



FACTORS DETERMINING BONE MORPHOLOGY IN PERIODONTAL DISEASE

P.Sivsankar¹, C.Sabarigirinathan², Vidya Sekhar³, H.Gayathri⁴, K.Vinayagavel⁵, L.Srinidhi⁶

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

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

³Reader, Srivenkateswara Dental College, Puducheery, India.

⁴Reader, ITS college of dental science and research, Ghaziyabad.

⁵Professor and HOD, Thiruvarur Medical college, Tamilnadu, India.

⁶Post Graduate Student, Tamilnadu Government Dental College, Chennai, Tamil Nadu, India.

ABSTRACT

Sites with infrabony defects have been shown to be at higher risk of disease progression in subjects who did not receive systematic periodontal therapy. On the other hand, evidence that persistence of an infrabony defect after completion of active periodontal treatment may increase the site-specific risk of progression of periodontitis is only indirect and associated with the reported increased risk of periodontal breakdown at sites with residual pockets.

Key words: Infra bony defects, Crestal bones, Fenestrations, Dehiscences.

INTRODUCTION

Periodontal diseases are infections associated with pathogenic bacteria that colonize the subgingival area, causing inflammation, which may lead to the destruction of periodontal tissues. Periodontal disease alters the morphologic features of the bone in addition to reducing bone height.

Normal variation in alveolar bone:

Considerable normal variation exists in the morphologic features of alveolar bone, which affects the osseous contours produced by periodontal disease. The anatomic features that substantially affect the bone destructive pattern in periodontal disease include the following.

Thickness, width and crestal angulation of the interdental septa:

The width and shape of the interdental septum and the angle of the crest normally vary according to the convexity of proximal tooth surfaces and the level of the CEJ of the approximating teeth. The interdental space and therefore the interdental septum between teeth with prominently convex proximal surfaces are wider anteroposteriorly than those between teeth with relatively flat proximal surfaces. The faciolingual diameter of the bone is related to the width of the proximal root surface. The angulation of the crest of the interdental septum is generally parallel to a line between the CEJs of the approximating teeth. When there is a difference in the level of the CEJs, the crest of the interdental bone appears angulated rather than horizontal.

Thick and thin periodontium:

Corresponding Author :- **Dr.C.Sabarigirinathan, M.D.S** Email:- sabarigirinathandr@yahoo.co.in

The “thin” periodontium consists of bell-crowned teeth with the concomitant open embrasures form found with such teeth. The roots are invested with rather thin bone. Fenestrations, dehiscences, and gingival recession are common in such an arrangement. Both gingival and bony architecture reveal thin margins and relatively distinct festooning and undulant contour of bone and gingiva.

Bony architecture in the thin periodontium is not conducive to crater formation, thick margins, and circumferential funnel-shaped resorptive lesions simply because the mass of bone adjacent to roots is not sufficient to provide the various aberrations. In the presence of inflammatory periodontal disease, thin marginal bone resorbs totally, resulting in the exaggeration of the normal festooned form. Interproximal bone in this configuration is usually pyramidal, and so resorption from adjacent inflammation really flattens the septum. This resorption rarely occurs to the extent that an interproximal crater is formed.

On the other hand, the thick periodontium is quite different in almost all respects. Not only is the alveolar housing is thick and bulky, but the teeth, particularly molars, are distinctly different from those found in thin periodontium. Crowns are thickened and blocky, resulting in constricted interproximal embrasures, and the roots are relatively thick and club-shaped.

The pattern of bone destruction is unlike that encountered in thin alveolar process. Because bone resorption follows the localized presence of inflammation, a thick alveolar housing has strong predilection to circumferential, funnel-shaped (angular) patterns of bone loss.

The presence of fenestrations and dehiscences:

Isolated areas in which root is denuded of bone and the root surface is covered only by periosteum and overlying gingiva are termed fenestrations. In these areas the marginal bone is intact. When the denuded areas extend through the marginal bone, the defect is called a dehiscence (Figure-8). Such defects occur on approximately 20% of teeth; they occur more often on the facial bone than on the lingual bone, are more common in anterior teeth than on posterior teeth, and are frequently bilateral. Microscopic evidence of lacunar resorption may be present at the margins [1].

The cause of these defects is not clear. Prominent root contours, malposition and labial protrusion of the root combined with a thin bony plate are predisposing factors. The Northern European skull is often dolicocephalic, i.e. long-headed, the jaws narrow and overcrowded and alveolar plates thin; so fenestrations and dehiscences are more common. Fenestration and dehiscence are important because they may complicate the outcome of periodontal therapy².

The alignment of the teeth:

On teeth in labial version, the margin of the labial bone is located farther apically than teeth on proper alignment. The bone margin is thinned to a knife-edge and presents an accentuated arc in the direction of the apex. On teeth in lingual version, the facial bony plate is thicker than normal.

Root morphology:

Where roots diverge, as they do especially on first upper molars or where the root is markedly convex as it may be on both upper and lower canines, the overlying bone may be very thin or deficient. This may not manifest in health, but where some tissue destruction has taken place a divergent palatal root of an upper first molar can be related to gross recession [2].

Root position within the alveolar process:

The effect of the root-to-bone angulation on the height of the alveolar bone is most noticeable on the palatal roots of maxillary molars. The bone margin is located farther apically on the roots, which forms relatively acute angles with the palatal bone. The cervical portion of the alveolar plate is sometimes considerably thickened on the facial surface, apparently as reinforcement against occlusal force [3].

Proximity with another tooth surface:

If interproximal spaces are reduced as a result of crowding, root proximity may occur. This leads to less effective removal of plaque and subgingival calculus in the inaccessible interproximal area. Even surgically, these teeth may be difficult to treat, leading to a compromised prognosis. Nonetheless, a well motivated and dexterous patient will likely prevent plaque accumulation regardless of tooth shape or position [4].

Exostoses:

Exostoses are localized, peripheral overgrowths of bone (Figure-9). Depending on their location in the jaw they are identified as torus palatinus (hard palate) or torus mandibularis (lingual mandibular plate). Sometimes, several bony overgrowths occur on the vestibular alveolar bone and are simply called multiple exostoses.

A peculiar condition consisting of bone exostoses has been reported to occur in some patients after undergoing either a skin graft vestibuloplasty or an autogenous free gingival graft. A slowly growing exostosis develops at the recipient site of the gingival graft. A definite female sex predilection is characteristic of this condition which usually presents in the canine-premolar area of the mandible or maxilla [5].

It can be suggested that the sequence of events leading to the formation of exostoses in the site of a previous free gingival graft, begins with a soft-tissue

trauma in the receptor site, which produces the release of inflammatory mediators, including insulin-promoting growth factor. It also has been shown that a soft tissue injury in the vicinity of bone induces a periosteal proliferation.

Vascular disruption as a consequence of the surgical trauma, resulting in transient ischemia in the periosteum, would produce hypertrophy and hyperplasia of the periosteal cells, with an osteogen differentiation. Finally, the presence of excessive forces would then favor, in the long run, the formation of exostosis. However, although these mechanisms have been suggested as an explanation of the formation of exostoses and callus after bone injuries, their role in cases of exostoses after free gingival grafts has not been determined yet [6].

Microscopically, the bone exostosis consists of lamellar or woven bone, or both, surrounded by the fibrous connective tissue with or without scarce inflammatory cells. Surgical removal of this lesion is indicated if interference with prosthetic appliance occurs or if esthetic concerns emerge.

Trauma from occlusion:

Trauma from occlusion may be a factor in determining the dimension and shape of bone deformities. It may cause a thickening of the cervical margin of alveolar bone or a change in the morphology of the bone (e.g., angular defects, buttressing bone) on which inflammatory changes will later be superimposed.

Buttressing bone formation (Lipping):

When bone is resorbed by excessive occlusal forces, the body attempts to reinforce the thinned bony trabeculae with new bone. This attempt to compensate for lost bone is called buttressing bone formation and is an important feature of the reparative process associated with trauma from occlusion. It also occurs when bone is destroyed by inflammation or osteolytic tumor. When it occurs within the jaw, it is termed *central* buttressing bone formation.

When it occurs on the external surface, it is referred to as peripheral buttressing bone formation. In central buttressing the endosteal cells deposit new bone, which restores the bony trabeculae and reduces the size of the marrow spaces. Peripheral buttressing occurs on the facial and lingual surfaces of the alveolar plate. Depending on its severity, peripheral buttressing may produce a shelf like thickening of the alveolar margin, referred to as lipping, or a pronounced bulge in the contour of the facial and lingual bone [7].

Food impaction

Interdental bone defects often occur where proximal contact is abnormal or absent. Pressure and irritation from food impaction contribute to the inverted bone architecture. In some cases the poor proximal relationship may result from a shift in tooth position because of extensive bone destruction preceding food impaction. In such patients, food impaction is a complicating factor other than the cause of the bone defect.

Dehiscence on the canine and fenestration of the first premolar (dry skull specimen)



Fenestrations & dehiscences on lingual aspect of mandibular incisors



Bony exostosis in relation to mandibular molars



Horizontal bone loss in relation to maxillary posterior region



CONCLUSION

The destruction of the alveolar bone in periodontal disease is determined by the balance of resorption and regeneration of bone. The role of inflammatory cytokines in the destruction of periodontal

tissues during disease process has been widely. Besides the role of inflammatory cytokines, recent studies have identified key genes, core binding factor and osteoprotegerin that have a central role in the formation and activity of bone forming and bone-resorbing cells.

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