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Abstract:

Objectives – To test the effect of bisphosphonate (BP) treatment for up to 3 years on bone necrosis and osteocyte death in the mandible using a canine model. Materials and Methods – Dogs were treated with clinical doses of oral alendronate (ALN, 0.2 or 1.0 mg/kg/day) for 1 or 3 years. In a separate study, dogs were treated with i.v. zoledronate (ZOL) at 0.06 mg/kg/day for 6 months. En bloc staining was used to identify necrotic areas in the mandible; viable osteocytes were identified using lactate dehydrogenase. Results – None of the treatments was associated with exposed bone, but 17–25% of dogs treated for 1 year and 25–33% of dogs treated for 3 years with ALN showed pockets of dead bone. Necrotic areas had no viable osteocytes and were void of patent canaliculi. No control animals demonstrated necrotic bone. ZOL treatment for 6 months was associated with osteocyte death greater than that seen in animals treated with ALN or saline. It is not clear whether osteocyte death occurs because of direct toxic effects of BPs, or because suppressed remodelling fails to renew areas that naturally undergo cell death. Necrotic areas are also associated with bone other than the mandible, e.g. the rib, which normally undergo high rates of remodelling. Conclusions – Reduced remodelling rate

using BPs may contribute to the pathogenesis of bone matrix necrosis. The development of an animal model that mimics important aspects of BP-related osteonecrosis of the jaw is important to understanding the pathogenesis of osteonecrosis.

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