The concept of root resorptions or *Root resorptions are not multifactorial, complex, controversial or polemical!*

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Abstract

The mechanisms of root resorptions are known, and their causes are well defined. They are clinically asymptomatic and do not induce pulp, periapical or periodontal changes; rather, they are usually consequences of these phenomena. Root resorptions are local and acquired defects, and not dental signs of systemic diseases. Root resorptions occur when structures that protect teeth from bone remodeling, particularly cementoblasts and epithelial rests of Malassez, are eliminated.

Keywords: Root resorption. Tooth resorption. Resorption. Orthodontics.

A concept is a mental representation of an object or phenomenon described by the mind using its general characteristics. A concept may also represent the formulation of an idea by means of words. Still, a concept may be defined as a synonym to conception, definition and characterization. In short, to establish a concept means to identify, describe and classify the different elements and aspects of reality.

In studies about root resorptions, the first paragraph is invariably used to define their concept. In several cases, that concept is limited to a certain type of resorption or restricted to the context of a single clinical case, and does not take into consideration all the aspects of root resorptions. Concepts should be general because, if restrictive, may limit the understanding of the phenomenon as a whole. Therefore, we have set here to discuss the concept of root resorptions to serve as a humble contribution to the preparation of future studies about this topic.

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FIGURE 1 - Concepts are jewels of accurate communications between people at all levels, particularly in clinical and scientific exchanges. The accurate concept of root resorption implies in clear and concise classifications and definitions on the subject.

ROOT RESORPTIONS ARE NOT COMPLEX, POLEMICAL OR CONTROVERSIAL

Root resorptions have two basic mechanisms of occurrence, both very well understood: inflammation or replacement.

» **Inflammatory resorption.** Cementoblasts "recover" or "hide" the root surface, and the Sharpey collagen fibers are inserted among them. Teeth are very close to the bone and separated by the periodontal ligament, which has a mean thickness of 0.25 mm, ranging from 0.2 to 0.4 mm.

Bone is constantly under remodeling thanks to the stimulation of local and systemic factors. Bone dynamics contributes to the stable level of mineral salts in blood and grants it a great capacity to adapt to everyday functional demands. Bone remodeling depends on the receptors that osteoblasts and macrophages have on their membranes, so that local and systemic mediators may manage osteoclast activities. Osteoclasts do not have receptors for bone remodeling mediators and are functionally dependent on osteoblasts and osteoclasts.

On the other side of the periodontium, cementoblasts on the root surface, although very close to the bone, do not have receptors for bone remodeling mediators. They do not respond to or "hear" the biochemical messages to resorb or form new mineralized tissue on the root surface: they are "deaf" to bone remodeling mediators, although they have receptors for other mediators that are essential to cell life, such as growth hormone and insulin.

Any cause that is active in the site where cementoblasts are found and that removes them

from the surface will expose the mineralized root surface; the bone cells, which are very close, will promote root resorption, though maybe only temporary. Root resorptions have local causes that eliminate cementoblasts from the root surface, and no systemic cause is known to produce the same effect.

» **Replacement resorption.** Bone remodeling leads to the constant resorption of mineralized structures, and, at the same time, constant new bone formation also on the periodontal surface of the tooth alveolus. Naturally, each new bone layer deposited on the alveolar periodontal surface is closer to the tooth and, at a mean thickness of 0.25 mm, dentoalveolar ankylosis may soon be seen. Cementoblasts and osteoblasts interfere and form areas of fused cement and bone, alternating with randomly distributed areas of resorption and new bone formation. However, that does not usually happen due to the presence of epithelial rests of Malassez, a net made up of cords that are 20 cells long and 4 to 8 cells wide and that form a structure that resembles a basketball basket in the periodontal ligament around the tooth root.

Epithelial rests of Malassez, as all other epidermal tissues in the organisms, constantly release epidermal growth factor (EGF) for self-stimulation to proliferate and maintain their structures. However, at the same time this mediator in the ligament stimulates bone resorption in the alveolar periodontal surface. Therefore, the periodontal space is preserved and dentoalveolar ankylosis is avoided.

Dentoalveolar ankylosis occurs practically only when epithelial rests of Malassez are eliminated, usually by dental trauma, which may range from light trauma, such as concussion, to more severe trauma, such as avulsion. Like dentoalveolar ankylosis, bone remodeling also affects mineralized dental tissues, which are gradually and inexorably resorbed and replaced with bone, which explains why it is called replacement resorption. In teeth that remain unerupted for a long time, severe periodontal ligament atrophy due to lack of use may facilitate the development of dentoalveolar ankylosis.

Based on the descriptions of these two mechanisms of root resorption, it does not seem logical to say that they are complex or unknown. Also, it seems unacceptable to say, therefore, that their causes are polemical or controversial.

ROOT RESORPTION DOES NOT HAVE A "MULTIFACTORIAL ETIOLOGY"

The term "multifactorial etiology" suggests that a certain disease or phenomenon requires that several causes should act at the same time for their occurrence, although dictionaries do not explicitly assign this connotation to the strict meaning of "multifactorial".

Dental caries is a classical example of a disease with a multifactorial etiology. For dental caries to occur, there should be bacterial plaque on the tooth as a result of lack of oral hygiene, a diet based on carbohydrates, teeth with susceptible enamel, and time for these factors to interact and generate the disease. In other words, one cause depends on the other for the disease to occur.

Diabetes is also multifactorial because the disease occurs if individuals inherit the gene responsible for autoimmunity against the pancreatic insulin-producing cells and inherited factors interact with environmental conditions, such as obesity, inadequate nutrition, a sedentary life-style and stress.

Root resorptions have several causes that act independently from each other. In some special cases, the causes may be associated in root resorption, but that is not usual. From a conceptual point of view, we should avoid saying that root resorptions are multifactorial; rather, we may say, to be more accurate, that it has multiple or several causes. The term "multifactorial" may convey the equivocal connotation of simultaneity of causes for root resorptions to occur.

THE CAUSES OF ROOT RESORPTIONS ARE KNOWN

In inflammatory root resorptions, the causes remove cementoblasts from the surface, such as in the situations described below:

- Chronic periapical lesions: toxic bacterial products, such as lipopolysaccharides, as well as other toxic agents resulting from microbial metabolism, are released in the periapical environment or reach the apical root surface via dentinal tubules.
- Orthodontically applied forces may completely close the lumen of blood vessels, and nutrition may not reach them. Rarely, the contact between tooth and bone resulting from excessive force may physically eliminate cementoblasts from the root surface due to compression.
- Unerupted teeth may compress blood vessels of neighboring teeth when they come closer due to eruptive forces, as it sometimes happens with maxillary canines and third molars.
- Accidental dental trauma may break vessels and may put the tooth in contact with the alveolar bone surface. Dental trauma may be surgical, operative or anesthetic.
- Long periods of occlusal trauma may lead to the death of cementoblasts and, in more severe cases, induce inflammatory root resorptions.

In cases of replacement resorption, the causative factors remove the epithelial rests of Malassez from the periodontal ligament. The main and practically single cause that eliminates this ligament component is dental trauma, which may range from concussion, in its milder form, to avulsion and replantation, in its more severe forms. Dental trauma may be:

- Accidental, during leisure activities, car crashes, violence or other incidents.
- Surgical, such as in cases of inadequate luxation of unerupted canines and trauma to second molars during maneuvers to extract unerupted third molars.
- Trauma due to the action of laryngoscopes over teeth during intubation for general anesthesia.

In teeth unerupted for a long time, excessive periodontal ligament atrophy may create the conditions for dentoalveolar ankylosis and consequent replacement resorption.

ROOT RESORPTIONS ARE NOT SYSTEMIC OR HEREDITARY

In the human species, cell and tissue events occur according to genetic information and tend to be genetically determined, but that does not assign a hereditary nature to all events. Root resorptions, as biological phenomena, include genetically-determined cell and tissue events, but there is no hereditary determination in its initiation or in the development of individual or familial susceptibility.

In the human species there are no diseases, conditions or susceptibilities transmitted from parent to child that may assign a greater tendency towards or prevalence of root resorption. The causes of root resorptions are local and affect cementoblasts, epithelial rests of Malassez, or both. In endocrine diseases, as well as in other systemic diseases, root resorptions are not part of clinical or imaging signs.

Apical and root morphology, the crown-to-root ratio, and the shape of the alveolar bone crest affect the predictability of root resorptions during orthodontic treatment. If asked, we may even say that patients with triangular tooth roots, pipette-shaped or dilacerated apices and rectangular bone crests are more susceptible or predisposed to root resorption during orthodontic treatment, but these tendencies are morphological, and not genetic or hereditary.

ROOT RESORPTION TREATMENT AND PROGNOSIS

The main therapeutic principle for inflammatory root resorptions is the elimination of causes. When inflammation and cell stress are

removed from the area of resorption, bone remodeling units and their osteoclasts are demobilized and leave the root surface; that is, mediators disappear. The pH in the region goes back to neutral, and new cementoblasts are formed and colonize the root surface again in a few days. Immediately after that, new cement is formed, with the reinsertion of collagen fibers into the new cementoblast layer. The root surface is again biologically normal.

If the cause is bacterial contamination through the root canal, adequate endodontic treatment removes the cause, and inflammatory resorption is repaired. If the cause is an orthodontic force, appliance deactivation and the dissipation of forces stop the process. When the possible cause is eliminated and inflammatory root resorption does not stop, the true cause might not have been eliminated.

Replacement resorptions always follow dentoalveolar ankylosis and, once established, the process cannot be stopped. When ankylosis is detected before it progresses into replacement resorption, luxation followed by extrusion may, in most cases, restore the periodontal ligament at the bridges or foci of bone-tooth connection. However, if dealing with replacement resorption in which part of the root was resorbed and replaced with bone, the physical union prevents the separation of both.

In summary, inflammatory root resorption may be controlled and cured, and its prognosis is good, but replacement resorption has a poor prognosis because the tooth will be lost, sooner or later.

ROOT RESORPTIONS DO NOT INDUCE PAIN OR PULP NECROSIS

No matter how close they are to the pulp, inflammatory or replacement root resorptions do not cause pain. The number of mediators present and necessary for mineralized tissue resorption is not enough to induce pain or discomfort for the patient. If there is a painful sensation in teeth with any type of resorption, another cause should be investigated to explain the pain: root resorption is an asymptomatic and, after all, silent biological process.

Although very close to the pulp, or even in the structure of the pulp itself, as in the case of internal resorption, root resorptions do not induce pulp necrosis. Root resorption does not release products that are toxic to cells. The resorption of mineralized tissues has the only purpose of deconstructing them to recycle their mineral and nonmineral contents, which are reused as ions, amino acids and peptides.

Root resorptions alone are clinically asymptomatic and do not induce pulp, periapical or periodontal changes; rather, they are usually consequences of these phenomena, and not their cause.

FINAL CONSIDERATIONS

A way to express the concept of resorption in general and of root resorption in particular

Resorptions in the organism as a whole are phenomena seen in several clinical situations. They are part of the mechanism of structural disarrangement of mineralized tissues. In the interface between osteoclasts and odontogenic mineralized tissue, acids and enzymes are released, and the resulting molecules are carried along the cytoplasm inside vacuoles in a process known as transcytosis. They are then secreted into the extracellular space in the form of amino acids, peptides and ions. In the extracellular matrix and in the body fluids, such as blood and lymph, these components are used by other organs, tissues and cells.

Root resorption is a process to disarrange mineralized odontogenic tissues by the action of bone cells found on their surfaces when the structures that protect teeth against bone remodeling are removed, particularly cementoblasts and epithelial rests of Malassez.

Resorptions are pathological events in permanent teeth, and physiological in primary teeth. In some clinical situations, such as orthodontic treatment, root resorptions are frequent and acceptable, if predicted and mitigated, and are part of the biological cost to have esthetically pleasing and functional teeth.

The mechanisms of root resorptions are known and their causes are well defined. They are clinically asymptomatic and, alone, do not induce pulp, periapical or periodontal changes; rather, they are usually consequences of these phenomena. Root resorptions are local and acquired defects, and not dental signs of systemic diseases.

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