Connective tissue pathology

Benign connective tissue tumors

Fibroma

- **(Irritation Fibroma, Traumatic Fibroma, Focal Fibrous Hyperplasia, Fibrous Nodule)**

- **Hyperplasia of fibrous connective tissue that evolves in response to chronic irritation in which there is extensive elaboration of collagen resembling scar tissue.**

Clinical Features

- Exophytic
- Firm nodule
- Assymptomatic
- Usually smaller than 2 cm
- Buccal mucosa, tongue, lower lip, gingiva
- May be hyper-keratinized
- Due to trauma or chronic irritation
- May be ulcerated

Etiology

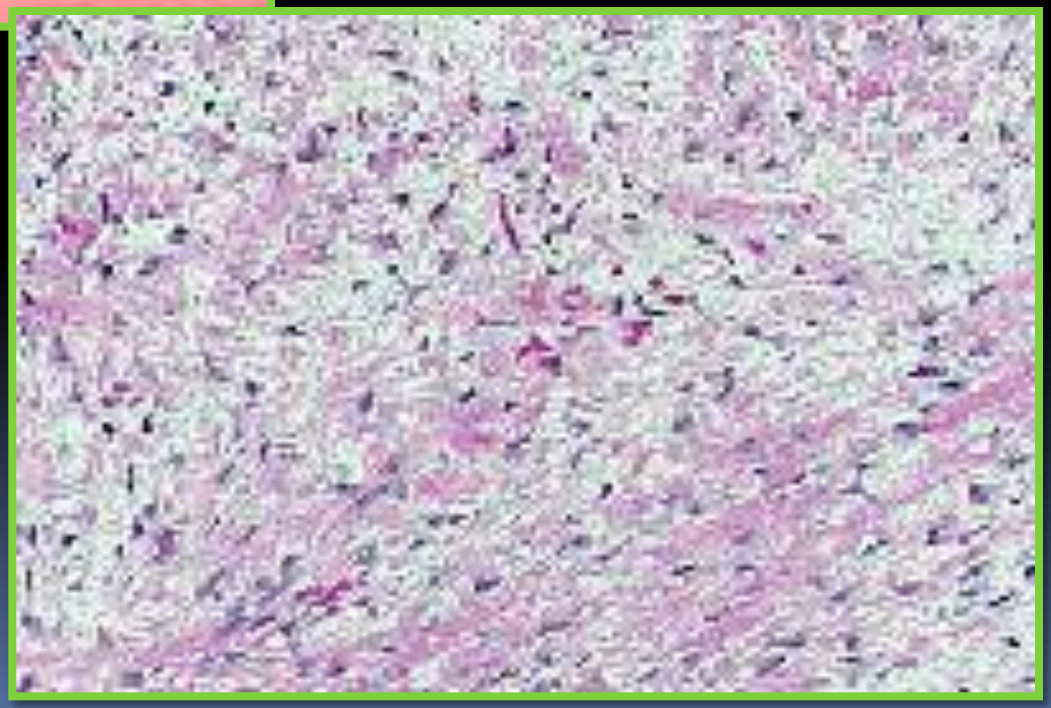
- Chronic irritation or trauma

Tissue of origin

- Fibrous connective tissue

Histological Features

- Stratified squamous epithelial (maybe keratinized) – appears stretched
- Proliferation of fibroblasts and collagen fibers (thick bundles of fibers) – Whorled pattern
- Compressed blood capillaries



Main Pathologic Process

- Benign hyperplasia (reactive)

Treatment

- Excision; remove source of irritation

Giant cell Fibroma

- The giant cell fibroma is a subtype of irritation fibroma, i.e. it is a localized inflammatory fibrous hyperplasia.

- It differs from routine fibromas in that its stroma contains scattered fibroblasts with very large, usually angular (stellate), but **not hyper-chromatic** nuclei.

Clinical Features

- Any age
- Any oral mucosal surface, but it has a predilection for gingival, lateral tongue and tip of tongue locations.
- Seldom becomes more than 0.5 cm. In greatest diameter and often are comprised of two or three lobules, or may have a few small papules on the surface.

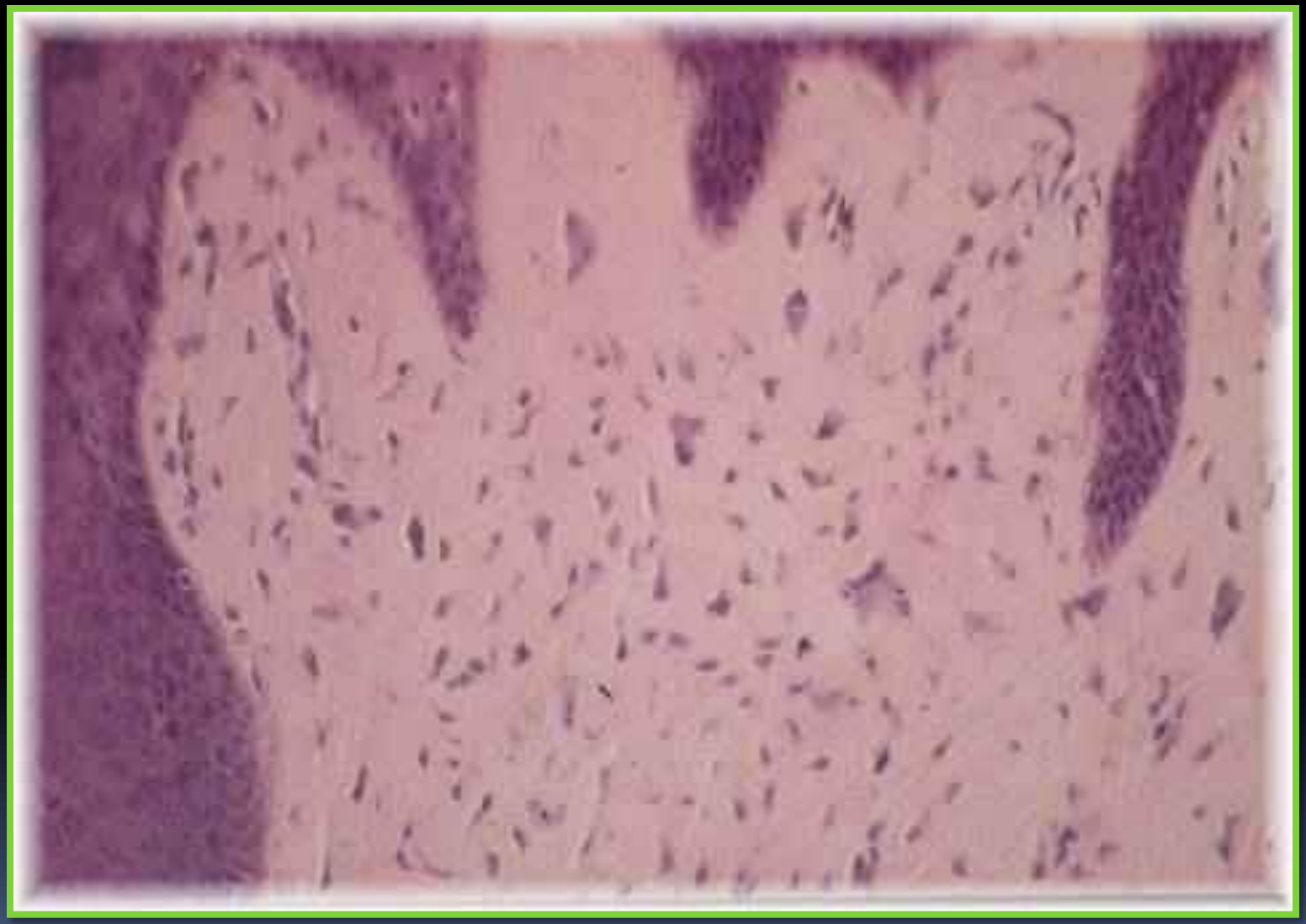
Clinical Features

- Most lesions are pedunculated
- The surface is almost never ulcerated, unless acutely traumatized
- Color is that of the surrounding normal mucosa

Histopathology

- Diffuse, somewhat immature, rather avascular collagenic stroma
- Small bipolar and slightly stellate fibroblasts scattered throughout in moderate numbers.
- Occasional fibroblasts will be quite large and angular, and may have more than one nucleus
- These pathognomonic cells are never hyperchromatic, as they would be if they were truly dysplastic fibroblasts.

- They are most numerous in the zone immediately beneath the covering epithelium.
- The epithelium is usually normal but may have elongated and narrow rete-ridges.
- Occasional lymphocytes may be seen beneath the epithelium or around capillaries.



Treatment and Prognosis

- Conservative surgical removal but is more likely to recur with this treatment than a more routine irritation fibroma
- If left alone, the giant cell fibroma will not enlarge indefinitely, but will reach its maximum size within a few months and remain unchanged thereafter, unless acute trauma produces edema or areas of granulation tissue proliferation

PERIPHERAL GIANT CELL GRANULOMA

PERIPHERAL GIANT CELL GRANULOMA

- Goddorn & gross,1856-fungus excrescence of gingiva
- Giant cell epulis, myeloid epulis,
- Jaffe 1952 – giant cell reparative granuloma
- Bernier & cahn Peripheral giant cell reparative granuloma

DEFINITION

- Peripheral Giant Cell Granuloma is an oral, non-neoplastic, tumorous proliferation that frequently involves gingival tooth bearing areas.

Thomas Oral Pathology 6th ed

Etiology

Local Irritation

Hormonal Influence (*Whitaker & Bouquet*)

Pathogenesis

- Phagocytic response , osteoblastic function
- Proliferation of cells

CLINICAL FEATURES

AGE

5^{th} –6^{th} decade

2^{nd} –3^{rd} decade(20 – 33%)

SEX

Females (60%)

SITE

Post mandible- Premolar- Molar ,
Incisor- Canine

Attached gingiva & alveolar mucosa

PRESENTATION

- Hourglass Appearance
- Size : 0.5cm – 5cm , < 2cm in 94% cases
- Color: Red,Purple or Blue
- Surface: Smooth, Nodular or Ulcerated

SIGNS & SYMPTOMS

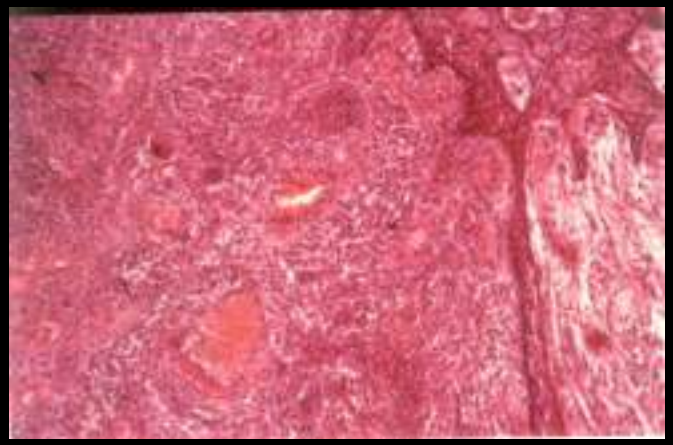
- Asymptomatic
- Displacement of teeth
- Speech & mastication

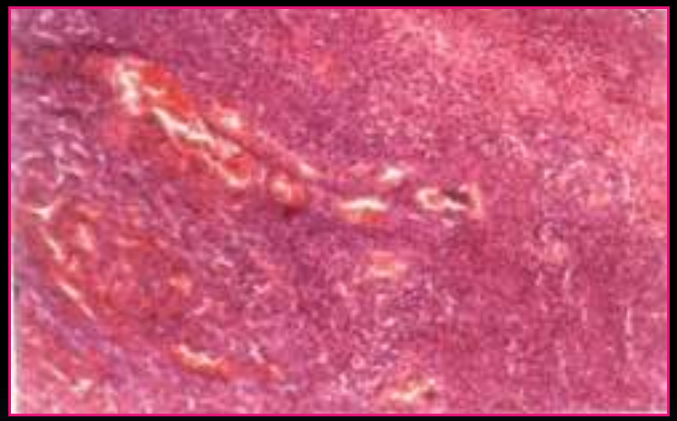
RADIOGRAPHIC FEATURES

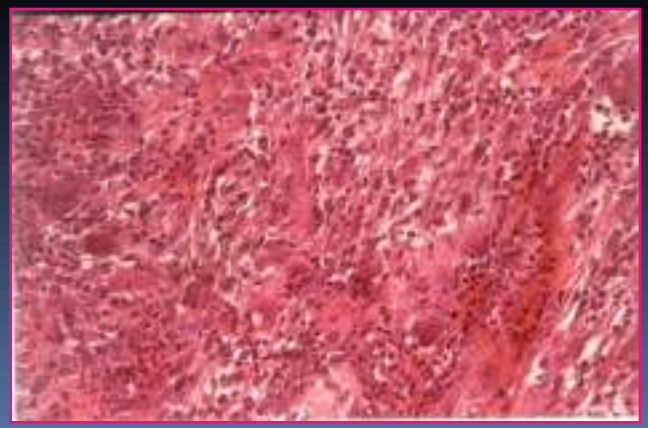
- Cupping or Superficial bone resorption
- Focal loss of alveolar bone
- Widening of periodontal ligament

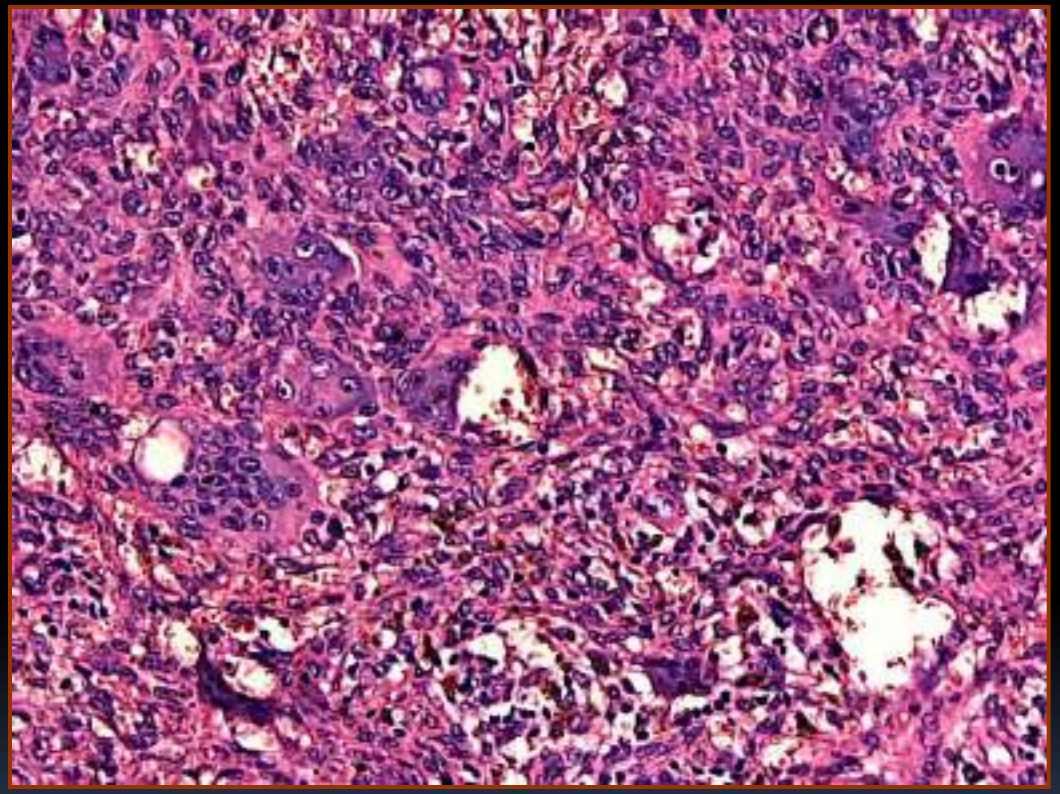
HISTOPATHOLOGIC FEATURES

- Keratinised Stratified Squamous Epithelium
- Multinucleated Giant Cells
- Hemorrhage & Hemosiderin









DIFFERENTIAL DIAGNOSIS

- Irritation Fibroma
- Peripheral Counterpart of CGCG
- Peripheral Ossifying Fibroma
- Peripheral Odontogenic Fibroma
- Epulides
- Acquired Hemangiomas
- Malignant Tumors
- Metastatic Tumors

DIAGNOSIS

- Clinical Presentation
- Incisional Biopsy
- Serum Electrolytes

MANAGEMENT

- Surgical Excision
- Elimination of Local Factors
- Surgical Defect

RECURRENCE RATE - *10 – 12%*

Katsikeris et al 9.8%

Bhaskar et al – 12%

Sorensen – 7.7 %

Giasanti & Waldron – 4.9%

Andersen et al – 70.6%

COMPLICATIONS

- Loss of Adjacent Teeth
- Compromised oral Function

CENTRAL GIANT CELL GANULOMA

Introduction

Central giant cell granuloma (CGCG) is an intraosseous destructive lesion of the anterior mandible and maxilla in which larger lesions expand the cortical plate, cause movement of teeth, and produce root resorption; Composed of multinucleated giant cells in a background of mononuclear fibrohistiocytic cells and red blood cells.

- **Non-aggressive CGCG:**

  - slow growth.

  - less recurrent rate.

  - less common root resorption & cortical perforation.

- **Aggressive CGCG:**

  - rapid growth.

  - high recurrent rate.

  - common root resorption & cortical perforation.

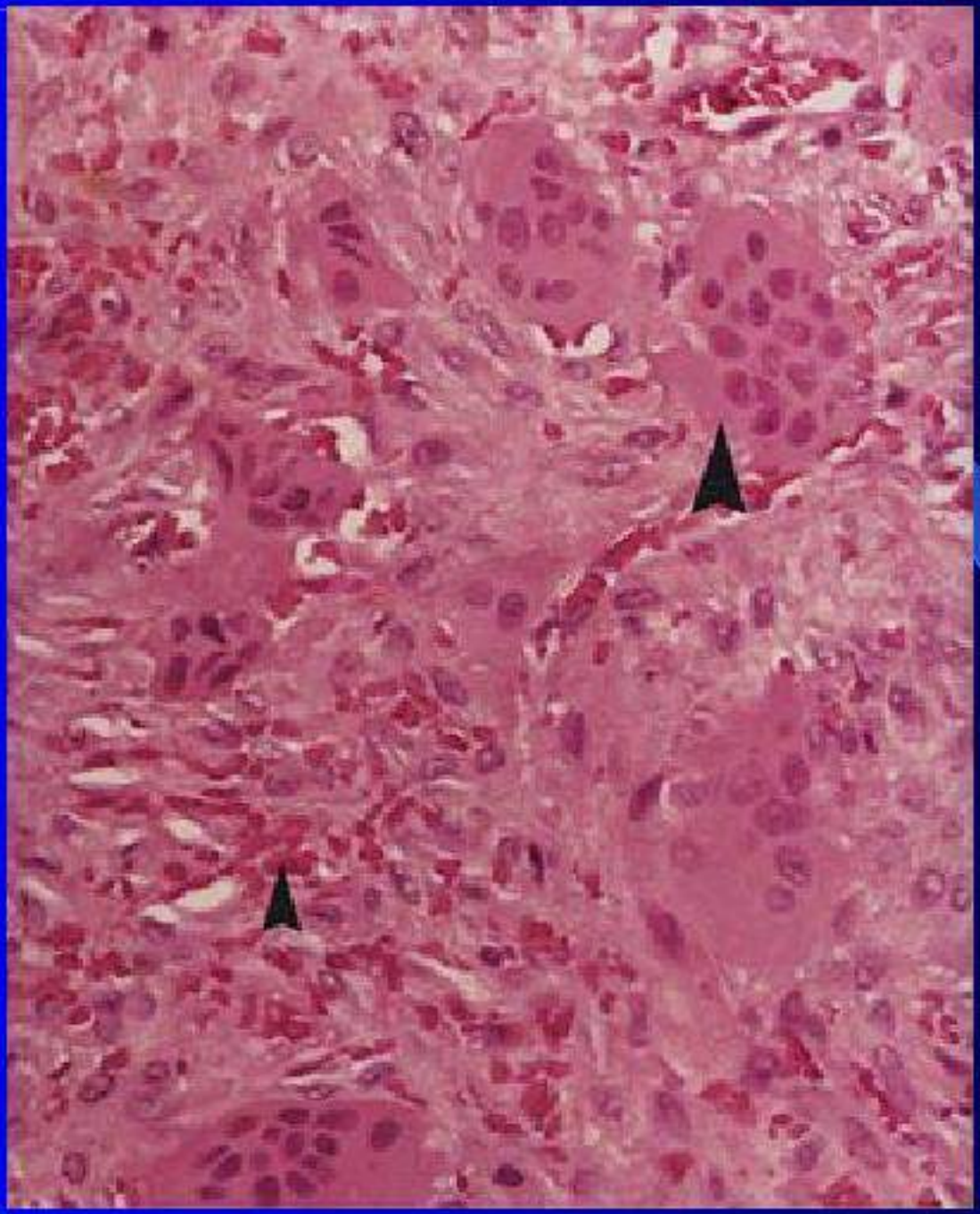
  - likelihood of being painful & paresthesia.

Radiographic appearance

- Radiolucency.
- Indistinct line of demarcation.
- Complete cortical bone loss.
- Buccal & lingual expansion.
- Resorption of tooth roots.
- Unilocular or multilocular.

Histopathology

- Proliferating endothelial cells.
- Small capillaries.
- Multinucleated giant cells. (char. of osteoclast)
- Mitotic figures of mononuclear cells.
- Active fibroblast & myofibroblast.
- Fibrous stroma.
- Extravasated RBC.
- Hemosiderin pigment.
- Osteoid & newly forming bone.



Treatment

- **Surgical :**
  - Curettage
  - Block resection (if aggressive lesion)
  - Extraction if unerupted tooth involved.

- **Non-surgical :**
  - Systemic Calcitonin therapy
  - Intralesional Glucocorticosteroids.

ANEURYSMAL BONE CYST

- ABC
- **An uncommon expansile osteolytic lesion of bone consisting of a proliferation of vascular tissue that forms a lining around blood filled cystic lesion**
- **Reactive non-neoplastic**

Etiology

- Unknown
  - May be primary or secondary
- secondary ABC in 30% of cases
  - GCT
  - CHONDROBLASTOMA
  - OSTEOBLASTOMA
  - OSTEOSARCOMA

Pathogenesis

- Persistent local alteration in the hemodynamics
- Increased venous pressure
- Dilation of vascular bed
- Resorption of surrounding bone (giant cells)
- Area replaced by fibrous,osteoid &new bone

- Hematoma or any existing bone lesion

  (fails to heal)

  ↓

- Connection of circulatory system with hematoma or existing lesion

  ↓

- Welling up of blood into the lesion

Clinical features

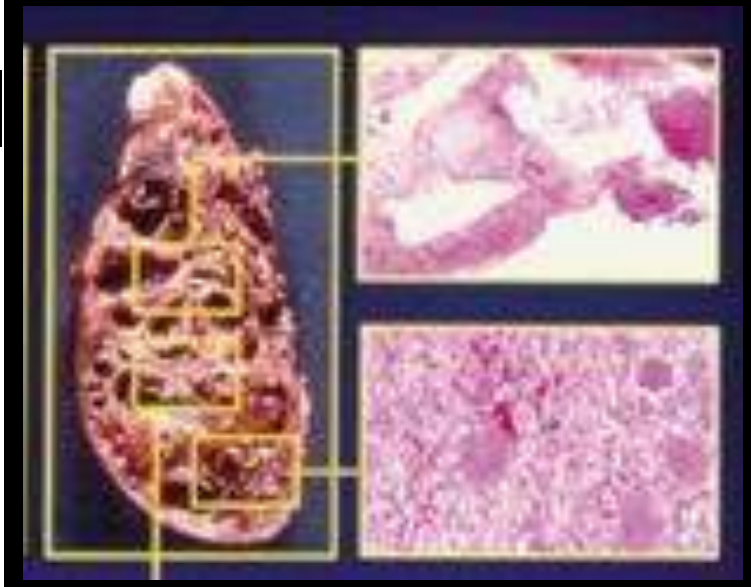
- Young individuals. M=F, young age, around 20 yrs
- History of traumatic injury
- Clavicle, rib, mandible , maxilla, other skull bones, bones of hands & feet
- Gross appearance at the time surgery BLOOD WELLING UP from the tissues
- BLOOD- SOAKED SPONGE appearence

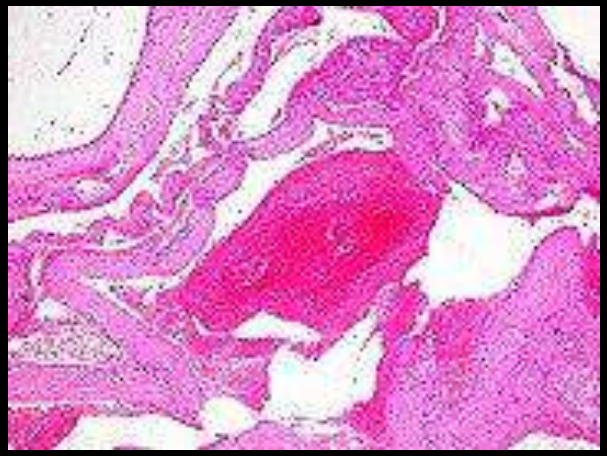
Radiography

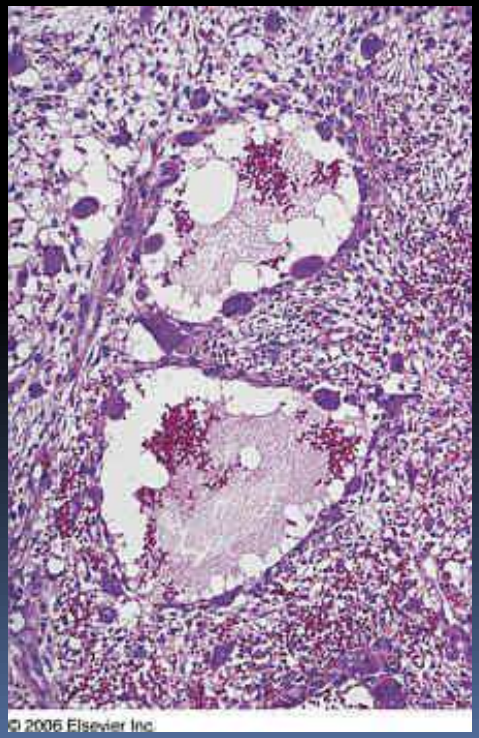
- Early lesion, Radiolucent
- Later lesions appears Expanded cortex with honeycomb or soap bubble appearance
- Destruction of cortical bone in eccentrically ballooned manner making a thin Shell Of Bone
- Marked cortical thinning and erosion and periosteal elevation

Histopathology

- Fibrous connective tissue stroma containing many blood filled cavernous or sinusoidal spaces
- Multinucleated giant cells, haemosiderin pigments , osteoid formation.



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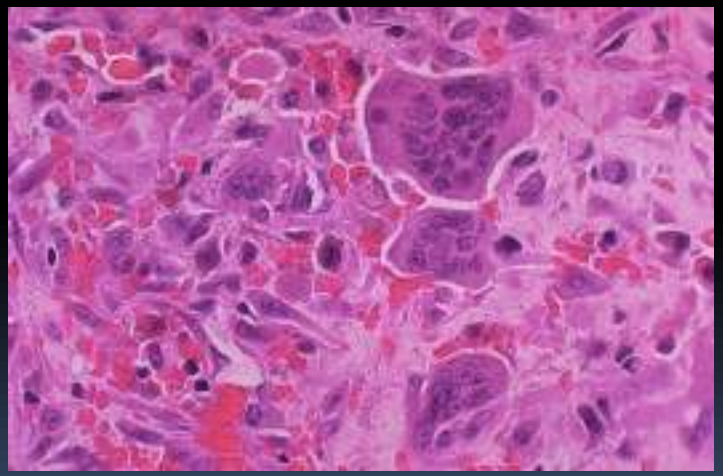

Treatment

- Curettage and bone grafting has a 20-40% recurrence rate
- Recurrance --- more aggressive curettage or excision
- Marginal excision or wide excision with bone grafting is preferable

OSSIFYING FIBROMA (CEMENTIFYING-FIBROMA; CEMENTO-OSSIFYING FIBROMA)

Ossifying fibroma is a true neoplasm with a significant growth potential & are relatively rare

The tumor is limited to the tooth bearing areas of maxilla & mandible

OSSIFYING FIBROMA (CEMENTIFYING-FIBROMA; CEMENTO-OSSIFYING FIBROMA)

- The neoplastic cells elaborate bone & cementum & hence thought to arise from progenitor cells (capable of dual differentiation) of periodontal ligament or odontogenic
- The cementum-like material present in ossifying fibromas is considered as a variation of bone (Neville)

OSSIFYING FIBROMA (CEMENTIFYING-FIBROMA; CEMENTO-OSSIFYING FIBROMA)

- Ossifying fibroma and cementifying fibroma are now considered to be the two extremes of the same spectrum; because both frequently contain both bone and cementum-like tissue; these lesions are now called cemento-ossifying fibroma (COF) *WHO

- Variations will be combined under the term, cemento-ossifying fibroma (Neville)

- OSSEOUS DYSPLASIAS

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Clinical features

- COF presents clinically as painless expansion of the jaw
- Small lesions are discovered in routine radiographs
- Occurs over a wide age range, mainly seen during the third and fourth decades of life
- Definite female predilection seen (5:1)

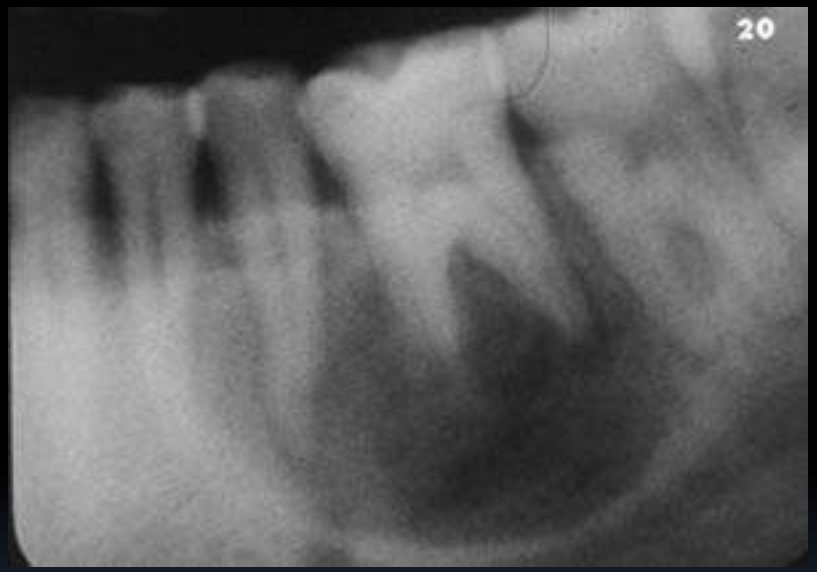
Mandible > Maxilla

Mandibular premolar and molar

Large COFs involving mandible may show a characteristic **downward bowing of the inferior cortex of the mandible**

Larger lesions causes facial symmetry. Pain & parasthesia are rarely seen

Radiographic features



- Well defined & unilocular
- Some may show a sclerotic border
- It may be radiolucent, or with varying degrees of radiopacity depending on the amount of calcified material produced
- well demarcated with different quantities of mineralized bone
- Root divergence or resorption of roots of teeth is present

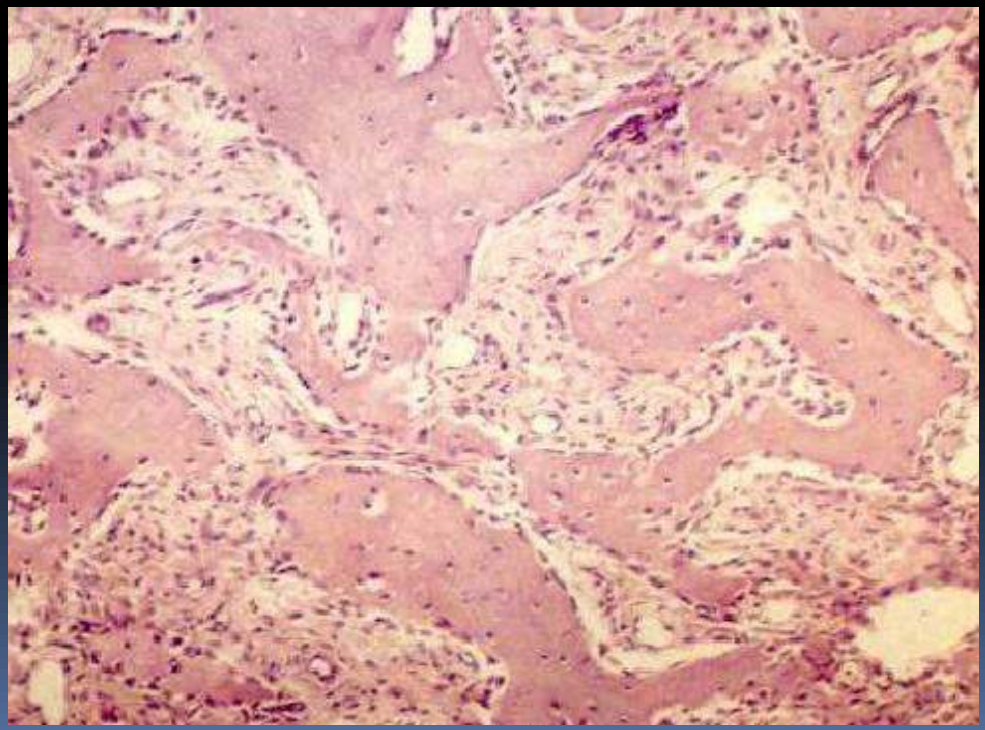
Histopathology

- Most lesions are not encapsulated but are well demarcated grossly & microscopically, from the surrounding bone & can easily be removed from its bony bed
- Consist of fibrous tissue that exhibits varying degrees of cellularity and contains mineralized material

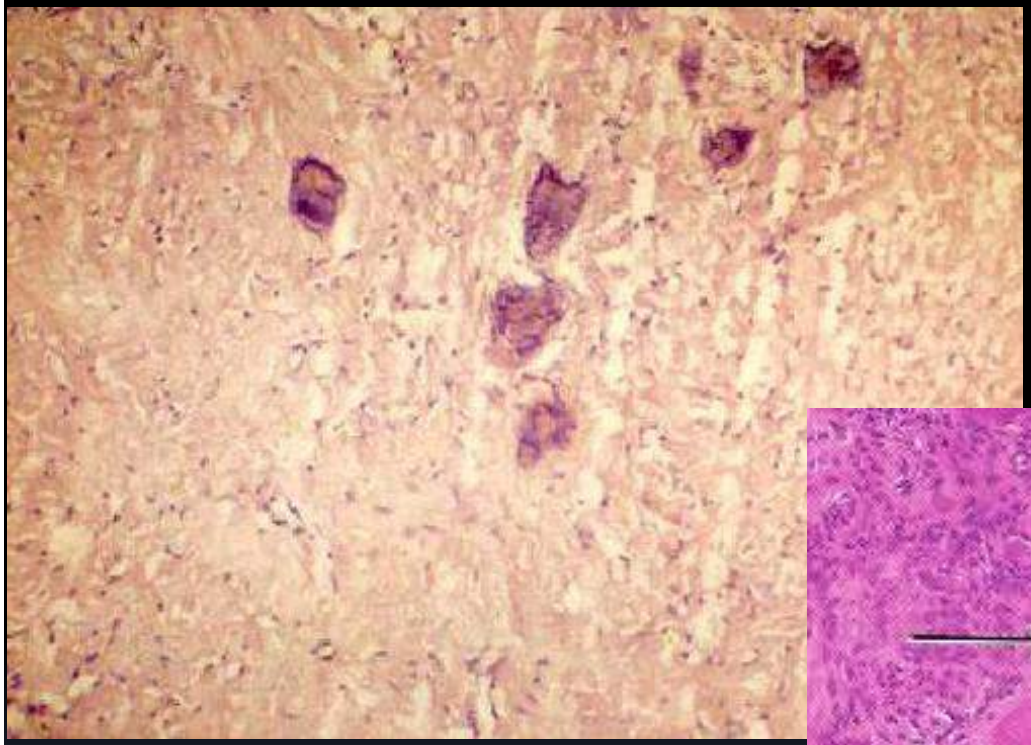
- Trabeculae of bone and droplets of cementum-like material can be seen forming within a background of cellular fibrous connective tissue
- Trabeculae of osteoid and bone OR basophilic and poorly cellular spherules that resemble cementum are seen
- Admixtures of the two types are typical
- Fibroblastic cells may show hyper chromatic nuclei
- Osteoclast-like multinucleated cells may be seen

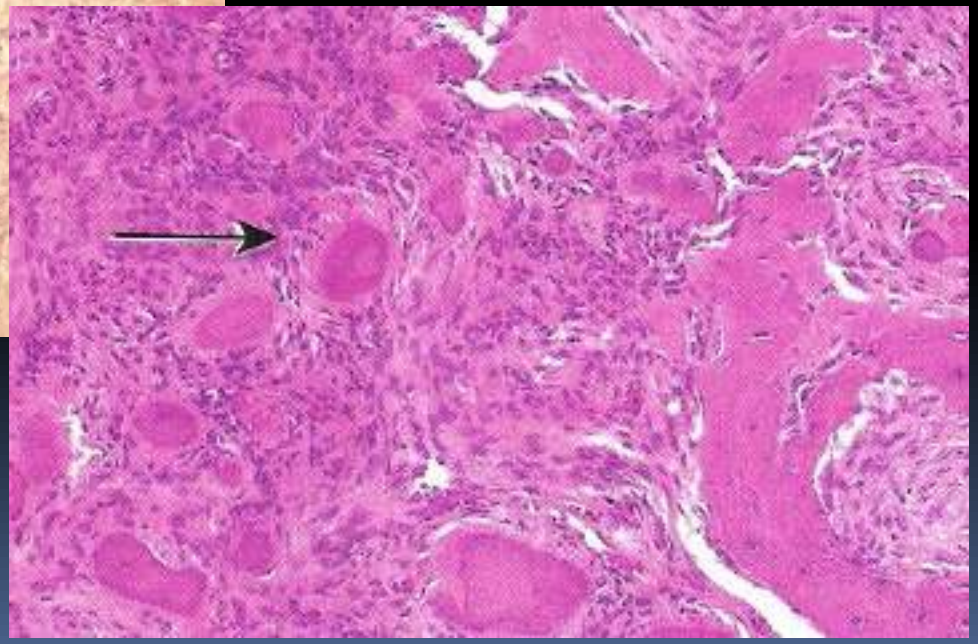
Histopathology

- Bony trabeculae with osteoid rimming
- Hyalinized stroma with dystrophic calcification

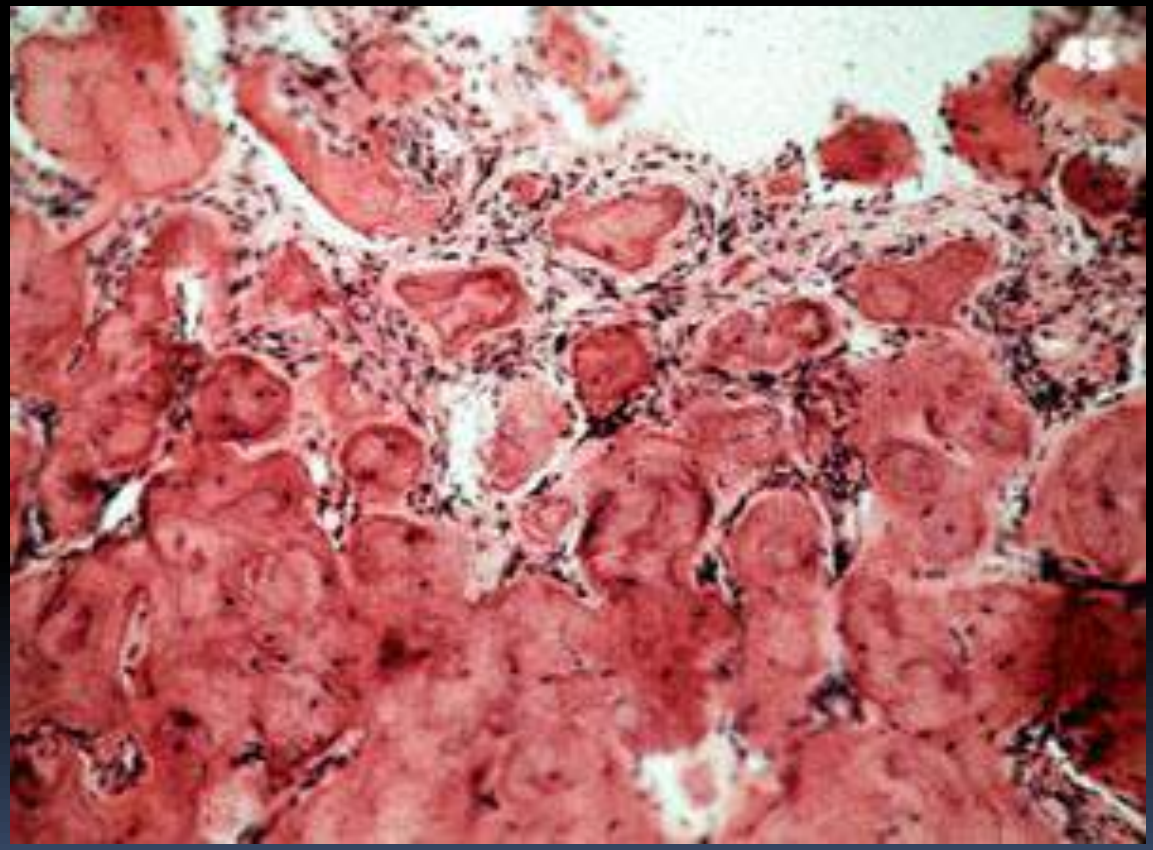


Histopathology





Histopathology



Treatment and Prognosis

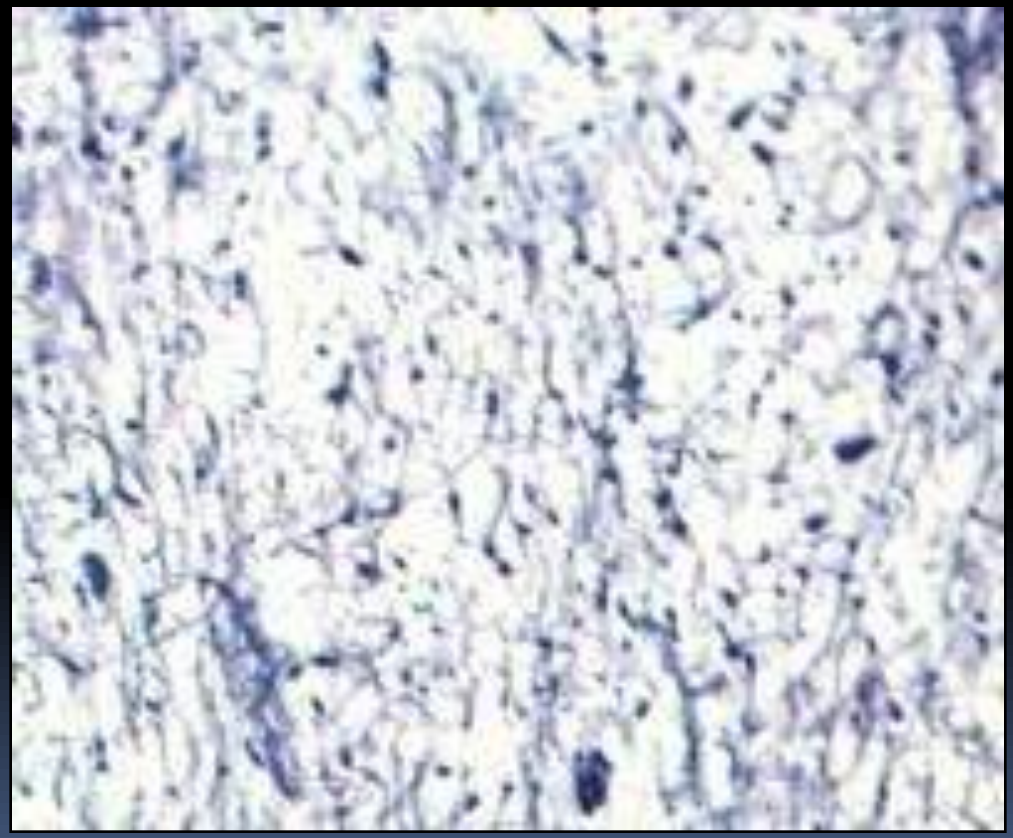
- The circumscribed nature of the ossifying fibroma generally permits enucleation of the tumor with relative ease
- Untreated tumors of massive size may require surgical (enbloc) resection and bone grafting
- prognosis is very good
- Recurrence after removal of the tumor is rare
- No evidence of COFs undergoing malignant change

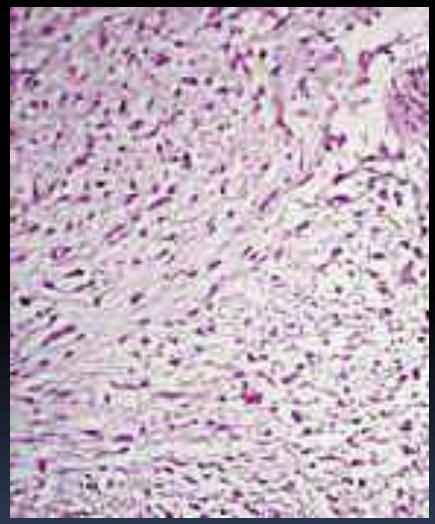
Myxoma

- This is a heterogeneous group of soft tissue tumors which have a common histologic appearance of abundent myxoid ground substances
- Commonly composed of mucopolysacchrides and hyalurinic acids
- True neoplasm made up of tissue resembling primitive mesenchyme
- stellate shaped cells in loose mucoid stroma

Clinical features

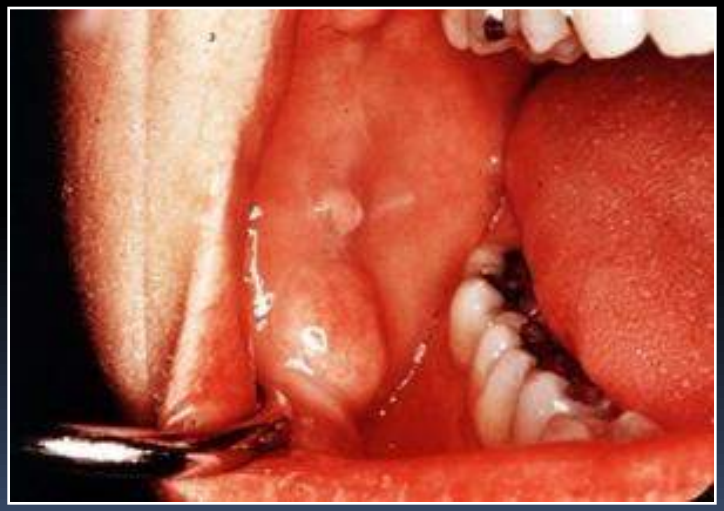
- Commonly deeply situated lesions, observed frequently in skin, sub cutaneous tissue, GU tract, liver, & salivary gland
- Any age, M=F
- O/M: rare lesion,majority of cases represents as myxomatous degeneration of fibrous tumour
- D/D: nerve sheath myxoma, oral focal mucinosis
- H/F: stellate cells in loose mucoid stroma
- Treatment: surgical, recurrence quite common





LIPOMA

- Yellow epulis
- Benign neoplasm of normal fat cells that appears as a soft, movable swelling, often with a slight yellowish coloration



Clinical types

- Diffuse
- Superficial
- Encapsulated

Clinical Features

- Well defined
- Soft on palpation
- Freely movable
- M=F
- Mostly in adults
- Arise within the superficial submucosal connective tissue
- Often yellow
- Smooth overlying surface
- Most often occurs on the buccal mucosa, ventral tongue and floor of the mouth

Differential Diagnosis:

- Fibroma
- Neuroma
- Neurofibroma
- Salivary gland neoplasms

Etiology:

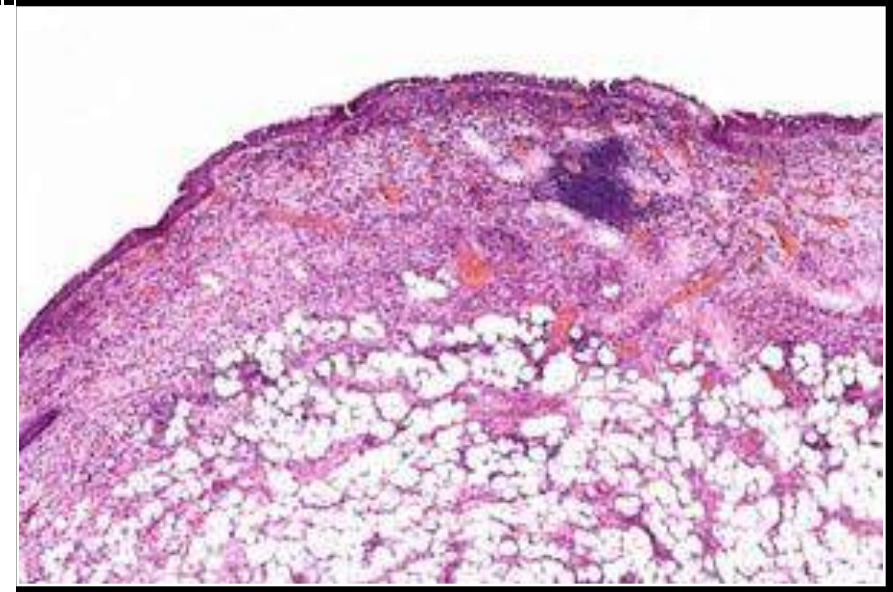
- unknown

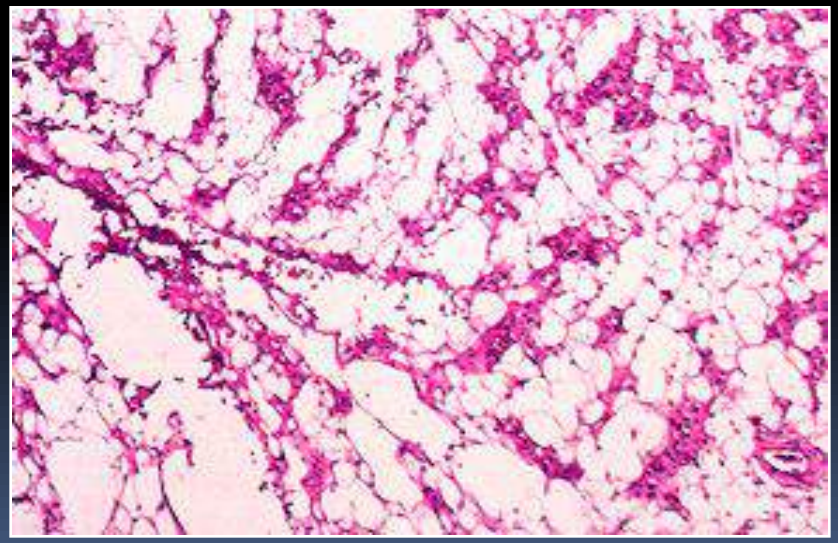
Tissue of Origin

- adipose tissue of the mucosa

Histologic Features

- Adipose tissue prolifertion
- Lesion covered by epithelium
- Clear mature adiposed cells
- Nuclei pushed to edge of cytoplasm
- May or may not have chronic inflammation in this lesion





Hemangiomas

- Vasoformative tumor
- Hamartomas
- Tumours identified by rapid endothelial cell proliferation in early infancy, followed by involution over time.

Hamartoma - Fault or defect

Benign non neoplastic tumour like malformation made up of an abnormal mixture of cells and tissues found in areas of the body where growth occurs (native to the origin).

Choriastoma -- seperated

A mass formed by mall development of tissue of a type not normally found at the site.

Eg . Ectopic Brest, ectopic liver etc

Mass of histologically normal tissue at abnormal location

Teratoma

A type of germ cell tumour that may contain several different type of tissues and sometimes mature elements such as hair,muscle and bonetumour composed of tissues not normally present at the site

1. Undifferentiated capillary network stage
2. Reteform developmental stage
3. Final developmental stage

- 1. --- Hemangioma
- 2. --- capillary or venous malformation
- 3.--- lymphatic malformation

Classification of vaso-formative tumours

- Hemangiomas
- Vascular malformation

Hemangiomas

- Capillary hemangioma
- Cavernous hemangioma
- Angioblastic hemangioma
- Racemose hemangioma
- Diffuse systemiic hemangioma
- Metastasizing hemangioma
- Nevus vinosus or portwine stain
- Hereditary hemorrhagic telangectasia

Vascular malformation

- Venous
- Capillary
- Arterio-venous
- Lymphatic malformation
  lymphangiomas

Hemangioma

- Manifest with in first month of life
- Involutes over time

Vascular malformation

- Manifest in early childhood/later
- Stable, does not regress

- **Clinical features:**
- Early childhood
- Female predilection
- Usually located in skin or mucosal tissue; may also occur in bone or muscle
- Lips is a common site
- a soft, moderately well circumscribed, painless mass which is red or blue in coloration
- Usually less than 2 cm.

- Adult-onset lesions usually occur on lip mucosa, buccal mucosa or lateral border of the tongue
- May be part of a syndrome

1. Rendu-osler-weber syndrome
2. Sturge-weber syndrome
3. Kasabach-merritt syndrome
4. Maffucci syndrome

- **Differential Diagnosis:**

- ***soft tissue lesion***

1. arteriovenous malformation

- ***intraosseous lesions***

1. ameloblastoma
2. giant cell lesion

Etiology:

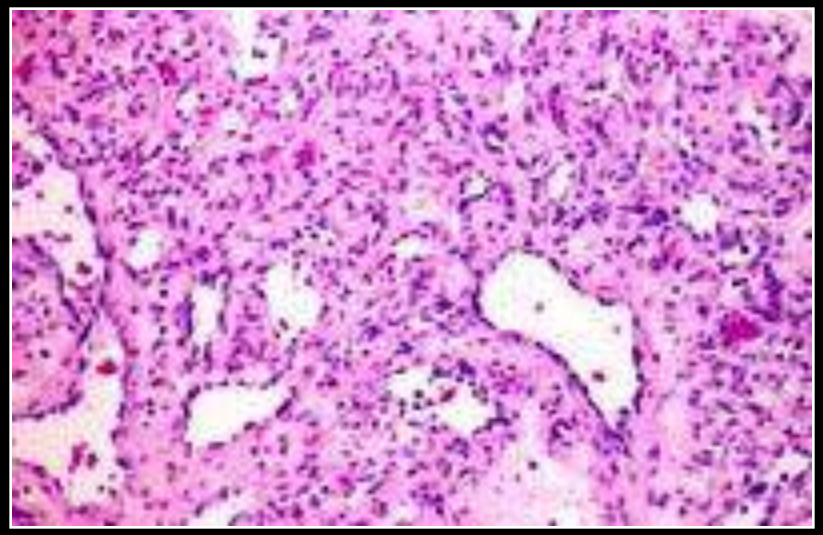
- Hamartomas
- Trauma
- Factors of a syndrome

Histologic features:

- Proliferation of blood vessels

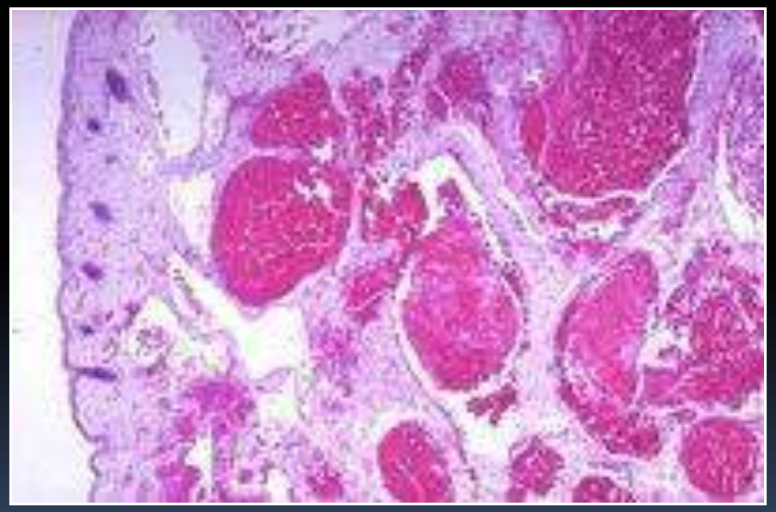
- **Capillary hemangioma**
- Multiple, small capillary channels

- **Cavernous hemangioma**
- Large tortuous dilated vascular spaces densely packed with erythrocytes



capillary hemangioma

cavernous hemangioma



- **Treatment:**
- may be left untreated until puberty
- surgical excision for functional or cosmetic reasons
- sclerosing agents
- laser therapy
- cryosurgery
- strangulation of the feeder vessel

- **Prognosis:**
- good
- capillary hemangiomas usually will regress or disappear

Lymphangioma

- Benign proliferation of lymphatic vessels that occurs as a focal superficial lesion within the oral cavity and as a massive diffuse lesion of the neck (cystic hygroma).

- **Clinical Features:**
- mucous membrane; tongue, cheek most common
- raised, diffuse, bubbly nodules or vesicles
- no gender predilection
- evident at birth or early childhood
- range in color from clear to pink, dark red, brown or black
- asymptomatic
- soft, fluctuant
- varies in size
- usually painless

- **Differential Diagnosis:**
- hemangioma
- angiosarcoma
- Kaposi sarcoma

- **Etiology:**
- Hamartomas

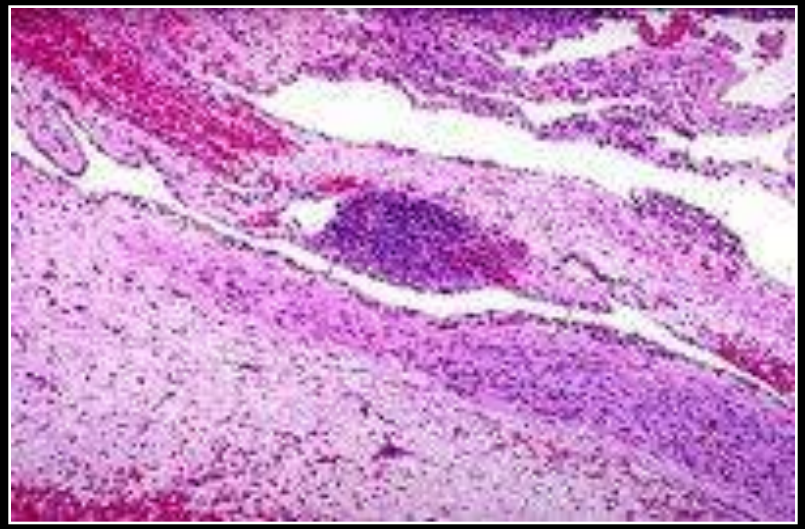
- **Histologic Features:**
- multiple, intertwining lymph vessels in a loose fibrovascular stroma
- lymphatic vessels are lined by a single layer of endothelial cells
- no encapsulation

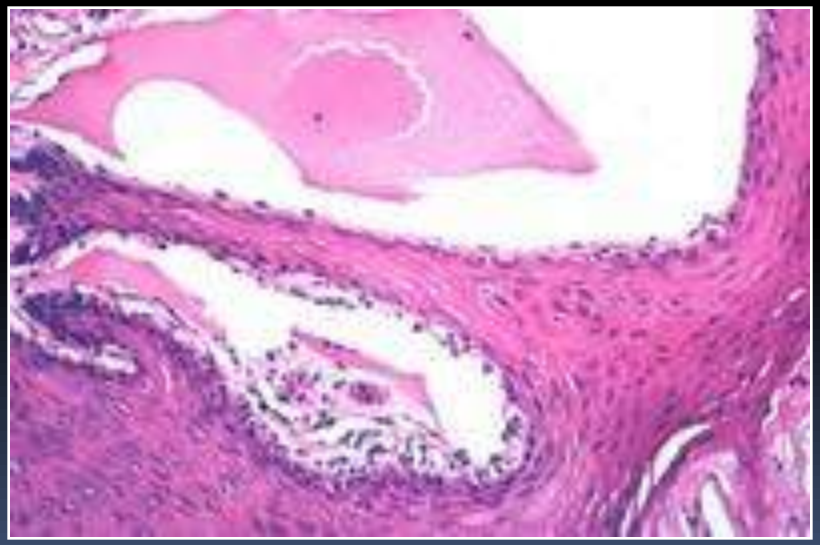
types:

- lymphangioma simplex (capillary lymphangioma) - composed of small, thin-walled lymphatics
- cavernous lymphangioma - comprised of dilated lymphatic vessels with surrounding adventitia

- cystic lymphangioma (cystic hygroma) - consisting of huge, macroscopic lymphatic spaces with surrounding fibrovascular tissues and smooth muscle

- benign lymphangioendothelioma (acquired progressive lymphangioma) - lymphatic channels appear to be dissecting through dense collagenic bundles





- **Treatment:**
- surgical excision

- **Prognosis:**
- good
- may recur

Sturge-Weber syndrome

- Encephalo-trigeminal hemangiomatosis
- Uncommon, congenital condition
- Hamartomas affecting eyes, skin, CNS

Etiology

- Somatic Mutation--- overproduction of angiogenic factors

- Primary defect is developmental insult to precursors of mesencephalic neural crests resulting in malformations in meninges,eye,skin..

- Clinical features:
- Facial cutaneous capillary venous angioma
- Area of 5^{th} CN
- Intracranial calcifications
- Eye: glaucoma, exopthalmus, angioma of choroid.
- Neurology: convulsions, spastic hemiplegia, mental retardation.

- Angiomatous lesions may involve gingivae, buccal mucosa.

Chondroma

- Benign tumour composed of mature cartilage
- Uncommon in jaw bones, common in other long bones

Clinical features

- Any age,
- M=F
- Painless, slow growing tumour
- Anterior maxilla, near the midline
- Nasal septum and nasal spine
- Root resorption, and root divergence

R/F --- irregular radiolucency

H/F – masses of hyaline cartilage with areas of calcification

Treatment --- surgical excision

Osteoma

- Benign tumour characterized by proliferation of either cancellous or compact bone, usually in an endosteal or periosteal locations

C/F— Rare,any age, M=F, slow growing painless, multiple osteomas – Gardner's syndrome

R/F – well circumscribed radiopaque mass

H/F – extremely dense compact or cancellous bone

Torus palatinus

- Slow growing, flat based bony protuberance or excrescence which occurs in the midline of the palate
- Heridetary – mendelian dominant ????

C/F – M>F, 3 rd decade, small out growth, can be flat based, spindle shaped or nodular ,overlying mucosa appears blanched

R/F – radiopaque mass

H/F – dense compact bone with a center of cancellous bone

Torus mandibularis

- Exostosis found on the lingual surface of the mandible
- C/F – children, high frequency in mangoloids, lingual side in mandible near canine - premolar, commonly bilateral

Multiple exostosis

- Exostosis found on the buccal surface of the maxilla below the mucobuccal fold in the molar region
- Overlying mucosa is intact and appears blanched

T.P
T.M
M.E

BENIGN TUMOURS OF NERVE TISSUE ORIGIN

- TRAUMATIC NEUROMA
- MEN'S SYNDROME
- NEURO FIBROMA
- NEURELEMMOMA
- MELANOTIC NEUROECTODERMAL TUMOUR OF INFANCY

TRAUMATIC NEUROMA

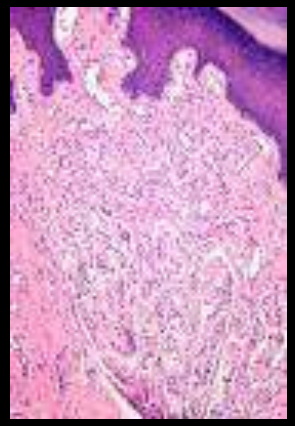
- Amputation neuroma
- Not a true neoplasm, rather an exuberant attempt at repair of damaged nerve trunk.
- Hyperplasia of nerves.

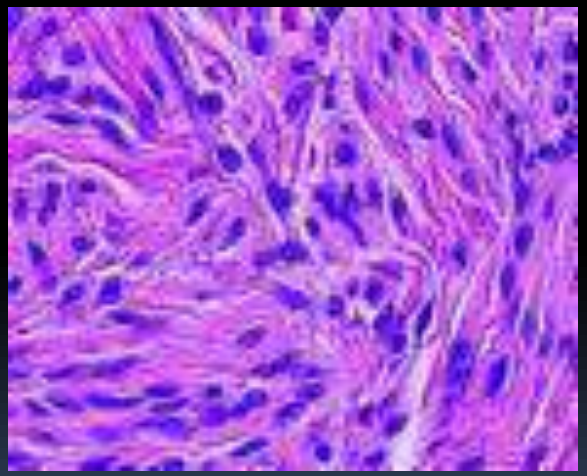
Clinical features

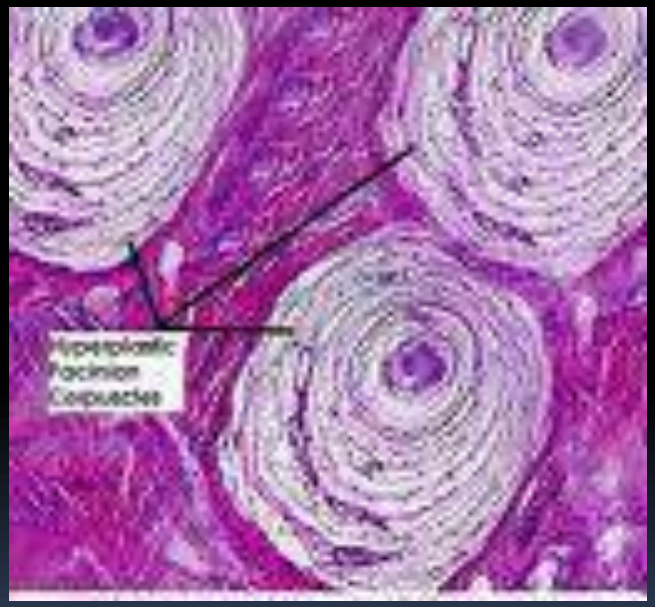
- Small nodule or swelling of the mucosa.
- Slow growing , less than a centimeter.
- Near mental foramen in the alveolar ridge, lips & tongue.
- Central lesions may also occur.
- Digital pressure may induce pain, some times along the course of the nerve. (neuralgic type of pain.)

Histological features

- Mass of irregular & often interlacing neurofibrils and schwann cells.
- Proliferation of nerve fibers, in the form of small bundles or diffused.






Hyperplastic
Pacinian
Corpuscles

Treatment

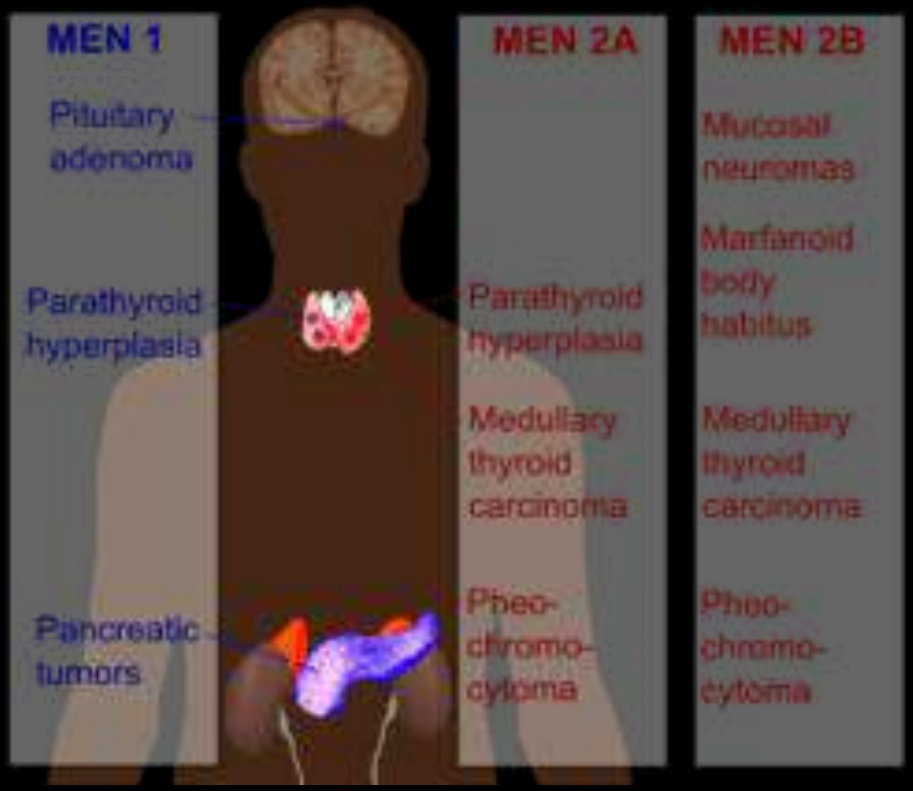
- Surgical
- Recurrence – rare.

Multiple endocrine neoplasia syndrome.

- MEN
- Adrenal pheochromacytoma, medullary thyroid carcinoma, diffuse alimentary tract ganglioneuromatosis, & multiple small submucosal neuromas.
- Autosomal dominant.

Clinical features

- Tall, lanky, marfanoid body type,narrow face, muscle wasting
- Adrenal & thyroid tumors not present until puberty.
- Oral neuroma's early adult hood.
- Painless nodules –lip ant tongue & buccal commisures
- BUMPY LIPS
- MEN TYPE I,II A, & II B


MEN 1
Pituitary adenoma
Parathyroid hyperplasia
Pancreatic tumors
MEN 2A
Parathyroid hyperplasia
Medullary thyroid carcinoma
Pheo-chromo-cytoma
MEN 2B
Mucosal neuromas
Marfanoid body habitus
Medullary thyroid carcinoma
Pheo-chromo-cytoma

Histological features

- Partially encapsulated aggregation of proliferation of nerves,often with thickned perineurium,intrwined in a plexiform pattern.
- Loose fibrous stroma.
- TREATMENT : surgical

Neurofibroma

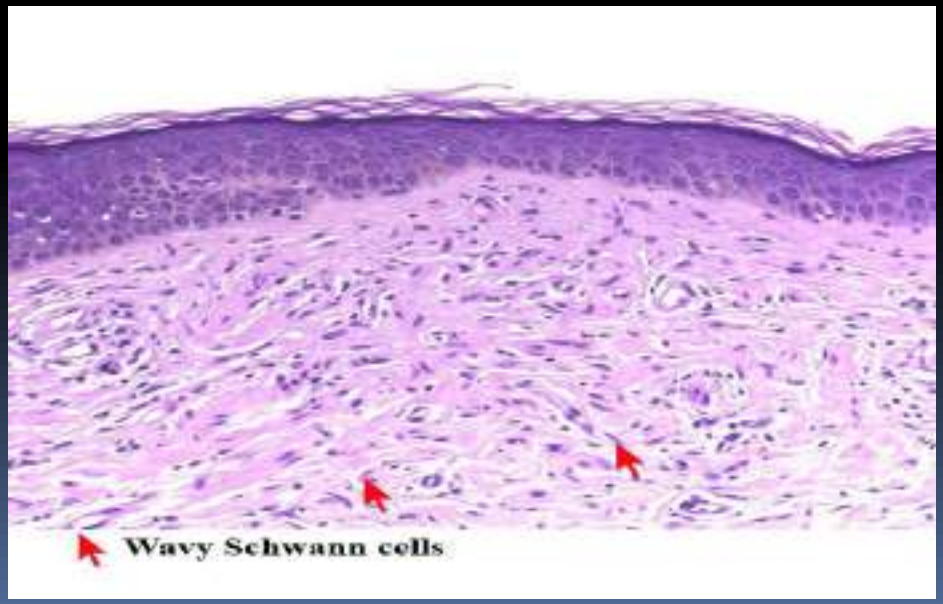
- It is a benign tumor of nerve tissue origin.
- Derived from the cells constituting the nerve sheath.
- Seen as solitary or generalized.
- Generalized – von ricklinghausan's diseases
- Perineural fibroblasts. – neuroectodermal in origin
- ETIOLOGY: un known – inherited as autosomal dominant some cases spontaneous mutation.
- Type I, & type II.

Clinical features.

- All race ,all age & M=F.
- Multiple small elivted nodules skin, mucsa, gingiva.tongue.
- Tendency of these lesions transform in to malignanacy.
- Solitory rare – malignancy.
- Osseous changes, mental defect,ocular diseases with congenital deffect.
- Intraosseous lesions are also reported.
- Associated with odontogenic fibroma.

Histological features

- Proliferation of delicate spindle cells with wavy nuclei,inermingled with each other.
- Cellular and myxoid pattern.



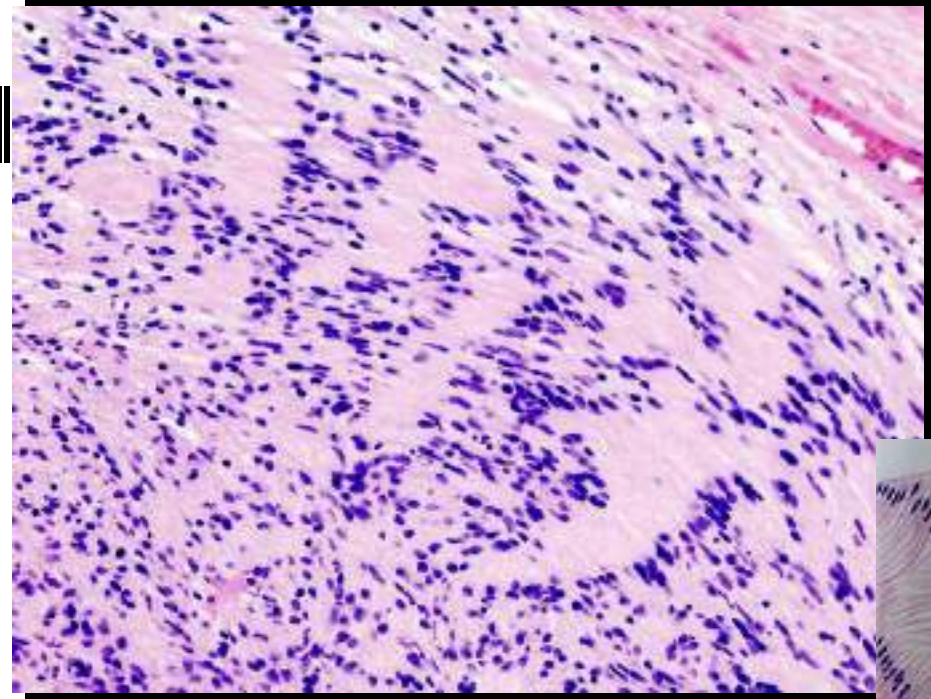
- Treatment. - surgical

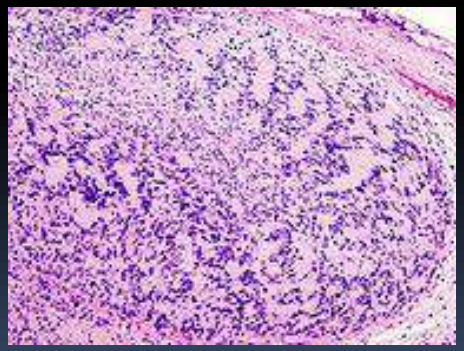
Neurilemmoma. (schwannoma)

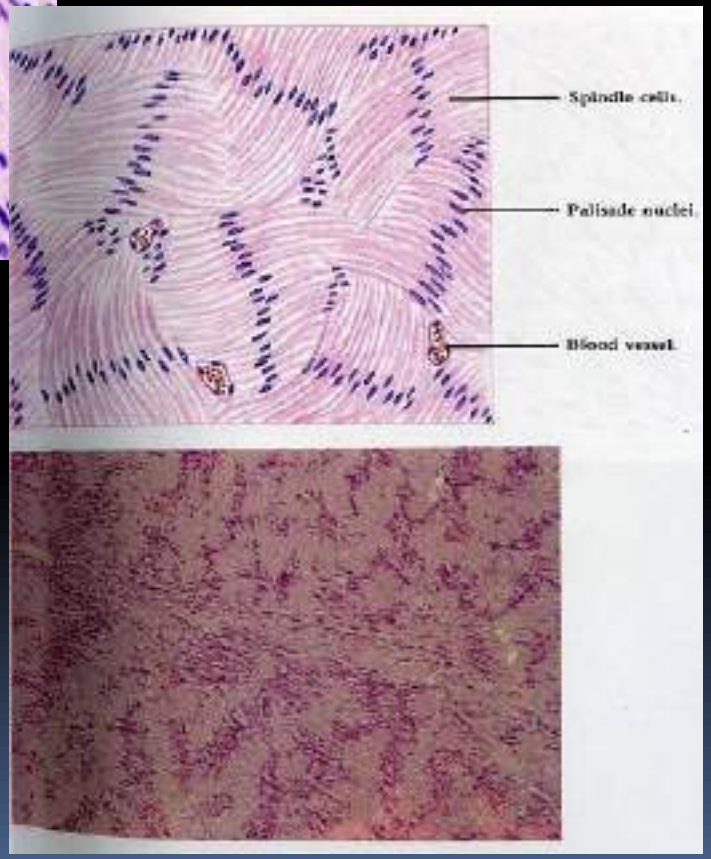
- Rather common tumor of schwann cells.
- C/F: slow growing, association with neurofibromatosis.
- At any age – First decade, M=F.
- Painless until causing pressure on adjacent tissues.
- Palate , Gingiva, lip, floor of the mouth, max sinus, salivary glands.
- Central lesions are also reported.- mandible. Solid expansion with destruction of cortical plates – more painful.

Histological features

- Composed of two types of tissue, Antony A & B
- Ant A type: cells with elongated or spindle shaped nuclei which are aligned to form characteristic pallisading pattern.
- In some plane appears in whorles or swirls.
- Ant B type: disorderly arranged cells & fibers
- Verocy bodies: small hyaline structures characteristic of the tumor.

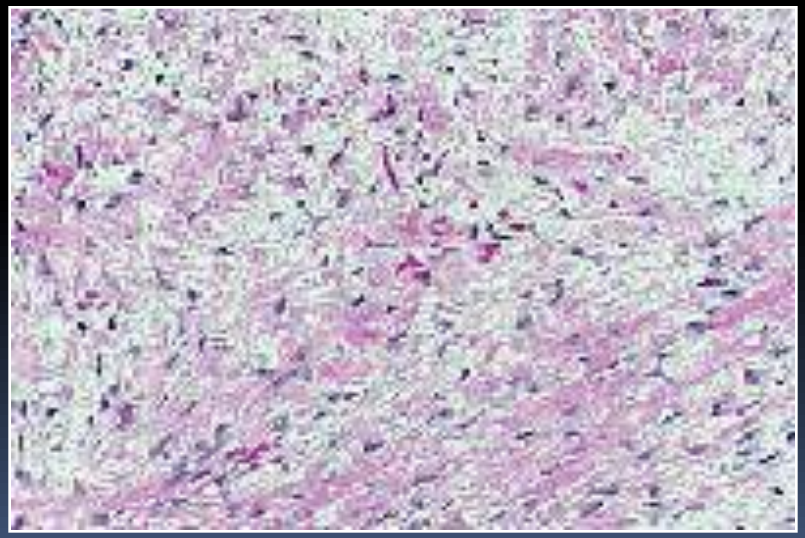


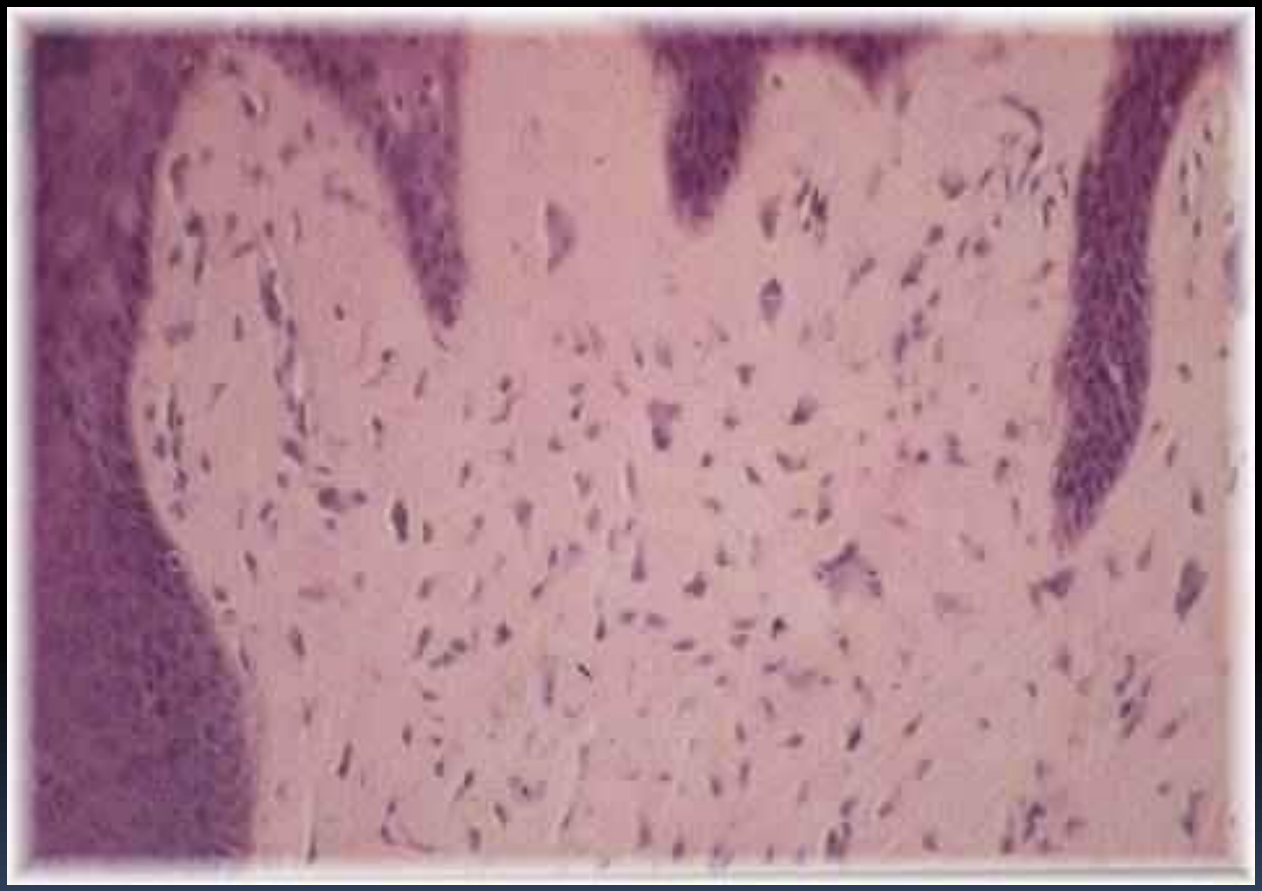


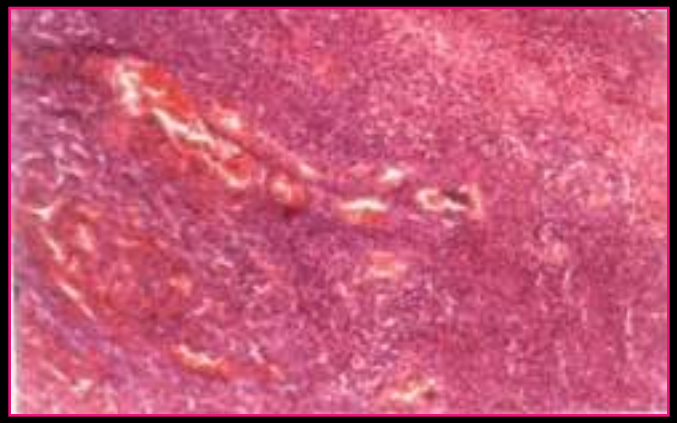

Spindle cells.
Palisade nuclei.
Blood vessel.

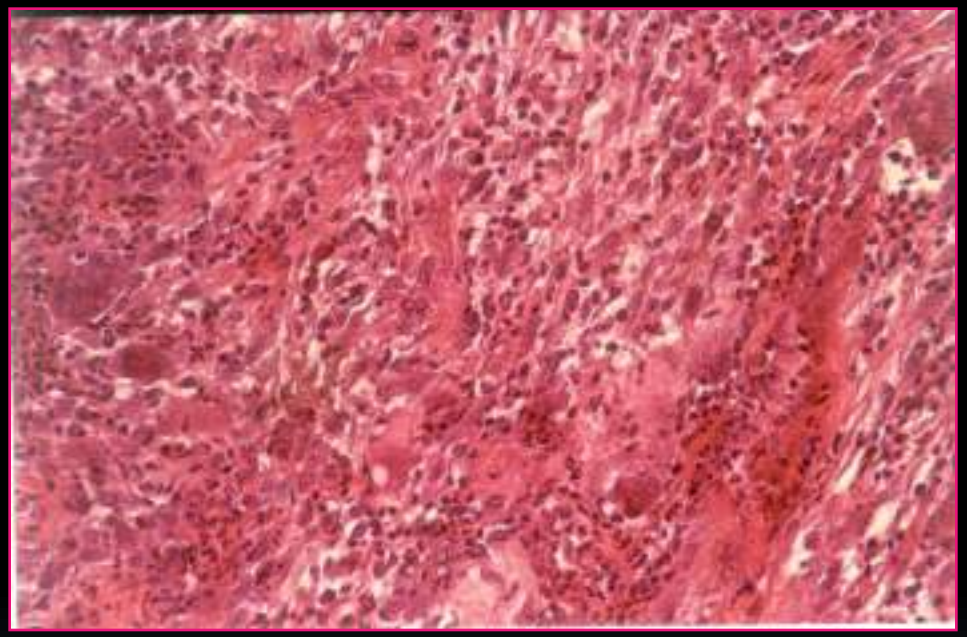
- Treatment. - surgical

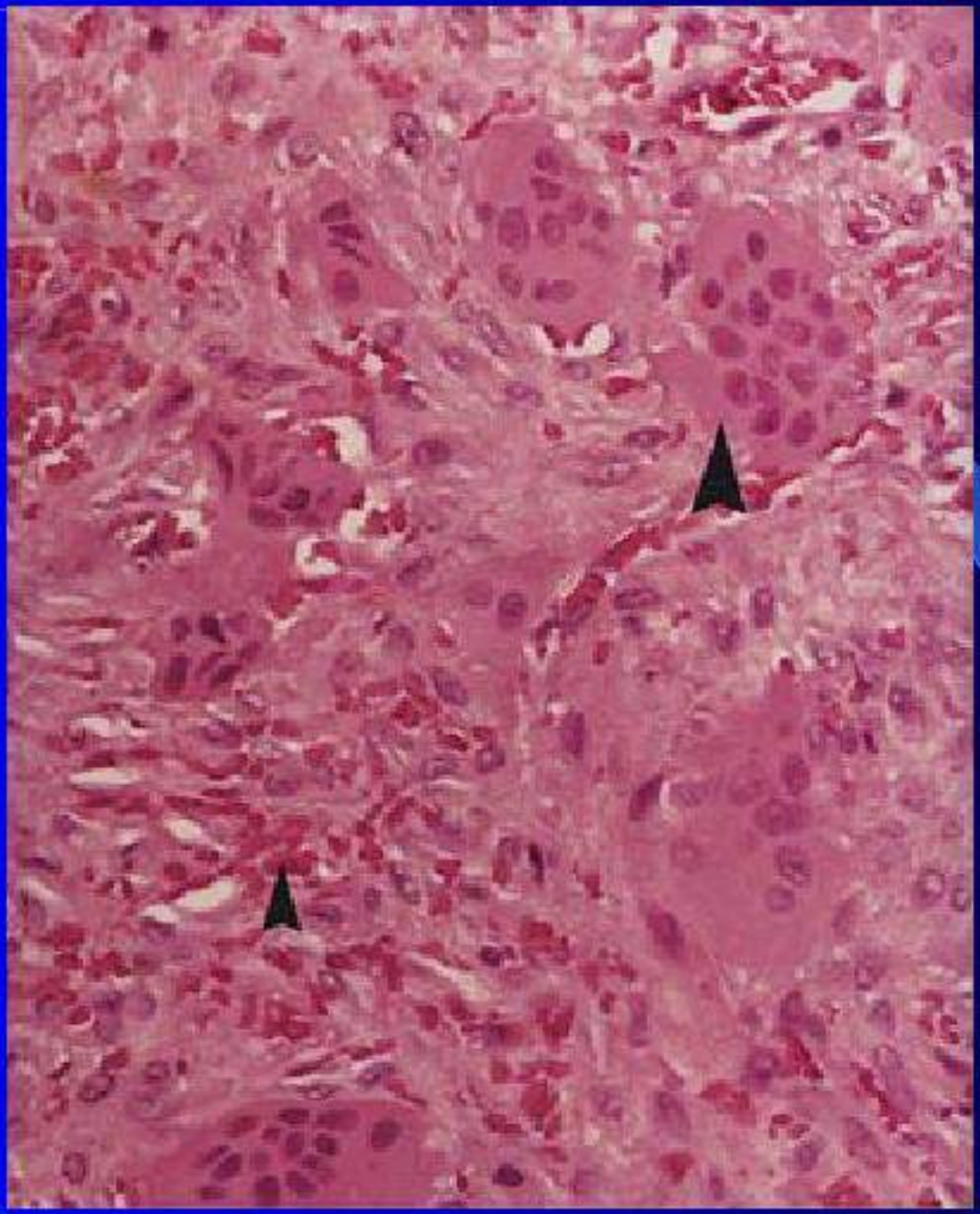
RECAP

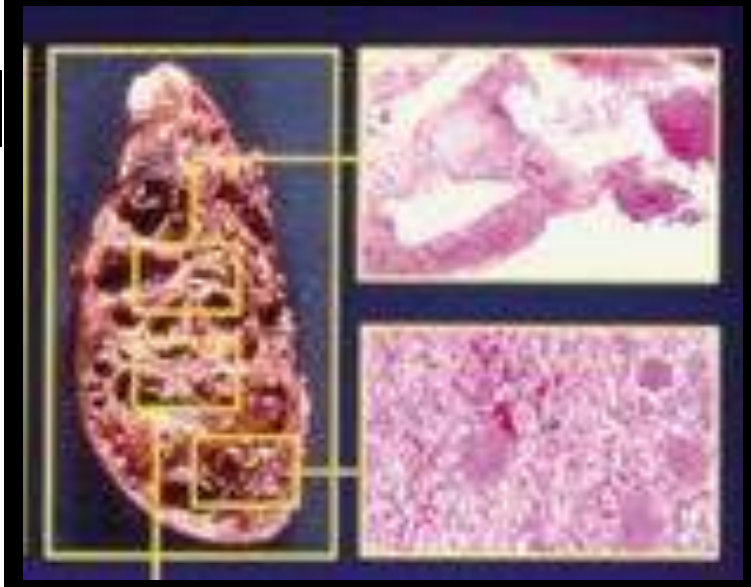


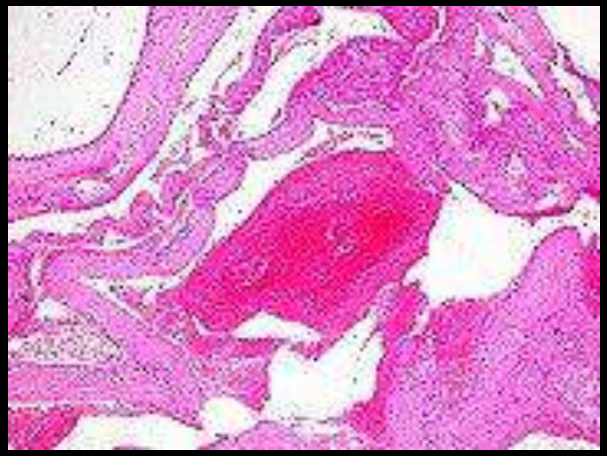


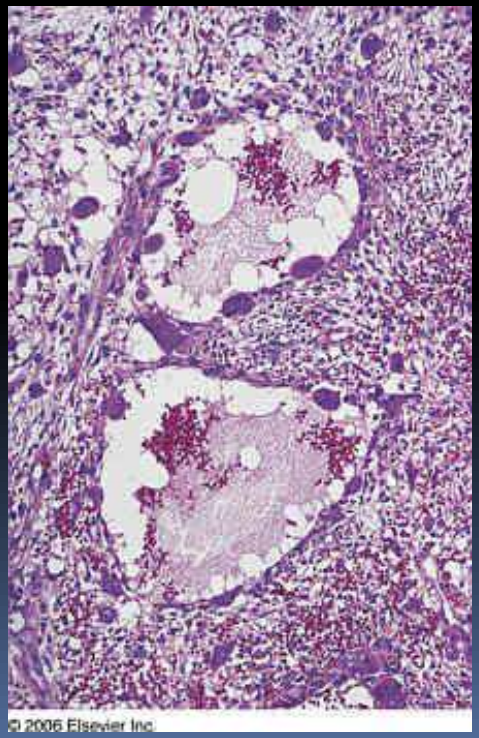








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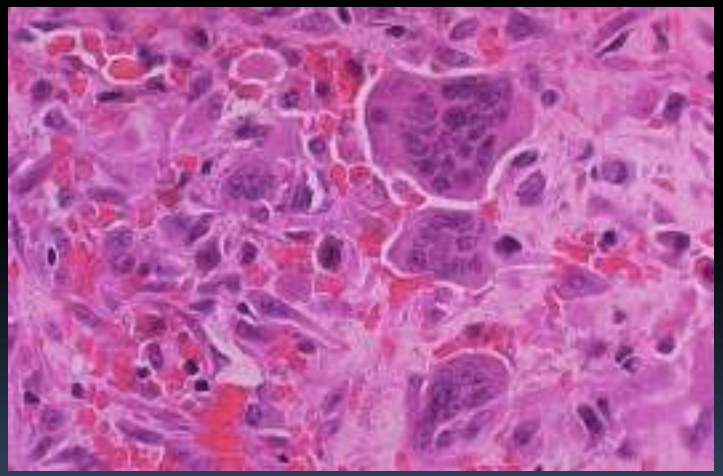
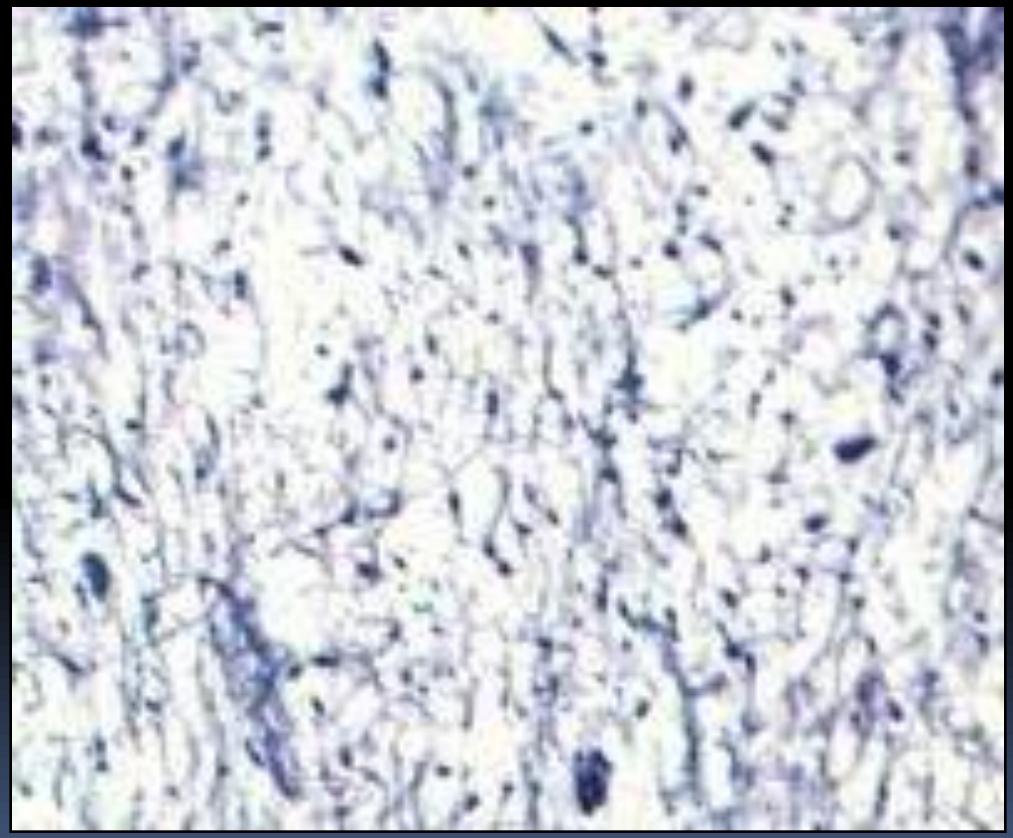
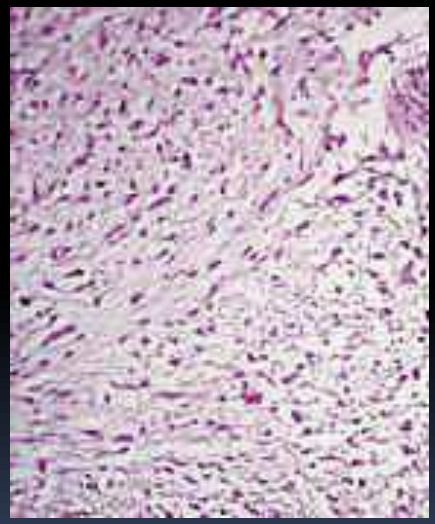
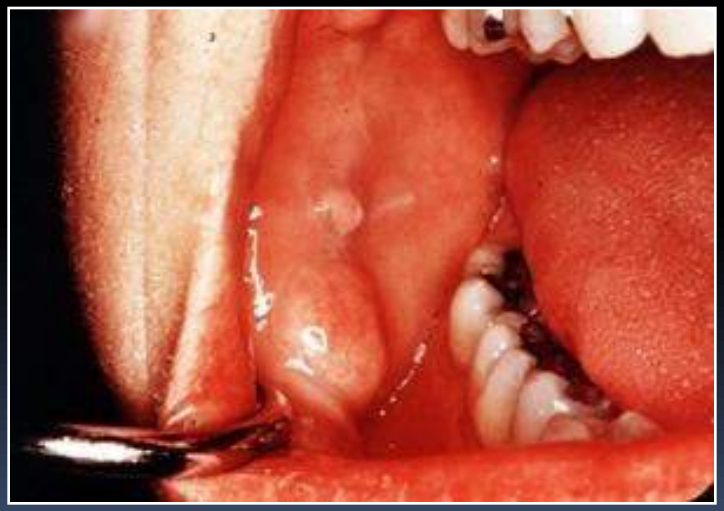
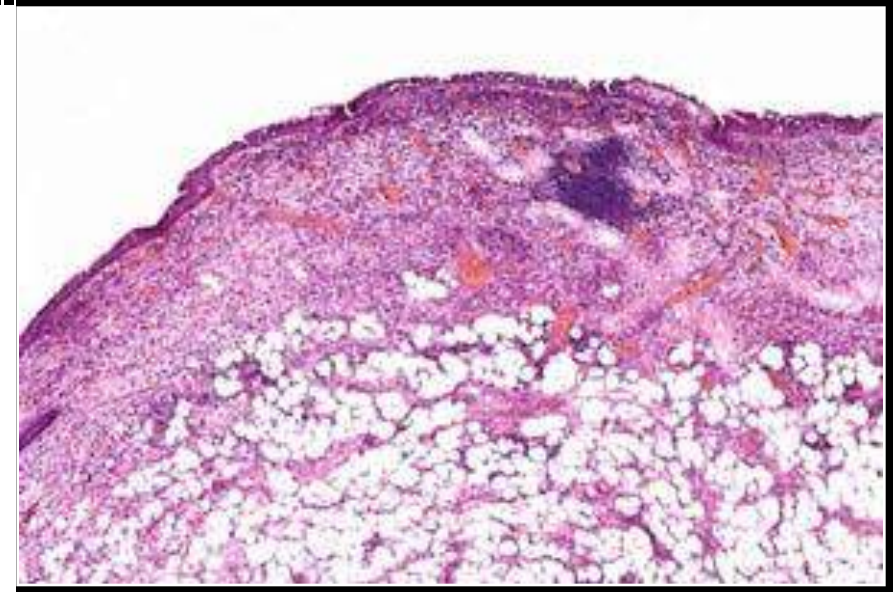
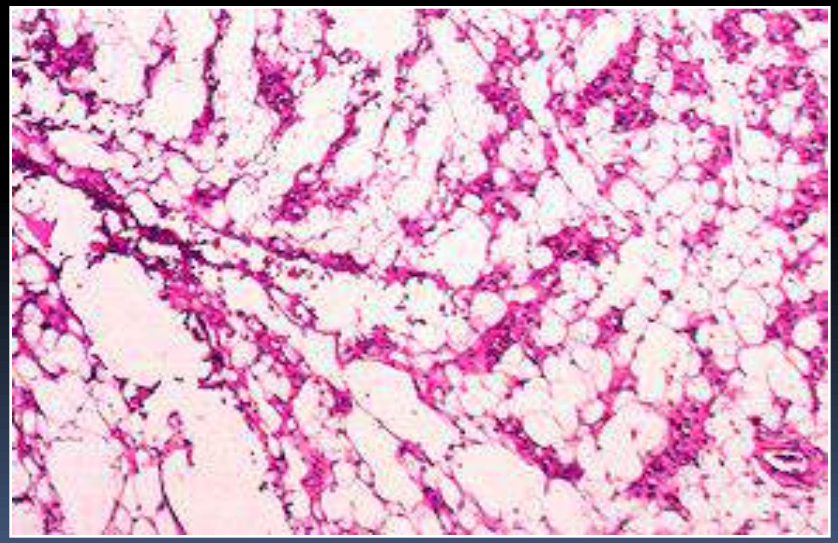


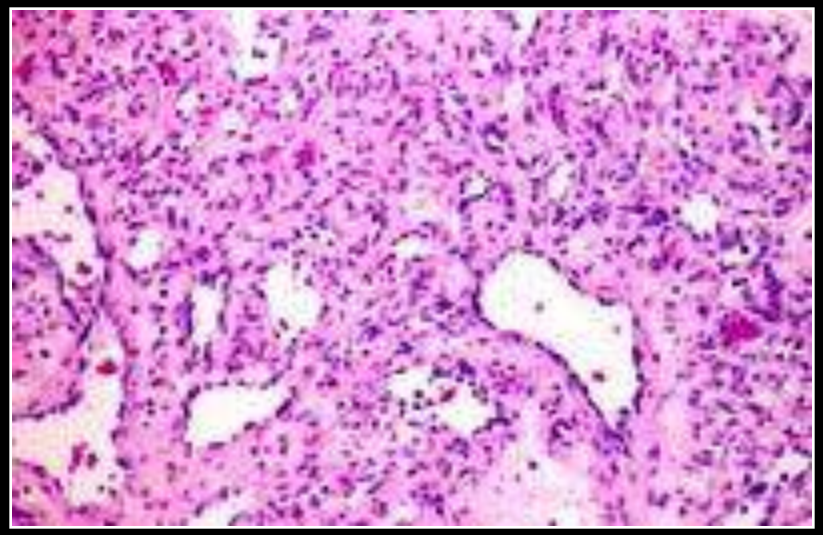


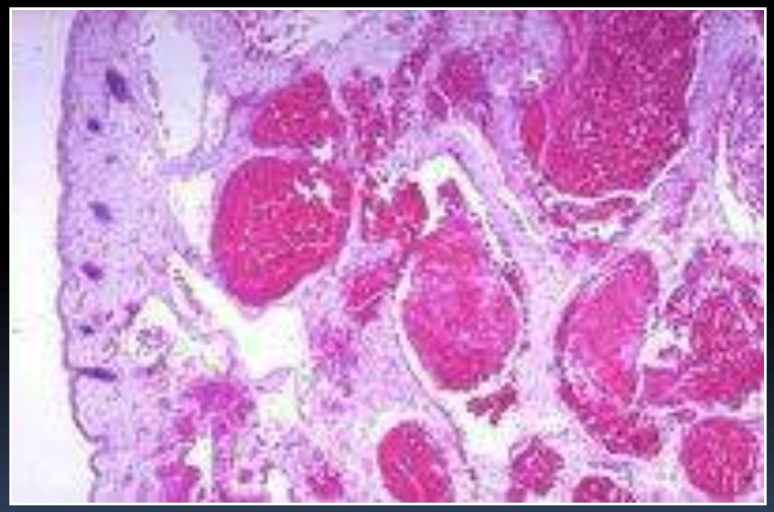


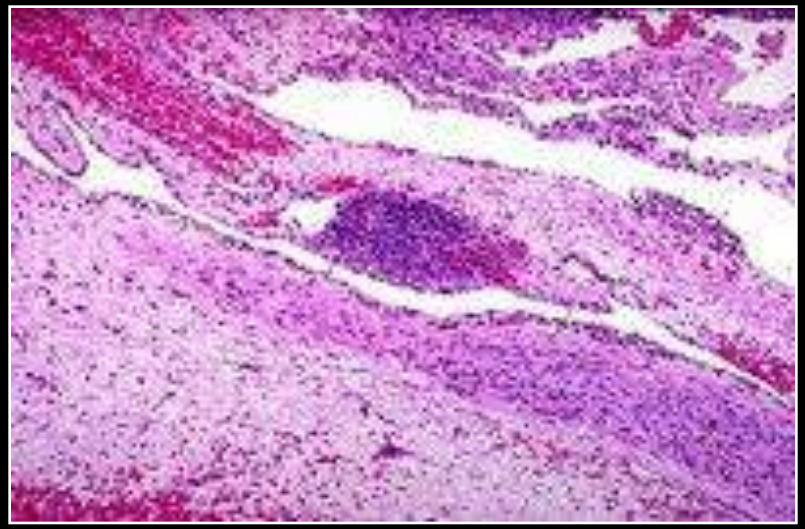


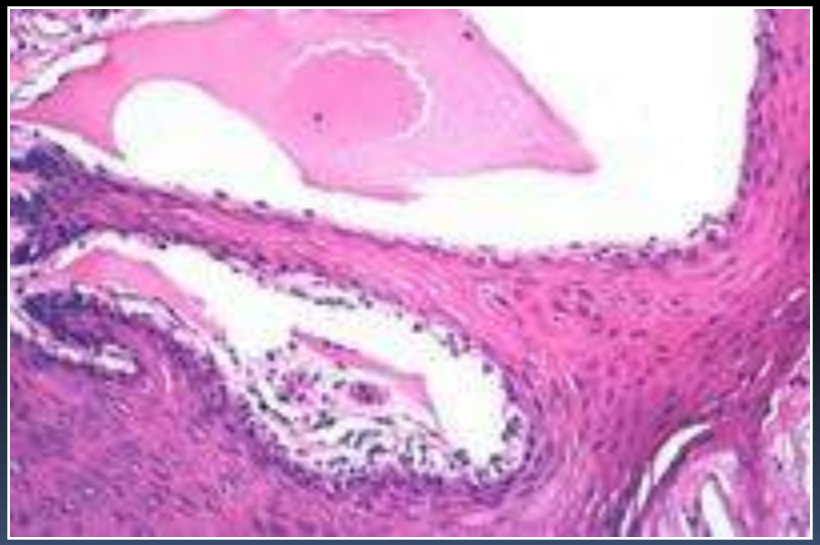


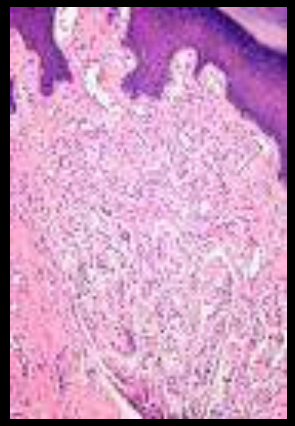


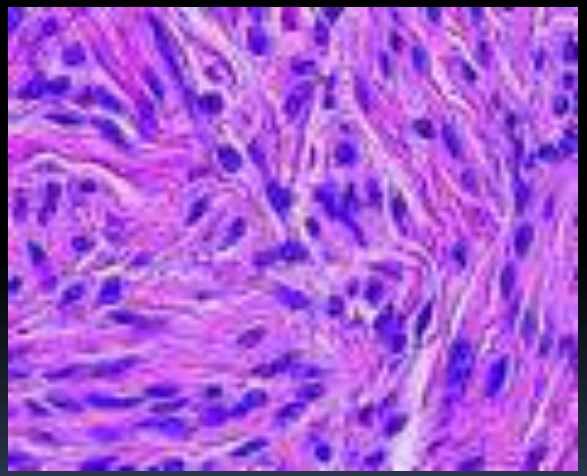


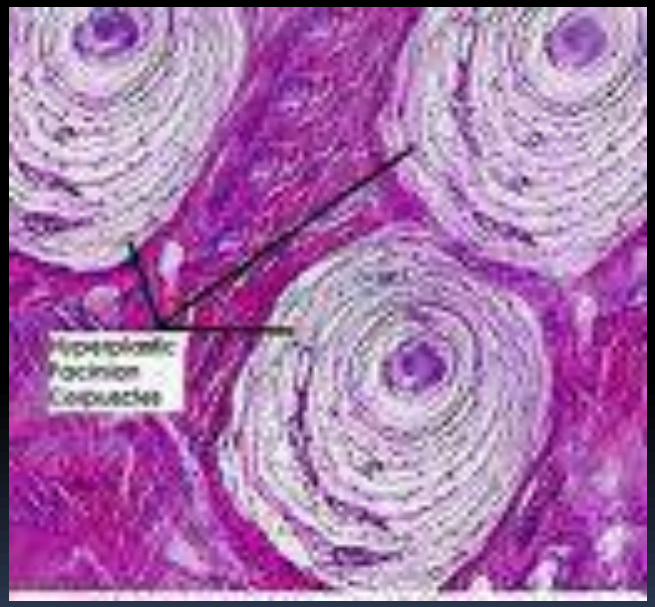


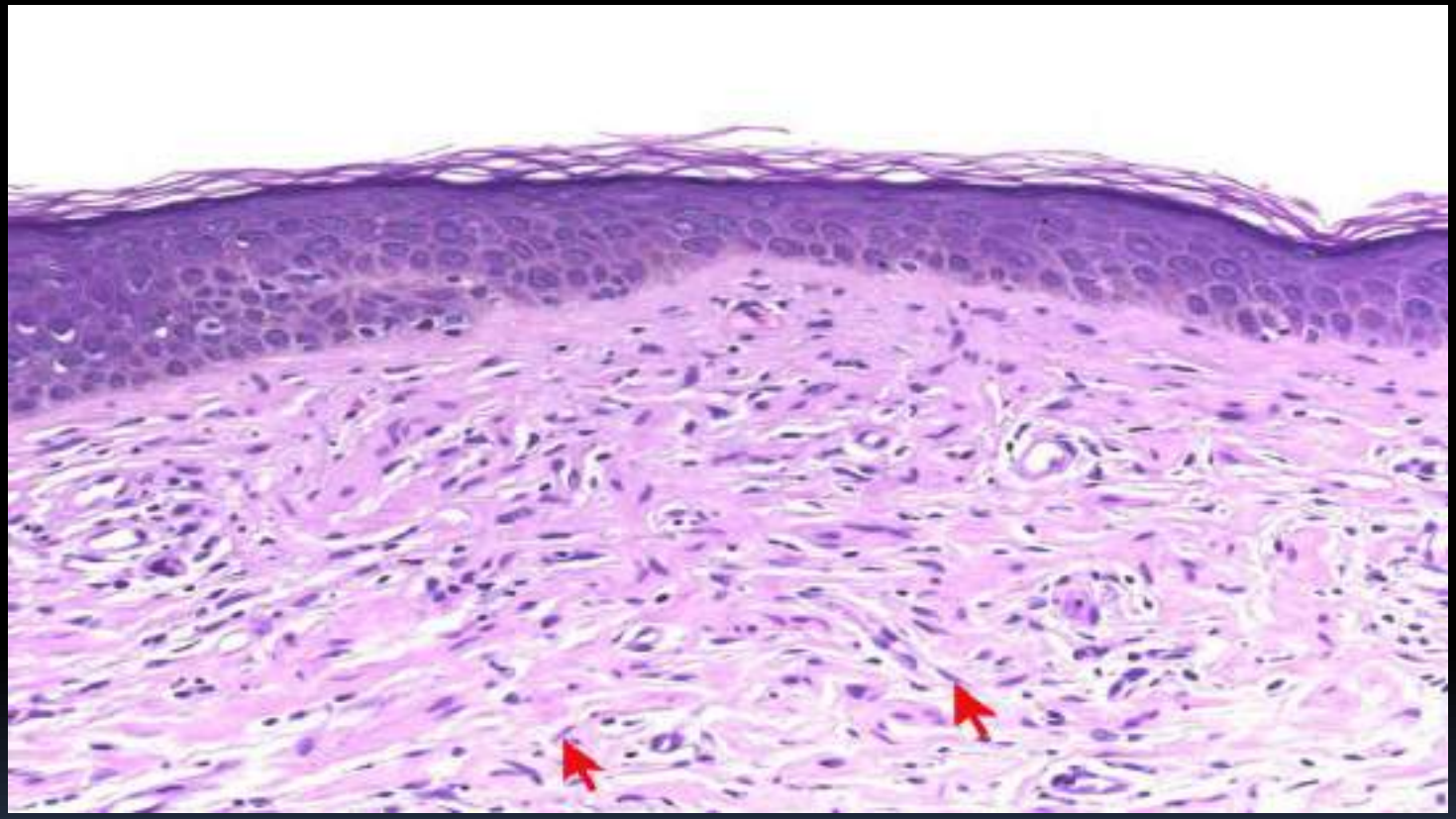


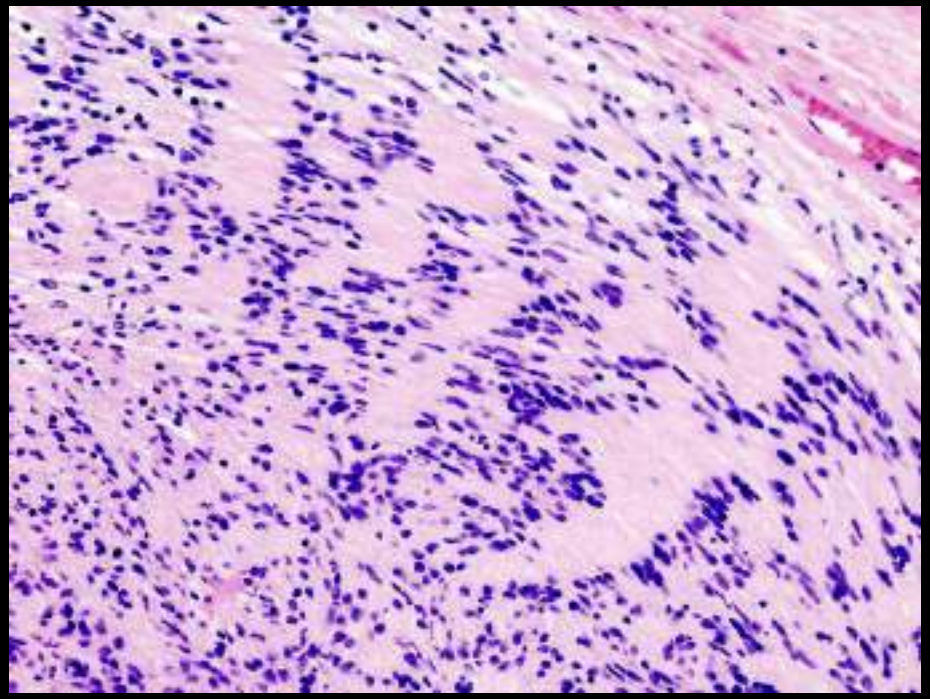


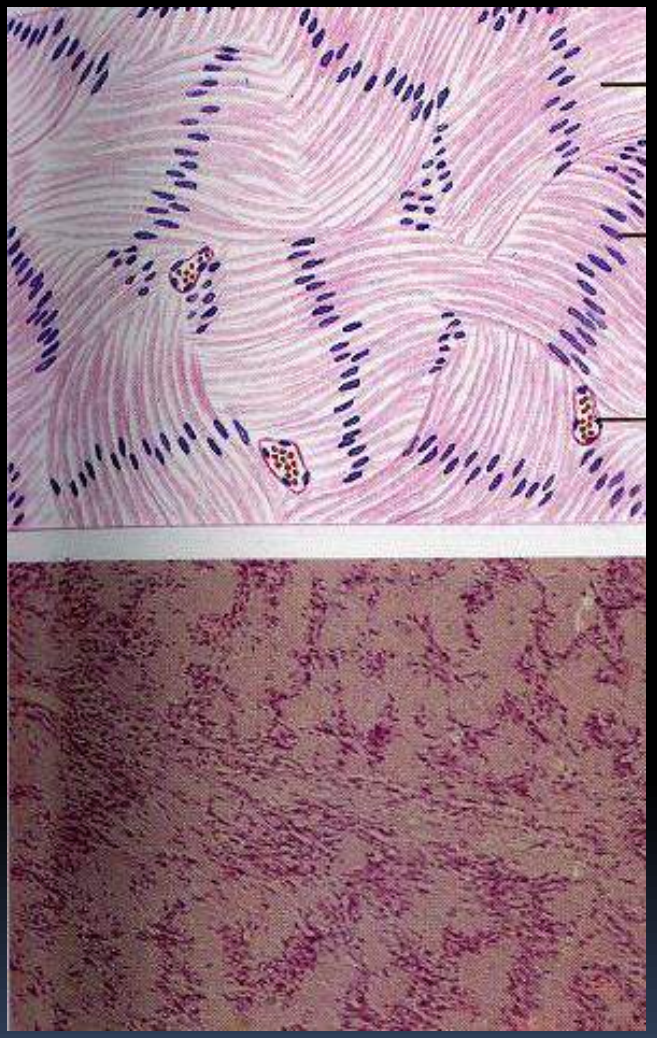


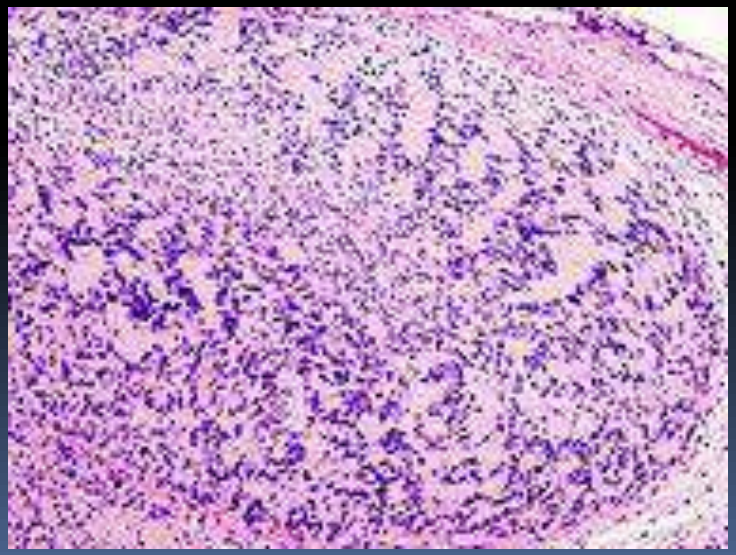



Hyperplastic
Pacinian
Corpuscles









THANK U

DERMATOLOGIC DISEASES

Classification

- Genodermatosis
- Immune mediated diseases

Genodermatosis

- Ectodermaldysplasia
- White Sponge Nevus
- Hereditary Benign Intra Epithelial Dyskeratosis
- Dyskeratosis Congenita
- Xeroderma Pigmentosum
- Darier Disease
- Peutz Jeghers Syndrome
- Hereditary Hemorrhagic Telangetasia
- Epidermolysis Bullosa
- Cowden Syndrome
- Ehlers Danlos Syndrome.

Immune Mediated Diseases

- Pemphigus
- Para- Neoplastic Pemphigus
- Cicatricial Pemphigoid
- Bullous Pemphigoid
- Erythema Multiforme
- Erythema Migranes
- Ritter's Syndrome
- Lichen Planus
- Psoriasis
- Lupus Eryhematosus
- Systemic Sclerosis
- Crest Syndrome
- Graft Versus Host Diseases

Ectodermal Dysplasia

"Group of inherited conditions in which two or more ectodermally derived anatomic structures fail to develop"

Clinical features

- Relatively rare
- Autosomal dominant, recessive, X linked
- More than 150 subtypes
- hypohidrosis, hypotrichosis and hypodontia
- M>F

Clinical features

- Soft, smooth, thin, dry skin with partial or complete absence of sweat glands
- Hyperpyrexia
- Infants: unexplained fever
- Sebaceous glands & hair follicles: absent or defective
- Hair of Scalp, Eyebrows: fine, scanty, blond
- Bridge of Nose: depressed
- Supra-orbital ridges & frontal bosses: pronounced, protuberant lips

Oral manifestations

- Anodontia or oligodontia
- Malformation of teeth
- Truncated or cone shaped
- Growth of jaw : not impaired
- Reduction: vertical dimension
- High palatal arch
- Cleft palate
- Salivary glands: xerostomia
- Lips: dry & cracked
- Mucous glands: chronic rhinitis & pharyngitis

a

b

White Sponge Nevus

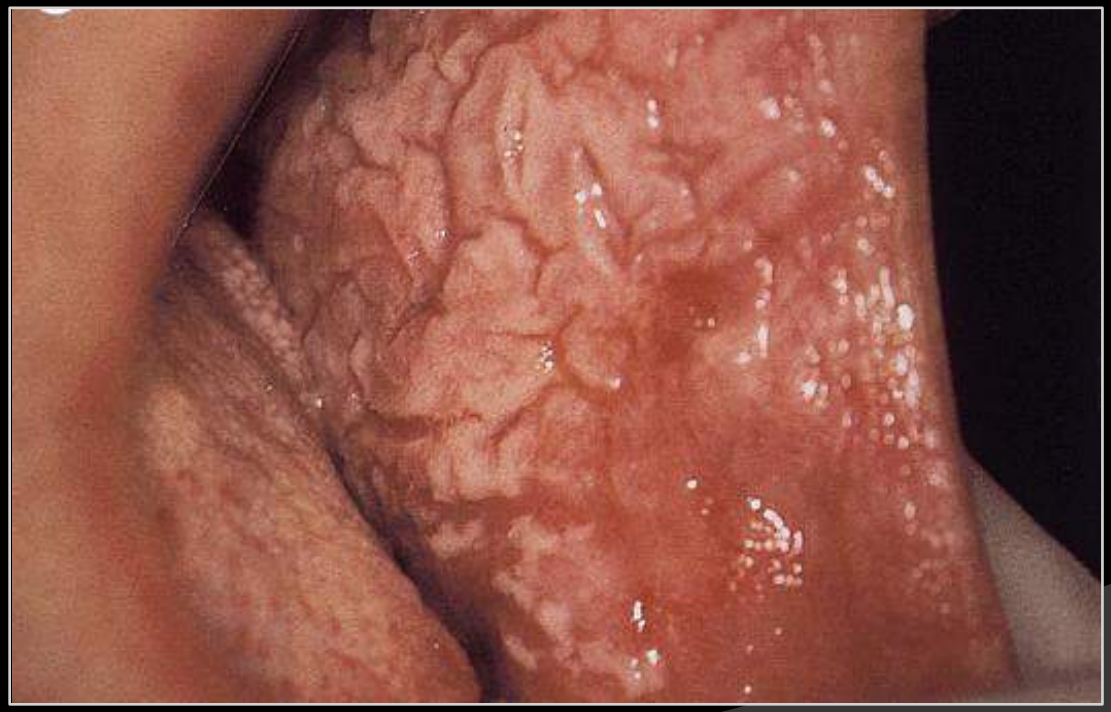
Cannon's disease

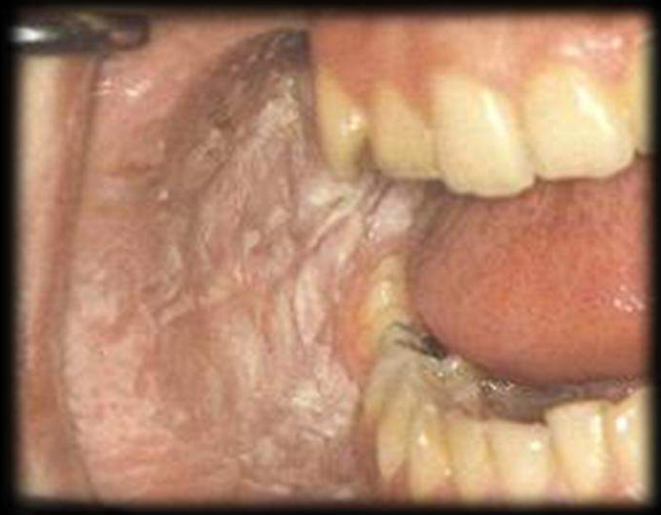
Familial white folded dysplasia

- Relatively rare genetically determined skin disorder
- Defect in the normal keratinization pattern in the oral mucosa

Clinical features

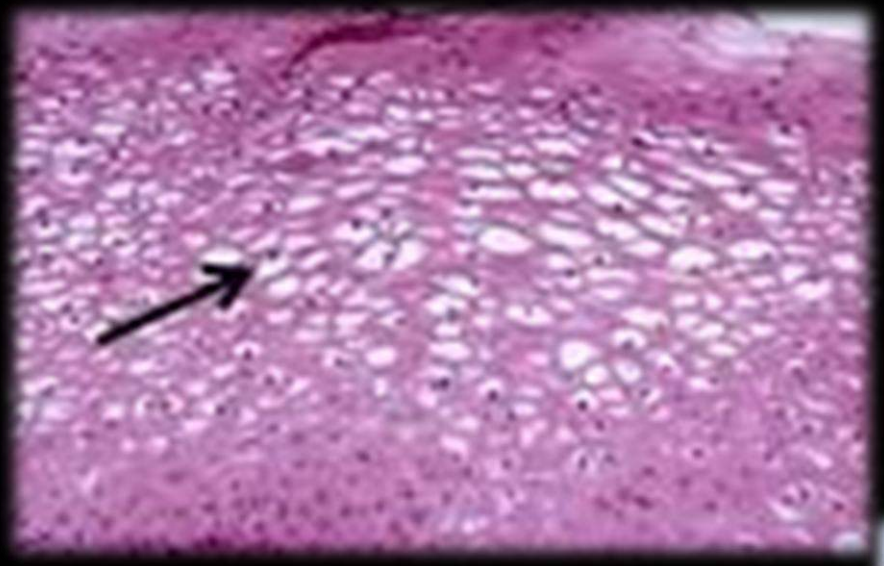
- Congenital
- Symmetric, thickened, white, corrugated or velvety diffused plaques
- Commonly Bilateral
- Buccal mucosa
- Ventral tongue, labial mucosa, soft palate, alveolar mucosa, floor of the mouth
- Asymptomatic





Histopathology

- Hyperkeratosis
- Acanthosis
- Intracellular edema: spinous layer
- Perinuclear eosinophilic condensation
- Basal layer intact
- Mild inflammatory inflitrate



Elhers - Danlos syndrome

- Group of inherited connective tissue disorders
- Abnormal collagen production
- Hypermobilty of the joints, easy bruisabilty, marked elasticity of the skin
- ‘Rubber’ man contortionist – Circus shows

Epidermolysis Bullosa

"It is a heterogeneous group of inherited blistering muco-cutaneous disorders"

Types :

Depending on the defective mechanism of cellular cohesion

I. Simplex
II. Junctional
III. Dystrophic

EB simplex

- Autosomal dominant
- Keratin gene defect
- Blistering of the hands and feet
- Mucosal involvement uncommon
- Heals without scarring
- Good prognosis

EB junctional

- Autosomal recessive
- Severe blistering at birth
- Granulation tissue around the mouth
- Pitted enamel hypoplasia
- Oral erosions
- Often fatal

EB dystrophic

- Dominant & recessive types

Dominant:

- Autosomal dominant
- Not usually life threatening
- Generalized blistering
- Vesicle- bullae – rupture – erosion/ulcerations– healing with scarring
- Extremities primarily affected
- Disfigurement
- Gingival erythema,recession, reduction in the depth of buccal vestibule

EB dystrophic

Recessive :

- Severe mucosal involvement
- Mitten like scarring
- Deformities of hand &feet
- Involvement of groin and axilla
- Fatal

Immune mediated diseases

- Erythema multiforme
- Lichen planus
- Pephigus
- Pemphigoid
- Lupus erythematosus
- Psoriasis
- Systemic sclerosis

Erythema multiforme

- It is an acute inflammatory disease of the skin & mucous membrane that causes a variety of skin lesions.
- “Multiforme”.
- Three forms –

  **Self limiting

  ** Stevens - Johnson ‘s syndrome

  ** Herpes associated EM

- Etiology:
- Immune mediated,
- Certain drugs - sulfonamides, digitalis, birth control pills, penicillin etc.
- Micro organisms – herpes & mycoplasma pneumonia.
- Radiation therapy , severe infectious diseases.

Clinical features:

- Children and young adults
- M> F
- Hands, feet, extensor surface of the elbows, knees.
- Acute onset, fever & malaise.
- Maculae or papule .5 -2 cm in diameter,
- Target or bulls eye lesions

Oral manifestations:

- 45%.
- Lips, buccal mucosa, tongue and palate
- Bullae with erythematous base - break rapidly into irregular shaped ulcers.
- Difficulty in mastication, and speech.
- Burning sensation.
- Blood tinged saliva.
- Crustation over affected area.
- Heals within two weeks.

Stevens – Johnson's syndrome

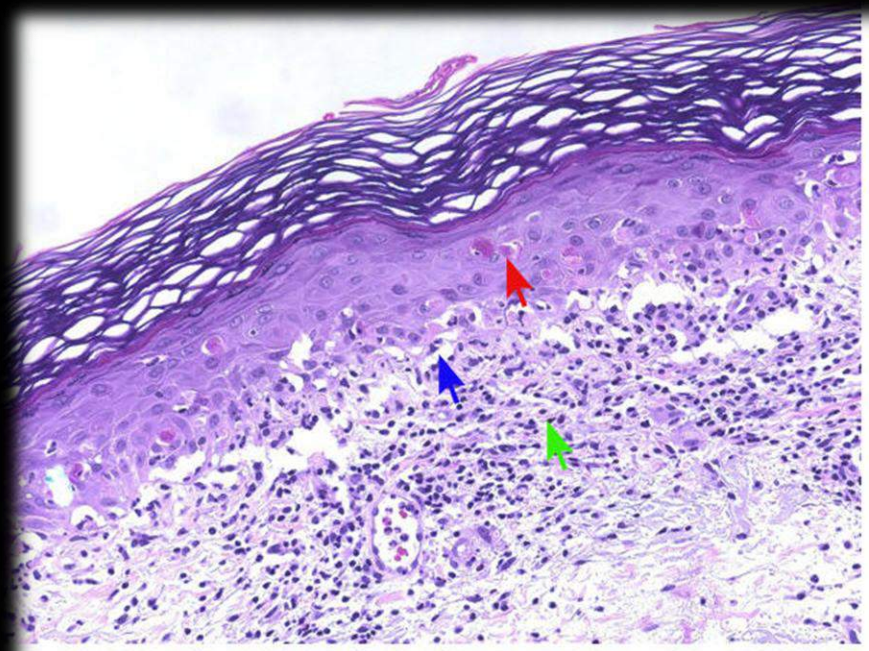
- Severe form of EM
- Wide spread involvement skin oral cavity genitalia and eyes.
- Cutaneous lesions – vesicular or bullous with hemorrhage,
- Eye lesions – conjunctivitis, corneal ulcerations.
- Genital – urethritis, balanitis, and vagina ulcers.
- Oral lesions are extensive and extremely painful.

Toxic epidermolysis necrolysis

- TEN
- Lyell's disease
- Generally occurs secondary to drug reaction and results in sloughing of skin and mucosa in large sheets.

Histopathology

- Intercellular edema in the spinous layer
- Edematous connective tissue.
- Intraepithelial vesicle formation.
- Dilated blood capillaries, peri vascular neutrophilic infiltration.
- Bacterial colonies , eosinophils.



Dead keratinocytes

Vacuolar change

Lymphocytes

LICHEN PLANUS

- Syn: LICHEN RUBER PLANUS
- Erasmus Wilson – 1869
- Common dermatologic disease that often affects oral mucosa.

CLINICAL FEATURES:

- Bilateral
- Small flat topped papules.
- Discrete or coalesce into large groups
- Sharply demarcated..
- Wickham's striae.
- F>M

- Bilaterally symmetrical.
- Flexor surfaces of wrist & forearms, inner aspect knees & thighs.
- Primary symptom: pruritus.
- Females: common.
- PATHOGENESIS:
- Autoimmune disorder.

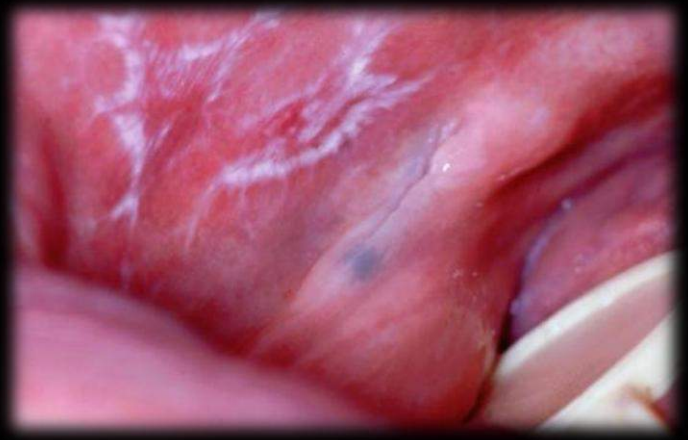
Types:

RETICULAR
EROSIVE
BULLOUS
PIGMENTED

Reticular lichen planus:

- Skin lesions appear as small, angular, flat topped papules only few mm in diameter.
- Gradually coalesce to a larger plaques
- Early lesions – red, gradually – violaceous hue.

- Posterior buccal mucosa.
- Wickham’s striae.- interlacing lines
- surface of the lesions are covered by characteristic ,very fine grayish white lines.
- Over tongue: appear as keratotic plaques with atrophy of the papillae

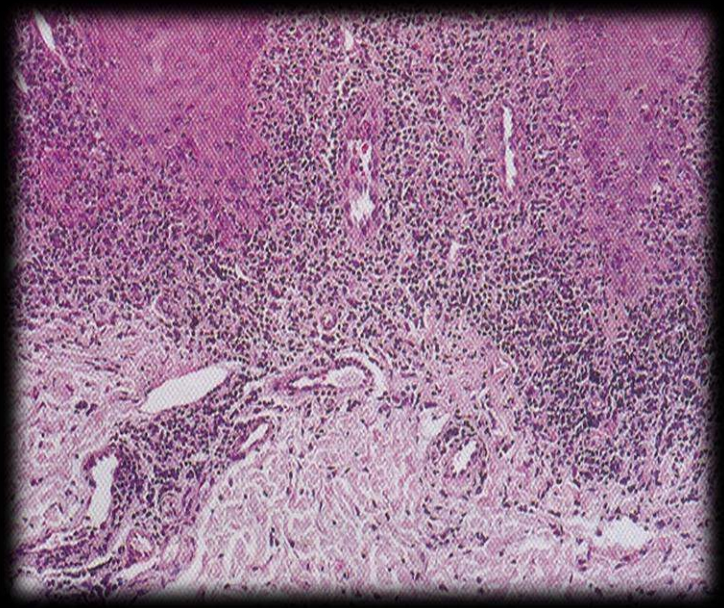


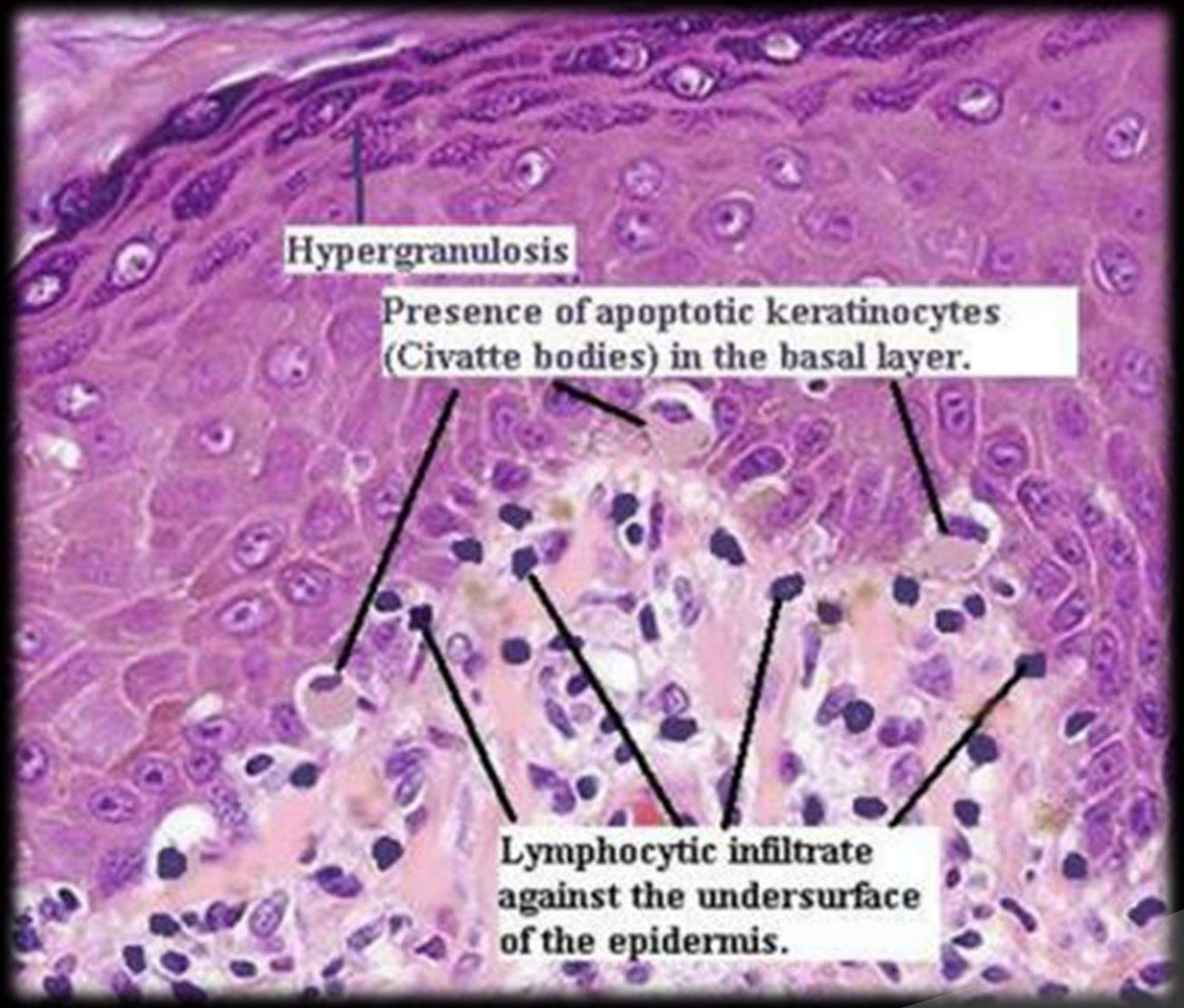
Erosive lichen planus:

- Symptomatic.
- Atrophic, erythematous areas with central ulcerations.
- Periphery: Wickham’s striae.
- Gingiva: desquamative gingivitis.

HISTOLOGICAL FEATURES

- Saw tooth shape rete pegs.
- Acanthosis.
- Hyperkeratosis or orthokeratosis.
- Intense T-lymphocyte infiltrate.
- Degenerating keratinocytes- colloid, cytoid, hyaline, or Civatte bodies.




Hypergranulosis
Presence of apoptotic keratinocytes (Civatte bodies) in the basal layer.
Lymphocytic infiltrate against the undersurface of the epidermis.

- **DIFFERENTIAL DIAGNOSIS**

- Leukoplakia
- Candidiasis
- Pemphigus
- Erythema multiforme
- Lupus erythematosus
- Cicatricial pemphigoid.

- **GRINSPAN'S SYNROME:** LICHEN PLANUS+ HYPERTENSION+ DIABETES MELLITUS.

PEMPHIGUS

- Autoimmune disorder.
- Vesiculobullous lesion.
- It is a serious chronic disease characterized by the appearance of vesicles and bullae, small or large fluid filled blisters, that develop in cycles.
- Intercellular auto-antibodies: epithelium of skin & oral mucosa.
- Circulating auto-antibodies are present in their serum.

- 4 types:

- Pemphigus vulgaris
- Pemphigus vegetans
- Pemphigus erythematosus
- Pemphigus foliaceous

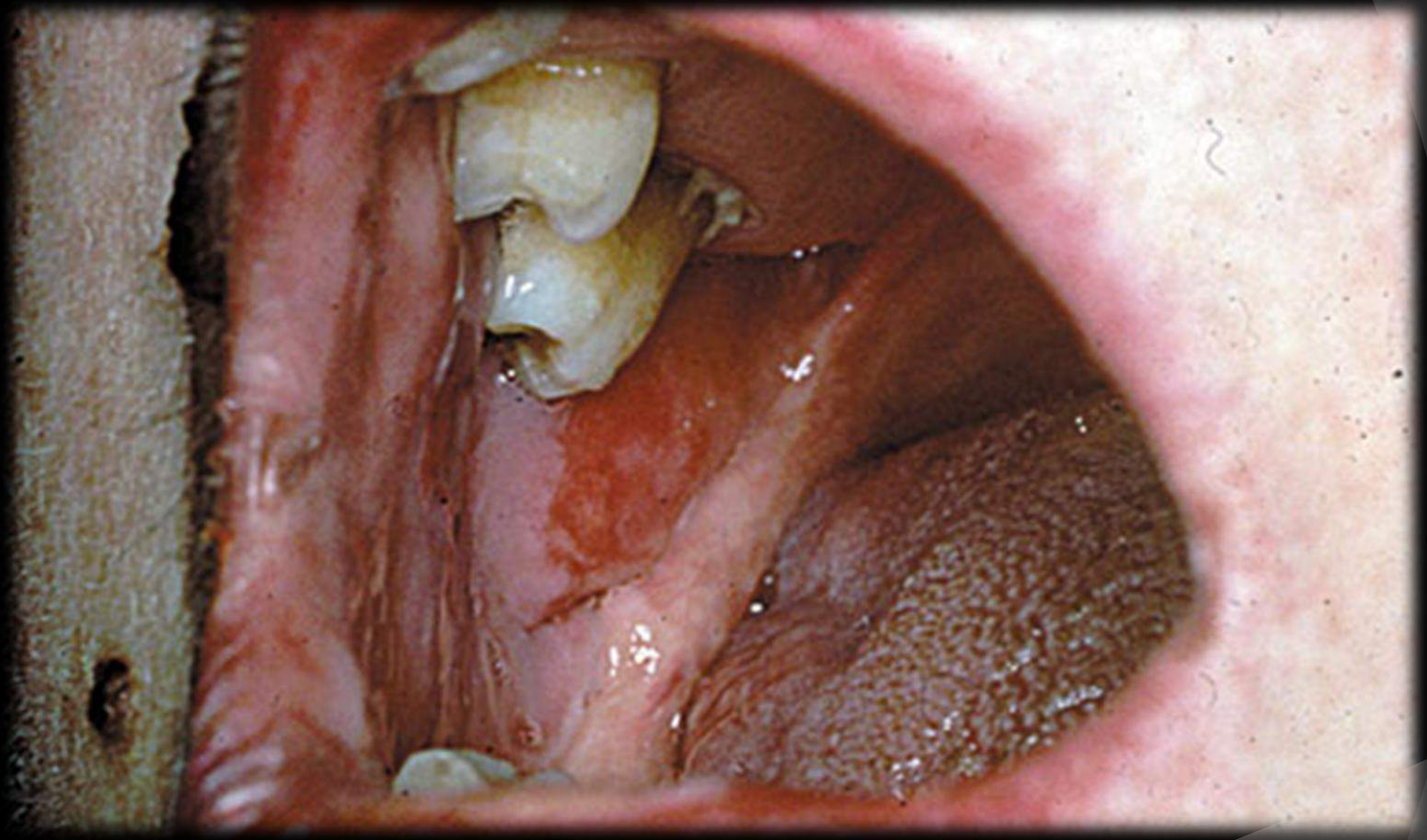
- Vulgaris & vegetans affect oral mucosa.

- ***Pathogenesis:*** Pemphigus vulgaris is due to abnormal production of autoantibodies against the epidermal cell surface glycoproteins, desmoglein 1 & desmoglein 3.

- CLINICAL FEATURES
- 50yrs.
- No sex predilection.
- Complain of soreness, ragged erosions and ulcerations.
- Palate, labial mucosa, ventral tongue, gingiva.

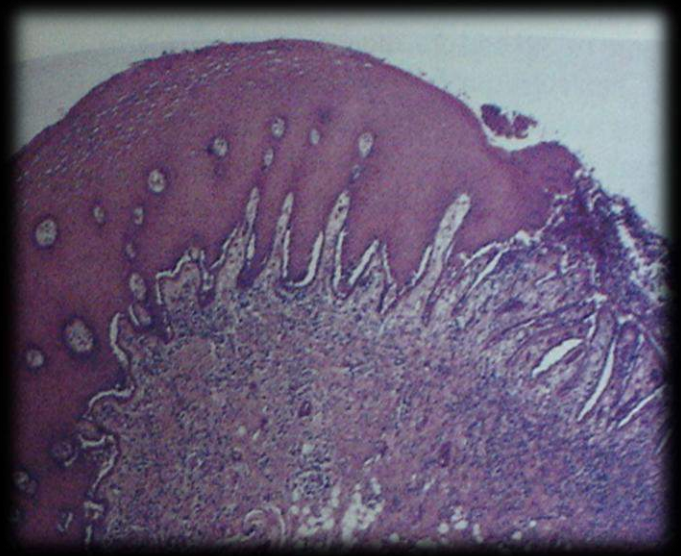
.

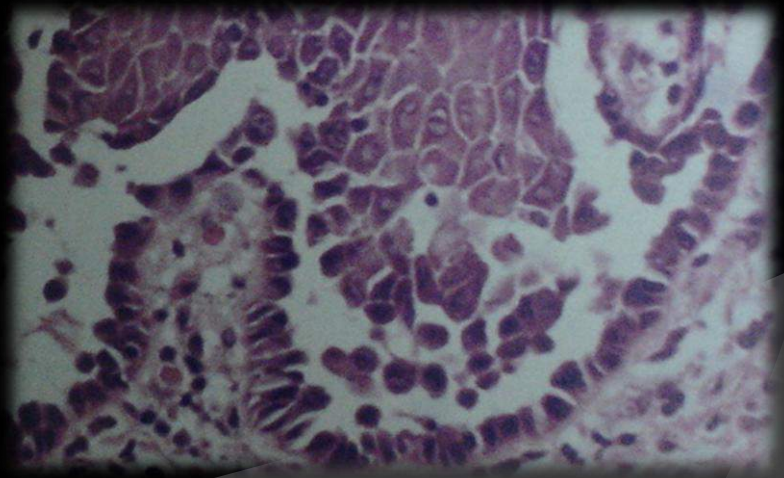
- Vesicle or bulla formation – early rupture of thin, friable roof of blisters.
- Skin lesions-flaccid vesicles & bullae leaving an erythematous, denuded surface
- Ocular involvement: bilateral conjunctivitis
- *Nikolsky sign*: Bulla is induced on normal appearing skin if firm lateral pressure is exerted.



HISTOPATHOLOGICAL FEATURES

- Intraepithelial separation.
- Superficial cells are stripped away----basal cell layer: “row of tombstones”.
- Acantholysis - round cells.
- Exfoliative cytology: round cells-Tzanck cells.
- Direct immunofluroscence-intracellular.
- Mild chronic inflammatory infiltrate in connective tissue.





PARANEOPLASTIC PEMPHIGUS

- Neoplasia induced pemphigus
- Vesiculobullous disorder: lymphoma or chronic lymphocytic leukemia.

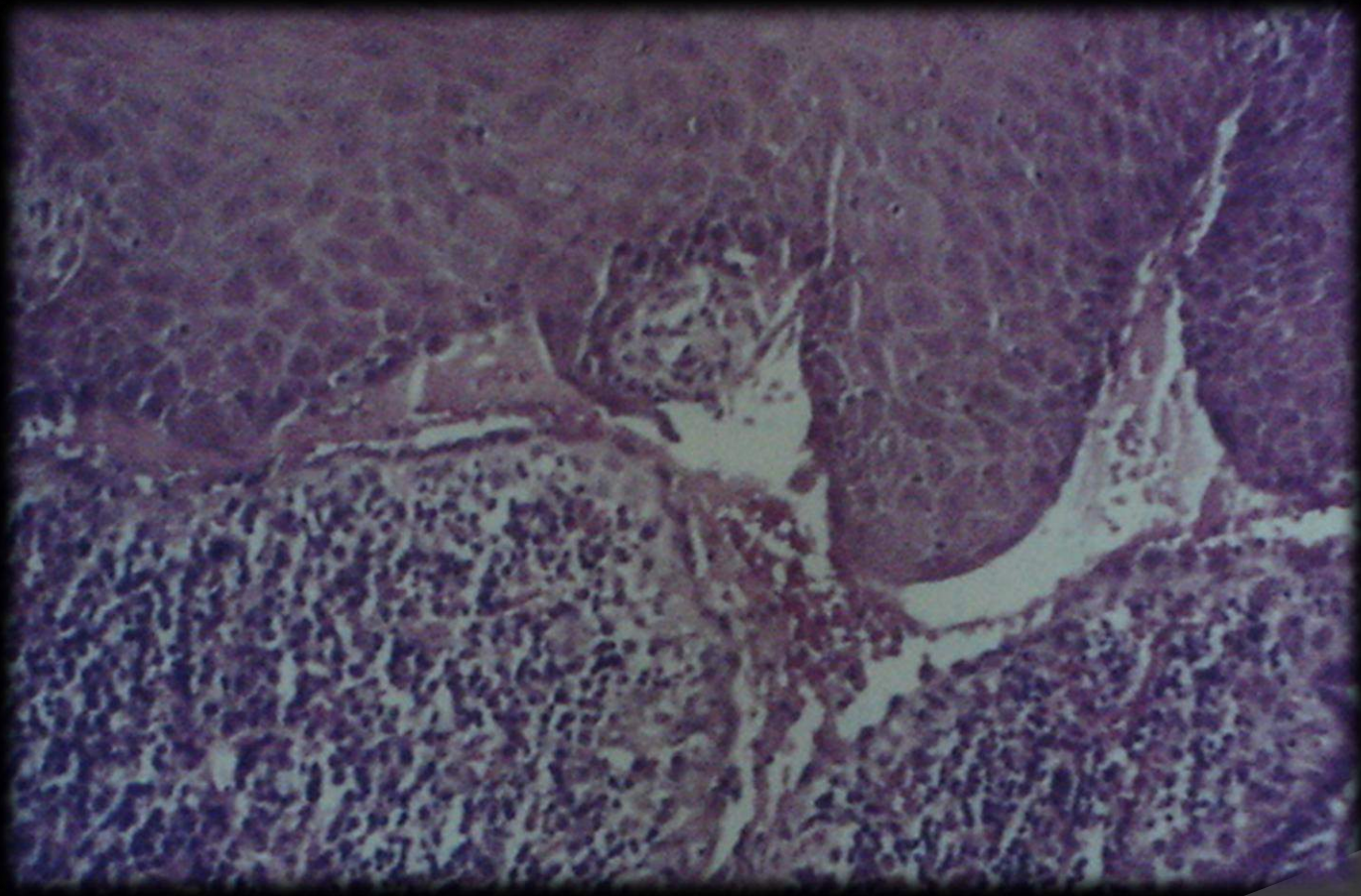
- ***Pathogenesis:*** cross-reactivity develops between antibodies produced in tumor & antigens associated with desmosomal complex & BM of epithelium.

Clinical features

- History of lymphoreticular neoplasm or thymoma.
- Mutiple vesiculobullous lesions affects the skin and oral mucosa.
- Crusted, hemorrhagic lip lesions may be mistaken for erythema multiforme or herpes simplex infection.
- Ocluar involvement.
- Oral ulceration are quite painful.

HP FEATURES

- Subepithelial and intra epithelial clefting (supra basilar)
- Lymphocytic infiltration.



Pemphigoid

- Cicatricial (mucous membrane)
- Bullous

Mucous membrane pemphigoid
Clinical features

- Autoimmune disease that primarily affects the mucous membrane ,including mouth, oro-pharynx, conjunctiva, and the geniitalia.
- Affects older adults, with an average age of 50-60yrs
- F>M 2:1

Etiology ;

- AUTOIMMUNE, autoanibodies against basement membrane zone antigens. BP 2 and laminin 5.

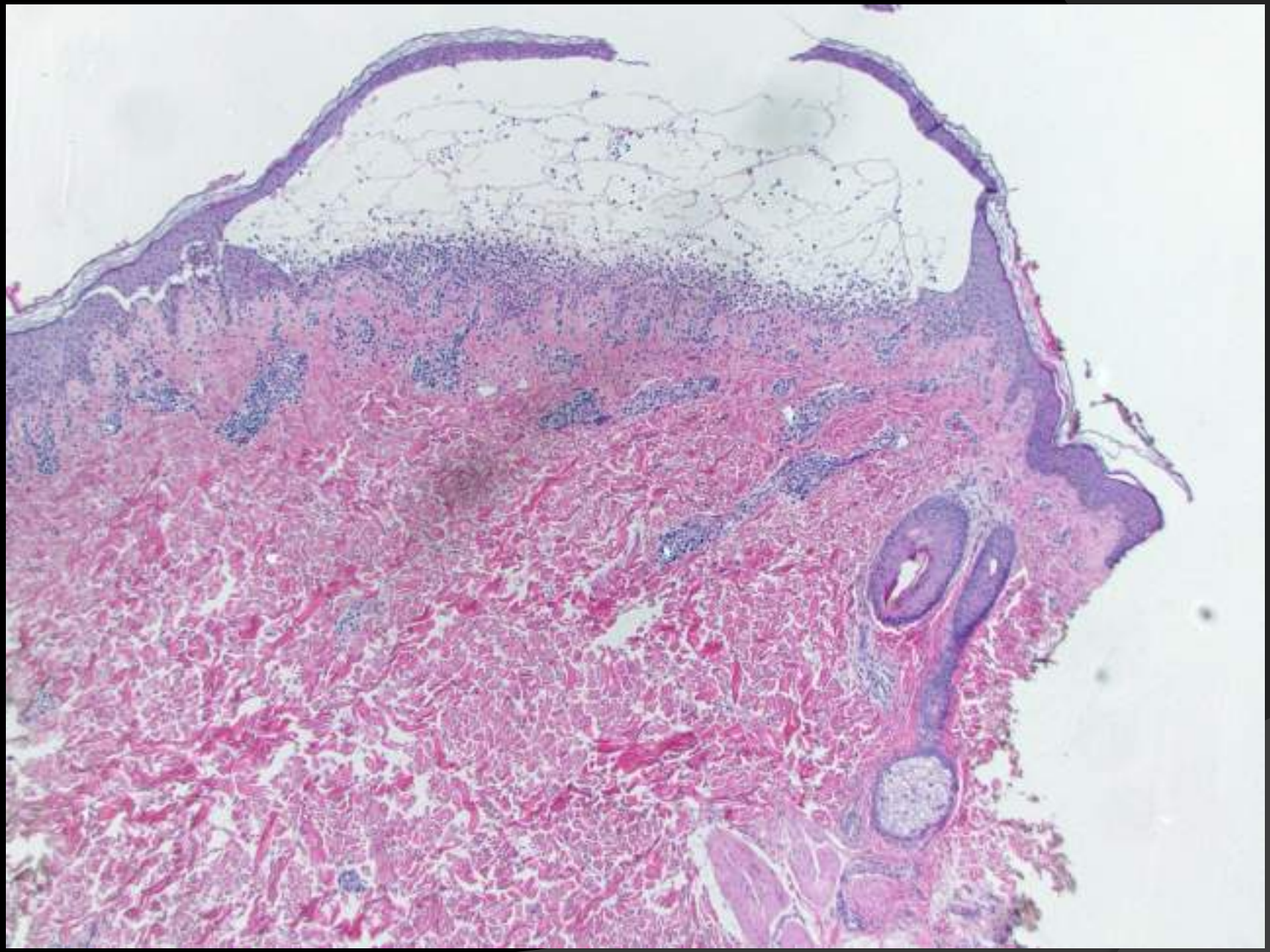
Clinical features

- Large, irregular oral ulcerations characterize the lesions after the initial rupture of bullae
- One or more intra oral vesicles, - soft palate
- Often the gingival tissues are the only affected site,(desquamative gingivitis, erosive LP)

- Patient with ocular involvement shows severe conjunctival inflammation
- The disease has caused the upper eyelid of the patient to turn inward, resulting in the eyelashes rubbing against the eye itself
- Complete scaring between conjunctival mucosa and the eyelids themselves, producing blindness

HP Features

- Sub epithelial clefting
- Direct immunoflorence-basement membrane zone,



Bullous pemphigoid

- Chronic autoimmune sub epidermal blistering skin disease that rarely involves mucous membrane
- Etiology – autoimmune ---- auto antibodies against hemidesmosomal BP antigens, BP230 & BP180

Clinical features

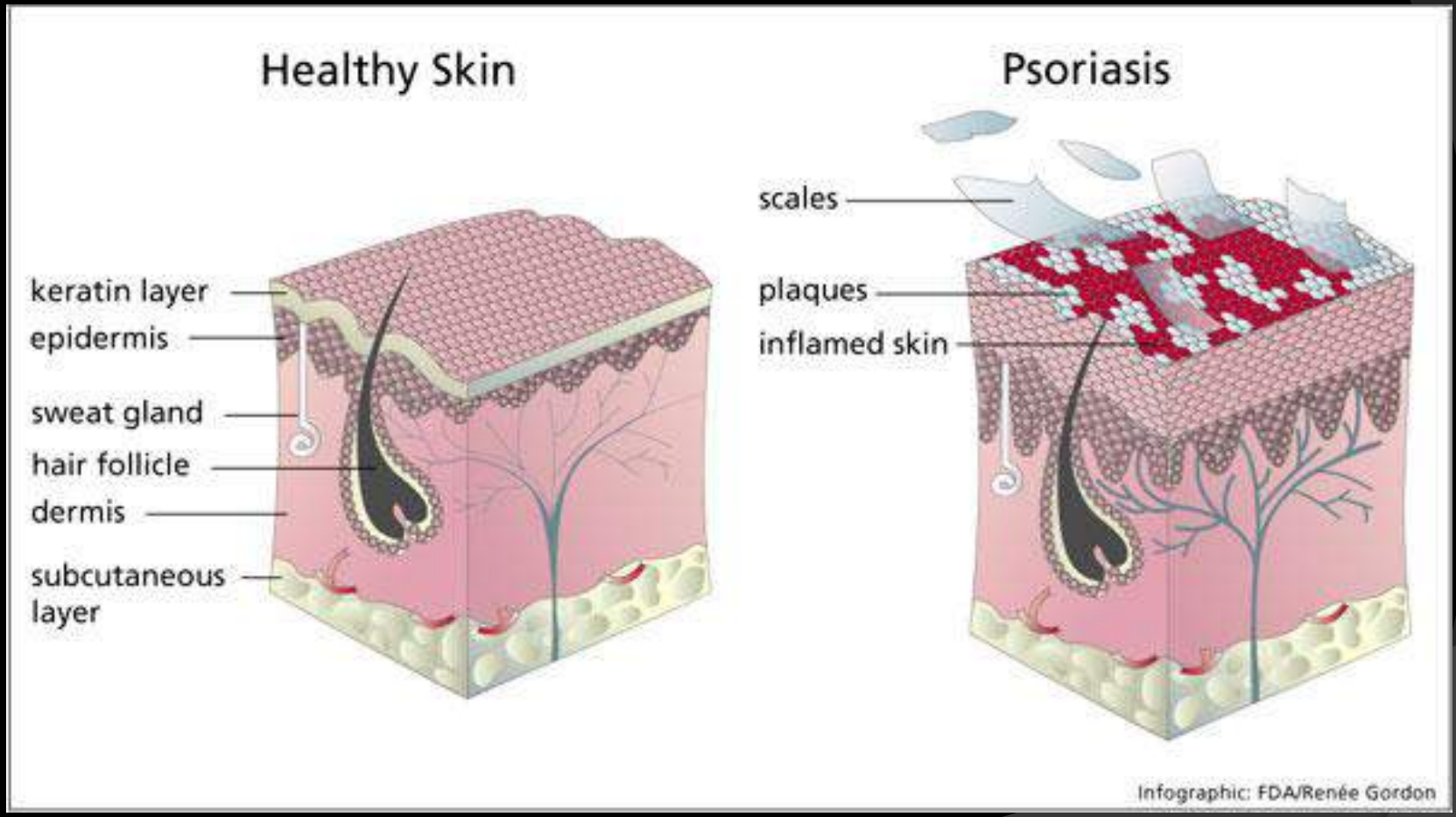
- Elderly 60>
- Cutaneous lesions – non specific rashes, common on limbs
- Utricarial and eczematous
- Vesicles and bullae (thick walled)
- Rupture do not always seen, when it does – eroded areas

Histopathology

- Sub-epithelial blisters, fibrinous exudate and inflammatory cells

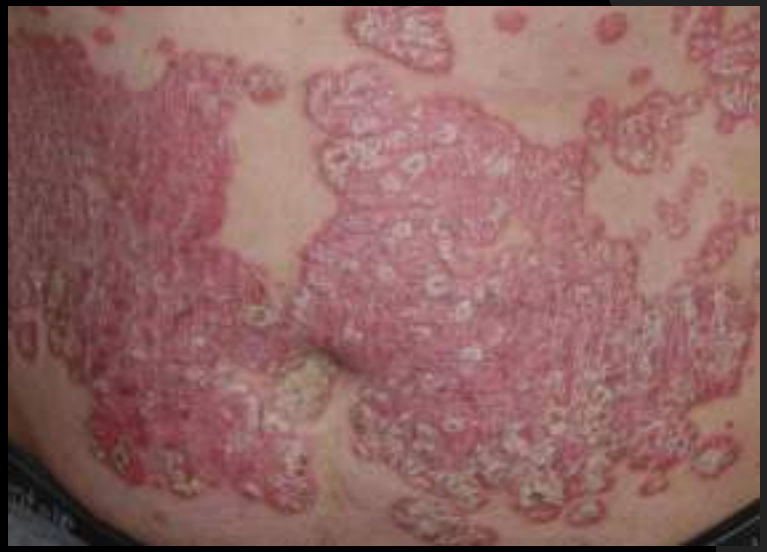
PSORIASIS

- Common inflammatory dermatologic disorder
- PATHOGENESIS:
- Increased proliferative activity of the cutaneous keratinocytes
- Activated T- lymphocyte: abnormal production of cytokines
- Genetic factors
- Environmental conditions


Healthy Skin
keratin layer
epidermis
sweat gland
hair follicle
dermis
subcutaneous layer
Psoriasis
scales
plaques
inflamed skin
Infographic: FDA/Renée Gordon

CLINICAL FEATURES

- 2nd to 3rd decade.
- Periods of exacerbation and quiescence
- Improve during summer and worsen during winter
- Scalp, elbows and knees
- Asymptomatic, itching
- Psoriatic arthritis: TMJ

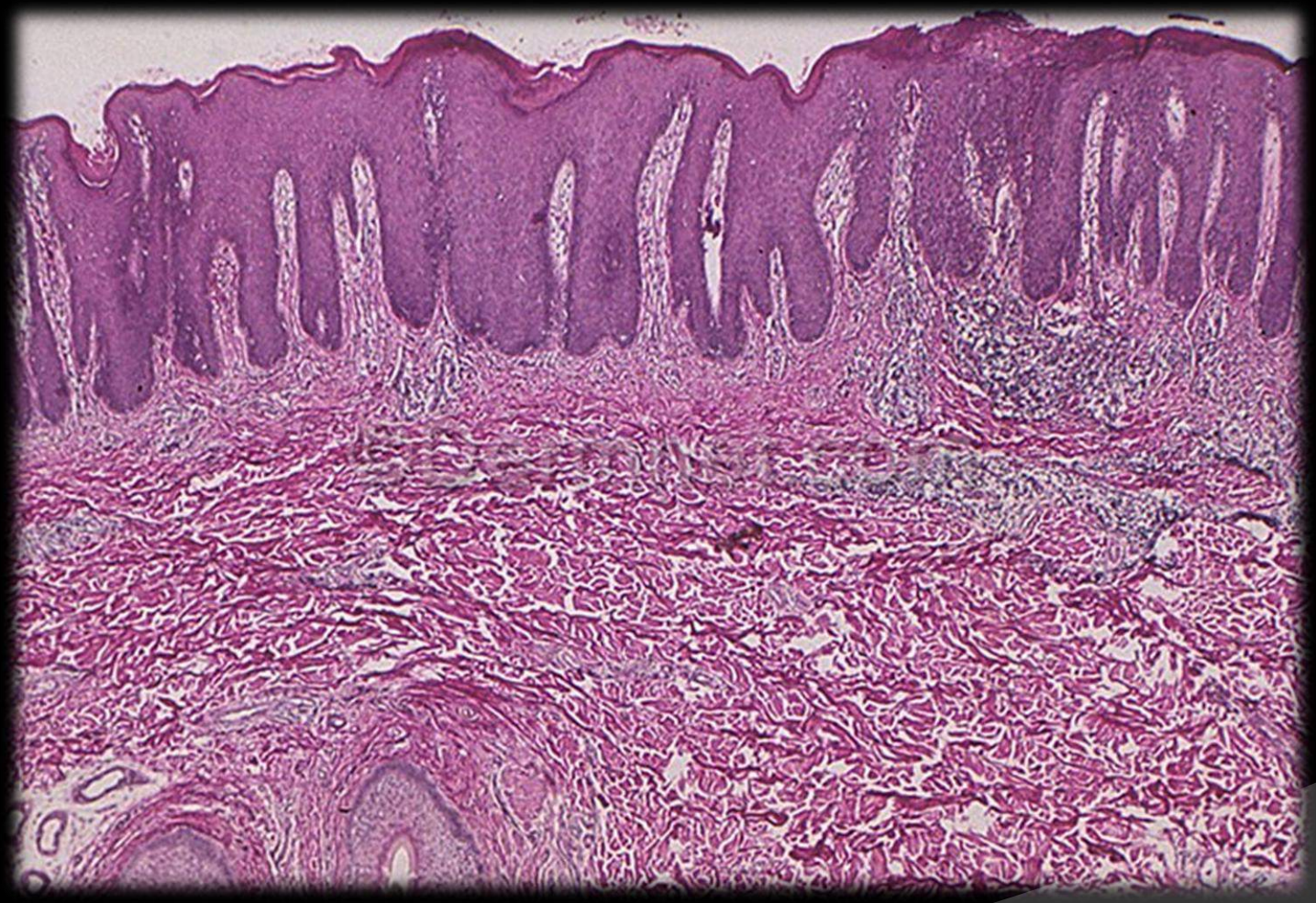


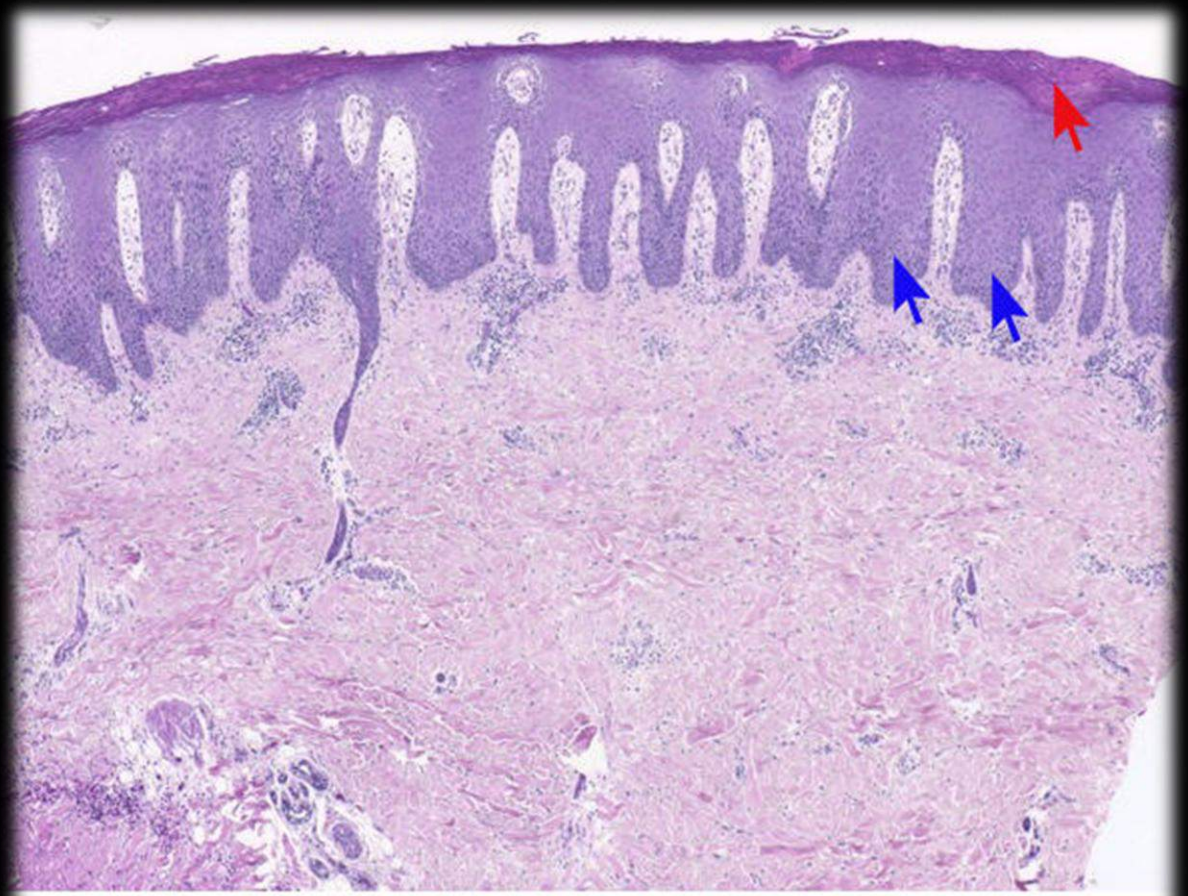
ORAL LESIONS

- Uncommon.
- White plaques to red to ulcerations.
- Erythema migrans: intraoral psoriasis.

HISTOPATHOLOGICAL FINDINGS

- Marked parakeratin production.
- Epithelial reteridges are elongated.
- CT: dilated capillaries.
- Perivascular ch.inflammatory cells.
- Munro’s abscess: collection of neutrophils.





Hyperkeratosis and parakeratosis

Regular elongation of rete ridges

LUPUS ERYTHEMATOSUS

- Lupus erythematosus is a classic example of immunologically mediated condition

- It is the most common collagen vascular disorder in USA

- **3 clinical forms: Severity and distribution**.
- Mildest form, Discoid lupus erythematosus [DLE].
- Intermediate form, Subacute cutaneous lupus erythematosus [SCLE].
- Severest form, Systemic lupus erythematosus [SLE].

- Discoid – confined to skin & mucosa
- Systemic -- multi organ involvement

CRITERIA BY AMERICAN RHEUMATISM ASSOCIATION

- Malar rash
- Neurologic
- Hematologic
- Photosensitivity
- Immunologic
- Oral ulcers
- Antinuclear antibody
- Arthritis
- Kidney involvement

Etiology

- Genetic
- Immunological abnormality possibly mediated by viral infection
- Autoimmune disease
- Endocrine

SYSTEMIC LUPUS ERYTHEMATOSUS

- 31yrs.
- M:F=1:10
- Fever, weight loss, arthritis,fatigue,general malaise :40%-50%.
- Characteristic rash,having the pattern of butterfly, develops over malar area and nose.

- Sunlight worsens the lesion.
- Kidneys are affected:40-50%.
- Cardiac involvement-commonly pericarditis.

ORAL LESIONS:

- 5% - [25-50%].
- Palate, buccal mucosa, gingiva.
- Lupus cheilitis.
- Ulceration, pain, erythema.
- Xerostomia,stomatodynia, candidiasis,periodontal disease.

DISCOID LUPUS ERYTHEMATOSUS

- Common disease.
- 3rd & 4th decades of life.
- More common in women.
- Most common sites : face, oral mucous membranes, chest, back,& extremities.

SKIN LESIONS: Slightly elevated red or purple macules that are often covered by gray or yellow adherents.

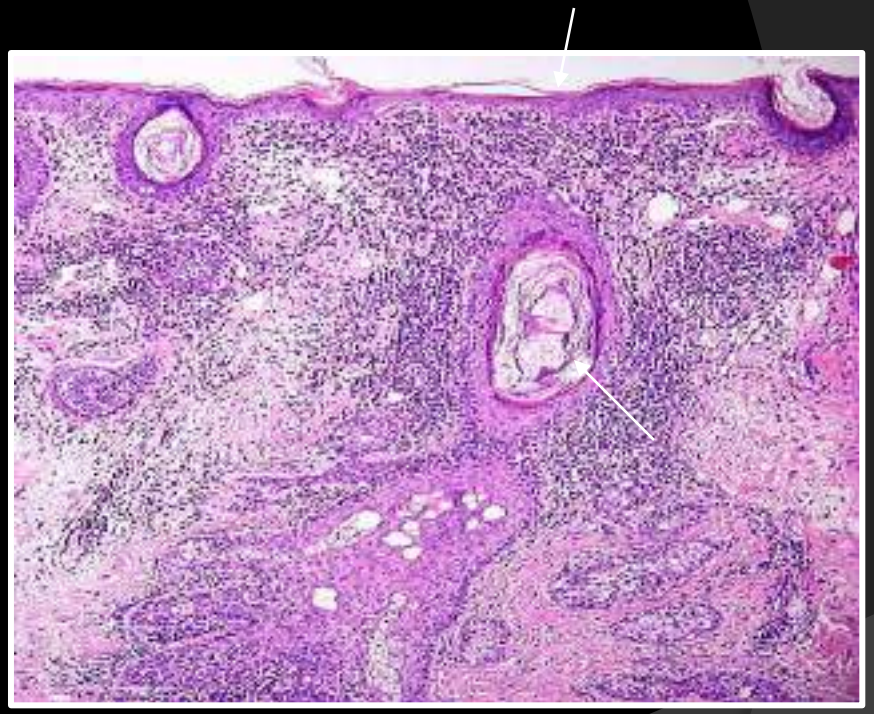
- Increase in size by peripheral growth.
- Periphery: pink or red, central :atrophic & scarring

ORAL LESIONS:

- Erythematous areas.
- Slightly elevated or depressed with white spots.
- Buccal mucosa, palate, tongue.
- Most common site: vermilion border of lower lip.
- Fine white striae radiate out from the margin.

HISTOPATHOLOGICAL FEATURES

- Hyperkeratosis, ”follicular plugging”.
- Degeneration of basal cell layer.
- Chronic inflammatory cell infiltrate in the CT.
- Infiltrate surrounds the blood vessels.



TREATMENT

- Avoid exposure to sunlight.
- Mild-NSAID’s combined with antimalarial drugs.
- Severe-Systemic corticosteroids combined with immunosuppressive agents.
- Oral lesions-systemic corticosteroids.

CREST SYNDROME

Acrosclerosis

- It is an uncommon mild variant of systemic sclerosis.

C- Calcinosis cutis.
R- Raynaud's phenomenon
E- Esophageal dysfunction
S- Sclerodactyly
T- Telangiectasia.

Calcinosis- calcium deposits in the skin

Raynaud's phenomenon- spasm of blood vessels in response to cold or stress

Esophageal dysfunction- acid reflux and decrease in motility of esophagus

Sclerodactyly- thickening and tightening of the skin on the fingers and hands

Telangiectasias- dilation of capillaries causing red marks on surface of skin

A.D.A.M.

TELANGIECTASIA

- Finger become stiff.
- Skin –smooth, shiny appearance.
- Undergo permanent flexure- claw deformity.
- Due to abnormal collagen deposition.

FOCAL DERMAL HYPOPLASIA

- [Goltz-Gorlin syndrome]

CLINICAL FEATURES

- Female predominance.
- Focal absence of dermis.
- Skin atrophy.
- Streaky pigmentation.

Characteristic linear, erythematous, raised
and depressed macules.

Oligodactyly of the hand.

- Sclerodactyly

- Oligodactyly of the feet.
- Erythematous hyperpigmentation on the limbs.

- Reticulate, mildly atrophic, erythematous macules and soft, rounded nodules.
- Mental retardation.
- Eye anomalies
- Multiple papillomas of mucosa /skin.
- Sparse eyebrows and scalp hair.

ORAL MANIFESTATIONS

- Papillomas of lips, buccal mucosa or gingiva.
- Teeth are defective in shape, structure and size.
- Microdontia & enamel hypoplasia-common.
- Cleft lip/palate.

HISTOPATHOLOGICAL FINDINGS

Characteristic absence of dermal collagen and the accompanying appearance of adipose tissue in the dermis.

DEVELOPMENTAL DISTURBANCES

OF

ORAL SOFT TISSUES

Malformation

A structural defect in the body due to abnormal embryonic or fetal development.

Deformation

A change from the normal size or shape of an anatomic structure due to mechanical forces that distort an otherwise normal structure

It is different from malformation – In malformation evelopment of a structure is arrested, delayed or misdirected in embryonic life

Congenital

A condition that is present at birth, whether or not it is inherited

Hereditary

Conditions capable of being transmitted from parent to offspring through genes

Anomaly

Deviation from the normal

Anomalad

A malformation together with its subsequently derived structure

Contents

- Developmental disturbances of Tongue
- Developmental disturbances of Lips
- Developmental disturbances of Cheek
- Developmental disturbances of Gingiva
- Developmental disturbances of Soft palate
- Developmental disturbances of Salivary glands

DEVELOPMENTAL DISORDERS OF TONGUE

- Aglossia / Microglossia
- Macroglossia
- Ankyloglossia
- Fissured tongue
- Bifid tongue/ cleft tongue
- Median rhomboid glossitis
- Benign migratory glossitis
- Hairy tongue
- Lingual thyroid

Aglossia /microglossia

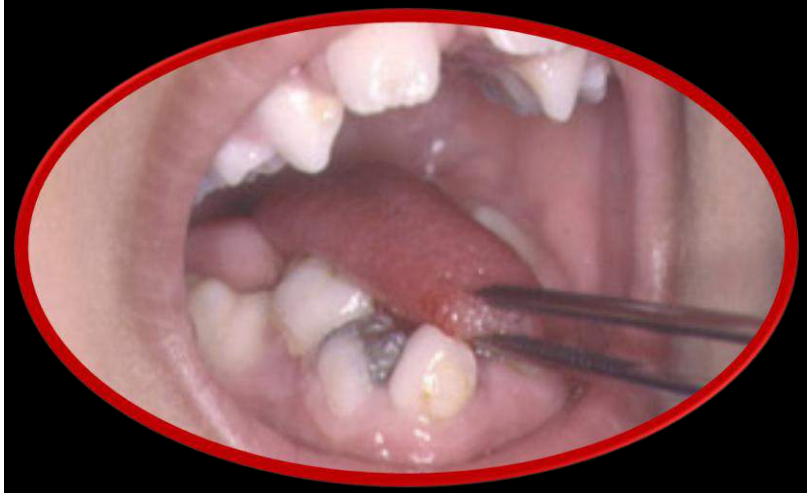
- Aglossia and microglossia syndrome.
- Condition where the size of the tongue is abnormally small.
- The congenital anomaly/malformation is extremely uncommon.
- Fewer than 50 cases have been described till date.
- Cases of complete absence of the tongue have also been reported which is termed as **AGLOSSIA.**
- True sense – Aglossia is extreme microglossia with glossoptosis.

Hypoplasia of the mandible and lack of muscular stimulus between the arches.

Failure of mandible to develop in anterior and transverse directions

Extremely small tongue

- Difficulties in speech and mastication.
- Severe malocclusion.



Macroglossia

- Macro – large
- Glossia – Tongue
- Relatively uncommon disorder.

- Possible causes

- Congenital syndromes - Down syndrome, Beckwith-Wiedemann syndrome
- Dilation of lymphatics & veins - Lymphangioma & lingual varix
- Muscle hypertrophy
- Inflammation/ infections - Syphilis,pneumonia.TB,candidiasis,smallpox
- Endocrinal – Diabetes, cretinism ,hypothyroidism, acromegaly.
- Neoplastic
- Infiltrative – Amyloidosis, sarcoidosis

- Two broad category – true and pseudo

Macroglossia leads to variety of consequences.

- Tongue protrusion, which exposes the tongue to trauma leads to mucosal drying and recurrent upper respiratory tract infections.
- Noisy breathing
- Speech impediment
- Swallowing difficulties
- Drooling of saliva
- Airway obstruction

Treatment

- Successful treatment require appropriate rehabilitation and long term follow up.
- Treatment options varies from observation, orofacial therapy and surgery.
- In adults the initial treatment should be conservative but main treatment is partial glossectomy.

Ankyloglossia

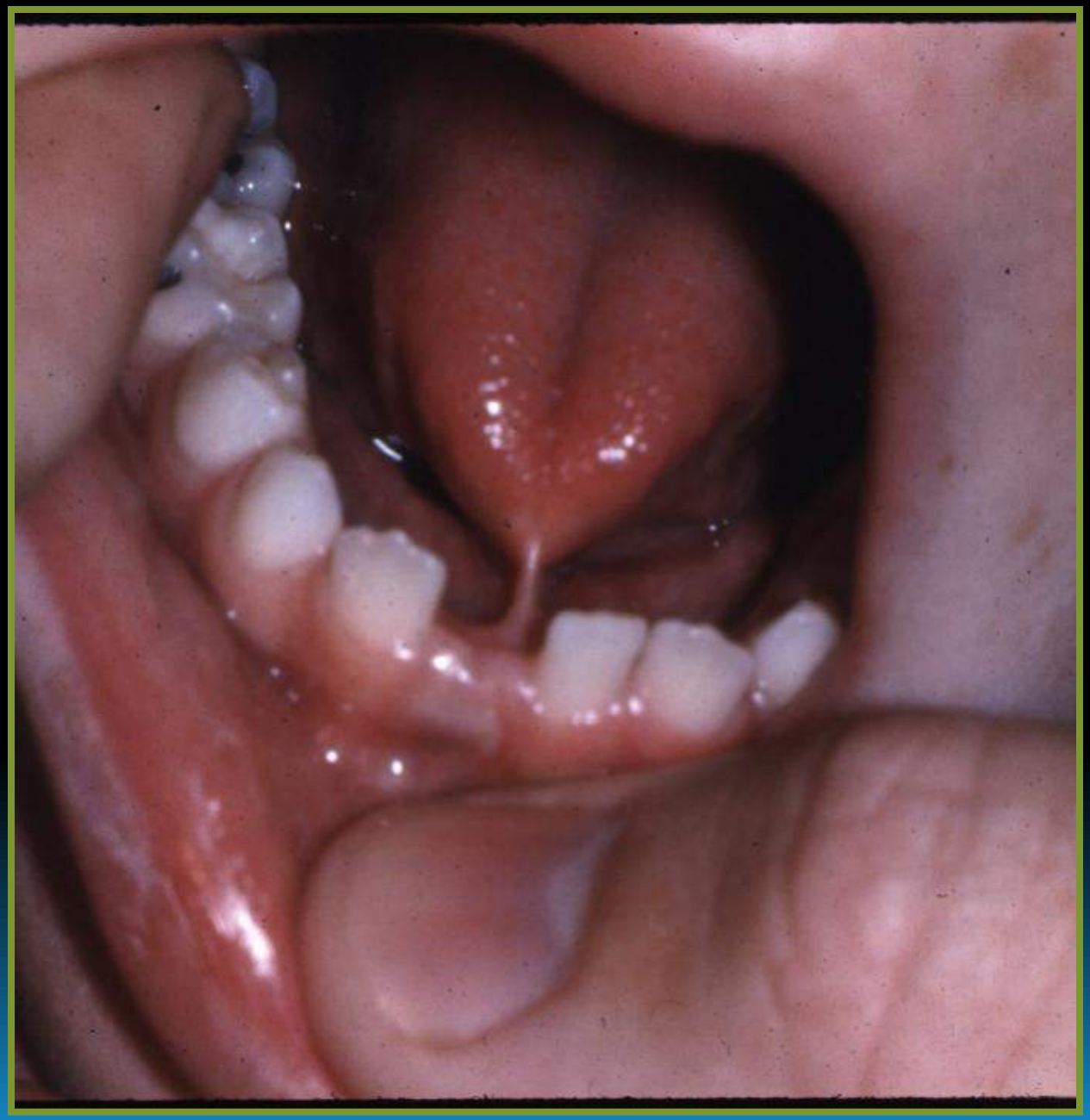
- ‘Ankyloglossia', ‘short frenum', 'short frenulum', & 'tongue tie’.

- Refers to restricted tongue movement.

- Fusion of the lingual frenum to the floor of the mouth or due to high lingual frenum attachment.

- Partial ankyloglossia or "tongue-tie" is a much more common condition.

- This leads to a myriad of speech problems such as lisping and stuttering, and swallowing problems.



- The treatment is to surgically sever the connection between the frenum and the floor of the mouth.
- In young children treatment is postponed until 4 – 5 years since it is difficult to access severity of disorder in early life.

Cleft-tongue

•Bifid tongue.

•Partial or complete.

• complete cleft is rare and due to Lack of merging of the two lateral swellings of this organ.

•Partial cleft is more common and manifests as a deep groove on the dorsal surface of the tongue.

•Partial cleft is due to incomplete merging and failure of groove obliteration by underlying mesenchymal proliferation.

• Partial cleft is a common feature in the oro-facial-digital syndrome (thick fibrous bands in lower anterior mucobuccal fold and clefting of hypoplastic mandibular alveolar process).

FISSURED TONGUE

- **Furrowed Tongue**
- **Scrotal Tongue**
- **Grooved Tongue**
- **Lingua Fissurata**
- **Lingua Plicata**
- **Lingua Scrotalis**
- **Plicated Tongue**
- **Cerebriform Tongue**

- **Fissured tongue** is a condition frequently seen that is characterized by grooves that vary in depth and are noted along the dorsal and lateral aspects of the tongue.

- Definitive etiology is unknown.

- Depth of the fissures varies - 6 mm in diameter.

- The fissures or grooves may be interconnected, separating the tongue dorsum into several lobules.

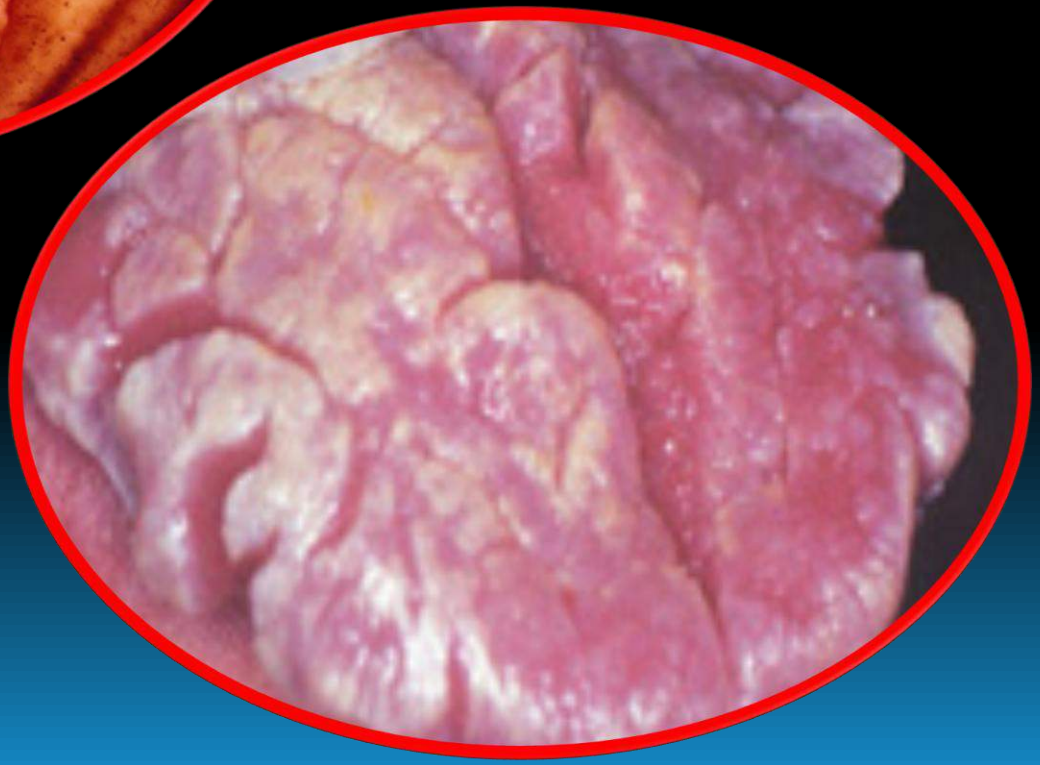
- May develop in association with infection or malnutrition.

- Melkersson-Rosenthal syndrome, Down syndrome and benign migratory glossitis.

- Melkersson-Rosenthal syndrome - persistent or recurring lip or facial swelling, intermittent seventh (facial) nerve paralysis and fissured tongue.

- In Melkersson-Rosenthal syndrome, the morbidity is not due to the fissured tongue but is secondary to the granulomatous inflammation of the lips/facial soft tissues and facial paralysis.

- Slight male predilection.



- **B**enign condition and is considered to be a variant of normal tongue architecture.

- Usually asymptomatic unless debris is entrapped within the fissure which causes irritation.

- May be congenital or become apparent during childhood or later.

- Diagnosed more frequently in adulthood.

- The prominence of the condition increase with the age.

Microscopy

- Hyperplasia of retepegs.
- Loss of keratin hairs on surface of filliform papilla.
- Papillas are seperated by deep grooves.
- Micro abscesses in upper epithelial layers and poly morpho nuclear infiltrate into epithelium.
- Mixed inflammatory infiltrate in the lamina propria.

Treatment

- No specific treatment required.
- Advised to brush tongue, since grooves may act as a source of bacterial accumulation and cause irritation.

Geographic tongue

- Benign migratory glossitis.
- Erythema migrans.
- Psoriasiform mucositis.
- Wondering rash of the tongue

Benign condition that occurs in about 3% of the general population.

Etiology

Poorly understood may be

- Hypersensitivity or environmental or hormonal factors.
- Although this is an inflammatory condition histologically, a polygenic mode of inheritance has been suggested because it is seen clustering in families.
- Associated with human leukocyte antigen DR5 (HLA-DR5), human leukocyte antigen DRW6 (HLA-DRW6), human leukocyte antigen Cw6 (HLA-Cw6).
- Associated with psoriasis and in patients with fissured tongue.

Clinical Features

- Affect all age groups , more predominant in adults.
- F:M = 2:1
- Lesions affect different areas of the tongue at different times .
- Burning sensation.
- The classic manifestation is an area of erythema, with atrophy of the filiform papillae of the tongue, surrounded by a serpiginous, white, hyperkeratotic border.
- Similar lesions may be present concurrently on other aspects of the tongue or other mucosal sites.

Histological findings

- Elongation of the rete ridges , hyperparakeratosis and acanthosis.
- Towards the center of the lesion, corresponding to the erythematous area clinically, loss of filiform papillae with migration and clustering of neutrophils within the epithelium is seen(monro's abscess).
- The predominant inflammatory infiltrate in the lamina propria is neutrophils with an admixture of chronic inflammatory cells.

Treatment

- No medical intervention is required because the lesion is benign and most often asymptomatic.
- In severe cases topical corticosteroids in the form of fluocinonide & beta methasone gel with zinc suppliments can be prescribed.

Median rhomboid glossitis

- Central papillary atrophy .
- Posterior lingual papillary atrophy.
- Posterior midline atrophic candidiasis as a more appropriate diagnostic term.

ETIOLOGY

- First suggested in 1914 by Brocq and Pautrier.
- The embryonic tongue is formed by two lateral processes (lingual tubercles) meeting in the midline and fusing above a central structure from the second branchial arch (copula) with hypobranchial eminence.
- During this period of fusion posterior dorsal point of fusion is occasionally defective, leaving a rhomboid-shaped, smooth erythematous mucosa lacking in papillae or taste buds derived from copula.
- The erythematous clinical appearance is due to the absence of filiform papillae in this region rather than to local inflammatory changes .

CLINICAL FEATURES

- The lesion is found in one of every 300-2,000 adults, depending on the rigor of the clinical examinations
- Shows 3:1 male predilection.
- Presents in the posterior midline of the dorsum of the tongue, just anterior to the V-shaped grouping of the circumvalate papillae.
- The long axis of the rhomboid or oval area of red depapillation is in the anterior-posterior direction.

- Lesions are typically less than 2 cm. in greatest dimension and most demonstrate a smooth, flat surface, although it is not unusual for the surface to be lobulated
- Occasional lesions have surface mamillations raised more than 5 mm above the tongue surface located somewhat anterior to the usual location. None have been reported posterior to the circumvallate papillae.
- Lesions associated with atrophic candidiasis are usually more erythematous but some respond with excess keratin production and, therefore, show a white surface change.
- Infected cases may also demonstrate a midline soft palate erythema in the area of routine contact with the underlying tongue involvement this is referred to as a kissing lesion.

MICROSCOPY

- Silver staining & PAS often reveal candida hyphae and spores in the superficial layers of the epithelium.

- This pseudoepitheliomatous hyperplasia may be quite pronounced, and the tangential cutting of such a specimen may result in the artifactual appearance of cut rete processes as unconnected islands of squamous epithelium.

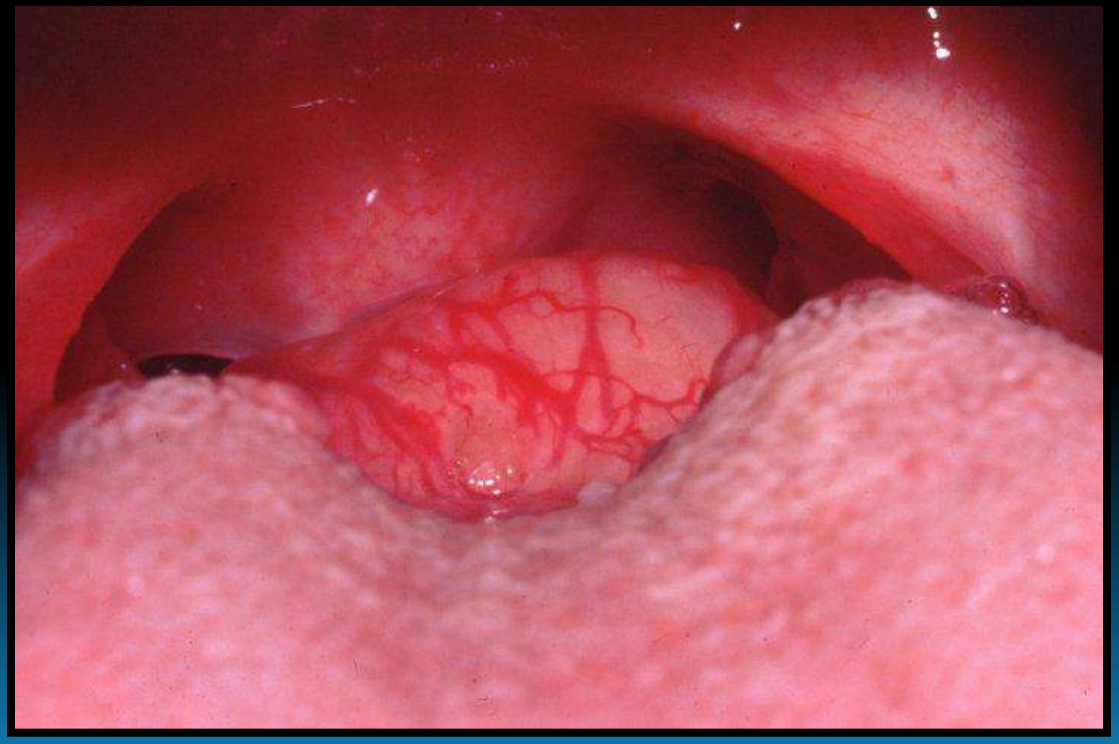
- Hence, it is recommended that the patient be treated with topical antifungals prior to biopsy of a suspected median rhomboid glossitis .

TREATMENT

- No treatment is necessary for median rhomboid glossitis, but nodular cases are often removed for microscopic evaluation.
- Recurrence after removal is not expected, although those cases with pseudoepitheliomatous hyperplasia should be followed closely for at least a year after biopsy to be certain of the benign diagnosis.
- Antifungal therapy (topical troches or systemic medication) will reduce clinical erythema and inflammation due to candida infection.
- Some lesions will disappear entirely with antifungal therapy.

LINGUAL THYROID NODULE

Accessory accumulation of thyroid tissue that is usually functional within the body of the posterior tongue.



ETIOPATHOGENESIS

- Late in the first month of Intra Uterine life the anlage of the thyroid gland descends from the posterior dorsal midline of the tongue (actually the floor of the pharyngeal gut) to its final position in the lower neck.

- If the embryonic gland does not descent normally, ectopic or residual thyroid tissue may be found between the foramen caecum and the epiglottis.

- Approximately two-thirds of patients with lingual thyroid lack thyroid tissue in the neck.

CLINICAL FEATURES

- The lingual thyroid is four times more common in females than in males.
- It presents as an asymptomatic nodular mass of the posterior lingual midline, usually less than a centimeter in size but sometimes reaching more than 4 cm in size .
- Larger lesions can interfere with swallowing and breathing, but most patients are unaware of the mass at the time of diagnosis, which is usually in the teenage or young adult years.
- Up to 70% of patients with lingual thyroid have hypothyroidism and 10% suffer from cretinism.
- Other sites of ectopic thyroid deposition include the cervical lymph nodes, submandibular glands and the trachea. Rarely, parathyroid glands are associated with the ectopic thyroid tissue.

MICROSCOPY

- The lingual thyroid consists of a non-encapsulated collection of embryonic or mature thyroid follicles which may extend between muscle bundles, raising suspicions of malignant invasion.

- The follicular cells are normal or atrophic in appearance.

- All diseases capable of affecting the normal thyroid gland can of course, affect the glandular tissue entrapped in the tongue.

- Thyroid adenoma, goiter, hyperplasia, inflammation, and carcinoma occur in lingual thyroids and therefore, be evaluated in the same fashion as would any biopsied thyroid gland.

- In those patients lacking thyroid tissue in the neck, the lingual thyroid can be excised and autotransplanted to the muscles of the neck.

- However, Most cases require no treatment and in cases where biopsy is necessary it should be considered with caution because of the potential for hemorrhage, infection or release of large amounts of hormone into the vascular system (thyroid storm).

- Rare examples of thyroid carcinoma arising in the mass have been reported, almost always in males.

LINGUAL VARICOSITIES

- Prominent lingual veins, usually observed on the ventral and lateral surface of the tongue are called lingual varicosities.
- These are hemmorhoid (dialated veins) caused by the decrease in the amount of surrounding connective tissue.
- A normal variant in adults over 60 years of age believed to be related to the aging process.
- It's occurance increases with age or increased blood pressure

- Enlarged veins usually purple or red or clusters on ventral and lateral surface of the tongue.

- There is no association with other systemic diseases; however, a relationship between varicosities in the legs and prominent lingual veins has been reported.

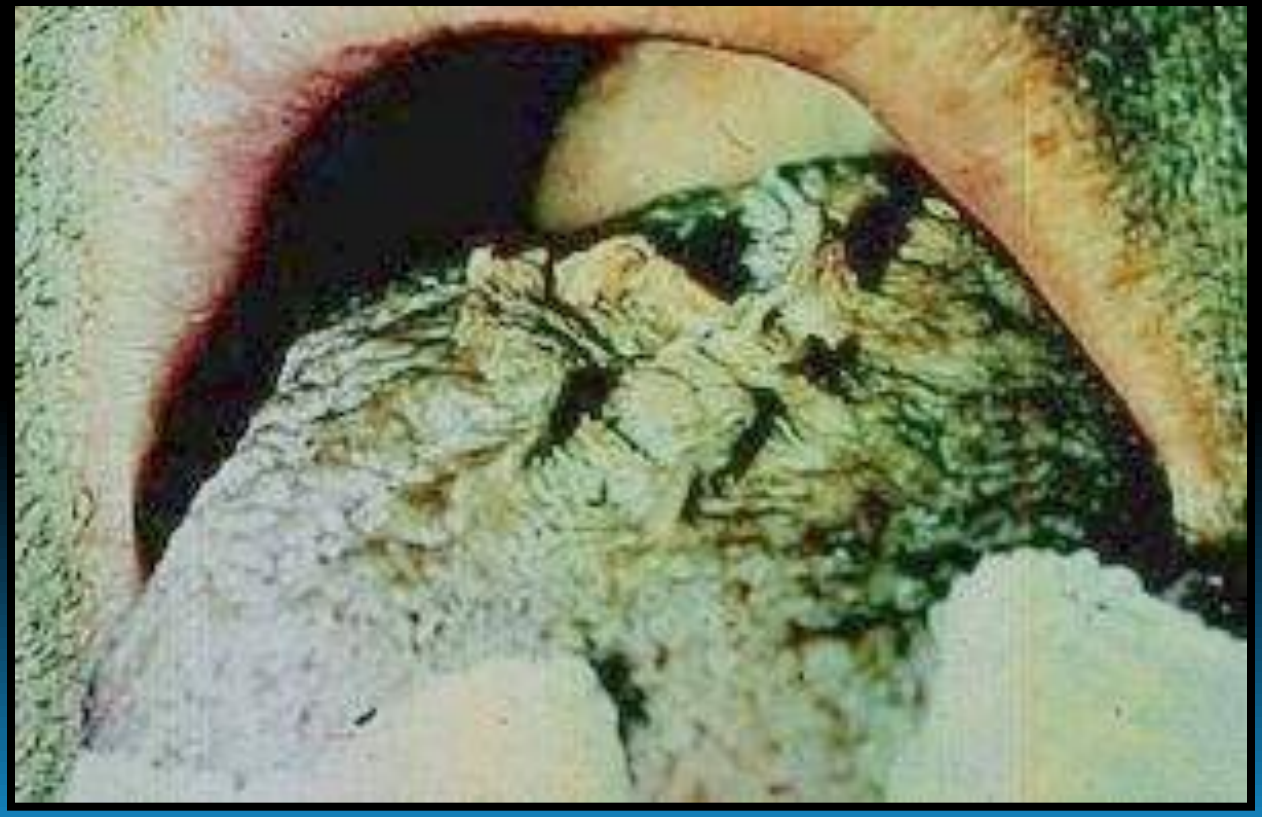
- **Microscopy** reveal dilated veins with walls showing little smooth muscle and poorly developed elastic tissue .

- Thrombosis show lines of zahn which may result in recanalization or phlebolith formation

- **Treatment** is not necessary.

HAIRY TONGUE

- Commonly observed condition of defective desquamation of the filiform papillae that results from a variety of precipitating factors.



- The condition is most frequently referred to as black hairy tongue (lingua villosa nigra), hairy tongue may also appear brown, white, green, pink, or any of a variety of hues depending on the specific etiology and secondary factors.

ETIOLOGY

- The basic defect in hairy tongue is a hypertrophy of filiform papillae on the dorsal surface of the tongue, with a lack of normal desquamation.

- Contributory factors for hairy tongue are numerous and include poor oral hygiene, tobacco use, coffee or tea drinking, the use of medications (especially broad-spectrum antibiotics) and therapeutic radiation of the head and the neck.

CLINICAL FEATURES

- Normal filiform papillae are approximately 1 mm in length, whereas filiform papillae in hairy tongue have been measured at more than 15 mm in length.
- Hairy tongue is rarely symptomatic, although overgrowth of *Candida albicans* may result in glossopyrosis (burning tongue).
- Patients frequently complain of a tickling sensation in the soft palate and the oral pharynx during swallowing.
- In more severe cases, patients may actually complain of a gagging sensation. Retention of oral debris between the elongated papillae may result in halitosis.
- Although hairy tongue is reported more often in males, it is not uncommon in females, especially those who drink coffee or tea and/or those who use tobacco.

- The incidence and the prevalence of hairy tongue increases with age.
- Patients occasionally notice the condition of the tongue during tooth brushing and present to the office with concerns regarding potential malignancy.
- The tongue has a thick coating in the middle, with a greater accentuation toward the back.
- Bacterial and fungal overgrowth play a role in the color of the tongue.

MICROSCOPIC FINDINGS

- Consist of elongated filiform papillae, with mild hyperkeratosis and occasional inflammatory cells.
- Finding accumulated debris intermingled among the papillae and candidal pseudohyphae is not unusual.
- Culture of the tongue's dorsal surface may be taken if a superimposed oral candidiasis or other specific oral infection is suspected.
- Cytologic smears stained with Gram stain or periodic acid-Schiff stain may reveal candidal organisms.

DEVELOPMENTAL DISORDERS OF

LIPS

COMMISSURAL LIP PITS

- Small mucosal invaginations that occur at the corners of the mouth on the vermilion border.

ETIOLOGY

- Family history --- Autosomal dominant transition.

- Their location suggests that they may represents a failure of normal fusion of the embryonal maxillary and mandibular process.

CLINICAL FEATURES

- Common in male
- Unilateral or bilateral
- Blind fistulas that may extend to a depth of 1-4 mm.
- Small fluid may expressed when they squeezed – representing drained saliva in to these pits.

CONGENITAL LIP PITS

- Para median lip pits or Congenital invagination of the lower lip.
- Believed to arise from persistent lateral sulci on embryonic mandibular arches. (usually disappears by 6^{th} week)
- 1-1.5 cm in depth.
- Occasionally single, occurs at the center of the lip.
- Vander Woulde syndrome.
- Popliteal pterygium syndrome

HISTOPATHOLOGY

- Narrow invagination of the surface epithelium lined by stratified squamous epithelium.

- Connective tissue reveals minor salivary gland ducts draining into the sulcus.

TREATMENT

- No specific treatment. Surgical excision is considered.

DOUBLE LIP

- Rare developmental anomaly characterized by redundant fold of tissue on the mucosal side of the lip.

ETIOPATHOGENESIS

CONGENITAL DOUBLE LIP

- During fetal development the upper lip mucosa consists of two transverse zones ,an outer zone, which is smooth and similar to skin, (pars glabra), & the inner zone, which is villous and similar to the oral mucosa,(pars villosa).

- The furrow dividing the double lip represents an exaggerated boundary line between the two zones.

- In the double lip buccal villous part becomes hypertrophic.

- Upper lip > lower lip

- Though the deformity can occur at birth, it usually becomes evident after the eruption of the permanent teeth.

ACQUIRED DOUBLE LIP

- Develops after a trauma to lips.

- Associated with Ascher ‘s syndrome.

  Double lip, Blephrocholasis, Non toxic thyroid enlargement.

CHELITIS GRANULOMATOSA

CLINICAL FEATURES

Cheilitis granulomatosa is episodic with nontender swelling and enlargement of one or both lips.

- The upper lip is involved slightly more often than the lower lip, and it may feel soft, firm, or nodular on palpation.

- The earliest manifestation is sudden diffuse or occasionally nodular swellings of the lip or the face.

- The first episode typically subsides completely in hours or days. After recurrent attacks, swelling may persist and slowly increase in degree, eventually becoming permanent.

- Granulomatous cheilitis is a chronic swelling of the lip due to granulomatous inflammation.

CAUSES

- Unknown.
- Genetic predisposition may exist in Melkersson-Rosenthal syndrome.
- Dietary or other antigens are the most common identified cause of orofacial granulomatosis.

PATHOPHYSIOLOGY

- In granulomatous cheilitis, normal lip architecture is eventually altered by the presence of lymphoedema and noncaseating granulomas in the lamina propria.

- Attacks sometimes are accompanied by fever and mild constitutional symptoms (eg, headache, visual disturbance).

- The enlarged lip appears cracked and fissured with reddish brown discoloration and scaling.

- The fissured lip becomes painful and eventually acquires the consistency of firm rubber.

- Swelling may regress very slowly after some years.

- Regional lymph nodes are enlarged (usually minimally) in 50% of patients.

Histologic Findings

- Histologic changes are not always conspicuous or specific in many cases of long duration;

- Chronic inflammatory cell infiltrate, peri & para vascular aggregations of lymphocytes, plasma cells and Histiocytes

- The infiltrate becomes denser and pleomorphic, and small focal granulomas are formed that are indistinguishable from Crohn disease or sarcoidosis.

- Small granulomas occur in the lymphatic walls in some cases. Similar changes may be present in cervical lymph nodes.

CHEILITIS GLANDULARIS

The condition is characterized by progressive enlargement and eversion of the lower labial mucosa that results in obliteration of the mucosal-vermilion interface.

CLINICAL FEATURES

- Enlargement and the loss of elasticity of the lip with pain.
- Initially Asymptomatic lip swelling occurs with clear viscous secretion expressible from dilated ductal openings on the mucosal surface.
- Periods of relative quiescence interrupted by transient or persistent painful episodes associated with suppurative discharge.

- A burning discomfort or a sensation of rawness referable to the vermilion border may be reported.
- This is associated with atrophy, speckled leukoplakic change, erosion, or frank ulceration with crusting.
- Affects the lower lip almost exclusively. It manifests as progressive, often multinodular enlargement, eversion, and induration.
- Salivary gland duct orifices may be dilated and appear as red or black puncta.

HISTOLOGIC FINDINGS

- Epithelium can be essentially normal or show evidence of disturbance that ranges from varying degrees of atypia or dysplasia to frank carcinoma.

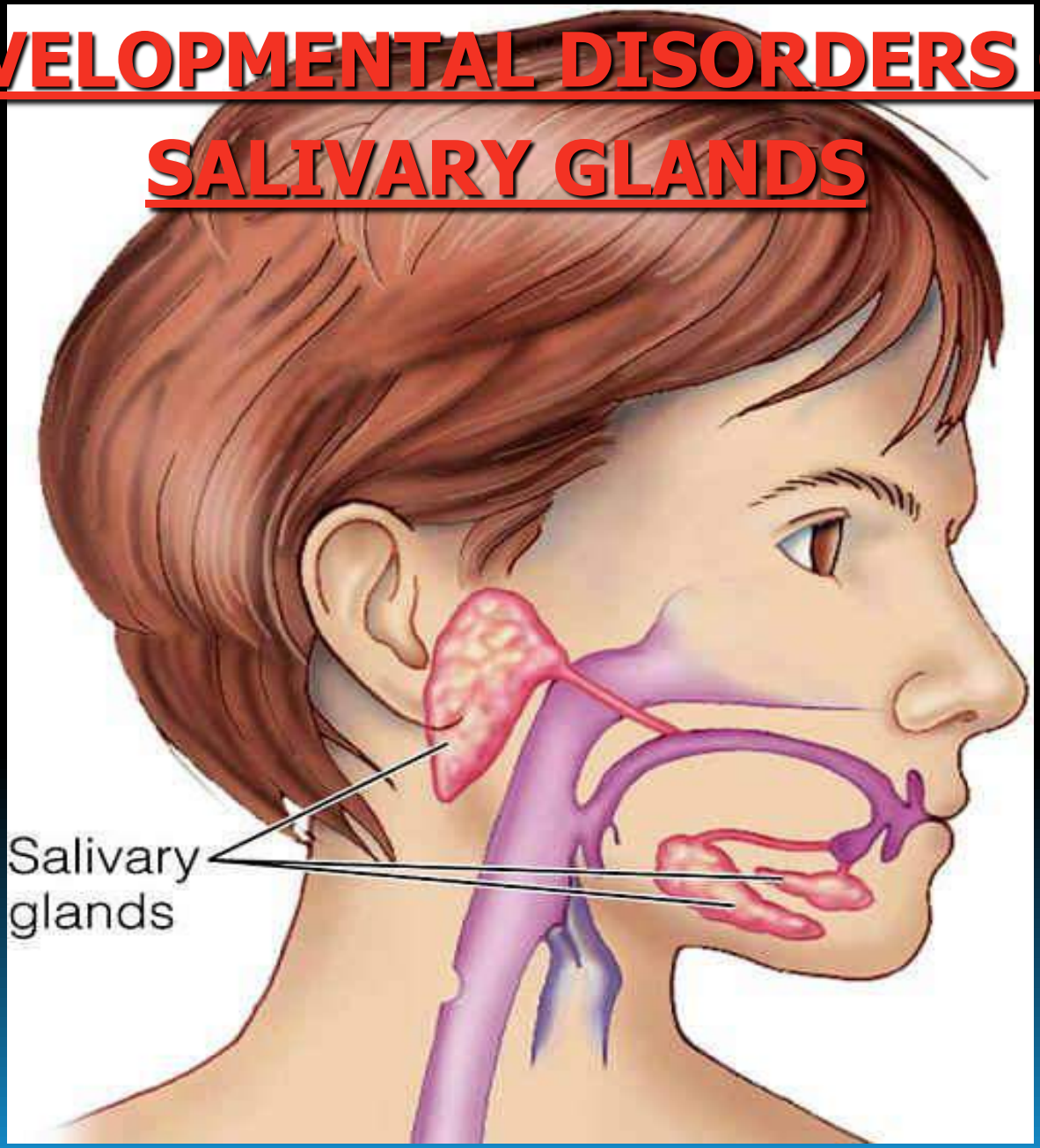
- The severity of epithelial maturational disturbance tends to correspond with the degree of risk for progression to carcinoma.

- Minor salivary glands show changes that include atrophy or distention of acini, with or without squamous metaplasia.

- Chronic inflammatory infiltration and replacement of glandular parenchyma, and interstitial fibrosis.

- Other possible histologic findings include stromal edema, hyperemia, surface hyperkeratosis, erosion, or ulceration

DEVELOPMENTAL DISORDERS OF SALIVARY GLANDS



APLASIA (AGENESIS)

- **Aplasia** of the salivary glands is an occasional finding.
- Any one or group of salivary glands may be absent, unilaterally or bilaterally.
- Aplasia becomes manifested with the development of xerostomia.
- A diagnosis of salivary gland aplasia is made after exclusion of the common causes of xerostomia - medications, sjogren's syndrome and radiation.

- The CT scan or MRI will indicate the gland's absence and its replacement by fat and fibrous tissue.
- Scinti-scanning with a radioisotope will confirm the initial diagnosis.
- The absence of the salivary duct orifice / papilla is an additional clue.
- Aplasia occurs for unknown reasons as an isolated finding or in conjunction with other developmental defects such as

Hemifacial microsomia,

LADD syndrome and

Mandibulo-facial dysostosis (Treacher Collins).

ATRESIA

- An abnormality of salivary gland duct.
- Congenital occlusion of the duct or absence of one or more salivary duct
- Exceedingly rare condition
- Atresia of the duct can be diagnosed by use of contrast sialography.
- Submandibular duct commonly involved

ABBERANCY

- Salivary gland tissue may arise in various aberrent locations within the head and neck.
- The most common site is in periparotid lymph nodes.
- Other sites include the lower neck, sternoclavicular joint, thyroglossal duct, middle ear, tonsils, hypopharynx, and mandible.
- This aberrant tissue may be located within or on the surface of the mandible.
- If located within the mandible, may be associated with the development of a malignant neoplasm (most commonly mucoepidermoid carcinoma), while that which is on the surface usually represents benign salivary gland tissue.

LINGUAL MANDIBULAR SALIVARY GLAND DEPRESSION

- **Stafne's Cyst**
- **Stafne's Bone Defect**
- **Static Bone Cyst**
- **Latent Bone Cyst**
- **ETIOLOGY** – unknown
- A developmental concavity of the lingual cortex of the mandible, usually in the third molar area, caused by overextension of an accessory lateral lobe of the submandibular gland.

CLINICAL FEATURES

- Most often adult males are affected
- Asymptomatic anatomic indentation of the posterior lingual mandible located below mandibular canal in molar region.
- Occasionally bilaterally present.
- Radiographically resembles a cyst with discrete corticated margin, oval radiolucency beneath the level of the inferior alveolar canal within the bone.

DEVELOPMENTAL DISTURBANCES OF BUCCAL MUCOSA

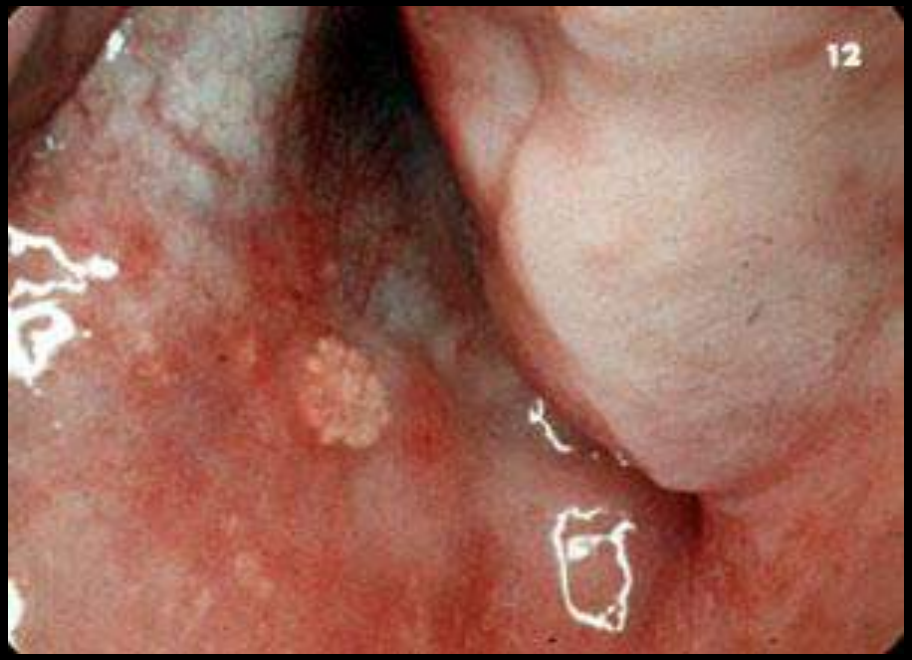
- Fordyce Granules
- Leukoedema
- Focal Epithelial Hyperplasia

FORDYCE GRANULES

- These are sebaceous glands that occur in the oral mucosa.
- These have been reported in 80% of the population, so their presence is considered as a normal anatomic variation.

CLINICAL FEATURES :

- Present as a multiple yellow or yellowish- white papular lesions.
- Buccal mucosa & lateral portion of the vermilion border of upper lip are commonly involved.
- Adults > children.
- Asymptomatic, few to hundred in number.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HISTOPATHOLOGY

- Sebaceous lobules are seen just beneath the epithelium.

- Similar to normal skin except for the absence of hair follicle.

LEUKOEDEMA

- Common oral mucosal condition of unknown cause.
- Leukoedema is an anatomic variation that represents hydration of the buccal mucosal epithelium and requires no treatment.
- It is a type of normal oral mucosa rather than a disease.
- Although, some studies pointed towards the developmental origin and some smoking, explanation is not satisfactory.

CLINICAL FEATURES

- Diffuse grayish – white milky opalescent appearance of the mucosa.
- The surface appears folded, resulting in wrinkles.
- Lesions do not rub off.
- Lesions diminishes or disappears when the cheek is stretched.

HISTOPATHOLOGY

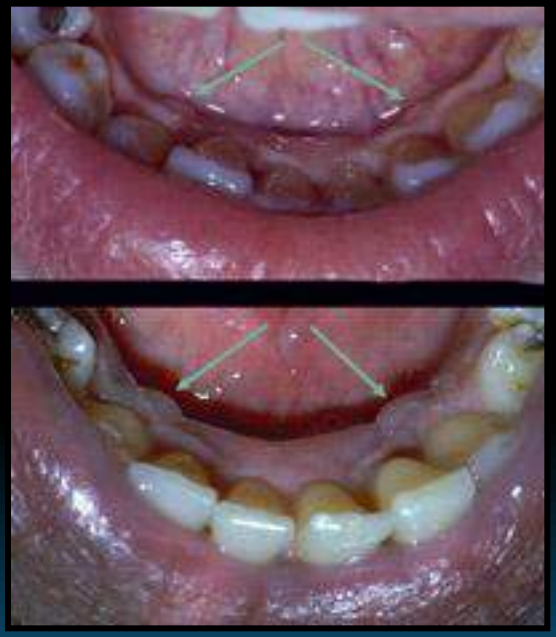
- Demonstrate increase in the thickness of the epithelium with striking intracellular edema of the spinous layer.
- Epithelial surface is usually Para keratinized & rete ridges are broad and elongated.
- Vacuolated epithelial cells show pyknotic nuclei.

DEVELOPMENTAL DISTURBANCES OF GINGIVA

- Retro cuspid Papillae
- Hereditary Gingival Fibromatosis

RETROCUSPID PAPILLAE

- Normal structures seen particularly in the younger age groups.
- They are found on the attached gingivae of the mandible, often bilaterally, lingual to the cuspid.
- Raised, nodules, usually 0.5 mm, with a broad base (sessile) or a pedunculated base (emerging from a stalk).
- They may regress with age, may be mistaken for disease but require no treatment.



HEREDITARY GINGIVAL FIBROMATOSIS

- Idiopathic gingival fibromatosis.
- The term Gingival Fibromatosis is used for those, which have an hereditary pattern or part of a more extensive syndrome.
- Localized & Generalized.
- Symmetrical fibromatosis of the tuberosity is a localized variant of gingival fibromatosis. This form is more common than generalized gingival fibromatosis.
- Appears to be a developmental type of phenomenon, although its true etiology is unknown.

CLINICAL FEATURES

- Generalized gingival fibromatosis presents as irregular enlargement of the facial and lingual aspects of the attached and marginal gingiva.
- A portion of one quadrant may be involved or all four quadrants of the gingival tissues may be involved.
- Enlargement is painless, slowly progressive and dependent to a great extent on the oral hygiene of the individual.
- The hyperplastic tissues are firm to palpation, inflammation and edema may make some surface areas spongy, erythematous and easily bleeding.
- It is not unusual for the fibromatosis to completely cover the teeth.

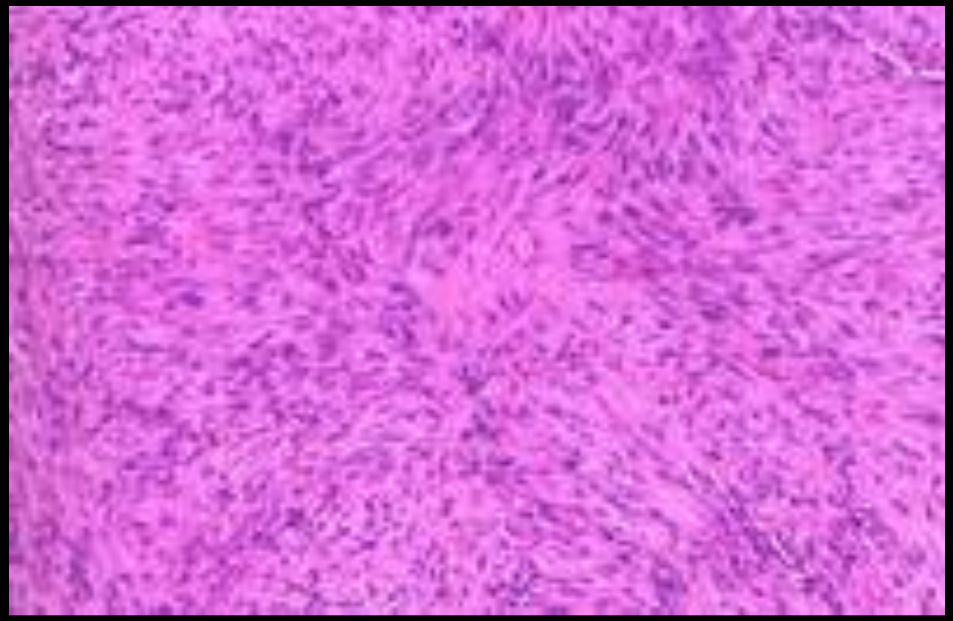
Histopathology

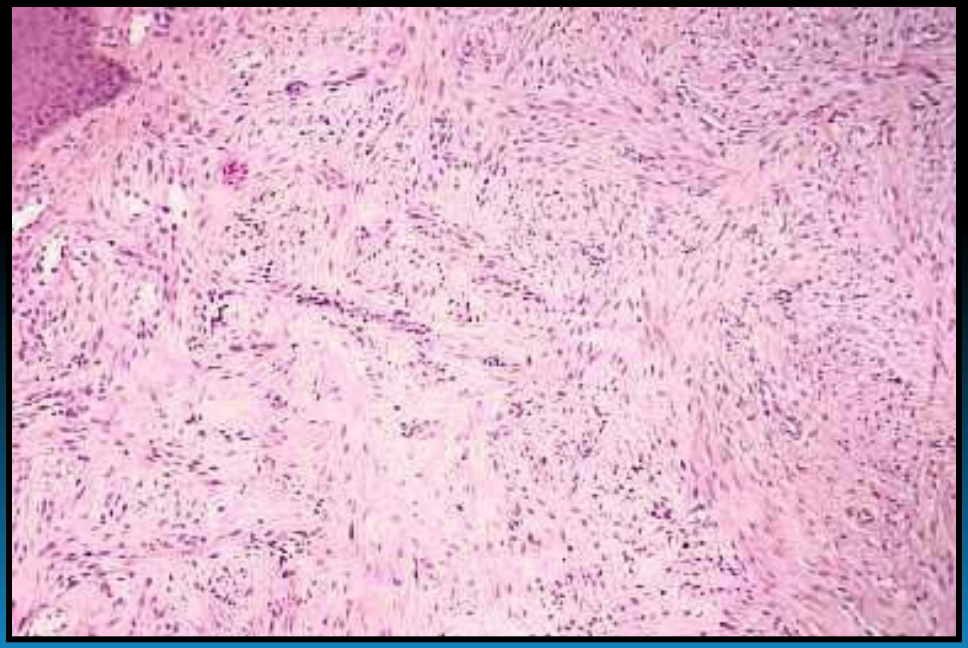
- Gingival fibromatosis shows dense or moderately dense, rather avascular, bland of collagenic connective tissue with scattered chronic inflammatory cells, especially beneath the surface epithelium.

- The attached gingival epithelium may have extreme elongation of rete processes.

- The crevicular epithelium facing the tooth surfaces usually shows considerable degeneration, subepithelial edema, and more extensive inflammatory cell infiltration because of the gingivitis or periodontitis

- When evaluating the inflammatory cell infiltrate, some cases of generalized gingival hyperplasia show focal collections of histiocytes intermixed with lymphocytes and foreign-body type multinucleated giant cells.





Treatment and Prognosis

- Gingival fibromatosis is removed by gingivectomy, with recurrences being treated in the same fashion or by more conservative removal of local areas of hyperplasia.
- Improved oral hygiene will greatly diminish the risk of recurrence.
-

VANDER WOUDE SYNDROME

- VWS is inherited as autosomal dominant pattern.
- Most cases of van der Woude syndrome have been linked to a deletion in chromosome 1q32-q41. however, a second chromosomal locus at 1p34 has also been identified
- About three percent of people with a cleft palate have VWS.
- Features of VWS include:

1. Mounds or depressions (pits) on the lower lip
2. Cleft lip with or without cleft palate
3. Missing teeth

- People who show signs of VWS can have one or more of these features. It is the lip pits or mounds that help set VWS apart from other types of cleft syndromes.

- Lip pits

  - Lower lip pits are fairly distinctive.

  - The pits are usually medial, often (but not always) on the vermilion portion of the lower lip.

  - They tend to be centered on small elevations in infancy but become simple depressions by adulthood;

- They are usually bilateral but are occasionally median or paramedian or unilateral (more commonly on the left side than on the right).
- Because they may be associated with accessory salivary glands, saliva, either visible or expressible, may be present in the lip pits
- Pits may lead to tracts that are surprisingly long, making surgical removal challenging.
- Lip pits may be the only manifestation of the syndrome.

Developmental Disturbances Affecting The Teeth

Classification

Size

Shape

Number

Structure

Classification

Intiation

Morpho – differentiation

Apposition

Congenital diseases

Malformation

A structural defect in the body due to abnormal embryonic or fetal development.

Deformation

A change from the normal size or shape of an anatomic structure due to mechanical forces that distort an otherwise normal structure

It is different from malformation – In malformation evelopment of a structure is arrested, delayed or misdirected in embryonic life

Congenital

A condition that is present at birth, whether or not it is inherited

Hereditary

Conditions capable of being transmitted from parent to offspring through genes

Anomaly

Deviation from the normal

Anomalad

A malformation together with its subsequently derived structure

Initiation

- Anodontia
- Accessory & supernumerary teeth
- Natal and neonatal teeth

Morpho-differentiation

- Microdontia
- Dilaceration
- Macrodontia
- Taurodontism
- Dens in Dente
- Enamel Pearl

- Fusion
- Supernumerary roots
- Gemination
- Concrescence
- Dens Evaginatus
- Hypercementosis
- Talon Cusp

Apposition

I. Enamel Hypoplasia

II. Amelogenesis Imperfecta

III. Dentinal Dysplasia

IV. Dentinogenesis Imperfecta

V. Regional Odontoplasia

Congenital disease

- Congenital syphilis

  Mulberry molars & Hutchinson's incisors

Size of the teeth

Developmental disturbances during the Bud stage

Microdontia

Macrodontia

Microdontia

- It means teeth that are smaller than normal
- Microdontia is a common condition usually associated with pituitary dwarfism
- Generalized or localized
- True or Relative
- Peg-laterals

Macrodontia

- It refers to teeth that are larger than normal
- Macrodontia is much less common than microdontia
- Macrodontia is not a common condition usually associated with pituitary gigantism
- Generalized or localized
- True or Relative

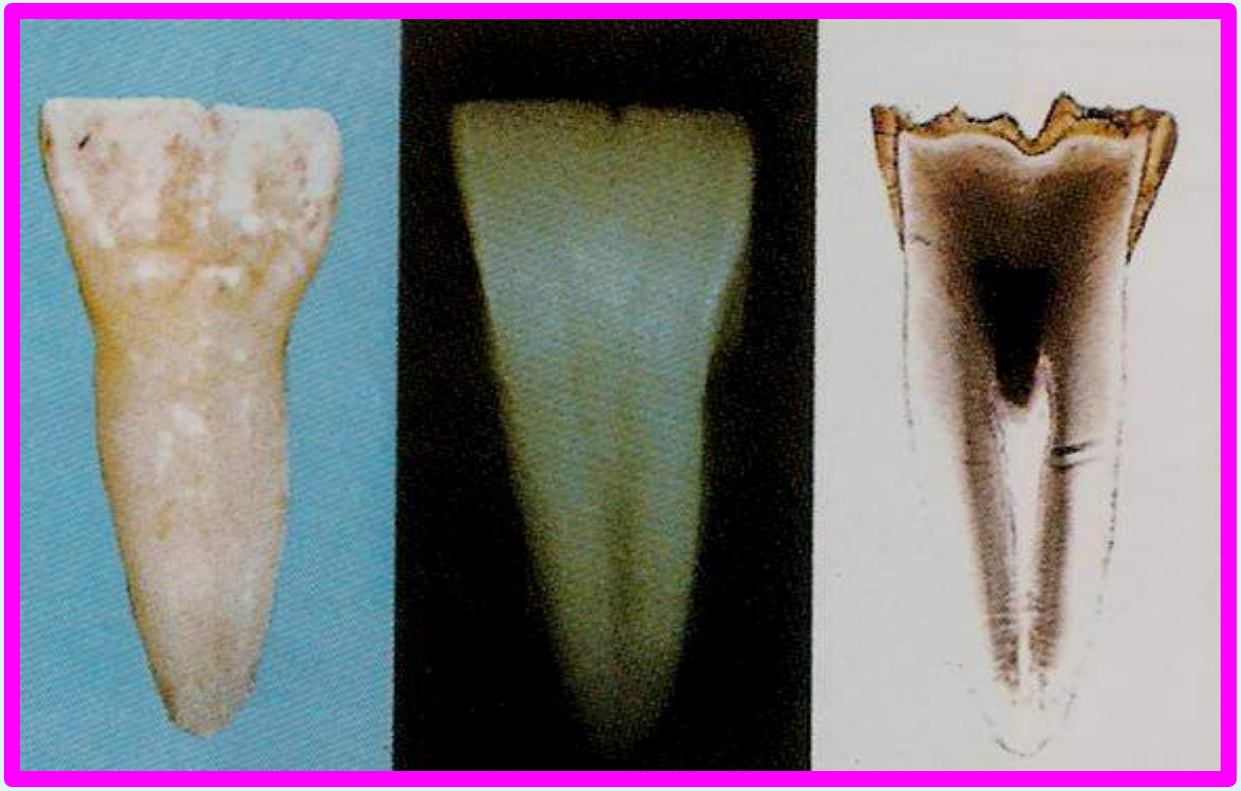
Shape of the teeth

- Gemination
- Fusion
- Concrescence
- Dilaceration
- Talon's cusp
- Dens in dente
- Dense evaginatus
- Taurodontism
- Supernumerary roots

Gemination

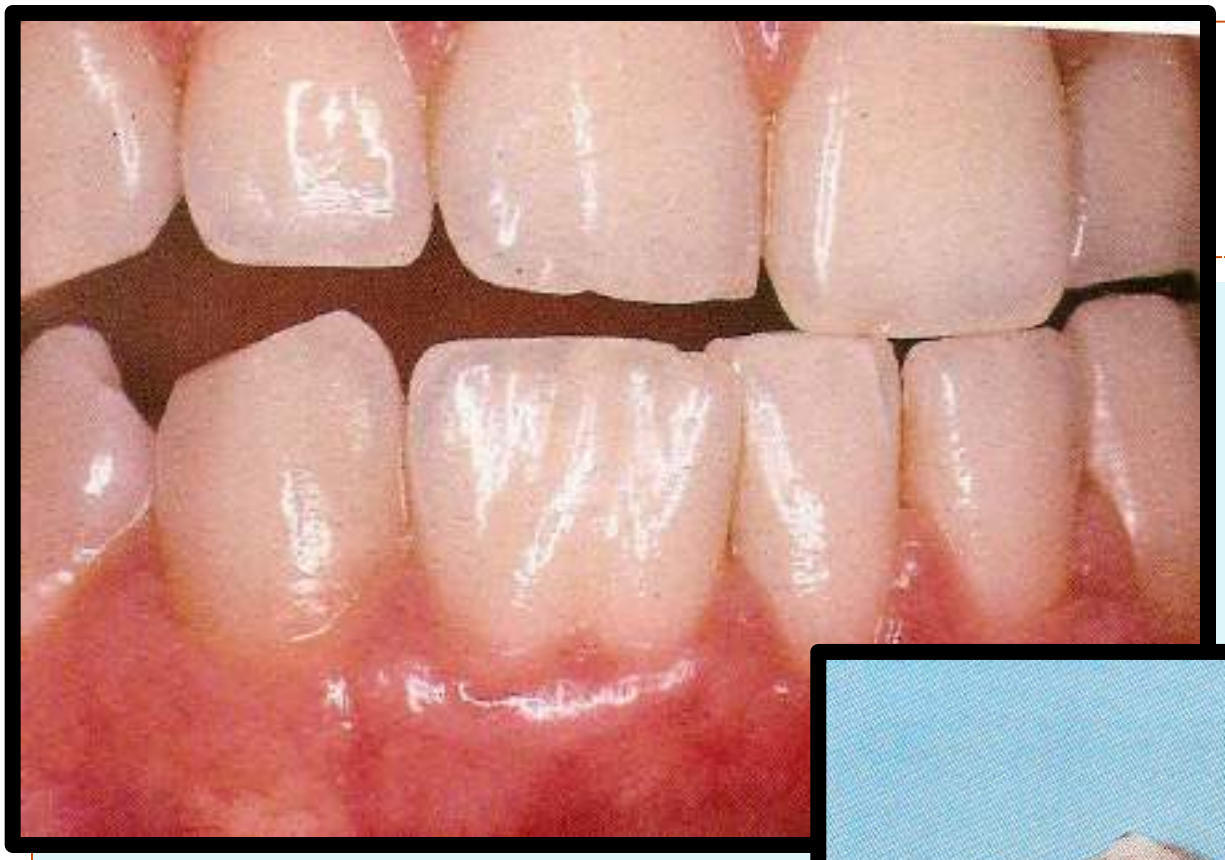
- Disturbances in cap stage, abnormal invagination of enamel organ into dental papilla
- Anomalies which arise from an attempt at division of single tooth germ by an invagination, resulting incomplete formation of two teeth
- Completely or incompletely separated two crowns having single root or root canal

- Occurs in both primary and permanent dentition
- Higher frequency in anterior and maxillary region
- Bifid crown may be seen overlying with root fused having one enlarged root with a single canal



Fusion

- Union of two normally separated tooth germs
- Fusion may involve the entire length of the teeth, or only the roots, depending on the stage of development of the teeth at the time of the union
- The root canal can be shared or separate
- Fusion is seen in the deciduous as well as in the permanent dentition
- It may be difficult or even impossible to differentiate fusion of supernumerary teeth from gemination



Concrescence

- Form of fusion occurring after the completion of root formation
- Teeth are united by only cementum

Dilaceration

Refers to abnormal angulation or sharp bend or curve, in the root of a formed tooth

Etiology-

Trauma during tooth formation resulting in change in position of calcified portion of tooth and remainder of tooth is formed at an angle

Clinical features

- Permanent maxillary incisors most commonly affected
- Curve may occur anywhere along the length of tooth
- Difficulty during extractions

Treatment

- No treatment is required
- Detection is of critical importance when Endodontic therapy or Extraction is undertaken

Talon's cusp

- It is a well differentiated additional cusp arising lingually or palatally from the cingulum areas of maxillary or mandibular permanent incisor
- Rarely in deciduous teeth
- Can be Unilateral or bilateral
- It is composed of normal enamel, dentin and contains horn of pulp tissue
- Seen associated with other anomalies like Supernumerary teeth, Odontomas, Impacted teeth, Dens invaginatus
- Rubinstein-Taybi syndrome and Sturge-Weber syndrome

Dens in dente

- Tooth within tooth
- Dense invaginatus
- **It arises as a result of invagination in the surface of tooth crown before calcification has occurred**

Etiology

- Increased localized external pressure
- Focal growth retardation and stimulation in certain areas of tooth bud

Clinical features

- Most commonly occurs in permanent lateral incisors, premolars, canines and molars

- The depth of invagination varies from slight enlargement of cingulum pit to extension into the apex

Types

- Coronal
- Radicular

- Type I
- Type II
- Type III

DENS EVAGINATUS

It is an accessory cusp or globule of enamel on the occlusal surface.

Clinical features

- Usually bilateral.
- Rarely in molars and cuspids.

TREATMENT

Dens Evaginatus- Results in occlusal problems and is prone to fracture resulting in pulp exposure.

Cuspal removal with direct pulp capping can be done.

Pulpal necrosis may result in cessation of root formation, so Apexification with calcium hydroxide is required to achieve closure.

Extraction may be occasionally recommended.

TAURODONTISM

It is an enlargement of the body and pulp chamber of a multi-rooted tooth with apical displacement of the pulpal floor and bifurcation of the roots.

Clinical and Radiographic Feature

- Affected teeth tend to be rectangular in shape and exhibit pulp chambers with increased Apico-occlusal height and a bifurcation close to the apex.

TREATMENT

- It may exhibit decreased strength and stability as an abutment
- No specific treatment is required.
- If endodontic treatment is required, the shape of the pulp chamber increases difficulty of locating, instrumenting and obturating the root canals.

Number

- Anodontia
- Supernumerary teeth
- Natal & neonatal teeth

Disturbances during initiation of tooth germs

- Anodontia
- Hypodontia
- Pseudo or false Anodontia

Anodontia

It is defined as condition in which there is congenital absence of teeth in the oral cavity.

It can be :

- Total or Complete Anodontia
- Partial Or Subtotal Anodontia
- Pseudo Anodontia
- False Anodontia

Complete or total Anodontia-

- It is rare condition in which there is neither deciduous or permanent tooth in the oral cavity
- It is seen associated with hereditary Ectodermal Dysplasia which is inherited as an x-linked recessive trait and primarily affects males, but an autosomal dominant form can affect females.
- It may sometimes occur among children who have received high doses of radiations which causes destruction of tooth germs resulting in complete lack of tooth formation in future

Partial Or Subtotal Anodontia

- Congenital absence of one or few teeth, this is also known as hypodontia or oligodontia.
- In this when a deciduous tooth is congenitally missing, it is very likely that its permanent successor will also be missing thereby suggesting some genetic influence.
- Third molars are most commonly missing teeth.
- Maxillary lateral incisors and mandibular premolars are next most commonly affected.
- In deciduous maxillary lateral incisors are most commonly missing.

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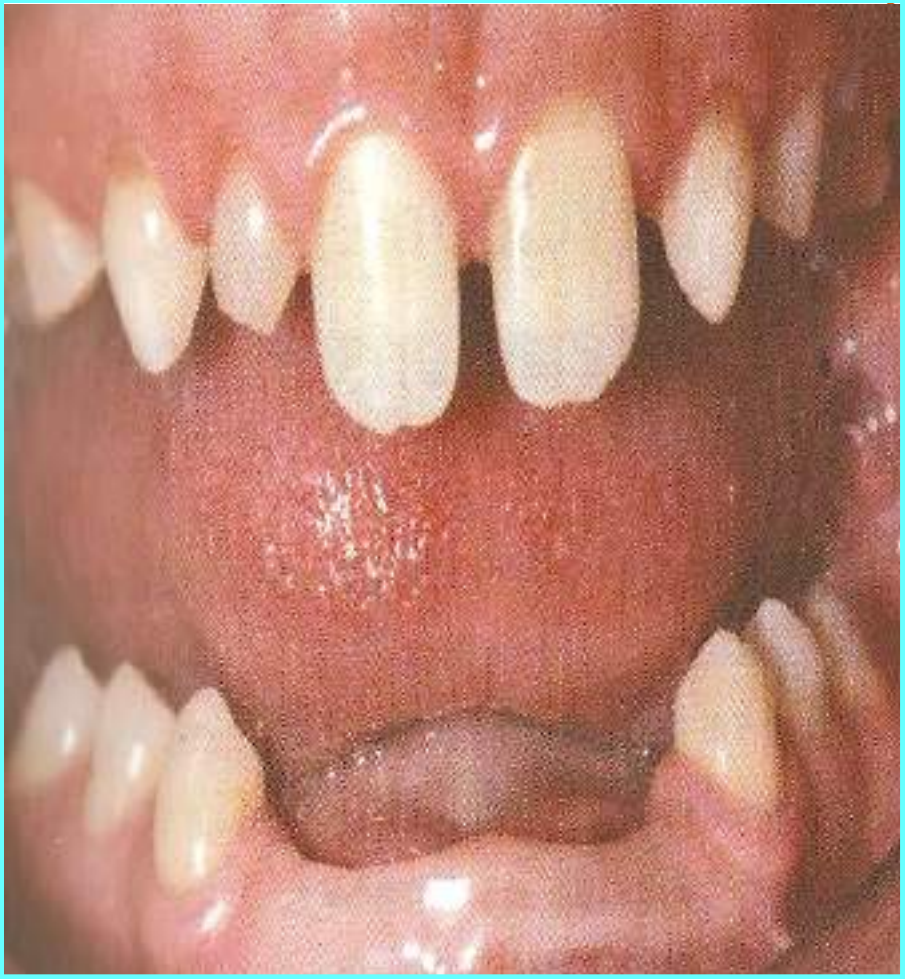

- Pseudo anodontia – It refers to condition in which teeth are present within the jaw bone but are not clinically visible in the mouth, as they have not erupted eg. impacted teeth or submerged teeth.
- False Anodontia – In this condition teeth are missing in the oral cavity because of their previous extractions or exfoliation.

Treatment

- Single or multiple missing tooth may be replaced by fixed prosthodontics or osteointegrated implants with associated prosthetic crowns.

SUPERNUMERARY TEETH

- Abnormal initiation may result in the development of one or more extra teeth.
- Dental hyperplasia may result in additional enamel organs being formed, resulting in development of teeth beyond the normal number (32 permanent teeth).
- Examples of supernumerary are:
  - Mesiodens (a tooth located between the permanent maxillary central incisors)
  - Fourth molar(paramolars)
  - Third mandibular premolars
  - Extra maxillary laterals

Treatment

- They may produce crowding or malocclusion and cause cosmetic problems.
- They may be directly or indirectly responsible for increase caries incidence and periodontal problems.
- Supernumerary teeth are mostly non functional and should be extracted.
- Impacted teeth should be surgically removed.

Structure of teeth

DEVELOPMENTAL DISTURBANCES

- Amelogenesis imperfecta
- Dentinogenesis imperfecta
- Dentin dysplasia
- Regional odontodysplasia
- Dentin hypocalcification

Amelogenesis imperfecta

- A structural defect of the tooth enamel
- Hereditary enamel dysplasia, hereditary brown enamel, hereditary brown opalescent dentin
- Clinical presentation and likely stage of enamel formation – HYPOPLASTIC,HYPOCALCIFIED,HYPOMATURATIVE

Hypoplastic TYPE I	Hypomaturaive TYPE II	Hypocalcified TYPE III	Hypomaturaton-hypoplastic with taurodontism TYPE IV
IA Hypoplastic,pitted autosomal dominant(AD)	IIA Hypomaturation, pigmented AR	III A AD	IV A hypomaturation-Hypoplastic with taurodontism
IB Hypoplastic, local , AD	IIB Hypomaturation , X linked recessive	IIIB AR	IV B Hypoplastic - hypomaturation with taurodontism
IC Hypoplastic, local, autosomal Recessive(AR)	IIC Snow capped teeth		
ID Hypoplastic, smooth,AD			
IE Hypoplastic , Smooth,,X linked Dominant			
IF Hypoplastic, Rough, AD			
IG Enamel agenesis,AR			

Clinical features

- Overlapping clinical features
- Type I –70%
- Type II – 20- 30 %
- Type III – 7%
- Absence or poorly formed enamel
- Occlussal and incisal wear and tear
- Increased inter-proximal space
- Decreased radiodensity
- Both the dentition are affected, generally only enamel is affected.

Hypoplastic

Hypocalcified

Hypomaturative

a
b
c
d
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Environmental enamel hypoplasia

- Incomplete or defective formation of enamel matrix of the teeth caused by environmental factors
- Both the dentitions are affected, single are multiple teeth involvement

- Both enamel and dentin are affected

- Factors capable of producing injuries to the ameloblasts are responsible

- Nutritional deficiency (vit A, C, D)
- Exanthematous diseases (Measels, chicken pox, scarlet fever)
- Congenital syphilis
- Birth injuries (prematurity, Rh Hemolytic diseases)
- Local infection Or Trauma
- Ingestion of chemicals (Flouride)

Clinical features

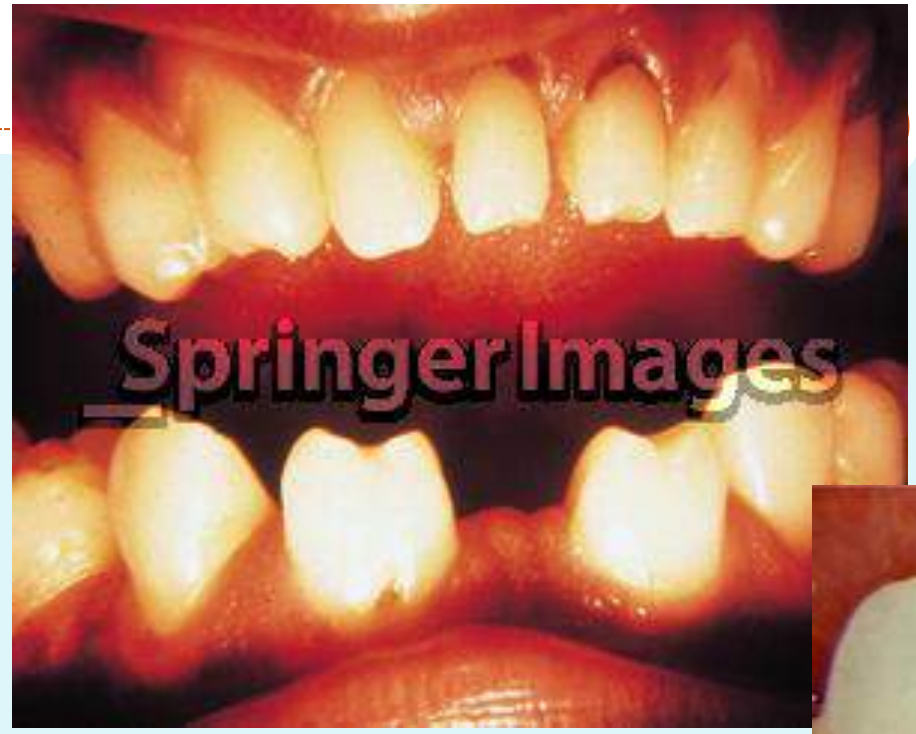
- Mild cases- few small grooves, fissures, and pits on the enamel surface
- Moderate cases – rows of deep pits arranged horizontally across the surface of the tooth. Single or multiple rows based on number of injuries
- Severe cases– considerable portion of the enamel may be absent

Nutritional deficiencies

- Vit A, C & D

Exanthematous fever

- Measles, scarlet fever, chicken pox, congenital syphilis
- Hypoplasia due to congenital syphilis – maxillary and mandibular permanent incissors, (Hutchinson's Incisors) and molars (mulberry molars, moon's molars)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EH due to local infection or trauma

- Localized
- Turners hypoplasia

EH due to fluoride

- Mottled enamel
- Dental fluorosis
- Ingestion of fluoride containing drinking water
- 0.9 – 1 part per million

Clinical features

- Questionable changes characterized by white flecking or spotting of the enamel
- Mild changes manifested by white opeque areas involving more of the tooth surface area
- Moderate and severe changes showing pitting and brownish staining of tooth surface
- Corroded appearance of the tooth

Dentinogenesis imperfecta

- Autosomal dominant condition affecting both deciduous and permanent dentition.
- Grey – yellowish brown colored teeth.
- Broad crown with constricted cervical area.
- Tulip shaped tooth.
- Chromosome 4 – dentin sailophospho protein

- Broken enamel with exposed dentin.
- Attrition of teeth
- Lack of pulp chamber in the tooth.

Classification

- Dentinogenesis imperfecta I
- Dentinogenesis imperfecta II (Brandywine type)

DI I

- Opalescent dentin
- Not associated with OI
- Teeth are amber- grey, or amber brown
- Bulbous crown, shorter & narrow roots, pulp chamber & root canals are smaller, sometimes completely obliterated

1x

DI II (Brandywine type)

- Brandywine triracial isolates in southern Maryland
- Affects both Dentitions
- Multiple pulp exposures after rapid wear and tear of teeth
- deciduous - - large pulp chambers, later reduces in size.
- Large pulp chamber, & thin enamel, dentin – shell teeth.

Histopathology

- Irregular dentinal tubules
- Large areas of uncalcified matrix
- Less in number but larger sized dentinal tubules
- Degenerating odontoblasts

Dentin dysplasia

- Rootless teeth
- Rushton 1939
- Hereditary , autosomal dominant pattern of inheritance
- Two types – Type I (radicular dentin dysplasia)
  Type II (coronal Dentin dysplasia)

Type I Radicular DD

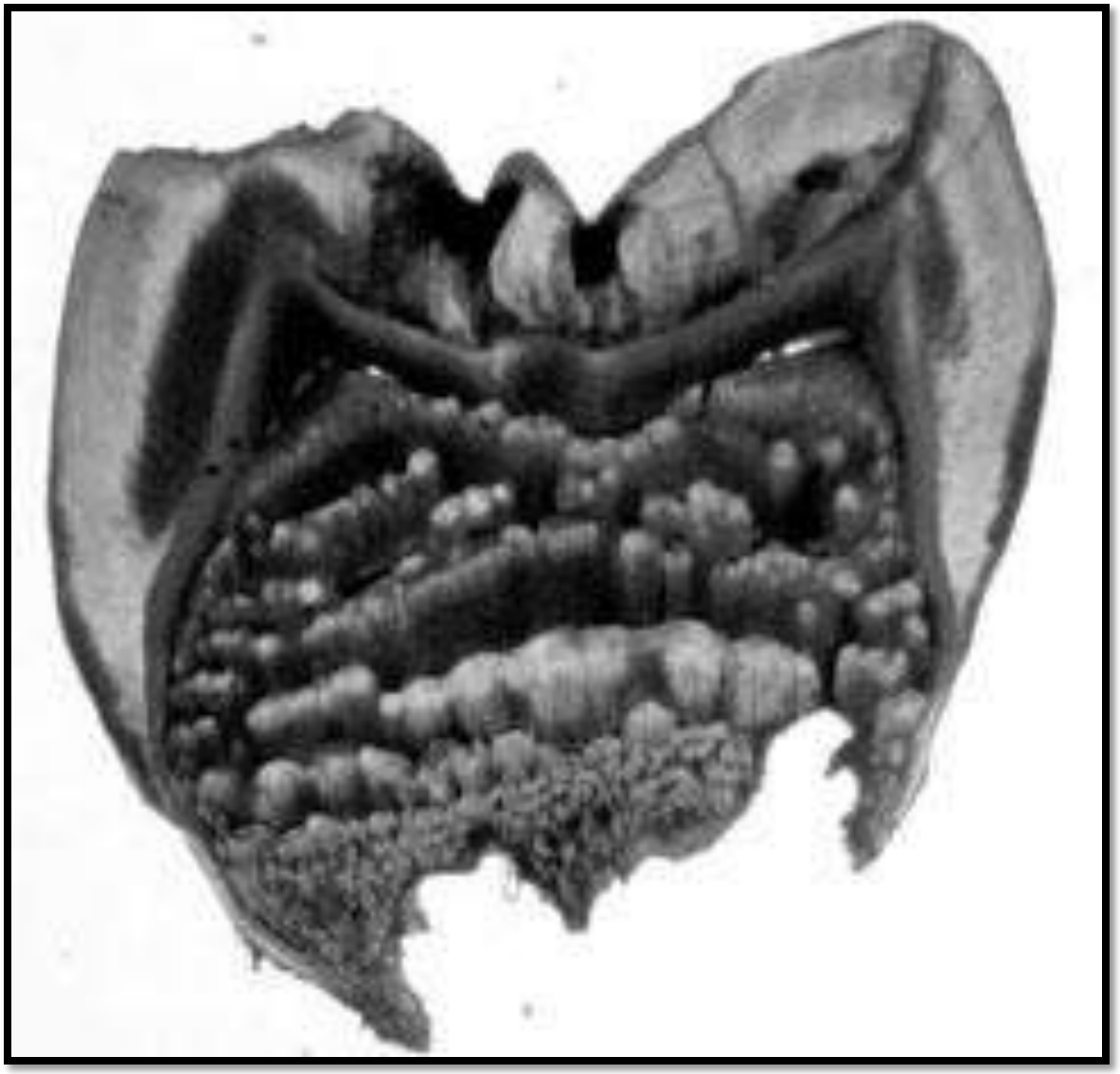
- Both dentitions are affected
- Teeth appear clinically normal in morphology and colour
- Slight amber translucency
- Extreme mobility of the teeth
- Premature loss of teeth after minor trauma
- Short roots

Radiographic features

- Roots are short and blunt
- Malformed roots
- Pulp chamber and canals are completely obliterated
- Crescent shaped pulpal remnant may be seen

Histopathology

- Part of coronal dentin – normal
- Apical dentin shows complete obliteration of canals
- Pulpal calcification
- Lava flowing around the boulders – normal dentinal tubule formation appears to be blocked so that new dentin formation around the obstacles



Type II Coronal DD

- Both dentitions are affected
- Involvement of deciduous common
- Yellow brown, bluish grey opalescent teeth
- Permanent appears normal clinically
- Large pulp chamber in coronal portion of the tooth – thistle tube appearance
- Calcifications in pulp

Histopathology

- Multiple pulp stones
- Amorphous a tubular dentin

Regional odontodysplasia

- Ghost teeth
- Unusual dental anomaly in which one or several teeth in a localized area are affected.
- Max > mand
- Permanent central, lateral, cuspids
- Both dentitions are affected
- Etiology – unknown, trauma or systemic illness, vascular defects

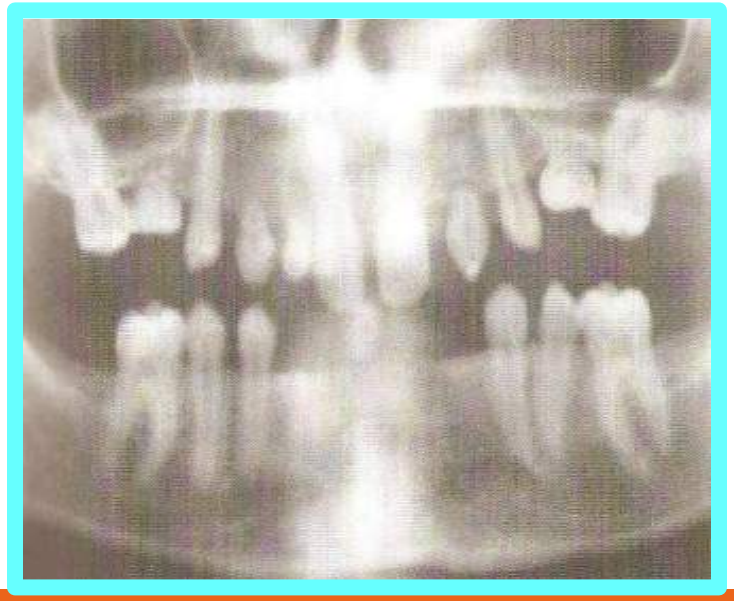
Clinical features

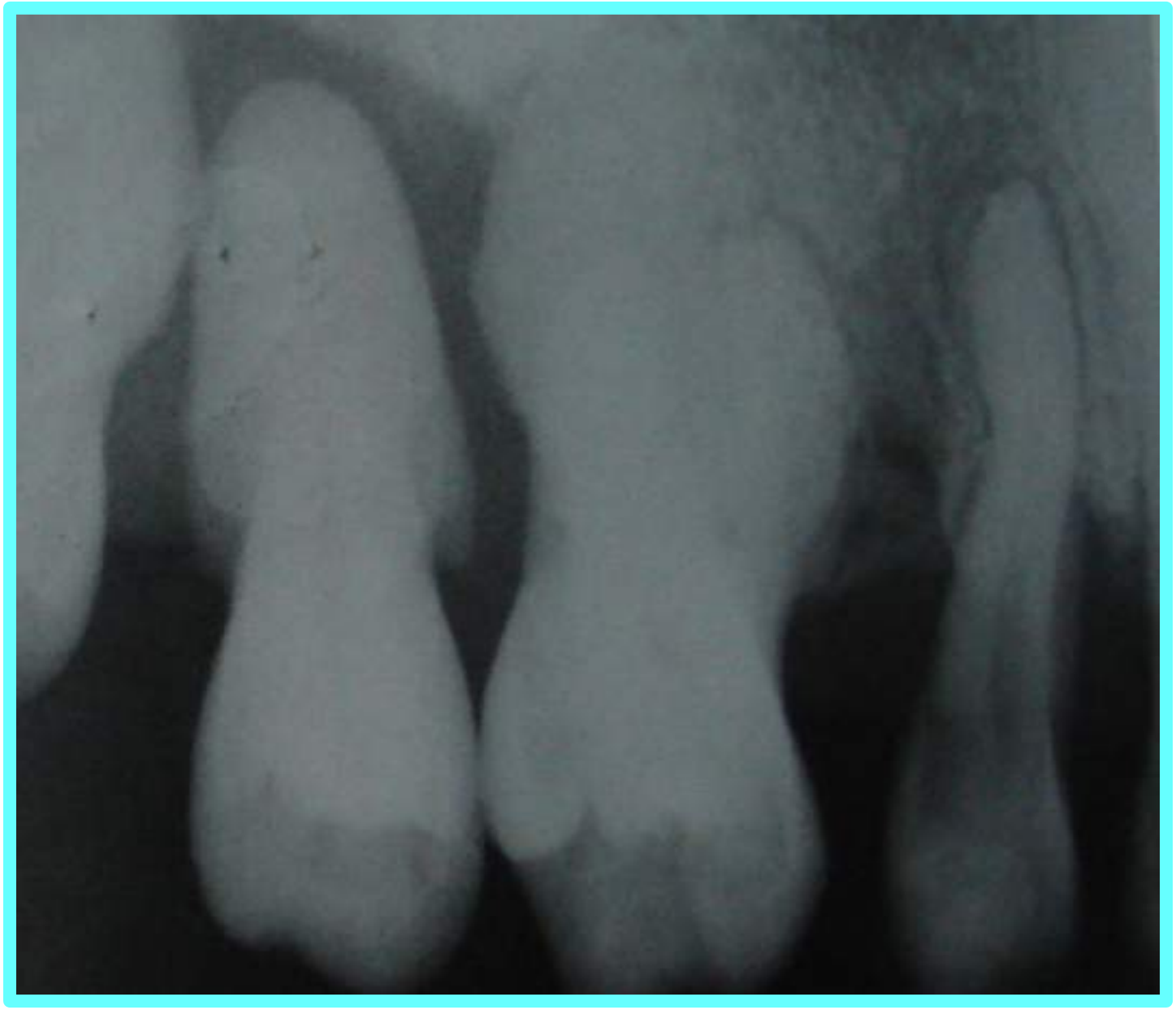
- Delay or total failure in the eruption
- Marked alteration in the shape, very irregular in appearance
- Defective mineralization
- Radiographic features – marked reduction in the radio density of the enamel and dentine leading to thin enamel and dentine and large Pulp chambers – GHOST TEETH

IN

Histopathology

- Marked reduction in the amount of dentine, the widening of pre-dentine layer
- Presence of large areas of inter-globular dentine
- REE around the non erupted teeth, shows numerous calcifications





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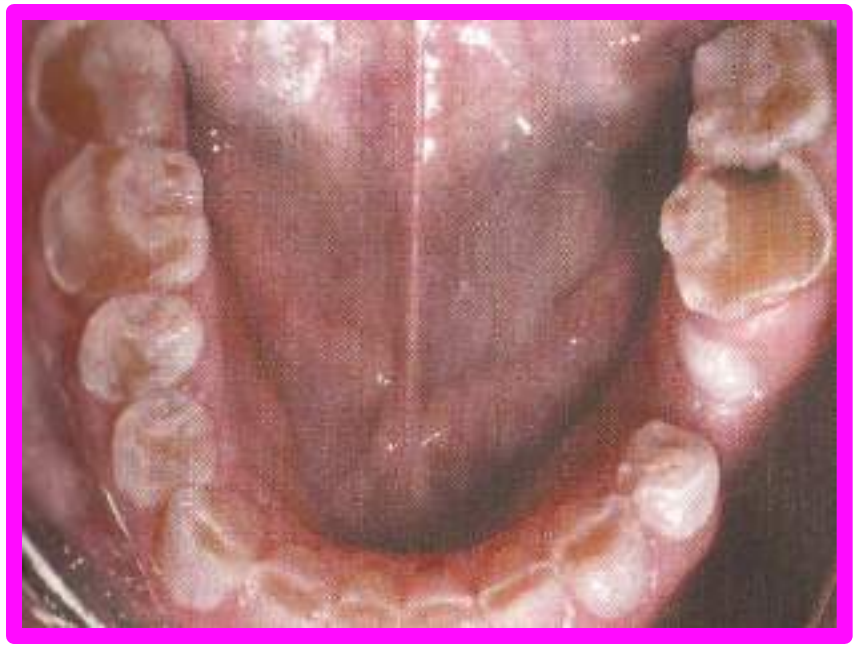

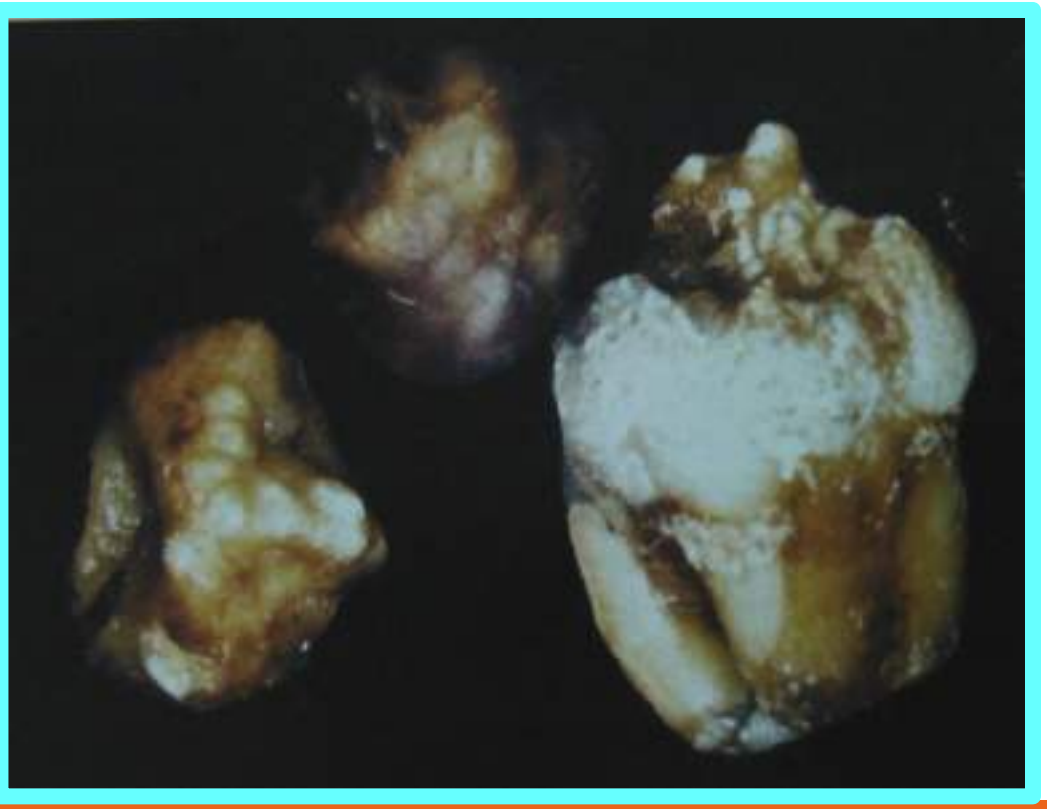

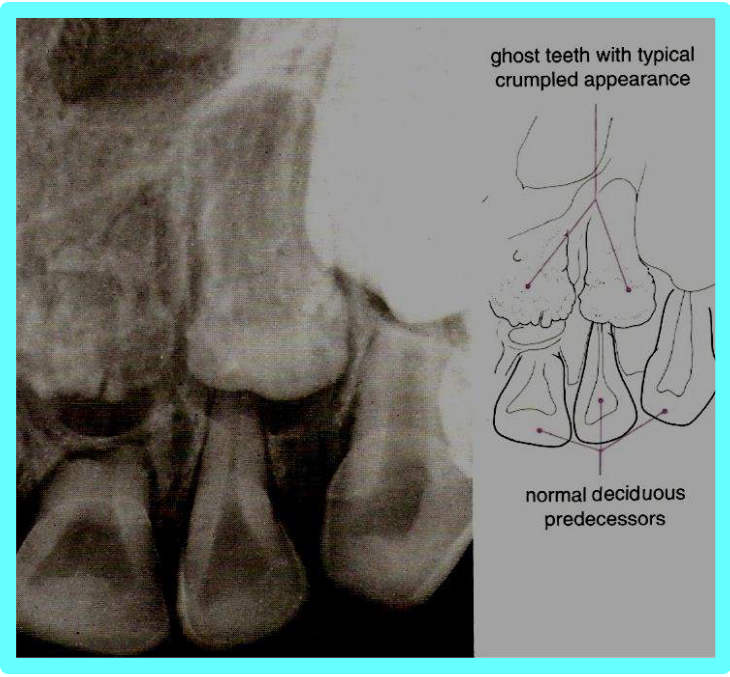
ghost teeth with typical crumpled appearance
normal deciduous predecessors

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DEVELOPMENTAL DISORDERS OF LIPS

- Congenital lip pit
- Commissural lip pit and fistulas
- Double lip
- Cheleilitis glandularis
- Cheilitis granulomatosa
- Labial and oral melanotic macule
- Cleft lip & palate
- Peutz – jeghers syndrome
- Melkersson – rossenthal syndrome
- Van Der Woude syndrome

CONGENITAL LIP PITS

- *Para median lip pits or Congenital invagination of the lower lip.*
- Believed to arise from persistent **lateral sulci** on embryonic mandibular arches. (usually disappears by 6th week)
- 1-1.5 cm in depth, occasionally single, occurs at the center of the lip.
- Van Der Woude syndrome.
- Popliteal pterygium syndrome

HISTOPATHOLOGY

- Narrow invagination of the surface epithelium lined by stratified squamous epithelium.
- Connective tissue reveals minor salivary gland ducts draining into the sulcus.

TREATMENT

- No specific treatment. Surgical excision is considered

VAN DER WOUDE SYNDROME

- ✓ Mounds or depressions (pits) on the lower lip
- ✓ Cleft lip with or without cleft palate
- ✓ Missing teeth
- Inherited as autosomal dominant pattern.
- linked to a deletion in chromosome 1q32-q41. however, a second chromosomal locus at 1p34 has also been identified.
- 3 % of people with a cleft palate have VWS.

COMMISSURAL LIP PITS

- Small mucosal invaginations that occur at the corners of the mouth on the vermilion border.
- They may represents a failure of normal fusion of the embryonal maxillary and mandibular process.

DOUBLE LIP

- Rare developmental anomaly characterized by redundant fold of tissue on the mucosal side of the lip.

Etiopathogenesis:

- During fetal development the upper lip, mucosa consists of two transverse zones ,an ***outer zone***, which is smooth and similar to skin, (pars glabra), & ***inner zone***, which is villous and similar to the oral mucosa,(pars villosa).
- In the double lip buccal villous part becomes hypertrophic.

- Upper lip > lower lip
- Though the deformity can occur at birth, it usually becomes evident after the eruption of the permanent teeth.

ACQUIRED DOUBLE LIP

- Develops after a trauma to lips.
- Associated with Ascher ‘s syndrome.
  Double lip, Blephrocholasis, Non toxic thyroid enlargement.

CHELITIS GRANULOMATOSA

- Cheilitis granulomatosa is a chronic swelling of the lip due to granulomatous inflammation.
- Miescher cheilitis, melkerson rosenthel syndrome(Facial palsy & fissured tongue)
- In granulomatous cheilitis, normal lip architecture is eventually altered by the presence of lymphoedema and noncaseating granulomas in the lamina propria
- Episodic with nontender swelling and enlargement of one or both lips.
- Upper lip > Lower lip
- Soft, firm, or nodular on palpation.

- Earliest manifestation is sudden diffuse or occasionally nodular swellings of the lip or the face.
- First episode typically subsides completely in hours or days. After recurrent attacks, swelling may persist and slowly increase in degree, eventually becoming permanent.
- Enlarged lip appears cracked and fissured with reddish brown discoloration and scaling.
- Fissured lip becomes painful and eventually acquires the consistency of firm rubber.
- Swelling may regress very slowly after some years.
- Regional lymph nodes are enlarged (usually minimally) in 50% of patients.
- Attacks sometimes are accompanied by fever and mild constitutional symptoms (eg, headache, visual disturbance).

Histopathology

- Chronic inflammatory cell infiltrate, peri & para vascular aggregations of lymphocytes, plasma cells and Histiocytes
- The infiltrate becomes denser and pleomorphic, and small focal granulomas are formed that are indistinguishable from Crohn disease or sarcoidosis.
- Small granulomas occur in the lymphatic walls in some cases. Similar changes may be present in cervical lymph nodes.

CHEILITIS GLANDULARIS

- Actinic cheilitis.
- It is a clinical diagnosis that refers to an un common and poorly understood inflammatory disorder of the lip.
- The condition is characterized by progressive enlargement and eversion of the lower labial mucosa that results in obliteration of the mucosal-vermilion interface.
- Potential pre disposing factors for the development of actinic cheilitis and squamous cell carcinoma

CLINICAL FEATURES

- Enlargement and the loss of elasticity of the lip with pain.
- Initially asymptomatic, lip swelling occurs with clear viscous secretion expressible from dilated ductal openings on the mucosal surface.
- Periods of relative quiescence interrupted by transient or persistent painful episodes associated with suppurative discharge.
- A burning discomfort or a sensation of rawness referable to the vermilion border may be reported.

- This is associated with atrophy, speckled leukoplakic change, erosion, or frank ulceration with crusting.
- Affects the lower lip almost exclusively. It manifests as progressive, often multinodular enlargement, eversion, and induration.
- Salivary gland duct orifices may be dilated and appear as red or black.

Histopathology

- Epithelium can be essentially normal or show evidence of disturbance that ranges from varying degrees of atypia or dysplasia to frank carcinoma.
- The severity of epithelial maturational disturbance tends to correspond with the degree of risk for progression to carcinoma.
- Minor salivary glands show changes that include atrophy or distention of acini, with or without squamous metaplasia.
- Chronic inflammatory infiltration and replacement of glandular parenchyma, and interstitial fibrosis.
- Other possible histologic findings include stromal edema, hyperemia, surface hyperkeratosis, erosion, or ulceration

DEVELOPMENTAL DISORDERS OF SALIVARY GLANDS

- Aplasia (agenesis)
- Atresia
- Aberency
- Hyperplasia of salivary gland.
- Developmental lingual mandibular salivary gland depression.

APLASIA (AGENESIS)

- Aplasia of the salivary glands is an occasional finding.
- Any one or group of salivary glands may be absent, unilaterally or bilaterally.
- Aplasia becomes manifested with the development of xerostomia.
- A diagnosis of salivary gland aplasia is made after exclusion of the common causes of xerostomia - medications, sjogren's syndrome and radiation.

- The CT scan or MRI will indicate the gland's absence and its replacement by fat and fibrous tissue.
- Scinti-scanning with a radioisotope will confirm the initial diagnosis.
- The absence of the salivary duct orifice / papilla is an additional clue.
- Aplasia occurs for unknown reasons as an isolated finding or in conjunction with other developmental defects such as

Hemifacial microsomia,
LADD syndrome and
Mandibulo-facial dysostosis (Treacher Collins).

ATRESIA

- An abnormality of salivary gland duct.
- Congenital occlusion or absence of one or more of major salivary gland ducts.
- Exceedingly rare condition.
- Atresia of the duct can be diagnosed by use of contrast sialography.

ABERRANCY

- Salivary gland tissue may arise in various aberrent locations within the head and neck.
- The most common site is in periparotid lymph nodes.
- Other sites include the lower neck, sternoclavicular joint, thyroglossal duct, middle ear, tonsils, hypopharynx, and mandible.
- This aberrant tissue may be located within or on the surface of the mandible.
- If located within the mandible, may be associated with the development of a malignant neoplasm (most commonly mucoepidermoid carcinoma), while that which is on the surface usually represents benign salivary gland tissue.

LINGUAL MANDIBULAR SALIVARY GLAND DEPRESSION

- **Stafne's Cyst**
- **Stafne's Bone Defect**
- **Static Bone Cyst**
- **Latent Bone Cyst**
- **ETIOLOGY** – unknown
- A developmental concavity of the lingual cortex of the mandible, usually in the third molar area, caused by overextension of an accessory lateral lobe of the submandibular gland.

DEVELOPMENTAL DISTURBANCES OF BUCCAL MUCOSA

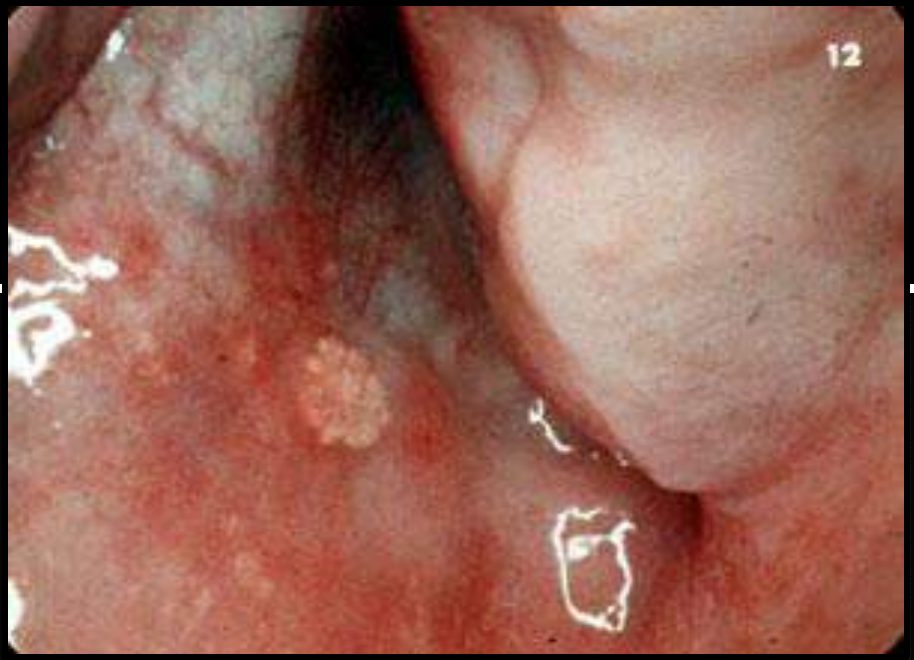
- Fordyce Granules
- Leukoedema
- Focal Epithelial Hyperplasia

FORDYCE GRANULES

- These are sebaceous glands that occur in the oral mucosa.
- These have been reported in 80% of the population, so their presence is considered as a normal anatomic variation.

CLINICAL FEATURES :

- Present as a multiple yellow or yellowish-white papular lesions.
- Buccal mucosa & lateral portion of the vermilion border of upper lip are commonly involved.
- Adults > children.
- Asymptomatic, few to hundred in number.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LEUKOEDEMA

- Common oral mucosal condition of unknown cause.
- Leukoedema is an anatomic variation that represents hydration of the buccal mucosal epithelium and requires no treatment.
- It is a type of normal oral mucosa rather than a disease.
- Although, some studies pointed towards the developmental origin and some smoking, explanation is not satisfactory.

CLINICAL FEATURES

- Diffuse grayish – white milky opalescent appearance of the mucosa.
- The surface appears folded, resulting in wrinkles.
- Lesions do not rub off.
- Lesions diminishes or disappears when the cheek is stretched.

HISTOPATHOLOGY

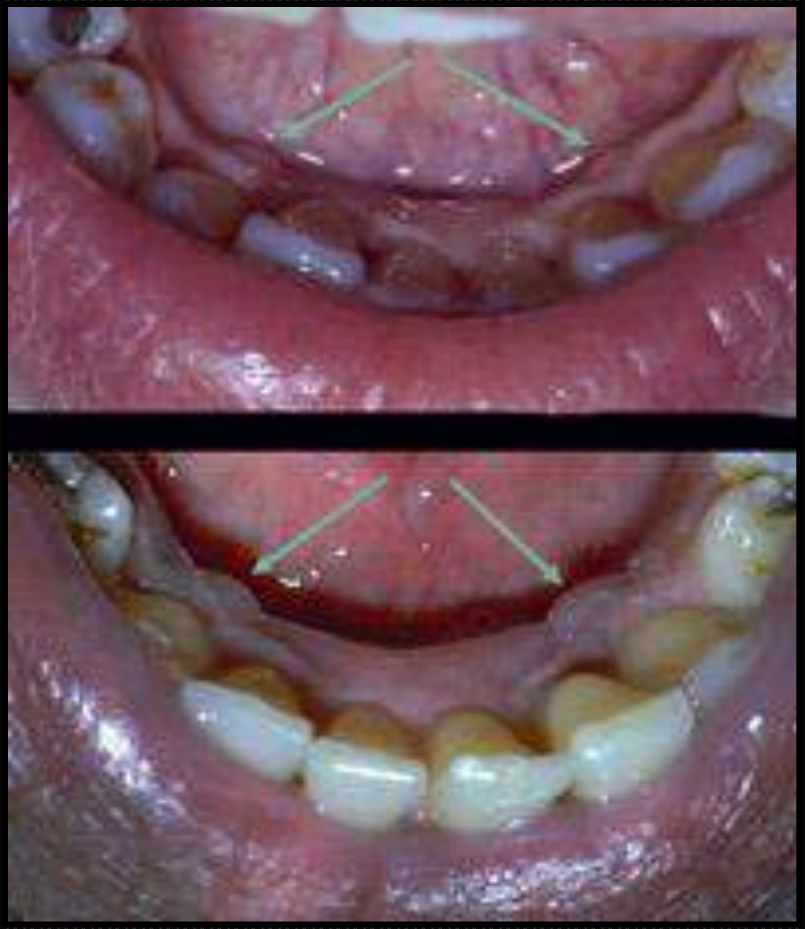
- Demonstrate increase in the thickness of the epithelium with striking intracellular edema of the spinous layer.
- Epithelial surface is usually Para keratinized & rete ridges are broad and elongated.
- Vacuolated epithelial cells show pyknotic nuclei.

DEVELOPMENTAL DISTURBANCES OF GINGIVA

- Retro cuspid Papillae
- Hereditary Gingival Fibromatosis

RETROCUSPID PAPILLAE

- Normal structures seen particularly in the younger age groups.
- They are found on the attached gingivae of the mandible, often bilaterally, lingual to the cuspid.
- Raised, nodules, usually 0.5 mm, with a broad base (sessile) or a pedunculated base (emerging from a stalk).
- They may regress with age, may be mistaken for disease but require no treatment.

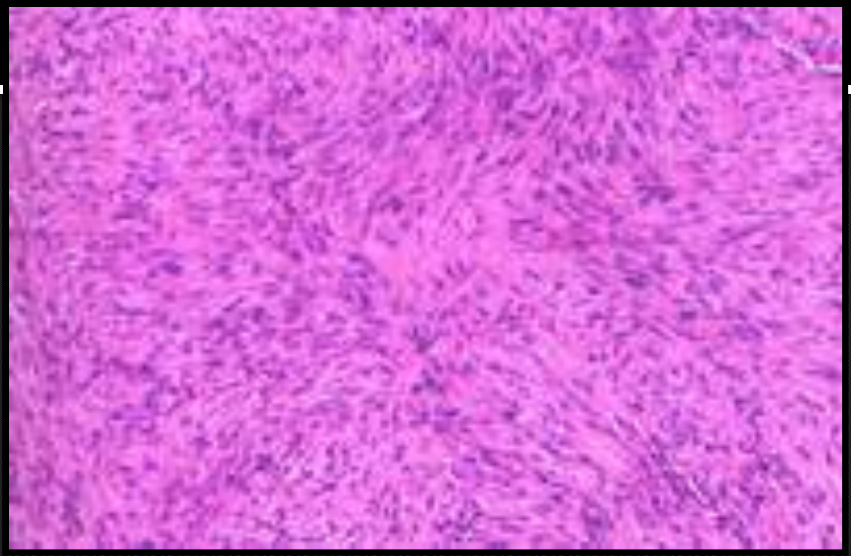


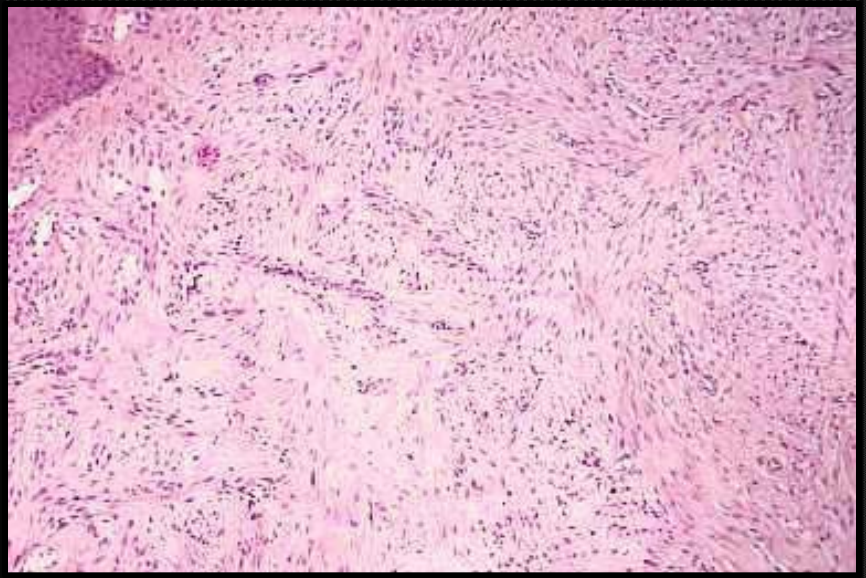
HEREDITARY GINGIVAL FIBROMATOSIS

- Idiopathic gingival fibromatosis.
- The term Gingival Fibromatosis is used for those, which have an hereditary pattern or part of a more extensive syndrome.
- Localized & Generalized.
- Symmetrical fibromatosis of the tuberosity is a localized variant of gingival fibromatosis. This form is more common than generalized gingival fibromatosis.
- Appears to be a developmental type of phenomenon, although its true etiology is unknown.

Histopathology

- Gingival fibromatosis shows dense or moderately dense, rather avascular, bland of collagenic connective tissue with scattered chronic inflammatory cells, especially beneath the surface epithelium.
- The attached gingival epithelium may have extreme elongation of rete processes.





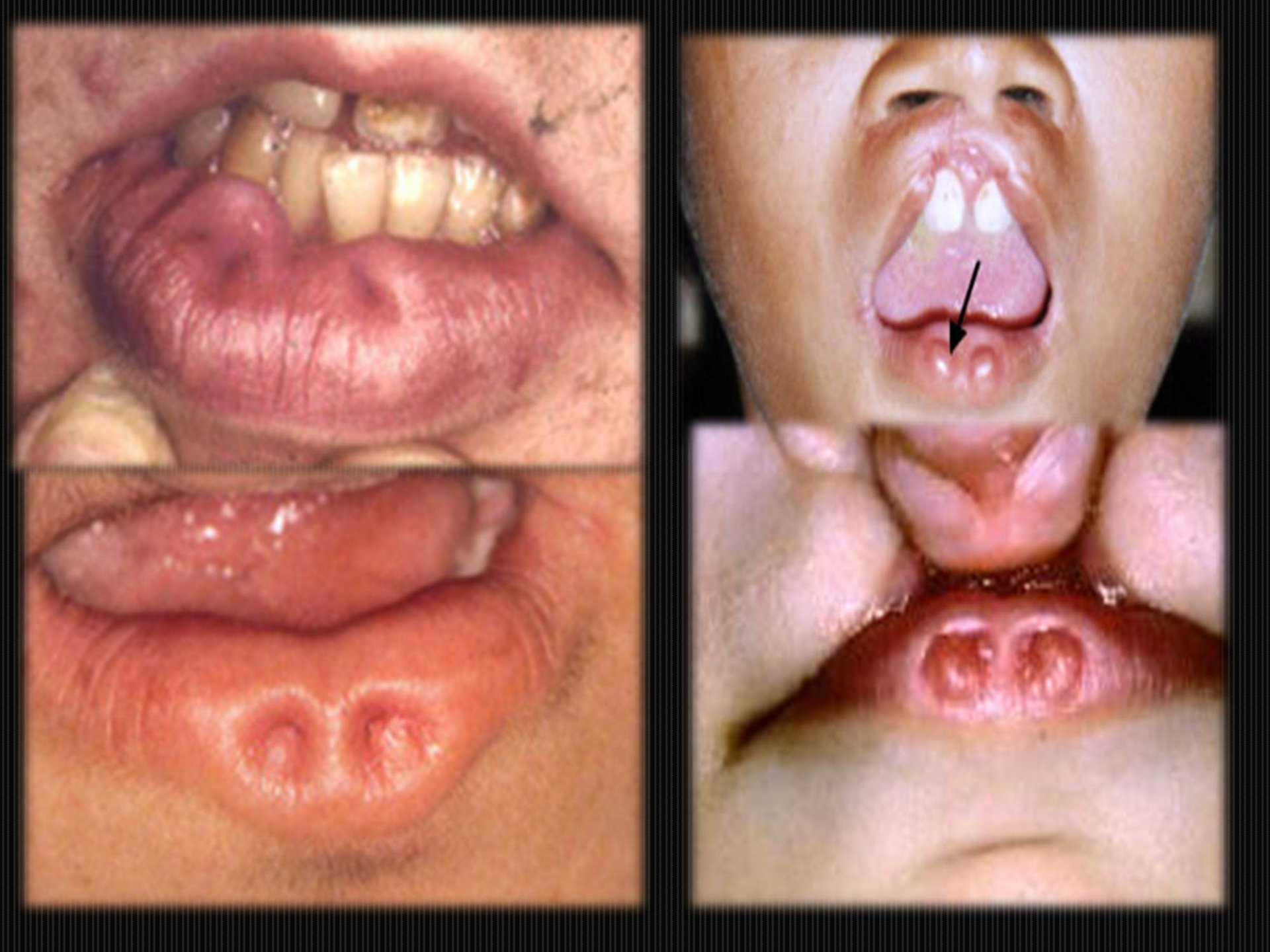
VANDER WOUDE SYNDROME

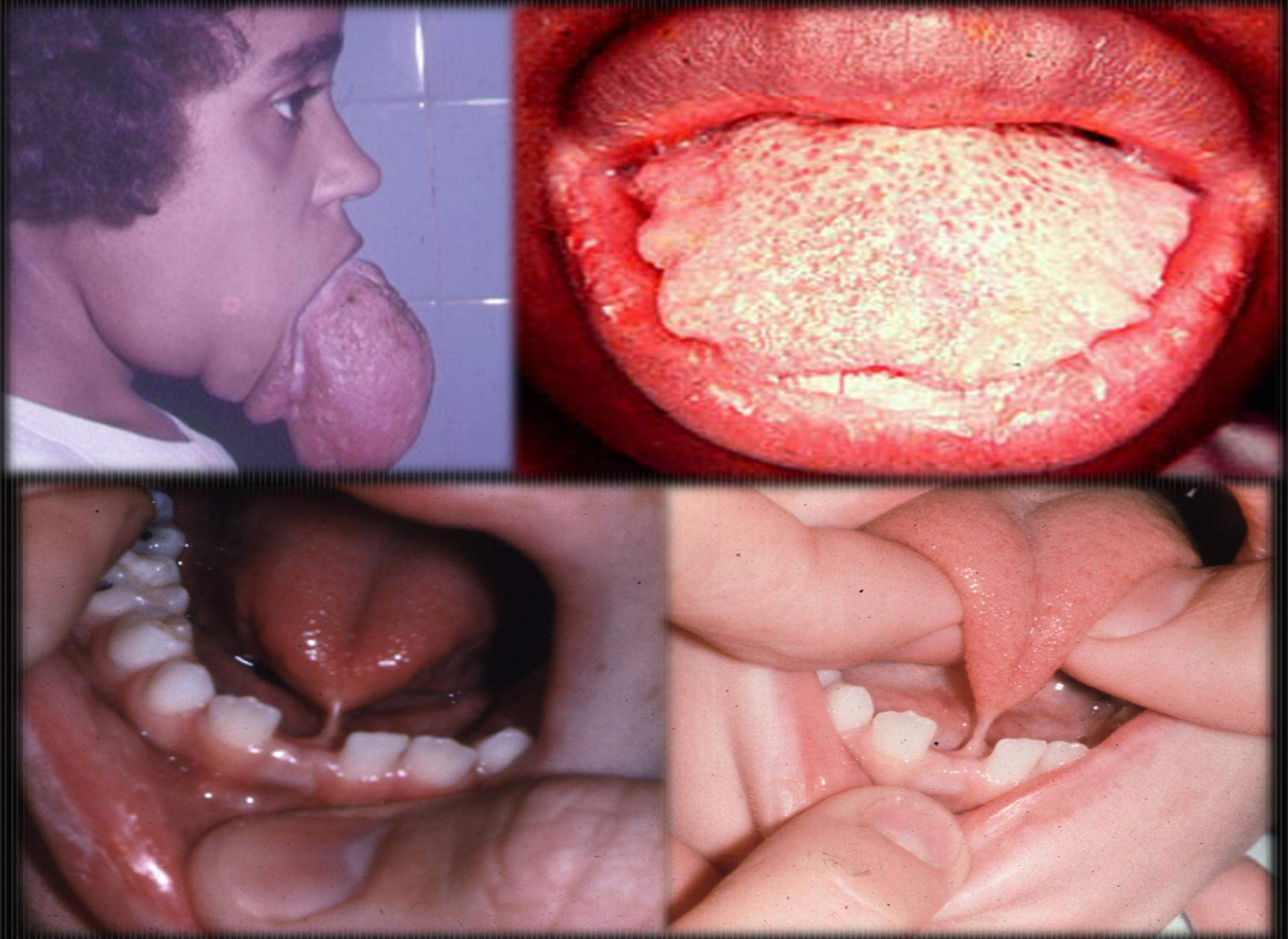
- VWS is inherited as autosomal dominant pattern.
- Most cases of van der Woude syndrome have been linked to a deletion in chromosome 1q32-q41. however, a second chromosomal locus at 1p34 has also been identified
- About three percent of people with a cleft palate have VWS.
- Features of VWS include:

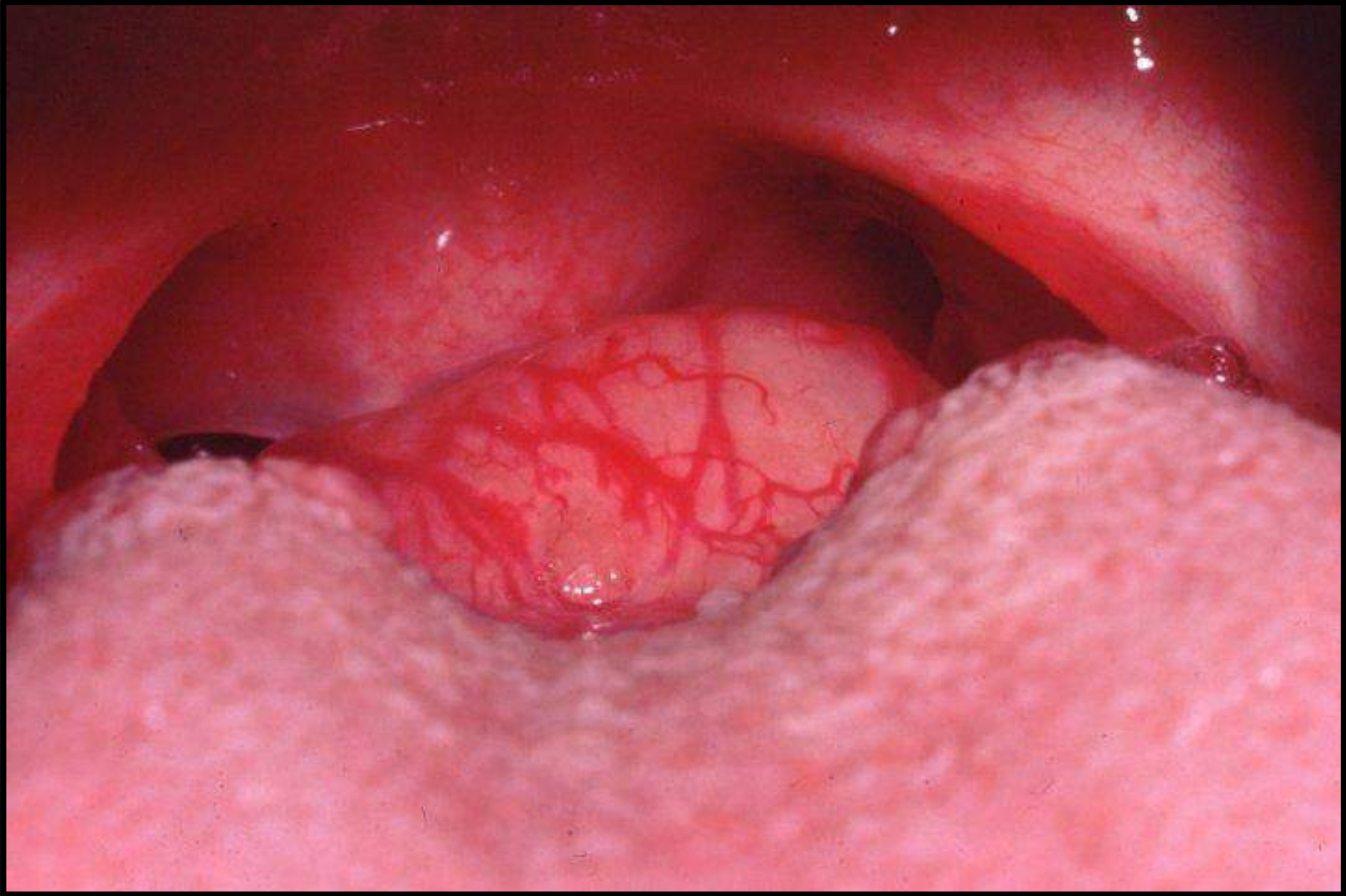
1. Mounds or depressions (pits) on the lower lip
2. Cleft lip with or without cleft palate
3. Missing teeth

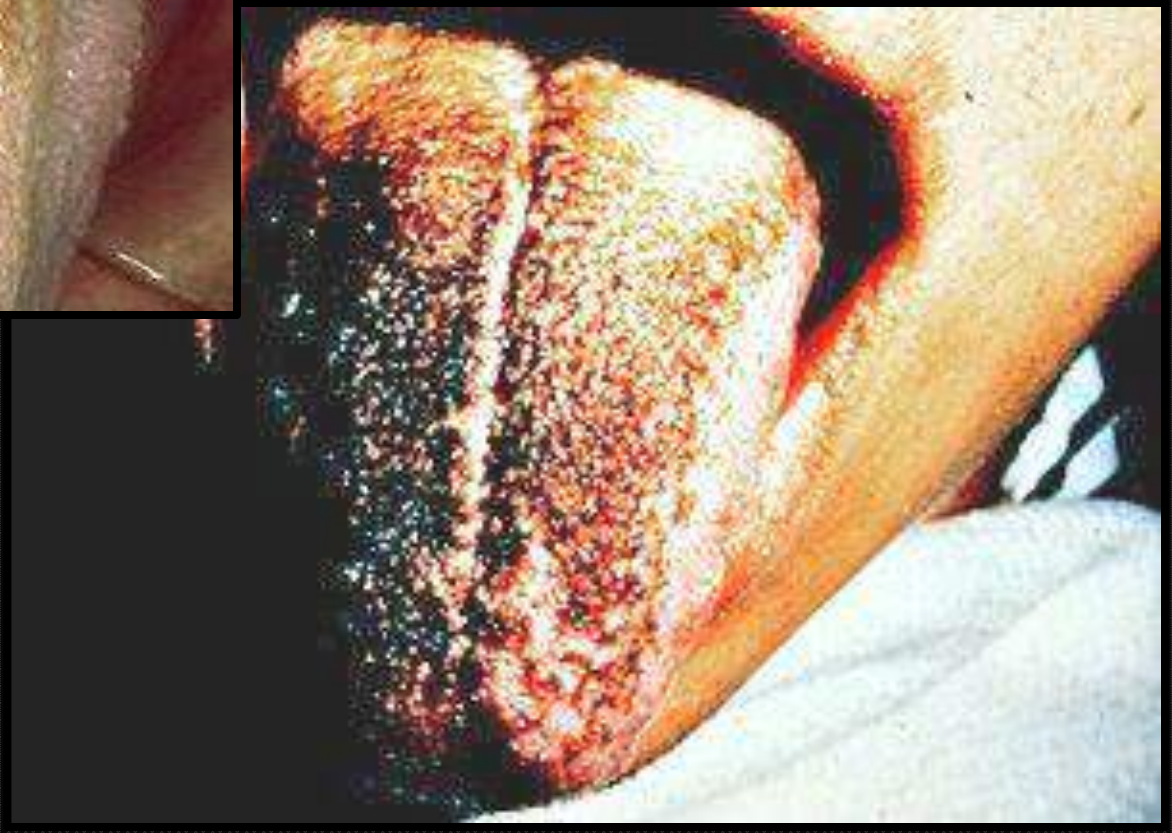
- People who show signs of VWS can have one or more of these features. It is the lip pits or mounds that help set VWS apart from other types of cleft syndromes.
- Lip pits
  - Lower lip pits are fairly distinctive.
  - The pits are usually medial, often (but not always) on the vermilion portion of the lower lip.
  - They tend to be centered on small elevations in infancy but become simple depressions by adulthood;

- They are usually bilateral but are occasionally median or paramedian or unilateral (more commonly on the left side than on the right).
- Because they may be associated with accessory salivary glands, saliva, either visible or expressible, may be present in the lip pits
- Pits may lead to tracts that are surprisingly long, making surgical removal challenging.
- Lip pits may be the only manifestation of the syndrome.









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DEVELOPMENTAL DISTURBANCES AFFECTING THE TEETH

Classification

- Intiation
- Morpho – differentiation
- Apposition
- Congenital diseases

Initiation

- Anodontia
- Accessory & supernumerary teeth
- Natal and neonatal teeth

Morpho-differentiation

- Microdontia
- Dilaceration
- Macrodontia
- Taurodontism
- Dens in Dente
- Enamel Pearl

- Fusion
- Supernumerary roots
- Gemination
- Concrescence
- Dens Evaginatus
- Hypercementosis
- Talon Cusp

Apposition

I. Enamel Hypoplasia
II. Amelogenesis Imperfecta
III. Dentinal Dysplasia
IV. Dentinogenesis Imperfecta
V. Regional Odontoplasia

Congenital disease

- Congenital syphilis

  Mulberry molars & Hutchinson's incisors

Classification

- Size
- Shape
- Number
- Structure

Developmental disturbances

STRUCTURE OF TEETH

- Amelogenesis imperfecta
- Dentinogenesis imperfecta
- Dentin dysplasia
- Regional odontodysplasia
- Dentin hypocalcification

Amelogenesis imperfecta

- A structural defect of the tooth enamel
- Hereditary enamel dysplasia, hereditary brown enamel, hereditary brown opalescent dentin
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- Incomplete or defective formation of enamel matrix of the teeth caused by environmental factors
- Both the dentitions are affected, single are multiple teeth involvement

- Both enamel and dentin are affected

- Factors capable of producing injuries to the ameloblasts are responsible

- Nutritional deficiency (vit A, C, D)
- Exanthematous diseases (Measels, chicken pox, scarlet fever)
- Congenital syphilis
- Birth injuries (prematurity, Rh Hemolytic diseases)
- Local infection Or Trauma
- Ingestion of chemicals (Flouride)

Clinical features

- Mild cases- few small grooves, fissures, and pits on the enamel surface
- Moderate cases – rows of deep pits arranged horizontally across the surface of the tooth. Single or multiple rows based on number of injuries
- Severe cases– considerable portion of the enamel may be absent

Nutritional deficiencies

- Vit A, C & D

Exanthematous fever

- Measles, scarlet fever, chicken pox, congenital syphilis
- Hypoplasia due to congenital syphilis – maxillary and mandibular permanent incissors, (Hutchinson's Incisors) and molars (mulberry molars, moon's molars)

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EH due to fluoride

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- Questionable changes characterized by white flecking or spotting of the enamel
- Mild changes manifested by white opeque areas involving more of the tooth surface area
- Moderate and severe changes showing pitting and brownish staining of tooth surface
- Corroded appearance of the tooth

Dentinogenesis imperfecta

- Autosomal dominant condition affecting both deciduous and permanent dentition.
- Grey – yellowish brown colored teeth.
- Broad crown with constricted cervical area.
- Tulip shaped tooth.
- Chromosome 4 – dentin sailophospho protein

- Broken enamel with exposed dentin.
- Attrition of teeth
- Lack of pulp chamber in the tooth.

Classification

- Dentinogenesis imperfecta I
- Dentinogenesis imperfecta II
  (Brandywine type)

DI I

- Opalescent dentin
- Not associated with OI
- Teeth are amber- grey, or amber brown
- Bulbous crown, shorter & narrow roots, pulp chamber & root canals are smaller, sometimes completely obliterated

1x

DI II (Brandywine type)

- Brandywine triracial isolates in southern Maryland
- Affects both Dentitions
- Multiple pulp exposures after rapid wear and tear of teeth
- deciduous - - large pulp chambers, later reduces in size.
- Large pulp chamber, & thin enamel, dentin – shell teeth.

Histopathology

- Irregular dentinal tubules
- Large areas of uncalcified matrix
- Less in number but larger sized dentinal tubules
- Degenerating odontoblasts

Dentin dysplasia

- Rootless teeth
- Rushton 1939
- Hereditary , autosomal dominant pattern of inheritance
- Two types – Type I (radicular dentin dysplasia)
  Type II (coronal Dentin dysplasia)

Type I Radicular DD

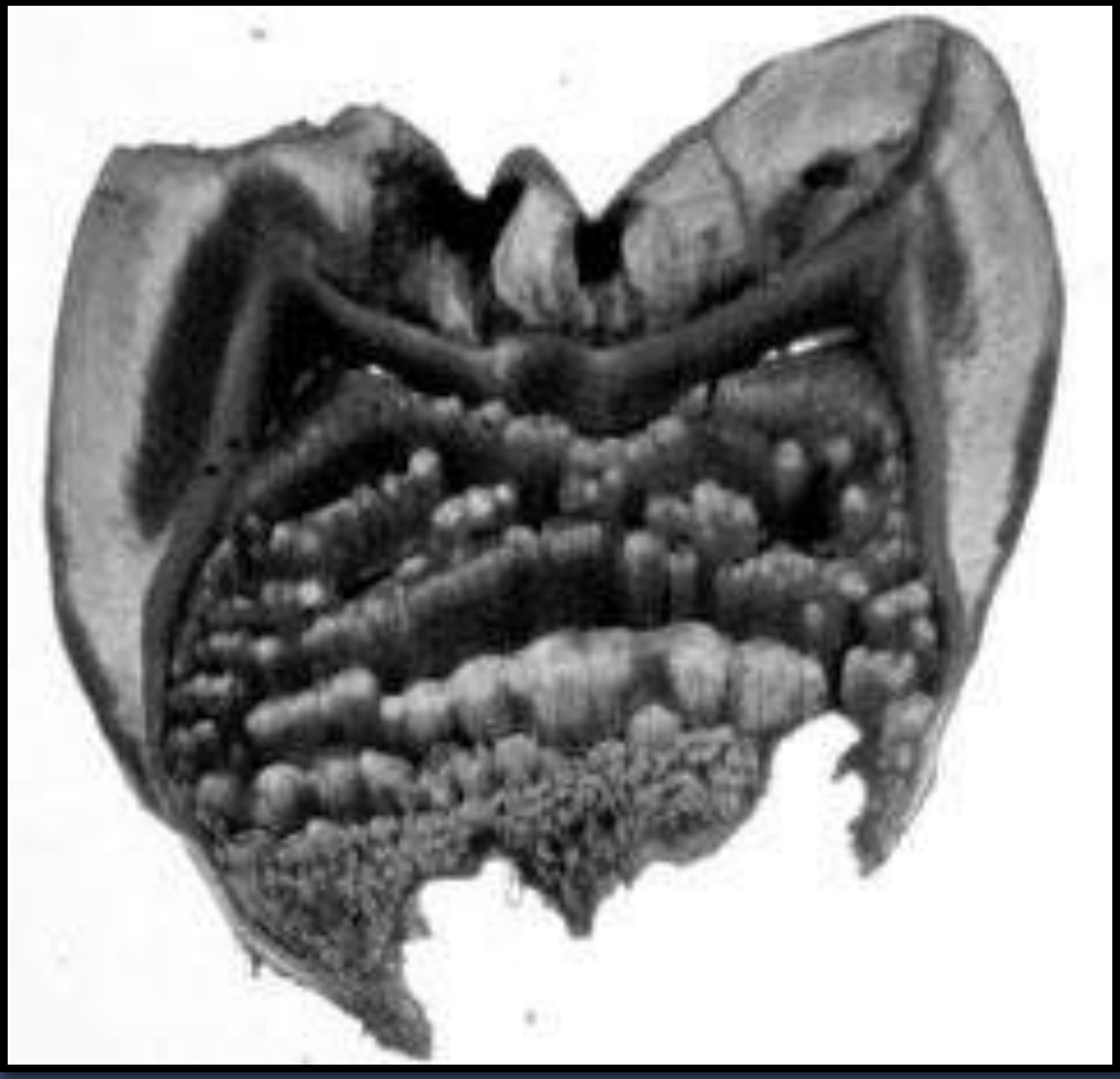
- Both dentitions are affected
- Teeth appear clinically normal in morphology and colour
- Slight amber translucency
- Extreme mobility of the teeth
- Premature loss of teeth after minor trauma
- Short roots

Radiographic features

- Roots are short and blunt
- Malformed roots
- Pulp chamber and canals are completely obliterated
- Crescent shaped pulpal remnant may be seen

Histopathology

- Part of coronal dentin – normal
- Apical dentin shows complete obliteration of canals
- Pulpal calcification
- Lava flowing around the boulders – normal dentinal tubule formation appears to be blocked so that new dentin formation around the obstacles



Type II Coronal DD

- Both dentitions are affected
- Involvement of deciduous common
- Yellow brown, bluish grey opalescent teeth
- Permanent appears normal clinically
- Large pulp chamber in coronal portion of the tooth – thistle tube appearance
- Calcifications in pulp

Histopathology

- Multiple pulp stones
- Amorphous a tubular dentin

Regional odontodysplasia

- Ghost teeth
- Unusual dental anomaly in which one or several teeth in a localized area are affected.
- Max > mand
- Permanent central, lateral, cuspids
- Both dentitions are affected
- Etiology – unknown, trauma or systemic illness, vascular defects

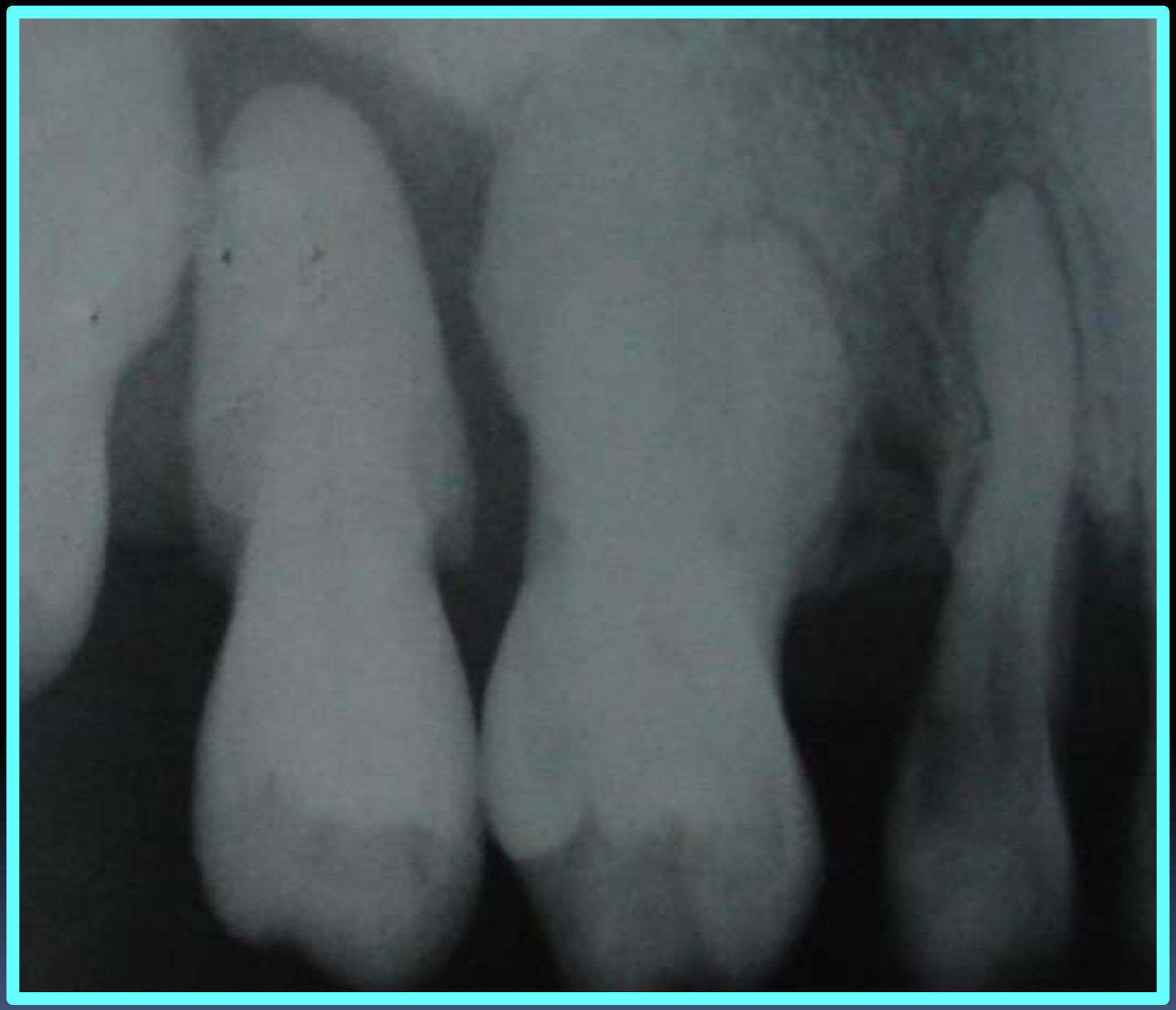
Clinical features

- Delay or total failure in the eruption
- Marked alteration in the shape, very irregular in appearance
- Defective mineralization
- Radiographic features – marked reduction in the radio density of the enamel and dentine leading to thin enamel and dentine and large Pulp chambers – GHOST TEETH

IN

Histopathology

- Marked reduction in the amount of dentine, the widening of pre-dentine layer
- Presence of large areas of inter-globular dentine
- REE around the non erupted teeth, shows numerous calcifications



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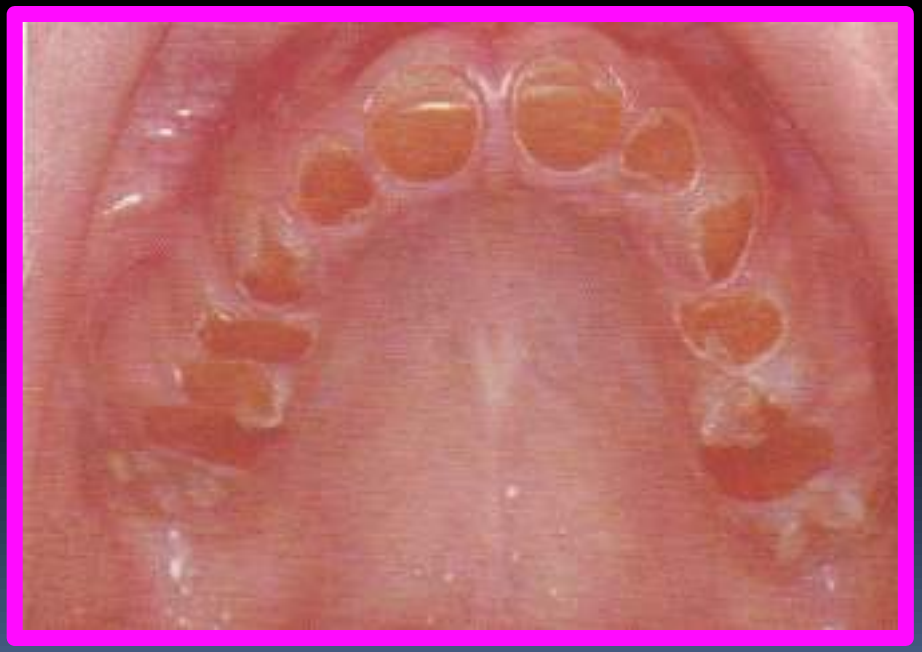

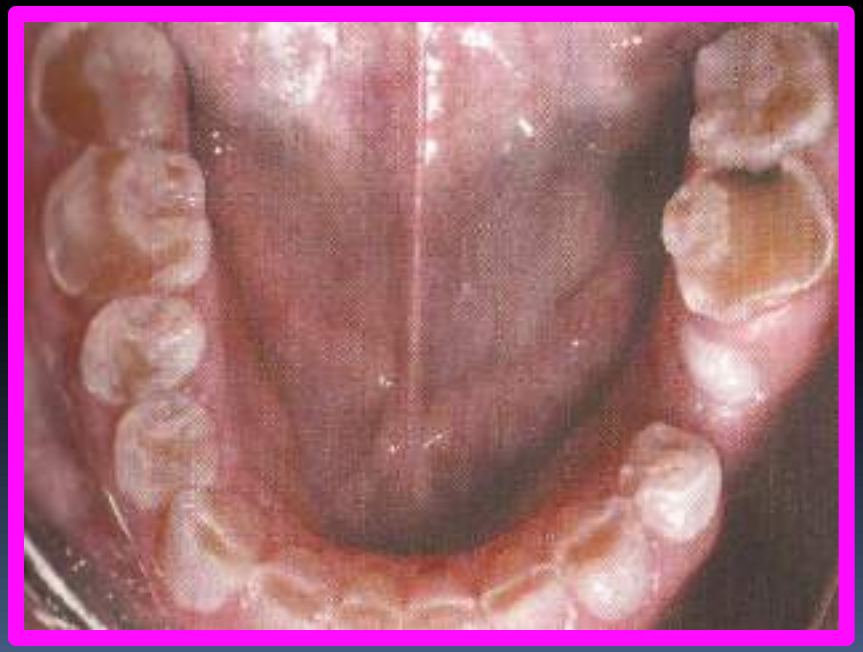

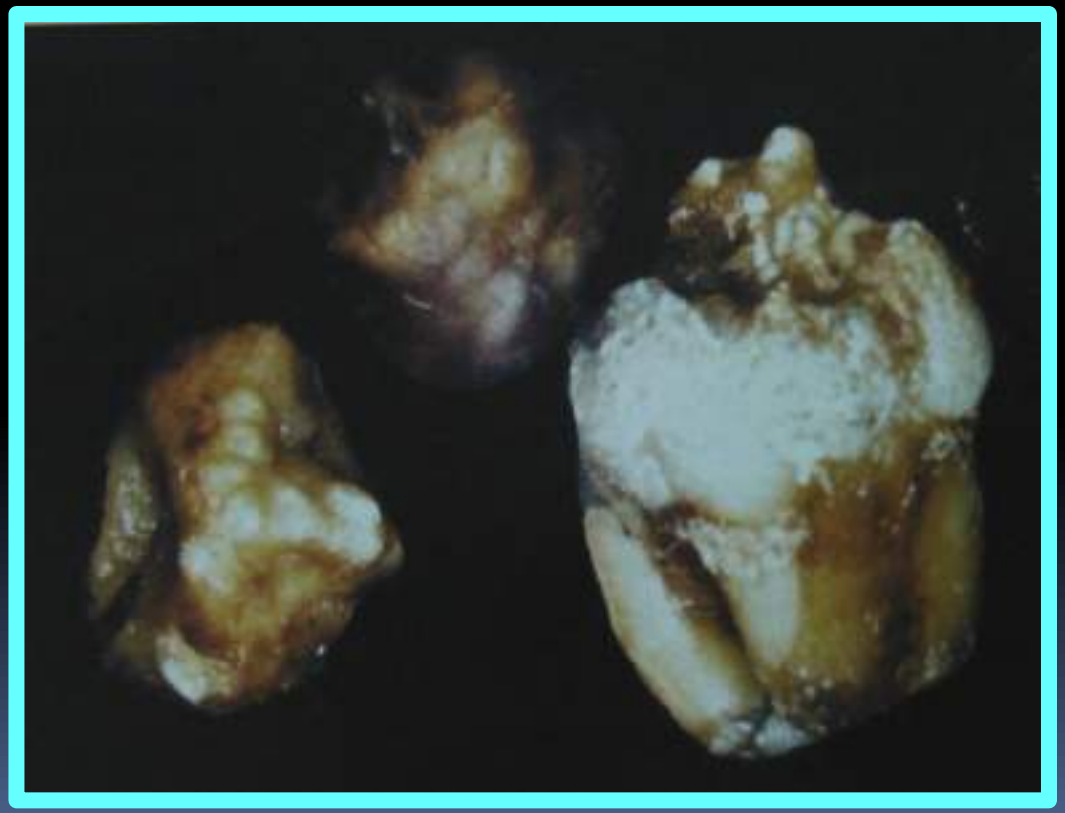

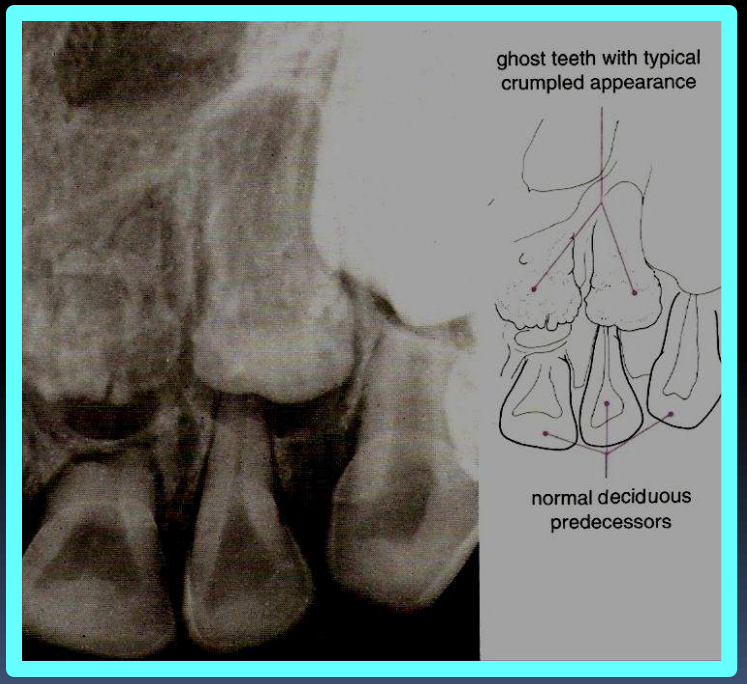
ghost teeth with typical crumpled appearance
normal deciduous predecessors

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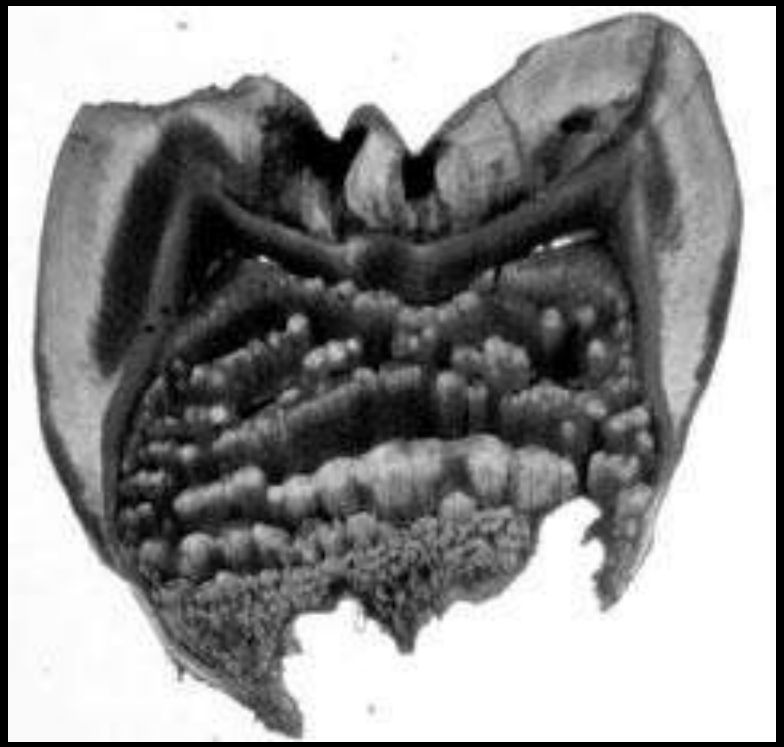


Candidiasis

- Candidiasis is a well known but incompletely understood opportunistic mycotic infection in humans.
- Oral candidiasis is a collective term given to a group of disorders caused by the yeast Candida and its species.
- Among the species of genus Candida -Candida albicans , C.tropicalis, C.glabrata, C.stellatoidea, C.parapsilosis, C.Krusei and C.kyffer.
- oral candidiasis may serve as a clinical marker that indicates the presence of significant underlying disease - “disease of the diseased”

- The effect of oral candidiasis may range from localized infections to acute, systemic disseminated disease .

- Oral candidiasis assumed a renewed importance in the recent times due to its association with Acquired Immuno Deficiency Syndrome.

- '*Moniliasis'* -- fungal infections in plants

- 'Candidiasis' -- United States , 'Candidosis' Canada, England, France, and Italy.

Predisposing Factors

- Local host factors
- Systemic factors
- Iatrogenic factors and miscellaneous factors .

Local host factors

1. Mucosal Barrier

- Exogenous epithelial changes
  - Trauma
  - Laceration
- Endogenous epithelial changes
  - Atrophy
  - Hyperplasia
  - Dysplasia

2. Saliva

- Quantitative changes
  - Xerostomia
  - Sjogren's syndrome
  - Radiotherapy
- Qualitative changes
  - pH
  - Glucose concentration

3. High Carbohydrate diet

4. Commensal flora

Systemic Factors

Altered physiological states
- Infancy
- Old age

Altered hormonal states
- Diabetes
- Hypothyroidism
- Hypoparathyroidism
- Hypo-adrenocortism

Altered nutritional state
- Hypovitaminosis
- Iron deficiency
- Malnutrition

Altered Immune Mechanisms
- Decreased number of phagocytes
- Intrinsic defects in immune cells
- Defects in cell mediated immunity
- Due to infective states

Iatrogenic And Miscellaneous Factors

Iatrogenic factors

- Antibiotics
- Corticosteroids
- Contraceptives
- Immunosuppressive drugs

Miscellaneous factors

- Cigarette smoking

Classification

- Primary Oral Candidiasis (Group I)
- Secondary Oral Candidiasis (Group II)

Group I

Acute

- Pseudomembranous

Chronic

- mucocutanoeus candidiasis
- Pseudomembranous
- Erythematous
- Hyperplastic
- Plaque – like
- Nodular

Candida-associated lesions

- Denture stomatitis
- Angular cheilitis
- Median rhomboid glossitis

Group II

- Familial chronic mucocutaneous candidiasis
- Diffuse chronic mucocutaneous candidiasis
- Candidiasis endocrinopathy syndrome
- Familial mucocutaneous candidiasis Severe combined immunodeficiency
- Di-George’s syndrome
- Chronic grnaulomatous disease
- HIV disease and Acquired Immuno Deficiency Syndrome (AIDS)

- Recently keratinized primary lesions superinfected with candida
  - Leukoplakia
  - Lichen planus
  - Lupus erythematosus

Pseudomembranous Candidiasis

- Classically an acute infection and chronic in patients using corticosteroids, HIV infected individuals, and in other immunocompromised patients.

- This form may be seen in neonates and among terminally ill patients.

- Characterized by white membranes on the surface of the oral mucosa, tongue and elsewhere.

- The lesions develop to form confluent plaques that resemble milk curds, and can be wiped off to reveal a raw, erythematous and sometimes bleeding base.

- Oropharyngeal thrush may sometimes spread to the adjacent mucosa, particularly those of the upper respiratory tract and the esophagus.
- combination of oral and esophageal candidosis -- HIV-infected patients.

Erythematous Candidosis

- Erythematous candidiasis - atrophic candidiasis.
- Atrophic candidiasis -- uncommon and poorly understood condition associated with corticosteroids and topical or systemic broads spectrum antibiotics or in HIV disease.
- persistent acute pseudo membranous candidosis when pseudo membranes are shed or may develop de novo.

Clinical Features

- Erythematous areas generally on the dorsum of the tongue, palate or buccal mucosa.
- Lesions on the dorsum of the tongue present as depapillated areas.
- Red areas are often seen in the palate in HIV disease.
- Can be an associated angular stomatitis.
- Other features include, mouth being scaled by hot beverages, burning sensation and bald appearance of tongue.

Thrush associated with erythematous candidiasis
Picture credit: Pr J. Samson, Geneva

Plaque-like or Nodular Variant of Candidosis

- Hyper plastic candidosis.
- Lesions are almost always chronic in nature presenting as discrete raised lesions that vary from small, palpable, translucent, whitish areas to large, dense, opaque plaques.
- Nodular lesions - leukoplakias (3 - 50%)
- 2 - 6% risk of malignant transformation
- Iron and folate deficiencies and with defective cell mediated immunity. -- etiology

Candida-associated denture stomatitis

- chronic atrophic candidosis or denture-induced stomatitis or denture stomatitis.
- Most common oral fungal infection in the elderly denture wearers.
- chronic erythema, edema of the mucosa that contacts the fitting surface of the denture.
- Angular stomatitis is often the only presenting complaint.

Candida-Associated Angular Cheilities

- Angular cheilitis, also called Perleche or angular stomatitis.
- Multifactorial in origin and affect the angles of the mouth, either unilaterally or bilaterally, especially in the elderly denture wearers.
- characterized by soreness, erythema and fissuring.
- Angular stomatitis is, very occasionally, an isolated initial sign of anemia or vitamin deficiency.
- angular cheilities have been described in HIV disease

Median Rhomboid Glossitis

- Midline glossitis, or glossal central papillary atrophy, is characterized by an area of papillary atrophy, which is elliptical or rhomboid in shape, symmetrically placed centrally at the midline of the tongue anterior to the circumvallate papillae.

- The relevance of candida to the condition has been controversial .
- However it has been demonstrated that the condition frequently shows a mixed bacterial/fungal micro flora .

- **Etiology:**
- Thought to be due to persistence of tuberculum impar during formation of tongue (developmental anomaly).

- As a result of candidal infection in this area.

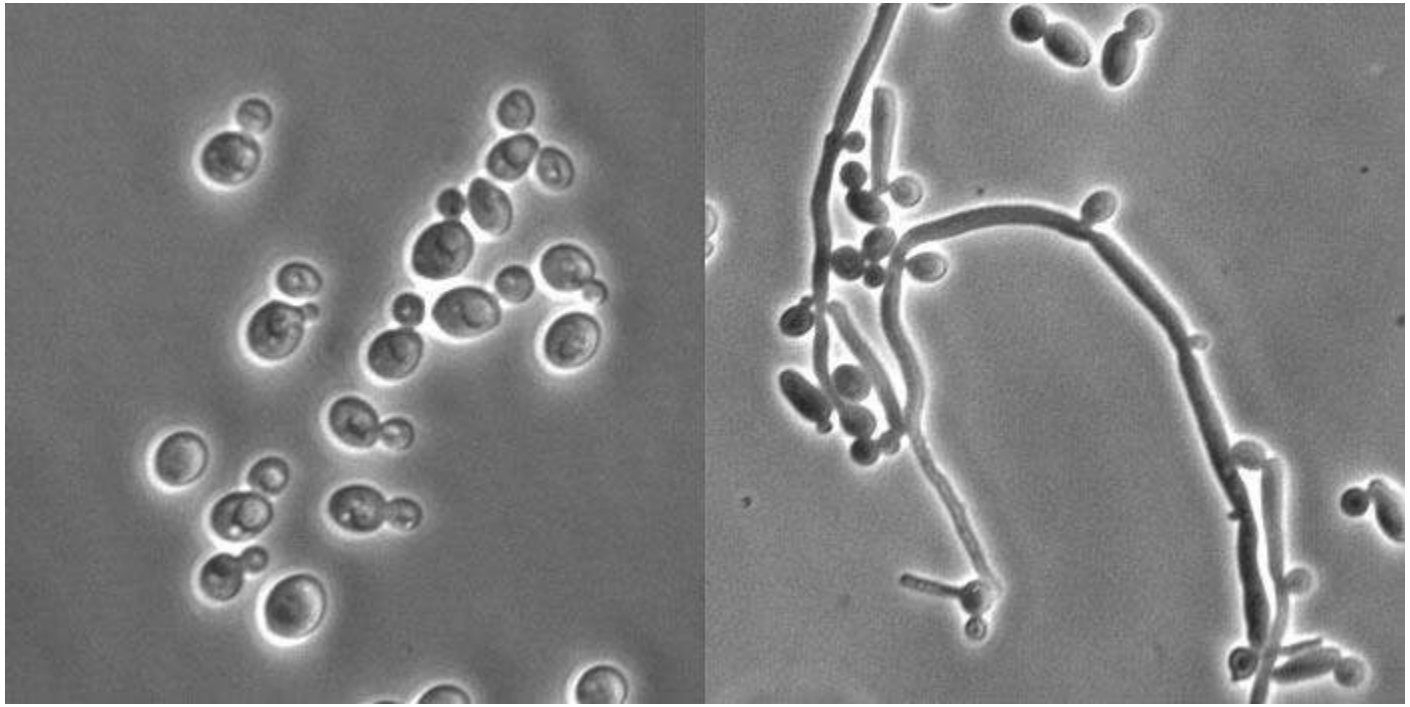
- **HIV related oral candidiasis may present as 4 distinct clinical variants, namely ;**
  - Pseudomembraneous
  - Erythematous (atrophic)
  - Hyperplasic
  - Angular cheilitis

lab diagnosis

- Smear
- Swab
- Paper Points.
- Concentrated Oral Rinse.
- Imprint Culture
- Impression culture
- Salivary culture technique.
- Biopsy/ Histopathology.
- Culture media.

Smear/ Histopathology.

- Direct examination of smears stained with grams stain.
- Water mount/ wet film :
- Potassium hydroxide technique (KOH)
- H& E and PAP
- Grocott's – Gamori's methanamine silver (GMS) technique.
- Periodic acid-Schiff reaction (PAS)



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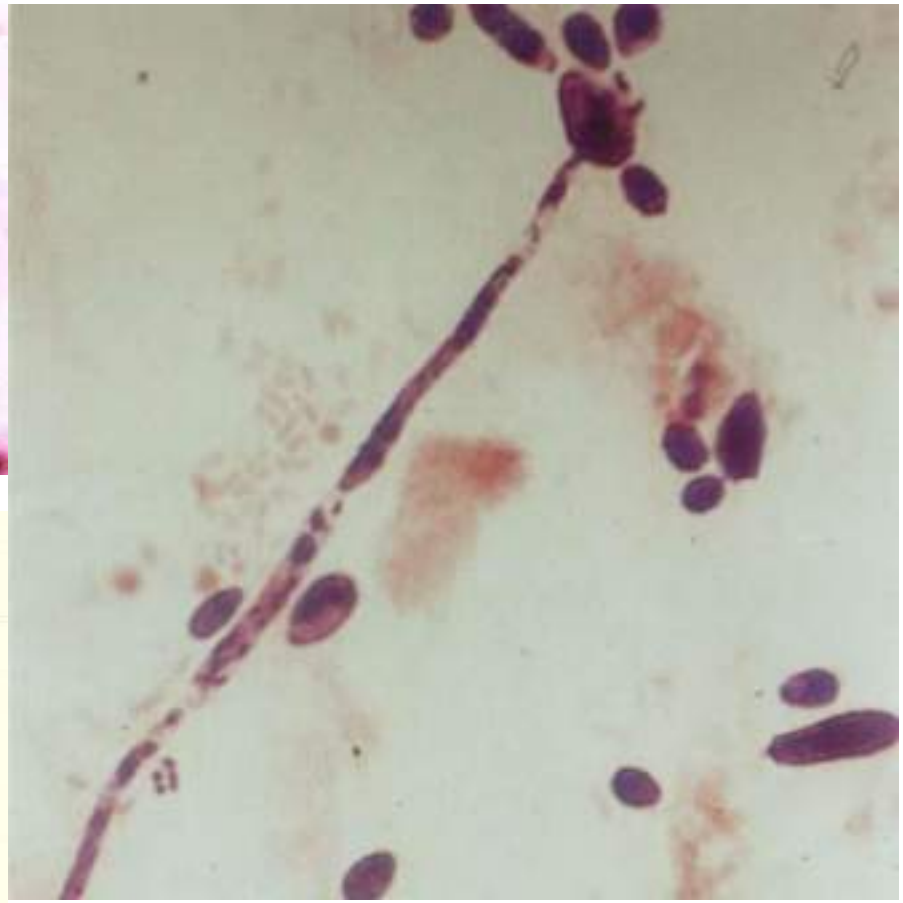
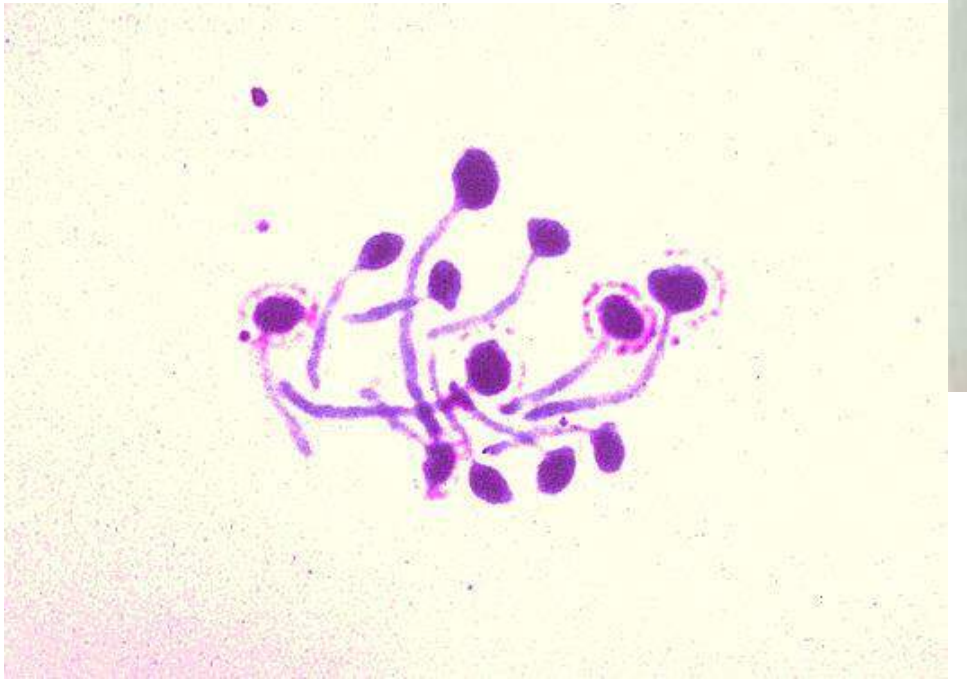
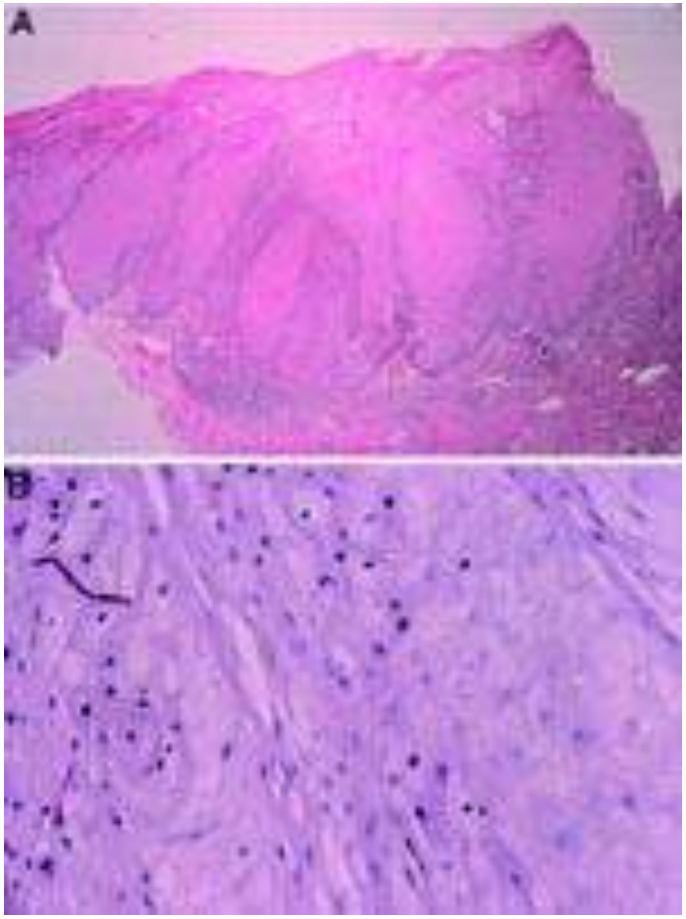





A
B

Culture Methods

- **Sabouraud's Dextrose Agar**
- **Corn-meal agar (CMA)**
- **Pagano- Levin**
- **CHROM agar**

Treatment

- **Topical and systemic Antifungal Medications**
- Nystatin
- Ketoconazole tablets
- Fluconazole
- Itraconazole
- Miconazole

DISEASES OF PERIODONTIUM

Classification

Gingival diseases

- Plaque induced Gingival Disease
- Non Plaque induced gingival disease

Chronic Periodontitis

- Localized /Generalized

Periodontitis as Manifestation of Systemic Diseases

- Necrotizing periodontal diseases
- Necrotizing ulcerative gingivitis
- Necrotizing ulcerative periodontitis

Abscesses of the periodontium

- Gingival Abscess
- Periodontal abscess
- Pericoronal Abscess

Classification

Periodontitis associated with endodontic lesions

- Endodontic –periodontal lesion
- periodontal –Endodontic lesion
- Combined Lesions

Developmental or Acquired deformities & Conditions

- Localized tooth related factors that predispose to plaque induced gingival diseases or pericoronitis
- Muco gingival deformities & Conditions around teeth
- Muco gingival deformities and conditions on edentulous ridges

Occlussal Trauma (TFO)

Dental plaque induced gingival diseases

- Gingivitis – inflammation of the gingiva.
- Plaque induced is the common form.
- It's a result of interaction between plaque bacteria and host defense.

Local factors:

- Micro organisms, calculus. food impaction, faulty, or irritating appliances, mouth breathing, tooth malposition.

Systemic factors:

- Nutritional deficiency, drug reaction, endocrine changes associated with – puberty ,pregnancy, menstruation, DM, allergy, heredity, granulomatous infection, neutrophil dysfunction and immunopathies.

Clinical features

- Acute and chronic.
- Localized and generalized.
- Alteration in color in the marginal gingiva – deep pink – reddish.
- Bleeding from the gingival sulcus.
- Swelling and edema – absence of stippling.
- Marked enlargement ,edema , fibrosis – chronic hyper plastic gingivitis.
- Pus discharge – later stages of chr.gingivitis

Radiographic features

- No bony changes, if any – periodontitis.

Histopathology

- Engorged blood capillaries, infiltration of inflammatory cells into the CT.edematous epithelium.

Necrotizing ulcerative gingivitis

- *Rare Bacterial Disease : Acute & Sub acute
- Vincent's infection (trench mouth disease).
- The inflammatory condition involves mainly free gingival margins, the crest of Gingiva and interdental papillae.
- When soft palate and tonsilar areas are involved- Vincent's angina.
- Epidemic pattern- world war I& II.

- **Etiology**

- **Bacterias Involved**
- Treponema
- Fusioform Bacillus

- **Local Predisposing Factors**
- Injury To Gingiva
- Smoking
- Deep Periodontal Pockets And Periocoronal Flaps.

- **Systemic Predisposing Factors** :

- Nutritional Deficiency Eg. : Vit. C, Vit. B2

- **Debilitating Disease**
- Chronic Diseases Like Syphititis Etc.
- Cancers
- Gastrointestinal Disorders Like Ulcerative Collitis
- Blood Dysplasia Such As Leukemia And Anaemia
- AIDS

ANUG

- **Clinical Features**
- History : Sudden Onset
- May Follow Debilitating Disease Or Acute Respiratory Tract Infection.
- Psychological And Physical Stress.
- May Have A History Of Repeated Remissions And Exacerbations

Oral Signs

- Characteristic Lesions Are Punched Out, Crater-like Depression At The Crest Of Interdental Papillae.
- May Extend Upto Marginal Gingiva Or Rarely To Attached Gingiva And Mucous Membrane.

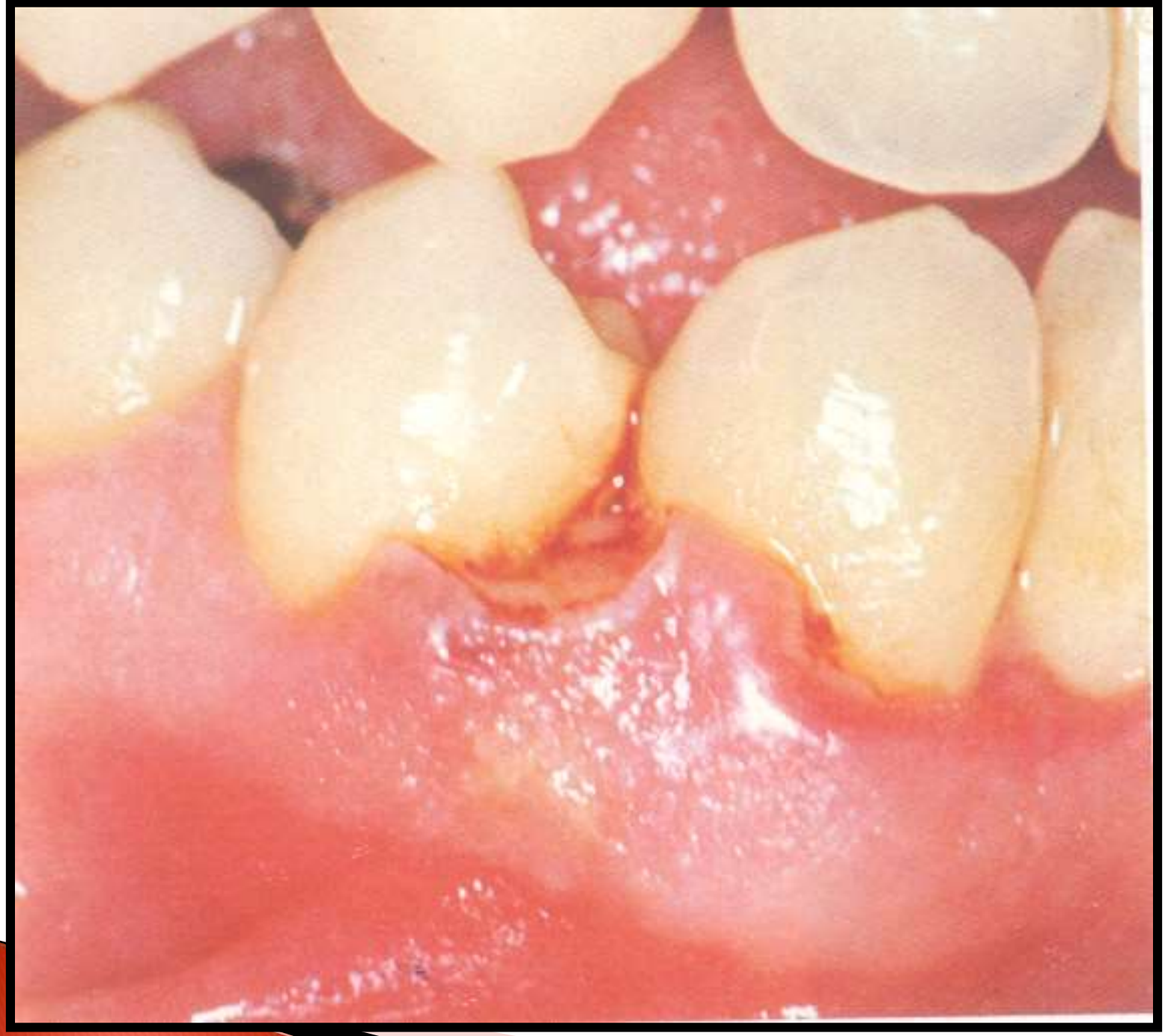
❖Gingival Crater Is Covered By Gray, Pseudomembranous Slough, Demarcated From The Remainder Of The Gingival Mucosa By A Pronounced Linear Erythema.

❖At Times The Pseudomembrane May Be Denuded Exposing The Gingival Margin, Which will be Red, Shiny And Hemorrhagic.

❖Spontaneous Gingival Hemorrhage Or Pronounced Bleeding On Slightest Stimulation Is Characteristic Feature.

❖Fetid Odor

❖Increased Salivation



Oral Symptoms :

- Extremely Sensitive To Touch.
- Constant Radiating, Gnawing Pain, Which Aggravates On Eating Spicy Or Hot Foods.
- Metallic Foul Taste
- Excessive Amount Of ‘Pasty’ Saliva

Extra-oral Or Systemic Signs & Symptoms

- Local Lymphadenopathy
- Slight Elevation Of Temperature

<u>In Severe Cases.</u>

- High Fever
- Increased Pulse Rate
- Leukocytosis
- Loss Of Appetitie
- Insomnia, constipation, GI disorder headache and mental depression. Sometimes accompany the condition.
- In severe cases Noma or gangrenous stomatitis may be seen

DIFFERENTIAL DIAGNOSIS

- Herpetic Gingivostomatitis
- Chronic Periodontitis
- Desquamative Gingivitis
- Streptococcal Gingititis
- Apthous Stomatitis
- Diptheritic And Syphilitic Lesions
- Tuberculous Gingival Lesion
- Candidiasis
- Agranulocytosis
- Dermatoses (Pemphigus, Erythema Multiforme In Lichen Planus

- **H/P**: Non Specific Inflammatory lesion with areas of necrosis.

- Final diagnosis is by correlating with clinical features.

- **TREATMENT:** Prophylaxis, antibiotics & recontouring of gingival papillae in severe cases.

Desquamative gingivitis

- Clinical term characterized by intense redness and desquamation of the surface epithelium of the gingiva.
- Not a disease entity – oral manifestation of some gingival diseases.
- Cicatricial pemphigoid, pemphigus, and lichen planus.
- F>M, 40 -45 yrs.

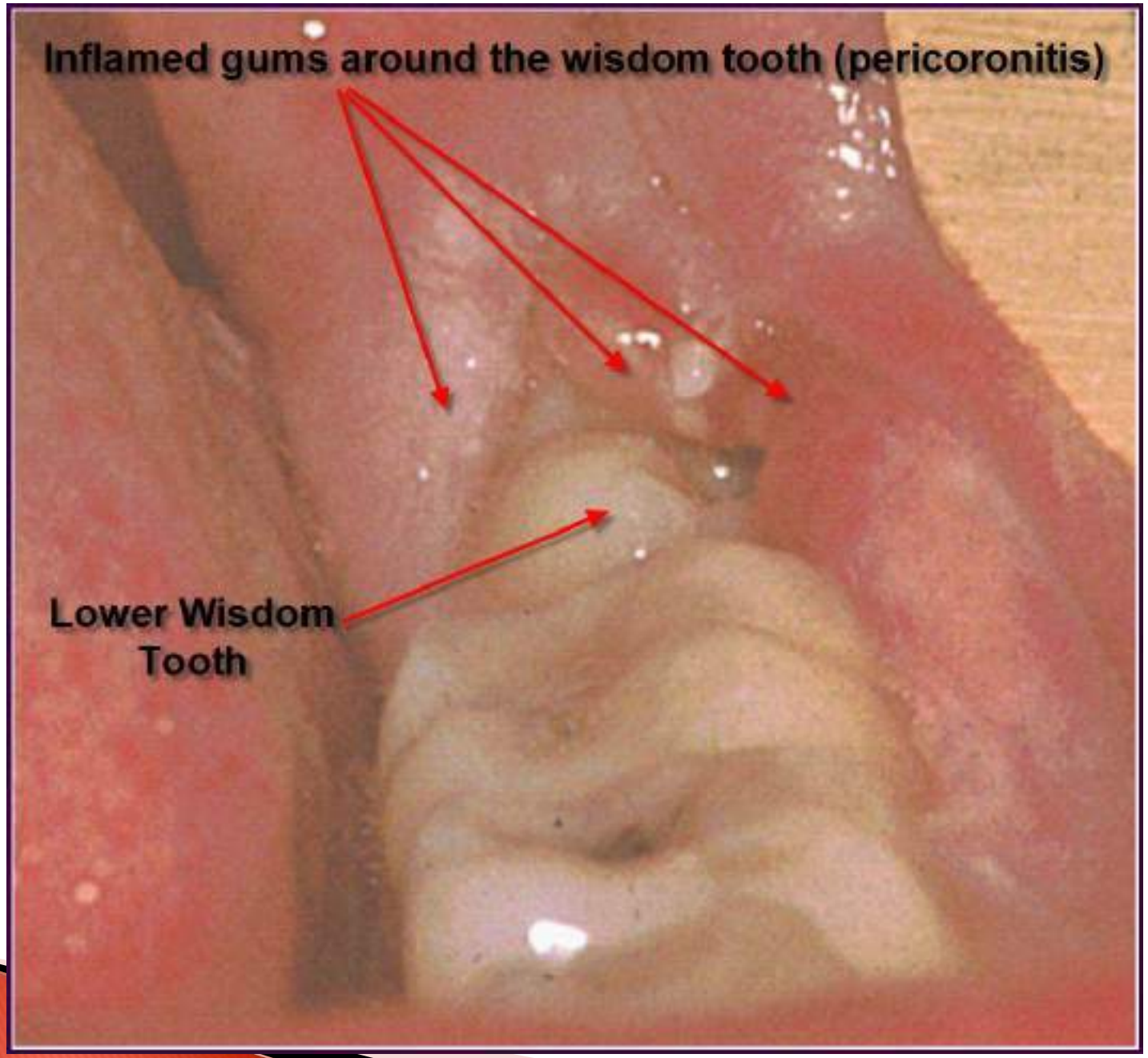
- Red, swollen, glossy pearing gingiva with loss of stippling.
- Multiple vesicles and superficial denuded areas.
- Nikolsky's sign + ve -- peeling of tissues at the dermal - epidermal junction at slight pressure.

Gingival abscess

- An acute, localized painful lesion caused by the forceful penetration of any foreign objects.
- Limited to marginal gingiva.
- Initially red & shiny, within hours becomes fluctuant and exhibits pointing through which pus discharges.

Pericoronitis

- An inflammatory lesion occurring around the impacted or partially erupted tooth.
- Incomplete eruption – food stagnation under gingival flap --- infection – inflammation.
- May leads to abscess – pericoronal abscess.
- third molar , mand > max.


Inflamed gums around the wisdom tooth (pericoronitis)
Lower Wisdom Tooth

- Difficulty in chewing, opening of the mouth.
- lymphedenopathy, mild fever.
- H/p -- epithelial hyperplasia, intercellular edema, lymphocytic infiltration, increased vascularity.

Gingival enlargement.

- Increase in the size of the gingival tissues, which may be the result from a number of different conditions.

- Etiologic factors:

1. Inflammatory.
2. Drug induced.
3. Enlargement associated with systemic factors
4. Idiopathic gingival enlargement
5. Neoplastic

Inflammatory enlargement

- Prolonged chronic inflammation.
- Local factors, poor oral hygine, acucumulation of dental caluclus, mouth breathing.
- Gingiva are soft edematous, hyperemic, and sensetive to touch.
- Easy to bleed, non stippled & glossy.

Drug induced enlargement

- Anti convulsants, immunosuppressants and calcium channel blockers.
- Dilantine sodium,
- Cyclosporine,
- Nefidefine, Verapamil.

Enlargement associated with systemic factors

- Conditional enlargement.
- Systemic condition of the patient which exaggerates the usual gingival response to plaque.
- Plaque is essential for initial enlargement.

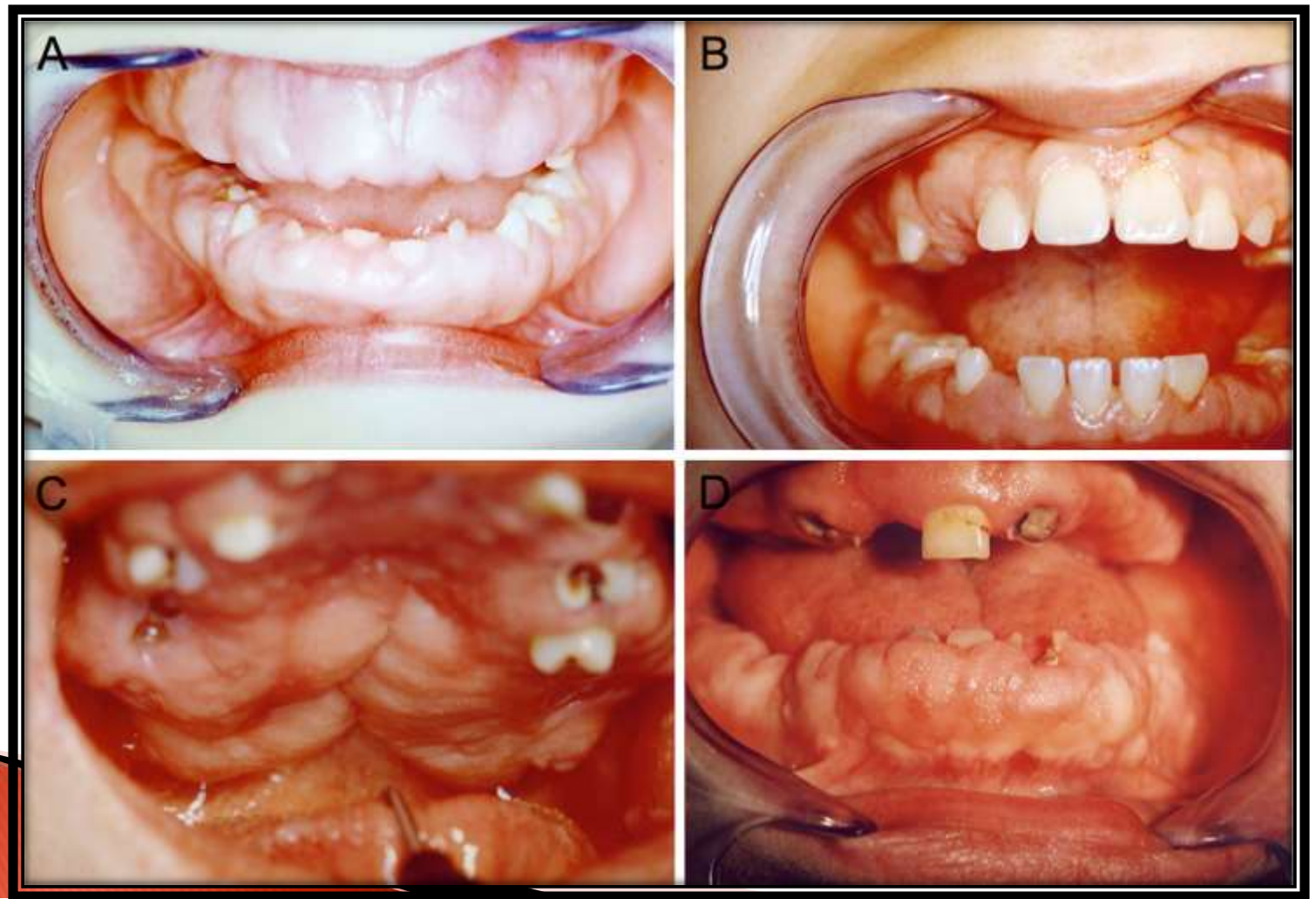
- Hormonal , Nutritional, Allergic .
- Puberty, pregnancy.
- Vitamin C deficiency -- scurvy.
- Plasma cell gingivitis.

Enlargement due to systemic diseases

- Leukemia
- Granulomatous diseases – Crohn's disease, Sarcoidosis, Wegner's granulomatosis.

Idiopathic gingival enlargement.

- Fibromatosis gingiva.



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Neoplastic enlargement

- Benign and malignant.
- Fibroma, granular cell tumour, PGCG, peripheral odontogenic tumours.
- Fibrosarcoma, leukemia, SCC.

Periodontitis

- Inflammatory disease of the supporting tissues of the tooth caused by specific microorganisms or the group of microorganisms, resulting in progressive destruction of the periodontal ligament and the alveolar bone with pocket formation.

- Chronic
- Aggressive
- TFO – Acute form

Chronic periodontitis

- Most common form.
- Associated with local irritation.
- Preceded by local irritation and gingivitis.
- Local factors – microbial plaque, calculus, food impaction & irritating margins of the fillings.

- Actinobacillus actinomycetem comitans,
- Bacteroids forsythus,
- Porphromonas gingivalis,
- Fusobacterium nucleatum etc.

Clinical features

- Begins as a simple marginal gingivitis as a reaction to plaque or calculus.
- Tiny ulcerations of the crevicular epithelium.

- Untreated – more n more accumulation of local irritant --- more inflamed and edematous gingiva – ulceration or detachment of epithelium in gingival sulcus (deepening of gingival sulcus) ---- periodontal pocket formation – destruction of alveolar bone -- detachment of PDL fibres – gingival recession ----- PERIODONTITIS

- Bleeding gums
- Mobility of the teeth.
- Gingival recession.
- PDL pocket formation.
- Alveolar bone loss.

Aggressive periodontitis

- Rapidly progressing type of periodontitis in patients who do not have large accumulation of plaque or calculus.
- Localized or generalized.
- JUVANILE PERIODONTITIS.
- Defect in the immune response than local factor influence.

Clinical features

Localized form:

- Occurs during puberty
- Familial tendency
- First molars and incisors.
- Absence or minimal influence of local factor.
- Higher rate of bone loss.

Generalized form:

- Under the age 30
- It involves three teeth other than first molar and incisor
- Presence of marked accumulation of local factors.

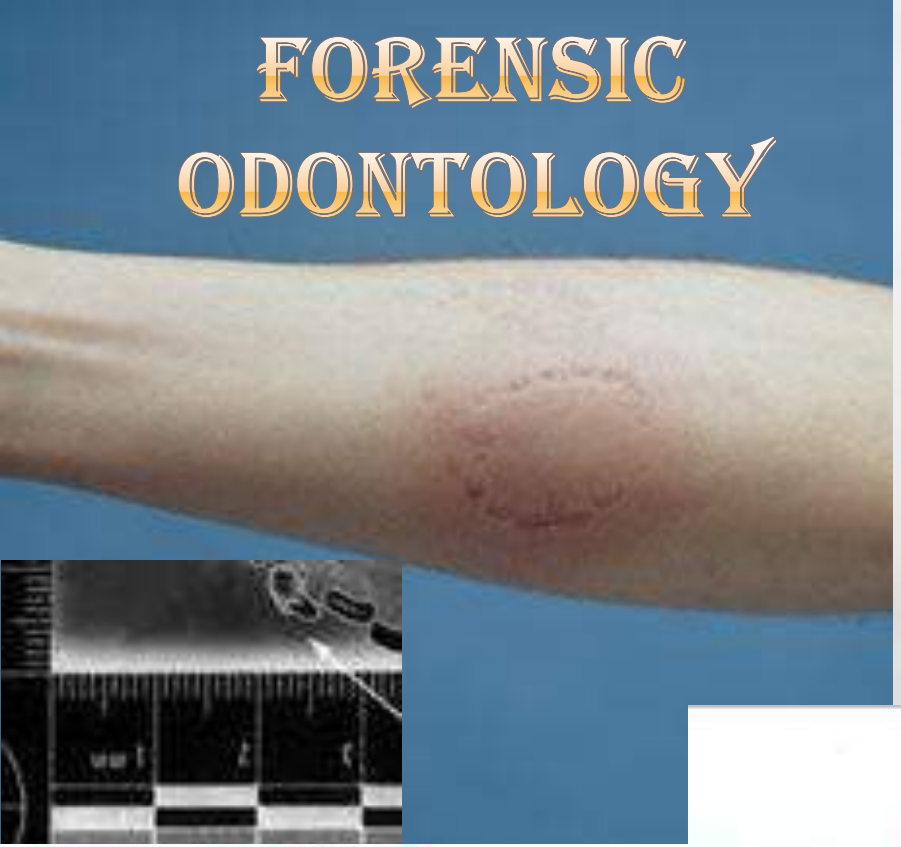
Periodontal abscess

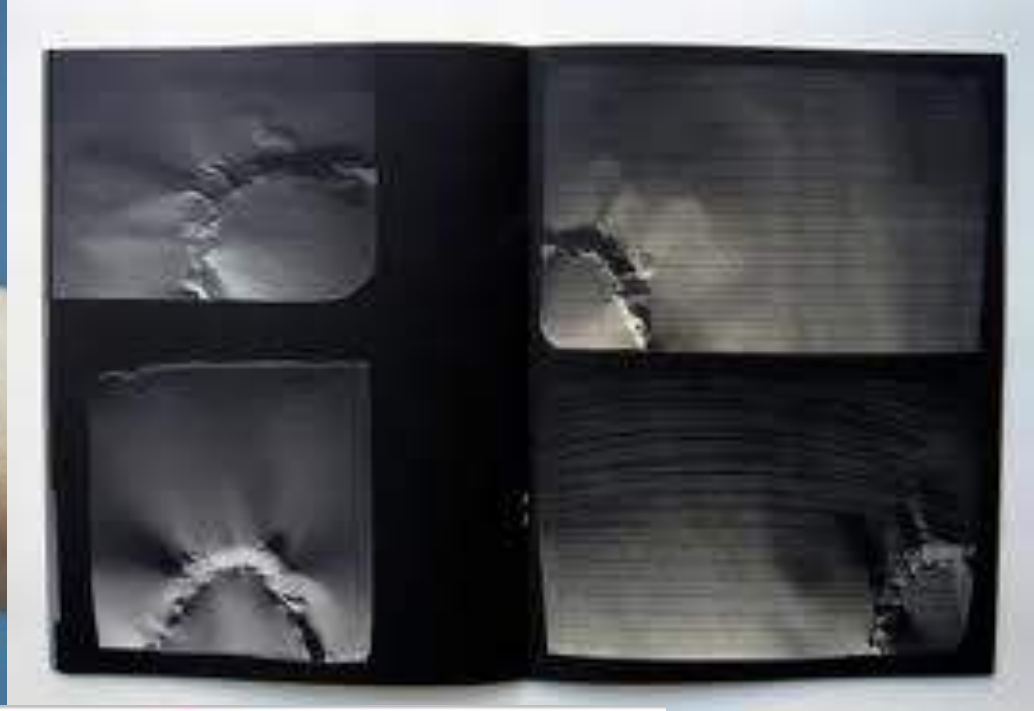
ANUP

TFO

FORENSIC ODONTOLOGY

FORENSIC ODONTOLOGY





INTRODUCTION

- Forensic - Latin word Forensics.
- Forum - Meeting place - civic and legal matters were discussed by those with the public responsibility –court of law.
- Forensic science -- study and practice of the application of science to the purposes of justice.
- Criminalistics pathology, toxicology, physical anthropology, odontology, psychiatry.

INTRODUCTION

- Forensic dentistry (Odontology) -- Dental specialty that relate and applies dental knowledge to legal problems.

- Indestructibility of the teeth & their uniqueness in each individual's age, sex and race.
- Dental anthropology, photography, serology bite-mark analysis, microscopic analysis and soft tissue investigations.

DEFINITION

- Forensic odontology as the application of dental science to the law; that is the use of dental evidence in the interest of the justice. -- American Society of Forensic Odontology.

- FDI – branch of dentistry which in the interest of justice, deals with the proper handling and examination of dental evidences, and with proper evaluation and presentation of dental findings.

- Deals with identification based on the recognition of unique features present in an individual's dental structures.
- Important role in personal identification.
- Mass disaster – natural or man made, where conventional methods are failure.

- Forensic anthropology
- Bite mark identification
- Mass disaster identification
- Dental prosthesis identification
- Forensic photography
- Charting and Record keeping
- Clinical laboratory analysis
- Testimony and expert witness
- Malpractices
- Peer review
- Child abuse
- Research in Forensic Dentistry

PERSONAL IDENTIFICATION

- Identity - characters by which a person may be recognized.

- Settlement of property.
- Facilitate remarriage of a surviving spouse.
- Crimination of body according to relevant religion and cultural methods.

- Traditional methods – clothing, jewellery, etc.
- Surgical scars, moles, fractures etc.

IMPORTANCE DENTAL IDENTIFICATION

- Resistant to post mortem changes, and decomposition.
- Material – majority of the restorative materials and others are also resistant to PM changes.

DENTAL IDENTIFICATION PROCEDURES

1. Comparative identification.

2. Reconstructive identification.

COMPARATIVE IDENTIFICATION

- Conventional method of post-mortem dental identification.

1. Oral autopsy
2. Obtaining dental records
3. Comparing PM & AM data
4. Writing report &drawing conclusion

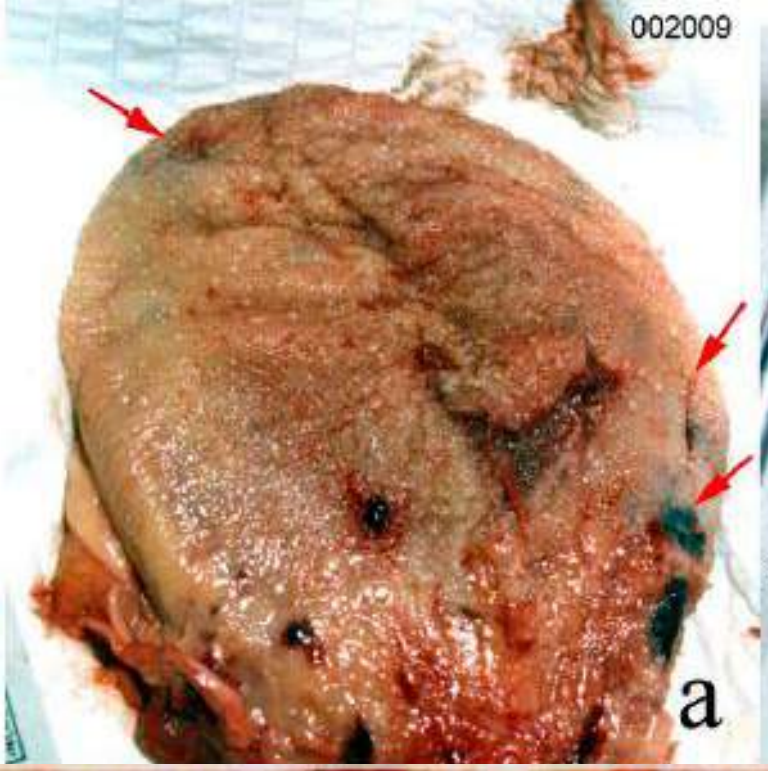
RECONSTRUCTIVE IDENTIFICATION

- Dental profiling
- Virtually no clue available about the deceased.
- Age, sex, race, gender, DNA sampling.

ORAL AUTOPSY

- Necropsy or post mortem.
- Examination of deceased, usually with dissection to expose the organs, to determine cause of death.
- Critical examination of external features of the body – gender, ethnicity, build, wound, scars, tatto, body piercing, hair sample, finger prints, finger nail scraping.

- Oral examination
- Mouth gag or intra oral myotomy – jaw separation.
- Burn cases – tooth are brittle – coating with cyanoacrylate cement, polyvinyl acetate, or clear acrylic paint.
- Thorough examination of hard & soft tissues, injuries, fractures, photographs, radiographs.
- Interpol PM changes form – PINK.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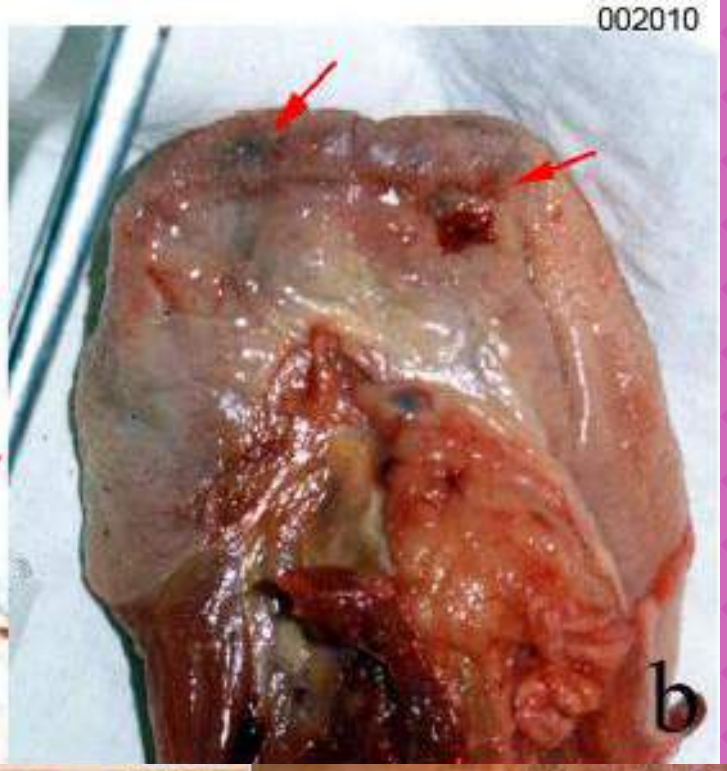
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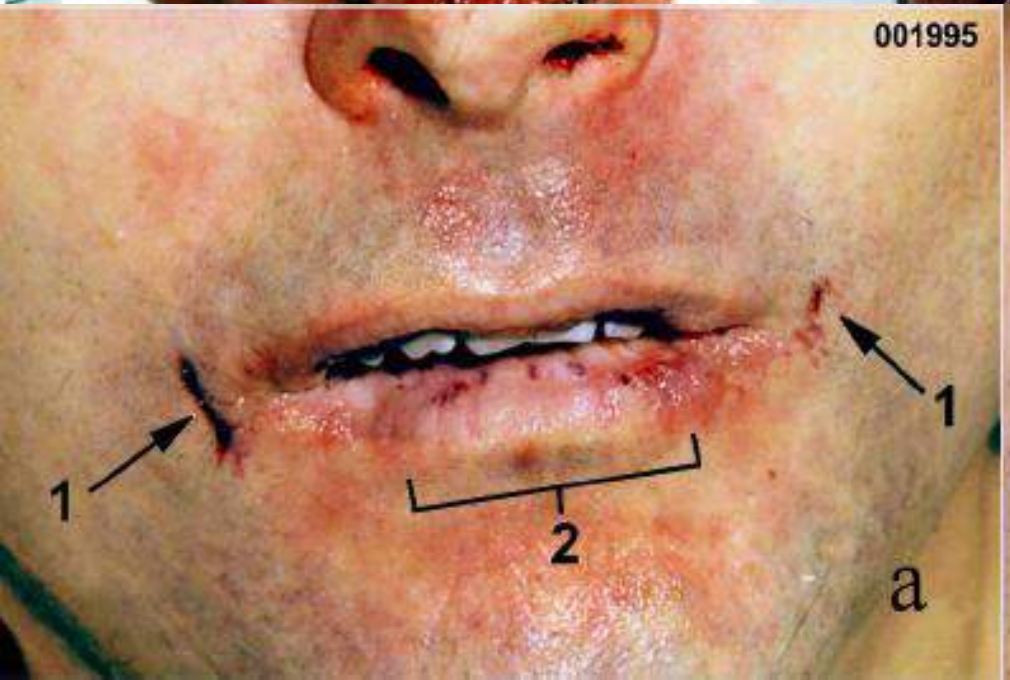
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1
1
2
a

001999
b

P.M. Mass (print) **VICTIM IDENTIFICATION FORM** F2

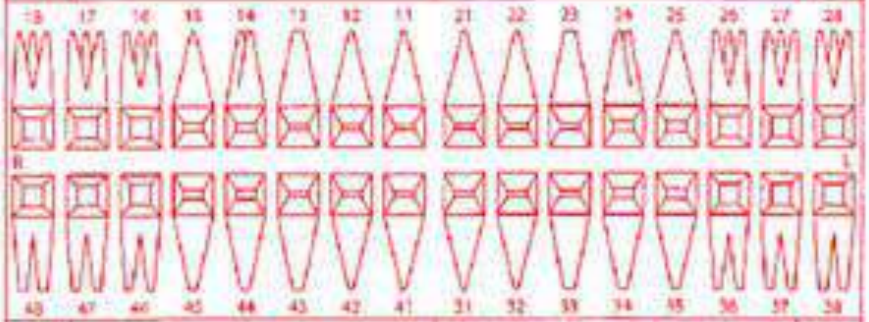
DEAD BODY

Nature of disaster: _____ No: _____

Place of disaster: _____ Sex unknown ☐

Date of examination: ☐☐ Day ☐☐ Month ☐☐ Year Male ☐ Female ☐

86	DENTAL FINDINGS		
51-11			21-61
52-12			22-62
53-13			23-63
54-14			24-64
55-15			25-65
16			26
17			27
18			28



48		38
47		37
46		36
85-45		35-75
84-44		34-74
83-43		33-73
82-42		32-72
81-41		31-71

87	Specific description of Crowns, bridges and dentures	88	Further findings: Occlusion, attrition, anomalies, implants, periodontal status, etc.
89	X-rays taken of:		
90	Supplementary examinations		
91	Age evaluation (method):		

Odontologist
Name _____
Address _____
Phone number _____

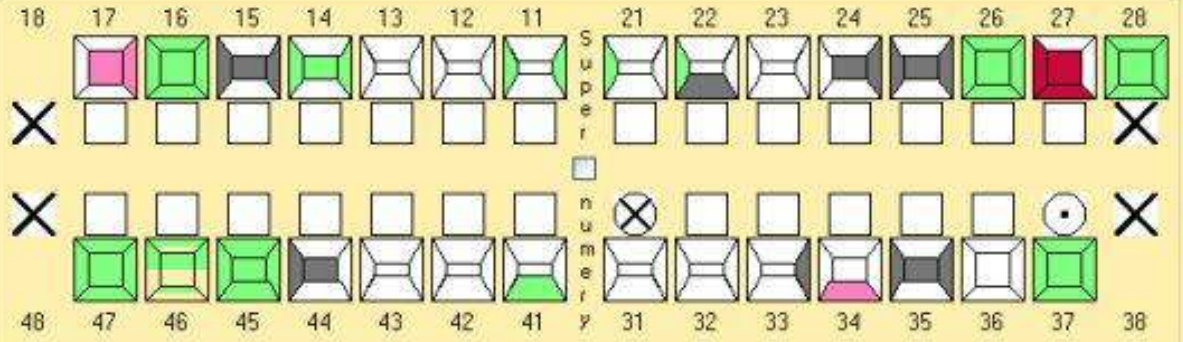
Signature

OBTAINING DENTAL RECORDS.

- Obtaining his/her dental information- treatment / other hospital records.
- Casts, dental charts, radiographs and photographs.
- Interpol AM form – YELLOW.

86 Dental information

11	cof MD	cof M	21
12	sou	cof M, amf L	22
13	sou	sou	23
14	cof MOD	amf OD	24
15	amf MOD	amf MOD	25
16	mcc	mcc	26
17	car MO	goi MOL	27
18	mis	mcc	28



48	mis	mis	38
47	abu mcc	mcc rfx	37
46	mam mcp	sou	36
45	abu mcc	amf MOD	35
44	amf OD	car V	34
43	sou	amf D	33
42	sou	sou	32
41	tcf V	mpm	31

87 Specific data

Upper Jaw

- ☐ Crown
- ☐ Bridge
- ☐ Partiel denture
- ☐ Edentulous

Lower Jaw

- ☐ Crown
- ☐ Bridge
- ☐ Partiel denture
- ☐ Edentulous

COMPARISON OF AM &PM DENTAL DATA

- Tooth morphology , bony restoration, soft tissue changes, fractures, dental restorations, and other pathology.

WRITING REPORT &DRAWING CONCLUSION

- CONFIRMS IDENTIFICATION – AM – PM match each other.
- PROBABLE IDENTIFICATION – data is consistent but can not confirm identity.
- POSSIBLE IDENTIFICATION – explainable difference between AM – PM.
- INSUFFICIENT INFORMATION – data is insufficient or minimal.
- EXCLUDES IDENTIFICATION – data are clearly inconsistent.

IDENTIFICATION FROM DENTAL DNA

- PCR – polymerase chain reaction.
- Amplification of highly degraded DNA.
- Unavailability of AM report – comparison of DNA pattern to parent or sibling.
- TYES – nuclear DNA Or Mitochondrial DNA.
- mDNA – each cell will high number of mDNA, inherited exclusively from mother – similar strands are noticed in siblings and many maternal relatives.

AGE DETERMINATION

- The dentition is one of the four systems used in estimating physiologic age.
  - Bone development.
  - Secondary sex characters.
  - Stature and weight.
- Accuracy of an age estimation varies with the age.
- During the development of the teeth, upto about 15 year of age - ± 1

MEDICOLEGAL SIGNIFICANCE OF AGE

- Significance in both civil and criminal cases.

Marriage contracts.

Kidnapping, rape.

Attainment of majority.

Eligibility of employment.

judicial punishment.

Criminal abortion.

Infanticide.

METHODS FOR DETERMINATION OF AGE

1. Visual method
2. Radiographic method
3. Histological
4. Physical and chemical methods

VISUAL METHOD
RADIOGRAPHIC METHOD

1	2	3	4	5	6	7
I_1	I_2	C	PM_1	PM_2	M_1	M_2

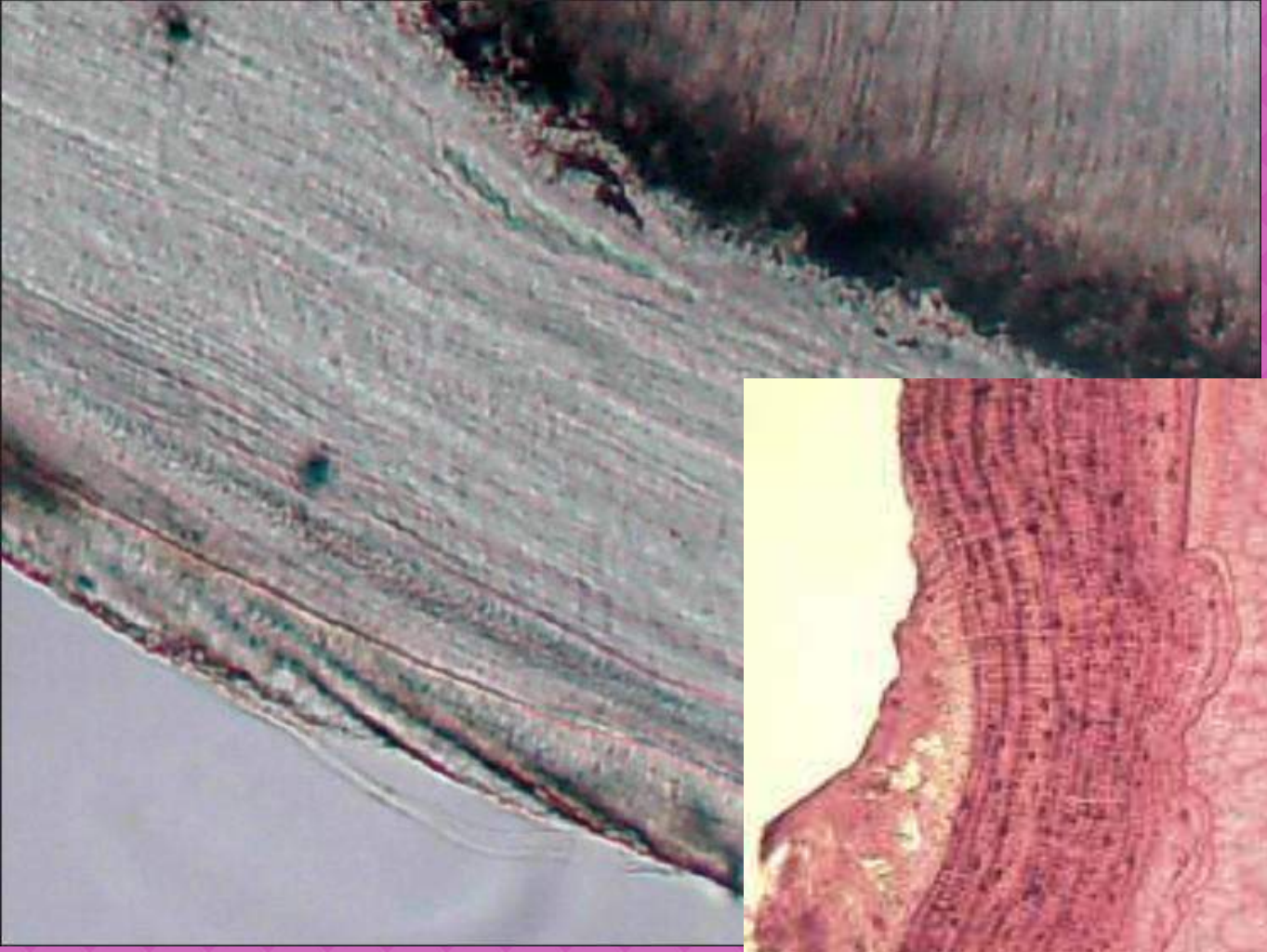
age 6		6						
7		6	1					
8		6	1	2				
9		6	1	2	4			
10		6	1	2	4	5		
11		6	1	2	4	5	3	
12		6	1	2	4	5	3	7

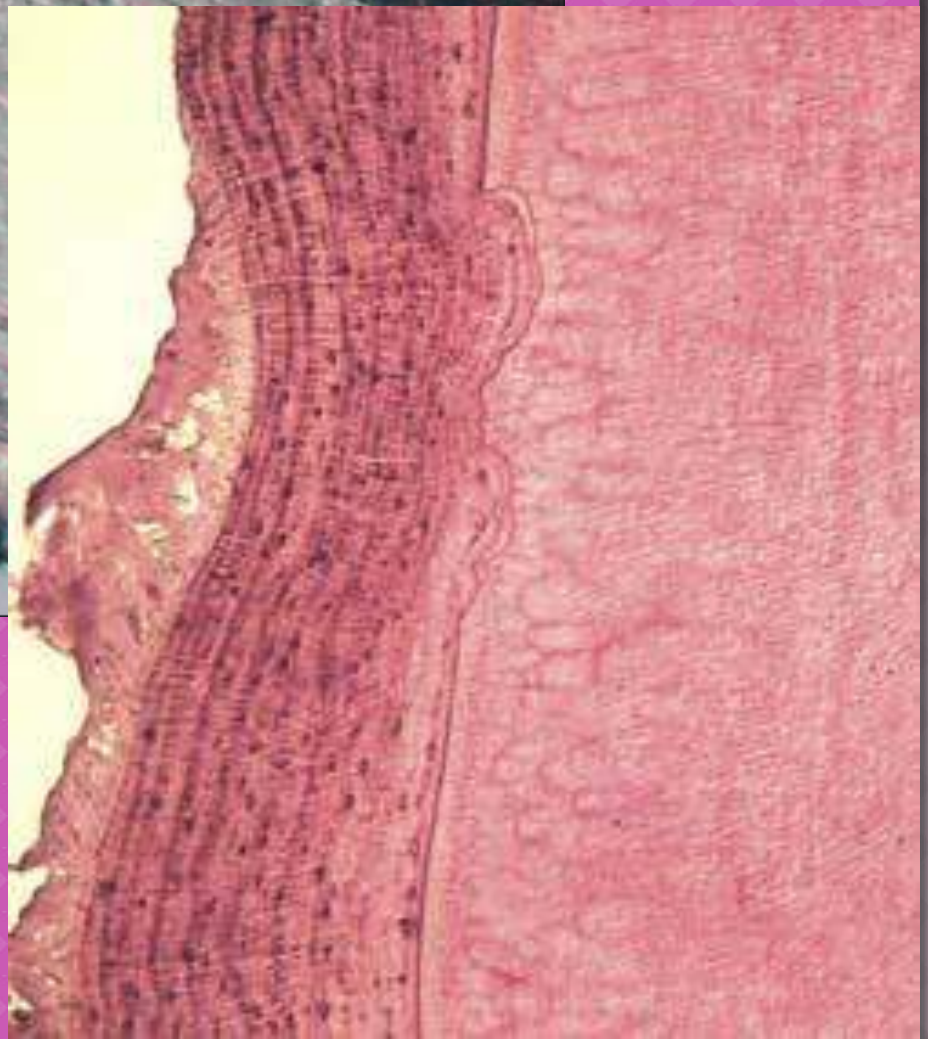
Teeth present

KUSRI'S TRIANGLE

HISTOLOGICAL

1. Commencement of mineralization.
2. Completion of crown formation
3. Completion of tooth eruption
4. Termination of root formation





Cemental lines

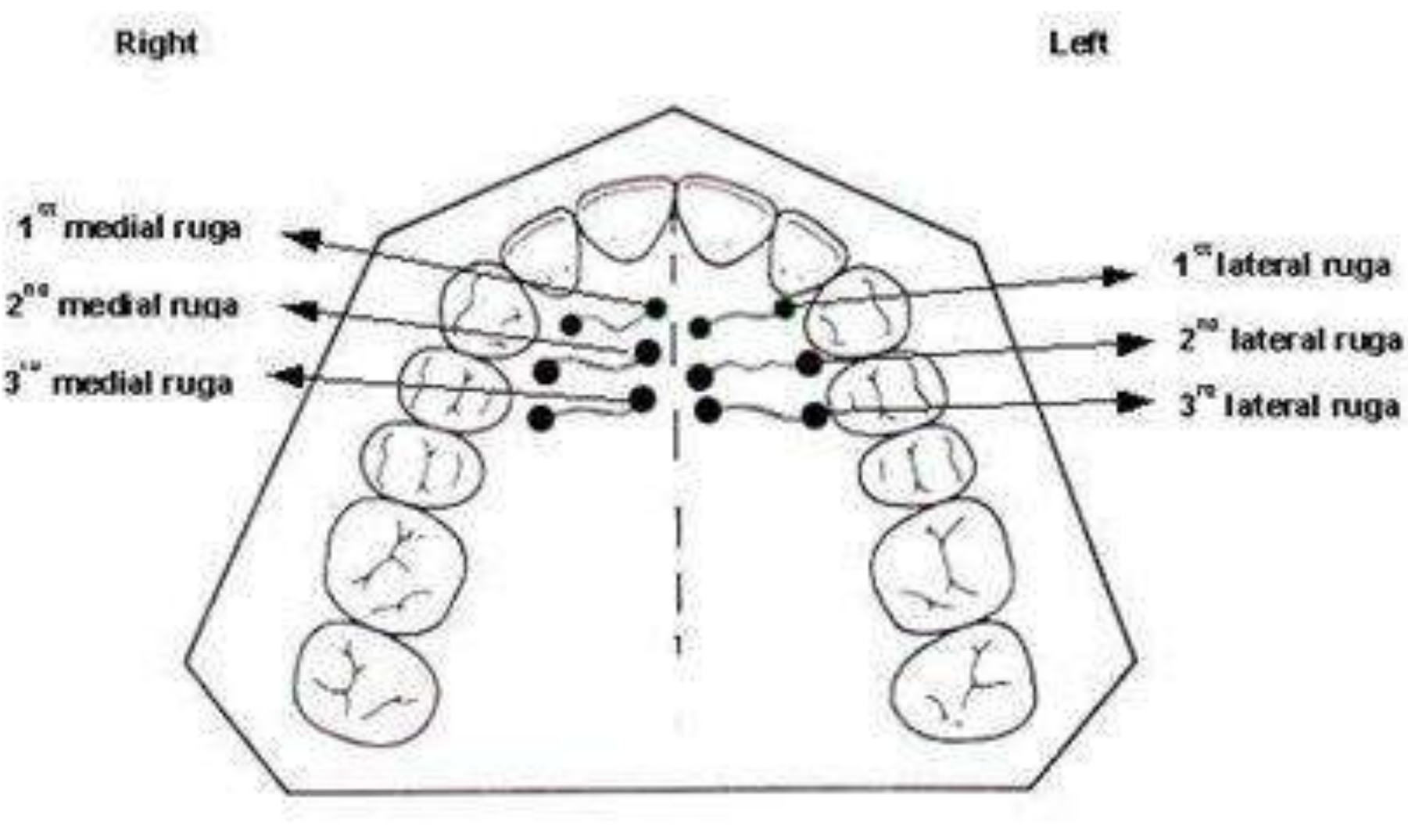
OTHER METHODS

- CALCIUM AND PHOSPHATE RATIO -- electron probe microanalysis of human teeth.
- NUMBER OF DENTINAL TUBULES -- scanned electron microscopy.
- Number of dentinal tubules decreased with increasing age.
- FLUORESCENCE FROM DENTIN AND CEMENTUM
- ROOT CANAL SHRINKAGE

Forensic odontology

Palatal rugae pattern

- Plicae palatinae transversae.
- Rugae palatine.
- Refers to the ridges on the anterior part of palatal mucosa, each side of the median palatal raphe and behind the incisive papilla.
- Useful method- edentulous individual.
- Do not change shape with age and after trauma or surgical procedure.


Right
Left
1st medial ruga
2nd medial ruga
3rd medial ruga
1st lateral ruga
2nd lateral ruga
3rd lateral ruga

Classification

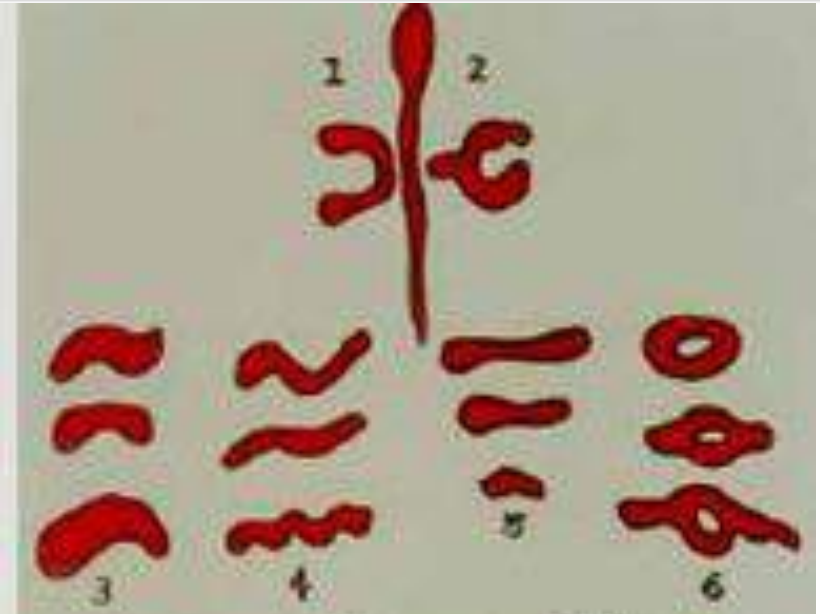
LYSELL

- Primary rugae > 5mm.
- Secondary rugae 3-5mm.
- Fragmentary rugae 2-3mm.

(Rugae < 2mm are not considered)

THOMAS & KOTZE – primary rugae,

- Branched, united, cross-linked, annular, & papillary



Various Shapes of Rugae:

1. Diverge
2. Converge
3. Curve
4. Wavy
5. Straight
6. Circular

Analysis

- Tracing manually on to a clear acetate, then superimposing the tracings on photographs or plaster models.
- Computer software program. – RUGFP-ID.

Lip prints

- Lip prints are the normal lines and fissures in the form of wrinkles and grooves present in the zone of transition of the lips, between the inner labial mucosa and outer skin.
- No anatomical name given yet.
- Analogous to finger prints.
- Personal identification and sex determination.

Types

- Type 1 : Clear-cut vertical grooves that run across the entire lips

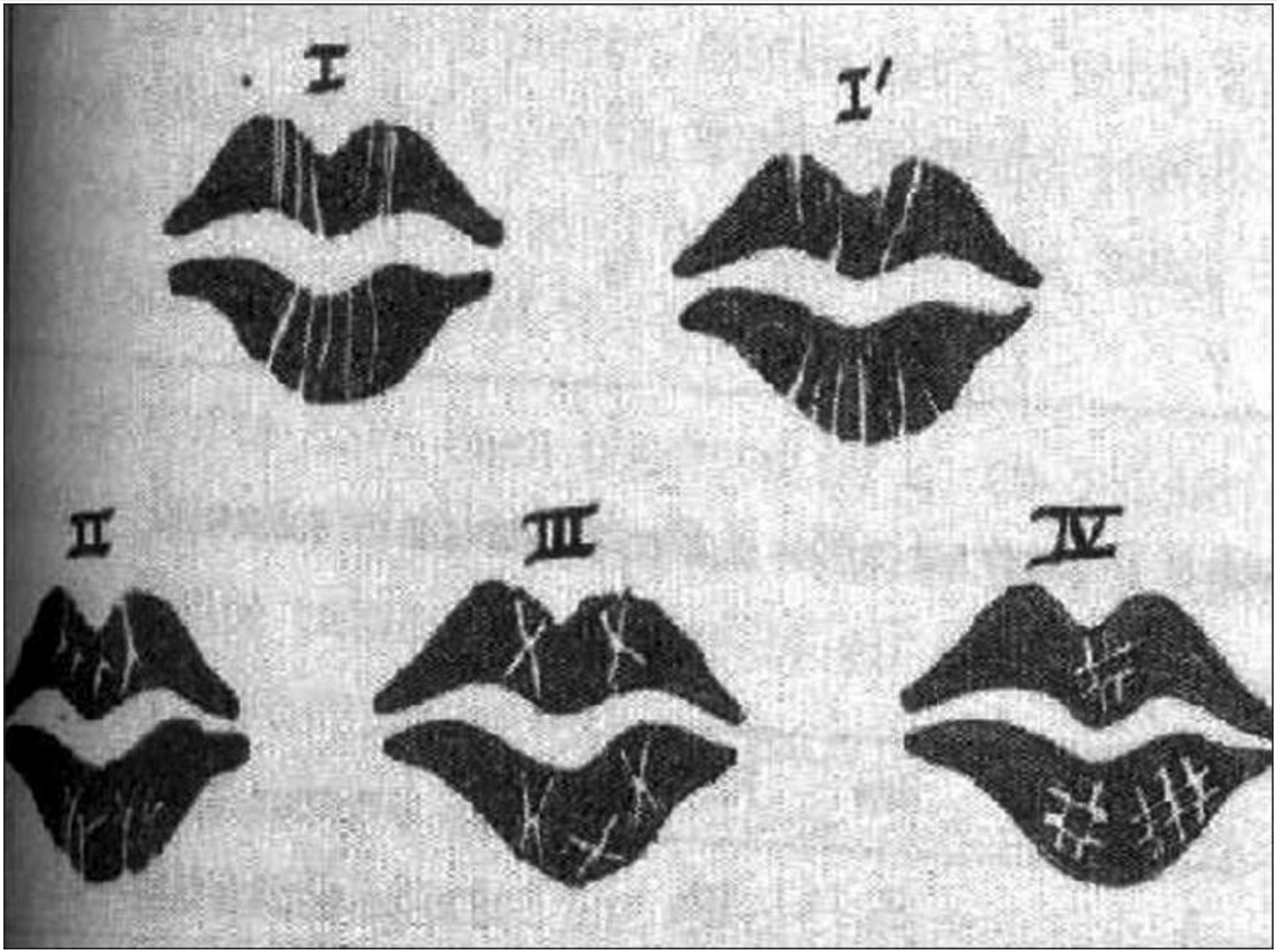
  Type 1' : Similar to type 1, but do not cover the entire lip

  Type 2 : Branched grooves

  Type 3 : Intersected grooves

  Type 4 : Reticular grooves

  Type 5 : Grooves do not fall into any of the type 1-4 and cannot be differentiated morphologically (undetermined).


I
I′
II
III
IV

- Type 1, 1′ : Pattern dominant - Female

  Type 2 : Patterns are dominant - Female

  Type 3 : Pattern present - Male

  Type 4 : Male

  Type 5 : (varied patterns) - Male

  Same patterns in all quadrants - Female

Steps

- Applying lipstick over the lip.
- Sticking cello tape over the lipstick applied area.
- Remove the tape and transfer the impression to a clear sheet.
- Compare the given data with suspect print by magnification method (visual comparison) or by computer.

Bite marks

- A mark caused by the teeth either alone or in combination with other mouth parts.
- Tissue, food stuff or objects.
- Humans or animals.

Differences

- Human bites – U shaped indentations.
- Usually present on breasts, abdomen, nipple, thigh, back & shoulder – sexual assault.
- Other extremities – fight & violence.
- Animal bites – V shaped indentations.
- Prominent canines and narrow centrals.
- Generally on extremities.

FORENSIC EDUCATION SERVICES

Classification

Mc Donald's:

Tooth pressure marks.

- Direct application of pressure by teeth, incisal & occlusal surface indentations.

Tongue pressure marks.

- Sufficient amount of tissue is taken into mouth.
- Tongue pressure on lingual surface of teeth as well as on the palate. – suckling marks.

Tooth scrape marks.

- Scraping of tooth across the bitten material.
- Usually caused by the anteriors & present as scratches or superficial abrasions.

Bite mark investigations

?

- Is the injury a bite mark?
- If yes, was it caused by humans?
- Was it caused by child or adult?
- Age of the marks correspond to the time and type of the crime?
- Any unique features?
- Can these marks be compared or recorded?

Steps

- Visual examination.
- Photography.
- Saliva or swab collection.
- Collecting the bite mark impression.
- Collecting impression of suspect.
- Analysis and comparison.
- Conclusion- positive, possible or exclude.

Sex determination.

- Mesiodistal width of the crown
- Mandibular canine index
- Eruption of deciduous and permanent teeth
- Sex chromatin in the pulp
- Amelogenin gene and the DNA from the pulp
- Cephalometric and craniometric analysis
- Palatal dimensions

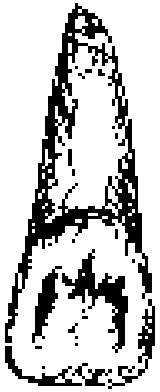
CRANIAL MEASUREMENT USEFUL IN SEX DETERMINATION

	Male	Female
Cranial variables		
Cranial base length	112.5	103
Mastoid height	12.5	9.6
Mastoid width	24	19
Total face height	127	114
Bizygomatic breadth	129	122
Basion-nasion length	102	96
Cranial length	188	179
Nasal height	54	50
Maximum frontal breadth	120	115.5
Mandible variables		
Bicondylar breadth	117	111
Bigonial breadth	100	91.5
Minimum ramus breadth	31	28.5
Gonion-gnathion length	77	73
Total mandibular length	111	108

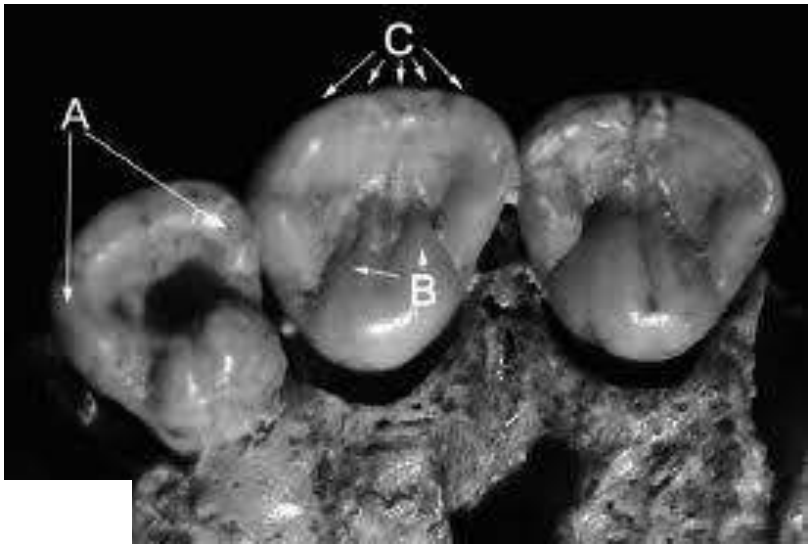
ODONTOMETRIC DIFFERENCES

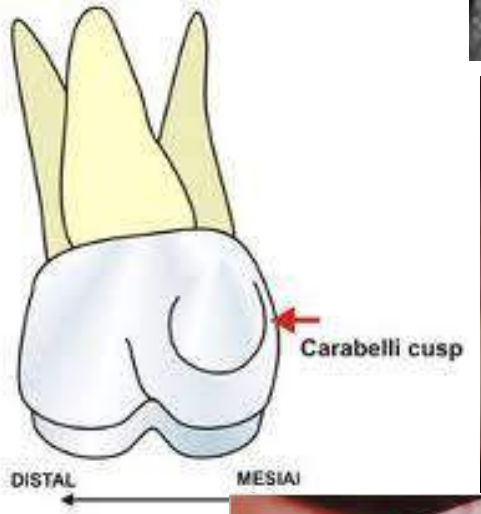
Tooth No.	Mesiodistal		Buccolingual	
	(♂)	(♀)	(♂)	(♀)
11	8.9	8.5	7.1	7.0
12	7.0	6.65	6.5	6.2
13	8.3	7.6	8.4	7.9
14	6.9	6.8	9.3	8.9
15	6.7	6.65	9.8	9.3
16	11.0	10.6	11.0	10.9
17	10.4	9.9	11.0	10.7
41	5.5	5.3	6.2	6.1
42	6.1	5.9	6.5	6.5
43	7.2	6.6	7.55	7.4
44	7.1	7.0	7.9	7.6
45	7.4	6.9	8.6	8.2
46	11.1	10.8	10.4	10.2
47	10.5	10.2	10.3	9.9

	Male	Female
Skull features		
1. Architecture	Rugged	Smooth
2. Frontal and parietal eminences	Small	Large
3. Forehead	Sloping	Vertical
4. Supraorbital ridges	Well developed	Absent or slightly developed
5. Glabella	Prominent curvature	Relatively flat
6. Nasion	Marked depressed	Rarely depressed
7. Orbits	Squarish, with rounded margins	Rounded, with sharp margins
8. Cheek bones	Bulky and laterally arched	Lighter, compressed
9. Posterior root of zygoma	Well developed	Slight
10. Supramastoid crest	Marked development	Faint or absent
11. Mastoid process	Large	Small
12. Mastoid crest	Slight to moderate	Marked
13. Tympanic plate of temporal bone	Well developed	Delicate
14. Occipital condyles	Large	Small
15. Glenoid fossa	Deep	Shallow
16. Digastric fossa	Broad	Narrow
17. Palate	Larger, V-shaped	Smaller, parabolic shape
Mandible features		
18. General features	Larger, thicker; broader ascending ramus	Smaller, thinner; narrower ascending ramus
19. Condyles	Larger	Smaller
20. Body height	Greater depth at symphysis	Shallow at symphysis
21. Shape of chin	Squarish	Rounded/pointed
22. Gonial angle	Less obtuse	More obtuse



Shovel- Shaped Incisor





Thank – U

Practical Application of Forensic Odontology

Forensic Odontology

- Forensic Odontology or Forensic Dentistry is the proper handling, examination and evaluation of dental evidence, which will be then presented in the interest of justice.
- Evidence
  - Age(especially in children)
  - Identification of the person, etc

APPLICATIONS

- Age Estimation
- Injuries – S. 320 IPC
- Identification of the deceased
  - Unknown bodies
  - Mass disaster cases
- Bite Mark examination & Identification
- Dental Malpractice litigations

AGE ESTIMATION

AGE ESTIMATION IN ADULTS

- GUSTAFSON'S METHOD
- DENTIN TRANLUCENCY
- AMINO ACID RACEMISATION

GUSTAFSON'S METHOD

- This method is based on morphological and histological changes of the teeth.
- This assessed regressive changes such as:

a) Amount of occlusal attrition(A)

b) Coronal secondary dentine deposition(S)

c) Loss of periodontal attachment(P)

d) Cementum apposition at the root apex(C)

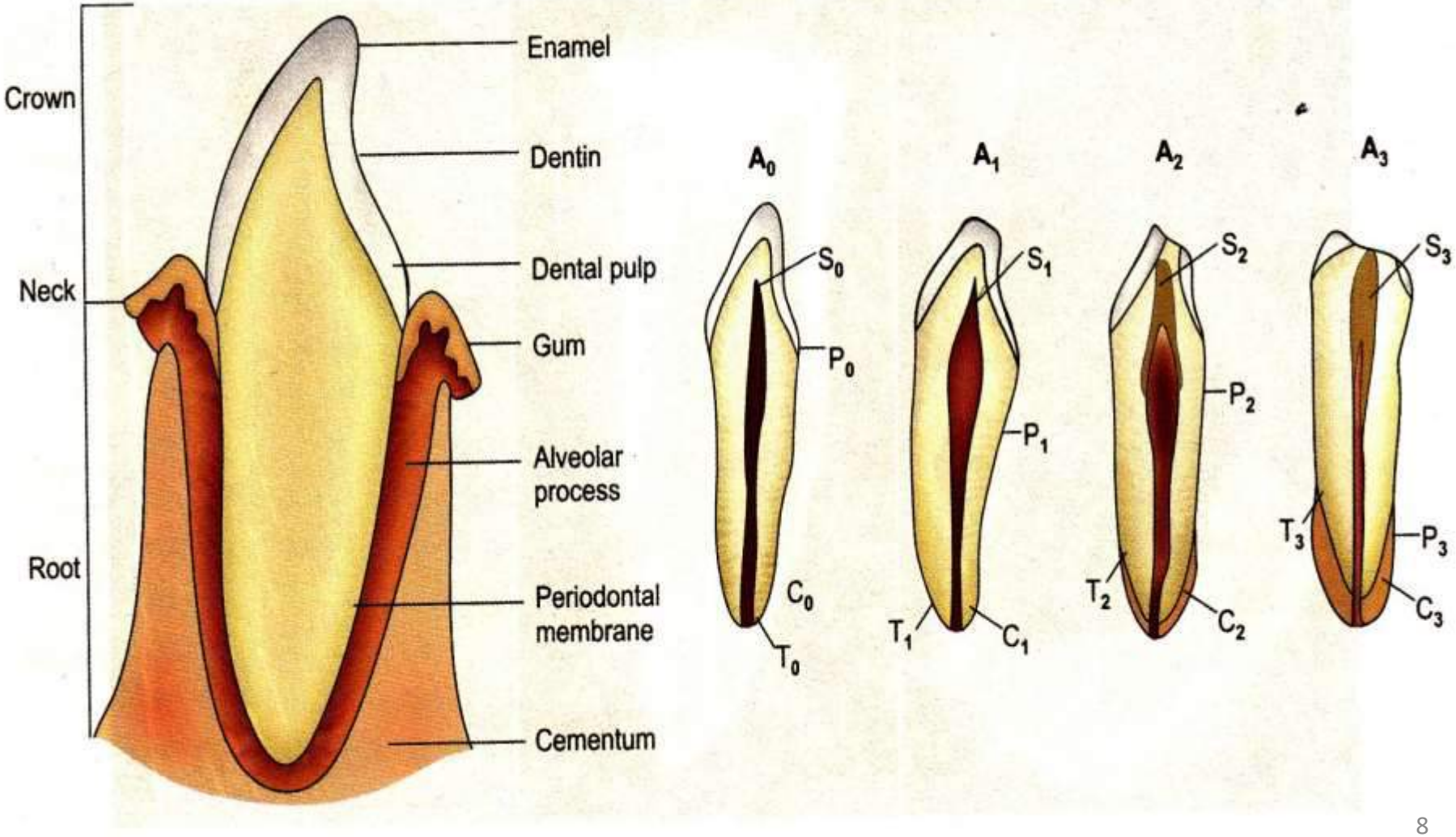
e) Root resorption at the apex(R)

f) Dentine translucency(T)

Age from teeth >25 yrs of age

- Gustafson' s Method
  - Attrition
  - Periodontosis
  - Secondary dentine deposition
  - Cementum apposition
  - Root resorption
  - Root translucency

Gustafson's Method: Schematic



No Translucency

(Score- 0)

Translucency
Lower 1/3rd

(Score – 1)

Translucency
2/3rd of root

(Score – 2)

Translucency
whole root

(Score – 3)

VARIOUS STAGE OF ROOT TRANSLUCENCY AND ITS SCORING

DENTIN TRANSLUCENCY

Gustafson (1950) first observed that root dentin becomes transparent with age.

Root dentin is considered to become translucent during third decade of life, beginning at the apex and advancing coronally.

The root dentine translucency appears to be related directly to chronological age.

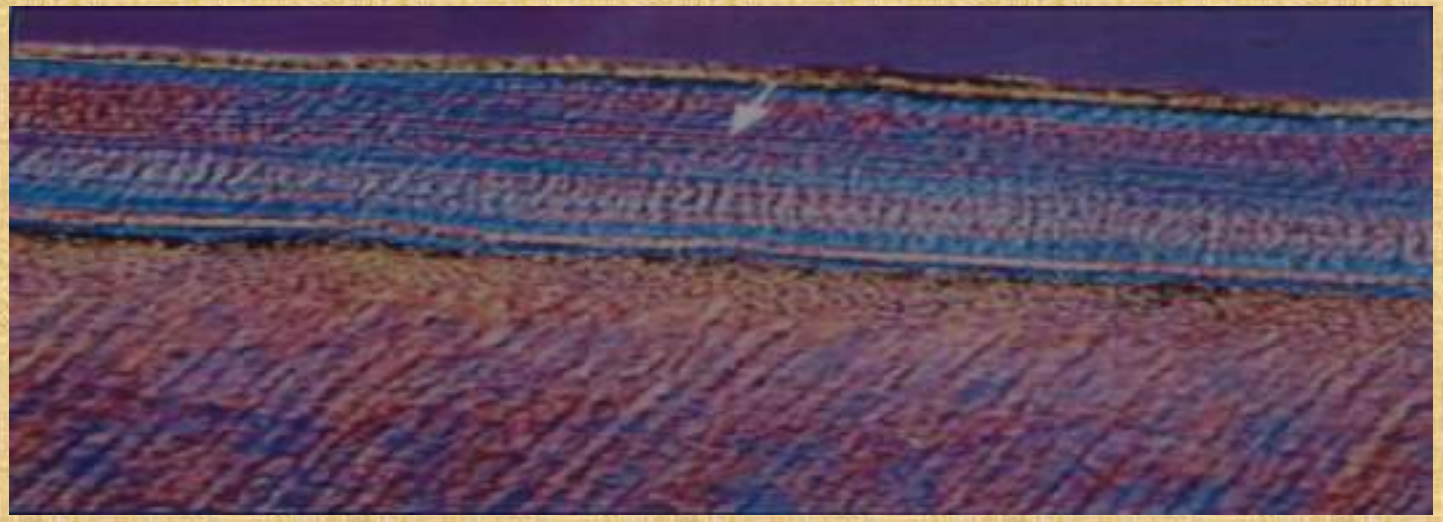
Age= B0+B1X where B0 –regression constant
B1-regression coefficient
X-length of translucency

Tooth section from the three age groups depicting progressive increase in translucency with age·

AGE ESTIMATION FROM INCREMENTAL LINES OF CEMENTUM

- The premise for the incremental lines analysis is based on the fact that these lines have the same pattern in an individual whose enamel formed at the same time in a given dentition.

- The different teeth developing in one individual give the same pattern of incremental lines creating a "FINGERPRINT" of Enamel development specific to an individual.



Arrow depicting Cementum Annulations in polarized microscope.

- **Age of an individual is calculated by adding the number of incremental bands to that of an eruption age of individual tooth.**

- **Counts of cemental annulations can be a mean of age determination.**

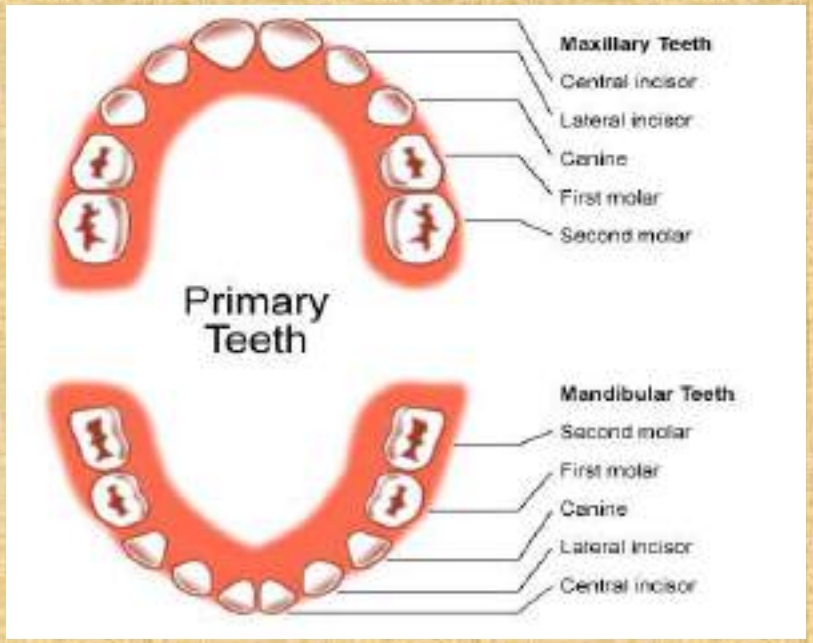
- **Cementum thickness and number of annulations increase with age.**

Age Estimation

- Eruption of teeth
- Two set of teeth
  - –Temporary or Deciduous
  - –Permanent
    - Successional teeth
    - Superadded teeth

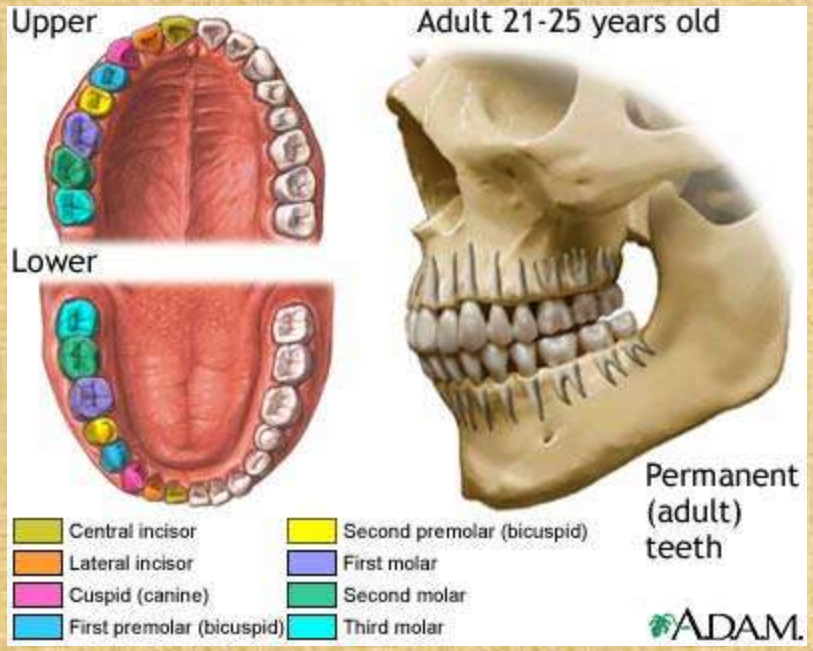
Teeth through the years

Childhood



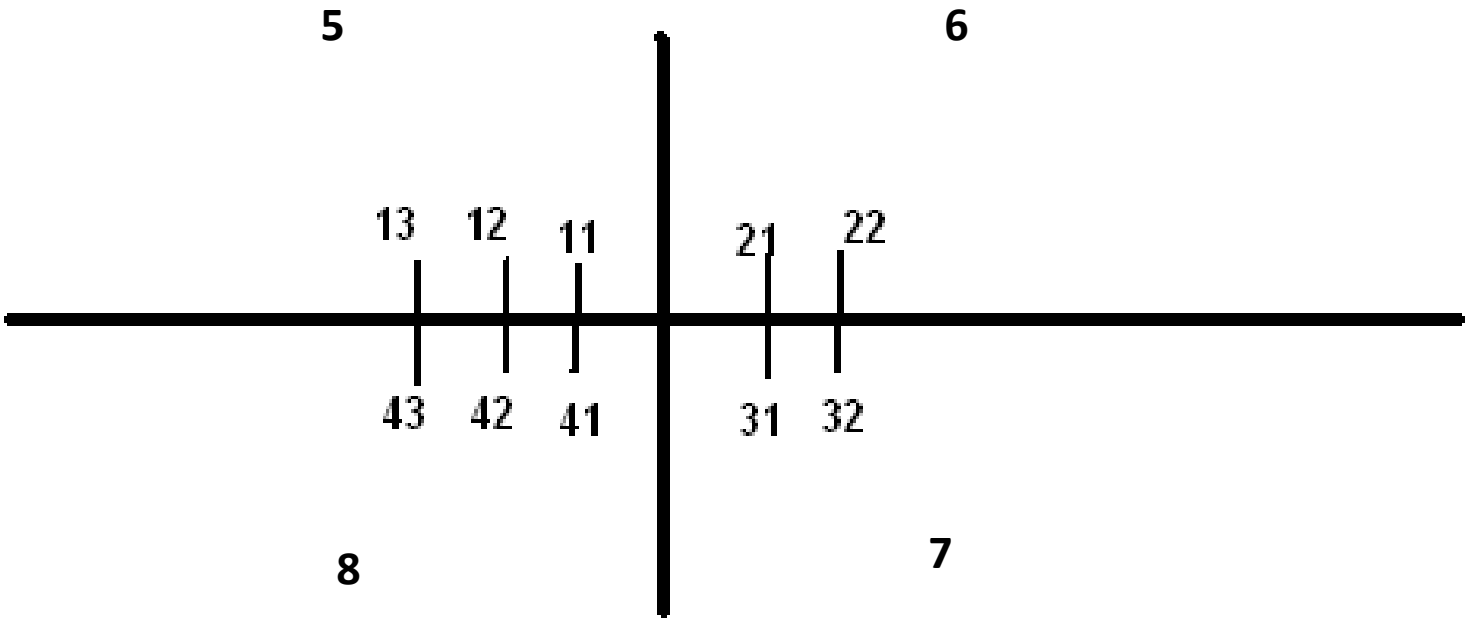
- **Primary teeth sprout from milk buds and are temporary. Once they fall out, permanent teeth as seen on the other side appear.**

Adulthood



- **Permanent adult teeth come in when primary teeth fall out; they are permanent because they establish roots inside the gums. Third molar come in around the mid teenage years.**

Dental Formula



FDI SYSTEM OF DENTAL FORMULA

Age Chart - Deciduous & Permanent teeth

Teeth	Temporary (Mth)	Permanent (Yrs)
Central Incisors	L 6–8 U 7-9	6 – 8
Lateral Incisors	L 10-12 U 7-9	7 – 9
Canines	17 – 18	11 – 12
1 st Premolars	Absent	9 – 11
2 nd Premolars	Absent	10 – 12
First Molars	12 – 14	6 – 7
Second Molars	20 – 30	12 – 14
Third Molars	Absent	17 – 21

Period of mixed dentition

- 6 to 12 years of age
- 21 – 24 teeth

Radio. Assessment - Teeth Development

- **Orthopantomogram-** provided facility available

- X-ray of oral cavity - **Oblique tangential view with mouth open at 90^0**

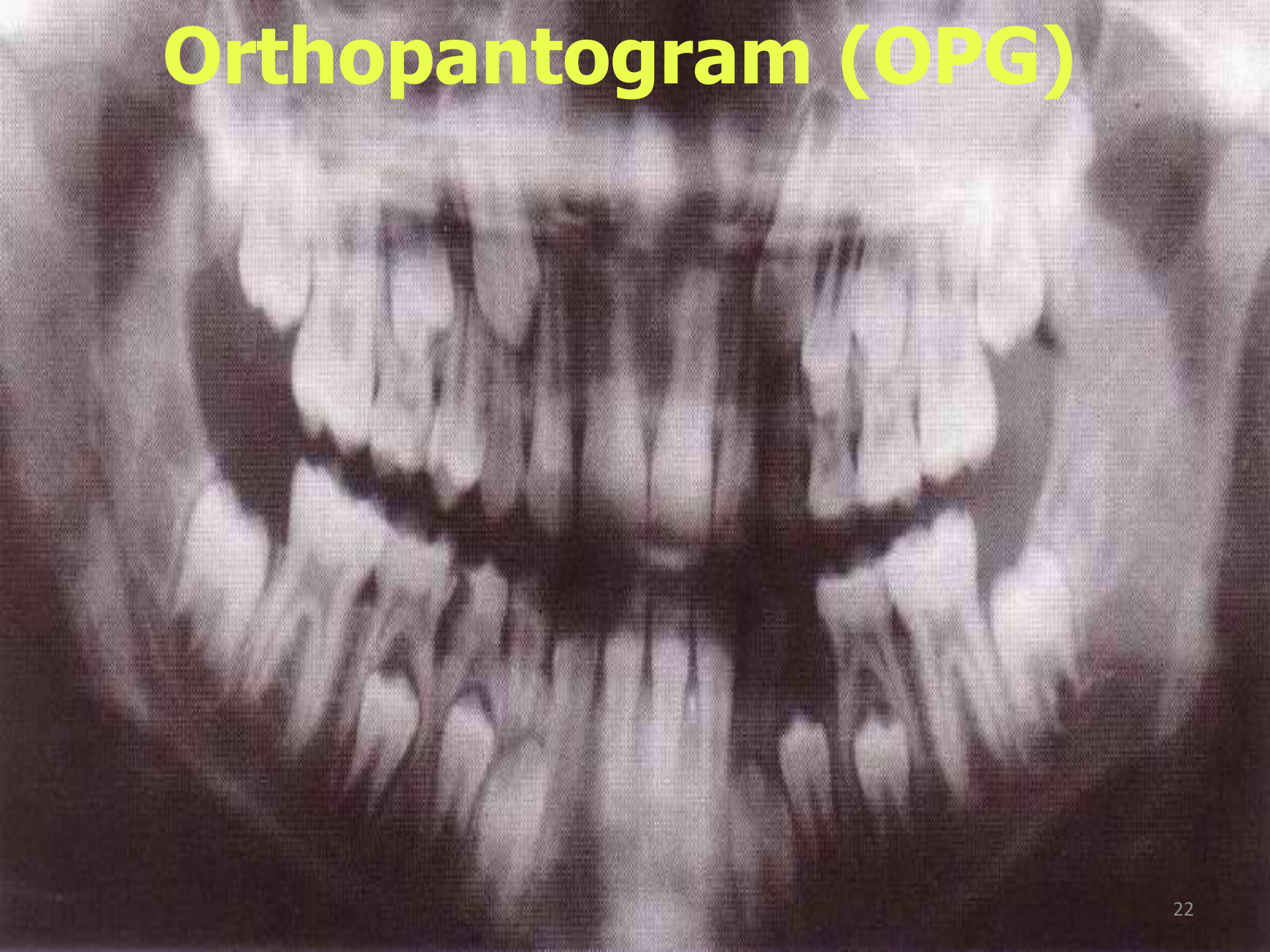
- Teeth develop earlier in females by 1 yr

Crown & Root Development of Third Molar

Crown Formation (On X-ray)	**Age (Yrs)**
• Complete Crown Formation	15
• Crown with 1/3rd Root Formation	16
• Crown with 2/3rd Root Formation	17
• Crown with complete Root Formation	18
• Apical Closure of Root (Calcification of root complete)	21

X-RAYS

Orthopantogram (OPG)



INJURY

Whether injury Grievous or not ?

Fracture/ Dislocation of tooth

- Total oral hygiene - condition of gums, looseness of tooth, disease conditions, bleeding gums
  - Taken in to account before forming opinion

- Each case decided on its merit

- Based on scientific facts and & is unbiased

IDENTIFICATION

Dental comparison

- Dental records are useful in helping to identify unknown persons
  - who have been the victim of foul play or
  - who have been simply reported missing
- The Forensic expert / Dentist compares antemortem records with postmortem findings to determine if there is a positive match

Individual Characteristics

- Size of tooth
- Shape of tooth
- Shape of root
- Placement of tooth
- Quantity of teeth
- Combinations of dental work done:
  - Crowns
  - Extractions
  - Bridge
  - Fillings
  - Root canals

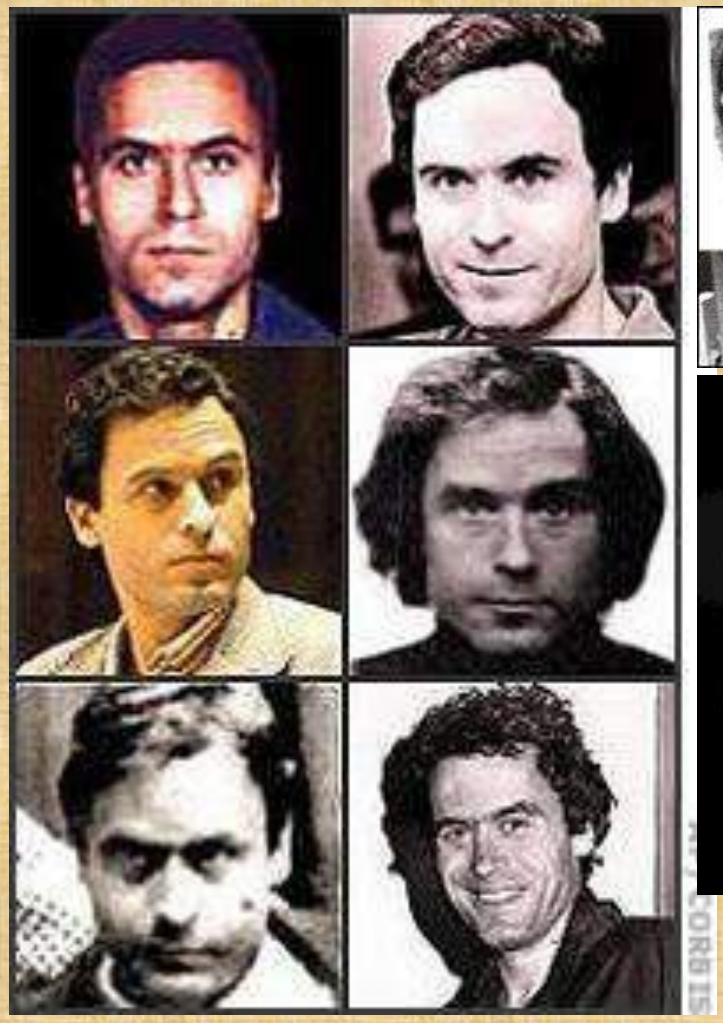
Various dental work

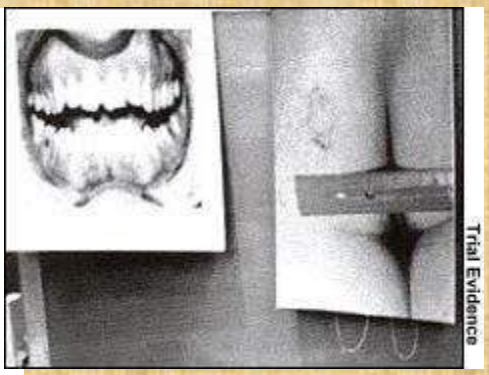
Other information from teeth

- Sex from teeth
- Race from teeth
- Occupation from teeth
- Social status
- Teeth used as weapon of offence

BITE MARKS

State of Florida v. Ted Bundy





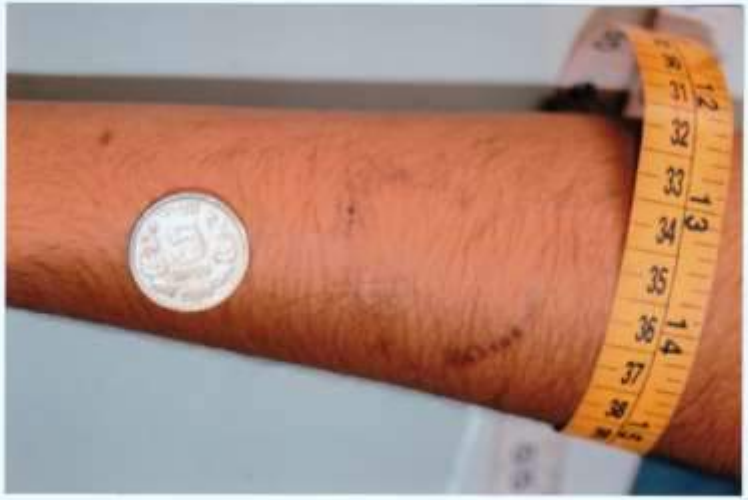
Ted Bundy, was an American serial killer who murdered numerous young women between 1974 and 1978.

He confessed to 30 murders, however the total amount of victims remains unknown. He would bludgeon his victims, then strangle them to death. He engaged in rape and necrophilia.

Where are bite marks found?

- Impressions left on food, skin or other items left at scene
  - Impressions vary
    - Depending on the pressure applied, the impression will show accordingly.
    - More the pressure, the more detail to bite mark

Various Bite Marks



Can you tell the differences of these bite marks?

Things to note….

- The shape or curvature
- No. of tooth marks
- Horizontal diameter
- Vertical diameter
- Depth of depression
- Distances between two teeth
- Orientation of each tooth

BITE MARKS

There are five (5) types of bite marks which can be classified by four (4) degrees of impression:

1. **hemorrhage -- small bleeding spot**
2. **abrasion -- undamaging mark on skin**
3. **contusion -- ruptured blood vessel, bruise**
4. **laceration -- punctured or torn skin**
5. **avulsion -- removal of skin**

I. **clearly defined -- significant pressure**

II. **obviously defined -- first-degree pressure**

III. **quite noticeable -- violent pressure**

IV. **lacerated -- skin violently torn from body**

***Possible Bitemark*:** An injury showing a pattern that may or may not be caused by teeth; could be caused by other factors but biting cannot be ruled out.

- *Criteria:* general shape and size are present but distinctive features such as tooth marks are missing, incomplete or distorted or a few marks resembling tooth marks are present but the arch configuration is missing.

***Probable Bitemark*:** The pattern strongly suggests or supports origin from teeth but could conceivably be caused by something else.

- *Criteria:* pattern shows some (basic/ general) characteristics of teeth arranged around arches.

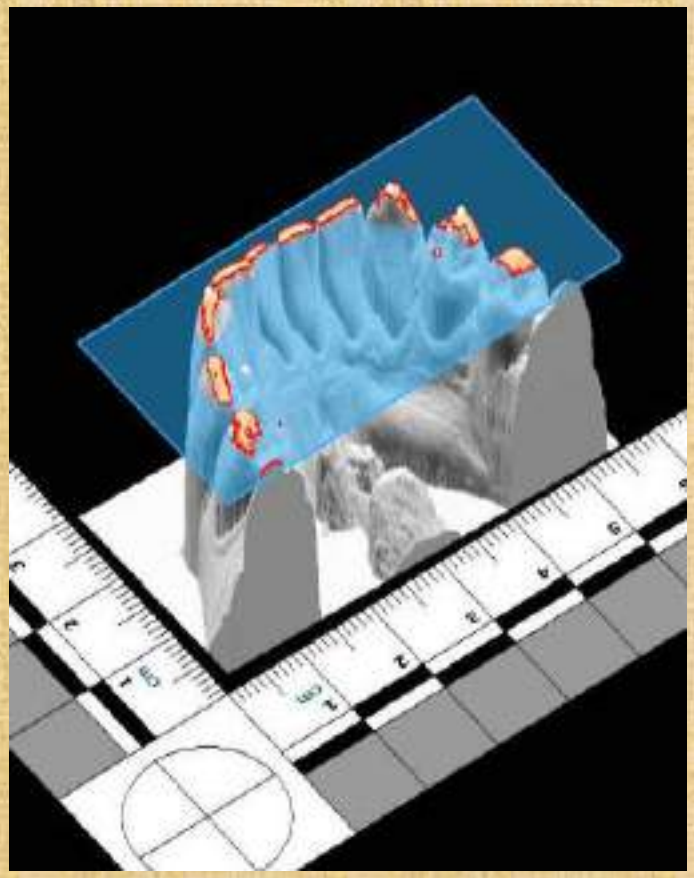
***Definite Bitemark*:** There is no reasonable doubt that teeth created the pattern; other possibilities were considered and excluded.

- *Criteria*: pattern conclusively illustrates classic features (all the characteristics) of dental arches and human teeth in proper arrangement so that it is recognizable as an impression of the human dentition.

Analyzing bite marks

- Bite marks are photographed with a scale
- Casts of impression are taken
- Impression traced onto transparencies
- Casts of suspects teeth are taken
- Comparison between suspect cast and bite mark

Computer Odontology



- Automatic dental code matching
  - Bites are run through the computer to find a match
- OdontoSearch
  - Compare a data base of missing peoples, felons, government workers
- Automatic dental identification system
  - Produce a list of people having same dental code No.
- 3D Bite mark analysis
  - 3D scans of dental casts used to generate overlays
  - The overlays are compared with the photograph of the bite marks.

DENTAL IDENTIFICATION PROCEDURES

COMPARATIVE IDENTIFICATION

STEPS:

- Oral autopsy
- Obtaining dental records.
- Comparing post and antemortem dental data.
- Writing the final report
- Drawing the conclusion

RECONSTRUCTIVE IDENTIFICATION (DENTAL PROFILING)

- When Ante-mortem Records Are Not Available
- Includes A Triad Of Information → ethnic Origin , Gender , Age Etc
- Information From This Process Will Enable A More Focussed Search For Ante-mortem Records

DENTAL PROFILING

INDICATIONS

- When ante mortem records are not available.
- When circumstantial evidence may not give an indication about the putative identify of the deceased, in which case dental records are not traceable.
- Also to confirm known individuals

ROLE OF DENTAL PROFILING IN IDENTIFYING UNKNOWN INDIVIDUAL

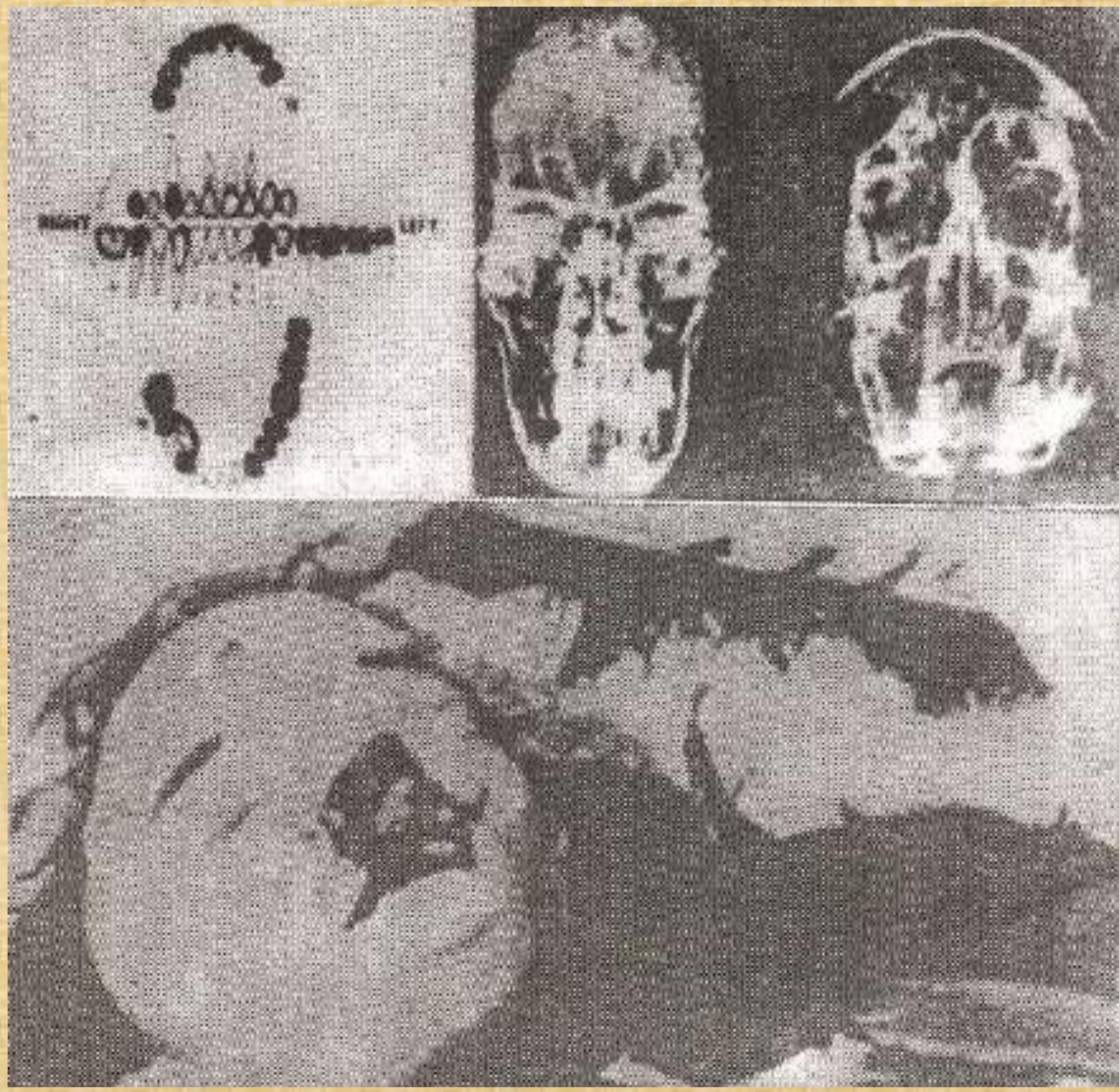
- Dental profiling is used not only to establish the identity of an unknown individual, but also to confirm the identity of a known person, which was done in cases of Saddam Hussein, Muammar Gaddafi and Osama bin Laden.

ADOLF HITLER

Identified by: Kathe Echtmann

Forensic odontology

Dental evidence played a vital role in the historical case of identification of Adolf Hitler



RAJIV GANDHI

Methods Of HUMAN Identification

- Using enamel
- By dental DNA
- By anatomy of the tooth- Odontometry
- Using skulls- Anthropometry

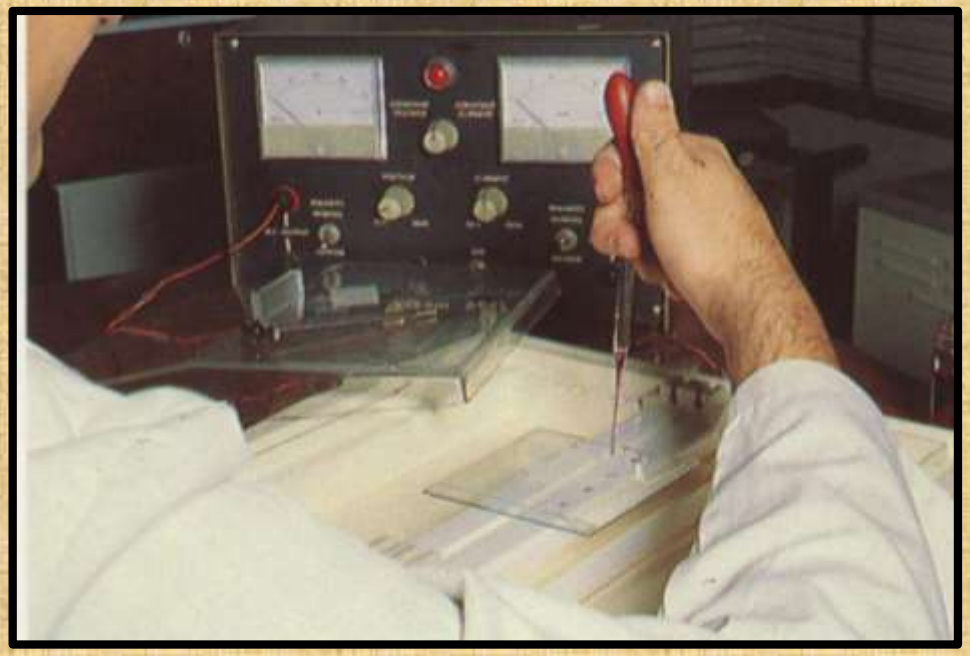
Using Enamel

- **Amelogenesis- has a different site & pattern of nucleotide sequence in male & female enamel.**
- **'Amel' gene-located on the Y chromosome**
  - **Males-2 different amel genes- XY**
  - **Females-2 same amel genes- XX.**

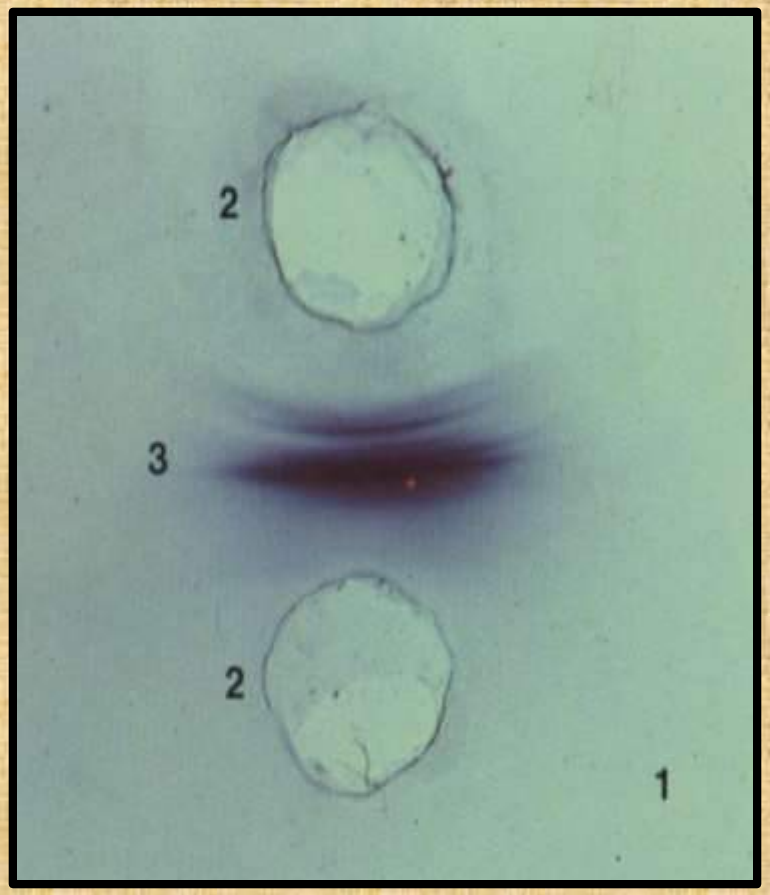
Species Identification

Using Dentine:

- A fragment containing less than 1 cu. mm of dentine will contain identifiable antigens.



Precipitation Reaction



Fluid extracted from dentine

↓

Placed in circular wells in electrophoresis gel

↓

Suitable diluted antibodies from known species placed in adjacent wells

↓

Post reaction occurs when correct antibody reacts

Methods Of using Dental DNA

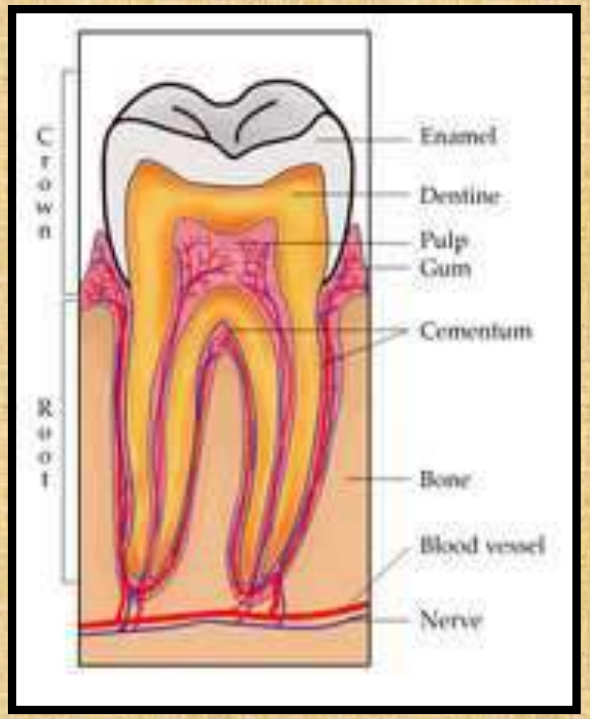
- The identification of sex using dental DNA is a very sophisticated method as compared to the methods used earlier.

Tooth As A Source

In the tooth DNA may be found in :

- Pulp-coronal & radicular
- Odontoblastic processes
- Accessory canals
- Adherent bone
- Periodontal ligament fibers

Also from personal articles-like tooth brush



Other DNA sources:

- Blood & blood stains
- Semen & seminal stains
- Hair & nails
- Saliva, urine & other biological fluids
- Tissues & organs

From hair

From saliva

Techniques Of Obtaining DNA

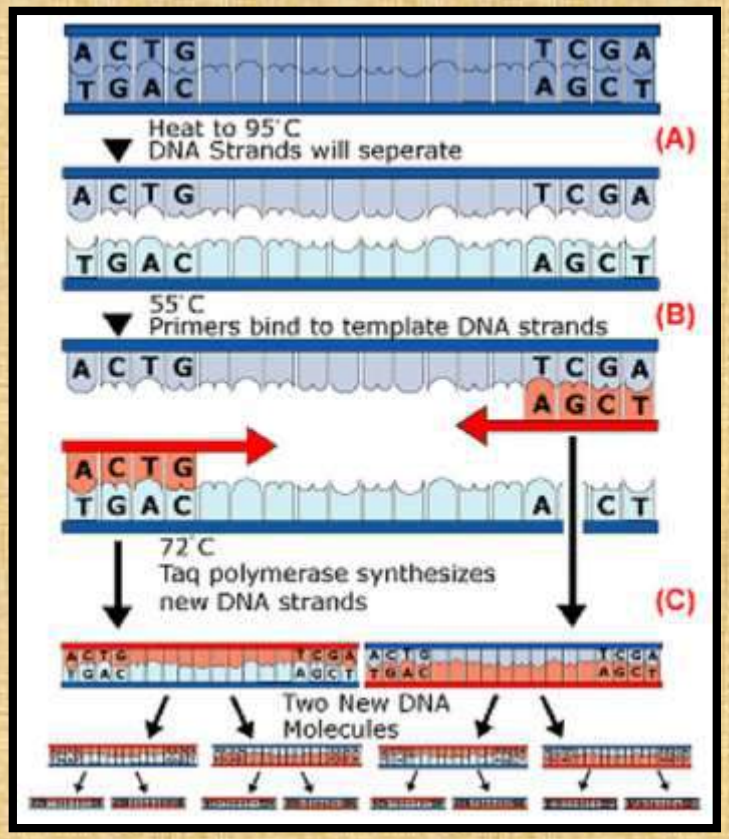
1st given by Smith:

- ***Crushing the entire tooth***
- ***Conventional endodontic access***
- ***Vertical split***
- ***Horizontal split***

Methods Of DNA Identification

- **Restriction fragment length Polymorphism- RFLP**
- **Amplified restriction fragment length Polymorphism - Amp RFLP**
- **Short tandem repeats**
- **Polymerase chain reaction**

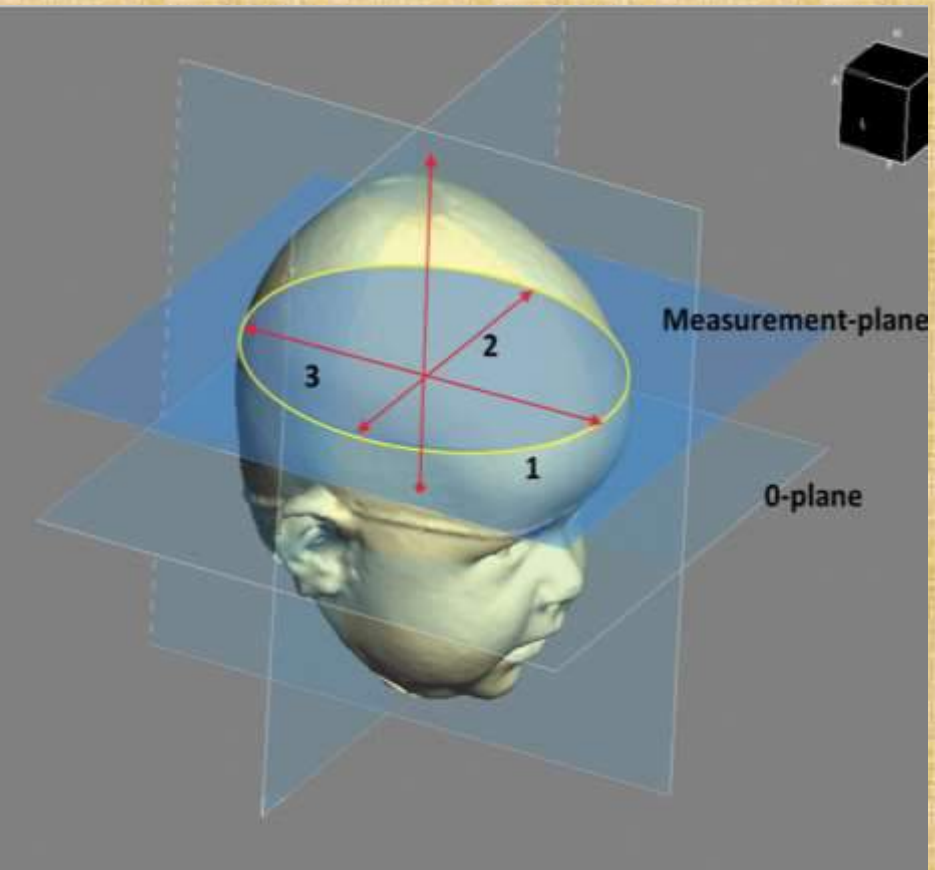
PCR



Short Tandem Repeats

Skull anthropometry using cephalometry

Skull anthropometry alongwith cephalometric analysis plays a vital investigative role in legal and forensic odontology.



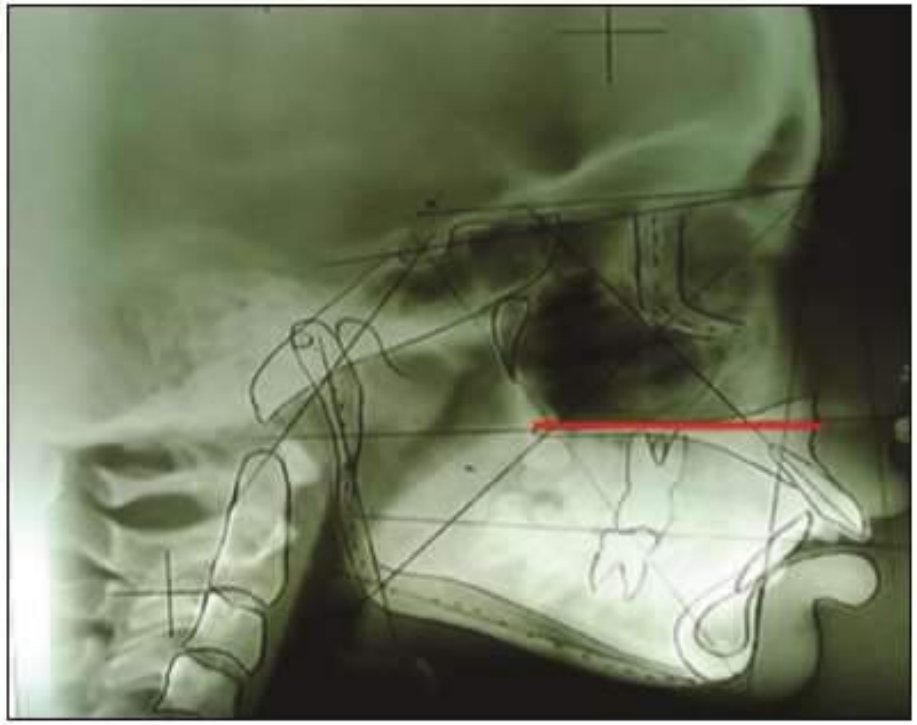


Figure 1: Maxillary extent using Rakosi Jarabak analysis on lateral cephalographs

a
b

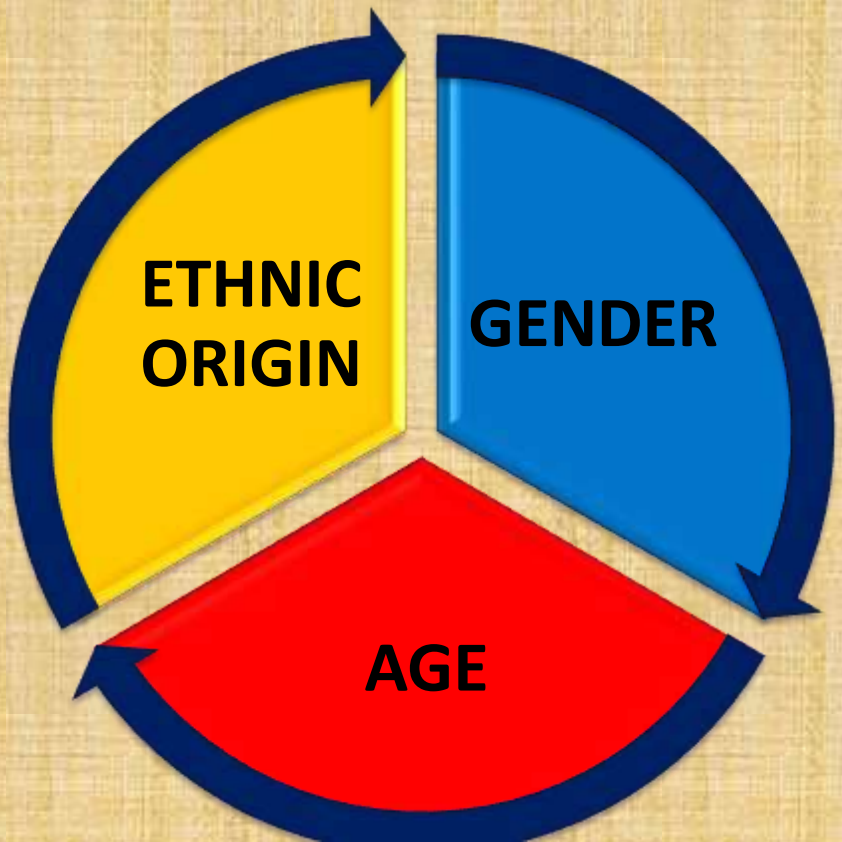
TRIAD OF DENTAL PROFILING

- **Dental profiling includes extracting a triad of information**

1) THE DECENDENT'S ETHINIC ORIGIN

2) GENDER

3) AGE



IDENTIFY ETHNIC ORIGIN FROM TEETH

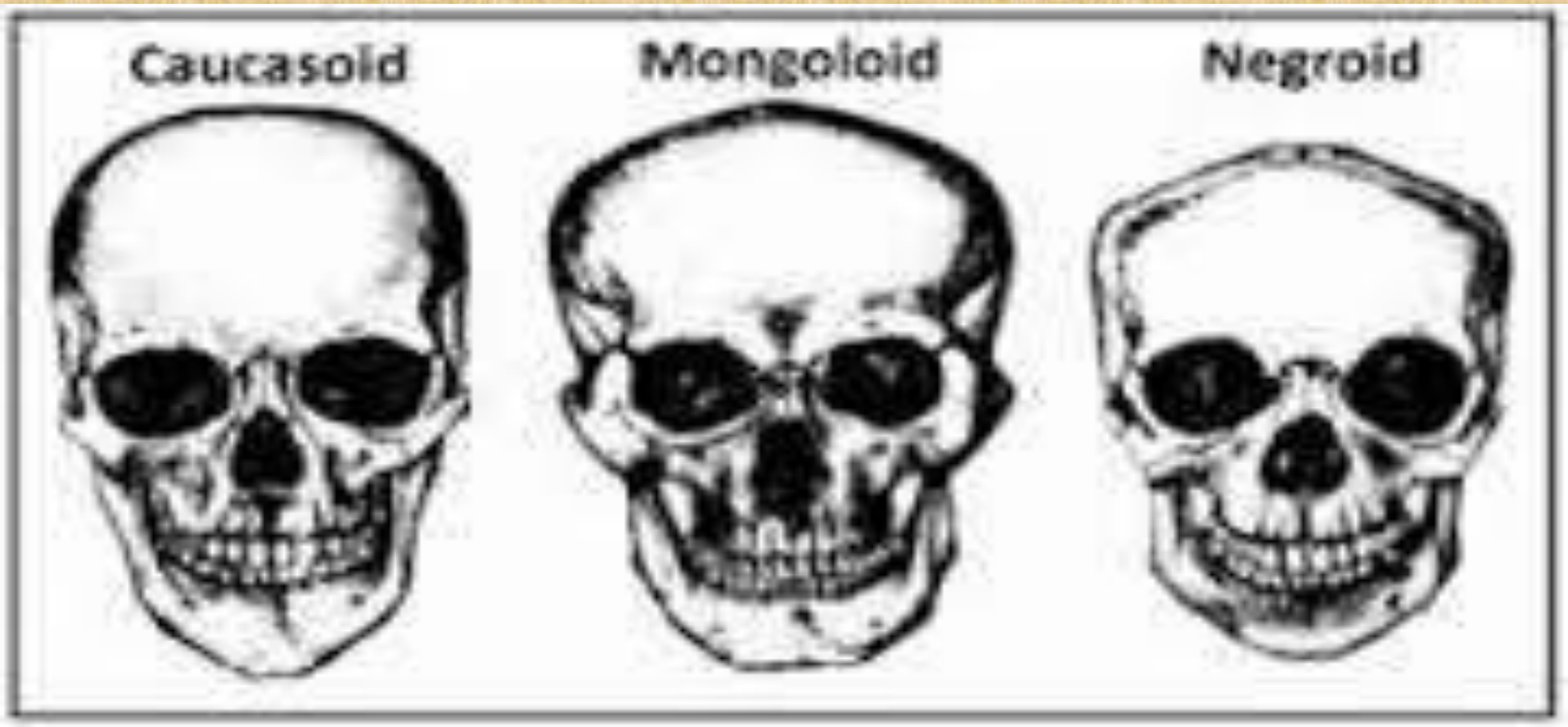
- **Human races have been divided into 3 races-**

1.Caucasoid
2. Mongoloid
3. Negroid

Characteristics	Caucasoid	Negroid	Mongoloid
General appearance	High, wide	Long, low	Rounded
Skull length	Varied	Long	Long
Skull breadth	Varied (often broad)	Narrow	Broad
Skull height	Medium to high	Low	Medium
Sagittal contour	Round to arched	Flat	Arched
Face breadth	Narrow to wide	Narrow	Very wide
Face height	Medium to high	Low	High
Cheek	No projection	Some lateral projection	Anteriorly projected
Orbital opening	Round to angular	Rectangular	Rounded
Facial profile	Orthognathic	Prognathic	Medium to flat
Nasal opening	Narrow to medium	Wide	Narrow to wide
Lower nasal margin	Sharp	Smooth and/or guttered	Sharp
Nasal profile	Straight	Downward slant	Straight
Palate shape	Narrow to wide	Wide	Moderately wide

M C N

Nasal spine	medium	prominent, straight	Reduced
Nasal sill	medium	sharp	dull/absent
Incisor form	shoveled	blade	Blade



Caucasoid incisor

Mongoloid incisor

- It is possible today to identify an individual's ethnic origin based purely on one's dentition

GENETIC & ENVIRONMENTAL INFLUENCE ON TOOTH

- **Dental features have complex mode of inheritance and are combination of heredity & environmental factors to which person is exposed.**

Dental features in population divided into-

1) METRIC(tooth size)

2) NON METRIC(tooth shape)

METRIC DENTAL TRAITS

- It is based on measurements.
- It is influenced by intra oral environmental factors.

EXAMPLES-

- Missing lateral incisors cause compensatory increase in central incisors.
- Space constraints in jaws result in compression of third molar.

NON-METRIC DENTAL TRAITS

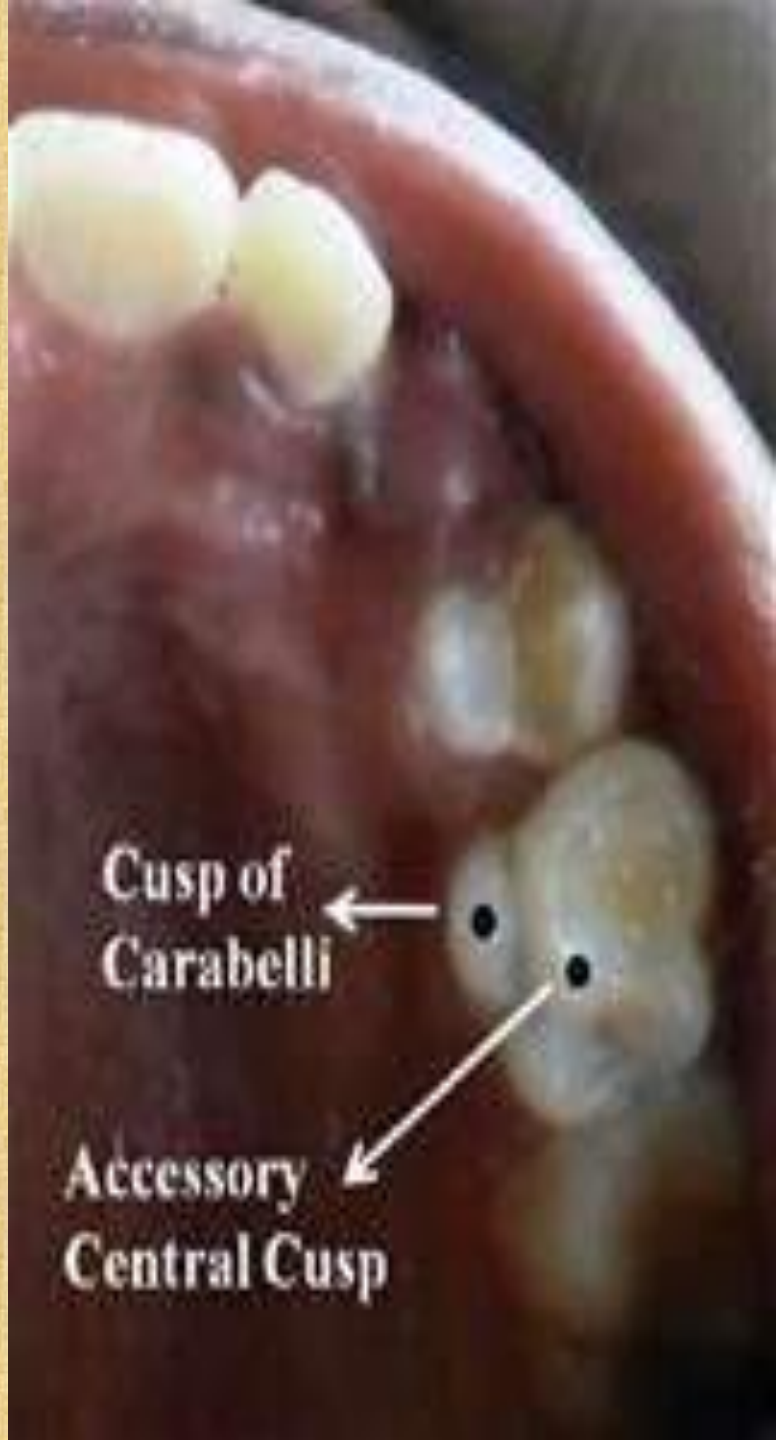
- **Is defined in terms of presence and absence of particular features.**

1. **SHOVELING- presence of prominent mesial & distal marginal ridges on the lingual surface of the maxillary & mandibular anterior teeth.**

2. CARABELLI TRAIT

It is a cingular derivative expressed on the mesiolingual or aspect of the mesiolinguallingual cusp of maxillary 1st molar.

The Trait may be absent or expressed as a minor depressions or well developed tubercules with free apexes.



3. THREE CUSPED MAXILLARY SECOND MOLARS-

The distolingual (distopalatal) cusp of maxillary second molar tends to be reduced size or absent.

4. WINGING-

It is characterized by bilateral labial rotation of the distal margins of maxillary central incisors, the incisal edge appears (V) shaped from occlusal aspect.

SEX DETERMINATION

- ✓ **Assessing the gender of unknown human skeletal remains is the second step in the triad of building a dental profile.**

- ✓ **Sex can be asessed based on data from morphology of skull & mandible, tooth measurement & by analysis of DNA from teeth.**

GENDERING FROM CRANIOFACIAL MORPHOLOGY & DIMENSIONS

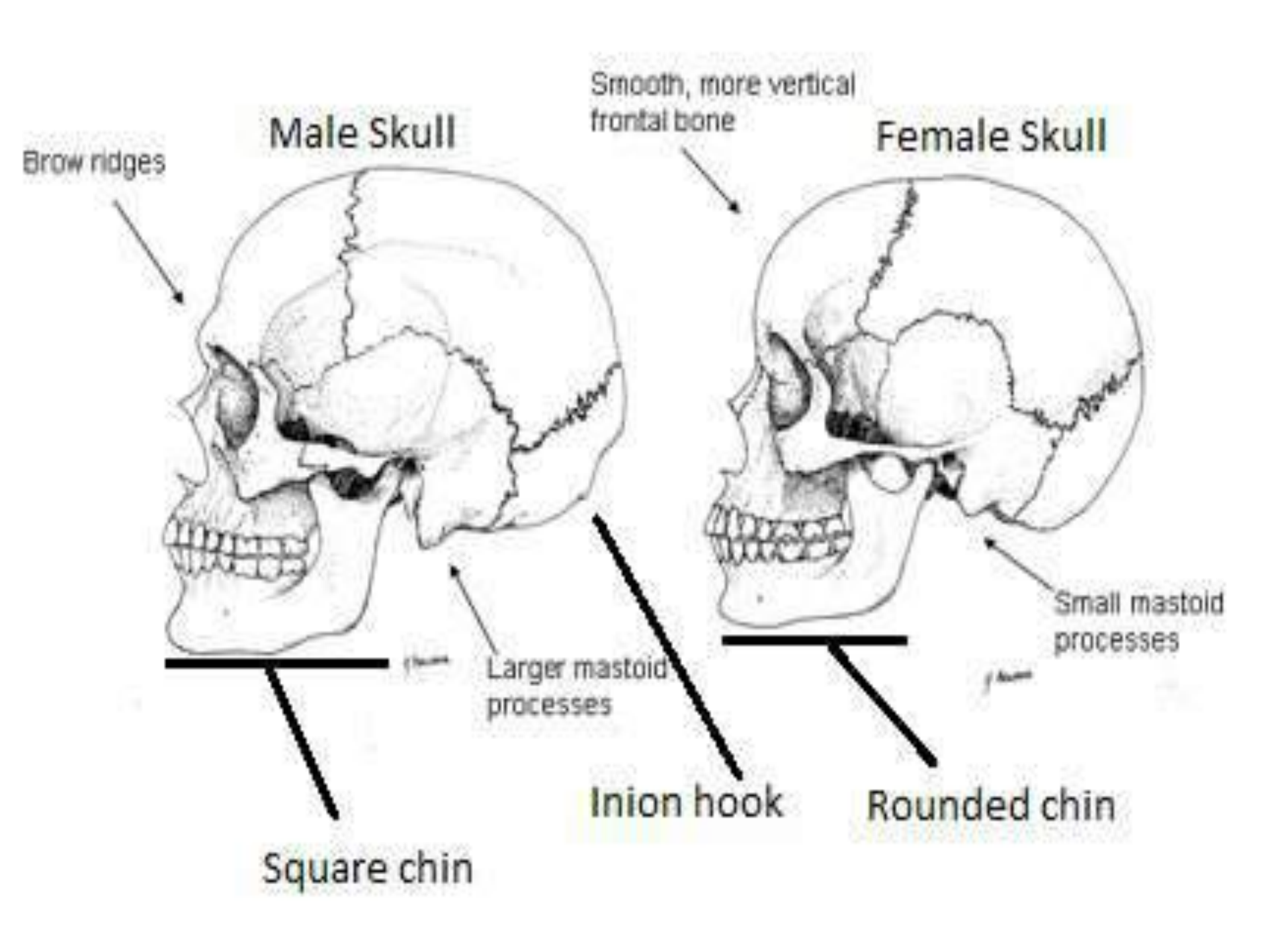
- ✓ **The use of morphological features of the skull & mandible is a common approach used by anthropologist in gendering.**

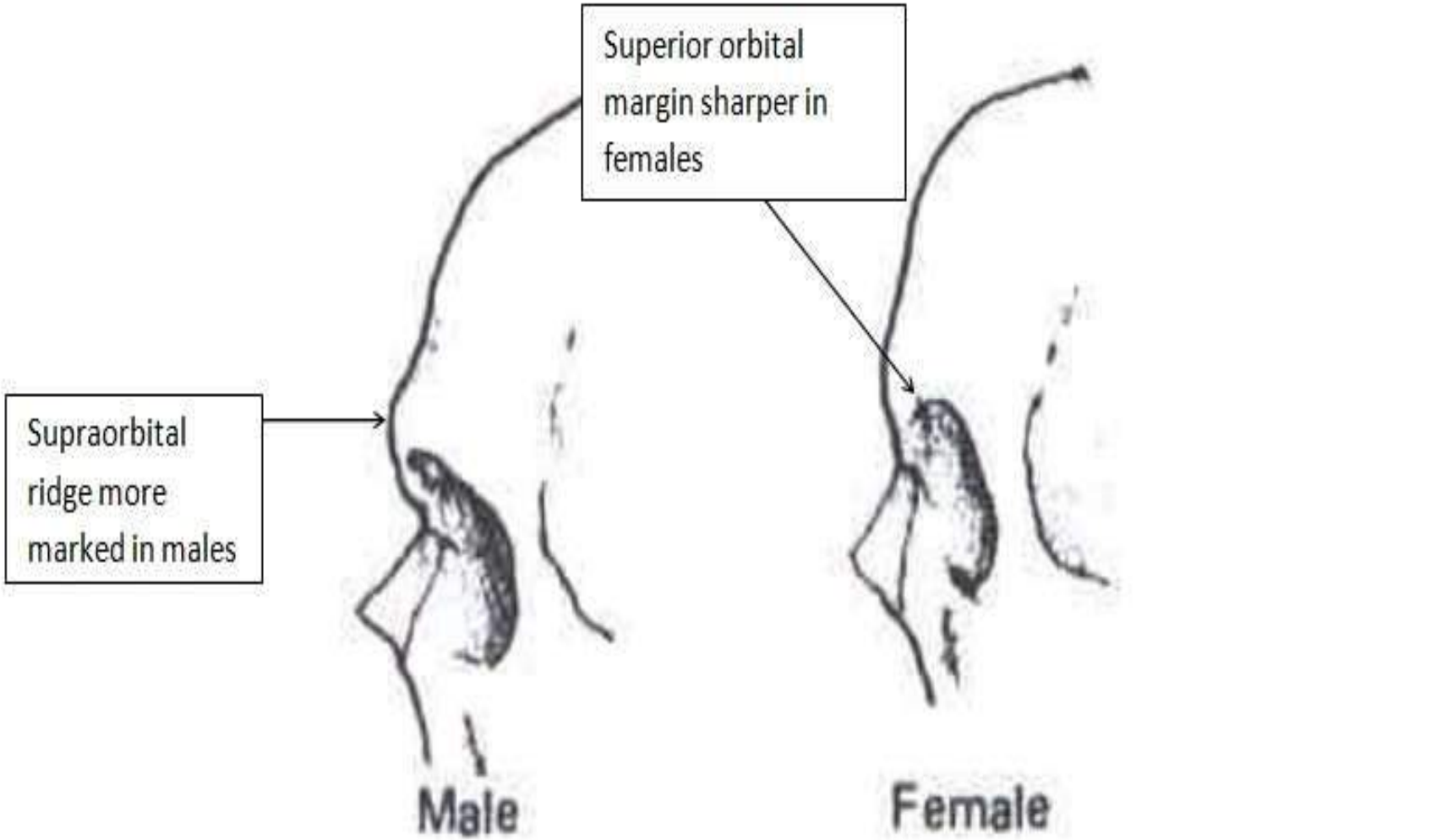
- ✓ **A number of features are known to show variation between the genders.**

CRANIOFACIAL MORPHOLOGIC INDICATORS OF GENDER

SKULL FEATURES	MALE	FEMALE
Size/architecture	Big , rugged	Small, smooth
Frontal & parietal eminence	Small	Large
Forehead	Sloping	Vertical
Supraorbital ridges	Medium - large	Small- medium
Glabella	Moderate-marked curve	Flat-slight curve
Orbits	Squared, low, rounded margins	Rounded, high, sharp margins

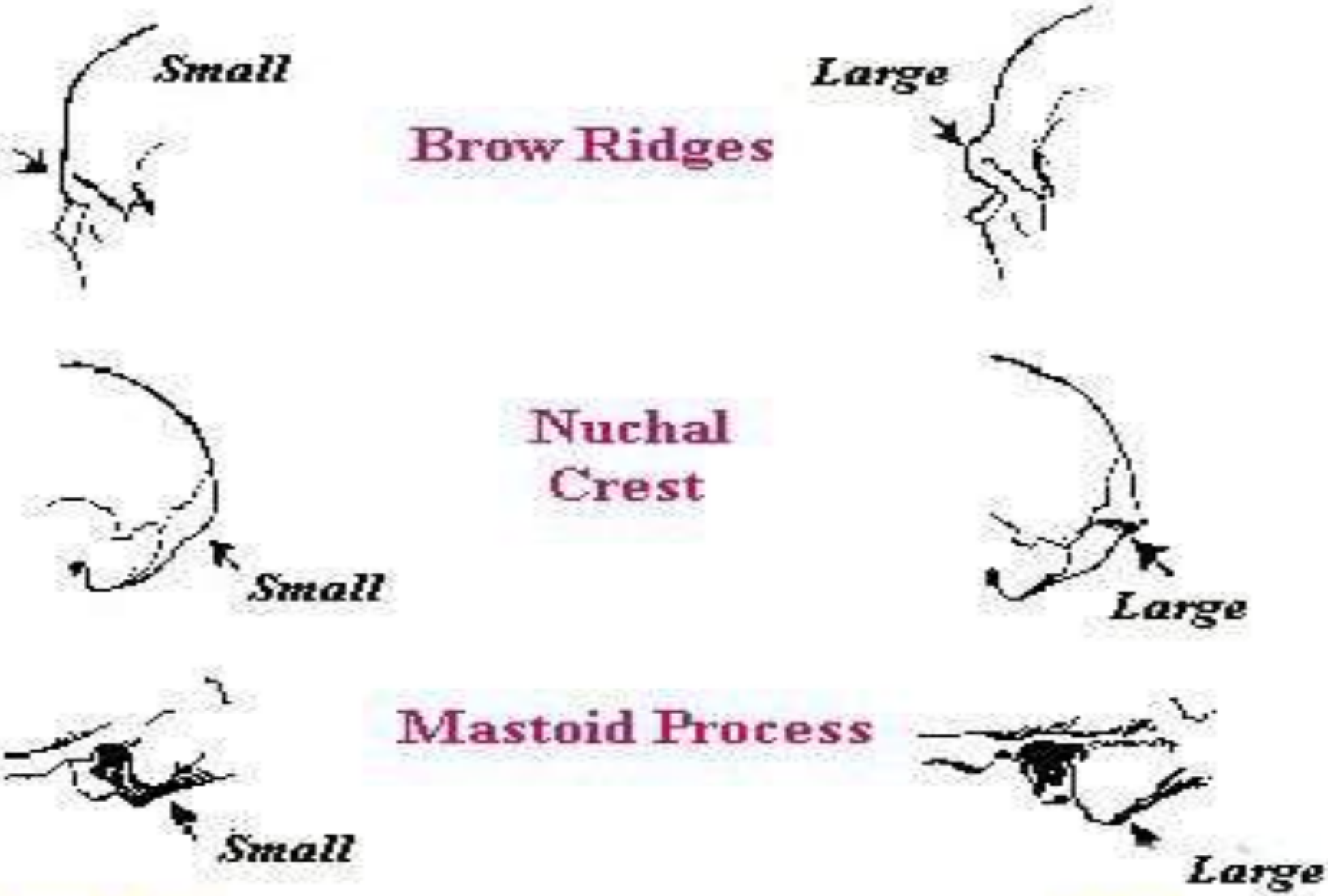
NASAL APERTURE	High, thin sharp margins	Lower, wider rounded margins
ZYGOMATIC ARCH	Extends	Does not extend
OCCIPITAL	Muscle lines marked	Muscle lines not marked
MASTOID PROCESS	Medium-large	Small-medium
OCCIPITAL CONDYLE	Large	Small
GLENOID FOSSA	Deep	Shallow
FORAMEN MAGNUM	Large-long	Small-round
PALATE	Large, U- shaped	Smaller, parabolic


Male Skull
Brow ridges
Larger mastoid processes
Square chin
Inion hook
Smooth, more vertical frontal bone
Female Skull
Small mastoid processes
Rounded chin



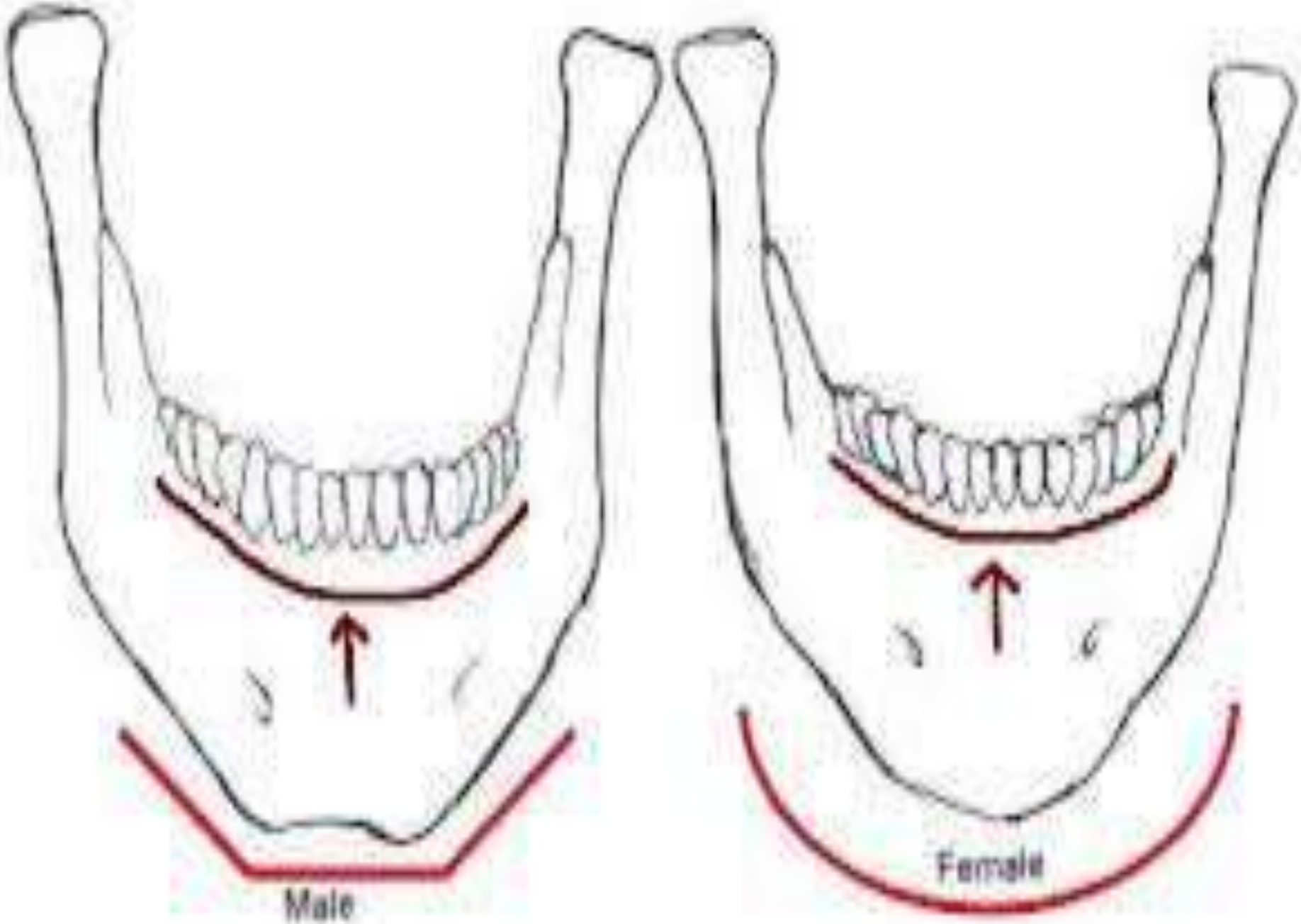
Picture adapted: Bass, William M. 1995. 4th ed. Human Osteology:A Laboratory and Field Manual of the Human Skeleton. Specials Publication No. 2. Missouri

Major Sexing Features of the Skull



MANDIBLE FEATURES

	MALE	FEMALE
General features	Large, broad ascending ramus	Small, narrow ascending ramus
Condyles	Large	Small
Shape of chin	Square	Rounded, pointed
Gonial angle	Less obtuse, flares	More obtuse, does not flare
Body height	High symphysis	Low symphysis


Male
Female

- ✓ **WILLIAMS & ROGERS found that gender could be predicted correctly in 96% of cases using different features of the skull & mandible.**

- ✓ **They observed that constellation of just 6 features**

i. **Mastoid process**

ii. **Supraorbital ridge**

iii. **Size & Architecture of skull**

iv. **Extention of Zygomatic arch beyond the external auditory canal.**

v. **Nasal aperture**

vi. **Gonial angle**

SEX DIFFERENCE IN TOOTH SIZE

- ✓ Tooth is used for differentiating gender by measuring their **mesiodistal & buccolingual dimensions.**

- ✓ **Numerous study have shown that the** male dentition is statistically larger than the female permanent & deciduous tooth crown dimensions.

Using Anatomy Of Tooth

Male Teeth

Female Teeth

- Male teeth larger both mesio-distally& labio-lingually.
- Size difference is more in maxillary centrals & laterals.
- Incisal edges are rounded off in females.
- Tooth eruption & development processes take place earlier in females.

Average male & female tooth measurements (in mm) on the right & left side combined

TOOTH	MALE	FEMALE	MALE	FEMALE
MAXILLA	BUCCOLINGUAL		MESIODISTAL	
CI	7.24	7.09	8.42	8.29
LI	6.35	6.27	6.61	6.50
CI	8.08	7.76	7.63	7.41
PM1	9.26	9.11	6.81	6.82
PM2	9.23	9.05	6.42	6.55
M1	11.16	10.83	9.85	9.80
M2	10.86	10.52	9.34	9.18

MANDIBLE

	BUCCOLINGUAL		MESIODISTAL	
TOOTH	MALE	FEMALE	MALE	FEMALE
CI	5.85	5.86	5.35	5.26
LI	6.08	6.12	5.88	5.78
CI	7.36	7.00	6.66	6.46
PM1	7.93	7.74	6.77	6.82
PM2	8.54	8.26	6.85	6.88
M1	10.50	10.17	10.80	10.58
M2	10.07	9.85	9.83	9.71

GENDER DETERMINATION BY DNA ANALYSIS

- ✓ **Forensic DNA analysis for gender determination can give highly accurate result.**

- ✓ **Sex can be determined by very minute quantities of DNA & from very old specimens of teeth.**

- ✓ **"AMELOGENIN (AMEL) is one of the major matrix proteins secreted by the ameloblasts of the enamel.**

- ✓ **The AMEL gene, coding for highly conserved protein, is located on the X- & the Y-chromosomes in humans.**

- ✓ **The 2 alleles are similar for the exonic sequences but differ in the intronic sequences.**

- ✓ Thus the **females (XX)** have **two identical AMEL genes** but the **males (XY)** have **two non identical genes”.**

- ✓ **Preparing DNA from teeth by ultrasonication & subsequent PCR amplification, there is 100% success in determining sex of the individual**.

DENTAL AGE ESTIMATION

- Ageing In Prenatal, Neonatal And Early Postnatal.
- Age Estimation In Children And Adolescents.
- Age Estimation In Adults.

Phases in tooth development and eruption

1. **Utero:** (from 6 - 8 weeks) to eruption of 1st tooth at 6 months

2. **Primary dentition (milk teeth):** from 6 months to 6 years.

3. **Mixed dentition:** 6 to 12 years.

4. **Permanent dentition:** From 12 years on.

Age Estimation

- Presence of **neonatal line** indicates a live birth.
- **Examining teeth eruption patterns.**
- **Gustafson method (looking for six signs of wear)**
- **Lamendin method (looking at transparency of roots/dentin translucency)**
- **Age estimation based on ratio of D-Aspartic acid to L-Aspartic acid.**

Malignant Connective Tissue Tumours

Willis Definition:

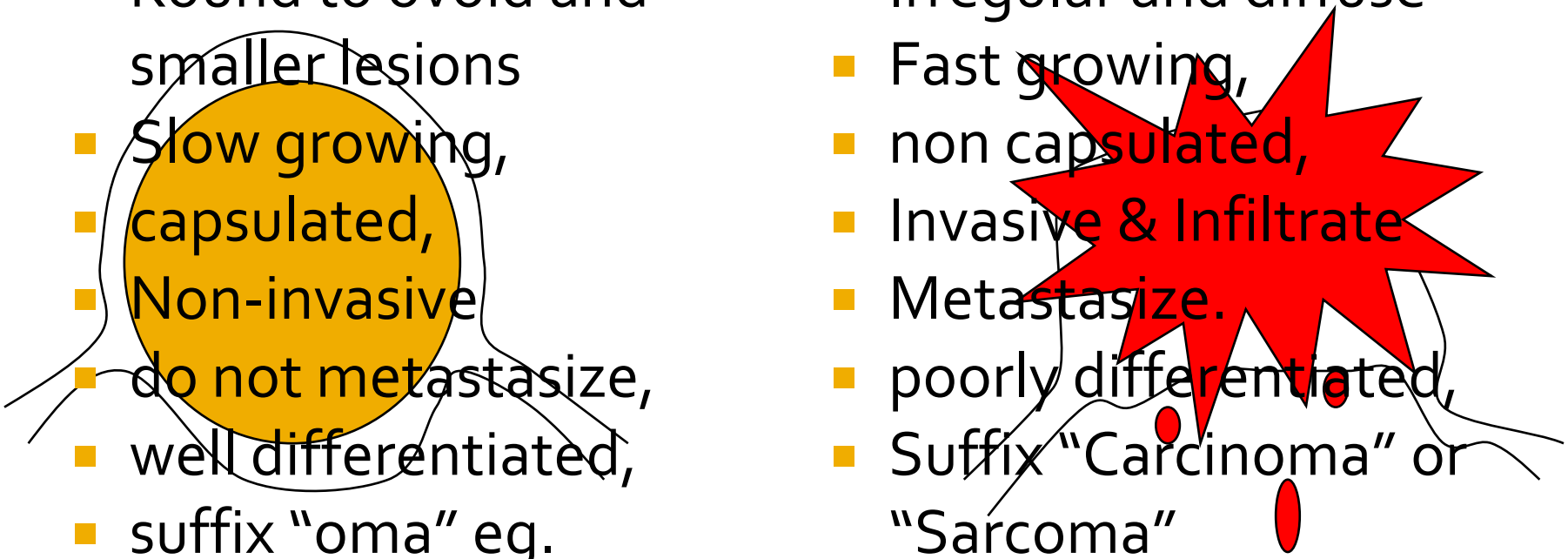
Neoplasm is an abnormal mass of tissue the growth of which exceeds and is uncoordinated with that of normal tissue and persists in the same excessive manner after cessation of the stimuli which evoked the change

Benign

- Round to ovoid and smaller lesions
- Slow growing,
- capsulated,
- Non-invasive
- do not metastasize,
- well differentiated,
- suffix “oma” eg. Fibroma.

Malignant:

- Irregular and diffuse
- Fast growing,
- non capsulated,
- Invasive & Infiltrate
- Metastasize.
- poorly differentiated,
- Suffix “Carcinoma” or “Sarcoma”

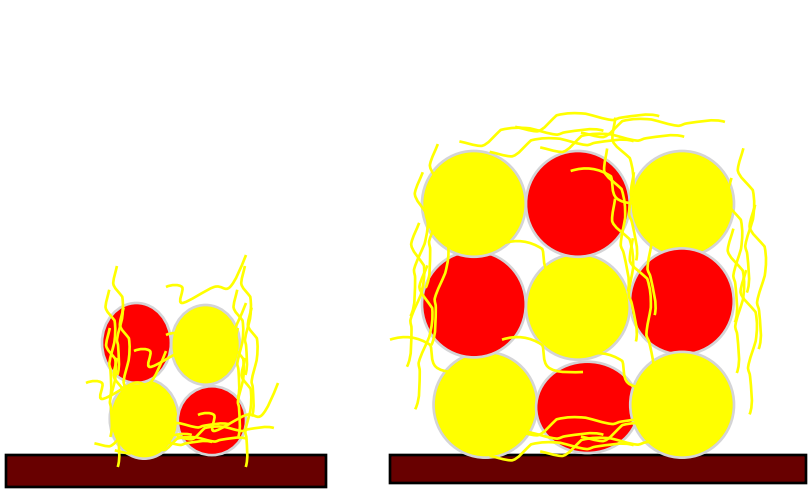


Carcinoma v/s Sarcoma

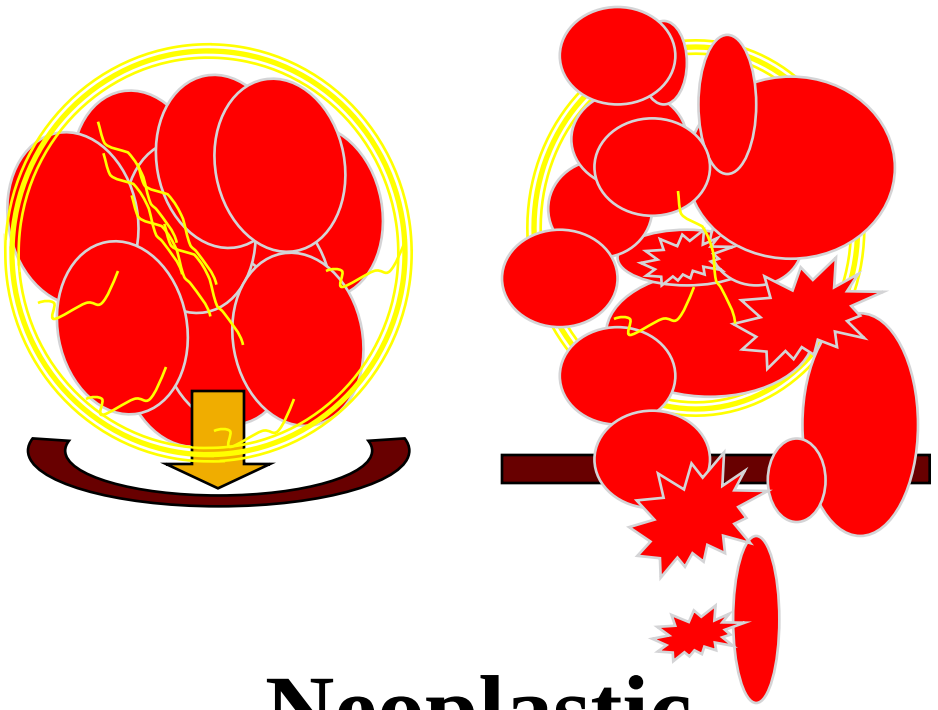
Features	Carcinoma	Sarcoma
Age Incidence	Middle age & old age (can occur at any age)	Young adults (can occur at any age)
General incidence	More common	Less common
Structure	Typically arranged in groups except in anaplastic tumours	Arranged in diffused sheets intimately related to stroma
	Stroma often well formed	Stroma always poorly formed and inadequete for the growth of the tumour
	Haemorrhage and necrosis Less common	Haemorrhage and necrosis more common

Features	Carcinoma	Sarcoma
Growth pattern	Relatively slow growing	Very rapid
Metastasis	Early : lymphatic Late stage: haematogenous (very rare) Commonest site : liver	Lymphatic spread very rare haematogenous spread common Commonest site: lungs
Radiosensitivity	Highly radiosensitive	Highly radioluresistant

Mechanism of Neoplams



Non-Neoplastic
(Polyclonal)



Neoplastic
(Monoclonal)

SARCOMAS

Latin -- FLESHY

Common Characteristics

- Relatively younger individuals
- Faster metastasis
- Hematogenous spread
- Lesser five year survival rate
- Difficult to diagnose because of overlapping clinical and histopathologic features
- IHC confirmatory diagnosis

Tissue of origin	Benign	Malignant
Fibroblasts	Fibroma	Fibrosarcoma
Muscle	Leomyoma Rabdomyoma	Leomyosarcoma Rabdomyosarcoma
Fat	Lipoma	Liposarcoma
Nerve	Neuroma Schwonnoma Neurofibroma	Neurofibrosarcoma Malignant peripheral nerve sheath tumour(MPNST)
Endothelial cells	Hemangiomas Lymphangiomas	Hemangio endothelioma Hemangiopericytoma Angiosarcoma Kaposi's sarcoma
Cartilage	Chondroma	Chondrosarcoma
Bone	Osteoma	Osteosarcoma
Lympho-reticular cells	----------	Lymphomas Multiple myeloma Ewing's sarcoma

Fibrosarcoma

Tumour of mesenchymal cell origin that is composed of malignant fibroblasts in collagenous background

Soft tissue tumour or primary or secondary bone tumour

Uncommon neoplasm

PRIMARY	SECONDARY
▪ Primary lesion ▪ Fibroblastic malignancy that produces variable amount of collagen ▪ Central or peripheral	▪ Arises from a preexisting lesion or after radiotherapy to an area of bone or soft tissue ▪ Central or peripheral ▪ Poor prognosis

Etiopathogenesis

No definite causes

Translocation t(12;15)(p13;q25)

Multiple neurofibromas – 10 % risk

Pre existing lesions like Fibrous dysplasias, Chronic Osteomyelitis, Bone infracts, Paget's disease

Clinical features

- 10% of musculo-skeletal and 5% of bone
- No racial predilection
- M>F
- 4^{th} decade
- Lower extremities– femur & tibia , soft tissues of the thigh, post knee
- Infantile fibrosarcoma -< 10 yrs – good prognosis

Clinical features

- Pain and swelling
- Pathologic fracture

} Central

- Painless mass
- Spreads faster and grows to huge size

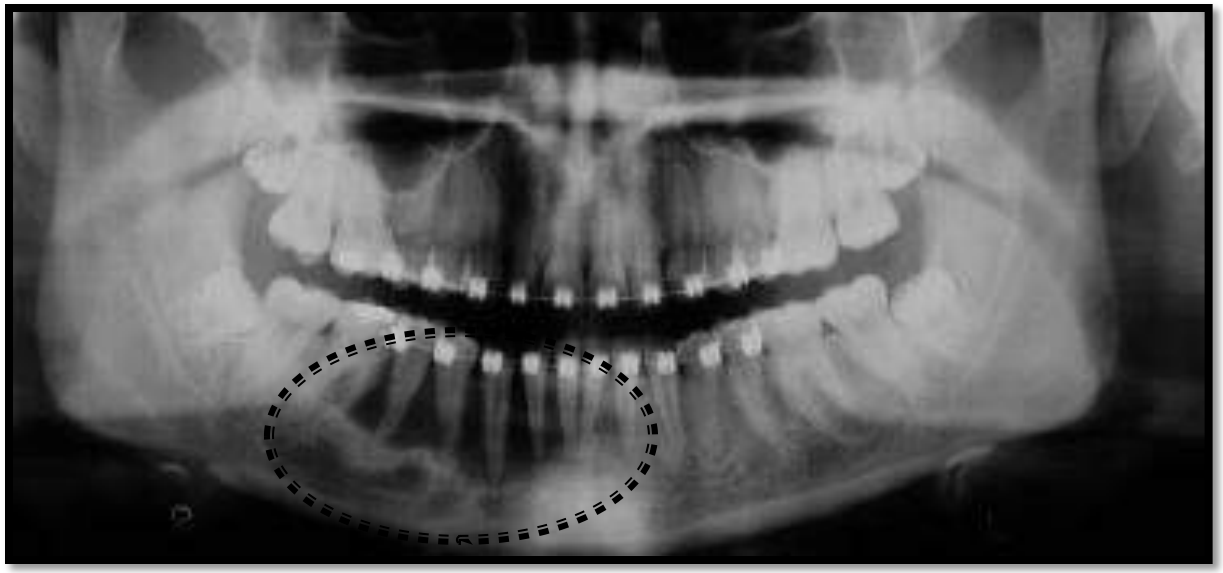
} Soft tissue

Differential diagnosis

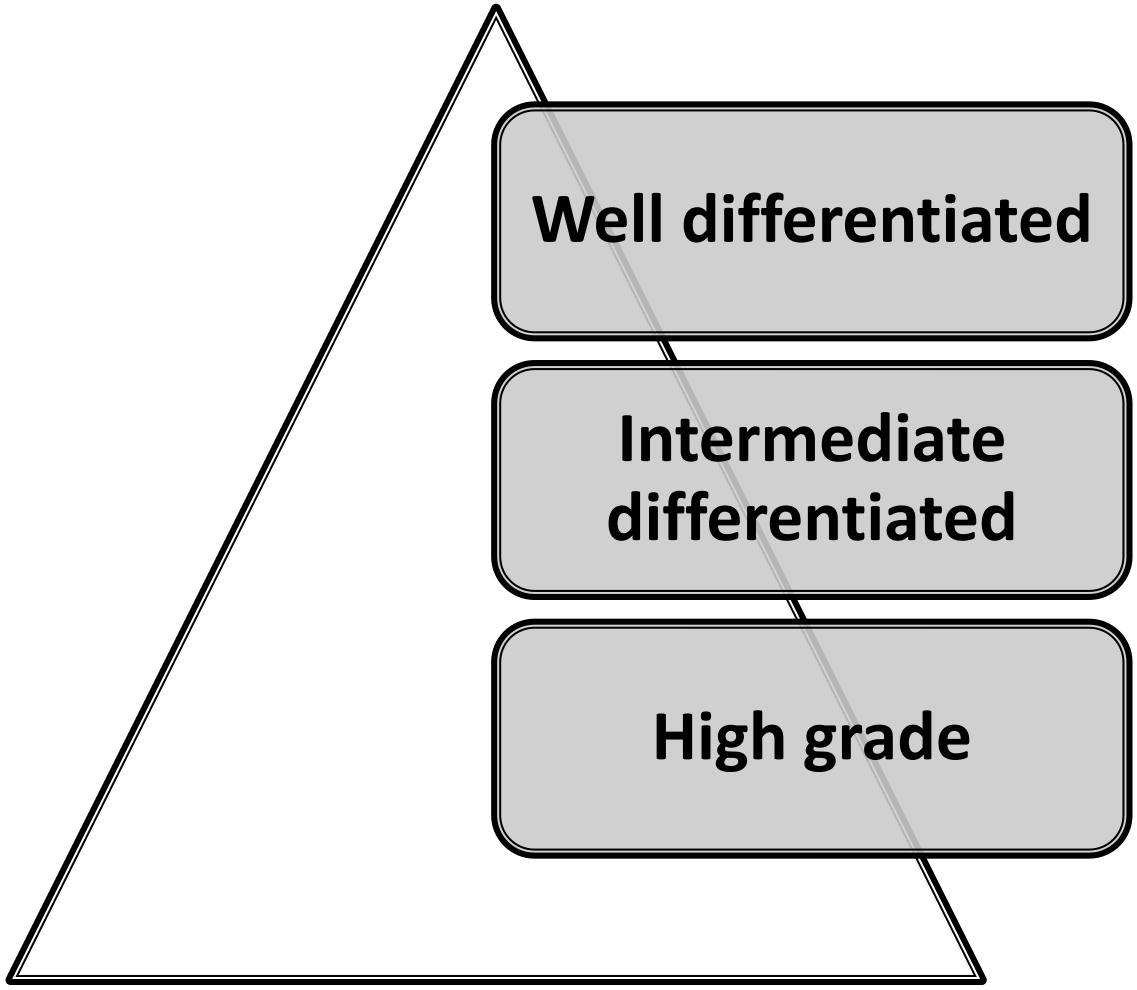
- Fibrous dysplasia
- Fibrous histiocytoma
- Osteosarcoma
- Paget's disease
- Malignant neuro sarcoma

Radiographic features

- Irregular radiolucency with some radiopaque mass



Histopathology



Well

- Malignant cells are dispersed in rich collagen background
- Plump fibroblasts with deeply staining spindle shaped nuclei & tapered ends
- Less pleomorphism of tumour cells
- Normal mitotic figures

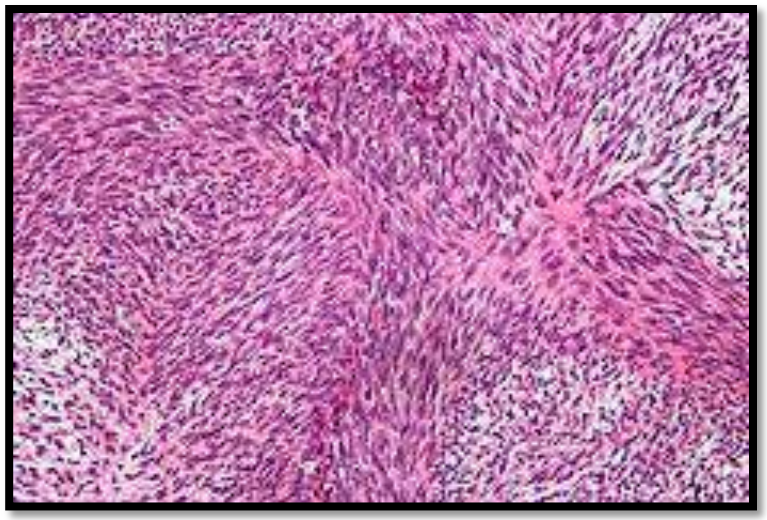
Intermediate

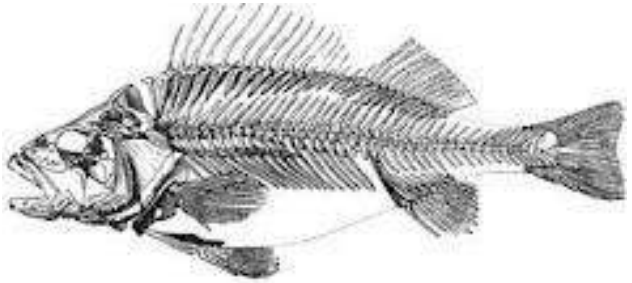
More cellular

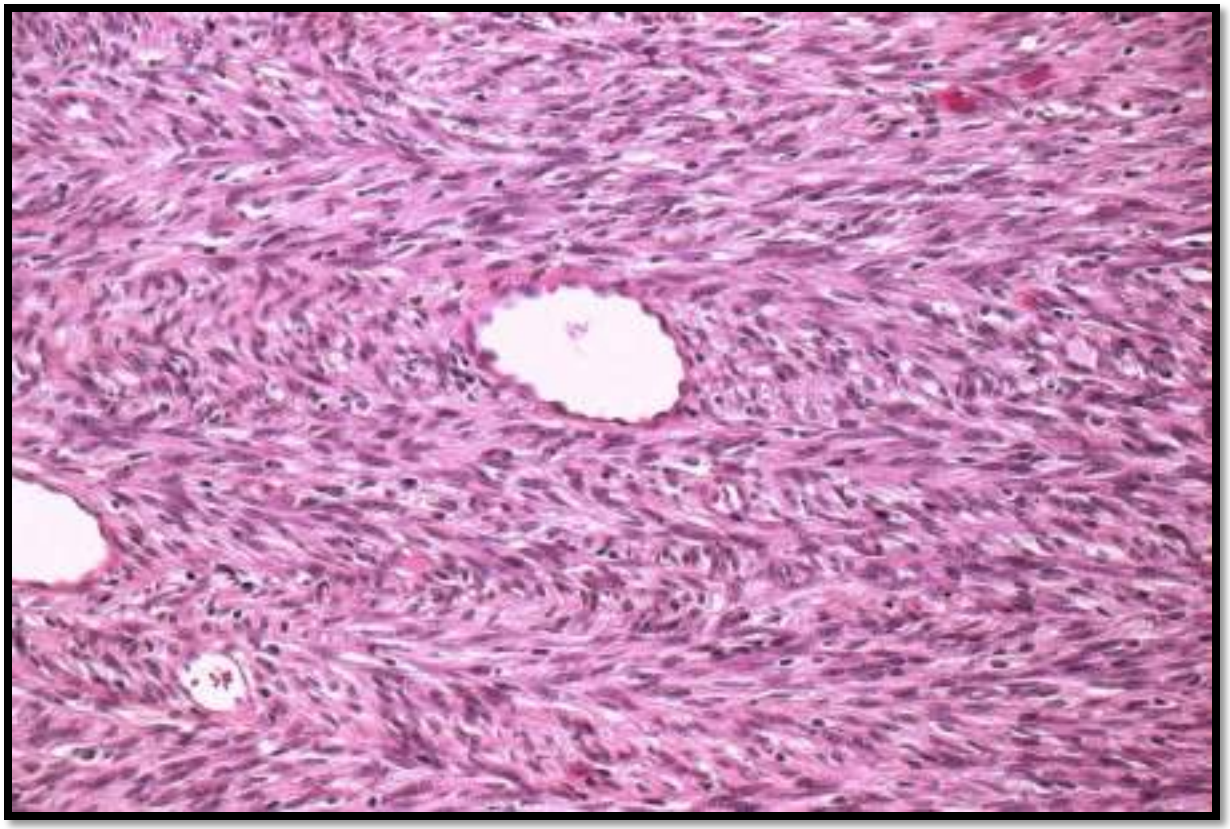
Parallel sheets of cells arranged in intertwining whorls – **HERRINGBONE PATTERN**

Slight degree of pleomorphism

Areas of hyalinization with collagen maturation

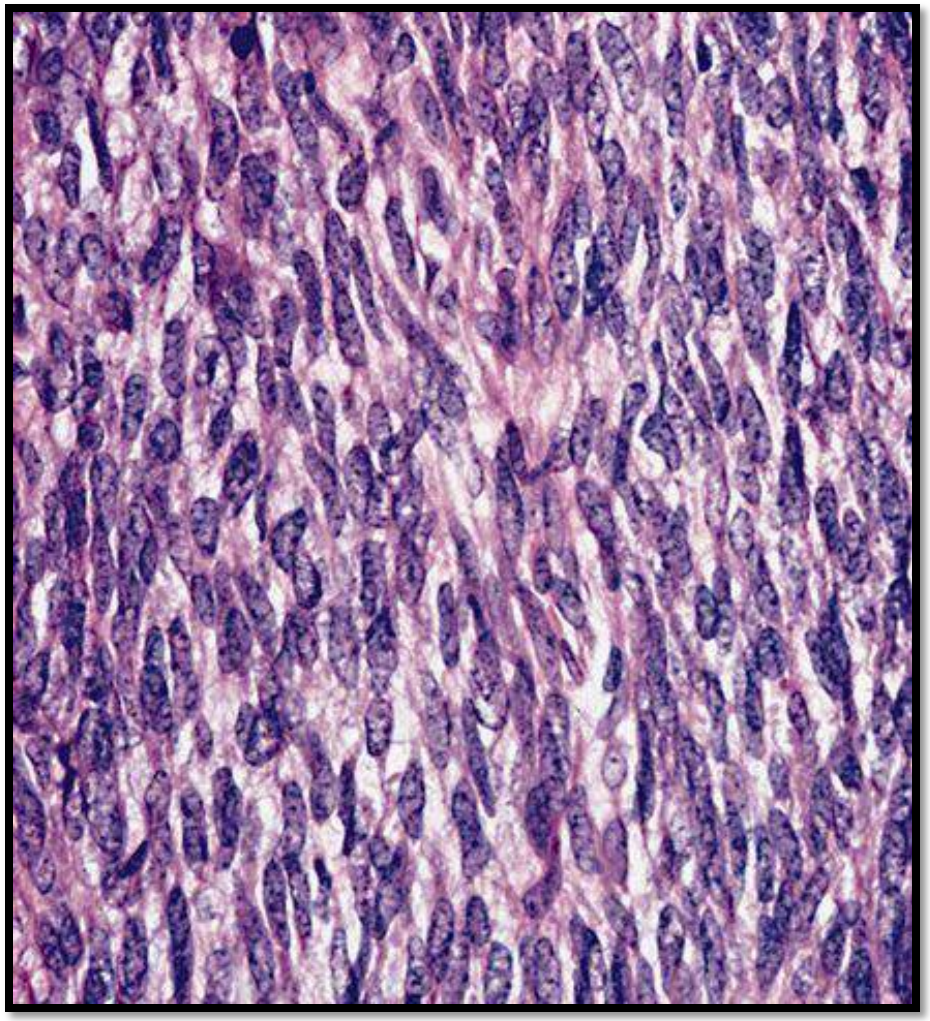


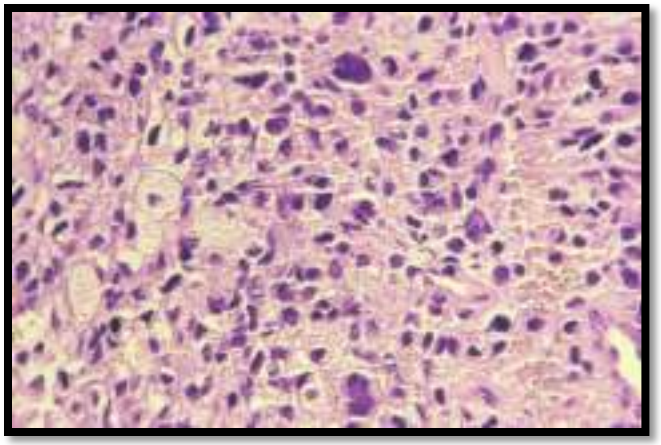


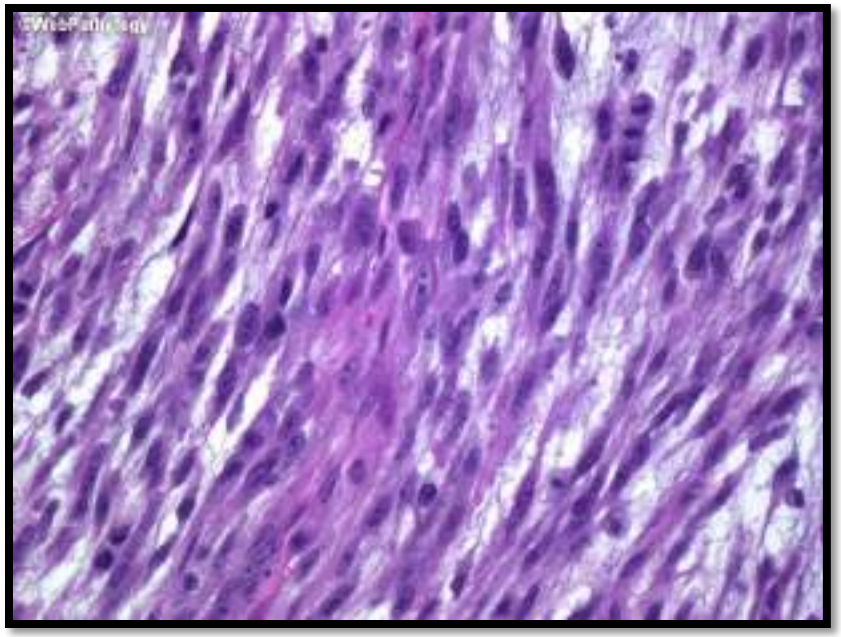


High

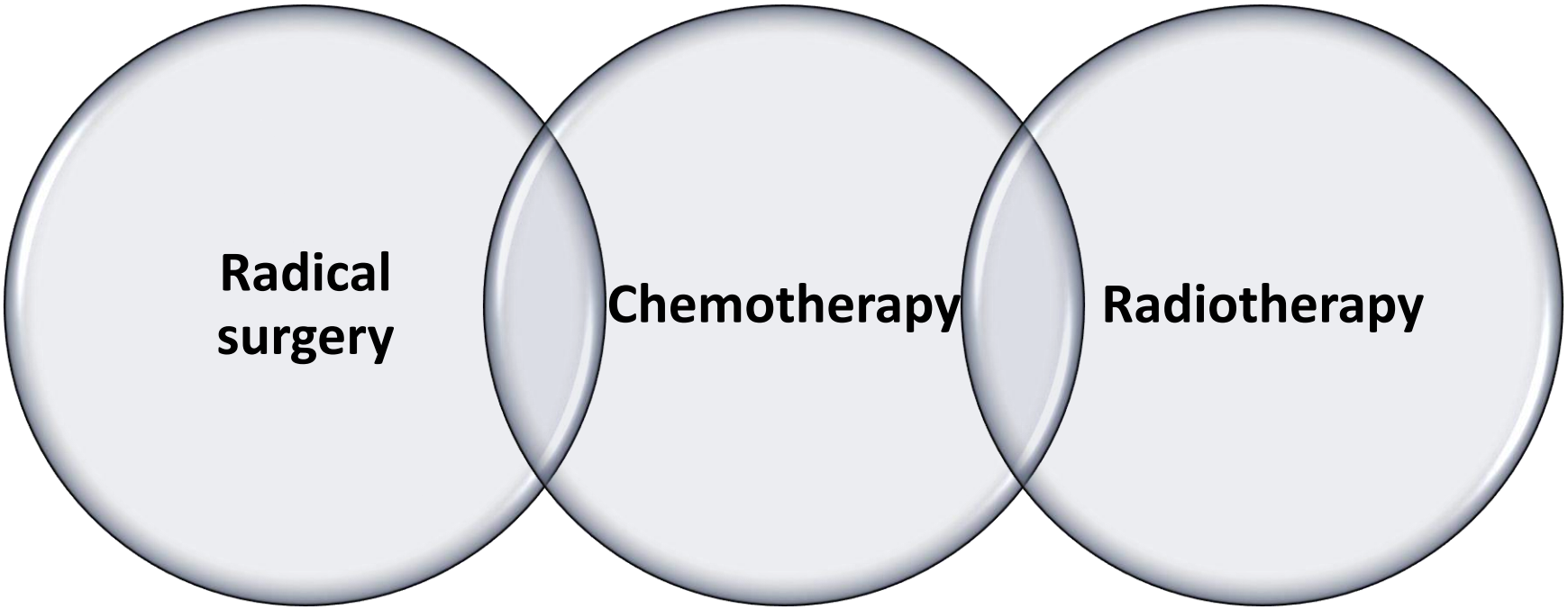
- Highly cellular
- Marked cellular atypia
- Sparse matrix
- Anaplastic cells with bizarre mitosis
- Multinucleated giant cells







Treatment



Kaposi's sarcoma

- Multiple idiopathic hemorrhagic sarcoma of kaposi
- Angioreticuloendothelioma
- **Multi-centric proliferation of vascular and spindle cell components**
- 1872 – Moritz Kaposi

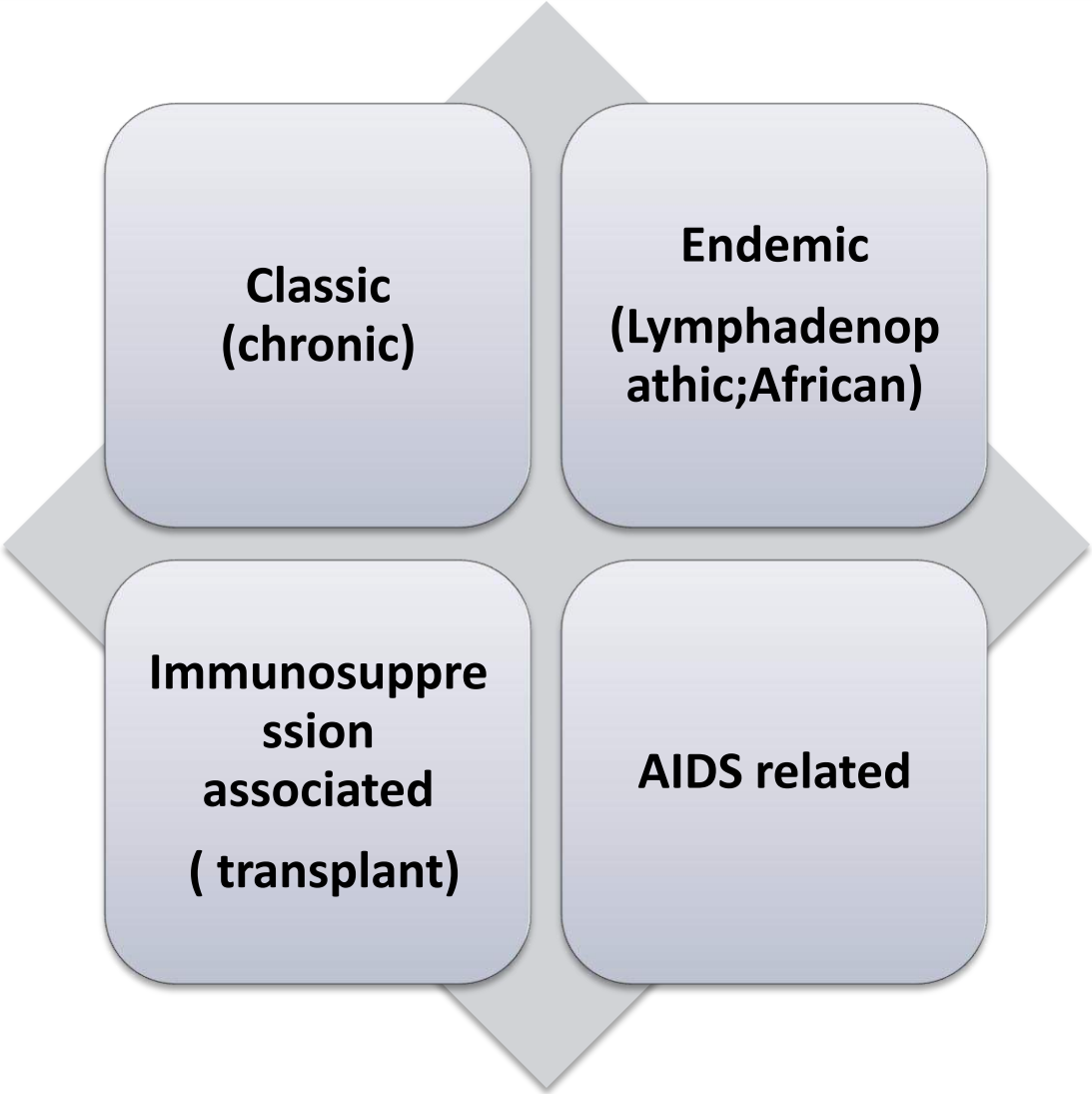
Etiology

Unknown

- Immunosuppression
- Sexually transmissible infectious agents
- Human herpes virus 8(HHV8 or KSHV)

An angiogenic disorder with widespread cellular proliferation occurring in response to circulating growth factors

Clinical Features



Classic

- Associated with altered immune status and some lympho-reticular malignancies
- Cutaneous multifocal blue - red nodules on the lower extremities
- Slowly growing, increase in number
- Oral involvement – rare
- Soft bluish nodules on palatal mucosa & gingiva

Endemic

- Young African children
- localized or generalized enlargement of lymph nodes
- Fulminant course with visceral involvement
- Skin and oral cavity – rare
- Head & neck – salivary glands & cervical nodes

Transplant

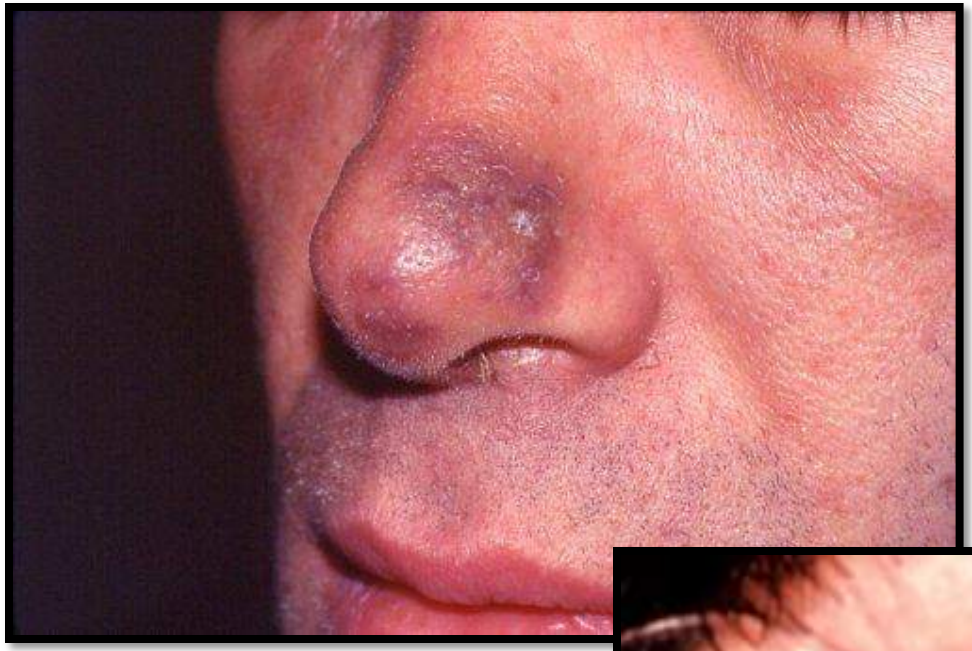
1-4 % of renal transplant

Oral cavity – rare

Sarcomatous involvement of skin &internal organs

AIDS related

- 40% of homosexual AIDS patients
- Cutaneous lesions are common – tip of the nose
- Oral lesions – common, palate and gingiva
- Early lesions -- Flat blue/red/purple patch , focal or diffuse, asymptomatic (patch stage)
- Deeply discolored & surface papules/nodules with exophytic growth (plaque/ nodular stage)
- Cervical lymphedenopathy/salivary gland enlargement



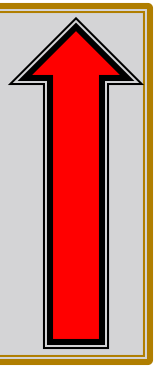
Histopathology

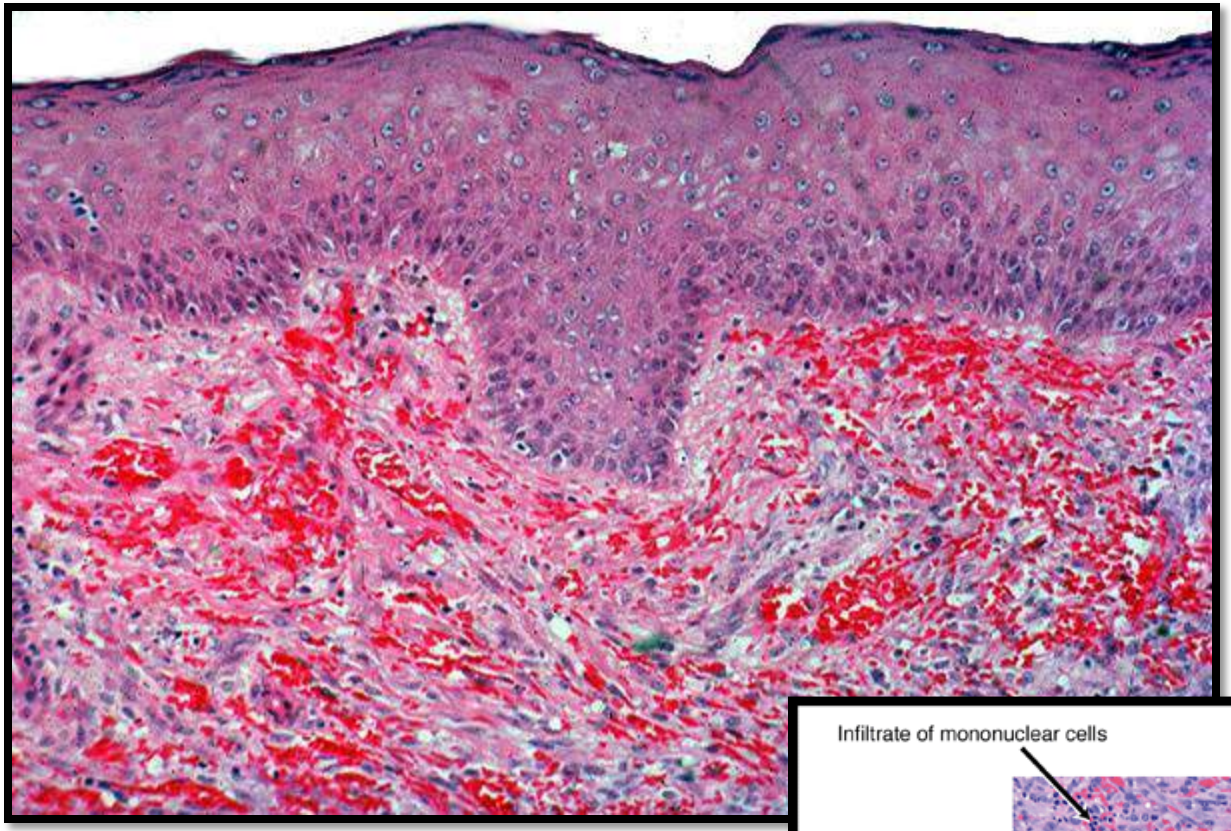
Patch stage

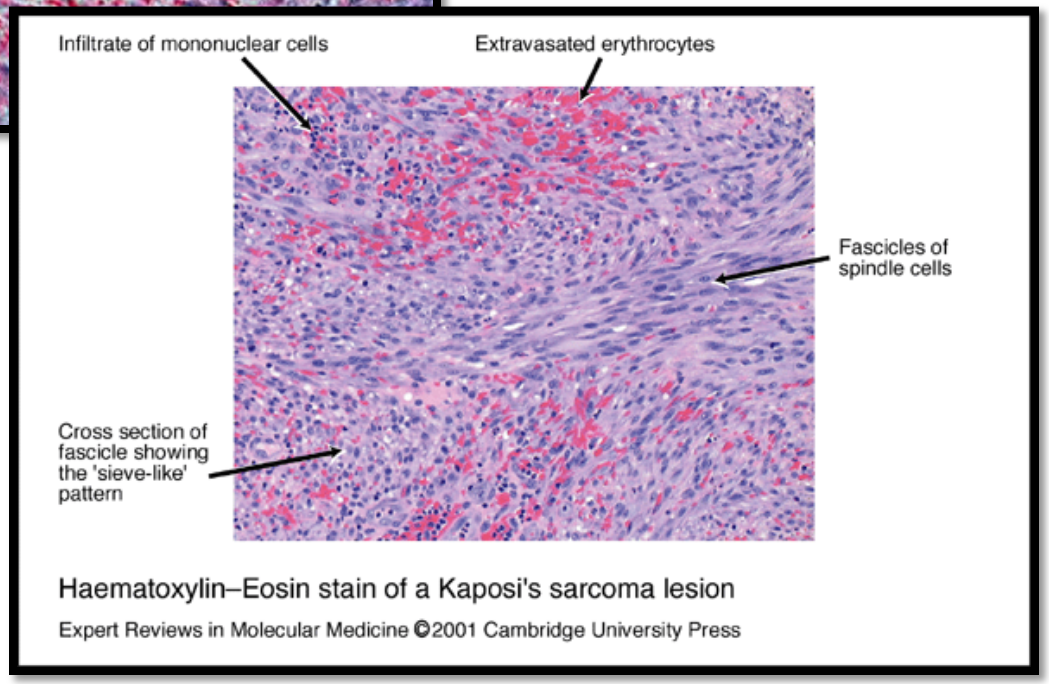
- ✓Proliferation of small veins and capillaries around dilated vessels
- ✓Slit like vessels around preexisting vessels
- ✓Vessels lined by plump ,atypical endothelial cells
- ✓Pronounced mononuclear infiltrate

Plaque stage

- ✓Proliferation of small veins and capillaries around dilated vessels
- ✓Slit like vessels around preexisting vessels
- ✓Vessels lined by plump ,atypical endothelial cells
- ✓Pronounced mononuclear infiltrate

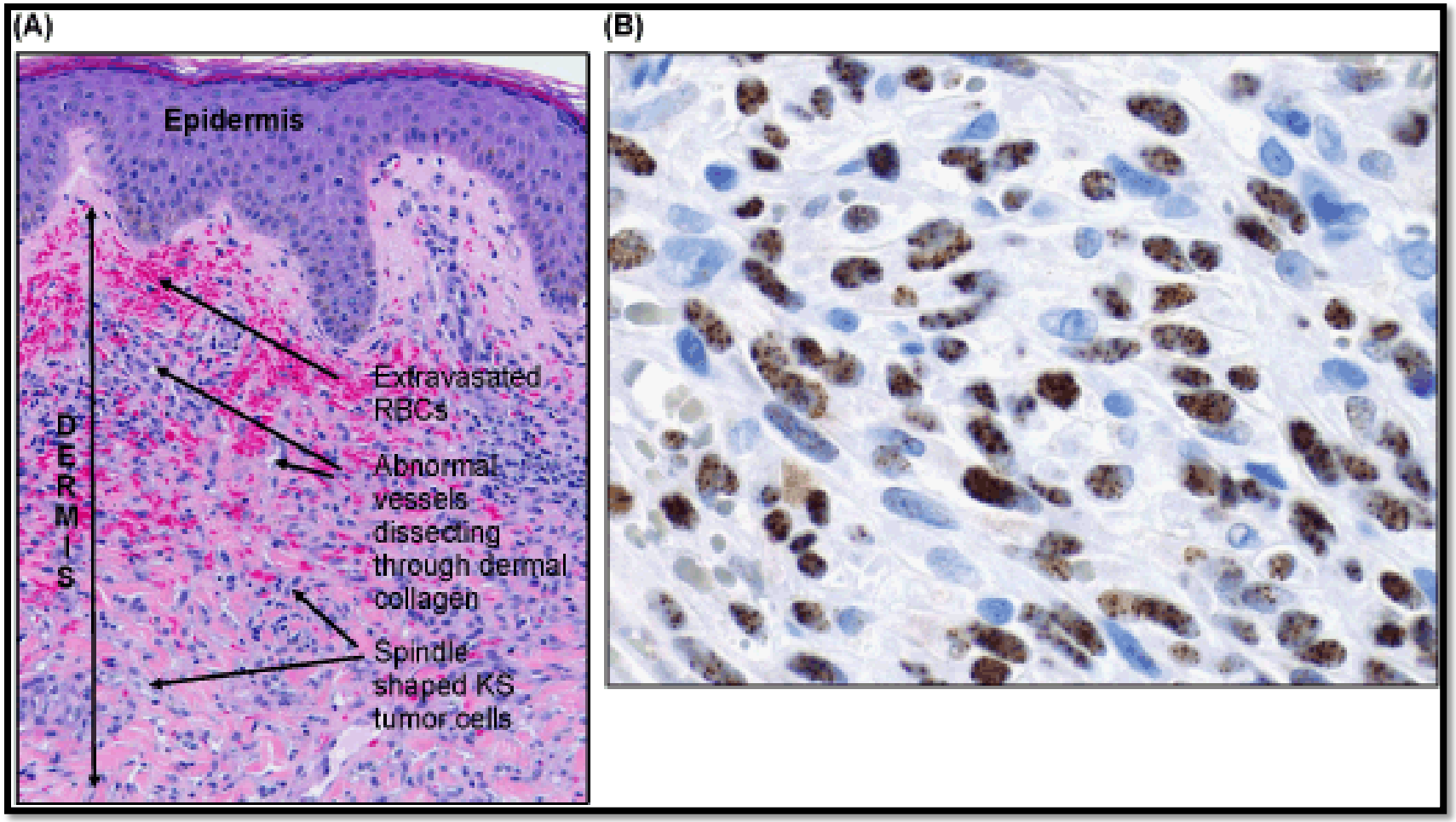






Haematoxylin–Eosin stain of a Kaposi's sarcoma lesion

Expert Reviews in Molecular Medicine ©2001 Cambridge University Press


(A)
Epidermis
DERMIS
Extravasated RBCs
Abnormal vessels dissecting through dermal collagen
Spindle shaped KS tumor cells
(B)

Treatment

Low dose radiation with chemotherapy

Ewing's sarcoma

- Endothelial myeloma
- Round cell tumour

- **James Ewing in 1921**

- Highly malignant small round to ovoid cell sarcomas with clinical and radiographic characteristics of primary osseous lesion

Etiology

Unknown

GENE TRNASLOCATION

Clinical features

- Children and young adults (5-25 yrs)
- Central & Peripheral
- Long bone & pelvic region
- Mand > Max – mandible ramus
- M>F
- Pain and swelling
- Facial deformity
- Destruction of alveolar bone with loosening of the teeth

Radiography

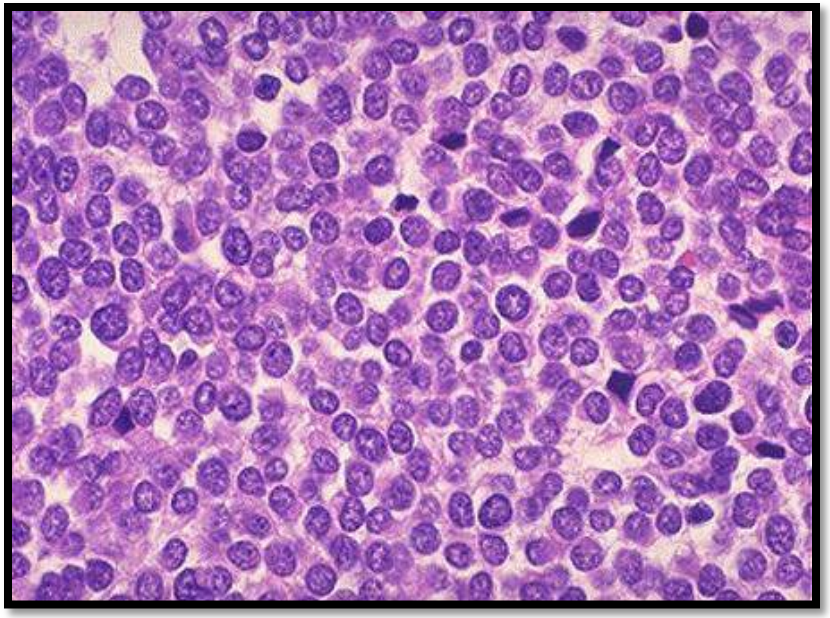
Moth eaten radiolucency with erosion of the cortex

Expansion of bone

Periosteal **“ onion –skin”** appearance may be seen

Histopathology

- Extremely cellular
- Solid sheets or masses of small round cells
- Dispersed chromatin & hyper chromatic nuclei, high mitotic figures
- **FILIGREE** pattern
- Positive for glycogen



Tretment

Surgery +Radiation +chemotherapy

Malignant lymphomas

Malignant lymphomas constitutes a group of neoplasms of varying degrees of malignancy which are derived from the basic cells of lymphoid tissue, the lymphocytes & histiocytes in any of their developmental stages

TABLE 9-1 Revised European-American Lymphoma (REAL) Classification of Lymphoid Neoplasms

	B-Cell Neoplasms	T-Cell and Postulated NK-Cell Neoplasms
Precursor cell neoplasms	Precursor B-lymphoblastic lymphoma/leukemia	Precursor T-lymphoblastic lymphoma/leukemia
Peripheral (mature) cell neoplasms	B-cell chronic lymphocytic leukemia/small lymphocytic lymphoma (B-CLL/SLL) Lymphoplasmacytoid lymphoma Mantle cell lymphoma Marginal zone B-cell lymphoma (extranodal or nodal) Splenic marginal zone B-cell lymphoma Hairy cell leukemia Plasmacytoma Diffuse large B-cell lymphoma Burkitt's lymphoma	T-cell chronic lymphocytic leukemia Large granular lymphocytic leukemia (T-cell or NK-cell type) Mycosis fungoides Peripheral T-cell lymphoma, unspecified Angioimmunoblastic lymphoma Intestinal T-cell lymphoma Adult T-cell lymphoma/leukemia Anaplastic large cell lymphoma

NK, Natural killer; *B-CLL/SLL*, B-cell chronic lymphocytic leukemia/small lymphocytic lymphoma.

Hodgkin’s lymphoma

Non Hodgkin’ s lymphoma

Hodgkin's lymphoma

I. **Nodular lymphocyte predominant lymphoma**

II. **Classical Hodgkin's lymphoma**

A. *Nodular sclerosis*
B. *Lymphocyte rich*
C. *Mixed cellularity*
D. *Lymphocyte depleted*

Hodgkin's lymphoma

Hodgkin's disease

Thomas Hodgkin -- 1832

Primarily disease of lymph nodes

Etiology

EB virus

HIV related

Clinical features

- Bi-model
- 30-35yrs ---- 50-55 yrs
- Whites >blacks
- Painless enlargement of the lymph-nodes
- URT infections, fever, weight loss, night sweats, splenomegaly, hepatomegaly

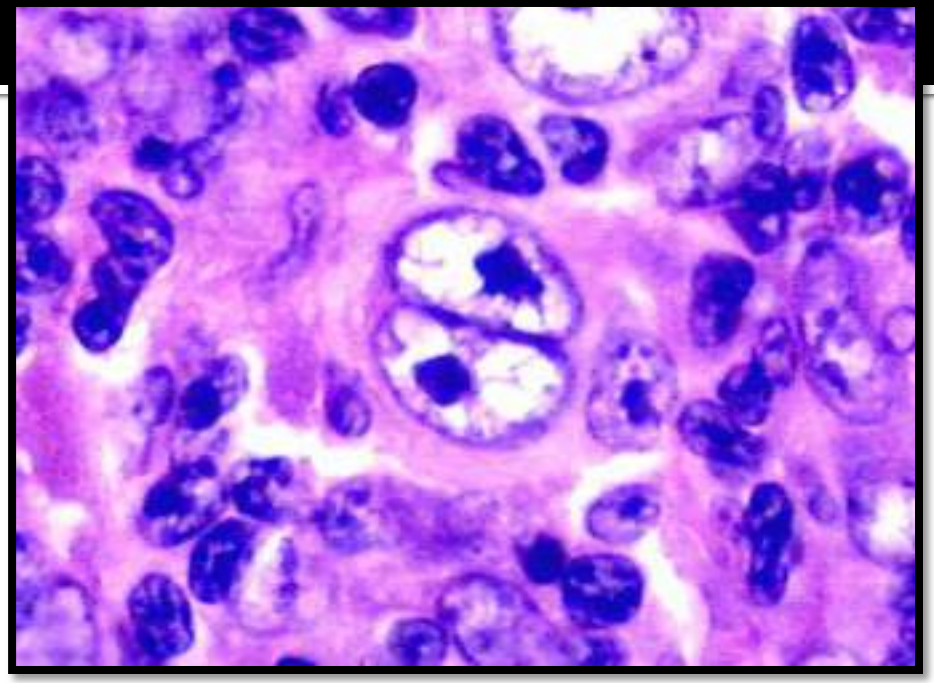
Histopathology

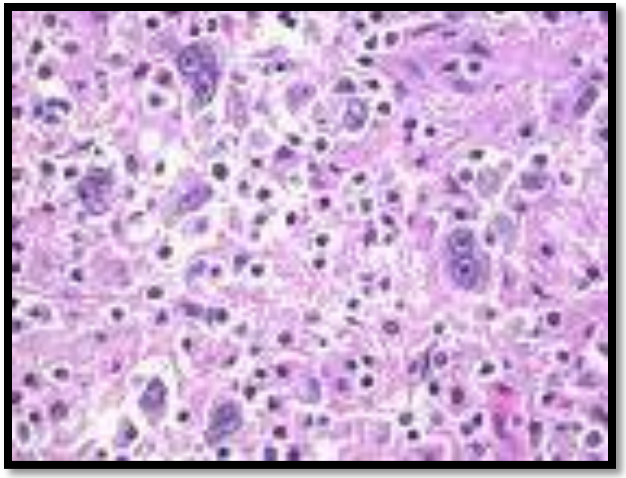
Classical Hodgkin's lymphoma

Reed -Sternberg cells

Mirror image nuclei **OWL EYES**

Finely granular cytoplasm

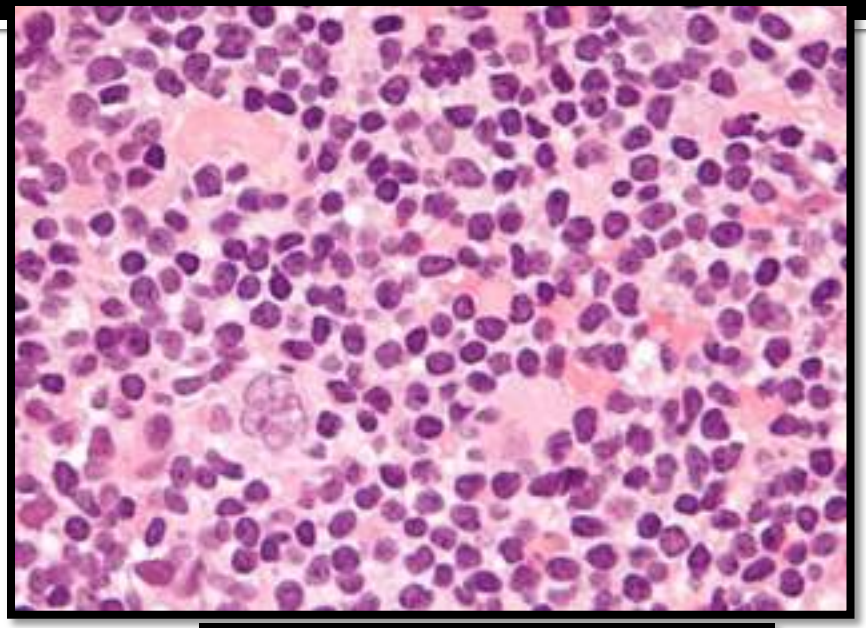


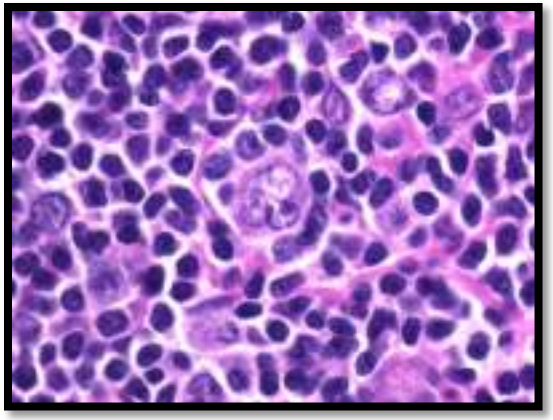


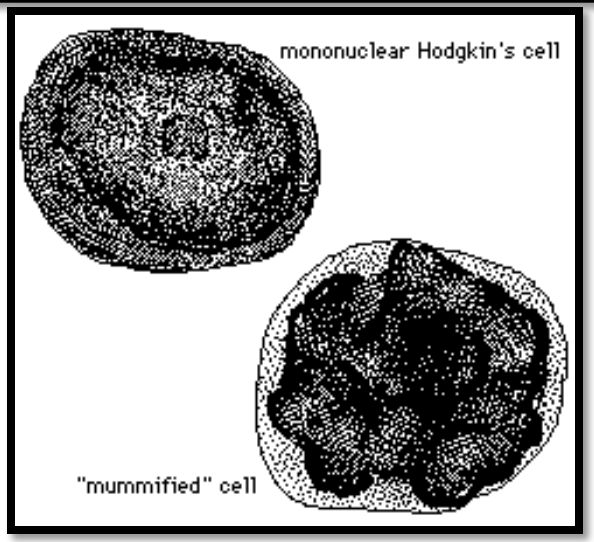
Histopathology

Nodular lymphocyte predominate lymphoma

POPCORN cells







Tretment

Surgery + Radiation +chemotherapy

Non Hodgkin's lymphoma

Lympho proliferative malignancies which can involve both lymph nodes & extra nodal tissues

2nd most common sarcoma

Relatively late complication of HIV infection

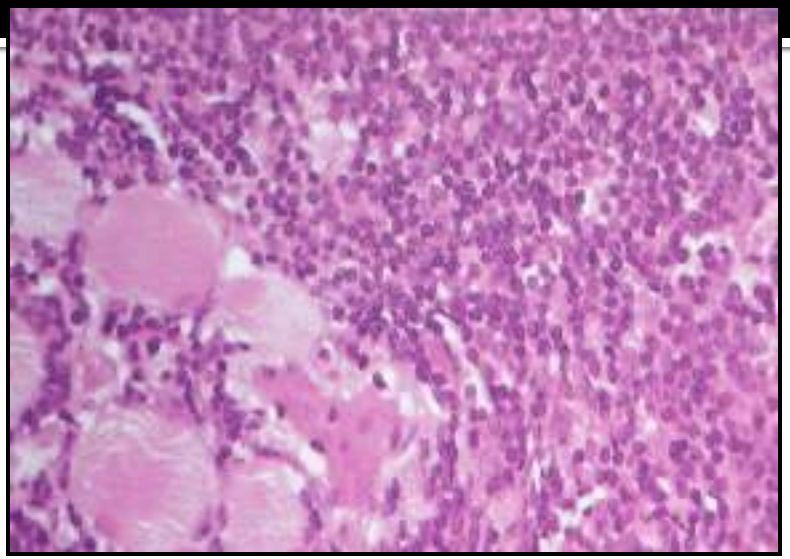
Etiology

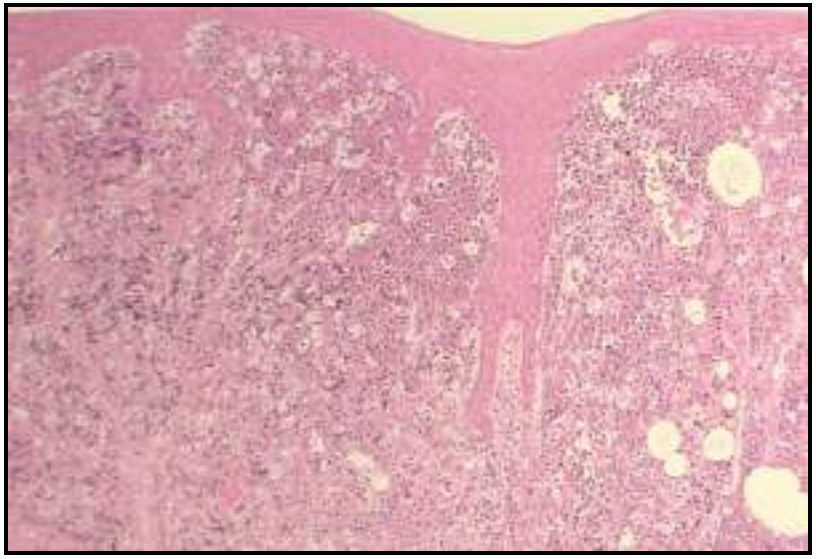
- Chromosomal abnormality
- Co existence with other sarcomas
- Certain chemicals – pesticides, organic chemicals
- EB virus
- Immunodeficiency & HIV related

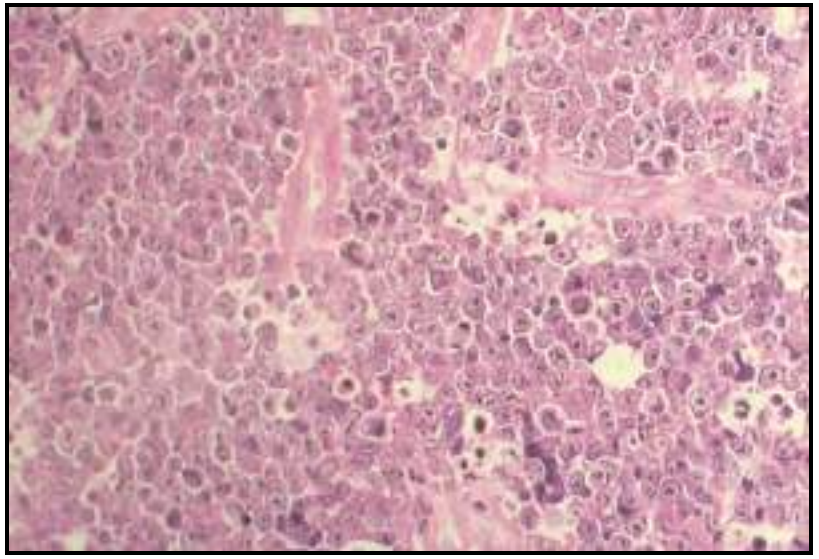
Clinical features

- Typically exhibited in extra nodal locations (75%)
- CNS (1/5th) the most common site
- Gingiva, faucial pillars & palate
- Rapidly growing soft tissue mass, non – tender & diffuse
- Boggy, erythematous / purplish, may / may not be ulcerated.
- Lymphedenopathy, and systemic symptoms of illness

Histopathology







Tretment

Surgery + Radiation +chemotherapy

Burkitt's lymphoma

- African jaw lymphoma
- Denis Parsons Burkitt

High grade B cell lymphoma

1. Endemic (African)
2. Non endemic

Etiology

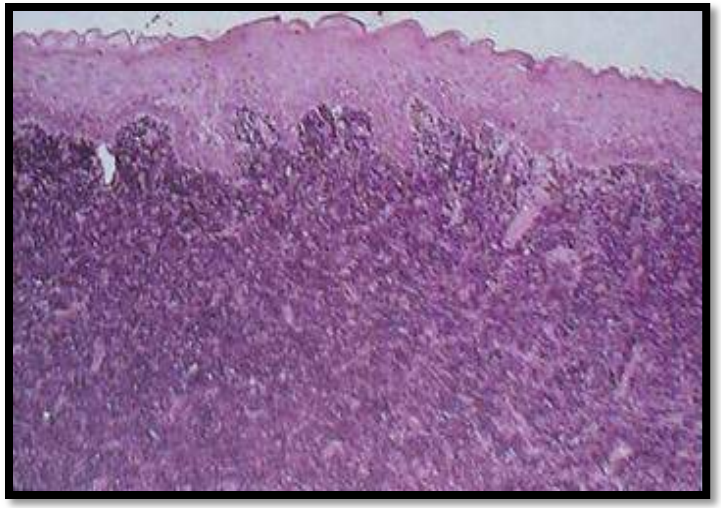
EB virus

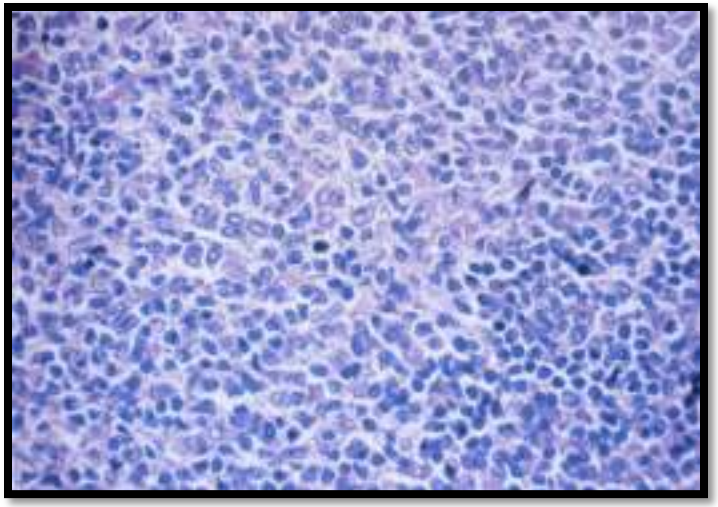
Clinical features

AFRICAN	NON AFRICAN
Jaw commonly involved	Abdominal organs ileum, cecum
Swelling of the affected bone	Swelling and pain in the affected area
Loosening of teeth	Bowel obstruction
Swelling of lymph nodes (non tender)	
Rapidly growing	

Histopathology

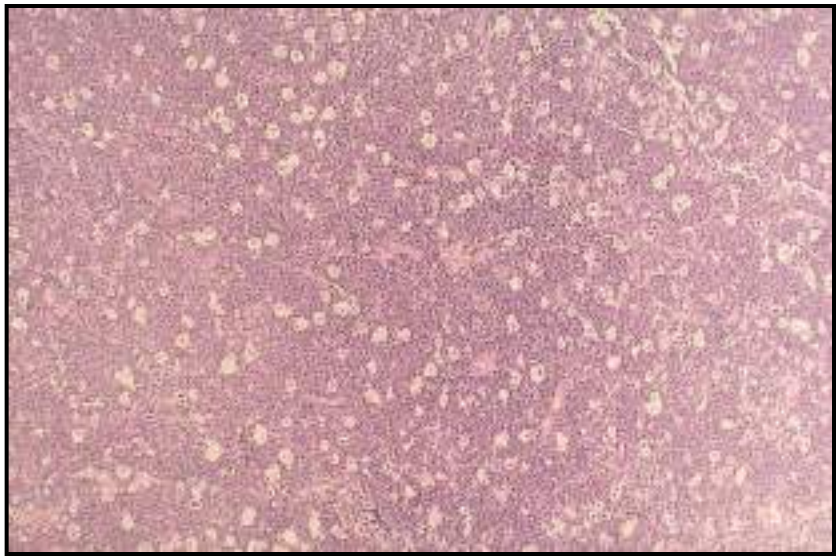
- Sheets of large lymphoid cells, less differentiated
- Nodular or follicular pattern
- Minimal cytoplasm and altered n:c
- Destruction of the normal adjacent host tissue by infiltration

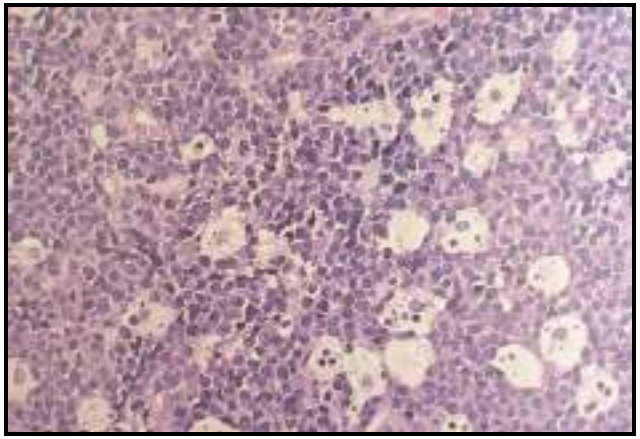




“Starry – sky”

Interspersed macrophages with abundant cytoplasm (stars) set against a background of malignant darkly staining lymphoma cells (night sky)





Tretment

Surgery + Radiation +chemotherapy

Multiple Myeloma

- Most common primary neoplasm of the skeletal system
- Malignancy of plasma cells

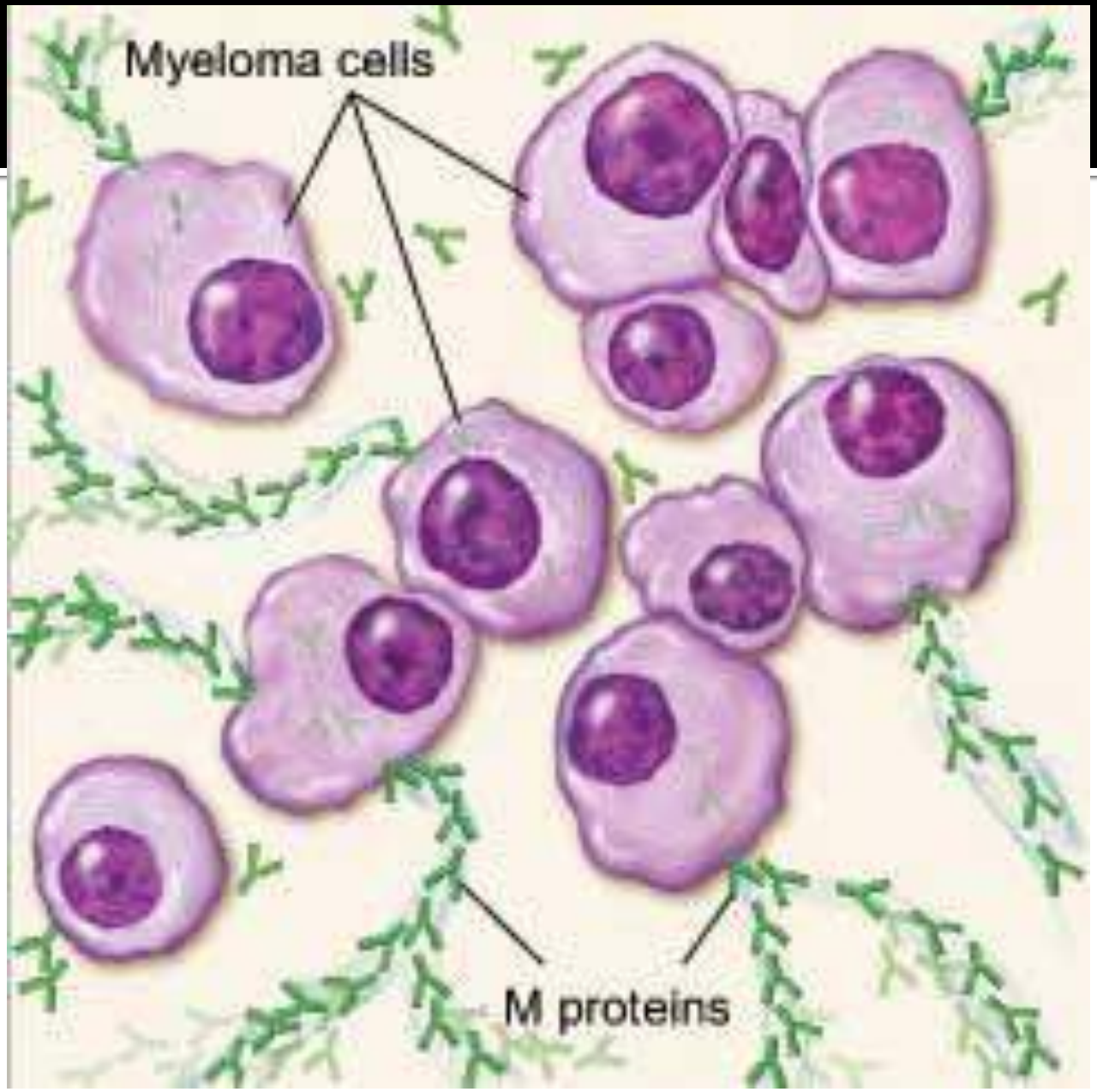
Multiple Myeloma

- B- lymphocytes → Plasma cells → Antibodies

- Plasma cell → Malignant transformation → Clonel proliferation → Production of single type(monoclonal) of antibody

Pathogenesis

Expansion(proliferation) of single line of plasma cells that replace normal bone marrow and produces monoclonal immunoglobulins


Myeloma cells
M proteins

Etiology

- Radiation exposure
- Occupational exposure -- certain chemicals like benzene, formaldehyde, hair dyes, paint sprays, asbestos
- Leather industries, agriculture – rubber plantation
- Atomic bombing in Japan – increased incidence of MM

Clinical feature

- Any age group – middle aged
- M>F
- Diffuse disease of the bone marrow
- 90% cases – osseous involvement

Clinical feature

- Vertrabral column, ribs, pelvis, femur bone
- Bone pain specially from compression fractures of ribs
- Approximately 20% free of symptoms – diagnosed accidentally
- Number of lytic foci or diffused demineralization of bone
- Lymphedenopathy & hepato-splenomegaly

Oral manifestations

- Mandible > maxilla
- Ramus , angle and molar region
- Pain, swelling and expansion of the jaw
- Numbness and mobility of the teeth
- Extra osseous lesions – gingival epulids

Radiographic features

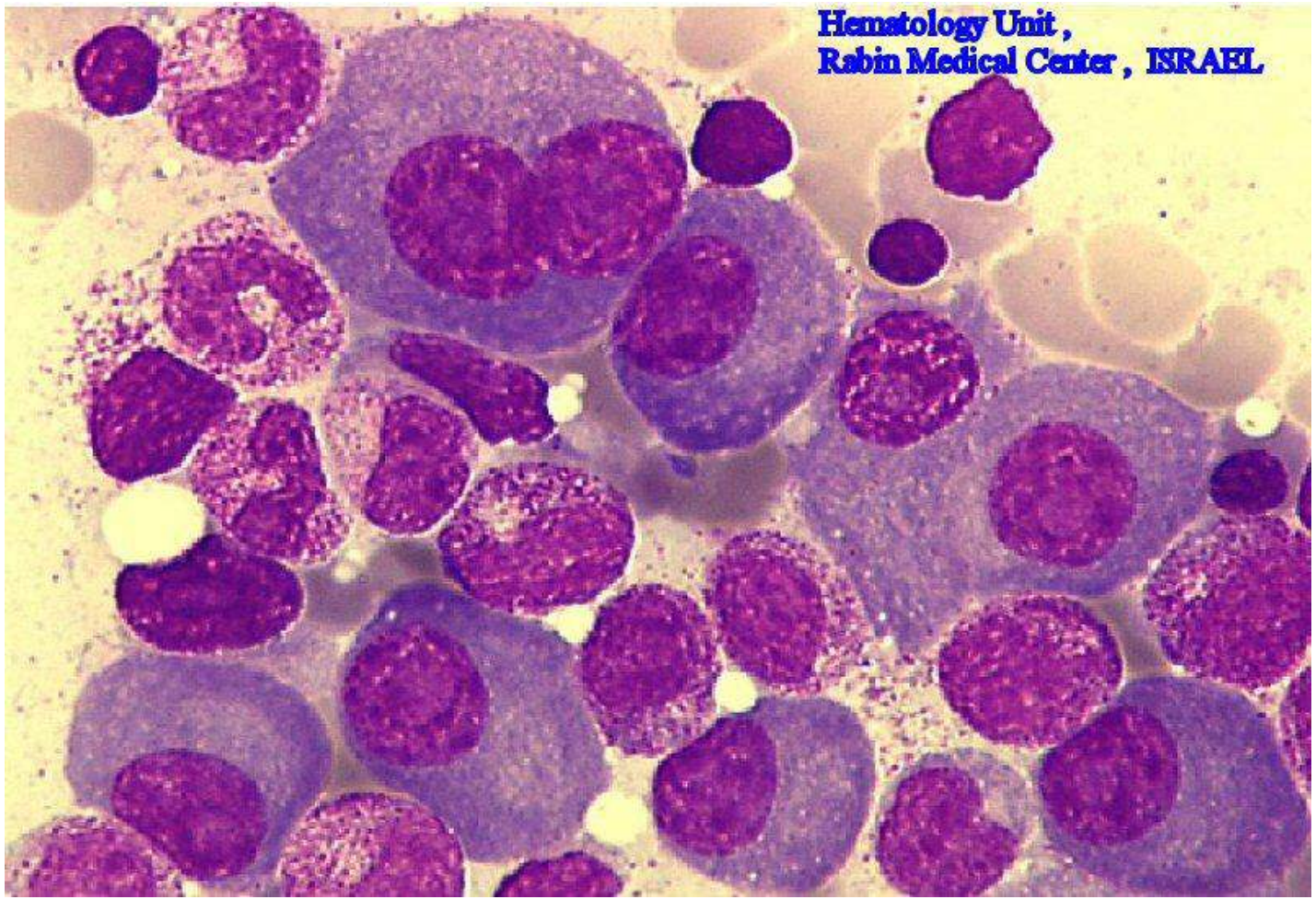
- Sharply punched out areas
- Occurs in the active sites of haemopoesis
- Few mm to cm
- Sometimes diffuse destruction of the bone

Laboratory findings

- Hyperglobulenemia (monoclonal gammopathy)
- Total Serum protein level –8-16gm percent
- BENCE – JONES PROTEIN in the urine (60-65% of patients)
- Un usual protein which coagulates when urine is heated to 40-60^0 C, and then disappears when the urine is boiled, reappears when it is cooled
- Monoclonal hyper gamma globulinemia, IgG myeloma, IgA myeloma
- Hypercalcemia – bone destruction

Histological features

- Sheets of closely packed cells resembling plasma cells
- Round to ovoid cells with eccentrically placed nuclei exhibiting chromatin clumping in a **cartwheel or checkerboard** appearance
- Perinuclear halo
- Russell bodies


Hematology Unit ,
Rabin Medical Center , ISRAEL

Treatment

Mainly chemotherapy

OSTEOSARCOMA

Common primary cancer of bone

Young adults - 10 and 25 years, Mandible> maxilla. Males>females – 1.6:1

Rare in later age – Secondary to previous irradiation or Paget's disease

The five-year survival ~ 60%

Commonly affects long bones

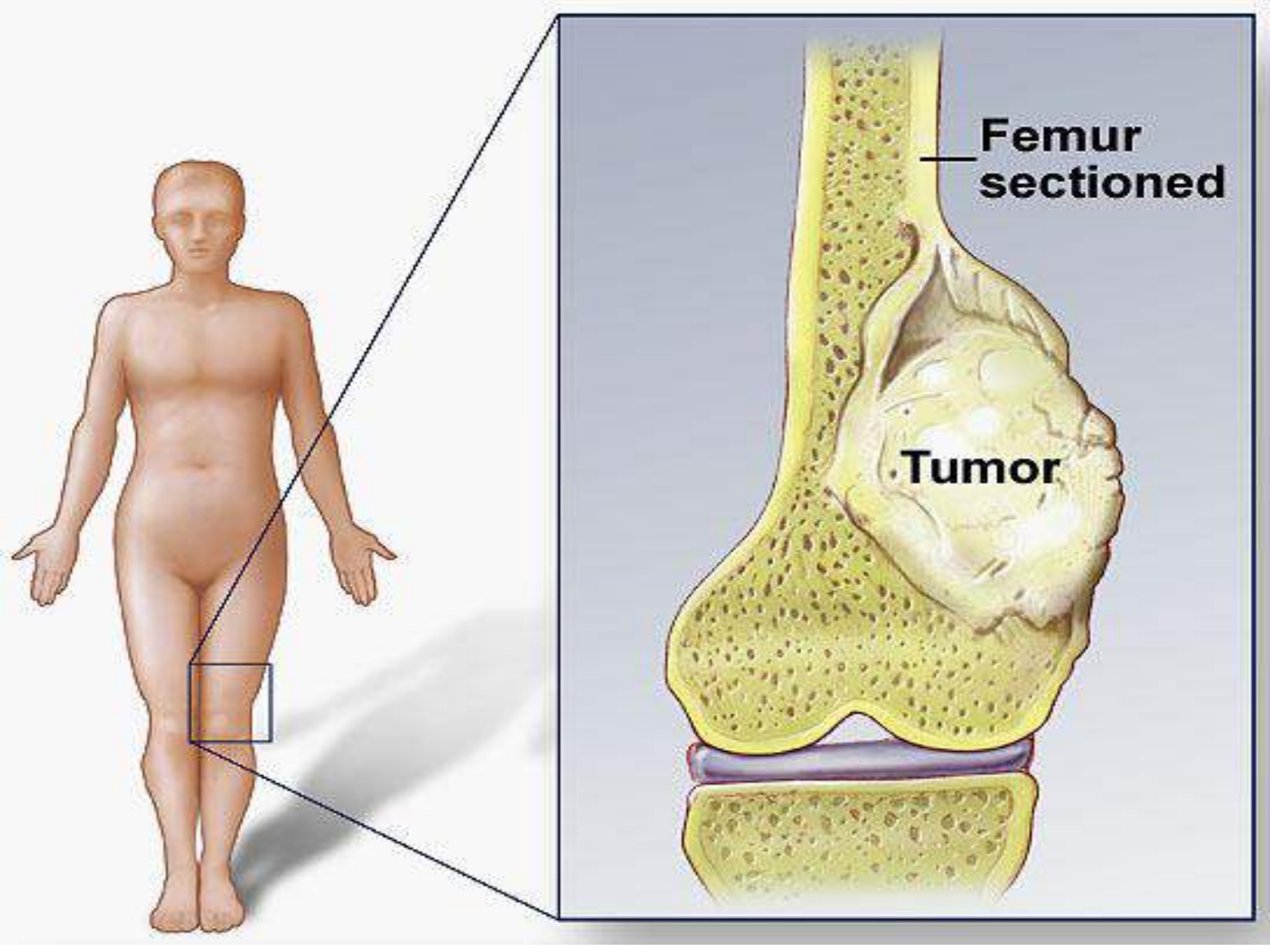
Etiology

Unknown.

Ionizing radiation, trauma, Fibrous dysplasia,

Paget's disease are considered the predisposing factors.

Li-Fraumeni syndrome (Chromosomal aberrations in p53 and retiniblastoma gene localized to 17p13 & 13q14respectevely).


Femur
sectioned
Tumor

Clinical Features -- Jaw osteo-sarcoma:

- Painless swelling and growth of jaw as first signs.
- Mobility of the teeth of the affected areas.
- Asymmetry of the face
- Parasthesia

Additional features:

- If maxilla is involved: Epistaxis, nasal obstruction, eye problem,(diploplia)
- Headache and trismus may be present.
- The tumour may extend to the infratemporal fossa and to the maxillary sinus.

- Osteosarcoma can be broadly classified

Intra-medullary
&
Juxta-cortical

Osteosarcoma – gross

Osteosarcoma – gross

Radiography – Variable findings;

1.Sometimes seen as a periapical radiolucency.
Sometimes tumour invades periodontal ligament space - widening of Peridontal space on one or both sides of the tooth- **Garrington's sign**.

2. Radiolucent –radio-opaque lesions-ragged ill defined borders, opaqueness resulting in excessive bone production, intermingled with Radiolucent foci of bone destruction giving **"Cumulus Cloud appearance"**

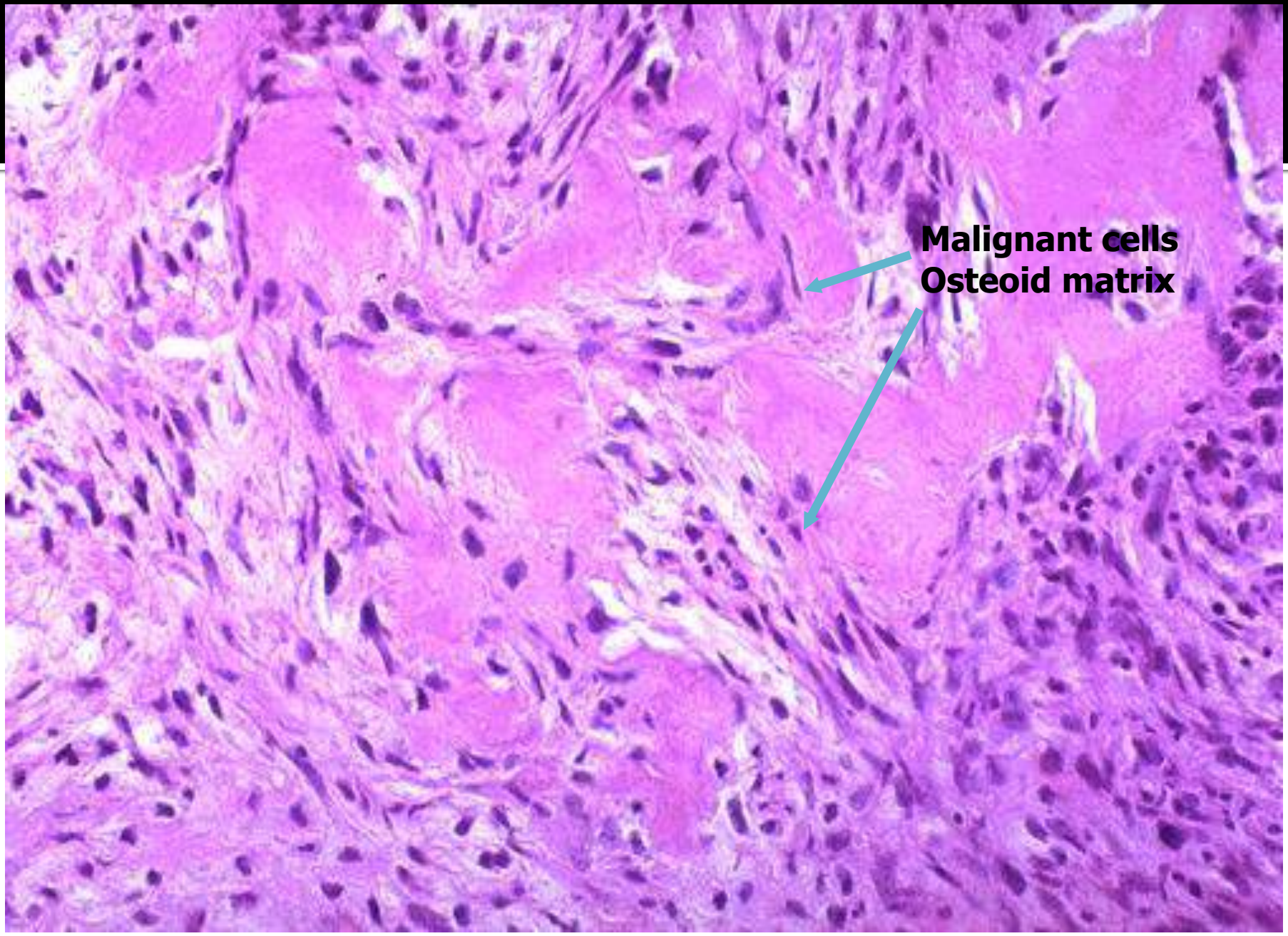
3.Sclerotic type with thin striae of bone projecting perpendicullarly from tumour surface giving **" sunburst appearance."**

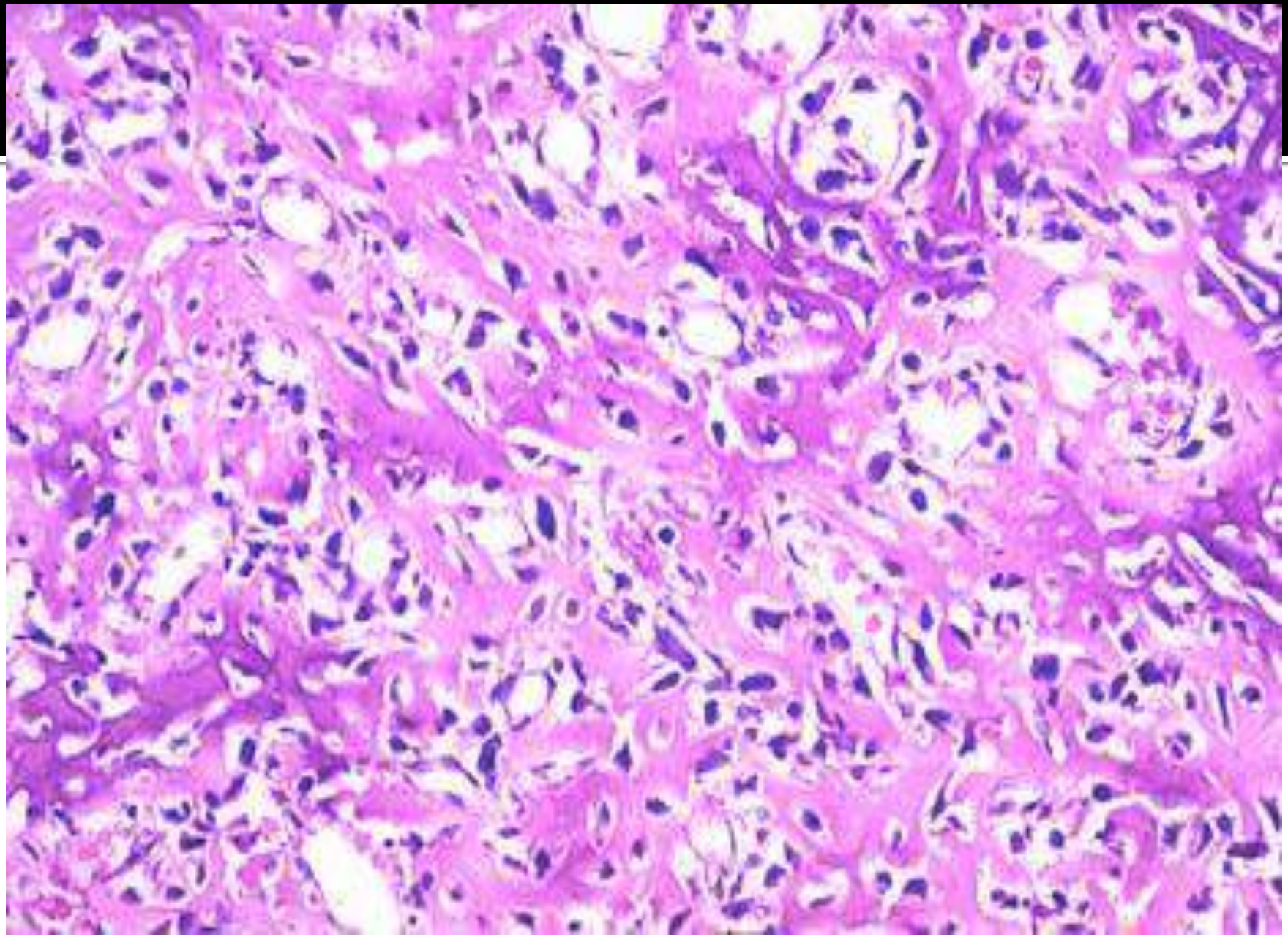
HISTOLOGICALLY,

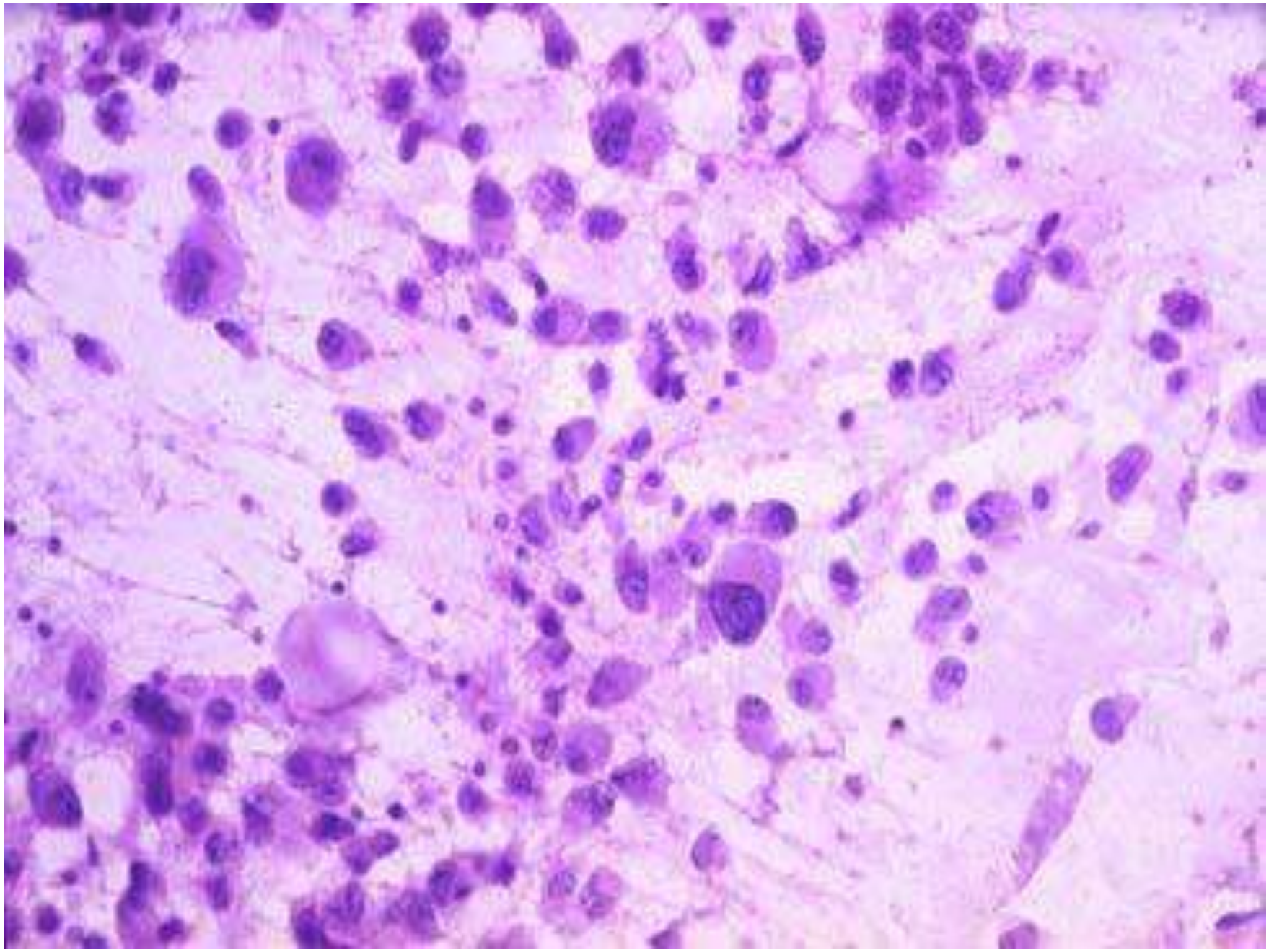
1. **Osteoblastic,**
2. **chondroblastic,**
3. **fibroblastic,**
4. **Telengiectatic,**
5. **small cell,**
6. **giant cell,**
7. **spindle cell variant.**

Histopathology

- Osteosarcoma is characterised by proliferating Malignant spindle cells which directly forms osteoid or immature bone
- These neoplastic Osteoblasts are pleomorphic, hyperchromatic and arranged in a disorderly fashion.
- Chondroblastic type shows cartilagenous tissue rimmed by malignant spindle cells.
- Other Investigations: X rays, CT and MRI –


Malignant cells
Osteoid matrix

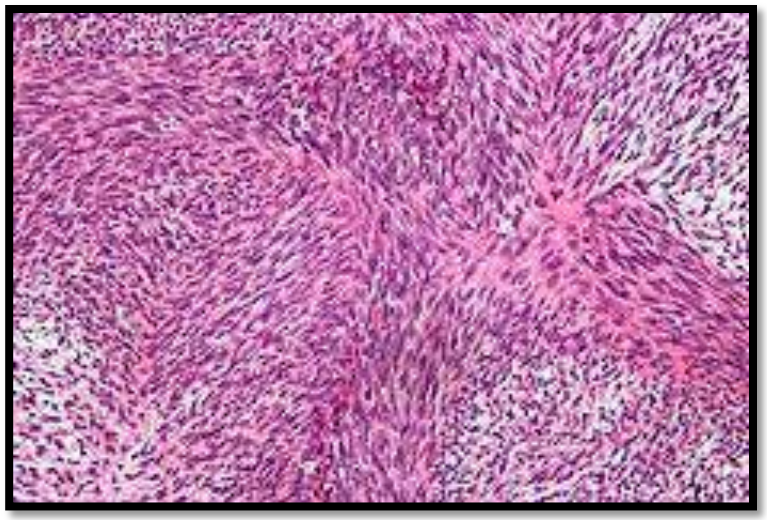


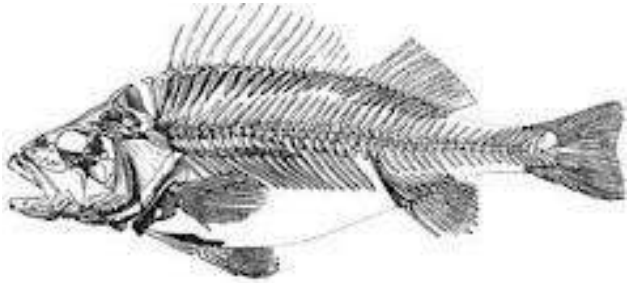


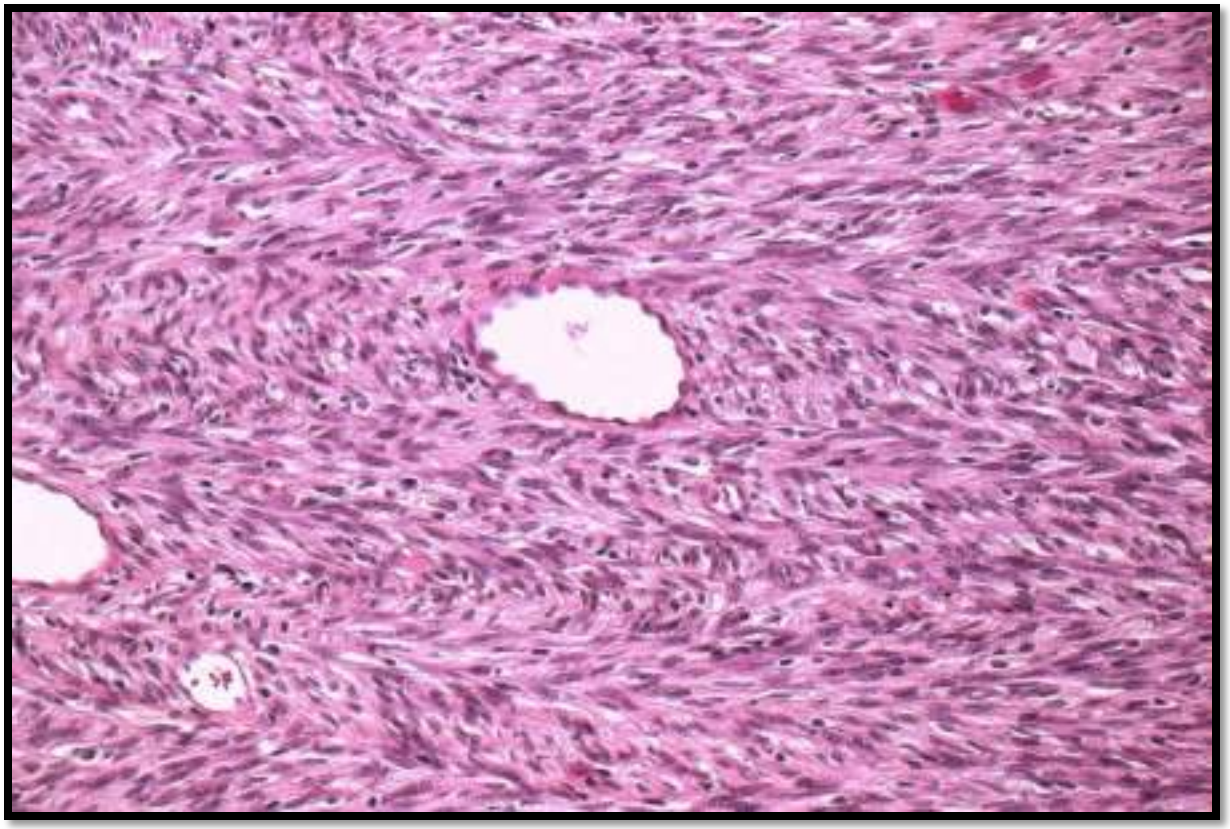
TREATMENT

Surgery
&
Chemotherapy

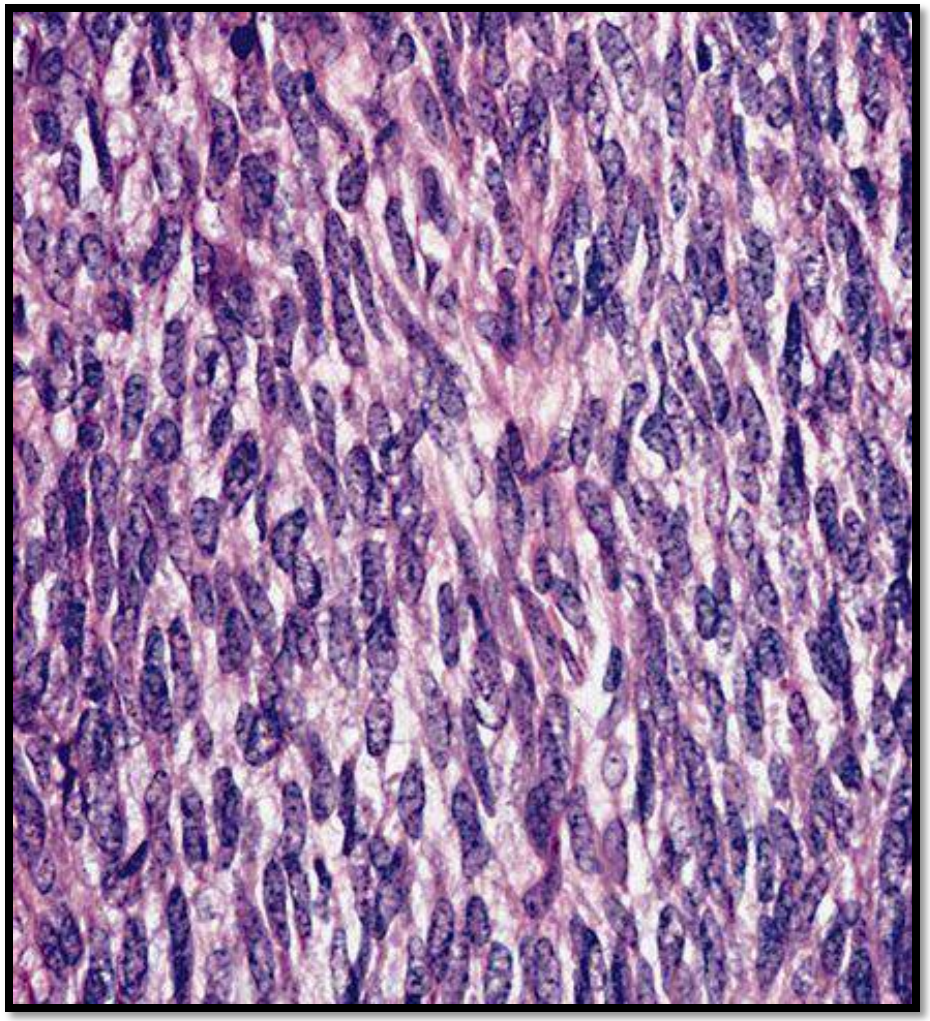
Re - cap

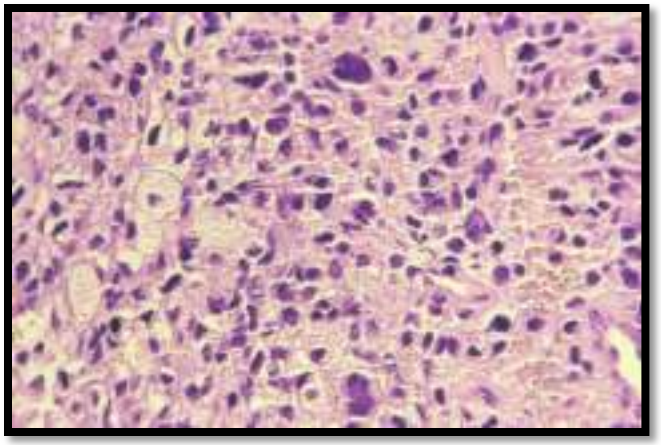


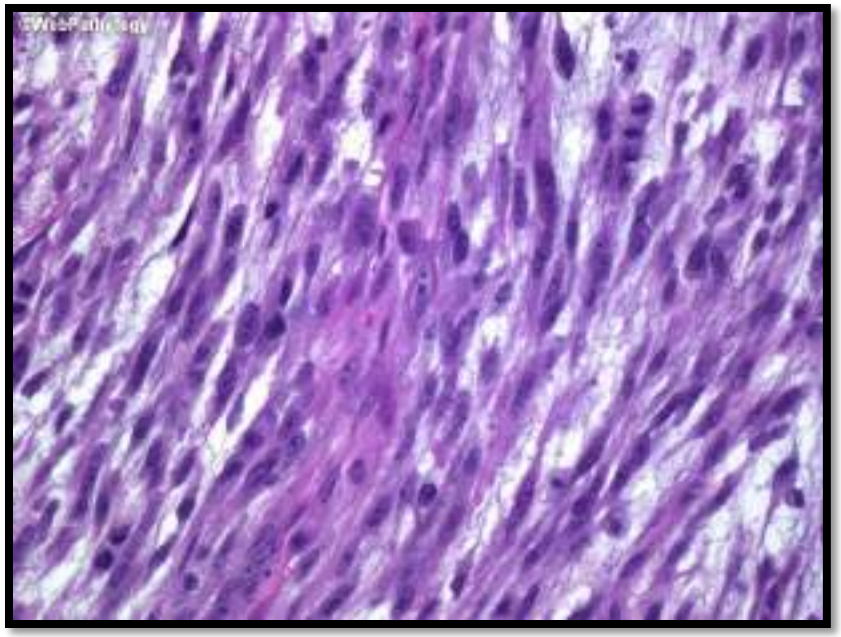




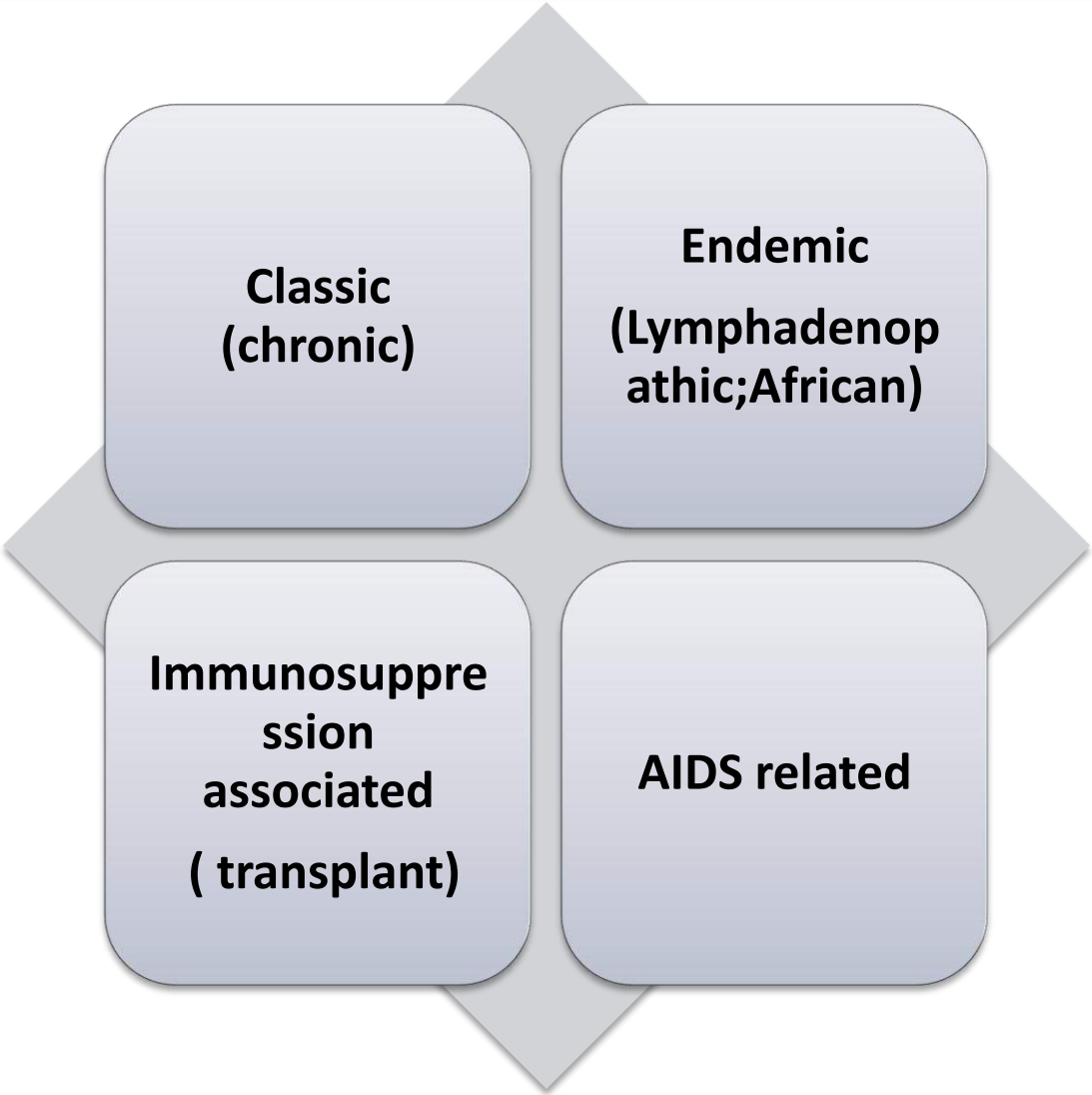
Intermediated grade of fibro sarcoma



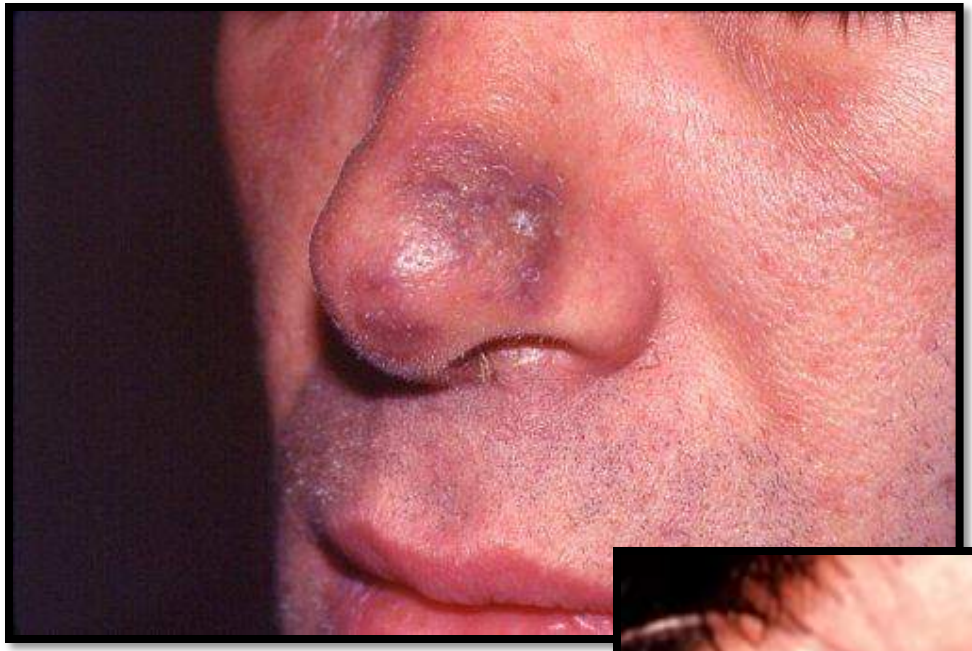




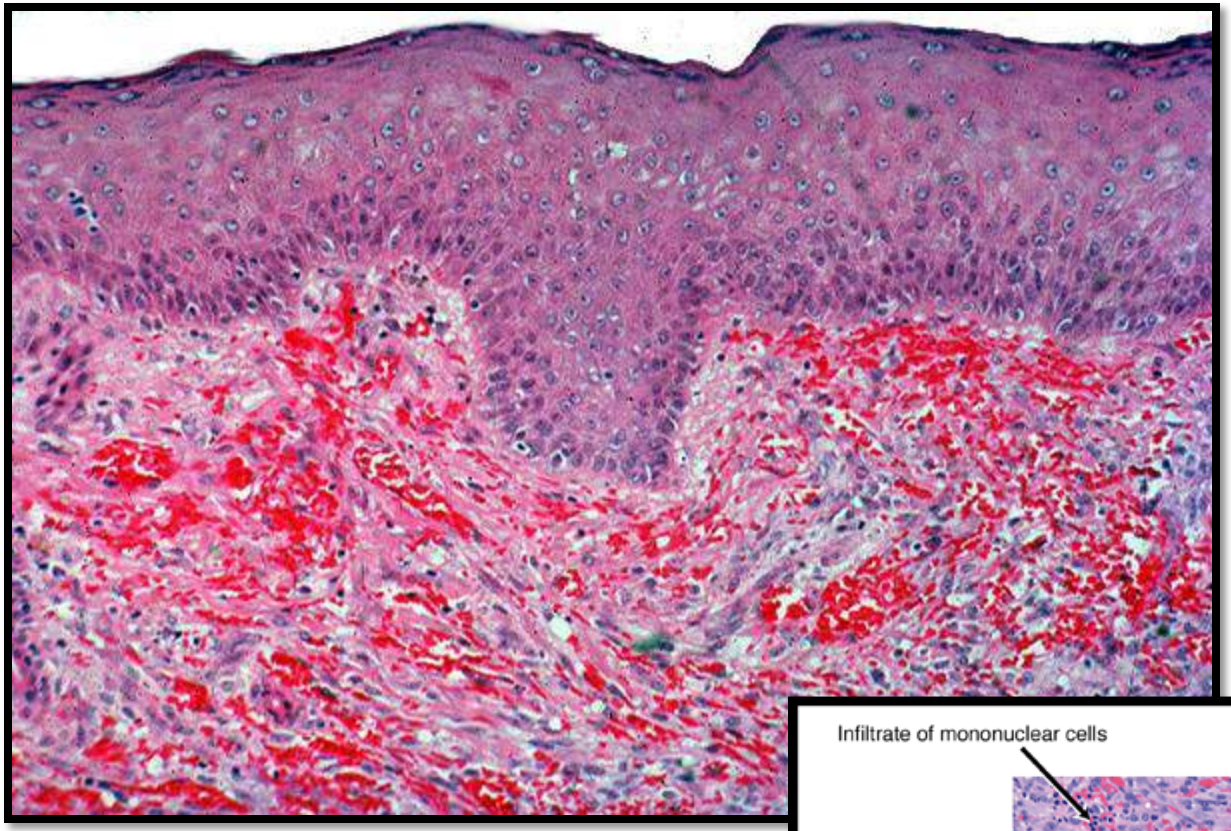
High differentiated fibro sarcoma

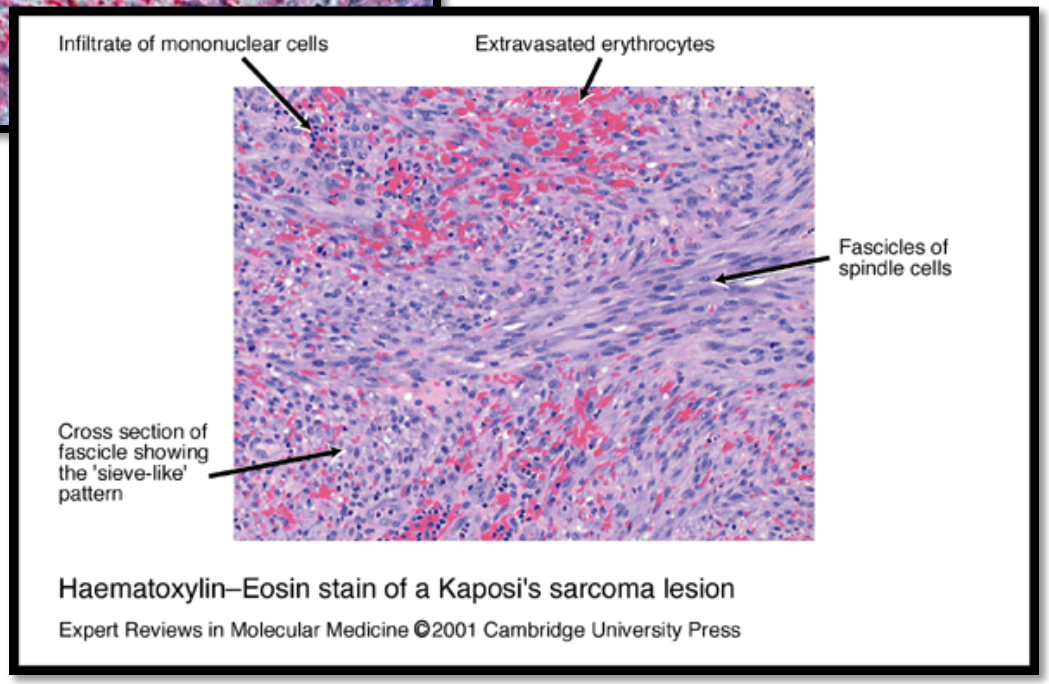

Classic
(chronic)
Endemic
(Lymphadenopathic;African)
Immunosuppression associated
(transplant)
AIDS related

Kaposi sarcoma



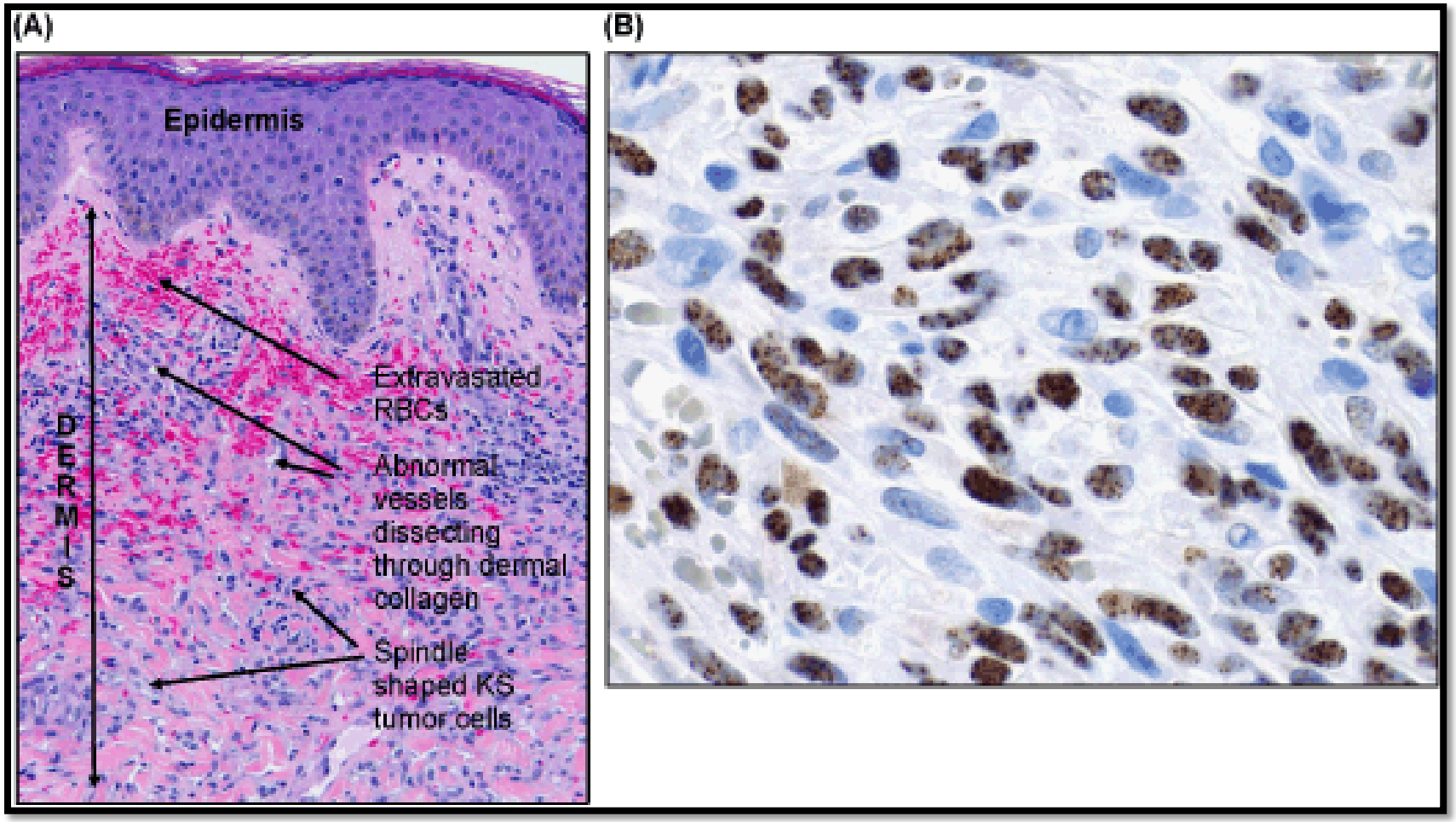
Kaposi sarcoma





Haematoxylin–Eosin stain of a Kaposi's sarcoma lesion

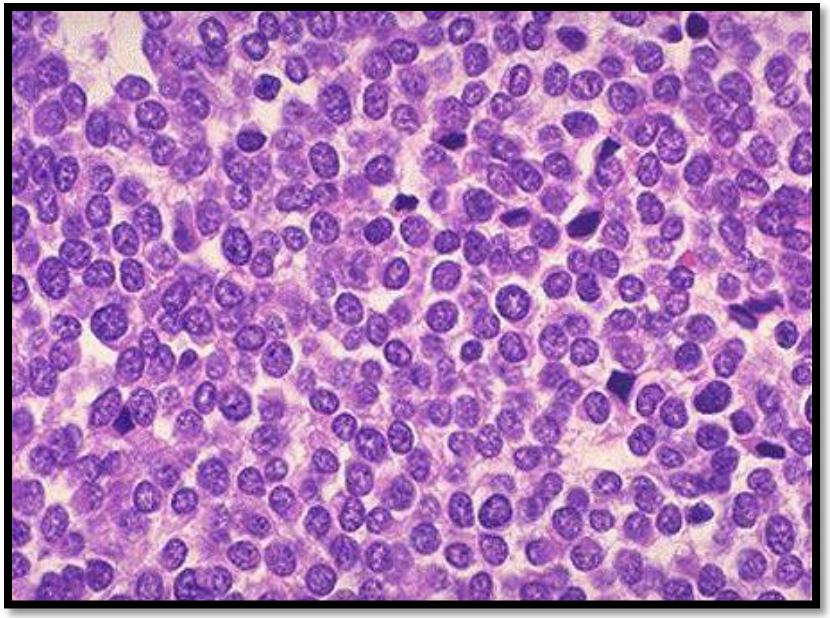
Expert Reviews in Molecular Medicine ©2001 Cambridge University Press


(A)
Epidermis
DERMIS
Extravasated RBCs
Abnormal vessels dissecting through dermal collagen
Spindle shaped KS tumor cells
(B)

Kaposi sarcoma

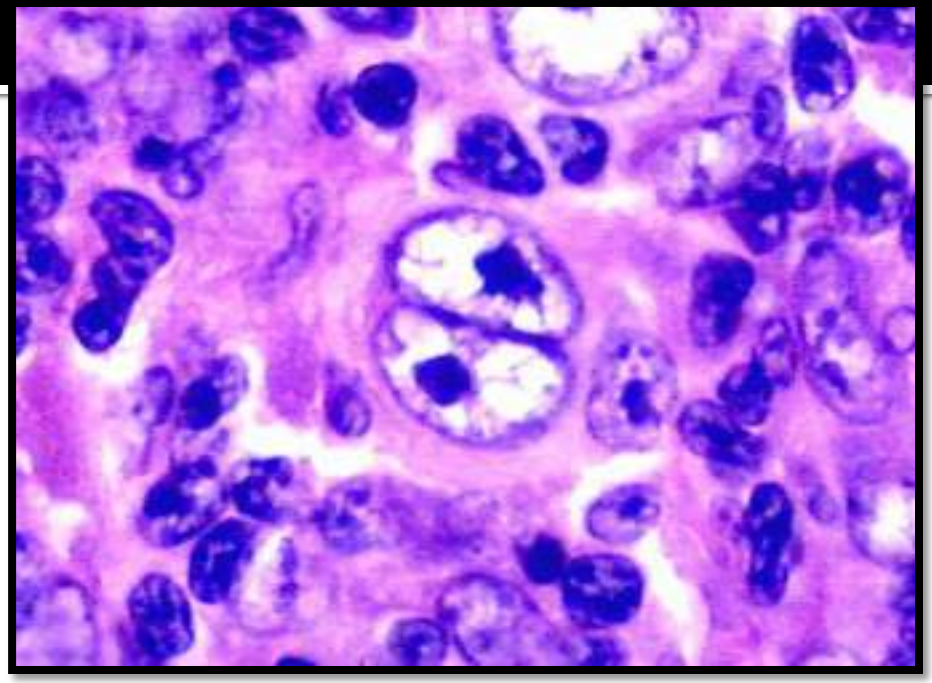
Histopathology

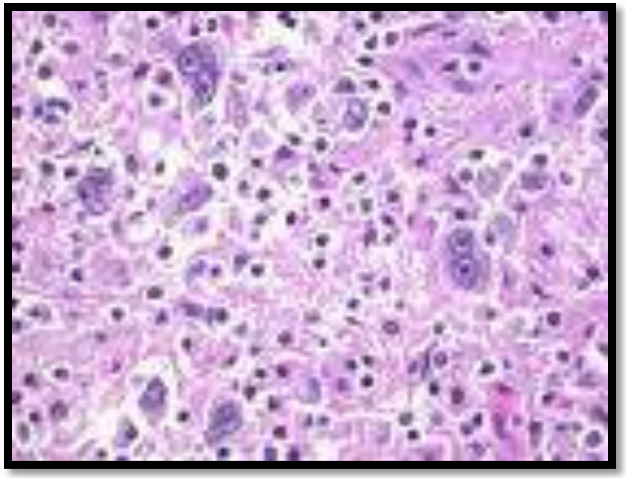
- Extremely cellular
- Solid sheets or masses of small round cells
- Dispersed chromatin & hyper chromatic nuclei, high mitotic figures
- **FILIGREE** pattern
- Positive for glycogen



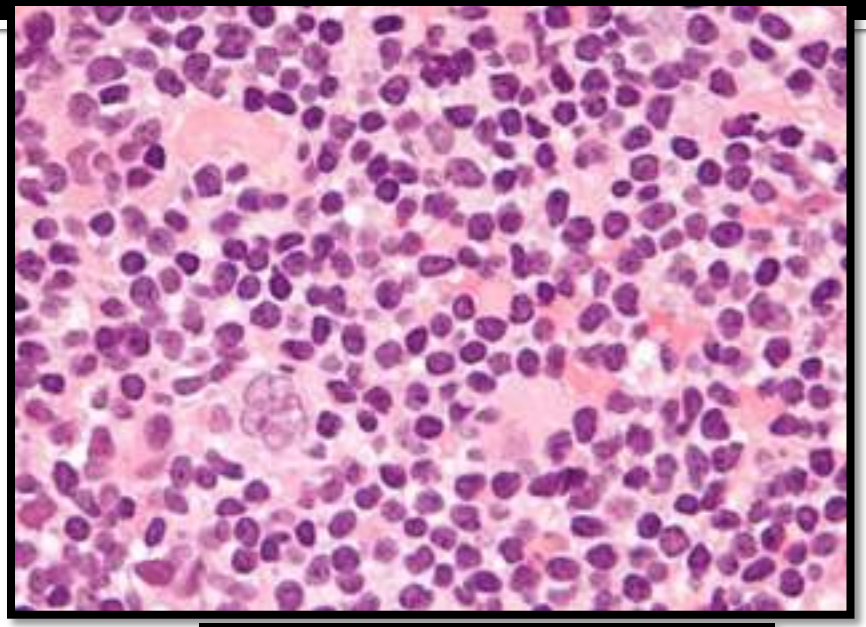
Ewing's sarcoma

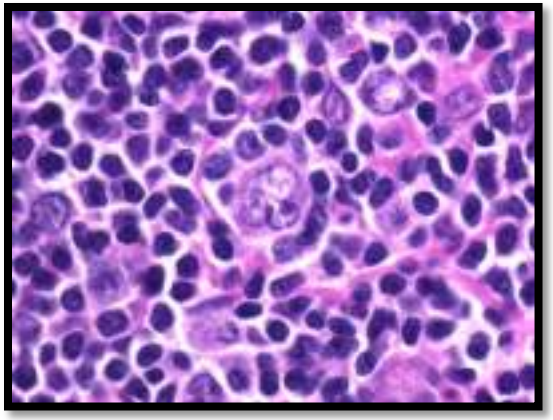
Histopathology

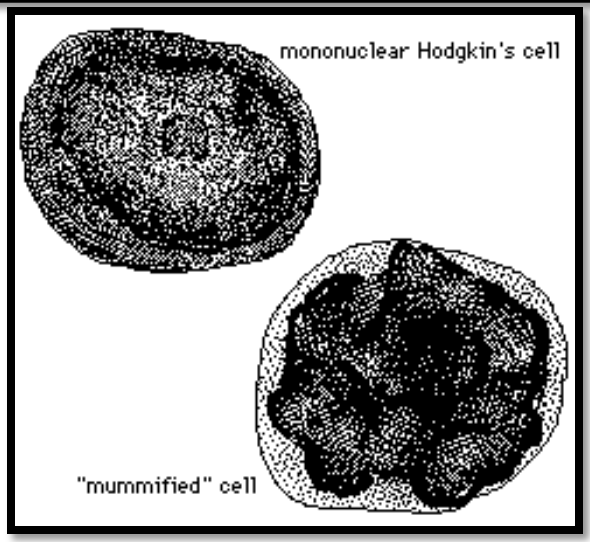




Histopathology



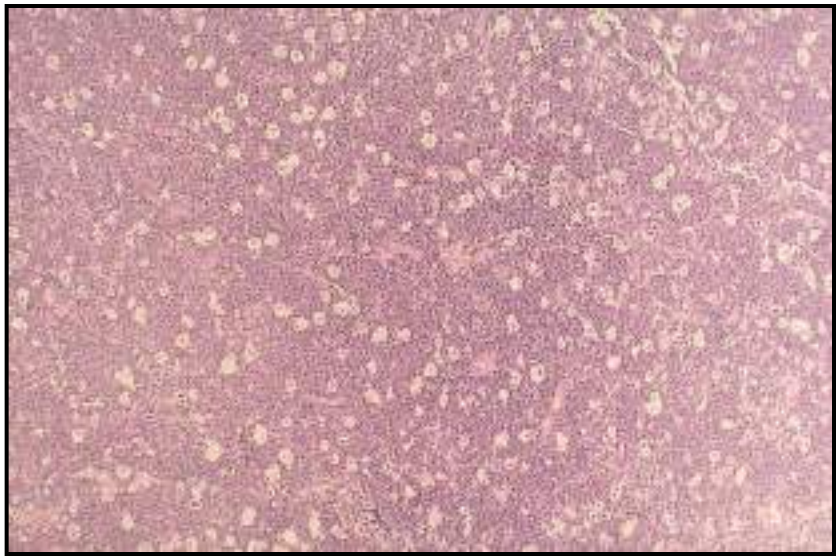


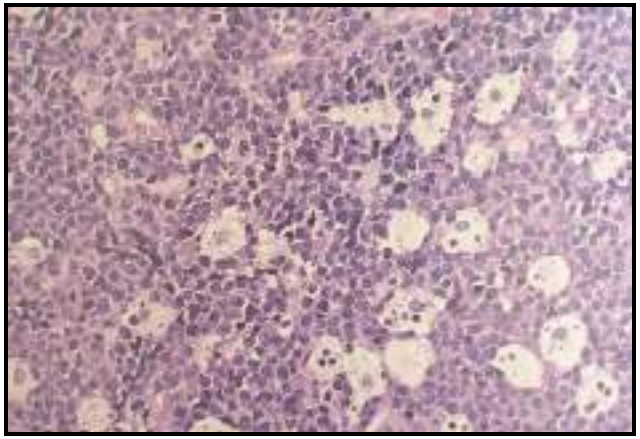


Hodgkin’ s lymphoma
Reed sten berg cells
pop corn cells

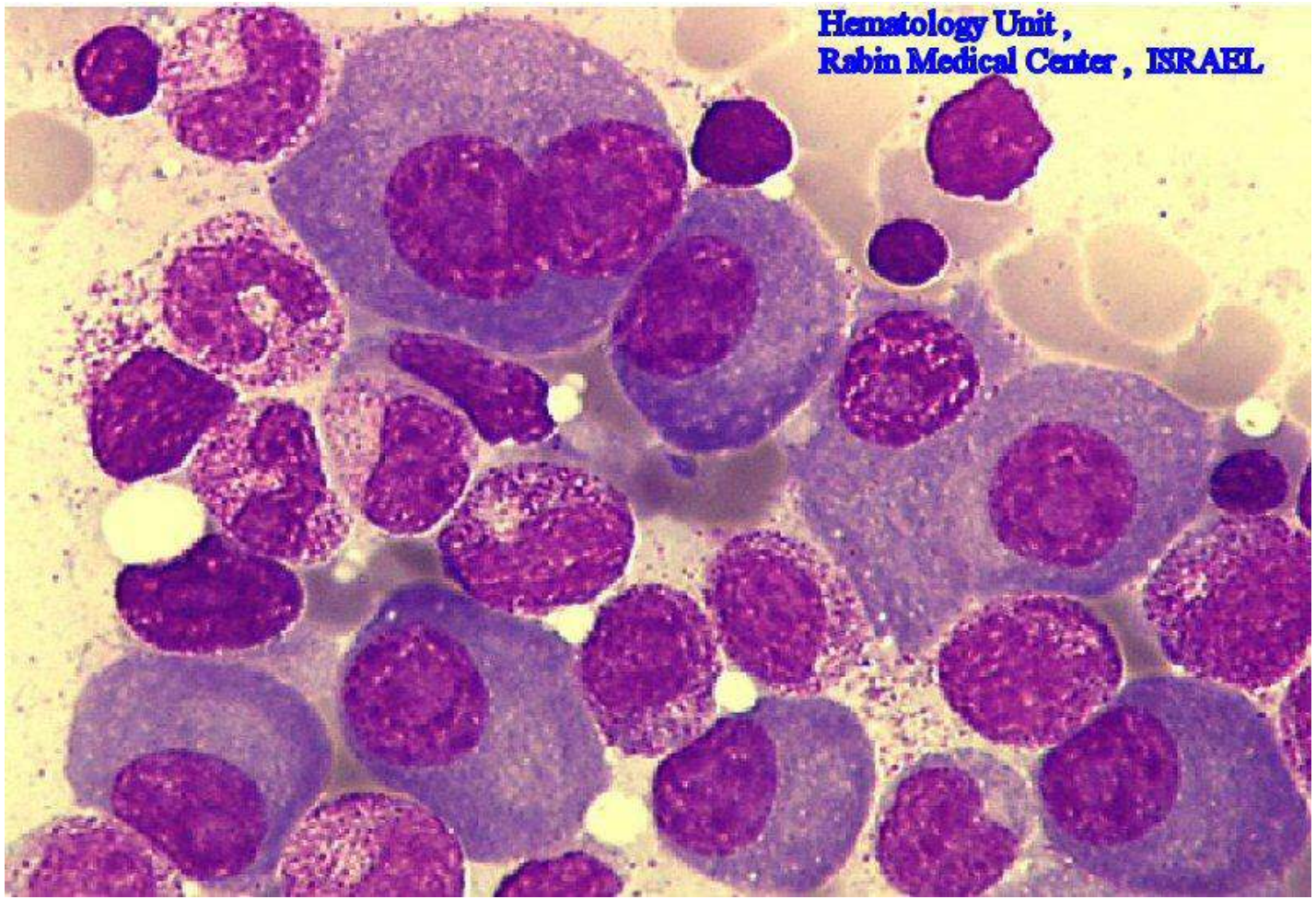
“Starry – sky”

Interspersed macrophages with abundant cytoplasm (stars) set against a background of malignant darkly staining lymphoma cells (night sky)

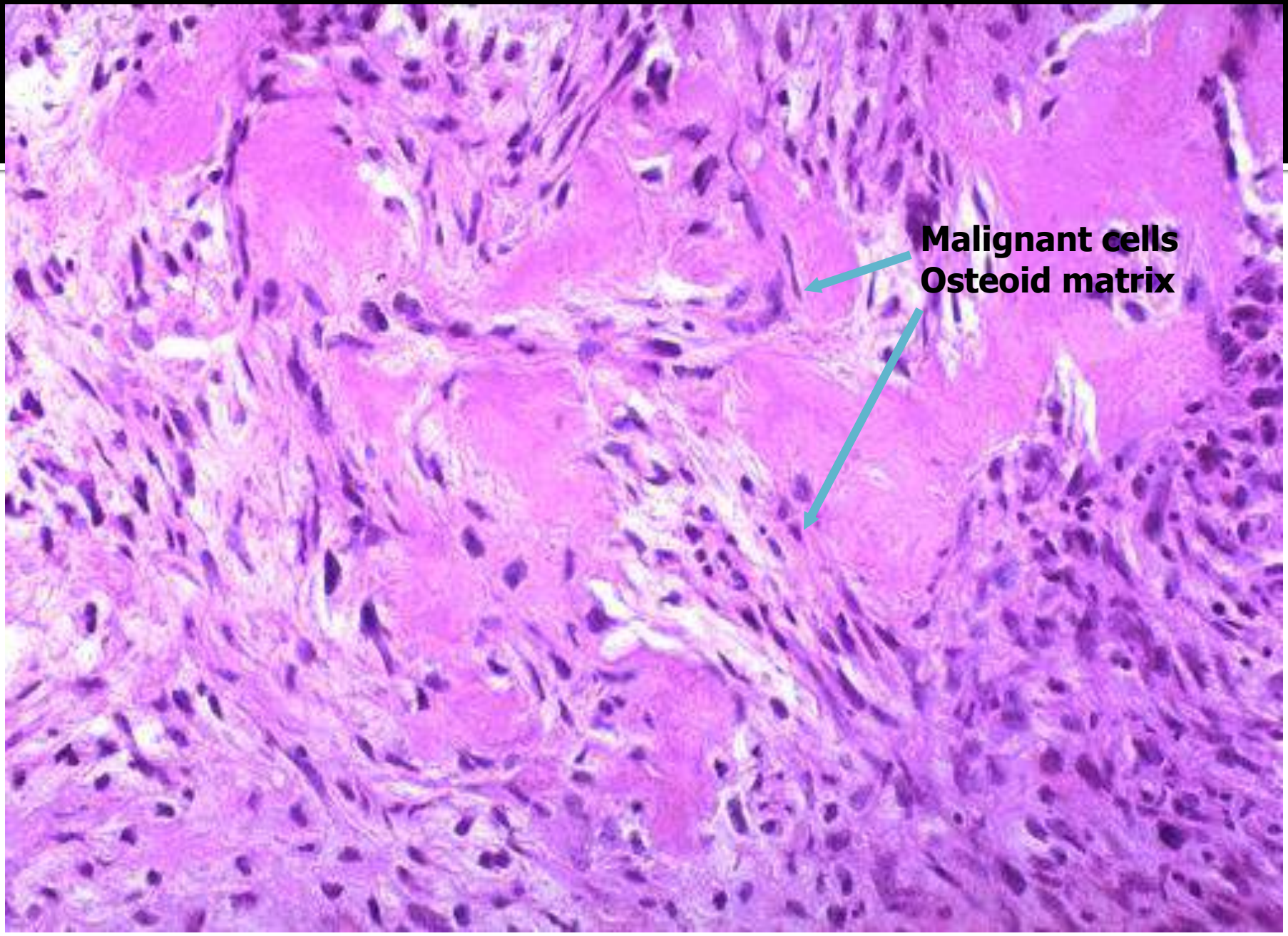


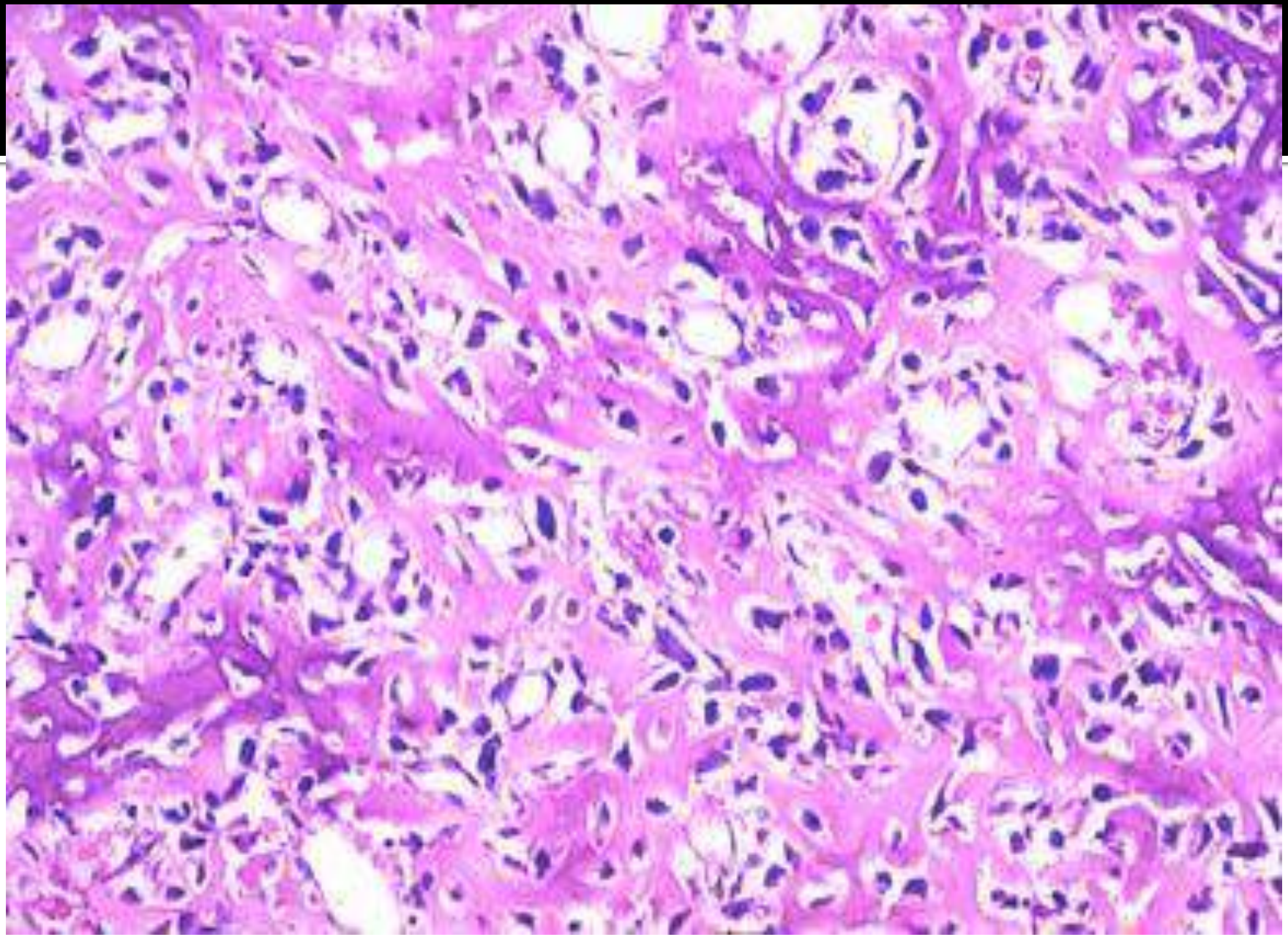


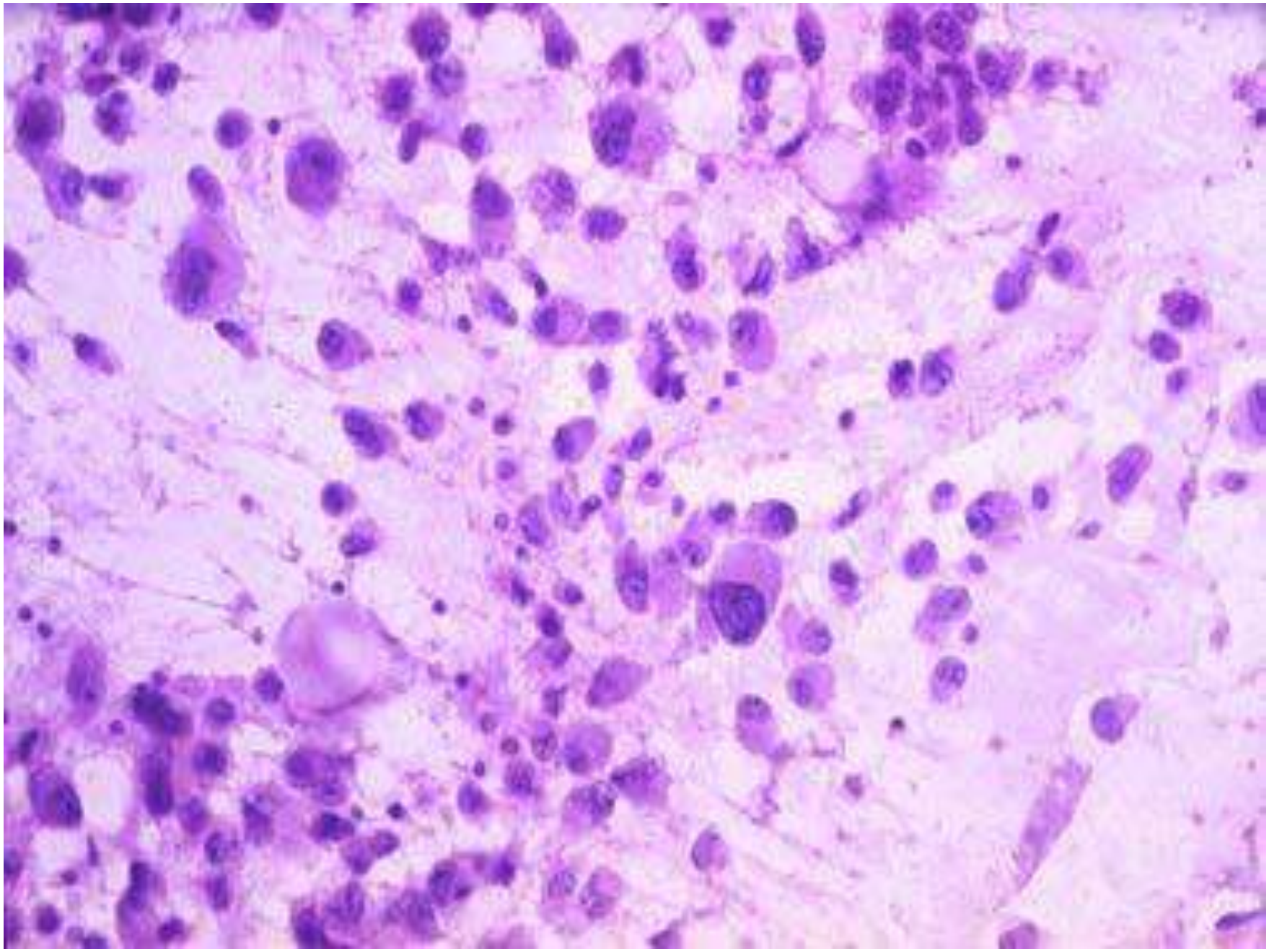
Burkit’ s lymphoma


Hematology Unit ,
Rabin Medical Center , ISRAEL

Multiple myeloma


Malignant cells
Osteoid matrix



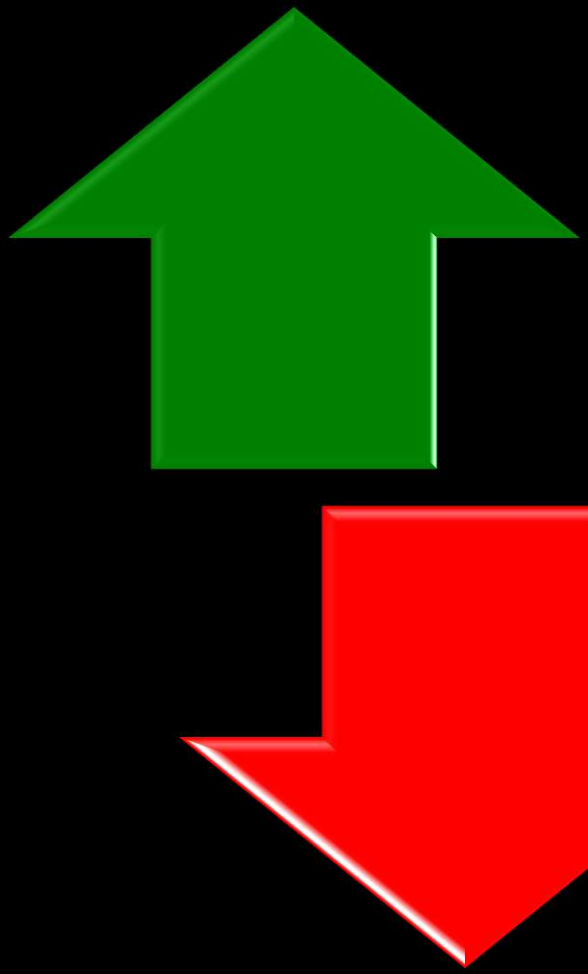


Osteo sarcoma

Thank –u

Odontogenic cysts

‘A cyst is a pathological cavity having fluid, semi fluid, or gaseous contents and which is not created by the accumulation of pus. It is frequently, but not always, lined by epithelium’



True

Epithelial lining

Pseudo

No epithelial lining

Central cavity

Epithelial lining

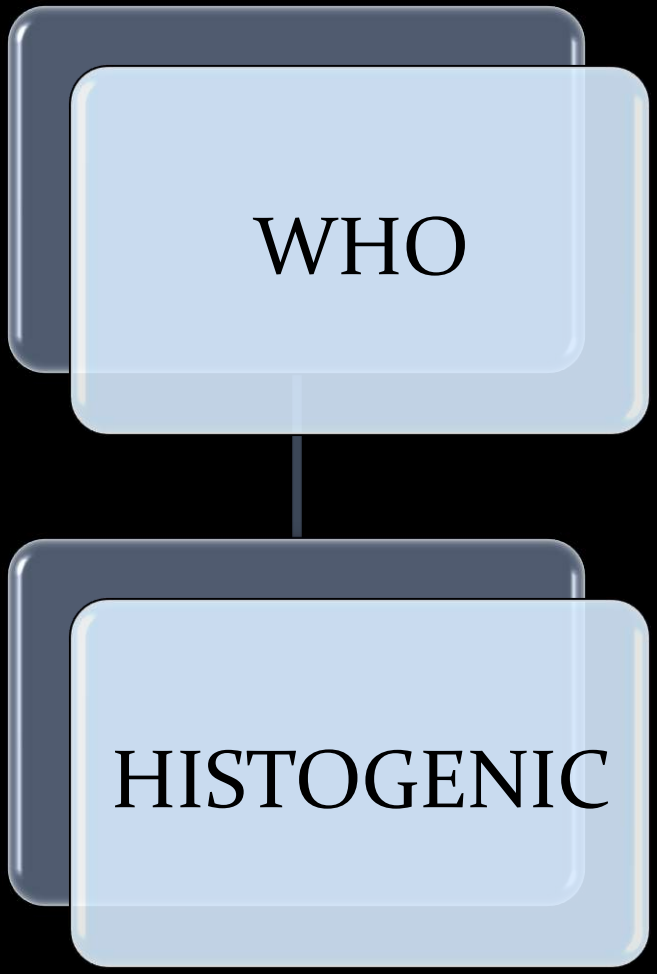
An outer wall (capsule)

Cystic cavity	Fluid, semisolid material, cellular material, debris, keratin, or mucous
Epithelial lining	Keratinized or non keratinized, stratified squamous , pseudo stratified, columnar, cuboidal .
Capsule	Connective tissue components Fibers, vessels, odontogenic cell rests, inflammatory cells.

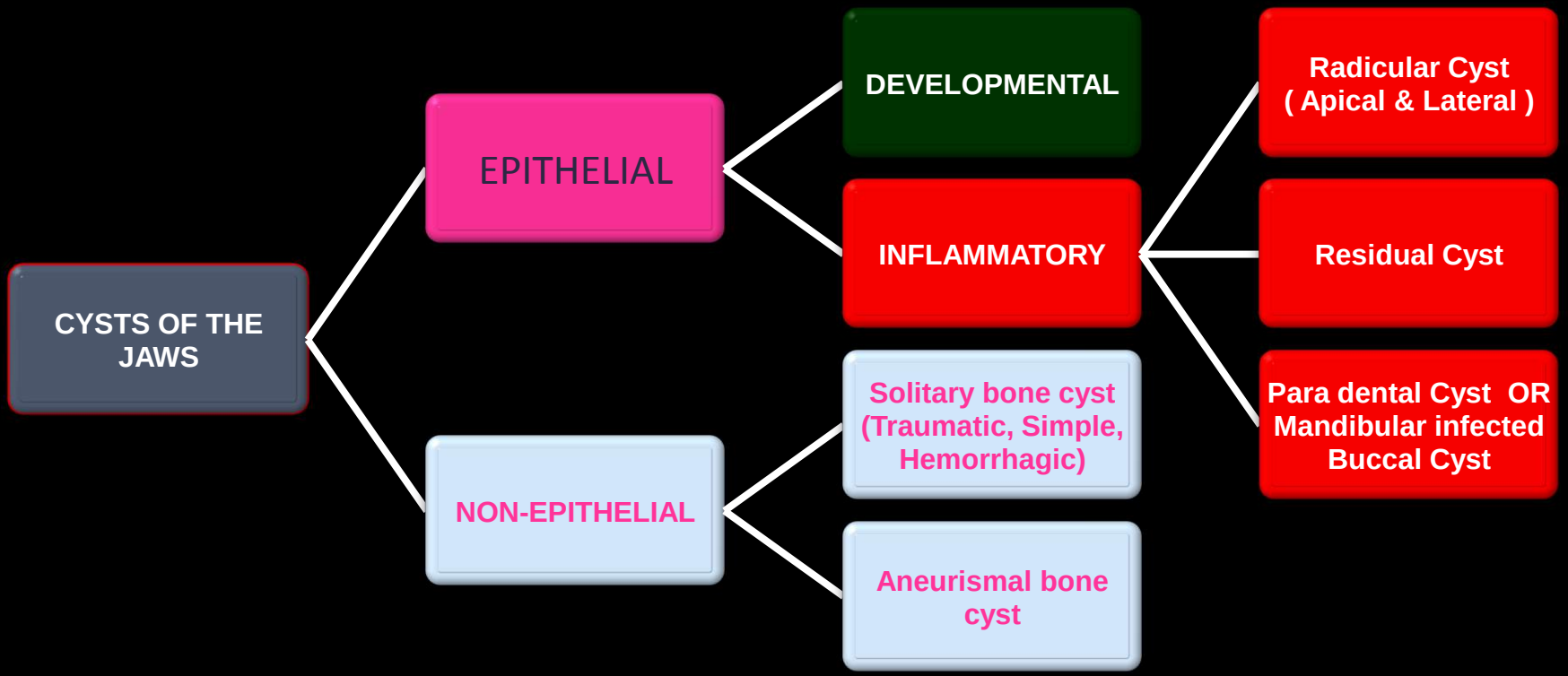
Odontogenic cyst

A cyst in which lining of the lumen is derived from epithelium involved in tooth development.

Classification



WHO


CYSTS OF THE JAWS
EPITHELIAL
NON-EPITHELIAL
DEVELOPMENTAL
INFLAMMATORY
Solitary bone cyst (Traumatic, Simple, Hemorrhagic)
Aneurismal bone cyst
Radicular Cyst (Apical & Lateral)
Residual Cyst
Para dental Cyst OR Mandibular infected Buccal Cyst

DEVELOPMENTAL

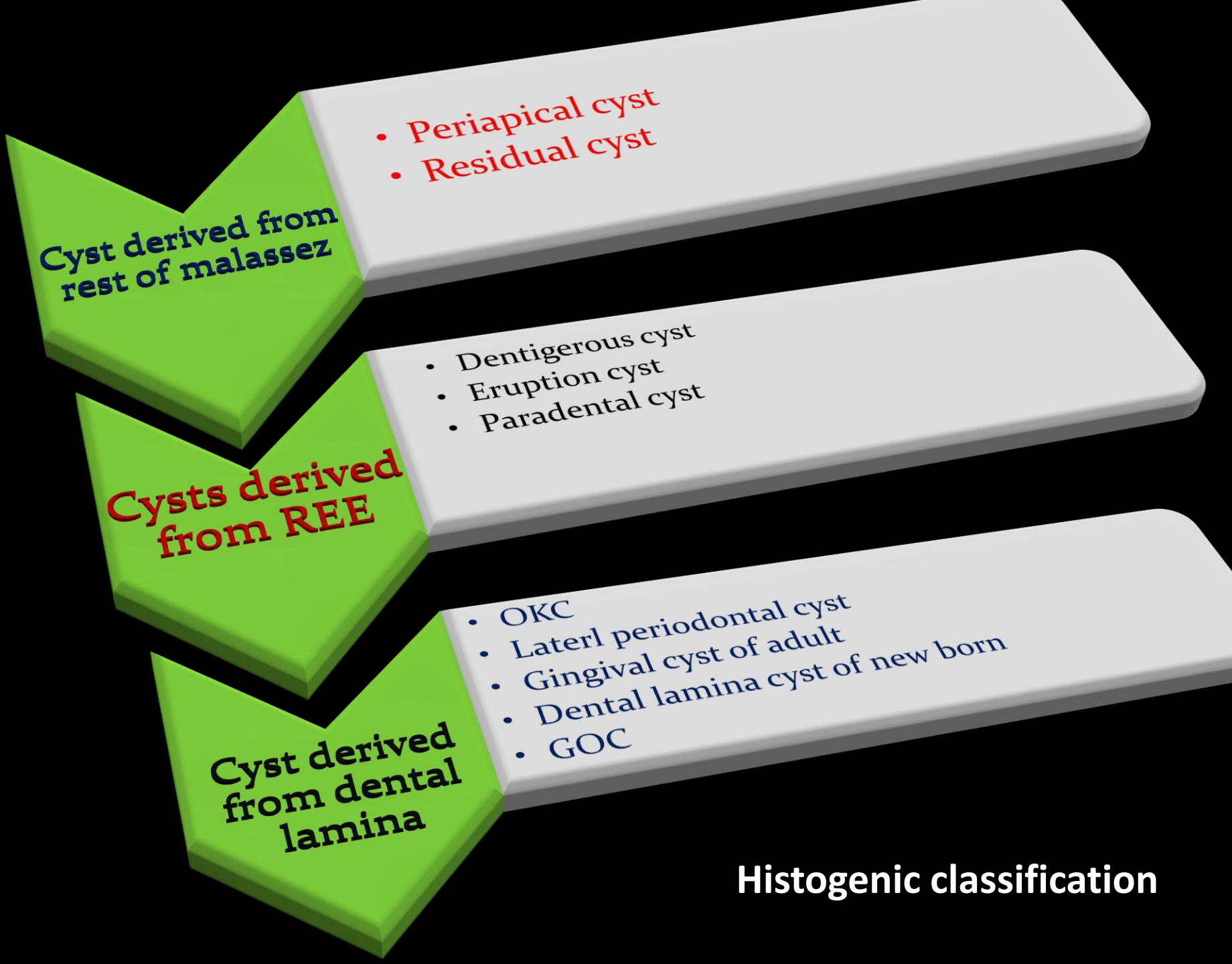
ODONTOGENIC CYST

NON ODONTOGENIC CYST

1. Nasopalatine Duct Cyst
2. Nasolabial Cyst
3. Mid Palatal Raphe Cyst of Infants
4. Median Palatine, Median Alveolar & Median Mandibular Cyst
5. Globulomaxillary Cyst

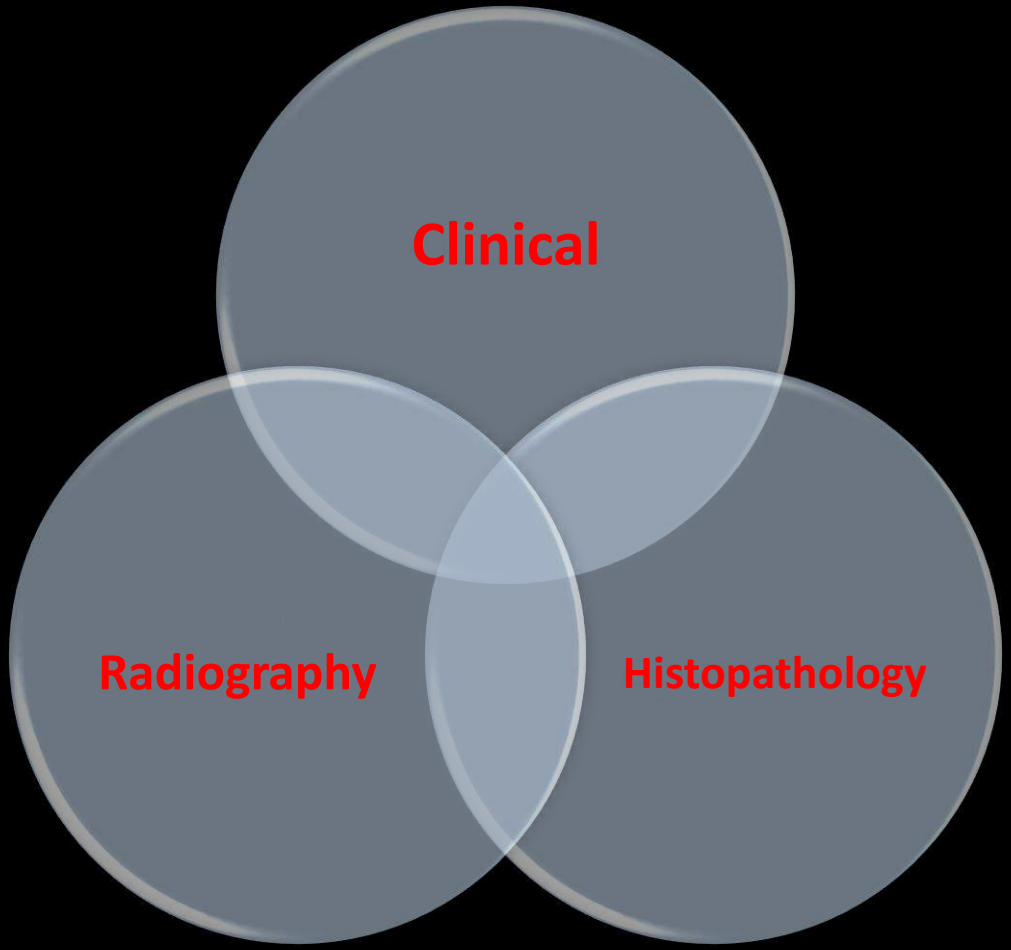
ODONTOGENIC CYSTS
GINGIVAL CYST OF INFANTS
ODONTOGENIC KERATOCYST
DENTIGEROUS CYST
ERUPTION CYST
LATERAL PERIODONTAL CYST
GINGIVAL CYST OF ADULTS
GLANDULAR ODONTOGENIC CYST
CALCIFYING ODONTOGENIC CYST

Rests of malassez	• Remnants of Hertwig's epithelial root sheath that persists in the periodontal ligament after root formation .
Derived from REE	• Residual epithelium that surrounds the crown of the tooth after enamel formation is complete.
Derived from dental lamina	• Islands or strands of epihtelium originates from dental lamina after late bell stage (rests of serrae).



Histogenic classification

Diagnosis



Inflammatory origin

- ✓ **Radicular Cyst**
  (Apical & Lateral)
- ✓ **Residual Cyst**
- ✓ **Para dental Cyst**

Introduction

Etiopathogenesis

- ✓ Source of epithelium
- ✓ Epithelial proliferation and Cyst formation
- ✓ Cyst enlargement

Clinical feature

Radiographic features

Histopathology

Treatment and prognosis

Radicular cyst

- ✓ An odontogenic cyst derived from cell rests of malassez that proliferate in response to inflammation.
- ✓ Periapical cyst.
- ✓ Most common odontogenic cyst
- ✓ Inflammatory in origin

Etiopathogenesis

- ✓Develops at the root apex of an erupted devitalized tooth.
- ✓Trauma or dental caries.
- ✓Epithelial rests of Hertwig’ s epithelial root sheath.

Inflammation

Proliferation of cell rests of malassez

Progressive growth of the epithelial island

Liquifactive necrosis of the inner cells of the island

Central cavity surrounded by variable epithelium

Establishment of osmotic gradient

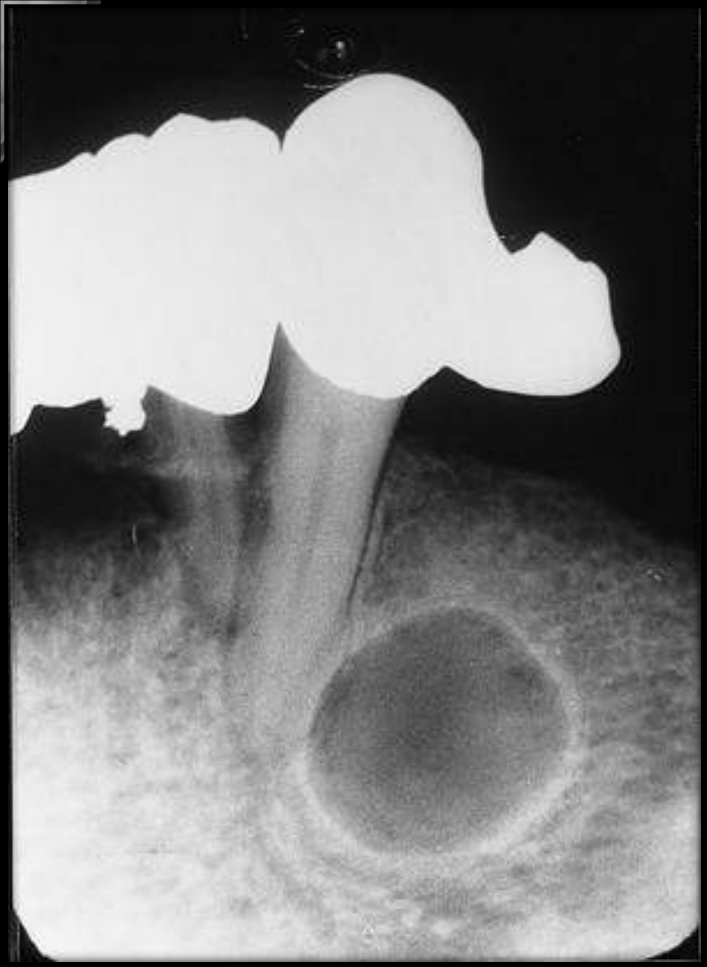
Enlargement cyst

Clinical features

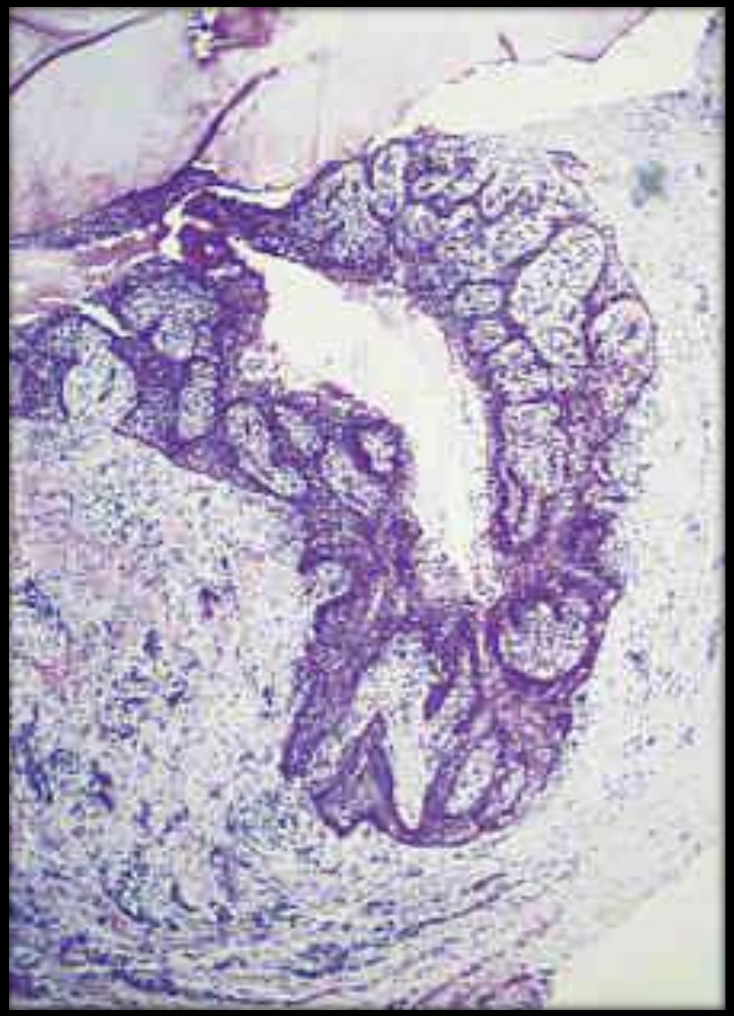
- ✓ Develops at the root apex adjacent to pulpal opening.
- ✓ Periapical or lateral Periapical.
- ✓ Pain, swelling, facial asymmetry.
- ✓ Root resorption, displacement of tooth.
- ✓ Maxilla> mandible.
- ✓ Maxillary anterior, mandibular molar, premolar.

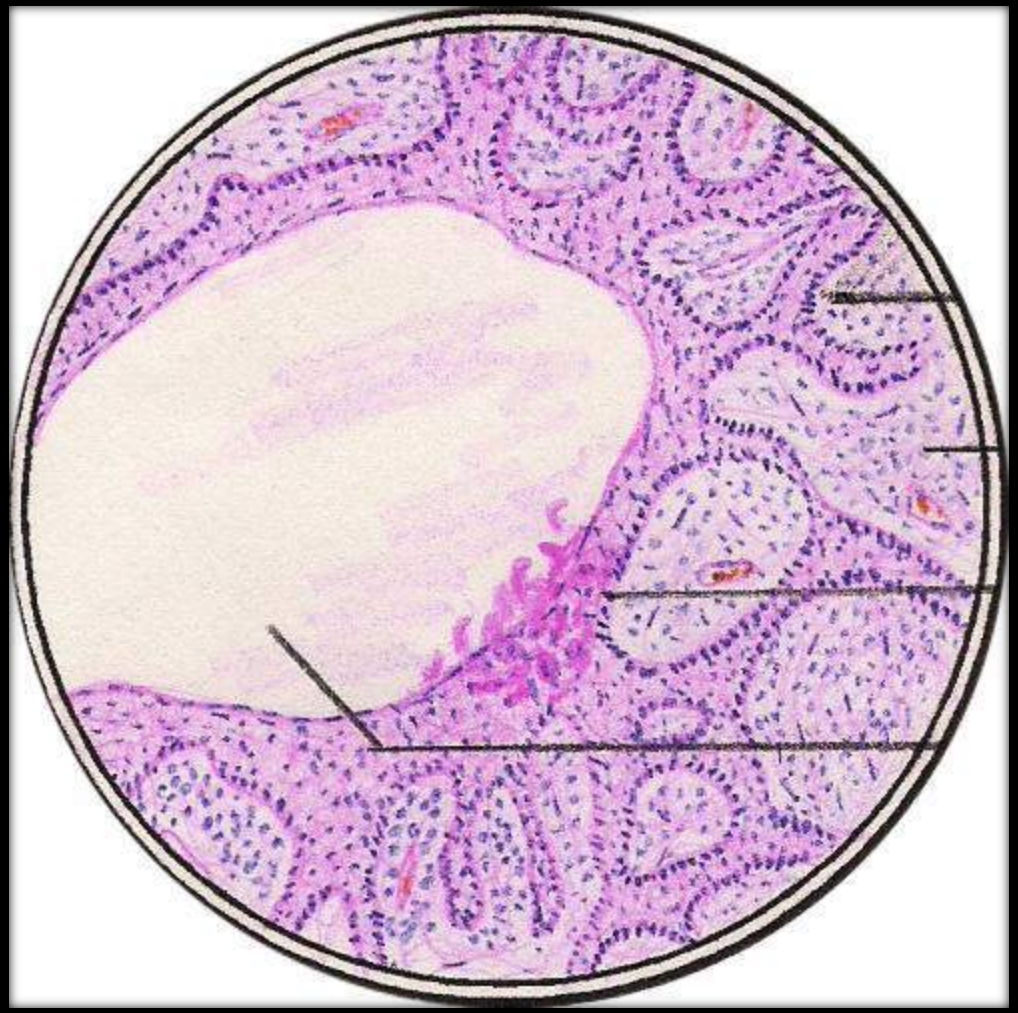
Radiographic features

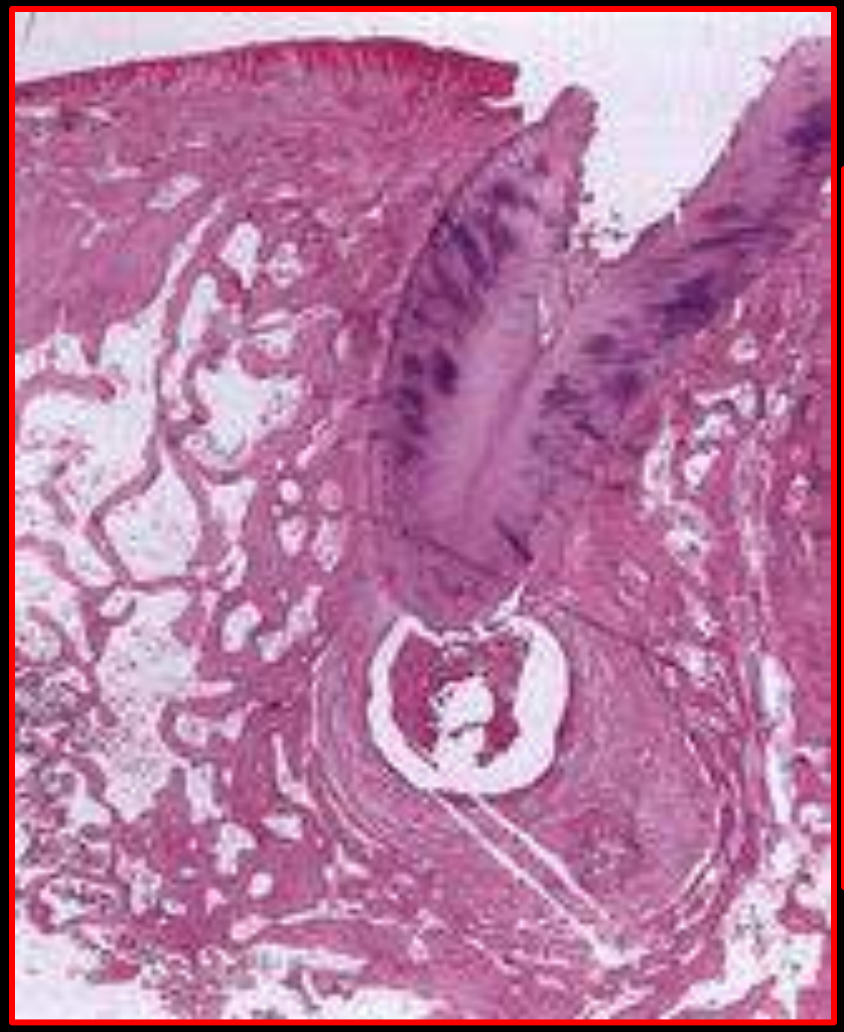
Rounded, well circumscribed, often corticated radiolucency at the apex of a non vital tooth.

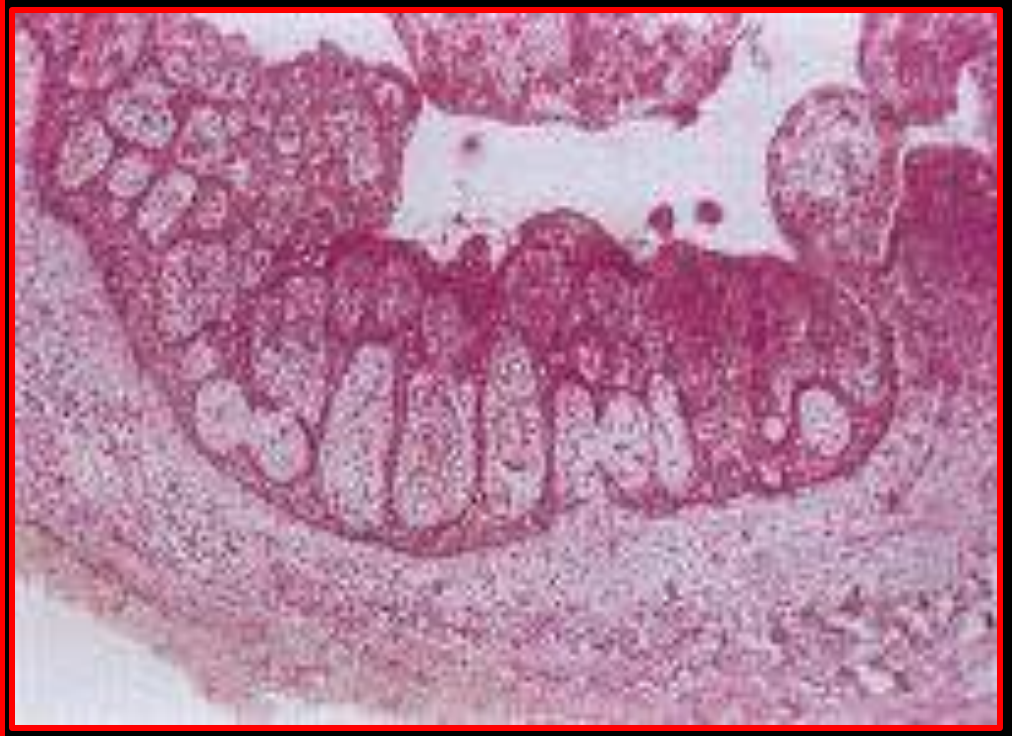


Histopathology



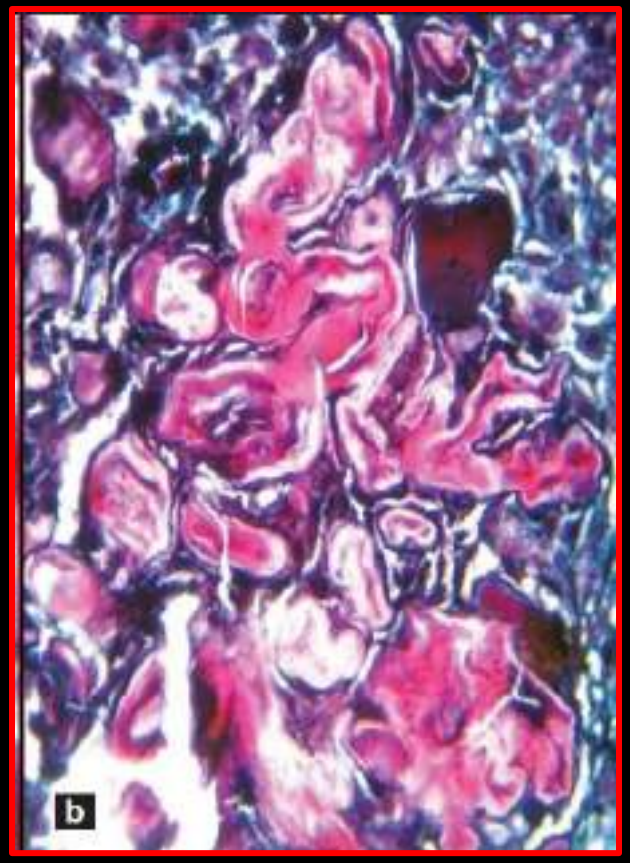


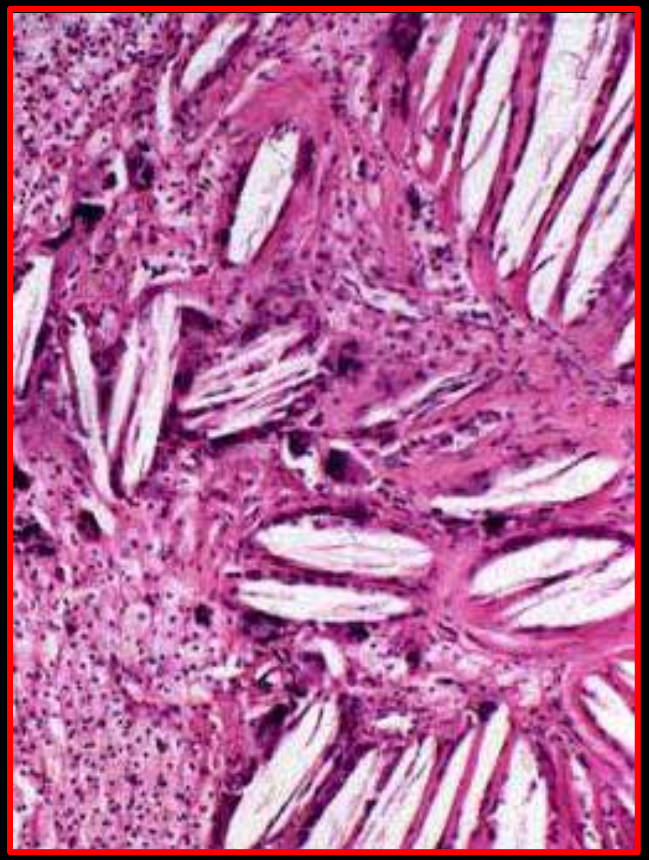


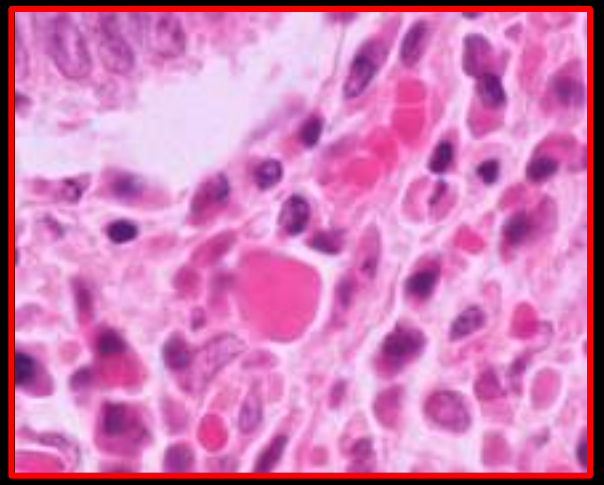


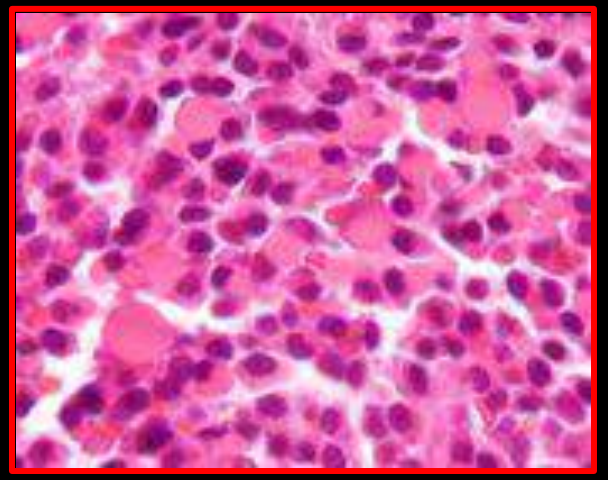
- ✓ Lined by non keratinized stratified squamous epithelium of variable thickness.
- ✓ Proliferation of epithelium in Arcading pattern.
- ✓ Discontinuous at times because of inflammation.
- ✓ Intense inflammation in the connective tissue capsule.
- ✓ Lymphocytes and plasma cells.
- ✓ Extravassated RBC's, numerous blood capillaries.
- ✓ Rushton body(hyaline body), Russell body, cholesterol crystals.

Rushton body, Russel body & cholestrol crystals









Lumen

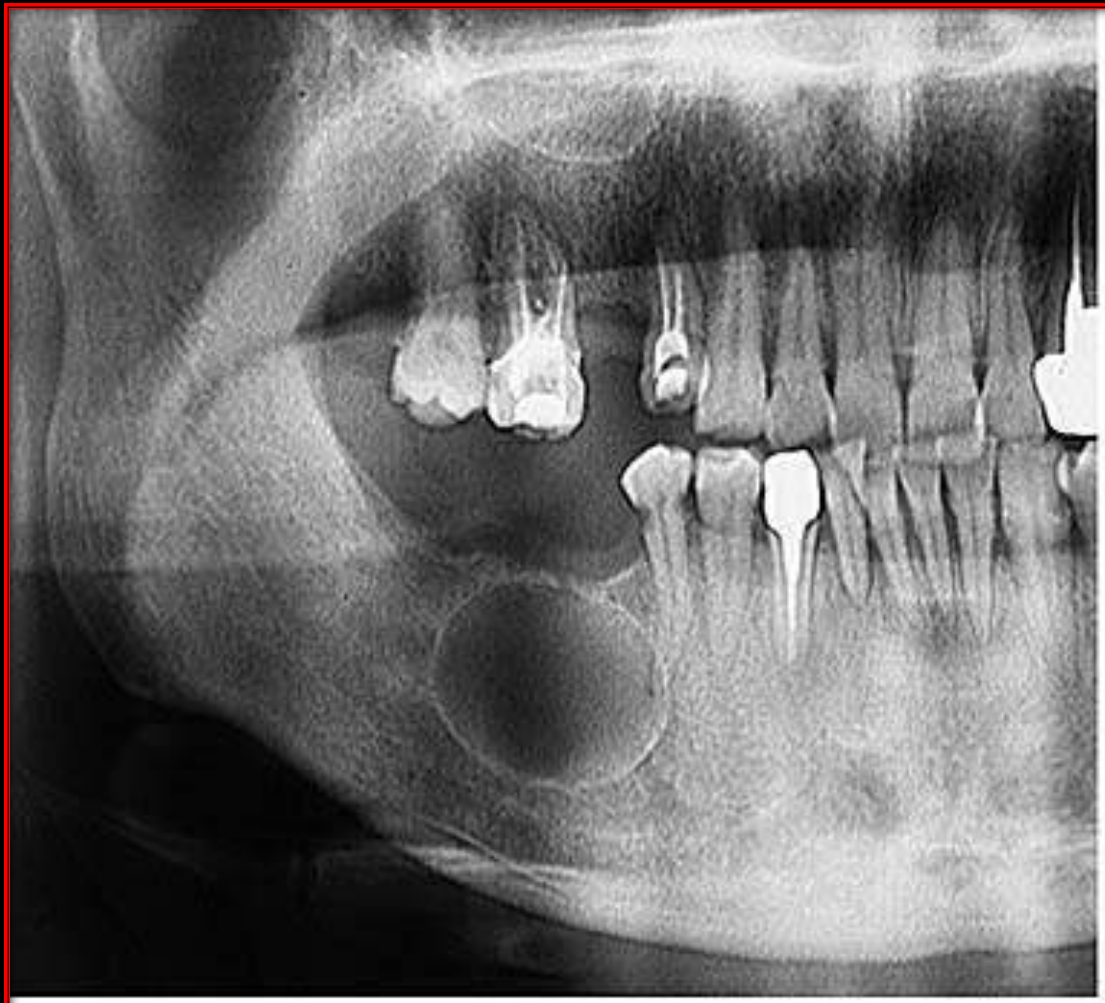
- Blood tinged.
- Higher in protein content.

Treatment

- Enucleation and marsupilization

Residual cyst

- ✓ Represents retained periapical cysts from teeth that have been removed.
- ✓ Develops from the left over granuloma after tooth extraction.
- ✓ Any tooth bearing area.
- ✓ Well defined radiolucent lesions.
- ✓ Few mm to cm.
- ✓ Usually asymptomatic, does not expand the cortical bone.
- ✓ Surgical curettage.



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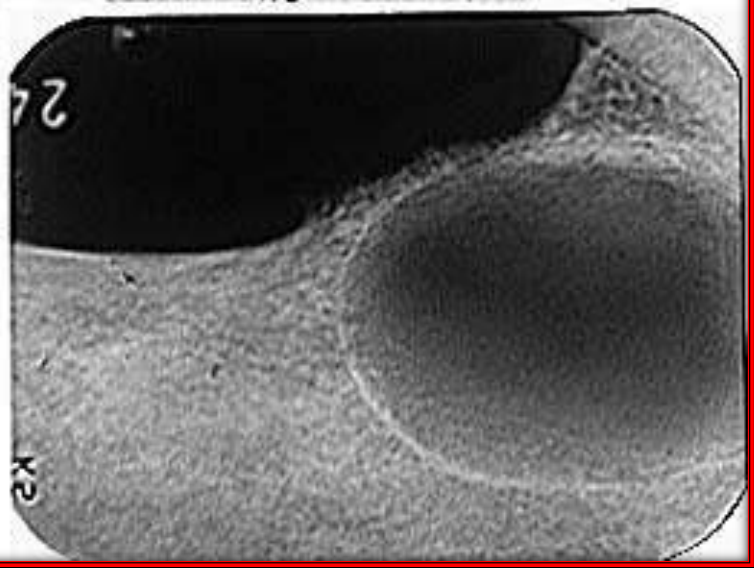

2x

Paradental cyst

‘ A cyst of odontogenic origin, commonly located sub gingivally on the buccal aspect of an erupted mandibular molar or distal surface of a partially erupted mandibular third molar ’

Buccal bifurcation cyst (Craig cyst)

Inflammatory collateral cyst

Etiopathogenesis

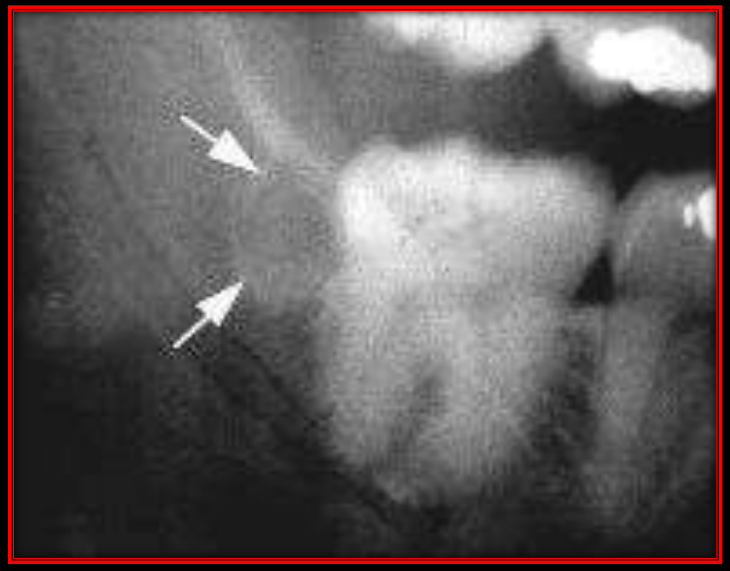
- ✓ Inflammatory origin
- ✓ Reduced enamel epithelium
- ✓ cell rests of malassez ?

Clinical feature

- ✓ Third molar lateral side – distally, buccally or distobuccally.
- ✓ Vital teeth.
- ✓ Asymptomatic.
- ✓ Few mm to cm.

Radiographic feature

- Difficult to visualize in routine radiographs – buccal aspect.
- Well circumscribed radiolucent lesion.



Histopathology

- ✓ Non keratinized, stratified squamous epithelium.
- ✓ slight hyperplasia of the epithelium.
- ✓ Neutrophilic infiltration into the epithelium.
- ✓ Chronic inflammatory infiltrate in the connective tissue.

Treatment

- ✓ Surgical enucleation.

Developmental origin

- ✓ Gingival cyst of infants
- ✓ Gingival cyst of adults
- ✓ OKC
- ✓ Dentigerous cyst
- ✓ Eruption cyst
- ✓ Lateral periodontal cyst
- ✓ Glandular odontogenic cyst
- ✓ Botryoid odontogenic cyst

Gingival cyst of infants

Dental lamina cysts of newborn

'Multiple occasionally solitary superficial raised nodules on edentulous alveolar ridges of infants that resolve without treatment'

- ✓ Derived from dental lamina.
- ✓ Keratin producing epithelial lining.
- ✓ Epstein pearls – keratin filled nodules on the mid palatine raphe.
- ✓ Bohn's nodule – keratin filled cysts scattered over the palate predominantly at the junction of hard and soft palate.

Represents entrapped epithelial remnants at the time of embryonic fusion.

Clinical feature

- ✓ Soft tissue cysts.
- ✓ Alveolar ridges, small often multiple.
- ✓ Asymptomatic.
- ✓ Pre deciduous dentition.

Microscopy

- ✓ Thin epithelial lining.
- ✓ Lacks retepegs.
- ✓ Lumen filled with desquamated keratin.
- ✓ Occasional dystrophic calcification may be seen in connective tissue capsule.

Gingival cyst of the adult

‘A small developmental odontogenic cyst of the gingival soft tissue derived from the rests of the dental lamina, containing a lining of embryonic epithelium of cuboidal cells and distinctive focal thickenings similar to the lateral periodontal cyst’

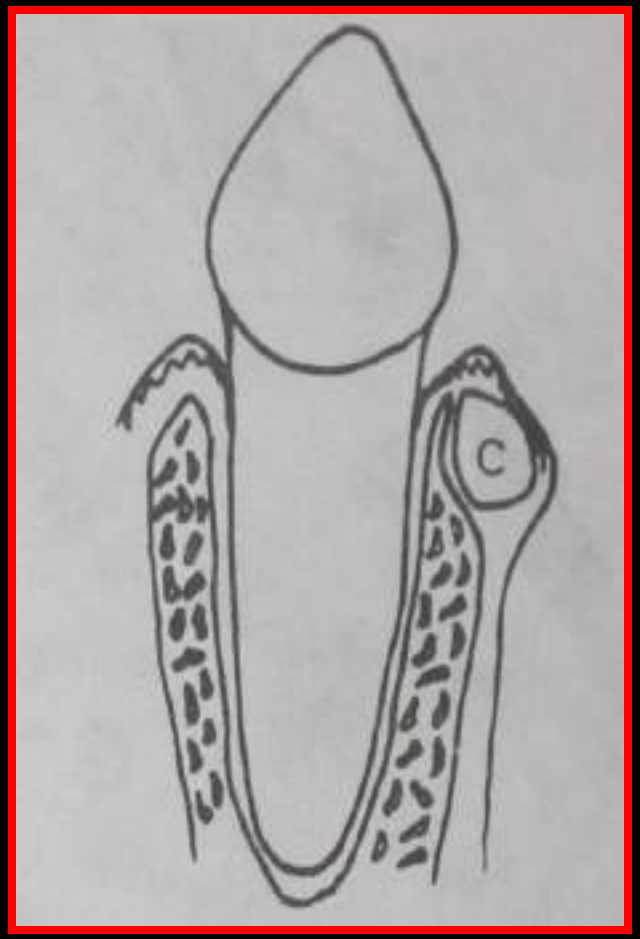
Etiopathogenesis

- Remnants of dental lamina
- Epithelial islands of periodontal membrane
- Traumatic implantation of epithelium

- Cystic transformation of the dental lamina rests.
- Traumatic implantation of surface epithelium – true odontogenic cyst ????
- Soft tissue counter part of lateral periodontal cyst ???

Clinical features

- ✓ 30 – 40 years.
- ✓ Mandibular bicuspid – cuspid incisor area.
- ✓ Small, well circumscribed, painless swelling on the gingiva.
- ✓ Closely resembles Mucocele.
- ✓ Free or attached gingiva.


C

Radiographic features

- ✓ Soft tissue cyst does not manifests radiographic details.
- ✓ Superficial erosion of the cortical plate.

Microscopy

- ✓ Flattened to several layer thickened epithelium.
- ✓ Presence of epithelial plaques.
- ✓ Glycogen rich clear cells.
- ✓ Fibrous capsule.
- ✓ Presence of dental lamina rests in the capsule.

Dentigerous cyst

‘ An odontogenic cyst that surrounds the crown of an impacted tooth; it is caused by the accumulation of fluid between REE and enamel surface ,resulting in a cyst in which the crown is located within the lumen and the root outside’.

Follicular cyst

Etiopathogenesis

Reduced enamel epithelium

Tooth covered by follicle lined by REE.

Failure to erupt (IMPACTED).

Transfer of fluid from surrounding connective tissue into the follicle through osmosis.

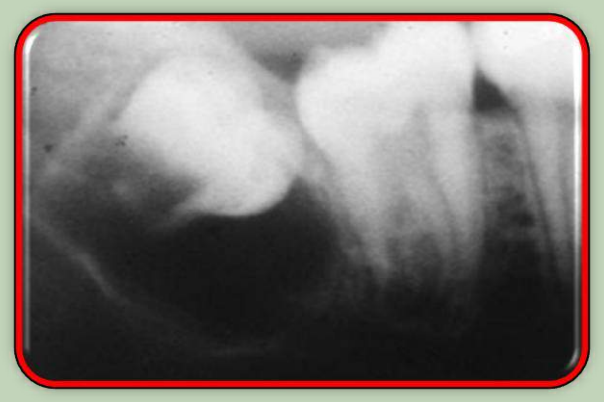
Formation of cyst.

Clinical feature

- ✓ Impacted, embedded or unerupted tooth.
- ✓ May be associated with odontome.
- ✓ 2nd to 3rd decade.
- ✓ Slight male prediction.
- ✓ Solitary, mandibular molar, max cuspid.
- ✓ Bilateral or multiple – cleidocranial dysplasia, and maroteax-lamy syndrome.

- ✓ Expansion of the bone – facial asymmetry.
- ✓ Displacement of the teeth, root resorption and pain.
- ✓ Cyst involving impacted third molar in the ramus area -- *Hollowing out* of the entire ramus.
- ✓ Potentially capable of becoming an aggressive lesion.
- ✓ Ameloblastoma, Squamous Cell Carcinoma, Muco epidermoid Carcinoma, Adenomatoid Odontogenic Tumour.

Radiographic features



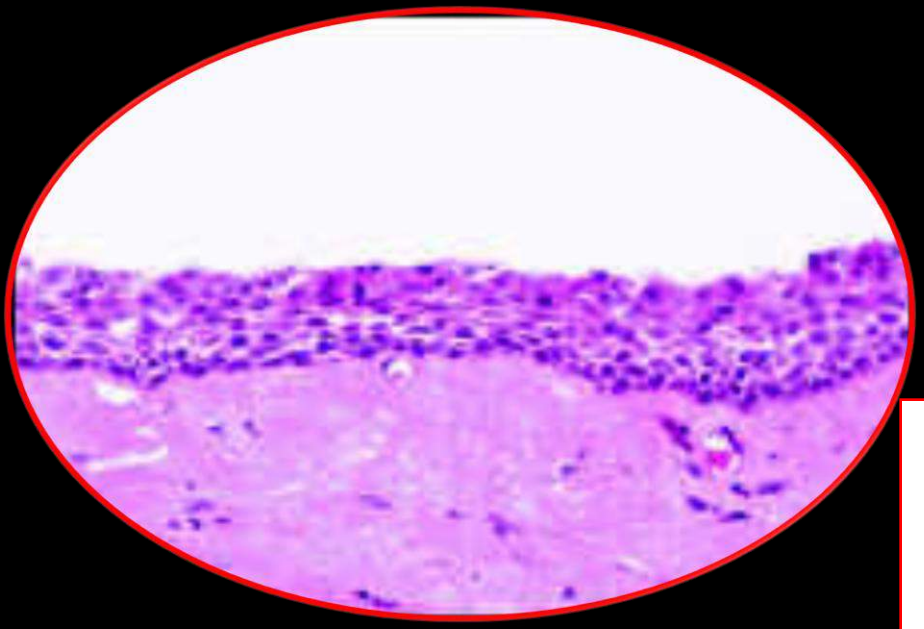
Central

Circumferential

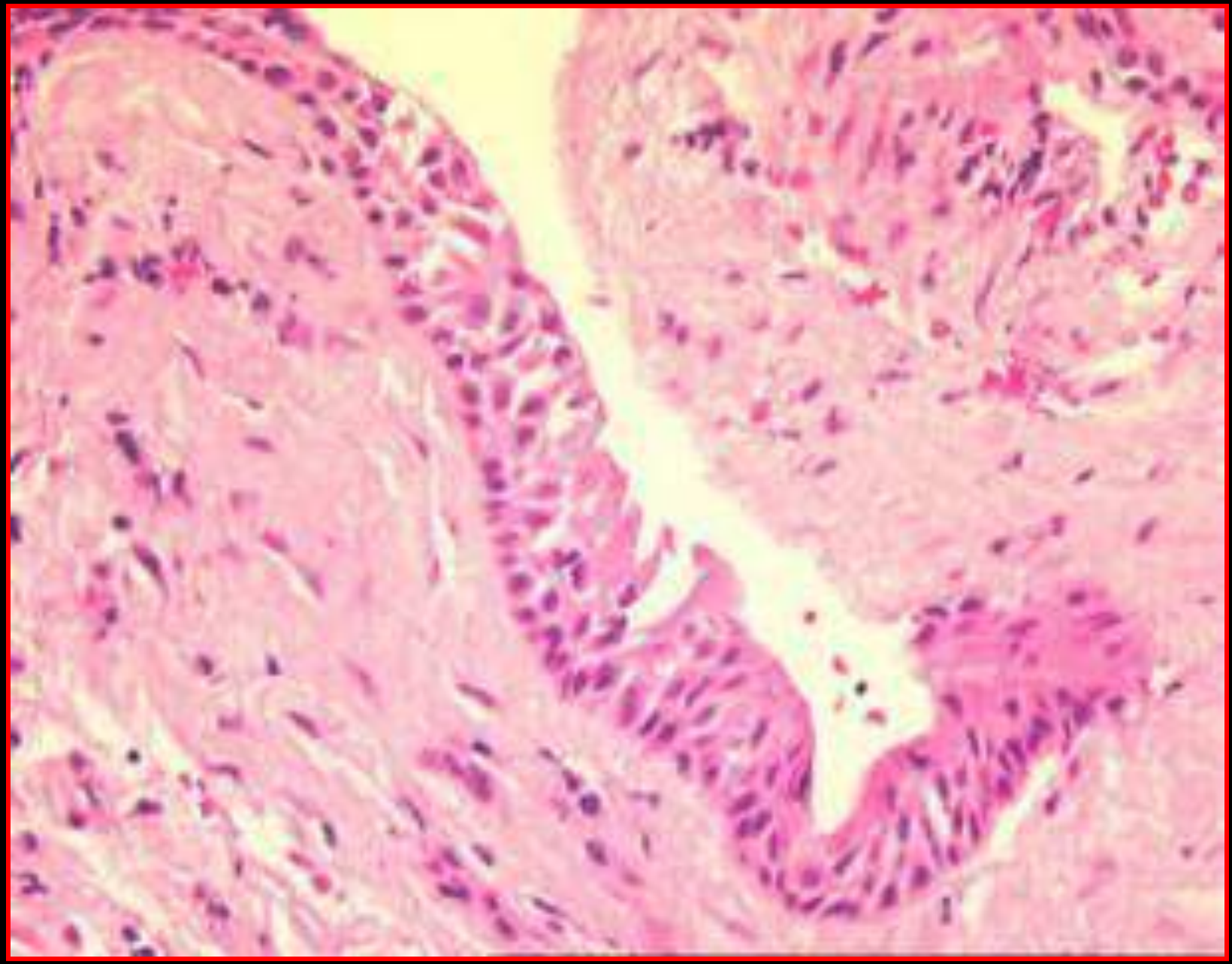
lateral

Histopathology

- ✓ Thin layer of non keratinized stratified squamous epithelium.
- ✓ Cubical- 2-4 layer thick.
- ✓ Retepeg formation generally absent unless infected.
- ✓ Mucous metaplasia of epithelial cells.
- ✓ Thin connective tissue wall.
- FOLLICULAR KERATOCYST



- ✓ Connective tissue capsule is composed of very loose fibrous OR Myxomatous tissue.
- ✓ Varying numbers of islands of odontogenic epithelium in the capsule.
- ✓ Presence of Rushton bodies in the epithelium



LUMEN

- ✓ Thin watery yellow, occasionally blood tinged.

Eruption cyst

‘An odontogenic cyst with the histologic features of a dentigerous cyst that surrounds a tooth’s crown that has erupted through the bone but not soft tissue and is clinically visible as a soft fluctuant mass on the alveolar ridges’

- ✓ Eruption hematoma
- ✓ Reduced enamel epithelium

Clinical features

- ✓ Dentigerous cyst occurring in soft tissue.
- ✓ Tooth is impacted in soft tissue (gingiva) not inside the bone.
- ✓ Small fluctuant swelling on the alveolar ridges.
- ✓ Common in children.
- ✓ Circum oral cystic cavity contains blood swelling appears purple – *Eruption hematoma.*

Microscopy

- ✓ Superficial aspect – stratified squamous epithelium of the gingiva.
- ✓ Thin fibrous connective tissue separates this from the cystic lining.
- ✓ Lining epithelium similar to dentigerous cyst lining.
- ✓ Capsule composed of loosely arranged fibers and capillaries.

Odontogenic keratocyst

OKC/ KCOT

‘ A cyst derived from the rests of dental lamina, with a biologic behavior similar to benign neoplasm, with a distinctive lining of six to ten cells in thickness, and that exhibits a basal layer of palisaded cells and a surface of corrugated keratin ’

Primordial cyst

keratocystic odontogenic tumour

- ✓ Most interesting odontogenic cyst.
- ✓ Different clinical behavior.
- ✓ Typical histopathology.
- ✓ Associated with nevoid basal cell carcinoma syndrome.
- ✓ Potential to behave aggressively , that can recur – benign cystic neoplasm – keratocystic odontogenic tumour.

Etiopathogenesis

- ✓ Two main sources of epithelium

  Dental lamina or its remnants

  Extensions of basal cells from the overlying oral epithelium.

- ✓ Genetic tendency

- ✓ Associated with syndrome

- ✓ Degeneration of stellate reticulum of the enamel organ before the formation of tooth due to environmental and genetic causes

- ✓ Degeneration of stellate reticulum of the enamel organ before the formation of tooth due to environmental and genetic causes.
- ✓ Epithelium proliferate along the cancellous component of the bone.
- ✓ Role of osmolality of cyst fluid.
- ✓ Multi-centric hydrostatic expansion of cyst
- ✓ Epithelial cells producing keratin - semisolid cyst content

Enlargement of CYST

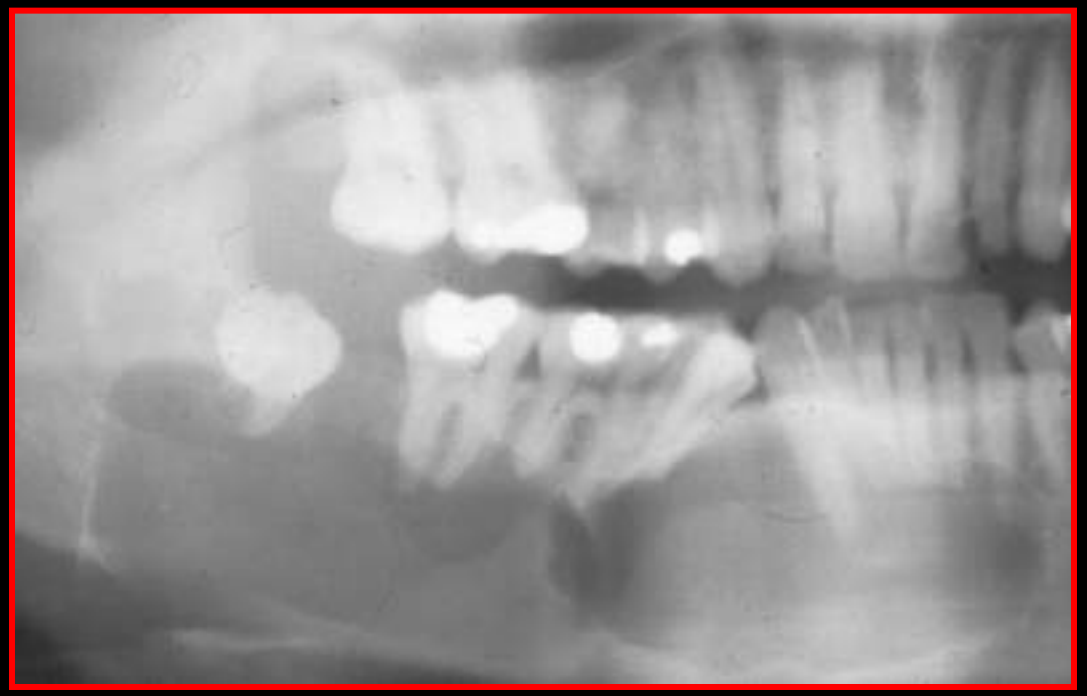
Clinical features

- Bimodal age distribution 2^{nd} -3^{rd} , 5^{th} decade.
- ✓ Males > females; Mandible > Maxilla
- ✓ Body and ascending ramus of the mandible extensively, with little or no bony expansion.
- ✓ OKCs tend to extend along the cancellous component of the bone – less or no expansion of the cortical plates.
- ✓ It frequently reaches a large size, particularly at the angle of mandible and ascending ramus, before they are diagnosed.

- ✓ Displacement of the teeth.
- ✓ Perforation of bone .
- ✓ Multiple OKC- 7%
- ✓ Naevoid basal cell carcinoma Syndrome (Gorlin - Goltz syndrome.

Radiographic features

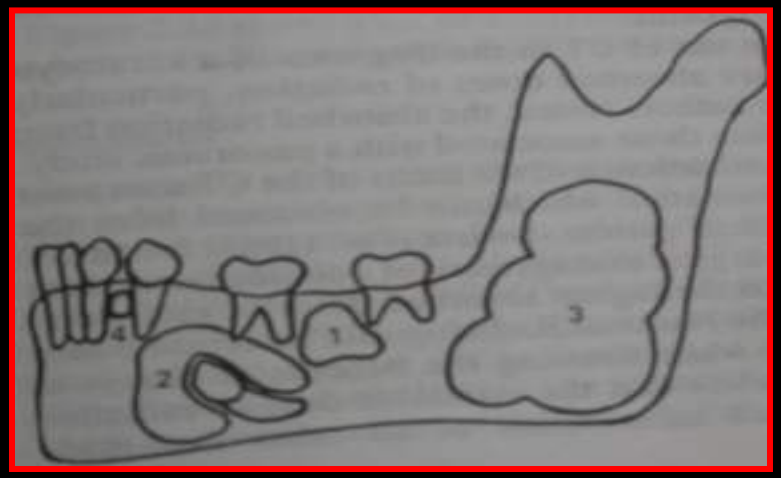
- ✓ Small, round or ovoid radiolucent areas.
- ✓ Most of them are well - demarcated with a distinct sclerotic margin.
- ✓ Some of the uni-locular lesions have scalloped margins and these may be misinterpreted as multi-locular lesions.



R

Radiographic variants of OKCs

Envolopmental (2)	Extraneous (3)	Collateral (4)	Replacemental (1)
Keratocyst embracing an adjacent un erupted tooth.	In ascending ramus away from the teeth	Adjacent to the roots of teeth, usually in the mand. P.M region, which are indistinguishable radiologically from lateral periodontal cyst.	Replaces a tooth germ and present at the site of missing tooth



Histopathology

Two variants

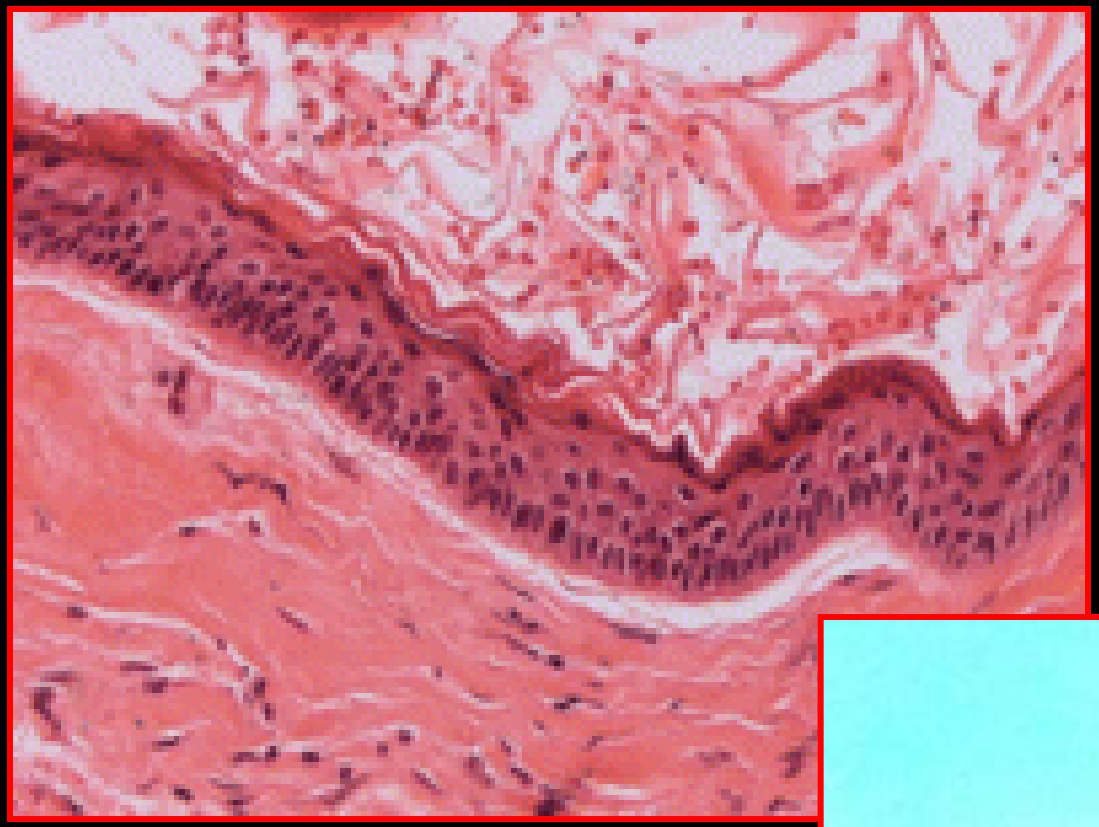
Orthokeratinized

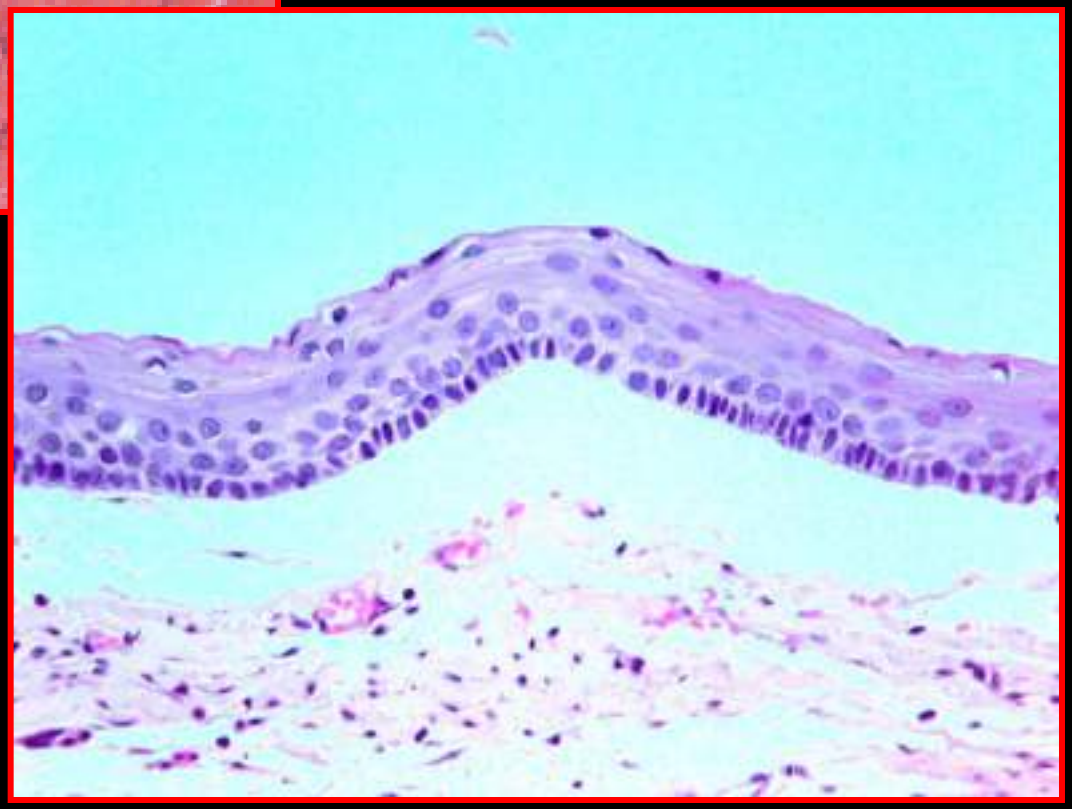
Para keratinized

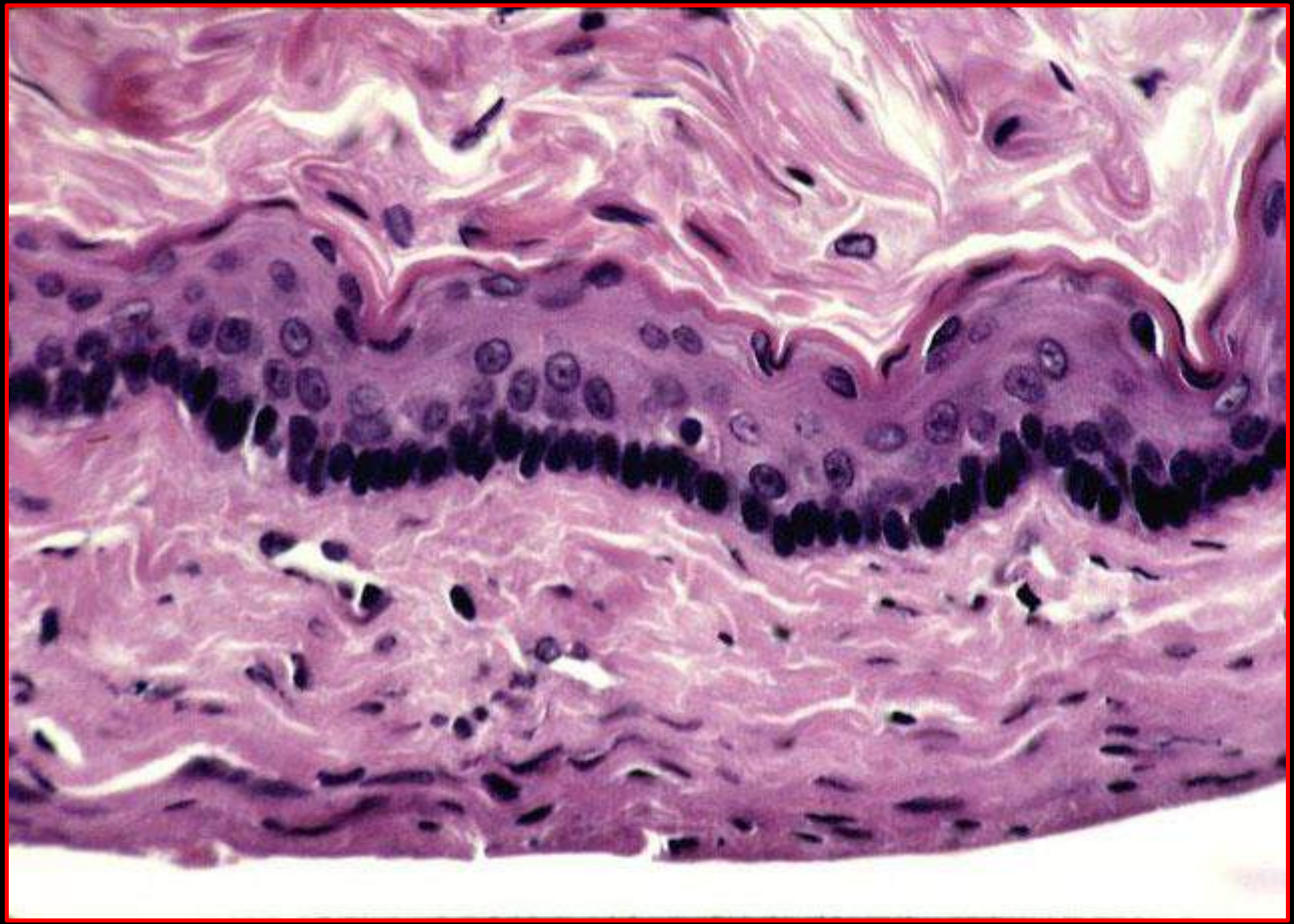
GROSS/ MACROSCOPY

- The linings of the keratocysts are rarely received intact in the laboratory.
- They are usually thin walled, collapsed and folded.

- Lined by a regular keratinised stratified squamous epithelium which is usually about 6-10 cell layer thick and without rete ridges.
- The from of keratinization is exclusively parakeratotic in about 80-90% of cases, but is sometimes orthokeratotic (10%).
- Surface corrugation.
- Palisaded and polarized nucleus in the basal layer cells.
- Tombstone appearance of basal layer cells.







- The fibrous capsule of the keratocyst is usually thin with relatively few cells widely separated by a stroma.

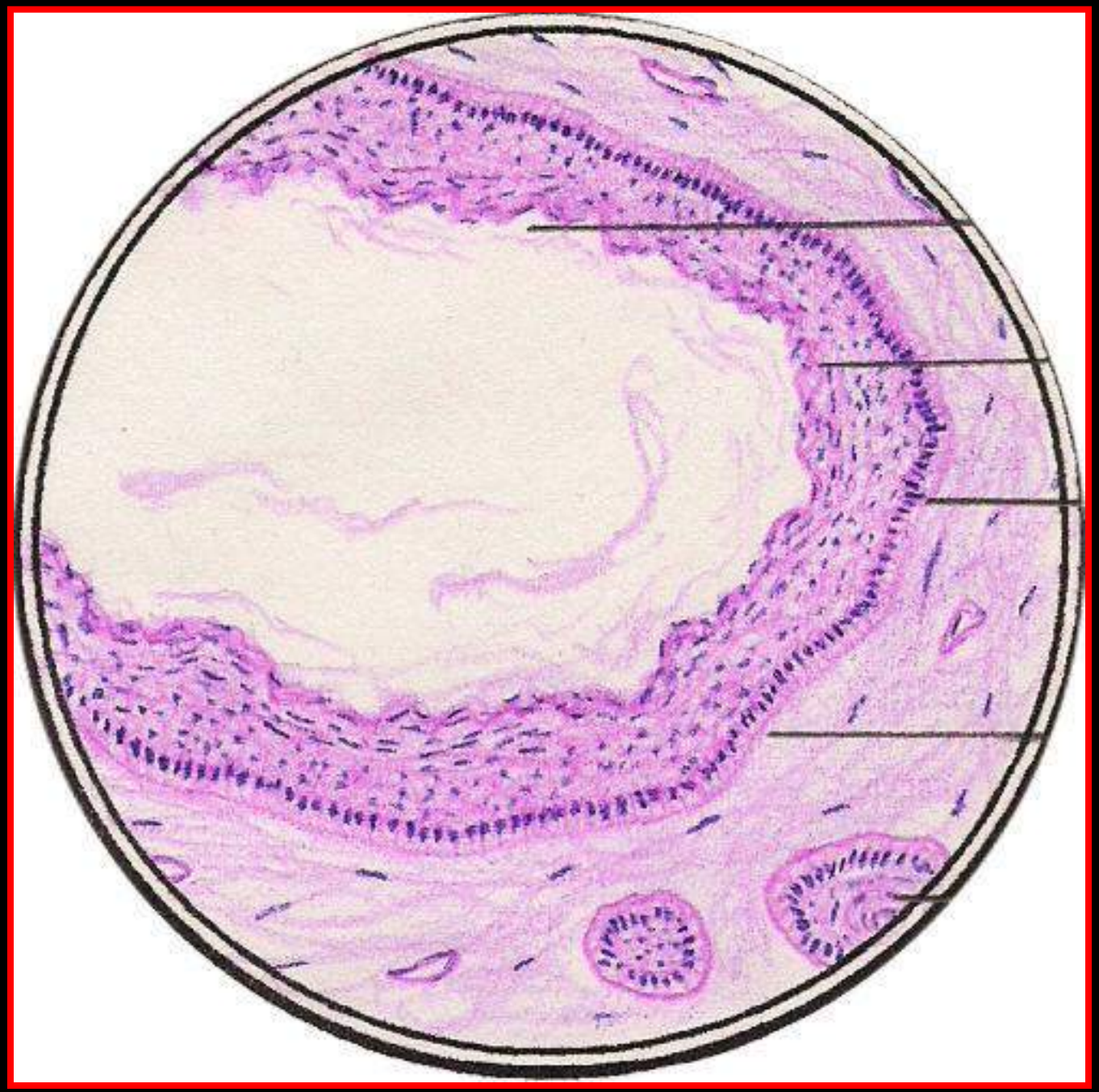
- Inflammatory cells are very infrequent but there may be a mild infiltration of lymphocytes and monocytes.

- In the presence of an intense inflammatory process, the adjacent epithelium looses its keratinised surface, may thicken & develop rete processes or may ulcerate.

- Presence of daughter cysts within the connective tissue.

Lumen

- Straw colored fluid
- Keratin filled areas.
- Protein level of less than 4.0 gm / 100 ml indicate a diagnosis of keratocyst.



Treatment

- ✓ Marsupialization & Enucleation. – more recurrences.
- ✓ Resection part of the jaw with overlying epithelium.
- ✓ Attempts have been made to devitalize the cells of the cyst lining before enucleation by instilling Carnoy's fluid.
- ✓ Regular follow up.

Recurrence

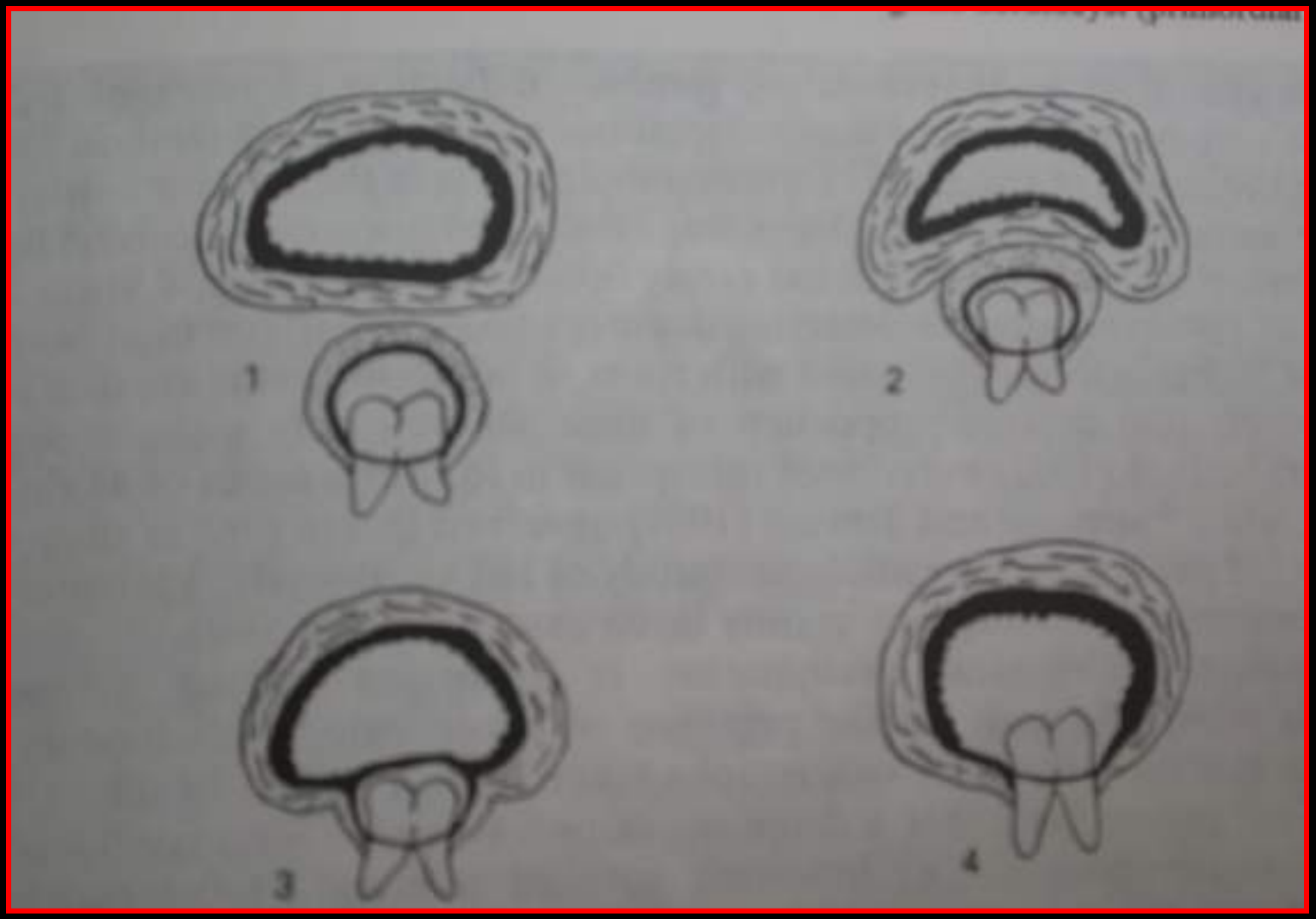
- ✓ High recurrence rate.
- ✓ The size of the cyst does not influence the recurrence rate.
- ✓ Recurrences were more frequent with cysts in patients with the naevoid basal cell carcinoma syndrome.
- ✓ Keratocysts enucleated in one piece has less recurrence potential.
- ✓ Those cysts whose radiographic appearance was multilocular had a higher recurrence rate than those with a unilocular appearance

?????

- ✓ Daughter or satellite cysts.
- ✓ Thin and fragile lining – difficult to remove in single piece.
- ✓ Proliferation of epithelium into the cancellous components of bone.
- ✓ Improper surgical removal.
- ✓ Neoplastic and aggressive behavior.

Follicular keratocyst

- ✓ Tooth erupting into the existing odontogenic keratocyst.
- ✓ Radio graphically resembles dentigerous cyst.
- ✓ Histologically shows features of both OKC & dentigerous cyst.
- ✓ Area of cyst attached to the tooth – dentigerous cyst.
- ✓ Rest of the area – OKC.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Nevoid basal cell carcinoma syndrome.

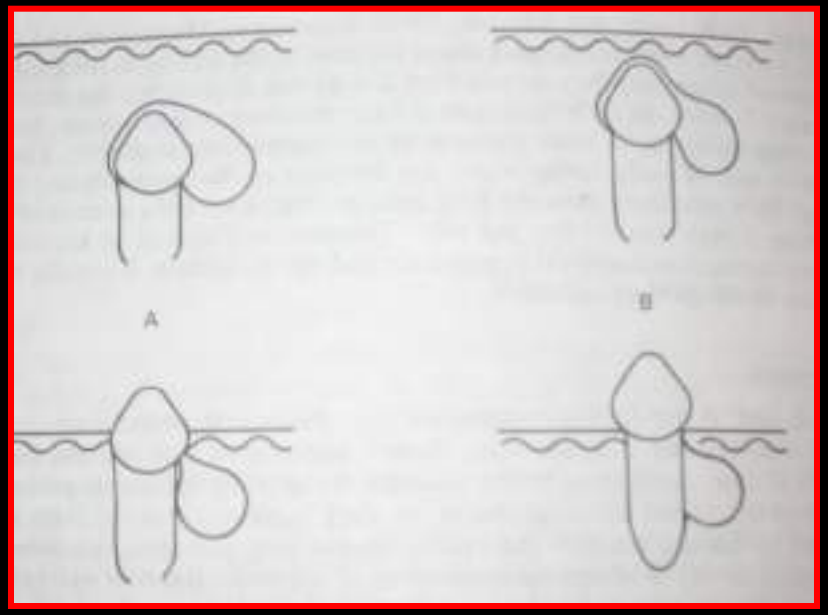
- ✓ Gorlin- Goltz syndrome.
- ✓ Jaw cyst – Basal cell nevus, bifid rib syndrome

Cutaneous	• Basal Cell Carcinoma, Benign Dermal Cysts, Palmer Pitting, Palmer Planter Keratosis
Dental and osseous	• Multiple OKCs Mandibular Prognathism, Bifid Rib, Veritable Abnormality
Ophthalmologic	• Hypertelorism With Wide Nasal Bridge
Sexual	• Hypogonadism & Ovarian Tumours
Neurologic	• Mental Retardation, Dural Calcification

Lateral periodontal cyst

‘A slow growing, non expansile, developmental odontogenic cyst derived from rests of dental lamina, containing an embryonic lining of one to three cells and distinctive focal thickenings(plaques)’

- ✓ Developmental in origin.
- ✓ Cystic degeneration of clear cells of dental lamina
- ✓ Initially as a dentigerous cyst developing along the lateral surface of the crown, later somehow tooth erupts living behind the cyst in the periodontal space.



Clinical features

- ✓ 50 years
- ✓ M>F
- ✓ Mandibular bicuspid/cuspid/incisor.
- ✓ Asymptomatic
- ✓ Discovered during routine radiogrphy.

Radiographic features

- ✓ Small well defined, delicately corticated, unilocular radiolucency located between the roots.
- ✓ Multilocular variant – Botryoid odontogenic cyst.

Microscopy

- Thin lining of non keratinized epithelium.
- 1-3 cell thickness.
- Focal epithelial thickenings proliferating in to the lumen.
- Clear, vacuolated glycogen rich cells.

Glandular odontogenic cyst

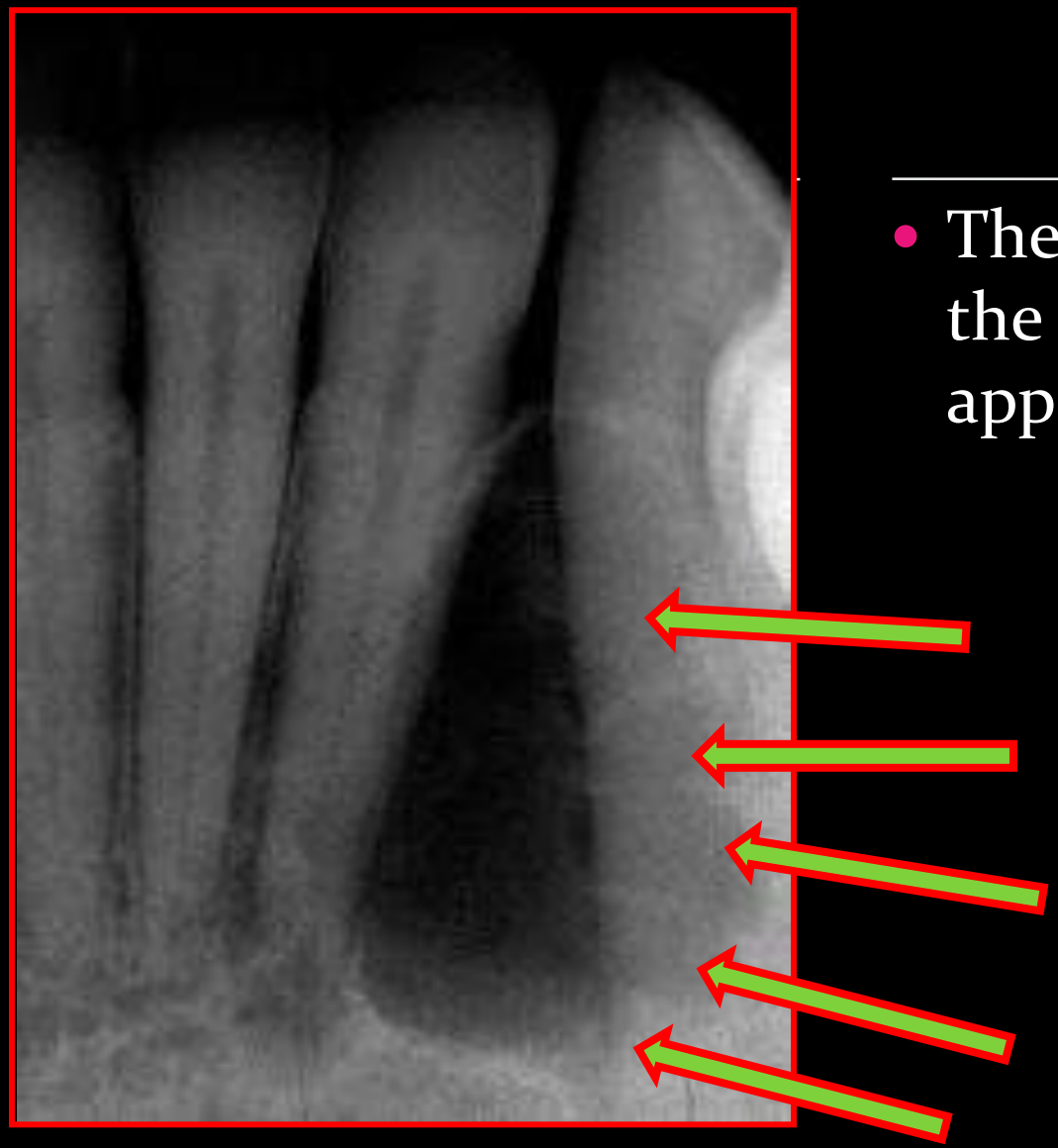
- “A cyst arising in the tooth-bearing areas of the jaws characterised by an epithelial lining with cuboidal or columnar cells both at the surface and lining crypts or cyst-like spaces within the thickness of the epithelium“

- Sialo-odontogenic cyst

- REE

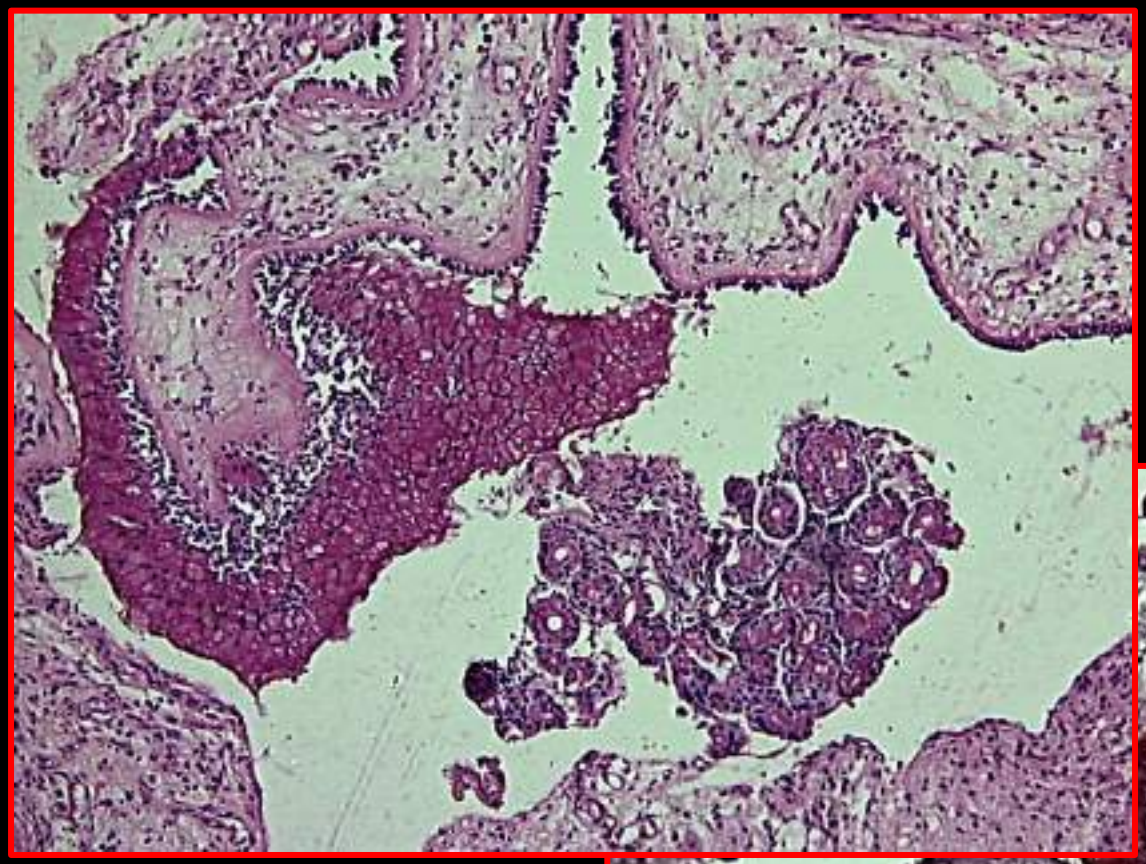
- ✓ Usually presents as a painless, slow-growing swelling.
- ✓ Anterior part of the jaws.
- ✓ 4^{th} and 5^{th} decades of life.
- ✓ Expansion of jaws with or without pain or paraesthesia.
- ✓ The aggressive nature of the lesion has been reported as 25% to 55% cases recur following curettage.

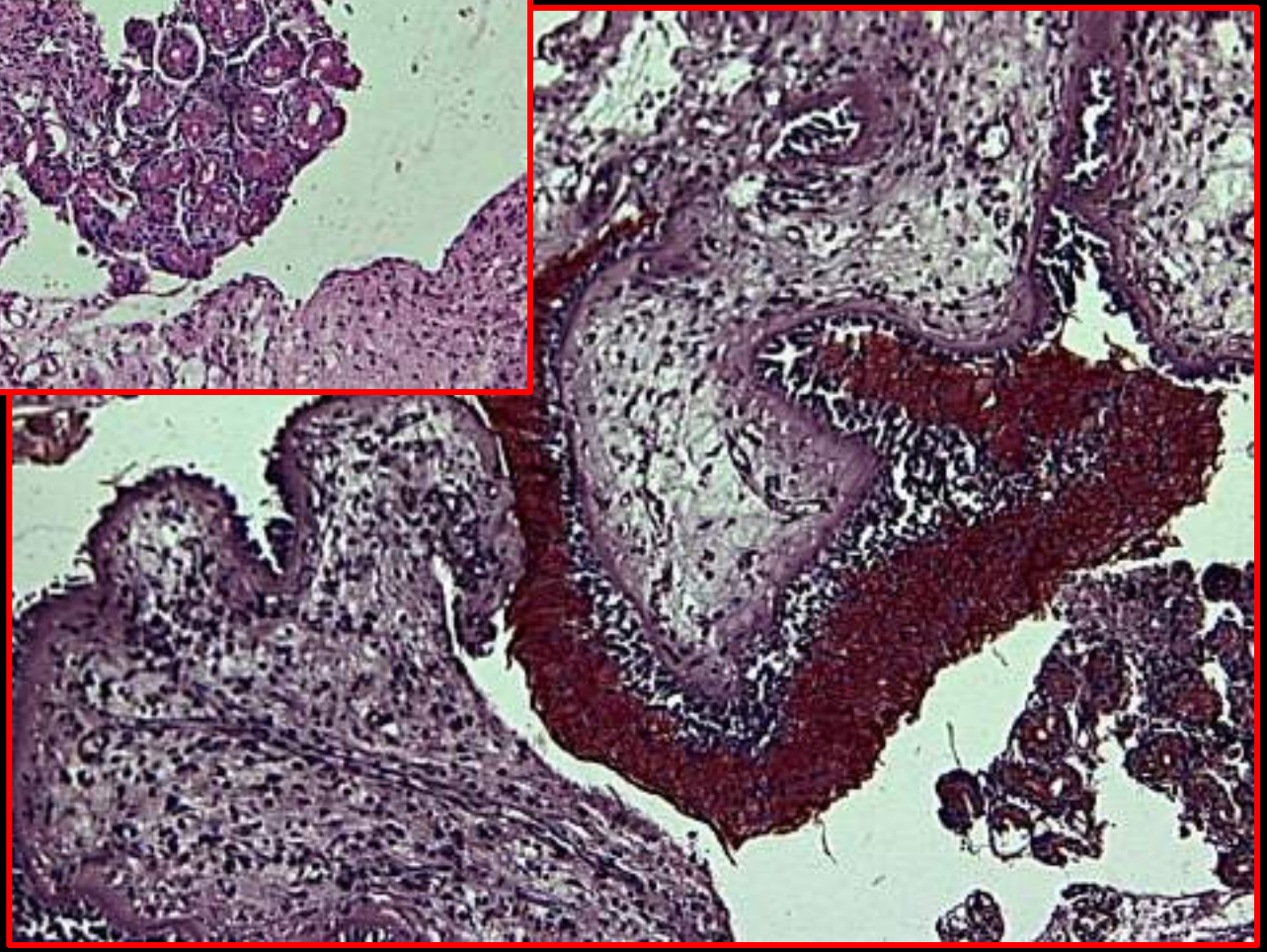
Radiographic features



- The radiograph shows the typical multi-locular appearance.

- ✓ Non-keratinized stratified squamous epithelial cystic lining .
- ✓ Cystic lining was of variable thickness, exhibiting focal plaques of epithelium with a flat connective tissue interface.
- ✓ The superficial layer of the cystic lining consisted of columnar cells, goblet cells and few ciliated cells that formed papillary projections.
- ✓ Within the thickness of the epithelium, areas exhibiting intra-epithelial gland like structures .
- ✓ Connective tissue reveals the formation of daughter cysts.





Calcifying Epithelial Odontogenic Cyst

CEOC/CCOT

- Gorlin cyst.
- Cystic variant two types.

- **Cystic component *** Neoplastic component
  - Type Ia,
  - Type IIb,
  - Type IIIc.
- CEOC--- Central[intra bony]
- Peripheral variant [gingiva]

CLINICAL FEATURES

-SWELLING [HARD -BONY EXPANSION].
-RARELY PAIN.
-MAY BE ASYMPTOMATIC

[DISCOVERED ON ROUTINE RADIOGRAPH]

TYPE 1A-----SIMPLE UNICYSTIC TYPE.
TYPE 1B-----THE ODONTOME PRODUCING TYPE.
TYPE 1C----AMELOBLASTOMATOUS PROLIFERATING TYPE

TYPE 1A----**EPITHELIAL LINING IS LOW CUBOIDAL OR SQUAMOUS.**

2-3 layer **thick** ----**Any age**

"GHOST CELLS" - DENTINOID

[Ameloblastic fibroodontoma, odontomas, cranio pharyngioma, ameloblastoma]

PALE, EOSINOPHILIC, SWOLLEN EPITHELIALCELLS THAT HAVE LOST THE NUCLEUS BUT SHOW A FAINT OUTLINE OF CELLULAR & NUCLEAR MEMBRANE

- Age-2nd Decade [any age]
- Sex-equal distribution
- Race- no racial predilection
- Mand = max
- Common site-anterior part of jaw
- May cross the midline

- **TYPE II**—[Dentinogenic ghost cell tumour]
- **Neoplasm like lesion**
- **Intra ossious usually occurs later inlife**
- **--Extra ossious**
- **Ameloblastoma like strands & islands of odontogenic epitelium infiltrating connective tissue.**
- **+Ghost cells, + dentinoid material**

Radiological features ;

unilocular radiolucent area with regular outline
or poorly defined margins

****Mulicystic

+ Irregular calcified bodies appears as opacities of various sizes

--Displacement of teeth
--Resorption of root of adjacent teeth
--Perforatoin of cortcal plate may occur

HISTOLOGICAL FEATURES:

- **Prominent basal cell layer & palisaded or cuboidal cells and hyperchromatic nuclei, polarised away from basement membrane**

6 to8 layer thick.

- **Ghost cells are seen in epithelium. Ghost cells are degenerating**

Epithelial cells.

- **They may represent the product of coagulative necrosis of odontogenic epithelium**

- **Exhibits yellow flourescence with rhodamine b Stain.**

Calcifications may occur in ghost cells.

•Ghost cells may be in close contact with
Ct and evoke a foreign body reaction of
Multi nucleated giant cells.

•Ct capsule may contain odontogenic epithelium
Satellite cysts may be present.

• Dentinoid may
Also be present.

Sometimes complex odontomas are seen in the wall of ceoc.

Rx; enucleation, or excision.

recap

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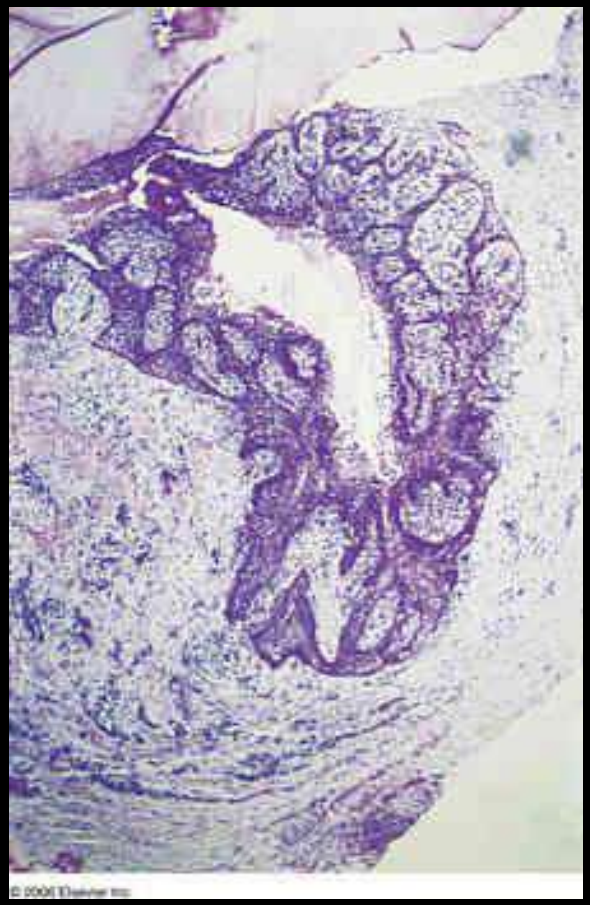
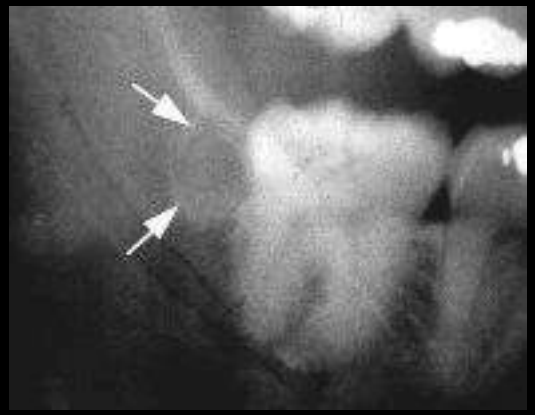
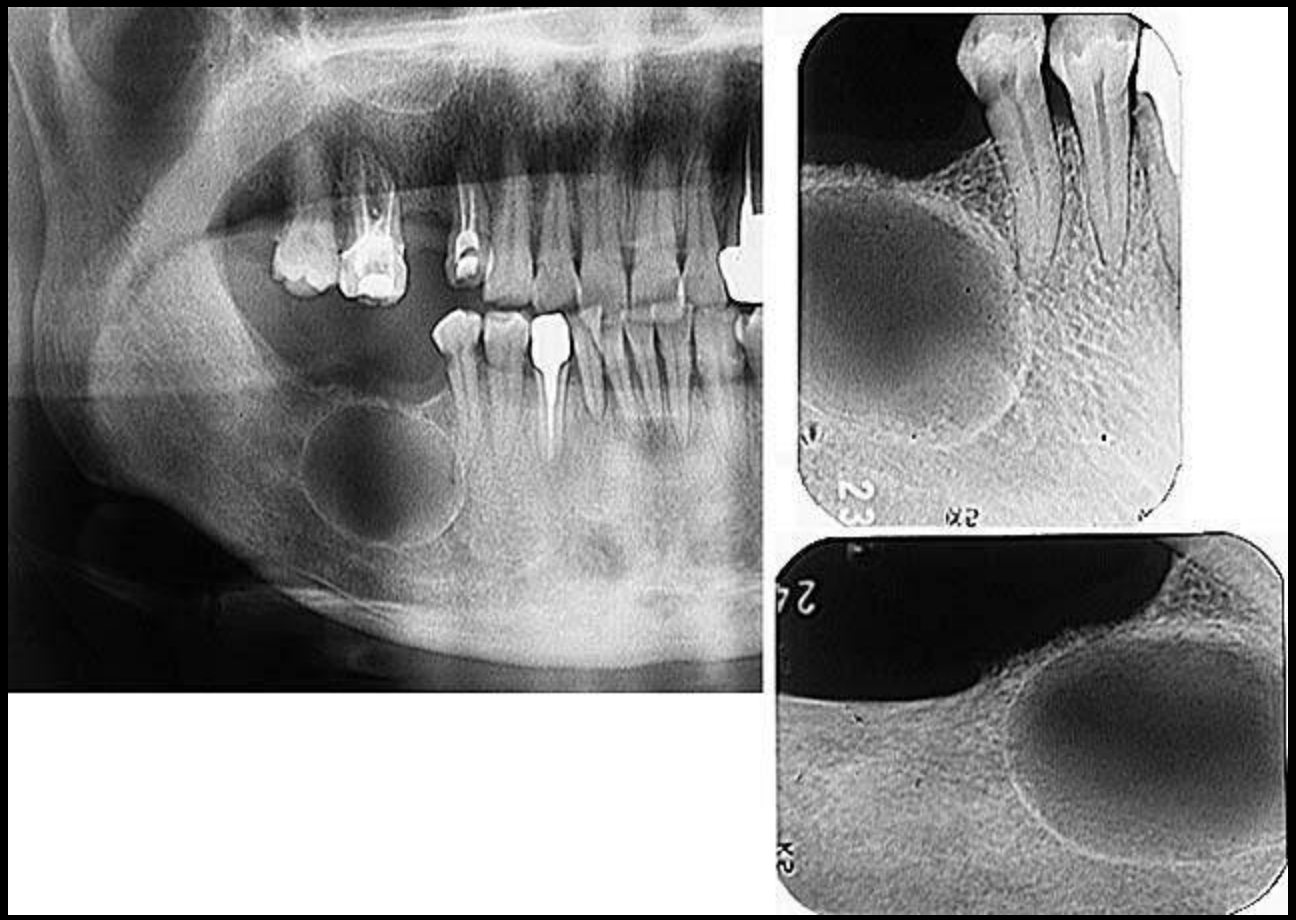






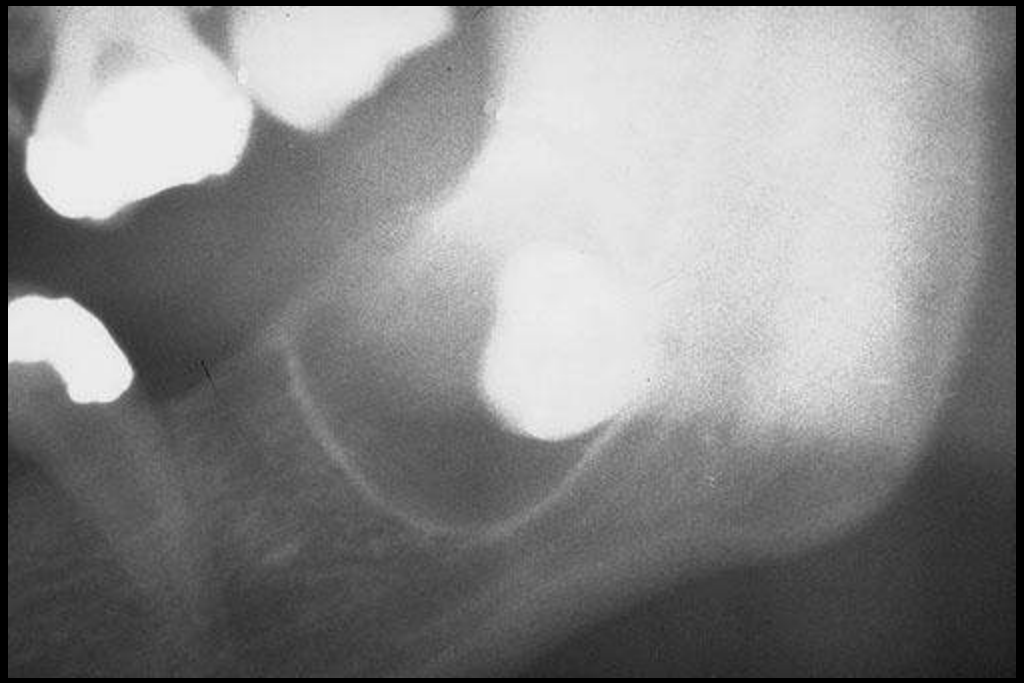
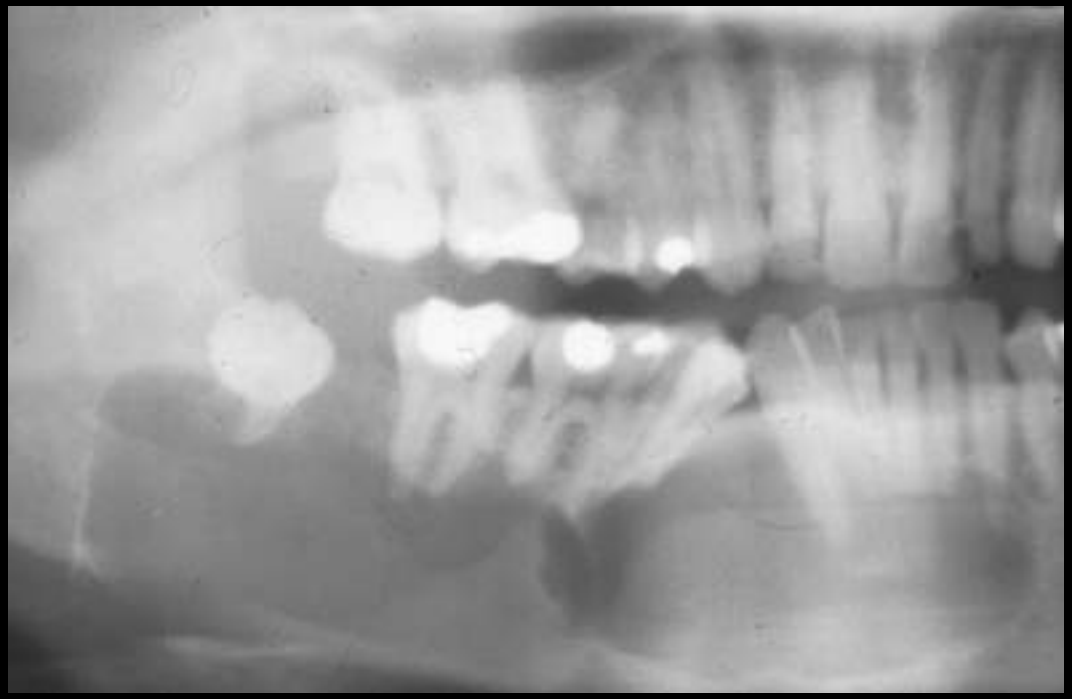


Fig. 55



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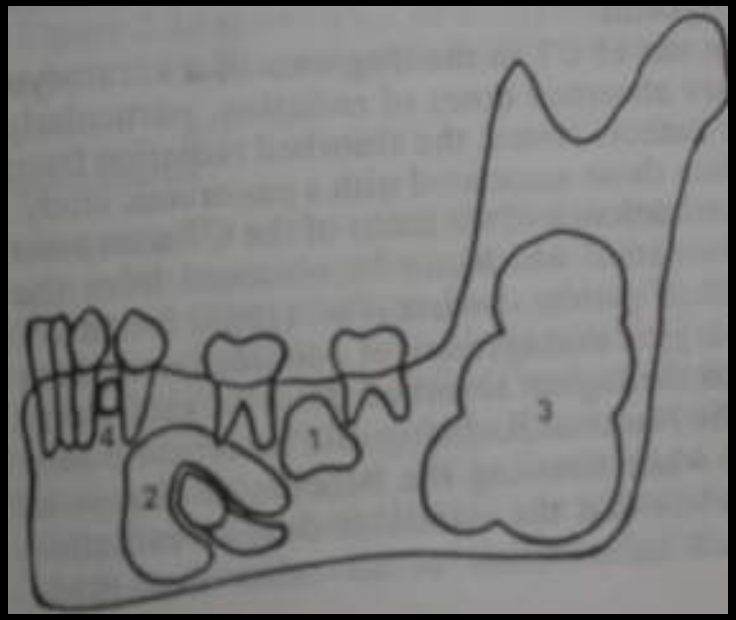
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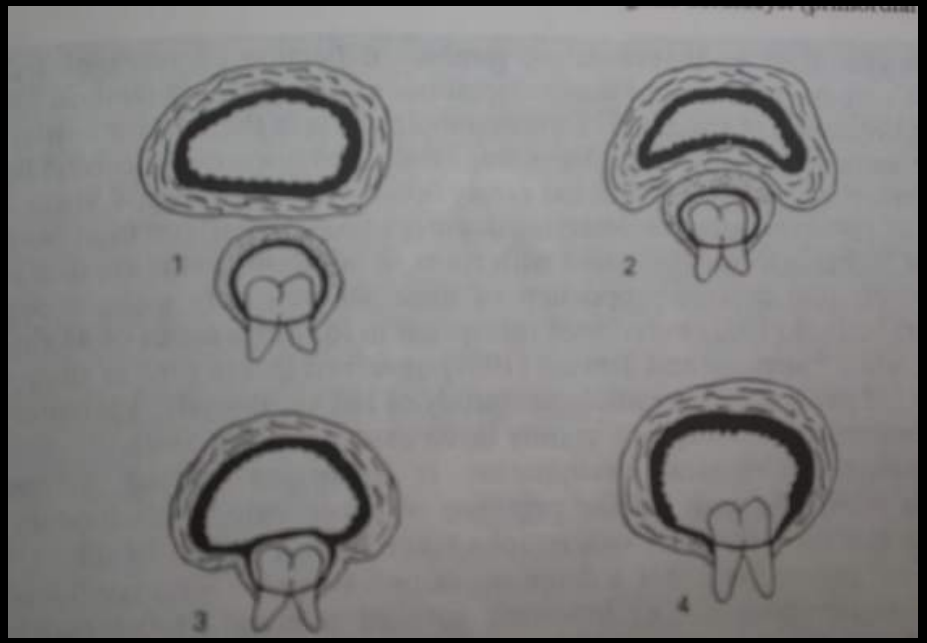
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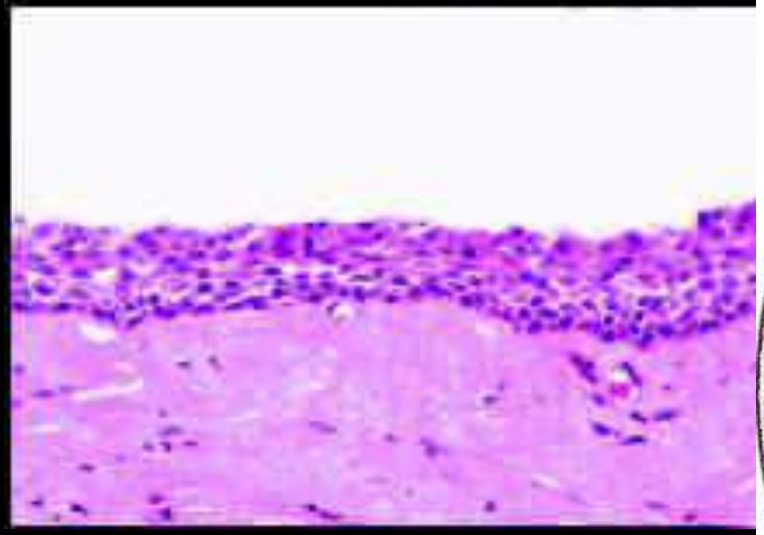

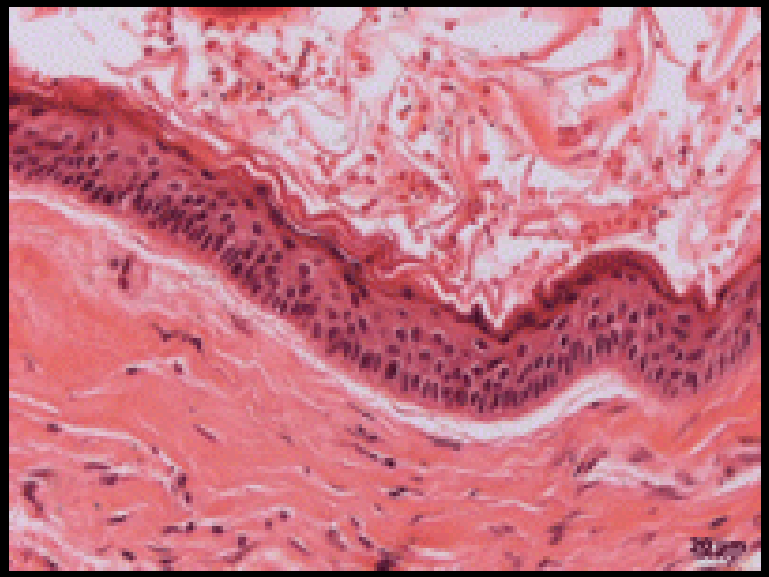

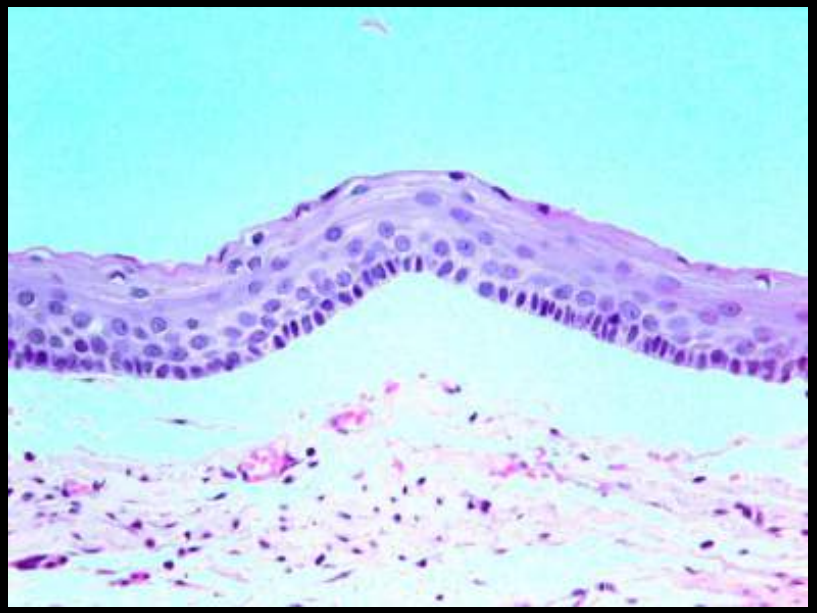

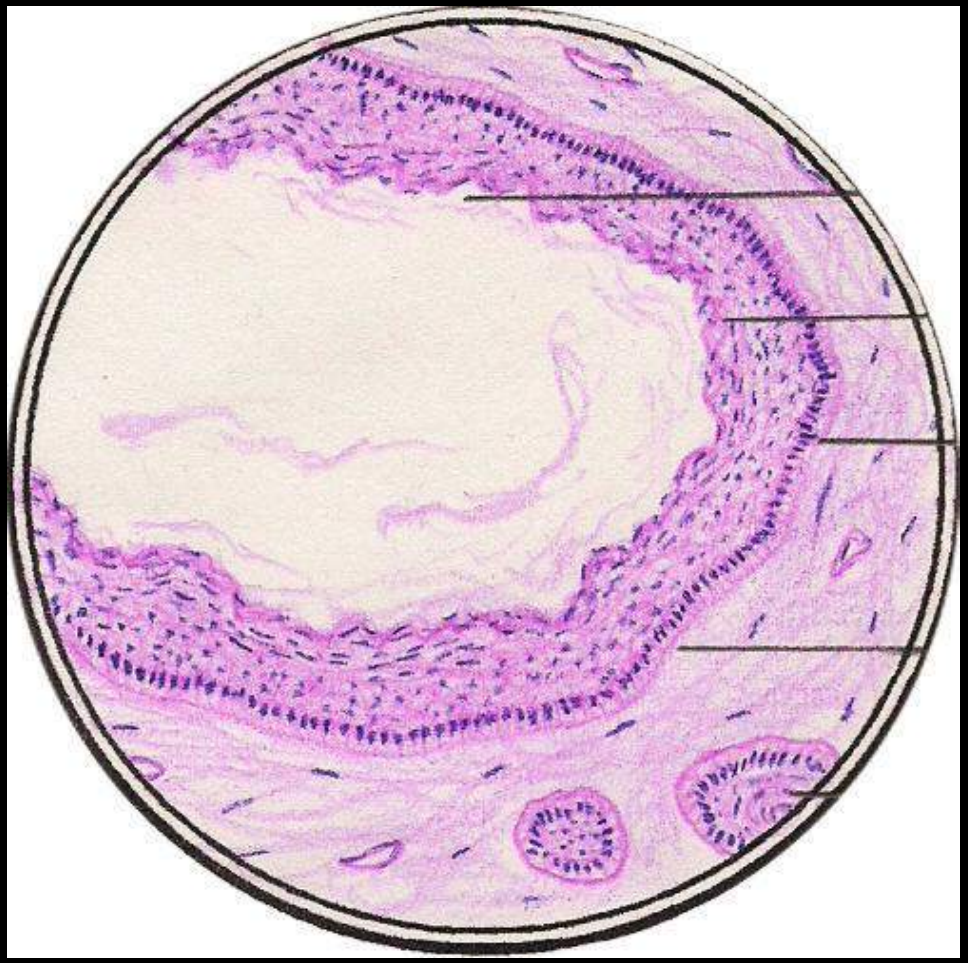

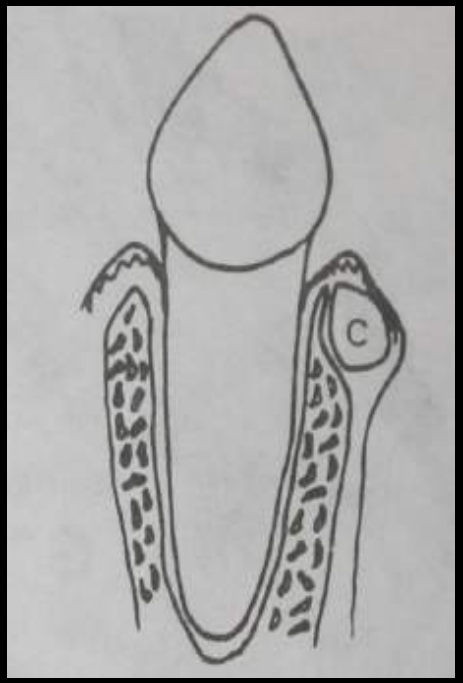
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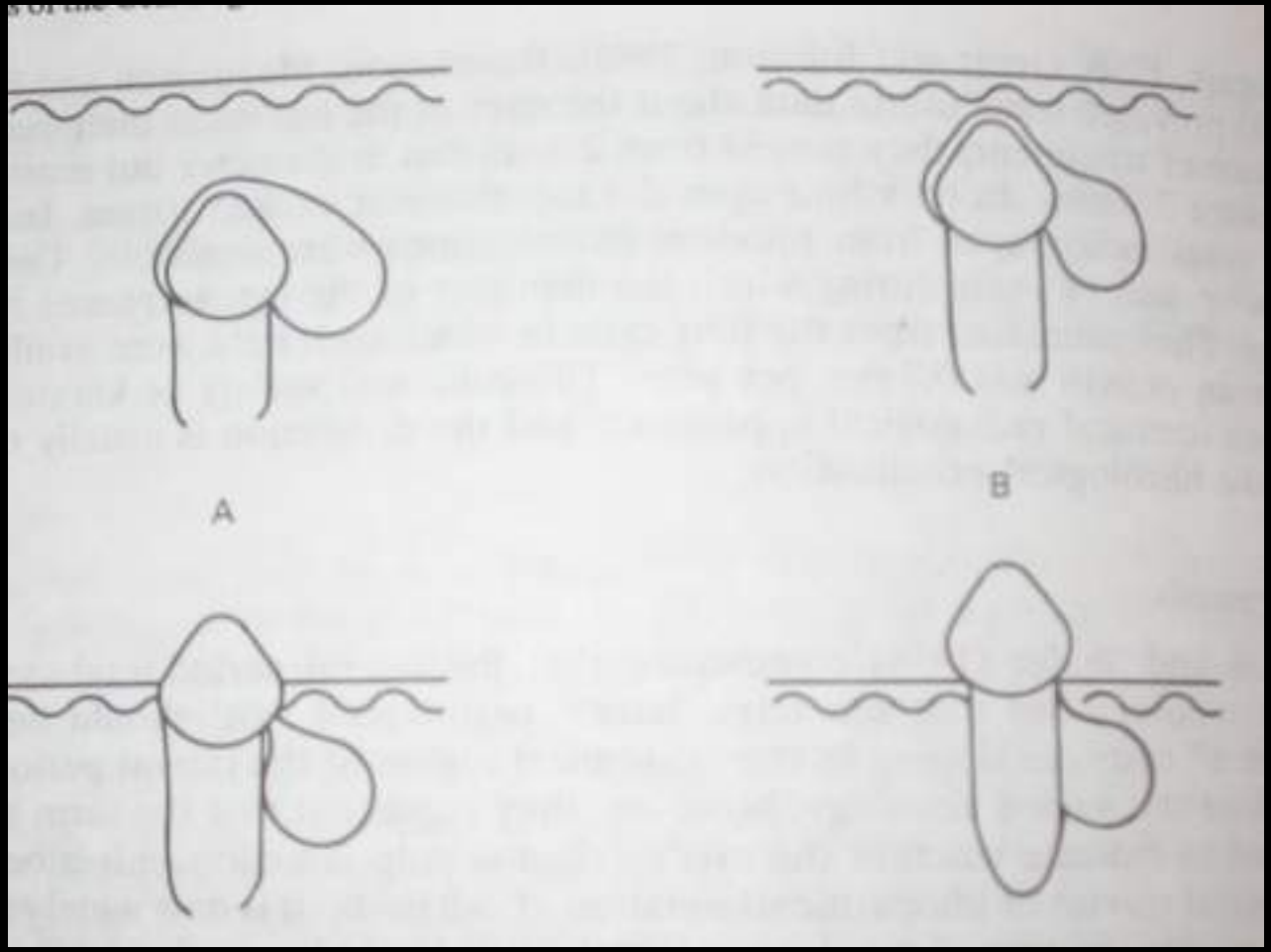
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Nutrition Facts
thank yu

Odontogenic tumours

- Odontogenic tumours and tumour-like lesions constitute a group of heterogeneous diseases that range from hamartomatous or non-neoplastic tissue proliferations to benign neoplasms to malignant tumours with metastatic potential.
- They are derived from epithelial, ectomesenchymal and/or mesenchymal elements of the tooth-forming apparatus.
- Odontogenic tumours are rare, some even extremely rare, but can pose a significant diagnostic and therapeutic challenge.

Found exclusively within the maxillofacial skeleton

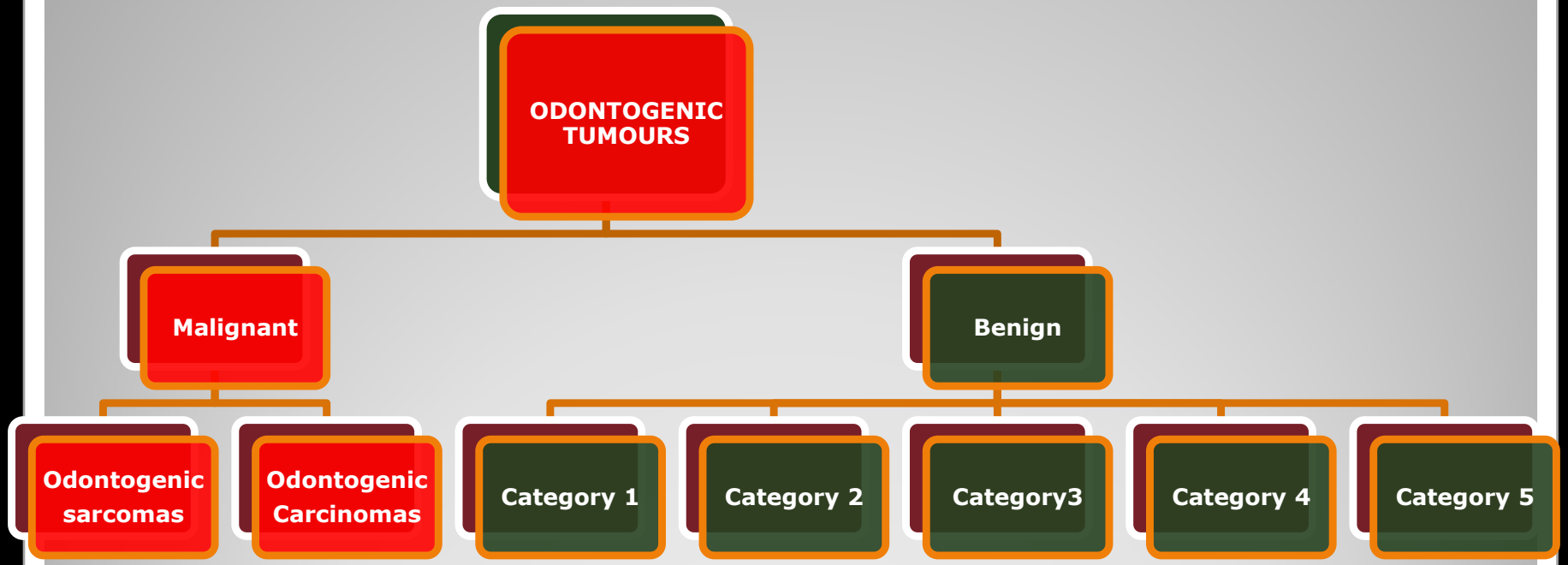
Intraosseous / Centrally Located

OR

Extraosseous or Peripherally located

(Soft tissue (gingiva) overlying tooth-bearing areas or alveolar mucosa in edentulous regions)

Classification


ODONTOGENIC TUMOURS
Malignant
Benign
Odontogenic sarcomas
Odontogenic Carcinomas
Category 1
Category 2
Category3
Category 4
Category 5

MALIGNANT TUMOURS		
Odontogenic carcinomas		
1	Metastasizing (malignant) ameloblastoma	9310/3
2	Ameloblastic carcinoma • Primary Type • Secondary Type – Dedifferentiated (Intraosseous) • Secondary Type -Dedifferentiated (Peripheral)	9270/3
3	Primary intraosseous squamous cell carcinoma • Solid type • Derived from keratocystic odontogenic tumour • Derived from odontogenic cysts	9270/3
4	Clear cell odontogenic carcinoma	9341/3
5	Ghost cell odontogenic carcinoma	9302/3

ODONTOGENIC SARCOMAS		
1	Ameloblastic fibrosarcoma	9330/3
2	Ameloblastic fibrodentino–and fibro-odontosarcoma	9290/3

BENIGN TUMOURS

1. Odontogenic epithelium with mature, fibrous stroma without odontogenic ectomesenchyme

1	Ameloblastoma, solid / multicystic type (intra osseous)	9310/0
2	Ameloblastoma, solid / multicystic type (Extraosseous/peripheral)	
3	Ameloblastoma, desmoplastic type	
4	Ameloblastoma, unicystic type	
5	Squamous odontogenic tumour	9312/0
6	Calcifying epithelial odontogenic tumour	9340/0
7	Adenomatoid odontogenic tumour	9300/0
8	Keratocystic odontogenic tumour	9270/0

BENIGN TUMOURS

2. Odontogenic epithelium with odontogenic ectomesenchyme, with or without hard tissue formation

1	Ameloblastic fibroma	9330/0
2	Ameloblastic fibrodentinoma	9271/0
3	Ameloblastic fibro-odontoma	9290/0
4	Odontoma	9280/0
5	Odontoma, complex type	9282/0
6	Odontoma, compound type	9281/0
7	Odontoameloblastoma	9311/0
8	Calcifying cystic odontogenic tumour	9301/0
9	Dentinogenic ghost cell tumour	9302/0

BENIGN TUMOURS		
3. Mesenchyme and/or odontogenic ectomesenchyme with or without odontogenic epithelium		
1	Odontogenic fibroma	9321/0
2	Odontogenic myxoma / myxofibroma	9320/0
3	Cementoblastoma	9273/0

BENIGN TUMOURS		
4. Bone-related lesions		
1	Ossifying fibroma	9262/0
2	Fibrous dysplasia	
3	Osseous dysplasias	
4	Central giant cell lesion (granuloma)	
5	Cherubism	
6	Aneurysmal bone cyst	
7	Simple bone cyst	
5. OTHER TUMOURS		
1	Melanotic neuroectodermal tumour of infancy	9363/0

Definitions

- 'An odontogenic tumor that is usually 'unicentric, nonfunctional, intermittent in growth, anatomically benign and clinically persistent' - Robinson
- U N I A C
- It is a true neoplasm of enamel organ type tissue which does not undergo differentiation to the point of enamel formation – Shafer
- A benign but locally invasive polymorphic neoplasm consisting of proliferating odontogenic epithelium , which usually has a follicular or plexiform pattern , lying in a fibrous stroma. - WHO

AMELOBLASTOMAS

Synonyms

- Ameloblastoma -- Churchill in 1934
- Adamantinoma - Malassez
- “Epithelioma Adamantin“ – Malassez
- Adamantoblastoma

Clinical Types

- Conventional/ Solid multicystic ameloblastoma (Peripheral or Central) 86%
- Unicystic ameloblastoma 13%
- Pituitary ameloblastoma / Ameloblastoma of the long bones 1%

- Source of cell origin
- Initiation of the tumor
- Growth of the tumor

Etiopathogenesis

Odontogenic epithelial origin

- Rests of dental lamina
- Developing enamel organ
- Epithelial lining of odontogenic cysts
- Basal cells of the oral mucosa
- Heterotopic epithelium in other parts of the body, especially the pituitary gland.

Source of cells/histogenesis

Initiation

- **UKNOWN**

- It is thought that it is likely the result of alterations or mutations in the genetic material of cells that embryologically pre -programmed for tooth development.
- **Defective genes**

  Ras, Myc, Fos, p53

- Multifactorial
- Environmental factors and individual patient variables (e.g. general health status, nutritional status) also likely have a role in modulating the nature of the disease.

Growth of the tumor

Incidence

- Most common odontogenic tumor next to Odontome – worldwide
- In India – first most commonest odontogenic tumor(61.5%)
- Age - wide age range of occurrence 10 to 90 yrs, most commonly seen in 4^{th} decade
- Gender – no significant sex predilection, but slightly more common in males
- Race – more common in Blacks than Whites
- Site –mandible > Maxilla
- Ramus of the mandible > Angle of the mandible> Anterior mandible (more common in Blacks)> Posterior maxilla > mandibular Premolar area > Anterior Maxilla

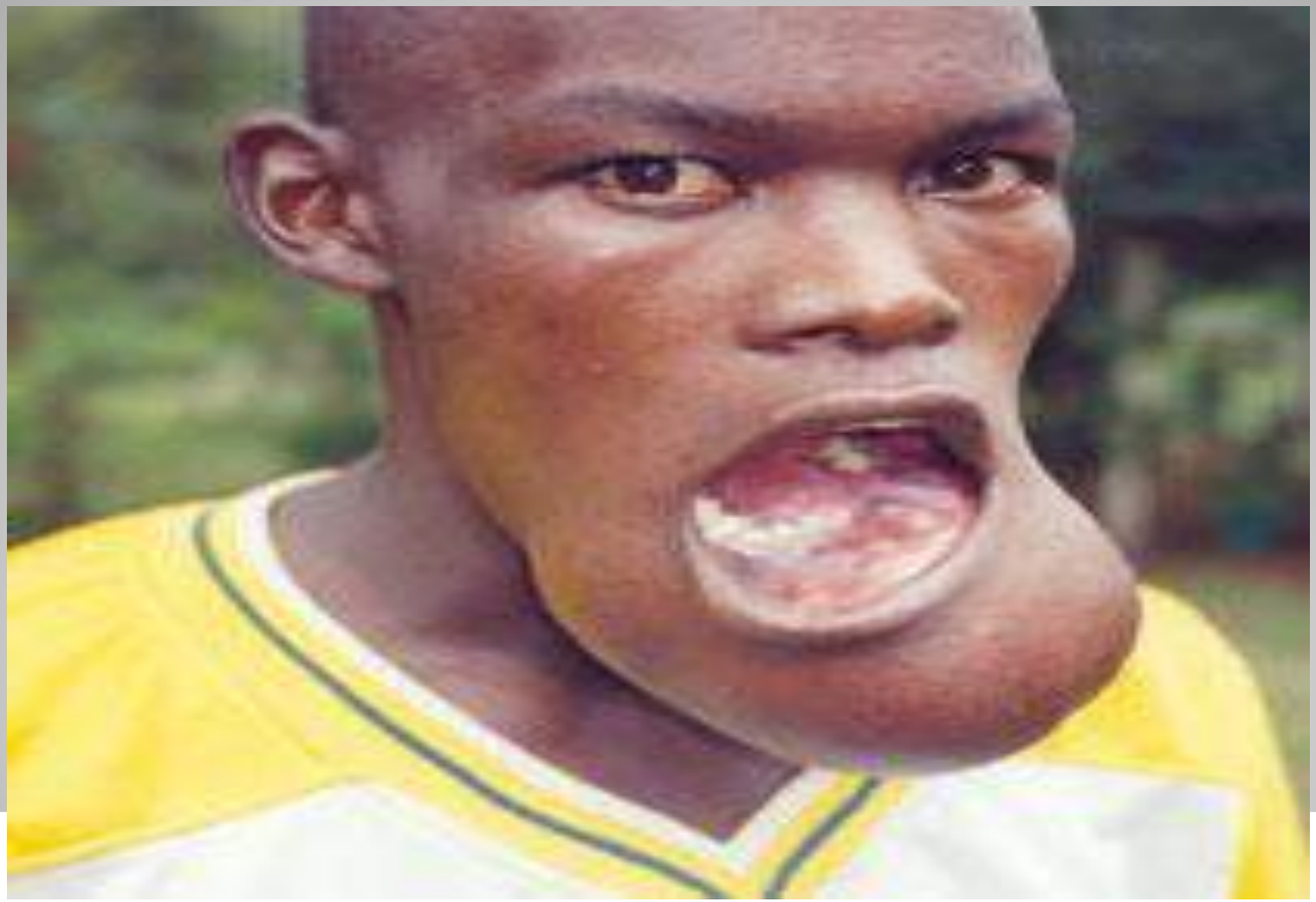
Clinical features

- Asymptomatic
- Smaller lesions detected during routine radiographs

Common clinical presentation

- A painless swelling
- Expansion of the jaws.
- If untreated the tumor may grow to massive size
- Usually no pain and paresthesia even with large tumors.
- Facial asymmetry
- Displacement of tooth

Ameloblastoma



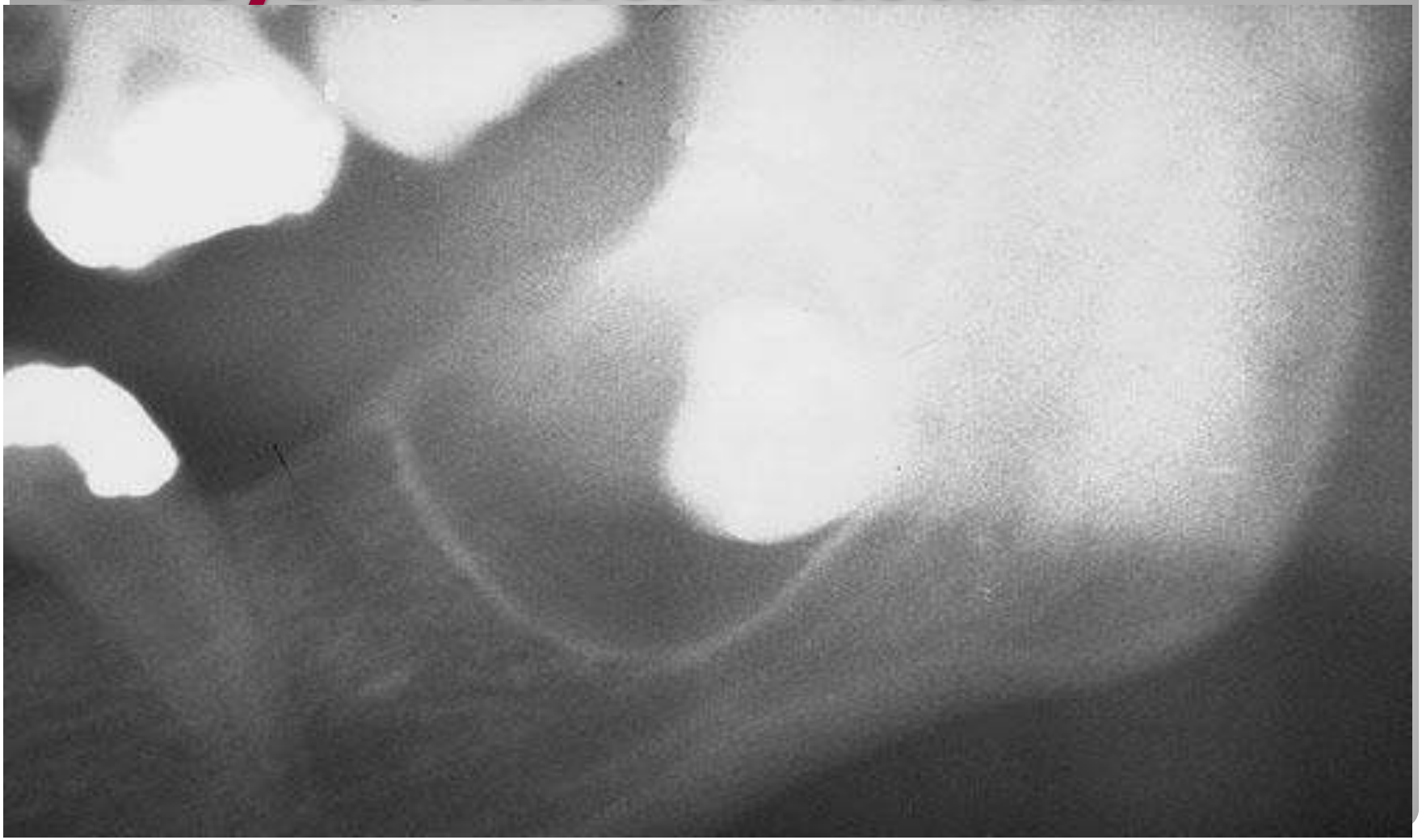
Radiographic features

- Multilocular radiolucent lesion
- Soap bubble appearance - radiolucent loculations are large.
- Honey comb - radiolucent loculations are small.
- Buccal and lingual cortical expansion
- Resorption of root adjacent to the tumor
- Many cases are associated with an unerupted tooth, most commonly mandibular third molar will be associated with (Unicystic).
- The margins of the radiolucent defect often show irregular scalloping.
- Mixed radiolucency and opacity may be seen with in the tumor in desmoplastic variants.

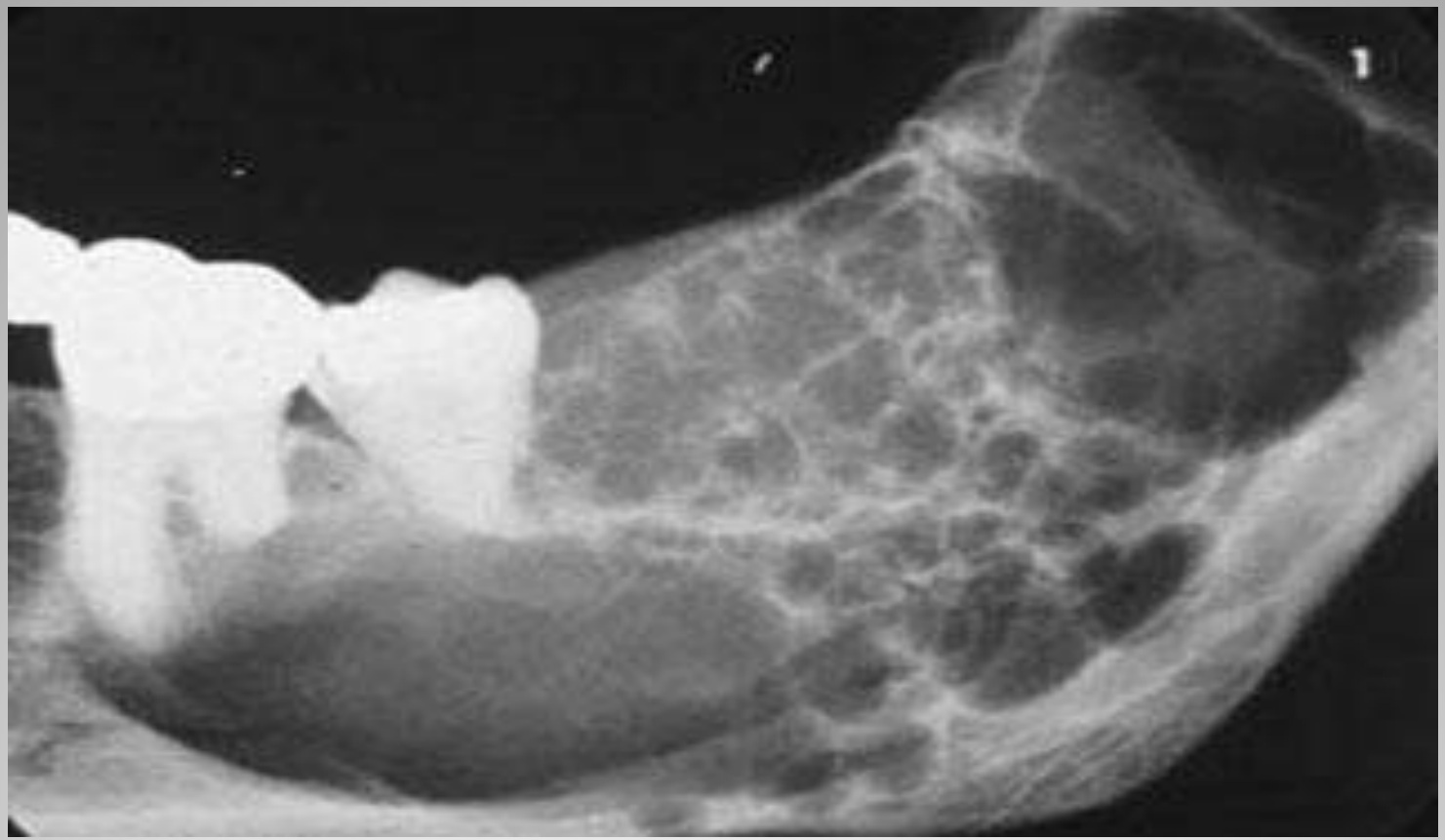
18

5

Unicystic Ameloblastoma



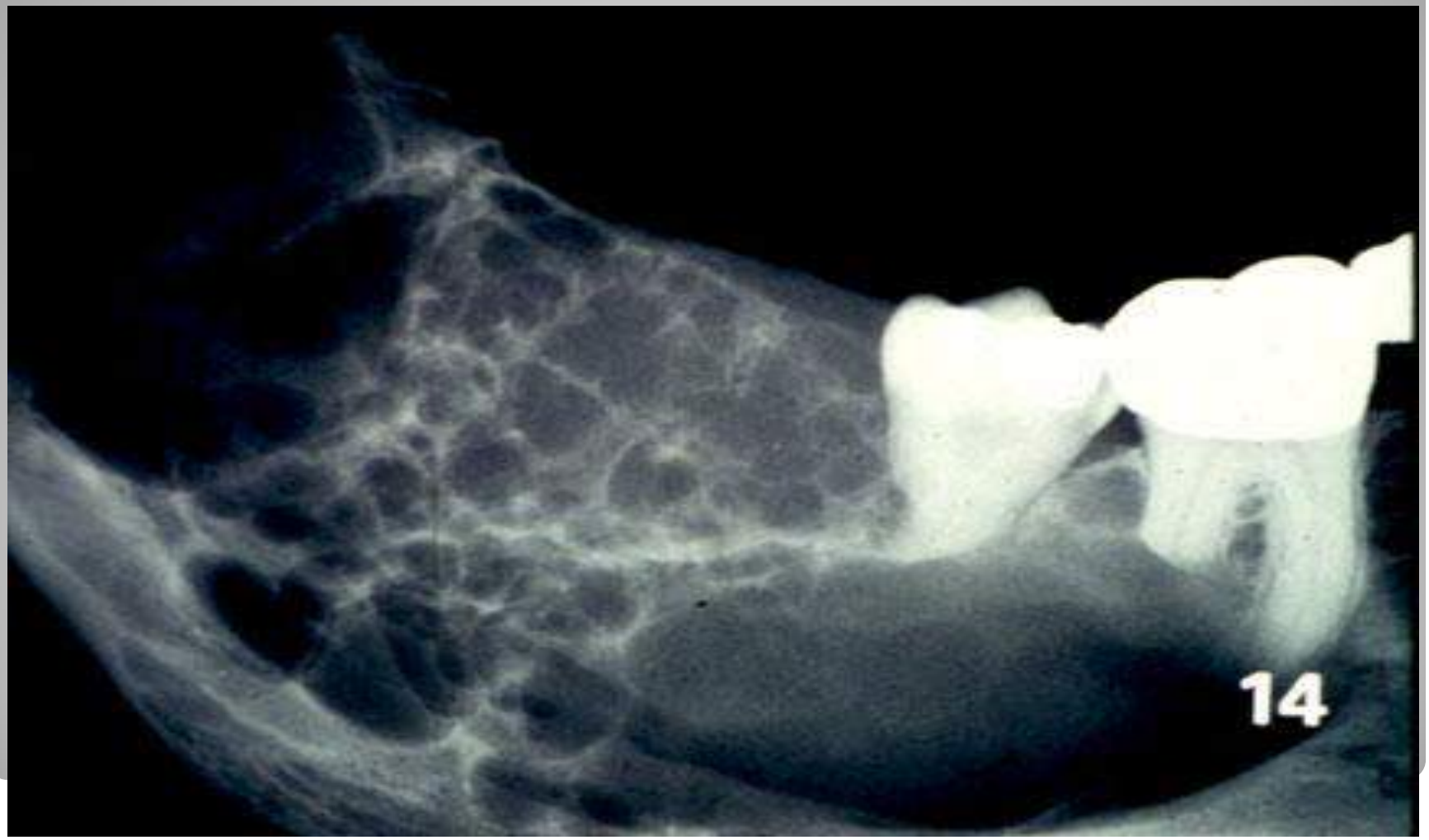
Ameloblastoma, soap bubble appearance



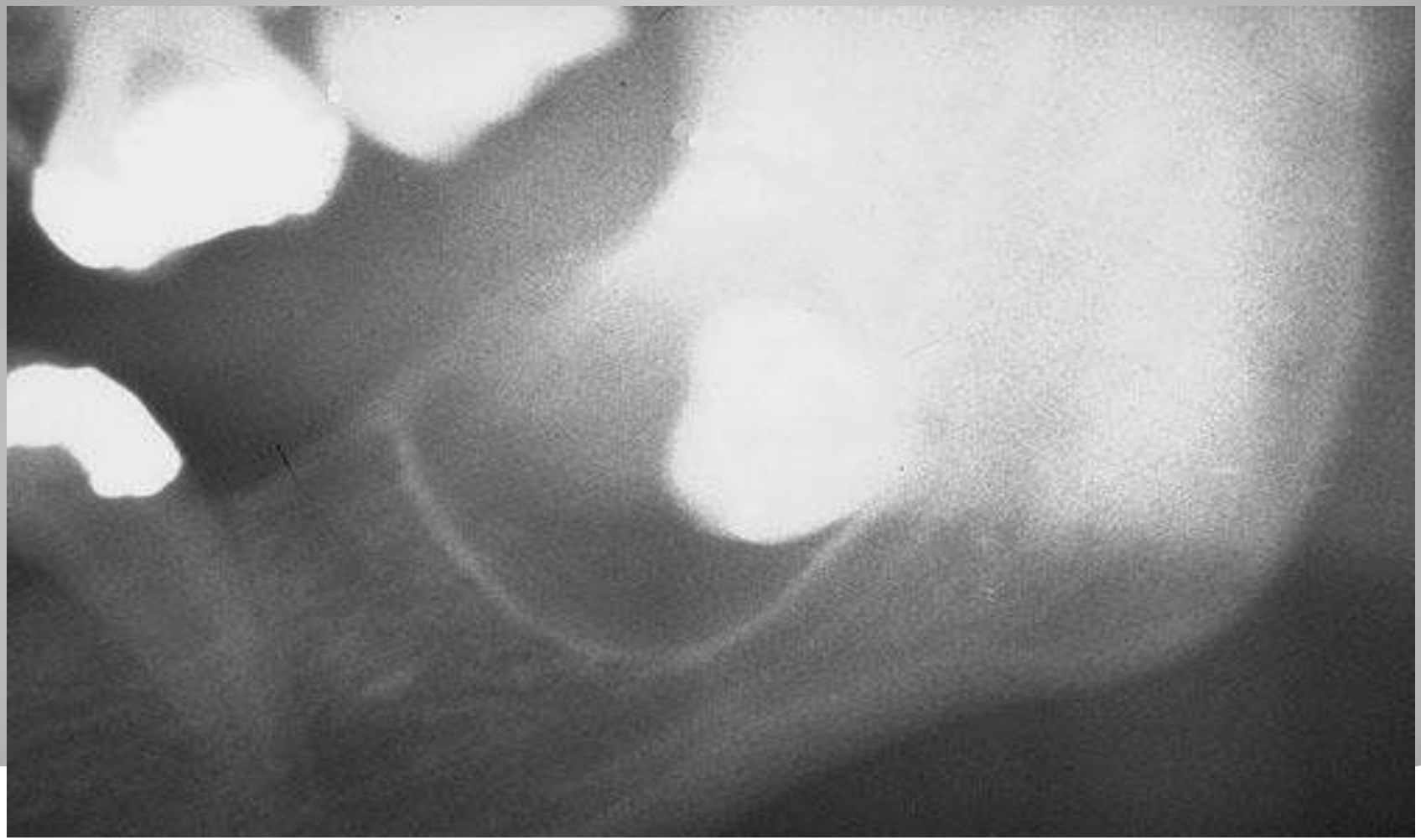
Ameloblastoma, Honeycomb appearance

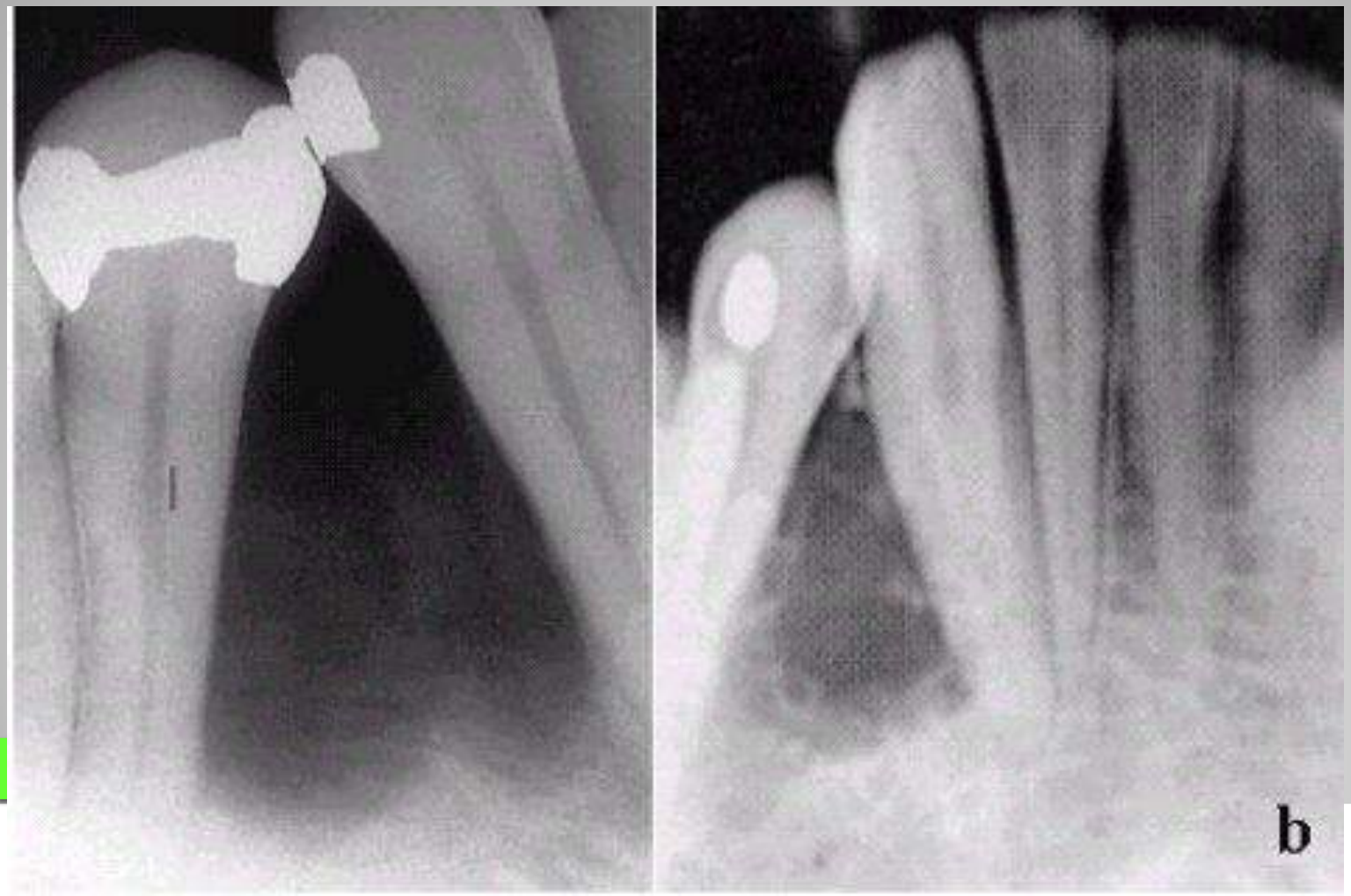
Ameloblastoma

Multicystic Ameloblastoma Honeycomb



Unicystic Ameloblastoma

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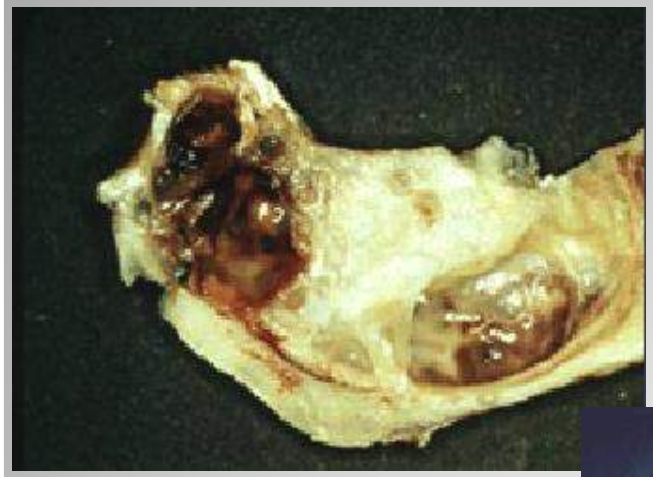
Peripheral ameloblastoma

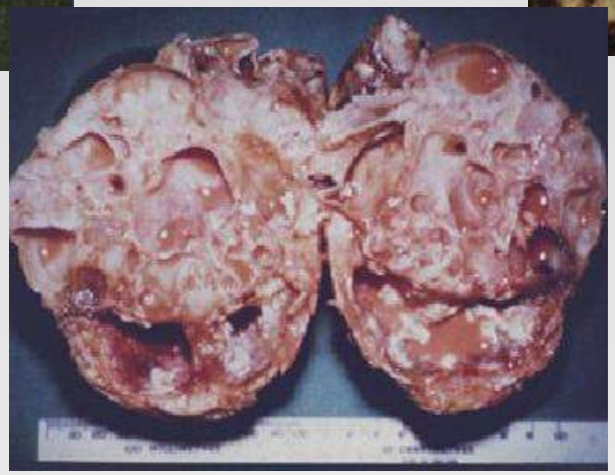
Peripheral Ameloblastoma

- A tumor which histologically resembles the typical central or intraosseous ameloblastoma but which occurs in the soft tissue outside and overlying the alveolar bone
- **'Basal cell carcinoma of the gingiva'**
- Gingiva and alveolar ridges
- Swelling, superficial ulcerations, pain as the lesion matures
- This tumor appears to originate from either surface epithelium or remnants of dental lamina.

Gross features

Varying combinations of cystic and solid features.





Histopathological features

Histopathological Subtypes

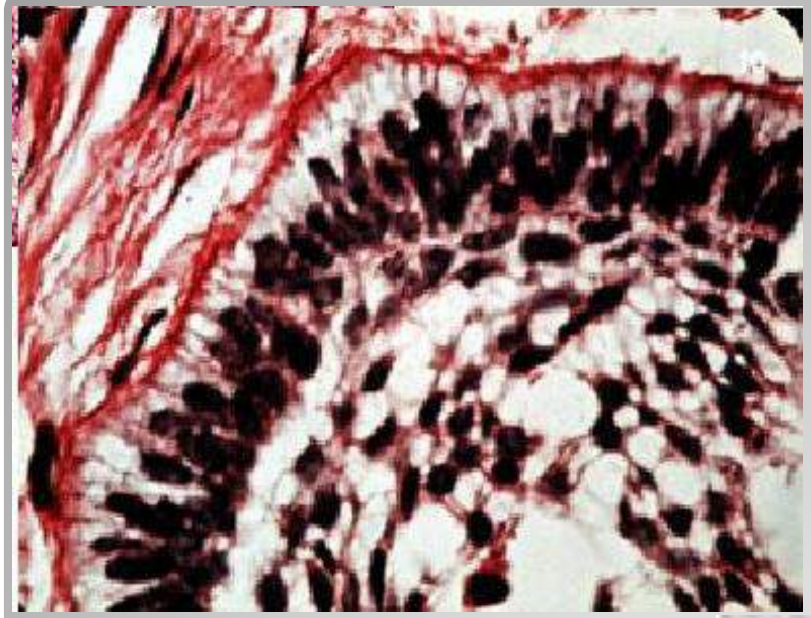
- Follicular Ameloblastoma
- Plexiform Ameloblastoma
- Acanthomatous Ameloblastoma
- Desmoplastic Ameloblastoma
- Granular Cell Ameloblastoma
- Basal Cell Ameloblastoma

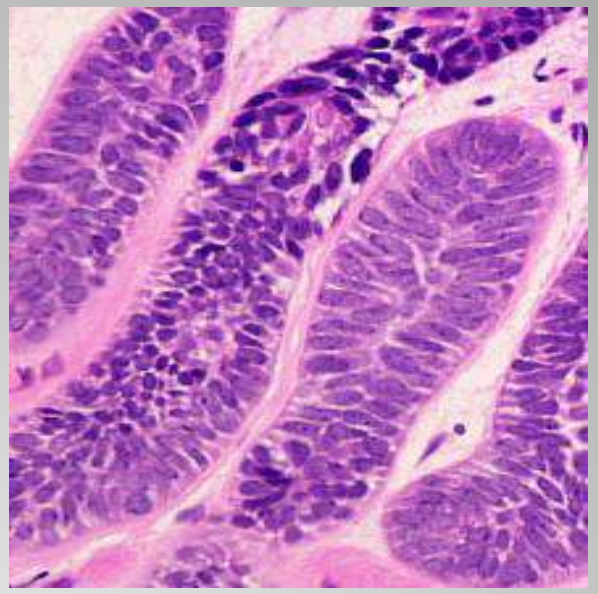
Histopathology

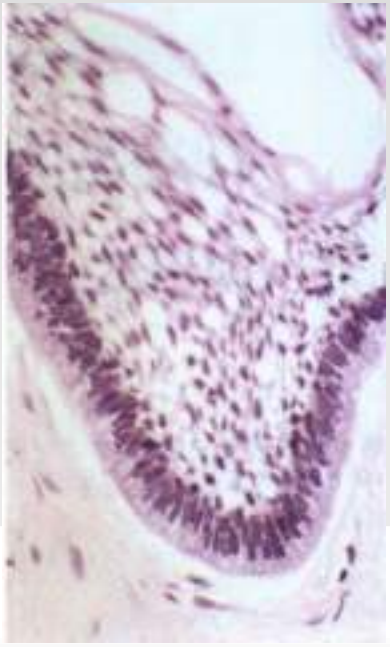
- The epithelial component of the neoplasm proliferates in what seems to be disconnected islands, strands, and cords within the moderately dense collagenized fibrous connective tissue stroma.
- The islands, strands, and cords may vary considerably in size and mimics various stages of enamel organ formation.
- In high power magnification, the darkly staining periphery is composed of tall columnar cells with hyperchromatic nuclei.
- The nuclei tend to be round to oval in shape, and the nuclei of adjacent cells are in roughly the same location within the cytoplasm (Palisading arrangement).
- The palisaded nuclei are oriented away from the basement membrane area of the cell, and a small clear vacuole can be seen between the nucleus and the basement membrane.

Histopathology

- This peripheral layer of **tall columnar cells** with **nuclear hyperchromasia**, **reverse polarity of the nuclei**, and **subnuclear vacuole formation** mimic the normal embryologic development of the tooth bud at the stage of enamel matrix production.
- Central cells of the islands or strands mimics stellate reticulum
- These classic features of ameloblastoma originally were described by Vickers and Gorlin in 1970 **(VICKERS AND GORLIN CRITERIA).**
- Proliferating epithelium is seen to exert an inductive effect on the surrounding connective tissue stroma. In these areas, a zone of hyalinization of the collagen is present immediately adjacent to the epithelium.

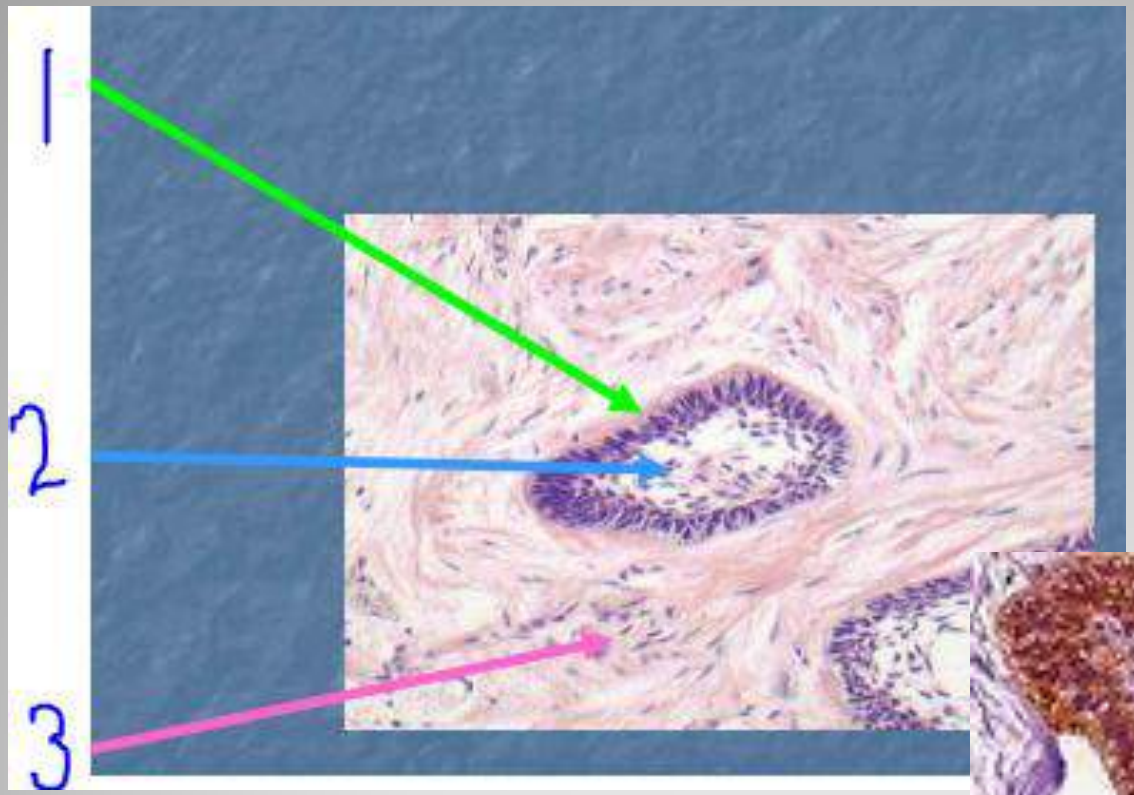






Follicular Ameloblastoma

- Most commonly encountered variant
- Simple Ameloblastoma
- Composed of many small discrete islands with peripheral layer of cuboidal or columnar cells whose nuclei are generally well polarized.
- These cells strongly resemble ameloblasts or preameloblasts and these enclose a central mass of polyhedral, loosely arranged cells resembling the stellate reticulum.
- Occassionaly Stellate reticulum like tissue has undergone complete breakdown or cystic degeneration– Cystogenic ameloblastoma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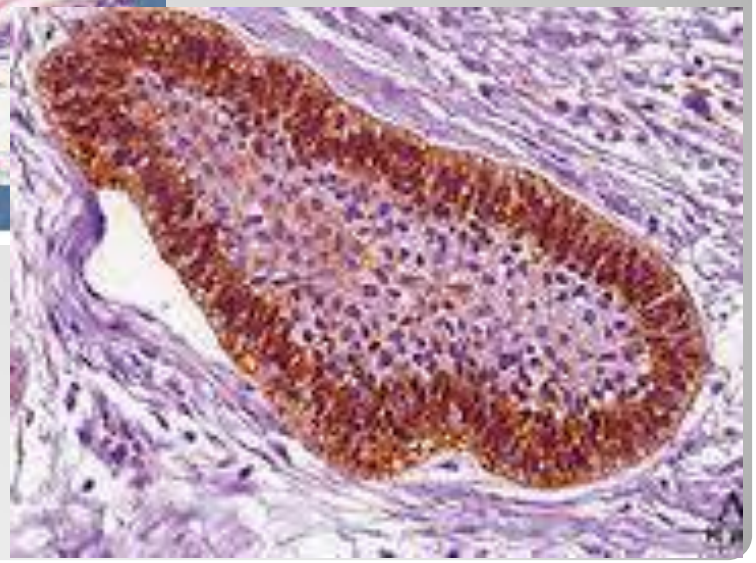
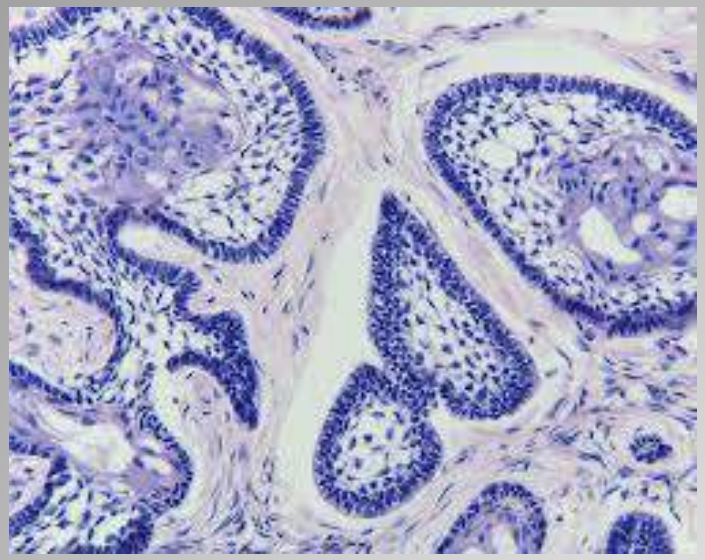
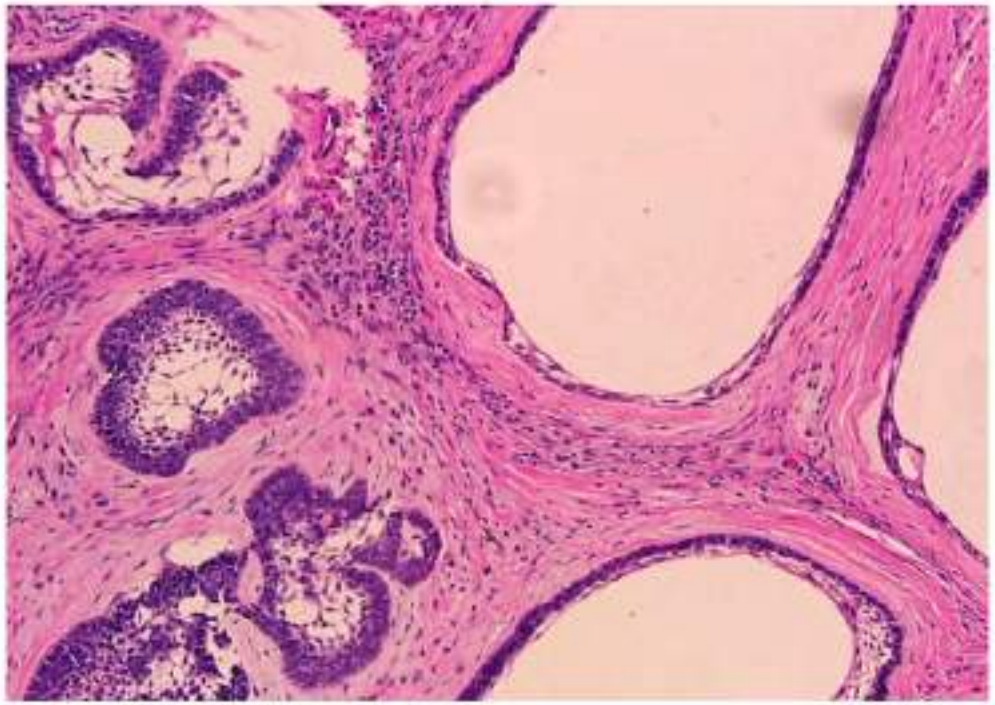
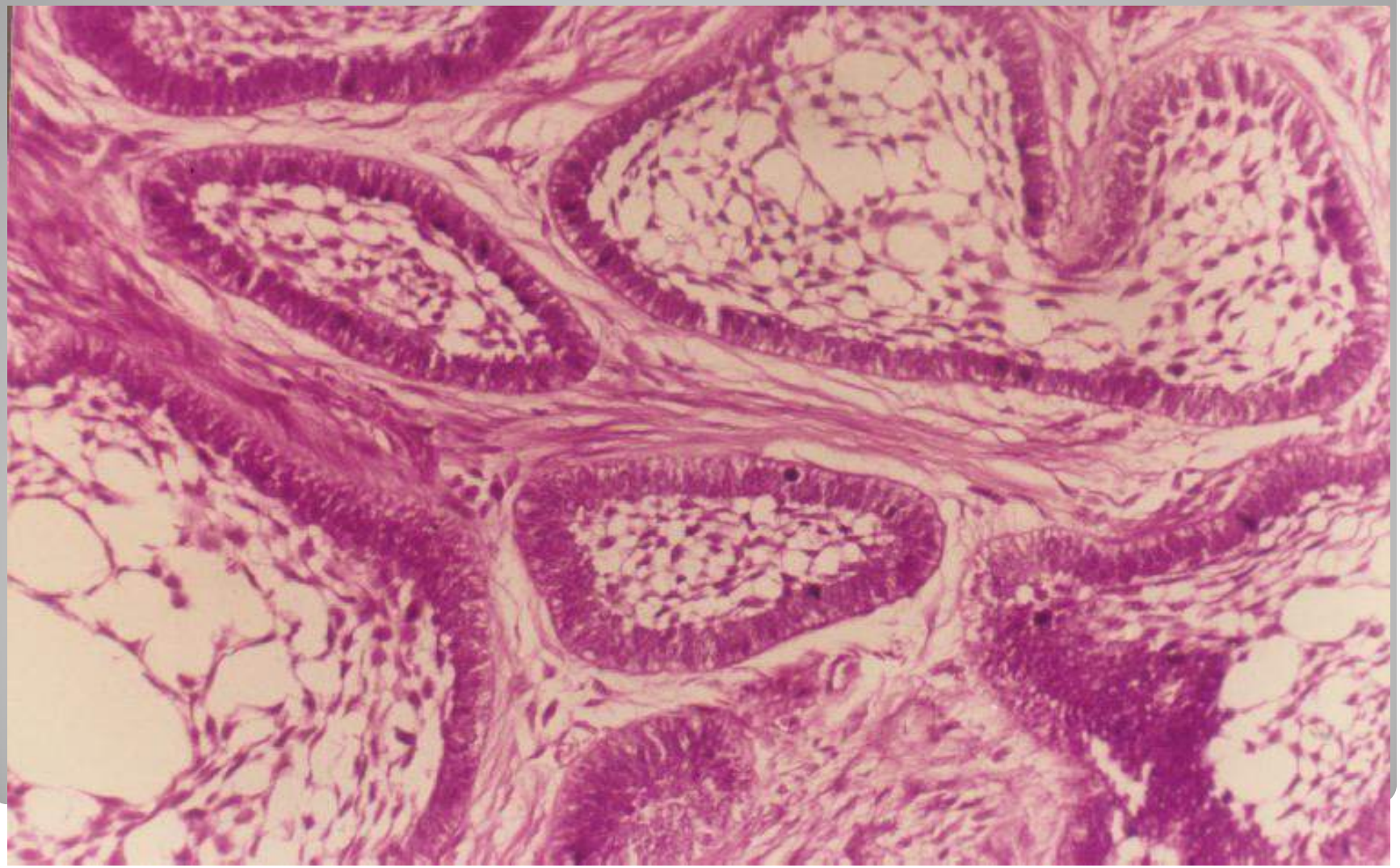


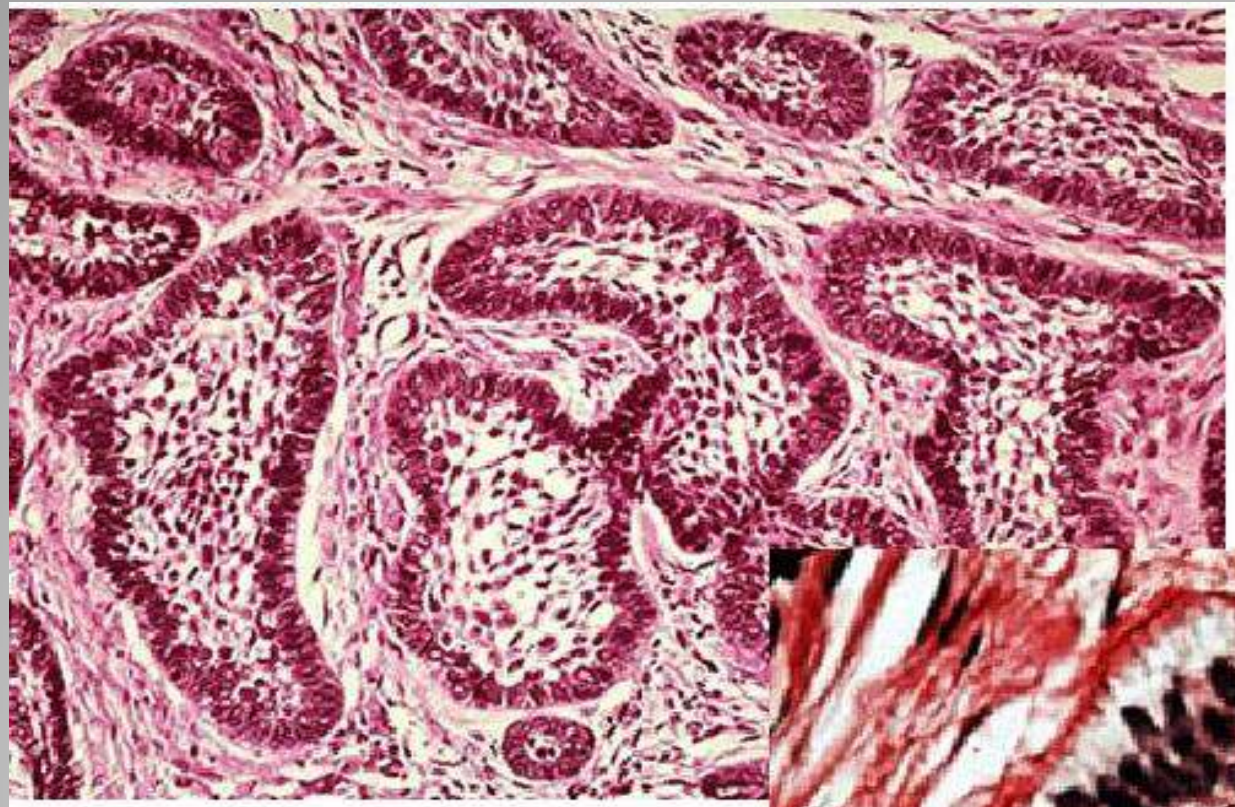


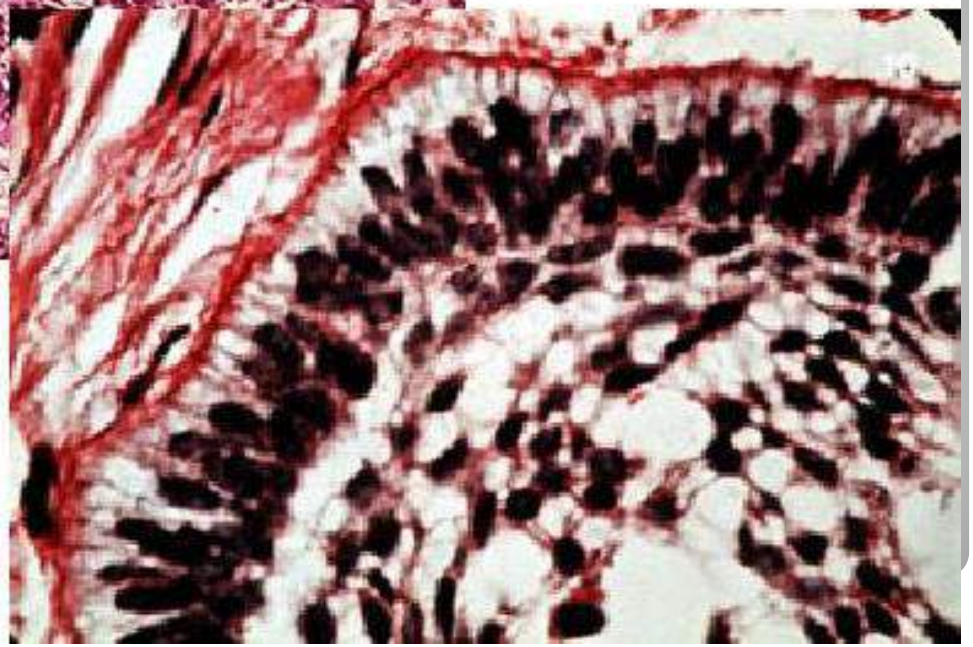


Cystogenic Ameloblastoma

Follicular Ameloblastoma







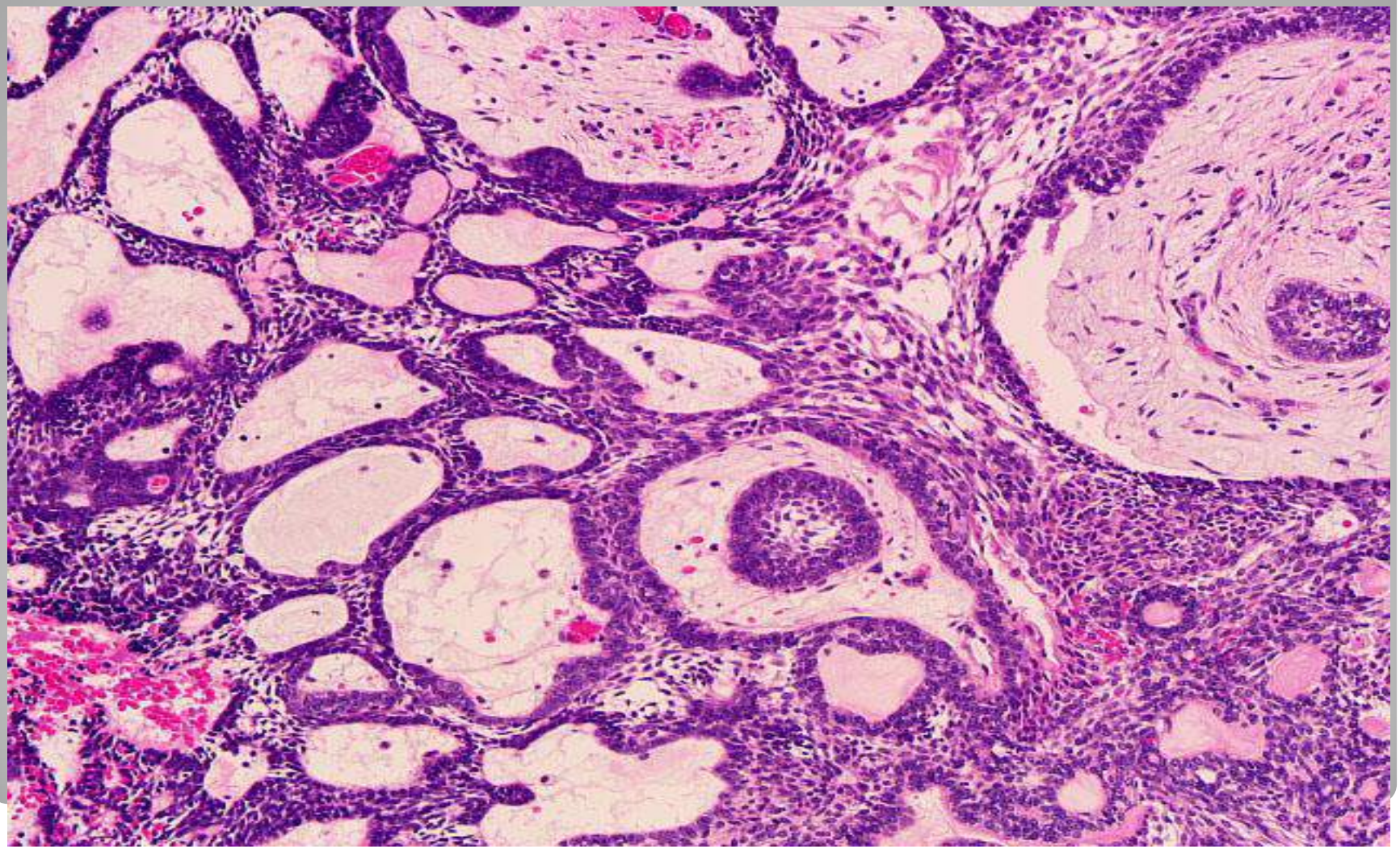
Follicular Ameloblastoma

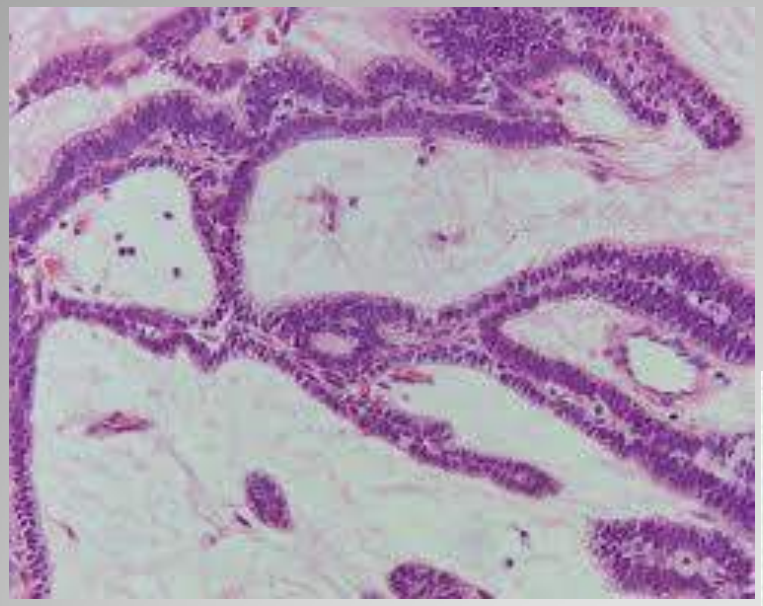
- Ameloblast like tumor cells are arranged in irregular masses, or more frequently, as a network of interconnecting strands of cells, between these layers may be found stellate reticulum like cells.

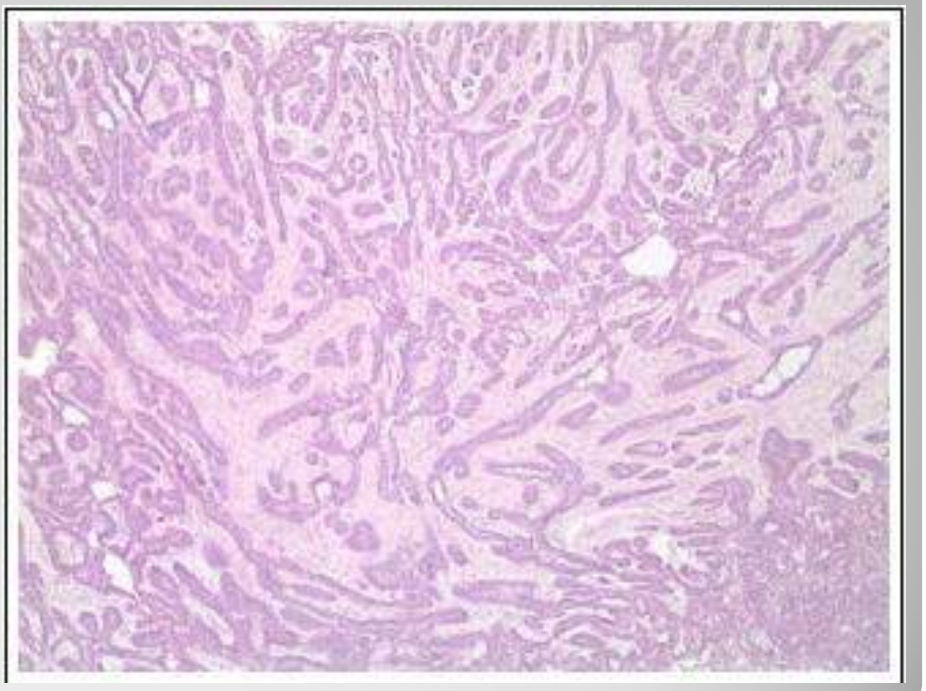
- Sometimes double rows of columnar cells are lined up back to back. However, the stellate reticulum like tissue is much less prominent in the plexiform type than in the follicular type of ameloblastoma.

Plexifom Ameloblastoma

Plexiform Ameloblastoma

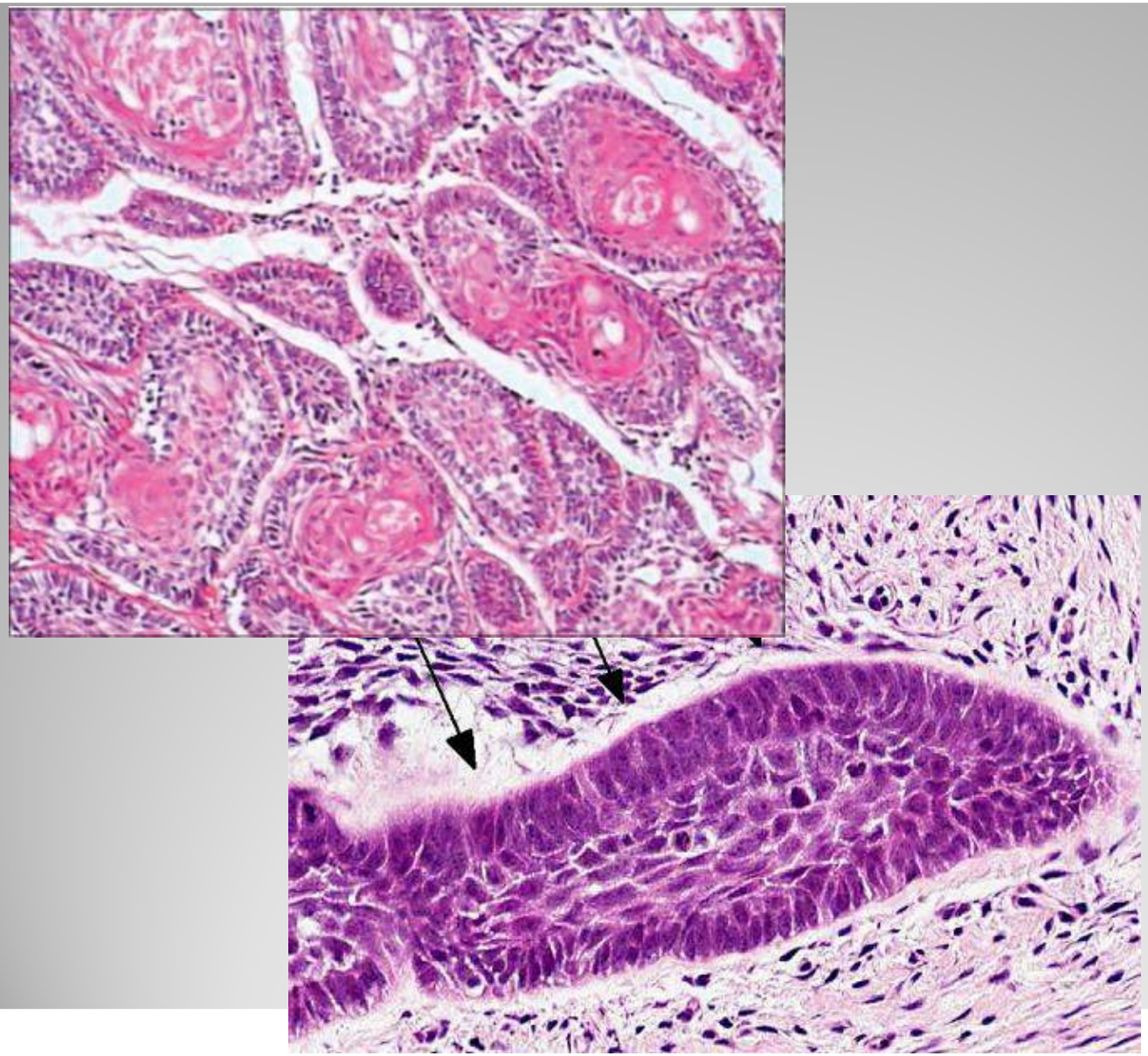


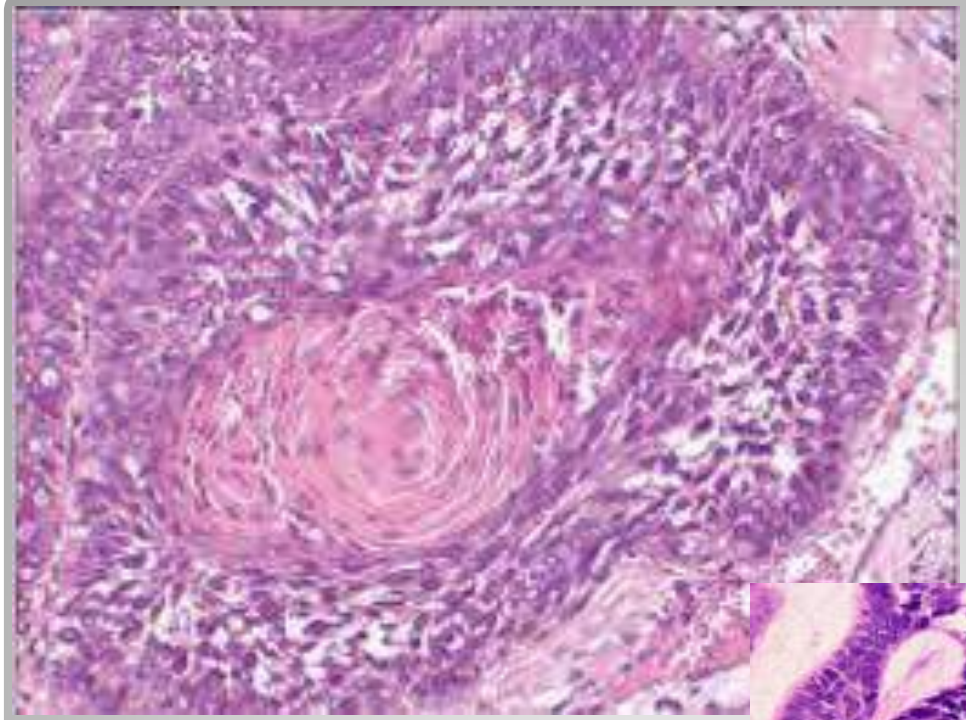


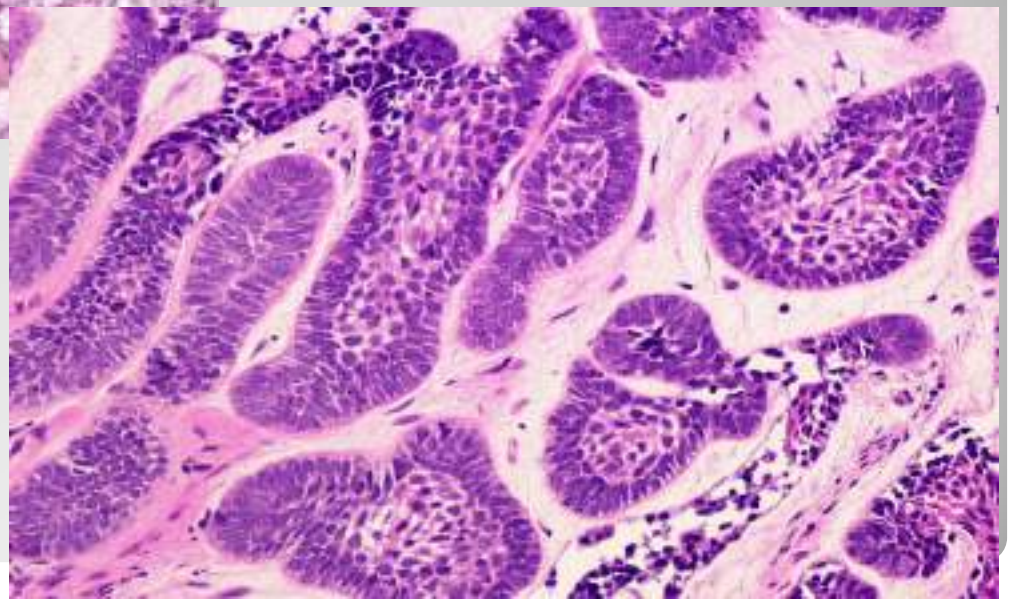


- Cells occupying the position of the stellate reticulum undergo squamous
- metaplasia, sometimes with keratin formation in the central portion of the tumor islands.
- This usually occurs in the follicular type of ameloblastoma.
- On occasion, epithelial or keratin pearls may even be observed.

Acanthomatous Ameloblastoma

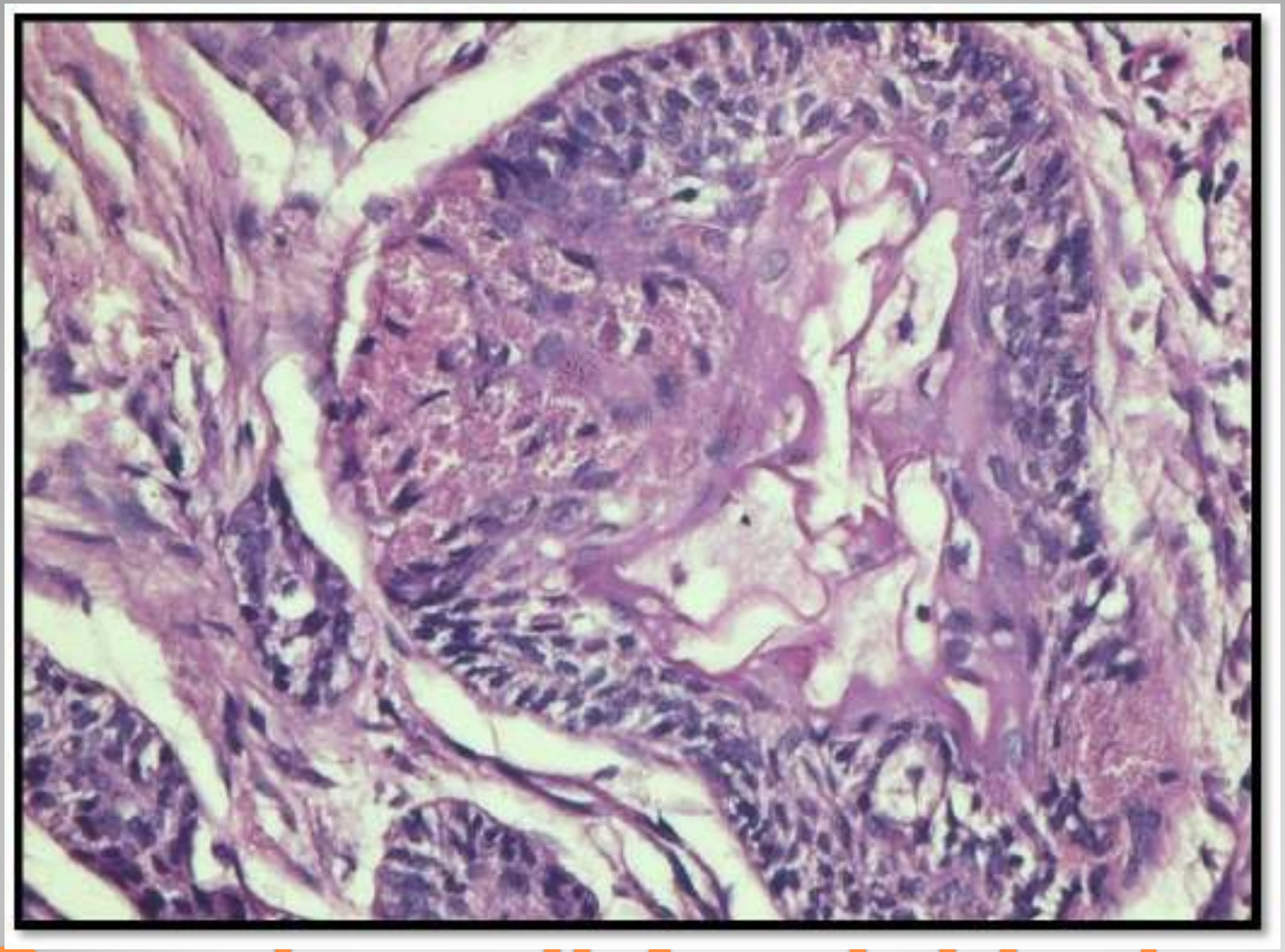




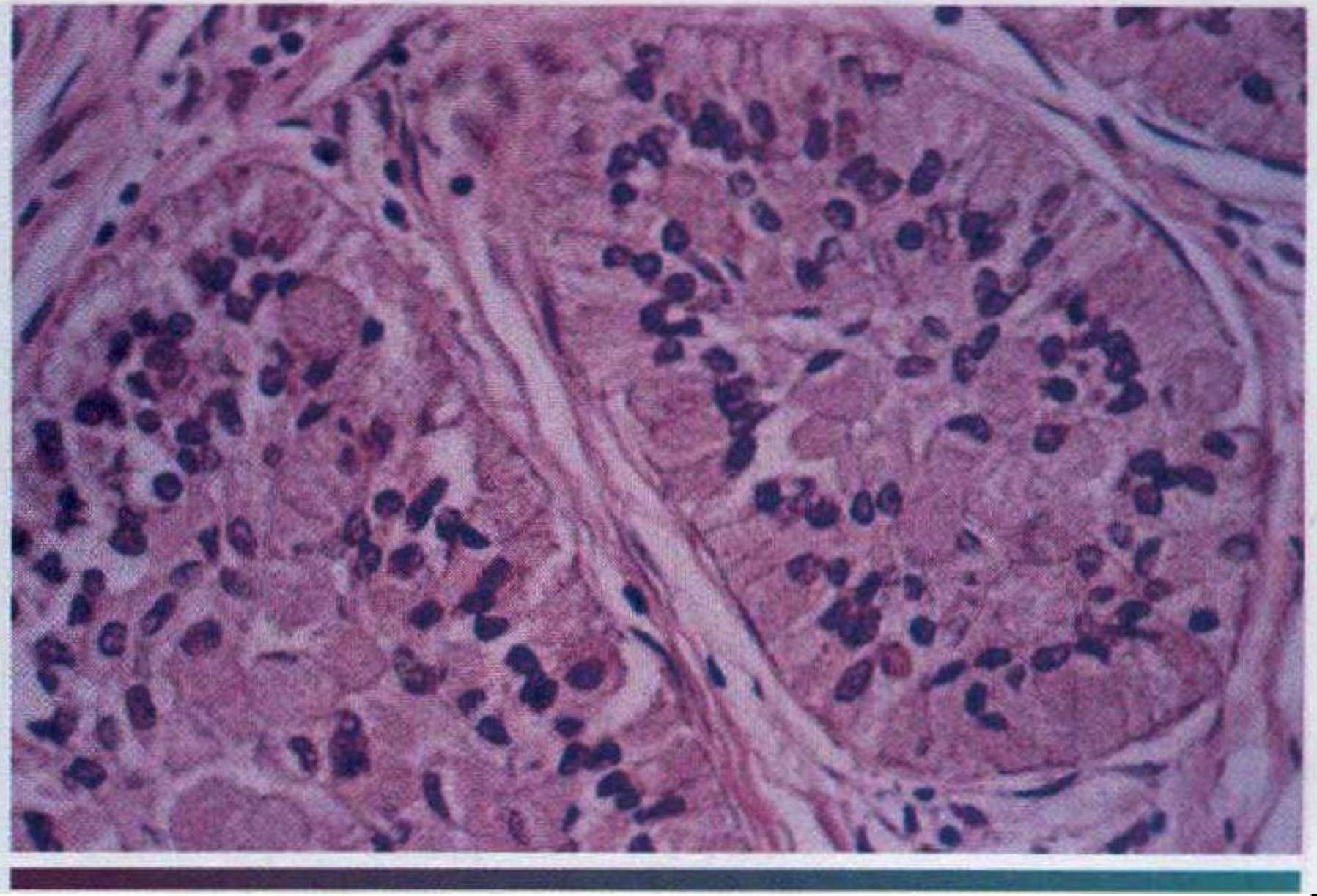


Granular cell Ameloblastoma

- Marked transformation of the cytoplasm, usually of the stellate reticulum like cells, so that it takes on a very coarse, granular, eosinophilic appearance.
- Ultrastructural studies -- these cytoplasmic granules represent lysosomal aggregates with no recognizable cellular components.
- Granular cell type appears to be an aggressive lesion with a marked proclivity for recurrence.
- In addition, several cases of this type have been reported as metastasizing. However, all other clinical features of the lesion appear similar to the other forms.

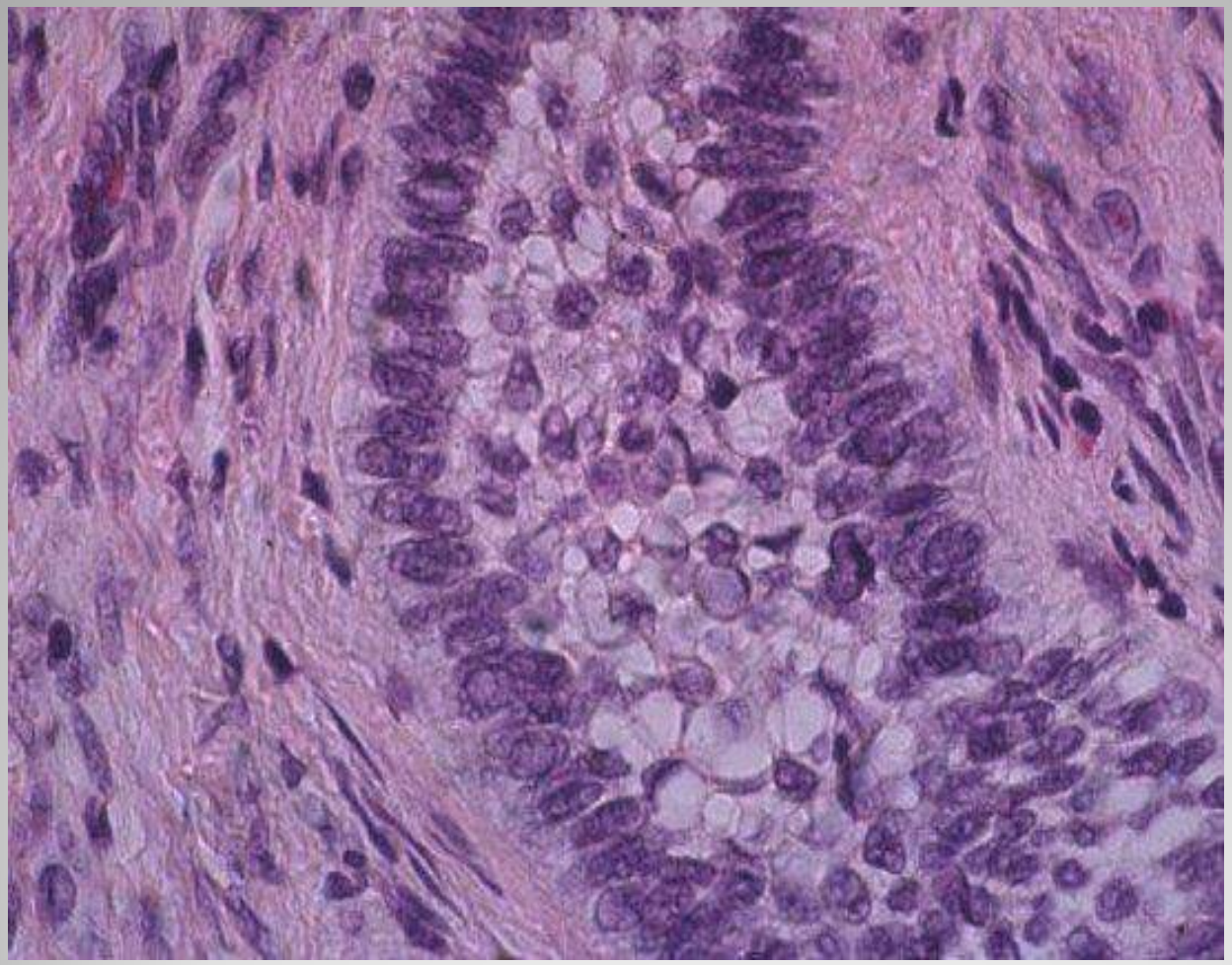


Granular cell Ameloblastoma



Basal Cell Ameloblastoma

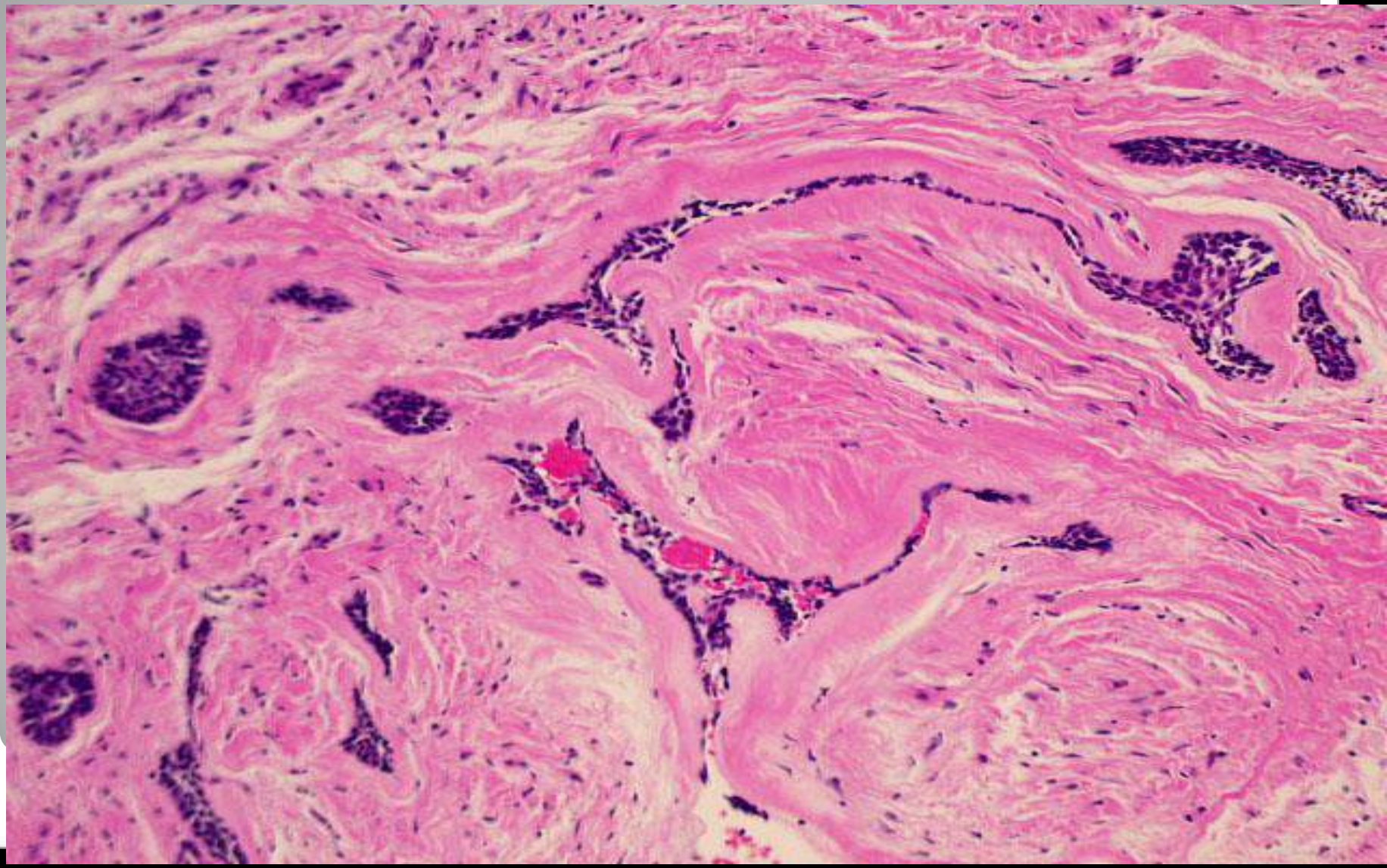
- The **basal cell type of ameloblastoma bears considerable** resemblance to the basal cell carcinoma of the skin.
- It is believed that this is the rarest histologic subtype and the epithelial tumor cells are more primitive and less columnar, are generally arranged in sheets, more so than in the other tumor types.



- Found in a dense collagen stroma that may appear hyalinized and hypocellular.
- It has a greater tendency to grow in thin strands and cords of epithelium rather than in an island like pattern.
- The epithelial proliferation almost seems to be compressed and fragmented by the dense hyalinized stroma.
- Central cells are often scant in the epithelial proliferation, and the cells making up the periphery of the strands and cords often are flattened or cuboidal rather than tall columnar in appearance.
- Reverse polarity of nuclei and subnuclear vacuole formation may be difficult to recognize.
- Ant region of the jaws
- More aggressive

DESMOPLASTIC AMELOBLASTOMA

Desmoplastic Ameloblastoma



- Unicystic type –approximately 6% of ameloblastomas.
- It tends to occur in a younger population (average age -22 years)
- A high percentage of these lesions are associated with an impacted tooth, and the most commonly cited provisional diagnosis is dentigerous cyst.
- OKC and Radicular cyst transforming into Ameloblastomas are also been described.
- unicystic type has a considerably better overall prognosis

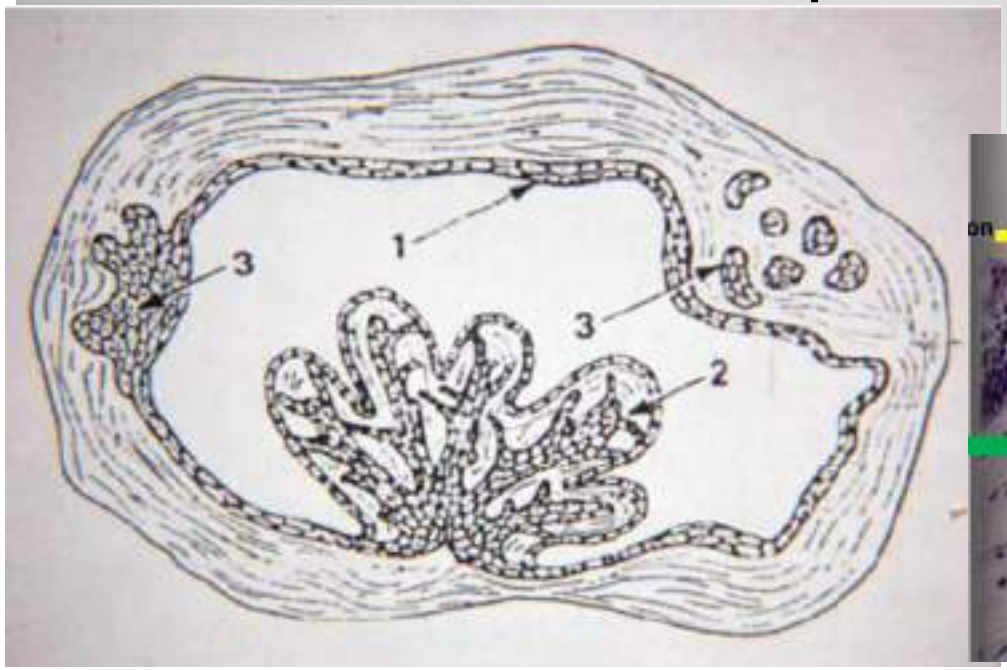
Unicystic Ameloblastoma

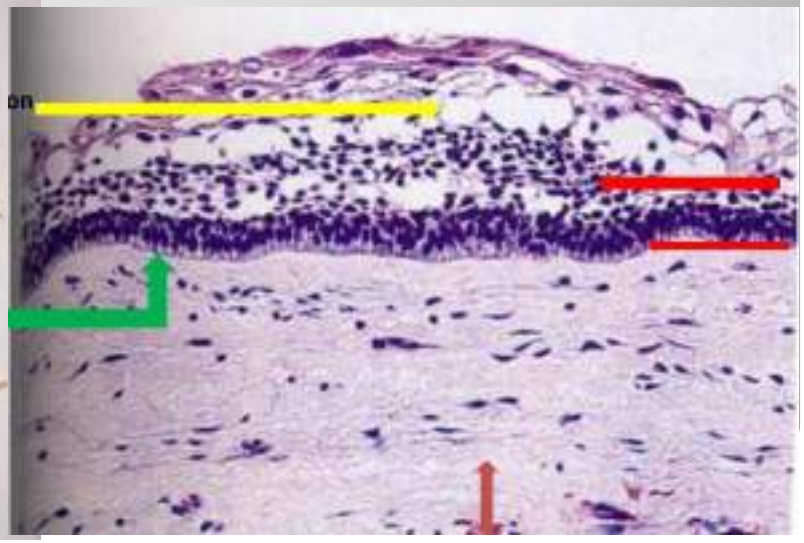
- Lining epithelium exhibiting alterations virtually identical with those described by Vickers and Gorlin as representing early ameloblastomatous changes in the dentigerous cyst
- Nodules of tumor projecting intraluminally.
- Ameloblastomatous lining epithelium proliferating into the connective tissue wall.
- Islands of ameloblastoma occurring isolated in the connective tissue wall

Unicystic Ameloblastoma

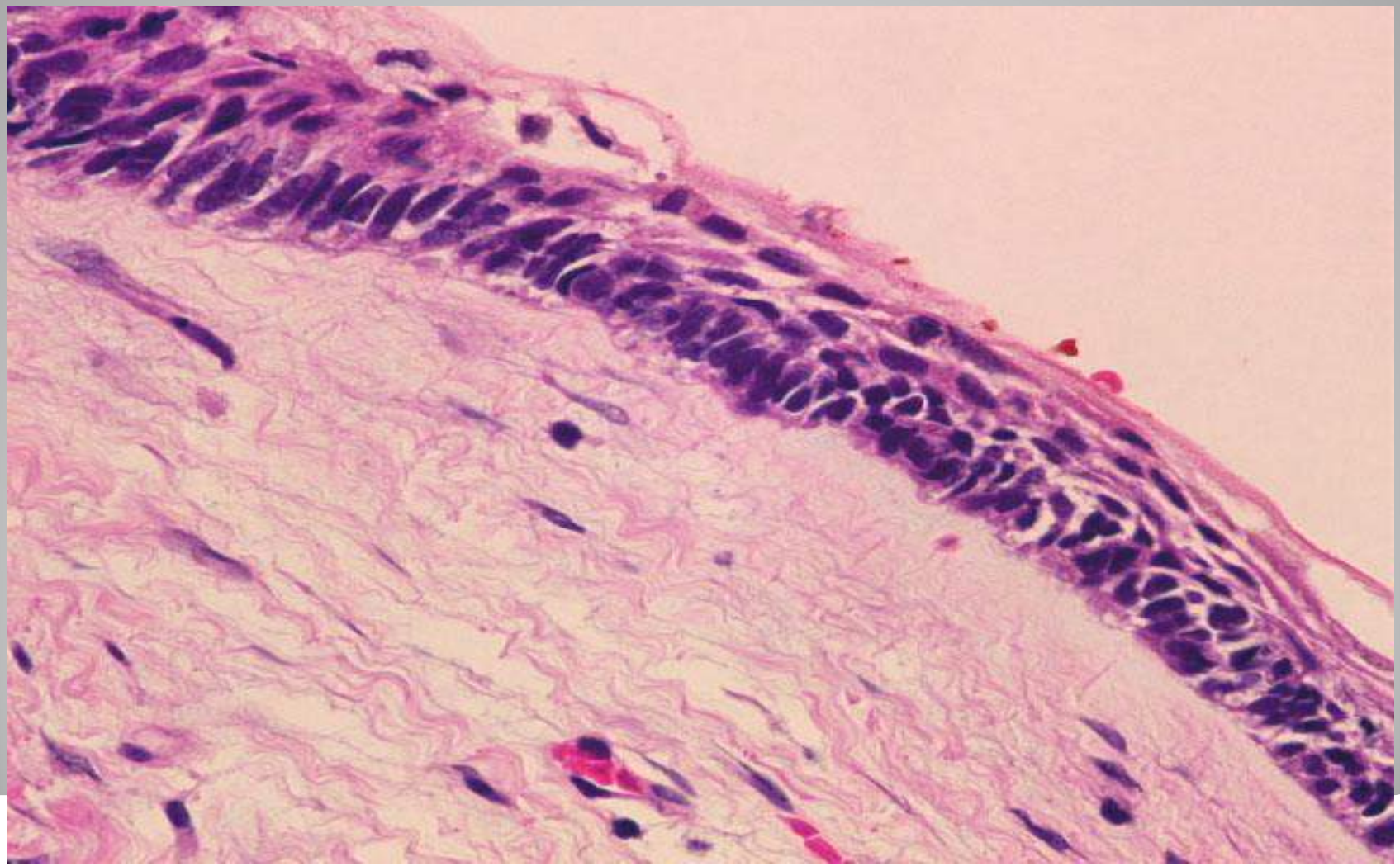
The luminal type - subgroup 1

- Defined as having an epithelial lining of which parts may show t ransformation to cuboida l or columnar basal cells with hyperchromatic nuclei, nuclear palisading with polarization , cytoplasmic vacuolization and subepithelial hyalinization.





Unicystic luminal Ameloblastoma



Subgroup Interpretation

1 - Luminal UA

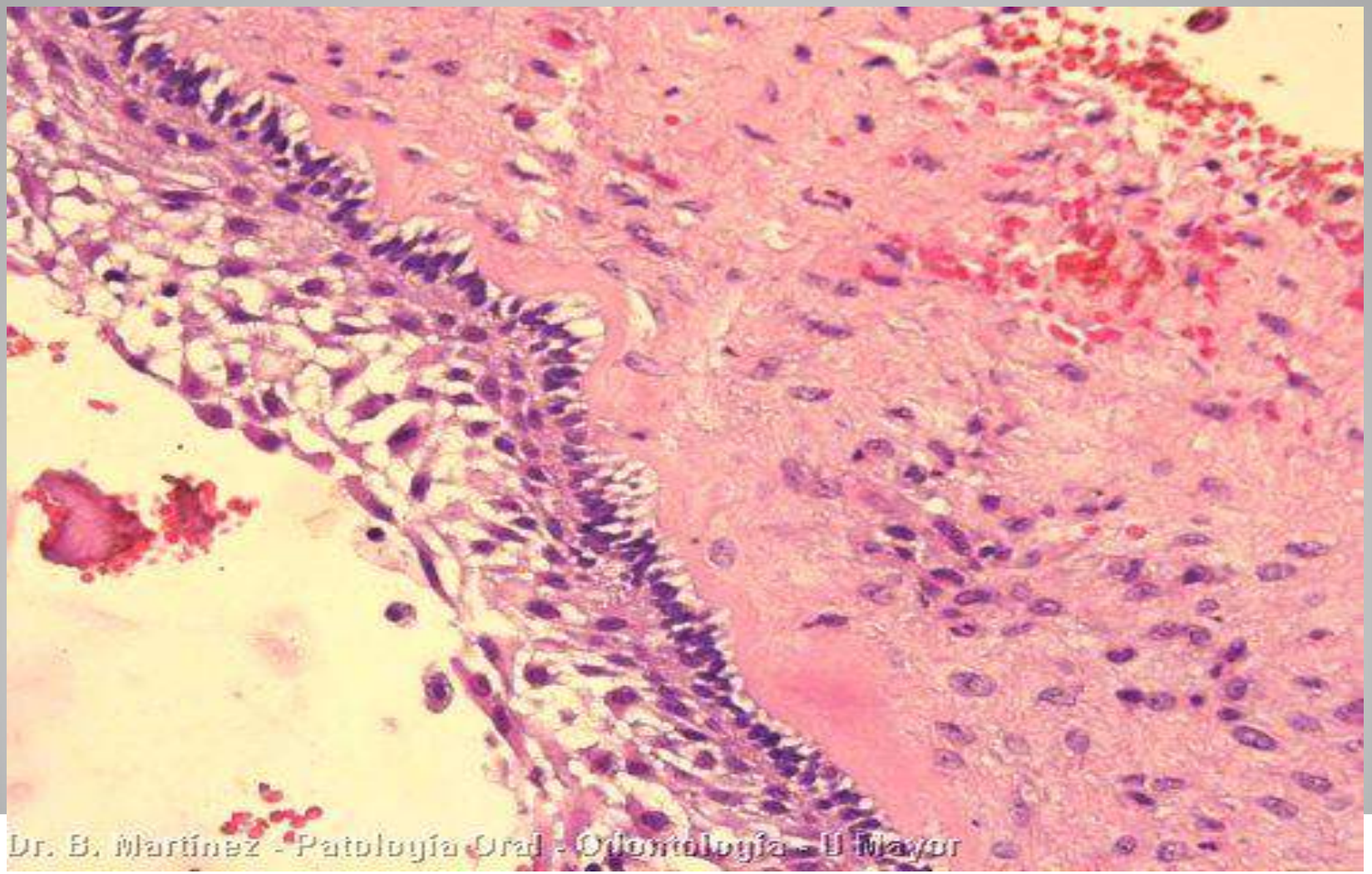
1.2 - Luminal and intraluminal UA

1.2,3 - Luminal, intraluminal , and intramural UA

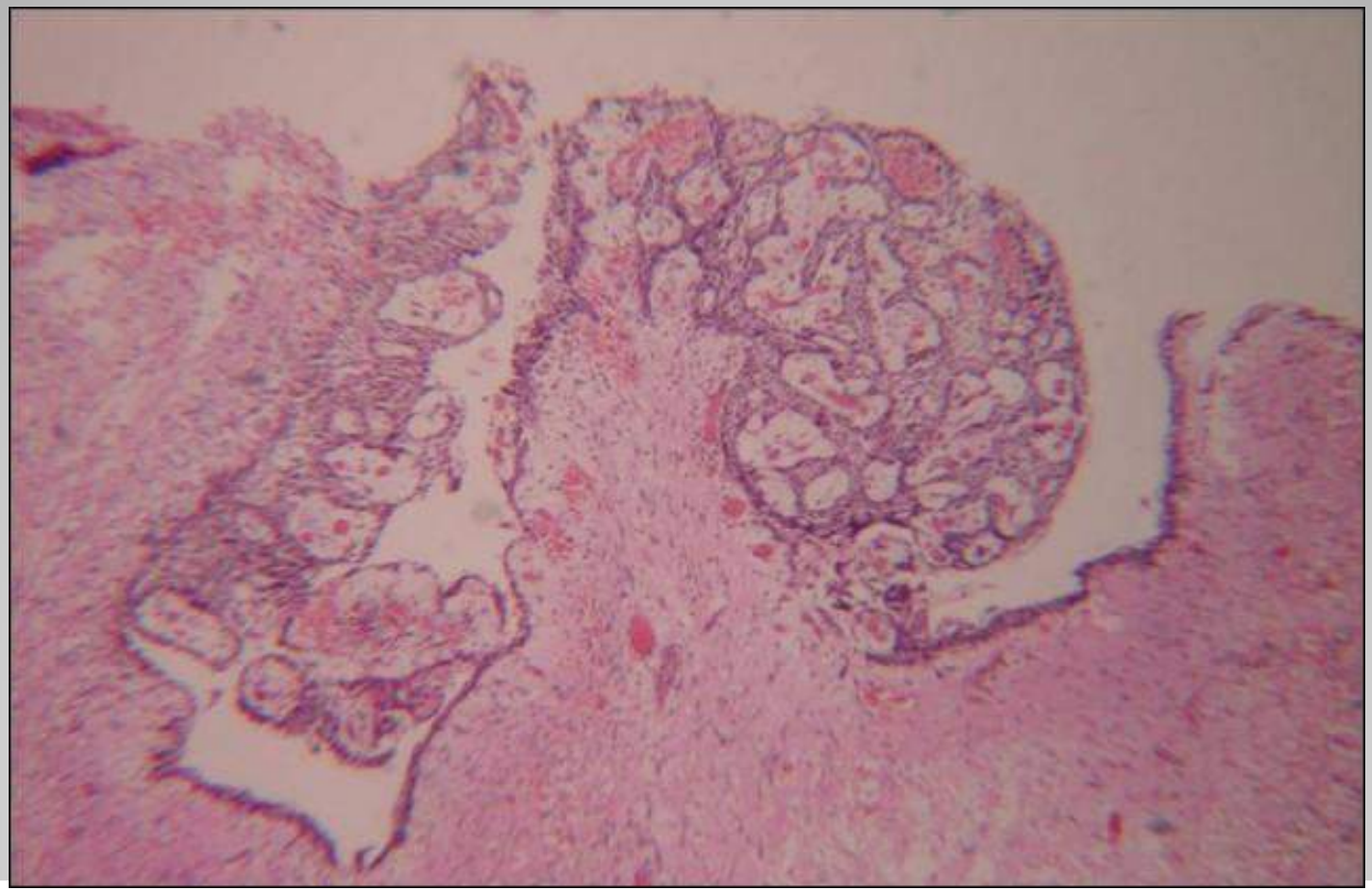
1.3 - Luminal and intramural UA

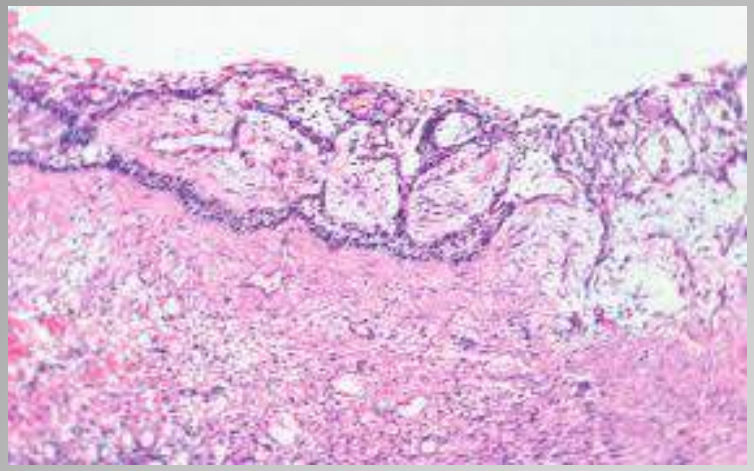
Unicystic Ameloblastoma

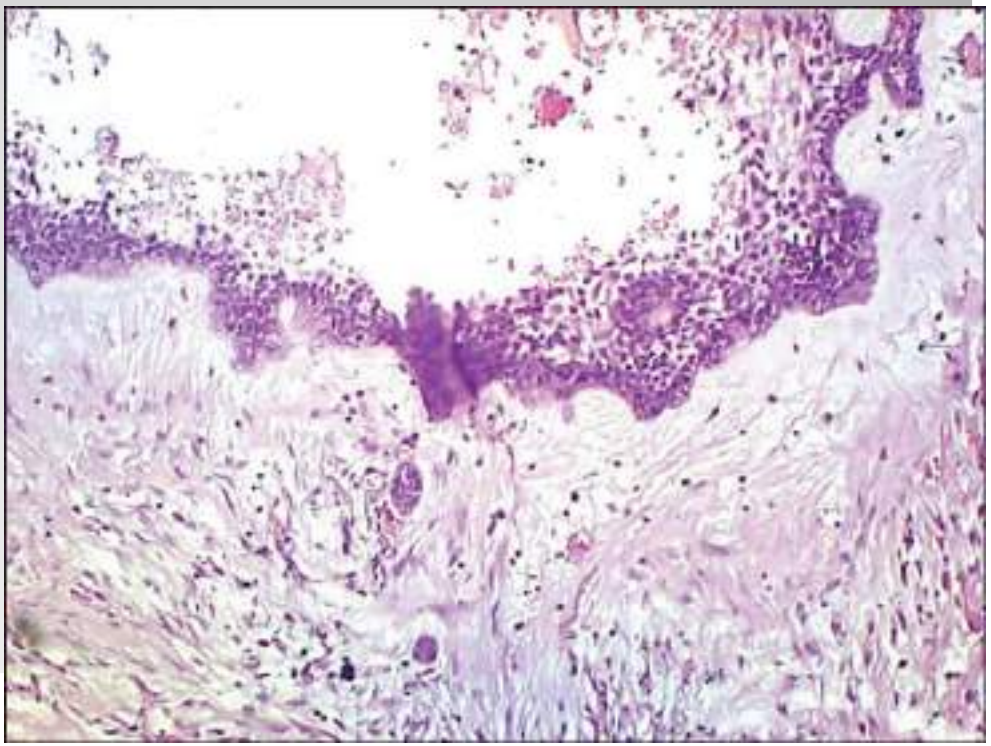
Unicystic luminal Ameloblastoma

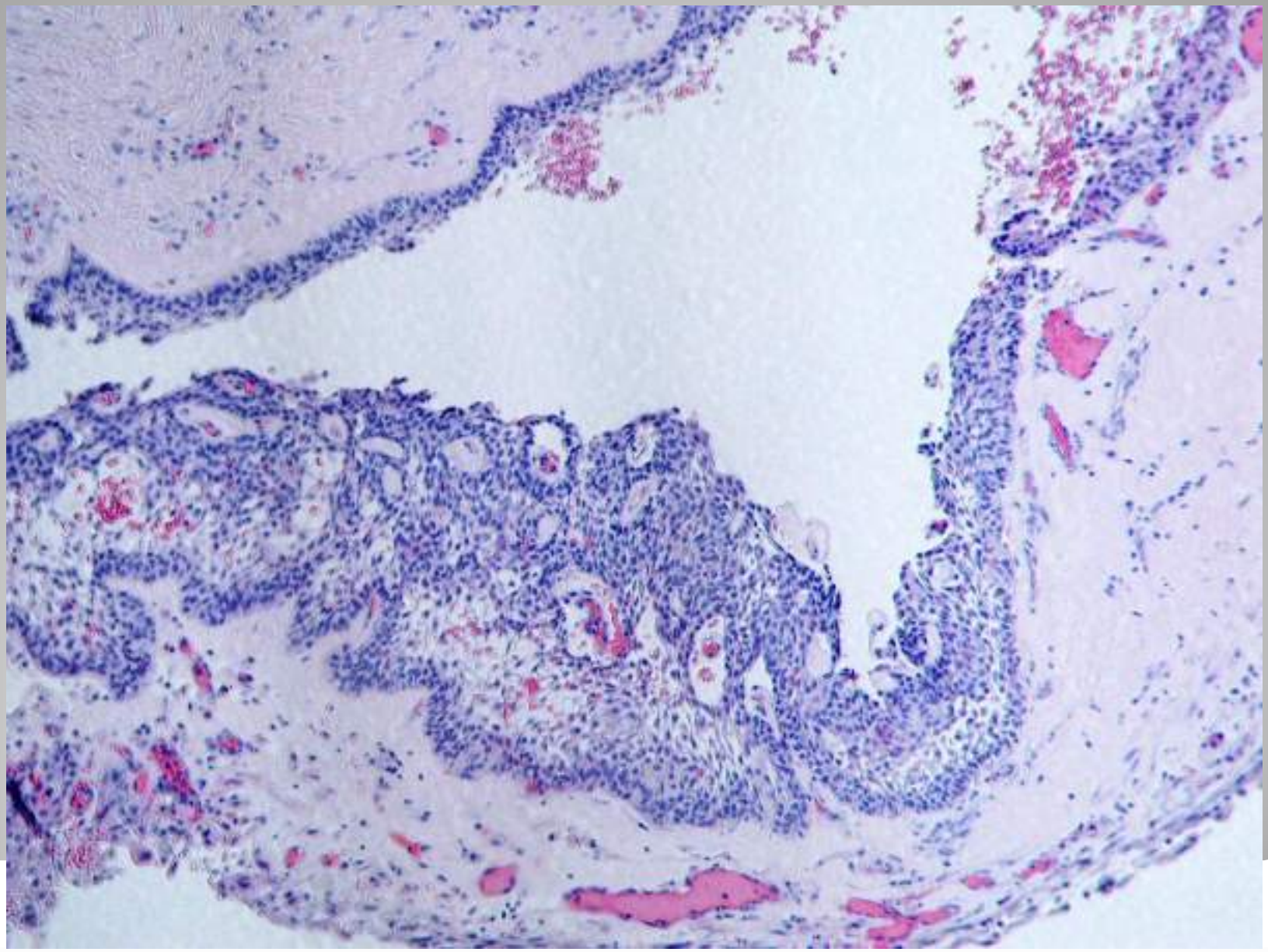


- subgroup 1,2 - luminal and intra luminal shows simple and intraluminal features.

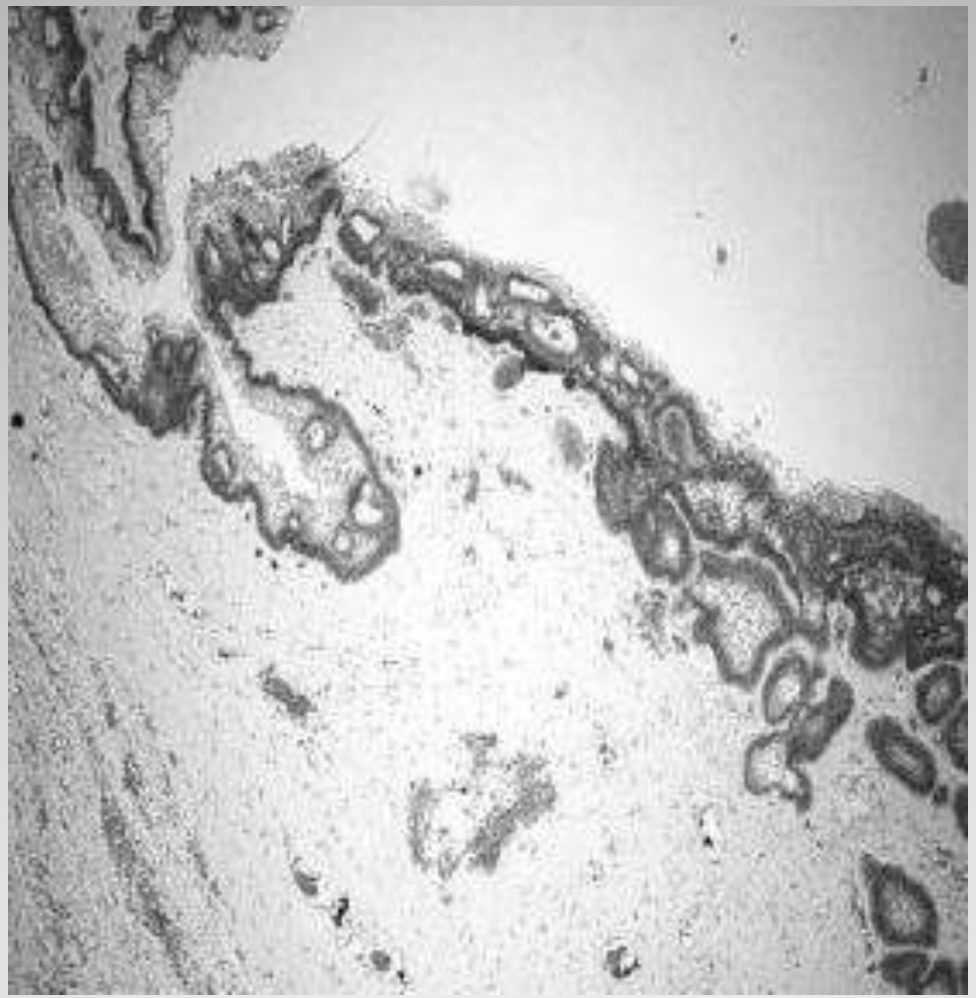


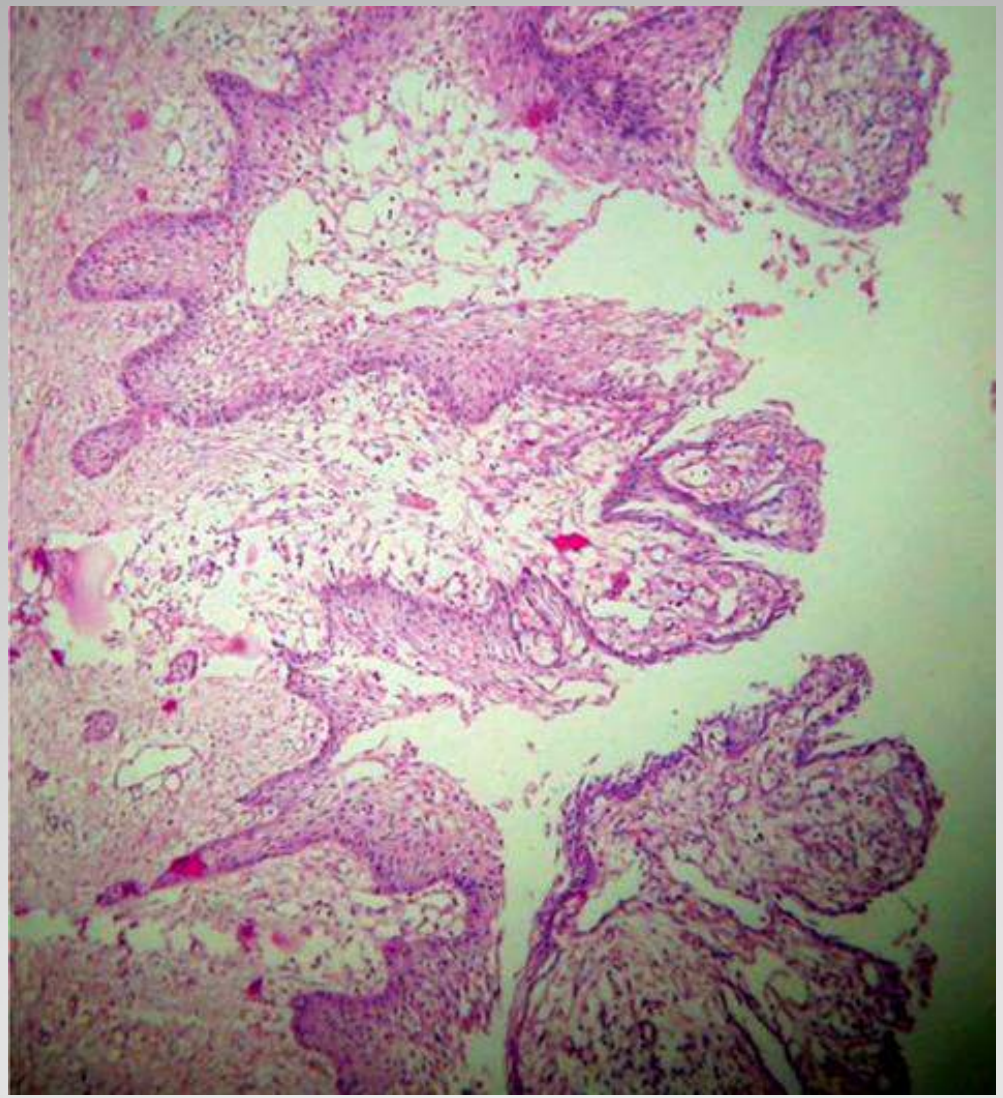






- 1.2,3 - Luminal, intraluminal , and intramural UA





- 1.3 Luminal and intra mural exhibits a cyst with a luminal lining in combination with intramural nodules of ameloblastomatous tissue.

Differential diagnosis

Other odontogenic tumors:

Ameloblastic fibroma,odontogenic myxoma.

Non odontogenic tumors:

Central giant cell granuloma,aneurysmal bone cyst.

Odontogenic cysts:

Dentigerous cyst,odontogenic keratocyst

Treatment

Treatment of ameloblastoma ranges from conservative curettage to radicular resection.

Treatment varies according to site,size&characteristics of the ameloblastoma

Curettage

En-block resection

Segmental resection

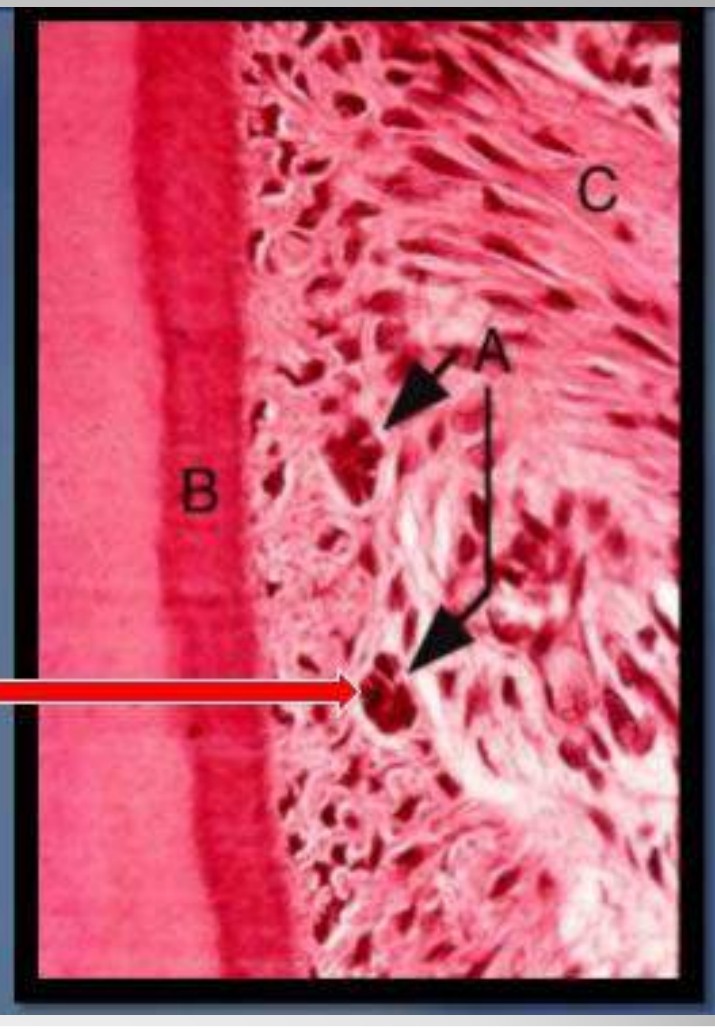
Definition

- Squamous odontogenic tumour (SOT) is a locally infiltrative neoplasm consisting of islands of well-differentiated squamous epithelium in a fibrous stroma.
- ICD-O code 9312/0
- The SOT is a rare tumour with less than 50 cases reported.
- Mean age - 38.7
- The gender ratio is 1.4:1 (men:women).
- Usually occurs intraosseously & develops in the periodontal ligament between the roots of vital erupted, permanent teeth.
- Mandible> Maxilla.

Squamous odontogenic tumour

- Etiology - UNKNOWN

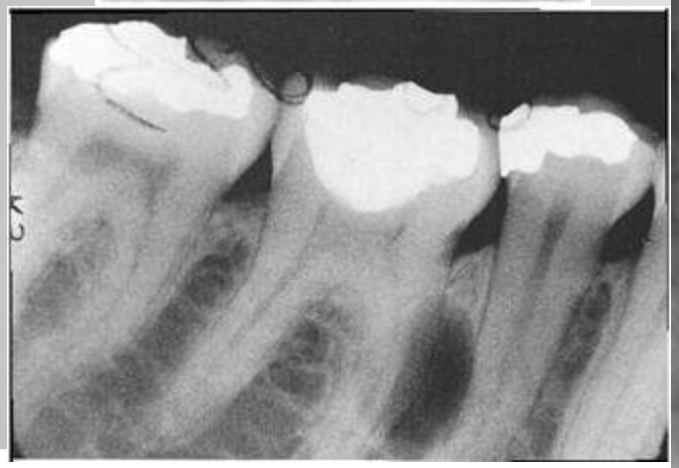
- Rare tumour, believed to be arise from neoplastic transformation of rest of malassiz



Rest of malassiz

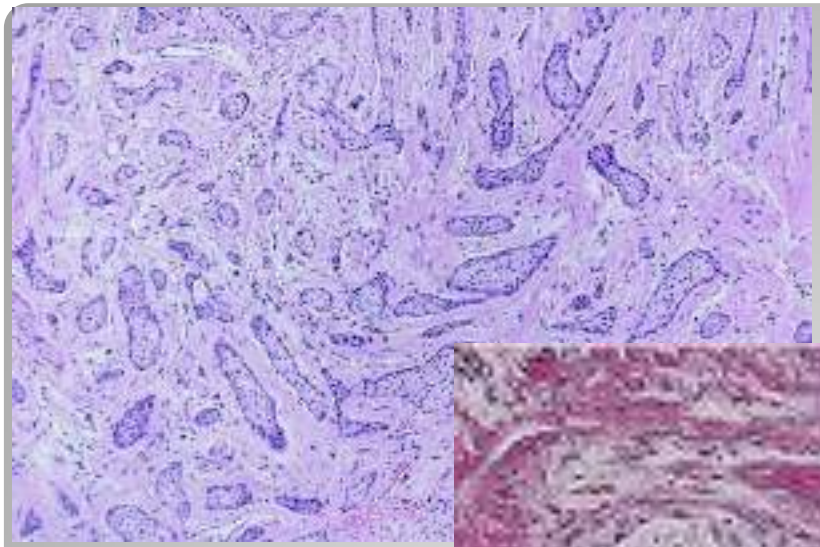
Clinical features / Imaging

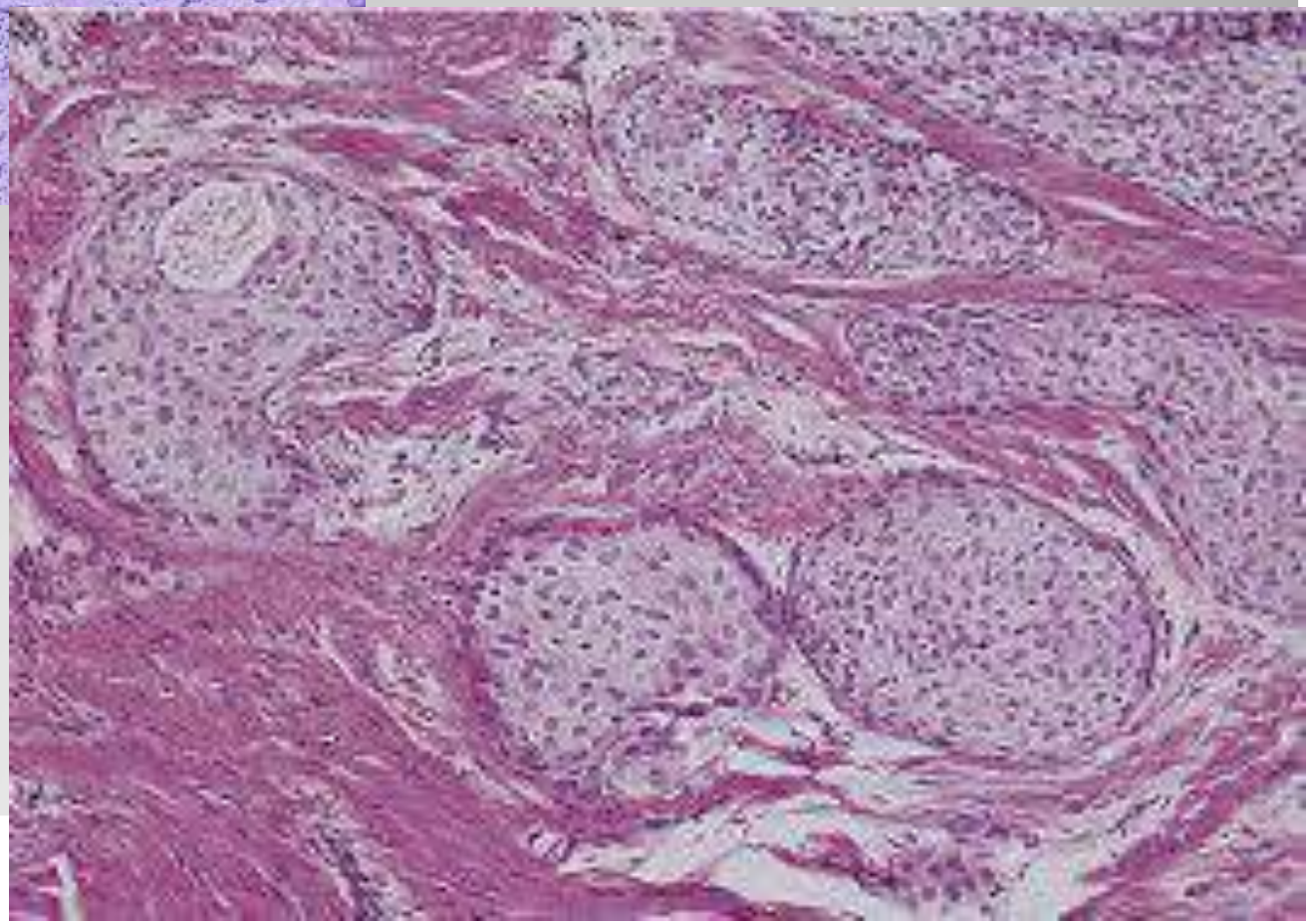
- Mobility of teeth, local pain, swelling of the gingiva, osseous expansion or mild gingival erythema.
- Radiographically, a unilocular or triangular radiolucency between the roots of adjacent teeth is seen.
- Extensive SOTs may present a multilocular pattern.
- The peripheral variant may produce some ‘saucerization’ of the underlying bone – a result of pressure from tumour



- SOTs are composed of islands of well differentiated squamous epithelium of varying size and shape.
- Islands are rounded or oval, occasionally, irregular and cordlike structures may be seen.
- Individual tumour islands reveal a peripheral layer of low cuboidal or flat epithelial cells.
- Epithelial islands may undergo central microcystic degeneration.
- Individual islands may contain calcified material.
- SOTs may be mistaken for an acanthomatous ameloblastoma, a desmoplastic ameloblastoma, a well-differentiated squamous cell carcinoma, or pseudoepitheliomatous hyperplasia.

Histopathology





- Conservative surgical treatment is usually sufficient.
- Recurrences are rare and are probably due to incomplete removal.

Prognosis and predictive factors

- Pindborg Tumour
- First described in 1956 by the late Dr Jens J Pindborg.

Definition

- CEOT is a locally invasive epithelial odontogenic neoplasm, characterized by the presence of amyloid material that may become calcified.
- **ICD-O code 9340/0**

Calcifying Epithelial Odontogenic Tumour (CEOT)

- The definite etiology of this neoplasm is still UNKNOWN
- Tumour cells may originate from the stratum intermedium layer of the enamel organ in tooth development.
- Some hypothesize that the Pindborg tumor arises from remnants of the primitive dental lamina found in the initial stage of odontogenesis, and these epithelial rests are the more likely true progenitor cell.

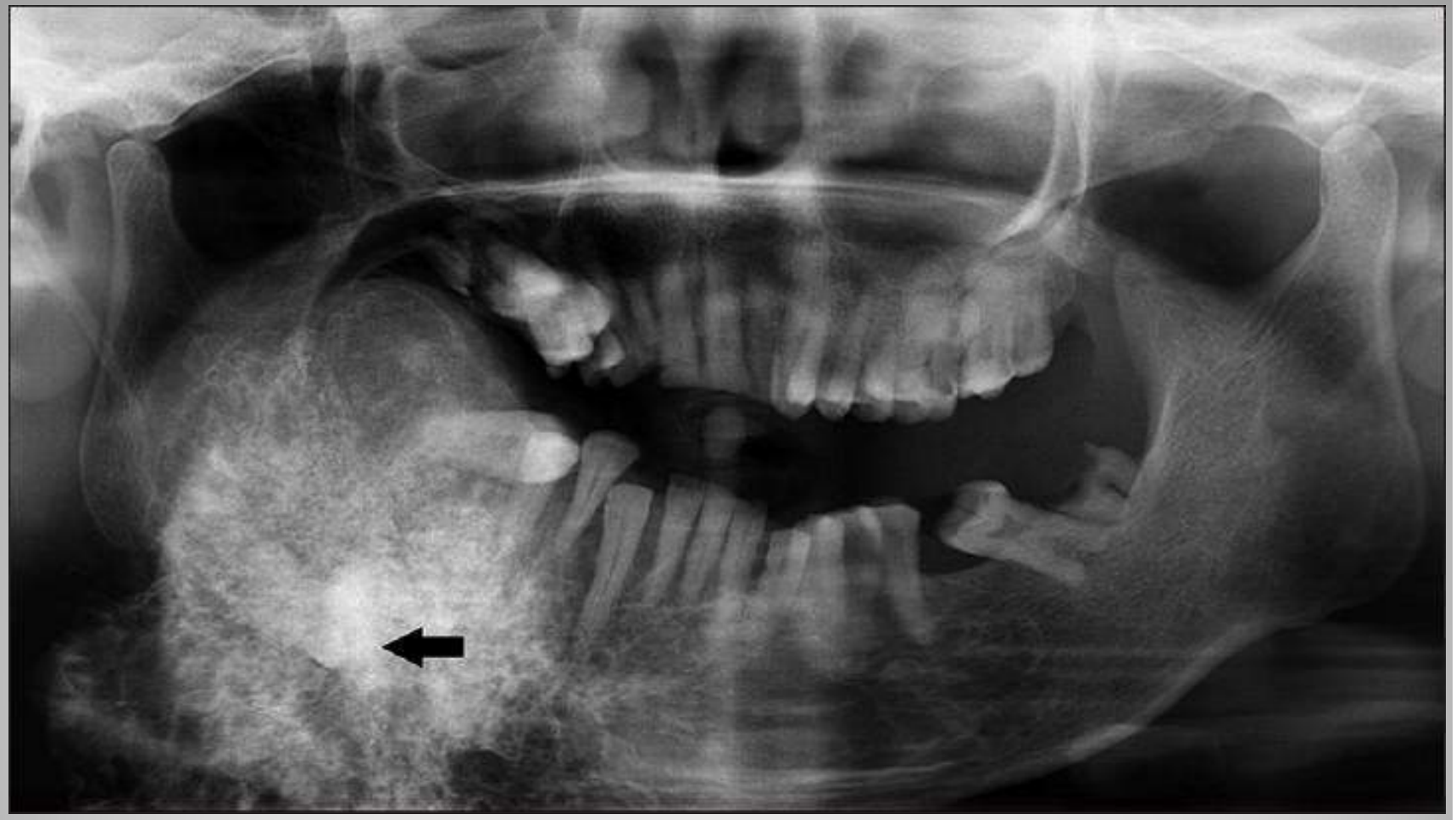
Histogenesis/Etiology

Epidemiology

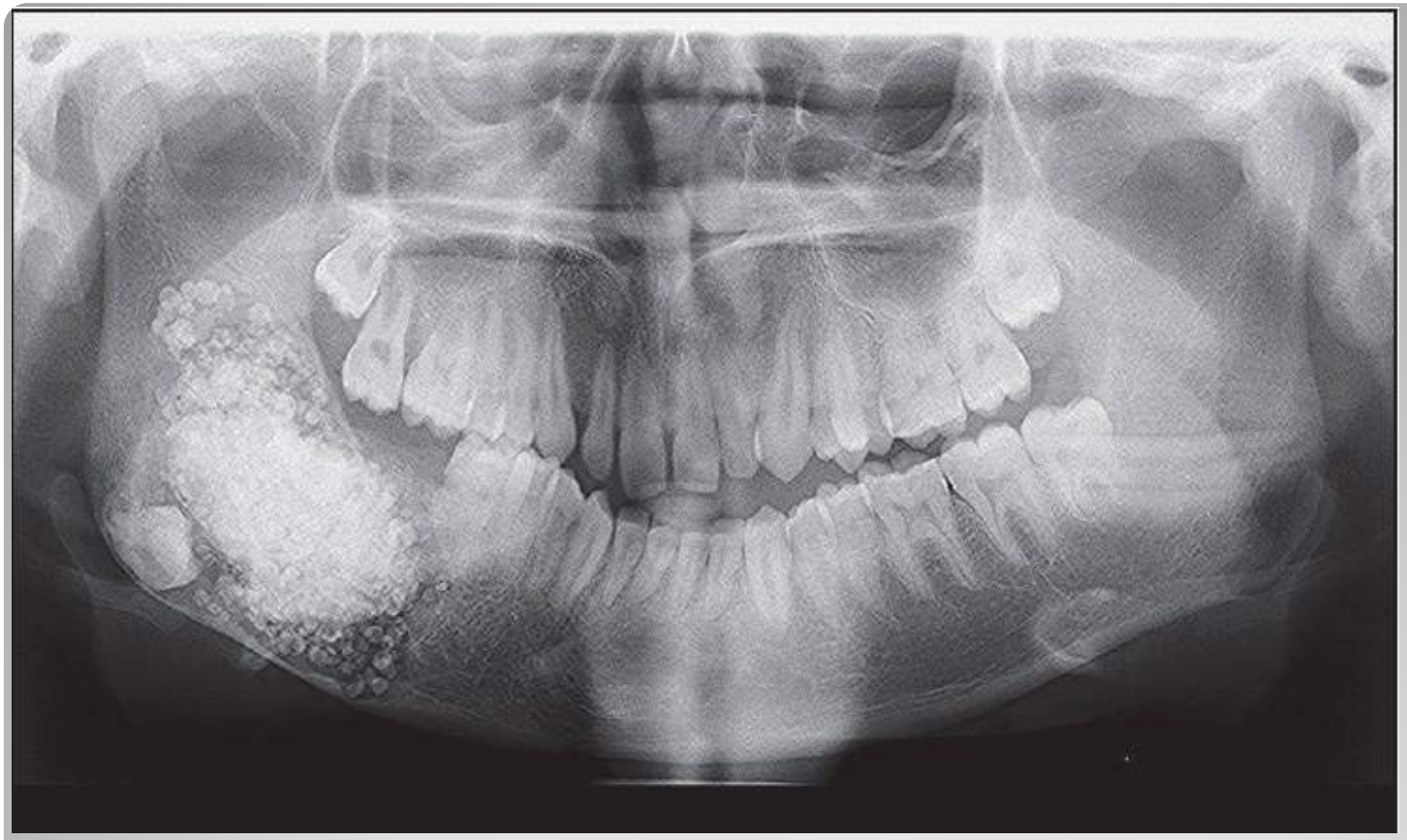
- CEOT accounts for approximately 1% of all odontogenic tumours
- Age -20 and 60 years - mean around 40 years
- There is no gender predilection.
- Most cases are intraosseous, approximately 6% arise in extraosseous locations.
- Mandible > Maxilla 2:1
- Premolar/molar region
- Peripheral lesions usually occur in the anterior gingiva.

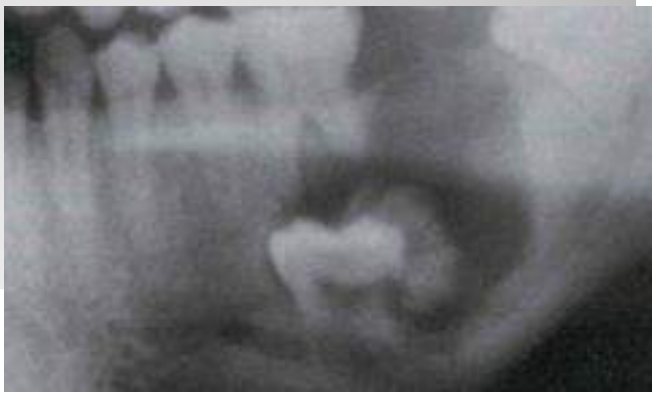
- The tumour presents as an asymptomatic slow-growing expansile mass of the jaw.
- Peripheral gingival lesions are firm painless masses.
- Radiographically, they may be unilocular or multilocular, most CEOTs present as mixed radiolucent- radiopaque lesions, but they may show considerable variation.
- Scattered flecks of calcification throughout the radiolucency have given rise to the descriptive term of a 'driven snow' appearance.
- In about half of the cases, an unerupted tooth, most often a mandibular third molar, is associated with the lesion.

Clinical features / Imaging



Driven Snow Appearance



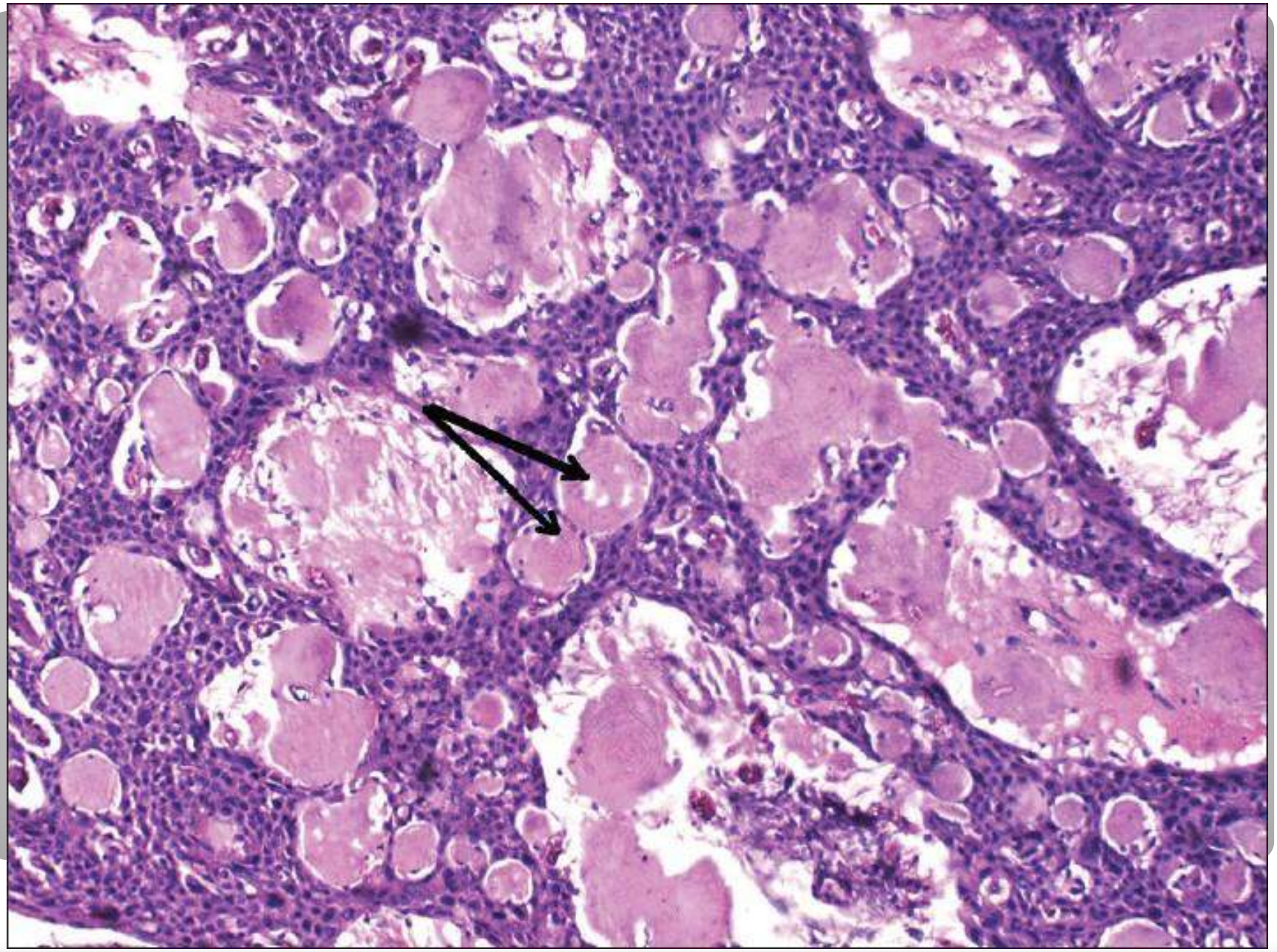


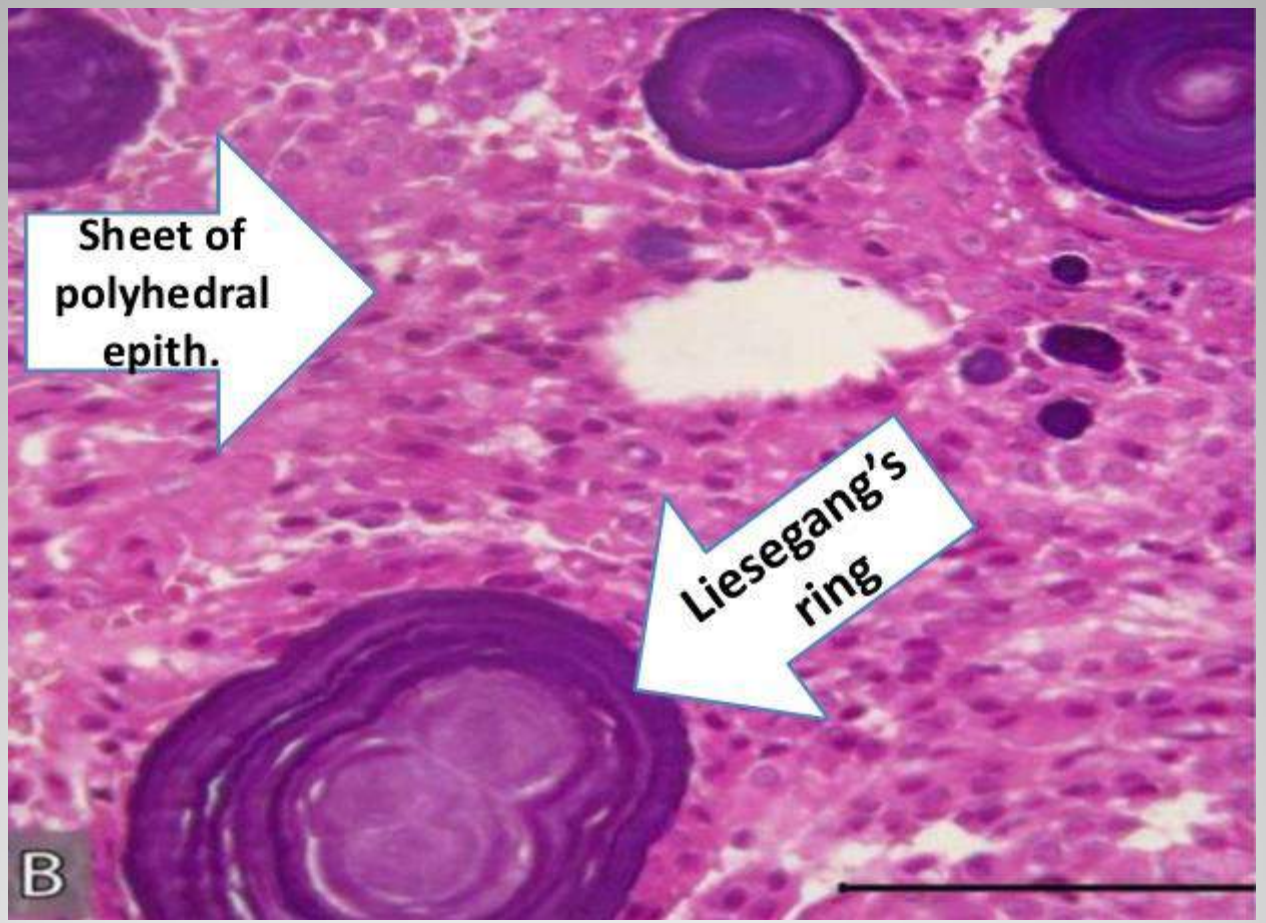
- The tumour consists of a fibrous stroma with islands and sheets of polyhedral epithelial cells with abundant eosinophilic granular cytoplasm, sharply defined cell borders and well-developed intercellular bridges.
- Their nuclei are frequently pleomorphic, with giant nuclei being common.
- Mitotic figures are rarely encountered; in case of malignant transformation, mitoses are frequent.
- Eosinophilic, homogeneous hyaline material that is often calcified in the form of concentric rings is present within or around the sheets of tumour cells.

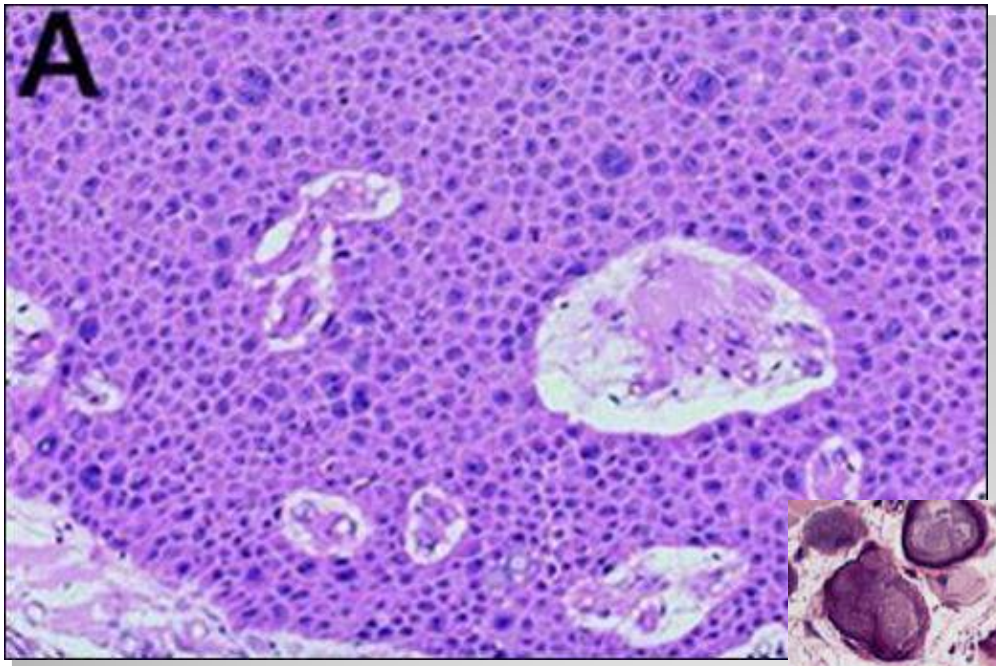
Histopathology

- Positive staining with Congo red and fluorescence with thioflavine T show this material to be amyloid.
- Apple green birefringence –polarized microscope
- Calcifications are characteristic concentric lamellar (Leise gang Rings), but non-calcifying variants also occur.
- Lack of calcification is more common in extraosseous tumours.
- Clear cells may be present within the epithelial nests; they contain glycogen.
- In some cases, the clear cells make up a significant proportion of the tumour – clear cell variant

Histopathology




Sheet of polyhedral epith.
Liesegang's ring
B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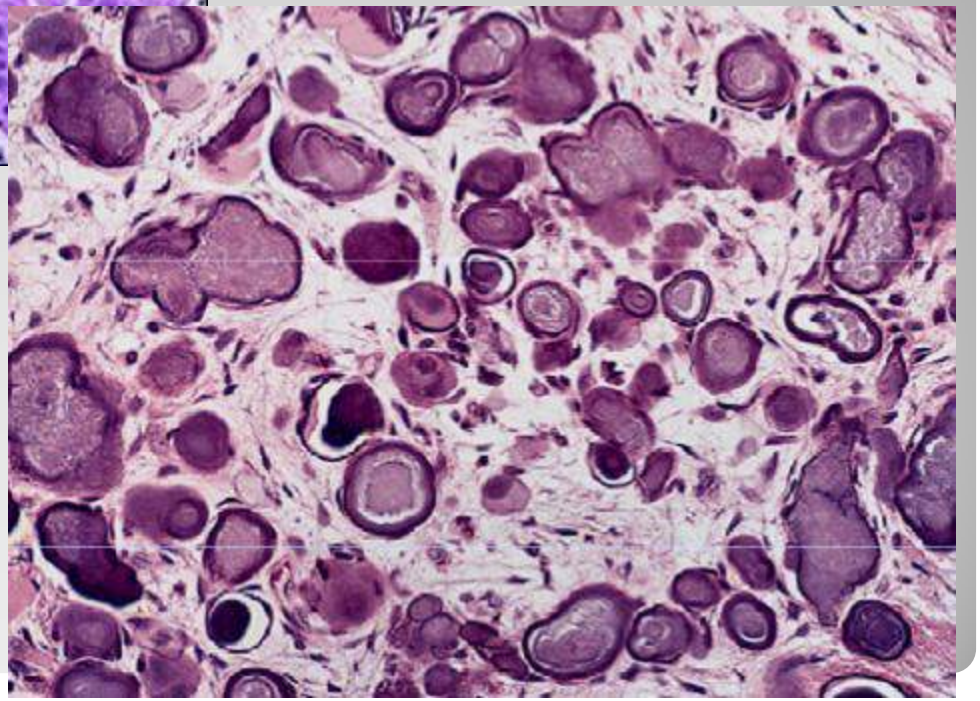
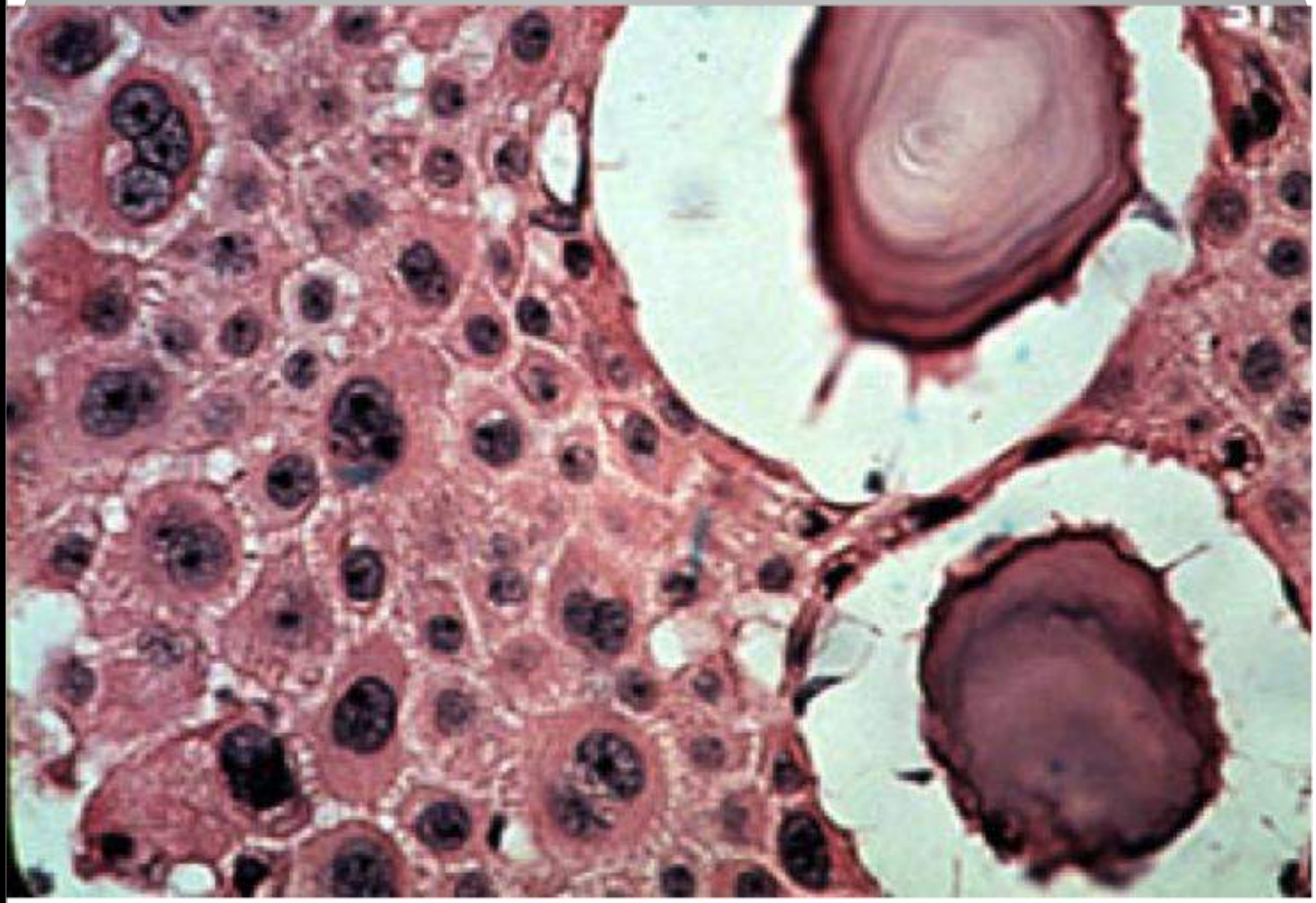
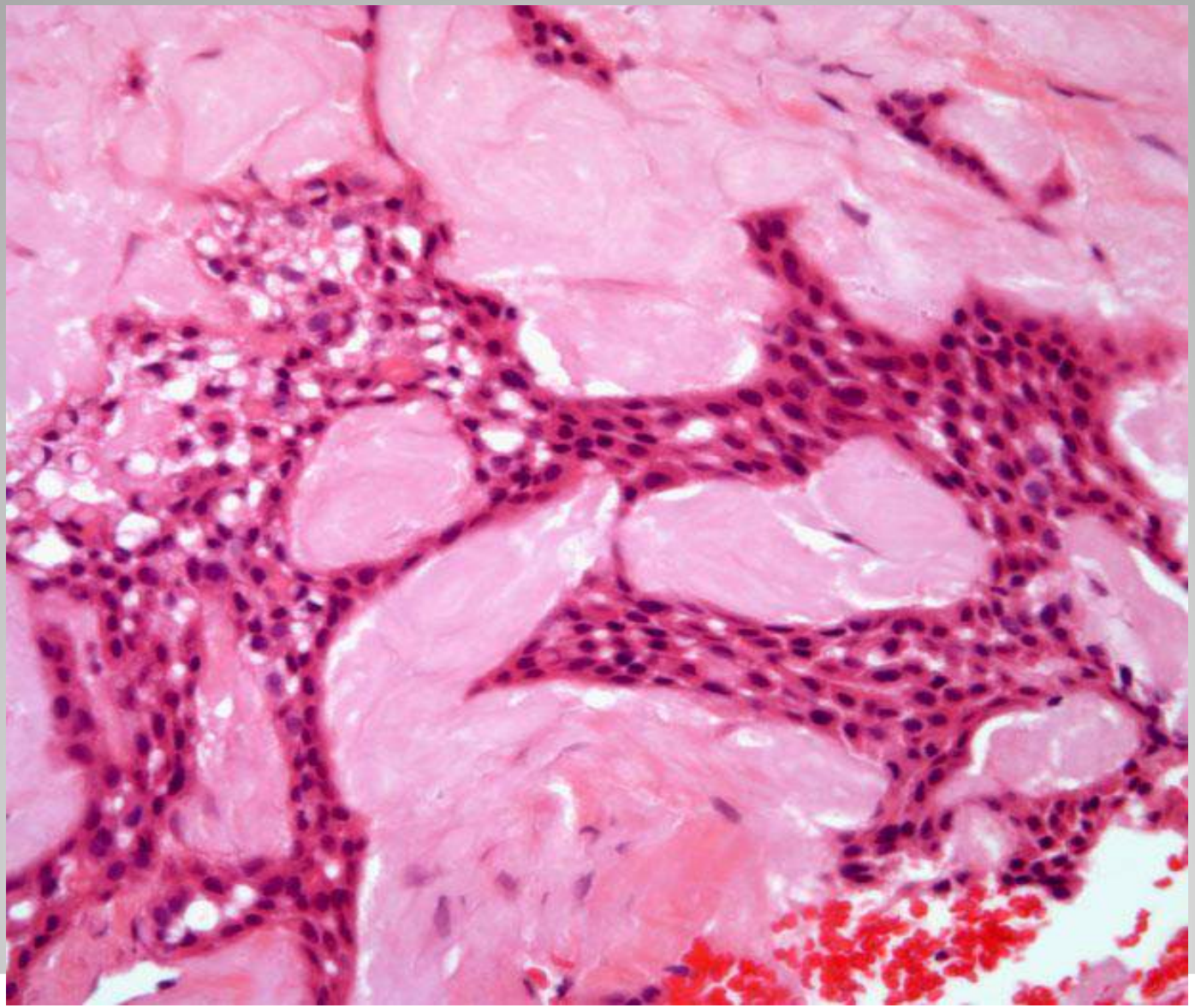
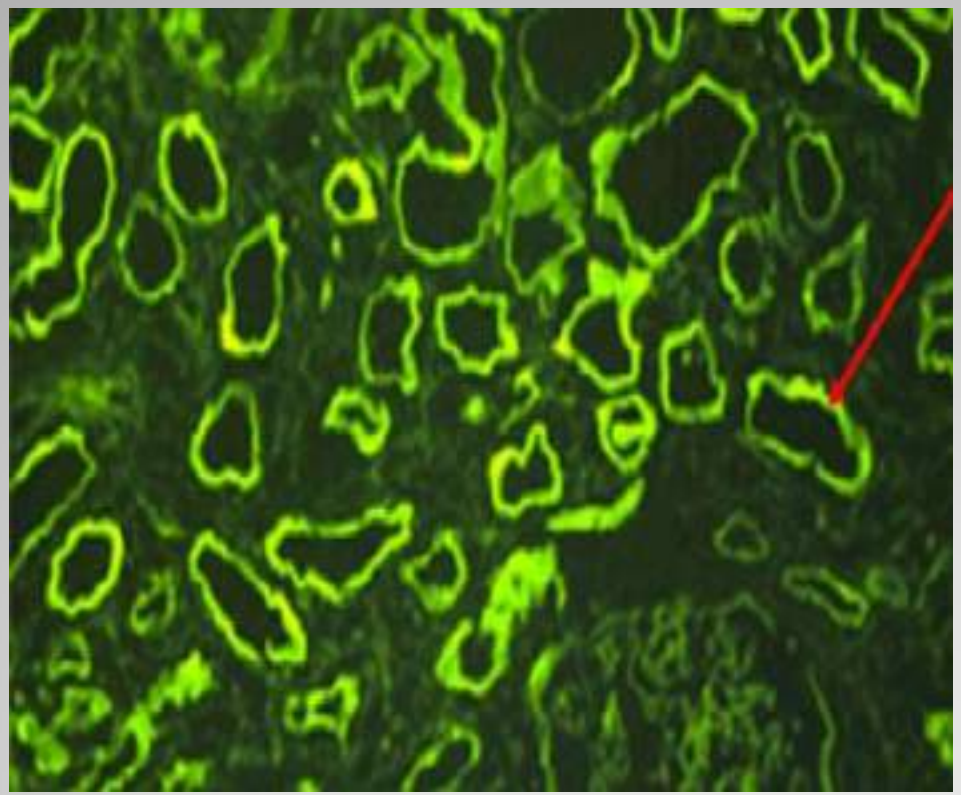








- Intra epithelial squamous cell carcinoma
- Clear cell carcinoma
- Muco-epidermoid carcinoma

Differential diagnosis

Treatment & Prognosis

- The CEOT is a locally invasive tumour. Small tumours may be enucleated, but larger ones require local resection.
- An overall recurrence rate of about 14% has been noted.
- A relatively higher recurrence rate of 22% has been noted for the clear cell variant.
- Longterm follow-up is recommended.

- AOT
- Adenomatoid odontogenic tumour is composed of odontogenic epithelium in a variety of histoarchitectural patterns, embedded in a mature connective tissue stroma and characterized by slow but progressive growth.
- **ICD-O code 9300/0**
- **2/3rd tumour**

2/3rd – females
2/3rd – maxilla
2/3rd – anterior
2.3rd – young adults

Adenomatoid Odontogenic tumour

Epidemiology

- Accounts for 2-7% of all odontogenic Tumours
- Age – Range 3 to 82 years, mean 2nd & 3rd decade
- Male : female ratio is 1:1.9, In Asian countries 1:3.2
- Almost exclusively occurs intraosseously with a preference for the maxilla over the mandible with a ratio of 2.1:1- maxillary cuspid region.
- The rare peripheral type occurs exclusively in the anterior maxillary gingiva.

- Specific stimulus that triggers proliferation of the progenitor cells of AOT is unknown.
- Because of its exclusive occurrence within the tooth-bearing areas of the jaws (most often associated closely with an unerupted or impacted tooth) and its cytologic resemblance to the dental lamina and components of the enamel organ, there is no disagreement that the AOT is of odontogenic origin (Rick GM, 2004).

Histogenesis

- Majority of the lesions measured between 1.5 and 3.0 cm,
- Obvious clinical swelling but asymptomatic
- High percentage of these lesions being associated with unerupted teeth and present as dentigerous cysts would strongly suggest that they are related to some late disturbance in odontogenesis.
- Since none of the associated teeth were described as morphologically defective, the disturbance must occur after odontogenesis is complete.

Clinical presentation

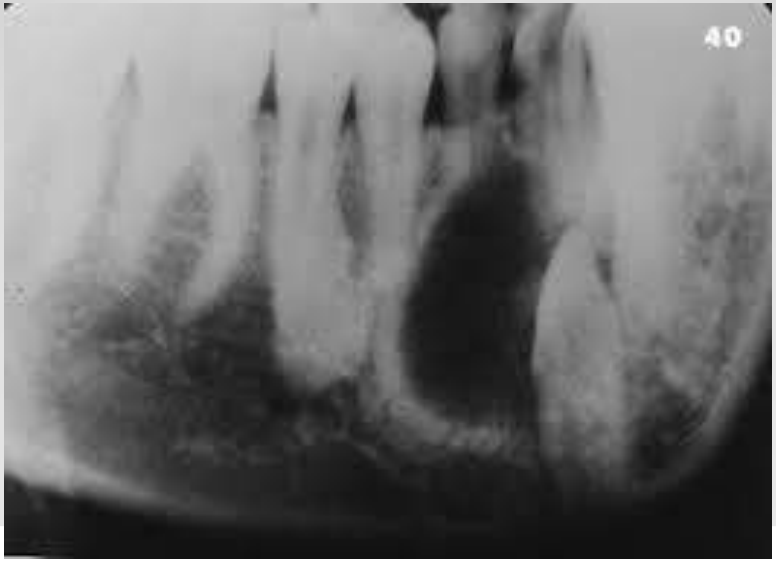
- Peripheral lesions present as a painless, gingival colored mass that ranges from 1–1.5 cm in diameter.
- Gingival lesions cannot be differentiated clinically from gingival fibromas, peripheral cemento-ossifying fibromas, peripheral giant cell lesions, or from other peripheral odontogenic tumors, such as odontogenic fibroma, ameloblastoma, calcifying odontogenic cyst, and calcifying epithelial odontogenic tumor.

Clinical presentation

Radiographic features

- Both follicular and extra follicular AOT, shows a well-defined, unilocular radiolucency around the crown of an unerupted permanent tooth, mimicking a dentigerous cyst.
- Sometimes mixed raadiolucency, showing small radioopeque foci
- The peripheral variant may disclose erosion (saucerization) of the alveolar bone crest.

R
L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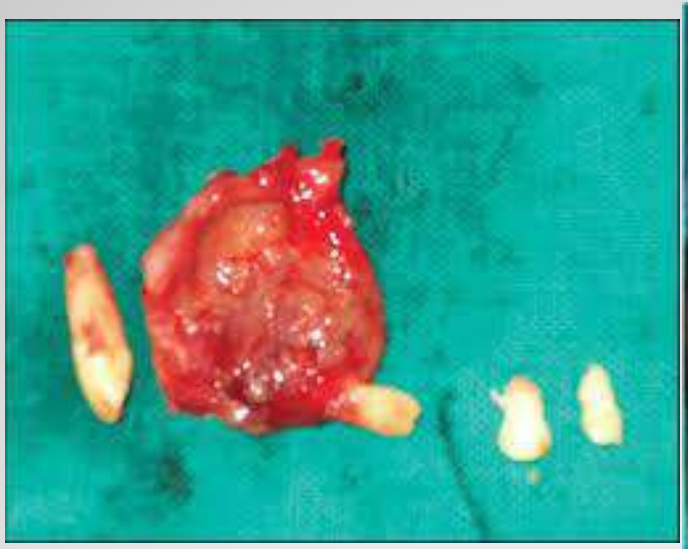
Macroscopic features

- Soft, roughly spherical mass with a distinct fibrous capsule.
- Upon gross sectioning, the tumour may exhibit white to tan, solid to crumbly tissue.
- Sometimes one or more cystic spaces of varying sizes with yellowish brown fluid or semisolid material; fine, hard 'gritty' granular material; and one to several larger calcified masses.
- Additionally, intact specimens demonstrate the crown

Histopathology

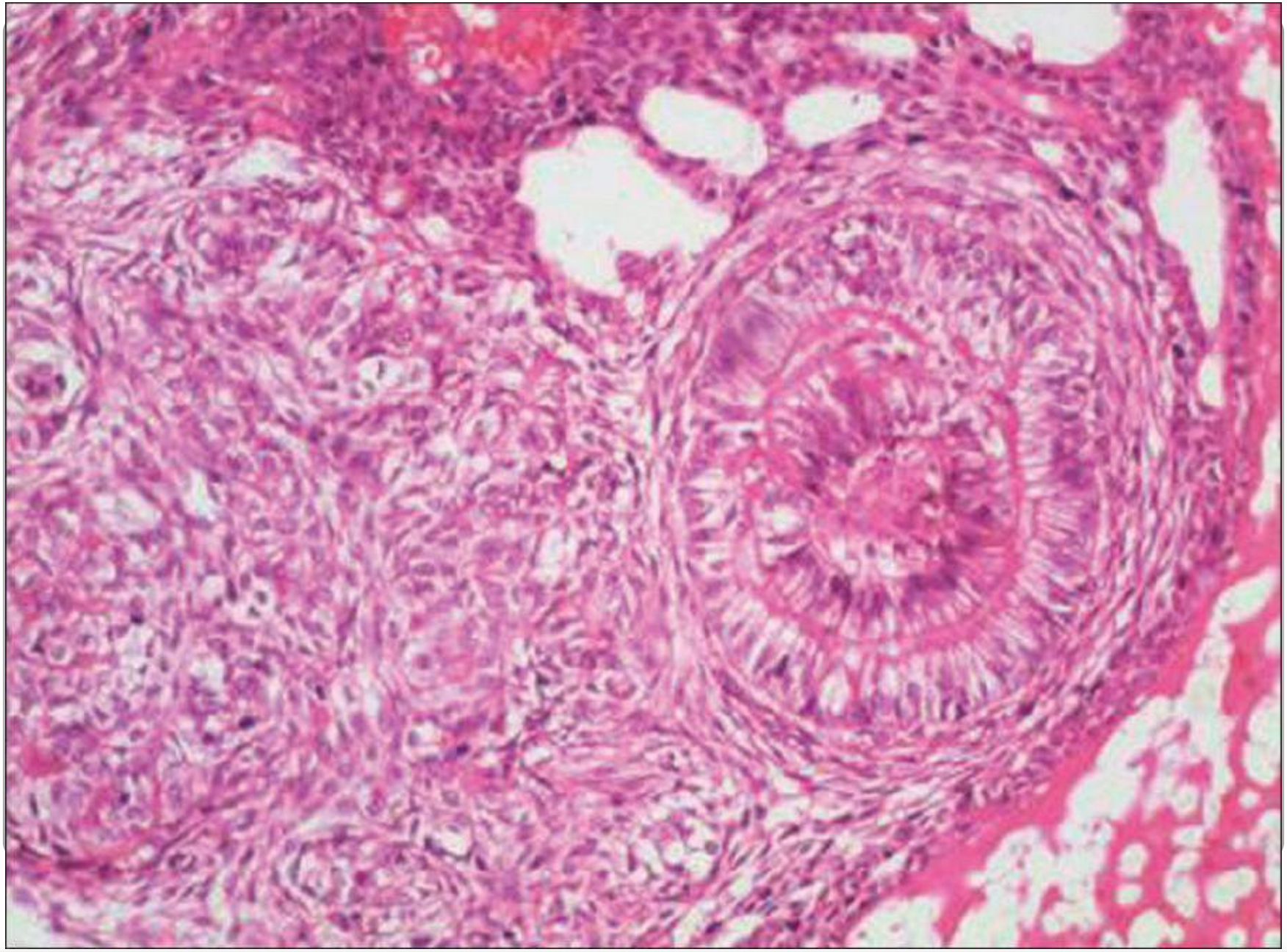


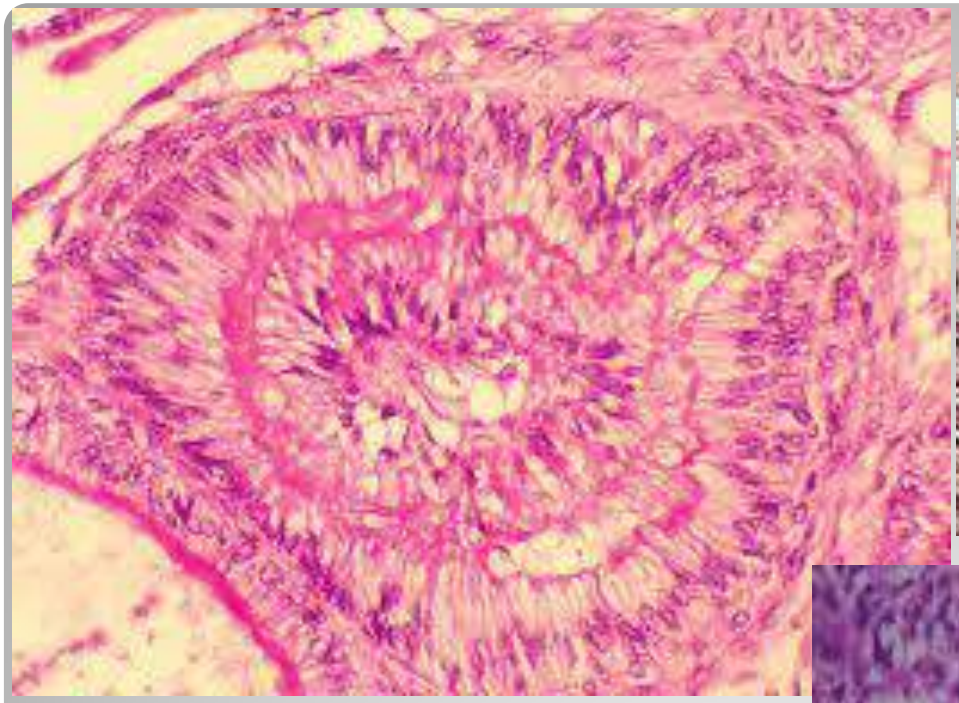
- AOT exhibits diverse histomorphologic features
- low magnification the most striking pattern is that of variably sized solid nodules of cuboidal or columnar cells of odontogenic epithelium forming nests or ROSETTE-LIKE structures with minimal stromal connective tissue. (Multiple layer of odontogenic cells)
- Between the epithelial cells and in the centre of the rosette-like configurations, eosinophilic amorphous material ("tumour droplets") is present.
- Small amounts of eosinophilic material or calcifications also may be present between these cells.

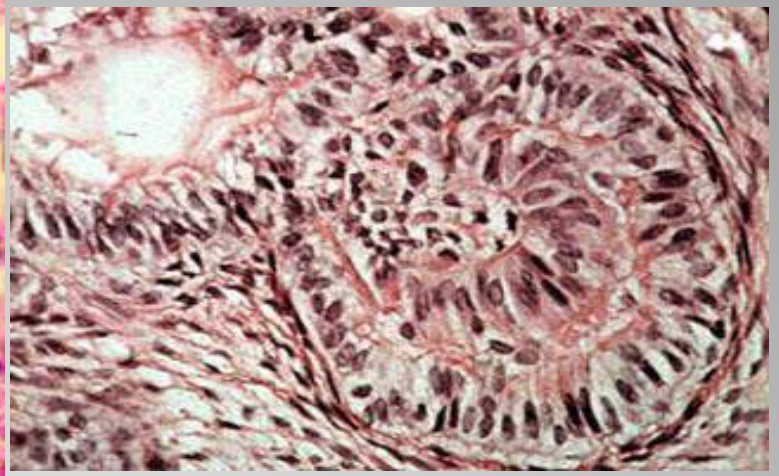
Histopathology

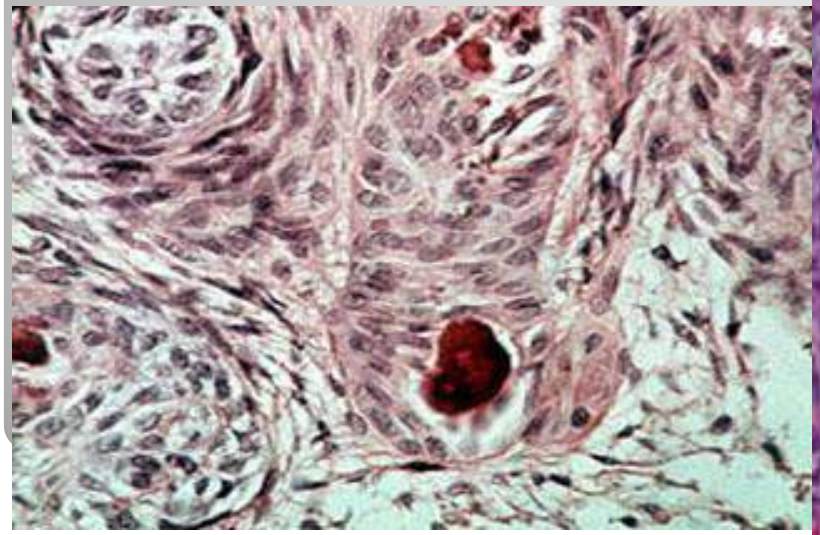
Histopathology

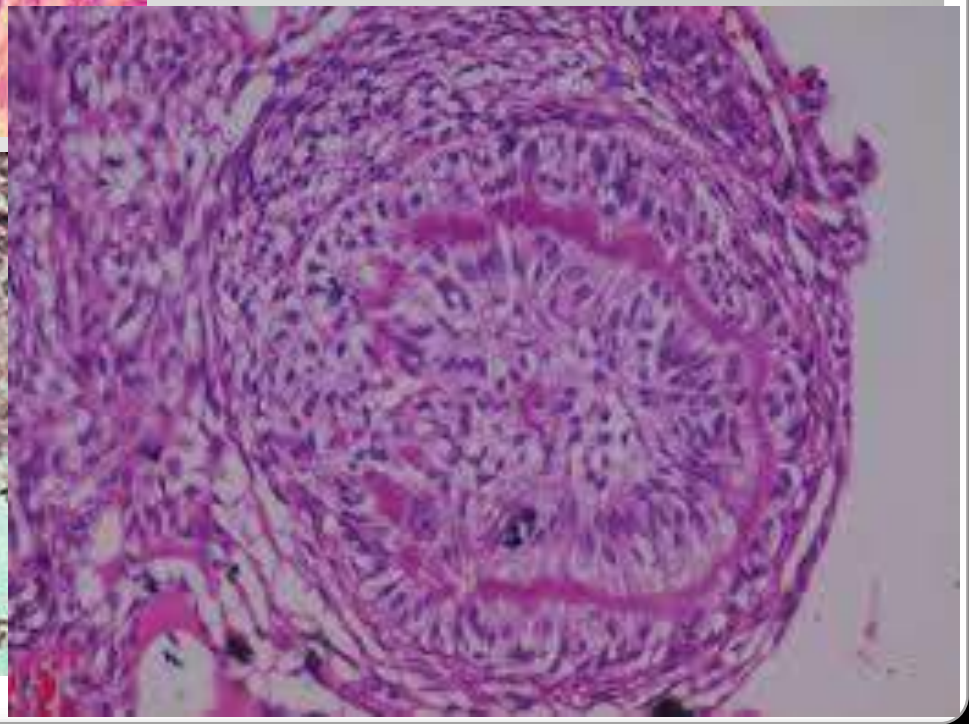
- Cellular areas demonstrates tubular or duct-like appearance (convoluted pattern).
- The duct-like spaces are lined by a single row of columnar epithelial cells, with the nuclei polarized away from the luminal surface.
- The duct-like spaces represent pseudolumina formed by secretion of the columnar epithelial cells.
- The lumen may be empty or contain eosinophilic material or cellular debris.
- These duct like or microcyst lumina frequently are lined by an eosinophilic rim of varying thickness (the so-called **hyaline ring**).

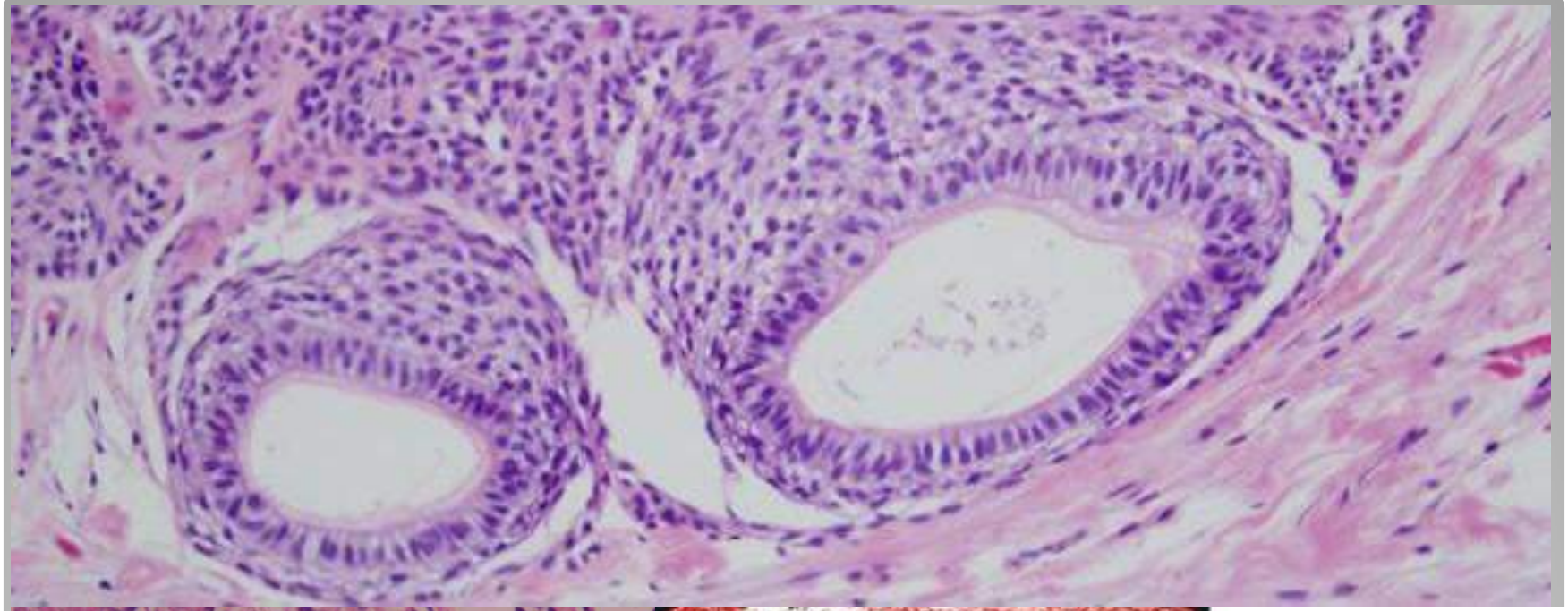


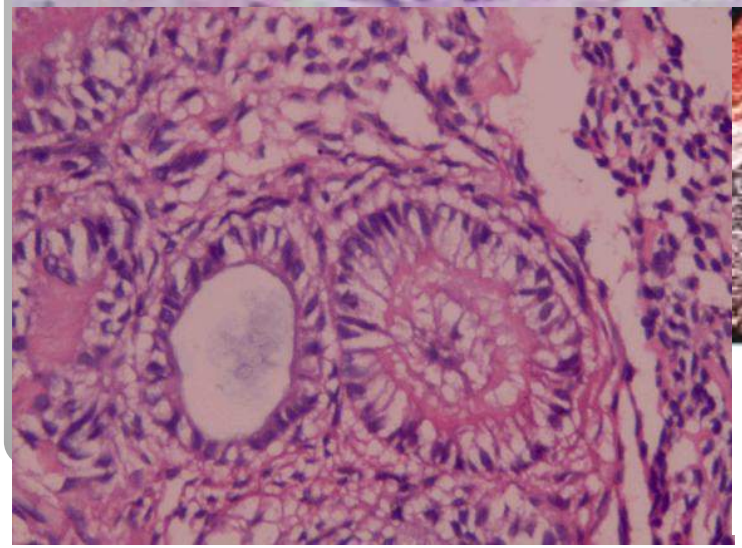


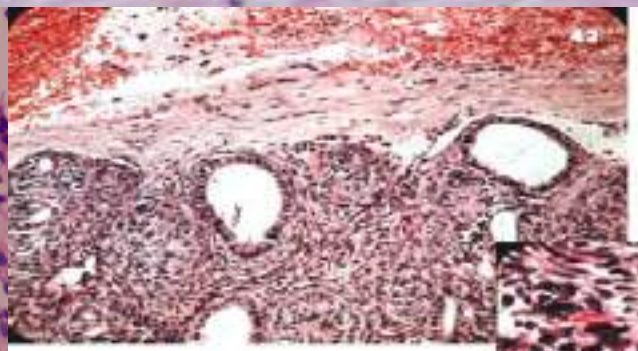




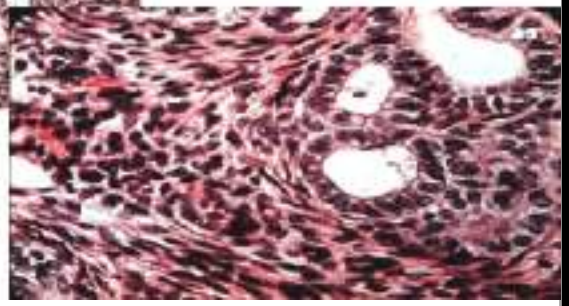








AOT



Definition

- A benign uni- or multicystic, intraosseous tumour of odontogenic origin, with a characteristic lining of parakeratinized stratified squamous epithelium and potential aggressive, infiltrative behaviour. It may be solitary or multiple. The latter is usually one of the stigmata of the inherited naevoid basal cell carcinoma syndrome (NBCCS).
- **ICD-O code 9270/0**
- The traditional designation is odontogenic keratocyst (OKC), which stresses the benign behaviour of this lesion.
- However, the WHO Working Group recommends the term keratocystic odontogenic tumour (KCOT) as it better reflects its neoplastic nature.
- Other synonyms include odontogenic keratocystoma and primordial cyst.

Keratocystic odontogenic tumour

- It is generally agreed that KCOT arises from odontogenic epithelium. The available evidence points to two main sources of the epithelium: the dental lamina or its remnants and extensions of basal cells from the overlying oral epithelium.

Genetics

- The NBCCS or PTCH gene has been mapped to chromosome 9q22.3-q31 & probably functions as a tumour suppressor.
- Studies on NBCCS and sporadic KCOT have provided molecular evidence of a two-hit mechanism in the pathogenesis of these tumours demonstrating allelic loss, at two or more loci, of 9q22 leading to over expression of bcl-1 and TP53 in the NBCCS.
- This supports the concept that KCOT represents a neoplasm
- There is also accumulating evidence that the PTCH gene might be a significant factor in the development of sporadic KCOT
- Furthermore, preliminary investigations suggested over-expression and amplification of genes located in 12q.

Histogenesis

Odontogenic epithelium with odontogenic ectomesenchyme, with or without dental hard tissue formation

Ameloblastic fibroma
Ameloblastic fibrodentinoma
Ameloblastic fibro-odontoma
Odontoma, complex type
Odontoma, compound type
Odontoameloblastoma
Calcifying cystic odontogenic tumour
Dentinogenic ghost cell tumour

- Ameloblastic fibroma (AF) consists of odontogenic ectomesenchyme resembling the dental papilla and epithelial strands and nests resembling dental lamina and enamel organ. No dental hard tissues are present. If there is dentin formation, the lesion is referred to as ameloblastic fibro dentinoma (AFD).

ICD-O code

- Ameloblastic fibroma 9330/0
- Ameloblastic fibrodentinoma 9271/0
- **Soft mixed odontogenic tumor**
- **Soft mixed odontoma**
- **Fibro-adamantoblastoma**

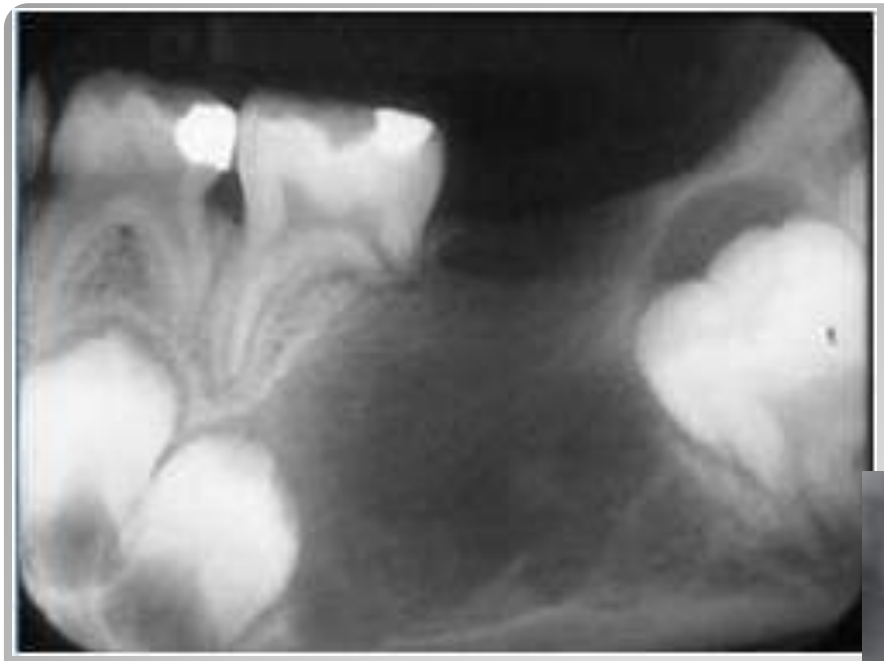
Ameloblastic fibroma /fibrodentinoma

Epidemiology

- AF/AFO is a rare odontogenic tumour
- Mean age is 14.8 years
- AF/AFO mainly occurs in the posterior Mandible.

- Most cases of AF present as painless swelling
- Discovered due to disturbances of tooth eruption.
- Radiographically, the tumour presents as a well demarcated unlocular or multi locular radiolucency with smooth border.
- Often seen in connection with a malpositioned tooth.
- Mixed radiolucent if AFO

Clinical /Imaging



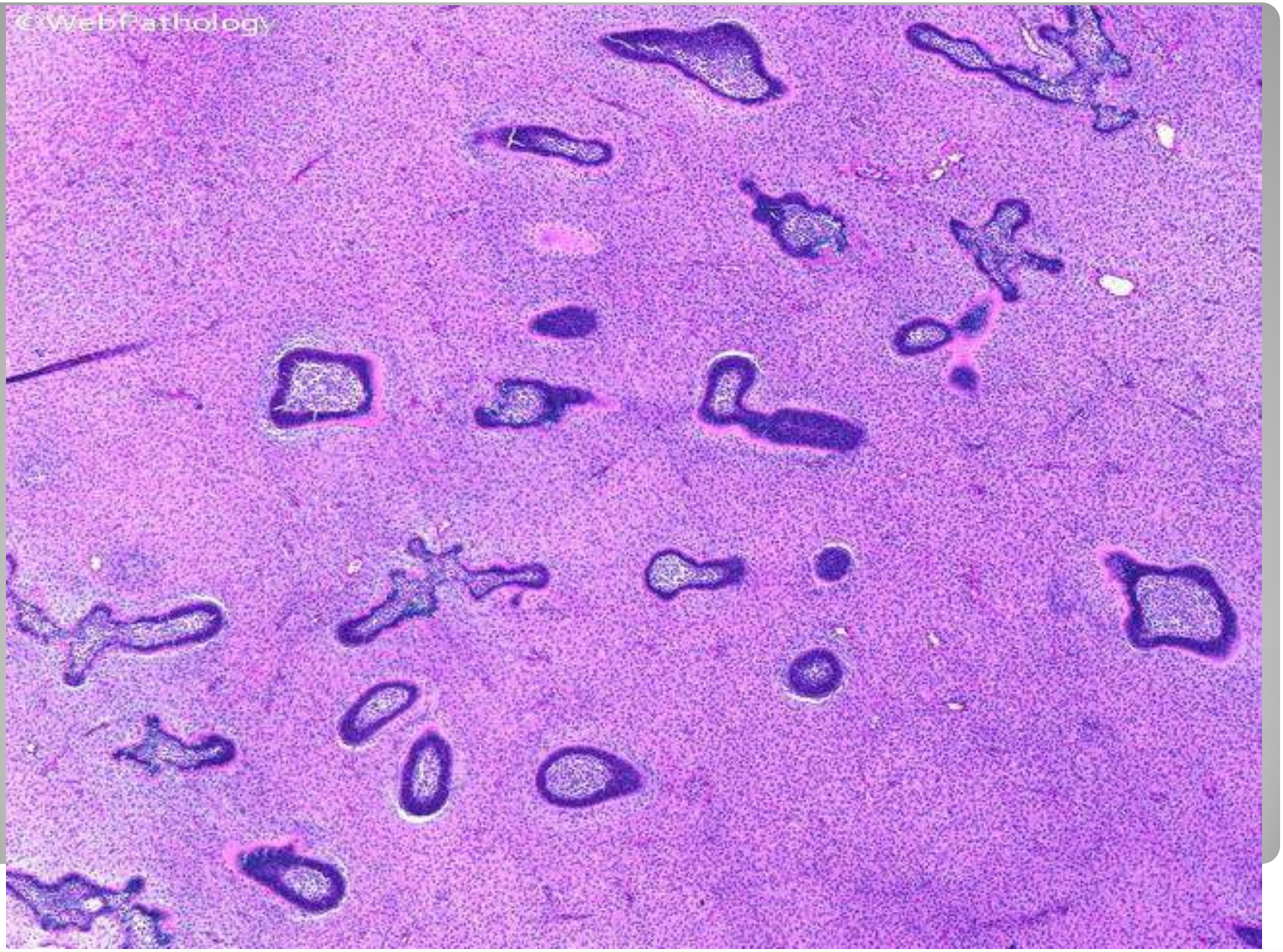
A

- Epithelial portion consists of scattered islands of epithelial cells in a variety of patterns, including rosettes, long finger like strands (alien foot appearance), nest and cords showing budding outgrowths.
- These epithelial cells are usually of a cuboidal or columnar type and bear close resemblance to primitive odontogenic epithelium.
- Mitotic activity is not common.
- Tissue resembling stellate reticulum is often not seen/ occasionally may be found.

Histopathology

Histopathology

- Mesenchymal component is made up of a primitive connective tissue that, in some cases, shows closely intertwining fibrils interspersed by large connective tissue cells closely resembling those of the dental papilla.
- On occasion, this may even resemble dysplastic dentin -- AFO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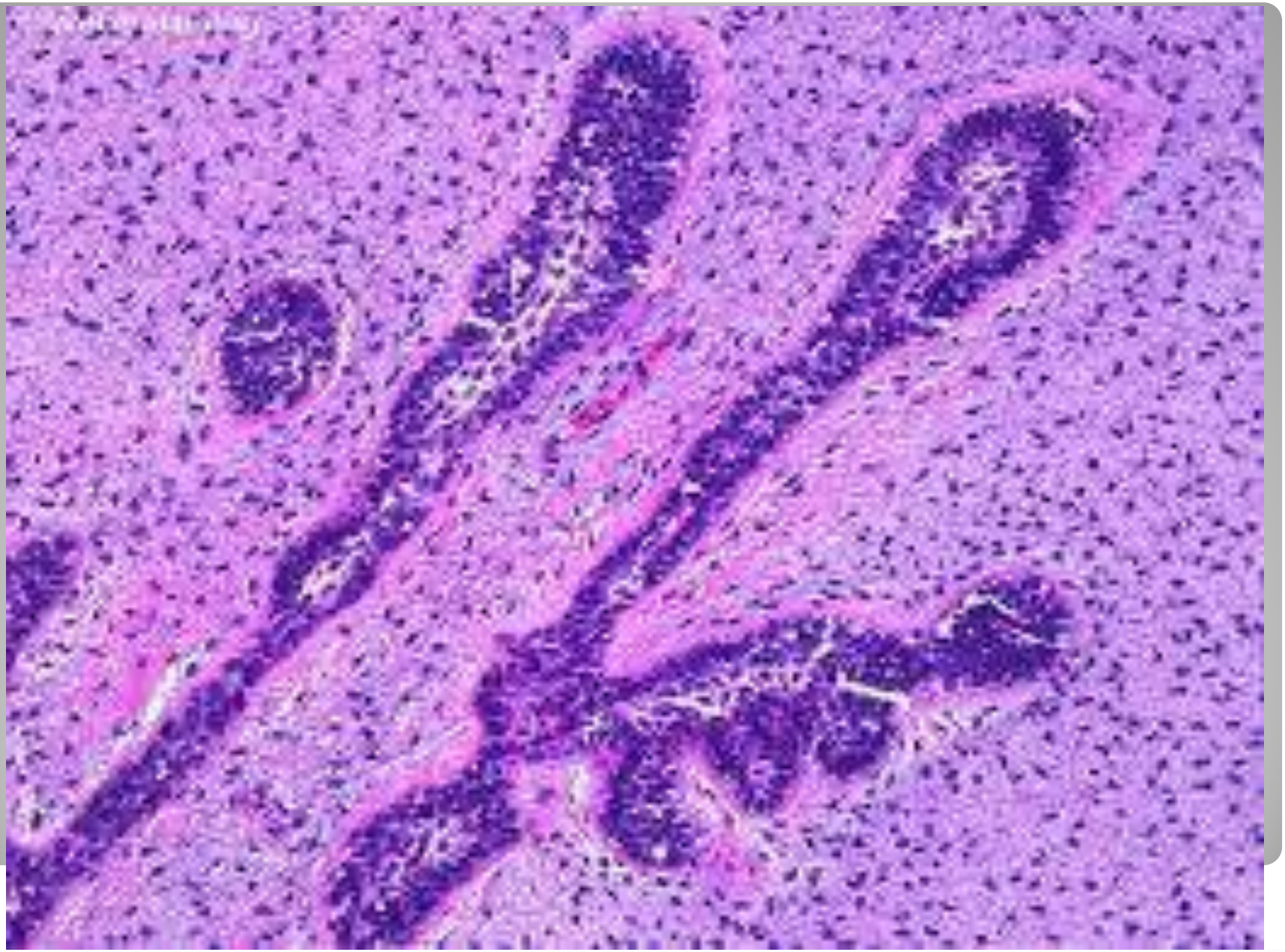

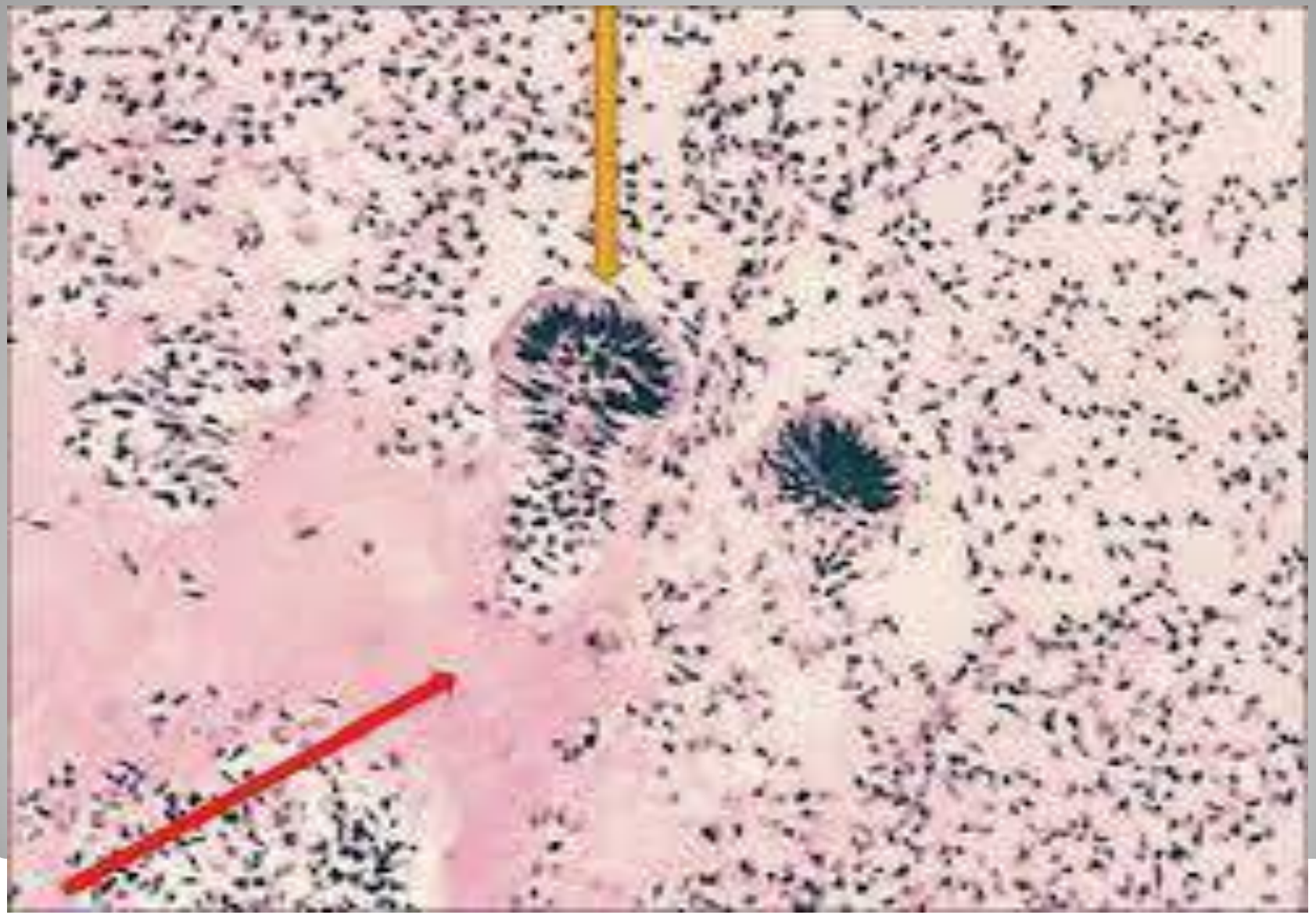

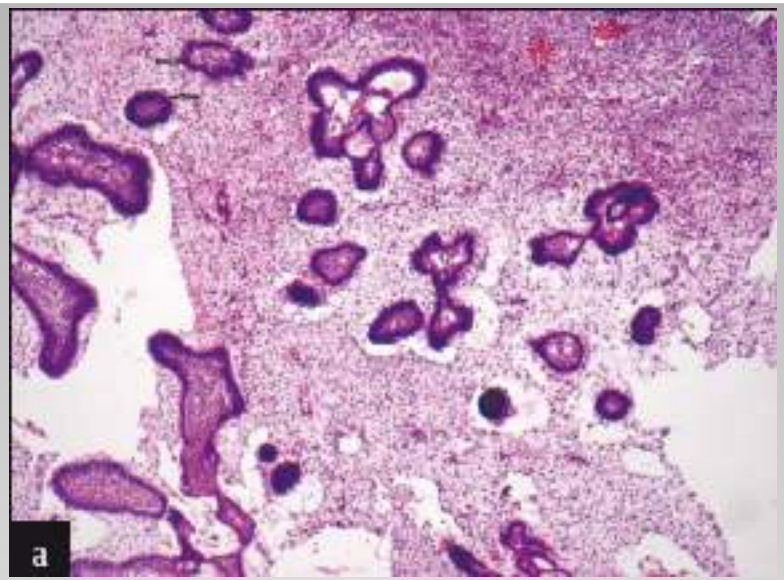
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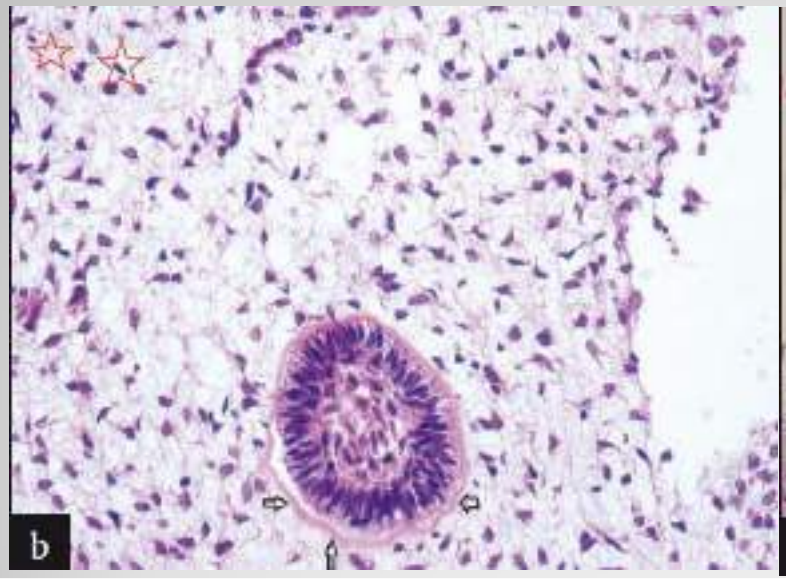
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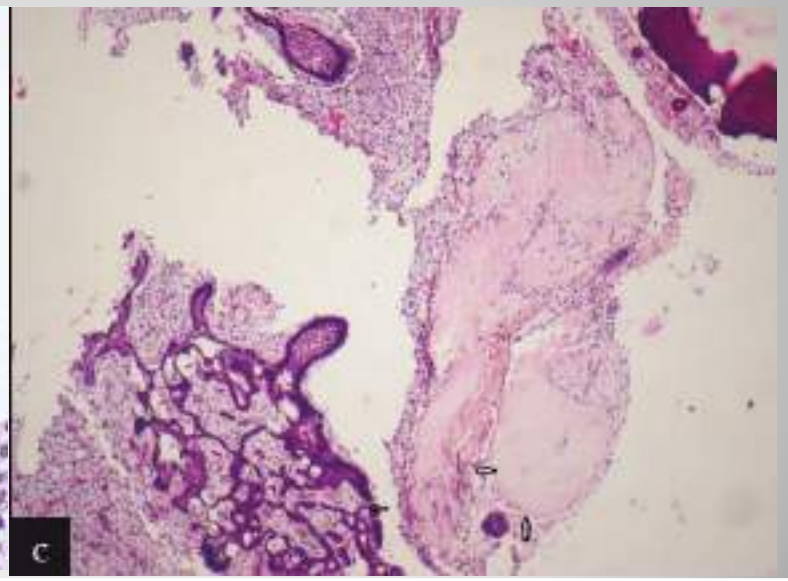
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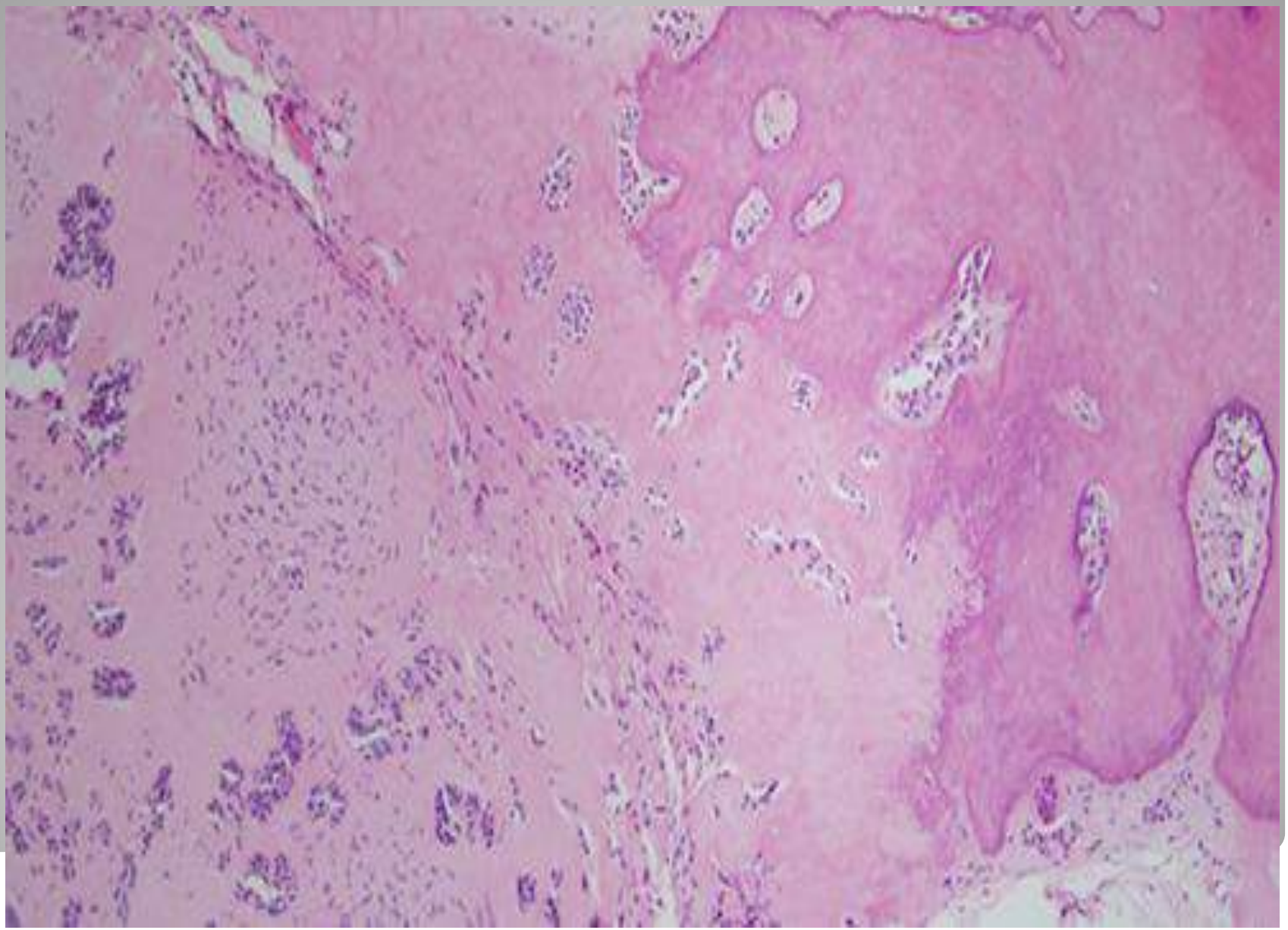

Treatment

- Treatment consists of enucleation and curettage.
- Recurrence may occur but this does not justify initial aggressive treatment
- Rarely, AF may progress to malignancy (ameloblastic fibrosarcoma).

Definition

- Ameloblastic fibro-odontoma (AFO) is a tumour, which has the histologic features of ameloblastic fibroma (AF) in Conjunction with the presence of dentin and enamel - WHO
- It is a rare odontogenic tumor composed of morphologic features characteristic of amelobtastlc fibromas on the one hand and complex odontomas on the other. – Reichart
- **ICD-O code 9290/0**

- Ameloblastic fibroma represents an early, undifferentiated complex odontoma and that the ameloblastic fibro-odontoma is simply a further differentiated maturing odontoma.
- After investigating this problem, Slootweg concluded that the ameloblastic fibroma is a true neoplasm and does not differentiate into an ameloblastic fibro-odontoma.
- He also concluded that the ameloblastic fibroodontoma, in fact, represent an immature complex odontoma, and therefore, represented a hamartomatous rather than a neoplastic odontogenic lesion.

Epidemiology

- Male: Female - 3.3 : 1
- Age ranging from 0.5–39 years ,mean being 11.5 years of age.
- Maxilla = Mandible
- Anterior = posterior

- AFO is often asymptomatic
- May be detected as a result of failure of tooth eruption.
- Radiographically, AFO exhibits a well-circumscribed unilocular or multilocular radiolucency with varying levels of radiopacity depending on the extent of mineralization.
- AFO is often associated with an unerupted tooth.

Clinical /Imaging

- Lesion consists of the same apparent type of tissue as seen in the ameloblastic fibroma.
- Thus, cords, fingers, strands and rosettes of primitive odontogenic columnar or cuboidal epithelial cells, often resembling dental lamina, are found.
- The mesenchymal component is an embryonic fibrous connective tissue with delicate fibrils interspersed by large primitive fibroblasts, all resembling dental papilla. In addition, typical composite odontoma is found.

Histopathology

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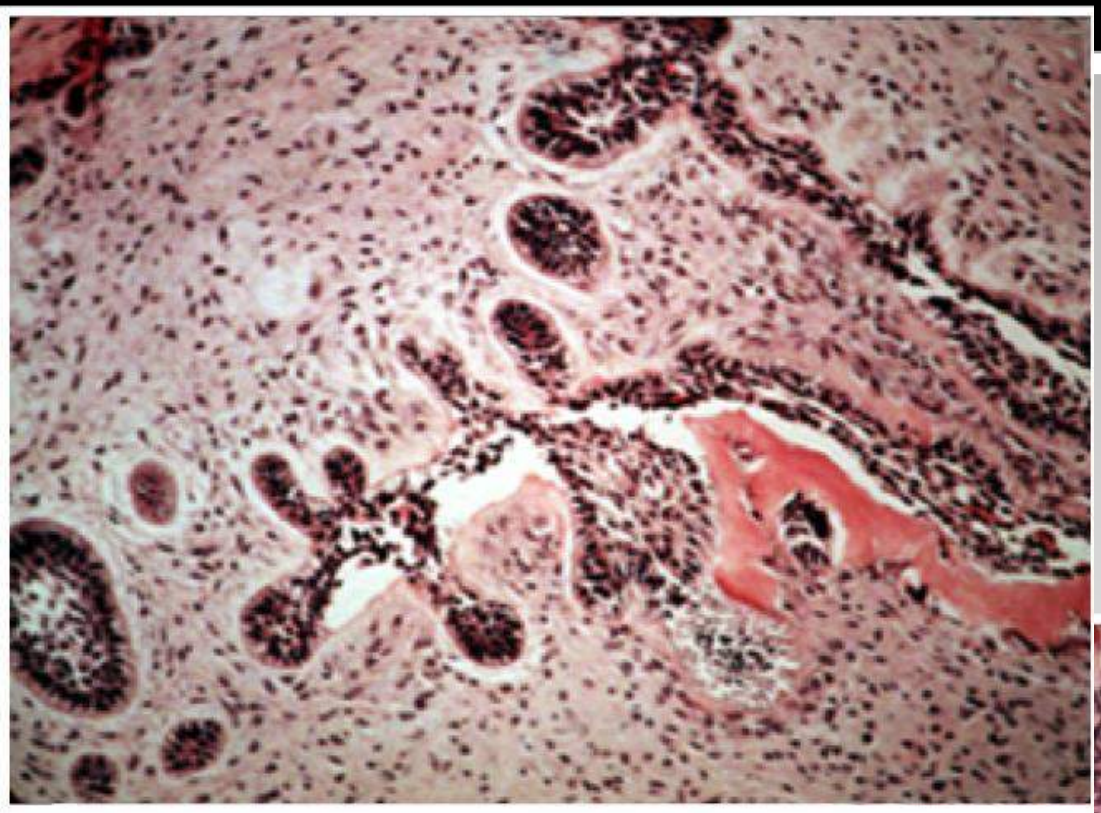
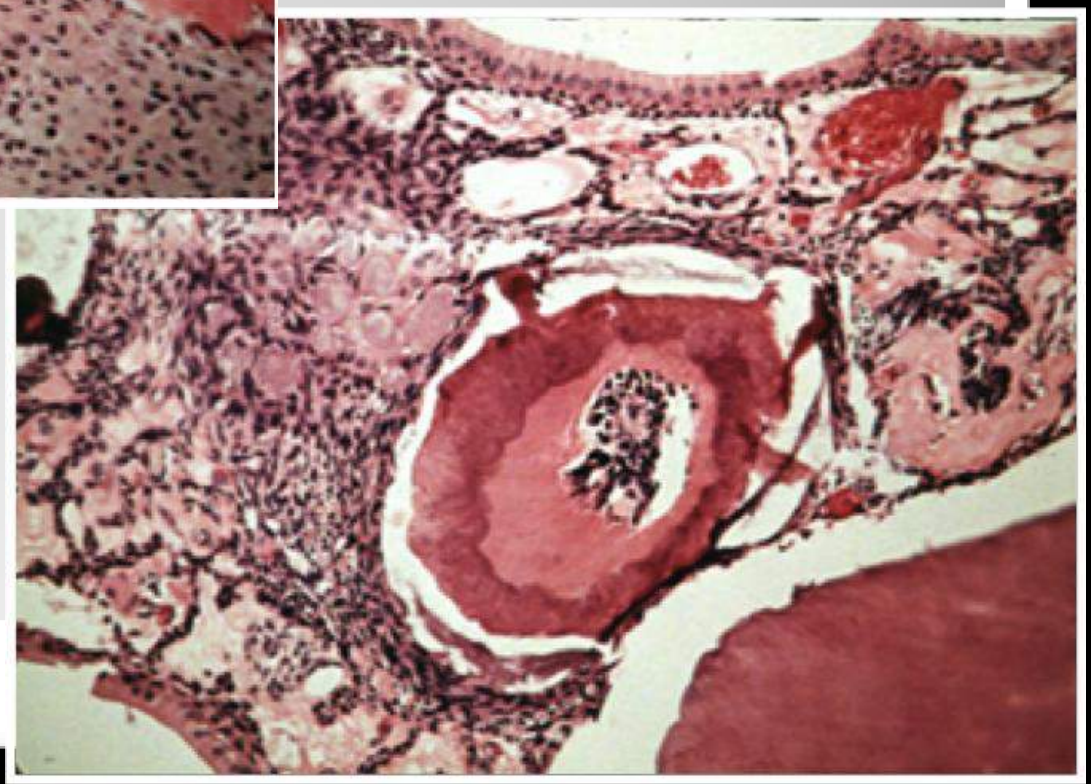




- Treated by curettage, since it does not appear to locally invade bone.
- Little tendency for recurrence of the ameloblastic fibro-odontoma.

Treatment

Definition

- Odontoameloblastoma (OA) combines features of ameloblastoma with those of an odontoma.

ICD-O code 9311/0

Synonyms

- Ameloblastic odontoma, odontoblastoma

Odontoameloblastoma

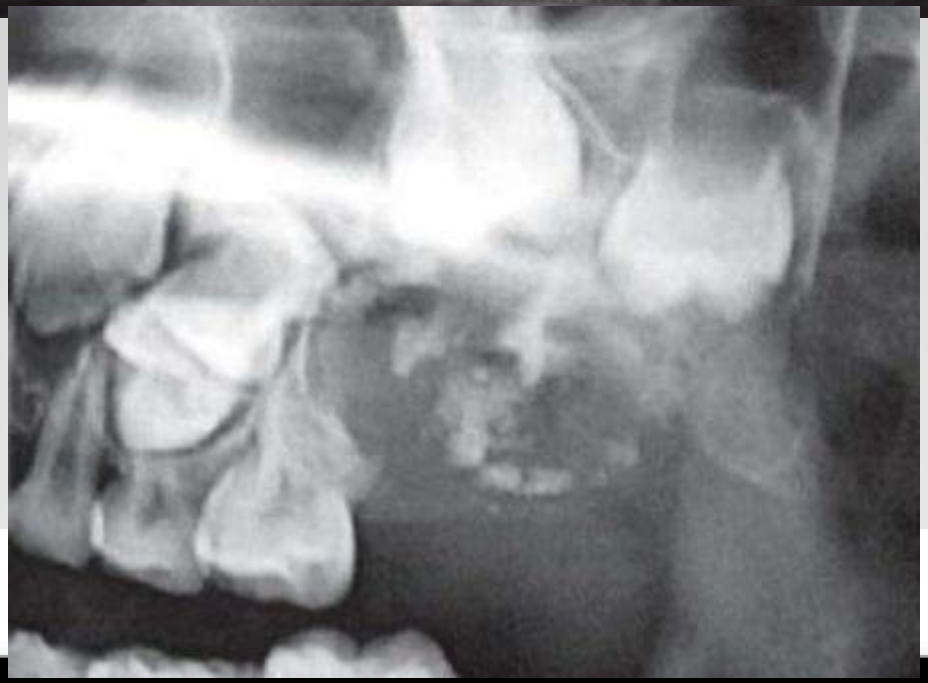
- Any statistical information may not be valid – because of very few cases reported
- More frequently in children, and is somewhat more common in the mandible than in the maxilla.
- It is a slowly expanding lesion of bone which produces considerable facial deformity or asymmetry if left untreated.
- Since it is a central lesion, considerable destruction of bone occurs.
- Mild pain may be a presenting complaint, as well as delayed eruption of teeth.

Clinical presentation

- Central destruction of bone with expansion of the cortical plates is prominent.
- Numerous small radiopaque masses which may or may not bear resemblance to miniature teeth.
- In other instances there is only a single, irregular radiopaque mass of calcified tissue present.
- Thus the radiographic appearance of the ameloblastic odontoma is identical with that of the composite odontoma.

Imagining

L



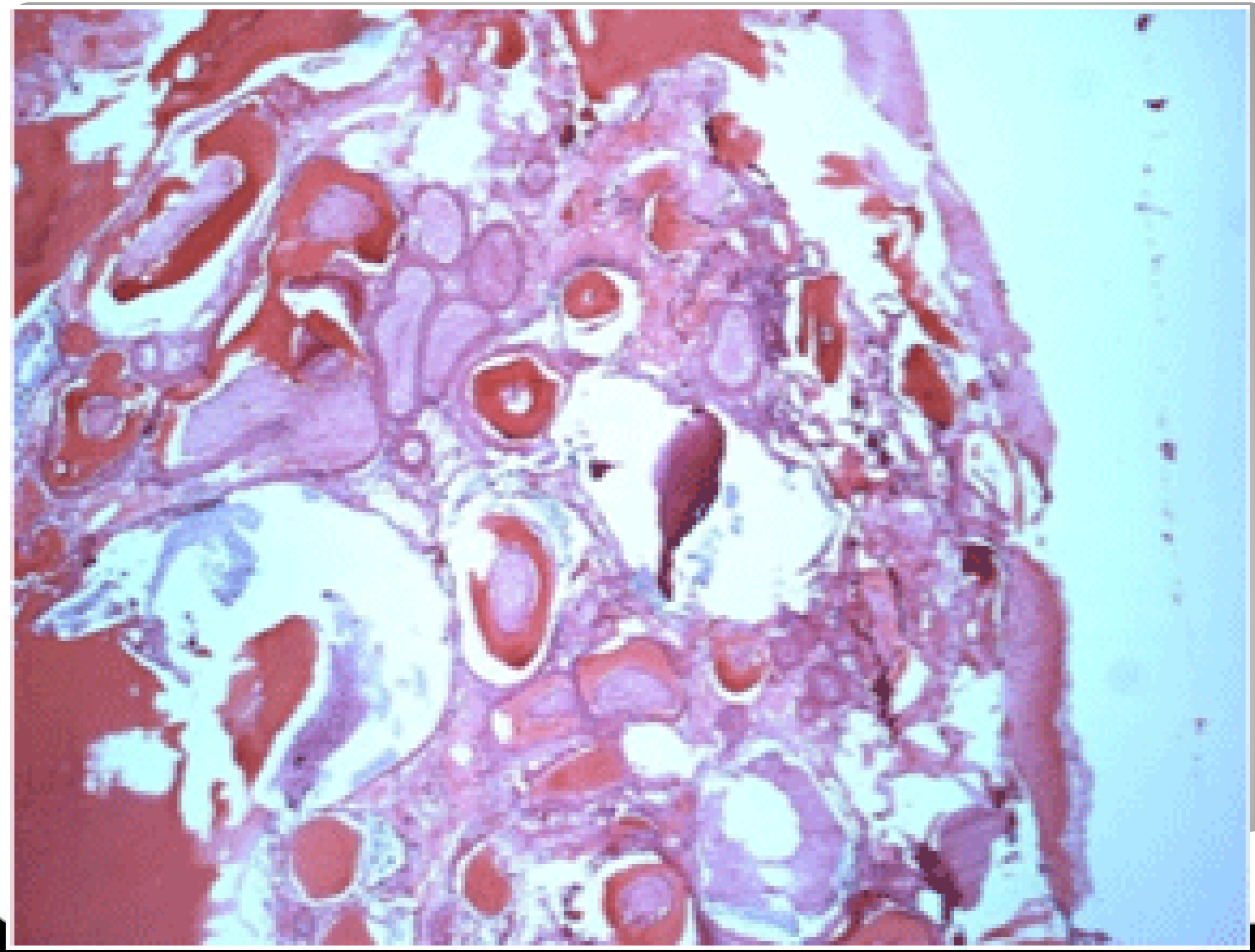
4mm/rot
4sp

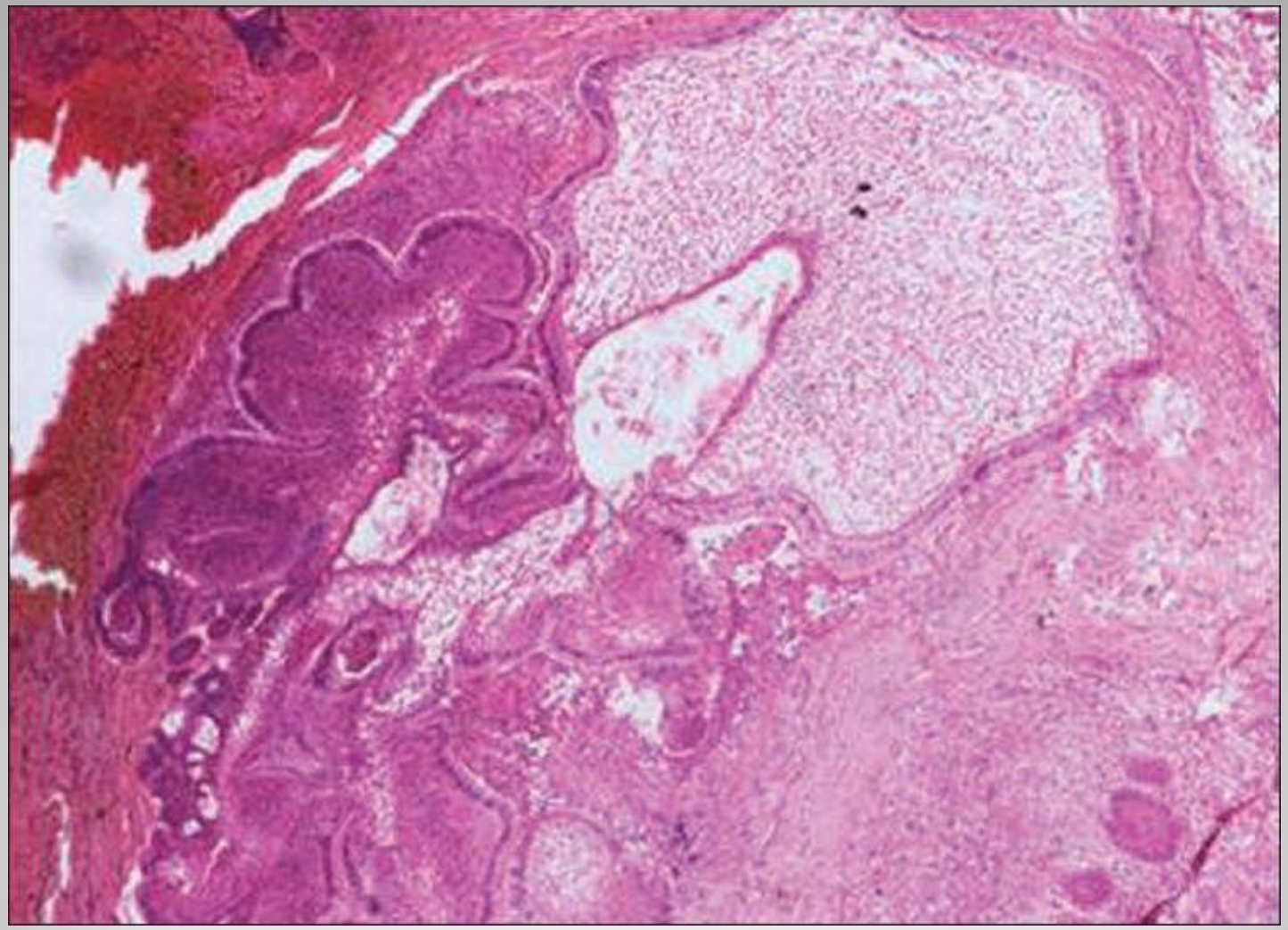
- Most OAs are unencapsulated.
- On cut section, the lesion has a multinodular architecture with soft and hard tissue components.
- The amount of mineralized tissue may appear as large lobulated masses or as rudimentary teeth scattered within the soft tissue.

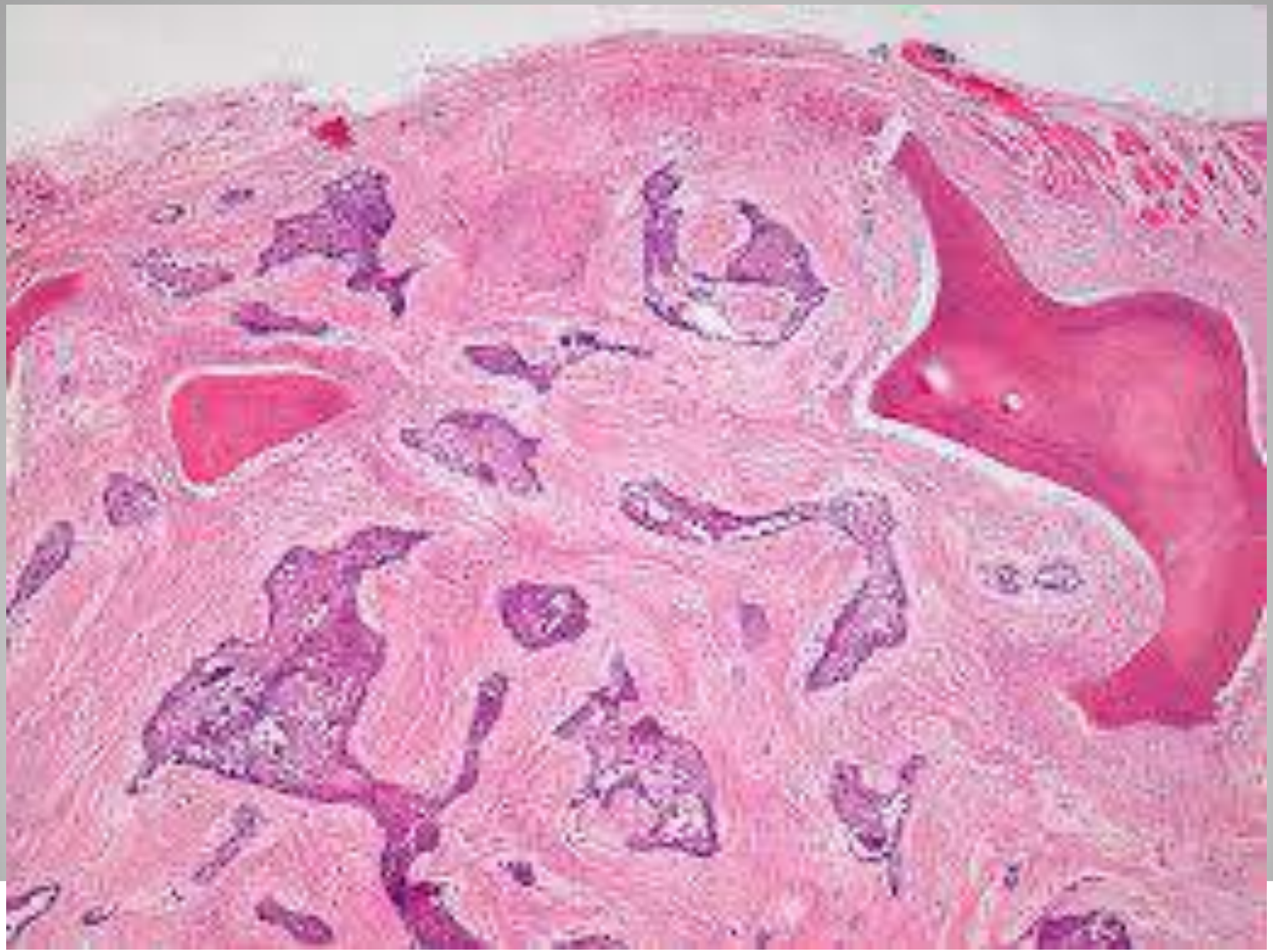
Macroscopy

- The epithelial component consists of islands and cords of odontogenic epithelium demonstrating follicular and plexiform patterns, typical of ameloblastoma.
- In addition to the fibrous stroma, this tumour shows a variable amount of cellular myxoid tissue adjacent to the epithelium, where mineralized dental tissues are formed as in odontomas
- Isolated small foci of ghost cells may occasionally be found

Microscopy







Treatment

- The general behavior of this lesion is the same as that of the ameloblastoma component, and therefore, the same philosophy of management would also apply here as for the ameloblastoma.
- Curettage & enucleation (Recurrence observed)
- Segmental resection
- Enblock excision
-

Definition

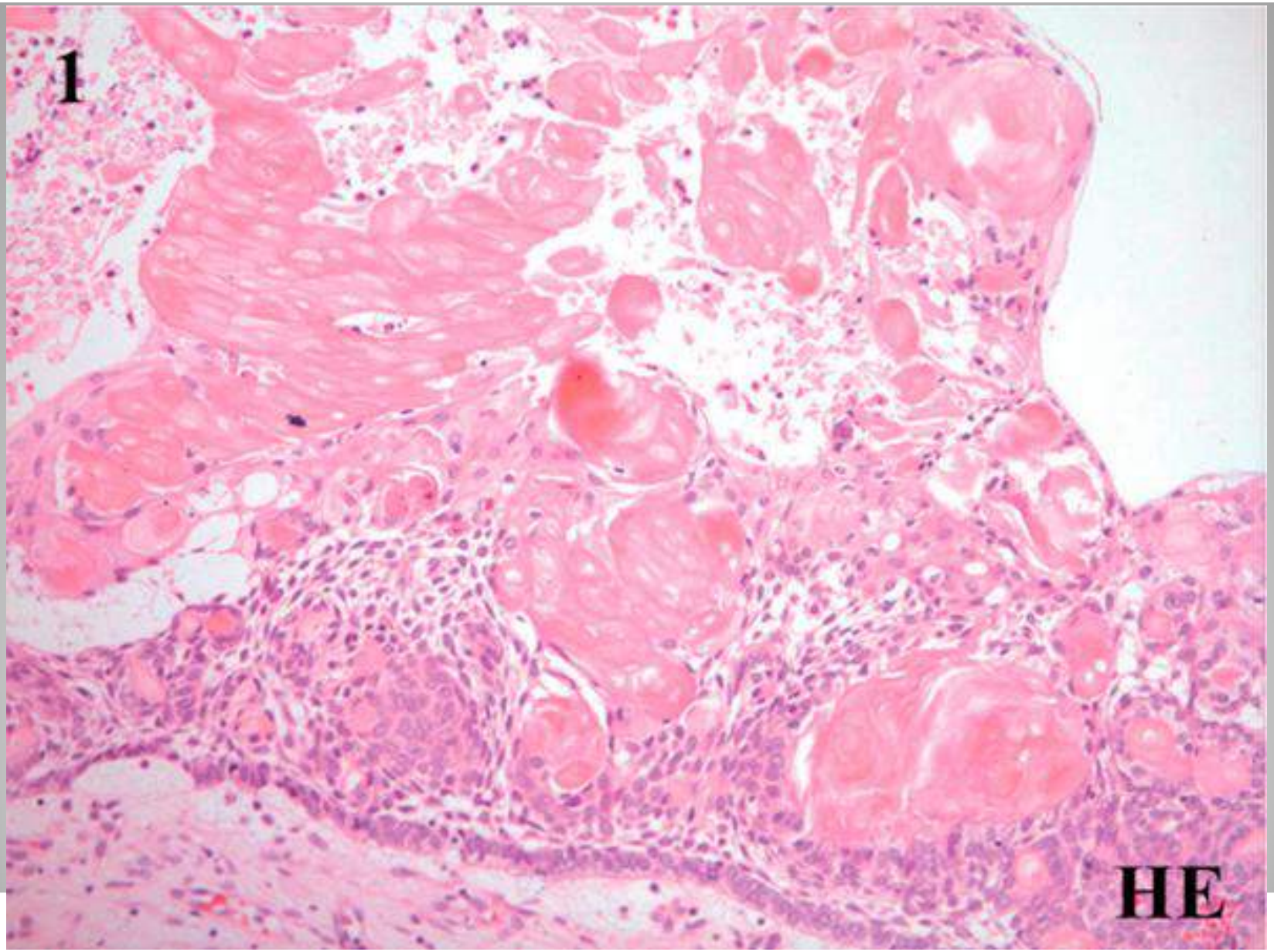
Calcifying cystic odontogenic tumour (CCOT) is a benign cystic neoplasm of odontogenic origin, characterized by an ameloblastoma-like epithelium with ghost cells that may calcify.

A locally invasive neoplasm characterised by ameloblastoma-like islands of epithelial cells in a mature connective tissue stroma. Aberrant keratinization may be found in the form of ghost cells in association with varying amounts of dysplastic dentin.

Calcifying cystic odontogenic tumour & Dentinogenic ghost cell tumour (CCOT & DGCT)

Group 1 - 'Simple' cysts • Calcifying odontogenic cyst (COC)
Group 2 - Cysts associated with odontogenic hamartomas or benign neoplasms: calcifying cystic odontogenic tumours (CCOT). The following combinations have been published • CCOT associated with an odontome • CCOT associated with adenomatoid odontogenic tumor • CCOT associated with ameloblastoma • CCOT associated with ameloblastic fibroma • CCOT associated with ameloblastic fibro-odontoma • CCOT associated with odonto-ameloblastoma • CCOT associated with odontogenic myxofibroma
Group 3 - Solid benign odontogenic neoplasms with similar cell morphology to that in the COC, and with Dentinoid formation • Dentinogenic ghost cell tumour
Group 4 - Malignant odontogenic neoplasms with features similar to those of the dentinogenic ghost cell tumour • Ghost cell odontogenic carcinoma

Suggested classification of the odontogenic ghost cell lesions. (From Praetorius, 2006)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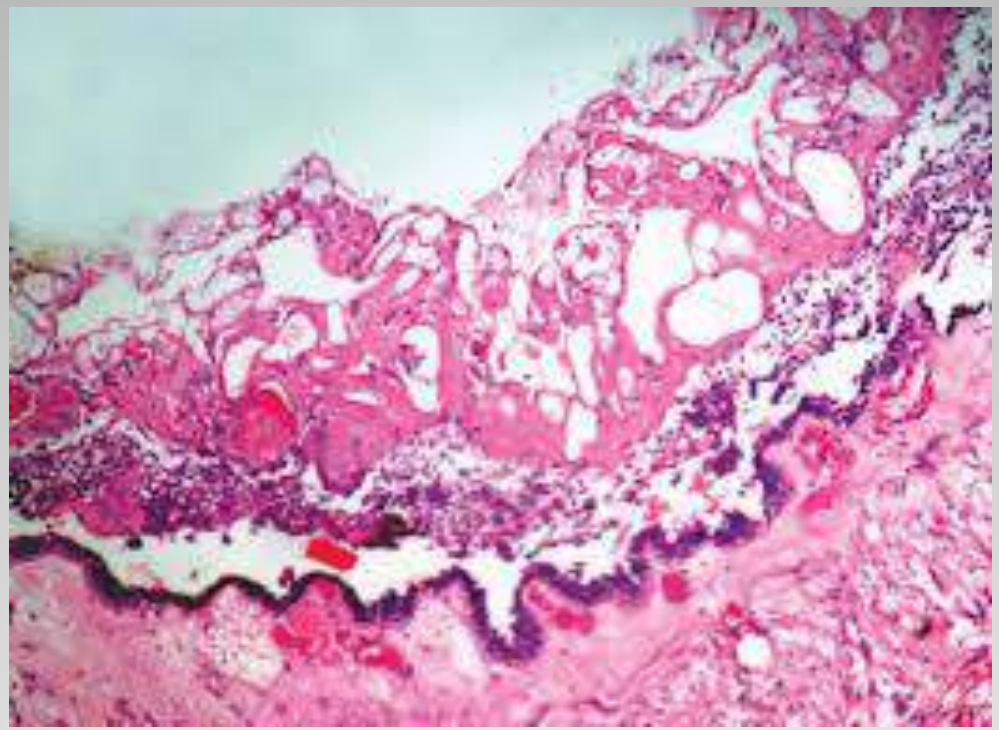

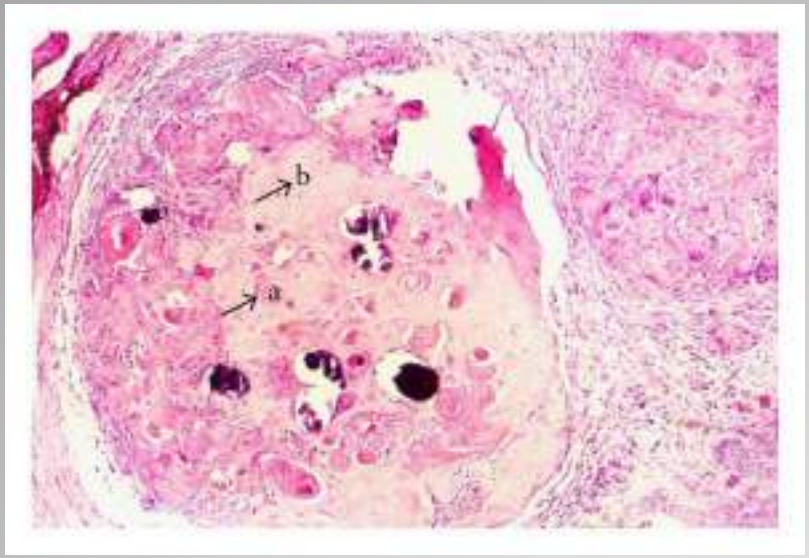
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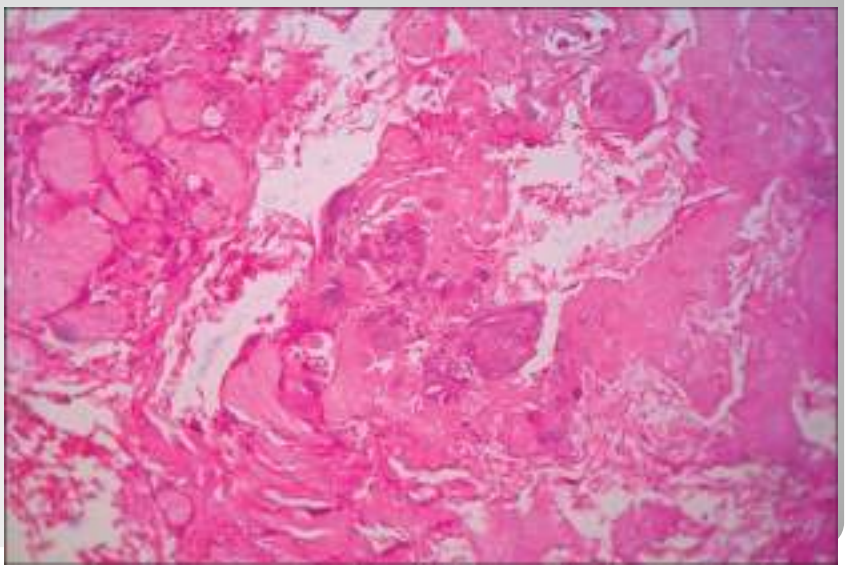

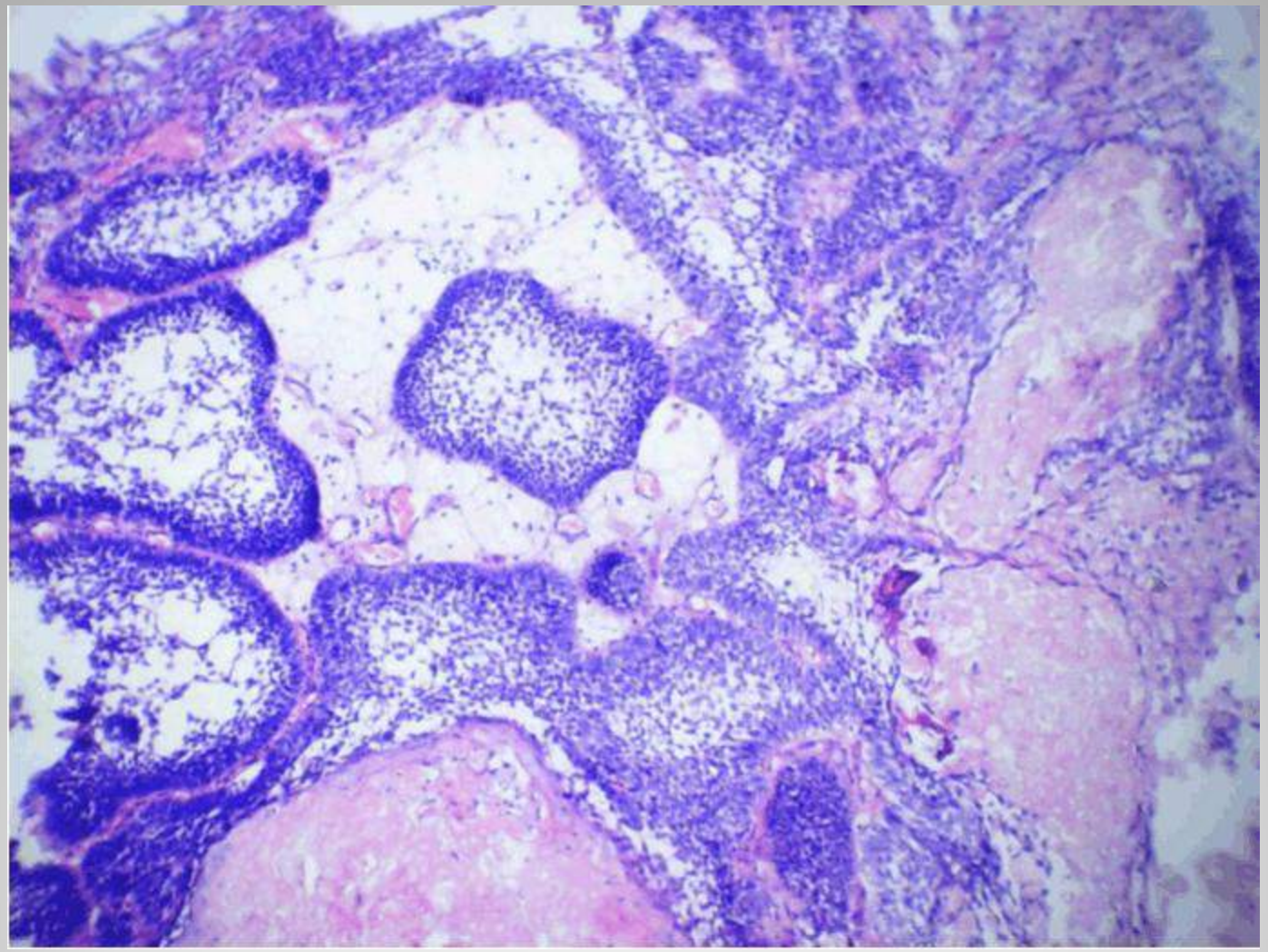

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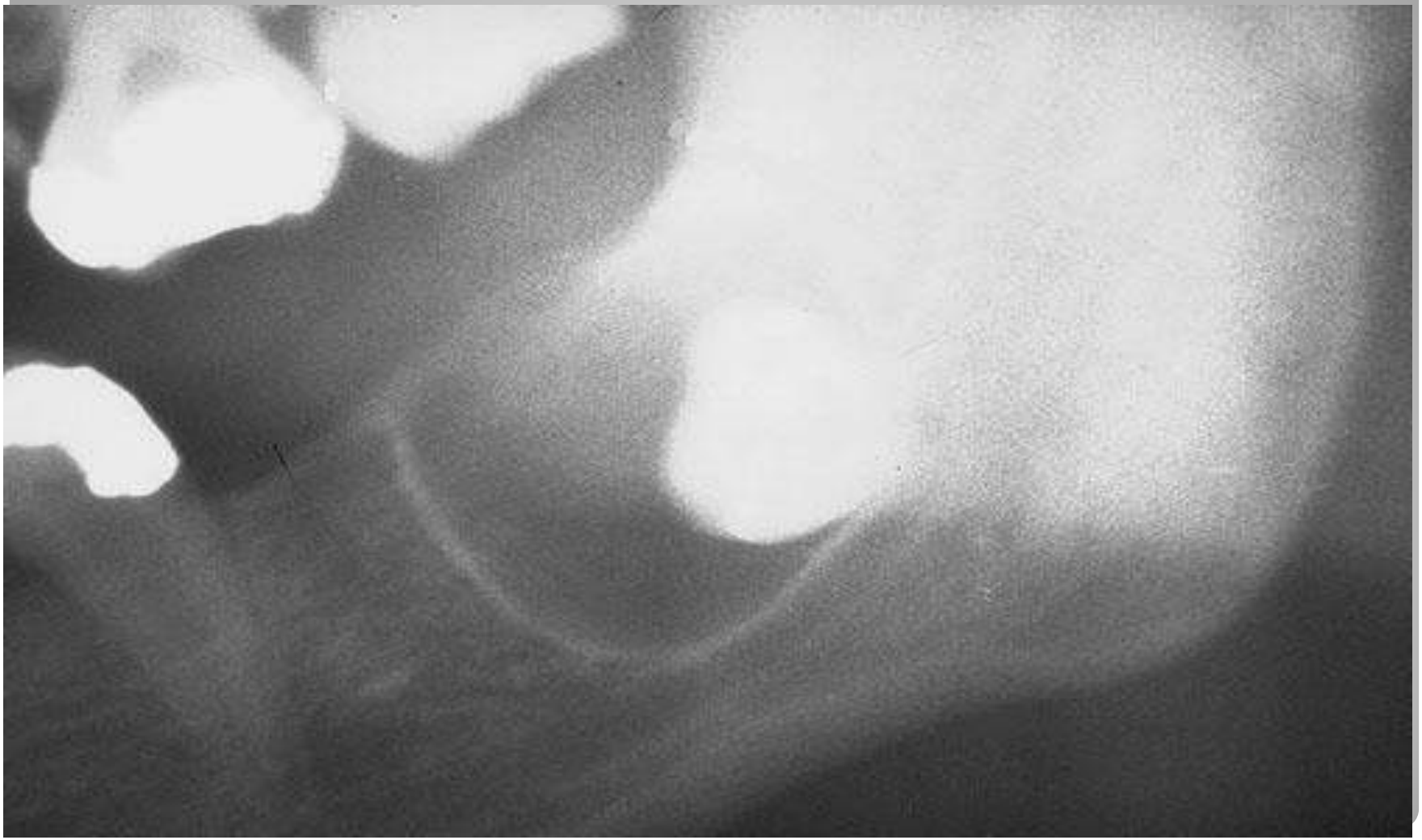

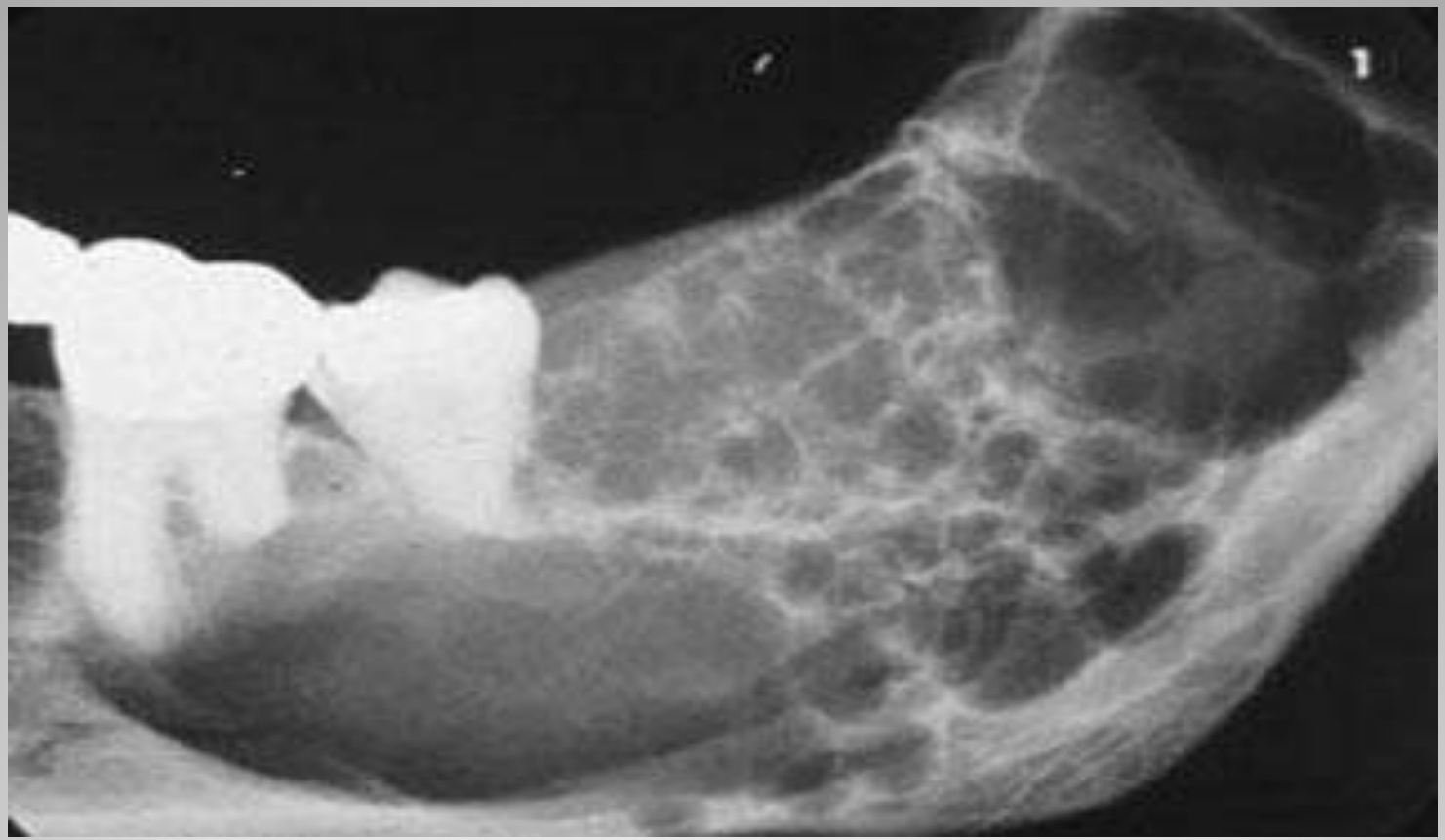

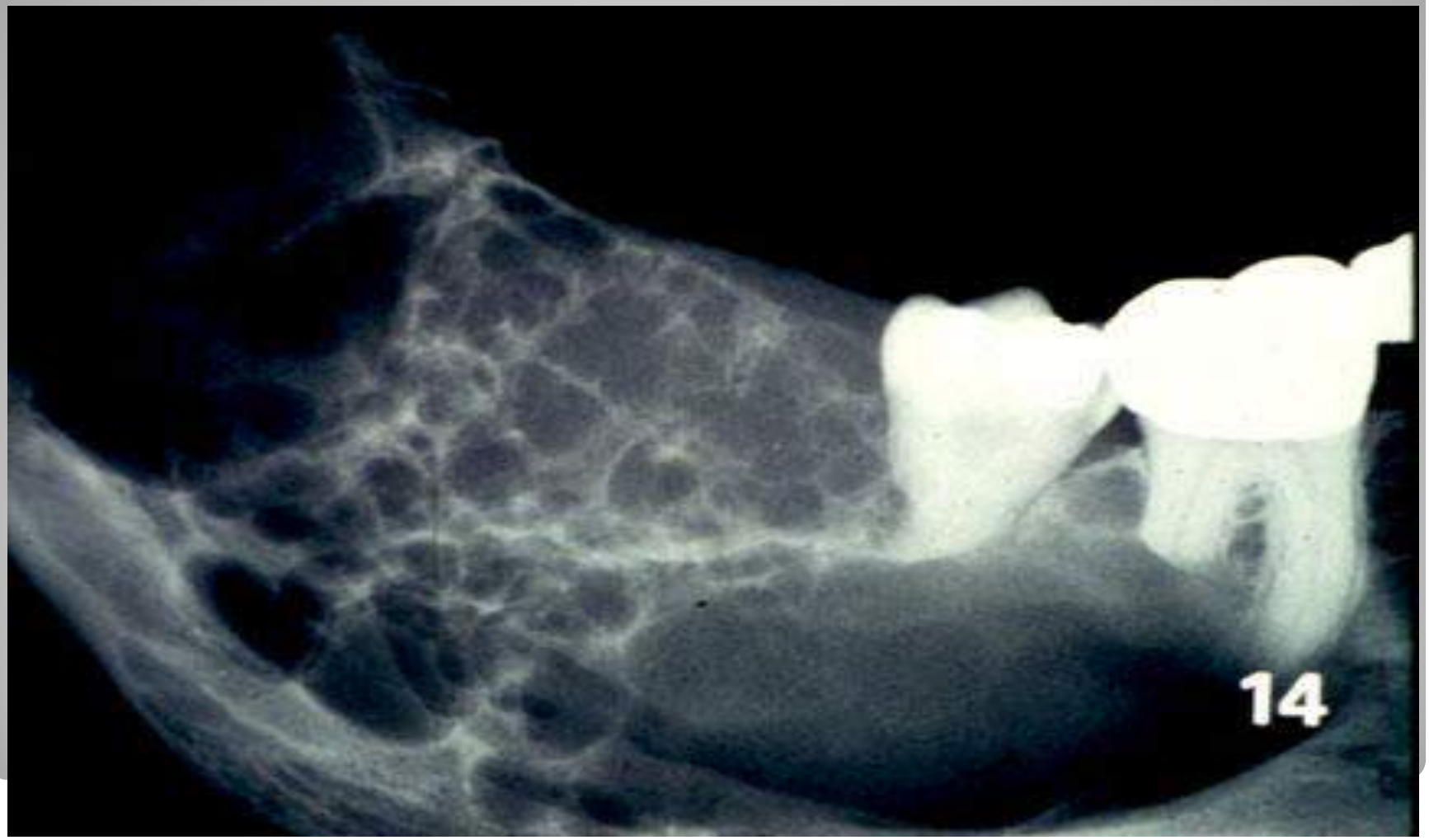
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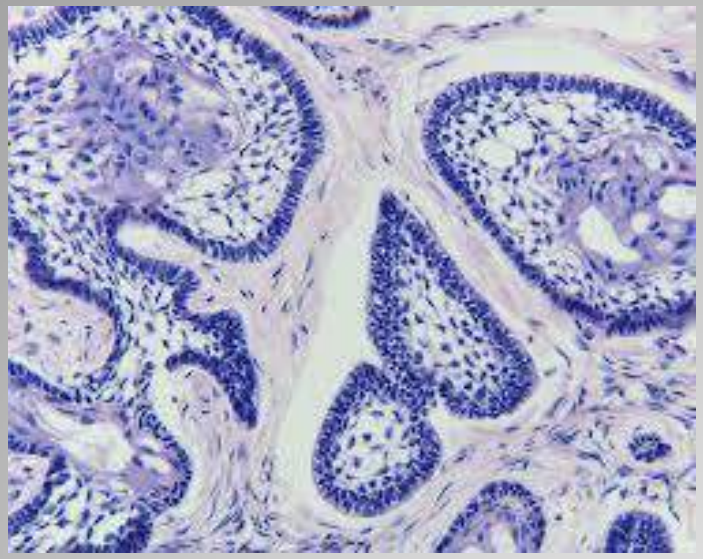
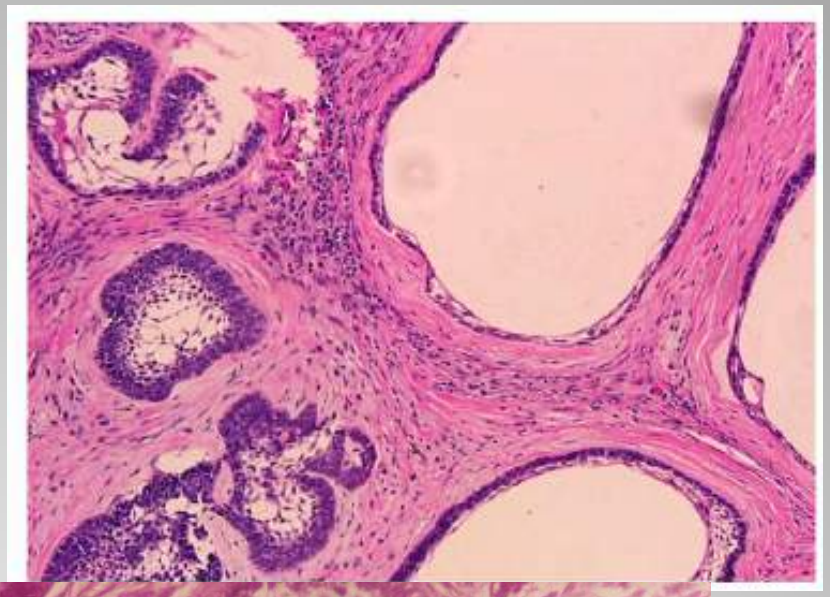
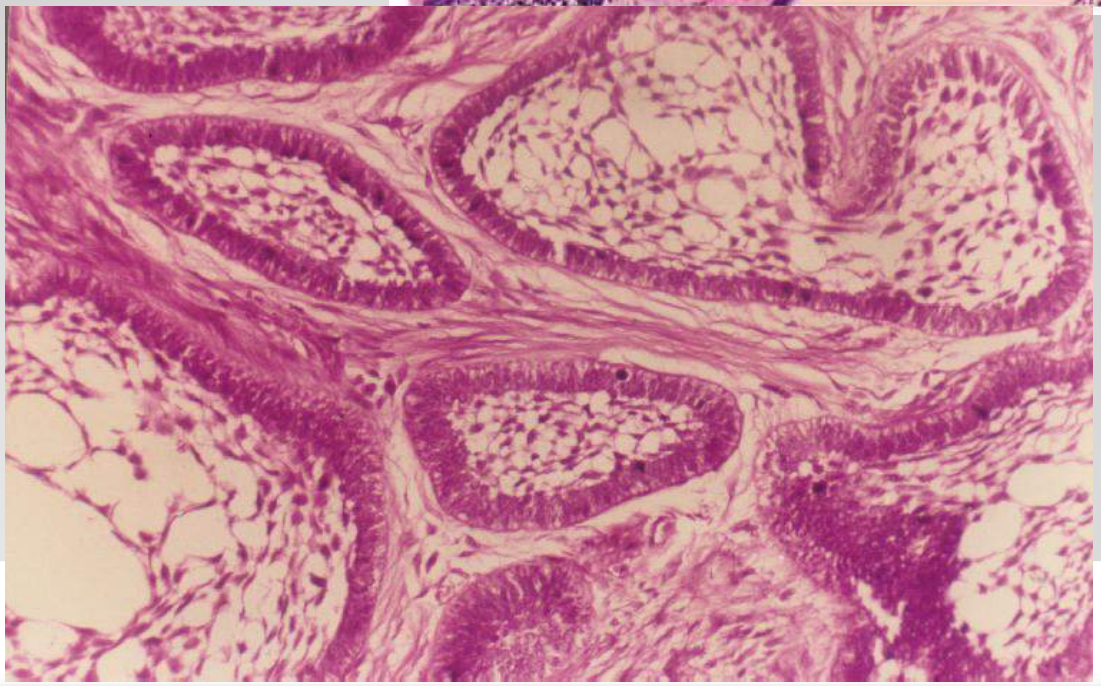
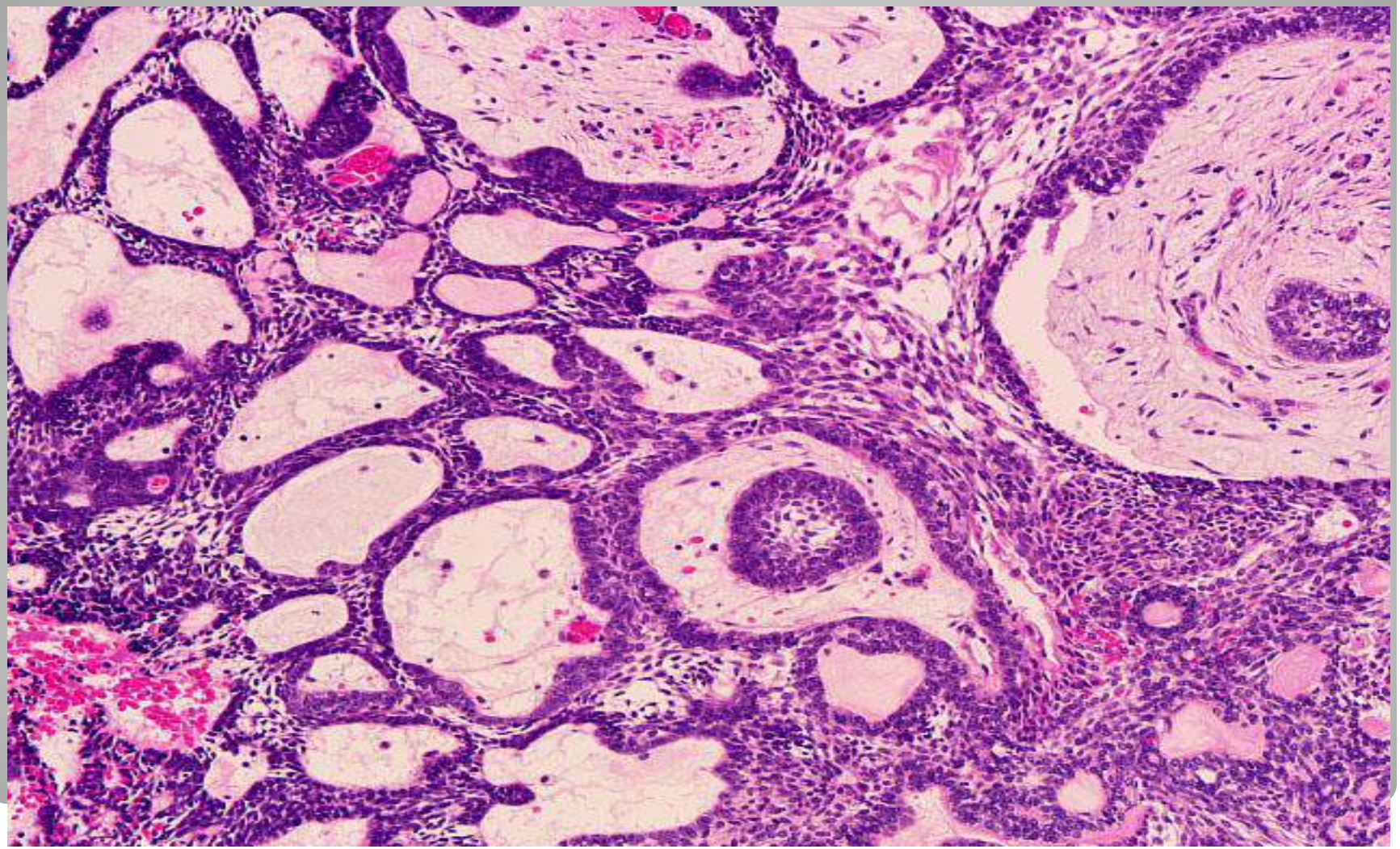
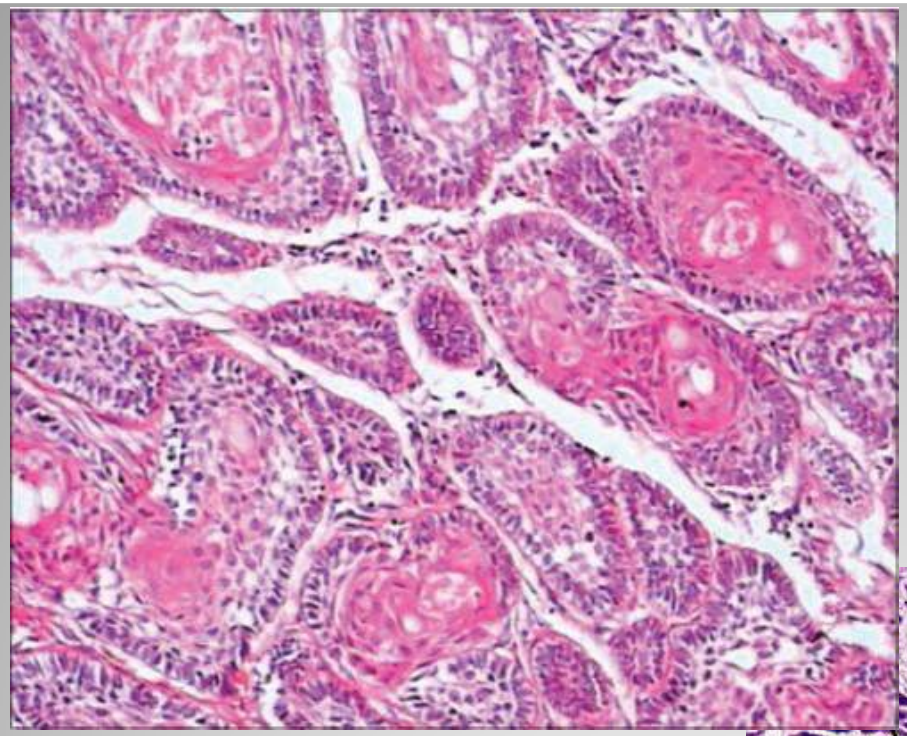
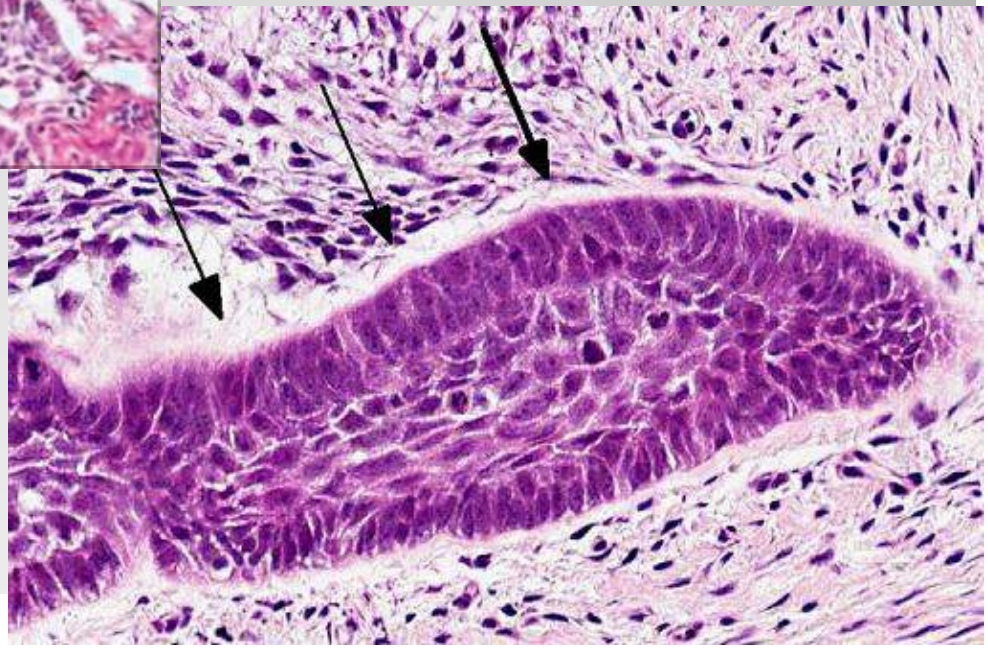


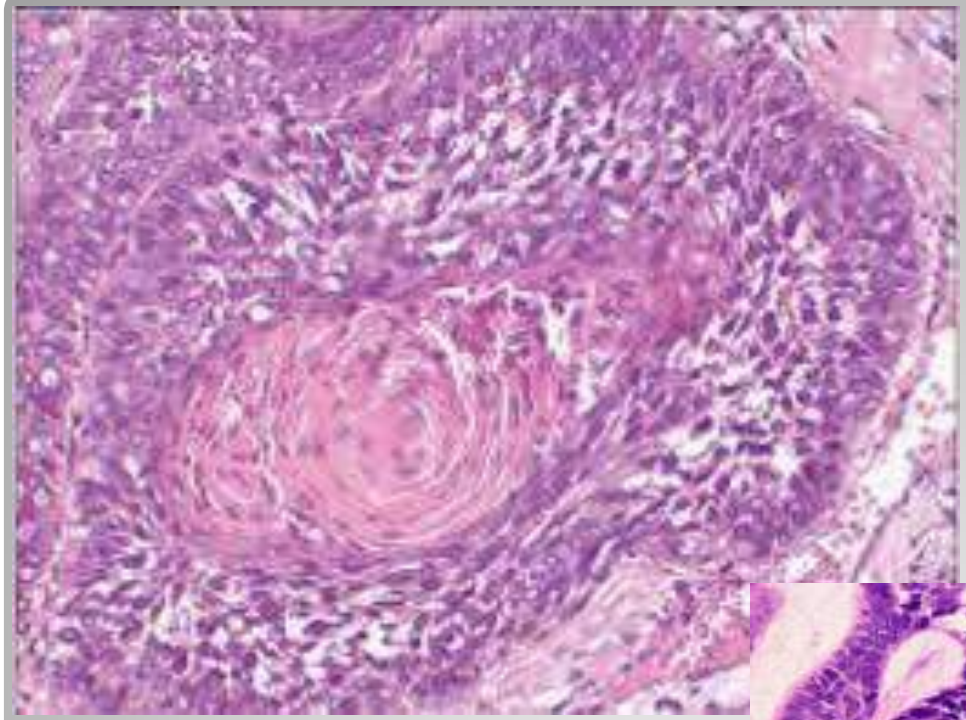


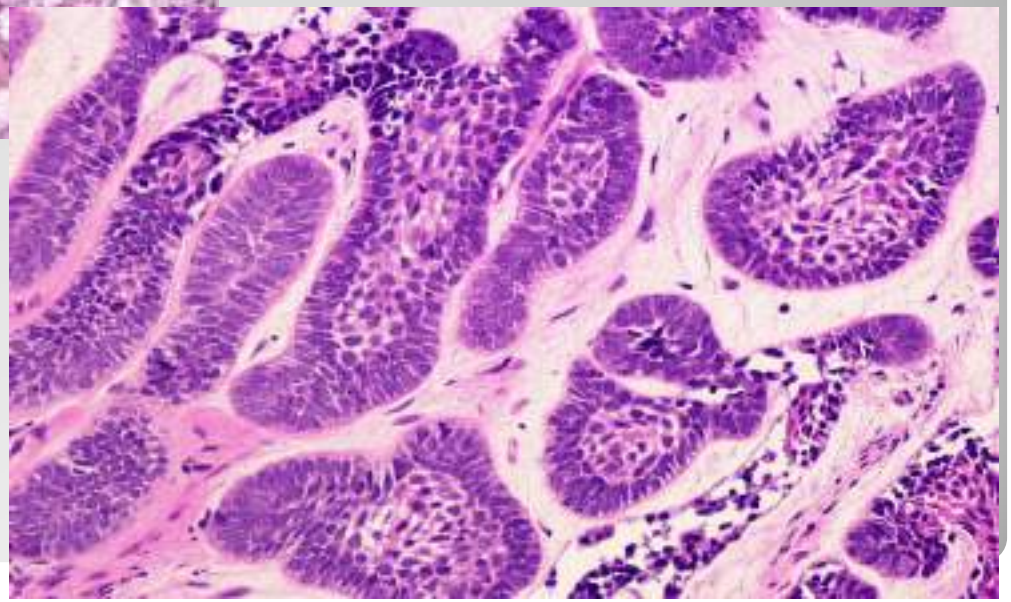


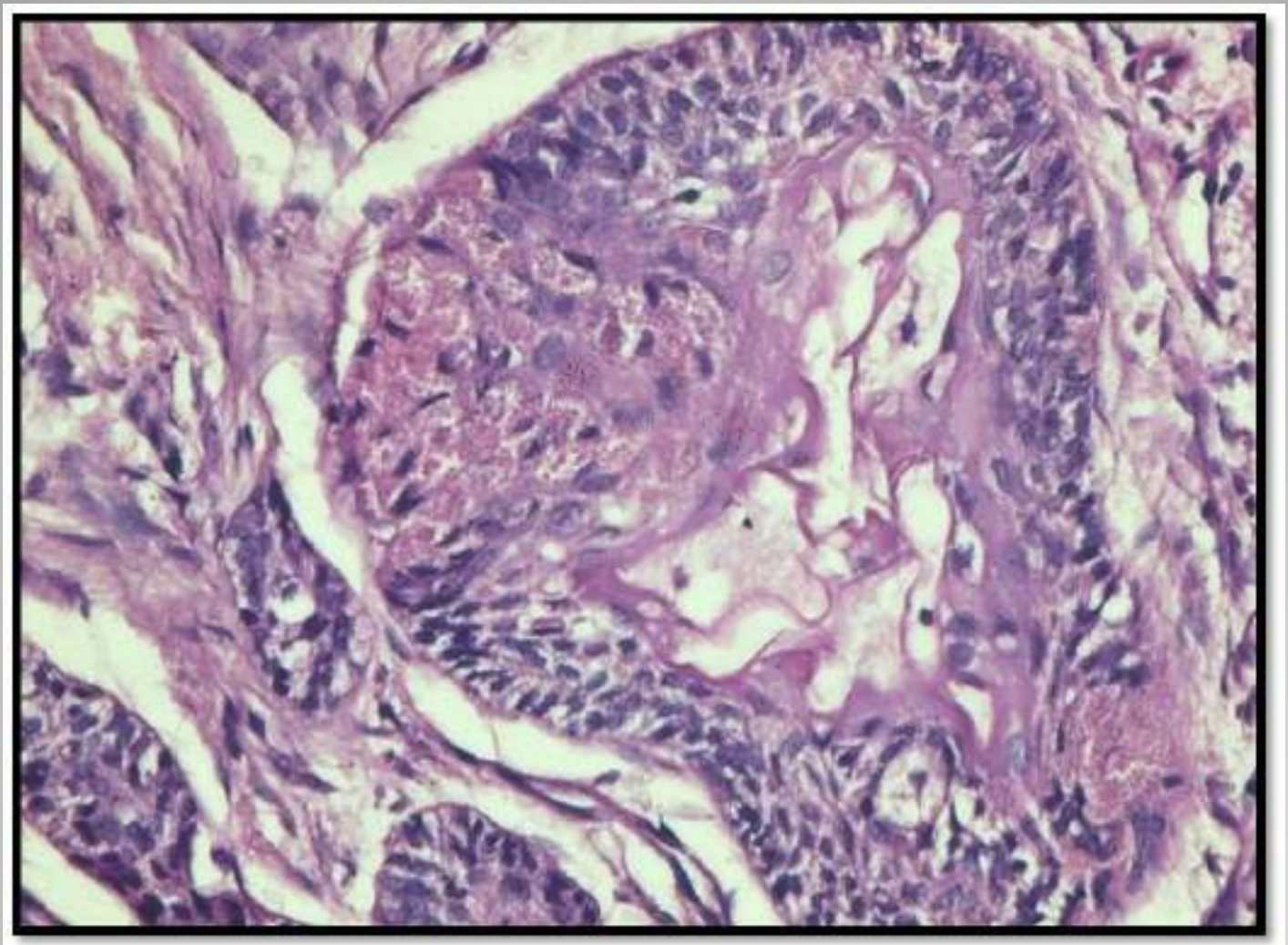


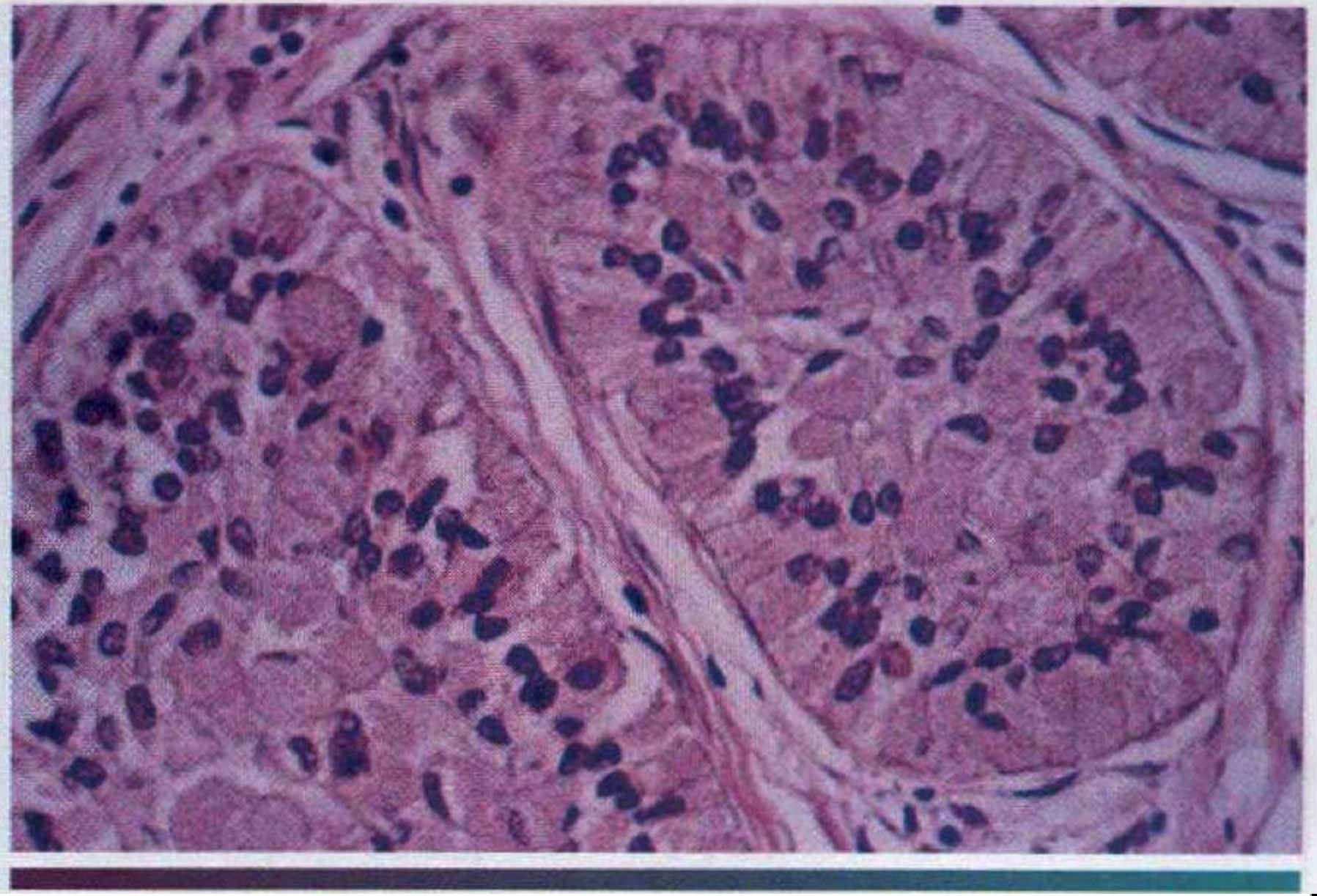




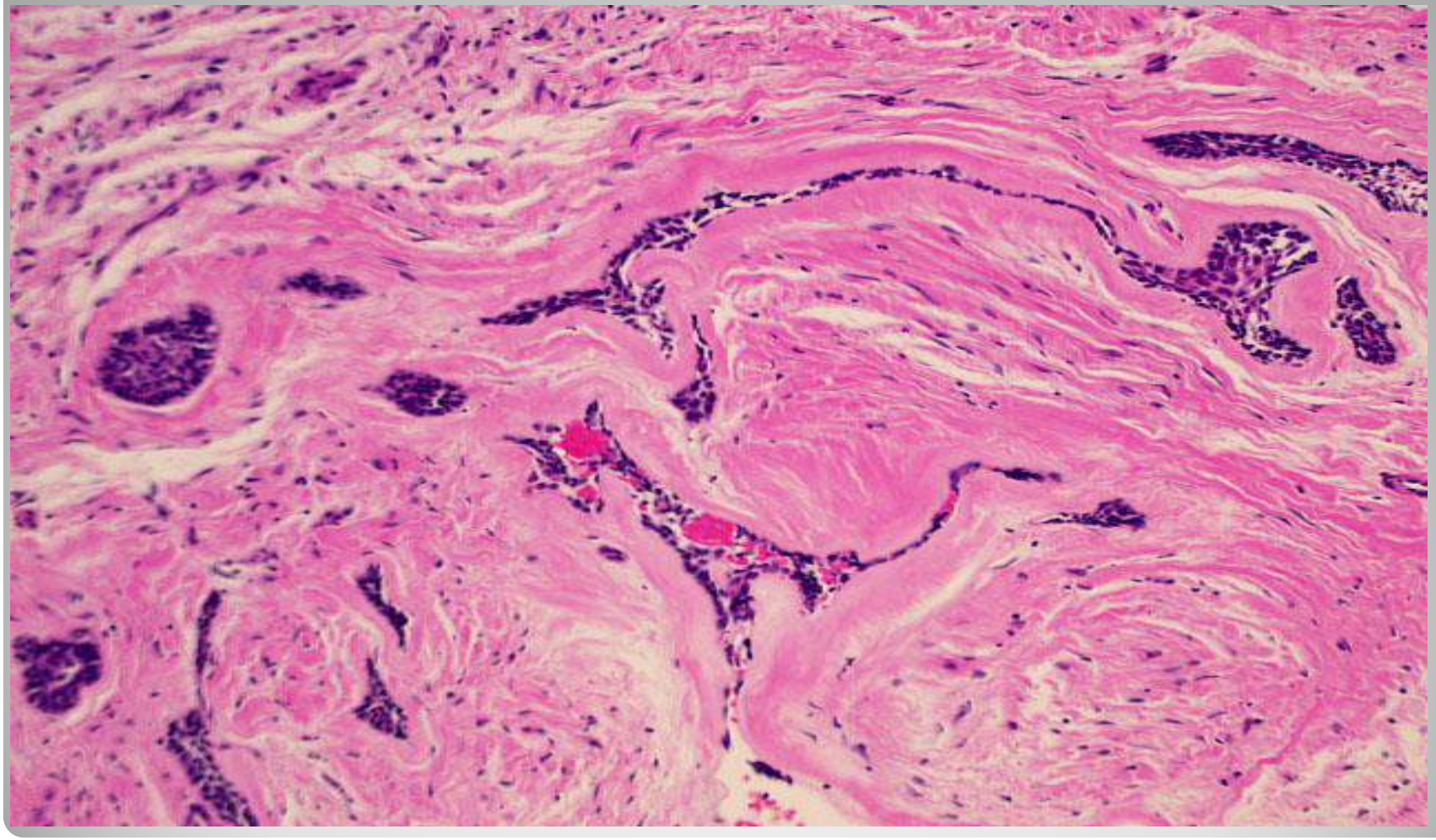


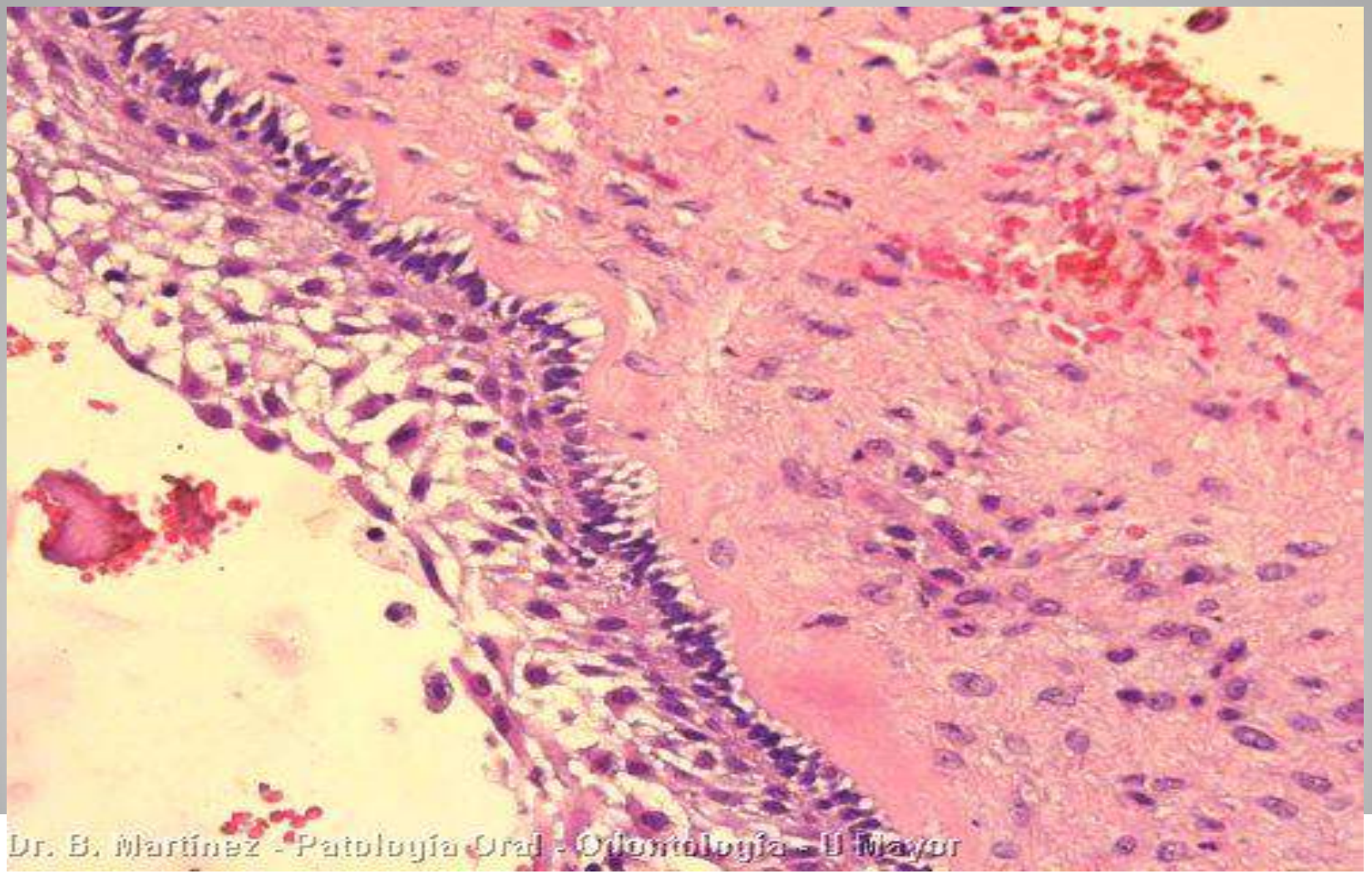






Desmoplastic Ameloblastoma

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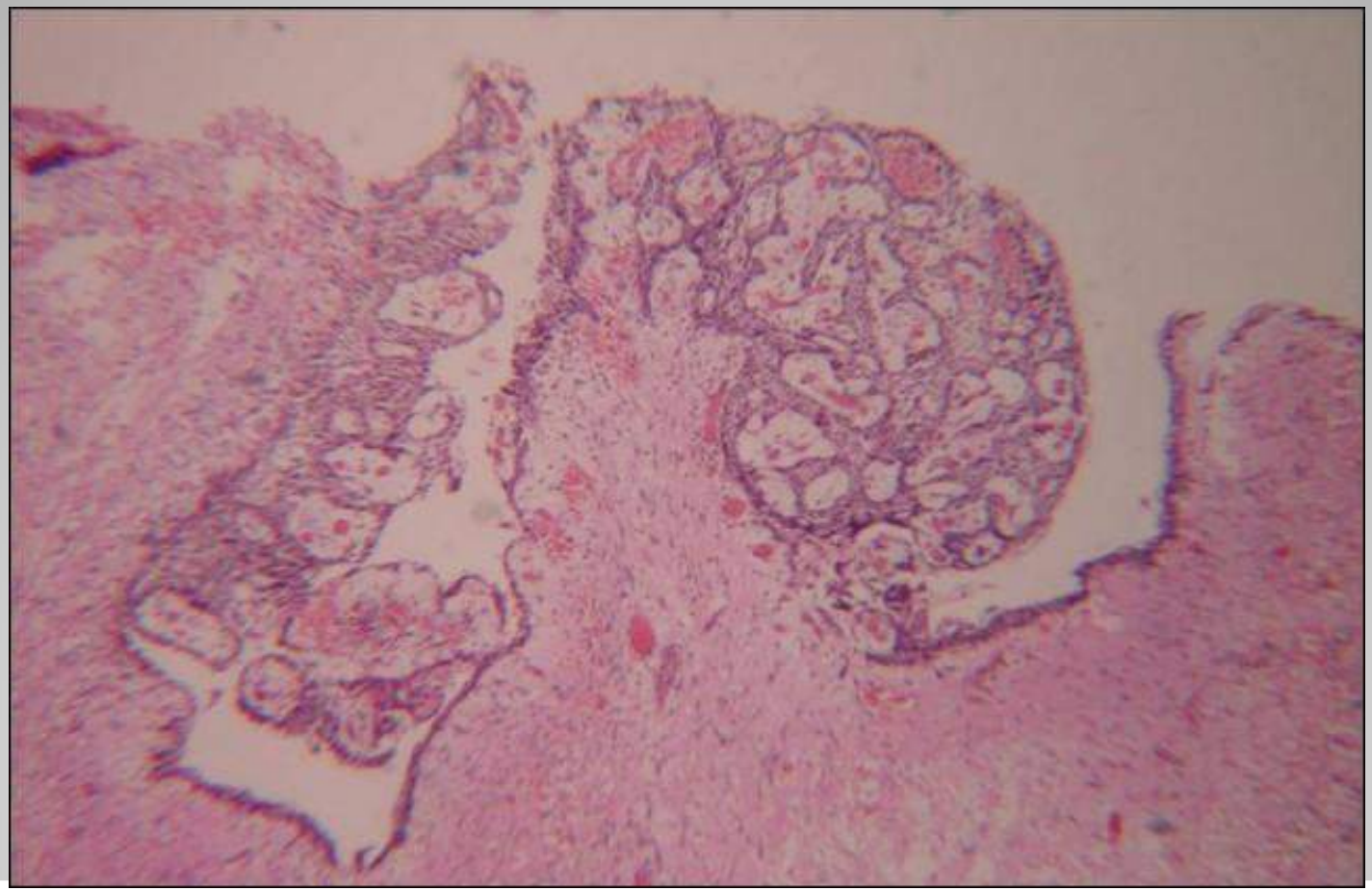

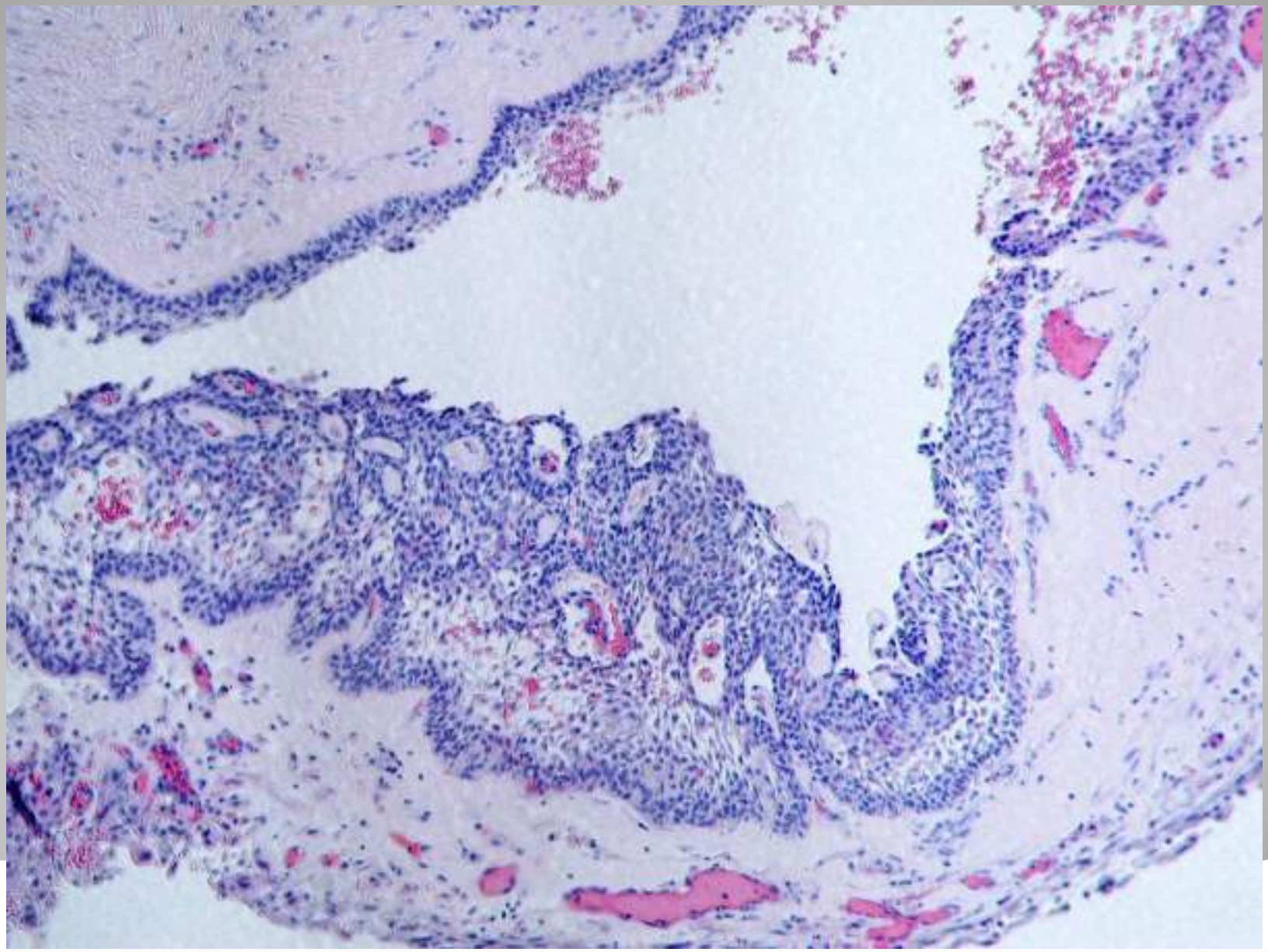

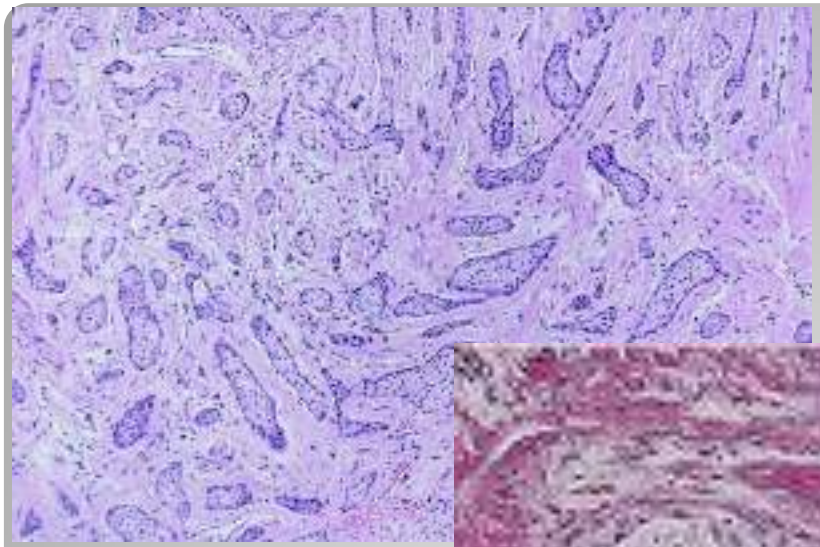

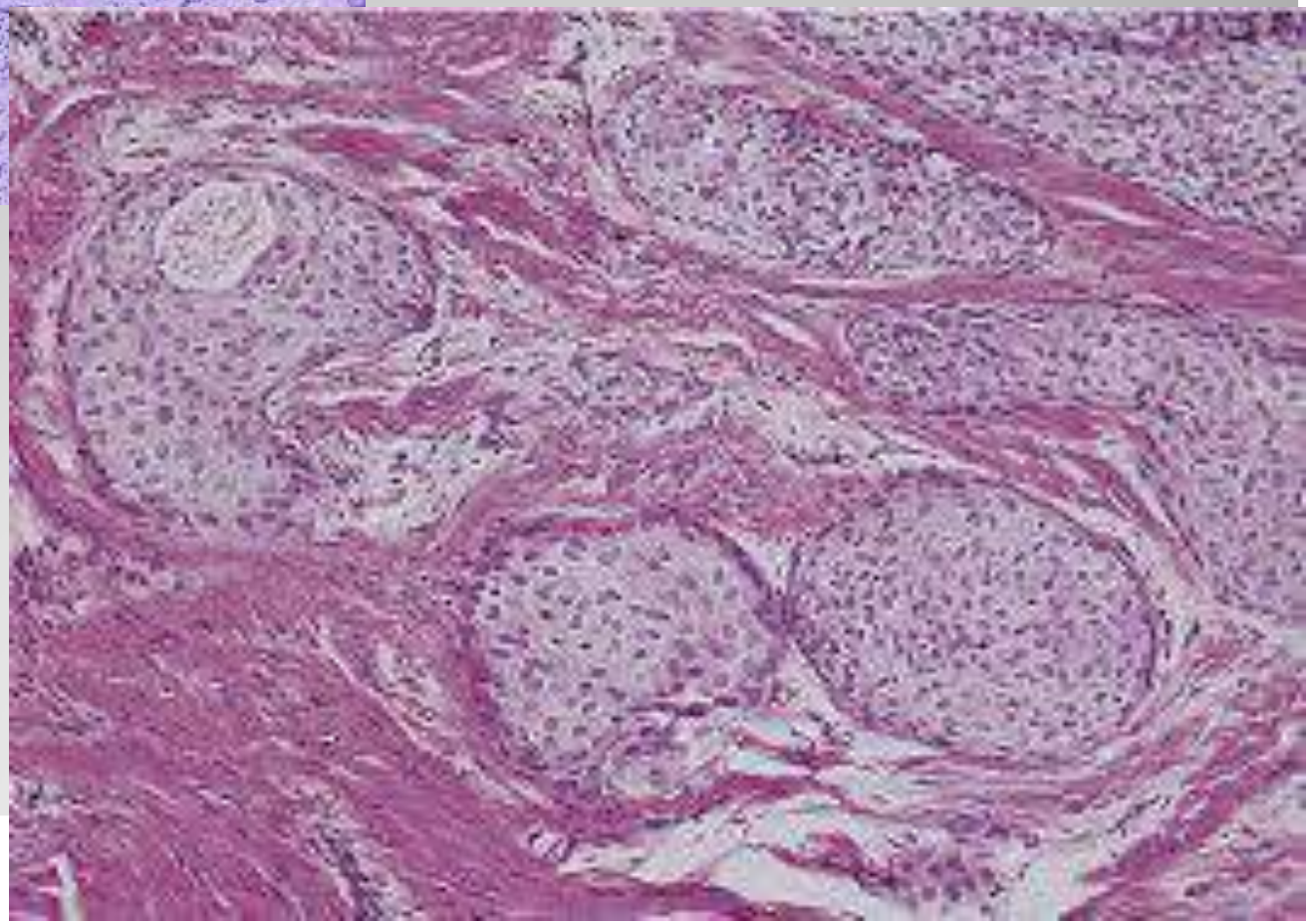

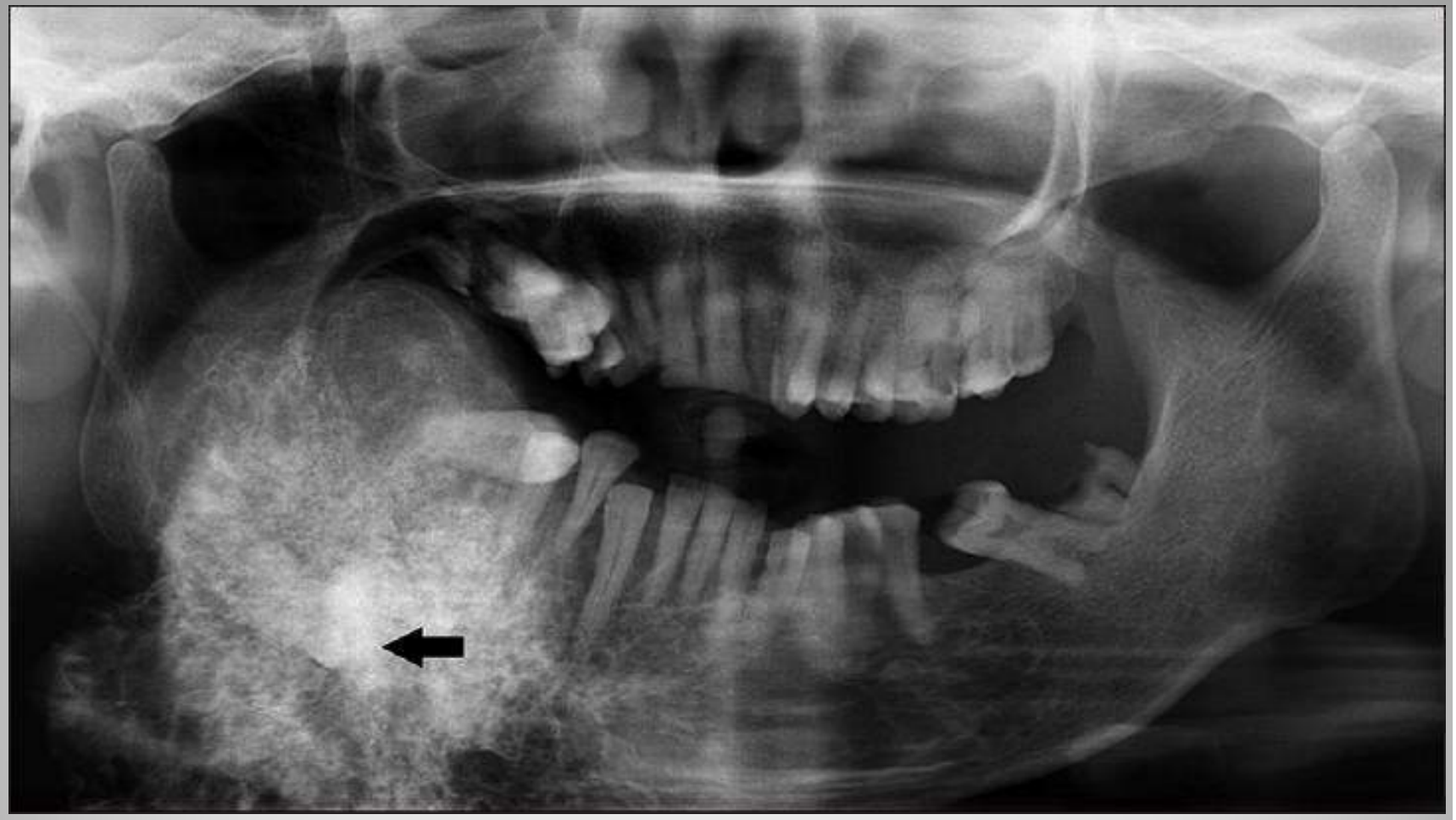

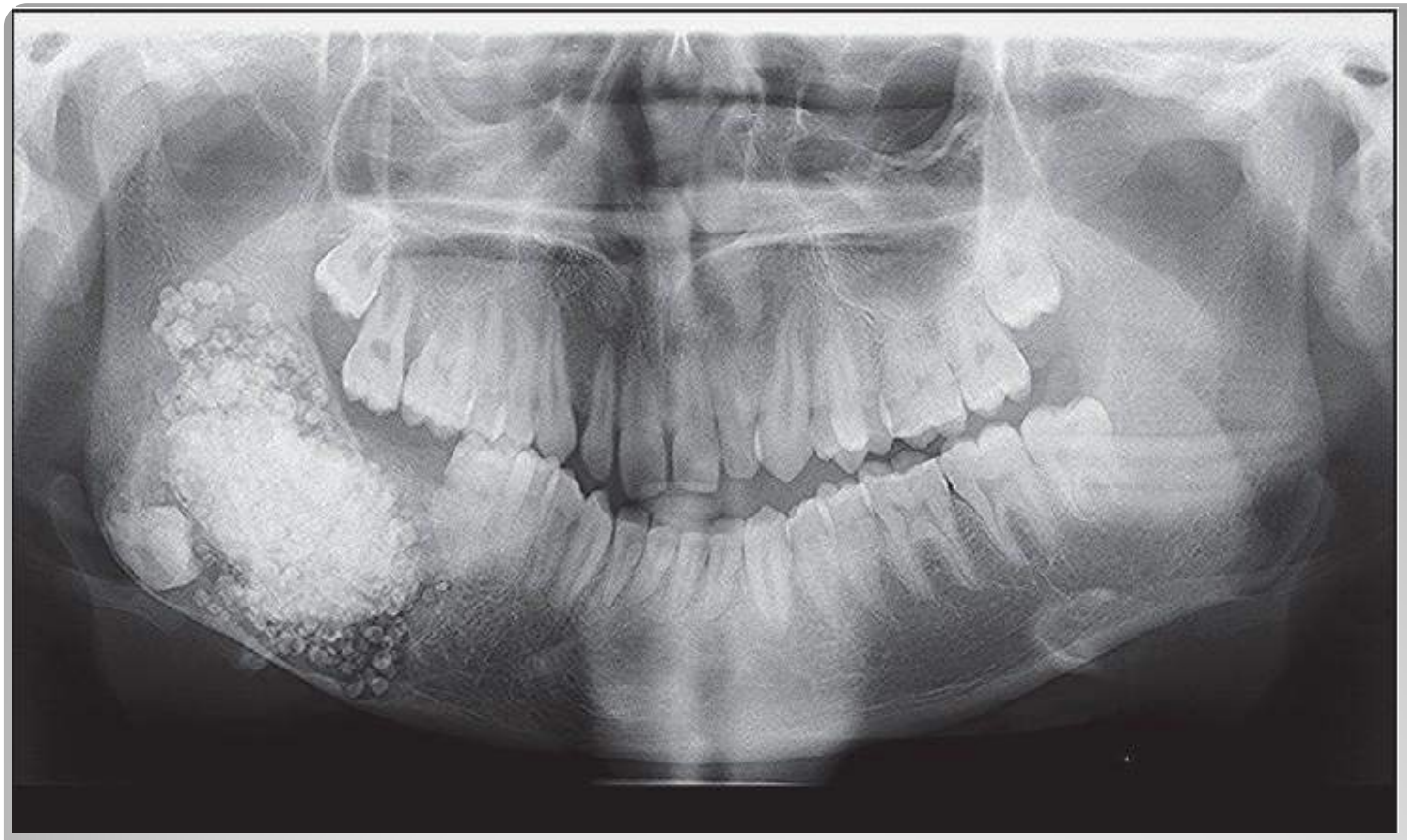

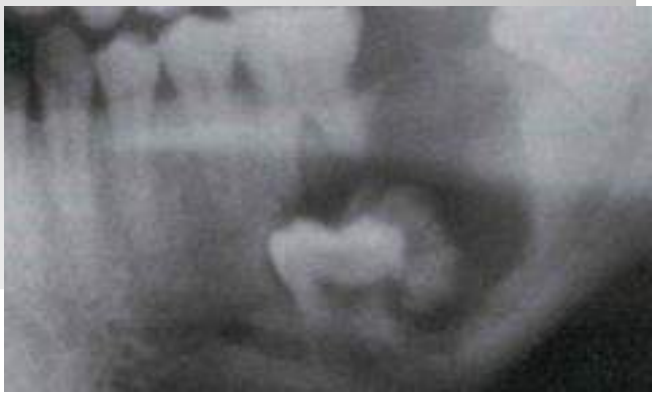

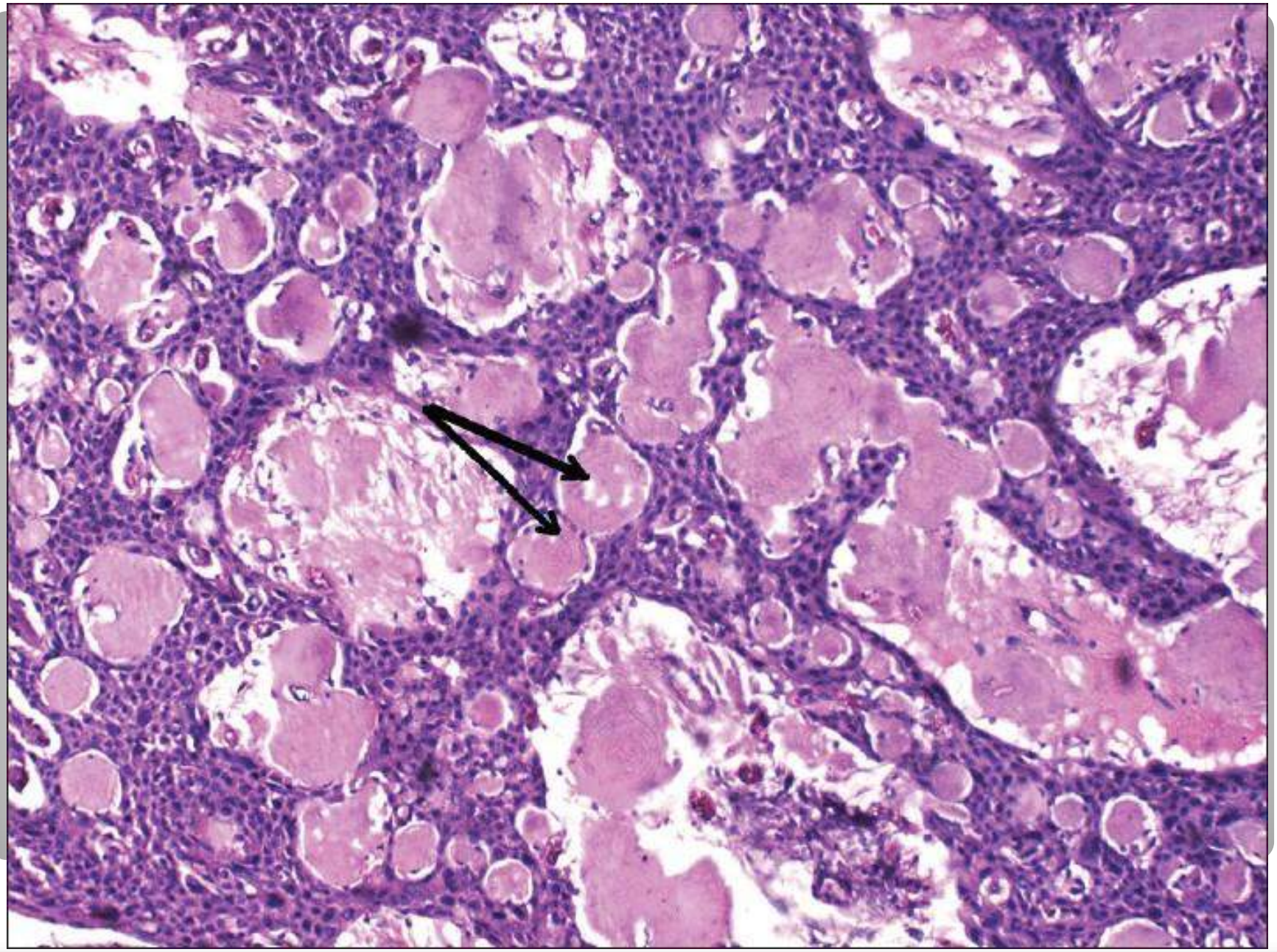

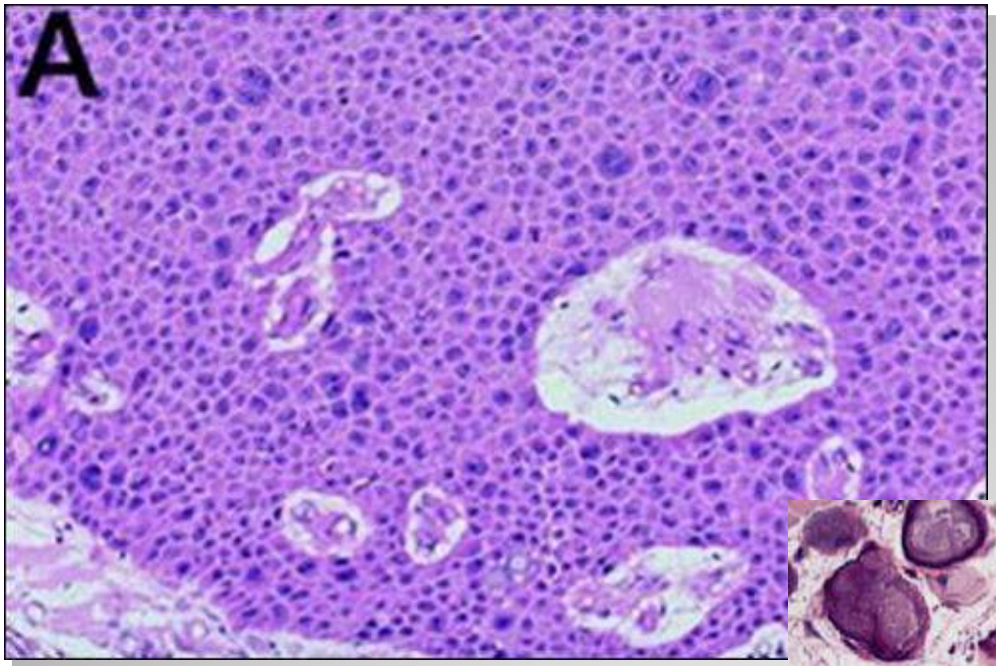
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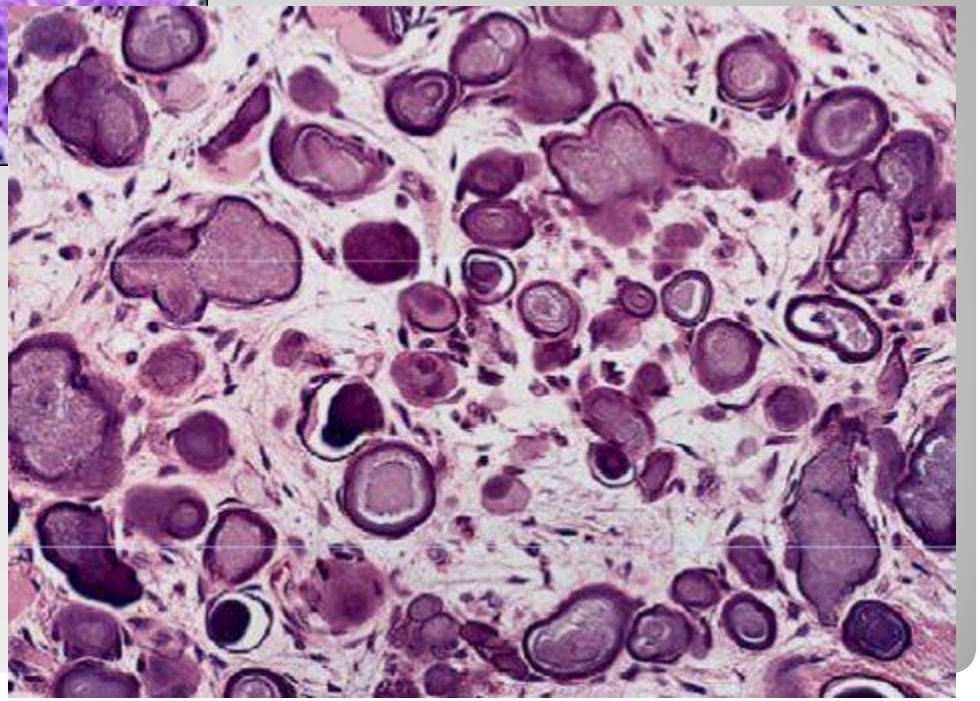

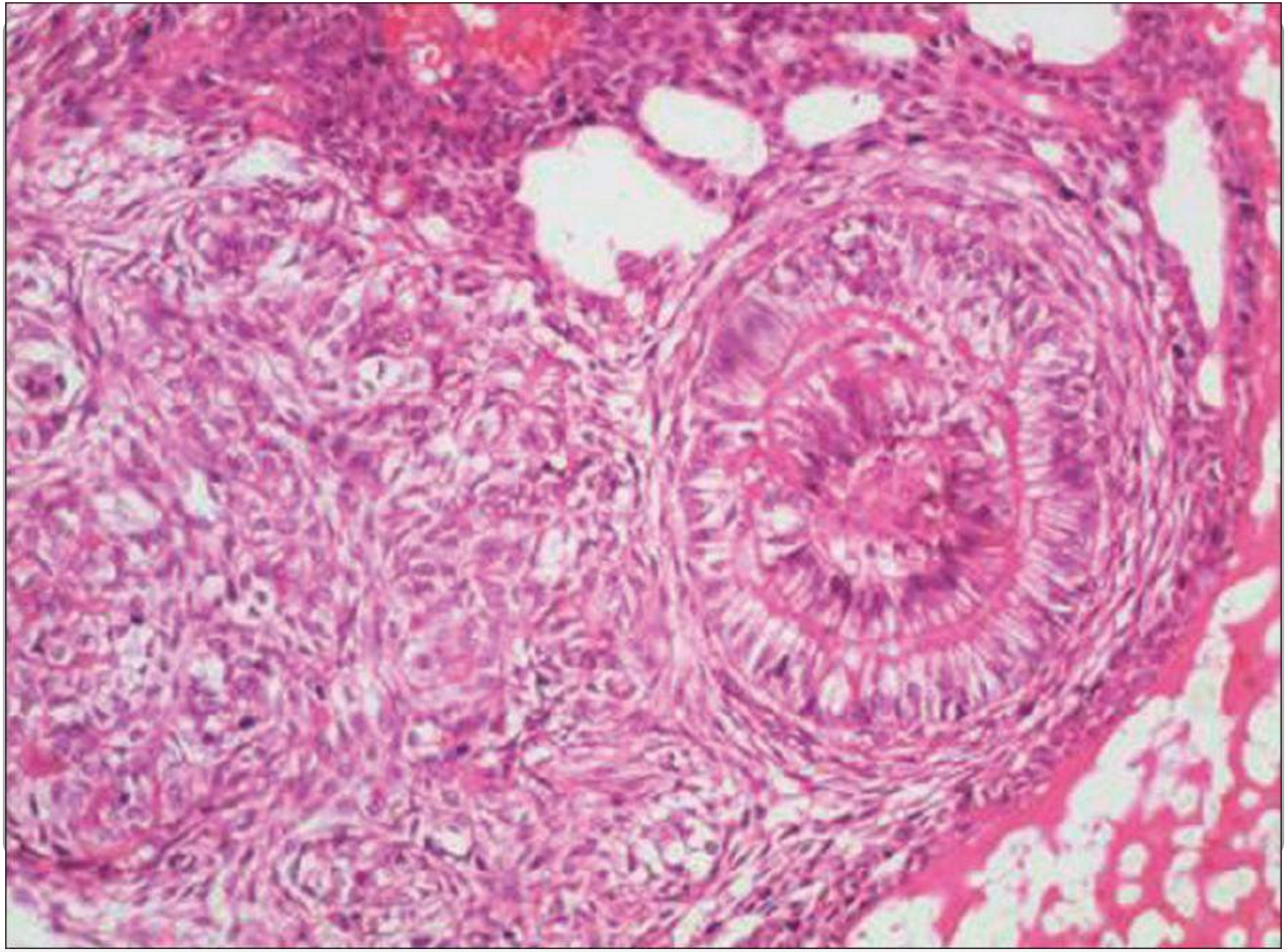

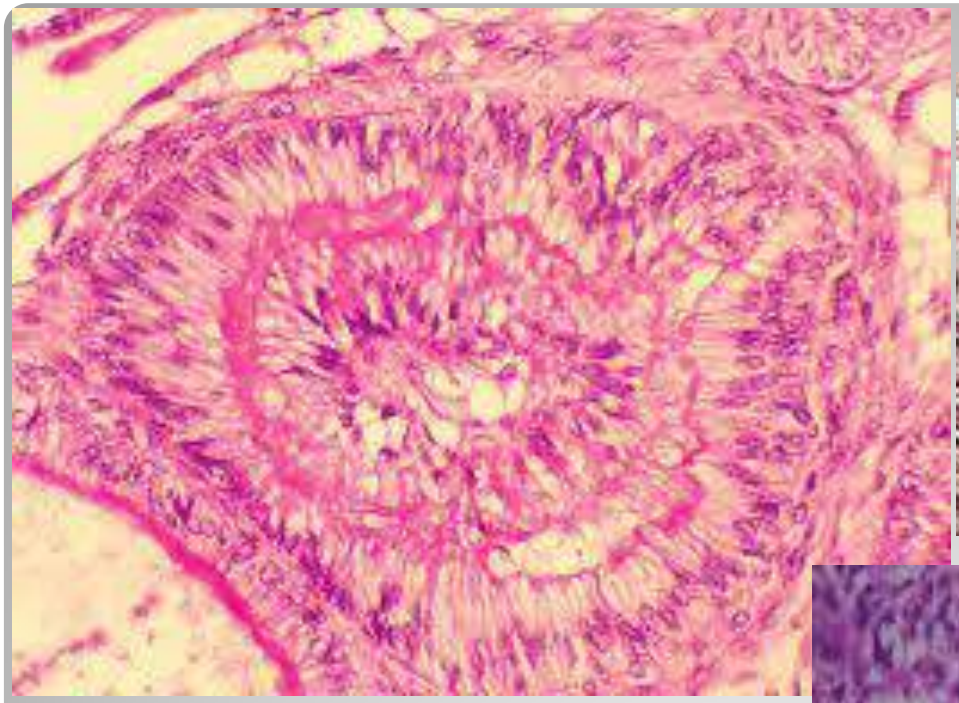

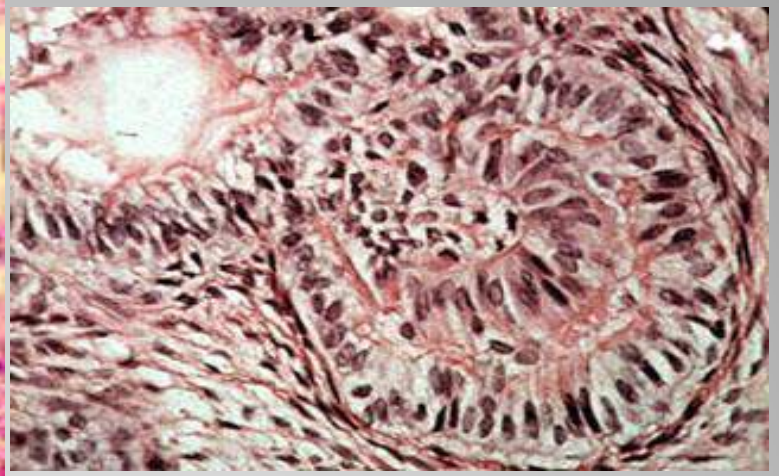

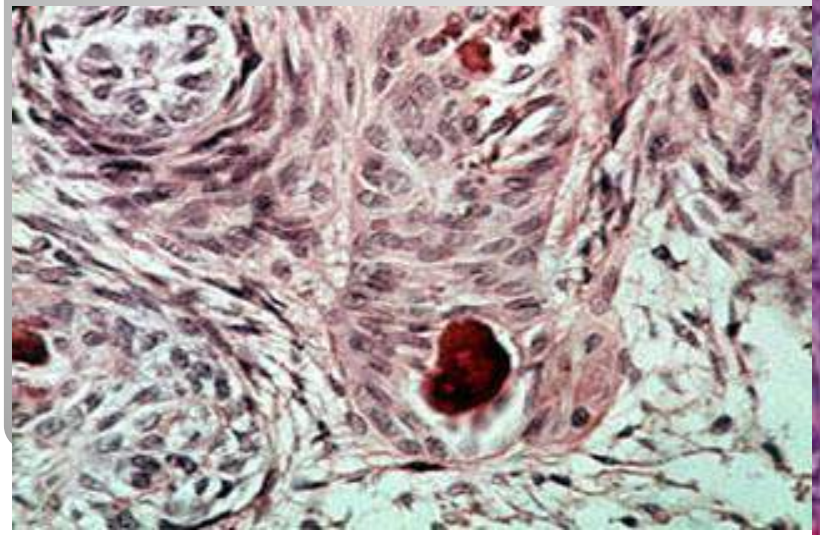

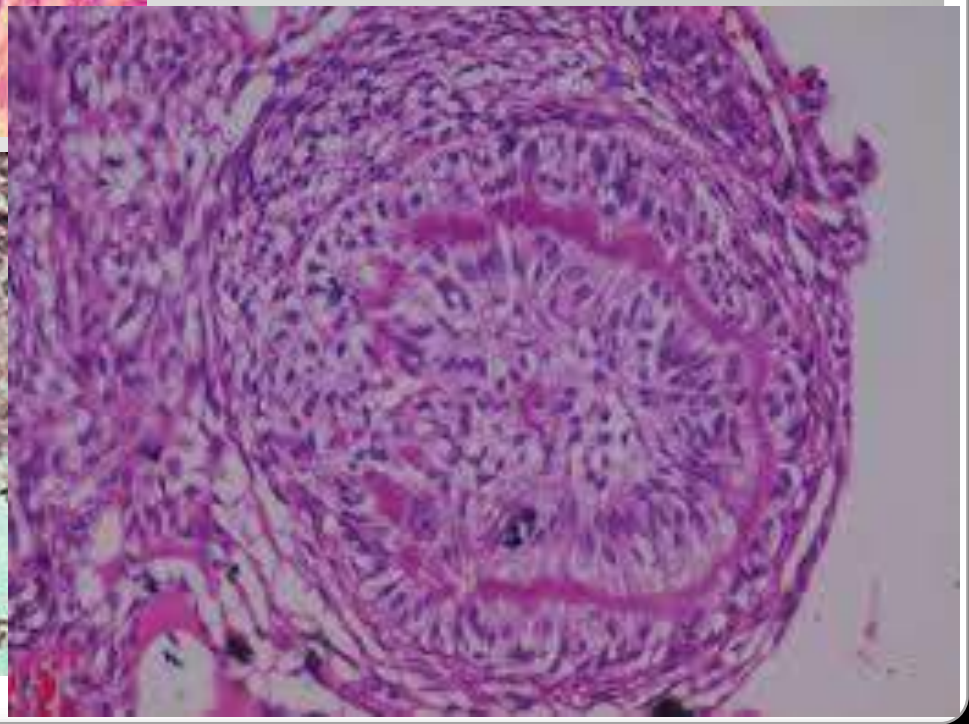

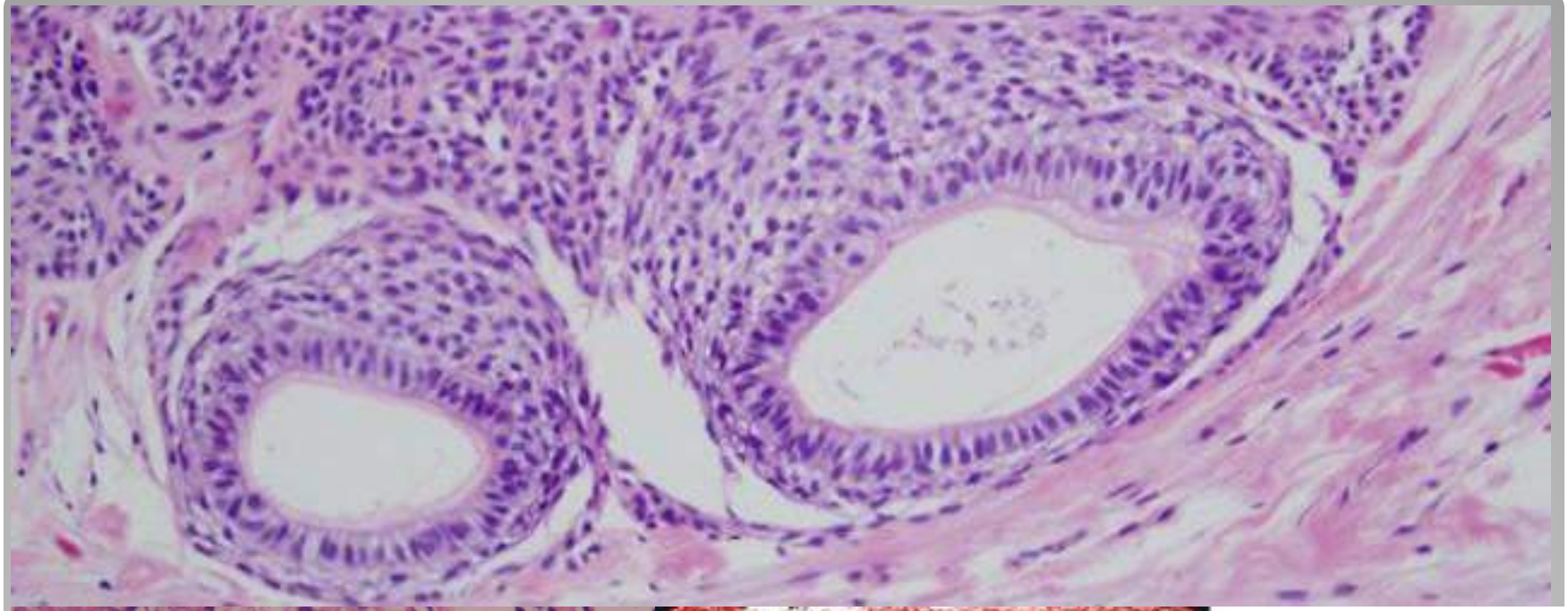

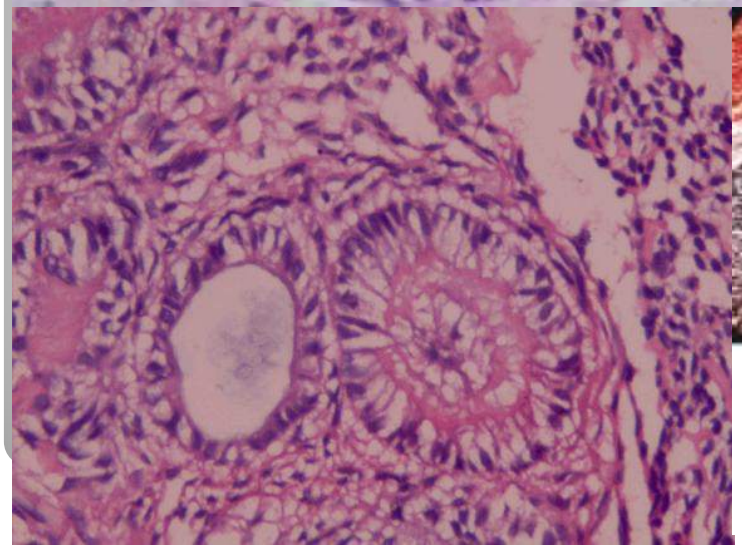

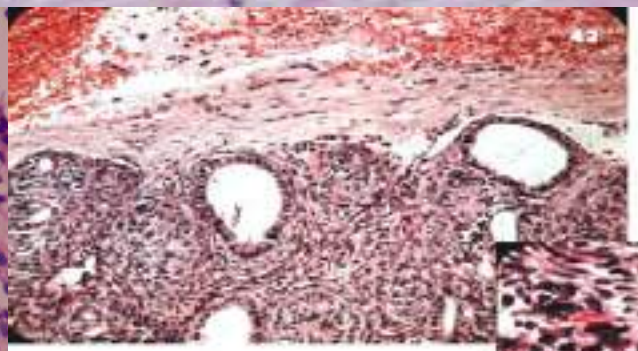

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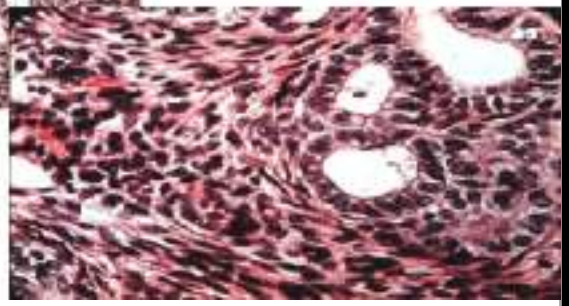

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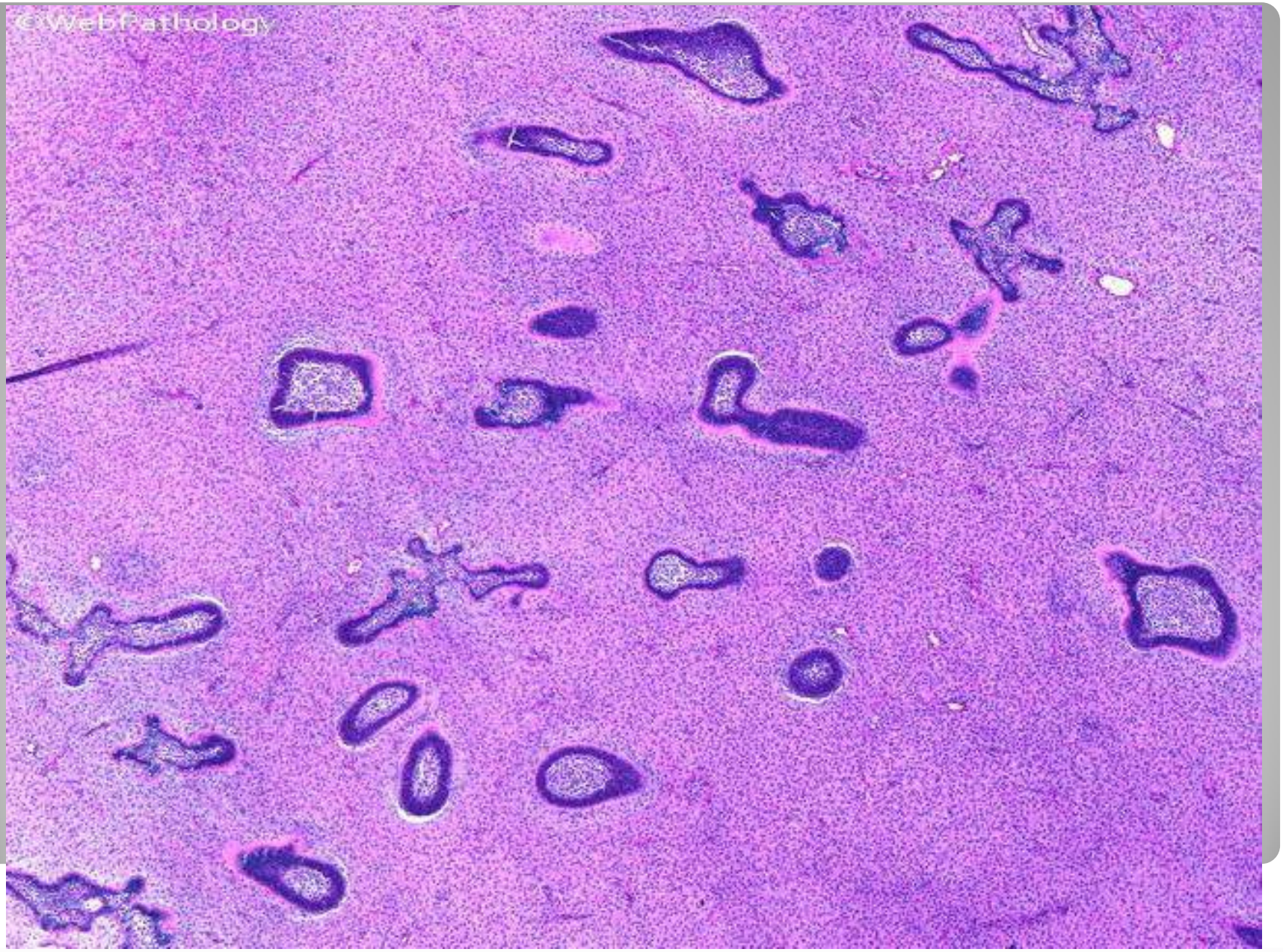
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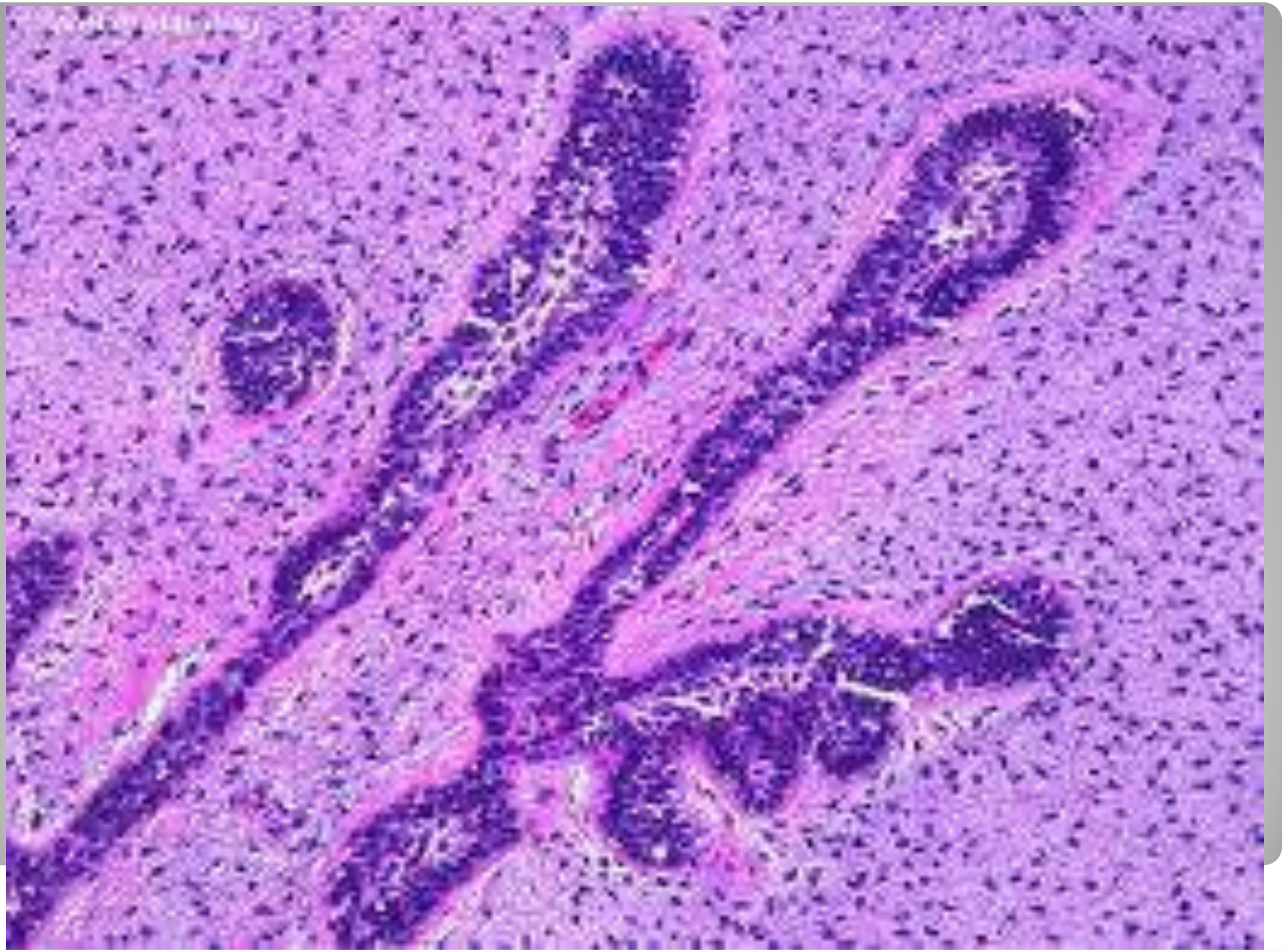

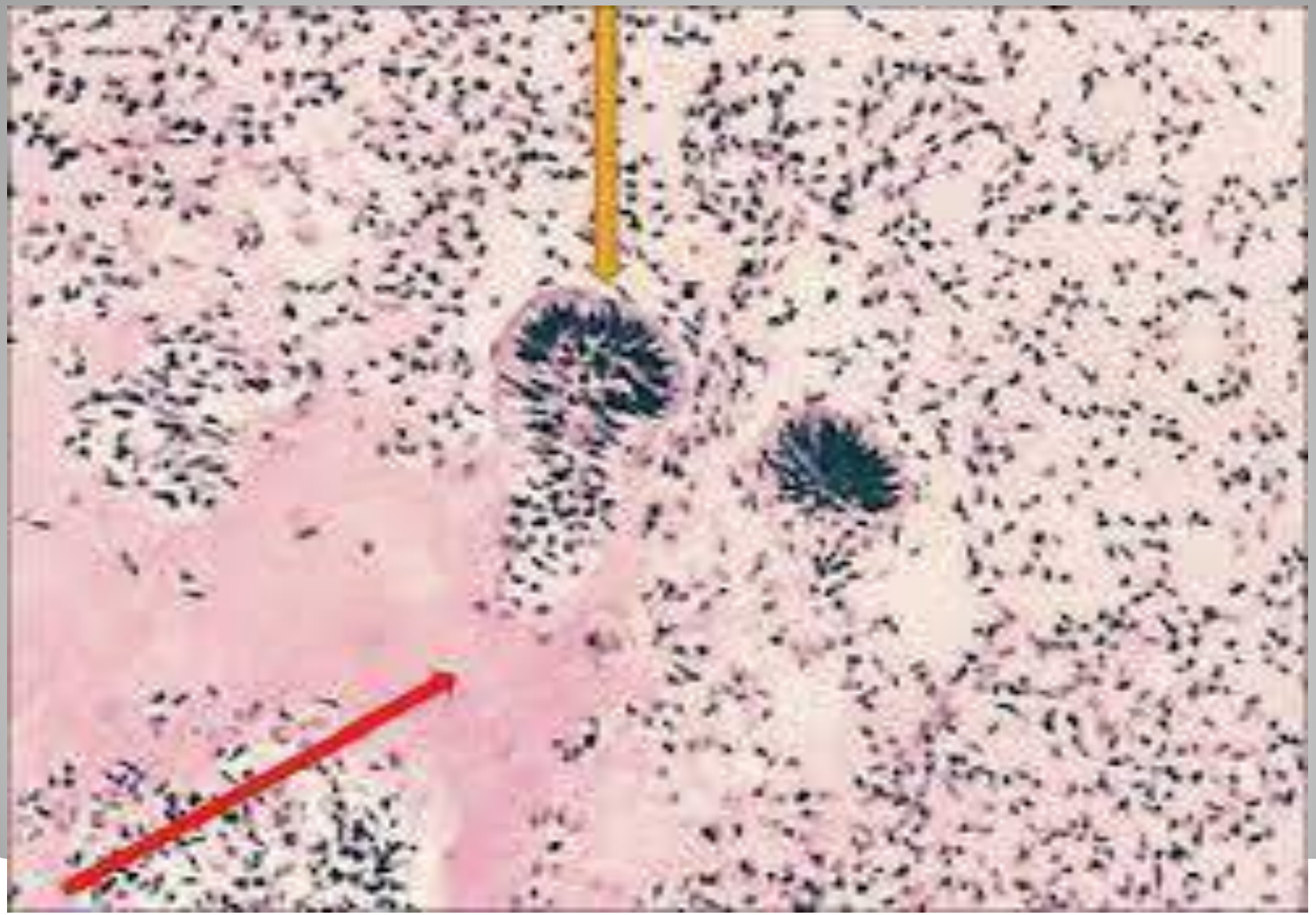

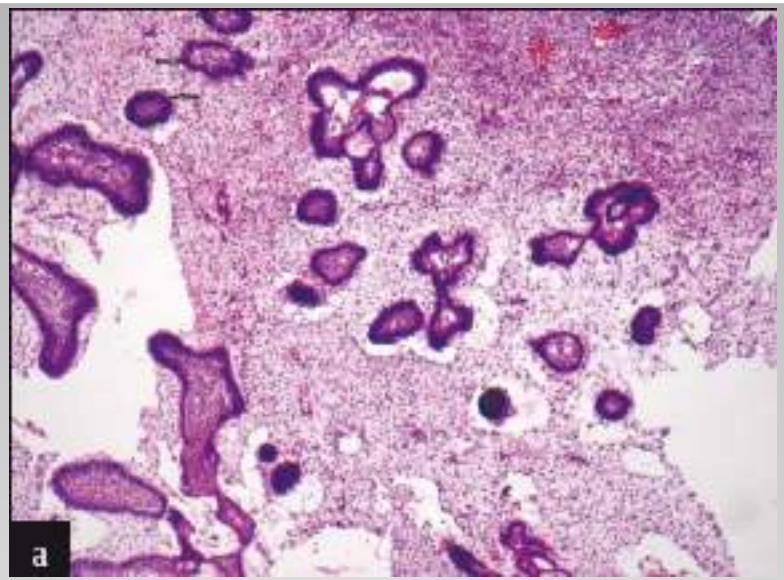
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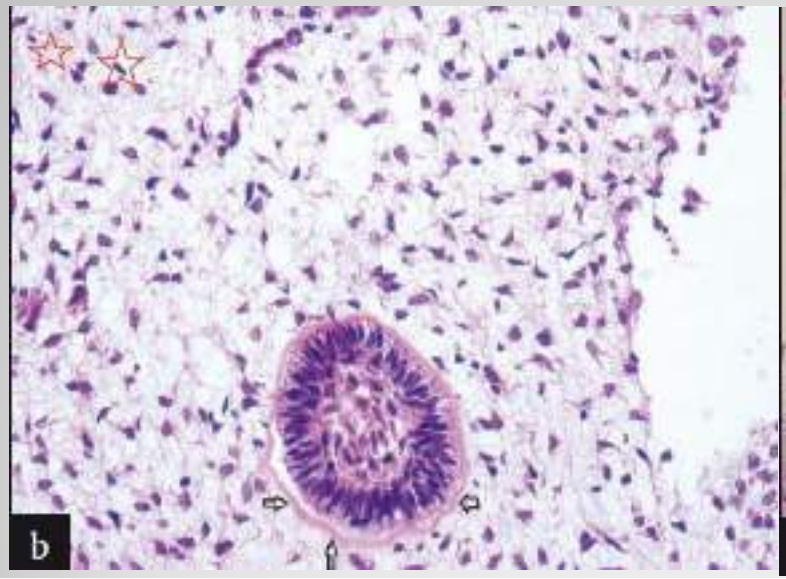
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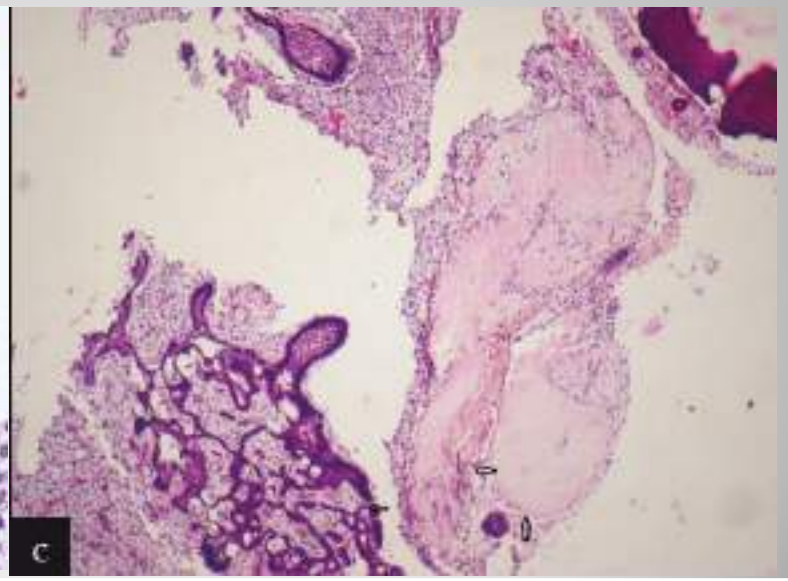
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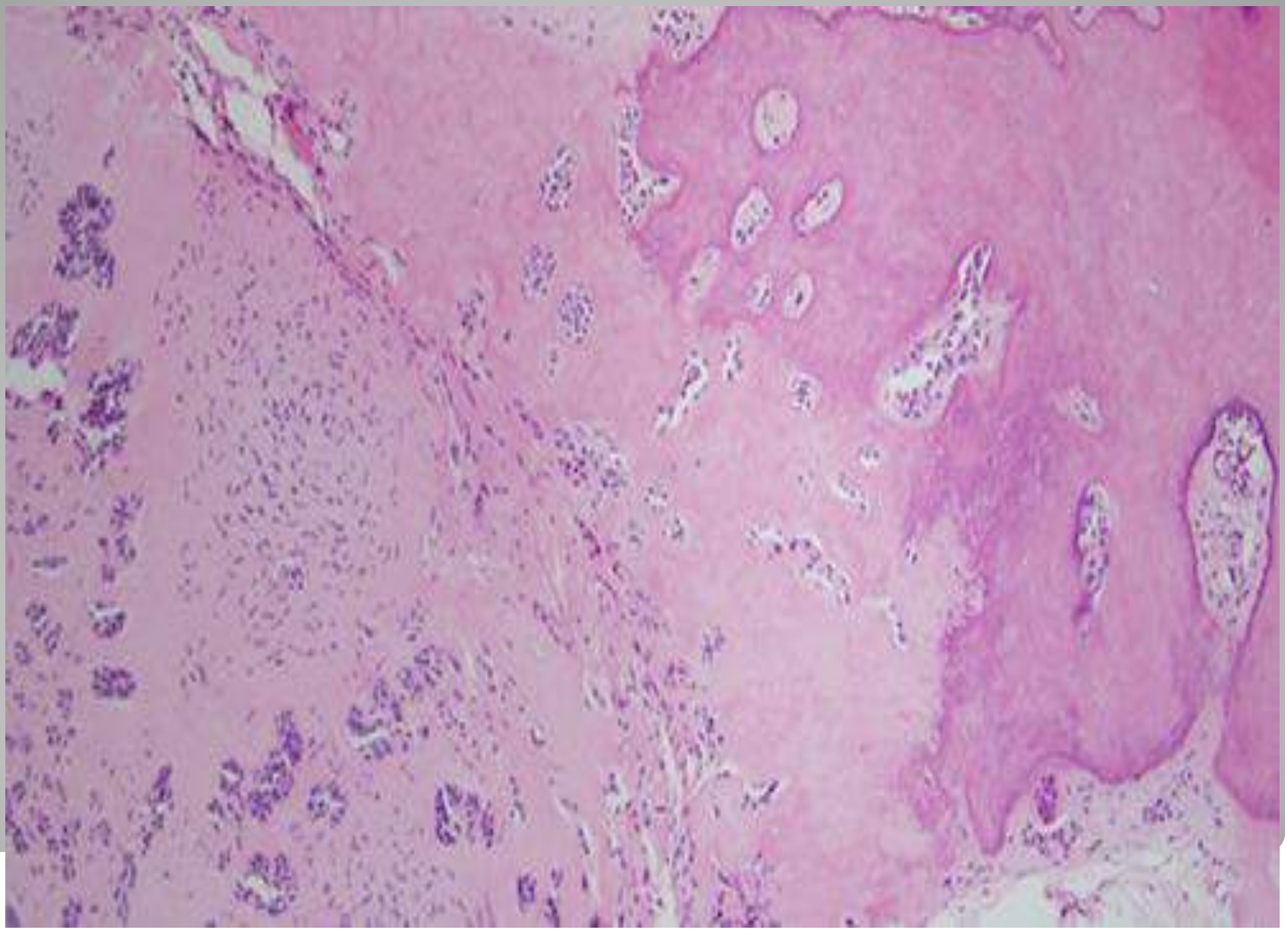

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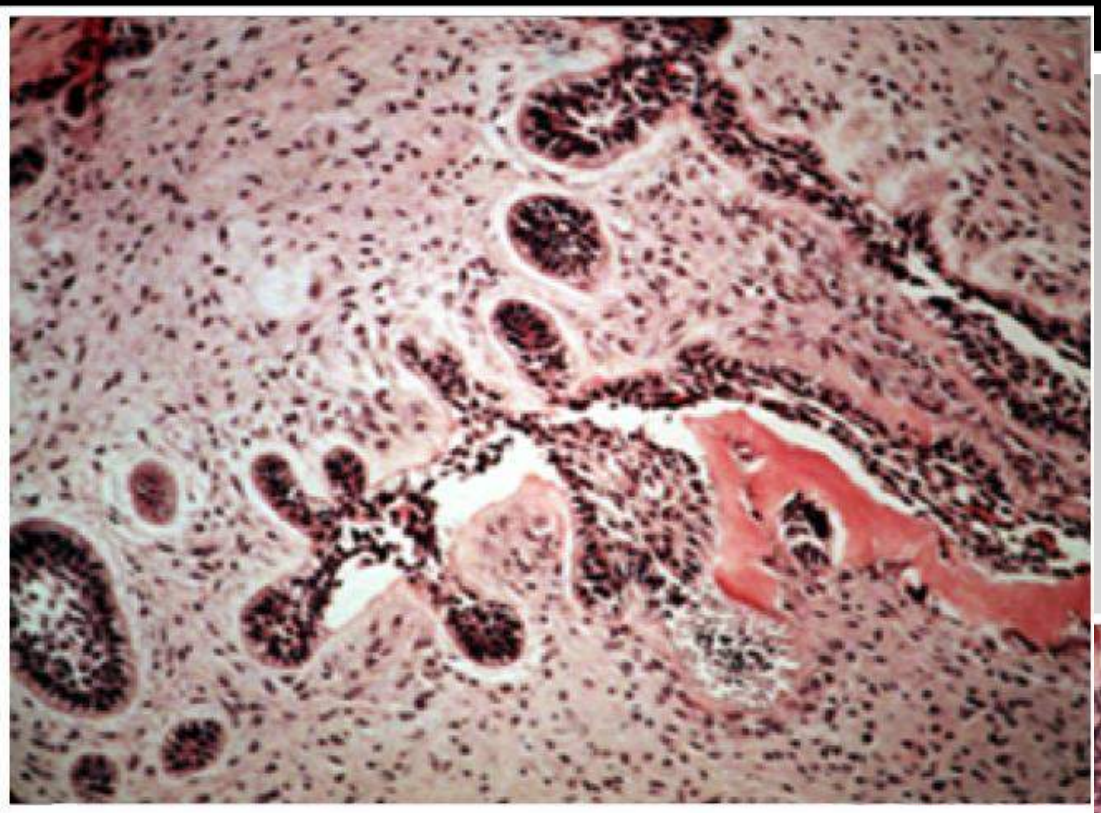
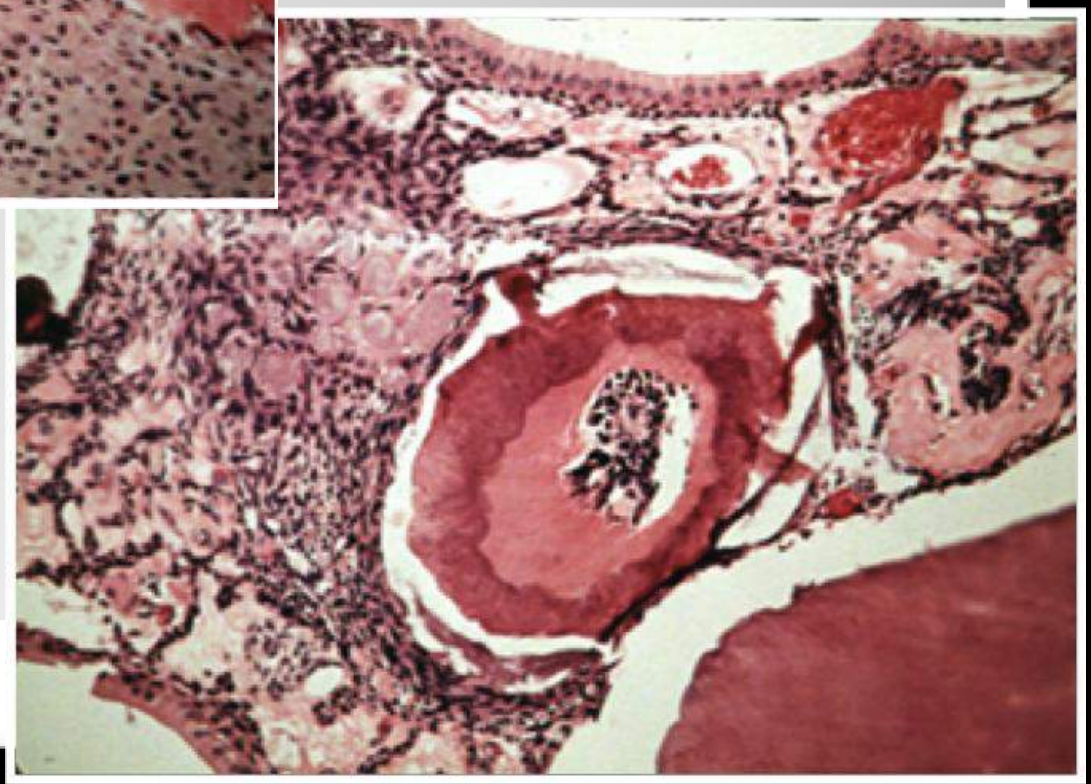
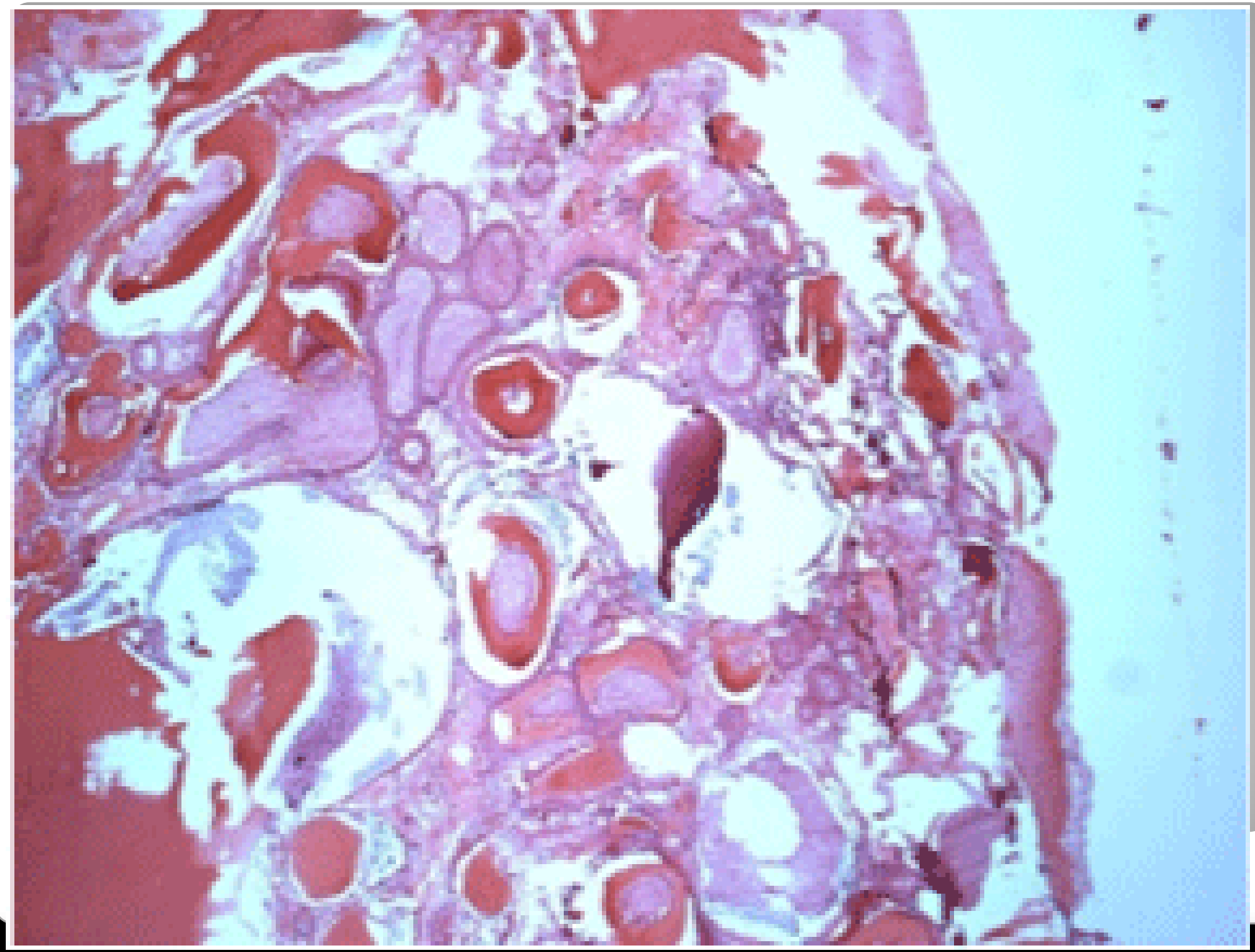
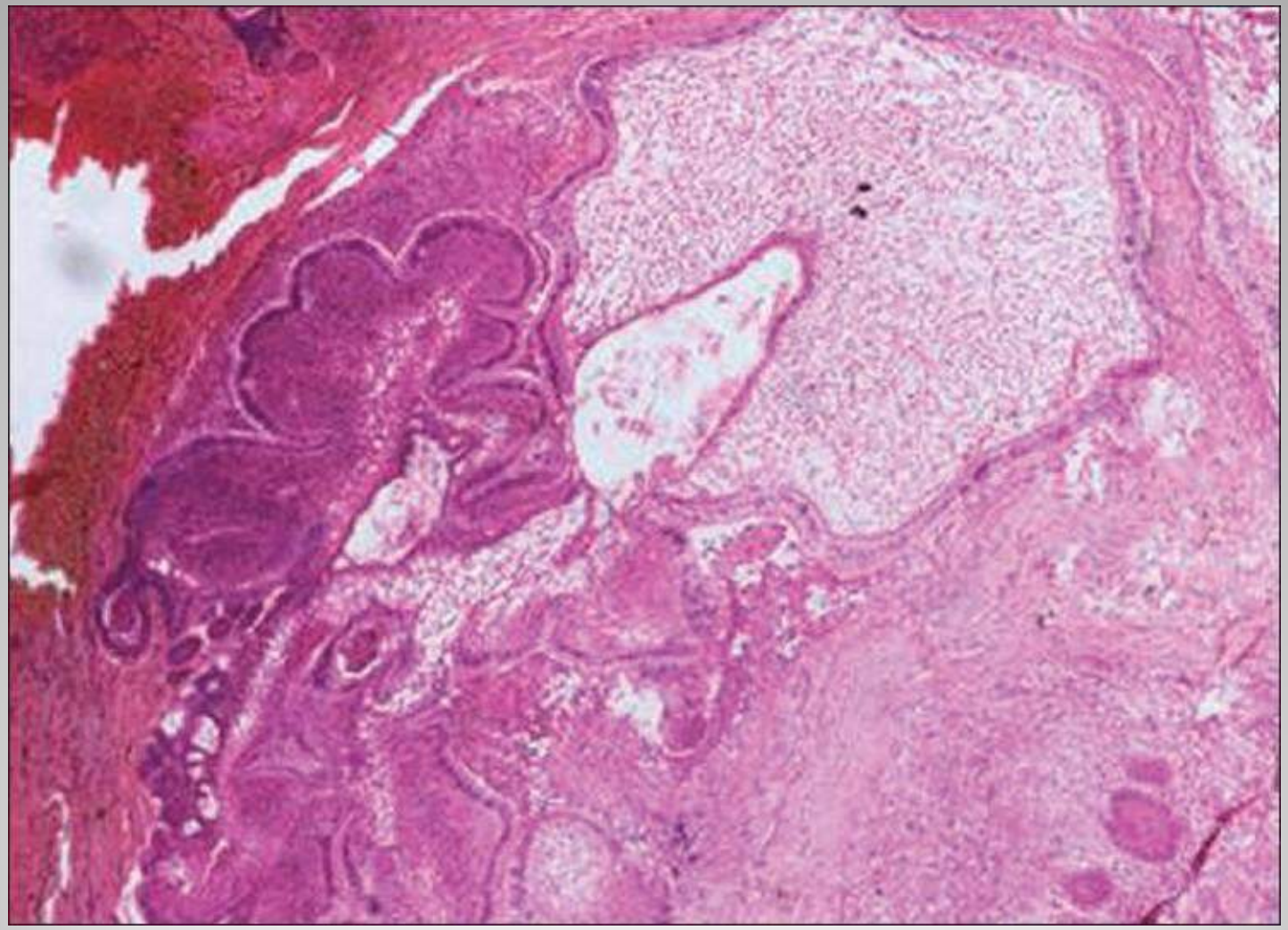
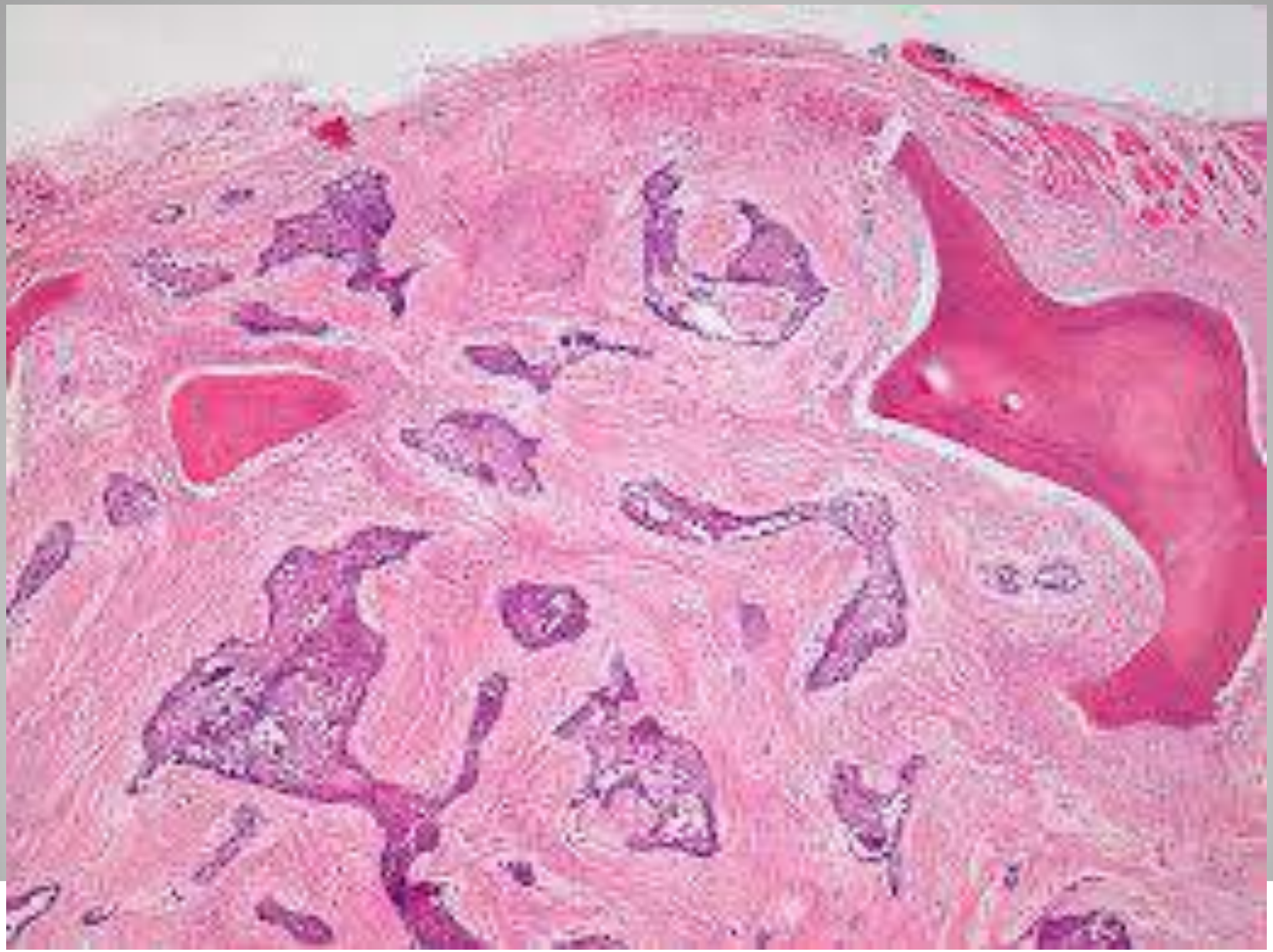










Odontogenic ectomesenchyme with or without included odontogenic epithelium

Odontogenic fibroma

Odontogenic myxoma / myxofibroma

Cementoblastoma

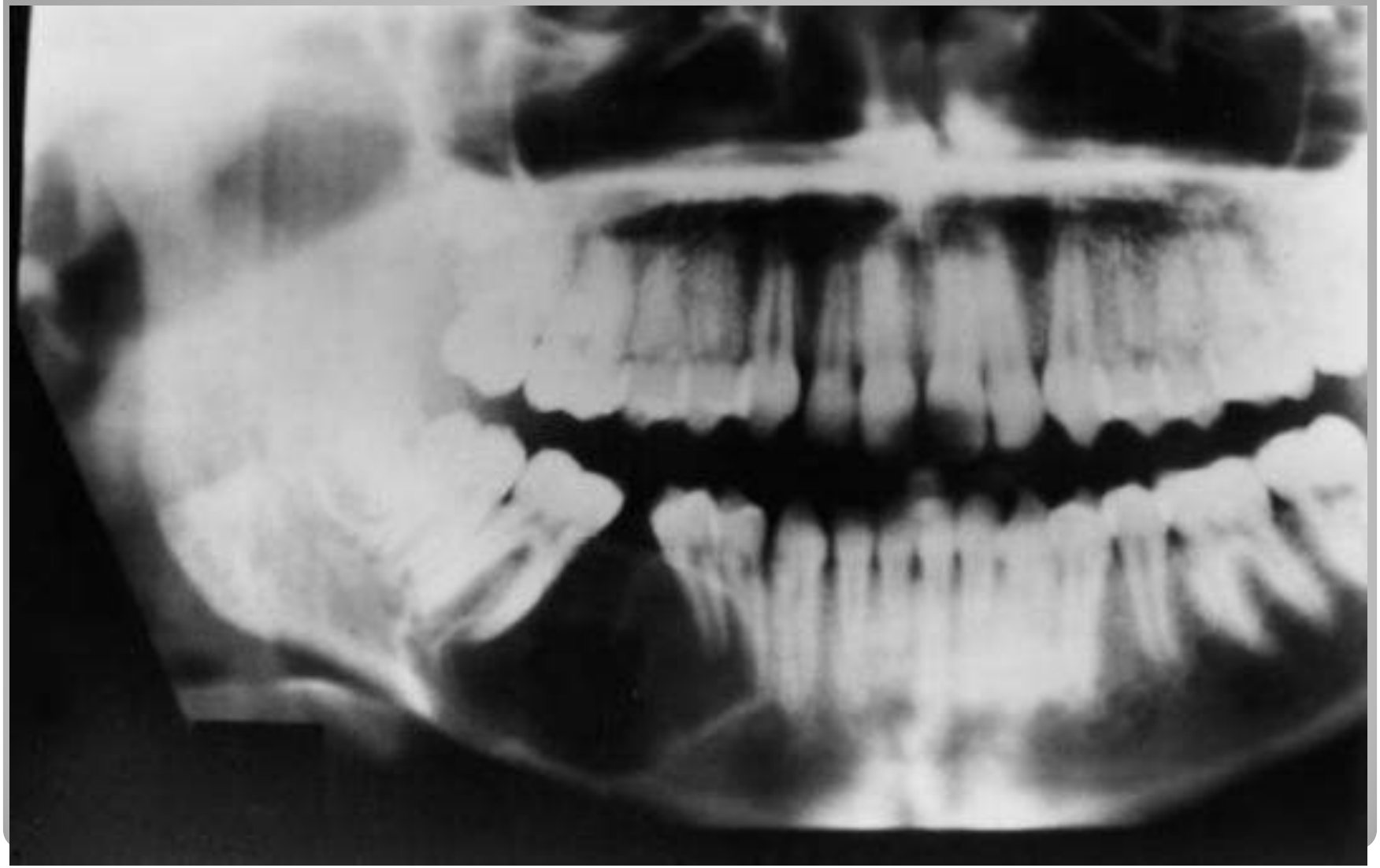
Odontogenic fibroma

Definition

- The odontogenic fibroma (OF) is a rare neoplasm characterized by varying amounts of inactive-looking odontogenic epithelium embedded in a mature, fibrous stroma.
- **ICD-O code 9321/0**
- **Age -40 yrs**
- **Mandible > maxilla**
- **male =Female**
- **Central and peripheral**

- Slow growing
- Painless swelling
- Cortical expansion
- Unilocular radiolucent area with well defined often sclerotic borders
- Rarely, the occurrence of calcified material may
- produce a mixed radiolucent / radio - opaque appearance.
- Adjacent teeth may be displaced.
- Some tumours are associated with the crown of an un erupted tooth.

Clinical features / Imaging



Histopathology

Two histological types of lesions

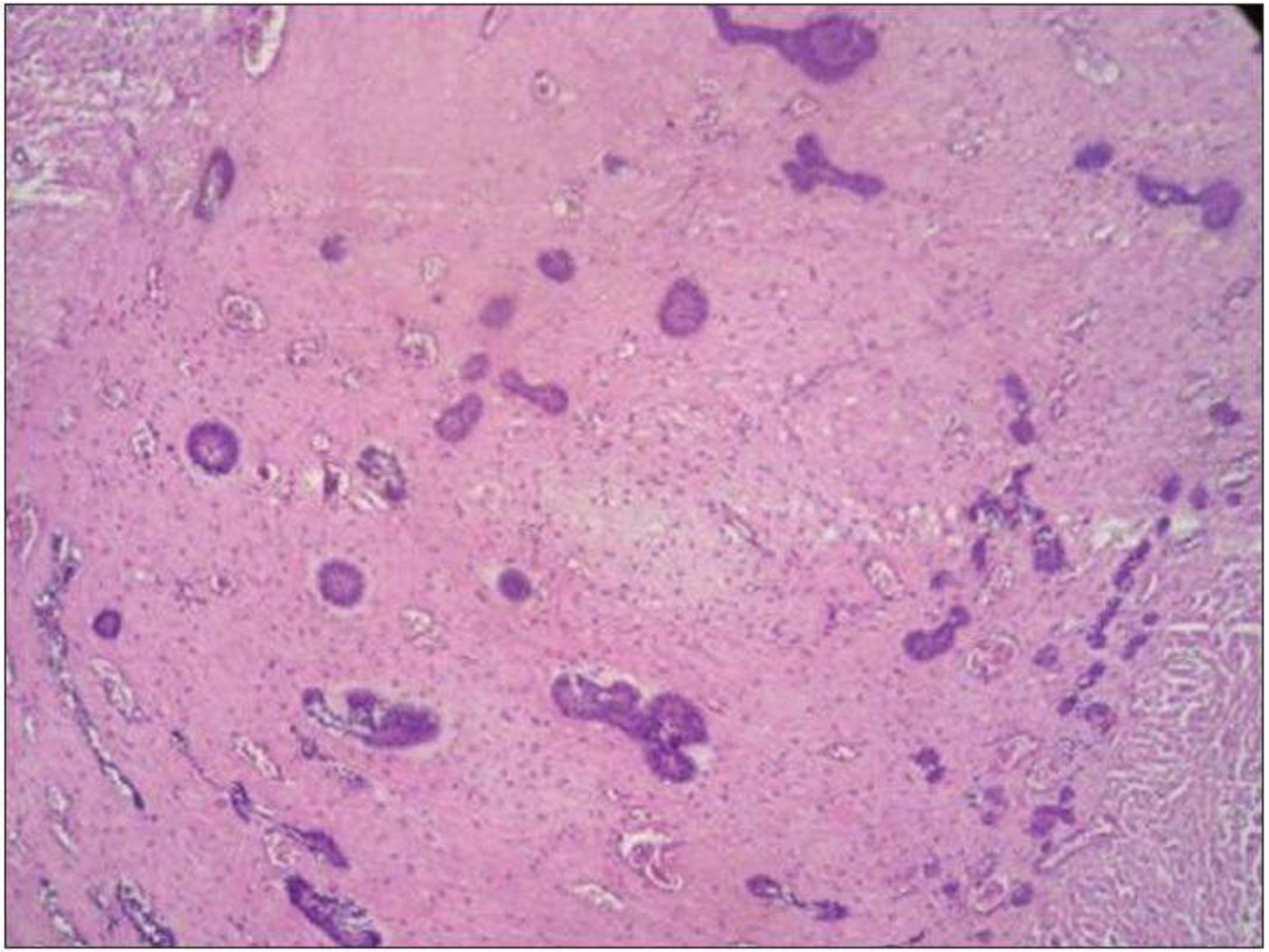
- The epithelium-poor type (formerly termed simple type)
- The epithelium- rich type (formerly termed complex or WHO-type).

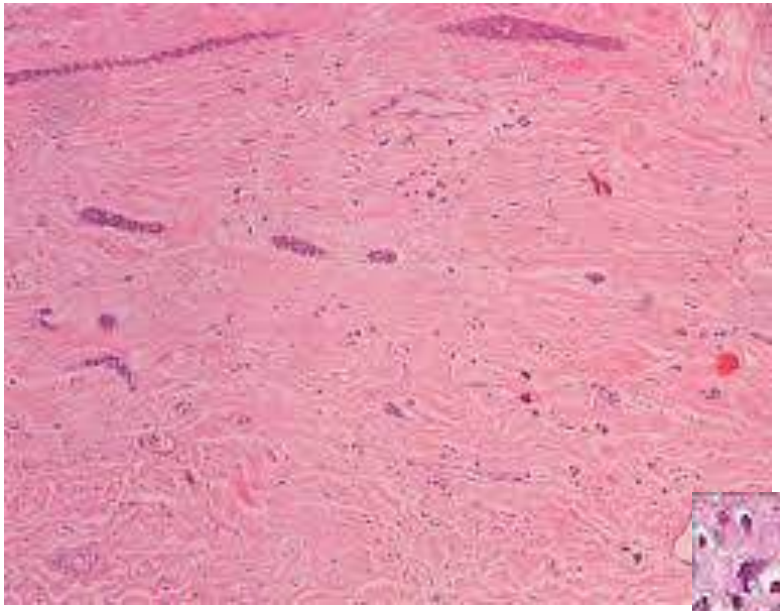
Histopathology

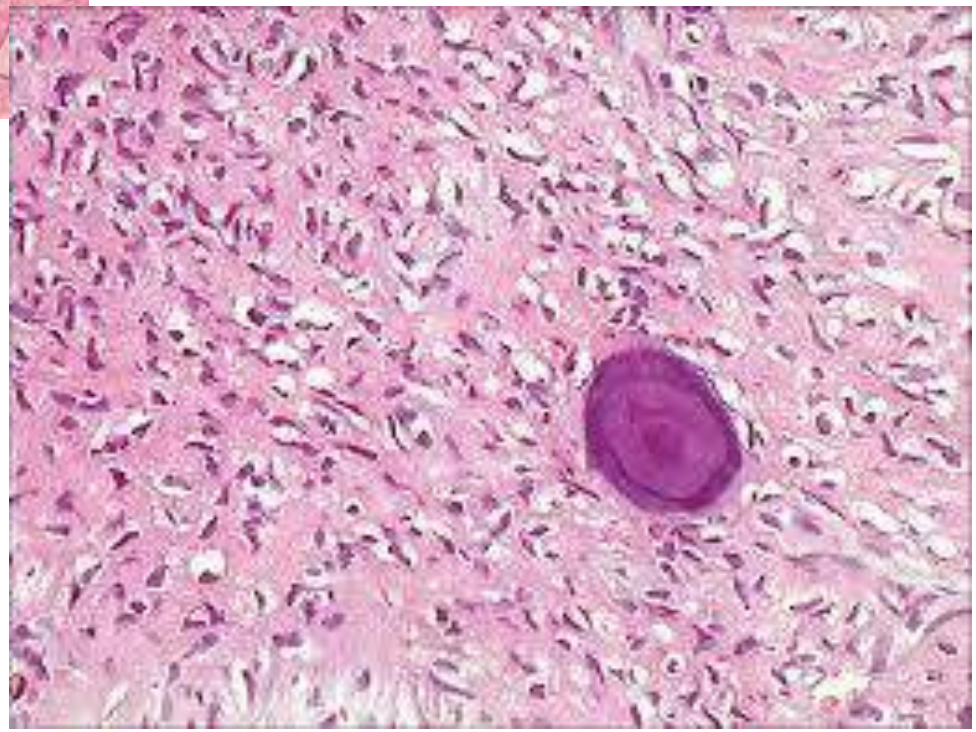
- The epithelium-poor type of COF is a non-infiltrating connective tissue lesion resembling a dental follicle.
- It is minimally cellular with dispersed delicate collagen fibres.
- A considerable amount of ground substance produces a fibro-myxoid quality to the background.
- Scattered remnants of inactive-looking odontogenic epithelium appear as small irregular islands and cords.
- Occasionally, variably- formed calcifications may occur

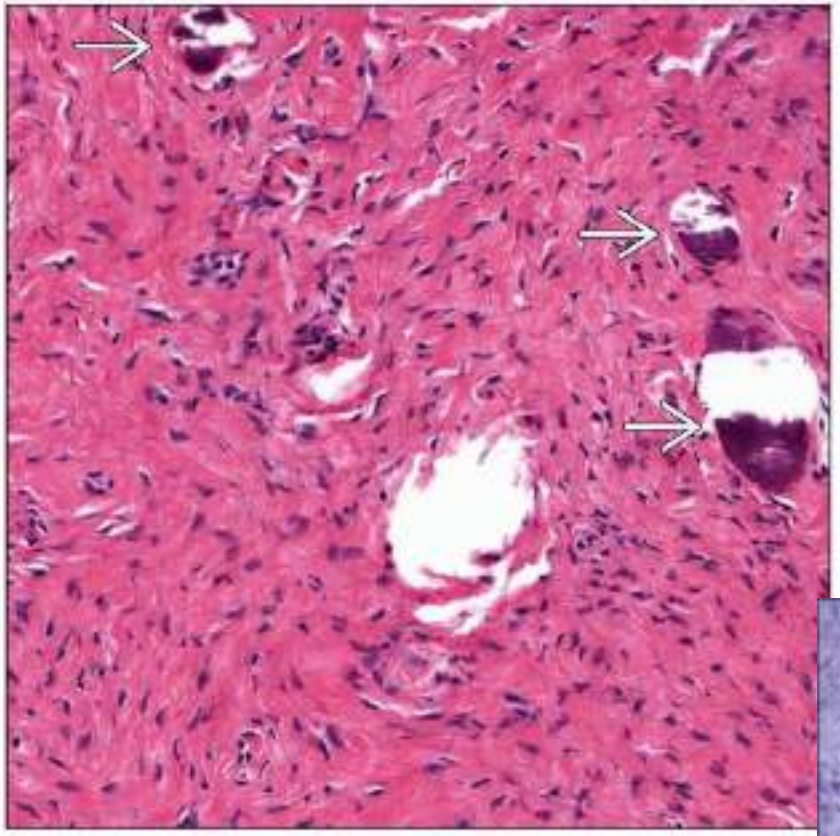
Histopathology

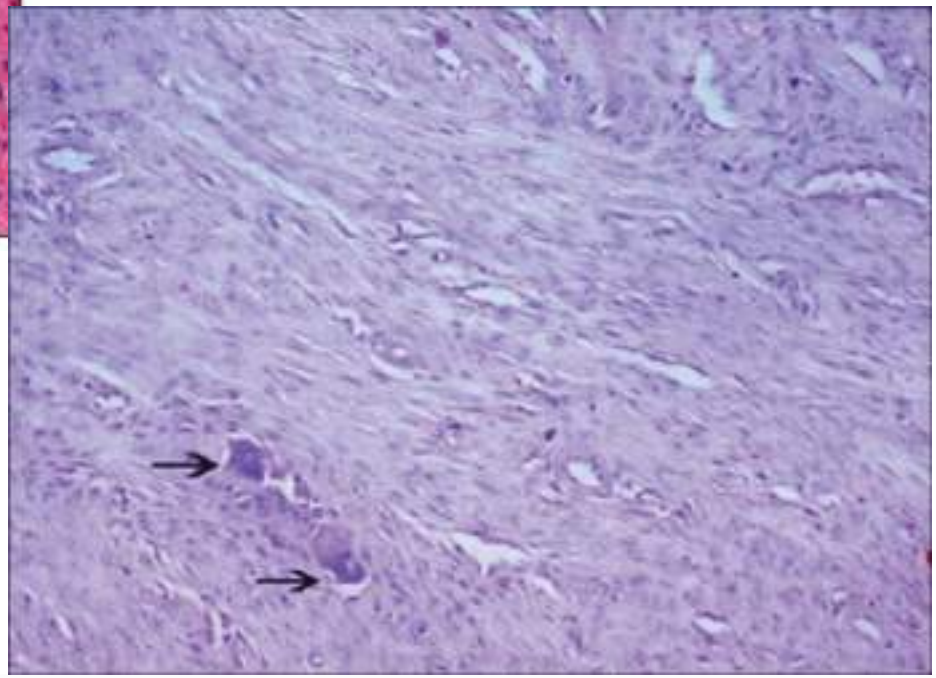
- The epithelium-rich type of COF is composed of cellular, fibroblastic connective tissue interwoven with less cellular and often vascular areas.
- Islands or strands of inactive-looking odontogenic epithelium are an integral component; they may be sparse but are often conspicuous.
- Foci of calcified material considered to be metaplastically produced dysplastic cementum/osteoid/ dentin.











- **Definition**

Odontogenic myxoma (OM) is an intraosseous neoplasm characterized by stellate and spindle-shaped cells embedded in an abundant myxoid or mucoid extracellular matrix. When a relatively greater amount of collagen is evident, the term myxofibroma may be used.

ICD-O code 9320/0

Synonym

- Odontogenic fibromyxoma

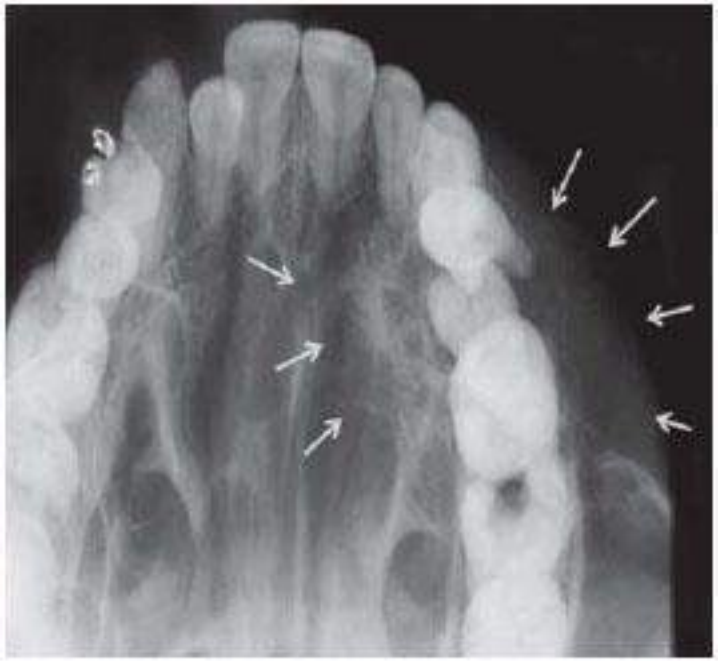
Odontogenic myxoma / myxofibroma

Epidemiology

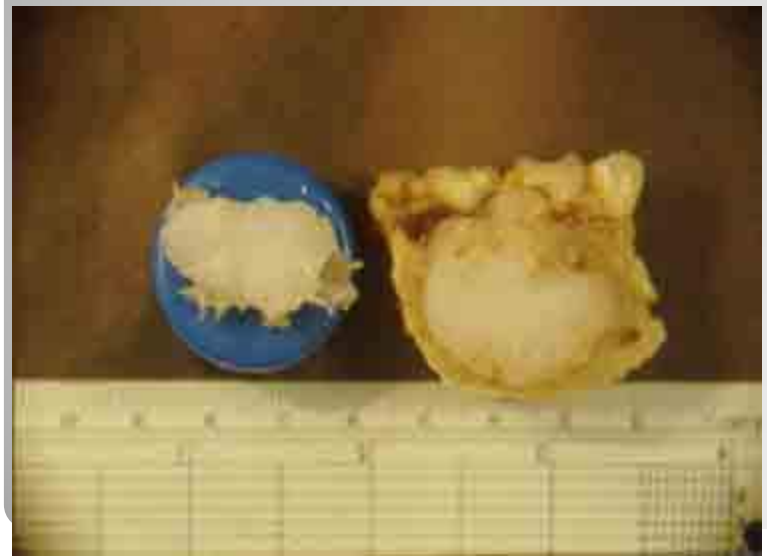
- Third most frequent odontogenic tumour (after odontoma and ameloblastoma).
- Age range varies from 1-73 years, with a mean age of 30 years.
- Slightly more common in females
- Mandible > maxilla

- Smaller lesions are asymptomatic.
- Larger lesions cause painless expansion with cortical perforation
- Unilateral sinonasal obliteration may mimic nasal polyposis (Maxillary involvement).
- Radiographically, unilocular or multilocular radiolucency, sometimes showing a fine “soap bubble” or “honeycomb” appearance occasionally with fine trabeculations.
- The borders of the tumour are usually well-defined
- Root displacement occurs, as does root resorption.

Clinical presentation and Imaging



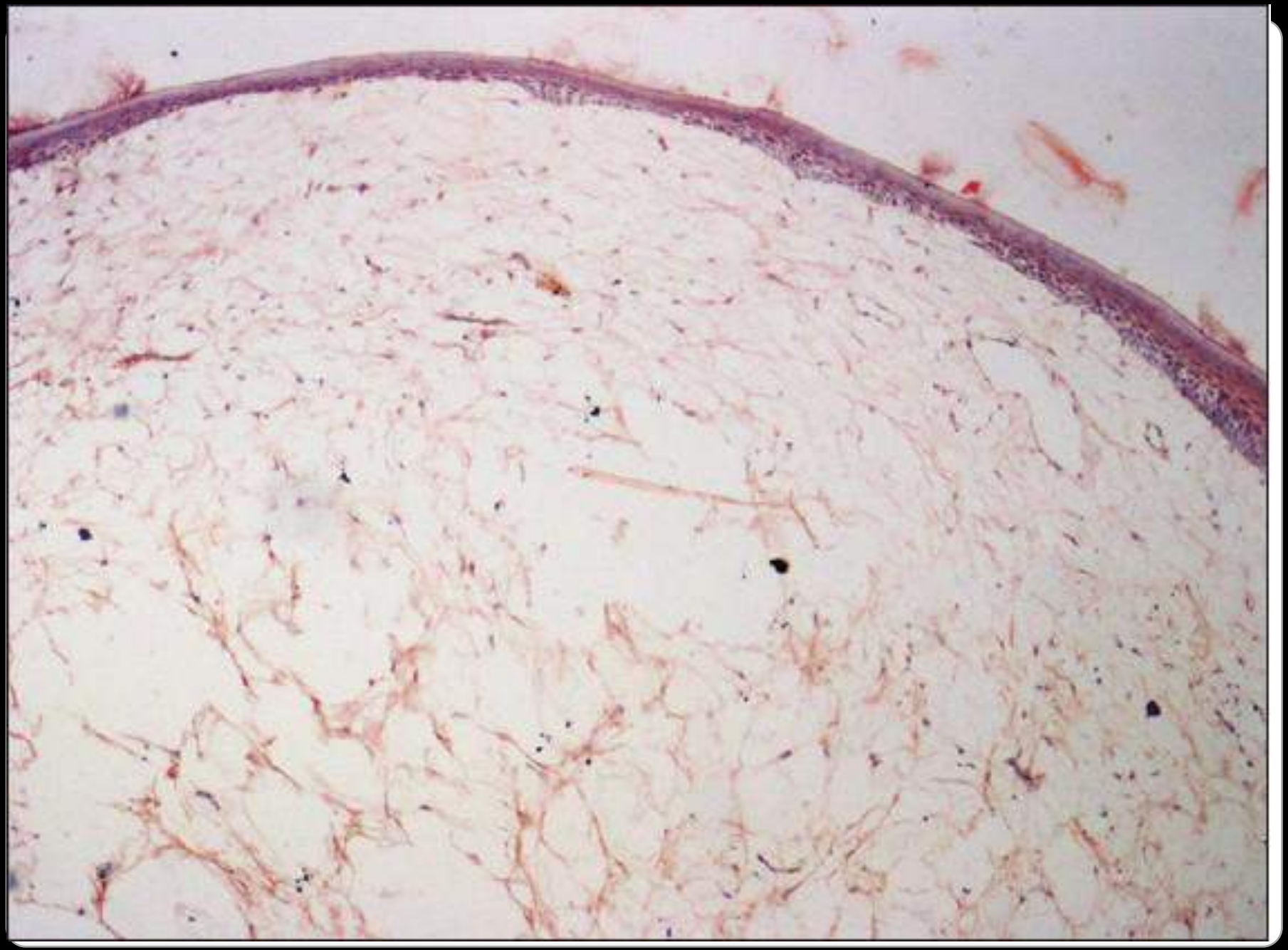
- Gross examination reveals a grey-white mass with a typical translucent mucinous appearance.
- The consistency varies from gelatinous to firm, depending on the amount of collagen present and fine white bands of collagen may be visible on the cut surface.

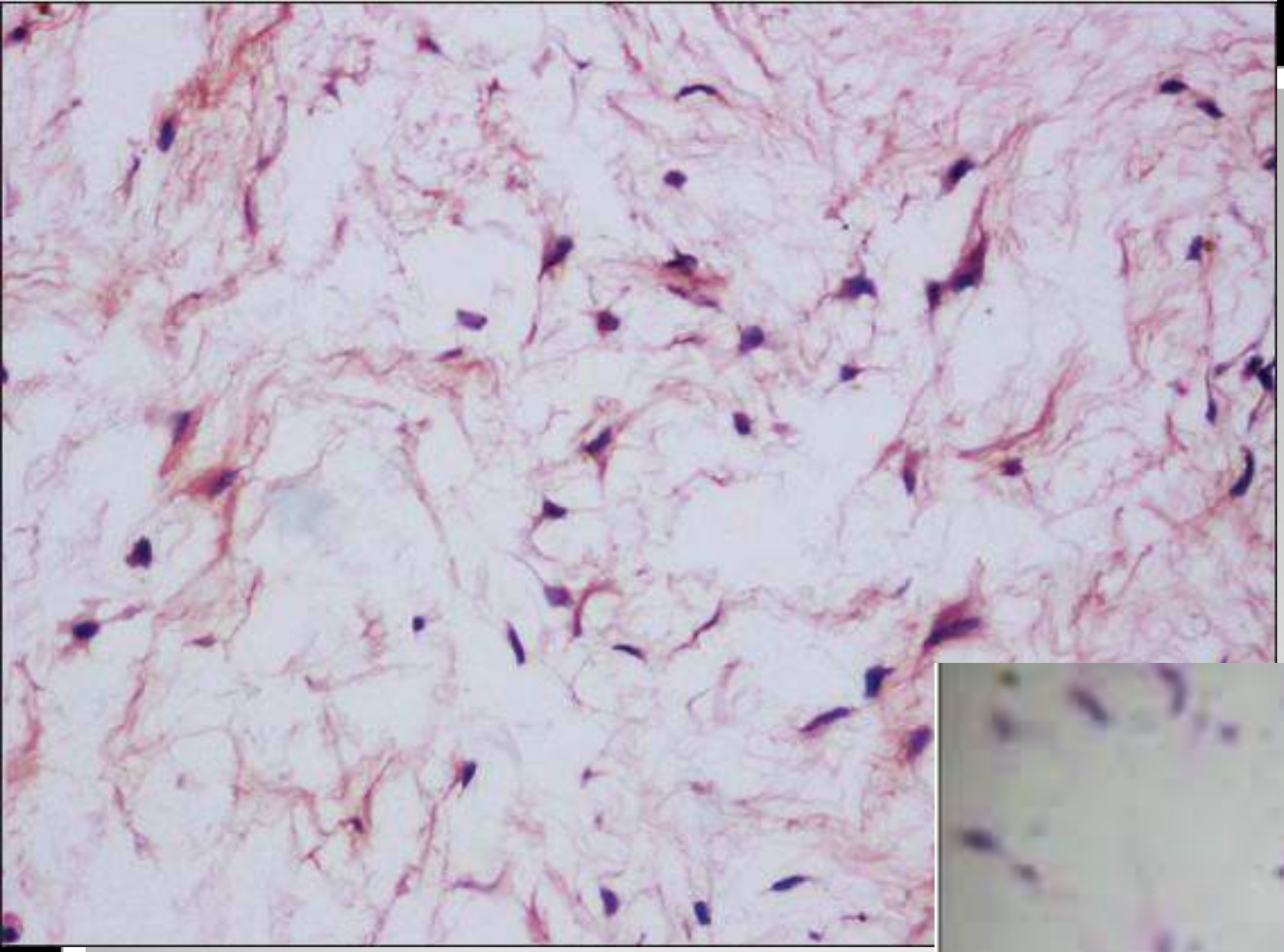


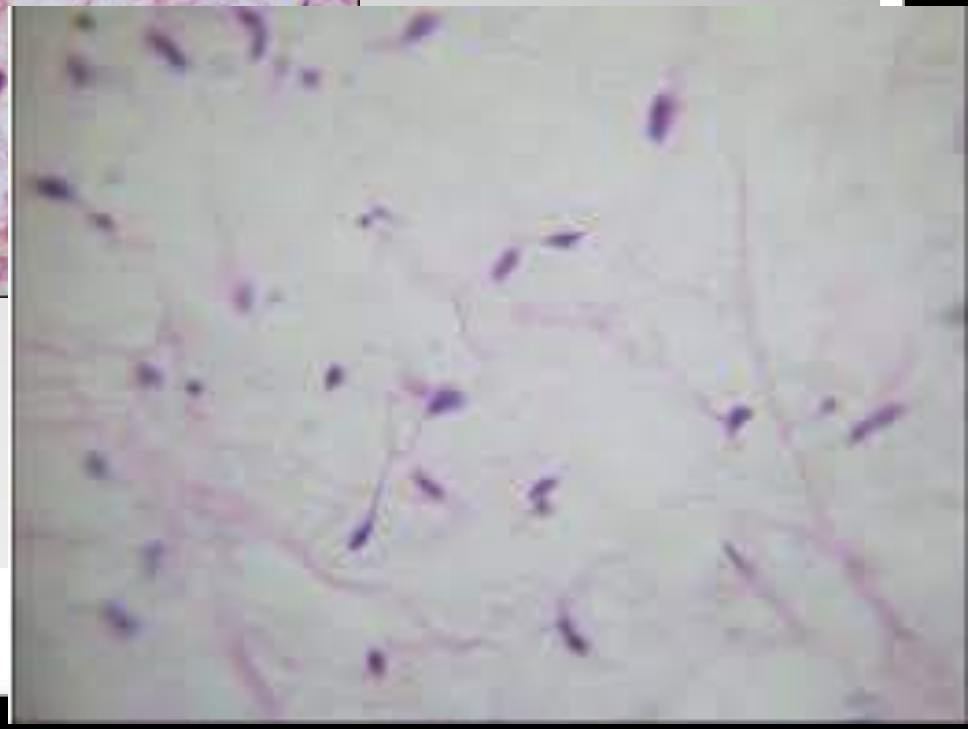
Macroscopy

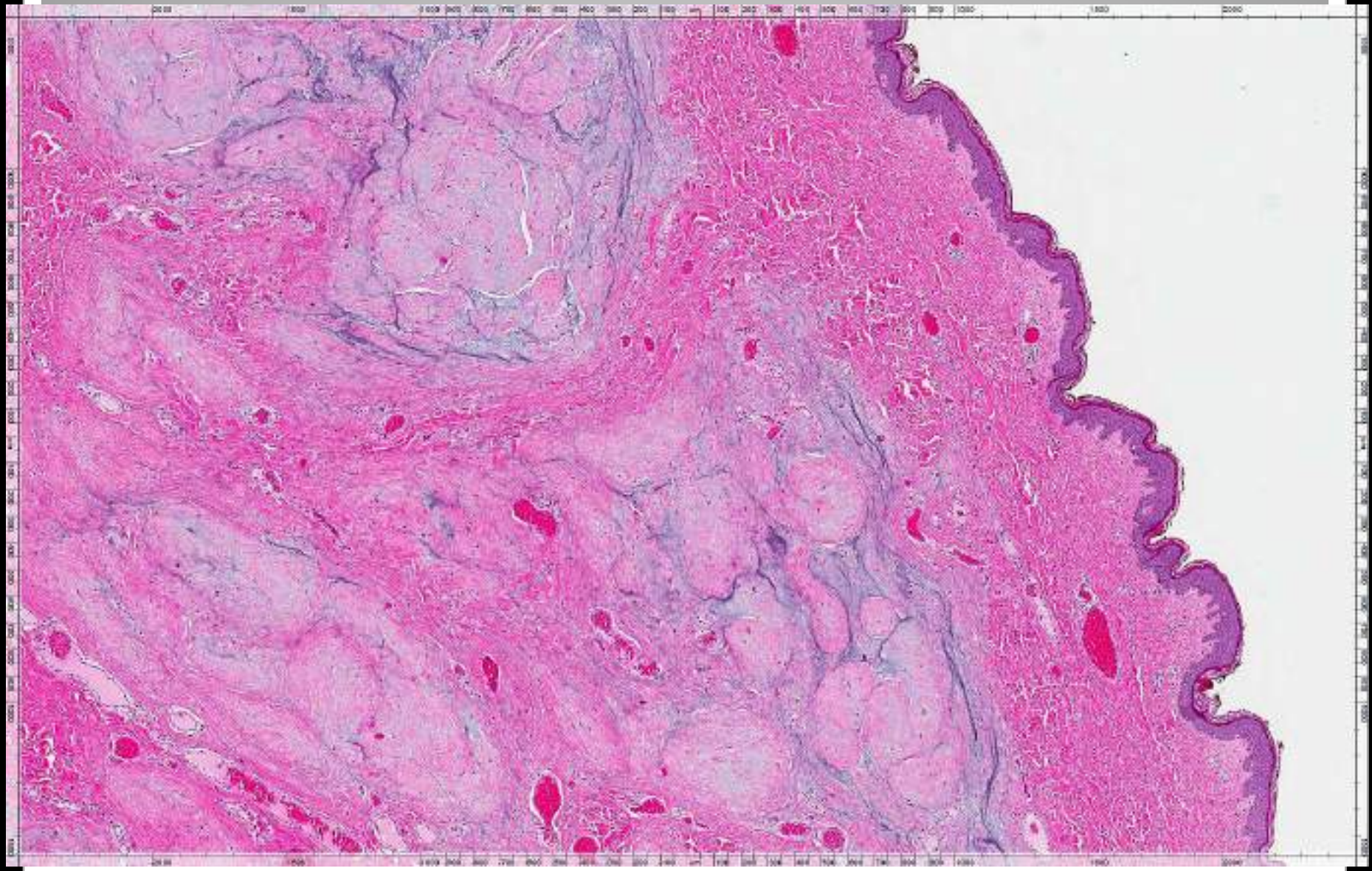
- Characterized by randomly oriented stellate, spindle-shaped and round cells with long, fine, anastomosing pale or slightly eosinophilic cytoplasmic processes extending from the centrally placed nucleus.
- Cells are evenly dispersed in an abundant mucoid or myxoid stroma that contains only a few fine collagen fibres.
- Binucleated cells, mild pleomorphism and mitotic figures may occur.
- Rests of odontogenic epithelium are not obvious in most lesions and are not required for establishing final diagnosis.
- Some lesions may have a tendency to produce collagen fibres and are designated myxofibroma.
- Larger lesions shows areas permeating into marrow spaces

Microscopy









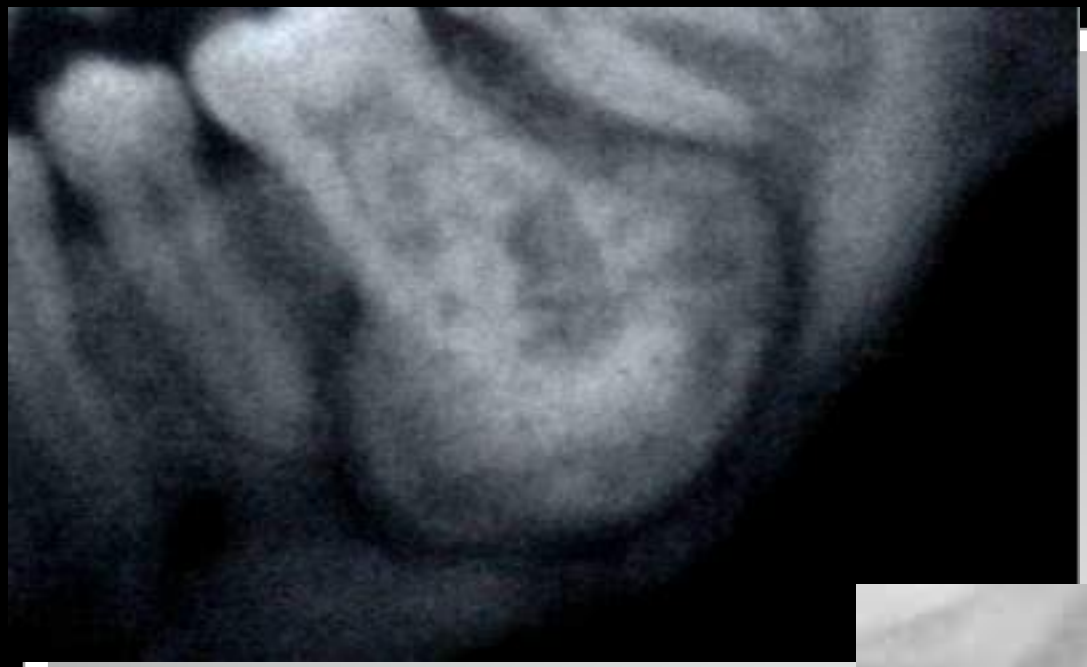
Definition

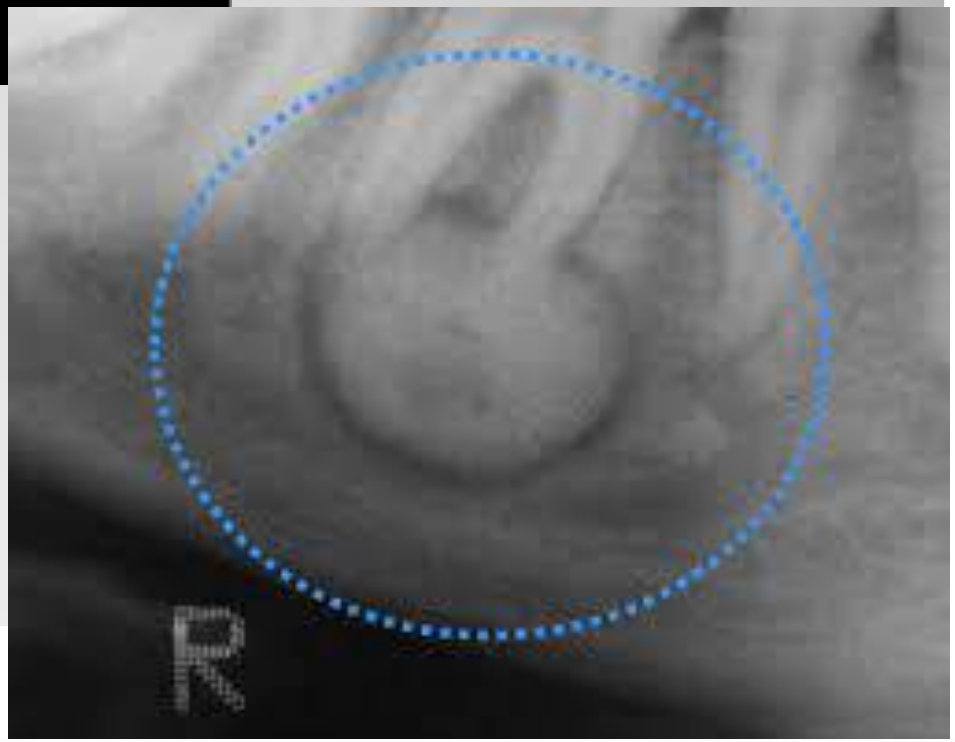
- A cementoblastoma is characterized by the formation of cementum-like tissue in connection with the root of a tooth.
- ICD-O code 9273/0
- Just over a hundred cases have been reported.
- The age range is from 8 up to 44 years, the mean being approximately 20 years.
- There is no distinct gender preference.
- The majority of cementoblastomas are located in the mandible, particularly related to the permanent first molar; association with a primary tooth is exceptional

Cementoblastoma

- The most common finding is a painful swelling at the buccal and lingual/palatal aspect of the alveolar ridges.
- The vitality of the involved tooth remains intact.
- Lower-lip paresthesia or a pathologic fracture of the mandible are rarely reported
- Radiographically, the tumour is well-defined and is mainly of a radiopaque or mixed-density, surrounded by a thin radiolucent zone.
- Root resorption, loss of root outline and obliteration of the periodontal ligament space are common findings.

Clinical presentation and imaging

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Macroscopy

- The tumour consists of a rounded or nodular mass attached to one or more tooth roots and is surrounded by a grey to tan layer of irregular soft tissue.

- A cementoblastoma consists of dense masses of acellular cementum-like material in a fibrous, sometimes rather vascular stroma that may contain multinucleated cells.
- The tumour mass blends with the root of a tooth with simultaneous root resorption.
- In the more mature parts of the tumour, basophilic reversal lines may produce a Paget disease-like pattern.
- At the periphery sheets of unmineralized tissue may be seen, often being arranged in radiating columns.

Microscopy