Review Article

INTERNAL RESORPTION: A REVIEW ARTICLE

UNDER PEER REVIEW

ABSTRACT:

Tooth Resorption is classified majorly into internal and external resorption as given by Andreason. The lesion puts the dentist in a predicament for treatment of the lesion, as it is often discovered accidentally, decreasing the prognosis as it is found later. Since the lesion usually affects the esthetically important region, it places the dentist in a quandary on what to do. Various factors affecting the pathophysiology of the lesion are the inflammatory process, stimulation of clastic cells. The etiology for the lesion is trauma in the majority of the cases. However, it may also be caused by excessive heat production during cavity preparation and in untreated carious lesions. Clinically it appears to be typically asymptomatic, although when it is actively progressing, the lesion shows symptoms of pulpitis as it mainly occurs due to chronic inflammation of the pulp. Histopathologically the lesion shows connective tissue of pulp which is inflamed and infiltrated by neutrophils, lymphocytes, plasma cells, and macrophages. The granulation tissue dominates the lesion. Radiographically the lesion occurs as oval to round radiolucency in the crown. The treatment performed is root canal treatment (RCT), as terminating the progressing resorptive process is the goal, and by doing so, further damage to the tooth can be prevented. Internal resorption will result in loss of tooth unless it is treated.

KEYWORDS: internal resorption, pink tooth, inflammatory resorption, tooth resorption, external resorption.

INTRODUCTION:

The term *internal resorption* is defined as a process that results in the loss of cementum and dentin or bone. Once the loss of dentin has occurred, the internal resorption area appears to be a *pink spot* due to an increased number of blood vessels in the area. There are two ways in which this lesion manifests, which can be identified in the histopathology of the lesions. One type is destructive, where there is the destruction of mineralized tissues of the tooth, and the other is reparative, in which the lesion is substituted by cementum and dentin-like tissue in the adjoining area of the resorption. The pink tooth is produced by granulation tissue ingrowth when the resorption process

reaches the cervical portion of the crown. This lesion is usually clinically asymptomatic, with maxillary incisors being the most affected teeth in the jaw. [(1)] When diagnosing internal resorption, it is obligatory to use a radiograph. Consequently, the radiograph reveals oval radiolucency with smooth and precise margins [(2)]. The oval radiolucency in the pulp space indicates enlargement of the pulpal space. The lesion can also be detected using clinical examination based on tooth-color changes, conventional radiography, cone-beam computed tomography, light microscopy, and electron microscopy. **[(3)]** The treatment of choice is RCT, as terminating the progressing resorptive process is the goal, and by doing so, further damage to the tooth can be prevented [(4).] The treatment also helps remove the granulation tissue and necrotic pulp. The tooth affected with internal resorption has a weaker tooth structure (due to loss of hard tissues dentin, cementum). Hence, care must be taken to keep the shaping of the root canal as conservatively as possible during the treatment. As there is an increased number of blood vessels in the area, the bleeding might affect visibility during the procedure. Internal resorption will result in loss of tooth unless it is treated.

Tooth Resorption is classified majorly into internal and external resorption as given by *Andreason* (MODIFIED):

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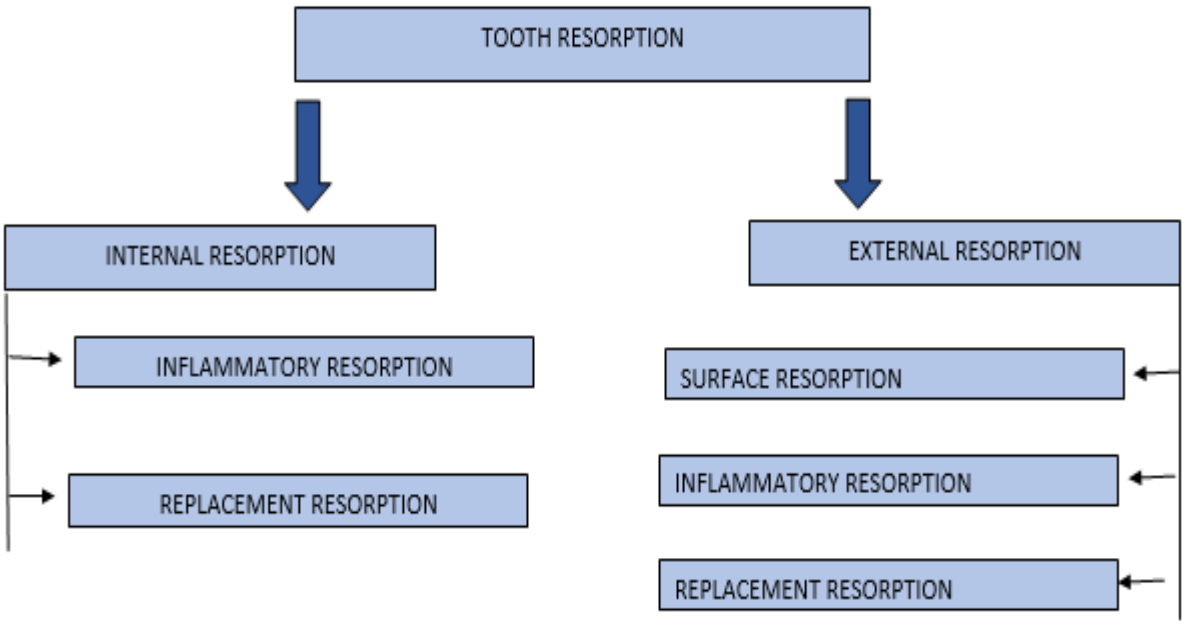

Fig 1. Andreasen classification of tooth resorption; GS Heithersay Australian Dental Journal Supplement 2007;52

ETIOLOGY

Internal resorption is initiated due to trauma to the tooth physically or thermally (excessive heat produced during access cavity preparation). In most cases, it was also observed that untreated

carious lesions might also result in internal resorption of the tooth due to chronic inflammation of the pulp.

PATHOPHYSIOLOGY

The viable blood supply present in the pulp and the necrotic debris provides stimulus, which stimulates clastic cells' formation. Various factors are affecting this, such as the inflammatory process and the intensity of the stimulus. [10]. The onset of resorption is caused by hyperemia in the pulp and low ph, leading to the accumulation of numerous macrophages (11). Caries, when extended deep, it causes damage to the predentin leading to its loss, odontoblastic degeneration leading to inflammation and necrosis of pulp which then leads to activation of odontoblasts, consequently causing internal resorption, which is then replaced by granulation tissue (Fig 2). Granulation tissue is formed when the connective tissue undergoes metaplasia after resorptive activity (5). The entire pulp tissue undergoes necrosis due to continuously progressing infection, limiting the process of resorption, thereby preventing its progression (17). The ongoing process of resorption can be maintained by collateral blood supply present at the site of resorption emerging from the periodontal ligament via the accessory canals (6). The lesion typically spreads symmetrically and radially into the surrounding dentin. It has been found that its development can only be initiated if the vital pulpal tissue is substituted by a connective tissue analogous to the periodontium (6). This process of dissolution of tooth structure involves two mechanisms, the stimulating factor and the factor that provides a nidus for the process to continue (5). When the surface of the root is separated from the blast cell layer that protects it, which may have occurred due to trauma, it provides a trigger that promotes the resorptive process. The infection is present in the canal caused by the continuously acting mechanical forces on the tooth. The tooth with apical periodontitis, which may be produced following the death of pulpal tissue, may develop apical root resorption (19).

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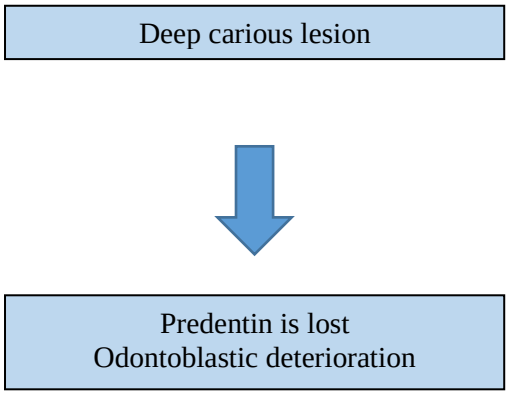

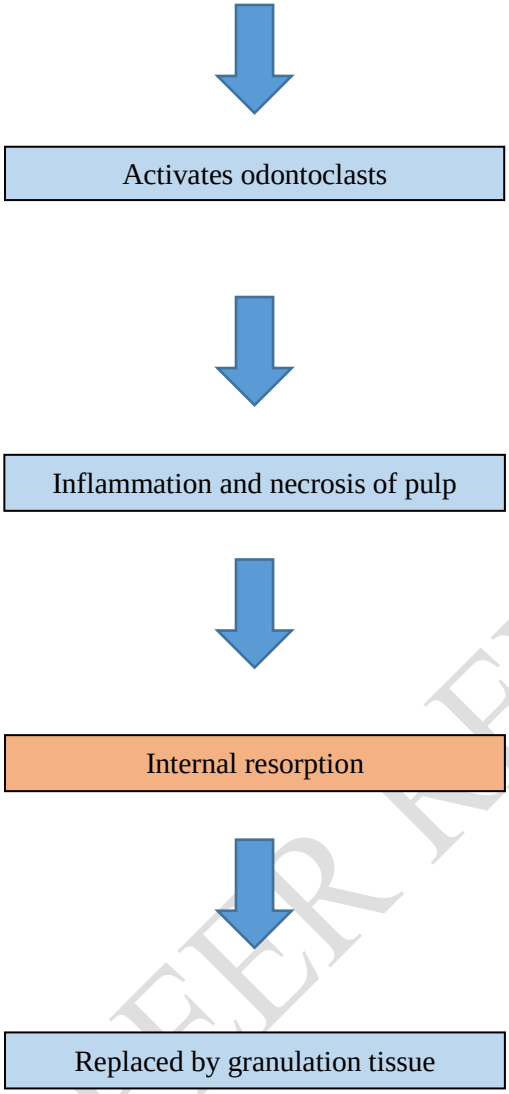

Fig: 2. Pathogenesis of Internal Resorption

CLINICAL FEATURES:

Tooth affected with Internal resorption appears reddish/pinkish caused by the presence of granulation tissue which then changes to dark grey/grey once the pulp becomes necrotic [6,16]. When the lesion is actively progressing, it shows symptoms of pulpitis as it mainly occurs due to chronic inflammation of the pulp. Since the pulp is vital initially, the vitality test gives a positive result till the pulp becomes necrotic and perforation of the canal has taken place [6]. After the pulp becomes necrotic, it will result in apical periodontitis, which develops into periodontal abscess and formation of the sinus tract due to progressing inflammatory process. Extensive, irreversible tissue loss occurs due to root perforation, and an external connection between a root canal and the surrounding tissue might develop [17].

HISTOPATHOLOGY:

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The connective tissue in the pulp is inflamed and is infiltrated by neutrophils, lymphocytes, plasma cells, and macrophages. These macrophages and odontoclasts are connected to the mineralized dentinal surface and resorptive bays [18]. The granulation tissue dominates the lesion. Under microscopy, bacteria are found in abundance in the dentinal tubules and coronal gangrenous zone of the root canal, which communicates with the necrotic and granulation tissue experiencing resorption [19]. There are two ways in which this lesion manifests, which can be identified in the histopathology of the lesions. One type is destructive, where there is the destruction of mineralized tissues of the tooth, and the other is reparative, in which the lesion is substituted by cementum and dentin-like tissue in the adjoining area of the resorption [11].

RADIOGRAPHIC FEATURES:

Radiographically the lesion occurs as oval to round radiolucency in the crown whose margins are precise, smooth with deformation of the initial profile of the root canals [3]. The lesion goes undetected in cases where it rapidly spreads in the root canal, causing the death of the pulp and thereby terminating the resorptive process. The lesion starts in different ways in single-rooted and multirooted teeth. Single-rooted teeth originate symmetrically from the coronal pulp [6]. In contrast, it originates from one part of the pulp in the multirooted teeth and then extends to the adjacent dentin. [12,13] Internal resorption is differentiated from external resorption in a way where if the lesion (radiolucency) shifts towards the canal after taking intraoral periapical radiograph from two separate angles, it is internal resorption, and if not, then otherwise [24].

DIAGNOSIS

After correlating clinical investigation discoveries and intraoral radiographs, the diagnosis should be made [19]. The lesion must be thoroughly examined to check if it is perforating or non-perforating type of lesion. An iatrogenic perforation is consistently identified as a large amount of bleeding following the injury. This perforation is often directly visible when the coronal portion of the tooth is perforated. However, sometimes more apical perforations within the root canal reveal bleeding when the paper point is inserted into the canal. Unless local anesthesia is administered, an abrupt, unexpected pain during treatment can indicate a perforation [26]. Apex Locator is handy for detecting perforation. Inserting the file in the perforation will display a zero-reading suggesting connection with the periodontal ligament. Operating microscopes have become infamous for detecting perforations. It is easy to visualize the location and extent of perforations with bright operating light and magnification [26]. The lesion takes place in both the dentition and appears to be clinically, etiologically, and morphologically similar in both, apart from the rate at which the lesion

progresses. Internal resorption is quicker in milk teeth than permanent dentition [20]. Bacteria were found in abundance in teeth in which the lesion was progressing quite rapidly [19]. The cells causing resorption in the tooth and other parts of the body show similarity in showing the robust activity of an enzyme termed tartrate-resistant phosphatase [21]. The changes in vascularity of pulp can be effectively detected by MRI [22]. On MRI, the control teeth and the affected teeth differ in the form of tooth, and the intensity of the signals received from the pulp at the site of resorption. The lesions are hyperintense in short- tau inversion recovery and T1- T2 weighted sequences [25]. Positive signals were received in diffusion-weighted whole-body imaging with background body signal suppression. The intensity of the signal was more significant in the tooth with inflammatory pulp than the control tooth [25]. However, apical shortenings, lateral or cervical root gaps, widening of the root canal, and external root radiolucencies in the early stages, when they are small or because of the limitations of this two-dimensional method, are typically not detectable on x-rays [3]. CBCT is another optional diagnostic instrument for the early detection of the lesion. Early detection can lead to timely intervention and better treatment outcomes. the extension of resorption is identified by analyzing all dimensions of the lesion can be obtained with CBCT [29]. Consequently, using CBCT we can ascertain whether the lesion is in a state of arrest, repair, or progression. A seminal benefit of this imaging modality is the ability to record comparable images with significantly lower radiation doses than multi-detector CT. Studies have found that CBCT is more error-less and precise when compared to periapical x-rays [30].

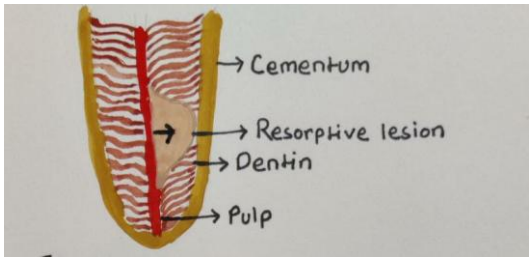

Fig 3a- Internal Resorption

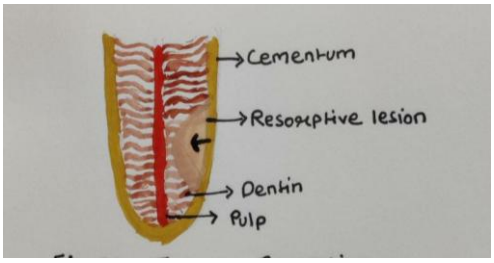

Fig 3b- External Resorption

Internal resorption differs from external resorption in a way that internal resorption originates from the pulp chamber and appears to be progressing towards cementum (Fig 3a). However, in external resorption, the lesion originates from the cementum and progresses towards the pulp chamber (Fig 3b). In internal resorption the oval and well-defined radiolucency is present and radiographically the root canal cannot be seen in the area of defect. In contrary to external resorption in which the root canal is intact and the radiolucency is irregular [6]. In cbct, we can differentiate between the lesions by locating the site and the direction of progression of the lesion as done in radiographs. In case of external resorption, the radiolucency observed tends to shift in different angles horizontally

in contrary to the case of internal resorption where the radiolucency tends to be close to the root canal [31].

TREATMENT:

Three treatment modalities are possible for this lesion. Until the lesion is symptomatic, no treatment may be done. If the lesion becomes symptomatic, then extraction can be done. The tooth can also be immediately extracted if the prognosis after restoration is low. Several criteria must be considered during the decision-making process, which include: The patient's age, medical history, the position of the teeth, occlusion, the place where the resorption takes place. The presence or absence of root perforations, as well as the extent to which they are present, the remaining root hard tissue's resistance or weakness, the ability to perform a restorative procedure on the tooth in question based on the data gathered during the clinical exam CBCT are also essential factors to be considered for the same. There are a few things to consider, like the absence of therapeutic abstinence, monitoring symptoms of an infectious disease plays an important role. There are three possibilities for orthograde root canal therapy depending on whether perforation is present or not. If the lesion is non-perforated to the radicular wall, the root canal may be filled with gutta-percha. If the lesion appears to be of mixed type, the root canals can still be filled with gutta-percha or MTA. [11] The perforated area is then filled with bioactive material like MTA or Biodentine in lesions with short root length. [7] However, the cleaning and shaping of the root canal are challenging due to complex irregularities present in the root canal system. The long-term success of the endodontic treatment depends on the presence of bacteria and organic debris after endodontic treatment [8]. Calcium hydroxide causes necrosis of the remaining pulp and also aids in controlling excessive bleeding. In addition to this, it also makes the necrotic pulp more soluble to sodium hypochlorite [9]. Calcium hydroxide can be used as a sealant in the canals for 1-2 weeks in cases where perforation has not taken place. At the next appointment, the removal of residual tissue can be done using irrigation and instrumentation. Calcium-silicate-based materials can also be used to seal the resorptive cavity with perforating defects as it forms a barrier between the obturating material and the resorptive cavity. Calcium hydroxide as an intracanal medicament is also done, which assists in dissolving any leftover granulation tissue before completing inter-radicular treatment, which is followed by gutta-percha canal obturation. Recently there are new advances taking place in the field of regenerative endodontics which can also be used alternatively to treat a perforated type of lesions [14,15]. To ensure that the root canal is effectively cleaned and debrided, curettage is done after resection of the root end, and it is done up to the area where the perforation has extended. In cases where the lesion may be perforating from the apical to lingual side, the expansion of the access opening on the lingual side is not always accepted; however, if required, it can be done. Since the process of dissolution of the mineralized tooth structure makes the tooth weaker in terms of strength, a post

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and core restoration long with composite may be used to regain the strength of the tooth so that it resists fracturing or chipping under occlusal forces [23].

CONCLUSION

Internal resorption is a rare insidious and usually asymptomatic condition. It shows more affliction towards females and maxillary incisors as they are easily more prone to trauma. The diagnosis of the lesion can be made visually by the color change seen in the tooth crown. Radiographically the lesion can appear as oval to round radiolucency. Internal resorption is differentiated from external resorption in a way where if the lesion (radiolucency) shifts towards the canal after taking intraoral periapical radiograph from two separate angles, it is internal resorption, and if not, then otherwise. For the management of internal resorption, RCT still stands to be the therapy of choice as it eradicates the necrotic pulp and necrotic tissue, hence terminating the resorptive activity. The onset of resorption is caused by hyperemia in the pulp and low ph, leading to the accumulation of numerous macrophages. The liveliness of the periodontal membrane is an essential factor for the development of the lesion. It has been noted that the good vitality of the periodontal ligament(pdl) will lead to a lesser extent of resorption as compared to the teeth with poor vitality of (pdl). When a tooth gets traumatized and advances to avulsion of the tooth, if during reimplantation of the tooth there is damage to pdl, it will lead to a massive inflammatory response, further increasing the chances of resorption. The tooth with apical periodontitis, which may be produced following the death of pulpal tissue, may develop apical root resorption.

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