



REVIEW ARTICLE

CLINICAL DIAGNOSIS OF DENTAL PAIN

***Dr. Aji Markose**

Department of Conservative Dentistry, Vivekanada Dental College, Thiruchengode (Namakkal dt),
Tamilnadu - 637205

ARTICLE INFO

Article History:

Received 08th August, 2016

Received in revised form

25th September, 2016

Accepted 19th October, 2016

Published online 30th November, 2016

ABSTRACT

Dental pulp consist of vascular connective tissue contained within the rigid dentin walls. It is the principal source of pain in the oral cavity and also a major site of attention in endodontics and restorative procedures. Thus the knowledge to pulp is essential not only for providing dental treatment but also to know the rationale behind the treatment provided.

Key words:

Dental pulp, Pain, Inflammation, Pulpitis,
Dentin, Tooth fracture.

Copyright©2016, Aji Markose. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Citation: Aji Markose, 2016. "Clinical diagnosis of dental pain", *International Journal of Current Research*, 8, (11), 42447-42450.

INTRODUCTION

The dental pulp is subjected to various insults in the mouth. These may be in the form of physical, chemical or bacterial agents. The pulp responds to these irritants most often by an inflammatory response and sometimes by pulpal degeneration. The nature of the irritant and the duration of exposure to the irritant determine the reaction of the pulp. Mild irritation of a short duration may produce reversible inflammation of the pulp while long standing severe irritation may lead to irreversible changes. Once the inflammation is irreversible it invariably progresses to necrosis of the pulp.

Barodontalgia/Aerodontalgia

It is pain experienced in a recently restored tooth during low atmospheric pressure. Pain is increased either during ascent or descent. Chronic pulpitis which appears asymptomatic in normal conditions, may also manifest as pain in high altitude because of low pressure.

Reversible Pulpitis

Pain: is sharp but of brief duration ceasing when irritant is removed. It is usually caused by cold, sweet, and sour stimuli.

Visual examination and history: May reveal caries, traumatic occlusion and undetected fracture.

Radiographs:

- Show normal PDL and lamina dura
- Depth of caries or restoration may be evident

Percussion test: Shows negative response i.e tooth is normal to percussion and palpation without any mobility.

Vitality tests: Pulp responds readily to cold stimuli. Electric pulp tester, requires less current to cause pain.

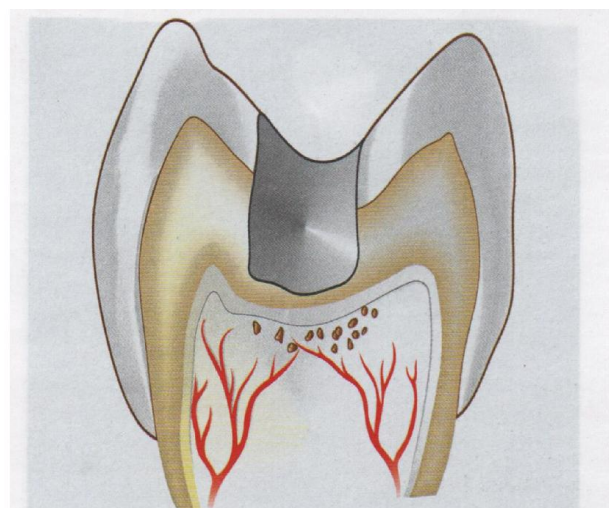


Fig. 1. Insertion of deep restoration causing pulp inflammation

*Corresponding author: Dr. Aji Markose,

Department of Conservative Dentistry, Vivekanada Dental College,
Thiruchengode (Namakkal dt), Tamilnadu - 637205

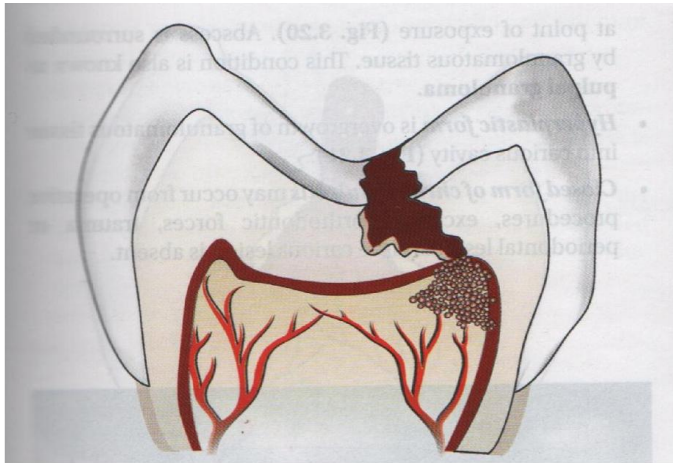


Fig. 2. Tooth decay causing pulpitis

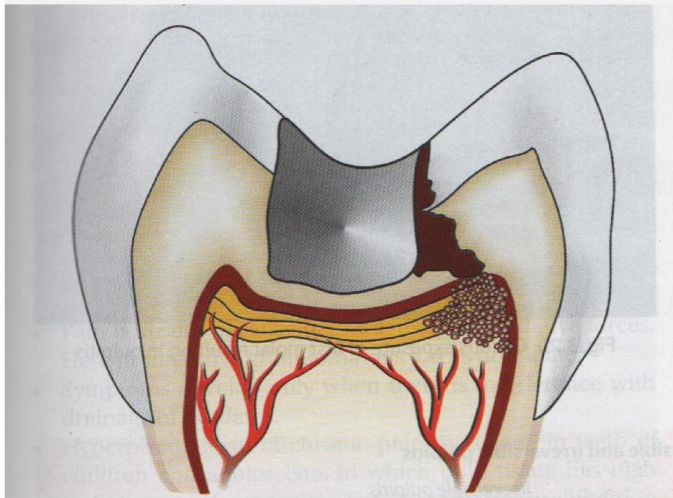


Fig. 3. Secondary caries under restoration



Fig. 4. Carious exposure of first molar resulting in pulpitis



Fig. 5. Deep caries involving the pulp in mandibular first molar. Note widening of periodontoal ligament space

Irreversible Pulpitis

Visual Examination and history: Examination of involved tooth may reveal previous symptoms. On Inspection one may see deep cavity involving pulp or secondary caries under restoration.

Radiographic findings:

- May show depth and extent of caries.
- Periapical area shows normal appearance but a slight widening may be evident in advanced stage of pulpitis.

Percussion: Tooth is tender on percussion (due to increased intrapulpal pressure as a result of exudative inflammatory tissue)

Vitality tests: Thermal test;- Hyperalgesic pulp responds more steadily to cold stimulation than for normal tooth, pain may persist even after removal of the irritant. As the pulpal inflammation progresses, heat intensifies the response because it has expansible effect on blood vessels Cold tends to relieve pain because of its contractile effect on vessels, reducing the intrapulpal pressure.

Electric test: Less current is required in initial stages. As tissue becomes more necrotic, more current is required to generate the response.

Acute Apical Periodontitis: The Signs and Symtoms are

- Tooth is tender on percussion
- Dull, throbbing and constant pain
- Pain occurs over a short period of time
- Negative or delayed vitality test
- No swelling
- Pain on biting
- Cold may relieve pain or no reaction
- Heat may exacerbate pain or no reaction
- No radiographic signs, Sometimes widening of periodontal ligament space.

Acute Apical Abscess

- In early stages, there is tenderness of tooth which is relieved by continued slight pressure on extruded tooth to push it back into alveolus.
- Later on throbbing pain develops with diffuse swelling of overlying tissues.
- Tooth becomes more painful, elongated and mobile as infection increases in later stages.

- Patient may have systemic symptoms like fever, increased WBC count.
- Spread of lesion towards a surface may take place causing erosion of cortical bone or it may diffuse and spread widely resulting in formation of cellulitis.

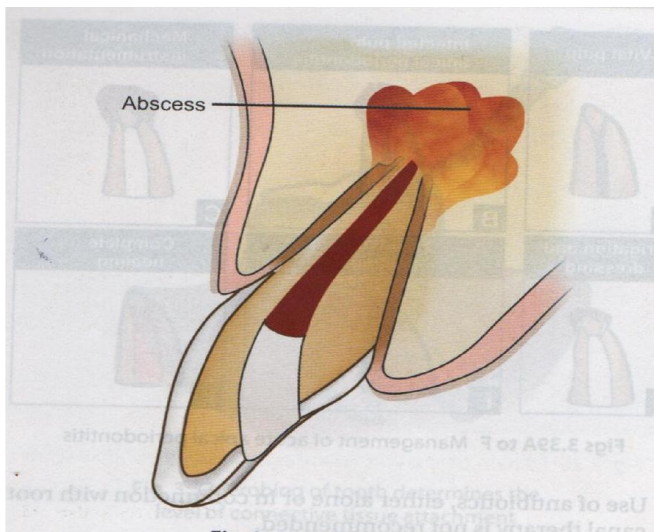


Fig. 6. Periapical abscess



Fig. 7. Radiograph showing periapical abscess in relation to 21

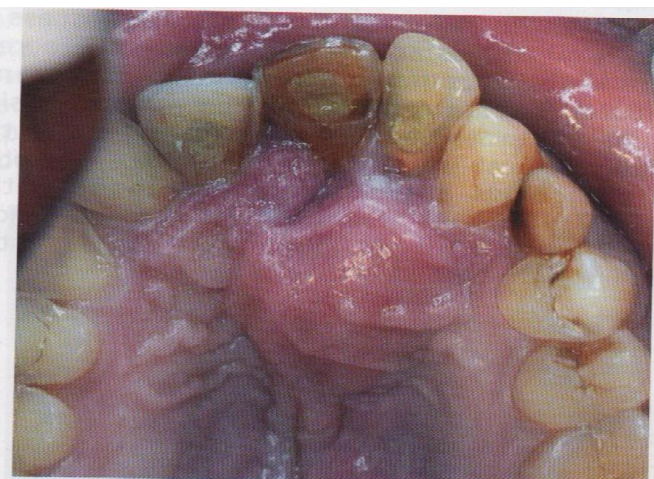


Fig. 8. Acute apical abscess in relation to maxillary incisors presenting as a swelling on the palate



Fig. 9. Purulent discharge seen on opening the access of a maxillary central incisor associated with an acute apical abscess

Phoenix Abscess

- Clinically often indistinguishable from acute apical abscess.
- At the onset tenderness of tooth and extrusion of tooth from socket
- Tenderness on palpating the apical soft tissue
- Most commonly associated with initiation of root canal treatment
- History from patient
- Pulp tests shows negative response
- Radiographs show large area of radiolucency in the apex

Persistent apical periodontitis

- It is post treatment apical periodontitis in an endodontically treated tooth.
- It may persist because of complexity of pulp space which cannot be reached by instruments or irrigants and thus obturating material. Nair Listed following extracellular factors which contribute to persistent apical periodontitis.

- Foreign body reaction to gutta-percha
- Periapical biofilm
- Cholesterol crystals
- Periapical scar tissue
- Actinomyces infection

Exposed Sensitive dentin

- Primary cause for dentin hypersensitivity is exposed dentin tubules which can occur due to loss of covering periodontal structures (gingival recession) or by loss of enamel.
- The causes of loss of enamel include Attrition, Abrasion and Erosion
- Tooth hypersensitivity differs from dentinal or pulpal pain. In case of dentin hypersensitivity, patients ability to locate the source of pain is good, whereas in case of pulpal pain, it is very poor.

- The character of the pain does not outlast the stimulus, the pain is intensified by thermal changes, sweet and sour.
- Intensity of pain is usually mild to moderate.
- The pain can be duplicated by hot or cold application or by scratching the dentin. The pulpal pain is explosive, intermittent and throbbing and can be affected by hot and cold.

Tooth Infractions

- It is defined as “Incomplete tooth fracture extending partially through a tooth”. The fracture commonly involves enamel and dentin, but sometimes pulp and periodontal structures may also get involved.

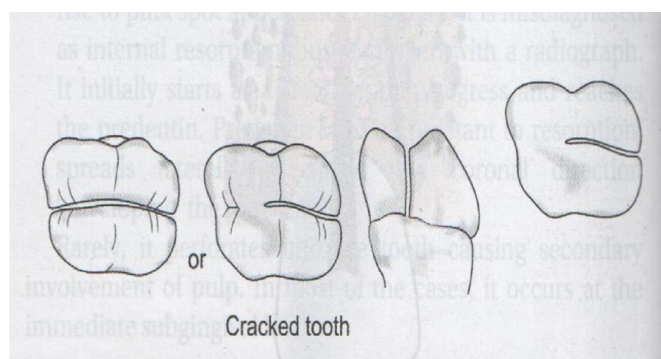


Fig. 10 Diagrammatic representation of cracked teeth

- Tooth infractions include Craze line, Cracked teeth and cuspal fracture.
- Mandibular second molars, followed by mandibular first molars and maxillary premolars are the most commonly affected teeth.
- While the crack tends to have a mesiodistal orientation in most teeth, it may run buccolingually in mandibular molars.
- Pain on biting that ceases after the pressure has been withdrawn, is a classical sign.
- The patient may have difficulty in identifying the affected tooth as there are no proprioceptive fibers in the pulp chamber.
- Vitality testing gives a positive response

- The tooth is not tender to percussion in an axial direction
- Pain increases as the occlusal forces increases, and relief occurs once the pressure is withdrawn.

Conclusion

The inflammatory changes in the pulp can produce reactions in the periapical tissues. Apart from pulpal disease, periodontal disease, iatrogenic factors and occlusal trauma are other etiologic factors for periapical diseases.

REFERENCES

- Ailor JE. 2000. Managing incomplete tooth fractures. *J Am Dent Assoc.*, 131:1168-74
- Baume IJ. 1970. Diagnosis of diseases of the pulp. *Oral Surg Oral Med Oral Pathol.*, 29:102-16
- Bhasker SN. 1966. Periapical lesions. Types, Incidence and clinical features. *Oral Surg Oral Med Oral Pathol.*, 21:657-71
- Cameron CE. 1964. Cracked tooth syndrome. *J Am Dent Assoc.*, 68:405-11
- Dummer PMH, Hicks R, Humes D. 1980. Clinical Signs and symptoms in pulp disease. *Int Endod J.*, 13:27-35
- Hasler JE, Mitchell DF. 1970. Painless pulpitis. *Int Am Dent Assoc.*, 81: 671-7
- Marton, Kiss C. 1993. Characterization of inflammatory cell infiltrate in dental periapical lesions. *Int Endod J.*, 26:131-6
- Michaelson PL, Holland GR. 2002. Is pulpitis painful? *Int Endod J.*, 35:829-32
- Morse DR, Seltzer S, Sinai I, Biron G. 1977. Endodontic Classification. *J Am Dent Assoc.*, 94: 685-9
- Nisha Garg and Amit Garg. 2014. Text Book of Endodontics, Third Edition.
- Pitt ford TR. 1982. The effects of the periapical tissues of bacterial contamination of filled root canal, *Int Endod J.*, 15: 16-22
- Ramya Raghu, Raghu Srinivasan. Endodontics, Current Concepts and Practice, First Edition, 2015
- Seltzer S. 1972. Classification of pulpal pathosis. *Oral Surg Oral Med Oral Pathol.*, 34:269-80
- Swepston JH, Miller AW. 1985. The incompletely fractured tooth. *J Prosthet Dent.*, 55:413-6.
