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# RESEARCH ARTICLE

# BONE LOSS PATTERNS IN PERIODONTITIS-AN OVERVIEW

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**ABSTRACT**

Periodontal disease is an inflammatory condition that affects the supporting structures of the teeth which eventually lead to bone loss, tooth mobility and tooth loss if left untreated.Bone loss in periodontal disease is a hall mark and the patterns of bone loss is based on the type of periodontal destruction present.A number of factors contribute to bone loss in periodontitis like bacterial derived factors, antigens that stimulate a local inflammatory response and activation of innate immune system.The present review gives us an overview of classification of bone defects, bone loss patterns specific to each periodontal condition and factors influencing bone loss.



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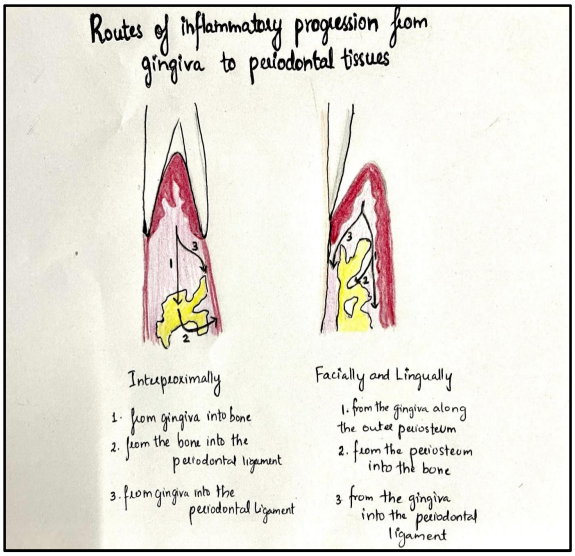
# INTRODUCTION

Alveolar bone is the bony portion of the maxilla and the mandible in which roots of the teeth are held by fibers of periodontal ligament.[GPT -8][1] "Periodontitis is a bacterial-induced inflammatory condition that affects the teeth's supporting structures, leading to damage and potential tooth loss."[2] Changes that occur in bone which are responsible for tooth loss is a crucial factor, as the destruction of bone occurs due to periodontitis. Periodontal disease changes the morphological features of the bone along with reduction in bone height. Hence the present review focused on the pathogenesis , classification and patterns of bone loss in periodontitis.[3]

**Pathway of spread of inflammation in periodontitis**

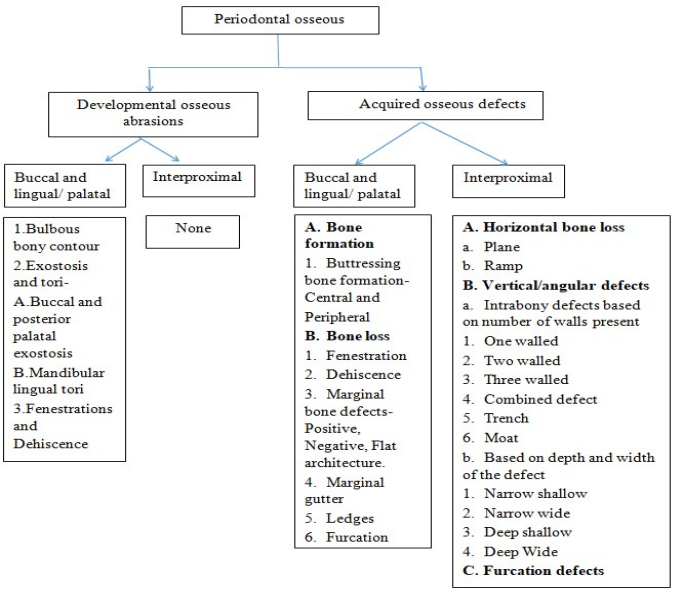
Gingivitis progresses along the collagen fibers and blood vessels, infiltrating the surrounding loose tissue and eventually reaching the alveolar bone.[4] Although the primary site of inflammation is in the marginal periodontium, the response is more widespread, often affecting the bone and triggering a reaction before any visible signs of crestal resorption or attachment loss appear.[5] In the upper molar region, the inflammation can spread to the maxillary sinus, leading to thickening of the sinus membrane.[6] In the areas between teeth, inflammation propagates through the loose connective tissue surrounding blood vessels, along fiber bundles, and into the bone via vascular channels that penetrate the interdental septum at various points, including the center, sides, or angles of the crest. Moreover, inflammation can enter the bone through multiple channels. Less commonly, the inflammation spreads directly from the gingiva into the periodontal ligament and then into the interdental septum.[7] On both the facial and lingual aspects, the inflammatory response from the gingiva propagates along the outer surface of the bone, following the periosteum, and enters the marrow spaces through vascular channels in the outer cortical layer of the bone.[8]



## Classification

1. Goldman and Cohen
   a. Suprabony
   b. Infrabony
      - Intrabony
      - Craters
2. Goldman and Cohen (1958)
   a. One wall
   b. Two wall
   c. Three wall
   d. Combined defect[9]
3. According to Glickman [1964]
   - Osseous craters
   - Hemiseptal defects
   - Intrabony defects
   - Bulbous bone contours
   - Inconsistent margins and ledges
   - Reverse architecture
4. According to Prichard
   - Furcation involvement
   - Anatomic aberrations of alveolar process
   - Exostoses and tori
   - Dehisence and fenestration
5. According to Clarke[10]
   A. Vestibular, lingual or palatal structures or defects
      i. Normal anatomic structures
         - External oblique ridge
         - Retromolar triangle
         - Mylohyoid ridge
         - Zygomatic process
      ii. Exostoses and tori
         - Mandibular lingual tori
         - Buccal and posterior palatal exostoses
      iii. Dehiscence
      iv. Fenestrations
      v. Reverse osseous architecture
   B. Vertical defects
      i. One walls
      ii. Two walls
      iii. Three wall
      iv. Combination with different number of walls at the various levels of the defects.
   C. Furcation defects
      i. Class I or Incipient
      ii. Class II or partial
      iii. Class III or through and through

6. Modified classification by Vandana and Bharath [11]




## Radius of action

Garant and Cho[12] discussed how locally produced bone resorption factors and bacterial biofilm affect bone loss. They suggest that bone resorption factors need to be close to the bone surface to work effectively. The range of effectiveness for bacterial biofilm inducing bone loss is between 1.5mm to 2.5mm. Beyond 2.5mm, the effect diminishes. Interproximal angular defects are likely to occur in spaces wider than 2.5mm, leading to horizontal bone loss.[13] Tal's[14] measurements in human patients supported this concept. The presence of bacteria in the tissues can cause large defects that are more than 2.5mm away from the tooth surface, especially in aggressive types of periodontitis.[15] Factors determining bone morphology in periodontal disease.

## 1. Normal variation in alveolar bone

The morphology of alveolar bone exhibits significant natural variation, which influences the osseous contours resulting from periodontal disease. Several key anatomic features play a crucial role in shaping the pattern of bone destruction caused by periodontal disease, including:

- The dimensions and angulation of interdental septa (thickness, width, and crestal angle)
- The thickness of the alveolar plate on both facial and lingual surfaces
- The presence of fenestrations (openings) and dehiscences (gaps) in the bone

These factors contribute to the complexity and variability of periodontal disease's impact on alveolar bone. Alignment of the teeth

- Root and root trunk anatomy
- Root position within the alveolar process
- Proximity with another tooth surface.

## 2. Exostoses

Exostoses are bony growths that can vary in size and shape, and can occur in different forms, such as small or large nodules, sharp ridges, spike-like projections, or a combination of these.[16] Interestingly, palatal exostoses are relatively common, found in approximately 40% of human skulls.In rare instances, exostoses have been reported to develop after the placement of free gingival grafts, highlighting the dynamic nature of bone growth and adaptation.[17]

## 3. Trauma from occlusion

Occlusal trauma may play a role in shaping the size and form of bone deformities. It can lead to a thickening of the cervical margin of the alveolar bone or alter the bone's morphology, resulting in changes such as angular defects or buttressing bone. These changes can then provide a foundation for subsequent inflammatory alterations to occur.

## 4. Buttressing bone formation

In response to weakened bony trabeculae due to resorption, the body may attempt to compensate by forming new bone tissue to provide support. This process is known as buttressing bone formation. When it occurs within the jawbone, it is referred to as central buttressing bone formation. Conversely, when it occurs on the external surface of the bone, it is termed peripheral buttressing bone formation.[18] The latter can lead to an expansion of the bone contour, which may be associated with the formation of osseous craters and angular defects.

## 5. Food impaction

Interdental bone defects frequently arise when proximal contact is abnormal or absent, leading to pressure and irritation from food impaction, which contributes to the inverted bone architecture. In some instances, the poor proximal relationship may be a consequence of tooth displacement resulting from extensive bone destruction that occurs prior to food impaction. In such cases, food impaction is a secondary complication rather than the primary cause of the bone

defect. Food impaction can occur in two directions: vertically, due to occlusal forces, or laterally, due to pressure from the lips, cheeks, and tongue.

**6. Aggressive periodontitis**

Aggressive periodontitis typically leads to attachment and bone loss around the incisors and first molars, especially in teenage cases. Notably, the first molars often exhibit a vertical or angular pattern of alveolar bone destruction in aggressive periodontitis. The underlying cause of this localized bone destruction in this type of periodontal disease remains unknown**.**

**MECANISM OF BONE DISTRUCTION**

Periodontal bacteria can translocate through the ulcerated epithelium of the periodontal pockets , entering the blood stream and causing bacteremia and systemic inflammation. This leads to an increase in serum levels of IL-6,induced by blood borne Porphyromonas gingivalis. Consequently, an osteoclast precursor population in the bone marrow expands. The precursor cells of osteoclasts show a strong tendency towards becoming osteoclasts and are found in different areas where bone is being broken down. These precursor cells can mature into fully developed osteoclasts when they are exposed to receptor activator of NF-κB ligand (RANKL) produced locally. This idea suggests a potential way in which the bone marrow could connect periodontitis with other conditions that involve bone loss, like rheumatoid arthritis.[19]

**BONE FACTOR CONCEPT**

In periodontal diseases, the bone factor concept describes the systemic influence of the response of alveolar bone. When there is generalized tendency towards bone resorption, bone loss is initiated by a local inflammatory process that maybe magnified. Local and systemic factors regulate the physiologic equilibrium of the bone.[20]

**TYPES OF BONE LOSS**

**HORIZONTAL BONE LOSS**

Horizontal bone loss is the most frequently observed bone loss pattern in periodontitis. The height of the bone is described, but the bone margin still remains almost perpendicular to the tooth surface. Interdental septa and facial and lingual plates are also affected, but not at a degree equal to that present around the tooth.

**VERTICAL OR ANGULAR DEFECTS**

Vertical or angular defects are when the bone alongside the roof forms a hollowed-out trough in an oblique direction, with the base of the defect located below the surrounding bone. Typically, angular defects are accomplished by intrabony periodontal pockets, while intrabony pockets always have an underlying angular defect. Angular defects can be classified based on the number of osseous walls they possess, which can range from one, two, or three. If the number of walls in the apical part of the defect is higher than in its occlusal part, we use the term 'Combined osseous defect'. Vertical defects that happen between the teeth can often be seen on X-rays, although thick bony plates might make then harder to spot. Angular defects may also occur on the facial and lingual/palatal surfaces, but they aren't visible on radiographs. Surgical exposure is the definitive way to confirm the existence and shape of vertical osseous defects. Vertical defects tend to become more prevalent as a person gets older.[21] Around 60% of individuals with interdental angular defects typically have just one defect. Radiographically, vertical defects tend to be most prevalent on the distal and mesial surfaces.[22] However, when it comes to three-wall defects, they are commonly found on the mesial surfaces of upper and lower molars.[23]

**OSSEOUS CRATERS**

Osseous craters, which are indentations in the interdental bone, are typically limited to the facial and lingual walls. Craters account for approximately one third of all defects and about two thirds of all mandibular defects. They tend to occur twice as frequently in posterior segments compared to anterior segments.[24] In 85% of cases, the heights of the facial and lingual crests of a crater are found to be identical. In the remaining 15% of cases, the lingual crests are slightly higher.

High frequency of interdental craters are due to; [25]

- The interdental area is notorious for collecting plaque and can be quite challenging to clean.
- The normal flat or even concave faciolingual shape of the interdental septum in lower can actually contribute to the formation of craters.
- The vascular patterns extending from the gingiva to the center of the crest can be potentially serve as a pathway for inflammation.

**BULBOUS BONE CONTOURS**

Bulbous bone contours can be caused by exostoses, which are bony enlargements. They can also result from adaptation to function or buttressing bone formation. They are more often in maxilla than mandible.

**REVERSED ARTCHITECTURE**

When interdental bone, including the facial and lingual plates, is lost without losing radicular bone, it reverses the normal architecture, causing reversed architecture defects. These defects are common in maxilla of patients with periodontitis.[26]

**LEDGES**

Ledges are formed when the bone plates thickens and are then resorbed, creating plateau like bone margins.

## CONCLUSION

In periodontal diseases, the pathophysiology of bone destruction involves a mix of destructive and reparative processes. The host response aims to repair the injury to restore the the tissues normal structure and function. However, if the infection persists, these processes continue without resolution, leading to various tissue response patterns to the injury. Surgical periodontal therapy aims to achieve the restoration of the normal physiological bone contour.

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