Bone 83 (2016) 276–282

Contents lists available at ScienceDirect

Bone

journal homepage: www.elsevier.com/locate/bone

Abstracts from the XXXI Annual Meeting of the Argentine Association of Osteology and Mineral Metabolism, Buenos Aires, Argentina

Bone repair: A comparative study of the effect of calcium hydroxide [$Ca(OH)_2$] in short and long bones

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The last researches have focused on the study of biomaterials. $Ca(OH)_2$ would promote the acceleration of bone repair. Our aim is to evaluate the effect of $Ca(OH)_2$ in bone regeneration of experimental cavities in tibia and in dental post-extraction. Wistar rats were divided into two groups: A—Both tibias were exposed and a cavity of 1.5 mm diameter was made. The left was filled with $Ca(OH)_2$ powder and the right was not filled (control). B—Both first mandibular molars were extracted. A collagen sponge was placed in the alveoli. The left alveoli was previously treated with $Ca(OH)_2$. Both groups were sacrificed at 7, 15 and 30 days. The tibias and hemimandibles were processed for microscopic analysis. The bone repair was similar in the control cases, both tibia and mandible. At 7 days hyalinization of the bone matrix in alveoli problems, loss of viable osteocytes and/or osteocytic cavities, and material added in bone matrix could be observed. In tibia cavities we observed a large amount of reactive trabecular bone with a high cell density. At 15 days in the alveoli an inflammatory infiltrate was observed. Chondroid tissue cores surrounded by an acidophilus hyaline substance and bone tissue with osteoblasts at the area in contact with the $Ca(OH)_2$ could be noted. Tibial images showed typical endochondral ossification in the repair area. At the surface of the cavity, tissue adopted a compact bone pattern. At 30 days in the alveoli the chondroid tissue was surrounded by newly formed bone and capillary neoformation in Haversian canal. Conclusions: In both experimental situations we can establish that $Ca(OH)_2$ caused inflammatory processes in short terms. In the long terms $Ca(OH)_2$ bone repair occurred both in tibia and alveoli. The chondroid images in the repair of the post-extraction alveoli is remarkable.

doi:10.1016/j.bone.2014.12.026

Effect of testosterone on the regulation of protein and gene expression related to oxidative stress damage in C2C12 cells

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The loss of muscle mass and strength with aging, also referred to as sarcopenia, is a prevalent condition among the elderly, associated with a deficit of sex hormones. In our previous works we have demonstrated that testosterone protects against H_2O_2-induced apoptosis in C2C12 at different levels: morphological, physiological, biochemical and molecular. In the present study we evaluated some of the components upstream of the classical apoptotic pathway which could trigger this response, the expression levels of genes related to these pathways and the role of the hormone during these events. By Western blot and immunocytochemistry, we observed that H_2O_2 treatment induces the activation of p53 in a time-dependent way. Phosphorylation of p53 induced by H_2O_2 is reduced by testosterone treatment prior to H_2O_2. One of the ways that p53 induces apoptosis is through p66Shc activation, an adaptor protein which amplifies the generation of mitochondrial hydrogen peroxide. Our studies showed that hormone incubation prior to H_2O_2 reduces p66Shc activation and its mitochondrial localization. Furthermore, testosterone diminished JNK phosphorylation induced by the apoptotic agent. So JNK could be acting as a p66Shc activator, since pretreatment with a JNK inhibitor decreased the phosphorylation not only of JNK but also of p66Shc. Finally, mRNA levels of different pro and anti-apoptotic genes were determined by real time PCR, during the apoptotic stimuli in the presence or absence of the hormone, showing that testosterone has opposite effects over gene expression to H_2O_2. Testosterone would therefore favor the expression of genes related to proliferation and survival respect of those associated to apoptosis.

doi:10.1016/j.bone.2014.12.027

Severe and persistent hypocalcemia following denosumab in a patient with osteoblastic bone metastases

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Advanced prostate cancer has a strong propensity to metastasize to bone. Serious complications from bone metastases include severe pain, pathological fractures, and compression of the spinal cord. These skeletal-related events (SRE) can negatively impact quality of life for patients. Bisphosphonates and recently denosumab, have been shown to reduce SRE. Both are associated with adverse effects including hypocalcemia, which is generally mild and transient. We report a case of severe and prolonged hypocalcemia requiring hospitalization. A 67-year-old man was presented for management symptomatic hypocalcemia. Prostate cancer was diagnosed three years before and progressed despite of androgen-deprivation therapy. When osteoblastic bone metastasis was diagnosed, he received radiotherapy and started denosumab (120 mg monthly). Six months later he was admitted because of hypocalcemia that persisted for 30 days despite high intravenous and oral calcium and calcitriol doses. The next 5 months he needed high oral calcium and calcitriol supplementation. At 6 months osteoblastic activity was reduced by Docetaxel (expressed by decrease in bone scintigraphy uptake and alkaline phosphatase) and denosumab effect ceased (increase in CTXs that were very low at admission). Then reduction in calcium and calcitriol supplementation were possible. **Conclusions:** 1) We postulate that hypocalcemia was due to avid calcium uptake by excess osteoblastic activity unopposed by compensatory mechanisms. Bone resorption was profoundly depressed as showed very low CTXs. 2) Denosumab related hypocalcemia in osteoblastic metastasis may be more severe and prolonged than previously reported. 3) In those patients a close followup is mandatory.

doi:10.1016/j.bone.2014.12.028

Bone properties of rats treated with fluoride in combination with mechanical stress due to physical activity

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Fluorine is found in nature as fluoride (F) in drinking water and food. Fluoride has an important role in the prevention and control of dental caries. However in areas where the water fluoride concentration exceeds the limit of WHO (1.5 ppm), bone alterations and metabolic disorders such as insulin resistance have been reported. Our laboratory has demonstrated that the insulin resistance induced by fluoride, could be reversed with physical activity, due to an increased incorporation of F into bones. Therefore, the aim of this study was to evaluate the strength and composition of bone material in animals that reversed their fluoride-induced insulin resistance through physical activity. Sprague-Dawley rats were divided into four groups in which exercise and F intake were evaluated: (n = 6/group): S (no exercise and drinking

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