Pulp and Periradicular Pathosis

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Many irritants can cause pulp diseases, they are either:

1- **Viable**  
   *(Microorganisms, mainly bacteria)*

2- **Non-Viable**  
   *(Mechanical, thermal, and chemical irritants)*
Bacterial Endotoxins may travel through dentinal tubules & cause pulp inflammation

The bacterial species that cause dental caries are: **Streptococcus mutans, Lactobacilli, and Actinomyces.**
Initially, when the pulp gets inflamed it becomes infiltrated locally by chronic inflammatory cells which are:

1- Macrophages
2- Lymphocytes
3- Plasma Cells.

Then after the exposure of the pulp, Polymorphoneuclear (PMN) leukocytes will infiltrate the area.
Inflammation and subsequent necrosis depend on several factors:

1. Virulence of bacteria.
2. Ability to release inflammatory fluids.
3. Host resistance.
5. Lymph drainage.

These factors affect the progress of the disease.
Defense mechanism of the pulp (what the pulp does to protect itself):

1. Formation of Tertiary Dentine.
2. Immune cells are protective mechanism also.

These mechanisms may slow the progression of the disease, but if the irritant has not been removed the disease will progress more and more.
The role of bacteria in pulp and periradicular diseases is well established i.e. Bacteria is number one cause of pulpal diseases.

There is a very important study that was done on gnotobiotic rats (germfree rats).

In these rats when they opened the pulp and exposed it to the oral cavity that doesn't have bacteria they noticed that the pulp healed even without sealing it, which indicates that bacteria is the number one cause of pulp diseases and the turning of reversible pulpitis to irreversible pulpitis then to necrotic pulp.
They come usually from restorative procedures. The more dentinal tubules get closer to the pulp the more they increase in number and diameter so that these following procedures may cause irritation to the pulp and cause damage to odontoblasts and affect the blood supply:

1- Deep cavity preparation.
2- Removal of tooth structure without proper cooling.
3- Impact trauma.
4- Occlusal trauma.
5- Deep periodontal curettage
6- Orthodontic movement.
Also periapical tissues can be damaged as a result of:

1. Impact trauma.
2. Hyperocclusion (having a restoration with high spot, like amalgam)
3. Endodontic procedures and accidents (like working beyond the working length)
4. Perforation of the root.
5. Overextension of the filling materials.
They represent the material that we use such as:

1- Components of the filling materials.
2- Dentin cleansing, sterilizing and desensitizing agent *(we don't use them anymore because they are toxic and don't have a big effect).*
3- Intracanal medicament, irrigants, plus some components of obturation materials.

These all may cause irritation to periradicular and pulpal tissues.
In pulp disease, odontoblasts will die and will cause subsequent inflammation.

The degree of inflammation is proportional to the intensity and severity of the tissue damage.

The Severity of tissue damage ranges from reversible pulpitis to irreversible pulpitis to necrosis, and this depends on the severity, duration of the irritant and the host capacity to respond.
Whenever there is irritation of the pulp, multiple biological systems will be activated, which are:

1. **Nonspecific inflammatory mediators** that are available in any connective tissue e.g. histamine, bradykinin and arachidonic acid metabolites.

2. **PMN lysosomal granule products** (elastase, cathepsin G and lactoferrine).

3. **Protease inhibitors** such as antitrypsin.

4. **Neuropeptides** have a long inflammatory reaction in the pulp such as Calcitonin Gene-Related Peptide (CGRP) and Substance P (SP).
1- It is located in low-compliance environment.
2- Lack of collateral circulation, whenever there are collapsed venules there won't be new venules to take the edema outside the pulp.

However, the response to inflammation will be in a way similar to other connective tissue.
Mast cells are not present in healthy pulp but they are present in inflamed pulp. They produce histamine which has a significant role in pulpal inflammation.

Pulpal nerves are protective in nature and may be involved in the recruitment of inflammatory cells to the injured pulp.
Healthy pulps contain T-lymphocytes with very minimal number of B-cells that have protective mechanisms.

However, their immune reaction in the pulp can result in the formation of small necrotic foci and eventually total pulp necrosis.
What happens when there is inflammation in the pulp?
There will be increase in capillary pressure and subsequent elevation of capillary permeability which will lead to edema since there is no enough space for the pulp to expand which will affect the vessels, and if there is no enough drainage from veins and lymphatic system this will cause collapse and ischemia.
This will cause pain, in addition to inflammatory mediators that will cause \textbf{lowering of pain threshold} and the sensation of pain as a result.

In this situation where there are collapsed vessels plus bacteria (number one cause of pulp necrosis, this will lead to progression of the lesion with the persistence of the irritant in these stages:

1- Reversible pulpitis.
2- Irreversible pulpitis.
3- Hyperplastic pulpitis.
4- Pulp necrosis.
A) Pulpal

B) Periapical
A - Pulpal Diagnoses

1- Normal Pulp
2- Reversible Pulpitis
3- Irreversible Pulpitis
   A- Symptomatic
   B- Asymptomatic
4- Pulp Necrosis
5- Previously Treated
6- Previously Initiated Therapy

B - Apical Diagnoses

1- Normal Apical Tissues
2- Apical Periodontitis
   A- Symptomatic
   B- Asymptomatic
3- Chronic Apical Abscess
4- Acute Apical Abscess
5- Condensing Osteitis
1- Normal Pulp

- The pulp is symptom-free & normally responsive to pulp testing.

- Mild or transient response to thermal cold testing, lasting no more than one to two seconds after the stimulus is removed.
* **1- Normal Pulp**

* It is important to compare the tooth in question with adjacent and contralateral teeth.

* It is best to test the adjacent teeth and contralateral teeth first so that the patient is familiar with the experience of a normal response to cold.
Diagnostic Terminology
A) Pulpal

* 2- Reversible Pulpitis

* Stimulus (cold or sweet) \(\rightarrow\) Discomfort

Discomfort goes away within 2 seconds following the removal of stimulus

* Causes
* 1- Exposed dentine
* 2- Caries
* 3- Deep restoration
2- **Reversible Pulpitis**

- Periapically, no significant radiographic changes
- No spontaneous pain

Following the management of the etiology (e.g. caries removal plus restoration; covering the exposed dentin), the tooth requires further evaluation to determine whether the “reversible pulpitis” has returned to a normal status.
3- Irrversible Pulpitis

Two Types:

1) Symptomatic
2) Asymptomatic
Symptomatic Irrversible Pulpitis

1) Sharp pain upon thermal stimulus, lingering pain (30 seconds or longer after stimulus removal)
2) Spontaneity (unprovoked pain)
3) Referred pain
4) Sometimes ... pain with lying down
5) Analgesics are not effective

Difficult to diagnose because inflammation has not yet reached the periapical tissues >> no pain to percussion
Asymptomatic Irreversible Pulpitis

1) The vital inflamed pulp is incapable of healing and RCT is indicated
2) No clinical symptoms
3) Responds normally to thermal testing
4) Trauma or deep caries that would result in exposure following caries removal

Treatment >>> RCT.
Diagnostic Terminology

A) Pulpal

4- Pulp Necrosis

1) The pulp is non-responsive to pulp testing & is asymptomatic

2) Pulp necrosis per se does not cause apical periodontitis (pain to percussion or radiographic evidence of osseous breakdown) unless the canal is INFECTED
5) **Previously Treated**

1) The tooth has been endodontically treated & the canals are obturated with various filling materials other than intracanal medicaments

2) The tooth does not respond to thermal or electrical pulp testing
6- Previously Initiated Therapy

1) Tooth is previously treated by partial endodontic therapy (pulpotomy or pulpectomy)

2) The tooth **may** or **may not** respond to pulp testing modalities
Diagnostic Terminology

B) Periapical

1) Normal Apical Tissues

1) Not sensitive to percussion or palpation testing

2) Radiographically, the lamina dura is intact & the PDL space is uniform

3) As with pulp testing, comparative testing for percussion and palpation should always begin with normal teeth as a baseline for the patient.
Apical Periodontitis

Two Types:

* A) Symptomatic
* B) Asymptomatic
A) Symptomatic Apical Periodontitis

1) Represents inflammation of the periodontium
2) Painful response to biting and/or percussion
3) **May** Or **MAY NOT** be accompanied by radiographic changes (depending upon the stage of disease)
B) Asymptomatic Apical Periodontitis

1) Inflammation & destruction of the apical periodontium that is of pulpal origin
2) Appears as apical radiolucency and does not present clinical symptoms
3) No pain on percussion or pulpalation
3) **Chronic Apical Abscess**

1) Pulpal infection and necrosis
2) Gradual onset
3) little or no discomfort
4) Intermittent discharge of pus through sinus tract
5) Radiographically, signs of osseous destruction
6) Tracing gutta percha is indicated
Diagnostic Terminology

B) Periapical

4) **Acute Apical Abscess**

1) Inflammatory reaction to pulpal infection & necrosis
2) Rapid onset, spontaneous pain, extreme tenderness of the tooth to pressure, pus formation & swelling of associated tissues
3) There may be no radiographic signs of destruction
4) The patient often experiences fever and lymphadenopathy
5) **Condensing Ostetis**

A diffuse radiopaque lesion with localized bony reaction to low-grade inflammatory stimulus usually seen at the apex of the tooth.
Tooth No. 46 (lower right 6)

1) Hypersensitive to cold & sweet over the past few months
2) Symptoms have subsided
3) Now, no response to thermal testing,
   Pain on biting & percussion
4) Radiographically, diffuse radiopacity
   around the tooth apices

Diagnosis ????
**Examples**

**Diagnosis is**

Pulp Necrosis, Symptomatic apical periodontitis with condensing osteitis

**Treatment:**

Nonsurgical RCT
Build up & Crown

Over time The condensing osteitis will regress partially or totally
Tooth No. 42 (Lower right 2)

1) Apical radiolucency during routine examination
2) History of trauma more than 10 years ago
3) Tooth was slightly discoloured
4) It is not responding to cold test or EPT
5) No tenderness to percussion or palpation in the region

Diagnosis ????
**Example 2**

* **Diagnosis is:**
  Pulp Necrosis, Asymptomatic Apical Periodontitis

**Treatment**

Non-surgical RCT followed by bleaching & permanent restoration
Tooth No. 26 (Maxillary Left 1st Molar)

1) Occluso-mesial Caries
2) Sensitivity to sweets & cold liquids
3) No discomfort to biting or percussion
4) The tooth is hyper-responsive to Endo-Ice with no lingering pain

Diagnosis ????
Diagnosis is:
- Reversible Pulpitis, Normal Apical Tissues

Treatment:
- Excavation of the caries followed by placement of a permanent restoration.
- If the pulp is exposed, treatment would be non-surgical endodontic treatment followed by a permanent restoration such as a crown.
THANK YOU