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Oral Pathology module

Infections of Teeth and Jaws

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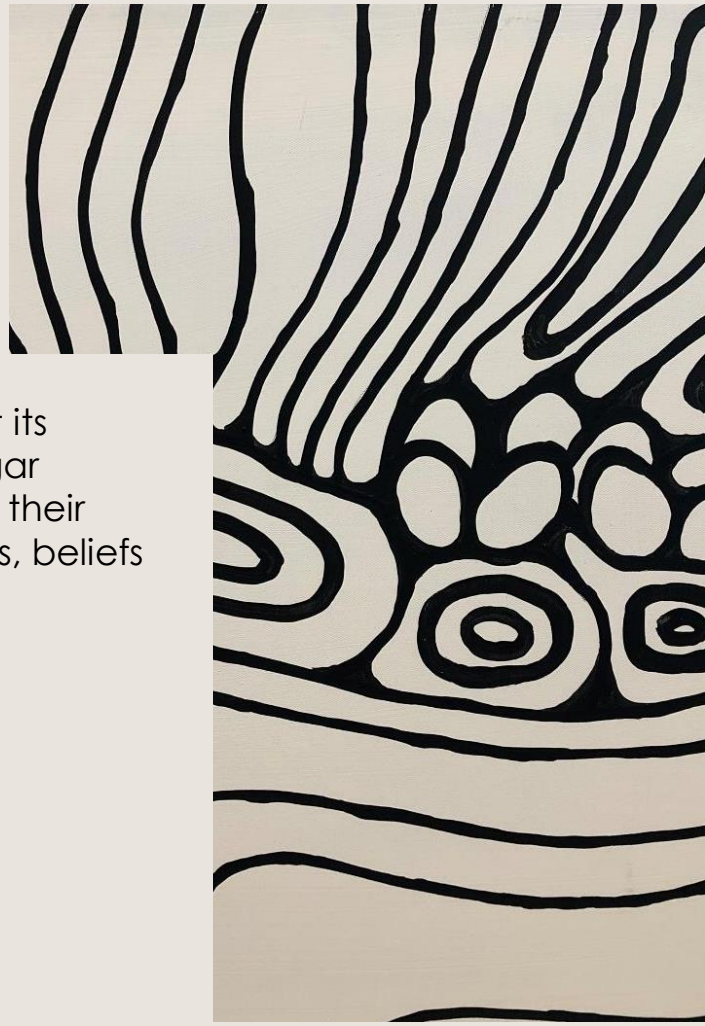
DDS DipOPath MDSc MFDS RCPS FHEA FRCPath FFOMP(RCPA)PhD

Acknowledgement
of country

The University of Western Australia acknowledges that its campus is situated on Noongar land, and that Noongar people remain the spiritual and cultural custodians of their land, and continue to practise their values, languages, beliefs and knowledge.



Artist: Dr Richard Barry Walley OAM

Learning outcomes

1. Describe common teeth and jaws infections
2. Identify the aetiology of teeth and jaws infections.

Infections

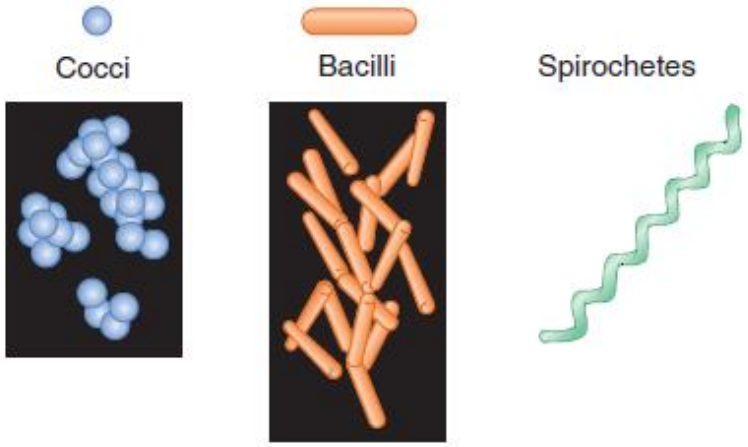
- **Microorganisms** cause infectious diseases**.**
- **Microorganisms:**
  - bacteria
  - Viruses
  - Fungi
  - Prions
  - Protozoa
  - Helminthes
  - arthropods.
- Not all microorganisms are **pathogenic**
- The most common infectious agents in the oral cavity are **periodontal** pathogens and **cariogenic** microorganisms.

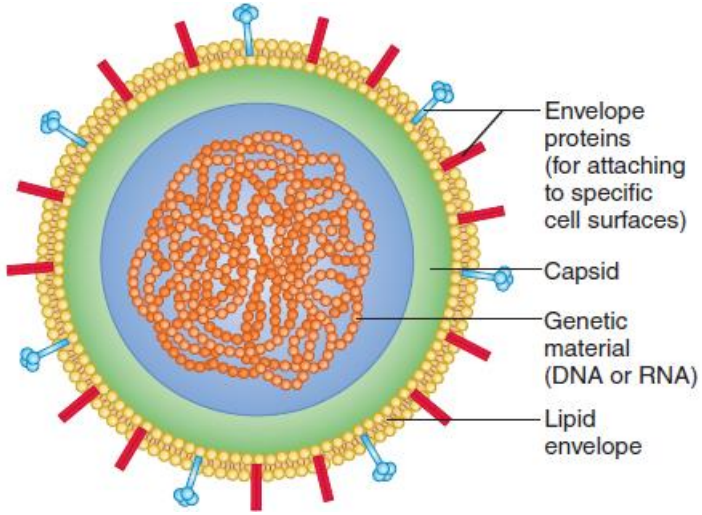
Infections

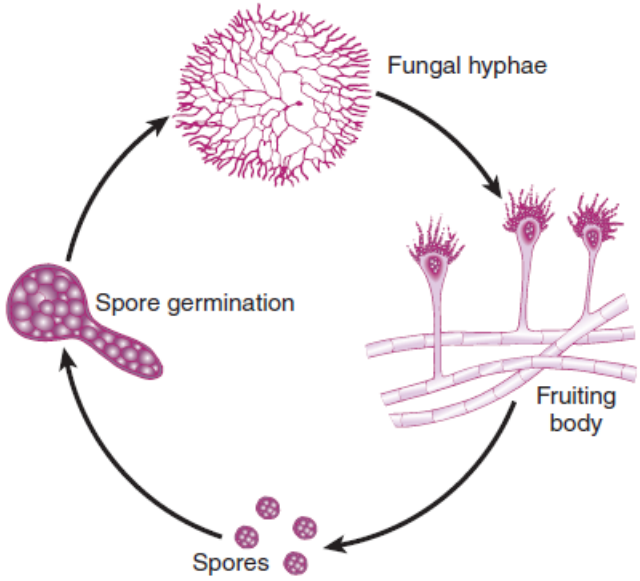
- **Opportunistic infection** is caused by microorganisms that usually do not produce disease in a person with a healthy immune system. When the immune system is compromised in some way, and the host's natural defenses against the pathogen are reduced or eliminated, these microorganisms may cause disease.

- Opportunistic infections are especially common in patients with immunosuppressive conditions such as AIDS.

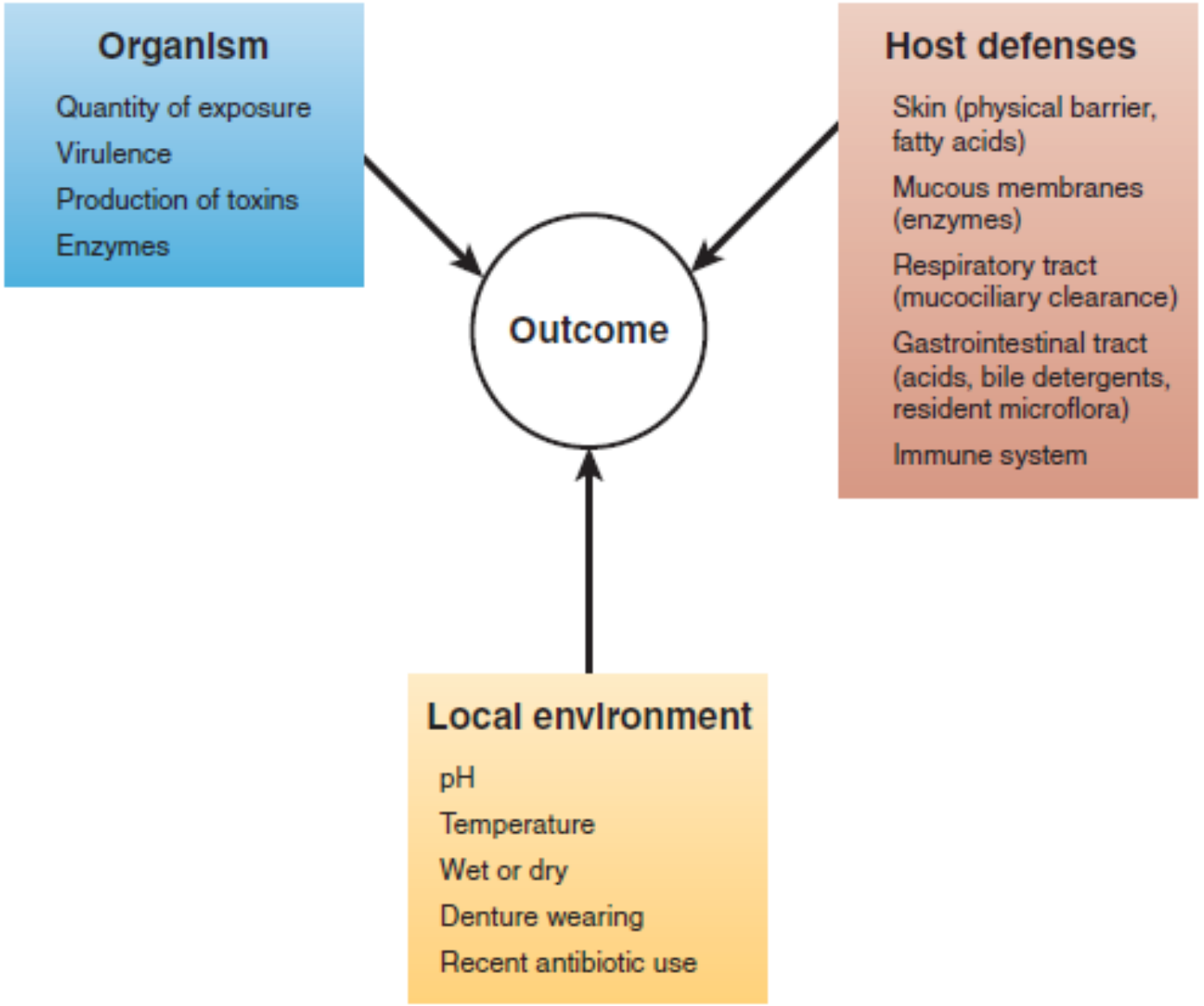
Microorganisms

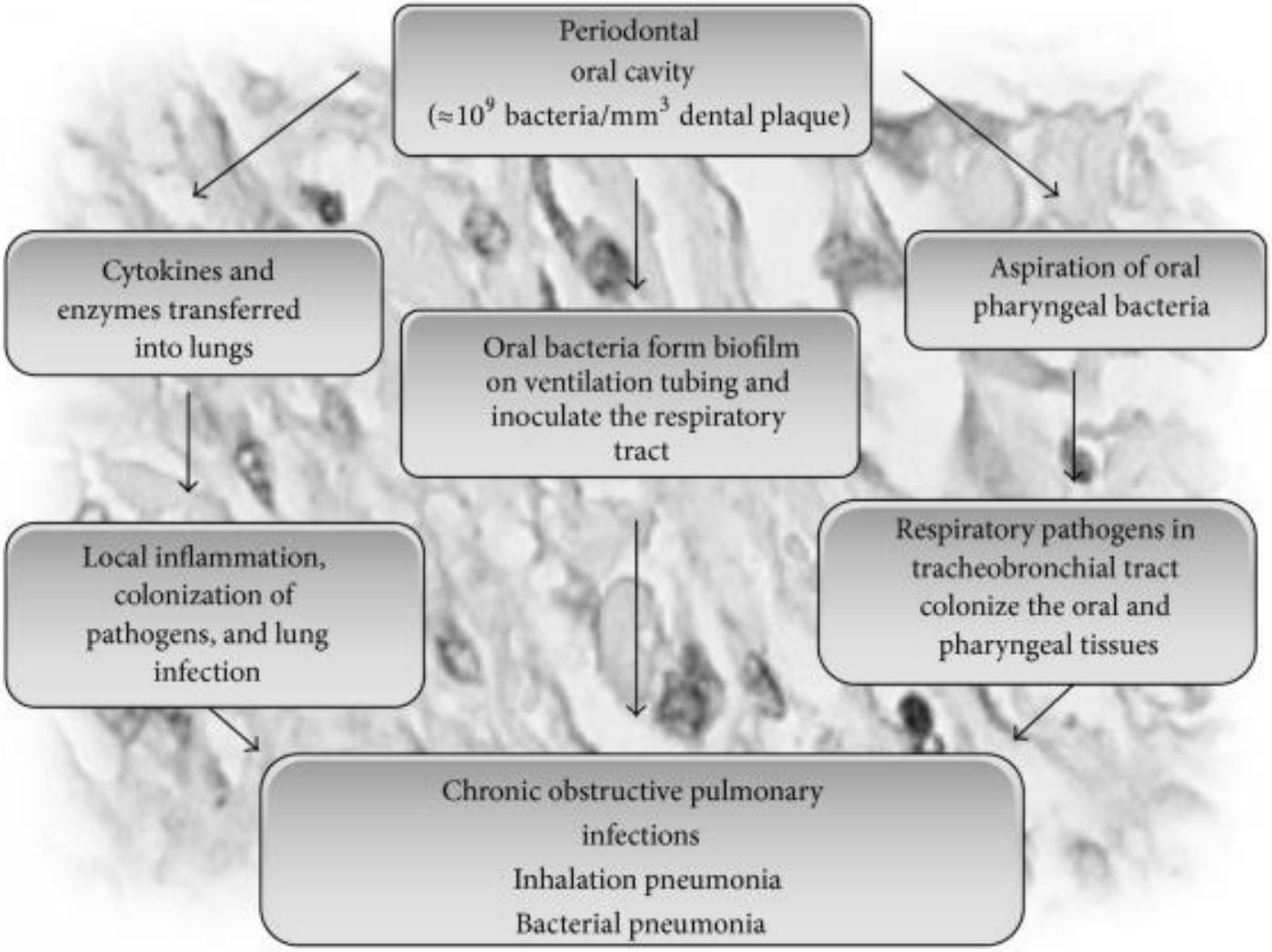






Variables affecting outcome of exposure to infectious agent

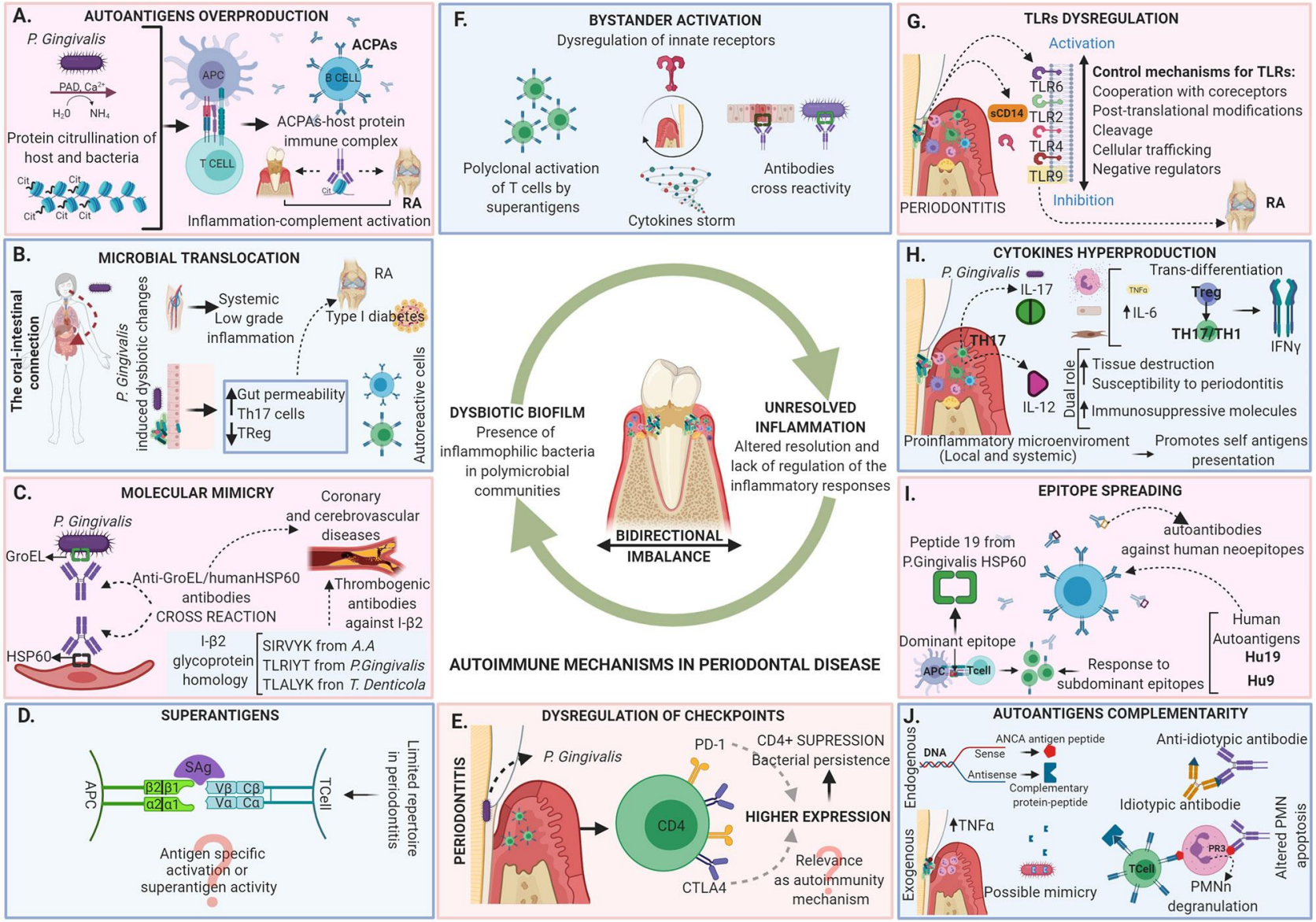




https://onlinelibrary.wiley.com/doi/10.1155/2015/793898

Oral dysbiosis and autoimmunity

https://www.frontiersin.org/articles/10.3389/fimmu.2020.591255/full



Clinical Signs and Symptoms of Infection

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- Signs and symptoms of infection may be caused by the organism directly or by the host response to being infected.
- Infections may be localized, systemic, or disseminated.
- Localized infections tend to cause pain in a specific body part. For example, if a cut on the skin is infected with bacteria, pain will occur at the site of the infection. The lesion will show redness, swelling, heat, edema, possibly a foul odor, and some form of exudate, such as pus.
- Systemic and disseminated infections tend to show extreme fatigue, weight loss, low-grade or spiking fever, night sweats and chills, and generalized body aches.

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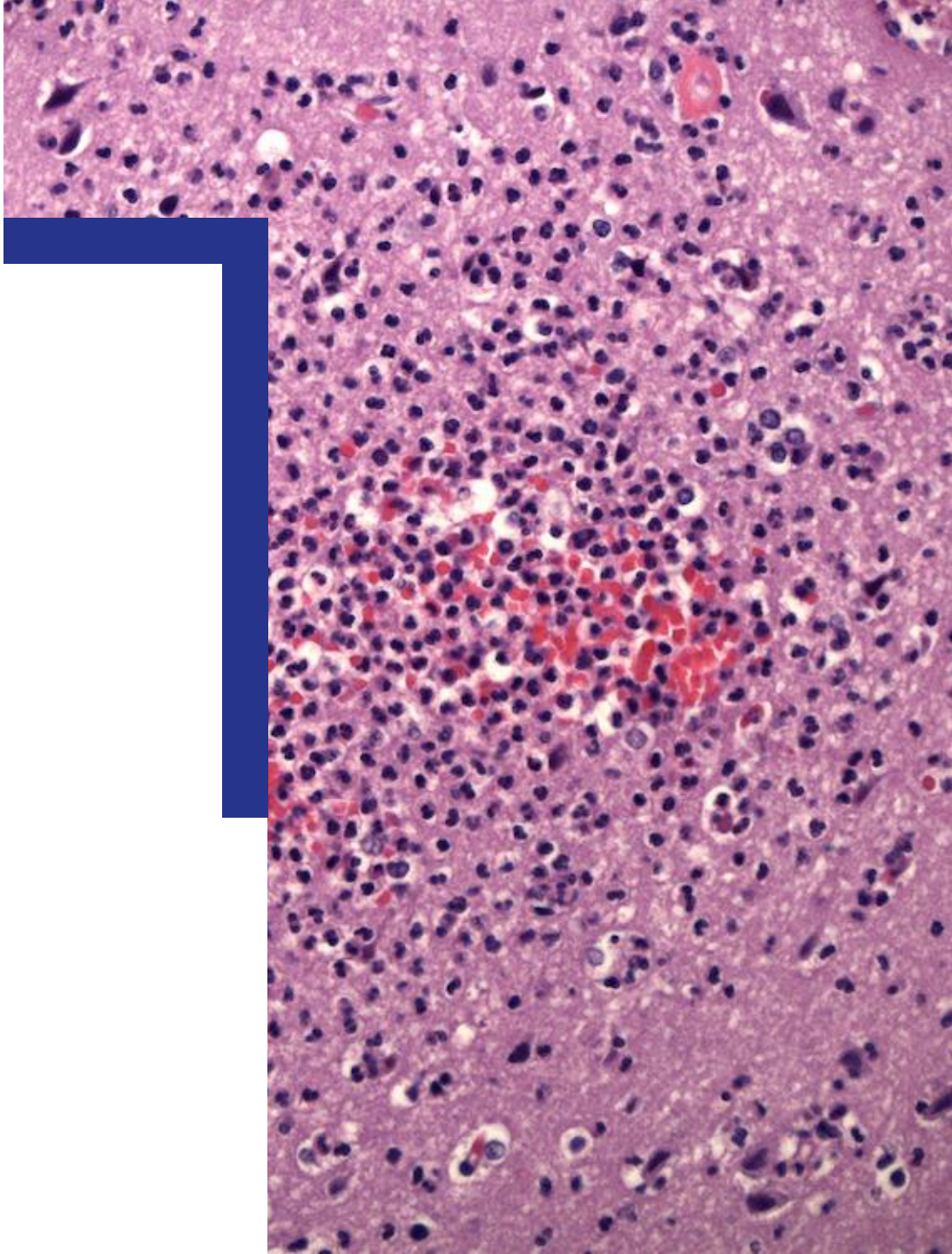
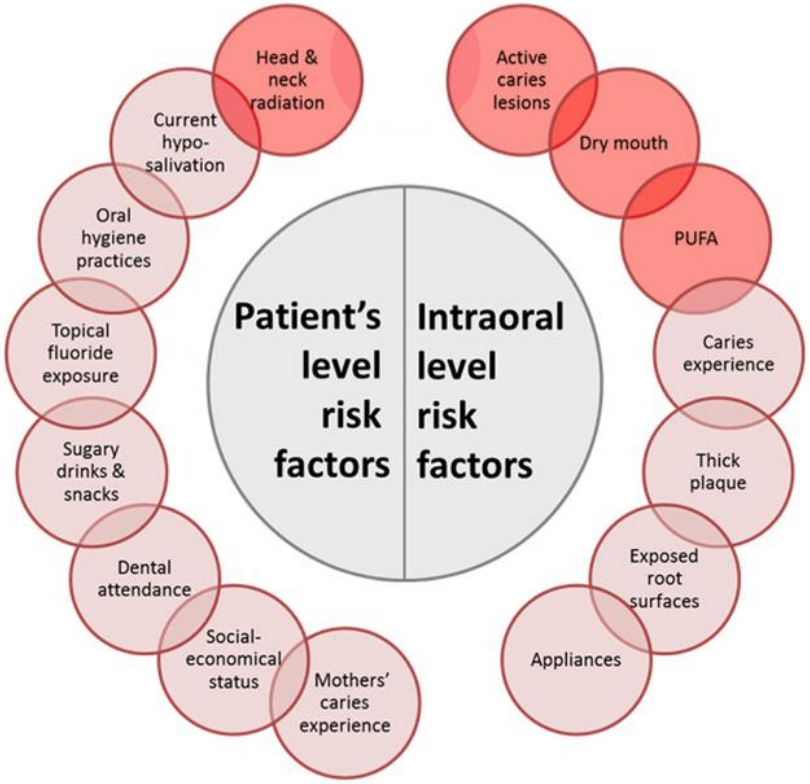
Bacterial infections



Dental caries



https://bmcoralhealth.biomedcentral.com/articles/10.1186/1472-6831-15-S1-S12

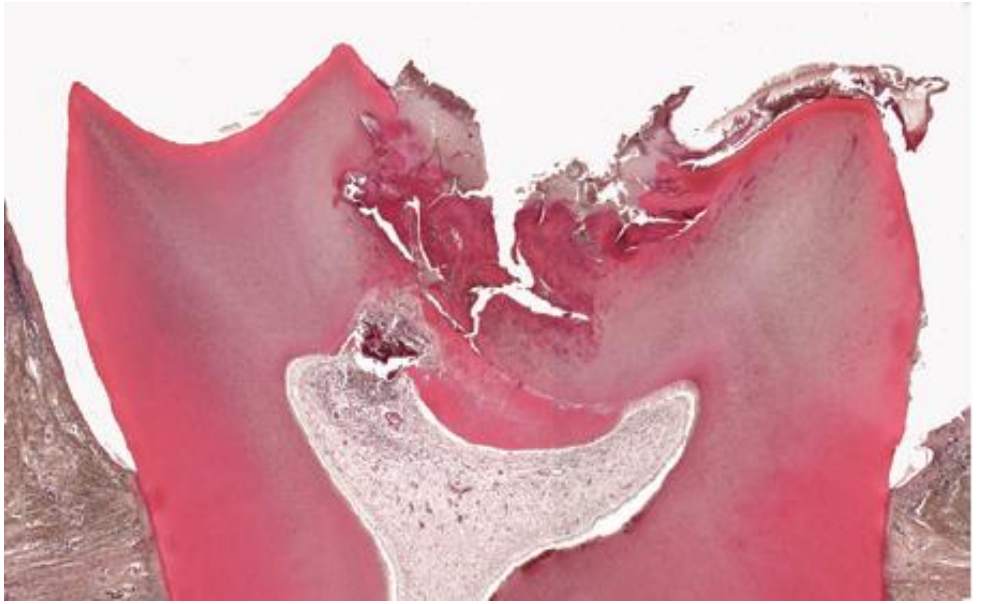
Pulpitis

Causes of pulpitis

- Dental caries
- Traumatic exposure of the pulp
- Fracture of a crown or cusp
- Cracked tooth
- Thermal or chemical irritation

Types of pulpitis

- Acute closed pulpitis
- Chronic closed pulpitis
- Open pulpitis
- Chronic hyperplastic pulpitis



PERIAPICAL PERIODONTITIS, ABSCESS AND GRANULOMA

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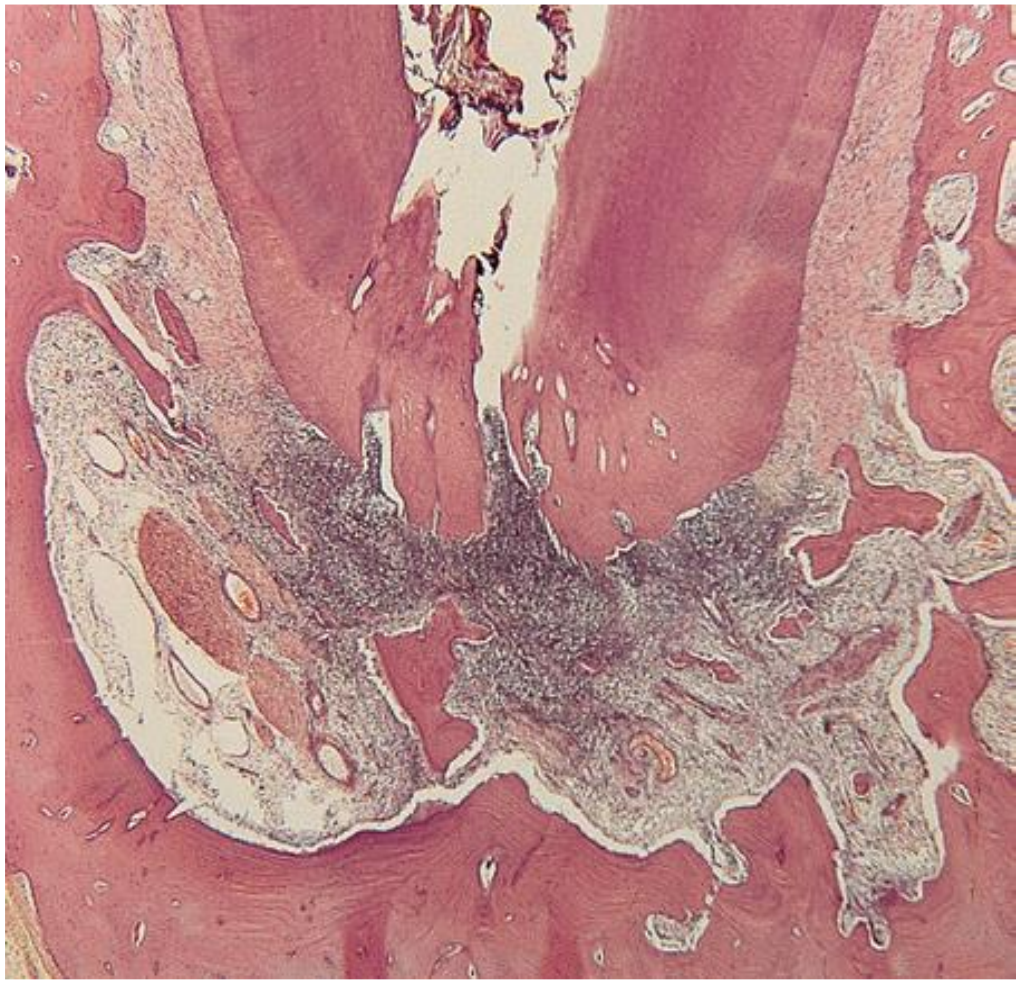
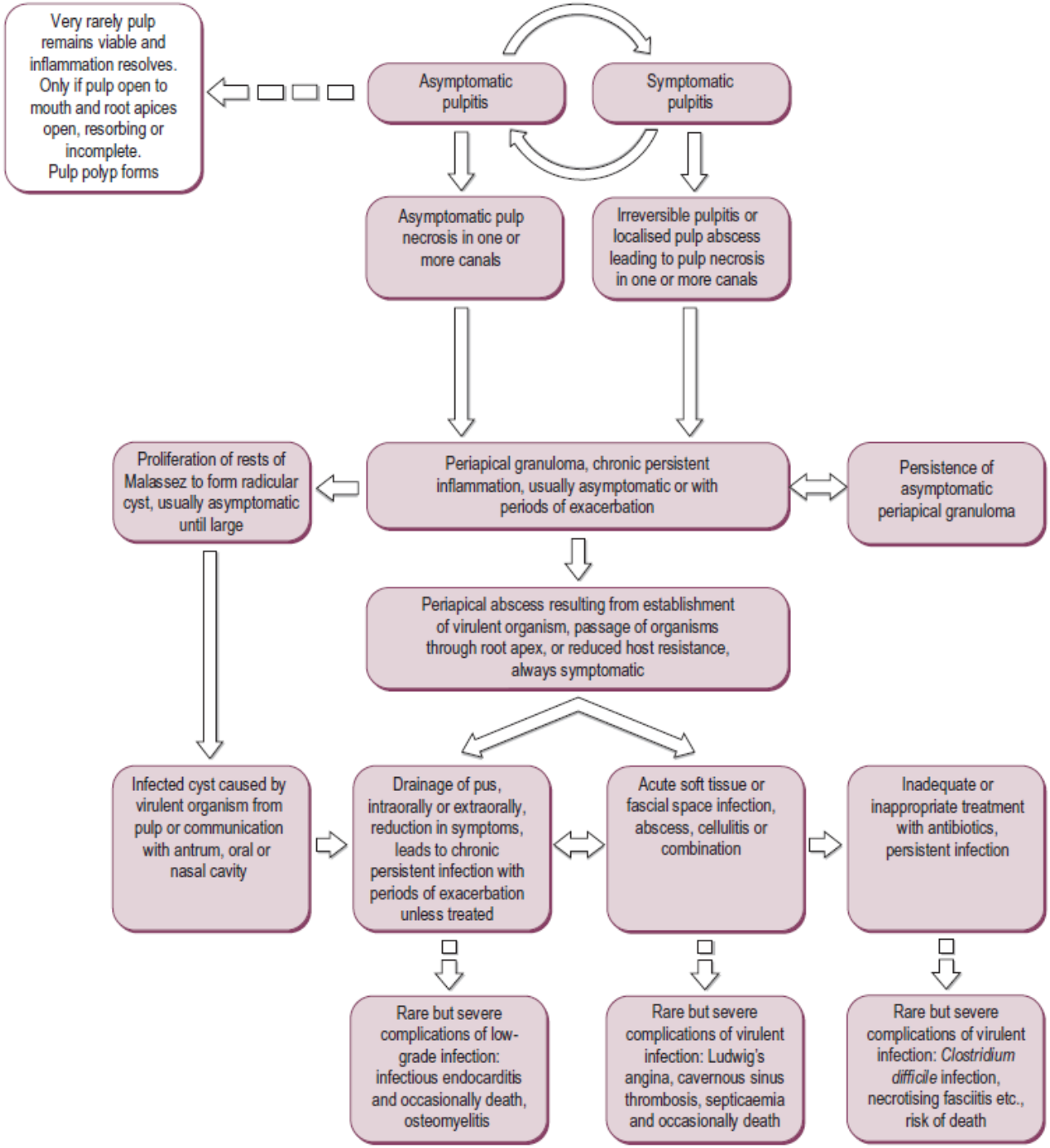
- Periapical inflammation is due to spread of infection, bacterial products or other irritants through the apex into the periodontal ligament following death of the pulp.

Causes of apical periodontitis

- Infection
- Trauma
- Chemical irritation




Very rarely pulp remains viable and inflammation resolves. Only if pulp open to mouth and root apices open, resorbing or incomplete. Pulp polyp forms
Asymptomatic pulpitis
Symptomatic pulpitis
Asymptomatic pulp necrosis in one or more canals
Irreversible pulpitis or localised pulp abscess leading to pulp necrosis in one or more canals
Proliferation of rests of Malassez to form radicular cyst, usually asymptomatic until large
Periapical granuloma, chronic persistent inflammation, usually asymptomatic or with periods of exacerbation
Persistence of asymptomatic periapical granuloma
Periapical abscess resulting from establishment of virulent organism, passage of organisms through root apex, or reduced host resistance, always symptomatic
Infected cyst caused by virulent organism from pulp or communication with antrum, oral or nasal cavity
Drainage of pus, intraorally or extraorally, reduction in symptoms, leads to chronic persistent infection with periods of exacerbation unless treated
Acute soft tissue or fascial space infection, abscess, cellulitis or combination
Inadequate or inappropriate treatment with antibiotics, persistent infection
Rare but severe complications of low-grade infection: infectious endocarditis and occasionally death, osteomyelitis
Rare but severe complications of virulent infection: Ludwig's angina, cavernous sinus thrombosis, septicaemia and occasionally death
Rare but severe complications of virulent infection: *Clostridium difficile* infection, necrotising fasciitis etc., risk of death

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Toothache or pain felt in teeth or alveolus

Pain of pulpal origin Sensitivity to sweet, hot or cold, poorly localised			Pain of periodontal ligament origin Pain on biting or pressure on tooth, usually well localised to one or more teeth					
Dentine hypersentivity	Pulpitis Tooth is vital or partially vital and may be hypersensitive to testing		Cracked tooth or cusp	Periapical periodontitis	Periodontal abscess	Sinusitis	Neurological or vascular pain	Neuralgic or psychogenic pain
	Reversible pulpitis	Irreversible pulpitis						
Pain of short duration more or less limited to period of stimulus, particularly cold	Symptoms may be limited to duration of stimulus or persist for varying period afterward, caries or other cause may be evident	Poorly defined entity, usually identified by severe continuous or spontaneous pain	Shooting or electric shock-like pain on biting, often only on one cusp or in one direction, also when a fracture line involves periodontal ligament	Pain on pressure to single tooth, caries or other cause of pre-existing pulpitis may be present, periapical, lateral canal or furcation radiolucency only in longstanding cases	Pain on pressure to single tooth, tooth vital, abscess in ligament visible or revealed by probing furcation or deep pocket	Tenderness on pressure to teeth with apices near sinus, usually concurrent or recent nasal or sinus symptoms, not usually severe pain	Teeth vital unless previously devitalised for other reasons	Unusual localisation trigger or perceived cause, associated with depression, anxiety or delusional states, teeth vital unless previously devitalised for other reasons
Confirm diagnosis by identifying exposed dentine or tooth wear and applying appropriate treatment	May resolve on treating cause but once established may progress to irreversible pulpitis, even after an asymptomatic period	Responds most reliably to extirpation of pulp or extraction	Confirm by identifying crack	Resolves on drainage or extirpation of pulp or extraction	Resolves on drainage and local treatment or extraction	Resolves on treatment of sinusitis	Consider mimics of pulpitis such as trigeminal neuralgia and the prodromal symptoms of facial Herpes zoster infection	Consider atypical odontalgia, atypical facial pain, 'phantom tooth' etc., but only after excluding organic causes

CHRONIC APICAL PERIODONTITIS AND PERIAPICAL GRANULOMA

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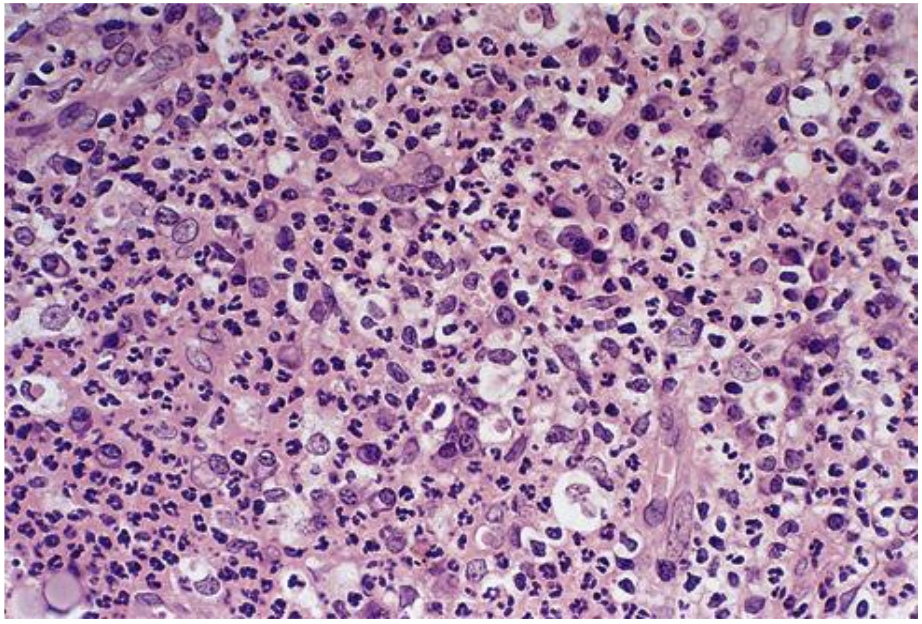
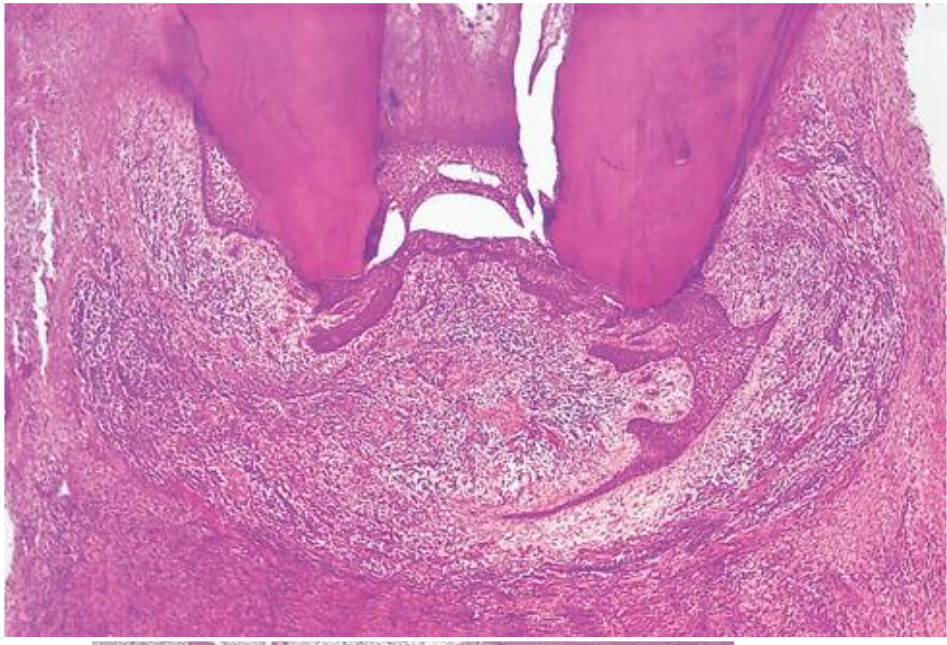
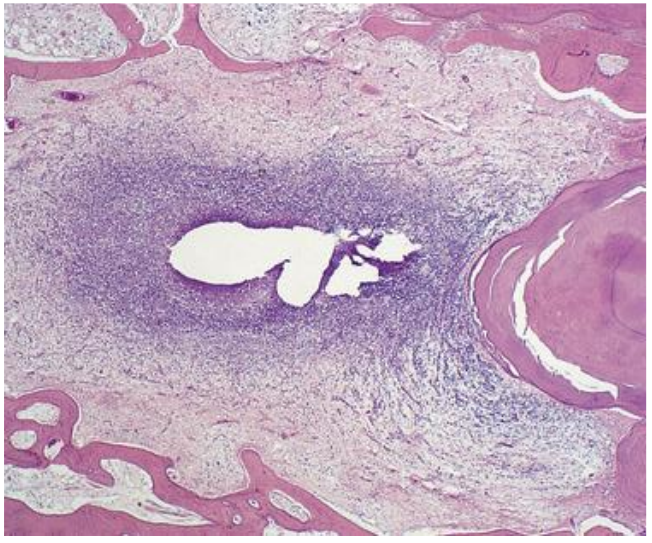
- Chronic periapical granuloma is the most frequent outcome of necrotic pulp.
- Most develop without symptoms.
- They can also arise from acute apical periodontitis
- A periapical granuloma is caused by frustrated healing.
- The granuloma itself is sterile in almost all cases, but bacteria and irritants from necrotic tissue remain in the pulp chamber, inaccessible to the host response.
- The tooth is non-vital and may be slightly tender to percussion; otherwise, symptoms may be minimal.

Periapical granuloma

- A periapical granuloma is a typical focus of chronic inflammation characterised by lymphocytes, macrophages and plasma cells in loose edematous fibrous tissue.







Periapical granuloma

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Possible outcomes of chronic apical periodontitis

- Periapical granuloma formation
- Radicular cyst formation
- Suppuration, sinus formation or spread
- Periodic acute exacerbations of inflammation of infection

Spontaneous resolution does not occur.

Gingivitis and periodontitis

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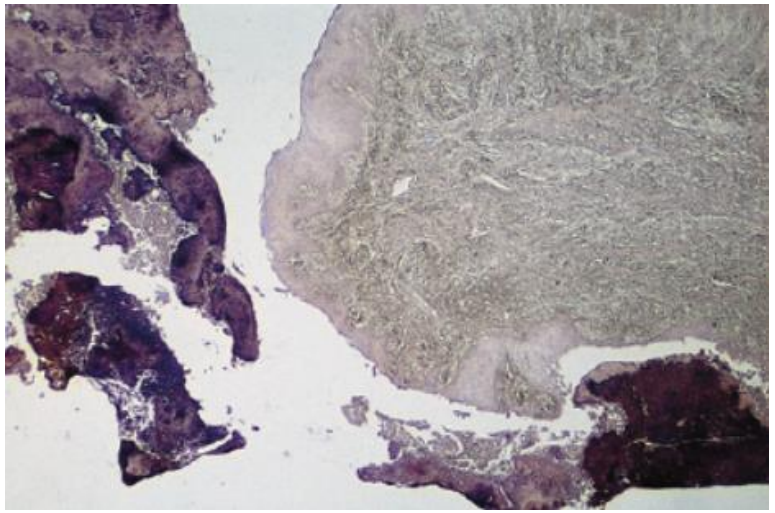
Pathological processes in chronic periodontitis

- Chronic inflammation
- Destruction of periodontal ligament fibres
- Resorption of alveolar bone
- Migration of the epithelial attachment to the apex
- Formation of pockets around the teeth
- Formation of subgingival plaque and calculus

Acute pericoronitis

- Pericoronitis is a localized, intraoral soft tissue infection most commonly associated with erupting lower third molars.
- The microflora of pericoronitis is diverse and differs from pathogens that cause periodontitis.
- In a study of microbiota of pericoronitis, *Actinomyces oris, Eikenella corrodens, Eubacterium nodatum, Fusobacterium nucleatum, Treponema denticola,* and *Eubacterium saburreum* were present in high levels
- Usually is triggered by trauma from the opposing dentition



Acute pericoronitis

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Differential diagnosis

- Foreign body impaction
- Pyogenic granuloma
- Peripheral ossifying fibroma
- Dental caries
- Periodontitis
- Periapical abscess or granuloma

Gingival inflammatory swellings

- *Orofacial granulomatosis*
- *Sarcoidosis*
- *Acute leukaemia*
- *Wegener's granulomatosis*
- *Scurvy*

Alveolar osteitis

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Key features

- The most common painful complication of dental extractions
- Loss of clot normally filling extraction socket
- Loss of clot may be due to excessive local fibrinolytic action or bacterial enzymes or both
- Bare, whitish lamina dura exposed in socket.
- Pain relieved by irrigation and repeated dressing of socket
- Dead bone usually shed as crumblike fragments
- Eventual healing of socket from its base by granulation

- Alveolar osteitis develops more frequently after 1%–2% of extractions for lower-third molar extractions.

Predisposing factors for alveolar osteitis

- Excessive extraction trauma
- Limited local blood supply
- Gingival infection such as acute ulcerative gingivitis, pericoronitis or abscess
- Local anaesthesia with vasoconstrictor
- Smoking
- Oral contraceptives
- Osteosclerotic disease: Paget's disease, cemento-osseous dysplasia
- Radiotherapy
- History of previous dry socket

ACUTE OSTEOMYELITIS

- In acute osteomyelitis bacteria and inflammation spread through the medullary bone from a focus of infection.
- The most common cause is spread of infection from a periapical infection.
- Adult males who have more dental infections than females are mostly affected.
- Almost all cases affect the mandible, which is less vascular than the maxilla.
- Early complaints are severe, throbbing, deep-seated pain and swelling with external swelling due to inflammatory edema. Later, distension of the periosteum with pus and, finally, subperiosteal bone formation cause the swelling to become firm. The overlying gingiva and mucosa is red, swollen and tender. Associated teeth are tender

ACUTE OSTEOMYELITIS

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Important predisposing conditions for osteomyelitis

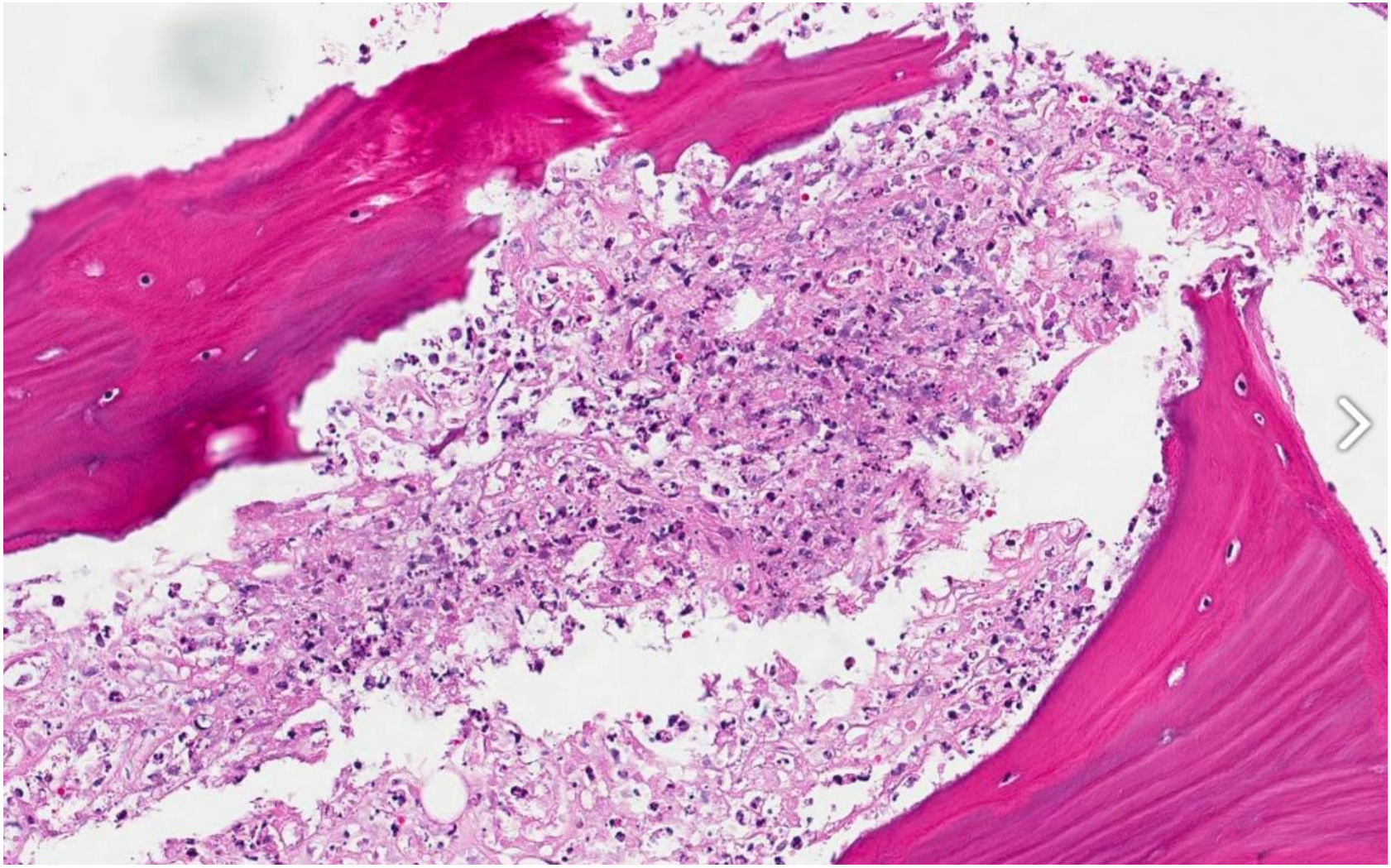
Local damage to or disease of the jaws

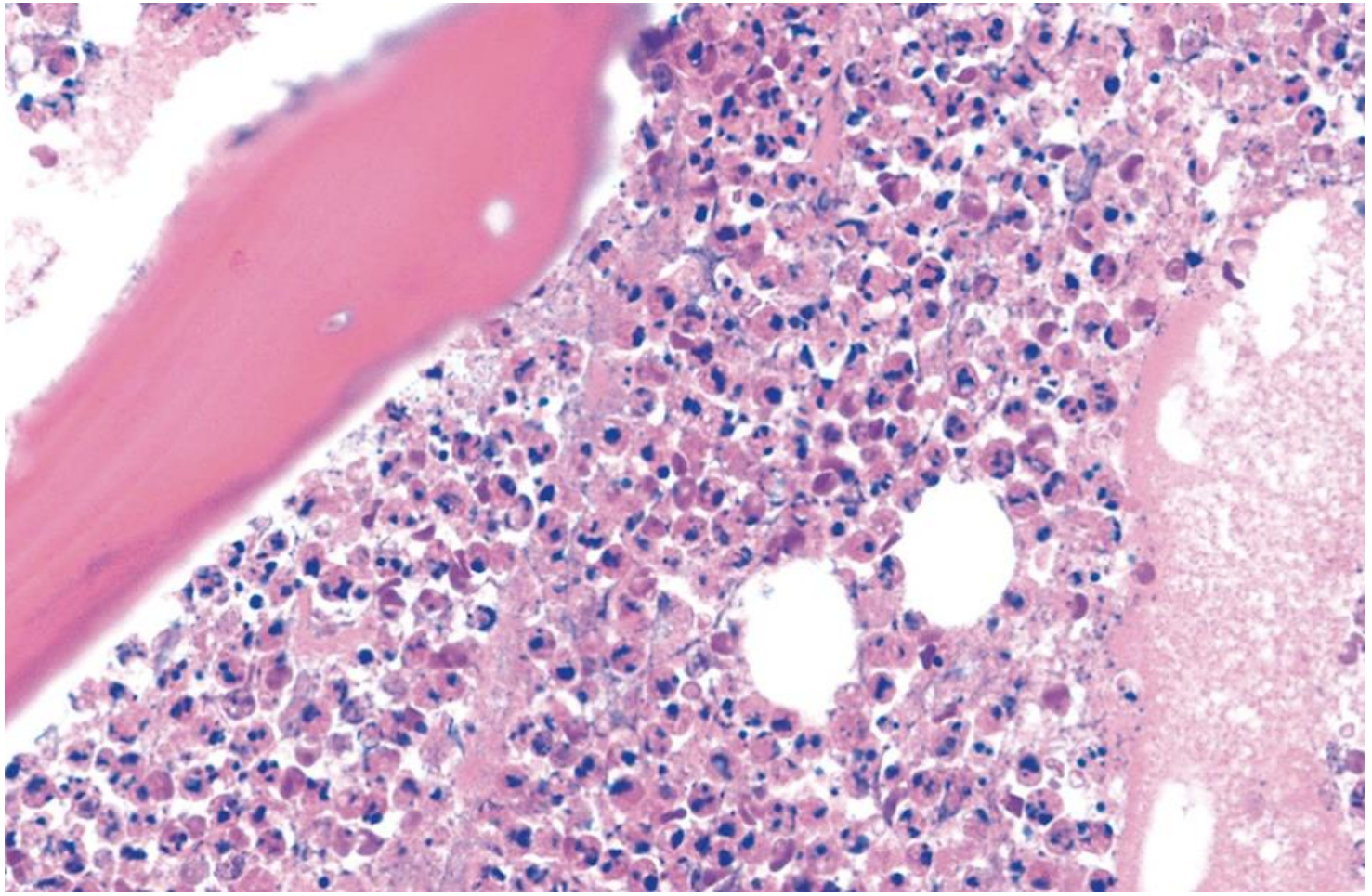
- Radiation damage
- Causes of osteosclerosis
- Paget's disease
- Fibro-osseous lesions, particularly cemento-osseous dysplasia
- Osteopetrosis

Impaired immune defences

- Poorly controlled diabetes mellitus
- Sickle cell anaemia
- Chronic alcoholism or malnutrition
- Drug abuse
- Tobacco smoking
- Malignant neoplasms and their treatment

ACUTE OSTEOMYELITIS



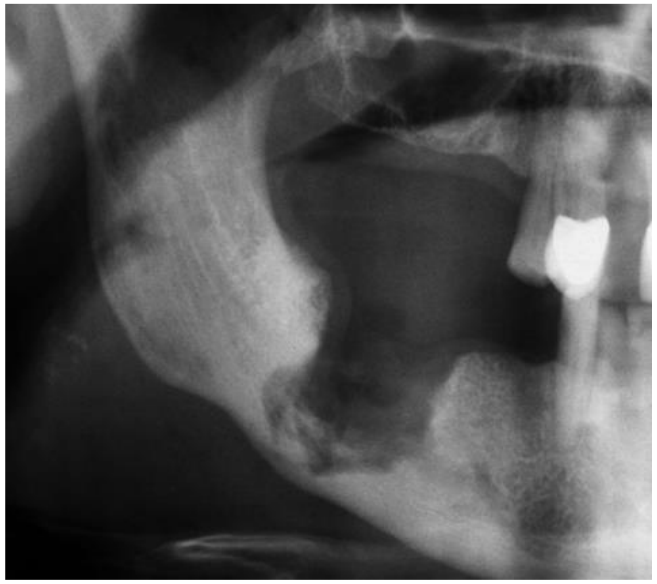


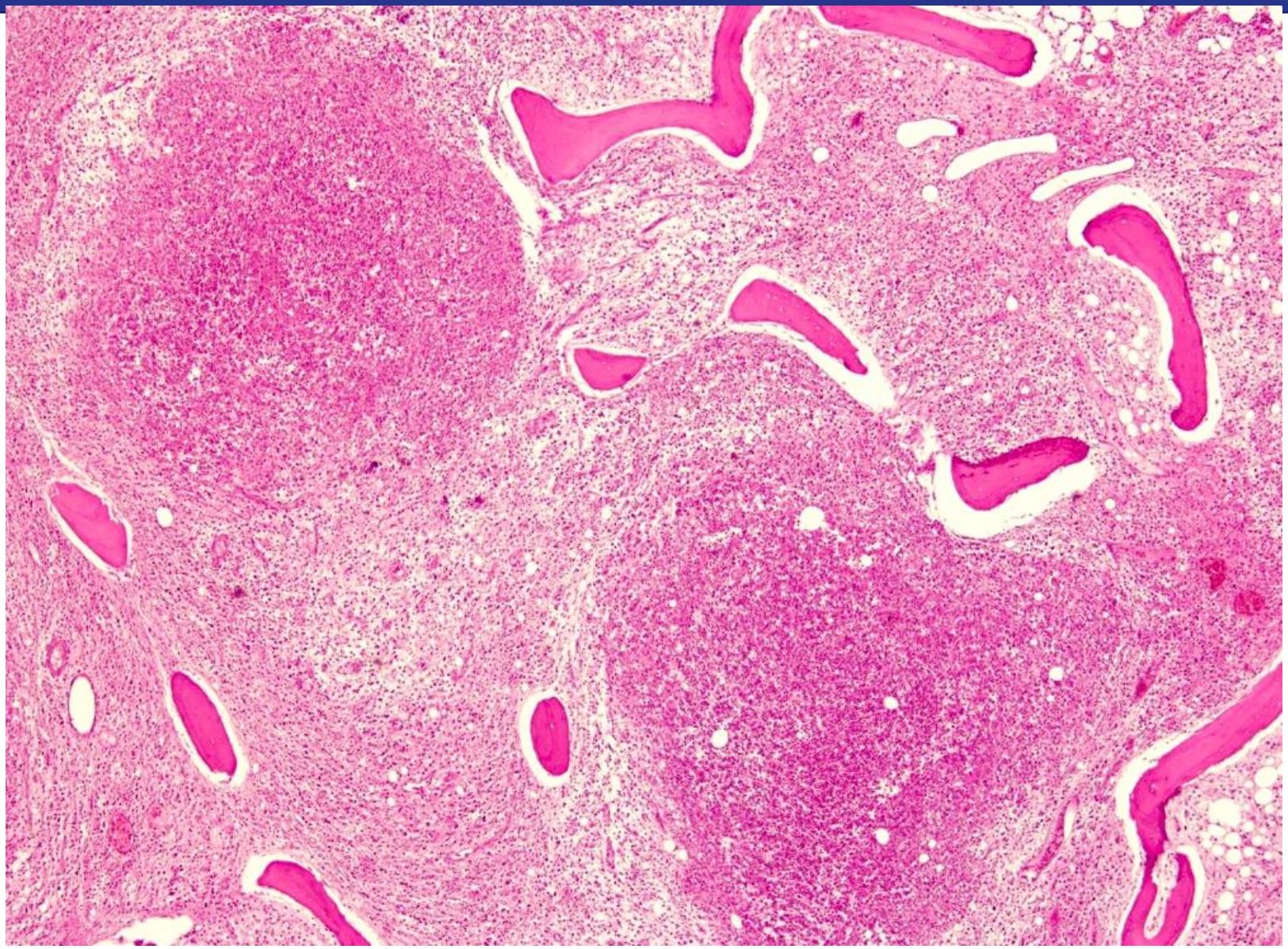
CHRONIC OSTEOMYELITIS

- More common than acute osteomyelitis and arises from infection by weakly virulent bacteria or in avascular bone.
- Most cases develop without a prior acute phase and only rarely does acute osteomyelitis lead to chronic osteomyelitis

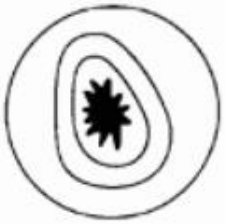
Key features

- Mandible mainly affected
- Infection of dental origin
- Low-grade pain
- Sclerosis or avascular bone is often a predisposing factor
- Resistant to treatment
- Prolonged antibiotic treatment required
- Role for surgery to remove sequestra and sclerotic bone





Stage I (Medullary osteomyelitis)



Necrosis limited to medullary contents and endosteal surfaces.
Etiology: Hematogenous
Treatment:
Early: Antibiotics/host alteration
Late: Unroofing, intramedullary reaming

Stage II (Superficial osteomyelitis)

Necrosis limited to exposed surfaces.
Etiology: Contiguous soft tissue infection
Treatment:
Early: Antibiotics/host alteration
Late: Superficial debridement/coverage
Possible ablation

Stage III (Localized osteomyelitis)

Well marginated and stable before and after debridement.
Etiology: Trauma, evolving stages I and II, latrogenic
Treatment:
Antibiotics/host alteration
Debridement, dead space management
Temporary stabilization, bone graft optional

Stage IV (Diffuse osteomyelitis)

Circumferential and/or permeative.
Unstable prior to or after debridement
Etiology: Trauma, evolving stages I, II, and III, latrogenic
Treatment:
Antibiotics/host alteration
Stabilization - ORIF, external fixation (Ilizarov)
Debridement, dead space management
Possible ablation

DIFFUSE SCLEROSING OSTEOMYELITIS

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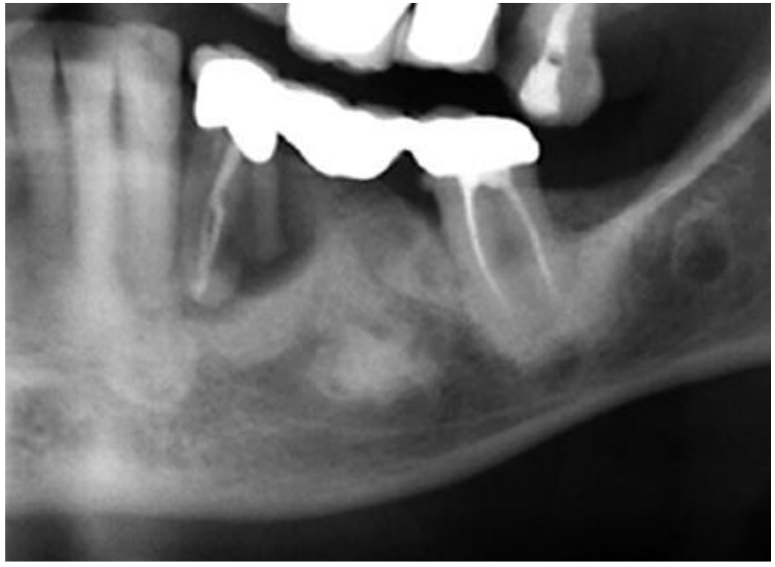
Key features

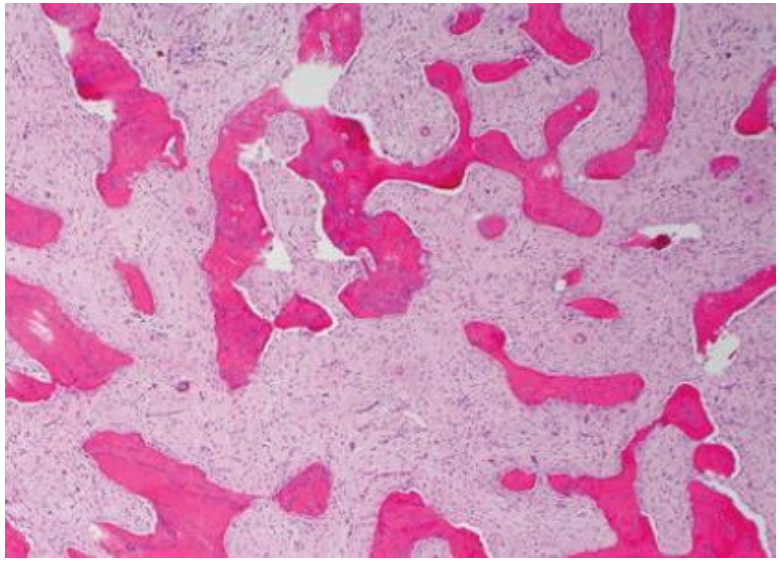
- Affects adults
- No sex predilection
- Affects mandible almost exclusively
- Patchy diffuse sclerosis in the alveolar process
- Changes more marked around sites of periapical or periodontal chronic inflammation
- Persistent ache or pain but no swelling
- Radiographically resembles but is distinct from florid cemento-osseous dysplasia

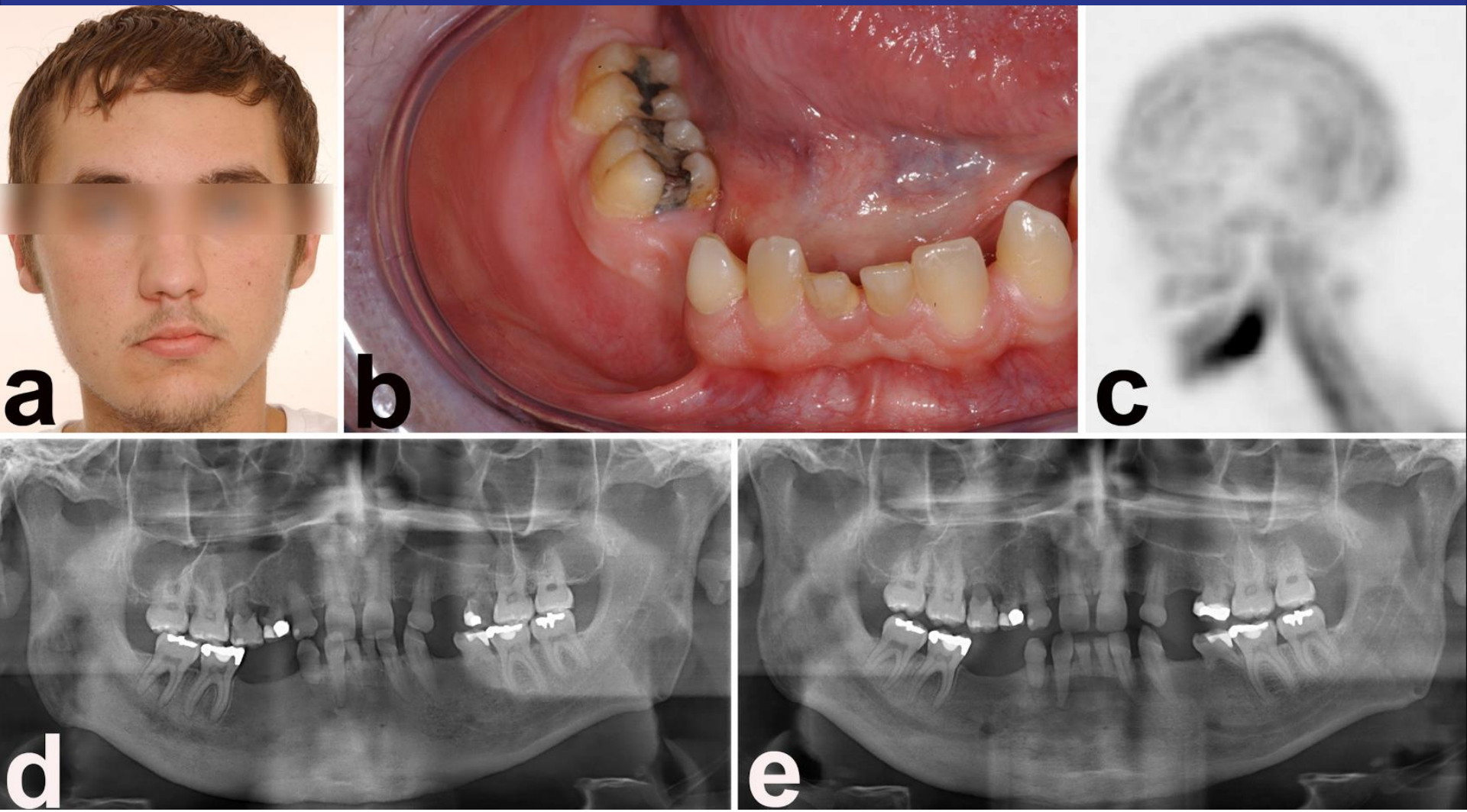


Pathology

- Bone sclerosis and remodelling
- Scanty marrow spaces and little or no inflammatory

infiltrate, although adjacent to areas of inflammation





https://www.sciencedirect.com/science/article/pii/S1010518215003066

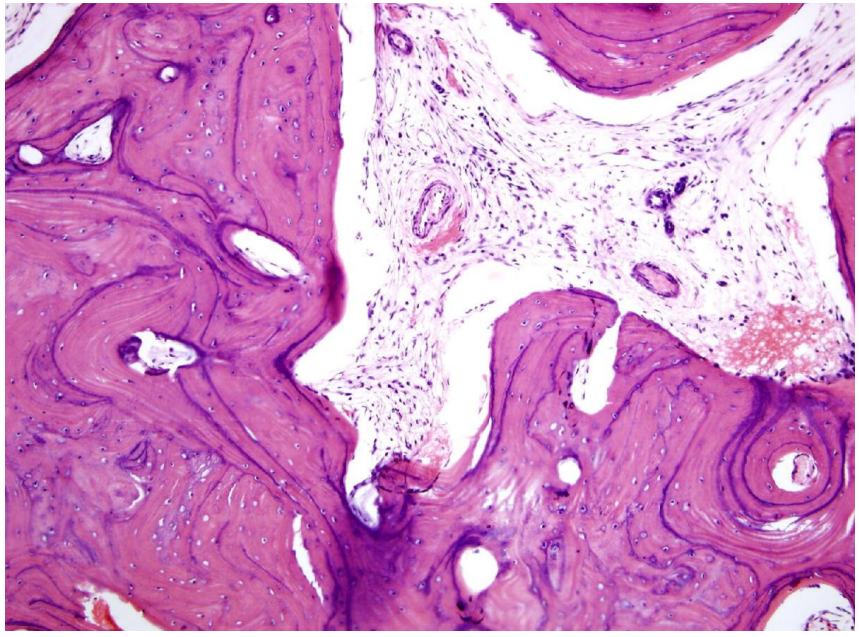
Focal sclerosing osteomyelitis

Key features

- Bony reaction to low-grade periapical infection
- Children and young adults affected
- Premolar or molar region of mandible affected
- Bone sclerosis associated with a non-vital tooth
- Localised but uniform radiodensity related to tooth with widened periodontal ligament space or periapical area
- No expansion of the jaw

Pathology

- Dense sclerotic bone with scanty connective tissue or inflammatory cells



Quiz

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Questions