Alveolar bone loss and mineralization in the pig with experimental periodontal disease

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Abstract

Objective: To address how experimental periodontal disease affects alveolar bone mass and mineral apposition in a young pig model.

Materials and methods: Seven three-month-old pigs were periodically inoculated with 4 types of periodontal bacteria, along with a ligature around the last maxillary deciduous molar for 8 weeks to induce periodontal disease (PG). Eight same-aged pigs served as the control (CG). Segmentations of 3D cone-beam CT images were performed to quantify volumes of the total alveolar bone, alveolar ridge, and all roots of the target molar. Calcein and alizarin were administered for labeling mineral apposition before euthanasia. The harvested molar blocks were sectioned and examined under epifluorescence. The inter-label distance between the two vital markers at regional bone surfaces were measured and mineral apposition rate (MAR) was calculated.

Results: A significant reduction of total alveolar bone volume was seen in PG with the major loss at the alveolar ridge. MAR was significantly higher at the root furcation region than those at both buccal and palatal ridges in CG. Compared
with CG, PG animals showed more interrupted labeled bands with significantly lower MAR at the furcation region. MARs were positively associated with both the volumes of total alveolar bone and ridge in CG, but only with the total alveolar bone in PG.

**Conclusions:** In young growing pigs, mineral apposition is region specific. The experimental periodontal disease not only leads to alveolar bone loss, but also perturbs mineral apposition for new bone formation, thus impairing the homeostasis of alveolar bone remodeling.

Keyword: Dentistry

1. **Introduction**

Periodontal disease is a serious infection that damages soft and hard tissues of the periodontium, including the alveolar bone, thus the destruction of alveolar bone is a hallmark of periodontal disease. A number of studies have placed a focus on how inflammation from the periodontal disease and involving bacteria leads to alveolar bone loss through various proinflammatory molecules and cytokine networks (Di Benedetto et al., 2013; Henderson and Kaiser, 2018; Hienz et al., 2015), and other studies characterized additional features of alveolar bone loss resulting from periodontal disease (Cochran, 2008). Since coupled bone resorption and formation is the key mechanism to maintain homeostasis of the healthy skeletal system (Hienz et al., 2015; Rodan, 1998), excessive alveolar bone resorption may be compensated by more active bone formation to restore or preserve the homeostatic status. However, this homeostatic mechanism could be perturbed by the existence of destructive proinflammatory molecules and cytokines along with mechanic traumas from altered occlusion in periodontal disease, tipping the balance in favor of bone resorption over bone formation (Hienz et al., 2015; Rodan and Martin, 2000). Due to this imbalance, bone loss takes place and the reduction of regional mineralization is anticipated. Although matrix vesicle and nuleation theories have been proposed for calcification process in the formation of new bone (Hienz et al., 2015), few studies have addressed how alveolar bone loss is related to the bone calcification process through regional mineralization in periodontal disease. This knowledge gap prevents us from better understanding the mechanism of bone pathophysiology in periodontal disease. Therefore, the purpose of the current study was to quantify the regional bone loss vs regional mineral apposition on alveolar bone in a young pig model with experimental periodontal disease. It is hypothesized that mineral apposition at the surfaces of alveolar bone is region-specific in normal growth and remodeling, but experimental periodontal disease impedes this process, distorting the normal interactions between alveolar bone loss and mineral apposition.
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