Pulpal and peri-radicular disease-aetiology, progression and classification

Year 4 DDS - November 2014

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Outline:

i. Aetiology of pulpal and peri-radicular disease

ii. The pulp-dentine complex

iii. Reaction of the pulp-dentine complex to dental caries

iv. Progress of pulpal disease

v. Classification of pulpal and peri-radicular disease

vi. Microbiology of intra-canal infection

vii. Diagnostic procedures
i- Aetiology of pulpal and peri-radicular disease:

**Mechanical:** Trauma, iatrogenic pulp exposure, over-instrumentation...

**Thermal:** Pulp test, cavity/ crown prep with insufficient cooling..

**Chemical:** Toxic materials, cements (e.g: zinc-phosphate).

**Microbial:** Caries, periodontal disease.

**Ageing??**
i- The pulp-dentine complex:

- One unit: embryologically, structurally and functionally

**Embryonic development:**

- Tooth development begins approximately during the sixth week in utero.
- A complex interaction between the oral epithelial cells of the ectoderm and the underlying ectomesenchymal cells.
- Development is divided into three stages according to the shape of the oral epithelium.

![Embryonic development stages](image)
Embryonic development:

a) Bud stage:

- Epithelial cells of the ectoderm proliferate into the subjacent ectomesenchyme and form "bud-like" projections.
Embryonic development:

b) Cap stage:

- The ectodermal epithelium proliferates around the ectomesenchymal cells, taking on the appearance of a cap.
- This epithelial aggregate is known as the enamel organ and is composed of outer and inner enamel epithelium separated by stellate reticulum and stratum intermedium cells.
- The enamel organ later produces enamel.
- Ectomesenchymal cells enclosed by the enamel organ are known as the dental papilla which represents the future pulp.
- A layer of ectomesenchymal cells referred to as the dental follicle condenses and surrounds the enamel organ and the dental papilla.
Embryonic development:

c) Bell stage:

- The enamel organ continues to grow until the dental papilla is partially enclosed by the invaginating epithelium, taking on a bell shape.
- Histodifferentiation and morphodifferentiation takes place
- The inner enamel epithelial cells differentiate into ameloblasts.
- Peripheral cells of the dental papilla differentiate into odontoblasts.
- Dental follicle cells give rise to cementoblasts, osteoblasts and fibroblasts, which collectively form the periodontium.
- The rim at which inner and outer enamel epithelia join is known as the cervical loop. Its apical growth forms Hertwig’s epithelial root sheath and is responsible for the root formation.
**Types of dentine:**

1- Mantle dentine
2- Primary dentine
3- Secondary dentine
4- Tertiary dentine (reactionary or reparative)
5- Predentine
6- Dentinal tubules
7- Peritubular dentine
8- Intertubular dentine
9- Sclerotic dentine
The dental pulp:

Loose connective tissue that consists of water (75-80%), cells, fibrous matrix and ground substance and contains vascular, lymphatic and nervous elements.

**Cells:** odontoblasts, fibroblasts, macrophages, lymphocytes and undifferentiated mesenchymal cells.

**Fibrous matrix:** collagen fibers type I and II.

Type I collagen is thought to be produced by the odontoblasts, while Type II is probably produced by the pulp fibroblasts as this type increases in frequency with the age of the tooth.

**Ground substance:** the environment that surrounds both cells and fibers of the pulp is rich in proteoglycans, glycoproteins and large amounts of water.

Divided into the following layers: odontoblastic layer, cell-free zone, cell-rich zone and pulp proper.
ii- Reaction of the pulp-dentine complex to caries:

Three basic reactions of the pulp-dentine complex to protect the pulp against caries:

- Sclerosis of the dentinal tubules
- Deposition of tertiary dentine
- Inflammation and immune response
Enamel caries

1: Translucent Zone
2: Dark Zone
3: Body of the lesion
4: Surface Zone
Dentinal caries:

Zone of Destruction

- Following cavitation in enamel, bacteria infect the dentine which becomes necrotic, and liquified.
- This zone is soft and yellow and is easily removed with an excavator.
Dentinal caries:

Zone of Bacterial Invasion

• The tubules are invaded by bacteria.

• Acids produced dissolve both the inorganic (hydroxyapatite) and organic (collagen) components of dentine.
Dentinal caries:

Zone of Demineralisation

The advancing front of the carious lesion

Very small (less than 1mm).

No bacteria found in this zone.
Dentinal caries:

Sclerotic (Translucent) Zone

A defence response.
Odontoblasts lay down mineral deposits within the tubules, which become plugged.
This slows down the acid advance, giving the pulp some protection.
Occasionally seen on radiographs as a radio-opaque area below the caries.
Dentinal caries:

Tertiary dentine:
To keep the pulp at distance from the coronal infection.
Reactionary or reparative
Enamel Cavitation

Destruction and ADJ Spread

Bacterial Invasion

Demineralisation

Sclerotic

Tertiary Dentine
iii- Progress of pulpal and peri-radiccular disease:

Like any other connective tissue, the pulp responds to tissue injury by means of inflammation.

Pulp inflammation develops long before the pulp becomes directly exposed and infected.

The extent of pulp inflammation depends on:

- Depth of bacterial intra-tubular invasion
- Bacterial virulence
- Degree of reduction of dentine permeability.

The pulp is not invaded by bacterial cells as long as it remains vital.

At this stage, appropriate clinical management leads to resolution of the inflammatory reaction. Hence the term “reversible pulpitis”.
iii- Progress of pulpal and peri-radicular disease:

- If caries remains untreated and active, the pulp increases the magnitude of its inflammatory response.
- Polymorphonuclear neutrophils (PMNs) becomes progressively attracted and accumulate in the subjacent pulp area.
- Vascular events including vasodilatation and increased vascular permeability take place which result in exudation.
- The inflammatory reaction may be severe and the damage is beyond repair.

A clinical state referred to as “irreversible pulpitis”.
iii- Progress of pulpal and peri-radiccular disease:

- Oedema formation results in increased hydrostatic pressure, which can exceed that of thin-walled venules and may result in impeded drainage and stagnation of blood flow.
- PMNLs continue to accumulate near the exposure area and contribute to tissue damage by releasing enzymes and free-radicals which degrade the pulp.
- Bacterial metabolites and enzymes also contribute to tissue damage
- Localized abscess formation can develop in the pulp adjacent to the exposed area.
- Accumulation of localised abscesses and dead cells eventually results in **pulp necrosis**.
iii- Progress of pulpal and peri-radicular disease:

• Following localised necrosis, bacterial infection gradually moves in an apical direction.

• The pulp tissue immediately adjacent to the infected region responds with the same inflammatory events described earlier.

• Therefore, after carious exposure, the pulp undergoes a series of events including, injury, inflammation, necrosis and infection until the entire pulp is necrotic and infected.

• The inflammatory response to the advancing infection may extend to the peri-radicular tissues even before the necrosis and infection reach the apical or lateral foramena.
iii- Progress of pulpal and peri-radicular disease:

- As the advancing infection reaches the apical (or lateral) foramen, the peri-radicular tissues develop a protective inflammatory response (both innate and adaptive immune responses) in an attempt to contain the spread of the infection to the bone and other body sites.
- The resulting peri-radicular inflammatory response depends on both:
  a) number and virulence of pathogenic bacteria and
  b) host defence
- The resulting peri-radicular inflammatory response can be either:
  i) Acute: **acute apical periodontitis**
     **acute apical abscess**
  ii) Chronic: **chronic apical periodontitis**
     **chronic apical abscess**
iv- Classification of pulpal and peri-radicular disease:

Pulp:
- Normal
- Reversible pulpitis
- Irreversible pulpitis
- Necrotic

Peri-radicular tissues:
- Normal
- Apical periodontitis
- Apical abscess
  - Acute
  - Chronic
  - Acute
  - Chronic
Normal pulp:

- Asymptomatic
- Responds normally to pulp tests:
  - Mild transient sensation
  - Does not cause distress to the patient
  - Sensation reversed immediately following stimulus removal
- Radiographically:
  - Normal apical anatomy:
    - Intact lamina dura
    - 0.2-0.3 mm PDL space
  - No evidence of resorption or pulp exposure
**Reversible pulpitis:**

- The pulp is inflamed and symptomatic due to certain irritants.
- Pulp stimulation is uncomfortable to the patient but symptoms subside quickly.
- Caries, defective restoration, exposed dentine, recent dental treatment
- Radiographically:
  - Normal lamina dura
  - No evidence of resorption or pulp exposure
- Must be differentiated from dentinal hypersensitivity.
- The symptoms are relieved and inflammation is reversed once the irritation source is removed.
Irreversible pulpitis:

An established state of pulpal inflammation from which the pulp cannot recover.

Pain:

• Usually intermittent and spontaneous
• Usually dull and poorly localized
• Aggravated upon exposure to temperature changes (esp. cold).
• Lasts for a prolonged period following stimulus removal

Deep caries, defective deep restoration, pulp exposure or any insult to the pulp.
No or minimal radiographic changes (i.e: thickening of the apical PDL space may be evident)

Occasionally, deep caries does not produce any symptoms, even though clinically and radiographically the caries may well be into the pulp. A state known as “asymptomatic irreversible pulpitis”
**Pulp necrosis:**

The ultimate outcome of pulpitis.

May also occur after trauma that can deprive the pulp from its blood supply.

If bacteria gain access to a necrotic pulp, a lack of blood supply means that no inflammatory and immune cells are present to fight against infection.

Also bacteria are capable of degrading organic necrotic tissue for use as a substrate.

Tooth is typically asymptomatic (before the pathosis extends into the periodontium)

Tooth non-responsive to electric or cold pulp tests. However, applying heat may cause pain due to remnants of pulpal fluids or gazes.

Radiographic changes range from thickening of the apical PDL space to the development of peri-apical radiolucent lesion.
Acute apical periodontitis (AAP):

Tooth painful to biting pressure or percussion

May or may not respond to pulp testing.

May be caused by:

- Extension of pulpal inflammation
- Reaction to intra-pulpal infection
- Trauma

Radiographically: widened PDL space but no peri-apical radiolucent lesion.

Primary AAP: inflammatory response starts within a healthy tissue.

Secondary AAP: acute response starts in an already chronically inflamed tissue.

(Acute exacerbation of a chronic inflammation).
Chronic apical periodontitis:

- A long standing inflammatory reaction characterized by the presence of granulation tissue known histologically as a periapical granuloma.
- Generally presents with no symptoms. Though the tooth may feel “different” to the patient upon percussion.
- Negative response to pulp tests.
- Radiographically: an apical radiolucency that reflects a state of equilibrium between the host defensive response and the infectious process.
- Any disturbance in the equilibrium that favors the growth of the infectious microflora can lead to an acute disease in the form of secondary acute apical periodontitis or secondary apical abscess (phoenix abscess).
**Apical abscess:**

A category of apical periodontitis where there is localized collection of pus.

**Acute apical abscess:**

An extremely painful condition characterized by:

- Intense pain and tenderness to biting, pressure, percussion or palpation
- Intra-oral or extra-oral swelling
- Varying degrees of mobility
- Possibly systemic signs of fever, malaise and lymphadenopathy.

The culprit tooth has an infected pulpal space. Therefore negative response to pulp tests.

Radiographic changes may range from a widened apical PDL space to a periapical radiolucency.

Acute apical abscess can be primary or secondary.
Primary acute apical abscess:

Develops as a result of primary acute apical periodontitis.

No radiographic radiolucent changes apart from slight thickening of the periodontal membrane space which occurs as a result of the tooth being slightly extruded from its normal position in the socket by the inflammatory process and fluid build-up.

Secondary acute apical abscess:

Develops as an acute exacerbation of chronic apical periodontitis. Characterized by all the symptoms of primary acute apical abscess in addition to a periapical radiolucent area.
Chronic apical abscess (chronic suppurative periodontitis)

- A draining sinus tract is the hallmark of chronic apical abscess.
- Usually not associated with pain due to the relief provided by the draining sinus.
- Can be drained either intra or extra-orally.
- Radiographically, a radiolucent periradicular area is evident.
- Can be traced by inserting a gutta percha point in the sinus tract and exposing a radiograph, the offending root, can be identified readily.
Cysts:

Definition: A pathological bone cavity containing liquid, semi-liquid or gaseous content, frequently but not always lined with epithelium.

- Epithelial
  - Odontogenic
  - Inflammatory
    - Radicular: Apical, Lateral, Residual
    - Paradental
  - Developmental
    - Keratocyst
    - Dentigerous
    - Eruption
    - Lateral-periodontal
    - Gingival

- Non-odontogenic
  - Non-odontogenic
  - Nasopalatine
  - Nasolabial
  - Median

- Non-epithelial
  - Solitary bone cyst
  - Aneurysmal
  - Stafne’s idiopathic
Cysts:

Theories of cyst expansion:

1- Hydrostatic theory: The cyst wall acts as a semi-permeable membrane which draws fluid only from outside to inside.

2- Proliferation of epithelial lining: Surface area of cystic sac increases by division of cells surrounding the cyst.

3- Prostaglandin theory: epithelial cells release bone resorption factors which stimulate the breakdown of bone cells.
Apical radicular cysts:

Epithelium-lined pathological cavities of inflammatory origin.

Epithelium: from Hertwig’s epithelial root sheath or remnants or enamel epithelium

They are of two types: those self sustaining cavities entirely lined by epithelium where no communication with the root canal exists are termed true cysts. When the epithelium-lined cavity is open to the root canal of the offending tooth, it is known as pocket cyst.
Chronic hyperplastic pulpitis (pulp polyp):

- Seen particularly open carious lesions in primary molars and newly erupted permanent molars (children and young adult)
- Due to the presence of good blood supply and large root opening.
- Not sensitive to touch (little nerves in the hyperplastic tissue),
- Can be confused with hyperplastic gingiva which extended into the carious defect.
- Because the tooth is open to the oral cavity, transudates and exudates from the inflamed pulpal tissue drain freely and do not accumulate within the restricted and rigid confines of the tooth.
Chronic hyperplastic pulpitis (pulp polyp):

- Tissue necrosis with destruction of the microcirculation that usually accompanies irreversible pulpitis does not occur in part because of this lack of significant intra-pulpal pressure.

Histopathologic features:

- A mass of granulation tissue.
- Inflammatory cell infiltration, chiefly lymphocyte and plasma cells sometimes mixed with polymorphs.
- Sometimes covered with stratified squamous epithelium.

Treatment: by RCT or extraction
Condensing osteitis (Chronic focal sclerosing osteomyelitis/ Garre's disease):

A periapical inflammatory disease, a reaction to an infection of endodontic origin.

Causes more bone production destruction in the area

Most common site is near the root apices of premolars and molars

The lesion appears as a radiopacity in the periapical area

The sclerotic reaction results from good patient resistance and a low degree of virulence of the offending bacteria.

The associated tooth may be carious or contains a large restoration and usually associated with a non-vital tooth
Condensing osteitis (Chronic focal sclerosing osteomyelitis/ Garre's disease):

**Aetiology**: Infection of periapical tissues of a high immunity host by organisms of low virulence.

**Treatment**: The offending tooth should be tested for vitality of the pulp, if inflamed or necrotic, then endodondtic treatment is required, while hopeless teeth should be extracted

**Differential Dx**:  
- Idiopathic osteosclerosis  
- Cementoblastoma
<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
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<tbody>
<tr>
<td>Normal pulp</td>
<td>A clinical diagnostic category in which the pulp is symptom-free and normally responsive to pulp testing.</td>
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<tr>
<td>Reversible pulpitis</td>
<td>A clinical diagnosis based on subjective and objective findings indicating that the inflammation should resolve and the pulp return to normal.</td>
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<tr>
<td>Symptomatic irreversible pulpitis</td>
<td>A clinical diagnosis based on subjective and objective findings indicating that the vital inflamed pulp is incapable of healing. Additional descriptors: lingering thermal pain, spontaneous pain, referred pain.</td>
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<tr>
<td>Asymptomatic irreversible pulpitis</td>
<td>A clinical diagnosis based on subjective and objective findings indicating that the vital inflamed pulp is incapable of healing. Additional descriptors: no clinical symptoms but inflammation produced by caries, caries excavation, trauma.</td>
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<td>Pulp necrosis</td>
<td>A clinical diagnostic category indicating death of the dental pulp. The pulp is usually nonresponsive to pulp testing.</td>
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<tr>
<td>Previously treated</td>
<td>A clinical diagnostic category indicating that the tooth has been endodontically treated and the canals are obturated with various filling materials other than intracanal medicaments.</td>
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<tr>
<td>Previously initiated therapy</td>
<td>A clinical diagnostic category indicating that the tooth has been previously treated by partial endodontic therapy (eg, pulpotomy, pulpectomy).</td>
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**Apical tissues:**

<table>
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<tr>
<th>Normal tissues</th>
<th>Teeth with normal periradicular tissues that are not sensitive to percussion or palpation testing. The lamina dura surrounding the root is intact, and the periodontal ligament space is uniform.</th>
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<tbody>
<tr>
<td>Symptomatic apical periodontitis</td>
<td>Inflammation, usually of the apical periodontium, producing clinical symptoms including a painful response to biting and/or percussion or palpation. It might or might not be associated with an apical radiolucent area</td>
</tr>
<tr>
<td>Asymptomatic apical periodontitis</td>
<td>Inflammation and destruction of apical periodontium that is of pulpal origin, appears as an apical radiolucent area, and does not produce clinical symptoms.</td>
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<tr>
<td>Acute apical abscess</td>
<td>An inflammatory reaction to pulpal infection and necrosis characterized by rapid onset, spontaneous pain, tenderness of the tooth to pressure, pus formation, and swelling of associated tissues.</td>
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<tr>
<td>Chronic apical abscess</td>
<td>An inflammatory reaction to pulpal infection and necrosis characterized by gradual onset, little or no discomfort, and the intermittent discharge of pus through an associated sinus tract.</td>
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<tr>
<td>Condensing osteitis</td>
<td>Diffuse radiopaque lesion representing a localized bony reaction to a low-grade inflammatory stimulus, usually seen at apex of tooth.</td>
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