IDIOPATHIC EXTERNAL CERVICAL ROOT RESORPTION: REVIEW WITH A CASE REPORT

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ABSTRACT

Resorption of the dental tissues is an inflammatory process and manifests in different forms and with varied symptoms. Idiopathic cervical resorption is a rare entity and usually comes to light at a later stage. This article presents a case report of this lesion along with a comprehensive review of the literature and treatment options available

Key Words: Resorption, Idiopathic, Cervical

INTRODUCTION

The extracanal invasive resorptive process begins in the gingival attachment or the periodontal ligament. It can be considered as a form of external resorption. It is usually asymptomatic and the patient may realise the condition only when the teeth develop mobility or colour change.

CASE REPORT:

A 25 year old male patient reported with a complaint of an exfoliated tooth and mobile lower front teeth since six months. He had consulted a dentist who had performed oral prophylaxis with an ultra sonic scaler. The patient however did not have any relief in the symptoms and the mobility increased with time until one day a tooth spontaneously and painlessly exfoliated. The patient was alarmed regarding some serious condition and decided to have a second opinion.

The medical history was non contributory and he was in a state of good health. He had suffered neither from trauma nor undergone any orthodontic, oral surgical or periodontal procedure except for oral prophylaxis after which he had noticed the mobility. Extra oral findings were normal.

Intra oral examination showed a root stump of mandibular left first pre molar (34) of which the exfoliated crown was brought by the patient (Fig 1). The gingival tissues exhibited marked swelling but there was no bleeding on probing (Fig 2). Mandibular anterior teeth exhibited grade I/II mobility from pre molar to pre molar region. However it was realized that the mobility was restricted to the crowns and the roots were not mobile. When a No. 23 explorer was used, the moon shaped end went deep inside at the level of CEJ (Fig 3).

IOPA Radiographs showed:

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a) Deep resorptive defect in 33 and beginning of resorption in 35 with root stump of 34 (Fig 4)

b) Deep resorptive defect in 33,32,31,41,42 (Fig 5)

c) No significant findings in the maxillary anterior region (Fig 6)

d) OPG confirmed the findings and it was noted that the resorption was limited to mandibular anterior region (Fig 7)

The patient was advised to get blood work up for Serum Calcium, Phosphorus and Para Thyroid hormones which were found to be under normal levels.

A clinical diagnosis of Idiopathic External Cervical Root Resorption was made.

The patient was further advised scanning by CBCT and explained the treatment options of:

1. Endodontic treatment followed by crown lengthening, post and core and crowns.
2. Endodontic treatment followed by over denture.
3. Extraction followed by conventional/ Implant supported partial denture.

However the patient refused any further diagnostic tests did not return for follow up.

DISCUSSION:

Root resorption is the loss of hard dental tissues i.e. dentin and cementum as a result of odontoclastic action [1]. It might occur as physiologic or pathologic phenomenon as intra radicular or apical according to the location in which the condition is observed. Physiologic resorption is observed in primary teeth which is desirable. However root resorption in permanent teeth is unfavorable as it might result in irreversible damage and/ or eventual tooth loss [2].

Root resorption might be classified by its location in relation to the root surface i.e. internal or external resorption. Internal resorption is an inflammatory condition that results in progressive destruction of the intra radicular dentin. The resorbed spaces are filled by granulation tissue only or in combination with bone like or cementum like mineralised tissue. A high prevalence of the condition has been associated with teeth that had undergone specific treatment procedures like auto transplantation [11].

External resorption can be further classified as surface resorption, external inflammatory resorption, external replacement resorption (ankylosis), external cervical resorption and transient apical breakdown. Other terms used to describe ECR include odontoclastoma, peripheral cervical resorption, peripheral inflammatory root resorption, extra canal invasive resorption, supra osseous extra canal invasive resorption and sub epithelial external root resorption [3].

ECR usually occur immediately below the epithelial attachment of the tooth at the cervical level. The remnants of the epithelial sheath surround the root like a net, thus imparting a resistance to resorption and subsequent ankylosis. The cementum and predentin covering on dentin are esential elements in resistance to resorption.

Another function of the cemental layer is related to its ability to inhibit the movement of toxins if persent in the root canal space into the surrounding periodontal tissue. An intact root is resistant to resorption however if an injury removes or alters the (protective) pre dentin or pre cementum, inflammation from the pulp or the periodontium may induce resorption with multi nucleated clastic cells similar to those seen in bone resorption. Damage or deficiency in this protective layer below the epithelial attachment exposes the root surface to osteoclasts which may resorb the dentin [10].

Thus in order for root resorption to occur, two things must happen [11]:

1. Loss or alteration of the protective layer

Damage to the protective layer occurs directly, due to trauma or indirectly, when inflammatory response occurs in pulp or periodontium. The only exception to this rule is intrusive injury, where forcing of the conical root apically into a similarly shaped socket will cause tremendous damage to the entire root surface. Inflammation in response to a traumatic injury varies according to the stimulus. If the periodontal ligament cells are allowed to dry out before replantation will provide a stimulus for an inflammatory response.

2. The inflammatory response

A. *The destructive phase*:

This is the first phase in which active resorption occurs with multinucleated giant cells. It continues as long as the stimulus is present. It will continue till either the entire root is resorbed or the stimulus is removed.

B. *The healing phase*:

The critical factor in determining the outcome after an injury is the type of cells that repopulate the root surface during the healing pahse. If cementoblasts are able to cover the damaged root surface, a type of healing termed as cemental healing or surface resorption will occur and the outcome is favorable.

If on the other hand bone producing cells are able to cover the root surface then healing conditions are unfavorable as direct contact of bone and root will occur leading to ankylosis. The root is slowly replaced by bone which is known as replacement resorption or osseous replacement. Destruction of over 20% root surface is required for it to occur.

Invasive cervical resorption is a clinical term used to describe a relatively uncommon, insidious and often aggressive form of external tooth resorption [1]. Characterized by its cervical location and invasive nature, it leads to a progressive and destructive loss of the tooth structure. There is invasion of fibro vascular tissue that progressively resorbs dentin and cementum. The common contributing factors include trauma, periodontal inflammation, internal bleaching and orthodontic forces.

Hiethersay's Classification [3]:

Class 1: early lesion which may show a slight irregularity in the gingival contour.

Class 2: in this class there might be pink discoloration of the crown while the radiographic picture shows extensive irregular radiolucency extending from the cervical area into the crown and projected over the canal outline.

The resorptive cavity is filled with a mass of fibrous tissue, blood vessels and clastic cells adjacent to the dentin surface. Pulpal symptoms occur only when the resorption penetrates this barrier and is secondarily invaded by microbes.

Class 3: in this class the resorptive process has extensions into but not beyond the coronal third of the root. Clinically the crown may show a pink discoloration and there might be a cavitation of the overlying enamel.

The radiographic image shows an irregular moth eaten image and the outline of the canal may be seen as a radiopaque line demarcating the canal from the adjacent irregular radiolucency.

Class 4: this class includes invasive process that has extruded beyond the coronal third of the root while the crown shows a pink discoloration in the cervical region.

Clinical signs:

1. Located in the cervical region of the tooth.
2. Pink spot is usually seen.
3. Tooth usually responds positively to thermal tests untill pulp is involved.

Radiographic signs –

1. Detected as chance radiologic findings because tooth is usually asymptomatic.
2. Varies from asymmetrically located radiolucency with irregular margins in cervical/proximal region of tooth to uniformly round radiolucency centered over the root.
3. Early lesions are usually radiolucent in appearance.
4. Advanced lesions might have mottled appearance because of fibro-osseous nature of the lesion.
5. Root canal should be visible and intact (indicating lesion is external) [10]

Multiple resorptions can occur, particularly when there has been a history of orthodontic treatment and a full mouth radiographic examination is essential. [7]

The extra canal invasive resorptive process begins in the gingival attachment or periodontal ligament. It can be considered as a form of external resorption. A relatively small hole develops in the periphery of the root during the initial resorptive process and extends through the cementum into the dentin. A number of small holes may be associated with a single extra canal invasive resorptive process. Extra canal invasive resorptive process extends as finger like projections into the dentin. With in the body of dentin it tends to extend in a vertical direction apically, coronally or both. The radiolucency may appear to be parallel to the canal space and may assume a mottled appearance. [1] The status of the gingival tissue in patients appeared to have no influence on the occurrence or extent of multiple cervical resorption. [4]

External resorption is a process that leads to an irreversible loss of cementum, dentin and bone. It takes place in both vital and

pulp less teeth. Clinically, cervical external resorption is associated with inflammation of the periodontal tissues and does not have any pulpal involvement.[7]

Cervical external resorption occurs immediately below the epithelial attachment of the tooth. As a result, it must be noticed that the location is not always cervical but related to the level of the marginal tissues and the pocket depth. In particular, teeth with cementum deficiencies related to previous trauma or a cemento-enamel disjunction (10%) due to histological variations an unprotected, locally destroyed or altered root surface which has become susceptible to resorbing clastic cells during an inflammatory response of the periodontal ligament to traumatic or bacterial stimulus, maintained by infection in the adjacent marginal tissues. [9]

Other types of root resorption:

Pulp space infection-

Apical periodontitis with apical root resorption:

The predominant cause is a bacterial challenge through caries. When the pulp defenses are overcome and the pulp is necrotic and infected, the inflammatory stimulators will contact the surrounding periodontal attachment. The periodontal inflammation is accompanied by slight resorption of the root at the cement-dentinal junction. [2]

Lateral periodontitis with root resorption:

When the root loses its cemental protection, lateral periodontitis with root resorption can result the periodontal infiltrate consists of granulation tissue with lymphocytes, plasma cells, and polymorphs nuclear leukocytes. [8]

Sulcular infection-

The progressive external root resorption, which is of inflammatory origin, occurs immediately below the epithelial attachment of the tooth, usually but not exclusively in the cervical area of the tooth it is referred to as sub epithelial external root resorption. Causes of the root damage immediately below the epithelial attachment of the root include orthodontic tooth movement, trauma, non-vital bleaching and other less definable cause. The pulp plays no role in cervical root resorption and is mostly normal in these cases. [5]

The invading tissue arises from the periodontal ligament but differs from periodontal tissues in both structure and behavior. For invasion to occur, a defect in the cementum/cementoid layer is a likely prerequisite. This may be of developmental origin in a small zone near the cervical area, or the result of physical or chemical trauma. Presence of inflammatory cells is not necessarily indicative of a microbiological etiology. [11]

Internal (root canal) inflammatory resorption –

This type of resorption might occur in any area of the root canal systems. It is characterized by the radiographic appearance of an oval shaped enlargement within the pulp chamber resulting in a perforation or symptoms of acute or chronic apical periodontitis after the entire pulp has undergone necrosis and the pulp space has become infected. It involves a progressive loss of intraradicular dentin

without adjunctive deposition of hard tissues adjacent to the resorptive sites. It is frequently associated with chronic pulpal inflammation, and bacteria might be identified from the granulation tissues. [10]

The coronal part of the pulp is usually necrotic, whereas the apical part of the pulp must remain vital for the resorptive lesion to progress and enlarge in the presence of living osteocytes, osteoclasts fail to produce actin rings, which are the hallmark of active resorbing cells. [11]

Internal (root canal) replacement resorption-

Internal root canal replacement resorption is characterized by an irregular radiographic enlargement of the pulp chamber, with discontinuity of the normal canal space. Because the resorption process is initiated within the root canal outline of the original canal appears distorted. The enlarged canal space appeared radio graphically to be obliterated by a fuzzy-appearing material of mild to moderate radio density. This form of resorption is typically asymptomatic to be caused by a low grade inflammation of the pulpal tissues such as chronic irreversible pulpitis or partial necrosis.

Resorption of the intraradicular dentin is accompanied by subsequent deposition of a metaplastic hard tissue that resembles bone or cementum instead of dentin. Variant of internal root canal replacement resorption previously been reported as "*internal tunnelling resorption*". This entity is usually found in the coronal portion of root fractures but might also be seen after luxation injuries. The resorption process tunnels into the dentin adjacent to the root canal, with concomitant deposition of bonelike tissues in some regions. These bone-like tissues have the appearance of cancellous bone instead of compact bone.[4]

Different hypotheses have been proposed regarding the origin of the metaplastic hard tissues that are formed within the canal space. The first hypotheses suggest that the metaplastic tissues are produced by postnatal dental pulp stem cells present in the apical, vital part of the root canal as a reparative response to the resorptive insult. This is a analogous to the formation of tertiary reparative dentin by odontoblasts like cells after the death of the primary odontoblasts.

The second hypotheses propose that both the granulation tissues and metaplastic hard tissues are of nonpulpal origin. Those tissues might be derived from cells that transmigrated from the vascular compartments or originated from the periodontium the pulpal tissues are replaced by periodontium like connective tissues.

Causes of Root Resorption: [3, 12]

1. **Orthodontic Treatment**: Excessive orthodontic forces at the cervical region of the tooth might result in tissue necrosis adjacent to exposed root dentin. This might result in mononuclear precursor cells being stimulated to differentiate into odontoclasts, which are attracted to and resorb the exposed root dentin.
2. **Trauma**: ECR is a recognized complication of luxation and avulsion injuries. Dental trauma

might cause developmental defects in the cervical region on the unerupted permanent successor teeth as a result of direct trauma of the root apices on the unerupted successor. The use of splints (especially interdental wiring) might also potentially damage the cement enamel junction and therefore predispose to ECR.

3. **Intracoronal Bleaching**: Hydrogen Peroxide might denature dentin and provoke an immunologic response. In addition, the pH at the root surface of teeth is reduced to about 6.5 by intracoronal placement of a "walking bleach" paste. This slightly acidic environment is known to enhance osteoclastic activity, which might result in ECR Resorptive process might be related to injury to the periodontium from the heat lamp or bleaching tool used, rubber dam clamp placement during root canal treatment causing damage to the root surface.

4. **Surgery**: Surgical procedures removal of adjacent partially or fully erupted third molars or supernumerary teeth, transplantation of canine teeth, the surgical exposure of an unerupted canine, and periodontal surgery for root amputation.

5. **Other Factors**: Bruxism and intracoronal restorations. Developmental defects such as hypoplasia or hypo mineralization hyperoxaluria and oxalosis could cause root resorption. This is due to an increased concentration of oxalates in blood caused by kidney failure, resulting in precipitation of crystals in hard tissues, which the author claims could initiate the root resorptive process.

Molecular aspects of Root Resorption: [8,12]

Osteoclasts are multinuclear cells responsible for resorption of bone; while odontoblasts are corresponding cells resorbing dental hard tissues. Mononuclear odontoclasts can also actively resorb dental hard tissue, although during progressive resorption most cells have several nuclei.

Resorption of teeth results from the activation of clastic cells, RANK (receptor activator of nuclear factor *k* B) signaling network in osteoclasts. The factor which have been analyzed include a family of biologically related tumor necrosis factor (TNF), tumor necrosis factor receptor (TNFR)/TNF-like proteins: osteoprotegerin (OPG), RANK and RANK ligand (RANKL).

PTH or PTHrP bind to osteoblasts, bone forming cells, which increases their expression of 'Receptor Activator of Nuclear Factor *k* B Ligand,' (RANKL), which can be bind to the RANK receptor of osteoclasts precursor cells, the latter become active osteoclasts through cell fusion. Osteoprotegerin (OPG) is a glycoprotein that is a secreted member of the tumor necrosis factor (TNF) receptor super family and has a variety of biological functions including the regulation of bone turnover. OPG is a potent inhibitor of osteoclastic bone resorption by competitively inhibiting the

association of the OPG ligand with the RANK receptor on osteoclasts and osteoclasts precursor's preosteoclast-like cells (POC) in the presence of conditioned medium of osteoblastic cells. TNF-alpha also aided differentiation of hematopoietic progenitor cells into POCs Extremely low levels of TNF-alpha increased the level of mRNA for calcitonin receptor and cathepsin-K of the POC cell surface-associated proteins (SAP) from S. aureus are potent stimulators of bone resorption. Bone resorption was due to proteins and not the result of contamination with lipoteichoic acid or muramyl dipeptide from the peptidoglycan. Damage to the organic sheath, predentin and odontobiasts cells covering mineralized dentine inside the root canal must occur to expose the mineralized tissue to pulpal cells with resorbing potential that internal root resorption can be divided into two different types: Transient resorption and progressive internal root resorption.

Diagnosis: [7]

As its name suggests, ECR is usually found at the cervical region of the tooth. This discoloration is a result of the highly vascular granulation (resorptive) tissue within the tooth become visible through the thinned out (resorbed) dentin and translucent overlying enamel differentiate ECR from sub gingival caries, which will feel sticky on probing and does not present with the pink spot. The base of an ECR defect will feel hard and also result in a scraping sound when probed. Probing the ECR defect and/or the associated periodontal pocket will cause profuse bleeding of the underlying highly vascular resorptive tissue. The edges of the cavity usually appear sharp and narrow. Teeth with ECR will respond positively to sensitivity testing because the pulp only becomes involved in very advanced cases of ECR.

The use of parallax radiographic techniques is advocated for differentiating internal from external resorption defects. A second radiograph taken at a different angle often confirms the nature of the resorptive lesion. Radiologically, internal (root canal) replacement resorption presents as a cloudy, mottled, radiopaque lesion with irregular margins.

Use of Cone Beam CT (CBCT) is very helpful in accurately diagnosing the condition.

Treatment: [13]

Treatment directed at avoiding or minimizing the severity of the initial inflammatory response.

1. **Prevention**: In athletics, the mouth guard is a proven protective device against traumatic damage to the teeth.
2. **Minimizing Additional Damage after the Injury**: Decisive and correct action taken at the site of injury is probably, the most critical step in minimizing additional attachment damage after an injury the tooth should be gently repositioned back into its original position as soon as possible if splinting is necessary it should be performed with a functional splint for 7-10 days. The extraoral dry time must be minimized by placing the tooth in an appropriate storage medium. Practical alternatives include sterile saline or saliva

(vestibule of the mouth). Water, is an unsuitable medium for the storage of avulsed teeth. However, newer specialized media such as Hank's Balanced Salt Solution (HBSS), which is a common culture medium, or Via Span™, a liver transplant medium, can be used.

3. **Pharmacological Manipulation of the Inflammatory Response**: Tetracycline has been widely used in the treatment of periodontal disease because of its sustained antimicrobial effects. Recently, tetracycline has been shown to possess anti-resorptive, as well anti-microbial, properties; specifically, it has a direct inhibitory effect on osteoclasts and collagenase. Glucocorticoids have been widely used to reduce the deleterious effects of inflammatory responses; more specifically, they have been shown to reduce osteoclastic bone resorption. Topical dexamethasonne was found to be useful while systematic usage was not. Ledermix, a drug combing tetracycline and corticosteroids may be helpful.

4. **Stimulate Cemental Healing:** The supernatant of cultured gingival fibroblasts, if used as a storage medium, might contain a number of biologically-active factors that might promote periodontal ligament regeneration and repair Emdogain ™ may be extremely beneficial in teeth with extended extra oral dry time.

5. **Slow Down 'Inevitable' Osseous Replacement**: When the periodontal ligament on the root surface is definitely destroyed and osseous replacement of the root with bone.

A. All remaining periodontal ligament debris is removed from the root by thorough curettage or with the use of acid.

B. Fluoride has been shown to effectively slow down remodelling of the root to bone and the root is soaked in fluoride for 5 min before replantation.

C. Bisphosphonates are drugs that have been found to slow down osteoporosis in post-menopausal females.

Root canal disinfection removes the stimulus for the peri-radicular inflammation and the resorption will stop. Favorable healing with cementum will occur but, if a large area of root is affected, osseous replacement may result by the mechanism described earlier. In cases where the pre-radicular inflammation is stimulated by pulpal infection, bone resorption is usually extensive.[6]

1. Internal Approach:

A. Maintain the vitality of the pulp. If the pulp is vital, the canal will be free of bacteria and, thus, this type of external inflammatory root resorption will not occur. In severe injuries where vitality has been lost it is possible, under some circumstances, to promote revascularization of the pulp space. Revascularization is possible in

young teeth with incompletely formed apices (1.1 mm wide radiographically) if they are replaced close to their original position soon after the injury. Revascularization after a traumatic dental injury is a race between the new tissue replacing the ischemically necrotic pulp and the bacteria moving into the unprotected pulp space.

B. Prevent root canal infection by root canal treatment at 7-10 days. In teeth with closed apices, revascularization cannot occur. These teeth should be endodontically treated within 7-10 days of the injury before the ischemically necrosed pulp becomes infected, a creamy mix of calcium hydroxide is placed. Calcium hydroxide can be applied long- term (upto 6 months) to ensure periodontal health prior to filling the root canal with gutta-percha.

C. The elimination of pulp space infection. When root canal treatment is initiated later than 10 days after the accident or if active external inflammatory resorption is observed, the preferred antibacterial protocol consists of a chemo mechanical preparation, followed by long-term dressing with densely packed calcium hydroxide.

In situations when the root canal wall has been perforated, Mineral Trioxide Aggregate (MTA) or BioDentin ™ should be considered for repair. A hybrid technique might also be used to obturate canals; the canal apical to the resorption defect is obturated with gutta-percha, and then the resorption defect and associated perforation are sealed with MTA or BioDentin.

2. External Approach:

A. When the resorptive defect extends apically, remove the entire root cementum surface adjacent to the granulomatous tissue in the dentin. The defect and denuded root surface is covered with a spacer (freeze-dried bone), and the entire area covered with a Gore-Tex ™ membrane.

B. Force eruption entrance of the defect into the root can now easily be found and the defect cleaned and restored. Reshaping of the ‘raised’ bony contour then allows an ideal architecture to remain in place.

C. Forced eruption/re-intrusion: After the repair is complete the tooth can be orthodontically moved into its original position.

D. Intentional replantation: It is the last resort to manage the tooth failing which extraction is required.

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FIGURES:

Figure 1

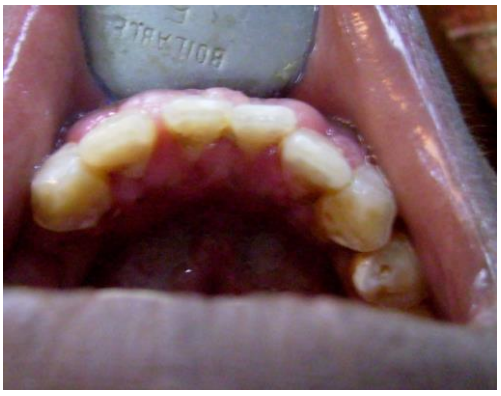

Figure 2

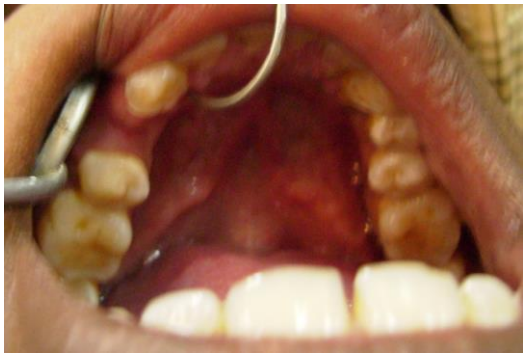

Figure 3

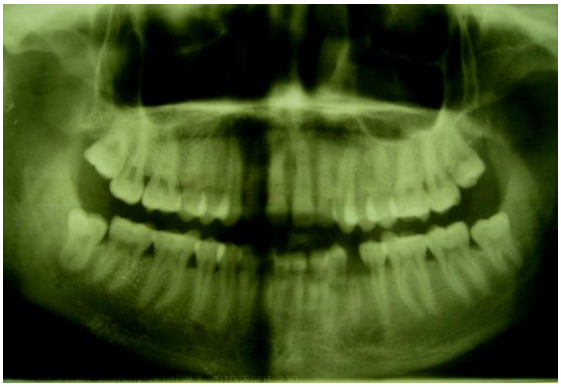

Figure 7

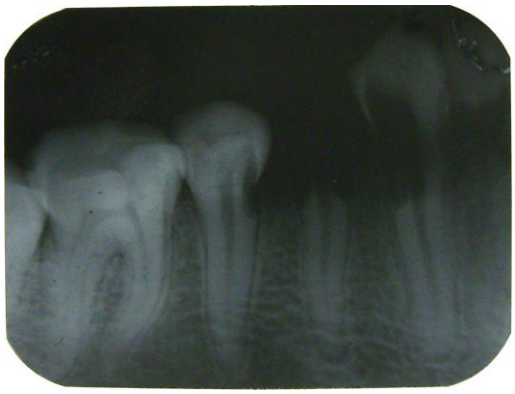

Figure 4

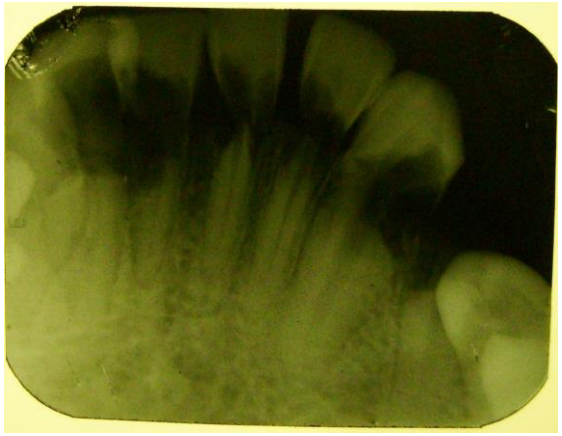

Figure 5

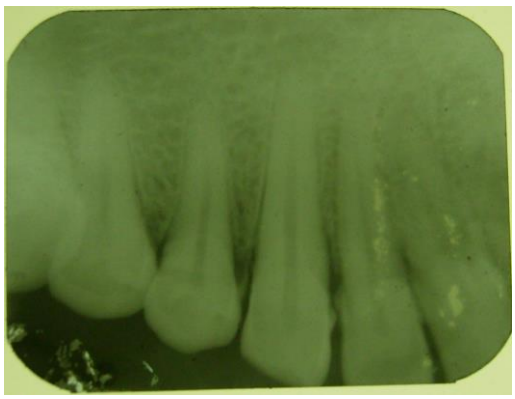

Figure 6