Root resorption

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Abstract
Over the years root resorption has been a complicated aspect of dentistry with regard to the aetiology, classification and treatment options This review of literature has been put forward so as to assist the dentist in ensuring the longevity and retention of affected teeth with the aim of reducing costs for prosthetic treatment.

Introduction

Root resorption has long been identified in the dental literature and was first documented in the 16th Century dental text by Artzney Buchlein[1]. It is a condition associated with either a physiologic or pathologic process resulting in a loss of dentine, cementum or bone[2]. Frequently, this pathologic condition is difficult to predict, diagnose and treat[3]. In the various dental disciplines, it has been traditional to consider root resorption as a unique phenomenon pertaining only to a particular treatment procedure[4]. Being a serious complication of treatment and often resulting in failure of attempted procedures, root resorption in these situations is mediated by an unwanted and uncontrolled activity of hard tissue resorbing cells[5]. Frank and Torabinejad[6] say that resorption can also occur as a result of idiopathic factors.

The mineralised tissues of the permanent teeth are not normally resorbed. They are protected in the root canal by the predentine and odontoblasts and on the root surface by the precementum and cementoblasts[7]. If the predentine or precementum becomes mineralised, or mechanically damaged or scraped off, multinucleated cells will colonize the mineralised or denuded surfaces and resorption will ensue[8]. The process of root resorption involves an elaborate interaction among inflammatory cells, resorbing cells and hard tissue structures[3]. Root resorption has long been a challenging problem for dental practitioners. The aim of this paper is to review root resorption with emphasis on the aetiology, classification and treatment options.

Aetiology of Resorption

The etiological factors involved in root resorption include mechanical pressure, inflammation and presence of denatured host tissues and unknown (idiopathic) causes[6]. Root resorption occurs frequently in traumatised teeth and in teeth that have undergone orthodontic and periodontal treatment, but is also seen in other teeth, apparently as a result of wear and tear[9]. Infective necrotic debris and toxic products move through the dentinal tubules to the root surface to sustain the inflammatory resorptive process[10]. Bleaching of discoloured teeth has been associated as causing root resorption. Concentrated hydrogen peroxide may penetrate dentinal tubules and lead to vascular emboli, cell damage, inflammation and resorption of dental hard tissues[11]. Tumours that produce root resorption are most frequently those in which growth and expansion are not rapid, such as cysts, ameloblastomas, giant cell tumours, and fibro-osseous lesions. Rapidly growing and malignant tumours produce much less resorption[9]. Root resorption is also seen in conjunction with systemic diseases with dento-alveolar manifestations[9].

Mechanism of Resorption

Root resorption, which is initiated by a denuded area of the root surface, may be sustained by mechanical irritation, increased pressure in the tissue or infection of the root canal and tubules of the crown and root dentine[12]. The process of tooth (root) resorption involves an elaborate interaction among inflammatory cells, resorbing cells and hard tissue structures[3].

Formation of Osteoclasts.

Injuries to and irritation of bone, dentine or cementum leads to chemical changes within these tissues; the result being the formation of multinucleated giant cells referred to as clasts[13]. The clastic cell is the key cell type responsible for all hard tissue resorptive processes[14]. Osteoclasts are bone-resorbing cells derived from hemopoietic cells of the monocyte-macrophage lineage; osteoclasts have a life span of approximately two weeks. The osteoclast is a very active cell and it has the ability to move between resorbing sites. It is a multinucleated giant cell, formed from the fusion of mononuclear precursor cells that arrives at the site of resorption via the blood stream. Osteoclasts are found in tiny depressions, pits or irregular grooves, termed "Howship lacunae" in cementum, dentine and bone[13].

The resorptive process

According to Andreasen[4] tooth and bone resorption as a result of osteoclast activity can be seen as the expression of three main functions of these cells:

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A maintenance function, where osteoclasts, together with osteoblasts, during continuous activity maintain or change the structure of jaws.
A role in the defence against infection.
Its participation in repair of traumatised tooth or bone. In this function the osteoclasts act as specialised macrophages and is actively participating in the advancing front of cells involved in the healing process.

The resorptive process itself can be described as being bimodal, involving the degradation of the inorganic crystal structure of hydroxyapatite and the organic structure of collagen[15]. The activated osteoclasts produce an acidic pH (3.0 – 4.5) in their microenvironment. At pH 5.0 or lower, the solubility of hydroxyapatite increases dramatically, and resorption of hard tissues can occur[16].

Osteoclasts, the cells that resorb bone, attach themselves to the bone surface via a circular sealing zone of cytoplasm called the clear zone. The later encircles a series of inter-digitating folds of cell membrane (ruffled border) under which the resorption process occurs[17]

Dentinoclasts are morphologically similar, if not identical, to osteoclasts but do not appear to reach the size or nuclear numbers of their osseous counterparts, which may result from their differences in their respective resorption substrata. Both osteoclast and dentinoclasts appear to resorb in a similar fashion, producing large resorption pits or lacunae on the hard tissue surface as they move between sites of resorption and it can be difficult to distinguish whether the clastic cells not currently engaged in resorption are dentinoclasts or osteoclasts[17]. Bacteria are integral to the process of tooth resorption and there are two possibilities in the mechanism of bacteria-induced resorption:

1) Bacteria produce acids and proteases that destroy the bone matrix components
2) Bacteria stimulate the production of osteoclast factor, which promotes osteoclastic activity[16]

One of the mechanisms involved in the protection or modification of the resorption of the root is thought to be that cementum, being more resistant to resorption than the dentine, to some extent protects the root from resorption[18].

Classification

Tooth destruction may occur by root resorption, which is accomplished by dental pulp cells (internal resorption) or by cells of the periodontal ligament (external resorption)[19].
Root resorption is divided into internal (root canal resorption) and external resorption (root surface resorption). Each of these resorption types can be further subdivided into an active, arrested or repaired phase according to the activity of resorption[20]. Both internal and external root resorption may occur on any surface of the tooth that contacts vital tissue[21].

External Root Resorption

External root resorption is extremely common, but varies widely in clinical presentation due to an unwanted and uncontrolled imbalance in osteoclast function[6]. External resorptive lesions according to Gartner, et al[21] have the following radiographic features:

(1) The borders may be ill defined,
(2) Their distribution is not symmetrical and may occur on any external surface of the root.
(3) May have less well defined margins and show variations in radio-density that may appear striated.
(4) If the lesion is superimposed on the root canal system, it should be possible to follow the canal walls unaltered through the area of the defect.

External resorption is further classified into surface, inflammatory or replacement (ankylosis) resorption[21]

External surface root resorption

External surface resorption is a transient phenomenon in which the root surface undergoes spontaneous destruction and repair. It is found in all teeth, in varying degrees, and is likely to be a normal physiologic response [22]. It is the least destructive form of external root resorption and is a self-limiting process; hence requires no treatment[23]. The resorptive lacunae are superficial and frequently observed on non-traumatised teeth as well. Repair is occasioned by the spontaneous formation of new cementum[24]. Surface resorption is usually demonstrated radio-graphically as a rounding of the peripheral part of the fracture line and is a common finding[25].

Surface resorption is a self-limiting process occurring in small outlining areas of disrupted periodontal ligaments, which are repaired from adjacent intact parts of the periodontal ligament without significant inflammatory changes in the soft tissues[6]. Since it is now understood that the process occurs not only on the root surface, but in the root canal as well, a better term would seem to be transient root resorption[9].

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External inflammatory root resorption

Is best described as a bowl-shaped resorptive defect that penetrates dentine and it represents the most common type of external root resorption[26]. External inflammatory root resorption is maintained by an inflammation in the periodontal membrane caused by bacteria in the pulp and dentinal tubules[27]. It can occur on any part of the root, however, it is frequently subcategorised according to that part of the tooth to which it has occurred.

a) Cervical External Inflammatory Root Resorption (Also known as invasive cervical resorption) can occur following injury to the epithelial cervical attachment apparatus and to the area of the root surface just below the attachment apparatus. Single rather than multiple teeth are affected and the process tends to be slow. Also mostly seen following the use of 30% hydrogen peroxide in conjunction with bleaching of teeth[12]. The pulp is not involved until the condition is well advanced[28].

   Invasive cervical resorption is an insidious and often aggressively destructive form of external root resorption that is characterized by invasion of the cervical region of the root by fibro-vascular tissue derived from the periodontal ligament[29]. It is a relative uncommon and clinically challenging condition. A similar invasive resorption may also be noted in the crowns of erupting teeth. For these cases the term "invasive coronal resorption" seems appropriate, where as resorption of a similar nature, but situated more apical may be termed "invasive radicular resorption"[30].

   Tronstad[9] hypothesized that cervical resorption is probably initiated by injury to the cervical periodontal ligament apparatus apical to the epithelial attachment. This may be transient initially, but if bacterial irritation is superimposed, then the process is perpetuated and becomes progressive. The bacterial source was thought to be the gingival sulcus rather than the root canal. Cervical resorption is of considerable endodontic importance because by penetrating the root canal it becomes an endodontic problem though it is not a pulpal reaction nor is it maintained by pulp necrosis or root canal infection[12].
   While some teeth show external signs such as pinkish discolouration, quite often it is only detected by radiographs, though it is possible to probe a defect cervically[31]. Cervical resorption is the type of external resorption most often misdiagnosed as internal resorption[12].

   In external cervical resorption the periodontal tissues are inflamed first and may cause a vague gnawing discomfort typical of periodontal irritation[12]. Cervical resorption is usually asymptomatic because the pulp remains protected by a thin layer of predentine and dentine[10], however if the pulp subsequently becomes involved as the resorption progresses, pulpal symptoms may be superimposed[12]. Here the periodontal pain is preceded by pulpal symptoms. Gingival and periodontal inflammation may also be accompanied by gingival bleeding and bad taste[32].

b) Apical External Inflammatory Root Resorption. Regularly, all teeth with periradicular periodontitis will exhibit some degree of external inflammatory root resorption at the apex[33]. The capacity for repair of root resorption is diminished with greater severity of periodontitis[34]. Intense and progressive inflammation confined to the apex causes sufficient pressure to overcome the "resistance" of the cemental layer to resorption[35]. It is hypothesised that the excessive forces initiate inflammation either as a result of stimulation of a phagocytic process by tissue breakdown products or through some form of neurogenic inflammation[36].

Replacement Resorption (Ankylosis)

Occurs after extensive necrosis of the periodontal ligament with formation of bone on the denuded area of the root surface[26]. Dentoalveolar (replacement) ankylosis is a common complication of replanted teeth that leads to a gradual resorption of the dental hard tissues and their replacement by bone[37]

Ankylosis is the union of tooth and bone, with no intervening connective tissue, following external inflammatory resorption[38]. The development of dentoalveolar ankylosis has been described as a process in which bone trabeculae, formed on the socket wall, gradually grow across the periodontal space and fuse with the root surface. These bone trabeculae then unite and form a solid plate directly upon the intact cementum or the previously resorbed cementum or dentine[39]. If less than 20%of the root surface is involved, reversal of ankylosis may occur. If not, ankylosed teeth are incorporated in the alveolar bone and will become part of the normal remodelling process of the bone. Consequently, they will gradually resorb and be replaced by bone, hence the term

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replacement resorption[40]. Replacement resorption occurs as a "mistake" because the cells involved in the remodelling of bone are not able to distinguish between root cementum, dentine and bone. The osteoclasts will resorb the dental tissues just as readily as they resorb the bone, and the osteoblasts that are not capable of forming dentine or cementum will replace the resorbed areas of the root with bone[9]. Recent studies have indicated that this initial root resorption may be associated with an inflammatory reaction against an infected periodontal membrane of a replanted tooth or with the phagocytosis of its necrotic periodontal soft tissue[41]. The pathogenesis of replacement resorption is manifested in two ways: either permanent (progressive) replacement resorption which gradually resorbs the entire root, or transient replacement resorption in which a once-established ankylosis later disappears[40,42]. If greater than 20% of the root surface is damaged the likelihood of progressive ankylosis increases[9].

Internal Root Resorption

Internal resorption is rare in permanent teeth[23]. Probably due to the relative protection of the internal dentine wall from mechanical trauma, compared with external root surface, would account for the lower incidence of internal resorption in traumatised teeth. In contrast, pulpal infection is a common finding in such teeth[43]. Traditionally internal root resorption has been associated with a long standing, chronic inflammation in the pulp[12]. Initiation of internal resorption requires the removal of odontoblasts and predentine. Consequently, trauma may be recognised as an initiating factor of internal resorption[43]. The production of stimulation may be from potent stimulators of hard tissue resorption released by inflammation. The stimulation may be necessary for the progression of internal resorption, as macrophages have been shown to require continuous stimulation during phagocytosis[43]. The dentinal tubules have to have a special and tortuous course. They have to be open to an area of the root canal where the tissue is necrotic and infected so that micro-organisms may enter the tubules and then lead to an area of the canal with vital pulp tissue. This is a rather unlikely occurrence and probably explains why progressive internal resorption is a rarity in permanent teeth[12]

According to Wedenberg and Lindskog[40] internal resorption may be divided into:

a) Transient type – similar to external root surface resorption, which developed in the absence of pulpal infection. This type of resorption may be caused by the disappearance of odontoblasts and predentine, thus exposing dentine.

b) Progressive type – which requires continual stimulation by a bacterial inflammation and which resembles external inflammatory resorption.

Being typically asymptomatic, it is usually discovered during a routine radiographic evaluation and is often misdiagnosed as external resorption[44]. When internal resorption lacunae are seen radio graphically it is an indication for endodontic treatment. The lesion may present with a pink discolouration called a "pink tooth" caused by inflamed pulp tissue. Clinically the pulp necrosis is in the pulp chamber and usually in the root canal to a level somewhere coronally to the resorption lacunae. The resorptive area and the root canal apical to this area will contain vital tissue. In some cases the entire pulp will be necrotic. The resorptive process will then have stopped since obviously vital cells are needed for the resorption to go on[12]. Gartner, et al[19] attributed the following radiographic features to internal resorptive lesions:

(1) margins are smooth and clearly defined
(2) the distribution over the root is symmetrical but may be eccentric
(3) the radiolucency is of uniform density
(4) the pulp canal or chamber cannot be followed through the lesion
(5) the walls of the root canal system may appear to balloon out.

With internal resorption, the pulp is involved first; so pulpal symptoms may be a feature. If the pulp succumbs completely and periradicular tissues become inflamed, symptoms of periodontitis may be evident. Therefore pulpal symptoms precede periradicular symptoms.

Treatment

All resorptive lesions should not be treated as endodontic problems. It is necessary to localise the site of the resorption by means of radiographic and clinical examination and after that make a diagnosis and treatment plan[45].

Calcium hydroxide pastes are routinely used in endodontic therapy in an attempt to prevent or stop the resorptive process. Diffusion of hydroxyl ions through dentine into the periradicular tissues neutralises the acid pH of the resorptive area caused by the action of the osteoclasts. If the level of diffusion of hydroxyl ions from calcium hydroxide cements was similar to the diffusion of hydroxyl ions from calcium hydroxide pastes, an attempt could be made to treat teeth undergoing resorption by using a sealer containing calcium hydroxide during obturation of the root canal. The calcium hydroxide containing cements, although suitable for the use as root canal sealants, do not produce an alkaline pH at the root surface and thus the putative effect on root resorption is moot[46].

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Calcium Hydroxide has an alkaline pH of 12.5, making it a desirable medicament in the treatment of resorptive processes[47]. Diffusion of the hydroxyl ions from the root canal raises the pH at the surface of the root adjacent to the periodontal tissues, thereby possibly interfering with osteoclastic activity[12]. Calcium hydroxide when placed in the canal effectively kills the bacteria, and in addition will influence the local enviroment at the resorption sites on the root surface through the dentinal tubules. Due to its high pH, it neutralises lactic acid from the osteoclasts thus preventing dissolution of the mineral content of the root. It appears therefore, that calcium hydroxide would prevent continuation of the resorptive process and in addition might stimulate repair[12].

Internal resorption

Once internal resorption is diagnosed, treatment with non surgical root canal therapy is necessary. It is critical that the pulp and granulation tissue with odontoclasts be removed to arrest the process[23]. However endodontic surgery can be performed in cases with poor prognosis[48].

External resorption

According to Benenati[23] there is no treatment regime for external surface resorption as it undergoes spontaneous destruction and repair. The treatment of external inflammatory root resorption is dependent on the aetiology but in the event that the pulp is necrotic non surgical root canal therapy with use of calcium hydroxide as an interim medicament is recommended[3]. A success rate of 96% has been reported for the treatment of external inflammatory root resorption with this method[12].

According to Tronstad[9] there is no treatment for ankylosis at present. Ultimately the crown of the tooth will fracture off at the gingival crest as ankylosis progresses, resulting in a complete replacement of the root by bone. Nevertheless antibiotic treatment can only delay not prevent, the root resorption associated with permanent ankylosis[49]. Careful consideration is necessary if extraction is the treatment of choice for ankylosed incisors because this may significantly reduce alveolar bone height that will be difficult to restore[3].

Internal and external resorption can occur simultaneously on the same tooth. This can be detected at various stages of progression and may appear as separate or joined defects. As the resorption advance inward from the external surface and outward from the internal surface, the defects will eventually communicate[16].

An effective strategy to prevent tooth resorption associated with familial bone dysplasia is by therapy using alendronate, a biphosphonate compound and a potent inhibitor of osteoclastic activity[50].
Pressure resorption (due to pressure) may be seen during tooth eruption and in patients with certain tumours impinging on the roots of the teeth. Also seen during orthodontic movements of teeth. If diagnosed late may be quite destructive, however, the resorptive process will be arrested when the situation of the resorbing cells stops[12]

Conclusion

Root resorption is a condition that is difficult to predict, diagnose and treat and therefore it is of critical importance to be able to distinguish between the types of resorption and between teeth with a good or poor prognosis.

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