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Pulpal Disease

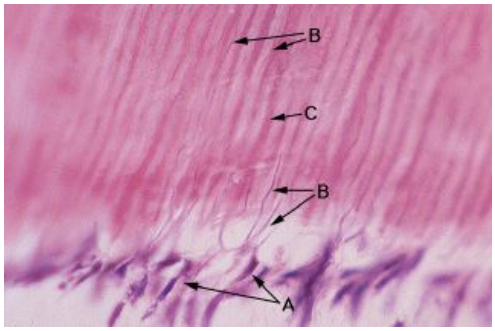
Objective – Describe the consequences of pulpal inflammation and pulp necrosis

DENTINAL CARIES

The dental pulp, dentine and peri-radicular tissues are all embryologically derived from the same tissues, are histologically intimately related and functionally respond in unison. Hence, it is referred to as the **Dentino-pulp-PDL complex**.

The dental pulp, with its odontoblasts, and other cells of a less specialised nature, is continuous with the tissue of the periapical area and that of the bone marrow spaces. In addition, both pulp and periapical tissues communicate with the rest of the body via the vascular system.

Pulp-Dentine Interface:



- A – Odontoblasts
- B – Odontoblastic Processes
- C – Predentine

Although pulp and periapical tissue are different histologically, both consist essentially of vascular connective tissue. It is therefore not surprising that the responses of these tissues to injurious agents fall almost entirely into the broad area of inflammation. The fact that pulp and periapical tissue form a continuum means that almost inevitably involvement of one tissue in a disease process will be followed by involvement of the other. Most irritants affecting pulp and periapical tissues arrive via the hard tissues of the crown of the tooth so that changes in the pulp occur first and are followed by changes in the periapical tissue.

Irritants affecting the pulp:
Carious Lesion
Removal/restoration of carious lesion
Damage from tooth cracks/fractures (trauma/occlusal forces)

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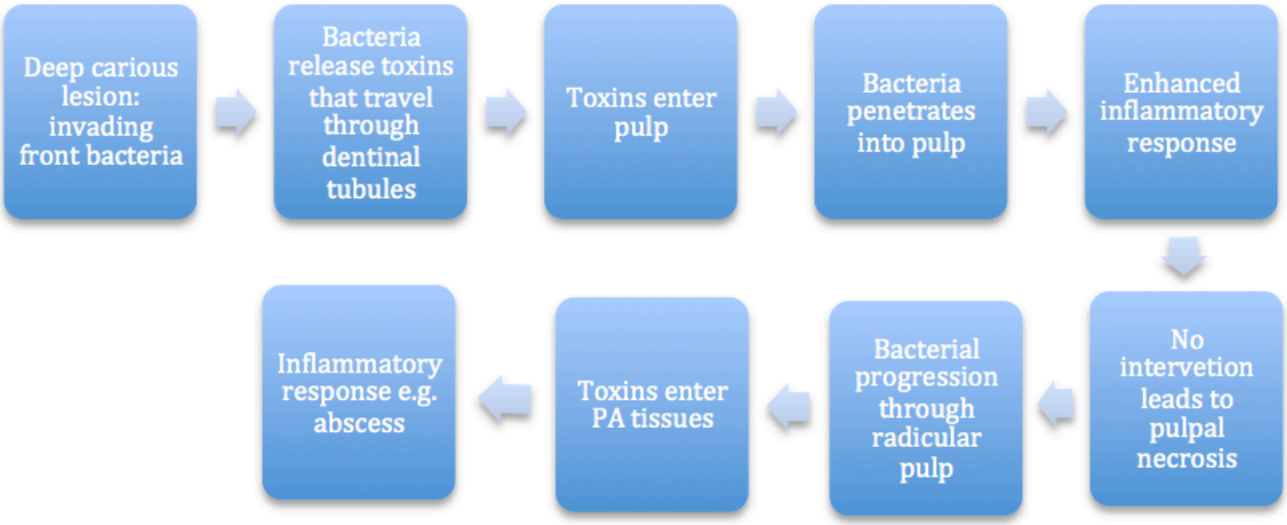
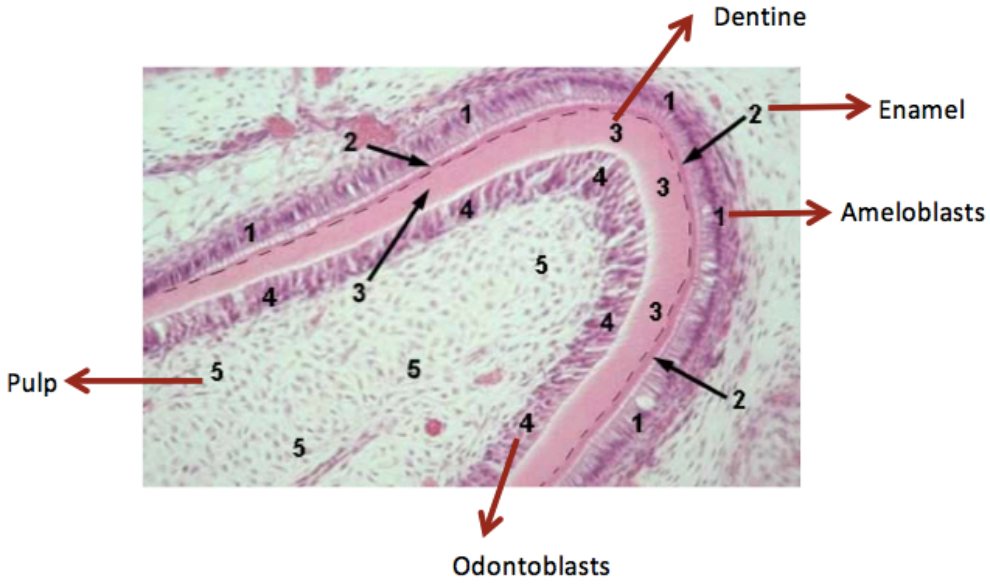
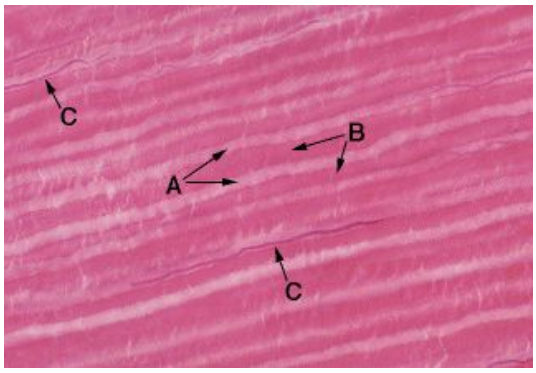


Normal Tooth Tissue:



Dentinal Tubules:



- A – Dentinal Tubules
- B – Intertubular Dentine
- C – Odontoblastic Processes

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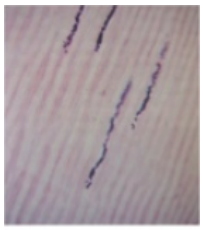
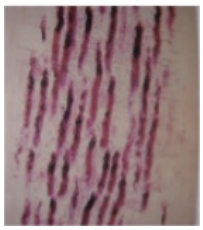
Histological Features of Dental Caries:

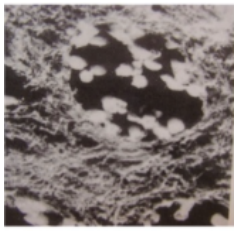


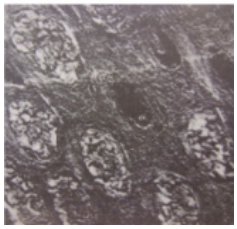
Although the protective nature of the pulp attempts to prevent the spread of the caries process, dentine is still destroyed.

Therefore, behind the area of sclerotic dentine, there is a narrowing zone of decalcification just ahead fo the bacterial invasion of the dentinal tubules.



Only a few tubules are invaded.





In advanced carious lesions:

Dentinal tubule confluence results from continued decalcification of the tubules, even though the organic matrix structure may still be maintined for some time.

This confluence occurs because of packing of the tubules with invading bacteria.

Liquefactive foci and **transverse clefts.**

Dentinal tubule coalescence and loss of minerals in the regions where bacteria is present in the tubules leads to formation of **Liquefaction Foci**. These are ovoid areas of destruction of tubules parallel to the course of the tubules. It is packed with necrotic debris from the tubule destruction.

Liquefactive foci

See the following link for more histology slides:

http://www.uky.edu/~brmacp/oralhist/module5/lab/oh5main.htm

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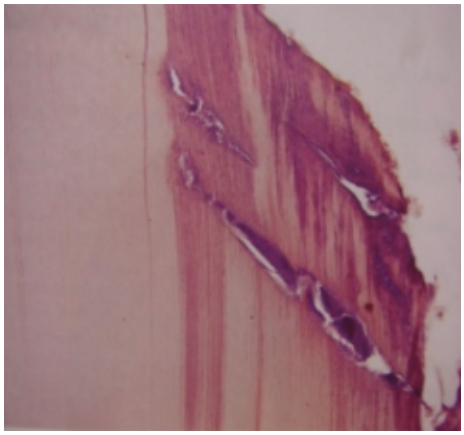

Continued dentinal destruction and proteolysis occurs at several focal areas which ultimately coalesce forming a necrotic leathery mass of dentine.

In this mass, **transverse clefts** are formed. These clefts occur perpendicular to the tubules and parallel to the course of lateral branches of tubules or along the collagen fibres of organic matrix.

It is because of these clefts that carious dentine can be removed in thin layers by hand instruments.

Transverse Clefts

Using the information above, try and identify the histological features of Dental Caries in the slides below:

Figure 1:

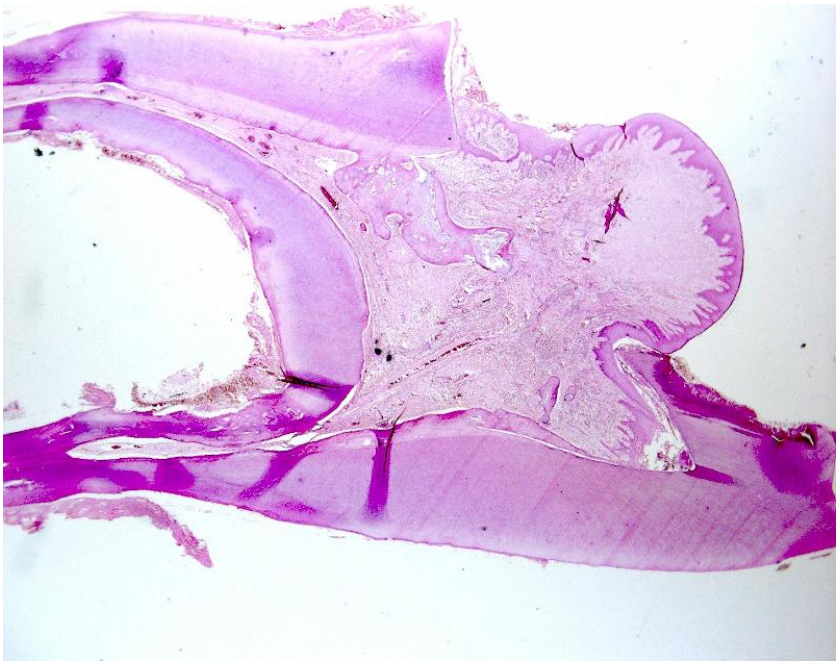

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Figure 2:

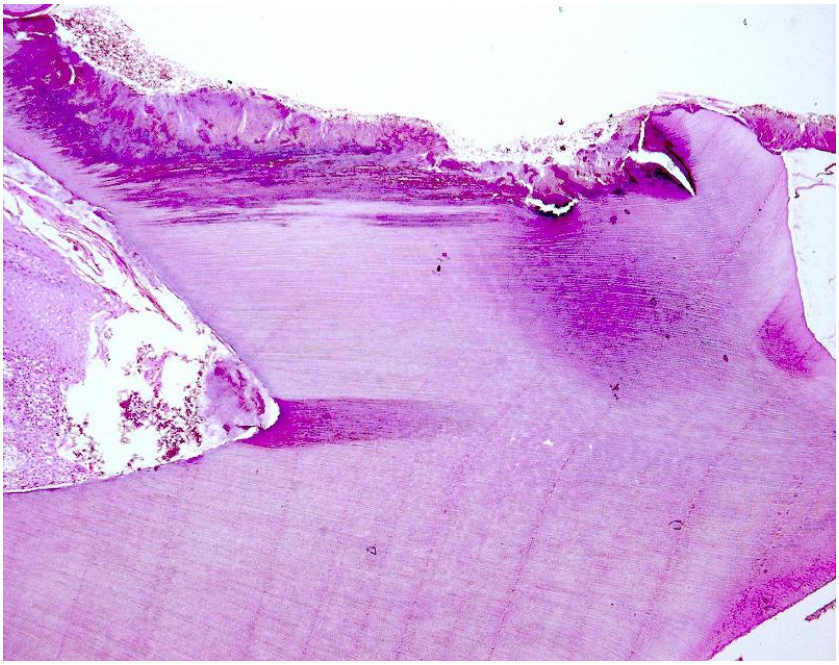
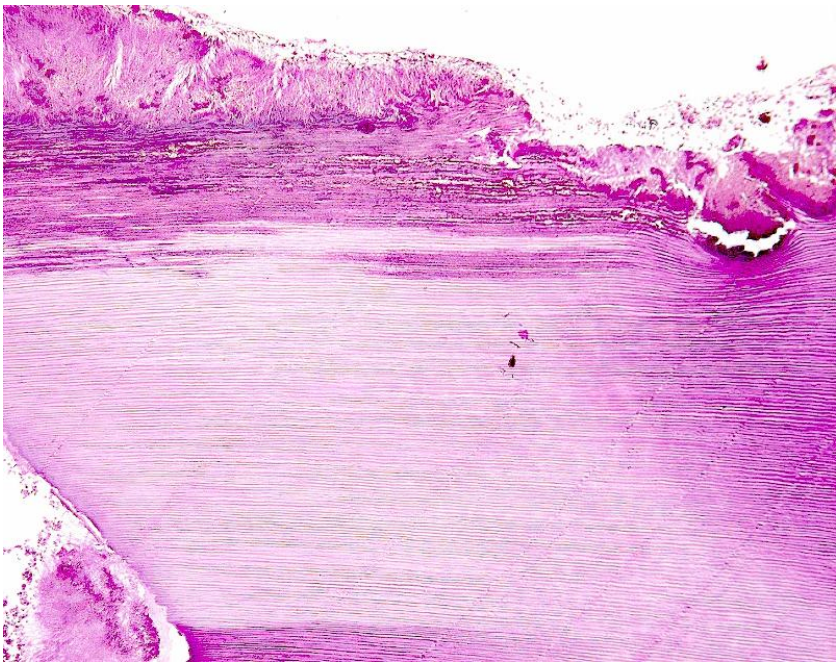


Figure 3:



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THE PULPAL RESPONSE TO A CARIOUS LESION:

If the carious lesion increases in size and extends further into the tooth, the pulp could be exposed to the oral environment.

The slowly advancing carious lesion usually evokes a response from the odontoblasts, namely, the formation of a calcio-traumatic line, loss of the odontoblast process and the laying down of reparative dentine.
As irritants from the carious lesion increase in intensity, odontoblasts degenerate and die and the dentine laid down becomes irregular (reparative dentine).

At about the time when degeneration of odontoblasts is occurring, there is a gradual infiltration of the pulp with chronic inflammatory cells, especially lymphocytes. This latter occurrence can be regarded as early chronic inflammation occurring in the pulp and is the starting point for its progression.

Pulpal protective responses:
Tertiary Dentine - stimulated by mediators in remaining dentine activated by pulpal insult
Sclerotic Dentine - narrowing of tubules to protect against an insult

THE PULPAL RESPONSE TO AN ADVANCED CARIOUS LESION

As the carious lesion advances deeper into the dentine, there may be an increasing build-up of chronic inflammatory cells and degeneration and death of odontoblasts.

Because of destruction and loss of tooth tissue, the pulp sometimes becomes "exposed" to the oral cavity before it has become grossly infected and totally destroyed by an acute inflammatory reaction which proceeds through to pus formation (acute abscess formation).

The changes of acute inflammation may be initiated by a wide variety of irritants and, in the earlier stages may be independent of the causative agent. However, the irritant itself has a profound bearing on the progress of acute inflammation.

For example, if an irritant is short acting and does not cause any irreversible tissue damage, then the affected tissue will return to normal. In the pulp, **reversible pulpitis** may occur because of thermal or mechanical shock (for example). With a severe insult and/or infection widespread inflammation of the pulp develops which may cause pulpal necrosis which rapidly involves the whole pulp. Inflammation of the pulp may spread to involve the periapical aspect of the periodontal ligament (apical periodontitis). AP may be acute and lead on to an acute periapical abscess.

If the micro-organisms win the battles against the pulpal and periapical defences and invade the periapical tissue, the periapical tissues will then contain miro-organisms, resulting in the development of an abscess.

Clinically, this abscess is described as Suppurative Apical Periodontitis.
Abscess is a histological term for the swollen area within the body tissue which contains an accumulation of pus. The pus is due to the microorganism pathogens and the defending neutrophils. The pathogens produce toxins that kill the neutrophils. As neutrophils attack the pathogens, lysosomal enzymes are secreted and these digest dead and live cells. Many of the neutrophils die in this battle.

When the periapical lesion progresses from a localised inflammatory response to a more extensive inflammatory defence response with blood vessels and connective tissues, a periapical granuloma results.

CASE – ACUTE PULPITIS WITH PULP ABSCESS FORMATION

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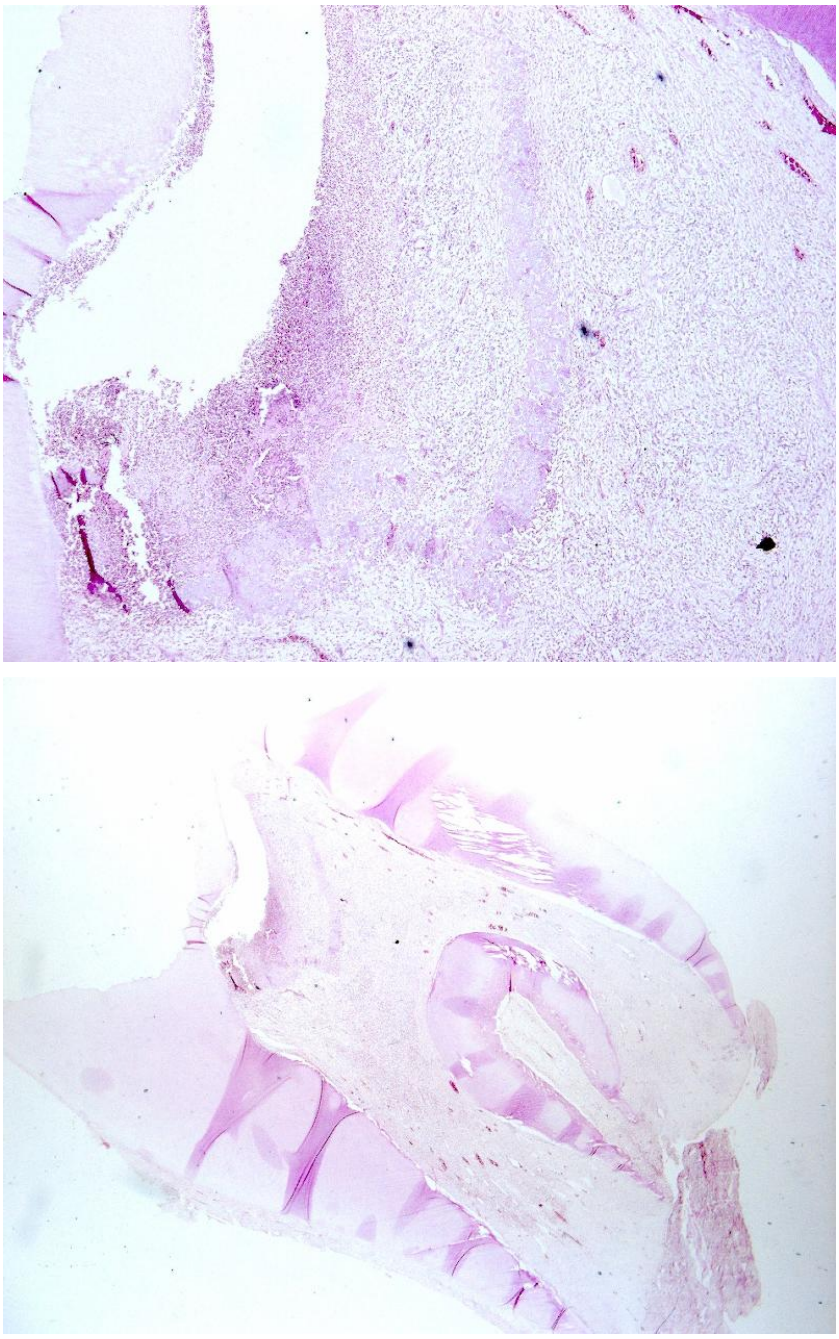

Infection has occurred, followed by acute inflammation which has become the dominant reaction and has progressed to the formation of an acute abscess. There is a dense accumulation of neutrophils, some of which are degenerate, which is the histological appearance of pus. Some of this accumulation has been lost causing artefactual spaces.

Vessels in the remainder of the pulp show marked dilatation and neutrophils are present in much of the tissue, which includes many chronic inflammatory cells present from the already existing chronic inflammatory state. This condition will progress rapidly to total pulpal necrosis.

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PERIAPICAL GRANULOMA: Acutely inflamed periapical granuloma

Periapical Granulomas, are the histological manifestation of apical periodontitis, which dentists and dental students are more familiar with.

Periapical disease is initially due to intra-canal bacteria that cause a periapical response. In the first instance, the periapical tissues do not contain microorganisms. This periapical lesion then progresses from a localized inflammatory response to a more substantial inflammatory defence response with blood vessels and connective tissues called a periapical granuloma.

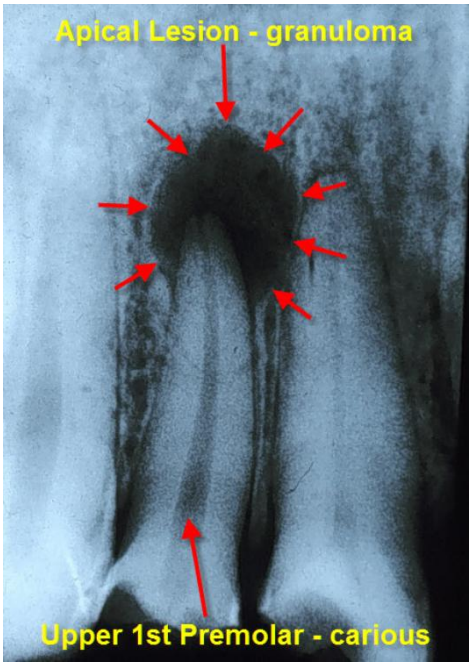
The periapical granuloma may become infected with pyogenic organisms. The resulting acute inflammation will often progress to the formation of an acute abscess in the granuloma. In the absence of treatment, this will progress further to form a dentoalveolar abscess which will usually "point" (burst) and drain into the mouth or (less commonly) on to the face. Other complications may ensue (e.g. spread of infection down the neck).

Symptoms:

- Intermittent pain over a long period of time
- Can have a history of pulpitis or be asymptomatic

Signs:

- Lesion is tender to percussion
- Pulp may or may not respond to pulp test.
- May have dental caries/large restorations
- Evidence of trauma



Radiographic features:

- As the soft tissue mass grows, bone is resorbed so that the granuloma appears as a radiolucency on a radiograph as, also, does a radicular cyst. It is not possible to distinguish between a granuloma and a radicular cyst radiographically

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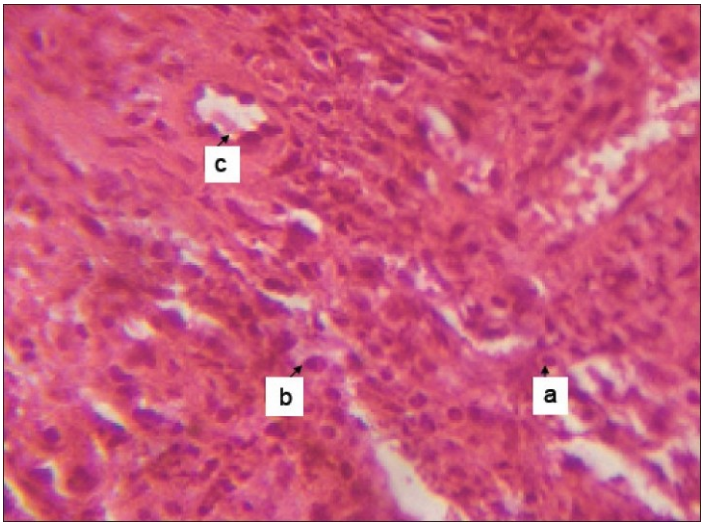
Histological features:

- When sectioned and examined histologically, periapical granulomas display a wide variety of appearances which are the result of the nature and intensity of these irritants and the particular host's response to them. In most instances, the tissue consists of fibrous connective tissue with chronic inflammatory cells and a variable number of small blood vessels. In some, endothelial cell proliferation and plump fibroblasts can be seen as granulation tissue proliferates.
- Cells found in the fibrous connective tissue:
    - Plasma cells
    - Lymphocytes
    - Foamy macrophages.



a)Lymphocytes **b)**Plasma cells **c)** Blood vessels

Investigations and treatment:

- Periapical radiographs are recommended to search for periapical radiolucencies. However, it the lesion is non healing a biopsy may be indicated to determine if it is a cyst.
- Treatment involves treating the cause:
    - Pulpitis?Root canal treatment
    - Mechanical/physical trauma
    - Chemical irritant such as a restorative material? Replace it.
    - Previous root canal treatment? Re-do root canal treatment
    - mechanical irritation such as occlusal trauma? Adjust occlusion

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Epithelial Proliferation and acute inflammation in the granuloma

Epithelial proliferation is common in dental granulomas which is concerning as they can then form a radicular cyst. Furthermore, epithelial proliferation can be initiated by root canal treatments especially instrumentation and obturation beyond the apex. This is because when gutta percha for example is pushed in the periapical space epithelium tends to grow around it. It should also be noted that epithelium proliferation is less likely to occur when obturation and instrumentation is confined to canal.

Clinical manifestations:

- May be associated with previous root canal treatment.

Radiographic features: apart from the presence of a granuloma, there isn't an indication of epithelium proliferation

Histological features:

- Stratified squamous epithelium
- macrophages
- Lymphocytes
- inflammatory cells
- collagen fibres around lesion

RC: root canal **Triangles A&B:** depict several small cysts surrounded by epithelium

Investigations and treatment:

- Biopsy is required to confirm proliferation
- Generally most of the epithelium degenerates, which may heal without surgical intervention
- Some remain in the form of cysts.
- Surgical intervention if cysts don't heal

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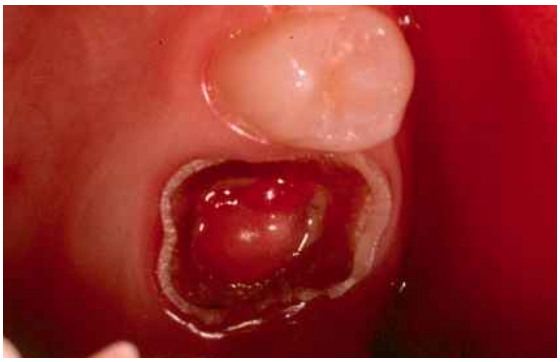
CHRONIC HYPERPLASTIC PULPITIS

It is a form of irreversible pulpitis/chronic pulpitis which is also known as a pulp polyp. It occurs in molar teeth with open apices (both deciduous and permanent) when the tooth has had a large occlusal carious lesion which has eventually opened into the roof of the pulp chamber. Instead of becoming necrotic, the pulp becomes hyperplastic, producing a mass of granulation tissue which protrudes through the pulp chamber.

"This occurs in response to the carious pulp exposure, which expands slowly, and finally results in a large open cavity. The chronic inflammatory tissue within the pulp chamber may at times proliferate through the opening and extend beyond the boundary of the tooth in form of a polyp.

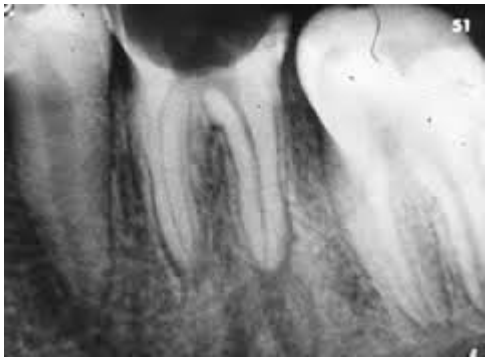


Clinical manifestations:

- Pink fleshy mass
- May cover most of the clinical crown of the involved tooth.
- A unique characteristic is the rate with which it becomes epithelialised
- May respond positively to thermal and electrical pulp tests

Histological findings:

- Pulp polyp proliferating through a large carious exposure.
- Prior to the lesion expanding, its surface layer contains a mass of necrotic cells and leukocytes, below that is inflammatory tissue of differing sizes.
- As the size increases, it can either retains its surface layer of necrotic connective tissue or have a cap of stratified squamous epithelium.
- The tissue in the pulp chamber often changes into granulation tissue, which rises from the pulp into the carious lesion.
- There may be fibrosis and calcific degeneration in the pulp chamber



Radiographic findings:

- large, open cavity which directly accesses the pulp chamber.
- If there is an increased amount of pulpal involvement or the lesion has been active for a long period a periapical radiography can reveal an incipient chronic apical periodontitis.

Investigations and treatment:

- It can be easy to identify it clinically but radiographic evidence may be required
- Extraction of tooth
- Root canal treatment if the intra-radicular floor is intact

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MCQs:

1) Why does the pulp react before periapical tissues?
    a) Pulp has a greater number of nociceptors and therefore reacts at a higher speed that the periapical tissue
    b) Periapical tissue is resistant to bacterial invasion
    c) Most insult comes from the crown
    d) The pulp has a more extensive vascular system and thereby an inflammation can occur quicker
    e) Most insults come from the root

2) Liquefication foci are found in?
    a) Candidiasis
    b) Leukoplakia
    c) Dental caries
    d) Lichen planus
    e) Metallic restorations

3) What mechanism is responsible for formation of liquefaction foci?
    a) Focal coalescence and break down of enamel lamellae
    b) Focal coalescence and break down of enamel tufts
    c) Generalised breakdown of dentinal tubules
    d) Focal coalescence and break down of dentinal tubules
    e) Generalised breakdown of apical cementum

4) Dentino-pulp-PDL complex. Why is referred to as that?
    a) Due to their close proximity
    b) Irritants tend to affect all three components
    c) They are all embryologically derived from the same tissues; mesenchymal tissue
    d) They are all embryologically derived from the same tissues; ectodermal tissue
    e) That is the order which irritant attack the tooth tissue

5) What is the order of the caries invasion process?
    a) Bacterial invading front, toxins released and travel down the dentinal tubules, toxins enter pulp, Bacteria enters pulp, inflammation, pulp necrosis, Bacterial enter radicular pulp, toxins enter PA tissue, Inflammation
    b) Bacterial invading front, toxins released and travel down the dentinal tubules, Bacteria enters pulp, toxins enter pulp, inflammation, pulp necrosis, Bacterial enter radicular pulp, toxins enter PA tissue, Inflammation
    c) Bacterial invading front, toxins released and travel down the dentinal tubules, toxins enter pulp, Bacteria enters pulp, inflammation, tertiary dentine formation, Bacterial enter radicular pulp, toxins enter PA tissue, Inflammation

d) Bacterial invading front, bacteria travel down the dentinal tubules, toxins enter pulp, Bacteria enters pulp, inflammation, pulp necrosis, Bacterial enter radicular pulp, toxins enter PA tissue, Inflammation
e) Bacterial invading front, toxins released and travel down the dentinal tubules, toxins enter pulp, Bacteria enters PA tissue, inflammation, pulp necrosis

6) You take a radiograph and biopsy of what you expect is a periapical granuloma. What do you expect to see?
   a) periapical radiolucency, fibrous connective tissue containing: plasma, cells, macrophages and keratinocytes
   b) periapical radiolucency, elastic connective tissue containing: plasma, cells, macrophages and keratinocytes
   c) periapical radiopacity, fibrous connective tissue containing: plasma, cells, macrophages and keratinocytes
   d) periapical radiopacity, elastic connective tissue containing: plasma, cells, macrophages and lymphocytes
   e) periapical radiolucency, fibrous connective tissue containing: plasma, cells, macrophages and lymphocytes

7) Signs and symptoms of a periapical granuloma may include:
   a) TTP, positive response to sensibility testing and intermittent pain
   b) Short sharp pain, previous caries incidence, TTP
   c) TTP, large restoration, short sharp pain
   d) No response to pulp testing, evidence of trauma, TTP
   e) A &D

8) Which is true? Epithelial proliferation in periapical granulomas:
   a) Generally require surgical intervention
   b) Will always turn into cysts
   c) Can be diagnosed radiographically
   d) Require a biopsy to confirm
   e) If it is associated with a previous RCT, treatment must be re-done

9) Histological findings which indicate epithelial proliferation include:
    a) Stratified squamous epithelium, Merkel cells, and lymphocytes
    b) Stratified squamous epithelium, Merkel cells, and lymphocytes and macrophages
    c) Stratified squamous epithelium,oligodendrocytes, and lymphocytes and inflammatory cells
    d) Stratified squamous epithelium, inflammatory cells, and lymphocytes and macrophages
    e) Stratified squamous epithelium, Merkel cells, and lymphocytes and oligodendrocytes

10) Clinical manifestations of chronic hyperplastic pulpitis includes:
    a) Clinically diagnosable pink mass
    b) Lesion covers most of clinical crown
    c) a unique characteristic is the rate it becomes epithelialised
    d) Responds to pulp testing
    e) All the above

References:

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