



## REVIEW ARTICLE

# Disease of Pulp and Periradicular Tissue: An Overview

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### Abstract:

Dental pain is the most common reason due to which patient seek dental treatment. Pain occur due to diseases involving pulp and periradicular tissues, as these tissues are richly innervated and have ample of blood supply. Also it is enclosed by surrounding tissues that are incapable of expanding, such as dentin and also has terminal blood flow and small-gauge circulatory access the periapex. All of these characteristics severely constrain the defensive capacity of the pulp tissue when faced with the different aggressions it may be subjected to. In addition to above mentioned characteristics, pulp tissue can also be affected by a retrograde infection, arising from the secondary canaliculi, from the periodontal ligament or from the apex during the course of periodontitis. this review article basically concentrates on structure of pulp, classification of diseases related to pulp and periradicular tissue and detailed explanation of diseases.

**Keywords:** Abscess, Periodontitis, Pulp, Pulpitis

## 1 | INTRODUCTION

**P**ulp has been described as a highly resistant organ and sometime as an organ with little or no resistance as its resistance depends on cellular activity, nutritional supply, age and other metabolic and physiologic parameters. (1) The dental pulp consists of vascular connective tissue contained within rigid dentin walls. (2) It is the principal source of pain within the mouth and also the major site of attention in endodontics and restorative treatment. (3)

**Supplementary information** The online version of this article (<https://doi.org/10.15520/jcmro.v3i10.351>) contains supplementary material, which is available to authorized users.

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Stimuli----> deposition of secondary dentin ---->  
reducing size of pulp chamber and volume----->  
reduces the cellular, vascular and neural content of  
the pulp results in atrophy (3)

## 2 | DISCUSSION

The first and foremost reaction of pulp tissue to irritation is “Inflammation”, but the basic disease process that is involved in pulp and periapical disease is “Infection”. Infection can start from pulp and spread to periodontal tissue and vice versa is also possible. (4)

*Causes of Pulp Disease (5, 6)*

- Physical

### a) Mechanical

#### 1. Trauma

A. Accidental (contact sports) – traumatic injury to pulp due to violent blow to the tooth during a fight, sports or household accidents. Habits such as opening booby pins with the teeth and nail biting may also cause pulp injury.

B. Iatrogenic dental procedures such as

- during excavation of carious tooth structure, there might be accidental exposure of the pulp
- Rapid movement of teeth by means of mechanical separator and during orthodontic treatment
- The use of pins for mechanical retention of amalgam.

2. Pathologic wear – attrition , abrasion etc

3. Crack tooth syndrome

4. Barodontalgia

Crack Tooth Syndrome

- Incomplete fractures through the body of tooth.
- Symptom – pain ranging from mild to excruciating, at the initiation or release of biting pressure

- Diagnostic method by – Tooth may disclose an enamel crack which may be better visualized by using a dye or transilluminating the tooth with fibre optic light
- Treatment-a full crown restoration immobilising the fragments.

### Barodontalgia

Toothache occurring at low atmospheric pressure. It occurs in high altitude. For example, in chronic disease no symptoms occur at ground level but at high altitude pain occurs due to low atmospheric pressure.

#### b) Thermal

- Heat from cavity preparation , at either low or high speed
- If there are deep fillings without protective base there will be conduction of heat.
- Polishing of restoration will lead to production of frictional Heat

#### c) Chemical cause-

- Phosphoric acid and acrylic monomer etc.
- Erosion by acids

#### d) Infection caused by bacteria and their by products enter pulp through-

- Caries associated toxins.
- Direct invasion of the pulp from caries or trauma
- Microbial colonization in pulp by blood borne micro organisms (anachoresis)
- Entry of bacteria through developmental groove

**TABLE 1:** Fibers associated with dental pain

A DELTA FIBERS		C FIBRES
2-5um	Diameter	0.3-1.2um
5-30m/s	Conduction velocity	0.4-2m/s
Yes, No	Myelinated	No
Subodontoblastic zones.	Location	Near the blood vessels throughout the pulp
Sharp, pricking, unpleasant, bearable	Pain characteristics	lingering, throbbing, unbearable

Pulp fibres associated with dental pain have been described in Table 1 (7)

*Classification of Diseases of Pulp*

*According to Grossman (8)*

- Inflammatory diseases of dental pulp

(a) Reversible pulpitis

1. Symptomatic (acute)
2. Asymptomatic (chronic)

(b) Irreversible pulpitis

1. Acute – 1 abnormally responsive to cold  
2 abnormally responsive to heat
2. Chronic -1 Asymptomatic with pulp exposure  
2 Hyper plastic pulpitis
- 3 Internal resorption

1. Pulp degeneration- 1.calcific 2. atrophic

- Pulp necrosis

*Classification (Ingles ) (9)*

- 1.Hyperreactive pulpalgia.

- a. Dentinal hypersensitivity.
  - b. Hyperemia.
2. Acute pulpalgia.
    - a. Incipient.
    - b. Moderate.
    - c. Advanced.
  3. Chronic pulpalgia.
  4. Hyperplastic pulpitis.

1. Necrotic pulp.
2. Internal resorption.
3. Traumatic occlusion.
4. Incomplete fracture

*Classification (Weine) (10)*

- Inflammatory changes

1. Hyperplastic (reversible pulpitis).

- a. Hypersensitive dentin.
- b. Hyperemia

1. Acute pulpalgia (acute pulpitis)
2. Chronic pulpalgia (subacute pulpitis)
3. Chronic pulpitis
4. Chronic hyperplastic pulpitis
5. Pulp necrosis

B. Retrogressive changes

1. Atrophy
2. Dystrophic calcifications

*Reversible Pulpitis (6, 10, 11)*

It is one of the earliest form of pulpitis and at one time referred to as “pulp hyperaemia”.

Symptoms

- The pulp is inflamed to the extent that thermal stimuli- usually cold- cause a quick, sharp, hypersensitive response that subsides as soon as stimulus is removed- symptomatic reversible pulpitis.
- Pain is not spontaneous. Occurs due to stimulation of A delta nerve fibres.
- If the irritant is removed by sealing the tubules, pulp will revert to asymptomatic. Or if the symptoms persist, it leads to irreversible pulpitis.
- Asymptomatic reversible pulpitis

### Histopathology

There is capillary bed engorgement with oedema — prolonged vasodilation—increased capillary pressure—increased vascular permeability—increased blood volume—increased intrapulpal pressure—pain occurs.

### Diagnosis

- Pain short duration
- Stimulus – required
- History - recent dental procedures
- Percussion test - negative
- Referred pain - negative
- On lying down pain - negative
- Color change - negative
- Radiograph - normal
- Vitality test - cold intensifies pain

### Treatment

Prevention is best treatment .Periodic care is done to prevent development of caries. Use of cavity varnish or base followed by proper filling and polishing. If pain persists even after removal of stimulus irreversible pulpitis occurs- treated by pulp extirpation.

### *Irreversible Pulpitis (6, 11)*

Is a persistent inflammatory condition of the pulp, symptomatic or asymptomatic, caused by noxious stimulus. Pain occurs spontaneously and it persists for several min to hrs and lingers on even after removal of stimulus.

#### Early symptoms

- Pain is sharp, piercing, shooting and may be intermittent or continuous which occurs due to stimulation of C- fibres.

#### In later stages

- Pain is so severe in later stages and can be described as boring, gnawing, or throbbing. Its intensity is increased by heat and sometimes relieved by cold. Patients often kept awake at night by pain.

### Histopathology

As decay reaches the pulp following changes are seen:

venules become congested causing necrosis— necrosis attract PMN's by chemotaxis— Acute inflammation—phagocytosis— PMN's die —release lysozyme —purulent exudates—microabcess is formed—pulp protects itself with fibrous connective tissue.

Microscopically, one sees the area of abscess, zone of necrotic tissue, with micro organisms present in the late carious state, along with lymphocytes, plasma cells, macrophages.

### Diagnosis

- Pain-Continuous &throbbing
- Stimulus-Not required , spontaneous pain
- History-Deep caries, trauma , extensive restoration
- Percussion test-Positive

- Referred pain-Positive
- On lying down pain-positive
- Color -Positive due to tissue lysis
- Radiograph-Widening of PDL space
- Vitality test-Heat intensifies pain cold relieves pain

#### Treatment

Complete removal of the pulp or pulpectomy and surgical removal is considered if tooth is not restorable.

#### *Chronic Hyperplastic Pulpitis (Pulp Polyp) (12–14)*

- A reddish cauliflower like growth of pulp tissue through & around a carious exposure of young pulp characterized by development of granulation tissue

#### Histopathology

Microscopically, a complex of new capillaries, proliferating fibroblasts and inflammatory cells are present in pulp polyp. The tissue in the pulp chamber is often transformed into granulation tissue, which projects from the pulp into the carious lesion.

#### Diagnosis

- If hyperplastic tissue grow beyond the cavity of tooth, it appears as if gum is growing into the cavity. Differentiate it by raising the tissue back to its origin.
- Radiograph shows large open cavity with direct access to pulp chamber.

#### Differential Diagnosis

It should be differentially diagnosed from gum polyp by raising the stalk back to its origin.

#### Treatment

Elimination of polypoid tissue, removal of pulp followed by restoration.

#### *Internal Resorption (10, 15–17)*

It is an idiopathic slow or fast progressive resorptive process occurring in the dentin of pulp chamber or root canals of tooth. It is painless condition stimulated by trauma which produces dentin destruction. Tooth is asymptomatic but on perforation of root pain occurs.

Appearance: Pink Spot

#### Histopathology

It is the result of osteoclastic activity. The resorptive process is characterized by lacunae, which may be filled in by osteoid tissue and presence of granulation tissue accounts for profuse bleeding when the pulp is removed. Multinucleated giant cells are usually seen when pulp is chronically inflamed.

#### Diagnosis

##### Radiographic features

- Radiographically appears as uniform, round or ovoid radiolucent enlargement of pulp chamber
- It does not involve the bone, if perforation occurs the bone adjacent to it is resorbed

Vitality test: occurs in teeth with vital pulps & give positive response to sensitivity tests.

#### Treatment

Pulp extirpation stops the internal resorption, after repair defect is obturated with plasticized gutta percha.

#### *Pulp Degeneration (18)*

Generally present in older people. It may be the result of persistent, mild irritation. May also be induced by attrition, abrasion, erosion, bacteria etc. It occurs in 2 forms

1. Calcific degeneration
2. Atrophic degeneration

Calcific Degeneration: It occur when part of pulp tissue is replaced by calcific material i.e pulp stones (denticles)

Pulp stones are classified as –

a) according to location - 1) free 2) embedded 3) attached

b) according to structure- 1) true 2) false

**True Denticles**

Made up of localized mass of calcified tissue that resemble dentin because of their tubular structure, more common in pulp chamber than in root canal and nodules bear great resemblance to secondary dentin.

**False Denticles**

Localised mass of calcified tissue, does not exhibit tubular structure. Nodules appear to be made up of concentric layers or lamellae deposited around a central nidus. More common in pulp chamber than in root canal.

**Free Denticles**

Denticles lying entirely within the pulp chamber and not attached to dentin walls

**Attached Denticles**

Denticles lying within the pulp chamber and is continuous with the dentin walls

Radiographically, appears as intracanal radiopacity similar to surrounding dentin. Calcific degeneration of complete pulp space occurs as a sequale to traumatic injury is known as Calcific Metamorphosis

**Atrophic Degeneration**

Decrease in size occurs slowly as tooth grows old. Fewer stellate cells are present & intracellular fluid is increased. Replacement of cellular elements with fibrous tissue is called as fibrous degeneration. (10)

**Pulp Necrosis (19)**

Necrosis is death of pulp.

**Types**

- In Coagulation necrosis soluble part of tissue is converted into solid material
- Caseation
- Liquefaction necrosis

Cause: Noxious insult or injury to pulp such as trauma, bacteria, chemical irritation.

Symptoms: Discoloration of tooth, no painful symptoms occur

**Diagnosis**

- Radiographic changes – shows large cavity & thickening of PDL.
- Vitality test – tooth is not responding to vitality test
- Visual examination: due to lack of normal translucency tooth appear opaque .
- Histopathology – inflammation is seen.

**Treatment**

- Root canal treatment or extraction in non restorable tooth.

*Diseases of Periradicular Tissue*

Pulpal diseases are one of the most common cause of periradicular diseases, as there is inter relationship between pulp and periradicular tissues through the various foramina of root canals and give rise to inflammatory and immunologic reactions through the passage of bacterial products and toxins. It is said that even pulp inflammation causes inflammatory changes in the periodontal ligament even before pulp becomes totally necrotic. (20)

*Classification*

*According to Grossman (8)*

1. Acute periradicular diseases
  - (A) Acute apical periodontitis
  - (B) Acute alveolar abscess
  - (C) Acute exacerbation of chronic apical periodontitis
2. Chronic periradicular diseases
  - (A) Chronic apical periodontitis
    - (a) Chronic alveolar abscess
    - (b) Cystic apical periodontitis
  - (B) Persistent apical periodontitis
3. Condensing osteitis

- 4. External root resorption
- 5. Diseases of periradicular tissue of non endodontic origin

*Classification (Ingles) (9)*

Periradicular lesions are divided into three main clinical groups.

- a) Symptomatic (acute) apical periodontitis (SAP)
- b) Asymptomatic (chronic) apical periodontitis (AAP)
  - i) Periapical granuloma
  - ii) periradicular cyst
  - iii) condensing osteitis
- c) Apical abscess
  - i) Symptomatic Apical Abscess
  - ii) Asymptomatic Apical Abscess

Non-Endodontic Periradicular Lesions

Odontogenic Cysts

- Dentigerous cyst
- Lateral periodontal cyst
- Odontogenic keratocyst
- Residual apical cyst

Bone Pathology: fibrous- osseous lesions

- Periradicular cemental dysplasia
- Osteoblastoma & cementoblastoma
- Cementifying & Ossifying fibroma

Odontogenic Tumor

- Ameloblastoma

Non-Odontogenic Lesions

- Central giant cell granuloma
- Nasopalatine duct cyst
- Simple bone cyst
- Globulomaxillary cyst

*Classification (Weine) (10)*

- i) Apical periodontitis
  - Acute apical periodontitis (AAP)
  - Chronic apical periodontitis( CAP)
  - Condensing osteitis
- ii) Apical abscess
  - Acute apical abscess (AAP)
  - Chronic apical abscess (CAP)
- iii) Non-endodontic periapical lesions
  - Odontogenic cyst
    - Dentigerous cyst
    - Lateral periodontal cyst
    - Odontogenic keratocyst
    - Residual apical cyst
  - Odontogenic tumor
  - Non-Odontogenic tumor .

*Routes of Micro-organism Ingress*

Through the open cavity, dentinal tubules, gingival sulcus, periodontal ligament, blood stream, broken occlusal seal or faulty restoration of a tooth previously treated by endodontic therapy and extension of a periapical infection from adjacent infected teeth. (20)

*Acute Apical Periodontitis (21, 22)*

Painful inflammation around the apex as an extension of pulpal inflammation into periapical tissue, mechanical or chemical trauma by endodontic instruments or materials or occlusal trauma caused by hyperocclusion or bruxism.

Causes

1. Abnormal occlusal contacts
2. Recently inserted restoration extending beyond the occlusal plane.
3. Traumatic blow to the tooth
4. Bacterial or noxious products from an inflamed, necrotic pulp
5. over instrumentation during cleaning and shaping root canal
6. Forcing of irritants through the apical foramen

7. Extension of obturating material through the apical foramen.

#### Symptoms

Patient complains of pain on closure and mastication and tenderness of tooth. Tooth may feel slightly sore and extruded.

#### Histopathology

- An inflammatory reaction occurs in the PDL.
- Dilatation of blood vessels, PMN's are present, and an accumulation of serous exudates distends the PDL and extrudes the tooth slightly.
- Severe irritation lead to activation of osteoclast resulting in breakdown of periradicular bone, and the acute alveolar abscess may follows

#### Diagnosis

- Tender on percussion is classical diagnostic feature. Radiographically thickening of PDL or a small area of rarefaction is present.

Treatment: Occlusal adjustment and removal of irritants is the immediate line of the treatment

#### *Acute Alveolar Abscess (20, 23, 24)*

Localised collection of the pus in the alveolar bone at the root apex of the tooth from the necrotic pulp, with extension of infection through apical foramen into periradicular tissues.

#### Causes

- Trauma or of chemical or mechanical irritation.
- The immediate cause is generally bacterial invasion of dead pulp tissues

#### Symptoms

- Tenderness is the first symptom felt which is relieved by continuous slight pressure on extruded tooth to push it back into the alveolus.
- Later severe, throbbing pain occur with attendant swelling of the overlying soft tissue.

- Osteitis, periostitis, cellulites or osteomyelitis may occur if left unattended.
- Sinus tract is formed usually on labial and buccal mucosa though which pus drainage occur.

#### Diagnosis

- Generally made quick and accurately from the clinical examination and from the subjective history give by the patient.
- A radiograph may help one to determine the tooth affected by showing a cavity, a defective restoration, thickened PDL space, or evidence of breakdown of bone.
- Affected tooth may be tender to percussion or patient may state that it hurts to chew with the tooth.
- Apical mucosa tender on palpation and tooth may be mobile and extruded.

#### Histopathology

- The marked infiltration of PMN'S and the rapid accumulation of inflammatory exudates in response to an active infection distend the PDL & there by elongate the tooth. If the process continue, periodontal fibers will separate.
- Although some mononuclear cells may be found, the chief inflammatory cells are PMN'S.
- As the bony tissue in the region of the root apex is resorbed, and as more and more of the PMN'S die in their battle with the microorganisms, pus is formed.
- Microscopically, one sees an empty space or spaces, where suppuration has occurred, surrounded by PMN'S & some mononuclear cells.

#### Treatment

- The immediate treatment consists of establishing the drainage and controlling the systemic reaction.

*Acute Exacerbation of Chronic Periodontitis (Phoenix Abscess) (25)*

An acute periapical exacerbation that arises from a previously existing chronic lesion.

Cause

1. Noxious stimuli from a diseased pulp with chronic periradicular disease.
2. While chronic periradicular diseases, Such as granulomas and cysts, are in a state of equilibrium, these apical reactions can be completely asymptomatic.
3. At times, because of an influx of necrotic products from a diseased pulp, or because of bacteria and their toxins, these apparently dormant lesions may react and may cause an acute inflammatory response.
4. Lowering of the body's defenses in the presence of bacteria and the bacteria toxins released from the root canal.
5. Root canal instrumentation leads to mechanical irritation that may also trigger an acute inflammatory response.

Symptoms

- Tender on touch during initial stages.
- As inflammation progresses, the tooth may be elevated in its socket and may become sensitive.
- The mucosa over the radicular area may be sensitive to palpation and may appear red and swollen.

Histopathology

In the granuloma or cyst and the adjacent periradicular tissues are areas of liquefaction necrosis with disintegrating polymorphonuclear neutrophils and cellular debris (pus). These areas are surrounded by infiltration of macrophages and some lymphocytes and plasma cell

Diagnosis

- The exacerbation associated with the initiation of root canal therapy in a completely asymptomatic tooth.
- In such a tooth, radiographs show well-defined periradicular lesion.
- The patient may have a history of a traumatic accident that turned the tooth dark after a period of time or of postoperative pain in a tooth that had subsided until the present episode of pain.
- Lack of response to vitality tests points to a diagnosis of necrotic pulp.

Treatment

- Establishing drainage and controlling of systemic reaction.

*Periapical Granuloma (26, 27)*

Dental granuloma is a granulomatous tissue continuous with the PDL, resulting from death of the pulp & diffusion of bacteria and bacterial product toxins from the root canal into the surrounding periradicular tissues through the apical and lateral foramen.

Inflammation in Dental Granuloma is misnomer. Because its tissue is chronic in nature & not a tumor. A granuloma contains "Granulomatous" tissue, that is granulation tissue & chronic inflammatory in composition. A granuloma may be seen as a chronic low grade defensive reaction of the alveolar bone to irritation from the root canal.

Cause

- Death of pulp followed by mild infection or irritation of the periradicular tissue.
- In some cases granuloma preceded by a chronic alveolar abscess.
- Experimental evidence has shown that a granuloma is cell mediated response to pulpal bacterial products.

Symptom

No subjective symptom, except in rare cases when it breaks down & undergoes suppuration.

Diagnosis

- Generally discovered during routine radiographic examination.
- Area of rarefaction is well defined, with lack of continuity of the lamina dura.
- Exact diagnosis can be made only by microscopic examination.
- Generally tooth non tender to percussion & it's not loose.
- Mucosa over the root may or may not tender on palpation
- A sinus tract may be present. Tooth does not respond to thermal or EPT.
- Patient may give a history of pulpalgia that sub sided

#### Histopathology

Granulomatous tissue replaces the alveolar bone and PDL.

It consists of a rich vascular net work, fibroblasts and a moderate infiltration of lymphocytes and plasma cells. Macrophages and foreign – body giant cells may also be present.

As the inflammatory reaction continues, because of irritation from bacterial or their products, the exudates accumulates at the expense of the surrounding alveolar bone,

This process is followed by clearing of the dead osseous tissue by macrophages or foreign-body giant cells while, at the periphery, fibroblasts actively build a fibrous wall.

Some granulomas consist foam cells, macrophages containing lipid material, and cholesterol. The alveolar bone may show resorption and osteoclasts may be present.

#### Treatment

- Extraction of involved tooth
- Root canal therapy
- If left untreated, may undergo transformation into an apical periodontal cyst through proliferation of epithelial rests in the area.

#### *Radicular Cyst (28, 29)*

A radicular cyst is a cyst most likely results of epithelial cells (Malassez) in the periodontal ligament are stimulated to proliferate and undergo cystic degeneration by inflammatory products from a non vital tooth. The lumen of the cyst is filled with a low – concentration of proteinaceous fluid.

#### Causes

- Physical
- Chemical
- Bacterial

#### Symptoms

- No symptoms are associated with the development of a cyst, except those incidentals to necrosis of the pulp.
- Tooth movement occur due to pressure of the cyst, owing to accumulation of cystic fluid. As a result the root apices of the involved teeth become spread apart, so the crowns are forced out of alignment.
- Tooth also become mobile.
- If left untreated, cyst continue to grow at the expense of the maxilla or the mandible.

#### Diagnosis

- Tooth with a radicular cyst does not react to electrical or thermal stimuli, and results of other clinical tests are negative, except the radiograph. The patient may report a previous history of pain.
- Usually, on radiographic examination, shows loss of continuity of the lamina dura with an area of rarefaction. The radiolucent area is generally round in outline, except where it approximates adjacent teeth, in which case it may be flattened and may have on oval shape.

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- The radiolucent area may be larger than a granuloma and may include more than one tooth.

Location 60% of cysts are found maxilla, especially around incisors & canines.

### Histopathology

Radicular cysts consist of a cavity lined with stratified squamous epithelium derived from epithelial cell rests of Malassez present in the periodontal ligament. Periradicular inflammatory changes cause the epithelium to proliferate. As the epithelium grows into a mass of cells, the center loses the source of nutrition from the peripheral tissues. These changes produce necrosis in the center; a cavity is formed, and a cyst is created.

### Treatment

- Extraction if involved tooth
- Root canal treatment

### *Condensing Osteitis/Chronic Focal Sclerosing Osteomyelitis (30)*

Condensing Osteitis is the response to a low-grade, chronic inflammation of the periradicular area as a result of a mild irritation through the root canal.

### Etiology

Alveolar bone is stimulated by osteoblastic activity from pulpal disease resulting in degeneration.

### Symptoms

- Tooth may be asymptomatic/ sensitive to stimuli.
- Vitality tests: the tooth may or may not respond

### Diagnosis

- Radiographs shown localized area of radiopacity surrounding the affected root.
- It is an area of dense bone with reduced trabecular pattern.

- The mandibular posterior teeth are most frequently affected.

### Histopathology

Microscopically, condensing osteitis appears as an area of dense bone with trabecular borders lined with osteoblasts. Chronic inflammatory cells, plasma cells, and lymphocytes are seen in the scant bone marrow.

Treatment: Root canal treatment is a treatment of choice

Prognosis: Excellent, lesion may persist after endodontic treatment

### *Asymptomatic Apical Abscess (20, 24)*

A chronic alveolar abscess is a long standing, low-grade infection of the periradicular alveolar bone. The source of the infection is in the root canal.

### Etiology

Natural sequelae of death of the pulp with extension of the infective process periapically or it may result from a pre-existing acute abscess.

### Symptoms

A tooth is generally asymptomatic, no response to pulp vitality test such an abscess is detected only during routine radiographic examination Continuously / intermittently draining sinus tract which may be seen as a stoma on the oral mucosa.

### Diagnosis

- May be painless or only mildly painful.
- At times, radiographic evidence of osseous breakdown is seen during routine examination or discoloration of the crown of the tooth.
- The radiograph often shows a diffuse area of bone rarefaction, but the radiographic appearance of the lesion is non diagnostic.
- Thickening of PDL. The rarefied area may be so diffuse as to fade indistinctly into normal bone.

### Histopathology

As the infective process extends to the periapical tissues or as toxic products diffuse through the apical

foramen. Some of the periodontal fibers at the root apex are detached or lost, followed by destruction of the apical periodontal ligament. The apical cementum may also become affected. Lymphocytes and plasma cells are generally found toward the periphery of the abscessed area, with variable numbers of PMN'S at the center. Mononuclear cells may also be present. Fibroblasts may start to form a capsule at the periphery. The root canal itself may appear to be empty or cellular debris may be present.

#### Treatment

Drainage must be established by either opening the pulp chamber or extracting the tooth. Root canal therapy may be carried out. If not treated lead to – osteomyelitis, cellulitis, bacteremia, fistulous tract formation opening on the skin.

### 3 | SUMMARY AND CONCLUSION

Pulp and periodontal tissue form basis of dental practice. Patient mainly seek dental treatment because of pain and discomfort. So every dentist should have proper knowledge of every disease related to pulp and periodontal tissue on the basis of which proper diagnosis can be made on the basis of this diagnosis proper treatment plan can be made and implemented for the well being of patients.

### REFERENCES

- Morotomi T, Washio A, Kitamura C. Current and future options for dental pulp therapy. *Jap Dent Sci Rev* 2019;55(1): 5-11
- Rudolph P. *Essentials of oral histology and embryology: a clinical approach* 3rd ed. St.Louis,MO,USA: Mosby Elsevier;2006
- Bergenholtz G, Horsted-Bindslev P, Reit C. *Textbook of endodontology*. 2nd ed. Hoboken,NJ,USA: Wiley-Blackwell;2009.
- Heyeraas KJ, Kvinnsland I. Tissue pressure and blood flow in pulpal inflammation. *Proc Finn Dent Soc* 1982;88 (Suppl 1): 393-401.
- Al Reader, Nusstein J, Hargreaves KM. *Local anesthesia in endodontics. Pathways of pulp*. 9th ed. St.Louis, Missouri: Mosby/Elsevier 2006.
- Ali SG, Mulay S. Pulpitis: A review. *IOSR J Dent Med Sci* 2015;14(8): 92-97.
- Trowbridge HO. Review of dental pain-Histology and Physiology. *J Endod* 1986;12(10): 445-52.
- Grossman LI. *Endodontic Practice*. 9th edn. Philadelphia: Lea &Febiger, 1978:51-75.
- mulsen MH, Sieraski SM. Histophysiology and diseases of the dental pulp. In: Weine FS, ed. *Endodontic therapy*. 4th edn. St.Louis: Mosby, 1989:128-150.
- Abbott PV, Yu C. A clinical classification of the status of the pulp and the root canal system. *Aus Dent J* 2007;52(1 Suppl): S17-S31.
- Dabuleanu M. Pulpitis reversible/irreversible. *J Can Dent Assoc* 2013;79: 90-94.
- Dayal PK, Subhash M, Bhat AK. Pulpoperiapical periodontitis. A radiographic study. *Endodontolgy* 1999;11:60-4.
- Neville BW, Damm D, Allen CM, Bouquot JE. *Oral and Maxillofacial Pathology*. Philadelphia: W.B. Saunders Company; 1995. p. 97-8.
- Škaljac-Staudt G, Galić N, Katunarić M, Ciglar I, Katanec D. Immunopathogenesis of chronic periapical lesions. *Acta Stomatol Croat* 2001;35:127-31.
- Patel S, Ricucci D, Durak C, Tay F. Internal root resorption: A review. *J Endod* 2010;36(7): 1107-1121.

16. Lyroudia KM, Dourou VI, Pantelidou OC, Labrianidis T, Pitas IK. Internal root resorption studied by radiography, stereomicroscope, scanning electron microscope and computerized 3D reconstructive method. *Dent Traumatol* 2002;18:148–52.
17. European Society of Endodontology. Quality guidelines for endodontic treatment: consensus report of the European Society of Endodontology. *Int Endod J* 2006;39:921–30.
18. Talbot ES. Pulp degeneration. *Jour AMA* 1904.
19. Andreasen JO, Bakland LK. Pulp regeneration after non-infected and infected necrosis, what type of tissue do we want? A review. *Dent Traumatol* 2011: 1-6.
20. Gutmann JL, Baumgartner JC, Gluskin AH, Hartwell GR, Walton RE. Identify and define all diagnostic terms for periapical/periradicular health and disease states. *J Endod* 2009;35(12): 1658-1674.
21. Graunaite I, Lodiene G, Maciulskiene V. Pathogenesis of apical periodontitis: A literature review. *J Oral Maxillofac Res* 2011;2(4)e1: 1-15.
22. Nair PN. Pathogenesis of apical periodontitis and the causes of endodontic failures. *Crit Rev Oral Biol Med*. 2004 Nov 1;15(6):348-81.
23. Johnson WT. Color atlas of endodontics. Philadelphia: WB Saunders Co; 2002:10–1
24. Morse DR, Seltzer S, Sinai I, et al. Endodontic classification. *J Am Dent Assoc* 1977;94:685–9.
25. Chaudhary M, Chaudhary SD. Essentials of pediatric oral pathology. 1st ed Jaypee Brothers Publication, New Delhi: 2011.
26. Omoregie FO, Ojo MA, Saheeb BDO, Odukoyo O. Periapical granuloma associated with extracted teeth. *Nig J Clin Prac* 2011;4(3): 293-96.
27. Peters E, Monica L. Histopathologic examination to confirm diagnosis of periapical lesions: A review. *J Can Dent Assoc* 2003;69:598-600.
28. Grover N, Tyagi KK, Kanwar M, Sharma V, Tyagi AK. Radicular cyst of permanent incisors and its management: A case report. *Annals Dent Special* 2014;2(2): 79-81.
29. Shear M. Cysts of the oral regions. 3rd ed. Boston: Wright; 1992. Pg.136-70.
30. Holly D, Jurkovic R, Mracna J. Condensing osteitis in oral region. *Bratisl Lek Listy* 2009;110(11): 713-15.

**How to cite this article:** Singh G., Paul R S., Arora A., Kumar S., Jindal L., Raina S. **Disease of Pulp and Periradicular Tissue: An Overview.** *Journal of Current Medical Research and Opinion.* 2020;652–664. <https://doi.org/10.15520/jcmro.v3i10.351>

