



International Journal of Pharmacology & Toxicology

www.ijpt.org

BONE DESTRUCTION PATTERNS IN PERIODONTAL DISEASES

P.Sivsankar¹, C.Sabarigirinathan^{2*}, H.Gayathri³, Vidya Sekhar⁴, L.Srinidhi⁵,
S.Balasiddharth⁶

¹Senior Assistant Professor, Tamilnadu Government dental College, Chennai Tamil Nadu India - 600003

²Professor and Vice principal, Tamilnadu Government Dental College, Chennai Tamil Nadu India, 600003

³Reader, Srivenkateswara Dental College, Puducherry, India.

⁴Reader, ITS college of dental science and research, Ghaziabad, India.

⁵Post Graduate Student, Tamilnadu Government Dental College, Chennai, TamilNadu, India 600003

⁶Private practitioner, Chennai-600102, Tamilnadu, Chennai. India.

ABSTRACT

The presence of periodontal osseous lesions is clinically significant in many ways. It relates to the associated loss of tooth support, to the site specificity of periodontal destruction, and to the possibility that ecological niches (deep pockets and furcation involvement) associated with some osseous lesions may represent site-specific risk factors or indicators for disease progression.

Keywords: Bone loss, Bone deformity, Bony pockets.

INTRODUCTION

Among the various patterns of bone destruction in periodontal disease described in literature, vertical or angular bone defects and furcation involvements have received particular attention. The reason for this fact may be that these particular entities are relatively more prevalent and prone to deterioration and recurrence even after periodontal therapy.

Periodontal disease alters the morphologic features of the bone in addition to reducing bone height. The extent and the severity of alveolar bone loss in the dentition are usually assessed by a combination of radiographic and clinical means and are important adjuncts to the clinician in the diagnosis, treatment planning, and assessment of prognosis of the periodontal patient [1].

Horizontal Bone Loss

It is the most common pattern of bone loss in periodontal disease. The bone is reduced in height, but

bone margins remain roughly perpendicular to the tooth surface. The interdental septa and facial and lingual plates are affected, but not necessarily to an equal degree around the same tooth (Figure-3) [2].

The configuration of the interdental septum always seemed to be determined by the level of the plaque on the two neighboring tooth surfaces. Thus, if the plaque reaches the same level on both sides, the crest of the interdental septum would assume a horizontal bone loss; if plaque proliferates down to different levels, the crest of the interdental septum would be oblique and an angular defect would be established [3-5].

Horizontal bone loss may be mild, moderate, or severe, depending on its extent. Mild bone loss is defined as approximately 1 mm of attachment loss, and moderate loss is anything greater than 1 mm up to the mid-point of the length of the roots or to the furcation level of the molars. Severe bone loss is anything beyond this point.

Horizontal bone loss is known as the least predictable type of periodontal defects in regenerative

treatment approaches. Recently, the use of enamel matrix proteins (EMP) as an adjunct to periodontal surgery has shown promising results in treatment of sites with horizontal bone loss [6].

Bone Deformities: (Osseous Defects)

Different types of bone deformities can result from periodontal disease. These usually occur in adults but have also been reported deciduous dentition. They occur as a result of the uneven apical propagation of the subgingival plaque front at different root surfaces or at distinct areas of a single root. Loss of attachment has, in other words, de facto progressed at a higher speed along certain “root paths” than others, for a variety of reasons.

Morphology of periodontal osseous defects is determined by a variety of factors which include, Location of the causative microorganisms on the root surface, Root and root trunk anatomy, Thickness of the alveolar bone, Root position within the alveolar process, and the steric relationship with adjacent periodontal lesions.

Classification

Many attempts, however, have been made to classify periodontal osseous defects. Classifications are generally based upon specific morphological criteria and are aimed at guiding clinicians with their diagnosis, treatment and prognosis. A first level of classification differentiates between suprabony defects, infrabony defects, and interradicular or furcation defects.

Suprabony defects are those where the base of the pocket is located coronal to the alveolar crest. Infrabony defects are those where the base of the pocket is located apical to the alveolar crest. With regard to infrabony defects, two types of defects can be recognized: intrabony defects and craters.

Diagnosis

The diagnosis of the presence and the morphology of periodontal osseous lesions represent a major clinical challenge. It is primarily performed combining clinical information derived from the evaluation of the attachment level with information derived from diagnostic-quality parallel-technique intraoral radiographs. A precise knowledge of root anatomy and its variations is also an important component for the diagnosis of periodontal osseous defects, and interradicular defects in particular.

Diagnostic quality radiographs provide additional information on the morphology of the alveolar bone resorption. In this context, the interpretation of the radiographic image of the interdental septum is complicated, since the radiograph provides a two-dimensional illustration of a three-dimensional anatomy consisting of superimposed structures including alveolar bone, hard tooth substances and soft

tissue.⁹Radiographically detectable bone loss does not exclude the presence of an osseous lesion).

. In particular, the site-specific comparison of radiographic bone loss with clinical attachment loss allows the clinician to make a qualified guess of the true osseous architecture, whose exact morphology, however, can only be established after flap elevation. Detection of the defect, its location and extension, along with its major morphological features should be performed before flap elevation. A further aid to this end is the use of transgingival probing or bone sounding [7].

Vertical or Angular or Infrabony defects

These defects occur in an oblique direction, leaving a hollowed-out trough alongside the root; the base of the defect is located is located apical to surrounding bone. In most instances, angular defects have accompanying infrabony pockets; such pockets always have an underlying angular defect.

Classification

Intrabony defects have been classified according to their morphology in terms of residual bony walls, width of the defect (or radiographic angle), and in terms of their topographic extension around the tooth. On the basis of the number of residual alveolar bone walls, intrabony defects can be primarily classified into one-wall, two-wall and three-wall defects (Figure-4).

The three-wall defect was originally called an intrabony defect and the one-wall defect is also called a hemiseptum. Frequently, intrabony defects present a complex anatomy consisting of a three-wall component in the most apical portion of the defect, and two- and/or one-wall components in the more superficial portions. Such defects are frequently referred to as combination defects.

Etiology

A variety of factors have been associated with the formation of intrabony defects. These includes, Trauma from occlusion, Food impaction, Plaque-retaining local elements, Distance between adjacent root surfaces, Overzealous flossing³

Recently, one 10-year study demonstrate that vertical bone loss was grater in smokers compared to non-smokers. In addition, the vertical bone loss increased comparably more in heavy exposure smokers than in light exposure smokers indicating an exposure-response relation.

Prevalence

True epidemiological studies that assess the prevalence of bony defects in large random samples from various populations are sparse The occurrence of vertical bone loss as estimated from cross-sectional studies varies from 23% to 61% in patients seeking dental care and from 30% to 40% in population studies [1,5].

Vertical defects increase with age. There is an increase of infrabony defects from the anterior to posterior areas.⁶ With respect to topography of bony defects in the dentition, it appears that mesial tooth surfaces are affected more frequently than distal ones, but again, the information on whether certain teeth are more prone to develop defects than other is rather inconsistent.⁷ Three wall defects are more frequently found on mesial surfaces of upper and lower molars.

Defect characteristics

The treatment of infrabony defect with bone graft and/or GTR shows extensive variability both in terms of efficacy and predictability. This variability has been partly attributed to patient characteristics, variations in defect anatomy, design of the surgical approach, characteristics of the employed barrier membrane, post-operative infection control and follow-up regimen and the level of experience of the surgeon.

If A_1 represents the CEJ at a tooth with a vertical defect, D_1 the most apical extension of the intrabony lesion (the bottom of the defect), and B_1 the most coronal position of the crest adjacent to the vertical defect, the angle between the two lines A_1D_1/B_1D_1 can be used as a diagnostic radiographic parameter.

Eickholz et al. 2004 has evaluated two different definitions of defect depth and their influence on treatment outcome in terms of bony fill. The defect depth was calculated as – (i) CEJ-BD minus CEJ-AC (INFRA1), and (ii) horizontal projection of the most coronal extension of the bony wall to the root surface to BD (INFRA2). The authors found that INFRA2 was a better predictor of bony fill than that determined by INFRA1 [6].

Role in progression of periodontal disease

A study by Steffensen & Weber (1989) indicated that “wide” bony defects suffered further bone loss after periodontal surgery, whereas “narrow” defects displayed radiographic bone gain. A clear association between the presence of angular bony defects and further periodontal bone loss as well as tooth loss has also been demonstrated.

Osseous Craters

The most common bony lesion described and encountered in periodontal disease is the interdental crater. It is defined as a cup- or bowl-shaped defect in the interdental alveolar bone with bone loss nearly equal on the roots of two contiguous teeth and more coronal position of the buccal and lingual alveolar crest; the facial and lingual/ palatal walls may be of unequal height (AAP 1993) (Figure-5)

This defect can be considered as the result of the apical spread of periodontitis along two adjacent roots in a relatively narrow (mesiodistally) interproximal area. Craters have been found to make up about one third (35.2%) of all defects and about two thirds (62%) of all

mandibular defects. They are twice as common in posterior segments as in anterior segments.⁸ The interdental area collects more plaque and is difficult to clean. The normal flat or even concave faciolingual shape of interdental septum in lower molars may favor crater formation. Vascular patterns from the gingiva to the center of the crest may provide a pathway for inflammation.

Buccal & Lingual Resorptions

The bone and gingival configurations are different buccally and lingually over the root prominences. In addition, the blood supply is a different type; the area is served by a circulatory bed or vascular network instead of by a central complex of nutrient vessels.

For this reason, the pattern of resorption is random. Narrow, deeply penetrating interradicular lesions and circumferential, funnel-shaped lesions are possible and commonly occur in this region. The resorption still follows the pathway for inflammation, but that pathway is different.

The pattern of resorption clearly depends on the mass of bone available in the region. Marginal ledges of bone are not a factor if the root of the tooth is invested in a thin sleeve of bone, and this occurrence is by no means rare when the alveolar process is slender, or the roots are large, or both. In fact, these areas are subject to gingival recession because the roots are often in dehiscence under the gingiva. This dehiscence is sometimes caused by marginal gingival inflammation, which initiates the resorption that destroys the entire labial plate marginally over the root prominences.

The interproximal structure is such that the sudden reduction in root volume from the canine to the lateral incisor is not reflected in the bone topography. Frequently, a thick margin of bone is labial to the lateral incisor and may be adjacent to thin bone over the canine. This arrangement, coupled with the inefficient contact relationship, is most conducive to pocket formation. Because of relative root proximity in this region, the mesiodistal dimension to the interproximal bone is extremely constricted.¹¹

In the interproximal bony septa between the anterior teeth, on the other hand, the volume and configuration of the bone are different, as is the topographic arrangement. A peak is a frequent feature here and results from several factors. Firstly, the labial and lingual marginal bone forms an arc of a shorter radius than that formed posteriorly. This feature alone is sufficient to establish a peaked interproximal septum. Secondly, the labiolingual dimension is much smaller than the posterior dimension.

Reversed Architecture

Numerous terms have been developed to describe the topography of alveolar housing. Examples of morphologically descriptive terms include negative,

positive, flat, and ideal. The architecture is said to be “positive” if the radicular bone is apical to the interdental bone. The bone is said to have “negative” or “reversed” architecture if the interdental bone is more apical than the radicular bone (Figure-6). These defects are produced by loss of interdental bone, including the facial plates, lingual plates or both, without concomitant loss of radicular bone, thereby reversing the architecture. Such defects are more common in maxilla. Flat architecture is the reduction of the interdental bone to the same height as the radicular bone. Osseous form is considered to be “ideal” when the bone is consistently more coronal on the interproximal surfaces than on the facial and lingual surfaces. The ideal form of the marginal bone has similar interdental height, with gradual, curved slopes between interdental peaks.

Physicochemical parameters of the powdered drug were determined and reported as total ash, moisture content, total protein value, total carbohydrate, lipid content and vitamin c. These are determined by standard procedure(8).

Ledges

These are plateau-like bone margins caused by resorption of thickened bony plates (Figure-1). Ledgeing of the marginal bone results when or of a wide ledge on

the buccal aspect in the mandibular molar region as a result of the external oblique ridge. There is a loss in height of the bone without an accompanying reduction in thickness.

Furcation Involvements

The term furcation involvement refers to the invasion of the bifurcation or trifurcation of multirooted teeth by periodontal disease. Detailed knowledge of the morphology of the multirooted teeth and their position in the dental arch is a fundamental prerequisite for a proper understanding of problems which may occur when such teeth become involved in destructive periodontal disease.

Numerous tooth-specific anatomical variations are thought to predispose teeth to furcation involvement. Recognizing these factors is necessary for the treatment of the furcation, regardless of the treatment modality. Anatomical factors that make molars particularly susceptible to periodontal disease include: Accumulation of bacterial plaque as a result of difficult access for oral hygiene procedures. Aberrant root morphology, Enamel projections or pearls, Presence of accessory canals, Size and location of the furcation, Length of the root trunk, Bifurcation ridges, Root concavities. furcation involvement.

Fig 1: Dehiscence on the canine and fenestration of the first premolar



Fenestrations & dehiscences on lingual aspect of mandibular incisors (dry skull specimen)



Fig. 2 - Bony exostosis in relation to mandibular molars



Fig. 3 - Horizontal bone loss in relation to maxillary posterior region



Fig. 4 – Vertical bony defects

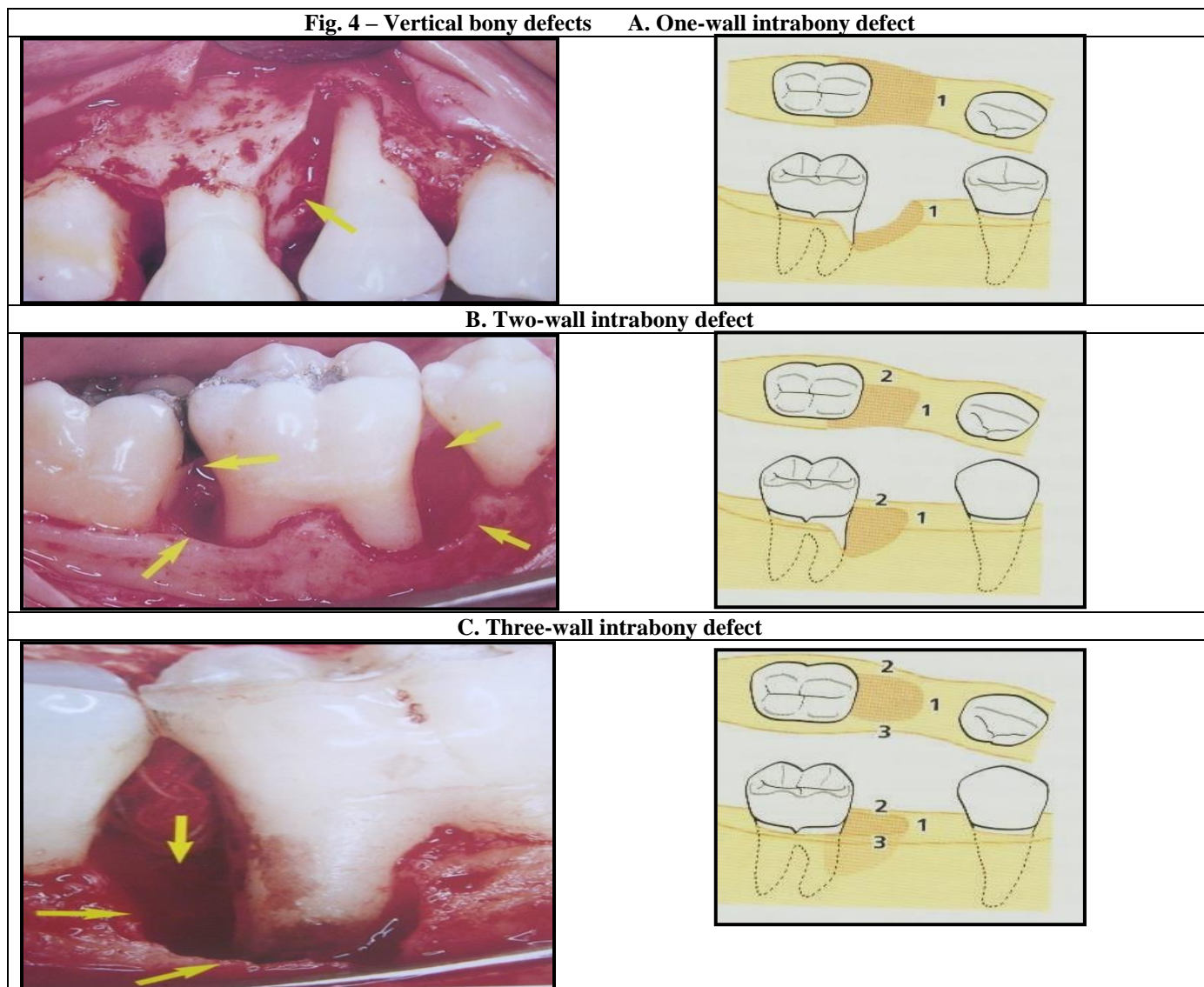


Fig. 5 – Osseous crater (c) between maxillary lateral incisor & canine

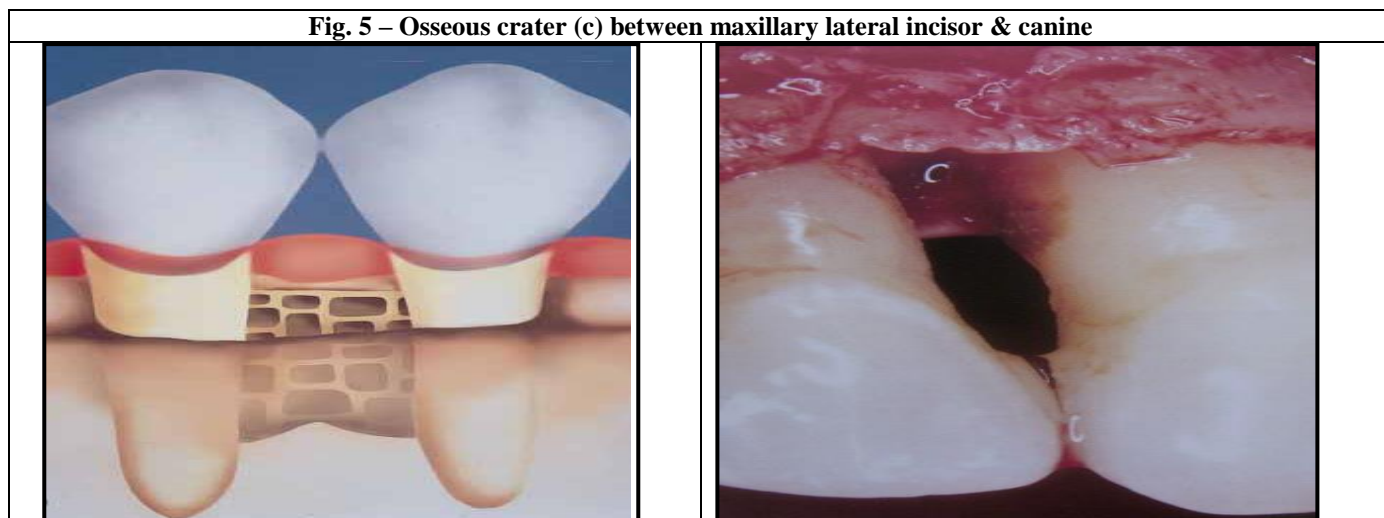


Fig. 6 – Reverse architecture of mandibular posterior region

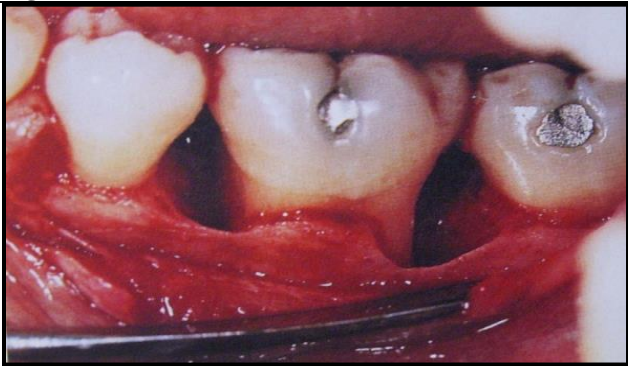


Fig. 7 – Ledge produced by interproximal resorption

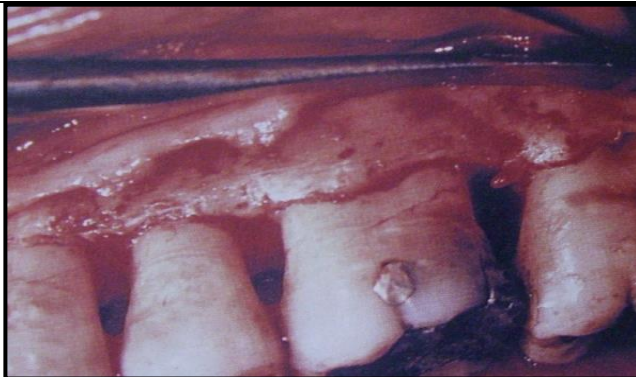
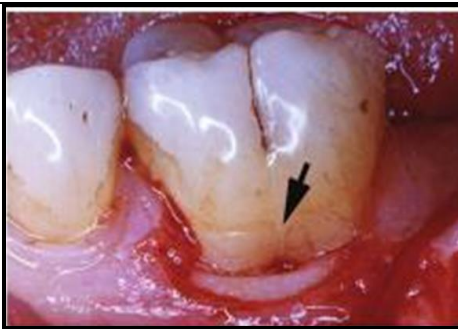


Fig.-8. Classification of Cervical Enamel Projections (Master & Hoskins 1964)



Grade I



Grade II



Grade III

Classification of Furcation Involvement

Fig. 9 – Horizontal classification

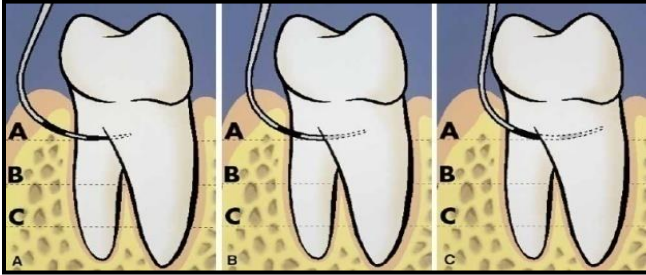
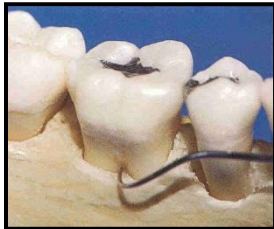
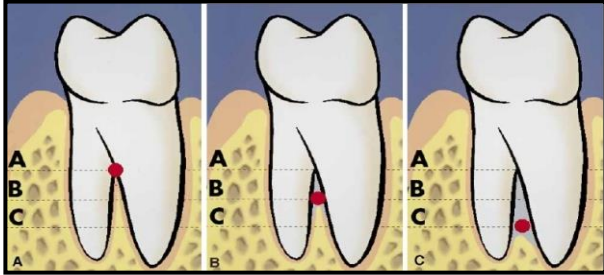


Fig. 10 – Vertical classification



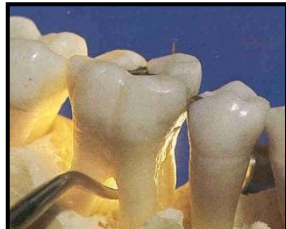
Grade I



Grade II



Grade III



Grade IV

CONCLUSION

Periodontal osseous defects are a frequent sequela of periodontitis. Diagnosing their presence and establishing their morphology before surgical access requires a careful clinical examination combined with diagnostic quality radiographs. This poses a clinical challenge that should not be underestimated. The presence

of periodontal osseous lesions is clinically significant in many ways. It relates to the associated loss of tooth support, to the site specificity of periodontal destruction, and to the possibility that ecological niches (deep pockets and furcation involvement) associated with some osseous lesions may represent site-specific risk factors or indicators for disease progression.

REFERENCES

1. Manson JD. Bone morphology and bone loss in periodontal disease. *J Clin Periodontol* 1976; 3: 14-22.
2. G.Wennstrom JL: Prevalence of furcation involvements in patients referred for periodontal treatment. *J Clin Periodontol* 1996; 23: 1093-1099.
3. Muller HP, Eger T. Furcation diagnosis. *J Clin Periodontol* 1999; 26: 485–498.
4. Albandar JM, Brunelle JA. Kingman A. Destructive periodontal disease in adults 30 years of age and older in the United States, 1988–1994. *J Periodontol* 1999; 70: 13–29.
5. DeSanctis M, Murphy KG. The role of resective periodontal surgery in the treatment of furcation defects. *Periodontology* 2000, Vol. 22, 2000, 154–168.
6. Matthews DC, Tabesh M. Detection of localized tooth-related factors that predispose to periodontal infections. *Periodontology* 2000 2004; 34: 136-150.
7. Loos B, Claffey N and Egelberg J. Clinical and microbiological effects of root debridement in periodontal furcation pockets. *J Clin Periodontol* 1988; 15: 453-63.