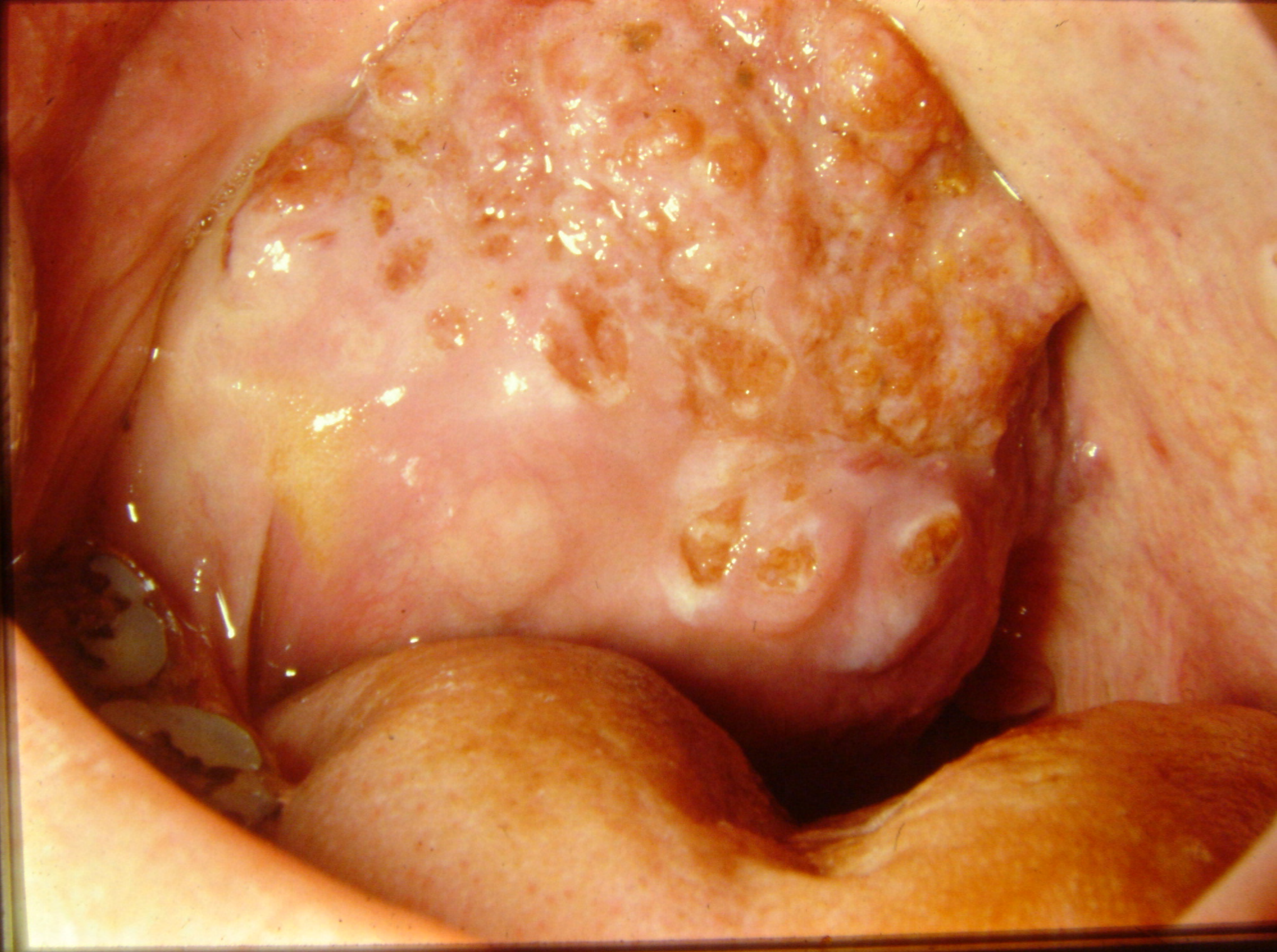


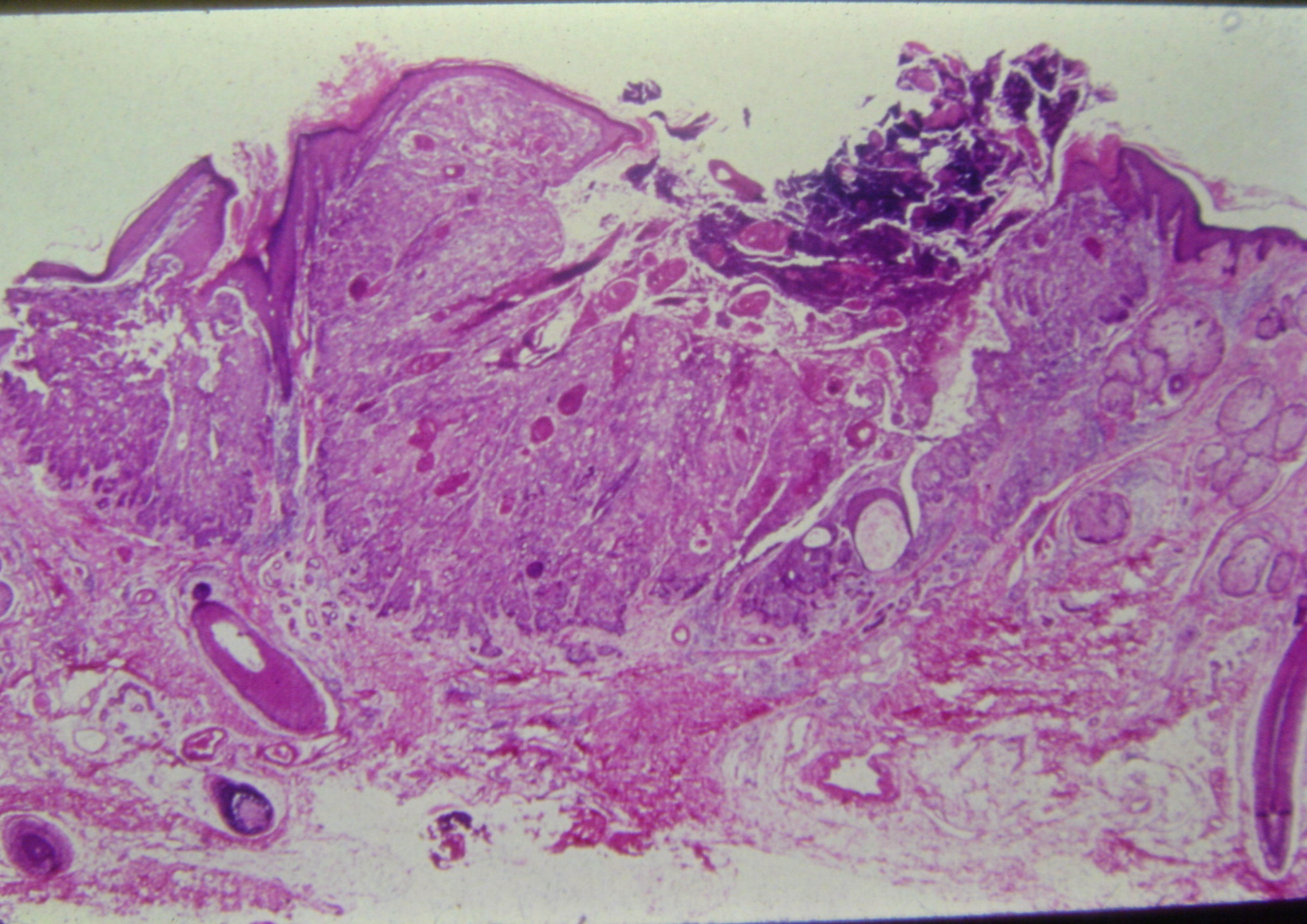


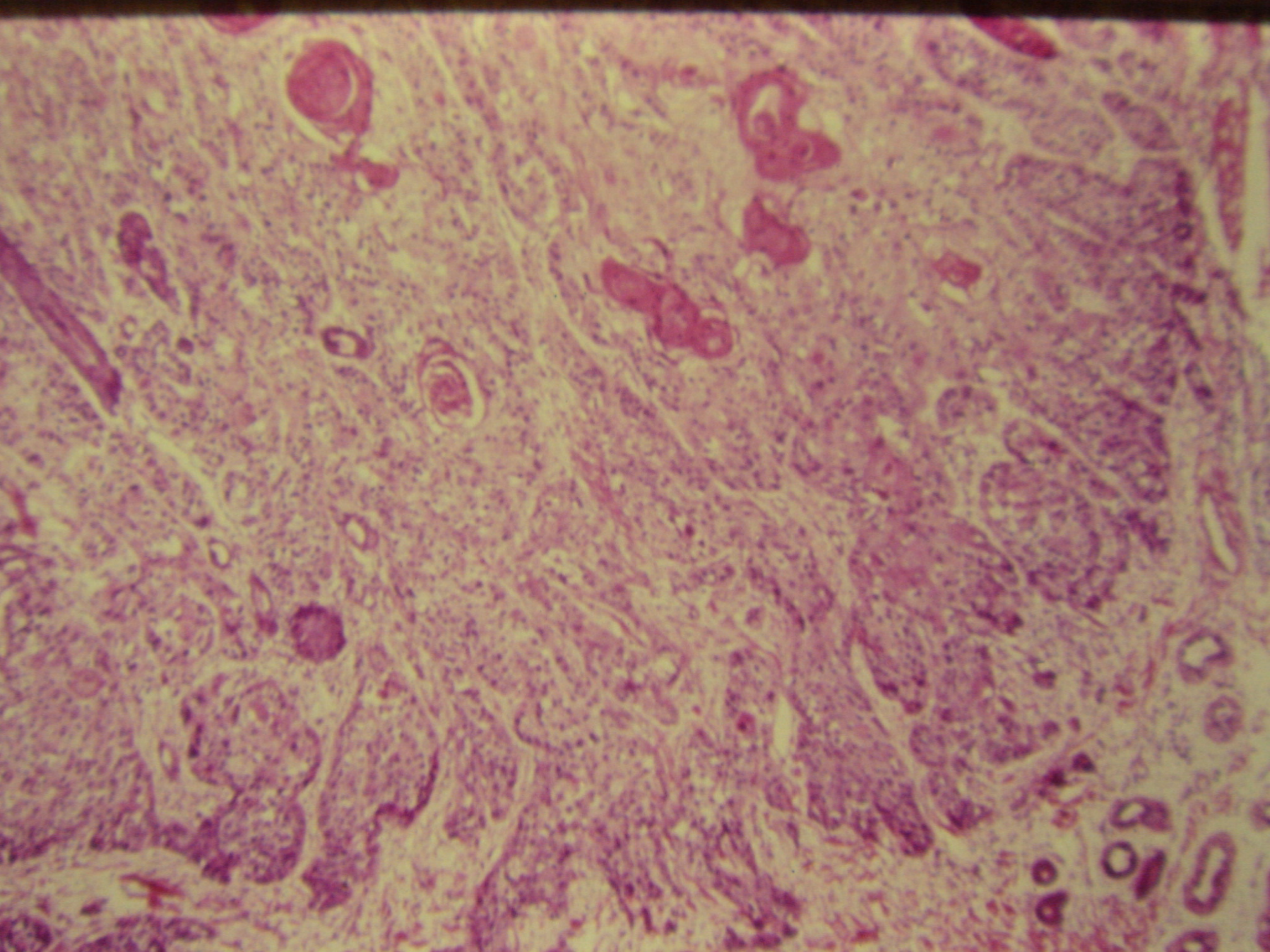
Carcinoma of maxillary sinus

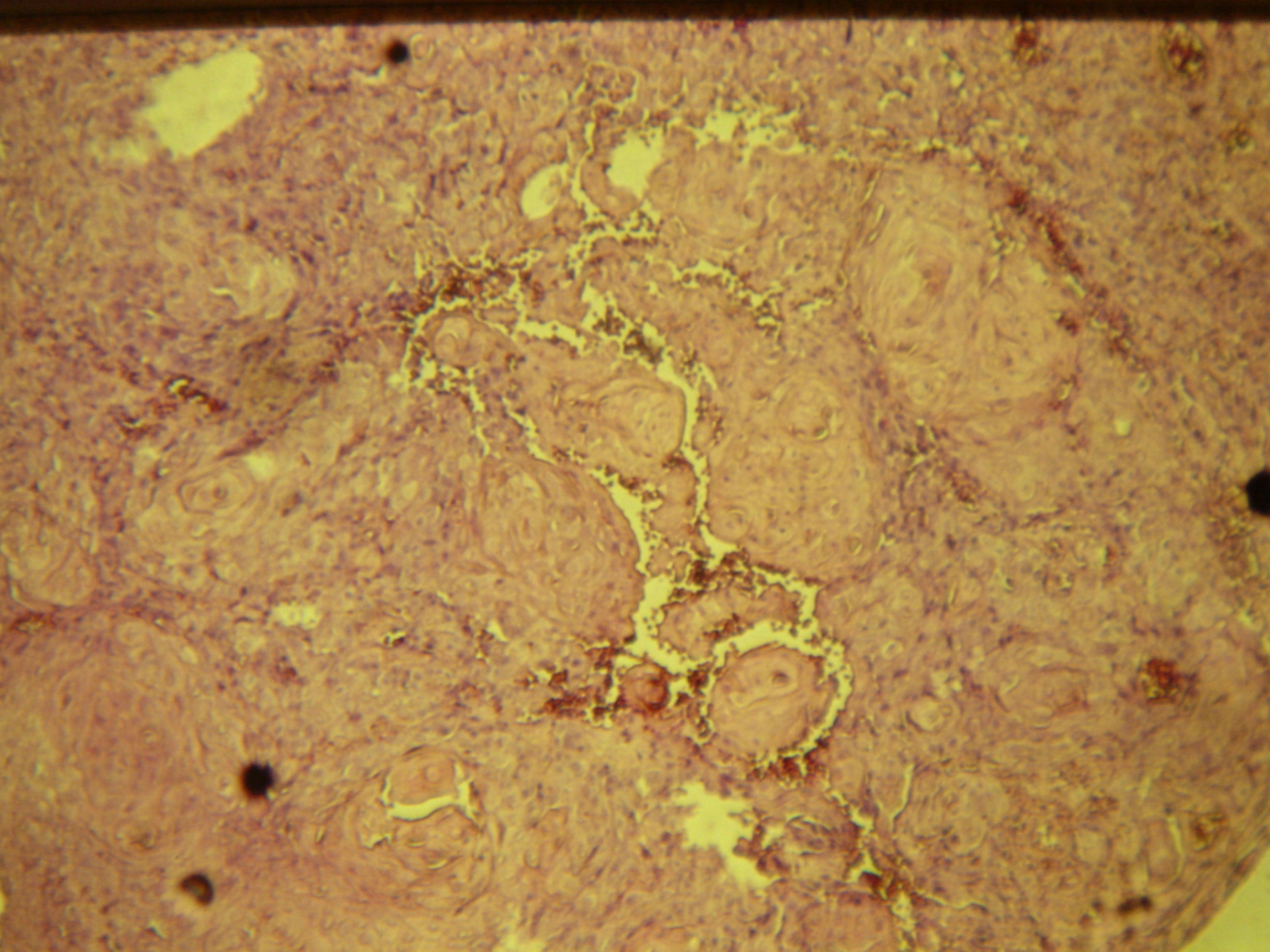
* Antral carcinoma is an exceedingly dangerous
* Chr. sinusitis doesn't related.
* Swelling or bulging of max. alveolar ridge
* palate or mucobuccal fold.
* loosening or elongation of molars.
* unilateral stuffiness , discharge.

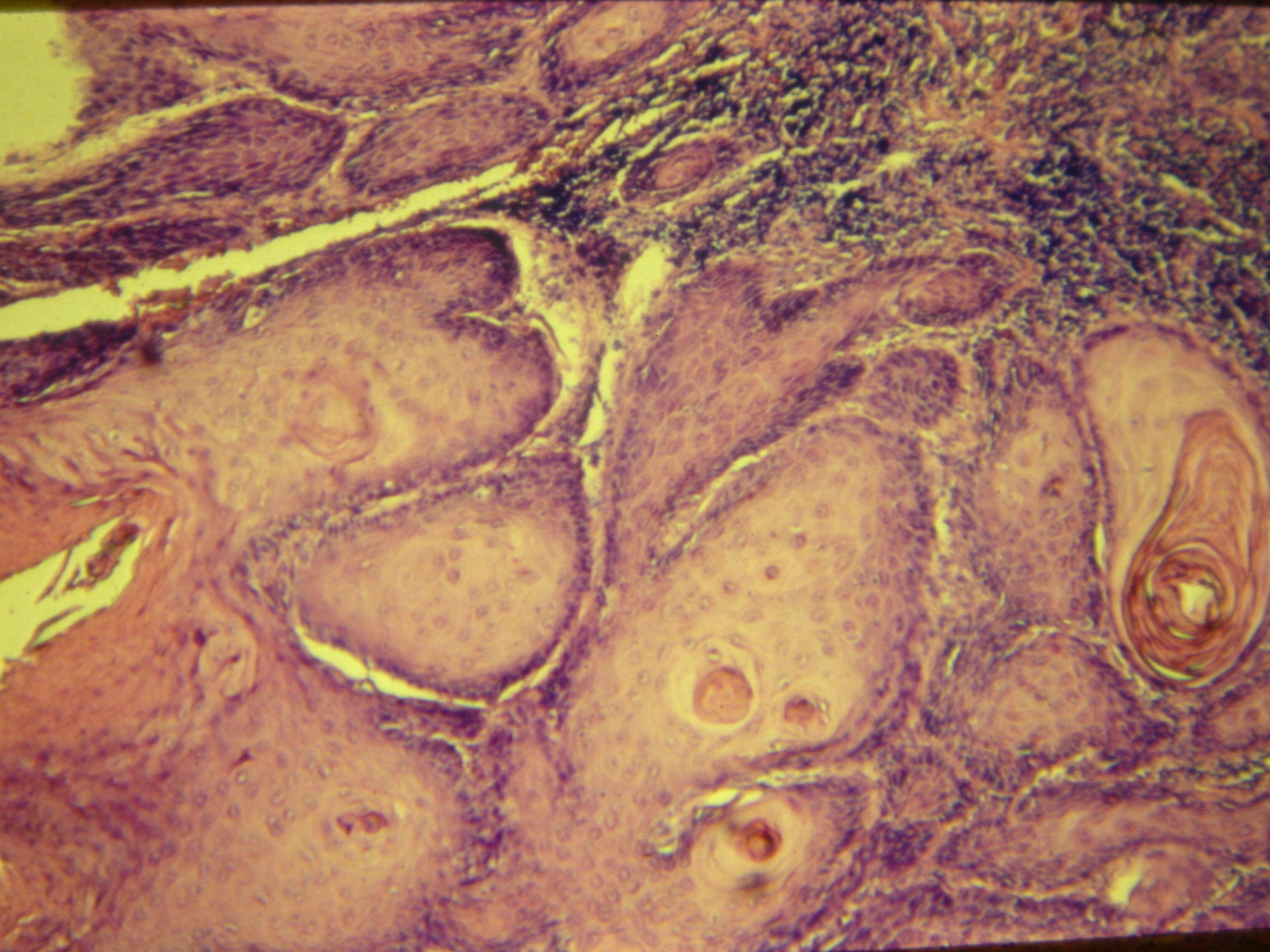








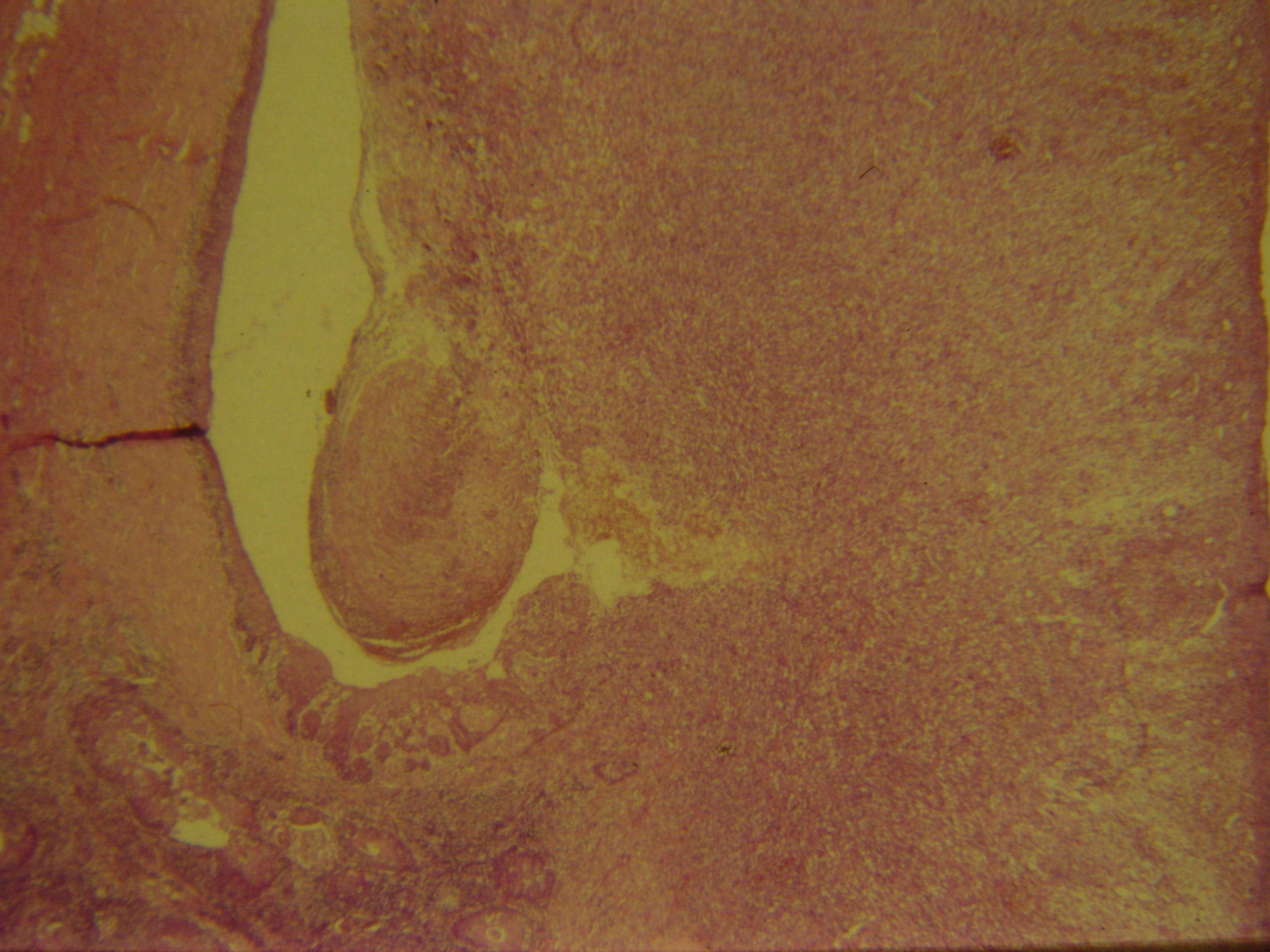


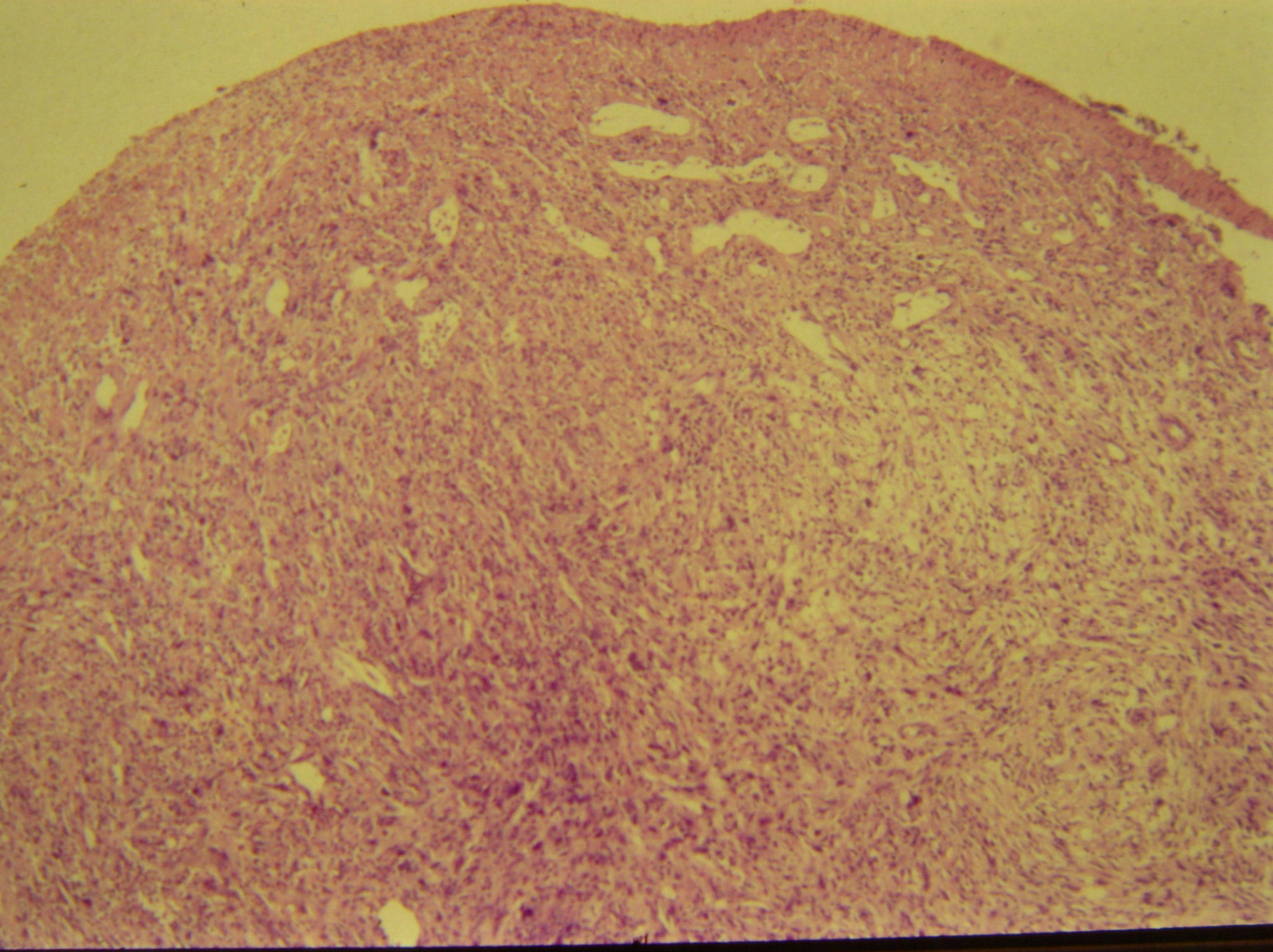


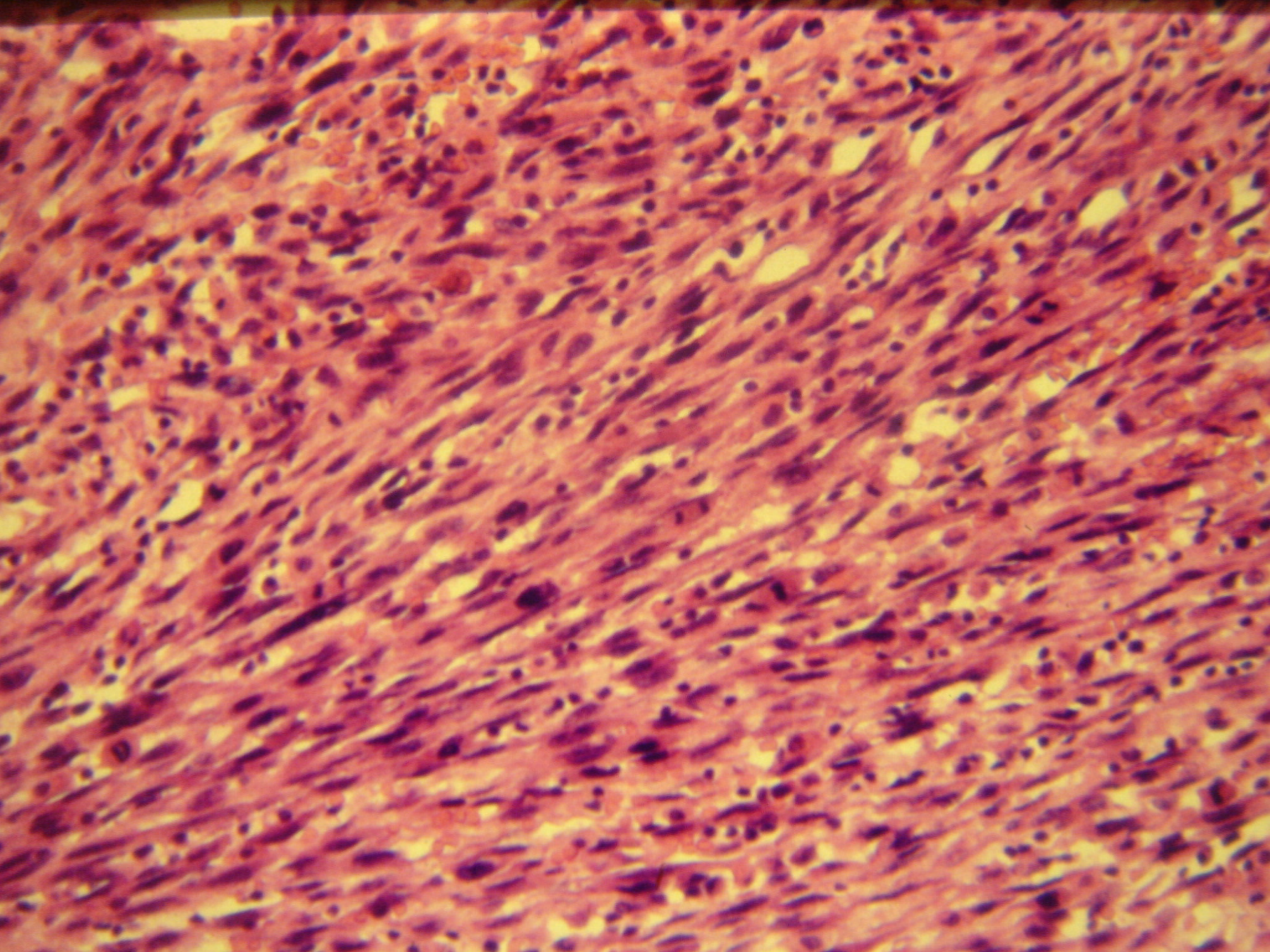
Spindle cell carcinoma

* upper respiratory & alimentary tracts.
* pseudosarcoma, carcinosarcoma.
* lower lip (42%) tongue (20%)
  alveolar redge or gingiva (19%)
* polypoid, exophytic or endophytic.
* Histo types- fasciculated, myxomatous,
  streaming.
* 81% recorded meta.









*Adenoid squamous cell carcinoma

*Lymphoepithelioma and transitional cell carcinoma

*Malignant melanoma

Adenoid squamous cell carcinoma

* frequency on the lip.
* originates from the pilosebaceous structure
* or arise from the area of senile keratosis with acantholysis.

* 93% in head & neck.
* ulcerated, hyperkeratotic or exophytic.
* tubular ductal structure, with dyskeratotic cells.

Lymphoepithelioma & transitional cell carcinoma

* nasopharynx, oropharynx, tongue, tonsil.
* young or middle- aged persons.
* transitional type of stratified epi.
* schneiderian membrane.
* extremely malignant.
* rapid clinical, meta. widely.

Clinical features

* elevated ulcerated granular eroded surface.
* indurated, exophytic or fungating growth.
* swelling of regional lymph nodes
* sore throat, nasal obstruction, headache epistaxis, ocular symptoms.

Tx & prognosis

highly radiosensitive

unfavorable Px due to wide meta.

5 year survival rate 30%.

SCC in this area ---> 5 yr. rate 11%

Treatment and prognosis

*grave prognosis

*a late sign of the neoplastic dis.

*x-ray radiation

*sex hormones

*adrenalectomy, hypophysectomy

*chemotherapeutic agent(ACP)

Malignant melanoma

* neoplasm of epidermal melanocytes
* 3rd most common cancer of skin
* Southern Arizona 6 x than other are
* arise from Junctional nevi.
* radial- growth & vertical- g. phase

Melanoma

* eye, m.m., parotid gl.
* most common sites for Japanese
* palate, max. gingiva / alveolar ridge.
* atypical melanocytes c̄ in the basal layer.
* > 0.75 mm. in thickness and located in BANs (back, arm, neck & scalp) have a greater tendency to meta.

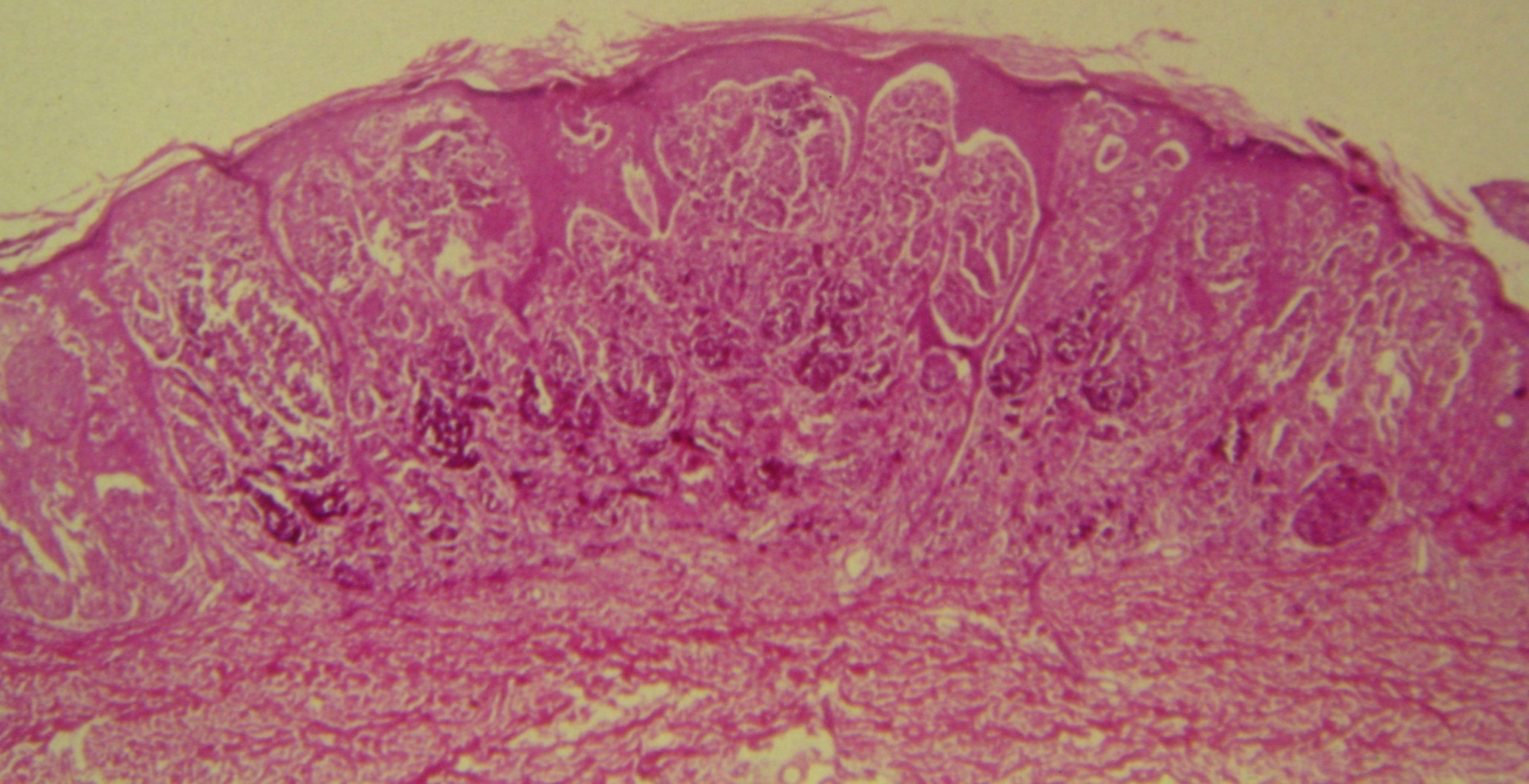


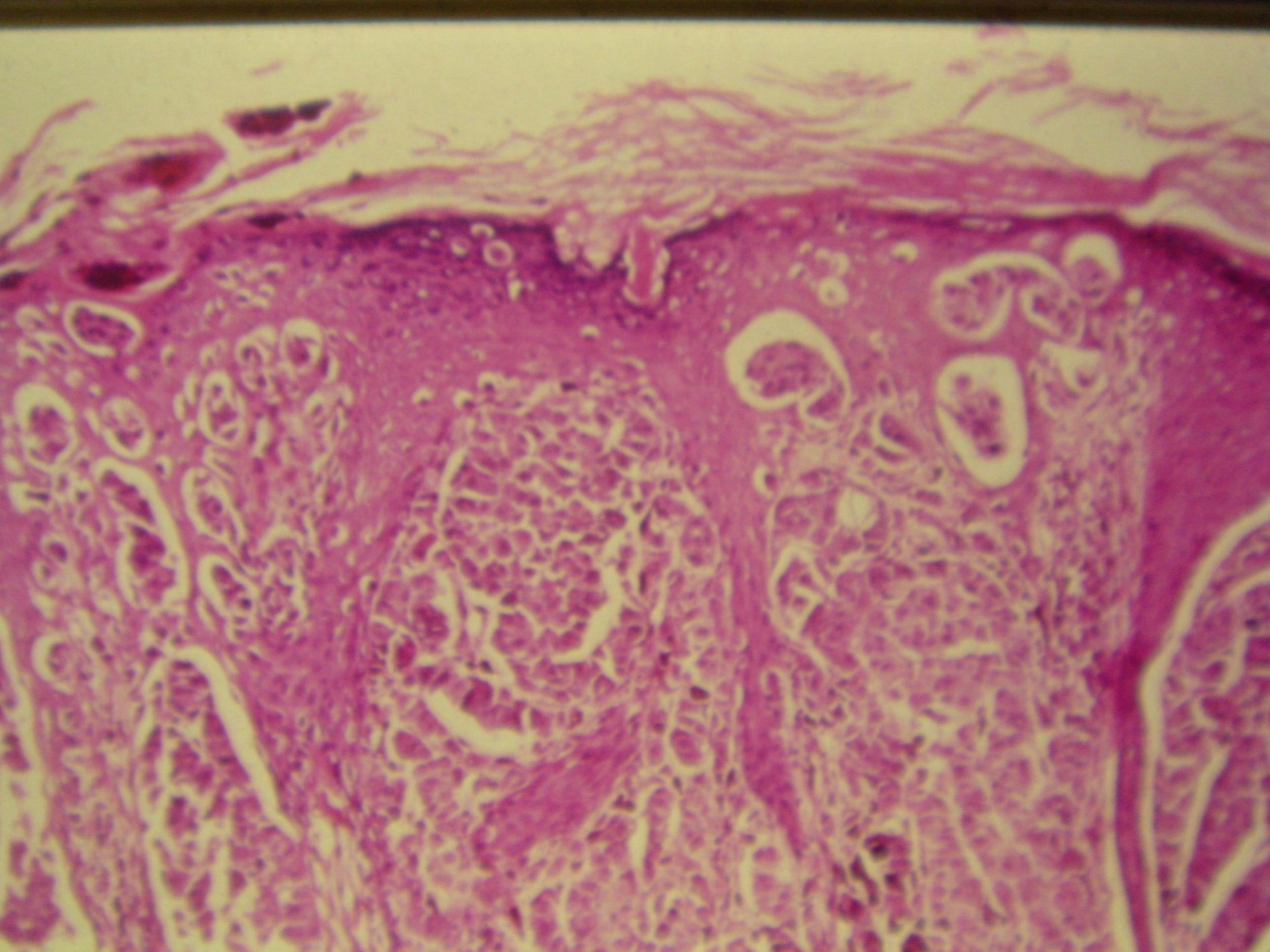


Types: 1. superficial spreading 65%,
radial growth - pagetoid melanoma
in situ, back, sun-exposed skin.

2. Nodular melanoma 13%
no radial growth just vertical
amelanotic melanoma

3. Lentigo maligna melanoma 10%
melanotic freckle of Hutchinson.





Tx

* surgical excision
* cryosurgery, immunotherapy.
* radiation, chemotherapy .
* 5 year survival rate - 7%