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Periodontic- Endodontic Lesions - A Review

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Abstract

Periodontics-Endodontic lesions arise from inflammation or degeneration of both pulpal and periodontal tissue as a result of the intimate anatomic relationship. Pulpal pathosis can play a major role in the initiation and perpetuation of periodontal attachment loss. Pulpal infection may cause a tissue destructive process that proceeds from the gingival margin. The term “retrograde periodontitis” was suggested in order to differentiate this from marginal periodontitis in which the infection spreads from the gingival margin toward the root apex. Another term, pulpodontic-periodontic syndrome, has been used to define a syndrome involving inflammation or degeneration of pulp with a periodontal pocket adjacent to the same tooth. This syndrome can be initiated by either pulpal or periodontal disease and may manifest pulpal and periodontal symptoms. Pulpal infection has the potential to initiate inflammatory changes in the alveolus at both apical and non-apical locations of teeth. Nevertheless the effect of periodontal disease on the pulpal tissue is controversial. Many examples have been cited and confirmed through animal studies and histological data demonstrating restoration of the periodontal tissues through successful endodontic therapy. Periodontists, being mainly concerned with treating gingival and periodontal disease, have little specialized knowledge about the problems of the diseased pulp. On the other hand, endodontists frequently have a lack of understanding concerning the treatment of the diseased periodontal tissue. It is important to correlate these two entities and treat periodontal endodontic lesions via inter disciplinary approach. The purpose of this article is to review the literature of periodontal-endodontic problems and the management of the same.

Keywords: perio-endo lesions, pathosis, pulpo periodontal, pulpal infection

Introduction

Tooth is a complex structure. Scientific advancements have segmented the field of dentistry into a vast number of specializations. Each specialized person performs a specific task. But there are instances where treatment from single specialist will not eradicate the disease, the reason being intricate nature of tooth, with its surrounding environment. There may be more than one implication of a particular disease. So it becomes mandatory to recognize various problems individually and treat them accordingly. An interdisciplinary approach among professionals becomes important for complete rehabilitation.

Over the years, there has been much discussion regarding the interrelationship between periodontic and endodontic disease. Pulpal and periodontal problems are responsible for more than 50% of tooth mortality. Many individuals have both pulpal and periodontal disorders that involve the same teeth. When this occurs, it is frequently difficult for the clinician to determine which disorder produced a specific sign or symptom. The diagnostic criteria used to distinguish between disease that may have originated from the pulpal necrosis or from attachment loss are not always sufficiently specific to allow determination of the disease etiology. This is especially true with advanced angular defects, pocket suppuration, swelling of the marginal gingiva, tenderness to percussion and increasing tooth mobility. All these may have more causes than plaque associated attachment loss. In all instances of concurrent periodontal and pulpal disorders, the clinician must determine whether the existing tissue change came from one or both processes so that appropriate treatment can be given. One process may have started the destruction and the other may have contributed at a later time or the lesion may be the result of one process alone.

It has been hypothesized that irritants may be transmitted between the pulpal and the periodontal structure. These tissues share common embryonic derivation, vasculatures, lymphatics, neural pathways and microflora. This juxtaposition of these tissues creates an intimate relationship. The apical foramen is the most important but by no means the only location where these tissues meet. Lateral and accessory canals, mainly in the apical area and in the furcation of molars, also connect the dental pulp with the periodontal ligament. In addition, a great number of dentinal tubules extend from the pulp to the cementum.

Anatomical considerations in pulpo-periodontal disease

1. Enamel

1**.** Deep pits fissures and lamella, favors plaque accumulation leading to pulpal disease. 2. The wedging effect of ideal cusp-fossa relations in teeth frequently produces incomplete or complete crown-root fractures which may produce a pulpo-periodontal lesion. 3. The lesion may start from the pulpal tissue extending to the periodontal tissues via the root apex or an accessory canal or from the periodontal side extending to the pulpal tissues via the apical or accessory foramen. 4. Enamel projections into furcations are involved which occur frequently and many be directly involved in pocket formation. 5. In an area where accessory canals into the pulp may lead to pulpo-periodontal disease.

2.Cementum

Exposure of cementum either hidden or visible is vulnerable to bacterial invasion. The breakdown of millions of sharpey's fibers leaves a sieve like surface full of canals which may be filled by the bacteria and toxins. Fissures and areas of incomplete calcification in cementum serve to channel inflammation into the deeper periodontal tissue. Lateral and accessory canals transverse the cementum which becomes exposed by root instrumentation and permit bacteria to move from periodontal to pulpal tissue or vice versa.[1]

3. Dentin

Open tubules may be penetrated by bacteria and their toxins from either the periodontal or the pulpal side. Tubules exposed from the periodontal side by caries, root instrumentation or fracture may cause an initial pulpal lesion. From the pulpal side, bacteria from caries may penetrate the cemental border if present or to the periodontal tissues leading to inflammation through dentinal tubules. If plaques of bacteria are permitted to accumulate on the surface, more ions may leave the dentin than are replaced and the result is likely to be surface demineralization and root sensitivity with the possibility of progressive pulp disease from a periodontal origin.[1]

4. Pulp

The pulp horns are more likely to assume clinical significance in the management of a carious lesion, whereas multiple apical foramina and accessory canals may serve to carry pulpal tissues, particularly in the furcation of molars.[1]

5. Periodontal ligament

The pocket in retrograde periodontitis is found over a prominent root suggesting alveolar dehiscence. Further, such a dehiscence may offer less resistance to advancing inflammation than the normal ligament. Inflammation, either periapical or periodontal may cause some elongation of the tooth because of swelling in the tissues and may produce secondary occlusal traumatism. In such instances, the ligament loses some of its protective-defensive ability and breaks down with advancing disease. Thus, an area of function, which is protective against many of the insults from inflammation and trauma, may become a channel for the progress of disease.

Etiological Factors Leading To Pulpo-Periodontal Problems

1. Live Pathogens

a. Bacteria:- *Aggregatibacter actinomycetemcomitans, Tannerella forsythia, Eikenella corrodens, Fusobacterium nucleatum, Porphyromonas gingivalis, Prevotella intermedia, and Treponema denticola*. These pathogens were found in all endodontic samples and the same pathogens were found in teeth with chronic apical periodontitis and chronic (adult) periodontitis. They concluded that periodontal pathogens often accompany endodontic infections and supported the idea that endodontic–periodontal interrelationships are a critical pathway for both diseases.[2]

b- Fungi (yeasts):- *C. albicans* has been detected in 21% of infected root canals using 18S rRNA directed species-specific primers. *C. albicans* also showed the ability to colonize canal walls and penetrate into dentinal tubules. Other species such as *Candida glabrata, Candida guillermondii,* and *Candida incospicia* and *Rodotorula mucilaginosa* were also detected.[3]

c-Viruses:-There is increasing evidence to suggest that viruses play an important role in both endodontic and periodontal diseases. In patients with periodontal disease, herpes simplex virus is frequently detected in gingival crevicular fluid and in gingival biopsies of periodontal lesions. Human cytomegalovirus was found in about 65% of periodontal pocket samples and in about 85% of gingival tissue samples. Epstein–Barr virus type I was detected in more than 40% of pocket samples and in about 80% of the gingival tissue samples. Gingival herpes viruses were associated with increased occurrence of subgingival *P. gingivalis, T. forsythia, P. intermedia, Prevotella nigrescens, T. denticola, and A. actinomycetemcomitans,* suggesting that they may play a role in promoting overgrowth of pathogenic periodontal bacteria. In endodontics, the presence of viruses in the dental pulp was first reported in a patient with AIDS. DNA of HIV virus has also been detected in periradicular

lesions. However, it has not been established that HIV virus can directly cause pulpal disease. Herpes simplex virus was also studied in relation to endodontic disease. However, unlike its role in periodontal disease, it appears that herpes simplex virus is not associated with endodontic disease. Human cytomegalovirus and Epstein– Barr virus play a role in the pathogenesis of symptomatic periapical lesions.[4]

2. Non - Living Etiologic Agents

a-Extrinsic Agents :

Foreign Bodies : Foreign bodies are frequently found to be associated with inflammation of the periradicular tissues. Although endodontic and periodontal diseases are primarily associated with the presence of microorganisms, some treatment failures may be explained by the presence of certain foreign substances in situ. These include substances such as dentin and cementum chips, amalgam, root canal filling materials, cellulose fibers from absorbent paper points, gingival retraction cords, leguminous foods, and calculus like deposits.

b- Intrinsic Agents

1.**Cholesterol**

The presence of cholesterol crystals in apical periodontitis is a common histopathologic finding. With time, the cholesterol crystals would be dissolved and washed away, leaving behind the spaces they occupied as clefts. The reported prevalence of cholesterol clefts in periapical disease varies from 18% to 44%. It has been suggested that the crystals could be formed from cholesterol released by disintegrating erythrocytes of stagnant blood vessels within the periapical lesion, lymphocytes, plasma cells and macrophages, which die in great numbers and disintegrate in chronic periapical lesions, or by the circulating plasma lipids.

2. Russell bodies

Russell bodies can be found in most inflamed tissues throughout the body including the periradicular tissues. These are small, spherical accumulations of an eosinophilic substance found within or near plasma cells and other lymphoid cells. The presence and occurrence of Russell bodies in oral tissues and periapical lesions is well documented. [5]

3.Rushton hyaline bodies: The presence of Rushton hyaline bodies (RHB) is a feature unique to some odontogenic cysts. Their frequency varies from 2.6% to 9.5%. RHB usually appear within either the epithelial lining or the cyst lumen. They have a variety of morphologic forms, including linear, irregular, rounded and polycyclic structures, or they may appear granular. The exact nature of RHB is not fully understood.[6]

4.Charcot-Leyden crystals: Charcot-Leyden crystals (CLC) are naturally occurring hexagonal bipyramidal crystals derived from the intracellular granules of eosinophils and basophils. Their presence is most often associated with increased numbers of peripheral blood or tissue eosinophils in parasitic, allergic, neoplastic, and inflammatory diseases. Activated macrophages were reported to have an important role in the formation of CLC in several disease processes. CLC and damaged eosinophils, along with their granules, have been observed within macrophages.[7]

Contributing Factors Leading To Pulpo-Periodontal Problems

1.Poor Endodontic Treatment: Poor endodontic treatment allows canal reinfection, which may often lead to treatment failure. Clinical signs and symptoms as well as radiographic evidence of periradicular lesions are usually associated with endodontic failure. Endodontic failures can be treated by either orthograde or retrograde retreatment with good success rates.

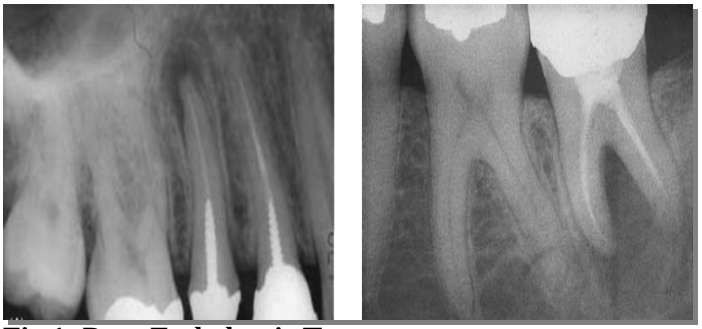


Fig 1: Poor Endodontic Treatment

2. Poor Restorations: Coronal leakage is an important cause of failure of endodontic treatment. Root canals may become decontaminated by microorganisms due to delay in placement of a coronal restoration and fracture of the coronal restoration and/or the tooth, it was therefore recommended that excess of gutta-percha filling should be removed to the level of the canal orifices and that the floor of the pulp chamber be protected with a well-sealed restorative material. Coronal restoration is the primary barrier against coronal leakage and bacterial contamination of endodontic treatment. Therefore it is essential that the root canal system be protected by good endodontic obturation and a well-sealed coronal restoration. However, even popular permanent restorative materials may not always prevent coronal leakage. Cemented full crowns as well as dentin-bonded crowns also showed leakage.[8]

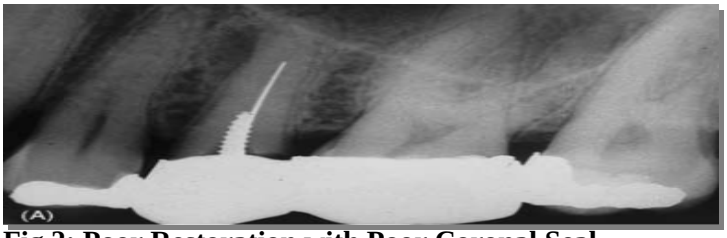


Fig 2: Poor Restoration with Poor Coronal Seal

3. Trauma: Trauma to teeth and alveolar bone may involve the pulp and the periodontal ligament. Both tissues can be affected either directly or indirectly. Dental injuries may take many shapes but generally can be classified as enamel fractures, crown fractures without pulp involvement, crown fractures with pulp involvement, crown–root fracture, root fracture, luxation, and avulsion. Treatment of traumatic dental injuries varies depending on the type of injury and it will determine pulpal and periodontal ligament healing prognosis.[9]

4.Resorption:[10]Root resorption is a condition associated with either a physiologic or a pathologic process resulting in a loss of dentin, cementum and/or bone. It may be initiated in the periodontium and affect initially the external surfaces of the tooth (external resorption) or it may start within the pulp space affecting primarily the internal dentin surfaces (internal resorption). If not diagnosed and treated, external root resorption may invade cementum, dentin and ultimately the pulp space. In cases of untreated internal resorptions the process may advance and perforate to the external root surface.

External root resorption may be divided into three main categories:

1) Progressive inflammatory resorption

2) Invasive resorption (non inflammatory)

3) Replacement resorption (non inflammatory)

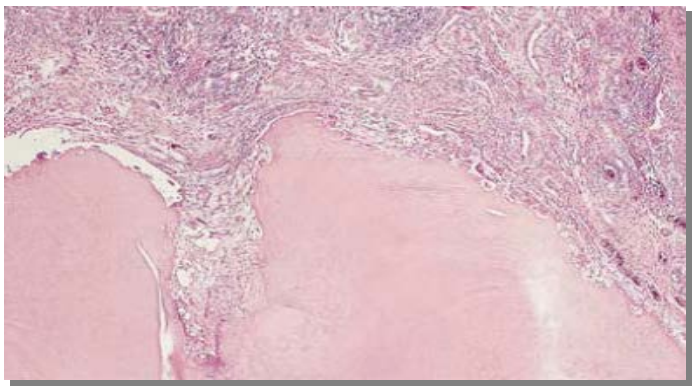


Fig 3: Periapical lesion showing multiple resorptive areas, inflammatory infiltrate, and osteoclasts.

5. Perforations :

Root perforations are undesirable clinical complications that may lead to treatment failure. When root perforation occurs, communications between the root canal system and either periradicular tissues or the oral cavity may

often reduce the prognosis of treatment. Root perforations may result from extensive carious lesions, resorption, or from operator error occurring during root canal instrumentation or post preparation.[11]

6. Developmental Malformations

Teeth with developmental malformations tend to fail to respond to treatment when they are directly associated with an invagination or a vertical developmental radicular groove. Such conditions can lead to an untreatable periodontal condition. These grooves usually begin in the central fossa of maxillary central and lateral incisors crossing over the cingulum, and continuing apically down the root for varying distances. Such a groove is probably due to the failure of the tooth germ to form another root. As long as the epithelial attachment remains intact, the periodontium remains healthy. However, once this attachment is breached and the groove becomes contaminated by bacteria, a self-sustaining infrabony pocket can be formed along its entire length.

Pathways of Communication Between The Pulp And The Periodontium.

Despite our substantial understanding of the etiology of periodontal and endodontic disease, we are frequently at a loss to explain how one process occurring primarily in the pulp or periodontal tissue might affect the other process. A preponderance of has established pathways of communication between the pulp and the periodontal tissue.

The various pathways of communication between the pulp and the periodontium may be classified as follows,

Developmental

Apical Foramen.

Lateral or Accessory Canals.

Dentinal Tubules.

Developmental or Lingual Grooves.

Tooth / Root Anomalies

Pathological

Empty Spaces Created by Destroyed Sharpey's Fibers.

Root Fractures Following Trauma.

Idiopathic Resorption (Internal or External).

Cemental Agenesis or Hypoplasia

Iatrogenic

Exposure of dentinal tubules following root planing.

Accidental lateral perforations during endodontic treatment.

Classification of Endodontic-Periodontal Lesions

Because of the close relationship between endodontics and periodontics, various classifications have been suggested for lesions affecting the pulpal and periodontal tissues.

Weine's classification of endo-perio problems: [12]

This classification is based on the fact that four types of endodontic-periodontal cases are commonly encountered. Divisions of the cases are based on the etiology of the disease which determines the types of therapy required and the probable diagnosis.

Class 1: Tooth in which symptoms clinically and radiographically simulate periodontal disease but are in fact due to pulpal inflammation and/or necrosis.

Class 2: Tooth that has both pulpal and periapical disease concomitantly.

Class 3: Tooth that has no pulpal problem but require endodontic therapy plus root amputation to gain periodontal healing.

Class 4: Tooth that clinically and radiographically simulates pulpal or periapical disease but in fact has periodontal disease.

The classification of teeth with pulpo-periodontal problem suggested by **P.H.A. Guldener**[13]is based mainly on the etiology of the disease.

Class 1: Primary endodontic lesion

Class 1(a): Accidental perforations (intra-alveolar) or resorptive perforations (internal resorption)

Class 1(b): Chronic periradicular lesion (granuloma or cyst) or acute Periradicular lesion(alveolar abscess)

Class 2: Primary periodontal lesion.

Class2 (a):Advanced periodontal disease with or without extension to the apical area (pulp vital)

Class2 (b): Secondary endodontic involvement. Infection through lateral canals or Dentinal tubules. Pulpal necrosis with or without secondary periapical involvement (pulp non-vital)

Class 3: Combined lesion-True combined lesion (coalescence between periodontal and endodontic lesion or vertical crown root fracture with pulpal involvement.

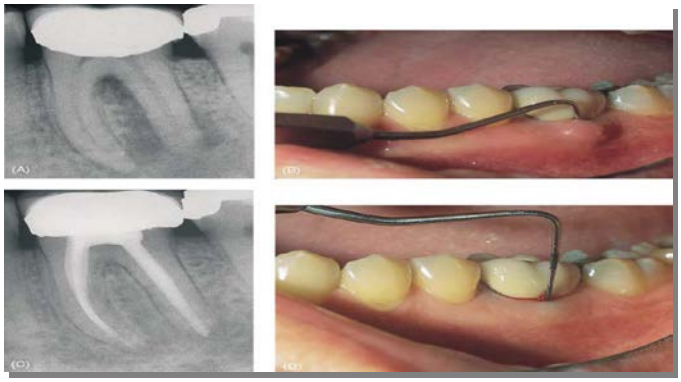


Fig 4: Primary endodontic disease in a mandibular first molar with a necrotic pulp.

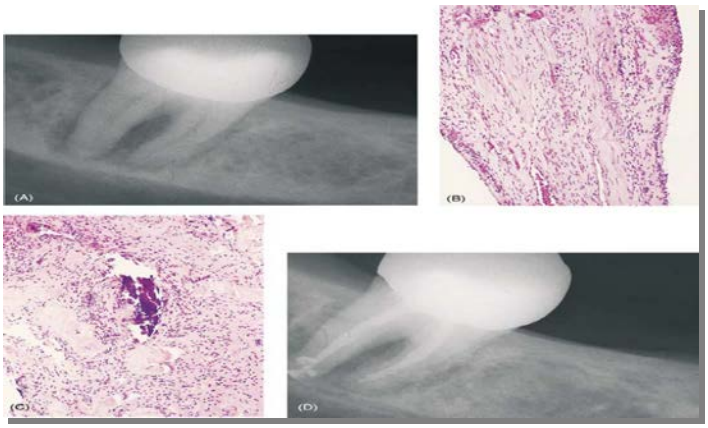


Fig 5 : Primary periodontal disease in a mandibular second molar. Patient was referred for endodontic therapy

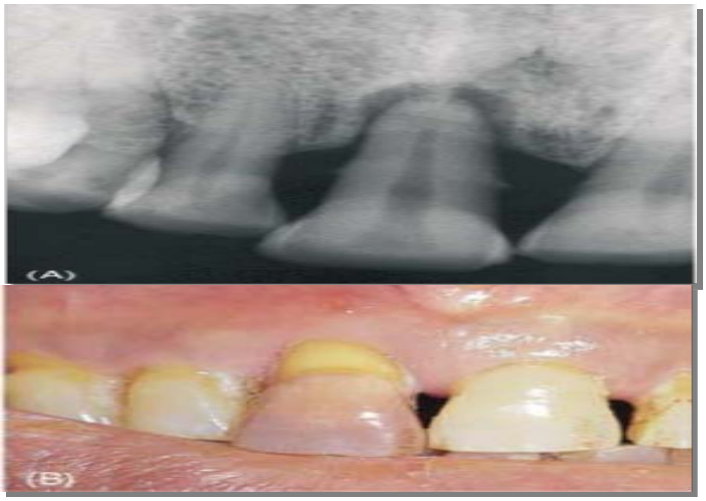


Fig 6: True combined endodontic periodontal disease.

Glickman's classification[14]of the pulpal periodontal disease depends on the pathways of the spread of inflammation as;

i. A periapical lesion originating in pulpal infection may have a pathway of fistulization from the apex and along the root to the gingival.

ii. Marginal periodontitis can progress to the apex of a root or to the emergence of an accessory canal and induce a secondary pulpal involvement.

iii. A true combined lesion is present when two separate lesion of endodontic and periodontal origin coalesce.

A similar form of classification is given by

William H. Hiatt.[15]

1. Pulpal lesions with secondary periodontal disease of short duration.
2. Pulpal lesion with secondary periodontal disease of long duration.
3. Periodontal lesion of short duration with secondary pulpal disease.
4. Periodontal lesion of long duration with secondary pulpal disease.
5. Periodontal lesion treated by hemisection or root amputation.
6. Complete and incomplete crown-root fracture.

7. Independent pulpal and periodontal lesion which merge into a combined lesion.

8. Pulpal lesion which evolve into periodontal lesion following treatment.

9. Periodontal lesion which evolve into pulpal lesion following treatment.

According to **Simon, Glick and Frank**[16] endodontic-periodontal problem could be classified as follows:

A. An originally endodontic problem, with fistulation from the apex and along the root to the gingival from apex or a lateral canal.

B. Fistulation through the apex or a lateral canal causing bifurcation involvement.

C. A long standing periapical lesion draining through the periodontal ligament can become secondarily complicated leading to "Retrograde Periodontitis"

D. A periodontal pocket can deepen to the apex and secondarily involve the pulp.

E. Secondary endodontic involvement- The primary periodontal involvement at the cervical margin can expose the lateral canal to the oral environment resulting in pulpal necrosis.

F. Two independent lesions, periapical and marginal, can co-exist and eventually fuse with each other.

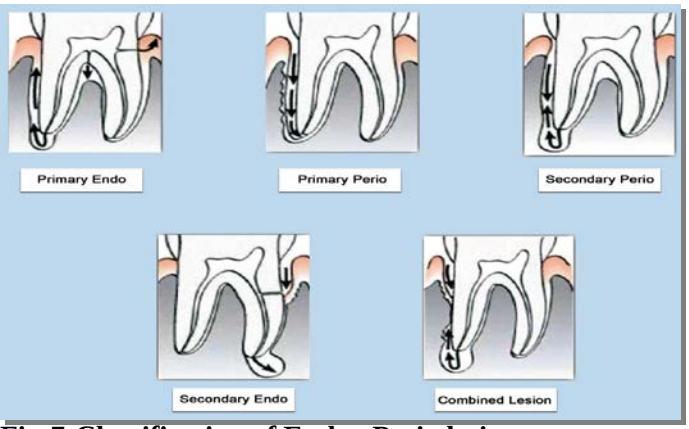


Fig-7-Classification of Endo –Perio lesions

Diagnosis

It is incumbent to gather all relevant information via history and examination. So, appropriate diagnostic aids are needed to correlate this information to determine the condition which might be consistent with the findings. It also helps to differentiate between various conditions and to arrive at the correct diagnosis.

History

Considerable information may be gained from the patients medical and dental history. A through history of the onset, duration and progress of the problem should be noted. This should include sign and symptoms relating to present or past pulpal or periodontal disease and also a history of trauma to the tooth. The chief complaint itself may establish the diagnosis. Usually pulpal problems are of acute onset whereas periodontal problems are chronic in nature.

Clinical Examination

1. Visual examination A thorough visual examination of the lips, cheeks, oral mucosa, tongue, palate and muscles should be done routinely. Digital examination of the same tissues is performed simultaneously. The alveolar mucosa and attached gingiva are examined for the presence of inflammation, ulcerations, or sinus tracts. Frequently, the presence of a sinus tract is associated with a necrotic pulp. Changes in color, texture and architecture of the gingival tissues may indicate periodontal pathology. Likewise aberrations of the teeth must be assessed in detail. Restoration of various types, fracture lines, carious lesions, etc suggests the possibility of pulpal pathology. The teeth are examined for abnormalities such as caries, defective restorations, erosions, abrasions, cracks, fractures, and discolorations. A discolored permanent tooth may often be associated with a necrotic pulp. A ''pink spot'' detected in the tooth crown may indicate an active internal resorption process. A conclusive diagnosis for pulpal disease cannot be achieved by visual examination alone. It therefore must always be accompanied by additional tests. Visual examination is

dramatically improved by the use of enhanced magnification and illumination.

2. Pain

Several aspects of pain should be considered when differentiating between pulpal and periodontal pathosis. They include the type, intensity, frequency, duration and activators of pain. Early pulpitis gives pain on cold, which is mild and is of short duration. Pain occurs spontaneously during these early stages of pulpal inflammation, where there is poor localization and the pain may be referred to other sites. Pain due to the pulpal involvement is usually acute in onset and severe. Heat produces pain that is sharper, more severe and of longer duration. It does not usually respond to potent analgesics. As the inflammation increases in intensity, the pain becomes more severe, of longer duration and is felt with hot or cold. Finally continuous severe pain may be felt even at room temperature, suggesting pulpal degeneration and necrosis.

3. Swelling

Swelling caused by pulpal infections often occurs in the mucobuccal folds or spreads to the facial planes. Muscle attachments and root length determine the route of drainage. Swelling caused by periodontal infection is usually found in the attached gingival and rarely spreads beyond the mucogingival junction and usually does not cause facial swelling.

Suppuration or Abscess Formation

Suppuration can occur with either pulpal or periodontal pathosis. If associated with periodontal disorders, it may be linked with an acute periodontal abscess, a chronic periodontal disease, or an acute exacerbation of a chronic disorder. Suppuration associated with pulpal pathosis may be with either an acute or chronic condition such as an acute alveolar abscess or a chronic alveolar abscess draining through a fistula.

5. Probing:

In case of periapical abscess, due to pulpal involvement the drainage is through the fistula, so the conventional periodontal probing cannot determine the origin of the sinus tract as no pocket is present. If the guttapercha point goes to the mid root, furcation, or any other portion of the tooth, a lateral canal or periodontal involvement is diagnosed.

In case of chronic periodontal disease conditions where there is bone loss, the periodontal probe may reach the apex of the root. Thus the periodontal probing to the apex may not always indicate a pulpal involvement. In such cases, only pulp test can determine the diagnosis.

6. Mobility:

Mobility testing can be performed using two mirror handles on each side of the crown. Pressure is applied in a facial–lingual direction as well as in a vertical direction and the tooth mobility is scored. Tooth mobility is directly proportional to the integrity of the attachment apparatus or to the extent of inflammation in the periodontal ligament. Teeth with extreme mobility generally have little periodontal support, indicating that the primary cause may be periodontal disease. Fractured roots and recently traumatized teeth often present high mobility. [17]

7.Tests

a. Percussion

Percussion is performed by tapping on the incisal or occlusal surfaces of the teeth either with the finger or with a blunt instrument such as the back end of a mirror handle. The tooth crown is tapped vertically and horizontally. Although this test does not disclose the condition of the pulp, it indicates the presence of a periradicular inflammation. An abnormal positive response indicates inflammation of the periodontal ligament that may be either from pulpal or periodontal origin.

b. Palpation

Palpation is performed by applying firm digital pressure to the mucosa covering the roots and apices. With the index finger the mucosa is pressed against the underlying cortical bone. This will detect the presence of periradicular abnormalities or ‘‘hot’’ zones that produce painful response to digital pressure. A positive response to palpation may indicate active periradicular inflammatory process. However this test does not indicate whether the inflammatory process is of endodontic or periodontal origin. Also, as with any other clinical test, the response should be compared to control teeth.[18]

c. Pulp Vitality Testing

These tests are designed to assess the response of the pulp to different stimuli. It also helps the clinician to determine whether the lesion is of pulpal or periodontal origin. An abnormal response may indicate degenerative changes in the pulp. In general, no response indicates pulp necrosis, and moderate transient response indicates normal vital pulp. A quick painful response may often indicate reversible pulpitis and lingering painful response indicate irreversible pulpitis. Since some of these tests may provoke a painful reaction they should be carefully performed and their nature and importance explained to the patient. When correctly performed and adequately interpreted these tests are reliable in differentiating between pulpal disease and periodontal disease.

d. Cold Test

This test is performed by applying a cold substance, or agent, to a well-isolated tooth surface. Tooth isolation can be achieved by drying the crown surfaces with cotton rolls, gauze and a very gentle air blast. Several cold methods are used: ice sticks, ethyl chloride, carbon dioxide (dry ice), and refrigerants such as dichlorodifluoromethane (DDM). Carbon dioxide (–78 8C) and DDM (–50 8C) are extremely cold and are only used when the pulp does not respond to less cold agents. An intense and prolonged pain response often indicates abnormal pulpal changes and irreversible pulpitis. Lack of response may indicate pulp necrosis.

e. Electric Pulp Test

This test is performed by applying an electric stimulus to the tooth using a special pulp tester device. The tooth is first cleaned, dried and isolated. A small amount of toothpaste is placed on the electrode of the pulp tester, which is then put into contact with the clean tooth surface. Only sound tooth structure should be contacted. Electric current is gradually applied until the patient reports sensation. No response frequently indicates pulp necrosis. A positive response may be interpreted as either intact vital pulp or partially necrotic pulp. [19]

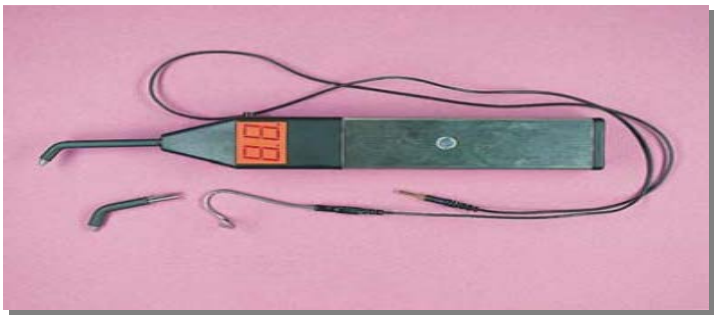


Fig 8: Pulp Vitality Tester

f. Heat Test

The normal response of a healthy pulp to heat is pain that increases in intensity and decreases immediately once the heat is removed. Lingering pain indicates an irreversible inflamed pulp. In case of periodontally involved teeth, persistence of pain even after removal of stimulus indicates an endodontic involvement.

g. Blood Flow Test

This test is designed to determine the vitality of the pulp by measuring its blood flow rather than the response of its sensory nerve fibers. Different systems such as dual wavelength spectrophotometry, pulse oximetry, and laser Doppler have been developed to measure either oxyhemoglobin, low concentration of blood, or pulsation

of the pulp. Sensors are applied to the external surfaces of the crown and the pulp blood flow is recorded and compared to controls. The procedure is non-invasive and painless. These tests are relatively new and are not used routinely.

h. Test cavity

This test is highly reliable in determining the vitality of the pulp. It basically consists of creating a cavity in the tooth without anesthesia. A high-speed handpiece with a new sharp bur is generally used. A positive response indicates presence of vital pulp tissue, while a negative response accurately indicates pulp necrosis. If no response is obtained, the cavity is extended into the pulp chamber and endodontic treatment is initiated.

i. Restored Teeth Testing

ii. Testing teeth with extensive coronal restorations is somewhat more challenging. Whenever possible, the restoration should be removed to facilitate pulp testing. In cases where restoration removal is not feasible, a small access opening is made through the restoration until sound tooth structure is reached. Cold test and cavity test will give the most reliable results. In most instances electric pulp testing will not prove beneficial.[20]

Access through full gold crowns can usually be done without affecting the strength and stability of the restoration. Access repair is done with amalgam, or another permanent filling material. Access for pulp testing can be done through porcelain restorations as well. In such cases, access is done slowly and with copious water irrigation.

8. Fistula Tracking

Endodontic or periodontal disease may sometimes develop a fistulous sinus track. Identifying the source of inflammation by tracking the fistula will help the clinician to differentiate between diseases of endodontic and periodontal origin. Fistula tracking is done by inserting a semi-rigid radiopaque material into the sinus track until resistance is met. Commonly used materials include gutta-percha cones or presoftened silver cones. A radiograph is then taken that will reveal the course of the sinus tract and the origin of the inflammatory process.[21]

9. Radiographic Examination

Radiographs are essential for detection of anatomic landmarks and a variety of pathological conditions. Radiographic examination will aid in detection of carious lesions, extensive or defective restorations, pulp caps, pulpotomies, previous root canal treatment and possible mishaps, stages of root formation, canal obliteration, root resorption, root fractures, periradicular radiolucencies, thickened periodontal ligament, and alveolar bone loss.[22]

10. Cracked Tooth Testing [23]

Transillumination

This test is designed to aid in the identification of cracks and fractures in the crown. A fiberoptic connected to a high-power light source is used to illuminate the crown and gingival sulcus. The contrast between the dark shadow of the fracture and the light shadow of the surrounding tissue will clearly reveal the size and orientation of the fracture line. An existing restoration may need to be removed to enhance visibility.

Wedging

This technique aids in the identification of vertical crown fractures or crown–root fractures. Such fractures cause a painful response to the patient at the time of chewing. During the test, wedging forces are created as the patient is instructed to chew on a cottonwood stick or other firm material. This test is fairly reliable in identifying a single tooth causing pain during mastication. Many of these fractures involve only the tooth crown and terminate in the pulp chamber. Such cases are treated successfully with endodontic therapy.

Staining

Staining identifies lines of fracture in the crown and root and is often used in conjunction with the wedging test. The tooth crown is dried and a cotton pellet soaked with methylene blue dye is swabbed on the occlusal surface of the tooth. The patient is asked to bite on a stick and perform lateral jaw movements. This way the dye penetrates well into the zone of the fracture. The dye is then rinsed from the tooth surfaces and visual examination with magnifying loops or the microscope will reveal a distinctive fracture line darkened with dye.

11. Selective Anesthesia Test

This test is useful in cases where the source of pain cannot be attributed to a specific arch. Disappearance of pain following a mandibular block will confirm the source of pain originating from a mandibular tooth. The periodontal ligament injection is often used to narrow down the zone in question; however, it cannot anesthetize a single tooth without affecting adjacent teeth.[24]

Differential Diagnosis [26]

For differential diagnostic purposes the ‘‘endoperio lesions’’ are best classified as endodontic, periodontal or combined diseases. They can also be classified by treatment depending on whether endodontic, periodontal or combined treatment modalities are necessary. They include: primary endodontic disease, primary periodontal disease, and combined diseases. The combined diseases include: primary endodontic disease with secondary periodontal involvement, primary periodontal disease with secondary endodontic involvement, and true combined diseases.

Primary Endodontic Disease

An acute exacerbation of a chronic apical lesion on a tooth with a necrotic pulp may drain coronally through the periodontal ligament into the gingival sulcus. This condition may mimic clinically the presence of a periodontal abscess. In reality, it is a sinus tract from pulpal origin that opens through the periodontal ligament area.

Primary Periodontal Disease

These lesions are caused primarily by periodontal pathogens. In this process, chronic periodontitis progresses apically along the root surface. In most cases, pulp tests indicate a clinically normal pulpal reaction. There is frequently an accumulation of plaque and calculus and the pockets are wider. The prognosis depends upon the stage of periodontal disease and the efficacy of periodontal treatment. The clinician must also be aware of the radiographic appearance of periodontal disease associated with developmental radicular anomalies.

Combined Lesions:

Primary Endodontic Disease With Secondary Periodontal Involvement

If after a period of time a suppurating primary endodontic disease remains untreated, it may become secondarily involved with periodontal breakdown. Plaque forms at the gingival margin of the sinus tract and leads to plaque-induced periodontitis in the area. When plaque or calculus is detected, the treatment and prognosis of the tooth are different that those of teeth involved with only primary endodontic disease. The tooth now requires both endodontic and periodontal treatments. If the endodontic treatment is adequate, the prognosis depends on the severity of the plaque-induced periodontitis and the efficacy of periodontal treatment. With endodontic treatment alone, only part of the lesion will heal to the level of the secondary periodontal lesion.

Primary Periodontal Disease with Secondary Endodontic Involvement

The apical progression of a periodontal pocket may continue until the apical tissues are involved. In this case

the pulp may become necrotic as a result of infection entering via lateral canals or the apical foramen.

True Combined Disease

True combined endodontic–periodontal disease occurs less frequently than other endodontic–periodontal problems. It is formed when an endodontic disease progressing coronally joins with an infected periodontal pocket progressing apically. The degree of attachment loss in this type of lesion is invariably large.

	Endodontic condition	*Periodontal condition*	*Iatrogenic condition*
Visual examination	☐ Presence of sinus tract ☐ Presence of decay ☐ Large restoration ☐ Fractured restoration or tooth ☐ Erosions ☐ Abrasions ☐ Cracks ☐ Discolorations ☑ Poor RCT	☑ Gingivitis, periodontitis ☐ Gingival recession ☐ Accumulation of plaque ☐ Subgingival calculus ☐ Intact teeth ☐ Periodontal abscess	☑ Gingivitis, periodontitis ☐ Swelling around tooth ☐ Presence of pus, exudate ☑ Defective restoration ☑ Poor root canal treatment
Pain	☐ Sharp	☐ Dull ache ☐ Sharp only in acute condition	☐ Dull ache ☐ Sharp only in acute condition
Palpation	☐ Pain on palpation	☐ Pain on palpation	☐ Pain on palpation
Percussion	☐ Tender on percussion	☐ Tender on percussion	☐ Tender on percussion
Mobility	☐ Fractured roots and traumatized tooth present high mobility	☐ Localized and generalized mobility	☑ High mobility related to the involved tooth
Pulp vitality	☐ Irreversible pulpitis ☐ Necrotic pulp	☐ The pulp is vital	☐ Nonvital pulp
Sinus tracing	☐ A radiograph with gutta percha points to apex	☐ Sinus tract mainly at the lateral aspect of the root	☑ Difficult to trace out the origin of the lesion radiographically
Radiographs	☐ Deep carious ☑ Defective restorations ☑ Previous poor RCT ☐ Possible mishaps ☐ Root fractures ☐ Periapical radiolucency	☐ Vertical bone loss ☐ Bone loss wider coronally ☑ Previous poor RCT ☐ Possible mishaps ☑ Root resorption	☑ Periradicular bone loss ☑ Defective restorations
Cracked tooth test	☐ Painful response with chewing	☐ No symptoms	☐ Painful response with chewing

Fig. 9: Diagnosis of exact lesion[25]

	Pulpal	Periodontal
Etiology	Pulp infection.	Periodontal infection.
Pulp test	Non-vital.	Vital.
Microbial	Few organisms.	Complex microbial plaque.
Trauma (occlusion)	May be primary or secondary.	May be primary cause of a wide periodontal space or secondary cause in pocket formation.
Inflammation	Acute.	Chronic.
Ph	Usually acid.	Usually alkaline.
Root surface	No calculus.	Calculus.
Pocket	Narrow, one surface, often facial.	Wide coronally, narrow apically usually interproximal and part of a generalized disease.
Bone loss	One surface, often facial, wider apically, narrow coronally.	Multiple surfaces, usually interproximal wider coronally, often intrabony.
Radiographic findings	Funnel shaped, wider at apex but often superimposed over the root in facial pocket.	Generalized bone destruction coronally not involving periapical regions.
Restorative	Deep restorations.	Caries or deep restorations' not related.

HISTOPATHOLOGY		
1. Epithelium	No down growth.	Proliferation and down growth
2. Connective tissue	Fibre residue on root surface, minimal granulation tissue.	No fibre except apical portion, large quantity of granulation in pocket
3.Gingival	Little or no recession.	Gingival recession in chronic.
4.Therapy	Pocket closure with Endodontic treatment or with simple additional periodontal procedures.	Regeneration possible only with advanced periodontal treatment, except in pocket of recent origin from acute inflammation.

DIFFERENTIAL DIOGNOSIS OF PULPAL, PERIAPICAL, PERIODONTAL FINDINGS

	Pulpal	Periapical	Periodontal
Type of pain	Sharp lancinating	Dull or continuous	Dull or absent
Swelling	Absent	Generalized	Localized
Colour of tooth	Normal	Darkened	Normal
Percussion	Normal	Sensitive	Usually normal*
Extrusion	Absent	Extruded	Absent
Lymphatic	Absent	Enlarged	Absent
Vitalometer	1-Low range 2-Normal range 3-High range	No response	Normal
Periodontal	No probable defects	Normally single probable defect	Probable defects in many areas of the mouth
Radiographic examination	Caries, recent shallow filling. trauma	Deep caries Deep fillings (* Except in cases of traumatic occlusion)	Pocket calculus

Treatment of Endodontic Periodontal Lesions [27]

Primary endodontic lesions occur as a result of pulpal pathology and can be treated with root canal therapy alone. Surgical endodontal therapy is not regarded as necessary, even in the presence of large periapical radiolucencies and the presence of periodontal abscesses.

Primary endodontic lesions with secondary periodontal involvement have plaque, calculus and endotoxins within the periodontal pocket. Endodontic treatment must be completed first and may result in partial closure of the pocket if the periodontal irritants have not invaded all the way to the apex. Periodontal therapy should follow the endodontic treatment to ensure removal

of plaque, calculus and infected cementum that has developed on the root surface within the pocket.

Primary periodontal lesions usually require periodontal therapy alone. However, pulpal pathology maybe induced while carrying out the periodontal therapy. This should be considered when treating lesions in the furcation region where many pulps receive accessory blood supply.

The treatment of **primary periodontal lesions with secondary endodontic involvement** depends largely on the extent of the pulpal involvement. If diagnosed at an early stage, the involvement may be limited to reversible pulpal hypersensitivity. Treatment of the periodontal lesion removes the source of pulpal irritation and secondary mineralization of the dentinal tubules allows resolution of the pulpal hypersensitivity. When the periodontal lesion has progressed to involve accessory canals and caused irreversible pulpal inflammation or pulpal necrosis combined endodontic and periodontal therapy is again required. Endodontic therapy should be completed first

"True" combined lesions also require combined endodontic and periodontal therapy. The prognosis of teeth with "true" combined lesions is often poor' particularly when the periodontal lesions are chronic, with extensive loss of attachment. In some situations, it may be felt that entire tooth cannot be saved even with extensive combined therapy. However, root amputation, hemisection or separation may allow the root structure to be saved. Prior to surgery, palliative periodontal therapy should be completed and root canal therapy carried out on the roots to be saved.

The sequence of therapy for all endodontic-periodontic lesions is important and should be carefully considered when planning treatment. In some situation it may be felt that the entire tooth cannot be saved even with extensive combined therapy. Root amputation, hemisection or root separation may allow the root configuration to be changed sufficiently for part of root structure to be saved.

Hemisection:-[28]

Hemisection is the splitting of a two-rooted tooth into two separate portions. This process has been called *hienspillization* or *separation* because it changes the molar into two separate roots. Hemisection is most likely to be performed on mandibular molars with buccal and lingual class II or Ill furcation involvements. As with root resection, molars with advanced bone loss in the interproximal and interradicular zones are not good candidates for hemisection. After sectioning of the teeth, one or both roots can he retained. This decision is based on the extent and pattern of bony loss, root trunk and root length, ability to eliminate the osseous defect, and endodontic and restorative considerations. The anatomy of the mesial roots of mandibular molars often leads to their extraction and the retention of the distal root to facilitate both endodontic and restorative therapy.

Indications for hemisection

1. To divide the prognosis of the root of a given tooth.
2. To control the environment of the furcation.
3. To eliminate untreatable carious and/or endodontic lesions

Root Resection/Hemisection Procedure[29]

The most common root resection involves the distobuccal root of the maxillary first molar, --' as diagrammed in After appropriate local anesthesia, a full thickness mucoperiosteal flap is elevated. Root resection or hemisection of teeth with advanced attachment loss usually requires opening both facial and lingual/palatal flaps. Typically, a root cannot be resected without elevating a flap. !he flap should provide adequate access for visualization and instrumentation and minimize surgical trauma. After debridement, resection of the root begins with the exposure of the furcation on the root .The

removal of a small amount of facial or palatal bone may he required to provide access for elevation and facilitate root removal . A cut is then directed from just apical to the contact point of the tooth, through the tooth, and to the facial and distal orifices of the furcation. This cut is made with a high-speed, surgical-length fissure or crosscut fissure carbide bur. The placement of a curved periodontal probe into or through the furcation aids in orienting the angle of the resection. For hemisection, a vertically oriented cut is made faciolingually through the buccal and lingual developmental grooves of the tooth, through the pulp chamber, and through the furcation. If the sectioning cut passes through a metallic restoration, the metallic portion of the cut should be made before flap elevation. This prevents contamination of the surgical field with metallic particles.

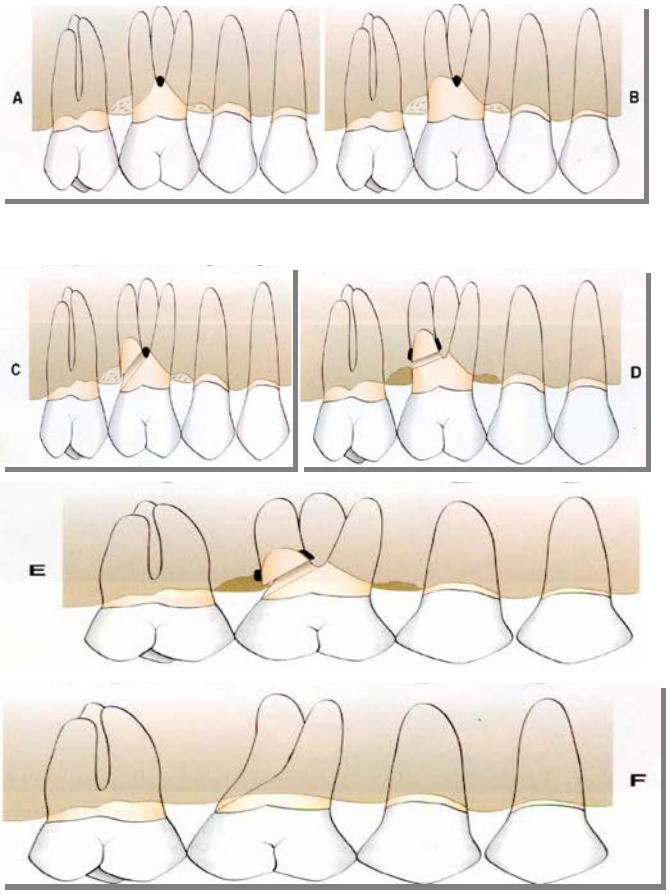
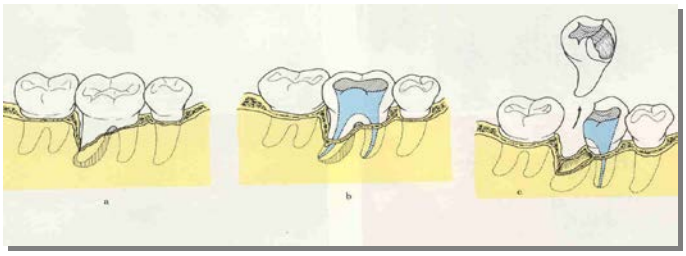


Fig 10: Steps In Root Resection Procedure



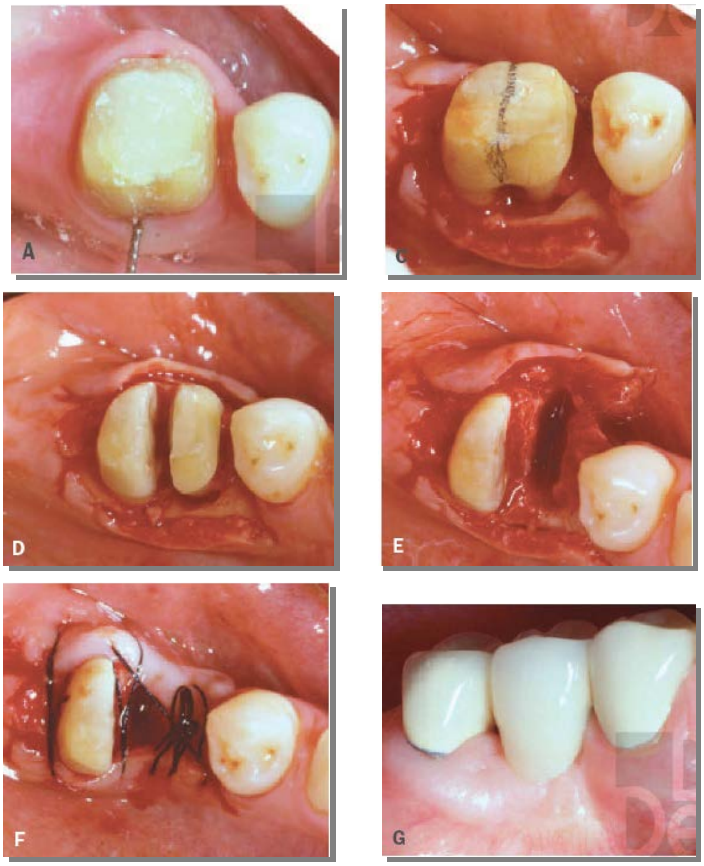


Fig-11: Hemisection procedure

Indications for root resection

1.The periodontal principle of strategic extraction.

2.Control of compromised embrasure due to tight root proximity.

3.Furcation control.4.Severe carious destruction.

5.Endodontically untreatable root.

Contraindications for Root Resection and Hemisection

1. Fused roots (along their length or at apex).
2. Roots in acutely close proximity.
3. Inability to treat tooth endodontically.

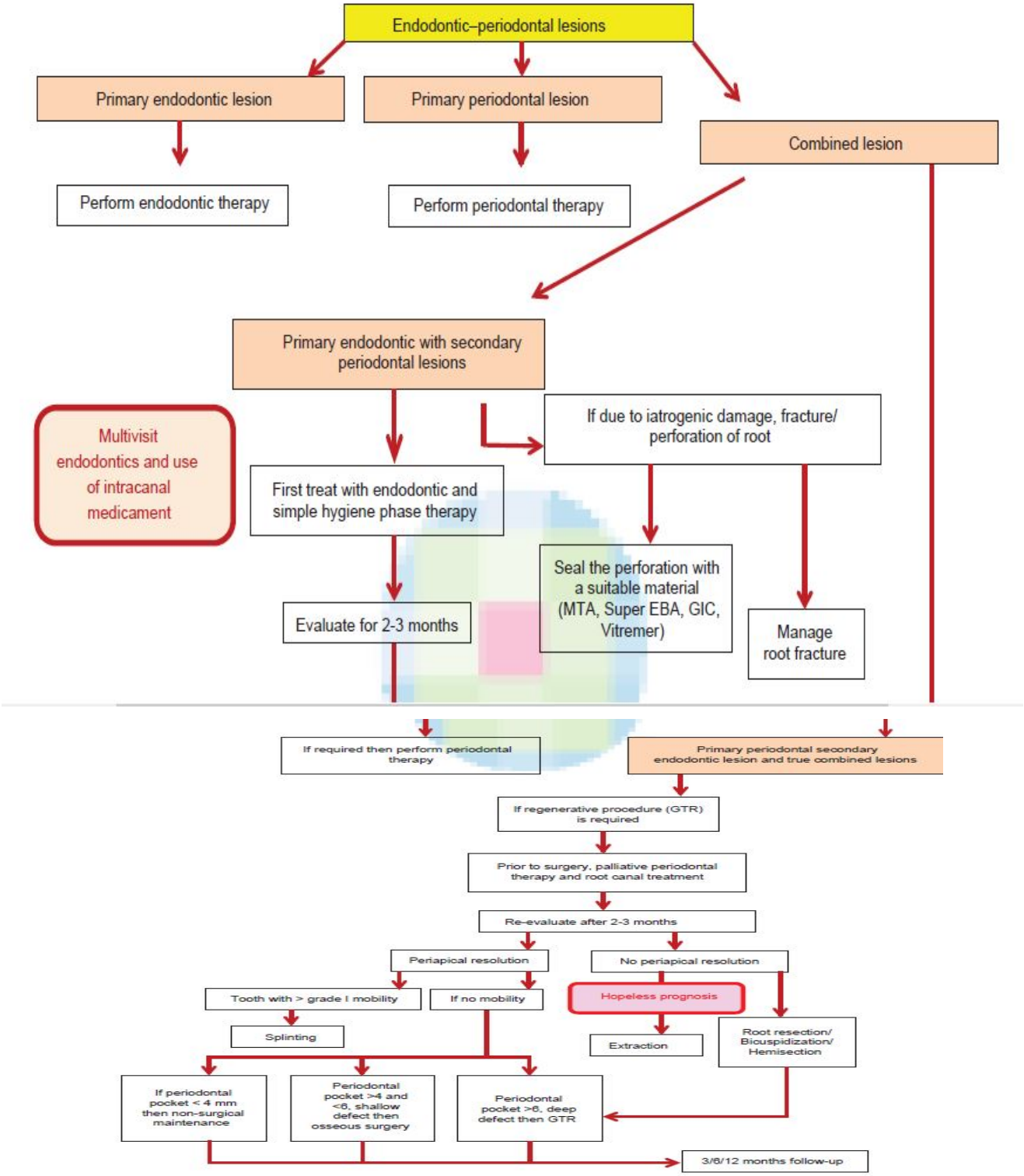


Fig. 12:Treatment Strategy in the Management of Endo-Perio Lesions

Effect of immediate periodontal surgical treatment on periodontal healing in combined endodontic–periodontal lesions with communication[30]

Periodontal healing in terms of improvement in periodontal parameters after 6 months of endodontic therapy in immediate surgical periodontal treatment was comparable to healing after 9 months of endodontic therapy in delayed surgical treatment in endodontic-periodontal apically communicating lesions which suggested that there may not be need to wait for 3 months after endodontic treatment for definitive periodontal therapy. There may not be any detrimental effect of immediate periodontal surgery on periodontal healing and results of the study are in favour of performing both treatments immediately as it has less treatment duration and better patient compliance.

Conclusion

All dental treatment seeks to preserve and maintain the teeth and supporting structures in a state of health and function. Both periodontics and endodontics are vitally concerned with the health of supporting structures of the teeth. The scope of periodontics and endodontics varies only by location of the involved portion of tooth and surrounding periodontal structures. The periodontal role focuses on the preservation or restoration of the periodontium. Whereas the root canal therapy is cleaning and shaping of the root canal space followed by its 3 dimensional obturation to restore or maintain the apical periodontium to a biologically acceptable state. The differential diagnosis of periodontal disease and puplal disease is essential to formulate a definitive treatment plan. A thorough understanding of the causative factors, pathways of communication common clinical signs and symptoms and treatment modalities will enable the clinician to achieve successful results.

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