

Alveolar bone in disease

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Abstract

Anatomically, alveolar bone is the part of the maxilla and mandible that surrounds and supports the teeth. It undergoes continual remodelling process which maintains the balance between the apposition and resorption of alveolar bone. This balance if disturbed produces changes in terms of quality and quantity of alveolar bone. Changes in both quality and quantity of alveolar bone (alveolar bone defects) can be significant with respect to the progression of a particular disease or in response to a therapy.

This review describes the alveolar bone defects associated with various pathological conditions and the effect of systemic factors on the architecture of alveolar bone.

Keywords: Remodelling process, Alveolar bone defects, Systemic factors.

Introduction

In a healthy individual alveolar bone constantly undergoes bone formation by osteoblasts and bone resorption by osteoclasts. But certain disorders of the endocrine system, bone metabolism and other systemic diseases are known to disturb this balance and may alter the form and function of alveolar bone. Systemic influences on bone resorption may be exerted by several mediators, including parathyroid hormone (PTH), interleukin-1, tumour necrosis factor (TNF), transforming growth factor (TGF), and 1,25-dihydroxy vitamin D3. This increased bone resorption modifies the quality and quantity of alveolar bone which provides an easier pathway for the spread of inflammation.⁽¹⁾

Mechanism of bone resorption: Bone resorption is considered as normal destruction process in the bony remodelling phenomenon which is mediated by osteoclasts. This occurs usually at random sites or are specific to the areas that require repair. Bony remodelling comprises of six phases:

- 1. Quiescent phase:** In this phase the bone is at rest. Factors that initiate this are still unknown.
- 2. Activation phase:** This phase starts with the activation of bone surface through dissolution of endosteal surface by collagenases and retraction of osteoblasts on the endosteal surface. This leads to activation of osteoclast precursors from blood circulation which in turn results in differentiation, migration and fusion of multinucleated osteoclast cells. Furthermore, osteoclast cells orient towards the bone surface.
- 3. Resorption phase:** Upon orientation of osteoclast cells over the site of resorption cathepsin k, reactive oxygen species produced by Tartrate resistant acid phosphatase (TRAP) are secreted at ruffled border into the resorptive pit. The

effectiveness of the secretion by osteoclast cell depends on sealing zone formed on the resorption compartment. Within this sealing zone pH reduces and results in degradation of bone matrix. This resorption produces irregular scalloped cavities on bone surface known as 'Howship lacunae'. Bone resorption usually takes 2-4 weeks in each remodelling cycle.

- 4. Reversal phase:** This phase is characterized by transition from bone resorption to bone formation under the influence of coupling signals like TGF- β 3, IGF-1, IGF-2, bone morphogenetic proteins, PDGF, or fibroblast growth factor.
- 5. Formation phase:** under the influence of these coupling signals osteoblast are formed from preosteoblastic precursors. The osteoblasts then lay down the osteoid matrix.
- 6. Mineralization phase:** This phase occurs 30 days after the osteoid formation. Once mineralization is complete again quiescent phase starts.⁽²⁾

In various pathologic conditions this remodelling cycle is characterized by prolonged resorption phase and an impaired reversal phase which leads to significant bone loss without bone formation.

Alveolar bone and periodontitis

Periodontal disease or periodontitis, is defined as a disease of the tooth-supporting (periodontal) tissues caused by various pathogenic bacteria.⁽¹⁾ Various authors like Saglie et al. (1987) have postulated that the connective tissue invasion by various bacterial species elicits an abnormal host response leading to rapid bone resorption. Similarly, Newman et al. (1979) also associated rapid bone loss with increased presence of loose, unattached and motile gram negative bacterial species in pocket.⁽³⁾

The initial response to bacterial invasion is the localized inflammatory reaction that activates the innate

immune system. Amplification of this initial localized inflammatory reaction causes the release of an array of cytokines and other mediators through which the inflammation propagates in the gingival tissues. The failure to regulate this “inflammatory front” within gingival tissue results in expansion of the response in alveolar bone. The inflammatory process thus results in the destruction of connective tissue and alveolar bone that may lead to tooth loss.⁽⁴⁾

Spread of inflammation into alveolar bone

Facially and lingually, inflammation propagates from gingiva along outer periosteum to involve the bone and periodontal ligament. Interproximally, inflammation spreads to the loose connective tissue around the blood vessels, through the fibers, and then into the bone.⁽⁵⁾

This pathway of inflammation becomes a crucial factor in determining the type of alveolar defect that is seen in a disease.

Alveolar bone defects: Glickman (1964) classified the alveolar defects into osseous craters, hemiseptal defects, Infrabony defects, Bulbous bone contours, Reversed architecture, Inconsistent margins and Ledges. Whereas Prichard (1967) expanded Glickman’s classification by including furcation involvement, anatomic aberrations of alveolar process, exostoses and tori, dehiscence and fenestrations.

Goldman and Cohen (1958) classified alveolar defects into:

1. Suprabony defects: Where the base of pocket is located coronal to the alveolar crest.
2. Infrabony defects: Where the base of the pocket lies apical to the alveolar crest.
3. Intrabony defects: Bony defects whose infrabony component affects primarily one tooth.
4. Craters: The defect affects two adjacent root surfaces to a similar extent.^(5,15)

Types of alveolar bone defect

1. **Horizontal Defects:** It is the commonest pattern of bone loss. In this defect, the bone is reduced in such a way that the bone margin is approximately perpendicular to the teeth surface. Interdental septa, facial and lingual plates of bones are affected, but to an equal degree around the same tooth.⁽⁶⁾
2. **Vertical /Angular Defects:** It occurs in oblique direction usually. The base of the defect is situated apical to the surrounding bone. Vertical defects are seen adjacent to a tooth and form a triangular area of missing bone, known as triangulation. In most instances, angular defects are accompanied by an infra bony pocket.⁽⁶⁾

Angular defects are classified by Goldman and Cohen on basis of number of walls involved:

- i. **One wall defect/Hemiseptum:** Only one wall of the interseptal wall remains and there is complete

destruction of mesial and distal portion of interseptal bone which is visible on radiographs.

- ii. **Two walled defects:** Characterized by saucer shaped cavity in interdental bone, facial and lingual walls intact; most commonly found in posterior segment of maxilla/mandible which is visible on radiograph.
 - iii. **Three walled defects:** In this the bony walls are present on three sides and tooth forms the fourth wall.
 - iv. **Combined defect:** Number of walls in apical portion of defect are greater than its occlusal portion.⁽⁵⁾
3. **Fenestrations:** Are the isolated areas in which the root is denuded of bone and the root surface is covered only by periosteum and overlying gingiva. Predisposing factors includes prominent root contours, malposition, labial protrusion of the root combined with a thin bony plate. It is seen more often on facial bone than on lingual bone more common on anteriorly than posteriorly. It occurs bilaterally.⁽⁷⁾
 4. **Dehiscences:** When the denuded areas extend through the marginal bone then defect is called a dehiscence. Alveolar bone dehiscences were classified into the following types, based on the dehiscence height along with other accompanying alveolar bony defects. These classifications were based on the measurements obtained in the sagittal planes.^(8,9)

Class I: This includes dehiscences which are present on either buccal or lingual side of the tooth, without any alveolar bone defects. Then tooth root was divided into three equal parts, from the cemento enamel junction to the apex of root for further classifying into subdivisions.

Division I: Dehiscences of the coronal one-third of the root.

Division II: Dehiscences of the middle one-third of the root.

Division III: Dehiscences of the apical one-third of the root, without involving the apical foramen.

Class II: This includes dehiscences with alveolar bone defects which are present peri apically either in buccal or lingual side of the tooth.

Division I: Dehiscences of the whole root involving the apical foramen.

Division II: Dehiscences with periapical lesions. Wherein periapical lesion occurred as radiolucency in the apical part of root that exceeded twice the width of the periodontal ligament space.⁽¹⁰⁾

Division III: Dehiscences with fenestrations surrounding the apex of the root. Though fenestration is an alveolar bone defect without involving the alveolar margin.⁽¹¹⁾

Class III: Dehiscences located on both sides (buccal or lingual) of the tooth. Further

classification is done referring to the divisions of Class I and Class II whichever is more severe.

5. **Osseous Crater:** Osseous craters are characterized by the concavities in the crest of the interdental bone confined within the facial and lingual walls. Craters have been found to make up to 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.⁽⁴⁾
6. **Bulbous Bone Contours:** Bulbous bone contours are bony enlargements that are caused by exostosis, adaptation to function, or even buttressing bone formation. They are found more frequently in the maxilla than in the mandible.
7. **Reversed Architecture:** Reversed architecture 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 normal architecture. Such defects are more common in the maxilla.⁽¹²⁾
8. **Ledges:** Ledges occur as the defects with plateau-like bone margins resulting from loss of thickened bony plates.⁽⁵⁾
9. **Furcation Involvement:** The furcation involvement is defined as the invasion of the bifurcation and trifurcation of multirooted teeth by periodontal disease. The prevalence of furcation involved molars is not clear. Though mandibular first molars are most commonly involved, and the maxillary premolars are the least common. The number of furcation involvements increases with the age.⁽¹⁰⁾
Furcation involvement is classified based on the amount of tissue destruction by Glickman in 1953.^(13,14)
 - i. Grade I is associated with incipient bone loss.
 - ii. Grade II is associated with partial bone loss.
 - iii. Grade III is associated with total bone loss leading to through-and-through passage from the furcation.
 - iv. Grade IV is same as grade III, but with gingival recession resulting in the exposure of furcation.
10. **Trench shaped defect:** Trench defect occurs when such bone loss affects two or three confluent surfaces of the same tooth. Trenches can be similarly identified by the tooth surfaces involved (e.g., mesiofacial, mesio-lingual-distal, etc.). Hence, there are eight possible combinations seen in this defect i.e. mesio-facial, mesio-lingual, disto-facial, disto-lingual, mesial-facial-distal, mesial-lingual-distal, facial-mesial-lingual, facial-distal-lingual.⁽¹⁶⁾
11. **Moat shaped defects:** Moat shaped defects occur circumferentially around the teeth i.e. when bone loss deformity involves all the four surfaces of a tooth.⁽¹⁶⁾

12. Ramp shaped defects: Ramp shaped defects occur when both alveolar bone and its supporting bone are lost such that the margins of the defect lie at different levels.⁽¹⁶⁾

13. Plane defect: Plane defect occurs when both alveolar bone and supporting bone is lost such that the margins of the defect lie at the same level.⁽¹⁶⁾

Trauma from Occlusion: Trauma from occlusion may be considered a factor in determining the dimension and shape of bone deformities. It may result in the thickening of the cervical margin of alveolar bone or a change in the morphology of the bone on which inflammatory changes will later can be superimposed.⁽¹⁷⁾

Sometimes bone formation occurs in an attempt to buttress weakened bony trabeculae by resorption. For example:

- i. Central buttressing bone formation which occurs within the jaw.
- ii. Peripheral buttressing bone formation when it occurs on the external surface. Peripheral buttressing may cause bulge in the bone contour, called lipping, which is accompanied by the production of osseous craters and angular defects.^(5,18)

Systemic Diseases

Systemic Diseases affecting the bone architecture include Osteoporosis, Vitamin D deficiency, Diabetes, Hyperparathyroidism, Haematological disorders, Paget's disease, Fibrous dysplasia.

Osteoporosis is a systemic bone disease characterized by reduced bone strength, low bone mineral density (BMD), and altered macroscopic and microscopic architecture, and is associated with increased risk of fractures. A reduced buccolingual width in the dentate alveolar process may occur as a result of periosteal resorption. Osteoporosis could also influence the rate of tooth movement. Persistent trabeculae are found along the planes of bone stress. Trabeculae may be arranged radially; in between wide spaces are present. Radiographically, there is reduced density accompanied by thinning of cortical boundaries.⁽¹⁹⁾

Vitamin D at standard level maintains the calcium balance in the body. This calcium can be used for mineralization of bone. In hypo-calcaemic, alveolar bone showed hypomineralization and demonstrated a cellular and matrix organization, similar to the immature woven bone. Vitamin D deficiency results in osteoporotic bone with thinned dental crypts. Also, Vitamin D excess can affect nuclear receptors in the osteoblasts resulting in bone resorption. The trabeculae become reduced in number. In severe cases, jaws appear completely radiolucent, so that teeth appear to be suspended in air. Thus, it can be concluded that deficiency as well as excess of Vitamin D can lead to osteopenia and bone resorption.⁽²⁰⁾

Diabetes results in poor bone quality because of formation of advanced glycation end products, eventually resulting in fractures. Taylor et al postulated that inadequate glycemic control can result in increased alveolar bone loss. It alters the response of the periodontal lesion to local irritants, hastening bone loss and retarding postsurgical healing of the periodontal lesions. This disease exhibits a fulminating periodontitis with periodontal abscess formation. This will give rise to mobility of teeth. There is severe and rapid alveolar bone resorption takes place. Diabetes also increases apoptosis and decreased the number of bone-lining cells, osteoblasts, and periodontal ligament fibroblasts. Thus, diabetes caused a more persistent inflammatory response, greater loss of attachment and more alveolar bone resorption, and impaired new bone formation.⁽²¹⁾

In *leukemia*, the infiltrate fills the marrow spaces and the periodontal ligament which results in osteoporosis of alveolar bone and the supporting bone along with disappearance of periodontal fibers. Destruction of alveolar bone is the most common manifestation of leukemia. Bone loss may be in the form of transverse lines of increased radiolucency or irregular areas and bone loss produced gives so called, moth eaten appearance.⁽²²⁾

Paget's disease of bone/ Osteitis deformans is associated with abnormal remodelling of bone that results in the weakening of the affected bone with pain, fractures and arthritis in the joints near the affected bones. In this disease coarse and sparse trabecula are seen that tend to converge towards the midline of mandible. In the skull, early lytic lesion may be seen as discrete radiolucent areas termed as osteoporosis circumscripta. The margins are somewhat irregular. Bone scan may demonstrate marked uptake throughout the entire mandible which is seen radiographically as Lincoln's sign or black beard. In later stages, rounded radiopaque patches of abnormal bone are often seen giving a cotton wool appearance. Also, there is increase in alveolar width associated with flattening of palate when maxilla is involved.⁽²³⁾

Fibrous dysplasia is a disorder where fibrous tissue replaces normal bone and marrow that makes the bone weak. Fibrous dysplasia appears as areas of whorled amorphous calcified materials that are well circumscribed. Radiographically, this gives a characteristic ground glass appearance. As the lesions grows, dysplastic bony trabeculae increase in size and number and give an appearance of smoky mottled radiopacities. The replaced structure of bone resembles the ring of orange which is called as 'orange peel'. Enlarging deformities of alveolar process mainly in buccal and labial cortical plates are also seen.^(24,26)

In *Osteomalacia* affecting adults, there is incomplete mineralization of osteoid that leads to pseudo fractures. There is decrease in Ca/PO₄ ratio, increase in alkaline phosphatase, and decrease in

calcium excretion. Osteoid tissue is formed in the defect but there is no calcium available to be deposited in the osteoid and the zone is called as Looser's zone. A poorly calcified ribbon like zone extending into bone at approximately right angles to the periosteal margin is also seen radiographically. There is increased tendency towards fracture, peculiar waddling or penguin gait, tetany and green stick bone fractures.^(25,27)

Conclusion

Alveolar bone is a dynamic tissue that undergoes a constant state of flux in response to physiological and pathological influences. These pathological influences alter the key factors involved in the remodelling processes of the alveolar bone, results in net bone loss and, therefore, in a lower bone mass and increased risk of fracture.

Therefore, understanding the structural changes occurring in the alveolar bone as a result of systemic influences gives an upper hand in the diagnosis and in employing the adequate treatment modality to restore the anatomical and functional form of alveolar bone.

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