ABSTRACT: Internal resorption is an unusual condition and is often seen in teeth with past history of injury. It is usually asymptomatic and has a classic clinical sign “pink spot” in the crown. It’s a chance discovery on a routine radiographic examination. Prognosis improves with early diagnosis and treatment of the condition.

INTRODUCTION

According to the Glossary of the American Association of Endodontists, resorption is defined as a condition associated with either a physiologic or a pathologic process resulting in the loss of dentin, cementum, or bone. Internal resorption is a rare, pathological condition which starts in the pulpal space and spreads to the surrounding dentin. This spread is thought to begin from chronic inflammation of the coronal pulp caused by continuous bacterial stimulation. Histologically the area involved consists of multinucleated giant cells, osteoclasts, odontoblasts and a cascade of numerous other enzymes. The condition is more frequently observed in male than in female subjects. Although Internal Resorption is a relatively unusual condition, a higher frequency of the condition has been related with teeth that had undergone specific treatment procedures such as auto-transplantation. Maxillary incisors are the most commonly affected teeth. Radiographically, the lesion appears as uniform, round to oval radiolucent enlargement of the pulp space. The margins are smooth and clearly defined with distortion of the original root canal outline. For internal resorption to take place, vital pulp tissue is required. Therefore non-surgical root canal therapy is the treatment of choice to arrest the destruction process.

CLASSIFICATION

Andreasen classification remains the most widely accepted classification:

Classification of tooth resorption proposed by Lindskog subdivides resorption into 3 broad groups namely:

1. Trauma induced tooth resorption
2. Infection induced tooth resorption
3. Hyperplastic invasive tooth resorption
**Etiology**

It has been documented that the process is initiated by a variety of stimuli such as trauma, pulpotomy, extreme heat produced during cutting of dentin, chronic inflammation of the pulp following caries perpetuated by bacterial factors, cracked tooth, tooth transplantation, and orthodontic treatment.  

The literature also cites the association of herpes zoster with resorption and the degeneration of odontoblast due to systemic viral infection. Solomon et al. reported a case of internal resorption affecting 21 and 23, with a history of herpes zoster affecting the maxillary branch of the left trigeminal nerve. The effect on the dental pulp could be possibly explained by the virus being attracted to the nerve endings in the pulp. Ramchandani and Mellor and Wadden have also accounted cases of internal resorption linked with herpes zoster infection. Genetic factors have also been implicated in the development of internal resorption. The link between interleukin-1 (IL-1) gene polymorphism and root resorption has been reported in a study of monozygotic twins.

**Pathophysiology**

*See Figures 1 and 2.*

Internal resorption usually occurs as a result of a continuous chronic inflammatory process. Progress of internal resorption is dependent on two things: presence of vital pulp tissue at/below the resorption area and partially or completely necrotic pulp, coronal to the site of resorption, thereby allowing a constant entry of microorganisms and its antigens into the root canal. Microbial stimulus is an essential factor for the persistence of resorption.
The extent of progression is also determined by the intensity of the stimuli and inflammatory process. The origin of clastic cells is related to the viable blood supply and the necrotic tissue acts as a stimulus for the formation of these cells. This probably explains as to why IR is stated as a rare occurrence compared to external root resorption. The vascular changes in the pulp produce hyperaemia, causing an increased oxygen tension resulting in low pH levels, thus attracting numerous macrophages to the site, thereby piloting the onset of resorptive process. The connective tissue, following the resorptive activity, may undergo metaplasia to form granulation tissue. Predominance of a progressing infection causes necrosis of the entire pulp tissue and limits the resorptive process and this acts as a protective mechanism preventing its progression. The presence of a collateral blood supply through an accessory canal from the periodontal ligament to the resorption site can add to maintaining the resorptive process. Internal inflammatory root resorption in its most classical form spreads symmetrically in all directions into the dentin surrounding the pulp.

**Figure 1:** Schematic representation of pathogenesis of internal resorption.

**Figure 2:** Diagrammatic representation of pathogenesis of internal resorption.
Replacement type of internal resorption results from a low grade irritation of pulpal tissues such as chronic irreversible pulpitis or partial necrosis usually localized to a small area in the root canal. This associated low grade chronic infection produces more of a reactive lesion bringing about deposition of metaplastic tissue resembling bone or cementum. Wedenberg and Zetterqvist examined cases of progressive internal resorption and observed that the normal pulp tissue was replaced by a periodontal-like connective tissue with osteogenic potential. Furthermore, the process appeared to alternate between resorption of dentin and apposition of mineralized tissue, the extent of the former being related to the intensity of stimuli. Transient apical resorption may follow luxation injuries and this can be viewed through serial X-rays showing a reduction in radiolucency over a period of a few months. It is an affirmative response, as the traumatized pulp heals rapidly aided by the presence of a viable vascular network. An associated colour change may evolve due to intrapulpal haemorrhage and might resolve instinctively upon the revascularization of the coronal pulp chamber. As this is a transient process, the internally resorbed apex will resolve unevenly.

**DIAGNOSIS**

Various diagnostic tools used for detection of internal resorption are:

- Visual examination based on changed color in tooth crown
- Radiographic diagnosis
- Conventional and cone beam computed tomography
- Light microscopy
- Electron microscopy

Teeth in which resorptive process reaches cervical area of the crown may have a pinkish color, known as ‘pink tooth’ resulting from granulation tissue ingrowth.

A radiograph of the affected tooth usually shows an oval enlargement (ballooning out) of the root canal space. The pulp chamber and canal cannot be followed throughout the lesion. Radiograph performed at different angulation confirms that the resorptive lacunae is a continuation of the distorted border of the root canal.

Cone beam computed tomography (CBCT) is a relatively new three dimensional imaging technique requiring a significantly lower radiation dose than conventional computed tomography. With traditional computed tomography, a narrow fan shaped X-ray beam makes a series of rotations around the patient’s head as they are incrementally moved through the machine. The raw data from each rotation is then reconstructed to produce tomographic images. CBCT differs from conventional computed tomography imaging in that the whole volume of data is acquired in the course of a single sweep of the scanner.

Axial, transverse, and tangent slices, number of root surfaces, and actual root resorption extension can be analyzed.

Light microscope shows different levels of inflammation of the pulp tissue with infiltration of predominant lymphocytes, macrophages and some leukocytes, dilated blood vessels and multinucleated dentinoclasts in resorative lacunae on the pulpal-dentin surface.

Electron microscope shows the pulpal-dentin wall without odontoblasts. Dentinoclasts, large in number, have size of 50μm and with numerous filipods are turned towards dentin surface and attached to it.

**DIFFERENTIAL DIAGNOSIS**

There are certain key factors that may be looked into for the diagnosis of internal resorption.

**Etiology.** History of trauma, other etiological factors like crown preparation or pulpotomy, and positive pulp sensitivity test should be ruled out for its diagnosis.

**Sensitivity Testing.** A negative response to sensitivity testing is obtained, as the coronal pulp has often been removed or is necrotic and the active resoring cells are more apical in the canal.

**Pink Spot.** The pink tooth caused by the granulation tissue undermining the enamel can also be a feature of subepithelial external inflammatory root resorption/cervical resorption (progressive external root resorption, of inflammatory origin, occurring immediately below the epithelial attachment of the tooth), which must be ruled out before the diagnosis of internal resorption is established.

**Radiology.** Progressive internal resorption or those with perforations of root can be distinguished from external resorption by varied radiographic techniques. In teeth with internal resorption, the radiolucent lesion “moves” with the canal when the radiographs are taken at different angles, while in external resorption the radiolucent lesion “moves” outside of the canal. Internal resorption has a uniform enlargement of the canal space with regular bone structure, whereas external resorption has an irregular border with an alteration in the adjacent bone, and the canal can often be visualized through the radiolucent area.

**MANAGEMENT OF RESORPTION**

**Surface Resorption:**

- Monitor radiographically.
- Endodontic treatment only if signs of infection.

**Transient apical internal resorption**

- Monitor radiographically.
- Endodontic treatment only if signs of infection or ongoing discoloration.

**Pressure**

- Remove cause e.g. unerupted cusp, neoplasm

**Orthodontic**

- Should stabilize on completion of orthodontic treatment
Replacement resorption

- Mature tooth in normal occlusion; leave and monitor for ultimate implant replacement. In infra-occlusion; in selective cases surgical reposition and treat root surface with emdogain.
- Immature tooth in infraocclusion; in selected cases surgically reposition and treat root surface with emdogain; or decoronate and submerge. Implant therapy, if necessary, when alveolar growth completed.

Internal Inflammatory (infective) root resorption

1. Apical
- Endodontic treatment to the level of resorption.
- Long term calcium hydroxide dressing before placement of root filling.

2. Interradicular
- Endodontic treatment and root canal filling (hot GP technique, Obtura etc)

External inflammatory root resorption

- Endodontic treatment and intracanal medication with either Ledermix paste followed by long term calcium hydroxide or calcium hydroxide alone. Root fill when resorption controlled.
- Prevention: following replantation of mature tooth pulp extripation and ledermix paste dressing as soon as possible.

Communicating internal external inflammatory resorption

- Endodontic treatment to resorptive defect. Induce calcification by use calcium hydroxide alone or following careful topical application of 90% trichloracetic acid. ProRoot MTA may also be used.

Internal replacement resorption

- Pulpectomy and root filling

DISCUSSION

The reciprocal activity between the newly formed granular tissue and dentinoclasts initiates and progresses the resorption process inside the endodontic space which could be compared to pathogenetic changes in the periapical region. The early diagnosis and therapy is very important in order to stop the resorption process. The success or failure of therapy should be followed clinically and by radiographic control. Naturally, if the resorption is stopped actually is not progressing, we believe that our treatment is successful. We saved a tooth and the objective of our therapy has been accomplished. The outcome of treatment of teeth with internal root resorption depends primarily on the size of the lesion. Large lesions cause a reduction in the resistance of the tooth to shear forces that may lead to tooth fracture. Therefore, it is imperative to initiate endodontic treatment as soon as possible to arrest the progression of the resorative process and to prevent root or cervical crown fracture.

CONCLUSION

Internal resorption is a rare insidious process with the majority of the cases being idiopathic. The detection of internal resorption is easy only in simple cases, while others require high quality periapical radiographs or advanced diagnostic techniques like dental CT scans for its diagnosis. Proper patient history, early diagnosis, and appropriate treatment at the correct time prevent tooth loss. The outcome of the treatment is good and depends on the amount of remaining dentin wall thickness. Care should be taken to distinguish internal resorption from other types of tooth resorption for appropriate management.

REFERENCE


