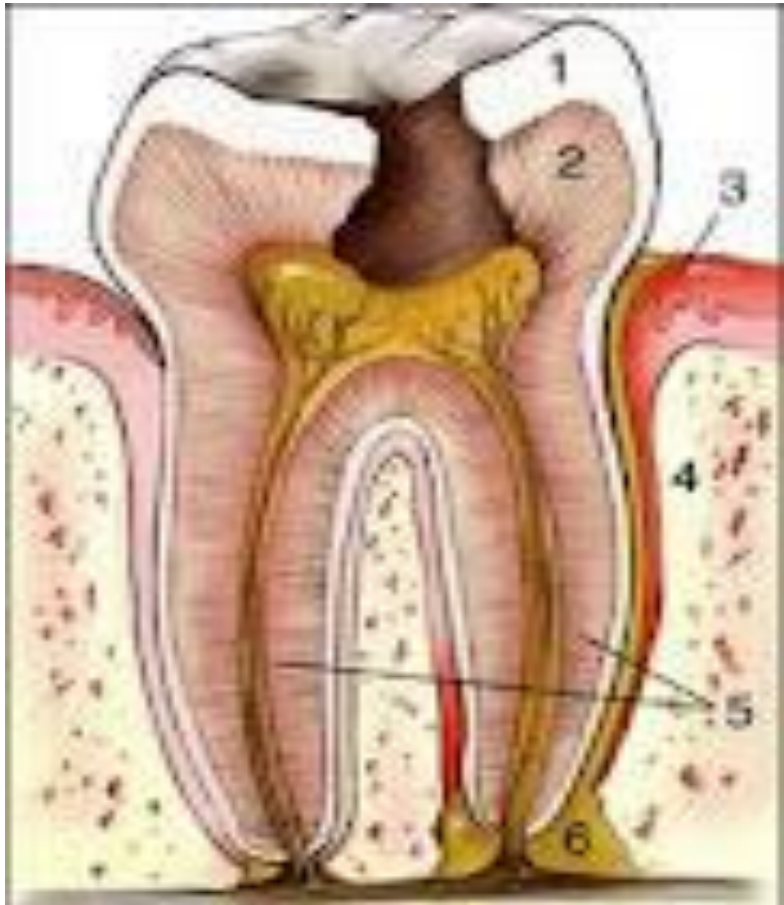
Periapicalperiodontitis

Diseases of periapical tissues
Periapical periodontitis

Definition: it is the extention of inflammation of the pulp beyond the tooth into periapical area of the peridontal ligament.

Differences: Get resorption of adjacent bone & occasionally root. (confined space)

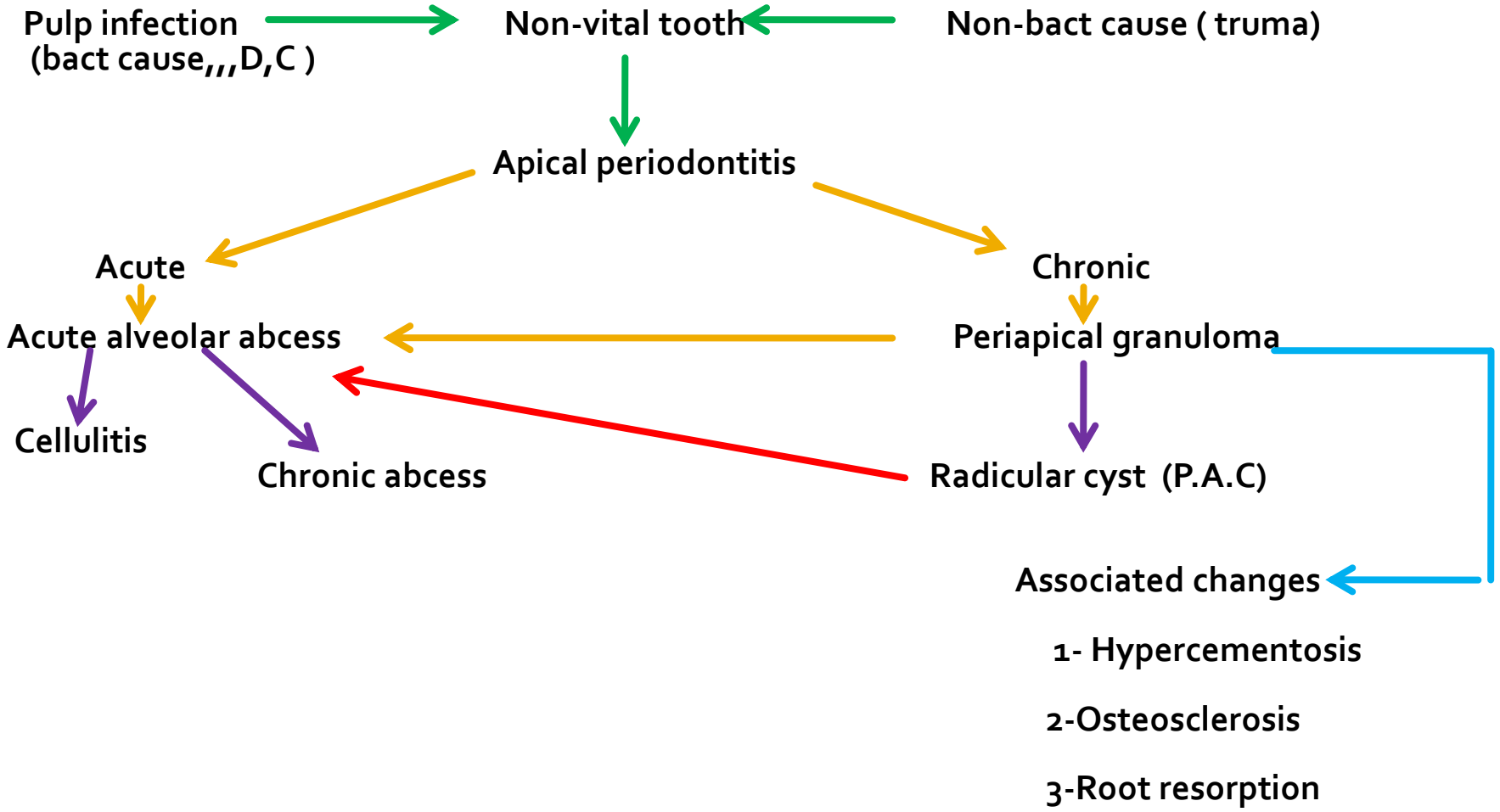


Comparison with pulpitis

1-Healed # No healing (why?)-No collateral blood supply

2- Localized affected tooth # can not be localized (why?)--No properioceptive nerve fiber

Possible lesions occur around a non-vital tooth



These sequences depend on many factors

1. No. & virulence of M.O
2. Type & severity of irritant (chemical or mechanical)
3. Resistance of patient

P.A.P must be distinguished from
chronic marginal periodontitis

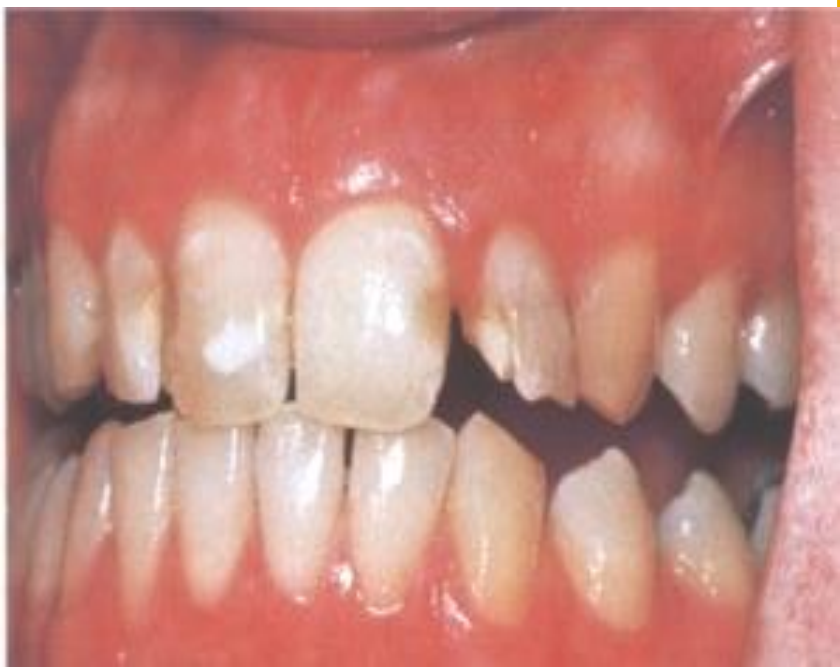
Etiology

- Infection (Bact. ,Bacterial toxin , infl. Product) → P.A area (time)

  → P.A periodontitis

- Traum (blow...?,occlusion...?.filling...?.instrumentation..)

- Chemicals (R.C.filling materials)

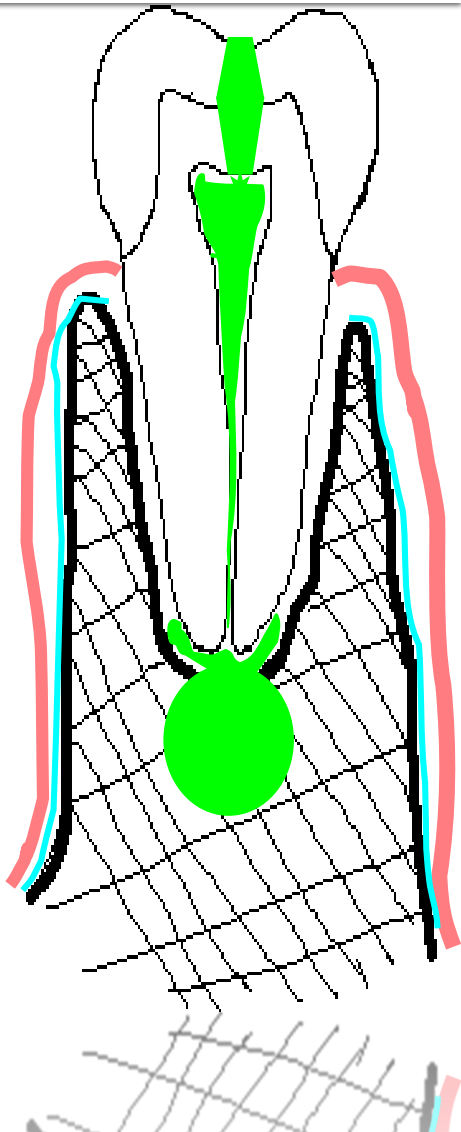


1- Periapical granuloma
(Chronic periapical periodontitis)

-An inflamed granulation tissue,
(resulting from irritation following pulp disease or endodontic treatment) , at the periapical area oftooth.

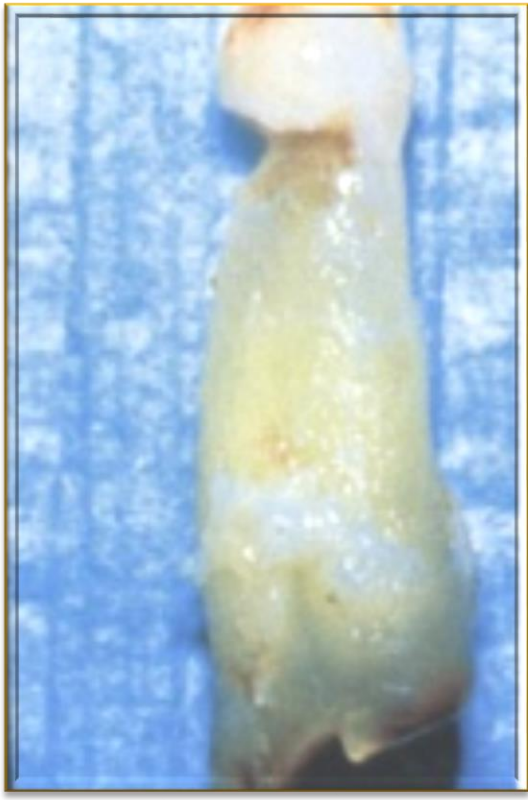
- Resorption of P.A alveolar bone,in response to long standing inflam , is replaced by this granulation tissue.
- It is the commonest sequalae of pulpitis & pulp necrosis.

-S.T. be lateral granuloma (why?)



Clinical features

- Asymptomatic
- Progress slowly
- Mild pain (during eating) & sensitivity can develop (why?)
- Soft tissue over apex may be tender (to percussion). (why?)
- Non vital tooth



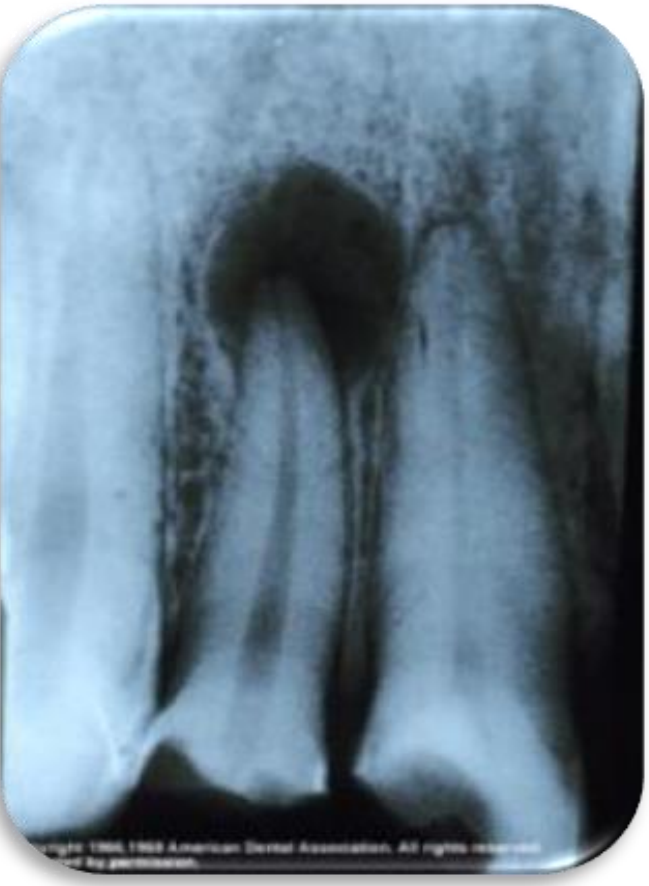
- Any carious tooth with pulp exposure, mainly 1st molars (why?)

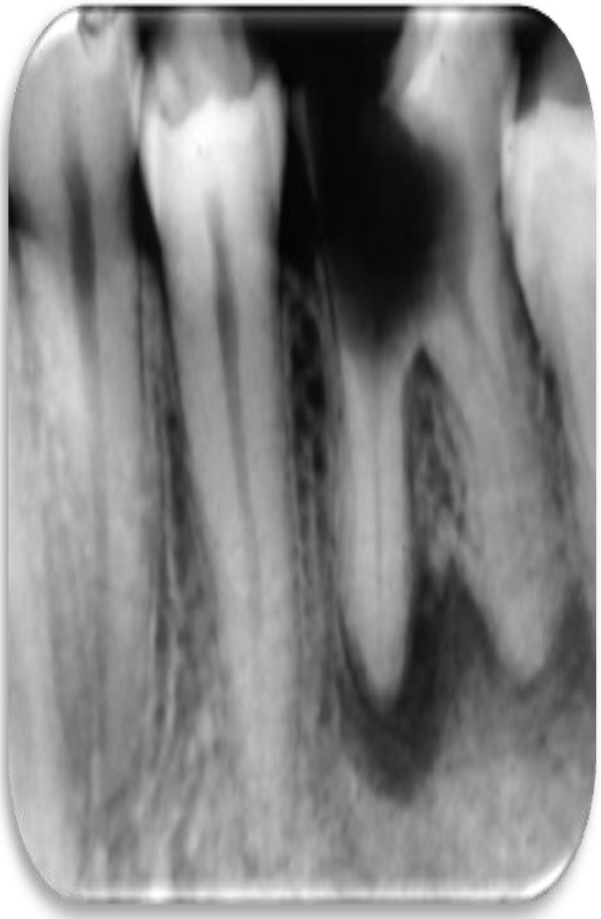
Radiographical feature

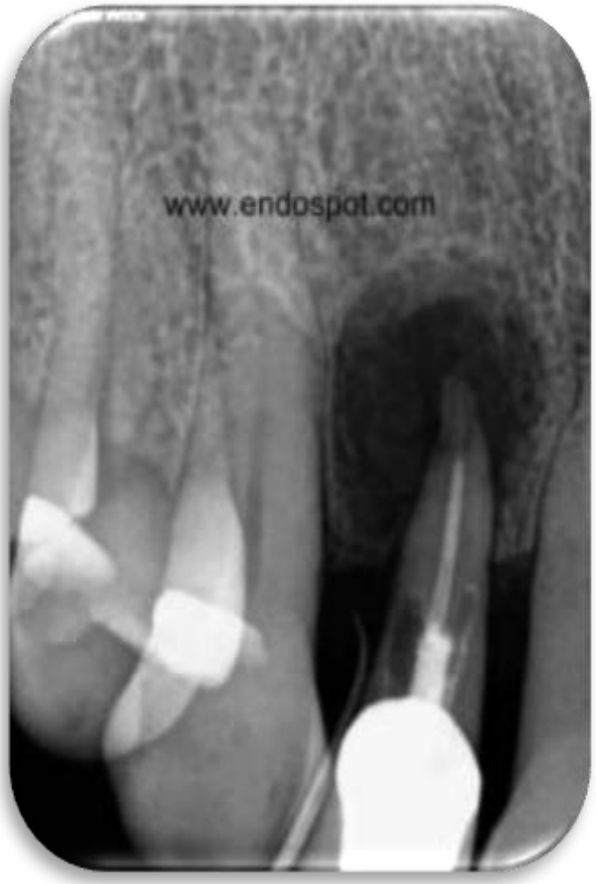
-Earliest change is a thicking of PDL at tooth apex.

-As lesion expand,a definite oval or round clearly demarcated RL at apical area of the involved tooth.

-RL may be laterally displaced . (rare)





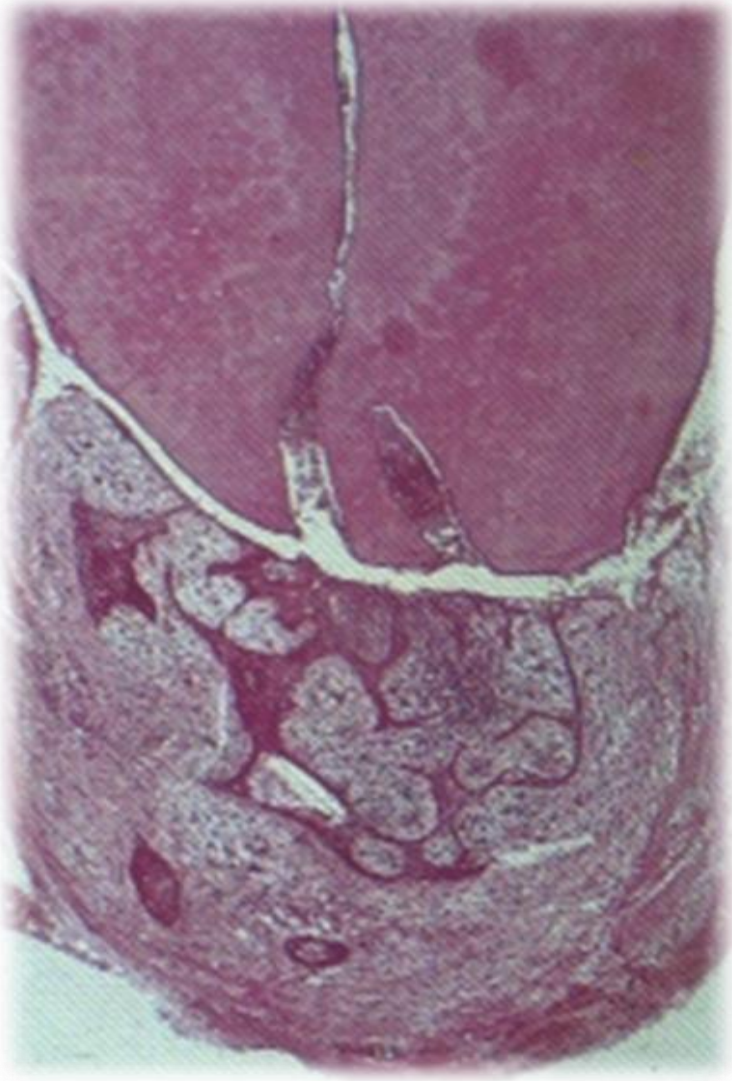


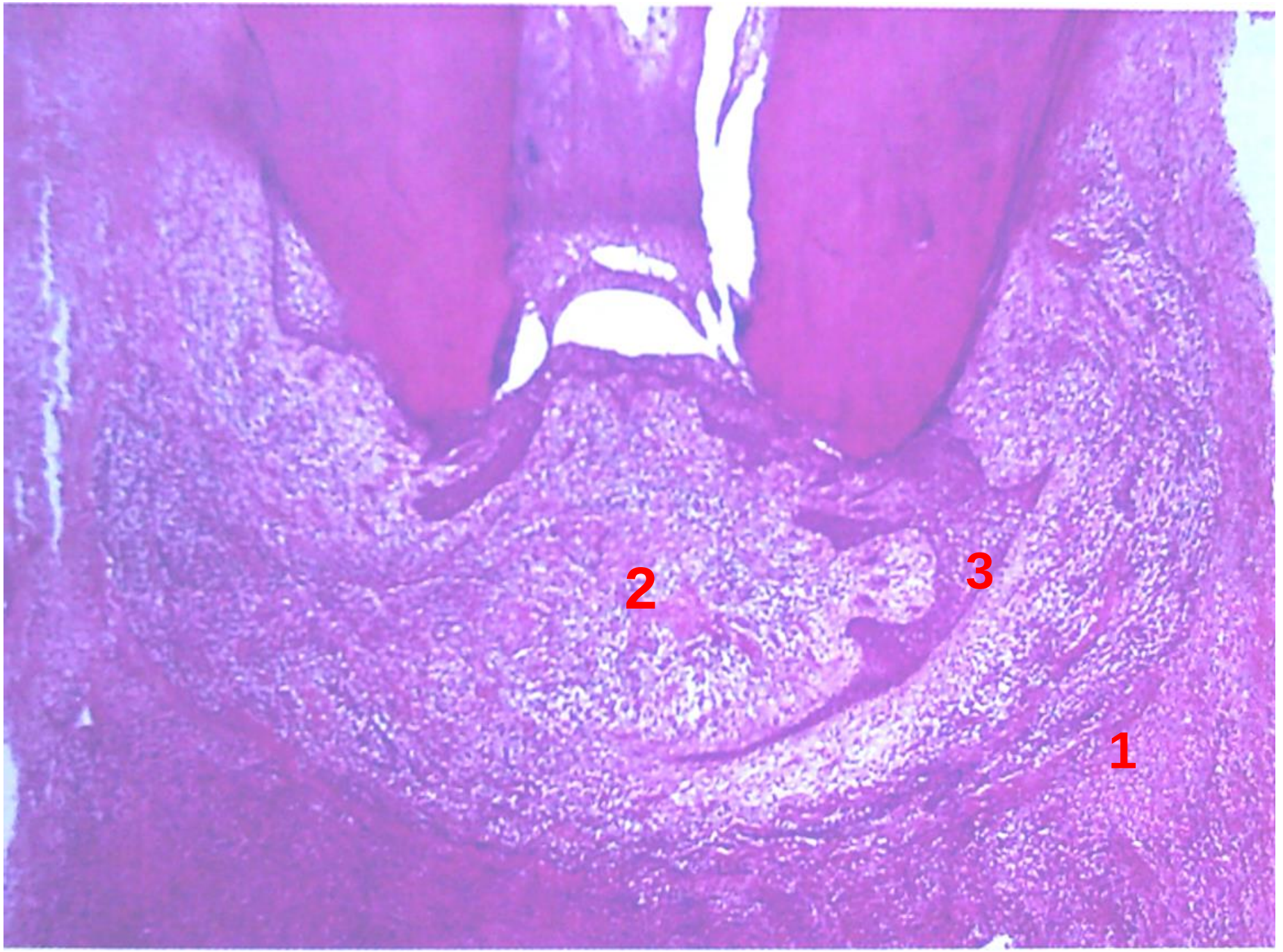
Histopathology

- Dense fibrous tissue capsule.
- Central zone of granulation tissue (large no. of capillaries with proliferating fibroblast & collagen fibers) with macrophage & foamy cells.
- May have some cholesterol crystals surrounded by multinucleated giant cells.
- Irregular islands of epithelium (why?)

Stimulated by growth factor released from inflamm cells

- PMNL ? And plasma cells.




2
3
1

Origin of epithelium in periapical granuloma

1-Epithelial cell rest of Malassez ..? Most common
2-Respiratory epith of maxillary sinus.? In upper teeth
3-Oral epitelium growing through a .? In fistula
4-Oral epithelium from..? In peridontal pocket

- **Treatment:**

  Root canal therapy, apicoectomy, or extraction

- **Prognosis:**

  good,

  could develop into a periapical cyst if untreated

Sequelae of PAG

1-Remain localized within bone (body resistance =virulance of bact)

↓ resistance & / or ↑ virulance of bact → bone resorption

2-Suppuration&abcess formation → P.A.A → Pus reach buccal gingiva over affected tooth → nodule of granulation T (Gum boil) formed marked opening of sinus tract.

3-Radicular cyst.

4- Osteosclerosis (low-grade irritation)--bone apposition rather resorption

5-Hypercementosis

2- Periapical cyst (Radicular cyst)

- Commonest cystic lesioin in the jaw. 75% of all odontogenic cyst.
- Derived from untreated , long standing P.A.G.
- True cyst.

Pathological cavity lined by epithelium & the lumen contain fluid &/or semifluid materials.

PATHOGENESIS

- **PAC arise from proliferation of epith cell rest of Malassez present in PDL or within the preceeding PAG.**

Not all PAG form a cyst

-Two mechanisms for cyst formation:

1-Contineous proliferation → mass of epith cells → mass ↑ in size →
central part loss nutrition → degeneration & liquifactive necrosis →
cavitation → cyst formation with contineous expantion (drain fluid from surrounding T).

2-Degeneration & liquifactive necrosis of the granulation T (bact & dead pulp release toxic product) → epith proliferate to surround necrotic area →
cyst formation.

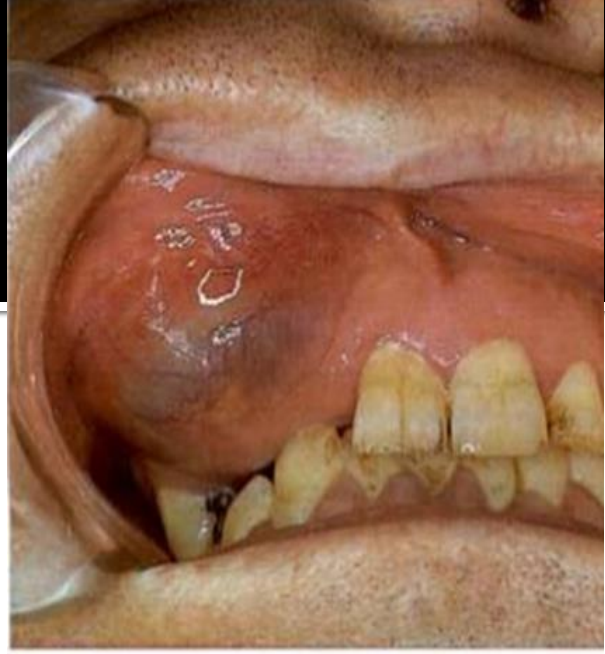
The main factors responsible for expansion of P.A.C:

- 1-Proliferation of epithe lining .
- 2-Hydrostatic pr.of cyst fluid. (Ballon-like fashion)
- 3- Resorption of surrounding bone

(cyst tissue release bone –resorbing factor---
prostaglandin E2 &E3 &collagenase , from fibroblast in cyst capsule.
This increased by action of cytokines **IL-1 &IL- 6**
which synthetized by epith. cyst lining)

Rate of expansion 5 mm / year

Clinical features

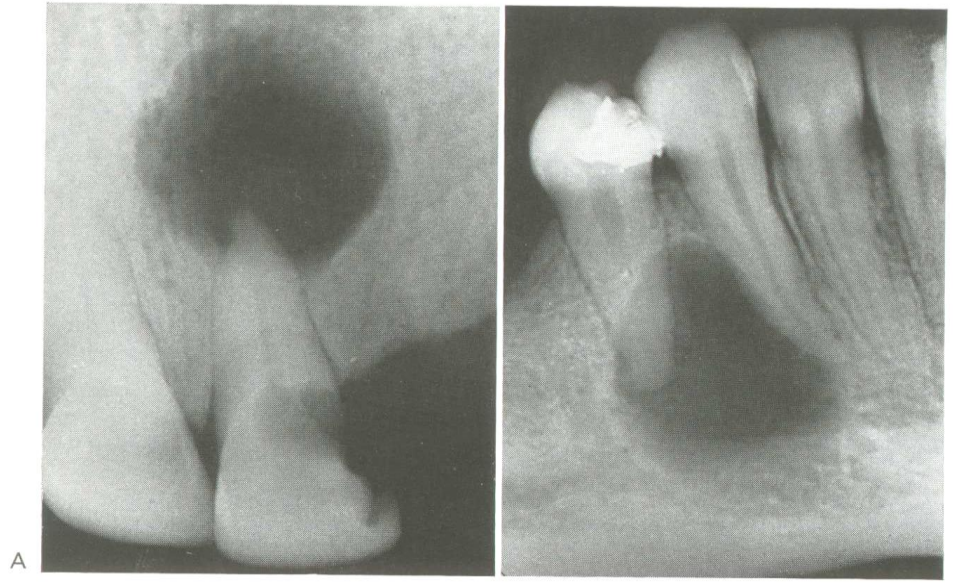


-Asymptomatic -----if exacerbation → abcess formation--- (pain)

-Mobility of adjacent teeth---------if enlarged

Radiographical

-Well-circumscribed RL at root apex,surrounded by thin rim of RO margin



A

Fig. 126. Radicular cysts. A: From maxillary left central incisor. B: From mandibular first premolar

Histopathology

- -Cavity liend by non keratinized

str.Sq.epith.**Epith.thickness correlated with degree of inflam**

- Presence of Rushton bodies

  -eosinophilic & brittle

(rest of RBC or epith secretory products)

- -Wall composed of:

  --C.T rich in collagen fiber.

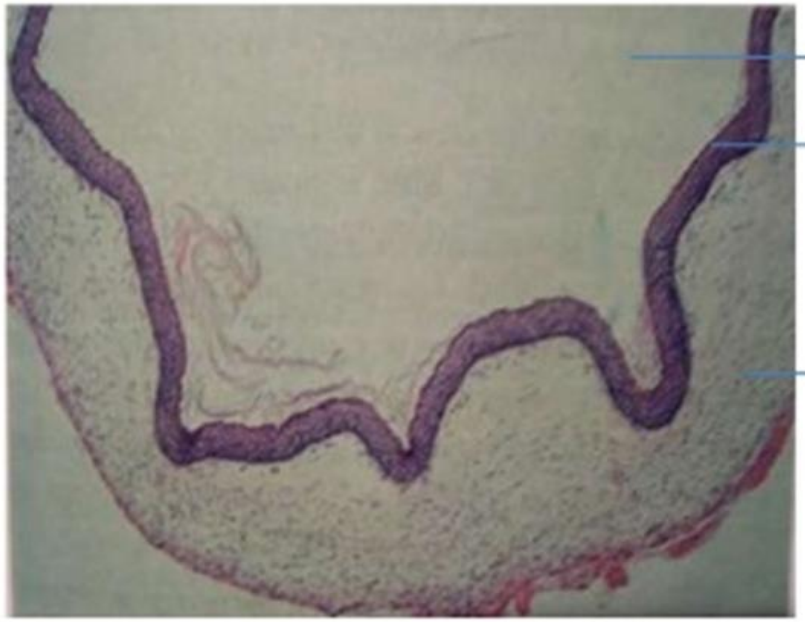
  -- Inflam cells infiltrate (plasma cell, lymphocytes,macrophages)

  -- Foam cells. (Lipid-laden macrophages).

  -- Cholestrol crystals surrounded by giant cell.

- -Fibrous T capsule surrounding the wall.
- -Lumen contains thick protinaceous fluid & cellular debries.

- **Treatment:-** Exo ,, RCT



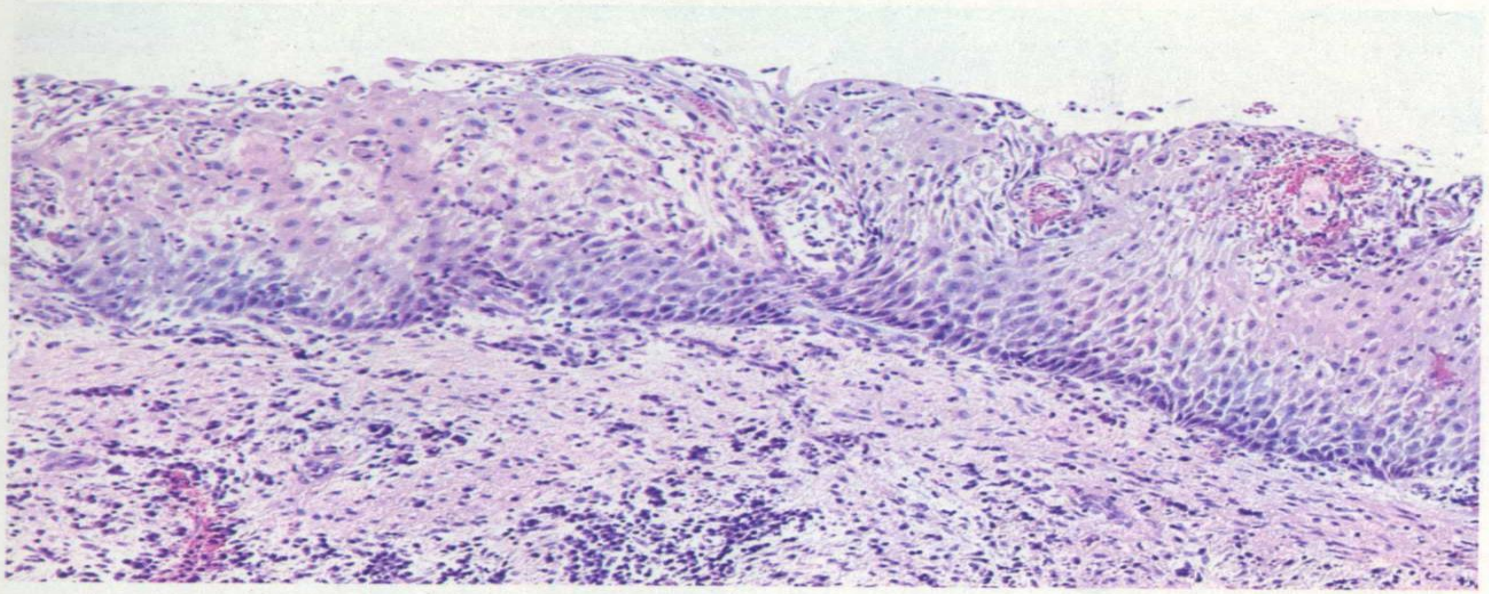


Fig. 127. Radicular cyst x 90

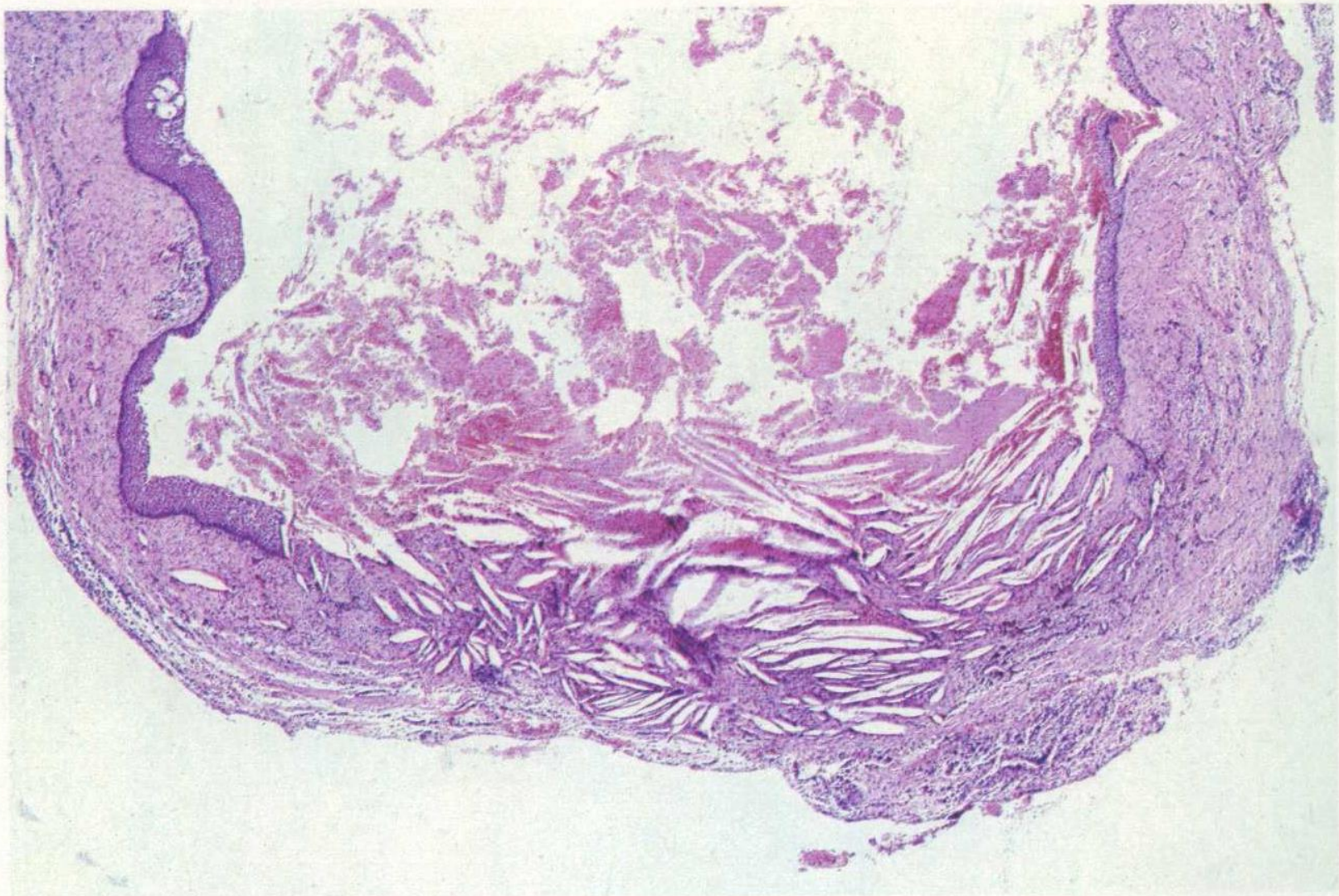
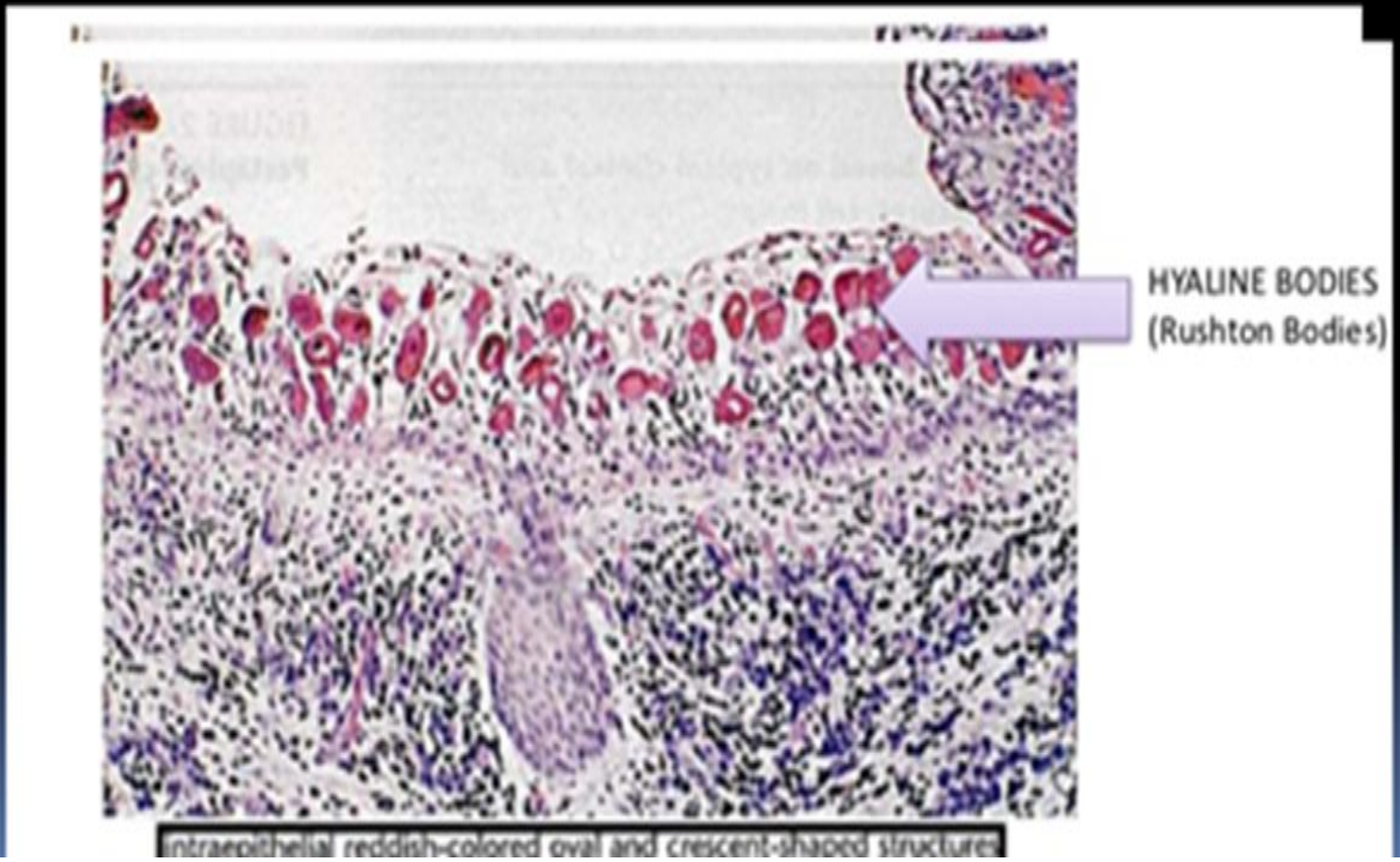


Fig. 133. Radicular cyst. Deposit of cholesterol crystals x 21

- Hyaline bodies may be seen in the epithelium and mucous cells are often present as a result of metaplasia.



Residual cyst

-Radicular cyst,remain in the jaw & faild to resolve after exo of the involved tooth.

-**20%** of radicular cyst is a residual in type.

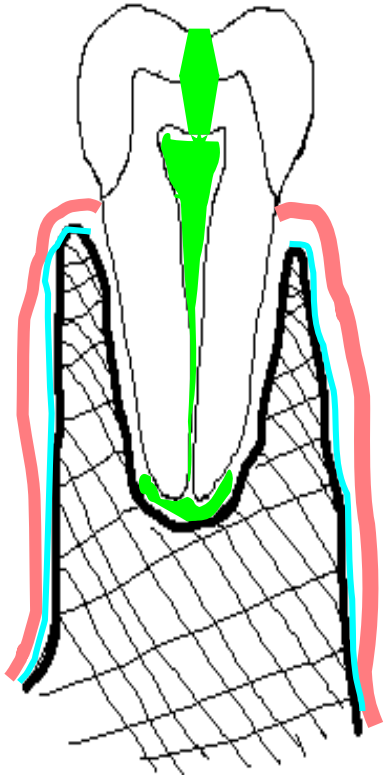
Lateral radicular cyst

- Uncommon.
- Extention of inflam from pulp to PDL,along lateral root canals.

3- Periapical abscess

- A progression of an acute pulpitis in which exudate extends into the adjacent periapical soft and hard tissues.

(Suppurative process at apex of the tooth)



Clinical Features:

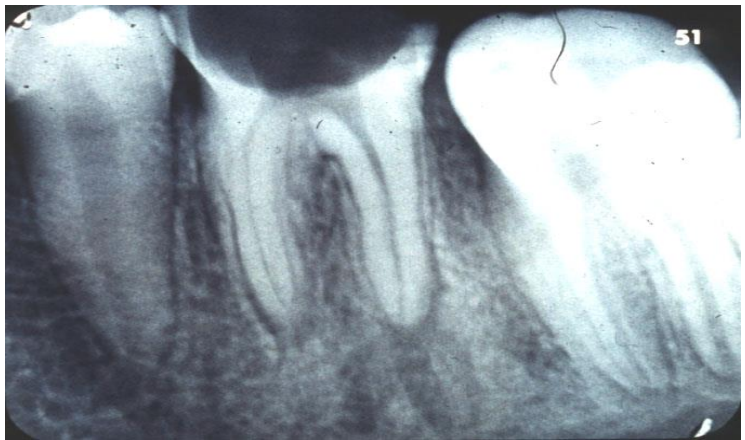
- Painful swelling (why?) of varying size and position
- Extrusion of the tooth from its socket
- Tenderness to percussion
- Fever and malaise with regional lymphadenopathy
- Erythema and possibly draining sinus, intraoral or extra-oral
- Unresponsive to thermal and electrical stimuli
- Affected tooth can be localized

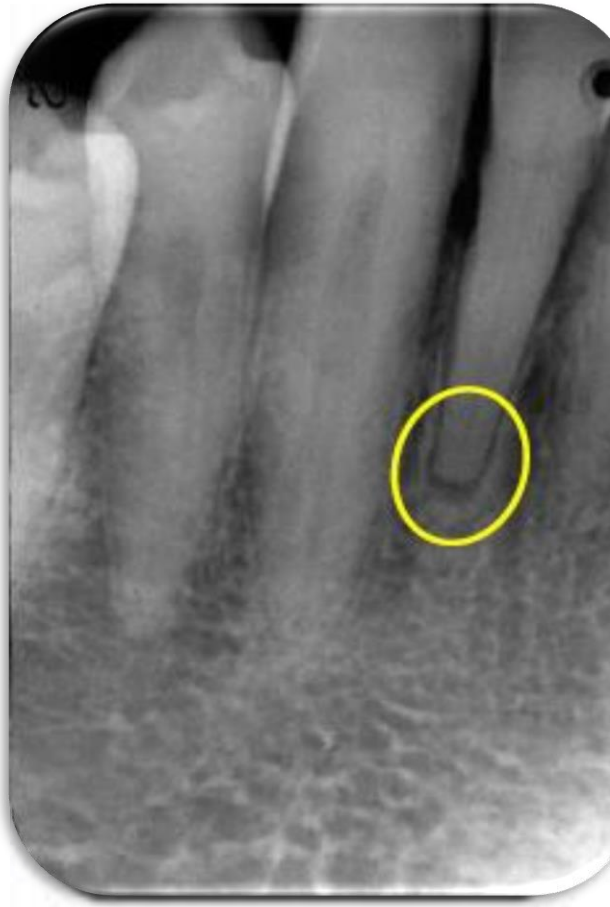
Radiographically

-Acute abcess----no radiographical indication or slight widening of PDL at apex.

-Chronic abcess----RL, no distinct demarcated line.

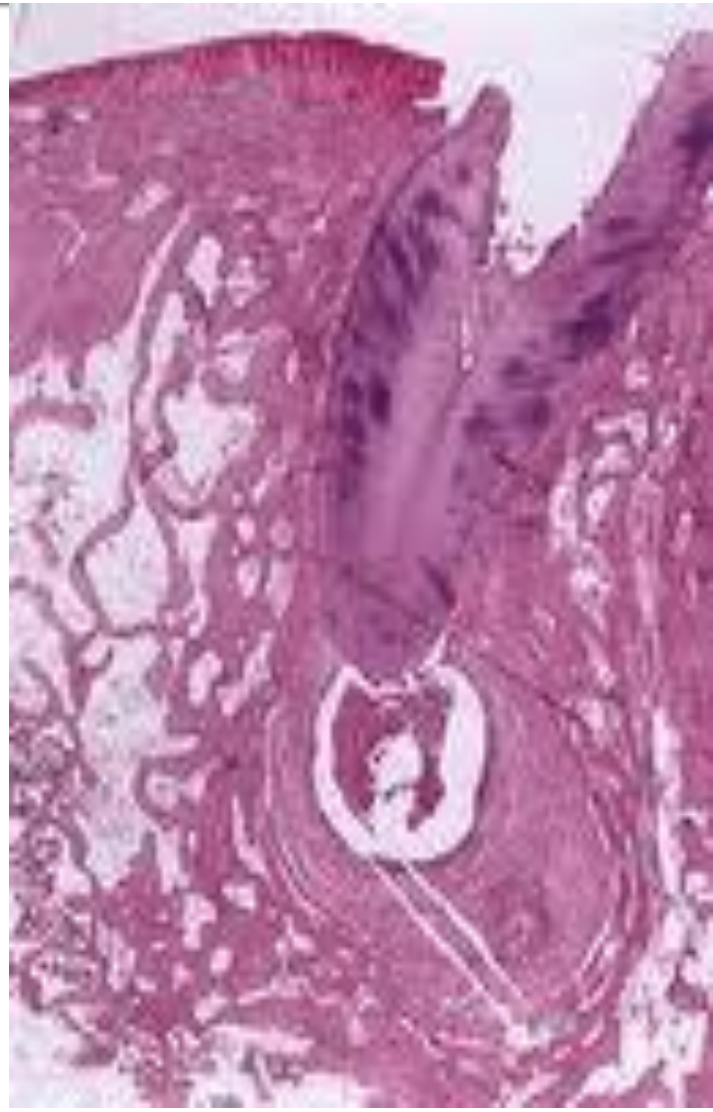
(Abcess rapidly lytic process)

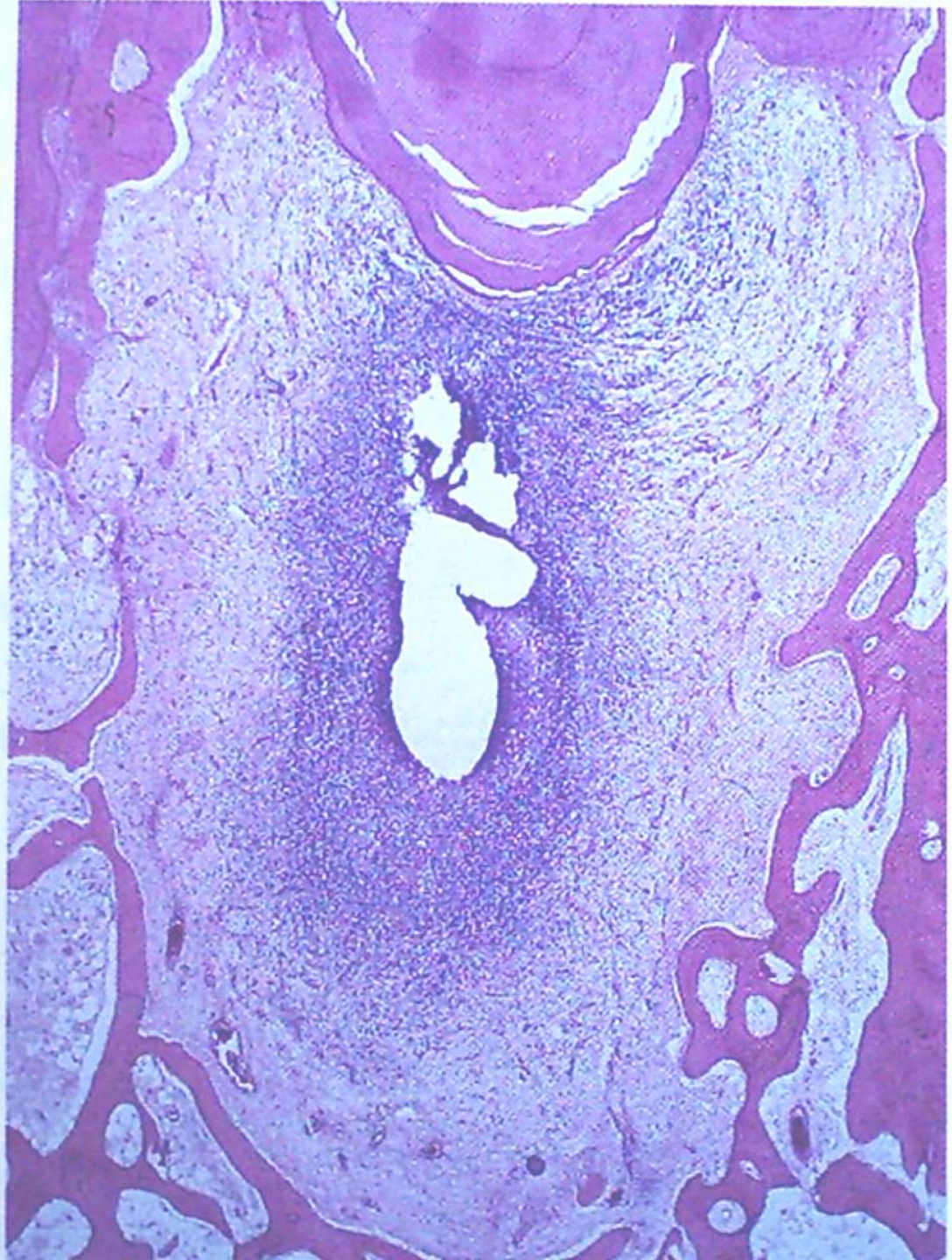




Histopathology

- 1-Outer FT capsule
- 2-Wide zone of granulation T
- 3-Central area of pus





Treatment

- 1-Drainage (emergency)
-
- 2-Analgesic & antibiotics
- 3-R.C.T
- 4-Exo
- Prognosis ---------Good

Acute Periapical Abscess & Spread of Inflammation: Routes of Spread

- If cause is not removed by extraction, RCT or antibiotics:

1. Suppuration continues and abscess enlarges.

2. Balance between irritant and host defenses may cause abscess to become chronic and localized.

3. Increase in hydrostatic pressure causes pus to track in one of a number of directions:

a) Through an open root canal.
b) Through gingival sulcus.
c) Through cancellous bone then perforating the cortex, then buccally or lingually, intraorally or extraorally depending on anatomy and muscle attachments relative to root apices.

Acute Periapical Abscess & Spread of Inflammation: Routes of Spread

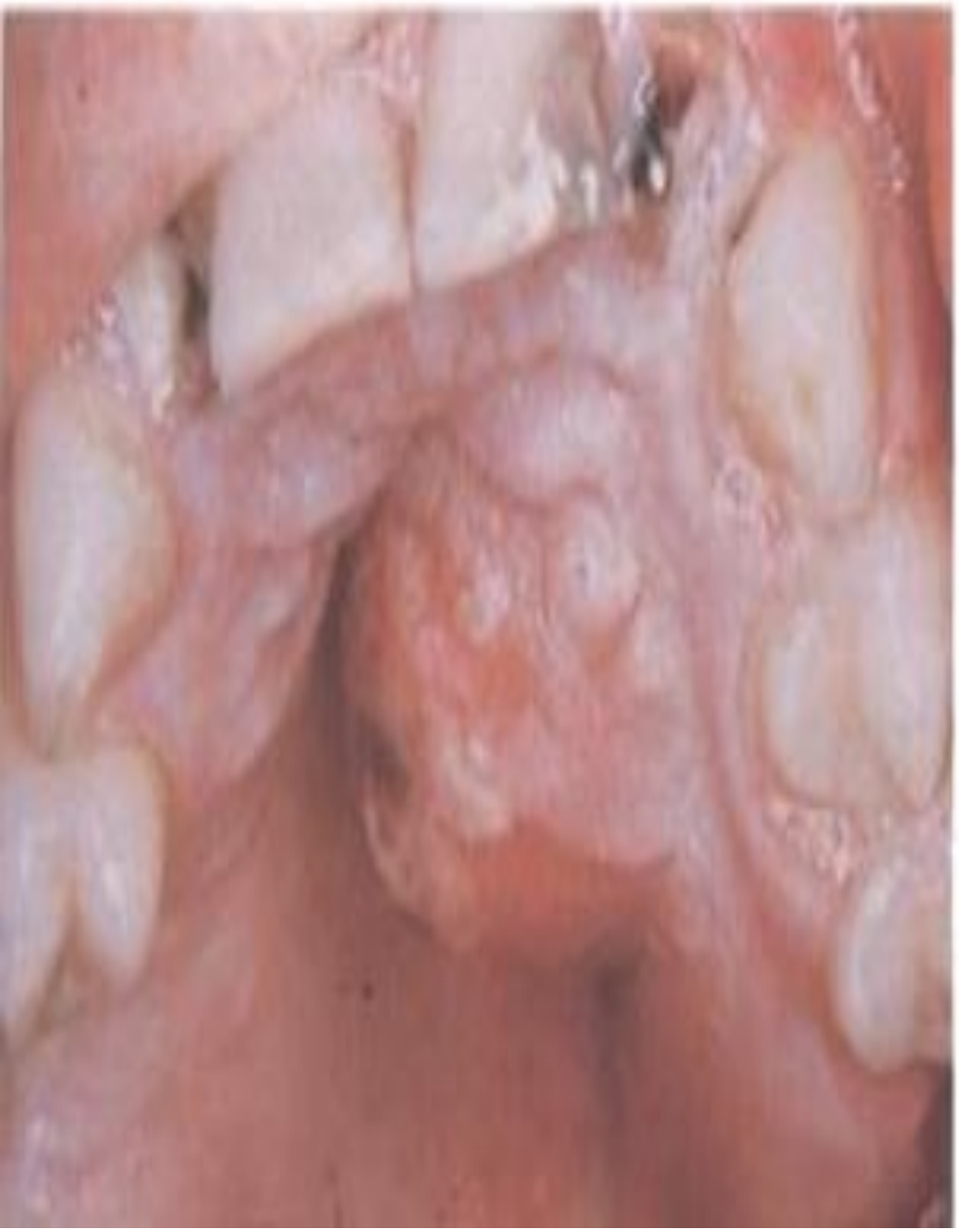
After the pus has perforated the cortical plate its subsequent routes of spread are dictated largely by anatomical factors.

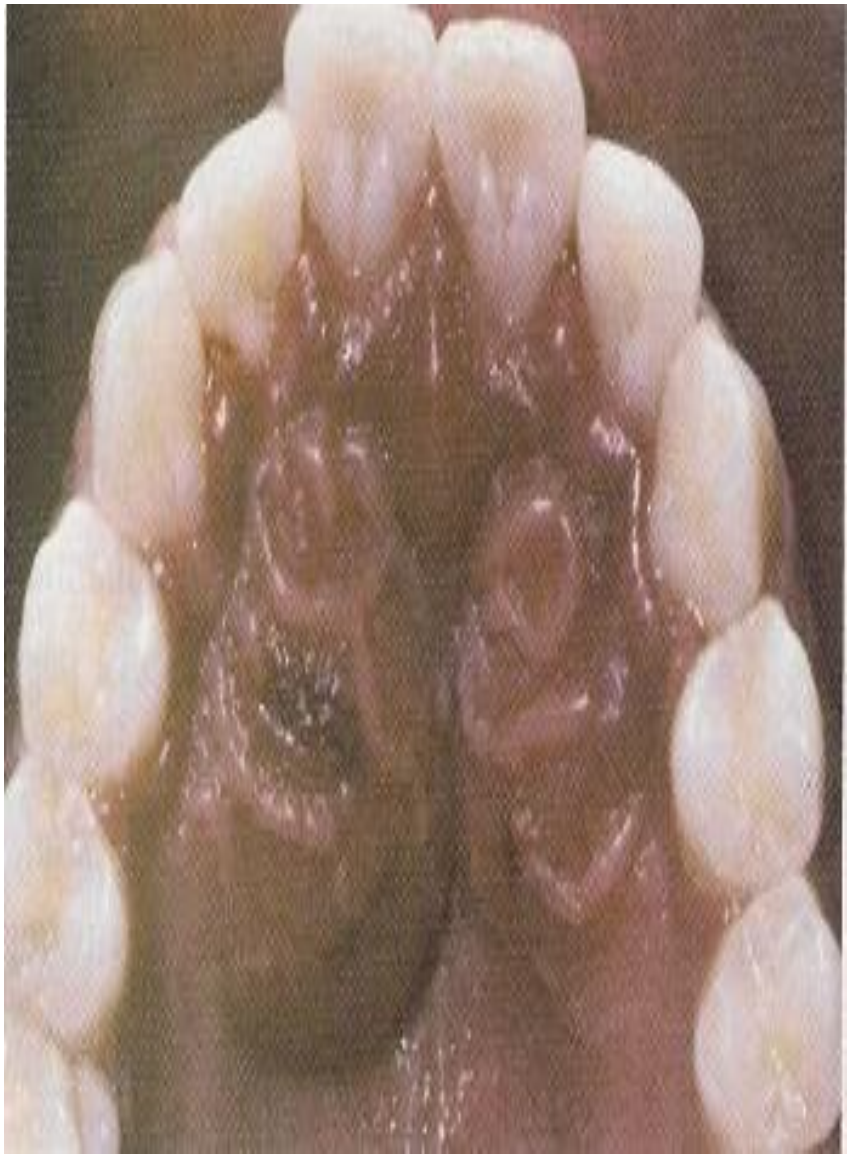
- **Possible outcomes:**

1- **Pus may discharge directly into oral cavity through a sinus track.**

- **This may occur with or without pain or swelling.**
- **A nodule of granulation tissue often forms & marks the opening of sinus track- "*gumboil*" or "*parulis*".**

2- **Pus tracking palatally may spread under the dense palatal mucoperiosteum posteriorly to the junction of hard and soft palate, presenting as a palatal abscess.**



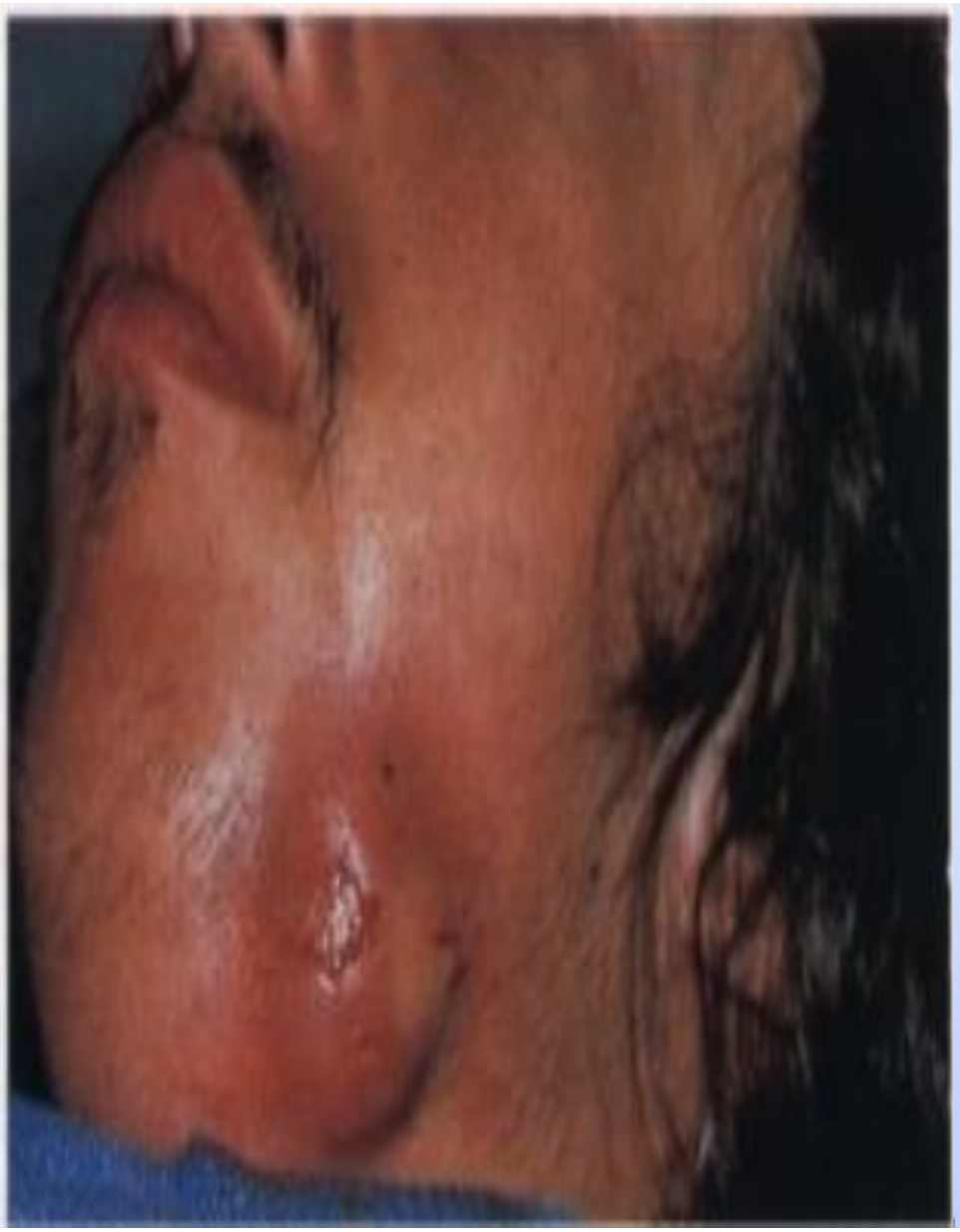


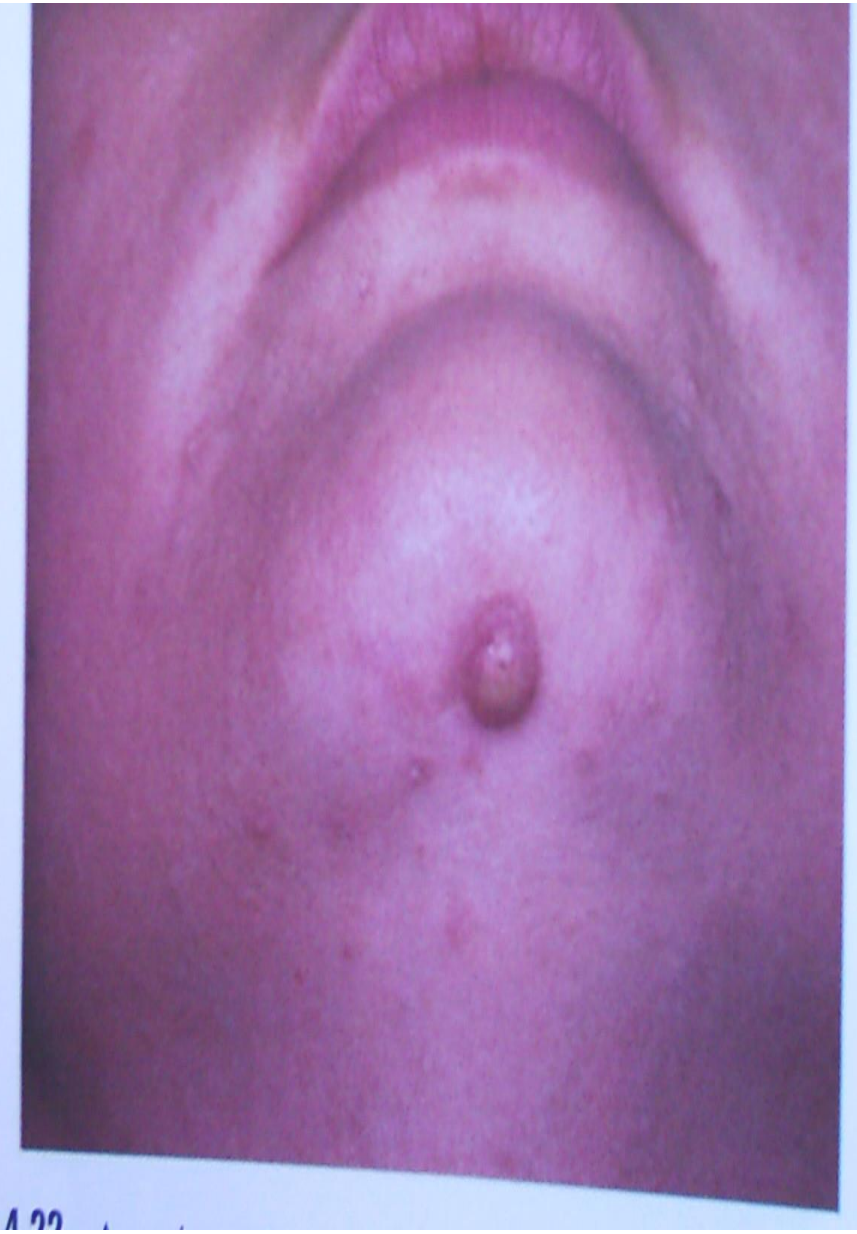
Acute Periapical Abscess & Spread of Inflammation: Routes of Spread

3- Abscesses in molar regions may penetrate the buccal cortical plate above (maxilla) or below (mandible) attachments of buccinator muscle.

- In such cases, the acute inflammatory edema and suppuration may spread into soft tissues of face or neck, → presenting as *cellulitis*

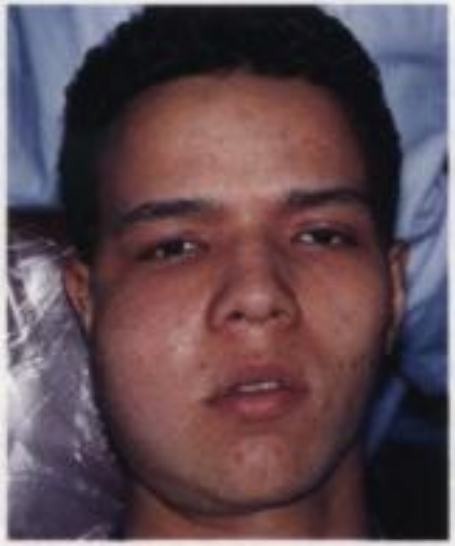
-- Or it may stay as a localized soft tissue abscess.
Such an abscess may discharge through a sinus on skin surface
(extra oral abcess) → Scarring of skin (if chronic)





Cellulitis

- Rapidly spreading inflammation of the soft tissues particularly associated with streptococcal infections,involving fascial planes that separete muscle T.
- In contrast to an abscess, it is not well-localized.
- Release of streptokinase and hyaluronidase---(rapid spread)
- Diffuse, tense & painful soft T swelling.
- Malaise & elevated temperature.
- Extension of cellulitis associated with maxillary teeth towards the eye is a potentially serious complication.
- Involvement of veins at inner canthus of eye may result in cavernous sinus thrombosis.



gure 34.1. **Buccal space infection:** a rapidly spreading and inful infection of the buccal space from an infected first andibular molar.

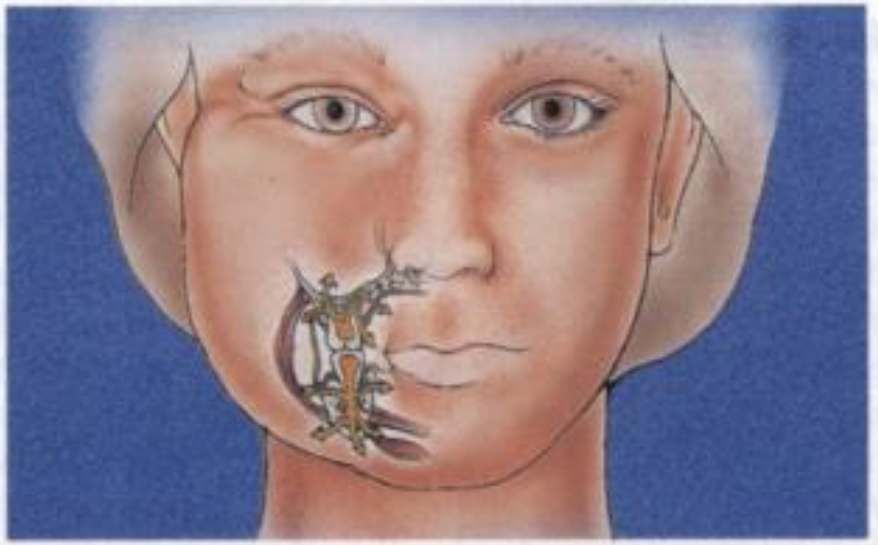
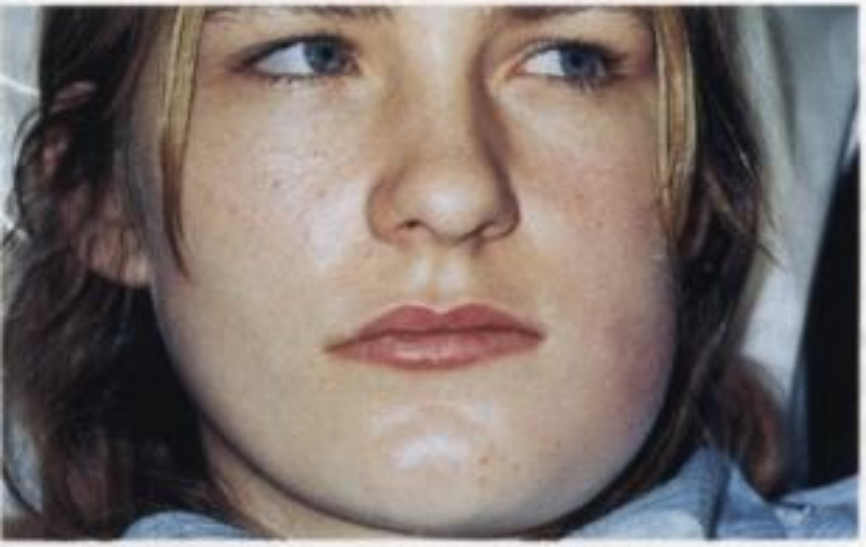


Figure 34.2. Buccal space infection: illustration of spread into the buccal space from an infected mandibular molar.



gure 34.3. **Masseteric space infection:** slowly spreading infec- on below the masseter muscle adjacent to the ramus.

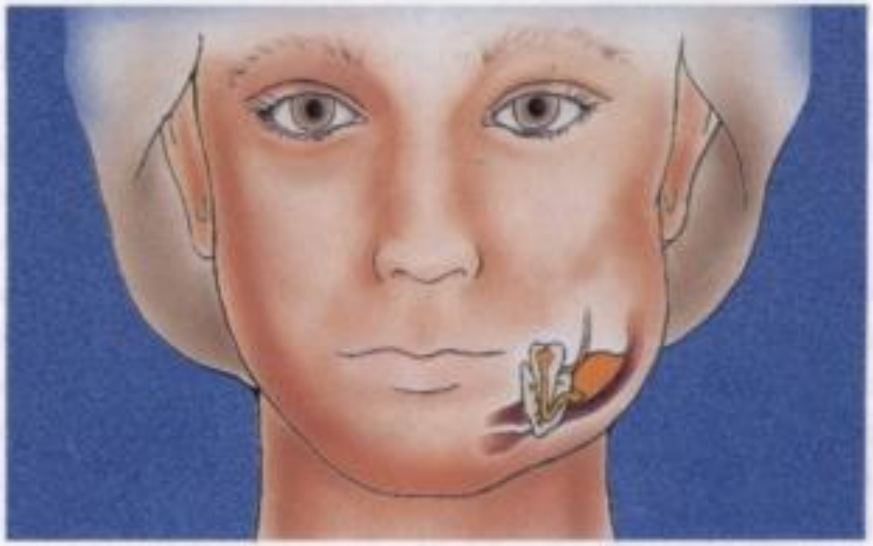


Figure 34.4. Masseteric space infection: illustration of spread into the space between the masseter muscle and mandibular ramus.

Cavernous sinus thrombosis

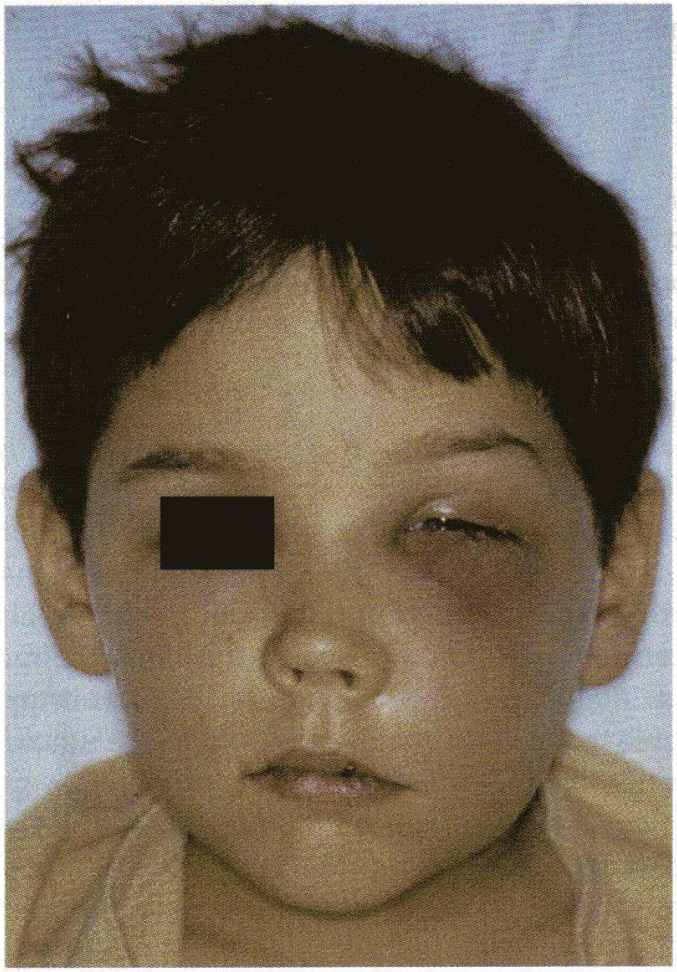
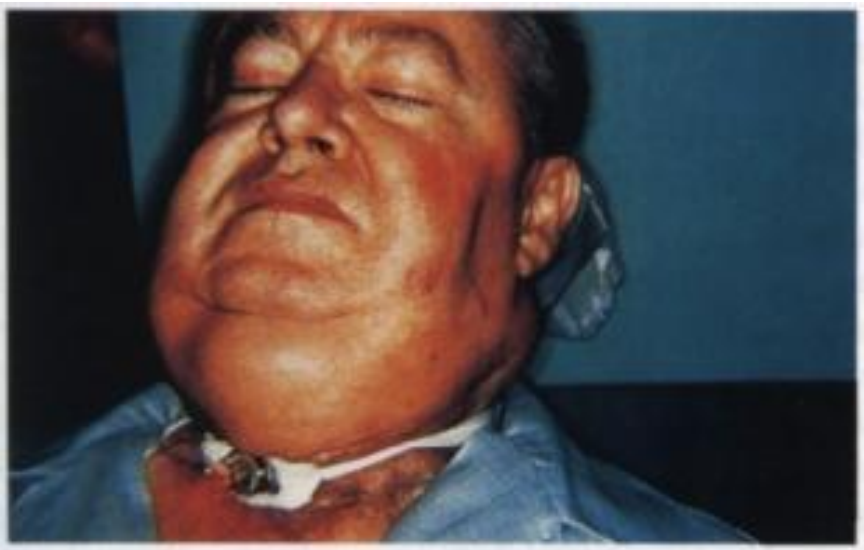


Figure 3-46 • Cellulitis involving canine space. Erythematous and edematous enlargement of the left side of the face with involvement of the eyelids and conjunctiva. Patients with odontogenic infections involving the canine space are at risk for cavernous sinus thrombosis. (Courtesy of Dr. Richard Ziegler.)

Ludwig's Angina

- Severe cellulitis involving the submandibular, sublingual, and submental spaces.
- A result of initial involvement of the submandibular space.
- Rare since advent of antibiotics.
- Board-like swelling of floor of mouth, elevated tongue, difficult eating , swallowing, & breathing.
- If it tracks backwards to involve pharynx and larynx, with edema of the glottis, suffocation may occur.

Ludwig's angina



igure 34.7. **Ludwig's angina:** massive swelling of the subman-
ibular, submental, and sublingual spaces requiring tracheos-
omy. (Courtesy Dr Geza Terezhalmy)

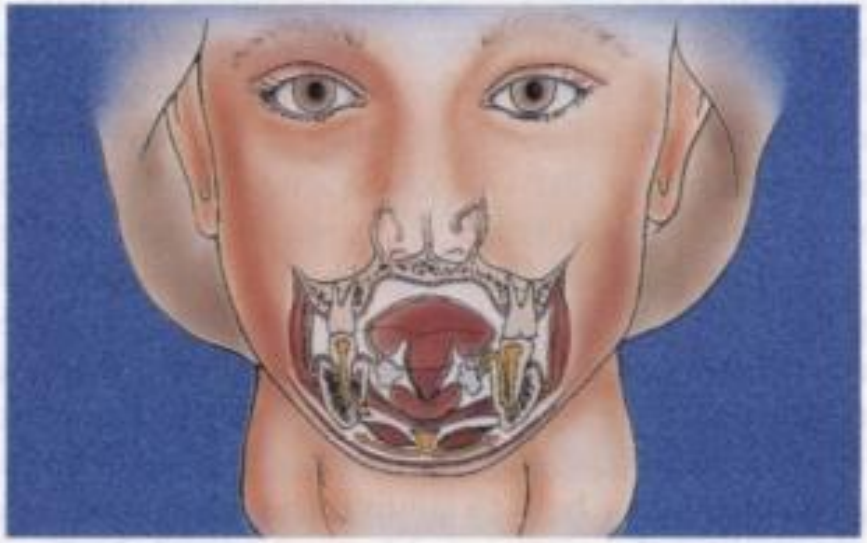


Figure 34.8. Ludwig's angina: illustration of spread of infection from an infected molar.