Review Article

Indian Journal of Dental Education
Volume 9 Number 2, April - June 2016
DOI: https://dx.doi.org/10.21088/ijde.0974.6099.9216.8

Periapical Bone Loss

Prashanth Kumar Katta

Abstract

Periapical infection leads to bone loss in that region the mechanism behind bone loss is very subtle and it should be understood to arrest and allow deposition of bone so that the periapical bone will function normally and better survival of the tooth. This article describes how the bone loss occurs and how deposition takes place after the treatment is completed.

Keywords: Aetiology; Diagnosis; Healing; Mechanism.

Introduction

After the infiltration of pathogenic bacteria to the periodontium, the bacteria and/or bacterial products initiate the inflammatory process, consequently activating the inflammatory response. Tissue destruction is caused by the inflammatory cells and their extracellular enzymes. An inflammatory infiltrate is formed, followed by the destruction of the connective tissue, the encapsulation of the bacterial mass and pus formation [1,2].

What is the Apical Lesion?

A Protective Host Response with a Price Tag

Apical lesions represent a protective activity of the host response that is successful most of the time. Nevertheless, this protection has a price tag, which is destruction of the surrounding apical bone. Bone destruction is one of the primary indicative signs of an apical lesion. The gradual disappearance of the bone defect that was caused by this destructive response is commonly used as a major clinical sign and tool to monitor the healing of these lesions [2,3].

Under normal conditions, bone metabolism represents a balance of osteoclastic bone resorption and osteoblastic bone production. This is a complex, interdependent relationship in which osteoblasts mediate the resorptive activity of the osteoclasts. Mediators of inflammation (cytokines, prostaglandins, and many growth factors) tip this balance to favour either bone resorption or bone formation.

Bacteria colonizing the necrotic root canal start inducing damage to the periradicular tissues and give rise to inflammatory changes. In fact, periradicular inflammation can be observed even before the entire root canal is necrotic.

Inflammatory Mediators

The cyclooxygenase enzymes, COX-1 and COX-2, catalyze the conversion of arachidonic acid to prostaglandins.The inflammatory process in periapical chronic lesions is still not fully understood, although four main biochemical factors are known:

1. kinin system
2. vasoactive amines
3. complementary system
4. metabolites of arachidic acid.

The periapical inflammatory reaction is composed of a mixed inflammatory infiltrate characterized by the presence of neutrophils, T and B lymphocytes, plasmocytes, and macrophages, with higher or lower prevalence of certain cell types depending on the stage of the disease. The neutrophils are present in the initial phase of the development of the periapical lesion, playing an important role in the pathogenesis of the disease. With the timedependent progression of the

Author's Affiliation: Assistant Professor, Department of Endodontics, King Khalid University, Abha, kingdom of Saudi Arabia.

Reprints Requests: Prashanth Kumar Katta, Assistant Professor, Department of Endodontics, King Khalid University, Abha, Kingdom of Saudi Arabia.
E-mail: drprashanthkumar@yahoo.com

lesion, the inflammatory process becomes chronic and initiates the recruitment of mononucleated inflammatory cells. Macrophages are the main inflammatory cell type characteristic of this stage [1,4].

Development of Apical Lesions [2,4,5]

Lesions of endodontic origin pose a particular challenge since that bacteria persist in a protected reservoir that is not readily accessible to the immune defenses. In healthy conditions, dental pulp is protected from microorganisms of the oral cavity by enamel and dentin. The exposure of dental pulp to microorganisms as a consequence of dental caries, fractures or operative procedures triggers a local inflammatory response. The progression of such infection and inflammation results in necrosis of the pulp and involvement of periapical tissues, generating a PL (Nair, 1997).

The body's response to the bacteria emerging from the apical foramen is initiated in the adjacent periodontal ligament in the form of apical periodontitis. This response, which is aimed at containing and killing the bacteria, also causes local damage to the host in the form of bone resorption. Among the cytokines that are produced by the cells of the apical inflammatory response, IL-1β and tumor necrosis factor â (TNF β) have the capacity to activate local osteoclastic bone resorption. The first (IL-1β) is produced mainly by activated macrophages, while the second (TNFβ) is a product of activated T-lymphocytes [6,7].

IL-1β and TNF β are the primary causes of the local apical bone resorption. When lining cells of the bone are exposed to these cytokines, they express on their surfaces a signaling molecule, the receptor activator of nuclear factor kappa β-ligand (RANKL). This ligand engages the RANK receptor, which is present on the surface of the neighboringpreosteoclasts and osteoclasts, thus causing the maturation of preosteoclasts into mature osteoclasts and the activation of existing osteoclasts, which express ruffled borders and begin the bone resorbing actively.

The resulting local bone resorption is first radiographically expressed as a widening of the apical periodontal space; this space gradually increases, eventually resulting in a radiolucent lesion in the apical bone, that is, an apical lesion.

Apical bone resorption may thus be considered a side effect of the protective host response. The activation of an effective host response that is aimed at eliminating harmful bacteria results in the local production of cytokines that cause resorption of the surrounding bone.

Six possible biological factors have been described as causing asymptomatic apical periodontitis following root-canal treatment: persistent intraradicular infection, extraradicular infection (principally actinomycosis), foreign body reaction related to the root filling material, the accumulation of endogenous cholesterol crystals that irritate the periapical tissue, true cystic lesions, and scar tissue.

Differential Diagnosis for a Periapical Radiolucency [3, 8, 9]

1. Granuloma
2. Cyst
3. Abscess
4. Scar
5. Foreign body reaction
6. OKC
7. Ameloblastoma (multilocular)
8. Central Giant Cell Granulom (multilocular)
9. Metastatic malignancy (breast, prostate, kidney)

Differential Diagnosis for a Periapicalradioopacity

1. Periapicalcemental dysplasia - cementoma
2. Focal sclerosing osteomyelitis
3. Idiopathic osteoscleroses
4. Cementoblastoma
5. Calcifying odontogenic cyst
6. Calcifying epithelial odontogenictumor
7. Adenomatoid odontogenictumor

Healing [10,11]

Endodontic treatment there is residual inflammation in the apical/periapical tissues which can be visualized radiographically.The host body tries to repair and regenerate the diseased tissue is compromised due to the presence of bacterial contamination; this becomes a chronic process unless adequate clinical treatment to eliminate the infectious agents is initiated.

Conclusion

The extent of the bone loss depends upon the severity of the infection. Treatment should be started immediately bone loss is detected on the radiograph. In reality bone loss is more severe than we can see on

the radiograph. Appropriate treatment will stop further progression of the disease and promote the deposition of the bone, which is a success.

Conflict of Interest

None

Source of Funding

Nil

Acknowledgements

Nil

Ethical Clearance

Not needed as it is a review article

References

1. Role of endotoxin in the etiology of periapical lesions: molecular mechanisms involved in endotoxin's recognition and cell activation, lucisanopaulo nelson-filhoraquel assed bezerra da silvaléa assed bezerra da silva1 andiara de rossi, rgo, rev gaúchodontol, portoalegre. out./dez., 2014; 62(3): 289-298.
2. Yamasaki M, Kumazawa M, Kohsaka T, Nakamura H, Kameyama Y. Pulpal and periapical tissue reactions after experimental pulpal exposure in rats. J Endod. 1994; 20: 13-7.
3. Stashenko P, Wang CY, Riley E, Wu Y, Ostroff G, Niederman R. Reduction of infection-stimulated periapical bone resorption by the biological response modifier PGG glucan. J Dent Res. 1995; 74: 323-330.
4. Armada-Dias L, Breda J, Provenzano JC, Breitenbach M, Rôças IN, Gahyva SM, Siqueira JF, Jr. Development of periradicular lesions in normal and diabetic rats. J Appl Oral Sci. 2006; 14: 371-375.
5. Bacterial Pathogenesis and Mediators in Apical Periodontitis José F. SIQUEIRA Jr Isabela N. RÔÇAS, Braz Dent J. 2007; 18(4): 267-280.
6. Immunonopatogenesis of Chronic Periapical Lesions, Greta ©kaljac-Staudt, Nada GaliÊ, Marina KatunariÊ, Ivana Ciglar, DavorKatanec, ActaStomatol Croat. 2001; 35(1).
7. Periodontal abscess: a review, punitvaibhavpatel, sheelakumar g, amrita patel, journal of clinical and diagnostic research. 2011 Apr; 5(2): 404-409.
8. Wound healing of apical tissues after root canal therapy: a long-term clinical, radiographic, and histopathologic observation study DomenicoRicucci, MD, DDS, Louis M. Lin, BDS, DMD, PhD, Larz S. W. Spångberg, DDS, PhD, Oral Surg Oral Med Oral Pathol Oral RadiolEndod. 2009; 108: 609-621.
9. Brynolf I. A histological and roentgenological study of the periapical region of human upper incisors. Odont Revy. 1967; 18(Suppl 11): 1-176.
10. Ørstavik D, Kerekes K, Eriksen HM. The periapical index: a scoring system for radiographic assessment of apical periodontitis. Endod Dent Traumatol. 1986; 2:20-34.
11. Review of osteoimmunology and the host response in endodontic and periodontal lesions Dana T. Graves, Thomas Oates, Gustavo P. Garlet, Journal of Oral Microbiology. 2011; 3: 5304.